

Title : THE EFFECTS OF PEEP ON LUNG FLUID AND PROTEIN EXCHANGE DURING LUNG VASCULAR INJURY

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Introduction. Studies indicate both increase and decrease in the extravascular lung water content after the application of positive end-expiratory pressure (PEEP). The present study examines the effects of PEEP on lung transvascular fluid and protein exchange in normal lungs and after lung vascular injury and pulmonary edema induced by alloxan.

Methods. The study was made in anesthetized (halothane) sheep since it was possible to collect pure lung lymph (i.e., the net lung fluid and protein filtrate) after cannulating the efferent duct of the caudal mediastinal node (1). Catheters were passed into the main pulmonary artery and into the most dependent portion of the left lower lobe to measure the pulmonary arterial (P_{pa}) and pulmonary arterial wedge (P_{pw}) pressures. Pulmonary blood flow (\dot{Q}_L) was measured by thermodilution and pulmonary vascular resistance (PVR) was calculated. Arterial oxygen tension was maintained in the normal range during the experiment. We used the following protocol: (i) 2 hrs baseline period; (ii) 1 to 2 hrs of PEEP (20 cm H₂O) until a new steady-state lymph flow was achieved; (iii) alloxan (200 mg/kg) until a new steady-state; (iv) PEEP (20 cm H₂O) until a new steady-state. The lung lymph flow (\dot{Q}_{lym}) was determined every 15 minutes and the protein concentrations of simultaneously collected plasma (P) and lymph (L) samples were determined. The lung transvascular protein flow was calculated by $\dot{Q}_{lym} \times L$ protein concentration.

Results. The effects of PEEP on steady-state pulmonary hemodynamics and on \dot{Q}_{lym} , L/P and protein concentration ratio are indicated below. The data are shown as mean \pm 1 SEM.

	Base- line	PEEP 20cm H ₂ O	Alloxan Base- line	PEEP 20cm H ₂ O
\dot{Q}_{lym} ml/hr	5.68 $\pm .78$	4.70 ± 1.09	12.54 ± 2.02	5.49+ $\pm .58$
L/P ratio	.70 $\pm .03$.74 $\pm .06$.87 $\pm .06$.84 $\pm .04$
Lymph pro- tein flow g/hr	26.49 ± 4.84	23.11 ± 7.13	61.43 ± 9.67	26.15+ ± 6.19
P_{pa} mm Hg	20.0 ± 1.2	26.2* ± 1.7	17.1 ± 1.4	20.4 ± 2.9
P_{pw} mm Hg	4.9 $\pm .1$	6.1* ± 1.0	4.9 $\pm .7$	6.2+ $\pm .3$

\dot{Q}_L l/min	2.41 $\pm .16$	1.60* $\pm .34$	1.79 $\pm .19$	1.07+ $\pm .17$
PVR mm Hg/l/min	7.33 ± 1.0	14.81* ± 2.44	7.66 $\pm .58$	15.52+ ± 1.77

* different from baseline ($p < 0.05$)

+ different from alloxan baseline ($p < 0.05$)

Discussion. The findings that PEEP in the normal lung had little effect on \dot{Q}_{lym} and lung lymph protein flow are consistent with observations at 10 cm H₂O (2). The unchanged fluid and protein fluxes may be due to the transmission of the high airway pressure to the interstitium and lung microvessels so that increase in the interstitial hydrostatic pressure is matched by an increase in the microvascular hydrostatic pressure (2). Unlike the normal lung, PEEP during lung vascular injury resulted in decreases in \dot{Q}_{lym} and protein flow to baseline levels. The different response cannot be explained by differences in the decrease in vascular surface area since the increases in PVR with PEEP in the normal and injured conditions were similar. The data may be explained by a greater transmission of the high airway pressure to the edematous lung interstitium due to decreased interstitial compliance, so that the fluid filtration pressure is reduced during PEEP. Another possibility is that the high airway pressure is not transmitted to the pulmonary microvessels because they are less compressible during edema, so that microvascular pressure does not increase as in the normal lung to match the increase in the interstitial hydrostatic pressure, thereby also resulting in decreased filtration pressure during PEEP. The unchanged pulmonary arterial pressure during PEEP with vascular injury supports the latter possibility. Therefore, the data indicate that PEEP decreases \dot{Q}_{lym} and lymph protein flow during edema produced by lung vascular injury.

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References

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