Type A Aortic Dissection After Nonaortic Cardiac Surgery

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Background—Cardiac surgery with cardiopulmonary bypass is associated with mechanical manipulation of the ascending aorta that occasionally leads to type A aortic dissection (AAD).

Methods and Results—One hundred three patients with surgical repair for AAD following nonaortic cardiac surgery were identified. With the use of logistic regression modeling, coronary artery bypass surgery (CABG), either isolated or combined with another procedure in the initial operation, was associated with significantly higher operative mortality in comparison with patients with non-CABG procedures at the time of AAD repair both for all patients (odds ratio, 2.90; 95% confidence interval, 1.09–7.72; P=0.033) and for patients with acute and chronic AAD ≥30 days after the initial operation (odds ratio, 3.62; 95% confidence interval, 1.13–11.54; P=0.03). In patients who developed AAD late after the initial operation, operative mortality was highest in patients without preoperative coronary angiography and appropriate management of their native coronary artery disease and graft disease (odds ratio, 5.36; 95% confidence interval, 1.68–17.0; P=0.002). Nearly all the intimal dissection tears were located at sites of previous surgical trauma. Most of the ascending aortas that had dissected initially had a diameter ≥40 mm with histological evidence of medial degeneration in resected tissue samples.

Conclusions—In patients who have undergone previous cardiac surgery, preexisting aortic wall pathology contributes to AAD with typical intimal damage at sites of mechanical trauma. The operative mortality was the highest in patients with previous CABG in comparison with patients with non-CABG procedures. Preoperative coronary angiography and operative management of native coronary and graft disease were significantly associated with outcome in patients with previous CABG. (Circulation. 2013;128:1602-1611.)

Key words: aorta ■ cardiopulmonary bypass ■ hypertension ■ pathology ■ surgery
with a history of cardiac surgery and cardiopulmonary bypass (CPB), and, specifically, AAD may occur in 0.6% of patients who have had previous aortic valve replacement (AVR) surgery. Furthermore, =1 in every 7 to 8 patients with acute AAD had previously undergone cardiac surgery. It is therefore reasonable to believe that cardiac surgery itself contributes to a low but significant percentage of all aortic dissections. Patients who experience AAD during or after cardiac surgery represent a unique population with important differences in aortic pathology, cause of dissection, and clinical presentation. Although predisposing factors for AAD remain ill-defined, previous cardiac surgery is increasingly recognized as a risk factor for AAD. However, current guidelines do not provide conclusive information on the characteristic features and management requirements in this group of patients. Operative mortality rates for these patients have been reported to be between 6% and 66%, suggesting substantial differences in patient management and thus great potential for improvement. Several potential sites of iatrogenic trauma during primary cardiac surgery have been suggested as the origin for later aortic dissection, including sites of cross-clamping injury, suture line, and cannulation sites, especially in the presence of concomitant vessel wall pathologies. Most reports provide no comment on either the aortic morphology at the time of the primary surgery, or any information on the associated histopathology or the precise entry tear location. Interpretation of reported data is further limited by unclear definitions, varying inclusion and exclusion criteria, and differing patient management. For example, previous case series have focused exclusively on AVR patients, or have included cases with congenital repair, and previous aortic surgery for dissection and aneurysms. Other reports have not clearly distinguished acute from chronic cases and intraoperative from postoperative AAD.

The underlying pathology and its progression, the symptoms and clinical presentation, and the diagnostic and treatment strategies are all crucial elements in the management and outcome of AAD after cardiac surgery. Realizing the associated high mortality in this patient group, identification of predictive conditions allowing for improved management and outcome are of the upmost importance. The purpose of this study was therefore to determine conditions associated with the surgical mortality of AAD repair as a complication of non-aortic cardiac surgery and to identify management pathways to improve outcome.

Methods

Study Population

We retrospectively reviewed the data of all adult cardiac surgical patients who underwent cardiac surgery with CPB between January 1, 1990 and December 31, 2012 (n=68249) at 9 university institutions in Austria, Germany, Switzerland, and the United Kingdom. A total of 103 patients (0.15%) with AAD and a history of previous cardiac surgery were identified (Vienna [AKH and Hietzing] [n=12], Graz [n=19], Salzburg [n=11], Innsbruck [n=14], Berne [n=19], Freiburg [n=6], London [Brompton: n=17], and Liverpool [n=5]) and made up our study group. Patients with previous aortic surgery for aneurysm or dissection (including Marfan syndrome and other connective tissue disorders), dissection following cardiac catheter intervention and transcatheter aortic valve implantation, traumatic rupture, and coarctation of the aorta were excluded.

