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**Question 1**

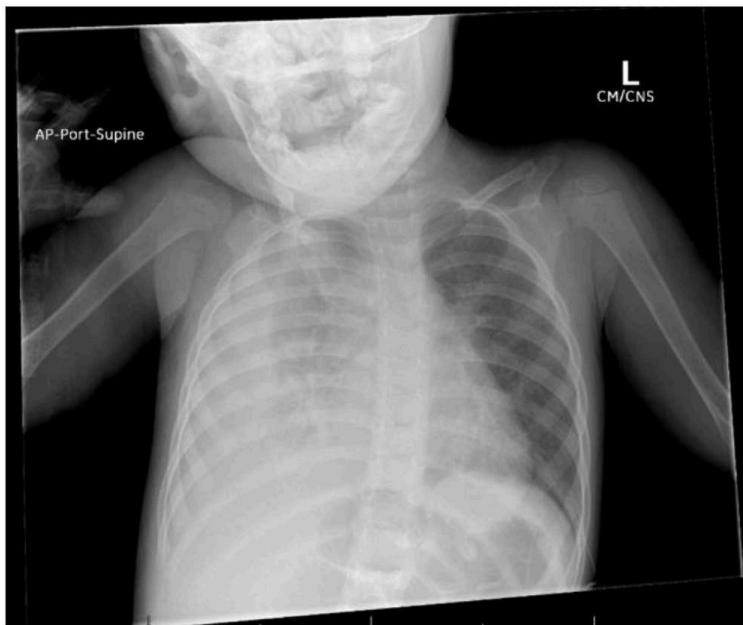
Correct

1.00 points out of 1.00

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A 2-year-old child presents with a 3-day history of cough, runny nose, malaise, and high fevers. This morning, she developed shortness of breath and was taken to the emergency department. She has a temperature of 40.1 °C, heart rate of 154 beats/min, blood pressure of 108/50 mm Hg, respiratory rate of 42 breaths/min, and pulse oximeter saturation is 82% in room air. She is placed on a 100% nonrebreather facemask, and her oxygen saturations increase to 88%. On physical examination, she has decreased breath sounds on the right, with retractions and grunting. A chest radiograph is obtained (**Figure**). She is admitted to the pediatric intensive care unit where she is placed on bilevel positive air pressure. Her saturations increase to 92%. Chest ultrasonography reveals a large right-sided simple pleural effusion. She is sedated with ketamine, and a right chest tube is placed percutaneously in the midaxillary line at the sixth intercostal space under ultrasonographic guidance. The chest tube is attached to a drainage system at -20cm H<sub>2</sub>O suction, and 1.5 L of straw-colored fluid is immediately drained from the pigtail catheter. There is no bubbling seen in the water chamber.

**Figure.** Chest radiograph of the patient in the vignette.



Courtesy of A. Au

Twenty minutes later, the patient develops increased work of breathing with desaturation to 88%, hypotension, and blood pressure of 62/32 mm Hg. On examination, the patient has crackles on the right lung, cardiac examination has a regular rate and rhythm with normal S<sub>1</sub> and S<sub>2</sub>, and the abdomen is soft, nontender, and nondistended.

Of the following, the MOST likely cause of this patient's clinical decompensation is

- A. hemothorax

- B. hepatic injury
- C. pneumothorax
- D. re-expansion pulmonary edema ✓

Your answer is correct.

### PREP Pearl(s)

- Re-expansion pulmonary edema can occur immediately or up to 24 hours following lung re-expansion with chest tube placement.
- Chest tube should be inserted between the third to fifth intercostal space in the mid to anterior axillary line, over the top of the rib.
- Complications of chest tube placement include pneumothorax, hemothorax, injury to the diaphragm, liver, or spleen, or development of a bronchopulmonary fistula.

### Critique

The patient in this vignette has re-expansion pulmonary edema. This may occur following reinflation of a collapsed lung, as seen following chest tube drainage of a pleural effusion or pneumothorax. The pathophysiological mechanism of this phenomena is unclear, but may be attributed to ischemia reperfusion injury to the lung during re-expansion, or disruption of vascular permeability, lymphatic flow, or surfactant production.

Re-expansion pulmonary edema typically occurs immediately to 1 hour following re-expansion of the lung, but may occur up to 24 hours later. Clinically, patients develop tachypnea, cough, or chest pain, and in severe cases become hypoxemic with associated hypotension. Treatment of re-expansion pulmonary edema is supportive. Prevention of re-expansion pulmonary edema may involve slowing the time of fluid or air drainage and reducing or eliminating the application of negative pressure.

Chest tube insertion is typically performed between the third to fifth intercostal space in the mid to anterior axillary line. The fourth intercostal space can be identified by the level of the nipple. The needle should be inserted over the top of the rib to avoid injuring the neurovascular bundle. Complications of chest tube placement include pneumothorax, hemothorax, injury to the diaphragm, liver, or spleen, or development of a bronchopulmonary fistula.

