5-1-1934

Amoebic dysentery

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A M O E B I C D Y S E N T E R Y

By

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Omaha, Nebraska
April 1934

Preface

This paper is presented to the University of Nebraska College of Medicine to fulfill the senior requirements.

The subject of amoebic dysentery was chosen due to the interest aroused from the previous epidemic, which started in Chicago last summer (1933).

This disease has previously been considered as a tropical disease, and was rarely seen and recognized in the temperate zone. Except in individuals who had been in the tropics previously.

In reviewing the literature, I find that amoebic dysentery may be seen in any part of the world, and from surveys made, the incidence is five in every hundred which harbor the Entamoeba histolytica, it being the only pathogenic amoeba of the human gastro-intestinal tract.

This disease is one of great importance and should be more thoroughly understood by the medical profession than it has in the past. One reason for this is that it is important to treat carriers, and the profession should be able to recognize these individuals, which is the all-important factor in control of the disease, and I hope that this paper will be of some aid to its readers in making a diagnosis and being able to handle this disease.

H. C. Dix.

Amoebic Dysentery

History

The Amoeba histolytica was first discovered in 1859 by Lambl (1) in the stools of a child suffering from enteritis, but he did not think of pathogenic importance. They were again noted some years later, both by Cunningham and Lewis, in the stools of cholera patients, and even in the stools of apparently healthy individuals.

The name Amoeba Coli was first conferred by Lösch, who in 1875 found the organisms in the stools of a chronic dysenteric patient, who at necropsy, was found to have extensive ulceration of the colon. He claimed for them, in part only, an etiological relationship with dysentery. He described them minutely as to shape, size, and character of motility.

Lösch attempted to produce the disease in dogs. He injected the fecal material containing the amoebae into the rectum of four dogs. In one of them eight days later he found amoebae in the stools, and in another, which was killed on the eighth day, he found rectal swelling, injection and slight ulceration.

Kruse and Pasquale also injected fecal material in the rectum of dogs and retained it by suturing the anus and they produced a condition similar to the condition found in man. They also used necrotic material

from a secondary liver abscess and produced a condition similar to that found in man which was the first proof that secondary liver abscesses contained the amoeba.

Marchaux succeeded in producing the disease in cats, those having lived over fifteen days developed secondary liver abscesses, "usually single".

Robert Koch was the first man to really give definite knowledge of its parasitic properties. While investigating cholera in Egypt in 1883, he found the amoeba in the deeper portions of the ulcers in the intestines of several cases of dysentery, and because of their deep position, he looked upon them as exciting a causal relationship. It was his influence that caused Kartulis to study them.

Kartulis in 1885 started his work on the disease. He found amoebae in the stools of five hundred cases of dysentery in Egypt, and also found them in the necrotic contents and wall of liver abscesses complicating the disease. He observed, too, that their number was seemingly related to the severity of the attack. He also failed to find amoebae in other cases of dysentery other than amoebic dysentery. They noted that amoebae were found in patients of other diseases such as cholera and also apparently normal individuals.

Osler, in 1890, was the first man in the United States to report the finding of amoebae in the stools and hepatic abscesses of a case of amoebic dysentery. His patient came from the Panama Canal Zone.

The finding of amoebae in stools of normal individuals and of those ill with other conditions than dysentery, and the fact that rectal injections in animals sometimes failed to produce dysentery in them, led to the question whether the amoeba was really pathogenic or whether more than one variety existed, one type being pathogenic and the other non-pathogenic.

Several observers had reported the finding of amoebae which were larger than that described by Lösch as Amoeba Coli, and presenting a different encysted form. Quineke and Roos were among the first, who endeavored to show that variations doubtless exist. With one type they succeeded in inducing, in cats, both dysentery and liver abscesses, while the amoebae from another source failed absolutely. They also describe an intermediate type which caused mild intestinal symptoms when injected by rectum into cats. This led them to name these Amoeba Coli, retaining the name by Lösch to that type inducing most severe results, and Amoeba Coli Mitis, the mild variety and Amoeba Coli Vulgaris, the non-pathogenic type. These later they obtained

from the stools of normal individuals after the administration of carlshad salts, finding amoebae in 9 out of 24. Schulberg found them in 10 out of 20 in liquid stools of normal individuals, but failed to find them in normal individuals.

Calandruccio swallowed amoebae and though he continued to find them in his stools for some days, no marked condition resulted.

Then Councilman and Lafleur (3) started to change the previous classification and put them under pathogenic and non-pathogenic types. The pathogenic form was termed Ameba dysenteriae and the non-pathogenic, Amoeba Coli. This classification lasted up until the time of Schaudinn in 1903. After his experiments he termed the non-pathogenic type of amoeba "Entameba Coli" and the pathogenic "Entameba histolytica" and this term has been unchanged up to the present time. Soon after Schaudinn had printed his paper and given the new classification, Viereck, in 1907, described a third form which was called Entameba tetragena, but not until 1913 was the identity of Entameba tetragena and Entameba histolytica brought out. So by this it was concluded that there was only one pathogenic form and this was Entameba histolytica. The family history is given by both Schaudinn and Viereck.

Due to the work of Walker (2) in 1911, and Mathis in 1913, and many others, we have been lead to turn

from the trophozoite form to the study of cysts which was described at this time.

Rogers, in 1912, introduced the use of emetine. Emetine had been used many years before. In 1891, Tull, Walsh and Warden had found the total alkaloid of epecacuanha. The emetine of that period cured cases of dysentery which were uneffected by epecacuanha deprived of the alkaloid emetine, but this was forgotten for some time, until when amoebic dysentery was differentiated from bacillary dysentery. Then it was rediscovered. This was impressed when Vedder observed that the growth of free-living amoeba was inhibited by high dilutions of emetine and cephoeline, and when Rogers described the immobility and death of Entameba histolytica obtained from dysenteric stools, by contact with a solution of emetine hydrochloride one part to one hundred thousand.

In the years from 1912 to 1916 there was considerable progress made in the identification and classification of the different types of amoebae.

In 1919 to 1920 Dr. Craig (5) worked out the complement fixation reaction which was of some aid in diagnosis.

In 1921 an article was published by Kofoid and Swezy (6) on the effects of amoebiosis on arthritis, therefore considering the disease as a systemic disease.

There has also been some recent work and many publications made since the recent epidemic in Chicago, which has made the United States amoebic minded. Many new staining processes have been developed and culture medium has been changed, but in trying to culture the parasites, it has been very difficult to get growth.

