of Baudrimont and St. Ange[7] and Dareste[8–10] who found that obliteration of the respiratory air space by varnishing the large end of the shell led to asphyxial death of the embryo. Varnishing over the small end did not affect development in any way.

The results of oiling the shell membrane are not in agreement with Kuo's reports that chicks develop normally when the inner shell membrane is vaselined. It appears that such a procedure likewise engenders anoxemia. This fact, considered in the light of recent studies on behavioral development in bird and mammalian embryos,[11–12] may explain why Kuo frequenty encountered prehatching respiratory activity and somatic "mass motility" in his specimens.[2–5] Even in the normal, undisturbed, incubating egg an adequate gaseous exchange between the chick and the outside atmosphere is maintained with difficulty.[13] Vaselining the inner shell membrane in the region of the air space, designated by some as the "avian placenta," leads to abnormal, asphyxial behavior patterns.

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Pathogenicity of "Carrier" Strains of *Endamoeba histolytica* in the Experimental Dog.

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For many years it has been recognized that individuals may harbor *Endamoeba histolytica* without exhibiting clinical symptoms. Such individuals have often been termed healthy carriers. It has been suggested that this organism may not always invade tissue but that it may live in the large bowel as a commensal. Brumpt[1] holds this belief and consequently divides the organism into 2 separate species according to its pathogenicity or non-pathogenicity

7 Baudrimont, A., and St. Ange, M., *Ann. Chim.* (Phys.), 1847, Series 3, **21**, 195.

8 Dareste, M. C., *C. R. Acad. Sci.*, Paris, 1855, **41**, 963.

9 Dareste, M. C., *Ann. Sci. Nat.*, 1855, Series 3 (Zool.), **4**, 119.

10 Dareste, M. C., *C. R. Soc. Biol.*, 1857, **9**, 99.

11 Windle, W. F., and Barcroft, J., *Am. J. Physiol.*, 1938, **121**, 684.

12 Windle, W. F., and Becker, R. F., *Arch. Neurol. and Psychiat.*, 1940, **43**, 90.

13 Romijn, C., and Roos, J., *J. Physiol.*, 1938, **94**, 365.

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1 Brumpt, E., *Trans. Roy. Soc., Trop. Med. and Hyg.*, 1928, **22**, 101.

for experimental kittens. Craig[2] is one of the strongest advocates of the belief that all strains are actually or at least potentially pathogenic to man. He has repeatedly stated that *E. histolytica* is essentially a tissue parasite and that it cannot live and propagate for any length of time in the human intestine without producing some tissue damage.

The present investigation was designed to determine experimentally the pathogenicity of human "carrier" strains of *Endamoeba histolytica.* By inoculating dogs with cysts or trophozoites of *E. histolytica* obtained from the feces of human "carriers," it was expected that evidence could be obtained with reference to the percentage of animals which became infected, the incubation period, the clinical manifestations of the infection and the amount of pathology produced by the amebae.

The amebic strains selected for experimentation were obtained from a group of orphan children who had never shown symptoms of diarrhea or dysentery. They were thus placed in the so-called "healthy carrier" group of individuals harboring *E. histolytica.* Full-grown dogs were utilized as experimental animals, since the course of the disease in them closely simulates that in man. The dogs were either inoculated intragastrically with cysts or intracecally with trophozoites, which had been isolated in culture.

Out of 26 dogs inoculated with 16 different "carrier" strains of *E. histolytica* all became infected with the organism and all showed amebic lesions at autopsy. This 100% infection is remarkably high when one remembers that these strains are considered by many workers to be non-pathogenic. Walker and Sellards[3] found in their human experiments that the incubation periods varied from 1 to 33 days with an average of 11 days. In the present series of dog experiments the incubation periods varied from 2 to 21 days, with an average of 6 days.

On the basis of objective symptoms, presence of amebae and extent and numbers of lesions, the animals were divided into 4 groups: (1) Acute dysentery, with numerous amebae and extensive lesions (4 dogs); (2) mild, evanescent dysentery, with amebae and lesions (6 dogs); (3) no dysentery, with amebae and lesions (3 dogs), and (4) no dysentery, with no detectable amebae but with lesions (13 dogs).

Three types of gross lesions were observed in the 26 experimentally infected dogs: (1) Extensive superficial denudation of

2 Craig, C. F., *Amebiasis and Amebic Dysentery,* 1934.

3 Walker, E. L., and Sellards, A. W., *Phil. J. Sci.,* 1913, **8**, 253.

the mucosa; (2) ragged, round and slit-like ulcers; and (3) typical pin-point lesions.

From the above results it is observed that all of the 16 different "carrier" strains of *E. histolytica* were pathogenic to dogs. Every animal became infected and showed amebic lesions at autopsy. The severity of the infections was different for the various strains and for the individual experimental animal but all strains were shown to be pathogenic and in many cases very active tissue invaders. The gross lesions were very typical of amebic infections but in histological sections amebae were never observed beneath the muscularis mucosae. The average incubation period of 6 days is considerably shorter than the 11 days of Walker and Sellards[8] for their human experiments but this is probably due to the fact that the infections could be detected by cecal aspiration at a much earlier date. If the conditions of an amebic infection in the dog are at all comparable to those in man, it is probable that there is no such thing as a healthy carrier condition in man and that all individuals harboring the organism have amebic lesions in the large bowel.

Conclusions. From the evidence cited above it is highly probable that all "carrier" strains of *E. histolytica* are pathogenic and that "healthy human carriers" as well as those individuals exhibiting clinical manifestations of the disease should be regarded as clinical cases.

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Conversion of Dibenzyl Disulfide to Hippuric Acid in the Rat.

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It has been demonstrated that benzyl chloride, S-benzyl-*l*-cysteine and S-benzylglutathione, when administered to animals, yield in the urine N-acetyl-S-benzyl-*l*-cysteine.[1-3] S-benzyl-*d*-cysteine undergoes optical inversion in the rat, via the oxidative deamination, to yield N-acetyl-S-benzyl-*l*-cysteine.[4] The optical inversion of S-

1 Stekol, J. A., *J. Biol. Chem.*, 1938, **124**, 129.

2 Stekol, J. A., *J. Biol. Chem.*, 1939, **128**, 199.

3 Stekol, J. A., PROC. SOC. EXP. BIOL. AND MED., 1940, **43**, 108.

4 du Vigneaud, V., Wood, J. L., and Irish, O. J., *J. Biol. Chem.*, 1939, **129**, 171.