

Pulseless Electrical Activity in a Pediatric Patient: A Case Report and Review of Causative Factors and Treatment

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Pulseless electrical activity, an arrhythmia that leads to cardiac arrest, is defined as the presence of organized electrical activity without a palpable pulse or arterial blood pressure. When this arrhythmia presents during anesthesia, it has become routine practice to initiate advanced cardiac life support according to the American Heart Association guidelines. This arrhythmia is usually associated with a poor prognosis unless a reversible cause is investigated and treated immedi-

ately. The purpose of this article is to summarize the causative factors of pulseless electrical activity and its treatment modalities. This case report describes the successful resuscitation of a pediatric patient who presented with pulseless electrical activity during anesthesia for a rigid bronchoscopy.

Keywords: Cardiac arrest, pediatric, pulseless electrical activity.

It is estimated that approximately 16,000 American children will suffer a cardiac arrest each year.¹ Many factors contribute to the survival of the pediatric patient after a cardiac arrest. These factors include the environment where the arrest occurred, the patient's preexisting condition, the length of time with no pulsatile flow before successful resuscitation, the electrocardiographic (ECG) rhythm, and the quality of the interventions provided during the resuscitation.¹

The approach to cardiopulmonary resuscitation (CPR) in children differs from the approach in adults because of the differences in cause and pathophysiology of the cardiac arrest. Adults usually present with ventricular fibrillation during cardiac arrest and often have a history of coronary artery disease (CAD).² The focus during the resuscitation of an adult is prompt defibrillation to improve survival. Children who experience cardiac arrest rarely have CAD. Worsening tissue hypoxia, acidosis due to respiratory failure, or circulatory shock usually causes cardiac arrest in children.² In the pediatric population, ECG rhythms usually progress from bradycardia to asystole or pulseless electrical activity (PEA) rather than ventricular fibrillation. The survival rate of children presenting with these cardiac arrhythmias is higher than in adults presenting with the same arrhythmias.² It is essential that the anesthesia provider recognize cardiac arrest early in the pediatric population and provide immediate treatment in order to increase the survival of the patient.

The American Heart Association (AHA) has developed guidelines for the resuscitation of the pediatric patient who experiences cardiac arrest. The ultimate goal of the resuscitation guidelines is to improve the survival of the patient, with good neurologic outcomes. According to the AHA, hospitalized children are 3 times more

likely to survive a cardiac arrest now than they were 10 years ago.³ In a multicenter prospective observational study from hospitals in the United States and Canada, Nadkarni et al² found that the rate of survival to hospital discharge following pulseless cardiac arrest is higher in children than in adults: 27% vs 18%, respectively. Most of the children who experience cardiac arrest during a hospitalization typically have respiratory illnesses, severe infections, or a history of heart surgery.³ The improvement in survival is primarily due to the clinical practice guidelines for acute resuscitation.

The pediatric resuscitation guidelines are built on the foundation that the healthcare provider will provide basic life support to maintain the oxygen supply to the vital organs and tissues. The most vital step during a cardiac arrest is to recognize the rhythm. Once the rhythm has been identified as a shockable or nonshockable rhythm on the ECG, the provider can deliver the treatment. In children, rhythm disturbances are usually caused by nonshockable rhythms such as asystole or PEA.⁴ Pulseless electrical activity exists when there is organized electrical activity on the ECG without a palpable pulse. The goal of resuscitation is to achieve the return of spontaneous circulation while investigating and reversing the cause of the cardiac arrest.

The treatment of PEA is to provide basic life support and pediatric advanced life support. When the child has a diagnosis of a potentially lethal arrhythmia, CPR should be initiated (Figure). Once the arrhythmia has been diagnosed on ECG as a nonshockable rhythm such as PEA, CPR should be continued, and epinephrine (0.01 mg/kg) should be administered intravenously.⁵ The epinephrine dose can be repeated every 3 to 5 minutes. Once an airway has been secured, the patient should be ventilated