Epidemiology

Amoebic dysentery is characteristically an endemic disease, which is primarily of the tropics and subtropics, but at the present time, it is known to be where every man may live. The cysts are undoubtedly the propogative stage of the entamebae. It is always necessary for a parasite to be able to leave a host and then re-infect a new host without being killed. The cyst being quite resistant, carries out the external part of the life cycle of this parasite. No multiplicity of the cysts or trophozoites takes place out side of the host (7). The trophozoites degenerate very rapidly outside the host, and if the trophozoite is taken into the host by mouth, it will not set up any infection due to the fact that it can not resist the digestive action of the secretions of the stomach. The cysts being quite resistant are not destroyed when taken in with food, but set up an infection very readily. In some cases symptoms develop, while in others no symptoms appear, but virulent cysts are passed in the stools. In this way the individual becomes a very serious menace to society.

In handling dejecta of an amoebic dysentery patient it should always be destroyed in a manner which will kill all trophozoites and cysts. The examination of the stool may show only the vegatative forms, but you never know when cysts will appear in the stool in large quantities.

The causative factor producing excystation of the vegetative form is unknown. Certainly warmth and moisture are necessary, nutrient matter and enzymes, singly or in conjunction with each other, may be essential factors. The life cycle is of great value in showing the infective stages.

Life cycle of Entameba histolytica (8). It is comparatively simple, consisting of two distinct stages. The trophozoites being the vegatative stage. They multiply and are motile, being found in the intestinal tract, producing lesions and symptoms of the disease.

The motile forms are passed in large numbers in the feces, but are destroyed in a short time after leaving the body, and when ingested, are destroyed by gastric juice and secretions of the jejunum. In the course of an established infection, conditions unfavorable for growth eventually develop. The exact nature of these unfavorable conditions is still obscure. The trophozoites cease to ingest food, "which is red blood cells to a great extent", round up, become motionless and filled with glycogen. The size diminishes and newly formed cysts containing one nucleus are found. This signifies the immature cysts. Then the nucleus divides and two nuclei are present, and the two nuclei divide once more giving four nuclei. This is a mature cyst. Many of the cysts seen in the feces are immature.

They vary in size from about seven microns to fifteen microns.

As previously stated, the propagation from one host to another is entirely dependent upon the cysts. When the acute symptoms subside, some of the trophozoites in the colon usually undergo encystment. These cysts pass out in the feces and these cysts are quite resistant, but when ingested by mouth, excystation occurs in the small intestine and colon, a four nucleated amoeba emerges from the cyst or four small amoeba. "This is not definitely known." These amoeba are arrested at points of stasis in the large bowel. They penetrate the mucosa and form the typical lesion of amoebic dysentery.

Dr. Hegner (9) while in Panama did many very interesting experiments on the life cycle of the Entameba histolytica. He and his associates worked with the trophozoites of Entamoeba histolytica from human cases of acute intestinal amoebiosis.

He gives a new classification of the different types of Entameba histolytica based upon morphology and locomotion. Type one is the tissue invading amoeba, two is the precystic amoeba, and third the cysts.

He describes the tissue dwelling amoeba as occur in the mucosa, submucosa and even in the muscular layers.

The term Lumen Amoebae is proposed for a second type which lives in the lumen of the intestine. It is

usually assumed that amoebae enter the tissue where they multiply rapidly. Some of the offsprings pass out into the lumen where they become the precystic type and are evacuated in the feces.

The tissue dwelling amoebae are located at the base and the sides of the ulcers and not in the exudate and none were seen in the material on the way out of the ulcers. It seems hardly possible for the enormous number of amoebae in the lumen to originate from the comparatively few tissue dwelling amoebae.

In carriers where there are few is any tissue dwelling amoebae, so Dr. Hegner thinks, there is reproduction of the trophozoites in the lumen and not all amoibae are from tissue dwelling amoebae.

Precystic amoebae are lumen amoebae that for some unknown reason, "probably some unfavorable condition", has been stimulated to encyst. A few precystic amoebae may originate in the tissues, but most of them are from the lumen amoebae.

The cysts are of particular interest to those engaged in public health work because they are recognized as the effective stage that may set up an infection when swallowed by a susceptible host. A number of problems involving cyst were studied by Hegner, which included their development outside the body, and places where excystation occurs. The time required for excystation and the excystation of immature cysts.

Development of cysts outside the body. Material from a stool containing large numbers of cysts of Entameba histolytica was fexed and injected into the intestine of monkeys at definite intervals, and this showed evidence that either a differential destruction of cysts occured or else development from uninucleated cysts.

Time required for excystation. Entamoeba histolytica cysts were injected into monkeys orally. One monkey was killed in two hours and twenty minutes, a second in two hours and a half, and a third in three hours. Excystation had begun in the first two, but was well under way in the third monkey. This shows that excystation occurs in about three hours after the cysts are taken into the gastro-intestinal tract.

The place of excystation is largely in the small intestine and also in the proximal portion of the colon. Hegner could not get excystation to occur by rectal injection of monkeys, as was reported in kittens by Sellard and Theiler in 1924 and Hoore in 1925.

The process of excystation. The first sign of excystation is the movement of the organism within the cyst, then a break occurs in the cyst wall and part of the cyst flows out. The cytoplasm flows in again and out and in a number of times before the organism actually escapes from the cyst.

The excystation of immature cysts. Amoeba carriers frequently pass large numbers of cysts containing only one or two nuclei. It seems certain that cysts in any stage of development are capable of excystation and hence infective to susceptible hosts. It is not known if these cysts reach the stage of maturity or not before excystation, but it has been proven that a host can become infected from the ingestion of immature cysts.

Much experimental work has been done on this subject, and there is evidence to show that excystation may even occur in the descending colon. But many men argue against this so there is no definite proof as to the exact location of excystation.

Immunity. The infection of the Entamoeba histolytica affords no resistance to a re-infection. The patients are prone to relapses. (10) Although there seems to be some racial resistance to the complications of the infection, it has been noted that in the native people of the tropics, there is much less liver abscess in per centage of cases than there is in the people of the temperate zone. The reason for this is unknown unless the natives have acquired some natural immunity over the centuries that this disease has been prevelant in the tropics. The disease is much more prevelent in the temperate zones in recent years, probably due to

the rapid transportation and the much more intermingling of races in the last few centuries.

Prophylaxis. There is no method of specific protection against amoebic infection. The personal measures depending upon individual cleanliness and hygienic precautions are important.

