Rupture of the Device Landing Zone During Transcatheter Aortic Valve Implantation
A Life-Threatening But Treatable Complication

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Background—Iatrogenic damage of different structures of the aortic root, in the region of the so-called “device landing zone,” may occur during transcatheter aortic valve implantation (TAVI). It is mostly considered difficult to treat or even untreatable.

Methods and Results—We performed a retrospective analysis of the occurrence, clinical presentation, treatment, and outcome of iatrogenic rupture in the device landing zone in a series of 618 consecutive patients who underwent TAVI at our institution between April 2008 and October 2011. The incidence of rupture was 1% (6 patients). The correct diagnosis was established during TAVI procedures in 4 and postmortem in 2 patients. The major sign of the aortic rupture was apparent bleeding in 4 patients and failure of myocardial recovery after valve implantation in 1; it was asymptomatic in 1 patient. The iatrogenic rupture in the region of the device landing zone was treated surgically in 5 patients and only conservatively in the patient without symptoms. When the diagnosis was established correctly during TAVI, only 1 of 4 patients died (25%). The overall mortality rate was 50% (3 of 6 patients died).

Conclusions—Rupture of different structures in the device landing zone during TAVI is a life-threatening complication that can be treated successfully if it is immediately recognized and adequately managed. (Circ Cardiovasc Interv. 2012; 5:424-432.)

Key Words: valves • prosthesis • surgery • TAVI • annulus rupture • transapical • transcatheter

Transcatheter aortic valve implantation (TAVI) poses a high risk of possible dangerous and life-threatening complications that can occur at any moment during the procedure.1–7 Different iatrogenic damages may take place at various levels of the aortic root8–11 in the so-called “the device landing zone.”8 In contrast to a standard surgical procedure, if the complications do occur, they are very difficult to control.9 Therefore, it is necessary to be aware of that fact and to take all possible measures to prevent the complications.3

We report on our single-center experience with iatrogenic rupture in the device landing zone in a series of more than 600 TAVI procedures and retrospectively analyzed the occurrence, risk factors, diagnostic and therapeutic errors, treatments, and outcome.

Methods

Patients
Between April 2008 and October 2011, 618 patients with severe aortic valve disease underwent TAVI. The mean age of the patients was 79.5±8.1 years (range, 29–99 years). There were 369 (59.7%) female and correspondingly 40.3% (249) male patients. There were 189 (30.6%) patients in New York Heart Association class IV. The mean logistic EuroSCORE (European System for Cardiac Operative Risk Evaluation) was 34.6±20.9% (range, 2% to 97%) and the mean STS (Society of Thoracic Surgeons) score was 16.0±14.2% (range, 1% to 90%). Transapical TAVI was used in most patients (77.4%; n=478). The transfemoral approach was used in 122 (19.7%), transaxillary in 16 (2.6%), and others in 2 (0.3%). Balloon-expandable valves (Edwards-Sapien THV, Edwards Lifesciences, Irvine, CA) were used in 502 (81.2%) patients and self-expandable valves (CoreValve Revalving System, Medtronic Inc, MN) in 116 (18.8%). The overall 30-day mortality was 5.8% (36/618), and in patients without cardiogenic shock it was 5.0% (29/585). The retrospective analyses identified 6 patients with rupture in the region of the device landing zone. Written informed consent for the TAVI procedures was obtained from all patients or their representatives. The study was approved by our institutional review board.

General Procedural Considerations
All procedures were performed by our heart team, consisting of 5 surgeons, 2 interventional cardiologists, and 2 anesthesiologists with

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WHAT IS KNOWN
- The aortic annulus and other structures of the aortic root may be damaged during TAVI.
- Rupture is mostly considered difficult to treat or even untreatable.

WHAT THE STUDY ADDS
- Early recognition and treatment can improve outcome.
- Consider any arterial bleeding with no identifiable cause as suspected annulus rupture.
- Immediately institute cardiopulmonary bypass, convert TAVI to conventional aortic valve replacement, and repair the lesion, even in patients who are considered formally “inoperable.”

Expertise in transesophageal echocardiography (TEE). The procedures were performed in our hybrid operating room with a monoplane angiography system (Siemens Artis zee, Siemens AG, Munich, Germany), using the standard technique with some modifications. The procedures were monitored by fluoroscopy, angiography, and continuous TEE.

The patient selection, indications, and contraindications for TAVI, reasons for preferred use of transapical approach, and outcomes from our institution have been published elsewhere.

Assessment of the Device Landing Zone (Left Ventricular Outflow Tract, Annulus, Cusps, Sinuses of Valsalva, Proximal Aorta)
The annulus was assessed preoperatively using transthoracic echocardiography (TEE) (parasternal long-axis view) and multislice computed tomography (MSCT) (coronal and sagittal views) and immediately before TAVI by TEE (midesophageal short- and long-axis views; [for the measurement of the annulus diameter: long-axis view during the maximal diameter of LVOT in midsystole]). The reference level was set immediately below the insertion of the cusps (“subannularly”). In the hybrid operating room, immediately before TAVI, the diameter of the annulus was measured first by our TAVI anesthesiologist(s) with expertise in TEE and then by the surgeon(s) and the cardiologist designated for the particular procedure, while the numeric values were hidden (“blinded fashion”). Next, the results were revealed and one value was defined as the “reference value.” In general, the TTE result was used only as an orientational value for screening if the annulus was too large for the use of the contemporaneous transcatheter valves.

