Comparison of transcatheter aortic valve and surgical bioprosthetic valve durability: A fatigue simulation study

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ABSTRACT

Transcatheter aortic valve (TAV) intervention is now the standard-of-care treatment for inoperable patients and a viable alternative treatment option for high-risk patients with symptomatic aortic stenosis. While the procedure is associated with lower operative risk and shorter recovery times than traditional surgical aortic valve (SAV) replacement, TAV intervention is still not considered for lower-risk patients due in part to concerns about device durability. It is well known that bioprosthetic SAVs have limited durability, and TAVs are generally assumed to have even worse durability, yet there is little long-term data to confirm this suspicion. In this study, TAV and SAV leaflet fatigue due to cyclic loading was investigated through finite element analysis by implementing a computational soft tissue fatigue damage model to describe the behavior of the pericardial leaflets. Under identical loading conditions and with identical leaflet tissue properties, the TAV leaflets sustained higher stresses, strains, and fatigue damage compared to the SAV leaflets. The simulation results suggest that the durability of TAVs may be significantly reduced compared to SAVs to about 7.8 years. The developed computational framework may be useful in optimizing TAV design parameters to improve leaflet durability, and assessing the effects of underexpanded, elliptical, or non-uniformly expanded stent deployment on TAV durability.

1. Introduction

Transcatheter aortic valve (TAV) intervention is now the standard-of-care treatment for inoperable patients and a viable alternative option for high-risk patients with symptomatic aortic stenosis (AS) (Haussig et al., 2014). Since the first TAV procedure in 2002 (Cribier et al., 2002), TAVs have been implanted in more than 150,000 patients worldwide (Zhao et al., 2014). Short and mid-term clinical results are promising: TAV intervention can significantly improve valve hemodynamics and patient quality of life with the added benefits of lower operative risk and shorter recovery time compared to surgical aortic valve (SAV) replacement (Kodali et al., 2012; Milburn et al., 2014). However, little is known about the long-term durability of these devices, owing in part to the relative immaturity of TAV intervention and the advanced age and illness of the patients selected for this treatment. To date, TAV intervention is still not considered for lower-risk patients: SAV replacement with either a mechanical or bioprosthetic valve remains the gold standard.

Bioprosthetic SAVs display superior hemodynamics to mechanical valves and eliminate the need for anticoagulant therapy. The caveat for these valves is the durability of the tissue leaflets due to calcification or fatigue-induced structural deterioration over time. The second generation pericardial valve from Edwards Lifesciences, the Carpentier–Edwards Perimount (CEP), has an in vivo durability of up to 20 years, which is a significant improvement over first generation valves such as the Ionescu–Shiley (IS) valve which has since been taken off the market for its subpar durability of approximately 5 years (Gabbay et al., 1984a,b; Brais et al., 1985, Reul et al., 1985; Cooley et al., 1986; Nistal et al., 1986a,b). The improved durability of the CEP valve has been attributed to valve design factors, such as the flexible stent which acts as a cushion to reduce leaflet stresses (Singhal and Luk, 2013), thicker leaflets, and improved leaflet coaptation (Vesely, 2001).

TAV leaflets are made of similar materials as the IS and CEP SAVs, generally either glutaraldehyde-treated bovine pericardium (GLBP) or porcine pericardium; however, TAV leaflets must be thinner to permit transcatheter delivery ($\sim$0.25 mm compared to $\sim$0.4 mm in SAVs). Furthermore, TAV stents do not allow for deflection to cushion the leaflets. It has been shown through finite element (FE) analysis that due to these constraints, TAV leaflets

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experience higher stresses and strains (Li and Sun, 2010) than traditional SAV leaflets, particularly in the presence of aortic calcification resulting in non-circular, asymmetric stent deployment (Sun et al., 2010; Gunning et al., 2014). Therefore, TAV devices can be expected to have reduced durability compared to SAVs. However, while there have been numerous biomechanics studies on TAV devices, they have been confined to the TAV function (Dwyer et al., 2009a; Li and Sun, 2010; Sun et al., 2010; Gunning et al., 2014; Kuetting et al., 2014) and interaction with the surrounding tissue (Dwyer et al., 2009b; Capelli et al., 2012; Wang et al., 2012, 2015; Auricchio et al., 2014; Morganti et al., 2014) immediately following implantation. The durability and potential failure modes of TAV devices remain unknown. Before TAV intervention can be considered a potential alternative treatment for lower-risk patients with longer life expectancies, there is a pressing need for new strategies to evaluate and improve leaflet durability.

