

- Entamoeba nipponica*. *Centralbl. f. Bakt., etc.*, 1909, I. Orig. II, p. 650.
- *v. Prowazek, S. Beitrag. zur Entamoeba-Frage (*Entamoeba williamsi*) *Arch. f. Protistenk.* 1911, xx, 3, p. 345.
- *Idem. *Entamoeba*. (*Entamoeba polecki*.) *Arch. f. Protistenk.*, 1912, xxv, 2, p. 273.
- *Idem. Weiterer Beitrag zur Kenntnis d. Entamoeben. *Arch. f. Protistenk.*, 1912, xxvi, 2, p. 241.
- *Ibid.
- *Butschli, O. Beitrag. zur Kenntnis der Flagellaten u. einiger verwandter Organismen. *Zeitschr. f. wissensch. Zool.* 1878, 30, p. 273.
- *Grassi, Atti della Società Ital. d. Scienze natural. Vol. 24, p. 182. 1881.
- *Idem. Ibid., p. 181. 1881.
- *Liebetanz. *Amoeba bovis Liebetanz*. *Berliner tierärzt. Wochenschr.*, 1905. No. 18.
- *Castellani, A. Note on a Liver Abscess of Amoebic Origin in a Monkey. *Parasitology*. 1908, I, p. 101.
- *Hartmann, M. Entamoeba testudinis. Note in Doffein's Lehrbuch der Protozoen kunde. Berlin, 1911, p. 598.
- *Prowazek, S. v. Weitere Beitrag zur Entamoeba-Frage. *Arch. f. Protistenk.*, 1912, xxvi, 2, p. 241.
- *Musgrave and Clegg. Amebas. Their Cultivation and Etiological Significance. Bureau Govt. Laboratories, Manila, 1904. No. 13.
- *Hartmann, M. Die Dysenterie-Amoben. Handbuch d. Path. Protozoen. Prowazek, S. v. Leipzig, 1912, I, p. 50.
- *Werner, H. Studies regarding Pathogenic Amoebae. *Indian Med. Gazette*, 1909, xlv, p. 241.
- *Whitmore, E. Parasitäre u. Freilebende Amoben a. Manila u. Saigon. *Arch. f. Protistenk.*, 1911, xxiii, p. 70.
- *Walker, E. L. Loc. cit.
- *Liston and Martin. *Quar. Journal Micro. Sciences*. N. S. 226, 1911, p. 279.
- *Wells, R. T. Aerial Contamination as a Factor in the Study of Amoebic Infections by Cultural Methods. *Parasitology*, 1911, iv, p. 204.
- *Craig, C. F. Observations upon the Morphology of Parasitic and Cultural Amoebae. *Jour. Med. Research*, xxvi, I, 1912, p. 1.
- *Loesch. Ibid.
- *Hlava. Ibid.
- *Kovacs, F. Beob. u. vers. d. Amoben-Dysenterie. *Zeitschr. f. Helikde*, 1892, xiii, p. 509.
- *Kartulis. Loc. Cit.
- *Zancoral. Pathogenie des absces du foie. *Centralbl. f. Bakt., etc.*, 1893, I. A. Abt. xiv, p. 638.
- *Strong and Musgrave. Etiology of the Dysenteries of Manila. *Ann. Rep. Surg. Gen'l Army*, 1900, p. 251.
- *Hartmann, M. Untersuch. u. parasitische Amoben. I. *Entamoeba histolytica*. *Schaudinn. Arch. f. Protistenk.*, 1909, xviii, 2, p. 207.
- *Viereck, H. Loc. cit.
- *Werner, H. Loc. cit.
- *Schaudinn, F. Loc. cit.
- *Vedder, E. An Examination of the Stools of One Hundred Healthy Individuals, etc. *Jour. Am. Med. Assoc.*, 1906, xxvi, p. 870.
- *Sistrunk, W. E. Intestinal Parasites found in Individuals Residing in the Northwest. *Jour. Am. Med. Assoc.*, 1911, lvii, p. 1507.
- *Ashburn and Craig. *Entamoeba coli*, etc. *The Military Surgeon*, 1907, I, p. 21.
- *Craig, C. F. Observations upon Amoebae Infecting the Human Intestine, etc. *Am. Med. Phila.*, 1905, ix, pp. 854, 897, 937.
- *Stiles, C. W. The Presence of *Entamoeba histolytica* and *Entamoeba coli* in North Carolina. *Pub. Health Rep. Washington*, 1911, xxvi, 34, p. 1276.
- *McCarrison, R. Observations on the Amoebae in the Intestine of Persons suffering from Goitre, etc. *Quar. Jour. Micro. Sciences*, 1909, 53, p. 723.
- *Whitmore, E. Loc. cit.
- *Walker, E. L. Loc. cit.
- *Darling, S. T. Loc. cit.
- *James, W. T. Personal Communications.
- *Elmassian. Loc. cit.
- *Prowazek, S. v. Weiterer Beitrag zur Kenntnis der Entamoeba-Frage. *Arch. f. Protistenk.*, 1912, xxvi, 2, p. 241.
- *Kartulis. Loc. cit.
- *Kruze and Pasquale. Studium des Dysenterie und Leberabscesse. *Deutsche Med. Wochenschr.*, 1893, 15, p. 354; 16, p. 368.
- *Celli and Flocca. Ueber die Aetiologie der Dysenterie. *Centralbl. f. Bakt., etc.*, 1895, I, xvii, p. 309.
- *Strong and Musgrave. Loc. cit.
- *Jurgens. Zur Kenntnis der Darmamoeben u. d. Amobenenteritis. *Veroff. a. d. Geb. d. Milit-Sanitätswes.*, Berlin, 1902, 20, p. 110.
- *Wellman, Creighton. Personal Communications.
- *Craig, C. F. The Parasitic Amoebae of Man. *Phila.*, 1911, p. 107.
- *Craig, C. F. Studies upon the Amoebae in the Intestine of Man. *Jour. Infec. Dis.*, 1908, 5, 3, p. 324.
- *Werner, H. Loc. cit.
- *Wellman, Creighton. Personal Communication.
- *Viereck, H. Loc. cit.
- *Hartmann, M. Untersuch. u. Parasit. Amoben. I. *Entamoeba histolytica*. *Schaudinn. Arch. f. Protistenk.*, 1909, xviii, p. 207.
- *Werner, H. Loc. cit.
- *Fantham, H. B. Personal Communication.
- *Darling, S. T. Loc. cit.
- *Franchini, G. Experimentelle Tropendysenterie, etc. *Centralbl. f. Bakt.*, 1912, I. Abt. Orig. 61, p. 590.
- *Musgrave and Clegg. Loc. cit.
- *Greig and Wells. Dysentery and Liver Abscess in Bombay. *Scientif. Mem. Sanit. Depts. Govt. of India*, N. S. 47, 1911.

