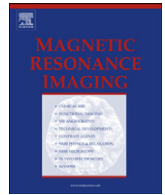




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Original contribution

Phase-contrast magnetic resonance imaging for the evaluation of wall shear stress in the common carotid artery of a spontaneously hypertensive rat model at 7 T: Location-specific change, regional distribution along the vascular circumference, and reproducibility analysis[☆]

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ABSTRACT

Purpose: To measure wall shear stress (WSS) in the common carotid arteries (CCA) of a spontaneously hypertensive rat (SHR) model and a normotensive Wistar Kyoto rat (WKY) model by 2D phase-contrast magnetic resonance imaging (PC-MRI).

Materials and methods: PC-MRI was performed on 7 SHR and 7 WKY at ages of 4 and 7 months at a 7 T scanner. Images in the middle CCA (CCA_{mid}) and in the bifurcation of CCA (CCA_{bifur}) were acquired. The WSS values for differentiating characteristics between two models were calculated. Further, its location-specific change, regional distribution along the CCA circumference, and the reproducibility were evaluated.

Results: In the 4-month-old rats, SHR showed lower temporal averaged WSS (WSS_{avg}) and peak systolic WSS (WSS_s) in the CCA_{bifur} in comparison with WKY (WSS_{avg}: 0.95 ± 0.18 vs. 1.30 ± 0.36 N/m² ($P < 0.01$); WSS_s: 1.68 ± 0.70 vs. 3.22 ± 2.49 N/m² ($P < 0.05$)). We observed the same trends in the 7-month-old rats. In the SHR model, the WSS_{avg} was lower in the CCA_{bifur} than in the CCA_{mid}. The regional distribution of WSS_{avg} along the circumference of CCA showed lower values in WKY, particularly in posterior segments of CCA_{bifur}. The intra-observer, intra-scan and inter-scan reproducibility was acceptable and the disagreements were ranged from -0.05 to 0.06 N/m².

Conclusion: This study evaluated WSS in SHR and WKY models by 2D PC-MRI. High reproducibility analyses further indicated the reliability of measurements of WSS in the CCA of SHR and WKY models using PC-MRI at 7 T.

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1. Introduction

Atherosclerosis is a systemic disease and a leading cause of death in developing countries [1]. The risk factors for atherosclerosis include hypertension, cigarette smoking, and hypercholesterolemia. Within a vessel, certain regions are prone to atherosclerosis

development, such as bifurcations, branches, and the inner walls of curvatures. Several studies have shown an association between the hemodynamic parameter of wall shear stress (WSS) and the pathogenesis of atherosclerosis [2–4]. The WSS is the frictional force exerted by the circulating blood on the endothelium of the vessel walls. If endothelial cells are exposed to environments with higher WSS, they form flat elongated shapes. This morphology reduces the accumulation of cholesterol and thus the risk of developing atherosclerosis [5]. In contrast, if the endothelial cells are exposed to a relatively lower WSS environment, they form rounder shapes, which are associated with increased intercellular permeability [6]. If cholesterol compounds accumulate in the more permeable arterial walls, fibrous atheromatous plaques can form

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easily on the vessel walls [6]. Therefore, lower WSS values accelerate the development of atherosclerosis in endothelial cells [2–4,6].

With the advantages of excellent resolution and noninvasion, phase-contrast magnetic resonance imaging (PC-MRI) has been a useful tool to assess blood flow in vivo for decades. In addition, the hemodynamic parameters, such as pulse wave velocity (PWV) and WSS, from PC-MRI for animal models have extended the field of clinical applications along with new technique developments [7]. Zhao et al. have demonstrated higher PWV and lower WSS in aorta of ApoE deficient mice model [8]. In terms of WSS in CCA, the application of PC-MRI in the mice model of stable and vulnerable carotid artery atherosclerotic plaques has been presented by van Bochove et al. They concluded that the WSS measured from the common carotid artery (CCA) with atherosclerotic plaques was significantly lower when compared to that from the normal contralateral side in the mice model [9].

Previous studies have established the spontaneously hypertensive rat (SHR), a rat with native hypertension, as a suitable animal model for investigations on atherosclerosis [10,11]. Using an invasive scheme of aortic cannulas connected to a pressure recording system, Marque et al. showed that the old SHR model was associated with marked stiffening of the aortic wall and a lower WSS with increasing age [12]. Based on a ball flowmeter, Huang et al. showed that SHR exhibited the reduced WSS in the cremasteric arterioles when compared to the normotensive Wistar Kyoto rat (WKY) [13]. Furthermore, in human studies, a number of researches have demonstrated that the WSS of carotid artery was significantly lower in hypertensive patients than normotensive controls [14,15]. In combination with computational fluid dynamics (CFD), Greve et al. have studied flow velocity and WSS in the infrarenal aorta of mice, rats, and human and reported the allometric equation for WSS [16]. Since the WSS may vary from species to species and even not identical throughout the vascular system [17], these evidences raise an interest in exploring the characteristics of WSS in CCA in SHR and WKY models via PC-MRI technique.

