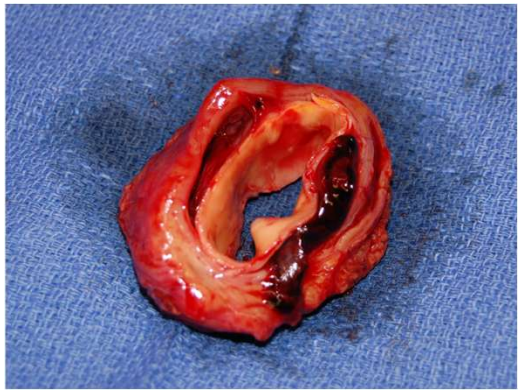
Yale NewHaven Health

Management of Type A Aortic Dissections Patients on Anti-Coagulants

The Houston Aortic Symposium

March 5-7, 2026



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Disclosure

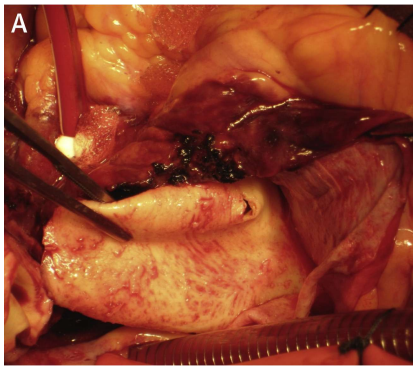
Nothing to disclose

Acute Type A Aortic Dissection (ATAAD)

Emergent proximal aortic surgery is the standard of care for ATAAD

ATAAD surgery accomplishes:

- life preservation
- correct malperfusion
- repair of valve dysfunction
- repair of aortic aneurysm



Over all 30-day mortality for ATAAD is 9.4-18%[1,2]

Re-exploration rates for bleeding , ranges between 14% and 22%.

1.STS National Database
2.Zhu Y, ed al Type A Aortic Dissection- Experience over Five Decades. JACC Vol.76 No. 14 2020

From: **The International Registry of Acute Aortic Dissection (IRAD): New Insights Into an Old Disease**

JAMA. 2000;283(7):897-903. doi:10.1001/jama.283.7.897

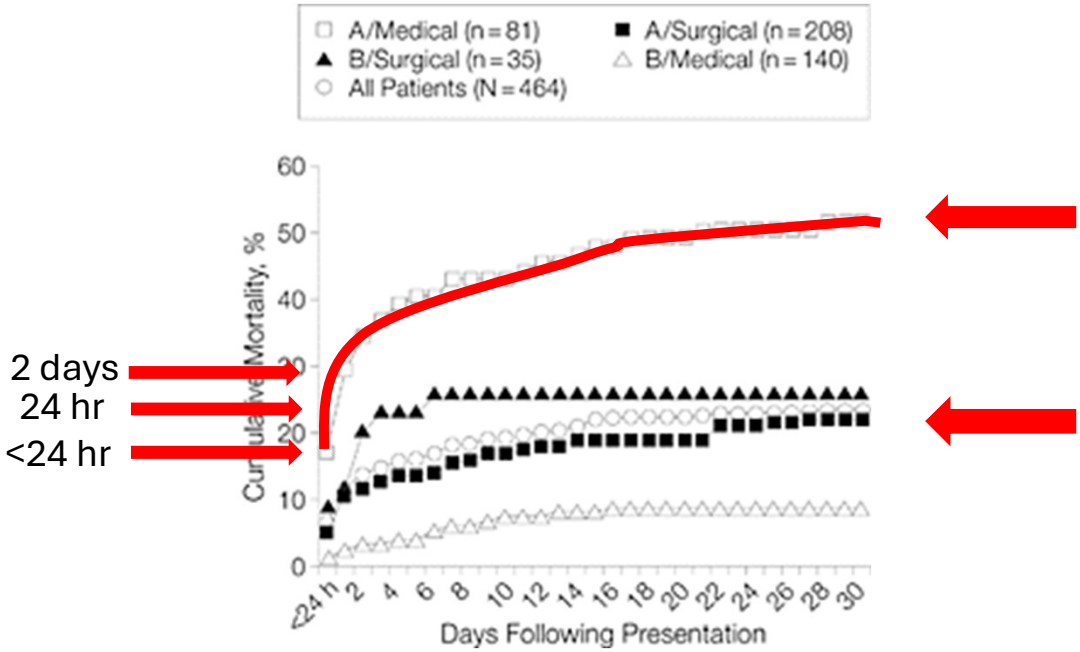


Figure Legend:

See footnote to Table 1 for descriptions of type A and type B dissections.

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Table 4. Management and Outcomes of Acute Aortic Dissection

	Type A (n = 289) Management, No. (%)		Type B (n = 175) Management, No. (%)	
	Surgical	Medical	Surgical	Medical
No.	208 (72)	81 (28)	35 (20)	140 (80)
In-hospital mortality	54 (26)	47 (58)	11 (31.4)	15 (10.7)
Total*	101 (34.9)		26 (14.9)	

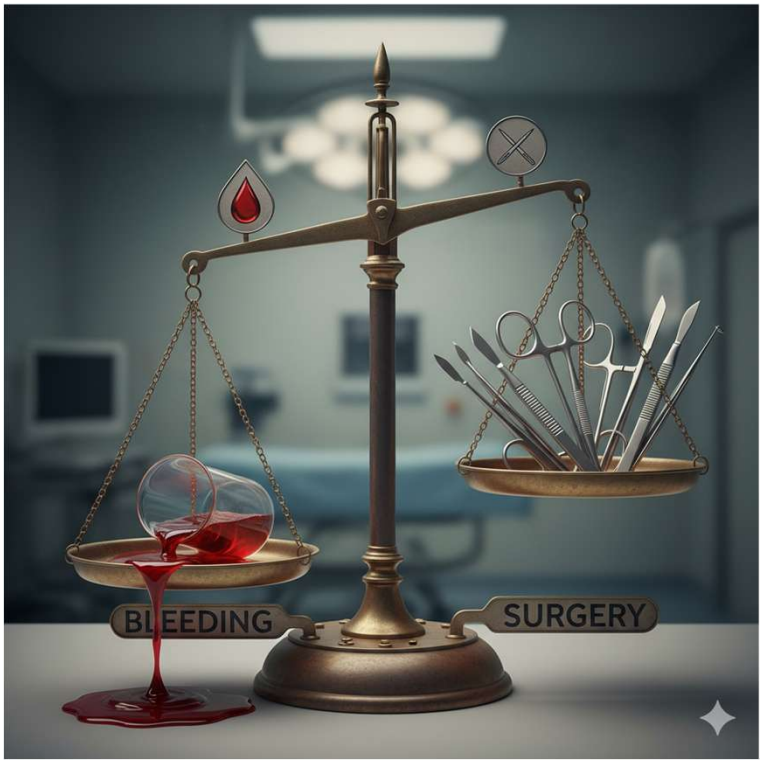
*Total mortality for both groups was 127 (27.4%). For definitions of type A and B dissections, see footnote to Table 1.

Acute Type A Aortic Dissection (ATAAD)

Unknowns of ATAAD Surgery

- **Incomplete patient medical history** (h/o CAD, stoke, HF etc)
- **Medication history**
- **Neurological status** (intubated and transfer patients)
- **Incomplete/inadequate imaging** (PE study or only Chest CT scan)
- **?ER medication administration** (r/o MI patients)
- **?pre-cath medication**
- **? Sequelae of elevated lactate**

Complex ATAAD Scenario



NOACs
clopidogrel
prasurgrel
tricagrelor

Frequency of and Inappropriate Treatment of Misdiagnosis of Acute Aortic Dissection

Mark S. Hansen, MD[a,*], Gustavo J. Nogareda, MD[b], and Stuart J. Hutchison, MD[b]

Sixty-six patients
(type A dissection: n 43; type B dissection: n 20; intramural hematoma: n 3)
Most (n 52, 79%) were transferred from other hospitals.
Correct initial diagnoses were made in 40 patients (61%).
Forty-two patients (64%) underwent surgical repair, and 24 (36%) were managed medically.
Surgery was performed in 40 of 43 patients (93%) with type A dissection and 2 of 20 patients (10%) with type B dissection.
After misdiagnosis, inappropriate antithrombotic treatment occurred in **26 patients** (100%), including antiplatelet agents in 26 (100%; acetylsalicylic acid in 26, acetylsalicylic acid and clopidogrel in 1), antithrombin agents in 21 (81%; unfractionated heparin in 13, low-molecular-weight heparin in 8), and fibrinolytic agents in 3 (12%).

Frequency of and Inappropriate Treatment of Misdiagnosis of Acute Aortic Dissection

Mark S. Hansen, MD[a,*], Gustavo J. Nogareda, MD[b], and Stuart J. Hutchison, MD[b]

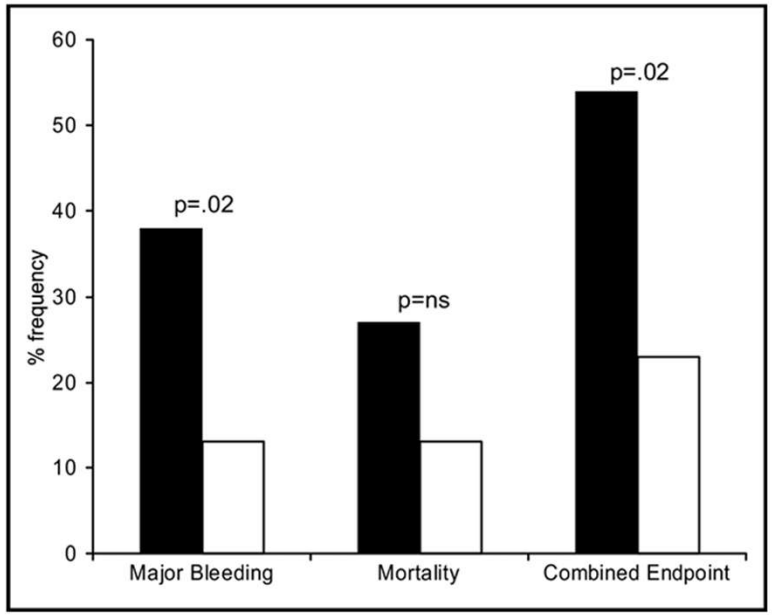


Figure 1. Rates of major bleeding and mortality in patients with AAS exposed to antithrombotic (antiplatelet, antithrombin, or fibrinolytic) agents *(black bars)* compared with those who were not exposed *(white bars)*.

