Flexner in his bacteriological studies of Philippine dysentery in 1899 obtained cultures of two types of bacilli from the intestinal walls and contents. Type I. was found in acute cases; it was pathogenic for laboratory animals. Type II. was present in all dysenteric cases, but less prominent than Type I. in acute cases. It was not found in healthy persons or in those suffering from other diseases. These observations confirm Shiga's results.

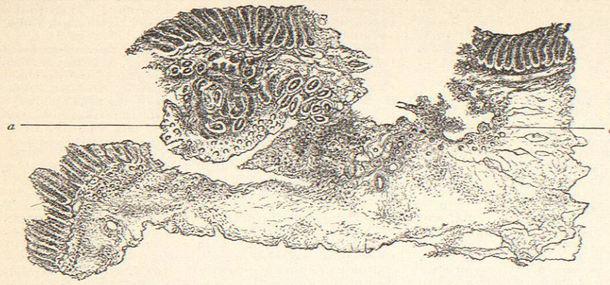

FIG. 1673.—Acute Amœbic Dysentery. Spreading Ulcer. *a*, *a*, Amœbæ in the Submucosa. Enlarged 80 times. (After Kartulis.)

The report of the Surgeon-General of the United States Army, for the year ending June 30th, 1900, contains a very full and interesting report by Strong and Musgrave on the etiology of the dysenteries of Manila. In a period of ten months there were, in the First Reserve Hospital, Manila, 1,328 cases of dysentery, being 14.64 per cent. of the total of 9,063 cases of illness admitted. One hundred and eleven autopsies were made on dysenteric cases; 21 of these were classed as "acute specific dysentery," 11 as "subacute specific dysentery," and 79 as "amœbic dysentery." A bacillus was isolated in 17 out of the 21 cases of acute dysentery and in 2 out of the 11 cases of subacute dysentery; this bacillus was similar to that found by Shiga at Tokyo in 1897. Strong and Musgrave regard this Bacillus dysenteriæ as the cause of these acute and subacute forms, and thus their conclusions agree with those of Shiga and Flexner.

This opinion is supported by the voluntary ingestion, by an Indian criminal condemned to death, but in perfect health, of a forty-eight-hour bouillon culture of Bacillus dysenteriæ in warm milk. In twenty-four hours characteristic dysenteric stools appeared, with fever; the stools contained mucus, cells, and bacteria, but no amœbæ. This attack ended in recovery.

Flexner, Strong, and Musgrave describe minutely the bacilli found by them; they were pathogenic for mice, guinea-pigs, rats, and rabbits; injected into the rectum of cats and dogs they did not produce dysenteric lesions or symptoms. In 26 cases of dysentery which presented different lesions and in which amœbæ were present the Bacillus dysenteriæ was not found.

The blood serum of acute and subacute cases of the acute specific dysentery has an agglutinative reaction with the B. dysenteriæ by the third day; it is marked by the fifth or sixth day.

(1) AMŒBIC DYSENTERY.—This form of dysentery is endemic in India, Egypt, the Philippine Islands and elsewhere in the Tropics. It has been met with as a sporadic form in the United States. Osler says that the cases of acute and chronic dysentery admitted to his wards in Johns Hopkins Hospital have been almost exclusively of this form.

Pathological Anatomy.—The sigmoid flexure is the chosen seat of disease; it may involve the ascending or descending colon, or the whole of the large bowel at one time. The intestine is thickened; the mucosa coated with a muco-sanguineous secretion is intensely hyperæmic, swollen, and in places ecchymotic. The solitary follicles appear as grayish-white points, surrounded with an areola of injected vessels. Nodular projections of the mucosa are seen; they result from œdema and cellular infiltration of the submucosa. This condition is followed by an ulcerative process, beginning in the submucosa and undermining the mucous layer above. These overhanging layers next fall off, leaving large exposed ulcers, which may deepen until the muscular coat is exposed and forms the base of the ulcer. The ulcerative process is thought by Kruse and Pasquale to begin in the solitary follicles, but according to Councilman and Lafleur the origin is in the submucosa.

Amœbæ and other micro-organisms are found clinging to the mucous surface and penetrating into the intertubular spaces. Kartulis believes that the amœbæ destroy the epithelium, penetrate the lymph channels and finally reach the submucosa, exciting inflammation and necrosis. He does not agree with Kruse and Pasquale that the bacteria co-operate in causing the lesion of dysentery, but is more in accord with Councilman and Lafleur as to the exclusive agency of amœbæ.

