

Settings

1

2

3

4

5

Item 1 of 5

Question Id: 841

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

A 17-year-old girl with a history of depression is brought to the emergency department after attempting suicide. Her parents report that she consumed 2 bottles of insecticide after having an argument with her sister approximately 2 hours prior to presentation. The patient's symptoms include nausea, vomiting, abdominal pain, and copious watery diarrhea. She has a known history of self-cutting and suicidal ideations but no prior suicide attempts. Blood pressure is 86/42 mm Hg and pulse is 120/min. Her breath has a faint garlic odor, and she has numerous healing linear scars on her forearms. Which of the following medications should be administered immediately?

☐

A. CaNa_2EDTA

☐

B. Deferoxamine

☐

C. Dimercaprol

☐

D. Hydroxycobalamin

☐

E. Methylene blue

Submit

Block Time Remaining: 00:00:11

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

II

Suspend

End Block

4:45 PM

2/13/2019

Settings

1

2

3

4

5

Item 1 of 5

Question Id: 841

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

A 17-year-old girl with a history of depression is brought to the emergency department after attempting suicide. Her parents report that she consumed 2 bottles of insecticide after having an argument with her sister approximately 2 hours prior to presentation. The patient's symptoms include nausea, vomiting, abdominal pain, and copious watery diarrhea. She has a known history of self-cutting and suicidal ideations but no prior suicide attempts. Blood pressure is 86/42 mm Hg and pulse is 120/min. Her breath has a faint garlic odor, and she has numerous healing linear scars on her forearms. Which of the following medications should be administered immediately?

☐

A. CaNa₂EDTA [22%]

☐

B. Deferoxamine [8%]

☒

C. Dimercaprol [41%]

☐

D. Hydroxycobalamin [11%]

☐

E. Methylene blue [16%]

Omitted

Correct answer
C

41%

Answered correctly

13 Seconds

Time Spent

12/20/2018

Last Updated

Explanation

Arsenic poisoning

Block Time Remaining: 00:00:13

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

4:45 PM

2/13/2019

Settings

1

2

3

4

5

Item 1 of 5

Question Id: 841

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

Arsenic poisoning	
Mechanism	• Binds to sulfhydryl groups • Disrupts cellular respiration & gluconeogenesis
Sources	• Pesticides/insecticides • Contaminated water (often from wells) • Pressure-treated wood
Manifestations	• Acute: Garlic breath, vomiting, watery diarrhea, QTc prolongation • Chronic: Hypo/hyperpigmentation, hyperkeratosis, stocking-glove neuropathy
Treatment	• Dimercaprol (British anti-Lewisite) • DMSA (meso-2,3-dimercaptosuccinic acid, succimer)

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Arsenic is odorless, tasteless, and easily absorbed after ingestion or inhalation. Toxicity can result from exposure to pesticides/insecticides, contaminated water, pressure-treated wood, metallurgy, mining, or glass-making. Arsenic **binds to sulfhydryl groups, impairing cellular respiration** via **inhibition of pyruvate dehydrogenase** and disruption of gluconeogenesis and glutathione metabolism.

Acute arsenic poisoning affects most organ systems and presents with abdominal pain, vomiting, **severe watery diarrhea**, delirium, and **hypotension** from dehydration. **QTc prolongation** can progress to life-threatening torsades de pointes. A **garlic odor** is often noted on the patient's breath or stool.

Block Time Remaining: 00:00:13

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

Windows icons

System tray: 4:45 PM, 2/13/2019, notification icon

Settings

1

2

3

4

5

Item 1 of 5

Question Id: 841

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

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Arsenic is odorless, tasteless, and easily absorbed after ingestion or inhalation. Toxicity can result from exposure to pesticides/insecticides, contaminated water, pressure-treated wood, metallurgy, mining, or glass-making. Arsenic **binds to sulfhydryl groups, impairing cellular respiration** via **inhibition of pyruvate dehydrogenase** and disruption of gluconeogenesis and glutathione metabolism.

Acute arsenic poisoning affects most organ systems and presents with abdominal pain, vomiting, **severe watery diarrhea**, delirium, and **hypotension** from dehydration. **QTc prolongation** can progress to life-threatening torsades de pointes. A **garlic odor** is often noted on the patient's breath or stool.

The first-line chelating agent is **dimercaprol** (eg, British Anti-Lewisite), which increases urinary excretion of heavy metals by forming stable, nontoxic soluble chelates. The sulfhydryl group of dimercaprol combines with arsenic and displaces arsenic ions from the sulfhydryl groups of enzymes involved in cellular respiration. Dimercaprol has a very narrow therapeutic index, and serious side effects include nephrotoxicity, hypertension, and fever.

