Lymph Circulation in Congestive Heart Failure

Effect of External Thoracic Duct Drainage

By Marlys Hearst Witte, M.D., Allan E. Dumont, M.D.,
Roy H. Clauss, M.D., Bertha Rader, M.D., Norman Levine, B.S.,
and Ernest S. Breed, M.D.

SUMMARY
The lymphatic circulation was evaluated after cannulation of the cervical thoracic duct in 12 patients with severe intractable congestive heart failure. After venting the distended duct, lymph flowed rapidly under increased pressure, and signs and symptoms of circulatory congestion were dramatically relieved. Dyspnea, orthopnea, anorexia, abdominal discomfort, distended neck veins, hepatomegaly, peripheral and scrotal edema, and ascites diminished or disappeared. Central venous pressure fell and arm-to-tongue circulation time decreased. In some patients arterial pressure, amplitude of peripheral pulses, central venous Po2, and urinary output rose further suggesting enhanced cardiac output. Considered together with previous observations in experimental animals, the findings indicate that elevated systemic venous pressure greatly increases formation of thoracic duct lymph but at the same time impedes the return of lymph into the great veins. While therapeutic drainage of lymph from the thoracic duct in patients with cardiac failure is limited, the capacity of the lymphatic system to transport excess capillary filtrate back to the blood stream constitutes a major control mechanism regulating the manifestations of this disorder.

Additional Indexing Words:
Central venous pressure Circulation time Metabolic balance studies

CONTROVERSY concerning the pathogenesis of edema in congestive heart failure has centered around the manner in which decreased cardiac output and increased systemic venous pressure promote formation of excess capillary filtrate.1,2 According to one view, failure of the heart leads directly to a rise in venous pressure which is reflected in a rise in capillary pressure, increased capillary filtration, and extravascular fluid accumulation. Salt and water are retained by the kidneys in an effort to restore circulating blood volume. The alternative view holds that reduction in cardiac output itself stimulates salt and water retention by the kidney. Hypervolemia follows and leads to increased venous and capillary pressure, increased capillary filtration, and accumulation of extravascular fluid which is aggravated by anoxic damage to the capillary endothelium.


From the Departments of Medicine and Surgery, New York University School of Medicine, New York, and the Department of Medicine (Cardiology Division), Washington University School of Medicine, St. Louis, Missouri.

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### Data from 12 Patients with Congestive Heart Failure in Whom External Thoracic Duct Drainage Was Performed

