AMEBIC DYSENTERY IN MICHIGAN.*

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The recording of a single case of amebic dysentery can be justified only by particular circumstances. In this case the geographic source of the infection is my chief reason, a secondary cause being the desire to emphasize the need of further work in this class of cases.

THE CASE IN POINT.

History.—H. S., farmer, 30 years old, was admitted to the medical clinic of the University Hospital, Ann Arbor, Dec. 6, 1901, on account of frequent loose stools, containing blood and mucus, pain in the abdomen, tenesmus and loss of weight and strength. The family history is negative. With the exception of chicken-pox, measles and diphtheria in childhood, the patient has always been well up to the present illness and continued his work until the day of admission. He states that his food and eating habits have always been good; does not use alcoholics. Drinks well water, the well being 24 feet deep, 100 feet from the barn, free from contaminations and the water considered unusually good. He does not remember having used other water within a recent period. Has not been out of the state since 1893, when he was in Chicago for a short time.

Present Disease.—The present disease began in August, 1901, with a griping pain in the lower abdomen. The stools soon became thin and watery. Bearing-down followed evacuation. There have usually been from six to twelve stools a day, sometimes as many as twenty. The stools have sometimes contained blood and mucus, but not always. They are often very small. Thinks he has not been free from diarrhea more than a day at a time since the disease began. He took treatment, the details of which are not known, but was not relieved; he grew weaker and lost about fifteen pounds in weight. The patient has no other subjective symptoms than those noted.

Examination.—Frame of medium size; muscles fairly well preserved, panniculus thin. Appearance not very ill; mind clear. Skin pale; no edema; tongue pale, faintly marked by teeth, thin white coat, otherwise negative. Examination of lungs negative. Heart negative, except soft systolic murmur in pulmonary area.

Abdomen: About an inch below level of ribs, slight fulness below and to left of umbilicus. Palpation shows a cylindric mass about an inch in diameter in this region; percussion over it tympanitic. No visible peristalsis. Examination of other parts of abdomen and spleen and liver regions negative.

Examination of rectum reveals blood-stains about the anus, a bloody and purulent fluid in the rectum. The mucous membrane of the rectum shows a number of small irregular, slightly elevated gray patches with red margins. The gray coat is very thin and can not be removed by mopping with cotton. Two thick and reddened valves can be seen.

The urine for 24 hours amounts to 700 c.c. sp. gr. 1025. Chemical and microscopic examination negative. The blood shows 4,730,000 red cells, 10,441 leucocytes per c.mm., hemoglobin 90 per cent.

A small quantity of reddish mucus was brought to me in the laboratory when the first rectal examination was made. The appearance of this was so characteristic of amebic enteritis that I mentioned the fact. At the first glance through the microscope, at a fresh drop, large numbers of Charcot's crystals were seen, and among these were several large and active amebæ, containing red blood corpuscles. The patient remained in the hospital three weeks. In this time the stools, the liquid, obtained through a speculum, from the rectum and the material adhering to a rectal tube, usually contained mucus, blood corpuscles, leucocytes, Charcot's crystals and amebæ. Sometimes there were small masses of columnar cells, or very small superficial sloughs. Eosinophile cells were rather numerous in the stools. (10 per cent. eosinophiles in the blood.)

The Amebæ.—The amebæ presented the same characteristics of size, refraction, inclusions and motion as those I reported in 1891, in Texas, and others that I have seen in dysenteric cases in other places. In the present case I measured a number of the parasites, alive, when as nearly round as possible. They ranged most frequently between 20 and 35 microns. The largest one was 38 microns. Not many were less than 20 microns. They usually contained red blood corpuscles, sometimes being as if stuffed full. The nucleus could occasionally be made out. In some cases there were highly refracting, somewhat greenish vesicles which at times showed changes of shape and size. The stools were usually soft, sometimes watery, generally small but sometimes amounting to 200 or 300 c.c. The amebæ were only found in the mucus or small sloughs.*

