Thoracic Duct-to-Pulmonary Vein Shunt in the Treatment of Experimental Right Heart Failure


SUMMARY
Elevated venous pressure in right heart failure leads not only to an increase in lymph formation but also to progressive resistance in the neck to the return of lymph to the circulation via the thoracic duct. Sequestration of fluid behind the failing heart tends to protect the circulation but at the same time leads to the clinical manifestations of heart failure.

The present study was performed on 40 dogs with combined tricuspid insufficiency and pulmonary stenosis. Thoracic duct lymph flow was greatly increased. Pressure was considerably greater in the systemic veins than in the pulmonary vein beyond the right heart obstruction. Lymph flow was substantially enhanced when the thoracic duct was connected to the lower pressure pulmonary veins. Furthermore, direct anastomosis of the thoracic duct to the pulmonary vein resulted in fall in systemic venous pressure, increased in renal excretion of salt and water, and reduction in ascites. These results indicate that alterations in the flow of thoracic duct lymph have important bearing on the manifestations and treatment of right heart failure.

Additional Indexing Words:
Lymph            Lymphatic-venous shunt           Pulmonic stenosis
Tricuspid insufficiency    Venous pressure           Ascites

THE LYMPHATIC SYSTEM is the final pathway for return of excess interstitial fluid to the vascular compartment. The presence of edema from any cause, therefore, may be considered to represent failure of this system.1-3 Inadequate lymphatic drainage and edema formation due to mechanical blockage of lymphatic channels are well recognized. On the other hand, the concept that a normal or "hypertrophied" lymphatic system may be incapable of carrying off an excess volume of lymph is more subtle.

Increased pressure in the systemic veins behind the failing right heart leads to increased transfer of fluid into the interstitial space. This same increased central venous pressure provides progressive resistance to the return of lymph to veins in the neck.4,5 When an external thoracic duct fistula is created in the patient with right heart failure, systemic venous hypertension and edema are relieved.6 Observations on the dog with hepatic outflow obstruction indicate that, even

From the Departments of Surgery, Medicine, and Pathology, Washington University School of Medicine, and St. Louis City Hospital, St. Louis, Missouri.

Investigation was supported by Grant HE-09073-CV-03 from the U.S. Public Health Service, and grants from the St. Louis Heart Association and the Institute of Medical Education and Research of St. Louis City Hospital.

Paper was presented in part at the meeting of the American Heart Association, New York City, October 21-25, 1966.

Dr. Witte is an Established Investigator of the American Heart Association. Mr. Kash is a third year medical student.

Circulation, Volume XXXVI, October 1967
without external loss of lymph, ascites disappears rapidly when splanchnic lymph removal is enhanced by anastomosis of the thoracic duct to an intrathoracic systemic vein of lower pressure than the subclavian vein. The present study was designed to evaluate the effect of improving lymph drainage by diverting the flow of thoracic duct lymph around the failing right heart into the low pressure pulmonary veins.

Table 1

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Effects of Shunts on Venous Pressure and Sodium and Water Excretion in Dogs with Right-Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of dogs in group</td>
</tr>
<tr>
<td>Venous pressure (cm H₂O) above atrium</td>
<td>17</td>
</tr>
<tr>
<td>Sodium excretion (mEq/hour)</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>7</td>
</tr>
<tr>
<td>Chronic</td>
<td>9</td>
</tr>
<tr>
<td>Water excretion (ml/hour)</td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>8</td>
</tr>
<tr>
<td>Chronic</td>
<td>12</td>
</tr>
</tbody>
</table>

*Calculated as average of the individual % changes.
†Average of consecutive hourly measurements within 24 hours prior to surgery.

Methods

Forty mongrel dogs were placed in metabolic cages and fed a constant 60-mEq sodium diet. Weight, abdominal girth, urinary output of water and sodium, and central venous pressure were followed before and after creation of right heart failure by simultaneous production of tricuspid insufficiency and pulmonary stenosis.

In seven dogs with ascites following this maneuver, the thoracic duct was cannulated in the right chest under pentobarbital anesthesia (25 mg/kg), and the chest was closed. Phasic and mean pressures in the superior vena cava, subclavian vein, pulmonary vein, and thoracic duct were recorded simultaneously on a Hewlett-Packard multichannel polygraph. Multiple determinations of lymph flow were made within the range of mean venous pressures observed.

In 17 other dogs with ascites, a permanent connection between the thoracic duct and left inferior pulmonary vein was created by several different methods—direct end-to-end or end-to-side anastomosis with 7-0 silk or Dacron or sutureless anastomosis. In eight dogs hourly measurements (acute) of urinary sodium and water excretion were made up to 6 hours following this procedure. The animals were then returned to their metabolic cages, and preoperative observations (chronic) were continued. Patency of shunts was evaluated several days later either by lymphangiography or direct examination.

