TABLE I.
The Production of Polycythemia by Cobalt in the 3 Dogs Whose Blood Volume Changes are Shown in Fig. 1. Erythrocyte Numbers Are Given in Millions.

	Dog No. 13	Dog No. 14	Dog No. 15
Before Cobalt	5.17	5.78	5.07
	5.26	5.81	5.16
	5.06	5.75	5.28
			5.09
After Cobalt	5.93	6.75	6.29
	6.07	6.84	6.32
	6.22	6.72	6.25
	6.13	6.77	6.30

I, which presents erythrocyte numbers observed before and after the production of cobalt polycythemia. The increases are not great but are significant, ranging from 17 to 22%.

These experiments on dogs confirm, in a qualitative way, the work of Orten, Underhill, Mugrage, and Lewis,[2] which was done on rats.

Conclusion. Cobalt polycythemia in the dog is accompanied by an increase of blood volume which is due chiefly to an increased volume of cells.

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Adenomatous Stomach Lesion of the Rat Associated with Heavy *Cysticercus fasciolaris* Infestation.*

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In connection with an extensive compilation of their data on spontaneous tumors of the rat, Bullock and Curtis[1] recorded the interesting observation that a hypertrophic gastritis was frequently found in rats infested with large numbers of *Cysticercus fasciolaris,* the larval form of the cat tapeworm, *Taenia taeniaeformis (crassicollis).* Following the ingestion of the *Taenia* eggs by the rat, the oncospheres localize in the liver, where they lead to the formation of cysts from which the well-known *Cysticercus* sarcomas often develop.[2] The gastritis seems to be definitely associated with the

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1 Bullock, F. D., and Curtis, M. R., *J. Cancer Res.*, 1930, **14**, 1.

2 Bullock, F. D., and Curtis, M. R., *Proc. New York Path. Soc.*, n.s., 1920, **20**, 149.

heavy infestations produced by massive feedings of *Taenia* eggs.[3] During the course of experiments on feeding *Taenia taeniaeformis* eggs, we also have observed thickened and enlarged stomachs in the rats. In view of the scarcity of experimental approaches to research on tumors of the glandular stomach,[4] it is desirable to call attention to the adenomatous character of the gastric lesions that have been produced in our animals.

Infestations were carried out by feeding portions of terminal proglottids of mature worms or by feeding small pieces of bread on which counted suspensions of eggs had been dropped. Grossly thickened stomachs were observed in all of 7 rats, 3 males and 4 females, which were sacrificed after a heavy *Cysticercus* infestation of 235 to 272 days' duration. In these animals the enlarged livers contained 150 to 200 cysts, displacing most of the liver tissue. An eighth rat, with a less pronounced lesion, had an infestation of 61 cysts for 303 days. These animals included piebald rats of the McCollum strain and albinos of the Wistar and Buffalo strains, all of which were maintained on the McCollum stock diet without supplements. Different animals of the group had been infested at 3 different times by means of *Taenia* worms secured from 3 different cats.

By contrast, in a group of 9 rats of the same strains that were lightly infested, with 5 to 33 cysts of 239 to 330 days' duration, no significant changes were present in the stomachs. The tenth rat of the group had a practically normal stomach, although it had an infestation of 63 liver cysts for 295 days. Likewise, the stomachs of a non-infested group of control rats were normal. In neither the heavily nor lightly infested animals had there yet developed any gross evidence of *Cysticercus* sarcomas at the time of autopsy.

The thickened stomachs varied in size from only slightly larger than normal up to more than twice the normal size. When the animals were sacrificed, the forestomach was usually found contracted and containing little food. The glandular stomach was always practically empty or covered with a thick coating of mucus. As a result of the pronounced thickening, the lumen of the stomach was considerably narrowed, usually leaving scant space for food.

Grossly the lesion appeared to be confined to the glandular part of the stomach; the forestomach was essentially normal. The mucosal surface of the stomach appeared rough, with an opaque pink or white color. Sometimes small superficial hemorrhagic areas were seen. The

[3] Curtis, M. R., and Dunning, W. F., personal communication.

