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Flow and anastomosis in vascular networks

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HIGHLIGHTS

- ▶ We analyze the effect that anastomosis has on blood flow.
- ▶ Details on how anastomosis affects flow are strongly dependent on network geometry.
- ▶ Flow in a network with anastomosis is determined by its local structure.
- ▶ Our model is able to interpret the anastomotic effect for tree-like in vivo vasculature networks.
- ▶ Results are robust to the consideration of the myogenic effect.

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ABSTRACT

We analyze the effect that the geometrical place of anastomosis in the circulatory tree has on blood flow. We introduce an idealized model that consists of a symmetric network for the arterial and venous vascular trees. We consider that the network contains a viscoelastic fluid with the rheological characteristics of blood, and analyze the network hydrodynamic response to a time-dependent periodic pressure gradient. This response is a measurement of the resistance to flow: the larger the response, the smaller the resistance to flow. We find that for networks whose vessels have the same radius and length, the outer the level of the branching tree in which anastomosis occurs, the larger the network response. Moreover, when anastomosis is incorporated in the form of bypasses that bridge vessels at different bifurcation levels, the further apart are the levels bridged by the bypass, the larger the response is. Furthermore, we apply the model to the available information for the dog circulatory system and find that the effect that anastomosis causes at different bifurcation levels is strongly determined by the structure of the underlying network without anastomosis. We rationalize our results by introducing two idealized models and approximated analytical expressions that allow us to argue that, to a large extent, the response of the network with anastomosis is determined locally. We have also considered the influence of the myogenic effect. This one has a large quantitative impact on the network response. However, the qualitative behavior of the network response with anastomosis is the same with or without consideration of the myogenic effect. That is, it depends on the structure that the underlying vessel network has in a small neighborhood around the place where anastomosis occurs. This implies that whenever there is an underlying tree-like network in an in vivo vasculature, our model is able to interpret the anastomotic effect.

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1. Introduction

Anastomosis in the context of blood vasculature refers to the bridging between vessels. It leads to the formation of loops. In

healthy mammals anastomosis exists at many levels in the circulatory tree. Nevertheless, it becomes more and more frequent as one goes down towards the inner vessels (Secomb and Pries, 2011).

Anastomosis is particularly relevant in vascular diseases. Patients with ischemic vascular diseases tend to develop collateral vessels that bypass major arterial obstructions. This may explain why some patients have no – or minimal – symptoms of ischemia (Helisch and Schaper, 2003). For instance, collateral

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arteries are a common, adaptive response, in patients with coronary heart disease (CHD). Collateral arteries may prevent myocardial ischemia in healthy subjects and in patients with CHD (Berry et al., 2007). An increase in the number of anastomotic vessels also occurs in various pathologies associated with micro-neovascularization, such as diabetic retinopathy (Ashton, 1963) or the Moyamoya disease (Czabanka et al., 2003).

Collateral circulation constitutes an important aspect of cerebrovascular circulation. A highly interconnected network of arterioles overlies mammalian cortex to route blood to the cortical mantle. Such cortical arteriolar network, presents a high degree of redundancy, supports collateral flow, and plays a pivotal role in the pathophysiology of cerebral ischemia (Schaffer et al., 2006; Brozici et al., 2003; Liebeskind, 2003). Experimentally, it has been found that efficient collateral blood supply, delays ischemic injury after middle cerebral artery occlusion (Maeda et al., 1999). Understanding the effect of collateral anastomoses may explain differences in clinical outcome, enable risk stratification for individual subjects, and expand treatment options for acute stroke and chronic cerebrovascular disorders (Liebeskind, 2003).

In the above mentioned cases, the loop structure created by anastomosis has the effect of providing robustness to a network by means of making it redundant. That is, by assuring the preservation of blood flow and maintaining the delivery of nutrients to a tissue despite the presence of local obstructions (Blinder et al., 2010; Goldman and Popel, 2000; Axelsson et al., 1997). The presence of anastomosis is particularly relevant around cancer tumors. These ones, often promote the creation of a more profuse vascular network, mainly at the arterioles and capillary levels. At these levels, the network is a complex structure of interconnected loops (Pries et al., 2010; Less et al., 1991). A local enhancement of flow could be therefore speculated, since tumors require nutrients and oxygenated blood to grow. In other cases, tumors kidnap a large artery that bypasses many levels and directly feeds the tumor (Yamada et al., 2004). In these cases, anastomosis might also be suspected to have an effect of flow enhancement.

