NITRIC OXIDE, A PROTECTIVE MOLECULE IN THE CARDIOVASCULAR SYSTEM

Lei J, Vodovotz Y, Tzeng E, Billiar TR[1]

Abstract

Nitric oxide (NO) is an intra- and inter-signaling molecule that regulates vessel dilatation, neuronal transmission, cardiac contraction, immunomodulation, and stem cell differentiation and proliferation. NO plays an important protective role in the cardiovascular system. NO inhibits smooth muscle cells proliferation and migration; enhances proliferation and migration of endothelial cell and inhibits apoptosis; suppresses platelet aggregation; and prevents platelet, leukocyte and monocyte adhesion to endothelium. NO exerts an inhibitory effect on the development of intimal hyperplasia in mechanically or immunologically injured vessel. New therapeutic approaches aimed at enhancing NO bioavailability or assisting delivery of NO locally may help patients with cardiovascular disease **[Nitric Oxide. 2013;35:175-85]. PMID: 24095696**

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A REVIEW OF METHODS TO DETERMINE THE FUNCTIONAL ARTERIAL PARAMETERS STIFFNESS AND RESISTANCE

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Abstract

Objectives: In treatment of hypertension not only the pressure response is of interest, but also the effect on arterial parameters, for example, stiffness and resistance, is essential. We therefore reviewed what quantitative information on arterial stiffness can be obtained from pressure wave analysis.

Methods: Using data from published large cohort studies, we derived relations between stiffness and the pressure-derived variables systolic pressure, pulse pressure, augmentation index (AIx), return time of reflected wave and reflection magnitude.

Results: All pressure-derived variables give limited information on arterial function in terms of stiffness and resistance, except AIx (in low stiffness range only). Input impedance as a comprehensive description of the arterial system is too complex to derive and interpret in practice, but is accurately described by three parameters: systemic vascular resistance, total arterial stiffness, and aortic characteristic impedance (outflow tract size and proximal aortic stiffness). These parameters predict aortic pressure well in terms of magnitude and shape: with measured flow the predicted (p) and measured (m) systolic, Ps, and diastolic, Pd pressures relate as Ps,p=0.997 Ps,m-1.63 and Pd,p=1.03 Pd,m-3.12 mmHg (n=17). Therefore, methods should be developed to determine, preferably noninvasively, these three arterial parameters.

Conclusion: Variables derived from pressure wave shape alone (e.g. inflection point, AIx among others), and wave separation (e.g. reflection magnitude), while predicting cardiovascular events, give little information on arterial function. We propose to develop new, and improve existing, noninvasive methods to determine systemic vascular resistance, total arterial stiffness, and aortic characteristic impedance. This will allow quantifying the response of arterial function to treatment **[J Hypertens. 2013 Sep; 31(9):1769-75]. PMID:23777762**

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WHAT IS THE MECHANISM OF FLOW-MEDIATED ARTERIAL DILATATION

Markos F, Ruane O'Hora T, Noble MI[1]

Abstract

The present review attempts to explain the controversies concerning the mechanism of shear stress-mediated arterial dilatation, commonly called flow-mediated arterial dilatation (FMD). Flow-mediated dilatation occurs in an artery when the blood flow to the organ supplied by the artery is increased. There are two hypotheses regarding the stimulus for FMD: (i) a wave of endothelial and smooth muscle hyperpolarization, conducted in a retrograde fashion from the vasodilated peripheral vascular bed towards the relevant conduit artery; and (ii) an increase in shear stress sensed by the endothelial cells. The latter hypothesis is associated with two further postulates concerning the method of mechanotransduction of the shear stress stimulus: (i) direct transmission from endothelial cell cytoskeleton to the vascular smooth muscle to induce dilatation; and (ii) indirect transmission to the endothelial cell cytoskeleton via the glycocalyx. The virtues and inconsistencies of these hypotheses are discussed. The first hypothesis is excluded because a vasodilated peripheral vascular bed does not cause dilation of the upstream conduit artery if an increase in flow within the conduit artery is prevented and because FMD is completely blocked by inhibition of nitric oxide synthase (NOS). It is probable that the stimulus is an increase in shear stress between the blood and the adjacent layer of the arterial wall, the glycocalyx. Ultimately, a change in the endothelial cell cytoskeleton is the likely event that leads to activation of NOS and this activation does not occur without a functioning glycocalyx **[Clin Exp Pharmacol Physiol. 2013; 40(8): 489-94].PMID:23692253**

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