Effect of Bile Upon Gastric Mucosa.* (27674)

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Superficial gastritis occurs frequently after operations upon the stomach(1,2,3). Although the etiology of postoperative gastritis is unknown, Palmer(1) and Schindler(4) suggest that reflux of intestinal contents into the stomach is the etiological factor. Because this explanation includes several possible causative agents, it is necessary that these be studied individually and in various combinations. The following experiments were undertaken to study the effect of a constant perfusion of concentrated bile upon gastric mucosa.

Methods. Under pentothal and ether anesthesia, a 2 × 3 cm, full-thickness, graft was taken from the greater curvature of the stomach in each of 9 healthy, mongrel dogs. The blood supply of the graft which was derived from the left gastro-epiploic and splenic vessels was carefully preserved. The gallbladder was partially mobilized and a 2 × 3 cm portion of the fundus excised. The gastric graft was brought anterior to the stomach and sutured into the defect of the gallbladder. The suture line was protected by omentum. Eight dogs that survived operation were subsequently sacrificed at intervals of 1, 3, 5, 7, 9, and 12 months after operation. The gallbladders and gastric grafts were examined microscopically.

Results. It was ascertained at time of sacrifice that each graft was viable and its original blood supply was patent. In each dog, the biliary system was also patent and the graft was in contact with an unobstructed flow of concentrated gallbladder bile. The mucosa of each gallbladder and gastric graft appeared grossly normal (Fig. 1).

In all dogs, the microscopic appearance of the gastric mucosal grafts resembled normal gastric tissue (Fig. 2). No distinction could be made in the grafts on the basis of their duration of implantation in the gallbladder wall. The muscularis mucosa showed no evidence of round cell infiltration or fragmentation. The gland layer displayed no atrophy, interstitial fibrosis, glandular dilatation or tortuosity. The foveolar layer from the neck region to the mucosal surface was normal. All signs of necrobiosis of the neck region including degeneration of neck-cells, hyperactive neck-cell regeneration and hyperchromatic nuclei of the columnar lining-cells were absent. The foveolae were not dilated or tortuous and there was no interstitial infiltration. The surface epithelium showed no evidence of ulceration. The cells were columnar and the nuclear position was basal. There was no cellular infiltration of the interstitium.

The microscopic appearance of the gallbladders demonstrated varying degrees of inflammation as manifested by edema, fibrosis and infiltration with neutrophils, lymphocytes, plasma cells and macrophages (Fig. 3). There were Rokitanski-Aschoff Sinuses in the gallbladder removed after one year.

Comment. The findings of Palmer(4) indicate that simple gastroenterostomy is the most likely surgical procedure to lead to postoperative gastritis. He suggests that transstomal regurgitation of jejunal contents is the most important causative factor. Our failure to demonstrate gastritis in segments of stom-

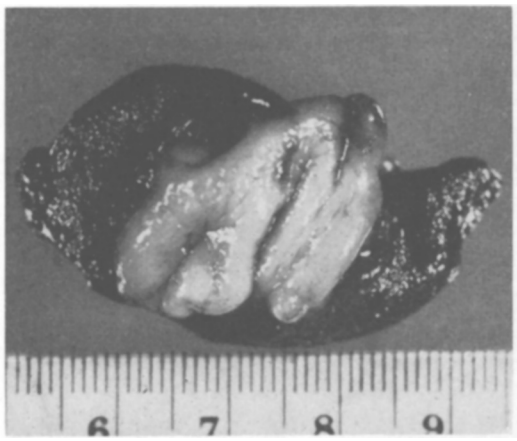

FIG. 1. Gastric mucosa after implantation in gallbladder wall of a dog for 12 mo.

