Title : FAILURE OF STEROIDS TO IMPROVE HEMORRHAGIC PULMONARY EDEMA

Authors : E. Goldman, M.D., T. de Campo, M.D., and J. A. Aldrete, M.D.

Affiliation: Department of Anesthesiology, University of Colorado Medical Center, Denver, Colorado 80262

Introduction. The effectiveness of massive doses of steroids in the treatment of acute respiratory insufficiency both in animals and in humans is controversial. This experiment was carried out in order to determine whether pharmacologic doses of methylprednisolone (MP) would influence the water content of the lung (LW) as well as the physiological parameters during acute hemorrhagic pulmonary edema induced by oleic acid (OA) in a 24 hour experiment.

Methods. Twenty-three dogs anesthetized with pentobarbital were intubated and mechanically ventilated with a Harvard pump (FIO_2 0.5). A pulmonary artery thermodilution catheter and direct arterial pressure monitoring were used. The dogs were divided into three groups. Four dogs were the control group. Nineteen dogs received OA, 0.1 ml/kg, which was followed by MP 30 mg/kg in 10 dogs. The remaining nine dogs became the OA-untreated group. Hemodynamic parameters as well as arterial and mixed venous blood samples were obtained at baseline and then sequentially for a 24-hour period. At the conclusion of the study the lungs were excised. LW was obtained by the wet weight/dry weight method. All values were corrected for the residual blood in the lungs, and expressed as ml of water/g dry lung.

Results are summarized in Table I. There was no significant difference between OA-treated and OA-untreated groups in any of the parameters studied, either at baseline or at the end of the study.

1. In both OA groups LW, PVR, PaO_2 and $\dot{Q}s/\dot{Q}t$ differed significantly from control.
2. There was no significant difference between OA-treated and OA-untreated groups in any of the parameters studied.
3. In both OA groups PaO_2 decreased ($p < .001$) and $\dot{Q}s/\dot{Q}t$, $\overline{PAP}$ and PVR increased with time ($p < .001$). The changes observed in $\dot{Q}s/\dot{Q}t$ did not correlate with cardiac output alterations.

TABLE I.

	Control	OA-untreated	OA-treated
BASELINE			
$\overline{PAP}$ torr	14±2	14.4±1.7	12.8±1
PCWP torr	4±.8	4.9±.7	4.6±.6
$\dot{Q}t$ L/min	3.0±.3	3.5±1.4	2.8±.4
PVR*	209±74	214±91	280±169
PaO_2 torr	291±19	246±17	179±16
$\dot{Q}s/\dot{Q}t$	.07±.02	.07±.01	.11±.02
LW ml/g	--	--	--
AFTER 24 HOURS			
$\overline{PAP}$ torr	13±6	25±2	26±3
PCWP torr	6.0±.9	5.6±.6	4.2±.9
$\dot{Q}t$ L/min	2.6±.5	2.9±.3	2.2±.4
PVR*	330±29	617±75	832±136
PaO_2 torr	220±16	62±5	48±6
$\dot{Q}s/\dot{Q}t$	.12±.05	.50±.05	.58±.06
LW ml/g	4.8±1.1	9.2±.6	11±1.1

Values are mean ± SEM.

* dynes/sec/cm^{-5}

4. $\dot{Q}t$ decreased ($p < .001$) and $\overline{PAP}$ increased ($p < .001$) as PVR increased during the experiment, reflecting the marked restriction in pulmonary vasculature. This finding was similar in the two OA groups.

Conclusions. 1. MP did not change the course of severe lung injury, as could be assessed by the hemodynamic parameters and shunt fraction. 2. Lung water content in severe, long-term edema was unaffected by early administration of high doses of MP.