General Hospital with a 2-week history of dyspnea, nonproductive cough, and weight loss. Despite a strong history of ethanol abuse, he denied any prior history or symptoms of pancreatitis. The initial chest x-ray film demonstrated a large left pleural effusion and a small right pleural effusion. The left effusion was hemorrhagic with an elevated amylase concentration of 3,230 Somogyi units. The serum amylase concentration was 66 Somogyi units. Pleural biopsy was negative for acid-fast bacilli and malignancy. The right pleural effusion was transudative with an amylase concentration of 45 Somogyi units. The patient underwent tube thoracostomy and drainage of the left pleural effusion. A subsequent computed tomographic (CT) scan of the chest and abdomen showed a posterior mediastinal cystic mass with extension into the abdominal cavity, suggesting the diagnosis of a mediastinal pseudocyst. Conservative management with hyperalimentation was attempted; however, 5 weeks later the patient developed *Staphylococcus aureus* bacteremia and hemodynamic instability necessitating the administration of vasopressors. We proceeded with CT-guided percutaneous catheter drainage of the intra-abdominal cyst, which resulted in concurrent drainage of the thoracic cyst. After initial improvement, the patient's condition deteriorated over the ensuing 4 weeks. Candidemia developed, and the patient eventually died.

This case demonstrates an alternative approach to that of Zeilen-der et al to a patient who is not a surgical candidate if conservative therapy and hyperalimentation are not effective. Often, CT-guided drainage of the abdominal cyst will also result in drainage of the thoracic component and clinical improvement. There have been two previous reports documenting this technique in the management of complications arising from a mediastinal pancreatic pseudocyst. Our case underscores several points in the approach to these patients. First, although a history of pancreatitis or abdominal trauma can be elicited in most cases, symptoms relating to the pleural effusion may be the sole manifestation of pancreatitis. Second, the elevated fluid amylase concentration was the first clue to the diagnosis; therefore, the amylase level should be determined routinely in evaluation of chronic effusions. Some reports suggest that a pleural fluid amylase value significantly higher than the simultaneous serum value is pathognomonic for a pancreaticogenic effusion. To a lesser degree, this can also be seen in some malignant effusions. Third, we concur that CT is a sensitive tool in confirming the diagnosis.

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Figure 1. Image from ERCP study shows tracking of contrast material (curved arrows) superiorly from head of pancreas to mediastinal pseudocyst (open arrow). Injection into ampulla also filled dilated pancreatic duct (solitary straight arrow) and dilated common bile duct (double straight arrows), changes consistent with chronic pancreatitis.

To the Editor:

We thank Drs Read and Richardson for their comments. We agree that CT-guided drainage of the abdominal cyst will often result in drainage of the thoracic component and is a therapeutic alternative in the patient who is not a surgical candidate.

Our patient eventually underwent endoscopic retrograde cholangiopancreatography (ERCP), which demonstrated the fistulous tract between the pancreatic duct and the mediastinal pseudocyst (Fig 1). Thus, ERCP may be an alternative or complementary modality for diagnosis of pancreatic pseudocyst formation.

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Self-extubation

To the Editor:

We read with interest the article by Coppolo and May regarding self-extubation. We would like to share a similar prospective study with a smaller number of patients done at our institution over a 7-month period from February 1989 to September 1989.

We compared the effects of sedation and restraints on patients who extubated themselves while on a ventilator. An average of 16 patients per day were followed up over a 7-month period. The
Lymphoid Interstitial Pneumonitis and Infection with Human Immunodeficiency Virus Type 2

To the Editor:

Lymphoid interstitial pneumonitis (LIP) was frequently seen in children with acquired immunodeficiency syndrome (AIDS) infected by human immunodeficiency virus type 1 (HIV-1). LIP have also been reported in HIV-1 infected adult patients. Human immunodeficiency virus type 2 (HIV-2) was identified in 1986 and has been associated with AIDS. To our knowledge, LIP associated with HIV-2 infection has not yet been described. We report such a case.

A 55-year-old French homosexual man presented in February 1984 with cryptococcal meningitis. In April 1985, chest x-ray film showed a bilateral reticular pattern without mediastinal lymph-node enlargement. Open lung biopsy was performed and a diagnosis of LIP was established. Specific staining for pathogens was negative. On immunologic testing, he had negative tuberculin skin tests and low absolute numbers of CD4 + lymphocytes (50/µm³) and a CD4 + /CD8 + ratio of 0.18. From April 1985 to June 1986 he was chronically ill with intermittent diarrhea and weight loss. He died in July 1986. Autopsy findings showed a high-grade non-Hodgkin’s lymphoma made up of diffuse, large cells with involvement of the lungs, liver, spleen, and myocardium.

Serum samples taken and stored in 1985 were tested for the presence of HIV-1 and HIV-2 antibodies using an enzyme-linked immunosorbent assay and Western blot analysis (Diagnostics Pasteur). No antibodies to HIV-1 were detected, but HIV-2 infection was diagnosed by the presence of antibodies to HIV-2 envelope glycoproteins gp 41 and gp 130-165. Serologic test for human T-cell leukemia virus type 1 was negative.

Our patient was probably infected with HIV-2 by multiple homosexual contacts while travelling in West Africa (Guinea-Bissau). In this case, LIP showed some features distinctive from previously reported cases of LIP in adults with HIV-1 infection. First, the occurrence of LIP in a white man contrasts sharply with the usual occurrence of LIP in black HIV-1 infected patients originating from Central Africa. Second, the patient did not show salivary gland enlargement or persistent generalized lymphadenopathy, as reported in the HIV-1 infected patient with LIP (this being the pulmonary manifestation of a more diffuse CD8 + lymphocytic visceral infiltration). Infection with HIV-2 may have a direct causal role in the development of LIP, as has been suggested with HIV-1. HIV-2 infection should be looked for in patients with LIP.

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To the Editor:

We thank Dr Brandstetter for his comments regarding our article. It is interesting that his findings about sedation and restraints are comparable with ours. We agree that a better method of restraint would be of value. However, we have also found that a truly determined patient will exhale himself despite the best attempts at restraint.

After reviewing our data, we believe that virtually 90 percent of patients who deliberately exhale themselves needed more timely weaning and extubation rather than more formidable restraint. We hope that our observations will serve as a stimulus for all of us to critically examine our current criteria for weaning and extubation of ventilated patients.