Intrinsic PEEP: An Underrecognized Cause of Pulseless Electrical Activity

Sandeep Mehrishi, MD, FCCP, DABSM
Liziamma George, MD, FACP, FCCP
Aamir Awan, MD

Cardiopulmonary arrest associated with pulseless electrical activity (PEA) carries a poor prognosis unless a reversible cause is recognized and is treated promptly. Rapid initiation of advanced cardiac life support (ACLS) has become a routine practice in cardiopulmonary arrest and is performed according to American Heart Association guidelines.[1] PEA, a rhythm frequently encountered during cardiac arrest, is characterized by organized electrical activity without palpable pulse or measurable arterial blood pressure. Although survival rate for primary PEA is only 0% to 5%, the cardiac dysfunction can be reversed when secondary causes are treated promptly.[2,3] The common causes of PEA and their management are given in **Table 1**. Stueven et al[4] found that only 19% of 503 adult patients who sustained cardiorespiratory arrest on presentation to a prehospital system could be successfully resuscitated (as defined by presence of pulse and a rhythm on arrival into emergency department [ED]). A minority of these patients (4.4%) were discharged alive from the hospital.

Positive-pressure ventilation (PPV), whether manual or mechanical, is a standard component of cardiopulmonary resuscitation (CPR). Currently, PPV is performed during CPR without any regard to patients' previous medical and especially pulmonary history. Patients with underlying pulmonary disease usually have abnormal airway resistance and compliance, which are the two most important physiologic variables related to development of intrinsic positive end-expiratory pressure (PEEP), also called auto-PEEP. Woda et al[5] suggested that rescuers inadvertently may hinder the resuscitation effort because there is no adequate method of ventilation to account for development of intrinsic PEEP during CPR. We report a case in which a patient may have developed intrinsic PEEP during the course of CPR, thus limiting resuscitative efforts.

CASE PRESENTATION

A 68-year-old woman with history of chronic obstructive pulmonary disease (COPD) was referred to the ED by her primary care physician for shortness of breath. On arrival in the ED, the patient was in severe respiratory distress and was unable to complete her sentences without stopping intermittently. Her past medical history included a 90 pack-year history of cigarette smoking, hypertension, bursitis, and anxiety. Her medications included celecoxib, oxycodone hydrochloride, doxazosin mesylate, and a combination of propoxyphene napsylate and acetaminophen.

Physical examination revealed that the patient's blood pressure was 160/92 mm Hg, her heart rate was 106 bpm, and her respiratory rate was 26 breaths/min. Physical examination also was significant for diffuse wheezes. Due to her severe respiratory distress, she was immediately intubated with a 7.5-mm endotracheal tube and placed on mechanical ventilation with assist control mode, tidal volume of 500 mL, respiratory rate of 14 breaths/min, and inspired oxygen concentration of 0.8. The arterial blood gas (ABG) analysis following initiation of mechanical ventilation revealed a pH of 7.04, $Paco_2$ of 109 mm Hg, and Pao_2 of 63 mm Hg. She was started on treatment with albuterol nebulizers, intravenous magnesium sulphate, and intravenous methyl prednisolone. Based on the ABG results, the ventilator settings were adjusted and tidal volume was increased to 600 mL and respiratory rate to 20 breaths/min. A repeat ABG obtained after this change in ventilator settings revealed pH of 7.28, $Paco_2$ of 52 mm Hg, and Pao_2 of 125 mm Hg. A chest radiograph revealed significant hyperinflation.

Shortly thereafter, she suffered a cardiac arrest and an ACLS protocol was initiated. During CPR, it was evident that there was increasing difficulty to ventilate the

Dr. Mehrishi and Dr. George are assistant clinical professors of medicine at the State University of New York, Stony Brook, NY. Dr. Awan is in private practice in the Department of Medicine, Jackson Medical Center, Jackson, MN.

patient and her chest became progressively distended. The patient was switched from mechanical ventilation to manual ventilation. At this time, the cardiac monitor revealed PEA. The recommended ACLS algorithms for PEA as outlined by the American Heart Association[1] were followed. Bilateral chest tubes were placed emergently during the CPR, and a pericardiocentesis attempt was made without any beneficial result. Empiric aggressive intravenous fluid administration was performed for unsuspected hypovolemia. The ACLS protocol was continued for a period of 20 minutes after which it was discontinued.