[4] Klein, A. J., and Palmer, W. L., *Arch. Path.*, 1940, **29**, 814.

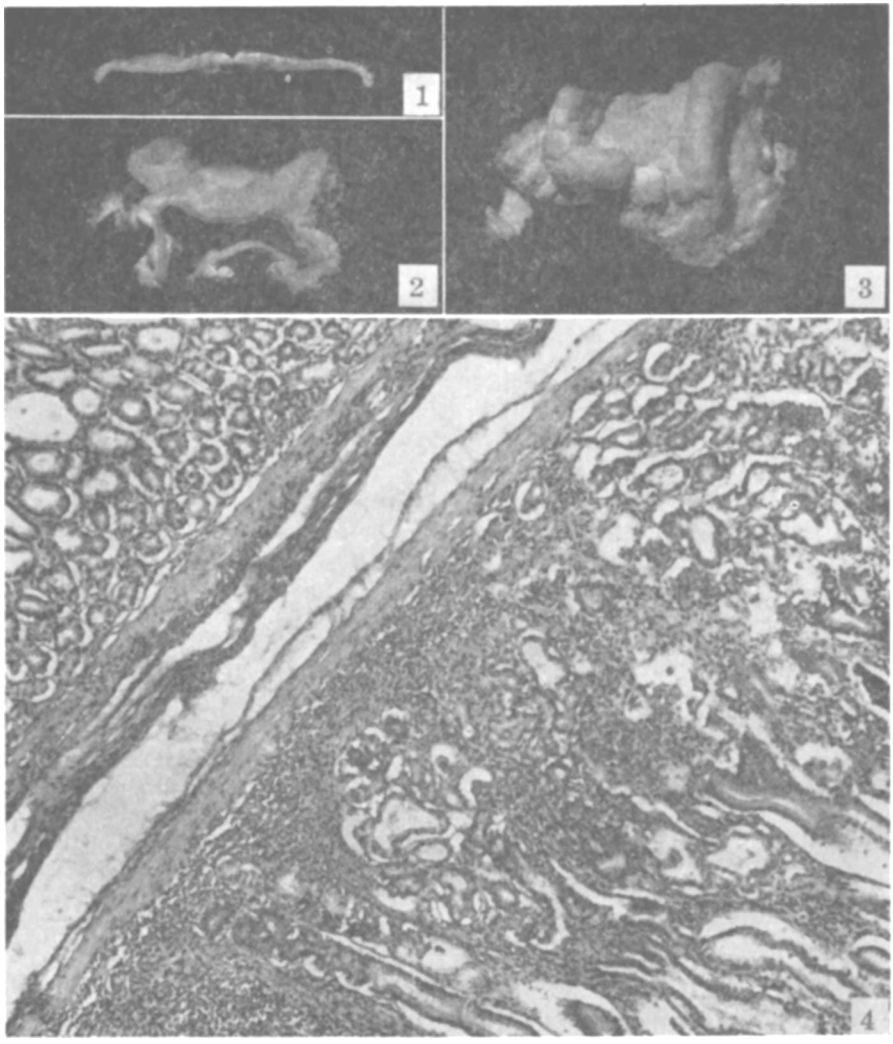

FIG. 1. Cross section of normal glandular stomach of rat. (Actual size.)

FIG. 2. Cross section of adenomatous glandular stomach of rat bearing heavy liver infestation of *Cysticercus*. The stomach has been turned almost inside out to show the size of the 2 large papillary overgrowths, which projected into the lumen. (Actual size.)

FIG. 3. Top view of sectioned stomach shown in Fig. 2. Note the 2 long ridges. (Actual size.)

FIG. 4. Photomicrograph taken near base of right ridge of stomach shown in Fig. 2. Note connective tissue stalk, inflammatory changes in stroma, and abnormal glands. ($\times$ 75.)

thickening was in some cases almost general, while in others there were in addition papillary extensions of as much as 7 mm beyond the

normal stomach thickness (Fig. 1, 2). The most pronounced part of the overgrowth was in various sites, such as at the border of the forestomach, as symmetrical longitudinal ridges (Fig. 3), or in one case as an enlarged ring around the pylorus.