Definitions

AAD was defined as intraoperative when the dissection occurred and was recognized during the primary cardiac surgery (before leaving the operating room) and was treated at the time of recognition. Acute AAD within 2 to 4 weeks after cardiac surgery is thought to have a particularly high risk of rupture and operative mortality that can be expected to change later when adhesions have formed and inflammation has resolved. The early postoperative period was therefore defined as AAD within 30 days of primary surgery. Late acute dissection was defined as occurring >30 days after primary surgery, with surgical repair within 2 weeks after the onset of specific symptoms leading to the diagnosis of aortic dissection. Late chronic AAD cases were most often incidental findings with no acute or specific symptoms. These AADs had presumably occurred >30 days after the initial cardiac surgery, and the patients underwent elective surgery.

The major and most specific presenting symptom that patients with acute AAD experience is severe or worst ever chest pain having an abrupt onset and a sharp, tearing, or ripping quality; this was therefore defined as the typical presentation of AAD. All other symptoms considered nonspecific for AAD or difficult to allocate (eg, focal neurological deficits, shortness of breath, congestive heart failure, supraclavicular swelling, tachycardia, or palpations) were defined as atypical presentation. Impending tamponade was defined as clinical or echocardiographic evidence of a pericardial effusion (and right atrial or ventricular compression) with palpable pulses and no systemic hemodynamic compromise. Severe tamponade was defined as clinical and imaging documentation of pericardial effusion with pulse deficit and hemodynamic compromise.

Data Collection

The local ethics committees approved review of the data, and consent was waived for the retrospective analysis. The hospital data and records available for post hoc analyses included medical notes for all hospital stays, perfusionist and anesthetist reports, the operating notes for the secondary procedure for AAD (all 100% complete), and 76 of the primary operating notes, of which 45 included the operating surgeon’s description of the aorta. Because routine non-aortic procedures do not usually require preoperative imaging of the aorta, computed tomography (CT) scans and aortic measurements were limited to 36 preoperative investigations. The entry sites of dissection were identified intraoperatively with certainty in 94 cases, documented by the surgeon, and confirmed with imaging reports (CT/MRI or transesophageal echocardiography imaging). Furthermore, 54 histopathologic reports of resected aortic tissues were available for review. Patients were seen at outpatient clinics regularly, and follow-up was 100% complete (the closing date for telephone interviews was December 15, 2012) with a mean follow-up of 3.5±4.5 years (maximum, 22.8 years) and a total of 362 patient-years.

Surgical Technique

Primary surgical procedures are shown in Table 1. All operations were performed with standard techniques through a median sternotomy and with CPB. Techniques for surgical repair of AAD are displayed in Table 2. Operations were performed without delay as emergencies in 80 patients, whereas 23 cases of chronic AAD were classified as elective procedures. Ten patients received one or more new coronary bypass grafts, and 12 had their proximal anastomoses reimplanted into the prosthetic aortic graft with a button of native aorta. Intraoperative cases were initially cannulated according to their planned procedure, and the arterial cannula was changed to the femoral artery as soon as the AAD was noticed. For all other surgical procedures (n=77), femoral arterial cannulation was used in 42 patients (55%), and the remaining patients had arterial cannulation via the right subclavian or axillary artery (31%) or through direct cannulation into the aortic arch (14%).
Venous drainage was established via the femoral vein (49%), via the right atrium (45%), or by bicaval (5%) cannulation. Deep hypothermic circulatory arrest (DHCA, 18–22°C) was used in 79 patients (78%). The operations were performed by 48 experienced staff surgeons.

### Statistical Analysis
Continuous variables are expressed as mean and standard deviation or as medians with first and third quartiles (Q1-Q3) for nonnormally distributed variables. Categorical variables are expressed as raw numbers and percentages. For the univariate statistical comparison, we used the χ² or Fisher exact test for variables with an expected cell count of <5 patients for categorical variables, and the Mann-Whitney U test or Kruskal-Wallis test in case of >2 groups for continuous variables. Kaplan-Meier estimates were used to calculate cumulative probabilities of overall survival rates. We used logistic regression and Cox proportional hazards regression for the analyses of 30-day (operative) and long-term mortality, respectively. Multivariable logistic and Cox models were adjusted for 2 different sets of variables: first, for cross-clamp time, CBP time, and interval groups; and, second, for preoperative coronary angiography and new coronary bypass grafting. Overall model fit was assessed by using the Hosmer-Lemeshow test. A P value of <0.05 was considered significant; all P values and confidence intervals (CIs) were 2 sided. Outcome data are presented as operative mortality, defined as death from any cause during or after surgery for AAD within 30 days if the patient is discharged or within any interval if the patient is not discharged. All analyses were performed by using SPSS 17.0.

