Predictors of Proximal Aortic Dissection at the Time of Aortic Valve Replacement

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Background—Type I aortic dissection develops in 0.6% of patients late after aortic valve replacement (AVR), and 13% of patients with type I aortic dissections have a history of AVR. Predictors of aortic dissection at AVR, however, have not been characterized.

Methods and Results—A study group of 33 patients with type I aortic dissection had aortic surgery 49±55 months after routine AVR. A group of 101 controls, who did not have morphological progression of aortic diameters ≥6 years after AVR, was used to identify predictors of postsurgical dissection. Multivariate analysis identified aortic regurgitation (P<0.002) and fragility (P<0.001) or thinning of the aortic wall (P<0.007) at AVR as predictors, associated with a 14%, 22%, and 7% probability of late aortic dissection, respectively. Clamping times, types of valve prostheses, concomitant coronary artery bypass grafting, and mean ascending aortic diameters of 43±10 mm at AVR did not predict late dissection. A separate analysis of 29 nondissecting aneurysms of the ascending aorta developing 104±64 months after routine AVR revealed younger age at AVR (P<0.003) and congenitally bicuspid aortic valves (P<0.03) as predictors of late aneurysm formation.

Conclusions—Aortic regurgitation combined with fragile and thinned aortic walls in patients with moderate aortic dilation may reflect aortic root disease, with a high risk for postsurgical aortic sequelae if it is treated incompletely by isolated valve replacement. (Circulation. 1999;100[suppl II]:II-287–II-294.)

Key Words: aneurysm ■ aorta ■ risk factors ■ surgery ■ valves

Type I aortic dissection occurs in 0.6% of patients late after aortic valve replacement (AVR).1–13 Moreover, 13% of patients with acute type I aortic dissections have a history of previous AVR.1–13,14–16 A lethal outcome is reported in 50% of such dissections occurring within 1 month to 16 years after AVR.1,2,7,9,12,17–31 Combining AVR with prophylactic replacement of the ascending aorta is a treatment that may prevent aortic dissection; criteria for prophylactic aortic surgery at AVR, however, are established exclusively for Marfan patients.8,12,29,32

This retrospective study was performed to assess clinical and pathoanatomical characteristics of non-Marfan patients developing type I aortic dissection late after routine AVR. A control group with stable aortic diameters who were followed for a minimum of 6 years after AVR was used to identify predictors of late dissection. These predictors may be used as an aid to estimate the risk of subsequent dissection at the time of routine AVR. A similar analysis was performed to identify predictors for progressive nondissecting aneurysm of the ascending aorta at AVR.

Methods

Patients

Between January 1979 and December 1997, 666 consecutive patients had surgery for type I aortic dissection (210 men and 90 women with a mean age of 54±13 years)13 or a nondissecting aneurysm of the ascending aorta (231 men and 135 women with a mean age of 54±15 years). Aortic surgery was performed at the University Hospital of Eppendorf, Hamburg; St. Georg General Hospital, Hamburg; Hannover Medical School, Hannover; and Christian-Albrechts-University, Kiel; all in Germany. A total of 33 of the type I aortic dissections and 29 of the nondissecting aneurysms of the ascending aorta were associated with previous AVR (Tables 1 and 2). In the group of type I aortic dissections, 28 cases were acute and 5 were chronic (15%; cases 4, 19, 24, 25, and 30), with onset of pain within or after 14 days of admission, respectively.34 In the group of patients with nondissecting aneurysms after AVR, aortic surgery was performed electively for progression of an aortic diameter >50 mm in 24 cases and as an emergency repair for rapid expansion or rupture in 5 cases (17%; patients 38, 47, 52, 57, and 62).

In an attempt to identify features at AVR associated with the risk of either late postsurgical dissection or aneurysm, we selected a group of 101 consecutive patients (74 men and 27 women; mean age, 63±17 years) who had routine AVR performed from January 1989...
through December 1991 at the University Hospital of Eppendorf. This control group had $6$ years of uneventful follow-up at our institution (mean, 94±9 months), with no morphological progression in the diameter of the ascending aorta, as assessed by transesophageal echocardiography, computed tomography, or magnetic resonance imaging (MRI). In this group, the aortic diameter measured at AVR was 41±10.1 mm. Stigmata of Marfan syndrome, acute endocarditis, and concomitant mitral valve surgery at the time of AVR excluded patients from this group, and complete documentation was required for inclusion in a prediction model.