The necrosis once begun leads to various forms of destructive metamorphosis—small cavities in the submucosa, deep round and irregular ulcers with undermined edges, serpiginous ulcers, and small follicular ulcers; the ulcers extend into the submucosa; the base is rarely formed of the muscular or serous layer. Some ulcers are covered with a slough of necrotic tissue.

The ulcers in the early stages are scattered at varying distances throughout the colon. Later, by the spreading of neighboring ulcers and by the undermining and destruction of the intervening tissue, the mucosa is converted into a ragged surface with hanging shreds and fringes of undestroyed mucous membrane. This advanced process may be limited to certain small areas or may extend throughout a considerable portion of the colon. (See also article on *Amœbæ Pathogenic for Man*, in Vol. I.)

Abscess of the liver was found in six out of eight of the autopsies of amœbic dysentery (Councilman); in four cases there was more than one abscess, and in two cases they were very numerous and small; in every case the right lobe was the seat of this change, and in two cases there were abscesses in the left lobe. In the smaller abscesses there was a small amount of glairy, semi-transparent fluid; in the large ones the contents were more fluid, of a grayish color, sometimes brownish-red from admixture with blood. A distinct limiting wall, sometimes dense and hard, is found in the larger abscesses, but it has not been seen in the smaller ones.

The fluid of the smaller abscesses is composed of fatty, granular material, fragments of liver cells, and pus and lymphoid cells; that of the larger abscesses contains fat, red blood corpuscles, fragments of liver cells, and very few pus cells. Amœbæ were contained in both, but were more numerous in small and recent abscesses.

The following table gives a summary of the relations of abscess of the liver to dysentery, as deduced from observations in India.

TABLE A.

Authority.	Locality.	Number of cases of dysentery.	Number of cases of abscess of liver with dysentery.	Other alterations in the liver.	Liver healthy.	Per cent. of abscess of liver in cases of dysentery.
Murchison	India	.....	102	.....	.....	
Horton	India	55	6	.....	.....	10.9 +
Dr. McPherson	Calcutta Hospital	160	21	.....	.....	13.1 +
	Calcutta Med. College Hospital	245	.....	124		
Mr. McGregor	India	21	.....	16		
Dr. Shanks	Madras	96	36	.....	.....	37.5 —
Dr. Janis	Madras	43	8	.....	.....	18.3
Dr. Monat	Madras	61	13	30	18	21.32
Dr. Hamilton	Madras	17	0	.....	.....	
Dr. William Dix	Madras	26	9	.....	.....	34.6 +
Dr. Thompson	Madras	11	2	.....	.....	18.18 +
Dr. McGregor	Madras	5	1	.....	.....	20.—
		259	69			26.6
Dr. McPherson	Bengal	293	46	.....	.....	16.04 +
Dr. Morehead	Bombay	32	13	.....	.....	40.6 +
Dr. Stovell	Bombay	49	21	.....	.....	42.8 +
		81	34			41.9 +
	Madras, Bengal, Bombay	633	149	.....	.....	23.53
Waring	India	.....	96			
Fayrer quotes Moore		1,663	347	.....	.....	20.87 +
Parkes	India	98	43	50	6	43.9
Reynolds	Netley Path. Museum	25	3	.....	.....	12.0
Schneider		1,400	57	385	10	4.07
Béranger-Féraud	Senegal	411	143	170	98	34.79
Annesley		29	21	.....	.....	72.41
Haspel		25	13	.....	.....	52.0
Gluck		151	16	.....	.....	10.59
Grand total		4,916	1,028	775	132	20.91

Strong and Musgrave found liver abscesses in 14 out of 97 cases. Other changes in the liver consist in extensive necrosis in the centre of the lobules, the capillaries and necrotic cells containing numerous leucocytes; but amœbæ are not associated with the necrotic process; they are found only in association with the abscesses.

