anesthesia machine, and maintaining desflurane 1 MAC. When hemodynamic instability began and temperature increased, the diagnosis was confirmed.

This case of MH involved desflurane and SCh. The relatively slower onset may relate to the delaying effect of nondepolarizing relaxants and depressants in slowing the onset of MH.[4] Further, increased ventilation for management of increased expired CO_2 may in part mask and delay the diagnosis of MH.[5] Comprehensive monitoring and a high degree of suspicion, *i.e.*, vigilance, remain fundamental factors for a timely diagnosis.

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Negative-pressure Pulmonary Edema Associated with Saber-sheath Trachea

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NEGATIVE pressure pulmonary edema (NPE) has been associated with the relief of upper airway obstruction in otherwise healthy patients.[1-5] The suggested pathophysiologic mechanisms have included increased venous return, reduced ventricular compliance, interventricular septal shift, and increased afterload.[2] "Saber-sheath" is a morphologic description applied to a trachea that is narrowed in the coronal plane and elongated in the sagittal plane.[6,7] It has been associated with obstructive lung disease and hypothesized to develop as a result of a type of tracheomalacia (Todd T, Winton T: Personal communication, April 1998).[7,8] This case report describes an episode of NPE associated with a saber-sheath trachea.

Case Report

A 77-yr-old man with a fractured proximal humerus presented for shoulder arthroplasty. The patient described symptoms of dyspnea on minimal exertion and was clinically diagnosed with obstructive lung disease. He had a history of congestive heart failure, a remote myocardial infarction, gastroesophageal reflux disease (GERD), and a 50 pack-yr smoking history. He had undergone laparoscopic cholecystectomy 2 yr previously, reportedly uneventfully. Reduced breath sounds in all lung fields were noted. Electrocardiogram (ECG) showed a left bundle branch block (LBBB). Chest radiograph revealed flattened hemidiaphragms.

The patient was premedicated with ranitidine and metoclopramide and taken to the operating room where the usual monitors were applied, including ECG, pulse oximeter, and noninvasive blood pressure cuff. An arterial line was placed in the nonoperative extremity. The patient was preoxygenated and induced in a rapid sequence fashion (fentanyl, 100 μg; propofol, 100 mg; and rocuronium, 50 mg), with cricoid pressure applied. Mild resistance to passage of the endotracheal tube (ETT) was encountered at 22 cm, which was overcome using slight rotation of the ETT, completing insertion to 24 cm.

The surgery was completed uneventfully, with minimal blood loss and infusion of 1.0 l of crystalloid. After return of consciousness and reversal of muscle relaxation (glycopyrrolate, 0.4 mg; pyridostigmine, 10 mg), the patient's respirations were rapid and shallow. Assessment of neuromuscular function indicated probable complete reversal (sustained tetanus for 5 s at 50 Hz), and positive pressure ventilation was resumed. Auscultation revealed coarse bilateral crackles. Chest radiograph showed bilateral infiltrates and a saber-sheath trachea (fig. 1), the latter having been overlooked on preoperative films. ECG showed LBBB in the same configuration as preoperatively. Diuresis was initiated with intravenous furosemide, 40 mg, (30 min postoperatively), and the patient was transferred to the intensive care unit. The differential diagnosis included high-pressure pulmonary edema caused by heart failure and low-pressure pulmonary edema caused by aspiration or NPE. NPE was suspected because of the sudden onset of symptoms on emergence. A pulmonary artery catheter was placed (90 min postoperatively), which showed a pulmonary artery occlusion pressure (PAOP) of 12 mmHg and pulmonary artery pressure

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Key words: Dyspnea; postoperative.

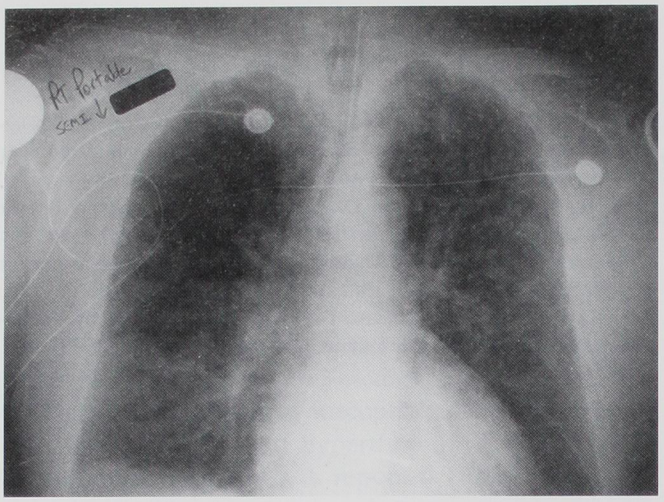

Fig. 1. Chest radiograph taken in recovery room, illustrating characteristic distal tracheal tapering of saber-sheath trachea and bilateral infiltrates compatible with pulmonary edema.

(PAP) of 30/15. During endotracheal suctioning, passage of the catheter had been noted to be difficult. Subsequent fiberoptic bronchoscopy revealed an apparent external compression of the right tracheal wall, extending from the ETT orifice to a point approximately 4 cm proximal to the carina. The tracheal lumen was reduced in coronal caliber by approximately 75% but was unchanged sagitally. Subsequent computed tomography scan of the thorax (fig. 2) demonstrated that the tracheal obstruction was caused by partial collapse of its wall rather than by external compression. The proximal extent of the collapse was unknown because of the stenting effect of the ETT, however, and it was unclear that the patient would tolerate extubation from continuous positive airway pressure (CPAP). The patient was taken to the operating room again (24 h later) for controlled extubation, with the room prepared for emergency rigid bronchoscopy or tracheostomy. While awake and spontaneously breathing on CPAP, the patient was extubated and tolerated this well. Serial myocardial enzymes were negative for infarction. Arterial blood gases on discharge from hospital 5 days later showed *p*H, 7.34; PO_2, 62 mmHg, PC_{O_2}, 55 mmHg, and HCO_3, 31 mM. Preoperative ABGs were not available. Pulmonary function tests at 4 weeks showed an FEV_1 of 0.91 l and forced vital capacity of 2.1 l, and concurrent flow–volume loop was compatible with a variable intrathoracic obstruction.

