ORIGINAL ARTICLE

An audit of risk factors for wound infection in patients undergoing coronary artery bypass grafting or valve replacement

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Objectives To investigate the epidemiology of surgical site infection in cardiac surgery patients operated on in 2006.

Design Retrospective study of a case-control sample.

Setting Cardiac surgery unit of a university teaching hospital in Hong Kong.

Patients Cardiac surgery patients with surgical site infection were matched by procedure type, sex, and year of surgery with non-infected patients.

Main outcome measures Identification of risk factors for surgical site infection.

Results The infected and non-infected cardiac surgery patients did not differ in age, sex, or smoking history; however, patients with surgical site infection were significantly heavier (mean body mass index, 26.6 vs 23.9 kg/m^2, P<0.046). Almost 41% of the subjects had a history of diabetes mellitus, there being a significantly greater proportion among infected than non-infected patients (53.1% vs 28.1%, P<0.042). All 37 of the patients without a diagnosis of diabetes had normal (ie <8 mmol/L) preoperative glucose levels, but 99% of them yielded evidence of subsequent glycaemic dysfunction during or after surgery. Overall, 50% of the patients had a blood transfusion during the operation, with infected patients significantly more likely to have been transfused than the non-infected ones (65.6% vs 34.4%, P<0.008).

Conclusions There appears to be a relationship between surgical site infection in cardiac surgery patients and pre-existing (diagnosed and covert) diabetes mellitus and blood transfusion. Future studies should consider these factors in relation to surgical site infections, both in the wider surgical population and from a risk-minimisation perspective.

Key words
Blood transfusion; Cardiac surgical procedures; Diabetes mellitus; Risk factors; Surgical wound infection

Hong Kong Med J 2008;14:371-8

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Introduction

As many as 5% of patients develop surgical site infections (SSIs),[1] of which approximately two thirds are confined to the incision. Wound infections are either superficial or deep, and may involve the organs or spaces accessed during the operation.[2] Classified according to the extent of wound contamination, SSIs are described as clean, clean contaminated, contaminated, or dirty, depending on wound appearance, discharge, and the likelihood of peri-operative bacterial contamination from disruption of specific structures. The incidence of SSIs varies for each operative procedure, surgeon, and institution, suggesting that there are modifiable workplace and practice contributing factors.

Following open-heart surgery, sternal wound infections (SWIs) occur in up to 5% of patients and can be potentially life-threatening.[3] Significant preoperative risk factors associated with SWI include diabetes mellitus (DM), respiratory disease, a history of smoking, peripheral vascular disease, renal failure, obesity, hypertension, and angina (class 3 or 4).[4,5] The risk for SWI is increased if surgery involves both internal thoracic arteries, a valve procedure, or a ventricular assist device.[4,5] Leg wounds at donor sites account for more than 70% of cases with severe infection following cardiac surgery.[6] In over 50% patients with SSIs, the pathogen implicated is staphylococcus. In one study, they comprised methicillin-susceptible *Staphylococcus epidermidis* (26.7%), methicillin-resistant *S epidermidis* (15.5%), methicillin-resistant *Staphylococcus aureus* (12.1%), and methicillin-susceptible *S aureus* (3.2%).[6] Up to 20% of such clinically suspected wound infections are

引致接受冠狀動脈搭橋術或瓣膜置換術病人出現傷口感染的風險因素評估

目的 以2006年接受心臟手術的病者為病例對照樣本，探討手術部位感染的流行病學情況。

設計 對病例對照樣本進行回顧研究。

安排 香港一所大學教學醫院的心臟外科部。

患者 按手術種類、年份、病人性別，把手術部位受感染的心臟病患者與沒有受感染的患者配對。

主要結果測量 在研究對象中，識別出造成手術部位感染的風險因素。

結果 受感染和無感染的心臟病患者在年齡、性別以至吸煙情況並無差別，但受感染的病人明顯較重（平均身體質量指數比較：26.6對23.9 kg/m^2, P<0.046）。研究的病人中，曾患糖尿病的幾近41%，其中受感染患者所佔的比例明顯較高（53.1%對28.1%, P<0.042）。32位沒有診斷出患糖尿病的病人，術前血糖水平均屬正常（即<8 mmol/L），但99%相繼在術中和術後出現血糖功能異常。整體來説，五成病人接受術中輸血，其中又以受感染的病人的人數尤其多（65.6%對34.4%, P<0.008）。

