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A 46-year-old man with a 30-pack-year smoking history develops right leg pain and swelling after returning from an overseas trip. He is diagnosed with a lower extremity deep venous thrombosis and is started on intravenous heparin. Shortly after being hospitalized, he develops right-sided weakness and facial droop. CT scan of the head reveals a left middle cerebral artery stroke. Which of the following physical examination findings is most likely to be present in this patient?

A.

Diastolic decrescendo-type murmur that decreases following amyl nitrite inhalation

B.

Ejection-type systolic murmur that increases on standing

C.

Presystolic murmur that disappears with atrial fibrillation

D.

Midsystolic click that occurs earlier with inspiration

E.

Wide splitting of S2 that does not change with respiration

Submit

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A 46-year-old man with a 30-pack-year smoking history develops right leg pain and swelling after returning from an overseas trip. He is diagnosed with a lower extremity deep venous thrombosis and is started on intravenous heparin. Shortly after being hospitalized, he develops right-sided weakness and facial droop. CT scan of the head reveals a left middle cerebral artery stroke. Which of the following physical examination findings is most likely to be present in this patient?

☐ A. Diastolic decrescendo-type murmur that decreases following amyl nitrite inhalation [6%]

☐ B. Ejection-type systolic murmur that increases on standing [9%]

☐ C. Presystolic murmur that disappears with atrial fibrillation [15%]

☐ D. Midsystolic click that occurs earlier with inspiration [7%]

☒ E. Wide splitting of S2 that does not change with respiration [60%]

Omitted

Correct answer
E

60%
Answered correctly

35 Seconds
Time Spent

10/29/2018
Last Updated

Explanation

A cerebrovascular event (eg, transient ischemic attack, stroke) in the setting of known venous thromboembolic disease is suspicious for **paradoxical embolism**. Paradoxical emboli originate in the systemic venous circulation (lower or upper extremities) and enter the systemic arterial circulation via an intracardiac or intrapulmonary shunt. They can occur in patients with **patent foramen ovale**, **atrial septal defects**, **ventricular septal defects**, or large **pulmonary arteriovenous malformations**.

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A cerebrovascular event (eg, transient ischemic attack, stroke) in the setting of known venous thromboembolic disease is suspicious for **paradoxical embolism**. Paradoxical emboli originate in the systemic venous circulation (lower or upper extremities) and enter the systemic arterial circulation via an intracardiac or intrapulmonary shunt. They can occur in patients with **patent foramen ovale, atrial septal defects, ventricular septal defects, or large pulmonary arteriovenous malformations**.

Atrial septal defects with left-to-right shunting typically cause **wide and fixed splitting** (no change with respiration) of the second heart sound (S2). Patients may also have a mid-systolic ejection murmur over the left upper sternal border resulting from increased flow across the pulmonic valve and a mid-diastolic rumble due to increased flow across the tricuspid valve. Importantly, a paradoxical embolism can occur even in patients with net left-to-right shunting due to **transient reversal of the shunt** during periods of elevated right-sided pressure (eg, early ventricular systole, straining during coughing or defecation).

(Choice A) An early diastolic decrescendo murmur is characteristic of aortic regurgitation. Inhaled amyl nitrite produces marked vasodilatation, which reduces systemic arterial pressure and regurgitant flow (lessening the murmur).

(Choice B) A systolic ejection murmur that increases in intensity with **standing** can be heard in patients with hypertrophic cardiomyopathy (due to a decrease in left ventricular outflow tract size). In contrast, the murmur of valvular aortic stenosis decreases in intensity upon standing.

(Choice C) Mitral or tricuspid valve stenosis can cause a diastolic murmur with presystolic accentuation due to atrial contraction. With mild stenosis, the murmur may only be audible during the accentuation phase in late diastole; atrial fibrillation may cause the murmur to disappear completely.

(Choice D) The midsystolic click of mitral valve prolapse occurs **earlier** with inspiration due to the reduced left ventricular volume caused by decreased venous return to the left heart.

Educational objective:

Paradoxical embolism occurs when a thrombus from the venous system crosses into the arterial circulation via an abnormal connection between the right and left cardiac chambers (eg, patent foramen ovale, atrial septal defect, or ventricular septal defect). Atrial left-to-right shunts cause

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ventricular septal defects, or large pulmonary arteriovenous malformations.

Atrial septal defects with left-to-right shunting typically cause **wide and fixed splitting** (no change with respiration) of the second heart sound (S2). Patients may also have a mid-systolic ejection murmur over the left upper sternal border resulting from increased flow across the pulmonic valve and a mid-diastolic rumble due to increased flow across the tricuspid valve. Importantly, a paradoxical embolism can occur even in patients with net left-to-right shunting due to **transient reversal of the shunt** during periods of elevated right-sided pressure (eg, early ventricular systole, straining during coughing or defecation).

(Choice A) An early diastolic decrescendo murmur is characteristic of aortic regurgitation. Inhaled amyl nitrite produces marked vasodilatation, which reduces systemic arterial pressure and regurgitant flow (lessening the murmur).

(Choice B) A systolic ejection murmur that increases in intensity with **standing** can be heard in patients with hypertrophic cardiomyopathy (due to a decrease in left ventricular outflow tract size). In contrast, the murmur of valvular aortic stenosis decreases in intensity upon standing.

(Choice C) Mitral or tricuspid valve stenosis can cause a diastolic murmur with presystolic accentuation due to atrial contraction. With mild stenosis, the murmur may only be audible during the accentuation phase in late diastole; atrial fibrillation may cause the murmur to disappear completely.

(Choice D) The midsystolic click of mitral valve prolapse occurs earlier with inspiration due to the reduced left ventricular volume caused by decreased venous return to the left heart.

Educational objective:

Paradoxical embolism occurs when a thrombus from the venous system crosses into the arterial circulation via an abnormal connection between the right and left cardiac chambers (eg, patent foramen ovale, atrial septal defect, or ventricular septal defect). Atrial left-to-right shunts cause wide and fixed splitting of S2 and can facilitate paradoxical embolism due to periods of transient shunt reversal (eg, during straining or coughing).

References

Paradoxical embolism

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The public health department of a developing country performs an epidemiologic study to assess the nationwide incidence of upper respiratory infections among children. The data show a high rate of childhood bacterial pharyngitis. Current practice guidelines indicate that a rapid test should be performed in children to identify the presence of bacterial antigens. If this test is negative, a throat culture, the gold standard for definitive diagnosis, is then performed. To offset cost and avoid losing patients to follow-up, the department is considering recommending the empiric use of penicillin for the treatment of suspected bacterial pharyngitis. Which of the following would be expected to decrease after long-term implementation of this guideline?

A. Deaths associated with diarrheal illness

B. Diagnoses of serum sickness-like reactions

C. Incidence of nephritic syndrome

D. Need for cardiac surgery

E. Use of broad-spectrum antibiotics

Submit

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Settings

The public health department of a developing country performs an epidemiologic study to assess the nationwide incidence of upper respiratory infections among children. The data show a high rate of childhood bacterial pharyngitis. Current practice guidelines indicate that a rapid test should be performed in children to identify the presence of bacterial antigens. If this test is negative, a throat culture, the gold standard for definitive diagnosis, is then performed. To offset cost and avoid losing patients to follow-up, the department is considering recommending the empiric use of penicillin for the treatment of suspected bacterial pharyngitis. Which of the following would be expected to decrease after long-term implementation of this guideline?

☐ A. Deaths associated with diarrheal illness [2%]

☐ B. Diagnoses of serum sickness-like reactions [2%]

☐ C. Incidence of nephritic syndrome [30%]

☒ D. Need for cardiac surgery [53%]

☐ E. Use of broad-spectrum antibiotics [10%]

Omitted

Correct answer
D

53%

Answered correctly

12 Seconds

Time Spent

12/04/2018

Last Updated

Explanation

Acute rheumatic fever

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Explanation

Acute rheumatic fever	
Epidemiology	• Endemic in developing countries
Pathogenesis	• Occurs 2-4 weeks after acute group A streptococcal pharyngitis • Molecular mimicry: Anti-streptococcal antibodies attack cardiac & neuronal antigens
Clinical features	• Acute/subacute ◦ Migratory arthritis ◦ Pancarditis (mitral regurgitation) ◦ Sydenham chorea • Chronic ◦ Mitral stenosis
Prevention	• Prompt treatment of streptococcal pharyngitis with penicillin

Early **penicillin** treatment of **group A streptococcal pharyngitis** is important for the **prevention of acute rheumatic fever** (ARF), a major cause of cardiovascular death in many developing nations. ARF primarily affects the heart and central nervous system because host antibodies produced in response to streptococcal antigens cross-react with host antigens in these organs. Chronic cardiac inflammation can progress to rheumatic heart disease, specifically valvular disease. The **mitral valve** is the most commonly affected and gradually thickens, fibroses, and calcifies, eventually requiring valvotomy or more invasive surgical intervention.

Empiric therapy for a condition must be considered in the context of the host characteristics, pre-test probability of the disease, benefits/risks of waiting for a definitive diagnosis, and cost of therapy versus its potential complications. If all cases of acute streptococcal pharyngitis were treated empirically, the incidence of rheumatic heart disease and associated cardiac procedures would likely decrease.

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or cardiovascular death in many developing nations. ARF primarily affects the heart and central nervous system because most antibodies produced in response to streptococcal antigens cross-react with host antigens in these organs. Chronic cardiac inflammation can progress to rheumatic heart disease, specifically valvular disease. The **mitral valve** is the most commonly affected and gradually thickens, fibroses, and calcifies, eventually requiring valvotomy or more invasive surgical intervention.

Empiric therapy for a condition must be considered in the context of the host characteristics, pre-test probability of the disease, benefits/risks of waiting for a definitive diagnosis, and cost of therapy versus its potential complications. If all cases of acute streptococcal pharyngitis were treated empirically, the incidence of rheumatic heart disease and associated cardiac procedures would likely decrease.

(Choice A) Antibiotic use can promote diarrhea by altering the gut microbial milieu. This alteration can lead to overgrowth of *Clostridium difficile*, a gram-positive bacterium that can cause profuse, watery diarrhea and life-threatening fulminant colitis. Therefore, increasing antibiotic use would likely result in increased diarrheal illness and potential complications.

(Choice B) Serum sickness-like reactions are associated with certain infections (eg, hepatitis B) and antibiotics (eg, penicillin). The development of circulating drug-specific immune complexes may cause fever, rash, and arthritis. If penicillin was used more frequently, serum sickness-like reactions would potentially increase, not decrease.

(Choice C) Post-streptococcal glomerulonephritis (PSGN) is the most common cause of nephritic syndrome in children worldwide. It is caused by deposition of immune complexes in glomeruli following pharyngitis or skin infections with group A *Streptococcus*. Although early antibiotics are effective in preventing ARF, they have not been shown to reduce the risk of PSGN.

(Choice E) Empiric use of potentially unnecessary antibiotics may cause an increase in antibiotic resistance, thereby increasing the need for broad-spectrum antibiotics. For example, methicillin-resistant *Staphylococcus aureus* (MRSA) evolved from methicillin-sensitive *S aureus* via alteration of the protein binding site for beta-lactam antibiotics. As a result, broad-spectrum antibiotics (eg, vancomycin, daptomycin) are required for MRSA infections.

Educational objective:

Acute rheumatic fever is a complication of untreated group A streptococcal pharyngitis. Rheumatic heart disease is the most common cause of acquired valvular heart disease and cardiovascular death in developing countries. The incidence of acute rheumatic fever and rheumatic heart

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Educational objective:

Acute rheumatic fever is a complication of untreated group A streptococcal pharyngitis. Rheumatic heart disease is the most common cause of acquired valvular heart disease and cardiovascular death in developing countries. The incidence of acute rheumatic fever and rheumatic heart disease has been reduced in industrialized nations with prompt treatment of streptococcal pharyngitis with penicillin.

References

- Primary prevention of acute rheumatic fever and rheumatic heart disease with penicillin in South African children with pharyngitis: a cost-effectiveness analysis.
- Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 Update by the Infectious Diseases Society of America.

Diagnostic criteria of acute rheumatic fever

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A 53-year-old man comes to the office with progressive exertional dyspnea. He has smoked 2 packs of cigarettes per day for the last 35 years. Physical examination shows increased anteroposterior diameter of his chest. Auscultation reveals decreased breath sounds and scattered wheezes throughout his lungs. Examination of his extremities is unremarkable. An echocardiogram reveals moderate dilation of the right ventricle and increased central venous pressure. The absence of peripheral edema in this patient is best explained by which of the following compensatory mechanisms?

☐

A. Decreased capillary permeability

☐

B. Decreased circulating aldosterone levels

☐

C. Decreased interstitial fluid pressure

☐

D. Increased plasma oncotic pressure

☐

E. Increased tissue lymphatic drainage

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☐

A. Decreased capillary permeability [8%]

☐

B. Decreased circulating aldosterone levels [23%]

☐

C. Decreased interstitial fluid pressure [5%]

☐

D. Increased plasma oncotic pressure [22%]

☒

E. Increased tissue lymphatic drainage [38%]

Omitted

Correct answer

E

38%

Answered correctly

4 Seconds

Time Spent

11/18/2018

Last Updated

Explanation

This patient has dyspnea, signs of lung hyperinflation, and a heavy smoking history, suggesting chronic obstructive pulmonary disease (COPD). Chronic hypoxia in COPD can lead to pulmonary vasoconstriction, increased pulmonary artery pressure, and right heart failure (ie, cor pulmonale).

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Explanation

This patient has dyspnea, signs of lung hyperinflation, and a heavy smoking history, suggesting chronic obstructive pulmonary disease (COPD). Chronic hypoxia in COPD can lead to pulmonary vasoconstriction, increased pulmonary artery pressure, and right heart failure (ie, cor pulmonale), which is confirmed by this patient's echocardiogram. In turn, right heart failure leads to increased central venous pressure and excessive capillary hydrostatic pressure, which predisposes him to developing **peripheral edema**.

Fluid movement across the capillary wall (filtration) into the interstitium is dependent on hydrostatic pressure, oncotic pressure, and capillary permeability. It is tightly balanced by lymphatic drainage, which returns interstitial fluid to the vasculature. Peripheral edema (accumulation of excess fluid in the interstitial space) develops when transcapillary plasma filtration exceeds the resorptive capacity of the lymphatics. Factors that favor development of edema include:

- **Elevated capillary hydrostatic pressure:** Higher capillary pressures increase plasma filtration into the interstitium, particularly in dependent tissues. Causes include arteriolar dilation (eg, dihydropyridine calcium channel blockers) and impaired venous return (eg, high central venous pressure, venous thrombosis).
- **Decreased plasma oncotic pressure:** Plasma proteins (eg, albumin) generate oncotic pressure that pulls interstitial fluid back into the capillary bed venules. Edema develops in conditions with decreased albumin levels (eg, nephrotic syndrome, cirrhosis, malnutrition).
- **Sodium and water retention:** Increased intravascular volume raises capillary hydrostatic pressure and decreases plasma oncotic pressure (dilutes plasma proteins). Causes include kidney disease and heart failure.
- **Lymphatic obstruction:** Decreased lymphatic return impairs removal of excess interstitial fluid. Common causes of lymphatic obstruction include filariasis, invasive malignancies, and iatrogenic etiologies (eg, surgical lymph node dissection and radiation therapy).

Increased capillary fluid transudation **raises interstitial hydrostatic pressure**, resulting in **increased lymphatic drainage**. This compensatory response can delay the development of clinically apparent edema. However, progressive heart failure, cirrhosis, or renal failure can eventually overwhelm lymphatic return capacity and produce significant edema.

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- Decreased plasma oncotic pressure: Plasma proteins (eg, albumin) generate oncotic pressure that pulls interstitial fluid back into the capillary bed venules. Edema develops in conditions with decreased albumin levels (eg, nephrotic syndrome, cirrhosis, malnutrition).
- Sodium and water retention: Increased intravascular volume raises capillary hydrostatic pressure and decreases plasma oncotic pressure (dilutes plasma proteins). Causes include kidney disease and heart failure.
- Lymphatic obstruction: Decreased lymphatic return impairs removal of excess interstitial fluid. Common causes of lymphatic obstruction include filariasis, invasive malignancies, and iatrogenic etiologies (eg, surgical lymph node dissection and radiation therapy).

Increased capillary fluid transudation **raises interstitial hydrostatic pressure**, resulting in **increased lymphatic drainage**. This compensatory response can delay the development of clinically apparent edema. However, progressive heart failure, cirrhosis, or renal failure can eventually overwhelm lymphatic return capacity and produce significant edema.

(Choices A and D) Although increased plasma oncotic pressure and decreased capillary permeability oppose edema formation, neither is associated with COPD. Severe COPD may cause polycythemia due to hypoxia, but erythrocyte concentration does not affect plasma oncotic pressure.

(Choice B) In patients with heart failure, activation of the renin-angiotensin-aldosterone system (RAAS) leads to increased aldosterone levels that cause sodium and water retention (exacerbating edema formation). Volume expansion causes increased atrial/brain natriuretic peptide release, which can partially counteract the effects of RAAS activation; however, aldosterone levels remain above normal in patients with heart failure.

(Choice C) Decreased interstitial fluid pressure favors flow into the interstitium and would contribute to the development of peripheral edema.

Educational objective:

Peripheral edema results from the accumulation of fluid in the interstitial spaces. Factors that promote edema include elevated capillary hydrostatic pressure, decreased plasma oncotic pressure, sodium and water retention, and impaired lymphatic drainage. In chronic heart failure, increased lymphatic drainage initially offsets factors favoring edema, whereas acute changes (eg, venous thrombosis, heart failure decompensation) are more likely to produce edema.

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A 32-year-old man comes to the emergency department with chest pain that started earlier in the day. The pain is midline and sharp and increases with deep breaths but decreases when the patient leans forward. He had a mild respiratory illness a week ago. Other medical history is unremarkable. The patient is a lifetime nonsmoker and has no family history of early-onset heart attack, sudden death, or cardiomyopathy. Blood pressure is 120/70 mm Hg and pulse is 110/min and regular. Which of the following physical examination findings is most expected in this patient?

A. Friction rub

B. Kussmaul sign

C. Pericardial knock

D. Pulsus paradoxus

E. S3

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A 32-year-old man comes to the emergency department with chest pain that started earlier in the day. The pain is midline and sharp and increases with deep breaths but decreases when the patient leans forward. He had a mild respiratory illness a week ago. Other medical history is unremarkable. The patient is a lifetime nonsmoker and has no family history of early-onset heart attack, sudden death, or cardiomyopathy. Blood pressure is 120/70 mm Hg and pulse is 110/min and regular. Which of the following physical examination findings is most expected in this patient?

✓

☒

A. Friction rub [81%]

☐

B. Kussmaul sign [5%]

☐

C. Pericardial knock [3%]

☐

D. Pulsus paradoxus [7%]

☐

E. S3 [1%]

Omitted

Correct answer
A

81%

Answered correctly

5 Seconds

Time Spent

10/03/2018

Last Updated

Explanation

The chest pain of **acute pericarditis** is sharp and pleuritic and characteristically **decreases** when the patient sits and **leans forward**, which decreases pressure on the parietal pericardium. Fibrinous/serofibrinous pericarditis is the most common type of pericarditis, and a **pericardial friction rub** (described as high pitched, leathery, and scratchy) is the most specific physical finding. Fibrinous pericarditis may be caused by myocardial infarction, rheumatologic disease (eg, systemic lupus erythematosus), uremia, or viral infection. This patient's history of a recent upper

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Explanation

The chest pain of **acute pericarditis** is sharp and pleuritic and characteristically **decreases** when the patient sits and **leans forward**, which decreases pressure on the parietal pericardium. Fibrinous/serofibrinous pericarditis is the most common type of pericarditis, and a **pericardial friction rub** (described as high pitched, leathery, and scratchy) is the most specific physical finding. Fibrinous pericarditis may be caused by myocardial infarction, rheumatologic disease (eg, systemic lupus erythematosus), uremia, or viral infection. This patient's history of a recent upper respiratory illness suggests a **viral etiology**.

(Choice B) During inspiration, jugular venous pressure normally decreases as blood is pulled into the right side of the heart. Kussmaul sign is a paradoxical increase in jugular venous pressure on inspiration. It occurs because of impaired right-sided diastolic filling in conditions such as constrictive pericarditis, restrictive cardiomyopathy, and tricuspid stenosis. Although constrictive (chronic) pericarditis can result from prolonged pericardial inflammation (over months to years), this patient's presentation is consistent with acute pericarditis, which is not associated with impaired right-sided diastolic filling.

(Choice C) A pericardial knock is a brief, high-frequency, precordial sound heard in early diastole (shortly after S2) in patients with constrictive pericarditis. It occurs earlier than S3 and may be confused with the opening snap of mitral stenosis.

(Choice D) **Pulsus paradoxus** is a drop in systolic blood pressure >10 mm Hg during inspiration. It represents an exaggeration of the normal reduction in systolic blood pressure during inspiration. It can be seen in cardiac tamponade, constrictive pericarditis, and severe asthma or chronic obstructive pulmonary disease.

(Choice E) An S3 typically results from rapid cessation of diastolic filling into an enlarged ventricle. It is usually associated with heart failure such as occurs with dilated cardiomyopathy, severe mitral regurgitation, or severe aortic regurgitation. A right-sided S3 can occur with right-sided heart failure.

Educational objective:

Acute-onset, sharp, and pleuritic chest pain that decreases with leaning forward is characteristic of acute pericarditis. Fibrinous/serofibrinous pericarditis is the most common form of pericarditis and a pericardial friction rub is the most specific physical finding. Viral pericarditis is often



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respiratory illness suggests a **viral etiology**.

(Choice B) During inspiration, jugular venous pressure normally decreases as blood is pulled into the right side of the heart. Kussmaul sign is a paradoxical increase in jugular venous pressure on inspiration. It occurs because of impaired right-sided diastolic filling in conditions such as constrictive pericarditis, restrictive cardiomyopathy, and tricuspid stenosis. Although constrictive (chronic) pericarditis can result from prolonged pericardial inflammation (over months to years), this patient's presentation is consistent with acute pericarditis, which is not associated with impaired right-sided diastolic filling.

(Choice C) A pericardial knock is a brief, high-frequency, precordial sound heard in early diastole (shortly after S2) in patients with constrictive pericarditis. It occurs earlier than S3 and may be confused with the opening snap of mitral stenosis.

(Choice D) **Pulsus paradoxus** is a drop in systolic blood pressure >10 mm Hg during inspiration. It represents an exaggeration of the normal reduction in systolic blood pressure during inspiration. It can be seen in cardiac tamponade, constrictive pericarditis, and severe asthma or chronic obstructive pulmonary disease.

(Choice E) An S3 typically results from rapid cessation of diastolic filling into an enlarged ventricle. It is usually associated with heart failure such as occurs with dilated cardiomyopathy, severe mitral regurgitation, or severe aortic regurgitation. A right-sided S3 can occur with right-sided heart failure.

Educational objective:

Acute-onset, sharp, and pleuritic chest pain that decreases with leaning forward is characteristic of acute pericarditis. Fibrinous/serofibrinous pericarditis is the most common form of pericarditis and a pericardial friction rub is the most specific physical finding. Viral pericarditis is often preceded by an upper respiratory infection.

References

- [Acute pericarditis](#).

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Text Zoom

A 34-year-old man comes to the office due to heart palpitations that are particularly prominent at night. With moderate exertion he also experiences head-pounding accompanied by involuntary head-bobbing. The patient recently emigrated from Southeast Asia and remembers being diagnosed with a heart murmur years before, but he cannot recall the type of murmur and has never received any treatment. He has no other medical conditions and takes no medications. He does not use tobacco, alcohol, or illicit drugs. The patient's father has coronary artery disease and his mother has type 2 diabetes mellitus. Based on this patient's history, which of the following findings is most likely to be present on further evaluation?

A. Impaired left ventricular contractility

B. Left ventricular outflow obstruction

C. Restricted left ventricular filling

D. Systolic and diastolic hypertension

E. Widening of the pulse pressure

Submit

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TUTOR

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A 34-year-old man comes to the office due to heart palpitations that are particularly prominent at night. With moderate exertion he also experiences head-pounding accompanied by involuntary head-bobbing. The patient recently emigrated from Southeast Asia and remembers being diagnosed with a heart murmur years before, but he cannot recall the type of murmur and has never received any treatment. He has no other medical conditions and takes no medications. He does not use tobacco, alcohol, or illicit drugs. The patient's father has coronary artery disease and his mother has type 2 diabetes mellitus. Based on this patient's history, which of the following findings is most likely to be present on further evaluation?

- ☐ A. Impaired left ventricular contractility [2%]
- ☐ B. Left ventricular outflow obstruction [9%]
- ☐ C. Restricted left ventricular filling [5%]
- ☐ D. Systolic and diastolic hypertension [2%]
- ☒ E. Widening of the pulse pressure [79%]

Omitted

Correct answer
E79%
Answered correctly7 Seconds
Time Spent10/28/2018
Last Updated

Explanation

Chronic aortic regurgitation

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TUTOR





Chronic aortic regurgitation

Common etiologies	• Congenital bicuspid aortic valve • Postinflammatory (eg, rheumatic heart disease, endocarditis) • Aortic root dilation (eg, Marfan syndrome, syphilis)
Pathophysiology	• Backflow from aorta into LV → ↑ LV end-diastolic volume • LV initially compensates with eccentric hypertrophy → ↑ SV & CO • Eventual LV dysfunction → ↓ SV & CO → heart failure
Clinical findings	• Diastolic decrescendo murmur • Widened pulse pressure (↑SBP & ↓DBP) • Rapid rise-rapid fall ("water-hammer") pulsation • Abrupt carotid distension & collapse, "pistol-shot" femoral pulses

CO = cardiac output; DBP = diastolic blood pressure; LV = left ventricle; SBP = systolic blood pressure; SV = stroke volume.

This patient has a number of clinical findings that are characteristic of **chronic aortic regurgitation (AR)**. Failure of the aortic valve to tightly seal off the aorta from the left ventricle at the end of systole leads to rapid loss of aortic pressure and an overall **decrease in diastolic blood pressure**. In addition, the left ventricle undergoes **eccentric hypertrophy** to initially compensate for an increase in left ventricular end-diastolic volume, which leads to increased stroke volume and cardiac output. Because pulse pressure (the difference between diastolic and systolic blood pressure) is proportional to stroke volume, the compensatory **increased stroke volume** on top of reduced diastolic pressure causes the characteristic **widened pulse pressure** seen with AR.

This high-amplitude pulsation with rapid diastolic collapse is responsible for the unusual pulsation findings in AR. Forceful pulsations in the intracranial arteries can cause **head bobbing** with each heart beat (de Musset sign), and patients may experience **palpitations** due to forceful left ventricular contraction. In addition, physical examination can show abrupt distension and collapse of the carotid arteries (Corrigan sign) and peripheral arteries ("water-hammer" pulse), and reveal "pistol-shot" femoral pulses (Traube sign) on auscultation.

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Text Zoom

pressure. In addition, the left ventricle undergoes **eccentric hypertrophy** to initially compensate for an increase in left ventricular end-diastolic volume, which leads to increased stroke volume and cardiac output. Because pulse pressure (the difference between diastolic and systolic blood pressure) is proportional to stroke volume, the compensatory **increased stroke volume** on top of reduced diastolic pressure causes the characteristic **widened pulse pressure** seen with AR.

This high-amplitude pulsation with rapid diastolic collapse is responsible for the unusual pulsation findings in AR. Forceful pulsations in the intracranial arteries can cause **head bobbing** with each heart beat (de Musset sign), and patients may experience **palpitations** due to forceful left ventricular contraction. In addition, physical examination can show abrupt distension and collapse of the carotid arteries (Corrigan sign) and peripheral arteries ("water-hammer" pulse), and reveal "pistol-shot" femoral pulses (Traube sign) on auscultation.

(Choice A) Impaired left ventricular contractility occurs later in the course of AR and eventually leads to heart failure. Because head-bobbing and other characteristic findings in AR are due to increased stroke volume and high-amplitude, rapid rise-rapid fall pulsation, these findings are less prominent once impaired left ventricular contractility (and decreased stroke volume) sets in.

(Choices B and C) Left ventricular outflow tract obstruction, such as occurs with aortic stenosis, leads to reduced stroke volume and characteristic weak and delayed pulses (pulsus parvus et tardus). Restriction of left ventricular filling, as occurs in mitral stenosis, also reduces stroke volume due to reduced left ventricular end-diastolic volume (reduced preload). Clinical findings associated with high-amplitude pulsation will not be present with reduced stroke volume.

(Choice D) Combined systolic and diastolic hypertension is not necessarily accompanied by widened pulse pressure; the signs of high amplitude, rapid rise-rapid fall pulsation are more characteristic of chronic AR.

Educational objective:

Chronic aortic regurgitation (AR) causes a reduction in diastolic blood pressure and a compensatory increase in left ventricular stroke volume. These changes create a high-amplitude, rapid rise-rapid fall pulsation (ie, widened pulse pressure) and the other characteristic findings of AR (eg, head bobbing, "pistol-shot" femoral pulses).

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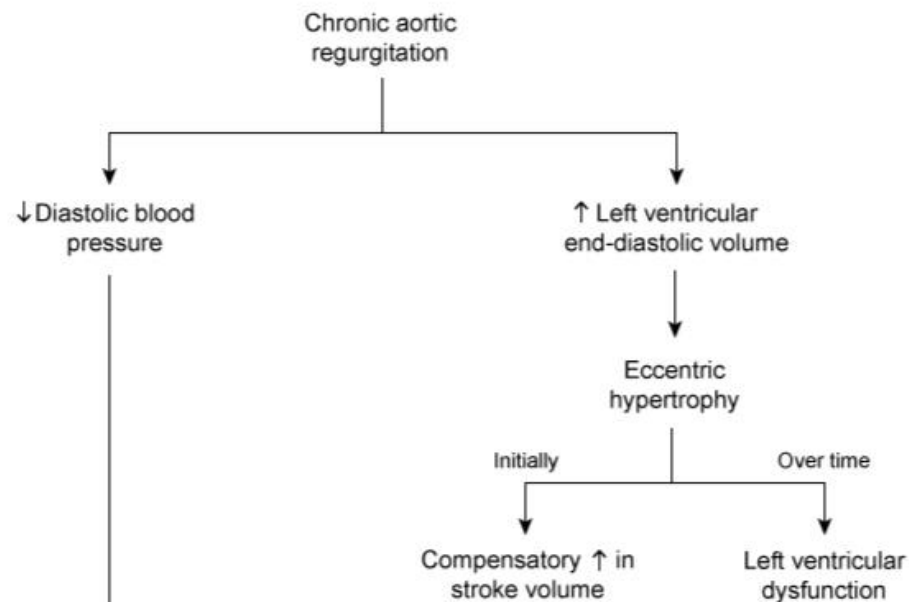
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Exhibit Display

Pathophysiology of chronic aortic regurgitation



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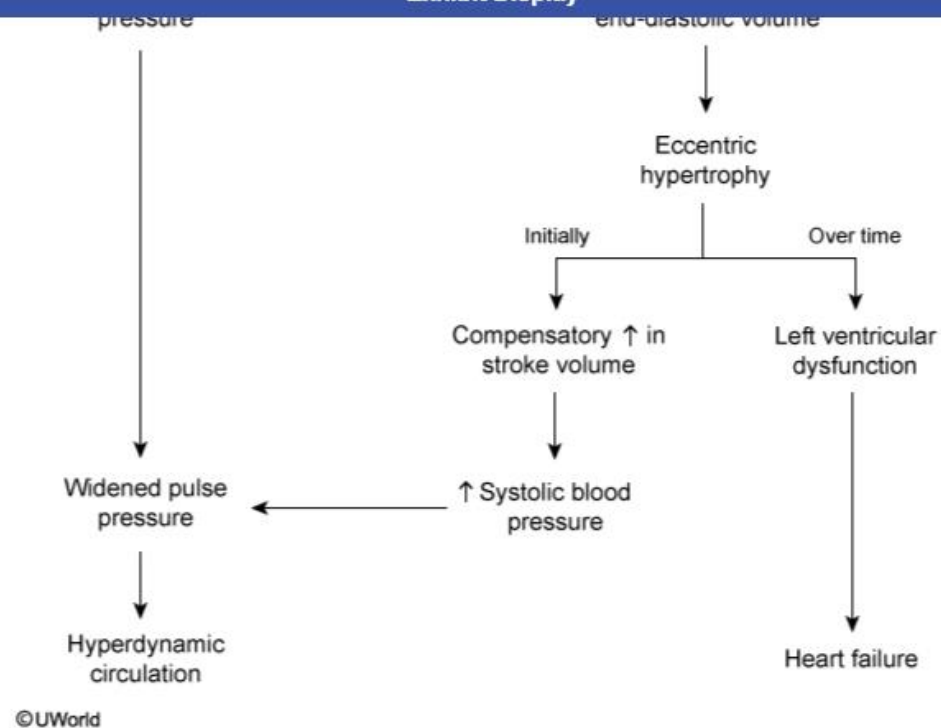


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Exhibit Display



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Text Zoom

A 34-year-old man comes to the emergency department with fatigue and lightheadedness. The patient had an upper respiratory infection last week, and since then his energy level has been low with shortness of breath on mild exertion. Otherwise, his medical history is insignificant. He is a lifetime nonsmoker. The patient's temperature is 37 C (98.6 F), blood pressure is 80/60 mm Hg, and pulse is 120/min and regular. His pulse becomes undetectable to palpation during inspiration. The lungs are clear to auscultation, but the jugular veins are distended. Which of the following is the most likely diagnosis?

☐ A. Acute myocardial infarction

☐ B. Acute viral myocarditis

☐ C. Cardiac tamponade

☐ D. Constrictive pericarditis

☐ E. Septic shock

☐ F. Tension pneumothorax

Submit

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TUTOR

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A 34-year-old man comes to the emergency department with fatigue and lightheadedness. The patient had an upper respiratory infection last week, and since then his energy level has been low with shortness of breath on mild exertion. Otherwise, his medical history is insignificant. He is a lifetime nonsmoker. The patient's temperature is 37 C (98.6 F), blood pressure is 80/60 mm Hg, and pulse is 120/min and regular. His pulse becomes undetectable to palpation during inspiration. The lungs are clear to auscultation, but the jugular veins are distended. Which of the following is the most likely diagnosis?

- ☐ A. Acute myocardial infarction [0%]
- ☐ B. Acute viral myocarditis [13%]
- ☒ C. Cardiac tamponade [48%]
- ☐ D. Constrictive pericarditis [33%]
- ☐ E. Septic shock [3%]
- ☐ F. Tension pneumothorax [1%]

Omitted

Correct answer
C48%
Answered correctly4 Seconds
Time Spent09/22/2018
Last Updated

Explanation

This presentation of **hypotension**, tachycardia, **jugular venous distension** with clear lungs, and **pulsus paradoxus** (manifesting as loss of

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This presentation of **hypotension**, tachycardia, **jugular venous distension** with clear lungs, and **pulsus paradoxus** (manifesting as loss of palpable pulse during inspiration) is consistent with **cardiac tamponade**. This is most likely due to the patient's recent viral illness causing viral pericarditis with significant pericardial fluid accumulation. Normally there is a <10 mm Hg decrease in systolic blood pressure during inspiration. **Pulsus paradoxus** refers to an abnormal exaggerated decrease in systolic blood pressure >10 mm Hg on inspiration, a common finding in patients with large pericardial effusions causing tamponade.

