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EDITOR'S HIGHLIGHT

TECHNICAL CORNER



First-in-Man 4-Chamber Pressure-Volume Analysis During Transcatheter Aortic Valve Replacement for Bicuspid Aortic Valve Disease

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ABSTRACT

This report constitutes a first-in-man description of pressure-volume analysis in all 4 cardiac chambers before and after transcatheter aortic valve replacement. Pressure-volume analysis demonstrated that the hemodynamic consequences of valve replacement are chamber-specific and influenced by all aspects of the procedure (i.e., rapid ventricular pacing), not just valve deployment. (**Level of Difficulty: Advanced**.) (J Am Coll Cardiol Case Rep 2021;3:77-81) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

he hemodynamics of bicuspid aortic valve disease and how they change in response to transcatheter aortic valve replacement (TAVR) are not well characterized. Pressure-volume (PV) analysis is the gold-standard technique for myocardial performance quantification. Herein, we present the first-ever report of PV analysis in all 4

LEARNING OBJECTIVES

- To review the technique for performing and interpreting pressure-volume analysis in all 4 cardiac chambers.
- To appreciate the hemodynamics consequences of transcatheter aortic valve replacement in the left ventricle as well as the other cardiac chambers.

chambers of the human heart immediately before and after TAVR.

HISTORY OF PRESENTATION

The measurements were performed in a 71-year-old man with severe bicuspid valve aortic stenosis (AS) and New York Heart Association functional class III symptoms as well as multiple comorbidities that precluded surgical valve replacement. Pre-procedural echocardiography confirmed a calcified bicuspid aortic valve (Sievers type 1) with a peak velocity of 4.6 m/s, mean gradient of 49 mm Hg, and aortic valve area of 0.8 cm². The patient provided informed written consent to participate in the study and the local institutional review board approved the study protocol.

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institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

ABBREVIATIONS AND ACRONYMS

- AS = aortic stenosis
- LA = left atrial/atrium
- LV = left ventricle/ventricular
- PV = pressure-volume
- **RA** = right atrial/atrium
- RV = right ventricle/ventricular

RVP = rapid ventricular pacing

TAVR = transcatheter aortic

valve replacement

INVESTIGATIONS

PV loops were measured with a 7-F conductance catheter (CD Leycom, Hengelo, the Netherlands) that was maneuvered into the desired cardiac chamber with an Agilis steerable introducer sheath (Abbott Cardiovascular, Chicago, Illinois). This novel technique overcame previous challenges guiding the stiff conductance catheter and minimized artifact associated with suboptimal catheter positioning (Figure 1). Measurements were

calibrated with hemodynamics from Swan-Ganz catheterization and chamber volumes derived from 3-dimensional echocardiography performed at the time of TAVR. During valve deployment, the conductance catheter was positioned in the left ventricular (LV) apex via an antegrade transseptal approach, generating the first-ever LV PV loops during valve deployment. A 29-mm S3 valve (Edwards Lifesciences, Irvine, California) was ultimately deployed after a 26-mm valve resulted in significant aortic regurgitation.

Baseline PV loops are highlighted in Figure 2. LV stroke work is predictably high. Although the LV is able to generate substantial systolic pressure (>160 mm Hg), the lower-than-expected ratio of endsystolic elastance/effective arterial elastance (normal is approximately 1) is due to the increased effective ventricular afterload secondary to the stenotic valve. The right ventricular (RV) PV loops appear similar in shape to the LV loops because of elevated pulmonary pressures, contrasting with the typical dome-shaped RV PV loops seen with normal pulmonary pressures.

Little is known about right atrial (RA) or left atrial (LA) PV loops in human health or disease, and the interpretation of atrial loops is complex due to the absence of inflow valves that regulate chamber volume. Evaluation of their passive (conduit) and active (contractile) contributions to ventricular filling from PV loops is a matter of active research. Nevertheless, quantification of their intrinsic systolic and diastolic properties based on PV analysis is well established (1,2). In this case, these loops show that although atrial contractility does not change significantly pre- versus post-TAVR, the RA is unloaded (reduced systolic and diastolic pressures), whereas the LA was slightly more loaded (higher pressures), perhaps secondary to the reduction of LV contractility.