Five minutes after cessation of resuscitative efforts and discontinuation of PPV, the patient was noted to have spontaneous respiration, sinus tachycardia, and a palpable systolic blood pressure of 80 mm Hg. Massive pulmonary embolism as a cause of PEA was thought to be unlikely on the basis of improvement in blood pressure upon discontinuation of PPV. She was transferred to the medical intensive care unit where she died 2 weeks later with severe anoxic encephalopathy.

DISCUSSION

Pathology of Intrinsic PEEP

CPR, a procedure that dates back to the mid-1800s, came into common practice in hospitals after the first description of closed cardiac massage in 1960.[6] Since then, the goal of CPR has been to provide adequate ventilation at the time of closed cardiac massage. Little has been written to describe the necessity for altering the method of providing ventilation in patients with underlying pulmonary disorders such as asthma, COPD, or chronic bronchitis. A significant number of patients (~5%) who undergo CPR may have underlying pulmonary disease.[5,7]

Mechanical ventilation with ventilator settings that do not permit complete exhalation (eg, increased respiratory frequency, high tidal volume, decreased expiratory time, or a combination of these factors) results in air trapping or hyperinflation of the lungs.[8] In addition, dynamic airway collapse also can contribute to this process. Consequently, the alveolar pressure at the end expiration is higher than atmospheric pressure. This process is called *dynamic hyperinflation*, and the positive end-expiratory alveolar pressure associated with a higher than resting lung volume is called *intrinsic* or *auto-PEEP*.[9] Woda et al[5] found that the ventilatory variables can be increased to a point at which intrinsic PEEP could develop even in patients with normal lungs. They also demonstrated that utilizing current ACLS standards of ventilation, significant intrinsic PEEP could develop in patients with lung disease.

Table 1. Common Causes and Treatment of Pulseless Electrical Activity

Causes	Management
Hypovolemia	Volume infusion
Tension pneumothorax	Decompression (chest tube, needle)
Acidosis	Hyperventilation, sodium bicarbonate
Cardiac tamponade	Pericardiocentesis
Hypothermia	Rewarming
Electrolyte disturbances	
Hyperkalemia	Sodium bicarbonate, glucose/insulin, calcium chloride, dialysis
Hypokalemia	Replace IV potassium, may add magnesium
Hypoxia	Oxygenation, ventilation
Myocardial infarction/injury	Nitrates, aspirin, thrombolytics, etc
Embolism (pulmonary, air, amniotic fluid)	Thrombolytics, heparin, surgical intervention, supportive therapy
Drug overdose	Mechanical ventilation, activated charcoal, lavage, specific antidotes

IV = intravenous.

Intrinsic PEEP adversely affects lung compliance and cardiovascular function, increases the work of breathing, and can worsen hypoxemia and hypercapnia. The hemodynamic consequences of positive airway pressure were first recognized by Cournanad et al[10] in 1948. An important consequence of dynamic hyperinflation is that it can cause "occult" intrinsic PEEP, which can lead to hypotension by increasing pulmonary vascular resistance[11] and intrathoracic pressure.[12] This effect is particularly evident in hypovolemic patients and in patients with increased lung compliance.[13,14] A case of a patient with emphysema who developed severe cardiovascular instability secondary to ventilator changes and who had simultaneous chest radiographic abnormalities suggestive of intrinsic PEEP has been reported.[15]

The left ventricular performance also is affected by intrinsic PEEP. Jardin et al[16] demonstrated that for each stepwise increase in PEEP at or above 15-cm H_2O, mean arterial pressure, cardiac index, and stroke index decrease, intrapleural pressures increase, and transmural pressures remain unchanged. They demonstrated that with increasing level of PEEP, there is a

