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HISTOLOGIC ASPECTS OF FAT DEPOSITION FOLLOWING THORACIC DUCT LIGATION AND PORTAL INFUSION AND A CONSIDERATION OF PORTAL PRESSURE VARIATIONS IN THE DOG

By

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SUMMARY

Ligation

Three of the twelve dogs that were ligated developed ascites. The intestines of all ligated fat-fed animals appeared yellow and edematous.

Frozen sections of duodenal biopsies taken from control animals and stained with oil red O, demonstrated low amounts of neutral lipid in the epithelium, lamina propria and central lacteals. Ligated animals presented moderate amounts and ligated fat-fed animals the greatest amounts. This was thought to be attributable to the blockage of lymph absorption. Lipid stratification in the epithelium was found in ligated fat-fed animals but not in control and ligated animals. It was thought to be the effect of hydrophobic bonding.

Lipid accumulation was first noted in the basal portion of the epithelial cells, while one in 60 villi examined appeared ruptured in ligated fat-fed animals.

The jejunum demonstrated a greater amount of lipid than the duodenum and ileum owing to its prominent role in lipid absorption. Concentrations observed within the epithelium, lamina propria and central lacteals all appeared least in control animals, more in ligated animals and most pronounced in ligated fat-fed animals. Stratification of lipid granules appeared intracellularly in epithelial cells. Both the jejunum and duodenum demonstrated swollen and ruptured villi in approximately the same frequency.

The number of lipid granules present within the ileum was insignificant. As before, lipid granules were located in the basal portion of the cells.

Liver tissue in control animals contained lipid granules in zone one and three but not in two. In ligated animals concentrations were greater in zones one and three, while livers of ligated fat-fed animals showed the greater concentration in all zones. Sinusoidal diameter increased 3.8% in ligated and 5.3% in ligated fat-fed animals. Sinusoidal lipid also increased in ligated fat-fed dogs. This indicates that due to the ligation of the thoracic duct, there was an increase in lipid concentration in all three zones of Rappaport ('54), and was attributable to mobilization or obstruction of neutral lipid absorption.

Intralipid

A study was made of portal infusion of Intralipid in dogs. Frozen histological sections of the liver of control animals revealed granules of neutral lipid concentrated intracellularly in zones one and three of Rappaport ('54), and minimal amounts in zone two. Sinusoids were free of neutral lipid. Neutral lipid was also concentrated at the base of the cell of the bile duct epithelium. No lipid was seen in the lymphatics but some granules were present within the lumen of the portal vein branch of the triad.

In experimental animals neutral lipid was equally concentrated in zones one and three. However, zone two appeared to begin to acquire lipid at the fourth hour of

Intralipid infusion. All deposition was intracellular. Slight increases were noted in the sinusoids and the lumen of the sublobular veins, while the portal canal revealed increased lipid concentration at the apical portions of bile duct epithelium and in the lumen of the portal vein.

Hemodynamic Considerations

In mongrel dogs the thoracic duct and portal vein were cannulated to measure TDF and PVP. Varying dosages of angiotensin II were administered systemically to ascertain the relationship between TDF and PVP. It would appear that angiotensin II brings about an increase in TDF by increasing post hepatic resistence, which results in elevated PVP and Pc in intestinal and hepatic vasculature. The increased TDF when converted to pressure (Werner, '65) and compared to PVP provides a pressure gradient which could maintain flow in an LTPB.

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