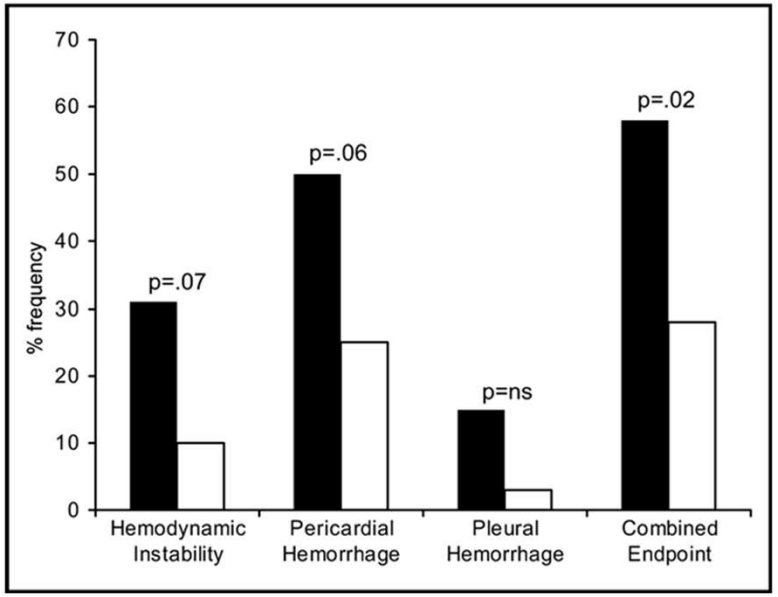


Figure 2. Rates of hemodynamic instability and hemorrhagic complications before intervention in patients with AAS exposed to antithrombotic (antiplatelet, antithrombin, or fibrinolytic) agents *(black bars)* compared with those who were not exposed *(white bars)*.

Anti-platelet Agents

Oral Antiplatelet Agents and Reversal Strategies

Drug Class	Agent(s)	Mechanism of Action	Reversal/Management Strategy
COX-1 Inhibitor	Aspirin (ASA)	Irreversibly inhibits COX-1, reducing thromboxane A2 production	**Platelet transfusion** (1-2 units). Discontinue medication.
P2Y12 Inhibitors (Thienopyridines)	Clopidogrel (Plavix), Prasugrel (Effient)	Irreversibly blocks P2Y12 receptor (ADP)	**Platelet transfusion** (often requires higher doses/multiple units). Desmopressin (DDAVP).
P2Y12 Inhibitor (Non-thienopyridine)	Ticagrelor (Brilinta)	Reversibly binds P2Y12 receptor	**Platelet transfusion** (most effective 24-48 hrs after last dose). **Bentracimab** (experimental Fab).
PDE Inhibitors	Cilostazol, Dipyridamole	Increases cAMP, inhibiting platelet aggregation	Discontinue medication.
PAR-1 Antagonist	Vorapaxar	Inhibits thrombin-induced platelet activation	Discontinue medication.

Oral Anti-coagulants

Oral Anticoagulants and Specific Reversal Agents

Oral Anticoagulant (Class)	Generic Name (Brand)	Specific Reversal Agent (Antidote)
Direct Thrombin Inhibitor	Dabigatran (Pradaxa)	Idarucizumab (Praxbind)
Factor Xa Inhibitor	Apixaban (Eliquis)	Andexanet alfa (Andexxa)
Factor Xa Inhibitor	Rivaroxaban (Xarelto)	Andexanet alfa (Andexxa)
Factor Xa Inhibitor	Edoxaban (Savaysa)	*None approved (use 4F-PCC)*
Factor Xa Inhibitor	Betrixaban (Bevyxxa)	*None approved (use 4F-PCC)*
Vitamin K Antagonist	Warfarin (Coumadin)	Vitamin K & 4F-PCC (Kcentra)

Coagulopathy-associated with ATAAD

- Blood contact between blood and subendothelial tissue factor, collagen, and adventitial layer
- Causes a consumptive coagulopathy like DIC
- Platelet consumption due to contact with collagen +CPB ➡ worsens platelet consumption and activation
- Hypothermia ➡ increased platelet activation, aggregation, reversible thrombocytopenia caused by splenic and hepatic sequestration
- Decrease factor II, V, VII, X and XII levels and higher fibrin/fibrinogen degradation products ➡ higher PT/INR
- CPB causes a dilution to all these factor, decrease in platelets and fibrinogen

Coagulopathy-associated with ATAAD

- preoperative malperfusion and extent of aortic dissection - risk factors for massive bleeding
- It has been hypothesized that increased false lumen extent, and thereby more exposed subendothelial tissue, might lead to increased coagulopathy
- This could explain why DeBakey type I dissection is associated with increased risk for bleeding

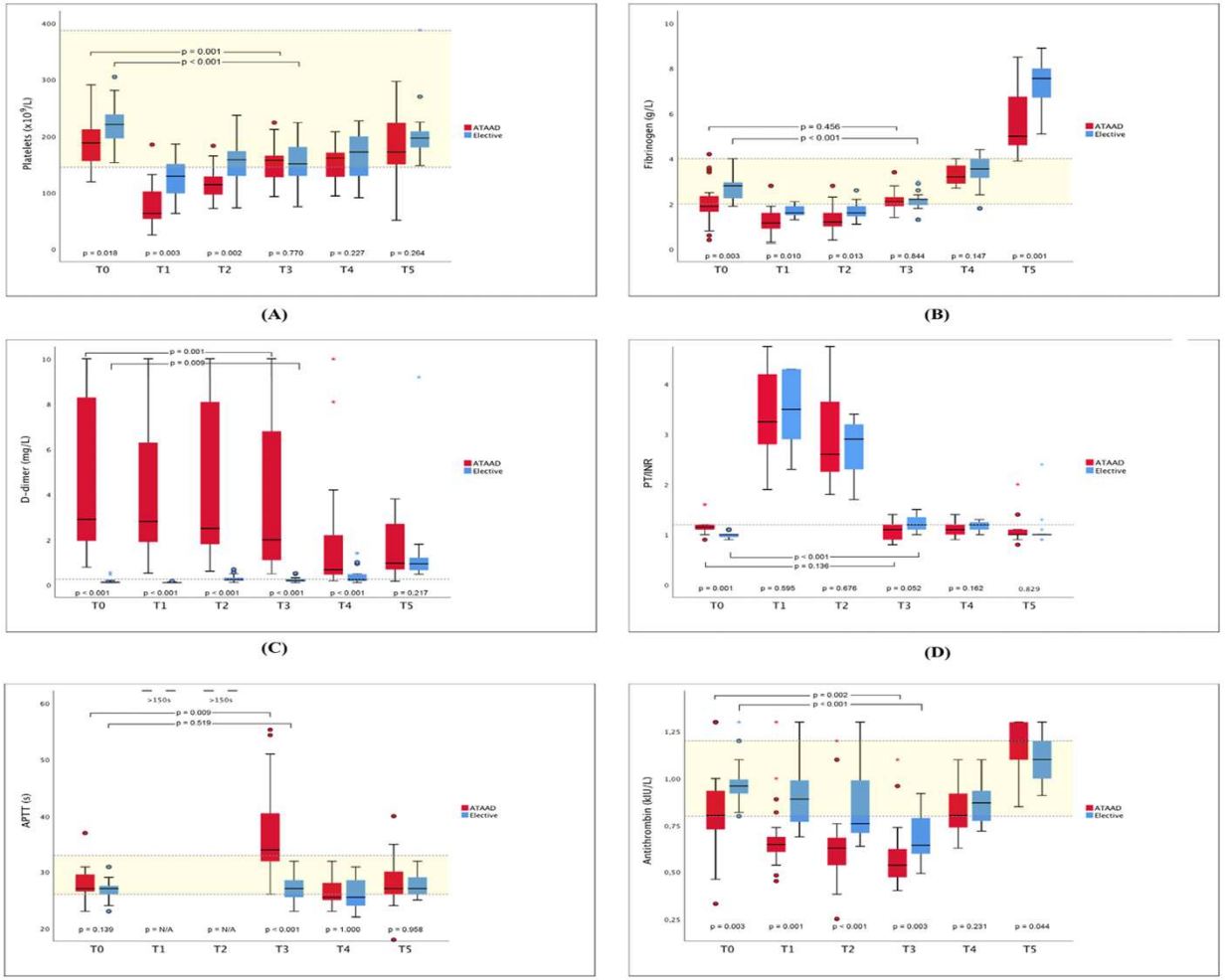


Fig 2. Box plots illustrating levels of platelets (A), fibrinogen (B), D-dimer (C), prothrombin time/international normalized ratio (D), activated partial thromboplastin time (E), and antithrombin (F). APTT, activated partial thromboplastin time; ATAAD, acute type A aortic dissection; PT/INR, prothrombin time/international normalized ratio; T0, anesthesia induction; T1, core temperature nadir; T2, before protamine reversal; T3, end of surgery; T4, 24 hours after surgery; T5, 5 days after surgery, stratified by group.

I. Zindovic et al. / Journal of Cardiothoracic and Vascular Anesthesia 33 (2019) 2746 2754

Bleeding is associated with severely impaired outcomes in surgery for acute type A aortic dissection

- UDPB massive bleeding is defined as including one or more of the following criteria: postoperative blood loss more than 2000 ml within 12 h, 11or more allogenic red blood cell (RBC) units transfused,11 or more plasma units transfused, or the use of recombinant-anti factor VIIa.

Table 1. Patient characteristics and surgical procedure.

