Fluid Dynamics of Blood Flow during Reperfusion and Post-conditioning

La Barck, Anthony J.[1], Docimo, Jennifer[2], and Merrill, Thomas L.* [3]
[1,3] Rowan University, [2] FocalCool, LLC
*Corresponding author: 201 Mullica Hill Road, Glassboro, NJ 08028, merrill@rowan.edu

Introduction

Reperfusion injury is caused by the rapid restoration of blood flow to oxygen-starved tissue. Animal studies show that intermittent periods of occlusion (also called post-conditioning) during reperfusion can limit tissue damage to vital organs such as the heart and brain[1]. These studies suggest that the protective effects of post-conditioning relate to the dynamics of blood flow[2, 3].

COMSOL is being used to model blood flow during post-conditioning. A 2-dimensional middle cerebral artery bifurcation will be used. The model will initially simulate pulsatile blood flow to predict pressure-flow relationships. A blockage in an artery will then be included to intermittently stop blood flow, simulating post-conditioning. This model will lay the foundation in understanding how fluid dynamics may impact post-conditioning protective mechanisms.

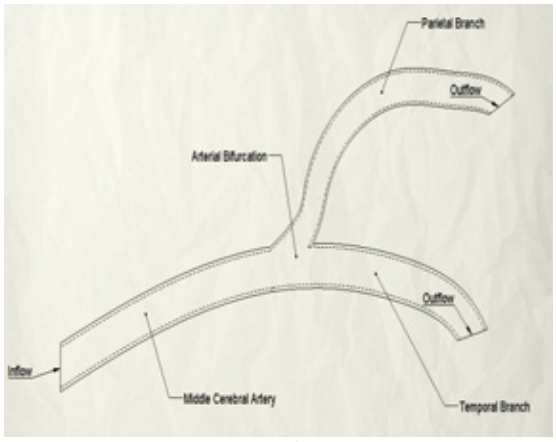

Figure 1 – Schematic of 2-D Artery Model

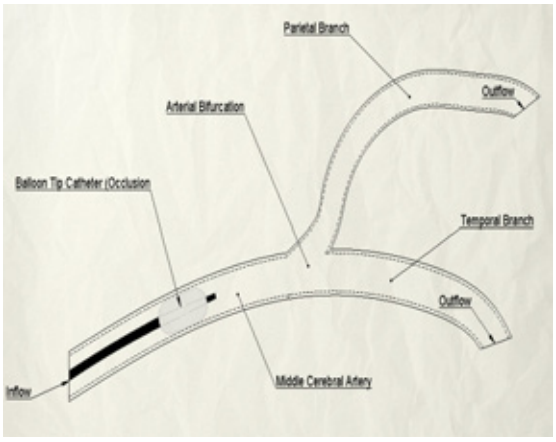

Figure 2 – 2-D Artery Model with Occlusion

Use of COMSOL Multiphysics

COMSOL Multiphysics will be used in this model in two different ways. First, a 2-D model of blood flow is necessary, so the Fluid Dynamics (Incompressible Navier-Stokes) module will be used to observe the pressure-flow fields in an arterial structure (Figure 1). The Structural Mechanics (Eigen frequency Analysis) module will then be used to simulate the elasticity of the artery geometry. These two modules combined will produce a pulsatile blood flow model. The model will then simulate post-conditioning by the insertion of an occlusion (Figure 2). A balloon tipped catheter will create the occlusion, inflating then deflating cyclically, thus varying the inlet velocity and pressure of blood over time.

Expected Results

Initially, we expect the velocity field of the model to be representative of Figure 3. Turbulent mixing will develop at the divider wall, producing high velocity and shear stresses. These areas indicate potential sites where tissue damage from oxidative stress and inflammation can occur. Turbulent wakes will also occur from post-conditioning near the occlusion site (Figure 4). We expect to observe that downstream turbulent blood flow is reduced as a result of post-conditioning.

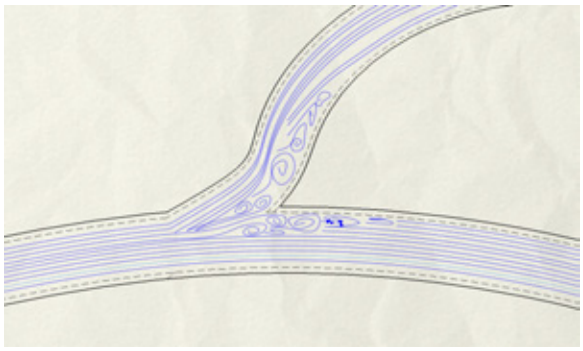

Figure 3 – Turbulence Effect at Divider Wall

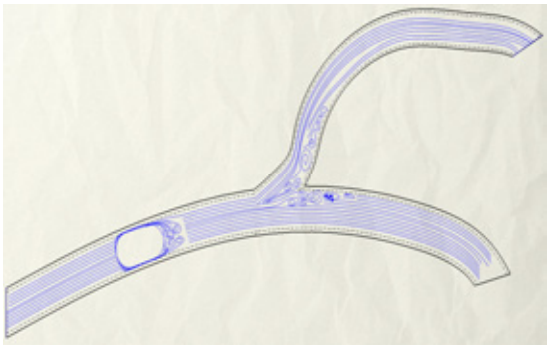

Figure 4 – Turbulence Effect at Occlusion Site

Conclusions

It is unclear what biological mechanisms aid tissue salvage during post-conditioning[4]. Understanding the fluid dynamics is reasonable initial step. Looking ahead, we will add species analysis to our model, measuring how fluid dynamics affects oxygen transport between blood and tissue using the Convection and Diffusion module in COMSOL.

To our knowledge, no study has examined how therapeutic hypothermia and post-conditioning combined can limit reperfusion injury. In the future, we plan to further develop our model to include cooled blood, simulating hypothermic conditions. COMSOL's General Heat Transfer module will help our model to show the thermal/fluid interaction of blood on oxygen transport as well.

References

1. Granfeldt A, Lefer DJ, Vinten-Johansen J. Protective ischaemia in patients: Preconditioning and postconditioning. Cardiovasc Res 2009 Jul 15;83(2):234-46.

2. Gao X, Ren C, Zhao H. Protective effects of ischemic postconditioning compared with gradual reperfusion or preconditioning. J Neurosci Res 2008 Aug 15;86(11):2505-11.

3. Xing B, Chen H, Zhang M, Zhao D, Jiang R, Liu X, Zhang S. Ischemic postconditioning inhibits apoptosis after focal cerebral ischemia/reperfusion injury in the rat. Stroke 2008 Aug;39(8):2362-9.

4. Yang D, Guo S, Zhang T, Li H. Hypothermia attenuates ischemia/reperfusion-induced endothelial cell apoptosis via alterations in apoptotic pathways and JNK signaling. FEBS Lett 2009 Jul 16.