



The Value of Extending Nasal Decolonization into the Postoperative Period for SSI Prevention

Tarina Garcia-Concheso, MSc, CPHQ*

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Introduction

Surgical site infections (SSIs) remain one of the most common and costly complications of modern surgery, occurring in approximately 1–3% of procedures in the United States.¹ SSIs lead to substantial morbidity and mortality, longer hospital stays, and increased readmissions making their prevention a central priority for healthcare systems.^{2,3}

Despite decades of implementation of standardized prevention bundles, including perioperative antibiotics and intraoperative sterile technique, progress in reducing SSI rates has plateaued.⁴ There is a pressing need to consider approaches beyond greater environmental sterility and broader perioperative antibiotics.⁵ Available evidence indicates that even perfect adherence to these current standards would yield only modest additional reductions in SSI rates. This trend suggests that current strategies do not fully address the continuum of microbial and clinical risks encountered throughout the perioperative period.^{3,4}

*Vice President Clinical Affairs, Global Life Technologies Corp.

Historically, the pathogenesis of SSIs has been attributed primarily to intraoperative contamination, directing preventive efforts toward minimizing exogenous sources such as operating room environment, instrument sterility, and surgical attire. While maintaining current sterile standards remains essential, simply layering on additional operating-room sterility requirements is unlikely to produce predictable gains in SSI reductions.^{5,6} Instead, data from microbiologic and genomic studies suggest that the patient's own nasal and skin microbiota are the predominant contributors to SSI. Across multiple cohorts, 70 to 95% of SSI pathogens match strains present in the patient's preoperative flora, providing further evidence that endogenous reservoirs, particularly the anterior nares, are major drivers of postoperative infection risk.^{4,7,8}

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In addition to preoperative factors, postoperative factors contribute substantially to infection risk. Daily wound manipulation, rapid recolonization of the nares and skin, and acquisition of new hospital-associated organisms all meaningfully increase susceptibility to SSI in the days following surgery.^{3,9}

Together, these findings are consistent with gaps in current prevention practices. Preoperative decolonization can reduce bacterial burden at the time of surgery; however, sustained protection during the postoperative period is not assured. Antibiotic-based regimens depend on full adherence and organism susceptibility, while antiseptic-based

approaches require continued application to maintain effect.¹⁰⁻¹²

Pathogenesis of SSI: The Role of Nasal and Skin Microbiota

The anterior nares are a key ecological niche for *Staphylococcus aureus* and an important endogenous reservoir for the development of surgical site infections. The nasal vestibule is particularly important as it harbors the highest and most stable bacterial burden of *S. aureus* within the body and is the most frequent carriage site implicated in transmission. Persistent or high-density colonization is strongly associated with an increased risk of postoperative infection.^{7,13}

Evidence indicates that the majority of SSI pathogens originate from the patient's own skin and nasal microbiome, with estimates suggesting that approximately 70-95% of SSIs arise from endogenous sources. Consistent with this, in a prospective spine surgery cohort, 86% of SSIs were linked to strains detected preoperatively, and more than half of SSI isolates were resistant to the prophylactic antibiotic administered.⁵ These findings illustrate that endogenous source and prophylaxis failure can coexist within the same case.¹⁴ Genomic investigations across surgical populations similarly demonstrate that a large majority of *Staphylococcus aureus* SSI isolates are closely related to endogenous strains.⁷

Nasal carriage also functions as a continuous source of contamination for the surrounding skin. Organisms are transferred frequently from the nares to the hands, face, and surgical field, creating repeated opportunities for pathogen inoculation during dressing changes, wound manipulation, and routine patient care.⁹ Even when skin antisepsis is performed according to guideline standards, recolonization of the skin from the nares can occur rapidly, sometimes within hours, re-establishing the reservoir of organisms capable of accessing the surgical wound.¹⁵ The postoperative period introduces additional

microbiologic risk, not just around incision care. Patients may acquire new nasal colonization during hospitalization through environmental exposure and cross-transmission, even when preoperative decolonization has successfully reduced or eliminated carriage.^{3,4,9} Evidence supports that methicillin-resistant *Staph aureus* (MRSA) can be acquired in the postoperative period, with newly acquired strains contributing to infectious complications.³ This dynamic nature of colonization highlights a key vulnerability: without continued suppression, the nares remain an active reservoir capable of seeding pathogens during the entire wound-healing period.

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Taken together, these data position nasal colonization of the anterior nares as a persistent, modifiable risk factor for SSI across the perioperative period—ongoing carriage, reacquisition, and contact-mediated redistribution can keep reintroducing pathogens during the wound-healing window.

Postoperative Factors as Overlooked Contributors to SSI Risk

Although SSI prevention efforts have traditionally focused on the preoperative and intraoperative periods, an increasing body of evidence suggests that postoperative factors contribute substantially to infection risk. This is not just theoretical. In adults undergoing joint arthroplasty or spine surgery, *S. aureus* nasal colonization within 24 hours after surgery was linked to about a threefold increase in subsequent SSI risk.¹⁰ During this time, the wound remains vulnerable while patients undergo dynamic

shifts in colonization, environmental exposure, and host defenses.