In particular, the following measurements were performed (TEE and/or MSCT): diameters of the left ventricular outflow tract (LVOT), annulus, and aorta (at the levels of the sinuses of Valsalva, sino-tubular junction and mid ascending aorta), distances between the coronary artery ostia and the aortic annulus, distances between the commissures themselves, and distances between each commissure and the aortic wall of the opposite sinus of Valsalva. The measurements were performed in conjunction with the evaluation of the morphology of the heart and aorta, determination of the projection of the left ventricular (LV) apex to the anterior chest wall, and identification of the shapes of the annulus (“round” versus “oval”) and proximal aorta (whether the sinuses of Valsalva are pronounced or not in comparison with the mid portion of the proximal aorta; “male-shaped” versus “female-shaped” [Figure 1]). Specific pathologies influencing the valve size selection, the procedure, and valve positioning, such as localized calcified masses, open or fused commissures, and amount of calcification in the neighborhood of the commissures, were identified. The number of open or fused commissures was counted (n = 0–3) and the amount of calcification in the device landing zone was assessed semiquantitatively by visual estimation (grade 0 to ++/+).

Selection of the Valve Size
Principally, the size of the valve used was determined according to the diameter of the native aortic valve annulus measured by intraoperative TEE (“reference value”). In general, a 2-mm oversized valve was used; the 23-mm Edwards-Sapien prostheses were applied for the aortic annuli with a diameter of <21 mm and the 26-mm Edwards-Sapien prostheses for annuli of 21 to 24 mm. For the annuli >24 mm and ≤27 mm, we used a 29-mm Edwards-Sapien XT valve (balloon expandable). For the larger annuli (>27 mm and ≤29 mm), we applied a 31-mm CoreValve (transfemoral or transaxillary approach) or reconsidered conventional aortic valve replacement (with cardioplectic arrest if the aortic cross-clamp time of about 20 minutes is predicted to be possible, otherwise on a beating heart using retrograde heart perfusion through the sinus coronarius). Before a 29-mm Edwards-Sapien XT prosthesis was available, for the annuli of between 24 and 27 mm, we used self-expandable 29-mm CoreValve prostheses (transfemoral or transaxillary approach).

In borderline cases, when measurements ranged about 21 mm (24 mm, respectively), MSCT influenced the valve size selection. In this case, the decision on valve size was made on an individual basis, taking into account all additional factors such as the distances from the annulus to the coronary artery ostia, the shape of the annulus (oval versus circular), the amount of material in the leaflets, aortic diameters at the level of the sinuses of Valsalva, the sino-tubular junction and ascending aorta, and the amount of calcification in the LVOT, anterior mitral leaflet, commissures, and aortic valve leaflets themselves. In borderline cases, the factors prone to a smaller valve (and vice versa for a larger prosthesis) are narrow aortic root with nonpronounced sinuses of Valsalva (“male-shaped” aorta), round shape of the aortic valve annulus, pronounced or severe calcification of the device landing zone, fused commissures (with 0–1 open commissure), short (<8 mm) distance between the annulus and the coronary artery ostia, female sex of the patient, and body surface area <1.8 m².

Statistical Analysis
Continuous variables are presented as mean±SD and range. Categorical variables are presented as number with percentage.

Results
The group of 6 patients with rupture in the region of the device landing zone represented 1% of all patients undergoing TAVI in this time period at our institution. The transapical means of valve implantation with balloon-expandable transcatheter valves was used in all 6 patients. The patient characteristics, intraprocedural and postprocedural data, treatments, and outcomes are summarized in the Tables 1 through 4. The mean logistic EuroSCORE was 26.3±12.6% (range, 11% to 48%) and the mean STS score was 15.2±9.6% (range, 5.3% to 31.6%). The diameters of the native aortic valve annulus varied from 20.0 to 25.0 mm (mean, 22.5±1.6 mm) in TTE, 19.7 to 22.4 mm (mean, 21.0±1.1 mm; sagittal view) and 20.0 to 25.6 mm (mean, 22.9±1.9 mm; coronal view) in MSCT, and 17.5 to 22.7 mm (mean, 20.9±2.1 mm) in TEE (Table 2).

In the patients with rupture in the region of the device landing zone, all segments of the TAVI procedure were performed without any technical procedural problems. The valves were placed in the desired position, and there was no coronary artery occlusion. Balloon redilatation of the implanted prosthetic valve was performed in 2 patients with relevant paravalvular leakage.
The correct diagnosis of the device landing zone rupture was established during TAVI procedures in 4 and postmortem in 2 patients. The major sign of rupture was apparent bleeding in 4 patients and failure of myocardial recovery after valve implantation in 1; in 1 patient, it was asymptomatic. Bleeding was obvious already intraprocedurally in 3 patients, and in 1 it was delayed and identified 8 hours after an initially uneventful postoperative course (Table 3).