### Results
Demographics, Initial Description of the Aorta, and Interval to Dissection
To avoid bias, we first checked whether a conjoint analysis of the data was justified, because we included records from
Because no differences were found for age, sex, interval groups, body mass index, CPB time, cross-clamp time, and operative mortality (all $P > 0.05$), the data were pooled. The study group consisted of 68 men (66%) and 35 women (34%) with a mean age of 66.8±11.6 years at the time of AAD repair (Table 1). Patients with late AAD (acute and chronic) were significantly younger at the time of AAD repair than intraoperative and early postoperative cases (64.2±12.3 versus 71.6±8.3 years, $P = 0.002$). The vast majority (83%) of patients were hypertensive. Nonsignificant patient characteristics between interval groups included left ventricular ejection fraction, body mass index, sex, age, and hypertension (all $P > 0.05$, Table 1). Nearly all (92%) of the aortas that later dissected were reported enlarged at the primary operation with a diameter of $\geq 40$ mm (mean, 46±3 mm; range, 40–57 mm). With the exception of a single case, all surgeons noted thinned, fragile, slightly dilated, or soft qualities of the aorta. Nearly all (92%) of these descriptions were confirmed histologically in corresponding pathological findings.

The intervals between primary cardiac surgery and AAD for the groups and according to type of surgery are shown in Table 1 and Figure 1. Intervals were significantly determined by the type of the primary procedure ($P = 0.042$). After AVR, 91% of the cases dissected late, whereas 56% of the cases of isolated coronary artery bypass surgery (CABG) dissected intraoperatively or early postoperatively. Restricting analyses to groups without mitral valve replacement, intervals for isolated AVR, AVR+CABG, and isolated CABG were 7.1±6.5 years, 3.6±4.0 years, and 1.4±2.6 years, respectively ($P \leq 0.001$). Excluding the intraoperative and early postoperative cases, the corresponding intervals were 8.2±6.3 years, 3.4±2.9 years, and 3.1±3.3 years, respectively ($P = 0.003$).

### Table 2. Operative Data in Patients With Type A Aortic Dissection and History of Nonaortic Cardiac Surgery

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Intraoperative</th>
<th>Early Postoperative</th>
<th>Late Postoperative (Acute)</th>
<th>Late Postoperative (Chronic)</th>
<th>$P$ Value</th>
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<tbody>
<tr>
<td>n</td>
<td>103</td>
<td>24</td>
<td>12</td>
<td>44</td>
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</tr>
<tr>
<td>Aortic replacement</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ascending, n (%)</td>
<td>43 (42)</td>
<td>13 (54)</td>
<td>5 (50)</td>
<td>15 (34)</td>
<td>10 (43)</td>
<td></td>
</tr>
<tr>
<td>Ascending-hemiarch, n (%)</td>
<td>25 (24)</td>
<td>8 (33)</td>
<td>5 (50)</td>
<td>10 (23)</td>
<td>2 (9)</td>
<td></td>
</tr>
<tr>
<td>AVR+ascending, n (%)</td>
<td>7 (7)</td>
<td>3 (13)</td>
<td></td>
<td>3 (7)</td>
<td>1 (4)</td>
<td></td>
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<tr>
<td>Bentall-hemis/total/arc, n (%)</td>
<td>7 (7)</td>
<td>—</td>
<td>—</td>
<td>5 (11)</td>
<td>2 (9)</td>
<td></td>
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<tr>
<td>Local repair, n (%)</td>
<td>1 (1)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1 (4)</td>
<td></td>
</tr>
<tr>
<td>Bentall, n (%)</td>
<td>18 (17)</td>
<td>—</td>
<td>—</td>
<td>11 (25)</td>
<td>7 (30)</td>
<td></td>
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<tr>
<td>Surgery abandoned, n (%)</td>
<td>2 (2)</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Previous CABG, n</td>
<td>55</td>
<td>18</td>
<td>10</td>
<td>16</td>
<td>11</td>
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<tr>
<td>Coronary angiography, n</td>
<td>12</td>
<td>—</td>
<td>—</td>
<td>8</td>
<td>4</td>
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<td>New bypass grafting, n</td>
<td>10</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td>1</td>
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</tr>
<tr>
<td>Cross-clamp, min (±SD)</td>
<td>129±55</td>
<td>136±41</td>
<td>86±62</td>
<td>119±55</td>
<td>118±60</td>
<td>0.058</td>
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<tr>
<td>DHCA, n (%)</td>
<td>79/101 (78)</td>
<td>20 (83)</td>
<td>8 (80)</td>
<td>36 (82)</td>
<td>15 (65)</td>
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<tr>
<td>DHCA, mean (±SD), min</td>
<td>34±18</td>
<td>28±11</td>
<td>34±18</td>
<td>36±21</td>
<td>37±17</td>
<td>0.669</td>
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**Entry of dissection**