結論 心臟病患者手術部位感染似乎與患者術前已患糖尿病（不論是醫生診斷到的或是隱性的）以及輸血有關。日後若進行更深入的研究，應以更多的手術病人為樣本，並以把風險降至最低的角度，考慮這些因素與手術部位感染的關係。

culture negative.[6]

Not surprisingly, patients who develop SSIs manifest a significant decline in the mental health component of quality-of-life scores.[7] Patients with wound infections in the early postoperative phase experience greater morbidity, require longer periods of hospitalisation, and incur greater treatment costs.[8] Cardiac SSI increases the length of hospital stay and costs in proportion to the severity of the infection; costs increase by 3.8%, 14.7% and 29.4% in mild, moderate and severe infections, respectively.[6] The corresponding figures for increase in length of hospital stay have been reported as 5, 9, and 15 days, respectively.[6]

The majority of SSIs are diagnosed after discharge, necessitating out-patient consultations, emergency room visits, radiology services, hospital readmissions, and home care services. The average total cost during the 8 weeks following discharge for an SSI patient has been estimated at US$5155.[7] Clearly, SSIs impose a substantial strain on health care personnel and hospital resources.[1]

Treatment is often confounded by the emergence of antibiotic-resistant pathogens[1] and because a substantial proportion of infected patients are elderly or have co-existing medical problems, in the past they would not have been considered for surgery.[2] As the population ages, it is reasonable to assume that older and sicker patients will be admitted for surgery, which will inevitably increase the risk and incidence of SSIs.

Within our institution, the infection rate of postoperative wounds has been under surveillance by the Infection Control Team since 2005. The aim of this study was to audit and report on SSIs, and identify likely risk factors among cardiac surgery patients receiving coronary artery bypass grafting (CABG) or valve replacement at our institution in 2006. The corresponding caseload ratio for our team was 60% versus 31%, whilst 9% of cases involved other forms of cardiac surgery.[3]

Methods

All patients attended the preoperative work-up out-patient clinic and were admitted to the cardiac ward the day prior to surgery. Preparation for surgery included routine showering and standard preoperative antibiotic prophylaxis with cefuroxime 1.5 g on induction of anaesthesia. Postoperative antibiotic cover was maintained for 48 hours using intravenous 750 mg cefuroxime boluses three times per day.

The US National Nosocomial Infection Surveillance System (NNIS) was used to identify patients with SSI. All surgical patients were observed on postoperative day 2 and then telephoned before day 30 and asked a standard set of questions related to their wounds. Classification of SSI was based on the report of purulent discharge, pain, erythema or swelling, the organisms cultivated, and any intervention by the surgeon or local doctor.

A case-control design was used to match the identified SSI cardiac surgery patients by gender and procedure type with non–wound infected patients for the year 2006. Patients were considered to be cases if they had been reported by the Infection Control Team to have an SSI following either a CABG or a cardiac valve replacement procedure during 2006. Control patients were selected based on matching the SSI cases by gender and procedure and also having been operated on in our department in 2006.

The exposures of interest were selected following a comprehensive review of independent risk factors for SSI in cardiac patients.[3-5,9,10] These variables included: smoking history, diagnosis of DM, infection site, postoperative number of days before the SSI, duration of surgery, American Society of Anesthesiologists (ASA) score, blood transfusion, and blood glucose levels at different time points (preoperative, intra-operative, recovery, intensive care unit [ICU], and high dependency unit). Data were extracted from patient charts and the cardiac surgery data registry by an independent auditor.

