cerebrum, as well as throughout the system of uremic subjects, at least one, and probably several toxic substances which exert their influence more or less locally on the cortex.

2. That it is equally certain that other portions of the brain than the cortex are also acted on, as in the production of coma, etc.

3. That no small part in the production of the uremic and eclamptic condition is played by intracranial pressure, due to a temporary excess of fluid, whether acting independently of or in conjunction with the toxic substances already mentioned.

4. That lumbar puncture will at least temporarily relieve certain of the symptoms most readily ascribed to localized intracranial pressure, and that in cases in which the pressure is the main factor, drainage of the spinal canal may save life. The procedure, together with free bleeding, purging and diuresis, should be added to our routine treatment of the condition.

5. That transfusion of normal salt solution by intravenous injection or hypodermoclysis, except in cases presenting anuria, or a greatly diminished urinary secretion, is contraindicated as tending to increase the liability to saturation of the tissues.

6. That the results of lumbar puncture in the three cases cited in this paper will not warrant the assumption that relief of intraspinal or intracranial pressure can alone be depended on to cure the uremic or eclamptic condition, provided the toxic influence is the prominent one in the particular case.

DISCUSSION.

DR. JAMES TYSON, Philadelphia—Dr. Willson's paper has given a fair presentation of this long-disputed subject. Pressure has long been held by some authorities to have an influence in the production of eclampsia, chiefly, however, on theoretical grounds. Dr. Willson has adduced some experimental evidence, of the value of which I am not prepared to judge without further study. I am not ready, however, to concede that there is not a toxic agent which plays a certain rôle in producing the phenomena of eclampsia. It has been a great many years since I prepared a paper to prove that puerperal eclampsia is due to a nephritis, the toxic products of which are responsible for the eclamptic symptoms. I think the same reasoning still holds at the present day. We must admit, too, that puerperal eclampsia, in very rare cases, may be a reflex phenomenon, just as convulsions in children may be a reflex event due to such irritation as teething, and the like. Convulsions thus caused usually occur in primipara after long and painful labors.

DR. ALEXANDER LAMBERT, New York City—Has any fatty degeneration of the liver been noted in these cases? In eclampsia the lesions are identical with those of acute yellow atrophy of the liver. In my experience and also the experience of the staff of the hospital, we have been struck with the fact of the apparent identity of the condition of the liver in uremia and in acute yellow atrophy. Even in the hyperemesis of pregnancy changes will appear in the urine with the development of leucin and tyrosin. There is no doubt that Dr. Willson clearly and carefully drew attention to the fact that the convulsive seizures were due to tension; but what is the cause of this intracranial tension? That is a question probably far behind that of toxemia. It is the same toxemia that kills in acute yellow atrophy. In eclampsia there are two types: that type with high tension, convulsive seizures predominating; the other type with a flat compressible pulse, and these patients die in spite of anything we may do for them. It is not uremic, but something back of the uremia. I have had experiences in which I have found acute yellow atrophy of the liver, and I think perhaps we have failed to recognize the frequency of the occurrence of that disease.

DR. ROBERT N. WILLSON—The subject is an interesting one,

especially from the fact that many may hold that these patients would have had a better chance of recovery if the procedure had not been carried out. In each of my cases in which lumbar puncture was done the patient died; in each, however, there was an immediate and positive improvement for a time. Vail has reported three cases, all of which recovered, the only treatment being lumbar puncture and rest in bed. Seiffert has also reported successful cases. It therefore seems to me certain that in some cases the dominant influence is pressure due to toxic or other causes. Dr. Lambert has referred to the resemblance between the pathologic findings in eclamptic cases and in acute yellow atrophy. I think there is no question as to the accuracy of his statement. In short, there are cases overwhelmingly toxic, while others appear to be overwhelmingly due to pressure. In the latter lumbar puncture may be a means of saving life.

I will refer to another matter that is rather interesting. Dr. Riesman has called my attention to the fact that all of his fatal cases gave a bloody cerebrospinal fluid. It would appear that when the fluid is clear, the case is much more apt to recover. In one of my own cases the fluid was clear after the first two punctures, but bloody after the third. All of these patients died. Vail mentions the fact that the fluid from his favorable cases was clear. Whenever failure has been experienced in getting into the canal it was due to the use of too short a needle. I now use a four-inch small trochar and canula, applied through an incision in the skin and subcutaneous tissues. I have never failed to get within the canal if I kept close to the spinous process with the needle point.