<table>
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<tr>
<th></th>
<th>Overall</th>
<th>Intraoperative</th>
<th>Early Postoperative</th>
<th>Late Postoperative (Acute)</th>
<th>Late Postoperative (Chronic)</th>
<th>$P$ Value</th>
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<tbody>
<tr>
<td>Proximal anastomosis, n</td>
<td>32</td>
<td>5</td>
<td>8</td>
<td>10</td>
<td>9</td>
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<tr>
<td>Aortotomy, n</td>
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<td>2</td>
<td>24</td>
<td>11</td>
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<tr>
<td>Cardioplegia, n</td>
<td>5</td>
<td>4</td>
<td>1</td>
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<td>—</td>
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<tr>
<td>Cannulation, n</td>
<td>10</td>
<td>7</td>
<td>—</td>
<td>1</td>
<td>2</td>
<td></td>
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<tr>
<td>Cross-clamp, n</td>
<td>10</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>9</td>
<td>1</td>
<td>—</td>
<td>7</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Operative mortality, n (%)</td>
<td>28 (27)</td>
<td>4 (17)</td>
<td>5 (42)</td>
<td>14 (32)</td>
<td>5 (22)</td>
<td>0.329</td>
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<tr>
<td>Previous CABG, n</td>
<td>20</td>
<td>4</td>
<td>4</td>
<td>7</td>
<td>5</td>
<td>0.488</td>
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<tr>
<td>Previous non-CABG, n</td>
<td>8</td>
<td>—</td>
<td>1</td>
<td>7</td>
<td>—</td>
<td>0.072</td>
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</table>

**Cause of death**

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Intraoperative</th>
<th>Early Postoperative</th>
<th>Late Postoperative (Acute)</th>
<th>Late Postoperative (Chronic)</th>
<th>$P$ Value</th>
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<tbody>
<tr>
<td>MCI/LCO, n</td>
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<td>4</td>
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<td>12</td>
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<tr>
<td>Bleeding, n</td>
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<td>—</td>
<td>2</td>
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<td>1</td>
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<td>Neurological, n</td>
<td>4</td>
<td>—</td>
<td>2</td>
<td>2</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>MOF, n</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>—</td>
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</tbody>
</table>

AVR indicates aortic valve replacement; Bentall, conduit; CPB, cardiopulmonary bypass; DHCA, deep hypothermic cardiac arrest; LCO, low cardiac output; MCI, myocardial infarction; MOF, multiorgan failure; Q1-Q3, first and third quartiles; and SD, standard deviation.
Clinical Presentation
Most cases of intraoperative AAD were diagnosed visually after dilatation and bluish discoloration of the aorta (n=14), or immediately after aortotomy (n=2) and puncture of graft holes (n=1). AAD was suspected when arterial line pressures suddenly increased or when unexplained no-flow on bypass grafts was noticed (n=3). Transesophageal echocardiography was used in all cases to diagnose or confirm the clinical suspicion of AAD.

Patients with early postoperative AAD presented with sudden onset of severe chest pain, central neurological deficit, or signs of impending pericardial tamponade. Four patients were still sedated and intubated, and AAD was noticed only by a sudden blood loss through chest drains. Two of these patients were taken to the operating room before imaging could be performed, but, owing to the dismal prognosis, the procedure was abandoned in due course and the diagnosis was made at the postmortem. Of 67 patients with late AAD, typical presentation with a sudden onset of sharp pain in the chest was noted in 50% of acute cases. There was only a single case of late acute AAD with rupture and severe tamponade. All other patients were hemodynamically stable on arrival in the operating room and without evidence of impending or severe tamponade.

Only 3 of 23 (13%) patients with late chronic aortic dissection reported previous episodes with chest pain. All other patients presented with unspecific symptoms or had never experienced any pain or symptoms when AAD was detected, usually on routine follow-up visits.

Histology and Entry of Dissection
The entry sites of dissection in the ascending aorta are shown according to the intervals between primary surgery and AAD (Table 2) and according to the type of the previous procedure (Table 3). The proximal graft anastomosis and aortotomy sites were identified as entries in approximately two-thirds of cases with previous isolated AVR (67.5%) and CABG (65.8%). Other dissection entry locations were sites of cannulation (n=8), cross-clamping and side-biting clamping (n=7), and cardioplegia (n=5). The entry was either not identified or not documented in 9 cases. With the exception of 4 cases, all other histology reports documented pathologies of the aortic wall: medial degeneration (MD, n=24), (athero)sclerosis (n=18), a combination thereof (n=6), and giant-cell arteritis (n=2).