The prophylactic measures have become a great problem of the public health departments. They have the problem of proper disposal of sewage which is a great factor in the protection of the people, also in the proper installment of plumbing as was brought out in the Hotel in Chicago this summer. (11).

Another very important measure in prophylaxis is the routine stool examination of all food handlers in public boarding houses. The falacy in this is that many cases of amoebiosis which present no symptoms will not show cysts in all stools. Therefore, they should be examined at least once every three weeks for five consecutive times.

In cases of dysentery with acute symptoms, the stools should be burned or sterilized with some caustic before disposed of. All cases of this type should have periodic stool examinations for a year before a cure is pronounced.

Morbid Anatomy

The gross appearance which the colon presents in all the cases of amoebic dysentery varies considerably in character in different cases and even in the same intestine. There are certain features which are met with in all cases. The most striking characteristic in all, and the area which attracts the attention to this as a special anatomical form of dysentery, was the great thickness of the intestine. This is present in every case, being much more marked in some cases than in others. Sometimes the thickening involves all the coats, while in other cases it is confined to the submucosa, and is always most marked in the submucosa. There is not only a general thickening due to edematous conditions, but there are found sharply circumscribed projecting nodular thickenings filled with gelatinous looking pus. These cavities communicate with the surface of the mucous membrane by a small opening. There are also sinus tracts, sometimes representing an extension of the cavity, and sometimes communicating with neighboring cavities of the same type. The elevated nodules varied considerable in size and the openings were frequently no larger than the head of a small pin, or so large that the cavity was freely exposed. Even then the surrounding mucous membrane was deeply undermined, and there were often found sinuses in the sub-

mucosa leading off from the ulcers.

These ulcers were filled with the same glairy, gelatinous-looking material as in the cavities. This can be lifted out and leave the ulcer clean, but sometimes portions of it may still cling to the wall.

The microscopic examination nearly always contains numbers of amoebae, red corpuscles, very round swollen cells and pus cells. These large round cells have a single nucleus. They are sometimes simply granular, others are filled with fat drops. They are probably enlarged connective tissue cells which have become free by softening of the tissue around the cells. The whole material is quite tenacious.

The undermined ulcers in connection with formation of cavities and sinuse tracts in the submucosa can be regarded as the form most frequently seen. They are found in the same intestine in connection with other forms of ulcers, and in some cases are much more prominent than others. They are most commonly seen in the transverse and descending colon.

In every case observed by Councilman and Lafleur, the submucous coat was the most affected. This was infiltrated and edematous, not only in the neighborhood of the ulcers, but in places which were free from ulcers. The ulcers increased in extent by the gradual infiltration and softening of this tissue with subse-

quent necrosis of the tisue immediately next to it. The roof which covers the more or less closed ulcer is broken through. The infiltration and softening continues at the sides, and an ulcer with deeply undermined edges is formed. The base of these ulcers are cleaned and formed by the muscular coat. Although the ulcer advances in the submucosa by gradual softening and disintegration of the tissue, the muscular coat offers a barier to this cellular distruction. The upper layers become necrotic, the cellular infiltration extends into the muscle along the connective tissue between the bundles of muscular fibers, but it holds together. The infiltration gradually extends through into the intermuscular connective tissue septa and the subserous coat, and the same process is repeated here as does in the submucosa. The circular muscular layer is destroyed and the vessels supplying this area are necrosed.

Kofoid and Swezy published an article in 1925 showing sections of the colon adjacent to an ulcer, outside the muscularis in the capillaries and small blood vessels of the serosa, abundant evidence that the amoebae have entered the blood stream and are thus in a position to find their way in the hepatic portal system to the liver. They also showed the trophozoites in the area around the ulcers. Dr. Boeck had the op-

portunity of observing several progressive stages in the early invasion, as well as the gradual and ultimate destruction of the lymph follicles as afforded by lesions occuring in Peyer's patches of the terminal ileum of five kittens. These amoebae showed an apparent selective action in attacking the follicles, as was evident from an initial necrosis of the epithelium covering the tip of the follicle. The adjacent epithelium of the intestinal mucous gland was not affected. Following the destruction of the epithelium over the follicle, the amoebae moved down into the follicle, and the sinus tract was formed.

The lymphoid cells of the follicle and the interstitial cells of the reticulum underwent necrosis. The nuclei became pyknotic and the cytoplasm underwent dissolution. There was practically no evidence of any cullular reaction to the infection in the early stages of the invasion, but later stages showed an infiltration of the submucosa at the outer limits of each follicle with plasma cells chiefly, scattered polymorphonuclear, large mononuclear, and a proliferation of connective tissue elements attempting to wall off the lesion. In the center of the gland there was progressive destruction of the lymph cells by the amoebae, resulting in much cellular detritus.

The amoebae, as they multiplied and formed a local abscess, appeared as large cells. The abscess thus produced continued to grow until the whole follicle was destroyed. Later the mucosa overlying the abscess in the submucosa may, in some cases, slough off and produce a large deep ulcer.

The follicles in the colon also showed early invasion by amoebae. This may go on simultaneous with the invasion with the erosion destruction of the mucosa adjacent to the follicle or elsewhere, depending upon location of the amoebic invasion. This invasion of lymphoid follicles was also noted in the colon and ileum in human cases reported by Councilman and Lafleur. Kuenen was the first to report a case which died of perforation of the ileum by amoebic invasion of Peyer's patches.

Kofoid, Bayers and Swezy, in 1922, reported several cases of Hodgkin's disease confirmed by pathological examination of excised glands. These glands contained cells which supposedly were Entamoeba histolytica. They also report finding and demonstrating amoebae in several cases of arthritis deformans.

Symptomatology

The onset of symptoms of amoebic dysentery may be very sudden or very insidious.

In the symptoms usually following an acute attack the patient usually come under observation in the following condition: Slightly emaciated and blanching of the mucous membranes, muscular weakness, decubitus indifferent, expression dull, sensorium clear, skin often dry and inelastic, distinctly sallow, tongue pale and flabby, moist and, more or less, fevered. The abdomen is of normal appearance or retracted, temperature not usually above 100° F., often being normal, pulse ranging from 70 to 90, respirations from 18 to 30, appetite impaired, and sleep disturbed by more or less frequent evacuations of the bowels. (13)

A departure from or accentuation of certain features in this clinical picture is observed in grave or in chronic dysentery. In the grave situation, the patient is more or less prostrate, the face is drawn, slightly cyanosed or flushed and the expression anxious, mind usually quite clear, almost complete anorexia, intense thirst and sleeplesness, abdomen greatly retracted and there may be free sweating. The temperature is frequently normal or subnormal. The pulse is small and rapid and respirations proportionately accentuated.

