Arterial grafting and the risk of sternal infection: How we can learn from our experience

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Deep sternal wound infection (DSWI) is a known and dreaded complication after coronary artery bypass grafting (CABG). The surgical literature suggests an incidence ranging from 0.7%^{1,2} to 2.3%.³ However, more recent reports from the Society of Thoracic Surgeons (STS) National Adult Cardiac Surgery Database report an overall incidence for isolated CABG in 2007 of 0.3%, which had decreased to 0.2% in the most recent 2014 report.⁴ Indeed, despite the increasing risk profile of surgical patients in recent years, the incidence of DSWI has not increased.^{5,6} This potentially life-threatening complication is associated with the need for subsequent procedures, with higher morbidity and higher early mortality—a mortality that can range from 1.5% when occurring as an isolated complication up to 46.3% when associated with multiple other complications.⁷⁻⁹ The impact of DSWI on late mortality is controversial and likely related to how patient groups are defined and cared for in various health care environments.^{8,10-12}

The surgical literature is replete with studies defining the risk factors for DSWI, with several validated risk scoring systems of varying levels of accuracy.¹³ The most recent STS Model for DSWI (one of the most accurate) reports a C-statistic (measure of discrimination) of 0.686,⁴ which is generally considered to lie in the “scarcely accurate” (0.5-0.7) range.¹⁴ The most commonly cited independent risk factors include diabetes and obesity, with immunosuppression, heart failure, chronic obstructive pulmonary disease, renal failure, peripheral vascular disease, smoking, prolonged operating times, use of the internal thoracic artery, blood transfusions, need for surgical reexploration, and need for postoperative cardiopulmonary resuscitation all being well documented in various series.¹³ More recently, intriguing evidence has documented a possible role for psychologic anxiety and depression—an area perhaps sorely in need of more investigation.^{15,16} Given

the low incidence, the ability of any one surgical series to identify independent predictors of DSWI is obviously limited by prevalence and sample size. However, the impact of center-level factors cannot be overemphasized. Shih and colleagues¹⁷ recently reported that in the state of Michigan, the predicted risk of hospital-acquired infection after CABG varied by 2.8% across centers (ie, patient level factors), whereas the actual observed rates of infection varied by 18.2%.¹⁷

It is in this regard that the report in this issue of the *Journal* from Kieser and colleagues¹⁸ makes a somewhat stunning contribution. Rather than applying statistical modeling to their results to report risk factors, the authors chose to chronicle their efforts to eliminate DSWI in the face of a dedicated and unusual commitment to bilateral internal thoracic artery (BITA) grafting, even in diabetic patients. As noted by the authors, despite a wealth of retrospective data documenting a long-term survival benefit for BITA versus single internal thoracic artery (SITA) grafting,¹⁹ use of BITA grafting in clinical practice is remarkably uncommon—4% in North America, 12% in Europe, and 30% in Japan.²⁰ It is specifically the risk of DSWI that has discouraged surgeons from using this approach in diabetic patients. This trend persists, despite increasing evidence that BITA grafting can be accomplished in diabetic patients, using a skeletonized harvesting approach, with no significantly increased risk of DSWI, yet with a significant improvement in long-term survival.^{21,22} Indeed, the aggressive and diffuse nature of diabetic coronary atherosclerosis may make BITA grafting specifically more suitable for the diabetic patient. The increased risk of infection does not seem to equal the survival benefit.

However, Kieser and colleagues¹⁸ were not satisfied to merely accept the risks of DSWI, but rather embarked on a journey to eliminate them, without compromising the integrity of the revascularization offered to the patient. As any good quality improvement effort to address a complex multifactorial problem in cardiac surgery, the authors' approach involved no less than 12 quality improvement efforts to reduce infection, 8 of which are specifically tracked in this report. Each one individually has a reasonable evidence base. The cumulative impact was to reduce the incidence of DSWI in the most recent 469 of 1001 CABG cases to zero. Two obvious questions inevitably arise. First, is zero really zero? Although the authors present interesting and somewhat compelling evidence based on statistical modeling that the probability

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of future infection based on the most recent algorithm is extremely low, the point is somewhat moot. The reality is that they were able to achieve an extremely low and markedly reduced infection rate using BITA grafting 76% of the time, and 35% of patients were diabetic. Whether the number is truly zero, or just incredibly low, only time will tell. Could the lower prevalence of insulin-dependent diabetes (but not diabetes in general) and chronic obstructive pulmonary disease in the “after change-point” patient group have played a role in the reduced infection rate? Probably, although the size of the reduction seems clearly out of proportion to the size of risk reduction. In any event, their patients are benefitting from advanced arterial revascularization without the traditionally reported incidence of DSWI.