(Choice A) Acute lead poisoning can present with constipation, anemia, and irritability and confusion. CaNa_2EDTA is a treatment option for lead toxicity and works by forming non-ionizing salts to increase urinary lead excretion.

(Choice B) Deferoxamine is the preferred chelating agent for iron overdoses or overload due to multiple blood transfusions. It binds circulating iron and facilitates its urinary excretion.

(Choice D) Cyanide poisoning presents with confusion, flushing or a "cherry-red" skin color, abdominal pain, and vomiting. Its antidote, hydroxycobalamin (vitamin B_{12} precursor), binds to intracellular cyanide forming cyanocobalamin, which can be easily excreted in urine.

(Choice E) Methylene blue is indicated for treatment of methemoglobinemia, which presents with gray- or blue-colored skin, shortness of breath, and "chocolate-colored" blood. Methylene blue acts as an artificial electron transporter for reduction of methemoglobin through the NADPH pathway.

Educational objective:

Acute arsenic poisoning impairs cellular respiration and presents with abdominal pain, vomiting, diarrhea, hypotension, and a **garlic odor** on the

Block Time Remaining: 00:00:13

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

4:45 PM

2/13/2019

Settings

1

2

3

4

5

Item 1 of 5

Question Id: 841

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

non-toxic soluble chelates. The sulfhydryl group of dimercaprol combines with arsenic and displaces arsenic ions from the sulfhydryl groups of enzymes involved in cellular respiration. Dimercaprol has a very narrow therapeutic index, and serious side effects include nephrotoxicity, hypertension, and fever.

(Choice A) Acute lead poisoning can present with constipation, anemia, and irritability and confusion. CaNa_2EDTA is a treatment option for lead toxicity and works by forming non-ionizing salts to increase urinary lead excretion.

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(Choice E) Methylene blue is indicated for treatment of methemoglobinemia, which presents with gray- or blue-colored skin, shortness of breath, and "chocolate-colored" blood. Methylene blue acts as an artificial electron transporter for reduction of methemoglobin through the NADPH pathway.

Educational objective:

Acute arsenic poisoning impairs cellular respiration and presents with abdominal pain, vomiting, diarrhea, hypotension, and a **garlic odor** on the breath. **Insecticides** and **contaminated water** are common sources of arsenic. **Dimercaprol** is the chelating agent of choice.

References

- Acute arsenic poisoning in two siblings.
- Homicidal arsenic poisoning.
- The role of chelation in the treatment of arsenic and mercury poisoning.

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Block Time Remaining: 00:00:13

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 2 of 5

Question Id: 11949

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

A 3-year-old boy is brought to the office by his parents due to constipation. The boy previously had soft stools 1-2 times daily. However, over the past 3 months, his bowel movements have decreased to 1 stool every 3-4 days with significant straining and production of hard stool balls. The boy has intermittent diffuse abdominal pain and increased irritability, but these symptoms have not affected his overall appetite. Family history is notable for a maternal aunt with celiac disease and a paternal grandmother with "thyroid problems." Four months ago, the boy and his parents moved into a house owned by their family for generations, and the parents are worried that the stress of the move might be contributing to his symptoms. Height and weight have been tracking appropriately. Physical examination shows a pale, tired boy with palpable stool in his abdomen but no abdominal tenderness. Which of the following is the most appropriate diagnostic test for this patient?

☐

A. Anorectal manometry

☐

B. Blood lead level

☐

C. Serum electrolytes

☐

D. Serum thyroid-stimulating hormone

☐

E. Serum tissue transglutaminase

Submit

Block Time Remaining: 00:00:16

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 2 of 5

Question Id: 11949

Mark

Previous

Next

?

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

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☐ A. Anorectal manometry [5%]

☒ B. Blood lead level [81%]

☐ C. Serum electrolytes [2%]

☐ D. Serum thyroid-stimulating hormone [7%]

☐ E. Serum tissue transglutaminase [3%]

Omitted

Correct answer
B

81%

Answered correctly

5 Seconds

Time Spent

10/05/2018

Last Updated

Explanation

Block Time Remaining: 00:00:18

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 2 of 5

Question Id: 11949

Mark

Previous

Next

?

