New Assessment of Endothelium-Dependent Flow-Mediated Vasodilation to Characterize Endothelium Dysfunction

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The vascular endothelium plays an important role in the regulation of vascular tone, cell growth, inflammation, and thrombogenicity. Endothelium dysfunction, then, is considered to promote several disorders that initiate the atherosclerosis process. Vascular tone dysfunction can be determined by high-resolution ultrasonographic imaging of the brachial artery, enabling one to assess endothelium-dependent flow-mediated dilation (FMD). It is based on the principle that an increase in blood flow, specifically in shear stress, provokes the release of nitric oxide and then a vasodilation that can be quantified. In this study, brachial artery diameter evolution was continuously followed during baseline and hyperemia after forearm occlusion using a custom designed software. Some techniques used to measure FMD are limited by operator dependence. We present a new, automated, and versatile method of FMD quantification based on B-mode echographic images and edge detection algorithms. Edges for each image in the acquired sequences are recognized as interfaces based on the grey-level profiles of the averaged pixel values. Within-reading and within-subject FMD% coefficients of variation were 7% and 10%, respectively. This technique largely improves manual measurements and was shown to be appropriate for wide clinical use.

Keywords: flow-mediated vasodilation, ultrasonography, endothelium, age

INTRODUCTION

The endothelium is a single-cell layer that plays a role as a mechanical and biological barrier between the blood stream and the vascular wall. This interface regulates the permeability between vascular tissue and blood cells, platelet aggregation, thrombogenicity, cellular proliferation, and ultimately modulates arterial vasomotor activity by way of the release of vasoactive substances such as nitric oxide (NO).1 The latter substances induce arterial vasodilatation, which is the most analyzed function in clinical studies. Endothelial dysfunction can be understood as a state in which this role is altered, inducing vascular constriction, leukocyte adherence, platelet activation, thrombosis, vascular inflammation, and ultimately atherosclerosis.2

Normally, blood vessels dilate as a response to flow rise. More specifically, a flow increase causes higher levels of friction between blood and endothelium cells ( ie, shear stress ) that releases NO synthesized from L-arginine under the influence of the enzyme NO synthase.3 This endothelium-derived relaxing factor is mainly responsible for vascular tone modulation, and a reduction in bioavailability characterizes endothelium dysfunction.

Endothelial function in humans can be assessed with several invasive1 and noninvasive4–7 methods.