TITLE: HALOTHANE AND ISOFLURANE EFFECTS ON MYOCARDIAL BLOOD FLOW DURING AORTIC CROSS CLAMPING IN DOGS WITH A CRITICAL CORONARY STENOSIS

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Introduction: Surgery of the aorta has become increasingly common in the past decade but operative surgical mortality for elective repair of intact abdominal aortic aneurysms persists at 3-9 %[1]. Half of these perioperative deaths are attributable to cardiac events. The deleterious effects of aortic cross clamping seem to be directly related to the increases in blood pressure, afterload and preload[2]. A great deal of attention has been given to anesthetic techniques that best preserve myocardial oxygen delivery while controlling myocardial oxygen demands. Use of the inhalational agents halothane and isoflurane.has been espoused to control the blood pressure and afterload increases seen with cross clamping[3]. Halothane is recognized to improve the oxygen supply/demand ratio. Its myocardial depressant effects may, however, be deleterious and it has been shown to worsen regional myocardial function in dogs. Isoflurane, despite its known in vitro myocardial depressant effects, does not decrease cardiac output, presumably due to a concurrent lowering of systemic vascular resistance. This effect on afterload is of potential benefit during aortic cross clamping; the recent evidence that isoflurane may induce myocardial lactate production or cause coronary steal in an animal preparation[4],may, however, prohibit its use in patients with coronary artery disease. We have investigated the alterations in regional myocardial blood flow and hemodynamic parameters during aortic cross clamping in dogs with a critical stenosis of the circumflex coronary artery under halothane or isoflurane anesthesia.

Methods: 22 acute mongrel dogs were anesthetized with Pentobarbital 30 mg/kg, endotracheally intubated and ventilated to maintain normal blood gas parameters. Catheters were placed to monitor mean aortic pressure (MAP), pulmonary artery pressure (PAP), cardiac output (CO), and to calculate systemic and pulmonary vascular resistance(SVR, PVR). A Millar micromanometer tipped catheter was placed into the left ventricle (LV) for measurement of LV pressure and its first derivative, dPdT. A left thoracotomy was performed and a left atrial line was placed for measurement of LA pressure and injection of radioactive microspheres for determination of myocardial blood flow (MBF). The left circumflex artery was dissected near its orgin and a doppler flow probe placed. A critical stenosis was created with a screw clamp such that resting flow was not diminished but the hyperemic response to a 5 second occlusion of the artery was abolished. Following 30 minutes for stabilization, Control (C) hemodynamic, metabolic and blood flow measurements were performed. A clamp was then placed on the descending thoracic aorta and all measurements were repeated (Control Crossclamp, CCX). The aortic clamp was removed and the dog permited to stabilize. The dog then received either halothane to .78% end tidal or isoflurane to 1.25% end tidal as determined with a mass spectrometer. After repeating all measurements (Halothane, H or Isoflurane,I) the aorta was again crossclamped and all measurements again repeated (Halothane Crossclamp, HCx or Isoflurane Crossclamp, ICx). Following completion of all data collection the dogs were sacrificed with KCl and the heart and kidneys removed and sectioned to determine regional blood flow. All data was analysized with student's t test to determine significance.

Results: There was a decrease in MAP during H and I compared to C and a decrease in CO during I but not H compared to C. There were no changes in SVR or PVR during H or I compared to C. The dramatic increase in MAP seen during CCx was significantly attenuated during HCx and ICx . CO decreased during all aortic crossclamping, but the decrease during ICx was greater than that seen from C to CCx. LAP was elevated during all periods of aortic crossclamping, but the increase was greater during ICX than during CCx . The fall in CO and the rise in LAP during HCx was comparable to that seen at CCx. HR was comparable at all time periods in all groups. There was a slight decrease in MBF during H anesthesis and HCx when compared to I anesthesia and ICx but there was no change from C to H or I or from CCx to HCx or ICx. We did not find any evidence of either regional or intramural "steal" at any intervention in either group.

Discussion: Halothane and isoflurane clearly attenuate the rise in blood pressure typically seen with aortic cross clamping. This attenuation of MAP seems to come at the expense of cardiac output and contractility, especially during isoflurane anesthesia. Myocardial blood flow was decreased during halothane anesthesia when compared to isoflurane and this decrease appeared greater in the normal zone than in the ischemic zone. There were no differences in MBF in any zone at any intervention when compared to control MBF. We certainly did not see clear cut evidence of either regional or intramural "steal" with either anesthetic. The myocardial depressant effects of either anesthetic as noted by decreased CO and dPdT and elevated LAP during HCx and ICx would seem to argue against use of either anesthetic during aortic crossclamping.

References:

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All values are means plus or minus standard error of mean