The chest tube in this scenario was placed at the sixth intercostal space. Placing a chest tube in this position increases the risk of injuring the diaphragm, liver, or spleen. However, hepatic injury would not account for the clinical decompensation in this vignette. The patient does not have a hemothorax, as the chest tube drainage was not bloody. The patient does not have a pneumothorax, as there is no evidence of air leak given the lack of bubbling in the water chamber.

### Suggested Reading(s)

Hirsch AW and Nagler J. Reexpansion pulmonary edema in pediatrics. *Pediatr Emerg Care*. 2018;34(3):216-220. doi:[10.1097/PEC.0000000000001435](https://doi.org/10.1097/PEC.0000000000001435)

Filosso PL, Guerrera F, Sandri A, Roffinella M, Solidoro P, Ruffini E, Oliaro A. Errors and complications in chest tube placement. *Thorac Surg Clin*. 2017;27(1):57-67. doi:[10.1016/j.thorsurg.2016.08.009](https://doi.org/10.1016/j.thorsurg.2016.08.009)

### Content Domain

- Critical Care, Medical Procedures (advanced)

## Learning Objectives

- Recognize the landmarks for chest tube insertion
- Review the complications of chest tube insertion

The correct answer is: re-expansion pulmonary edema

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**Question 2**

Correct

1.00 points out of 1.00

[Comment](#)


A 3-week-old infant weighing 5 kg has been receiving high-flow high-humidity nasal cannula (HFHH-NC) oxygen therapy for respiratory syncytial virus bronchiolitis on the regular pediatric unit for 6 hours. She has been receiving nothing by mouth and maintenance intravenous (IV) fluids during this time. Urine output has been 1.2 mL/kg/hour and capillary refill time is 2 seconds. A rapid response is called after the infant develops apnea. She is briefly stabilized with suctioning, increased high flow, and fraction of inspired oxygen (FiO<sub>2</sub>), but her mental status and respiratory effort continue to decline. The decision is made to intubate the infant.

Initial vital signs reveal a temperature of 39.1 °C, blood pressure of 75/45 mm Hg, pulse of 185 beats/min, respiratory rate of 65 breaths/min, and oxygen saturation of 85% while breathing 10 L/min of HFHH-NC oxygen at an FiO<sub>2</sub> of 1.0.

Intubation medications are ketamine 2 mg/kg IV, fentanyl 1 µg/kg IV, and rocuronium 1 mg/kg IV. Intubation is challenging due to copious secretions, and the patient requires bag and mask ventilation between the 2 attempts. A 3.5 mm cuffed endotracheal tube is utilized, and the patient is successfully intubated. She is placed on the following ventilator settings:

Tidal volume	45 mL
Positive end-expiratory pressure	10 cm H <sub>2</sub> O
Pressure support	10 cm H <sub>2</sub> O
Ventilation rate	30
FiO <sub>2</sub>	0.5

The patient's blood pressure 5 minutes after intubation is 51/30 mm Hg, with a heart rate of 150 beats/min. Of the following, the BEST explanation for this patient's hypotension is

- A. cardiac dysfunction due to viral infection
- B. decreased venous return 
- C. dehydration due to poor oral intake
- D. ketamine induced catecholamine depletion

Your answer is correct.

**PREP Pearl(s)**

- Changes in intrathoracic pressure affect systemic venous return and right ventricular preload.

- Increased right atrial pressure causes decreased venous return with varying effects on cardiac output, depending on volume status, cardiac function, and systemic blood pressure.
- Positive pressure ventilation reduces venous preload that can affect cardiac output, whereas negative pressure breathing promotes venous return.

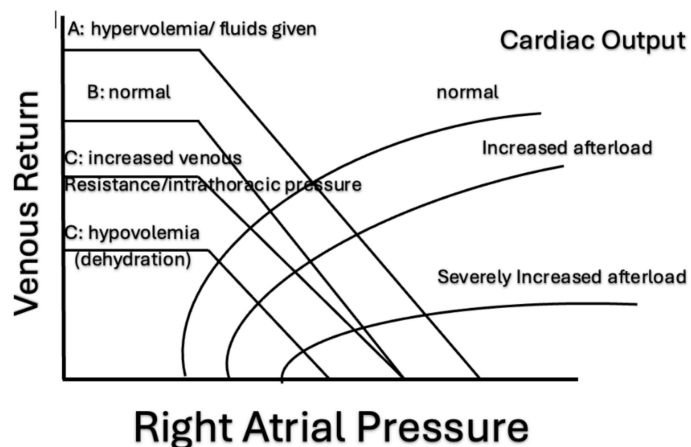
## Critique

The return of systemic venous blood back to the heart is the principal determinant of right ventricular preload. In 1954, Guyton proposed a model for venous return and defined its determinants, including the “mean systemic filling pressure,” also known as mean systemic venous pressure. Under most conditions, the mean systemic venous pressure also dictates steady-state cardiac output. Following a negative pressure gradient, blood redistributes from the venous system to the right atrium of the heart. Systemic venous return is therefore defined by the following equation:

