

## CONTRIBUTION OF THE LIVER TO THORACIC DUCT LYMPH FLOW IN A MOTIONLESS SUBJECT

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### ABSTRACT

To ascertain the contribution of the liver to thoracic duct lymph (TDL) flow in a resting subject, afferent hepatic blood flow was temporarily interrupted in dogs by placing an atraumatic clamp across the hepatoduodenal ligament containing the hepatic artery, portal vein and 80% of hepatic lymphatic drainage. To circumvent extrahepatic splanchnic venous sequestration, a side-to-side portacaval shunt (S-S-PCS) was constructed prior to interrupting blood flow. Portal venous pressure, cervical TDL flow, and total protein content were serially monitored.

TDL and total protein after S-S-PCS was comparable to that recorded in dogs without celiotomy ( $0.60 \pm 0.17$  ml/min and  $3.4 \pm 0.5$  g/dl, respectively). Interruption of hepatic blood flow was associated with a fall in TDL flow ( $0.38 \pm 0.8$  ml;  $p < 0.001$ ) and protein content ( $2.8 \pm 0.7$  g/dl;  $p < 0.01$ ) and TDL/plasma protein ratio ( $0.58 \pm 0.7$  to  $0.48 \pm 0.05$ ;  $p < 0.01$ ).

These data suggest that in the absence of supplemental fluid administration or other exogenous stimulation, hepatic lymph contributes one-third of resting TDL flow.

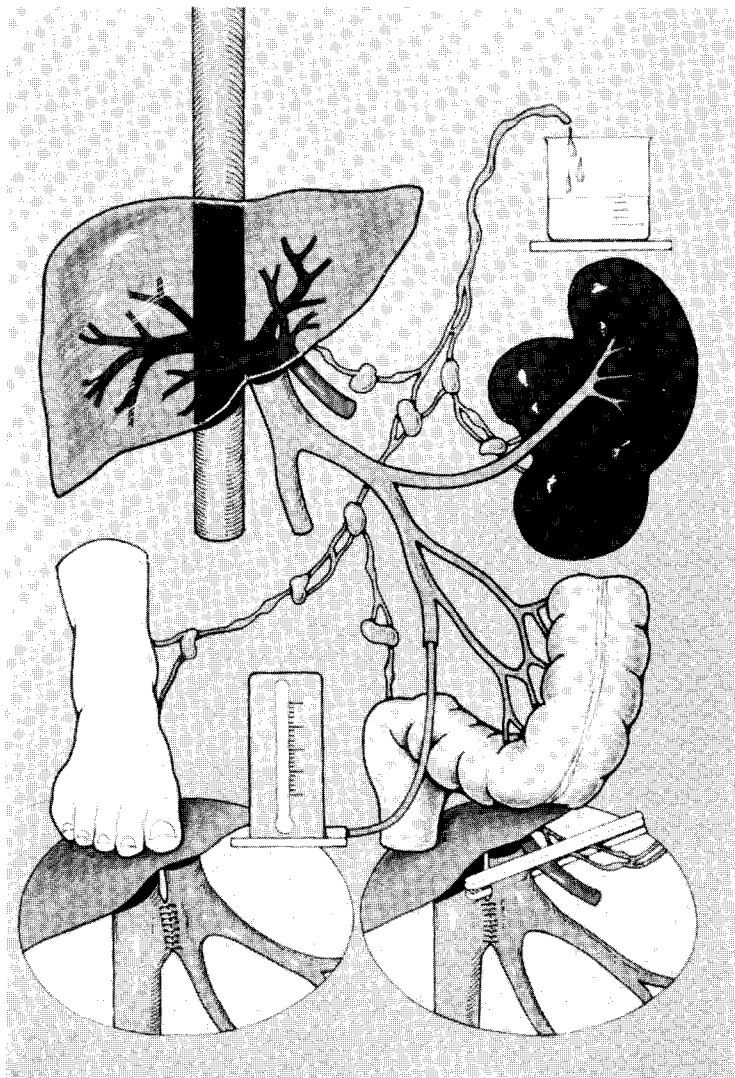
The thoracic duct transports 85-90% of total body lymph and essentially all lymph from the abdominal viscera and lower extremities. In a motionless subject, however, peripheral lymph flow is minuscule and accordingly, at rest, thoracic duct lymph derives primarily from the liver, gastrointestinal tract and its append-

ages, with a smaller contribution from the kidneys. Several studies have further suggested that the hepatic component in a motionless subject represents one-half or more of thoracic duct flow based largely on extrapolations from flow measurements in cannulated hepatic lymphatics alone or in conjunction with simultaneous measurements from exteriorized mesenteric and/or central lymph trunks (1,2).

