TRULY GETTING TO ZERO

Theoretical Considerations for Elimination of CLABSIs

By Steve Bierman, MD

If you have ever watched a fibrin sheath progress to a thrombus on the surface of a catheter — a process that initiates instantly upon entry into the bloodstream and proceeds quickly, often in just minutes — you will understand why the presence of contaminating bacteria on the surface of a catheter is something to be rigorously avoided. The rapidly forming fibrin sheath encases such surface bacteria, both shielding them and facilitating biofilm formation.

Understanding this sequence — bacterial surface contamination, fibrin shielding and biofilm formation — offers an important clue as to how CLABSIs might really be reduced to zero. Not the median rate of zero that we hear so often discussed, nor the “zero” we often hear touted when institutions pull every line at the first sign of an unexplained fever. Rather, I believe, a real CLABSI rate of zero is achievable for short, non-tunneled central venous catheters by instituting two simple measures.

Before we can fully understand those measures, we must first explore certain elements of catheter-associated infection that have long eluded our understanding — certain unexplained mysteries of our science.

The Enigmas

Consider these long-established, but unexplained, facts:

1. Short, non-tunneled (non-medicated) CVCs and radial artery lines often have approximately the same bloodstream infection rates when left to dwell for the same duration. These catheters are of different lengths, often different materials, and reside in distinctly disparate microbial environments. Why do they so often infect at the same rate?

2. Short non-tunneled CVC-associated bloodstream infections tend to occur early and, generally, to arise from bacterial biofilm on the external surface of the catheter. PICCs, in contradistinction to CVCs, tend to infect the bloodstream later owing largely to biofilm formation on the internal surface of the catheter. Both kinds of catheters have tips in the central circulation; they are often made of the same material; often managed by the same personnel; and, used to administer the same medications. Though it is a fact that skin flora generally cause infections in short CVCs, why aren’t they also the predominant cause of infections in PICCs?

3. Midline data, collected before the advent of the new power-injectable midlines, indicates a bloodstream infection rate associated with these catheters considerably lower than that of PICCs. Yet, these MST-inserted midline catheters have the same insertion site and the same catheter material as PICCs. Why don’t they have similar infection rates when compared with PICCs?

Until now, there has been no clear and coherent explanation to make sense of these enigmas. Maki and colleagues, brilliantly indicted cutaneous bacterial flora as the source of CVC-related bloodstream infections. They subsequently speculated that the diminished concentration of “cutaneous flora” at the usual site of midline insertion, as compared with the more concentrated cutaneous flora on the neck and chest, might explain the low BSI rate of midlines. But if concentration of bacteria were the only determinant, then midlines and PICCs should have the same infection rate and short CVCs, especially those placed in the neck, should have a remarkably higher rate of infection than radial artery catheters. Neither of these conditions holds true.

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The Solution
The solution becomes clear upon reading what I consider one of the most elegant studies in vascular access, by Livesley and his colleagues, published in 1998 in the European Journal of Clinical and Microbiological Infectious Diseases. What Livesley and his group did was simple: They randomly divided 60 open-heart patients into two groups of 30. Both groups received short CVC lines following chlorhexidine skin antisepsis. However, in Group 1 the lines were placed percutaneously using the Seldinger technique. In Group 2, the lines were placed through a Swan sheath introducer, that is, through a sterile conduit. Following placement, the patients underwent thoracotomy, their hearts were opened, and the distal portion of their CVCs was cultured (along with the glove used to obtain the culture).

Five of the Group 1 percutaneously placed catheters had skin bacteria “impacted upon the external catheter surface of the catheter.” In Group 2, whose catheters were placed into the bloodstream via a sterile conduit, only one catheter had skin bacteria on its external surface — an 80 percent reduction in bacterial surface contamination. In other words, introducing a short CVC over a wire and through the skin using the Seldinger technique resulted in 16.6 percent of catheters being “impacted with skin bacteria” as they entered the bloodstream. No wonder we commonly see CVC catheter-related bloodstream rates of 2 percent to 3 percent. As a side note: Livesley also demonstrated a 50 percent guidewire contamination rate using the standard Seldinger technique. This, as Levin and colleagues have shown, often leads to false-positive blood cultures due to intraluminal catheter colonization from these contaminated wires.

Imagine what must then happen on the surface of that “impacted” catheter: The bacteria (e.g., staphylococcus, streptococcus, etc.) are present upon entry into the bloodstream. Within minutes a dense fibrin sheath shields them from attack, they attach, aggregate, proliferate and ultimately—nourished by blood and abetted by chemical co-conspirators from platelets and other bacteria — develop into planktonic colonies seeding the bloodstream with billions of their kith and kin. It is not instantaneous; rather, it takes time—four, five, six days—depending on the size of the initial inoculum. The sooner the catheter is removed, the less time the fortress colony has to reach maturity. The longer it remains, the more likely its dissemination.