AMEBIC DYSENTERY.

ITS LOCAL LESIONS AND TREATMENT.*

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Our views of dysentery have greatly altered since the days when we considered it "inflammation of the bowels" without knowing exactly what we meant by the term inflammation. These changes of views are the result of pathologic studies and are in line with the great changes made in our views of surgical etiology and pathology.

With regard to dysentery they began in the discovery by Losch of the ameba coli, and the association of this germ with the disease by Councilman and Lafleur; for a while after this the profession accepted that ameba coli, afterward called "*Amœba dysenteriae*," as the established cause of the disease. In 1894 and 1895 the reports of Shiga, Flexner, Harris and others, seemed to establish the fact that at least in the epidemics of Japan and the Philippines the ameba was not always present, but there was a bacillus of a peculiar type universally present and to this they gave the name *Bacillus dysenteriae* and established its etiologic relations to that form of the disease.

After this various efforts were made to establish the unity of dysenteries and to prove that this bacillus was the only cause; that the presence of amebæ in dysentery was accidental. Clinical observations, however, have failed to justify such sweeping conclusions. The best authorities have finally concluded that not only are there cases of pure bacillary dysentery and cases of pure amebic dysentery, but there are also cases at least with the typical symptoms of dysentery in which neither the bacillus nor the ameba can be found. We therefore recognize these three types: simple catarrhal dysentery, bacillic dysentery and amebic dysentery.

* Read at the Fifty-fifth Annual Session of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. J. M. Anders, Frank Jones and W. S. Thayer.

It is to this last form of the disease that I wish to direct attention. What I have to say is based on personal experiences of seventy-four cases of amebic dysentery, together with what knowledge I may have gained from reading. In every case observed, with the exception of one, the diagnosis has been made on the finding of living motile *Amœba dysenteriae* in the stools or scrapings. To some it has seemed a presumption to look on the amebæ as the cause of ulcers and dysentery, but the fact that they are present with other pathogenic agents in all cases, and the fact that the dysenteric symptoms and local lesions disappear only when the amebæ have been eradicated and recur when the amebæ reappear is positive proof, it seems to me, of their etiologic influence in this disease. I, therefore, assume that amebic dysentery has been proved and we may proceed to consider its cause, its symptoms, its local lesions and its treatment.

ETIOLOGY.

The cause of the disease is the infection or invasion of the colon by *Amœba dysenteriae*. These amebæ consist of irregular oval masses of plasmodium, gelatinous in their appearance, scarcely visible when not moving, containing in their beds certain granular masses or vacuoles and appearing to be more or less refractile. They may be found in the mucus which is discharged in the stools, as well as in the scrapings from the ulcers in the rectum and sigmoid. Their source is unknown, nor is there any accepted theory with regard to it. The behavior of amebæ found in dysentery differs greatly from that of those found in fresh water, when exposed to heat or cold; it is this point alone that positively distinguishes the two. The latter remain motile at high and low degrees while the former are viable only at temperatures near that of the human body. Some have claimed that there was a difference in size, but my observation does not justify such an opinion, as the size of the amebæ varies greatly in different cases and even in the same case.

METHOD OF REPRODUCTION.

As to the method of production: It was formerly supposed that amebæ multiplied by binary division, after the manner of leucocytes in the blood. According to Craig of the United States Army, Dofflein and others, it is now claimed this reproduction occurs through ovulation and sporulation; the spots in the amebæ which stain deep blue with Wright's solution represent spores, and those which stain very faintly the vacuoles from which spores have been discharged. There is one point in Craig's experiments in which an assumption is necessary, viz., that the bodies observed stained are the same as those observed in the living state before the staining, for it is impossible to stain and preserve the amebæ motile. In fact, one can hardly positively distinguish non-motile amebæ from other substances in the stools. If, however, this assumption is admitted, the method of reproduction by sporulation would seem fairly well established, and it is confirmed by the clinical experience of recurrences long after the motile amebæ have entirely disappeared from the stools.

TEMPERATURE AND OTHER CONDITIONS AFFECTING THE AMEBÆ.