	Overall cohort			Propensity score-matched cohort		
	Massive bleeding $n = 403$	No massive bleeding $n = 594$	Standardized difference	Massive bleeding $n = 344$	No massive bleeding $n = 344$	Standardized difference
Previous cardiac surgery	23 (5.7%)	11 (1.9%)	0.2032	6 (1.7%)	9 (2.6%)	0.0597
Acetylsalicylic acid	125 (31.1%)	147 (25.0%)	0.1360	102 (29.7%)	105 (30.5%)	0.0190
Clopidogrel or ticagrelor	63 (15.7%)	53 (9.0%)	0.2035	45 (13.1%)	46 (13.4%)	0.0086
Warfarin	38 (9.5%)	28 (4.8%)	0.1830	25 (7.3%)	27 (7.8%)	0.0220
EuroSCORE II	9.1 (3.5-12.7)	7.0 (3.1-12.0)	0.2359	8.8 (3.3, 12)	7.4 (3.5, 13)	0.0548
History of smoking	144 (46.2%)	182 (41.5%)	0.0948	125 (46.8%)	95 (40.1%)	0.1361
History of aortic aneurysm	14 (4.8%)	19 (4.5%)	0.0139	14 (5.7%)	11 (4.3%)	0.0632
Bicuspid aortic valve	21 (5.2%)	38 (6.5%)	0.0517	19 (5.5%)	16 (4.7%)	0.0385
Previous cardiac surgery	23 (5.7%)	11 (1.9%)	0.2032	6 (1.7%)	9 (2.6%)	0.0597
Acetylsalicylic acid	125 (31.1%)	147 (25.0%)	0.1360	102 (29.7%)	105 (30.5%)	0.0190
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Bleeding is associated with severely impaired outcomes in surgery for acute type A aortic dissection

Table 2. Postoperative outcomes.

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	Overall cohort			Propensity score-matched cohort		
	Massive bleeding $n = 403$	No massive bleeding $n = 594$	p-value	Massive bleeding $n = 344$	No massive bleeding $n = 344$	p-value
Perioperative stroke	97 (24.2%)	70 (11.8%)	<.001	83 (24.3%)	51 (14.8%)	.002
Postoperative coma	64 (17.5%)	34 (6.4%)	<.001	53 (17.0%)	27 (8.8%)	.002
Perioperative myocardial infarction	35 (8.8%)	26 (4.4%)	.005	33 (9.7%)	20 (5.8%)	.060
Mechanical ventilation > 48 h	218 (55.6%)	124 (21.1%)	<.001	178 (52.8%)	77 (22.6%)	<.001
Postoperative cardiac arrest	35 (8.9%)	15 (2.5%)	<.001	28 (8.3%)	11 (3.2%)	.004
New-onset dialysis	93 (23.2%)	28 (4.7%)	<.001	77 (22.5%)	17 (4.9%)	<.001
Septicemia	72 (18.0%)	35 (5.9%)	<.001	60 (17.6%)	25 (7.3%)	<.001
Deep sternal wound infection	8 (2.0%)	16 (2.7%)	.51	8 (2.4%)	12 (3.5%)	.39
Intensive care unit stay, days	6 (3-13)	3 (2-5)	<.001	6 (3, 13)	3 (2, 5)	<.001
30-day mortality	79 (19.6%)	38 (6.4%)	<.001	59 (17.2%)	26 (7.6%)	<.001
Bleeding-related outcomes						
12-hour chest tube output, ml	1150 (530–2010)	470 (340–700)	<.001	1060 (510–2020)	500 (340–730)	<.001
Resternotomy for bleeding	157 (39.0%)	68 (11.4%)	<.001	138 (40.1%)	46 (13.4%)	<.001
Postoperative cardiac tamponade	113 (28.2%)	41 (6.9%)	<.001	96 (28.1%)	24 (7.0%)	<.001
Transfusions						
Red blood cell units	13 (9–20)	4 (2–6)	<.001	13 (9–19)	4 (2–6)	<.001
Platelet units	4 (2–8)	2 (1–4)	<.001	4 (2–8)	2 (1–4)	<.001
Plasma units	12 (6–21)	3 (2–5)	<.001	12 (6–20)	3 (2–6)	<.001
Hemostatic drugs						
Recombinant factor VIIa	169 (41.9%)	0	<.001	148 (43.0%)	0 (0.0%)	<.001
Fibrinogen concentrate	236 (70.4%)	225 (51.6%)	<.001	200 (69.4%)	130 (52.2%)	<.001
Tranexamic acid	291 (72.8%)	248 (79.5%)	.038	210 (78.9%)	169 (72.5%)	.094
Aprotinin	33 (11.3%)	69 (17.6%)	.021	28 (11.1%)	39 (17.0%)	.059

Data are mean ± standard deviation, n (%), or median (interquartile range).

Massive bleeding:

Morality more than 2x

RBC/Platelet transfusions 2x

FFP 4x

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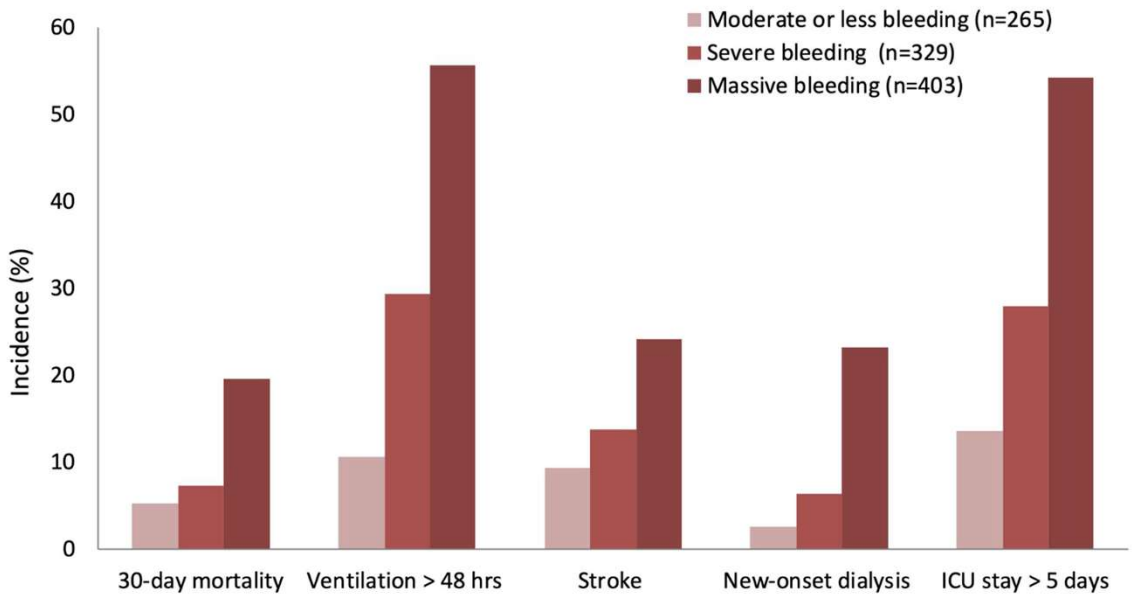

Figure 2. Incidence of 30-day mortality, mechanical ventilation more than 48 h, perioperative stroke, new-onset dialysis, and ICU stay more than 5 days in the overall cohort according to UDPB bleeding class. ICU: intensive care unit, UDPB: Universal Definition of Perioperative Bleeding.

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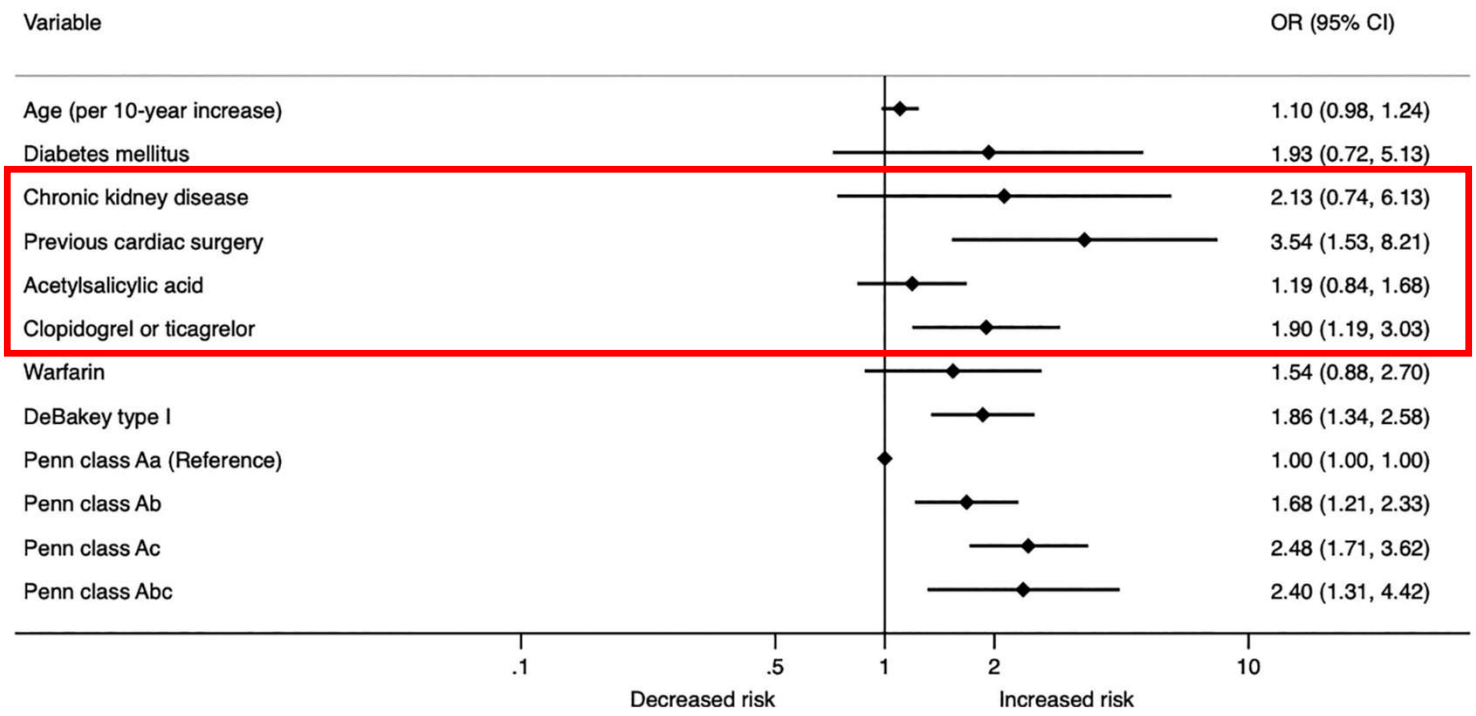

