

·临床研究 Clinical research·

流固耦合分析脉压差对颈内动脉狭窄处壁面剪切力的影响

李建伟, 纪盛章, 宋金玉

【摘要】 目的 基于流固耦合(fluid structure interaction, FSI)的方式,分析不同脉压差对颈内动脉狭窄处壁面剪切力(wall shear stress, WSS)的影响。**方法** 建模为颈内动脉轻度狭窄的 CT 血管造影(computed tomography angiography, CTA)图像,将模型导入 FSI 分析软件中。入口条件设定为 A、B 两组, A 组 8 种入口条件:舒张压设定为 80 mmHg,收缩压间隔 10 mmHg 由 90~160 mmHg 递增; B 组 8 种入口条件:收缩压设定为 120 mmHg,舒张压间隔 10 mmHg 由 110~40 mmHg 递减。获取两组数据后,分别测量颈内动脉狭窄处截面收缩期和舒张期的 WSS 数值和差值。**结果** 两组 WSS 差值随着脉压差增大而增大,当压差为 80 mmHg 时, A 组 WSS 差为 92.821 Pa, B 组 WSS 差为 98.203 Pa, 远高于正常压差(40 mmHg)的 WSS 差值 52.041 Pa; B 组脉压差越小,一个心动周期内的整体 WSS 越高, B 组脉压差为 10 mmHg 时,收缩期和舒张期的 WSS 分别为 220.384 Pa 和 204.744 Pa。**结论** 颈内动脉狭窄处的脉压差增高, WSS 的振荡幅度也会增大,易造成斑块破裂;当脉压差低且收缩压和舒张压同时偏高时, WSS 整体也偏高,同样是斑块的易损因素,通过 FSI 分析脉压差对颈内动脉狭窄处的 WSS 数值,有助于临床预防和治疗。

【关键词】 脉压差; 壁面剪切力; 颈内动脉狭窄; 流固耦合

中图分类号: R743.3 文献标志码: B 文章编号: 1008-794X(2023)-11-1131-04

Fluid structure interaction analysis of the effect of pulse pressure difference on the wall shear stress at the stenosis of internal carotid artery LI Jianwei, JI Shengzhang, SONG Jinyu. Tianjin Fourth Central Hospital, Tianjin 300140, China

Corresponding author: JI Shengzhang, E-mail: jsz0549@163.com

【Abstract】 Objective Using fluid structure interaction (FSI) method to analyze the effect of different pulse pressure difference on the wall shear stress(WSS) at the stenosis of internal carotid artery. **Methods** CT angiography(CTA) images of mild stenosis of internal carotid artery were modeled, and the model data were input into the FSI analysis software. The entry conditions were set to two groups: A and B. The entry conditions for group A included 8 items: fixing the diastolic pressure at 80 mmHg, separately setting eight systolic pressures (from 90 mmHg to 160 mmHg with an increasing interval of 10 mmHg). The entry conditions for group A included 8 items: fixing the systolic pressure at 120 mmHg, separately setting eight diastolic pressures (from 110 mmHg to 40 mmHg with an decreasing interval of 10 mmHg). After obtaining the data of the two groups, the systolic and diastolic WSS and difference value(D-value) at the stenosis of internal carotid artery were separately measured. **Results** In both groups, the WSS D-value increased with the pulse pressure difference increasing. When the pressure difference was 80 mmHg, the WSS D-value in group A was 92.821 Pa, which in group B was 98.203 Pa. The WSS D-value of both groups was much higher than 52.041 Pa which was obtained when the pressure difference was at normal 40 mmHg. In group B, within a single cardiac cycle the smaller the pulse pressure difference was, the higher the overall WSS would be. In group B, when the pressure difference was 10 mmHg, the systolic WSS and diastolic WSS were 220.384 Pa and 204.744 Pa, respectively. **Conclusion** With the increasing of pulse pressure difference at the stenosis of internal carotid artery, the oscillation amplitude of WSS will also increase, which is prone to plaque rupture. When the pulse pressure difference is low and both systolic and diastolic blood pressure are high, the overall WSS will also be high, which is also a vulnerability factor of plaque. The FSI analysis about the effect of pulse pressure difference on WSS at the stenosis of internal carotid artery is helpful for the clinical prevention and treatment. (J Intervent Radiol, 2023, 32: 1131-1134)

