relationship was depressed by LNA (P=0.03), whereas the slope of the extraction/MVO_2 relationship increased (P=0.003). In summary, increases in RV MVO_2 during hypoxia are met normally by increasing RC blood flow. When NO synthesis is blocked, the large RV O_2 extraction reserve is mobilized to maintain RV O_2 demand/supply balance. We conclude that NO contributes to RC vasodilation during systemic hypoxia, and, thus, NO is an important factor in maintaining right ventricular oxygen supply-demand balance under this condition. (This research was supported by U.S. National Institutes of Health grant HL-64785.)

NITRIC OXIDE CONTRIBUTES TO OXYGEN DEMAND – SUPPLY BALANCE IN HYPOPERFUSED RIGHT VENTRICLE

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The present study examined the role of nitric oxide (NO) in oxygen demand – supply balance in hypoperfused canine myocardium. The right coronary artery of anesthetized, open-chest dogs was perfused at pressures of 80, 60 and 40 mmHg, and right ventricular myocardial oxygen consumption, right coronary blood flow and other hemodynamic and cardiac function variables were measured. Right ventricular mechanical function was indexed as the product of heart rate X peak right ventricular systolic pressure X right ventricular dP/dt_{max}. NO synthesis blocker N^T–nitro-L-arginine methyl ester (L-NAME, 150 :g/min) was infused into the right coronary artery to block NO synthesis. Neither hypoperfusion nor L-NAME altered right ventricular function. Right ventricular myocardial oxygen consumption was significantly increased during L-NAME at right coronary perfusion pressure of 60 and 40 mmHg ($P < 0.05$ vs. untreated control condition). This increase in myocardial oxygen demand during coronary hypoperfusion was met by non-NO dependent vasodilation as reflected by a significant rise in right coronary blood flow during L-NAME ($P < 0.05$ vs. untreated control condition), but the relationship between oxygen consumption and flow became much steeper. Thus, NO improves right coronary conductance during hypoperfusion and also increases oxygen utilization efficiency. NO plays an important role in right ventricular oxygen demand – supply balance when oxygen delivery is restricted. (This research was supported by U.S. National Institutes of Health grant HL-64785.)