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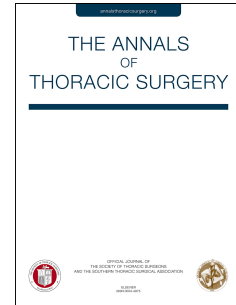
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Direct Aortic versus Peripheral Arterial Cannulation in Surgery for Type-A Aortic Dissection

Running head: Direct aortic cannulation

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Abstract

Background: It is controversial whether peripheral arterial cannulation may achieve better results than direct aortic cannulation during surgery for Stanford type A aortic dissection (TAAD).

Methods: Three-hundred and nine consecutive patients underwent surgical repair for acute TAAD from January 2005 to December 2017 at the Helsinki University Hospital, Finland. The early outcomes of patients who underwent surgery with direct aortic cannulation were compared with those in whom peripheral arterial cannulation was employed.

Results: Direct aortic cannulation was employed in 80 patients and peripheral arterial cannulation in 229 patients. Patients who underwent surgery with direct aortic cannulation had hospital mortality (13.8% vs. 13.5%, $p=0.962$) and stroke/global brain ischemia (22.3% vs. 25.0%, $p=0.617$) similar to those with peripheral arterial cannulation. The other secondary outcomes were equally distributed between the unmatched study cohorts. Among 74 propensity score matched pairs, direct aortic cannulation had hospital mortality (12.2% vs. 9.5%, $p=0.804$) and stroke/global brain ischemia rates (21.6% vs. 21.6%, $p=1.000$) comparable to peripheral arterial cannulation. The composite outcome of hospital mortality/stroke/global brain ischemia (29.7% vs. 27.0%, $p=0.855$), multiple stroke (16.2% vs. 17.6%, $p=1.000$), renal replacement therapy (11.8% vs. 13.0%, $p=1.000$) and length of stay in the intensive care unit (mean, 4.9 ± 4.5 vs. 4.8 ± 4.9 days, $p=0.943$) were also equally distributed between these matched cohorts.

Conclusions: In this institutional series, central arterial cannulation allowed a straightforward surgical repair of TAAD and achieved similar early outcomes to those of peripheral arterial cannulation.

Key-words: Aortic dissection; Type A aortic dissection; Aortic cannulation; Central cannulation; Peripheral cannulation.

Surgery for acute Stanford Type A aortic dissection (TAAD) is associated with substantial mortality and morbidity (1,2), which are attributable to concomitant cerebral, splanchnic and renal ischemia. Perfusion strategy is therefore a key issue for adequate end-organ protection during surgery for TAAD. It is controversial whether peripheral arterial cannulation is superior to direct aortic cannulation in preserving end-organ blood flow during surgery (3-6). The main argument against the use of direct aortic cannulation is the risk of suboptimal insertion of the arterial cannula leading to intraoperative malperfusion and progression of the dissection flap or even aortic rupture. On the other hand, the straightforward nature of direct aortic cannulation expedites surgical repair of TAAD, which may prove beneficial in hemodynamically unstable patients. This study was performed to investigate the benefits and harms of these two cannulation strategies during surgical repair for TAAD.

Patients and Methods

Three-hundred and nine consecutive patients underwent surgical repair for acute TAAD from January 2005 to December 2017 at the Helsinki University Hospital, Finland, and they are the subjects of this analysis. Permission to conduct this study was obtained by the Institutional Review Board. Data was retrospectively collected into an electronic datasheet with prespecified variables by two coauthors (C.M., H.P.M.) and underwent checking of its completeness and consistency by other two coauthors (M.I, P.R.). Clinical variables were defined according to the EuroSCORE II criteria (7). Operative risk was estimated according to the EuroSCORE II (8) and the Penn classification (8).

