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 blood was withdrawn to produce mean blood pressure of 79 mm.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P_{CO_2}, and arterial *p*H were measured before and after hemorrhage and again after intra-arterial reinfusion of the withdrawn blood.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 After arterial retransfusion of the withdrawn blood most values returned to control levels.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-

fusing capacity for carbon monoxide by increasing the instantaneous volume of pulmonary capillary blood available for carbon monoxide absorption. Following initial small increases in pulmonary vascular pressure, no further decreases in diffusing capacity were observed with further pressure increases, indicating an upper limit to the passive enlargement of the normal pulmonary capillary bed. The curve relating diffusing capacity to pressure suggests that passive enlargement is the consequence of either recruitment of a limited number of capillaries or limited dilation of capillaries as pressure is increased. Muscular exercise produces greater increases in diffusion capacity for carbon monoxide than can be produced by the maximal effect of passive congestion. This suggests that during muscular exercise diffusing capacity is increased by factors other than pressure alone. (*Daly, W. J., Giammona, S. T., and Ross, J. C.: Pressure-Volume Relationship of the Normal Pulmonary Capillary Bed, J. Clin. Invest. 44: 1261 (July) 1965.*)

SHOCK Ventilatory mechanics and pulmonary diffusion were studied in anesthetized dogs subjected to hemorrhagic or endotoxin shock. Acute hemorrhagic hypovolemia is associated with a transient but definite (50 per cent) increase in compliance and decrease in resistance to airflow (20 per cent); endotoxin shock showed opposite effects. All changes return to near control levels in a short time and remain so even if irreversible shock follows. At the same time, pulmonary diffusion is reduced during shock. This is related to both reduced pulmonary capillary blood volume and pulmonary capillary red-blood-cell volume. The fact that diffusion does not return to normal in irreversible shock, even after blood volume, systemic blood pressure and pulmonary artery pressure have returned to near normal, indicates increased pulmonary vascular resistance. The cause of this could be vasospasm, high critical reopening pressures, pooling or microcoagulation in the pulmonary circuit. (*Cahill, J. M., Jouasset-Strieder, D., and Byrne, J. J.: Lung Function in Shock, Amer. J. Surg. 110: 324 (Sept.) 1965.*)

SHOCK Cardiogenic shock was produced in dogs by microsphere embolization of the coronary arteries. Shock was treaetd by administration of low molecular weight dextran, 2 g. per kg. Compared to control animals, those treated had increased cardiac output, reduced peripheral resistance and improved blood flow through the viscera. The blood volume increased, resulting in increase of stroke volume, heart rate and venous pressure. There was no pulmonary edema. Of 23 controls, 15 developed shock and of these, 3 survived 48 hours. Of 20 treated animals, 9 developed shock and of these 7 survived 48 hours. (*Bloch, J. H., and others: Experimental Cardiogenic Shock, Arch. Surg. 91: 77 (July) 1965.*)

SHOCK In anesthetized artificially ventilated dogs subjected to acute hemorrhagic shock by bleeding 30 per cent of the blood volume and maintaining this deficit for two hours, application of 25 mm. of mercury external counterpressure (ECP) by means of a tank surrounding the animal improved survival. Three of 20 (15 per cent) control dogs survived beyond 24 hours; 8 of 14 (57 per cent) ECP dogs survived. Cardiac outputs remained higher in ECP animals than in controls during the period of shock. Apparently beneficial effect of ECP in hemorrhagic hypotension is due to compression of the venous bed with resultant increase in effective circulating blood volume. (*Shane, R. A., and Campbell, G. S.: Protective Influence of External Counterpressure in Acute Hemorrhagic Hypotension in Dogs, Amer. J. Surg. 110: 335 (Sept.) 1965.*)

SHOCK The role of the mesenteric sympathetic nervous system in hemorrhagic shock was studied. Two groups of dogs were subjected to a postganglionic mesenteric sympathectomy and exposed to hemorrhagic shock two and six weeks after denervation. In another group of dogs, the same structures were infiltrated with 1 per cent novocaine 30 to 45 minutes after the onset of hemorrhagic shock. The survival rates and hemodynamic parameters were compared with a control group and with another series that were pretreated with dibenzylene. The denervation operation provided protection similar to that of dibenzylene except to a much lesser degree. Chemical blockage of the mesenteric sympathetics after the onset of shock afforded no