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 Title : PEEP AND LUNG WATER MEASUREMENTS AFTER OLEIC ACID
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Introduction: Previous research has suggested that positive end expiratory pressure (PEEP) decreased lung water directly by increasing interstitial pressure or decreasing right ventricular filling pressure. We have applied an *in vivo* double indicator (thermal and indocyanine green) dilution technique^{1,2} to measure extravascular lung water (EVLW) in an oleic acid model of pulmonary edema. The objectives of this study were to: (1) evaluate the reliability of this technique in non-cardiogenic pulmonary edema; and (2) measure the effect of various levels of PEEP on EVLW as reflected by extravascular thermal volume (EVTV).

Methods: Nine mongrel dogs were anesthetized with pentobarbital and intubated in the supine position. The animals were divided into a control group (4) and a PEEP group (6). They were ventilated at a constant tidal volume (15 cc/kg) while the respiratory rate was adjusted to maintain P_{CO_2} between 35 and 45 torr. The F_{I,O_2} was 1.0. PEEP was applied by submersing the expiratory limb of the ventilator circuit under varying columns of water. A 20cm 5F thermister-tipped catheter was placed via cutdown in the femoral artery. A triple lumen pulmonary artery catheter was passed from the femoral vein. A central venous catheter was inserted in the internal jugular vein. Femoral and pulmonary arterial pressures, and ECG were recorded continuously. Arterial and mixed venous blood gases, hemoglobin and EVTV were determined sequentially at zero time (baseline), 15 min after oleic acid infusion, and at 30 min intervals thereafter until sacrifice at four hours. After baseline values were obtained, oleic acid, 0.18 cc/kg, was infused into the right atrium over 15 min. Normal saline was infused to establish an initial pulmonary capillary wedge pressure at 4-8 torr, and thereafter, in order to maintain the mean arterial pressure >80 torr. The expected increase in EVTV reached a plateau by 120 min in all animals. PEEP was added to the experimental group 150 min after oleic acid administration. EVTV measurements in the PEEP group were made 30 min after each change in end expiratory pressure. EVTV was measured by injecting 10 cc of cold (0°) green dye (2 mg) in the right atrium. Indicator dilution curves measured simultaneously via the femoral artery catheter were analyzed by a computer. EVTV was derived by the mean transit times of the two curves. Following the final data collection the animals were immediately sacrificed and the lungs removed. The EVLW was then measured by gravimetric technique and the values compared to pretermina-

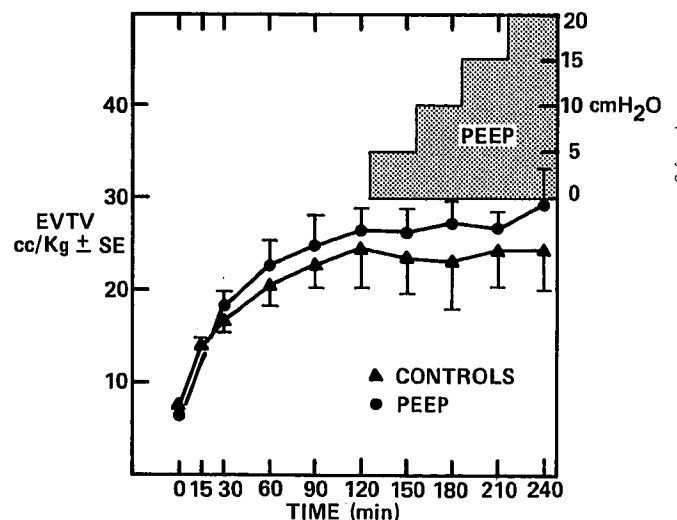
tion EVTV. The t-test was utilized to analyze the data at a $p < .05$ level.

Results: The initial EVTV for the two groups was the same and increased more than threefold at 120 min (figure). During PEEP therapy (final two hours of experiment) shunt fraction decreased ($Q_{san}/Q_t = .17$ vs $.53$, $p < .01$) in treated vs. control animals. During these two hours both mean EVTV (27 vs 24 cc/kg) and mean WEVTV/Wtime (.016 vs .015) between PEEP and control animals were not significantly different. Pretermination EVTV (cc/kg) was compared to EVLW (cc/kg) for oleic acid animals and 11 normal animals. The following linear regression was derived:

$$EVTV = 1.3 (EVLW) + 2.1 \quad (r = .96)$$

Discussion. PEEP does not alter extravascular lung water in this model of capillary leak syndrome. Improvement in oxygen gas exchange results from improvement in ventilation-perfusion mismatch. Since the slope of the EVTV curve was slightly positive and identical for the two groups, there is no evidence that PEEP retarded further fluid accumulation.

Although the variance increased with extreme degrees of pulmonary edema, this appears to be a reliable *in vivo* technique for quantifying EVLW in non-cardiogenic pulmonary edema.



Reference

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