Unilateral is comparable to bilateral antegrade cerebral perfusion in acute type A aortic dissection repair

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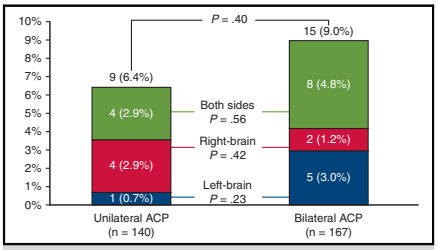
ABSTRACT

Objective: To compare the short- and long-term outcomes of unilateral and bilateral antegrade cerebral perfusion (uni-ACP and bi-ACP) in acute type A aortic dissection (ATAAD) repair.

Methods: From 2001 to 2017, 307 patients underwent surgical repair of an ATAAD using uni-ACP (n = 140) and bi-ACP (n = 167). Data were collected through the Department of Cardiac Surgery Data Warehouse, medical record review, and the National Death Index database.

Results: The demographics and preoperative comorbidities were similar between the uni-ACP and bi-ACP groups. Both groups had similar rates of procedures for aortic valve/root, ascending aorta, frozen elephant trunk, and other concomitant procedures. Perioperative outcomes were not significantly different between the 2 groups (30-day mortality: uni-ACP 3.4% vs bi-ACP 7.8%, $P = .12$) except reoperation for bleeding was significantly lower in uni-ACP (5% vs 12%, $P = .03$). Between the uni-ACP and bi-ACP groups, overall postoperative stroke rate (6% vs 9%, $P = .4$) and left brain stroke rate (0.7% vs 3.0%, $P = .23$) were not significantly different. The odds ratio of uni-ACP versus bi-ACP was 0.87 ($P = .80$) for postoperative stroke and 0.86 ($P = .81$) for operative mortality. The mid-term survival was better in the uni-ACP group, $P = .027$ (5-year: 84% vs 76%). The hazard ratio of all-time mortality for uni-ACP versus bi-ACP was 0.74 (95% confidence interval, 0.33-1.65), $P = .46$.

Conclusions: In ATAAD, both uni-ACP and bi-ACP are equally effective to protect the brain with low postoperative stroke rates and mortality in hemiarch to zone 3 arch replacement. Uni-ACP is recommended for its simplicity and less manipulation of arch branch vessels. (J Thorac Cardiovasc Surg 2020;160:617-25)



New-onset postoperative stroke after ATAAD repair with unilateral or bilateral ACP.

Central Message

Unilateral ACP provides adequate cerebral protection and achieves favorable short-term outcomes and mid-term survival in acute type A aortic dissection repair as effectively as bilateral ACP.

Perspective

Compared with bilateral ACP, unilateral ACP provides adequate cerebral perfusion with similar stroke rate, operative mortality, and mid-term survival. Bilateral ACP did not provide any additional benefits but may increase embolic stroke of the left brain. Unilateral ACP should be considered for aortic arch repair in acute type A aortic dissection for its simplicity and effectiveness.

See Commentaries on pages 626 and 627.

Acute type A aortic dissection (ATAAD), a life-threatening medical emergency, requires immediate surgical intervention and is correlated with high rates of mortality (17%-25%[1,2]) and neurologic complications. The overall surgical management of aortic disease has been evolving and the extent and complexity of ATAAD procedures has been increasing.[3] Rates of arch repair have increased from 27% of ATAAD procedures in 2003 to 2008 to 37% in 2013 to 2015[3] and although the use of circulatory arrest has not significantly increased, the total time of circulatory arrest has increased.[3] Due to this evolution, cerebral protection has become increasingly important to reduce neurologic complications and improve outcomes.

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Dr Yang is supported by the NHLBI of NIH K08HL130614 and R01HL141891, Phil Jenkins and Darlene & Stephen J. Szatmari Funds.

Received for publication Feb 17, 2019; revisions received June 21, 2019; accepted for publication July 12, 2019; available ahead of print Sept 5, 2019.

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0022-5223/$36.00

https://doi.org/10.1016/j.jtcvs.2019.07.108

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Abbreviations and Acronyms

ACP = antegrade cerebral perfusion
ATAAD = acute type A aortic dissection
bi-ACP = bilateral antegrade cerebral perfusion
CT = computed tomography
HCA = hypothermic circulatory arrest
LCC = left common carotid
NIRS = near-infrared spectroscopy
RCC = right common carotid
uni-ACP = unilateral antegrade cerebral perfusion

Antegrade cerebral perfusion (ACP) is used more frequently in ATAAD repair than retrograde cerebral perfusion[2-4] and within ACP, the superiority of unilateral or bilateral cerebral perfusion remains a controversial topic. Some surgeons advocate the use of bilateral cerebral perfusion as it is more physiologic, perfusing both hemispheres to avoid any unnecessary risk of unilateral perfusion[5] and in cases with longer circulatory arrest times.[6] Other surgeons advocate the use of unilateral ACP due to its shorter implementation time, simplicity, avoidance of manipulation of the dissected arch branch vessels, and equal effectiveness,[6-8] even in cases with circulatory arrest times longer than 1 hour.[7] In this study, we report our clinical experience using ACP in hemiarch to total arch replacement in the setting of ATAAD, comparing perioperative outcomes and mid-term survival between unilateral antegrade cerebral perfusion (uni-ACP) and bilateral antegrade cerebral perfusion (bi-ACP) to determine the effectiveness of cerebral protection with uni-ACP versus bi-ACP.

METHODS

This study was approved by the institutional review board at the University of Michigan (date and number of institutional review board approval: September 26, 2016, and HUM00119716) and was in compliance with Health Insurance Portability and Accountability Act regulations.

Study Population

All patients (n = 307, consecutive cases) who suffered an ATAAD from 2001 to January 2017 and underwent central aortic repair using ACP were identified, including uni-ACP (n = 140, 46%) and bi-ACP (n = 167, 54%). Investigators leveraged Society of Thoracic Surgeons data elements from the University of Michigan Cardiac Surgery Data Warehouse to identify the cohort and determine preoperative, operative, and postoperative characteristics. Electronic medical record review was conducted to confirm that patients underwent repair with ACP and to supplement data collection. Postoperative stroke was defined as in the Society of Thoracic Surgeons database, any confirmed neurologic deficit of abrupt onset following aortic repair caused by a disturbance in blood supply to the brain that did not resolve within 24 hours, or evidence of a stroke on computed tomography (CT) scan with or without clinical symptoms. If there is a stroke on CT scan, but symptoms resolved before discharge, they were counted as a stroke. Patients who had strokes due to malperfusion from aortic dissection before open aortic repair were not counted as new-onset postoperative stroke, but all were counted in the analysis of mid-term survival. However, if patients had a stroke before surgery but developed new neurologic deficit clinically or on CT scan following aortic repair, they were counted as new-onset postoperative stroke. Investigators used the National Death Index database through December 31, 2015,[9] and medical record review to obtain mid-term survival.

Surgical Techniques of ACP and Aortic Arch Surgery

ACP was used for all cases, including uni-ACP and bi-ACP. Choice of ACP was based on surgeon's practice. Uni-ACP was achieved through a graft to the axillary artery (n = 91), a graft to the intrathoracic right subclavian artery (n = 13), a graft to the innominate artery (n = 29) or direct innominate cannulation (n = 5), or a graft to the right common carotid artery (n = 2). The right axillary artery was the preferred site for arterial cannulation in the early stage. Lately, to avoid an additional incision, the arterial cannulation site has been migrated to the innominate artery, and more recently to the intrathoracic right subclavian artery with a chimney graft (8-mm Dacron graft). Two patients had carotid artery cannulation due to carotid artery occlusion and stroke preoperatively, and we cannulated the right common carotid (RCC) artery to perfuse the brain as quickly as possible. Both patients had no postoperative stroke.

