Title : MECHANISM OF RE-EXPANSION EDEMA IN RABBITS

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Introduction. Re-expansion edema is a clinical syndrome consisting of unilateral edema in a lung which is rapidly expanded following a period of prolonged collapse. We have developed a model of this syndrome in rabbits[1] by rapidly re-expanding lungs previously collapsed for one week by pneumothorax. The following experiments were done to determine whether altered pulmonary vascular permeability might be a contributing factor in the causation of this type of pulmonary edema. The method measures apparent concentration of ^{131}I-albumin (RISA) in extravascular, extracellular lung water (EVECW) as an indicator of pulmonary vascular permeability using ^{24}Na as an indicator of EVECW volume.

Methods: All experiments were performed in rabbits, 2.5-3.5 kg, of both sexes. ^{24}Na-NaCl, and RISA were injected intravenously into rabbits. When steady-state levels of isotope were achieved in plasma and lung, animals were divided into four groups: Group 1. Eight normal, awake animals breathed room air spontaneously for two hours. Group 2. In five animals with chronic right-sided pneumothoraces (6-8 days), lungs were reinflated by applying negative pressures of -100 torr to a chest tube communicating with the right pleural space. Animals were anesthetized with pentobarbital and breathed 100% oxygen spontaneously. Group 3. Eight awake animals with chronic pneumothoraces breathed room air spontaneously for two hours. Group 4. Ten awake animals with chronic pneumothoraces received oleic acid, 0.09 ml/kg intravenously and then breathed room air spontaneously for two hours. This latter group was included to determine how atelectasis might alter the effects of an immediate, diffuse pulmonary vascular injury caused by oleic acid. All animals were sacrificed and the amount of water, blood and radioactivity determined in excised lungs. EVECW (ml/gm dry lung) was calculated from the [^{24}Na cpm/gm dry lung/^{24}Na cpm/ml plasma]. The apparent concentration of RISA in EVECW compared to plasma ($[RISA]_L/[RISA]_{Pl}$ was obtained from [RISA cpm/gm dry lung/^{24}Na cpm/gm dry lung]/[RISA cpm/ml plasma/^{24}Na cpm/ml plasma].

Results. The results are shown in Fig. 1 and Table I. In Fig. 1, the results are compared with those obtained in previous experiments in which pulmonary edema was produced by oleic acid or increased left ventricular diastolic pressure, as models of edema due to vascular injury and increased hydrostatic pressure respectively.

Discussion. The results in Fig. 1 show a very significant increase in RISA concentration in EVECW in re-inflated lungs (Group 2) which is greater than that seen following increased hydrostatic pressure, or vascular injury due to oleic acid. This suggests increased pulmonary vascular permeability as the cause of re-expansion edema. Atelectasis alone (Group 3) caused no increase in EVECW or RISA concentration. In Table I, the effects of oleic acid in the collapsed right lung are not significantly different from the left lung showing that lung collapse does not mask the effects of a diffuse pulmonary injury. These experiments demonstrate that re-expansion edema is due to altered pulmonary vascular permeability, and is caused by mechanical stresses applied to the collapsed lung during re-expansion.

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References.

1. Pavlin J, Cheney FW: Unilateral pulmonary edema in rabbits after re-expansion of collapsed lung. J Appl Physiol 47:31, 1979

FIGURE 1

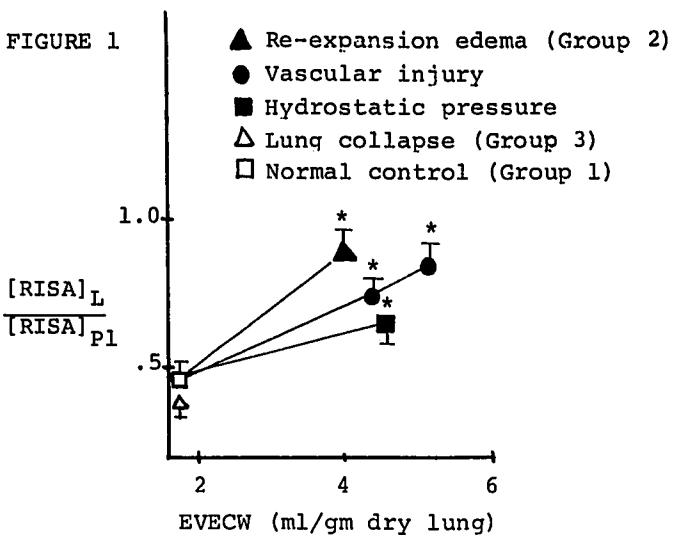

TABLE I
EFFECT OF OLEIC ACID IN GROUP 4

	Right	Left	Group 1
EVECW ml/gm dry lung	4.18* SD±1.2	4.40* ±1.7	2.04 ±.30
Total lung H_2O (gm/gm dry lung)	5.80* ±1.1	5.94* ±1.5	4.45 ±.32
$[RISA]_L/[RISA]_{Pl}$	.82* ±.19	.76* ±.16	.51 ±.22

*p < .05 compared to Group 1 controls