

FMD2013-16141

OPTIMIZATION OF BALLOON OBSTRUCTION FOR SIMULATING EQUIVALENT  
 PRESSURE DROP IN *IN-VIVO* CONDITIONS

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**ABSTRACT**

The study of hemodynamics in an animal model associated with coronary stenosis has been limited due to the lack of a safe, accurate, and reliable technique for creating an artificial stenosis. Creating artificial stenosis using occluders in an open-chest procedure has often caused myocardial infarction (MI) or severe injury to the vessel resulting in high failure rates. To minimize these issues, closed-chest procedures with internal balloon obstruction were often used to create artificial stenosis. However, it should be noted that the hemodynamics in a blood vessel with internal balloon obstruction as opposed to physiological stenosis hasn't been compared. Hence, the aim of this research is to computationally evaluate the pressure drop in balloon obstruction and compare with that in physiological stenosis. It was observed that the flow characteristics in balloon obstruction are more viscous dominated, whereas it is momentum dominated in physiological stenosis. Balloon radius was iteratively varied to get a pressure drop equivalent to that of physiological stenosis at mean hyperemic flow rates. A linear relation was obtained to predict equivalent balloon obstruction for physiological stenosis.

**INTRODUCTION**

Mortality associated with coronary artery disease is usually caused by myocardial ischemia due to one or more critically stenosed or occluded coronary arteries (Fig 1A, Fig 1B). Stenotic condition has been modeled *in-vivo* in different animals. In majority of the *in-vivo* studies, different degrees of area stenosis are created in anesthetized animals, either in open- or in closed- chest condition.

In order to create a model of coronary disease *in-vivo*, that reproduces the hemodynamic effects of a stenotic coronary vessel, an artificial stenosis must be introduced. Artificial stenosis in an open chested model can be achieved by a hydraulic occluder positioned

around the artery or by tightening a J-shaped screw clamp. Although these techniques have been shown to be effective in creation of an artificial coronary stenosis, they are known to cause complications and cannot accurately and reliably quantify the degree of obstruction. Further, they often cause severe injury to the vessel of interest.

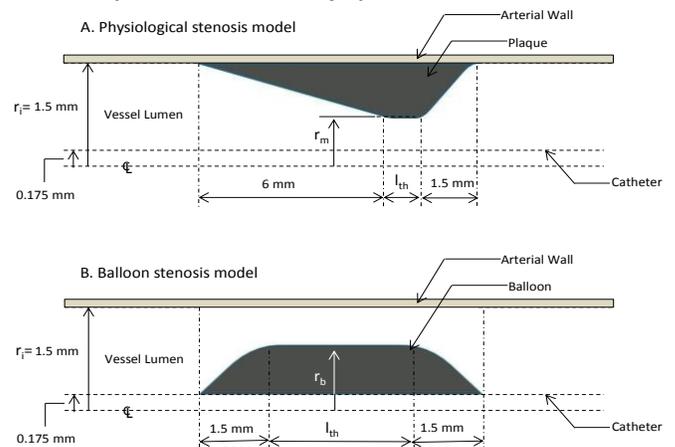


Figure 1: Geometry for (A) physiological and (B) balloon stenoses

On the other hand, artificial stenosis in a closed chest model can be achieved by inflation of an intracoronary balloon placed at the tip of catheter as shown in Fig 1B. The closed-chest model has an advantage as it minimizes tissue trauma, but the approach requires fluoroscopy for accurate positioning of the balloon obstruction. This technique provides control over the degree of stenosis to be created, and allows creation of a wide range of stenoses safely, quickly, and

repeatedly. However, it should be noted that the hemodynamics associated with internal balloon obstruction may not represent the hemodynamics associated with that of a physiological stenosis. Hence, the purpose of this study is to computationally determine equivalent balloon stenosis that results in a pressure drop similar to that of a physiological arterial plaque.

**METHODS**

**Geometry.** A physiological arterial plaque model and a balloon induced stenosis model were assessed for three levels of blockage having 64%, 80% and 90% area stenosis along with a 0.35 mm guidewire. The geometry (Fig 1A) and dimensions (Table 1) for the physiological stenosis model has been described previously [1]. The geometry (Fig 1B) and dimensions (Table 1) for the balloon stenosis model were based on angioplasty balloons. A balloon length of 7 mm and 20 mm balloon were assessed in this study. All the sharp corners of balloon and physiological stenosis were rounded with a fillet of 1 mm and 2.5 mm radius, respectively, to reflect *in-vivo* geometry.

