

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 pH 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. C., 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 sug-

gest capability for gas exchange. (Rosenzweig, 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 adrenocorticotrophic 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

on force of contraction. Since the separation of inotropic and chronotropic effects was transient and unpredictable, the combined therapy offered no advantage in the treatment of cardiogenic shock. (Stubbs, D., Pugh, D., and Bell, H.: *Combined Use of Isoproterenol and Propranolol in Cardiogenic Shock*, *Clin. Pharmacol. Ther.* 11: 244 (March) 1970.)

PLASMAPHERESIS No consistent abnormalities in clinical or laboratory findings were observed during six years of experience with plasmapheresis, in which more than 14,000 units of plasma were collected. Some donors gave as much as the plasma equivalent of four units of whole blood per week. Repeated plasmapheresis of a small pool of preselected donors provides a better product than plasmapheresis of random donors. Potential hazards for the plasmapheresis donor include hemoglobin, protein, and iron depletion, and misidentification of blood returned to the donor. Apparently the liver is capable of responding to plasmapheresis by increasing the rates of synthesis of albumin and other fatty plasma proteins. Recovery of normal gamma-globulin values is slower because it is dependent upon increased catabolism. Levels of total serum proteins and protein fractions should be monitored frequently in regular plasmapheresis donors. (Cohen, M. D., and Oberman, H. A.: *Safety and Long-term Effects of Plasmapheresis*, *Transfusion* 10: 58 (March) 1970.)

Respiration

INERTANCE Measurements of the inertance (inertia) of the respiratory system were made in five anesthetized, paralyzed subjects. Inertance values ranged from 0.018 to 0.035 cm H₂O·K·sec², after correction for the difference in density between the anesthetic gas mixture and air. (Bergman, N. A.: *Measurement of Respiratory Inertance in Anesthetized Subjects*, *Resp. Physiol.* 9: 65 (April) 1970.)

LUNG MECHANICS One pulmonary artery was occluded by inflation of an intraluminal balloon in each of a group of anesthetized, vagotomized dogs. Compliance decreased and resistance increased in both ipsilateral and contralateral lungs. End-expiratory P_{CO₂} decreased markedly in the oc-

cluded lung and increased in the contralateral perfused lung. Both inhalation of oxygen and intravenous administration of isoproterenol reversed the changes in compliance in both lungs. Inhalation of 2 to 8 per cent CO₂ increased compliance in the occluded lung but not in the perfused lung. Following multiple embolizations of one lung with iodinated oil, qualitatively similar alterations in pulmonary mechanics occurred; these were reversed by oxygen inhalation and by isoproterenol, but not by inhalation of CO₂. Observed effects of oxygen and isoproterenol on pulmonary mechanics following pulmonary artery occlusion suggested that the mechanical changes were due to smooth-muscle contraction secondary to ischemia. Compliance and resistance changes in contralateral perfused lungs may have been due to a circulating bronchoconstrictor substance elaborated in the occluded lung. (Tist, G. M., and others: *Effects of O₂ and CO₂ on Airway Muscle Following Pulmonary Vascular Occlusion*, *J. Appl. Physiol.* 28: 570 (May) 1970.)

ATELECTASIS Pulmonary hemodynamics associated with atelectasis were studied in dogs after recovery from surgical implantation of various flow- and pressure-sensing devices. Thirty minutes of acute unilateral atelectasis produced no remarkable change in cardiac output. A small but significant decrease in flow to the collapsed lung (4 per cent) occurred, and flow to the ventilated lung (1 per cent). Transpulmonary pressure was zero on the collapsed side and increased on the ventilated side. Pulmonary vascular resistance increased 15 per cent in the atelectatic lung but also increased 8 per cent in the expanded lung. The increase in vascular resistance in the expanded lung aids in maintenance of circulation in the collapsed lung and exaggerates hypoxia due to atelectasis. (Morgan, B. C., and Guntheroth, W. G.: *Pulmonary Blood Flow and Resistance during Acute Atelectasis in Intact Dogs*, *J. Appl. Physiol.* 28: 609 (May) 1970.)

OXYGEN UPTAKE Passive hyperventilation of anesthetized, paralyzed dogs increased oxygen uptake significantly and reproducibly. The increased uptake was not a result of an