The number of stools was from three to ten daily, except on one day, when there was only one. The odor was more offensive in the beginning than I had usually found it in amebic dysentery. The fecal part contained many bacteria, as usual. After ten days' treatment soft, partly formed stools appeared, without mucus, with few blood-streaks, and containing Charcot crystals but no recognizable amebæ. At this time, as there was a tendency to constipation, a dose of Carlsbad salts was given. This was followed by several watery stools. In a small bit of mucus in the second stool hundreds of active amebæ were found. The superficial diphtheritic patches in the rectum cleared up quickly, but left two superficial ulcers, not undermined. These quickly healed, and by the end of two weeks the mucous membrane was less red and swollen. The thickening in the left side disappeared. The rectal tube caused much less pain than at first. The mass in the left side of the abdomen gradually disappeared. The temperature reached 100.4 the day of admission, but then fell and after the third day never exceeded 99 F.

Treatment.—This consisted chiefly in the use of quinin enemata. This was given at first in quantities of 500 c.c., all the patient could take, of one to one thousand solution. The patient retained this for one to three hours usually. Later he took 2000 c.c. of 1:2000 solution. The amebæ continued active all the time and no marked evidence of improvement could be seen in the other elements of the stools, except that the feces became slightly more consistent. Later, benzozone was given by the mouth in quantities of 1500 to 2000 c.c. of the 1:1000 solution daily. At this time we were not aware of the best method of preparing the solution, and made it up fresh every morning. There was no effect on the activity of the amebæ. The stock of benzozone then gave out and could not be renewed in time to give a more thorough trial. The patient gained 3½ pounds while in the hospital. He was then obliged to go home, where he continued the quinin enemata for three months, keeping a record of his stools and other data and reporting at intervals. He steadily gained weight and had only one or two stools a day. Stools passed in the laboratory on his visits always contained live amebæ, though the stools had partly lost their dysenteric character. March 28 he was put on sublimed sulphur, 20 gr. t.i.d. During the next month he remained about the same. Blood and mucus occasionally appeared with the stools, but the stools were less fluid. Amebæ were always to be found. After a month, April 29, he was put on 10-grain doses of sulphur four times a day. In the three weeks after that he felt better than at any time since the disease began. He had one or two stools a day and amebæ could be found in only small numbers, in minute bits of mucus.

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* The patient's blood serum was tested with a culture of the Shiga bacillus kindly sent by Prof. Flexner, but did not cause agglutination. Cultures of the stools were negative so far as the specific bacillus was concerned. These observations, as well as injections in cats and other studies, were made by Dr. W. E. Griffin, who was obliged to interrupt the work in order to appear before the Naval examining board, and being ordered to report for duty soon after, his investigations were abandoned. I have to thank him and my assistant, Dr. D. S. Grim, for many of the notes made on this case.

RARITY OF THE CASE.

From the facts detailed there is no doubt the case is one of amebic dysentery. It is the first one I have seen or heard of in Michigan, and as the local origin can hardly be doubted, is of particular interest. "Dysentery" is frequently reported in Michigan, according to the reports of the State Board of Health, but very little has been written on its occurrence here, judging from the scanty references in the Surgeon-General's index catalogue and in periodic medical literature. I have not been able to see an epidemic. Many cases diagnosed as dysentery have been sent to my clinic, and I have seen others in consultation. None had histories closely resembling any of the clinical forms of amebic enteritis, and the conditions of the stools were in general not like those of acute dysentery or of the chronic amebic form. Most cases were chronic diarrheas secondary to disease of the stomach or to acute enteritis. Some were diarrheas of pernicious anemia and autopsies on some of these cases showed no characteristic lesion in the intestines. Others could be classed clinically as catarrhal dysentery, and in one of the most severe of these, seen with Dr. George W. Palmer of Chelsea, with perforation and abscess pointing in the femoral region, there were extensive superficial ulcers in the colon, altogether different from those in amebic cases. Perhaps some of the dysenteric cases reported in Michigan belong to the specific bacillary form, the relations of which have been so admirably worked up by Dr. Flexner, but the existing reports do not enable one to be certain of that.

THE DIAGNOSIS.