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

Complications of lead poisoning in children

Neurologic	• Loss of milestones/cognitive impairment • Behavioral problems • Encephalopathy
Gastrointestinal	• Constipation • Abdominal pain • Decreased vitamin D metabolism
Renal	• Interstitial nephritis
Hematologic	• Anemia

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This patient's constipation, abdominal pain, irritability, and pallor (suggesting anemia) are most likely due to lead poisoning. Policy efforts resulted in the elimination of lead, a toxic metal, in gasoline (1985) and paint (1977). Current lead exposure occurs through contact with **dust/paint in homes built before 1978**; this is the most likely cause of this patient's condition given the onset of symptoms after moving.

Lead binds to sulfhydryl groups on proteins, replaces calcium in calcium-dependent cellular functions, and directly inhibits enzymes in heme synthesis. Symptoms of lead poisoning are neurologic (**cognitive impairment**, irritability), gastrointestinal (**constipation**, **abdominal pain**), renal (**interstitial nephritis**), and hematologic (**anemia**).

Risk factors include having a sibling with prior elevated lead levels and living in older homes with recent renovation/construction. Because irreversible neurodevelopmental effects occur during exposure to low lead levels, screening is recommended for young children at high risk.

Block Time Remaining: 00:00:18

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

4:46 PM

2/13/2019

2

Settings

1

2

3

4

5

Item 2 of 5

Question Id: 11949

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

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Lead binds to sulfhydryl groups on proteins, replaces calcium in calcium-dependent cellular functions, and directly inhibits enzymes in heme synthesis. Symptoms of lead poisoning are neurologic (**cognitive impairment**, irritability), gastrointestinal (**constipation, abdominal pain**), renal (**interstitial nephritis**), and hematologic (**anemia**).

Risk factors include having a sibling with prior elevated lead levels and living in older homes with recent renovation/construction. Because irreversible neurodevelopmental effects occur during exposure to low lead levels, screening is recommended for young children at high risk. Diagnosis is made by measuring the **blood lead level**. Urine δ -aminolevulinic acid (heme synthesis substrate) is also **elevated**.

(Choice A) **Hirschsprung disease** typically presents in the newborn period with bilious vomiting and failure to pass meconium. Anorectal manometry measures rectal pressure via rectal catheter and shows absent relaxation of the internal anal sphincter. The diagnosis of Hirschsprung disease is confirmed by rectal biopsy.

(Choice C) Hypokalemia and hypercalcemia can present with constipation. However, patients with hypokalemia exhibit muscle weakness or arrhythmias, whereas hypercalcemia is accompanied by muscle weakness, confusion, and renal insufficiency.

(Choice D) Hypothyroidism can cause constipation, and primary hypothyroidism is associated with elevated TSH. However, constipation due to hypothyroidism is usually accompanied by other symptoms, such as decreased growth velocity and short stature in children.

(Choice E) Tissue transglutaminase is a screening test for celiac disease, which typically presents with diarrhea, weight loss, and abdominal pain. Some patients present with constipation instead of diarrhea. However, given this patient's appropriate growth and symptom onset after the recent move, lead poisoning is the more likely diagnosis.

Educational objective:

Lead poisoning can cause neurologic, gastrointestinal, renal, and hematologic complications. Although symptoms can be vague, lead poisoning should be suspected if these symptoms arise while residing in a home built before 1978. Diagnosis is made by measuring the patient's blood lead level.

Block Time Remaining: 00:00:18

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

Windows

Search

Task View

Edge

File Explorer

Shopping

Email

Calendar

Maps

Chrome

Firefox

Skype

System Tray

4:46 PM

2/13/2019

2

A 49-year-old man with long-standing hypertension comes to the emergency department with severe shortness of breath. He has not been compliant with outpatient follow-up or his prescribed medications. His blood pressure on arrival is 260/144 mm Hg and pulse is 100/min and regular. Chest examination shows bibasilar crackles. There are no heart murmurs. His serum creatinine level is 1.1 mg/dL. Intravenous furosemide and continuous nitroprusside infusion are started along with noninvasive positive pressure ventilation, and he experiences improvement in his symptoms. The next morning, the patient seems confused and lethargic. The nitroprusside infusion rate is found to be higher than recommended. A medication that acts as a donor of which of the following elements would most likely help reverse this patient's condition?

- ☐ A. Hydrogen
- ☐ B. Phosphorus
- ☐ C. Potassium
- ☐ D. Sodium
- ☐ E. Sulfur

Submit

Activate Windows

Go to Settings to activate Windows.