Cannulation technique

Direct aortic cannulation was accomplished by inserting the cannula into the true lumen of the ascending or aortic arch through two purse-string sutures reinforced with Teflon pledgets if required. Since June 2016, the arterial cannula was increasingly used with the Seldinger technique. The optimal position of the arterial cannula was verified by transesophageal and epiaortic ultrasound by checking first the correct position of the guidewire and after that of the arterial cannula into the true lumen of the aorta. The aorta was clamped, and systemic cooling was started. Myocardial protection was achieved using intermittent, antegrade and retrograde cold blood cardioplegia. When a target systemic temperature of 18-20°C was achieved, an ice

pack was put on patient's head, the aortic arch was explored, and the distal aortic repair was performed with or without antegrade cerebral perfusion. In 23 patients, the distal aortic anastomosis was performed with a clamp on site after having assessed the integrity of the aortic arch during a brief period of hypothermic circulatory arrest. When the distal aortic repair was accomplished, arterial blood flow was re-established through a side branch of the aortic Dacron prosthesis, rewarming was started, and the proximal aortic repair was performed.

Peripheral arterial cannulation was performed through an 8 mm Dacron prosthesis sutured to the peripheral artery. The site of peripheral arterial cannulation was according to the individual surgeon's preference and computed tomography findings. Cardiopulmonary bypass was started and the procedure was carried out as described above.

Outcomes

The primary outcomes of this study were hospital mortality and stroke/global brain ischemia. The secondary outcomes were multiple brain ischemic injuries, reoperation for bleeding, postoperative mechanical circulatory support, deep sternal wound infection/mediastinitis, acute kidney injury, and intensive care unit stay as well as a composite outcome including hospital mortality/stroke/global brain ischemia.

Hospital mortality was defined as all-cause death occurred during the index hospitalization. Stroke was defined as any focal (when limited to a specific vascular territory of the brain) or global (when diffusely involving several areas of the brain) neurological syndrome occurring during the index hospitalization caused by ischemia and/or hemorrhage not resolving within 24 h. The diagnosis and nature of stroke was based on computed tomography findings and was confirmed by a neurologist. Computed tomography of the brain was routinely performed in all patients with focal symptoms as well as in those with unconsciousness, delirium and disorientation. Multiple brain ischemic injury was defined as the presence of several focal ischemic findings in different regions of the brain in computed tomography. The severity of postoperative brain ischemic injury was assessed using the modified Rankin score. Deep sternal wound infection/mediastinitis was defined as an infection involving the deep tissues/sternal bone, and/or the mediastinum (9). Acute kidney injury was defined according to the KDIGO classification criteria (10).

Statistical Analysis

Statistical analyses were performed using Stata v. 15.1 (StataCorp LLC, Texas, USA) and SPSS v. 25.0 (IBM Corporation, New York, USA) statistical softwares. Continuous variables are reported as means and standard deviation, and categorical variables as counts and percentages. The Mann-Whitney, Fisher's exact and Chi-square tests were used for univariate analysis in the overall series. An acceptable balance between the cohorts was defined as standardized difference <0.10 . Since standardized differences of a few covariates were higher than 0.10 (Tables 1,2), one-to-one propensity score matching was performed using a caliper width of 0.2 the standard deviation of the logit. Propensity score was estimated using a non-parsimonious logistic regression model including all the covariates listed in Table 1 as well as aortic root replacement, partial/total arch repair and concomitant major cardiac procedure. Differences in preoperative and operative covariates as well as early outcomes were assessed using the McNemar test and the *t*-test for paired samples. Statistical significance was set at $p<0.05$.

Results

During the study period, 309 consecutive patients (mean age 61.7 ± 13.2 years; females 33.0%; mean EuroSCORE II $13.9\pm 13.3\%$) underwent surgical repair of TAAD at our Institution. The annual caseload of TAAD varied from 16 to 38/year (Supplemental figure 1). Surgery was performed by 24 surgeons with an individual volume ranging from one to 28 cases operated during the study period (nine surgeons operated ≥ 15 patients). The baseline characteristics and operative data of these patients are summarized in Tables 1 and 2. In the overall series, hospital mortality was 13.6% and stroke/global brain ischemia 23.9%. The other secondary outcomes are summarized in Table 3.