DIAGNOSIS AND TREATMENT OF AMEBIC DYSENTERY.*

By E. M. Mason, M.D.,
Birmingham, Ala.

When Osler, in 1890, reported the first case of amebic dysentery for America but little interest and no enthusiasm was aroused. The infection

*Read at the Sixth Annual Meeting of the Southern Medical Association, Jacksonville, Fla., November, 1912.

had occurred in Panama and no one supposed that amebiasis would ever prove of importance in the temperate zones. Time has changed this view, case reports having come from as far north as Canada, and from nearly every State in the Union. Indeed, so widespread is the infection that some recent writers express the opinion that amebiasis is second in importance only to hookworm disease. This extreme view I am not prepared to accept, but it is undoubtedly true that the disease is widely disseminated throughout the country; to a much greater extent, in fact, than is generally supposed to be the case. Of great interest, also, is the fact that in many localities the non-pathogenic *Entameba coli* abounds and must be recognized in order to avoid the grave error in diagnosis and prognosis that would follow were it mistaken for a pathogenic species.

Diagnosis.—When in a patient suffering from chronic diarrhoea or diarrhoea alternating with periods of constipation, we are led to suspect amebic infection; two questions present themselves for consideration. First, are amebae present? Second, if so, to what species do they belong? The answer to these questions lies in that *bête noir* of many clinicians, thorough examination of the feces.

Here the distant laboratory can be of no assistance to us. Rarely, and in warm weather, one finds motile amebae in a stool two or three hours old, but all motility ceases at a temperature but little below that of the body, hence our examinations must be made on a freshly passed stool which has not been allowed to become cold.

However sure experts may be of their ability to diagnose amebae in the encysted stage, it is not safe for the average clinician or laboratory worker to call any body ameba, however typical he may think it to be, unless he actually sees it in characteristic ameboid activity. It is an excellent plan to give a saline cathartic before collecting the specimen for examination, in order that the intestines may be well flushed out and the amebae washed away from the floors of the ulcers. One then collects the semi-fluid stools preceding the more liquid actions. Much depends on the portion of the stool selected for examination under the microscope. If mucus, which is frequently blood-tinged, be present, a bit of it is taken; otherwise, the more liquid part rather than the solid material should be chosen. If the weather be cool the slide should be quickly prepared and should be kept warm during the examination.

More often than not flagellates—*cercomonads* and *trichomonads*—are found in symbiosis with amebae, hence the presence of these actively

motile little bodies will often arrest our attention when first focussing on the preparation. Further examination may reveal the presence of motile amebae which we can then proceed to study for classification. Negative findings are by no means conclusive, however. Many times I have failed to find amebae on feces examination, only to discover them in great abundance when the rectal tube was brought into service. Only recently a patient was seen whose stools had been repeatedly examined by several men with negative results. On introducing the rectal tube, a bit of mucus was obtained which was teeming with amebae. In fact, feces examinations are so often negative and rectal tube examinations so uniformly satisfactory that in our work the latter method has almost entirely replaced the former. The tube may be passed in the office, if necessary, and one seldom fails to find a mass of amebae-laden mucus either in the tube contents, or scraped off by and adherent to one of the windows in the side of the tube. If the tube and contents must be transported from the home to the laboratory for examination, the necessary temperature may be maintained by placing the tube and contents in a warm vacuum bottle, or in a jar which is kept warm by being immersed in a larger jar filled with warm water.

Something like a dozen species of amebae have been described as parasitic in the intestines of man. No doubt several of these are the same species under different names. According to present knowledge, only three of them are of importance to us in this country. These are: *Entameba coli*, *Entameba histolytica* and *Entameba tetragena*. The last named species is rare outside of the tropics, but one should be on the lookout for it. *Entameba histolytica* is the common pathogenic ameba, while *Entameba coli* is a harmless commensal which is of importance only because it may be mistaken for the pathogenic species. Throughout the tropics the *Entameba coli* seems to be a normal inhabitant of the intestinal tract in a large proportion of the inhabitants. Craig found it in the feces of 71 per cent of 107 healthy soldiers in the Philippines, his results being confirmed by Vedder, who found it in 75 per cent of fifty healthy Filipino scouts whose feces he examined. Stiles, Craig and some other authorities hold that *Entameba coli* is almost equally prevalent in this country, a belief which I cannot accept for Maryland and Alabama, the States in which my work has been done. Dock in Michigan and Simon in Louisiana were also unable to find *Entameba coli* in more than a small per cent of the stools examined from normal per-

sons. However, there are numerous localities where infection with *entameba coli* does occur, either alone or associated with the pathogenic species, hence we must be on the alert to differentiate it from the other types.