Device landing zone rupture was treated surgically in 5 patients and conservatively in the 1 patient without symptoms (patient 4). When the diagnosis was established correctly during TAVI, only 1 of 4 patients died (25%) (Table 4). The overall mortality rate was 50% (3 of 6 patients died) for the patients with device landing zone rupture compared with 5.4% (33/612) in patients without rupture. Brief summaries of each case are given below.

**Patient 1**
An 88-year-old female patient underwent transapical TAVI with a 26-mm Edwards-Sapien valve. The values of the aortic annulus diameter varied significantly (Table 2) as assessed by several different MSCT and TEE assessments. The presence of the measurements larger than 21 mm led us to use a larger prosthesis to prevent its dislodgment. Massive bleeding occurred at the end of the procedure. It was masked by a large amount of adipose epicardial tissue and was falsely diagnosed as bleeding from suture holes in the hypertrophied myocardium of the LV apex. It needed additional sutures to be applied toward the LV base. At the end, the bleeding was controlled with deep myocardial sutures placed from outside through the enlarged left thoracotomy used for TAVI. The patient died in the hybrid operating room because of myocardial failure. The diagnosis of a LV rupture was retrospectively established after the postprocedural analyses of the case. The possible cause of the rupture was the use of a too-large prosthesis in the presence of annular and LVOT calcifications.

**Patient 2**
An 83-year-old female patient with a small aortic annulus (Table 2) and calcification of the aortic root including the LVOT and the proximal aorta underwent transapical TAVI with a 23-mm Edwards-Sapien valve (online-only Data Supplement Video 1). Immediately after valve implantation, myocardial recovery failed despite maximal inotrope support (online-only Data Supplement Video 2). The annulus rupture included the calcified left sinus of Valsalva and the ostium of the left coronary artery (Figures 2 and 3 and online-only Data Supplement Video 3). Despite composite graft implantation, the patient died in the hybrid room as a result of LV failure. The main reason for the rupture was the small aortic annulus in the presence of the aortic root calcification and the narrow aorta (online-only Data Supplement Video 1). Hypothetically, the use of a prosthesis smaller than 23 mm (not yet

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Table 1. Baseline Characteristics of the Patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>BMI, kg/m²</th>
<th>BSA, m²</th>
<th>NYHA Class</th>
<th>EuroSCORE, %</th>
<th>STS Score, %</th>
<th>FEV1 (l)</th>
<th>Creatinine, mg/dL</th>
<th>Hb, mg/dL</th>
<th>PAD</th>
<th>CAD</th>
</tr>
</thead>
<tbody>
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<td>1</td>
<td>88</td>
<td>Female</td>
<td>30</td>
<td>1.8</td>
<td>4</td>
<td>Yes</td>
<td>48.0</td>
<td>31.6</td>
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<td>1.9</td>
<td>11.7</td>
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<tr>
<td>2</td>
<td>83</td>
<td>Female</td>
<td>20</td>
<td>1.3</td>
<td>2</td>
<td>No</td>
<td>30.4</td>
<td>14.7</td>
<td>1.06</td>
<td>1.1</td>
<td>12.2</td>
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<tr>
<td>3</td>
<td>84</td>
<td>Female</td>
<td>25</td>
<td>1.6</td>
<td>4</td>
<td>No</td>
<td>24.4</td>
<td>17.8</td>
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<td>10.8</td>
<td>Yes</td>
</tr>
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<td>4</td>
<td>85</td>
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<td>26</td>
<td>1.8</td>
<td>4</td>
<td>No</td>
<td>11.0</td>
<td>16.0</td>
<td>1.29</td>
<td>1.3</td>
<td>13.1</td>
<td>No</td>
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<tr>
<td>5</td>
<td>89</td>
<td>Male</td>
<td>22</td>
<td>1.5</td>
<td>3</td>
<td>No</td>
<td>17.9</td>
<td>5.8</td>
<td>1.75</td>
<td>0.8</td>
<td>12.1</td>
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<tr>
<td>6</td>
<td>89</td>
<td>Female</td>
<td>27</td>
<td>1.7</td>
<td>3</td>
<td>No</td>
<td>26.2</td>
<td>5.3</td>
<td>N/A</td>
<td>0.8</td>
<td>12.0</td>
<td>No</td>
</tr>
<tr>
<td>All</td>
<td>86.3±2.7</td>
<td>...</td>
<td>25.0±3.6</td>
<td>1.6±0.2</td>
<td>3.3±0.8</td>
<td>1(17)</td>
<td>26.3±12.6</td>
<td>15.2±9.6</td>
<td>1.4±0.3</td>
<td>1.1±0.4</td>
<td>12.0±0.7</td>
<td>4(67)</td>
</tr>
</tbody>
</table>

BMI indicates body mass index; BSA, body surface area; NYHA, New York Heart Association; CS, cardiogenic shock; STS, Society of Thoracic Surgeons; FEV1, forced expiratory volume in 1 second; Hb, hemoglobin; PAD, peripheral arterial disease; CAD, coronary artery disease; and N/A, not available.