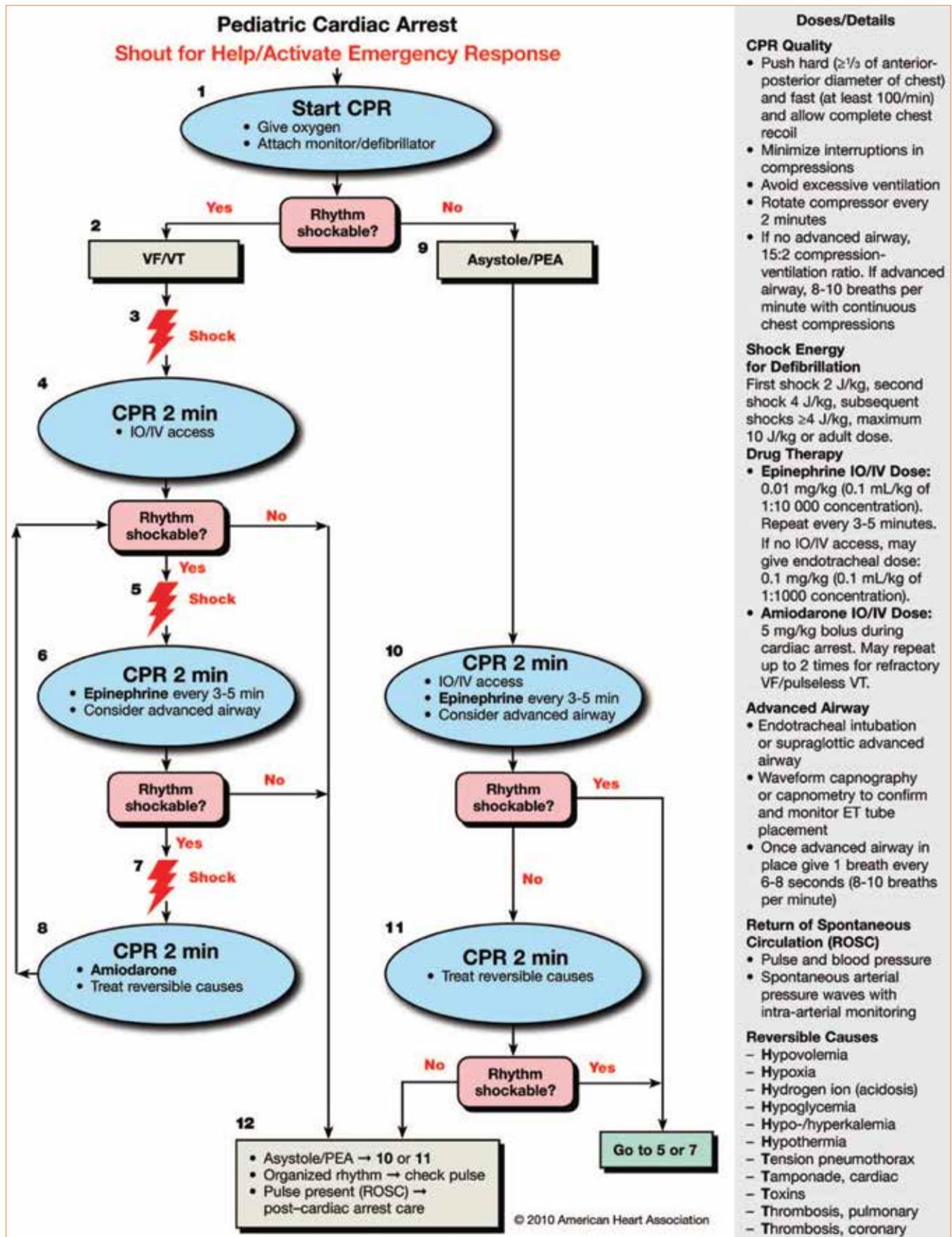


Figure. American Heart Association's Pediatric Advanced Life Support Pulseless Arrest Algorithm

Abbreviations: CPR, cardiopulmonary resuscitation; ET, endotracheal; IO/IV, intraosseous-intravenous; min, minutes; PEA, pulseless electrical activity; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia.

(From Kleinman et al.⁵ Used with permission.)

with 1 breath every 6 to 8 seconds.⁵ The provider should continue to assess the rhythm without minimal interruption in chest compressions. If the rhythm is not shockable, CPR should be continued until return of spontaneous circulation occurs.

In 2010 the AHA celebrated the 50th anniversary of the introduction of CPR to improve the survival of patients experiencing cardiac arrest. Despite the improved outcomes of in-hospital CPR, many children do not survive or they remain incapacitated after cardiac arrest.⁵ To improve survival, the AHA has put in place initiatives to promote the clinical practice guidelines in an effort to improve early recognition and management of cardiac arrest such as PEA.³ A step-by-step approach should be incorporated into practice when encountering a patient with PEA to seek out the reversible causes and improve survival.

There are a limited number of case reports in children who experience PEA. Most of the cases in the literature discuss PEA in adults who have experienced PEA because of severe retroperitoneal hemorrhage from rupture of the abdominal aortic aneurysm, lung hyperinflation, and hyperkalemia.⁶⁻⁹ One case report did describe the development of PEA in a pediatric patient. In 2008, Hyde and Puddy¹⁰ described the development of PEA in 2 extremely low-birth-weight, preterm neonates. In their case, PEA developed because of acute severe ionized hypocalcemia secondary to binding of calcium by citrate in the fresh frozen plasma.