Variable
OR (95% CI)
Age (per 10-year increase)
1.10 (0.98, 1.24)
Diabetes mellitus
1.93 (0.72, 5.13)
Chronic kidney disease
2.13 (0.74, 6.13)
Previous cardiac surgery
3.54 (1.53, 8.21)
Acetylsalicylic acid
1.19 (0.84, 1.68)
Clopidogrel or ticagrelor
1.90 (1.19, 3.03)
Warfarin
1.54 (0.88, 2.70)
DeBakey type I
1.86 (1.34, 2.58)
Penn class Aa (Reference)
1.00 (1.00, 1.00)
Penn class Ab
1.68 (1.21, 2.33)
Penn class Ac
2.48 (1.71, 3.62)
Penn class Abc
2.40 (1.31, 4.42)
.1
.5
1
2
10
Decreased risk
Increased risk

Preoperative dual antiplatelet therapy increases bleeding and transfusions but not mortality in acute aortic dissection type A repair

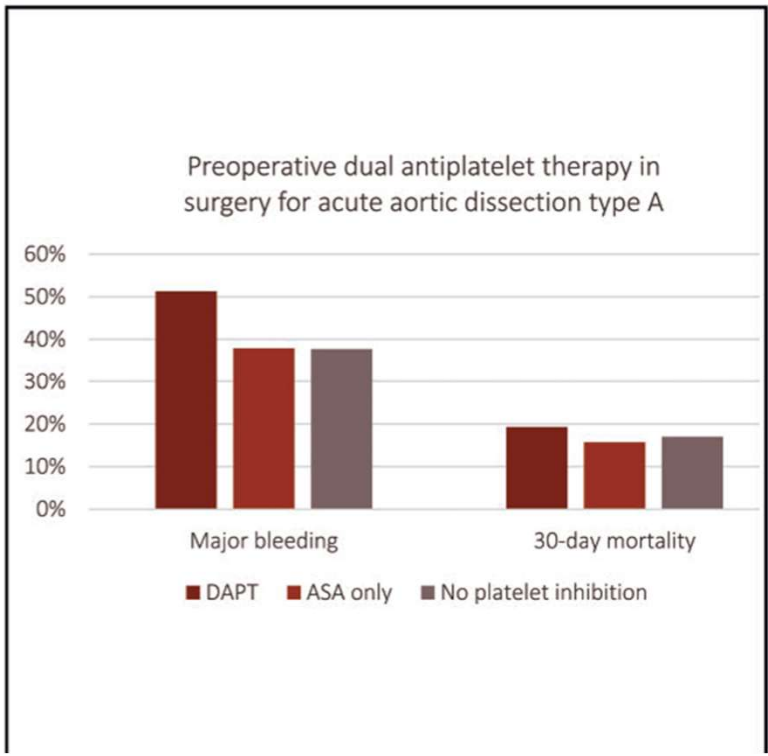
Emma C. Hansson[a,b,*], Arnar Geirsson[c,d], Vibeke Hjortdal[e], Ari Mennander[f], Christian Olsson[g], Jarmo Gunn[h], Igor Zindovic[i,j], Anders Ahlsson[k], Shahab Nozohoor[i,j], Raphaelle A. Chemtob[l], Aldina Pivodic[m], Tomas Gudbjartsson[c] and Anders Jeppsson[a,b] on behalf of the NORCAAD collaboration

Table 2: Clinical outcome variables after PS matching

	DAPT ($n = 119$)	No antiplatelets ($n = 270$)	ASA only ($n = 75$)	DAPT versus no antiplatelets *P*-value	DAPT versus ASA only *P*-value	Missing (n)
Major bleeding	61 (51.3)	106 (39.3)	26 (34.7)	0.037	0.034	0
Reoperation for bleeding	30 (26.8)	64 (23.9)	11 (14.9)	0.63	0.079	10
Postoperative bleeding (12 h)	800 (430–1410)	650 (400–1520)	840 (470–1500)	0.69	1.00	306
Transfusion RBC (units)	8 (4–15)	6 (2–12)	6 (2–11)	<0.001	0.012	61
Transfusion platelets (units)	4 (2–8)	2 (1–4)	3 (2–6)	<0.001	0.038	52
Transfusion plasma (units)	7 (3–14)	4 (2–8)	4 (2–12)	<0.001	0.067	59
Fibrinogen (g)	2 (0–4)	0 (0–2)	2 (0–3)	<0.001	0.039	161
aFVII	24 (27.6)	28 (19.7)	11 (26.8)	0.22	1.00	194
Tranexamic acid	77 (85.6)	80 (53.0)	30 (71.4)	<0.001	0.095	181
Aprotinin	19 (21.1)	30 (20.5)	8 (19.0)	1.00	0.98	186
Perioperative MI	10 (8.8)	19 (7.1)	6 (8.1)	0.69	1.00	9
Perioperative stroke	18 (15.8)	51 (19.0)	11 (14.9)	0.55	1.00	8
Deep sternal wound infection	1 (0.9)	6 (2.2)	1 (1.4)	0.67	1.00	10
Sepsis	6 (5.3)	21 (7.9)	9 (12.2)	0.51	0.16	10
Postoperative acute kidney injury	45 (39.8)	101 (41.1)	15 (20.8)	0.92	0.010	33
Postoperative renal replacement therapy	12 (10.8)	35 (13.1)	6 (8.1)	0.68	0.73	11
Postoperative tamponade	24 (21.6)	42 (15.7)	7 (9.5)	0.22	0.045	11
Length of stay in the ICU (days)	4 (2–7)	4 (2–7)	3.5 (2–6.5)	0.65	0.81	143
30-Day mortality	23 (19.3)	58 (21.5)	13 (17.3)	0.74	0.88	0

Data are presented as median (Q1–Q3) and n (%). *P*-values are from the Fisher's exact test, the χ^2 test or the Mann–Whitney *U*-test.

[a]FVII: recombinant activated factor VII; ASA: acetylsalicylic acid; DAPT: dual antiplatelet therapy; ICU: intensive care unit; MI: myocardial infarction; PS: propensity score; RBC: red blood cells.



Preoperative dual antiplatelet therapy increases bleeding and transfusions but not mortality in acute aortic dissection type A repair

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Major bleeding had more complications:

- such as perioperative stroke (22.5% vs 11.5%, P<0.001),
- postoperative acute kidney injury (51.0% vs 28.7%, P<0.001)
- sepsis (16.6% vs 6.0%, P<0.001)

30-day mortality was higher among patients with major bleeding (26.5% vs 10.9%, P<0.001).

For the entire cohort, 30-day mortality in patients with major bleeding was approximately 3 times higher (unadjusted OR 2.90, 95% CI 2.10–3.99; P<0.001, and after adjustment OR 2.44, 95% CI 1.72–3.46; P<0.001)

Prevalence, indications and appropriateness of antiplatelet therapy in patients operated for acute aortic dissection: associations with bleeding complications and mortality

Emma C Hansson,[1] Mikael Dellborg,[2,3] Vincenzo Lepore,[1] Anders Jeppsson[1,3]

43/133 patients with ATAAD received anti-platelet therapy

19 ASA

24 DAP

Intra-operative bleeding volume: 1800 ml with platelet inhibition

800 ml without platelet inhibition

Non-survivors more patients received DAT

More intra-operative and post-operative bleeding

CPB and HCA times were similar

Table 2 Survivors >30 days versus non-survivors

	Non-survivors	Survivors	p Value
N	21	110	
Gender (% women)	8 (38%)	36 (34%)	0.67
Age (years)	63±9	59±11	0.14
Ejection fraction (%)	49±13	56±9	0.024
Preoperative heart rate (BPM)	69±21	73±17	0.39
Preoperative systolic blood pressure (mm Hg)	114±30	128±33	0.039
Preoperative diastolic blood pressure (mm Hg)	62±12	72±22	0.07
Preoperative haemoglobin (g/l)	133±16	134±1 5	0.72
Platelet count ($\times 10^9$/l)	201±72	233±77	0.11
Serum creatine (μmol/l)	105±75	89±28	0.10
Cardiac tamponade	9 (43%)	22 (20%)	0.035
Intramural haematoma	4 (19%)	18 (16%)	0.76
Any antiplatelet treatment	8 (38%)	34 (31%)	0.52
Double antiplatelet therapy	7 (33%)	16 (15%)	0.038
Perioperative aprotinin treatment	2 (9.5%)	22 (20%)	0.19
CBP (min)	217±76	195±57	0.15
Circulatory arrest (min)	27±10	25±12	0.45
Intraoperative bleeding (ml)	2400 (500–5000)	1000 (500–2500)	0.020
Postoperative bleeding (ml)	2065 (580–2615)	550 (383–1198)	0.007
Postoperative bleeding >1000 ml	8 (67%)	33 (30%)	0.011
Transfusion RBC (u)	24 (14–38)	9 (3–16)	<0.001
Transfusion plasma (u)	20 (10–41)	9 (3–17)	0.006
Transfusion platelets (u)	7 (3–9)	4 (2–6)	0.07
Transfusions total (u)	48 (33–94)	21.5 (9–39)	0.002
Stroke	4 (19%)	13 (12%)	0.58
Massive bleeding	12 (57%)	21 (19%)	<0.001

Mean±SD, median (IQR) or number and %.
BPM, beats per minute; CBP, cardiopulmonary bypass; RBC, red blood cell.