In the chronic dysentery, the progressive anemia and loss of flush are special features, which dominate

the intestinal symptoms. The skin is dry, harsh and of a dull grayish-yellow color.

The anemia which is present in all cases of dysentery, deserves special mention. The defeciency exists both in the corpusclar elements and in the hemoglobin in about the same proportions. It is, no doubt, to be attributed partially to direct loss of blood in small quantities from the intestinal tract and the rest is attributed to mal-nutrition, but this does not explain the anemia occurring in chronic cases where the appetite and nutrition is comparatively good and has not lost any appreciable blood by rectum. So it is considered that it is partly due to the ingestion of corpuscles by the amoebae.

Diarrhea in some cases is the principal and only feature of disease. It is subject to great variation in character and frequency. The occurrence of intermission and exacerbation has been noted as very characteristic. This is the special characteristic of the disease.

The exacerbation often begins suddenly and subsides in the same manner, lasting from one to two days, up to a week to ten days, and are progressively more severe and longer duration in fatal cases.

The intermissions have a wide range of duration, from one day to three weeks, and during this time the

feces are soft or even formed, but mucous is usually adherent to them. It has been observed that the intermissions and exacerbations were most marked in cases complicated by liver abscess.

The involvement of the liver does not coincide with any exacerbation of intestinal symptoms. On the contrary, as may be seen by reviewing case reports, there is at this time either a continuous diarrhea of moderate severity or slight loseness of the bowels alternating with constipation, and exacerbations occur many days after there was evidence of hepatic or pulmonary involvement.

In Gangrenous dysentery, they may at first number thirty to forty bowel movements in the first twenty-four hours, but subsequently the decline to from twelve to eight or even to three or four at the end of a fatal case. This may be due to a gradual loss of expulsive power in the bowel.

The amount voided at each movement of the bowels is at first small and often consists entirely of clear masses of mucous mixed with more or less bright bloody and occasionally small fecal masses. As ulceration advances the stools become more copious, watery, and less homogeneous, blood is less frequently observed and small shreddy masses of a grayish or light yellow color appear, mixed with blood-stained mucous.

When extensive sloughing takes place the character of the stools is even varied. They are of a grayish, greenish (resembling spinach or green scum seen in stagnant water), reddish brown or variegated color. Sometimes quite liquid, at other times pultaceous, and have a very penetrating offensive odor, mixed with finely divided shreddy detritus, mucus and streaks of dark blood. There are seen larger, tough stringy masses of necrotic tissue of a grayish or yellowish brown color. No characteristically purulent stools are passed, but the some what slimy, gray, liquid movements contain more pus cells than any other.

In dysentery of moderate severety of abrupt onset, the stools are for a week to ten days similar to that of gangrenous dysentery. If the onset has been gradual liquid brownish-yellow stools containing mucus, streaks of blood, and many of the gelatinous grayish masses are found. Of this type there are usually four to ten stools in twenty-four hours, and a flux of this degree, but more irregular and without blood may continue for weeks.

In stools of chronic dysentery, they have a more homogeneous appearance. They are watery or of the consistency and appearance of a thin gruel and of an earthy or a dull yellow color, and contain few or many particles of clear mucus. During an acute exacerbation, blood and greenish, pultaceous material may be seen. In intermissions the stools may be formed or soft, but also a pres-

ence of mucus.

The reaction of dysentery stools is usually alkaline which is thought to be due to protein putrefaction.

Abdominal symptoms: Abdominal pain is the most constant symptom. It occurs most frequently in the early stages of gangrenous dysentery and dysentery of moderate severity with an abrupt onset with acute exacerbations, with the subsidence of the acute diarrhea, the pain decreases in severity, but, as it often disappears entirely as gangrene progresses, this does not have a prognostic value except in conjunction with other favorable symptoms.

In chronic cases, severe colic is not complained of, except during exacerbations, but this is often a dull aching or burning pain and a sensation of weight about the epigastrium. The pain is described by the patient as cramp-like, teasing or sometimes burning. It usually precedes or accompanies the movement of the bowels, when it is severe it is general, but subsequently is localized, usually to the lower abdomen.

Sensation to pressure is present in many cases. It is elicited most frequently by pressure along some part of the course of the colon.

Tenesmus. Great importance seems to have been attached to this symptom, which is considered patho-

manic to some doctors, and, no doubt, is so of catarrhal dysentery and of most cases of diphtheritic dysentery. Dr. Councilman and Lafleur's observations in the amoebic form of dysentery, however, coincides with those of Dutraulu who noted the infrequency of tenesmus in an extended series of cases of tropical dysentery. In Drs. Councilman and Lafleur's series of cases tenesmus was noted in four cases and it is to be observed that three of these were gangrenous forms of dysentery, which is again in accordance with Dutroulu, that tenesmus occurs chiefly in grave cases in which there is extensive sloughing, burning sensation in the rectum and anus during and after passage is very generally complained of.

Nausea and vomiting occur especially at the onset, but occurs in many instances at irregular intervals during the course of the illness. The ingestion of food and in the case complicated with liver abscess and lung abscess, paraxysmal coughing may occur.

Elevation of the body temperature is not a prominent feature in this form of dysentery, which in this respect is similar to other forms.

It is, never the less, true that if careful observations are made, an elevation of temperature above the normal point will be found at some period of the twenty-four hours daily throughout the period of the illness.

Exceptions to this must be made in severe gangrenous dysentery, in which the temperature is sometimes normal or subnormal for twenty-four hours or more, and in chronic dysentery, which may be absolutely afebrile for days or weeks.

An exacerbation of diarrhea may be accompanied by slight increase in fever, but is not at all constant. The accompanyment of liver and lung abscesses produce an elevation of temperature. The temperature curve is irregular. There may be alterations of continuations, remittent pyrexia throughout the illness and this occurs independently of any complications.

As convalescence is established, the fever declines through slight irregular elevations of temperature have been seen even when diarrhea is absent. The average range of temperature in uncomplicated cases is from 99°-101° or 102° F., and evening temperature is usually higher than morning temperature.

Rigors do not occur in cases which are uncomplicated, such as cases of liver abscess.

