Editorial

Risk Factors for Infection in Cardiac Surgery: Will the Real Culprit Please Stand Up?

Cardiac surgery is the epitome of contemporary hightechnology medical care. There are few phenomena in the history of medicine which rival cardiac surgery in the sheer complexity of its technology, its costs, and in the frequency with which it is employed. The average coronary artery bypass procedure involves an operating theatre which contains approximately \$250,000 worth of highly sophisticated equipment. Ten to fifteen elaborately trained physicians and hospital employees have designated responsibilities within the operating theatre itself; ten times that many employees will be involved in the perioperative and postoperative management of the patient. And the procedure is performed 800 times a day in American hospitals!

Given the complexity of the procedure and the potential for human error, it is somewhat of a modern miracle that complications are so infrequent. Infectious complications, particularly deep infections of the sternum, occur in well under 5% of patients undergoing cardiac bypass surgery. Having observed numerous bypass procedures for the express purpose of identifying potential infection hazards, I have been impressed with the resiliency of the human sternum. Following median sternotomy, this relatively avascular tissue is cauterized or coated with bone wax (or both), pushed, stretched, abraded, and dehydrated. After hours of such abuse, it is ignobly wired together and expected to heal, without complaint, in a matter of weeks.

Nevertheless, even under the best of circumstances, wound infections do occur. And, as when infection interferes with any clean surgical procedure, infections after cardiac surgery are unexpected, emotionally unsettling for both patient and physician, and often are associated with disastrous results. Deep sternal wound infections and

mediastinitis are associated with high mortality, high morbidity, and add significantly to the overall cost of cardiac surgery. Small wonder that the person most concerned with aseptic technique and meticulous infection control in a modern hospital is frequently the cardiac surgeon.

In this issue of *Infection Control* two institutions report clusters of excessive numbers of post-op infections in their respective cardiac surgery programs. In the study by de Silva and Rissing, 9% of patients developed postoperative wound infection following cardiac surgery during the first six months after a relocation to a new facility.² Performing cardiac surgery in a room used within the past 48 hours for a "contaminated" case was statistically associated with increased wound infections. In the study by Dandalides, Ratula, and Sarubbi, 23.7% of patients undergoing cardiac surgery developed a postoperative wound or nonwound infection during a two-month period in 1981.3 An extensive epidemiologic and microbiologic study of the cardiothoracic intensive care unit was undertaken. Contamination of the hands of personnel and environment was documented to occur only when water was turned on at a utility sink. Sternal wound infections were found to be related epidemiologically and bacteriologically to the cardiothoracic intensive care unit, suggesting that postoperative factors may be responsible for deep sternal wound infections.

The findings of both studies are unsettling. The contemporary concepts that: 1) the airborne route is of minor importance in the contamination of surgical wounds, and 2) postoperative factors do not influence infection rates in primarily closed wounds, have been challenged by these studies. Their findings, however, must be viewed very critically, indeed, skeptically. The problem is not with the results of the study—the airborne route and postopera-

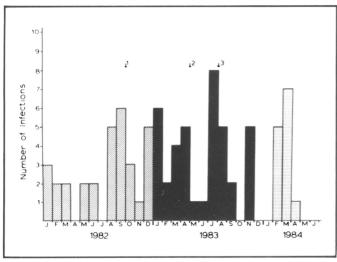


Figure. Sternal wound infections from January 1982 through June 1984. Approximately 100 median sternotomy procedures were performed each month during this period of time. Sternal wound infections have varied from 8 to 0 per month. Formal investigations of increased numbers of infections are noted by the arrows.

tive factors may be in fact very important in the pathogenesis of surgical wound infection. Rather, the problem is with the methodology.

