charts were reviewed for only those patients who extubated themselves and were reintubated. Of the eight patients who extubated themselves, four patients had been initially sedated, three patients had been restrained, one patient had been both sedated and restrained, and one had been neither. Following reintubation, five patients were put on restraints as well as sedation, and three were put on restraints only. The patients were followed up, and only one patient extubated himself again.

Our findings were consistent with those described by Coppolo and May, who also looked at the effects of sedation or restraints in preventing self-extubation. Most of the patients in their study who extubated themselves were either sedated, restrained, or both. In our study restraints seemed to be better than sedation since all of the eight patients who extubated themselves and were reintubated were put on restraints and only five of them received additional sedation. We believe that more effective means of restraint should be devised that are easily tolerable by the patient yet effectively prevent self-extubation.

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Chest 1990; 98:165-69

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 extubate himself despite the best attempts at restraint.

After reviewing our data, we believe that virtually 90 percent of patients who deliberately extubated 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.

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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). Cases of 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/mm³) 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 (Diagnosics 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 diffuse CD8+ lymphocytic 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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