Although the *Ameba coli* is a very striking object in its characteristic motion, those who have not seen it are likely to mistake for it various kinds of cells in stools. A number of these have been shown me in my laboratory by otherwise competent microscopists. The most puzzling things for the inexperienced observer are large epithelioid cells with violent protoplasmic motion in the body and with consequent movements in the sides. If one waits for the occurrence of true ameboid motion before making a diagnosis all such errors can easily be avoided, and if the stools are examined fresh, or what is often better, material obtained by passing a rectal tube, and the bits of mucus picked out, amebæ can easily be found if they are present.

HISTORY OF AMEBIC INVESTIGATION.

The finding of such a case as the one now reported recalls many interesting facts in the history of dysentery and of the amebæ found in stools. Although amebic dysentery was early looked upon as the dysentery of the tropics, largely owing to Kartulis' epoch-making observations and the striking relation of amebæ to "tropical" liver abscesses, it could not be forgotten that the parasites were first recognized in St. Petersburg (aside from the less important observations of Lambl, Lewis and Cunningham), and in chronic dysentery. I claimed, in a paper accepted for publication in October, 1890, but not printed until the next year, that ameba coli was probably a parasite having a world-wide distribution. The anatomic investigations of Councilman and Lafleur, the most important in many respects hitherto made in intestinal disease, and the discovery of the ameba in such different parts of the country as Baltimore, Texas, Philadelphia, Cincinnati, Chicago, Boston, Cleveland and Atlanta tended to fix the anatomic and geographic, as well as the clinical features of the disease. Owing to the difficulties in the way of assigning the exact etiologic relations of the amebæ, the observations just mentioned would have been of less value had it not been for discoveries in acute dysentery, both in and outside the tropics. The observations in these cases began as early as the most important ones in amebic dysentery, and culminating in the demonstrations of Shiga and Flexner, distinguish a very important group of cases, and enable us to study more accurately the nature of diseases belonging to the class of dysentery wherever found. At the same time, they have not lessened the diagnostic importance of *Ameba coli* nor made the need of further study of that parasite less imperative. On the contrary, they throw in a stronger light the close relation of the animal parasite to certain widespread, clinically important, and therapeutically difficult intestinal diseases. We must still learn the exact relation of the ameba coli to dysentery and intestinal ulceration, not to mention the remoter complications in the liver and thoracic organs. We can not as yet say whether the amebæ begin the anatomic alterations by themselves, or in association with bacteria introduced or normally living in the intestine, or whether they are nosoparasites, finding favorable seats for growth in lesions caused by other germs. That their association is purely accidental becomes less and less probable with the accumulation of data regarding the peculiarities of the intestinal changes. Direct and unequivocal proof, as by inoculation, is still lacking, and, from the difficulties in the way of cultivating amebæ, seems almost impossible. The results following the injection of material containing amebæ into the rectums of animals are not free from sources of error and have been contradictory in the hands of various investigators. The most important results are those of Dr. H. F. Harris, and, though they require confirmation, they deserve more attention than has hitherto been paid them. Harris[1] cultivated all the bacteria from dysenteric stools, but though he was unable to produce proctitis or colitis by injecting the cultures in animals, he succeeded (in young dogs) with the stools containing amebæ, even causing liver abscess, with amebæ. However, these experiments were few, and in view of the fact that Gasser produced colitis in cats by injections of garden earth, Sorgo with non-amebic dysenteric dejections, and also the fact that the previous observers have usually produced lesions not identical with those in human amebic enteritis, we must await further observations.

OPINIONS OF AUTHORS ON THE AMEBA.

The occurrence of amebæ in the stools of persons apparently well, or in those of patients with cholera or other diseases, or in the feces of lower animals, can not be considered proof that the amebæ found in dysentery have no pathogenic relation. I published an observation in 1891 showing the presence of severe ulceration in the cecum, with amebæ, in a patient without history of dysentery and with normal stools. Kruse and Pasquale pointed out very early that it was not necessary to assume the existence of two varieties, pathogenic and non-pathogenic, since the differences observed might be due to variations of disposition on the part of the host. On the other hand, our imperfect knowledge of amebæ in general, and the difficulties of distinguishing species or varieties make it highly probable that there are different kinds with different pathogenic characters.

