SciMedCentral

Journal of Cardiology & Clinical Research

Editorial

Blood Flow with Boundaries: Wall Shear Stress and Plaque Progression

Emad Rasoul-Arzrumly, Olivia Y. Hung, and Habib Samady*

Division of Cardiology, Andreas Gruentzig Cardiovascular Center, Emory University School of Medicine, USA

Corresponding author

Habib Samady, Andreas Gruentzig Cardiovascular Center, Emory University School of Medicine, 1364 Clifton Road, Suite F606, Atlanta, GA 30322, USA, Tel: 404-778-1237; Email: hsamady@emory.edu

Submitted: 10 August 2013

Accepted: 02 September 2013

Published: 07 September 2013

Shear stress results when fluids move along solid boundaries and is the tangential frictional force produced when fluid interacts with the boundary. In the human body, blood exerts shear stress on the arterial wall and its magnitude is dependent on regional geometry, blood flow, and blood viscosity. Given the complexity of these factors, measurement of coronary blood flow and viscosity, careful three dimensional reconstruction of vascular geometry, and computational fluid dynamics are required to calculate regional wall shear stress (WSS). WSS plays a crucial role in atherosclerotic plaque development, vascular remodeling, and endothelium maintenance [1,2].

Physiological WSS, derived from the pulsatile laminar flow of the cardiac cycle, protects and promotes healthy vasculature. It prevents endothelial cell turnover, enhances vasodilation, and augments anticoagulant, antiinflammatory, and antioxidant effects [1-3]. It occurs in regions of the arterial tree with uniform geometry, where the blood flow is generally unidirectional. In other areas, where the arterial tree has branching, curves, and narrowings, this flow is disrupted, thus creating uneven WSS as well as secondary oscillatory turbulent flows. Within the curvatures, WSS is higher on the outer side and lower along the inner edge. The non-uniform distribution of WSS may help explain the different localization of atherosclerotic lesions in the coronary arteries [4].

Atherosclerosis tends to develop in areas of low WSS. Figure 1 shows blood flow across a curved vessel. Diminished shear stress along the inner edge is linked to decreased endothelial repair and nitrous oxide production as well as accelerated endothelial cell turnover with distorted cell morphology and increased reactive oxygen species, leukocyte adhesion, and lipoprotein permeability [1]. These abnormalities play roles in the formation of the lipid core and the composition of plaque capsules. Low WSS has been shown to be an independent determinant of plaque progression [5-7]. At the other end of the spectrum, high WSS is associated with thinning of the fibrous cap and plaque ulceration as well as the expansion of calcified areas and extensive vascular remodeling [8]. The alteration between low and high WSS may be the destabilization factor which leads to the vulnerable plaque formation and eventually plaque rupture.

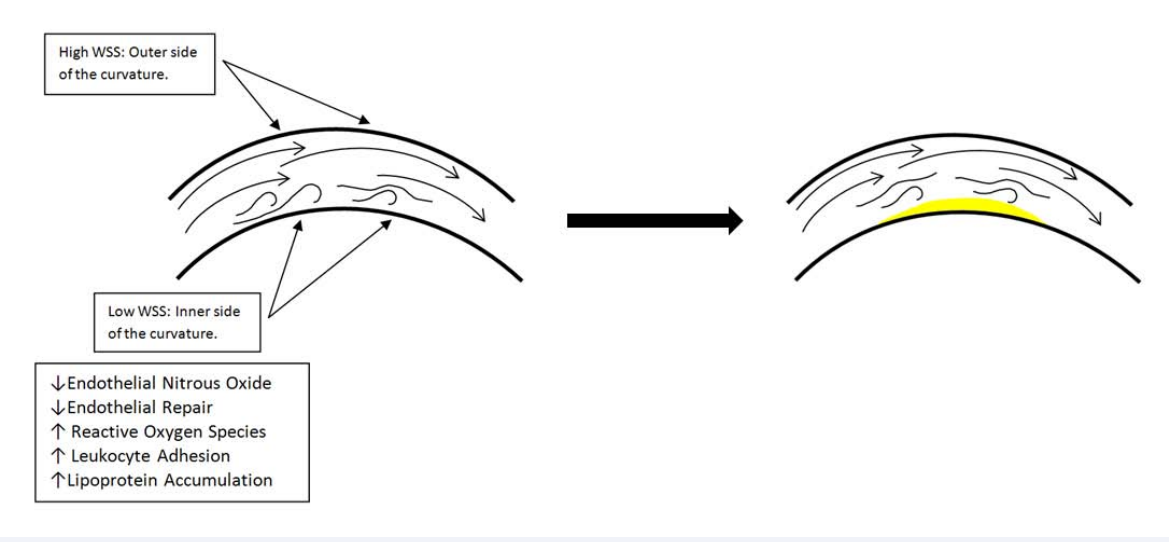

Figure 1 Curvature of the vessel results in different WSS contributing to plaque development.

