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Stroke in surgery of the thoracic aorta: Incidence, impact, etiology, and prevention

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Objectives: To determine the incidence, impact, etiology, and methods for prevention of stroke after surgery of the thoracic aorta.

Methods: A total of 317 thoracic aortic operations on 303 patients (194 male, 109 female) aged 13 to 87 years (mean 61 years) were reviewed. There were 218 procedures on the ascending aorta and arch and 99 on the descending aorta. Of the 218 procedures on the ascending aorta and arch, 86 involved cardiopulmonary bypass, 122 involved deep hypothermic circulatory arrest, 2 involved antegrade cerebral perfusion, and 8 involved "clamp and sew" or left heart bypass. Of the 99 procedures on the descending aorta, 20 involved "clamp and sew," 69 involved left heart or full bypass, and 10 involved deep hypothermic circulatory arrest. A total of 206 cases were elective and 97 were emergency operations.

Results: Twenty-three (7.3%) of 317 patients had a stroke. Fifteen strokes occurred in operations on the ascending aorta and 8 in operations on the descending aorta (6.9% vs 8.1%; $P = .703$). Stroke occurred in 16 (16.5%) of 97 emergency operations and 7 (3.4%) of 206 elective operations ($P = .001$). In the 300 patients surviving the operation, stroke was a significant predictor of postoperative death (9/23 [39.1%] vs 23/277 [8.3%]; $P = .001$). Analysis of operative reports, brain images, and neurologic consultations revealed 15 of the 23 strokes were embolic, 3 were ischemic, 3 hemorrhagic, and 2 indeterminate. Patients with stroke had longer intensive care unit stays (18.4 vs 6.8 days; $P = .0001$), longer times to extubation (12.7 vs 3.8 days; $P < .0012$), longer postoperative stays (31.4 vs 14.3 days; $P = .001$), and decreased age-adjusted survival (relative risk 2.775; $P = .0013$). After implementation of a rigorous antiembolic regimen, both strokes and mortality trended downward.

Conclusions: (1) Stroke complicates surgery of both the ascending and descending thoracic aorta and warrants consideration in decision making. (2) Strokes are largely embolic. (3) Antiembolic measures for particles and air are essential, including gentle aortic manipulation, thorough debridement, transesophageal echocardiography to identify aortic atheromas, carbon dioxide flooding of the field, and (in descending cases) proximal clamp application before initiating femoral perfusion.

Stroke is a devastating complication of surgery of the thoracic aorta. The surgeon must balance the life-threatening sequelae of untreated thoracic aortic disease with the risks associated with surgical repair. Surgeons have often focused on death, bleeding, and paraplegia as the major adverse outcomes of thoracic aortic surgery. This study aims to scrutinize specifically the complication of stroke after aortic surgery.

TABLE 1. Demographic and clinical characteristics

Characteristic	Incidence
Tobacco use	102 (32.2%)
Ethanol use	24 (7.6%)
Prior CVA	35 (11.0%)
COPD	30 (9.5%)
CAD	70 (22.1%)
Obesity	19 (6.0%)
Valvular heart disease	124 (39.1%)
CHF	32 (10.1%)
Prior vascular surgery	13 (4.1%)
Prior AAA	20 (6.3%)
Prior aortic surgery	39 (12.3%)
Prior cardiac surgery	29 (9.1%)
Chronic renal failure	18 (5.7%)
Hypertension	179 (56.5%)
Peripheral vascular disease	19 (6.0%)
Hypercholesterolemia	34 (10.7%)
Diabetes	13 (4.1%)

AAA, Abdominal aortic aneurysm; *CAD*, coronary artery disease; *CHF*, congestive heart failure; *COPD*, chronic obstructive pulmonary disease; *CVA*, cerebrovascular accident.

TABLE 2. Operative details

No. of operations	317
Age (y)	
Range	13-87
Mean	61
Sex	
Male	194 (64.0%)
Female	109 (36.0%)
Type of operation	
Emergency operation	97 (32.0%)
Aortic rupture	55 (17.4%)
Pharmacologic treatment	
Aminocaproic acid	78 (24.6%)
Aprotinin	87 (27.4%)
Barbiturates	45 (14.2%)
Steroids	175 (55.2%)
Location	
Ascending aorta/arch (n = 218)*	
Clamp and sew or left heart bypass†	8
CPB alone	86
Selective antegrade perfusion + CPB	2
DHCA + CPB	122
Descending aorta (n = 99)‡	
Clamp and sew	20
CPB (left heart or full)	69
DHCA	10

CPB, Cardiopulmonary bypass; *DHCA*, deep hypothermic circulatory arrest.
*Mean arrest time was 33.4 minutes at 17.7°C.
†Unusual cases (eg, base of innominate artery injury or distal arch operation).
‡Mean arrest time was 40.9 minutes at 16.2°C.

Most clinical reports on aortic surgical procedures simply state an overall incidence of stroke without specific analysis of this complication.[1-4] Stroke may not be adequately appreciated as a common sequel of descending aortic operations. Additionally, even regarding the ascending aorta, it is often difficult to compare interinstitutional data because of differences in the use of deep hypothermic circulatory arrest, retrograde cerebral perfusion, and antegrade cerebral perfusion during the arrest interval. Our study focuses specifically on the complication of stroke and the specific mechanisms by which the strokes in our patients occurred. The objectives of this study are as follows: (1) to determine the incidence of stroke as a complication of aortic surgery on the basis of data from Yale–New Haven Hospital, (2) to determine the clinical impact these strokes have on early and late survival, (3) to analyze the clinical details of those patients having strokes to determine the causes of these strokes, (4) to give suggestions for prevention of strokes on the basis of the pathologic mechanisms by which they occur, and (5) to analyze the effectiveness of those recommendations.