The objectives of this study were two-fold: first, to develop a computational framework for assessing TAV leaflet fatigue under cyclic loading and second, to compare leaflet fatigue in a TAV and SAV under identical loading conditions and with identical leaflet material properties. This computational approach to assess leaflet fatigue is advantageous in terms of efficiency (i.e. hours of simulation time versus the months it may take to fabricate and test valves via accelerated wear testers), and also because it enables well-controlled, side-by-side engineering design comparisons. For instance, the precise effects of various design features (leaflet shape, free edge height, leaflet thickness, etc.) on TAV leaflet durability can be quantitatively assessed and compared to that of the CEP SAV under the same loading conditions.

2. Methods

2.1. Constitutive modeling of tissue fatigue

In this study, GLBP was selected as a representative valve leaflet material. GLBP is comprised of stiff collagen fibers embedded in a compliant matrix of elastin and proteoglycans, and thus can be considered a fiber-reinforced continuum. Accordingly, the total tissue free energy, W, was decomposed into isochoric, W_{iso}, and volumetric parts, W_{vol}, as

$$W(\mathbf{C}) = W_{iso}(\bar{\mathbf{C}}) + W_{vol}(\mathbf{C}), \tag{1}$$

where $\mathbf{C}$ is the right Cauchy–Green tensor, $\bar{\mathbf{C}}$ is the deviatoric right Cauchy–Green tensor, and W_{iso} is further decomposed into distinct matrix and fiber contributions denoted with "m" and "f" subscripts respectively giving

$$W_{iso} = W_m(\bar{\mathbf{C}}) + W_f(\bar{\mathbf{C}}, \mathbf{M}), \tag{2}$$

where $\mathbf{M}$ is a structural tensor describing the fiber orientation.

2.1.1. Fatigued state tissue free energy function

Details on the constitutive modeling of the soft tissue fatigue response were described previously (Martin and Sun, 2012, 2013). Briefly, to incorporate changes to the valve leaflet material properties as a result of fatigue damage, W, was enhanced with the addition of a stress-softening parameter, D_s, and a permanent set parameter, $\mathbf{D}_{ps}$, given by

$$W(\mathbf{C}, D_s, \mathbf{D}_{ps}). = (1 - D_s)W_{iso}^0(\bar{\mathbf{C}}, \mathbf{M}) + W_{ps}(\bar{\mathbf{C}}, D_s, \mathbf{D}_{ps})$$
$$+ W_{vol}^0(\mathbf{C}), \tag{3}$$

where W_{ps} is the dissipated energy due to the permanent set, and W^0 indicates the initial (un-fatigued) strain energy. At the unfatigued state, both D_s and $\mathbf{D}_{ps}$ are inactive, i.e. $D_s = 0$ and $\mathbf{D}_{ps} = 0$,

thus W reduces to the strain-energy function, W^0. The parameters D_s and $\mathbf{D}_{ps}$ become active with the onset of fatigue damage induced by cyclic loading.

GLBP tissue fatigue damage evolution was considered to be a function of the peak equivalent strain per cycle as in our previous studies (Martin and Sun, 2012, 2013). The equivalent strain, Ξ_t, (Simo, 1987) at time $t \in [0, T]$, a scalar quantity proportional to the distortional energy, was defined for the matrix and fiber constituents distinctly as

$$\Xi_{t_m}(\bar{\mathbf{C}}(t)): = \sqrt{2W_m^0(\bar{\mathbf{C}}(t))} \text{ and} \tag{4}$$

$$\Xi_{t_f}(\bar{\mathbf{C}}(t), \mathbf{M}): = \sqrt{2W_f^0(\bar{\mathbf{C}}(t), \mathbf{M})}. \tag{5}$$