Another clinically relevant influence of WSS is observed in patients undergoing percutaneous coronary intervention. Stent placement in coronary arteries affects the vascular anatomy and the spatial distribution of WSS which can influence the progression of endothelialization, neointimal hyperplasia, and restenosis [9-12]. The disrupted blood flow occurs between the stent struts, leading to abnormal circulatory currents and low WSS that promotes re-endothelialization. These findings have led to the development of drug-eluting stents to help prevent restenosis by reducing the neointimal hyperplasia process. Post-stent computational fluid dynamics have also informed the structural design of vascular stents and bioabsorbable scaffolds. Indeed, biomechanical investigation of such devices focusing on stent diameter and length, scaffold flexibility, and strut arrangement is contributing to future stent and scaffold design to restore the natural geometry of the vasculature prior to atherosclerotic development.

In summary, assessment of WSS can be a substantial factor in the development of diagnostic and therapeutic strategies in managing patients with coronary artery disease. WSS is affected by any intervention which may distort the coronary anatomy and translating the mechanistic link between WSS and atherogenesis adds a novel dimension beyond plaque morphology. Continued development of methodology to calculate WSS on patients in real time could be used to identify susceptible areas, guide intervention, monitor therapeutic response, and predict outcomes.

DISCLOSURES/ACKNOWLEDGEMENTS

Dr. Samady has received research grants from Volcano Corporation and St. Jude Medical. Dr. Hung wishes to acknowledge the Ruth L. Kirchstein National Resarch Service Awards for a research training grant (5T32HL007745).

REFERENCES

1. Dhawan SS, Avati Nanjundappa RP, Branch JR, Taylor WR, Quyyumi AA, Jo H, et al. Shear stress and plaque development. Expert Rev Cardiovasc Ther. 2010; 8: 545-556.
2. Wentzel JJ, Chatzizisis YS, Gijsen FJ, Giannoglou GD, Feldman CL, Stone PH. Endothelial shear stress in the evolution of coronary atherosclerotic plaque and vascular remodelling: current understanding and remaining questions. Cardiovasc Res. 2012; 96: 234-243.
3. Malek AM, Alper SL, Izumo S. Hemodynamic shear stress and its role in atherosclerosis. JAMA. 1999; 282: 2035-2042.
4. Cecchi E, Giglioli C, Valente S, Lazzeri C, Gensini GF, Abbate R, et al. Role of hemodynamic shear stress in cardiovascular disease. Atherosclerosis. 2011; 214: 249-256.
5. Stone PH, Coskun AU, Kinlay S, Popma JJ, Sonka M, Wahle A, et al. Regions of low endothelial shear stress are the sites where coronary plaque progresses and vascular remodelling occurs in humans: an in vivo serial study. Eur Heart J. 2007; 28: 705-10.
6. Stone PH, Saito S, Takahashi S, Makita Y, Nakamura S, Kawasaki T, et al. Prediction of progression of coronary artery disease and clinical outcomes using vascular profiling of endothelial shear stress and arterial plaque characteristics: the PREDICTION Study. Circulation. 2012; 126: 172-81.
7. Samady H, Eshtehardi P, McDaniel MC, Suo J, Dhawan SS, Maynard C, et al. Coronary artery wall shear stress is associated with progression and transformation of atherosclerotic plaque and arterial remodeling in patients with coronary artery disease. Circulation. 2011; 124: 779-788.
8. Corban MT, Samady H. Wall shear stress and evolution of coronary atherosclerosis: an emerging intravascular imaging modality. Interventional Cardiology. 2012; 4: 155-7.
9. Timmins LH, Miller MW, Clubb FJ Jr, Moore JE Jr. Increased artery wall stress post-stenting leads to greater intimal thickening. Lab Invest. 2011; 91: 955-967.
10. Gundert TJ, Marsden AL, Yang W, LaDisa JF Jr. Optimization of cardiovascular stent design using computational fluid dynamics. J Biomech Eng. 2012; 134: 011002.
11. LaDisa JF Jr, Guler I, Olson LE, Hettrick DA, Kersten JR, Warltier DC, et al. Three-dimensional computational fluid dynamics modeling of alterations in coronary wall shear stress produced by stent implantation. Ann Biomed Eng. 2003; 31: 972-980.
12. Pant S, Bressloff NW, Forrester AI, Curzen N. The influence of strut-connectors in stented vessels: a comparison of pulsatile flow through five coronary stents. Ann Biomed Eng. 2010; 38: 1893-1907.

Cite this article

Rasoul-Arzrumly E, Hung OY, Samady H (2013) Blood Flow with Boundaries: Wall Shear Stress and Plaque Progression. J Cardiol Clin Res 1(2): 1009.