

CORRESPONDENCE

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Ventilator Circuit Changes and Nosocomial Pneumonia

To the Editor:—Hess *et al.*¹ suggest that ventilator circuit changes could be delayed to 7 days without increasing the incidence of ventilator-associated pneumonia. Indeed, inclusion of a very large number of patients adds tremendous credibility to their paper, which confirms our results.² This paper raises a few issues about which we wish to comment. The first concerns the duration of mechanical ventilation and its interaction with nosocomial pneumonia. According to the data reported by Hess *et al.*, the mean duration of ventilation was less than 6 days, during both the 48-h and 7-day circuit changes. These figures are different from those reported in most studies of ventilator-associated pneumonia (VAP), which were conducted with patients whose lungs were ventilated for much longer periods³⁻⁶ (10–25 days). Most studies also report that VAP occurs after a mean duration of mechanical ventilation of about 10 days.²⁻⁴ Thus, the data presented by Hess *et al.* leave some doubt as to whether the VAP cases were essentially early-onset pneumonia,⁷ which occurs during the first 4–5 days of ventilation and usually is due to such common pathogens as *Hemophilus influenzae* or *Streptococcus pneumoniae*, which do not cause major therapeutic problems because they are usually sensitive to most antibiotics. In contrast, late-onset pneumonia poses very different problems because it may be due to resistant pathogens such as *Pseudomonas aeruginosa* or *Acinetobacter spp.* and is responsible for a substantial increase in mortality.⁴ It would be interesting to know what pathogens were recovered in the study by Hess *et al.* and whether patients with pneumonia had been receiving mechanical ventilation for longer durations than patients without pneumonia and had different mortality rates. Second, despite the fact that we studied only 63 patients, we do not agree with Hess *et al.* that the power of our study was low. Power calculation is relevant to study design but not to the interpretation of data.⁸ Confidence intervals are appropriate when interpreting data.⁸ In our study, the confidence intervals for the incidence of pneumonia in patients with 48-h circuit changes and those with no change at all were strictly superimposable, and the *P* value for the pneumonia rates was 0.8. Because we used specific diagnostic criteria,⁹ it would be unlikely that increasing the number of patients would yield different results. Furthermore, in another study on a larger population,¹⁰ we confirmed that not changing circuits during ventilation with heated humidifiers does not put patients at greater risk of pneumonia. Our initial study² also involved extensive microbiologic surveillance with more than 110 quantitative cultures conducted on four patient and circuit sites in each group (for a total amount of more than 1,000 cultures). There was no difference in patient and circuit colonization, whether or not circuits were changed during the whole duration of mechanical ventilation. Given the pathogenesis of VAP,¹¹ these findings provide strong additional support for not changing circuits at all during mechanical ventilation. The recommendation to prolong the interval of circuit changes from 24 to 48 h was based on the publication by Craven *et al.*,¹² in which a similar number of cultures was performed. Notwithstanding, we believe that data from

a single center always must be questioned by others. We are delighted that the study by Hess *et al.* adds credibility to the possibility of reducing costs without detriment to the patient.

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In Reply:—We appreciate the comments of Dreyfuss and Djedaïni. Their work related to ventilator circuit changes and nosocomial pneumonia has been carefully conducted and has established a strong case favoring infrequent ventilator circuit changes.^{1,2} As Dreyfuss and Djedaïni indicate in their letter, there are several major differences between their work^{1,2} 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.¹ When placed into service, ventilator circuits quickly become contaminated, and the source of that contamination is almost always from the patient.^{4,5} As demonstrated by Dreyfuss *et al.*² and others,⁶ the incidence of ventilator-associated pneumonia does not change when a heat-and-moisture exchanger is used to prevent circuit colonization.² 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.*^{1,2} was the use of strict criteria to characterize pathogens producing ventilator-associated nosocomial pneumonia, including protected specimen brush with quantitative cultures. We did not conduct such intense microbiologic surveillance for several reasons. First, the microbiology of ventilator-associated pneumonia has been extensively investigated.^{1,2,7} 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.⁸ Our ventilator-associated pneumonia data were identified prospectively per the criteria of the Centers for Disease Control and Prevention (CDC),⁹ which is the standard used in the United States to identify infections and compare ventilator-associated pneumonia rates to the National Nosocomial Infection Surveillance System of the CDC.¹⁰

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.*¹ 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.¹¹ Elimination of patients whose lungs were mechanically ventilated 48-72 h^{1,2,7} 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 Djedaïni 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 Djedaïni 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% in the control group and 10% in the study group). However, when controlling for the duration of mechanical ventilation, the 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 Djedaïni 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 Djedaïni 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.^{1,2} 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.^{1,2} 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