Direct aortic cannulation was employed in 80 patients, and peripheral arterial cannulation in 229 patients. Direct aortic cannulation strategy was adopted in 6.7% of patients before 2016 and in 76.5% of patients later on (2016, 50.0%; 2017, 76.3%; 2018, 100%) (Supplemental figure 1). Direct aortic cannulation using the Seldinger technique was employed in 42 patients (52.5%) (Table 1). Three patients underwent cannulation of both the ascending aorta and the common femoral artery, and they were included in the direct cannulation cohort. No major problems were encountered during direct aortic cannulation.

In the peripheral arterial cannulation cohort, the common femoral artery was the cannulation site in 62.0% of patients and the axillary/subclavian artery in 41.9% of patients. Cannulation of two peripheral arteries was employed in 11 patients. Other peripheral arteries were the site of peripheral cannulation in two patients.

Outcomes in the Overall Series

The direct aortic cannulation and peripheral arterial cannulation cohorts had comparable operative risk, but a few baseline and operative covariates were not perfectly balanced between the study cohorts (Tables 1,2). Patients who underwent surgery with direct aortic cannulation had hospital mortality (13.8% vs. 13.5%, $p=0.962$) and stroke/global brain ischemia (25.0% vs. 22.3%, $p=0.617$) comparable to those with peripheral arterial cannulation (Table 3). The intensive care unit stay was significantly shorter in the direct aortic cannulation group (mean, 4.9 ± 4.5 vs. 6.7 ± 6.8 days, $p=0.037$). The other secondary outcomes were comparable in the study cohorts (Table 3).

Outcomes in Propensity Score Matched Cohorts

Propensity score matching yielded 74 pairs with similar baseline characteristics (Table 4). However, matching resulted in marginally high standardized differences for pulmonary disease, Penn classification, type of aortic root procedure and open distal anastomosis without significant differences in paired test (Tables 4,5).

Direct aortic cannulation had hospital mortality (12.2% vs. 9.5%, $p=0.804$) and stroke/global brain ischemia rates (21.6% vs. 21.6%, $p=1.000$) similar to peripheral arterial cannulation (Table 6). The composite outcome of hospital mortality/stroke/global brain ischemia (29.7% vs. 27.0%, $p=0.855$), multiple stroke (16.2% vs. 17.6%, $p=1.000$), renal replacement therapy (11.8% vs. 13.0%, $p=1.000$) and intensive care unit stay (mean, 4.9 ± 4.5 vs. 4.8 ± 4.9 days, $p=0.943$) were also equally distributed between these matched cohorts (Table 6, Figure 1).

Outcomes after Direct Aortic Cannulation with the Seldinger Technique

Patients who underwent direct aortic cannulation with the Seldinger technique (42 patients) had comparable operative risk (EuroSCORE II $11.4\pm 9.2\%$ vs. $16.8\pm 19.4\%$, $p=0.566$) to those without the Seldinger

technique (38 patients). Direct aortic cannulation with the Seldinger technique resulted in comparable hospital mortality (5/42 vs. 6/38 patients, 11.9% vs. 15.8%, $p=0.749$), stroke/global brain ischemia (9/42 vs. 11/38 patients, 23.8% vs. 28.9%, $p=0.621$) and composite outcome of hospital mortality/stroke/global brain ischemia (11/42 vs. 15/38 patients, 28.6% vs. 39.5%, $p=0.303$) to those without the Seldinger technique. The other secondary outcomes were also equally distributed between the matched cohorts.

Comment

The present findings suggest that direct aortic cannulation in surgery for TAAD achieved comparable early outcomes to peripheral arterial cannulation. Propensity score matching analysis showed that, when the prevalence of critical conditions such as preoperative acute neurological events, cardiogenic shock and aortic rupture were balanced between the study cohorts, direct aortic cannulation was safe during surgical repair of TAAD. These two cannulation strategies yielded similar rates of hospital mortality and stroke/global brain ischemia. Importantly, we observed similar rates of multiple stroke and renal replacement therapy in this series. These findings substantiate the hypothesis that restoring blood flow through the true lumen of the aorta is feasible through central cannulation and this may effectively prevent ischemic injury of end-organs. Based on these results, direct aortic cannulation is currently used in all patients undergoing surgery for TAAD at our institution.

