Is hemiarch replacement adequate in acute type A aortic dissection repair in patients with arch branch vessel dissection without cerebral malperfusion?

Elizabeth L. Norton, MS,[a] Xiaoting Wu, PhD,[b] Karen M. Kim, MD,[b] Shinichi Fukuhara, MD,[b] Himanshu J. Patel, MD,[b] G. Michael Deeb, MD,[b] and Bo Yang, MD, PhD[b]

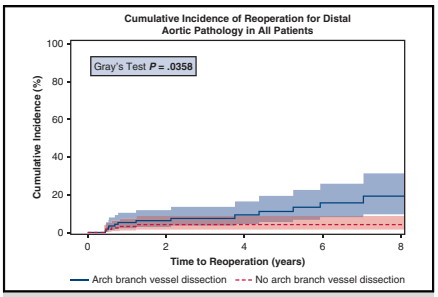
ABSTRACT

Objective: The study objective was to determine if hemiarch replacement is an adequate arch management strategy for patients with acute type A aortic dissection and arch branch vessel dissection but no cerebral malperfusion.

Methods: From January 2008 to August 2019, 479 patients underwent open acute type A aortic dissection repair. After excluding those with aggressive arch replacement (n = 168), cerebral malperfusion syndrome (n = 34), and indeterminable arch branch vessel dissection (n = 1), 276 patients with an acute type A aortic dissection without cerebral malperfusion syndrome who underwent hemiarch replacement comprised this study. Patients were then divided into those with arch branch vessel dissection (n = 133) and those with no arch branch vessel dissection (n = 143).

Results: The median age of the entire cohort was 62 years, with the arch branch vessel dissection group being younger (60 vs 62 years, $P = .048$). Both groups had similar aortic arch and descending thoracic aortic diameters, with significantly more DeBakey type I dissections (100% vs 80%) in the arch branch vessel dissection group. The arch branch vessel dissection group had more aortic root replacement (36% vs 27%, $P = .0035$) and longer aortic crossclamp times (153 vs 128 minutes, $P = .007$). Postoperative outcomes were similar between the arch branch vessel dissection and no arch branch vessel dissection groups, including stroke (10% vs 5%, $P = .12$) and operative morality (7% vs 5%, $P = .51$). The arch branch vessel dissection group had a significantly greater cumulative incidence of reoperation (8-year: 19% vs 4%, $P = .04$) with a hazard ratio of 2.89 (95% confidence interval, 1.01-8.27; $P = .048$), which was similar between groups among only DeBakey type I dissections (8-year: 19% vs 5%, $P = .11$). The 8-year survival was similar between the arch branch vessel dissection and no arch branch vessel dissection groups (76% vs 74%, $P = .30$).

Conclusions: Hemiarch replacement was adequate for patients with acute type A aortic dissection with arch branch vessel dissection without cerebral malperfusion syndrome, but carried a higher risk of late reoperation. (J Thorac Cardiovasc Surg 2021;161:873-84)



ABVD in ATAAD with hemiarch replacement had an increased cumulative incidence of reoperation.

CENTRAL MESSAGE

Hemiarch replacement produced adequate short-term outcomes for patients with ATAAD with ABVD without cerebral MPS, but could result in a higher risk of late reoperation.

PERSPECTIVE

Hemiarch replacement in ATAAD repair yields adequate short-term outcomes in patients with and without ABVD; however, because of the increased risk of reoperation in patients with ABVD, aggressive arch replacement could be considered by experienced surgeons for young, stable patients with ABVD.

See Commentaries on pages 885, 886, and 888.

Acute type A aortic dissection (ATAAD) is a lethal disease necessitating emergency surgical repair with an operative mortality of 17% to 26%[1-3] in large national and international registries. With the principal goal of having a live patient leaving the operating room, much debate exists regarding how limited or extensive aortic repair should be at the time of ATAAD while resecting the intimal tear. In regard to the aortic arch and distal extent of the repair, some surgeons advocate for a conservative approach (limited ascending/hemiarch replacement),[4,5]

From the [a]Creighton University School of Medicine, Omaha, Neb; and [b]Department of Cardiac Surgery, University of Michigan, Michigan Medicine, Ann Arbor, Mich.

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Address for reprints: Bo Yang, MD, PhD, 5155 Frankel Cardiovascular Center, 1500 East Medical Center Drive, Ann Arbor, MI 48109 (E-mail: boya@med.umich.edu).

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Abbreviations and Acronyms

ABVD = arch branch vessel dissection
ATAAD = acute type A aortic dissection
CI = confidence interval
CT = computed tomography
HR = hazard ratio
LCC = left common carotid
MPS = malperfusion syndrome
OR = odds ratio

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whereas others advocate a more aggressive approach (up to a total arch replacement plus a frozen elepxhant trunk) to address future deterioration of the aortic arch and risk for reoperation upfront. The conservative approach lessens operative time in an already complex cardiac procedure, whereas the more aggressive approach often results in longer operative times and more complex techniques, and has the potential to reduce future reoperations and improve long-term outcomes.[4,6-8]

Many surmise there is no one optimum operation for all patients, but an optimum operation exists for each individual patient, with the key being patient selection. However, no consensus exists on which specific patient factors contribute to the decision of hemiarch versus aggressive arch. Factors often considered include tear in the arch, arch aneurysm, involvement of arch branch vessels with cerebral malperfusion, and Marfan syndrome,[8-10] but the role of arch branch vessel dissection (ABVD) without cerebral malperfusion remains undetermined.

This study examined consecutive patients over 10 years with ATAAD without cerebral or upper-extremity malperfusion syndrome (MPS) who underwent hemiarch replacement comparing preoperative condition, operative procedures, postoperative outcomes, reoperation rates, and long-term survival among patients with and without ABVD. The aim of the study was to determine if ABVD without associated MPS can be effectively managed with hemiarch replacement as in patients without ABVD.

MATERIALS AND METHODS

The study was approved by the Institutional Review Board at Michigan Medicine in compliance with Health Insurance Portability and Accountability Act regulations, and a waiver of consent was obtained.

Study Population

Between January 2008 and August 2019, 479 patients underwent open repair of an ATAAD. After excluding patients with cerebral or right upper-extremity MPS (tissue/organ necrosis and dysfunction due to inadequate blood flow) (n = 34) and indeterminable ABVD (n = 1), the cohort consisted of 444 patients, of whom 276 patients underwent hemiarch replacement who were then divided into those with ABVD (ABVD group, n = 133) and those without ABVD (no ABVD group, n = 143) (Figure E1). Cerebral and right upper-extremity MPS were determined by surgeons at the time of presentation on the basis of physical examination (eg, focal neurologic deficits), laboratory values, and findings on computed tomography (CT) scans. For patients who were hemodynamically unstable, intubated, or sedated at time of presentation to Michigan Medicine (12%, 32/276), history of initial presentation was used to determine preoperative neurologic status. The ABVD group only included patients with dissection that extended into the main trunk of the innominate artery or left common carotid (LCC) artery. Dissection limited to the origin of the arch branch vessels was not counted as ABVD.