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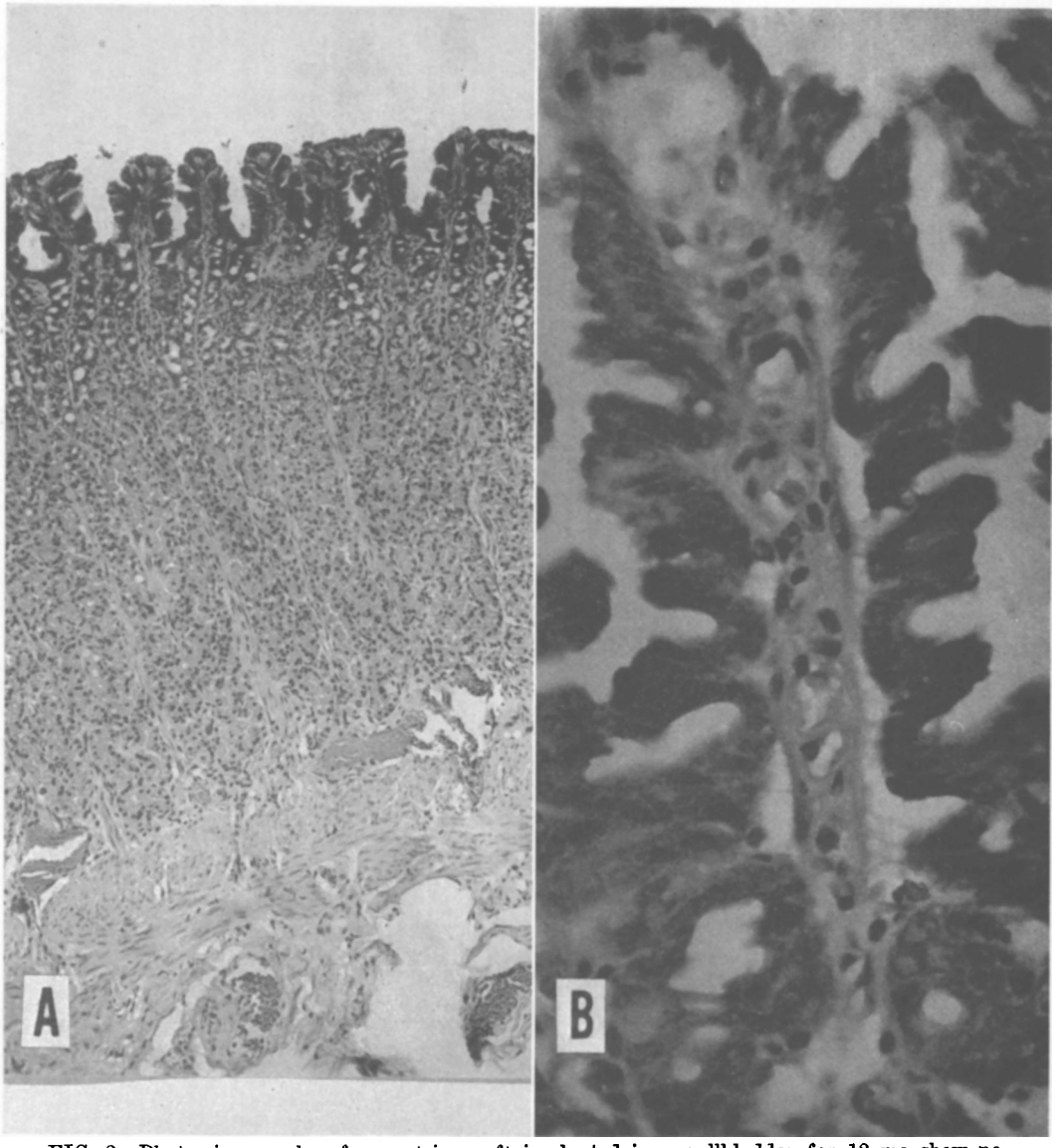

FIG. 2. Photomicrographs of a gastric graft implanted in a gallbladder for 12 mo show no evidence of gastritis (magnification A, ×110; B, ×550).

ach wall implanted into the gallbladders of dogs suggests that pure bile is not the etiological factor in production of superficial gastritis. The possibility that gastritis may result from reflux of other intestinal juices alone or in combination with bile is not excluded by these studies. These alternatives remain to be investigated.

There is some evidence to indicate that addition of vagotomy to gastroenterostomy affords a degree of protection for the gastric mucosa(4). This suggests the possibility that acid may be required to facilitate production of gastritis by trans-stomal regurgitation. Failure to obtain gastritis in our experiments might then be attributed to the lack of insufficient gastric juice in combination with the gallbladder bile. It is therefore of interest that the gallbladder mucosa demonstrated inflammation, for in our opinion this may have resulted from secretion of acid by the grafts.

