

# **FEA-based biomechanical evaluation of** transcatheter aortic valve implantation in patients with calcific aortic stenosis



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# Introduction

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**Background.** Transcatheter Aortic Valve (TAV) implantation is a novel technique for the endovascular replacement of stenotic aortic valves (AVs) (Fig. 1). Despite its increasing application, TAV still presents some complications: severe vascular injuries, heart block and prosthesis insufficiency [1].

Clinical studies showed that AV calcifications affect the may TAV outcomes implantation of procedures.





Stent crimping and *in situ* expansion. The stent was crimped from an external radius of 13.0 mm to 4.7 mm (Fig. 3.a), the latter was chosen consistently with the real dimensions of the crimped device. Crimping generated peak Von Mises stresses (425 MPa) at the joints of the beams (Fig. 3.b) where plastic hinges formed (13.8% plastic strain). Stent postexpansion geometry was consistent with *in vivo* CT measurements [3, 4]: its outer radius and internal orifice were 12.6 mm and 4.0  $cm^2$ , respectively. Von Mises stress in the plastic hinges increased by 5% (Fig. 3.c,d). Contact forces were lower on the ventricular outflow tract (6.6 N) than on the AV (50.8 N), as required by the device specifications.

**Aim:** Recently, finite element analysis (FEA) were used to analyze the mechanics of TAV implantation and its *in situ* function.

Here, for the first time we simulate both, within a realistic model of the aortic root with calcifiied AV.

Figure 1. Edwards Sapien<sup>®</sup> TAV and implantation procedure.



The aortic root model was based on MRI in vivo measures. AV calcifications were modeled by a layer of stiffened shell elements superimposed to the healthy valvular tissue (Fig. 2.a).

The balloon-expandable Edwards Sapien<sup>®</sup> (Fig. 2.b), based on the model previously described in [2], was considered. All tissues were modeled as hyperelastic, while the stent was elasto-plastic with isotropic hardening.

Using LS-DYNA<sup>©</sup> 971 (LSTC, Livermore, CA, USA), four phases were modeled. First, the crimping of the device: rigid planes placed around the TAV stent (Fig. 2.c) were moved radially, reproducing the action of the iris device used for the valve loading. Second, the stent expansion within the aortic root (Fig. 2d) was simulated by applying a uniform pressure to the inner surface of the stent.



Figure 3. a) Stent configuration before and after crimping simulation. b) Von Mises stress in the stent at the end of the crimping simulation and c) after expansion simulation. d) Zoom on the plastic hinges of the stent.

**TAV function.** TAV orifice area throughout the cardiac cycle was within the range of physiological AVs and comparable to *in vivo* echo-Doppler measurements following TAV implantation [4]. Due to AV calcifications, asymmetric stress concentration was observed on the lateral attachments of TAV leaflets, where higher risk of prosthesis failure could be expected.



Figure 2. a) Model of the AV with calcifications (in blue) b) TAV model. C) Sketch of the crimping simulation. d) Picture of the crimped stent model within the aortic root before stent expansion

Third, TAV leaflets were positioned into the expanded stent: a nonuniform displacement field was applied to the nodes of their lines of insertion, anchoring them on the corresponding site on the stent. Fourth, TAV leaflets function throughout the cardiac cycle was simulated by applying a physiological trans-valvular pressure on their surface.

Penalty-based contacts between the stent and the leaflets were used. Dynamic simulations were performed, with mass scaling and a proper



Figure 4. a) Complete model after TAV implantation and snapshots of TAV leaflets at peak systole (b), early (c) and late diastole (d).

## Conclusions

FEA allowed to simulate the implantation and the function of a real TAV device within a realistic aortic root with AV calcifications, which represents the actual clinical scenario. Future efforts will be focused on simulating TAV dynamics through a fluid-structure interaction (FSI) approach.

### References

**a)** 

[1] Rodés-Cabau, *Nat Rev,* 2011, v. 9, pp. 15-29. [2] Tzamtzis et al., *Med Eng Phys*, 2013, v. 35, pp. 125-30. [3].Willson et al., *JACC Cardiovasc Interv*, 2012, v. 5, pp. 525-32

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