Auto-PEEP during CPR*
An “Occult” Cause of Electromechanical Dissociation?

Paul L. Rogers, M.D.;† Robert Schlachtag, M.D.;† Adelaida Miro, M.D.,‡ and Michael Pinsky, M.D., F.C.C.P.‡

A 64-year-old man with severe COPD developed refractory nonperfusing sinus rhythm after intubation and positive-pressure ventilation. Fifteen minutes after resuscitative efforts were halted, the patient was noted to have spontaneous respirations and blood pressure, suggesting that dynamic hyperinflation was responsible for the observed electromechanical dissociation (EMD). We recommend a brief trial of apnea for patients with COPD and EMD when conventional measures are unsuccessful.

(Chest 1991; 99:492-93)

BCLS = basic cardiac life support; ACLS = advanced cardiac life support; EMD = electromechanical dissociation

Approximately 1,000 cardiopulmonary arrests occur each day in the United States.† Rapid initiation of basic cardiac life support (BCLS) and advanced cardiac life support (ACLS), which includes defibrillation and pharmacologic resuscitation, have decreased mortality associated with cardiac arrest.‡ Electromechanical dissociation (EMD), a rhythm frequently encountered during cardiac arrest, is characterized by organized electrical activity without palpable pulse or measurable arterial blood pressure. Although survival rates for primary EMD are only 0 to 5 percent, the cardiac dysfunction can be reversed when secondary causes of EMD such as hypovolemia, pulmonary embolism, tension pneumothorax, and pericardial tamponade are recognized and treated promptly.§ We report a potentially reversible cause of EMD during cardiopulmonary resuscitation that, to our knowledge, has not previously been described.

CASE REPORT

A 64-year-old man was admitted to the medical intensive care unit (MICU) with an acute exacerbation of chronic obstructive pulmonary disease (COPD) complicated by hypercapnia and respiratory acidosis. His medical history included 120 pack-years of cigarette smoking. Results of pulmonary function tests obtained before hospital admission were consistent with severe obstructive lung disease (FEV1 = 34 percent predicted). He responded rapidly to albuterol inhalation therapy and intravenous aminophylline, methylprednisolone, and antibiotics. Within 24 hours, his arterial blood gas values returned to baseline (pH of 7.44, Pco2 of 45 mm Hg, and Po2 of 55 mm Hg). He was transferred to a general medical ward for continued therapy.

Two days after the patient’s transfer from the MICU, acute bronchospasm and severe respiratory distress returned. Endotracheal intubation and manual ventilation were performed to improve the initial pH of 7.19, the Pco2 of 60 mm Hg, and the Po2 of 60 mm Hg; the endotracheal tube position was confirmed by auscultation. Shortly after intubation, the patient became hemodynamically unstable and required intravenous fluids and dopamine to maintain a systolic blood pressure of 60 mm Hg. Despite the administration of vasopressors and 3 L of normal saline solution, he became pulseless with sinus rhythm consistent with EMD. Repeated arterial blood gas analysis revealed pH of 7.31, Pco2 of 41 mm Hg, and Po2 of 39 mm Hg. The recommended ACLS algorithms for EMD, as outlined by the American Heart Association,¶ were followed. Possible reversible etiologic factors, including pulmonary embolism, tension pneumothorax, cardiac tamponade, and hypovolemia, were considered and were clinically excluded. During the resuscitation the patient never developed a palpable blood pressure, and after 20 minutes all efforts were discontinued. Fifteen minutes after resuscitative efforts were ended, the patient was noted by the nursing staff to have spontaneous respirations, sinus tachycardia, and a systolic blood pressure of 60 mm Hg. He was transferred to the MICU but died with profound hypotension in an hour despite aggressive therapy.

The final autopsy report revealed severe bilateral bullous emphysema and aspiration pneumonia of the right upper lobe. The endotracheal tube was positioned above the carina. There was no gross evidence of pericardial effusion, pulmonary emboli, or pneumothorax. Neither the gross nor the microscopic examination revealed any evidence of myocardial infarction.

DISCUSSION

Adverse hemodynamic consequences of positive airway pressure were first recognized by Courmand et al5 in 1948. Since then, others have reported similar hemodynamic effects, postulating that the reduction in cardiac output is due primarily to an increase in intrapleural pressure that impedes venous return.§ A decrease in venous return and a subsequent decrease in cardiac output are particularly evident in patients who are hypovolemic or who have an increased lung compliance, such as seen with emphysema.¶ Depression of cardiac output has been reported by Pepe and Marini6 in patients with COPD during mechanical ventilation. When selected ventilator settings do not permit adequate time for complete exhalation, dynamic factors such as increased respiratory frequency, airway collapse, tidal volume, percentage of inspiratory time (which reduces absolute expiratory time), or a combination of these factors results in air trapping or hyperinflation of the lungs.§ An important consequence of dynamic hyperinflation is that alveolar pressure remains positive throughout expiration leading to the development of “occult” positive end-expiratory pressure also termed “auto-PEEP” or “intrinsic” PEEP. The resulting hemodynamic consequences are identical to those caused by extrinsically applied PEEP, which can produce hypotension by increasing pulmonary vascular resistance7,8 and increasing intrathoracic pressure.¶,9