Observations showing the occurrence of amebæ in healthy men have been much used, as mentioned above, to disprove the possible causal relation of amebæ coli, and those of Schuberg have been reiterated over and over again, and often as if they were typical of the conditions in all parts of the world. Schuberg, in brief,

1. Virchow's Archiv. Bd. 166, p. 67.

gave laxatives to persons without evidences of intestinal diseases. In the examination of "about twenty stools," he found amebæ in "about the half," "sometimes in tolerable numbers." He thought it "probable that amebæ (and flagellates, which he also found in his cases) occur as commensals in the human intestine extraordinarily frequently if not regularly."

In the first place, it may be remarked that Schuberg's conclusions hardly fit his observations; in the second place, that even if a certain parasite occurred in every case in one locality, it would not follow that the same parasite would also be found as widespread elsewhere. The observations of many early workers were opposed to those of Schuberg, also many later investigations, such as those of Janowski in Warsaw and Strong in Manila. The latter found amebæ in only eight out of two hundred cases treated with Carlsbad salts. The most recent investigator in this field, Zorn[2] in Munich, also had negative results.

My own early control observations were so different from Schuberg's[3] that at once after reading his article I began some investigations, giving Carlsbad salts in dose of one-half to one ounce, to all patients who had no contra-indication, and examining the stools as soon after they were passed as possible. More than two hundred such cases were examined, when I discontinued the routine examinations, but I have since then examined many other cases in the same way. In no case, except the one now reported and another to be mentioned below, were amebæ found. Flagellates, such as Schuberg also found in his small series of cases, I found six times in the first two hundred cases, and several times since then, but only in cases of diarrhea, never in persons with previously normal stools.

The exceptional case was one of ulcerated cancer of the rectum with diarrhea. On two occasions amebæ were found in the stools. The parasites were smaller than those I had previously seen in dysentery in Texas, the largest measuring 20 mm.; they contained no red blood corpuscles and, in fact, very few inclusions of any kind. They had a more marked tendency to travel in one direction than is common in ameba coli, and less ameboid motion.

From these observations I think it can be assumed that amebæ are not present in "about one-half" of all normal intestines, in all parts of the world. The determination of the intestinal fauna in various localities may aid in clearing up the etiology of intestinal disease and its comparative neglect is difficult to explain.

But, admitting that there are different varieties of amebæ that may infest the intestine, the question remains to be answered whether they can be distinguished other than by the effects of inoculation in animals which, as we have seen, are not altogether conclusive.

NOMENCLATURE OF VARIETIES OF AMEBAE.

The difference in size of the amebæ in my case of cancer of the rectum as compared with those in dysentery is very striking, but in other cases the matter of size has not been so easy to use. In the literature there are certain contradictory observations and the subject has been still further confused by premature efforts at nomenclature. Future investigators should avoid adding to this, but should endeavor to clear up the present contradictions. When Loesch gave the name *Ameba coli* to the organism he found he did not define the specific characters with sufficient fullness, though he did as well as the existing knowledge of protozoa permitted. Councilman and Lafleur, in giving a new name, *Ameba dysenteriæ,* without adding to the zoologic knowledge of the parasite, hardly realized the needlessness of such an innovation. The fact that so eminent an authority in zoology as Blanchard had already suggested still another name, *Ameba intestinalis,* also without greater definiteness, was an unfortunate example, but not a justification. Quincke and Roos attempted a more scientific nomenclature when they made three varieties of intestinal amebæ, *Ameba coli Loesch, sive felis,* characterized by being pathogenic for man and cats, *A. coli mitis,* mildly pathogenic for men but not for cats, and *A. intestini vulgaris,* non-pathogenic. Unfortunately, there are discrepancies or contradictions between their further description of the different varieties and the descriptions of most other investigators. Quincke and Roos described *A. coli felis* as being small, 15-25 microns in diameter and without blood corpuscles. The size of the amebæ in dysentery varies considerably according to different observers: Kartulis, 12-30 microns; Lafleur, 6-35 microns; Osler, 10-20 microns; Kruse and Pasquale, 10-50 microns; Craig, 5-35 microns, most frequently 15-25 microns. Though large amebæ seem to have been more frequently noticed than small ones in tropical cases, this has not been uniformly so and some severe cases in temperate regions have shown small amebæ with red corpuscles, those found by Zorn in Munich (1 case) measuring 14-22 microns, or about the same size as in the Cicilian case of Quincke and Roos. One of the most recent, as well as one of the most competent students of dysentery, Strong, goes so far as to believe that small amebæ found in dysenteric cases are non-dysenteric, but in giving these the name *Ameba coli,* the larger forms that of *A. dysenteriæ,* he does not seem to have proved his right to act as a sponsor any more than did Roos.

