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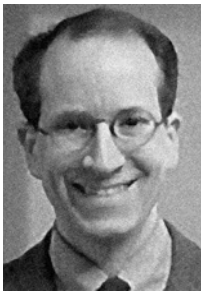
Intraoperative and Postoperative Variables Associated with Strokes following Cardiac Surgery



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ABSTRACT

Background: Strokes are a devastating complication of coronary artery bypass grafting (CABG) surgery. Previous work from 1992 to 2000 determined the principal mechanism of strokes occurring secondary to CABG. In the present study, we quantified the association between intraoperative and postoperative variables and stroke mechanisms while adjusting for patient and disease characteristics.

Methods: We conducted a prospective study of 13,897 patients who underwent isolated CABG in northern New England from 1992 to 2000. Data were collected on patient and disease characteristics, intraoperative and postoperative care, and outcomes. Strokes were classified as embolic, hypoperfusion, and mixed (hemorrhage, lacunar, thrombotic, other, multiple, and unclassified). We quantified the association between the intraoperative and postoperative treatment and course variables and the stroke mechanism while adjusting for patient and disease characteristics. Patients without strokes served as the reference group for the determination of odds ratios (OR).

Results: Variables associated with embolic strokes included cardiopulmonary bypass time greater than 2 hours versus less than 1 hour (OR, 1.5; *ptrend* .03) and postoperative atrial fibrillation (OR, 2.4; $P < .001$). The risk of hypoperfusion strokes was increased with the duration of cardiopulmonary bypass (OR, 6.4; *ptrend* .01) and postoperative atrial fibrillation (OR, 5.4; $P < .001$). Postoperative atrial fibrillation was associated with the risk of mixed strokes (OR, 1.7; $P = .04$).

Conclusions: After we adjusted for preoperative factors, postoperative atrial fibrillation and increasing duration of cardiopulmonary bypass remained significant predictors of embolic and hypoperfusion strokes, although to differing degrees. Prevention and management of atrial fibrillation and avoidance of prolonged exposure to extracorporeal circulation may offer leverage areas for the improvement of stroke outcomes.

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INTRODUCTION

Stroke is an uncommon yet devastating complication of coronary artery bypass grafting (CABG) surgery. Reported rates of stroke have ranged from 1.3% to 4.3% [Gonzalez-Scarano 1981, Gardner 1985, Jones 1991, Blossom 1992, Frye 1992, Lynn 1992, Ricotta 1995]. Most research to date has focused on the identification of preoperative risk factors. We recently studied the principal mechanisms of 388 strokes secondary to CABG and found that 62% were embolic and an additional 9% were hypoperfusion [Likosky 2003b]. Few published reports have focused on the associations between intraoperative and postoperative variables and a patient's risk of different stroke mechanisms.

Information concerning stroke mechanisms is inherently limited if the intraoperative and postoperative risk factors associated with each mechanism are not identified and understood. It is plausible that each mechanism has its own unique risk factors, which are in turn linked to events in a patient's intraoperative and postoperative care and course. Any substantial reduction in stroke risk would therefore necessitate understanding the relationship between mechanism and intraoperative and postoperative factors while adjusting for patient and disease characteristics.

We conducted a regional cohort study of patients who underwent isolated CABG surgery in northern New Eng-

land. The purpose of this study was to quantify the association between intraoperative and postoperative variables and a patient's risk of different stroke mechanisms while adjusting for preoperative characteristics. This study provides the foundation for a regional quality improvement effort with respect to stroke and CABG surgery.

MATERIALS AND METHODS

Setting

This study was conducted by the Northern New England Cardiovascular Disease Study Group (NNECDSG). The NNECDSG is a voluntary regional consortium representing all medical centers in Maine, New Hampshire, and Vermont and 1 medical center in Massachusetts that perform CABG surgery. Since its inception in 1987, the NNECDSG has maintained prospective surgical registries on all patients undergoing CABG, heart valve replacement, and percutaneous coronary interventions. The group fosters continuous improvement in the quality of care for patients undergoing these operations through the pooling of process and outcome data and providing timely feedback of these data back to clinicians through group meetings and region-, center-, and surgeon-specific reports [O'Connor 1996].

Institutional review board approval was achieved at each participating medical center.

