TITLE: EFFECT OF CARBON DIOXIDE (HYPOCAPNIA AND HYPERCAPNIA) ON REGIONAL MYOCARDIAL TISSUE OXYGEN TENSION IN DOGES WITH CORONARY STENOSIS

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Carbon dioxide (CO_2) has been well documented to act as a potent vasodilator of coronary vessels under normal conditions. But there is little data available on the effect of CO_2 on the collateral perfusion of patients with coronary insufficiency. We studied the effects of CO_2 on the myocardial tissue PO2 with critical coronary stenosis in the anesthetized dogs.

12 mongrel dogs were anesthetized with pentobarbital 30mg/kg and ventilated with 100% O_2 to maintain normocapnia. Following left thoracotomy, electromagnetic blood flow (BF) probe was applied to the left anterior descending artery (LAD). Two different regional PO2 were measured using two pairs of monopolar polarographic needle electrodes; one inserted in the epicardial (EPI), and the other in the endocardial (ENDO) layer, which were placed in the regions supplied by LAD and circumflex. Following the baseline recording, critical stenosis of LAD was produced by adjusting a copper-wire clamp occluder until LADBF reduced to 50%. After a stable normocapnic ventilation, hypocapnia was produced by hyperventilation which was kept fixed throughout the experiment. To induce hypercapnia, exogenous CO2 was added to the inspired gas stepwisely till End-tidal CO2 fraction (FECO2) reached 10%. In each step, the following variables were measured:cardiac output (CO), PaO2, PaCO2, LADBF, regional PO2 in both normal(N) and ischemic(I) myocardium(N-EPI PO2, N-ENDO PO2, and I-EPI PO2, I-ENDO PO2). The data were analyzed using paired-T test accepting $p<0.05$ as significant. The results are summarized in table 1. Hypocapnia resulted in a significant reduction of PO2 in both EPI and ENDO non-stenotic areas, while hypercapnia increased these PO2 values dose-dependently. After coronary stenosis, hypocapnia resulted in small but significant reduction of PO_2 in endocardiac ischemic area. Hypercapnia did not show any sign of reduced regional myocardial tissue PO_2 or evidence of regional or intramural "steal" phenomenon. Thus, we conclude that hypercapnia may not worsen the oxygenation of the ischemic myocardium and that maintenance of $PaCO_2$ and avoidance of hypocapnic hyperventilation are important for myocardial oxygenation in both normal and ischemic heart.

Table 1.	Normo-ventilation	Hyperventilation	CO2 inhalation	
FECO2(%)	5	2	6	<10
HR (b.p.m.)	155±4*	182±5	146±4*	130±3*
MAP(mmHg)	128±7	126±5	125±5	116±4*
C.O.(l/min.)	1.73±0.10	1.67±0.06	1.76±0.09	2.03±0.08*
PaCO2(mmHg)	38±1*	23±1	47±2*	93±2*
PaO2(mmHg)	434±37	487±17	453±23	431±13*
normal				
LADBF(ml/min.)	21±4*	17±3	28±4*	46±8*
N-EPI PO2(mmHg)	37±2*	33±2	42±2*	58±4*
N-ENDO PO2(mmHg)	51±5*	37±2	63±5*	88±7*
post-stenosis				
LADBF(ml/min)	11±2	11±1	12±1	15±2*
I-EPI PO2(mmHg)	20±2	18±2	20±3	26±3*
I-ENDO PO2(mmHg)	31±3*	25±3	36±4*	48±5*

Mean+SEM p<0.05 from 2% FECO2

TITLE: MYOCARDIAL O2 SUPPLY/DEMAND RELATIONS DURING PHENYLEPHRINE-INDUCED PRESSOR RESPONSES

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Phenylephrine is a vasoconstrictor acting via alpha-adrenergic receptors that is used in anesthesia practice to treat hypotension. Although alpha-adrenergic receptors are present in the coronary circulation (1), it is uncertain whether the plasma concentration of phenylephrine required for pressor responses under general anesthesia is sufficient to cause their activation. If so, this may impair ability of metabolic vasodilator mechanisms to satisfy the pressure-induced augmentation in myocardial oxygen demand. The present study was performed to evaluate changes myocardial blood flow (MBF), oxygen consumption (MVO2), and lactate extraction (LAC ext) during intravenous pressor infusions of phenylephrine in anesthetized dogs.

Seven mongrel dogs anesthetized with pentobarbital and fentanyl underwent left thoracotomy and were mechanically ventilated. Measurements were obtained of mean aortic pressure (MAP), heart rate (HR), left ventricular dP/dt max, and aortic blood flow (AOF). Radioactive microspheres (15 u) were used to measure mean left ventricular MBF and its transmural distribution (ENDO/EPI). Samples of blood were obtained from aorta and coronary sinus and analyzed for O2 content (CO-oximeter) and LAC. MVO2 was computed with Fick equation. Hemodynamic measurements were obtained under control conditions and following attainment of steady-state hemodynamic conditions during intravenous infusion of phenylephrine (2.8 ug/min/100g). Statistical analyses were performed with Student's t test for paired samples.

Phenylephrine increased MAP (+48%), decreased HR (-23%) and AOF (-23%), and had no effect on dP/dt max. It caused transmurally-uniform increase in MBF that was in proportion to increase in MVO2 resulting in no change in A-V O2 diff., coronary sinus PO2, or LAC ext (Table 1).

In conclusion, increases in myocardial MBF were sufficient to satisfy the augmented cardiac work requirement during phenylephrine-induced hypertension. This suggests that vasoconstrictor action of phenylephrine in coronary circulation did not impair ability of metabolic vasodilator mechanisms to maintain myocardial oxygen supply/demand balance.

References

1. Marcus, ML. The Coronary Circulation in Health and Disease. MacGraw-Hill, 1983, pp 130-132

Table 1. Myocardial effects of phenylephrine.

	Control	Phenylephrine
MBF, ml/min/100g	57 ± 8	101± 14*
ENDO/EPI	1.1 ± 0.1	1.1 ± 0.1
MVO2, ml/min/100g	7.8 ± 0.9	15.1 ± 2.7*
A-V O2, vol. %	14.1 ± 0.3	14.3 ± 0.9
CS PO2, mmHg	28 ± 2	28 ± 1
LAC ext., %	51 ± 9	57 ± 4

Values are Mean ±S.E. *P<0.05, from Control.