Both studies employed case control analysis to identify risk factors for the development of wound infection. Correcting the identified risk factors (ie, improving the air filtration system² and removal of the utility sink³) was associated with a marked decline in infection rates. Although this make-a-change-and-see-what-happens approach (post hoc ergo propter hoc) is commonly employed in the analysis of postsurgical wound infection, ^{4,5} the limitations must be appreciated. The observed declines in infection rate may not be causally related to the implementation of control measures. Especially when multiple pathogens are involved, it is dangerous to assume that a cluster of postsurgical infection is related to any single break in technique.

An analysis of sternal wound infections at Saint Thomas Hospital for the past two and a half years has revealed apparent clusters of infections (Figure). During the period of time represented by the Figure, open heart procedures were performed at a fairly steady 100 procedures per month. Months with clusters of six to eight sternal wound infections were followed by months with no infections. As indicated by the arrows, our first investigation of a cluster of infections occurred in late 1982 and was suspected to be related to improperly cleaned sternal saws. Following a change in procedure, the epidemic "resolved." Another cluster of infections was noted in early 1983. A second investigation ensued and a single surgical resident was found to be associated with threefourths of all infected patients. When the surgical resident rotated off the cardiac surgery service, the outbreak resolved. Finally, a large cluster of cases occurred in mid-1983. An intensive investigation again implicated sternal saws. The use of the malfunctioning saws was discontinued and new sternal saws were purchased. Infection rates promptly fell. Most importantly, however, clusters of infection recurred in November 1983 and early in 1984: investigations were not initiated and the "epidemics" resolved spontaneously.

The error in the make-a-change-and-see-what-happens analysis of postsurgical infection outbreaks is the assumption that problems discerned during the investigation of an epidemic did not exist before the epidemic. The resolution of the problem and the subsequent reduction in infection rates are thus thought to be causally related. If no "cause" is found, but the rate of infection declines, it is usually assumed that the activity of the infection control personnel has, a la the Hawthorne effect, led to an improvement in subtle infection control practices and the resolution of the outbreak. It is possible, however, that the rise and fall of infection rates may have occurred independently of the activities of the infection control personnel and that unidentified factors influenced the non-random distribution of infections.

Meakins and others have championed the need to prospectively stratify risk factors of patients undergoing surgery.⁶ Their initial data suggest that important differences in risk of infection following surgery can be discerned preoperatively with good reproducibility among various institutions. This prospective evaluation of patient risk factors for infection can be broadened to include surgical technique, operating room protocol, and environmental factors. The challenge to infection control practitioners interested in surgical wound infections is to develop intense prospective analyses of risk factors during the endemic (low infection rate) period of time. The relative importance of various risk factors may thus be discerned for both the epidemic and endemic periods of wound infection rates. In time, the technology of hospital epidemiologists may more closely complement that of the surgical procedures we are charged with monitoring. Meanwhile, the studies by deSilva and Rissing² and Dandalides et al³ provide a provocative focus on a very complex surgical procedure.

REFERENCES

- Bor DH, Rose RM, Modlin JF, et al: Mediastinitis after cardiovascular surgery. Rev Infect Dis 1983; 5:885-897.
- deSilva MI, Rissing JP: Postoperative wound infections following cardiac surgery: Significance of contaminated cases performed in the preceding 48 hours. *Infect Control* 1984; 5:000-000.
- Dandalides PC, Rutala WA, Sarubbi FA, Jr: Postoperative infections following cardiac surgery: Association with an environmental reservoir in a cardiothoracic intensive care unit. *Infect Control* 1984; 5:000-000.
- Engelman RM, Williams CD, Gouge TH, et al: Mediastinitis following open-heart surgery. Arch Surg 1973; 107:772-778.
- Ehrenkranz NJ, Bolyard EA, Wiener M, et al: Antibiotic-sensitive Serratia marcescens infections complicating cardiopulmonary operations: Contaminated disinfectant as a reservoir. Lancet 1980; 2:1289-1292.
- Nystrom P, Meakins JL: Comparison of two methods for scoring of surgical sepsis. Read before the Fourth Annual Meeting of the Surgical Infection Society, Montreal, April 30-May 1, 1984.

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