

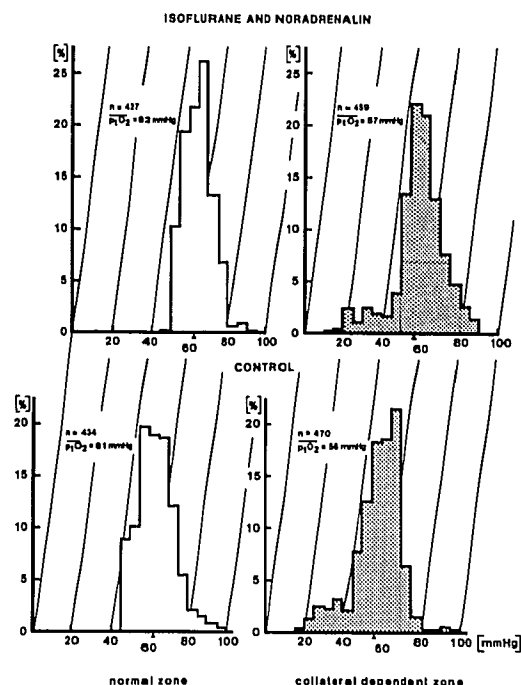
Title: OXYGENATION OF THE COLLATERAL FLOW DEPENDENT MYOCARDIUM DURING ISOFLURANE ANESTHESIA: ROLE OF CORONARY PERFUSION PRESSURE ?

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**INTRODUCTION:** We have reported recently that the volatile anesthetic isoflurane (ISO) reduces blood flow to and surface tissue oxygenation of the collateral dependent myocardium in dogs (1). As changes in myocardial blood flow (MBF) and tissue oxygenation ( $ptO_2$ ) in these experiments were accompanied by a reduction of mean arterial pressure (MAP), we could not differentiate definitely between a primary role of isoflurane on the coronary circulation or a secondary effect due to the reduced coronary perfusion pressure. We now report on a study, where coronary perfusion pressure was held constant during ventilation with the volatile anesthetic by intravenous infusion of norepinephrine (NE).

**METHODS:** Six dogs with body-weights between 30 and 40 kg had ameroid constrictors placed round their left anterior descending arteries (LAD) under aseptic conditions. After complete obstruction of the LAD, the animals were reanesthetized and underwent controlled ventilation. Anesthesia was maintained by the long acting narcotic piritramid (0.5 mg/kg  $\times$  h). The pericardium was opened through a left lateral thoracotomy and a catheter placed in the left atrium for radioactive microsphere injections (diameter 15 micron). Two 8-channel platinum electrodes (2) were placed on the surface of the beating left ventricle for surface tissue  $O_2$ -pressure recordings. One electrode was placed on the area supplied by the left circumflex artery (CX), the second on the myocardium distal of the occluded LAD. Control recordings of hemodynamic parameters, MBF and  $ptO_2$  were obtained and repeated after 45 minutes ventilation with end-expiratory 1.6-2.2 Vol% isoflurane, while MAP was kept close at its control value by infusion of NE. Correct positioning of both electrodes was verified when all determinations had been accomplished: This was done by lowering the MAP by isoflurane without infusion of NE;  $ptO_2$  on the normal CX area then remained constant, but decreased on the collateral flow dependent LAD area. The hearts were removed and the left ventricles divided into 160 tissue samples for determination of MBF. For statistical analysis the Wilcoxon tests for paired and unpaired observations were used. Values are expressed as mean  $\pm$  SE.  $p < 0.05$  is considered statistically significant.



**RESULTS:** The effects of ISO and NE on heart rate (HR), MAP, cardiac output (CO) and left ventricular  $O_2$  consumption are summarized in the first figure. For MBF to the healthy myocardium increased significantly during ISO and NE from  $1.1 \pm 0.1$  to  $2.8 \pm 0.2$  ml/min  $\times$  g. In the collateral dependent zone, only a slight and insignificant increase from  $0.8 \pm 0.1$  to  $1.3 \pm 0.3$  could be detected. MBF in the LAD area was significantly lower during ISO and NE than in the CX zone.  $ptO_2$  on the collateral dependent myocardium was slightly lower than on the CX area at control. Mean  $ptO_2$  remained essentially unchanged during ISO and NE: mean values were 62 mmHg on the normal myocardium and 57 mmHg on the collateral dependent myocardium, while arterial  $pO_2$  was about 100 mmHg. The summary histograms of all six animals are given together with the arterial  $pO_2$  values in figure 2.

**CONCLUSION:** In contrast to our previous study,  $ptO_2$  as well as MBF were not deteriorated in the collateral dependent myocardium when coronary perfusion pressure was kept close to its baseline value by infusion of NE during the application of isoflurane. We therefore conclude, that the disadvantageous effects of isoflurane are not caused by a direct influence on the coronary microcirculation, but rather by the lowered coronary perfusion pressure.

#### REFERENCES:

1. Conzen, P, et al., Anesthesiology 65: A5, 1986.
2. Kessler, M, et al., Anesthesiology 45: 184-197, 1976.