【Key words】 pulse pressure difference; wall shear stress; internal carotid artery stenosis; fluid structure interaction

DOI: 10.3969/j.issn.1008-794X.2023.11.016

作者单位: 300140 天津 天津市第四中心医院

通信作者: 纪盛章 E-mail: jsz0549@163.com

研究表明,脉压差对心脑血管疾病的影响比舒张压、收缩压更为重要,每增加 10 mmHg 的脉压差,在心脑血管疾病中的死亡风险就会增加 2%^[1]。急性缺血性卒中的重要危险因素之一就是颈动脉斑块^[2]。计算流体力学已广泛应用到血管疾病的研究中,尤其壁面剪切力(wall shear stress,WSS)作为血流动力学的重要参数直接与高危斑块相关^[3-4]。本研究以颈内动脉轻度狭窄的数据模型为例,用流固耦合(fluid structure interaction,FSI)有限元分析脉压差对颈内动脉狭窄处 WSS 的影响,为临床提供参考。

1 材料与方法

1.1 研究对象和标准

选取天津市第四中心医院超声显示颈内动脉内膜轻度增厚的 75 岁女性患者 1 例,采用无创 CTA 检查,诊断为颈内动脉轻度狭窄伴斑块。参考北美症状性颈动脉内膜切除协作研究组标准,血管狭窄度($\%$) $= (1-a/b) \times 100\%$ (a 为血管最窄处直径, b 为正常颈内动脉远端直径)。

1.2 有限元模型提取和建立

Mimics Medical 软件中导入 CTA 图像数据,自动阈值分割血管区,生成三维血管的模板库文件,在 Geomagic Studio 中将模板库文件不做任何变形和优化处理,分别生成血管和血液的曲面模型,最后导入到在 ANSYS Workbench 中划分血液和血管的有限元网格,并建立 FSI 的耦合面。

1.3 FSI 分析方法

ANSYS Workbench 软件运算遵守运动、质量、能量守恒定律,方程式为 Navier-Stokes,血液为层流方式,其属性为牛顿特性的不可压缩黏性流体,密度和

黏滞系数分别为 $\rho=1\,060\text{ kg/m}^3$ 和 $\mu=0.0\,035\text{ cP}$ ^[5-7]。血管壁属性为弹性固体,泊松比为 0.45,密度为 $1\,150\text{ kg/m}^3$,弹性模量为 0.6 MPa ^[8]。入口条件设定为 A 组和 B 组,A 组 8 种入口条件:舒张压设定为 80 mmHg,收缩压间隔 10 mmHg 由 90~160 mmHg 递增;B 组 8 种入口条件:收缩压设定为 120 mmHg,舒张压间隔 10 mmHg 由 110~40 mmHg 递减。出口条件为自由流动,各出口相对压力设为零^[9]。运算一个心动周期(1s),狭窄喉部做截面标记,并把 XYZ 轴坐标同平面标记于两组 16 条血管,求收缩期峰值 0.25 s 和舒张压末期 0.7 s 截面位置 WSS 平均值和差值结果。

2 结果

A 组 WSS 数值显示,随着脉压差的增大,WSS 的差值随着收缩期峰值(0.25s)增高而增大;脉压差减小时,WSS 的差值逐渐接近时整体的 WSS 较低,见表 1。从图 1 可以看出,随着脉压差的升高,A 组颈动脉狭窄处的 WSS 在收缩期峰值时由黄绿色逐渐变为橘红色,图示中蓝色为数值低,红色为数值高。

表 1 A 组舒张压 80 mmHg、收缩压递增 10 mmHg 的 8 组脉压下的 WSS 值

脉压(mmHg)	压差(mmHg)	0.25 s(Pa)	0.7 s(Pa)	WSS 差(Pa)
80~90	10	181.077	164.324	16.753
80~100	20	192.669	164.248	28.421
80~110	30	204.556	164.089	40.467
80~120	40	216.039	163.998	52.041
80~130	50	226.295	163.941	62.354
80~140	60	236.555	163.903	72.652
80~150	70	247.168	163.868	83.300
80~160	80	256.665	163.844	92.821

