CHARACTERIZATION OF TRICUSPID AND BICUSPID AORTIC VALVE HEMODYNAMICS UNDER NORMAL AND CALCIFIED CONDITIONS

Abstract

by

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Calcific aortic valve disease (CAVD) is the most prevalent form of heart valve disease, affecting 25% of the population 65 years and older with disease progression lasting 20-30 years. It is marked by the initial thickening and stiffening of the leaflets, followed by the formation of calcific lesions within the valve leaflets. In the bicuspid aortic valve (BAV), which is the most common type of aortic valve defect and is characterized by the formation of two functional leaflets instead of three, the onset of CAVD typically occurs at an earlier age and progresses to severe stenosis much more rapidly than in the normal TAV. While the etiology of CAVD has been linked to altered hemodynamics, alterations in hemodynamic performance due to anatomic and calcific defects have not been investigated. Therefore, the goal of this dissertation is to present a comparative in vitro assessment of the valvular flow and performance in a TAV and BAV under normal and calcified states.

Particle-image velocimetry was used to characterize the flow downstream of a model TAV, model BAV, model calcified TAV, and model calcified BAV mounted in an idealized aortic sinus geometry and subjected to a pulsatile flow environment. Valve performance was analyzed under steady flow conditions in terms of geometric orifice area, effective orifice area, and energy loss. Comparison of valve hemodynamics was based on velocity, vorticity, viscous shear stress, and Reynolds shear stress.

This study found that valvular anatomy and leaflet stiffening impact large-scale flow structures, but suggests that the BAV can function and generate energy loss at a level similar to a normal valve despite its intrinsic degree of stenosis. The main difference in valve performance between the BAV and TAV was in the elevated shear stress and turbulence experienced by the BAV. Calcification of the valve also led to a similar increase in the viscous shear stress and turbulence but significant decrease in valve performance. These similarities in the hemodynamic alterations in valves with anatomic or calcific defects as compared to normal TAV hemodynamics demonstrated a positive feedback cycle, which may be linked to CAVD development and progression.