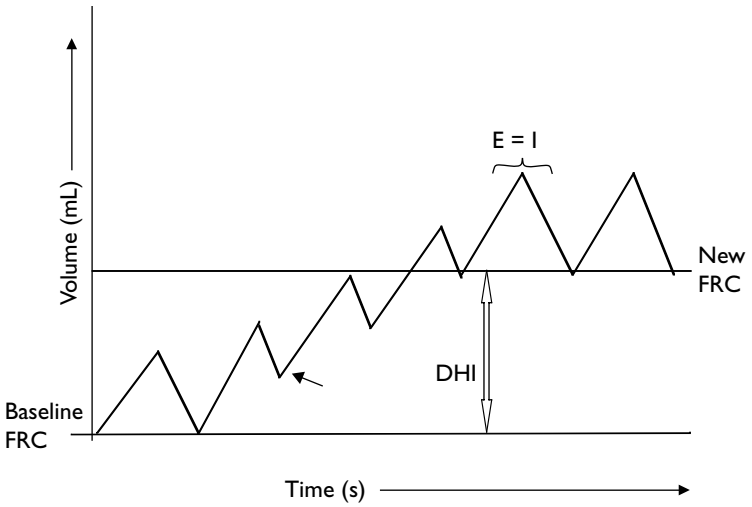

Figure. Flow time curve showing development of dynamic hyperinflation (DHI). The plotted line represents respirations, with the ascending lines denoting inhalations (I) and the descending lines denoting exhalations (E). The third breath is initiated (*arrow*) before complete exhalation of the second breath. Subsequent breaths are initiated on a gradually increasing functional residual capacity (FRC) until a new equilibrium is reached when exhalation equals inspiration. Significant DHI (*double-headed arrow*) has developed before a new FRC is attained. At end-exhalation, when the expiratory port is occluded, the manometer reads a positive pressure, indicating the presence of intrinsic positive end-expiratory pressure.

gradual increase in the radius of septal curvature at both end-diastole and end-systole; a progressive decrease in end-diastolic and end-systolic left ventricular dimensions; equalization of right and left ventricular transmural end-diastolic pressures; and unaltered or slightly increased myocardial contractility. When the lungs are hyperinflated, right ventricular loading restrains left ventricular performance through a leftward shift of the interventricular septum, which may be further enhanced with volume loading.[16] Reversal of cardiac arrhythmia with reduction in dynamic hyperinflation also has been reported.[17]

The case patient had severe COPD and was being ventilated with high tidal volumes and a high respiratory rate. Aggressive PPV, performed in an attempt to reduce the hypercapnia and respiratory acidosis, may have caused intrinsic PEEP. Development of intrinsic PEEP also has been reported with manual ventilation.[18,19] With termination of resuscitative efforts and PPV, the lungs have sufficient time to passively deflate, thus producing a fall in intrathoracic pressure that leads to an increase in venous return and results in palpable blood pressure.[12] Rogers et al[20] reported a patient with severe COPD who developed PEA while receiving mechanical ventilation during CPR. Fifteen minutes after stopping the CPR, patient showed spontaneous respiration, a palpable systolic blood pressure of 60 mm Hg, and sinus tachycardia. Lapinsky et al[21] suspected the role of intrinsic PEEP in the condition of 38% of patients with obstructive lung disease who underwent CPR secondary to electromechanical dissociation. In a multivariate analysis, Sutton et al[22] found the presence of pulmonary disease to be an independent predictor of electromechanical dissociation.

Diagnosis of Intrinsic PEEP

There are various methods to measure intrinsic PEEP.[23] Intrinsic PEEP should be suspected during expiration when the chest fails to come to rest before the onset of subsequent breath or wheezes persist until inspiration.[24] These findings can be confirmed reliably on graphic waveform displays on ventilators. On a flow time curve, an expiratory flow rate that does not return to zero before the next machine-delivered breath suggest the presence of intrinsic PEEP (**Figure**). Another method of determining intrinsic PEEP is by reading the ventilator manometer pressure when the expiratory port is occluded during a passive exhalation just before the onset of inspiratory flow. A positive pressure value indicates presence of intrinsic PEEP.

