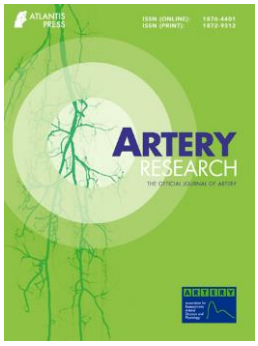

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

REGULATION OF ARTERIAL STIFFNESS: CELLULAR, MOLECULAR AND NEUROGENIC MECHANISMS

Alberto Avolio

To cite this article: Alberto Avolio (2011) REGULATION OF ARTERIAL STIFFNESS: CELLULAR, MOLECULAR AND NEUROGENIC MECHANISMS, Artery Research 5:4, 129–129, DOI: https://doi.org/10.1016/j.artres.2011.10.244

To link to this article: https://doi.org/10.1016/j.artres.2011.10.244

Published online: 14 December 2019

2. Finet G, Huo Y, Rioufol G, Ohayon J, Guerin P, Kassab GS. Structure-function relation in the coronary artery tree: from fluid dynamics to arterial bifurcations. EuroIntervention 2010;6:J10-J15.
3. Le Floc'h S., Cloutier G., Finet G., Tracqui P., Pettigrew RI., Ohayon J. On the potential of a new IVUS elasticity modulus imaging approach for detecting vulnerable atherosclerotic coronary plaques: in vitro vessel phantom study. Phys Med Biol. 2010;55(19):5701-5721.
4. Le Floc'h S, Ohayon J, Tracqui P, Finet G, Gharib A, Maurice R. L, Cloutier G and Pettigrew R. I. Vulnerable atherosclerotic plaque elasticity reconstruction based on Coupling dynamical segmentation with optimization of strain measurements. IEEE Trans Med Imaging. 2009;28(7):1126-37.
5. Ohayon J, Finet G, Gharib A, Herzka D, Tracqui P, Heroux J, Rioufol G, Kotys M, Elagha A and Pettigrew R.I. Necrotic Core Thickness and Positive Arterial Remodeling Index: Emergent Biomechanical Factors for Evaluating the Risk of Plaque Rupture. Am J Physiol Heart Circ Physiol. 2008 Jun;295:H717-727.
6. Ohayon J, Dubreuil O, Tracqui P, Le Floc'h S, Rioufol G, Chalabreysse L, Thivolet F, Pettigrew RI, Finet G. Influence of residual stress/strain on the biomechanical stability of vulnerable coronary plaques: potential impact for evaluating the risk of plaque rupture. Am J Physiol Heart Circ Physiol. 2007 Sep;293(3):H1987-96.
7. Finet G., Ohayon J., Rioufol G. Biomechanical interaction between cap thickness, lipid core composition and blood pressure in vulnerable coronary plaque: impact on stability or instability. Coron Artery Dis. 2004;15(1):13-20.

MRI IN CAROTID PLAQUE

C. Oppenheim, E. Touzé
Paris, France

The emergence of high-resolution imaging methods has enabled MRI to noninvasively image the fine internal structure of cervical arterial walls. This presentation will provide a comprehensive guide to perform high resolution MRI (HR-MRI) of cervical arteries, including the choice of coils, sequences, imaging parameters, and tips for optimal image quality. HR-MRI of carotid atherosclerosis has the potential to be used in treatment decisions or to monitor the effects treatment options. We will explain and illustrate how to quantify plaque volume, determine atherosclerotic plaque burden, detect plaque composition, and ultimately identify unstable plaque before it leads to a clinical event using HR-MRI. Finally, the role of HR-MRI in the diagnosis of cervical dissection and inflammatory disease of the arterial wall will be emphasized.

WHAT IS THE FUTURE FOR RESEARCH IN ARTERIAL STIFFNESS AND CENTRAL BLOOD PRESSURE ?

Michel E. Safar
Université Paris Descartes; Assistance Publique—Hôpitaux de Paris; Centre de Diagnostic et de Thérapeutique, Hôtel-Dieu, Paris, France

Please see Invited Speaker Extended Review Papers section.

MOLECULAR MECHANISMS OF THE VASCULAR REPSONSES TO HAEMODYNAMIC FORCES

S Lehoux
Montreal, Canada

Numerous studies in man and in animals support the relation between low or turbulent shear stress and atherosclerotic plaque location. However, little is known regarding the role of shear stress on the progression and composition of pre-established plaques. A recent prospective study in patients revealed that atherosclerosis regression is a realistic goal in patients, and it can be achieved through increased shear stress. We have used a model of arteriovenous fistula (AVF) developed in the lab, connecting the right common carotid artery with the jugular vein, to increase blood flow over established plaques in the brachiocephalic artery of LDL receptor knockout mice. Animals are placed on a high-fat diet for 12 weeks (wk0-wk12), then divided in three groups : control, sham, or AVF. Sham and AVF animals are maintained on high-fat for 4 weeks post-surgery. Atherosclerotic plaque size in the brachiocephalic artery averages $100,100\pm13,900\mu m^2$ at wk12 (control animals) and progresses to $134,200\pm15,100\mu m^2$ in sham by wk16, whereas it actually regresses by 53% in AVF in the same timeline ($63,500\pm15,100\mu m^2$; $p<0.02$). Relative smooth muscle cell, macrophage, and collagen content are equivalent between groups, but matrix metalloproteinase (MMP) activity is enhanced in the AVF mice, suggesting that these enzymes might participate in the remodeling process. Indeed, in mice treated with MMP inhibitors and TIMP plasmids, MMP expression and activity diminish, and AVF fails to reduce plaque size. Hence, a favorable local shear stress may reverse the atherosclerotic process through a process involving metalloproteinases.

VASCULAR REMODELLING OF THE RETINAL MICROCIRCULATION IN HYPERTENSION AND DIABETES

Roland E. Schmieder M.D.
Nephrology and Hypertension, University Hospital, Erlangen, Germany

In addition to the classical signs of hypertensive nephropathy (e.g. arterial narrowing, arterial venous nicking, retinal haemorrhages or microaneurysms) new technologies have emerged to characterize hypertensive retinopathy more precisely: Scanning Laser Doppler Flowmetry (SLDF) which allows the precise characterization of the remodelling processes in retinal arterioles, and techniques analyzing the retinal microvascular architecture. For example, in patients with essential hypertension and malignant hypertension the length to diameter ratio (indicating generalized narrowing) has been found to be increased and the number of terminal branches (a parameter of capillary density) reduced.
By applying SLDF with automatic full field perfusion imaging analysis, (AFFPIA) our data suggest that retinal arterioles and subcutaneous small arteries undergo the same type of remodelling in hypertension, and also the pattern in quantity of vascular changes are comparable. Most recently, a close correlation between the changes of the subcutaneous small arteries and retinal arterioles have been found. Hypertensive patients with a history of cerebrovascular events showed a greater wall to lumen ratio of retinal arterioles than hypertensive and normotensive controls. Of note, treated hypertensive patients with poor blood pressure control had a greater wall to lumen ratio than those with a better blood pressure control. In diabetic hypertensive patients hypertrophic outward remodelling occurs characterized by increased wall crossectional area and enlarged outer diameter. This pattern has been observed in subcutaneous and retinal vessels.

REGULATION OF ARTERIAL STIFFNESS: CELLULAR, MOLECULAR AND NEUROGENIC MECHANISMS

Alberto Avolio
Australian School of Advanced Medicine. Macquarie University, Sydney, Australia

Please see Invited Speaker Extended Review Papers section.