**Table 1: Dimensions (in mm) for the geometric model**

% Area Stenosis	All dimensions in mm				
	Physiological Stenosis Model		Balloon Stenosis Model (Internal Obstruction)		
	$r_m$	$l_{th}$	$r_b$	7 mm Balloon $l_{th}$	20 mm Balloon $l_{th}$
64	0.900	3.000	1.200	7.000	20.000
80	0.675	0.750	1.340	7.000	20.000
90	0.475	0.750	1.420	7.000	20.000

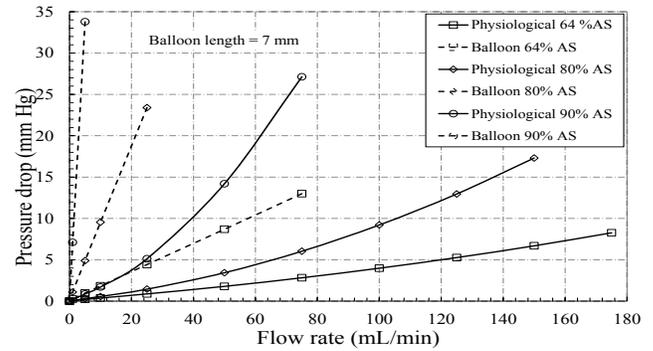
**Assumptions.** It is assumed that: (i) the arterial wall has a rigid and concentric shape; (ii) the stenosis geometry remains unchanged during basal and hyperemic (maximal flow condition) flows and, (iii) blood flow through the artery is averaged over time.

**Boundary and Initial conditions.** The no slip boundary condition was specified on the arterial wall, balloon surface and on the guide wire. Stress free boundary condition was applied at flow outlet and time averaged velocity [1] was used as input boundary condition. The blood was modeled as non-Newtonian carreau and incompressible fluid. The density of blood was considered to be 1.05 gm/cm<sup>3</sup>. The numerical model was solved using finite volume method (Ansys Fluent [v 14.5]).

**Solution Strategy.** The pressure drop for the physiological and balloon stenosis models at the three blockage levels were calculated using the corresponding mean hyperemic flow rates [1]. The pressure drop ( $\Delta p$ ) vs. flow rate (Q) variation was calculated for each stenosis model. Balloon radius was iteratively varied to get a pressure drop similar to that of physiological stenosis at mean hyperemic flow rates. The balloon geometry that meets the above criteria at the mean hyperemic flow rates (of physiological stenosis) was termed as equivalent balloon stenosis.

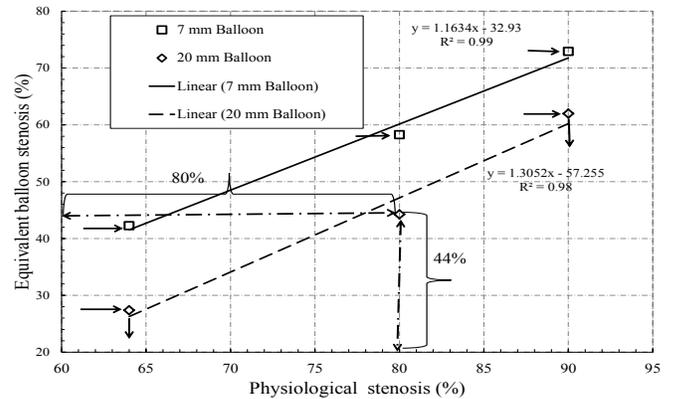
**RESULTS**

The relationship between stenosis pressure drop and flow is curvilinear and is described by  $\Delta p = aQ + bQ^2$  where  $a$  and  $b$  are stenosis-specific constants for viscous and momentum losses, respectively. From Figure 2, it can be observed that as stenosis severity increases for physiological stenosis, the  $\Delta p$ -Q shifts from almost linear (64%AS) to a non-linear curve (90 % AS), indicating the importance of momentum related losses with increasing stenosis severity. However, as stenosis severity increases for balloon stenosis, the  $\Delta p$ -Q remains almost linear (64% AS) to a linear curve (90% AS), indicating the predominance of viscous related losses in balloon obstructions.



**Figure 2:  $\Delta p$  vs. Q characteristic of 64%, 80% and 90% AS, for physiological stenosis and stenosis by a 7 mm long balloon**

Figure 3 shows the variation of balloon (internal) stenosis with respect to physiological stenosis for the same pressure drop. For a 7 mm balloon length, the equivalent balloon area stenosis of 42%, 58% and 73% AS is observed to have a pressure drop similar to that of physiological stenosis of 64%, 80% and 90% AS respectively. Similarly, for a 20 mm balloon length, the equivalent balloon area stenosis of 27%, 44% and 62% AS is observed to have a pressure drop similar to that of physiological stenosis of 64%, 80% and 90% AS respectively.



**Figure 3: Comparison of equivalent balloon and physiological stenosis**

**CONCLUSION**

Blood flow through a balloon obstruction and physiological stenosis has been computationally analyzed. It was observed that the flow characteristics in balloon obstruction are more viscous dominated, whereas its momentum dominated in physiological stenosis. Further, a linear relation to predict equivalent balloon obstruction is proposed. In future, it is of interest to extend this study to account for compliance effects of arterial wall.

**ACKNOWLEDGEMENT**

This work is supported by financial support from Department of Veteran Affairs through VA Merit Review Grant (101CX000342-01).

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