### Study Variables

Sixteen variables were assessed in each patient. These included (1) age at the time of AVR, (2) sex, (3) cardiac functional class (New York Heart Association), (4) reduced left ventricular ejection fraction ($<25\%$), and (5) an angiographic diagnosis of coronary heart disease, as derived from preoperative medical records. The type of aortic valve disease was categorized according to echocardiographic and/or angiographic findings as (6) aortic stenosis, (7) aortic regurgitation, or (8) a combination of both. Surgical records at AVR were used to assess (9) a congenitally bicuspid aortic valve, (10) the maximum diameter of the ascending aorta (mm), and (11) thinning or (12) fragility of the aortic wall (according to intraoperative inspection and palpation). (13) Concomitant coronary artery bypass grafting and (14) time of aortic cross-clamping (minutes) were also assessed in each patient. In 15 patients with no intraoperative measurements available at AVR (24%), images from preoperative transesophageal echocardiography, computed tomography, MRI, and/or angiography were reevaluated for aortic diameters. Images were also screened for (15) aortic coarctation in all patients. (16) Chronic systemic hypertension was considered present with evidence of long-standing hypertension $\geq 2$ years before AVR.

### TABLE 1. Characteristics of Patients With Aortic Dissection After Previous AVR

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Time From AVR to AD, mo</th>
<th>Risk Factors</th>
<th>Findings at AVR</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55/M</td>
<td>43</td>
<td>-</td>
<td>AR 40</td>
<td>Alive 15.2 d</td>
</tr>
<tr>
<td>2</td>
<td>58/M</td>
<td>23</td>
<td>-</td>
<td>AR 41 Thin +</td>
<td>5.6 y</td>
</tr>
<tr>
<td>3</td>
<td>55/M</td>
<td>52</td>
<td>+</td>
<td>AR ... Thin, frag +</td>
<td>8.2 mo</td>
</tr>
<tr>
<td>4</td>
<td>63/M</td>
<td>39</td>
<td>-</td>
<td>AR ... +</td>
<td>12.2 y</td>
</tr>
<tr>
<td>5</td>
<td>64/M</td>
<td>24</td>
<td>-</td>
<td>AR 36 ... +</td>
<td>3.7 y</td>
</tr>
<tr>
<td>6</td>
<td>58/F</td>
<td>2</td>
<td>-</td>
<td>AR 30 Thin frag</td>
<td>5.0 mo</td>
</tr>
<tr>
<td>7</td>
<td>48/F</td>
<td>6</td>
<td>-</td>
<td>AR 40 Thin</td>
<td>1.6 d</td>
</tr>
<tr>
<td>8</td>
<td>51/M</td>
<td>2</td>
<td>+</td>
<td>AR 49 Soft thick</td>
<td>6.4 d</td>
</tr>
<tr>
<td>9</td>
<td>62/M</td>
<td>12</td>
<td>-</td>
<td>AR ... Frag</td>
<td>1.2 y</td>
</tr>
<tr>
<td>10</td>
<td>53/M</td>
<td>4</td>
<td>+</td>
<td>AR 35 Thin +</td>
<td>4.2 y</td>
</tr>
<tr>
<td>11</td>
<td>35/M</td>
<td>21</td>
<td>-</td>
<td>AR 58 +</td>
<td>4.1 y</td>
</tr>
<tr>
<td>12</td>
<td>70/F</td>
<td>36</td>
<td>-</td>
<td>AR 50 Frag</td>
<td>2.0 mo</td>
</tr>
<tr>
<td>13</td>
<td>63/M</td>
<td>68</td>
<td>-</td>
<td>AS/AR 29 Thin +</td>
<td>4.9 y</td>
</tr>
<tr>
<td>14</td>
<td>54/M</td>
<td>6</td>
<td>+</td>
<td>AR 73 Thin -</td>
<td>Operative death</td>
</tr>
<tr>
<td>15</td>
<td>59/F</td>
<td>48</td>
<td>-</td>
<td>AR 54 Thin +</td>
