

Biomechanical Model of Brain Vasculature

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Introduction

The vascular compartment plays a very important role in the response observed during a BOLD (blood oxygenation level-dependent) contrast experiment. Principally, it contains the contrast agent that makes us sensitive to changes in metabolic demand (which is related to neuronal activity). As a consequence of changes in the metabolic demand, dynamic changes take place in the vascular system in order to satisfy the new demand levels of increased brain activity. This complicates the dynamics of the observed signal in magnetic resonance imaging since the amount of tracer in the compartment will depend not only on the demand, but also on the dynamics of the vascular compartment. The objective of this work is to present a relatively simple biomechanical model of brain microvasculature and compare its behavior to various situations encountered in typical brain function experiments.

Methods

The structure of the vascular system is extremely complicated. Models that attempt to describe blood flow in the vascular system make use of numerous assumptions (and this is no exception). Suppose a portion of the vascular tree (in the order of arterioles, capillaries, venules) can be modeled as a single, elastic, cylindrical tube where blood (assumed to be a Newtonian fluid) is flowing in laminar fashion. Flow in a tube is maintained by a pressure gradient, and the relationship between flow rate and pressure is given by Poiseuille's formula,

$$dP / dx = - 8 \mu F / \pi r_0^4 \quad [1]$$

where P is the pressure, F is the flow rate, and r_0 is the radius of the tube if it were to be uniform. Since the tube is elastic we expect it to distend more in higher pressure areas compared to lower pressure areas (note that this pressure drop is across the vessel wall), so that the tube radius is a function of axial position. If we assume the vessel wall to be thin compared to the radius, and for it to behave as a Hookean elastic material, then an expression for the radius as a function of position can be obtained through the computation of the average radial stress and strain at the vessel wall (see [1] for details).

$$r(x) / r_0 = 1 / (1 - r_0 p(x) / E h) \quad [2]$$

In the above expression $r(x)$ is the radius as a function of position (due to pressure p across the wall), E is the elasticity of the vessel wall and h is the wall thickness. To model the viscoelastic nature of the vessel wall, its dynamics were simulated to resemble those of the changes in blood flow (a first-order linear system), but with a temporal delay and a longer time constant (see [2] for details), maintaining the validity of the expressions above over a range of values. To compute the flow out of the tube, the following expression was used,

$$dV / dt = F_{in} - F_{out} \quad [3]$$

Throughout these preliminary simulations equation 1 was used to calculate the corresponding pressure gradient (flow-rate drives the system) and equation 2 is used to calculate the geometry of the tube. The following parameters were used: $r_0 = 100 \mu\text{m}$, $h = 0.15 r_0$, $P_{end} = 10 \text{ mmHg}$, $x = 1 \text{ cm}$. The flow rate into the tube is assumed to follow first-order dynamics with a baseline velocity of 0.5 cm/s and increases by 50% with neuronal activity.

Results

Figure 1 presents the in-flow and volume changes obtained in the vascular model when the elasticity remains constant ($E = 2 \times 10^4 \text{ N/m}^2$). The outcome of keeping a constant elasticity yields similar temporal dynamics for both volume and in-flow (the change in amplitude is related to the magnitude of the elasticity parameter). Figure 2 presents the in-flow, out-flow and volume changes when the elasticity follows a more viscoelastic behavior ($E = 8 \times 10^4 \text{ N/m}^2$). The outcome of viscoelastic behavior yields a slower decay in the blood volume, a result reported in [3] (although the time constant produced here appears somewhat shorter). Note that since the temporal pressure changes affect the geometry of the vessel instantaneously, initially there is a brief period where the out-flow decreases below baseline and a larger increase is produced after the pressure (which is proportional to in-flow) begins returning to baseline.

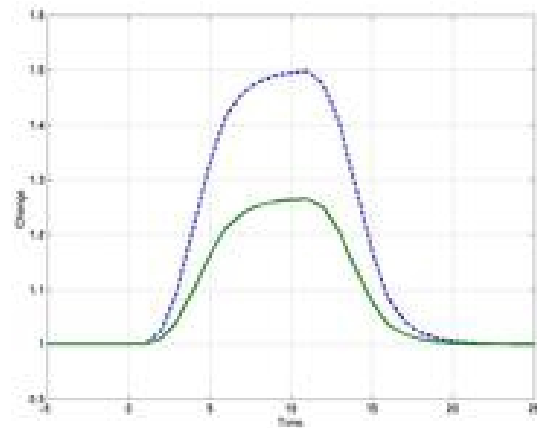


Figure 1: Changes of blood volume (solid) and in-flow (dashed) over baseline for a vascular compartment with constant elastic properties. The compartment is driven by the in-flow which follows first-order dynamics.

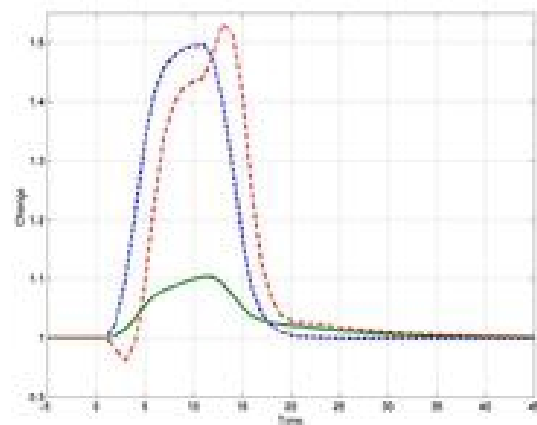


Figure 2: Changes of blood volume (solid), in-flow (dashed) and out-flow (dash-dot) over baseline for a vascular compartment exhibiting viscoelastic properties.

Discussion

The model developed here is able to describe some of the observed dynamic behavior of flow and volume reported in the literature through the elastic nature of the compartment. It is the interplay between the dynamics of at least blood flow, volume, and the amount of deoxy-hemoglobin (which is related to the metabolic supply/demand of oxygen), that provide the dynamics observed in the BOLD response. Actually, the elevated volume response (Figure 2) is hypothesized to be the source of the post-undershoot observed in BOLD and also the source of some observed non-linearities. These new models have the potential to accurately describe the underlying physiological changes (for better quantitation) as well as location in the vascular tree (for better localization). Further development of such models is currently under investigation.

References

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