Our patient had both bullous emphysema and an episode of acute bronchospasm. We postulate that once manual ventilation was initiated, alveolar pressures were easily transmitted to the pleural space, causing an increase in intrathoracic pressure and ultimately reducing venous return. Auto-PEEP may have developed as a result of aggressive manual ventilation in an attempt to reduce hypercapnia and correct respiratory acidosis. Finally, the hyperinflation-induced increases in pulmonary vascular resistance may impede right ventricular ejection, leading to a decreased left ventricular preload.10,11 When resuscitative efforts were discontinued, the lungs had sufficient time to passively deflate, intrathoracic pressure fell, and venous return increased. With adequate left ventricular preload, the cardiac...
output increased sufficiently to produce a palpable blood pressure. A "mechanism" was proposed by Pepe and Marini9 when moderate to severe hypotension developed as changes in ventilator settings resulted in intrinsic PEEP: Once positive pressure ventilation was temporarily discontinued, the patient's hemodynamic indices improved and stabilized.10

In a recent retrospective review of 261 cases of cardiac arrest,11 the presence of COPD was associated with an increased risk of EMD. Although not discussed by the authors, it may be auto-PEEP, not simply COPD, that is the important factor associated with the development of EMD.

A primary goal of resuscitative therapy in patients with EMD should be to improve venous return with aggressive fluid administration. When fluid administration proves ineffective in patients with severe bronchospasm, it may be worthwhile to include auto-PEEP in the differential diagnosis of potentially reversible etiologic factors. A brief trial of apnea (15 to 30 s) may be beneficial in differentiating absent pulse due to auto-PEEP from other causes. Recognizing that patients with COPD may develop hyperinflation-induced hypotension after intubation and mechanical ventilation and being prepared for its occurrence appear to be essential in the effective care of these patients.

REFERENCES
3 Friedman HS. Diagnostic considerations in electromechanical dissociation [editorial]. Am J Cardiol 1976; 38:268-69
6 Morgan BC, Crawford EW, Guntheroth WG. The hemodynamic effects of changes in blood volume during intermittent positive-pressure ventilation. Anesthesiology 1969; 30:297-305
8 Pepe PE, Marini JJ. Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction: the auto-PEEP effect. Am Rev Respir Dis 1982; 126:166-70
9 Bergman NA. Intrapulmonary gas trapping during mechanical ventilation at rapid frequencies. Anesthesiology 1972; 37:626-33

Endobronchial Actinomycosis Simulating Bronchogenic Carcinoma

Diagnosis by Bronchial Biopsy

Ilana Ariel, M.D.;* Raphael Breuer, M.D.;† Nidal S. Kamal, M.D.; Issachar Ben-Dov, M.D.;‡ Paul Moide, M.D.;§ and Eliezer Rosenmann, M.D. ¶

Five cases of actinomycosis of the main bronchi or trachea which were suggestive clinically of bronchogenic carcinoma are described. In four patients the correct diagnosis was made by a bronchial biopsy or wash, or both. Three of them recovered following antibiotic treatment, and one died a few days after bronchoscopy. In one case the Actinomyces were found in the bronchial wash retrospectively following diagnosis of pulmonary actinomycosis in the lobectomy specimen. A concomitant endobronchial lipoma was found in one of the patients. The diagnosis of pulmonary actinomycosis by bronchial biopsy may save the patient major surgical intervention.

(Chest 1991; 99:493-95)

The clinical presentation of thoracic actinomycosis has changed considerably over the last decades together with the reduction of its incidence.14 Cases with extensive destruction of the chest wall and formation of discharging sinus tracts are now seldom seen. The presenting symptoms of actinomycosis nowadays are unresolved pneumonia or a pulmonary infiltrate or a mass evidenced on routine chest x-ray films. When these are found in an elderly person they are naturally suspected to be bronchogenic carcinoma, unless proven otherwise.

We present five cases of actinomycosis of the trachea or major bronchi, which were suggestive of bronchogenic carcinoma, that were diagnosed by bronchial biopsy or wash or both.

Case Reports

Case 1

A 59-year-old woman was admitted because of a cough productive of purulent sputum for four months and a recent episode of hemoptysis. Chest x-ray films revealed a segmental infiltrate in the anterior medial segment of the left lower lobe. A diagnosis of

*Department of Pathology, Hadassah University Hospital Mount Scopus. Senior Lecturer of Pathology, the Hebrew University—Hadassah Medical School, Jerusalem, Israel
†Pulmonary Unit, Hadassah University Hospital, Kibbutz Hadassah, Lecturer of Internal Medicine, the Hebrew University—Hadassah Medical School.
‡Pulmonary Unit, Ittihad Hospital, Nablus, Israel.
§Department of Internal Medicine, Hadassah University Hospital Mount Scopus. Lecturer of Internal Medicine, the Hebrew University—Hadassah Medical School.
¶Department of Radiology, Hadassah University Hospital Mount Scopus. Clinical Associate Professor of Radiology, the Hebrew University—Hadassah Medical School.
Reprint requests: Dr. Rosenmann, Department of Pathology, Hadassah University Hospitals, Professor of Pathology, the Hebrew University—Hadassah Medical School.