PULMONARY INTERSTITIAL PRESSURE The effects of alveolar and pleural pressures on the interstitial pressure surrounding the fluid-exchanging vessels of the lung were studied in dogs. Transudation of fluid into the lungs was produced by the combination of inflating a balloon in the left atrium and rapidly infusing isotonic saline solution. The rate of fluid accumulation was expressed as g water/g dry lung/hour. Increasing mean airway pressure did not reduce the rate of fluid accumulation, indicating its lack of effect on interstitial pressure. There was, in fact, a tendency for fluid to accumulate in the presence of elevated airway pressure. Two possible explanations for this are: 1) Increased alveolar capillary surface; 2) Decreased interstitial pressure. The latter explanation implies that increased alveolar pressure is not transmitted to the interstitial space surrounding the fluid-exchanging vessels, possibly because of interference by surface tension of the alveolar air-liquid interface. Elimination of this factor by ventilation with naphthalene resulted in decreased fluid accumulation. (Melvin, R. B., and others: Interstitial Pressure of the Lung, Circ. Res. 24: 197 (Feb.) 1969.) ABSTRACTER'S COMMENT: The apparent salutary effect of positive airway pressure in the treatment of clinical pulmonary edema are in no way negated by this report, since left atrial pressure was kept high and pulmonary capillaries were kept open. This is in contrast to what probably occurs clinically with IPPB, with its effects on decreasing central blood volume.

VENA CAVAL LIGATION Thirteen dogs were anesthetized with pentobarbital and their lungs artificially ventilated. Inferior vena cava (IVC), femoral arterial (FA) and right ventricular (RV) baseline pressures were measured through indwelling catheters. Heart rate, cardiac output, blood gases and pH also were measured. IVC was exposed and ligated below the renal veins. Measurements were repeated every five to ten minutes for at least an hour. Ligation of IVC resulted in a 10 per cent reduction in mean systemic pressure, 30 per cent reduction in cardiac output, a reduction in RV pressure (from a mean of 6.4 to 5.5 mm Hg) and a reduction in stroke volume (from 12.6 to 9.6 ml) within five minutes. These parameters progressively returned to control levels over the next 20 to 60 minutes. Heart rate and blood gases remained unchanged. IVC pressures increased abruptly from 2.6 to 28.2 mm Hg at five minutes, decreased to 15.5 during the next hour and remained elevated for as long as seven days (duration of application of ligature in one dog). It was suggested that such deleterious acute hemodynamic changes in patients with cardiopulmonary disease should be avoided by using volume expanders or myocardial stimulants. (Harsanyi, P. G., Ruiz-Garriga, J., and Moser, K. M.: Acute Hemodynamic Consequences of Ligation of the Inferior Vena Cava, J. Thorac. Cardiov. Surg. 27: 442 (March) 1969.)

SHOCK Temperature was recorded from the digital pad of the third finger, the large toe, deltoid region, lateral thigh and rectum in 100 patients with clinical signs of shock. A significant correlation between cardiac output and great toe temperature was seen. In addition to aiding in assessing blood flow, toe temperature provided a helpful indication of prognosis. If toe temperature three hours after admission was lower than 27 C, or if the difference between toe and ambient temperature was less than 2 C, the likelihood of death was high. Because of its simplicity, reliability and low cost, toe temperature measurement is of value in the clinical monitoring of shock. (Joly, H. R., and Weil, M. H.: Temperature of the Great Toe as an Indication of the Severity of Shock, Circulation 39: 131 (Jan.) 1969.)

HYPOXICO HEMOLYSIS Tocopherol-deficient mice were exposed to 100 per cent oxygen at varying pressures. It was shown that hemolysis could occur at pressures below 60 psia provided the duration of exposure was increased. The time required for hemolysis to occur increased linearly as a log-log function of the decrease in pressure. The data imply that any degree of hyperoxia can cause hemolysis if it is sustained. (Goldstein, J. R., and Mengel, C. E.: Hemolysis in Mice Exposed to Varying Levels of Hyperoxia, Aerospace Med. 40: 12 (Jan.) 1969.)