Catheter Material Inhibits Bacterial Attachment

By Michelle DeVries, MPH, CIC, VA-BC

A newly published study in *Medical Devices: Evidence & Research* by Dr. Rahul Pathak reported that a new catheter material prevents bacterial attachment and biofilm formation.[1] Pathak's team demonstrated that if a bacterium does attach to this material, it is 1.5 times more likely to die as compared with a polyurethane catheter.[1] These discoveries, especially if proven in a randomized clinical trial, could have dramatic implications for vascular access and infection control. Other recent studies looking at polyurethane catheters have raised the question of potential "bloom" from surface additives with the potential to facilitate bacterial attachment. [2-3] Certainly, more work (reproducibility) is needed to make this data more robust, but as a starting point it raises some questions.

Since I have studied catheters made of this same material reviewed by Dr. Pathak in my own institution, I was eager to speak with him about this work particularly since it might explain, at least in part, the complete absence of infections we have reported across hundreds of devices and more than two years of use.

MD: Let me begin by asking where your work is published and why it's so important?

RP: Our work appears in Medical Devices: Evidence & Research, and it is open access—so anyone can read the study without having to pay. As to why this work is important: I think this readership is probably more aware than most of the continuing scourge of catheter-related bloodstream infections (CRBSIs) in hospitals throughout the world. While much good work has been done in recent years to reduce CRBSIs, this complication is still far too common. Now that we understand how polyurethane catheters "facilitate bacterial attachment," it seems especially important to look for new, safer ways to get around this problem.

MD: And you believe your work offers an explanation and a new way to avoid CRBSIs?

RP: It's a start. Let's step back for a second. My team decided to explore bacterial attachment and biofilm formation on ChronoFlex C® with BioGUARD™ after my original study on midline catheters made of this material was published.[4] In that study, conducted in a ventilator ICU, we demonstrated a 37% decrease in central line days and a 100% decrease in CLABSI due to use of the POWERWAND midline, which is made of the study material. Beyond those findings, what struck me was that even in bacteremic patients, these catheters did not become infected. Why? How?

MD: I've witnessed the lack of midline infections in my own facility with POWERWAND catheters. Have you now figured out the' why' and the 'how'?

RP: For the most part, yes. Here is what we did. First, we devised a simple in vitro percutaneous insertion model that simulates skin. We know from Livesley that 1 out of every 6 catheters placed through the skin picks up Staph. aureus during insertion.[5] This happens despite chlorhexidine anti-sepsis. But since we wanted to explore the worst case, our model exposed every catheter to a living bacterial colony. Then, we incubated the exposed ChronoFlex C and polyurethane (control) catheters, cut their tips and—using the newest fluorescent microscopy techniques—counted bacteria, both living and dead.

MD: This technique, is it considered better than, say, the semi-quantitative roll-plate method?

RP: Immensely better. The roll-plate technique is at best a rough approximation. To perform it, biofilm is disrupted and lost. Moreover, it cannot discern bacteria that attach but subsequently die.

MD: So, what did you find?

RP: We found that bacteria were 5.4 times more likely to attach and grow to the polyurethane catheter than to the ChronoFlex C with BioGUARD catheter ($p = 0.0020$). Moreover, those rare bacteria that did attach to the ChronoFlex had a 1.5 times greater chance of dying.

MD: Why might that be?

RP: Two reasons. First, the manufacturer removes surface additives from the POWERWAND catheter. Second, the surface of the POWERWAND has what is called micro-patterning. There is very little surface for even one or two bacteria to attach to. Bacteria need to form communities to thrive and form a biofilm. Absent a community of adherent neighbors, they die.

MD: Your work is in vitro. What really intrigued me is that it bears out my two-year in vivo experience with POWERWAND midline catheters. But mine is a prospective observational study. Do you plan to extend your work to include a randomized controlled trial (RCT)?

RP: Provided we can secure adequate funding, we would very much like to conduct a RCT comparing standard polyurethane catheters to ChronoFlex C with BioGUARD. In the meantime, there are six published studies (and two more studies pending publication, including yours) that total more than 35,000 POWERWAND catheter-days of use. In every study, although not a primary endpoint, zero bloodstream infections were reported. We recognize that is not definitive proof; but it is strongly suggestive, especially as no other midlines have performed nearly so well.

MD: Right now, my experience has been with the POWERWAND midlines but I have seen similarly strong data with the 6 cm catheter from Access Scientific. Am I correct that the same material is used for those shorter catheters as well? And nothing would prevent it from being used across an even broader range of devices?

