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second stage of an amobarbital interview. This is not usually the case if there is no psychiatric disease.<sup>8</sup> The mechanism of amobarbital has been shown to be the enhancement of the GABA receptor function.<sup>9</sup>

In this case report, the patient had a brief atypical catatonic psychosis, most likely resulting from stimulation of the reticular formation with antagonism of the GABA receptors function by the high dose of steroids. Her stuporous condition, together with her myasthenia gravis, resulted in respiratory difficulties. We believe that etomidate reversed this effect by antagonizing the steroid-induced changes. Hence, etomidate caused arousal rather than sedation in this particular patient.

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## Fatal Pulmonary Fat Embolism in the Early Postoperative Period

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PULMONARY fat embolism is a well-recognized complication of major orthopedic surgery.<sup>1-5</sup> Most cases occur intraoperatively during pressurization and manipulation of long-bone intramedullary canals.<sup>1-3,5,6</sup> Embolic events that occur in the postoperative period may be insidious, with their clinical sequelae evolving progressively.<sup>4</sup> We report an unusual case of acute, massive pulmonary fat

embolism that occurred in the early postoperative period.

## Case Report

A 76-yr-old man was scheduled for left total hip arthroplasty during combined regional-general anesthesia. Two previous left total hip arthroplasties were uncomplicated. He had a history of hypertension, a 6-cm infrarenal abdominal aortic aneurysm and hypothyroidism. There was no documented history of coronary vascular disease. Medications included isosorbide dinitrate, metoprolol, and L-thyroxine. Results of physical examination were within normal limits; baseline preoperative systolic blood pressure was 120-150 mmHg and heart rate was 50-60 beats/min. Preoperative laboratory data were normal. The electrocardiogram showed only sinus bradycardia. Chest radiograph was normal.

Lumbar plexus psoas compartment and sciatic Mansour approach nerve blocks were performed using bupivacaine. General endotracheal anesthesia was induced and maintained with fentanyl, isoflurane, nitrous oxide, and oxygen. Arterial and central venous pressure monitoring and intraoperative autologous blood salvaging were used.

Despite an estimated 3,000-ml blood loss, the patient remained

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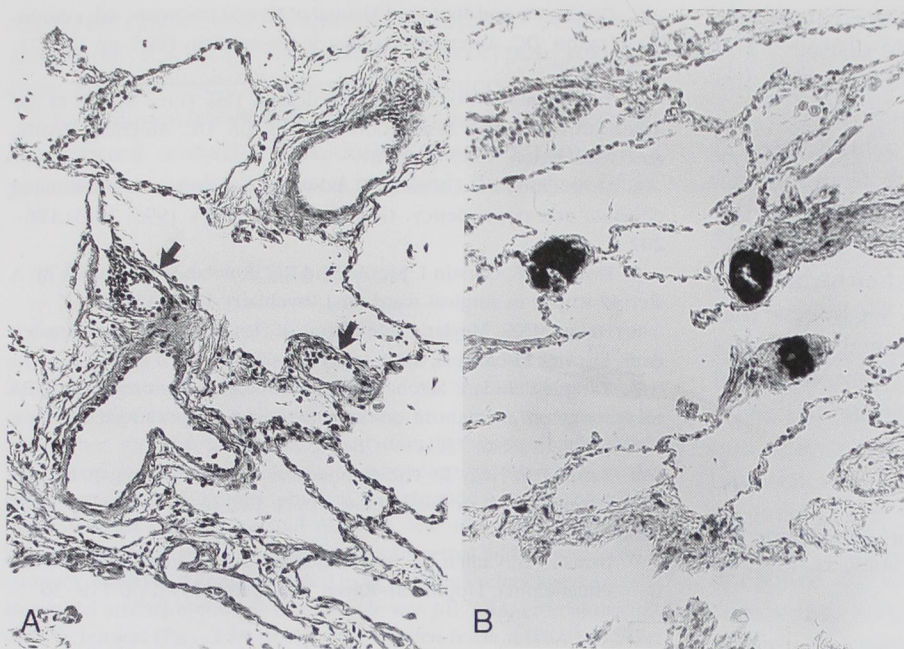


Fig. 1. (A) Histopathology of lung parenchyma taken at autopsy of this patient (hematoxylin and eosin stain,  $\times 200$ ). Note capillary lumens (arrowheads). Intraluminal fat was washed out by organic solvent staining before paraffin embedding. (B) Histopathology of lung parenchyma taken at autopsy of this patient (osmium tetroxide stain,  $\times 200$ ). Osmium-stained fat appears black, filling capillary lumens in central portion of slide.

hemodynamically stable throughout the intraoperative period, with systolic blood pressure ranging from 90–130 mmHg, heart rate ranging from 50–70 beats/min, central venous pressure ranging from 8–14 mmHg, and a urine output of 250 ml during the 6 h procedure. Oxygen saturation ( $Sp_{O_2}$ ) remained above 95% throughout the procedure. There were no untoward events intraoperatively. Intraoperative fluid replacement consisted of 5 units of packed erythrocytes, 1,400 ml of autologous salvaged blood, 6 l of crystalloid and 1 l of hetastarch solution.

