Pulmonary emboli are complex syndromes of altered coagulation and perfusion that remain prevalent among the population, especially the hospitalized. Adequate preparation by the clinicians and realization of the subtle yet potentially catastrophic nature of pulmonary emboli are critical when surgical intervention is required.

This case report describes a 49-year-old woman with a diagnosis of massive pulmonary embolism who was brought to the operating room for emergent pulmonary artery embolectomy. Despite a profound obstruction in her main pulmonary artery, she arrived in no acute distress and with stable hemodynamic values. During induction of general anesthesia, she quickly decompensated, requiring emergent cardiopulmonary bypass. Intraoperative transesophageal echocardiography guided the multistep surgery, resulting in the recognition of a recurrent right atrial embolus. The patient tolerated the procedure and ultimately had a favorable hospital course.

Keywords: Echocardiogram, embolectomy, pulmonary embolus.

Venous thromboemboli are estimated to occur among the US hospitalized population at an astounding 1 per 1,000.¹ Most patients with pulmonary thromboemboli have concurrent venous thromboses, thus representing a persistent health problem for which the prevalence has changed very little over the past 20 years.²,³ This rate may be artificially low due to fatal but undiagnosed emboli in the absence of an autopsy. Of those with venous thromboembolism, mortality is reported to be 12% to 18%.¹,³

Pulmonary thromboembolus is a frequently discussed complication of surgical and medical patients, in which alterations in coagulation and perfusion result in debris collecting within the branches of the venous side of the body between the right atrium and the alveolar-capillary interface.⁴ Pulmonary emboli may be composed of a mobile plaque from a deep vein thrombus (DVT), products of gestation (amniotic fluid emboli), air, fat, or other foreign bodies.

The presentation of pulmonary thromboembolus may involve a range of symptoms, including shortness of breath, chest pain, hypotension, tachycardia, acidosis, and evidence of right-sided heart failure.⁴ Physiologic responses may be limited with smaller emboli and escalate to severe and life-threatening with larger occlusions. The severity may be gauged by the number, size, and position of the emboli.⁵ Although smaller emboli travel to the lower portions of the lung, larger emboli obstruct primary vessels up to and including the main pulmonary artery (PA).⁶ Additionally, comorbidities such as coronary artery disease, preexisting congestive heart failure, and lung disease decrease the threshold for tolerating this abnormal condition.

Essential to the recognition of a pulmonary thromboembolus is an appreciation of the physiologic manifestations of impeded blood flow from the right side of the heart to the pulmonary capillary bed. In essence, blood is prevented from interfacing the ventilated oxygen in the lungs while the systemic left side of the heart is prevented from receiving the sequestered volume on the right. Treatment involves supporting the oxygenation and perfusion of the body, prevention of additional pulmonary thromboemboli with DVT prophylaxis, placement of an inferior vena cava filter, and medical vs surgical management of the in situ pulmonary thromboembolus.⁷,⁸

Case Summary
This report describes a 49-year-old woman who presented to the emergency department by ambulance after a syncopal episode at home. Presenting symptoms included tachycardia, hypotension, and complaints of ongoing chest pain and shortness of breath that had worsened over the last week. Although this was the first incident of syncope, she stated the accompanying symptoms began shortly after her bilateral mastectomy approximately 2 weeks previously. Evaluation in the emergency department included an extremity Doppler ultrasound showing a DVT in the right leg, as well as a computed tomography scan with angiography showing a large saddle embolus in the main PA. The patient was admitted to the intensive care unit and underwent placement of an inferior vena cava filter. She received cardiology and cardiothoracic surgery consultations, was evaluated with transthoracic echocardiography (TTE), and was anticoagulated with intravenous (IV) heparin and enoxaparin (Lovenox). An anesthesia provider was consulted to prepare the patient
for emergency pulmonary artery embolectomy after it was determined by TTE that additional emboli were located in the right atrium, placing her at risk for cumulative clots in the lung and for catastrophic sequelae.