Values are mean ± SD or n (%).
available for clinical use) would possibly have prevented this complication. We believe that we lost too much time before the correct diagnosis of the problem was made, during which the coronary perfusion of a hypertrophic heart was not secured and too much time passed until cardioplegia was given.

Patient 3

An 84-year-old female patient underwent technically uneventful transapical TAVI with a 26-mm Edwards-Sapien valve (Table 2). Just before closure of the thoracotomy wound, slight but continuous arterial bleeding appeared. Initially, the cause of bleeding could not be identified clinically. The patient remained hemodynamically stable.

Table 2. Preprocedural Data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
<th>Patient 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF, %</td>
<td>38</td>
<td>50</td>
<td>50</td>
<td>65</td>
<td>55</td>
<td>50</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>60</td>
<td>46</td>
<td>50</td>
<td>35</td>
<td>42</td>
<td>45</td>
</tr>
<tr>
<td>dPmean, mm Hg</td>
<td>69</td>
<td>71</td>
<td>84</td>
<td>50</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>AVA, cm²</td>
<td>0.8</td>
<td>0.4</td>
<td>0.8</td>
<td>0.5</td>
<td>0.6</td>
<td>0.8</td>
</tr>
<tr>
<td>LVOT by TEE, mm</td>
<td>18.4</td>
<td>14.6</td>
<td>18.8</td>
<td>17.7</td>
<td>19.9</td>
<td>22.5</td>
</tr>
<tr>
<td>Annulus diameter, mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TTE</td>
<td>22.0</td>
<td>20.0</td>
<td>25.0</td>
<td>23.0</td>
<td>22.0</td>
<td>23.0</td>
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<tr>
<td>CT sagittal</td>
<td>22.4</td>
<td>19.7</td>
<td>21.9</td>
<td>20.4</td>
<td>20.0</td>
<td>21.5</td>
</tr>
<tr>
<td>CT coronal</td>
<td>23.6</td>
<td>20.0</td>
<td>23.6</td>
<td>22.0</td>
<td>22.6</td>
<td>25.6</td>
</tr>
<tr>
<td>TEE</td>
<td>20.6</td>
<td>17.5</td>
<td>22.5</td>
<td>19.5</td>
<td>22.4</td>
<td>22.7</td>
</tr>
<tr>
<td>Sinuses of Valsalva,* mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>CT</td>
<td>35.3</td>
<td>24.2</td>
<td>30.9</td>
<td>28.0</td>
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<td>29.6</td>
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<tr>
<td>TEE</td>
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<td>27.6</td>
<td>29.2</td>
<td>29.9</td>
<td>27.5</td>
</tr>
<tr>
<td>“Male-shaped” aorta†</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CT</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
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</tr>
<tr>
<td>TEE</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Fused commissures,‡ n</td>
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<td>2</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>TEE</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Calcification,† grade 0 to +++</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Device landing zone by CT</td>
<td>++</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>LVOT by CT</td>
<td>+</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>LVOT by TEE</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>AML by CT</td>
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<td>0</td>
<td>++</td>
<td>0</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>AML by TEE</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>0</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Annulus by CT</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Annulus by TEE</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Edge of the cusps by CT (n)</td>
<td>++ (3)</td>
<td>+++ + (2)†</td>
<td>++ (3)</td>
<td>+++ (3)</td>
<td>+++ (3)</td>
<td>+++ (3)</td>
</tr>
<tr>
<td>Edge of the cusps by TEE (n)</td>
<td>++ (3)</td>
<td>+++ + (2)†</td>
<td>++ (3)</td>
<td>+++ (3)</td>
<td>+++ (3)</td>
<td>+++ (3)</td>
</tr>
<tr>
<td>Inside the cusps by CT (n)</td>
<td>++ (3)</td>
<td>++ + (2)‡</td>
<td>++ (3)</td>
<td>+ (3)</td>
<td>+++ (3)</td>
<td>+++ (3)</td>
</tr>
<tr>
<td>Inside the cusps by TEE (n)</td>
<td>+ (3)</td>
<td>+ (2)‡</td>
<td>+ (3)</td>
<td>+ (3)</td>
<td>+ (3)</td>
<td>+ (3)</td>
</tr>
<tr>
<td>Proximal aortic wall by CT</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Proximal aortic wall by TEE</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

LVEF indicates left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; dPmean, mean transvalvular gradient; AVA, aortic valve area; LVOT, left ventricular outflow tract; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; CT, multislice computed tomography; and AML, anterior mitral leaflet.

*Aortic diameter.
†Visual assessment.
‡Both left and noncoronary cusps.

