Liver that had been autoclaved at a pressure of 15 pounds for 15 minutes was sterile. Pieces of such liver were placed into the peritoneal cavity of other animals without causing death. Some of such autoclaved liver was reinfected by organisms isolated in pure culture from fresh liver. Such a piece free in the peritoneal cavity or abdominal wall caused the death of the animals within 24 hours.

Foetal liver, secured aseptically from ceserean section pups was proven sterile by culture. When placed into the peritoneal cavity of other animals it was autolyzed completely without injury to the animals.

Conclusions. (1) The uncontaminated liver of normal healthy adult dogs regularly contains anaerobic bacteria. (2) It is probable that the experimental so-called "*In Vivo* aseptic autolysis of the liver," is always complicated by this infection, and that this is the cause of death. (3) *In vivo,* aseptic autolysis of fetal liver (proved sterile by culture) does not produce toxic effect. (4) Autolytic enzymes of liver may be destroyed by heating, but such pieces of liver cause the death of an animal in 24 to 36 hours. An anaerobic, spore-bearing bacterium may be recovered from the liver at autopsy. (5) Autoclaved liver is sterile, and causes no harm to an animal if placed into the peritoneal cavity. If autoclaved liver is re-infected with the anaerobic organisms before being placed into the peritoneal cavity, or abdominal wall, death results within 24 hours.

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Fatal Effect of Total Loss of Gastric Juice.*

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The present experiments were undertaken to determine the effect of the total loss of gastric juice from the body under conditions not complicated by obstruction such as are present after closure of the pylorus. Recourse was made to a method described by Lim, Ivy, and McCarthy[1] with this important modification that the vagus nerves to the isolated stomach were left intact. The stomach was cut across at the cardia and the pylorus. The cardiac end of the

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[1] Lim, Ivy, and McCarthy, *Quar. J. Exp. Physiol.*, 1925, xv, 13.

stomach was then inverted and closed and the open pyloric end brought to the surface as a fistula. An anastomosis was made between the lower end of the esophagus and the duodenum. Great care was taken in sectioning the cardia not to interfere with the vagus fibers to the stomach and as much of the blood supply to the isolated stomach was left as was technically possible. To date we have made 3 such preparations but the data to be presented here are from a single animal. At autopsy the vagus nerves to the isolated stomach in this animal were found intact. A daily record was kept of the water and food intake (food exclusively milk and muscle), the urinary output, and the quantity and composition of the gastric secretion collected from the isolated stomach. In addition blood was drawn at the end of each 24 hr. period and determinations made of the CO_2 combining power of the blood plasma, the blood pH, and the NPN, urea N, and chloride concentration.

The following points have been established: (1) The total loss of the gastric secretion from the body causes death in the dog with symptoms of weakness, anorexia, oliguria and profound depression. (2) Accompanying these symptoms and proportionate to their severity occurs a prompt fall in blood chloride, an increase in the CO_2 combining power of the plasma, an increase in the pH, and a late increase in the NPN and urea N. (3) The symptoms may be relieved and the blood chemistry returned toward the normal by the intravenous injection of from 2,000 to 3,000 cc. of Ringer's solution or 0.9% NaCl solution per 24 hrs. (4) The isolated stomach, with intact blood and vagal supply, will frequently secrete in 24 hrs. from 1000 to 1600 cc. of gastric juice with a chloride concentration of 0.5 to 0.55%. There is thus excreted by the gastric mucosa in 24 hours more than twice the calculated amount of chloride in the circulating blood. It is this loss of chloride which is chiefly responsible for the death of the animal. (5) The gastric mucosa can continue to secrete gastric juice of high free and total acidity (0.312% and 0.363% respectively) even when the blood chloride has been reduced to less than a third of its normal concentration (108 mg. of Cl per 100 cc.) and an extreme alkalosis exists (Plasma CO_2 104). Under these conditions, however, the volume of secretion is lessened. (300 cc. per 24 hrs.). It should be mentioned that this is continuous secretion since the animal is depressed and refuses all food. The ordinary stimuli for secretion are thus absent. (6) When as a result of alkalosis, hypo-chloremia, and absence of food taking the volume of juice is depressed, the intravenous injection of Ringer's solution causes a pronounced increase in secretion (from 300 cc. per 24 hrs. to 900 cc. and this without the stimulus of food).

(7) When the animal is in severe hypo-chloremia and considerably dehydrated, the secretion of urine being greatly lessened, it apparently does not experience thirst for it drinks but little water. If then it is given a 1000 cc. of Ringer's solution intravenously, it promptly becomes thirsty and drinks large quantities of water. (8) The body temperature remains normal throughout the periods of dehydration. (9) Evidence of tetany has been uniformly absent except that occasional slight twitchings of the legs have been observed during the intravenous injection of Ringer's solution. For the most part the deviation from the normal has been in the direction of depression rather than excitation.

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Action of Iodized Oils on Serous Membranes.

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Iodized oils (Lipiodol "Lafay", and Lipoiodin "Ciba") have been injected into the pericardium, pleura, peritoneum, and joint cavities in an endeavor to study the pathogenicity of these substances. Dogs were used throughout. Ten animals were injected intrapericardially with 15 to 20 cc. iodized oil; in each case the dog developed a sterile pericarditis with marked effusion. Death was in most cases due to a pneumonia which was probably secondary to an embarrassed circulation caused by the pericardial effusion. The pericardium was covered with a fibrinous exudate resembling that of the "shaggy heart" of pneumonia; there was thickening of the pericardium and it appeared congested. A few adhesions were commonly present, especially about the auricles. The exudate consisted of from 90 to 300 cc. of creamy fluid, which on microscopic examination showed oil droplets, fibrin, and leucocytes (chiefly lymphocytes). Direct smears of the effusion were always negative for bacteria, as were cultures. Microscopic examination of frozen sections stained with Sudan III and of ordinary hematoxylin and eosin preparations showed the fibrin layer covering the inner surface of the pericardium to be loaded with leucocytes and oil droplets. The pericardium itself contained many leucocytes and a few red blood cells. The first few layers of muscle fibers under the pericardium had undergone fatty degeneration.