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<th>Patient</th>
<th>Age (yr) and Sex</th>
<th>Diagnosis and clinical findings</th>
<th>CV pressure (cm H2O)</th>
<th>Previous treatment</th>
<th>Thoracic duct lymph flow (ml/min)</th>
<th>Thoracic duct lymph pressure (cm H2O)</th>
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</thead>
<tbody>
<tr>
<td>1. R.J.</td>
<td>47 F</td>
<td>RHD: MS, TS, TR, PH, AF, PVC, digitalis toxicity, cardiac cirrhosis IV-E</td>
<td>40</td>
<td>d, m, t</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>2. A.D.</td>
<td>33 M</td>
<td>RHD: MS, PAT, PVC, bigeminy, 7 years post MC, IV-E</td>
<td>28</td>
<td>d, a, t</td>
<td>7</td>
<td>28</td>
</tr>
<tr>
<td>3. M.B.</td>
<td>45 F</td>
<td>RHD: MS, PH (Ppa mean 75) AF, IV-E, CI 1.4 L (fixed), serum Na 128, K 5.9</td>
<td>24</td>
<td>d, acl, m, t, paracentesis, phlebotomy</td>
<td>5</td>
<td>30</td>
</tr>
<tr>
<td>4. C.B.</td>
<td>51 M</td>
<td>Marie Strümpell spondylitis: associated AR, 1 yr post-aortic valvuloplasty and aortoplasty, pulmonary emboli, intractable angina IV-E</td>
<td>26</td>
<td>d, a, acl, am, m, t, paracentesis, antithyroid drugs, morphine, abt</td>
<td>11</td>
<td>80</td>
</tr>
<tr>
<td>5. M.C.</td>
<td>73 F</td>
<td>RHD: MS, MR, TR, cardiac cirrhosis, IV-E obtunded, serum Na 127, K 5.6</td>
<td>27</td>
<td>d, acl, m, t, Southey tubes</td>
<td>9</td>
<td>30</td>
</tr>
<tr>
<td>6. E.B.</td>
<td>50 F</td>
<td>RHD: MS, MR, TR, 3 years post MC, IV-E</td>
<td>25</td>
<td>d, a, acl, m, t</td>
<td>12.5</td>
<td>23</td>
</tr>
<tr>
<td>7. R.Z.</td>
<td>41 M</td>
<td>RHD: AS, AR, MS, MR, SBE (active), cerebral and pulmonary emboli, IV-E obtunded, serum Na 122, K 3.9</td>
<td>27</td>
<td>d, a, acl, m, t, Southey tubes, abt, aeg</td>
<td>4</td>
<td>43</td>
</tr>
<tr>
<td>8. E.M.</td>
<td>58 M</td>
<td>HHD, ASHD: MI, ruptured IV septum, pulmonary emboli, IV-E, serum Na 126, K 3.6</td>
<td>23</td>
<td>d, a, acl, am, dx, m, t, thoracentesis, aeg</td>
<td>2</td>
<td>26</td>
</tr>
<tr>
<td>9. T.M.</td>
<td>47 F</td>
<td>CHD: IASD + ? MS, IV-E, anuria, severe hypotension, no palpable pulses. Serum Na 112, K 6.3</td>
<td>37</td>
<td>d, a, m, t, abt, aeg</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>10. V.T.</td>
<td>49 F</td>
<td>RHD: MS, 9 years post MC, PH (Ppa 110/30), AF, cardiac cirrhosis, IV-E hypotension, severe oliguria serum Na 118, K 6.8</td>
<td>30</td>
<td>d, a, am, m, t, morphine, Kayexalate</td>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td>11. M.V.</td>
<td>47 F</td>
<td>UHD, extreme obesity, PVC, digitalis toxicity, acute and chronic respiratory insufficiency with coma, IV-E, anuria</td>
<td>90</td>
<td>d, a, acl, m, t, positive pressure ventilation</td>
<td>3</td>
<td>43</td>
</tr>
<tr>
<td>12. J.B.</td>
<td>45 M</td>
<td>Idiopathic cardiomyopathy, pulmonary emboli, IV-E gangrene RLE</td>
<td>18</td>
<td>d, a, m, t, phlebotomy, aeg</td>
<td>4.5</td>
<td>19</td>
</tr>
</tbody>
</table>

**Abbreviations:** AF = atrial fibrillation; AR = aortic regurgitation; AS = aortic stenosis; CI = cardiac index (L/min/m²); CV = central venous; HD = heart disease; AS = arteriosclerotic, C = congenital, H = hypertensive, R = rheumatic, U = unknown; IASD = interatrial septal defect; IVSD = interventricular septal defect; IV-E = functional-therapeutic classification; LA = left atrium; LV = left ventricle; MC = mitral commissurotomy; MI = myocardial infarction; MR = mitral regurgitation; MS = mitral stenosis; Ppa = pulmonary artery pressure (mmHg); PAT = paroxysmal atrial tachycardia; PH = pulmonary hypertension; PM = postmortem; PV = plasma volume; PVC = premature ventricular contractions; RLE = right lower extremity; RLL = right lower lobe; RV = right ven-