<td>3.6 y</td>
</tr>
<tr>
<td>16</td>
<td>66/M</td>
<td>47</td>
<td>-</td>
<td>AR 35 Thin frag</td>
<td>Operative death</td>
</tr>
<tr>
<td>17</td>
<td>61/M</td>
<td>63</td>
<td>+</td>
<td>AS/AR ... Normal +</td>
<td>6.2 y</td>
</tr>
<tr>
<td>18</td>
<td>71/M</td>
<td>41</td>
<td>+</td>
<td>AR ... +</td>
<td>4.8 y</td>
</tr>
<tr>
<td>19</td>
<td>48/M</td>
<td>12</td>
<td>-</td>
<td>AR 41 Thin +</td>
<td>3.3 y</td>
</tr>
<tr>
<td>20</td>
<td>42/M</td>
<td>164</td>
<td>-</td>
<td>AR 39 Thin</td>
<td>4.5 mo</td>
</tr>
<tr>
<td>21</td>
<td>16/M</td>
<td>23</td>
<td>-</td>
<td>AS/AR 40 ... +</td>
<td>4.7 y</td>
</tr>
<tr>
<td>22</td>
<td>33/M</td>
<td>190</td>
<td>-</td>
<td>AS/AR 27 Normal</td>
<td>3.9 y</td>
</tr>
<tr>
<td>23</td>
<td>35/F</td>
<td>72</td>
<td>-</td>
<td>AR 49 Extr thin -</td>
<td>4.5 y</td>
</tr>
<tr>
<td>24</td>
<td>62/M</td>
<td>54</td>
<td>-</td>
<td>AR/AS 50 Extr thin</td>
<td>3.6 y</td>
</tr>
<tr>
<td>25</td>
<td>45/M</td>
<td>22</td>
<td>-</td>
<td>AR 51 Frag, thick +</td>
<td>6.3 y</td>
</tr>
<tr>
<td>26</td>
<td>72/F</td>
<td>5</td>
<td>-</td>
<td>AR/AS 40 Extr thin +</td>
<td>6.3 mo</td>
</tr>
<tr>
<td>27</td>
<td>59/M</td>
<td>4</td>
<td>-</td>
<td>AR 49 Thin +</td>
<td>6.1 y</td>
</tr>
<tr>
<td>28</td>
<td>77/M</td>
<td>64</td>
<td>+</td>
<td>AS ... Thin, frag +</td>
<td>3.8 y</td>
</tr>
<tr>
<td>29</td>
<td>66/F</td>
<td>16</td>
<td>-</td>
<td>AS/AR ... Normal</td>
<td>3.7 y</td>
</tr>
<tr>
<td>30</td>
<td>38/M</td>
<td>84</td>
<td>+</td>
<td>AS ... Thin -</td>
<td>3.4 y</td>
</tr>
<tr>
<td>31</td>
<td>52/M</td>
<td>196</td>
<td>+</td>
<td>AS 46 +</td>
<td>3.6 mo</td>
</tr>
<tr>
<td>32</td>
<td>57/M</td>
<td>180</td>
<td>-</td>
<td>AR 39 Normal +</td>
<td>4.5 y</td>
</tr>
<tr>
<td>33</td>
<td>60/M</td>
<td>1</td>
<td>+</td>
<td>AR 48 Frag</td>
<td>2.4 d</td>
</tr>
</tbody>
</table>

HTN indicates chronic systemic hypertension; CMN, cystic media necrosis; CBAV, congenitally bicuspid aortic valve; +, present; −, absent; zzz, not available; MDA, maximum diameter of the ascending aorta; AD, type I aortic dissection; F/U, follow-up; extr thin, extremely thin; frag, fragile; AR, aortic regurgitation; and AS, aortic stenosis.
Both location and distal extent of post-AVR dissection or aneurysm, maximum ascending aortic diameter, and leakage of the aortic valve prosthesis were assessed from findings on transesophageal echocardiography, computed tomography, MRI, and/or angiography performed before aortic surgery. Surgical records were used to assess the location of the entrance tear to the aortic dissection. Histological examination of the ascending aorta was done for evidence of cystic media necrosis (considered present in at least a moderate degree) in all study patients but not in controls. Follow-up after aortic surgery was obtained through repeat visits at the outpatient clinic and/or by communication with the patient’s primary physician; 9 patients (14%; patients 3, 26, 31, 45, 48, 51, 53, 54, and 55) were lost to follow-up.

