Surgical Site Infections and Postoperative Factors

To the Editor—I read with interest the article by Farrin Manian regarding the role of postoperative factors in surgical site infections (SSIs) [1]. I agree with Dr Manian that, although the majority of SSIs are almost certainly determined during surgery, there are definitely factors after the patient leaves the operating room that can lead to infection. Dr Manian cites several references regarding the risk of infection when surgical drains are left after an operation. It would be astounding if this were not the case. The drain crosses the epidermal barrier to microorganisms and provides a ready route to the surgical site just as a central line directs bacteria into the bloodstream. Although not well studied, the same principles that reduce the risk of central line–associated bloodstream infection (CLABSI) should reduce the risk of drain-associated SSI, including not using drains whenever possible, removing them as soon as possible, and using strict sterile management and dressings, perhaps including chlorhexidine gluconate–impregnated dressings. More studies, such as the one by Degnim et al [2], are definitely needed. In my own practice I have seen a dramatic decline in the use of drains for many surgical procedures compared with their use in the 1970s, but we still have a long way to go in this area.

Dr Manian also cites the association between bacteremia and SSI. This connection was demonstrated in animal models many years ago, showing conclusively that in that setting the surgical incision was at risk for infection from bacteremia [3–5]. Of the studies cited by Manian, that by Le Guillou et al [6] is the most convincing; in their study, 9% of the SSIs observed followed a central line–associated bacteremia with the same organism. This, combined with the strong animal evidence, suggests that the association is real, and provides yet another reason to redouble our efforts to prevent CLABSI. Another article that suggests this association is an interesting report by Ehrenkranz and Pfaff [7] from 1991, when they observed that a cardiac surgical practice that operated in 2 hospitals had a significantly higher mediastinitis rate in one hospital than in the other. Investigation found no difference in processes in the operating rooms, but quite different infection control practices in the intensive care units (ICUs) of the 2 hospitals. When the poorly performing ICU was corrected, the infection rate decreased without any change in other patient care practices.

Manian cites the association of skin closure techniques and anticoagulation and hematomas with SSI. Interestingly, there are also animal models that show conclusively that recent incisions are susceptible to infection from the outside after the conclusion of surgery for several days until the incisions are sealed [8, 9]. In addition, work by Olsen et al [10] that demonstrates a higher rate of SSI after spinal surgery in patients with postoperative fecal incontinence also suggests infection from external sources after leaving the operating room. In my own practice, when I perform a clean operation, especially one with placement of a prosthetic device, I seal the incision and place an impermeable sterile dressing on in the operating room, which I leave on for a minimum of 5 days. If wound seepage requires dressing change, this is done in a sterile manner as if we were back in the operating room, and the area is repped and redressed in the same manner and the dressing left until the wound is dry and sealed. Unfortunately, most surgeons seem to derive pleasure from taking the dressing off within 24 hours to admire the incision, and there are no conclusive data in humans to support either my practice or the early removal. However, leaving the dressing on has no potential adverse consequences that I can think of.

An area not mentioned by Manian is the issue of perioperative glucose control. A growing body of literature ties perioperative hyperglycemia to a dramatically increased risk for SSI both in diabetic and nondiabetic patients, and most publications in this field demonstrate an increased risk associated with hyperglycemia for at least 2 days after the operation as well as, of course, for hyperglycemia in the operating room [11–20]. However, at this time, the precise level of perioperative glucose control that should be achieved and the optimal method for doing that remain controversial.

Finally, I would like to caution readers regarding the interpretation of the article by Murphy et al [21], cited by Manian as possible acquisition of methicillin-resistant Staphylococcus aureus (MRSA) infection in the postoperative period. In that article, patients scheduled for orthopedic surgery were screened for MRSA and, if it was found, decolonized. They were then scheduled for surgery after decolonization was confirmed, and there was a higher rate of MRSA infection in these “decolonized” patients. However, the interval between decolonization and surgery was as long as 3 months, they were not screened again, and they received a cephalosporin for prophylaxis. We know that many patients colonized by S. aureus tend to remain colonized or become recolonized after decolonization, and thus the most likely explanation for these patients is that they went into surgery colonized with MRSA and received inappropriate prophylaxis.

I thank Dr Manian for bringing up this topic, and I hope that the readers will take this into account as they work in their own institutions to try to minimize the morbidity of SSI.

Note

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