Surgical Management and Outcome
Overall operative mortality after surgery for AAD was 27%. Of 28 mortalities, 2 patients died during the initial hospitalization at day 31 and 45 after surgery; all others died <30 days postoperatively (14 patients on the day of surgery, another 10 patients at ≤6 days). The mortality rates for intraoperative, early postoperative, late acute, and chronic AAD were 17%, 42%, 32%, and 22%, respectively (P=0.329 between groups, Table 2). The cause of death in these 28 patients was myocardial infarction/low cardiac output (n=20), bleeding (n=3), neurological (n=4), and multi-organ failure (n=1). Patients with primary CABG (isolated or concomitant) had a significantly higher risk of death than patients with previous non-CABG surgery as observed in the entire population (odds ratio [OR], 2.90; 95% CI, 1.09–7.72; P=0.033) and particularly in patients with acute and chronic AAD ≥30 days after the initial operation (OR, 3.62; 95% CI, 1.13–11.54; P=0.03). These odds ratios correspond to the crude risk ratios for the whole group [relative risk, 2.86; 95% CI, 1.12–7.30; P=0.028] and for the group with late AAD [relative risk, 3.77; 95% CI, 1.24–11.49; P=0.026], respectively. In contrast to patients with previous AVR,
operative mortality in patients with previous CABG was significantly determined by preoperative angiography and appropriate treatment with new coronary bypass grafts. In patients who developed AAD late after the initial operation, operative mortality was highest in patients without preoperative coronary angiography and appropriate management of their native coronary artery disease (CAD) and graft disease (OR, 5.36; 95% CI, 1.68–17.0; P=0.002). Only 12 of 27 (44%) patients with late AAD and previous CABG underwent preoperative coronary angiography (Table 2). Eleven of 14 (79%) of the patients without preoperative coronary imaging and who had not received new bypass grafts died of myocardial infarction or low cardiac output. Patients with late AAD who received new graft(s) blindly without preoperative imaging of the coronary status, most likely as bailout for failure of weaning from CPB, had a nearly 4-fold mortality in comparison with patients who underwent preoperative imaging (83% versus 24%; OR, 16.79; 95% CI, 1.81–155.8; P=0.006). In contrast, all patients with previous CABG who underwent preoperative coronary angiography and received new bypass grafts survived. Patients with late AAD after isolated AVR had a lower operative mortality than patients with isolated CABG that did not reach statistical significance (17% versus 33%; OR, 2.42; 95% CI, 0.65–9.01; P=0.298) (Table 2). After adjustment for preoperative angiography and new bypass grafting, isolated or concomitant AVR (OR, 0.55; 95% CI, 0.21–1.41; P=0.21) and mitral valve replacement (OR, 1.43; 95% CI, 0.38–5.35; P=0.59) were not associated with increased risk for operative mortality.

No other variables were associated with increased mortality in univariate analyses, including the acuity of dissection, emergency status, cardiac tamponade, delay time between presentation and surgical treatment, sex, center of treatment, arterial hypertension, entry site of dissection, diabetes mellitus, age, primary procedure, smoking status, body mass index, body surface area, left ventricular ejection fraction, CPB, cross-clamp time, interval, and aortic valve pathologic (all P>0.05). After adjustment for cross-clamp time and interval groups, CPB time (per minute) was independently associated with operative (OR, 1.007; 95% CI, 1.001–1.014; P=0.032) and overall mortality (hazard ratio, 1.006; 95% CI, 1.002–1.010; P=0.005). The surgical procedures for repair of AAD are listed in Table 2. Neither concomitant valve replacement nor (hemi)arch replacement was significantly associated with survival (P=0.460). Most patients (78%) were operated on with DHCA with a mean arrest time of 34±18 minutes (P=0.391 between groups). Significantly more patients operated on with DHCA (78%) than without DHCA (54%) survived (OR, 0.32; 95% CI, 0.12–0.85; P=0.034).

Forty-seven patients recovered without major complications and had a postoperative length of stay of a median of 11 (Q1–Q3: 9–16) days. Twenty-eight patients experienced one or more complications that included respiratory failure with slow weaning from the respirator, reintubation and tracheostomy, hemofiltration, sepsis, revision/reexploration for bleeding and secondary wound healing, implantation of a permanent pacemaker, and neurological complications including transitory psychotic syndrome, stroke, hemi- and paraplegia, focal neurological deficits, and polyneuropathy. One patient required coronary stenting after successful resuscitation. These 29 patients with one or more complications had a length of stay of a median of 24 (Q1–Q3: 19–51) days.