**Literature Review**

The English, French, and German literature was screened for reports on aortic dissection or aneurysm developing after AVR using Medline (key words: aneurysm, aorta, risk factors, surgery, valves) and literature lists provided in articles on this subject. Detailed results of this survey are shown in Tables 3 and 4.

**Statistical Analysis**

The risk of developing aortic dissection or aneurysm after AVR associated with the presence of each of the 16 variables was evaluated separately for aortic dissection and aneurysm by univariate, unconditional logistic regression analysis with SAS. Age, aortic diameters, and time of aortic cross-clamping were analyzed as continuous variables, and they are given as mean ± SD. Variables emerging as significant predictors of risk at the 5% level were included in a multivariate model. Estimates of risk (odds ratios) were calculated based on the coefficients from the logistic models. Predicted probabilities of disease (P[D]) given a particular variable (Xi) were calculated from the coefficients (bi) in the final multivariate models using the equation

\[
P(D) = \frac{e^{b_0 + b_1 X_1}}{1 + e^{b_0 + b_1 X_1}}
\]

where b0 is a constant. Comparison between groups and characteristics was performed by Fisher’s exact test; the level of significance was set at 5%.

**Results**

**Findings at Aortic Surgery**

Patients with type I aortic dissection and nondissecting ascending aortic aneurysm after previous AVR had similar age (55±13 versus 51±13 years) and sex distributions (79%
men in both groups). Surgery for aortic dissection and nondissecting aneurysm was performed 49±55 and 104±64 months, respectively, after previous AVR (P<0.01). Diameters of the ascending aorta were similar after postsurgical dissection and aneurysm (67±21 versus 63±9 mm), whereas involvement of the descending aorta was found only in patients with aortic dissection (54%; patients 1 to 18).

Paravalvular leakage was more frequent in the aneurysm group than in the dissection group (59% versus 12%; P=0.01). In all postsurgical dissections, the entry site was located in the ascending aorta; in 45% of these, the entry was found 1 to 3 cm above the site of previous aortotomy, in 17%, previous cross-clamping and cannulation sites were excluded as entry locations, and in 38%, the precise location in the ascending aorta was not described. In 3 dissections, vena cava obstruction syndrome was observed (9%; patients 15, 24, and 25). Thirty-day survival was similar in the postsurgical dissection and aneurysm groups (82% versus 90%; Tables 1 and 2).

Findings at AVR

At AVR, patients with late dissection and aneurysm had similar maximum aortic diameters (43±10 versus 47±9 mm) and New York Heart Association functional classes (2.5±0.8 versus 2.0±0.7); the 2 groups also had an equal distribution of patients with systemic hypertension (33% versus 24%),