When uni-ACP was used, the left common carotid (LCC) was always clamped to prevent stealing of cerebral perfusion. Bilateral ACP was achieved through direct cannulation of the innominate/RCC and LCC arteries (n = 138), or through a graft to the innominate/right subclavian artery/right axillary artery and direct cannulation of the LCC artery (n = 29). If the vessel was dissected, we cannulated the true lumen of the branch vessel. We did not routinely clamp the left subclavian artery. The flow rate for ACP was 10 mL/kg/min for both uni-ACP and bi-ACP in all cases. Blood pressure during ACP was maintained around 60 mm Hg (50-70 mm Hg). Cerebral saturation was maintained around >50% in most cases. Three cases (2%) were converted from uni-ACP to bi-ACP due to minimal back-bleeding from the LCC artery or near-infrared spectroscopy (NIRS) dropping by >30% from baseline right before hypothermic circulatory arrest (HCA). Patients were cooled to 18°C before 2012 and to 24°C to 28°C since 2012 for ACP. The blood temperature for ACP during HCA was at the cooling temperature of the whole body or 18°C to 22°C since 2017 to further cool the brain. Topical (head) cooling was achieved via placement of an ice pack around the head for every case. Mannitol and solumedrol with or without barbiturates were administrated right before HCA. NIRS was used for every patient.

Indications for zone 1 to 3 arch replacement included an arch aneurysm >4 cm, intimal tear located in the arch that could not be resected by a hemiarch replacement, or dissection of arch branch vessels with malperfusion.[10] In hemiarch or zone 1 arch (arch divided between innominate and LCC arteries) replacement (n = 162), after the distal anastomosis at arch, the brain was perfused bilaterally. In zone 2 (arch divided between LCC and left subclavian arteries) and zone 3 (arch divided distal to the left subclavian artery) arch replacements (n = 145), after the distal arch anastomosis, the LCC artery was reimplanted/replaced separately first to achieve bilateral cerebral perfusion, then the innominate artery was reimplanted/replaced, and the left subclavian artery last. All arch branch vessels were reimplanted/replaced individually to branch grafts. Arch branch vessels were resected and replaced if they were thrombosed and significantly occluded due to dissection. Separate incisions were made at the neck to replace the whole common carotid arteries if the false lumen of the common carotid arteries was thrombosed and the arteries were severely stenotic or occluded. In the last 5 years, a frozen elephant trunk (cTAG-10 cm, manufactured by W. L. Gore & Associates, Inc, Flagstaff, Ariz) has been placed into the true lumen of the descending thoracic aorta distal to the left subclavian artery if the intimal tear was found in the proximal descending aorta to cover the intimal tear or a narrow true lumen was found in the distal thoracic or abdominal aorta on CT angiogram to prevent lower body

malperfusion. The root procedures were performed as previously described.[11] Malperfusion syndrome was diagnosed based on imaging (CT angiogram) and clinical presentation (neurologic deficit, abdominal pain, paralysis of extremities, pulse deficit, troponin I, lactate, creatinine, creatinine phosphokinase, etc) and was managed with endovascular fenestration/stenting and delayed open central aortic repair as described previously.[12]

Statistical Analysis

Initial analysis provided descriptive information on the demographic, clinical, and surgical characteristics. Continuous variables were reported as median (25%, 75%) and categorical variables as n (%) in frequency tables. Univariate comparisons between uni-ACP and bi-ACP groups were performed using χ^2 tests for categorical data and Wilcoxon rank sum tests for continuous data. Multivariable logistic regression was used to assess the risk factors for operative mortality and postoperative stroke by adjusting to group, age, sex, year of operation, peripheral vascular disease, ejection fraction, surgeon, history of stroke, acute stroke, HCA time, crossclamp time, and hemiarch/zone 1 versus zone 2/3 arch replacement. Crude survival curves of all patients since operation were estimated using the non-parametric Kaplan–Meier method. Log-rank test was used to compare the survival of groups (uni-ACP vs bi-ACP). Cox proportional hazard regression was performed to calculate the hazard ratio for all-time mortality by adjusting group, age, sex, year of operation, coronary

TABLE 1. Demographics and preoperative data

Variables	Total (n = 307)	Unilateral ACP (n = 140)	Bilateral ACP (n = 167)	*P* value
Patient age, y	59 (49, 67)	59.5 (52, 69.5)	57 (48, 66)	.03
Sex, male	217 (71)	94 (67)	123 (74)	.21
Preexisting comorbidities				
Hypertension	228 (74)	105 (75)	123 (74)	.79
Diabetes mellitus	22 (7.2)	11 (7.9)	11 (6.6)	.67
History of smoking				.78
Current	85 (28)	41 (29)	44 (27)	
CAD	44 (15)	19 (14)	25 (15)	.78
COPD	33 (11)	14 (10)	19 (11)	.69
History of stroke	8 (2.6)	3 (2.1)	5 (3.0)	.73
History of renal failure	15 (4.9)	6 (4.3)	9 (5.4)	.66
On dialysis	6 (2.0)	2 (1.4)	4 (2.4)	.69
Marfan syndrome	13 (4.2)	5 (3.6)	8 (4.8)	.59
Other connective tissue disorder	3 (1.0)	2 (1.4)	1 (0.6)	.59
Bicuspid aortic valve	22 (8.0)	9 (6.7)	13 (9.2)	.65
PVOD	46 (15)	27 (19)	19 (11)	.053
Previous cardiac surgery	32 (10)	14 (10)	18 (11)	.82
Preoperative AI				.13
Severe	63 (22)	23 (17)	40 (26)	
Ejection fraction	55 (55, 60)	56 (55, 65)	55 (50, 60)	.02
Acute myocardial infarction	7 (2.3)	4 (2.9)	3 (1.8)	.10
Acute stroke	17 (5.5)	11 (7.9)	6 (3.6)	.12
Acute renal failure	43 (14)	15 (11)	28 (17)	.25
Acute paralysis	7 (2.3)	2 (1.4)	5 (2.9)	.46
Cardiogenic shock	23 (7.5)	12 (8.6)	11 (6.6)	.51
Preoperative creatinine	1.0 (0.8, 1.3)	1.0 (0.8, 1.3)	1.0 (0.9, 1.4)	.26
Malperfusion				
Coronary	6 (2.0)	4 (2.9)	2 (1.2)	.42
Cerebral	17 (5.5)	10 (7.1)	7 (4.2)	.26
Spinal cord	7 (2.3)	2 (1.4)	5 (2.9)	.46
Celiac/hepatic	6 (2.0)	2 (1.4)	4 (2.4)	.69
Mesenteric	27 (8.8)	10 (7.1)	17 (10)	.35
Renal	24 (7.8)	10 (7.1)	14 (8.4)	.69
Extremity	33 (11)	12 (8.6)	21 (13)	.26
Delayed open repair	50 (16)	14 (10)	36 (22)	.006

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *ACP*, Antegrade cerebral perfusion; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *PVOD*, peripheral vascular occlusive disease; *AI*, aortic insufficiency.

artery disease, preoperative renal failure, acute paralysis, acute myocardial infarction, cardiogenic shock, and zone 2 or 3 arch replacement. All statistical calculations used SAS 9.4 (SAS Institute, Cary, NC) and were considered significant at $P < .05$.