Predicted 30-day mortality in univariate testing:

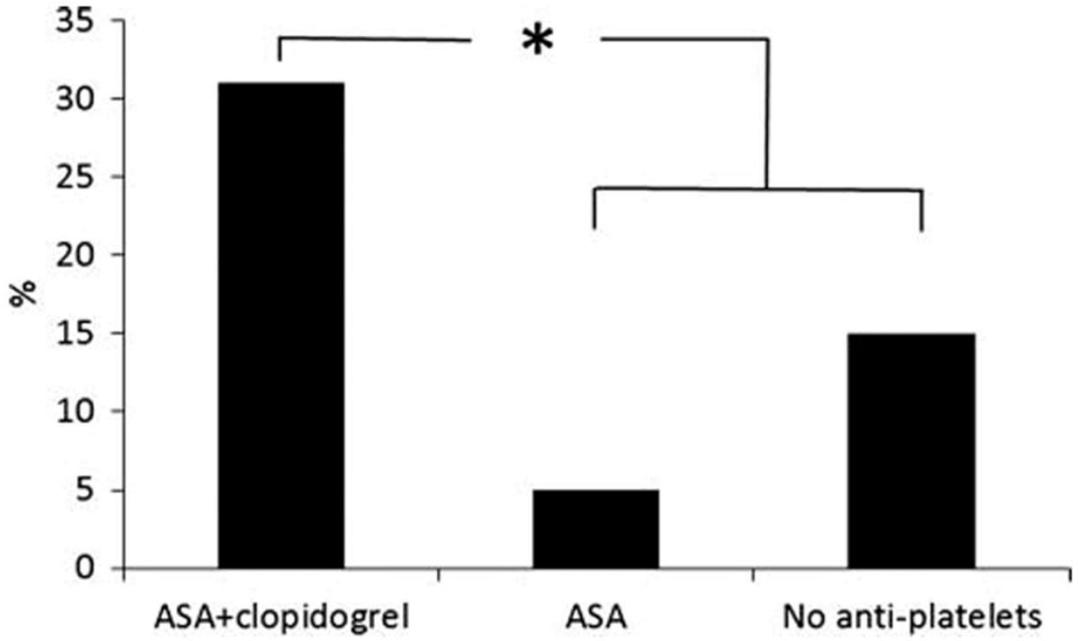
- **red blood cell transfusion (**OR 1.04 per unit, 95% CI 1.02 to 1.07, p=0.001),
- **postoperative bleeding** (OR 1.05 per 100 ml, 95% CI 1.01 to 1.09, p=0.012),
- **cardiac tamponade** (OR 3.00, 95% CI 1.12 to 8.01, p=0.028),
- **left ventricular ejection fraction** (OR 0.95, 95% CI 0.89 to 0.99, p=0.035),
- **systolic blood pressure** (OR 0.98, 95% CI 0.97 to 0.99, p=0.042)
- **DAT** (OR 2.94, 95% CI 1.03 to 8.40, p=0.044).

Prevalence, indications and appropriateness of antiplatelet therapy in patients operated for acute aortic dissection: associations with bleeding complications and mortality

Emma C Hansson,[1] Mikael Dellborg,[2,3] Vincenzo Lepore,[1] Anders Jeppsson[1,3]

Mortality during the first 30 days after surgery was not significantly different in patients who did or did not undergo antiplatelet therapy: 15% versus 19% (p=0.56).

Subgroup analysis



Adult Cardiac

Adult Cardiac Rapid Fire Orals II

Patients Undergoing Surgery for Acute Type A Aortic Dissection on Preoperative Anticoagulants and Antiplatelets

Sunday, January 26, 2025 10:05am - 10:12am PT

Location: Exhibit Hall Theater 1

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Purpose: Little is known about the surgical outcomes of patients taking direct oral anticoagulants (DOACs), warfarin, or dual anti-platelet therapy (DAPT) who present with acute Type A aortic dissection (ATAAD). We investigated this patient population using a national cohort in the Society of Thoracic Surgeons Adult Cardiac Surgery Database (STS ACSD).

Methods: A matched retrospective cohort study was conducted used data from 23,597 patients who underwent repair of ATAAD in the STS ACSD from 2017 to 2022. The primary analysis used optimal 1-to-2 matching within propensity score caliper to pair each DOAC (n=710), warfarin (n=387), and DAPT (n=610) patient to 2 control patients (n=20,230) with similar baseline characteristics. Mantel-Haenszel tests were used to assess the association between clinical outcomes and preoperative anticoagulant or antiplatelet use in each of the DOAC, DAPT, and warfarin matched comparisons. A second matched cohort analysis further compared the clinical outcomes among DOAC patients who had surgery on the day of admission and those for which surgery was delayed least one day post admission. The primary outcome was operative mortality. Secondary outcomes included reoperation for bleeding and blood product transfusion.

Results: Compared to their matched controls, the DOAC, warfarin, and DAPT groups had similar mean age (DOAC: 69.8 vs 69.2, p=0.235; warfarin: 68.1 vs 67.4, p=0.369; DAPT: 64.6 vs 64.2, p=0.462), incidence of prior cardiac surgery (DOAC: 45.6% vs 44.1%, p=0.527; warfarin: 61.2% vs 58.4%, p=0.386; DAPT: 59.0% vs 57.2%, p=0.493), and presentation in Penn class A malperfusion (DOAC: 63.2% vs 63.9%, p=0.968; warfarin: 56.6% vs 59.6%, p=0.758; DAPT: 49.2% vs 51.1%, p=0.217). Operative mortality was significantly higher for patients on DOACs (25.9% vs 21.5%, OR

1.30 [1.04,1.62], p=0.020) and DAPT (28.0% vs 23.6%, OR 1.28 [1.01,1.61], p=0.039), and trended higher for those on warfarin (25.0% vs 19.8%, OR 1.35 [0.99,1.84], p=0.053). Reoperation for bleeding was significantly higher for all 3 medication groups (DOAC: 11.7% vs 8.7%, OR 1.41 [1.03,1.93], p=0.027; warfarin 11.4% vs 7.6%, OR 1.56 [1.01,2.42], p=0.037; DAPT 14.4% vs 6.9%, OR 2.24 [1.62,3.11], p< 0.001). Blood products were more often transfused in patients on DOACs, warfarin, and DAPT (Table 1). Most DOAC patients (74.4% [520/699]) underwent surgery on day 0 of admission. There was a trend towards reduced mortality for those who underwent surgery on day ≥1 (17.6% vs 25.6%, OR 0.64 [0.4,1.01], p=0.06).

Conclusion: Patients undergoing surgery for ATAAD who were taking DOACs, warfarin, or DAPT preoperatively had increased mortality, reoperation for bleeding, and requirement for blood transfusions.

Identify the source of the funding for this research project: Penn Aorta Center, University of Pennsylvania, Philadelphia, PA

Potential Remedies

Anti-platelet Agents

Oral Antiplatelet Agents and Reversal Strategies

Drug Class	Agent(s)	Mechanism of Action	Reversal/Management Strategy
COX-1 Inhibitor	Aspirin (ASA)	Irreversibly inhibits COX-1, reducing thromboxane A2 production	**Platelet transfusion** (1-2 units). Discontinue medication.
P2Y12 Inhibitors (Thienopyridines)	Clopidogrel (Plavix), Prasugrel (Effient)	Irreversibly blocks P2Y12 receptor (ADP)	**Platelet transfusion** (often requires higher doses/multiple units). Desmopressin (DDAVP).
P2Y12 Inhibitor (Non-thienopyridine)	Ticagrelor (Brilinta)	Reversibly binds P2Y12 receptor	**Platelet transfusion** (most effective 24-48 hrs after last dose). **Bentracimab** (experimental Fab).
PDE Inhibitors	Cilostazol, Dipyridamole	Increases cAMP, inhibiting platelet aggregation	Discontinue medication.
PAR-1 Antagonist	Vorapaxar	Inhibits thrombin-induced platelet activation	Discontinue medication.

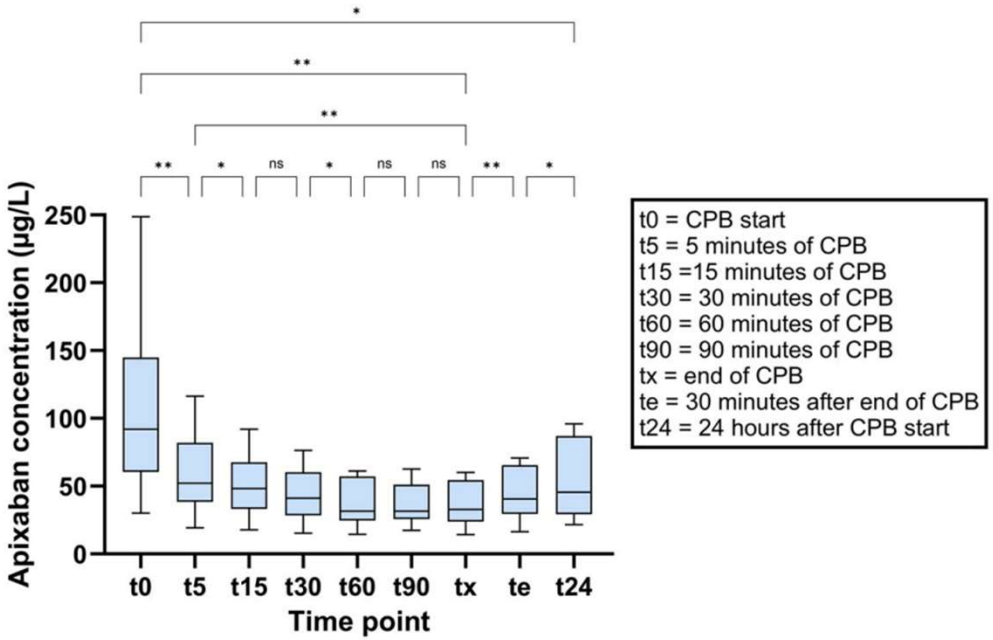
Oral Anti-coagulants

Oral Anticoagulants and Specific Reversal Agents

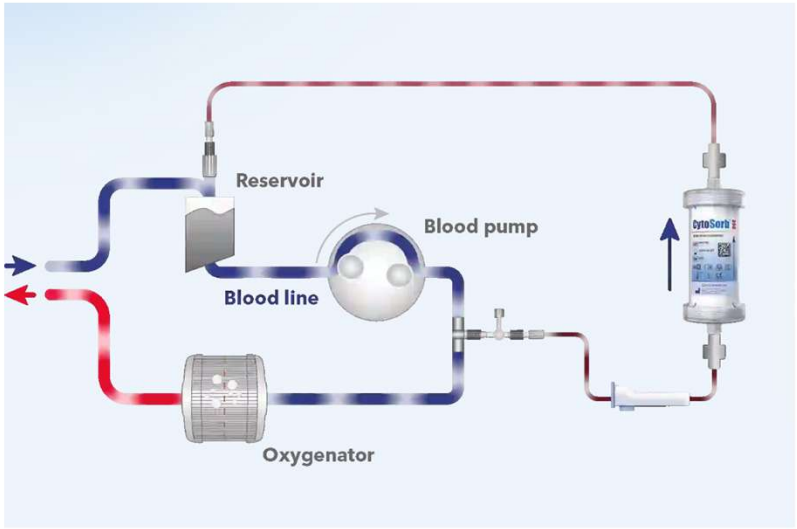
Oral Anticoagulant (Class)	Generic Name (Brand)	Specific Reversal Agent (Antidote)
Direct Thrombin Inhibitor	Dabigatran (Pradaxa)	Idarucizumab (Praxbind)
Factor Xa Inhibitor	Apixaban (Eliquis)	Andexanet alfa (Andexxa)
Factor Xa Inhibitor	Rivaroxaban (Xarelto)	Andexanet alfa (Andexxa)
Factor Xa Inhibitor	Edoxaban (Savaysa)	*None approved (use 4F-PCC)*
Factor Xa Inhibitor	Betrixaban (Bevyxxa)	*None approved (use 4F-PCC)*
Vitamin K Antagonist	Warfarin (Coumadin)	Vitamin K & 4F-PCC (Kcentra)