The data were presented as means and standard deviations (or medians and interquartile ranges, wherever appropriate). The differences in variables between the groups were tested using either Student's unpaired *t* tests or the Mann-Whitney *U* test. The differences in variables between different time points were compared using the paired *t* test. Associations between variables and the outcomes were tested by the Chi squared or Fisher's exact test. Logistic regression analysis on the variables identified with P<0.1 in the univariate analysis, was used to create a predictive model for outcome. Significance was set at P<0.05.

Results

According to the NNIS criteria and surveillance by the Infection Control Team at our institution, the point prevalence of SSI in our surgical patients ranged between 7.6% and 10% over the last 3 years. In 2006, there were 32 cases among our cardiac patients; the entire case-control group consisted of 63 patients. Their characteristics are shown in Table 1. The overall mean age was 61 (range, 29-78) years. The SSI and non-infected cardiac surgery patients did not differ in terms of age, gender, or smoking history. However, the SSI patients were significantly heavier, being classified as overweight (mean body mass index [BMI], 25.7 kg/m^2), whereas the non-SSI patients were within the normal BMI range (mean, 23.9 kg/m^2; P=0.047); BMI >25 kg/m^2 is considered overweight.

A total of 50% of the SSI cases occurred in the sternum, and 34% of infections were in the lower limb donor sites. The mean number of postoperative days before infection was 17, with a range from 5 to 30 days. Diabetic patients had wound breakdown at a mean of 16.6 days (range, 9-29 days), no difference from the mean of 17.4 days (range, 5-30 days) was noted in non-diabetic patients.

The overall mean duration of surgery was 175 minutes, being 177 minutes for CABG and 165 minutes for valve replacements. There was no difference in the duration of surgery between the infected and non-infected groups. The wound infection patients had a mean ASA co-morbidity score of 3, which denotes severe systemic disease that limits activity but is not incapacitating. Our non-infected patients had a mean ASA score of 2.9, despite evidence that an ASA score higher than 2 is associated with wound infections.

Almost 41% of the 63 patients had a history of DM. Significantly more SSI than non-infected patients were known to be diabetic (53% vs 28%, P<0.042). The mean intra-operative blood glucose levels did not differ, nor was there any difference in the absolute change of blood glucose levels between the infected and non-infected groups (Table 2). As can be seen from the Figure, in both groups the mean blood glucose levels remained significantly elevated from the baseline at all time points during the postoperative phase. A preoperative blood glucose level of 8 mmol/L or higher (the acknowledged cut-off point for hyperglycaemia) was noted in nine SSI patients and three without wound infections (P<0.055). The mean blood glucose levels of all patients at peri-operative and postoperative time points were within

TABLE 1. Patient demographics, clinical characteristics, and surgery undergone

Characteristic	Cases (n=32)	Controls (n=31)	P value
Sex			
Male	19	19	0.877
Female	13	12	
Mean age (SD) [years]	59.1 (11.7)	63.0 (9.4)	0.156
Mean body mass index (SD) [kg/m^2]	25.7 (4.0)	23.9 (2.7)	0.047
Smoking status*			
Never	14	14	1.000
Ever	17	17	
Type of surgery			
Coronary artery bypass grafting	25	24	0.946
Valve replacement	7	7	
Mean (SD) No. of postoperative days before the infection	17 (7)	-	-
Site of infection		-	-
Sternum	16		
Lower limb	11		
Septicaemia	1		
Multiple	4		

* Data were missing for one case

TABLE 2. Blood glucose levels at different peri-operative time points

Time points*	Mean±SD glucose level (mmol/L)		P value
	SSI group†	Control group	
Preoperative	6.5±2.5	5.8±1.7	0.207
Mean for intra-operative period	9.6±2.9	8.7±2.0	0.140
Mean during recovery	10.2±2.7	9.2±1.9	0.183
Peak during recovery	11.7±3.6	10.6±2.5	0.298
Mean during ICU stay	9.5±1.3	9.3±1.8	0.051
Peak during ICU stay	10.5±1.9	10.0±1.9	0.040
Mean during HDU stay	10.0±2.9	9.9±1.7	0.152
Peak during HDU stay	14.0±4.4	11.5±2.5	0.618
Peak glucose level at any one time	13.1±3.9	11.3±2.8	0.914