The case report presented here offers a detailed description of the successful resuscitation of a pediatric patient in whom PEA developed during a bronchoscopy. A thorough review of the causative factors and treatment modalities of PEA will be discussed.

Case Summary

An 18-month-old, 12-kg, girl presented to the preoperative area for a rigid bronchoscopy. The patient's medical history included reactive airway disease. The patient had no allergies to medication. Her current medications included fluticasone, albuterol, and methylprednisone. There was no prior surgical history or family history of anesthetic complications. Two days before admission to the hospital, the patient started having difficulty breathing and persistent coughing.

The preoperative physical assessment revealed audible stridor, chest retractions, bilateral inspiratory wheezing, regular heart rate and rhythm, a noninvasive blood pressure (BP) of 80/40 mm Hg, heart rate (HR) of 101/min, respiratory rate of 35/min, and arterial oxygen saturation (SaO₂) of 97% at room air. The patient's preoperative hemoglobin and hematocrit values were 11.2 and 34%, respectively.

The patient's guardian consented to a general anesthetic. No preoperative sedative was given to the patient. Nebulized albuterol was administered to the patient

in the preoperative holding area, with improvement in inspiratory wheezing. Lactated Ringer's solution was infused intravenously and continuously through a 24-gauge peripheral intravenous catheter at 44 mL/h.

In the operating room, standard monitors were applied. Preoxygenation with 100% oxygen was instituted, and the intravenous induction agents lidocaine (12 mg) and propofol (36 mg) were given. An oral airway was inserted, and mask ventilation was maintained with 100% oxygen. A propofol infusion was started at a dosage of 75 µg/kg/min. The patient received 10% topical nebulized lidocaine puffs to the oropharynx. The patient was positioned supine with a rolled towel across the back between the scapulae to extend the neck and bring the trachea forward. The patient's oxygenation was maintained with intermittent positive pressure ventilation through a T-piece connected to the side arm of a 20-cm, size 3.5 rigid bronchoscope. Chest wall movement was observed during the procedure and end-tidal carbon dioxide (ETCO₂) was monitored. The ETCO₂ was maintained within 35 to 40 mm Hg, with the tracing returning to baseline at end-expiration. Oxygen was administered by face mask if SaO₂ decreased to less than 90%.

Twenty minutes into the procedure, there was a sudden loss of ETCO₂ and SaO₂. Chest wall movement was decreased while ventilating through the rigid bronchoscope. The ECG tracing on the monitor displayed sinus rhythm in the 90s. The BP monitor did not register a numerical reading, and no carotid pulse was appreciated. The propofol infusion was discontinued immediately, and the procedure was terminated.

Chest compressions were started, and a 4.0-mm endotracheal tube was placed in the trachea by the surgeon. Bilateral breath sounds were auscultated after the airway was secured by the surgeon. A bolus of 250 mL of lactated Ringer's solution and 0.12 mg of epinephrine was administered intravenously. Chest compressions and manual ventilation were continued until an ETCO₂ waveform was visible on the monitor and a pulse was palpated in the carotid artery. The HR, BP, SaO₂, and ETCO₂ were monitored and kept within normal limits until the patient awoke from anesthesia. The endotracheal tube was removed after the patient was awake, responsive, and kicking up both legs. The vital signs after resuscitation were HR of 130/min, BP of 93/50 mm Hg, and SaO₂ of 99%.

A day after the resuscitation, the patient was awake and responsive. Two days after the resuscitation, the patient returned to the operating room for successful completion of the procedure. The patient was discharged home 4 days after the resuscitation, with no sequelae.

Discussion

Pulseless electrical activity (PEA), previously known as electromechanical dissociation, is a clinical condition that presents as a lack of a palpable pulse in the pres-

ence of organized electrical activity. Metabolic, respiratory, or mechanical derangements that lead to changes in preload, afterload, or contractility can lead to PEA.¹¹ The weakened myocardium will be compromised by worsening acidosis, hypoxia, and increased vagal tone. The compromised inotropic state of the myocardium will lead to inadequate mechanical activity, even though the electrical activity of the heart is functional.

During PEA, the cardiac muscle is unable to generate enough force during electrical depolarization. It is always caused by a direct insult to the myocardium. Many of the causes are treatable if diagnosed early. The causes of PEA include hypovolemia, hypoxemia, hydrogen ions, hypokalemia or hyperkalemia, hypoglycemia, hypothermia, toxins, cardiac tamponade, tension pneumothorax, thrombosis, and trauma.¹¹ The anesthesia provider must attempt to diagnose the cause in order provide the appropriate treatment.

