Alterations in Splenic Hilar Lymphatics in Patients with Congestion of the Spleen (35112)

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Experimental ligation of the splenic vein, a model for studying effects of splenic congestion in animals, immediately increases splenic lymph formation (1). Whether or not a similar alteration develops in patients with splenic congestion secondary to cirrhosis or congestive heart failure is unknown. Splenic lymph vessels are diaphanous structures almost invisible during life and collapsed after death. For this reason studies of deranged splanchnic lymph formation in patients with cirrhosis or congestive heart failure have focused on liver and intestinal lymphatics and have neglected those of the spleen (2–4).

Histologic studies of clamped sections of the hepatoduodenal ligament by Baggenstoss and Cain demonstrated that abnormally large and numerous hepatic hilar lymph vessels characterize patients with cirrhosis or congestive heart failure (5–7). An attempt to similarly consider splenic lymph drainage forms the basis for this report. As abnormalities in size, number, and contents of splenic hilar lymph vessels would reflect alterations in splenic lymph formation, these vessels were examined microscopically in clamped sections of the splenic pedicle removed at autopsy or following splenectomy. Sections from patients who had splenic congestion secondary to cirrhosis or congestive heart failure were compared to sections from patients without these disorders.

Methods. The procedures used in this study are similar to those of Baggenstoss and Cain's investigation of hepatic hilar lymphatics (5–7). Specimens were collected at autopsy or at operation in the following manner: before removal of the spleen, the splenic pedicle was cross clamped at two sites, the first as close to the spleen as possible and the second about 2–3 in. away, to encompass a component of the pedicle. With the clamps still in place the specimen was resected and fixed in formalin. Representative sections embedded in paraffin were cut 5 μ in thickness and stained with hematoxylin and eosin and by Weigert's method for elastic fibers (8). All sections which included the splenic artery were examined with a 32$\times$ scanning lens and only lymph vessels that could be identified at this magnification were counted. The largest number identified in a single field was taken as the final count. When a section included lymph nodes, only vessels outside the capsule of the node were counted. At the time of microscopic examination, the examiner had no knowledge of the source of the specimen. In addition to the number of vessels, size and contents were also observed. Based on the technique of Baggenstoss and Cain, certain characteristics were used to distinguish lymph vessels from veins namely, extremely thin walls, lack of significant musculature, and absence of a continuous elastic membrane.

Clinical Data. In 5 patients without portal or systemic circulatory congestion specimens were obtained at autopsy following death from carcinomatosis (3), pulmonary embolism, and transection of the spinal cord. Five additional specimens were obtained at laparotomy in patients with Hodgkin's disease, congenital hemolytic anemia, hemolytic anemia of unknown etiology, and in 2 with traumatic rupture of a normal spleen.

In patients with portal or systemic circulatory congestion specimens were obtained at autopsy in 4 patients who died in congestive

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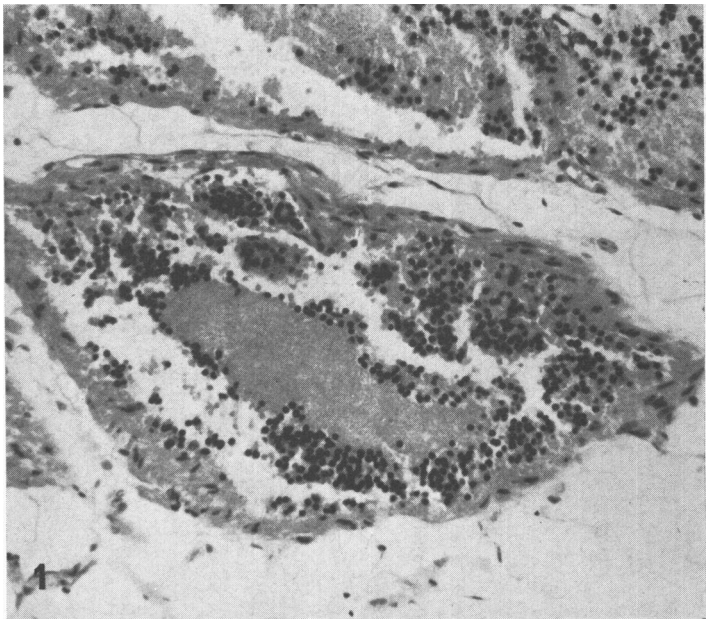