Investigators used the 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 records, including CT scans, were systematically reviewed to supplement data collection and confirm ABVD. The National Death Index database through December 31, 2018,[11] was used as well as electronic medical record review to obtain data on long-term survival. Further survival and reoperation data were collected from a thorough medical record review in addition to surveys (letters and phone calls, January 2018). Reoperation included reoperations for the aortic arch, descending thoracic, or thoracoabdominal aortic aneurysms via sternotomy, thoracotomy, or thoracic endovascular aortic repair. Reoperations for aortic root pathology and other cardiac, nonaortic surgeries were not included as a reoperation. Loss of follow-up and end of the study period were treated as censors during the time to event analysis.

Surgical Techniques

The operative strategy in patients with ATAAD has been extensively described previously.[8,12-15] Briefly, the indication for aortic root replacement in patients with ATAAD included (1) intimal tear at the aortic root, (2) root diameter 4.5 cm or more, (3) connective tissue disease, and (4) unrepairable aortic valve pathology.[12,13] The root procedures include direct repair[12,13] or replacement as inclusion root, Bentall procedure, or David procedure.[12,13,16] Root repair includes preservation of the native tissue of the aortic root including the aortic sinuses and aortic valve, comprising aortic valve resuspension, aortic valve repair, and closure of the false lumen at the sinotubular junction. All patients in this study underwent hemiarch replacement, because dissection of arch branch vessels without MPS is not currently an indication for aggressive arch replacement (zone 1/2/3 arch replacement) with replacement of arch branch vessels in our group. Hemiarch replacement includes resection of the lesser curvature of the aortic arch to varying degrees without reimplantation of any arch branch vessels. In zone 1 arch replacement, the arch is divided between the innominate and LCC arteries with replacement of the innominate artery to its bifurcation; in zone 2 arch replacement, the arch is divided between the LCC and left subclavian arteries with replacement of the innominate and LCC arteries; and in zone 3 arch replacement (total arch replacement), the arch is divided distal to the left subclavian artery with replacement of all arch branch vessels. Indications for zone 1 to 3 arch replacement would be arch aneurysm greater than 4 cm or intimal tear in the arch, both of which were unable to be resected via hemiarch replacement, or dissection of arch branch vessels with MPS,[8] and these patients were excluded from this study. If needed, a frozen elephant trunk (cTAG 10 cm, WL Gore and Associates, Flagstaff, Ariz) was placed into the true lumen of the descending thoracic aorta distal to the left subclavian artery as described.[8] MPS of endovascular amenable vascular territories (visceral, extremity) was managed

with initial endovascular fenestration/stenting and delayed open aortic repair.[17,18]

Statistical Analysis

Continuous variables are presented as median (25 percentile, 75 percentile), and categorical variables are reported as n (%) in frequency tables. Univariate comparisons between no ABVD and ABVD groups were performed using chi-square tests or Fisher exact test for categorical data and Wilcoxon rank-sum tests for continuous data. Multivariable logistic regression was used to assess risk factors for postoperative stroke by adjusting ABVD, age, and hypothermic circulatory arrest time. Cumulative incidence curves adjusting for death as a competing risk were generated to assess reoperation rates over time. Gray's test was used to test the difference in cumulative incidence curves between the 2 groups. Incidence rates were calculated for long-term events (transient ischemic attack; stroke; reoperation for arch, descending, or thoracoabdominal aneurysm), in which the numbers of events were divided by total patient-years of follow-up. Rate ratio tests were used to compare the incidence rates between groups. Cox proportional hazard regression was performed to calculate the hazard ratio (HR) for reoperation for distal aortic pathology by adjusting ABVD, age, gender, and connective tissue disease. Crude survival curves since operation were estimated using the nonparametric Kaplan–Meier method. Log-rank test was used to compare the survival between groups. All statistical calculations used SAS 9.4 (SAS Institute, Inc, Cary, NC).

RESULTS

Demographics

The median age of the entire cohort was 62 years and predominantly consisted of men (68%), similar between groups, which is consistent with the general ATAAD population. The ABVD group had more peripheral vascular disease (24% vs 14%, $P = .03$) and more severe aortic insufficiency ($P = .0001$). Eighty-nine percent of patients had DeBakey type I aortic dissections with the ABVD group having more DeBakey type I dissections (100% vs 80%, $P < .001$). Other preoperative characteristics were similar between the ABVD and no ABVD groups (Table 1). Among the ABVD group, the innominate artery was the most commonly involved in the dissection (92%), whereas the right subclavian artery was the least commonly involved (14%) (Tables 1 and E1).

Intraoperative Data

Aortic dimensions at the root, ascending, mid-arch, and proximal descending were similar between groups at time of ATAAD repair (Table 2). The ABVD group had significantly more aortic root replacement (36% vs 27%) and longer aortic crossclamp times (153 vs 128 minutes, $P = .007$), but similar other concomitant procedures, cardiopulmonary bypass and hypothermic circulatory arrest times, cerebral perfusion strategies, and intraoperative units of packed red blood cells ($P > .05$) compared with the no ABVD group (Table 2). The ABVD group had more use of the axillary artery (38% vs 25%, $P = .02$) and less use of the innominate (3.0% vs 10%, $P = .01$) and intrathoracic right subclavian (2.3% vs 13%, $P = .001$) arteries compared with the no ABVD group with similar use of the femoral artery and aorta for cannulation for cardiopulmonary bypass (Table E2).

Postoperative Data

Overall, there were no significant differences in postoperative complications, including reoperation for bleeding, stroke (9.8% vs 4.9%, $P = .12$), new-onset renal failure, and operative mortality (6.8% vs 4.9%, $P = .51$) between the ABVD and no ABVD groups (Table 3). The no ABVD group had more bilateral strokes, and the ABVD group had more unilateral strokes, with 90% of all strokes being embolic in origin (Table 4). ABVD conferred a 2 times increased risk of postoperative stroke (odds ratio [OR], 2.2; 95% confidence interval [CI], 0.82-5.68; $P = .12$).