Inspiration causes an increase in systemic venous return, resulting in increased right heart volumes. Under normal conditions, this results in expansion of the right ventricle into the pericardial space with little impact on the left side of the heart. However, in conditions that impair expansion into the pericardial space (eg, cardiac tamponade), the increased right ventricular volume that occurs with inspiration leads to bowing of the interventricular septum toward the left ventricle. This leads to a decrease in left ventricular (LV) end-diastolic volume and forward stroke volume, with a resultant decrease in systolic pressure during inspiration.

(Choice A) Acute myocardial infarction typically presents with sudden onset of substernal discomfort, diaphoresis, and/or dyspnea, and can progress to cardiogenic shock (hypotension, tachycardia, jugular venous distension, and pulmonary edema). Patients can have weak or undetectable peripheral pulses; however, pulsus paradoxus is typically not seen.

(Choice B) Acute viral myocarditis can lead to severe LV systolic dysfunction and present with acute congestive heart failure and cardiogenic shock. Patients with severe LV failure can have pulsus alternans - a variation in pulse amplitude with alternate beats. Pulsus paradoxus is typically not seen in such patients.

(Choice D) Pulsus paradoxus can also occur in patients with asthma, chronic obstructive pulmonary disease, and (less frequently) constrictive pericarditis. Constrictive pericarditis is caused by scarring and loss of elasticity of the normal pericardial sac; it takes several weeks to months to develop and only rarely occurs after recurrent episodes of acute pericarditis.

(Choice E) The initial stage of septic shock is a hyperdynamic circulatory state with lowered systemic vascular resistance and increased cardiac output (warm shock). Weak pulses and pulsus paradoxus would not be expected in such patients.

(Choice F) Intentional pneumothorax breath sounds are typically absent on the affected side with hyperresonance to percussion. Other features

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expansion into the pericardial space (eg, cardiac tamponade), the increased right ventricular volume that occurs with inspiration leads to bowing of the interventricular septum toward the left ventricle. This leads to a decrease in left ventricular (LV) end-diastolic volume and forward stroke volume, with a resultant decrease in systolic pressure during inspiration.

(Choice A) Acute myocardial infarction typically presents with sudden onset of substernal discomfort, diaphoresis, and/or dyspnea, and can progress to cardiogenic shock (hypotension, tachycardia, jugular venous distension, and pulmonary edema). Patients can have weak or undetectable peripheral pulses; however, pulsus paradoxus is typically not seen.

(Choice B) Acute viral myocarditis can lead to severe LV systolic dysfunction and present with acute congestive heart failure and cardiogenic shock. Patients with severe LV failure can have pulsus alternans - a variation in pulse amplitude with alternate beats. Pulsus paradoxus is typically not seen in such patients.

(Choice D) Pulsus paradoxus can also occur in patients with asthma, chronic obstructive pulmonary disease, and (less frequently) constrictive pericarditis. Constrictive pericarditis is caused by scarring and loss of elasticity of the normal pericardial sac; it takes several weeks to months to develop and only rarely occurs after recurrent episodes of acute pericarditis.

(Choice E) The initial stage of septic shock is a hyperdynamic circulatory state with lowered systemic vascular resistance and increased cardiac output (warm shock). Weak pulses and pulsus paradoxus would not be expected in such patients.

(Choice F) In tension pneumothorax, breath sounds are typically absent on the affected side with hyperresonance to percussion. Other features of tension pneumothorax include tachypnea, tachycardia, hypotension, distended neck veins, and tracheal deviation to the contralateral side.

Educational objective:

Cardiac tamponade typically presents with hypotension with pulsus paradoxus, elevated jugular venous pressure, and muffled heart sounds (Beck's triad). Pulsus paradoxus refers to an abnormal exaggerated decrease in systolic blood pressure >10 mm Hg on inspiration, and is a common finding in patients with pericardial effusion with cardiac tamponade.

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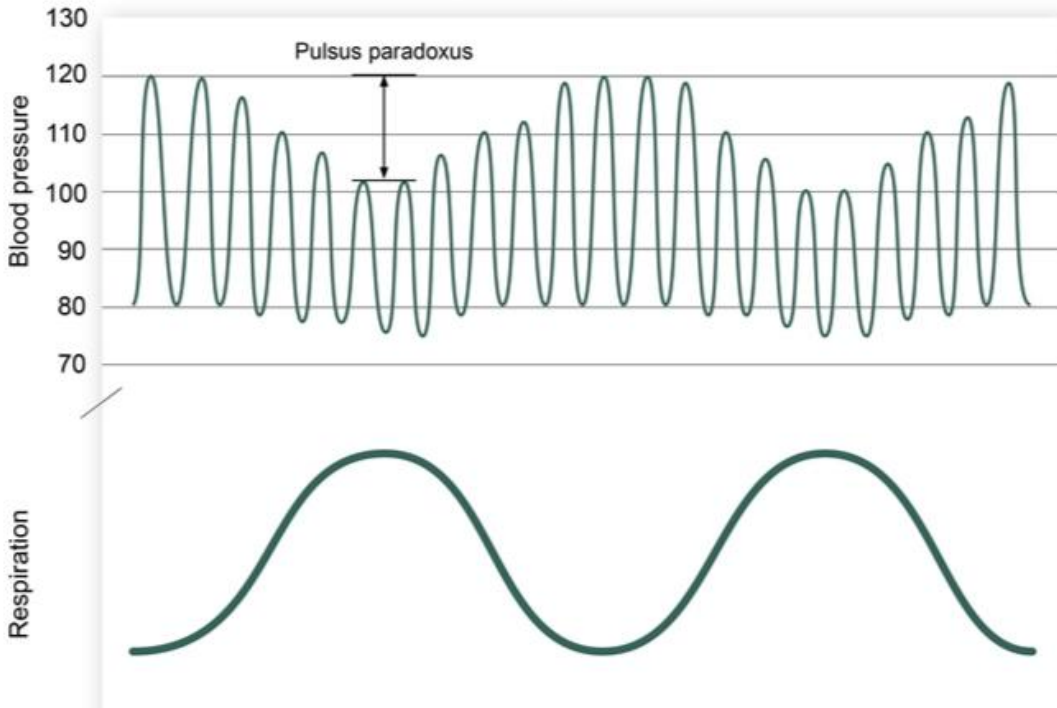
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Text Zoom

Septic shock (3%)

Exhibit Display

Pulsus paradoxus



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undetectable peripheral pulses; however, pulsus paradoxus is typically not seen

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A 12-year-old Caucasian male is found to have a wide, fixed splitting of the second heart sound (S2) on routine physical examination. He denies any symptoms. If present, the congenital heart disease in this patient may require surgical repair to prevent irreversible changes in the:

- ☐ A. Right ventricle
- ☐ B. Right atrium
- ☐ C. Left ventricle
- ☐ D. Left atrium
- ☐ E. Pulmonary vessels
- ☐ F. Coronary vessels

Submit

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A 12-year-old Caucasian male is found to have a wide, fixed splitting of the second heart sound (S2) on routine physical examination. He denies any symptoms. If present, the congenital heart disease in this patient may require surgical repair to prevent irreversible changes in the:

A. Right ventricle [24%]

B. Right atrium [18%]

C. Left ventricle [10%]

D. Left atrium [6%]

E. Pulmonary vessels [39%]

F. Coronary vessels [0%]

Omitted

Correct answer
E

39%

Answered correctly

3 Seconds

Time Spent

02/06/2019

Last Updated

Explanation

Wide, fixed splitting of S2 that does not vary with respiration is a characteristic auscultatory finding of an atrial septal defect (ASD). ASD creates a left-to-right shunt because of the high pressure in the left atrium. The result is increased blood flow through the pulmonary artery. The muscular pulmonary arteries may develop laminated medial hypertrophy that can become so severe over time as to increase the pulmonary vascular resistance above the total systemic vascular resistance. At this point, the original left-to-right intraseptal shunt reverses, and flow becomes right-to-left.

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Explanation

Wide, fixed splitting of S2 that does not vary with respiration is a characteristic auscultatory finding of an atrial septal defect (ASD). ASD creates a left-to-right shunt because of the high pressure in the left atrium. The result is increased blood flow through the pulmonary artery. The muscular pulmonary arteries may develop laminated medial hypertrophy that can become so severe over time as to increase the pulmonary vascular resistance above the total systemic vascular resistance. At this point, the original left-to-right intracardiac shunt reverses, and flow becomes right-to-left. This switch to right-to-left shunting manifests as late-onset cyanosis, with clubbing and polycythemia. Eisenmenger syndrome is the name for reversal of shunt flow through a congenital cardiac defect that occurs as a result of chronic pulmonary hypertension. Over time, the pulmonary vascular sclerosis becomes irreversible and closure of the cardiac septal defect can no longer be hemodynamically tolerated by the right ventricle.

(Choices A and B) Pulmonary hypertension produced by an ASD could result in right ventricular hypertrophy and right atrial enlargement. However, these changes are not necessarily irreversible. If the pulmonary hypertension is corrected, the right heart can revert to a more normal morphology.

(Choices C and D) Left ventricular failure is uncommon in patients with ASD. Left atrial enlargement can be present due to volume overload, but the main changes are in the right side of the heart.

Educational Objective:

Wide, fixed splitting of the second heart sound is a characteristic auscultatory finding in patients with ASD. A hemodynamically significant ASD can produce chronic pulmonary hypertension as a result of left-to-right intracardiac shunting. Eisenmenger syndrome is the late-onset reversal of a left-to-right shunt due to pulmonary vascular sclerosis resulting from chronic pulmonary hypertension. Closure of the ASD may be required to prevent irreversible pulmonary vascular sclerosis and a permanent Eisenmenger syndrome.

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Text Zoom

A 35-year-old man comes to the office with progressive fatigue, dyspnea on exertion, and lower extremity edema over the last 2 weeks. Preceding these symptoms, he had an episode of fever, runny nose, and myalgias that resolved after several days. The patient has no other medical history, takes no medications, and has no significant family history. He occasionally drinks alcohol and does not use tobacco. Blood pressure is 112/74 mm Hg, and pulse is 98/min and regular. Physical examination reveals jugular venous distension, bibasilar crackles on lung auscultation, and 2+ pitting edema involving the lower extremities. Which of the following echocardiographic findings is most likely to be seen in this patient?

A. Concentric left ventricle thickening with abnormal diastolic relaxation

B. Dilated ventricles with abnormal systolic ventricular function

C. High systolic pressure gradient across the aortic valve

D. Regional wall motion abnormality

E. Systolic anterior motion of the mitral valve

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A 35-year-old man comes to the office with progressive fatigue, dyspnea on exertion, and lower extremity edema over the last 2 weeks. Preceding these symptoms, he had an episode of fever, runny nose, and myalgias that resolved after several days. The patient has no other medical history, takes no medications, and has no significant family history. He occasionally drinks alcohol and does not use tobacco. Blood pressure is 112/74 mm Hg, and pulse is 98/min and regular. Physical examination reveals jugular venous distension, bibasilar crackles on lung auscultation, and 2+ pitting edema involving the lower extremities. Which of the following echocardiographic findings is most likely to be seen in this patient?

A. Concentric left ventricle thickening with abnormal diastolic relaxation [8%]

✓

B. Dilated ventricles with abnormal systolic ventricular function [58%]

C. High systolic pressure gradient across the aortic valve [4%]

D. Regional wall motion abnormality [7%]

E. Systolic anterior motion of the mitral valve [20%]

Omitted

Correct answer
B

58%

Answered correctly

3 Seconds

Time Spent

11/05/2018

Last Updated

Explanation

Causes of nonvalvular heart failure				
	Ventricular wall thickness	Ventricular cavity size	Contractile function	Diastolic function

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Causes of nonvalvular heart failure

	Ventricular wall thickness	Ventricular cavity size	Contractile function	Diastolic function
Ischemic heart disease	↓ or normal	↑	↓	↓ or normal
Dilated cardiomyopathy*				
Hypertensive heart disease	↑	↓	normal	↓
Hypertrophic cardiomyopathy*	↑ septal thickening	↓	normal	↓
Restrictive cardiomyopathy*	normal (but stiff)	↓ or normal	normal	↓

*Cardiomyopathies are characterized by primary myocardial dysfunction (eg, viral myocarditis, genetic disorder, infiltrative disease). Myocardial disease caused by secondary factors (eg, hypertension, coronary artery disease, valvular dysfunction) is not classified as a cardiomyopathy.

This patient's presentation with dyspnea, lower extremity edema, jugular venous distension, and pulmonary crackles is consistent with **decompensated heart failure**. In a young patient who develops heart failure following a symptomatic viral prodrome (eg, fever, sinus congestion, myalgias), **dilated cardiomyopathy** (DCM) due to **viral myocarditis** should be suspected. A number of viruses have been implicated in causing DCM including coxsackievirus, adenovirus, and influenza virus. It is believed that an inadequate immune response to the viral infection allows virus to infect and persist inside cardiomyocytes, resulting in damage due to a direct cytotoxic effect or a destructive autoimmune reaction.

Depletion of cardiomyocytes with subsequent fibrosis causes weakened myocardial contractility (**systolic dysfunction**), volume overload, and ventricular dilation (**eccentric hypertrophy**) as surviving myocytes respond to increased wall stress. Other causes of DCM include genetic disease, pregnancy, infiltrative disease (eg, late manifestation of amyloidosis or hemochromatosis), and drugs/toxins (eg, anthracyclines, chronic alcohol abuse).

(Choice A) Concentric left ventricular (LV) **hypertrophy** with thickened walls and abnormal diastolic relaxation is characteristic of hypertensive



Item 8 of 40

Question Id: 75



Depletion of cardiomyocytes with subsequent fibrosis causes weakened myocardial contractility (**systolic dysfunction**), volume overload, and ventricular dilation (**eccentric hypertrophy**) as surviving myocytes respond to increased wall stress. Other causes of DCM include genetic disease, pregnancy, infiltrative disease (eg, late manifestation of amyloidosis or hemochromatosis), and drugs/toxins (eg, anthracyclines, chronic alcohol abuse).

(Choice A) Concentric left ventricular (LV) **hypertrophy** with thickened walls and abnormal diastolic relaxation is characteristic of hypertensive heart disease due to long-standing uncontrolled hypertension. Initially, the LV cavity is reduced in size and there is normal systolic function; however, over time, high afterload pressures can cause progressive systolic dysfunction, LV cavity enlargement, and heart failure.

(Choice C) High systolic pressure gradient across the aortic valve is present in aortic stenosis; concentric LV hypertrophy can occur due to LV pressure overload. Aortic stenosis can develop in relatively young patients due to calcification of a bicuspid aortic valve; however, a crescendo-decrescendo systolic murmur is expected on examination.

(Choice D) Regional wall motion abnormality is suggestive of ischemic heart disease (eg, myocardial infarction). Contractile function is impaired in the damaged portion of myocardium, resulting in systolic dysfunction with LV volume overload, eccentric hypertrophy, and LV cavity enlargement. Ischemic heart disease is unlikely in this young patient without significant risk factors for cardiovascular disease.

(Choice E) In patients with **hypertrophic cardiomyopathy**, systolic anterior motion of the mitral valve may worsen LV outflow tract obstruction. Clinical features of hypertrophic cardiomyopathy include exercise-induced syncope (due to outflow obstruction) and sudden cardiac death (due to ventricular arrhythmia) in young athletes.

Educational objective:

Dilated cardiomyopathy results from direct damage to cardiomyocytes leading to myocardial contractile dysfunction (systolic dysfunction), volume overload, and ventricular dilation. Viral myocarditis is a common cause of dilated cardiomyopathy and should be suspected in young patients who develop heart failure following a symptomatic viral prodrome.

References

- [Cardiomyopathies: Evolution of pathogenesis concepts and potential for new therapies.](#)

Block Time Remaining: 00:01:20

TUTOR





The following vignette applies to the next 2 items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 34-year-old Russian immigrant comes to the physician with a 2-month history of exertional dyspnea and progressive lower extremity swelling. He also reports abdominal distension and decreased appetite. The patient has gained 4.5 kg (10 lbs) over the last 2 months. Past medical history is significant for hypertension and recurrent lung infections requiring prolonged antibiotic therapy. He has no history of coronary artery disease. The patient has smoked a pack of cigarettes daily for the past 15 years. He undergoes noninvasive cardiac testing followed by cardiac catheterization. A jugular venous pressure tracing is shown on the slide below.

Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

- ☐ A. Atrial contraction
- ☐ B. Carotid pulse upstroke
- ☐ C. Isovolumetric ventricular contraction
- ☐ D. Tricuspid valve closure
- ☐ E. Tricuspid valve opening

Submit

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Item 9 of 40

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Lab Values

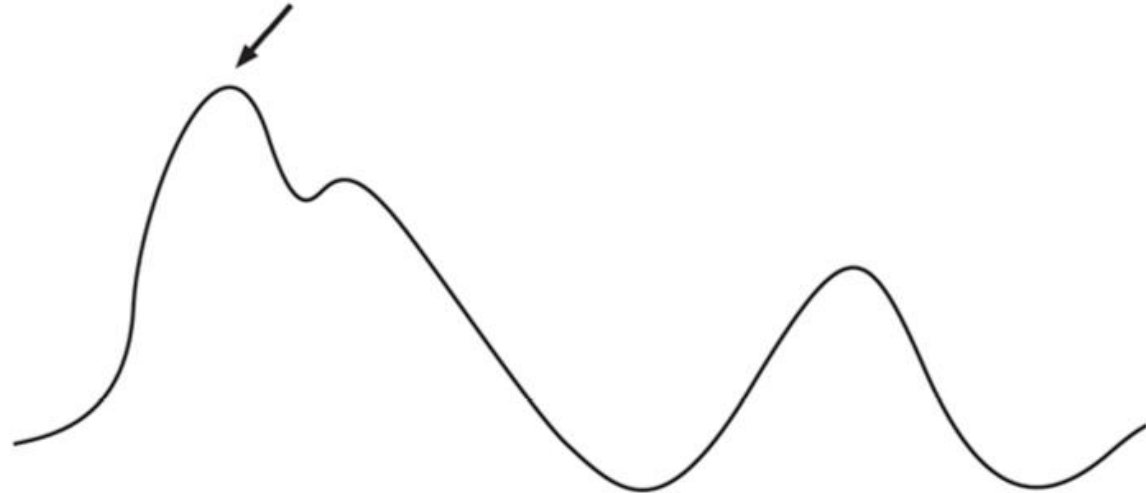
Notes

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Exhibit Display



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Block Time Remaining: 00:19:52

TUTOR

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2/9/2019



The following vignette applies to the next 2 items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 34-year-old Russian immigrant comes to the physician with a 2-month history of exertional dyspnea and progressive lower extremity swelling. He also reports abdominal distension and decreased appetite. The patient has gained 4.5 kg (10 lbs) over the last 2 months. Past medical history is significant for hypertension and recurrent lung infections requiring prolonged antibiotic therapy. He has no history of coronary artery disease. The patient has smoked a pack of cigarettes daily for the past 15 years. He undergoes noninvasive cardiac testing followed by cardiac catheterization. A jugular venous pressure tracing is shown on the slide below.

Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

- ☐ A. Atrial contraction
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- ☐ D. Tricuspid valve closure
- ☐ E. Tricuspid valve opening

Submit

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TUTOR



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The following vignette applies to the next 2 items. The items in the set must be answered in sequential order. Once you click **Proceed to Next Item**, you will not be able to add or change an answer.

A 34-year-old Russian immigrant comes to the physician with a 2-month history of exertional dyspnea and progressive lower extremity swelling. He also reports abdominal distension and decreased appetite. The patient has gained 4.5 kg (10 lbs) over the last 2 months. Past medical history is significant for hypertension and recurrent lung infections requiring prolonged antibiotic therapy. He has no history of coronary artery disease. The patient has smoked a pack of cigarettes daily for the past 15 years. He undergoes noninvasive cardiac testing followed by cardiac catheterization. A jugular venous pressure tracing is shown on the slide below.

Item 1 of 2

The waveform indicated by the arrow most likely corresponds to which of the following?

✓

☒

A. Atrial contraction [72%]

☐

B. Carotid pulse upstroke [4%]

☐

C. Isovolumetric ventricular contraction [6%]

☐

D. Tricuspid valve closure [8%]

☐

E. Tricuspid valve opening [7%]

Omitted

Correct answer
A

72%

Answered correctly

3 Seconds

Time Spent

12/26/2018

Last Updated

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C. Isovolumetric ventricular contraction [6%]

D. Tricuspid valve closure [8%]

E. Tricuspid valve opening [7%]

Omitted

Correct answer
A

72%
Answered correctly

3 Seconds
Time Spent

12/26/2018
Last Updated

Explanation

A normal jugular venous pulse wave tracing has 3 positive waves (a, c, and v) and 2 negative waves (x and y descent). The **a wave** (indicated by the arrow on the diagram above) is generated by **atrial contraction**, the c wave is caused by bulging of the tricuspid valve into the right atrium in early systole, and the v wave reflects the passive increase in pressure and volume of the right atrium as it fills in late systole and early diastole. The troughs of the jugular venous pulse are known as the x-descent and the y-descent. The x-descent is primarily caused by relaxation of the right atrium. The y-descent represents the abrupt decrease in right atrial pressure during early diastole after the tricuspid valve opens and the right ventricle begins to passively fill.

Educational objective:

On jugular venous pressure tracings, the first peak is the a wave, which is generated by atrial contraction. This is notably absent in patients with atrial fibrillation.

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Item 2 of 2

The patient is admitted to the hospital for further workup and treatment. After initial evaluation, a CT scan of the chest is obtained, an image of which is shown on the slide below.

Which of the following is the most likely diagnosis?

☐ A. Aortic dissection

☐ B. Constrictive pericarditis

☐ C. Hypertrophic cardiomyopathy

☐ D. Ischemic heart disease

☐ E. Viral myocarditis

Submit

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Item 10 of 40

Question Id: 2071

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Lab Values

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Text Zoom

Item 2 of 2

The patient is admitted to the hospital for further workup and treatment. After initial evaluation, a CT scan of the chest is obtained, an image of which is shown on the slide below.

Which of the following is the most likely diagnosis?

☐ A. Aortic dissection

☐ B. Constrictive pericarditis

☐ C. Hypertrophic cardiomyopathy

☐ D. Ischemic heart disease

☐ E. Viral myocarditis

Submit

Block Time Remaining: 00:01:24

TUTOR

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System Tray



Item 2 of 2

The patient is admitted to the hospital for further workup and treatment. After initial evaluation, a CT scan of the chest is obtained, an image of which is shown on the slide below.

Which of the following is the most likely diagnosis?

- ☐ A. Aortic dissection [4%]
- ☒ B. Constrictive pericarditis [59%]
- ☐ C. Hypertrophic cardiomyopathy [27%]
- ☐ D. Ischemic heart disease [2%]
- ☐ E. Viral myocarditis [6%]

Omitted

Correct answer
B59%
Answered correctly3 Seconds
Time Spent12/26/2018
Last Updated

Explanation

The above CT image shows thickening and calcification of the pericardium suggestive of constrictive pericarditis. Although the pericardium is normally only 1-2 mm thick, it can range in thickness from 4-20 mm in patients with constrictive pericarditis. Patients with this condition typically have slowly progressive dyspnea, chronic edema, and ascites. A rapidly descent that becomes both deeper and steeper during inspiration is

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Item 10 of 40

Question Id: 2071

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Lab Values

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Text Zoom

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Explanation

The above CT image shows thickening and calcification of the pericardium suggestive of constrictive pericarditis. Although the pericardium is normally only 1-2 mm thick, it can range in thickness from 4-20 mm in patients with constrictive pericarditis. Patients with this condition typically have slowly progressive dyspnea, chronic edema, and ascites. A rapid y-descent that becomes both deeper and steeper during inspiration is often observed on the jugular venous pressure tracing. Possible causes include radiation therapy to the chest, cardiac surgery, and tuberculosis (which is possibly the cause in this patient who is an immigrant from an endemic region).

(Choice A) An **aortic dissection** causes sudden chest pain that radiates to the back. Chest radiographs often show widening of the mediastinum, and a double aortic lumen would be seen on a CT scan of the chest.

(Choice C) Hypertrophic cardiomyopathy also presents with dyspnea and changes in the jugular venous pressure, specifically a prominent a wave. CT may show thickening of the interventricular septum but would not show thickening or calcification of the pericardium.

(Choice D) Ischemic heart disease classically causes pressure like substernal chest pain that radiates to the left shoulder, and calcifications in the coronary arteries and aorta are often seen on CT.

(Choice E) Patients with viral myocarditis have a variable clinical presentation ranging from asymptomatic to severe chest pain and cardiogenic shock. Pericardial thickening is not a feature of this condition.

Educational objective:

Calcification and thickening of the pericardium are common features of constrictive pericarditis on CT. Clinical findings include slowly progressive dyspnea, peripheral edema, and ascites.

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A 24-year-old man with a documented harsh systolic ejection murmur on cardiac auscultation dies suddenly. His father and uncle died suddenly in their 30s. A gross autopsy specimen of his heart is shown below.

Which of the following would have most likely increased the intensity of this patient's murmur?

- ☐ A. Passive leg raising
- ☐ B. Phenylephrine infusion
- ☐ C. Squatting
- ☐ D. Sudden standing
- ☐ E. Sustained hand grip

Submit

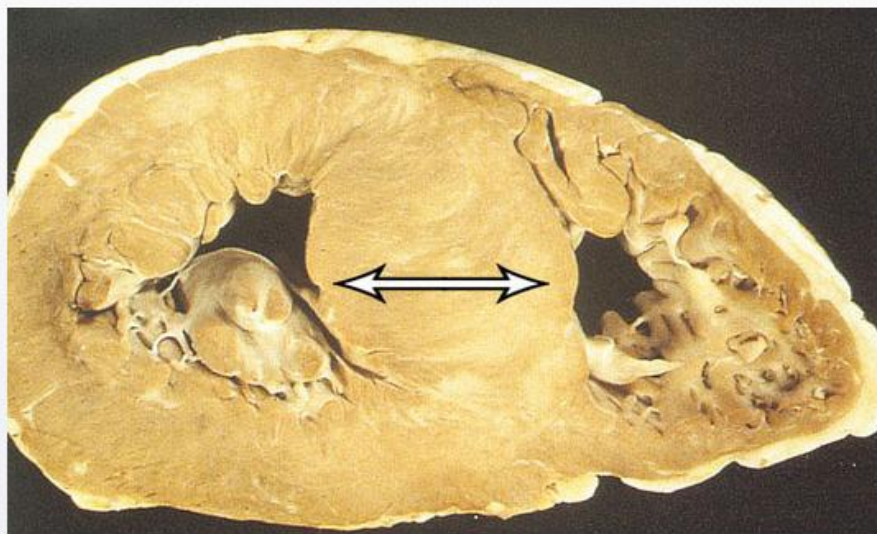
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TUTOR





A 24-year-old man with a documented harsh systolic ejection murmur on cardiac auscultation dies suddenly. His father and uncle died suddenly in their 30s. A gross autopsy specimen of his heart is shown below.



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Which of the following would have most likely increased the intensity of this patient's murmur?

- ☐ A. Passive leg raising [5%]
- ☐ B. Phenylephrine infusion [3%]
- ☐ C. Squatting [20%]

Block Time Remaining: 00:19:52

TUTOR





A 24-year-old man with a documented harsh systolic ejection murmur on cardiac auscultation dies suddenly. His father and uncle died suddenly in their 30s. A gross autopsy specimen of his heart is shown below.

Which of the following would have most likely increased the intensity of this patient's murmur?

- ☐ A. Passive leg raising
- ☐ B. Phenylephrine infusion
- ☐ C. Squatting
- ☐ D. Sudden standing
- ☐ E. Sustained hand grip

Submit

Block Time Remaining: 00:01:27

TUTOR





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Which of the following would have most likely increased the intensity of this patient's murmur?

- ☐ A. Passive leg raising [5%]
- ☐ B. Phenylephrine infusion [3%]
- ☐ C. Squatting [20%]
- ☒ D. Sudden standing [53%]
- ☐ E. Sustained hand grip [16%]

Omitted

Correct answer
D



53%
Answered correctly



2 Seconds
Time Spent



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Explanation

This patient's heart shows asymmetric ventricular septal hypertrophy, a finding characteristic of **hypertrophic cardiomyopathy (HCM)**. Dynamic **left ventricular outflow tract (LVOT) obstruction** is seen in about 25% of patients with HCM and causes a harsh crescendo-decrescendo systolic ejection-type murmur that is best heard along the lower left sternal border and apex.

The degree of LVOT obstruction (and intensity of the murmur) **varies** based on **LV end-diastolic volume**. **Mechanisms** that decrease preload

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Omitted

Correct answer
D

53%

Answered correctly

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Last Updated

Explanation

This patient's heart shows asymmetric ventricular septal hypertrophy, a finding characteristic of **hypertrophic cardiomyopathy** (HCM). Dynamic **left ventricular outflow tract (LVOT) obstruction** is seen in about 25% of patients with HCM and causes a harsh crescendo-decrescendo systolic ejection-type murmur that is best heard along the lower left sternal border and apex.

The degree of LVOT obstruction (and intensity of the murmur) **varies** based on **LV end-diastolic volume**. **Mechanisms** that decrease preload (ie, venous return) or afterload reduce LV chamber size, which decreases the separation between the mitral valve and interventricular septum, increasing obstruction. Sudden standing (from a sitting or supine position), Valsalva (straining phase), or nitroglycerin administration decreases preload and will result in **increased murmur intensity**. In contrast, squatting (from a standing position), sustained hand grip, or passive leg raise will increase preload and/or afterload, thereby decreasing murmur intensity (**Choices A, C, and E**).

(Choice B) Phenylephrine (a selective α_1 -agonist) infusion would increase systemic vascular resistance (afterload), thereby increasing LV end-diastolic volume and reducing the gradient across the LVOT. This would result in decreased murmur intensity.

Educational objective:

Hypertrophic cardiomyopathy is characterized by asymmetric ventricular septal hypertrophy and variable dynamic left ventricular outflow tract obstruction. Maneuvers that increase preload or afterload will decrease murmur intensity by increasing left ventricular end-diastolic volume and lessening the outflow tract obstruction.

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Omitted

Correct answer

D

Explanation

This patient's heart shows asymmetric septal hypertrophy and a **left ventricular outflow tract (LVOT) obstruction**, which produces a systolic ejection-type murmur that is loudest at the base of the heart.

The degree of LVOT obstruction is determined by the degree of obstruction (ie, venous return) or afterload. Sudden increases in preload and will result in **increased obstruction**. Sudden increases in afterload will increase preload and/or afterload.

(Choice B) Phenylephrine (a vasoconstrictor) increases diastolic volume and reducing the degree of obstruction.

Educational objective:

Hypertrophic cardiomyopathy is a genetic condition characterized by LVOT obstruction. Maneuvers that increase preload and/or afterload lessen the outflow tract obstruction.

Exhibit Display

Effect of maneuvers on hypertrophic cardiomyopathy

	Physiologic effect	Left ventricular blood volume	Murmur intensity
Valsalva (strain phase)	↓ Preload	↓	↑
Abrupt standing			
Nitroglycerin administration			
Sustained hand grip	↑ Afterload	↑	↓
Squatting	↑ Afterload & preload		
Passive leg raise	↑ Preload		

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Text Zoom

A 65-year-old man comes to the office due to exertional dyspnea and easy fatigability. He has no other medical problems. The patient has not seen a physician in the last 10 years. He takes no medications but smokes a pack of cigarettes per day and drinks approximately 7-10 alcoholic beverages a week. His blood pressure is 170/90 mm Hg and pulse is 80/min. Physical examination reveals bilateral lung crackles. Which of the following sets of diagnostic findings would be most consistent with isolated diastolic heart failure?

	Left ventricular end-diastolic pressure	Left ventricular end-diastolic volume	Left ventricular ejection fraction
☐ A.	Increased	Increased	Decreased
☐ B.	Normal	Increased	Decreased
☐ C.	Normal	Normal	Decreased
☐ D.	Increased	Normal	Decreased
☐ E.	Increased	Normal	Normal
☐ F.	Normal	Increased	Normal

Submit

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Item 12 of 40

Question Id: 186

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Text Zoom

A 65-year-old man comes to the office due to exertional dyspnea and easy fatigability. He has no other medical problems. The patient has not seen a physician in the last 10 years. He takes no medications but smokes a pack of cigarettes per day and drinks approximately 7-10 alcoholic beverages a week. His blood pressure is 170/90 mm Hg and pulse is 80/min. Physical examination reveals bilateral lung crackles. Which of the following sets of diagnostic findings would be most consistent with isolated diastolic heart failure?

	Left ventricular end-diastolic pressure	Left ventricular end-diastolic volume	Left ventricular ejection fraction
☐ A.	Increased [23%]	Increased	Decreased
☐ B.	Normal [9%]	Increased	Decreased
☐ C.	Normal [5%]	Normal	Decreased
☐ D.	Increased [13%]	Normal	Decreased
☒ E.	Increased [40%]	Normal	Normal
☐ F.	Normal [8%]	Increased	Normal

Omitted

40%

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Omitted

Correct answer

E

40%

Answered correctly

2 Seconds

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Explanation

Diastolic heart failure (DHF) is a common cause of acute decompensated HF. It is characterized by **normal left ventricular ejection fraction** (LVEF, >50%) and **end-diastolic volume** in the setting of increased LV filling pressures (**Choice D**). Diastolic dysfunction can be due to conditions that decrease LV compliance, such as impaired myocardial relaxation (eg, from ischemia) or increased intrinsic ventricular wall stiffness (eg, from amyloid deposition). This patient most likely has long-standing hypertension leading to LV wall hypertrophy, which can impair myocardial relaxation and increase intrinsic wall stiffness.

LV diastolic pressure is determined by the blood volume in the LV cavity and compliance of the left ventricle. Conditions that reduce ventricular compliance lead to increased LV end-diastolic pressures (LVEDP) at the same LVED volumes. This causes an upward shift in the **pressure-volume curve** (point a → b).

As diastolic dysfunction worsens, LVEDP continues to rise as the heart attempts to maintain a near-normal stroke volume and cardiac output. Decompensation occurs when the increased LVEDP (transmitted to the left atrium and pulmonary veins) causes pulmonary edema and dyspnea.

(Choice A) In contrast to diastolic HF, systolic HF is caused by a primary decrease in myocardial contractility rather than ventricular compliance. As a result, **systolic HF** is characterized by a reduced LVEF (<50%), along with progressive chamber dilation with increased LV volume and elevated LVEDP.

(Choices B, C, and F) LVEDP is elevated (not normal) in patients with diastolic HF. This increase in LVEDP is transmitted to the left atrium, pulmonary veins, and capillaries; the result is pulmonary congestion, dyspnea, and exercise intolerance.

Educational objective:

Diastolic heart failure is caused by decreased ventricular compliance and is characterized by normal left ventricular (LV) ejection fraction, normal

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LV diastolic pressure is determined by the blood volume in the LV cavity and compliance of the left ventricle. Conditions that reduce ventricular compliance lead to increased LV end-diastolic pressures (LVEDP) at the same LVED volumes. This causes an upward shift in the **pressure-volume curve** (point a → b).