Study Design

This regional prospective study comprises 13,897 patients who underwent isolated CABG surgery between April 1992 and June 2000. Details of our data collection have previously been reported [O'Connor 1992]. We collected data on procedural characteristics (use of an intra-aortic balloon pump [IABP], duration of cross-clamping, duration of cardiopulmonary bypass, and low cardiac output syndrome [return to cardiopulmonary bypass or use of an intraoperative or postoperative IABP]) and postoperative outcomes (use of ≥2 inotropes at 48 hours, atrial fibrillation, and reoperation for bleeding). To study a relatively homogeneous surgical procedure, we excluded patients who underwent off-pump CABG and patients who underwent CABG associated with heart valve surgery, resection of ventricular aneurysm, or any other surgical procedure.

We defined a stroke as a new focal neurologic deficit occurring during or following the CABG procedure that appeared and was established prior to discharge and was still at least partially evident more than 24 hours after its onset.

Classification System

This study relies on the development and implementation of the NNECDSG stroke classification system [Likosky 2003b]. A listing of stroke mechanisms and operational definitions for each are listed in the "Appendix." Using clinical end-point committees staffed with cardiothoracic surgeons, cardiovascular anesthesiologists, stroke neurologists, and neuroradiologists and an extensive review of medical records, we divided strokes into 2 principal mechanisms: hemorrhagic and ischemic. We further divided ischemic strokes into 2 subcategories: thromboembolism (embolic or some type of in situ clot) and hypoperfusion.

We previously reported that 62.1% of strokes that occurred after isolated CABG surgery were secondary to embolism, whether carotid, aortic, or cardiac in source [Likosky 2003b]. Because of the dominance of this embolic mechanism, we collapsed the classification system into 3 groups: embolic, hypoperfusion, and mixed (hemorrhage, lacunar, thrombotic, other, multiple, and unclassified).

Analysis

Rates of stroke subtypes were calculated across preoperative, intraoperative, and postoperative risk factors by dividing the number of strokes identified in each subgroup by the total number of patients in that subgroup. Rates were displayed as events per 1000 patients.

Multinomial logistic regression (MLR) with the Stata 7.0 statistical software package (StataCorp, College Station, TX, USA) was used to quantify the association between stroke mechanisms and patient and disease characteristics, intraoperative and postoperative care, and outcomes. MLR allows the analyst to model the log-odds of a nominal (any positive integer) event's occurrence. MLR offers an advantage, stemming from its relative efficiency, compared with traditional multivariate logistic regression. It allows one to make direct comparisons across stroke mechanisms instead of creating multiple multivariate logistic prediction models. In the current study, comparisons were made between a patient's risk of stroke by an embolic, hypoperfusion, or mixed mechanism and patients who did not have strokes.

In previous work, we created both preoperative and intraoperative/postoperative prediction models of strokes after CABG surgery [Charlesworth 2003, Likosky 2003a]. In our preoperative model, we identified the following patient and disease characteristics to be significantly associated with stroke: age, sex, diabetes, vascular disease, acuity, and ejection fraction. In our latter model once we adjusted for the aforementioned preoperative risk factors, the following factors remained significantly associated with the risk of stroke: atrial fibrillation, duration of cardiopulmonary bypass, and low cardiac output syndrome. In the present study, we adjusted for factors in our preoperative model while we used the intraoperative and postoperative factors as independent variables in our current MLR prediction model.

RESULTS

Univariate Analysis

Rates of stroke subtypes across the intraoperative and postoperative treatment and course variables are summarized in Table 1. Rates of embolic strokes increased with increasing duration of cardiopulmonary bypass, increasing cross-clamp duration, use of an intraoperative or postoperative IABP, incidence of low cardiac output, postoperative atrial fibrillation, postoperative myocardial infarction, and any return to cardiopulmonary bypass. Rates of hypoperfusion strokes increased with increasing duration of cardiopulmonary

Table 1. Associations between the Intraoperative or Postoperative Treatment and Course and the Risk of Stroke Subtype (Rates per 1000 Patients)

Variables	Embolic	Hypoperfusion	Mixed
Cardiopulmonary bypass duration			
<60 min	6.55	0.50	2.52
60-89 min	7.13	0.64	4.02
90-119 min	10.80	1.58	4.08
≥120 min	13.63	3.56	6.82
Cross-clamp duration			
<45 min	13.10	1.31	6.93
45-64 min	14.52	2.12	6.36
≥65 min	14.39	2.71	6.67
Intraoperative or postoperative intra-aortic balloon pump			
No	8.85	1.10	4.01
Yes	12.59	8.99	10.79
Low cardiac output at 48 h			
No	8.68	1.03	4.05
Yes	17.33	9.33	8.00
Postoperative atrial fibrillation			
No	9.39	0.66	4.46
Yes	26.02	4.44	8.57
Postoperative myocardial infarction			
No	7.96	0.81	3.83
Yes	10.00	1.77	4.51
Return to bypass			
No	7.18	1.06	4.00
Yes	23.62	7.87	3.94

bypass, increasing cross-clamp duration, use of an intraoperative or postoperative IABP, incidence of low cardiac output, use of CABG surgery, postoperative atrial fibrillation, postoperative myocardial infarction, and any return to cardiopulmonary bypass. Rates of mixed-stroke etiologies increased with increasing duration of cardiopulmonary bypass, use of an intraoperative or postoperative IABP, incidence of low cardiac output, postoperative atrial fibrillation, and postoperative myocardial infarction.