Apixaban removal during emergency surgery for type A acute aortic dissection: a prospective cohort study

Røed-Undlien, Henriette MD[a]; Schultz, Nina H. MD, PhD[b,c]; Husebråten, Inger M. MSc[d]; Wollmann, Birgit M. PhD[e]; Akerkar, Rupali R. PhD[f]; Molden, Espen PhD[e,g]; Amundsen, Erik K. MD, PhD[h,i]; Bjørnstad, Johannes L. MD, PhD[a,d,*]



t0 = CPB start
t5 = 5 minutes of CPB
t15 =15 minutes of CPB
t30 = 30 minutes of CPB
t60 = 60 minutes of CPB
t90 = 90 minutes of CPB
tx = end of CPB
te = 30 minutes after end of CPB
t24 = 24 hours after CPB start



Hemoadsorption of Rivaroxaban and Ticagrelor during Acute Type A Aortic Dissection Operations

Kambiz Hassan,[1] Tabea Brüning,[1] Michael Caspary,[2] Peter Wohlmuth,[3] Holger Pioch,[1] Michael Schmoeckel,[1] and Stephan Geidel[1]

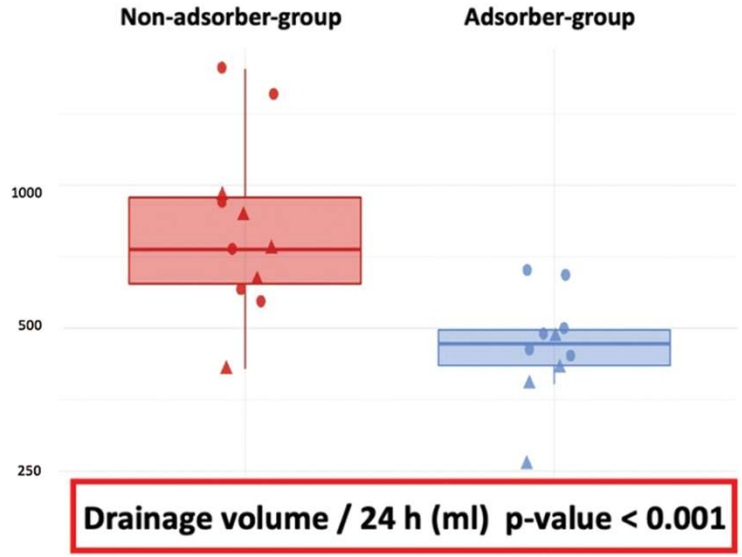
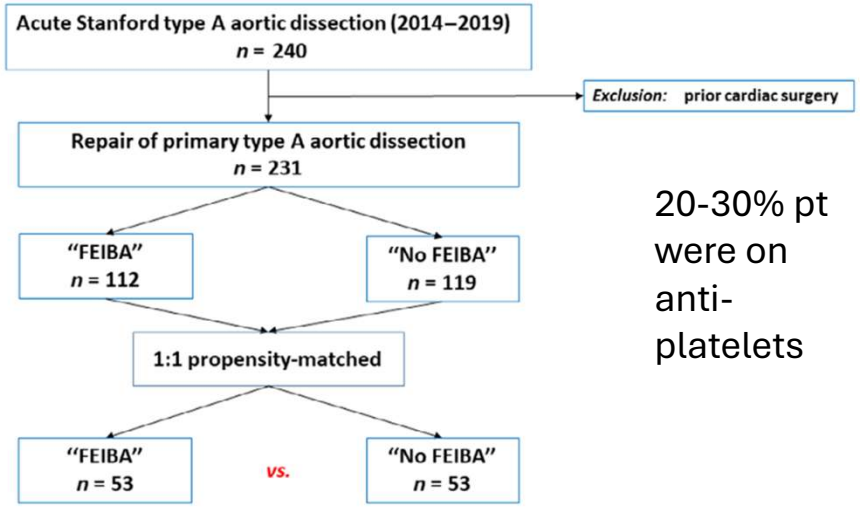


Fig. 2 The 24-hours drainage volume was significantly lower in adsorber patients; patients treated with ticagrelor ○ or rivaroxaban △ and with hemadsorption (blue) and without hemadsorption (red).

Factor eight inhibiting bypass activity for refractory bleeding in acute type A aortic dissection repair: A propensity-matched analysis

Stevan S. Pupovac[1] | Randy Levine[2] | Ashley T. Giammarino[3] | Samuel Jacob Scheinerman[3] | Alan R. Hartman[1] | Derek R. Brinster[3] | Jonathan M. Hemli[3]



20-30% pt were on anti-platelets

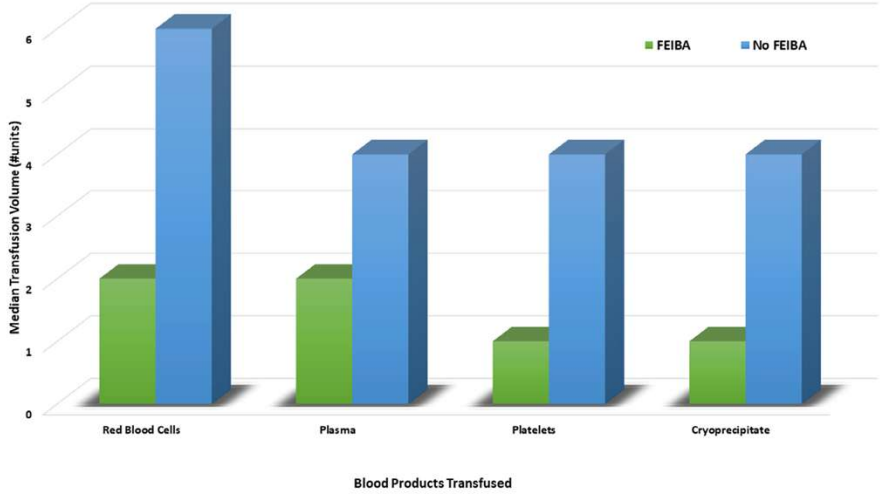


FIGURE 2 Volume of blood products transfused in the first 48 hours after acute type A dissection repair. FEIBA, factor eight inhibiting bypass activity [Color figure can be viewed at wileyonlinelibrary.com]

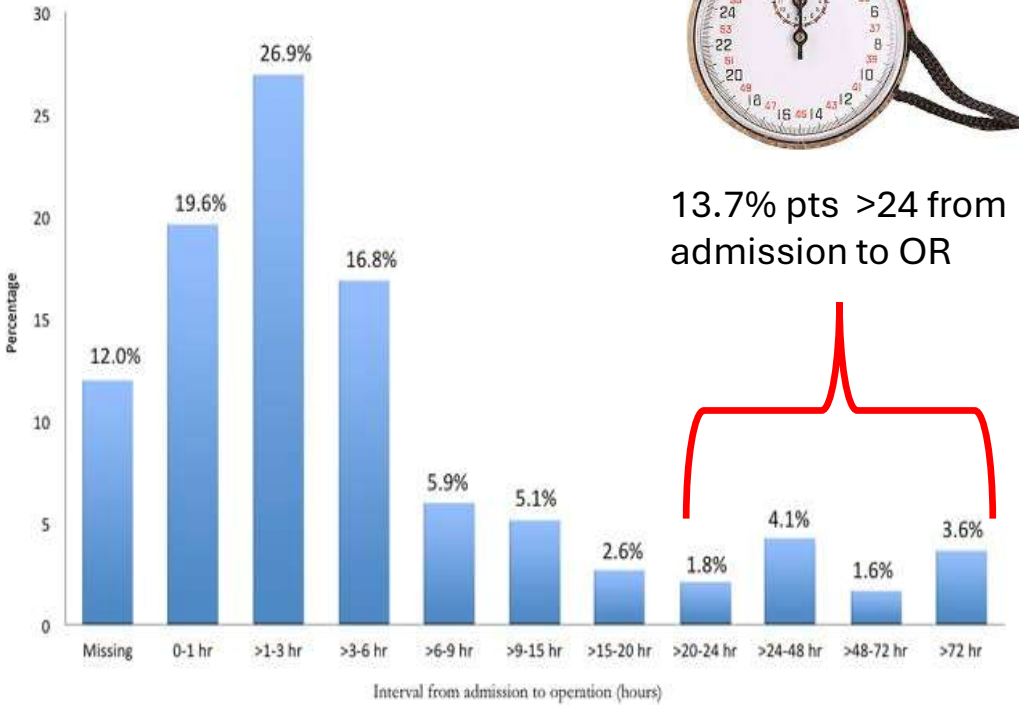
Factor eight inhibiting bypass activity for refractory bleeding in acute type A aortic dissection repair: A propensity-matched analysis