The matter of inclusion of red blood corpuscles, which has been used as a partial diagnostic feature, is still somewhat uncertain. Roemer[4] thought the presence or absence of blood corpuscles accidental, depending on the presence of blood in the intestinal contents. Sorgo[5] saw the amebæ become free from blood corpuscles, though there was still blood in the stools, in cats. In my case of cancer with small amebæ, there were no blood-inclusions, but there was very little blood in the stools at that time. The point is one that should be capable of experimental proof, and may be of more value than it now seems.

In conclusion, I wish to add that in view of the rapid advances made in our knowledge of acute dysentery in the United States, largely by Flexner and his pupils, and the fundamental work done in chronic dysentery by Councilman and Lafleur, it seems as if the inviting field offered by the study of the amebic form should attract more workers in this country than has been the case.

DISCUSSION.

DR. THOMAS MCCRAE, Baltimore—The paper just read is of special interest in reference to the question asked in Baltimore whether or not amebic dysentery is traveling northward. Several years ago one of these cases was admitted in Dr. Osler's ward, the patient never having been out of the state of Maryland and at that time this was contrary to the opinion expressed that practically all cases came from further south. Since then, however, there have appeared many cases that have never been out of the state of Maryland, where the disease is not infrequent. A point of especial interest,

2. Deutsches Archiv für klin. Med., Bd. 72, p. 366.
3. Centralblatt für Bakteriologie und Parasitenkunde, Bd. 13, 1893.
4. Münchener med. Woch., 1898, No. 41.
5. Wiener klin. Wochenschrift, 1897, No. 18

recently observed, is that an increasing number of cases are to be found in children and it has been found that some of these have occurred in children who have become infected through drinking "gutter-water." The report made by Dr. Dock has been of special interest as going to prove the wider distribution of the disease in the north.

DR. JAMES J. WALSH, New York—During the past year at least two cases of true amebic dysentery have been reported in New York City. One of the patients had not been outside of New York City for years, the other had never been out of the city. It seems, therefore, that the ameba has crept as far north in its endemicity as New York City.

DR. E. LIBMAN, New York—At Mt. Sinai Hospital we have encountered a large number of cases of amebic liver abscess. During the past three years we have examined specimens from at last ten cases of this kind in which no history could be obtained of any residence outside of New York City.

DR. DOCK, in closing—I should have mentioned in my report that I excluded any possibility of infection from soldiers who returned from Cuba or the Philippines. Since the Cuban war there have been a number of cases of tropical infections from returned soldiers. In view of the wide spread of the disease, the vague symptoms that are present and the absence of the symptoms known as dysenteric, it is very important that all stools be examined more carefully than they have been in the past. Finding the organisms early gives a better basis for making a prognosis than anything else.

WHAT CASES OF PLACENTA PREVIA CAN BE BEST TREATED BY CESAREAN SECTION?*

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It is not my intention to go over the whole subject of placenta previa and the various methods of treatment, because this has been done so thoroughly in the past two years as to be unnecessary. It is worthy of remark, however, that the importance of this condition has been forcibly brought home to the profession by the advocates of Cesarean section, and the treatment of this serious condition has taken on new interest.