After a median sternotomy, slight but obvious arterial bleeding was identified from an adventitial hematoma of the proximal aorta in the region of the right coronary artery (Figure 4 and online-only Data Supplement Video 4). The patient underwent an uneventful conventional aortic valve replacement with a biological prosthesis using standard cannulation for cardiopulmonary bypass (CPB). A longitudinal lesion of the aortic wall in the right coronary sinus of Valsalva coming from the ruptured calcified native aortic annulus was repaired with several pledgeted sutures. The postoperative course was characterized by prolonged respiratory weaning, but finally the patient recovered well. Severe calcification was believed to be a highly relevant factor for the rupture. It remained unclear—and questionable—whether
implantation of a smaller valve (23 mm) in the presence of a severely calcified and narrow aortic root could have prevented this complication.

**Patient 4**

An 85-year-old female patient with an aortic annulus of 19.5 to 22 mm (Table 2) and calcification of the LVOT and aortic annulus underwent technically uneventful transapical TAVI with a 23-mm Edwards-Sapien valve. Completion angiography showed an optimal position of the valve but a finding of an unusually contrasted area in the region of the native aortic annulus (Figure 5 and online-only Data Supplement Video 5). The patient remained hemodynamically stable and was treated conservatively. She underwent close postoperative monitoring. MSCT performed 3 months later showed no pathological findings. Hypothetically, the use of a prosthesis smaller than 23 mm (presently not available for clinical use) would possibly have prevented this complication. However, the mechanism suspected for this patient is only speculative because the smallest measurement of 19.5 mm fits into the recommended size for the use of the 23-mm prosthesis.

**Patient 5**

Uneventful transapical TAVI with a 26-mm Edwards-Sapien prosthesis was performed in an 89-year-old male patient with severe annulus and LVOT calcification. The diameter of the native aortic annulus ranged 20 to 23 mm (Table 2). Additional reballooning of the valve was performed because of relevant paravalvular leakage. The initial postoperative course was uneventful. Suddenly, 8 hours after the procedure, the patient became hemodynamically unstable and massive bleeding through the left pleural drain occurred. The patient was immediately transferred to an operating room, and revision was performed by a surgeon on duty. The bleeding was described as bleeding from the coronary artery from the left lateral wall of the left ventricle. It was controlled by sutures but the patient died intraoperatively due to myocardial failure. In this patient, LV rupture mimicked the misleading diagnosis of injury of a coronary artery. The correct diagnosis was established retrospectively. The most likely explanation for the rupture is the importance of extensive calcification.

**Patient 6**

An 89-year-old female patient with an aortic annulus with an enhanced oval shape (Table 2) and calcification of the LVOT underwent uneventful transapical TAVI with a 26-mm Edwards-Sapien valve. Initially, the valve was not completely deployed because of the calcification seen in the LVOT. Additional complete inflation of the implanted valve was performed because of relevant paravalvular leakage. During closure of the chest, a small amount of arterial blood appeared in the wound. Inspection of the already-sutured apex showed no bleeding source. Repeated angiography showed rupture of the native aortic valve annulus in the region below the left coronary sinus of Valsalva with contrast propagation toward

---

**Table 3.** Data Regarding Rupture in the Region of the Device Landing Zone

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Time of Diagnosis</th>
<th>Location</th>
<th>Main Anatomic Pathology*</th>
<th>Reballooning During TAVI</th>
<th>Main Symptom</th>
<th>Intraoperative Diagnosis</th>
<th>Timely Treatment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Postmortem</td>
<td>Left ventricle</td>
<td>Annulus calcification</td>
<td>No</td>
<td>Bleeding</td>
<td>Incorrect</td>
<td>No</td>
<td>Surgical</td>
</tr>
<tr>
<td>2</td>
<td>During TAVI</td>
<td>Left coronary sinus, LM</td>
<td>Small annulus, root, and LVOT calcification</td>
<td>No</td>
<td>Myocardial failure</td>
<td>Correct</td>
<td>No</td>
<td>Surgical</td>
</tr>
<tr>
<td>3</td>
<td>During TAVI</td>
<td>Right coronary sinus</td>
<td>Annulus calcification</td>
<td>No</td>
<td>Bleeding</td>
<td>Correct</td>
<td>Yes</td>
<td>Surgical</td>
</tr>
<tr>
<td>4</td>
<td>During TAVI</td>
<td>Native annulus</td>
<td>Annulus and LVOT calcification</td>
<td>No</td>
<td>Asymptomatic</td>
<td>Correct</td>
<td>Yes</td>
<td>Conservative</td>
</tr>
<tr>
<td>5</td>
<td>Postmortem</td>
<td>Left ventricle</td>
<td>Root, annulus, and LVOT calcification</td>
<td>Yes</td>
<td>Bleeding</td>
<td>Incorrect</td>
<td>No</td>
<td>Surgical</td>
</tr>
<tr>
<td>6</td>
<td>During TAVI</td>
<td>Left ventricle</td>
<td>LVOT calcification</td>
<td>Yes</td>
<td>Bleeding</td>
<td>Correct</td>
<td>Yes</td>
<td>Surgical</td>
</tr>
</tbody>
</table>

TAVI indicates transcatheter aortic valve implantation; LM, main trunk of the left coronary artery; and LVOT, left ventricular outflow tract.

