

Title : THE EVALUATION OF DOG LUNG CAPILLARY PERMEABILITY CHARACTERISTICS

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**Introduction.** The capillary pressure required to cause progressive edema (Pcc) should be decreased and the capillary filtration coefficient (Kf) increased by an increase in capillary membrane permeability in the lung. In order to test this we estimated Pcc and Kf in both normal dogs and in dogs with increased pulmonary capillary permeability caused by alloxan administration.

**Methods.** We evaluated five control dogs and six dogs given 75-100 mg/kg alloxan. We used a recently developed technique of weighing the left lower lobe of intact dog lungs. The arterial and venous pressures to the left lower lobe (Pa and Pv) were increased in steps and the change in lobar weight was recorded. Pulmonary capillary pressure (Pc) was calculated as  $(Pa + Pv)/2$ . Lobar weight became constant after each increase in pressures until Pc exceeded Pcc. When  $Pc > Pcc$  lobar weight increased at a constant rate (S). For each experiment we found the relationship between S and Pc and estimated Kf as  $\Delta S/\Delta Pc$ . Pcc was taken as the extrapolated Pc at which  $S=0$ .

**Results.** In the control experiments Pcc averaged  $22.5 \pm 5.1$  mmHg (mean  $\pm$  SD) and Kf was  $.036 \pm .013$  ml/min/mmHg/lobe. In the alloxan group Pcc was  $12.4 \pm 3.3$  mmHg and Kf was  $1.43 \pm 1.69$ . Kf was higher and Pcc significantly lower in the alloxan group. We also found a relationship between Pcc, the critical capillary pressure required for progressive edema to develop, and the plasma oncotic pressure ( $\pi_c$ ) in the control experiments ( $r = .70$ ). There was no relationship between Pcc and  $\pi_c$  in the alloxan group ( $r = .04$ ).

**Discussion.** These results show that changes in Pcc and Kf are indicators of changes in capillary membrane permeability. The significant relationship between the Pcc and  $\pi_c$  in the control lungs and the absence of this relationship after alloxan has direct clinical implications. The importance of maintaining  $\pi_c$  when Pc is elevated in normal lungs and the futility of such efforts when the exchange vessel permeability has been significantly altered is clearly demonstrated.