Examining the removed nasotracheal tube (fig. 1) revealed a large 15-cm intraluminal thrombus. A significant volume of blood was entrapped in the nasotracheal tube during intubation. The supine position of the patient pooled the blood in the dependent portion of the tube, and clot formation followed. Numerous case reports have documented the hazards of nasotracheal intubation. To our knowledge, this particular complication has been reported only once before, but not in the anesthesia literature.1

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Hemoptysis from a Pulmonary-artery Catheter

To the Editor:—We have observed a patient in whom hemoptysis associated with a fan-shaped density on chest roentgenography developed following placement of a pulmonary-artery catheter. An 83-year-old paraplegic woman arrived in our emergency room in septic shock (mean blood pressure 40 mm Hg) from pyonephrosis. Following treatment with fluids, antibiotics, steroids and digoxin, the patient was brought to the operating room for a right pyelolithotomy. A triple-lumen balloon-tipped catheter was inserted into the right subclavian vein using an introducer and its location verified by pressure tracings. Pulmonary arterial pressure was 32/13 mm Hg and pulmonary capillary wedge pressure (PCWP) was 11 mm Hg. Anesthesia was induced with thiopental and maintained with nitrous oxide, fentanyl and pancuronium. After the patient was turned to the left lateral decubitus position, adequate tracings from the pulmonary-artery catheter were difficult to obtain despite balloon inflation, 1-inch withdrawal of the catheter, and frequent flushings. Arterial pressure was maintained at 140/90 mm Hg and pulse 80–90/min.

In the recovery room, with the patient supine, pulmonary arterial tracings were obtained. Pulmonary arterial diastolic (PAD) pressure and pulmonary capillary wedge pressure (PCWP) were zero during a period of supraventricular tachycardia (144/min) and arterial hypotension (60/40 mm Hg). A roentgenogram of the chest at this time was normal. Eighteen hours after operation bright red blood (100–200 ml) was suctioned from the endotracheal tube. Fiberoptic bronchoscopy did not reveal a source of bleeding. A portable chest roentgenogram taken at this time showed bilateral pleural effusions and a fan-shaped localized infiltrate in the right lower lung distal to the catheter tip (fig. 1). The pulmonary-artery catheter was removed, the hemoptysis stopped, and over the next four days the fan-shaped density resolved.

Asymptomatic fan-shaped roentgenographic densities at the tips of pulmonary-artery catheters have been reported previously.1 Mechanical erosion or perforation2 from catheter tip motion, or an actual tear of the artery wall from balloon overinflation, coupled with a large catheter loop, may have caused
the hemoptysis. If hemoptysis occurs or new pulmonary changes near the catheter tip are seen, the catheter should be removed without delay.

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Reductive Metabolism of Halothane and Hepatotoxicity

To the Editor:—Eger1 rightly points out that the currently identified reductive metabolites of halothane have not been shown to be toxic in themselves. However, we have recently reported hepatic functional and morphologic abnormalities that accompany increases in concentrations of volatile reductive halothane metabolites in Fischer 344 rats subjected to enzyme induction and hypoxia.2,3 Specifically, concentrations of 1,1-difluoro-2-chloroethylene (CDF) and 1,1,1-trifluoro-2-chloroethane (CTF) were low in the absence of abnormalities in hepatic function or morphology. However, a four- to eightfold increase in reductive metabolism of halothane resulted from enzyme induction and moderate hypoxia (oxygen, 14 per cent) and this was associated with hepatic necrosis in all animals at 24 hours after anesthesia; appropriate controls showed no such abnormalities. These findings satisfy accepted criteria for hepatic damage due to direct cellular injury. The formation of CDF and CTF could only proceed via an intermediate free radical or carbamion,3,4 so it is most likely that these metabolites merely serve as a "marker" of reductive metabolism and that hepatic cellular injury results from the intermediate carbamion or free radical. These observations were made in an animal model with oxidative and reductive halothane metabolism similar to that of man, and the lesion described was similar to that previously reported in cases of presumed halothane hepatitis. Thus, the findings in our animal model have pertinence to the important question of hepatic effects of halothane in man.3,4

We found in man that reductive halothane metabolites were present in the exhaled air during halothane anesthesia with 100 per cent oxygen at concentrations higher than those reported after anesthesia by Sharp et al.5 Our studies also show that concentrations of volatile reductive halothane metabolites rapidly increase from the start of anesthesia, reach a plateau after one hour, and decline rapidly after anesthesia. In conclusion, there seems little doubt that the reductive metabolism of halothane has significance for human hepatic toxicity of halothane, and that the volatile reductive metabolites CDF and CTF are useful markers for reductive metabolism.

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