Sweating is observed in all abscess cases and in gangrenous dysentery is often associated with a subnormal temperature and feeble circulation. In moderate cases of dysentery it is seldom seen. In chronic cases the skin is persistantly dry.

Circulation and respirations are affected in the

same degree that they are in other diseases attended by moderate pyrexia. With the abrupt onset there is accentuation of the pulse which ranges from 80 to 100 and is full and regular. The most common range throughout the disease in uncomplicated cases is from 80 to 90. In grave cases, 100 to 110, while in chronic cases the pulse rate only occasionally exceeds the normal. With progressive exaustion in the fatal cases, the pulse becomes feeble, compressible and more rapid, ranging from 120 to 140.

The respirations are increased in ratio with the pulse. The condition of the circulation and respiration in cases complicated by hepatic and pulmonary abscess is considerably more rapid.

The urine is usually slightly affected in all cases, having some albuminuria. There is usually no disturbance in kidney function in cases of chronic dysentery. In gangrenous cases, there may be retention, quantity very slight, and urine highly concentrated.

Complications

Abscess of the liver is one of the most common and serious complications of amoebic dysentery. It developes in about twenty-two per cent of the cases of amoebic dysentery. It is much more frequent in males, more common in foreigners to the tropics than in the natives, and rare in children under ten years of age. It may develope at any time during the course of the intestinal infection. Not uncommonly it occurs after all symptoms of dysentery have ceased for a long period of time, or sometimes before any noticeable intestinal symptoms have developed. In some fatal cases of liver abscess there have been no evidences of intestinal lesions at autopsy and no history of dysentery during life. A history of dysentery may, however, be obtained in about sixty to ninty per cent of the cases according to some statistics. In the majority, the abscess becomes evident in the first month after the onset of the dysentery (14). The use of alcohol in cases having amoebic dysentery, seems to be a predisposing factor in liver abscess as well as other dietetic excesses and exposure.

The most common seat of the abscess is the upper and posterior portion of the right lobe of the liver. In the greatest majority of cases, only one abscess is found, but multiple abscesses are seen in some cases.

Rarely, as many as two or three hundred small abscesses may be found. The amoebae usually invade the liver by way of the portal vein and these parasites are frequently found lying in the veins of the submucosa and in the portal capillaries and veins. In about half of the abscesses bacteria may be obtained by culture. The remainder are sterile in regard to microorganisms. The small amoebic abscesses consist of thick glairy, yellowish masses of mucus which are not fluid. In the large abscesses the contents are more liquid and of a creamy, gelatinous, purulent consistency. In color they are yellowish, grayish red, brownish red or greenish from the adjacent mixture of bile. Shreds of necrotic liver tissue are mixed with the fluid portions. Microscopically, one is struck usually with the absence or presence in a small number only, of polymorphonuclear leucocytes. The contents consist of granular material, consisting of fragments of cells, swollen degenerated liver cells, red blood corpuscles, fat globules, cholesterin crystals and amoebae. The amoebae are sometimes difficult to find in the pus, but can almost always be found in scrapings from the wall of the abscess.

In abscesses where no bacteria are found, you find that the amoebae have caused necrosis and liquifaction of the tissues without any pronounced inflammatory reaction.

In the very early abscesses you may find liver cells still present. There is edema of the surrounding tissue and there are a few mononuclear phagocytic cells in the neighborhood.

Symptoms and Diagnosis of Liver Abscess. (15)

The condition may develope very slowly and for this reason is frequently overlooked, and perforation may be the first indication. In liver abscess there is no single symptom that is constant proof that the liver is involved. The general condition and appearance of the case may lead to the diagnosis, which sometimes is confirmed by aspiration. A history of previous diarrhea may suggest the condition, but an absence of such history can not exclude such a diagnosis of liver abscess.

If the onset is acute the diagnosis is made much more readily. Rogers recognizes a stage which he terms pre-suppurative stage of amoebic hepatitis in which amoebae from dysenteric lesions have lodged in the portal capillaries of the liver, but actual abscess has not taken place. There may be a low remittent fever and a leucocytosis in which the polymorphonuclear leucocytes are increased very little in per centage. He thinks at this time the administration of emetine may prevent liver abscess formation. In differentiating

between the pre-suppurative and suppurative is often very difficult without puncture and this sometimes is very unsatisfactory. If chills and sweating are present and the condition does not improve by emetine, suppuration may be expected.

Fever, pain, enlargement and functional disturbances are the more frequent indications of liver abscess. Fever is usually present at some time, but is often insufficient to attract the attention of the physician. Sometimes it is irregular, from 99° to 102° F., and it may be of a septic type rising in the evening to 103° to 104°. Chills and fever may occur and the symptoms resemble, more or less, malaria. Sometimes the conjunctiva shows a slight amount of jaundice, and persistent vomiting may occur (16).

In certain cases the facies may suggest the diagnosis; emaciation occurs in some cases, and the appetite is poor and the tongue becomes coated and fuzzy.

Pain is a variable. It may occur in the right shoulder when due to irritation of branches of the phrenic nerve, or over the hypochondrium or epigastrium. When not present spontaneously, it may be elicited on pressure over the liver. Enlargement may be detected by physical examination or by x-ray. The enlargement is usually upward and may reach as high as the angle of the scapula, or it may progress so as to cause a

bulging on the right side, a swelling may also be seen over the sixth and seventh rib in some extensive cases.

The movement of the right side of the chest during respiration may be limited and the right rectus may show some rigidity. Percussion and auscultation frequently give no information of the condition. You may get an increase in hepatic dullness.

Functional disturbances may also be associated in making a diagnosis of liver abscess. When considerable destruction of the hepatic substance has taken place, the area of excretion may be diminished. Stitt believes that the most specific test for liver function is to be that of urobilin. The per centage of nitrogen eliminated as ammonia may also be increased in diminished liver function. An absolute diagnosis can often only be made by aspiration and finding the Entamoeba histolytica in the pus.

Lung abscess is next in frequency to liver abscess. It may be secondary to a liver abscess developing from direct extension, or it may occur primary. The amoebae entering the lung through the hepatic veins. The abscess is very similar in character to that of the liver. The formation of abscess may be preceded by an irregular fever and an irritating cough. Respirations are frequently increased in number and shallow. Pleursy usu-

ally precedes perforation. Cough appears and expectoration appears. When the abscess ruptures into a bronchus, the respirations become less frequent and a definite sputum is found. It is called anchovy-sauce-like sputum. The abscess may continue to discharge for years or death may occur in short time. In favorable cases, it may clear up in a few months, but the prognosis in these cases is very much guarded.