$$\text{systemic venous return} = \frac{P_{MSV} - P_{RA}}{R_{VR}},$$

where  $P_{MSV}$  is the mean systemic venous pressure,  $P_{RA}$  is the right atrial pressure, and  $R_{VR}$  is the resistance to flow in the systemic veins. Therefore, venous return to the heart is determined by the difference between the mean systemic pressure and the right atrial pressure. As seen in the **Figure** (as noted by point C on the venous return curve), as right atrial pressure increases, there is a linear decrease in systemic venous return, whereas lower right atrial pressures facilitate increased venous return, and therefore, increased cardiac output. Venous return is maximal at a right atrial pressure of zero. In addition, a rise in mean systemic venous pressure (ie, blood pressure) also increases venous return by this equation. It is important to note that at right atrial pressures less than zero, systemic venous return is limited by increases in venous resistance because the vena cavae are very compliant and collapse at entry into the thoracic cavity.

Figure. Effect of mechanical ventilation on venous return and cardiac output.



Courtesy of E. Reade

Interactions between the cardiovascular and respiratory systems influence systemic venous return. The distinction between spontaneous and mechanical ventilation is important; during spontaneous inspiration, intrathoracic pressure decreases as the diaphragm descends. Right atrial (RA) pressure also decreases, resulting in increased venous return according to the relationship above. This phenomenon is described as

the “thoracic pump.” Mechanical positive pressure ventilation (PPV) changes this paradigm with inspiration, causing increased intrathoracic pressure, increased RA pressure, and decreased venous return. This response is exaggerated in an already hypovolemic patient, such as a patient with vasodilation due to sepsis or a profoundly dehydrated patient (preload dependent). Again, as  $\text{venous return} = P_{MSV} - P_{RA}$ , patients with lower  $P_{MSV}$  are more sensitive to significant increases in  $P_{RA}$  when intubated and provided PPV.

The dominant effects of mechanical ventilation are decreased RV preload and decreased LV afterload. However, multiple factors influence this relationship. In patients with cardiac failure, they likely have a high  $P_{MSV}$ . In these patients, the reduction in left ventricular afterload from PPV may be more significant than the potential decrease in venous return and RV preload, and may actually improve cardiac output.

Decreased venous return due to positive pressure ventilation is the best explanation for the hypotension in the patient described in the vignette. Positive pressure ventilation with relatively high tidal volume and positive end-expiratory pressure (PEEP), transmitted to the right atrium, increases right atrial pressure ( $P_{RA}$ ) and impedes venous return. Decreased venous return is essentially due to an increase in right ventricular afterload in this case. With higher levels of PEEP (thought to be  $>10$  cm  $H_2O$ ) in animal models, there is likely an increase in RVr due to flow limitation and the collapse of the superior and inferior vena cavae as they enter the right atrium.

Cardiac dysfunction due to viral infection would not explain the patient’s hypotension as described in the vignette, because there is no evidence that the patient has cardiac dysfunction related to her RSV infection. Dehydration due to poor oral intake would also not explain the hypotension in this case because the patient appears to be fairly well hydrated, as evidenced by normal urine output, adequate blood pressure for age just prior to intubation, and capillary refill time of 2 seconds. This patient may be mildly hypovolemic, but this is not the primary cause of her acute hypotension. Ketamine generally causes elevation of heart rate and blood pressure due to dose-dependent stimulation of the central nervous system and increased sympathetic outflow. However, it is also a direct negative inotrope. There are case reports of cardiovascular collapse due to catecholamine depletion in critically ill patients with depletion of endogenous catecholamines, but these are rare and usually seen only with higher doses of ketamine than used here.

## Suggested Reading(s)

Bronicki RA, Ahmed M, Flores S, Fuhrman BP. Cardiopulmonary interactions. In: *Fuhrman and Zimmermans’ Pediatric Critical Care*. 6th ed. Elsevier; 2022.

Funk DJ, Jacobsohn E, Kumar A. The role of venous return in critical illness and shock; part I: physiology. *Crit Care Med*. 2013;41(1):255-262. doi:[10.1097/CCM.0b013e3182772ab6](https://doi.org/10.1097/CCM.0b013e3182772ab6)

Funk DJ, Jacobsohn E, Kumar A. Role of the venous return in critical illness and shock; part II: shock and mechanical ventilation. *Crit Care Med*. 2013;41(2):573-9. doi:[10.1097/CCM.0b013e31827bfc25](https://doi.org/10.1097/CCM.0b013e31827bfc25)

Persichini R, Lai C, Teboul JL, Adda I, Guérin L, Monnet X. Venous return and mean systemic filling pressure: physiology and clinical applications. *Crit Care*. 2022;26(1):150. doi:[10.1186/s13054-022-04024-x](https://doi.org/10.1186/s13054-022-04024-x)

