The development of focal arteriolar and capillary damage is, then, the anatomical result of the excessively high pressure associated with malignant hypertension; the physiological result appears to be a breakdown in the relationship between pressure and flow; and both these manifestations are the direct result of a pressure that exceeds the yield point of resistance arterioles.


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# Diagnostic Ascitic Tap in Cirrhosis

The dangers of immediate shock[1] or hyponatraemia[2] or both have discouraged the use of paracentesis as a treatment for ascites in patients with cirrhosis. However, aspiration of 10-20 ml by syringe should be a routine, for this can be of great value in diagnosis, particularly in bacterial or tuberculous peritonitis, which may be clinically silent. Textbook descriptions refer to protein concentrations of less than 25 g/l in uncomplicated cirrhosis, values greater than this being suggestive of tuberculous infection or hepatoma development.[3] This view has now been challenged by Sampliner and Iber,[4] who found that 19 of 98 consecutive patients with cirrhosis and ascites admitted to their care had an ascitic protein concentration of more than 25 g/l, the highest value being 43 g. In none of these patients was there any evidence of the textbook complications.

One possible explanation for high-protein ascites in cirrhosis has been suggested by Witte *et al.*,[5] who drew attention to the higher protein concentration of hepatic lymph when compared to splanchnic lymph. If ascites is derived largely from hepatic interstitial fluid, as occurs with postsinusoidal portal hypertension, it will have a higher protein concentration than that occurring when the portal hypertension is predominantly presinusoidal, when the ascites is derived from the splanchnic interstitial fluid. Interestingly, in the series of Witte *et al.*, patients with "early" cirrhosis had a much higher ascitic fluid protein concentration than those with "late" cirrhosis. They therefore suggested that the postsinusoidal area was the earliest site of vascular obstruction and that as the cirrhosis progressed pressure was transmitted back through the portal venous system, resulting in an increasing contribution of splanchnic interstitial fluid to the ascites.

Though it need not indicate a serious complicating condition, nevertheless, when a high protein fluid is found it should always be examined by microscopy and culture for tubercle bacilli—a protein concentration of more than 25 g/l is almost invariable in tuberculous peritonitis.[6 7] Too often this diagnosis is not made until the patient reaches necropsy.

Pancreatic ascites is another condition associated with ascitic fluid of high protein concentration. Eighty-five cases have recently been reviewed by Donowitz *et al.*[8] This condition occurs predominantly in alcoholics, and not surprisingly a number of the patients have associated liver disease. The mechanism of the ascites is not clear, but it usually occurs in association with either a pseudocyst or a ruptured pancreatic duct. The ascites is massive and often resistant to diuretics. Abdominal pain and tenderness need not be present, and there is no close temporal association to bouts of acute pancreatitis. Macroscopically the fluid is usually serous but may be serosanguinous. The amylase concentration in the ascitic fluid, which may exceed 20 000 Somogyi units, is of diagnostic importance.

Spontaneous bacterial peritonitis is being increasingly recognized in cirrhosis with ascites.[9 10] The condition carries a high mortality (80-95%) and is not rare. Both Conn[9] from U.S.A. and Caroli[11] from Paris have reported frequencies of 8% in patients with cirrhosis and ascites admitted to hospital. Characteristically, the patient has fever, abdominal pain, reduced or absent bowel sounds, and impending hepatic coma.[9] Rebound tenderness is less common, and sometimes the condition can be entirely silent. The ascitic fluid is usually cloudy in appearance with a white cell count—predominantly polymorphonuclear—above 1 x $10^9$/l (1000/$mm^3$) and the responsible organism can often be identified from a Gram stain of the centrifuged deposit. In two thirds of cases this is a Gram-negative bacillus, the pneumococcus accounting for most of the remaining cases. The protein concentration of the fluid, in contrast to that in tuberculous peritonitis, is almost invariably low, a mean value of 18 g/l being recorded in Conn's series.[9] Why the peritonitis develops is unknown, but it has never been described in the absence of ascites.


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# Congenital Defects of the Anterior Abdominal Wall

In Britain and the U.S.A. the declining birth rate has probably reduced[1] the numbers of congenital abnormalities requiring urgent surgery by at least 20%. Furthermore the change in the climate of opinion about the need for urgent operative closure of severe myelomeningocele has reduced the number of long-stay cases in many neonatal surgical units. There has thus been an apparent increase in the incidence of some of the less common congenital lesions; defects of the anterior abdominal wall are a noteworthy example. Indeed, there may have been an actual increase[2 3] in such cases seen in neonatal surgical units, since the improved survival rates, particularly in ruptured exomphalos and gastroschisis, achieved over the past ten years may have encouraged referral of cases previously considered hopeless.

Two major congenital abnormalities occur in the region of the umbilicus: their combined incidence is around 1 per 5000 live births.[4] Exomphalos (called omphalocele in North America) is a herniation of intra-abdominal viscera through the umbilical ring; there may be an intact sac or it may rupture before, during, or after birth. Gastroschisis is a defect in the abdominal wall separate from the umbilicus, usually