Methods

Data collection for this study occurred in two steps. In 1999, we retrospectively analyzed 227 consecutive thoracic aortic operations performed on 222 patients from January 1989 through September 1998. All surgical procedures for thoracic aortic disease (aneurysms, dissections, penetrating atherosclerotic ulcers, or intramural hematomas) were included. Reimplantation of the coronary arteries, concomitant coronary revascularization procedures, and resuspension or replacement of the aortic valve were performed as indicated. Cerebrospinal fluid drainage was routinely used in descending aortic operations. After analyzing these patients, we instituted specific antiembolic preventive measures, as described fully in the "Results" section. These measures included gentle aortic manipulation, thorough debridement, transesophageal echocardiography to identify aortic atheromas, flooding of the field with carbon dioxide, proximal clamp application before initiating femoral perfusion (in operations on the descending aorta), and avoidance of left atrial cannulation in cases of atrial fibrillation. Subsequent to these changes, between October 1998 and December 2000, we collected another 90 procedures on 84 patients. This second group is compared with the first to assess the impact of these antiembolic measures.
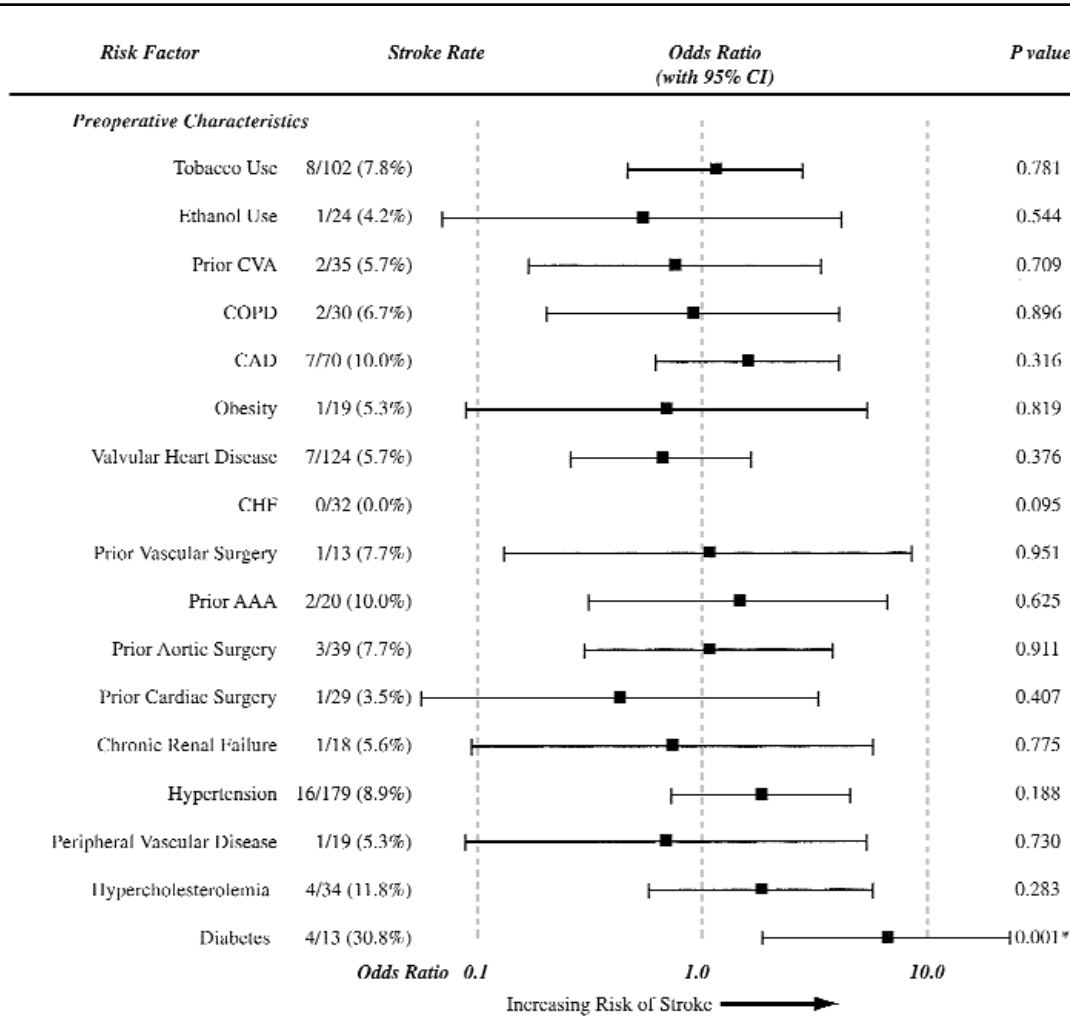
Charts were reviewed and data points collected as listed in Table 1. Our review of 317 operations yielded 303 patients, 194 of whom were male (64.0%) and 109 female (36.0%). Patient age ranged from 13 to 87 years, with a mean age of 61 years. Operative details are summarized in Table 2. No retrograde cerebral perfusion was used in our study. Carbon dioxide flooding was used uniformly in all operations and cerebrospinal fluid drainage was used for operations on the descending aorta. A total of 206 (68.0%) operations were elective and 97 (32.0%) were emergency procedures, with 55 aortic ruptures, defined as free blood in the pericardium or mediastinum. Long-term follow-up was conducted

TABLE 3. Stroke rates

	Overall (n = 317)	Ascending (n = 218)	Descending (n = 99)	Elective (n = 206)*	Emergency (n = 97)*
Overall	23 (7.3%)	15 (6.9%)	8 (8.1%)	7 (3.4%)	16 (16.5%)
		P = .703		*P* = .001	

*The number of elective and emergency cases does not equal the total of 317 cases because inadequate data were available to accurately classify 14 procedures.