Peritonitis. Peritonitis is a quite common complication in amoebic dysentery. Local peritonitis may result from extension of inflammation from the ulcerations in the intestinal wall until the peritoneal coat is invaded with deposition of lymph, fibrin, and other inflammatory products on the surface. Patches of fibrous adhesions are frequent in chronic cases. These may cause abdominal soreness and pain.

Peritonitis which generally proves fatal may follow perforation of a liver abscess or of an intestinal ulcer. Perforation of an ulcer usually results from a deep sloughing ulcer. It has occurred in the cecum and has been mistaken for appendicitis. Perforation occurs in only about three to four per cent of the cases which are hospitalized. It is almost always fatal when it occurs. Perforation may also occur retroperitoneal and affect the psoas muscle and abscess will point in the inguinal region as a psoas abscess.

Amoebic appendicitis has been identified, but is very difficult to make a definite diagnosis in the living. It is usually an extension from the cecum (17). Brain abscess is a very serious complication and is always fatal. There are many cases reported in the literature. Kartulis reported several cases found in the literature.

In reviewing case reports, all the cases but one had occurred in the cerebrum either in one hemisphere or the other. The abscess are usually single, but may be multiple. The fluid in the abscess appears similar to that of the liver. It is usually reddish brown and contains necrotic brain tissue and thrombosed blood vessels attached to the walls. The meninges are seldom affected, therefore, a lumbar puncture will yield a clear fluid. The toxic evidence of suppuration are not prominent and there is usually little or no evidence of intracranial pressure. The disease proceeds rapidly and death occurs usually within six to ten days after onset. Patient loses consciousness and coma rapidly develops. If the abscess ruptures into the ventricle the course is very acute.

Convulsions may occur and the temperature may rise, Some cases go into a sudden mania, others develop coma without any previous symptoms. Cases have been reported

where Jacksonian epilepsy was the first symptom. The usual methods used in diagnosing brain abscess or tumor is much help in some cases.

Arthritis deformans has been recently worked out and reported by Eby, Reed, Wycoff and Kofoid (18) as a complication of amoebic dysentery. They have reported the finding of amoebae in the joints of cases with arthritis deformans during amoebiosis. This has not been generally accepted by the medical profession as yet, but is a subject which is well worth considering.

Etiology and Diagnosis

The diagnosis of amoebic dysentery is very difficult in some cases, and many times it is only made in the laboratory. There are many other forms of dysentery which resemble the clinical picture of amoebic dysentery very much. It is possible to differentiate bacillary from amoebic by the more sudden and acute onset of the bacillary type together with fever and other signs of toxemia. The pulse rate is definitely more rapid in the bacillary type than in the amoebic type. The number of stools being more frequent and less in amount. (19).

The diagnosis (20) in any case of amoebic dysentery should be confirmed by the finding of the amoebae in the stools, or the typical four nucleated cysts which are characteristic of the Entameba histolytica. The examination of the stool should be made as soon as possible after the stool has been passed. If the stool is allowed to stand the amoebae soon die or disentegrate and are not found on examination. If you want to keep the amoebae living you should have a heated substage which keeps the amoebae alive and quite actively motile. In studying the living amoebae you must have them alive and motile, then they will not be mistaken for a phagocytic cell. After the amoebae have been demonstrated to be alive and active, the clinician should be able to differentiate the three most common amoebae found

in man. The distinction between these amoebae is very difficult in many cases and the experience of the clinician is very necessary. The most popular belief of the men having a great deal of experience in the observation of amoebic dysentery say that, Entamoeba histolytica is the only one which ingests red blood cells, and when an amoeba is found to have ingested red blood cells it is quite conclusive that it is Entameba histolytica. Many men debate this previous statement and say that Limax and Entamoeba Coli will also ingest red blood cells.

The distribution of the chromatin in the nucleus is frequently of aid in differentiation. The examination of cysts in stained preparations gives more accurate results in the determination of species.

In staining the cysts, there are many methods. Schaudinn's method which is an alcoholic sublimate iron hematoxylon. Donaldson's method is most successful; he used iodin eosin solution. This should be freshly prepared. It is made up of a saturated solution of eosin in normal salt, two parts; five per cent potassium iodide in normal salt solution, two parts. The smear is prepared by rubbing out a minute portion of the feces by rolling it around an applicator stick in a small drop of normal saline solution, then add a drop of the iodin eosin stain. The cysts stand out

clearly as bright spherules which soon become tinged with the iodin to varying tones of yellow, while their glycogen filled vacuoles, when present, turn light or dark brown according to their mass. The nuclei become more clearly defined as the iodine penetrates, especially in Entamoeba histolytica and Entamoeba coli. In the Endalimax nana they are detected with difficulty. The cysts of Entamoeba histolytica measure from 5 to 20 cc. on the average. They are spherical or ovoid, and the cyst wall is clear and perfectly smooth and is formed of a single layer.

When the cyst is first formed it has only one nucleus and it measures about one-third the size of the whole cyst.

The cysts are passed in the feces in the uninucleated, binucleate and quadranucleate stages.

The cysts of Entamoeba coli measure from 10 to 30 cc. or more. The cysts containing one, two, four or eight nuclei may occur in the stool, the cysts containing eight nuclei when mature. It is considered that 80 per cent of the cysts found in the stool have eight nuclei.

The mature cysts of Endalimax nana are typically oval and measure usually from eight to ten microns in length and seven to eight in width. Kofoid and Wenyon believe that the structure of the nucleus of Endalimax

nana when stained is an absolute diagnostic criterion of this species.

In Entamoeba histolytica there is a central karyosome and the peripheral chromatin is scattered over the nuclear membrane in granules of a small size, whereas, in Endalimax nana there is no central karyosome and the peripheral chromatin is massed in a single large clump at one point on the nuclear membrane.