The safety of direct aortic cannulation has been documented in a few previous studies. In 2009, Kamiya et al. (11) showed that direct aortic cannulation achieved comparable rate of 30-day mortality (14% vs. 23%, $p=0.07$) and of stroke (4.9% vs. 4.5%, $p=0.09$) to femoral artery cannulation. Reece et al. (6) reported on significantly lower 30-day mortality (0% vs. 17%, $p=0.04$) with direct aortic cannulation compared with axillary and femoral artery cannulation.

Kreibich et al. (12) recently reported on 355 TAAD patients who underwent surgery with direct aortic cannulation and whose outcome was compared with 101 patients with axillary artery cannulation and 128 patients with femoral artery cannulation. After inverse probability weighting, similar rates of hospital mortality, stroke and renal replacement therapy were observed in these three study cohorts. These authors observed that direct aortic cannulation was associated with shorter aortic cross-clamping and cardiopulmonary bypass duration.

On the contrary, Sabashnikov et al. (5) reported on trend for increased 30-mortality (35.3% vs. 13.7%, $p=0.074$) after direct aortic cannulation compared to axillary artery cannulation.

It is worth noting that the rate of stroke and global brain ischemia in the present series is somewhat high (24%). Other series (6) reported on high rate of neurological complications as well. In our series this was likely attributable to a rather high prevalence of preoperative acute neurological events (23.7%) as well as to our institutional strategy to perform brain computed tomography in all patients with postoperative neurological derangement of any severity.

Limitations

The retrospective nature is the main limitation of this study because does not allow a throughout evaluation of the possible contraindications to direct aortic cannulation encountered in this series. However, when this cannulation technique was implemented at our institution, direct aortic cannulation was employed in 76.5% of patients. Secondly, direct aortic cannulation was used during the most recent years and we cannot exclude the confounding effect of changes in perioperative care over time. Thirdly, the Seldinger technique was employed only recently in our series and it is not clear whether this adjunct might have optimized the arterial cannulation of the aorta and contributed to improved results. However, from a clinical point of view, the routine use of transesophageal ultrasound might have warranted the correct position of the arterial cannula even when the Seldinger technique was not used. Fourthly, this series is not adequately powered to reliably detect a treatment effect. Indeed, a non-inferiority study assuming hospital mortality rates of 13.5% and 13.8% for the treatment groups with a non-inferiority limit of 5% (α 5%, β 80%) would require the recruitment of 540 patients per group. Fifthly, we attempted to adjust the results for baseline characteristics using the propensity score method, but we cannot exclude that several patients underwent direct aortic cannulation because of severe unstable hemodynamic conditions. This may introduce a significant bias, particularly when analyzing the outcome of patients treated before the implementation of the direct aortic cannulation strategy. Sixthly, surgery was performed by a rather large number of surgeons with a small individual volume, which might affect the outcome of these patients as well as be a source of bias in this analysis. Finally, the limited size of this series does not allow a separate analysis of the prognostic impact of

different peripheral arterial cannulation strategies. A larger study population is needed to address this important issue.

Conclusions

This institutional series showed that direct aortic cannulation allowed a straightforward surgical repair of TAAD and achieved similar early outcomes to those of peripheral arterial cannulation. These findings seem to substantiate the results of a few previous studies, which demonstrated the safety of the direct aortic cannulation strategy in patients with TAAD.

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Table 1. Baseline characteristics of patients who underwent surgical repair for type-A aortic dissection.