TABLE 4. Risk factors for stroke—univariate analysis (χ^2 test) of preoperative risk factors

Risk Factor	Stroke Rate	Odds Ratio (with 95% CI)	P value
Preoperative Characteristics			
Tobacco Use	8/102 (7.8%)		0.781
Ethanol Use	1/24 (4.2%)		0.544
Prior CVA	2/35 (5.7%)		0.709
COPD	2/30 (6.7%)		0.896
CAD	7/70 (10.0%)		0.316
Obesity	1/19 (5.3%)		0.819
Valvular Heart Disease	7/124 (5.7%)		0.376
CHF	0/32 (0.0%)		0.095
Prior Vascular Surgery	1/13 (7.7%)		0.951
Prior AAA	2/20 (10.0%)		0.625
Prior Aortic Surgery	3/39 (7.7%)		0.911
Prior Cardiac Surgery	1/29 (3.5%)		0.407
Chronic Renal Failure	1/18 (5.6%)		0.775
Hypertension	16/179 (8.9%)		0.188
Peripheral Vascular Disease	1/19 (5.3%)		0.730
Hypercholesterolemia	4/34 (11.8%)		0.283
Diabetes	4/13 (30.8%)		0.001*

Odds Ratio   0.1   1.0   10.0

Increasing Risk of Stroke ➤

Bars on graph indicate 95% confidence intervals; odds ratios cannot be calculated when the incidence of disease is zero. *CI,* Confidence intervals; *CVA,* cerebrovascular accident; *COPD,* chronic obstructive lung disease; *CAD,* coronary artery disease; *CHF,* congestive heart failure; *AAA,* abdominal aortic aneurysm; *SCA,* subclavian artery; *IABP,* intra-aortic balloon pump.
*Statistically significant result.

through office visits, telephone interviews, and analysis of office records. In this review, we used operative reports, computed tomographic scans, magnetic resonance imaging, and neurologic consultation reports in combination with neuroradiologic consultation to determine the causes of these strokes.

Univariate and multivariate logistic regression analysis was performed to investigate preoperative and intraoperative predictors of stroke by means of the logistic regression procedure of the SAS System (SAS Institute, Inc, Cary, NC). Preoperative risk factors were stratified according to previously published standards.[5] Univariate analysis of number of days intubated, intensive care unit length of stay, and postoperative length of stay was performed to determine significant differences in clinical outcomes of patients who had a stroke. The Student *t* test was employed for univariate

TABLE 5. Risk factors for stroke—univariate analysis (χ^2 test) of operative risk factors

Risk Factor	Stroke Rate	Odds Ratio (with 95% CI)	P value
Type of Operation			
Operation for Dissection (vs. Aneurysm)	14/112 (12.5%)		0.008*
Operation for Chronic Dissection (vs. Acute)	4/28 (14.3%)		0.774
Operation in Descending/TA (vs. Asc/Arch)	8/99 (8.1%)		0.703
Emergency Operation	16/97 (16.5%)		0.001*
Aortic Rupture	9/55 (16.4%)		0.004*
Most Common Operator	10/182 (5.5%)		0.160
Pharmacologic Treatment			
Barbiturate Use	3/45 (6.7%)		0.869
Aminocaproic Acid Use	4/78 (5.1%)		0.404
Aprotinin Use	9/87 (10.3%)		0.192
Steroid Use	17/175 (9.7%)		0.061
Type of Clamping, Cannulation and Manipulation			
Clamp Proximal to Left SCA (Desc/TA)	2/17 (11.8%)		0.693
"Clamp and Sew" Use	2/20 (10.0%)		0.625
DHCA Use	14/133 (10.5%)		0.100
Arterial Cannulation in Femoral Artery	15/161 (8.5%)		0.925
Arterial Cannulation in Aorta	2/29 (6.9%)		0.724

Odds Ratio   0.1   1.0   10.0

Increasing Risk of Stroke ⟶

Bars on graph indicate 95% confidence intervals; odds ratios cannot be calculated when the incidence of disease is zero. *CI*, confidence intervals; *CVA*, cerebrovascular accident; *COPD*, chronic obstructive lung disease; *CAD*, coronary artery disease; *CHF*, congestive heart failure; *AAA*, abdominal aortic aneurysm; *SCA*, subclavian artery; *IABP*, intra-aortic balloon pump; *DHCA*, deep hypothermic circulatory arrest; *Asc*, ascending; *TA*, thoracoabdominal. *Statistically significant result.

analysis with the use of the analysis of variance procedure of the SAS System. Kaplan-Meier survival analysis was performed and the log-rank calculated; results were also adjusted for age by means of proportional hazards regression, both with the SAS System.

Results
Stroke Incidence
Overall, 23 (7.3%) of 317 operations resulted in cerebrovascular accident, defined as a permanent, new, central neurologic deficit. Spinal cord ischemia was not included in this analysis, because our intent was to focus on discrete lesions of the brain. The incidence of stroke is compared for location of aortic disease and urgency of procedure in Table 3. Tables 4, 5, and 6 illustrate univariate analyses of risk factors predictive of stroke in this population.