* ICU denotes intensive care unit, and HDU high dependency unit
† SSI denotes surgical site infection

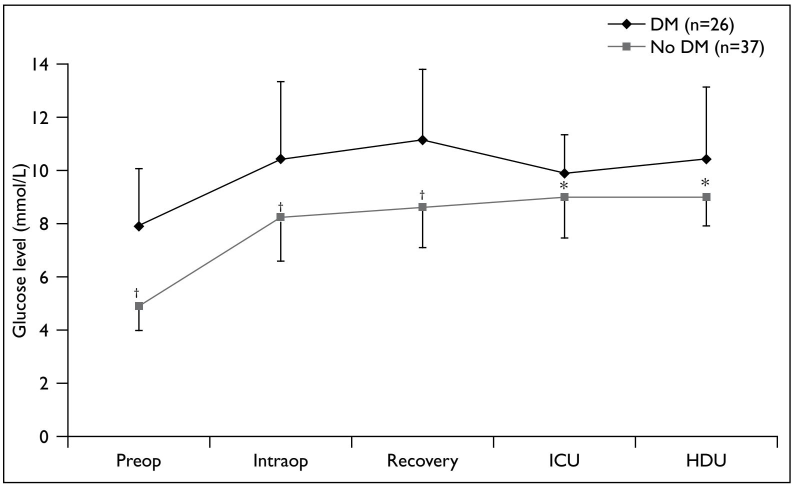

FIG. Mean blood glucose levels at different peri-operative phases in patients with and without surgical site infections

Preop denotes preoperative, Intraop intra-operative, ICU intensive care unit, HDU high dependency unit, and DM diabetes mellitus; error bars denote SD

* P<0.05

† P<0.001

the dysfunctional range. At all time points, higher blood glucose readings were noted in the DM than non-DM patients.

All 37 of the patients without a diagnosis of DM had normal (ie <8 mmol/L) preoperative glucose levels, but 99% of them manifested evidence of glycaemic dysfunction (ie blood glucose >8 mmol/L) at some time point during or after surgery. Intra-operative hyperglycaemia was significantly associated with surgery that lasted longer than 3 hours (P<0.005). Half of the patients who developed hyperglycaemia did so during the operation, whereas in the rest its onset was during recovery or in the ICU. There was a significant association between hyperglycaemia during the operation and the duration of the recovery period (P<0.028), but no association with the ASA score, a BMI over 25 kg/m^2, or in-hospital blood transfusion.

Overall, 50% of the patients had blood transfusions during the operation or at some point before discharge from the hospital. Patients with SSIs were significantly more likely to have had a blood transfusion than the controls (65.6% vs 34.4%, P<0.008). Patients who had received a blood transfusion and those who had not, manifested their SSI after a mean of 18 and 17 days, respectively.

Table 3 shows the association between exposure and SSI. The logistic regression retained the two variables of 'known diagnosis of diabetes' (P=0.031) and having received a 'blood transfusion while hospitalised' (P=0.003) as the significant independent risk factors.

Discussion

The incidence of wound infection in our service was high compared to that of other institutions in Hong Kong. As it is known that wound infection occurs when bacterial contamination overcomes a host's defenses against bacterial growth,[11] the parameters of the individuals should also be considered. It is possible that some patients had greater systemic compromise, more severe disease, or had received dissimilar peri-operative management. Our findings suggest that, at least in the sub-population of cardiac surgery patients, the first two factors may be important.

In this study, the risk of SSI increased nearly four-fold if a blood transfusion had been given, and three-fold if the patient was either known to be a diabetic or obese. Smoking status and absolute blood glucose values and changes at various time points were not associated with the subsequent development of an SSI.