## Content Domain

- Physiology, Cardiovascular

## Learning Objectives

- Recognize the venous return curve and factors that increase or decrease venous return and cardiac output

The correct answer is: decreased venous return

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**Question 3**

Correct

1.00 points out of 1.00

[Comment](#)

An 11-year-old female with chronic headaches has had worsening vision over the past month. She went to the optometrist for an eye examination and was found to have papilledema. She has now been admitted to the pediatric intensive care unit for neurologic monitoring after brain imaging revealed no hydrocephalus or mass. Her lumbar puncture had an opening pressure of 35 cm H<sub>2</sub>O. On examination, she is alert and conversant but continues to report headache and diplopia. Her vital signs are a heart rate of 76 beats/min, a respiratory rate of 14 breaths/min, blood pressure of 98/66 mm Hg. She weighs 84 kg, and is 162 cm tall (body mass index is 32). She has a sexual maturity rating of 2. She has been well except for moderate to severe acne that improved markedly with treatment over the past month, including taking oral minocycline and applying topical retinoic acid daily.

Of the following, the factor that likely has the MOST impact on her condition is

- A. body mass index >30
- B. female sex
- C. minocycline ✓
- D. topical retinoic acid

Your answer is correct.

**PREP Pearl(s)**

- Tetracycline and its derivatives are the most common medication cause of pseudotumor cerebri in prepubertal children.
- Risk factors for pseudotumor cerebri change for children after puberty include obesity, female sex, and polycystic ovary syndrome.
- If the opening pressure is above 52 cm H<sub>2</sub>O, then surgical interventions may be necessary.

**Critique**

Pseudotumor cerebri (also known as idiopathic intracranial hypertension) is a relatively rare disease in children. Incidence is estimated to be 0.6-0.71/100,000 which is one-third of the rate seen in adults. However, there are important differences between risk factors for adults and children. In adults, recent weight gain, female sex (postpubertal), obesity, and polycystic ovary syndrome are all risk factors for primary pseudotumor cerebri syndrome. However, in children 12 years of age and younger, there is no difference among males and females, and being thin is a risk factor as opposed to obesity in postpubertal females. These differences likely reflect the importance of the pubertal state as a risk-stratifying factor.

Most younger children develop pseudotumor cerebri as a result of secondary causes. Medications are the largest category of factors associated with secondary pseudotumor cerebri, with tetracycline and its derivatives (minocycline and doxycycline) being the most frequently indicated. Lithium and vitamin A derivatives are also associated with the condition when taken in the oral form. Isotretinoin is given for the

treatment of cystic acne but requires a negative pregnancy test and close monitoring. There have been a few case reports of topical retinol potentially causing pseudotumor cerebri. However, oral administration of transretinoic acid or isotretinoin are more commonly noted with this condition. These agents should not be given in combination with tetracycline or its derivatives, since this can lead to irreversible vision loss if pseudotumor cerebri develops when these medications are taken together.

Genetic diseases such as Turner syndrome and trisomy 21 are both risk factors for pseudotumor cerebri. In addition, there are many vascular conditions that can cause secondary pseudotumor cerebri. These conditions include central sinus venous thrombosis, mastoiditis, superior vena cava syndrome, arteriovenous fistula, and prior intraventricular infection or hemorrhage. However, the incidence of pseudotumor cerebri in these conditions is much lower than what is seen with medications.

Documenting the initial lumbar puncture opening pressure is important since although most cases of pediatric pseudotumor cerebri respond to stopping the medication, in more severe cases, additional treatment may be needed. The mainstay of treatment is reduction of intracranial pressure with drainage of cerebrospinal fluid and initiation of acetazolamide therapy. Acetazolamide is a carbonic anhydrase inhibitor and is thought to reduce the production of cerebrospinal fluid. Papilledema has been shown to more effectively improve with acetazolamide therapy than headaches. If headaches persist, then addition of a loop diuretic (eg, furosemide) may be needed. If the opening pressure is above 52 cm H<sub>2</sub>O, surgical interventions may be necessary. This is particularly relevant when the secondary cause is due to prior intraventricular hemorrhage or there is severe papilledema. These cases have a higher failure rate with medication or lumbar drainage alone, and optic nerve sheath fenestration may be needed to reduce papilledema. Risk factors such as vascular ischemia have kept this procedure from becoming standard of care, and there is variation between centers in their surgical approach to this condition. All of these patients require close monitoring and subsequent follow-up until their papilledema and vision have stabilized.