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<tr>
<th>Thoracic duct lymph</th>
<th>Duration drainage (days)</th>
<th>Weight loss (kg)</th>
<th>Additional studies</th>
<th>Course</th>
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<tbody>
<tr>
<td>3.3 (82%)</td>
<td>7</td>
<td>10.9</td>
<td>Figs. 3, 4 (top), 5</td>
<td>2 wks later: CI 2.85L, PPA mean 48. Discharged after wt loss of +30 kg on d,a,e,m,t. Doing well +1 yr on d,a,m,t.</td>
</tr>
<tr>
<td>1.4 (21%)</td>
<td>7</td>
<td>12.3</td>
<td>Figs. 3, 4 (top), 5</td>
<td>Cannula removed. Sudden death during PAT 1 day later.</td>
</tr>
<tr>
<td>6.0 (75%)</td>
<td>7</td>
<td>3.9</td>
<td>Figs. 3, 4 (top), 5</td>
<td>Discharged on d,a,m,t. Severe CHF for 2 yr. PM: mitral valve diameter 2 mm, obliteratorive thrombus LA, multiple pulmonary emboli.</td>
</tr>
<tr>
<td>1.7 (31%)</td>
<td>8</td>
<td>25.0</td>
<td>Figs. 2–4 (top), 5, 7</td>
<td>Lymph clot in cannula. Died in pulmonary edema. PM: valvuloplasty incomplete with torn sutures, MI, bronchopneumonia</td>
</tr>
<tr>
<td>2.6 (39%)</td>
<td>3</td>
<td>10.9</td>
<td>Figs. 2–4 (top), 5, 7</td>
<td>Cannula fell out. Discharged on d,a,m. Doing well 6 mo later.</td>
</tr>
<tr>
<td>6.0 (85%)</td>
<td>2</td>
<td>0.45</td>
<td>Figs. 3–5</td>
<td>Surgery: mitral and tricuspid annuloplasty. Died 1 day PO.</td>
</tr>
<tr>
<td>2.0 (33%)</td>
<td>1.05 (17%)</td>
<td>3</td>
<td>Figs. 3–7 urine aldosterone</td>
<td>Discharged on d. Died suddenly at home 2 mo later.</td>
</tr>
<tr>
<td>3.8 (52%)</td>
<td>1</td>
<td>1.09</td>
<td>Figs. 3, 4 (top), 5</td>
<td>Surgery: IVSD repaired. Died PO.</td>
</tr>
<tr>
<td>3.3 (53%)</td>
<td>5</td>
<td>15.5</td>
<td>Figs. 2–5, 7</td>
<td>Pulses became palpable, extremities warm; CV oxygenation ↑. Cannula removed. Died suddenly 2 days later.</td>
</tr>
<tr>
<td>1.6 (35%)</td>
<td>1.46 (34%)</td>
<td>5</td>
<td>Figs. 3, 4 (top), 5–7 t 1/2 plasma aldosterone</td>
<td>Cannula clamped. Hypotension reappeared. Died 1 day later.</td>
</tr>
<tr>
<td>2.8 (32%)</td>
<td>0.75 (22%)</td>
<td>5</td>
<td>Figs. 3, 4 (top), 5–7</td>
<td>Awakened from coma. Urine output rose to normal. CV oxygenation ↑. Day 5: died after massive aspiration of food. PM: UHD.</td>
</tr>
<tr>
<td>0.7 (11%)</td>
<td>1.75 (25%)</td>
<td>3</td>
<td>Figs. 3, 4 (top), 5, 7, 9</td>
<td>Surgery: RLE amputation. Died 2 days later. PM: thrombi RV, LV, and common iliac artery; RLL pneumonia and infarction</td>
</tr>
</tbody>
</table>

tricle; SBE = subacute bacterial endocarditis; TR = tricuspid regurgitation; TS = tricuspid stenosis.

Abbreviations concerning treatment: a = Aldactone (spironolactone); abt = antibiotic; acg = anticoagulant; acl = ammonium chloride; am = aminophylline; d = digitalis; dx = Diamox (acetazolamide); e = ethacrynic acid; m = meralluride (Mercuhydrin); t = thiazide.

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The controversy remains unresolved, for while either theory explains certain clinical and experimental observations, neither one is compatible with all.

Fluid that leaves the intravascular compartment normally returns via venous and lymphatic pathways. However, once the heart fails as a pump, reabsorption is gradually impeded, the vascular space becomes overloaded, and fluid shifts into tissues. As venous congestion becomes intense, edema accumulates at a rate limited only by lymph flow and compliance of the interstitial space.\(^3\)

Starling was the first to suggest that derangements in the formation and transport of lymph bear directly on the pathogenesis of circulatory congestion in heart disease. He conceded, however, that his views, based in part on direct observations in a single dog with spontaneous heart failure,\(^4\) were largely “matters of surmise.” In the present study, the lymph circulation was evaluated in 12 patients with severe congestive heart failure. Flow, pressure, and composition of thoracic duct lymph were determined, and the effects of external lymph drainage on the manifestations of heart failure were examined. These observations extend and amplify earlier findings.\(^5\)

**Methods**

Twelve patients with severe congestive heart failure of several years’ duration were studied. Pertinent clinical data are presented in table 1. Marked dyspnea, orthopnea, anorexia, distended neck veins that filled from below, enlarged tender liver, ascites, and peripheral edema were present in all patients. Treatment with rest in bed, low-salt diet, digitalis, a variety of diuretic drugs, and mechanical removal of intracavitary or peripheral fluid had failed, and most patients were moribund with associated severe renal and electrolyte disturbances. In three patients closed cardiac surgery and in one patient open heart surgery had been performed previously. Functional-therapeutic classification was IV-E in all patients.