TABLE 3. Reported Prevalence of Type A Aortic Dissection After Previous AVR

<table>
<thead>
<tr>
<th>Reference</th>
<th>Cases with AD after AVR, n</th>
<th>AVR All Procedures, n</th>
<th>Late AD, %</th>
<th>AD All Cases, n</th>
<th>Previous AD, n (%)</th>
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</thead>
<tbody>
<tr>
<td>Muna1</td>
<td>26</td>
<td>3785</td>
<td>0.7</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Miller15</td>
<td>6</td>
<td>...</td>
<td>...</td>
<td>121</td>
<td>6 (5)</td>
</tr>
<tr>
<td>Bachter2</td>
<td>5</td>
<td>1117</td>
<td>0.4</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Burckhardt3</td>
<td>2</td>
<td>88</td>
<td>2.3</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Cohn4</td>
<td>2</td>
<td>912</td>
<td>0.2</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Douglas5</td>
<td>1</td>
<td>187</td>
<td>0.5</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Crawford6</td>
<td>54</td>
<td>...</td>
<td>...</td>
<td>288</td>
<td>54 (19)</td>
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<td>Lytle6</td>
<td>8</td>
<td>1689</td>
<td>0.5</td>
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<td>...</td>
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<tr>
<td>Hedoire7</td>
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<td>640</td>
<td>0.6</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Carrel8</td>
<td>11</td>
<td>1593</td>
<td>0.7</td>
<td>...</td>
<td>...</td>
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<tr>
<td>Presbitero9</td>
<td>10</td>
<td>1499</td>
<td>0.7</td>
<td>134†</td>
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</tr>
<tr>
<td>Aris10</td>
<td>1–5‡</td>
<td>378</td>
<td>0.3–1.3</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>David11</td>
<td>1</td>
<td>374</td>
<td>0.3</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Prenger12</td>
<td>4</td>
<td>677</td>
<td>0.6</td>
<td>42</td>
<td>10 (24)</td>
</tr>
<tr>
<td>Epperlein14</td>
<td>8</td>
<td>...</td>
<td>...</td>
<td>79</td>
<td>8 (10)</td>
</tr>
<tr>
<td>von Kodolitsch15</td>
<td>12</td>
<td>1503</td>
<td>0.8</td>
<td>80</td>
<td>12 (15)</td>
</tr>
<tr>
<td>Total</td>
<td>154</td>
<td>14 442</td>
<td>0.6</td>
<td>744</td>
<td>101 (13)</td>
</tr>
</tbody>
</table>

*2 cases with rupture of a nondissecting aneurysm of the ascending aorta; †11 cases with types I and II aortic dissection; ‡unclear how many patients between 1 and 5 had aortic dissection. Abbreviations as in Table 1.

### TABLE 4. Reports on Type A Aortic Dissection After Previous AVR

<table>
<thead>
<tr>
<th>Reference</th>
<th>Time from AVR to AD, y</th>
<th>HTN</th>
<th>CMN</th>
<th>CBAV</th>
<th>Risk Factors</th>
<th>Findings at AVR</th>
<th>30-day Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Derkac17</td>
<td>1.3</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>–</td>
</tr>
<tr>
<td>Miller15</td>
<td>6</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>–</td>
</tr>
<tr>
<td>Spitzer18</td>
<td>6.7</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>–</td>
</tr>
<tr>
<td>Fukuda19</td>
<td>0.8</td>
<td>+</td>
<td>–</td>
<td>+</td>
<td>AR/AS</td>
<td>Mono</td>
<td>–</td>
</tr>
<tr>
<td>Muna1</td>
<td>0.3</td>
<td>+</td>
<td>+</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>–</td>
</tr>
<tr>
<td>Charnsangarvej20</td>
<td>0.7</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>AR/AS</td>
<td>Mono</td>
<td>–</td>
</tr>
<tr>
<td>Gooch21</td>
<td>13</td>
<td>–</td>
<td>+</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>+</td>
</tr>
<tr>
<td>Orszulak22</td>
<td>9</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>AR/AS</td>
<td>Caged ball</td>
<td>+</td>
</tr>
<tr>
<td>70/M</td>
<td>4</td>
<td>+</td>
<td>–</td>
<td>–</td>
<td>AS</td>
<td>Bio</td>
<td>–</td>
</tr>
<tr>
<td>Reference</td>
<td>Age/Sex</td>
<td>Time from AVR to AD, y</td>
<td>Risk Factors</td>
<td>Findings at AVR</td>
<td>Prosthesis</td>
<td>30-day Survival</td>
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<tr>
<td>-------------------</td>
<td>---------</td>
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<td>-----------------</td>
<td>------------</td>
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<tr>
<td>Le Heuzey</td>
<td>56/M</td>
<td>15</td>
<td>– + + +</td>
<td>AR ...</td>
<td>Caged ball</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td></td>
<td>61/M</td>
<td>0.6</td>
<td>+ + +*</td>
<td>AR + Mono</td>
<td>+</td>
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</tr>
<tr>
<td>74/M</td>
<td></td>
<td>2</td>
<td>– – + AR/AS</td>
<td>+ Caged ball</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bachet</td>
<td>. . /F</td>
<td>4</td>
<td>– – (MFS)</td>
<td>AR +</td>
<td>Caged ball</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40/M</td>
<td>5</td>
<td>– + –*</td>
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<td>. . /M</td>
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<td>– + –</td>
<td>AR + Mono</td>
<td>+</td>
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<td>. . /M</td>
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<td>– + –*</td>
<td>AR + Mono</td>
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<td></td>
<td></td>
<td>6</td>
<td>– – + AR</td>
<td>+ Caged ball</td>
<td>+</td>
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<td>Stone</td>
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<td>– – –</td>
<td>AR ...</td>
<td>Bio</td>
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<td></td>
</tr>
<tr>
<td></td>
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</table>