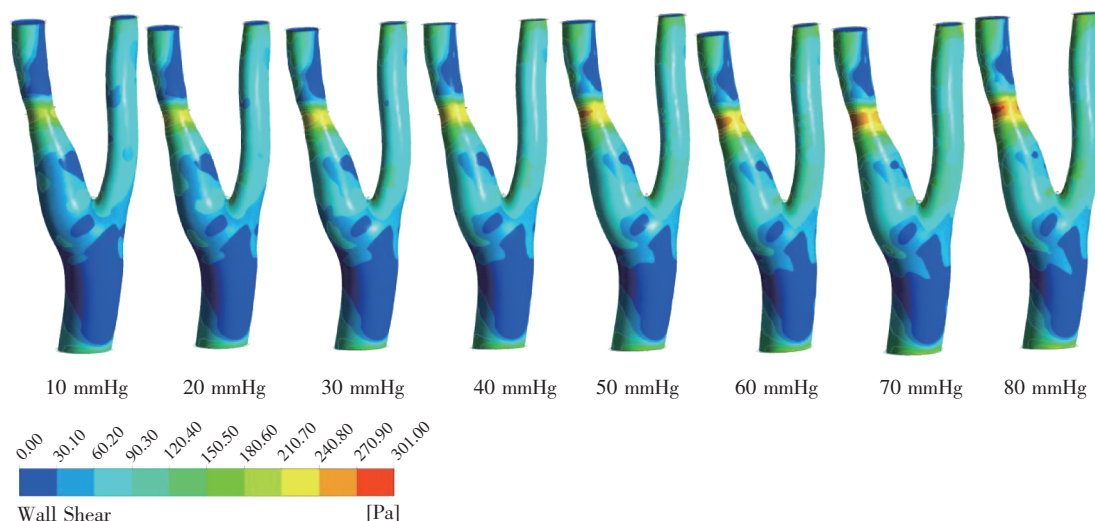


图 1 A 组脉压差以 10 mmHg 递增,收缩期峰值的 WSS 值图

B 组 WSS 数值显示,随着脉压差增大,舒张末期(0.7 s)WSS 值随着舒张压的降低而减小,而心动周期内的 WSS 的差值变大;脉压差减小时的舒张压和收缩压接近,整体 WSS 在此时偏高,见表 2。从图 2 可以看出,随着脉压差的增大,舒张末期的颈内动脉狭窄处 WSS 图示由橘红色逐渐降低为黄绿色,图示中蓝色为数值低,红色为数值高。

表 2 B 组收缩压 120 mmHg、舒张压递减 10 mmHg 的 8 组脉压下的 WSS 值

脉压(mmHg)	压差(mmHg)	0.25 s(Pa)	0.7 s(Pa)	WSS 差(Pa)
110~120	10	220.384	204.744	15.940
100~120	20	218.372	192.496	25.876
90~120	30	217.566	181.043	36.523
80~120	40	216.039	163.998	52.041
70~120	50	215.587	156.066	59.521
60~120	60	213.251	140.599	72.652
50~120	70	211.568	126.498	85.070
40~120	80	210.023	111.820	98.203

3 讨论

随着年龄逐渐增加,血管壁脂质沉积、钙化等因素使血管的弹性降低,收缩期大动脉扩张能力减弱,使舒张期的回缩提前到了收缩期,收缩压进一步升高导致脉压差加大^[10],正常人的脉压差为 20~60 mmHg。可以直接量化的压力、流速和线迹分布等血流动力学重要参数已广泛应用于临床研究^[11-12];尤其 WSS 作为一种平行于血管壁内皮的局部应力,参与调节内皮细胞的形态和管壁重构,管壁的重构与狭窄程度密切相关,标志着斑块的易损性增加^[13-14]。实验中设计两组脉压差为 10~80 mmHg,就是量化狭窄喉部 WSS 与脉压差规律递增以后的关系,为动脉粥样硬化性斑块形成和发展提供