<i>Covariates</i>	<i>Overall dataset 309 patients</i>	<i>Peripheral cannulation 229 patients</i>	<i>Direct aortic cannulation 80 patients</i>	<i>Standardized difference</i>	<i>P-value</i>
Age (years)	61.7±13.2	61.0±12.8	63.7±14.0	0.203	0.046
Octogenarians	17 (5.5)	11 (4.8)	6 (7.5)	0.112	0.363
Female	102 (33.0)	67 (29.3)	35 (43.8)	0.304	0.018
Body mass index (kg/m ²)	27.6±5.6	27.8±5.8	27±5.1	0.138	0.462
Hemoglobin (g/L)	128±18	129±18	127±18	0.138	0.498
eGFR (mL/min/1.73 m ²)	74.5±33	74±34	75±30	0.912	0.082
Bicuspid aortic valve	26 (8.4)	17 (7.4)	9 (11.3)	0.132	0.289
Previous cardiac surgery	13 (4.2)	10 (4.4)	3 (3.8)	0.031	0.554
Peripheral vascular disease	13 (4.2)	7 (3.1)	6 (7.5)	0.200	0.088
Diabetes	25 (8.1)	18 (7.9)	7 (8.8)	0.032	0.482
Stroke	17 (5.5)	9 (3.9)	8 (10.0)	0.240	0.044
Pulmonary disease	22 (7.1)	16 (7.0)	6 (7.5)	0.020	0.878
Coronary artery disease	29 (9.4)	22 (9.6)	7 (8.8)	0.030	0.821
Acute myocardial infarction	32 (10.4)	27 (11.8)	5 (6.3)	0.194	0.115
LVEF ≤50%	57 (19.3)	42 (19.3)	15 (19.2)	0.001	0.995
Emergency	298 (96.4)	220 (96.1)	78 (97.5)	0.081	0.552
Critical preop. state	85 (27.5)	67 (29.3)	18 (22.5)	0.155	0.154
Acute neurological event	73 (23.7)	52 (22.7)	21 (26.6)	0.090	0.485
Cardiogenic shock	68 (22.0)	51 (22.3)	17 (21.3)	0.025	0.850
Resuscitation in operating room	7 (2.3)	5 (2.2)	17 (2.5)	0.021	0.579
Cardiac tamponade	87 (28.3)	70 (30.7)	17 (21.3)	0.217	0.106
Aortic rupture	49 (15.9)	38 (16.6)	11 (13.8)	0.079	0.549
Preop. malperfusion at CT ^a	138 (46.3)	106 (48.0)	32 (41.6)	0.129	0.332
Epi-aortic arteries dissection ^a	108 (35.0)	82 (35.8)	26 (32.5)	0.253	0.209
Mesenteric arteries dissection ^a	14 (4.8)	11 (5.2)	3 (3.8)	0.065	0.766
Renal arteries dissection ^a	36 (12.4)	28 (13.2)	8 (10.3)	0.092	0.499
Limb arteries dissection ^a	41 (14.1)	30 (14.2)	11 (14.1)	0.001	0.992
Penn classification				0.182	0.648
A	165 (53.4)	121 (52.8)	44 (55.0)		
B	74 (23.9)	55 (24.0)	19 (23.8)		
C	54 (17.5)	39 (17.0)	15 (18.8)		
B+C	16 (5.2)	14 (6.1)	2 (2.5)		
EuroSCORE II (%)	13.9±13.3	13.9±12.7	14.0±15.1	0.002	0.337

CT, computed tomography; ^a, computed tomography was not performed preoperatively in 19 patients.

Table 2. Operative data of patients who underwent surgical repair for type-A aortic dissection.

