Transcatheter aortic valve-in-valve implantation: Go with the flow?

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Transcatheter aortic valve-in-valve (ViV) implantation has emerged as an attractive alternative to reoperative surgical aortic valve replacement (AVR) for degenerated bioprosthetic aortic valves (BPV).¹ The Valve-In-Valve International Data (VIVID) Registry, the largest multinational registry examining outcomes of ViV implantation, reported mortalities of 7.6% and 16.8% after 1 and 12 months, respectively.² In a systematic review pooling data of 18 trials with 823 patients, transcatheter ViV insertion was associated with reduced incidences of both stroke (1.9% vs 8.8%) and bleeding (6.9% vs 9.1%) relative to reoperative surgical AVR while providing similar valve hemodynamics and perioperative mortality (7.9% vs 6.1%).³

On the basis of the promising results of ViV implantation and of patients' preference for less-invasive procedures without permanent oral anticoagulation, the treatment strategy for patients requiring AVR is currently reshaping. The 2017 updated American Heart Association and American College of Cardiology guidelines for the management of patients with valvular heart disease have lowered the age limit for recipients of mechanical prostheses from 60 to 50 years and recommend an individually tailored choice of either a mechanical or bioprosthetic valve for patients between 50 and 70 years of age (class IIa, level B-NR).⁴ Consequently, it can be expected that the need for ViV implantation for structural deterioration of BPV is going to increase in the future and that patient selection will shift from patients judged by the heart team to be at high or prohibitive risk for reoperative surgical AVR to those considered to be at intermediate or low risk.

It is therefore time to improve our understanding of the underlying hemodynamic mechanisms after ViV insertion, which is currently based predominantly on feasibility trials and registries reporting clinical outcomes.¹⁻³ The report by Hatoum and colleagues⁵ in this issue of the *Journal* elucidates some important aspects by quantifying in vitro changes of aortic sinus blood flow dynamics by

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Central Message

Decreased aortic sinus flow and shear stress observed after transcatheter aortic ViV implantation, especially in the noncoronary sinus, may be a potential mechanism of thrombosis.

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high-resolution 2-dimensional particle image velocimetry before and after ViV insertion of 2 commercially available models (CoreValve 26 mm [Medtronic, Minneapolis, Minn] and Sapien 23 mm [Edwards Lifesciences Corp, Irvine, Calif]). ViV insertion was performed in an acrylic aortic valve chamber with a 23-mm Hancock bioprosthesis (Medtronic), and velocity fields within the coronary and noncoronary sinuses were captured. Two important results should be highlighted. First, both implanted ViV models reduced peak sinus flow velocity, vorticity, and shear stress, especially during systole, and may promote thrombus formation. Second, higher flow velocities and shear stress was measured in the coronary sinus than in the noncoronary sinus, making the coronary leaflets less susceptible to thrombus formation.

Translating these in vitro observations into direct clinical relevance seems premature. They do, however, certainly add another useful piece to the puzzle of leaflet thrombosis that has been reported recently after both transcatheter AVR and surgical AVR with bioprosthetic valves,⁶⁻⁸ a finding that could be even more frequent after ViV procedures and potentially could be counteracted by

strict oral anticoagulation. Although this work does not consider other important mechanisms that may impact sinus flow and promote thrombus formation, such as differing designs (internally vs externally mounted BPV), size and calcification degree of the BPV,⁹ and finally appropriate sizing and positioning of the valve during ViV implantation,¹⁰ Hatoum and colleagues⁵ are to be congratulated for their fundamental work.

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