Hegner and Cort (21) made out the following table in 1921 which differentiated the three main types of amoeba in man:

Characteristics	Entamoeba Hist.	Entamoeba Coli.	Endalimax nana
Unstained alive Size	Small 5-30u	Large 10-30u	Very small 5-15u
Ingestion of corpuscles	common	rare	rare
Shape in motion	One pseudopodium protruded in direction of movement.	One pseudopodium protruded in direction of movement.	One or more small pseudopodia at one time.
Locomotion	Active, pseudopdia protruded explosively.	Sluggish, pseudopodia formed by flowing out slowly.	Sluggish in formed stool, active in liquid stool.
Appearance	Ectoplasm is strongly refractile, endoplasm finely granular.	Endoplasm small in amount; less refractile; endoplasm vacuolated.	Endoplasm very clear and refractile.
Stained in iron hematoxylin	Chromatin: a small central granule and many small granules on nuclear membrane.	Chromatin: a larger central granule often eccentric, and many large granules on nuclear membrane.	Chromatin: no central granule, large mass and one or more smaller masses.

In many cases of amoebic dysentery it is very difficult to demonstrate the protozoan as being present in the stool and the cysts are also very difficult to find in many cases. Where the most difficulty is found is in the chronic cases and during the remissions, and also in cases of suspected carriers. Therefore, I think that John Gordon Thomas has outlined a method of which should be observed in sending stools for examination. The first point he brings out is that the whole stool should be sent when possible. This is of aid, since the general appearance of the stool is frequently characteristic. Thus the parasitologist can see at a glance whether the stool is diarrheic, semi-solid, normally formed, or constipated, and whether mucus or blood are present, and is so in what quantity. Next, the stool should reach the laboratory as soon as possible. The vegative stage of amoebae is greatly affected by adverse circumstances. The activity will be present only at a temperature of about 37° C. The amoebae become rounded up and immobile, but become active sometimes when a warm stage microscope is used. Cysts withstand external conditions much better, but often a short period will cause them to become degenerate and more difficult to diagnose. When the entire stool cannot be sent the clinician

should carefully select the portion from the stool which is called Sago-grains or nodules of mucus. A platinum loop full may contain hundreds of amoebae, where the remainder of the stool may contain only a few.

The macroscopic appearance of an amoebic dysentery stool is quite characteristic. In the exacerbation period, it may contain only blood and mucus. It should be noted that the general color of the stool is a reddish brown, or, as it has sometimes been described, chocolate shade, and not bright as ordinary blood.

The stool may be a fluid in which the mucus is so intimately mixed with the fecal material as to be almost imperceptible to the naked eye. This is not commonly present. The stool may consist of mucus intimately mixed with the fecal material in which the mucus is seen as small masses or blabs so aptly described as "Sago grains" appearance. In the more latent cases the stool if formed of normal consistency and to the naked eye presents only one feature, unfortunately not always present, which are small glistening pin-points of thick tenacious mucus on the surface. In many of such cases the stool appears to be perfectly normal, but many variations may occur, such as the sudden passage of pure blood from an eroded artery.

In many incidences it is necessary to use a saline cathartic the night before and produce a liquid stool before the amoebae or cysts may be found in the stools.

In questionable cases where the diagnosis is difficult to make and the amoebae are not found or may be present and not distinguishable from the Entamoeba coli, it becomes necessary to use a proctoscope or a sigmoidoscope and frequently the ulcerations will be seen in the sigmoid or descending colon. By the use of a long applicator swab, wipe out one of the ulcers thoroughly and, in many instances, you will find the active amoebae if examined immediately. I think this procedure is of great importance in many questionable cases where the amoebae are not found in the stools.

A complement fixation test has been worked out by Dr. Charles F. Craig (22). This is a method very similar to that of the complement fixation test of syphilis used in the army. He uses an alcoholic extract of the cultures of Entamoeba histolytica grown on a modified Boeck-Dibohlav medium and develops until there are available at least one hundred twenty culture of organisms for extraction with absolute alcohol. It is necessary to use great care in titrating this antigenic extract from hemolytic qualities, anticomplementary qualities and antigenic strength. The technic of the test is important and should be attempted only by a trained

serologist. The practical value of the test, therefore, is limited particularly by difficulty of preparing and titrating the antigenic extract. Craig described this technique in September 1929. He reports it is of value in diagnosing cases of amoebic abscesses of the liver which has no intestinal symptoms and in diagnosing healthy carriers without symptoms. The test is the strongest in cases of carriers and mild infections. In some acute cases it is negative or doubtful. The test is not positive with other species of amoebae of flagellates. Normal persons or those ill with other diseases do not give positive reactions. The complement fixation bodies disappear from patients' blood serum after antiamoebic treatment and the disappearance of Entamoeba histolytica from the feces. It is positive during relapses, unless repeatedly negative for several weeks. Therefore, a negative reaction does not prove the absence of amoebic infection or that antiamoebic treatment has resulted in a cure.

It is thought by many clinicians that this test is of little value due to the fact that the Entamoebae are so difficult to culture and the antigen and complement is so difficult to titrate. Also the test does not give a positive reaction in a great enough per centage of cases to make it a test of any great diagnostic value.

Treatment of Amoebic Dysentery

In the treatment of amoebic dysentery the patient should be put to bed and kept in bed all the time during treatment. In the past century there has been many methods of treatment introduced which indicates that there is no one treatment which will give a positive cure.

Emetine (23) has become the most efficient treatment in late years. Ipecac was formerly advocated for treatment regardless of the cause. The Brazilian root was first taken to Europe by Pias in 1658, where it was successfully used by Helvetius in the treatment of Louis the XIV and was sold as a secret remedy to the French government. Two centuries later, Docker introduced the use of large doses, 60 gr. two or three times a day, of powdered ipecacuahae in the treatment of severe dysentery in Mauritius. His excellent results were confirmed by others.

In 1886 Maclean and Chevers (24) advocated the use of ipecac in acute hepatitis. Later the drug fell into more or less disrepute, but its use has been reserved as a result of Manson's advocacy of its employment in dysentery and Rogers in hepatitis. Meanwhile, many other drugs have been used in dysentery, such as quinine, bismuth, tannin, salines, opium, salvarsan which

have been given in all methods.

The chief interest in the treatment of amoebic dysentery is centered around ipecac and its derivatives, emetine being the most efficient. The drawback in its use is first the reaction received and by the fact that some cases do not respond. Epecac contains five alkaloids of which emetine and cephaelin are the chief ones. In 1912 Vedder (25) reported the effect of ipecac on amoebae and this showed that emetine killed amoebae in a dilution of 1-100,000. From this work he concluded that it was a very good amoebacide. Following this Sir Lenard Rogers, in India, tried hypodermic administration of emetine and reported strikingly good results especially in early acute cases.