For detailed descriptions of these parasites, reference may be had to the original communication of Schaudinn as regards *entameba histolytica* and *entameba coli*, or to the excellent monograph of Craig, where all the types are described. I shall only enumerate the chief differential points here.

Entameba coli averages from two to three times the size of a red blood corpuscle. The distinction between ectoplasm and endoplasm is never marked and often is made out with great difficulty. The nucleus is prominent, centrally located and has a definite nuclear membrane. Vacuoles are rare and poorly defined.

Pseudopodia are short and blunt, and the movements of the parasite are usually quite sluggish. Contained bodies, including red blood cells, are infrequent.

Entameba histolytica averages somewhat larger than *entameba coli*, being from three to six times the diameter of the red blood cell. The ectoplasm is strikingly differentiated from the endoplasm, the sharply outlined, clear, glassy ectoplasm being one of the chief earmarks of this organism. Craig speaks of a peculiar greenish color, due to pigment from the contained blood cells, as characteristic of this parasite. *Entameba histolytica* seldom shows a nucleus; when one is present it is poorly defined and excentrically placed. One or more well-marked vacuoles are to be seen in the ameba of average size. Significant also is the fact that in this species red blood cells are frequently contained in the body of the parasite, often a dozen or more being seen in one ameba. Pseudopodia are more finger-like than in *entameba coli* and are always formed by the clear ectoplasm, the endoplasm flowing in after the pseudopod is well formed. In fresh specimens motility is very active.

Entameba tetragena is not thought to be a common parasite in this country, but a number of cases have been reported. In size it shows no distinction from the forms already described. Ectoplasm and endoplasm are clear and distinct, while a large, well-defined nucleus is constantly present. Vacuoles may or may not be present. Motility is active, the pseudopodia being formed primarily by the clear ectoplasm.

Ordinarily anyone who has had a fair amount of experience in amebic work can readily classify the organisms found. In doubtful cases recourse

must be had to the staining reactions and methods of reproduction of the parasites. This entails a considerable amount of time and work, but fortunately one seldom has to proceed so far for diagnosis alone.

The staining reactions are not specific, but when properly stained with Hasting's or Wright's stain, *entameba coli* shows the deeper stain in the endoplasm, *entameba histolytica* in the ectoplasm, while *entameba tetragena* is said to take the stain practically uniformly throughout.

For study of the reproductive process one must have a warm stage, or, better still, a warm box for the microscope, and follow the development of the parasite through many hours.

Aside from the process of simple division, which is common to all, each species has a definite and characteristic method of reproduction by schizogony by means of which the parasite reproduces under unfavorable conditions.

Entameba coli undergoes encystment and forms eight daughter amebae within the cyst, *entameba histolytica* by the formation of spores which are extruded by budding from the periphery of the parent organism, while *entameba tetragena* reproduces like *entameba coli* by the formation of daughter amebae within a cyst, but with the difference that only four daughter amebae are formed.

Treatment.—Amebic infection occurs sometimes through infected water; fruits and green vegetables are often at fault and contact infection, either direct or indirect, very likely plays an important part in the dissemination of the disease. Proper control of these avenues of infection would mean the eradication of the disease.

For the individual patient, rest in bed for at least two weeks is essential to the first step in treatment. The diet should be confined to liquids at first, with the gradual addition of light solids as the patient improves. Should the patient apply for treatment during the stage of acute diarrhoea, it is preferable not to institute specific treatment until the urgent symptoms have been controlled by means of rest in bed, proper diet, simple colonic lavage and, perhaps, the use of opium and bismuth. Should there be constipation, saline cathartics should be used freely until the bowel is thoroughly cleansed.

Until quite recently colonic irrigation with germicidal solutions was the mainstay in specific treatment, nearly all of the well-known antiseptics having been used at one time or another, quinine, silver nitrate and mercuric chloride being the favorites. Of these, quinine was most commonly

employed. Starting with a solution of 1 to 1,500, the strength was gradually increased until a 2 to 3 litre irrigation of 1 to 500 strength was used twice daily, the patient changing from knee-chest to right lateral position and retaining the solution for fifteen to twenty minutes. Under this plan of treatment some undoubted cures resulted, but a number of objections arose. Not all patients can retain a two-quart enema for fifteen minutes. The physician must himself, in the majority of instances, give the enema, since proper introduction of the rectal tube cannot be left to the ordinary hospital orderly. Cinchonism rapidly supervenes in many patients and in others the inflamed condition of the bowel makes a continuation of the treatment impossible. Finally, the treatment is tedious and not always successful, for when deep-seated, undermined ulcers are present the fluid does not reach the amebae at the bottom of the ulcers. Some writers have even asserted that the beneficial effects of this plan of treatment were due to the systematic colonic lavage rather than to the quinine. Be that as it may, it is certainly true that irrigation with cold water or plain salt solution has resulted in marked improvement in many instances.