RESULTS

Demographics and Preoperative Data

Bi-ACP was used more in the early stage (before 2011), whereas uni-ACP was used more in the late stage of the study period (after 2011) (Figure E1). The demographics and preoperative comorbidities were similar between uni-ACP and bi-ACP groups except patients in the bi-ACP group were significantly younger (57 vs 59.5 years old, $P = .03$). There was no significant difference in presence of malperfusion syndrome. The 2 groups were very comparable (Table 1).

Intraoperative Data

The complexity of aortic root procedures and concomitant procedures was similar between the 2 groups. Overall, the bi-ACP group had significantly more zone 2/3 arch replacements but fewer hemiarch replacements; longer aortic crossclamp and HCA times, which was the lower body circulatory arrest time; and lower body temperatures during HCA. The bi-ACP group required significantly more intraoperative transfusion of packed red blood cells (Table 2).

Postoperative Outcomes

Overall, there were no significant differences in major postoperative outcomes. However, the bi-ACP group had significantly more reoperation for bleeding than the uni-

TABLE 2. Intraoperative data

Variables	Total (n = 307)	Unilateral ACP (n = 140)	Bilateral ACP (n = 167)	*P* value
Aortic root procedure				.76
AVR only	7 (2.3)	2 (1.4)	5 (2.9)	
Aortic root replacement	84 (27)	41 (29)	43 (26)	
Aortic root repair	187 (61)	84 (60)	103 (62)	
Arch replacement				
None	1 (0.3)	1 (0.7)	0 (0)	.46
Hemiarch	122 (40)	87 (62)	35 (21)	<.0001
Zone 1 arch	39 (13)	16 (11)	23 (14)	.64
Zone 2 arch	109 (36)	32 (23)	77 (46)	<.0001
Zone 3 arch	36 (12)	4 (2.9)	32 (19)	<.0001
Frozen elephant trunk	30 (10)	15 (11)	15 (8.9)	.61
CPB time, min	227 (190, 281)	224.5 (191.5, 280)	230 (188, 285)	.86
Crossclamp time, min	160 (116, 205)	144 (103, 184.5)	173 (133, 224)	<.0001
HCA time, min	38 (27, 49)	29 (22.5, 38)	45 (38, 55)	<.0001
Lowest temperature, °C	18 (17, 22)	20 (18, 24)	17 (16, 18)	<.0001
Cannulation				<.0001
Innominate	34 (11)	34 (24)		
Axillary	91 (30)	91 (65)		
RCC	2 (0.7)	2 (1.4)		
RSc	13 (4.2)	13 (9.3)		
Innominate+LCC	134 (44)		134 (80)	
RSc/axillary+LCC	27 (8.8)		27 (16)	
RCC+LCC	6 (2.0)		6 (3.6)	
Concomitant operations				
CABG	17 (5.5)	9 (6.4)	8 (4.8)	.53
MV	2 (0.7)	0 (0)	2 (1.2)	.5
TV	4 (1.3)	2 (1.4)	2 (1.2)	1
Blood transfusion (PRBCs)				<.0001
0 units	64 (21)	47 (34)	17 (10.6)	
1 unit	23 (7.7)	13 (9.4)	10 (6.3)	
2 units	28 (9.4)	14 (10)	14 (8.8)	
≥3 units	184 (62)	65 (47)	119 (74)	

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *ACP*, Antegrade cerebral perfusion; *AVR*, aortic valve replacement; *CPB*, cardiopulmonary bypass; *HCA*, hypothermic circulatory arrest; *RCC*, right common carotid; *RSc*, right subclavian; *LCC*, left common carotid; *CABG*, coronary artery bypass graft; *MV*, mitral valve; *TV*, tricuspid valve; *PRBCs*, packed red blood cells.

ACP group but a similar reoperation for bleeding rate after propensity score match (Table E1). The bi-ACP group had 15 (9%) new-onset postoperative strokes, whereas the uni-ACP group had 9 (6.4%), which was not significantly different (Table 3, Figure 1, *A*). The majority (92%) of new-onset postoperative strokes were embolic in the whole cohort, and 11% (1 patient) of all strokes in the uni-ACP group were located in the left brain only, whereas 33% (5 patients) of all strokes in the bi-ACP group were in the left brain only (Table 4, Figure 1, *A*). The severity of the strokes was similar between the 2 groups (Table 4). Logistic regression showed uni-ACP, age, zone 2/3 arch replacement, HCA and crossclamp times, year of surgery, and individual surgeon were not significant risk factors for postoperative stroke. The odds ratio (OR) of uni-ACP versus bi-ACP was 0.87 (95% confidence interval, 0.29-2.59), $P = .80$ (Table 5).

The operative mortality, which includes mortality in the hospital or within 30 days of surgery, was not significantly different between the uni-ACP and bi-ACP groups (5.7% vs 9.6%), $P = .21$. Age at operation (OR, 1.04; $P = .04$) and acute preoperative stroke (OR, 5.5; $P = .04$) were significant risk factors for operative mortality but not uni-ACP (OR, 0.86 vs bi-ACP), HCA time (OR, 1.02), crossclamp time (OR, 1.01), year of surgery (OR, 1.05), or individual surgeon (Table 5).

Mid-Term Outcomes

The follow-up time was 5 ± 4 years (maximum 17 years). There were 51 deaths total in the whole cohort during follow-up. The mid-term survival was better in the uni-ACP group compared with the bi-ACP group, $P = .027$ (5-year survival: 84% vs 76%) (Figure 1, *B*). However, in Cox proportional hazard regression, only age, preoperative renal failure, acute myocardial infarction, acute paralysis, and cardiogenic shock were significant risk factors for all-time mortality after surgery but not bi-ACP (vs uni-ACP) or zone 2/3 arch replacement (vs hemi/zone 1 arch replacement) (Table 6, Figure 1, *C*). There was no difference in incidence rate of stroke during follow-up between uni-ACP (0.49%/year) and bi-ACP (0.33%/year).

DISCUSSION

In this study, we found that the postoperative neurologic complications and operative mortality were not significantly different between uni-ACP and bi-ACP groups. The mid-term survival was significantly better in the uni-ACP group compared with that in the bi-ACP group by Kaplan–Meier analysis. However, multivariable Cox proportional hazard analysis did not show that bi-ACP was a significant risk factor of all-time mortality after surgery (bi-ACP vs uni-ACP, hazard ratio, 1.36; $P = .46$) (Video 1).

