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INSIGHTS INTO REGIONAL ADAPTATIONS IN THE GROWING PULMONARY ARTERY USING A MESO-SCALE STRUCTURAL MODEL: EFFECTS OF ASCENDING AORTA IMPINGEMENT

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Delineating the normal postnatal development of the PA can inform our understanding of congenital abnormalities, as well as the effects of related diseases such as pulmonary hypertension. In particular, we were interested in the effects of increasing impingment of the ascending aorta on the regional mechanical behavior of the growing PA (Fig. 1). Structurally motivated models incorporate significant mechanical aspects of the underlying microstructure. Hence, structurally based models can help elucidate the mechanisms governing the structure-function relationship of biological tissues and better predict the alteration in mechanical behavior as a result of a disease condition. Such approaches have been utilized for arterial tissues including the PA. However, the models developed so far have not addressed the mechanical behavior of normal growing vessels from the early juvenile to the adult states. Moreover, the reliability of such models considerably depends on the accurate quantification of organization and load-bearing behavior of fibrous components of the tissue. In the case of the PA, these are collagen, elastin, and to a lesser extent, smooth muscle as the mechanically significant structural components. Thus, the elastin and collagen structure-function relationship of the normal PA and in connection to growth changes need to be utilized in such models. Utilizing the extensive experimental structural and biaxial mechanical measurements from our previous study (J Biomech Eng. 2013 Jul 1;135(7):71010-12), including collagen fiber engagement with strain, we developed a structural constitutive model for the PA. The constitutive model developed also takes account of contributions of the ground matrix, consisting of non-fibrillar elastin, smooth muscle cells and other non-cellular materials, to delineate the structure-function relationship of PA wall in the postnatal growth period by comparing the juvenile and adult stages. The model fit the available b iaxial data well (Fig. 2). Our key findings were that while there are regional variations with age, the major effects of the age related changes were exhibited in the medial aspect of the PA wall (Fig. 1). Specifically, we observed that structurally, the elastin and collagen fibers' in-plane splay underwent opposite changes, and a trend towards more rapid collagen fiber recruitment with respect to strain. The most profound changes were observed with the fiber moduli, with the elastin modulus increasing by ~50% and the collagen modulus reducing to only ~25% of the juvenile value (Fig. 3). While this change was in part driven by the observed increase in total tissue elastin volume fraction relative to the collagen volume fraction, these changes were also a direct result of the changes in local tissue structure. As observed in our geometric studies the focal changes observed in the medial region are likely due to the impingement of relatively stiff ascending aorta on the growing PA. Intuitively, the effects of the local impingement would be to lower the local wall stress. If so, then this would be consistent with the observed decrease in collagen modulus. However, it does not explain the concomitant increase in elastin modulus. Clearly, some type of compensatory mechanism is in play in here, but the nature of which remains unknown. What can be said with some certainty is that, during the postnatal somatic growth, local stresses can substantially modulate the development of regional tissue microstructure and mechanical behaviors in the PA. Finally, we underscore that in our previous studies suggest an increase in effective PA wall stress with postnatal maturation. This observation is contrary to the accepted theory of maintenance of homeostatic stress levels in the regulation of vascular function, and suggests alternative mechanisms regulate postnatal somatic growth. Understanding the underlying mechanisms, including incorporating important structural features during growth, will help to improve our understanding of congenital defects of the PA and lay the basis for functional duplication in their repair and replacement by simulation techniques to be discussed.