Of all the remedies proposed, ipecac has, in my experience, given the most prompt and satisfactory results. Its action seems to be truly specific, and it is not uncommon to note the entire disappearance of amebae from the stools after only one dose of the drug. The nausea and vomiting that follow the improper administration of ipecac have caused it to be looked upon with disfavor by many physicians, yet these untoward effects may be easily averted by proper preparation of the patient and of the drug.

Before beginning ipecac treatment the patient should be placed on a liquid diet and the bowels should be thoroughly flushed by means of a saline cathartic. For three hours before giving the drug the patient should receive no nourishment. The ipecac should be of proved potency and should be administered in the form of freshly prepared salol-coated pills or capsules. Much of the trouble that has arisen from the use of ipecac has been due to the use of improperly coated pills, or pills that were so old and hard that they passed through the intestines without being dissolved. For some time it has been my custom to use the powdered drug in the form of salol-coated five-grain capsules instead of pills, each lot of capsules being freshly prepared by a competent pharmacist at the time when needed. The object of the salol coating being merely to protect the capsule during its passage through the

stomach, the salol covering should only be of sufficient thickness to serve this purpose. The amount of salol used is perfectly harmless. The proper dose may be given at intervals during the day, or, preferably, in one large dose at bedtime. The usual custom is to give on an empty stomach at bedtime from sixty to eighty grains of ipecac with as small a quantity of water as possible, the patient being directed to lie on the right side for an hour after swallowing the capsules. In nervous or apprehensive patients it may be desirable to give a small dose of opium. Given in this way, ipecac but rarely causes nausea or discomfort, the patient passing a comfortable night. The same dose is repeated nightly for three or four nights, then, if the stools be free of amebae, the nightly dose is decreased gradually until ten grains only are given. This dose is continued for from one to two weeks. At the end of this time silver nitrate irrigations may be used if rectal ulceration is pronounced.

Under this plan of treatment patients are frequently enabled to return to work at the end of two or three weeks, but they should be cautioned to be on the lookout for a return of symptoms, since the well-known tendency to recurrence may make a second course of treatment necessary.

Another virtue claimed for ipecac is that it lessens the likelihood of liver infection, with the formation of abscess, which occurs in something like 20 per cent of untreated or imperfectly treated patients. Since using the ipecac treatment I have never seen a case of amebic abscess of the liver.

In conclusion, a word may be said with reference to the surgical treatment of amebiasis by means of appendicostomy and irrigation through the caecum. I do not feel that operative treatment is justifiable in the ordinary case, though there may be a field for it in the occasional chronic and intractable case that fails to respond to the usual form of treatment. In the two cases in which I have seen surgical intervention resorted to the results were far from satisfactory. Should an operation be done, the caecum rather than the appendix should be opened.

Summary and Conclusions.—Amebic dysentery is not confined to the tropics, but may be encountered in any part of the United States and even in Canada.

Outside of the tropics infection with *Entamoeba coli* is probably not so common as has been supposed. The rectal tube is an indispensable adjunct to technique in diagnosis, and the finding of definitely motile amebae is the first step in diagnosis. Careful study of the fresh specimen

will enable one to differentiate the amebae in practically every case. Proper classification of the parasites found must always be made, otherwise the diagnosis of amebic dysentery may be erroneously based on the presence of harmless *Entameba coli*.

The ipecac treatment of amebic dysentery is practically specific.

DISCUSSION.

Dr. Sidney K. Simon, New Orleans—I think we are to be congratulated on having been able to listen to such instructive and comprehensive papers on this subject. Dr. Craig's work in this field has been that of a master mind, so to speak, and to those of you who have not yet read his work on the parasitic amebae of man, I would advise you to do so, provided you are sufficiently interested in the subject, and I think all Southern physicians should be sufficiently interested in this subject to read that work, which, to me, reads almost like a novel.

The point Dr. Craig brought up in regard to the mode of transmission by the roach or the mouse is certainly a new point and a very interesting one. I had always believed in a sort of theoretical way, without practical demonstration, that the banana was largely accountable for the distribution of amebic infection in this country. As you know, the bananas are imported from the Central American countries in large numbers, shipped through the Southern ports of the United States, and shipped to all sections of this country in cars without, in many instances, having touched the hands of any of the American dock laborers, and so forth. Since we know that the amebae grow rapidly or readily on the over-ripe banana, it struck me as a possible means of transmission from the South American countries that the banana might be a source of infection. I was particularly struck by that the summer before last, when in Rochester, Minn., when I learned that they had discovered forty cases of amebic dysentery originating in that city. Minnesota has one of the most frigid climates in the United States in winter. It is certainly not a sub-tropical climate, and still the doctor who gave me the information was able to prove conclusively that these forty cases had never been out of the Northwest, and in many instances not out of Rochester, Minn., showing the widespread distribution of this infection not only in this country, but in all northern climates, and how little justice there is in claiming that amebic dysentery is distinctly a tropical disease.