Midterm Outcomes

The completeness of follow-up for reoperation and long-term events was 83%, and the completeness of follow-up for survival was 100%. Our total follow-up time for long-term events was 895 patient-years. Mean follow-up time was 3.2 ± 3.0 years. Midterm transient ischemic attack and stroke were similar between the ABVD and no ABVD groups. The ABVD group had a significantly higher incidence rate of reoperation during follow-up (3.17 vs 1.03%/year, $P = .04$) (Table 5). The ABVD group had a significantly higher cumulative incidence of reoperation adjusting for death as a competing factor compared with the no ABVD group (8-year: 19% vs 4%, $P = .04$) (Figure 1, *A*). In patients with DeBakey type I dissections, the cumulative incidence of reoperation was still higher in the ABVD group (8-year: 19% vs 5%, $P = .11$) (Figure 1, *B*). In addition, ABVD was an independent risk factor for reoperation of the aortic arch and distal aorta in the Cox model (HR, 2.89; 95% CI, 1.01-8.27; $P = .048$), whereas age (HR, 1.03; $P = .76$), female gender (HR, 0.75; $P = .64$), and connective tissue disease (HR, 1.42; $P = .76$) were not significant risk factors for reoperation (Figure 1, *C*). The 8-year survival was similar between the ABVD (76% [95% CI, 63-85]) and no ABVD (74% [95% CI, 59-84], $P = .30$) groups (Figure 2).

DISCUSSION

In this study comparing patients with and without ABVD without cerebral MPS undergoing ATAAD repair with hemiarch replacement, patients with ABVD had similar perioperative outcomes, but a significantly higher incidence rate and cumulative incidence of reoperation on the aortic arch and distal aorta. In addition, ABVD was an independent risk factor for reoperation on the aortic arch and distal aorta (Video 1).

ATAAD requires emergency surgery because of the risk of (1) acute aortic insufficiency and subsequent acute heart failure, (2) malperfusion of end organs (brain, heart,

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TABLE 1. Demographics and preoperative outcomes

	Total (n = 276)	No ABVD (n = 143)	ABVD (n = 133)	*P* value
Patient age (y)	62 (52, 70)	62 (54, 71)	60 (50, 67)	.048
Sex, male	189 (68)	91 (64)	98 (74)	.07
BMI (kg/m^2)	28.3 (35.4, 33.2)	28.1 (24.6, 32.6)	28.7 (25.7, 33.3)	.31
Preexisting comorbidities				
Hypertension	206 (75)	110 (77)	96 (72)	.37
Diabetes	25 (9.1)	11 (7.8)	14 (11)	.41
Smoking status				.65
Never	115 (42)	56 (39)	59 (45)	
Former	72 (26)	39 (27)	33 (25)	
Current	88 (32)	48 (34)	40 (30)	
CAD	54 (20)	29 (21)	25 (20)	.76
COPD	30 (11)	18 (13)	12 (9.0)	.34
History of MI	16 (5.8)	7 (4.9)	9 (6.8)	.51
History of renal failure	8 (2.9)	4 (2.8)	4 (3.0)	1.0
History of CVA	10 (3.6)	5 (3.5)	5 (3.8)	1.0
PVD	52 (19)	20 (14)	32 (24)	.03
Connective tissue disorder	10 (3.6)	3 (2.1)	7 (5.3)	.20
Bicuspid aortic valve	23 (9.0)	13 (9.9)	10 (8.1)	.59
Previous cardiac surgery	19 (6.9)	11 (7.7)	8 (6.0)	.58
DeBakey classification				
I	247 (89)	114 (80)	133 (100)	<.0001
II	29 (11)	29 (20)	0 (0)	<.0001
Distal extent				
Ascending	29 (11)	29 (20)	0 (0)	<.0001
Arch	39 (14)	24 (17)	15 (11)	.19
Descending thoracic	35 (13)	21 (15)	14 (11)	.30
Abdominal	71 (26)	34 (24)	37 (28)	.44
Iliac(s)	96 (35)	32 (22)	64 (48)	<.0001
Unknown*	6 (2.2)	3 (2.1)	3 (2.6)	.93
Extent of ABVD				
Innominate	-	123 (92)	-	-
RCC	-	45 (34)	-	-
RSc	-	18 (14)	-	-
LCC	-	74 (56)	-	-
LSc	-	61 (46)	-	-
Preoperative AI				.0001
None	70 (27)	47 (34)	23 (19)	
Trace	29 (11)	17 (12)	12 (10)	
Mild	74 (28)	43 (31)	31 (25)	
Moderate	43 (16)	19 (14)	24 (19)	
Severe	45 (17)	11 (8.0)	34 (27)	
Ejection fraction	58 (55, 65)	58 (55, 65)	58 (55, 65)	.67
Acute MI	10 (3.6)	7 (4.9)	3 (2.3)	.34
Acute stroke	1† (0.4)	1 (0.7)	0 (0)	1.0
Acute renal failure	21 (7.6)	13 (9.1)	8 (6.0)	.34
Acute paralysis	5 (1.8)	3 (2.1)	2 (1.5)	1.0
Cardiogenic shock	29 (11)	16 (11)	13 (9.8)	.70
Preoperative creatinine	1.0 (0.8, 1.3)	1.0 (0.8, 1.2)	1.0 (0.8, 1.3)	.03
MPS	43 (16)	25 (17)	18 (14)	.37
Coronary	10 (3.6)	7 (4.9)	3 (2.3)	.34
Spinal cord	5 (1.8)	4 (2.8)	1 (0.8)	.37

(Continued)

TABLE 1. Continued

	Total (n = 276)	No ABVD (n = 143)	ABVD (n = 133)	*P* value
Celiac/hepatic	4 (1.5)	3 (2.1)	1 (0.8)	.62
Mesenteric	16 (5.8)	8 (5.6)	8 (6.0)	.88
Renal	13 (4.7)	5 (3.5)	8 (2.9)	.32
Lower extremity	20 (7.3)	9 (6.3)	11 (8.3)	.53
Delayed operation	28 (10)	14 (9.8)	14 (11)	.84

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *P* value indicates the difference between the no ABVD and ABVD groups. *ABVD*, Arch branch vessel dissection; *BMI*, body mass index; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *MI*, myocardial infarction; *CVA*, cerebrovascular accident; *PVD*, peripheral vascular disease; *RCC*, right common carotid; *RSc,* right subclavian; *LCC*, left common carotid; *LSc*, left subclavian; *AI*, aortic insufficiency; *MPS*, malperfusion syndrome. *Specific distal extent unable to be determined because of the limitation of the imaging studies: at least arch (n = 3), at least entire thoracic descending (n = 1), at least very upper abdomen (n = 1), and at least renal level (n = 1). †Anoxic brain injury during preoperative cardiac arrest.