As diastolic dysfunction worsens, LVEDP continues to rise as the heart attempts to maintain a near-normal stroke volume and cardiac output. Decompensation occurs when the increased LVEDP (transmitted to the left atrium and pulmonary veins) causes pulmonary edema and dyspnea.

(Choice A) In contrast to diastolic HF, systolic HF is caused by a primary decrease in myocardial contractility rather than ventricular compliance. As a result, **systolic HF** is characterized by a reduced LVEF (<50%), along with progressive chamber dilation with increased LV volume and elevated LVEDP.

(Choices B, C, and F) LVEDP is elevated (not normal) in patients with diastolic HF. This increase in LVEDP is transmitted to the left atrium, pulmonary veins, and capillaries; the result is pulmonary congestion, dyspnea, and exercise intolerance.

Educational objective:

Diastolic heart failure is caused by decreased ventricular compliance and is characterized by normal left ventricular (LV) ejection fraction, normal LV end-diastolic volume, and elevated LV filling pressures.

References

- Heart failure with normal ejection fraction.
- Management of Chronic Heart Failure in Primary Care: What Evidence do we have for Heart Failure with Preserved Systolic Function?

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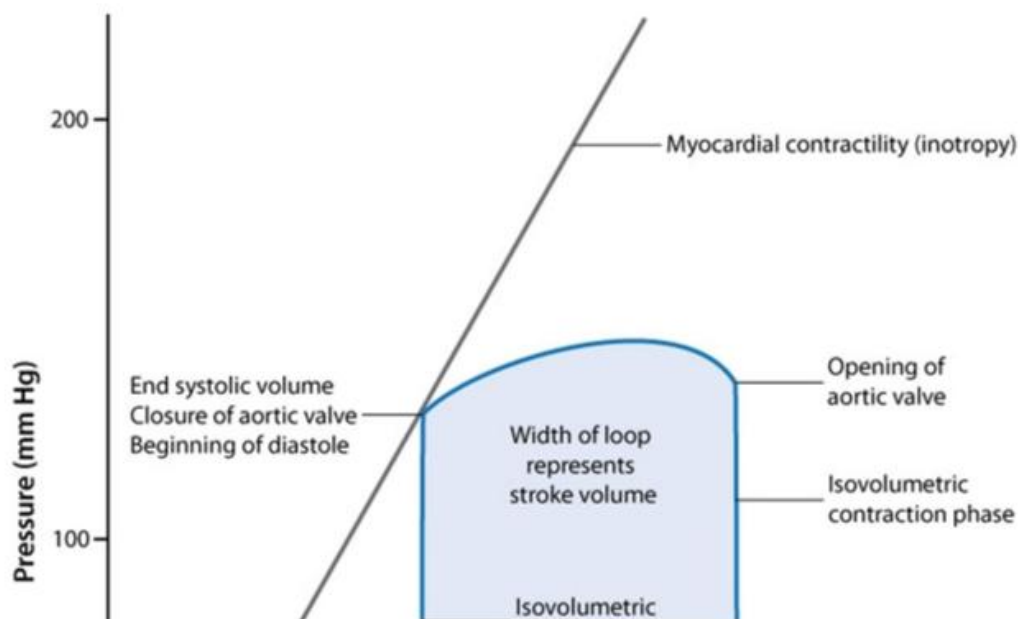
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Exhibit Display

Normal left ventricular function, pressure-volume curve



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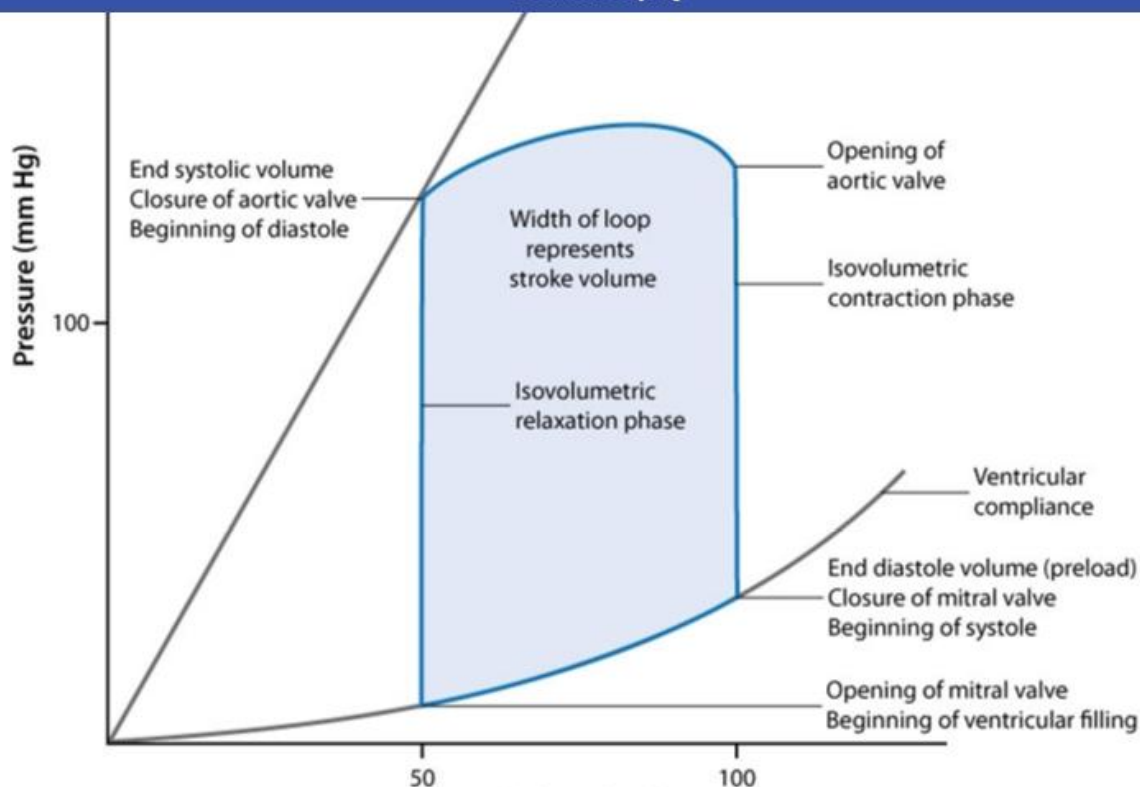
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(eg. from amyloid deposition). This patient most likely has long-standing hypertension leading to LV wall hypertrophy which can impair myocardial

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Question Id: 952



Atherosclerotic lesions of the coronary arteries can limit blood flow to the myocardial regions supplied by the affected vessels. Certain medications can cause a redistribution of blood flow away from the ischemic areas, exacerbating existing myocardial ischemia. Which of the following drug effects is most likely to produce this phenomenon?

- ☐ A. Coronary arteriolar dilation
- ☐ B. Decreased myocardial contractility
- ☐ C. Epicardial vessel dilation
- ☐ D. Negative chronotropic effect
- ☐ E. Systemic venous dilation

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TUTOR





Atherosclerotic lesions of the coronary arteries can limit blood flow to the myocardial regions supplied by the affected vessels. Certain medications can cause a redistribution of blood flow away from the ischemic areas, exacerbating existing myocardial ischemia. Which of the following drug effects is most likely to produce this phenomenon?

- ☒ A. Coronary arteriolar dilation [59%]
☐ B. Decreased myocardial contractility [5%]
☐ C. Epicardial vessel dilation [11%]
☐ D. Negative chronotropic effect [4%]
☐ E. Systemic venous dilation [18%]

Omitted

Correct answer
A59%
Answered correctly3 Seconds
Time Spent12/03/2018
Last Updated

Explanation

Hemodynamically significant atherosclerotic lesions (>50% occlusion) reduce the amount of blood supplied by a coronary vessel to the corresponding myocardial territory. In response, the myocardium releases locally acting, endogenous substances (eg, adenosine, nitric oxide) that cause **dilation of coronary arterioles**, leading to a downstream reduction in pressure and recruitment of additional blood flow to that region. Arterioles that supply areas of significantly **ischemic myocardium** can become **maximally dilated** at rest, limiting their capacity to further

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corresponding myocardial territory. In response, the myocardium releases locally acting, endogenous substances (eg, adenosine, nitric oxide) that cause **dilation of coronary arterioles**, leading to a downstream reduction in pressure and recruitment of additional blood flow to that region. Arterioles that supply areas of significantly **ischemic myocardium** can become **maximally dilated** at rest, limiting their capacity to further increase blood flow.

Pharmacologic stress agents (eg, adenosine, dipyridamole) are used during myocardial perfusion imaging to simulate the generalized coronary arteriole dilation caused by exercise to assist in identifying areas of ischemic myocardium. Administration of these drugs will result in **no change in blood flow** to ischemic myocardium and a significant increase in blood flow to nonischemic myocardium; the amplified relative difference in blood flow allows for easy detection of ischemic myocardial areas.

When recruitment of additional upstream blood flow is limited (eg, due to a proximal atherosclerotic lesion), these drugs can lead to an absolute **decrease in blood flow to ischemic myocardium**, as most of the limited blood supply is redirected toward the newly vasodilated areas of nonischemic myocardium. This redistribution of blood flow is referred to as **coronary steal**, and most commonly occurs in individuals with extensive coronary artery disease.

(Choices B, D, and E) Decreased myocardial contractility, negative chronotropy, and systemic venous dilation (leading to reduced preload) all decrease myocardial oxygen demand through a reduction in cardiac work. These mechanisms act to improve myocardial ischemia globally and will not result in a redistribution of blood flow away from areas of ischemic myocardium.

(Choice C) The epicardial vessels are the large coronary arteries of the heart (eg, right coronary, left circumflex). Although dilation of these large arteries plays a role in increasing myocardial blood flow, coronary blood flow regulation is mostly mediated by the arterioles. Dilation of large coronary arteries would improve global cardiac blood flow and would not trigger coronary steal.

Educational objective:

Increases in resting blood flow to ischemic myocardium are primarily mediated by locally-acting substances (eg, adenosine, nitric oxide) that trigger coronary arteriolar vasodilation. Pharmacologic arteriolar vasodilators (eg, adenosine, dipyridamole) mimic the vasodilation that occurs with exercise and may cause redistribution of blood flow from ischemic to nonischemic areas of myocardium, so-called coronary steal.

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corresponding myocardial territory. In response, the myocardium releases locally acting, endogenous substances (eg, adenosine, nitric oxide) that

Exhibit Display

At rest

With adenosine

Flow-limiting stenosis

Arteries

Arterioles

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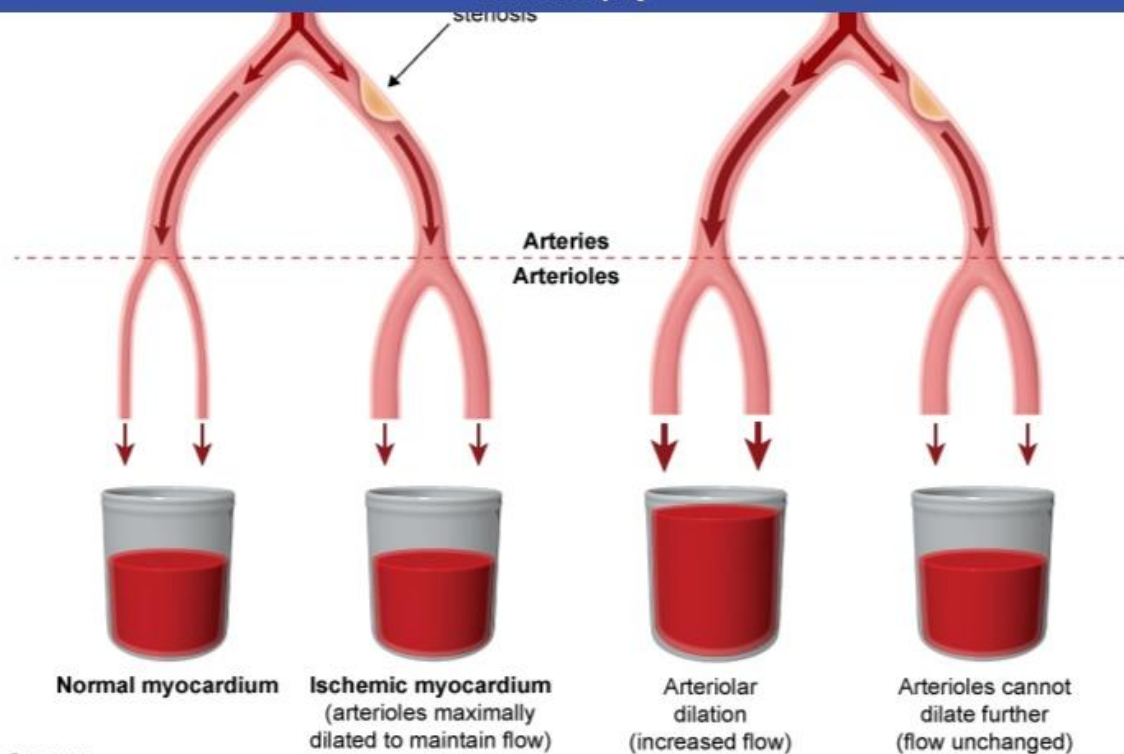
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corresponding myocardial territory. In response, the myocardium releases locally acting, endogenous substances (eg, adenosine, nitric oxide) that

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Text Zoom

A 72-year-old man is hospitalized due to fatigue, orthopnea, and exertional dyspnea. The patient takes no medications and has not seen a physician in many years. He is a lifelong nonsmoker. His blood pressure is 110/60 mm Hg and pulse is 64/min. Examination reveals bibasilar crackles, elevated jugular venous pressure, and bilateral lower extremity pitting edema. His left ventricular end-diastolic pressure-volume is shown in the graph below (purple curve).

Which of the following is the most likely cause of this patient's condition?

A. Alcoholic cardiomyopathy

B. High-dose doxorubicin therapy

C. Selenium deficiency

D. Transthyretin deposition

E. Viral myocarditis

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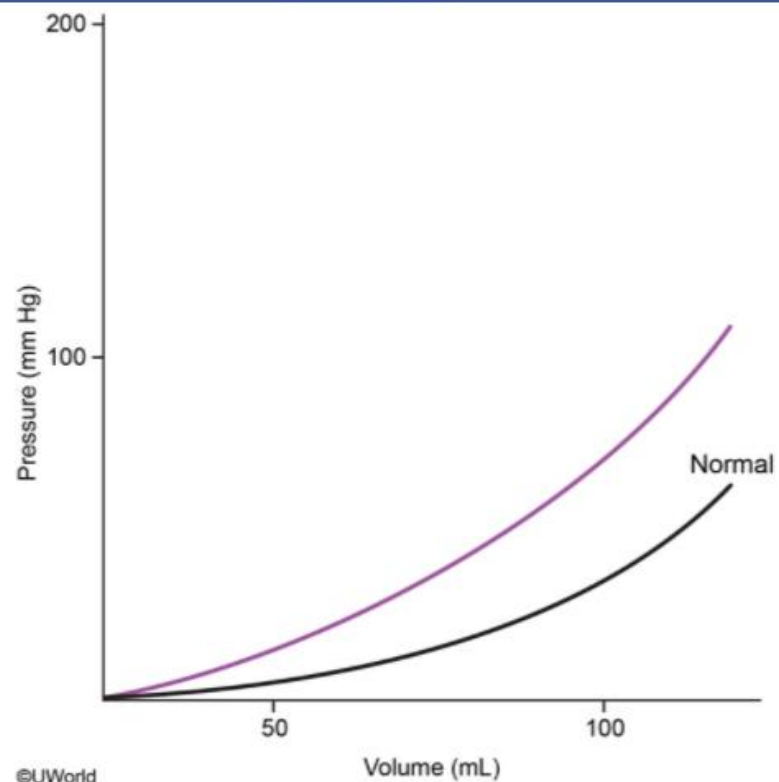


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Text Zoom

A 72-year-old man is hospitalized due to fatigue, orthopnea, and exertional dyspnea. The patient takes no medications and has not seen a physician in many years. He is a lifelong nonsmoker. His blood pressure is 110/60 mm Hg and pulse is 64/min. Examination reveals bibasilar crackles, elevated jugular venous pressure, and bilateral lower extremity pitting edema. His left ventricular end-diastolic pressure-volume is shown in the graph below (purple curve).

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B. High-dose doxorubicin therapy

C. Selenium deficiency

D. Transthyretin deposition

E. Viral myocarditis

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TUTOR

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Windows Taskbar

System Tray



A 72-year-old man is hospitalized due to fatigue, orthopnea, and exertional dyspnea. The patient takes no medications and has not seen a physician in many years. He is a lifelong nonsmoker. His blood pressure is 110/60 mm Hg and pulse is 64/min. Examination reveals bibasilar crackles, elevated jugular venous pressure, and bilateral lower extremity pitting edema. His left ventricular end-diastolic pressure-volume is shown in the graph below (purple curve).

Which of the following is the most likely cause of this patient's condition?

- ☐ A. Alcoholic cardiomyopathy [21%]
- ☐ B. High-dose doxorubicin therapy [11%]
- ☐ C. Selenium deficiency [3%]
- ☒ D. Transthyretin deposition [48%]
- ☐ E. Viral myocarditis [14%]

Omitted

Correct answer
D

48%
Answered correctly

4 Seconds
Time Spent

09/22/2018
Last Updated

Explanation

This presentation (exertional dyspnea, orthopnea, bibasilar crackles, elevated jugular venous pressure, and lower extremity edema) is consistent with **decompensated congestive heart failure** (CHF). The left ventricular (LV) diastolic pressure-volume curve (purple curve in graph above)

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Explanation

This presentation (exertional dyspnea, orthopnea, bibasilar crackles, elevated jugular venous pressure, and lower extremity edema) is consistent with **decompensated congestive heart failure** (CHF). The left ventricular (LV) diastolic pressure-volume curve (purple curve in graph above) shows **reduced LV compliance**, indicating that **diastolic dysfunction** is the main contributing factor of the patient's CHF.

LV diastolic pressure is determined by the blood volume in the LV cavity and compliance of the left ventricle. Conditions that decrease compliance lead to increased LV end-diastolic (LVED) pressures at the same LVED volumes. This causes a shift in the pressure-volume curve that goes upward and to the left. Higher LV filling pressures are transmitted back to pulmonary vasculature, leading to pulmonary edema. Impaired right ventricular filling or neurohormonal activation from low cardiac output is responsible for the peripheral edema and elevated jugular venous pressure.

Restrictive cardiomyopathy (idiopathic or infiltrative disorders [eg, amyloidosis, sarcoidosis, hemochromatosis]) is an important cause of diastolic heart failure, also known as heart failure with preserved ejection fraction. **Transthyretin** (TTR), a protein tetramer produced in the liver, acts as a carrier of thyroxine and retinol. Mutations in the TTR gene can increase the tendency of TTR to misfold, producing an **amyloid protein** that infiltrates the myocardium (**infiltrative cardiomyopathy**).

(Choices A, B, C, and E) Alcoholic cardiomyopathy, doxorubicin therapy, selenium deficiency, and viral myocarditis can all lead to dilated cardiomyopathy with LV systolic dysfunction. Systolic heart failure is typically characterized by increased LV volume and shifting of the pressure-volume curve to the right due to thinning of the ventricular wall (increased compliance).

Educational objective:

Diastolic heart failure (DHF) is caused by decreased ventricular compliance and is characterized by normal left ventricular (LV) ejection fraction, normal LV end-diastolic volume, and elevated LV filling pressures. Hypertension, obesity, and infiltrative disorders (eg, transthyretin-related amyloidosis, sarcoidosis) are important causes of DHF.

References

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System Tray



A 67-year-old male presents to your office with a history of progressive dyspnea. He cannot tolerate moderate exertion and sleeps in a half-sitting position due to orthopnea. He has also noticed some swelling of his ankles. He does not smoke or consume alcohol. His past medical history is significant for hypercholesterolemia and recurrent chest pain. Which of the following do you expect to be increased in this patient?

- ☐ A. Cardiac output
- ☐ B. Renal perfusion
- ☐ C. Arteriolar resistance
- ☐ D. Mixed venous oxygen content
- ☐ E. Lung compliance

Submit

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TUTOR





A 67-year-old male presents to your office with a history of progressive dyspnea. He cannot tolerate moderate exertion and sleeps in a half-sitting position due to orthopnea. He has also noticed some swelling of his ankles. He does not smoke or consume alcohol. His past medical history is significant for hypercholesterolemia and recurrent chest pain. Which of the following do you expect to be increased in this patient?

- ☐ A. Cardiac output [4%]
- ☐ B. Renal perfusion [3%]
- ☒ C. Arteriolar resistance [72%]
- ☐ D. Mixed venous oxygen content [10%]
- ☐ E. Lung compliance [8%]

Omitted

Correct answer
C72%
Answered correctly5 Seconds
Time Spent02/06/2019
Last Updated

Explanation

The symptoms described in this clinical scenario are strongly suggestive of congestive heart failure (CHF). Left-sided CHF frequently presents with dyspnea on exertion, orthopnea (dyspnea while lying flat) and paroxysmal nocturnal dyspnea (waking from sleep gasping for air). Symptoms of right-sided CHF include lower extremity edema and hepatomegaly. Most commonly combined left- and right-ventricular failure is observed owing to the fact that the most common cause of right heart failure is left heart failure.

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The symptoms described in this clinical scenario are strongly suggestive of congestive heart failure (CHF). Left-sided CHF frequently presents with dyspnea on exertion, orthopnea (dyspnea while lying flat) and paroxysmal nocturnal dyspnea (waking from sleep gasping for air). Symptoms of right-sided CHF include lower extremity edema and hepatomegaly. Most commonly combined left- and right-ventricular failure is observed owing to the fact that the most common cause of right heart failure is left heart failure.

CHF occurs due to a progressive decrease in myocardial contractility (systolic dysfunction) or due to inability of heart to relax sufficiently to accommodate incoming blood in diastole (diastolic dysfunction). CHF of both origins ultimately leads to decreased cardiac output and inadequate oxygen delivery to tissues. Decreased perfusion of peripheral tissues induces a number of neuroendocrine compensatory mechanisms, such as:

1. **Renin-angiotensin-aldosterone mechanism:** Decreased renal perfusion leads to diminished stretch of glomerular afferent arterioles and increased synthesis of renin. Increased angiotensin I is converted to angiotensin II, which is a potent vasoconstrictor. By causing vasoconstriction, angiotensin II increases arterial resistance and afterload (**Choice C**). The failing heart is unable to pump blood to the tissues against the increased afterload induced by angiotensin II, so tissue perfusion decreases and more renin is secreted by the kidney creating a vicious circle. Angiotensin II also stimulates the secretion of aldosterone which functions to increase the circulating blood volume (preload) and further exacerbate CHF.
2. Increased sympathetic output is stimulated by baroreceptors that sense decreased perfusion. Epinephrine and norepinephrine increase heart rate and contractility, but these neurotransmitters also increase peripheral arterial resistance thereby increasing afterload.

(Choice A) Cardiac output is decreased in both systolic and diastolic heart failure.

(Choice B) Decreased cardiac output leads to diminished perfusion of the viscera, including the kidneys. Decreased renal perfusion activates the renin-angiotensin-aldosterone mechanism.

(Choice D) As less arterial blood is delivered to peripheral tissues, the contact of tissues with venous blood is increased. Uptake of oxygen from venous blood is higher, and mixed venous oxygen content is decreased.

(Choice E) Increased pressure in pulmonary circulation results in transudation of fluid into the lung interstitium and air spaces. This decreases lung compliance and oxygen diffusion.





accommodate incoming blood in diastole (diastolic dysfunction). CHF of both origins ultimately leads to decreased cardiac output and inadequate oxygen delivery to tissues. Decreased perfusion of peripheral tissues induces a number of neuroendocrine compensatory mechanisms, such as:

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(Choice A) Cardiac output is decreased in both systolic and diastolic heart failure.

(Choice B) Decreased cardiac output leads to diminished perfusion of the viscera, including the kidneys. Decreased renal perfusion activates the renin-angiotensin-aldosterone mechanism.

(Choice D) As less arterial blood is delivered to peripheral tissues, the contact of tissues with venous blood is increased. Uptake of oxygen from venous blood is higher, and mixed venous oxygen content is decreased.

(Choice E) Increased pressure in pulmonary circulation results in transudation of fluid into the lung interstitium and air spaces. This decreases lung compliance and oxygen diffusion.

Educational Objective:

Decreased cardiac output triggers a number of compensatory mechanisms. Renin-angiotensin-aldosterone activation and increased sympathetic output raise arterial resistance (afterload) and exacerbate heart failure by making it more difficult for the failing heart to pump blood to the tissues.

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A 62-year-old woman comes to the hospital with intermittent but progressive substernal chest pain over the last 36 hours. Medical history includes hypertension and hyperlipidemia, but the patient has been poorly compliant with her medication regimen and outpatient follow-up. She previously smoked a pack daily for 30 years but quit last year. Her blood pressure is 130/75 mm Hg and pulse is 73/min. ECG on admission shows normal sinus rhythm with a 2-mm ST segment elevation in leads V2-V5. The patient is treated with medical management. However, on the fifth day of hospitalization she dies suddenly, despite adequate resuscitation. Which of the following is the most likely cause of death in this patient?

☐ A. Carotid artery occlusion

☐ B. Hypertensive emergency

☐ C. Increased venous return

☐ D. Profound hypotension

☐ E. True ventricular aneurysm

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TUTOR

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System Tray



A 62-year-old woman comes to the hospital with intermittent but progressive substernal chest pain over the last 36 hours. Medical history includes hypertension and hyperlipidemia, but the patient has been poorly compliant with her medication regimen and outpatient follow-up. She previously smoked a pack daily for 30 years but quit last year. Her blood pressure is 130/75 mm Hg and pulse is 73/min. ECG on admission shows normal sinus rhythm with a 2-mm ST segment elevation in leads V2-V5. The patient is treated with medical management. However, on the fifth day of hospitalization she dies suddenly, despite adequate resuscitation. Which of the following is the most likely cause of death in this patient?

- ☐ A. Carotid artery occlusion [16%]
- ☐ B. Hypertensive emergency [2%]
- ☐ C. Increased venous return [3%]
- ☒ D. Profound hypotension [41%]
- ☐ E. True ventricular aneurysm [36%]

Omitted

Correct answer
D41%
Answered correctly2 Seconds
Time Spent11/21/2018
Last Updated

Explanation

This patient most likely died from profound hypotension due to **ruptured left ventricular free wall**. Her initial presentation of substernal chest discomfort and ST segment elevation in anterior precordial leads is consistent with acute **anterior wall myocardial infarction (MI)**. Free wall

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This patient most likely died from profound hypotension due to **ruptured left ventricular free wall**. Her initial presentation of substernal chest discomfort and ST segment elevation in anterior precordial leads is consistent with acute **anterior wall myocardial infarction (MI)**. Free wall rupture is a catastrophic mechanical complication that usually occurs within the first 5-14 days after a large anterior transmural MI (from left anterior descending occlusion). During this timeframe, the infarcted myocardium is substantially weakened by coagulative necrosis, neutrophilic and macrophage infiltration, and enzymatic lysis of myocardial connective tissue. Abrupt rupture of the left ventricle leads to **hemopericardium** and **cardiac tamponade**. Patients have sudden onset of chest pain and **profound hypotension and shock**, with rapid progression to pulseless electrical activity and death. Various forms of cardiac rupture (listed from most common to least common) and their associated clinical signs are as follows:

- Free wall rupture – hemopericardium and cardiac tamponade
- Ventricular septal rupture – acute ventricular septal defect and left-to-right shunting
- Papillary muscle rupture – acute onset of severe mitral regurgitation

(Choice A) Carotid artery occlusion can lead to ischemic symptoms (eg, hemiparesis, vision changes) but does not typically cause sudden death.

(Choice B) Hypertensive emergency can lead to aortic dissection that typically presents with tearing chest or back pain. Hypotension and shock can occur with retrograde extension of the dissection into the pericardial cavity or coronary arteries, with resultant hemopericardium or MI, respectively. However, this patient's presentation of initial normal blood pressure and ST segment elevation is more suggestive of acute MI with subsequent cardiac rupture.

(Choice C) Abrupt rupture of the left ventricle leads to hemopericardium and cardiac tamponade. This leads to decreased venous return to the right heart due to increased pressure in the pericardial cavity.

(Choice E) True aneurysms of the ventricular wall are bound by scarred myocardium that is tough and fibrotic. They are a late complication (>1 month) of large transmural infarcts and can cause mural thrombus, arrhythmias, and heart failure, but rarely rupture.

Educational objective:

Rupture of the left ventricular free wall is a catastrophic mechanical complication of anterior wall myocardial infarction (MI) that usually occurs





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electrical activity and death. Various forms of cardiac rupture (listed from most common to least common) and their associated clinical signs are as follows:

- Free wall rupture – hemopericardium and cardiac tamponade
- Ventricular septal rupture – acute ventricular septal defect and left-to-right shunting
- Papillary muscle rupture – acute onset of severe mitral regurgitation

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(Choice E) True aneurysms of the ventricular wall are bound by scarred myocardium that is tough and fibrotic. They are a late complication (>1 month) of large transmural infarcts and can cause mural thrombus, arrhythmias, and heart failure, but rarely rupture.

Educational objective:

Rupture of the left ventricular free wall is a catastrophic mechanical complication of anterior wall myocardial infarction (MI) that usually occurs within the first 5-14 days after MI. Rupture leads to hemopericardium and cardiac tamponade, causing profound hypotension and shock with rapid progression to pulseless electrical activity and death.

References

- Identification and treatment of complications of myocardial infarction.
- Left ventricular free-wall rupture in acute myocardial infarction: a blow-out type case.

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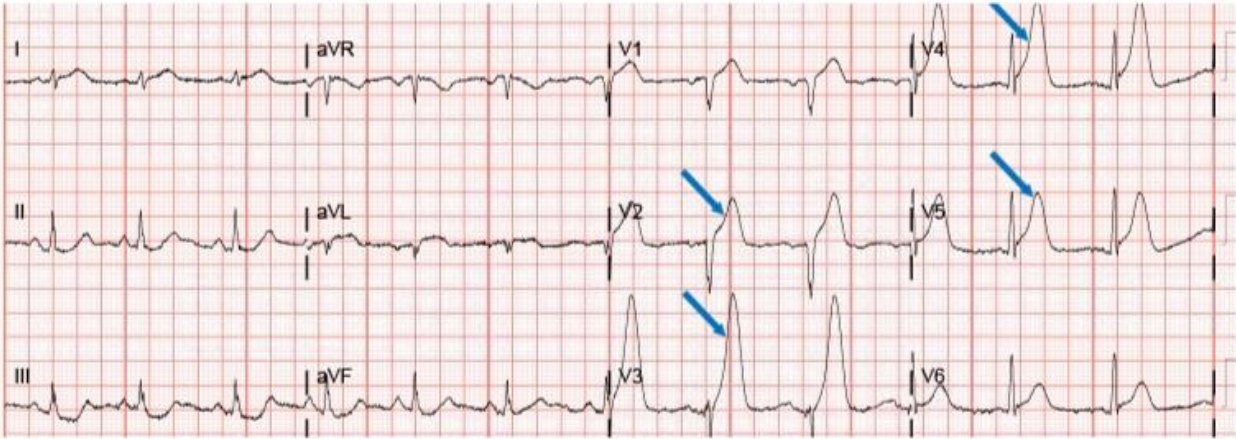
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ECG tracing showing leads I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, V5, and V6. Blue arrows point to ST-segment depression in leads V4, V5, and V6.

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subsequent cardiac rupture.

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Text Zoom

A 54-year-old woman comes to the office due to exertional dyspnea and fatigue. The patient gets short of breath while walking to a store 2 blocks away from her home. She also has trouble breathing at night and sleeps with 2 or 3 pillows to prop her up. She has had occasional palpitations but no chest pain. The patient takes no medications on a regular basis, and does not use tobacco, alcohol, or illicit drugs. After initial evaluation, she is scheduled for a left heart catheterization, the findings of which are shown below.

Which of the following is the most likely diagnosis?

A. Aortic regurgitation

B. Aortic stenosis

C. Mitral regurgitation

D. Mitral stenosis

E. Tricuspid regurgitation

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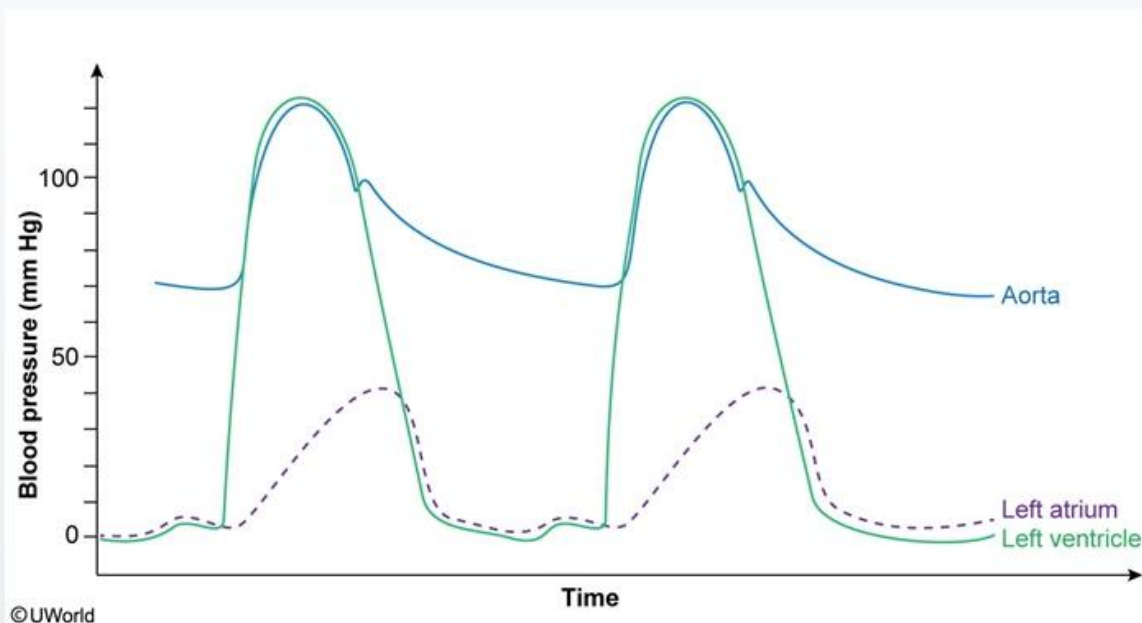
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Windows Taskbar

1:36 PM 2/7/2019



A 54-year-old woman comes to the office due to exertional dyspnea and fatigue. The patient gets short of breath while walking to a store 2 blocks away from her home. She also has trouble breathing at night and sleeps with 2 or 3 pillows to prop her up. She has had occasional palpitations but no chest pain. The patient takes no medications on a regular basis, and does not use tobacco, alcohol, or illicit drugs. After initial evaluation, she is scheduled for a left heart catheterization, the findings of which are shown below.



Which of the following is the most likely diagnosis?