One such treatable cause is massive external or internal bleeding.⁶ The massive bleeding may be caused by gastrointestinal bleeding, trauma to the structures in the thorax, or a severe pelvic fracture. Pulseless electrical activity caused by internal bleeding is difficult to diagnose unless a thorough assessment is performed with diagnostic studies. Ultrasonography findings may reveal an empty right ventricle during severe hypovolemia or a shock state.⁶ The empty ventricle should suggest a search for the cause of the bleeding to include the aorta, pleural spaces, and peritoneal spaces.

Any patient condition contributing to an obstruction to systemic or pulmonary circulation can also lead to PEA. Major causes of obstruction to circulation may include cardiac tamponade, tension pneumothorax (TP), and pulmonary embolus (PE).¹² Cardiac tamponade will impede the circulation by blocking the filling of the ventricles during diastole. The major causes of cardiac tamponade include myocardial infarction, inflammation of the pericardium, and trauma to the ventricle.¹³ Tension pneumothorax will impede the venous return to the heart. Even though TP is a rare cause of PEA, it should be considered in the trauma patient and the patient receiving mechanical ventilation. The data supporting PE as a cause of PEA are scarce. Even though it is very difficult to diagnose, a study by Comess et al¹⁴ revealed that 9 of 25 patients who presented with PEA were diagnosed with having a PE. The PE was diagnosed by transesophageal echocardiography that revealed right ventricular enlargement and dysfunction.¹⁴

A rare but lethal cause of circulatory obstruction is air trapping during mechanical ventilation.⁸ Lung hyperinflation is a unique phenomenon in which the end-expiratory volume may exceed the predicted end-expiratory volume because the rate of lung emptying is interrupted by the next inspiratory effort before exhalation to the static relaxation volume.¹² A recent case

report implicated lung hyperinflation as the cause of PEA in a mechanically ventilated patient. Lung hyperinflation occurs when enough time is not allowed for complete exhalation; therefore, air is trapped in the lungs, developing excessive airway pressures. Lung hyperinflation could potentially lead to compression of the heart and right ventricular afterload. In addition, if mediastinal pressures are increased and venous return is decreased, the result is loss of cardiac output.⁸ Lung hyperinflation can be caused by bronchospasm, mucus plug, a partially obstructed endotracheal tube, and aspirated secretions.

Hypoxia and acidosis that leads to respiratory failure is considered another cause of PEA. Respiratory failure is not the initiator of the cardiac arrest, but the outcome of hypoxia and acidosis leads to negative inotropic effects on the heart.¹² Advanced cardiac life support will improve oxygenation and acidosis in the patient with respiratory failure. In pediatric cardiac arrest, hypoxia and acidosis are major factors that contribute to acute respiratory insufficiency and circulatory shock.¹

Additionally, some toxins such as an overdose of therapeutic drugs can lead to PEA by causing massive vasodilation that will result in a lower BP and decreased cardiac output. Overdoses of verapamil, atenolol, digoxin, and propranolol have been implicated in several case studies resulting in PEA.¹⁵⁻¹⁸

Other unusual causes of PEA can include electrolyte disturbances, hypoglycemia, and hypothermia. Electrolyte disturbances, such as hypokalemia and hyperkalemia, have been shown to contribute to the development of PEA when it is associated with changes in the ECG such as peaked T waves, widened QRS complex, or sinusoidal QRS pattern.¹² Hyperkalemia is commonly seen in patients with chronic renal failure. When the patient presents with PEA resulting from hyperkalemia, calcium chloride and glucose infusion along with cardiopulmonary resuscitation, will assist in the return of spontaneous circulation.⁹ Hypoglycemia, a condition defined as inadequate levels of glucose in the blood, has been implicated as a cause of PEA even though evidence to support this is sparse.¹⁹ Hypothermia is not considered a cause of in-hospital cardiac arrest but may be considered in patients who live in high altitudes or are exposed to extremely cold climates.²⁰

Early recognition of cardiac arrest and prompt initiation of CPR is essential for successful resuscitation of the pediatric patient experiencing PEA. It may be difficult to recognize a patient who is displaying the signs and symptoms of PEA. A problem should be suspected when there is a sudden loss of ETCO₂ and noninvasive BP. When the pulseless patient is identified, CPR should be initiated immediately. It is essential to promptly recognize the cause of PEA in order to resume immediate return of a spontaneous circulation.