The fact that we have been unable to isolate or obtain a pure culture of this ameba renders its study more difficult than that of ordinary pathogenic agents. Its behavior, however, under certain circumstances has been observed clinically, and from this we may formulate some of its characteristics. At the temperature of the body, or slightly higher, the life of the ameba is indefin-

ite; if, however, the temperature of the vessel in which it is obtained gets below that of 70 degrees F., the motility and life of the ameba become extinct and no amount of heat will restore the same.

Bichlorid of mercury 1,-10,000, at the temperature of the body, checks the motility but does not destroy it except after very prolonged contact. Rogers states that a solution of quinin 1-1,000, at the temperature of the body, failed to destroy the amebæ after several hours' contact, but that a 1-500 solution stopped all movements in from five to fifteen minutes; when the amebæ were in the tissues, however, they were only destroyed by prolonged soaking in 1-500 or 1-100 solution of quinin. Saline solutions at the temperature of the body will not destroy the motility. According to Harris of Atlanta, peroxid of hydrogen is fatal to the germ. He does not state, however, whether this fluid was used cold or warm. Five per cent. of the 15 volume peroxid of hydrogen in water, however, does not seem to destroy the amebæ at a temperature of the body. Nitrate of silver in 1 per cent. solution checks the motility but does not seem to destroy the germ. All these solutions, however, when used at a temperature below 70 degrees, are fatal to the motility and life of the amebæ. In at least twelve of the cases which have been under my care the stools had been examined in vain for amebæ before they came to me. In every instance the failure to find the bodies has been due to the fact that the stools were examined after they had cooled off. In order to recognize the amebæ with any certainty the examination must be made while the stools are still warm and on warm slides. In my office these examinations are made from scrapings obtained from ulcers in the rectum and from the mucus collected on the rectal wall. These are placed at once on warm slides, put under a microscope and slightly heated now and then in order to maintain the temperature at about 98 degrees F. In the hospital or laboratory the stools are collected in warm pans, transferred immediately to the oven of the laboratory and kept there until an examination can be made at the temperature of the human body. These precautions are absolutely necessary to make an accurate diagnosis.

CHARACTERISTICS OF THE DISEASE.

Amebic dysentery is characterized by an insidious onset, variable but usually mild constitutional symptoms, frequent recurrences, extreme chronicity and latent existence. It may begin as an acute diarrhea, with blood and mucus in the stools and burning in the rectum, or it may come on as a simple looseness of the bowels, some of the stools being practically normal while others are thin and contain much mucus, with or without blood, and causing little or no pain. The acute cases are associated with burning in the rectum and frequent desire to defecate, loss of appetite, and slight elevation of temperature. The stools may be very frequent; the demand to defecate is imperative, but they are not associated with tormina nor excessive burning in the rectum. In most instances castor oil or salines, followed by moderate doses of opium and bismuth, control the disease for the time being. In two instances in the series of cases observed periods of quiescence lasting for six months in one and four months in the other have been observed. We must make some allowances with regard to the statements of these patients regarding these periods of absolute quiescence, for they are only periods of comparative activity of the amebæ. In a few instances the patients have actually become constipated during these periods, but such cases are exceptionally rare. In al-

most every instance the patients have reported periods of feeling greatly improved at times, but when we have carefully pinned them down to the facts these patients have considered themselves practically well when they were having two to four mucous stools a day without any pain.

LATENT DYSENTERY.

By latent dysentery we refer to the condition in which *Amœba dysenteriae* are present in the intestine, without the diarrheal symptoms or the local manifestations in the form of rectal and sigmoidal ulcers. Rogers has established this fact, and I have myself observed four cases in which there was no diarrhea and yet there was amebic colitis with malaise, loss of flesh and strength and moderate temperature at night. One of these cases suffered from abscess of the liver, which recurred from time to time and which was operated on twice in five years. He could not recall ever having had a diarrhea. He was referred to me with the view of discovering some cause for these recurring abscesses of the liver in possible ulcers in the rectum and infection from these through the portal circulation. No ulcers were found in the rectum, but certain small cicatrices and some fetid mucus suggested a careful examination of the stools; these we found swarming with *Amœba dysenteriae*. Two other cases with tenderness over the caput coli, loss of appetite and gradual decline in weight, mucous colitis but no diarrhea, have been observed, in each of which the stools contained *Amœba dysenteriae* in abundance. In one of these cases amebic ulcers were found in the rectum, and in the other two small ulcers were observed in the sigmoid. The fact, therefore, that the patient does not have frequent, bloody, mucous stools does not prove that he is absolutely free from *Amœba dysenteriae*. From cases recently observed, I am inclined to believe that no inconsiderable number of the sufferers from mucous colitis are the victims of amebic infection notwithstanding the absence of diarrhea.