Although blood transfusions are regarded as essential in many surgical procedures, they carry significant risks, including: transfusion reactions, receipt of infectious agents, contaminated red blood cells,[8] and compromised immunity.[12] The pro-inflammatory effects of transfused blood can mediate tissue destruction. Our finding related to the association between blood transfusion and SSIs is in agreement with other recent reports that it is an independent risk factor for postoperative bacterial infection (odds ratio=1.18, P=0.007), SSI, and post-injury multiple-organ failure.[13-15] The risk from blood transfusions does not appear to be related to the type of surgery, as the association has been described in cholecystectomy patients (7.7 times greater risk after ≥4 units of blood).[16] It is also present in colorectal cancer sufferers, as well as those having major pulmonary resections,[17] ileal-pouch anal anastomoses,[18] oral cavity cancer surgery,[19] bowel surgery,[20] and cardiac surgery.[21]

Interestingly, the occurrence of wound infections appears to increase incrementally with each unit of blood transfused,[22] and is also greater if the blood has been in storage for 21 days or more[23] or has been depleted of nitric oxide. An individual's disease process and systemic compromise are likely to provide an intermediate link with these factors in the causal pathway to SSIs.

The length of hospital stay is a variable commonly used as an outcome of efficacy. Although it was not measured in this study, cardiac surgery patients who received more than 4 units of blood have been reported to stay significantly longer in ICUs than those who were not transfused (6.1±7.2 days vs 3.7±2.8 days, respectively; P<0.01).[21] This finding may be confounded by the severity of cardiac disease and the associated co-morbidities that precipitate haemorrhage.

Within our institution, one of the initiatives

TABLE 3. Association of exposure with surgical site infection (SSI)

Variable*	SSI	Non-SSI	Odds ratio (interquartile range)	P value
Known diagnosis: diabetes mellitus				
Yes	17	9	2.770 (0.978-7.845)	0.052†
No	15	22		
Obesity (BMI ≥25 kg/m²)				
Yes	18	10	2.70 (0.967-7.541)	0.055†
No	14	21		
Blood transfusion in hospital				
Yes	21	11	3.818 (1.332-10.942)	0.011†
No	10	20		
Smoking				
Yes	17	17	1.00 (0.368-2.719)	1.00†
No	14	14		
ASA score				
1	0	1	-	0.559†
2	1	2		
3	27	19		
4	3	2		
Preoperative mean blood glucose >8.0 mmol/L				
No	23	28	0.274 (0.066-1.131)	0.062†
Yes	9	3		
Intra-operative mean blood glucose >8.0 mmol/L				
No	11	14	0.636 (0.230-1.757)	0.382†
Yes	21	17		
Recovery mean blood glucose >8.0 mmol/L				
No	4	9	0.254 (0.067-0.959)	0.036†
Yes	28	16		
Recovery peak blood glucose >8.0 mmol/L				
No	3	5	0.414 (0.089-1.931)	0.280‡
Yes	29	20		
ICU mean blood glucose >8.0 mmol/L				
No	4	6	0.452 (0.112-1.821)	0.380‡
Yes	28	19		
ICU peak blood glucose >8.0 mmol/L				
No	3	2	1.190 (0.183-7.727)	1.000‡
Yes	29	23		
HDU mean blood glucose >8.0 mmol/L				
No	5	3	2.222 (0.442-11.18)	0.434‡
Yes	12	16		
HDU peak blood glucose >8.0 mmol/L				
No	1	0	--	0.472‡
Yes	16	19		
Blood glucose >8.0 mmol/L at any one time point				
No	1	0	-	1.000‡
Yes	31	28		
Surgery duration >3 hours				
Yes	16	15	1.067 (0.397-2.865)	0.898†
No	16	16		

* BMI denotes body mass index, ASA American Society of Anesthesiologists, ICU intensive care unit, and HDU high dependency unit
† Chi squared test
‡ Fisher's exact test

supported by the infection control service was comprehensive peri-operative blood glucose control. Following in-service presentations from this unit, surgeons are becoming aware of the association between high blood glucose levels in diabetic patients and the risk of SSIs. However, to date no consensus protocol for managing blood glucose in non-diabetic patients has been established. During the cardiopulmonary bypass phase of cardiac surgery, blood glucose levels can increase by 20% in non-diabetics and fall by 10% in known diabetic patients.[24] An increase of only 1 mmol/L of blood glucose during surgery has been associated with a greater risk of postoperative complications.[25]