RP: Absolutely. The same materials are used in their 6, 8 and 10 cm catheters as well as their arterial lines and CVC. The possibilities with this new information are huge now that we can explain what clinicians across the country have been reporting for years.

MD: Well, Dr. Pathak, I will certainly look forward to your continuing work. Thank you for your important contribution.

RP: And thank you, Chellie, for yours.

Disclosures: Michelle DeVries is a member of the speaker's bureaus for Access Scientific, Becton Dickinson, Eloquest and Ethicon. ICT

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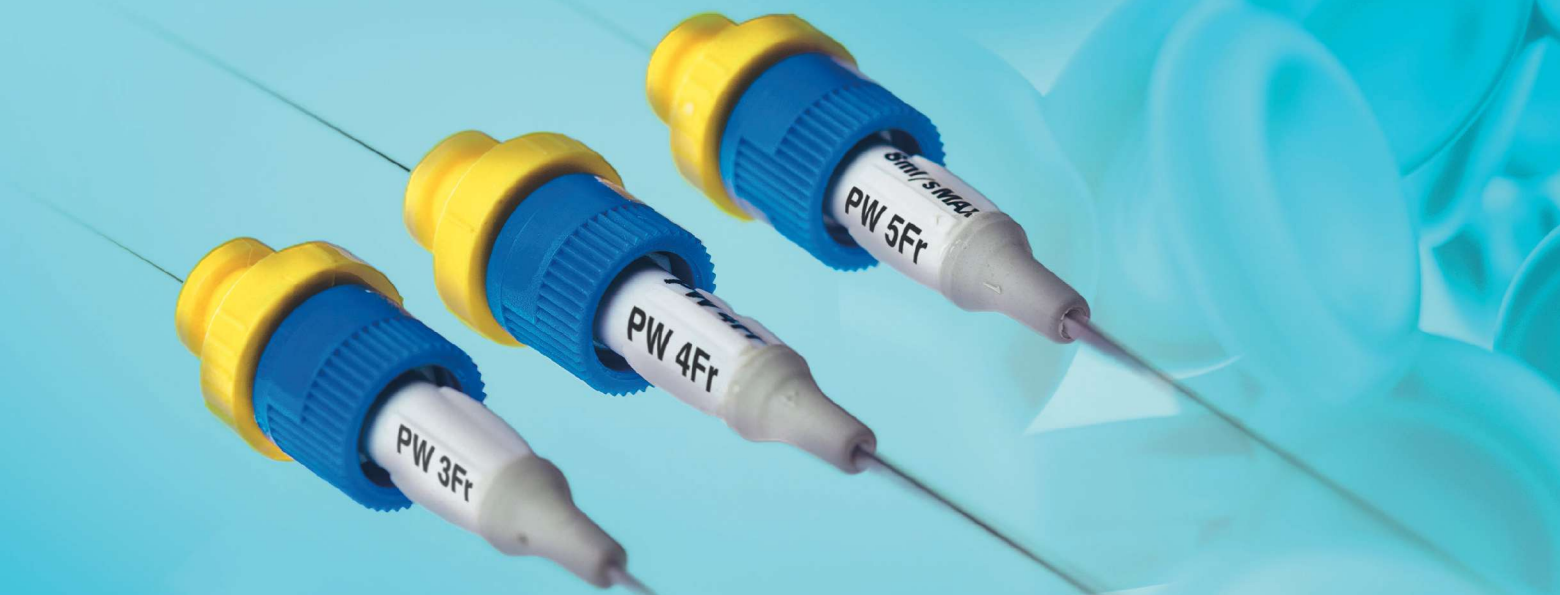

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2. The POWERWAND is proven in vivo to be thromboresistant with respect to both thrombus on the surface of the catheter and thrombus on the wall of the vein. Based on canine jugular vein thromboresistance study, correlations to clinical applications has not been ascertained.

†Based on laboratory test results which may not be indicative of clinical results. Data on file and refer to publication[3]. Preclinical in-vitro evaluations do not necessarily predict clinical performance with respect to catheter-related bloodstream infection.

3. Pathak R, Bierman S, d'Arnaud P. Inhibition of bacterial attachment and biofilm formation by a novel intravenous catheter material using an in vitro percutaneous catheter insertion model. Medical Devices: Evidence and Research. 2018;11:1-6. https://doi.org/10.2147/MDER.S183409.

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LC-0576 Rev. B