GEOGRAPHICAL DISTRIBUTION OF THE DISEASE.

The locations in which amebic dysentery is likely to be found are worthy of consideration. Formerly the disease was called "tropical dysentery" under the false impression that it was only found in the tropical zones. While it is a fact that it is more frequently found in warm climates, yet it is well known that it may also occur in almost any climate. I have seen three cases of acute amebic dysentery in patients who have never been more than fifty miles south of New York and fifteen cases in which the patients have always resided above the thirty-seventh degree of latitude. The majority of the seventy-four cases observed, however, have contracted the disease in the Philippines, the West Indies and the extreme southern portions of the United States. The possibility of amebic dysentery can not, therefore, be eliminated on account of the patient's residence. The disease is nearly always sporadic even in the tropical zones, and may thus be distinguished clinically from bacillary dysentery, which is generally epidemic.

LOCAL LESIONS OF AMEBIC DYSENTERY.

The local lesions occurring in amebic dysentery differ in the acute and chronic forms. The disease is practically confined to the colon, although there are instances in which the small intestine has been involved for a short distance above the ilocecal valve. In some cases the disease begins in the rectum and travels upward, while in others it follows the reverse course. Rogers

and Fitcher state that the typical cases of infection begin in the caput coli and travel down. At all events the rectum and sigmoid flexure become involved sooner or later in almost every case. In the three acute cases observed the mucous membrane at the anus was pouting, bright red and swollen, the radial folds were edematous and painful; the walls of the rectum were in close apposition with each other, hot and tender to the touch, bright red in appearance and in one case there were several patches of pseudo-membrane. At this stage of the disease no actual ulcerations were observed, but later on these developed. These ulcers were irregularly oval in shape with gelatinous, yellowish, elevated centers, surrounded by bright red or purplish zones. They are usually superficial and heal rapidly under local treatment.

In the chronic form the ulcers begin as small red papules on the mucous membrane, rapidly becoming more and more prominent until a yellow spot develops in the center, due to the loss of epithelium, and this constitutes the center of the ulcer. I have frequently seen these in chronic cases of amebic dysentery, but did not observe them in either of the acute cases examined. The ulcers are usually situated on the summits of the mucous folds; a favorite site is on the folds of Houston. The size and shape of the ulcers vary greatly; some are small and irregularly oval, while others occupy almost the entire circumference of the gut. The long axis of the ulcer is usually at right angles with the long axis of the gut.

In very chronic cases the ulcers sometimes assume the form of little troughs cut out of mucous membrane with overhanging edges which resemble very much worm-holes in wood. These trough-like ulcers sometimes cross one another, forming stellate ulcers. In this type of ulcer the base is hard and quite red and may extend through the muscular layer to the peritoneal layer, causing adhesions. The yellow gelatinous mass which forms the center of the ulcers is composed of submucosa infiltrated with *Amœba dysenteriae*. Strange as it may seem, the amebæ are more abundantly found in the smaller ulcerations than in the large. In some cases large sloughs composed of detached shreds of mucous membrane are seen at the site of the ulcer or in the stools. Rogers has pointed out that these cases are generally associated with abscess of the liver which contain streptococci in addition to amebæ, thus showing a mixed infection. As pointed out by him, the odor in these cases is gangrenous and fetid, which is entirely unlike that found in amebic dysentery. One might here call attention to the peculiar odor of the stools in amebic dysentery, which is entirely different from that observed in other types of colitis. As the ulcers heal the yellow centers melt away, assume a grayish tint and gradually disappear, leaving a clean granular base more or less indurated; the small ulcers heal rapidly and all evidence of their having been present is lost; the larger ones, however, and the trough-shaped variety leave cicatrices which have been known to result in strictures of the gut.

DISTRIBUTION OF THE LESIONS.

