A MULTIPHASIC FORMULATION FOR MODELING THE BLOOD FLOW-MEDIATED DILATION IN ARTERIES AND ARTERIOLES

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ABSTRACT

Resistance arteries and arterioles play a pivotal role in regulating peripheral vascular resistance within the microcirculation. They possess the remarkable ability to dynamically adjust their lumen diameter and thickness in response to various stimuli, including mechanical, chemical, and electrical signals. This responsiveness allows them to finely tune blood flow to meet the metabolic demands of tissues and organs, thereby playing a crucial role in maintaining overall cardiovascular homeostasis [1]. The physiology of vasodilation involves a combination of passive mechanical properties within the tissue layers and the active contractility of vascular smooth muscle cells (VSMCs) located in the Media layer. One of the primary active mechanisms of autoregulation is the myogenic response, also known as the Baylis effect [2]. This response entails the artery dilating in response to a rapid increase in transmural pressure, mediated by changes in VSMC tone. Additionally, alterations in hemodynamics trigger changes in shear stress, prompting the release of shear-induced endothelial-derived vasoactive metabolites, such as nitric oxide (NO) [3]. To better understand the biomechanics of microvessels in the presence of blood flow, we propose a multiphasic Fluid-Structure Interaction model that accounts for the two-phase blood flow (i.e., the blood cell-rich phase in the central core and the blood plasma in the annular part of the arteriole) and the vascular wall, which consists of the Media (also incorporates the Intima) and Adventitia layers. A thixotropic elasto-viscoplastic (TEVP) model is adopted for the rheological modeling of the blood core [4], whereas blood plasma is presented as a viscoelastic material. The passive response of both solid layers is described by the Holzapfel–Gasser–Ogden model [5], which accounts for the anisotropic nature of the material due to the collagen fibers. The Media also features an active contractile apparatus, which is regulated by the intercellular calcium concentration, $[Ca^{2+}]_i$, and myosin phosphorylation. An increase in $[Ca^{2+}]_i$ intensifies the generated active forces, which in turn diminishes arteriole expansion. In addition, we include the regulation of VSMC relaxation through the NO - phosphodiesterase-catalysed (cGMP) pathway [6]. To solve the resulting FSI problem, we adopt the Finite Element Method (FEM). To calibrate the parameters of the model, we use experimental reports that are mainly based on human individuals. Thus, the proposed model will give a better understanding of autoregulation in human vasculature.

KEYWORDS: Resistance Arteries, Active Response, Myogenic Tone, Finite Element Method

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