Letters to the Editor

Infections Linked to Anesthetic

To the Editor:

A recent article[1] describing investigations conducted by the Centers for Disease Control and Prevention (CDC) following postoperative infections at various hospitals was reported briefly in *Infection Control and Hospital Epidemiology*.[2] In the report by Bennett et al,[1] some findings, mainly epidemiological correlations, indicate that extrinsic contamination of propofol was responsible for infectious symptoms following surgery. However, definite proof could not be provided in any patient due to problems with some of the data. In no single case-patient has it been demonstrated conclusively that an anesthetist or any other healthcare worker transferred microorganisms recovered later from patients into a vial or an ampule of propofol and from these containers to the patient (for discussion, see references 3, 4).

It is interesting to note a major discrepancy between the first CDC report of 1990[5] and the updated report issued in 1995.[1] The first report included five patients in a California hospital who developed surgical wound infections after clean surgical procedures. A throat culture from the anesthetist involved grew *Staphylococcus aureus*, and the phage type was identical to that found in the patients' wounds.[5] In the second report, these patients are presumably among the 16 cases of postoperative infection in Hospital 1. However, no throat culture from an implicated anesthetist is mentioned now, but rather a scalp lesion.[1]

Furthermore, the first report states that the outbreak period for these five patients was 8 days.[5] In the second report, however, there is no outbreak period of 8 days that fits exactly to five patients. If we assume these hospitals to be identical, several more cases, including two fatalities, must have occurred *after* the first CDC investigation. If, on the other hand, the hospitals are not identical, the five patients mentioned in the first report are not included in the second one.[1]

Perhaps there is an easy explanation for these discrepancies. In any case, the authors must be congratulated for their repeated efforts to warn anesthesia personnel about the potential danger to the patients by breakdowns in aseptic technique when handling propofol.

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The author replies.

Thank you for your letter. You are correct that there is a simple explanation for the discrepancies that you note in the reports of infectious complications associated with the use of propofol published in the *Morbidity and Mortality Weekly Report (MMWR)* and the *New England Journal of Medicine (N Engl J Med)*.[1,2] The California hospital investigation included in the *MMWR* was conducted by the County Health Department in California and not directly by my staff at the Centers for Disease Control and Prevention. Therefore, although this investigation was included in the *MMWR,* it was not included in the *N Engl J Med* paper. The *N Engl J Med* paper only included investigations that my staff conducted on-site. Although we assisted several state or local health departments in their conduct of additional investigations, these were not included in the *N Engl J Med* paper. The hospital numbers in the *MMWR* bear no relationship with the numbers of the hospitals in the *N Engl J Med* paper. I hope this clarifies any confusion.

REFERENCES

1. Carr S, Waterman S, Rutherford G, et al. Postsurgical infections associated with an extrinsically-contaminated intravenous anesthetic agent—California, Illinois, Maine, Michigan. *MMWR* 1990;39:426-433.
2. Bennett SN, McNeil MM, Bland LA, et al. Postoperative infections traced to contamination of an intravenous anesthetic, propofol. *N Engl J Med* 1995;333:147-154.

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Clostridium difficile and Sucralfate

To the Editor:

We were delighted to see that our initial study provoked additional inquiry in this area, and we offer the following comments. In our study of 147 critically ill patients, we identified a statistically significant negative association (adjusted odds ratio=0.15, $P<.001$) between sucralfate exposure and a positive *Clostridium difficile* toxin assay.[1] Watanakunakorn et al[2] found no such association in their retrospective study. What might explain these results? The answers may lie in methodological differences and study setting.

In the latter report, controls were selected by a non-random method; exposure assessment was not defined clearly, and it is uncertain whether data abstractors were masked to case-control status of the patient. What was the definition of sucralfate exposure? What was the duration of exposure, and were patients receiving the agent on the day the toxin assay was done? These factors are important in the design and interpretation of case-control studies.[3,4] Furthermore, cases were older, were more likely to be from nursing homes, and were hospi-

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