ure was the intravenous administration of corticosteroids in large doses for several days. Bronchoscopy was considered contraindicated. Epidural anesthesia was recommended to avoid this obstetrical complication. (*Berris, B., and Kasler, D.: Pulmonary Aspiration of Gastric Acid-Mendelson's Syndrome, Canad. Med. Ass. J. 92: 905 (Apr. 24) 1965.*)

GAS EXCHANGE WITH HYPOTENSION Dogs under general anesthesia and muscle relaxants were ventilated at a fixed rate and volume. From 24 to 32 ml./kg. blood was withdrawn to produce mean blood pressure of 79 mm. of mercury. Oxygen consumption, carbon dioxide elimination, arterial P_{O_2} and P_{CO_2}, and arterial *p*H were measured before and after hemorrhage and again after intra-arterial reinfusion of the withdrawn blood. Cardiac output decreased by more than 50 per cent, arterial oxygen saturation fell from 94 to 88 per cent, arterial P_{CO_2} rose from 41 to 46 mm. of mercury and physiologic dead space increased by 27 per cent. The gradient of arterial to alveolar P_{CO_2} increased from 2.1 mm. to 4.8 mm. of mercury which indicates an increase in wasted ventilation. A metabolic acidosis occurred simultaneously, the arterial *p*H falling from 7.29 to 7.00 and buffer base from 42 to 38 mEq. During the control period, the amount of available oxygen was about 4 times the oxygen consumption. After hemorrhage this relation fell to 2:1 as the result of low cardiac output. When, during hemorrhagic hypotension, hyperventilation with greatly diminished arterial oxygen saturation was superimposed, the amount of available oxygen may critically approach the rate of oxygen consumption. After arterial retransfusion of the withdrawn blood most values returned to control levels. (*Rehder, K., Teichert, P., and Hessler, O.: Effect of Hemorrhagic Hypotension on the Pulmonary Gas Exchange during Controlled Respiration, Thoraxchirurgie 13: 289 (August) 1965.*)

PULMONARY VASCULAR RESISTANCE Blood flow was determined by means of a scanning technique following injection of radioactive xenon in saline into the pulmonary artery of the isolated perfused canine lung. With elevation of pulmonary venous pressure, blood flow in dependent portions of the lung decreased while vascular resistance increased. This effect was rapidly reversible by lowering the venous pressure. Histologic study of quick frozen sections showed interstitial edema, especially around small arteries and veins. Perivascular edema may counteract the normal "tethering" action of lung parenchyma which acts to keep the vessels open and thus allows the natural tendency of vessels to collapse to prevail. Such a mechanism may explain the increased vascular resistance found in patients with pulmonary venous hypertension as in mitral stenosis or left ventricular failure. (*West, J. B., Dollery, C. T., and Heard, B. E.: Increased Pulmonary Vascular Resistance in the Dependent Zone of the Isolated Dog Lung Caused by Perivascular Edema, Cir. Res. 17: 191 (Sept.) 1965.*)

PULMONARY HYPERTENSION On 50 patients, age 29 to 70, there were 20 lobectomies (group I) and 30 pneumonectomies (group II). Preoperatively the right ventricular pressure averaged 24/0 in group I and 29/1 in group II. Postoperatively these values rose to 34/0 in group I and 40/2 in group II. Pulmonary arterial pressures were 24/7 and 27/10 preoperatively, 33/12 and 39/15 postoperatively in the two respective groups. The hematocrit was 38.2 and 38.9 versus 47.3 and 49.3, respectively, before and after operation. In some of the pneumonectomies, the postoperative pulmonary blood pressure was more than 100 per cent higher than before operation. Particularly in group II a latent right heart failure should always be suspected. Pulmonary hypertension is not always caused by an increase in vascular resistance. Rise of the hematocrit as the result of chronic hypoxemia and the relative polycythemia entail a rise in viscosity which, in turn, effects further resistance to perfusion and rise of pulmonary blood pressure, eventually resulting in cor pulmonale. (*Schickedanz, H., and others: Pathophysiology of Lung Resection, Langenbeck Arch. Klin. Chir. 311: 12 (July 15) 1965.*)

PULMONARY CIRCULATION Acute pulmonary vascular congestion produced by body tilting pressure suit inflation, or occlusive thigh tourniquets increases breath-holding dif-