Long-term survival for each interval group is shown in Figure 2. Overall estimated 1- and 5-year survival rates were 66% (95% CI, 61%–71%) and 59% (95% CI, 54%–65%), respectively. With the use of Cox regression modeling, long-term mortality was significantly associated with previous CABG (hazard ratio, 2.04; 95% CI, 1.05–3.93; P=0.034), but not AVR, mitral valve replacement, time interval to AAD, preoperative angiography, new bypass grafting, or type of surgical repair.

Discussion

Nonaortic cardiac surgery is associated with a low but significant incidence of AAD. Operative mortality of AAD repair in this group of patients is high and depends on the type of initial surgery, time to AAD, and management factors. Patients with previous CABG had the poorest outcome. Preoperative coronary angiography and operative management of native CAD and graft disease had the strongest impact on survival.

The overall incidence of 0.15% in our cohort is consistent with previous reports that found AAD as a complication of cardiac surgery procedures in 0.12% to 0.6% of retrospective case series. Differences can be explained with varying definitions and inclusion criteria, such as cardiac catheterization and other invasive vascular treatments, aortic surgery including Ross procedures, or exclusive AVR. Other investigators have excluded intraoperative or chronic cases, or were unable to specify the time of AAD. The true incidence of cardiac surgery–associated
AAD is most likely underestimated. Studies usually report mortality rates only of patients who actually undergo surgery. Patients with previous cardiac surgery are twice as likely to be managed medically, perhaps because of a perceived excessive risk of surgery, and might not even be referred to a cardiac surgeon. Furthermore, a considerable number of patients do not reach the emergency department alive, and many immediate fatalities from acute AAD may go undiagnosed altogether. It has been estimated that early postoperative AAD underlies 3% to 5% of deaths after cardiac surgery.

Because there are important differences in clinical presentation, surgical management, and prognosis, we believe that intraoperative AAD must be distinguished from cases that occur in the early postoperative phase and from late postoperative AAD characterized by adhesions and the progression of the underlying disease. For the accurate comparison of data, it is essential to standardize definitions and be clear about inclusion and exclusion criteria. We suggest using the terms and definitions for intraoperative, early, and late (acute, chronic) postoperative AAD as applied here for future reporting.

In our study, we observed that AAD occurred mainly in patients with preexisting aortic wall pathology and hypertension at sites of iatrogenic mechanical trauma. The aortic wall was described at the initial surgery as thin or fragile in nearly all cases. Furthermore, the aortic diameter was 46±3 mm in all available imaging (CT/MRI) reports. This is in accordance with von Kodolitsch et al who found an aortic diameter of 43±10 mm in their series of patients who experienced AAD following AVR. According to convention and guidelines, this diameter per se does not necessitate aortic replacement at the time of AVR or CABG, but it is suggestive of aortic wall pathology. We found that most patients with aortic valve pathology at the initial surgery were diagnosed with aortic regurgitation. Taken together, aortic regurgitation, hypertension, and the fragility and thinning of the aortic wall at AVR were identified as strong and independent predictors for future aortic dissection, associated with a 14%, 22%, and 7% probability individually, or 64% to 96% with 2 or all 3 predictors combined.

A thin, fragile, and dilated aorta is typically associated with histopathologic changes such as MD. Nearly all histological examinations of excised aortic tissues in our study showed evidence of MD, atherosclerosis, or both. MD is characterized by an accumulation of basophilic ground substance (mucopolysaccharides) in the media with cystlike lesions, disruptions of collagen, elastic fragmentation, and apoptosis or necrosis of smooth muscle cells that results in weakening of the aortic tissue and higher wall stress. The presence of MD is typical of connective tissue diseases, but it was shown to precede aortic dissection in non-Marfan aortas as well. Chronic hypertension causes intimal thickening, fibrosis, calcification, and extracellular fatty acid deposition that affect arterial wall composition. In parallel, the extracellular matrix undergoes accelerated degradation, apoptosis, and elastolysis with hyalinization of collagen. Arterial hypertension is thus a key element in the etiology of MD. In particular, patients with an enlarged aorta (eg, >40 mm) at AVR in combination with hypertension and a thin or fragile aortic wall are likely to benefit from prophylactic aortic replacement without additional operative risk in experienced centers. Intuitively, a pathological aortic wall appears susceptible to dissection with mechanical trauma acting as a trigger. Luk et al analyzed the aortic histology in a series of patients with late AAD after previous cardiovascular surgery. All cases showed at least mild to moderate MD changes near or at the site of traumatic injury (stitch holes and knots, suture lines of aortotomy, or vein grafts) as demonstrated by a sharp discontinuity in the media of the aorta. Whereas most other reports could not comment on the entry tears owing to uncertainty and the lack of documentation, we were able to identify the site of entry with certainty in most (91%) of the cases. Typically, the aortotomy and the proximal graft anastomosis sites were identified as entry tear locations in two-thirds of cases with AVR and CABG, respectively. Dissections in the remaining patients originated from sites of clamping, cardioplegia, or cannulation. It is therefore imperative to minimize intimal and medial damage during surgery.

