Flow-Mediated Slowing as a Methodological Alternative to the Conventional Echo-Tracking Flow-Mediated Dilation Technique for the Evaluation of Endothelial Function: A Preliminary Report

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Abstract

The Moens-Korteweg equation predicts changes in pulse wave velocity (PWV) after changes in arterial radius; therefore, an increase in arterial radius, as seen in a reactive hyperemia (RH) condition, should slow PWV over a given arterial segment. If this assumption is true, then the deceleration of PWV over the brachial artery (flow-mediated slowing [FMS]) should be an equivalent signal of endothelial function during a conventional RH flow-mediated dilation (FMD) procedure. Our aim was to compare FMS with FMD after RH in healthy individuals as part of a study that seeks to evaluate the clinical usefulness of FMS as a noninvasive approach to characterize endothelial function. This cross-sectional study included 25 healthy participants (18 women [72%]) with a mean ± SD age of 21.12±0.73 years. The FMD and FMS were simultaneously measured. A significant correlation was observed between both measures of FMS (absolute difference and percentage variation) and echo FMD: $R=-0.42$ ($P=.04$) and $r=0.46$ ($P=.02$), respectively. The FMS was shown to depend on the baseline brachial diameter, with smaller variations depicted for smaller baseline brachial diameters. It seems to be a promising and feasible method for measuring changes after RH, although further studies are needed to evaluate how this correlation holds in different clinical conditions and to demonstrate its clinical usefulness.

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The role of endothelial dysfunction (ED) in the natural history of cardiovascular disease is well-known and is acknowledged as one of the earliest stages in the physiopathologic continuum of atherosclerosis.[1] Thus, ED is an important cardiovascular risk marker, with significant prognostic implications.[2-7] To date, flow-mediated dilation (FMD) is recognized as the reference method for assessing endothelial function (EF) noninvasively. The percentage increase in brachial artery diameter after reactive hyperemia (RH) defines the FMD and relates to the endothelium's ability to release and respond to nitric oxide, thus indicating its functional health.[7] Although this technique has strong advantages, it also has some important disadvantages, such as being highly operator-dependent, which could determine unacceptable variability unless a dedicated technician and adequate equipment are present.[8] As an alternative to the conventional FMD technique, pulse wave velocity (PWV) could offer a much easier and more reproducible methodological alternative to studying EF. In fact, the Moens-Korteweg equation links PWV to the arterial radius, as follows: $PWV = \frac{\sqrt{E \cdot h}}{D \cdot \delta}$, where E is the elastic modulus; h, the wall thickeness; D, the arterial diameter; and δ, the blood density.[9] The theoretical consequence of this is that if the arterial radius R increases after RH, a decrease in PWV is expected to occur. If this assumption is true, then the deceleration of PWV over the brachial artery (what we designate as flow-mediated slowing [FMS]) should be an equivalent signal of EF during a conventional RH procedure. Because PWV is a validated

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TABLE. Demographic Characteristics of the Study Population[a,b]

Characteristic	Total (N=25)	Men (n=7)	Women (n=18)	*P* value
Age (y)	21.12±0.73	21.00±10	21.17±0.62	.62
Height (m)	1.68±0.10	1.80±0.05	1.63±0.06	<.001
Weight (kg)	62.10±10.47	72.42±9.59	58.06±7.81	.001
BMI	22.10±3.19	22.30±2.62	22.04±3.45	.86
SBP (mm Hg)	114.20±10.80	121.57±8.12	111.33±10.50	.03
DBP (mm Hg)	66.72±8.48	65.43±6.45	67.22±9.27	.65
PP (mm Hg)	47.48±11.11	56.14±10.19	44.11±9.74	.01
MBP (mm Hg)	88.55±7.70	84.14±5.16	81.93±8.55	.53
HR (beats/min)	70.92±8.79	68.14±9.94	72.00±8.35	.34
Basal diameter (mm)	3.46±0.59	4.17±0.43	3.12±0.22	<.001
Basal carotid-radial PWV (m/s)	8.58±1.16	9.23±1.33	8.33±1.02	.08

[a]BMI = body mass index; DBP = diastolic blood pressure; HR = heart rate; MBP = mean blood pressure; PP = pulse pressure; PWV = pulse wave velocity; SBP = systolic blood pressure.
[b]Data are presented as mean ± SD.

method, easy to perform, highly reproducible, and low cost, the demonstration of its usefulness for evaluating ED should be of major interest.[8] To date, just a few studies have explored this hypothesis.[10,11]

Based on this premise, we present the preliminary results of an ongoing project that aims to appraise the clinical usefulness of FMS for the evaluation of ED, particularly focusing on the comparision between FMS and FMD after RH in healthy individuals.

METHODS

We conducted a cross-sectional study aimed at comparing FMS and FMD estimations of EF in 25 clinically healthy participants (18 women [72%]) with a mean ± SD age of 21.12±0.73 years.

Evaluations were conducted in a laboratory with controlled luminosity, temperature, and humidity. All the evaluations took place in the morning, with participants fasting and deprived from smoking, exercise, alcohol, and cafeine for 12 hours before the study. Demographic data were collected for each participant. The participants were then placed in the supine position, and after a 10-minute rest period, brachial blood pressure was measured using an automatic and clinically validated sphygmomanometer (ri-champion N; Rudolf Riester GmbH) with a cuff that adjusted for the arm diameter. Afterward, FMD was obtained, complying with the methodological recommendations.[8,12] A Vivid 3 echograph (General Electric) equipped with a linear probe (frequency range, 7-12 MHz) was used. A basal measurement of the diameter of the right brachial artery in a linear plane was made, approximately 2 to 3 cm off the antecubital fossa. Baseline carotid-radial PWV was then acquired in the same arm using the Complior Analyse device (Alam Medical). For this, a probe with piezoelectric crystals was placed on the right radial artery and another on the ipsilateral carotid artery. The probes were adjusted to ensure the acquisition of pulse waves with suitable reproducible, stability, and amplitude. The distance between the 2 points was measured directly. The brachial PWV corresponded to the distance (d) divided by the pulse wave transit time (PTT) between the 2 arterial territories considered (carotid-radial), where PWV = d/PTT (m/s).

Subsequently, a cuff was placed on the forearm, distal to the site of the ultrasound assessment, and was inflated into a suprasystolic pressure (~50 mm Hg above the previously measured systolic blood pressure), keeping the ischemia for a 5-minute period, after which the cuff was deflated. Approximately 1 minute after complete deflation of the cuff, the diameter of the brachial artery and the brachial PWV were simultaneously measured. The FMD was calculated as the percentage increase in brachial diameter after RH. The FMS was calculated using the baseline PWV and the PWV after RH, as ΔPWV, quantifying the absolute difference between the 2 moments of PWV, and as %PWV, quantifying the percentage of variation. All measurements were performed in the right arm.

All the tests were performed by the same experienced operator (T.P.) to ensure the necessary reproducibility conditions. All the

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