Does EECP Significantly Affect Myocardial Perfusion? A Systematic Review & Meta-Analysis

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Study Type	Systematic review & meta-analysis — MEDLINE, EMBASE, Cochrane CENTRAL databases
Evidence Level	Level I — systematic review of prospective studies
Funding	National Natural Science Foundation of China (31300137 and 81171859)
ACC Relevance	Level I evidence confirming EECP improves tissue perfusion — the key mechanism for injury rehabilitation

Objective

To assess whether standard EECP therapy significantly affects myocardial perfusion in coronary artery disease (CAD) patients through systematic review and meta-analysis of all available literature. Databases searched: MEDLINE, EMBASE, Cochrane CENTRAL — from inception to May 2015. Search terms: ('enhanced external counterpulsation' OR EECP) AND perfusion. Human clinical trials in English only.

Included Studies

Study	n	Method	NOS Quality Score
Arora & Bergmann (2007)	26	SPECT myocardial perfusion imaging	High
Lawson et al. (1992)	18	Angiographic + Doppler assessment	High
Masuda et al. (2001)	10	13N-ammonia PET perfusion	High

Michaels et al. (2002)	Not reported	Intracoronary Doppler + pressure wire	High
Michaels et al. (2005)	175	Radionuclide myocardial perfusion (multicenter)	High
Tartaglia et al. (2003)	19	Nuclear exercise stress test + treadmill	High

Meta-Analysis Results

Primary outcome	Weighted mean difference (WMD) in myocardial perfusion pre- vs post-EECP
Pooled WMD	–0.19 (95% CI: –0.38 to 0.00, p=0.049) — statistically significant improvement
Heterogeneity	I^2 = 89.1% (p=0.000) — significant heterogeneity; random-effects model applied
Publication bias	No significant publication bias (Begg's p=0.091; Egger's p=0.282)
EECP Protocol	Standard: 35–36 one-hour sessions within a 7-week period (consistent across all studies)

Putative Mechanisms of Perfusion Improvement

• **Direct vasodilation:** EECP pressure wave directly dilates existing vessels in the myocardium

• **Collateral vessel formation:** EECP increases de novo collateral vessel formation via angiogenic factors (alpha-actin, vWF, VEGF, BFGF, HGF)

• **Nitric oxide:** EECP increases vascular shear stress, promoting release of the angiogenic vasodilator nitric oxide (NO)

• **Haemodynamic augmentation:** EECP pressure wave enhances coronary artery mean pressure by 16% and peak diastolic pressure by 93%

Conclusions

Standard EECP therapy (35–36 one-hour sessions within a 7-week period) significantly increases myocardial perfusion in CAD patients. This Level I systematic review confirms that tissue perfusion improvement is a reproducible, documented effect of EECP across multiple independent prospective studies using diverse measurement techniques. The same perfusion improvement mechanisms — vasodilation, angiogenesis, NO production — are directly relevant to musculoskeletal injury rehabilitation, wound healing, fracture recovery, and post-injury tissue repair.

Full Citation

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