## Suggested Reading(s)

- Barmherzig R, Szperka CL. Pseudotumor cerebri syndrome in children. *Curr Pain Headache Rep.* 2019;23(8):58. doi:[10.1007/s11916-019-0795-8](https://doi.org/10.1007/s11916-019-0795-8)
- Beres SJ. Update in pediatric pseudotumor cerebri syndrome. *Semin Neurol.* 2020;40(3):286-293. doi:[10.1055/s-0040-1708847](https://doi.org/10.1055/s-0040-1708847)

## Content Domain

- Critical Care, Neurological disorders and support

## Learning Objectives

- Recognize how different factors affect the likelihood of pseudotumor cerebri at different ages

The correct answer is: minocycline

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**Question 4**

Correct

1.00 points out of 1.00

[Comment](#)

A 14-year-old 40-kg adolescent born prematurely presents for an elective posterior spinal fusion extending from T4 to L4. History is remarkable for idiopathic scoliosis and gastrostomy tube. Their preoperative Cobb angle was 80°. The estimated blood loss is 1L. They receive a total of 30 mL/kg of packed red blood cells and approximately 250 mL of salvaged red blood cells during the case. The anesthesia team keeps them intubated following the case, and they are brought to the pediatric intensive care unit.

The patient is receiving a fentanyl infusion at 2 µg/kg/hour for pain control; the patient is hemodynamically stable, hemoglobin is 9 mg/dL, and they have a negative pressure dressing. They remain nothing-by-mouth overnight, and are extubated the following day to high-flow, high-humidity nasal cannula oxygen therapy. The pain control is converted from continuous fentanyl to a morphine patient-controlled-anesthesia (PCA) with a basal rate, and scheduled ketorolac and acetaminophen. The high-flow oxygen therapy is weaned over 24 hours from 20 L of oxygen per minute (LPM) to 10 LPM. On postoperative day 3, the patient's home gastrostomy feedings are restarted at 5 mL/hour.

On postoperative day 4, the patient develops abdominal distention and vomiting, and has not yet stoolled since the operation. The abdominal radiograph reveals multiple distended loops of small bowel, moderate stool burden in the descending colon, and no free intraperitoneal air.

Of the following, the factor that would MOST likely decrease the risk of feeding intolerance in this patient is

- A. changing morphine PCA to hydromorphone PCA
- B. initiating gastrostomy tube feeds on postoperative day 1 ✓
- C. scheduled methylnaltrexone
- D. use of bilevel positive pressure ventilation

Your answer is correct.

**PREP Pearl(s)**

- While a seemingly benign complication, postoperative ileus in patients with neuromuscular scoliosis undergoing posterior spinal fusion can lead to prolonged fasting times, need for parenteral nutrition, and respiratory compromise from abdominal distention. This can delay wound healing, mobilization, and hospital discharge.
- Patients with spastic quadriplegia or cerebral palsy who have gastrostomy tubes, high intraoperative blood loss, more severe curvature, and more extensive spinal fusions are at increased risk for gastrointestinal complications following spinal fusion.
- Earlier introduction of enteral feeding have been shown to decrease the incidence of postoperative ileus in surgical patients.

**Critique**

Postoperative ileus is defined as an abnormal pattern of gastrointestinal motility characterized by nausea and vomiting, the inability to tolerate enteral nutrition with abdominal distention, and delayed passage of flatus and stool. Ileus is the most common postoperative gastrointestinal complication of posterior spinal fusion surgery. Other gastrointestinal complications following spinal fusion include pancreatitis, superior mesenteric artery (SMA) syndrome, and Olgivie syndrome. Ileus often leads to delayed patient recovery from surgery, postoperative morbidity, increased hospital length of stay, and higher hospitalization costs. From a clinical standpoint, ileus may cause respiratory distress due to abdominal distention, prohibit enteral feeding, lead to parenteral nutrition, delay wound healing, and decrease participation in rehabilitation therapies.

Studies have examined risk factors for postoperative complications after posterior spinal fusion in pediatric patients, including specifically those with neuromuscular scoliosis. One 2020 prospective multicenter study of 425 children with cerebral palsy undergoing posterior spinal fusion found the following factors increased the odds of gastrointestinal complications:

- Being gastrostomy tube fed
- Blood loss over 3 mL/kg/level of fusion
- Larger Cobb angle > 70°
- Higher number of spinal levels fused

In general, narcotics decrease intestinal motility, and minimizing postoperative narcotic use in favor of nonsteroidal anti-inflammatory medications, acetaminophen, and in some cases epidural anesthesia, is an important part of clinical pathways for spinal fusion recovery. However, there is little evidence that there is any difference in rates of ileus or opioid-induced constipation seen with morphine or hydromorphone. Therefore, changing morphine PCA to hydromorphone PCA would not be the best choice.

Methylnaltrexone is a pure peripheral  $\mu$ -opioid antagonist that is FDA-approved for the treatment of opioid-induced constipation in adults. Most experience with this medication is in adults on chronic opioid therapy, particularly in oncology patients. There are case reports describing its use in children in the oncology/palliative care population as well as one brief report detailing its use in two intensive care unit patients with cancer. There may be future applications for this medication in this population. However, this medication is not currently indicated in postoperative ileus, and there are no studies describing its use as a preventative agent. Therefore, scheduled methylnaltrexone would not be the factor among the given options most likely to reduce feeding intolerance.