While all writers agree with the general statement that amebic dysentery is practically confined to the colon, there is considerable difference between them in regard to the portion of this organ most frequently affected. Fitcher and Rogers state that the cecum, appendix, hepatic flexure and ascending colon are the chief sites of the lesions. In 116 cases Fitcher rarely, if ever, found the lesions in the rectum and sigmoid. These

statements are entirely at variance with my experience, as well as that of Strong, Leonard, Craig and others. In the seventy-four cases of amebic dysentery observed by me, typical ulcers of the rectum and sigmoid were found in seventy instances. In every case except one the character of the lesions was proved by the demonstration of living motile amebæ in the stools and scrapings from the ulcers. Fitcher himself, although he claims that the chief seat of the disease is in the cecum, bases his diagnosis on the finding of *Amœba dysenteriae* in scrapings from the rectal wall.

Why these experiences should differ it is impossible to say unless it be due to the fact that my observations have all been made, with one exception, on living subjects, while those of the authors quoted were made postmortem. The one patient who died while under my care from acute miliary tuberculosis showed dysenteric ulcers in the caput coli and in the ileum just above the ileocecal valve. It is possible, of course, that the other cases had ulcers so high up in the intestinal canal that it was impossible to observe them through the sigmoidoscope. In the majority of instances, however, the ulcers appeared to decrease in size and number from the rectum upward until they disappeared entirely in the upper portion of the sigmoid flexure, and as the patients entirely recovered under local treatment, it is reasonable to suppose that all the ulcers were reached. There is no doubt, therefore, in my mind that the rectum and sigmoid flexure are involved in a large percentage of chronic cases.

DIAGNOSIS.

The diagnosis of amebic dysentery rests on finding in the stools or in the scrapings, living motile *Amœba dysenteriae*. Sometimes it requires several examinations before their presence can be established, but when once seen, in any considerable numbers along with the typical ulcers, the character of the disease is beyond doubt. This diagnosis will be more positive if the amebæ are observed in the scrapings from the ulcers, rather than in the stools, for it is known that *Amœba coli* may be present in the intestinal canals of perfectly healthy individuals. The simple presence, therefore, of one or two amebæ is not sufficient, without the ulcers and typical symptoms, to justify the diagnosis of true amebic dysentery.

COMPLICATIONS.

The complications of amebic dysentery are physical exhaustion, anemia, autointoxication, peritoneal adhesions and abscess of the liver. The first two are not uniformly present; patients may suffer from the disease for a long time without emaciation or any great loss of strength. Some cases, however, become rapidly emaciated; the hemoglobin is greatly reduced and they are practically bedridden early in the disease. In all instances the patients have a feeling of lassitude and are just a little below par in strength. The color is usually sodden rather than pale, and there is often the hectic flush on the cheeks which suggests the possibility of tuberculosis.

These conditions are due to the autointoxication and drain on the system brought about by the presence of amebic ulcers in the intestine. When the ulcers have healed and the intestine is restored to its normal conditions, these symptoms rapidly disappear.

Abscess of the liver is the one complication which commands our highest respect and demands the most rapid eradication of the amebæ. It is not my purpose to discuss here how these organisms reach the liver, whether

through the portal circulation or across the peritoneal cavity; the fact remains that the large majority of cases of abscess of the liver are due to infection by *Amœba dysenteriae*, and these have their habitat in the colon.

As Rogers states, no case of amebic abscess of the liver has ever been observed in which there were not present amebic ulcers in the colon. This fact justifies the statement that wherever the patient has symptoms of abscess of the liver or has suffered from it, he should be treated for amebic dysentery whether amebæ are found in the stools or not. The fact that they are not found at the time of examination does not prove that the spores are not buried in the submucosa, lying dormant and ready to reproduce the disease at any time.

TREATMENT.

The treatment of amebic dysentery is based on two facts which have already been mentioned, viz.: The disease is due to an infection of the colon by *Amœba dysenteriae*, and the seat of the infection is in the crypts of the mucous membrane or in the submucosa. Whether the original infection is due to the burying of the amebæ themselves in these tissues or to the deposit of the spores it is impossible to state. From recent observations I am of the impression that the latter is more probable, but this is not important from a practical point of view. Our efforts should be directed toward getting rid of them and preventing their reproduction. In the beginning we may observe that there are usually no serious constitutional manifestations in this disease except when these are due to complications such as have been mentioned.