Multinomial Logistic Regression

The reference group for the remainder of our analyses consists of patients who did not have a stroke. Associations between intraoperative and postoperative factors and risk of stroke via different mechanisms are summarized in Table 2. Compared with patients without atrial fibrillation, postoperative atrial fibrillation was associated with a 2.8-fold increased risk of embolic strokes, a 6.9-fold increased risk of hypoperfusion strokes, and a 2.0-fold increased risk of mixed strokes, versus patients who did not have strokes. A cardiopulmonary bypass duration greater than 2 hours (versus less than 1 hour) was associated with a 2-fold increased risk of embolic stroke (*ptrend*, .002), a 7-fold increased risk of hypoperfusion stroke (*ptrend*, <.001), and a nearly 3-fold increased risk of mixed strokes (*ptrend*, .003). The occurrence of low cardiac output syndrome was associated with a 2.0-fold increased risk of embolic strokes, a 9.3-fold increased risk of hypoperfusion strokes, and a 2.0-fold increased risk of mixed strokes.

We used MLR to test the association between stroke subtypes and the variables from Table 2 while adjusting for patient and disease characteristics. The regression model significantly (χ^2_{18} = 212.55; P < .001) predicted the occurrence of stroke subtypes (Table 3). For embolic strokes, we found that postoperative atrial fibrillation was associated with a 2.4-fold increased risk, cardiopulmonary bypass greater than 2 hours was associated with a 1.5-fold increased risk, and the occurrence of low cardiac output syndrome was associated with a 1.6-fold increased risk. For hypoperfusion strokes, we found that postoperative atrial fibrillation was associated with a 5.4-fold increased risk, cardiopulmonary bypass greater than 2 hours was associated with a 6.4-fold increased risk, and the occurrence of low cardiac output syndrome was associated with a 2.6-fold increased risk. For mixed strokes, we found that postoperative atrial fibrillation was associated with a 1.7-fold increased risk, cardiopulmonary bypass greater than 2 hours was associated with a 1.9-fold increased risk, and the occurrence of low cardiac output syndrome was associated with a 1.4-fold increased risk.

DISCUSSION

In this regional prospective study of patients who underwent isolated CABG surgery, we quantified the association

Table 2. Univariate Associations between the Intraoperative and Postoperative Treatment and Course and the Stroke Subtypes*

	Embolic		Hypoperfusion		Mixed	
Variables	Odds Ratio (95% CI)	*P*	Odds Ratio (95% CI)	*P*	Odds Ratio (95% CI)	*P*
Atrial fibrillation, postoperative	2.84 (2.13, 3.80)	<.001	6.86 (2.80, 16.83)	<.001	1.97 (1.24, 3.14)	.004
Cardiopulmonary bypass duration						
<60 min	Reference		Reference		Reference	
60-89 min	1.09 (0.70, 1.71)	.697	1.27 (0.26, 6.14)	.762	1.60 (0.81, 3.19)	.179
90-120 min	1.66 (1.07, 2.59)	.025	3.16 (0.71, 14.13)	.132	1.64 (0.80, 3.34)	.178
≥120 min	2.12 (1.30, 3.43)	.002	7.18 (1.60, 32.12)	.010	2.75 (1.31, 5.79)	.008
	ptrend	.002	*ptrend*	.000	*ptrend*	.003
Low cardiac output syndrome	2.04 (1.16, 3.59)	.013	9.25 (4.00, 21.38)	<.001	2.02 (0.88, 4.62)	.095

*Reference group consists of patients not experiencing strokes. CI indicates confidence interval.

