

Contribution of nitric oxide to metabolic coronary vasodilation in the human heart.

[Quyyumi AA](#), [Dakak N](#), [Andrews NP](#), [Gilligan DM](#), [Panza JA](#), [Cannon RO 3rd](#).

National Institutes of Health, Cardiology Branch, NHLBI, Bethesda, MD 20892-1650, USA.

BACKGROUND: The vascular endothelium contributes to smooth muscle relaxation by tonic release of nitric oxide. To investigate the contribution of nitric oxide to human coronary epicardial and microvascular dilation during conditions of increasing myocardial oxygen requirements, we studied the effect of inhibiting nitric oxide synthesis with NG-monomethyl-L-arginine (L-NMMA) on the coronary vasodilation during cardiac pacing in patients with angiographically normal coronary arteries with and without multiple risk factors for coronary atherosclerosis.

CONCLUSIONS: During metabolic stimulation of the human heart, nitric oxide release contributes significantly to microvascular vasodilation and is almost entirely responsible for the epicardial vasodilation. This contribution of nitric oxide is reduced in patients exposed to risk factors for coronary atherosclerosis and leads to a net reduction in vasodilation during stress. An important implication of these findings is that reduced nitric oxide bioavailability during stress in patients with atherosclerosis or risk factors for atherosclerosis may contribute to myocardial ischemia by limiting epicardial and microvascular coronary vasodilation.

[Circulation](#). 1995 Aug 1;92(3):320-6.

PMID: 7634444 [PubMed - indexed for MEDLINE]