viscera, limbs), (3) tamponade, and (4) aortic rupture, with the first priority being the patient's life. Replacement of dissected aortic branch vessels without malperfusion is not a goal in the emergency setting. We also frequently leave dissected visceral branches or iliac arteries alone unless there is malperfusion of those branches. It is controversial to replace the aortic arch and dissected arch branch vessels in the absence of malperfusion. We found that with hemiarch replacement, patients with ABVD had similar postoperative complications, including operative mortality (Table 3), as those with no ABVD. In addition, more patients in the ABVD group required aortic root

TABLE 2. Intraoperative data

	Total (n = 276)	No ABVD (n = 143)	ABVD (n = 133)	*P* value
Aortic dimensions at time of ATAAD (mm)*				
Root	44 (40, 50)	43 (39, 49)	45 (40, 51)	.12
Ascending	49 (46, 55)	49 (46, 55)	49 (45, 55)	.64
Arch	37 (34, 42)	37 (35, 42)	37 (33, 41)	.29
Descending	35 (31, 38)	35 (31, 39)	35 (31, 38)	.65
Aortic root procedure				.0035
None	18 (6.6)	16 (11)	2 (1.5)	
AVR only	5 (1.8)	4 (2.8)	1 (0.8)	
Root replacement	87 (32)	39 (27)	48 (36)	
Root repair	166 (60)	84 (59)	82 (62)	
Frozen elephant trunk	38 (14)	19 (13)	19 (14)	.81
Concomitant procedures				
CABG	16 (5.8)	9 (6.3)	7 (5.3)	.71
Mitral valve	1 (0.4)	1 (0.7)	0 (0)	1.0
Tricuspid valve	3 (1.1)	2 (1.4)	1 (0.8)	1.0
CPB time (min)	212 (176, 268)	210 (171, 260)	214 (185, 272)	.26
Crossclamp time (min)	138 (106, 187)	128 (97, 178)	153 (113, 200)	.007
HCA	276 (100)	143 (100)	133 (100)	
HCA time (min)	30 (23, 37)	30 (24, 37)	29 (23, 36)	.44
Cerebral perfusion				.74
None	0 (0)	0 (0)	0 (0)	
Antegrade	139 (50)	73 (51)	66 (50)	
Retrograde	122 (44)	61 (43)	61 (46)	
Both antegrade and retrograde	15 (5.4)	9 (6.3)	6 (4.5)	
Lowest temperature (°C)	18 (17.7, 21.8)	18.2 (17.8, 24)	18 (17.7, 19.9)	.035
Blood transfusion (PRBCs), units	2 (0, 5)	2 (0, 5)	2 (0, 4)	.82

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *P* value indicates the difference between the no ABVD and ABVD groups. *ABVD*, Arch branch vessel dissection; *ATAAD*, acute type A aortic dissection; *AVR*, aortic valve replacement; *CABG*, coronary artery bypass graft; *CPB*, cardiopulmonary bypass; *HCA*, hypothermic circulatory arrest; *PRBC*, packed red blood cells. *Dimensions were taken at the aortic sinus for the root, mid-ascending for the ascending, mid-arch for the arch, and proximal descending for the descending diameter.

TABLE 3. Postoperative data

	Total (n = 276)	No ABVD (n = 143)	ABVD (n = 133)	*P* value
Reoperation for bleeding	18 (6.5)	11 (7.7)	7 (5.3)	.41
Tamponade	3 (1.1)	1 (0.7)	2 (1.5)	.61
Deep sternal wound infection	1 (0.4)	1 (0.7)	0 (0)	1.0
Sepsis	7 (2.5)	5 (3.5)	2 (1.5)	.45
Postoperative MI	3 (1.1)	0 (0)	3 (2.3)	.11
Atrial fibrillation	103 (37)	56 (39)	47 (35)	.54
Cerebrovascular accident	20 (7.3)	7 (4.9)	13 (9.8)	.12
Location				
Left brain	4 (1.4)	1 (0.7)	3 (2.3)	1.0
Right brain	8 (2.9)	1 (0.7)	7 (5.3)	.16
Both sides	8 (2.9)	5 (3.5)	3 (2.3)	.06
Etiology				
Embolic	18 (6.5)	7 (4.9)	11 (8.3)	.52
Hemorrhagic	2 (0.7)	0 (0)	2 (1.5)	.52
Permanent*	8 (2.9)	2 (1.4)	6 (4.5)	.64
New-onset paraplegia	2 (0.7)	2 (1.4)	0 (0)	.50
New-onset acute renal failure	31 (11)	14 (9.8)	17 (13)	.43
Requiring dialysis	13 (4.7)	6 (4.2)	7 (5.3)	.68
Permanent	6 (2.2)	2 (1.4)	4 (3.0)	.43
Gastrointestinal complications	32 (12)	19 (13)	13 (9.8)	.36
Pneumonia	42 (15)	18 (13)	24 (18)	.21
Prolonged ventilation (>24 h)	154 (56)	78 (55)	76 (57)	.66
Hours intubated	38 (22, 90)	37 (22, 86)	43 (22, 94)	.43
Reintubation	14 (5.1)	9 (6.3)	5 (3.8)	.34
Tracheostomy	5 (1.8)	3 (2.1)	2 (1.5)	1.0
Postoperative LOS (d)	10 (7, 16)	10 (7, 15)	10 (7, 16)	.81
Total LOS (d)	11 (7, 17.5)	11 (7, 18)	11 (7, 17)	.68
Intraoperative mortality	1 (0.4)	0 (0)	1 (0.8)	.48
In-hospital mortality	15 (5.4)	6 (4.2)	9 (6.8)	.35
30-d mortality	13 (4.7)	5 (3.5)	8 (6.0)	.32
Operative mortality†	16 (5.8)	7 (4.9)	9 (6.8)	.51

Data presented as median (25%, 75%) for continuous data and n (%) for categorical data. *P* value indicates the difference between no arch vessel dissection and arch vessel dissection groups. *ABVD*, Arch branch vessel dissection; *MI*, myocardial infarction; *LOS*, length of stay. *Permanent stroke was defined as stroke not fully recovered at postoperative visit or before in-hospital death. †Operative mortality includes 30-day mortality and in-hospital mortality.

replacement and had longer aortic crossclamp times. The fact that there was no significant difference in postoperative stroke or mortality (Table 4) supported that hemiarch was probably adequate for patients with ABVD but no malperfusion. There was no significant difference in long-term survival between the ABVD and no ABVD groups (Figure 2), which further supports the adequacy of hemiarch replacement. Taken together, hemiarch replacement produced adequate short-term outcomes in patients with and without ABVD but no malperfusion.

