

chick. These conditions would appear to be due to one of three factors:

(1) An *inhibition* of the organisms owing to an unfavorable environment or to antagonistic action of other organisms;

(2) The *destruction* of the bacteria due to (a) the influence of an abnormally high temperature on these soil organisms; (b) the chemical activities in the body of the chick; (c) the chemical activities of other organisms.

(3) The *transformation* of the citrate-using bacteria into the non-citrate using type in the intestinal tract of the chick.

Of these factors, the temporary inhibition of the organisms appears to be the least probable; a month after inoculation of a known mixture of the two types into citrate medium, both are found viable when transferred to eosin-methylene-blue plates, thus eliminating any shorter time factor; yet when similar transfers are made from negative citrate tubes, no growths of citrate users develop. Investigation of these separate points is now in progress in the hope of obtaining more definite information concerning the fate of the citrate-using bacteria in the intestinal tracts of young chicks.

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Chronic ulcerations in the dog's stomach produced by x-ray.

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Progress in the study of gastric ulcer has been decidedly delayed by the fact that it has been impossible to produce, experimentally, chronic ulcerative lesions in the stomach of the lower animals, notably in the dog. Innumerable attempts have been made in the past twenty or more years, and, with one possible exception, they have all been failures. In certain instances, Dragstedt was able to produce a chronic ulcer by injecting 4 per cent silver nitrate solution beneath the mucosa, and then stitching in and out through this area, using non-absorbable material.

In about 60 per cent of cases, a chronic lesion was produced, but the procedure was by no means dependable, and added the element of a foreign body imbedded in the tissues.

In October, 1923, I began an experimental study of gastric ulcer. Accepting the suggestion given me by Dragstedt, I began a series of experiments to determine whether the exposure of the mucous membrane of the dog's stomach to X-ray would produce a destructive lesion that would be slow in healing. Our opinion was that, since X-ray burns of the skin were slow in healing, often resisting for months all forms of treatment, if such a lesion could be produced in the wall of the dog's stomach, it might be considered a pathological and clinical counterpart of chronic ulcer in the human. This may be questioned, at least until microscopic evidence will indicate whether these experimentally produced ulcers are histologically similar to human chronic ulcers.

After a number of failures due to insufficient exposure, it was finally discovered that an ulceration which runs a definite chronic course, can be produced in the dog's stomach by the X-ray.

The following technique and protocols are submitted as a preliminary report:

The dog's stomach is delivered as far as possible through an abdominal incision. An incision over the pre-pyloric antrum is made through the anterior wall, parallel to the long axis of the stomach. Bleeding vessels are ligated if necessary. The posterior wall of the stomach is everted through the incision of the anterior wall, and four sutures are placed in the posterior wall so as to represent the four angles of a square. These sutures are threaded through a lead plate which has a foramen about 3 cm. in diameter. When these sutures are tied, an area of the posterior wall is fixed in the foramen of the lead plate, and is then exposed to the rays. Occasionally it will be necessary to place a few extra sutures, fixing the posterior to the anterior wall, to prevent leakage of gastric contents. An exposure of 150 milli-ampere minutes, 140 KWV, unfiltered, at a distance of nine inches is given. The fixation sutures are then cut, and the wall of the stomach is closed by two layers of sutures.

In a relatively small number of dogs which have been examined, this method has not failed in producing a chronic ulcer.

PROTOCOLS.

Dog 7. Exposure 3-20-24. On 5-28-24, a definite chronic ulcer, 14 mm. long, 6 mm. wide and 2 mm. deep, present. A scar on the posterior serous coat. On 2-26-25, ulcer still present, practically same size, edges more indurated. Animal still alive.

Dog 8. Exposure 5-11-24. On 3-12-25, a definite chronic ulcer present. An area about $2\frac{1}{2}$ cm. in diameter, indurated and roughened. In the center of this area, a fissure 8 mm. in length, 3 mm. in width, and about 3 mm. in depth. No scar on the posterior serous coat. Animal still alive.

Dog 9. Exposure 6-13-24. On 3-5-25, a chronic pear-shaped ulcer, 1 cm. in diameter and 2 mm. deep, present. Floor of the ulcer granulating. Dog still alive.

Dog 15. Exposure 2-24-25. On 5-15-25, an ulcer 4 mm. long, 2 mm. wide, oval in shape, punched out appearance. Dirty grey floor, edges indurated, surrounded by a hyperæmic area. Dog still alive.

Dog 12. Exposure 3-17-25. On 5-19-25, died of a generalized peritonitis due to a gastric perforation. Perforation about 6 mm. in diameter, edges thickened. Microscopic examination of the margin of the perforation shows but little sign of inflammatory reaction. Most of the necrotic tissue digested away.

Proper anesthesia was used in all cases.

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Analogous action of insulin and epinephrin on the liver.

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Insulin and epinephrin are frequently considered as antagonistic because hypoglykemia and hyperglykemia follow after the respective administration of these substances.

Since we know the mechanism of insulin action,¹ the alleged

¹ E. F. Müller, H. E. Wiener, and R. vE. Wiener, *Proc. Soc. Exp. Biol. and Med.*, 1925, **xxii**, 375.