An approach to diagnosing the cause of PEA was re-

viewed by Desbiens.¹² An algorithm using the 3 and 3 rule will allow the practitioner to recognize the cause of PEA and immediately treat it. After chest compressions are started, if a pulse is palpated in the femoral artery, cardiac failure may be the cause of PEA. The treatable causes of cardiac failure include hypoxemia, acidosis, hypokalemia, hyperkalemia, hypoglycemia, hypothermia, and toxins. If no pulse is palpated in the femoral artery during chest compressions, an obstruction to circulation or severe volume depletion should be suspected. Differential diagnoses in obstruction to circulation may include TP, cardiac tamponade, and PE. Deviation of the trachea may assist in the diagnosis of TP. An echocardiogram would aid in the diagnosis of cardiac tamponade and PE. Severe volume depletion may be suspected in the trauma patient with suspected external or internal injuries. Many of the causes of PEA are treatable if diagnosed early. A systematic approach to diagnosis and treatment will improve survival in the patient.

In PEA, the treatment of choice is to determine the cause and to administer epinephrine in conjunction with CPR. A standard dose of epinephrine (0.01-0.02 mg/kg) was used in this case study, but one study implicated that high-dose epinephrine (0.05-0.2 mg/kg) had dramatic improvement during resuscitation and outcome of the patient.²¹ Most studies in the literature refute this claim and have found no benefit in administering high-dose epinephrine instead of a standard dose during pediatric cardiac arrest. A study by Carpenter and Stenmark²² concluded there is no benefit from the use of high-dose epinephrine compared with a standard dose. Fourteen of 24 resuscitations using high-dose epinephrine resulted in the return of spontaneous circulation (58%), with a mean time of 19 minutes; in comparison, 24 of the 34 resuscitations using standard-dose epinephrine resulted in return of spontaneous circulation (71%) in 12 minutes.²² Another study, by Dieckmann and Vardis,²³ concluded that high-dose epinephrine does not seem to improve survival in the pediatric patient with an out-of-hospital cardiac arrest due to asystole and PEA compared with standard-dose epinephrine.

Vasopressin has been compared with epinephrine. This long-acting endogenous hormone acts at V₁ receptors to cause systemic vasoconstriction and V₂ receptors to increase the reabsorption of water in the renal tubule. In swine models undergoing cardiac arrest, vasopressin shunted more blood to the vital organs, brain, and heart, compared with epinephrine administration.²⁴ Results of studies in humans have concluded that vasopressin had no obvious benefit over epinephrine during CPR in pediatric patients.^{25,26}

This case study presented the unusual development of PEA in a child undergoing anesthesia for rigid bronchoscopy. When a patient undergoes a rigid bronchoscopy, it is important to maintain adequate oxygenation and

communication with the surgeon since the airway is being shared. During a bronchoscopy, a rigid or flexible scope is inserted into the trachea for diagnostic or therapeutic purposes. If adequate assisted ventilation is not provided during the case by the anesthesia provider, the inadequate ventilation can lead to hypercarbia and hypoxemia. Hypercarbia can occur during a bronchoscopy because the expiratory pressure generated by the passive recoil of the chest and lung may not be sufficient to expel air through the smaller scopes.²⁷ Air trapping can also occur in the lungs during a bronchoscopy if enough time is not allowed for passive expiration.²⁷ The air trapping can lead to barotrauma, diminished venous return, and reduced cardiac output.

The patient in this case study had no history of complications under general anesthesia. The preoperative anesthetic evaluation revealed active respiratory problems that could have contributed to the development of PEA in this case. The combination of the respiratory problems, hypoxia, and air trapping during the bronchoscopy were presumed to be the cause of the cardiac arrest that led to PEA. During manual ventilation, if enough time is not allowed for complete exhalation, air could get trapped in the lungs. This trapped air leads to lung hyperinflation that could potentially lead to a decrease in venous return and a loss in cardiac output. To prevent this event from occurring during a bronchoscopy, the provider should carefully monitor the patient's ventilations by monitoring the ETCO₂ and SpO₂. It is imperative to allow a long time constant of 5 to 10 seconds for expiration during manual ventilation to prevent the scenario that occurred in this case.²⁷

Pulseless electrical activity is a rare medical emergency. The cause of PEA in a child undergoing anesthesia is generally assumed to be caused by respiratory failure, but other causes should also be investigated. The anesthesia provider should approach the cardiac arrest with an algorithmic approach to adequately diagnose the cause of PEA and administer the appropriate treatment. The goal of treatment during a cardiac arrest should always be to increase the child's chance of survival.

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