tion might result in dislodgement of tracheal membranes leading to airway obstruction. Furthermore, an endotracheal tube could aggravate existing edema and inflammation. Anesthesia *via* mask should be considered for these patients when general anesthesia is necessary. Regardless of the choice of airway management, the availability of a skilled bronchoscopist for diagnosis and therapy of airway obstruction is advised.

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Intraoperative Re-expansion Pulmonary Edema

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Re-expansion pulmonary edema has been reported following evacuation of both chronic pneumothorax[1,2] and pleural effusion.[3] It is an uncommon, but potentially life-threatening, condition which has not been described in the perioperative period. We describe a case of intraoperative pulmonary edema that occurred following rapid re-expansion of a lung that had been chronically collapsed due to a malignant pleural effusion.

CASE REPORT

A 60-yr-old, 88.5-kg, 177-cm man presented with a 6-month history of dyspnea due to a large right pleural effusion caused by a previously diagnosed pleural mesothelioma. The patient's past history was significant for hypertension, a 40 pack-year history of smoking, and a 12-yr

exposure to silica. The patient was taking no medications. Preoperative pulmonary function studies revealed restrictive disease with a vital capacity of 1640 ml (45% of predicted), and obstructive disease with a 1-s forced expiratory volume of 1310 ml. Ventilation-perfusion studies revealed a matched defect in ventilation and perfusion involving the entire right lung field both in the anterior and posterior views. Echocardiography showed normal left and right heart chambers with no evidence of pericardial effusion. Preoperative arterial blood gas analysis demonstrated a respiratory alkalosis with a pHa of 7.50, Pa_{CO_2} = 31 mmHg, and a Pa_{O_2} = 79 mmHg. The patient received digitalis preoperatively and iv aminophyline.

After breathing 100% oxygen, anesthesia was induced with thiopental, 250 mg iv, and fentanyl, 100 μg iv. Succinylcholine, 100 mg iv, was given to facilitate endotracheal intubation, and anesthesia was maintained with 1% isoflurane, 50% N_2O in O_2, and incremental doses of fentanyl, 50 μg IV. The patient was placed in the left lateral decubitus position and, shortly after surgery began, 3,200 ml of pleural effusion was rapidly removed through a right thoracotomy incision. The patient underwent a right sub-total pleurectomy with re-expansion of the right lung, implantation of I[125] needles, and a partial pericardiectomy with marlex mesh reconstruction.

The patient's intraoperative course was stable initially, with the Pa_{O_2} ranging from 100–181 mmHg during controlled ventilation with a FI_{O_2} of 0.5. The central venous pressure ranged between 10–12 cm H_2O, and the urine output was 90 ml per hour. Three hours after the evacuation of the pleural effusion, and while the I[125] implants were being placed, straw-colored, slightly hemorrhagic fluid was noted to be exiting from the endotracheal tube. Despite frequent endotracheal suctioning, the anesthetic circuit had to be changed four times over the next 1–2 h due to the volume of fluid coming from the endotracheal tube. Chemical analysis of the fluid revealed a protein value of 4.0 g/dl and an albumin of 2.6 g/dl, values consistent with pulmonary edema fluid. Pa_{O_2} was 229 mmHg, while being ventilated with 100% oxygen during this time.

Fiberoptic bronchoscopy was conducted while the patient was in the left lateral decubitus position. There was no sign of airway obstruction,

but straw-colored fluid was noted to be exiting only from the right main stem bronchus. Over the next 2–3 h (6 h after induction of anesthesia), the pulmonary edema subsided and clinically disappeared. The patient was brought to the recovery room breathing spontaneously with the endotracheal tube in place. A chest radiograph at that time showed a small right apical pneumothorax and multiple implants consistent with the operation performed. There was no residual evidence of pulmonary edema in either lung field.