Because the aortic arch is frequently dissected in ATAAD, most arch repairs are managed with circulatory arrest. The increasing adoption of strategies for cerebral perfusion and protection may be improving perioperative and long-term outcomes compared with deep hypothermia and circulatory arrest alone in patients undergoing aortic arch surgery,[13] and ACP is an important adjunct to HCA in arch surgery.[2] In most reports of aortic arch repair in elective cases or

TABLE 3. Postoperative outcomes

Variables	Both (n = 307)	Unilateral ACP (n = 140)	Bilateral ACP (n = 167)	*P* value
Myocardial infarction	3 (1.0)	1 (0.7)	2 (1.2)	1
New-onset cerebrovascular accident	24 (7.8)	9 (6.4)	15 (9.0)	.40
New-onset renal failure on dialysis	25 (8.2)	11 (7.9)	14 (8.6)	.82
Reoperation for bleeding	27 (8.8)	7 (5.0)	20 (12)	.03
Sepsis	6 (2.0)	3 (2.1)	3 (1.8)	1
Paraplegia	2 (0.7)	1 (0.7)	1 (0.6)	1
Need for tracheostomy	9 (2.9)	4 (2.9)	5 (3.1)	1
Prolonged ventilation	176 (58)	78 (56)	98 (59)	.60
Hours intubated	45.5 (23, 99)	41 (22, 91)	51 (24, 106)	.24
Postoperative length of stay, d	11 (7, 17)	11 (7, 16)	12 (7, 18)	.34
Intraoperative mortality	4 (1.3)	0 (0)	4 (2.4)	.13
In-hospital mortality	21 (6.8)	7 (5.0)	14 (8.4)	.24
30-d mortality	18 (5.9)	5 (3.4)	13 (7.8)	.12
Operative mortality*	24 (7.8)	8 (5.7)	16 (9.6)	.21

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. Intraoperative mortality was defined as mortality in the operating room, and was counted in both in-hospital mortality and 30-day mortality. In-hospital mortality was defined as mortality in the hospital before discharge. 30-day mortality was defined as mortality within 30 days after surgery independent of patients being in the hospital or discharged. There is overlapping in in-hospital mortality and 30-day mortality. *ACP*, Antegrade cerebral perfusion. *Operative mortality was defined as all mortalities in the hospital or within 30 days after surgery.

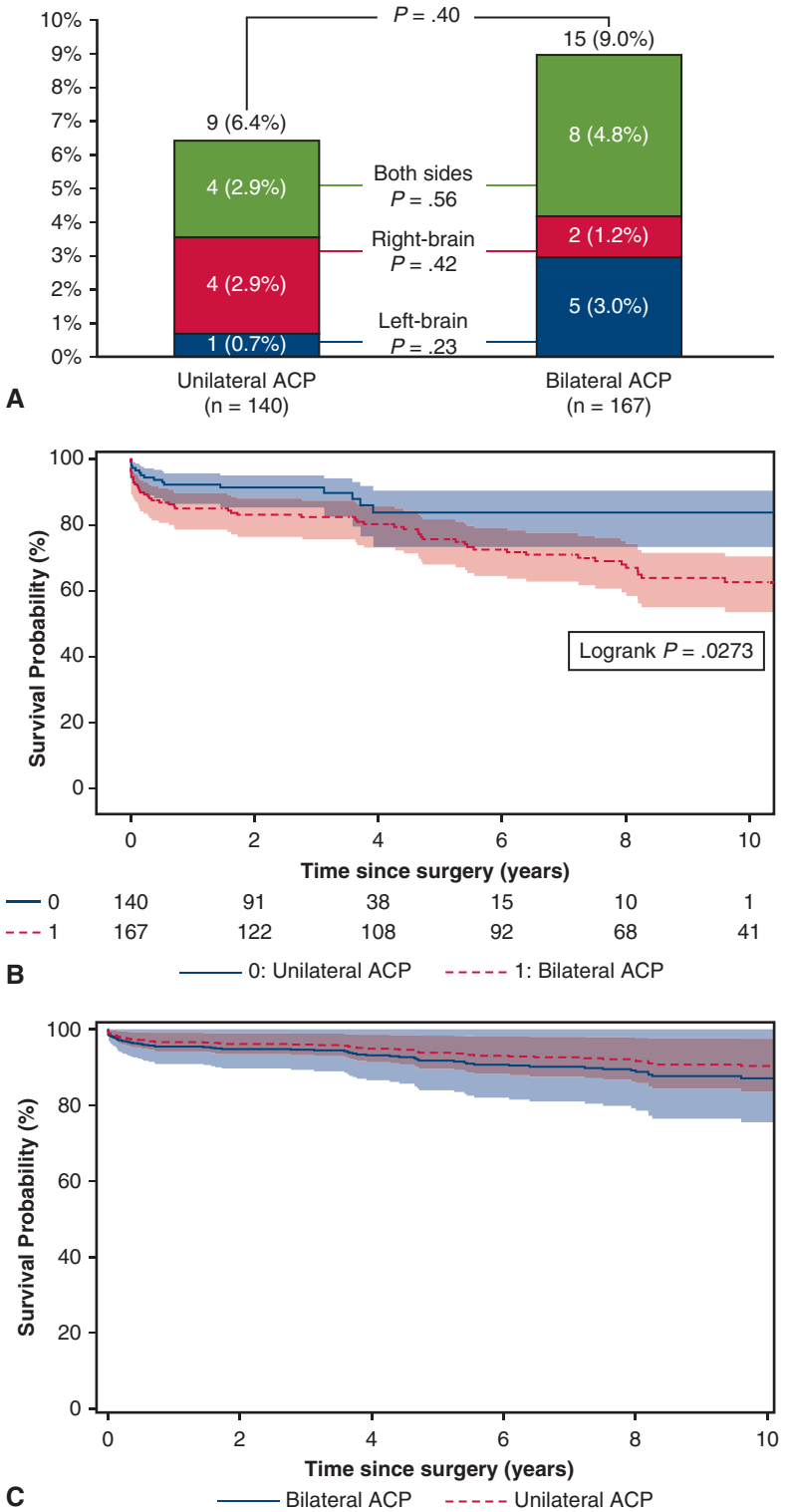


FIGURE 1. A, The distribution of new-onset postoperative stroke after ATAAD repair with uni-ACP or bi-ACP. Only 1 patient had an embolic isolated left cerebral stroke, and no patients had an isolated left cerebral stroke due to hypoperfusion in the uni-ACP group. B, Survival (Kaplan–Meier analysis) of all patients with ATAAD repair using uni-ACP or bi-ACP. The 5-year survival was better in the uni-ACP group compared with the bi-ACP group (84% vs 76%). C, Cox proportional hazard regression model: survival of a 60-year old male patient without comorbidities (including coronary artery disease, preoperative renal failure, acute myocardial infarction, acute paralysis, or cardiogenic shock), operated in 2017 using uni-ACP versus bi-ACP (hazard ratio, 0.74; 95% confidence interval, 0.33-1.65, $P = .46$). The curve was truncated at 10 years. *ACP*, Antegrade cerebral perfusion.

TABLE 4. Details of stroke in unilateral ACP and bilateral ACP groups

	Unilateral ACP (n = 9)	Bilateral ACP (n = 15)	*P* value
Location			
Left-brain	1 (11)	5 (33)	.35
Right-brain	4 (44)	2 (13)	.15
Both sides	4 (44)	8 (53)	1.0
Etiology			
Embolic	9 (100)	13 (87)	.51
Hemorrhagic	0 (0)	2 (13)	.51
Severity			
Coma/brain death	1 (11)	2 (13)	1.0
Motor deficit	8 (89)	13 (87)	1.0
Speech deficit	4 (44)	8 (53)	1.0
Temporary	5 (56)	4 (27)	.21
Permanent*	4 (44)	11 (73)	.21

Data presented as n (%) for categorical data. *ACP*, Antegrade cerebral perfusion. *Permanent stroke was defined as stroke not fully recovered at postoperative visit or before in-hospital death.