Abscesses are found also in the lungs; they occurred in three out of eight cases, and were seated in the lower lobe of the right lung in every case. In two cases the lung abscess communicated with a liver abscess through a perforated diaphragm. The abscess cavity in the lung was surrounded by consolidated tissue of great extent, embracing the entire lobe. The contents of the abscesses were granular detritus, round lymphoid cells, red blood corpuscles, pus cells, and amœbæ. A definite wall sometimes included the abscess. The amœbæ are more numerous in alveoli immediately adjoining the abscess, and in those places where the abscess is most rapidly advancing, and where there is no limiting wall or connective tissue; but all the neighboring tissues contain them.

Symptoms.—The symptoms of amœbic dysentery are not uniform or characteristic. The cases are either acute or chronic.

Acute Amœbic Dysentery.—There is a catarrhal and an ulcerative stage. In the former a moderate fever and even a slight chill may be present at the onset. The temperature soon returns to normal, or, if it continues high, this points to a severe intestinal lesion or to the involvement of other organs, *i.e.*, liver or lungs. The stools are at first diarrhœal in character, not very numerous, and soon become typically dysenteric. They may, however, be dysenteric from the beginning, with little or no odor, and alkaline in reaction. Amœbæ when present are found in the muco-bloody portions. Pain is usually present in the region of the navel; the entire colon, and frequently the cæcum, are sensitive to pressure. Tenesmus appears coincidently with dysenteric stools, and points to beginning ulceration or to involvement of the lower colon and rectum.

The ulcerative stage may begin with cholera-like symptoms, chill, rapid rise in temperature, diarrhœa and vomiting, burning thirst, and pain in the legs. The tongue is dry and chalky. The stools are usually very numerous, from twenty to thirty in twenty-four hours; they contain much blood and soon become dark brown and fœtid. The colic and tenesmus are severe, and large pieces of necrotic mucous membrane are often passed in the stools. Micturition may also be very painful and the entire abdomen, especially in the region of the colon, is acutely sensitive. In the more severe cases, with gangrenous destruction of the mucous membrane, the temperature rapidly sinks to normal or subnormal, the extremities become cold, and the patient soon dies in col-

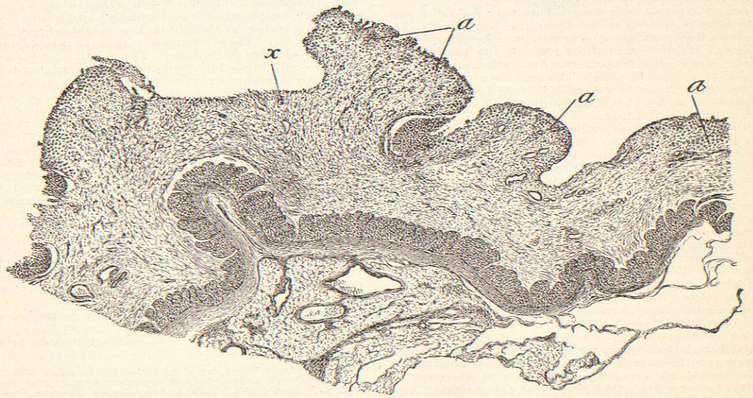

FIG. 1674.—Dysentery Due to Bacillus Dysenteriæ. Section of colon showing thickening of submucosa; *a*, *a*, *a*, superficial necrotic layer of mucosa with areas of hemorrhage; *x*, loss of superficial layer. Enlarged 80 times. (Section from a specimen in the Army Medical Museum.)

lapse. Consciousness is usually maintained to the end. Hemorrhage and perforation of the bowels may occur.

Severe attacks of amœbic dysentery may follow a very mild beginning, and with early and proper treatment even

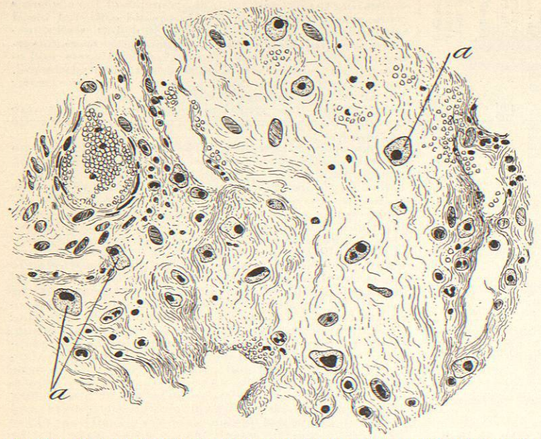

FIG. 1675.—Dysentery Due to Bacillus Dysenteriæ. (Section of area x in Fig. 1674.) Fibrinous infiltration and plasma cells (a, a) in the submucosa. (Enlarged 300 times.)

severe attacks may be cured. Spontaneous cure is very infrequent. The course of the disease is irregular, with frequent intermissions and exacerbations, and shows a strong tendency to become chronic.