TABLE 3 Clinical outcomes

	All patients				Propensity-matched patients			
Variable	**FEIBA ($n = 112$)**	**No FEIBA ($n = 119$)**	***p* value**	**SMD**	**FEIBA ($n = 53$)**	**No FEIBA ($n = 53$)**	***p* value**	**SMD**
30-day mortality	15 (13.4)	12 (10.1)	.56	0.10	5 (13.2)	7 (9.4)	.38	0.12
Thrombotic complication	11 (9.8)	15 (12.6)	.32	0.05	5 (9.4)	7 (9.4)	.63	0.09
Stroke	10 (8.9)	9 (7.6)	.89	0.05	4 (7.5)	3 (2.8)	>.99	0.08
Pulmonary embolism	0	2 (1.7)	.26	0.08	0 (0.0)	2 (3.8)	.25	0.06
Deep vein thrombosis	1 (0.9)	6 (5.0)	.07	0.01	1 (1.9)	2 (3.8)	.50	0.04
Reoperation for bleeding	5 (4.5)	5 (4.2)	.92	0.07	2 (3.8)	1 (1.9)	.5	<0.001
Perioperative myocardial infarction	0 (0.0)	1 (0.8)	>.99	0.13	0 (0.0)	0 (0.0)	-	<0.001
Deep sternal wound infection	0 (0.0)	2 (1.7)	.5	0.19	0 (0.0)	1 (1.9)	.50	0.12
New-onset renal failure requiring dialysis	13 (11.6)	10 (8.4)	.55	0.11	4 (7.5)	4 (7.5)	>.99	<0.001
Sepsis of any cause	7 (6.3)	9 (7.6)	.89	0.05	3 (5.7)	7 (13.2)	.16	0.26
Ventilation time, hours	142.4 ± 281.7	124.0 ± 385.9	.68	0.05	146.5 ± 207.1	100.32 ± 165.5	.20	0.30
Duration of ICU care, hours	301.1 ± 527.4	193.4 ± 215.7	.04	0.27	383.4 ± 662.1	235.3 ± 233.1	.3	0.36
Length of postoperative stay, days, median (IQR)	10 (7 to 17)	10 (7 to 18)	.78	0.04	10 (7 to 17)	10 (7 to 17)	.77	0.06

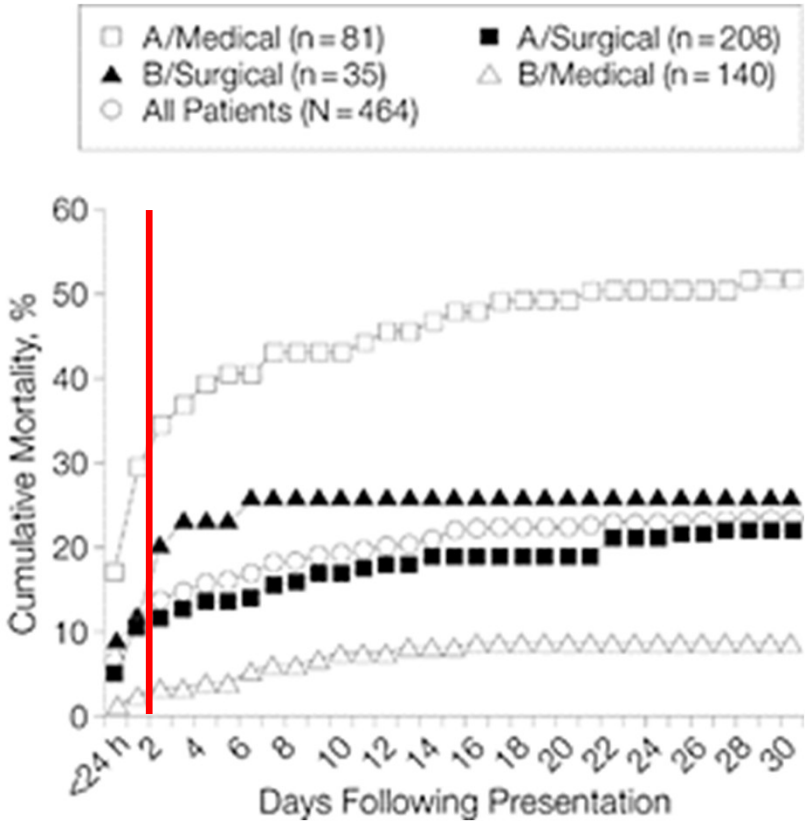
Note: Values are *n* (%) or mean ± standard deviation unless otherwise specified.
Abbreviations: FEIBA, factor eight inhibiting bypassing activity; ICU, intensive care unit; IQR, interquartile range; SMD, standardized mean difference.

Timing



13.7% pts >24 from admission to OR

vs.



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Eranki et al. Journal of Cardiothoracic Surgery (2023) 18:247
https://doi.org/10.1186/s13019-023-02341-y

Journal of Cardiothoracic Surgery

REVIEW Open Access

The role of delayed aortic surgery in type A aortic dissection and mesenteric ischemia: a systematic review and meta-analysis

Aditya Eranki[1,7*], Ashley R Wilson-Smith[2,3,4], Michael L Williams[5], Aashray Gupta[6,7], Campbell Flynn[7], Jim Iliopoulos[8] and Con Manganas[7]

Abstract

Introduction Approximately one third of patients with Acute Type A Aortic Dissection (ATAAD) present with pre-operative malperfusion syndromes (MPS). Of these, mesenteric malperfusion represents the greatest risk to patients with respect to increased short-term mortality. In select patients, it may be feasible to offer a staged approach by treating the mesenteric malperfusion first, optimizing the patient in the intensive care setting and then, following with a central aortic repair. The aim of this systematic review is to summarize cohort studies assessing the role of pre-operative interventions for mesenteric malperfusion.

Methods An electronic literature search of five databases was performed to identify all relevant studies providing studies examining short-term mortality on patients who underwent either endovascular or open revascularisation of

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Original Paper

Perfusion

Perfusion
2024, Vol. 39(6) 1098–1105
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Night-time surgery for type a aortic dissection: Immediate or delaying?

Qian Sichong,[1,2,*] Liu Hong,[3,*] Wang Shipan,[1,2] Xue Yuan,[1,2] Li Haiyang[1,2] and Zhang Hongjia[1,2]

Abstract

Objectives: This study aims to investigate whether surgery performed during night compared with daytime were associated with an increased risk of operative mortality of type A aortic dissection (TAAD) patients.

Methods: A total of 2015 TAAD patients who underwent surgical repair were collected from two cardiovascular centers from Jan 2015 to Jan 2021. According to the start time of surgery, patients were divided into daytime group (06:01 a.m. to 06:00 p.m.) and night-time group (06:01 p.m. to 06:00 a.m.), and retrospective analyses were performed between them.

Results: The operative mortality of night-time group (12.2%, 43/352) was dramatically higher than daytime group (6.9%, 115/1663; $p = 0.001$). There was significant difference between night-time and daytime groups in terms of 30-days mortality (5.8% vs 10.8%; $p = 0.001$) and in-hospital mortality (3.5% vs 6.0%; $p = 0.03$). The night-time group had a longer duration of

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Original Paper

Perfusion

When to Consider Deferral of Surgery in Acute Type A Aortic Dissection: A Review

Check for updates

Ashraf A. Sabe, MD, Edward D. Percy, MD, Tsuyoshi Kaneko, MD, Ryan P. Plichta, MD, and G. Chad Hughes, MD

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Background. Acute type A aortic dissection (ATAAD) is a surgical emergency with an operative mortality of up to 30%, a rate that has not changed meaningfully in more than 2 decades. A growing body of research has highlighted several comorbidities and presenting factors in which delay or permanent deferral of surgery may be considered; however, modern comprehensive summative reviews are lacking. The urgency and timing of this review are underscored by significant challenges in resource use posed by the coronavirus disease 2019 (COVID-19) pandemic. This review provides an update on the current understanding of risk assessment, surgical candidacy, and operative timing in patients with ATAAD.

Methods. A literature search was conducted through PubMed and Embase databases to identify relevant studies relating to risk assessment in ATAAD. Articles were selected by group consensus on the basis of quality and relevance.

Results. Several patient factors have been identified that increase risk in ATAAD repair. In particular, frailty, advanced age, previous cardiac surgery, and use of novel anticoagulant medications have been studied. The understanding of malperfusion syndromes has also expanded significantly, including recommendations for surgical delay. Finally, approaches to triage have been significantly influenced by resource limitations related to the ongoing COVID-19 pandemic. Although medical management remains a reasonable option in carefully selected patients at prohibitive risk for open surgery, endovascular therapies for treatment of ATAAD are rapidly evolving.

Conclusions. Early surgical repair remains the preferred treatment for most patients with ATAAD. However, improvements in risk stratification should guide appropriate delay or permanent deferral of surgery in select individuals.

(Ann Thorac Surg 2021;111:1754-62)

Acute type A aortic dissection (ATAAD) is a surgical emergency, with a mortality rate of up to 90% in patients who do not undergo timely operative intervention.[1–3] Despite significant advances in imaging, perioperative care, and surgical technique, operative mortality rates have remained relatively unchanged, at between 10% and 30%, over the past 2 decades (Figure 1).[4–6] Given this relative stagnation in rates of morbidity and mortality associated with ATAAD repair, more recent studies have focused on the identification of factors important for risk stratification in these patients. Furthermore, new options for endovascular management have emerged and are evolving to become efficacious treatment options for carefully selected individuals with ATAAD.[7] Finally, in the era of the coronavirus disease 2019 (COVID-19) pandemic, traditional algorithms for the treatment of surgical emergencies must be carefully challenged and reexamined with a focus on minimizing infectious spread, as well as timely and appropriate resource allocation.[8] The aims of this review were to provide a commentary on contemporary approaches to the identification of high-risk features in ATAAD and to ascertain when delay, permanent deferral of surgical treatment, or transfer to a specialized center may be considered (Table 1).

Material and Methods

Articles discussed in this narrative review were identified through a literature search of English language articles in PubMed (1946 to the present) and Embase (1974 to the present), last updated April 25, 2020. The search strategy focused on the identification of articles studying contemporary factors influencing outcomes of ATAAD repair: frailty, age, malperfusion, malperfusion syndrome (MPS), previous cardiac surgery, anticoagulant medications, and risk stratification tools. Additionally, current articles on the ongoing COVID-19 pandemic were identified through additional search of in-press articles in relevant surgical and cardiothoracic surgical journals. We selected the articles on the basis of quality and relevance.