combined ATAAD cases, uni-ACP is just as effective as bi-ACP.[14] In the studies comparing uni-ACP and bi-ACP in only ATAAD cases in the past 10 years, all 4 studies[5-8] reported similar outcomes (including postoperative stroke and 30-day mortality) between uni-ACP and bi-ACP, even though the HCA time was longer than 1 hour,[7] except in the study of Tong and colleagues.[5] In their study,[5] the 30-day mortality (20.7%) and stroke rate (16.9%) in total arch replacement are much greater in the uni-ACP group compared with the bi-ACP group, which are also much greater than those (around 10%-12%) reported in other studies.[6-8] In our study, the 30-day mortality (3.4%) and postoperative stroke rate (6.4%) in the uni-ACP group were one half of those in the bi-ACP group (Table 3). After propensity score match (n = 49 pairs) based on age, sex, extent of arch repair (hemiarch, zone 1-3 arch), and year of surgery there was still no significant difference of postoperative stroke or mortality (Table E1). Even in more extensive arch replacement (zone 2/3 arch), the 30-day mortality and postoperative stroke rates were both 2.8%, compared with 7.3% and 9.2% in the bi-ACP group (Tables E2-E4). We think the difference in the study of Tong and colleagues[5] was an institutional difference and not due to the different approaches of ACP (ie, uni-ACP vs bi-ACP). In addition, we also found the rate of intraoperative blood transfusion and postoperative reoperation for bleeding was significantly greater in the bi-ACP group. The favorable 30-day mortality, stroke rate, and transfusion rate in the uni-ACP could be associated with simplified ACP strategy and less manipulation of the dissected/thrombosed aortic arch and/or arch branch vessels.

The mechanisms of stroke in ATAAD repair are unknown. Multivariable logistic analysis did not show a significant factor associated with new-onset postoperative stroke, including the type of ACP, HCA time, zone 2/3 arch replacement, individual surgeon, or year of surgery (Table 5). The potential causes of stroke could be thromboembolization due to significant intra-arterial instrumentation and intramural thrombus, global hypoperfusion due to unstable hemodynamics, and compression of the true lumen from a thrombosed false lumen of the carotid arteries which compromises cerebral perfusion. The fact that we had more left-sided embolic stroke in the bi-ACP group (Table 4) conveys that direct cannulation of the LCC artery in patients undergoing ATAAD could increase the risk of left-sided embolic stroke. To prevent embolic stroke, we would recommend minimizing manipulation of the dissected aortic arch and arch branch vessels from both outside and inside those vessels, cannulating the true lumen

TABLE 5. Risk factors for postoperative stroke and operative mortality (multivariable logistic regression)

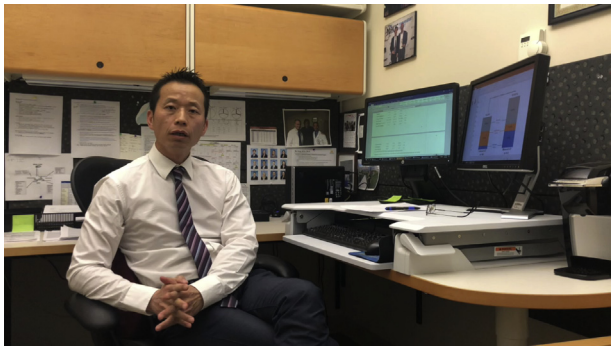
	Postoperative stroke		Operative mortality*	
	Odds ratio (95% CI)	*P* value	Odds ratio (95% CI)	*P* value
Bilateral ACP	1.62 (0.57-4.59)	.36	1.20 (0.36-4.00)	.77
Age	0.99 (0.96-1.02)	.41	1.04 (1.00-1.08)	.04
Sex, female	1.085 (0.45-2.635)	.86	0.36 (0.12-1.14)	.08
Ejection fraction <40%	0.55 (0.03-10.50)	.69	4.14 (0.60-28.68)	.15
PVOD	0.93 (0.29-3.00)	.91	1.07 (0.30-3.76)	.92
History of stroke	0.61 (0.03-11.16)	.74	0.69 (0.02-20.55)	.83
Acute stroke	1.185 (0.20-6.92)	.85	5.17 (1.06-25.30)	.04
HCA time	1.02 (0.99-1.04)	.18	1.01 (0.99-1.04)	.32
Zone 2/3 arch	0.69 (0.30-1.62)	.39	0.76 (0.30-1.90)	.55
Crossclamp time	1.00 (0.99-1.00)	.46	1.005 (0.999-1.01)	.10
Year of surgery	1.03 (0.92-1.16)	.61	0.975 (0.85-1.11)	.71

CI, Confidence interval; *ACP*, antegrade cerebral perfusion; *PVOD*, peripheral vascular occlusive disease; *HCA*, hypothermic circulatory arrest. *Operative mortality was defined as all mortalities in the hospital or within 30 days after surgery.

TABLE 6. Risk factors for late mortality after surgery (multivariable Cox proportional hazard regression)

	HR (95% CI)	*P* value
Uni-ACP	0.74 (0.33-1.65)	.46
Age	1.06 (1.04-1.08)	<.0001
Male	1.21 (0.69-2.10)	.51
Surgery year	0.93 (0.85-1.01)	.07
CAD	1.28 (0.71-2.30)	.42
History of renal failure	2.57 (1.16-5.69)	.02
Preoperative acute MI	6.03 (1.55-12)	.01
Acute paralysis	3.66 (1.22-11)	.02
Cardiogenic shock	2.95 (1.43-6.11)	.003
Zone 2/3 arch replacement	1.27 (0.73-2.23)	.40

HR, Hazard ratio; *CI*, confidence interval; *Uni-ACP*, unilateral antegrade cerebral perfusion; *CAD*, coronary artery disease; *MI*, myocardial infarction.



VIDEO 1. Discussion of short- and mid-term outcomes of patients with acute type A aortic dissection treated with unilateral versus bilateral antegrade cerebral perfusion during aortic arch repair. Video available at: https://www.jtcvs.org/article/S0022-5223(19)31700-3/fulltext.

in non-dissected peripheral arteries, such as the intrathoracic right subclavian artery, right axillary artery, or distal non-dissected carotid artery, and also avoiding femoral artery cannulation and retrograde perfusion of the body during cardiopulmonary bypass. To avoid global hypoperfusion, we recommend avoiding hypotension as much as possible and maintaining a greater blood pressure if patients have known carotid stenosis. If the carotid artery is occluded due to compression of thrombosed false lumen, we recommend reperfusing the brain as quickly as possible with a separate graft anastomosed to the carotid artery distal to the occlusion and replacing the occluded carotid arteries with arch replacement.

The concern with the use of uni-ACP is that the left side of the brain is underperfused due to an incomplete or variation of the circle of Willis. However, when we use uni-ACP through the innominate artery, right subclavian or axillary artery, or RCC artery, we perfuse the left side of the brain through collaterals between the right and left common carotid arteries and right and left external carotid arteries,[15] in addition to the circle of Willis. During HCA, the back-bleeding from the LCC artery is another piece of evidence of the perfusion of the left brain. If the back-bleeding from the LCC artery is minimal or the left NIRS decreases by >30%, the collaterals between the left and right common carotid arteries may not be robust. We would recommend cannulating the LCC artery to ensure perfusion of the left brain, unless the LCC artery is occluded by thrombosed false lumen. ATAAD is frequently associated with aortopathy due to genetic defect, rarely atherosclerosis; therefore, most patients undergoing ATAAD have robust normal RCC-LCC collaterals, which is different from patients with atherosclerotic arch aneurysms. In our study, only 3 cases were converted from uni-ACP to bi-ACP, and none had postoperative strokes. If uni-ACP causes hypoperfusion to the left brain during HCA, one would expect most of the strokes in the uni-ACP group to be of the left brain, global injury, and more severe stroke. However, in the uni-ACP group, there was only 1 patient (0.7%) who had an isolated left cerebral embolic stroke (focal hypodensity seen within the genu and posterior limb of the left internal capsule) compared with 5 patients (3%) in the bi-ACP group. Zero patients in the uni-ACP group had an isolated left cerebral stroke due to global hypoperfusion, and the severity of strokes was similar between the 2 groups. (Table 4, Figure 1, *A*) All of these findings indicated uni-ACP alone was as effective as bi-ACP for cerebral protection in arch repair with HCA in most patients undergoing ATAAD.