Chronic Amœbic Dysentery.—This may follow a subacute attack or be chronic from the beginning. The progress is irregular, exacerbations alternating with periods of improvement during which the patient goes about. If a soldier he performs his duties, entering the hospital from time to time with subacute attacks. During the interval the bowels may be constipated, and the patient looks well and is well nourished. The duration is long. Death results from liver abscess, from perforation peritonitis, from other complications, or from exhaustion.

(2) DYSENTERY DUE TO THE BACILLUS DYSENTERLÆ.—This variety, first described by Shiga in Japan and by Flexner and Barker, and by Strong and Musgrave in the Philippines, is endemic in these localities, but may become epidemic, as noted by Shiga. A small epidemic occurring in Germany was described by Kruse. Flexner has observed one case in the Pennsylvania Hospital, but further study is necessary to decide whether it prevails in this country.

Pathological Anatomy.—The disease resulting from infection with B. dysenteriæ appears in both an acute and a chronic form, each having peculiar lesions.

In the acute form the serous coat of the large intestine is hyperæmic. Near the sigmoid flexure and upper part of the rectum the vessels of the mesentery are distended. The wall of the colon is thickened; the degree and extent of this change bearing some relation to the intensity and duration of the attack. The mucosa and submucosa show a relatively greater increase in size as compared with the other coats. The surface of the mucosa, from the anus to the colon, is red or brownish-red in color, with ecchymotic patches and covered with a layer composed of necrotic tissue, blood, mucus, and epithelium. The mucosa is hypertrophied, forming nodulated areas that give to the surface an irregularly corrugated appearance. These ridges are probably more marked in cases of longer duration and less marked when death takes place within the first week. The change is somewhat greater in the lower colon than elsewhere.

There are no ulcers, but there is an irregular superficial erosion of the surface of the mucosa, that gives to it a worm-eaten appearance.

One of the most characteristic features of this form of dysentery is the extension of the inflammatory process into the ileum for a distance of from 10 to 15 cm. This condition does not exist in amœbic dysentery and is found in one-third of the cases. Peyer's patches and the solitary glands may be moderately swollen.

In the subacute form the mucosa is less red, the solitary follicles are swollen, hemorrhagic, and of a dark red color. The surface of the bowel is uneven, often mammillated, and the necrotic layer is less marked. There are superficial erosions, but no ulcers as in amœbic dysentery, and less thickening of the intestinal walls than in the acute form.

*Pathological Histology.**—The surface of the mucosa is covered with a pseudo-membrane composed of a fine network of fibrin enclosing multinuclear cells and free red blood corpuscles, or, as shown in Fig. 1674, x, the mucosa may also be involved in this process of coagulative necrosis and, in small areas, may separate from the submucosa. To the change in the submucosa is chiefly due the great thickening of the gut. It is very œdematous, appears in places almost homogeneous, and shows a large quantity of fibrin. There is an absence of infiltration with leucocytes. Scattered hemorrhages are abundant and large lymphoid cells and Unna's plasma cells appear in great number, especially in the neighborhood of the blood-vessels. These changes may take place beneath an intact mucous membrane. The serous coat is usually œdematous, and the blood-vessels are injected, but hemorrhages are uncommon. The bacilli and cocci are abundant in the necrotic mucous membrane and are said by Strong to be seen in all the coats.

Symptoms.—The incubation period lasts not over forty-eight hours. The onset is sudden, with frequent stools, consisting of mucus which is soon mixed with blood. The movements accompanied with tenesmus increase in frequency, except toward the end of fatal cases, when they become less frequent. The tongue is coated with a whitish fur and there is excessive thirst. Nausea and vomiting may occur.

The abdomen is not usually distended and there may be tenderness on deep pressure over the colon. The spleen is not often enlarged.

The temperature rises to 102° or 103°, or even to 104° F.; it runs an irregular course and rises or falls before death. The pulse ranges from 100 in the early stages to 150 or more as the disease advances.

Lobar pneumonia, broncho-pneumonia, acute bronchitis, and fibrino-purulent pleurisy are met with.