FIG. 1. Dilated splenic lymph vessel in patient W. D., who died in congestive heart failure. Lymphocytes as well as an occasional red blood cell can be seen in the lumen; 125×.

heart failure (pulmonary and peripheral edema, no ascites) and in 2 who died from hepatic cirrhosis and bleeding esophageal varices. An additional autopsy specimen was obtained from a patient who had cirrhosis and who died 5 days after porta caval shunt was performed for bleeding varices. One specimen was obtained at laparotomy from a patient who had combined schistosomal and postnecrotic cirrhosis.

Results. In sections from the 5 patients who died of causes unrelated to cirrhosis or congestive heart failure and from the 5 who underwent splenectomy for reasons unrelated to these 2 disorders, no dilated lymph vessels could be identified.

In sections from 4 patients who died in congestive heart failure the number of dilated lymph vessels per section varied from 2 to 4, all were markedly distended and in two the lymphatics contained many red blood cells (Fig. 1). The spleens weighed from 120 to 420 g and in only 1 patient could the spleen be palpated prior to death. Sections of the spleen disclosed findings consistent with chronic passive congestion in 3 of the 4. None of these patients had hypersplenism.

In the two patients with hepatic cirrhosis who died of bleeding varices and in one with cirrhosis who underwent splenectomy and spleenorenal shunt for variceal bleeding the number of dilated splenic lymph vessels varied from 2 to 4 and were markedly distended (Fig. 2A and B). In 2 of these 3 patients the lymph vessels contained innumerable red blood cells (Fig. 3). Ascites was present prior to death or operation in all three and splenomegaly and hypersplenism in 2. In one patient portal pressure was 28 cm of water 4 days before death and in the patient undergoing splenectomy it was 47 cm of water. The spleens of these 3 patients varied in weight from 540 to 1150 g and changes consistent with chronic passive congestion were seen in all. From both patients who died there were long histories of alcoholism and markedly deranged tests of liver function. Histologic sections of the liver from the third patient showed postnecrotic and schistosomal cirrhosis. In the remaining patient with cirrhosis and esophageal varices who died 5 days after an end-to-side portacaval shunt, no dilated splenic lymphatics were seen. Prior to portacaval shunt, portal pressure was 39 cm of water, and ascites was present but the spleen was not palpable. At