Discussion

This case illustrates probable NPE in a patient with a saber-sheath trachea. Although causation of NPE by the saber-sheath trachea is difficult to prove unequivocally, circumstances were strongly suggestive. At first, congestive heart failure caused by ischemic heart disease intuitively seemed more likely. Although myocardial ischemia was difficult to exclude completely given the LBBB on the patient's ECG, no myocardial infarction was detected by serial enzyme evaluation. Furthermore, his PAOP and PAP were normal shortly after the start of diuresis. Aspiration of gastric contents was possible with the history of GERD, but the rapid resolution of symptoms excluded it.

The etiology of NPE is multifactorial and has been reviewed elsewhere.[1,2] It is probably caused by a combination of preload and afterload stress, septal shift, and transient ventricular dysfunction during a hypoxemic, hyperadrenergic state. Although this patient's pulmonary function was limited, negative pleural pressure of −50 cm H_2O has been reported as being sufficient to cause NPE.[2]

The saber-sheath trachea is a morphologic description of a rare form of tracheomalacia. It is thought to be caused by calcification, stiffening, and subsequent microfracture and destruction of the normally elastic tracheal rings (Todd T, Winton T: Personal communication, April 1998).[7,8] Alternatively, deposition of abnormal collagen may be the cause. It may extend from the glottis to involve the mainstem bronchi and is untreatable in the latter. More limited disease in a younger patient may be considered for radical treatment, such as tracheal resection or stenting.

A saber-sheath trachea was previously reported in association with difficulty forming a seal with an ETT cuff.[6] The patient in that case had an unusually large saber-sheath trachea. The usual saber-sheath trachea is narrowed substantially in the coronal plane, causing an overall reduction in caliber,[7] as was the case with this patient.

The possible reasons for this patient's symptoms on emergence and resolution at extubation were multiple. On emergence, the combination of an ETT and zero end-expi-

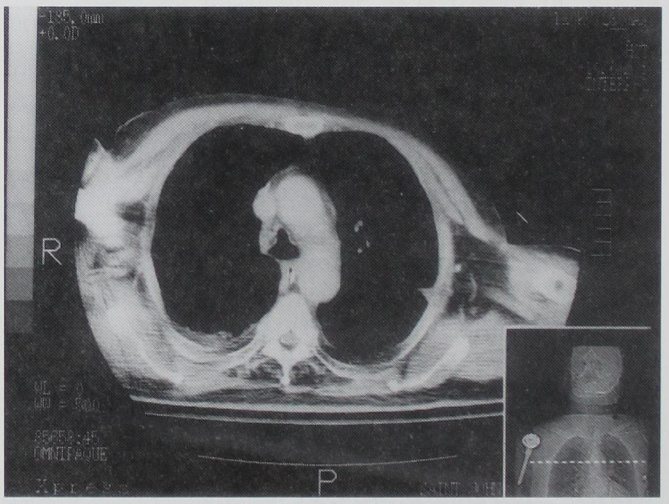

Fig. 2. Computed tomographic scan of thorax showing partial collapse of distal tracheal wall and normal surrounding structures. The tracheal index (coronal diameter divided by the sagittal diameter) was 0.4, well below the upper limit reported.[7]

ratory pressure may have superimposed sufficient work of breathing in this patient with decreased pulmonary reserve to cause symptoms. At successful extubation, this patient was on CPAP, which may have stented his airway sufficiently to relieve partial collapse. Residual neuromuscular blockade, although not apparent in this patient, might still have been detected had more sophisticated evaluation been available, *e.g.,* double-burst stimulation. Finally, the patient's ventricular function was known to be decreased and probably was unable to adapt to changing preload and afterload stressors.

Although the diagnosis of NPE in this patient was primarily by exclusion, its management was straightforward and consisted of ventilation and diuresis. However, the patient's ability to maintain adequate ventilation after extubation was uncertain because the proximal extent of tracheal collapse was unknown. Consequently, extubation was performed in the operating room with the availability of a rigid bronchoscope and tracheostomy equipment.

In summary, this patient with chronic obstructive pulmonary disease (COPD) and saber-sheath trachea suffered pulmonary edema associated with emergence from general endotracheal anesthesia. Saber-sheath trachea in this patient, as is typical, resulted in significant reduction in tracheal caliber caused by reduction in strength of the tracheal cartilage. Pulmonary edema in this patient was most likely low pressure, compatible with the diagnosis of NPE. Awareness of the association of saber-sheath trachea with COPD[7] in similar patients is probably warranted.

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Anaphylactic Shock to Neuromuscular Blocking Agent: A Familial History

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THE incidence of anaphylactic reactions during anesthesia is estimated in France to be approximately 1/3,500 general anesthesia.[1–3] Muscle relaxants are the most common cause of anaphylactic reactions during anesthesia.[3,4] We report two cases of anaphylactic shock in relation to the administration of nondepolarizing neuromuscular blocking agents that occurred in the members of a family, which suggests that there may be a familial predisposing factor to anaphylactic reactions induced by muscle relaxants.

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Key words: Anaphylaxis; muscle relaxants; heredity.

Case Report

A 62-yr-old woman weighing 50 kg without preexisting disease was undergoing thyroidectomy during general anesthesia for euthyroid goiter. Anesthesia was induced with 150 μg fentanyl and 350 mg