An 80-year-old woman was admitted to our institution because of shortness of breath (New York Heart Association grade III) and stable angina pectoris (Canadian Cardiovascular Society [CCS] grade II).

Two years ago, the patient had a posterior wall infarction successfully treated by percutaneous coronary intervention and stent implantation. A low-gradient aortic stenosis with an aortic valve area of 1.1 cm² was also diagnosed but was considered to be clinically insignificant at the time.

Because of the progression of symptoms within the last 2 years, a re-evaluation of coronary and aortic valve disease was performed. Coronary angiography revealed a 1-vessel coronary disease with a patent stent in the right coronary artery without any evidence of restenosis. The left ventricular ejection fraction was only moderately impaired (55%). However, the aortic valve area, as determined invasively, had decreased to 0.5 cm². This was confirmed by echocardiography; the cusps of the aortic valve were calcified and their motion was impaired (Supplemental Data Movie 1).

On the basis of these findings, the necessity of an aortic valve replacement was discussed. However, because the patient was anxious and refused conventional aortic valve replacement, percutaneous treatment of the aortic stenosis using the second generation of the CoreValve ReValving system (CoreValve Inc, Irvine, Calif) was proposed¹ (Figure 1A). This prosthesis has an inner diameter of 23 mm and consists of a self-expanding nitinol frame, which carries 3 leaflets of porcine pericardial tissue. The upper part of the frame anchors the system in the aorta; the middle part of the prosthesis is constrained to avoid interference with the coronaries and carries the valve. The lower part of the stent exerts high radial forces (which pushed aside the calcified leaflets), avoids recoil, and is covered with pericardium to minimize paravalvular leaks.¹

Under general anesthesia and extracorporeal circulatory support, a valvuloplasty was performed, followed by successful CoreValve implantation via a transfemoral access (Supplemental Data Movies 2 and 3). The patient was extubated the same day, recovered quickly after the procedure, and left the hospital 10 days later. At 1-year follow-up, the mean gradient across the valve was 3 mm Hg, the aortic valve area was 1.7 cm², and there was no evidence of paravalvular leak (Supplemental Data Movie 4). These changes in hemodynamics were accompanied by an improvement in symptoms in this patient.

Despite the increasing use of percutaneous aortic valve replacement, little is known about ingrowths of prostheses and durability in humans.¹⁻³ The autopsy of our patient, who unfortunately committed suicide 425 days after implantation, might give some insight with regard to the CoreValve prosthesis.

The calcified native valve was pushed aside by the CoreValve frame, and neither the native cusps nor the implanted bioprosthesis had any interference with the coronaries (Figure 2A). The bioprosthetic valve did not show any signs of morphological deterioration at autopsy (Figures 1B and 2B). The lower part of the stent, which was covered by porcine pericardium (Figure 1A) and positioned in the left ventricular outflow tract, was completely enclosed by fibromyoblasts (Figures 1B, 3A, and 3B). The ingrowths of the stent in the left ventricular outflow tract might explain why paravalvular leaks that are evident immediately after implantation disappear over time.¹ Conversely, in the ascending aorta, only those struts of the frame that had close contact to the aortic wall were covered by endothelium (Figures 1B and 2A). All other parts of the frame, in particular the struts at the top, which extended into the aorta, had a surface without discoloration (Figures 1B and 2A).

Despite the high flow velocity in the aorta, these nonendothelialized and noncovered parts of the nitinol frame might be a place of origin for thrombi formation and thromboembolic events. Therefore, platelet inhibition for >1 year after CoreValve implantation might be considered.
Disclosures
Dr Linke received speaker honoraria from CoreValve Inc, is a proctor and consultant for CoreValve Inc, and was a subinvestigator in a clinical study supported by a restricted grant from CoreValve Inc. Dr Schuler was a subinvestigator in a clinical study supported by a restricted grant from CoreValve Inc.

References
Figure 2. A, Association between coronary arteries and the prosthesis. A probe is positioned in the left main stem and the right coronary artery. There is no interference between the cusps of the native aortic valve or the bioprosthesis and the ostium of the coronaries. B, Cor-eValve prosthesis in situ (top view). The bioprosthesis does not show any signs of morphological deterioration. The nitinol frame is well adapted to the annulus and the ascending aorta.
Figure 3. A, View from the apex into the left ventricular outflow tract. The lower part of the bioprosthesis is completely grown in. B, Histological evaluation (hematoxylin/eosin staining) of a tissue section cut out between the stent struts at the left ventricular outflow tract (corresponding to the rectangle in Figure 3A). Ingrowths of the prosthesis into the left ventricular outflow tract are shown. The porcine pericardium (a) covering the lower part of the prosthesis is surrounded by some inflammatory cells (primarily lymphocytes, b) that are always found in the direct vicinity of an artificial valve. They are covered by fibroblast (c) and a layer of endocardium (d) at the luminal side of the prosthesis (e, left ventricular outflow tract).
Ingrowths of a Percutaneously Implanted Aortic Valve Prosthesis (CoreValve) in a Patient With Severe Aortic Stenosis

Axel Linke, Robert Höllriegel, Thomas Walther, Katrin Schierle, Christian Wittekind, Jörg Ender, Friedrich W. Mohr and Gerhard Schuler

doi: 10.1161/CIRCINTERVENTIONS.108.796094
Circulation: Cardiovascular Interventions is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-7640. Online ISSN: 1941-7632
The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circinterventions.ahajournals.org/content/1/2/155
Data Supplement (unedited) at:
http://circinterventions.ahajournals.org/content/suppl/2008/10/14/1.2.155.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Interventions can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Interventions is online at:
http://circinterventions.ahajournals.org//subscriptions/