Title: LEFT VENTRICULAR VOLUME RESPONSE TO PEEP

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Introduction. PEEP is an effective technique for management of respiratory failure. However, the application of PEEP may cause decreased cardiac output (CO). Despite the large volume of literature on PEEP, the mechanism reducing the CO remains controversial. On the basis of estimates of left ventricular transmural pressure, a decrease in LV contractility as the cause of decreased CO with PEEP has been proposed.[1] To determine if decreased contractility, rather than decreased LV preload is the cause of decreased CO, we measured radiographic LV volumes under three conditions: I CONTROL, II PEEP, and III PEEP with volume load to bring CO to the control value.

Methods. Nine (9) mongrel dogs were anesthetized with pentobarbital 30 mg/kg, 70% N_2O, 30% O_2, paralyzed with pancuronium, and mechanically ventilated to $PaCO_2$'s of 39±4. Temperature and hematocrits were maintained. Blood 15 ml/kg was removed and stored for reinfusion. Ringers lactate 45 ml/kg was administered concurrently to maintain blood volume. Atrial pacing was instituted to maintain a constant heart rate. The dogs were placed RAO in supine position and studied under three conditions: I CONTROL, II PEEP, 15 cm H_2O, and III PEEP + VOLUME load to return CO to control. The following variables were observed or calculated and recorded via appropriate physiological monitoring devices and/or standard formulas: wt, BSA, hematocrit, temperature, CO, CI, SV, SI, MAP, SVR, CVP, PAP, PADP, $\overline{PAP}$, $\overline{PCWP}$, PVR, LVEDP, esophageal pressure (peso), transmural pressures (TM), airway pressure, respiratory rate, CO_2ET, VT, airflows, total compliance, blood gases, LVEDV, LVESV, SV, and EF. The ventricular volumes were obtained by single plane cine, recorded in order that single frame sinus beats could be obtained in end-systole and end-diastole, traced and volumes calculated by the Dodge[2] regression formula. Paired t tests were used to calculate significance.

Results. Summarized in Table 1.

As expected with the application of PEEP cardiac output decreased approximately 25%, associated with a decreased stroke index both as measured radiographically and as calculated from hemodynamic data. These values returned to control values with volume loading. Upon application of PEEP, LV filling, as estimated by end-diastolic volume, decreased substantially, and with volume loading increased to control levels. Ejection fraction was unchanged with the application of PEEP, and there was a small increase in EF with volume loading. Thus it is apparent that the decreased cardiac output with PEEP is solely a result of decreased preload.

There were small and insignificant changes in the transmural LVEDP.

Table 1

	I CONTROL	II PEEP	III PEEP+VOLUME	SIGNIFICANCE
CI	3.5	2.45	3.5	†#
SI(xray)	20.8	15.4	22	†#
SI	20.8	14.1	21	†#
LVEDVI	42.3	30.6	39	†#
LVESVI	21.7	15.6	16.9	†
EF	.49	.49	.56	
Peso	-1.6	1.9	2.4	†θ
TMcvp	4.6	6	7.2	
TMpcwp	6	7	9.9	θ
TM1vedp	6.6	6	7.8	
MAP	132	125	134	#
LVSWI	35.4	22.9	36	†#

SD omitted for clarity, pressures in torr, $p<.005$, †(I vs II), #(II vs III), θ(I vs III)

Discussion. The transmural LVEDP is calculated as the difference between LVEDP and esophageal pressures. Both LVEDP and esophageal (or pleural) pressures are difficult to measure with precision and the difference between these two pressures is likely to be imprecise. However at low volumes, left ventricular filling is known to vary widely with small differences in LVEDP, and we assume the lack of correlation of transmural LVEDP with diastolic volume reflects these considerations, although a change in LV compliance with PEEP cannot be precluded.

We conclude that there is no change in LV function with PEEP and that the decreased cardiac output is due to decreased preload. However, the decreased preload is not readily detected by hemodynamic measurements. The cause of the decreased preload with PEEP is presumably related to RV filling or preload, and cannot be precisely determined from this study, but volume loading with PEEP restores LV preload and cardiac output to control levels without hemodynamic evidence for RV failure.

References

1. Liebman, S.S., Patten, M.T., Manny, J., et al. The Mechanism of Depressed Cardiac Output on PEEP. Surg. 83 (5): 594-598, 1978.
2. Dodge, H.T. Determination of LV Volume and Mass. Rad. Clin. North Am. IX (3), 1971.