At the completion of the procedure, the patient was awakened in the operating room, his trachea was extubated, and he was taken to the postanesthesia care unit. At arrival, he was drowsy but conversant and receiving oxygen *via* nasal cannula. For the first 3 h, the patient remained sleepy with easy awakening and was pain free requiring no supplemental analgesia. Systolic blood pressure was 100–140 mmHg, heart rate was 70–80 beats/min, central venous pressure was 10–14 mmHg, and urine output was 50–100 ml/h.  $Sp_{O_2}$  remained more than 94%. Electrocardiography remained without significant change. Hematocrit concentration was 35% and potassium concentration was 5.6 mEq/l, but electrolytes were otherwise normal. Approximately 3.5 h after arrival in the postanesthesia care unit, the patient became mildly confused, although he remained hemodynamically stable and well oxygenated. Thirty minutes later, in the absence of any physical manipulation of the patient,  $Sp_{O_2}$  decreased to 70–80% and the patient became severely dyspneic. The patient's trachea was intubated but, within 2 min, systolic blood pressure decreased to 50–60 mmHg. The electrocardiogram showed 4-mm ST-segment elevation in leads  $V_2$ – $V_5$ . Multifocal premature ventricular complexes, ventricular tachycardia, and ventricular fibrillation developed. Resuscitation efforts were unsuccessful.

At autopsy, the primary cause of death was diagnosed to be acute, massive pulmonary fat embolism with acute cor pulmonale. In addition, early, mild, left ventricular ischemia and moderate coronary vascular disease were present: 20% left anterior descending coronary artery and 60% right coronary artery stenoses. There was no evidence

of intracardiac or pulmonary vascular thromboembolic clot. The diagnosis of pulmonary fat embolism was based on autopsy findings of massive, diffuse osmium tetroxide-stained fat embolization in all pulmonary capillaries and several larger pulmonary vessels and of acute cor pulmonale at autopsy, confirming the basis for clinical deterioration in our patient.

At autopsy, histologic sampling of lung tissue with hematoxylin and eosin stain was unrevealing. Tissue treated with osmium tetroxide stain, one of the few staining techniques available for assessing the presence of fat in histologic tissue samples, showed large fat globules throughout the pulmonary vasculature, allowing confirmation of the diagnosis (figs. 1A and 1B).

## Discussion

Pulmonary fat embolism resulting from traumatic long-bone injury was first described in detail by Ernst von Bergmann in 1873. He documented the clinical course, deterioration, death, and autopsy findings of a patient with a comminuted fracture of the distal femur. At autopsy, von Bergmann's patient was found to have had massive pulmonary fat embolism.<sup>7</sup>

Cardiopulmonary collapse during this patient's early postoperative course had several possible causes. Acute myocardial infarction, ruptured abdominal aortic aneurysm, and postoperative surgical bleeding were considered to be unlikely because of the absence of angina, electrocardiographic abnormalities, or hemodynamic instability before collapse. The rapid progression of the patient's deterioration, in the absence of these findings

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and in the presence of sudden, severe dyspnea and hypoxia, suggested a primary respiratory basis for the clinical decompensation.

The presence of fat diffusely throughout the pulmonary vasculature explains the mechanism of clinical decompensation in patients with massive fat embolization. Massive obstruction of the pulmonary vasculature leads to marked intrapulmonary shunting, hypoxia, and respiratory failure. Furthermore, the marked increase in pulmonary vascular resistance leads to right-sided heart failure.

Surgical technique may influence the incidence and severity of fat embolization. Prevention of pulmonary fat embolism includes careful intramedullary lavage before placement of the prosthesis to decrease the amount of debris available for embolization.<sup>8</sup> In addition, the severity of embolism during revision arthroplasty may be influenced by the tools used for dismantling the prosthesis and removing the intramedullary cement. Ultrasonic tools produce a greater embolic load than either osteotomes or high-speed burr drills.<sup>9</sup> Another technique available to decrease the severity of embolization is venting of the femoral canal. Femoral venting works by decompressing the medullary canal before pressurization,<sup>2,10</sup> but such venting may increase the likelihood of shaft fracture or lead to extravasation of cement after the vent is removed. As a result, femoral venting techniques are not used at our institution, but meticulous medullary lavage was used and osteotomes were used to remove cement from the medullary canal.

Although pulmonary fat embolization is generally subclinical in nature,<sup>9</sup> patients at an especially high risk for clinical compromise include those who have reduced cardiopulmonary physiologic reserve, patients with pathologic fractures, and those with the presence of a route for intracardiac right-to-left shunting, especially patent foramen ovale.<sup>4,6</sup>

It is important in the setting of acute perioperative decompensation of patients undergoing or having undergone major operative procedures that the possibility of pulmonary fat embolism be entertained. In situations in which fat emboli are suspected, it is imperative that tissues be stained with osmium tetroxide, before the application of routine stains, to confirm the clinical diagnosis.

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