Despite an apparently normal respiratory excursion and respiratory rate, arterial blood gases in the recovery room were poor, with a $P_{a_{O_2}}$ of 50 mmHg while breathing 40% oxygen via a T-piece. Morphine was given iv for pain, and the patient received several IPPB treatments with bronkosol in an effort to alleviate the obstructive component of his respiratory condition. When no further evidence of respiratory compromise was evident, the trachea was extubated (5 h after admission to the post-anesthesia care unit). Shortly thereafter, the $P_{a_{O_2}}$ was 95 mmHg while breathing 40% O_2 via a face mask. After spending 16 h in the post-anesthesia care unit, the patient was returned to his hospital room and apparently remained stable over the next 24 h.

On the third postoperative day, the patient developed new onset atrial fibrillation, and impending respiratory failure was diagnosed. A chest radiograph showed right lung collapse, despite the presence of functioning chest tubes. He was transferred to the intensive care unit. Endotracheal intubation was performed and ventilatory support was instituted. The patient continued in a progressive downhill course and, as a result of his extensive mesothelioma, expired 2 weeks later following a cardiopulmonary arrest.

DISCUSSION

This case demonstrates an unusual type of non-cardiac pulmonary edema after re-expansion of a lung that had been collapsed for many months. The unilateral nature of the pulmonary edema and the absence of cardiac or other known etiologies strongly suggests, by exclusion, a relationship between the process of rapid pulmonary re-expansion and the development of intraoperative pulmonary edema.

Development of unilateral pulmonary edema in a re-expanded lung is a rare but recognized complication of both pneumothorax evacuation[1,2] and rapid removal of pleural effusion.[3,4] It has not been reported as an intraoperative complication. The earliest accounts of this syndrome were published in 1875.[5] In these cases, the pulmonary edema followed percutaneous evacuation of large volumes of pleural fluid, and it was noted as early as 1902 that this problem seldom occurred unless more than 2 l of fluid was removed.[4,5] A symptom-free period of several minutes to hours was also noted, as was the fact that the pulmonary edema often cleared rapidly. However, there were some patients who subsequently expired, usually secondary to acute respiratory insufficiency.[6,7]

The pathophysiology of re-expansion pulmonary edema is poorly understood. Currently, it is thought that there is injury to pulmonary capillaries during the period of lung collapse, resulting in an increased alveolar capillary membrane permeability.[8] With sudden re-expansion of the lung, there is an increase in pulmonary capillary blood flow, that results in the movement of plasma ultrafiltrate from the capillaries into the interstitium and alveolar spaces.[3,6]

We feel that this case report is unique in that the re-expansion pulmonary edema occurred intraoperatively. Our patient had prolonged collapse of the lung, documented by ventilation-perfusion scan, and over 3 l of fluid were quickly removed intraoperatively. While it has been recommended that patients with massive pleural effusion should undergo slow aspiration of no more than 1500 ml at a time, this is clearly not possible during a thoracotomy.[3,6] In addition, a subtotal pleurectomy was performed, thus permitting rapid re-expansion of the lung. The pulmonary edema occurred intraoperatively within 1 h of lung re-expansion, and clinically disappeared over the next 2–3 h. With positive pressure ventilation, the patient maintained adequate oxygenation. While the use of PEEP is a standard therapeutic modality for pulmonary edema, it was not needed in this case for oxygenation, and the surgeon expressed concern that PEEP might interfere with the surgical procedure. By the time the patient was taken to the recovery room, the pulmonary edema was no longer present either clinically or radiographically. The patient was awake, alert, and seemed to have adequate ventilatory exchange. His respiratory status appeared to be normal for a patient following a major thoracotomy; it was not until 48 h later that he developed lung collapse and respiratory insufficiency. The patient's extensive malignancy also contributed to his continued respiratory deterioration. Undoubtedly, it was a combination of the re-collapse of the affected lung and his underlying disease that ultimately led to his demise.

In conclusion, we believe that anesthesiologists should be aware of the possibility that re-expansion pulmonary edema may occur intraoperatively. Furthermore, it is crucial to keep in mind that possibly fatal total lung collapse may occur up to 48 h later. While prevention of intraoperative re-expansion pulmonary edema may not be possible, it is clear from our experience that close postoperative observation and ventilatory support is required even though the patient may have a symptom-free period early in the postoperative course.

