Nitroglycerin Reduces Blood Viscosity: A Novel Mechanism to Explain the Enhancement of Nutrient Blood Flow to Ischemic Zones

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Introduction: Nitroglycerin (NTG) is thought to relieve ischemia by increasing coronary blood flow (CBF) via dilation of conduit vessels or from collateral, but its effects on hemodynamics. We have shown, however, that increases in hyperemic CBF are limited mainly by capillary or myocardial vascular resistance (MVR), which depends on blood viscosity. We therefore hypothesized that enhancement of nutrient blood flow to ischemic zones during NTG are secondary not to decreases in stenosis resistance (SR) or collateral flow, but to reduced blood viscosity.

Methods: We studied 5 open chest dogs with a critical LAD or LCx stenosis (group 1). To eliminate collateral flow, the LCx was stenosed while the LAD was occluded in 5 other dogs (group 2). NTG (0.2-0.5ugkg^-1min^-1) was infused into the left main coronary artery to minimize effects on systemic hemodynamics. At baseline and during NTG, mean aortic pressure (MAP), distal coronary pressure (dCP), right atrial pressure (RAP), and whole blood viscosity (WBV) were determined. SR and MVR were calculated using MAP-dCP/CBF and dCP-RAP/CBF, respectively. MCE was performed to determine myocardial blood volume (MBV).

Results: No changes in hemodynamics were seen after NTG compared to baseline in either group of dogs. In group 1 dogs, there was no significant decrease in stenosis resistance or increase in MBV after NTG. A significant decrease in WBV (17±15 %, p=0.01), and a concurrent decrease in MVR in the ischemic bed (1.8±14 %, p=0.01) was found in group 2 dogs with NTG.

Conclusion: Increases in CBF during NTG are not reliant on changes in stenosis resistance, MBV, or collateral flow, but are secondary to reductions in blood viscosity which enhances microcirculatory red cell velocity. This phenomenon might explain in part the anti-ischemic effect of the drug.