Continued Vulnerability of the Surgical Wound

After closure, surgical wounds undergo a prolonged healing process during which tissues are not yet fully sealed.³ Pathogens may access fresh surgical sites postoperatively through hematogenous spread, surgical drains, or slowly healing wounds, and many SSIs reflect postoperative exposures rather than events confined to the operating room.³ One proposed explanation is the Trojan Horse hypothesis, in which pathogens remote from the incision are taken up by immune cells and trafficked to the wound site. This mechanism has been demonstrated in mice using MRSA and has been proposed to help explain infections due to organisms not detected in the wound at the end of surgery.⁶ Factors such as wound drainage, anticoagulation, and impaired perfusion further prolong tissue susceptibility, increasing the likelihood that organisms colonizing the nares or skin reach the surgical site.³

Daily Care Activities Create Repeated Pathogen Inoculation Opportunities

Routine postoperative activities—including dressing changes, wound assessments, manipulation of drains and catheters, and patient self-contact—provide repeated opportunities for bacteria from the nares or skin to reach the incision. Hospital microbiota readily colonize patient surfaces, and organisms from the nares frequently reseed nearby skin sites, sustaining the potential for contamination throughout recovery.^{15,16}

Risk of New Colonization Acquired in the Hospital

Even when preoperative decolonization is initially effective, patients may reacquire or newly acquire nasal colonization during hospitalization through

environmental exposure, healthcare contact, or medical devices.^{3,4} The postoperative period is characterized by rapid microbial turnover, as patients both shed their own microbiota and acquire organisms from surrounding surfaces, staff, and shared spaces. This dynamic exchange underscores that colonization is not static and can shift quickly during recovery.⁹

Together, these postoperative factors create a clinically significant risk window that extends well beyond the operative event. Without measures that continue to suppress nasal and skin bacterial burden during this period, patients remain vulnerable to both endogenous reseeding and newly acquired colonization. This persistent risk underscores the need for postoperative proactive treatment of the microbiome of the nares in further controlling SSIs.¹¹

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Limitations of Preoperative-Only Decolonization

Preoperative nasal decolonization can reduce bacterial burden at the time of incision, but its protective effect is often transient once patients re-enter the hospital microbial environment. Risk is uneven across carriers: higher nasal burden and carriage within 24 hours after surgery are associated with increased postoperative infection risk. As a result, a single preoperative suppression window may be mismatched to the biology of high-burden carriers.^{13,15,17} Recolonization may occur rapidly or variably after treatment, indicating that short-term “resetting” of the nares may not reliably ensure suppression through recovery.¹⁷

Preoperative screening for the presence of nasal colonization or other targeted approaches cannot address infections driven by postoperative acquisition or shifts in colonization during hospitalization; while most SSIs involve strains already carried by the patient, a meaningful minority arise from newly acquired or nosocomial organisms not detected preoperatively.³

Decolonization guidelines recommend topical chlorhexidine gluconate (CHG) for daily bathing along with either mupirocin or an intranasal antiseptic agent (e.g., alcohol-based or povidone-iodine).¹⁸ Routine use of mupirocin presents challenges, including emerging resistance, variable adherence to the five-day preoperative regimen, and infeasibility in urgent surgeries.¹⁹ When the full course is completed and resistance is not present, mupirocin can provide sustained decolonization that extends into the wound-healing period. In contrast, nasal antiseptic regimens act immediately and do not require advance treatment, but their effect is not prolonged after discontinuation, so reapplication during the healing window may be needed to help maintain a low bacterial burden.¹⁰

The AORN 2021 Preoperative Skin Antisepsis Guideline suggests that “surgical patients may benefit from relatively short-term decolonization or until the surgical incision has healed,” signaling that protection limited solely to the preoperative window may not be sufficient for some patients.¹⁸

Professional guidelines have begun to acknowledge this post-operative gap. The AORN 2021 Preoperative Skin Antisepsis Guideline suggests that “surgical patients may benefit from relatively short-

term decolonization or until the surgical incision has healed,” signaling that protection limited solely to the preoperative window may not be sufficient for some patients.¹⁸ While not prescribing a specific postoperative protocol, this language supports implementing protocols for continued decolonization during the healing phase.

Taken together, the evidence supports a central limitation of preoperative-only approaches: they can meaningfully reduce burden at incision, yet they may not reliably cover the full period of postoperative vulnerability or the within-hospital colonization dynamics that can sustain or reintroduce risk.^{3,4,13,15} These findings reinforce the need for strategies that maintain suppression of nasal and skin bacterial burden beyond the preoperative window and through postoperative recovery when clinically appropriate.

Evidence Supporting Combined Pre- and Postoperative Nasal Decolonization

Emerging evidence suggests that extending nasal decolonization into the postoperative period may offer additional protection during the timeframe when patients are most vulnerable to recolonization and hospital-acquired microbial exposure. Studies incorporating both pre- and postoperative nasal antisepsis have reported reductions in SSIs in procedures including total joint arthroplasty, indicating that maintaining suppression of nasal bacterial burden beyond the day of surgery may help mitigate short-term recolonization or de novo acquisition after the procedure.^{20,21}

Universal decolonization programs—where all surgical patients receive pre- and post-operative nasal antisepsis and skin cleansing regardless

of colonization status—have also demonstrated reductions in SSIs and improvements in patient outcomes and cost metrics.²² These findings support the feasibility of implementing broader, extended-use strategies across the surgical episode of care.

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Meta-analytic evidence further shows that protocols including both pre- and postoperative nasal antisepsis are associated with lower SSI rates than preoperative-only approaches, although specific postoperative durations vary across studies.²⁰

Despite these promising findings, important evidence gaps remain. Large, controlled studies may be useful to evaluate the degree to which postoperative extension enhances outcomes beyond preoperative-only regimens and to define the optimal duration, frequency, and candidate populations for extended use. Additional research could also further assess adherence, tolerability, and potential resistance risks, as well as to better understand postoperative recolonization and acquisition patterns that may inform decolonization strategies throughout the full wound-healing period.

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