In this study, we aim to measure cross-sectional flow-related parameters and WSS in the CCA of an SHR model and a WKY model by 2D PC-MRI for the differentiation of WSS in the CCA in two models at a 7 T MR scanner. We also calculated the regional distribution of the WSS along the circumference of the vascular lumen in CCA to identify local abnormalities in the WSS. Besides, the potential dependences of WSS on different locations in CCA as well as the reproducibility of measurements were investigated.

2. Material and methods

2.1. Animal preparation

This study was approved by the local institutional animal ethics committee. Two-month-old SHR ($n = 7$, number of investigated CCA = 14, including both right and left CCAs) and WKY ($n = 7$, number of investigated CCA = 14, including both right and left CCAs) were maintained under standard conditions (lights on from 6 AM to 6 PM). A standard rodent diet and water ad libitum was provided to the same 14 rats until 4 months and 7 months of age when the MRI examinations were performed. The mean weights of the SHR and WKY at the age of 4 months were 332 ± 14 g and 335 ± 13 g, respectively. During MRI examinations, the animals were anesthetized with isoflurane through a nose cone. The anesthesia dose was 5% for initial induction and 1.5% for maintaining the heart rates of rats within the range of 56–65 bpm. The respiratory monitoring and electrocardiography (ECG) gating signals were recorded simultaneously during scanning.

2.2. MRI acquisition

All MRI examinations were conducted on a 7 T animal MRI scanner (ClinScan 70/30, Bruker, Germany) with a gradient strength

of 630 mT/m. A 2D time-of-flight (TOF) sequence was performed for noncontrast enhanced angiography, which provided anatomical information on the CCA (TR/TE = 20/3.46 ms, field of view (FOV) = 40×40 mm, matrix size = 256×256 , slice thickness = 1.2 mm, number of average = 1). Fig. 1(a) and (b) show the reconstructed sagittal and coronal views of the CCA of a WKY rat. A 2D single slice time-resolved PC-MRI with fast low angle shot (FLASH) sequence was performed for the measurement of flow with parameters of TR/TE = 15.75/4.51 ms (minimum TE), flip angle = 30° , FOV = 40×40 mm, matrix size = 256×256 , slice thickness = 2 mm, number of averages = 10, and unidirectional velocity encoding (VENC) = 1.5 m/s in the through-plane direction. The temporal resolution was around 31.5 ms. This VENC value was chosen to avoid phase wrap in WKY and SHR models. To ensure synchronicity with the heartbeat, a prospective ECG gating technique was used to acquire flow information within 90% of the R-R intervals. To investigate the location-specific changes in the flow velocity and WSS along the CCA, two imaging planes (approximately 8 mm apart) perpendicular to the long axis of blood flow direction were prescribed for the measurement of the through-plane flow in the middle CCA (CCA_{mid}) and the bifurcation of the CCA (CCA_{bifur}), as shown in Fig. 1. The slice position for the CCA_{bifur} was around 2 mm proximal to the bifurcation of CCA (Fig. 1(a)). After defining the slice position of CCA_{bifur}, the slice position of CCA_{mid} was placed at the middle between CCA_{bifur} and the arch of the subclavian artery.

To evaluate the reproducibility of measurements of blood flow velocity and WSS, intra-scan (in total 28 scans, number of investigated CCA = 56) and inter-scan (in total 28 scans, number of investigated CCA = 56) exams were respectively conducted when the rats were 4 months and 7 months of age. In the intra-scan tests, the animals were scanned twice consecutively, using an identical localizer. In the inter-scan tests, two consecutive scans were performed on each rat, using different localizers. After the first scan, the rat was removed from the table and was re-positioned for the second examination. The operator would repeat the localizers and re-prescribe the imaging planes on newly acquired TOF images.

2.3. Quantitative data analysis

Before the delineation of the regions of interest (ROIs), the phase-offset errors from noncompensated eddy-current-induced fields and concomitant gradient terms were corrected as a previous study [18]. The ROIs ($\sim 5 \times 5$ pixels) of the CCA_{mid} and CCA_{bifur} were determined manually on magnitude images for each time frame by using an in-home developed program written in Matlab (The MathWorks, Natick, MA, USA) [19]. The selected ROIs were transferred to the phase images to calculate the blood flow velocity, flow rate, flow volume, and the derived WSS for the CCA_{bifur} and CCA_{mid}, as shown in Fig. 1(c) and (d). The flow-related indices of both of right and left CCAs were calculated.

For a Newtonian and incompressible fluid, the WSS tensor ($\vec{\tau}$) could be expressed by the following equation [19]:

$$\vec{\tau} = 2\eta\epsilon \quad (1)$$

where η is the viscosity of fluid and ϵ is the deformation tensor. If a 1D problem is considered, the WSS can be simplified to the following equation, as described by Stalder et al. [19]:

$$WSS = \eta \frac{dv}{dr} \quad (2)$$

where η is estimated as 3.5×10^{-3} N s/m² in a rat model [20], v is the fluid velocity, and r is the vascular radius. The temporal averaged WSS (WSS_{avg}) was calculated to determine the mean WSS during the

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