In a univariate analysis, preoperative characteristics had little impact on the incidence of stroke (Table 4). Stroke occurred at similar rates in operations on the ascending (6.9%) and descending (8.1%) aorta (Table 3). On the other hand, patients requiring emergency aortic surgery had significantly higher rates of stroke (odds ratio 5.62, 95% confidence intervals 2.23-14.16) than those undergoing elective operations (Tables 3 and 5). Other operative characteristics associated with higher stroke rates included operations for dissection and those for aortic rupture (Table 5). Although the use of deep hypothermic circulatory arrest had no impact on stroke rates, longer arrest times were associated with increased stroke rates (Table 6).

Multivariate logistic regression analysis revealed some additional factors (both preoperative and intraoperative) that
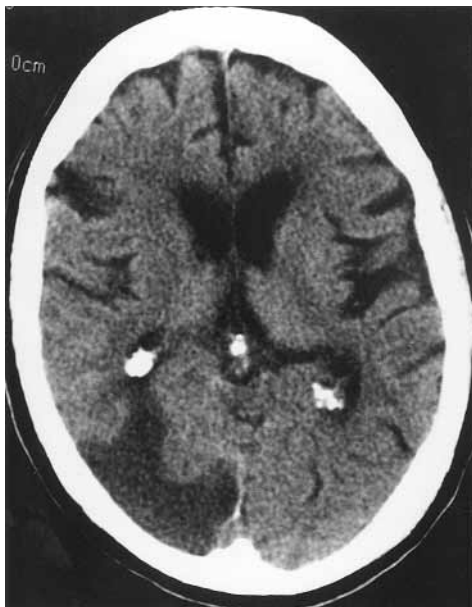
Figure 1. Computed tomographic scan of embolic stroke. Note the hypodense infarct in the right occipital region, with marked loss of brain substance. There is no midline shift.
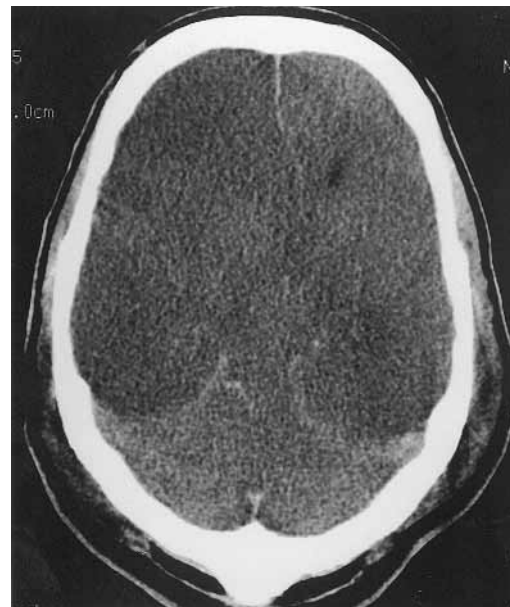

Figure 2. Computed tomographic scan of ischemic stroke. Note the diffuse edema of the entire brain, with obliteration of all cisterns.

increased a patient's risk of stroke during aortic surgery; these included surgery in the descending aorta, emergency surgery, the use of deep hypothermic circulatory arrest, increased use of fresh frozen plasma, and a history of diabetes mellitus (Table 7). None of the other variables from the univariate analysis met the threshold ($P < .30$) for entry into the regression model. In addition, there was no significant association between the location of the proximal clamp in descending aortic surgery or the arterial cannulation site and the incidence of stroke.

The surgeon performing the procedure had no statistically significant effect on stroke incidence but did influence the mortality of the procedure ($P = .001$) (Table 8). Overall 30-day mortality was 49 (15.5%) deaths in 317 operations, but for the surgeon who performed the most procedures (182), the 30-day mortality rate was only 8.8% ($P = .001$). Mortality also varied in these patients on the basis of the urgency of the operation. Elective operations had a 7.3% (15/191) 30-day mortality versus a 34.0% (33/97) 30-day mortality for emergency operations ($P = .0001$). Mortality was higher in patients with descending operations than in those with either ascending or thoracoabdominal operations (Table 9), reflecting the relatively large number of ruptured aortas in the descending category (15/63, 23.8%).

Etiology of Stroke

Using operative reports (specifically, surgeons' reports of severe atheroma or clot), neurologic clinical consultation, and neuroradiologic evaluation of stroke pattern on computed tomography or magnetic resonance imaging, we were
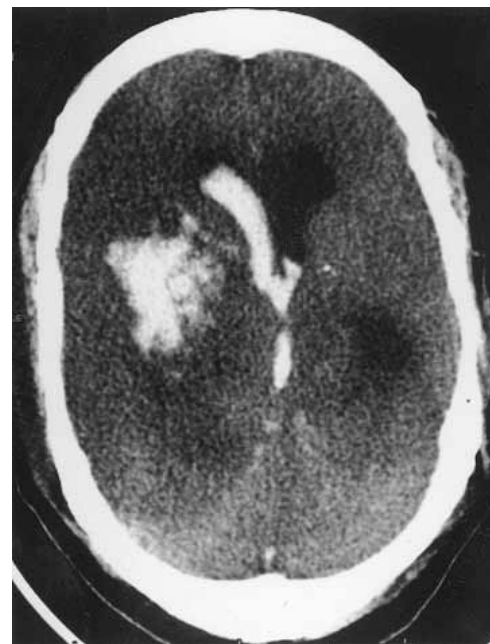

