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# Left Tension Pneumothorax Mimicking Myocardial Ischemia after Percutaneous Central Venous Cannulation

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Although many complications associated with central venous cannulation have been described,[1–4] electrocardiographic (ECG) changes are rarely thought of as a sign of pneumothorax. This case report is presented to heighten awareness of this possibility. It also reviews the literature describing ECG changes due to a pneumothorax that may mimic myocardial ischemia.

## CASE REPORT

An 88-yr-old man was admitted with an intertrochanteric fracture of the right femur following a fall. The patient denied any history of dyspnea on exertion, stating that he frequently walked two blocks without shortness of breath.

On examination, he weighed 63 kg and appeared dyspneic at rest, with a respiratory rate of 18 breaths/min, blood pressure of 140/88 mmHg, and pulse of 88 beats/min. Physical examination revealed findings consistent with congestive heart failure: jugular venous distension, bilateral 2+ pitting edema up to the knees, and rales at the bases of both lungs. Preoperative echocardiogram showed mild aortic sclerosis, concentric left ventricular hypertrophy, and adequate left ventricular systolic function. The patient was treated with furosemide and nitrates preoperatively for heart failure.

Two days after admission, the patient was brought to the operating room for open reduction and internal fixation of the right femur. Because of the signs and symptoms of congestive heart failure, pulmonary artery catheterization prior to induction was planned to monitor the patient's fluid status and left ventricular compliance. Monitors were applied, including pulse oximeter and ECG leads II and $V_5$. The patient's initial vital signs were blood pressure of 145/75 mmHg, pulse of 80 beats/min, respiratory rate of 18 breaths/min, and oxygen saturation of 96% on room air. Despite multiple attempts, the internal and external jugular veins on both the right and left sides could not be cannulated. During these attempts, the patient was very cooperative, was comfortable breathing room air with hemoglobin oxygen saturations by pulse oximetry ($Sp_{O_2}$) between 95 and 97%, did not cough or complain of chest pain or shortness of breath, and did not have more than 10% change in vital signs. Finally, the right internal jugular was cannulated, and a pulmonary artery catheter was passed without difficulty. The initial pressures were pulmonary artery pressure of 30/10 mmHg, pulmonary artery occlusion pressure of 8 mmHg, and central venous pressure of 12 mmHg with appropriate respiratory variation.

Induction of anesthesia was performed using fentanyl 350 μg, thiamylal 50 mg, and vecuronium 8 mg, and the lungs were ventilated *via* mask prior to tracheal intubation. Hemodynamic variables remained within 10% of baseline values during induction and intubation. Breath sounds were initially diminished on the left side, causing us to withdraw the endotracheal tube so that the tube was taped at 22 cm measured from the right angle of the mouth. Breath sounds on the left side improved, and mechanical ventilation was begun. At this time, it was noted that the QRS amplitude of the $V_5$ lead had changed compared to that before induction. No other hemodynamic change was noted. A 12-lead ECG was obtained in the operating room (fig. 1). The QRS axis had a rightward shift, suggesting the possibility of anterolateral

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Key words: Anesthesia techniques: central venous cannulation. Complications: pneumothorax. Heart: ischemia.

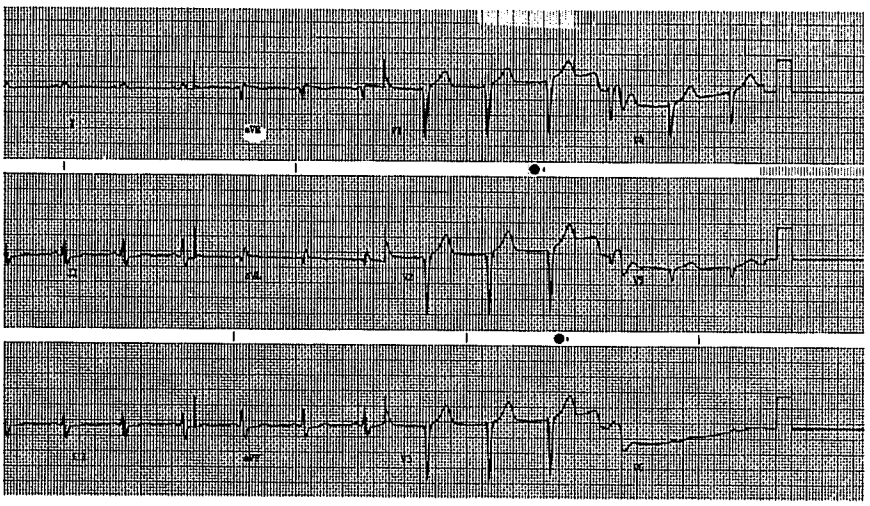

FIG. 1. Postintubation ECG showed diminished R-wave progression from V2 to V4.

