LETTERS TO THE EDITOR

Staphylococcus aureus Bacteremia and Peripheral Vascular Catheters

To the Editor—We were interested in the article by Trinh et al[1] that reported on the risks of peripheral vascular catheters for *Staphylococcus aureus* bacteremia (PVC-SAB). We (E.T.C. and J.R.) have also commented on the importance of PVCs as a cause of *S. aureus* bacteremia (SAB) on the basis of the following data:[2] (1) a national prevalence of PVCs among hospitalized patients of 30.3%; (2) a report that PVCs cause more SABs than are caused by central vascular catheters; (3) reports of PVC care, when audited, being suboptimal; and (4) studies of mortality indicating that PVCs pose a considerable risk.

Trinh et al[1] report that PVCs associated with SABs had a longer mean dwell time than did PVCs that were not associated with SABs ($P < .001$).[1] Their comparison was based on completed PVC episodes for the group of patients who developed SABs. They compared these times with the PVC dwell times obtained from a group of patients who were identified in a point-prevalence study. However, it is clear that this latter group included PVC episodes that could not have been completed, because the PVCs were still in situ. This would have resulted in the dwell times of the comparator group being underestimated, leading to a likely overestimation of the SAB risk associated with the duration of insertion of PVCs.

Although we concur with Trinh et al[1] that patients' risks of developing SABs increase with an increased duration of PVC insertion, we do not believe that their analysis supports this conclusion. We believe that additional work is still needed to highlight the importance of duration of PVC exposure, to reduce patients' risks of developing SABs while receiving healthcare interventions. It would also be useful to understand the rationale for variation in PVC prevalence (30.3% in Scotland[3] and 76% reported by Trinh et al[1]).

In addition, the use of the PVC point-prevalence data, multiplied by bed occupancy data, to serve as a denominator for incidence density may have also underestimated or overestimated incidence if the PVC use varied during the study period.

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Reply to Curran et al

To the Editor—We appreciate the interest in our recent publication regarding peripheral venous catheter–related infection.[1] Curran et al[2] raise a concern that our patients with peripheral venous catheter–related *Staphylococcus aureus* bacteremia were compared with a control group that consisted of patients with peripheral venous catheters who were identified in a point-prevalence survey. They erroneously conclude that all of our patients with peripheral vascular catheter–related *S. aureus* bacteremia had their episodes at the completion of therapy through the catheter. This was not the case. Some of the patients had infections that were detected while the peripheral venous catheter was indwelling, and the catheter would otherwise have been left in place had the event not occurred, whereas others received a diagnosis at the time that the catheter was scheduled to be replaced. We realize that our control group was less than ideal; however, the increased dwell time in the study patients cannot be solely attributed to the detection of infection at the completion of therapy through the peripheral venous catheter that was in place at the time that the infection was suspected. We agree with Curran et al[2] that more data are needed regarding the relationship between peripheral venous catheter dwell time and the risk of infection.

Regarding the number of patients who received a peripheral venous catheter during a hospitalization, other authors have suggested on the basis of the available literature that 30%–80% of patients receive such catheters.[3] Differences in the prevalence of peripheral venous catheter use in different

hospitals and in different countries likely reflect variability in the acuity of illness, the frequency with which other forms of intravascular access are utilized, and other variables. Certainly, understanding the underpinnings of these differences is a fruitful area of future investigation. Last, we agree with Curran et al[2] that our derivation of the denominator for our incidence density calculation was less than ideal. Nevertheless, at the very least, we hope that our calculations allow an approximation of the frequency with which such infections occur, and we hope that our study raises awareness in the healthcare community at large that serious bloodstream infections still arise from peripheral venous catheters and that such infections may fly under the radar of detection, because we have focused so much of our infection control efforts on central venous catheters and other device-related infections.

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Public Reporting of *Clostridium difficile* and Improvements in Diagnostic Tests

To the Editor—Fong and colleagues[1] make some important points about the impact of changing methods for the laboratory diagnosis of *Clostridium difficile* on public reporting schemes. Like that of the authors, our own institution decided to discontinue the use of a toxin enzyme immunoassay (EIA) due to widespread reports of poor sensitivity and specificity. We, too, encountered a significant increase in our laboratory positivity and disease incidence rates.

In line with international guidelines,[2-4] we adopted a 2-step diagnostic approach, screening first with glutamate dehydrogenase and then confirming with a reflex polymerase chain reaction (PCR) test (Cepheid GeneXpert). For the 4 months before the change, we had a laboratory positivity rate of 2.49% (53 of 2,146 specimens tested); this more than doubled to 5.55% (98 of 1,767 specimens tested) in the 4 months after the change. This also had a dramatic affect on our rate of *C. difficile* infection (CDI), which increased from 3.6 per 10,000 (0.036%) to 7.1 per 10,000 (0.071%) occupied bed–days. We have demonstrated that this change was not due to change in antimicrobial prescribing, change in patient population or increasing nosocomial transmission, or change in environmental contamination rates.[5]

PCR and other methods will detect colonized patients as well as infected ones, and although both groups pose a potential reservoir for transmission, they should be treated differently for clinical management as well as epidemiological data reporting. The rate of patient colonization is not well understood, with a wide range of figures quoted in the literature. It is important, therefore, to corroborate the laboratory test with clinical history and examination; we have found that approximately 10% of patients with a positive PCR test are probably colonized rather than truly infected.

Figure 1 shows the rate of CDI experienced in our organization from September 2009 to July 2011. (The improved diagnostic algorithm was introduced in September 2010.) It appears that 10 months after the introduction of the new testing method, rates of CDI have stabilized and are beginning to decrease. This may be the result of improved case ascertainment (of both infected and colonized patients), which, when appropriately treated and/or isolated, could be expected to result in decreased ongoing transmission.

In England, the Department of Health introduced a mandatory reporting scheme for *C. difficile* in 2004, with a target for all acute National Health Service (NHS) trusts to reduce CDI by 30% compared to a base level in 2007–2008. This was achieved 2 years ahead of schedule in 2009, and a new *C. difficile* objective was applied to NHS organizations from April 2011. This is based on a sliding scale, requiring the worst performers to make the greatest improvements, with a maximum target CDI rate of 4.5 per 10,000 occupied bed–days. There are severe financial penalties for trusts failing to meet this target, amounting to 0.1% of the contract value for each percentage above the baseline, capped at a maximum of 2%.

For a 1,100-bed organization with a target of no more than 155 cases of *C. difficile* per year, such as ours, exceeding the target by 20 cases (13%) could amount to a fine of 1.4% of contract value (this equates to around £9 million, with each additional case costing more than £400,000). These contract terms are part of the standard mandatory NHS contract, and