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Unexpected Focal Neurologic Deficit on Emergence From Anesthesia: A Report of Three Cases

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The occurrence of a new neurologic deficit on emergence from anesthesia may be the result of a focal or global cerebral insult. Patients experiencing a global cerebral insult from profound hypotension, hypoxia, or cardiac arrest may have diffuse neurologic abnormalities without localizing signs. A systemic insult can also occur in a patient who has a focal lesion making one region of the central nervous system more vulnerable to injury, such as hypotension in a patient with carotid artery stenosis. A patient emerging from anesthesia with a new and unexpected focal neurologic abnormality must be assumed to have suffered an acute focal injury from either ischemia or hemorrhage. The three cases below demonstrate various etiologies for unexpected focal neurologic deficit on emergence from general anesthesia.

CASE REPORTS

Case 1. A 65-yr-old, 72-kg man with a horseshoe kidney and a large staghorn calculus in his right renal pelvis underwent percutaneous ultrasonic lithotripsy following percutaneous placement of a wire into the renal pelvis under local anesthesia. His medical problems included hypothyroidism treated with synthyroid 0.125 mg daily, chronic pulmonary interstitial fibrosis, coronary artery disease with an old anterior myocardial infarction, and history of one episode of cardiac syncope due to episodic atrial fibrillation 1½ yr prior to this hospitalization. Preoperative physical examination revealed bibasilar rales, but was otherwise normal. Laboratory values were normal. ECG showed

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normal sinus rhythm, old anterior myocardial infarction, and a T wave abnormality. No premedication was administered.

General anesthesia was induced with thiopental 400 mg iv and maintained with 50% nitrous oxide and 0.5–1.5% enflurane in oxygen. Endotracheal intubation was facilitated with succinylcholine 60 mg iv. The patient was placed in the prone position with his head turned to the left. During positioning and patient preparation for the procedure, the patient's arterial blood pressure fell from an initial awake 140/85 mmHg to 90/55 mmHg. This hypotension responded over a 20-min period to an infusion of 1 liter of 5% dextrose in lactated Ringer's solution and mephenteramine 7.5 mg iv. The remainder of the intraoperative period was uneventful and the patient remained hemodynamically stable. The patient was transported to the recovery area with his trachea intubated and breathing spontaneously.

Ten minutes postoperatively, it became apparent that the patient was more lethargic than expected and had no motor function in the right extremities. On physical examination, sensory function was normal, but there was dense right hemiplegia, positive right Babinski sign, right facial motor deficit, and marked dysphasia, expressive more than receptive. The cranial nerve reflexes were intact. There were no carotid bruits and the heart was normal on auscultation. The patient underwent immediate computerized tomography (CT) head scan that showed no focal changes. He remained hemodynamically stable during the postoperative period and, with spontaneous respiration supplemented by 70% O_2 through a close-fitting mask, Pa_{O_2} was 150 mmHg, Pa_{CO_2} 38 mmHg, and pHa 7.38. Further evaluation included normal oculoplethysmography and digital venous angiography revealing normal extracranial carotid arteries with diffuse disease of the medium-sized intracranial vessels. Echocardiography revealed mild left atrial enlargement, apical left ventricular aneurysm, and mural thrombus. Diagnosis was felt to be focal left cerebral hemispheric infarction, probably due to emboli of cardiac origin.

The patient showed gradual improvement in his motor and speech deficit, beginning 8 h postoperatively. Therapy included anticoagulation for 3 months begun 4 days postoperatively, and physical and speech therapy. Within 2 weeks, the patient had near complete return of motor function. The patient was discharged 2 weeks postoperatively with a slight foot drop and weakness of the biceps and flexor muscles of the wrist and hand. The patient was interviewed by telephone 2 yr following the procedure, and reported complete functional recovery occurring within a few weeks after discharge.

Case 2. A 32-yr-old, 61-kg woman with persistent pelvic pain underwent pelvic laparoscopy. Her medical problems included chronic diarrhea and pelvic pain of 3 yr duration. Her medications included only