

Review

Large variations in absolute wall shear stress levels within one species and between species

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Abstract

Wall shear stress (WSS), the frictional force between blood and endothelium, is an important determinant of vascular function. It is generally assumed that WSS remains constant at a reference value of 15 dyn/cm². In a study of small rodents, we realized that this assumption could not be valid. This review presents an overview of recent studies in large and small animals where shear stress was measured, derived from velocity measurements or otherwise, in large vessels.

The data show that large variations exist within a single species (human: variation of 2–16 N/m²). Moreover, when we compared different species at the same location within the arterial tree, an inverse relationship between animal size and wall shear stress was noted. When we related WSS to diameter, a unique relationship was derived for all species studied.

This relationship could not be described by the well-known r^3 law of Murray, but by the r^2 law introduced by Zamir et al. in 1972.

In summary, by comparing data from the literature, we have shown that: (i) the assumption of a physiological WSS level of ~15 dyn/cm² for all straight vessels in the arterial tree is incorrect; (ii) WSS is not constant throughout the vascular tree; (iii) WSS varies between species; (iv) WSS is inversely related to the vessel diameter. These data support an “ r^2 law” rather than Murray’s r^3 law for the larger vessels in the arterial tree. © 2006 Elsevier Ireland Ltd. All rights reserved.

Keywords: Wall shear stress; Animal; Clinical; Patients; Mice; Mouse; Rat; Rabbit; Dog; In vivo; Human; Doppler

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1. Introduction

Wall shear stress (WSS), the frictional force between blood and endothelium, is an important determinant of endothelial cell function, gene expression, and structure. Indeed, a variety of studies provided evidence that WSS has to be maintained between certain limits in order to maintain vascular haemostasis. WSS is actively maintained within limits during intrauterine growth, during the neonatal period and early childhood, and during exercise in the adult. Inappropriate values of WSS have been associated with maladaptive growth, patent ductus arteriosus, congenital malformations of the heart and atherosclerosis [1–3]. Indeed, when WSS is reduced by 30% in vivo in ApoE mice, the expression of several atherogenic genes is induced, which triggers the development of large atherosclerotic lesions [4]. To avoid these conditions, the endothelium in the arterial system should be responsive to WSS within a narrow range of values that are considered “normal”.

At present, it remains unclear whether the endothelium throughout the arterial system is primed with the same range of WSS values. Currently, researchers in the field often assume mean WSS levels of $\sim 15 \text{ dyn/cm}^2$ ($1 \text{ dyne/cm}^2 = 0.1 \text{ N/m}^2$) as acceptable, because it represents the average WSS values over the cardiac cycle of the large straight arteries experiencing steady laminar flow. This is based on studies in patients and animal models, which provide evidence that WSS actively influences vessel wall remodeling [5–8]. This compensatory response mediated by the endothelium aims at the maintenance of a WSS magnitude of approximately $15\text{--}20 \text{ dyn/cm}^2$. Partially based on this notion, it is also commonly assumed that this acceptable range of WSS is rather constant throughout the vascular system [9–14].

Another argument for a constant WSS value of $\sim 15 \text{ dyn/cm}^2$ at different locations in the arterial system is derived from the principle of minimal work for the cardiovascular system as proposed by Murray [15]. He stated that the total energy to drive the blood and to maintain blood volume is minimized in the arterial system. Deducted from this principle is Murray's law [16], which states that the cube of the radius of the mother vessel equals the sum of cubes of the radii of the daughter vessels. While this optimization principle predicts a constant WSS throughout the vascular system [17], a number of recent publications show a broad range in the actual mean WSS levels that could be measured in the different types of arteries in humans [18–21]. Flow measurements in animal models also show differences in WSS levels between species [22–25]. These data therefore indicate that

WSS varies with the location across the cardiovascular system within one species, and that there are cross-species differences. In spite of this, a paucity of data exists which compare WSS at different anatomical locations [21,26] or between species in one type of vessel. Until now, no reviews are available which summarize the separate WSS values found in literature to provide an adequate overview about this subject.

In this review, we present evidence from literature that supports the concept that WSS levels are not identical throughout the vascular tree. We also provide evidence that WSS levels differ between species. The interpretation of these data will be discussed in relation to a modification of Murray's law. Acceptance of this concept would have significant implications for further WSS research, as the importance of the effects of anatomical localization of the studied endothelial cells and the species from which they are derived is often overlooked in current studies.

2. Variations in mean WSS in the arterial system

2.1. Human individuals

Vascular disease affects the vascular remodeling capacities of arteries, which could result in an alteration of the mean WSS level. Accordingly, only studies performed in healthy human subjects are included in this review.

In Table 1A, the WSS values in different types of arteries derived from experimental data of several investigators are summarized [18–21,26–39]. All WSS values are derived from in vivo measurements in conscious human subjects, by applying either ultrasound or MRI techniques. All values are stationary WSS values or recalculated to become stationary WSS values, and therefore the time dependence of the WSS measurements are not taken into account. Excellent studies and reviews are present on this topic [40–42]. For more information on the studies that we have included in Table 1A concerning background, and details in methods, we refer to Table 1B. It should be mentioned that the WSS values in Table 1A are obtained from studies testing different hypotheses (e.g. the effect of smoking or exercise on blood flow), with different study designs testing specific parameters. However, only WSS values obtained from the control (non-treatment) groups were included in this review. Selection criteria for the incorporated studies are as followed: subjects of the selected control group are younger than 40 years old, do not show clinical manifestation of cardiovascular disease, are non-smokers and do not receive any prescribed

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