The second obvious question that arises is which intervention is it that accounted for the decrease? A purist might relish the opportunity to examine the impact of each measure in isolation. Unfortunately, for the 8 measures alone this would involve 255 possible combinations—for all 12 no less than a staggering 4095—well beyond the scope of any reasonable clinical investigation, especially when exploring an outcome with an incidence in the 1% range (or less). In reality, this is not what surgeons do. We address a problem with the full vigor of available knowledge. In fact, others have reported the success of this “bundled” approach to reducing DSWI after cardiac surgery.²³ What is more instructive than the actual measures used is the process by which the authors addressed the topic. Not satisfied with improvement, they persisted in their efforts until they had, at least for now, eliminated the problem. During the process, they then applied sophisticated statistical modeling to attempt to retrospectively analyze their process in an attempt to discern which element seemed to have had the most profound impact. They then used this information in an ongoing basis to change clinical behavior. We might want to debate whether the incremental potential survival benefit of BITA grafting in a given vigorous, young, obese, diabetic woman might not warrant the risk of DSWI, but the authors’ experience with this group of patients certainly warrants caution. Perhaps ironically, the long-term survival benefit of BITA versus SITA grafting is less clear for women than it is for men. In the largest series reported to date, which unfortunately consisted of only 329 women, BITA grafting did not confer a late survival benefit compared with SITA grafting in propensity-matched female patients, even though operative survival was better.²⁴

In any event, it is not certain that what determines DSWI in one center will necessarily be applicable to another. Even large data repository reports like that from the STS may have limited applicability to other patient populations.²⁵ These large registry findings are certainly corroborated by the hospital-level data reported by Shih and colleagues¹⁷

from the Michigan experience, which essentially teach us that hospital-level, rather than patient-level factors account for more of the variance in postsurgical infection. The true message of Kieser and colleagues¹⁸—aside from the definitively actionable information related to minimizing the risk of DSWI while maintaining a commitment to arterial revascularization—is that it is ultimately the dedication to clinical improvement through rigorous self-examination and evidence-based programmatic adaptation that will drive surgical quality. Both her professional colleagues and her patients are the true beneficiaries.

References

1. Borger MA, Rao V, Weisel RD, Ivanov J, Cohen G, Scully HE, et al. Deep sternal wound infection: risk factors and outcomes. *Ann Thorac Surg.* 1998;65: 1050-6.
2. Lua JCY, Graysonb DA, Jhaa P, Srinivasana AK, Fabria BM. Risk factors for sternal wound infection and mid-term survival following coronary artery bypass surgery. *Eur J Cardiothorac Surg.* 2003;23:943-9.
3. The Parisian Mediastinitis Study Group. Risk factors for deep sternal wound infection after sternotomy: a prospective, multicenter study. *J Thorac Cardiovasc Surg.* 1996;111:1200-7.
4. Society of Thoracic Surgeons. Data Analyses of the Society of Thoracic Surgeons National Adult Cardiac Surgery Database; report modules 2007 and 2014.
5. Ferguson TB Jr, Hammill BG, Peterson ED, DeLong ER, Grover FL, STS National Database Committee. A decade of change—risk profiles and outcomes for isolated coronary artery bypass grafting procedures, 1990-1999: a report from the STS National Database Committee and the Duke Clinical Research Institute. Society of Thoracic Surgeons. *Ann Thorac Surg.* 2002;73: 480-9.
6. Doherty C, Nickerson D, Southern DA, Kieser T, Appo J, Dawes J, et al; for the Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease (APPROACH) Investigators. Trends in postcoronary artery bypass graft sternal wound dehiscence in a provincial population. *Plast Surg.* 2014;22: 196-200.
7. Tang GH, Maganti M, Weisel RD, Borger MA. Prevention and management of deep sternal wound infection. *Semin Thorac Cardiovasc Surg.* 2004;16: 62-9.
8. Toumpoulis IK, Anagnostopoulos CE, DeRose JJ. The impact of deep sternal wound infection on long term survival after coronary artery bypass grafting. *Chest.* 2005;127:464-71.
9. Filsoofi F, Castillo JG, Rahmanian PB, Broumand SR, Silvay G, Carpentier A, et al. Epidemiology of deep sternal wound infection in cardiac surgery. *J Cardiothorac Vasc Anesth.* 2009;23:488-94.
10. Cayci C, Russo M, Cheema F, Martens T, Ozcan V, Argenziano M, et al. Risk analysis of deep sternal wound infections and their impact on long-term survival: a propensity analysis. *Ann Plast Surg.* 2008;61:294-301.
11. deMoraes AAI, Abboud CS, Chammas AZL, Aguiar YS, Mendes LC, Neto JM, et al. Long term mortality of deep sternal wound infection after coronary artery bypass surgery. *Rev Bras Cir Cardiovasc.* 2012;27:377-82.
12. Colombier S, Kessler U, Ferrari E, von Segesser LK, Berdajs DA. Influence of deep sternal wound infection on long-term survival after cardiac surgery. *Med Sci Monit.* 2013;19:668-73.
13. Buja A, Zampieron A, Cavalet S, Chiffi D, Sandona P, Vinelli A, et al. An update review on risk factors and scales for prediction of deep sternal wound infections. *Int Wound J.* 2012;9:372-86.
14. Swets JA. Measuring the accuracy of diagnostic systems. *Science.* 1988;240: 1285-93.
15. Williams JB, Alexander KP, Morin JF, Langlois Y, Noiseux N, Perrault LP, et al. Preoperative anxiety as a predictor of mortality and major morbidity in patients aged >70 years undergoing cardiac surgery. *Am J Cardiol.* 2013;111: 137-42.
16. Theodore DA, Goodwin RD, Schneider N, Goldsmith L, Gordon RJ. Depression is associated with increased risk of sternal wound infection after cardiothoracic surgery. Presented at: the 54th Interscience Conference on Antimicrobial Agents and Chemotherapy; September 5-9, 2014, Washington, DC.