The patient was transported to the preoperative holding area for anesthetic assessment. She presented sitting upright on the hospital bed, was pleasant, and was in no acute distress. Initial findings included height and weight of 167.6 cm and 77.9 kg, respectively; no allergies; and a past medical history of hypertension, hyperlipidemia, and breast cancer. Past surgical history revealed no anesthetic complications. It had been more than 12 hours since her last oral intake. Vital signs were blood pressure (BP), 114/68 mm Hg; heart rate 98/min in sinus rhythm; respirations, 18/min and unlabored; and oxygen saturation, 97% on 3 L/min of nasally administered oxygen. Physical assessment included clear breath sounds bilaterally, regular heart rate and rhythm, a Mallampati II airway class, and no shortness of breath or chest pain while at rest. Her laboratory values were all within normal range with the exception of her coagulation profile, which showed an induced international normalized ratio (INR) of 1.9. Of note, her family history included a pulmonary thromboembolus in her father. Although presence of a pulmonary thromboembolus in a family member itself does not signify a greater risk of embolism, certain coagulopathies that promote emboli have been identified as hereditary.9 Two of these coagulopathies are the mutation of factor II and factor V, for which she tested negative.

In preparation for surgery, 2 mg of midazolam was given IV in the existing 18-gauge peripheral catheter. A second IV access was established with a 16-gauge catheter, and a radial arterial line was placed to monitor BP directly. Throughout the procedures, the patient remained interactive and without distress. Blood products were ordered and made immediately available. The patient was then taken to the operating room (OR).

While in the sitting position, preoxygenation with 100% oxygen by mask was begun and 5-lead electrocardiogram, pulse oximeter, noninvasive BP cuff, and arterial line BP monitors were applied. With the surgeon immediately available, the patient was placed supine as induction was begun. A rapid sequence induction with cricoid pressure was administered using 20 mg of etomidate, 250 μg of fentanyl, 100 mg of lidocaine, and 160 mg of succinylcholine. Immediately upon induction, the arterial BP was noted to be rapidly falling. Intubation proceeded without difficulty, and a 7.5-mm oral endotracheal tube was placed with a clear view of the glottis. Bilateral breath sounds, end-tidal carbon dioxide, and chest rise confirmed placement of the tube. The bed was placed in a steep Trendelenburg position, and a central venous line was placed into the right internal jugular vein. Both peripheral IV lines were opened while epinephrine and vasopressin boluses were administered for treatment of systolic BPs that had rapidly decreased below 60 mm Hg and a heart rate that had suddenly dropped under 40/min. Chest compressions immediately followed after arterial line BP monitoring demonstrated no appreciable pulsatile flow (flat arterial tracing). Within 1 to 2 minutes of medication administration, BP and heart rate resumed preoperative levels, and general anesthesia was maintained with desflurane in oxygen, scopolamine, and pancuronium.

A transesophageal echocardiography (TEE) probe was placed to evaluate the embolus and cardiac function. Initial findings included profoundly large and extensive emboli in the right atrium, an enlarged and strained right ventricle, and a decreased left ventricular end-diastolic volume (LVEDV; Figures 1 to 4). These were
all expected findings given the known severity of the emboli. Epinephrine and dobutamine infusions were started to sustain the systemic BP and cardiac output while a crystalloid fluid bolus was started through the central venous line.

As the preparation and draping process was being completed, the arterial line BP trends began to decline again despite the vasoactive infusions. The surgeon quickly ordered heparinization and proceeded to make a median sternotomy to initiate emergency cardiopulmonary bypass. The TEE revealed that despite resuscitative chest compressions, the emboli in the right atrium appeared unchanged.

After the heart was arrested, the right atrium was opened, and a large amount of clot was removed. The main PA was also opened, and a large Y-shaped clot was removed from the bifurcation of the right and left PA (Figure 5). In addition, multiple smaller emboli were removed from distal pulmonary arteries. Once the surgeon had removed all visible clots and all vessels and the atrium were closed, the patient was warmed, and standard interventions were performed to wean the patient from cardiopulmonary bypass.

As blood flow was allowed through the heart, it was observed on TEE that the right atrium was beginning to fill with additional emboli. It was hypothesized that a thrombus previously attached on the proximal side of the inferior vena cava filter had broken free and traveled up the vena cava into the heart. Literature supports this finding of multiple clots outside the lung identified by TEE in the presence of in situ pulmonary emboli. In fact, a 26% incidence of concurrent extrapulmonary emboli (in the right atrium and vena cava) has been reported in patients having PA embolectomy. The surgeon again arrested the heart and opened the atrium to find a very long embolus, conforming much to the appearance of the inferior vena cava and mobile in the right atrium. This was the sole clot observed; thus, the atrium was again closed, and preparations were made to wean from cardiopulmonary bypass.