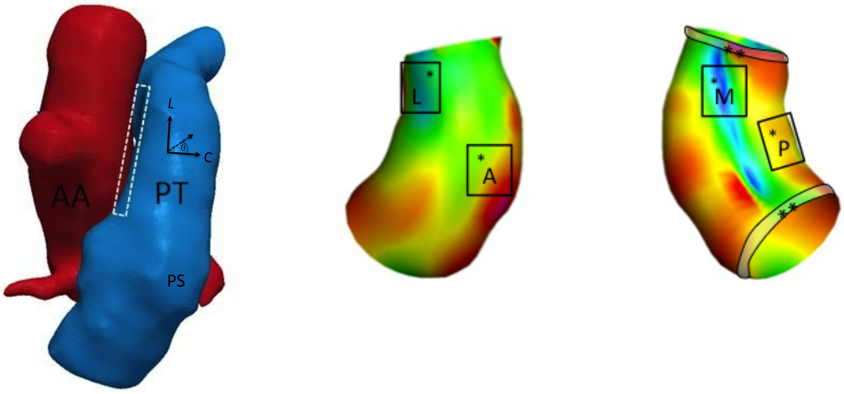

Figure 1. (a) 3D surface reconstructions of the ovine ascending aorta (AA) and pulmonary trunk (PT), with the white box showing the region of impingement between the two great vessels, (b) Two views showing the Posterior, Anterior, Lateral, and Medial regions of the PT studied.

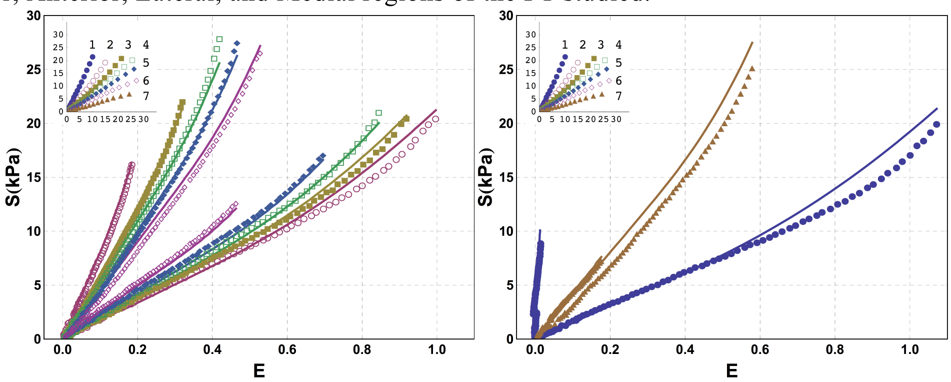

Figure 2. Constitutive model fit to the average five-protocol biaxial stress-stretch data of juvenile and adult medial PA wall specimens.

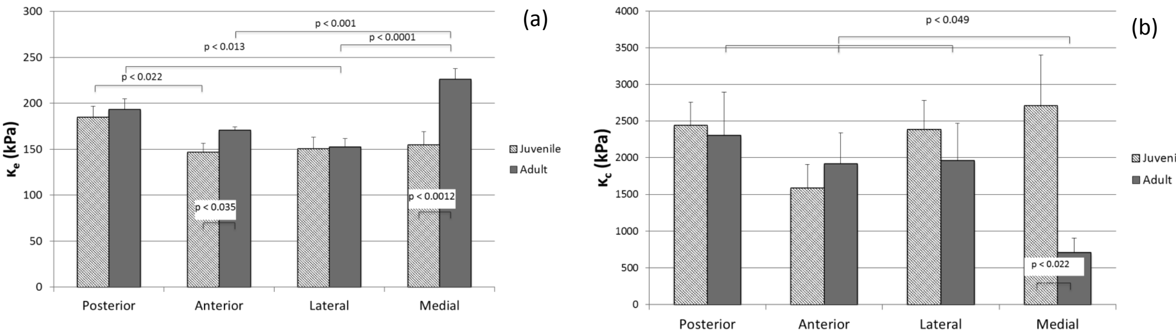

Figure 3. Predicted collagen (κc) and elastin (κe) fiber moduli regions at both age time points. The most drastic changes were for the elastin modulus ke increasing by ~50% and the collagen modulus kc decreasing to only ~25% of the juvenile value in the medial region.

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