实际理论依据。FSI 分析颈内动脉狭窄处 WSS 的结果中看出,当脉压差升高时,两组的 WSS 差值均增大,当压差在 80 mmHg 时,A 组 WSS 差为 92.821 Pa, B 组 WSS 差为 98.203 Pa, 远远高于脉压差标准值 40 mmHg 时的 WSS 差 52.041 Pa。Doddasomayajula 等^[15]发现,更低的 WSS 和更高的 WSS 表现为高 WSS 振幅,是斑块易损的重要环境因素。脉压差越大 WSS 的振幅越大,对斑块处内膜的作用力越强,势必对斑块造成损伤,形成脑血管意外,因此脉压差增大在计算流体力学 WSS 数值分析上能够直观反映出斑块的易损信号。高 WSS 影响内皮细胞分子机制,局部的血小板撕裂后释放出细胞活性因子并聚集,导致斑块的失稳进阶破裂脱落,最终发生脑卒中^[16-17]。斑块内出血是判断不稳定斑块的重要特征,有研究表明,颈动脉轻度狭窄局部的斑块内出血与 WSS 最大值相关^[18]。A 组数据显示压差越大,收缩压峰值的 WSS 越大,而且远远高于一般水平,这种情况是因为设定时 A 组固定了舒张压,收缩压以 10 mmHg 递增造成,因此要关注压差较大时的收缩压情况,如果收缩压超过正常值而且较大时,就要提示临床高 WSS 对于斑块的高危影响;从 B 组数据能看出,设定收缩压属于一般水平,脉压差越小舒张压越大,其舒张末期的 WSS 越大,当临床面对压差小,但是收缩压和舒张压均较高时,说明一个心动周期内的 WSS 始终保持在一个较高的水平,同样是斑块的易损因素。A 组、B 组数据均显示,收缩压或是舒张压异常升高都会影响斑块局部的血流动力学 WSS 异常改变,对斑块形成易损环境,尤其是压差较大并且收缩压较高时需要临床

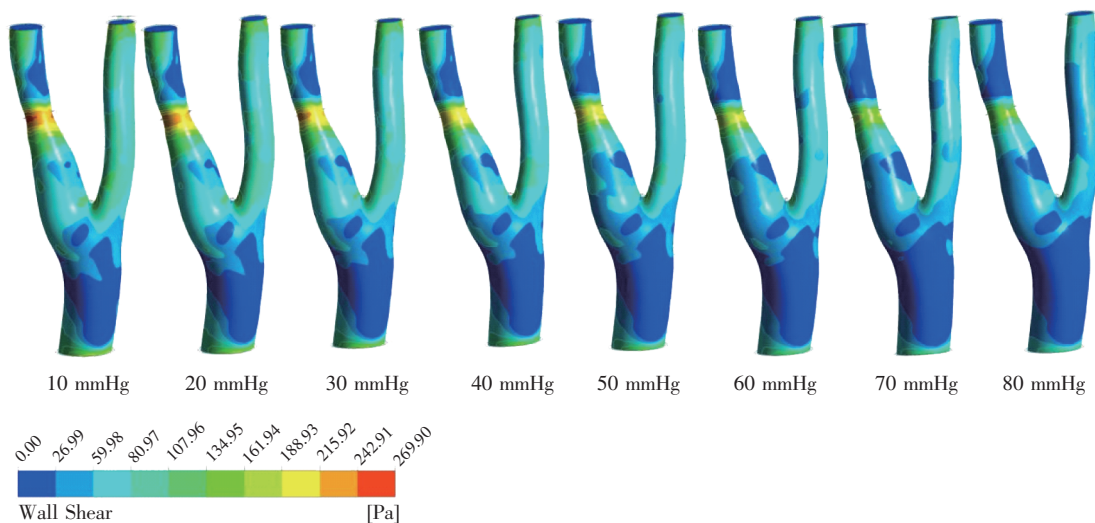


图 2 B 组脉压差以 10 mmHg 递减,舒张末期的 WSS 值图

密切关注。

WSS 是血流动力学中的关键参数,尤其对斑块的形成和发展起着至关重要的作用,当脉压差出现异常时,WSS 随着脉压差过大而产生高振幅,斑块处在一个极不稳定的环境,易损度大大增加;脉压差低于正常值,并且收缩压和舒张压整体偏高时,会使 WSS 处于一个较高水平,这种持续的高 WSS 同样是斑块的易损因素。用 FSI 分析颈内动脉狭窄处 WSS 可以看出,脉压差过大和脉压差过小且收缩压、舒张压整体偏高时都要引起临床关注。本研究设定入口条件为理想脉压,建立个性化的入口条件,并且获得更多的临床病例数据是以后的研究方向。