The HCA time was significantly longer in the bi-ACP group, most likely due to the extra work to insert the cannula into the LCC artery. Sometimes, the LCC artery was dissected, which required very careful insertion of the cannula. In addition, we set a separate pressure monitoring system just for LCC perfusion, which took more time. When patients had bi-ACP, surgeons felt the brain was well-protected and were more meticulous with the anastomosis of the Dacron graft to the distal arch during HCA. Teflon felt was used before 2012 for the arch anastomosis with bi-ACP, which could have increased HCA time. The longer HCA time resulted in longer aortic crossclamp time but not CPB time (Table 2). Although both HCA and crossclamp times were longer in the bi-ACP group, neither of them were significant risk factors for postoperative stroke or mortality with ORs close to 1 (Table 5).

To our surprise, the mid-term survival was significantly better in the uni-ACP group compared with the bi-ACP group (Figure 1, *B*); however, the difference became nonsignificant after propensity score match (Figure E2), which is consistent with the previous study[6] (no difference of 3-year survival). The mid-term survival statistics could be impacted by the lower number of patients with uni-ACP that have been followed after 4 years postoperatively (Figure 1, *B*). However, the mid-term survival in the uni-ACP group was not any

worse, if not better, than that in the bi-ACP group. The hazard ratio of uni-ACP versus bi-ACP for all-time mortality after surgery in the Cox model was 0.74 (Table 5). Taken together, we would recommend uni-ACP over bi-ACP for aortic arch repair in ATAAD for its simplicity and effectiveness. Since 2012, 80% of ATAADs have been repaired with uni-ACP at our institution (Figure E1). Some surgeons still use retrograde cerebral perfusion for hemiarch replacement sometimes at our institution, which is a different topic and not included in this study.

This study has limitations as a single-center, retrospective study. More bi-ACP was used in 2001 to 2011 and more uni-ACP was used in 2012 to 2017 (Figure E1). However, the year of surgery was found not to be a significant risk factor of operative mortality or late mortality (hazard ratio, 0.93) (Table 5). Using uni-ACP or bi-ACP was based on surgeon's practice, and some surgeons could choose bi-ACP if more extensive arch work was anticipated. This could create selection bias, as is seen in most retrospective studies using a different practice as control. However, the preoperative conditions were very comparable between the 2 groups (Table 1). There was no difference in outcomes among individual surgeons by logistic regression. The surgeons used the same criteria for arch replacement as we discussed in the methods. Both propensity score match analysis and subcohort analysis of zone 2/3 arch replacement showed similar outcomes in uni-ACP group compared with the bi-ACP group (Tables E1-E4). Therefore, we do not think this limitation weakened our conclusion that uni-ACP is simple and as effective as bi-ACP for cerebral protection. The sample size was relatively small; therefore, the study may be underpowered. We used a clinical diagnosis of postoperative stroke based on the Society of Thoracic Surgeons definition or imaging, which could underestimate the subclinical strokes.

CONCLUSIONS

In conclusion, both uni- and bi-ACP perfusion provide adequate cerebral protection in ATAAD with good short-term outcomes and mid-term survival. Unilateral ACP should be considered for aortic arch repair in ATAAD for its simplicity and less manipulation of dissected arch branch vessels.

Conflict of Interest Statement

Authors have nothing to disclose with regard to commercial support.

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Key Words: antegrade cerebral perfusion, aortic arch replacement, aortic dissection, stroke, survival

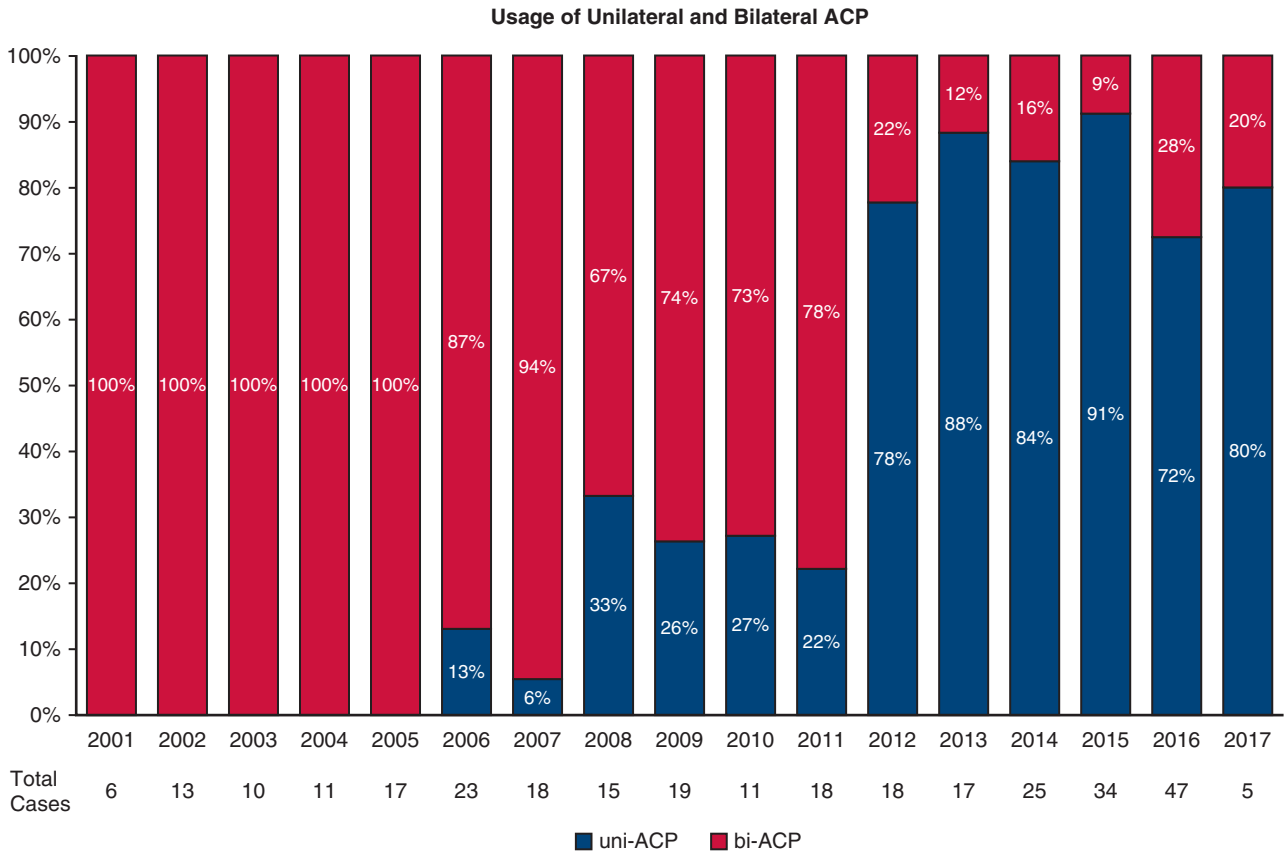

FIGURE E1. Usage of unilateral and bilateral antegrade cerebral perfusion over time. Bi-ACP was used more in the early stage while uni-ACP was used more in the late stage of the study period. *Uni-ACP*, Unilateral antegrade cerebral perfusion; *Bi-ACP*, bilateral antegrade cerebral perfusion.