Microscopically the lesion appeared adenomatous. There was glandular hypertrophy, sometimes arising from large connective tissue stalks (Fig. 4). Many glands appeared abnormal, showing hyperplasia, metaplasia, and necrosis. The stroma showed inflammatory changes in which an extensive eosinophilia was sometimes noted. Enlarged acini were frequently present.

Although the presence of some unrecognized factor, such as another concomitant infection, can not be ruled out, the consistent association of the heavy liver infestation and the adenomatous stomach lesion would seem to indicate that the *Cysticercus* infestation itself is responsible for the adenomatous stomach changes. No gross local parasitic infection has as yet been observed in the routine inspection of the stomachs, or after incubation of the stomachs for one hour at 37°C in physiological saline solutions. The absence of the lesion in lightly infested rats, or in control animals maintained in the same cage with heavily infested rats, is evidence against a contagious infection as the etiological factor. Portions of thickened stomach are now being fed to young rats in attempts to transmit any local infectious agent present.

Andervont and Stewart[5] have reported a spontaneous adenomatous lesion of the stomach in Strain I mice, in which susceptibility to the lesion is inherited. In the case of the adenomatous rat lesion, its production in a piebald strain and in two albino strains apparently eliminates hereditary susceptibility as a major factor in the etiology of the lesion associated with *Cysticercus* infestation.

Bonne and Sandground[6] have reported the experimental production of adenopapillomatous growths in the Javanese monkey by means of *Nochtia nochti*, a nematode living in the stomach. In the case of the *Cysticercus* stomach lesion, it must be noted that the infestation is apparently confined to the liver, thus seeming to eliminate local tissue irritation by the larval worm as a cause of the proliferation. Since the development of the lesion appears to depend upon the presence of a large number of liver cysts, it is likely that the adenomatous change is brought about at a distance from the infestation through the medium of some toxic metabolic product elaborated by the encysted worms. It is also possible that some nutritional alteration takes place as a result of the displacement of

[5] Andervont, H. B., and Stewart, H. L., *Science*, 1937, **86**, 566.

[6] Bonne, C., and Sandground, J. H., *Am. J. Cancer*, 1939, **37**, 173.

the major part of the liver tissue by the cysts. It is interesting that the same organism that often leads to the formation of liver sarcomas in older animals is also associated with the adenomatous gastric lesion which has been described.

Further studies are now in progress on the effect of dietary modifications on the development and possible prevention of the lesion.

Summary. The production of adenomatous stomach lesions of the rat, associated with heavy liver infestations of *Cysticercus fasciolaris,* is reported. The adenomatous stomach lesions have been produced in 3 different strains of rats.

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A Virus Pneumonia of Syrian Hamsters.*

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In the course of attempts to infect Syrian hamsters (*Cricetus auratus*) 2 to 4 months old, with influenza A, virus strain W. S.,[1] another filterable agent capable of producing a fatal pneumonia in these animals was found. One hamster was inoculated intranasally with mouse-passage influenzal virus (W.S.) and developed slight nasal symptoms. This animal was sacrificed after 8 days and suspensions of lung and turbinate were passed to a second hamster which had large plum-colored pneumonic areas in its lungs when killed at 6 days. Subsequently suspensions of lung and turbinate have been carried through 17 serial intranasal passages in hamsters with the production of pneumonia in each passage except the first and third. The suspensions at each hamster-passage when inoculated intranasally into mice produced a pneumonia which was usually fatal within 6 to 9 days. Attempts to produce a similar pneumonia in hamsters with several other mouse-passage strains of influenzal virus were unsuccessful. We were also unable to repeat the original experiment starting with another mouse-passage of the strain W.S.

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1 Taylor, R. M., PROC. SOC. EXP. BIOL. AND MED., 1940, **43**, 541.