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0003-4975/$36.00
https://doi.org/10.1016/j.athoracsur.2020.08.002

Surgical Delay for Acute Type A Dissection With Malperfusion

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Background. **An acute type A aortic dissection is considered a surgical emergency. Review of the risk factors for a type A dissection showed that preoperative malperfusion was associated with a 22% (2/9) intraoperative mortality and an 89% (8/9) hospital mortality. Intraoperative deaths were secondary to pulmonary failure resulting from capillary leak; the remaining patients died of multiorgan failure resulting from reperfusion injury.**

Methods. **The surgical delay approach was adopted for malperfused patients, and treatment in these patients included percutaneous reperfusion, with aortic fenestration and branch stenting where appropriate. Twenty patients had a type A dissection and malperfusion shown by pulsed-wave Doppler echocardiography, transesophageal echocardiography, or spiral computed tomographic scanning. Malperfusion was documented by angiography. After reperfusion, all patients' conditions were stabilized in the intensive care unit; intravenous beta-blockers were administered to decrease the maximum rate of increase of left ventricular pressure. Once patients completely recovered from the consequences of malperfusion, surgical repair was performed. Statistical comparison of the non-delay and delay groups was performed using Fisher's exact test and Student's *t* test. Multiple logistic regression analysis was used to establish independent predictors for mortality.**

Results. **The mean delay to repair was 20 days (2 to 67 days). Four (31%) patients were discharged home and readmitted for operation. Three patients (15%) died preoperatively, 1 of retrograde dissection and rupture and 2 of reperfusion injury. Seventeen underwent surgical repair, with two deaths (12%); 15 (75%) were discharged, with an average follow-up of 16.8 months ($p < 0.003$). Delay was the only independent predictor of outcome.**

Conclusions. **Patients with an acute type A dissection and malperfusion should undergo percutaneous reperfusion, and surgical repair should be delayed until the reperfusion injury resolves.**

(Ann Thorac Surg 1997;64:1669–77)

Malperfusion is a devastating complication of type A acute aortic dissection, with a reported incidence of 16% to 33% [1–3]. Review of our population of patients with a type A dissection seen from January 1992 through May 1994 showed that preoperative malperfusion was associated with the highest risk for death of all variables analyzed. Malperfusion is defined as a complication in an organ system secondary to ischemia and resulting in organ dysfunction and systemic metabolic abnormalities. Clinical presentations of malperfusion included myocardial, visceral, neurologic, and limb ischemia. Despite corrective procedures and an intact surgical repair, these patients died of metabolic abnormalities and end-organ failure.

Because of previous poor results in malperfused patients, in June 1994 we began using another approach that included surgical delay, nonoperative reperfusion where appropriate accomplished using percutaneous fenestration and stenting, and intensive medical management of the systemic blood pressure. By correcting all the metabolic abnormalities and allowing for end-organ failure to resolve before surgically repairing the aortic dissection, we hoped to improve both the short-term and long-term outcome.

In this article we describe the delayed approach to a type A dissection with malperfusion and compare the results in a retrospective manner with those in our earlier malperfused patients with an acute type A dissection who were treated with emergent surgical repair.

Material and Methods

At the University of Michigan from January 1992 through May 1994, 44 patients with an acute type A aortic dissection were treated by emergent surgical repair regardless of their perfusion status. Patients who presented with the malperfusion syndrome (end-organ damage due to ischemia secondary to dissection) underwent emergent surgical repair in the belief that it would restore blood flow to the threatened organs and help resolve end-organ damage.

Presented at the Thirty-third Annual Meeting of The Society of Thoracic Surgeons, San Diego, CA, Feb 3–5, 1997.

Address reprint requests to Dr Deeb, Section of Thoracic Surgery, The University of Michigan Hospitals, 1500 E. Medical Center Dr, 2124 Taubman Center, Box 0344, Ann Arbor, MI 48109-0344 (e-mail: mdeeb@umich.edu).

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FEATURE ARTICLES

Delayed Repair of Acute Type A Aortic Dissection in a Patient with Gastrointestinal Bleeding and Pulse Deficit

Gabriele Di Luozzo, MD, Henry J. Tannous, MD, Sharif H. Ellozy, MD, Scott Nowakowski, MD, Konstadinos A. Plestis, MD, David H. Adams, MD, and Randall B. Griepp, MD

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Acute type A aortic dissections are considered surgical emergencies because these patients are at risk for life-threatening complications. Patients who present with significant neurologic and other end-organ malperfusion may benefit from a more conservative approach. We present a patient with type A aortic dissection and concomitant mesenteric and limb ischemia.

(Ann Thorac Surg 2007;84:2097–9)

Acute type A aortic dissection (AAD) is a catastrophic event that carries a significant perioperative mortality. Reportedly, contemporary operative outcomes after the repair of AAD have a perioperative mortality of approximately 15% [1]. The majority of patients present with chest pain that radiates to the back; however, a variety of other presentations have been documented. Preoperative presentation and signs have a significant impact on operative and postoperative outcomes [2]. The most common treatment for AAD is urgent repair of the ascending aorta with reconstitution of blood flow into the true lumen. In selective cases of AAD, conservative management may be prudent when confronted with significant neurologic or other end-organ compromise.

Twenty percent of patients with AAD will present with a pulse deficit or mesenteric ischemia. Mortality of mesenteric ischemia in the setting of AAD has been reported to be approximately 30% [3]. Likewise, preoperative pulse deficit and limb ischemia have both been shown to

Accepted for publication June 27, 2007.

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0003-4975/07/$32.00
doi:10.1016/j.athoracsur.2007.06.077

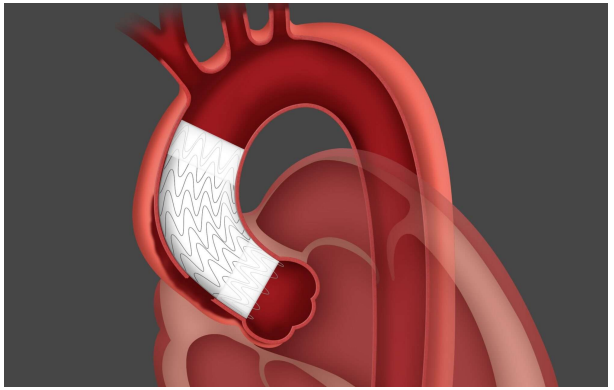
Alternatives to Surgery

Pros

- Avoid ill-effect of CPB
- No delays
- Allow for anti-coagulant washout period
- Potential bridge to more definitive surgery

Cons

- Anatomy of ascending aorta (?)
- Malperfusion
- Access vessels



Conclusions

Massive bleeding is common ATAAD surgery

Coagulopathy secondary to ATAAD + DAPT +/-NOAC

Muti-modality approach

ICU monitoring and anti-impulse therapy

Patient comorbidities and planned extent of operation should be carefully considered

Global coagulation assay (TEG)

Delay surgery until half-life clearance of the agent

Reversal agent administration

Previous surgery patients + NOAC/DAP consider delayed surgery-high risk

Post-repair

Transfusion of platelets, FFP and cryoprecipitate

FEIBA

PCC

Thank you

Cause of Death Following Surgery for Acute Type A Dissection

Evidence from the Canadian Thoracic Aortic Collaborative

R. Scott McClure, MD, SM[1,2], Maral Ouzounian, MD, PhD[3], Munir Boodhwani, MD[4], Ismail El-Hamamsy, MD, PhD[5], Michael W.A. Chu, MD[6], Zlatko Pozeg, MD[7], Francois Dagenais, MD[8], Khokan C. Sikdar, MSc, MAS, PhD[9], Jehangir J. Appoo, MDCM[1]*

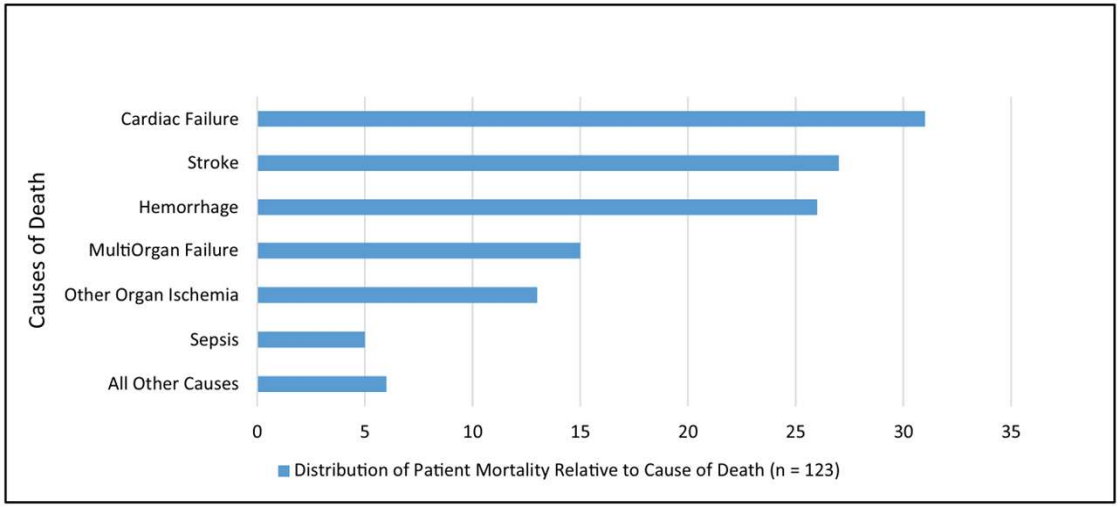


Figure 1. Distribution of patient mortality depending on cause of death during surgical repair for acute Type A dissection at nine surgical centers across Canada between 2007 and 2013.

Table 5. Postoperative complications prior to death (n = 123).

Complication	n (%)
Cardiac complications	58 (47)
Respiratory failure	46 (37)
Stroke	37 (30)
Renal failure (requiring dialysis)	2 (26)
Gastrointestinal complications	30 (24)
Re-exploration for bleeding	25 (20)
Multiorgan failure	25 (20)
Sepsis	13 (11)
Paraplegia	9 (7)