Diabetes mellitus is a prevalent condition in Hong Kong. The age-adjusted prevalence (known and unknown) for the 35-64 age-group was 9.7% in men and 10.6% in women in 1995.[26] Among Hong Kong's elderly (aged 65-74 years), the prevalence increases sharply to 21.7% in men and 29.3% in women.[26] Previous studies have shown that nearly 50% of patients with DM are undiagnosed.[27,28] In our study, 99% of the non-diabetic patients manifested glycaemic dysfunction at some time point during or after surgery. Blood glucose levels are expected to fluctuate, but if glucose regulatory systems are intact, they should not extend beyond 3 to 7 mmol/L despite prolonged fasting or extreme stress. At-risk patients, notably those who carry genetic factors for DM, may be expected to decompensate during surgery.

It is not routine practice in our institution to include glucose tolerance testing or determine haemoglobin A_{1c} (to seek out glycaemic dysfunction) during the preoperative work-up. It is possible that due to the cardiovascular disease risk factors, carrying out baseline tests would increase the proportion of known diabetic patients entering cardiac surgery theatres. Thus, the association between known DM and SSI (marginally significant in our study) may be stronger in a larger patient group whose true diabetic status was known. Indeed, DM has previously been identified as a risk factor for the development of deep SWIs following cardiac surgery, with a 2.5-fold increased risk.[5]

The increased infection risk in diabetics can be attributed to the impaired neutrophil chemotaxis, phagocytosis, the adherence and glycosylation of collagen matrix proteins, all of which lead to weakened antibacterial defenses and delayed wound healing.[29] However, a recent study reported similar rates of deep SWIs in both diabetic and non-diabetic cardiothoracic patients.[10] Moreover, the level of glucose dysfunction and its control at the point of surgery and in the postoperative period are highly predictive of wound infection.[6,30] Studies of ICU patients have demonstrated significantly fewer SSIs when the blood glucose level is maintained below 7 mmol/L.[31]

The current study found obesity to be associated with the development of SSIs. Although a link with SWI was recently reported in a large cohort of cardiac patients,[32] there is also a clear causal link between DM and being overweight. This study did not control for body weight, as it would have compromised the investigation of DM as an exposure of interest.

The fact that smoking status was not associated with SSIs in our sample of cardiac patients was surprising. If the classification had been based on current smoking status, then it would have been expected that patients with known disease may have ceased smoking in an effort to reduce their risk of death. However, we compared 'ever smoked' with 'never smoked' and were unable to find any association.

The current study has the obvious limitation of being small and covering a short period. This was necessary to capture the incidence of SSIs in the cardiac surgery population, before procedural changes were implemented. Several variables have become more tightly controlled since 2006. Preoperative showering has become a three-stage process instead of a one-shower event. The intra-operative antibiotic regimen has been revised so that drugs are re-administered after 4 hours of surgery to provide greater cover during long procedures. Following an audit of blood product use, there has been a significant reduction in the number of blood transfusions in cardiac surgery patients; now 58% of patients receive blood versus 73% previously ($P<0.002$). These changes have coincided with a marked reduction in the incidence of SSIs in this patient group.

A further drawback was that our findings cannot be generalised to other types of surgery, due to the intermediary link in the causal pathway between co-morbidities and coronary disease. The logical progression of this audit is to extend it across the various divisions of surgery and to evaluate the risk factors for SSI in the wider surgical population more comprehensively. It is well known, for example, that procedures that involve anastomosis are more often associated with wound infection than cleaner procedures such as CABG.

Similarly, studies of stress hyperglycaemia have primarily reported its incidence and association with morbidity and mortality in cardiac surgery or acutely ill patients admitted to ICUs, rather than in general surgery patients.[5,8,25,30,33-36] Future work should collect routine glucose tolerance data from all surgical patients, to allow a more accurate DM classification and facilitate and unravel the role of glycaemic dysregulation in all types of surgery.

