When ascitic fluid accumulates in patients with chronic hepatic congestion, its protein content is often high, in distinct contrast to low protein levels (usually less than 1.5 g/100 ml) accompanying cirrhosis of the liver. The explanation for this difference is not fully understood but observations, both in patients and experimental animals, suggest that elevated hepatic sinusoidal pressure drives plasma across highly permeable sinusoidal membranes into the interstitial space of Disse and across the liver capsule. When the capacity of the hepatic lymphatic drainage system is exceeded, high protein lymph "weeps" into the peritoneal cavity as ascitic fluid of high protein concentration (1, 2). Dive and his colleagues (3) report that the transport of plasma proteins across dog hepatic sinusoids and into hepatic lymph has two major components: molecular sieving of larger proteins and bulk flow. Using their techniques, we studied the transfer of proteins from plasma to ascitic fluid in a patient with gross ascites of high protein concentration resulting from chronic constrictive pericarditis.

A 17-year-old girl developed secondary amenorrhea in March 1975. Following a negative pregnancy test she was treated unsuccessfully with medroxyprogesterone acetate. She returned 6 months later with gross ascites. Pelvic examination was unremarkable. Chest x-ray demonstrated small bilateral pleural effusions. The hemoglobin level was 8.6 g/100 ml; the serum bilirubin, LDH, SGOT, and albumin were within normal limits. Abdominal paracentesis failed to reveal malignant cells in the aspirated fluid. Following blood transfusion a laparotomy was performed which revealed the liver to be enlarged and nodular. A wedge biopsy was taken and the ascites was drained.

One year later she was readmitted with the additional complaint of easy fatiguability. Despite continued administration of a low-salt diet, spironolactone, and furosemide, the ascites had recurred and persisted. On examination there was an umbilical hernia and massive ascites without peripheral edema. The liver and spleen were not palpable and cardiovascular findings suggested pericardial constriction. The pulse was 80/min, the blood pressure 100/60 mm Hg lying and standing, and the systolic blood pressure fell 10 mm Hg on deep inspiration. Jugular venous pressure was markedly elevated with little discernible pulsation. A diastolic right ventricular impulse was palpable, and a third heart sound and fine basal pulmonary rales were audible.

Investigations at this time revealed linear calcification in the anterior pericardium on chest x-ray; there was no pleural effusion. EKG showed widespread T-wave inversion with right ventricular hypertrophy. Right and left heart catheterization revealed elevation and equalization of the diastolic pressure in all four chambers to 20-25 mm Hg.

At this time, hemoglobin, white blood cell count and differential, blood smear, and platelet count were unremarkable. Blood urea nitrogen, bilirubin, alkaline phosphatase, LDH, SGOT, and prothrombin time were also normal. Urinalysis demonstrated a trace of albumin on one examination. Total serum protein was 7.2 g/100 ml and serum albumin 4.4 g/100 ml. Radiologic studies of the small intestine and histologic section of jejunum after