<i>Covariates</i>	<i>Overall dataset 309 patients</i>	<i>Peripheral cannulation 229 patients</i>	<i>Direct aortic cannulation 80 patients</i>	<i>Standardized difference</i>	<i>P-value</i>
Ultrasound-guided Seldinger technique	42 (13.6)	-	42 (52.5)	-	-
Peripheral arterial cannulation					
Common femoral artery	145 (46.9)	142 (62.0)	3 (3.8)	1.581	<0.0001
Axillary/subclavian artery	96 (31.1)	96 (41.9)	0	1.201	<0.0001
Aortic root replacement	94 (30.4)	79 (34.5)	15 (18.8)	0.362	0.008
Type of aortic root repair				0.380	0.110
Interposition graft procedure	210 (68.0)	147 (64.2)	63 (78.8)		
Interposition graft+AVR	5 (1.6)	3 (1.3)	2 (2.5)		
Bentall-DeBono procedure	84 (27.2)	71 (31.0)	13 (16.3)		
David procedure	9 (2.9)	7 (3.1)	2 (2.5)		
Yacoub procedure	1 (0.3)	1 (0.4)	0		
Concomitant major cardiac procedure	43 (13.9)	35 (15.3)	8 (10.0)	0.160	0.240
Concomitant CABG	40 (12.9)	33 (14.4)	7 (8.8)	0.178	0.194
Open distal anastomosis	289 (93.5)	217 (94.8)	72 (90.0)	0.180	0.113
Distal aortic anastomosis				0.302	0.306
Ascending aorta	277 (89.6)	203 (88.6)	74 (92.5)		
Between innominate a. and left common carotid a.	4 (1.3)	4 (1.7)	0		
Between left common carotid a. and left subclavian a.	6 (1.9)	6 (2.6)	0		
Descending aorta	22 (7.1)	16 (7.0)	6 (7.5)		
Hemiarch repair	264 (85.4)	192 (83.8)	72 (90.0)	0.183	0.179
Partial or total arch repair	32 (10.4)	26 (11.4)	6 (7.5)	0.132	0.330
Frozen elephant trunk procedure	4 (1.3)	3 (1.3)	1 (1.3)	0.005	0.724
Aortic cross clamp time (min)	103±39	106±41	98±33	0.218	0.183
Cardiopulmonary bypass time (min)	198±62	203±64	183±56	0.330	0.006
Hypothermic circulatory arrest	273 (88.3)	204 (89.1)	69 (86.3)	0.086	0.497
Hypothermic circulatory arrest time (min)	27±11	27±12	27±9	0.064	0.647
Antegrade cerebral perfusion	57 (18.5)	53 (23.1)	4 (5.0)	0.491	<0.0001
Bilateral antegrade cerebral perfusion	20 (35.1)	18 (34.0)	2 (50.0)	0.329	0.439
Duration of antegrade cerebral perfusion (min)	39±25	39±25	27±26	0.453	0.196
Lowest core temperature (°C)	19±2	19±3	18±1	0.501	0.018

Continuous variables are report as mean and standard deviation; categorical variables are reported as counts and percentages; CABG, coronary artery bypass grafting; AVR, aortic valve replacement.

Table 3. Outcomes of patients who underwent surgical repair for type-A aortic dissection.

<i>Outcomes</i>	<i>Overall dataset 309 patients</i>	<i>Peripheral cannulation 229 patients</i>	<i>Direct aortic cannulation 80 patients</i>	<i>P-value</i>
Hospital death	42 (13.6)	31 (13.5)	11 (13.8)	0.962
Death in operating room	6 (1.9)	5 (2.2)	1 (1.3)	1.000
Stroke/global brain ischemia	71 (23.0)	51 (22.3)	20 (25.0)	0.617
De novo stroke/global brain ischemia ^a	36 (15.3)	28 (15.8)	8 (13.8)	0.710
Multiple stroke lesions	49 (15.9)	36 (15.7)	13 (16.3)	0.911
Modified Rankin score	4.2±1.6	4.2±1.6	4.2±1.6	0.868
Hospital death/stroke/global brain ischemia	95 (30.7)	69 (30.1)	26 (32.5)	0.693
Reoperation for bleeding	40 (12.9)	32 (14.0)	8 (10.0)	0.362
Postop IABP or ECMO	4 (1.3)	3 (1.3)	1 (1.3)	1.000
DSWI or mediastinitis	14 (4.5)	11 (4.8)	3 (3.8)	1.000
Acute kidney injury	137 (47.9)	98 (46.2)	39 (52.7)	0.337
Renal replacement therapy	42 (13.6)	33 (14.5)	9 (11.3)	0.572
Intensive care unit stay (days)	6.2±6.3	6.7±6.8	4.9±4.5	0.037

Continuous variables are report as mean and standard deviation; categorical variables are reported as counts and percentages; IABP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; DSWI, deep sternal wound infection; ^a, excluding patients with preoperative acute neurological event.

