flow through the lungs, the shunts were twice as large in the IPPB group as the CPPB group. Increasing levels of ventilation decreased the amounts of shunt in both groups, but extremely high and impractical levels of ventilation were required in the IPPB animals compared with the CPPB animals. These studies demonstrate that the method of delivering ventilation is as important as the amount of ventilation in reducing physiologic shunting across the lung. (*Ashbaugh, D. G.: Effect of Ventilatory Methods and Patterns on Physiologic Shunt, Surgery 68: 99 (July) 1970.*)

PULMONARY EDEMA Retrospective studies of arterial blood-gas values in 101 patients with pulmonary edema due to myocardial infarction showed that a severe combined metabolic and respiratory acidosis was present in 55 (mean *p*H 7.11; P_{CO_2} 70 torr; base excess −10 mEq/l). Metabolic acidosis alone was found in 14 patients, respiratory acidosis in 12, and respiratory alkalosis in eight. The remaining 12 patients had normal blood-gas values. Analysis of arterial blood-gases is an invaluable guide to bicarbonate therapy in the management of patients with acute pulmonary edema. (*Avery, W. G., and others: The Acidosis of Pulmonary Edema, Amer. J. Med. 48: 320 (March) 1970.*)

PULMONARY CIRCULATION Increases in intravascular pressure caused linear increases in pulmonary blood volume in the isolated, perfused lungs of greyhounds when filling was either from artery to vein (antegrade) or from vein to artery (retrograde). At constant static intravascular pressure, pulmonary blood volume increased when transpulmonary pressure (and hence lung volume) was increased. The magnitude of the increase in pulmonary blood volume depended upon the level of intravascular pressure. When either the arterial or the venous cannulae were occluded, communication with the opposite side of the pulmonary circulation still occurred when alveolar pressure exceeded intravascular pressure. India ink injections demonstrated that the site of the arteriovenous communications may be vessels at alveolar septal junctions. These vessels are not considered to constitute shunts since their size and location suggest capability for gas exchange. (*Rosenzweit, D. Y., Hughes, J. M. B., and Glazier, J. B.: Effects of Transpulmonary and Vascular Pressure on Pulmonary Blood Volume in Isolated Lung, J. Appl. Physiol. 28: 553 (May) 1970.*)

PULMONARY PERFUSION Following myocardial infarction, perfusion of lung bases was markedly reduced (approximately 30 per cent) in 15 patients studied by radioactive xenon techniques. The patterns of blood flow and regional ventilation–blood flow ratios resembled those found in mitral stenosis. The patients sustaining myocardial infarction showed no clinical evidence of congestive heart failure. (*Kazemi, H., and others: Distribution of Pulmonary Blood Flow after Myocardial Ischemia and Infarction, Circulation 41: 1025 (June) 1970.*)

SHOCK Adrenal blood flow and steroid output in hypophysectomized dogs subjected to hemorrhagic shock were measured. Normotensive oligemia did not result in progressive decreases in adrenal venous flow and steroid secretion. Sensitivity of the adrenal cortex to adrenocorticotropic hormone was not altered. Replacement of relatively small amounts of isotonic saline solution either systemically or into the adrenal artery resulted in increases in adrenal flow and steroid secretion despite persistence of systemic hypotension. Adrenal perfusion seems to be an important factor in adrenal cortical secretion in hemorrhagic shock. (*Mack, E., and Egdahl, R. H.: Adrenal Blood Flow and Corticosteroid Secretion in Hemorrhagic Shock, Surg. Gynec. Obstet. 131: 65 (July) 1970.*)

CARDIOGENIC SHOCK Nine patients with cardiogenic shock or low cardiac output following open-heart surgery were treated with isoproterenol and propranolol. Isoproterenol alone was effective in increasing cardiac output in either condition. In four of nine patients, isoproterenol plus propranolol caused increases in stroke volume and cardiac output and decreases in heart rate. Within 30 to 75 minutes, cardiac output and stroke volume returned to near baseline values. In these patients, the antagonistic effects of propranolol on heart rate were seen earlier than the effects