Use of bilevel positive pressure ventilation for post-extubation respiratory support would likely cause increased abdominal distention due to aerophagia and exacerbate ileus. High-flow high-humidity nasal cannula therapy at the flow rates described in this patient (1 L/kg/min initially, weaned to 0.5 L/kg/min) are not thought to distend the lower esophageal sphincter.

Thus, initiating gastrostomy tube feeds on postoperative day 1 is the factor most likely to reduce feeding intolerance, because a shorter fasting time and earlier introduction of enteral feeding have been shown to decrease the incidence of postoperative ileus in surgical patients. The 2017 Consensus-Based Best Practice Guidelines for postoperative care following posterior spinal fusion for adolescent idiopathic scoliosis recommend that clear liquids be started immediately postoperatively, and that the patient's regular diet may be started as soon as the patient is tolerating liquids regardless of the presence of bowel sounds or other clinical criteria. Benefits of early enteral feeding after major surgery include the earlier return of bowel sounds and passage of flatus as well as faster time to first bowel movement. By the time this patient was started on low-volume feeds, they had likely been nothing-by-mouth for at least 72 hours. While it was reasonable to leave the patient nothing-by-mouth while intubated overnight with the intent to extubate the following day, withholding enteral feeds well after extubation, combined with the use of opioids and the requirement of gastrostomy tube feedings likely contributed to the development of ileus.

## Suggested Reading(s)

Fletcher N, Glotzbecker M, Marks M, Newton P. Development of consensus-based best practice guidelines for postoperative care following posterior spinal fusion for adolescent idiopathic scoliosis. *SPINE*. 2017;42(9):E547-E554. doi:[10.1097/brs.0000000000001865](https://doi.org/10.1097/brs.0000000000001865)

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Sundar S, Enders J, Bailey K, et al. Use of a standardized perioperative care path for adolescent idiopathic scoliosis leads to decreased complications and readmissions. *Clin Spine Surg*. 35(1):E41-E46. doi:[10.1097/bsd.0000000000001236](https://doi.org/10.1097/bsd.0000000000001236)

Verhofste BP, Berry JG, Miller PE, et al. Risk factors for gastrointestinal complications after spinal fusion in children with cerebral palsy. *Spine Deform*. 2021;9(2):567-578. doi:[10.1007/s43390-020-00233-y](https://doi.org/10.1007/s43390-020-00233-y)

## Content Domain

- Gastroenterology, Gastrointestinal Motility

## Learning Objectives

- Recognize the factors associated with development of postoperative ileus

The correct answer is: initiating gastrostomy tube feeds on postoperative day 1

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**Question 5**

Correct

1.00 points out of 1.00

[Comment](#)

A previously healthy 17-year-old adolescent presents as a direct admission to the pediatric intensive care unit due to altered mental status. Parents report that he was found in the garage with his friends as they were spraying spray paint into brown paper bags and inhaling the vapor as a recreational activity, reportedly to get a "buzz." The boy began to have more unusual behavior, and ultimately became significantly altered and confused with slurred speech and depressed consciousness. Examination shows an altered mental status with a Glasgow Coma Score of 10. His vital signs include a temperature of 98.7 °F, heart rate of 120 beats/min, respiratory rate of 24 breaths/min, blood pressure 125/85 mm Hg, and SPO<sub>2</sub> 96%. He has a faint ring of paint around his lips and nose. Lungs are clear to auscultation bilaterally with scant wheezing but no distress. Cardiac examination is without murmur. Neurologic examination reveals a disoriented patient with no focal neurologic findings otherwise.

Of the following, the MOST severe complication commonly reported as a result of this condition is

- A. cardiac dysrhythmia ✓
- B. cerebral edema
- C. hepatic failure
- D. renal failure

Your answer is correct.

**PREP Pearl(s)**

- Intentional hydrocarbon intoxication via inhalation is a common method for adolescents to achieve a "high" as a result of the chemical psychoactive properties.
- Known as huffing, sniffing, or bagging, hydrocarbon inhalation intoxication can lead to neurologic derangements including altered mental status and coordination deficits, and in severe intoxication, even coma.
- Inhaled hydrocarbons can sensitize the myocardium to endogenous and exogenous catecholamines, and predispose patients to fatal ventricular arrhythmias.

**Critique**

Intentional inhalational hydrocarbon intoxication and abuse among adolescents poses a significant public health concern with more than 2 million adolescents endorsing this type of behavior at least once. Adolescent misuse of volatile hydrocarbons for their psychoactive properties is a well-documented phenomenon. Several factors contribute to this, including affordability, widespread availability, and ease of administration of these substances. Volatile hydrocarbons are found in numerous household products such as glues, solvents, lighter fluid, gasoline, and spray paints. Virtually any pressurized aerosol product can be abused due to the presence of volatile hydrocarbon propellants. The most common methods of inhalation

include sniffing concentrated fumes directly (sniffing glue), holding soaked rags to the face, or inhaling from a secondary container like a brown paper bag (huffing or bagging). The child in the vignette has a history consistent with huffing or bagging spray paint.