The fact that amebae can be transmitted by roaches and mice is very interesting, and I shall keep it in mind. The differentiation between the different forms of amebae is a very serious problem to the general practitioner. We have had some difficulty among our students at home in teaching them to find amebae, in teaching them to look for amebae and find them. Now we have, according to our present knowledge, not only to instruct the practitioner of medicine to examine the stools, which is a somewhat difficult thing to do, because

somehow or other the examination of the stools is considered beneath the dignity of the majority of doctors, and they do not seem to realize the very great importance of examination of the stools, especially in the South, but now we have to teach them not only to examine the stools, but that not every ameba they find in the stools is a pathogenic ameba; that some of the amebae they see do not cause disease. What Dr. Craig has said is true, that it is not such a very easy matter, even for the experienced always to differentiate *Entameba coli* from the ameba *histolytica* or *tetragena*. I believe for the average practitioner, who has not a great deal of time to delve into these matters of differentiation, the various staining methods, such as Dr. Craig has worked out so beautifully, a very good practical point for him to remember is this: If you find an ameba in the stool which moves and which contains red blood vessels or cells, you are perfectly safe in saying this is a harmful ameba, and not the harmless variety, and you will not go astray if you consider that case of movable ameba containing red cells as true ameba *histolytica*, and that, therefore, it is a case for treatment.

As to the distribution of the *Entameba coli*, I have been interested in it for several years, and I am prepared to say, although not ready to formulate statistics at the present time, that the *Entameba coli* is not the common variety of species in Louisiana, and in the neighboring Southern States it has not been found common in the stools of individuals I have examined. Just a point in connection with that to show it does occur, I recently had occasion to see a case of dysentery which presented the clinical picture of amebic dysentery, but in whose stools I could never find anything but the *Entameba coli*. Try as hard as I could with large doses of salines, there would always be the *Entameba coli*. This case turned out to be one of chronic Flexner-Harris infection, demonstrated by finding the agglutination reaction in the blood and isolating the Flexner-Harris bacillus in the stools. This case would not respond to the ipecac treatment.

Dr. Mason's points were practical, and for one having been an advocate of the ipecac treatment for many years, I am always glad to hear of a new convert. His point in regard to repeated examinations of the stools is important. You cannot hope to make a diagnosis of amebic dysentery always with one examination of the stools. My method is to make, on my first visit to the patient, an examination, passing a rectal tube with several windows cut into it, with snares to catch the mucus. I pass the tube about one foot, pull it out with a sudden jerk, and pull out the window portion of mucus-containing blood. If I do not find amebae readily, I will, in addition to that, pass the proctoscope and try to pick off from the ulceration in the rectum pieces of mucus-containing blood. If that is not successful, I give the patient salines to wash down these amebae which may be present in the cecum or ascending or transverse colon, as well as in the rectum. But as I have the conviction that the clinical picture is one of amebic dysentery, it may take four or five days to be absolutely certain, because there is

only one way of diagnosing amebic dysentery, and that is by finding amebae in the stools. There is no other possible way. The clinical history is not sufficiently characteristic to make the diagnosis.

In regard to the ipecac treatment, I have a few words to say. The ipecac treatment was gone over so carefully by Dr. Mason that I only want to bring up one or two points. The mode of administration is the main thing. Again, I have found, by personal conversation and in the literature, that there are those who have failed with the ipecac treatment because they have not followed the detailed methods of administering it. You cannot hope to succeed in treating amebic dysentery with ipecac if you allow a patient to walk around. The patient must go to bed for two weeks. There should be nothing short of that, no compromise. The diet during the first week is absolutely liquid, and the bowel is first washed out with saline cathartics before administering the ipecac. The idea in my mind is this: In order for the ipecac to reach the amebae, the bowel must be fairly clear of all solid material, so that it can spread and get into the crevices in every possible way. The dosage is an immaterial matter. I have never seen anybody harmed by ipecac, and I have given 100 grains in one dose and figured the salol contents of the pill as high as fifty grains in one dose. I have examined the urine to find traces of salicylic acid poisoning. Nobody in my experience has ever been harmed in the slightest by the use of ipecac or by the salol.

Dr. John T. Halsey, New Orleans.—The question of the diagnosis and treatment of amebiasis is so important that it justifies me in taking a moment or two of your time. I also am a convert to ipecac, although one of the older ones. I was taught in my early practice that quinine irrigations and various other irrigations were useful in the treatment of this disease, but they failed over and over again, and I had the conviction that amebic dysentery was often an incurable disease. I saw patients get well, but I saw a number of them who had apparently gotten well in whom the disease recurred, so that I heard of them later as suffering from the old trouble and dying from it. I think there is a possibility, when we are discussing a disease like this, that the impression is conveyed that the ipecac treatment of dysentery is certain to cure every case of amebiasis that is treated with it and treated correctly. I must confess that although I have endeavored to follow out this treatment absolutely and in every detail as it has been worked out by Simon and others, I find it is not always successful. There is one great difficulty one meets with, and that is, we cannot always get properly prepared salol-coated pills, and we probably very seldom can get the salol-coated capsules, with a coating just thin enough and not too thick. I have also repeatedly seen the pills pass through the intestine undissolved. These have probably often been badly coated pills, but especially in the cases of long standing those poor individuals who come to one after years and months of dysentery, and who are practically living skeletons, there are, it seems to me, physiological or pathological reasons why

salol-coated pills pass through the bowel intact. The secretions, particularly the alkaline secretions of the intestine, the pancreas and liver, are very markedly diminished in cases of extreme emaciation, and I am convinced that has a bearing on the passage in the undissolved state through the bowel of the salol-coated pills. Understand, I am not knocking these pills, as they are the most practical way of giving ipecac, and are all right if they are broken up in the intestines, but we will meet with some cases in which they will not be dissolved, and we ought to have an alternative method of administration, and this we have in the old method that goes back, how many centuries I do not know, of giving a patient who has been on a liquid diet ten or fifteen minims of the tincture of opium, and half an hour or an hour later, giving him twenty or thirty or forty or more grains of finely powdered ipecac in suspension. This method has its own great advantages, and I believe is the only way in which you can give ipecac efficiently in the particular cases which will not dissolve the salol. That is a point that ought to be brought out. It will stand you in good service.

