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Conference Abstract P.02 Differential 'Mediators' of Low-Flow 'Mediated' Constriction in Healthy vs Patients of Ischemic Heart Disease

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ABSTRACT

Background: Low-flow mediated constriction (LFMC) has emerged as a non-invasive tool for assessment of endothelial dysfunction [1]. There is insufficient data on association between change in artery diameter during occlusion with its possible stimulus; 'low flow' state. This study aims to evaluate the association between change in brachial-artery diameter during constriction with alterations in retrograde, anterograde and oscillatory flow profile in healthy subjects and patients with ischemic heart disease (IHD).

Methods: Brachial-artery responses to occlusion were assessed from artery diameter and blood flow using B-mode and pulsedwave doppler ultrasound respectively in 89 patients with IHD and 29 healthy subjects. Change in anterograde, retrograde and net flow velocity, shear rate (AFV, RFV, NFV, ASR, RSR and NSR respectively) and oscillatory shear index (OSI) during forearm occlusion at 50 mmHg above systolic pressure, from baseline was calculated.

Result: Diameter deceased significantly in healthy subjects and patients during occlusion compared to baseline. Interestingly, in stepwise forward-selection analysis, change in maximum AFV, ASR, RSV and RSR emerged in best fit model, explaining 76.2% of total variability in delta LFMC in IHD patients, with maximum contribution by ASR (70.4%). On the other hand, in healthy, change in maximum RFV, RSR, NFV and NSR emerged in best-fit model explaining 89.9% of total variability, of which 47% was by NSR and 33% by RSR.

Conclusion: Brachial-artery constriction during occlusion is 'mediated' by decrease in ASR in patients of IHD and decrease in NSR and increase in RSR in healthy, highlighting the possibility of differential 'mediators' of constriction in healthy vs diseased.

REFERENCE

[1] Gori T, Dragoni S, Lisi M, Di Stolfo G, Sonnati S, Fineschi M, et al. Conduit artery constriction mediated by low flow: a novel noninvasive method for the assessment of vascular function. J Am Coll Cardiol 2008;51:1953–8.

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