At the time when I reported[1] the first entirely successful Saenger-Cesarean section for placenta previa there was little on record of value regarding the frequency and mortality in the various forms of this complication. At that time I had delivered but one case with placenta previa, and that only partial, in which rupture of the membranes allowed the head to engage, and mother and child survived, but I had in mind other cases with less happy results. All the statistics, however, that were available emphasized the serious nature of certain forms of placenta previa. The study made at that time of the literature bore out the statement made "that this was the most fatal complication with which the obstetrician had to deal, and under the most favorable conditions (meaning by that the results of well-conducted lying-in-hospitals) and in the hands of experts, showed a higher death rate, fetal and maternal, than almost any other obstetrical condition." The statistics, it is true, as later writers[2] have stated, were old, but the obstetric teaching in regard to this subject was no more modern, at least, so far as treatment was concerned.

It is not necessary to go over the entire ground on which the resort to Cesarean section in certain cases was held justifiable, but a review of those indications in the light of subsequent additions to our knowledge of the subject, will be of interest.

The indications appeared to be as follows:

1. Cases of complete previa.
2. Cases of previa in primiparæ when signs of fetal or maternal exhaustion are evident.
3. When the condition of rigid os is present.
4. Where there is a history of previous operative delivery.
5. In transverse positions and in cases of prolapsed cord, if the cord is not easily returnable.

Advocating section for these conditions is far from advocating section for all cases of placenta previa, as my paper has been quoted. We should not proceed too fast in the advocacy of new treatment, and while the surgical invasion of the obstetric field has only begun, I do not believe that section will be accepted as a cure for all obstetric complications until we admit that the pains of normal labor should be accepted as indications for interference.

The paper read by Dr. Gillette[3] at the meeting last year was a notable contribution to our knowledge of this subject, and his statistics of mortalities show that the condition is still a serious one. His report of the results of 216 cases in the hands of various men show 34 mothers lost, or a death rate of 15 1/3 per cent., and 96 children, or 44 1/3 per cent. As to the variety of implantation, there were 88 cases of centralis, 20 mothers lost (22 8/11 per cent.); while 66 children were lost (70.5 per cent.). Of the other forms of implantation (partialis and marginalis), 128 were reported, with 14 deaths for the mothers, a death rate of 11 per cent., and 41 of the children, or a death rate of 31.5 per cent.

Shauta[4] in a recent article, while arguing against section, gives the following figures: Placenta previa centralis, 50 cases, with the death of 9 mothers (18 per cent.), and 35 children (70 per cent.), while in 184 cases of partial previa he lost 7 mothers (3.8 per cent.), and 92 children (50 per cent.). These statistics do not differ from those quoted in my original paper, of Jardine,[5] who (1896-8) reported 12 complete, with 16 2/3 per cent. maternal deaths and 66 2/3 per cent. fetal, except that they are enough higher to prove that it is even more fatal than we originally believed.

In primiparæ this condition can not be said to be so rare as not to require consideration, as it occurs in about one in eight cases of previa. Fry[6] in reporting 14 cases, reports 7 cases in primiparæ.

Reason would seem to indicate that operative interference in primiparæ must be slower and more difficult than in multiparæ, because of the firmness of the parts.

It has been said, however, that the os with placenta previa is more easily dilated than if normal. This statement, however, should be changed, for there have been cases enough reported and evidence enough adduced in the past two years, to warrant the statement that the os is more easily torn than normally, while doing manual dilation and extraction; in fact, there are many cases of spontaneous rupture of the uterus in the presence of placenta previa.[7] [8]

Schultze,[9] in a discussion of uterine rupture, gives as a cause paralysis of the region of placental insertion. He refers to the fact that during pregnancy the uterine wall develops less in the portion where the placenta is attached than elsewhere, and that when the placenta is adherent, the portion of the uterine wall left after its separation is thin. Failure of this area to contract he terms paralysis of the area of placental insertion.

Anatomically no part of the uterus is more liable to rupture than the lower segment during labor, not only on account of the retraction of the muscular fibers of the uterus and the consequent thinning of the lower

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