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In Reply.—We appreciate the comments of Dreyfuss and Djedaini. Their work related to ventilator circuit changes and nosocomial pneumonia has been carefully conducted and has established a strong role favoring frequent ventilator circuit changes.1–2 As Dreyfuss and Djedaini indicate in their letter, there are several major differences between their work1,2 and ours.3 First, they conducted extensive microbiologic surveillance of ventilator circuits, heated humidifiers, and respiratory secretions. Second, they routinely performed invasive diagnostic studies to establish the presence of pneumonia. Third, their study was limited to a single intensive care unit, and thus their sample size was smaller than ours.

We elected not to conduct cultures of circuits and humidifiers because it has been established that there is little, if any, relationship between ventilator circuit contamination and the incidence of ventilator-associated pneumonia.4–5 When placed into service, ventilator circuits quickly become contaminated, and the source of that contamination is almost always from the patient.5,6 As demonstrated by Dreyfuss et al.7,8 and others,9 the incidence of ventilator-associated pneumonia does not change when a heat-and-moisture exchanger is used to prevent circuit colonization.9 With no available evidence for a relationship between circuit colonization and ventilator-associated pneumonia, we believed that cultures of circuits and humidifiers would not add to the strength of our study design.

A major strength of the work of Dreyfuss et al.7,8 was the use of strict criteria to characterize pathogens producing ventilator-associated nosocomial pneumonia, including specimen brush with quantitative cultures. We did not conduct such intensive microbiologic surveillance for several reasons. First, the microbiology of ventilator-associated pneumonia has been extensively investigated.7–9 Second, invasive techniques are impractical for large studies and may be of limited use in patients already treated with antibiotics. Further, the use of invasive diagnostic testing for ventilator-associated pneumonia is controversial, and it has been suggested that this testing is unnecessary for high-quality patient care.6 Our ventilator-associated pneumonia data were identified prospectively per criteria of the Centers for Disease Control and Prevention (CDC),10 which is the standard used in the United States to identify infections and compare ventilator-associated pneumonia rates to the National Nosocomial Infections Surveillance System of the CDC.11

The purpose of our study was to compare ventilator-associated pneumonia rates with 48-h and 7-day circuit changes. By design, our selection criteria differed from Dreyfuss et al.8 We included all mechanically ventilated adult patients—we did not restrict the study to a single unit nor did we limit the enrollment to a minimum number of ventilator days. Our study included patients in seven intensive care units and patients mechanically ventilated outside of the intensive care unit. As suggested by the CDC, we used ventilator days as the denominator to report ventilator-associated pneumonia rates, because it controlled for the duration of mechanical ventilation.12 Elimination of patients whose lungs were mechanically ventilated 48–72 h13,14 or less skews the duration of mechanical ventilation to a mean that is greater than ours. At the Massachusetts General Hospital, 82% of patients are separated from mechanical ventilation by the 7th day. We agree with Dreyfuss and Djedaini that many of our cases of ventilator-associated pneumonia may have been early-onset pneumonia. However, we do not think that this differs from what commonly occurs in acute care hospitals or that it biases our results.

As Dreyfuss and Djedaini indicate, most studies have found a greater incidence of ventilator-associated pneumonia with a longer duration of mechanical ventilation. Our pneumonia rate was 3.9% for patients whose lungs were ventilated 10 days or less (4.2% in the control group and 3.7% in the study group) and 12% for patients whose lungs were ventilated more than 10 days (14.3% in the control group and 10% in the study group). However, when controlling for the duration of mechanical ventilation, our pneumonia rate was 13.9/1,000 ventilator days for patients ventilated 10 days or less (14.7/1,000 days in the control group and 13.1/1,000 in the study group) and 5.4/1,000 ventilator days for patients ventilated more than 10 days (5.9/1,000 days in the control group and 4.8/1,000 in the study group). Although Dreyfuss and Djedaini are correct in their suggestion that patients whose lungs are mechanically ventilated for longer periods of time are more likely to develop a ventilator-associated pneumonia, investigation of this issue was beyond the scope of our study and cannot be explored in retrospect from the data we collected. Further, we do not believe that mortality associated with 48-h or 7-day circuit changes adds anything to our study design because of the multifactorial nature of mortality.

Dreyfuss and Djedaini take issue with our statement regarding the statistical power of their study. They argue that confidence intervals are more appropriate when interpreting data. Our point was that the sample size in previous studies was relatively small.1,12 Statistically, this could be reflected as a low statistical power or as a wide confidence interval. The confidence intervals reported by Dreyfuss et al. are very wide.1,12 In their first paper,1 the confidence intervals are 16.9–49.3% for the control group (48-h circuit changes) and 13.2–48.7% for the study group (no circuit changes). In other words, it is statistically possible that the pneumonia rate in the control group was 20%, and the pneumonia rate in the study group was 40%. The

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only way to resolve this concern is to increase the sample size, which will narrow the confidence limits and increase the statistical power. Comparison of the Dreyfuss et al.1,2 studies further illustrates the issue related to small sample sizes. In the first study,1 the overall ventilator-associated pneumonia rate was about 50%, whereas it is only about 10% in the second study.3 This might cause one to question whether the subjects in these two studies represent the same population of patients. However, it is likely that these data represent the same population of patients because of the overlap of confidence intervals between the two studies. Statistically, one cannot rule out the possibility that the pneumonia rates would have been similar in the two studies if the sample size was large enough.

Although our study and that conducted by Dreyfuss et al. are methodologically different, we believe that our studies are complimentary, and each adds credibility to the other. Despite different study designs, our conclusions are similar. That is, ventilator circuits can be changed at 7-day or longer intervals, thus reducing costs without detriment to the patient.

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Low-frequency Component of Heart Rate Variability

To the Editor—The data provided by Hopf et al.1 are interesting and provocative. Their conclusions are in stark contrast to the large number of reports published during the last 10 yr and especially the last 2 yr3,4 that further specified the methodology needed to use the low-frequency spectral component as a marker of sympathetic modulation. These authors state that, after segmental epidural anesthesia

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