Autopsies were performed on all dogs. The experimental cardiac lesion was confirmed, the lymphatic-venous shunt examined, and the volume of ascites measured.
An example of the relationship between venous pressure (superior vena cava and pulmonary vein) and lymph flow in experimental right heart failure.

Results

Lymph flow rate after production of right-heart failure varied between 1 and 23 cc/min (normal flow rate is 0.25 to 0.5 cc/min). As the tip of the thoracic duct cannula was raised above the zero level (right atrium), lymph flow fell (fig. 1). In one dog typical of the group (fig. 2), mean pressure was 8.5 cm of H₂O lower in the pulmonary vein than in the superior vena cava. Lymph flow at the pulmonary vein pressure was 4.2 cc/min, and there was no flow at the superior vena cava pressure.

Following construction of a thoracic duct-to-pulmonary vein shunt, the thoracic duct appeared less distended. Important changes were subsequently noted in systemic venous hypertension, urinary sodium and water excretion, and ascites (table 1, fig. 3). Systemic venous pressure fell promptly in all dogs.
(mean change, −37.4%). Urinary sodium excretion increased within a few hours in all dogs (mean change, +1.190%) and remained higher than during the period of untreated heart failure (mean chronic change, +43%). Seven of eight dogs had a concomitant acute increase in water excretion (mean change, +157%) although the increase did not persist (mean chronic change, +7%). In the dogs whose urinary water excretion did not rise, the total sodium excretion nonetheless remained greater than in the pre-shunt period. Ascites diminished or disappeared in 10 of the 13 dogs (77%) who survived long enough for postoperative data to be obtained. Reaccumulation of ascitic fluid occurred in three of these 10 dogs (30%), and in each instance the shunt was found to be thrombosed. In one of the three dogs in which ascitic fluid reaccumulated, a subsequent anastomosis of the thoracic duct to the right superior pulmonary vein resulted in complete loss of ascites for the second time.

Changes in weight and abdominal girth in one dog are shown before and after a successful thoracic duct-to-pulmonary vein shunt (fig. 4). Following the shunt natriuresis (+928% acute and +650% chronic) and diuresis (+70% acute and +7% chronic) occurred as weight and abdominal girth returned to their pre-heart failure values.

**Discussion**

The clinical manifestations of heart failure are related to a fall in cardiac output leading to decreased perfusion of vital organs and to a rise in systemic venous pressure resulting in increased formation of interstitial fluid or lymph. Sequestration of plasma in the interstitial space serves as a "safety valve" to the circulation protecting the failing heart from volume overload. However, under the special circumstances when heart failure arises from an anatomic or functional obstruction in the right heart or lesser circulation, it is likely that "protection" of the left heart is neither necessary nor desirable. Increased return of lymph to the left heart beyond the obstruction might then be expected to relieve the consequence of venous hypertension (such as peripheral edema and ascites) without compromise to, or perhaps even with an improvement in, the output of the left heart.

The present studies demonstrate that thoracic duct lymph formation is greatly increased in experimental right heart failure as it is in patients with right heart failure. The data indicate that systemic venous hypertension poses a significant resistance to the return of thoracic duct lymph to the heart.
in this condition and that lymph flow can be enhanced by connection of the thoracic duct to low pressure pulmonary veins beyond the experimental obstruction in the right heart. Enhancement of lymph flow is even greater than in the dog with a caval constriction after connection of the thoracic duct to lower pressure veins in the chest rather than to the collapsible neck veins. Following a permanent anastomosis between the thoracic duct and pulmonary vein, systemic venous pressure falls, renal salt and water excretion rises, and ascites diminishes or disappears. These findings suggest that the systemic venous hypertension, renal salt and water retention, and ascites seen in right heart failure are related as much to the inadequate return of excess interstitial fluid by the lymphatics as to the cardiac lesion itself which initially caused the increased production of lymph.

The data already obtained in this study have direct pertinence to right heart failure in man when the left heart and kidneys are able to handle increased return of sequestered lymph to the arterial side of the circulation. This situation arises in isolated right-sided valvular lesions, chronic pulmonary disease, and pulmonary hypertension of diverse etiologies—disorders frequently intractable to current medical and surgical management. Further study of the role of the thoracic duct lymph system in right heart failure should provide important information on the relationship between discrepancies in the amount of lymph formed and returned to the circulation and the pathophysiology of right heart failure.

Acknowledgment

We are indebted to Miss Kathleen Kintner for her help in carrying out these studies.

References

Thoracic Duct-to-Pulmonary Vein Shunt in the Treatment of Experimental Right Heart Failure

WILLIAM R. COLE, MARLYS HEARST WITTE, STEPHEN L. KASH, MALCOLM RODGER, VIRGIL R. BLEISCH and GERHARD H. MUELHEIMS

Circulation. 1967;36:539-543
doi: 10.1161/01.CIR.36.4.539

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1967 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/36/4/539

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/