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TITLE: Halothane in Dogs with Left Ventricular Hypertrophy

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Introduction: It has been suggested, on theoretical grounds, that Halothane (H) might be a poor anesthetic choice in left ventricular-hypertrophy (LVH) secondary to valular aortic stenosis.[1] Some data indicates that stable LVH due to chronic pressure overload is associated with myocardial depression.[3] The addition of an exogenous agent with significiant cardiac depressant effects could result in hemodynamic deterioration especially if the outflow was severely obstructed. A dog model has been developed which produces animals with stable LVH[2] acceptably similar to the human condition. This study was initiated to examine the cardiovascular effects of Halothane in dogs with and without LVH secondary to supra-valvular aortic stenosis.

Methods: Groups of litter-mate mongrel pups underwent an aortic banding procedure at 5 weeks of age. Half of the animals had identical surgery without banding and served as controls. At 6 months of age the dogs were chronically instrumented.

Ten days after instrumentation anesthesia was induced with H:O_2, the trachea intubated and ventilation controlled. The dogs were studied at four H concentrations (0.85%, 1.3%, 1.6%, 1.9% end- tidal) in random sequence and then allowed to awaken. Each new end tidal state was allowed 30 minutes for equilibration. Controlled variables included P_aCO_2, arterial pH, body temperature, and H concentration. Recorded data included EKG, LV pressure, LVdP/dt, aortic and pulmonary artery pressure. Cardiac output was computed in duplicate by thermodilution and by the microsphere reference organ method. At sacrifice the hearts were separated into left and right ventricles and weighed.

Results: All banded dogs exhibited significant LVH as evidenced by LV wt./body wt., LV/RV wt., and systolic gradients across the stenosis (30 to 100 mmHg in the awake animal). Nine dogs with LVH and ten controls were studied. A summary of measured and calculated hemodynamic variables are shown in the Table. From 0.85% to 1.9% end-tidal H; MAP, CO, and SV declined 32%, 31%, and 31% respectively in the control group compared with 29%, 28%, and 27% in the banded animals. The index of contractile state, dP/dt, declined 43% in the normal dogs and 35% in those with LVH over the same concentration range. The Student T-test revealed changes with increasing H concentration within each group to be highly significant ($P<0.01$). There was no statistically significant difference in any parameter between the groups at a given H concentration. HR, SVR and LVEDP were essentially unchanged between and within the groups.

Hemodynamic Data

H%	0.85	1.3	1.6	1.9
(control)				
HR	102	99.3	100.0	100.2
MAP	77.8	67.2	57.3	52.3
CO	1.73	1.57	1.39	1.20
SVR	46.3	43.7	42.3	46.3
SV	17.0	16.0	14.3	11.8
dP/dt	15.4	12.8	10.7	8.7
LVEDP	5.0	4.7	4.7	5.6
(LVH)				
HR	108.2	101.9	102.1	104
MAP	83.1	70.3	64.9	59.4
CO	1.79	1.52	1.42	1.29
SVR	47.0	42.2	45.4	45.0
SV	16.6	15.2	13.9	12.2
dP/dt	15.5	13.2	11.4	10.0
LVEDP	6.5	6.1	5.8	6.7

HR(b/min), MAP(mmHg), CO(l/min), SVR(PRU's) SV(ml), dP/dt(arbitrary units), LVEDP(mmHg)

Discussion: In spite of similar cardiac depression at deepening H levels the dogs with hypertrophy were able to maintain cardiovascular stability as well as their normal counterparts. Although this result is remarkable, in light of the impressive myocardial thickening and stenotic narrowing, it should be noted that the LVH produced in this model was quite stable at the time of the investigation. The animals exhibited neither pulmonary congestion nor elevated LVEDP.

Hearts with concentric hypertrophy are susceptable to subendocardial ischemia under conditions of increased preload, afterload, contractile state and heart rate. The results of this study suggest that H may be safely used for general anesthesia for patients with stable LVH secondary to chronic pressure overload.

References: 1. Chambers, D.A. in Cardiac Anesthesia; Kaplan, J.A. Ed. 1979 pp 226 -2. O'Kane, H.O., et al: J. Thoracic Cardiovasc. Surg. 65:264 1973-3. Spann, J.F., Jr., et.al., Am. J. Med. Sci. 258:291, 1969