Figure 3. Computed tomographic scan of hemorrhagic stroke. Note the obvious extravasation of blood and midline shift resulting from the mass effect of the bleeding.

able to assign a specific cause for all except 2 of the strokes. Of the strokes encountered, 65.2% (15/23) were embolic in origin, 13.0% (3/23) were ischemic, and 13.0% (3/23) were hemorrhagic. The cause of 2 patients' strokes could not be
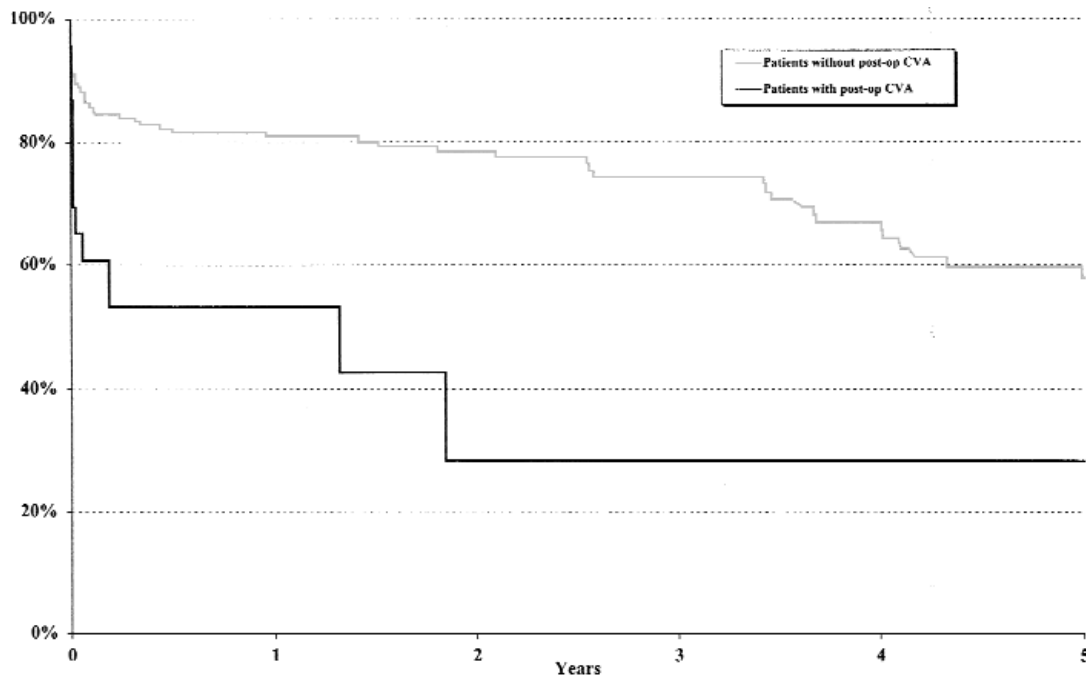
Figure 4. Kaplan-Meier survival analysis of patients having a stroke after aortic surgery. *CVA,* Cerebrovascular accident.

TABLE 6. Risk factors for stroke—univariate analysis (1-way analysis of variance tests)

Risk factor	Mean value		P value
	CVA	No CVA	
Operative date	1/4/96	5/5/96	.642
Anesthesia time (min)	454.4	420.0	.2774
Operative time (min)	363.8	336.0	.4701
CPB time (min)*	159.3	144.2	.3412
Aortic crossclamping (min)*	94.7	88.0	.5015
DHCA time (min)†	37.3	31.0	.0279‡
Lowest core temperature (°C)†	17.9	17.1	.4533
Crystalloids (intraoperative) (mL)	4352	3549	.2295
Colloids (intraoperative) (mL)	50.0	214.3	.3341
FFP (intraoperative) (units)	3.91	1.61	.0003‡
pRBCs (intraoperative) (units)	4.21	2.67	.096
Platelets (intraoperative) (units)	5.83	2.76	.0017†
Cell salvage (intraoperative) (units)	4.83	3.89	.7308
Urine output (intraoperative) (mL)	1317	1118	.286
Preoperative heart rate	80.9	77.3	.3035
Postoperative heart rate	92.0	93.0	.9183
Preoperative MAP	82.9	90.7	.3325
Postoperative MAP	54.9	55.9	.924
Postoperative PAD	16.8	15.7	.5718
Postoperative CI	2.95	2.64	.3784

CI, Cardiac index; *CPB,* cardiopulmonary bypass; *CVA,* cerebrovascular accident; *DHCA,* deep hypothermic circulatory arrest; *FFP,* fresh frozen plasma; *MAP,* mean arterial pressure; *PAD,* pulmonary artery diastolic pressure; *pRBCs,* packed red blood cells.
*Analysis performed only for those patients where relevant time (CPB or crossclamp) was not zero.
†Analysis performed only for those patients undergoing DHCA.
‡Statistically significant result.

determined. Preoperative and intraoperative data and detailed outcomes for patients with stroke are presented in Table 10. Neuroradiologic representations of embolic, ischemic, and hemorrhagic strokes are illustrated in Figures 1 to 3.

