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Abstract: P4757

Regulation of myocardial perfusion at high perfusion pressure during external counterpulsation

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Introduction: Little is known about regulation of myocardial perfusion at high perfusion pressures. External counterpulsation (ECP) is considered to increase blood flow in epicardial coronary arteries. However, there is no data on direct blood flow measurements in the myocardial microcirculation under ECP.

Methods: In 20 healthy subjects, quantitative myocardial contrast echocardiography (MCE) was performed during gradual ECP pressure increase (Baseline, 80, 160, 240 and 300mmHg). Three MCE clips were recorded at each step, and perfusion was calculated offline in end-diastolic frames using customized software. Parameters such as absolute myocardial blood flow (MBF, ml/min/g) and intramyocardial blood volume fraction (relative blood volume rBV, ml/ml) were retrieved. Arterial blood pressure curves were recorded sphygmometrically and digitized. Microvascular resistance was calculated as: diastolic pressure/MBF (mmHg*ml/min*g). Development of parameters under incremental ECP were analyzed by ANOVA repeated measures and Dunnett's post test. Linear trends were assessed by post test for significant linear trends.

Results: There was a significant increase in diastolic pressure under incremental ECP steps from initially 80±11 to a maximum of 134±14 mmHg ($p<0.0001$, significant from step 80mmHg upward). The rBV showed a significant increase from 0.11±0.04 to a maximum of 0.14±0.04 ($p = 0.01$). The MBF showed a slight but significant upward trend from 1.12±0.38 to 1.44±0.88 ml/min/g ($p < 0.05$). The microvascular resistance showed a significant increase from 78±29 at baseline to 127±77 mmHg*min/ml*g at maximal pressure ($p = 0.0004$). This effect was particularly pronounced from step 240mmHg upward.

Conclusions: High pressure-level ECP increases both, intramyocardial blood volume and microvascular resistance. The latter seems to prevent a more pronounced myocardial blood flow augmentation than observed. We interpret these findings as a form of autoregulation operative at the high "end" of coronary perfusion pressure. In previous studies, a much steeper blood flow augmentation in epicardial coronary arteries has been reported. We speculate that this flow is bypassing the microcirculation via recruitment of arterio-arterial or arterio-venous shunts.