not have active Pneumocystis carinii pneumonia (PCP). All of these patients had a history of single or recurrent episodes of PCP and were receiving aerosolized pentamidine isethionate as prophylaxis against recurrence of PCP. It was suspected that the pneumothoraces were caused by coughing due to the irritative effect of pentamidine therapy on the airways superimposed on abnormal decreased lung compliance secondary to interstitial fibrosis caused by previous PCP.

From January 1, 1988, to January 1, 1991, 1,200 known human immunodeficiency virus-positive patients were admitted to our institution. Thirty-two patients had spontaneous pneumothorax either on admission or during hospitalization. Twenty-four patients (75 percent) with spontaneous pneumothorax were not receiving PCP prophylaxis, and only three patients (10 percent) were receiving aerosolized pentamidine prophylaxis. The incidence of spontaneous pneumothorax in our AIDS group admitted to the hospital was 2.7 percent.

At the Fifth International Conference on AIDS, Newsome et al.3 reported that pneumothorax occurred in eight (2.5 percent) of the 327 patients with prior PCP who had been receiving aerosolized pentamidine prophylaxis for three to 13 months; the majority (75 percent) had evidence of active PCP.

When we compared the incidence of pneumothorax in patients receiving aerosolized pentamidine prophylaxis (2.5 percent) with that in our group, who for the most part received no prophylaxis, we found no statistical difference. Although we cannot exclude the possibility that inhaled pentamidine can directly cause pneumothorax, the evidence presented more likely implicates predisposing damage from prior episodes of PCP or, more likely, ongoing tissue destruction from recurrent or active infection.4 It would be helpful to know whether other institutions have any significant differences in the incidence of pneumothorax in patients treated with prophylactic aerosolized pentamidine and in those who receive no prophylaxis.

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Catheter-Related Infections and Associated Septicemia

To the Editor

We are writing to express our concern about the article by Norwood et al.,1 which appeared in the April 1991 issue of Chest. While we agree with the authors that much of the literature addressing this topic is plagued by varying definitions of catheter-related infection and anecdotal evidence, the recommendations that arterial catheters, pulmonary artery catheters, and central venous catheters be changed only when there is evidence of catheter-related local infection or bacteremia is disturbing for several reasons. First, the data presented by the authors are derived mainly from surgical (trauma) ICU populations, which represent a younger population without prior multiple medical conditions. Extrapolation of their data to other surgical and medical ICU patients may not be valid.

Second, the work of Norwood et al. suggests that patients with more severe underlying illness (eg, sepsis) may be at greater risk for catheter-related infection; however, their recommendations do not appear to take this into consideration.

Third, catheter-related infections, including cellulitis, septic thrombophlebitis, and bacteremia, are a major cause of nosocomial infection,2 the risk of which increases with duration of catheterization.3 Catheter-associated bacteremia is associated not only with endocarditis, metastatic infection, and septic shock, but also with prolonged hospitalization and extended intravenous antibiotic therapy. The mortality from staphylococcal bacteremia varies from 21 percent to 62 percent.2 Prevention of these complications should be at the heart of policies regarding intravascular catheters. The incidence of catheter-related infection in our medical ICU has decreased by at least 50 percent since the institution of strict guidelines regarding catheter insertion and duration of catheterization. Only one catheter-associated infection has been reported during the past four reporting months. Furthermore, the complication rate with more frequent catheter placement has remained unchanged at less than 1 percent.

The authors' recommendation to remove central venous and pulmonary artery catheters only after a catheter-related infection is clinically evident leaves us in the less-than-desirable situation of treating, rather than preventing, potentially fatal iatrogenic complications. The only way to minimize catheter-related infection is to limit the time in which central venous and pulmonary artery catheters are in situ. We applaud the efforts by Dr Norwood and his colleagues to help clarify a controversial topic; however, we question their conclusions and the decision by the ACCP Critical Care Council to publish this paper in what appears to be the form of a policy statement.

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To the Editor:

I thank Dr Lipchik and Dr Schapira for their comments about our review article, and I would like to reassure them that we share their concerns about catheter-related infections and associated bacteremia. Our recommendations should not be interpreted as representing a lackadaisical or hopeless attitude toward preventing infection. It is, rather, an attempt to arrive at some conclusions.