There is, therefore, no indication for serum therapy, as the disease is not in the blood and can not be reached through that channel. The whole treatment should be directed toward the local lesions, the destruction of the amebæ and the prevention of their development from the spores, and as these are buried in the tissues, it is evident that it will be impossible to destroy them by superficial washing or flushing of the intestinal canal, although this treatment is important and should be made use of to get rid of all the germs floating about in the colon. It is clear, however, that something should be employed by which the tissues may be penetrated and the germs or spores destroyed in order to completely eradicate the disease. While the remedies mentioned, bichlorid of mercury, nitrate of silver, saline solution, solutions of quinin, peroxid of hydrogen, etc., may be germicidal to the ameba when brought in direct contact with it, it is perfectly clear that none of these remedies will penetrate the submucosa.

This fact explains why so many cases treated by these remedies recur. The ideal treatment must, therefore, consist of some method by which these buried organisms can be reached and destroyed *in situ*. Early in our studies of *Amœba dysenteriae* it was discovered that when the specimen stools on the slides on which they were being examined cooled off below a temperature of about 70 degrees F. the motility of the amebæ was lost and could not be restored; this fact suggested that if the temperature of the parts containing these organisms could be reduced considerably below this degree the amebæ could be destroyed and their infecting and reproductive powers would be eliminated. The application of cold to the intestinal mucous membrane through prolonged douches seemed to offer a solution of the question. At first such remedies as krameria, ichthyol, bichlorid of mercury and nitrate of silver were introduced into the cold douches, but one after another

was discarded, as it was found that simple cold water served every purpose in destroying the amebæ.

When the bowel is tolerant of hydrogen peroxid 5 to 10 per cent. of this remedy is introduced into the water on account of its beneficial effect to the ulcers and to combat any mixed infections that may happen to be present.

In some patients it is necessary to use a long rectal tube in order to get the fluid high up in the colon, but in the majority of cases the simple rectal tip of a fountain syringe is sufficient. The patient is placed in the knee-chest posture, the bag or fountain being elevated not more than three feet above the level of the hips and the fluid is allowed to run in slowly. The amount of water used is limited only by the tolerance of the patient. Some take only a small quantity at first, but they soon learn to take even two or three quarts. Some can retain the water for only a few minutes, while others retain it for three-quarters of an hour. In the former class it is necessary to repeat the injections two or three times at each sitting in order to obtain the desired effect. The treatment is always begun by the administration of a large dose of sulphate of magnesium, and this is repeated once or twice every week, according to the necessity of the case. The time consumed in ridding the bowel of the amebæ depends entirely on the tolerance of the intestine to cold water. In those cases in which large amounts of very cold water can be retained the organisms disappear in a very short time, while in those who can only retain small quantities for short periods several weeks are required.

In the chronic cases, where there is tenderness over the cecum and hepatic flexures, an icebag is applied over this region for two hours twice a day.

A metal or glass reservoir is better than a fountain syringe for the injections because these vessels can be filled with cracked ice and the water kept very cold. The best results have been obtained in these cases in which the temperature of the water used was below 45 degrees F.

Local ulcers of the rectum and sigmoid are treated by applications of antinosin, argyrol and other astringent and antiseptic substances. This local treatment is of the utmost importance and should never be neglected, as many of these ulcers are complicated by a mixed infection.

Internal medication is indicated where the digestion is impaired or where the patient needs stimulation on account of exhaustion. In three instances the disease has been seated so high up in the colon and the patients have been so intolerant of cold water that it was impossible to reach the affected region in this manner and it became necessary to resort to surgical means in order to eradicate the amebæ. In one instance the Gibson method of valvular colostomy was employed; in the other two the appendix was drawn up into the abdominal opening and sutured to the skin, after the method of Weir, until the peritoneal cavity had closed off; it was then amputated close to its base and there was thus left an opening through which a catheter could be introduced for irrigating the colon. In two of these cases the improvement was rapid and recovery complete. In the third case the patient developed miliary tuberculosis and died at the end of three weeks, the regular treatment having never been instituted. This case showed typical amebic ulcers above the ileocecal valve, thus indicating the possible advisability of making the opening in the ileum instead of the colon in such cases. At the same

time recognizing the fact that the appendix may be the seat of amebic infection, it would be wise to remove this organ in all operative cases.

