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Heart rate, arterial distensibility, and optimal performance of the arterial tree

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ABSTRACT

In this study we explore the ability of a previously developed model of pulsatile flow for explaining the observed reduction of arterial distensibility with heart rate. The parameters relevant for the analysis are arterial wall distensibility together with permeability and reflection coefficients of the end capillaries. A non-specific artery and the ensemble of tissues supplied by that artery were considered in the model. The blood current within that artery was equalized to the sum of all micro currents in the tissues supplied by that artery. A formula emerged that relates changes in arterial distensibility with heart rate, and also with some particular aspects of microcirculation. Then, that formula was tested with data of distensibilities of the radial and carotid arteries observed at the heart rates of 63, 90, and 110 b.p.m. The formula correctly predicted the trend of decreased distensibility with heart rate for both arteries. Moreover, due to the fact that the carotid artery supplies the brain, and because the Blood–Brain barrier is highly restrictive to colloids in the blood, for the carotid artery the formula predicted a less marked decrease in distensibility than in the case of the radial artery feeding muscle tissue, which has a greater permeability to colloids, a trend that was confirmed by data. It was found that reduction of arterial distensibility with heart rate was greater in arteries that supply end capillaries with high permeability and low reflection coefficients.

1. Introduction

It has been long recognized that pulsatile blood flow performs the best than continuous flow because it induces both lower total peripheral resistance and mean arterial pressure (Mavroudis, 1978), and also better blood perfusion (Taylor et al., 1982). The distensibility coefficient of the vessel wall is defined as $\beta = (2/D)(dD/dP)$ where P is pressure within the vessel, and D is vessel diameter. Henceforth the term distensibility is used to mean distensibility coefficient.

On the other hand, a recent model of pulsatile flow predicts that if distensibility is kept constant, arterial impedance must decrease with pulse frequency (Silva and Reis, 2014). Actually, if pulse frequency (heart rate) and therefore the blood current increases in response to needs of organs in the body, then it makes sense that arterial impedance is reduced to ease the access of blood. On the other hand, if arterial impedance decreases with distensibility (Silva and Reis, 2014) one would expect that increased blood current would lead to decreased arterial impedance with heart rate.

However, many studies have shown that, in humans, arterial distensibility varies inversely with heart rate (Asmar et al., 1996; Liang et al., 1999; Giannattasio et al., 2003; Pitcher et al., 2010; Koskinen et al., 2011; Chrysohoou et al., 2013). The same effect has been observed in rats (Mircoli et al., 1999). Though, according to the model above referred (Silva and Reis, 2014) increased heart rate leads to lower arterial impedance, the observed increased arterial stiffness with heart rate actually increases impedance. Apparently, this behavior does not make sense because easing blood flow is sought to be the objective of the circulatory system.

An extended search in the pertinent literature also revealed some studies that concluded that arterial stiffness was not affected by heart rate (Wilkinson et al., 2002; Aizawa et al., 2009). However, these two studies have some particularities: in (Wilkinson et al., 2002) arterial stiffness changes with heart rate were indirectly estimated through the augmentation index calculated from the blood pressure waveform, while in (Aizawa et al., 2009) arterial stiffness was "determined before and 10 min after graded arm-cycling exercise".

On the other hand, some other studies (Kingwell et al., 1997; Currie et al., 2009; Jae-Bin et al., 2013) found that whole body arterial

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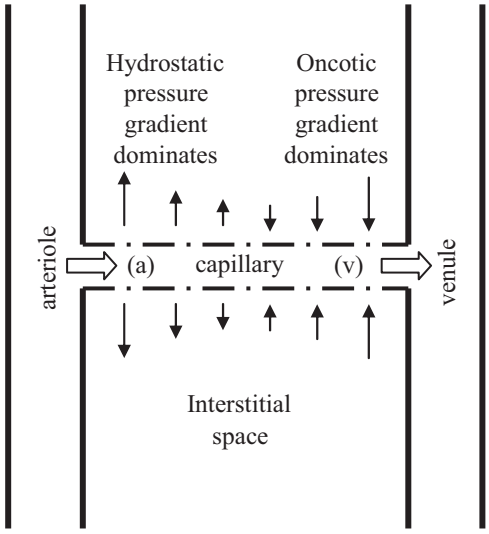

Fig. 1. Microcirculation: blood enters the capillary at the arteriolar end (a), water, salts and colloids are driven into the interstitial space by the capillary gradient, and return into the capillary driven by the oncotic gradient at the venular end (v).

compliance (WBAC) and then arterial distensibility was increased after cycling and treadmill exercise with relation to the values prior to exercise. This is a different result that compares arterial distensibility before and after the period in which heart rate is increased.

The decrease of arterial distensibility with heart rate is somewhat counter-intuitive, and challenges the current paradigm of human physiology. From the physiological point of view, no explanation has yet been presented. In the following we offer an explanation based on the assumption of optimal hemodynamic performance of the arterial tree, and show that increase in arterial stiffness with heart rate may be understood as the adjustment of the arterial tree on the way for global optimization of its performance. For this purpose we first take a closer look to microcirculation in the capillaries forming the end of the arterial tree.