AR indicates aortic root dilatation; MFS, Marfan syndrome; caged ball, Starr-Edwards or Smeloff-Cutter prostheses; and mech, mechanical valve prosthesis of unknown type. Other abbreviations as in Table 1.

*Aortic wall fragility; †this series includes 7 men and 5 women.
TABLE 5. Logistic Regression Analysis

<table>
<thead>
<tr>
<th>Variables</th>
<th>Univariate Analysis</th>
<th>Multivariate Analysis</th>
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<tr>
<td></td>
<td>OR 95% CI  P</td>
<td>OR 95% CI  P P(D)</td>
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<td>AVR with late aortic dissection</td>
<td></td>
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<tr>
<td>Fragile aortic wall</td>
<td>17.29 4.25–70.44 0.0001</td>
<td>22.34 3.31–150.58 0.001 22.1</td>
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<td>Aortic regurgitation</td>
<td>5.26 2.20–12.55 0.0002</td>
<td>12.88 2.63–63.09 0.002 14.1</td>
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<td>Thin aortic wall</td>
<td>5.43 2.17–13.57 0.0003</td>
<td>6.35 1.65–24.37 0.007 7.5</td>
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<td>Chronic arterial hypertension</td>
<td>3.39 1.34–8.57 0.01</td>
<td>3.02 0.51–17.94 0.22 3.7</td>
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<td>Age, y</td>
<td>0.97 0.95–0.99 0.02</td>
<td>0.97 0.94–1.01 0.20 ...</td>
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<td>AVR with late nondissecting aortic aneurysm</td>
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<tr>
<td>Age, y</td>
<td>0.96 0.94–0.98 0.002</td>
<td>0.96 0.93–0.99 0.003 ...</td>
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<td>Bicuspid aortic valve</td>
<td>5.98 1.74–20.63 0.005</td>
<td>4.68 1.21–18.13 0.03 57.3</td>
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</table>

OR indicates odds ratio; 95% CI, 95% confidence interval; and P(D), probability (percent) of developing a dissection or aneurysm after AVR.

Prediction of Dissection and Aneurysm at AVR

Of the 16 variables assessed in this study, multivariate analysis revealed aortic wall fragility (P<0.001), aortic regurgitation (P<0.002), and aortic wall thinning (P<0.007) as independent predictors of late dissection. According to this model, the probability of late dissection associated with each predictor is 22%, 14%, and 7%, respectively. Aortic wall fragility in combination with aortic thinning or aortic regurgitation increases the probability of late dissection to 64% and 79%, respectively; presence of all 3 predictors results in a 96% probability of dissection. Younger age at AVR (P<0.003) and a congenitally bicuspid aortic valve (P<0.03) were predictors of late nondissecting aortic aneurysm on multivariate analysis (Table 5).

Discussion

The 30-day survival in patients who had dissection after previous AVR was 82% in our study cohort, whereas analysis of the literature documents a lethal outcome in 50% of such patients (Tables 3 and 4). Moreover, in 14 442 patients who underwent AVR in 13 different centers, 0.6% developed type I aortic dissection 1 month to 16 years after AVR (Tables 3 and 4). Similarly, 13% of 744 reported type I aortic dissections revealed a history of AVR (Table 3). Thus, our analysis confirms a high prevalence of previous AVR in type I aortic dissection (11%) and documents a similar proportion of previous AVR in nondissecting aneurysms of the ascending aorta (8%).