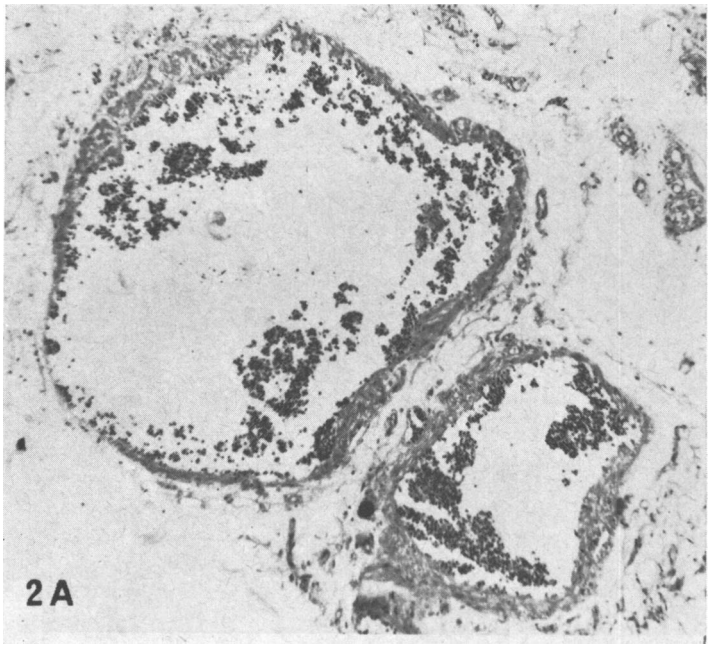
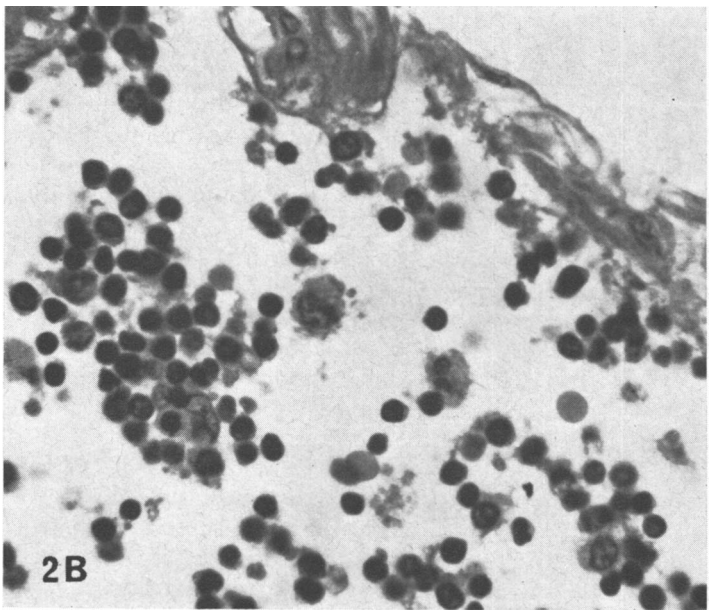


FIG. 2A. Two dilated splenic lymph vessels in patient S.B., who died of hemorrhage from esophageal varices; 65×. (B) Higher magnification of a section of the larger vessel in (A) demonstrating the thinness of the wall and absence of musculature. Lymphocytes are present in the lumen; 350×.



autopsy the spleen weighed 200 g and the shunt was patent. Clinical and autopsy data from these patients are summarized in Table I.

Discussion. In 1928 Barcroft and Florey found that following ligation of the splenic vein in cats excess splenic interstitial fluid formed and entered hilar lymphatics enroute

TABLE I. Chronic Circulatory Congestion.

Pt.	Age	Spleen wt (g)	No. of dilated lymphatics	Portal pressure (cm H_2O)	Platelets × 103	Autopsy or Clinical DX	Comments
S.B.	65	540	4			Laennec's cirrhosis	Exsanguinated from variceal hemorrhage
R.R.	27	1160	2	47	30	Schistosomal cirrhosis	Variceal hemorrhage spleno-renal shunt
E.R.	45	200	3	28	50	Laennec's cirrhosis	Exsanguinated from variceal hemorrhage
J.C.	47	200	0	39 preshunt	71	Laennec's cirrhosis	Died 5th day after patent porta caval shunt
W.D.	41	390	4		400	CHF	Old myocardial infarction
A.C.	94	145	3			CHF	ASHD
R.C.	42	420	2		280	CHF	Old myocardial infarction
E.T.	64	120	2		299	CHF	Cor pulmonale

to the thoracic duct (1).

The results reported here demonstrate that in patients with hepatic cirrhosis or congestive heart failure the splenic pedicle contains a number of dilated lymph vessels. As in the case of liver with dilated hilar lymphatics, such alterations reflect an increase in the formation of splenic lymph.

If local circulatory congestion is the predominant factor regulating the number of dilated splenic hilar lymph vessels, it seems curious that splenomegaly or hypersplenism or both were not a feature in patients with congestive heart failure who had striking alterations in these lymph vessels. In cirrhosis the origin of splenomegaly and hypersplenism may be more complicated than the mechanism suggested by the term "congestive splenomegaly" and involve derangements in hepatic sinusoidal blood flow, hepatic reticuloendothelial activity, and increased inflow into the portal system (9).