Whereas the liver has an enormous capillary surface area for fluid exchange and highly permeable endothelium, hydrostatic pressure is uniquely low ( $\sim 5$  mmHg) with little or no transcapillary oncotic gradient. Despite exquisite sensitivity of transsinusoidal water flux to intrahepatic microvascular hypertension (e.g., after thoracic inferior vena caval compression) (3), Starling forces in the liver under normal conditions are nonetheless closely balanced. Accordingly, the hepatic contribution to thoracic duct lymph (its major "lymph-shed") may, in a subject at rest, be considerably less than 50%.

### MATERIALS AND METHODS

In 7 large dogs (average weight 27kg) fasted overnight an *in situ* anhepatic model was prepared by excluding the liver without extirpation. Under light pentobarbital anesthesia (25mg/kg I.V.) total afferent hepatic blood flow and 80% or more of hepatic lymph drainage was simultaneously interrupted by cross-clamp-



*Fig. 1. Experimental design. Afferent hepatic blood flow was temporarily interrupted using an atraumatic clamp across the hepatoduodenal ligament containing the hepatic artery, portal vein and 80% of hepatic lymphatic drainage (lower right oval "window"). To circumvent splanchnic venous sequestration, a side-to-side portacaval shunt was constructed prior to interrupting hepatic blood flow (lower left oval "window"). Portal venous pressure was continuously monitored via a catheter in a jejunal vein. The thoracic duct was cannulated in the left neck and central lymph flow monitored at ten minute intervals by gravity drainage.*

ing with an atraumatic hemostat the hepatoduodenal ligament containing the hepatic artery, portal vein, and bulk of draining hepatic lymphatics (Fig. 1). To

circumvent extrahepatic splanchnic venous pooling and portal hypertension and with it systemic hypotension, a side-to-side portacaval shunt was constructed before-