Clinical Impact

Stroke was a significant predictor of postoperative mortality. Of the 300 patients surviving the operation, 9 (39.1%) of 23 patients with stroke died during the postoperative hospital stay, compared with 23 (8.3%) of 277 patients without stroke ($P = .001$). Patients with stroke accounted for 9 (28.1%) of the 32 postoperative deaths. Of those surviving the postoperative hospital stay, patients having a stroke stayed an average of 11.6 days longer in the intensive care unit than those without a stroke (18.4 vs 6.8 days; $P = .0001$). Additionally, patients with a stroke remained intubated an average of 8.9 days longer than those without stroke (12.7 vs 3.8 days; $P = .0012$). Finally, patients with a stroke stayed an average of 17.1 days longer in the hospital after the operation than those without (31.4 vs 14.3 days; $P = .001$).

Long-term follow-up of patients via office visits, telephone calls, and office records shows a marked difference in survival, with those patients who had a stroke exhibiting a greatly decreased long-term survival compared with those without a stroke ($P = .0001$) (Figure 4). This difference persists when the results are adjusted for patient age by means of proportional hazards regression (relative risk = 2.775, $P = .0013$).

TABLE 7. Logistic regression analysis of factors predicting rupture or acute dissection (dependent variables)*

Variable	Parameter estimate	SE	P value	OR	(95% CI)
Intercept term†	−4.6535	0.6719	.0001		
Desc/TA surgery	0.8325	0.575	.1477	2.299	(0.745-7.095)
Emergency surgery‡	1.6069‡	0.5117	.0017‡	4.987‡	(1.829-13.596)
History of DM‡	2.0097‡	0.7324	.0061‡	7.461‡	(1.776-31.350)
Use of DHCA§	1.2457§	0.0122	.0262§	3.475§	(1.159-10.424)
FFP (risk/unit)§	0.1143§	0.0534	.0322§	1.121§	(1.010-1.245)

CI, Confidence intervals; *Desc*, descending; *DHCA*, deep hypothermic circulatory arrest; *DM*, diabetes mellitus; *FFP*, fresh frozen plasma; *OR*, odds ratio; *SE*, standard error; *TA*, thoracoabdominal.

*This variable equals 1 if the patient incurred a stroke and 0 otherwise. Forward selection with entry at $P < .030$.

†Criteria for assessing model fit: −2 log L: intercept only: 161.526; intercept and covariates: 131.235; χ^2 for covariates: 30.291 with 5 degrees of freedom ($P = .0001$).

‡Statistically significant at $P < .01$ level.

§Statistically significant at $P < .05$ level.

TABLE 8. Complications by surgeon

Surgeon	Procedures performed	Stroke rate (%)	Mortality rate (%)
A	182	5.5	8.8
B	30	10.0	30.0
C	19	10.5	15.8
D	18	5.6	11.1
E	18	5.6	22.2
F	17	17.7	17.7
G	12	0.0	25.0
H	7	14.3	57.1
Others	1.75*	14.3	35.7
P value		.554	.001

*Average number of procedures performed per surgeon included in this group.

TABLE 9. Mortality rates

Procedure	No. performed	Intraop death	Postop deaths	30-Day mortality rate
Ascending/arch	218	8 (3.7%)*	21 (9.6%)	29 (13.3%)†
Descending	63	8 (12.7%)*	9 (14.3%)	17 (27.0%)†
Thoracoabdominal	36	1 (2.8%)*	2 (5.6%)	3 (8.3%)†

The *P* values shown are for the comparison between ascending/arch versus descending/thoracoabdominal.

*Statistically significant difference, $P = .047$.

‡Statistically significant difference: ascending/arch versus descending, $P = .010$; ascending/arch versus thoracoabdominal, $P = .020$; descending versus thoracoabdominal, $P = .001$.

ACD

This clinical information indicated the importance of embolism in the origin of stroke in these patients. A program of antiembolic measures was systematically instituted at our center thereafter, corresponding to operations performed during and after November 1998. These measures included use of intraoperative transesophageal echocardiography to identify aortic atheroma, thorough debridement of atheroma, use of the subclavian artery for cannulation (rather than the femoral) in cases of severe arteriosclerosis of the descending aorta, carbon dioxide flooding of the field, avoidance of the left atrium for cannulation in cases of atrial fibrillation, and routine clamping of the proximal thoracic aorta before starting left atrial–femoral artery bypass. Carbon dioxide flooding of the field was used throughout the entire experience, before and after the vigorous antiembolic regimen.

Since the institution of these changes, the overall stroke rate has decreased (from 7.9% to 5.6%; $P = .463$). This decrease is most pronounced in surgery on the descending aorta, where the stroke rate has decreased from 8 in 77 cases to 0 in 22 cases. Although some of the improvement may be accounted for by gradually increasing surgical proficiency, later operative date (as a continuous variable) was not associated with decreased stroke rates (Table 6). Mortality during this time period decreased commensurately, to 7.4% (5/68) for ascending and arch operations and 13.6% (3/22) for descending and thoracoabdominal operations ($P = .082$ and $P = .384$, respectively); there were no deaths except in those patients presenting on an urgent basis.

Discussion
Incidence

Stroke was found in this review to be disturbingly frequent after aortic surgery (6.9% for operations on the ascending aorta and 8.1% for those on the descending aorta). This incidence of stroke after aortic surgery is fully in line with prior reviews.[1,6-8] Little information is known to us from the literature regarding stroke after descending aortic surgery for comparison with our data.