Another point is with reference to the possibility of surgical help in these cases. I am sure we will all meet cases which we are not able to cure by the ipecac or any other method of medical treatment. Such cases should be treated surgically either by appendicostomy or even better by ileostomy or colostomy with artificial anus, which will keep from the inflamed, ulcerated bowel all irritating material, which will give it physiological rest, and at the same time and through the same opening permit of our applying medicinal solutions and astringents or ipecac decoctions directly into the lower bowel.

Dr. J. B. Wallace, Tampa, Fla.—As bearing on the importance of this subject, I would like to say something about the experience we had in Tampa a few months ago. I do not know how many cases of amebic dysentery we had down there; the diagnosis was made in the laboratory in thirty-five cases, but in addition to these, there were about fifty cases of abscess of the liver operated on at the local hospitals during the first four or five months of the year. The consensus of opinion was that the epidemic was due to infection from uncooked vegetables, lettuce particularly. It is possible that sporadic cases of amebic dysentery occurring in localities remote from the tropics may be due to the use of uncooked, infected vegetables coming from regions where it is prevalent. We have been thoroughly converted to the use of ipecac in these cases. In referring to the use of ipecac, Dr. Mason spoke of the time when it should be given; we first used it in a case where the patient was desperately ill, and after we had unsuccessfully tried practically everything else; after a few doses the patient began to recover. The majority of our cases were rather mild types of dysentery; in practically all the cases of abscess of the liver no diagnosis of amebic dysentery had been made. The history usually given was of a mild type of dysentery that had not been treated.

Dr. John L. Jelks, Memphis, Tenn.—If I loved a

fair little lady so well as some of my friends love ipecac, I would lean over and kiss her hand. That would be homage enough. I have seen two cases of amebic dysentery that originated in Denver, Col. Amebic dysentery had appeared sporadically. I am informed by the post surgeons in Yellowstone Park, while I was there. Lettuce and other raw vegetables, together with the indiscriminate use of them when the soil on which they are grown is fertilized with human excreta, is one of the prolific sources of amebic dysentery. Why should railroads be allowed to scatter amebae along their lines with utter disregard of State rights and with utter disregard of the protection of our people? We know that there is the indiscriminate interstate distribution of hookworms and amebae from one end of a railroad to another. This effete material should be controlled the same as any other dangerous products, and not scattered over the railroads for flies to carry amebae from there to the lettuce plants where they are likely to roost during a hot day. Human excreta distributed over vegetables and upon seeds is another dangerous thing that this Government should stop. Do not bring the excreta to your office; take your microscope to the patient. You may go into the rectum and scrape the mucosa, and if you find ulcers, there is where you are apt to find amebae. I have examined the excreta of human beings for ameba histolytica, and not being able to find it after repeated examinations, later I have found it. When you find amebae with the presence of pseudopodia, do not stop, but treat the case for amebic dysentery. It is unnecessary to have the symptom dysentery for amebiasis. I have operated on an enormous liver abscess in which there was absolutely no history of dysentery. Salol will do good, and I believe it will do more good than ipecac, and yet I know that these gentlemen are just as honest as I am, and I know that I am honest, and I am going to prove it to them. I have tried the use of ipecac by the mouth; I have tried ipecac through salol-coated pills, and have put it through the appendicostomy openings in large quantities. I have had doctors testify as to this and patients testify to it. For instance, when I asked a doctor, "Have you taken ipecac treatments?" he replied, "Oh, I have taken a neck of that." Why'd he not get well? He had lost 100 pounds in weight. I did not perform a dangerous operation. You have got no right to lose a patient by ceco-appendicostomy. One of three patients operated upon, one sat up the first day after the operation; another the second day, and the third sat up in about the third day. Is it a dangerous operation? No. The performance of appendico-cecostomy is preferable to any other procedure, and not appendicostomy, which is not suitable for these cases. By the use of formo-boric solution you can control the dysentery without any great danger and without any trouble. You can control the diarrhea. You may give ipecac if you want to; but, gentlemen, treat the disease locally. It is a local infection of the colon, the upper rectum and the sigmoid. But you can treat the disease locally by nitrate of silver applications to the diseased area below and by formo-boric irrigations. That is my treatment.

Dr. Stevens T. Harris, Highlands, N. C.—I have been very much interested in what has been said in reference to this subject. I have had considerable experience with this disease. It was my good fortune to be connected with the First Reserve Hospital in Manila for a while, where we had any amount of amebic dysentery. We used all manner of treatments there, and, of course, as the routine treatment we resorted to ipecac. I cannot say that we found it anything like a specific for the disease. I would like to say in regard to what Dr. Wallace said in reference to giving opium as a preliminary procedure, giving the ipecac in a bolus or in suspension is good treatment.

There is one treatment that has not been mentioned here today which, to my mind, is the summum bonum of treatment of amebic dysentery, and that is irrigations by peroxide of hydrogen. I have never yet seen a case that did not respond to this treatment.

There is one other treatment that is to be remembered, and which has a salutary effect, and that is the use of urotropin. In addition to this treatment, it is very essential that the patient should receive rest in bed. In fact, it is as important as any other part of the treatment.