☐ A. Aortic regurgitation [6%]

Block Time Remaining: 00:19:52

TUTOR



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A A A

Text Zoom

A 54-year-old woman comes to the office due to exertional dyspnea and fatigue. The patient gets short of breath while walking to a store 2 blocks away from her home. She also has trouble breathing at night and sleeps with 2 or 3 pillows to prop her up. She has had occasional palpitations but no chest pain. The patient takes no medications on a regular basis, and does not use tobacco, alcohol, or illicit drugs. After initial evaluation, she is scheduled for a left heart catheterization, the findings of which are shown below.

Which of the following is the most likely diagnosis?

A. Aortic regurgitation

B. Aortic stenosis

C. Mitral regurgitation

D. Mitral stenosis

E. Tricuspid regurgitation

Submit

Block Time Remaining: 00:01:45

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Windows Taskbar

1:36 PM 2/7/2019



A 54-year-old woman comes to the office due to exertional dyspnea and fatigue. The patient gets short of breath while walking to a store 2 blocks away from her home. She also has trouble breathing at night and sleeps with 2 or 3 pillows to prop her up. She has had occasional palpitations but no chest pain. The patient takes no medications on a regular basis, and does not use tobacco, alcohol, or illicit drugs. After initial evaluation, she is scheduled for a left heart catheterization, the findings of which are shown below.

Which of the following is the most likely diagnosis?

- ☐ A. Aortic regurgitation [6%]
- ☐ B. Aortic stenosis [5%]
- ☒ C. Mitral regurgitation [66%]
- ☐ D. Mitral stenosis [19%]
- ☐ E. Tricuspid regurgitation [1%]

Omitted

Correct answer
C

66%
Answered correctly

5 Seconds
Time Spent

09/11/2018
Last Updated

Explanation

This patient's left heart catheterization shows pressure tracings consistent with **mitral regurgitation** (MR). In the cardiac cycle, passive filling of the left atrium with blood from the pulmonary veins (atrial diastole) occurs essentially in sync with left ventricular (LV) systole. Left atrial pressure

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Explanation

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This patient's left heart catheterization shows pressure tracings consistent with **mitral regurgitation** (MR). In the cardiac cycle, passive filling of the left atrium with blood from the pulmonary veins (atrial diastole) occurs essentially in sync with left ventricular (LV) systole. Left atrial pressure gradually increases to approximately 10 mm Hg in the normal heart with a characteristic "V wave" created at the end of passive filling. However, in the setting of MR, an incompetent mitral valve allows backflow of blood from the left ventricle during ventricular systole, leading to markedly **elevated left atrial pressure** during this time. This creates an **early and large V wave**, the characteristic pressure tracing finding in patients with MR.

The elevated left atrial pressure in MR leads to atrial dilation and often precipitates atrial fibrillation, symptomatically manifesting with palpitations. Over time, decompensated heart failure, evidenced by dyspnea and orthopnea in this patient, can also occur.

(Choice A) In **aortic regurgitation**, backflow of blood from the aorta into the left ventricle during diastole results in rapid loss of aortic pressure and elevated LV end-diastolic volume with a compensatory increase in LV stroke volume. Pressure tracings show loss of the aortic dirotic notch (normally seen around aortic valve closure), rapid decline of aortic pressure during diastole, and elevated peak LV and aortic systolic pressures. There is also early closure of the mitral valve due to elevated LV diastolic pressure.

(Choice B) **Aortic stenosis** causes obstruction of LV outflow resulting in discordance of LV and aortic systolic pressure. Pressure tracings characteristically show peak LV systolic pressure significantly higher than aortic systolic pressure.

(Choice D) **Mitral stenosis** causes elevated left atrial pressures throughout the cardiac cycle with peak atrial pressure occurring at the time of atrial kick, just before the mitral valve closes at the end of ventricular diastole.

(Choice E) Tricuspid regurgitation causes an early and large V wave on right atrial pressure tracing, similar to that seen on left atrial pressure tracing in the setting of MR. Left-sided pressure tracings (left atrial, left ventricular, aortic) are minimally affected in tricuspid regurgitation.

Educational objective:

Left ventricular systole corresponds to the time of passive filling of the left atrium (atrial diastole). Mitral valve regurgitation leads to markedly elevated left atrial pressure during this period, creating the characteristic early and large V wave on left atrial pressure tracing.

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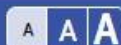
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System Tray



the left atrium with blood from the pulmonary veins (atrial diastole) occurs essentially in sync with left ventricular (LV) systole. Left atrial pressure gradually increases to approximately 10 mm Hg in the normal heart with a characteristic "V wave" created at the end of passive filling. However, in the setting of MR, an incompetent mitral valve allows backflow of blood from the left ventricle during ventricular systole, leading to markedly **elevated left atrial pressure** during this time. This creates an **early and large V wave**, the characteristic pressure tracing finding in patients with MR.

The elevated left atrial pressure in MR leads to atrial dilation and often precipitates atrial fibrillation, symptomatically manifesting with palpitations. Over time, decompensated heart failure, evidenced by dyspnea and orthopnea in this patient, can also occur.

(Choice A) In **aortic regurgitation**, backflow of blood from the aorta into the left ventricle during diastole results in rapid loss of aortic pressure and elevated LV end-diastolic volume with a compensatory increase in LV stroke volume. Pressure tracings show loss of the aortic diastolic notch (normally seen around aortic valve closure), rapid decline of aortic pressure during diastole, and elevated peak LV and aortic systolic pressures. There is also early closure of the mitral valve due to elevated LV diastolic pressure.

(Choice B) **Aortic stenosis** causes obstruction of LV outflow resulting in discordance of LV and aortic systolic pressure. Pressure tracings characteristically show peak LV systolic pressure significantly higher than aortic systolic pressure.

(Choice D) **Mitral stenosis** causes elevated left atrial pressures throughout the cardiac cycle with peak atrial pressure occurring at the time of atrial kick, just before the mitral valve closes at the end of ventricular diastole.

(Choice E) Tricuspid regurgitation causes an early and large V wave on right atrial pressure tracing, similar to that seen on left atrial pressure tracing in the setting of MR. Left-sided pressure tracings (left atrial, left ventricular, aortic) are minimally affected in tricuspid regurgitation.

Educational objective:

Left ventricular systole corresponds to the time of passive filling of the left atrium (atrial diastole). Mitral valve regurgitation leads to markedly elevated left atrial pressure during this period, creating the characteristic early and large V wave on left atrial pressure tracing.

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Question Id: 198

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Text Zoom

A 56-year-old man comes to the emergency department due to progressively worsening dyspnea. The patient can walk only a few blocks before becoming short of breath. He also finds it difficult to sleep lying flat and requires 3 pillows to prop himself upright when sleeping. Other medical problems include long-standing hypertension, for which he occasionally takes his prescribed antihypertensive medication. The patient does not use tobacco, alcohol, or illicit drugs. Blood pressure is 170/100 mm Hg, and pulse is 80/min and regular. Physical examination reveals bilateral basilar lung crackles, jugular venous distension, and bilateral lower extremity edema. Chest x-ray reveals cardiomegaly and hilar prominence. ECG shows left ventricular hypertrophy. Echocardiogram shows elevated pressures in the pulmonary artery. Which of the following is the most likely underlying cause of the observed echocardiographic finding in this patient?

A. Hypoxia-induced pulmonary vasoconstriction

B. Increased pulmonary arterial blood flow

C. Increased pulmonary venous pressure

D. Inflammatory pulmonary vascular disease

E. Obliteration of the pulmonary vascular bed

F. Thrombotic obstruction of the pulmonary arterial tree

Submit

Block Time Remaining: 00:01:50

TUTOR

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Feedback

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End Block

Windows Taskbar

System Tray



A 56-year-old man comes to the emergency department due to progressively worsening dyspnea. The patient can walk only a few blocks before becoming short of breath. He also finds it difficult to sleep lying flat and requires 3 pillows to prop himself upright when sleeping. Other medical problems include long-standing hypertension, for which he occasionally takes his prescribed antihypertensive medication. The patient does not use tobacco, alcohol, or illicit drugs. Blood pressure is 170/100 mm Hg, and pulse is 80/min and regular. Physical examination reveals bilateral basilar lung crackles, jugular venous distension, and bilateral lower extremity edema. Chest x-ray reveals cardiomegaly and hilar prominence. ECG shows left ventricular hypertrophy. Echocardiogram shows elevated pressures in the pulmonary artery. Which of the following is the most likely underlying cause of the observed echocardiographic finding in this patient?

- ☐ A. Hypoxia-induced pulmonary vasoconstriction [11%]
- ☐ B. Increased pulmonary arterial blood flow [9%]
- ☒ C. Increased pulmonary venous pressure [71%]
- ☐ D. Inflammatory pulmonary vascular disease [1%]
- ☐ E. Obliteration of the pulmonary vascular bed [4%]
- ☐ F. Thrombotic obstruction of the pulmonary arterial tree [1%]

Omitted

Correct answer
C71%
Answered correctly3 Seconds
Time Spent08/28/2018
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Explanation

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Explanation

This patient with dyspnea, orthopnea, and pulmonary crackles has **left-sided heart failure** (LHF) most likely due to long-standing, poorly controlled hypertension. Hypertensive heart disease typically manifests as heart failure due to concentric left ventricular (LV) hypertrophy and consequent LV diastolic dysfunction. LHF of any cause (eg, LV systolic dysfunction, valvular dysfunction) will result in **higher diastolic filling pressures**. This increase in pressure is **transmitted backward** to the left atrium and pulmonary veins, resulting in **pulmonary venous congestion** and consequent elevations in pulmonary capillary and pulmonary arterial pressure. The resulting **pulmonary hypertension** (PH) can lead to right-sided heart failure with jugular venous distension and peripheral edema.

Over time, **remodeling** of the pulmonary vasculature occurs with increased smooth muscle cell proliferation (medial hypertrophy) and collagen deposition (intimal thickening and fibrosis). The remodeling is less extensive than in (primary) **pulmonary arterial hypertension**; therefore, the PH is at least partially reversible with treatment of the LHF.

(Choices A and E) Hypoxia-induced vasoconstriction and emphysematous obliteration of the vasculature underlie PH that occurs secondary to chronic obstructive pulmonary disease. Although there is a small component of hypoxia-induced pulmonary vasoconstriction in LHF, pulmonary venous congestion is the major cause of PH.

(Choice B) Congenital heart disease that causes left-to-right shunting (eg, ventricular septal defect, atrial septal defect) can lead to PH via an increase in pulmonary arterial blood flow. In LHF, PH occurs due to backwards transmission of pressure from increased pulmonary venous blood volume; pulmonary arterial blood flow remains the same or decreases as LHF worsens.

(Choice D) Inflammatory large-vessel vasculitis (ie, Takayasu arteritis, giant cell arteritis) can sometimes involve the pulmonary arteries and cause PH. However, pulmonary vascular inflammation does not play a role in PH occurring secondary to LHF.

(Choice F) Thrombotic obstruction of the pulmonary arterial tree occurs in acute pulmonary embolism, and some patients can develop chronic thromboembolic PH despite receiving appropriate anticoagulation therapy. However, this patient has no history of thromboembolic disease, and

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deposition (intimal thickening and fibrosis). The remodeling is less extensive than in (primary) **pulmonary arterial hypertension**, therefore, the PH is at least partially reversible with treatment of the LHF.

(Choices A and E) Hypoxia-induced vasoconstriction and emphysematous obliteration of the vasculature underlie PH that occurs secondary to chronic obstructive pulmonary disease. Although there is a small component of hypoxia-induced pulmonary vasoconstriction in LHF, pulmonary venous congestion is the major cause of PH.

(Choice B) Congenital heart disease that causes left-to-right shunting (eg, ventricular septal defect, atrial septal defect) can lead to PH via an increase in pulmonary arterial blood flow. In LHF, PH occurs due to backwards transmission of pressure from increased pulmonary venous blood volume; pulmonary arterial blood flow remains the same or decreases as LHF worsens.

(Choice D) Inflammatory large-vessel vasculitis (ie, Takayasu arteritis, giant cell arteritis) can sometimes involve the pulmonary arteries and cause PH. However, pulmonary vascular inflammation does not play a role in PH occurring secondary to LHF.

(Choice F) Thrombotic obstruction of the pulmonary arterial tree occurs in acute pulmonary embolism, and some patients can develop chronic thromboembolic PH despite receiving appropriate anticoagulation therapy. However, this patient has no history of thromboembolic disease, and his orthopnea and crackles are more suggestive of PH due to LHF.

Educational objective:

Left-sided heart failure can cause secondary pulmonary hypertension via elevated left-sided diastolic filling pressures transmitting backward to the pulmonary veins, resulting in pulmonary venous congestion. Over time, pulmonary arterial remodeling (medial hypertrophy and intimal thickening with fibrosis) can occur, but not to the extent that occurs in (primary) pulmonary arterial hypertension.

References

- Pulmonary hypertension in left heart disease.

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Correct answer: C

Answered correctly

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Exhibit Display

Classification of pulmonary hypertension	
Pulmonary arterial hypertension (Group 1)	- Primary change in pulmonary arteries - Hereditary (eg, <i>BMPR2</i> mutation) - Connective tissue disease (eg, RA, SS) - HIV infection - Treatment targeted at endothelial dysfunction
Pulmonary hypertension (Groups 2-5)	- Secondary to another disease process - Left-sided heart failure - Chronic lung disease/hypoxia - Chronic pulmonary thromboembolism - Treatment aimed at underlying disease

RA = rheumatoid arthritis; SS = systemic sclerosis.

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Explanation

This patient with dyspnea, orthopnea, and controlled hypertension. Hyperoxia leads to consequent LV diastolic dysfunction and consequent pulmonary pressures. This increase in pulmonary pressures leads to pulmonary congestion and consequent edema, which can lead to right-sided heart failure.

Over time, remodeling of the pulmonary arteries and deposition (intimal thickening and hyperplasia) of the PH is at least partially reversible.

(Choices A and E) Hypoxia-induced pulmonary hypertension in chronic obstructive pulmonary disease and pulmonary venous congestion is the major cause of PH.

(Choice B) Congenital heart disease leads to an increase in pulmonary arterial blood flow, which leads to volume; pulmonary arterial blood flow.

(Choice D) Inflammatory large-vessel vasculitis (ie, Takayasu arteritis, giant cell arteritis) can sometimes involve the pulmonary arteries and cause PH. However, pulmonary vascular inflammation does not play a role in PH occurring secondary to LHF.

(Choice E) Thrombotic obstruction of the pulmonary arterial tree occurs in acute pulmonary embolism, and some patients can develop chronic PH.

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Text Zoom

A 33-year-old woman who recently immigrated to the United States is brought to the emergency department with severe shortness of breath and hemoptysis. Physical examination reveals a diastolic murmur. Chest x-ray shows severe pulmonary vascular congestion and edema. She is admitted to the hospital, treated with diuretics, and begins to feel better. However, during her hospitalization, she develops right-sided hemiparesis. Which of the following additional findings in this patient would be most suggestive of combined disease involving the mitral and aortic valves rather than exclusive mitral involvement?

A. Increased left ventricular diastolic pressure

B. Increased pulmonary artery pressure

C. Increased pulmonary capillary wedge pressure

D. Reduced pulmonary vascular compliance

E. Right ventricular dilation

F. Tricuspid regurgitation

Submit

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Text Zoom

A 33-year-old woman who recently immigrated to the United States is brought to the emergency department with severe shortness of breath and hemoptysis. Physical examination reveals a diastolic murmur. Chest x-ray shows severe pulmonary vascular congestion and edema. She is admitted to the hospital, treated with diuretics, and begins to feel better. However, during her hospitalization, she develops right-sided hemiparesis. Which of the following additional findings in this patient would be most suggestive of combined disease involving the mitral and aortic valves rather than exclusive mitral involvement?

✔

☐

A. Increased left ventricular diastolic pressure [73%]

☐

B. Increased pulmonary artery pressure [3%]

☐

C. Increased pulmonary capillary wedge pressure [14%]

☐

D. Reduced pulmonary vascular compliance [2%]

☐

E. Right ventricular dilation [5%]

☐

F. Tricuspid regurgitation [1%]

Omitted

Correct answer
A

73%

Answered correctly

3 Seconds

Time Spent

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Last Updated

Explanation

This patient's physical examination findings are suggestive of mitral stenosis (MS). In isolated MS, cardiac and pulmonary pressures proximal to

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Explanation

This patient's physical examination findings are suggestive of mitral stenosis (MS). In isolated MS, cardiac and pulmonary pressures proximal to the stenotic mitral valve are markedly elevated. However, diastolic pressure in the left ventricle (LV) is usually near normal or even decreased with severe stenosis. Increased LV diastolic pressure in a patient with suspected MS suggests that the aortic valve is also dysfunctional.

Rheumatic heart disease almost always affects the mitral valve, but both the mitral and aortic valves are affected in about 25% of cases.

Rheumatic heart disease typically causes combined aortic stenosis and regurgitation, both of which can increase LV diastolic pressure.

Rheumatic deformation of the aortic valve can also predispose to infective endocarditis even when the valve is functionally normal. This patient's right-sided hemiparesis is most likely due to embolism, which could have originated from an atrial mural thrombus secondary to atrial dilation from MS or an endocarditis-related valvular vegetation (less likely).

(Choices B and D) Severe, isolated MS can cause increased pulmonary artery pressure (pulmonary hypertension) via backward transmission of elevated pulmonary capillary wedge pressure. Longstanding pulmonary hypertension can result in reduced pulmonary vascular compliance due to endothelial-mediated pulmonary vasoconstriction, reactive hypertrophy of the arterial muscle layer, and partial obliteration of the pulmonary capillary bed.

(Choice C) Isolated MS can cause a significant elevation in left atrial pressure that is transmitted to the pulmonary veins and capillaries, increasing pulmonary capillary wedge pressure. In MS patients, the wedge pressure reflects only the left atrial end diastolic pressure and not the left ventricular end diastolic pressure.

(Choices E and F) Right ventricular dilatation can occur in MS when the resulting pulmonary hypertension is severe enough to cause right heart failure. Tricuspid regurgitation can occur as a complication of right ventricular dilatation.

Educational objective:

Isolated mitral stenosis elevates left atrial diastolic pressure and can therefore cause elevated pulmonary capillary wedge pressure, pulmonary hypertension, decreased pulmonary vascular compliance, right ventricular dilatation, and functional tricuspid regurgitation. Diastolic pressure in

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This patient's physical examination findings are suggestive of mitral stenosis (MS). In isolated MS, cardiac and pulmonary pressures proximal to the stenotic mitral valve are markedly elevated. However, diastolic pressure in the left ventricle (LV) is usually near normal or even decreased with severe stenosis. Increased LV diastolic pressure in a patient with suspected MS suggests that the aortic valve is also dysfunctional.

Rheumatic heart disease almost always affects the mitral valve, but both the mitral and aortic valves are affected in about 25% of cases. Rheumatic heart disease typically causes combined aortic stenosis and regurgitation, both of which can increase LV diastolic pressure. Rheumatic deformation of the aortic valve can also predispose to infective endocarditis even when the valve is functionally normal. This patient's right-sided hemiparesis is most likely due to embolism, which could have originated from an atrial mural thrombus secondary to atrial dilation from MS or an endocarditis-related valvular vegetation (less likely).

(Choices B and D) Severe, isolated MS can cause increased pulmonary artery pressure (pulmonary hypertension) via backward transmission of elevated pulmonary capillary wedge pressure. Longstanding pulmonary hypertension can result in reduced pulmonary vascular compliance due to endothelial-mediated pulmonary vasoconstriction, reactive hypertrophy of the arterial muscle layer, and partial obliteration of the pulmonary capillary bed.

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(Choices E and F) Right ventricular dilatation can occur in MS when the resulting pulmonary hypertension is severe enough to cause right heart failure. Tricuspid regurgitation can occur as a complication of right ventricular dilatation.

Educational objective:

Isolated mitral stenosis elevates left atrial diastolic pressure and can therefore cause elevated pulmonary capillary wedge pressure, pulmonary hypertension, decreased pulmonary vascular compliance, right ventricular dilatation, and functional tricuspid regurgitation. Diastolic pressure in the left ventricle is usually near normal or even decreased with severe mitral stenosis.

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Windows Taskbar

System Tray

Cellular physiologists are investigating the mechanisms of contraction cessation in cardiomyocytes in response to ischemic injury. In an animal experiment, the left anterior descending coronary artery is ligated at time 0, and changes in ATP levels within the affected myocardial cells are recorded. At which point on the graph does the affected portion of the myocardium stop contracting?

- ☐ A. A
- ☐ B. B
- ☐ C. C
- ☐ D. D
- ☐ E. E

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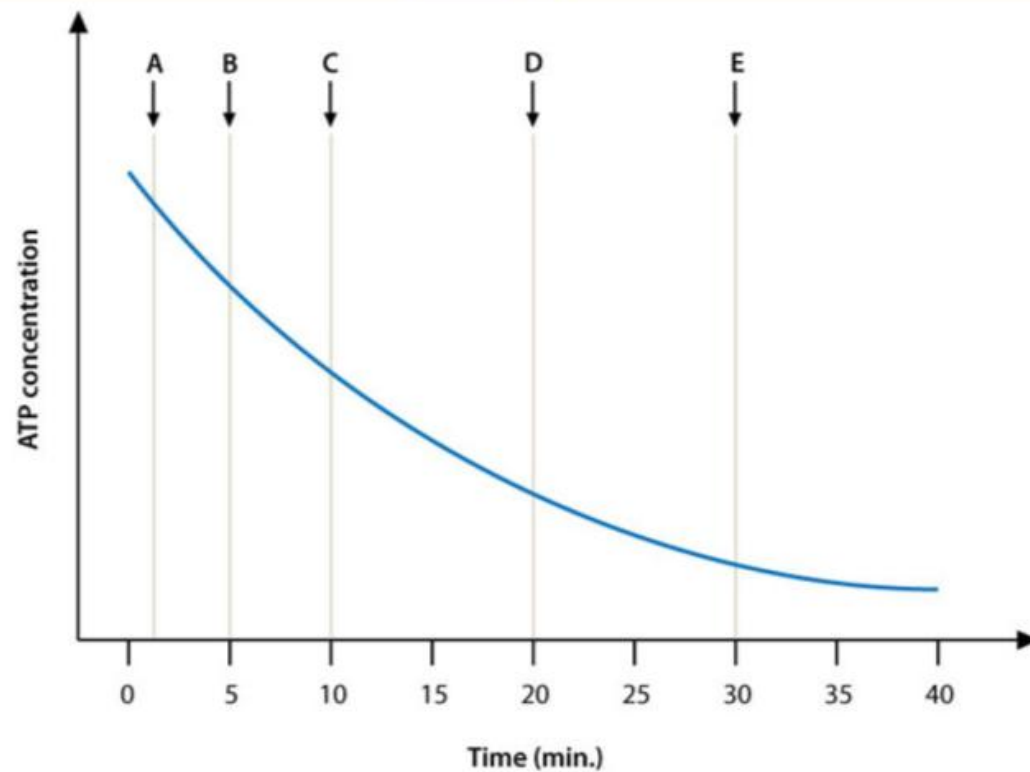
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Exhibit Display



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✓ A. A [25%]

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TUTOR



Cellular physiologists are investigating the mechanisms of contraction cessation in cardiomyocytes in response to ischemic injury. In an animal experiment, the left anterior descending coronary artery is ligated at time 0, and changes in ATP levels within the affected myocardial cells are recorded. At which point on the graph does the affected portion of the myocardium stop contracting?

- ☐ A. A
- ☐ B. B
- ☐ C. C
- ☐ D. D
- ☐ E. E

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Text Zoom

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Cellular physiologists are investigating the mechanisms of contraction cessation in cardiomyocytes in response to ischemic injury. In an animal experiment, the left anterior descending coronary artery is ligated at time 0, and changes in ATP levels within the affected myocardial cells are recorded. At which point on the graph does the affected portion of the myocardium stop contracting?

✔

☒ A. A [25%]

☐ B. B [11%]

☐ C. C [13%]

☐ D. D [25%]

☐ E. E [24%]

Omitted

Correct answer
A

25%

Answered correctly

2 Seconds

Time Spent

12/19/2018

Last Updated

Explanation

The major early biochemical consequence of total myocardial ischemia is cessation of aerobic glycolysis and initiation of anaerobic glycolysis. This transition occurs within seconds and results in inadequate production of high-energy phosphates (eg, ATP and creatine phosphate) and the accumulation of deleterious metabolites, including lactate. Although total ATP levels remain relatively normal during the first few minutes of ischemia, ATP is rapidly depleted from areas of the cell with high metabolic demand, such as the cytosol surrounding the contraction fibers and

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Explanation

The major early biochemical consequence of total myocardial ischemia is cessation of aerobic glycolysis and initiation of anaerobic glycolysis. This transition occurs within seconds and results in inadequate production of high-energy phosphates (eg, ATP and creatine phosphate) and the accumulation of deleterious metabolites, including lactate. Although total ATP levels remain relatively normal during the first few minutes of ischemia, ATP is rapidly depleted from areas of the cell with high metabolic demand, such as the cytosol surrounding the contraction fibers and electrolyte transport pumps. The depletion of ATP in critical cellular areas and the accumulation of toxins result in loss of contractility within about 60 seconds of total myocardial ischemia **(Choice A)**.

As ischemia persists, ATP levels within the affected cardiac myocytes continue to fall while lactate levels continue to rise. When ischemia lasts less than 30 minutes, loss of contractile function is reversible. However, on restoration of blood flow, full myocardial contractility is not immediately restored. Instead, there is prolonged dysfunction of the myocardium (myocardial stunning), with contractility gradually returning to normal over the next several hours to days. Increasing durations of ischemia prolong the time that the myocardium remains stunned.

Under hypoxic conditions, ATP within cardiomyocytes is degraded to ADP, AMP, and eventually adenosine. Adenosine is able to cross the cell membrane and function as a vasodilator when coronary blood flow is insufficient to meet myocardial demand. However, persistent ischemia can lead to depletion of massive amounts of adenosine. After 30 minutes of total myocardial ischemia, about half of the cellular adenine stores are lost. At this point, failure of cellular homeostasis is inevitable and ischemic injury becomes irreversible **(Choice E)**.

Educational objective:

Loss of cardiomyocyte contractility occurs within 60 seconds after the onset of total ischemia. When ischemia lasts less than 30 minutes, restoration of blood flow leads to reversible contractile dysfunction (myocardial stunning), with contractility gradually returning to normal over the next several hours to days. However, after about 30 minutes of total ischemia, ischemic injury becomes irreversible.

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Block Time Remaining: 00:01:57

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1:38 PM 2/7/2019

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 54-year-old Caucasian male complains of "dizzy spells" when walking his dog in the morning. He denies any chest pain, shortness of breath, weakness, or headaches. Cardiac auscultation findings at the right sternal border are given below. Which of the following is the most likely cause of this patient's symptoms?

A. Bicuspid aortic valve

B. Infective endocarditis

C. Marfan syndrome

D. Mitral valve prolapse

E. Ventricular septal defect

Play Media

Submit

Block Time Remaining: 00:01:59

TUTOR

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End Block

Windows Taskbar

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 54-year-old Caucasian male complains of "dizzy spells" when walking his dog in the morning. He denies any chest pain, shortness of breath, weakness, or headaches. Cardiac auscultation findings at the right sternal border are given below. Which of the following is the most likely cause of this patient's symptoms?

Play Media

✓

☒

A. Bicuspid aortic valve [69%]

☐

B. Infective endocarditis [5%]

☐

C. Marfan syndrome [2%]

☐

D. Mitral valve prolapse [12%]

☐

E. Ventricular septal defect [9%]

Omitted

Correct answer
A

69%

Answered correctly

166 Seconds

Time Spent

12/29/2018

Last Updated

Explanation

Block Time Remaining: 00:04:43

TUTOR

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End Block

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Text Zoom

Explanation

The cardiac auscultation findings in this patient demonstrate a harsh, crescendo-decrescendo systolic ejection murmur at the right sternal border, which are classic for aortic stenosis. Patients with aortic stenosis typically remain asymptomatic for a prolonged period of time. In more advanced cases, symptoms usually first present with exertion and can include syncope, dizziness, angina, or heart failure.

The three major causes of valvular aortic stenosis are a congenitally abnormal valve with calcification (e.g. bicuspid valve), a calcified normal valve, or rheumatic heart disease. Although rheumatic heart disease is the most common cause worldwide, calcific aortic valve disease (in either a bicuspid or tricuspid valve) is more common in the United States.

(Choice B) Although infective endocarditis can produce a heart murmur, involvement of the aortic valve is much less common than the other valves because of the relatively high blood flow. In addition, this patient does not have other clinical features suggestive of infective endocarditis (i.e. fever).

(Choice C) Marfan syndrome is associated with aortic root disease, which results in aneurysmal dilatation, aortic regurgitation, and aortic dissection. Aortic regurgitation would cause a diastolic murmur, not systolic.

(Choice D) Mitral valve prolapse produces a mid-systolic click and a late systolic murmur heard best over the cardiac apex, as opposed to the crescendo-decrescendo murmur in this case.

(Choice E) A holosystolic murmur is typically heard in patients with a ventricular septal defect, which can be differentiated from this patient's crescendo-decrescendo murmur.

Educational objective:

A bicuspid aortic valve is a common cause of aortic stenosis in the United States. The classic auscultatory finding in patients with aortic stenosis is a harsh, crescendo-decrescendo systolic ejection murmur heard best in the right second intercostal space with radiation to the carotids.

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Question Id: 2105

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 52-year-old man comes to the physician because of increasing fatigue over the last 2 weeks. He has also been experiencing exertional dyspnea and occasional headaches. Cardiac auscultation findings at the left sternal border are shown below. Which of the following is most consistent with this patient's auscultatory findings?

Play Media

☐

A. Aortic regurgitation

☐

B. Hypertrophic cardiomyopathy

☐

C. Mitral valve prolapse

☐

D. Papillary muscle dysfunction

☐

E. Tricuspid regurgitation

Submit

Block Time Remaining: 00:04:45

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Windows Taskbar

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 52-year-old man comes to the physician because of increasing fatigue over the last 2 weeks. He has also been experiencing exertional dyspnea and occasional headaches. Cardiac auscultation findings at the left sternal border are shown below. Which of the following is most consistent with this patient's auscultatory findings?

Play Media

✓

☒

A. Aortic regurgitation [34%]

☐

B. Hypertrophic cardiomyopathy [13%]

☐

C. Mitral valve prolapse [19%]

☐

D. Papillary muscle dysfunction [8%]

☐

E. Tricuspid regurgitation [24%]

Omitted

Correct answer
A

34%

Answered correctly

248 Seconds

Time Spent

01/07/2019

Last Updated

Explanation

Block Time Remaining: 00:08:50

TUTOR

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1:46 PM

2/7/2019

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Correct answer: A

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Explanation

Cardiac murmurs*

Systolic	Aortic stenosis		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid-left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2

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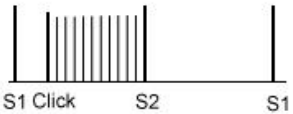
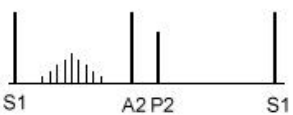
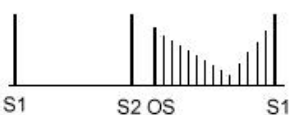
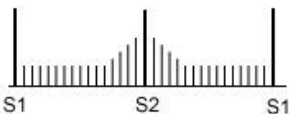
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Systolic		S1 S2 S1	
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis		Opening snap followed by mid-diastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machine-like in interscapular region (posteriorly)

*Pulmonic and tricuspid valve murmurs are similar in character to their aortic and mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.

LUSB = left upper sternal border. RUSB = right upper sternal border.

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	defect		sternal border
Diastolic	Aortic regurgitation		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machine-like in interscapular region (posteriorly)

*Pulmonic and tricuspid valve murmurs are similar in character to their aortic and mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.

LUSB = left upper sternal border; RUSB = right upper sternal border.

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This patient has an **aortic regurgitation (AR)** murmur. It is a high-pitched, blowing, **diastolic** murmur; the intensity is maximal right after A2 (sound of aortic valve closure) and proceeds in a **decrescendo** pattern for part or all of diastole. Higher overall intensity and longer duration of the murmur correlate with the severity of AR, and A2 may be absent in severe AR.

When AR is due to **aortic root dilation** (eg, in Marfan syndrome) the murmur is **best heard** at the **right upper sternal border** (classic aortic valve auscultatory location). However, for AR due to **valvular pathology** (eg, bicuspid aortic valve, rheumatic heart disease) the murmur is best heard at the **left third intercostal space**. In either scenario, the murmur is best appreciated at the end of expiration with the patient leaning forward (minimized lung volume with the heart closer to the chest wall enhances left-sided murmurs).

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Text Zoom

This patient has an **aortic regurgitation** (AR) murmur. It is a high-pitched, blowing, **diastolic** murmur; the intensity is maximal right after A2 (sound of aortic valve closure) and proceeds in a **decrescendo** pattern for part or all of diastole. Higher overall intensity and longer duration of the murmur correlate with the severity of AR, and A2 may be absent in severe AR.

When AR is due to **aortic root dilation** (eg, in Marfan syndrome) the murmur is **best heard** at the **right upper sternal border** (classic aortic valve auscultatory location). However, for AR due to **valvular pathology** (eg, bicuspid aortic valve, rheumatic heart disease) the murmur is best heard at the **left third intercostal space**. In either scenario, the murmur is best appreciated at the end of expiration with the patient leaning forward (minimized lung volume with the heart closer to the chest wall enhances left-sided murmurs).

(Choice B) Hypertrophic cardiomyopathy causes left ventricular outflow obstruction and produces a crescendo-decrescendo systolic murmur similar to that of aortic stenosis. The murmurs can be differentiated by maneuvers that increase left ventricular blood volume (eg, squatting); the murmur of hypertrophic cardiomyopathy decreases in intensity with increased left ventricular blood volume (outflow obstruction is reduced), whereas the murmur of aortic stenosis increases in intensity.

(Choice C) Mitral valve prolapse is classically associated with a midsystolic click and a late systolic murmur best heard at the cardiac apex.

(Choice D) Papillary muscle dysfunction can lead to mitral regurgitation, which causes a holosystolic murmur best heard at the cardiac apex.

(Choice E) Tricuspid regurgitation causes a holosystolic murmur that is best heard at the left lower sternal border. The murmur can be differentiated from that of mitral regurgitation by auscultation during inspiration; the murmur of tricuspid regurgitation will increase in intensity with inspiration as right ventricular blood volume is increased.

Educational objective:

Aortic regurgitation (AR) causes a high-pitched, blowing, diastolic murmur with a decrescendo intensity pattern. The murmur of AR due to aortic root dilation is best heard at the right upper sternal border, whereas the murmur of AR due to valvular pathology is best heard at the left third intercostal space.