Charles F. Craig (26) recommends particularly the use of emetine hydrochloride or emetine bismuth iodide. Emetine is specific for the relief of symptoms. The symptoms are relieved in the first few days after beginning administration. It is doubtful if it actually cures over one-third of the cases. They are always subject to recurrences and the word cured should be guarded against very carefully.

The drug may be given orally, subcutaneously or even intramuscularly. The patient gets quite a reaction out of oral administration and nausea and vomiting is quite common. When given intramuscularly or subcutane-

ously, the patient gets considerable pain and soreness at the area of injection.

When given orally, it must be given in a keratin-coated pill or capsule so that the drug is not absorbed in the stomach, but passes into the small intestine before the keratin coat is dissolved. This prevents considerable gastric distress. The dose for oral administration should not be more than a grain and a half at morning and night in equally divided doses. It is seldom used by mouth alone, but with subcutaneous or intramuscular injections, one-half grain given by mouth and one grain by intramuscular injection every morning for ten to twelve days, repeating this treatment if relapses occur.

Emetine is a toxic drug and when given in too large doses, causes severe diarrhea, myocarditis, neuritis, nervous prostration and great muscular weakness. Death may occur suddenly from cardiac failure. These symptoms should be constantly watched for when the drug is being given. If such symptoms develop the drug should be stopped at once.

Peripheral neuritis (27) after treatment with emetine is quite common. The trouble usually manifests itself in general muscular pain and weakness, especially in the legs and sometimes going on to paresis. Wrist and toe drop is quite common. These symptoms gradually

disappear after stopping the emetine. There has been many cases of death from emetine poisoning. In many of the cases reported as small a dose as four grains spread out in equal doses for four days. The toxic symptoms are first manifested by diarrhea and muscular weakness, and the heart becomes very weak with the usual cause of death being cardiac failure.

In cases in which a course of emetine treatment has been given and the patient received no releif, emetine has been stopped and chapparo amorgosa used. This drug was introduced by Wenyon and O'Connor. The drug is administered as a glass of fresh infusion four times a day, and a quart of the same infusion by rectum after a preliminary injection of sodium bicarbonate solution, one ounce to a quart of water. Sellards and McIver have reported successful treatment of four cases with this drug, but this treatment has been nearly forgotten in recent years.

Sodium iodoxyquinoline (28) sulfonate (Chinioform) may be given orally or as enema in treatment of acute amoebic dysentery. In the adult the drug is given one gram three times a day for eight to ten days. If this causes diarrhea, reduce the dose to one half. The enema given is made up of about 200 cc. of warm 2 per cent solution of chinioform which should be retained for several hours. This is given along with oral administra-

tion of seven and one-half grains three times a day. Chinioform is less toxic and is apparently just as effective as emetine hydrochloride or emetine bismuth iodide.

Oil of Chenopodium (30) has been used in treating many cases of dysentery. It was first found to be of value when Mendelson used it for the treatment of hook worm, and he noticed that the patients with dysentery were much relieved in many cases. From his investigation he found that oil of Chenopodium was a very good amoebacide.

Drs. Walker and Emrick also did considerable work on amoebic dysentery carriers in 1918. It is very important to treat carriers due to the fact that the carrier is the most important factor in the control of the disease. Emetine is not satisfactory in many cases of carriers. A satisfactory treatment of carriers of Entamoeba histolytica not only must be capable of destroying the intestinal parasites, but also it should not occupy too much of the patients' time.

The method of treatment outlined by Walker and Emrick was to give the patient first, magnesium sulphate, from one-half to one ounce at 6:00 a.m.; second, oil of Chenopodium, 16 minims in a keratin capsule or gelatin capsule at 8:00 a.m., 10:00 a.m. and 12:00 m.; third, castor oil, one ounce. This dose is for adults. Children

should have the dose regulated according to the age. The oil of chenopodium is given shortly after the purge, so that it will reach the intestine before the amoebae become encysted. If a single course doesn't result in a cure, then repeat again after a one-day interval.

The most important thing in this treatment is to be sure the purge is of sufficient quantity to produce a liquid stool. This also cleans out the bowel and gives the drug a better chance to attack the parasites and also there is less dilution.

Drs. M. E. Baines and E. C. Cart (31) introduced the use of oil of chenopodium in an emulsion of gum acacia by rectum. In the use of the enema, the rectal mucosa should be protected with petrolatum. The dose of the inert oil should be limited to two ounces. The buttock should be elevated and the enema given slowly and with great care. The first dose should not exceed eight ounces in the adult. The enema should be retained for an hour or two if possible.

Dr. Mason recommends giving the oil of chenopodium in two ounce doses of inert oil in six to eight ounces of olive oil.

This drug is toxic in over-doses and should not exceed the normal dose. It should not be given in less than ten days to two weeks after a full dose is given. An over dose is toxic and causes kidney damage and should be watched very closely.

Oil of Chenopodium relieves promptly the clinical symptoms in many patients with chronic and subacute dysentery. It is safely administered with castor oil when given orally. You may give the drug to the patient and let him take it by himself.

Summary

In summarizing amoebic dysentery, I find that the history of dysentery goes back many centuries, but its etiological parasite was not discovered until 1859, and not recognized or connected with the cause of dysentery until 1875. The definite pathology was first written by Councilman and Lafleur in 1891, and it was not until 1903 when Schaudinn gave the organism the name Entamoeba histolytica and proved it to be the etiological factor in producing one type of dysentery commonly seen in the tropics.

The symptoms of the disease may be sudden in onset or very insidious, or may never show any definite symptoms. In acute attacks the dysentery is one of the outstanding features and the appearance of the stool is of much significance. The patient also has abdominal discomfort and tenderness over the colon. In insidious cases you may have no symptoms that are definite. The diagnosis is made upon finding the amoebae or cysts in the stools and upon observation of lesions in the colon by proctoscopic examination. In some cases the clinical history is sufficient to make a diagnosis, but it should be confirmed by finding the trophozoites or cyst forms. The complement fixation method is not definite enough to depend upon, and culture methods

are very unsatisfactory and difficult to handle.

The treatment of this disease has been very variable. Many drugs have been used in treatment of amoebic dysentery, but at present there are only two drugs commonly used. One is emetine, which is given intramuscularly in the form of emetine hydrochloride, and orally in the form of emetine bismuth iodide. The other drug is Oil of Chenopodium which may be given in gelatin capsules or in solution of oil as an enema.

Both drugs have given very striking results, but the patient is still subject to remissions, and a cure should never be promised when starting to treat a patient with amoebic dysentery.

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