Title : LONG TERM PEEP THERAPY AND LUNG WATER IN OLEIC ACID ARF

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Introduction. Positive end-expiratory pressure (PEEP) has become the cornerstone of therapy in acute respiratory failure (ARF), improving oxygenation, re-establishing reduced lung volumes, and decreasing intrapulmonary shunt. The question that remains unanswered is whether PEEP can modify the basic pathophysiological process.

The objective of this study was to determine whether the administration of PEEP to dogs with severe ARF produced by oleic acid would decrease extravascular lung water (EVLW).

Methods. 30 mongrel dogs were used, and their tracheas intubated under pentobarbital Na anesthesia. A thermodilution balloon-tipped catheter and an arterial line were inserted. Hemodynamic measurements and blood samples for mixed venous and arterial blood gases were obtained sequentially. Volumes of fluids required in 24 hours were similar in all groups. ARF was produced by injecting oleic acid, 0.1 ml/kg, into a central venous line.

The dogs were assigned to three groups:

1. Control dogs (N = 10) were ventilated with intermittent positive pressure ventilation (IPPV) and FIO_2 of 0.5.
2. ARF-IPPV (N = 10). In this group, when the PaO_2 decreased to < 50 torr, the FIO_2 was raised from the baseline concentration of 0.5.
3. ARF-CPPV (continuous positive pressure ventilation) (N=10). In this group, when the shunt was higher than 15% PEEP was administered in increments, with the goal of decreasing the shunt to the preselected value. FIO_2 was kept constant (0.5).

At the end of twenty-four hours, blood samples for hemoglobin (Hb blood), mixed venous and arterial blood gases, as well as hemodynamic measurements, were performed. The dogs were then sacrificed and the lungs quickly excised. The total lung wet weight (TLWW) was recorded. Then twenty liters of normal saline were perfused through the pulmonary artery. The recovered volume (V_R) and its hemoglobin concentration (Hb_R) were determined. Finally, the lungs were allowed to dry until isogravimetric state was reached (total lung dry weight = TLDW). The following calculations were performed:

Total lung water (TLW) = TLWW - TLDW.

Extravascular lung water (EVLW) = TLW - LBW (lung blood weight), where

$$LBW = \frac{V_R \times Hb_R \times 1059}{Hb\ Blood}$$

Results. 1. Oleic acid produced severe ARF manifested by a three-fold increase in EVLW compared with the control group.

2. The dogs with ARF-CPPV (10 ± 4 mm Hg) had a lower Qsp/Qt than the group with ARF-IPPV ($p < 0.001$).

3. No significant difference in EVLW was noted between the two groups with ARF.

4. Pulmonary capillary wedge pressure and cardiac output at the end of the experiment were similar for all groups.

Conclusion. In severe ARF due to oleic acid, although PEEP effectively reduced intrapulmonary shunt this effect did not correlate with a concomitant change in EVLW measured after a prolonged period of respiratory support. Other etiological models of ARF have to be studied before a final conclusion is made concerning whether PEEP is a symptomatic or a pathophysiological treatment.

	CONTROL	ARF-IPPV	ARF-CPPV
TLW(gr)	210 (33)	572 (286)	659 (76)
EVLW(gr)	145 (35)	540 (169)	590 (86)
TLW(gr kg^{-1})	10.3 (1.3)	27.7 (5.9)	27.7 (4.4)
EVLW(gr kg^{-1})	7.08 (1.5)	24.6 (6.7)	25.0 (4.6)

p: CONTROL vs. ARF-IPPV or ARF-CPPV < 0.001.
ARF-IPPV vs. ARF-CPPV = N.S.

	CONTROL	ARF-IPPV	ARF-CPPV
Qsp/Qt (%)*	8.36 (2.68)	47.4 (18.0)	28.0 (15.3)
PCWP (mm Hg)	6.05 (2.5)	5.2 (2.1)	6.7 (4.2)
CO (L/M)	2.36 (0.7)	2.9 (1.3)	2.4 (0.9)

* Control vs. ARF-IPPV or ARF-CPPV, $p < 0.001$; ARF-IPPV vs. ARF-CPPV, $p < 0.001$.

Values expressed as a mean ± (standard deviation).

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