17. Shih T, Zhang M, Kommareddi M, Boeve TJ, Harrington SD, Holmes RJ, et al; for the Michigan Society of Thoracic and Cardiovascular Surgeons Quality Collaborative. Center-level variation in infection rates after coronary artery bypass grafting. *Circ Cardiovasc Qual Outcomes*. 2014;7:567-73.
18. Kieser M, Rose MS, Aluthman U, Montgomer M, Louie T, Belenkie I. Toward zero: deep sternal wound infection following 1001 consecutive coronary artery bypass procedures using arterial grafts: Implications for diabetic patients. *J Thorac Cardiovasc Surg*. 2014;148:1887-95.
19. Yi G, Shine B, Rehman SM, Altman DG, Taggart DP. Effect of bilateral internal mammary artery grafts on long-term survival: a meta-analysis approach. *Circulation*. 2014;130:539-45.
20. Kinoshita T, Asai T. Bilateral internal thoracic artery grafting current state of the art. *Innovations*. 2011;6:77-83.
21. Puskas JD, Sadiq A, Vassiliades TA, Kilgo PD, Lattouf OM. Bilateral internal thoracic artery grafting is associated with significantly improved long-term survival, even among diabetic patients. *Ann Thorac Surg*. 2012;94:710-5.
22. Dorman MJ, Kurlansky PA, Traad EA, Galbut DL, Zucker M, Ebra G. Bilateral internal mammary artery grafting enhances survival in diabetic patients: a 30 year follow-up of propensity score matched cohorts. *Circulation*. 2012;126:2935-42.
23. Miyahara K, Matsuura A, Takemura H, Mizutani T, Saito S, Toyama M. Implementation of bundled interventions greatly decreases deep sternal wound infection following cardiovascular surgery. *J Thorac Cardiovasc Surg*. April 13, 2014 [Epub ahead of print].
24. Kurlansky PA, Traad EA, Dorman MJ, Galbut DL, Zucker M, Ebra G. Bilateral internal mammary artery grafting reverses the negative influence of gender on outcomes of coronary artery bypass grafting surgery. *Eur J Cardiothorac Surg*. 2013;44:54-63.
25. Kirmani BH, Mazhar K, Saleh HZ, Ward AN, Shaw M, Fabri BM, et al. External validity of the Society of Thoracic Surgeons risk stratification tool for deep sternal wound infection after cardiac surgery in a UK population. *Interact Cardiovasc Thorac Surg*. 2013;17:479-84.