Q.1. You have just admitted to the PICU a 2-year-old child who was born at 25 weeks’ gestation. He has chronic lung disease and is treated with oxygen 0.5 L/min per nasal cannula at home. He presented to the emergency department with a 2-day history of worse cough, congestion, and increased work of breathing. He was found to be in acute hypoxemic respiratory failure and was intubated due to severe hypoxemia and an increased work of breathing. His chest x-ray has chronic bilateral densities and a new right-sided density. He is being ventilated in pressure control pressure support with peak inspiratory pressure (PIP) = 28 cm H₂O, pressure support (PS) 16 cm H₂O, positive end-expiratory pressure (PEEP) = 8 cm H₂O, ventilator rate = 35 per minute, and fraction of inspired oxygen (FiO₂) = 0.8. His peripheral oxygen saturation (SpO₂) is 98%, and the mean airway pressure is 15 cm H₂O. Do you think he meets the criteria for pediatric acute respiratory distress syndrome (PARDS)?

A. You think he meets the criteria based on his oxygenation saturation index = (80 × 15)/98 = 12.2.

B. You think he meets the criteria but realize that he does not have ARDS because he has a new unilateral infiltrate.

C. You think he meets the criteria but realize that he does not have ARDS because of his existing chronic lung disease.

D. You think he meets the criteria but realize that you cannot determine his oxygenation saturation index or his SpO₂/FiO₂ ratio because his SpO₂ is ≥97%.

Q.2. On the third day of the hospitalization, the patient in question 1 develops progressively worse hypoxemia. His oxygen saturation index (OSI) is now 22. Data suggest that pulmonary-specific ancillary therapies that are indicated in children include which of the following?

A. Exogenous surfactant
B. High-frequency oscillatory ventilation (HFOV)
C. Inhaled nitric oxide
D. There are no data in children supporting specific pulmonary ancillary therapies.

Q.3 Which of the following is a cause of direct lung injury?

A. Near-drowning
B. Sepsis
C. Sickle cell disease
D. Transfusions

Q.4 What can prevent the development of ventilator-associated lung injury in mechanically ventilated patients?

A. The application of low tidal volumes
B. The application of large tidal volumes
C. The application of a positive end-expiratory pressure (PEEP) level of 0 cm H₂O
D. The application of frequent recruitment maneuvers

Q.5 Which statement is true regarding the ventilator management of a patient with a severe acute respiratory distress syndrome?

A. A tidal volume of 10 mL/kg of predicted body weight can be safely applied in most patients.
B. A tidal volume of 6 mL/kg of predicted body weight can be safely applied in most patients.
C. A tidal volume of 6 mL/kg of predicted body weight is safe for all patients.
D. Prone positioning has not been shown to reduce mortality.

Q.6 Which of the following is most likely to promote ventilator-induced lung injury (VILI)?
A. Airway resistance
B. End-inspiratory lung volume
C. Level of positive end-expiratory pressure (PEEP)
D. Peak inspiratory pressure

Q.7 Which is the best ventilatory strategy for a 7-year-old patient with acute respiratory distress syndrome whose static pressure-volume curve is shown in Figure.1A?
A. PEEP 5; VT 200 mL
B. PEEP 5; VT 300 mL
C. PEEP 8; VT 180 mL
D. PEEP 10; VT 180 mL

Q.8 Which of the following statements regarding near-fatal asthma is true?
A. A ventilation strategy of low respiratory rates (<12 breaths/min), moderate-to-high tidal volumes (8–12 mL/kg), and permissive hypercapnia has been proved to be associated with increased mortality and a rate of pneumothorax approaching 100%.
B. Increasing positive end-expiratory pressure (PEEP) in mechanically ventilated asthma patients receiving neuromuscular blockade has been shown to have unfavorable effects on lung volumes, airway pressure, and hemodynamics.
C. Ketamine is contraindicated during intubation due to its slow onset of action and tendency to cause bronchoconstriction.
D. Nearly all subjects have a history of severe persistent asthma with frequent ICU admissions in the 1 year preceding the episode of near-fatal asthma