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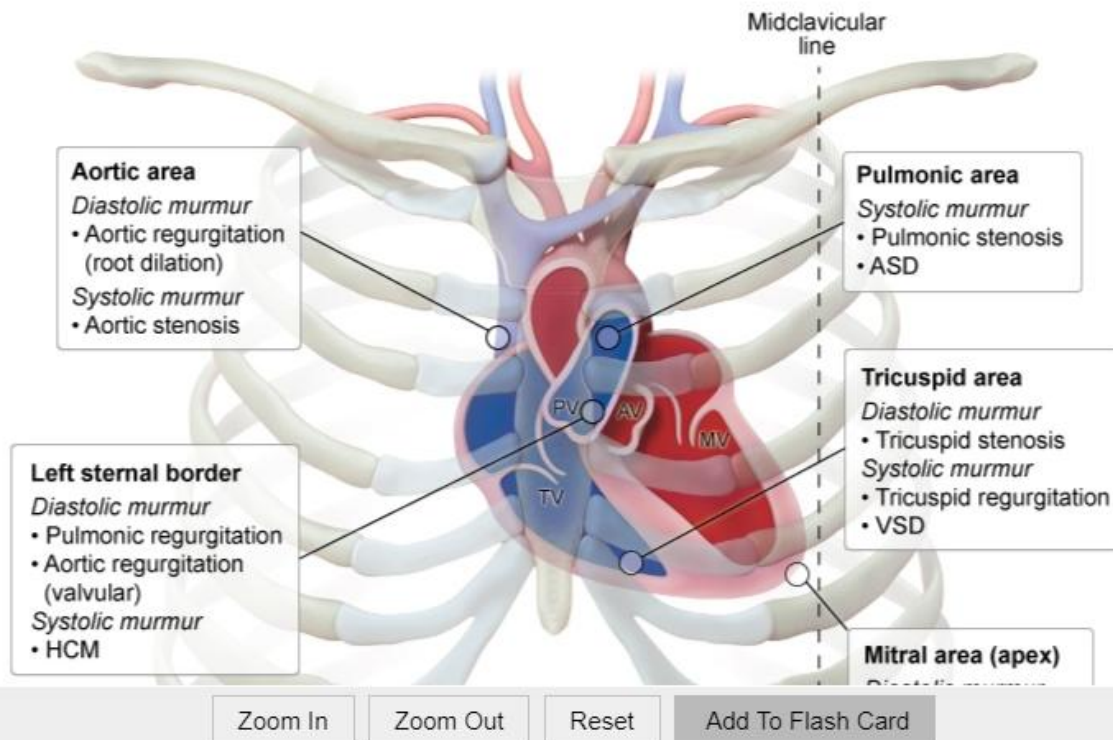
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Windows Taskbar



Exhibit Display

Cardiac auscultatory locations



Block Time Remaining: 00:08:50

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Exhibit Display

Left sternal border*Diastolic murmur*

- Pulmonic regurgitation
- Aortic regurgitation (valvular)
- Systolic murmur*
- HCM

Tricuspid area*Diastolic murmur*

- Tricuspid stenosis
- Systolic murmur*
- Tricuspid regurgitation
- VSD

Mitral area (apex)*Diastolic murmur*

- Mitral stenosis
- Systolic murmur*
- Mitral regurgitation
- MVP

ASD = atrial septal defect; AV = aortic valve; HCM = hypertrophic cardiomyopathy; MV = mitral valve; MVP = mitral valve prolapse; PV = pulmonic valve; TV = tricuspid valve; VSD = ventricular septal defect.
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Zoom In

Zoom Out

Reset

Add To Flash Card

Block Time Remaining: 00:08:50

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 12-month-old boy is brought to the pediatrician by his parents for an upper respiratory infection. His temperature is 38.3 C (101 F), blood pressure is 92/45 mm Hg, pulse is 110/min, and respirations are 25/min. Physical examination reveals erythema and swelling of the nasal mucosa and nasal discharge. Cardiac auscultation findings at the left sternal border are given below. The remainder of the examination is unremarkable. Which of the following is the most likely diagnosis?

Play Media

A. Aortic aneurysm

B. Aortic coarctation

C. Atrial septal defect

D. Mitral valve prolapse

E. Patent ductus arteriosus

Submit

Block Time Remaining: 00:08:53

TUTOR

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1:47 PM

2/7/2019

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 12-month-old boy is brought to the pediatrician by his parents for an upper respiratory infection. His temperature is 38.3 C (101 F), blood pressure is 92/45 mm Hg, pulse is 110/min, and respirations are 25/min. Physical examination reveals erythema and swelling of the nasal mucosa and nasal discharge. Cardiac auscultation findings at the left sternal border are given below. The remainder of the examination is unremarkable. Which of the following is the most likely diagnosis?

Play Media

A. Aortic aneurysm [1%]

B. Aortic coarctation [3%]

C. Atrial septal defect [25%]

D. Mitral valve prolapse [19%]

E. Patent ductus arteriosus [50%]

Omitted

Correct answer E

50%

Answered correctly

188 Seconds

Time Spent

08/31/2018

Last Updated

Explanation

Block Time Remaining: 00:11:58

TUTOR

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Feedback

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Explanation

The audio recording reveals a continuous murmur along with inspiratory splitting of S2. The key characteristic of this patient's murmur is its continuous nature, which is classic for patent ductus arteriosus (PDA). The symptoms caused by a PDA are quite variable depending on its length and size, both of which relate to the degree of left-to-right shunting. Because this patient's PDA was detected incidentally, he most likely has a small PDA. Diagnosis of small PDAs is often suspected on routine auscultation and confirmed with echocardiography. PDA is associated with a continuous murmur heard best in the left infraclavicular region with maximal intensity at S2. Patients at greatest risk of PDA are those born prematurely and those with cyanotic congenital heart disease.

(Choice A) An ascending aortic aneurysm is often associated with aortic regurgitation, which would result in an early diastolic murmur as opposed to a continuous murmur.

(Choice B) Coarctation of the aorta can be associated with a continuous murmur due to large collateral vessels, but this would be much less common than a PDA. In addition, other characteristics of aortic coarctation, such as differing blood pressure or pulse delay between the upper and lower extremities, would likely be evident on physical examination.

(Choice C) A small atrial septal defect can cause a continuous murmur in rare instances, and typically does so only when associated with mitral valve obstruction due to an elevated pressure gradient across the atria (Lutembacher syndrome). This would be much less common than a PDA.

(Choice D) Mitral valve prolapse classically results in a midsystolic click and a mid-to-late systolic murmur.

Educational objective:

Patent ductus arteriosus (PDA) is characterized by a continuous murmur heard best in the left infraclavicular region with maximal intensity at S2. A small PDA is often asymptomatic and is usually detected incidentally during routine cardiac auscultation. It occurs most commonly in patients born prematurely and those with cyanotic congenital heart disease.

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Block Time Remaining: 00:11:58

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Text Zoom

A 63-year-old man comes to the emergency department due to progressive dyspnea and fatigue. Over the last week, he has not been able to lie flat due to difficulty breathing and has had to sleep in a sitting position. He had an anterior myocardial infarction 6 months ago and has not been compliant with his medication regimen since that time. Medical history is also significant for hypertension. The patient is an ex-smoker with a 30-pack-year history. He does not drink alcohol. His father died of a heart attack at age 60. Blood pressure is 170/100 mm Hg, and pulse is 100/min and regular. Oxygen saturation is 90% on room air. He is afebrile. Auscultation reveals crackles at the lung bases, an S3 gallop, and a II/VI holosystolic murmur over the apex. The patient is admitted to the hospital, and after treatment with diuretics and vasodilators his condition improves significantly. Three days later there are no appreciable gallops or murmurs on cardiac examination. Which of the following best explains the murmur heard at the time of the initial examination?

A. Heavily calcified mitral annulus

B. Increased flow velocity through the aortic valve

C. Ruptured chordae tendineae

D. Secondary mitral regurgitation

E. Thickened and deformed mitral valve cusps

Submit

Block Time Remaining: 00:11:59

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Windows Taskbar

System Tray



A 63-year-old man comes to the emergency department due to progressive dyspnea and fatigue. Over the last week, he has not been able to lie flat due to difficulty breathing and has had to sleep in a sitting position. He had an anterior myocardial infarction 6 months ago and has not been compliant with his medication regimen since that time. Medical history is also significant for hypertension. The patient is an ex-smoker with a 30-pack-year history. He does not drink alcohol. His father died of a heart attack at age 60. Blood pressure is 170/100 mm Hg, and pulse is 100/min and regular. Oxygen saturation is 90% on room air. He is afebrile. Auscultation reveals crackles at the lung bases, an S3 gallop, and a II/VI holosystolic murmur over the apex. The patient is admitted to the hospital, and after treatment with diuretics and vasodilators his condition improves significantly. Three days later there are no appreciable gallops or murmurs on cardiac examination. Which of the following best explains the murmur heard at the time of the initial examination?

- ☐ A. Heavily calcified mitral annulus [1%]
- ☐ B. Increased flow velocity through the aortic valve [9%]
- ☐ C. Ruptured chordae tendineae [6%]
- ☒ D. Secondary mitral regurgitation [80%]
- ☐ E. Thickened and deformed mitral valve cusps [2%]

Omitted

Correct answer
D80%
Answered correctly3 Seconds
Time Spent11/15/2018
Last Updated

Explanation

Block Time Remaining: 00:12:01

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Explanation

This patient's presentation with fatigue, progressive dyspnea, orthopnea, pulmonary crackles, and an S3 heart sound is consistent with **decompensated heart failure**. Appropriate treatment with diuretics and vasodilators resulted in symptomatic improvement and disappearance of the apical holosystolic murmur, which was most likely due to **secondary (functional) mitral valve regurgitation** (MR).

Whereas primary MR is caused by an intrinsic defect of the mitral valve apparatus (eg, cleft in a valve cusp, myxomatous degeneration of the chordae tendineae), secondary MR occurs due to other factors. Decompensated heart failure is a common cause of secondary MR because it leads to an increase in left ventricular end-diastolic volume (LVEDV), or preload, with **dilation of the mitral valve annulus** (the tissue on which the mitral valve cusps are mounted) and taut stretching of the chordae tendineae. The dilated annulus and **restricted movement of the chordae tendineae** can cause insufficient closure of an intrinsically normal mitral valve, resulting in MR. Systemic hypertension can also contribute to secondary MR by favoring relatively lower-resistance regurgitant flow.

Treatment with diuretics to reduce LVEDV and vasodilators to reduce blood pressure can lead to resolution of heart failure-induced MR.

(Choice A) Calcification of the mitral valve annulus may occur with aging, and can be accelerated by hypertension or advanced kidney disease. It is usually an incidental finding that does not affect mitral valve function.

(Choice B) Increased flow velocity through the aortic valve occurs with aortic valve stenosis. The resulting murmur is crescendo-decrescendo and best heard at the right second intercostal space (rather than holosystolic and best heard at the apex).

(Choice C) Chordae tendineae rupture is a potential cause of primary MR that can occur in the setting of bacterial endocarditis, connective tissue disease (eg, Marfan syndrome), or acute myocardial infarction. The resulting murmur does not resolve with diuretics and vasodilators; surgical repair is typically required.

(Choice E) Thickened and deformed mitral valve cusps can occur in rheumatic heart disease and are a potential cause of primary MR. Such a

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secondary MR by favoring relatively lower-resistance regurgitant flow.

Treatment with diuretics to reduce LVEDV and vasodilators to reduce blood pressure can lead to resolution of heart failure-induced MR.

(Choice A) Calcification of the mitral valve annulus may occur with aging, and can be accelerated by hypertension or advanced kidney disease. It is usually an incidental finding that does not affect mitral valve function.

(Choice B) Increased flow velocity through the aortic valve occurs with aortic valve stenosis. The resulting murmur is crescendo-decrescendo and best heard at the right second intercostal space (rather than holosystolic and best heard at the apex).

(Choice C) Chordae tendineae rupture is a potential cause of primary MR that can occur in the setting of bacterial endocarditis, connective tissue disease (eg, Marfan syndrome), or acute myocardial infarction. The resulting murmur does not resolve with diuretics and vasodilators; surgical repair is typically required.

(Choice E) Thickened and deformed mitral valve cusps can occur in rheumatic heart disease and are a potential cause of primary MR. Such a murmur does not resolve with treatment of decompensated heart failure. In addition, mitral stenosis (evidenced by an opening snap and mid-diastolic rumble) is more likely than MR to occur with rheumatic heart disease.

Educational objective:

Decompensated heart failure is a common cause of secondary (functional) mitral valve regurgitation. Increased left ventricular end-diastolic volume causes dilation of the mitral valve annulus and restricted movement of the chordae tendineae with subsequent regurgitation. Treatment with diuretics and vasodilators can improve heart failure-induced MR.

References

- Functional mitral regurgitation.

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A 75-year-old man is brought to the emergency department by paramedics following a high-speed motor vehicle accident. Several attempts are made to resuscitate him but are unsuccessful. The patient had no significant past medical history and took no medications. He was known to be in good health and tolerant of moderate levels of physical activity. Electrocardiogram findings at his last office visit showed no abnormalities. At autopsy, heavy calcifications of the aortic valve are seen. Which of the following most likely preceded the aortic valve changes observed in this patient?

☐

A. Cell necrosis

☐

B. Extracellular amyloid deposition

☐

C. Hypercalcemia

☐

D. Intracellular hemosiderin accumulation

☐

E. Pathologic cell hypertrophy

Submit

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Text Zoom

A 75-year-old man is brought to the emergency department by paramedics following a high-speed motor vehicle accident. Several attempts are made to resuscitate him but are unsuccessful. The patient had no significant past medical history and took no medications. He was known to be in good health and tolerant of moderate levels of physical activity. Electrocardiogram findings at his last office visit showed no abnormalities. At autopsy, heavy calcifications of the aortic valve are seen. Which of the following most likely preceded the aortic valve changes observed in this patient?

Omitted

Correct answer
A

35%

Answered correctly

3 Seconds

Time Spent

01/20/2019

Last Updated

Explanation

This patient's aortic valve calcifications most likely represent **dystrophic calcification** affecting an aging aortic valve. Dystrophic calcification is considered a hallmark of cell injury and death, occurring in all types of **necrosis** (eg, coagulative, fat, caseous, liquefactive) in the setting of **normal calcium levels**. Grossly, dystrophic calcium deposits are seen as fine, gritty, white granules or clumps. On hematoxylin and eosin

Block Time Remaining: 00:12:04

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2/7/2019



Explanation

This patient's aortic valve calcifications most likely represent **dystrophic calcification** affecting an aging aortic valve. Dystrophic calcification is considered a hallmark of cell injury and death, occurring in all types of **necrosis** (eg, coagulative, fat, caseous, liquefactive) in the setting of **normal calcium levels**. Grossly, dystrophic calcium deposits are seen as fine, gritty, white granules or clumps. On hematoxylin and eosin staining, these deposits typically appear as dark-purple, sharp-edged aggregates. Deposits that develop lamellated outer layers are described as **psammoma bodies**.

Dystrophic calcification in aged (or damaged) cardiac valves is thought to be the result of endothelial and fibroblast death secondary to **chronic hemodynamic stress** (can be accentuated by valvular abnormalities) or atherosclerotic inflammation. Subsequent release of cellular degradation products into the valvular interstitium then promotes calcification and thickening of the valve leaflets and annulus. These changes are often benign in elderly adults (**aortic sclerosis**); however, over time, progressive valvular stiffening can lead to outflow obstruction (**calcific aortic stenosis**).

(Choice B) Extracellular amyloid deposition occurs in patients with amyloidosis. Amyloidosis can cause restrictive cardiomyopathy and heart failure, but it is not directly associated with calcification of the aortic valve.

(Choice C) Hypercalcemia is associated with metastatic calcification affecting normal tissues and organs. In this process, calcium deposition typically occurs in more alkaline tissues involved in acid excretion, such as kidneys, lungs, systemic arteries, and gastric mucosa.

(Choice D) Intracellular hemosiderin accumulation is common in patients who have hemolytic anemia or who undergo frequent blood transfusions. It is not directly associated with calcification of the aortic valve.

(Choice E) Pathologic cell hypertrophy of left ventricular cardiomyocytes (ie, left ventricular hypertrophy) can occur as the result of severe aortic stenosis.

Educational objective:

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Dystrophic calcification in aged (or damaged) cardiac valves is thought to be the result of endothelial and fibroblast death secondary to **chronic hemodynamic stress** (can be accentuated by valvular abnormalities) or atherosclerotic inflammation. Subsequent release of cellular degradation products into the valvular interstitium then promotes calcification and thickening of the valve leaflets and annulus. These changes are often benign in elderly adults (**aortic sclerosis**); however, over time, progressive valvular stiffening can lead to outflow obstruction (**calcific aortic stenosis**).

(Choice B) Extracellular amyloid deposition occurs in patients with amyloidosis. Amyloidosis can cause restrictive cardiomyopathy and heart failure, but it is not directly associated with calcification of the aortic valve.

(Choice C) Hypercalcemia is associated with metastatic calcification affecting normal tissues and organs. In this process, calcium deposition typically occurs in more alkaline tissues involved in acid excretion, such as kidneys, lungs, systemic arteries, and gastric mucosa.

(Choice D) Intracellular hemosiderin accumulation is common in patients who have hemolytic anemia or who undergo frequent blood transfusions. It is not directly associated with calcification of the aortic valve.

(Choice E) Pathologic cell hypertrophy of left ventricular cardiomyocytes (ie, left ventricular hypertrophy) can occur as the result of severe aortic stenosis.

Educational objective:

Dystrophic calcification occurs in damaged or necrotic tissue in the setting of normal calcium levels; metastatic calcification occurs in normal tissue in the setting of hypercalcemia.

References

- Pathogenetic mechanism of senile calcific aortic stenosis: the role of apoptosis.

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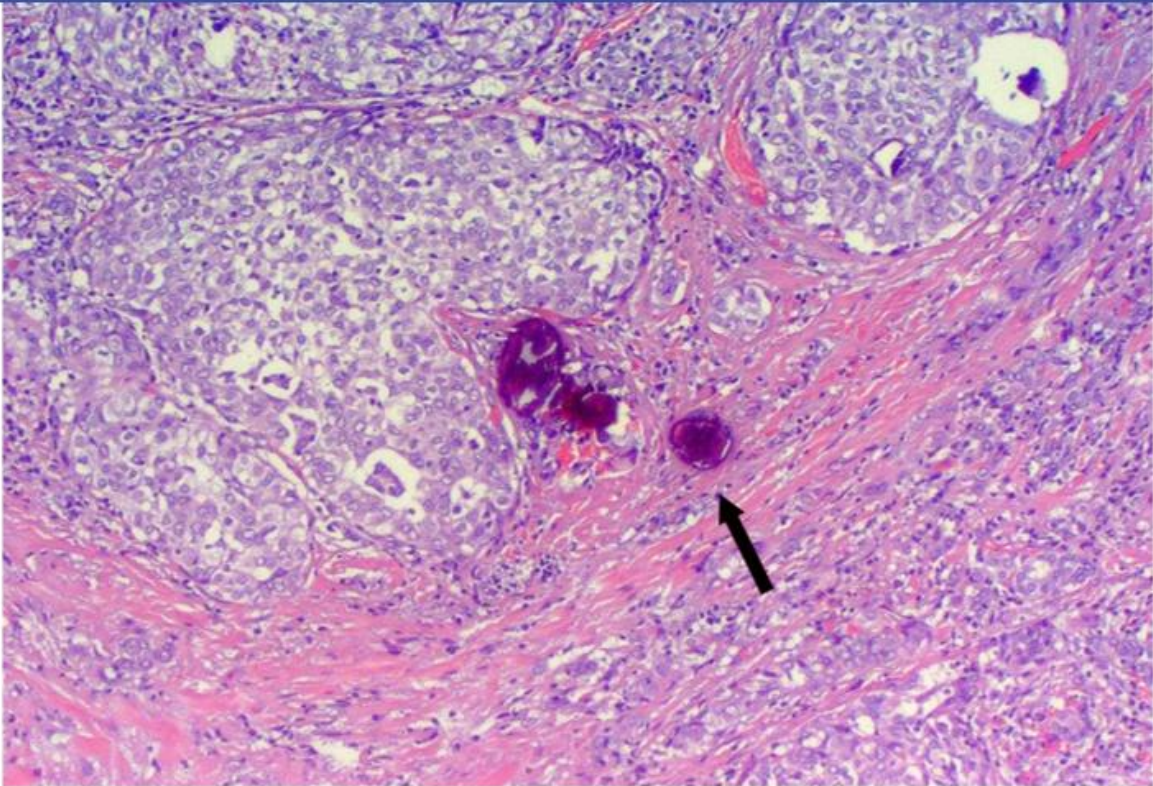
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Exhibit Display



Zoom In

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Educational objective:

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2/7/2019



This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 71-year-old man comes to the office with a 2-month history of progressive exertional dyspnea. The patient is unable to walk half a block without resting and can no longer climb stairs without stopping every few steps. He also has had difficulty sleeping due to repeatedly waking up at night feeling like he is suffocating. Physical examination shows bilateral lower extremity edema and distended neck veins. Cardiac auscultation findings over the apex are given below. This patient's auscultatory findings most likely reflect which of the following?

Play Media

- ☐ A. Aortic valve sclerosis
- ☐ B. Bulging of the interventricular septum
- ☐ C. Dynamic left ventricular outflow obstruction
- ☐ D. Elevated pulmonary arterial pressure
- ☐ E. Increased left ventricular end-systolic volume

Submit

Block Time Remaining: 00:12:07

TUTOR





This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 71-year-old man comes to the office with a 2-month history of progressive exertional dyspnea. The patient is unable to walk half a block without resting and can no longer climb stairs without stopping every few steps. He also has had difficulty sleeping due to repeatedly waking up at night feeling like he is suffocating. Physical examination shows bilateral lower extremity edema and distended neck veins. Cardiac auscultation findings over the apex are given below. This patient's auscultatory findings most likely reflect which of the following?

[Play Media](#)

- ☐ A. Aortic valve sclerosis [13%]
- ☐ B. Bulging of the interventricular septum [5%]
- ☐ C. Dynamic left ventricular outflow obstruction [10%]
- ☐ D. Elevated pulmonary arterial pressure [27%]
- ☒ E. Increased left ventricular end-systolic volume [43%]

Omitted

Correct answer
E

43%
Answered correctly

153 Seconds
Time Spent

09/26/2018
Last Updated

[Explanation](#)**Block Time Remaining: 00:14:34****TUTOR**



Explanation

Gallop heart sounds

	Features	Normal	Abnormal/associated conditions
Third heart sound (S3)	- Ventricular gallop sound (after S2) - Heard during rapid passive filling of ventricles in diastole - Sudden cessation of filling as ventricle reaches its elastic limit	- Age <40 - Pregnancy	- Systolic heart failure - Mitral regurgitation - High-output states
Fourth heart sound (S4)	- Atrial gallop sound (before S1) - Heard immediately after atrial contraction as blood is forced into a stiff ventricle	Healthy older adults	- Younger adults, children - Diastolic dysfunction (eg, LVH)

LVH = left ventricular hypertrophy.

Cardiac auscultation in this patient reveals a **low-frequency** sound occurring **after S2**, known as the **third heart sound (S3)**. Although the S3 can be normal in young individuals and pregnant patients, its presence in those age >40 is typically a sign of left ventricular (LV) **volume overload/failure** (causing exertional dyspnea and paroxysmal nocturnal dyspnea in this patient).

In early diastole, the ventricles relax and the atrioventricular valves open, allowing blood to rush in and fill the ventricles. An S3 develops with **forceful rapid passive filling** that exceeds the expansion capacity of the ventricle, leading to sudden deceleration of the entering blood column and reverberation of the ventricular walls. As a result, S3 is often heard in pathologic settings causing high ventricular filling pressures and/or volume overload, particularly aortic or mitral regurgitation and dilated cardiomyopathy.

(Choice A) Aortic valve sclerosis may produce a midsystolic ejection murmur but would not produce an extra heart sound. Progression to severe

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can be normal in young individuals and pregnant patients, its presence in those age >40 is typically a sign of left ventricular (LV) **volume overload/failure** (causing exertional dyspnea and paroxysmal nocturnal dyspnea in this patient).

In early diastole, the ventricles relax and the atrioventricular valves open, allowing blood to rush in and fill the ventricles. An S3 develops with **forceful rapid passive filling** that exceeds the expansion capacity of the ventricle, leading to sudden deceleration of the entering blood column and reverberation of the ventricular walls. As a result, S3 is often heard in pathologic settings causing high ventricular filling pressures and/or volume overload, particularly aortic or mitral regurgitation and dilated cardiomyopathy.

(Choice A) Aortic valve sclerosis may produce a midsystolic ejection murmur but would not produce an extra heart sound. Progression to severe aortic stenosis may eventually produce an S3 indicative of LV systolic failure.

(Choice B) In cardiac tamponade, accumulation of large quantities of pericardial fluid prevents the expansion of the ventricular free walls. During inspiration, venous blood return increases and the intraventricular septum bulges into the left ventricle to allow the right ventricle to accommodate the increased blood volume. The resultant decrease in LV filling volume causes pulsus paradoxus (>10 mm Hg decrease in systolic blood pressure during inspiration).

(Choice C) Hypertrophic cardiomyopathy can cause dynamic LV outflow obstruction, producing a crescendo-decrescendo ejection systolic murmur. The outflow obstruction can also cause systolic anterior motion of the mitral valve, resulting in mitral regurgitation (holosystolic murmur).

(Choice D) Elevated pulmonary arterial pressure (pulmonary hypertension) can present with exertional dyspnea and fatigue and eventual right ventricular failure (elevated jugular venous pressure, peripheral edema). However, auscultation will show increased intensity of the pulmonic closure sound (P2), and affected patients typically lack nocturnal dyspnea due to intact LV function.

Educational objective:

The third heart sound (S3) is a low-frequency sound occurring immediately after S2 that is commonly associated with increased ventricular end-systolic volume. S3 frequently occurs in the setting of mitral regurgitation and systolic heart failure (eg, dilated cardiomyopathy).

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Text Zoom

This item has associated media that may require the use of headphones. Please ensure your system/speaker volume is set to an audible level.

A 65-year-old man with a long history of hypertension and type 2 diabetes mellitus comes to the physician for a routine check-up. He is an ex-smoker with a 15 pack-year smoking history. He jogs 3 miles per day. His BMI is 28.5 kg/m². His family history is unremarkable. Cardiac auscultation findings over the apex are given below. The auscultatory findings are best explained by which of the following?

Play Media

A. Aortic root dilation

B. Atrial septal defect

C. Left ventricular hypertrophy

D. Mitral valve prolapse

E. Pericardial effusion

Submit

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TUTOR

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Windows Taskbar



Explanation

The auscultatory findings in this case demonstrate an extra low frequency heart sound at the end of diastole just before S1 known as S4. This fourth heart sound is often heard in older adults due to an age-related decrease in left ventricular compliance, and in such cases is a relatively benign finding. However, the louder the sound, the more likely it is due to a pathologically stiff left ventricle, which occurs in restrictive cardiomyopathy and in left ventricular hypertrophy following prolonged hypertension (likely the cause in this case). Unlike the S3 sound, auscultation of an S4 in younger patients is always pathologic. It is thought that the S4 coincides with atrial contraction and is produced by the resultant onrush of blood striking a stiff left ventricle.

(Choice A) Aortic root dilation can be associated with aortic regurgitation, which would result in an early diastolic murmur as opposed to an extra heart sound.

(Choice B) An atrial septal defect produces fixed splitting of the second heart sound. This can be distinguished from an S4, which occurs just before S1.

(Choice D) Mitral valve prolapse is classically associated with a mid-systolic click and a mid-to-late systolic murmur.

(Choice E) Physical examination signs of a pericardial effusion are neither sensitive nor specific, and a S4 is not an associated finding.

Educational objective:

The fourth heart sound (S4) is a low frequency sound heard at the end of diastole just before S1. It is due to decreased left ventricular compliance and is often associated with restrictive cardiomyopathy and left ventricular hypertrophy.

References

- Association of the fourth heart sound with increased left ventricular end-diastolic stiffness.
- Correlation of an audible fourth heart sound with level of diastolic dysfunction.

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A 65-year-old Caucasian male presents to the ER with sudden onset of right-sided calf and foot pain. His past medical history is significant for hypertension, type II diabetes mellitus, atrial fibrillation and stable angina. Physical examination reveals paleness of the right leg and diminished right popliteal pulse. Immediate angiography is ordered that reveals an obstructive thrombus in the right common femoral artery. The thrombus extraction is followed by a rapid surge of serum creatine kinase level, which is best explained by:

☐ A. Cellular swelling

☐ B. Cell membrane damage

☐ C. Glutathione peroxidase production

☐ D. Mitochondrial vacuolization

☐ E. Nuclear shrinkage

Submit

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1:57 PM

2/7/2019

2

A 65-year-old Caucasian male presents to the ER with sudden onset of right-sided calf and foot pain. His past medical history is significant for hypertension, type II diabetes mellitus, atrial fibrillation and stable angina. Physical examination reveals paleness of the right leg and diminished right popliteal pulse. Immediate angiography is ordered that reveals an obstructive thrombus in the right common femoral artery. The thrombus extraction is followed by a rapid surge of serum creatine kinase level, which is best explained by:

- ☐ A. Cellular swelling [6%]
- ☒ B. Cell membrane damage [70%]
- ☐ C. Glutathione peroxidase production [10%]
- ☐ D. Mitochondrial vacuolization [10%]
- ☐ E. Nuclear shrinkage [1%]

Correct answer
B

70%
Answered correctly

 3 Seconds
Time Spent

02/06/2019
Last Updated

Explanation

Ischemia is characterized by the reduction of blood flow, usually as a result of mechanical obstruction within the arterial system (eg, thrombus). If the flow of blood to the ischemic tissue is restored in a timely manner, those cells that were reversibly injured will typically recover. Sometimes, however, the cells within the damaged tissue will paradoxically die at an accelerated pace through apoptosis or necrosis after resumption of blood

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Explanation

Ischemia is characterized by the reduction of blood flow, usually as a result of mechanical obstruction within the arterial system (eg, thrombus). If the flow of blood to the ischemic tissue is restored in a timely manner, those cells that were reversibly injured will typically recover. Sometimes, however, the cells within the damaged tissue will paradoxically die at an accelerated pace through apoptosis or necrosis after resumption of blood flow. This process is termed reperfusion injury, and is thought to occur secondary to one or more of the following mechanisms: 1) oxygen free radical generation by parenchymal cells, endothelial cells, and leukocytes; 2) severe, irreversible mitochondrial damage described as "mitochondrial permeability transition"; 3) inflammation, which attracts circulating neutrophils that cause additional injury; and 4) activation of the complement pathway, causing cell injury and further inflammation. When the cells within heart, brain, or skeletal muscle are injured, the enzyme creatine kinase leaks across the damaged cell membrane and into circulation (as seen in this patient).

(Choice A) Cellular swelling arises secondary to changes in ion concentration and the influx of water. This state is considered a hallmark of reversible injury, and is not directly associated with the leakage of intracellular proteins such as creatine kinase.

(Choice C) Glutathione peroxidase actually reduces cellular injury by catalyzing free radical breakdown. The presence of this enzyme is not responsible for the release of creatine kinase.

(Choice D) Mitochondrial vacuolization reduces the cellular capacity for ATP generation and is associated with irreversible injury. Creatine kinase release is not directly associated with this mitochondrial change, however.

(Choice E) Nuclear shrinkage (pyknosis), fragmentation, and dissolution characterize irreversible injury of the cell. Creatine kinase release is not directly associated with such nuclear changes, however.

Educational Objective:

Reperfusion injury is thought to occur secondary to oxygen free radical generation, mitochondrial damage, and inflammation.

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A 16-year-old previously healthy boy is brought to the office due to palpitations. He was watching a documentary at a movie theater when his heart suddenly began to race and he became lightheaded. The patient says that he has felt "rapid heartbeats" in the past and thought that they were due to stress from school exams, but he has never had associated lightheadedness before. He has no chest pain or dyspnea. Physical examination is within normal limits. ECG strip is shown in the exhibit. Which of the following is most likely to be seen in this patient?