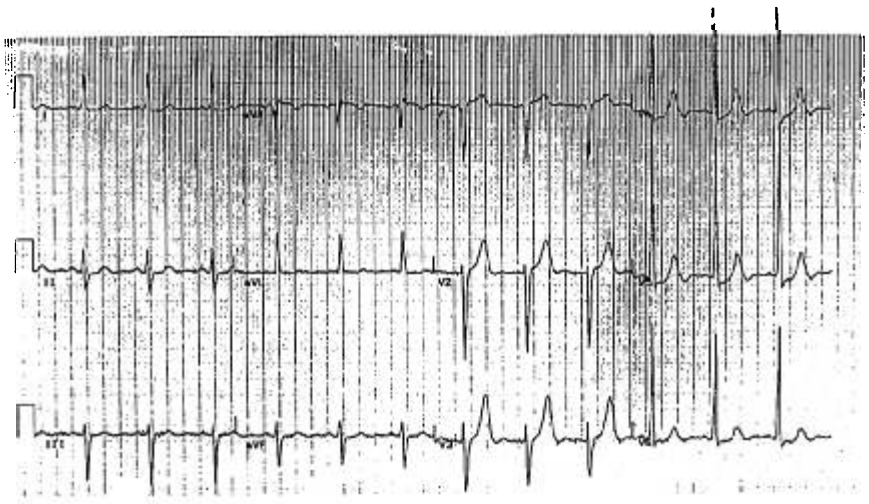

FIG. 2. Preoperative ECG showed positive R waves in leads V2–V4 and no evidence of ischemia.

ischemia. This finding was new compared to the patient's preoperative ECG (fig. 2), although it is possible that some of the QRS axis change was secondary to a difference in lead placement. Since the surgical procedure was urgent and the morbidity associated with a hip fracture was great, a decision was made to proceed with the surgery.

Anesthesia was maintained with 50% nitrous oxide, 0.5% isoflurane, and intravenous fentanyl. A nitroglycerin infusion was started intraoperatively and titrated to systemic blood pressure of 110/60 mmHg and pulmonary artery occlusion pressure of 10 mmHg in an attempt to resolve the ischemia diagnosed by QRS height and axis shift. Systolic blood pressure remained stable at 110 to 130 mmHg, peak airway pressure at 26 $cmH_2O$, and $Sp_{O_2}$ between 97 to 99% on $FI_{O_2}$ of 50%. No wheezing was heard through the esophageal stethoscope. The patient received two units of packed red cells for intraoperative blood loss.

At the end of the surgery, the patient was transferred to the postanesthesia care unit while still mechanically ventilated. At this time, it was noted that the left side of the patient's chest was not moving as much as the right. Postoperative chest x-ray revealed left pneumothorax with shift of mediastinum and trachea to the right (fig. 3). A chest tube was placed, and the ECG reverted to its preoperative pattern. Cardiac isoenzymes were normal, and the chest tube was removed after 3 days. The patient was subsequently transferred to a rehabilitative hospital for physical therapy.

