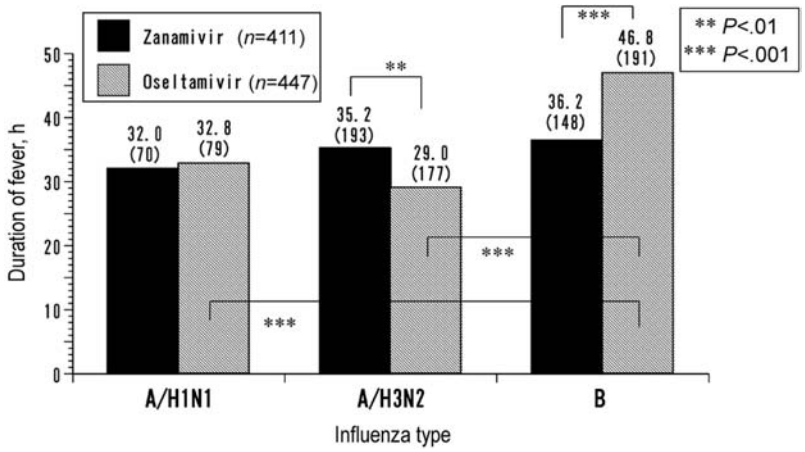

Figure 1. Duration of fever after the first dose of zanamivir or oseltamivir in patients with influenza A/H1N1, influenza A/H3N2, and influenza B virus infection. Numbers shown above the bars indicate percentage (number) of patients.

(±SD) of fever was significantly ($P<$ .01) shorter in those who received oseltamivir therapy (29.0 ± 24.8 h) than it was in those who received zanamivir therapy (35.2 ± 19.7 h). For patients with influenza B, the mean duration (±SD) of fever was significantly ($P<.001$) longer in those who received oseltamivir therapy (46.8 ± 29.6 h) than it was in those who received zanamivir therapy (36.2 ± 19.7 h). Among the patients who received oseltamivir therapy, the duration of fever was significantly longer in those with influenza B than it was in those with influenza A/H1N1 or A/H3N2 (figure 1). However, there were no statistically significant differences in the effectiveness of zanamivir therapy among patients with influenza A/H1N1, A/H3N2, or B (figure 1).

The respective reported mean values of inhibitory concentrations of 50% of zanamivir and oseltamivir were 1.14 and 0.90 nmol/L for influenza A/H1N1, 2.09 and 0.73 nmol/L for influenza A/H3N2, and 4.15 and 11.53 nmol/L for influenza B [8]. These findings may explain our results from a clinical context, because they reveal that oseltamivir is slightly more effective against influenza A/H3N2 and less effective against influenza B, compared with zanamivir therapy; this would indicate zanamivir for the treatment of influenza B virus infection. Recently, the World Health Organization reported that 39% of influenza A/H1N1 virus isolates worldwide were resistant to oseltamivir [9]. Zanamivir is indicated for use against influenza A/H1N1 virus strains that are resistant to oseltamivir. In conclusion, compared with zanamivir, oseltamivir was shown to be less effective against influenza B but more effective against influenza A/H3N2.

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Paradoxical Relationship between the Clinical Outcome of *Staphylococcus aureus* Bacteremia and the Minimum Inhibitory Concentration of Vancomycin

To the Editor—We investigated the relationship between the MIC of vancomycin and clinical outcomes in patients with *Staphylococcus aureus* bacteremia who were treated with vancomycin. Overall, patients who were infected with strains for which the MIC of vancomycin was >1.5 μg/mL had a lower 3-month mortality rate than did patients who were infected with strains for which the MIC was <1 μg/mL. This is in contrast to findings from previously published work [1].

A prospective clinical study was undertaken from August 2006 through March 2007 of cases of *S. aureus* bacteremia at a teaching hospital on the south coast of England. Clinical data were collected by patient interview and case note review. Patients were observed for 3 months after their first positive blood culture result to determine clinical outcome and survival. The genetic lineage of *S. aureus* isolates was determined using a rapid restriction-modification test [2], and vancomycin MICs were determined by Etest (AB Biodisk).

One hundred cases of *S. aureus* bacteremia were studied. Forty-five patients (45%) were treated with vancomycin; 14 (31.1%) of 45 were infected with methicillin-susceptible *S. aureus*, and 31 (68.9%) were infected with methicillin-resistant *S. aureus*. The MICs of vancomycin for these 45 isolates had a range of 0.75–2.00 μg/mL. Sixteen (35.6%) of the 45 patients died $\leq$3 months after diagnosis. An MIC of vancomycin <1.5 μg/mL was associated with a significantly greater risk of death at 3 months (OR, 12; 95% CI, 1.73–83.2; $P = .001$), compared with an MIC of vancomycin $\geq$1.5 μg/mL, as illustrated in figure 1. Clonal complexes (CC22, CC30 equating to EMRSA-15, and EMRSA-16) accounted for most isolates for which the vancomycin MIC was low, but genotype was not significantly associated with clinical outcome.

Soriano et al. [1] reported that higher MICs of vancomycin were associated with poor outcome when vancomycin was used for treatment. In our hospital in the United Kingdom, where EMRSA-15 and -16 are the dominant clones, we have found the reverse. This suggests that there may be geographical variation in the usefulness of the vancomycin MIC as a predictor of outcome.

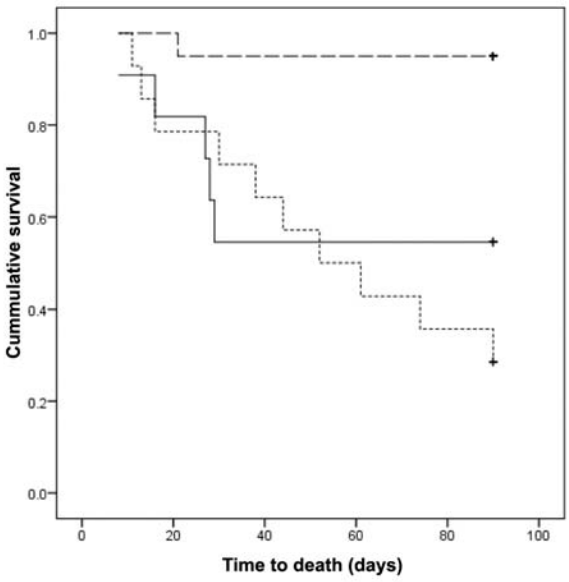

Figure 1. Kaplan-Meier survival curve for 45 patients with *Staphylococcus aureus* bacteremia who were treated with vancomycin and the different MIC ranges of vancomycin (MIC <1 μg/mL *[continuous line],* 11 patients; MIC of 1–1.49 μg/mL *[dotted line],* 14 patients; and MIC of 1.5–2 μg/mL *[dashed line],* 20 patients).

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