Q.9 Which of the following is the predominant mechanism by which terbutaline causes bronchodilation?
A. Activating the β-2 receptor, which increases cyclic adenosine monophosphate (cAMP) levels by augmenting its synthesis by adenylate cyclase
B. Blocking the acetylcholine receptor, reducing the level of cyclic guanosine monophosphate (cGMP)
C. Blocking the N-methyl-d-aspartate receptors in airway smooth muscle
D. Inhibiting phosphodiesterase, which increases cAMP levels by preventing its degradation

Q.10A 10-year-old boy is admitted to the pediatric intensive care unit with status asthmaticus refractory to initial treatment in the emergency department. The patient is agitated and exhibits severe intercostal and subcostal retractions with barely audible breath sounds. Mechanical ventilation is initiated because of hypoxemia and impending respiratory failure. Based on current best standards of care, which of the following ventilation strategies should be used after intubation?
A. Permissive hypercapnia with the use of low respiratory rates and long expiratory times to avoid dynamic hyperinflation
B. Pressure-controlled ventilation with a long inspiratory time and a short expiratory time
C. The application of high levels of positive end-expiratory pressure (10 to 15 cm H2O) to prevent derecruitment and atelectasis in the patient who has undergone neuromuscular blockade

D. The use of high tidal volumes and rapid respiratory rates to deliver supraphysiologic minute ventilation and correct the respiratory acidosis

Answers:

Q.1: D

There is a strong linear association between SpO2 and PaO2 as long as the SpO2 is ≤ 97%. The Pediatric Acute Lung Injury Consensus Conference (PALICC) definition of PARDS recommends use of the oxygenation index or oxygenation saturation index when a PaO2 is not available. Although the PALICC definition of PARDS does not require new bilateral infiltrates, it recommends future studies investigate whether bilateral versus unilateral infiltrates improves discrimination of risk stratification.

Q.2: D

There are no conclusive data supporting the specific pulmonary or pulmonary ancillary therapies in children. The most conclusive data supporting pulmonary therapy address limiting tidal volume in adults. Limiting the driving pressure and prone positioning seem to be beneficial in adults with ARDS, but there are no pediatric data. There are no studies supporting the use of exogenous surfactant, HFOV, or inhaled nitric oxide to improve mortality in children or adults.

Q.3: C

Causes of direct lung injury include pneumonia, gastric content aspiration, lung contusion, hydrocarbon ingestion, smoke inhalation, and sickle cell disease. Near drowning, multiple emergent transfusions, and sepsis lead to acute lung injury as part of multiorgan failure.

Q.4: A

The main measure that has been shown to improve prognosis in mechanically ventilated patients with the acute respiratory distress syndrome (ARDS) is tidal volume reduction to 6 mL/kg as compared to 12 mL/kg of predicted body weight. Increasing evidence suggests that such ventilator settings may also be beneficial in mechanically ventilated patients who do not meet criteria for ARDS. A PEEP level of 0 cm H2O is associated with the development of “low-volume” lung lesions and should be avoided. The other measures have not been shown to provide significant clinical benefit.

Q.5: B

The main measure that has been shown to improve prognosis in mechanically ventilated patients with the acute respiratory distress syndrome (ARDS) is tidal volume reduction to 6 mL/kg as compared to 12 mL/kg of predicted body weight. Prone positioning has also been associated with a reduction in the mortality of ARDS patients having a PaO2/FiO2 ratio < 150 mm Hg.

Q.6: B

Edema formation is now generally believed to be the hallmark of VILI. The respective roles of increased airway pressure and increased lung volume in this injury were clarified when mechanical ventilations at high and low tidal volumes (VT) were compared at identical (45 cm H2O) peak airway pressures. The injury was found only in rats subjected to high VT and not in those that underwent ventilation at high airway pressure in which lung distention was limited by thoracoabdominal strapping. Furthermore, pulmonary edema still developed in animals that underwent ventilation at high VT by negative external distending pressure, confirming that excessive airway pressure is not the causal factor of this type of injury. This VILI, which depends mostly on end-inspiratory volume, has been called volutrauma. The alveolar pressure corresponding to end-inspiratory volume is the plateau airway pressure (measured at no-flow), and its clinical importance has been emphasized in a consensus conference on mechanical ventilation. Several investigators reached the same conclusions in
other species using different protocols.