The stroke rate was twice as high in the ABVD group, although not significant (9.8% vs 4.9%, $P = .12$). Most strokes (5/7) in the no ABVD group were bilateral (Table 3), indicating those patients probably had an underlying general bilateral cerebral vascular pathology. However, the ABVD group had more unilateral strokes (10/13) and seemed to have more specific strokes related to the dissected arch branch vessels (Table 4), indicating that the dissection of arch branch vessels could be the cause of the strokes in the ABVD group. The OR of ABVD for stroke was 2.2 ($P = .12$), which also supported that ABVD could be a major cause of postoperative strokes in patients with ABVD. Most strokes were embolic (Table 4). The potential mechanism could be that the thrombus in the false lumen of

TABLE 4. Details of postoperative stroke among the arch branch vessel dissection group

	All ABVD (n = 133)	Isolated right-sided dissection (n = 59)	Isolated left-sided dissection (n = 10)	Bilateral dissection (n = 64)	*P* value
Stroke	13 (9.8)	2 (3.4)	2 (20)	9 (14)	.07
Location					
Left brain	3 (2.3)	0 (0)	0 (0)	3 (4.7)	1.0
Right brain	7 (5.3)	2 (3.4)	1 (10)	4 (6.3)	.71
Both sides	3 (2.3)	0 (0)	1 (10)	2 (3.1)	.71
Etiology					
Embolic	11 (8.3)	2 (3.4)	1 (10)	8 (13)	.54
Hemorrhagic	2 (1.5)	0 (0)	1 (10)	1 (1.6)	.54
Permanent*	6 (4.5)	1 (1.7)	1 (10)	4 (6.3)	1.0

Isolated right-sided: innominate or right common carotid artery dissection. Isolated left-sided: left common carotid artery dissection. Bilateral: innominate/right common carotid and left common carotid artery dissection. Univariate comparisons were performed using chi-square tests for categorical data and Wilcoxon rank-sum tests for continuous data. *ABVD*, Arch branch vessel dissection. *Permanent stroke was defined as stroke not fully recovered at postoperative visit or before in-hospital death.

the dissected arch branch vessels dislodged during intraoperative manipulation of those vessels resulting in embolic strokes in those patients. This mechanism could explain the greater incidence of right-sided strokes in patients with innominate artery and LCC artery dissection, because we used unilateral (right) cerebral perfusion in a majority of cases using antegrade cerebral perfusion (84%, 129/154), often with more manipulation of the innominate artery. Surgeons should minimize any manipulation of dissected arch branch vessels to decrease postoperative stroke rates in those patients.

Does more aggressive arch replacement improve short-term outcomes, such as a decreased rate of postoperative stroke? From our previous study[14] comparing hemiarch replacement and Zone 1/2/3 arch replacement among patients with ABVD without cerebral malperfusion, the postoperative stroke rate was similar between arch management strategies (11% vs 10%), as were other postoperative outcomes including operative mortality (8.2% vs 7.5%). In addition, aggressive arch replacement was not a significant risk factor for postoperative stroke (OR, 0.9) or operative mortality (OR, 0.7). Those findings indicate that aggressive arch replacement in patients with ABVD without malperfusion did not improve or worsen the perioperative stroke rate. Likewise, Rice and colleagues[19] found that total arch replacement had similar postoperative outcomes as hemiarch replacement. Performing an aggressive arch replacement compared with a hemiarch replacement results in similar short-term outcomes; however, additional factors than improved short-term outcomes contribute to the decision regarding arch management.

In addition to short-term survival in patients with ATAAD, surgeons must consider future reoperations, which is why some surgeons advocate a more extensive initial operation in regard to the aortic arch. Omura and colleagues[20] found that patients undergoing total arch replacement had significantly lower rates of distal aortic events (freedom from reoperation: 95% vs 84% at 5 years,

TABLE 5. Complications and reoperations during midterm follow-up

	No ABVD		ABVD		
	n = 125	Incidence rate (%/y)	n = 108	Incidence rate (%/y)	*P* value
TIA	3	0.65	0	0	.29
Stroke	1	0.29	1	0.24	1
Reoperation primarily for					
Arch aneurysm	0	0	4	0.97	.09
TAA/A	5	1.03	9	2.19	.27
Surgery type					
Endovascular	1	0.21	3	0.73	.51
Median sternotomy	0	0	4	0.97	.09
Open TAA/A repair	4	0.83	6	1.46	.56
Total procedures	5	1.03	13	3.17	.04

P value indicates the difference between no arch vessel dissection and arch vessel dissection groups. *ABVD*, Arch branch vessel dissection; *TIA*, transient ischemic attack; *TAA*, thoracic aortic aneurysm; *TAAA*, thoracoabdominal aortic aneurysm.