More than half of our group of patients with previous cardiac surgery had no or nonspecific symptoms when AAD was detected, in part, explaining the prolonged time from presentation to diagnosis and operative treatment of AAD. Besides a mandatory high index of suspicion, timely diagnosis crucially depends on imaging regardless of the clinical presentation. Nearly all of the patients with late chronic AAD in our cohort were either free of pain or had only mild and unspecific symptoms when AAD was detected, often incidentally. The explanation for the absence of pain in AAD is a matter of controversy. The tunica of the aortic wall is primarily composed of connective tissue and encloses the vasa vasorum and the nervi vasorum. Aortic pain is elicited by the stimulation of the nerve endings in the adventitia, notably through sudden stretching and tearing, converging in a pool of neurons in the posterior horn of the spinal cord. It has been suggested that slow or gradual dissection with less wall stretching and sparing of the adventitial layer may result in painless AAD. Previous mechanical trauma, inflammation, and consecutive scarring have the potential to damage the innervation of the vessel wall. Thus, painless AAD could be the result of denervation of the cardiac sympathetic nervous system secondary to the previous operation.

The most important acute and life-threatening symptom is the rupture of the proximal aorta into the pericardial cavity leading to cardiac tamponade. Up to 20% to 46% of patients with spontaneous AAD were reported to arrive in the operating room in unstable hemodynamic condition (shock or hypotension <90 mm Hg) as a clinical sign of tamponade. Conventional management therefore urges surgeons to operate on acute AAD without delay. Cardiac tamponade was predicted operatively in patients with spontaneous acute AAD, but was no longer statistically significant in the subgroup of patients with previous cardiac surgery. As an important finding in our study, neither cardiac tamponade nor the acuity of dissection or...
delay time (interval between presentation and surgical treatment) influenced operative mortality. Bayegan et al.\(^{26}\) distinguished impending and severe tamponade and found that only preoperative severe tamponade without palpable pulses, but not impending tamponade, was associated with an excessive risk for mortality. All 4 cases with cardiac tamponade in our study were classified as impending and referred to AAD in the early postoperative period. In that state, the tissue is still inflamed, sutures are not healed, coagulation is still compromised, and adhesions are not yet formed.

Importantly, severe compromising pericardial tamponade (rupture) occurred in only 1 of 67 patients (1%) with late AAD. The patient, however, was saved and survived for >13 years. All other patients were in a hemodynamically stable condition when they arrived in the operating room. This is in agreement with reports that found cardiac tamponade with hemodynamic compromise in only 4.0% to 5.9% of patients with AAD and previous cardiac surgery.\(^{11,18,25}\) It is supposed that the thickening of the aortic wall and scar adhesions from previous surgery contain the expansion of the dissecting aorta, so preventing or delaying rupture, tamponade, and instability.\(^{8,11,12,22}\) This low risk of rupture has come to suggest a less aggressive surgical approach, allowing time for clinical evaluation and coronary status assessment through preoperative coronary angiography.\(^{11,12,18,25}\) The strategy of delayed urgency substantially impacts operative planning and outcome.\(^{18,25,38}\)

Overall operative mortality in our entire cohort was 27%, in agreement with other reports.\(^{7,11,17}\) The cause of death in the majority of cases was myocardial infarction/low cardiac output, particularly in patients with previous CABG. Preoperative coronary angiography and operative management of native CAD and graft disease had the strongest impact on the outcome in patients with previous CABG. Gillinov et al.\(^{25}\) and Hirose et al.\(^{18}\) have reported an in-hospital mortality of only 5.6% and 14%, respectively, in patients with late AAD after previous cardiac surgery. The fundamental difference is the routine use of preoperative coronary angiography and aggressive coronary management in this patient group. Nearly all patients with a history of CABG (and 27% of patients without known CAD) required operative management of CAD at the time of AAD.\(^{18,25}\) Creswell et al.\(^{19}\) reported no operative mortality or perioperative myocardial infarction in patients who underwent preoperative coronary angiography and received new bypass grafts when CAD was known. In contrast, in-hospital mortality was 26% when angiography was not undertaken and CAD status remained unknown.