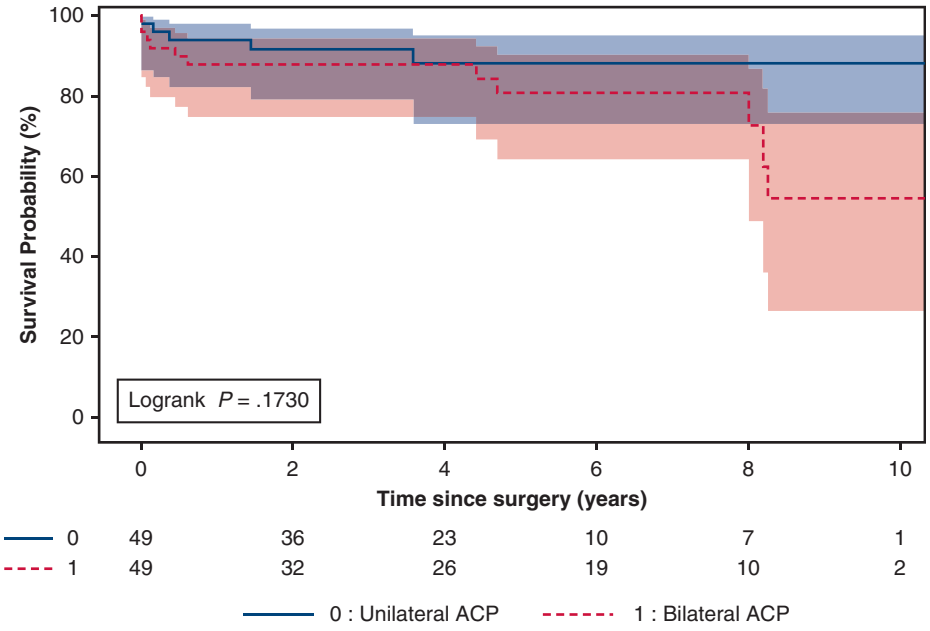

FIGURE E2. Survival (Kaplan-Meier analysis) of propensity-matched patients with acute type A aortic dissection repair using unilateral and bilateral antegrade cerebral perfusion. *ACP*, Antegrade cerebral perfusion.

TABLE E1. Postoperative outcomes following propensity score matching using age, sex, year of surgery, extent of arch replacement—hemiarch, zone 1, 2, 3 arch replacement

Variables	Both (n = 98)	Uni-ACP matched (n = 49)	Bi-ACP matched (n = 49)	*P* value
Myocardial infarction	0 (0)	0 (0)	0 (0)	
New-onset cerebrovascular accident	8 (8.2)	3 (6.1)	5 (10)	.71
New-onset renal failure on dialysis	8 (8.2)	5 (10)	3 (6.1)	.71
Reoperation for bleeding	6 (6.1)	1 (2.0)	5 (10)	.2
Sepsis	2 (2.0)	1 (2.0)	1 (2.0)	1
Paraplegia	1 (1.0)	0 (0)	1 (2.0)	1
Need for tracheostomy	3 (3.1)	1 (2.0)	2 (4.1)	1
Prolonged ventilation	61 (62)	30 (61)	31 (63)	.83
Hours intubated	50 (24, 103.5)	52 (27, 97)	46 (24, 103.5)	.94
Postoperative length of stay, d	12 (7, 18)	13 (7, 18)	11 (7, 18)	.88
Intraoperative mortality	0 (0)	0 (0)	0 (0)	
In-hospital mortality	5 (5.1)	2 (4.1)	3 (6.1)	1
30-day mortality	4 (4.1)	1 (2.0)	3 (6.1)	.62
Operative mortality*	6 (6.1)	2 (4.1)	4 (8.2)	.68

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. Intraoperative mortality was defined as mortality in the operating room and was counted in both in-hospital mortality and 30-day mortality. In-hospital mortality was defined as mortality in the hospital before discharge. Thirty-day mortality was defined as mortality within 30 days after surgery independent of patients being in the hospital or discharged. There is overlapping in in-hospital mortality and 30-day mortality. Operative mortality was defined as all mortalities in the hospital or within 30 days after surgery. *Uni-ACP*, Unilateral antegrade cerebral perfusion; *Bi-ACP*, bilateral antegrade cerebral perfusion.
*Operative mortality was defined as all mortalities in the hospital or within 30 days after surgery.

TABLE E2. Subcohort analysis: demographic and preoperative data

	None/hemiarch/zone 1 arch replacement			Zone 2/3 arch replacement		
	Unilateral ACP (n = 104)	Bilateral ACP (n = 58)	*P* value	Unilateral ACP (n = 36)	Bilateral ACP (n = 109)	*P* value
Patient age, y	63 (52.5, 71)	60 (44, 66)	.04	57 (48, 63.5)	57 (48, 66)	.85
Sex, male	74 (71)	43 (74)	.68	20 (56)	80 (73)	.04
BSA	2.1 (1.9, 2.2)	2.1 (1.9, 2.2)	.99	2.0 (1.8, 2.2)	2.1 (1.9, 2.2)	.30
Preexisting comorbidities						
Hypertension	79 (76)	46 (79)	.49	26 (72)	77 (71)	.79
Diabetes mellitus	8 (7.7)	5 (8.6)	.77	3 (8.3)	6 (5.5)	.69
History of smoking			.80			.23
None	46 (44)	28 (49)		16 (46)	46 (42)	
Former	30 (29)	14 (25)		6 (17)	34 (31)	
Current	28 (27)	15 (27)		13 (37)	29 (27)	
CAD	19 (19)	11 (19)	.99	0 (0)	14 (13)	.02
COPD	12 (11.5)	5 (8.6)	.58	2 (5.6)	14 (13)	.34
History of stroke	3 (2.9)	2 (3.4)	1	0 (0)	3 (2.7)	.57
History of renal failure	6 (5.8)	5 (8.6)	.52	0 (0)	4 (3.7)	.57
On dialysis	2 (1.9)	3 (5.2)	.35	0 (0)	1 (0.9)	1
Marfan syndrome	5 (4.8)	3 (5.2)	1	0 (0)	5 (4.6)	.33
Other connective tissue disorder	1 (0.9)	0 (0)	1	1 (2.8)	1 (0.9)	.43
Bicuspid aortic valve	9 (8.7)	9 (18)	.17	0 (0)	4 (4.4)	.57
PVOD	23 (22)	6 (10)	.07	4 (11)	13 (12)	1
Previous cardiac surgery	12 (11.5)	2 (3.4)	.09	2 (5.6)	16 (15)	.24
Preoperative AI			.03			.59
None	23 (23.5)	20 (36)		16 (47)	37 (37)	
Trace	18 (18)	4 (7.2)		2 (5.9)	15 (15)	
Mild	21 (21)	8 (15)		5 (15)	12 (12)	
Moderate	19 (19)	5 (9.1)		5 (15)	15 (15)	
Severe	17 (17)	18 (33)		6 (18)	22 (22)	
Ejection fraction	55 (55, 65)	55 (55, 60)	.06	59 (55, 60)	55 (50, 60)	.32
Acute myocardial infarction	4 (3.9)	2 (3.4)	1	0 (0)	1 (0.9)	1
Acute stroke	8 (7.7)	3 (5.2)	.75	3 (8.3)	3 (2.8)	.16
Acute renal failure	10 (9.6)	9 (16)	.26	5 (14)	19 (17)	.63
Acute paralysis	1 (0.9)	2 (3.4)	.29	1 (2.8)	3 (2.8)	1
Cardiogenic shock	9 (8.7)	5 (8.6)	.99	3 (8.3)	6 (5.5)	.69
Tamponade	14 (13.5)	3 (5.2)	.11	1 (2.8)	5 (4.6)	1
Preoperative creatinine	1.0 (0.8, 1.2)	1.1 (0.9, 1.5)	.035	1.1 (0.9, 1.5)	1.0 (0.8, 1.3)	.76
Malperfusion						
Coronary	3 (2.9)	1 (1.7)	1	1 (2.8)	1 (0.9)	.44
Cerebral	7 (6.7)	4 (6.9)	1	3 (8.3)	3 (2.8)	.16
Spinal cord	1 (0.9)	2 (3.4)	.29	1 (2.8)	3 (2.8)	1
Celiac/hepatic	2 (1.9)	3 (5.2)	.35	0 (0)	1 (0.9)	1
Mesenteric	6 (5.8)	6 (10)	.35	4 (11)	11 (10)	1
Renal	6 (5.8)	5 (8.6)	.52	4 (11)	9 (8.3)	.74
Extremity	8 (7.7)	7 (12)	.34	4 (11)	14 (13)	1
Delayed open repair	9 (8.7)	11 (19)	.06	5 (14)	25 (23)	.25

