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# Study on Lymphatico-Venous Shunts at the Cardia of Dogs with Acute Portal Hypertension

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**Summary**

Lymphatico-venous (L-V) anastomosis at the cardiac end of the stomach was found in 5 out of 19 dogs with severe lymphatic stress and hexamethonium injection. The significance of such a finding is discussed in relation to the genesis and rupture of oesophageal varices complicating portal hypertension.

## *Introduction*

L V anastomosis has been proven to exist in many sites of both experimental animals and man (1, 2, 4, 7–9, 12–14, 21–25), including the mesentery (20, 24). It has been the opinion of some authors that they do exist at the area where oesophageal varices develop (5, 6).

The aim of the present work is to investigate the presence of such a type of shunt at the cardiac end of the stomach of dogs.

## *Material and Methods*

Thirty four adult male and female mongrel dogs weighing from 17 to 24 kg were used in this work and divided into two groups: a) group 1 (15 dogs), in which the study was proceeded under normal physiological conditions, and b) group 2 (19 dogs), in which stress of the lymphatics of the area to be studied was created by induction of portal hypertension and ligation of the afferent lymphatics of the draining lymph nodes. Intravenous hexamethonium was administered to the same group (1 mg/kg body weight), one hour before dissection.

Anaesthesia was by thiopentone sodium in 10 ml saline (0.025 g/kg body weight) for induction and barbitone sodium solution in saline (0.25 g/kg body weight) given by I.V. drip for maintenance. The abdominal cavity was opened for passage of polyethylene catheter into the portal system by the way of a superior mesenteric vein tributary, for portal venous pressure measurements 30 minutes later. Portal hypertension was created by partial occlusion of the portal vein using a vascular clamp and the portal pressure was remeasured after 30 minutes. The afferent lymphatics of the draining lymph nodes of the stomach, near the portal vein were ligated and one hour before dissection hexamethonium was injected. Saline was administered to support arterial pressure when needed, without venous overloading. In all animals, dissection started 2 hours after creation of portal hypertension. The stomach with its omenta and the lower end of the oesophagus were dissected and inserted in a water bath at a temperature of 45 °C. The main stem of the left gastric vein was cannulated and washed with running water then coloured gelatin solution was injected at a temperature of 60 °C and a low pressure (20–40 mmHg). Depots (2–3 mm in diameter) of india ink were injected under the serosa of the cardiac end of the stomach and lower end of the oesophagus, and the points of injection were screened off with sponges so as to prevent serosal contamination with dye. The specimens were submitted to immediate gross observation for the distribution of lymphatics on the surface of the cardia and lower end of the oesophagus as well as for the presence of L-V shunts.

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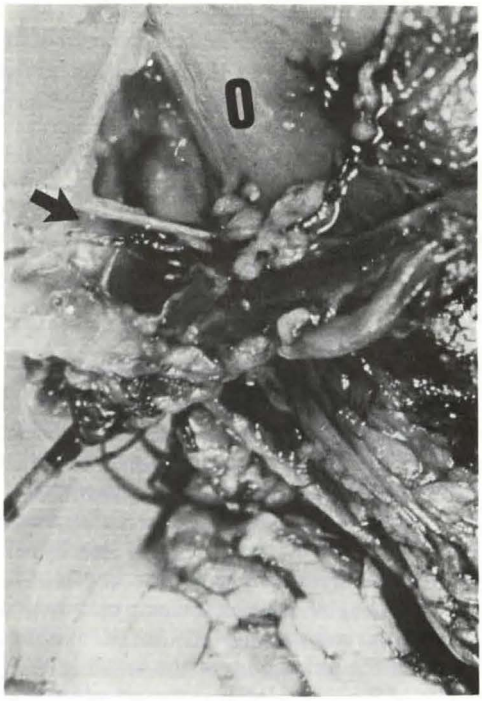


**Fig. 1** Photograph, showing lymphatico-venous anastomosis (arrow), between subserosal lymphatic of the cardiac end of stomach and tributary of left gastric vein. O = oesophagus

Junctional areas were fixed in buffered 10% formalin, then processed for microscopic examinations after staining with hematoxylin-eosin stain.

*Results*

None of the fifteen control dogs had lymphatico-venous anastomosis.

Among the 19 dogs of the second group, where the portal pressure was significantly elevated from 12.2 ± 2.3 to 32.4 ± 3.1 cm saline (mean ± S.E.), two hours before dissection, the afferent lymphatics of the draining lymph nodes of the stomach were ligated and hexamethonium was injected, L-V anastomosis was found in 5 dogs (Fig. 1–3).

*Discussion*

L-V anastomoses are probably enlargements of pathways normally present but do not function except under the stress of increased volume or pressure within the lymphatics (1, 3, 17, 18, 21, 29, 30). In the present study portal hypertension was created in 19 dogs and the afferent lymphatics of the draining lymph nodes of the stomach near the portal vein were ligated so as to produce both increased volume and pressure in the lymphatics


**Fig. 2** Photograph showing lymphatico-venous anastomosis (LV)
L = subserosal lymphatic
V = main stem of left gastric vein


**Fig. 3a** Photograph showing lymphatico-venous anastomosis at cardia (arrow)
L = subserosal lymphatic
V = tributary of left gastric vein

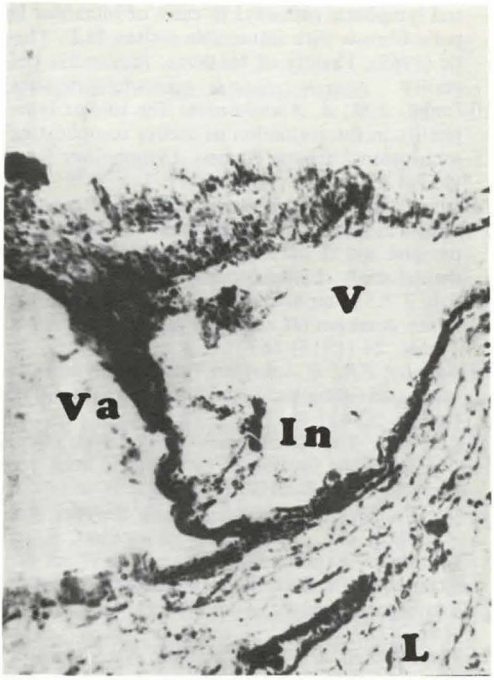


**Fig. 3b** Microphotograph of same specimen. The lymph vessel (L) passes within the adventitia of the vein (V).
In = India ink particles within lumen of the vein
Va = venous valve

of the area to be studied. In addition hexamethonium, the autonomic ganglionic blocking drug, was injected for the possibility of opening non functioning L-V communications (16). In 5 dogs with such severe lymphatic stress, L-V anastomoses were demonstrated. Portal hypertension leads to excessive lymph production (5, 6, 15, 18), that exceeds the thoracic duct drainage capacity (5, 9, 19, 31). As the sources of such excessive lymph in presinusoidal obstruction is the extrahepatic portal bed (10, 11, 26–28), the excess lymph from the cardiac end of the stomach is possibly diverted to the tributaries of the portal venous system via the L-V anastomosis that proved to exist in such area, and may play a role in the development and rupture of oesophageal varices.

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