2. Microcirculation and Starling forces

Blood is transported downstream in the arterial tree until it reaches the end capillaries that bridge arteriole and venule ends, which deliver it to the interstitial fluid that bathes every cell (see Fig. 1). Capillaries have opening of various widths according to the tissue to which blood is supplied. Many capillaries may turn impermeable to the bigger colloids in the blood, namely the proteins, therefore regulating their delivery to the interstitial fluid. Special classes of proteins called albumins constitute about 50% of human plasma protein and are very important as carriers of hydrophobic substances (e.g. lipid soluble hormones, bile salts, free fatty acids) (Farrugia, 2010). Water and other small molecules are generally free to pass through capillary openings.

In this way, filtration occurs along the capillary driven by the difference in hydrostatic pressure $\Delta P_{ci} = P_c - P_i$ between the capillary (P_c) and the interstitial space (P_i), therefore increasing the concentration of colloids that are not allowed to pass into the interstitial fluid. As the result a colloid osmotic pressure – oncotic pressure difference $\Delta\Pi_{ci} = \Pi_c - \Pi_i$ – develops between the interstitial fluid and the blood within the capillaries, which opposes the pressure gradient that drives the blood from the capillaries to the interstitial space (i.e. the space between cells that is bathed by the interstitial fluid). Within the capillary, hydrostatic pressure decreases from P_a (at the end of the arteriole) to P_v (at the beginning of the venule), $\Delta P_c = P_a - P_v$.

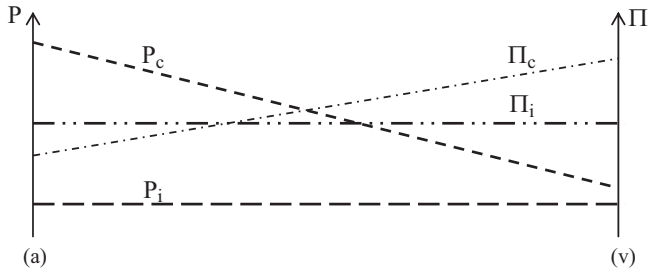

Fig. 2. Variation of capillary hydrostatic pressure (P_c), interstitial hydrostatic pressure (P_i), capillary oncotic pressure (Π_c), and interstitial oncotic pressure (Π_i), between arteriolar (a) and venular (v) ends of the capillary. Π_c increases within the capillary due to loss of fluid to the interstitial space. P_i and Π_i are constant in the interstitial space (Taylor, 1981; Seifter et al., 2005).

Hence, the blood leaving the end of the arteriole splits into two currents: one of them flows within the interstitial space; another one flows within the capillary. Both currents merge together at the entrance of the venula.

The current flowing into the interstitial space may be described by Starling's equation (Taylor, 1981; Seifter et al., 2005)

$$i_{ci} = K_{ci}\Delta P_{ci} - \sigma_{ci}\Delta\Pi_{ci} \tag{1}$$

where i_{ci} is the net current between the capillary and the interstitial space, K_{ci} and σ_{ci} respectively stand for filtration coefficient and reflection coefficient of the capillary section in which i_{ci} exists, Π_c and Π_i represent oncotic pressure of the colloids in the capillary and the interstitial space, respectively. Though in the literature, K_{ci} and σ_{ci} are termed "coefficients", in fact they represent conductances that are proportional to the extension of the capillary in which exchange of fluids occur. The reflection coefficient is null for a capillary wall permeable to all colloids in the blood. The first term in the r.h.s of Eq. (1) represents the current from the capillary to the interstitial space while the second one stands for the current from the interstitial space onto the capillary that is driven by oncotic pressure gradient. At the arteriolar end the hydrostatic driven current dominates, hence there is a net influx to the interstitial space, while the opposite occurs at the venular end where a net outflow towards the capillary (see Fig. 2).

The current within the capillary is driven by the hydrostatic pressure difference $\Delta P_c = P_a - P_v$ and is given by:

$$i_c = \Delta P_c / Z_c \tag{2}$$

where Z_c stands for capillary impedance. In this way, the total current leaving the arteriolar end is given by

$$i = i_i + i_c = K_{ci}\Delta P_{ci} - \sigma_{ci}\Delta\Pi_{ci} + \Delta P_c / Z_c \tag{3}$$

On the other hand, the total current entering the venular end is composed of the current from the interstitial space into the capillary:

$$i_v = i_{ic} + i_c = K_{ic}(\Delta P_c - \Delta P_{ci}) + \sigma_{ic}\Delta\Pi_{ci} + \Delta P_c / Z_c \tag{4}$$

where $K_{ic} \neq K_{ci}$ and $\sigma_{ic} \neq \sigma_{ci}$ respectively stand for filtration coefficient and reflection coefficient of the capillary section in which i_{ci} exists. Note that here we have considered the general case in which $K_{ci} \neq K_{ic}$ and $\sigma_{ci} \neq \sigma_{ic}$. Additionally, due to mass conservation for the steady state one has

$$i = i_v + i_{lymph} \tag{5}$$

where i_{lymph} stands for the rate at which lymph is drained from the interstitial space to the lymphatic circulation. Therefore, from Eqs. (3)–(5) one obtains

$$i_{lymph} = (K_{ic} + K_{ci})\Delta P_{ci} - K_{ic}\Delta P_c - (\sigma_{ci} + \sigma_{ic})\Delta\Pi_{ci} \tag{6}$$