Aortic tissue-stent mechanical interaction in transcatheter aortic valve replacement

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Statement of Purpose: Since the first procedure in 2002 [1], there has been an explosive growth in transcatheter aortic valve replacement (TAVR). By the end of 2011, about 50,000 TAVRs have been performed worldwide. Short- and medium-term outcomes after TAVR are encouraging with significant reduction in rates of death. However, adverse events associated with TAVR have been detected, including stroke, myocardial infarction, peripheral embolism, injury to the aorta, perivalvular leak, and access site injury [2, 3]. Furthermore, long-term durability and safety of these valves are largely unknown and need to be evaluated and studied carefully [4-6]. Successful deployment and function in TAVR is heavily reliant on the tissue-stent interaction. For instance, excessive radial force of the stent may cause aortic injury, while insufficient force may lead to paravalvular leakage and device migration. Therefore, a better understanding of the aortic tissue-TAV interaction is critical to TAVR success.

Methods: In this study, braided Nitinol stents were developed and tested to determine stent crimped diameter vs. stent radial force using a stent crimp experiment. We quantified important tissue-stent contact variables of self-expanding transcatheter aortic valve (TAV) stents when deployed into ovine and porcine aortic roots, such as the stent radial expansion force, stent pullout force, the annulus deformation response and the coefficient of friction on the tissue-stent contact interface. Furthermore, we conducted material and structural tests of tissues in the annulus region using uniaxial tensile and ring tests to quantify regional tissue properties. By doing so, we aimed to link the strength of tissues in the annulus region to the radial force of the stent to predict TAVI-induced tissue tearing.

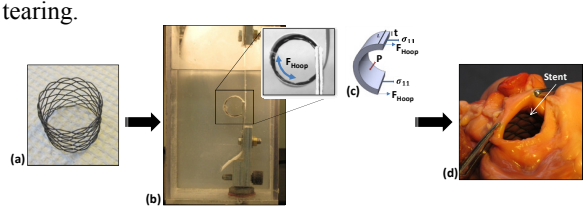

FIGURE 1. (a) Stent design. (b) Stent crimp experimental setup to measure hoop force at body temperature (37 C). (c) Free body diagram of stent cross-section labeled to show hoop force exerted on transcatheter valve stent from Dacron strap. (d) Stent in situ.

Results: The results indicated that when crimped at body temperature from 26 mm to 19, 21 and 23 mm stent radial forces were approximately 30-40% higher than those crimped at room temperature. Coefficients of friction leveled to approximately 0.10 ± 0.01 as stent wire diameter increased and annulus size decreased from 23 to 19 mm. Regardless of aortic annulus size and species tested, it appeared that a minimum of about 2.5 mm in annular dilatation, caused by about 60N of radial force from stent expansion, was needed to anchor the stent

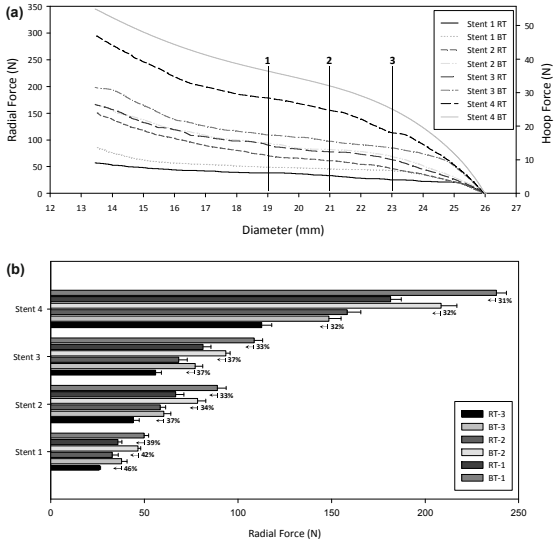

FIGURE 2. (a) Representative stent crimp diameter vs. radial force relationship of the four stents tested at (b) n= 4, crimped from 26 mm to 19, 21, and 23 mm, respectively (RT = room temperature; BT = body temperature).

against a pullout into the left ventricle. We found that the heterogeneous material properties at the annulus. The fibrous region of the aortic annulus could achieve radial force measures up to 230N before tissue rupture. The study of the contact biomechanics in animal aortic tissues may help us better understand characteristics of tissue-stent interactions and quantify the baseline responses of non-calcified aortic tissues.

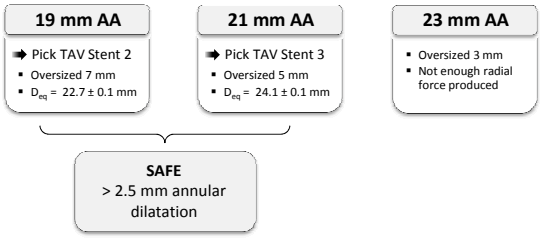

FIGURE 3. Transcatheter valve stent–tissue pairings. Oversize measure and corresponding equilibrium diameter position (Deq) for TAV stents offering an expansion force that meets the minimal required annulus dilation to counter migration in non-calcified aortic tissue.

Conclusion: This study helps to address important issues in the TAVR debate relating to device/aortic wall interactions; such data may facilitate TAV device design improvements to avoid aortic tissue rupture.

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References: 1.Cribier, A., et al., Circulation., 2002. **106**(24): p. 3006-8.
2.Grube, E., et al., J Am Coll Cardiol. , 2007. **1**(50): p. 69-76.
3.Webb, J., et al., Circulation, 2007. **7**(116): p. 755-763.
4.Movahed, M., Expert Rev Cardiovasc Ther., 2007. **6**(5): p. 997-8.
5.Ye, J., et al., Journal of Thoracic and Cardiovascular Surgery. **139**(5): p. 1107-1113.e1.
6.Webb, J.G. and D.A. Wood, Journal of the American College of Cardiology, 2012. **60**(6): p. 483-492.