☐

A. Atrioventricular conduction tract bypassing the atrioventricular node

☐

B. Cardiac myocyte degeneration below the level of the atrioventricular node

☐

C. Ectopic atrial foci that serve as the dominant pacemaker

☐

D. Prolonged repolarization due to myocyte ion channel abnormality

☐

E. Rapid electrical impulses originating in the pulmonary veins

Submit

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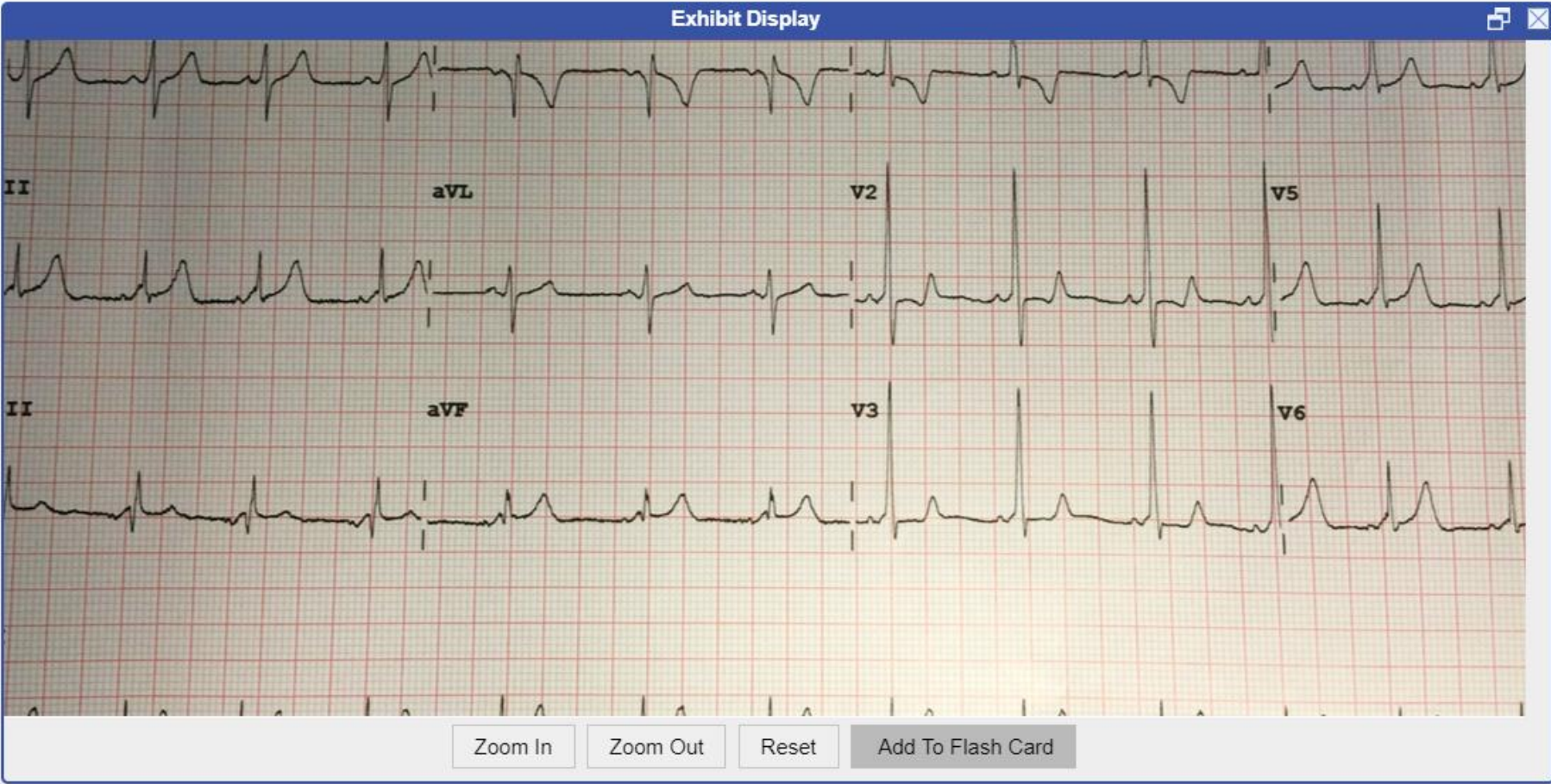
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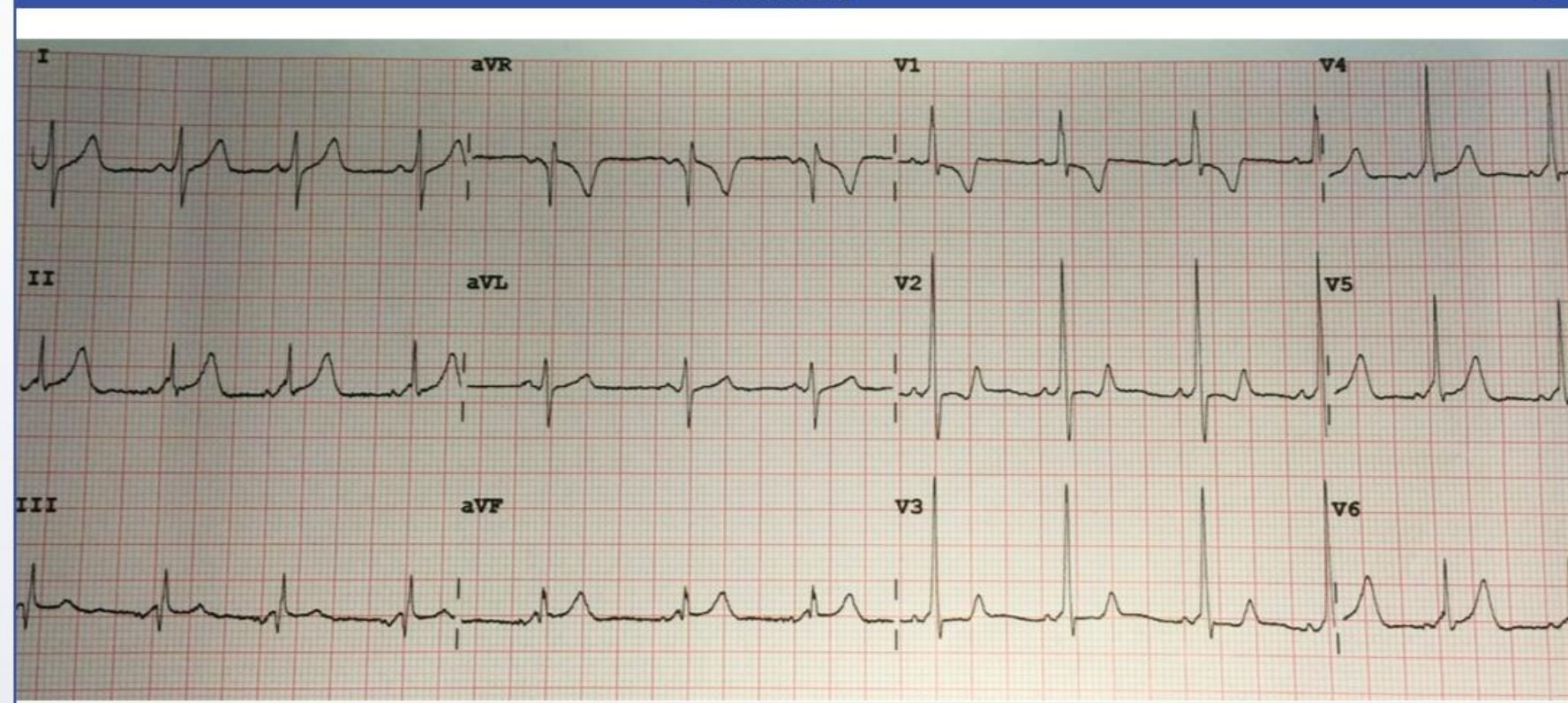
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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, Calendar, Photos, and other background applications.

System tray showing date and time: 1:57 PM 2/7/2019.

Exhibit Display



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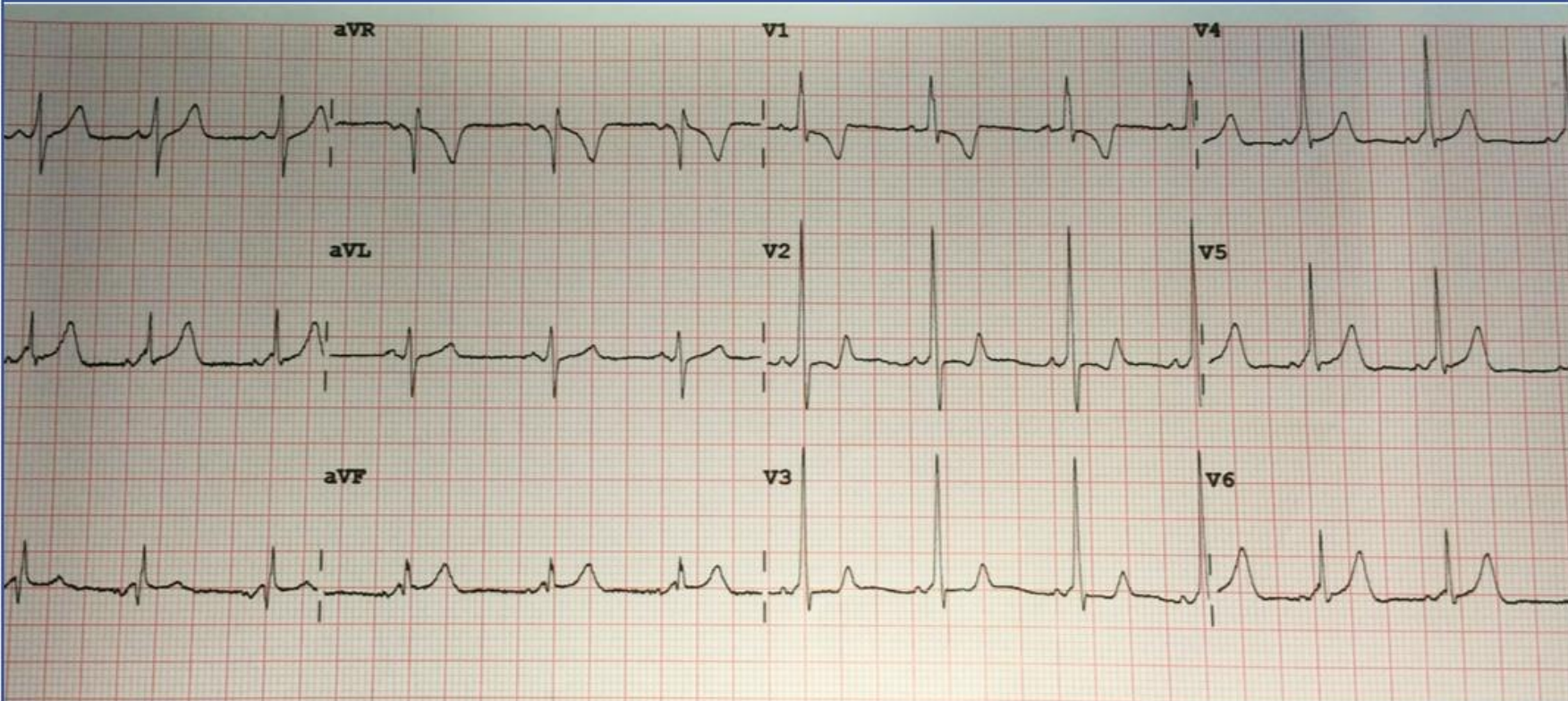
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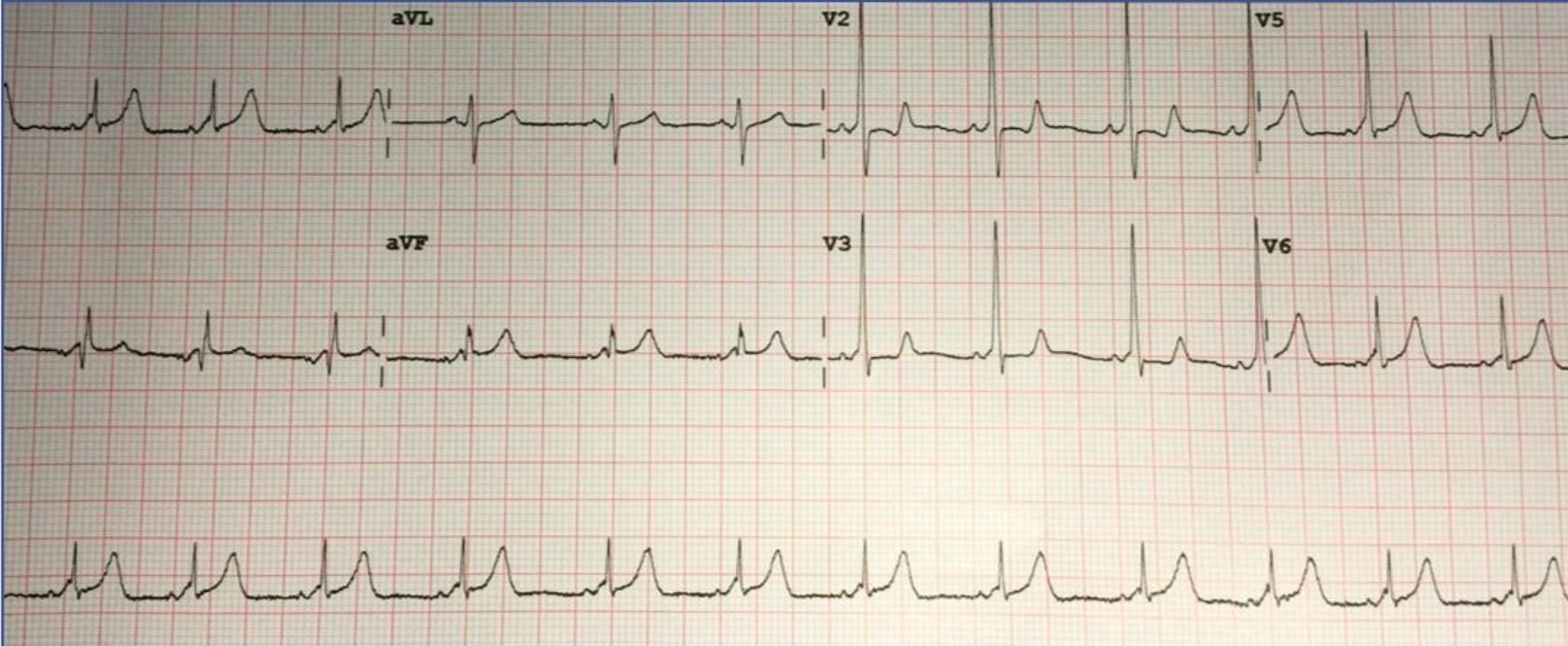
Notes

Calculator

Reverse Color

Text Zoom

Exhibit Display



Zoom In

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TUTOR

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1:58 PM

2/7/2019

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A

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Exhibit Display

ECG tracing showing leads II, aVL, V2, V5, III, aVF, V3, V6, and a repeat of II. The tracing shows a regular rhythm with a heart rate of approximately 75 bpm. The QRS complex is narrow and the T waves are upright in all leads.

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Windows Taskbar

System Tray



A 16-year-old previously healthy boy is brought to the office due to palpitations. He was watching a documentary at a movie theater when his heart suddenly began to race and he became lightheaded. The patient says that he has felt "rapid heartbeats" in the past and thought that they were due to stress from school exams, but he has never had associated lightheadedness before. He has no chest pain or dyspnea. Physical examination is within normal limits. ECG strip is shown in the [exhibit](#). Which of the following is most likely to be seen in this patient?

- ☒ A. Atrioventricular conduction tract bypassing the atrioventricular node [72%]
- ☐ B. Cardiac myocyte degeneration below the level of the atrioventricular node [1%]
- ☐ C. Ectopic atrial foci that serve as the dominant pacemaker [7%]
- ☐ D. Prolonged repolarization due to myocyte ion channel abnormality [15%]
- ☐ E. Rapid electrical impulses originating in the pulmonary veins [1%]

Omitted

Correct answer

A

72%
Answered correctly55 Seconds
Time Spent11/12/2018
Last Updated

Explanation

This patient's [ECG](#) demonstrates **Wolff-Parkinson-White (WPW) pattern**. The pattern is caused by an **accessory conduction pathway** (also known as an accessory bypass tract or a bundle of Kent) that directly connects the atria to the ventricles and allows electrical impulses to bypass the atrioventricular node, causing **preexcitation** (early excitation) of the ventricles. This preexcitation creates the characteristic **triad** of ECG

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the atrioventricular node, causing **preexcitation** (early excitation) of the ventricles. This preexcitation creates the characteristic **triad** of ECG findings that constitutes WPW pattern:

- Shortened PR interval
- Widening of the QRS interval
- Slurred and broad initial upstroke of the QRS complex, referred to as a **delta wave**

Many patients with WPW pattern on ECG are asymptomatic; however, some develop symptomatic cardiac arrhythmias. **WPW syndrome** is the combination of WPW pattern on ECG and the presence of symptomatic arrhythmia. **Atrioventricular reentrant tachycardia (AVRT)**, a type of paroxysmal supraventricular tachycardia that occurs due to reentry of electrical impulses through the accessory conduction pathway, is the most common associated arrhythmia and can manifest with intermittent palpitations, sensation of a racing heart, and lightheadedness or syncope.

(Choice B) Cardiac myocyte degeneration below the level of the atrioventricular node leads to variable degrees of conduction block and presents with slow heart rate.

(Choice C) Patients with wandering atrial pacemaker or ectopic atrial rhythm have ectopic atrial foci that replace the sinus node as the dominant pacemaker; P waves of varying morphology are seen on ECG. This patient has normal, uniform P waves suggesting normal sinus node function.

(Choice D) Long QT syndrome (LQTS) is a disorder of ventricular repolarization characterized by a prolonged QT interval on ECG. Congenital LQTS is due to a mutation in the genes encoding for cardiac sodium or potassium channels.

(Choice E) Atrial fibrillation is most often triggered by rapid electrical impulses originating in the pulmonary veins, and is characterized by an irregularly irregular rhythm and the absence of P waves.

Educational objective:

Wolff-Parkinson-White (WPW) pattern is characterized by a shortened PR interval, widening of the QRS complex, and slurred initial upstroke of the QRS complex (delta wave). It is caused by an accessory pathway that bypasses the atrioventricular node, causing preexcitation of the ventricles. Patients with WPW pattern can develop symptomatic arrhythmia (eg, atrioventricular reentrant tachycardia) due to reentry of electrical impulses through the accessory conduction pathway.

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- Shortened PR interval
- Widening of the QRS interval
- Slurred and broad initial upstroke of the QRS complex, referred to as a **delta wave**

Many patients with WPW pattern on ECG are asymptomatic; however, some develop symptomatic cardiac arrhythmias. **WPW syndrome** is the combination of WPW pattern on ECG and the presence of symptomatic arrhythmia. **Atrioventricular reentrant tachycardia (AVRT)**, a type of paroxysmal supraventricular tachycardia that occurs due to reentry of electrical impulses through the accessory conduction pathway, is the most common associated arrhythmia and can manifest with intermittent palpitations, sensation of a racing heart, and lightheadedness or syncope.

(Choice B) Cardiac myocyte degeneration below the level of the atrioventricular node leads to variable degrees of conduction block and presents with slow heart rate.

(Choice C) Patients with wandering atrial pacemaker or ectopic atrial rhythm have ectopic atrial foci that replace the sinus node as the dominant pacemaker; P waves of varying morphology are seen on ECG. This patient has normal, uniform P waves suggesting normal sinus node function.

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(Choice E) Atrial fibrillation is most often triggered by rapid electrical impulses originating in the pulmonary veins, and is characterized by an irregularly irregular rhythm and the absence of P waves.

Educational objective:

Wolff-Parkinson-White (WPW) pattern is characterized by a shortened PR interval, widening of the QRS complex, and slurred initial upstroke of the QRS complex (delta wave). It is caused by an accessory pathway that bypasses the atrioventricular node, causing preexcitation of the ventricles. Patients with WPW pattern can develop symptomatic arrhythmia (eg, atrioventricular reentrant tachycardia) due to reentry of electrical impulses through the accessory conduction pathway.

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End Block



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Lab Values



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Calculator



Reverse Color



Text Zoom



Correct answer

A



12%

Answered correctly



55 Seconds

Time Spent



11/12/2018

Last Updated

Explanation

This patient's **ECG** demonstrates **Wolff-Parkinson-White (WPW) pattern**. The pattern is caused by an **accessory conduction pathway** (also known as an accessory bypass tract or a bundle of Kent) that directly connects the atria to the ventricles and allows electrical impulses to bypass the atrioventricular node, causing **preexcitation** (early excitation) of the ventricles. This preexcitation creates the characteristic **triad** of ECG findings that constitutes WPW pattern:

- Shortened PR interval
- Widening of the QRS interval
- Slurred and broad initial upstroke of the QRS complex, referred to as a **delta wave**

Many patients with WPW pattern on ECG are asymptomatic; however, some develop symptomatic cardiac arrhythmias. **WPW syndrome** is the combination of WPW pattern on ECG and the presence of symptomatic arrhythmia. **Atrioventricular reentrant tachycardia (AVRT)**, a type of paroxysmal supraventricular tachycardia that occurs due to reentry of electrical impulses through the accessory conduction pathway, is the most common associated arrhythmia and can manifest with intermittent palpitations, sensation of a racing heart, and lightheadedness or syncope.

(Choice B) Cardiac myocyte degeneration below the level of the atrioventricular node leads to variable degrees of conduction block and presents with slow heart rate.

(Choice C) Patients with wandering atrial pacemaker or ectopic atrial rhythm have ectopic atrial foci that replace the sinus node as the dominant pacemaker; P waves of varying morphology are seen on ECG. This patient has normal, uniform P waves suggesting normal sinus node function.

(Choice D) Long QT syndrome (LQTS) is a disorder of ventricular repolarization characterized by a prolonged QT interval on ECG. Congenital LQTS is due to a mutation in the genes encoding for cardiac sodium or potassium channels.

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TUTOR



Feedback



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Reverse Color



Text Zoom



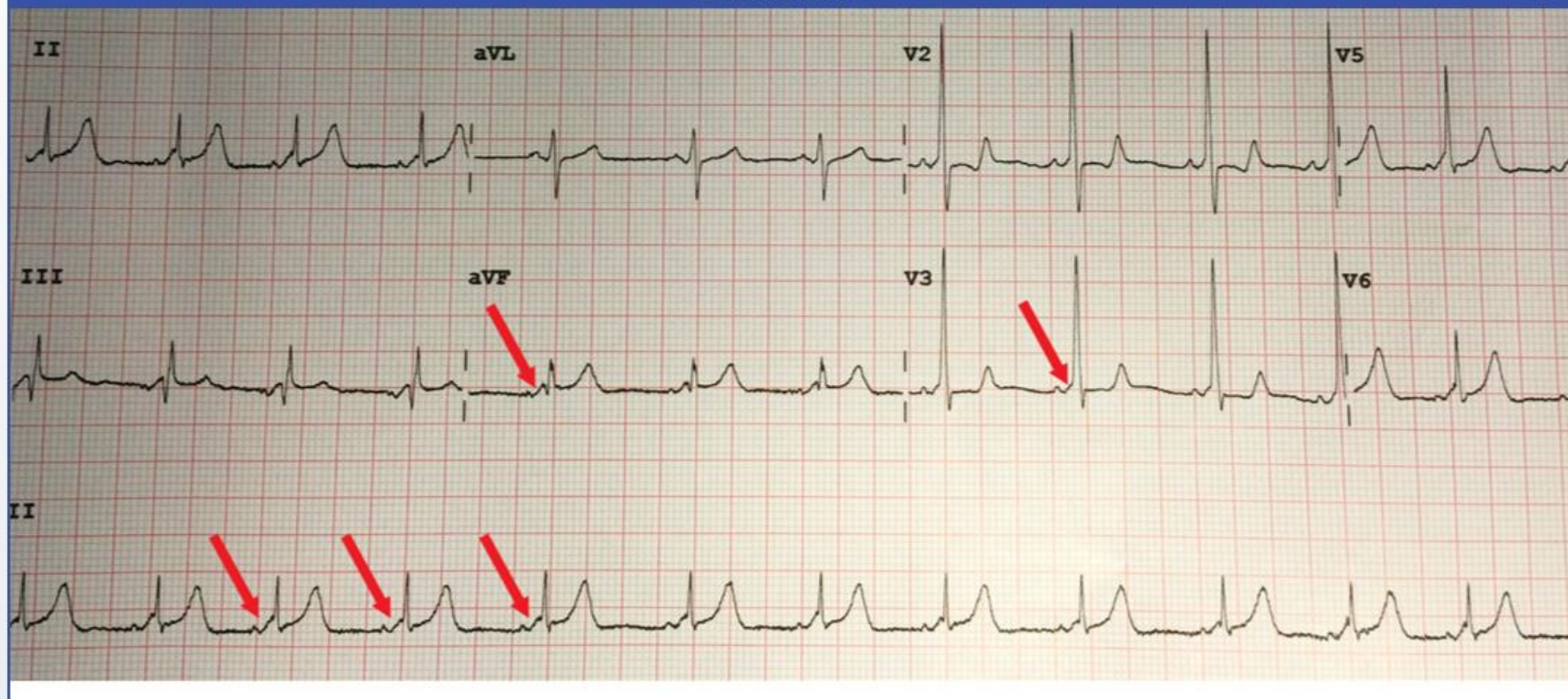
Correct answer

1/2%

55 Seconds

11/12/2018

Exhibit Display



Zoom In

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Add To Flash Card

ECG is due to a mutation in the genes encoding for cardiac sodium or potassium channels.

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Feedback



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55 Seconds

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Exhibit Display

Wolff-Parkinson-White triad

delta wave

Shortened PR interval

Widened QRS

Zoom In Zoom Out Reset Add To Flash Card

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TUTOR

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Suspend

End Block

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Tutorial

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Text Zoom

A 63-year-old man comes to the office with exertional dyspnea that has been progressing over the last 3 weeks. He sleeps in a recliner with his head elevated as he gets short of breath when laying flat in bed. His past medical history includes gout, dyslipidemia, and hypertension. On examination, blood pressure is 154/89 mm Hg and pulse is 85/min. Distended jugular veins are seen in the semirecumbent position. An apical heave is found on cardiac examination. Pitting edema is evident in the bilateral lower extremities. Levels of which of the following substances will be higher in the pulmonary vein compared to the pulmonary artery in this patient?

A. Aldosterone

B. Angiotensin I

C. Angiotensin II

D. Angiotensinase

E. Angiotensinogen

F. Arginine vasopressin

G. Prostaglandin E1

H. Renin

Submit

Block Time Remaining: 00:18:10

TUTOR

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II

Suspend

End Block

Windows Taskbar



A 63-year-old man comes to the office with exertional dyspnea that has been progressing over the last 3 weeks. He sleeps in a recliner with his head elevated as he gets short of breath when laying flat in bed. His past medical history includes gout, dyslipidemia, and hypertension. On examination, blood pressure is 154/89 mm Hg and pulse is 85/min. Distended jugular veins are seen in the semirecumbent position. An apical heave is found on cardiac examination. Pitting edema is evident in the bilateral lower extremities. Levels of which of the following substances will be higher in the pulmonary vein compared to the pulmonary artery in this patient?

- ☐ A. Aldosterone [1%]
- ☐ B. Angiotensin I [12%]
- ☒ C. Angiotensin II [59%]
- ☐ D. Angiotensinase [3%]
- ☐ E. Angiotensinogen [5%]
- ☐ F. Arginine vasopressin [3%]
- ☐ G. Prostaglandin E1 [9%]
- ☐ H. Renin [3%]

Omitted

Correct answer
C59%
Answered correctly3 Seconds
Time Spent01/16/2019
Last Updated

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A

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Explanation

This patient's exertional dyspnea, orthopnea (dyspnea in recumbent position that is relieved on sitting/standing), jugular venous distension, cardiac heave, and peripheral edema is consistent with **congestive heart failure** (CHF). The decreased cardiac output in CHF reduces renal perfusion, stimulating renin secretion by the juxtaglomerular cells of the **juxtaglomerular apparatus** (Choice H).

Renin acts to convert angiotensinogen (produced by the liver) into angiotensin I in the systemic circulation (Choices B and E). Angiotensin I is then converted to **angiotensin II**, primarily within the small pulmonary vessels by endothelial-bound angiotensin-converting enzyme. Therefore, levels of angiotensin II are **higher in the pulmonary vein** when compared to the pulmonary artery. Angiotensin II is a potent vasoconstrictor of the systemic circulation and causes a rise in blood pressure (increased afterload) that worsens cardiac output. Angiotensin II also acts on the adrenal gland to stimulate aldosterone release, causing sodium retention and further worsening the afterload (Choice A).

(Choice D) The angiotensinases are a group of enzymes found in multiple tissues and are responsible for the breakdown of angiotensin II and other structurally similar enzymes. Their production is not known to be directly regulated by the renin-angiotensin system.

(Choice F) Arginine vasopressin (antidiuretic hormone [ADH]) is released from the posterior pituitary in response to high serum osmolality (>280 mOsm/kg H₂O). The set point for ADH release is lowered in response to low effective intravascular volume (such as with low cardiac output in CHF). Therefore, ADH levels are elevated in patients with CHF, and the resulting tubular free water reuptake can lead to the development of hyponatremia.

(Choice G) Prostaglandins are produced in multiple cell types, including endothelial cells, mast cells, and macrophages. Prostaglandin E1 is a potent vasodilator. It maintains patency of the ductus arteriosus and causes afferent arteriolar vasodilation in the kidneys.

Educational objective:

Heart failure results in stimulation of the sympathetic nervous system and the renin-angiotensin-aldosterone system in an attempt to maintain effective intravascular volume. Inactive angiotensin I is converted into active angiotensin II by endothelial-bound angiotensin-converting enzyme in the small vessels of the lungs.

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Explanation

Exhibit Display

Juxtaglomerular cells

Macula densa

Extraglomerular mesangial cells

Juxtaglomerular apparatus

Afferent arteriole

Zoom In

Zoom Out

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2:00 PM

2/7/2019

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Text Zoom

Explanation

Exhibit Display

The diagram illustrates a cross-section of a nephron. A red, coiled glomerular capillary is enclosed within a double-walled glomerular capsule. The space between the two layers of the capsule is the capsular space. The outer layer of the capsule is the parietal epithelium, and the inner layer is the glomerular capillary wall. A distal tubule, shown as a thick orange cylinder, is connected to the capsule. An efferent arteriole, a red blood vessel, is shown exiting the capsule. Labels with leader lines point to the Efferent arteriole, Distal tubule, Glomerular capillary, Glomerular capsule, Capsular space, and Parietal epithelium.

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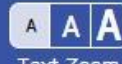
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2/7/2019



A 22-year-old man comes to the office for an exercise evaluation. He is an avid long-distance runner. One of his team members was disqualified recently due to health issues, which made him worried about his health. He has no medical history and does not take any medications or illicit drugs. His maternal grandfather died suddenly at age 40. Blood pressure is 125/75 mm Hg and pulse is 55/min. Transthoracic echocardiogram results are as follows:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	small
LV septal thickness	increased
LV posterior wall thickness	normal
LV mass	increased
LV ejection fraction	74%

The right ventricle appears normal and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- ☐ A. Anabolic steroid abuse
- ☐ B. Cardiac adaptation
- ☐ C. Hypertrophic cardiomyopathy
- ☐ D. Premature coronary artery disease
- ☐ E. Subclinical viral myocarditis





recently due to health issues, which made him worried about his health. He has no medical history and does not take any medications or illicit drugs. His maternal grandfather died suddenly at age 40. Blood pressure is 125/75 mm Hg and pulse is 55/min. Transthoracic echocardiogram results are as follows:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	small
LV septal thickness	increased
LV posterior wall thickness	normal
LV mass	increased
LV ejection fraction	74%

The right ventricle appears normal and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- ☐ A. Anabolic steroid abuse [1%]
- ☐ B. Cardiac adaptation [16%]
- ☒ C. Hypertrophic cardiomyopathy [81%]
- ☐ D. Premature coronary artery disease [0%]
- ☐ E. Subclinical viral myocarditis [0%]

Omitted

Correct answer

81%
Answered correctly5 Seconds
Time Spent12/02/2018
Last Updated

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Explanation

This patient most likely has **hypertrophic cardiomyopathy** (HCM). Recognition of HCM is important because it is one of the most common causes of sudden cardiac death (SCD) in young adults. Although patients may experience exertional dyspnea, chest pain, lightheadedness, or syncope, they are often asymptomatic. The condition is typically inherited in an autosomal dominant pattern, and patients will often have a **family history** of HCM or unexplained SCD.

Echocardiographic findings consistent with HCM include:

- Overall increase in left ventricular (LV) mass
- **Reduced LV cavity size** (leading to impaired diastolic function)
- **Asymmetric** increase in **LV wall thickness** predominantly affecting the **septum**
- Normal or **increased LV ejection fraction**
- Left atrial enlargement (secondary to increased LV end-diastolic pressure)

Another characteristic of HCM is a **poorly developed coronary capillary network** in hypertrophied regions with evidence of chronic ischemia (ie, fibrosis, scarring).

(Choice A) Anabolic steroid abuse can cause pathologic concentric LV hypertrophy, characterized by reduced diastolic function and poor coronary vascularization. However, uniform ventricular wall thickening would be expected.

(Choice B) Athlete's heart involves physiologic cardiac adaptations that improve cardiac function in response to high-level endurance training. There is typically predominant eccentric hypertrophy with enlarged LV cavity size and a small degree of concentric hypertrophy with uniformly increased LV wall thickness. The changes of athlete's heart must be differentiated from those associated with pathologic disease; this patient's reduced LV cavity size and localized septal wall thickening suggest underlying pathology due to HCM.

(Choice D) Severe coronary artery disease can lead to ischemic cardiomyopathy, which typically manifests as a dilated cardiomyopathy with an enlarged LV cavity and thin LV walls with impaired systolic function.

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- 21 Another characteristic of HCM is a **poorly developed coronary capillary network** in hypertrophied regions with evidence of chronic ischemia (ie, fibrosis, scarring).

23 (Choice A) Anabolic steroid abuse can cause pathologic concentric LV hypertrophy, characterized by reduced diastolic function and poor coronary vascularization. However, uniform ventricular wall thickening would be expected.

• 25 **(Choice B)** Athlete's heart involves physiologic cardiac adaptations that improve cardiac function in response to high-level endurance training.

• 26 There is typically predominant eccentric hypertrophy with enlarged LV cavity size and a small degree of concentric hypertrophy with uniformly

• 27 increased LV wall thickness. The changes of athlete's heart must be differentiated from those associated with pathologic disease; this patient's

• 28 reduced LV cavity size and localized septal wall thickening suggest underlying pathology due to HCM.

• 29 **(Choice D)** Severe coronary artery disease can lead to ischemic cardiomyopathy, which typically manifests as a dilated cardiomyopathy with an
• 30 enlarged LV cavity and thin LV walls with impaired systolic function.

(Choice E) Viral myocarditis may sometimes lead to dilated cardiomyopathy with eccentric hypertrophy and impaired LV systolic function. Subclinical disease is typically quite mild and resolves without sequelae.

34 Educational objective:

35 Patients with hypertrophic cardiomyopathy (HCM) may be asymptomatic and often have a family history of HCM or sudden cardiac death.

Findings consistent with HCM include an overall increase in left ventricular (LV) mass, reduced LV cavity size with impaired diastolic function, LV hypertrophy predominantly affecting the septum, and normal or increased LV ejection fraction.

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• 39 **Block Time Remaining: 00:18:17**

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A

Text Zoom

A 66-year-old man with diabetes mellitus is brought to the hospital due to sudden-onset chest pain and nausea. Blood pressure is 70/60 mm Hg and pulse is 60/min. Lungs are clear on auscultation. ECG shows ST-segment elevation in leads II, III, and aVF. Chest x-ray is unremarkable. The patient is diagnosed with an inferior wall myocardial infarction. Emergent cardiac catheterization reveals complete occlusion of the proximal right coronary artery. He is persistently hypotensive in the cardiac catheterization laboratory. Which of the following hemodynamic findings is most likely to be observed in this patient?

	Cardiac output	Pulmonary capillary wedge pressure	Central venous pressure
☐ A.	↓	↓	↑
☐ B.	↓	↑	↑
☐ C.	↑	↑	↓
☐ D.	↓	↑	↓
☐ E.	↑	↓	↓

Submit

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TUTOR

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Feedback

⏏

Suspend

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End Block

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Edge

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Shopping

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System Tray

2:00 PM

2/7/2019

2



A 66-year-old man with diabetes mellitus is brought to the hospital due to sudden-onset chest pain and nausea. Blood pressure is 70/60 mm Hg and pulse is 60/min. Lungs are clear on auscultation. ECG shows ST-segment elevation in leads II, III, and aVF. Chest x-ray is unremarkable. The patient is diagnosed with an inferior wall myocardial infarction. Emergent cardiac catheterization reveals complete occlusion of the proximal right coronary artery. He is persistently hypotensive in the cardiac catheterization laboratory. Which of the following hemodynamic findings is most likely to be observed in this patient?

	Cardiac output	Pulmonary capillary wedge pressure	Central venous pressure
☒ A. [39%]	↓	↓	↑
☐ B. [40%]	↓	↑	↑
☐ C. [1%]	↑	↑	↓
☐ D. [17%]	↓	↑	↓
☐ E. [1%]	↑	↓	↓

Omitted

Correct answer

A

39%
Answered correctly3 Seconds
Time Spent08/28/2018
Last Updated

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Explanation

This patient is in cardiogenic shock after suffering an **inferior wall myocardial infarction**. The inferior wall of the left ventricle (LV) is supplied by the posterior descending artery, which arises off the dominant **right coronary artery**. Because the right coronary artery also gives off marginal branches that supply most of the right ventricle (RV), inferior wall myocardial infarction is often associated with **right ventricular infarction**. Right-sided heart failure typically presents with hypotension and distended jugular veins, but the lungs are clear unless significant left-sided heart failure is also present. This patient's **lack of crackles** on lung auscultation and **absence of pulmonary edema** on chest x-ray is indicative of **isolated RV dysfunction**.

Right ventricular infarction decreases RV stroke volume, which in turn leads to **diminished** LV filling and **cardiac output** despite preserved LV systolic function, resulting in hypotension and shock. Because left-sided filling pressures are reduced, **pulmonary capillary wedge pressure** (PCWP) also **decreases** as it reflects left atrial pressure. In addition, patients have **elevated central venous pressure** due to impaired forward flow and backup of blood into the systemic venous system.

(Choices B and D) LV anterior wall infarcts (eg, due to left anterior descending artery occlusion) are more likely than inferior wall infarcts to cause LV systolic dysfunction, resulting in decreased cardiac output, elevated left-sided filling pressures (eg, increased PCWP), and cardiogenic pulmonary edema. Initially, central venous pressure may be normal; however, persistently high left-sided pressures may lead to right-sided heart failure, resulting in elevated central venous pressure.