Table 3. Multivariate Associations between the Intraoperative and Postoperative Treatment and Course and the Stroke Subtypes*

Variables	Embolic		Hypoperfusion		Mixed	
	Odds Ratio (95% CI)	*P*	Odds Ratio (95% CI)	*P*	Odds Ratio (95% CI)	*P*
Atrial fibrillation, postoperative	2.38 (1.77, 3.21)	<.001	5.42 (2.15, 13.70)	<.001	1.65 (1.03, 2.66)	.039
Cardiopulmonary bypass duration						
<60 min	Reference		Reference		Reference	
60-89 min	0.87 (0.53, 1.42)	.574	1.33 (0.15, 11.50)	.793	1.28 (0.59, 2.77)	.537
90-120 min	1.18 (0.72, 1.92)	.515	2.79 (0.35, 22.53)	.334	0.93 (0.40, 2.15)	.864
≥120 min	1.45 (0.84, 2.51)	.181	6.40 (0.79, 51.96)	.082	1.91 (0.81, 4.53)	.140
	ptrend	.032	*ptrend*	.005	*ptrend*	.328
Low cardiac output syndrome	1.62 (0.84, 3.16)	.153	2.62 (0.57, 12.02)	.215	1.44 (0.51, 4.07)	.494

*Data adjusted for age, sex, diabetes, vascular disease, creatinine ≥2 mg/dL or renal failure, acuity, and ejection fraction. Reference group consists of patients not experiencing strokes. CI indicates confidence interval.

between intraoperative and postoperative factors and the risk of different stroke mechanisms after adjusting for patient and disease characteristics. Our final prediction model uses readily obtainable preoperative, intraoperative, and postoperative variables and assigns an independent weight to each variable to provide quantitative information about the risk of different stroke mechanisms. By accounting for patient variability, the model provides a basis for tracking the outcomes of cardiac surgery programs. It may be useful for understanding the effects of treatment and course variables on stroke mechanisms. Clinicians may base their assessment of a patient's risk of developing a stroke by using our findings as a starting point for altering a patient's treatment and course.

Duration of Cardiopulmonary Bypass

We found a nearly 1.5-fold increase in risk of embolic strokes for patients who had cardiopulmonary bypass times lasting greater than 2 hours relative to the risk for patients who had cardiopulmonary bypass durations of less than 60 minutes. We believe that this variable may serve as a proxy for a difficult surgical case for the risk of both embolic and hypoperfusion strokes. Others have reported a risk of emboli with increasing duration of bypass. Brown et al, in an extensive study of brain specimens from patients who underwent CABG and valve-replacement surgery, found that total embolic load increased 90.5% for every hour of cardiopulmonary bypass [Brown 2000]. Although Brown et al did not comment on the length of cross-clamp time, these 2 variables are highly correlated in our data set ($r = 0.74$; $P < .001$).

We found a 30% increased risk of hypoperfusion stroke for patients who had a prolonged duration of cardiopulmonary bypass. We feel that this variable may be a proxy for a difficult surgical case. In addition, a prolonged cardiopulmonary bypass may be associated with a patient's inability to produce sufficient cardiac output. These patients are more prone to developing a hypoperfusion stroke.

Gardner et al found that patients who had severe hypotension had an 8.4-fold increased risk of stroke, whereas patients who had bypass times greater than 130 minutes had a 3.6-fold increased risk of stroke [Gardner 1985]. Controversy exists regarding the appropriate level of the mean arterial pressure for patients who undergo CABG surgery [Cartwright 1998, Hartman 1998]. In the current study, mean arterial pressure was lower for patients who had hypoperfusion strokes than for those who had embolic strokes, both on arrival in the intensive care unit (55.9 mm Hg versus 69.8 mm Hg; $P = .20$) and at 8 hours postoperatively (51.8 mm Hg versus 62.7 mm Hg; $P = .30$).

Low Cardiac Output

Evidence suggests that low cardiac output is a precursor to the development of hypoperfusion strokes, as seen in the work of Mathias [2000]. Surgenor et al described a regression model developed and used by the NNECDSG to predict the occurrence of fatal low cardiac output secondary to isolated CABG surgery [Surgenor 2001]. Reducing preload to patients with preexisting vascular disease may make them more susceptible to strokes because of the inadequate supply of oxygen to these territories during times of decreased arterial blood supply. These strokes result in a unique clinical pattern and presentation as well as a unique vascular lesion (ie, watershed).

More than 75% of our patients who had hypoperfusion strokes had hypotension or low cardiac output syndrome. We believe that this variable more accurately captures the risk associated with this disease state because patients with low cardiac output syndrome are more likely to have hypotension prior to cardiopulmonary bypass, an IABP (whether intraoperatively or postoperatively), and a need to return to cardiopulmonary bypass. An increased understanding of the factors leading to this diseased state, as well as of the management techniques for patients already experiencing low cardiac output, will certainly benefit this subgroup of patients.

