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Mitral leaflet modeling: Importance of *in vivo* shape and material properties

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ABSTRACT

The anterior mitral leaflet (AML) is a thin membrane that withstands high left ventricular (LV) pressure pulses 100,000 times per day. The presence of contractile cells determines AML *in vivo* stiffness and complex geometry. Until recently, mitral valve finite element (FE) models have neglected both of these aspects. In this study we assess their effect on AML strains and stresses, hypothesizing that these will differ significantly from those reported in literature.

Radiopaque markers were sewn on the LV, the mitral annulus, and AML in sheep hearts, and their four-dimensional coordinates obtained with biplane video fluoroscopy. Employing *in vivo* data from three representative hearts, AML FE models were created from the marker coordinates at the end of isovolumic relaxation assumed as the unloaded reference state. AML function was simulated backward through systole, applying the measured trans-mitral pressure on AML LV surface and marker displacements on AML boundaries.

Simulated AML displacements and curvatures were consistent with *in vivo* measurements, confirming model accuracy. AML circumferential strains were mostly tensile (1–3%), despite being compressive (−1%) near the commissures. Radial strains were compressive in the belly (−1 to −0.2%), and tensile (2–8%) near the free edge.

These results differ significantly from those of previous FE models. They reflect the synergy of high tissue stiffness, which limits tensile circumferential strains, and initial compound curvature, which forces LV pressure to compress AML radially. The obtained AML shape may play a role not only in preventing mitral regurgitation, but also in optimizing LV outflow fluid dynamics.

1. Introduction

The anterior mitral leaflet (AML) is a pivotal component of the mitral valve that swings open widely during diastole to facilitate left ventricular filling; then, during systole, it not only coapts tightly with the posterior mitral leaflets to prevent backward flow into the left atrium, but also provides an important part of the funnel-shaped outflow tract during left ventricular ejection. A better understanding of AML mechanics would increase knowledge of the function of entire mitral valve (MV). This could be useful from a clinical perspective, allowing for a deeper comprehension of the mechanisms underlying MV diseases and the development of more effective surgical procedures and prosthetic devices. It could also guide emerging applications in the tissue engineering field.

Different methods have been used to analyze AML and MV biomechanics; among them, finite element modeling (FEM) has proven useful to investigate the physiological behavior of MV (Kunzelman et al., 1993; Votta et al., 2008; Prot and Skallerud, 2009; Skallerud et al., 2011), its alterations with pathological conditions (Kunzelman et al., 1998; Prot et al., 2010), and the effects of surgical repair techniques (Votta et al., 2002; Avanzini, 2008).

Recently, FEM was combined with *in vivo* experiments in ovine hearts to focus such analysis on the *in vivo* mechanical properties of AML through a reverse-engineering approach. These studies have concluded that:

i) AML mechanical properties arise not only from a passive matrix of elastin and preferentially oriented collagen fibers, but also by active, contractile elements within AML that change the stiffness of leaflet during the cardiac cycle. These have been proposed to be myocardial cells (MCs), smooth muscle cells (SMCs), and valvular interstitial cells (VICs).
ii) Such contractile elements play a major role in determining AML mechanical properties. As a consequence, AML is stiffer

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