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Determination of pressure data from velocity data with a view towards its application in cardiovascular mechanics. Part 2. A study of aortic valve stenosis

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ABSTRACT

This paper is Part 2 of a study of blood flow across cardiovascular stenoses. In Part 1, we developed a rigorous mathematical approach for deriving a pressure field from experimental data for a velocity field that can be obtained by direct measurement. In this Part, existing methods for quantifying stenoses, with specific reference to cardiac valves, are reviewed. Using the mathematically rigorous and physically reasonable approach that we developed in Part 1, for a pre-specified flow velocity field proximal to the stenosis and pressure waveform field distal to the stenosis, we ascertain the intra-stenosis and distal flow velocity field, pressure field proximal to and within the stenosis, and energy dissipation, all as functions of position and time. The computed dissipation, kinetic energy and pressure are then presented in an idealized geometry, but relevant to a realistic geometry, with a symmetric stenosis.

1. Introduction

Stenotic cardiac valve and arterial occlusive diseases are among the leading causes of death worldwide; see Go et al. (2013). Interventional and surgical treatments have provided improvements in survival, cardiac function, and functional capacity. However, procedural therapies have substantial risks, and benefits are proportional to the physiological severity of the stenoses treated. Consequently, accurate and precise assessment of stenosis severity is required in order to appropriately decide whether and what type of treatment is warranted for a given lesion.

A stenosis in the cardiovascular system is a reduction in cross-sectional area of a structure across which blood flows. An anatomic stenosis, which is simply defined by its existence, may or may not result in a physiologically important stenosis. The physiological impact of a stenosis is the extent to which it poses increased impedance to blood flow, i.e., the extent to which energy of the flowing blood is dissipated or lost in order to generate and maintain flow and ultimately, to which blood flow becomes impaired. Stenoses are generally treated when they are physiologically important. Physiologically important stenoses satisfy two criteria: (1) hemodynamic severity, and more importantly, (2) adverse effects on proximal (e.g., the left

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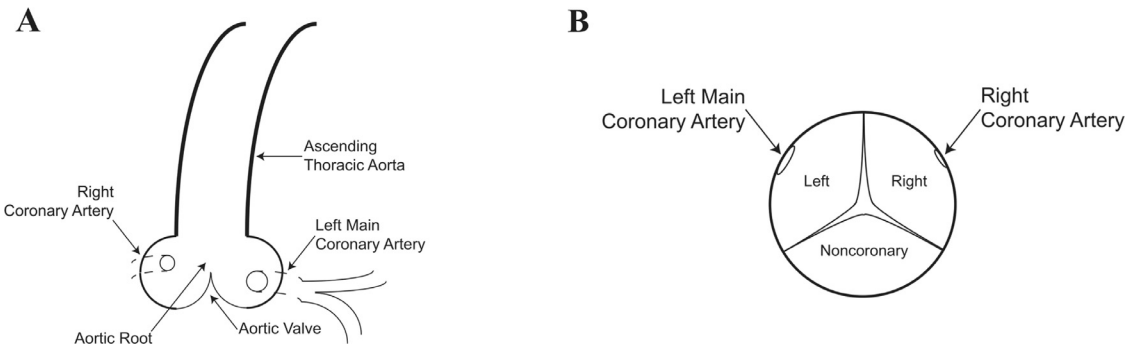

Fig. 1. Anatomy of the human aortic valve/root complex. The aortic valve, comprised of three cusps (leaflets), is shown in the diagram as being attached to the aortic root circumferentially, forming a circumferentially thickened ridge of fused cusp/aortic tissue termed the aortic valve annulus. The annulus is a three-dimensional crown-like structure, as opposed to a planar structure. In addition, the diameter of the base of the annulus (ventriculo-aortic junction) is smaller than the diameter of the aortic root-ascending thoracic aortic junction (a.k.a. sinotubular junction), such that the annulus is within a conic section; this is not depicted to scale in **A**. The aortic root constitutes the tissue housing for the valve; the root tissues are comprised of the three sinuses of Valsalva. At the initiation of left ventricular systolic ejection, the left ventricular outflow tract-to-aortic root pressure gradient forces the cusps of the aortic valve radially outwards, increasing the orifice cross-sectional area and permitting blood flow out of the left ventricle and into the aortic root throughout systolic ejection, and thence downstream. At the end of left ventricular systolic ejection, the aortic root-to-left ventricular outflow tract pressure gradient forces the cusps radially inwards, resulting in circumferential cusp coaptation and elimination of the potential orifice for regurgitant blood flow back into the left ventricular cavity. The left sinus of Valsalva is typically slightly smaller than the two other sinuses (not shown to scale), the right and noncoronary sinuses. The left main and right coronary arteries, which provide cardiac tissue/myocardial blood flow, each arise as separate ostia from the left and right sinuses of Valsalva, respectively. The segment of aorta downstream of/distal to the aortic root is termed the ascending thoracic aorta, which does not contain branch arteries. **A.** Long-axis view. In this view, not all three cusps can be seen. Rather, cross sections of the right and left cusps are shown. The view demonstrates the valve during either systolic isovolumetric contraction, or either phase of diastole. **B.** Short-axis view. In this view, all three cusps can be visualized. This view is an approximate representation of the surgeon's view of the aortic valve/root complex when the ascending thoracic aorta has been transected proximally. The view also demonstrates the valve during systolic isovolumetric contraction, or either phase of diastole. A central orifice is merely presented to highlight the separate nature of each valve cusp; in reality, a competent (non-regurgitant) valve has a minimal or absent central orifice.

ventricle in aortic valve stenosis) or distal (e.g., myocardial territory in the distribution of a stenotic coronary artery) tissues and organs.

Both invasive and non-invasive diagnostic techniques have been used to assess cardiovascular stenoses; see Bhattacharyya, Khattar, Chahal, Moat, and Senior (2013); Carroll (1993); Leggett and Otto (1996). While invasive techniques accurately determine hemodynamic severity and are the historical gold standard, they carry procedural risks. Consequently, non-invasive techniques have been used increasingly. However, as we outline below, current approaches to interpreting non-invasive data are incapable of ascertaining physiologic stenosis severity. In this manuscript, we develop an improved approach towards determination of the energy dissipation in the flowing blood and pressure gradients and differences across cardiovascular stenoses, which can be applied to non-invasive diagnostic modalities.

Background

Various methods have been used to evaluate stenoses by either anatomic or physiologic criteria. Broadly, anatomic approaches either directly measure cross-sectional area, or invoke conservation of mass to calculate cross-sectional area. In contrast, physiological approaches directly measure intraluminal pressure and/or flow velocity. Measured trans-stenosis pressure difference and calculated stenosis “resistance” or “impedance” are conceptually sound assessments of the physiologic impact of a stenosis. However, other physiological approaches used currently, notably calculated intraluminal pressure derived from measured flow velocity, or even calculated cross-sectional area derived from measured intraluminal pressure and volumetric flow rate, are fundamentally unsound from a fluid mechanical perspective. The relative strengths and weaknesses of these various approaches are reviewed below. Figs. 1–3 depict the left-sided cardiac valves (aortic and mitral) and the physiology of cardiovascular stenoses.

1.1. Anatomic

Direct measurement of valve or arterial cross-sectional area historically has been both inaccurate and imprecise. However, conservation of mass is applicable over one or more cardiac cycles because the circulation is a closed system and blood is an incompressible material, even with deformable conduits. Thus, the mean volumetric flow rate is constant along a given conduit, assuming the absence of branch vessels. The cross-sectional area at a given location along the length of the conduit thus may be calculated, as it equals the mean volumetric flow rate over a cardiac cycle divided by the magnitude of the mean flow velocity through the cross-sectional area in question over a cardiac cycle; see Kosturakis, Goldberg, Allen, and Loeber (1984); Warth, Stewart, Block, and Weyman (1984). Cardiac valve area and arterial (carotid) cross-sectional area have