In seventy-three cases treated by these methods the results have been seventy cures, one death, one abscess of the liver, which eventually recovered, and one case improved but not cured, the patient being necessarily called to Europe after two weeks' treatment. In five cases there were at times marked tenderness over the hepatic region and considerable rise in temperature, but these symptoms rapidly subsided with more frequent douching and the application of icebags over the caput coli and the hepatic region.

42 West Fiftieth Street.

DISCUSSION.

DR. FENTON B. TURCK, Chicago.—Dr. Tuttle called attention to the futile efforts to destroy the plasmodium; it is very interesting to see the efforts made to destroy them in some other way. It has been my experience that solutions have but little effect, and those that did had an effect on the circulation through the hyperemia that was produced by the drugs used. I have had a large experience in treating such infected cases while in the Army. In the treatment I have tried oil, but the oil method has not been successful. It has shown some effect in that it produced a better physiologic activity in the colon. Oil may become a powerful irritant and so excite a hyperemia of the mucosa and thus produce some effect on the plasmodium. The method I now use is hot water. At the Pan-American Congress in 1895-96 I presented a contribution on the good effects of ice water, using hot water first. I found that if I used cold water first there was danger of collapse in weak patients. Therefore I have found more benefit from using a high temperature. A temperature of 115 F. is not sufficient to produce any effect on the micro-organisms; one should use a temperature of from 122 to 131 F. This can be done by gradually raising the temperature, starting with a temperature of 105 F. This heat has a very beneficial effect, seems to destroy the plasmodium, and after every treatment the amebæ disappear. Then followed the use of ice water, and the effects produced are much better than if ice water be used alone. In very weak patients the effect of the heat followed by the ice is very beneficial.

In regard to this operation I have never experienced any difficulty in reaching the caput coli. The patient should be placed on an inclined plane and the tube should be introduced into the sigmoid and water run into the colon, let out again, reintroduced, and so one can douche the entire colon. When bismuth is used the x-ray will demonstrate the bismuth as high up as the transverse colon. There is no difficulty in the return of the water. Introduce a little air with the water; this increases the peristalsis and the fluid goes on. The vital condition of the patient should not be overlooked.

DR. A. W. WARDEN, Weehawken, N. J.—The most successful experience I formerly had in the treatment of these cases of dysentery was by the use of ipecac or quinin, but recently I have used a remedy that I consider better. This drug is *Chaparro amargoso*, which to adults may be given in doses of from 2 to 4 grains of the extract or of 30 to 60 minims of the fluid extract every two hours during the day. In dysentery *Chaparro amargoso* seems to have a specific action by altering the secretions of the bowel in destroying the protozoa and in nullifying their power. It is also tonic, antiperiodic and astringent. *Chaparro amargoso* is also of value in the treatment of enterocolitis or summer diarrhea of children, used in solution either alone or combined with opium.

DR. PHILIP MARVEL, Atlantic City.—One year ago in conversation with Dr. Tuttle about such a case he advised me to use ice water. I had been using different remedies, such as quinin, carbolic acid, nitrate of silver, argyrol, peroxid of hydrogen, and others, with little or no effect in controlling the ulcerations. At Dr. Tuttle's suggestion, I tried ice water,

and much to my satisfaction, the patient grew steadily better. It was my privilege to see her one week ago, and she was in very good condition. I should like to ask Dr. Tuttle if he has observed contractures or strictures after the water treatment. I do not know whether the stricture in my case is due to the disease or to the topical applications made by me prior to the time of beginning the ice water treatment. The patient I refer to is now perfectly well except for a contracture in the rectum. Previously, Tuttle's proctoscope could be used, i. e., passed without pain, but now this is impossible; the stricture will only admit a medium large size old style Kelly's cystoscope.

DR. SAMUEL S. GANT, New York—Was not Dr. Weir the first to suggest this opening of the appendix and using it for irrigating the colon?

DR. WILLIAM KRAUSS, Memphis, Tenn.—In reference to the diagnosis of amebic dysentery, I think I am right in stating that I never made a diagnosis unless my microscope and the patient were in the room together. I have never been able to revive a motionless ameba. The mucus from the discharge or swabbing should be transferred quickly to a warm slide. I have tried Dr. Tuttle's suggestion of cold water and with success. Water can not be used hot enough to injure the parasite.