Block Time Remaining: 00:00:24

TUTOR

Settings

1

2

3

4

5

Item 3 of 5

Question Id: 1254

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

A 49-year-old man with long-standing hypertension comes to the emergency department with severe shortness of breath. He has not been compliant with outpatient follow-up or his prescribed medications. His blood pressure on arrival is 260/144 mm Hg and pulse is 100/min and regular. Chest examination shows bibasilar crackles. There are no heart murmurs. His serum creatinine level is 1.1 mg/dL. Intravenous furosemide and continuous nitroprusside infusion are started along with noninvasive positive pressure ventilation, and he experiences improvement in his symptoms. The next morning, the patient seems confused and lethargic. The nitroprusside infusion rate is found to be higher than recommended. A medication that acts as a donor of which of the following elements would most likely help reverse this patient's condition?

☐ A. Hydrogen [19%]

☐ B. Phosphorus [10%]

☐ C. Potassium [10%]

☐ D. Sodium [15%]

☒ E. Sulfur [44%]

Omitted

Correct answer
E

44%

Answered correctly

8 Seconds

Time Spent

08/31/2018

Last Updated

Explanation

Management approaches to cyanide toxicity

Block Time Remaining: 00:00:26

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Management approaches to cyanide toxicity	
Treatment strategy	Mechanism of action
Sodium nitrite	Promotes methemoglobin formation, which combines with cyanide to form cyanmethemoglobin
Sodium thiosulfate	Serves as sulfur donor to promote hepatic rhodanese-mediated conversion of cyanide to thiocyanate, which is excreted in the urine
Hydroxocobalamin	Cobalt moiety binds to intracellular cyanide ions & forms cyanocobalamin, which is excreted in the urine

Activate Windows

Go to Settings to activate Windows



Feedback

 Suspend

WS.  End Block

Block Time Remaining: 00:00:26

TUTOR

Settings

1

2

3

4

5

Item 3 of 5

Question Id: 1254

Mark

Previous

Next

?

Tutorial

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

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This patient's clinical presentation is likely due to **cyanide toxicity** from **nitroprusside infusion**. Nitroprusside is a parenteral vasodilator with quick onset/offset mechanics that is commonly used for rapid blood pressure control in patients with hypertensive emergency. It is metabolized in the body to release nitric oxide and cyanide ions.

Cyanide is a potent mitochondrial toxin that binds to Fe^{3+} in **cytochrome c oxidase**, inhibiting the electron transport chain and halting aerobic respiration in the cell. Cyanide toxicity presents with altered mental status, seizures, cardiovascular collapse, **lactic acidosis**, and **bright red venous blood** (seen on venous blood gas and funduscopy). Nitroprusside-induced cyanide toxicity is most likely to occur in patients receiving higher doses/prolonged infusions or those with renal insufficiency.

Cyanide is normally metabolized in the tissues by rhodanese, an enzyme that transfers a sulfur molecule to cyanide to form thiocyanate, which is less toxic and excreted in the urine. Cyanide overdose depletes the available sulfur donors, allowing cyanide to accumulate in toxic amounts. **Sodium thiosulfate** works as an antidote by providing additional sulfur groups for rhodanese, enhancing cyanide detoxification. It is used in conjunction with **hydroxocobalamin** and **sodium nitrite** in the management of cyanide toxicity.

(Choices A, B, and C) Hydrogen, phosphorous, and potassium have no significant role in the detoxification reactions for cyanide poisoning.

(Choice D) Although sodium thiosulfate contains sodium, the sodium component has no role in the detoxification reaction in cyanide poisoning.

Educational objective:

Cyanide toxicity can occur in patients treated with nitroprusside. Cyanide toxicity presents with altered mental status, seizures, cardiovascular collapse, lactic acidosis, and bright red venous blood. Antidotal treatment of cyanide toxicity can be achieved by 3 different strategies: direct binding of cyanide ions (hydroxocobalamin), induction of methemoglobinemia (sodium nitrite), and use of detoxifying sulfur donors (sodium thiosulfate).

References

Block Time Remaining: 00:00:26

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

⏸

Suspend

⏹

End Block

Windows

Search

Taskbar Icons

System Tray

4:46 PM

2/13/2019

2

Settings

1

2

3

4

5

Item 4 of 5

Question Id: 172

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

A 14-year-old girl is brought to the emergency department following a suicide attempt. She swallowed several of her grandfather's theophylline tablets after an intense argument with her parents a few hours ago. The patient reports nausea and headache. She has no other prior medical problems and takes no medications. Her temperature is 37 C (98.6 F), blood pressure is 114/76 mm Hg, pulse is 88/min, and respirations are 16/min. This patient should be carefully monitored for which of the following?