It is true, as many have written, that bacteria can enter the bloodstream on the catheter’s external surface by migrating from skin to intracutaneous tract to intravascular surface. But though we casually refer to bacteria as “bugs,” they do not locomote as bugs do; rather, they must grow and expand their reach by means of cell division—a process that takes time and often must traverse prodigious distances (from a bacterium’s point of view).

Both routes of entry will result in a bloodstream infection caused by cutaneous flora. And given sufficient time, both routes can occur. But the mode of access suggested by Livesley’s research offers the bacteria a head-start. They arrive in the bloodstream simultaneously with the catheter. And since time mediates the pathogenesis of catheter-related bloodstream infections, I believe that the Livesley-route, so to speak, predominates. Moreover, as we shall see, there
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is highly suggestive evidence that supports this notion.

Consider: All catheters placed percutaneously infect the bloodstream predominantly from biofilm on their external surfaces. Short non-tunneled CVCs, arterial lines and even PIVs. Infection rates with these catheters correlate to their average dwell times: CVCs and arterial lines dwell an average of four to seven days and have similar rates, whereas PIVs dwell an average of 44 to 99 hours and have a lower BSI rate. In contrast, catheters that are placed through sheath introducers—i.e., PICCs and the older midlines—infect the bloodstream predominantly from biofilm that develops (from mishandling) on the intraluminal surface of the catheter. This process takes more time since bacteria are introduced at some time after insertion, and then must attach and grow. It is as if their starting line were pushed back when compared with the percutaneously impacted bacteria. Thus, as expected, these intraluminal infections in sheath-placed catheters take a longer time to result in bloodstream infection, and develop at a greater rate in the catheters that dwell the longest. This explains why PICCs (which historically have dwelled for longer times than most midlines) infect at a higher rate than the old MST-placed midlines, despite the fact they are placed in the same location using the same sheath-introducer technique. Also explained, is why the early midlines had lower rate of infection than PICCs namely, they were placed through a sterile conduit and removed relatively quickly (i.e., usually in less than two weeks). Not only does this schema solve all the aforementioned enigmas, but it also points to the two measures I alluded to that, in combination, should make ZERO CLABSI a truly attainable goal.

Getting to True-Zero CLABSI

By now, these measures are probably obvious: First, the Livesley study cogently suggests that short, non-tunneled CVCs should no longer be placed by means of a technique proven to result in a 16.6 percent contamination rate; rather these CVCs should be placed via sterile, removable conduits—that is, peel-away sheath introducers. (These sheaths must be removable because, being placed percutaneously, their external surfaces are contaminated.) The logic of our new understandings strongly dictates this insertion method for CVCs, provided such introducer sheaths do not entail other risks and unintended consequences that somehow heighten risk. I will return to this shortly.

The second measure is, of course, removal of the CVCs as early as possible so as to thwart biofilm maturation and bacterial dissemination. With our present understandings, one might even argue that owing to the potentially lethal risk of CLABSI all CVCs should be removed and/or replaced (when feasible) on or before the seventh day of dwell-time. Some exciting new evidence suggests this policy, coupled with the use of power-injectable midlines for
It is estimated that from 1 in 47 to 1 in 3,000 air emboli occur each year as a result of CVC placement. The estimated cost per incident is $71,636; the estimated mortality is as high as 30%. To avoid air embolism, a peelable sheath introducer with a competent valve should be used for CVC placement. Such a valve should be capable of withstanding a negative pressure of 160-170 cm H2O. While several valved peelable sheaths are presently on the market, most are only competent to restrict blood flow, but not to restrict the ingress of air. Presently, there are three valved peelable sheaths on market. Only one is “air-competent,” meaning capable of preventing air embolism under physiologic conditions. There are two other “valved” peelable sheaths designed to prevent excessive blood loss, but they both allow significant (and dangerous) air flow to occur at physiologic negative pressures. Hopefully, as our understanding of bacterial contamination of CVC catheters extends, other air-competent, valved, peelable sheath introducers will become available. If so, Livesley’s work may well prove to be among the most important and life-saving contributions to the quest for zero.

**Summary**

When non-cuffed CVCs are inserted percutaneously, using the Seldinger technique, 16.6 percent are contaminated with skin flora when they enter the bloodstream. A fibrin sheath rapidly encases and protects these bacteria, which over time form mature biofilm colonies and disseminate bacteria into the bloodstream. Using a valved, peel-away sheath introducer in conjunction with the CVC Bundle should reduce such contamination by 80 percent, based on Livesley’s study, while preventing air embolism. Coupled with early...
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References: