FLUID STRUCTURE INTERACTION NUMERICAL SIMULATIONS OF SERIAL PULMONARY ARTERY STENOSES

Eric Hong[1], Rosaire Mongrain[1,*], PhD, Olivier F. Bertrand[2], MD, Josep Rodes-Cabau[3], MD
[1]*Mechanical Engineering Department, McGill University, Montreal, QC*
[2]*Montreal Heart Institute, Montreal, QC,* [3]*Quebec Heart Institute, Sainte-Foy, QC*

Abstract

The focus of this research is to numerically investigate the effects of fluid structure interaction and spatial configuration of serial stenoses on the hemodynamics and wall motion of the pulmonary artery. Collapse and flow choking phenomena were observed by Kobayashi et al. during a pulsatile flow experiment involving a hydrogel stenosis tube model [1]. In a related study, Tang et al. observed that tube geometry is one of the most important factors affecting flow and wall behaviors [2]. The hypothesis of this study is that some 3D configurations of serial pulmonary artery stenoses are more susceptible to collapse under physiological conditions. i.e. they would exacerbate localized collapse and induce flow choking, leading to a near total occlusion of flow to the lung. This recurring state would produce a lethal ventilation-perfusion mismatch.

Keywords: Fluid Structure Interaction, Stenosis, Pulsatile Flow, Biomechanics

[*]Corresponding author: *rosaire.mongrain@mcgill.ca*, (514) 398-1576

INTRODUCTION

Pulmonary artery stenosis occurs in 10-60% of patients with tetralogy of Fallot with or without pulmonary atresia [3-5], in patients with Alagille or Williams syndromes [5-7], and occasionally after placement of systemic to pulmonary shunts [5, 8] or after the arterial switch operation [5, 9, 10]. For patients with congenital or postoperative branch pulmonary artery stenoses, the elevated right ventricular pressure is associated with right ventricule failure, arrhythmias and sudden death [11-13]. Within the venous system, hemodynamically significant stenoses, including cavopulmonary anastomoses, can contribute to superior vena cava syndrome, poor passive flow with poor cardiac output and atrial arrhythmias. Elevated central venous pressures in these patients is a risk factor associated with higher mortality [13, 14].

Surgical treatment with balloon angioplasty using low- or high pressure balloons is associated with significant morbidity, mortality, with success rate ranging from 53-72% [5, 15-17], and recurrence of stenosis secondary to scar tissue [5, 18, 19].

FDA phase one clinical trials have demonstrated that intravascular stents are an effective and safe therapy for the treatment of vascular stenoses in patients with congenital heart disease (1998) [13, 20-24]. Endovascular stents are now commonly implanted in the pulmonary circulation to treat pulmonary arterial stenoses with generally excellent results [20, 23-26].

However, stent implantation as an alternative to the balloon angioplasty does not eliminate the possibility of mortality. The risk of death directly attributed to the stent procedure has been documented. In one patient with familial congenital pulmonary artery branch stenosis and suprasystemic pulmonary pressure, the stent implantation into two small distal pulmonary segments increased the flow to the stented lung segments. This augmentation was so severe that segmental pulmonary edema developed with a progressive and lethal ventilation-perfusion mismatch [13]. The second case involves a small child 7 weeks status post tetralogy of Fallot repair with severe residual bilateral branch pulmonary artery stenosis. He had intractable right heart failure and was not considered a candidate for re-operation. During stent dilation, there was a main pulmonary artery tear resulting in a massive hemothorax, resulting in death [13].

The hypothesis of this study is that some 3D configurations of serial pulmonary artery stenoses are more susceptible to collapse under physiological conditions. i.e. they would exacerbate localized collapse and induce flow choking, leading to a near total occlusion of flow to the lung. This recurring state would produce a lethal ventilation-perfusion mismatch.

