
Flow-mediated vasodilation partially reflects nitric oxide-mediated endothelial function

To the Editor: In this Point:Counterpoint series, Green and Tschakovsky and Pyke address the issue of whether flow-mediated dilation (FMD) reflects nitric oxide (NO)-mediated endothelial function (1). However, this question is difficult to answer. It has been conclusively shown that FMD of the brachial artery after 5 min of wrist cuff occlusion can be entirely abolished by the NO synthase (NOS) inhibitor Nω-monomethyl-L-arginine (L-NMMA), suggesting a complete NO dependency of the process (2). Nevertheless, when the cuff was positioned on the arm above the probe, FMD was found to be 12% and was only reduced to 7.5% by L-NMMA (2). In contrast, Hornig reported a similar vasodilatory response of the radial artery (~12%) independent of whether the occlusion was performed upstream (upper arm) or downstream (wrist) of the site of measurement (3). Interestingly, the increase in radial artery diameter was even more pronounced after longer periods of vessel occlusion before assessment of FMD (~13% after 8 min vs. ~7% after 4 min). The amount of FMD abolished by L-NMMA was ~66% in healthy individuals but ~33% in patients with chronic heart failure (CHF), possibly due to endothelin-mediated vasoconstriction in CHF (1, 3). Therefore, FMD appears to partially reflect NO-mediated endothelial function. The abovementioned data are consistent with the hypothesis that key players other than NO mediating FMD, e.g., prostaglandins and myogenic factors, in different experimental settings depend on 1) the duration of occlusion, 2) the site of measurement in relation to the site of vessel occlusion, and 3) the subjects, in whom the measurements are performed.

REFERENCES

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