Following the removal of the aortic cross-clamp, an epinephrine infusion and milrinone loading dose were started to ensure cardiac contractility (especially of the right ventricle) and to minimize pulmonary hypertension. As bypass was discontinued, TEE showed no evidence of emboli in any location, the right ventricle was now ejecting well, and the left ventricle demonstrated an appropriate LVEDV. Following protamine administration, anticipated microvascular bleeding was observed and was addressed by giving 3 U of fresh frozen plasma and 2 U of platelets. A norepinephrine infusion was
initiated after persistently low systemic vascular resistance measurements and hypotension continued despite repeated boluses of phenylephrine.

Coagulation profile laboratory work was obtained because of continued multisite bleeding. After discussion with the surgeon, 4 U of cryoprecipitate were administered because of his concern over persistent surgical field bleeding. These units were given before the posting of the laboratory results (INR 1.6) because of suspected post-bypass coagulopathy. In addition to 800 mL of blood by intraoperative cell salvage, 1 U of packed red blood cells was given to maintain a hematocrit greater than 25%. As blood product administration achieved hemostasis and normovolemia, the hemodynamic trends stabilized and systolic BPs were sustained between 110 and 120 mm Hg. Pulse oximetry and arterial blood gas monitoring revealed satisfactory oxygenation with measurements between 95% and 100% and greater than 200 mm Hg, respectively. Following chest closure, the patient was transported to the cardiovascular intensive care unit, where she was extubated in less than 12 hours and all vasopressors discontinued.

On postoperative day 1, the patient was interviewed while sitting in a bedside chair. She was breathing well on nasally administered oxygen, had no complaints of shortness of breath or chest pain, and commented that her previous fatigue and air hunger were completely resolved. The remainder of her hospital course was uneventful, and she was discharged home.

Discussion
This patient initially presented with the classic symptoms of pulmonary thromboembolus. Although she presented with mild symptoms in the OR, her seeming stability masked the gravity of her severe underlying condition. Relative health before this incident likely influenced her resilience despite poor pulmonary blood flow. Two issues at the forefront of attention were, first, the subtlety of the profound pathology and, second, the necessity of having interventional equipment in place for thoracic cases. As to the former, this patient presented unassumingly on nasally administered oxygen and sitting up in bed. Her vital signs had normalized from the emergency department, and she gave no outward evidence of critical impedance to blood flow. Even as she was placed on the OR table, her vital signs did not change until induction was in progress and her position changed to supine. The clinical application is that the severity of disease is not linear with the intensity of given symptoms. To address the increased risk, some clinicians have suggested institution of femoral-femoral cardiopulmonary bypass before induction of general anesthesia because of the 19% incidence of cardiac collapse. Her nearly fatal pulmonary thromboembolus was primarily evident in the medical history and invasive testing results. Vigilance in assessment and knowledge of the pathology are paramount to being prepared for these cases.

With regard to preparedness, early insertion of an arterial line and large-bore IV catheter proved to be both the warning system and the treatment route (respectively) for the cardiovascular collapse. Establishing these lines after induction would have resulted in a delayed realization of the hypotension relative to the number of minutes between cycles of the noninvasive BP. Possibly even more critical would be the notably increased difficulty in obtaining such access in the face of severe hypotension, presence of vasopressors, and the mounting number of urgent tasks to be completed by the anesthesia care team.

Immediate availability of TEE and interpretation by a qualified practitioner served to assist in the anesthetic management of volume, inotropes, and vasopressors. Use of the TEE prevented the premature completion of the surgical procedure because of recognition of the subsequent emboli in the right atrium. Additionally, the TEE guided multiple stages of the operation, including vascular access placement and resuscitation management. It is the author’s opinion that attentiveness to the subtle embolic pathology and the anesthetic readiness directly facilitated this patient’s survival and management of a critical pulmonary thromboembolus and PA embolectomy.

REFERENCES
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