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Respiration

ABSENT HYPOXIC PULMONARY VASOCONSTRICTION AND HEPATIC CIRRHOSIS Evidence that patients with hepatic cirrhosis have increased pulmonary blood flow, arterial oxygen desaturation, decreased pulmonary vascular resistance, and hyperventilation has been presented. Although many explanations for arterial hypoxemia are available, none has been accepted. The shift to the right in the oxyhemoglobin-dissociation curve is not sufficient to explain the degree of desaturation. Common findings include a normal diffusion capacity, an increased alveolar–arterial oxygen tension gradient, and inability to saturate arterial blood fully during breathing of oxygen. These findings, and the results of various isotope gas studies, all favor venous admixture as the fundamental cause of the arterial hypoxemia. However, necropsy studies of patients with hepatic cirrhosis have revealed intrapulmonary arteriovenous anastomoses or portopulmonary venous communications in only a few patients. It has recently been theorized that, in the absence of a ventilatory defect, the arterial hypoxemia might be due to inappropriate distribution of pulmonary flow relative to ventilation. If the pulmonary system were not able to regulate perfusion, then the combination of arterial hypoxemia and low pulmonary vascular resistance might occur. The authors postulate that impairment of pulmonary hypoxic vasoconstriction might be responsible for both the low pulmonary vascular resistance and the resulting arterial hypoxemia in these patients. Therefore, the effects of low fractional inhalation concentrations of oxygen were studied in patients with cirrhosis, normal subjects, and finally, patients with clinical features similar to cirrhotics, i.e., increased pulmonary flow, anemia, and chronic debilitating illness. Ten patients with alcoholic hepatic cirrhosis breathed 10 per cent oxygen in nitrogen, but failed to demonstrate an increase in pulmonary vascular resistance. However, four patients with functional murmurs, three patients with hyperkinetic heart syndrome, six patients with normal pulmonary arterial pressures and intracardiac left-to-right shunts, and five patients with renal failure and anemia all increased their pulmonary vascular resistances when they breathed 10 per cent oxygen in nitrogen. These findings suggest that in hepatic cirrhosis the normal regulating mechanism of the pulmonary vascular bed, hypoxic vasoconstriction, may be impaired, resulting in failure of the lung to match perfusion to ventilation. (*Daoud, F. S., Reeves, J. T., and Schaefer, J. W.: Failure of Hypoxic Pulmonary Vasoconstriction in Patients with Liver Cirrhosis, J. Clin. Invest. 51: 1076–1080, 1972.*)