*Clinical judgment.

---

**Table 4.** Types of Treatment and Outcome

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Prosthesis Size, mm</th>
<th>Type of Additional Surgery</th>
<th>Packed RBC Units</th>
<th>Complications</th>
<th>Outcome</th>
<th>ICU Stay, Days</th>
<th>In-Hospital Stay, Days</th>
<th>Follow-Up Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>Myocardial U-stitches</td>
<td>9</td>
<td>Myocardial failure, death</td>
<td>Death</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>Composite graft</td>
<td>16</td>
<td>Myocardial failure, death</td>
<td>Death</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>Repair+aortic valve replacement</td>
<td>11</td>
<td>Prolonged respiratory support (tracheostomy)</td>
<td>Alive</td>
<td>9</td>
<td>15</td>
<td>369</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>Conservative treatment</td>
<td>0</td>
<td>Pacemaker implantation</td>
<td>Alive</td>
<td>2</td>
<td>8</td>
<td>145</td>
</tr>
<tr>
<td>5</td>
<td>26</td>
<td>Myocardial U-stitches</td>
<td>5</td>
<td>Myocardial failure, death</td>
<td>Death</td>
<td>1</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>6</td>
<td>26</td>
<td>Repair+aortic valve replacement</td>
<td>4</td>
<td>No</td>
<td>Alive</td>
<td>1</td>
<td>9</td>
<td>121</td>
</tr>
</tbody>
</table>

RBC indicates red blood cells; ICU, intensive care unit.
the left coronary artery (Figure 6 and online-only Data Supplement Video 6). The intensity of bleeding increased steadily, with the blood coming from the depth of the pericardium, and the patient became hemodynamically unstable. During medicamentous reanimation, an emergency sternotomy was performed, standard CPB was instituted, and an LV vent was placed through the right upper pulmonary vein. There was massive pulsatile arterial bleeding seen in the mid part of the LV lateral wall near a coronary artery and a diffuse hematoma located on the LV base near the left appendage. Interestingly, the first impression was very confusing because the bleeding imitated a lesion and bleeding from the coronary artery. (Possibly, the artery could be damaged at the end of the TAVI procedure by a forceps during the last check of the operating field before closure of the chest.) This was excluded by simple tests by loading and unloading the LV while the patient was on CPB. When the LV was unloaded by suction of the blood from the LV with the vent, the bleeding stopped. Contrary to this, when the vent suction was stopped and the volume was given into the heart by the perfusionist, the bleeding appeared again. (Otherwise, if the coronary artery was damaged, the bleeding should continue because the aorta was not cross-clamped.) The ascending aorta was then cross-clamped and blood cardioplegia was given. Again, the bleeding stopped when the heart was unloaded by vent suction. (If the coronary artery would have been damaged, the bleeding should have continued because of the continuous perfusion of the coronaries with blood cardioplegia.) This established the correct diagnosis of left ventricular wall rupture. After aortotomy and removal of the Edwards-Sapien

Figure 2. Transapical valve implantation in patient 2 with a small aortic annulus and calcified aortic root. Stopping the balloon inflation and incomplete dilation of the Edwards-Sapien prosthesis at this stage (A) would have prevented the annulus rupture shown in D. Note overdistension of the aorta (yellow arrows) by complete inflation of the balloon during valve deployment (B). Angiography was performed immediately after valve deployment to find the cause of failed myocardial recovery after valve deployment; contrast instillation through a pigtail catheter above the new valve showed no coronary artery occlusion and unusual tilting of the prosthesis below the ostium of the left coronary artery but no extravasation of the contrast is seen at this stage. A superstiff guide wire is still in place (C). An attempt at selective angiographic visualization of the left coronary artery (LCA) showed rupture of the left coronary sinus of Valsalva including the ostium of the LCA and extraluminal contrast extravasation (red arrows) in the region between the proximal aorta, LCA, and the trunk of the pulmonary artery (D). Note the venous cannula for emergency cardiopulmonary bypass. Ao indicates ascending aorta; LVOT, left ventricular outflow tract; and RCA, right coronary artery.

