aortic tissue and for the inferior vena cava. A tendency toward lower hexosamine synthesis was observed for the pulmonary artery and coronary artery samples. Assays performed on normal and lipid-arteriosclerotic aortic tissue specimens showed no significant difference between mean enzymatic activities exhibited by these tissue portions.

Discussion. The hexosamine-synthesizing enzyme has recently received increasing attention because this aminotransferase is involved in the synthesis of mucopolysaccharides. No investigations have previously been reported on the presence of this enzyme in human vascular tissue. The finding that one gram of human aortic tissue is capable of synthesizing .6 mg hexosamine in 20 minutes suggests an appreciable activity of this synthetase in the aortic wall. Since it has been suggested by various investigators that the mucopolysaccharides in the arterial wall may be closely connected with the pathogenesis of arteriosclerosis, quantitative biochemical studies of the various enzyme systems in arterial tissue associated with the intermediary metabolism of mucopolysaccharides may prove to be of definite significance in evaluating disease processes.

Summary. Determinations were made of the hexosamine-synthetase activities exhibited by intima-media layers of various types of human blood vessels. A total of 125 vascular specimens was included in the study. The activity measurements were performed by a modification of the procedure described by Bolognani *et al.* The average quantity of hexosamine synthesized in 20 minutes by one gram of thoracic descending aorta was .606 mg; corresponding values for the ascending aorta, abdominal aorta, pulmonary artery, coronary artery, cerebral arteries, and inferior vena cava were, respectively, .720, .726, .450, .450, .372, and .618. No significant difference was observed between the activity of normal and arteriosclerotic tissue from the same arterial specimens.

1. Leloir, L. F., Cardini, C. E., Biochim. Biophys. Acta, 1953, v12, 15.

2. Castellani, A. A., Zambotti, V., Nature, 1956, v178, 313.

3. Pogell, B. M., Gryder, R. M., J. Biol. Chem., 1957, v228, 701.

4. Bolognani, L., Montanari, L., Allieri, L., Zambotti, V., Bol. Soc. Ital. Biol. Sper., 1957, v33, 1777.

5. Schloss, B., Anal. Chem., 1951, v23, 1321.

Received September 2, 1964. P.S.E.B.M., 1965, v118.

Effect of Sham Feeding on Bile Flow in Cholecystectomized Dogs. (29881)

KEITH C. POWELL, LEONARD C. MILLER AND FRANK P. BROOKS
(Introduced by John R. Brobeck)
Departments of Medicine, Surgery and Physiology, University of Pennsylvania School of Medicine, Philadelphia

Insulin hypoglycemia has been shown(1) to produce a choleresis in chronic cholecystectomized dogs when gastric acid secretion was excluded from the duodenum. This action was blocked by a continuous intravenous infusion of an anticholinergic drug and by bilateral vagotomy. Feeding also elicited a choleresis which did not occur after vagotomy. These results were consistent with a vagally-mediated action on the liver. To obtain further evidence for the role of the vagus in choleresis and to detect a "cephalic" phase of bile secretion, bile flow was determined in the conscious cholecystectomized dog after sham feeding.

Method. The studies were carried out on 3 mongrel dogs weighing between 12-17 kg. Each dog was prepared surgically by cholecystectomy, ligation of the lesser pancreatic duct, installation of gastric and duodenal cannulae(2) and a 2-stage esophagostomy(3). The duodenal cannula was so placed that on opening it the ampulla of the common bile duct was visible. The experiments were car-

ried out on dogs that had been fasted for 15-24 hours.

Each experiment was performed with the dog upright in a Pavlov stand, the duodenal cannula open and an olive-tipped plastic catheter in the common bile duct. Bile was collected over a period of 5 hours. A steady flow of bile was maintained by intermittent suction with a 5 ml syringe or by careful attention to the position of the catheter tip.

The bile was collected in 5 one-hour samples in control experiments. In the sham feeding experiments the dogs were fed at the end of the first hour, bile was then collected for 4 hours following commencement of sham feeding. At least 3 control and 3 sham feeding experiments were done on each of the 3 dogs.

To sham feed a dog we gently pulled the posterior mucosal wall of the esophagus through the esophagostomy and kept it well forward by means of 3-6 hemostats. The mucosa was thus draped across the lumen of the lower part of the esophagus and so prevented food from reaching the stomach. Sham feeding was done slowly over 12-15 minutes with 100 g Ken-L-Burger. As the meat passed from the mouth, down to and out the esophagostomy it was collected and refed to the dog. By sham feeding slowly we avoided regurgitation from the esophagus and vomiting, both of which inhibit the flow of bile(4).

In all cases of sham feeding the gastric cannula was opened and the gastric juice aspirated through a Levine tube. This minimized the volume of acid passing into the duodenum and enabled us to assess the gastric acid response to sham feeding, as well as to check the presence or absence of food particles in the stomach. If a trace of food was seen in either the stomach or the duodenum the experiment was omitted from our results.

Volume was determined in graduated test tubes. The bilirubin concentration was estimated by the method of Evelyn and Malloy (5). The bile solids fraction was determined gravimetrically.

Results. A rapid flow of bile usually occurred immediately after catheterizing the common bile duct. This settled to a slow steady flow within 30-45 minutes.

TABLE I. Comparison of Control and Sham Feeding Responses for Bile on 3 Dogs.

