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Title: PHYSIOLOGIC EFFECTS OF KEROSENE ASPIRATION

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Introduction. Aspiration of hydrocarbon causes significant morbidity and mortality. Symptoms occur rapidly,[1,2] and pulmonary surfactant activity is altered;[3] however, the pathophysiology and response to therapy have not been described consistently in the literature. We investigated the physiologic effects of the aspiration of hydrocarbon in dogs.

Methods. Ten mongrel dogs were anesthetized with sodium pentobarbital, 25 mg/kg, intravenously, and intubated with cuffed endotracheal tubes. Lactated Ringer's solution was infused at approximately 4 ml/kg/hr. Additional anesthesia was given as needed. Catheters were inserted to measure systemic arterial (BP), central venous (CVP), pulmonary artery (PAP), pulmonary artery occlusion (PAOP), and intrapleural (Ppl) pressures. Heart rate (HR), respiratory rate (f), and temperature were monitored. Cardiac output ($\dot{Q}_t$) was determined by using a thermodilution technique and arterial and mixed venous blood samples were analyzed for pH, pCO_2, and pO_2 and hemoglobin content. Intrapulmonary shunt fraction ($\dot{Q}_{sp}/\dot{Q}_t$), arterial-venous oxygen content difference ($AVd0_2$), body surface area (BSA), cardiac index (CI), pulmonary vascular resistance (PVR), systemic vascular resistance (SVR), and stroke volume (SV) were calculated.

After control data were obtained, the animals aspirated kerosene, 0.5 ml/kg, through a catheter inserted into the endotracheal tube. While the animals continued to breathe room air spontaneously, all measurements and calculations were repeated 15, 30, 45, 60, 90, 120, 150, 180, and 240 min after aspiration. The data were analyzed by using Student's t-test for paired data. A level of probability (p) less than 0.05 was accepted as statistically significant; all values are reported as mean $\pm$ 1 S.D.

Results. The following changes occurred within 15 min: the $\dot{Q}_{sp}/\dot{Q}_t$ increased from .17 $\pm$ .07 to .59 $\pm$ .14 and remained elevated throughout the study; heart rate decreased from 159 $\pm$ 21 to 123 $\pm$ 20 bpm and BP from 122 $\pm$ 14 to 90 $\pm$ 17 torr, both returned toward control after 45 min; respiratory rate increased from 25 $\pm$ 9 to 85 $\pm$ 22 and remained elevated; respiratory acidosis occurred, but pHa and $paCO_2$ returned toward control after 60 min; arterial pO_2 decreased from 78 $\pm$ 9 to 41 $\pm$ 11 torr and did not change significantly over the next four hours; intrapleural pressure decreased from -4.0 $\pm$ 1.5 to -5.4 $\pm$ 2.5 torr and remained at this level. There was no change in PAP. No significant changes occurred in PAOP, PVR, SVR, $AVd0_2$, SV, $\dot{Q}_t$, or CI during the first 60 min, but PAOP decreased and PVR increased significantly after that. Late in the study, SVR and $AVd0_2$ significantly increased, while $\dot{Q}_t$, CI, and SV decreased significantly.

Discussion. The aspiration of kerosene caused severe pulmonary dysfunction resulting in intrapulmonary physiologic shunting of greater than 60% of the cardiac output and in severe hypoxemia. Marked tachypnea, combined with respiratory acidosis and decreased intrapleural pressure, suggests that pulmonary compliance decreased. Cardiovascular changes were minimal.

References.

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