A life-threatening condition known as “sudden sniffing death syndrome” occurs after the user is startled during or soon after inhalation and results in acute cardiac arrhythmia and death, which is the correct response above. The inhaled hydrocarbons sensitize the myocardium to endogenous and exogenous catecholamines, which can precipitate ventricular arrhythmia and sudden death. There are also numerous case reports which detail acute myocardial infarction as a complication following hydrocarbon inhalation, likely induced from coronary artery spasm, platelet aggregation, and thrombus formation. For these reasons, some advocate the avoidance of  $\beta$ -agonists, including albuterol, for patients with bronchospasm with suspected hydrocarbon inhalation.

Inhalation intoxication and injury from hydrocarbon fumes primarily impact two organ systems, including the central nervous system and pulmonary system. The hydrocarbon fumes are very lipophilic, allowing them to quickly pass from the lungs into the bloodstream and reach the brain within 15 to 30 minutes. Early symptoms can include confusion, feeling lost, and acting impulsively, sometimes with a sense of euphoria. Speech becomes slurred, and patients may have ataxia. In rare cases of repeated or prolonged exposure, coma may ensue. However, this is uncommon because people typically stop inhaling as drowsiness sets in. Cerebral edema resulting from inhalation of hydrocarbons has not been described.

In younger children, injury by hydrocarbon exposure tends to be the result of accidental ingestion resulting in concurrent pulmonary aspiration. Because of the low viscosity of hydrocarbon liquids, including gasoline and kerosene, these agents are easily aspirated during accidental ingestion leading to life-threatening aspiration pneumonia with severe hypoxia. Ingestion of hydrocarbon liquids can lead to gastrointestinal signs and symptoms, including nausea, vomiting, and abdominal pain. Even with small ingestions, there may be neurologic signs and symptoms, including dizziness, headache, confusion, drowsiness, and in severe cases, seizures or coma. Most hydrocarbon ingestions are in young children due to the exploratory nature of their development, and improperly stored chemical compounds in common-use containers such as empty water bottles. Although gastrointestinal distress is common, neither renal nor liver failure are commonly described as a consequence of ingestion of hydrocarbon liquids.

## Suggested Reading(s)

Hogge RL, Spiller HA, Kistamgari S, et al. Inhalant misuse reported to America’s Poison Centers, 2001–2021. *Clin Toxicol*. 2023;61(6):453-62. doi:[10.1080/15563650.2023.2216872](https://doi.org/10.1080/15563650.2023.2216872)

Kurtzman TL, Otsuka KN, Wahl RA. Inhalant abuse by adolescents. *J Adol Health*. 2001;1;28(3):170-80. doi:[10.1016/s1054-139x\(00\)00159-2](https://doi.org/10.1016/s1054-139x(00)00159-2)

Makrygianni EA, Palamidou F, Kaditis AG. Respiratory complications following hydrocarbon aspiration in children. *Ped Pulmonol*. 2016;51(6):560-9. doi:[10.1002/ppul.23392](https://doi.org/10.1002/ppul.23392)

Tormoehlen LM, Tekulve KJ, Nañagas KA. Hydrocarbon toxicity: a review. *Clin Toxicol*. 2014;52(5):479-89. doi:[10.3109/15563650.2014.923904](https://doi.org/10.3109/15563650.2014.923904)

## Content Domain

- Substance Use and Addictions

## Learning Objectives

- Recognize the complications of hydrocarbon inhalation injury
- Recognize signs and symptoms of hydrocarbon inhalation injuries

The correct answer is: cardiac dysrhythmia

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**Question 6**

Correct

1.00 points out of 1.00

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A 10-year-old child is admitted to the pediatric intensive care unit with influenza A infection and necrotizing pneumonia. They are intubated and ventilated. Despite treatment with vancomycin for 5 days, therapeutic levels of vancomycin have not been attained, and blood cultures are persistently positive for methicillin-resistant *Staphylococcus aureus* (MRSA) with high minimum inhibitory concentrations for vancomycin. Chest computed tomography scan shows necrotizing pneumonia with chest tubes in place and minimal residual pleural effusions. There is no other source of infection.

Of the following, the antibiotic MOST indicated in this situation is

- A. daptomycin
- B. doxycycline
- C. linezolid ✓
- D. trimethoprim sulfamethoxazole

Your answer is correct.

**PREP Pearl(s)**

- Vancomycin is the recommended drug of choice for the treatment of methicillin-resistant *Staphylococcus aureus* bacteremia and pneumonia.
- Linezolid, daptomycin, and ceftaroline are approved for use in methicillin-resistant *Staphylococcus aureus* bacteremia in children and can be used in combination with vancomycin.
- Daptomycin is inactivated by pulmonary surfactant and is not recommended for the treatment of methicillin-resistant *Staphylococcus aureus* pneumonia.
- Methicillin-resistant *Staphylococcus aureus* co-infection has been reported with influenza and is an independent risk factor for influenza-related mortality; these patients should be provided dual antimicrobial coverage with clindamycin and vancomycin.