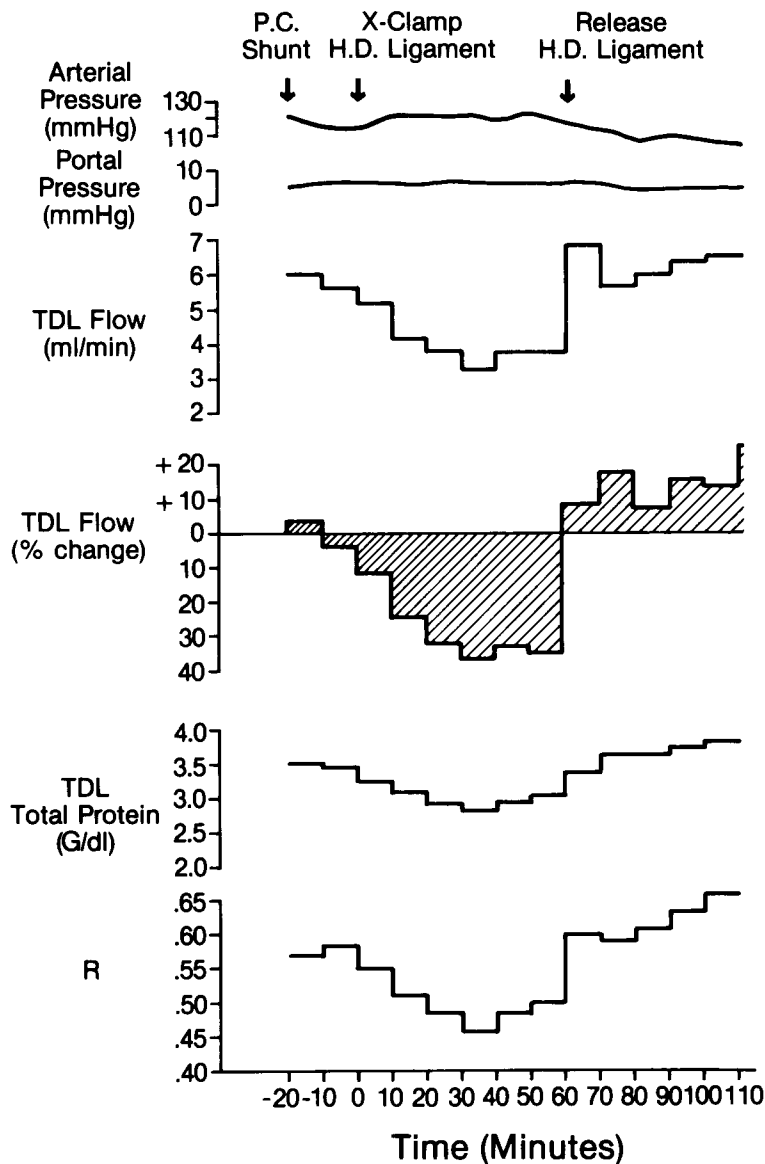


Fig. 2. Hemodynamics and lymph kinetics obtained in experimental preparation outlined in Fig. 1 (data represent the mean values of 7 dogs). With stable systemic and splanchnic hemodynamics, exclusion of the liver by interruption of hepatic afferent blood flow was associated with a sharp fall in central lymph flow and protein content. Unclamping with return of hepatic blood flow restored (with a slight "overshoot") these values. P.C. = portacaval; H.D. = hepatoduodenal ligament (contains hepatic artery and portal vein); TDL = thoracic duct lymph; R = thoracic duct lymph/plasma protein ratio.

hand. Changes in cervical thoracic duct lymph flow and total protein content were serially determined before, during, and

after interruption of the hepatic blood and lymph circulations (Fig. 1). No exogenous fluids were administered.

## RESULTS

In these large dogs, control thoracic duct lymph flow ( $0.60 \pm 0.17$  ml/min, mean  $\pm$  SD) and protein content ( $3.4 \pm 0.5$  g/dl) after side-to-side portacaval shunt was comparable to that recorded in dogs without celiotomy. Interruption of all afferent blood flow to the liver and adjacent periportal duct lymph flow was uniformly associated with a fall in thoracic duct lymph flow ( $0.38 \pm 0.08$  ml/min;  $p < .001$ ) and protein content ( $3.4 \pm 0.5$  to  $2.8 \pm 0.7$  g/dl;  $p < .01$ ) and thoracic duct lymph/plasma protein ratio (R) ( $0.58 \pm .07$  to  $0.48 \pm .05$ ;  $p < .01$ ). Plasma protein levels were unchanged (range 5.8 to  $6.2 \pm 0.7$  g/dl). Moreover, release of the ligature with reperfusion of the liver (one hour warm ischemia) promptly returned central lymph flow and protein composition toward normal (Fig. 2). Systemic and splanchnic hemodynamics were minimally altered as indicated by a stable arterial and portal venous pressure.

## COMMENT

In contrast to previous experimental designs, the contribution of the liver to thoracic duct lymph was examined using a technique that permitted *in situ* circulatory exclusion of the liver with easy reversibility. This preparation avoided injury to regional hepatic lymphatics (either by cannulation or ligation of accessory channels) while allowing lymphatic manipulation and lymph measurements in a central watershed (the thoracic duct) remote from the liver itself. Because the protein composition of hepatic lymph is close to 80-85% of plasma whereas that in the alimentary tract and urogenital system is closer to 50-60% of plasma, changes in

central lymph flow and composition in this preparation reflect solely the hepatic lymph contribution to the thoracic duct. Thus, liver exclusion produces on the average a 34% decrease in central lymph flow with a comparable fall in thoracic duct lymph protein and thoracic duct/plasma protein ratio. Upon release of the occluding clamp and restoration of hepatic perfusion and periportal lymph flow, thoracic duct lymph flow, protein content, and thoracic duct/plasma protein ratio promptly return as transsinusoidal pressure, microvascular filtration and hepatic lymph formation is reestablished.

Based on the alterations in thoracic duct lymph flow and protein composition before and after interruption of afferent blood flow to the liver, we conclude that in the absence of supplemental fluid administration or other exogenous stimulation, hepatic lymph contributes one-third of resting thoracic duct lymph flow.

## REFERENCES

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