

DIALYSIS FOR HEART FAILURE
Peritoneal dialysis significantly alleviated heart failure in 16 patients who did not respond to the conventional regimen of bed rest, salt restriction, digitalis and various diuretics including thiazides, mercurials and ethacrynic acid. Hyponatremia, hypochloremia, hypovolemia and markedly reduced cardiac indices accompanied the clinical picture of intractable failure. Since water equilibration between plasma and dialysate occurs before solute equilibrium, it was possible to remove relatively more water than sodium or chloride by limiting equilibration times to 30 minutes or less. Sustained clinical improvement in 12 of the 16 patients was associated with significantly increased cardiac indices, decreased blood volumes, weight loss and rises in serum plasma and chloride levels. (Cairnes, K. B., and others: *Clinical and Hemodynamic Results of Peritoneal Dialysis for Severe Cardiac Failure, Amer. Heart J.* 76: 227 (Aug.) 1968.)

PULMONARY CIRCULATION Respiratory acidosis, metabolic acidosis, infusion of serotonin and infusion of potassium are known to cause elevation of pulmonary artery pressure without remarkable alteration of cardiac output. During each of these conditions in anesthetized, artificially ventilated dogs, arterial oxygen tension was higher and alveolar-arterial oxygen tension difference and venous admixture were lower than under control conditions. These findings suggest that elevation of pulmonary artery pressure diverts blood from overperfused alveoli to less-well-perfused alveoli, resulting in a more homogeneous distribution of pulmonary capillary blood relative to inspired gas. Gravitational and positional factors may also change pulmonary artery pressure and influence the distribution of pulmonary capillary blood. (Haas, F., and Bergofsky, E. H.: *Effect of Pulmonary Vasoconstriction on Balance Between Alveolar Ventilation and Perfusion, J. Appl. Physiol.* 24: 491 (April) 1968.) **ABSTRACTER'S COMMENT:** Changes in pulmonary artery pressure during anesthesia may well be a highly significant factor contributing to the abnormally large alveolar-arterial oxygen tension differences fre-

quently observed in anesthetized subjects. Little is known about alterations in pulmonary hemodynamics during anesthesia, and there would seem a most fertile field for further investigation.

Respiration

PNEUMONECTOMY Arterial Pa_{O_2} and Pa_{CO_2} were measured in ten patients undergoing pneumonectomy in the lateral position. They were anesthetized with nembutal, morphine and nitrous oxide-oxygen and ventilated by an Engström ventilator. Initially, the oxygen concentration in inhaled gas was 34 per cent and the tidal volume was 133 per cent of the required value according to the Radford nomogram. After thoracotomy, four patients required increases in ventilation to 150 per cent of values predicted by the Radford nomogram to have Pa_{CO_2} values of 40 mm Hg or lower. Pa_{O_2} values were all above 100 mm Hg, the mean being 114.8 mm Hg. Following pneumonectomy, oxygen tension fell to an average of 94.5 mm Hg, and in three patients the tension did not rise above 85 mm Hg until the inhaled oxygen concentration was raised to 50 per cent. Compensation for this fall in oxygen tension was again seen at the ends of the operations when the patients were turned back to the supine position. After pneumonectomy, Pa_{CO_2} rose from 35.4 to 39.1 mm Hg, and this mean value was not changed when the patients were returned to the supine position. These changes were explained by an increase in capillary shunt, enhanced by the lateral position; decrease in ventilation/perfusion ratio; and increase in the deadspace/tidal volume ratio. A content of at least 50 per cent oxygen in the inhaled gas mixture and a ventilation of at least 150 percent of the Radford values are required during pneumonectomies. Because of the great variations in blood gases, even under these conditions, measurement of Pa_{O_2} and Pa_{CO_2} is recommended to ensure sufficient ventilation and oxygenation during pneumonectomy. (Bennicke, Knud A.: *Changes in Arterial Oxygen and Carbon Dioxide Pressure after Pneumonectomies, Der Anaesthetist* 17: 34 (Feb.) 1968.)

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