ROLE OF LYMPHATICS IN PREVENTING HYPOONCOTIC PULMONARY EDEMA

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The regulation of lung water depends on a precise balance between hydrostatic and osmotic forces across the pulmonary capillary. Marked

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Reprint from the Surgical Forum, Vol. XXVII, 1976
reduction of plasma colloid osmotic pressure (COP) usually does not result in pulmonary edema, even though peripheral edema may be prominent (1). We studied the role of lymphatics in protecting against the development of acute pulmonary edema when pulmonary capillary wedge pressure (PCWP) is normal and plasma COP is low.

METHOD

Five baboons weighing 21 to 28 kg were sedated (phencyclidine HCl), paralyzed (pancuronium bromide), and ventilated with a volume respirator. Left-sided lymph was collected by direct cannulation of the thoracic duct. The right cervical lymphatic chain was divided, and the remainder of right-sided lymph which included most of pulmonary lymph was collected through a right jugular-subclavian venous pouch. Plasmapheresis was performed three times by removing 20% of estimated blood volume and reinflushing the centrifuged red blood cells. Ringer's lactate was infused at a rate to hold PCWP constant. Cardiac output, central venous pressure, hematocrit, and urine output remained stable throughout the experiment. Colloid osmotic pressure was measured by a transducer membrane system (2). Albumin, protein, arterial and mixed venous blood gases, and dynamic compliance were measured and intrapulmonary shunt calculated.

RESULTS

Plasmapheresis reduced plasma COP from 19.6 ± 0.6 to 5.6 ± 1.8 mm Hg while PCWP remained stable at 4 ± 1 mm Hg. The COP-PCWP gradient decreased from 15.3 ± 1.9 to 12 ± 1.8 mm Hg. Right lymph COP, which may be taken as a reflection of pulmonary interstitial COP, decreased from 16.3 ± 2.7 to 5.9 ± 0.7 mm Hg. Thus, the net change in forces defined by the Starling equation was a 4-mm-Hg increase in filtration force across the pulmonary capillary.

Lymph flow increased fourfold: right lymph increased from 0.06 ± 0.03 to 0.28 ± 0.11 ml/min and left lymph increased from 0.27 ± 0.04 to 1.18 ± 0.22 ml/min. While left lymph albumin concentration decreased markedly (2.4 ± 0.1 to 0.1 ± 0.1 gm/100 ml), right lymph albumin concentration decreased (2.8 ± 0.2 to 1.0 ± 0.2 gm/100 ml) at a rate similar to the fall in plasma albumin (3.2 ± 0.1 to 1.1 ± 0.2 gm/100 ml). Right lymph:plasma albumin ratio was unchanged. Left lymph albumin flow (lymph flow × albumin concentration) decreased from 6.5 to 1.2 mg/min, while right lymph albumin flow increased from 1.7 to 2.8 mg/min.

Arterial blood gases, intrapulmonary shunt, and dynamic compliance did not change despite a 14% increase in body weight and the development of ascites and peripheral edema. At autopsy, pleural effusions were noted but no pulmonary edema was evident. Lung weight:
body weight ratio was 0.010 ± 0.001 and wet: dry lung weight ratio was 5.13 ± 0.25.

**DISCUSSION**

In these experiments, PCWP pressure was held constant by adjusting the infusion rate of Ringer's lactate so that the pulmonary microvascular pressure was unchanged. A fall in plasma COP would result in a corresponding increase in capillary filtration pressure if all other parameters of the Starling equation remained constant. However, right lymph COP, which probably reflects the lung interstitial COP, decreased along with the fall in plasma COP, thus neutralizing a large portion of this effect. In addition, excess filtrate was removed by an increase in volume of lymph flow. Fluid movement into the limited pulmonary interstitial space would cause an increase in interstitial pressure and also counter further filtration. Right lymph albumin remained the same as plasma, and no pulmonary edema developed while left lymph albumin fell towards zero and significant peripheral edema developed. The high right lymph albumin concentration could indicate that existing pulmonary extravascular protein was still being removed or that protein movement across the capillary was increased.

**CONCLUSION**

When plasma COP is lowered, massive peripheral edema develops, but the lungs are protected from edema by compensatory alterations in transcapillary forces and the rate of lymph removal.

**REFERENCES**