Conclusions

The costs of surgical wound infections in terms of prolonged hospital stays, increased medical expenses, and impaired quality of life are enormous. This audit identified an association between SSI and pre-existing diabetes and receipt of a blood transfusion but could not determine the direction of causality. The findings justify a review of available evidence and scrutiny of blood glucose and blood transfusion practices during all major surgical procedures.

Acknowledgements

The authors wish to thank Professors J Chan and PSA Kong from the Department of Medicine and Therapeutics at the Chinese University of Hong Kong for their assistance and interest in the project.

Declaration

This study was funded and materially supported by the Department of Surgery of the Chinese University of Hong Kong.

References

1. Cheadle WG. Risk factors for surgical site infection. Surg Infect (Larchmt) 2006;7(Suppl 1):7S-11S.
2. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol 1999;20:250-78.
3. Cardiac surgery report. Prince of Wales Hospital and Chinese University of Hong Kong website: http://www.surgery.cuhk.edu.hk/cardiothoracic/default.htm. Accessed Apr 2008.
4. Gummert JF, Barten MJ, Hans C, et al. Mediastinitis and cardiac surgery—an updated risk factor analysis in 10,373 consecutive adult patients. Thorac Cardiovasc Surg 2002;50:87-91.
5. Robinson PJ, Billah B, Leder K, Reid CM; ASCTS Database Committee. Factors associated with deep sternal wound infection and haemorrhage following cardiac surgery in Victoria. Interact Cardiovasc Thorac Surg 2007;6:167-71.
6. Shroyer AL, Coombs LP, Peterson ED, et al. The Society of Thoracic Surgeons: 30-day operative mortality and morbidity risk models. Ann Thorac Surg 2003;75:1856-65.
7. Perencevich EN, Sands KE, Cosgrove SE, Guadagnoli E, Meara E, Platt R. Health and economic impact of surgical site infections diagnosed after hospital discharge. Emerg Infect Dis 2003;9:196-203.
8. Mauermann WJ, Nemergut EC. The anesthesiologist's role in the prevention of surgical site infections. Anesthesiology 2006;105:413-21.
9. Bhatia JY, Pandey K, Rodrigues C, Mehta A, Joshi VR. Postoperative wound infection in patients undergoing coronary artery bypass graft surgery: A prospective study with evaluation of risk factors. Indian J Med Microbiol 2003;21:246-51.
10. Schmeltz LR, DeSantis AJ, Thiyagarajan V, et al. Reduction of surgical mortality and morbidity in diabetic patients undergoing cardiac surgery with a combined intravenous and subcutaneous insulin glucose management strategy. Diabetes Care 2007;30:823-8.
11. Woodfield JC, Beshay NM, Pettigrew RA, Plank LD, van Rij AM. American Society of Anesthesiologists classification of physical status as a predictor of wound infection. ANZ J Surg 2007;77:738-41.
12. Offner PJ. Age of blood: does it make a difference? Crit Care 2004;8(Suppl 2):24S-26S.
13. Kaya E, Yetim I, Dervisoglu A, Sunbul M, Bek Y. Risk factors for and effect of a one-year surveillance program on surgical site infection at a university hospital in Turkey. Surg Infect (Larchmt) 2006;7:519-26.
14. Chang H, Hall GA, Geerts WH, Greenwood C, McLeod RS, Sher GD. Allogeneic red blood cell transfusion is an independent risk factor for the development of postoperative bacterial infection. Vox Sang 2000;78:13-8.
15. Moore FA, Moore EE, Sauaia A. Blood transfusion. An independent risk factor for postinjury multiple organ failure. Arch Surg 1997;132:620-5.
16. Tang ZY, Guo EQ, Yan HC, Xie QP, Lian JA, Wu YL. Risk factors for infectious morbidity in gallbladder cancer patients treated surgically. Hepatogastroenterology 2007;54:22-7.
17. Harpole DH Jr, DeCamp MM Jr, Daley J, et al. Prognostic models of thirty-day mortality and morbidity after major pulmonary resection. J Thorac Cardiovasc Surg 1999;117:969-79.
18. Madbouly KM, Senagore AJ, Remzi FH, Delaney CP, Waters J, Fazio VW. Perioperative blood transfusions increase infectious complications after ileoanal pouch procedures (IPAA). Int J Colorectal Dis 2006;21:807-13.
19. Liu SA, Wong YK, Poon CK, Wang CC, Wang CP, Tung KC. Risk factors for wound infection after surgery in primary oral cavity cancer patients. Laryngoscope 2007;117:166-71.
20. Walz JM, Paterson CA, Seligowski JM, Heard SO. Surgical site infection following bowel surgery: a retrospective analysis of 1446 patients. Arch Surg 2006;141:1014-8.
21. Leal-Noval SR, Rincón-Ferrari MD, García-Curiel A, et al. Transfusion of blood components and postoperative infection in patients undergoing cardiac surgery. Chest 2001;119:1461-8.
22. Banbury MK, Brizzio ME, Rajeswaran J, Lytle BW, Blackstone EH. Transfusion increases the risk of postoperative infection after cardiovascular surgery. J Am Coll Surg 2006;202:131-8.
23. Mynster T, Nielsen HJ. The impact of storage time of transfused blood on postoperative infectious complications in rectal cancer surgery. Danish RANX05 Colorectal Cancer Study Group. Scand J Gastroenterol 2000;35:212-7.
24. Smith CE, Styn NR, Kalhan S, et al. Intraoperative glucose control in diabetic and nondiabetic patients during cardiac surgery. J Cardiothorac Vasc Anesth 2005;19:201-8.
25. Gandhi GY, Nuttall GA, Abel MD, et al. Intensive intraoperative insulin therapy versus conventional glucose management during cardiac surgery: a randomized trial. Ann Intern Med 2007;146:233-43.
26. Janus ED, Watt NM, Lam KS, et al. The prevalence of diabetes, association with cardiovascular risk factors and implications of diagnostic criteria (ADA 1997 and WHO 1998) in a 1996 community-based population study in Hong Kong Chinese. Hong Kong Cardiovascular Risk Factor Steering Committee. American Diabetes Association. Diabet Med 2000;17:741-5.
27. Cockram CS, Woo J, Lau E, et al. The prevalence of diabetes mellitus and impaired glucose tolerance among Hong Kong Chinese adults of working age. Diabetes Res Clin Pract 1993;21:67-73.
28. Ko GT, Cockram CS, Chan JC. How to minimize missing