Atrial Fibrillation

We found an association between postoperative atrial fibrillation and embolic strokes. Our experience is similar to that reported by Creswell et al for a series of 4507 patients who underwent CABG, mitral or aortic valve repair, or heart transplantation [Creswell 1993]. Patients were monitored continuously for arrhythmias until the time of discharge. Creswell et al found a stroke in 3.3% of the patients who had postoperative atrial fibrillation, whereas they found a stroke

in 1.4% of patients without arrhythmias. Although this study included patients who underwent surgeries other than isolated CABG, we found similar rates of atrial fibrillation in our study. In our surgical registry, we reported stroke rates of 2.63% for patients with and 1.18% for patients without a new onset of atrial fibrillation. We are confident that the association between embolic strokes and atrial fibrillation is an underestimate and that this underestimate reflects insufficient information regarding the timing of 27.7% of postoperative atrial fibrillation cases thought to be attributable to stroke mechanism. In our work classifying the etiology of these strokes, we found that atrial fibrillation was the primary mechanism for 24% of the patients who had embolic strokes.

In the present study, atrial fibrillation was also associated with hypoperfusion strokes. Atrial fibrillation adversely affects the coordination between the atrium and the ventricle, resulting in insufficient cardiac output, and consequently may increase a patient's risk of a hypoperfusion stroke. The deleterious effect of a new onset of atrial fibrillation may be magnified with preexisting low cardiac output syndrome. Our observed association of atrial fibrillation with the estimated risk of hypoperfusion strokes may be unstable and therefore may be an overestimate because of our low incidence (9% of all strokes) of this stroke subtype.

We acknowledge the following limitations in this study. We had insufficient observations for some stroke mechanisms to quantify substantially their association with potential risk factors. Because of this fact, we maximized our statistical power by collapsing our strokes into the 3 dominant categories (embolic, hypoperfusion, and mixed). In addition, we acknowledge that the mixed-stroke category represents combinations of many stroke mechanisms. Consequently, limited insight may be gleaned from the study of the intraoperative and postoperative factors associated with the occurrence of these strokes. The findings from this study, however, represent our experience of nearly 14,000 consecutive CABG operations. We realize that our findings should not be driven by *P* values but rather by formative judgments regarding the association between different stroke mechanisms and intraoperative and postoperative treatment and course variables. These judgments are based on pathophysiology and evidence supported in the medical literature. Lastly, this study lacks information regarding the timing of stroke mechanisms with respect to the new onset of postoperative atrial fibrillation and low cardiac output syndrome. Although this information would be useful, we are confident that any such misclassification would be randomly distributed among all stroke mechanisms and thus would serve only to underestimate our findings. We acknowledge that although work done by others has demonstrated that the management and extent of aortic disease are associated with the risk of stroke, we do not have reliable information among our patients concerning the management or the extent of aortic disease [Gardner 1985, Barbut 1997]. In addition, we did not have consistent data from echocardiograms that might have provided information regarding cardiac function and potential cardiac sources of emboli. Using information extracted from the review of medical records of patients who had strokes, we had data concerning the extent of aortic disease for 56 patients who had strokes.

We have quantified the association between intraoperative and postoperative factors and stroke mechanisms. Many of the preoperative, intraoperative, and postoperative factors contributing to embolic strokes (univariately and multivariately) suggest the need for further efforts with respect to the identification and prevention of postoperative atrial fibrillation and the development of management strategies for vasculopathy as well as for atherosclerotic aortas (using epiaortic probes or transesophageal echocardiography to map areas for cannulation that are free from atherosclerosis or shortening cross-clamp and cardiopulmonary bypass durations). We suspect that clinicians may better serve their patients at risk for hypoperfusion strokes by preventing and treating low output syndrome. Although we have identified areas of opportunity and further research concerning individual stroke subtypes, we realize and acknowledge that many of these factors are shared among all stroke mechanisms for a variety of reasons.

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APPENDIX

Stroke: New focal neurologic deficit that appears and is still at least partially evident more than 24 hours after its onset, occurring during or following the CABG procedure and established prior to discharge.

Hemorrhage: Intracranial bleeding that may be isolated or occur in other brain structures.

Thromboembolism:

Embolic: Ischemic stroke with 1 of 3 likely sources of thrombus: aortic, cardiac, or carotid identified by documented dysrhythmia, neurology, and/or imaging.

Lacunar: Ischemic stroke with classification determined by either neurologic and/or imaging.

Thrombotic: Ischemic stroke without evidence of embolic or lacunar origins.

Hypoperfusion: Stroke due to a mixture of extracranial stenoses and/or systemic hypotension.

Unclassified: Stroke caused by 2 or more competing mechanisms, or of unknown etiology.

Other: Stroke caused by an identified mechanism not listed above.