Values for 2nd hour after catheterization

	Control	Sham feeding
1. Volume, ml/hr	4.3 ± 2.1 (11)	6.1 ± 1.9 (10)*
2. Bile solids, mg/hr	589 ± 217 (10)	642 ± 252 (10)
3. Bilirubin, mM/hr	7.1 ± 4.0 (10)	6.7 ± 1.5 (10)

Values are means ± S.D.
No. of observations shown in parentheses.
* "p" value $<.05$; $>.01$.

The effects of sham feeding can be seen from Table I. The volume response to sham feeding was barely statistically significant ($p<0.05$; >0.01). Neither the output of bile solids nor bilirubin output approached a statistically significant figure ($p>.05$).

The efficacy of the sham feeding technique is shown by our collecting an average of 52 ml of gastric juice with a concentration of 102 mEq/l of titratable acid in the hour following feeding. In every case of sham feeding we obtained a good gastric response.

Discussion. The results show that sham feeding in 3 chronic dogs produced only a very slight hydrocholeresis. The stimulus of sham feeding was strong enough, however, to bring about a moderate output of gastric juice. We have concluded that the "cephalic phase" in the control of bile flow in dogs is not physiologically important.

Why insulin hypoglycemia causes a choleresis while sham feeding does not is unknown. Both sham feeding and insulin hypoglycemia cause a liberation of gastrin. Since a similar choleresis did not occur with sham feeding, it is reasonable to conclude that gastrin released by insulin hypoglycemia does not account for the choleresis of insulin hypoglycemia.

In a recent study(6) endoradiosondes, sewn into the antral mucosa of the stomach, indicated that insulin hypoglycemia and sham feeding gave different results in respect to the motility patterns of the fasting stomach. Olbe and Jacobson found that the fasting type II wave activity of the stomach was completely abolished for 1½ hours by sham

feeding, whereas insulin hypoglycemia increased the frequency and amplitude of such waves.

It is possible that insulin hypoglycemia is able to liberate pancreozymin and/or secretin from the antrum (or intestinal mucosa) in a way that sham feeding cannot. However, it is also known that antral motility is not essential for release of gastrin(7), and hence the differences in antral motility that occur with insulin hypoglycemia and sham feeding may be irrelevant.

It is of interest to note that in both humans(8) and dogs(9) insulin hypoglycemia has been shown to cause an increase in blood flow to the liver. Tanturi and Ivy(10) published studies on the effect of blood flow to the liver on bile flow. They concluded that in acute experiments an increased blood flow through the liver augmented bile output except when increased blood flow was associated with an increased intrahepatic vascular pressure. They acknowledged that the evidence was inconclusive. We have no knowledge of what the effect of sham feeding is on blood flow to the liver, and we do not know if the blood flow changes that occur with insulin hypoglycemia are relevant to the associated choleresis. Brauer(11) argues that bile flow rates are independent of blood flow or pressure when the oxygen supply is not a limiting factor.

Summary. In contrast to the true choleretic effect of insulin hypoglycemia and feeding in chronic cholecystectomized dogs, sham feeding produced only a slight hydrocholeresis. An increase in gastric secretion in the same dogs indicated effective vagal stimulation. These results add another difference between sham feeding and insulin-hypoglycemia in their effects on the digestive tract.

1. Fritz, M. E., Brooks, F. P., Am. J. Physiol., 1963, v204, 825.
2. Thomas, J. E., Proc. Soc. Exp. Biol. and Med., 1941, v46, 260.
3. Komarov, S. A., Marks, I. N., ibid., 1958, v97, 574.
4. Kocour, E. J., Ivy, A. C., Am. J. Physiol., 1938, v122, 325.
5. Malloy, H. T., Evelyn, K. A., J. Biol. Chem., 1938, v122, 597.
6. Olbe, L., Jacobson, B., Gastroenterology, 1963, v44, 787.
7. Thein, M. P., Schofield, B., J. Physiol., 1959, v148, 291.
8. Bearn, A. G., Billing, B. H., Sherlock, S., Clin. Sci., 1952, v11, 151.
9. Shoemaker, W., Mahler, R., Ashmore, J., Pugh, D., Am. J. Physiol., 1959, v196, 1250.
10. Tanturi, C. A., Ivy, A. C., ibid., 1938, v121, 61.
11. Brauer, R. W., J. Am. Med. Assn., 1959, v169, 1462.

Received September 2, 1964. P.S.E.B.M., 1965, v118.

The Antilactobacillus System of Saliva. Role of Salivary Peroxidase.* (29882)

S. J. KLEBANOFF† AND R. G. LUEBKE‡
USPHS Hospital, Department of Medicine and School of Dentistry, University of Washington, Seattle

More than 70 years ago, W. D. Miller proposed the chemico-parasitic theory of caries production(1). According to this concept, caries is initiated by decalcification of the surface of the tooth by acid and the source of the acid is the fermentation of carbohydrates by oral micro-organisms. Other processes, *e.g.*, proteolysis and chelation, also have been implicated in the pathogenesis of caries (2). However, it is still widely held that

* Supported in part by USPHS grant GM-10310 from Nat. Inst. Health.

† Research Career Development Awardee of Nat. Inst. Health (NIAMD).

‡ Supported by Training Grant 5T1 71-03 from Nat. Inst. Dental Research. Present address: College of Dentistry, Univ. of Kentucky Med. Center, Lexington.