Table 4. Baseline characteristics of propensity score matched patients who underwent surgical repair for type-A aortic dissection with or without direct aortic cannulation.

<i>Covariates</i>	<i>Peripheral cannulation 74 patients</i>	<i>Direct aortic cannulation 74 patients</i>	<i>Standardized differences</i>	<i>P-value</i>
Age (years)	64.4±12.4	63.6±14.4	0.059	0.723
Octogenarians	7 (9.5)	6 (8.1)	0.048	1.000
Female	33 (44.6)	34 (45.9)	0.027	1.000
Body mass index (kg/m ²)	26.6±5.1	27.0±5.2	0.082	0.598
Hemoglobin (g/L)	125±18	126±18	0.037	0.760
eGFR (mL/min/1.73 m ²)	83±36	87±40	0.097	0.663
Bicuspid aortic valve	7 (9.5)	8 (10.8)	0.045	1.000
Previous cardiac surgery	2 (2.7)	3 (4.1)	0.075	1.000
Peripheral vascular disease	6 (8.1)	5 (6.8)	0.051	1.000
Diabetes	8 (10.8)	7 (9.5)	0.045	1.000
Stroke	6 (1.4)	6 (1.4)	0.000	1.000
Pulmonary disease	1 (1.4)	3 (4.1)	0.167	0.625
Coronary artery disease	6 (1.4)	7 (9.5)	0.048	1.000
Acute myocardial infarction	6 (8.1)	5 (6.8)	0.052	1.000
LVEF ≤50%	13 (17.6)	13 (17.6)	0.000	1.000
Emergency	71 (95.9)	72 (97.3)	0.075	1.000
Critical preop. state	13 (17.6)	14 (18.9)	0.035	1.000
Acute neurological event	18 (24.3)	18 (24.3)	0.000	1.000
Cardiogenic shock	12 (16.2)	13 (17.6)	0.036	1.000
Resuscitation in operating room	1 (1.4)	1 (1.4)	0.000	1.000
Cardiac tamponade	18 (24.3)	14 (18.9)	0.132	0.556
Aortic rupture	7 (9.5)	8 (10.8)	0.045	1.000
Preop. malperfusion at CT ^a	32 (43.2)	29 (39.2)	0.082	0.728
Epaortic arteries dissection ^a	26 (35.1)	25 (33.8)	0.028	1.000
Mesenteric arteries dissection ^a	3 (4.1)	3 (4.1)	0.000	1.000
Renal arteries dissection ^a	8 (10.8)	13 (17.6)	0.195	0.359
Limb arteries dissection ^a	10 (13.5)	8 (10.8)	0.083	0.791
Penn classification			0.282	0.787
A	40 (54.1)	42 (56.8)	0.054	
B	22 (29.7)	19 (25.7)	0.089	
C	8 (10.8)	12 (16.2)	0.158	
B+C	4 (5.4)	1 (1.4)	0.222	
EuroSCORE II (%)	13.6±14.9	13.1±14.1	0.039	0.812

CT, computed tomography; ^a, computed tomography not performed preoperatively in 19 patients.

Table 5. Operative data of propensity score matched patients who underwent surgical repair for type-A aortic dissection with or without direct aortic cannulation.

