Title: EFFECT OF POSITIVE END-EXPIRATORY PRESSURE ON LEFT VENTRICULAR CONTRACTILITY

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Introduction. It is generally agreed that positive end-expiratory pressure (PEEP) reduces ventricular preload, but whether PEEP directly decreases left ventricular (LV) contractility remains controversial. A PEEP-related decrease in ventricular contractility has been attributed to pulmonary synthesis of a humorally-mediated negative inotropic factor[1], or secondary to LV coronary hypoperfusion.[2] Although traditional methods of assessing LV contractile performance are altered by changes in preload and afterload, the slope of the end-systolic pressure-volume relationship (termed E_{ES}) is a load-insensitive measurement of LV contractility. We, therefore, studied the effect of graded levels of PEEP on E_{ES} in a canine model.

Methods. Through a median sternotomy in 9 anesthetized dogs, transmural LV pressure was quantitated by on-line subtraction of pericardial pressure, measured by a fluid-filled catheter, from LV pressure, measured by a transducer-tipped catheter. Using the catheter impedance technique, LV volume was quantitated by a 7 Fr octapolar catheter connected to a signal conditioner-processor (Leycom Sigma-5, Oestgeest, The Netherlands). The end-systolic pressure-volume relationship was recorded during acute preload reduction from bicaval occlusion. Its slope, E_{ES}, and volume intercept at zero LV pressure (termed V_o) were determined by analysis of 7-10 pressure-volume loops. Changes in both E_{ES} and V_o represent sensitive indicators of myocardial contractile function. LV end-diastolic volume (EDV) and end-systolic volume (ESV) were determined by catheter impedance. The median sternotomy incision was reapproximated. After baseline measurements were recorded at 0 cmH_2O PEEP, increments of PEEP were added to 5, 10, and 15 cmH_2O. The effect of airway pressure on parallel conductance was determined by repeated injections of hypertonic saline at each measurement interval.

Data, expressed as mean ± SEM, were analyzed by analysis of variance for repeated measures. The study was approved by the Institutional Research Practices Committee.

Results. The various levels of PEEP had no effect on parallel conductance which allowed baseline values of parallel conductance to be used in the calculation of EDV and ESV (Table 1). Incremental levels of PEEP caused significant reductions in transmural LV end-diastolic pressure, EDV, and mean arterial pressure (MAP) (Table 1). The end-systolic pressure-volume relations were well-approximated by straight lines having correlation coefficients of 0.97 0.97, 0.96, and 0.95 at 0, 5, 10, and 15 cmH_2O PEEP, respectively. Overall, PEEP had no effect on ESV, V_o (Table 1) or E_{ES}. E_{ES} was 6.4 ± 1.7, 8.3 ± 1.8, 7.9 ± 1.7, and 9.2 ± 1.7 mmHg/ml with 0, 5, 10, and 15 cmH_2O PEEP, respectively.

Discussion. The predominant hemodynamic effect of PEEP was a reduction in LVEDV which is similar to that reported by others and attributed to a decrease in venous return and LV distensibility.[3] Moreover, in the range of PEEP tested, we found no evidence of depressed LV contractility as has been reported[1] from a circulating negative inotropic factor[1] or from myocardial ischemia.[2] In dogs with normal coronary anatomy, the PEEP-related hypotension was related to preload reduction without intrinsic abnormalities of LV contractility.

References.

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2. Tilley JG, Weisel RD, Evans PJ, et al: Hemodynamic and myocardial metabolic consequences of PEEP. Chest 88(4):496-502, 1985

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TABLE 1. HEMODYNAMIC EFFECTS OF PEEP

	PEEP			
	0 cmH_2O	5 cmH_2O	10 cmH_2O	15 cmH_2O
Parallel Conductance (ml)	24.9±6.1	23.2±5.0	24.5±4.6	21.1±5.6
MAP (mmHg)	98 ± 4	95 ± 4	76 ± 3[a]	63 ± 2[a]
LV end-diastolic pressure (mmHg)	4.1±.4	2.9±.7[a]	1.7±.6[a]	0.6±.5[a]
EDV (ml)	37.8±3.7	35.0±3.8[a]	28.7±2.9[a]	25.9±2.6[a]
ESV (ml)	22.9±3.1	23.4±3.0	19.3±2.6	18.0±2.5
V_o (ml)	3.4±4.8	1.4±4.5	3.8±2.7	8.0±3.2

[a] $p < 0.05$ compared with preceding value