A. Hematemesis and melena

B. Hypoglycemia and complete heart block

C. Jaundice and elevated liver enzymes

D. Sedation and respiratory depression

E. Seizures and tachyarrhythmias

Submit

Block Time Remaining: 00:00:28

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

Windows Taskbar

System Tray

Settings

1

2

3

4

5

Item 4 of 5

Question Id: 172

Mark

Previous

Next

?

Lab Values

Notes

Calculator

Reverse Color

A

A

A

Text Zoom

A 14-year-old girl is brought to the emergency department following a suicide attempt. She swallowed several of her grandfather's theophylline tablets after an intense argument with her parents a few hours ago. The patient reports nausea and headache. She has no other prior medical problems and takes no medications. Her temperature is 37 C (98.6 F), blood pressure is 114/76 mm Hg, pulse is 88/min, and respirations are 16/min. This patient should be carefully monitored for which of the following?

☐ A. Hematemesis and melena [1%]

☐ B. Hypoglycemia and complete heart block [9%]

☐ C. Jaundice and elevated liver enzymes [10%]

☐ D. Sedation and respiratory depression [13%]

☒ E. Seizures and tachyarrhythmias [65%]

Omitted

Correct answer
E

65%

Answered correctly

4 Seconds

Time Spent

10/04/2018

Last Updated

Explanation

In low to moderate doses, methylxanthines cause mild cortical arousal and insomnia, much like caffeine. Acute theophylline intoxication results in nausea/vomiting, abdominal pain, diarrhea, cardiac arrhythmias, and seizures. **Seizures** are the major cause of morbidity and mortality in **theophylline intoxication**. **Tachyarrhythmias** are the other major concern but usually do not cause QT prolongation.

Block Time Remaining: 00:00:30

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 4 of 5

Question Id: 172

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

Explanation

In low to moderate doses, methylxanthines cause mild cortical arousal and insomnia, much like caffeine. Acute theophylline intoxication results in nausea/vomiting, abdominal pain, diarrhea, cardiac arrhythmias, and seizures. **Seizures** are the major cause of morbidity and mortality in **theophylline intoxication**. **Tachyarrhythmias** are the other major concern but usually do not cause QT prolongation.

Treatment of theophylline intoxication includes administration of activated charcoal to reduce gastrointestinal absorption. Beta blockers are the drugs of choice for theophylline-induced cardiac tachyarrhythmias. Theophylline-induced seizures are difficult to treat, but benzodiazepines and barbiturates are the most effective agents.

(Choice A) Iron poisoning can cause hematemesis and melena as iron is directly toxic to gastric mucosal cells.

(Choice B) Bradycardia, hypotension, and hypoglycemia are seen with beta blocker toxicity. Glucagon is the specific antidote as it increases intracellular cyclic adenosine monophosphate and cardiac contractility.

(Choice C) Acetaminophen causes liver damage following a latent period. Acetylcysteine, a glutathione donor, is used to treat acetaminophen toxicity.

(Choice D) Severe sedation, respiratory depression, and constricted pupils are features of opioid intoxication. Treatment includes general supportive care and administration of opioid antagonists such as naloxone.

Educational objective:

Seizures are the major cause of morbidity and mortality from theophylline intoxication. Tachyarrhythmias are the other major concern.

References

- Role of extracorporeal drug removal in acute theophylline poisoning. A review.
- First-line therapy for theophylline-associated seizures.

Block Time Remaining: 00:00:30

TUTOR

Activate Windows

Go to Settings to activate Windows.

Feedback

Suspend

End Block

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 5 of 5

Question Id: 1448

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

A 24-year-old woman comes to the emergency department due to bloody emesis. She had 2 episodes of vomiting bright red blood and feels lightheaded and dizzy. The patient was recently diagnosed with factitious disorder after being hospitalized multiple times for a myriad of symptoms and undergoing several invasive procedures. Physical examination shows scattered ecchymoses. The abdomen is soft and nontender. Rectal examination shows maroon-colored, guaiac-positive stool. After much questioning, she admits to having ingested rat poison several days ago. Immediate treatment of this patient should include which of the following?

☐ A. Cryoprecipitate

☐ B. Desmopressin

☐ C. Fresh frozen plasma

☐ D. Platelet transfusion

☐ E. Protamine sulfate

Submit

Block Time Remaining: 00:00:32

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

II

Suspend

End Block

Windows

Search

Taskbar Icons

System Tray

4:46 PM

2/13/2019

Settings

1

2

3

4

5

Item 5 of 5

Question Id: 1448

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

A 24-year-old woman comes to the emergency department due to bloody emesis. She had 2 episodes of vomiting bright red blood and feels lightheaded and dizzy. The patient was recently diagnosed with factitious disorder after being hospitalized multiple times for a myriad of symptoms and undergoing several invasive procedures. Physical examination shows scattered ecchymoses. The abdomen is soft and nontender. Rectal examination shows maroon-colored, guaiac-positive stool. After much questioning, she admits to having ingested rat poison several days ago. Immediate treatment of this patient should include which of the following?