Figure 3. Intraprocedural transesophageal echocardiography (A, aortic valve short-axis view; B, aortic valve long-axis view) showing para-aortic hematoma (red circle) after implantation of an Edwards-Sapien valve in patient 2. The hematoma is located in the region of the proximal left coronary artery and between the left posterior side of the proximal aorta and the left atrium (LA) and the pulmonary artery (PA). This finding correlated to the contrast extravasation seen in Figure 1D. Ao indicates ascending aorta; LVOT, left ventricular outflow tract; r PA, right pulmonary artery; and RV, right ventricle.
valve, a 2.5x2.5x2.0 cm rupture in the LVOT myocardium was seen just below the left coronary annulus and lateral to the anterior mitral leaflet. It was caused by impression of a calcification from the LVOT into the myocardium. The defect in the LVOT was repaired from inside with a 3x3-cm autologous pericardial patch, and pericardially pledgeted sutures and conventional aortic valve replacement was performed. No attempt was undertaken to suture the myocardial tear from outside. The further operative and postoperative course was without problems. The most likely explanation for the rupture is the subsequent secondary balloon dilatation in the presence of calcification. Possibly, implantation of a smaller valve (23 mm) could have prevented this complication.

**Discussion**

This case series presents a collection of different manifestations of rupture in the region of the device landing zone during TAVI. Our limited experience has clearly shown that this type of procedure-related, life-threatening complications can be treated successfully if it is immediately recognized and adequately managed.

The principal problem was identical in all patients, namely, the discrepancy between the aortic annulus and the stent-valve size in the presence of additional possible predisposing factors, especially in patients with significant calcification. We observed this complication rarely and exclusively after the use of a balloon-expandable transcatheter valve but not after TAVI with self-expandable valves. However, our observation does not exclude it in the latter group, as rupture in the region of the device landing zone has already been reported after TAVI with a self-expandable valve as a consequence of overdilatation of the prosthesis to treat residual paravalvular regurgitation. Precise identification of the additional predisposing factors in our small group of patients is not possible. It might be that a combination of multiple anatomic and procedural factors rather than a single one is responsible for this complication. Most of our reported patients had a narrow aortic root (in terms of the diameter of the proximal aorta at the level of the sinuses of Valsalva) and/or calcified LVOT below the left coronary cusp, especially below the left part of the left coronary cusp, huge...
amount of calcified material in the leaflets, a narrow or calcified aortic root (proximal aorta), and asymmetrical septum hypertrophy. It might be that an enhanced oval shape in the presence of calcification may additionally increase the risk of this complication. Overdilatation of the prosthesis seems to be a procedural factor regardless of the type of the prosthesis used (self-expandable6 versus balloon-expandable).

There are several important aspects of possible conjunction between annular rupture, extensive calcification of the device landing zone, and the repeated balloon dilatation. Implantation of a balloon-expandable valve needs first balloon dilatation of the stenotic native aortic valve and second balloon dilatation for valve deployment. Additionally, if there is relevant paravalvular leakage after deployment of the new valve (a known important negative factor for long-term survival), it is treated by additional ballooning of the implanted valve. The native aortic valve annulus is frequently calcified in the presence of severe aortic valve stenosis. Therefore, it can be speculated that small tears in the native annulus occur during TAVI, probably more frequently than is diagnosed. It can be expected during deployment of a balloon-expandable valve rather than during balloon dilatation of the native valve. The reasons for this are, first, that a smaller balloon is used for dilatation than for valve deployment and, second, mechanical presence of the valve stent may cause additional damage of the tissue. However, it does not produce complications because the implanted new valve fits into the annulus, closes the tear and “seals” it. The clinically identified cases of “annulus rupture” should have some additional pathological changes (eg, calcification of the adjacent anatomical structures) causing simultaneous damage in the device landing zone (consisting of the aortic annulus, valvular cusps, and LVOT). Therefore, redilatation of the new valve (both balloon-expandable and self-expandable valves) because of relevant paravalvular leakage may even additionally increase this risk of rupture. The leakage frequently occurs in the presence of extensive calcification and therefore an additional balloon dilatation used to treat relevant paravalvular leakage after implantation of the prosthesis may lead to annular rupture under these circumstances.

Our valve size selection during TAVI is primarily based on TEE measurements. We have been using it for years during conventional aortic valve replacement. The results could have been checked immediately after excision of the native valve (they were mostly underestimated for 1–2 mm). As reported previously, we observed in roughly one-third of patients some difficulties regarding the determination of the valve size, mostly because of borderline values of 21 mm by TEE. In these cases, the decision was made on an individual basis, taking into account additional factors determined by TEE and MSCT. Our impression is that we generally tended to use “larger rather than a smaller valve.” In combination with a modified implantation technique, it resulted in a very low incidence of relevant paravalvular leakage. It might correlate with the recent reports that showed that the “real values” are more accurately assessed by MSCT and are underestimated by TEE due to an oval shape of the aortic annulus. The MSCT coronal view usually provides the largest annulus diameter, and the MSCT sagittal reconstructed views are usually 1 to 2 mm smaller than coronal and correlate well with TEE. Therefore, MSCT, including a 3-dimensional assessment of the aortic root, provides additional valuable information and may additionally help in valve size selection in these borderline cases. Another useful method to assess the optimal size of the new valve in this situation may be simultaneous aortography while inflating a 23-mm balloon across the valve.

