In vivo wall shear stress patterns in carotid bifurcations assessed by 4D MRI

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Summary We investigated the distribution of wall shear stress (WSS) within the carotid artery bifurcation of healthy volunteers and patients with internal carotid artery stenosis. WSS was determined by flow-sensitive 4D MRI and correlated with bifurcation angle, vessel tortuosity and the ratio of the diameter of the common (CCA) and internal carotid artery (ICA). Critical WSS occurred at the posterior wall of the physiologically dilated ICA bulb and the incidence of critical WSS values was dependent from individual bifurcation geometry. Moreover, we found that ICA stenosis changed physiological WSS distribution whereas carotid endarterectomy partially restored physiological WSS conditions.

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Introduction

Wall shear stress (WSS), the friction force of flowing blood that acts on the endothelial wall, can vary considerably throughout the vascular beds and has shown to be altered at the outlet or at the inner curvature of arteries, respectively. In an animal model, Cheng et al. \cite{1} showed that both low and oscillating WSS predispose to the development of atherosclerotic lesions and that oscillatory shear stress causes stable plaques whereas low WSS causes vulnerable lesions. Using computational fluid dynamics Lee et al. \cite{2} demonstrated that individual bifurcation geometry was correlated with the distribution of critical WSS values in healthy volunteers. Data from in vivo studies, however, are sparse. Therefore, we investigated the distribution of WSS along the carotid bifurcations of volunteers and patients using flow-sensitive 4D MRI in vivo \cite{3}. Findings of our previously published study \cite{3} are summarized here in brief.

Material and methods

64 carotid bifurcations of 32 healthy volunteers and 17 carotid arteries of patients with moderate ICA stenosis or recanalized high-grade ICA stenosis were evaluated. Blood flow velocities were measured using a 3 Tesla MRI system (TIM TRIO, Siemens, Erlangen, Germany) and a combined 12-element head and 6-element neck coil. Temporal and spatial resolution of flow-sensitive 4D MRI that was used for three-dimensional velocity acquisition were 45.6 ms and 1.1 mm × 0.9 mm × 1.4 mm \cite{3}. After postprocessing of raw data and based on a commercially available software (Ensight, CEI, Apex, USA) 7 analysis planes, were positioned along the common (CCA) and internal carotid artery stenosis (ICA) with an inter-slice distance of 4 mm. The use of an in house software (Matlab based Flowtool, The Mathworks, USA) and a lumen segmentation method allowed for individual WSS quantification as described previously \cite{4}. Following the study by Lee et al. \cite{2} individual bifurcation geometry (bifurcation angle, tortuosity and diameter ratio of the CCA and ICA) of healthy volunteers was manually determined by two readers based on time-of-flight MR

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http://dx.doi.org/10.1016/j.permed.2012.03.019
The carotid artery bifurcation of a patient with moderate internal carotid artery (ICA) stenosis is given. Blood flow is visualized using particle traces originating from an emitter plane at the distal common carotid artery (CCA) in early systole (i.e., 251 ms after the R wave of the ECG). Color coding represents absolute velocities in m/s. Accordingly, high velocities are represented by yellow and red and slow velocities by green and blues colors. Left: the stenotic segment of the proximal ICA can be appreciated (white arrow). Moreover, a poststenotic dilatation with the occurrence of a pronounced helical flow pattern in the distal ICA is demonstrated (yellow arrow). The external carotid artery (ECA) is characterized by a flow acceleration at the proximal vessel segment. In addition, a distal branch of the ECA is shown by the vessel contours (gold color, particles are only found at the outlet of the branch). Right: flow visualization of the bifurcation of this patient and at the same time point is presented from a posterior view. The stenotic segment (white arrow) and the pronounced helical pattern of blood flow distal to the ICA stenosis (yellow arrow) are demonstrated.

angiographies. The temporal average over the cardiac cycle of the absolute WSS (N/m²) and the degree of absolute WSS inversion over the cardiac cycle (oscillatory shear index, OSI in %) were extracted for 12 segments along the vessel circumference. Values of oscillatory and low wall shear stress of all healthy volunteers were pooled and the 10% and 20% highest and lowest values of absolute WSS and OSI of this cohort were defined as critical WSS. The distribution of critical WSS along the bifurcation of healthy volunteers and patients was then displayed and correlated with individual geometrical features [3].

Results and discussion

An example of three-dimensional blood flow visualization in a patient with ICA stenosis and thus significant changes compared to physiological blood flow patterns at the carotid bifurcation is given in Fig. 1. Critical WSS was consistently concentrated in proximal bulb regions of the CCA and ICA and thus at the site where carotid artery plaques typically develop. Multiple regression analysis revealed significant relationships between the vessel walls with critical WSS and the ICA/CCA diameter ratio. The size of regions that were exposed to critically OSI was significantly correlated with all geometrical parameters (bifurcation angle, tortuosity and ICA/CCA diameter ratio). Moderate ICA stenosis altered physiological WSS distribution whereas recanalization of previously high-grade ICA stenosis led to a similar distribution of WSS compared to healthy volunteers [3].

Flow-sensitive 4D MRI demonstrated the distribution of absolute and oscillatory WSS in vivo. Moreover, physiological and pathological blood flow parameter could be identified that were associated with atherosclerotic disease and recanalization procedures. This in vivo MRI technique seems very promising to study the influence of individual bifurcation geometry on local hemodynamics and the development and progression of carotid artery atherosclerosis.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ijmed.2012.03.019.

References