(Choice E) Increased cardiac output with decreased PCWP and central venous pressure is characteristic of distributive shock due to sepsis.

Educational objective:

The dominant right coronary artery perfuses both the inferior wall of the left ventricle and the majority of the right ventricle. Proximal occlusion can cause right ventricular myocardial infarction, which presents with hypotension (reduced cardiac output) and distended jugular veins (elevated central venous pressures). The lungs will be clear on auscultation and x-ray (lack of pulmonary edema) unless concomitant left-sided heart failure is also present.

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TUTOR

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Suspend

End Block

Windows Taskbar

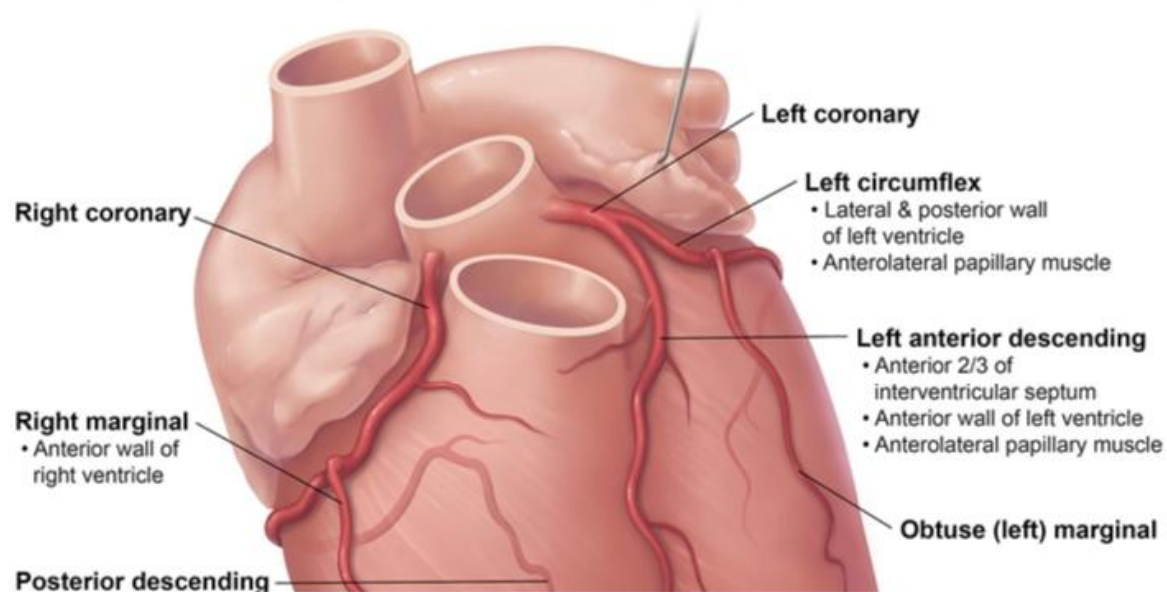
System Tray



Explanation

Exhibit Display

Right dominant coronary artery circulation



Zoom In

Zoom Out

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Block Time Remaining: 00:18:20

TUTOR





Explanation

Exhibit Display

Right coronary

- Lateral & posterior wall of left ventricle
- Anterolateral papillary muscle

Right marginal

- Anterior wall of right ventricle

Left anterior descending

- Anterior 2/3 of interventricular septum
- Anterior wall of left ventricle
- Anterolateral papillary muscle

Obtuse (left) marginal**Posterior descending**

- Posterior 1/3 of interventricular septum
- Inferior wall of left ventricle
- Posterior walls of ventricles
- Posteromedial papillary muscle

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Zoom In

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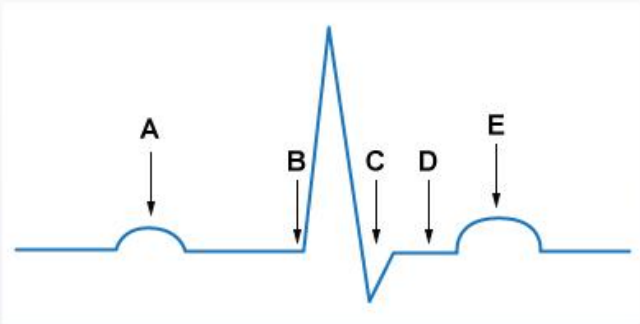
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TUTOR



A 21-year-old man comes to the office due to recurrent heart palpitations that start and stop abruptly. After initial workup, the physician suspects that there is an abnormal conduction pathway in this patient's heart that bypasses the atrioventricular node. Assuming the diagnosis is correct, which part of the patient's ECG is most likely to be affected during normal sinus rhythm?

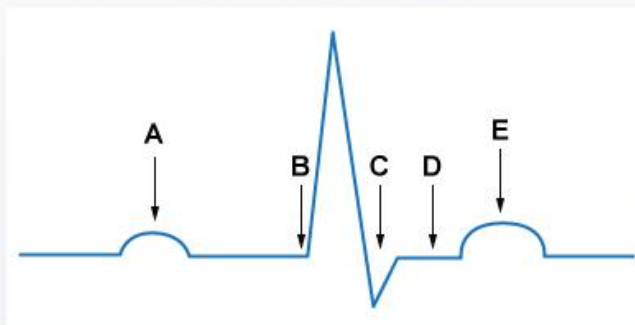


- ☐ A. A
- ☐ B. B
- ☐ C. C
- ☐ D. D
- ☐ E. E

Submit



A 21-year-old man comes to the office due to recurrent heart palpitations that start and stop abruptly. After initial workup, the physician suspects that there is an abnormal conduction pathway in this patient's heart that bypasses the atrioventricular node. Assuming the diagnosis is correct, which part of the patient's ECG is most likely to be affected during normal sinus rhythm?



- ☐ A. A [9%]
- ☒ B. B [87%]
- ☐ C. C [1%]
- ☐ D. D [1%]
- ☐ E. E [0%]

Omitted

Correct answer

87%
Answered correctly6 Seconds
Time Spent11/28/2018
Last Updated

Block Time Remaining: 00:18:23

TUTOR



Feedback

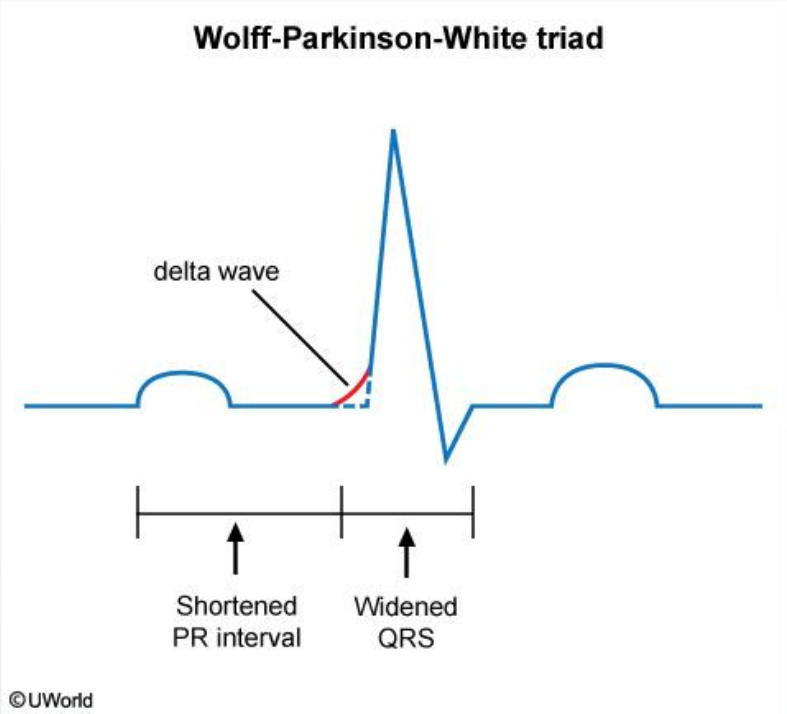


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Explanation



This young patient's repeated episodes of palpitations are suggestive of paroxysmal supraventricular tachycardia, and the physician suspects **Wolff-Parkinson-White (WPW) syndrome**. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows electrical conduction impulses to bypass the atrioventricular node and cause **preexcitation** of the ventricles. This preexcitation leads to

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Text Zoom

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This young patient's repeated episodes of palpitations are suggestive of paroxysmal supraventricular tachycardia, and the physician suspects **Wolff-Parkinson-White (WPW) syndrome**. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows electrical conduction impulses to bypass the atrioventricular node and cause **preexcitation** of the ventricles. This preexcitation leads to characteristic findings on baseline ECG, including a **shortened PR interval** (often <0.12 seconds), early upslope of the QRS complex (**delta wave**), and a **widened QRS complex**.

Many patients with an **accessory conduction pathway** can be asymptomatic and are diagnosed only when a WPW pattern is incidentally recognized on ECG. However, some patients will develop symptomatic arrhythmias. **Atrioventricular reentry tachycardia** (AVRT), a type of paroxysmal supraventricular tachycardia, is the most common arrhythmia that occurs with WPW. AVRT results from a rapid **reentrant circuit** that typically travels down the AV node to the ventricles and back up the accessory pathway, with patients often experiencing palpitations and a racing heart.

(Choice A) Except during periods of paroxysmal supraventricular tachycardia, a patient with an accessory conduction pathway should have a normal P wave on ECG.

(Choice C) The S wave on ECG is typically unaffected in patients with an accessory conduction pathway.

(Choices D and E) The ST segment and T wave roughly correspond to the plateau and repolarization phases, respectively, of the ventricular cardiomyocyte action potential. Due to changes in the normal geographic sequence of ventricular depolarization and repolarization as a result of preexcitation, there may be variable ST-segment/T-wave abnormalities. However, these findings are less common than the usual WPW triad of ECG abnormalities and, when present, are nonspecific for diagnosing an accessory conduction pathway.

Educational objective:

Wolff-Parkinson-White syndrome is characterized by symptomatic paroxysmal supraventricular tachycardia (eg, atrioventricular reentrant tachycardia) due to the presence of an accessory conduction pathway. During normal sinus rhythm, the presence of this accessory pathway causes ventricular preexcitation, which can be identified on ECG by the triad of a shortened PR interval, early upslope of the QRS complex (delta wave), and a widened QRS interval.

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Wolff-Parkinson-White (WPW) syndrome. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows electrical conduction impulses to bypass the atrioventricular node and cause **preexcitation** of the ventricles. This preexcitation leads to characteristic findings on baseline ECG, including a **shortened PR interval** (often <0.12 seconds), early upslope of the QRS complex (**delta wave**), and a **widened QRS complex**.

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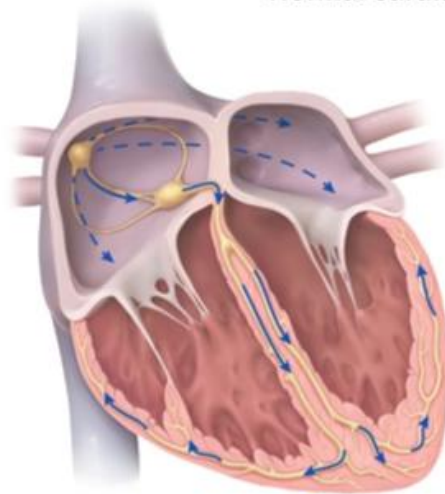
System Tray



Wolff-Parkinson-White (WPW) syndrome. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows

Exhibit Display

Normal cardiac conduction



Normal sinus rhythm



Ventricular preexcitation



Accessory pathway

WPW syndrome



Reentrant circuit

Zoom In

Zoom Out

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Add To Flash Card

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Feedback

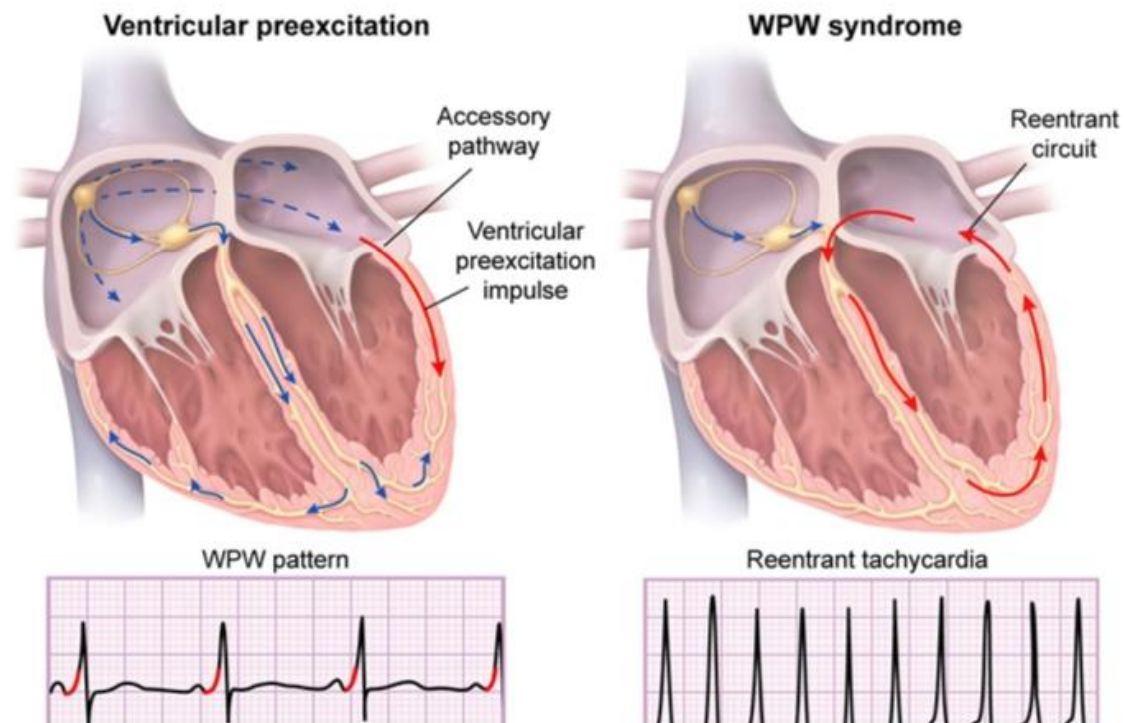
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Wolff-Parkinson-White (WPW) syndrome. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows

Exhibit Display



Zoom In

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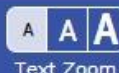
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Calculator



Reverse Color

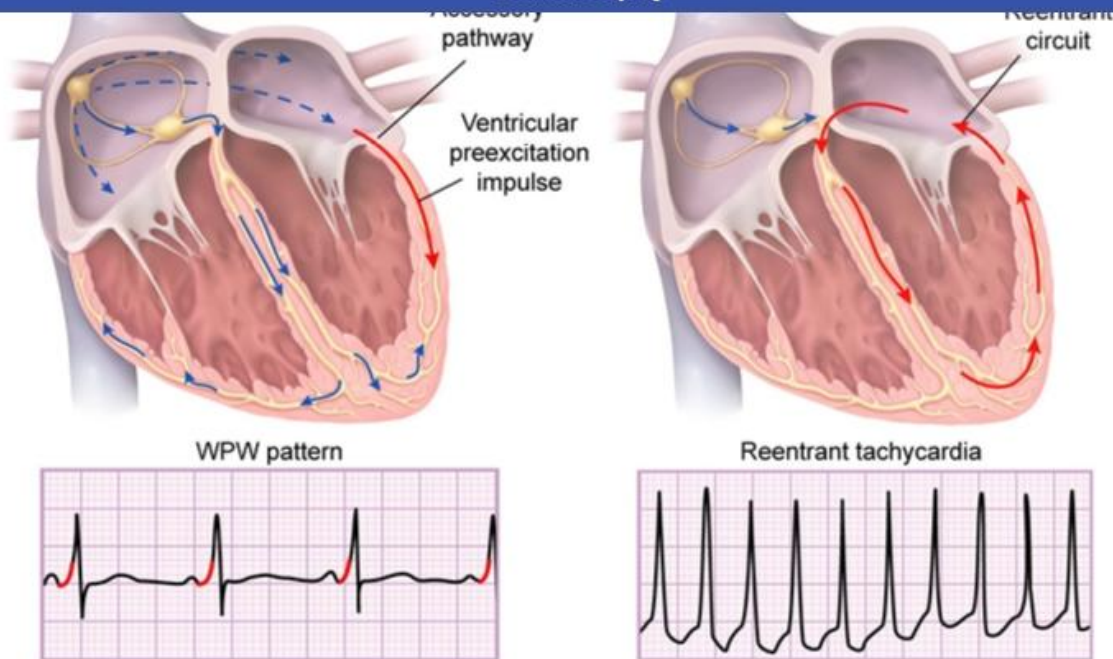


Text Zoom



Wolff-Parkinson-White (WPW) syndrome. WPW syndrome is caused by an **accessory conduction pathway** (bundle of Kent) that allows

Exhibit Display



WPW = Wolff-Parkinson-White.

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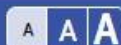
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A 64-year-old man is brought to the emergency department due to severe chest pain, diaphoresis, and shortness of breath. Symptoms began suddenly on awakening and have worsened over the past 2 hours. Blood pressure is 150/90 mm Hg and pulse is 102/min. Physical examination reveals an S4. ECG shows ST-segment depression and T-wave inversion in the lateral leads. Coronary angiography reveals a ruptured atherosclerotic plaque, with a thrombus in the left circumflex artery that causes near-total occlusion. Normal endothelial cells surrounding the lesion have released large amounts of a chemical substance that help decrease thrombus propagation by inhibiting platelet aggregation. Which of the following substances was most likely secreted by these endothelial cells?

- ☐ A. Hageman factor
- ☐ B. Kallikrein
- ☐ C. Prostacyclin
- ☐ D. Protein C
- ☐ E. Serotonin
- ☐ F. Thromboxane A₂

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Text Zoom

A 64-year-old man is brought to the emergency department due to severe chest pain, diaphoresis, and shortness of breath. Symptoms began suddenly on awakening and have worsened over the past 2 hours. Blood pressure is 150/90 mm Hg and pulse is 102/min. Physical examination reveals an S4. ECG shows ST-segment depression and T-wave inversion in the lateral leads. Coronary angiography reveals a ruptured atherosclerotic plaque, with a thrombus in the left circumflex artery that causes near-total occlusion. Normal endothelial cells surrounding the lesion have released large amounts of a chemical substance that help decrease thrombus propagation by inhibiting platelet aggregation. Which of the following substances was most likely secreted by these endothelial cells?

A. Hageman factor [5%]

B. Kallikrein [6%]

C. Prostacyclin [50%]

D. Protein C [17%]

E. Serotonin [1%]

F. Thromboxane A2 [17%]

Omitted

Correct answer
C

50%

Answered correctly

2 Seconds

Time Spent

10/29/2018

Last Updated

Explanation

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Explanation

The formation of a **platelet plug** (primary hemostasis) is essential for preventing bleeding after damage to vascular endothelium; it occurs in 3 steps:

- Platelet **adhesion** takes place via von Willebrand factor acting as a connector that binds platelets to underlying collagen
- Platelets become **activated** and secrete multiple substances, including adenosine diphosphate, ionized calcium, and fibrinogen, from their alpha and delta (dense) granules.
- Thromboxane A2 (**Choice F**), a prostaglandin, is released and acts as a vasoconstrictor and potent stimulator of platelet **aggregation**. Adenosine diphosphate also stimulates platelet aggregation.

Balance is required as excessive platelet plug formation can lead to a pathologic thrombus that restricts blood flow (eg, myocardial infarction). To oppose the functions of thromboxane A2, the endothelium secretes **prostacyclin** (prostaglandin I2), which is derived from arachidonic acid and synthesized from prostaglandin H2 by prostacyclin synthase. Once secreted, prostacyclin acts locally to **inhibit platelet aggregation** and adhesion to the vascular endothelium and to cause **vasodilation**. Nitric oxide aids in these functions as well. Atherosclerosis can impair the ability of endothelial cells to synthesize prostacyclin and nitric oxide, creating localized predisposition to excessive platelet thrombus formation.

A synthetic prostacyclin, epoprostenol, is used in the treatment of pulmonary hypertension, peripheral vascular disease, and Raynaud syndrome.

(Choice A) Hageman factor (factor XII) is synthesized by the liver and is activated by exposed collagen following damage to endothelial cells. It participates in secondary hemostasis by activating the intrinsic clotting pathway; it also activates fibrinolysis.

(Choice B) Kallikrein converts kininogen into bradykinin. It also may play a role in triggering the **fibrinolytic pathway**, but it does not inhibit platelet aggregation.

(Choice D) Protein C is a vitamin K–dependent factor synthesized by the liver. It regulates the coagulation cascade by inactivating factors Va and VIIIa.

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Windows Taskbar

System Tray

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A A A

Text Zoom

oppose the functions of thromboxane A2, the endothelium secretes **prostacyclin** (prostaglandin I2), which is derived from arachidonic acid and synthesized from prostaglandin H2 by prostacyclin synthase. Once secreted, prostacyclin acts locally to **inhibit platelet aggregation** and adhesion to the vascular endothelium and to cause **vasodilation**. Nitric oxide aids in these functions as well. Atherosclerosis can impair the ability of endothelial cells to synthesize prostacyclin and nitric oxide, creating localized predisposition to excessive platelet thrombus formation.

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(Choice D) Protein C is a vitamin K–dependent factor synthesized by the liver. It regulates the coagulation cascade by inactivating factors Va and VIIIa.

(Choice E) Serotonin has a wide range of functions, including mediation of mood (in the central nervous system) and gastrointestinal motility. It is also released from the delta granules of activated platelets and facilitates localized vasoconstriction.

Educational objective:

Prostacyclin (prostaglandin I2) is synthesized from prostaglandin H2 by prostacyclin synthase in vascular endothelial cells. Once secreted, it inhibits platelet aggregation and causes vasodilation to oppose the functions of thromboxane A2 and help maintain vascular homeostasis.

References

- Continuing education course #2: current understanding of hemostasis.

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TUTOR

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Feedback

Suspend

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Windows

Search

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Edge

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Shopping

Mail

Calendar

Chrome

Outlook

Skype

System Tray

2:02 PM

2/7/2019

2



A 42-year-old man comes to the emergency department due to acute-onset shortness of breath. He has no medical conditions and does not use alcohol, tobacco, or illicit drugs. The patient works as a box handler at a warehouse and has excellent exercise tolerance. Family history is insignificant. Blood pressure is 98/60 mm Hg and pulse is 112/min and regular. A short systolic murmur is heard at the apex. Bilateral crackles are present on lung auscultation. Bedside echocardiography reveals a flail posterior mitral leaflet due to chordal rupture and severe mitral regurgitation. Which of the following best reflects this patient's left ventricular hemodynamics compared to the normal heart?

- ☐ A. Afterload decrease, preload decrease, ejection fraction decrease
- ☐ B. Afterload decrease, preload increase, ejection fraction increase
- ☐ C. Afterload increase, preload decrease, ejection fraction decrease
- ☐ D. Afterload increase, preload decrease, ejection fraction increase
- ☐ E. Afterload increase, preload increase, ejection fraction increase

Submit

Block Time Remaining: 00:18:26

TUTOR





A 42-year-old man comes to the emergency department due to acute-onset shortness of breath. He has no medical conditions and does not use alcohol, tobacco, or illicit drugs. The patient works as a box handler at a warehouse and has excellent exercise tolerance. Family history is insignificant. Blood pressure is 98/60 mm Hg and pulse is 112/min and regular. A short systolic murmur is heard at the apex. Bilateral crackles are present on lung auscultation. Bedside echocardiography reveals a flail posterior mitral leaflet due to chordal rupture and severe mitral regurgitation. Which of the following best reflects this patient's left ventricular hemodynamics compared to the normal heart?

- ☐ A. Afterload decrease, preload decrease, ejection fraction decrease [24%]
- ☒ B. Afterload decrease, preload increase, ejection fraction increase [42%]
- ☐ C. Afterload increase, preload decrease, ejection fraction decrease [19%]
- ☐ D. Afterload increase, preload decrease, ejection fraction increase [2%]
- ☐ E. Afterload increase, preload increase, ejection fraction increase [10%]

Omitted

Correct answer
B42%
Answered correctly2 Seconds
Time Spent11/04/2018
Last Updated

Explanation

Hemodynamic changes in mitral regurgitation

Compensated Decompensated

Block Time Remaining: 00:18:27

TUTOR



Hemodynamic changes in mitral regurgitation			
	Acute MR	Compensated chronic MR	Decompensated chronic MR
Preload	↑↑	↑	↑
Afterload	↓	No change	↑
Contractile function	No change	No change	↓
Ejection fraction	↑↑	↑	↓
Forward stroke volume	↓	No change	↓



MR = mitral regurgitation.

This patient has **acute mitral regurgitation** (MR) due to a spontaneous chordae tendineae rupture. In acute MR, the left atrium (LA) is of normal size and compliance and is suddenly exposed to a large volume of regurgitant blood from the left ventricle (LV). This leads to very high LA pressures and subsequent **pulmonary edema**.

In addition, because the LV must accommodate the regurgitant blood volume along with normal pulmonary venous return, there is **increased LV end-diastolic volume** (preload). Meanwhile, the incompetent mitral valve forms a low-resistance regurgitant pathway for blood to flow into the LA during ventricular systole, **decreasing LV afterload**. The decreased afterload leads to **increased LV ejection fraction** (defined as $[\text{end-diastolic volume} - \text{end-systolic volume}] / \text{end-diastolic volume}$); however, much of the total ejection fraction is lost to regurgitation into the LA, resulting in overall decreased forward stroke volume and reduced cardiac output (manifesting as **hypotension** and cardiogenic shock).

In chronic MR, compensatory LA enlargement allows the LA to receive the regurgitant volume at lower filling pressures, preventing pulmonary edema from developing. Chronic volume overload also causes the LV to undergo substantial enlargement due to **eccentric hypertrophy**. Early on, the larger ventricular volume helps maintain forward stroke volume even in the setting of substantial regurgitant flow. However, with prolonged hemodynamic overload, progressive eccentric remodeling of the LV becomes maladaptive, resulting in an overwhelming increase in wall stress with eventual contractile dysfunction. The impaired contractility culminates in decompensation of chronic MR with reduced forward stroke volume (decreased cardiac output) and increased left-sided filling pressures (pulmonary edema).

Educational objective:

Regurgitant flow into the left atrium in acute mitral regurgitation leads to increased left atrial pressure and increased left ventricular end-diastolic volume (preload). The low-resistance regurgitant pathway also decreases left ventricular afterload with a resulting increase in ejection fraction but overall decrease in forward stroke volume. Increased left atrial pressure and decreased cardiac output result in pulmonary edema and severe hypotension, respectively.

References

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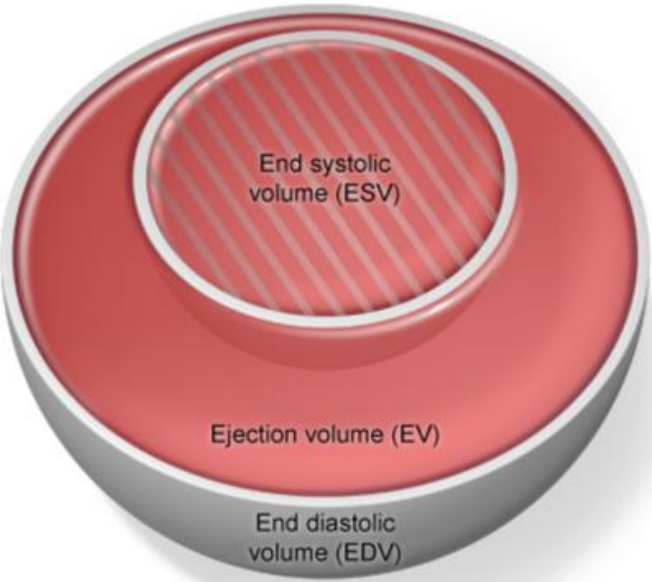
Calculator

Reverse Color

Text Zoom

Exhibit Display

$$\frac{EDV - ESV}{EDV} = \frac{EV}{EDV} = \text{Ejection fraction (\%)}$$



Zoom In

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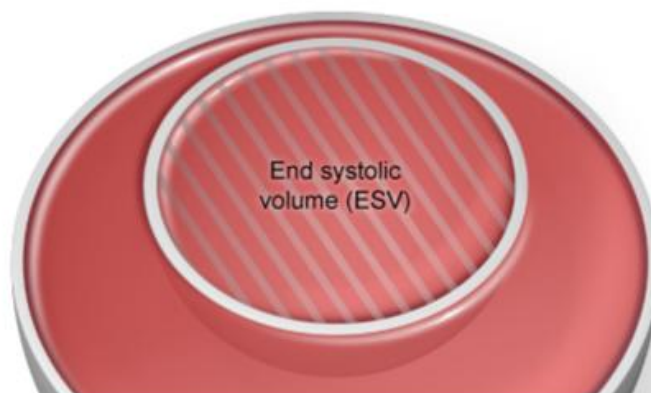
Text Zoom



Exhibit Display

Left ventricular ejection fraction

$$\frac{EDV - ESV}{EDV} = \frac{EV}{EDV} = \text{Ejection fraction (\%)}$$

End systolic
volume (ESV)

Zoom In

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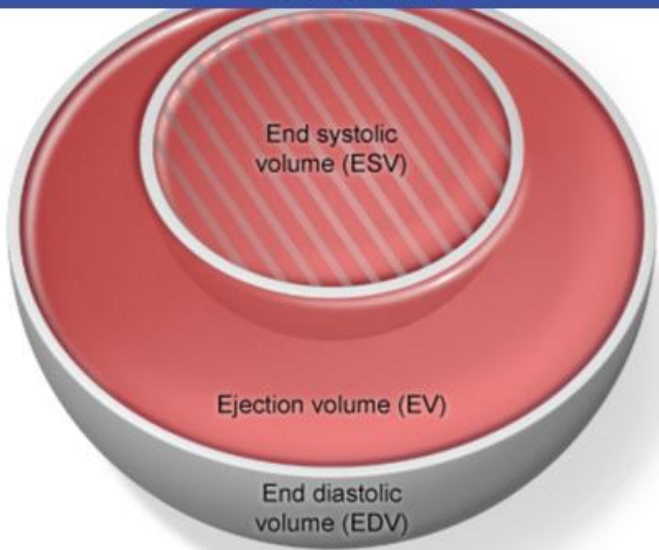
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Calculator

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End systolic volume (ESV)

Ejection volume (EV)

End diastolic volume (EDV)

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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Store, Mail, Calendar, Photos, and other background applications.

System tray showing network, volume, and date/time (2:02 PM 2/7/2019).

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Tutorial

Lab Values

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Calculator

Reverse Color

Text Zoom

Exhibit Display

Concentric & eccentric cardiac hypertrophy

Normal

RV

LV

Pressure overload

• Chronic hypertension

• Aortic stenosis

Volume overload

• Aortic or mitral regurgitation

• Ischemic heart disease

• Dilated cardiomyopathy

Sarcomere

Sarcomeres added in parallel

Sarcomeres added in series

Concentric left ventricular hypertrophy

Eccentric left ventricular hypertrophy

LV = left ventricle; RV = right ventricle.

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A A A

Text Zoom

A 44-year-old man with a history of intravenous drug use came to the emergency department due to fever and chills. Multiple blood culture sets were positive for *Staphylococcus aureus*, and the patient was diagnosed with infective endocarditis. He was successfully treated with a long course of antibiotics. Six months later, he returns to the clinic for a follow-up evaluation. The patient has no symptoms and reports good exercise tolerance. He is afebrile, blood pressure is 140/62 mm Hg, pulse is 82/min, and respirations are 16/min. Chest auscultation reveals a decrescendo diastolic murmur over the third intercostal space along the left sternal border. Echocardiogram shows severe aortic regurgitation, likely as a sequela to the prior infection. Which of the following changes is most responsible for maintaining cardiac output in the setting of this valvular abnormality?

A. Concentric left ventricular hypertrophy

B. Decrease in left ventricular preload

C. Increase in aortic elasticity

D. Increase in left ventricular afterload

E. Increase in left ventricular stroke volume

F. Sustained increase in heart rate

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Text Zoom

A 44-year-old man with a history of intravenous drug use came to the emergency department due to fever and chills. Multiple blood culture sets were positive for *Staphylococcus aureus*, and the patient was diagnosed with infective endocarditis. He was successfully treated with a long course of antibiotics. Six months later, he returns to the clinic for a follow-up evaluation. The patient has no symptoms and reports good exercise tolerance. He is afebrile, blood pressure is 140/62 mm Hg, pulse is 82/min, and respirations are 16/min. Chest auscultation reveals a decrescendo diastolic murmur over the third intercostal space along the left sternal border. Echocardiogram shows severe aortic regurgitation, likely as a sequela to the prior infection. Which of the following changes is most responsible for maintaining cardiac output in the setting of this valvular abnormality?