Occurrence of stroke is somewhat counterintuitive in operations on the descending aorta because the descending aorta, being downstream from the head vessels, might not be expected to be a significant source of air or particulate

TABLE 10. Patients with stroke: Preoperative and intraoperative characteristic

Patient	Disease	Rupture	HTN	Carotid disease
	Neurologic deficit			
0031	Type B dissection	–	Mild	None
	Listless, unresponsive			
0048	Desc aneurysm	+	Mild	None
	Seizures, generalized dysfunction			
0066	Type A dissection	–	Mild	None
	Seizures			
0113	Asc aneurysm	–	None	None
	Left hemiparesis, seizures			
0115	Type A dissection	+	None	None
	Right lower extremity weakness, visual changes			
0131	Type A dissection	–	Mild	None
	Brain death			
0132	Desc aneurysm	+	Mild	None
	Left hemiplegia and neglect, left facial droop			
0177	Type B dissection	–	Moderate	None
	Left arm and face paraplegia			
0195	Desc aneurysm	+	Mild	None
	Right hemiparesis, unresponsive			
0214	Asc aneurysm	–	Mild	None
	Right-sided weakness			
0230	Type A dissection	+	None	None
	Seizures, death			
0405	Type A dissection	+	Mild	None
	General slowing, bilateral weakness			
0555	Type B dissection	–	Mild	Mild
	Unresponsive, death			
0593	Asc aneurysm	–	Severe	None
	Left visual field deficit, left-sided weakness and paralysis, sensory loss below T3			
0602	Type A dissection	+	Mild	None
	Unresponsive, obtunded, brain death			
0609	Type A dissection	+	Mild	None
	Seizures, death			
0622	Type A dissection	+	None	None
	Brain death			
0652	Asc aneurysm	–	None	None
	Middle right hemiparesis			
0963*	Type A dissection	–	Mild	None
	Visual field deficits			
0991*	Type A dissection	–	None	None
	Right-sided weakness			
1006*	Type A dissection	–	None	None
	Left dense hemiparesis			
1014*	Asc aneurysm	–	Mild	Moderate
	Seizures, death			
1013*	Arch aneurysm	–	Mild	Moderate
	Bilateral weakness			

Desc, Descending; *Asc,* ascending; *arch,* aortic arch; *HTN,* hypertension; *N/A,* not available; *POD,* postoperative day; *PVD,* peripheral vascular disease; *RA,* right atrium; *Ao,* aorta; *FA,* femoral artery; *FV,* femoral vein; *SCA,* subclavian artery; *DHCA,* deep hypothermic circulatory arrest; *CPB,* cardiopulmonary bypass; *ACA, MCA, PCA,* anterior, middle, and posterior cerebral arteries.
*Indicates patient operated on after the systematic use of antiembolic measures.
†Stroke either embolic or due to hypoperfusion.

Cannulation site	Cerebral protection	Stroke etiology	30-Day survival
Imaging studies			
None	N/A	Hemorrhagic	Death POD 9
Extensive left frontal intraparenchymal, diffuse intraventricular and subarachnoid hemorrhages			
RA-Ao	DHCA	Embolic	Survived
Bilateral cerebellar and frontal, right caudate, and parietal infarcts			
LA-Ao	DHCA	Embolic	Survived
Bilateral thalmic infarcts			
RA-FA	DHCA	Embolic	Survived
Evolving right cerebellar and frontal lobe infarcts without hemorrhage			
RA-FA	CPB	Embolic	Survived
Bilateral caudate and periventricular white matter infarcts			
RA-FA	CPB	Ischemic	Death POD 5
Diffuse anoxic brain injury			
Ao-FA	DHCA	Embolic	Survived
Evolving right MCA infarcts			
RA-FA	CPB	Embolic	Survived
Early ischemia in the right MCA distribution			
FV-FA	DHCA	Embolic	Survived
Multiple bilateral infarcts			
RA-FA	DHCA	Embolic	Death POD 20
Right frontal infarcts			
RA-FA	DHCA	Embolic	Death POD 5
Extensive bilateral infarcts			
RA-FA	DHCA	Ischemic	Survived
Bilateral wedge-shaped parietal"watershed"infarcts			
LA-FA	None	Hemorrhage	Death POD 1
Extensive, diffuse intraparenchymal, intraventricular, and subarachnoid hemorrhages			
FV-FA	CPB	Embolic	Survived
Subacute infarcts in the right parieto-occipital region and posterior portion of the right temporal lobe			
RA-FA	DHCA	Unknown	Death POD 2
Imaging not performed			
RA-FA	DHCA	Ischemic	Death POD 4
Bilateral cerebellar infarcts and ACA/MCA "watershed" and left PCA infarcts			
RA-FA	CPB	Hemorrhage	Death POD 3
MCA infarctions with subsequent hemorrhagic conversion			
RA-FA	DHCA	Embolic	Survived
Right cerebellar infarct			
None	N/A	Unknown†	Survived
Occipital brain-stem/PCA infarct			
RA-FA	DHCA	Embolic	Survived
Left parietal infarct			
RA-Ao	CPB	Embolic	Survived
Right cerebellar infarcts			
RA-SCA	DHCA	Embolic	Death POD 4
Multiple bilateral infarcts			
RA-FA	DHCA	Embolic	Survived
Multiple bilateral infarcts, diffuse, all lobes, left greater than right			

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TABLE 11. Stroke rates

	Overall (n = 317)	Ascending (n = 218)	Descending (n = 99)	Elective (n = 206)	Emergency (n = 97)
Without antiembolic procedures (n = 227)	18 (7.9%)	10 (6.7%)	8 (10.4%)	6 (3.9%)	12 (16.2%)
With antiembolic procedures (n = 90)	5 (5.6%)	5 (7.4%)	0 (0.0%)	1 (1.9%)	4 (17.4%)
Overall	23 (7.3%)	15 (6.9%)	8 (8.1%)	7 (3.4%)	16 (16.5%)
P values	.463	.853	.115	.481	.894

matter. This study found that stroke does indeed occur in descending aortic operations, in fact fully as frequently as in ascending aortic operations. We believe that manipulation of the aortic arch for proximal control in descending aortic operations is one factor. We believe that retrograde perfusion from below (femoral artery) is another factor producing stroke in descending aortic operations.

