laboratory parameters indicative of blood loss would have been more reliable estimates of perioperative blood loss. These indicators should be considered as one of the primary outcome variables. Although transfusions may reflect bleeding, previous data\textsuperscript{15,16} suggest that transfusing blood in cardiac surgery is behavioral rather than a response to blood loss. Re-operation (exploration for bleeding) in coronary bypass surgery cannot be used as a measure of drug-induced coagulopathy unless one completely excludes other causes for bleeding, i.e., heparin-protease activity, large-vessel bleeding, and overall patient coagulation status. Simple changes in preoperative, intraoperative, and postoperative hematocrit and hemoglobin values would help clarify their claims, although even these are confounded by volume changes.

The patients selected in the study by Avorn et al\textsuperscript{1} were those who had received ≥ 3 U packed RBCs within 72 h after undergoing CABG procedure; all other CABG patients were control subjects. The higher comorbidity in the cases (HES group) may act as a confounder leading to higher transfusion rates as a result of lower transfusion thresholds for more severely ill patients.

The editorial accompanying this study recommends a change in practice without convincing evidence. Lack of sufficient data should not be replaced with personal interpretation of study results. This invariably can be misleading to the medical community.

\textbf{Communications to the Editor}

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\textbf{Unresolved Questions With the Use of Linezolid vs Vancomycin forNosocomial Pneumonia}

To the Editor:

We believe several issues must still be addressed in the study by Wunderink et al (November 2003)\textsuperscript{3} published in CHEST. First, it is very unlikely that only uncensored data were used, as mentioned under Fig 2, because not all of the patients died over the follow-up period. Moreover, the p value reported in the curves appears to have been based on a χ² test, as suggested by the statistical analysis section, whereas it should have been based on the log-rank test, which appropriately accounts for length of follow-up and censoring. Furthermore, it is not clear at what time point the crude death rate was calculated for the logistic regression analysis and why regression was used rather than proportional hazards regression, which would account for the length of follow-up and censoring.

Second, the clinical cure analysis in the methicillin-resistant \textit{Staphylococcus aureus} (MRSA) subgroup could be biased due to the different proportion of missing/indeterminate follow-up patients in each group, especially since those who died were counted as missing/indeterminate. A more appropriate analysis would have been based on cumulative incidence rates, which would account for the length of follow-up, censoring, and competing events such as death or toxicity.

Last, linezolid did not show significant beneficial results in the \textit{S aureus} subgroup. This is odd not only because the intention-to-treat (ITT) \textit{S aureus} subgroup was more than twice as large as the ITT MRSA subgroup (larger statistical power to detect a treatment effect), but also because vancomycin is obviously not an optimal therapy for non-MRSA strains, which made up more than half of the comparator group. Consequently, this potentially further decrease in the efficacy seen in the vancomycin arm might have amplified the treatment effect observed in the linezolid arm.
Therefore, the linezolid advantageous effects should be expected to be even more evident in the entire *S. aureus* subpopulation than the one observed in the MRSA subgroup. Nevertheless, a phase III clinical trial comparing linezolid with vancomycin in patients with *S. aureus* pneumonia (both MRSA and non-MRSA) is urgently needed to validate the results of this retrospective subgroup analysis.

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**Reference**