Dr. J. B. Elliott, New Orleans—If we understand thoroughly the pathology of amebic dysentery, I cannot see how we can use enemas or rectal injections to cure a case of amebic dysentery. When we realize that the amebae are buried in the mucosa and sub-mucosa, and even down into the muscle layer of the bowel, how can we ever believe that we can drive quinine or anything else up into that bowel, to have it hunt out, search for and kill the amebae? We must act through the blood. We must bring the opsonins, the antibodies to bear. We must increase the serum and therewith the antibodies through the whole course of the large bowel. The opsonins are the cause of the death of the amebae. They prepare the amebae for ingestion by the phagocytes. How irrigations can hunt out amebae and strangle them, I cannot understand.

I am a thorough convert to the use of ipecac. A patient who came to the Charity Hospital last week for hemorrhoids had a typical case of amebic dysentery. The next patient came in with a diagnosis of tuberculosis of the bowel, a very rare disease in our op'n'on. Tubercular enteritis is a rare thing unless the lung is very much involved. The second case gave a history of loose bowels extending over a period of eighteen months, with intervals of freedom from looseness of the bowels. These were cases of amebic dysentery. In the last four weeks I have seen seven cases of amebic dysentery, all of which came in either with a diagnosis of tuberculosis of the bowels or hemorrhoids. We found the amebae in the stools. We followed this plan: We had the patients walk over to the laboratories, where the students can examine the stools under the microscope, and we show them the amebae. When the diagnosis of amebic dysentery is made we follow with sixty grains of ipecac the first night, fifty the second night, and forty the third night, decreasing by ten grains each night; then ten grains of ipecac every

night for five days, and Ipecac off and on again if they should have a recurrence. In some cases I have used coal oil and water by enema, half and half, and the results were fairly good when it was first used, but I have given that up and am sticking now to Ipecac alone, plus rest and a careful diet.

Dr. J. E. Paullin, Atlanta, Ga.—I did not have the pleasure of hearing these papers on amebic dysentery, but I had quite an experience with the disease this summer, having seen an unusual number of cases. I treated the first series with the usual routine of Ipecac administered very much after the manner Dr. Elliott has been giving it. I used also in quite a number of these cases, and particularly in one man who had from twenty to thirty stools a day, and in each stool there were any number of the entameba histolytica, an injection of argyrol, one per cent of a two per cent solution, which is non-irritating, and the man retained it in his bowel over night. This was the most soothing thing as an irrigation which I could get for him. There was no other treatment resorted to for two or three days until his stools came down to three or four a day. I used in other cases equal parts of liquid alboline and coal oil. I found after these irrigations that the number of stools diminished and the amebae diminished temporarily. By accident, however, I happened to find out another drug which was used for another purpose, and which gave me a very surprising result. I realize fully the statement I am making is not a conclusion, but a simple statement, and that it is drawn from entirely too few cases. But in one gentleman who had amebic dysentery, and who was having from eighteen to twenty stools a day, and who had that dysentery for two years, off and on, examination of the stools showed any number of entameba. He happened to have syphilis at the same time; he was given an injection by another individual of salvarsan, and within two or three days he had a firm stool, the first solid stool he had had in several months. Examination of the stools after that did not show any entamebae. I do not know whether he is well yet or not. It may be simply a coincidence. I happened to have had one other case of amebic dysentery, the patient receiving an intravenous injection of neosalvarsan with the same results. This was only two weeks ago. The second patient did not have syphilis, but amebic dysentery. He received intravenous injections of neosalvarsan, and my attention was directed to the possibility in this case of the value of this drug, and I would not say that these cases are well. However, I have not found entameba in the stools since the injection. My attention was directed to that agent by Dr. Ballinger, who happened to have given salvarsan to a man who had had dysentery for a considerable length of time, and at the same time he had syphilis. As to whether it is worth anything or not, I do not know. As I said, there have been no symptoms of dysentery nor entamebae in the stools since. I have found some patients who could not take Ipecac, and in those recently I have given them the deodorized tincture of opium beforehand or a hypodermic injection of morphine. The Ipecac seems to upset them.

I have also used salol-coated pills in these cases who could not take Ipecac. I have treated them in that way, then I have put the salol-coated pill in a capsule, and then put the capsule in a one per cent solution of formaldehyde and water. This hardens the gelatin of the capsule, and by drying it for a certain length of time it will not dissolve in the stomach. I have found some patients who would become nauseated and depressed from the use of Ipecac. In these cases I have given the patients flower of sulphur. Of course, the flower of sulphur will produce in these patients a slight diarrhea. It has caused, at times, a temporary disappearance of amebae from the stools. As to whether that is worth very much or not, I do not know. The cases are very few.

Dr. William Litterer, Nashville, Tenn.—With reference to the distribution of the entameba coli, I cannot subscribe to the statement that has been made, that it is not widespread. I am only speaking from my investigations in the city of Nashville and State of Tennessee. I find that it is quite prevalent there. We found the entameba coli in a number of cases, and we have found it in conditions other than regular amebic dysentery. By giving the patients a saline purge it will bring out occasionally the amebae.

As to the differentiation in fresh specimens, I think it is quite difficult to tell the difference between entameba coli and histolytica and the tetragens, and this is especially true if you have not the phagocytosis or the red blood cell, a point made by Dr. Simon, and which I heartily endorse. I believe that is a safe rule to follow, that unless you have them it is difficult to tell the difference in the living culture without staining, and especially in the encysted stage. We get specimens sent in by means of which it is easy to diagnose entameba coli. By letting the specimen stand for forty-eight hours you can see the nuclei, and that will tell you at once as to whether it is non-pathogenic or not. As Dr. Simon has pointed out, where you find these red blood cells going on, and you are treating some of the cases, you will have ameba histolytica in which there will be no red blood cells engulfed.