☐

A. Cryoprecipitate [7%]

☐

B. Desmopressin [2%]

☒

C. Fresh frozen plasma [68%]

☐

D. Platelet transfusion [8%]

☐

E. Protamine sulfate [12%]

Omitted

Correct answer
C

68%

Answered correctly

4 Seconds

Time Spent

12/27/2018

Last Updated

Explanation

	Warfarin overdose	Heparin overdose

Block Time Remaining: 00:00:34

TUTOR

Activate Windows

Go to Settings to activate Windows.

0

Feedback

II

Suspend

End Block

4:46 PM
2/13/2019

	Warfarin overdose	Heparin overdose
Vitamin K	Effective (takes days)	Ineffective
FFP	Effective (contains all blood clotting factors & proteins)	Ineffective (contains antithrombin III → enhances heparin effect)
Protamine	Ineffective	Effective (heparin-specific antidote)

FFP = fresh frozen plasma.

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Most available **rodenticides** contain brodifacoum ("superwarfarin"), a long-acting 4-hydroxycoumarin derivative. By ingesting rodenticide, this patient depleted her vitamin K-dependent clotting factors, thereby causing an acquired coagulopathy (similar to **warfarin** toxicity) that led to bleeding (gastrointestinal hemorrhage with hematemesis and guaiac-positive stools, ecchymoses). The anticoagulant effect is generally seen approximately 48 hours following ingestion, which represents the time required for depletion of the coagulation factors.

To treat life-threatening bleeding in the setting of coumarin or warfarin toxicity, rapid replenishment of clotting factors II, VII, IX, and X is generally performed through intravenous administration of **fresh frozen plasma** (FFP), which contains all coagulation factors and other proteins present in the original unit of blood. Additional vitamin K should also be given. Vitamin K alone can be used in patients with abnormal coagulation tests (eg, prolonged prothrombin or partial thromboplastin times) but no bleeding.

(Choice A) Cryoprecipitate (precipitate obtained from FFP by centrifugation/thawing) contains factor VIII, factor XIII, von Willebrand factor, and fibrinogen. Therefore, it would not be of significant benefit when vitamin K-dependent clotting factors (II, VII, IX, and X) require replenishment.

(Choice B) Desmopressin increases plasma levels of von Willebrand factor and factor VIII; it is used in management of hemophilia A and von

Activate Windows

Go to Settings to activate Windows.

Settings

1

2

3

4

5

Item 5 of 5

Question Id: 1448

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

FFP = fresh frozen plasma.

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Most available **rodenticides** contain brodifacoum ("superwarfarin"), a long-acting 4-hydroxycoumarin derivative. By ingesting rodenticide, this patient depleted her vitamin K-dependent clotting factors, thereby causing an acquired coagulopathy (similar to **warfarin** toxicity) that led to bleeding (gastrointestinal hemorrhage with hematemesis and guaiac-positive stools, ecchymoses). The anticoagulant effect is generally seen approximately 48 hours following ingestion, which represents the time required for depletion of the coagulation factors.

To treat life-threatening bleeding in the setting of coumarin or warfarin toxicity, rapid replenishment of clotting factors II, VII, IX, and X is generally performed through intravenous administration of **fresh frozen plasma** (FFP), which contains all coagulation factors and other proteins present in the original unit of blood. Additional vitamin K should also be given. Vitamin K alone can be used in patients with abnormal coagulation tests (eg, prolonged prothrombin or partial thromboplastin times) but no bleeding.

(Choice A) Cryoprecipitate (precipitate obtained from FFP by centrifugation/thawing) contains factor VIII, factor XIII, von Willebrand factor, and fibrinogen. Therefore, it would not be of significant benefit when vitamin K-dependent clotting factors (II, VII, IX, and X) require replenishment.

(Choice B) Desmopressin increases plasma levels of von Willebrand factor and factor VIII; it is used in management of hemophilia A and von Willebrand disease.

(Choice D) Rat poison or warfarin does not affect platelet count.

(Choice E) Protamine sulfate is used to treat heparin overdose. It binds to heparin, forming a complex that has no anticoagulant activity.

Educational objective:

Most available rodenticides contain brodifacoum, a long-acting 4-hydroxycoumarin derivative. A patient who has ingested a quantity of rodenticide sufficient to cause coagulopathy and abnormal bleeding (similar to warfarin toxicity) requires immediate treatment with fresh frozen plasma in addition to vitamin K.