THE COMPUTATIONAL METHOD

The modeling method originates with the construction of the various geometries with the modeling software Rhinoceros (McNeel) (Figure 1). Meshing is then performed on the pre-processor of the finite element extension of ANSYS. The resulting data is converted to LS-DYNA (Livermore software technology corporation). The structural components are modeled with Langrangian shell elements. The surrounding fluid control volume is assembled with Eulerian brick elements. A flow reservoir attached to the inlet supplies the physiologically relevant pressure conditions to the fluid domain [27] (Figure 2). The Belytschko-Tsay shell element formulation will be linked with an isotropic elastic material model. The Young's Modulus is provided by [28, 29] with a specified poisson's ratio of 0.45 (Figure 3). More details about the governing process of the ALE formulation for fluid-structure interaction may be found in the literature. [30-33].

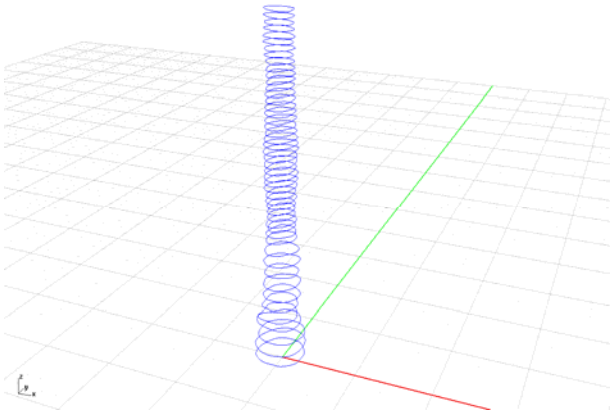

Figure 1: Typical artery geometry modeling

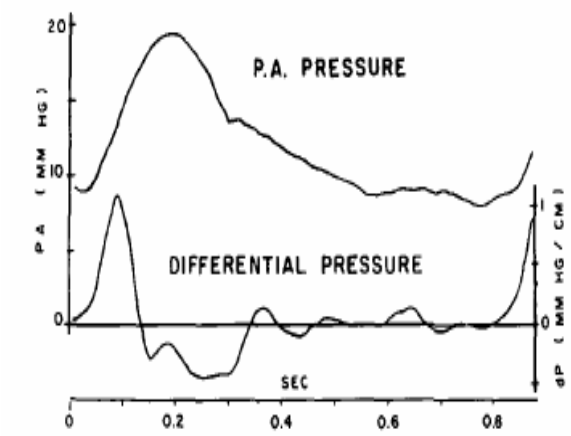

Figure 2: Physiological pulmonary artery pressure

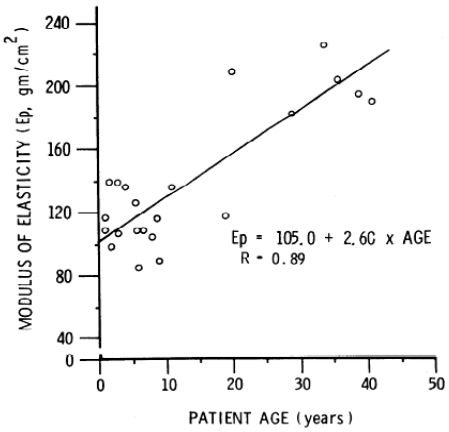

Figure 3: Modulus of elasticity v. patient age

PRELIMINARY RESULTS

Initially, a preliminary investigation was performed on a single stenosis on the left anterior descending (LAD) branch of the left coronary artery. The early results suggest that contribution of fluid structure interaction on the hemodynamics and wall motion is negligible. Furthermore, neither wall collapse nor flow choking were observed (Figure 4, 5, 6).

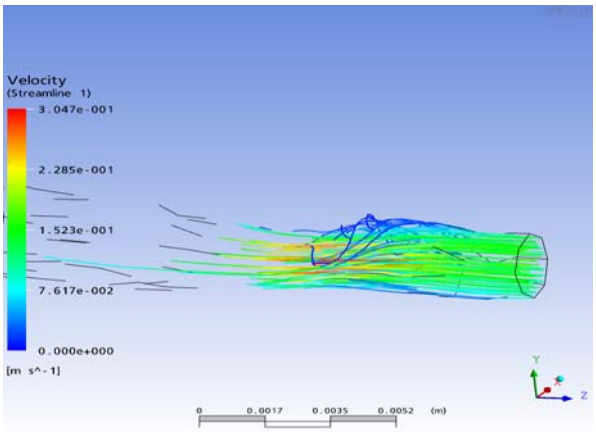

Figure 4: Streamline velocity of stenotic LAD

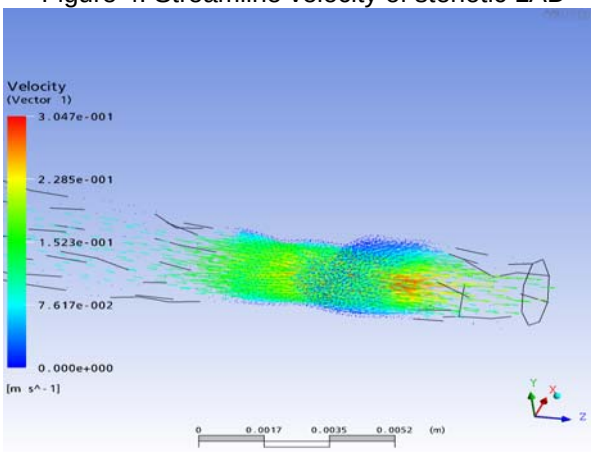

Figure 5: Velocity vector of stenotic LAD

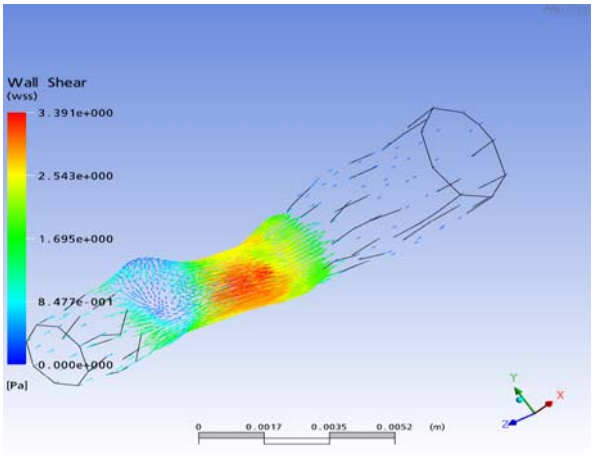

Figure 6: WSS of stenotic LAD

Physically, it may be argued that the required inner fluid pressurization to overcome the frictional pressure drop and transmural pressure gives rise to a tensile hoop prestress, and through the Poisson effect, to an axial tensile prestress. Since the ends are clamped, this will tend to stiffen and thus stabilize the shell [34]. i.e. the high pressure systemic arterial circulation combined with the bulky geometry (small diameter to membrane thickness) of the coronary artery will reduce the likelihood of collapse under physiological conditions.

From a numerical standpoint, the computations for the coronary artery were performed on a staggered time-integration scheme using ANSYS MFX. Refer to [35] for a thorough discussion about the stability, accuracy, convergence [36, 37], and load transfer between non-matching meshes [38-40] of this method. It was found that this loosely-coupled (partitioned) approach is inappropriate for large deformations dynamics. This is the technical basis for substituting for the ALE formulation in modeling fluid structure interaction of serial pulmonary artery stenoses, where large deformations are presumed to occur. This assumption is founded on the greater vascular distensibility and substantially diminished steady resistive opposition to flow (vascular resistance) of the pulmonary circulation in comparison to the systemic one. The pulsatile nature of ventricular ejection and the homeostatic requirement for an equivalent amount of blood ejected per beat from each ventricle, place these unique contraints upon the pulmonary arteries [41].

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