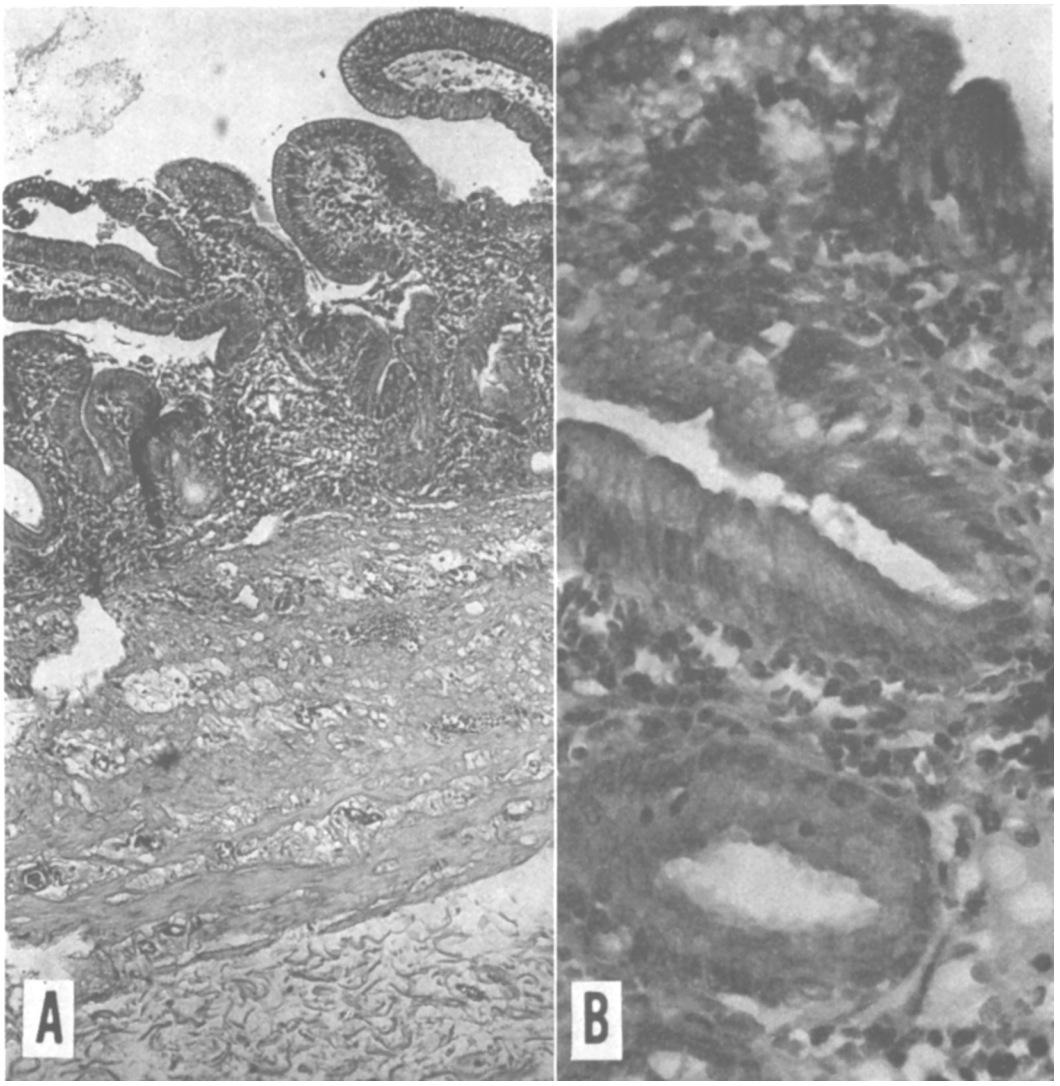

FIG. 3. Photomicrographs of a gallbladder in which a gastric graft was implanted for 12 mo demonstrate presence of chronic inflammation (magnification A, ×110; B, ×400).

Summary. 1. A full-thickness graft of the stomach, with preservation of its vascular supply, was implanted into the fundus of the gallbladder in each of 8 dogs. 2. Gastritis did not develop in any of the grafts studied 1 to 12 months after their preparation. Cholecystitis occurred in all dogs. 3. Perfusion of the gastric mucosa with pure bile does not appear to be a significant factor in production of gastritis.

1. Gray, H. K., Meyers, W. C., Dockerty, M. B., *Surg. Clinic. N. Am.,* 1948, v28, 965.

2. Moersch, H., Waters, W., *SGO,* 1940, v71, 129.

3. Christiansen, T., *Acta Med. Scand.,* 1943, v115, 496.

4. Palmer, E. D., *Gastroenterology,* 1953, v25, 405.

5. Schindler, R., *Gastritis,* New York, 1947.

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