return of excess interstitial fluid as to the cardiac lesion itself, which initially increased the production of lymph. Increased return of lymph to the left heart in patients with isolated right heart lesions might relieve consequences of venous hypertension and perhaps increase left heart output. (*Cole, W. R., and others: Thoracic Duct-to-Pulmonary Vein Shunt in the Treatment of Experimental Right Heart Failure, Circulation 36: 539 (Oct.) 1967.*)

**PULMONARY CIRCULATION** At moderately high lung volumes, the distribution of pulmonary blood flow in both the human and isolated dog lung can be explained by the relations between pulmonary arterial, alveolar and venous pressures. Employing a radioactive xenon and lung scanning technique in normal man in the upright position, it was found that blood flow increased over almost the whole vertical distance down the lung at total lung capacity. However, there was often a small area of higher vascular resistance in the most dependent zone. There was invariably a larger region of reduced blood flow near the base at functional residual capacity. At F.R.C., blood flow per alveolus usually decreased over the lower third of the lung, reaching two-thirds of its maximum value at the base. At residual volume, blood flow per alveolus commonly decreased from top to bottom of the lung. Similar patterns were found in the isolated dog lung where the effects of changing lung volume and of vasoconstrictor and vasodilator drugs showed that the increased vascular resistance was caused by pulmonary vessels outside the influence of alveolar pressure. These results suggest that any general increase in interstitial pressure in the human lung will affect the distribution of blood flow and pulmonary vascular resistance. (*Glazier, J. B., and others: Role of Interstitial Pressure in the Distribution of Pulmonary Blood Flow, J. Physiol. 190: 23 (May) 1967.*)

**PULMONARY EDEMA** The average rate of water accumulation in healthy dog lungs varied in a nonlinear way with the level of capillary hydrostatic-plasma colloid osmotic pressure difference, and was unaffected by the level of capillary hydrostatic pressure. At low levels of left artrial pressure minus plasma colloid osmotic pressure, water accumulated in the lung at an average rate of 0.09 ml./gm. of dry lung per hour per mm. Hg pressure difference. At higher levels the average rate of accumulation was 0.22 ml./gm. per hour per mm. Hg pressure difference. In most experiments water accumulated in the lungs slowly during the first 30 minutes of the test period, and more rapidly as the period was extended. Obstruction of right lymphatic duct outflow did not alter the rate of water accumulation. In the present experiments, pericapillary pressures were estimated to be of the order of −9 mm. Hg and the filtration coefficient for the pulmonary capillaries is estimated to be of the order of one-tenth to one-twentieth that for canine muscle capillaries. These data indicate that edema formation in lung tissue cannot be defined solely in terms of intravascular forces, but may be governed to a significant degree by changes in pericapillary forces in the pulmonary interstitium. (*Levine, O. R., and others: The Application of Starling's Law of Capillary Exchange to the Lungs, J. Clin. Invest. 46: 934 (June) 1967.*)

**PULMONARY EDEMA** Using a double isotope dilution technique, pulmonary extravascular fluid volume (PEV) was measured in normal subjects and in patients with valvular heart disease. PEV, in normals, mitral valve disease, and aortic valve disease was 107, 193, and 154 ml./m.$^2$, respectively. PEV correlates closely with mean pulmonary arterial pressure, mean left atrial pressure and functional classification, whereas there is little relation between PEV and cardiac output or pulmonary vascular resistance. (*McCredie, Michael: Measurement of Pulmonary Edema in Valvular Heart Disease, Circulation 36: 381 (Sept.) 1967.*)

**AIR EMBOLISM** Pulmonary air embolism occurred during insertion of a transvenous pacemaker in an 88-year-old woman. In reviewing the literature, it is felt that immediate therapy consists of placing the patient in the left lateral decubitus position, administering oxygen and vasopressors. More vigorous therapy includes closed-chest cardiac massage, aspiration of air from the heart and hyperbaric oxygenation. The pathophysiology of venous air embolism is such that a large bolus of air