北京航空航天大学四年级博士生和五年级直博生 学校奖学金申报表

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姓名	李晓银	学 号	BY1510116		指导教	师	樊瑜波&邓小燕	
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承担 科研 任务 情况	项目名称		课题来	源	课题负责		本人承担的具体工 作	
	动脉系统旋动流抑制动脉粥样硬化所起生理 作用的研究		国家自然科学基金		邓小燕		研究动脉粥样硬化的发生 机制	
	一氧化氮在动脉中的输运规律及其与动脉粥 样硬化局部性现象的相关性研究		国家自然基金		邓小燕		计算 NO 在家兔主动脉中的 运输规律	
	血管支架植入后力学环境变化影响血管支架 内再狭窄和晚期血栓形成的机制研究		国家自然基金		邓小燕		计算血管支架植入后力学 环境的变化	
己取得	论文题目		本人排	非名 发表年)		月	期刊(会议)	被检索
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Authors for correspondence:

Xiaoyan Deng

e-mail: dengxy1953@buaa.edu.cn

Yubo Fan

e-mail: yubofan@buaa.edu.cn

Numerical simulation of haemodynamics and low-density lipoprotein transport in the rabbit aorta and their correlation with atherosclerotic plaque thickness

Xiaoyin Li^{1,†}, Xiao Liu^{1,†}, Peng Zhang¹, Chenglong Feng¹, Anqiang Sun¹, Hongyan Kang¹, Xiaoyan Deng¹ and Yubo Fan^{1,2}

Two mechanisms of shear stress and mass transport have been recognized to play an important role in the development of localized atherosclerosis. However, their relationship and roles in atherogenesis are still obscure. It is necessary to investigate quantitatively the correlation among lowdensity lipoproteins (LDL) transport, haemodynamic parameters and plaque thickness. We simulated blood flow and LDL transport in rabbit aorta using computational fluid dynamics and evaluated plaque thickness in the aorta of a high-fat-diet rabbit. The numerical results show that regions with high luminal LDL concentration tend to have severely negative haemodynamic environments (HEs). However, for regions with moderately and slightly high luminal LDL concentration, the relationship between LDL concentration and the above haemodynamic indicators is not clear cut. Point-by-point correlation with experimental results indicates that severe atherosclerotic plaque corresponds to high LDL concentration and seriously negative HEs, less severe atherosclerotic plaque is related to either moderately high LDL concentration or moderately negative HEs, and there is almost no atherosclerotic plaque in regions with both low LDL concentration and positive HEs. In conclusion, LDL distribution is closely linked to blood flow transport, and the synergetic effects of luminal surface LDL concentration and wall shear stress-based haemodynamic indicators may determine plaque thickness.

1. Introduction

It is well documented that atherosclerosis is much more prone to occurring in particular regions of the arterial system where the geometry changes sharply, such as arterial branching, curvature and vascular stenosis, which is referred to the localization of atherosclerosis [1,2]. Two mechanisms have been proposed to explain the phenomenon [3]. One is the vascular responses to abnormal blood flow-induced shear stress [4–6]. The other one is the localized alterations in mass transport [4,7–15]. For the shear stress mechanism, it is believed that

¹Key Laboratory for Biomechanics and Mechanobiology of the Ministry of Education, School of Biological Science and Medical Engineering, Beihang University, Beijing 100191, People's Republic of China ²National Research Center for Rehabilitation Technical Aids, Beijing, People's Republic of China

⁽ii) XL, 0000-0001-7935-2444