In this paper we study the effect of anastomosis from a hydrodynamic point of view. We demonstrate that generally speaking, anastomosis causes an enhancement of flow in the network. However, details on how the geometrical place of the loops affects the flow, depend strongly on the network geometry. That is, even for a very idealized network – consisting of loops embedded in a tree-like branched structure – the geometrical place and the size of the loops caused by anastomosis can dramatically enhance the flow, have a minimal effect on it, or even be negligible. The effect of anastomosis depends on the sequence of radii and lengths of the vessels forming the underlying network.

Our model allows for the study of vessel networks at different scales. It can be applied to the circulatory system of a mammal or to certain microvascular networks. The topology of networks in microcirculation is very complex. However, there are tissues for which microcirculation occurs in a diverging tree of arterioles which feed the capillaries. This is the case for the mesentery (Secomb and Pries, 2011), and for the pial network of the middle cerebral artery (Blinder et al., 2010), at least at certain spatial scales. Our model can be applied in such scenarios.

The outline of the paper is as follows: in Section 2 we introduce the geometrical characteristics of a tree-like network in the absence of anastomosis. In Section 3 we describe the hydrodynamic model that we use for blood and show how flow and pressure gradient are related through a dynamic response function for the vessel network. This one in turn, is written in terms of the dynamic response function of the individual vessels

composing the network. Section 4 introduces the basic model of anastomosis through an electrical analogy. Section 5 incorporates anastomosis in a network when it connects different branches of the tree at the same bifurcation level. That is, we study the bridging between two parallel arteries (or veins) by an anastomotic vessel. We find that for underlying tree-like networks whose vessels have the same radius and length at all levels, the outer is the level in which anastomosis occurs, the larger the network response is. Section 6 incorporates anastomosis in a network in the form of bypasses that bridge vessels at different bifurcation levels. We find that for networks whose vessels have the same radius and length at all levels, the network response is larger the further are the levels bridged by the bypass. In Section 7 we apply the model to the available information for the dog circulatory system. We find that the effect that anastomosis causes at different bifurcation levels is strongly determined by the structure of the underlying network without anastomosis. We therefore rationalize the response dependence on the network geometry by introducing two idealized models in Section 8. We approximate the values of the dog circulatory system in two different ways. These idealized models allow us to understand mathematically the relevant quantities of the underlying network that determine the dynamic network response when anastomosis is present. In Section 9 we present a mathematical analysis and a discussion of our findings. We find that, to a large extent, the dynamic response is a local effect. Notably, our results say that whenever there is a jump in resistances – in the underlying network – there will be a jump in the network response when anastomosis is considered. Section 10 explores how the network response changes when the myogenic effect is considered at the level of arterioles. Finally, Section 11 presents our conclusions. We claim that, whenever there is an underlying tree-like network in an in vivo vasculature, our model could be used to interpret the anastomotic effect. This is particularly relevant since recent progresses on high resolution microscopy (Vakoc et al., 2009; Jung et al., 2010) have enabled a better characterization of vasculature in general and of vasculature associated to solid tumors in particular, thus contributing to improve the predictability of cancer progression. The concurrence of these sophisticated image-tracking systems and physical models as the one presented here, might provide a relevant tool to perform early prediction and diagnosis that could help to define the proper therapies to be followed. In this context, our model is capable of interpreting the effect of anastomosis in a particular kind of vessel networks. That is, the model could quantify the benefit that a particular solid tumor draws from its local vascular network.

2. Geometrical model for the network without anastomosis

Recently a network model has been introduced in order to study viscoelastic flow through a network of tubes (Flores et al., 2010). The model consists of a tree-like network in which vessels bifurcate always from one into two identical vessels giving rise to identical branches of the network. At each bifurcation step, one allows for the possibility of changes in the cross-sectional area and in the length of the vessels. In this way each level (or generation) of the network is constituted by vessels with the same length and cross section. Therefore, the network is characterized by its number of levels and by the cross section and length of the vessels at each level. A symmetrical network for the return flow is considered, in such a way that the network closes into itself. This is illustrated in Fig. 1, where it is also shown that segments at the same distance from the main branch – belonging to the same level – are labeled with the same index. We refer to outer levels of the network as the ones that are closer to the main

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