TABLE I.
Dog M38. Female, Pavlov pouch, weight 11.0 kilos.

Time	Urine		Gastric Juice		Remarks
	cc.	pH	cc.	Free acid (clinical units)	
9:05	115	5.95			Residual urine
10:05	5	5.96	4.8	23	Control period
(At 10:05, gave 1 mg. of histamine dichloride subcutaneously and 100 cc. water by stomach tube)					
11:05	4	6.95	37.0	115	
12:05	3	7.16	3.5	68	
1:05	4	5.99	5.2	0	
(At 1:05, gave 1 mg. histamine dichloride)					
2:05	5	7.82	39.0	115	
3:05	7	8.02	7.0	102	
4:05	3	5.90	3.5	0	

We believe that our experiments furnish additional evidence that the alkaline tide is due to the formation of HCl in the stomach. While we have found it always present in normal dogs, it seems less pronounced than in the pouch animals, perhaps because of the loss of acid from the fistulae in the latter.

Hiller[3] has reported that histamine lowers plasma pH while raising that of the urine. These observations would seem to indicate that the change in urinary pH after histamine is not brought about by any excess of base, and is perhaps unrelated to gastric secretion. We have confirmed her findings on blood pH following large doses of histamine (1 mg. per kilo or more) but not with such doses as were used in our urine studies (0.06 to 0.08 mg. per kilo). There is following the small doses a rise in blood pH, of 0.02 to 0.06, lasting for 1 to 2 hours.

[1] Ackman, F. D., *Canad. Med. Assn. J.*, 1925, xv, 1099.
[2] Hastings, A. B., and Sendroy, J., *J. Biol. Chem.*, 1924, lxi, 696.
[3] Hiller, Alma, *J. Biol. Chem.*, 1926, lxviii, 833.

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The Regeneration of Acid-Fastness by Animal Passage.

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By following up the regeneration of tubercle bacilli reported in a study of the granules of this organism,[1] several strains of non-acid-fast or partially acid-fast organisms have been studied. One or-

ganism or series of organisms obtained from the organs of a patient dying of a generalized miliary tuberculosis will be reported with special emphasis on the ability of the organism to regain acid-fastness on animal passage.

The peculiar aspect of the case was that in the numerous miliary tubercules there were very few acid-fast organisms found. After a search for many hours none were found in the spleen. Instead, however, the miliary tubercles and giant cells contained coccoid bodies (non-acid-fast) arranged singly and in pairs. These possessed a refractile nature not found in ordinary cocci. These cocci, although quite numerous, did not produce colonies on agar media, and sodium hydroxide treatment apparently destroyed their ability to produce infection in guinea pigs.

Pieces of the spleen were placed in celloidin capsules and embedded in the body cavities of guinea pigs. In 3 weeks numerous acid-fast bacilli were found in the capsules. One animal (T93) on which the capsule had broken, had a generalized tuberculosis. The spleen of this animal was inoculated into another animal (T38) that died in 12 days of a septicemia-like condition. The spleen was enlarged many times and red. The lungs were pneumonic, the liver was fatty and acutely inflamed. Numerous coccoid and bacillary organisms were observed as well as organisms that seemed to have a slight acid-fastness. The spleen was macerated, diluted and filtered and a single bacillus colony grown from a picked organism. This organism, a pleomorphic type, that possessed no constant morphology nor cultural characteristics, was inoculated into 2 guinea pigs. One (T78) received about 2 mg. of the organism and died in the same manner at T38 with similar pathology; the other (T82) received about 0.2 mg. and lived to develop a peculiar exudative type of disease resembling tuberculosis, with a few faintly acid-fast organisms in the hilus lymph glands. At this point it was observed that large doses administered in series to other animals produced a rapidly fatal septicemic disease in no way resembling tuberculosis. Small regulated doses produced in the third passage a typical guinea pig tuberculosis.

This process was repeated several more times—once with a single cell strain of a diphtheroid like organism—and the same general result was obtained. The final organism grown on a new special medium resembled the human type tubercle with the exception that it was not quite as virulent for rabbits as a typical human strain.

It is not *B. tuberculosis rodentinum* nor *B. pseudo-tuberculosis.*

According to the postmortem findings in more than 10 patients dying at this institution, a non-acid-fast form of organism has

played the dominant rôle until late in the disease. This is particularly true of the pneumonic phase of the primary complex in infants. Furthermore, it is thought that a great many of the associated conditions in tuberculosis may be attributed to these rapid growing forms that seem to emanate from the tubercle-forming organism in environments not suited for a development of the slowly growing waxy form.

[1] Sweany, H. C., *Amer. Rev. Tuber.*, 1928, xvii, 53.

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Nutritional Edema and Its Relation to the Incidence of Common Colds.

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In the course of 20 years of personal experimentation with fasting and various diets, edema was frequently manifested. It often occurred after periods of undernutrition but was most prominent after prolonged fasting. Edema has also been observed in others after fasting. It is apparently similar to the starvation edema *(Hungerödem)* which developed among the undernourished masses of Europe during the late war. The European studies of this "war edema" made it clear that nutritional factors and not impairment of the circulation or of kidney function were responsible. But the detailed analysis of the nutritional factors was complicated by the fact that the dietary of the afflicted individuals was not only insufficient in quantity but was also inadequate in other respects. Thus, some investigators were led to attribute the edema mainly to an excessive salt and vegetable intake while others considered it a consequence of deficiency in vitamins or fat.

The edema observed in the subject of the present study occurred independent of some of the factors which complicated the European studies. Hence, it is possible to say definitely that vitamin deficiency, fat starvation or an excessive salt intake were not fundamental factors in the development of this edema. Instead, the observations in this study indicate that protein starvation is the primary factor in giving rise to this type of hydration. The finding of Kohman[1] is hereby supported. However, the gross manifestation of nutritional edema seems to be possible only when the diet con-