The peak equivalent strains for each loading cycle were thus

$$\Xi_{n_m}^{peak} = \max_{t \in \left[\frac{n}{h}, \frac{n+1}{h}\right]} \sqrt{2W_m^0(\bar{\mathbf{C}}(t))}, \ n = 0, \ 1, \ 2, \ 3, \ \cdots, \ n_{tot_m}$$
$$\text{and} \tag{6}$$

$$\Xi_{n_f}^{peak} = \max_{t \in \left[\frac{n}{h}, \frac{n+1}{h}\right]} \sqrt{2W_f^0(\bar{\mathbf{C}}(t), \mathbf{M})}, \ n = 0, \ 1, \ 2, \ 3, \ \cdots, \ n_{tot_f}, \tag{7}$$

where h is the frequency and n is the number of loading cycles up to a maximum number of cycles, n_{tot_m} and n_{tot_f}. The number of cycles until failure (n_{tot}) were defined for the matrix and fiber constituents distinctly as (Martin and Sun, 2012):

$$n_{tot_k}\left(\Xi_{n_k}^{peak}\right) = \begin{cases} \infty & \text{if } \Xi_{n_k}^{peak} < \psi_{min_k} \\ \dfrac{\beta_k(n_{max_k} - 1)}{\Xi_{n_k}^{peak} - \psi_{min_k} + \beta_k}\left(\dfrac{1 - \exp^{\alpha_k\left(1 - \frac{\Xi_{n_k}^{peak}}{\psi_{max_k}}\right)}}{1 - \exp^{\alpha_{m,f_i}\left(1 - \frac{\psi_{min_k}}{\psi_{max_k}}\right)}}\right) \\ \quad + 1 \quad \text{if } \psi_{min_k} \leq \Xi_{n_k}^{peak} \leq \psi_{max_k}, \quad k = m, f, \\ 1 & \text{if } \Xi_{n_k}^{peak} > \psi_{max_k} \end{cases} \tag{8}$$

where α and β are material constants governing the amount of damage incurred by a single cycle at Ξ_n^{peak}, and ψ_{min} and ψ_{max}, define the boundaries of the fatigue damage evolution zone. Note that α, β, ψ_{min}, and ψ_{max} were also defined for the matrix and fiber constituents distinctly. The total amount of damage due to stress softening, D_s, after n tensile loading cycles was defined as

$$D_{s_k}\left(\Xi_n^{peak}\right) = \begin{cases} 0 & \text{if } \Xi_{n_k}^{peak} < \psi_{min_k} \\ \sum\limits_{n=1}^{n} \dfrac{1}{n_{tot_k}} & \text{if } \psi_{min_k} \leq \Xi_{n_k}^{peak} \leq \psi_{max_k}, \quad k = m, f. \\ 1 & \text{if } \Xi_{n_k}^{peak} > \psi_{max_k} \end{cases} \tag{9}$$

The tissue permanent set, D_{ps}, was considered to be due to damage to the matrix described by Martin and Sun (2013):

$$D_{ps_{ij}}\left(\Xi_{n_m}^{peak}\right) = \begin{cases} 0 & \text{if } \Xi_{n_m}^{peak} < \psi_{min_m} \\ \sum\limits_{n=1}^{n} \dfrac{1}{n_{tot_m}}D_{psmax}^n \dfrac{E_{n_{ij}}^{peak}}{E_n^{max}} \quad i, j = 1, 2 \\ \qquad\qquad\qquad \text{if } \psi_{min_m} \leq \Xi_{n_m}^{peak} \leq \psi_{max_m} \\ D_{psmax}^n & \text{if } \Xi_{n_m}^{peak} > \psi_{max_m} \end{cases} \tag{10}$$

Here the matrix permanent set is scaled by the peak strain ratio, $\dfrac{E_{n_{ij}}^{peak}}{E_n^{max}}$, to enforce anisotropy, where $E_{n_{ij}}^{peak}$ is the Green strain at $\Xi_t = \Xi_n^{peak}$ in direction ij, and $E_n^{max} = \max\{E_{n_{ij}}^{peak}\}$. The D_{psmax}^n refers to the maximum permanent set Green strain associated with