The role and safety of preoperative angiography in patients with acute AAD has been questioned, but the situation may fundamentally differ between spontaneous and late AAD following cardiac surgery. The safety and benefits of coronary angiography have repeatedly been shown in selected patients with spontaneous AAD, and with postcardiac surgery acute AAD, as well.\(^{18,25,38,39}\) Operative management of CAD is fundamentally different when the coronary status is known.\(^{18,25,38}\) The consequences of missing significant native CAD and occluded bypass grafts must therefore be weighted against the presumed risk of a slight delay. We found that severe tamponade is rare in this patient population, and it therefore appears prudent to obtain coronary angiography and schedule for surgery with delayed urgency in stable patients.\(^{11,18,25}\) As a future concept currently gaining popularity, the hybrid operating room enables the exact diagnosis of coronary status and aortic pathologies without time delay.\(^{39}\) Furthermore, electron beam CT and multislice spiral CT allow simultaneous diagnosis of aortic dissection and CAD as an alternative to intracoronary modalities.\(^{40}\)

This study shares the limitations associated with all post hoc analyses. Because our analysis was focused on patients presenting with AAD after previous nonaortic cardiac surgery, it was not designed to identify predicting risk factors for aortic dissection itself. Extensive data have been collected over a period of several years in multiple centers. Not all records were provided as complete data sets. Nevertheless, our data exceed and extend previous reports, particularly in combining primary documentation, identified entry sites of dissection, histopathologies, and complete follow-up. Descriptions in surgical records by definition carry an element of subjectivity. Nearly all surgeons' notes were supported by imaging and histology reports, but there could still be bias attributable to under- or overreporting because not all reports included a description. Because our study was based on multicenter-derived data, we were concerned about heterogeneity between centers, but found no differences for variables relating to patient characteristics, surgical management, and outcome. Differences in individual patient management reflect real-life conditions and furthermore allowed the analysis and identification of characteristics with potential for risk assessment and improving surgical outcome. Most likely, the true incidence of AAD following routine cardiac surgery is grossly underestimated. Further insight into the pathogenesis and the potential for early recognition, optimal patient management, and surgical outcome can be expected with larger patient numbers enabling extended analyses.

In conclusion, nonaortic cardiac surgery is associated with a low but significant incidence of AAD and must be expected at any time. Preexisting aortic wall pathology and hypertension are associated with AAD occurring at sites of iatrogenic surgical trauma. The operative mortality of AAD repair in this group of patients is high and depends on the primary procedure, the time interval between the initial cardiac surgery and AAD, and surgical management. The single most important factor that was associated with a lower operative mortality in our study was the implementation of preoperative coronary angiography and appropriate operative management of CAD and graft disease in patients with previous CABG. Because cardiac tamponade and hemodynamic instability are rare, patients with previous cardiac surgery who are in stable condition should undergo preoperative native coronary and graft status assessment and appropriate operative planning including the use of DHCA and, most importantly, aggressive revascularization.

**Disclosures**

None.
References


CLINICAL PERSPECTIVE

Nonaortic cardiac surgery is associated with a low but significant incidence of type A aortic dissection (AAD). Because there are important differences in clinical presentation, surgical management, and prognosis between spontaneous and late AAD following cardiac surgery, this large multicenter study sought to determine the conditions associated with the high surgical mortality of AAD repair as a complication of nonaortic cardiac surgery and to identify management pathways to improve outcome. We found that operative mortality depends on the type of initial surgery, time to AAD, and, importantly, influenceable management factors. AAD occurred mainly in patients with preexisting aortic wall pathology, identifiable with a slightly enlarged aorta, and hypertension at sites of iatrogenic mechanical trauma. This finding emphasizes most careful surgical handling and potentially a low threshold for prophylactic replacement of the ascending aorta that is without additional operative risk in experienced centers. Diagnosis of AAD warrants a mandatory high index of suspicion and depends primarily on imaging. The majority of patients with previous cardiac surgery had no or nonspecific symptoms when AAD was detected. The single most important factor that was associated with a lower operative mortality was implementation of preoperative coronary angiography and appropriate operative management of CAD and graft disease in patients with previous CABG. Because cardiac tamponade and hemodynamic instability are rare (1%), patients with previous cardiac surgery in stable condition should undergo preoperative native coronary and graft status assessment and appropriate operative planning, including aggressive revascularization and the use of deep hypothermic cardiac arrest.
Type A Aortic Dissection After Nonaortic Cardiac Surgery
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