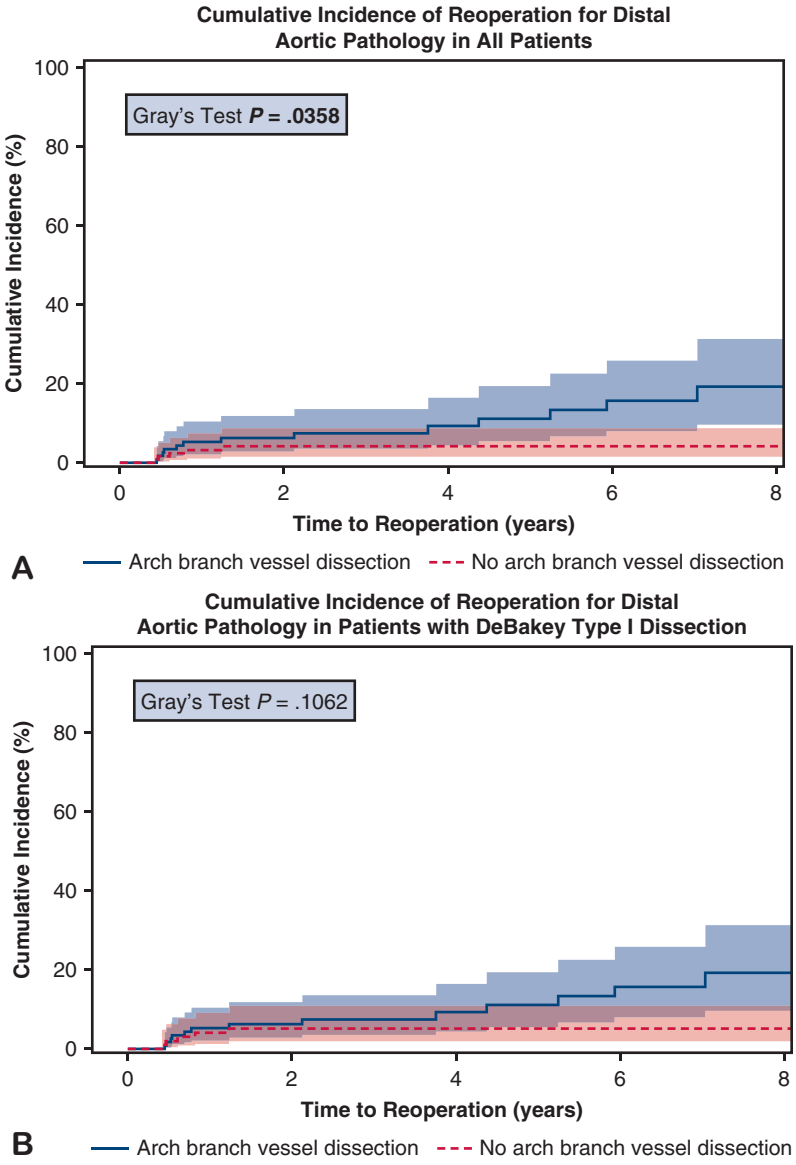


FIGURE 1. A, The cumulative incidence of reoperation for distal aortic pathology in all patients, including the distal arch, descending thoracic, and thoracoabdominal aorta, after ATAAD repair in patients with and without ABVD without cerebral or upper-extremity MPS undergoing hemiarch replacement. Death was treated as a competing factor. The 8-year cumulative incidence of reoperation was significantly higher in the ABVD group (8-year: 19.2% vs 4.1%, $P = .04$). B, The cumulative incidence of reoperation for distal aortic pathology in patients with DeBakey I dissection only after ATAAD repair with hemiarch replacement. Death was treated as a competing factor. The 8-year cumulative incidence of reoperation was higher in the ABVD group (8 years: 19.2% vs 5.1%, $P = .11$). C, Cox proportional hazard regression determined ABVD (HR, 2.89; 95% CI, 1.01-8.27; $P = .048$) was an independent risk factor for reoperation for distal aortic pathology, including distal arch, descending thoracic, and thoracoabdominal aortas, after ATAAD repair with hemiarch replacement, whereas age, gender, and connective tissue disease were not risk factors. *ABVD*, Arch branch vessel dissection.

$P = .01$) compared with those with hemi- and partial arch replacement, whereas we have previously found that rates of reoperation were similar between hemiarch and aggressive arch groups among all patients undergoing open ATAAD repair (10-year cumulative incidence: 14% vs 12%, $P = .89$).[8] However, when focusing on patients undergoing ATAAD repair with ABVD, we found that hemiarch replacement had a trend of higher cumulative incidence of reoperation compared with aggressive arch replacement (8-year: 23% vs 9% in zone 1/2/3 arch group, $P = .33$) but did not reach significant difference most likely because of the relatively small sample size.[14] In this study, looking at only patients undergoing hemiarch replacement with and without ABVD, those with ABVD had a

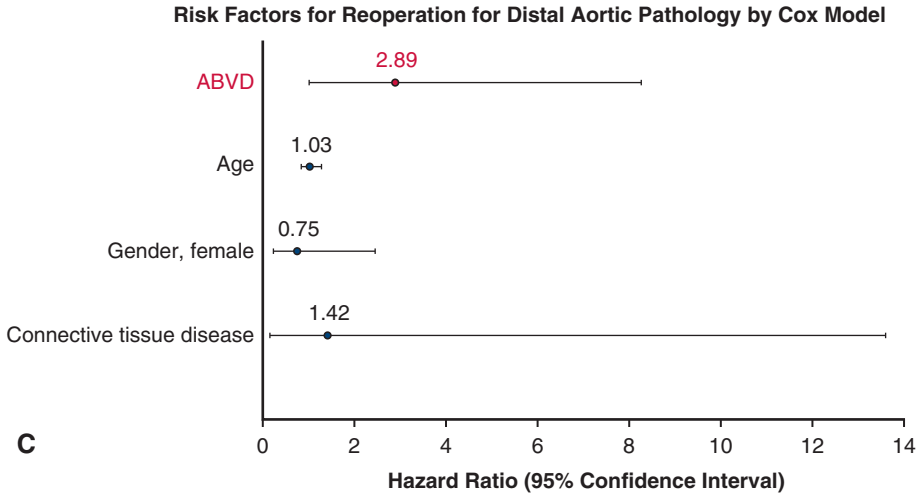


FIGURE 1. (*continued*).

significantly greater cumulative incidence of reoperation for aortic arch and distal aortic pathology (8-year: 19% vs 4% in no ABVD group, $P = .04$) with ABVD being an independent risk factor for reoperation (HR, 2.89, $P = .048$). Among only patients with DeBakey type I dissection, the difference in cumulative incidence of reoperation was similar (19% vs 5%, Figure 1, *B*), but not significant ($P = .11$), likely due to small sample size. These results supported that with ABVD, the aorta with residual dissection behaved differently than those without ABVD.

The reoperation for those patients with ABVD after hemiarch replacement was needed for the expanding aneurysms of the dissected aortic arch/descending thoracic aorta. The potential mechanism of continuous expansion of the distal aortic aneurysm could be that the dissected arch branch vessel continued to retrograde perfuse the false lumen of the aortic arch and descending thoracic aorta. This

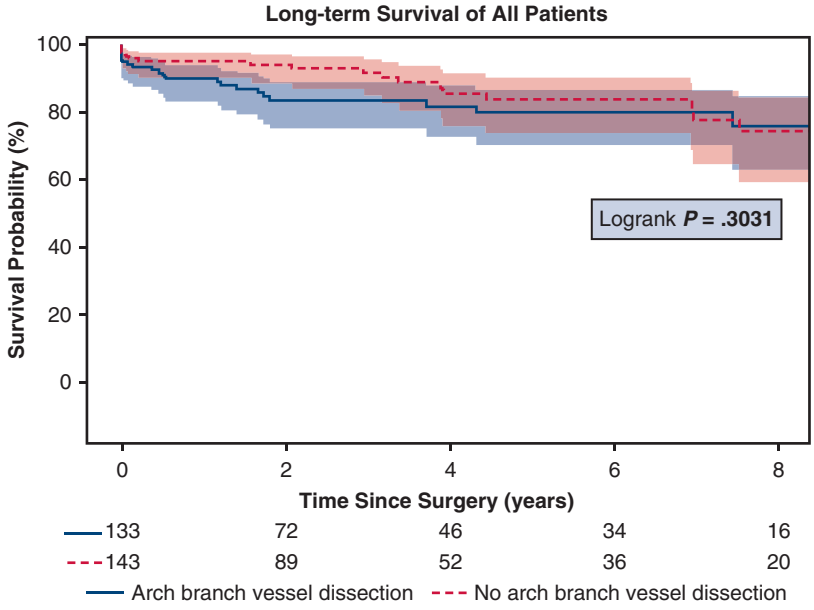
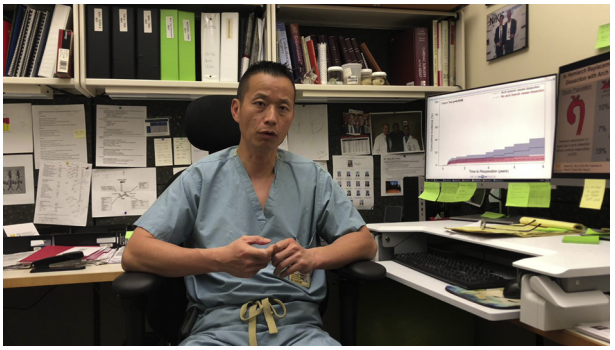