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2/13/2019

A 34-year-old woman is brought to the emergency department by ambulance due to nausea, an episode of vomiting, and dizziness. Her boyfriend arrives with her and says that he found her lying on the bed next to an empty bottle of aspirin tablets. When asked about the potential ingestion, the patient admits to swallowing a "bunch of aspirin pills a little more than 12 hours ago" after leaving work yesterday. She also has "an annoying buzzing sound that won't stop." Temperature is 38.3 C (101 F) and pulse is 102/min. She appears agitated and confused. Which of the following sets of laboratory results would most likely be found in this patient?

	pH	PaCO ₂	Plasma HCO ₃
☐ A.	7.30	34 mm Hg	16 mEq/L
☐ B.	7.38	20 mm Hg	12 mEq/L
☐ C.	7.42	40 mm Hg	26 mEq/L
☐ D.	7.48	50 mm Hg	36 mEq/L
☐ E.	7.60	25 mm Hg	24 mEq/L

Submit

Settings

1

Item 1 of 1

Question Id: 1544

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

A

A

A

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A 34-year-old woman is brought to the emergency department by ambulance due to nausea, an episode of vomiting, and dizziness. Her boyfriend arrives with her and says that he found her lying on the bed next to an empty bottle of aspirin tablets. When asked about the potential ingestion, the patient admits to swallowing a "bunch of aspirin pills a little more than 12 hours ago" after leaving work yesterday. She also has "an annoying buzzing sound that won't stop." Temperature is 38.3 C (101 F) and pulse is 102/min. She appears agitated and confused. Which of the following sets of laboratory results would most likely be found in this patient?

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[35%]			
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[35%]			
☐ C.	7.42	40 mm Hg	26 mEq/L
[5%]			
☐ D.	7.48	50 mm Hg	36 mEq/L
[12%]			
☐ E.	7.60	25 mm Hg	24 mEq/L
[12%]			

Omitted

Correct answer B

35%

Answered correctly

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4:54 PM

2/13/2019

Settings

1

Item 1 of 1

Question Id: 1544

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

Acute salicylate intoxication typically presents with nausea/vomiting, confusion, dizziness, **tinnitus** (eg, buzzing sound), **fever**, and **tachypnea** within several hours after ingestion. Two different acid-base abnormalities are characteristic.

1. **Respiratory alkalosis** occurs first as salicylates directly stimulate the medullary respiratory center, resulting in increased ventilation and loss of CO₂ in the expired air.
2. **Anion gap metabolic acidosis** begins to develop about 12 hours later as high concentrations of salicylates increase lipolysis, uncouple oxidative phosphorylation, and inhibit the citric acid cycle. This results in the accumulation of organic acids in the blood (eg, ketoacids, lactate, pyruvate), which bind bicarbonate, thereby increasing the calculated anion gap.

Because this patient is presenting >12 hours after salicylate ingestion, it is likely that the initial primary respiratory alkalosis has progressed to **mixed respiratory alkalosis/metabolic acidosis**. The **arterial blood gas** (ABG) in salicylate toxicity often shows a pH in the normal range as the 2 primary acid-base disturbances shift the pH in opposite directions. In addition, the ABG will show a low PaCO₂ (due to respiratory alkalosis and compensation for metabolic acidosis) and low HCO₃⁻ (due to metabolic acidosis). The PaCO₂ will be lower than predicted respiratory **compensation** (in this case, expected PaCO₂ = [1.5 * 12] + 8 ± 2 = 26 ± 2 = 24-28 mm Hg) due to the concurrent primary respiratory alkalosis.

(Choice A) These values represent a primary metabolic acidosis with appropriate respiratory compensation (eg, expected PaCO₂ = [1.5 * 16] + 8 ± 2 = 32 ± 2 = 30-34 mm Hg). Compensatory responses do not correct the pH completely.

(Choice C) These values are indicative of a normal ABG.

(Choice D) These values represent a primary metabolic alkalosis with respiratory compensation. Although patients with significant vomiting may develop a mild contraction metabolic alkalosis, the metabolic acidosis due to salicylate intoxication would predominate.

(Choice E) These values represent a primary respiratory alkalosis, which can be seen in early salicylate intoxication; they would be unlikely >12 hours after a massive ingestion.