A. Concentric left ventricular hypertrophy [22%]

B. Decrease in left ventricular preload [2%]

C. Increase in aortic elasticity [1%]

D. Increase in left ventricular afterload [4%]

E. Increase in left ventricular stroke volume [56%]

F. Sustained increase in heart rate [13%]

Omitted

Correct answer
E

56%

Answered correctly

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Time Spent

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Explanation

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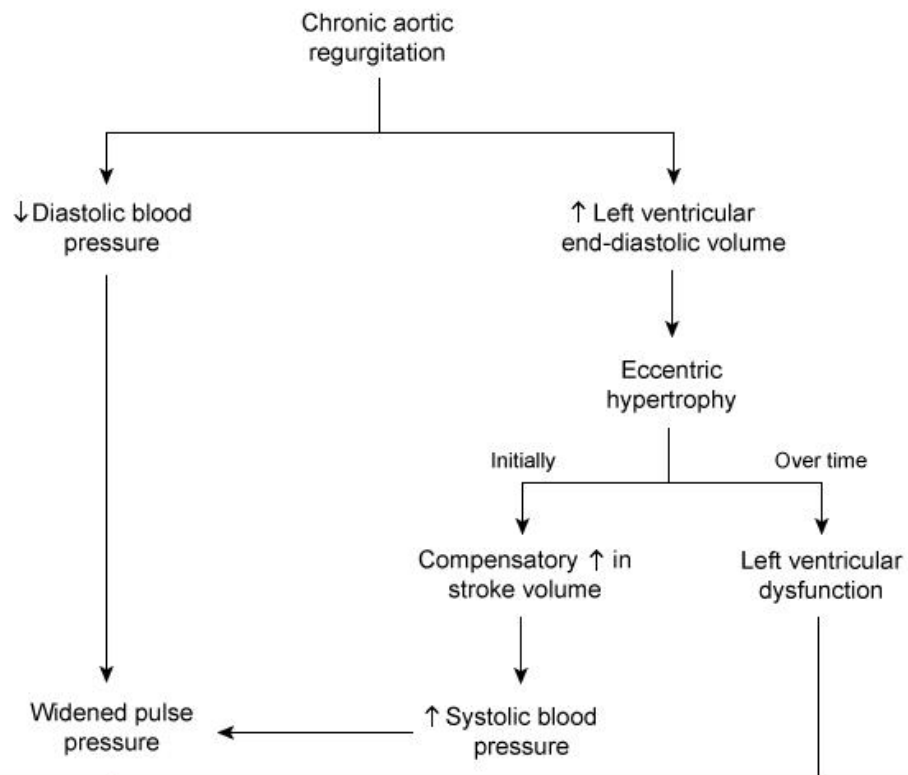
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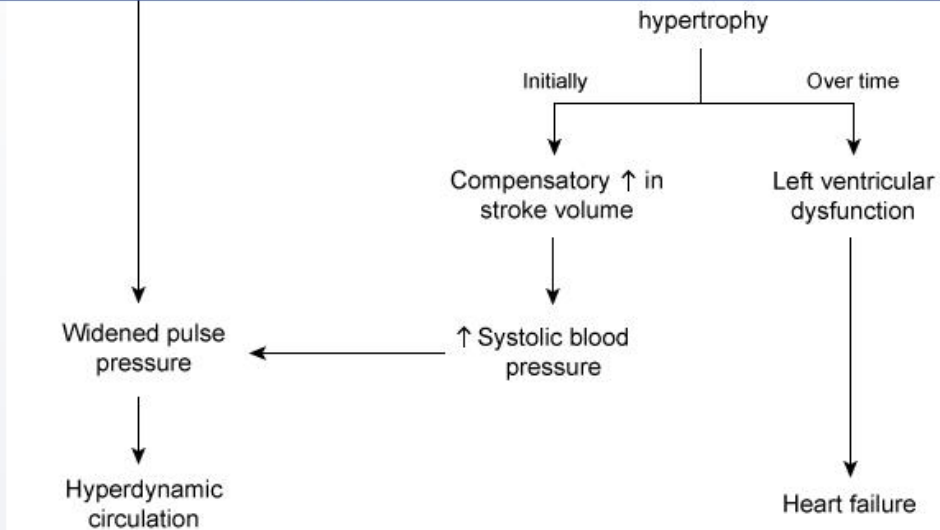
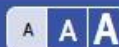
Pathophysiology of chronic aortic regurgitation



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This patient has developed **chronic aortic regurgitation** (AR) as a consequence of infective endocarditis. Incomplete closure of the aortic valve during diastole allows a portion of left ventricular stroke volume to leak back into the left ventricle; in severe AR this regurgitant flow volume may be up to 50% of total forward stroke volume. Left ventricular dilation caused by the increase in left ventricular end-diastolic volume (LVEDV) (**volume overload**) triggers **eccentric hypertrophy**, which involves ventricular wall lengthening due to the addition of myocardial contractile fibrils organized in series. This compensatory response causes an **increase in stroke volume** that is able to maintain cardiac output, and allows for a relatively long asymptomatic period (eg, several years) in most patients with chronic AR.

However, the compensatory eccentric hypertrophy in chronic AR is overall **maladaptive**. Over time, progressive left ventricular dilation leads to overwhelming wall stress with decreased stroke volume and eventual left ventricular failure.

(Choice A) Concentric hypertrophy occurs due to conditions that create ventricular pressure overload (eg, hypertension, aortic stenosis); it

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during diastole allows a portion of left ventricular stroke volume to leak back into the left ventricle, in severe AR this regurgitant flow volume may be up to 50% of total forward stroke volume. Left ventricular dilation caused by the increase in left ventricular end-diastolic volume (LVEDV) (volume overload) triggers eccentric hypertrophy, which involves ventricular wall lengthening due to the addition of myocardial contractile fibrils organized in series. This compensatory response causes an increase in stroke volume that is able to maintain cardiac output, and allows for a relatively long asymptomatic period (eg, several years) in most patients with chronic AR.

However, the compensatory eccentric hypertrophy in chronic AR is overall maladaptive. Over time, progressive left ventricular dilation leads to overwhelming wall stress with decreased stroke volume and eventual left ventricular failure.

(Choice A) Concentric hypertrophy occurs due to conditions that create ventricular pressure overload (eg, hypertension, aortic stenosis); it involves ventricular wall thickening due to the addition of myocardial contractile fibrils in parallel. Although some concentric hypertrophy may occur in AR, eccentric hypertrophy due to volume overload is the predominant change.

(Choice B) In AR, left ventricular preload or LVEDV is increased, rather than decreased. This helps the ventricle maintain cardiac output via the Frank-Starling mechanism. Decreased preload would cause a reduction in cardiac output.

(Choice C) Increased aortic elasticity (ie, decreased compliance or increased aortic stiffening) occurs with aging and results in higher systolic blood pressure, widened pulse pressure, and reduced cardiac output.

(Choice D) In chronic AR, there is an effective increase in left ventricular afterload due to the enlarged chamber size (increases wall stress) and high peak systolic pressures. This increases left ventricular work, making it more difficult to maintain cardiac output and eventually leads to heart failure.

(Choice F) Because cardiac output is maintained by an increase in stroke volume, there is typically minimal increase in heart rate in chronic AR. However, in acute AR, eccentric hypertrophy does not have time to develop and the capacity to increase stroke volume is limited. The resulting decrease in cardiac output triggers a reflexive increase in heart rate, but this response is typically insufficient and most patients develop severe cardiogenic shock with pulmonary edema.

Educational objective:

In chronic aortic regurgitation, persistent left ventricular volume overload triggers eccentric hypertrophy, which causes a compensatory increase in

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However, the compensatory eccentric hypertrophy in chronic AR is overall **maladaptive**. Over time, progressive left ventricular dilation leads to overwhelming wall stress with decreased stroke volume and eventual left ventricular failure.

(Choice A) Concentric hypertrophy occurs due to conditions that create ventricular pressure overload (eg, hypertension, aortic stenosis); it involves ventricular wall thickening due to the addition of myocardial contractile fibrils in parallel. Although some concentric hypertrophy may occur in AR, eccentric hypertrophy due to volume overload is the predominant change.

(Choice B) In AR, left ventricular preload or LVEDV is increased, rather than decreased. This helps the ventricle maintain cardiac output via the Frank-Starling mechanism. Decreased preload would cause a reduction in cardiac output.

(Choice C) Increased aortic elasticity (ie, decreased compliance or increased aortic stiffening) occurs with aging and results in higher systolic blood pressure, widened pulse pressure, and reduced cardiac output.

(Choice D) In chronic AR, there is an effective increase in left ventricular afterload due to the enlarged chamber size (increases wall stress) and high peak systolic pressures. This increases left ventricular work, making it more difficult to maintain cardiac output and eventually leads to heart failure.

(Choice F) Because cardiac output is maintained by an increase in stroke volume, there is typically minimal increase in heart rate in chronic AR. However, in acute AR, eccentric hypertrophy does not have time to develop and the capacity to increase stroke volume is limited. The resulting decrease in cardiac output triggers a reflexive increase in heart rate, but this response is typically insufficient and most patients develop severe cardiogenic shock with pulmonary edema.

Educational objective:

In chronic aortic regurgitation, persistent left ventricular volume overload triggers eccentric hypertrophy, which causes a compensatory increase in stroke volume to maintain cardiac output. This compensatory mechanism allows for a relatively long asymptomatic period in most patients; however, left ventricular dysfunction eventually occurs, leading to heart failure.

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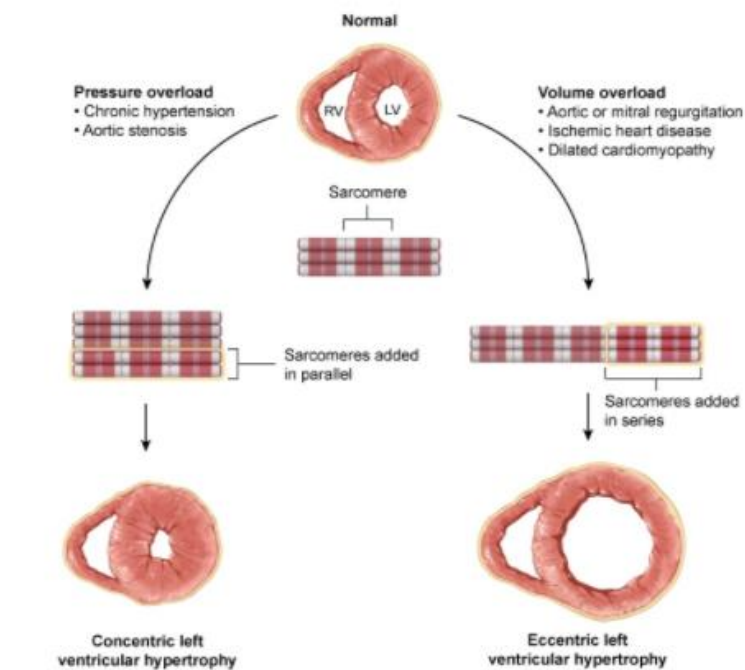
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System Tray



Exhibit Display

Concentric & eccentric cardiac hypertrophy



LV = left ventricle; RV = right ventricle.
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Text Zoom

A 52-year-old man comes to the office due to a 2-week history of progressive fatigue and exertional dyspnea. The patient is a postal delivery worker and reports that he gets short of breath after walking a few blocks. He has not had chest pain or palpitations. The patient does not use tobacco, alcohol, or illicit drugs. Cardiac auscultation reveals a murmur that is best heard when the patient sits up and leans forward. He brings to the office the report from a recent cardiac catheterization (graph shown below). This patient's murmur would most likely be loudest at which of the following time points on the graph?

A. A

B. B

C. C

D. D

E. E

Submit

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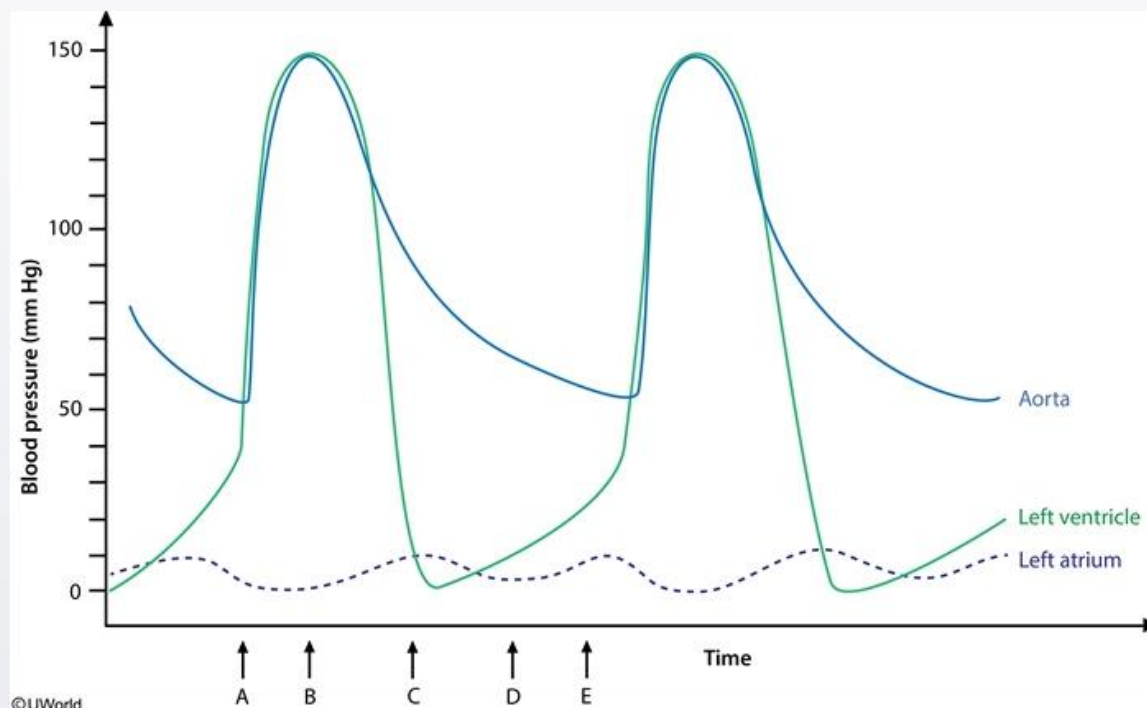


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Question Id: 227



A 52-year-old man comes to the office due to a 2-week history of progressive fatigue and exertional dyspnea. The patient is a postal delivery worker and reports that he gets short of breath after walking a few blocks. He has not had chest pain or palpitations. The patient does not use tobacco, alcohol, or illicit drugs. Cardiac auscultation reveals a murmur that is best heard when the patient sits up and leans forward. He brings to the office the report from a recent cardiac catheterization (graph shown below). This patient's murmur would most likely be loudest at which of the following time points on the graph?



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Text Zoom

A 52-year-old man comes to the office due to a 2-week history of progressive fatigue and exertional dyspnea. The patient is a postal delivery worker and reports that he gets short of breath after walking a few blocks. He has not had chest pain or palpitations. The patient does not use tobacco, alcohol, or illicit drugs. Cardiac auscultation reveals a murmur that is best heard when the patient sits up and leans forward. He brings to the office the report from a recent cardiac catheterization (graph shown below). This patient's murmur would most likely be loudest at which of the following time points on the graph?

A. A

B. B

C. C

D. D

E. E

Submit

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Windows Taskbar

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A 52-year-old man comes to the office due to a 2-week history of progressive fatigue and exertional dyspnea. The patient is a postal delivery worker and reports that he gets short of breath after walking a few blocks. He has not had chest pain or palpitations. The patient does not use tobacco, alcohol, or illicit drugs. Cardiac auscultation reveals a murmur that is best heard when the patient sits up and leans forward. He brings to the office the report from a recent cardiac catheterization (graph shown below). This patient's murmur would most likely be loudest at which of the following time points on the graph?

☐ A. A [14%]

☐ B. B [25%]

☒ C. C [47%]

☐ D. D [9%]

☐ E. E [3%]

Omitted

Correct answer C

47%
Answered correctly

2 Seconds
Time Spent

10/25/2018
Last Updated

Explanation

This patient's cardiac catheterization shows a hemodynamic profile consistent with **aortic regurgitation (AR)**; the characteristic decrescendo diastolic murmur is best heard with the patient leaning forward. The **peak intensity** of the murmur occurs **just after aortic valve closure** when

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This patient's cardiac catheterization shows a hemodynamic profile consistent with **aortic regurgitation** (AR); the characteristic decrescendo diastolic murmur is best heard with the patient leaning forward. The **peak intensity** of the murmur occurs **just after aortic valve closure** when the pressure gradient between the aorta and the left ventricle is maximal (point C). This pressure gradient falls progressively during diastole as blood regurgitates into the left ventricle, accounting for the **decrescendo** nature of the murmur.

Notable changes to the pressure tracings that occur in AR include:

- **Loss of the aortic dirotic notch:** On the normal aortic pressure tracing the dirotic notch is created by a small and abrupt increase in aortic pressure as blood is secured in the aorta by closure of the aortic valve. However, with an incompetent aortic valve there is immediate backflow of blood into the left ventricle, resulting in loss of pressure in the aorta and disappearance of the dirotic notch.
- **Steep diastolic decline of aortic pressure:** Aortic diastolic pressure declines more rapidly than normal as blood regurgitates into the left ventricle and nadirs at a lower than normal level.
- **High-peaking left ventricular and aortic systolic pressures:** The left ventricle attempts to compensate for regurgitant backflow by increasing stroke volume, resulting in higher systolic blood pressures, which combined with low aortic diastolic pressure leads to a **wide pulse pressure**.

(Choice A) This point corresponds to the start of left ventricular contraction, just after mitral valve closure and just prior to the opening of the aortic valve; it would mark the onset of the holosystolic murmur of **mitral regurgitation**.

(Choice B) This is the peak systolic pressure produced by the left ventricle during systole; the peak intensity of the crescendo-decrescendo murmur of **aortic stenosis** is heard here.

(Choice D) This point corresponds to mid-diastole. The murmur of AR is likely heard here as well, but at a lower intensity given the relatively lower pressure gradient between the aorta and left ventricle. The low-pitch rumble of **mitral stenosis** would also be heard at this time.

(Choice E) This point marks the onset of left atrial contraction (atrial kick) just before closure of the mitral valve. Accentuation of a mitral stenosis murmur may be heard at this time (presystolic accentuation).

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aortic pressure as blood is secured in the aorta by closure of the aortic valve. However, with an incompetent aortic valve there is immediate backflow of blood into the left ventricle, resulting in loss of pressure in the aorta and disappearance of the aortic notch.

- **Steep diastolic decline of aortic pressure:** Aortic diastolic pressure declines more rapidly than normal as blood regurgitates into the left ventricle and nadirs at a lower than normal level.
- **High-peaking left ventricular and aortic systolic pressures:** The left ventricle attempts to compensate for regurgitant backflow by increasing stroke volume, resulting in higher systolic blood pressures, which combined with low aortic diastolic pressure leads to a **wide pulse pressure**.

(Choice A) This point corresponds to the start of left ventricular contraction, just after mitral valve closure and just prior to the opening of the aortic valve; it would mark the onset of the holosystolic murmur of **mitral regurgitation**.

(Choice B) This is the peak systolic pressure produced by the left ventricle during systole; the peak intensity of the crescendo-decrescendo murmur of **aortic stenosis** is heard here.

(Choice D) This point corresponds to mid-diastole. The murmur of AR is likely heard here as well, but at a lower intensity given the relatively lower pressure gradient between the aorta and left ventricle. The low-pitch rumble of **mitral stenosis** would also be heard at this time.

(Choice E) This point marks the onset of left atrial contraction (atrial kick) just before closure of the mitral valve. Accentuation of a mitral stenosis murmur may be heard at this time (presystolic accentuation).

Educational objective:

Aortic regurgitation causes a decrescendo diastolic murmur with maximal intensity occurring just after closure of the aortic valve, when the pressure gradient between the aorta and left ventricle is the highest. The pressure tracing for aortic regurgitation is characterized by loss of the aortic aortic notch, steep diastolic decline in aortic pressure, and high-peaking systolic pressures.

References

- The rational clinical examination. Does this patient have aortic regurgitation?

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System Tray



A 24-year-old man comes to the office for an exercise evaluation. He is an avid long-distance runner. One of his team members recently died suddenly in his sleep, which made the patient worried about his health. He has no medical history, takes no medications, and does not use illicit drugs. His maternal grandfather had a heart attack at age 40 and underwent coronary artery bypass surgery. Blood pressure is 122/70 mm Hg and pulse is 49/min. Transthoracic echocardiogram shows the following:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	mildly increased
LV wall thickness	borderline increased uniformly
LV mass	increased
LV ejection fraction	69%

The right ventricle appears normal, and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- ☐ A. Anabolic steroid abuse
- ☐ B. Cardiac adaptation
- ☐ C. Hypertrophic cardiomyopathy
- ☐ D. Premature coronary artery disease
- ☐ E. Subclinical viral myocarditis

Submit

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A 24-year-old man comes to the office for an exercise evaluation. He is an avid long-distance runner. One of his team members recently died suddenly in his sleep, which made the patient worried about his health. He has no medical history, takes no medications, and does not use illicit drugs. His maternal grandfather had a heart attack at age 40 and underwent coronary artery bypass surgery. Blood pressure is 122/70 mm Hg and pulse is 49/min. Transthoracic echocardiogram shows the following:

Left atrium	mildly enlarged
Left ventricular (LV) cavity size	mildly increased
LV wall thickness	borderline increased uniformly
LV mass	increased
LV ejection fraction	69%

The right ventricle appears normal, and there are no valvular lesions. Which of the following best explains this patient's echocardiogram findings?

- ☐ A. Anabolic steroid abuse [2%]
- ☒ B. Cardiac adaptation [77%]
- ☐ C. Hypertrophic cardiomyopathy [17%]
- ☐ D. Premature coronary artery disease [0%]
- ☐ E. Subclinical viral myocarditis [0%]

Omitted

77%

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Explanation

This patient's echocardiogram findings are consistent with **athlete's heart** (ie, the physiologic cardiac adaptations that occur in highly trained athletes). **Endurance exercise** (eg, long-distance running) mostly increases the volume load on the heart (increased cardiac output) with a smaller component of increased pressure load (increased afterload). As a result, the heart predominantly undergoes eccentric hypertrophy (**cavity enlargement** improves diastolic function) with some degree of concentric **hypertrophy** (wall thickening increases systolic function). There is also **enhanced coronary capillary development** and a **decreased resting heart rate** due to improved efficiency of cardiac pumping.

Because these **physiologic changes improve cardiac function**, athlete's heart is not associated with an increased risk of cardiovascular morbidity or mortality. However, it is imperative that the changes of athlete's heart are differentiated from similar cardiac anomalies that may reflect serious underlying pathology that could increase the risk of sudden cardiac death (eg, hypertrophic cardiomyopathy). Findings that favor the presence of hypertrophic cardiomyopathy (**Choice C**) include LV hypertrophy predominantly affecting the septum, LV hypertrophy with reduced LV cavity size, impaired diastolic function, and a poorly developed coronary capillary network.

(Choice A) Anabolic steroid abuse can cause pathologic concentric LV hypertrophy, characterized by reduced diastolic function and poor coronary vascularization.

(Choice D) Severe coronary artery disease can lead to ischemic cardiomyopathy, which typically manifests as dilated cardiomyopathy with an enlarged LV cavity but thin LV walls with impaired systolic function.

(Choice E) Viral myocarditis may sometimes lead to dilated cardiomyopathy with eccentric hypertrophy and impaired LV systolic function. Subclinical disease is typically quite mild and resolves without sequelae.

Educational objective:

Athlete's heart refers to physiologic cardiac adaptations that improve cardiac function in response to high-level endurance training. There is predominant eccentric hypertrophy with a smaller component of concentric hypertrophy, leading to an overall increase in left ventricular (LV) mass, enlarged LV cavity size, increased LV wall thickness, and decreased resting heart rate.

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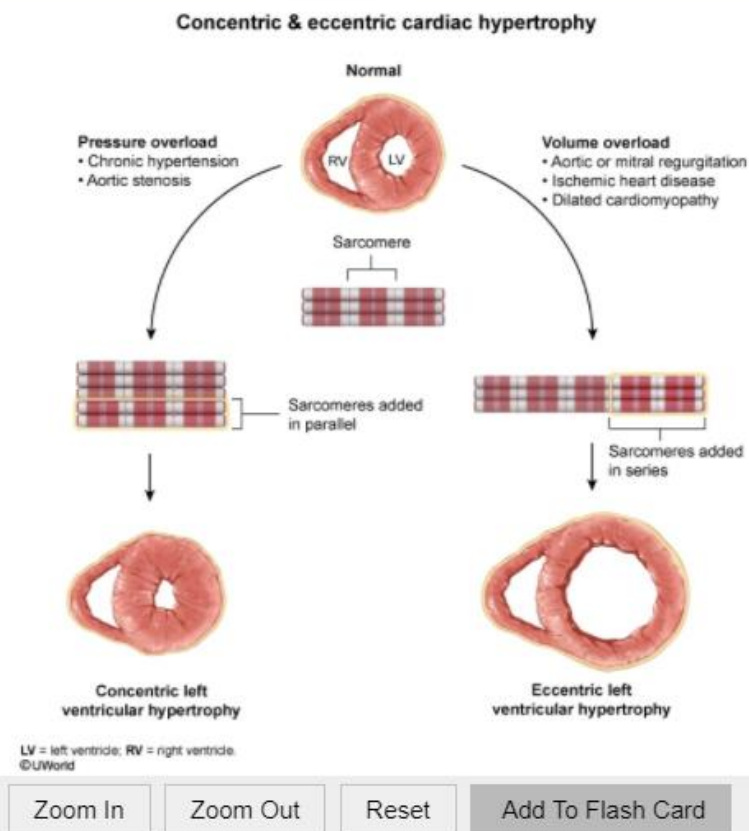
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Text Zoom

A 19-year-old man comes to the office due to difficulty seeing and blurred vision, which have worsened slowly over the past year. He is a second-year college student pursuing a degree in biochemistry. His grades are excellent, but he is concerned about the effect his poor vision has had on his classes this semester. The patient is also an avid swimmer. He weighs 71 kg (156.5 lb) and is 195 cm (6 ft 5 in) tall. Physical examination shows the findings seen in the [exhibits](#). This patient is most likely to die from which of the following conditions?

A. Aortic disease

B. Liver disease

C. Myocardial infarction

D. Overwhelming infection

E. Renal failure

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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Mail, and various background applications.

System tray showing network, volume, and date/time (2:05 PM 2/7/2019).

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Windows taskbar with icons for Start, Search, Task View, Edge, File Explorer, Mail, and various background applications.

2:05 PM

2/7/2019

2



A 19-year-old man comes to the office due to difficulty seeing and blurred vision, which have worsened slowly over the past year. He is a second-year college student pursuing a degree in biochemistry. His grades are excellent, but he is concerned about the effect his poor vision has had on his classes this semester. The patient is also an avid swimmer. He weighs 71 kg (156.5 lb) and is 195 cm (6 ft 5 in) tall. Physical examination shows the findings seen in the [exhibits](#). This patient is most likely to die from which of the following conditions?

- ☒ A. Aortic disease [88%]
- ☐ B. Liver disease [2%]
- ☐ C. Myocardial infarction [6%]
- ☐ D. Overwhelming infection [1%]
- ☐ E. Renal failure [1%]

Omitted

Correct answer

A

88%
Answered correctly73 Seconds
Time Spent01/23/2019
Last Updated

Explanation

Marfan syndrome (MFS) is one of the most common inherited connective tissue disorders. This condition is caused by a genetic defect in the glycoprotein **fibrillin-1** and results in abnormalities in the skeleton (eg, long extremities, scoliosis, pectus excavatum), eyes (eg, ectopia lentis), and cardiovascular system (eg, aortic root disease).

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88%

73 Seconds

01/23/2019

Correct answer

Answered correctly

Last Updated

A

Explanation

Marfan syndrome (MFS) is one of the most common inherited connective tissue disorders. This condition is caused by a genetic defect in the glycoprotein **fibrillin-1** and results in abnormalities in the skeleton (eg, long extremities, scoliosis, pectus excavatum), eyes (eg, ectopia lentis), and cardiovascular system (eg, aortic root disease).

The 2 most common cardiac abnormalities seen in MFS patients are **mitral valve prolapse** and **cystic medial degeneration of the aorta**. In more than 75% of MFS patients, cystic medial degeneration of the aorta results in aneurysmal dilation. If untreated, this can cause **aortic dissection**, the most common cause of death in MFS patients. The second most common cause of death is cardiac failure secondary to mitral valve prolapse and/or aortic regurgitation. For these reasons, MFS patients should be followed regularly by a cardiologist.

(Choices B, D, and E) With careful cardiovascular management, patients with MFS can have a normal life expectancy. MFS patients are not at increased risk for any of these other conditions.

(Choice C) Although homocystinuria is also associated with a **marfanoid body habitus**, distinguishing features of homocystinuria include intellectual disability and increased risk of thrombotic events (eg, myocardial infarction, stroke). However, this patient has no intellectual dysfunction.

Educational objective:

Cardiovascular lesions are the most life-threatening complications associated with Marfan syndrome. Early-onset cystic medial degeneration of the aorta predisposes to aortic dissection, the most common cause of death in these patients.

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A 73-year-old man is evaluated for exertional dyspnea. His exercise tolerance has decreased over the last year, and he can barely walk 2-3 blocks without stopping. The patient also reports occasional episodes of lightheadedness and palpitations. He has had no chest pain or syncope. He was diagnosed with hypertension in the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?

A. A

B. B

C. C

D. D

E. E

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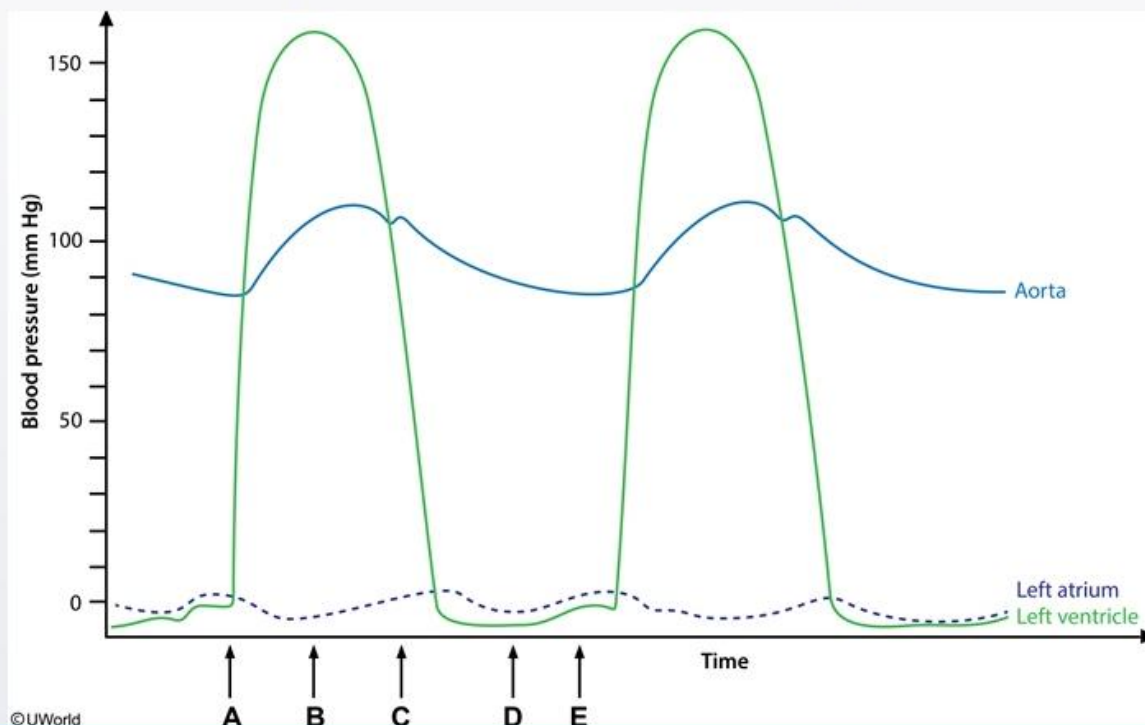


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Question Id: 243



A 73-year-old man is evaluated for exertional dyspnea. His exercise tolerance has decreased over the last year, and he can barely walk 2-3 blocks without stopping. The patient also reports occasional episodes of lightheadedness and palpitations. He has had no chest pain or syncope. He was diagnosed with hypertension in the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?



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A 73-year-old man is evaluated for exertional dyspnea. His exercise tolerance has decreased over the last year, and he can barely walk 2-3 blocks without stopping. The patient also reports occasional episodes of lightheadedness and palpitations. He has had no chest pain or syncope. He was diagnosed with hypertension in the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?

☐ A. A

☐ B. B

☐ C. C

☐ D. D

☐ E. E

Submit

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A 73-year-old man is evaluated for exertional dyspnea. His exercise tolerance has decreased over the last year, and he can barely walk 2-3 blocks without stopping. The patient also reports occasional episodes of lightheadedness and palpitations. He has had no chest pain or syncope. He was diagnosed with hypertension in the past but does not take any medications. Physical examination reveals a cardiac murmur. The patient is referred to a cardiologist for further evaluation. Cardiac catheterization is performed, and the findings are shown in the image below. Which of the following points most likely corresponds to the peak murmur intensity in this patient?

- ☐ A. A [15%]
- ☒ B. B [57%]
- ☐ C. C [17%]
- ☐ D. D [5%]
- ☐ E. E [3%]

Omitted

Correct answer
B57%
Answered correctly3 Seconds
Time Spent10/23/2018
Last Updated

Explanation

In the absence of any aortic valve pathology, the pressure in the aorta is similar to left ventricular pressure during systole. However, this patient's hemodynamic profile shows an abnormal pressure gradient between the left ventricular and aortic pressure tracings during systole (compare to **normal**), indicating significant aortic stenosis (AS). The intensity of the murmur of AS is directly related to the magnitude of the left ventricle to

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B

Explanation

In the absence of any aortic valve pathology, the pressure in the aorta is similar to left ventricular pressure during systole. However, this patient's hemodynamic profile shows an abnormal pressure gradient between the left ventricular and aortic pressure tracings during systole (compare to **normal**), indicating significant aortic stenosis (AS). The intensity of the murmur of AS is directly related to the magnitude of the left ventricle to aorta pressure gradient. The maximum pressure difference between the left ventricular and aortic pressure tracings is noted at point B and corresponds to the peak intensity of a cardiac murmur during auscultation.

(Choices A and C) The murmur of AS is a systolic ejection-type crescendo-decrescendo murmur that starts after the first heart sound following the opening of the aortic valve (point A). It typically ends before the A2 component of the second heart sound (point C). At points A and C, the left ventricular and aortic pressures are nearly equal with no or minimal flow across the aortic valve, and the murmur of AS would not be appreciated.

(Choices D and E) These points occur during diastole, when the aortic valve is closed and the mitral valve is open. There is no abnormally elevated pressure gradient between the left atrium and left ventricle. This is consistent with normal, unobstructed diastolic filling of the left ventricle, and no murmur would be heard during diastole.

Educational objective:

The murmur of aortic stenosis (AS) is a systolic ejection-type, crescendo-decrescendo murmur that starts after the first heart sound and typically ends before the A2 component of the second heart sound. The intensity of the murmur is proportional to the magnitude of the left ventricle to aorta pressure gradient during systole.

References

- The patient with a systolic murmur: severe aortic stenosis may be missed during cardiovascular examination.

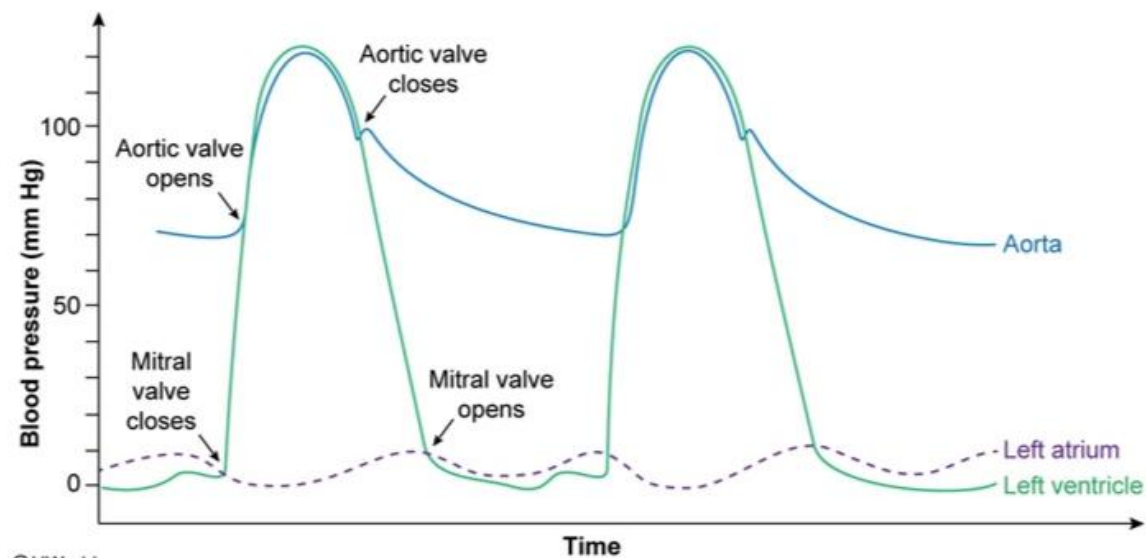




B

Exhibit Display

Normal pressure tracings



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