DR. TUTTLE—As stated in the paper it was Dr. Weir who first suggested making use of the appendix for this purpose. I modified Dr. Weir's operation by drawing out the appendix and absolutely getting rid of it, at the same time obtaining access to the seat of the amebic infection. I omitted to speak of the mixed infections associated with dysentery of this type. I also wished to speak of the importance of the local treatment of ulcerations down in the rectum and in the sigmoid, when using cold water irrigations; and of hot water for improving the circulation. There are certain cases in which the ulcers become infected with a secondary infection, and they must be treated individually by local applications of tincture of iodine, etc. With regard to ipecac and quinin, how can they affect the buried amebæ? The amebæ are not in the blood, but in the submucosa. The only way to reach them in this position is by the use of low temperatures. Dr. Morton of New York has recently reported a case in which the diagnosis of amebic dysentery was made and the case was treated by him by radiized quinin, and he claims the patient is well. Operation is to be resorted to only when all other methods of cure have failed. I do not believe it is a dangerous operation; it is certainly not more dangerous than leaving the amebæ to progress to a chronic state, with its dangerous complications. There is little danger in pulling up the appendix and holding it there until adhesions form and shut off the peritoneal cavity, and then amputating it.

THE PATHOLOGY AND ETIOLOGY OF ARTHRITIS DEFORMANS.*

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In discussing this subject there are two courses open; one is to go over the various theories and ideas that have been put forth, the other is to dispense with tradition so far as we can and state as closely as possible the condition of our knowledge in regard to this subject to-day. It is perhaps well to state frankly that we are more likely to outline our ignorance than to state our knowledge, but we can at any rate determine the lines in which investigation is most likely to be profitable, because I take it that no one would be likely to say to-day that we had

anything very firmly established in regard to the subject of this paper.

Through bacteriologic work we have been able to limit certain forms of arthritis definitely. It would seem best to restrict the term "infective arthritis" to those forms in which we have a distinct causal agent proved. The forms associated with the tubercle bacillus, gonococcus, pneumococcus, etc., belong to this group. Recent work suggests that acute articular rheumatism will before long be added to these. In regard to arthritis deformans, the evidence is as yet not positive, but very suggestive.

GENERAL CONSIDERATIONS.

Almost every point that one can raise in regard to this disease is a disputed one. The more important ones seem to me as follows:

1. Have we under this term one disease with many different manifestations, or have we included two, perhaps more, totally distinct diseases?

The decision in regard to this point is, of course, essential before one can get to any satisfactory conclusion as to the etiology or pathology, and I trust the discussion may clear up some of the points in regard to it. As has been frequently pointed out, there are certainly varying types of the disease. Some observers have distinguished these as the rheumatoid or atrophic type, and the osteoarthritic or hypertrophic type. In the former the disease may occur at any age from 2 to 70 years; the arthritis is usually polyarticular, may come on with great suddenness and recur perhaps at long intervals, or it may be a slow, progressive process. In this type the changes are more especially atrophic and often especially periarticular. As has been shown by Goldthwait, there is much increased excretion of calcium salts. In the second or osteoarthritic group there is in some ways a rather different picture; the classical description of the older writers, which laid special stress on the production of osteophytic growth, applies especially to these. These groups will be considered more in detail later.

2. The question of relationship of arthritis deformans to acute rheumatism. Of course, it is quite possible that a patient may have acute articular rheumatism and years afterward arthritis deformans, but I believe this is the only association between the two diseases, and that arthritis deformans has nothing whatever to do with acute articular rheumatism. Much of the confusion has been due to the mistake of considering attacks of arthritis deformans as acute articular rheumatism. Of course, this opinion is contrary to that of many of the authorities on the disease.

3. The question as to the essential etiologic factor, which we have to discuss under three heads: (a) A neural cause; (b) infection, with a definite specific organism; (c) infection, with various organisms.

One point has to be strongly kept in mind, that although the early and late conditions are quite different both clinically and pathologically, nearly all the older descriptions of the disease are based on the late changes, and no account is taken of the early ones. This has resulted in the process being commonly regarded as a degenerative one only. It would be as reasonable to base all our views as to the pathology of chronic nephritis on the conditions found in a much contracted kidney, ignoring the early stages.

Arthritis deformans appears under very different conditions and in varying forms; coming in the first decade of life, it differs very much from the form seen in adult life. In one it may be very acute, advancing rapidly to extreme deformity and destruction of the joints.

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