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Title : COLLATERAL VENTILATION AND PEEP IN NORMAL AND EDEMATOUS LUNGS OF DOGS

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Introduction. Total obstruction of segmental or subsegmental bronchi in excised lungs of dogs does not cause the obstructed segment to collapse. Ventilation distal to the obstruction is preserved and atelectasis is prevented by collateral respiration. Pores of Kohn and interbronchiolar and bronchial-alveolar communications have been described, but which pathway provides the route of collateral ventilation is unknown. Resistance to gas flow through small airways (R_{saw}) is normally minimal compared to that through collateral channels (R_{coll}). However, when R_{saw} is increased, R_{coll} may decrease so that gas would flow preferentially through collateral channels. Perry, et al.[1] have shown that this occurs in "pink-puffer" emphysema. The purpose of this study was to determine the effect of 10 cm H_2O positive end-expiratory pressure (PEEP) on R_{coll}, R_{saw}, and gas exchange of dogs before and after acute hemorrhagic pulmonary edema induced by oleic acid.

Methods. Nine dogs (14-23 kg), anesthetized with sodium pentobarbital, were ventilated with room air, a tidal volume of 20 ml/kg, and a rate of 12 breaths/min. A quadrilumen pulmonary artery catheter and a femoral artery catheter were placed transcutaneously. During each study period, heart rate, blood pressure, pulmonary artery pressure, pulmonary artery occlusion pressure, and cardiac output were measured. Arterial and venous blood gases were sampled for calculation of physiologic shunt fraction ($\dot{Q}_{sp}/\dot{Q}_t$) and alveolar-arterial oxygen tension gradient (A-aDO2) by using standard formulae. Animals were studied before and 30 min after central venous injection of oleic acid, 0.02 ml/kg. Measurements and calculations were obtained before, during, and after application of 10 cm H2O PEEP, before and after injection of oleic acid. The technique of measuring R_{coll}, described by Hilpert[2] and modified by Smith et al.,[3] allows measurement of R_{saw} in the same lung segment. A balloon-tipped catheter with two ports distal to the balloon was wedged in a dependent segment of either the right or the left lower lung lobe. One lumen allowed constant infusion of gas ($\dot{D}_{coll}$) and the second allowed measurement of pressure distal to the tip of the catheter (P_b). $\dot{D}_{coll}$ was terminated; P_b decayed exponentially as gas left the isolated segment through collateral channels. Measurements were repeated in triplicate for each study. R_{saw} and R_{coll} were calculated by using the methods described by Smith et al.[3] Analysis of variance and Duncan's multiple range test tested the significance of differences ($p < 0.05$).

Results. Oleic acid decreased PaO2 from 86 ± 3 to 64 ± 6 torr and concomitantly increased A-aDO2 from 35 ± 2 to 53 ± 5 torr and $\dot{Q}_{sp}/\dot{Q}_t$ from 0.15 ± 0.03 to 0.29 ± 0.05. With PEEP, the PaO2 increased to 78 ± 6 torr, A-aDO2 and $\dot{Q}_{sp}/\dot{Q}_t$ returned to control values. Cardiac output decreased after oleic acid was infused, but returned to control values. PEEP decreased cardiac output from 3.3 ± 0.4 to 2.1 ± 0.3 L/min and PaCO2 increased from 41 ± 3 to 48 ± 3 torr. R_{coll} decreased from 27.3 x 10^3 ± 9.6 x 10^3 to 2.1 x 10^3 ± 0.7 x 10^3 cm H_2O/L/sec after PEEP. After oleic acid, R_{coll} increased to 48 x 10^3 ± 18 x 10^3 cm H_2O/L/sec. PEEP decreased R_{coll} to 1.1 x 10^3 ± 0.4 x 10^3 cm H2O/L/sec. $\dot{D}_{coll}$ increased from 10 x 10^{-4} ± 5 x 10^{-4} to 40 x 10^{-4} ± 8 x 10^{-4} L/sec with PEEP. After oleic acid, $\dot{D}_{coll}$ decreased to 12 x 10^{-4} ± 6 x 10^{-4} L/sec and, after PEEP, increased to 60 x 10^{-4} ± 4 x 10^{-4} L/sec. R_{saw} did not change significantly either after oleic acid or PEEP.

Discussion. In normal lungs, PEEP decreased R_{coll} and, thus, more gas tended to flow through collateral channels. Intravenous injection of oleic acid produces a diffuse pulmonary injury causing collateral channels to close, which PEEP can reverse. Thus, part of the benefit of PEEP for gas exchange in diffusely injured lungs might be respiration through collateral ventilation. Oleic acid did not change R_{saw} which suggests that the resulting hypoxemia was secondary to alveolar-capillary injury, not to edema in small airways. Improved gas exchange and collateral flow without a decreased R_{saw} after PEEP suggests that the collateral channels involved are those of intra-alveolar pores of Kohn.

Conclusion. We conclude that pulmonary injury induced by oleic acid causes diffuse alveolar-capillary damage and closure of collateral channels. PEEP improves collateral ventilation, which may explain some of the previously reported benefits of PEEP for gas exchange of patients with acute respiratory failure.

References.

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