Impact

We have shown that stroke has a devastating impact on survival, producing death in the hospital in 28.3% of those affected and leaving only 28% alive at 2 years. Hospital morbidity was dramatically increased in stroke survivors, including intensive care unit stay, duration of intubation, and postoperative hospital stay. In addition to the usual concerns of death, bleeding, and paraplegia, surgeons must consider stroke heavily when deciding to operate on the thoracic aorta.

Etiology

In multivariable analysis, a number of risk factors were found to be predictive of stroke. These included a history of diabetes mellitus (possibly caused by chronic damage to the cerebral microvasculature), emergency surgery, the use and duration of hypothermic circulatory arrest, and the amount of transfused fresh frozen plasma (which may be a surrogate indicator of coagulopathy or intraoperative bleeding in these patients). Surprisingly, we found no correlation between the incidence of stroke and the location of either the proximal clamp in descending aortic surgery or the arterial cannulation site. The clinicoradiographic analysis revealed that the strokes in this study were predominantly (65.2%) embolic in nature. An embolic origin would explain the failure to correlate with measurable preoperative characteristics in multivariable analysis, because embolism is a purely technical issue. Ergin and associates[8] also concluded that permanent neurologic deficits were due to thromboembolic events and were not related to the type of cerebral protection used.

Prevention

Because intraoperative emboli are the overwhelming source of cerebrovascular accidents in this study, antiembolic precautions during the operative period become paramount.

The sobering impact of stroke on length and quality of life apparent in this review leads the surgeon to focus on potential modifications to operative technique to reduce the incidence of stroke in patients undergoing thoracic aortic surgery. Several practical recommendations arise as corollaries of the embolic etiologic mechanism of stroke.

In addition to the attention commonly given to minimize cardiopulmonary bypass time and deep hypothermic arrest interval, the surgeon must focus on antiembolic precautions. The following specific technical alterations in the conduct of surgery have been incorporated in our techniques and are recommended:

1. Mobilization. Even mobilization for operations on the severely diseased descending aorta can liberate debris to the cerebral vessels. Thus, extreme care is taken in manipulation of the aortic arch for proximal control in descending aortic operations.

2. Debridement. Severely diseased aortic cuffs are meticulously debrided of atheromatous debris before anastomosis and restoration of circulation, because remnants can easily be embolized to the head vessels.

3. Cannulation and perfusion. Transesophageal echocardiography can map the location and severity of aortic atheromatous disease and direct the surgeon toward different cannulation and perfusion approaches.[9-11] In patients with severe, mobile atheroma of the descending aorta, the surgeon may be presented with unfavorable conditions for retrograde femoral perfusion. In these cases, alternative perfusion sites, for example, the subclavian or axillary arteries, may prove effective at preventing embolization of debris, especially inasmuch as these sites provide "antegrade" flow. Retrograde flow may be more likely to lift the "shingle-like" atheromas molded by years of natural antegrade flow.

4. Carbon dioxide flooding. Our institution uses carbon dioxide flooding of the operative field. Because carbon dioxide is heavier than atmospheric air, it will displace this air. Theoretically, any gas embolized from the field will be carbon dioxide, which will more easily dissolve once in the cerebral circulation and pose less of a risk to the patient. This modality has an excellent therapeutic ratio: essentially no cost and very significant potential benefits.

5. Order of clamping. We recommend application of the proximal clamp (in descending aortic surgery) before the

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institution of femoral perfusion to prevent any disrupted atheromatous debris from reaching the cerebral circulation. Similarly, we discontinue perfusion before release of the proximal clamp.

6. Avoidance of the left atrium in atrial fibrillation. In patients with atrial fibrillation, we avoid cannulating the left atrial appendage for left atrial–femoral bypass for descending aortic surgery, for fear of liberating debris from the appendage by the cannulation process. Femoro-femoral bypass or deep hypothermic arrest may be preferable techniques.

The decrease in stroke rate from 8.1% to 0.0% for operations on the descending aorta in the time period after these observations were made and systematic antiembolic measures instituted suggests a beneficial impact of these measures. Stroke is an important cause of mortality, and mortality has also decreased during the postimplementation period. These outcomes at our institution have already shown moderate to strong beneficial trends in some parameters after implementation of a rigorous antiembolic regimen. To demonstrate precise statistical significances from the implementation of antiembolic measures would require huge patient numbers; we think the demonstrated trends are suggestive of a beneficial impact.

We conclude that potential for postoperative strokes is significant after surgery of the thoracic aorta and needs careful inclusion in weighing the pros and cons of surgical intervention for a particular patient. The risk of stroke is about 7%. Stroke is often lethal (28.3%), accounting for one quarter of all deaths from thoracic aortic surgery. Among those patients who survive to be discharged from the hospital after a stroke, only 28% will be alive 2 years after thoracic aortic resection. The majority of strokes are embolic—technically related. Specific technical correlates for prevention present themselves once the embolic origin is

appreciated. Application of these technical correlates may help to decrease the incidence of stroke, a devastating complication of thoracic aortic surgery.

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