FIGURE 2. Kaplan–Meier survival analysis of patients with and without ABVD without cerebral or upper-extremity MPS undergoing hemiarch replacement in ATAAD repair. The 8-year survival was similar between ABVD (76%, 95% CI, 63-85) and no ABVD (74%, 95% CI, 59-84, $P = .30$) groups.



VIDEO 1. Discussion of hemiarch repair in ATAAD repair in patients with ABVD without associated malperfusion. Video available at: https://www.jtcvs.org/article/S0022-5223(20)33297-9/fulltext.

persistent blood flow in the false lumen resulted in the continuous expansion of the false lumen of the aortic arch and proximal descending thoracic aorta. We have seen this phenomenon in patients with ABVD treated with the branched single anastomosis frozen elephant trunk repair (B-SAFER procedure).[21] Once the dissected arch branch vessels are replaced with aggressive arch replacement, there is no more persistent flow from the arch branch vessels into the false lumen of descending thoracic aorta. That is likely why aggressive arch replacement with replacement of the dissected arch branch vessels decreases the late reoperation rate in this patient population compared with hemiarch replacement.[14,20] Another speculation is that patients with ABVD could have more severe aortopathy/vasculopathy that resulted in continuous expansion of the distal aortic aneurysm. ABVD could be a sign associated with more severe aortopathy/vasculopathy as the ABVD group had a more than 2 times higher prevalence of connective tissue disease in this study (Table 1) and in another report.[21] Combined with the results from our previous study,[14] more aggressive arch replacement than hemiarch replacement could be considered by experienced surgeons in stable patients who are young with ABVD to prevent future reoperations given aggressive arch replacement does not increase operative mortality but could prevent future reoperations.[14]

Study Limitations

This study was limited as a single-center and retrospective experience. Because follow-up for reoperation was not 100% complete, the incidence of reoperation could be underestimated in both groups. In addition, the follow-up time was relatively short and the sample size was relatively small, which may have caused some type II error. At our institution, aortic surgeons primarily performed ATAAD repairs, and endovascular amenable MPS (visceral, lower extremity) was managed endovascularly before open aortic repair; therefore, our experience may not apply to all hospitals performing ATAAD repair.

CONCLUSIONS

Hemiarch replacement was adequate for ATAAD repair in patients with ABVD but no cerebral malperfusion. More aggressive arch management could be considered for select patients to prevent late reoperations (Figure 3).

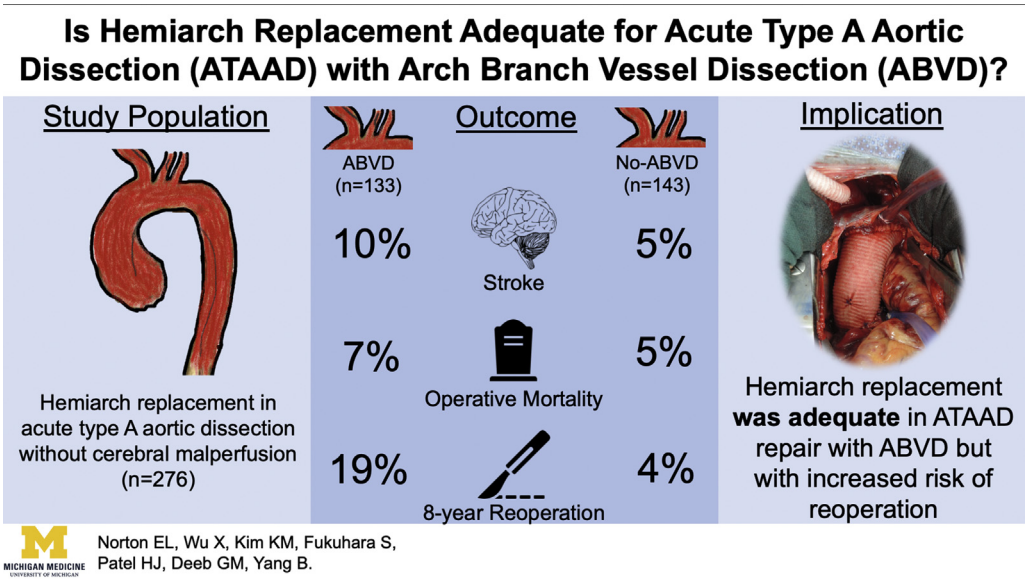


FIGURE 3. Hemiarch replacement is adequate in ATAAD repair in patients with ABVD without associated MPS but with an increased risk of reoperation on the aortic arch and distal aorta.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://aats.blob.core.windows.net/media/20AM/Presentations/Is%20Arch%20Branch%20Vessel%20Dissection%20wit.mp4.

Conflict of Interest Statement

H.J.P. is a consultant for WL Gore and Associates, Edwards, and Medtronic, and these efforts are modest. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: acute aortic dissection, aortic arch management, arch branch vessel dissection

Discussion

Presenter: Elizabeth L. Norton

Dr Rita C. Milewski *(Philadelphia, Pa)*. This timely analysis addresses several key questions in management of these patients. The specific repair for patients with catastrophic type A dissection must involve consideration of both the short-term and long-term outcomes. Two major questions in approaching type A dissection are what specific procedure should be performed to decreased stroke rate and at the same time decrease the need for further aortic reoperation? To address the issue of stroke after type A hemiarch repair, in your study, why do you think patients with ABVD treated with hemiarch replacement had a 2-fold higher stroke rate than patients without ABVD, though not significant?

Ms Elizabeth L. Norton *(Omaha, Neb)*. The real reasons for the higher postoperative stroke rate you mentioned are unknown. However, possible explanations could be a change in flow pattern, with the dissected branch vessel causing a pulsatile flow and then once being put on bypass more of a continuous flow and just that change in flow pattern. Another possibility would be a thrombus due to that dissection in the interrupted aortic arterial wall. In addition, during surgery

the manipulation of the head vessels could cause any thrombus to dislodge. Although we don't know the true reason for the increased stroke rate, I think those would be possibilities.