Management of PEA

A primary goal of resuscitative therapy in patients with PEA, in addition to excluding other recognized causes, should be to improve venous return with aggressive fluid administration. When fluid administration proves ineffective, intrinsic PEEP should be included in the differential diagnosis of potentially reversible etiologic factors. Although intrinsic PEEP is more likely to develop in the setting of severe airflow obstruction, it also can occur whenever minute ventilation is high.[25] It has been proposed that a brief trial of apnea (15–30 s) should be given in differentiating absent pulse due to intrinsic PEEP from other causes.[20]

Management of Intrinsic PEEP

Apart from disconnecting the patient from mechanical ventilation, other steps can be taken to manage intrinsic PEEP.[23] These steps include:

- **Treating airway constriction and inflammation with medications (eg, bronchodilators, corticosteroids).** This increases the expiratory flow rate, thereby minimizing air trapping.
- **Decreasing minute ventilation.** This can be achieved by decreasing either the tidal volume or the respiratory rate. This maneuver may lead to increased $PaCO_2$, termed permissive hypercapnia, which may not be harmful if contraindications (eg, increased intracranial pressure, cerebral edema) are excluded and a rapid and very high increase in $PaCO_2$ (> 90 mm Hg) is avoided.[26] However, permissive hypercapnia may have deleterious effects that must be considered in relation to those of intrinsic PEEP.
- **Increasing the time available for exhalation.** This can be achieved either by increasing the inspiratory flow rate or by decreasing the respiratory rate. The decrease in respiratory rate may lead to hypercapnia, and the patient may have to be heavily sedated and paralyzed.
- **Applying a small amount of external PEEP.** Development of intrinsic PEEP is one of the most common causes of "fighting the ventilator." Patients feel tremendous sensation of dyspnea as they lower their pleural pressure in an attempt to inhale but find that no air enters as alveolar pressure has not dropped below atmospheric pressure. Hence, the ventilator is not triggered. By applying extrinsic PEEP, the atmospheric pressure at end-exhalation is raised, thereby decreasing the work required to drop alveolar pressure below atmospheric pressure. In general, extrinsic PEEP of 80% to 85% of the measured intrinsic PEEP is applied. Various strategies that can be used to manage intrinsic PEEP are summarized in **Table 2**.

Table 2. Strategies to Prevent or Minimize Intrinsic PEEP in Patients on Positive-Pressure Ventilation

Decrease minute ventilation
Decrease respiratory rate
Decrease tidal volume
Decrease airway resistance
Bronchodilators
Anti-inflammatory medications (corticosteroids)
Large-size endotracheal tube, ventilator circuits, and devices
Increase of expiratory time
Increase peak inspiratory flow rate
Decrease respiratory rate
Sedate with or without paralyzation
Resort to permissive hypercapnia
Application of external PEEP (80%–85% of intrinsic PEEP)

PEEP = positive end-expiratory pressure.

Recognizing that patients with COPD may develop hyperinflation-induced hypotension after intubation and initiation of PPV, and being prepared for its occurrence, appears to be essential in the effective care of these patients. The present case suggests that it is a worthwhile effort to routinely disconnect the patient from PPV for a brief time if PEA develops during CPR, especially if the patient has a history of obstructive pulmonary disease.

CONCLUSION

Mechanical ventilation with ventilator settings that do not allow complete exhalation can result in intrinsic PEEP. This may occur, not only in patients with obstructive lung disease, but also in patients who are otherwise healthy. When PEA develops in patients receiving PPV, intrinsic PEEP should be included in the differential diagnosis. Apart from adjusting the ventilator settings to minimize the likelihood of inducing intrinsic PEEP, consideration should be given to disconnecting the patient from the PPV for a brief time. **HP**

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(continued on page 36)

(from page 33)

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