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Bilirubin Resorption in Obstructive Jaundice.

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There is a controversy as to the mechanism of resorption of bilirubin from the liver whose common duct is obstructed. Bloom,[1] Harley,[2] and Eppinger[3] have claimed that absorption in obstructive jaundice occurs through the lymphatics of the liver, the bilirubin reaching the blood stream through the thoracic duct. Others, Whipple and King,[4] Bollman, Sheard, and Mann,[5] Mendell and Underhill[6] were as emphatic in claiming that absorption occurs through the intralobular radicles of the hepatic veins.

The following investigation was conducted with the view of throwing some light on this mechanism. Twenty-eight experiments were performed on dogs to determine the relative amounts of bilirubin absorption by the blood and lymphatics of the liver when the biliary system was placed under experimentally controlled back pressures. This was accomplished by intubating the distal end of the common duct with a cannula connected by rubber tubing to a glass reservoir. The entire system was filled with the animal's own bile which was diluted with 2 volumes of physiological saline solution. The bile in each experiment was aspirated from the gall bladder, which was excluded from the biliary tree by ligation of the cystic duct. Each experiment was conducted under a constant and definite pressure (250 to 750 mm of H_2O) by raising the bile-containing reservoir to a measured height above the level of the liver. Quantitative van den Bergh estimations of bilirubin were made on the blood and lymph at 10- to 20-minute intervals during the course of the experiment. Lymph was obtained for bilirubin determination by cannulation of the cervical portion of the thoracic duct which was severed from its entrance into the subclavian vein. The blood was

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1 Bloom W., *Bull. Johns Hop. Hosp.*, 1923, **34**, 316.

2 Harley, U., *Arch. f. Anat. u. Physiol.*, 1893, p. 291.

3 Eppinger, H., *Beitr. z. Path. Anat. u. z. Allg. Path.*, 1902, **31**, 230.

4 Whipple and King, *J. Exp. Med.*, 1911, **13**, 115.

5 Bollman, Sheard, and Mann, *Coll. Pap. Mayo Clin.*, 1926, **18**, 360.

6 Mendell and Underhill, *Am. J. Physiol.*, 1905, **14**, 252.

taken from the femoral vein. The anesthetic used was pentobarbital sodium (nembutal) intravenously, 70 mg per kilo.

The experimentally produced intrabiliary pressures can be classified in 3 groups. (1) 250 mm H_2O pressure or less. In all 5 experiments the hydrostatic pressure was less than the secretory pressure of the liver as reported by Herring and Simpson[7] and subsequent investigators. (2) 300 mm H_2O pressure. In all 6 cases in this group, the intrabiliary pressure approximately equalled the secretory pressure of the liver. (3) 400 mm H_2O pressure or more. Such hydrostatic pressures were always greater than the secretory pressure of the liver.

In the first group, the experiments were done at a pressure of 250 mm of water. The liver secreted against this sub-secretory pressure and added to the reservoir from 5-12 cc of bile. At the end of a 3-hour experimental period, quantitative estimations of bilirubin in the blood and lymph were negative except for an occasional very faint trace of bilirubin in the lymph.

In the second group, the intrabiliary pressure was maintained at 300 mm H_2O. Of the 6 experiments, 3 showed bilirubin in the lymph and none in the blood throughout the duration of the 3-hour period. In the other 3 experiments, bilirubin appeared in the blood as well as in the lymph. However, the bilirubin always appeared first in greater concentration in the lymph and was later detectable in the blood, usually after 15 minutes. (Table I.)

TABLE I.

Bilirubin Concentration in the Blood and Lymph at an Intrabiliary Pressure of 300 mm of Water.

Exp.	Mg bilirubin per 100 cc	Minutes 10	20	30	60	80	100	120	140
A	Blood	.0	.0	.0	.0	.0	.0	.0	.0
	Lymph	.08	.17	.20	.36	.40	.90	1.30	1.60
B	Blood	.12	.12	.15	.38	.41	.40	.38	.40
	Lymph	.20	.30	.58	1.00	.98	1.20	1.50	1.60
C	Blood	.0	.05	.08	.10	.22	.30	.40	.55
	Lymph	.20	.40	.70	1.50	2.30	3.10	3.40	2.90
D	Blood	.0	.0	.0	.0	.0	.20	.30	.30
	Lymph	.0	.20	.30	1.20	1.30	1.50	1.60	1.90
E	Blood	.0	.10	.25	.40	.45	.45	.50	.55
	Lymph	.0	.20	.40	2.00	3.45	4.20	3.60	3.30
F	Blood	.0	.0	.0	.0	.0	.0	.0	.0
	Lymph	.0	.0	.0	.60	1.00	2.20	3.40	—

[7] Herring, P. T., and Simpson, S., *Proc. Roy. Soc. Lond.*, 1907, **79**, 517.

The experiments in the third group were conducted under constant pressures of 400, 500, 600, or 750 mm respectively. The average loss of bile from the reservoir was always more than 8 cc. Under these conditions, bilirubin was present in both the blood and the lymph. It is an interesting fact that while the bilirubinemia resulting from obstructive absorption tended to attain a common level irrespective of the degree of biliary pressure, the concentration of bilirubin in the lymph appeared to vary with the degree of intrabiliary pressure. (Table II.) The mechanism underlying this phenomenon is not yet clear.

Summary. Bilirubin did not appear in the blood or thoracic duct lymph when the intrabiliary pressure was lower than the secretory pressure of the liver. Obstruction of the common duct by raising the intrabiliary pressure above the secretory pressure of the liver resulted in bilirubinemia of approximately constant level, and in the appearance of bilirubin in the lymph in concentrations which increased as the intrabiliary pressure was increased.

TABLE II.
Typical Experiments Showing Bilirubin Concentration in the Blood and Lymph at Various Intrabiliary Pressures.

Pressure, mm of water	Mg bilirubin per 100 cc	Minutes							
		10	20	30	60	80	100	140	180
200	Blood	—	.0	—	.0	.0	.0	.0	—
	Lymph	—	.0	—	.0	.0	.0	.0	—
250	Blood	—	.0	—	—	—	.0	.0	.0
	Lymph	—	.0	—	—	—	.16	.21	.19
300	Blood	.0	.0	.0	.0	.0	.0	.0	—
	Lymph	.08	.17	.20	.36	.40	.90	1.60	—
400	Blood	.0	.50	.70	.56	.44	.52	—	—
	Lymph	.0	.37	.75	1.12	1.00	1.62	—	—
500	Blood	.58	.79	.63	1.40	1.60	—	—	—
	Lymph	1.80	3.00	3.00	3.30	3.30	—	—	—
750	Blood	.58	—	.63	.63	.55	—	.75	1.16
	Lymph	3.00	—	3.50	3.75	3.25	—	3.00	3.25