
The Lung and the Liver

Hepatic cirrhosis is associated with a wide variety of circulatory abnormalities. In the kidney, in severely decompensated cirrhosis, there may be renal cortical hypoperfusion leading to renal insufficiency of the hepatorenal syndrome. Cirrhotic patients commonly have increased cardiac output, decreased peripheral resistance, and expanded blood volume associated with hypoxemia and hypcapnia. Still other investigators have demonstrated increased pulmonary blood flow with decreased pulmonary artery pressure and resistance in patients with chronic liver disease. Paradoxically, an increasing number of cirrhotic patients have also been discovered to have idiopathic pulmonary hypertension. Tense ascites, hydrothorax, anemia, and fever are other obvious factors that may alter the circulation in cirrhotic patients.

Daoud and associates demonstrated the consistent loss of the hypoxic pulmonary vasoconstricting mechanism in ten severely decompensated cirrhotic subjects selected because they had clinical evidence of hyperdynamic systemic circulation. They postulated that impaired hypoxic vasoconstriction could lead to a relatively dilated pulmonary microvascular bed and a mismatch of ventilation and perfusion with resultant hypoxemia.

In this issue (see page 510) Naeije et al have reported valuable additional hemodynamic observations in cirrhotic patients. They showed that the loss of hypoxic pulmonary vasoconstriction ("nonresponders") in cirrhotic subjects is by no means uniform occurring in only seven of their 24 patients breathing 12.5 percent inspired oxygen for ten minutes. The other 17 "responders" increased pulmonary vascular resistance by greater than 20 percent over baseline. In both "responders" and "nonresponders,"
however, they confirmed a generally low mean pulmonary artery pressure of 11.5 mm Hg (normal 15 ± 3 [SD] mm Hg) in the face of arterial hypoxemia and a tendency for high cardiac output. Thus, they have extended the observations of abnormal pulmonary circulation and gas exchange even in cirrhotic patients with preservation of hypoxic pulmonary vasoconstriction. They did not postulate an explanation for these findings.

The difference in prevalence of the pulmonary hypoxic response in the studies of Daoud and Naeije may be related mostly to patient selection. Naeije et al did not select patients for their hyperdynamic circulatory state, and their patients appear to have less severe hepatocellular dysfunction compared to those studied by Daoud. Although complete data are not presented, seven out of ten patients in Daoud's study were jaundiced while the average serum bilirubin level in Naeije's patients was less than 2 mg/100 ml, the level below which clinical icterus usually cannot be detected. Unfortunately, neither study provides data for comparison on the degree of portal hypertension and extent of collateral circulation.

In alcoholic cirrhosis, the presence or absence of superimposed alcoholic hepatitis causes great variation in the severity of hepatocellular dysfunction, while the degree of portal hypertension may remain relatively fixed. Theoretically, injured parenchymal liver cells could elaborate or fail to detoxify a circulating vasoactive substance capable of altering pulmonary vascular control mechanisms. If, then, the alcoholic patients studied by Daoud and Naeije differed primarily in the severity of hepatocellular dysfunction, this could point the way for future investigations. For instance, the pulmonary hemodynamics of patients with portal vein thrombosis could be compared to patients with hepatocellular disease without significant portal hypertension. Likewise, it would be interesting to perform serial studies in alcoholic cirrhotic patients with stable portal hypertension but variable hepatocellular dysfunction from alcoholic hepatitis.

The relationship between liver disease and pulmonary hemodynamics is complex. To date, the findings are intriguing, perhaps conflicting, but surely too few for a solid pathophysiologic understanding. Clearly, further studies are needed.

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Computed Tomography of the Thorax
An Important New Diagnostic Tool

Computed tomography (CT) was introduced into the United States in 1973 at a time when politicians, bureaucrats, and the public were becoming increasingly concerned about the rising costs of health care. Utilizing roentgen rays and computer technology, the technique dramatically improved the ability to image the interior of the body. Because of the considerable expense of purchasing and operating the machine, CT became the focus of a continuing controversy over the value and cost of new medical technology.1 However, as Abrams and McNeil2 have recently pointed out, the implications of cost, distribution, and utilization of CT scanners can only be understood through continuing analysis of the impact of CT on medical diagnosis. At the level of patient care, the physician and the radiologist share the responsibility for confining the use of this expensive, but exciting new tool to instances where it offers a reasonable chance of diagnostic gain. Thus, the appearance of a report in this issue of Chest (see page 618) on the state of the art of thoracic CT is especially timely.

Although the number of papers dealing with CT scanning of the thorax has burgeoned, we are still in the early days of this rapidly developing technology. The understanding of a number of factors is important for success in its use. A scanning time of less than 18 sec permits breath-holding, greatly reduces artifact due to respiratory and cardiac motion, and improves the quality of images of thoracic structures.3 Because mediastinal structures are embedded in fat, the CT scan sharply etches the outline of normal structures so that they are visualized with a clarity unmatched by any other imaging technique. However, since CT scanning produces a series of transverse images, physicians who would use the technique must return to basics and become thoroughly familiar with the cross-sectional anatomy of the thorax.4 Concepts of the anatomy of the thorax that are more than sufficient for interpretation of frontal, lateral, and oblique chest roentgenograms and conventional chest tomograms are not sufficient background for interpretation of CT scans.

The CT image is generated by a computer analysis of the degree of attenuation of the roentgen beam as it passes through the body. Pulmonary physicians are familiar with the concept that a different contrast may be necessary to image the mediastinum than the lung parenchyma. A range of images of varying contrast can be generated by appropriately instructing the computer after a single exposure of the patient to the roentgen rays. Finally, the degree