Q.7:D

![Figure 1B: Static pressure-volume curves with upper and lower inflection points.](image)

Figure 1B: Static pressure-volume curves with upper and lower inflection points.

It has been recommended that the optimal PEEP should be set above the critical closing or critical opening pressure of the airways. This pressure can be deduced by the lower inflection point generated with static pressure-volume loops (Figure 1B). As the lung is inflated from zero end-expiratory pressure, in many lungs an abrupt change in compliance occurs as denoted by the lower inflection point. It is generally thought that this is the critical opening pressure of the airways above which the alveoli and airways remain open. As the lung is further inflated in increments, the pressure-volume slope increases and then abruptly changes direction as noted in (Figure 1B), as the upper inflection point. It is generally thought that the upper inflection point reflects overdistention of the alveoli. The general recommendation is to keep the PEEP level above the lower inflection point and to keep the end-inspiratory pause pressure below the upper inflection point. Currently, bedside use of static pressure-volume loops to set PEEP is not a standard practice in infants and children. Therefore the level of PEEP should be set by titrating the level of PEEP and selecting the level by the maximal level of improvement in oxygenation compliance seen without affecting systemic hemodynamics. The repeated collapse and reopening of the lung units at low lung volume have been shown to contribute to VILI. A strategy combining recruitment maneuvers, low VT, and higher PEEP has been shown to decrease the incidence of barotrauma or volutrauma. For the child in the vignette, the upper inflection point occurs with a VT of 200 mL, so a lung protective strategy would maintain the VT below 200 mL.

Q.8:B

Tuxen established in 1989 that increasing PEEP in patients with airway obstruction who are receiving neuromuscular blockade is associated with unfavorable increases in hyperinflation and intrathoracic pressures and frequent decreases in systemic blood pressure and venous oxygen saturation. In our practice, we set PEEP to zero in mechanically ventilated asthmatic patients during neuromuscular blockade (barring extenuating circumstances) and use minimal PEEP (less than auto-PEEP and not more than 8 cm H2O) during spontaneous breathing. Mortality from near-fatal asthma is approximately 4% in the United States. In a study by the Collaborative Pediatric Critical Care Research Network, 11 fatalities were observed out of 261 children with near-fatal asthma (4.2%). Of these 11 children, 10 had suffered a cardiac arrest prior to PICU admission. In that same publication the authors reported that 13% of subjects had no prior history of asthma (Answer D) and that only 29% of patients had an admission for asthma in the preceding year. Similarly, among 51 subjects who died from asthma in Australia, 32% of subjects had never been admitted to the hospital because of asthma prior to their death. Ketamine is commonly employed when intubating a child with near-fatal asthma (Answer C) due to its rapid onset, bronchodilatory effects, and beneficial hemodynamic outcomes. As described in the text, we suggest a ventilatory strategy that employs relatively low respiratory rates (6-12 breaths/min), tidal volumes of 8 to 12 mL/kg, and permissive hypercapnia (Answer A).
a similar strategy was used in 26 subjects, all patients survived and pneumothorax was uncommon.

Q.9:A
An increase in cyclic adenosine monophosphate (cAMP) levels through adenylate cyclase–mediated synthesis is the predominant mechanism by which the β-agonists cause bronchodilation. Answer B is the mechanism by which anticholinergic medications (eg, ipratropium bromide) work. Answer C is one mechanism by which ketamine causes bronchodilatation. The methylxanthines (eg, aminophylline) also cause bronchodilatation by increasing cAMP. However, they work by inhibiting phosphodiesterase 4 and preventing the breakdown of cAMP (Answer D).

Q.10:A
Mechanical ventilation of a child with asthma should follow a strategy based on long expiratory times, slow respiratory rates, and permissive hypercapnia to avoid dynamic hyperinflation. Attempts at normalizing arterial blood gases by delivering supraphysiologic minute ventilation increase the risk of air leak, hemodynamic instability, and death.

References