<i>Covariates</i>	<i>Peripheral cannulation 74 patients</i>	<i>Direct aortic cannulation 74 patients</i>	<i>Standardized differences</i>	<i>P-value</i>
Ultrasound-guided Seldinger technique	-	41 (55.4)	-	-
Peripheral arterial cannulation				
Common femoral artery	41 (55.4)	2 (2.7)	-	
Axillary/subclavian artery	37 (50.0)	0	-	
Aortic root replacement	18 (24.3)	14 (18.9)	0.132	0.424
Type of aortic root repair			0.345	0.467
Interposition graft procedure	55 (74.3)	58 (78.4)		
Interposition graft+AVR	1 (1.4)	2 (2.7)		
Bentall-DeBono procedure	17 (23.0)	12 (16.2)		
David procedure	0	2 (2.7)		
Yacoub procedure	1 (1.4)	0		
Concomitant major cardiac procedure	7 (9.5)	7 (9.5)	0.000	1.000
Concomitant CABG	5 (6.8)	7 (9.5)	0.099	0.754
Open distal anastomosis	71 (95.9)	65 (87.8)	0.300	0.109
Distal aortic anastomosis			0.374	0.763
Ascending aorta	69 (93.2)	68 (91.9)		
Between innominate a. and left common carotid a.	1 (1.4)	0		
Between left common carotid a. and left subclavian a.	2 (2.7)	0		
Descending aorta	2 (2.7)	6 (8.1)		
Hemiarch repair	7 (9.5)	8 (10.8)	0.045	1.000
Partial or total arch repair	5 (6.8)	6 (8.1)	0.052	1.000
Frozen elephant trunk procedure	0	1 (1.4)	0.165	1.000
Aortic cross clamp time (min)	93±33	99±34	0.188	0.231
Cardiopulmonary bypass time (min)	181±51	183±57	0.038	0.837
Hypothermic circulatory arrest	64 (86.5)	64 (86.5)	0.000	1.000
Hypothermic circulatory arrest time (min)	27±12	28±9	0.046	0.833
Antegrade cerebral perfusion	17 (23.0)	4 (5.4)	0.520	0.007
Bilateral antegrade cerebral perfusion	2 (11.7)	2 (50.0)	-	-
Duration of antegrade cerebral perfusion (min)	31±18	27±26	0.156	-
Lowest core temperature (°C)	19.3±2.7	17.9±1.5	0.662	<0.0001

Continuous variables are report as mean and standard deviation; categorical variables are reported as counts and percentages; CABG, coronary artery bypass grafting; AVR, aortic valve replacement.

Table 6. Outcomes of propensity score matched patients who underwent surgical repair for type-A aortic dissection with or without direct aortic cannulation.

<i>Outcomes</i>	<i>Peripheral cannulation 74 patients</i>	<i>Direct aortic cannulation 74 patients</i>	<i>P-value</i>
Hospital death	7 (9.5)	9 (12.2)	0.804
Death in operating room	2 (2.7)	1 (1.4)	1.000
Stroke/global brain ischemia	16 (21.6)	16 (21.6)	1.000
Modified Rankin score	4.0±1.6	4.2±1.6	0.696
De novo stroke/global brain ischemia ^a	9 (16.1)	7 (12.5)	0.678
Multiple stroke	13 (17.6)	12 (16.2)	1.000
Hospital death/stroke/global brain ischemia	20 (27.0)	22 (29.7)	0.855
Reoperation for bleeding	10 (13.5)	6 (8.1)	0.424
Postop IABP or ECMO	0	0	1.000
DSWI or mediastinitis	3 (4.1)	3 (4.1)	1.000
Acute kidney injury	32 (43.2)	37 (50.0)	0.868
Renal replacement therapy	9 (13.0)	8 (11.8)	1.000
Intensive care unit stay (days)	4.8±4.0	4.9±4.5	0.943

Continuous variables are report as mean and standard deviation; categorical variables are reported as counts and percentages; AIBP, intra-aortic balloon pump; ECMO, extracorporeal membrane oxygenation; DSWI, deep sternal wound infection; ^a, excluding patients with preoperative acute neurological event.

Figure Legend

Figure 1. Early outcome in patients who underwent surgical repair of Stanford type A aortic dissection with direct aortic cannulation or peripheral arterial cannulation. DSWI, deep sternal wound infection.

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