In accordance with the reported experience, we have modified our clinical practice. It includes the policy that any arterial bleeding with no identifiable cause should be considered as suspected annulus rupture, and therefore immediate institution of CPB and a median sternotomy should be performed to treat it, even in patients who are considered formally “inoperable” or “not suitable for conventional surgery.” Standard aortic valve replacement should be combined with repair of the additional lesion, for example, reconstruction of the damaged aortic wall. The optimal treatment for LVOT and myocardial rupture is reconstruction of the LVOT with an oversized pericardial patch. No attempts should be made to close the rupture of the left ventricle by using U-stitches from outside because bleeding stops when the LVOT is reconstructed. As known from mitral valve surgery, the danger of damaging the coronary arteries by myocardial sutures from the outside is very high with consequent myocardial infarction and unsuccessful weaning from CPB.

If the intraoperative findings are assessed as only minimal and there are no clinical signs (ie, bleeding), these patients can be treated conservatively under close surveillance with repeated CT checks. Importantly, postoperative TTE may underestimate the finding seen on TEE or it can even be barely detectable on TTE examination. The impact on survival of detected but asymptomatic rupture in the region of the device landing zone remains to be defined. Thus, all TAVI patients should be kept under close surveillance during the follow-up.

Possible prevention of this complication lies in an adequate balance between the oversizing of the new valve (to prevent...
or minimize paravalvular leakage) and the diameter of the native aortic annulus. Precise determination of the native aortic valve annulus and correct interpretation of the preprocedural findings with meticulous identification of possible factors for annulus rupture are mandatory. It should be emphasized that the discrepancies between the different measurements of the annulus provided by TTE, TEE, and CT—as found in several of our reported patients—should be considered an important indicator for possible annular rupture. Therefore, this discrepancy should be clarified before a definitive decision about valve size determination is made. Importantly, unusual findings when MSCT measurements are smaller than TEE measurements might indicate a screening failure in term of annulus assessment. Therefore, more accurate methods of the assessment of the diameter of the native annulus and that for valve size selection are necessary. They must eliminate the possible screening failure in terms of annulus assessment because it might be an important cause of annulus rupture. Additionally, a broader valve size spectrum of the transcatheter valves, including a smaller valve than presently available, in combination with precise determination of the annulus, should reduce the incidence of rupture.

Conclusions
Cardiac surgeons and interventional cardiologists involved in TAVI programs should be aware that the occurrence of rupture in the region of the device landing zone, a potentially fatal but treatable complication, may be underestimated. The devastating nature and severity of this complication presents an important limitation of this less-invasive aortic valve treatment. It is crucial to have the possibility to perform immediate conversion to conventional surgery when necessary. If there is a need for surgical revision, it should be performed by a surgeon with experience in TAVI. The precise, simple, and reproducible criteria of patient selection, annulus sizing, and determination of the proper size of a prosthetic valve should be established to minimize the risk of this possibly catastrophic complication and to optimize the overall results of TAVI.

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Disclosures
Drs Pasic, Unbehaun, Drews, Buz, and Dreyssse have been proctors to Edwards Lifesciences since July 2009.

References
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SUPPLEMENTAL MATERIAL

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LEGENDS FOR THE VIDEO FILES

Supplemental Video 1. Overdilation of the balloon during valve deployment in patient #2 with a small annulus and a narrow aortic root. Notice, during the last part of the release, sudden overdistension of the ascending aorta wall and overexpansion of the prosthetic stent in the region of the sinotubular junction (right margin of the image).

Supplemental Video 2. Transesophageal echocardiography (mid-papillary short axis) several minutes after valve deployment in patient #2 with lack of recovery showing severe hypokinesia of the hypertrophic myocardium in the region of the left coronary artery (anteroseptal, anterior, lateral and posterior left ventricular segments) but existing contractility of the septal and inferior segment (supplied by the right coronary artery).

Supplemental Video 3. An attempt to find the cause of failed myocardial recovery after valve deployment by selective angiographic visualization of the left coronary artery showed rupture of the left coronary sinus and massive extravasation of the contrast medium from the aorta towards the pulmonary artery (patient #2). The ostium and the main trunk of the left coronary artery are disrupted. The tip of the venous cannula for cardiopulmonary bypass placed through the femoral vein is in the right atrium. Note that there is still partial perfusion of the left main stem and LAD and the aortic prosthesis appears to be continent.

Supplemental Video 4. Regular angiography after valve deployment in patient #3 showing a flaw and irregular periaortal extravasation of the contrast after annulus rupture und lesion of the calcified right coronary sinus of Valsalva.

Supplemental Video 5. Completion angiography after valve deployment in patient #4 showing an optimal position of the new transcatheter valve and a finding of an unusual small
amount of contrast in the region of the aortoventricular connection below the left coronary artery.

**Supplemental Video 6.** Angiography in patient #6 showing rupture of the native aortic valve annulus in the region of the left coronary sinus of Valsalva with contrast propagation towards the left coronary artery. An additional very faint pulsatile contrast flow from the contrast filled cavity in the myocardium can be seen towards the pericardium, representing free myocardial rupture.