Editorial

Unachievable zeros

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We, as healthcare providers and surgeons, stand humbled before a formidable foe. For as long as surgeons have been performing operations, infections have arisen from our surgically created wounds. We have attacked these pathogens with antimicrobial drugs, sterilizing sprays, and process measures. Yet we struggle to achieve victory. As the bacteria and fungus responsible for these infections are exposed to broad-spectrum antimicrobials they develop resistance mechanisms faster than we can develop novel antibiotics. Process improvement strategies aimed at reducing these infections, in and of themselves, are inadequate; they do not fully address the dynamic interplay between host and pathogen. Unless we establish reasonable goals for our interactions with the microbial world we will be plagued by failure as we attempt to chase externally-imposed endpoints.

Sternal wound infections may be dramatic and lethal, and consequently have historically occupied much of the literature surrounding infection after cardiac surgery. Less is published about infection after so-called "secondary" procedures such as a groin access or saphenous vein graft (SVG) harvesting. To address this deficit, Gurlack and colleagues present a multi-institutional prospective cohort study using the Cardiothoracic Surgical Trials Network (CTSN) to examine predictors associated with development of secondary surgical site infection (SSI) after CABG with SVG conduit (1). Over a 9-month time period in 2010, they identified 2,174 patients of whom 65 (3%) developed a secondary SSI. This value is similar to the 3.5% of patients identified with superficial or deep sternal site infections from the same CTSN cohort (2), underscoring the frequency of these "secondary" infections.

But Gurlack *et al.*'s most consequential observation was the delayed presentation of these SSIs and the markedly high readmission rate among patients with secondary SSI. Median time to diagnosis of a secondary surgical site SSI was reported as 16 days [interquartile range (IQR), 11–29 days] (1). Most (86%) of the infections were diagnosed after discharge from the index hospitalization, 82% of these were identified within 30 days of discharge, a problem documented in prior studies examining SSI after cardiac surgery (1,3). Concerningly, the CTSN patients who developed a secondary SSI had an almost two-fold increased frequency of being readmitted within 65 days (1). Nearly one quarter were admitted greater than 30 days after their index operation (1). These delayed infections after clean surgery are particularly troubling.

The US healthcare system is transitioning as surgical outcomes of hospitals and surgeons are increasingly compared to, and tied to, re-imbursement. SSI and readmission are two commonly cited metrics of quality (4,5). Marrying these two outcome measures, and hitching them to reimbursement, has had several effects: hospital administrators are driven to push hospital-acquired infection rates toward zero, and similarly strive to minimize readmissions. Gurlack *et al.* correctly point out that "certain 'never' events [infections] will never have a zero incidence" (1). Any external endpoint aimed to achieve a zero incidence of infection, explicitly or otherwise, is missing the bigger picture. Instead of elimination, we should direct resources toward early detection of changes in host homeostasis signaling pre-clinical infection, with the goal of modulating developing infection through targeted anti-microbial therapy before it can progress and become clinically

detrimental. Similarly, if propensity for infection can be identified in patients nearing discharge, focused, aggressive, post-discharge monitoring can be initiated.

While this degree of pre-clinical risk factor assessment and prognostic prediction is not currently available for SSIs, evidence is building to support imminent emergence. For example, in critically ill patients with sepsis, a notoriously diverse and difficult-to-study cohort of patients, omics-based expression analysis has been shown to out-predict mortality when compared to clinical models and biochemical markers such as procalcitonin (6-8). Combining such sensitive preclinical omics-based testing with real-time, digital, patient-centered follow-up for SSIs is particularly promising (9,10). To address the challenging delayed SSIs described by Gurlack *et al.*, such combinatorial diagnostic methodology will be critical. But funding for the development, testing, and post-market surveillance of these novel diagnostic tests for SSI is crucial to encourage industry growth.

SSIs are unlikely to go away. As surgeons, we need to grow comfortable living in a human world intricately interwoven with the microbial. While we must do our best to minimize SSIs through appropriate antibiotic stewardship and process measures, we need to understand that, at best, our goals should be to modulate interactions between pathogenic bacteria and our patients, that elimination is not realistic. In the future infections will be identified preceding clinical deterioration, and before discharge. But to accomplish this goal, payment should be tied to early identification and treatment, rather than "unachievable zeros".

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Footnote

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