those subjects with high 2HR plasma glucose but 'normal' fasting plasma glucose levels? J Med 2001;32:53-65.
29. Talbot TR. Diabetes mellitus and cardiothoracic surgical site infections. Am J Infect Control 2005;33:353-9.
30. Furnary AP, Zerr KJ, Grunkemeier GL, Starr A. Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. Ann Thorac Surg 1999;67:352-62.
31. Grey NJ, Perdrizet GA. Reduction of nosocomial infections in the surgical intensive-care unit by strict glycemic control. Endocr Pract 2004;10(Suppl 2):46S-52S.
32. Salehi Omran A, Karimi A, Ahmadi SH, et al. Superficial and deep sternal wound infection after more than 9000 coronary artery bypass graft (CABG): incidence, risk factors and mortality. BMC Infect Dis 2007;7:112.
33. Cammu G, Lecomte P, Casselman F, et al. Preinduction glycemia and body mass index are important predictors of perioperative insulin management in patients undergoing cardiac surgery. J Clin Anesth 2007;19:37-43.
34. Rady MY, Johnson DJ, Patel BM, Larson JS, Helmers RA. Influence of individual characteristics on outcome of glycemic control in intensive care unit patients with or without diabetes mellitus. Mayo Clin Proc 2005;80:1558-67.
35. Finney SJ, Zekveld C, Elia A, Evans TW. Glucose control and mortality in critically ill patients. JAMA 2003;290:2041-7.
36. Collier B, Diaz J Jr, Forbes R, et al. The impact of a normoglycemic management protocol on clinical outcomes in the trauma intensive care unit. JPEN J Parenter Enteral Nutr 2005;29:353-9.