Surgical Techniques

In our population, the intimal tear was located 1 to 3 cm above the previous aortotomy in 45% of postsurgical dissections. However, the site of the aortotomy itself was intact in all cases, and it was not involved in the dissecting process in any case. Moreover, no evidence exists that any of the dissections arose from the site of previous aortic cross-clamping or cannulation. Similarly, logistic regression analysis excluded any association of aortic clamping time, concomitant coronary artery bypass grafting, or type of aortic valve prosthesis with postsurgical dissection or aneurysm. The literature review also failed to identify the predominance of a specific aortic valve prosthesis in postsurgical cases of dissection (Table 4).1,2,7,9,12,17–31 Dissections at clamping or cannulation sites are usually observed at the time or soon after surgery.39 Thus, we think that dissections late after AVR are usually not related to specific operative features or techniques.

Risk for Late Dissection

Aortic diameters <50 mm usually carry a low risk of dissection or rupture.32,40 Interestingly, the present study reveals that postsurgical dissection developed in 33 patients who had a mean aortic diameter of only 43±10 mm at the time of valve replacement. Moreover, multivariate analysis excludes the aortic diameter at AVR as an independent predictor of dissection. Thus, in patients with moderate aortic dilation at AVR, additional factors play a role in the subsequent development of late aortic dissection. Multivariate analysis identified fragility of the aortic wall, aortic regurgitation, and aortic wall thinning as independent predictors of dissection. Assessment of these independent risk variables at
AVR permits prediction of the risk of late dissection. In addition, bicuspid aortic valves, systemic hypertension, and cystic media necrosis were found in 15%, 33%, and 39% of postsurgical dissections, respectively; statistical analysis, however, failed to establish their association with aortic dissection. Analysis of 57 dissections reported late after AVR confirms a high prevalence of aortic regurgitation (92%), arterial hypertension (57%), cystic media necrosis (39%), and bicuspid aortic valves (24%) (Table 4).

It had been shown that—in contrast to previous AVR—previous isolated coronary artery bypass grafting is not an independent risk factor for type I aortic dissection. Intact native aortic valves probably exclude the presence of marked aortic root disease (comprising aortic regurgitation with aortic wall abnormalities) and may thus explain a lower risk for postsurgical aortic dissection in isolated coronary artery bypass grafting procedures.

**Risk for Late Nondissecting Aneurysm**

Younger age at AVR and presence of a congenitally bicuspid aortic valve in moderately dilated aortas predict nondissecting aortic aneurysms after AVR but not late dissection. Predominance of aortic valve stenosis, lower prevalence of aortic wall thinning, absence of wall fragility, and a longer time interval between AVR and aortic surgery in nondissecting aneurysms suggest that different mechanisms exist in the development of nondissecting aneurysms and dissection after AVR. Moreover, the high prevalence of paravalvular leakage after AVR indicates a potentially causative role in the pathogenesis of nondissecting progressive aneurysms.

**Study Limitations**

The present study was conducted in a retrospective fashion and includes some subjective data, such as thinness and fragility of the aortic wall. However, aortic wall thinness and/or fragility were described independently by 20 different surgeons from 4 surgical centers. In addition, reports from 4 centers confirm aortic wall fragility as a key finding in 5 cases of AVR later complicated by aortic dissection (Table 4). Future high-resolution tomography and aortic densitability measurements may be used for noninvasive and objective assessment of potential wall abnormalities.

**Conclusions**

Type I aortic dissection after AVR seems unrelated to surgical techniques. Patients with aortic diameters ≥43 mm at AVR should have intraoperative assessment of risk factors for potential late aortic dissection. Aortic wall fragility in combination with aortic thinning or aortic regurgitation carries a 64% or 79% probability, respectively, of late dissection, which increases to a 96% probability of dissection in the presence of all three predictors. Thus, any patient at AVR with an aortic diameter ≥43 mm and the presence of at least 2 predictors of late dissection will likely benefit from prophylactic aortic surgery. The combined presence of these predictors identifies a disease process of the entire aortic root rather than isolated valve disease. We thus suggest that in such patients, surgical treatment should encompass the entire aortic root. Prospective studies, however, may be required for defining optimal prophylactic surgical techniques.

**Acknowledgments**

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**References**


Predictors of Proximal Aortic Dissection at the Time of Aortic Valve Replacement
Yskert von Kodolitsch, Ognjen Simic, Ann Schwartz, Christoph Dresler, Roger Loose, Martin Staudt, Jörg Ostermeyer, Axel Haverich and Christoph A. Nienaber

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