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Title : FUROSEMIDE AND LUNG WATER DURING EXPERIMENTAL ARF

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Introduction. Although the use of diuretics is a well-established therapy for pulmonary edema secondary to left ventricular failure, its beneficial effects on pulmonary edema secondary to acute respiratory failure (ARF) are questionable.

The objective of this study was to determine whether furosemide may decrease the lung water accumulation during experimental ARF.

Methods. Twenty mongrel dogs received pentobarbital (30 mg/kg), their tracheas were intubated and connected to a Harvard ventilator.

A left thoracotomy was performed in each dog, and the left lower lobe (LLL) pulmonary artery and vein were cannulated for pressure recording. Then three plastic pads with a string were attached to the LLL (cyanoacrylate ester adhesive) with the purpose of hanging the lobe for continuous in situ weight determinations.

LLL pulmonary artery and vein pressure were kept constant through the experiments. When there was need to decrease PA pressure, partial constriction of the artery was produced with a band placed around it. When an increase in pressure was necessary, the right lung flow was diverted to the left by inflation of a balloon in the right main pulmonary artery (Swan-Ganz catheter).

The dogs were divided into three groups:

1. Control group (N=4).
2. ARF group (N=8)
3. ARF-L (L=furosemide) (N=8)

After an hour for stabilization, the experiment began (time 0): 16 dogs (ARF; ARF-L) received oleic acid (OA) 0.01 ml/gm lobe injected into the LLL pulmonary artery. One hundred and twenty minutes after the administration of OA, 8 dogs (ARF-L) received furosemide 1 mg/kg. The LLL weight rate of change (Δweight in %) was determined:

$$\frac{\text{actual weight - control weight}}{\text{control weight}}$$

At the end of the experiment (300 minutes) the dogs were sacrificed. The extravascular lung water (EVLW) of the LLL was determined by the method described by Pearce et al (1) and it was expressed as the corrected ratio for EVLW of wet weight/dry weight.

The data were analyzed with Student's t-test for unpaired observations, linear regression, and covariance analysis.

Results. 1. 120 min after OA administration, there was a significant weight gain in both groups with ARF (ARF $\bar{X}$ 0.40 ± SD 0.13%; ARF-L $\bar{X}$ 0.43 ± SD 0.06%) as compared with the control group ($\bar{X}$ 0.02 ± SD 0.02%) with $p < 0.001$ for both ARF and ARF-L vs. control. However, no difference was found between the two ARF groups.

2. After the administration of furosemide until the end of the experiment no difference was found in Δweight between the two groups with ARF:

	$\bar{X}$	± SD	p
ARF (180 min)	0.46	(0.12)	0.99
ARF-L (180 min)	0.46	(0.07)	
ARF (240 min)	0.54	(0.06)	0.87
ARF-L (240 min)	0.54	(0.03)	
ARF (300 min)	0.58	(0.09)	0.77
ARF-L (300 min)	0.57	(0.09)	

When the regression lines of time (120-300 min) vs Δweight for ARF and ARF-L were compared it was found that the slopes ($F:1.36_{1,300}$) and elevations ($F:0.028_{1,301}$) were not significantly different.

3. The EVLW (wet/dry) showed

	CONTROL	ARF	ARF-L
$\bar{X}$	4.02	7.49	7.54
± SD	(0.08)	(0.82)	(0.94)

ARF vs. ARF-L $p = 0.91$

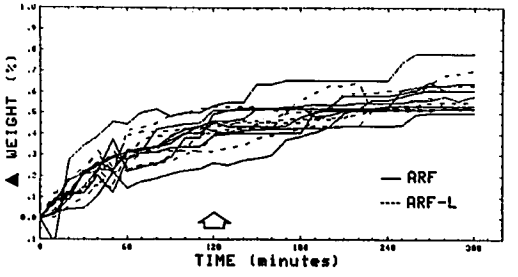
ARF or ARF-L vs. Control $p < 0.001$.

Conclusions. In this anesthetized, oleic acid-induced ARF model, furosemide did not alter the rate of increase of lung water accumulation when changes in vascular pressures were controlled.

It is postulated that if furosemide has any effect on pulmonary edema secondary to ARF, it may be due to changes in vascular pressure.

Reference.

1. Pearce ML, Yamashita J, Beazell J: Measurement of pulmonary edema. Circ Res 16:482-488, 1965.



Δweight vs. time for individual cases. The arrow indicates the administration of furosemide in the ARF-L group.