Cannulation of the thoracic duct in cirrhotic patients results in a marked decrease in size of the spleen (10). As splenic lymphatics drain to the cisterna chyli and thoracic duct it may be that facilitation of excessive lymph flow from the spleen is a factor in reduction in size and return toward normal function. Administration of chromium 51 tagged red cells to patient who had cirrhosis with bleeding varices and splenomegaly showed that with establishment of rapid lymph flow from the thoracic duct the spleen decreased in size and red blood cells sequestered in the spleen flowed out to be counted in the precordium.[3] Decrease in splenomegaly following thoracic duct cannulation thus tends to restore normal flow of formed blood elements through the spleen.

The observation that splenic lymph vessels in some patients with cirrhosis contain large numbers of intact red blood cells confirms an earlier finding by Jaeger (11). That this could be an important source of red blood cells in the thoracic duct lymph of cirrhotic patients is countered by the fact that thoracic duct lymph is grossly hemorrhagic in

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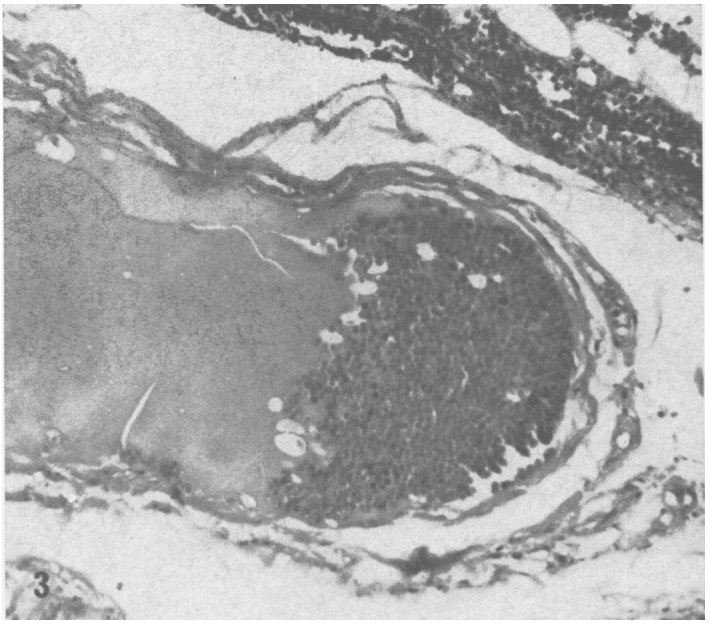

FIG. 3. Portion of dilated splenic lymph vessel in patient E.R., who died of hemorrhage from esophageal varices. The lumen contains lymph with a collectoin of red blood cells and lymphocytes along one side; 125×.

patients who have previously undergone splenectomy (12).

Presence or absence of red blood cells in splenic lymphatics bears directly on the question of whether the intermediate circulation of the spleen is open or closed. With one exception, previous attempts to answer this ignored the contents of splenic lymphatics and resulted in varied conclusions (13–15). Absence of red blood cells in splenic lymph vessels of experimental animals was noted previously by Godart and Hamilton (16). Considered together with the finding of red blood cells in dilated lymphatics of patients with splenic congestion these observations support the view that with respect to formed blood elements the intermediate circulation of the spleen is normally closed and in congested states may be open. This alteration may provide egress for excess fluid and cells from an overfilled vascular compartment into splenic lymph.

Summary. The number and content of dilated splenic hilar lymph vessels were examined in patients with and without congestion of the spleen. Sections from 10 patients without congestion showed no dilated lymph vessels. Sections from 7 patients with congestion all showed an increased number of dilated lymph vessels many of which contained red blood cells. One other patient who died after portal decompression demonstrated normal lymph vessels.

The results indicate that splenic hilar lymphatics enlarge as a result of splenic congestion and reflect an increase in splenic lymph formation. Absence of red blood cells in splenic lymph vessels under normal circumstances and their appearance in congested states supports the view that the intermediate circulation of the spleen is normally closed and that in congested states may be open.

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