Educational objective:

Aspirin intoxication should be suspected in a patient with the triad of fever, tinnitus, and tachypnea. Adults with aspirin toxicity initially develop a

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(Choice A) These values represent a primary metabolic acidosis with appropriate respiratory compensation (eg, expected PaCO₂ = $[1.5 \times 16] + 8 \pm 2 = 32 \pm 2 = 30-34$ mm Hg). Compensatory responses do not correct the pH completely.

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(Choice E) These values represent a primary respiratory alkalosis, which can be seen in early salicylate intoxication; they would be unlikely >12 hours after a massive ingestion.

Educational objective:

Aspirin intoxication should be suspected in a patient with the triad of fever, tinnitus, and tachypnea. Adults with aspirin toxicity initially develop a primary respiratory alkalosis followed by a mixed respiratory alkalosis and anion gap metabolic acidosis. When the PaCO₂ and HCO₃⁻ are outside the normal range, a normal pH suggests a mixed acidosis/alkalosis disorder, as compensatory responses do not correct the pH completely.

References

- Salicylate intoxication: a clinical review.

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Correct answer

50%

Answered correctly

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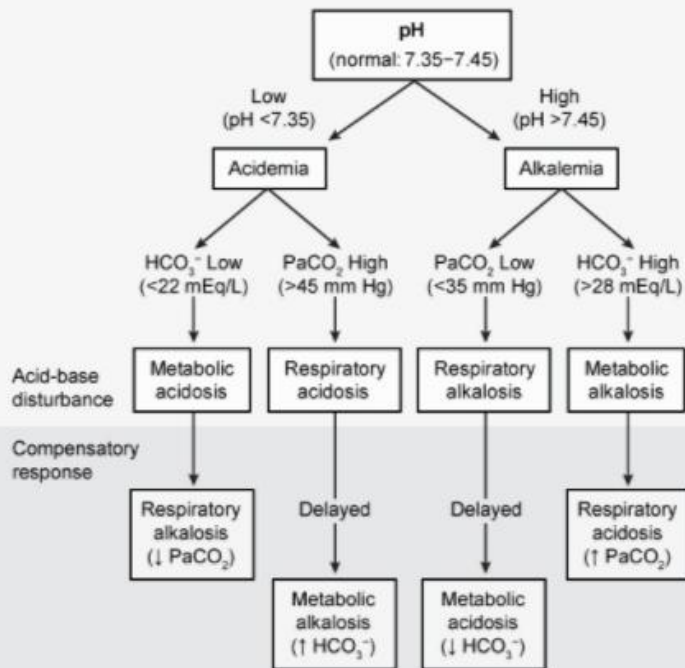
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Arterial blood gas interpretation of primary acid-base disorders

HCO₃⁻ = bicarbonate; PaCO₂ = partial pressure of carbon dioxide in arterial blood.

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TUTOR

Settings

1

Item 1 of 1

Question Id: 1544

Correct answer B

Mark

Previous

Next

Tutorial

Lab Values

Notes

Calculator

Reverse Color

Text Zoom

Explanation

Acute salicylate intoxication tachypnea within several hours

- Respiratory alkalosis** of loss of CO₂ in the expired
- Anion gap metabolic acidosis** (oxidative phosphorylation lactate, pyruvate), which

Because this patient is presenting **mixed respiratory alkalosis/m** the 2 primary acid-base disturbances and compensation for metabolic **compensation** (in this case, ex

(Choice A) These values represent $\pm 2 = 32 \pm 2 = 30-34$ mm Hg).

(Choice C) These values are indicative of a normal ABG.

(Choice D) These values represent a primary metabolic alkalosis with respiratory compensation. Although patients with significant vomiting may develop a mild contraction metabolic alkalosis, the metabolic acidosis due to salicylate intoxication would predominate.

Acid-base disorders

Primary disorder	Appropriate compensation
Metabolic acidosis	$\text{PaCO}_2 = 1.5 (\text{serum HCO}_3) + 8 \pm 2$
Metabolic alkalosis	$\uparrow \text{PaCO}_2$ by 0.7 mm Hg for every 1 mEq/L rise in serum HCO ₃
Acute respiratory acidosis	Acute: $\uparrow$ Serum HCO ₃ by 1 mEq/L for every 10 mm Hg rise in PaCO ₂ Chronic: $\uparrow$ Serum HCO ₃ by 3-4 mEq/L for every 10 mm Hg rise in PaCO ₂
Acute respiratory alkalosis	Acute: $\downarrow$ Serum HCO ₃ by 2 mEq/L for every 10 mm Hg decrease in PaCO ₂ Chronic: $\downarrow$ Serum HCO ₃ by 5 mEq/L for every 10 mm Hg decrease in PaCO ₂

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