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *ACP*, Antegrade cerebral perfusion; *BSA*, body surface area; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *PVOD*, peripheral vascular occlusive disease. *AI*, aortic insufficiency.

TABLE E3. Subcohort analysis: intraoperative data

	None/hemiarch/zone 1 arch replacement			Zone 2/3 arch replacement		
	Unilateral ACP (n = 104)	Bilateral ACP (n = 58)	*P* value	Unilateral ACP (n = 36)	Bilateral ACP (n = 109)	*P* value
Aortic root procedure			.54			.84
AVR only	2 (1.9)	2 (3.4)		0 (0)	3 (2.8)	
Aortic root replacement	36 (35)	22 (38)		5 (14)	21 (19)	
Aortic root repair	59 (56.7)	33 (56.9)		25 (69)	70 (64)	
Arch replacement						
None	1 (1.0)	0 (0)	1			
Hemiarch	87 (84)	35 (60)	.002			
Zone 1 arch	16 (15)	23 (40)	.0005			
Zone 2 arch				32 (89)	77 (71)	.028
Total Arch				4 (11)	32 (29)	.043
Frozen elephant trunk	9 (8.7)	3 (5.2)	.54	6 (17)	12 (11)	.39
CPB time, min	231.5 (191.5, 281.5)	226.0 (199, 285)	.84	217 (192, 242.5)	231 (188, 279)	.27
Crossclamp time, min	149 (97.5, 191)	178 (111, 226)	.06	136 (113, 161)	167 (136, 222)	.0008
HCA time, min	29 (23, 38)	41 (32, 50)	<.0001	27.5 (22, 37)	47 (39, 57)	<.0001
Lowest temperature, °C	20 (18, 24)	18 (17, 20)	.049	22 (20, 24)	17 (16, 18)	<.0001
Bladder	26.5 (22, 31)	27 (20, 34)	.48	26.5 (22, 29.5)	21 (19, 31)	.13
Esophageal	24 (18, 29)	25 (16, 30)	.85	25 (19, 30)	20 (17, 30)	.39
Cannulation			<.0001			<.0001
Innominate	22 (21)			12 (33)		
Axillary	74 (71)			17 (47)		
RCC	0 (0)			2 (5.6)		
RSc	8 (7.7)			5 (14)		
Innominate + LCC		46 (79)			79 (72)	
Innominate + LCC + LSc		1 (1.7)			8 (7.3)	
RCC + LCC		0 (0)			6 (5.5)	
Axillary + LCC		10 (17)			13 (12)	
Axillary + LCC + LSc		0 (0)			2 (1.8)	
RSc + LCC		1 (1.7)			1 (0.9)	
Concomitant operations						
CABG	9 (8.7)	3 (5.2)	.54	0 (0)	5 (4.6)	.33
MV	0 (0)	0 (0)		0 (0)	2 (1.8)	1
TV	2 (1.9)	2 (3.4)	.62	0 (0)	0 (0)	
Blood transfusion (PRBCs)			.11			<.0001
0 units	31 (30)	8 (15)		16 (44)	9 (8.6)	
1 unit	10 (9.7)	6 (11)		3 (8.3)	4 (3.8)	
2 units	9 (8.7)	5 (9.1)		5 (14)	9 (8.6)	
≥3 units	53 (51)	36 (65)		12 (33)	83 (79)	

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *ACP*, Antegrade cerebral perfusion; *AVR*, aortic valve replacement; *CPB*, cardiopulmonary bypass; *HCA*, hypothermic circulatory arrest; *RCC*, right common carotid; *RSc*, right subclavian; *LCC*, left common carotid; *LSc*, left subclavian; *CABG*, coronary artery bypass graft; *MV*, mitral valve; *TV*, tricuspid valve; *PRBCs*, packed red blood cells.

TABLE E4. Subcohort analysis: postoperative outcomes

	None/hemiarch/zone 1 arch replacement			Zone 2/3 arch replacement		
	Unilateral ACP (n = 104)	Bilateral ACP (n = 58)	*P* value	Unilateral ACP (n = 36)	Bilateral ACP (n = 109)	*P* value
Myocardial infarction	1 (0.9)	1 (1.8)	1	0 (0)	1 (0.9)	1
New-onset cerebrovascular Accident	8 (7.7)	5 (8.8)	1	1 (2.8)	10 (9.2)	.29
New-onset renal failure on dialysis	6 (5.8)	4 (7.0)	.74	5 (14)	10 (9.2)	.53
Reoperation for bleeding	6 (5.8)	5 (8.8)	.52	1 (2.8)	15 (14)	.12
Sepsis	2 (1.9)	2 (3.5)	.61	1 (2.8)	1 (0.9)	.44
Paraplegia	1 (0.9)	0 (0)	1	0 (0)	1 (0.9)	1
Need for tracheostomy	4 (3.9)	2 (3.5)	1	0 (0)	3 (2.8)	.57
Prolonged ventilation	60 (58)	32 (55)	.76	18 (50)	66 (61)	.27
Hours intubated	45 (22, 95)	45 (22, 110)	.72	34 (22, 90)	52 (24, 105)	.35
Postoperative length of stay, d	11.5 (7, 16)	12 (8, 17)	.88	8 (7, 15)	12 (7, 18)	.13
Intraoperative mortality	0 (0)	2 (3.4)	.12	0 (0)	2 (1.8)	1
In-hospital mortality	5 (4.8)	4 (6.9)	.72	2 (5.6)	10 (9.2)	.73
30-day mortality	4 (3.9)	5 (8.6)	.28	1 (2.8)	8 (7.3)	.45
Operative mortality*	6 (5.8)	6 (10)	.35	2 (5.6)	10 (9.2)	.73

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. Intraoperative mortality was defined as mortality in the operating room, and was counted in both in-hospital mortality and 30-day mortality. In-hospital mortality was defined as mortality in the hospital before discharge. 30-day mortality was defined as mortality within 30 days after surgery independent of patients being in the hospital or discharged. There is overlap in in-hospital mortality and 30-day mortality. *ACP,* Antegrade cerebral perfusion. *Defined as mortality in-hospital or within 30 days after surgery.