**Critique**

*Staphylococcus aureus* is a common cause of bacteremia in children. Most cases are caused by methicillin-susceptible *Staphylococcus aureus* (MSSA). Methicillin-resistant *Staphylococcus aureus* (MRSA) infections are associated with more prolonged bacteremia, and higher rates of hospitalization and intensive care admissions. Methicillin-resistant *Staphylococcus aureus* co-infection has been reported with influenza and is an independent risk factor for influenza-related mortality; these patients should be provided dual antimicrobial coverage with clindamycin and vancomycin.

Vancomycin is recommended for treatment of bacteremia as well as community-acquired and hospital-acquired pneumonia due to MRSA. There is long-standing evidence of effectiveness and lack of resistance to vancomycin. However, vancomycin has poor penetration into lung tissue, is nephrotoxic, and requires

therapeutic drug monitoring. Therapeutic drug levels have been reported to be difficult to achieve in patients with good renal clearance. Clindamycin, daptomycin, ceftaroline, and linezolid are possible alternatives to vancomycin.

The use of vancomycin alternatives has been suggested in patients initially treated with vancomycin who develop vancomycin-associated nephrotoxicity, have MRSA isolates with high minimum inhibitory concentrations, or have subtherapeutic vancomycin concentrations. Combination therapy with vancomycin has been suggested in patients with influenza and acute respiratory failure and suspected MRSA-coinfection and those with persistent bacteremia who are not candidates for source control. Persistent bacteremia should trigger a reassessment for foci of infection and source control; in most cases, source control will be more beneficial than addition on antimicrobials.

The choice of an agent as a substitute for or in addition to vancomycin should be determined by the site of infection, local antibiograms, and recommendations from infectious disease subspecialists. Clindamycin, the second-most prescribed antibiotic for MRSA, has antitoxin effects in vitro, and is recommended for MRSA pneumonia in stable children without bacteremia if clindamycin resistance rate is low. Daptomycin is dosed once daily, is not nephrotoxic, and is well tolerated; its use requires monitoring of creatinine kinase. Although daptomycin is the preferred drug for combination with vancomycin in MRSA bacteremia, and although acceptable concentrations are achieved in most tissues including cerebrospinal fluid, daptomycin is inactivated by surfactant and is not recommended in the treatment of MRSA pneumonia. Ceftaroline, a  $\beta$ -lactam, is clinically approved for MRSA community-acquired pneumonia and skin and soft tissue infections, but there is a lack of pharmacokinetic and outcome data in intubated children. It has good tissue penetration, but there is insufficient data to support its use in MRSA central nervous system infections. It is well tolerated with side effects similar to other cephalosporins. Linezolid, an oxazolidinone, has excellent penetration in the lung and is well tolerated. Longer courses are associated with myelosuppression; regular monitoring of blood cell counts is recommended. It should be used with caution in patients with existing cytopenias.

The patient in this vignette is intubated for acute respiratory failure due to influenza and MRSA necrotizing pneumonia and has not achieved therapeutic vancomycin levels in 5 days, likely secondary to high renal clearance. He is at risk for treatment failure and morbidity and mortality. It is prudent to add another antimicrobial agent in this case. Linezolid is the correct choice. Daptomycin is inactivated by pulmonary surfactant. Trimethoprim sulfamethoxazole and doxycycline are not recommended in patients with bacteremia or severe pneumonia due to MRSA.

## Suggested Reading(s)

Chiusaroli L, Liberati C, Rulli L, et al. Therapeutic options and outcomes for the treatment of children with gram-positive bacteria with resistances of concern: a systematic review. *Antibiotics (Basel)*. 2023;12(2):261. doi:[10.3390/antibiotics12020261](https://doi.org/10.3390/antibiotics12020261)

Haynes AS, Maples H, Parker S. Time for a change: considering vancomycin alternatives for pediatric methicillin-resistant *Staphylococcus aureus* bacteremia. *J Ped Inf Dis Soc*. 2023;(12)5:308–318. doi:[10.1093/jpids/piad032](https://doi.org/10.1093/jpids/piad032)

Randolph AG, Xu R, Novak T, et al. Vancomycin monotherapy may be insufficient to treat methicillin-resistant *Staphylococcus aureus* coinfection in children with influenza-related critical illness. *Clin Infect Dis*. 2019;68(3):365-372. doi:[10.1093/cid/ciy495](https://doi.org/10.1093/cid/ciy495)

## Content Domain

- Infectious Diseases, Antimicrobials

## Learning Objectives

- Select the appropriate antimicrobials to treat methicillin resistant *Staphylococcus aureus* infection

The correct answer is: linezolid

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