As to the treatment, I have been an advocate of the use of Ipecac in these cases. It is certainly convincing, after you once give this medicine, to see the disappearance of the histolytica. Often you cannot revive them if you have scraped the ulcers. They appear to be dead or paralyzed in some way.

With Dr. Jolks, I believe the best means in making a diagnosis is by examining the scrapings from the ulcers. The rectal tube is the second best means of making a diagnosis, but the scrapings of the ulcers is the best, and even then sometimes you may not be able to find the amebae.

Dr. C. C. Bass, New Orleans.—I should like to point out a possible error in depending upon scrapings from the rectum and lower bowel for the diagnosis of amebic infection. We must fully realize that the pathologic lesions of this disease are located chiefly considerably above that point, and especially about the flexures and frequently at the cecum.

With reference to examining the sigmoid with

the proctoscope, if we only go that far with our examination and treatment of the ulcers present. I am afraid it would not have a great deal to do with curing a case of amebic colitis. It may and probably is the best method of curing chronic ulcers or amebic ulcers, or any other kind of ulcers in the rectum and lower bowel, but when we consider the disease amebiasis, it is highly improbable that the treatment of a few ulcers in the rectum would have much to do with curing the disease, which usually extends to the cecum.

Dr. John L. Jelks, Memphis.—I find the amebae in the appendix oftentimes.

Dr. Bass—Yes, but you cannot reach the appendix or anywhere near that with the proctoscope. There is a great distance between the appendix and rectum that cannot be reached either by operation on the appendix or by the proctoscope.

Dr. Charles F. Craig, Washington, D. C. (closing the discussion).—In Dr. Mason's paper mention is made of the percentage of coli infections, and I stated that the percentage varies greatly in different localities, and I have no doubt in some localities you will find practically no infection with coli, while in others you will find a large percentage. While I was in San Francisco I examined about 100 young men, soldiers, men from all parts of the country, and found sixty per cent of them infected.

Dr. Mason—Had these soldiers been examined before going to the Philippines?

Dr. Craig—Yes; they were recruited from all parts of the United States and were examined before they went to the Philippines, and sixty per cent of them showed coli infection. It looks, therefore, as though the coli infection was pretty widespread. A point of great importance is the number of specimens examined. One ought to examine eight or ten to be sure the organism is not there. If that number of specimens be examined in every case you will find, I believe, that from twenty to fifty per cent will show coli infections. If a large number of people be examined. You will find local epidemics where people are re-infecting one another continually, and in such places you would have a high percentage.

I want to endorse the statements regarding the treatment of dysentery with ipecac. I had the good fortune to be stationed at San Francisco in the Army General Hospital when we had hundreds of cases of amebic dysentery returned from the Philippines. We gave quinine injections, and argyrol injections, and I agree with Dr. Elliott in asking, how one can expect to cure amebic dysentery by washing the amebae from the wall of the intestine, when they are also embedded deep in the sub-mucosa and the capillaries of muscular tissue?

Dr. Harris.—Did you try peroxide of hydrogen irrigations?

Dr. Craig—Yes, and with only temporary benefit. If we remember, these organisms are too deep in the tissues, the ones that are doing a great deal

of damage, for the irrigations to reach. While the irrigation treatment will do some good, and will get rid of a large number of amebae and prevent some ulceration, it will not affect the amebae situated deeply in the tissues. The ipecac does that, but how it acts we do not know.

Dr. Elliott's idea of the use of serum is a good one, but according to the recent work done by Rogers, ipecac is considered the specific against amebae. In other words, he is treating all dysentery cases by the intravenous injection of emetin, one of the active principles of ipecac, and is getting marvelous results. He is treating these cases from within instead of from without.

The relation of amebae to pellagra is one of coincidence. A large number of patients affected with pellagra will show amebae in the stools, and the amebae may aggravate the disease, but there is no direct etiological relationship, in my estimation.

As regards the method of transmission of amebic infection, the idea put forward by Dr. Simon regarding bananas is one that deserves attention. The class of people who pack bananas and handle them are apt to infect bananas with fecal material. There are a lot of people in tropical countries who are not very careful about hygiene. The idea we used to have of water being a great source of amebic infection is gradually being given up. I know when I was in Manila the water supply was badly infected, and we could cultivate amebae from the water, and a great deal of discussion arose as to whether, when we put in a new water supply there, we could get rid of the amebae by filtration. They have a new water supply, but the amebae still come through. But these amebae are not pathogenic amebae, and Walker has pointed out that if you add feces to distilled water or common water and allow it to stand, the parasite amebae cannot live for any great length of time in water. The general opinion of Walker, in the Philippines, and of Darling, in Panama, is that amebic dysentery is transmitted only occasionally by water. It is generally transmitted by direct infection of food with feces, simply contact cases.

With regard to the distribution of amebic dysentery, I agree with what has been said, that it is not confined to the South. I have seen cases originating in Illinois, Minnesota and Michigan, and they are being continually reported. Of course, we have more amebic dysentery in the South than in the North, but we have it all over the country.

Dr. E. M. Mason, Birmingham (closing the discussion).—I have been much edified by the liberal discussion on the subject of amebiasis. The time is passing rapidly, and I shall not continue the discussion except to say to my friends who oppose the ipecac treatment, that I sympathize with them thoroughly, because I felt exactly that way myself a year ago, and I believe that in time they, too, will be saved. (Laughter.)