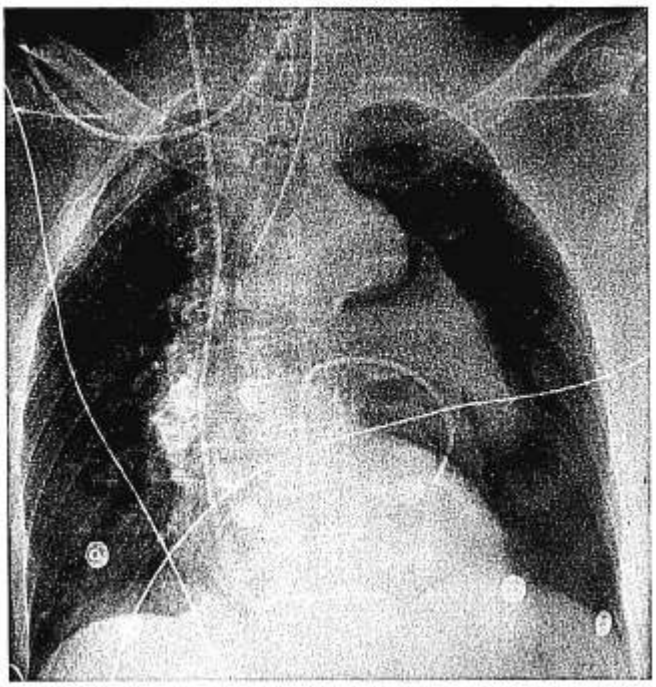

FIG. 3. Chest x-ray showing left-sided pneumothorax, collapsed left lung, and slight shift of mediastinum and trachea to the right.

## DISCUSSION

Symptoms of pneumothorax following percutaneous central venous catheterization include coughing, chest pain, and shortness of breath.[5] Signs include wheezing, asymmetric chest movement, decreased breath sounds on the affected side, tracheal shift away from the affected side, and decreased $Sp_{O_2}$. In our patient, none of the above was noticed, and the patient did not cough or become dyspneic. In this patient, the first and only sign of pneumothorax was the change in ECG as illustrated in figures 1 and 2. The QRS axis shifted to the right with the rotation of the heart from increased left-sided intrathoracic pressure. The patient's blood pressure, oxygen saturation, pulmonary artery wedge pressure, and end-tidal carbon dioxide tension did not change.

In our patient, the ECG did not show any ST–T wave changes associated with myocardial ischemia. The ECG changes that occurred during this case have been described in a previous case report[6] in which spontaneous left tension pneumothorax masqueraded as an anterior myocardial infarction. Gould *et al.*[7] described similar ECG changes in a patient after left pneumonectomy: loss of R-wave progression and diminished QRS voltage in precordial leads. ECG changes associated with 30% or more left pneumothorax include: 1) right shift of QRS axis, 2) di-

minished QRS amplitude, 3) decreased R-wave progression, and 4) inversion of precordial T waves.[8] The most important feature of ECG changes associated with a pneumothorax is the acute reversal of these changes after insertion of a chest tube.

We report this case as an example of tension pneumothorax mimicking myocardial ischemia to alert readers to include pneumothorax as part of the differential diagnosis of intraoperative ECG changes.

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# Postoperative Unilateral Pulmonary Edema: Possible Amiodarone Pulmonary Toxicity

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Amiodarone is a highly effective antiarrhythmic drug for treating otherwise refractory ventricular and atrial arrhythmias. It has been shown to have numerous serious adverse side effects, including pulmonary toxicity.[1] The overall incidence of amiodarone pulmonary toxicity (APT) is estimated at 5–15% with a reported mortality rate of 5–10%.[1–4] The cause of APT is not clear. One theory is that amiodarone enhances free oxygen radical production in the lung, and that the free oxygen radicals in turn oxidize cellular proteins, membrane lipids, and nucleic acids. It is suggested that high fractions of inspired oxygen ($F_{I_{O_2}}$) may accelerate these reactions.[5]

Some patients receiving amiodarone will eventually come to surgery for automatic internal cardiac defibrillator (AICD) insertion because of continued or recurrent refractory arrhythmias. This surgery is often performed *via* a left anterolateral thoracotomy requiring a period of one-lung ventilation. We report two cases of patients receiving amiodarone therapy who presented for this operation. The lungs of both patients were exposed to high $F_{I_{O_2}}$s during one-lung ventilation, and on the continuously ventilated side both patients developed unilateral pulmonary infiltrates that, though not indisputably proven, were consistent with APT.

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Key words: Lung: oxygen toxicity; unilateral pulmonary edema. Anesthetic techniques: one-lung ventilation. Pharmacology, antiarrhythmics: amiodarone.

## Case Reports

*Case 1.* A 59-yr-old, 75-kg man with a long history of coronary artery disease, three previous myocardial infarctions, and two separate coronary bypass procedures presented for placement of an AICD because of multiple episodes of ventricular tachycardia.

Two years prior to admission, treatment with amiodarone 600 mg per day was begun. Despite this, during the 4 months prior to this admission he developed further episodes of ventricular tachycardia. The amiodarone was discontinued on the day of admission to the hos-