Under local anesthesia the cervical portion of the thoracic duct was cannulated with polyethylene tubing and lymph was collected by gravity drainage. Flow rate (ml/min) and end pressure (cm H\(_2\)O above the right atrium) were determined. Body weight, oral and intravenous intake of fluid, and output of lymph and urine were measured daily. Central venous pressure was monitored manometrically via a catheter in the superior vena cava or right atrium. External lymph drainage was continued for 1 to 8 days. Based on vital signs, central venous pressure, urinary output, and frequent hematocrit readings, lymph losses were partially replaced by increasing oral intake of salt-containing fluids or intravenous infusion of balanced salt solutions, in some instances with added serum albumin. No diuretic drugs were administered, but the daily maintenance dosage of digitalis (0.1 to 0.2 g of digitalis leaf or 0.25 to 0.5 mg of digoxin) was continued except in two patients in whom digitalis toxicity was suspected. After conclusion of the drainage period, the cannula was removed at the bedside and a pressure dressing applied.

Additional studies were performed as indicated in table 1 and figures 1 to 7: total protein content of lymph and plasma (12 patients), serial determinations of arm-to-tongue circulation time dehydrocholic acid (Decholin; three patients), serial levels of renin in plasma and lymph (modified method of Hellmer and Judson;\(^6,\)\(^7\) three patients), serial determinations of urinary aldosterone excretion (double isotope dilution derivative assay;\(^8\) one patient), disappearance rate of injected 3H-d-aldosterone (one patient), lymph and plasma BSP content 45 min after intravenous injection of 5 mg/kg (four patients), and serial plasma volume determinations (Evans blue dye-dilution method; two patients).

The thoracic duct was also cannulated in four patients with “compensated” heart disease (no visible edema) prior to corrective cardiac surgery.
Diminution in distention of neck veins (top), genital and thigh edema (middle), and pedal edema (bottom) after drainage of lymph from the thoracic duct in three patients with congestive failure.

Results

In patients with congestive heart failure, the thoracic duct was enlarged to a diameter of 5 to 12 mm (normal, about 2 mm). Thoracic duct lymph flow measured promptly after placement of the cannula was greatly elevated (mean, 7.8 ml/min; range 2 to 30 ml/min; normal, 0.82 ± 0.27). Lymph end pressure was greatly increased (mean, 32.4 cm H₂O; range, 17 to 80 cm H₂O; normal, 11.6 ± 6.6) and closely approximated central venous pressure (mean, 32.9 cm H₂O) in most patients (table 1 and fig. 1).

There was no correlation between the magnitude of the initial thoracic duct lymph...
Total weight loss as related to lymph output and urine output during entire period of lymph drainage in patients with congestive failure. Lymph losses were partially replaced by oral and intravenous administration of salt-containing fluids.

Lymph flow (mean, 1.2 ml/min; range, 0.75 to 2.0 ml/min) and end pressure (mean, 10 cm) in four patients with “compensated” heart disease did not differ significantly from normal values (fig. 1).

Total protein content in thoracic duct lymph (table 1 and fig. 1) was decreased to 46% of plasma ($P < 0.01$; range, 11 to 82%) while values in patients with compensated heart disease (mean, 70%; range, 57 to 79%) did not differ significantly from control subjects (72% ± 11%). Lymph BSP at 45 min
Figure 7

Metabolic balance studies during thoracic duct lymph drainage in six patients with chronic congestive heart failure. Symptoms and signs of circulatory congestion markedly diminished or disappeared as body weight declined. In each patient venous pressure fell, but in four patients (cases 4, 5, 10, and 11) not to normal levels. Venous hematocrit was generally stable except for a transient rise in case 4. The response of urine output varied; urinary sodium excretion remained low. Levels of blood urea nitrogen (BUN) varied but did not correlate with changes in urinary output. Although lymph flow generally declined in the first few hours after thoracic duct cannulation, lymph output did not decrease further except in case 9 where it fell spontaneously and in case 4 where the tip of the draining cannula was elevated.

