DOES SEPSIS REDUCE THRESHOLD HYDROSTATIC PRESSURE FOR PULMONARY EDEMA?

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THE RELATIONSHIP of colloid oncotic pressure-pulmonary artery wedge pressure gradient (COP-PAW) in the development of pulmonary

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edema (PE) is not clear. Although an isolated increase in PAW beyond a threshold has been shown to cause PE, it is not clear whether an isolated reduction in COP causes PE. Moreover, sepsis might alter capillary permeability and contribute to the development of PE. The object of the present study was to determine whether sepsis reduced the threshold COP-PAW gradient necessary for pulmonary dysfunction.

MATERIALS AND METHODS

Seven adult male baboons underwent placement of a siliconized balloon catheter in the left atrial appendage by left anterolateral thoracotomy one week prior to study. Arterial and pulmonary artery catheters were inserted. Blood gases were taken and intrapulmonary shunts (Qs/Qt) calculated. COP-PAW gradients were randomly varied from 0 to -15 mm Hg in 5-mm increments. After the intended COP-PAW gradient was reached, the baboons were allowed to equilibrate for 30 minutes and measurements were taken. Pressures were then allowed to return to normal for 30 minutes before the next gradient was established. After the entire COP-PAW sequence was completed, infusion of *Escherichia coli* was begun. Two hours later the randomized sequence of COP-PAW gradients and all measurements were repeated.

A paired-comparison t test was used to determine significant differences between the septic and nonseptic periods at each COP-PAW gradient. The overall effect of sepsis on Qs/Qt was analyzed with a linear model.

RESULTS

The mean (± SEM) Qs/Qt at each COP-PAW gradient is shown in Figure 1. At each gradient point the average Qs/Qt of the septic period is higher than the nonseptic period and is consistently above 15%, a level generally

![Fig 1-Mean (± SEM) Qs/Qt at each COP-PAW gradient.](image-url)
agreed to represent clinically significant pulmonary dysfunction. In the nonseptic period, the intrapulmonary shunt exceeds 15% only when the COP-PAW gradient is lowered to -10 mm Hg. The linear model regression analysis indicated that the effect of sepsis significantly increases the Qs/Qt over and above the effect of COP-PAW gradient ($P < 0.005$).

DISCUSSION

The sum of forces across the normal pulmonary capillary membrane tends to keep the pulmonary interstitium dry. Increasing left atrial pressure above 22 mm Hg in the presence of normal COP has been shown to result in pulmonary edema (1). A decrease of the COP is a significant risk factor leading to pulmonary edema (2), but when pulmonary capillary hydrostatic pressures are normal, reduction of COP even to 0 mm Hg does not lead to pulmonary edema (3). Sepsis is said to cause pulmonary capillary dysfunction leading to loss of fluid into the interstitium. Our study confirmed that increasing left atrial pressure in the presence of normal COP results in pulmonary edema, and shows that sepsis lowers the threshold for development of edema. With the septic patient, meticulous attention must be paid to PAWP to avoid pulmonary edema.

REFERENCES