Catheter-Associated Urinary Tract Infections — Turning the Tide

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In the wake of an otherwise successful first phase of the National Action Plan to Prevent Health Care–Associated Infections, the reduction in catheter-associated urinary tract infections (UTIs) has been elusive.[1] Increasing rates of infection by 2012 signaled an inability to contain this common infection, even if some of the rise was attributable to broader definitions of catheter-associated UTI instituted earlier that year. A study of the On the CUSP: Stop CAUTI program, reported by Saint et al.[2] in this issue of the *Journal*, showed a significant reduction in catheter-associated UTIs in inpatient units that were not intensive care units (non-ICUs), despite the change in the definition. The study highlights the very real potential for reductions nationally.

Catheter-associated UTI is remarkable in that the technical protocols to prevent it have been known for a long time — sterile catheter insertion, maintenance of a closed drainage system, prevention of backflow, aseptic technique for collecting urine for culture, and minimal duration of catheter placement because of the propagation of low-level bacterial colony counts over time in a catheterized system.[3,4] These processes represent continually taught best practices.

Thus, we might assume that behavioral and decision-making aspects remain the dominant barriers to the reduction of catheter-associated UTIs. Nearly all campaigns rely on leadership, persuasion, and teamwork to be successful, but the On the CUSP: Stop CAUTI program's overt instruction to build a multidisciplinary team, focus on camaraderie and communication, and attend to failure points may be requisites to success. This approach may be critical for improving decision-making components of catheter-associated UTI prevention, such as indications for catheter placement and removal, avoidance of reflexive culturing of urine in response to fever, and advocacy for alternative strict toileting options (e.g., urinals for women or for men, external catheters for women or for men, measurement of diaper weight, intermittent catheterization, and measurement of daily weight).

The broad scope and scale of the study by Saint et al. also raises important questions. Why did ICUs not have the reductions in catheter-associated UTIs that non-ICUs had? There may be several explanations. Catheter-associated UTIs require a catheter plus a UTI. On the catheter front, patients are often sedated and unable to report on the presence of a catheter. In addition, the critical condition of patients in the ICU may make a nurse-driven program for placement and removal of urinary catheters less acceptable to physicians in the ICU. Catheters are also likely to be required for longer durations in ICU patients, and day-to-day ease of care with a catheter may outweigh concern about catheter-associated UTI prevention unless awareness is maintained. Regarding the diagnosis of UTIs, if reflexive culturing of urine in response to fever is not addressed, any reason for fever in patients in the ICU may result in unnecessary urine cultures that are positive for asymptomatic bacteriuria that could be treated with catheter removal rather than antibiotic therapy.

The multiplicity of reasons for fever in patients in the ICU is another reason why catheter-associated UTI rates in ICUs may not have declined. Changes to the National Healthcare Safety Network (NHSN) definition of catheter-associated UTI accounted for substantial increases in rates of catheter-associated UTI beginning in 2012 and substantial reductions in catheter-associated UTI rates in 2015.[5] In March 2012, NHSN changed the fever requirement from a subjective definition requiring attribution to catheter-associated UTI to an objective definition of the presence of fever regardless of its clinical source. Thus, it is likely that the NHSN rates reported here by both ICUs and non-ICUs underestimate the program's influence. In contrast, reverse causation is noted for the analysis of later cohorts from the On the CUSP: Stop CAUTI program, since they will benefit from the 2015 definition change that excludes yeast and restricts catheter-associated UTIs to cases with cultures with higher colony counts ($\geq 10^5$ colony-forming units [CFU] per milliliter vs. the current count of $\geq 10^3$ CFU per milliliter).

Ideally, repeat analyses, even in a random

subset of hospitals, should be performed with a uniform definition. In addition, subset analyses that stratify catheter-associated UTI rates according to whether decreases in catheter use were achieved should be performed. Several studies have shown that successful efforts to reduce catheter use cause the remaining population of catheterized patients to consist of higher-risk patients; as a result, rates of catheter-associated UTI can increase.[6-8] For this reason, if a substantial number of non-ICUs showed both reduction in catheter use and improvement in rates of catheter-associated UTI, it would be greater evidence of the benefit of the On the CUSP: Stop CAUTI approach.

Finally, what remains to be seen is whether health care providers can maintain activities resulting from innumerable best-practice campaigns. Does the focus on one campaign signal a trade-off of attention to other equally important processes? The sheer number of documentation and medical-practice requirements in place today requires a breadth of technological advances, facility design, human-factors engineering, and implementation science to be able to maximally prevent human errors and omissions. Successful enduring methods to attend to all best-practice measures are needed if we are to continue to improve patient safety.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

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The Sialylation Pathway and Coronary Artery Disease

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Plasma levels of non–high-density (non-HDL) cholesterol are causally linked to the risk of coronary artery disease. Non-HDL cholesterol levels are a better predictor of cardiovascular risk than levels of low-density lipoprotein (LDL) cholesterol alone, because non-HDL cholesterol comprises all known cholesterol-containing lipoproteins that can cause atherosclerosis, including LDL, very-low-density lipoprotein, intermediate-density lipoprotein, chylomicrons and their remnants, and lipoprotein(a).

Nioi et al. now report in the *Journal* important genetic evidence suggesting a causal association between *ASGR1*, a gene encoding the major subunit of the asialoglycoprotein receptor, and cardiovascular disease.[1] Nioi and colleagues sequenced the genomes of 2636 Icelanders and identified variants that were imputed into the genomes of approximately 398,000 additional Icelanders. They tested for an association between the imputed variants and non-HDL cholesterol levels in 119,146 participants and identified a rare 12-base-pair deletion (del12) in intron 4 of *ASGR1*. In the Icelandic population, del12 was associated with an average reduction of 13.6 mg per deciliter (0.35 mmol per liter) in plasma levels of non-HDL cholesterol as compared with the average level in the general population (relative reduction, 9%). This reduction included decreases of 9.5 mg per deciliter (0.25 mmol per liter) in LDL choles-