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was 25% (range, 17 to 34%) of the simultaneous plasma level (table 1). No significant retention of BSP was observed at 45 min in plasma of control subjects and in two patients with compensated heart disease.

Within a few hours after thoracic duct cannulation, important changes were noted in the clinical status of each patient, and these changes persisted throughout the entire period of lymph drainage. Dyspnea and orthopnea diminished or disappeared, and anorexia and abdominal discomfort were relieved. Distended neck veins, hepatomegaly, peripheral and scrotal edema, and ascites also diminished or disappeared (fig. 2). Body weight decreased (mean, 9.5 kg; range, 0.5 to 25 kg) and corresponded to unreplaced lymph losses (table 1, fig. 3).

Central venous pressure fell from a mean of 32.9 to 14.0 cm H₂O (table 1, figs. 4 top and 7). Lymph flow rates fell in most patients as venous pressure decreased acutely, but abnormally rapid flow persisted even after venous pressure returned to normal. Arm-to-tongue circulation time decreased (fig. 4 bottom). Urinary output increased in four patients who were oliguric or anuric prior to the procedure (figs. 5 and 7). Plasma volume decreased in two patients (table 1).

Renin levels in lymph and plasma did not change consistently and remained elevated (fig. 6). In case 7 (fig. 7), urinary

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**Figure 8**

Schematic comparison of thoracic duct lymph formation and return to the venous system in heart failure and hepatic cirrhosis. In heart failure, generalized venous hypertension promotes excess lymph (○) formation in both peripheral and splanchnic tissues. At the lymphatic-venous junction in the neck, local resistance and increased central venous pressure impede lymph return, which leads to lymphatic dilatation and hypertension and edema (●). In hepatic cirrhosis, venous hypertension is confined to the splanchnic bed and increased volumes of splanchnic lymph are returned via the thoracic duct to normotensive veins. When the lymphatic system is overloaded, excess capillary filtrate leaks into the peritoneal cavity to form ascites (●).

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**Figure 9**

Case 12. Postmortem dissection of junction between the thoracic duct (TD) and subclavian vein (SV). Note markedly dilated thoracic duct (1 cm diameter) proximal to narrow channel of entry (arrow) into subclavian vein. (Dissection by Dr. Charles L. Witte.)
Aldosterone excretion (154 μg/day before and 380 μg/day after drainage) and renin levels in lymph and plasma remained elevated despite marked clinical improvement. In case 10 (fig. 7), the biological half life of aldosterone remained prolonged (55 min compared with 50 min preoperatively; normal, 20 to 40 min)\(^{11}\) despite a decrease in venous pressure and diminution in edema.

Four patients were subsequently discharged with striking improvement in their clinical status (table 1). Three other patients underwent surgery immediately after lymph drainage. Two patients died during lymph drainage and the remaining three died within one week following removal of the cannula.

**Discussion**

The partition of extracellular fluid between the vascular and interstitial fluid compartments is determined by the net effect of capillary filtration, plasma leakage through large pores, venous reabsorption, and lymph flow. Generally, as venous pressure is raised, filtration of solutes increases more than bulk loss of protein-rich plasma.\(^8\) As a result, tissue protein content and thus oncotic pressure decrease and these changes initially counterbalance the rise in capillary hydrostatic pressure. As venous pressure rises still further, this compensatory mechanism is insufficient, and filtration is favored even at the venous end of the capillary. At this point, excess interstitial fluid accumulates and only increased lymph flow can remove it.\(^3\)

In patients with congestive failure, the increased tendency for fluid to leave the vascular space and the great capacity of the lymphatic system to transport excess tissue fluid is reflected in the increased flow of lymph under high pressure from the cannulated thoracic duct. Normally, thoracic duct lymph arises almost entirely from the liver and extrahepatic portal bed.\(^{12}\) Liver sinusoids are freely permeable to plasma protein, and the absence of an oncotic gradient between plasma and hepatic interstitial fluid means that small increases in hydrostatic pressure produce large increases in formation of liver lymph, which remains high in protein.\(^{13,14}\) On the other hand, extrahepatic portal capillaries are less permeable, and as venous pressure rises, the protein content of intestinal lymph falls.\(^{14}\) In chronic severe congestive failure, thoracic duct lymph protein is usually greatly diminished, a finding which indicates that excess lymph originates not only from the liver but also in large part from less permeable extrahepatic portal and peripheral capillaries which form a progressively more dilute filtrate as venous pressure rises. Similarly, 45-min BSP content of thoracic duct lymph (relative to plasma), a marker of liver lymph in the thoracic duct,\(^{10,15}\) is also low, as it is in patients with advanced hepatic cirrhosis in whom extrahepatic portal hypertension predominates.\(^10\)