[参考文献]

- [1] Jae SY, Kurl S, Kunutsor SK, et al. Association between pulse pressure and the risk of sudden cardiac death in middle-aged men: a 26-year follow-up population-based study[J]. Mayo Clin Proc, 2020, 95:2044-2046.
- [2] Bang OY, Kim BM, Seo WK, et al. Endovascular therapy for acute ischemic stroke of intracranial atherosclerotic origin-neuroimaging perspectives[J]. Front Neurol, 2019, 10: 269.
- [3] Hartman EMJ, de Nisco G, Kok AM, et al. Lipid-rich plaques detected by near-infrared spectroscopy are more frequently exposed to high shear stress[J]. J Cardiovasc Transl Res, 2021, 14: 416-425.
- [4] 刘文智,刘莹,罗院明. 斑块偏心分布影响下多组分两相血流动力学数值模拟[J]. 介入放射学杂志, 2019, 28:969-973.
- [5] Jozwik K, Obidowski D. Numerical simulations of the blood flow through vertebral arteries[J]. J Biomech, 2010, 43: 177-185.
- [6] Reid L. An introduction to biomedical computational fluid dynamics[J]. Adv Exp Med Biol, 2021, 1334: 205-222.
- [7] Chandran KB, Rittgers SE, Yoganathan AP. 生物流体力学-人体循环系统[M]. 北京:机械工业出版社, 2014.
- [8] Valencia A, Baeza F. Numerical simulation of fluid-structure interaction in stenotic arteries considering two layer nonlinear anisotropic structural model[J]. Int Commun Heat Mass, 2009, 36:137-142.
- [9] Tan FP, Borghi A, Mohiaddin RH, et al. Analysis of flow patterns in a patient-specific thoracic aortic aneurysm model[J]. Comput Struct, 2009, 87: 680-690.
- [10] Ohayon J, Gharib AM, Garcia A, et al. Is arterial wall-strain stiffening an additional process responsible for atherosclerosis in coronary bifurcations?: an in vivo study based on dynamic CT and MRI[J]. Am J Physiol Heart Circ Physiol, 2011, 301: H1097-H1106.
- [11] Ikoma T, Suwa K, Sano M, et al. Early changes of pulmonary arterial hemodynamics in patients with systemic sclerosis: flow pattern, WSS, and OSI analysis with 4D flow MRI[J]. Eur Radiol, 2021, 31: 4253-4263.
- [12] Paritala PK, Yarlalagadda T, Mendieta JB, et al. Plaque longitudinal heterogeneity in morphology, property, and mechanobiology[J]. Cerebrovasc Dis, 2021, 50: 510-519.
- [13] Chen Z, Li M, Li M, et al. Expansive arterial remodeling of carotid arteries in symptomatic ischemic patients[J]. J Interv Med, 2019, 2: 82-85.
- [14] Ngo MT, Lee UY, Ha H, et al. Comparison of hemodynamic visualization in cerebral arteries: can magnetic resonance imaging replace computational fluid dynamics? [J]. J Pers Med, 2021, 11: 253.
- [15] Doddasomayajula R, Chung BJ, Mut F, et al. Hemodynamic characteristics of ruptured and unruptured multiple aneurysms at Mirror and ipsilateral locations[J]. AJNR Am J Neuroradiol, 2017, 38: 2301-2307.
- [16] Eshthardi P, Brown AJ, Bhargava A, et al. High wall shear stress and high-risk plaque: an emerging concept[J]. Int J Cardiovasc Imaging, 2017, 33: 1089-1099.
- [17] Thim T, Hagensen MK, Horlyck A, et al. Wall shear stress and local plaque development in stenosed carotid arteries of hypercholesterolemic minipigs[J]. J Cardiovasc Dis Res, 2012, 3: 76-83.
- [18] Tuentner A, Selwaness M, Arias Lorz A, et al. High shear stress relates to intraplaque haemorrhage in asymptomatic carotid plaques[J]. Atherosclerosis, 2016, 251: 348-354.

(收稿日期:2022-10-10)

(本文编辑:新宇)