Rapid ventricular pacing (RVP) during valve deployment (Figure 2) induces a significant decline in LV contractility and pressure (see the gray PV loops and pressure-time tracing in the middle panel), and LV size and function do not return to baseline after RVP termination. In fact, the LV dilates considerably (end-diastolic volume increases by approximately 75 ml) and operates on an entirely new end-systolic PV relationship with reduced slope.

LV function improved by the time PV loops were repeated at the end of the procedure (Figure 2), but the slope of the end-systolic PV relationship suggests only a partial recovery. These loops also illustrate that ventricular afterload is multifactorial. Although TAVR relieved the mechanical obstruction caused by AS, other factors (i.e., reflex-mediated changes in heart rate and systemic vascular resistance) may still contribute to effective arterial elastance, or afterload, which did not decline immediately post-procedure.

DISCUSSION

These loops represent the first description of PV changes in all 4 cardiac chambers and provide a granular view of hemodynamics in severe bicuspid AS. They extend what is known from previously conducted studies, which are LV-centric, and provide a comprehensive picture of cardiac function during TAVR (3,4). Furthermore, the antegrade placement of the catheter facilitated LV PV measurements during valve deployment and eliminated any potential measurement bias created by the conductance catheter, which can introduce aortic regurgitation.

These methodological enhancements facilitated the observation that LV unloading with TAVR, as described in published reports (4,5), may not take into effect immediately, or may be overshadowed by the acute negative consequences associated with key procedural steps such as RVP. Extended further, this implies that whereas newer-generation transcatheter valves can be repositioned or retrieved, these manipulations may not be without hemodynamic consequences.

CONCLUSIONS

In summary, multichamber PV analysis is technically feasible and demonstrates that the hemodynamic ramifications of TAVR are heterogeneous and chamber-specific. Ventricular stunning may occur as a result of RVP and abrogate the short-term beneficial effects typically expected with TAVR. Future studies of RV and atrial mechanics through PV analysis may help explain short- and long-term effects of TAVR in different subgroups.



illustrating antegrade positioning of the conductance catheter–placed from a femoral vein, coursing from the RA to LA via transseptal puncture and into the LV–immediately after transcatheter valve deployment. 1 = conductance catheter; 2 = Agilis steering sheath; 3 = pulmonary arterial catheter; 4 = temporary transvenous pacemaker; 5 = transcatheter aortic valve delivery sheath; and 6 = pig-tailcatheter in the aorta. (**D**) Intraprocedural echocardiographic confirmation of the conductance catheter in the RV apex from the apical 4-chamber view. Ao = aorta; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.



Pressure-volume (PV) loops before transcatheter aortic valve replacement (TAVR) in the RA, RV, LA, and LV are displayed in the **blue box at the top of the figure**. Key hemodynamic parameters in the RV and LV before TAVR are presented in the **table below** the PV loops. The **gray box** focuses on LV hemodynamics during valve deployment (n.b. that these loops reflect LV hemodynamics after deployment of a 26-mm S3 prosthesis, which had to be retracted due to significant paravalvular regurgitation). The **top panel** highlights PV loops in the LV; the **blue** PV loops are captured immediately before the 29-mm S3 prosthesis being deployed, the **gray** PV loops represent the LV during rapid ventricular pacing; and the loops represented by the gradient of colors from **yellow to red** were captured immediately after valve deployment. The **dashed line** connecting the vertical axis with the point of end-systole represents the end-systolic PV relationship (ESPVR), which serves as a surrogate of ventricular contractility. The pressure-time tracing in the **gray box** below the PV loops illustrates how LV pressure changes in each of the perivalve deployment phases. An intracardiac electrogram from the conductance catheter (labeled as ECG) during the pressuretime tracing is provided for reference in the **bottom panel**. Post-TAVR PV loops in all 4 cardiac chambers are displayed in the red box, and accompanied by notable RV and LV hemodynamic parameters in the **table below**. Pre-TAVR deployment loops are presented in **light blue** for comparison. All ventricular PV loops feature the ESPVR and end-diastolic PV relationship (EDPVR) as **dashed lines**. Vascular resistance (VR) in the tables is presented as pulmonary VR for the RV, and systemic VR for the LV. E_a = effective arterial elastance; EDP = end-diastolic pressure; EDV = end-diastolic volume; HR = heart rate; SW = stroke work; V₀ = volume-axis intercept; other abbreviations as in **Figure 1**.

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AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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