Dr Milewski. Recent studies have addressed both the issue of stroke and long-term aortic reoperation in type A dissection. A large study using the Society of Thoracic Surgeons database by _?_ reported that total arch replacement was associated with greater risk for stroke versus hemiarch. Others, including Dr Desai from Penn and your group from Michigan, after adjusting for anatomic complexity, have reported no increased risk in stroke with total arch and a trend to decreased aortic reoperations. Because patients with ABVD treated with hemiarch had higher stroke rates, do you recommend all patients with arch branch muscle dissection have aggressive arch replacement and replacement of dissected arch branch vessels, a zone 1/2/3 arch?

Ms Norton. ABVD and MPS (cerebral or upper extremity) are indications for an aggressive arch replacement to restore flow. However, in patients with ABVD without malperfusion, the study shows that a hemiarch replacement is adequate. An aggressive arch replacement does not decrease the rate of postoperative stroke.

We had a previous study that compared hemiarch replacement with aggressive arch replacement in a cohort of patients with ABVD, and you can see that the stroke rate and the operative mortality are similar between the hemiarch and aggressive arch replacement groups. The stroke rate was 11% in the hemiarch group and 10% in the aggressive arch group.

The aggressive arch replacement does not improve short-term outcomes. However, it could prevent future reoperations. Also in this study, we looked at reoperations between hemiarch and zone 1/2/3 arch replacements and found that the hemiarch group had a reoperation rate of 23% at 8 years compared with 9% in those with an aggressive arch replacement. Therefore, an aggressive arch replacement does not improve short-term outcomes. However, it could have a benefit in preventing future long-term reoperations.

Dr Milewski. Certainly all cardiac surgeons are fully trained to care for all emergencies. However, given the data that you presented, what do you think a nonaortic cardiac surgeon should do when he or she is required to do an operative repair in a patient with type A dissection with ABVD without cerebral or upper-extremity malperfusion?

Ms Norton. In that scenario, hemiarch would be appropriate, especially for cardiac surgeons (not specialized aortic surgeons) who may be unfamiliar with an aggressive arch replacement—and those who don't perform ATAAD repairs often. The hemiarch replacement can save a patient's life, just recognizing the fact that there is a higher risk of reoperation in the future.

However, if a surgeon is experienced and comfortable with the procedure—per se an aortic surgeon, an aggressive arch replacement could be performed to prevent future reoperations. However, no immediate benefit in the short term would be applicable.

Dr Milewski. I agree. Great answer.

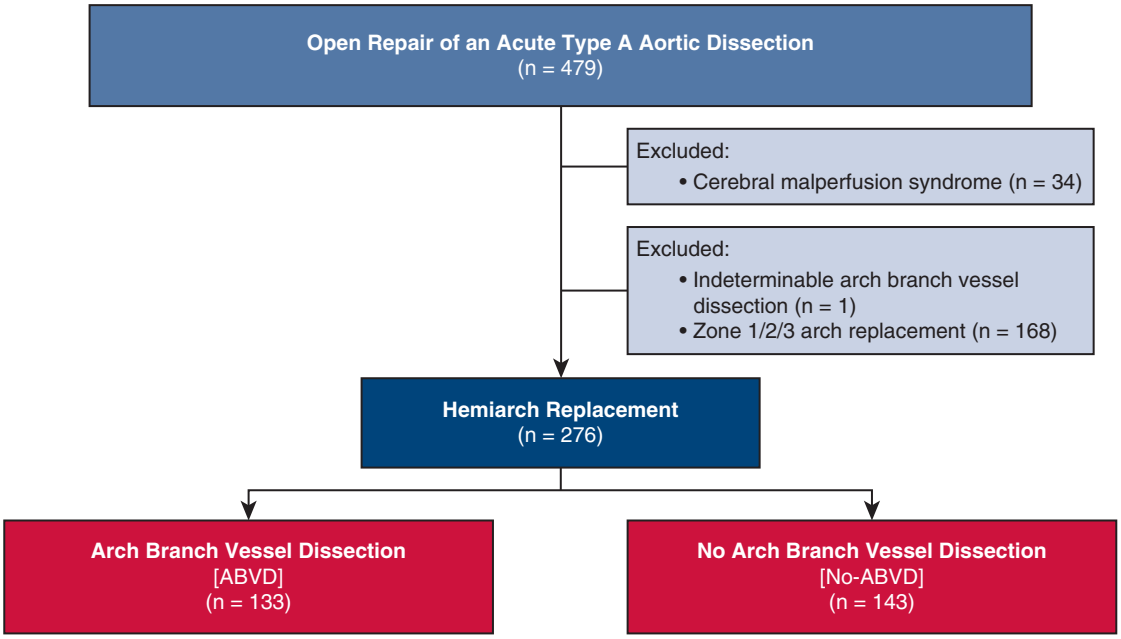

FIGURE E1. Algorithm of selection of study population.

TABLE E1. Extent of arch branch vessel dissection

	ABVD (n = 133)
Innominate	36 (27)
LCC	7 (5.3)
Innominate + LCC	7 (5.3)
Innominate + LCC + LSc	27 (20)
Innominate + LSc	4 (3.0)
Innominate + RCC	6 (4.5)
Innominate + RCC + LCC	9 (6.8)
Innominate + RCC + LCC + LSc	12 (9.0)
Innominate + RCC + LSc	4 (3.0)
Innominate + RCC + RSc	5 (3.8)
Innominate + RCC + RSc + LCC + LSc	7 (5.3)
Innominate + RCC + RSc + LSc	2 (1.5)
Innominate + RSc	2 (1.5)
Innominate + RSc + LCC + LSc	2 (1.5)
LCC + LSc	3 (2.3)

Data presented as n (%). *ABVD*, Arch branch vessel dissection; *LCC*, left common carotid; *LSc*, left subclavian; *RCC*, right common carotid; *RSc*, right subclavian.

TABLE E2. Cannulation strategy for cardiopulmonary bypass

	All patients (n = 276)	ABVD (n = 133)	No ABVD (n = 143)	*P* value
Axillary	87 (32)	51 (38)	36 (25)	.02
Aortic	33 (12)	16 (12)	17 (12)	.97
Innominate	19 (6.9)	4 (3.0)	15 (10)	.01
Right subclavian	21 (7.6)	3 (2.3)	18 (13)	.001
Femoral	113 (41)	58 (44)	55 (38)	.39
Axillary and aortic	1 (0.4)	0 (0)	1 (0.7)	1.0
Axillary and femoral	2 (0.4)	1 (0.8)	1 (0.7)	1.0

Data presented as n (%). *ABVD*, Arch branch vessel dissection.