Although lymph flows at an accelerated rate when the thoracic duct is cannulated, the presence of edema prior to this maneuver means that flow in the unvented lymphatic system has not kept pace with the rate of lymph formation\(^{16,17}\) (fig. 8). In 1937, McMaster\(^{18}\) observed that colored dyes injected subcutaneously into the edematous legs of patients with cardiac failure entered widely dilated, richly intercommunicating lymphatic channels but consistently failed to show "streamer" formation or forward flow. Instead, coloring matter pooled in lymphatics, extravasated into tissues, and even flowed retrograde. Great dilatation of the thoracic duct and its tributaries, ballooning of the duct as it approaches the relatively narrow junction of the thoracic duct with the jugular vein (fig. 9), equilibration of thoracic duct end pressure and central venous pressure, and sluggish to-and-fro motion of venous blood refluxing through the cervical junction further attest to generalized impairment to lymph flow in heart failure.

At least two important factors limit the flow of lymph into the venous system: local resistance at the thoracic duct-jugular vein junction\(^{19,20}\) and central venous pressure itself. In patients and experimental animals with right heart failure, thoracic duct lymph flow, measured by several different methods, is impeded by a high outlet pressure, although

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flow into the vein may be somewhat greater than normal for a given pressure.\textsuperscript{21-24} Thoracic duct end pressure, the net force propelling lymph onward to the great veins, is also increased,\textsuperscript{24, 25} but generally to levels no higher than corresponding central venous pressure, thus providing little or no gradient for lymph flow.

Cannulation of the distended thoracic duct results in a copious flow of lymph and mobilizes excess extravascular fluid. As lymph is drained externally and only partially replaced, the signs and symptoms of circulatory congestion diminish or disappear. The lymphatic system is again able to siphon off excess fluid from the venous system, and central venous pressure falls. Arm-to-tongue circulation time decreases, and this decrease suggests improvement in cardiac output, supported in some patients by a rise in arterial blood pressure, increased amplitude of peripheral pulses, improvement in central venous oxygenation, and increased urine output.\textsuperscript{26} However, urinary sodium excretion remains low and the renin-aldosterone system is not suppressed.

Furthermore, the manifestations of circulatory congestion can be relieved even without external lymph losses if lymph return is increased to match lymph production. For example, when the thoracic duct is anastomosed to a normotensive pulmonary vein in dogs with isolated right-heart failure from pulmonic stenosis and tricuspid insufficiency,\textsuperscript{24} sequestered lymph behind the right heart is effectively diverted to the left heart. Reduction in central venous pressure, natriuresis, diuresis, and loss of ascites follow. Similarly, in hepatic cirrhosis (fig. 8), another disorder characterized by increased production of thoracic duct lymph,\textsuperscript{10, 19} edema in the form of ascites is not always found. Here, venous hypertension is confined to the splanchnic bed, and as long as excess lymph drains into normotensive systemic veins as rapidly as it is formed in the splanchnic bed, ascites does not appear.\textsuperscript{27} Moreover, in dogs with ascites secondary to constriction of the supradiaphragmatic inferior vena cava or hepatic veins, either enlargement of the thoracic duct-jugular vein junction\textsuperscript{28} or anastomosis of the thoracic duct to intrathoracic systemic veins of lower pressure\textsuperscript{29, 30} leads to increased lymph flow in the thoracic duct and loss of ascites.

In patients with heart disease, when capillary filtrate forms more rapidly than it returns, excess interstitial fluid accumulates, and effective arterial volume decreases. A complex sequence of events is set into motion leading to retention of salt and water, which tends to restore this “lost” volume, but at the same time promotes further capillary filtration.\textsuperscript{27} The capacity of the lymphatic system to transport excess filtrate back to the blood stream plays a key role in regulating the manifestations of congestive heart failure.

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References
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