There is severe headache, and delirium may be present

* The description here given of the pathological changes in bacillary dysentery is based upon a study of specimens brought from Manila by Dr. E. R. Hodge, and now in the Army Medical Museum, Washington. By the kind permission of Major Walter Reed, U. S. A., Curator of the Museum, the drawings, illustrating the text of this article, were made from sections of these specimens.

in prolonged and fatal cases. The urine is decreased in amount and may contain a small amount of albumin. The liver is not enlarged or tender and liver abscess is never found in this form. There may be a moderate leucocytosis.

The progress and duration of the attack are variable. The most violent stage rarely lasts a week. Strong and Musgrave report that out of their 21 acute cases which came to necropsy, 4 patients died on the fourth and 4 on the fifth day of the disease; 10 others died before the fifteenth day, but none under three days' illness. In the cases that are early fatal the extent of the anatomical changes in the intestine indicates how virulent is the inflammatory process. It is remarkable how great a degree of thickening of tissue can be brought about in so short a time. Recovery is rare in the more severe cases.

In the cases that end in recovery improvement may set in at the end of the first week, the patient entering upon convalescence by the end of the second week; or the disease lasts in a subacute form for months, with thin and bloody mucous stools. One of Strong's patients died on the sixty-fourth day.

When convalescence begins, the patient is feeble and much emaciated and improves very slowly.

COMPLICATIONS.—There is no disease which has a larger number of complications than dysentery. Out of 1,537 cases of diarrhœa or dysentery observed in Egypt only 406 were uncomplicated; 1,131 were complicated with other more or less severe affections. The following list includes many of these, which may occur during the progress or as sequelæ of the disease: catarrh of the stomach and small intestines, acute bronchitis, pleurisy, pleuro-pneumonia, gangrene of the lung, albuminuria and anuria, ascites, anasarca, meningitis, thrombosis in the cerebral sinuses, convulsions, cerebral embolism with hemiplegia and aphasia, paraplegia, ulcer of the cornea, abscess of the liver, other lesions in the liver, peritonitis, perityphlitis, periproctitis, perineal fistula, parotitis, erysipelas, rheumatism, neuritis, arthritis, and polyarthritis.

Perforation and peritonitis occurred in 85 out of 880 cases collected by Béranger-Féraud.

DIAGNOSIS.—The recognition of acute dysentery is not difficult if the characteristic symptoms — tormina, tenesmus, and muco-sanguinolent stools—are present. In a case of hemorrhoids blood and mucus are passed, but the hemorrhoidal tumors can be seen, and the discharge follows a normal stool; the blood too is thinner and greater in quantity.

An error of diagnosis is not likely to occur in intussusception, although blood and mucus are passed with straining, but the tumor in the iliac or hypogastric region, the more frequent vomiting and tympanites, and the more rapid collapse are significant symptoms. Sometimes the disease proper may be overlooked when dysentery appears as a complication; this might be the case in typhoid or typhus fever.

The diagnosis of the nature of the lesion is based upon the general symptoms and the character of the stools. Slight illness and small mucous and bloody stools indicate colitis without infiltration and necrosis; but prostration and serious illness, with larger and more liquid stools, which may be offensive, point to infiltration of the deeper layers of the intestinal wall and to destruction of the mucous membrane. The higher up the inflammation is, the more fluid are the stools; the lower down, the more mucus and blood will be present.

In the future the diagnosis of dysenteric processes will not be complete until their nature is made clear by the discovery of the causative micro-organism in each case. This being known, the nature of the lesion and its progress are matters of certainty; upon this knowledge also the prognosis and perhaps in time the treatment will be based.

The diagnosis will be facilitated by the presence or absence of the agglutinative reaction with the pure culture of the B. dysenteriæ. The same test may be made applicable with other yet undiscovered dysenteric flora.

PROGNOSIS.—The fatality of dysentery varies very much with the conditions under which it appears. The mild form in temperate climates is a self-limited disease, tending to recover spontaneously. It rarely assumes a formidable nature outside the tropics, except when it is epidemic; but destructive epidemics are much more rare than formerly. In England the reduction in the death rate in dysentery has been marked since 1850. In 1880 the mortality was six hundred and sixty-eight times less than it was forty or fifty years before. In the United States the total death rate from dysentery, in 1850, was

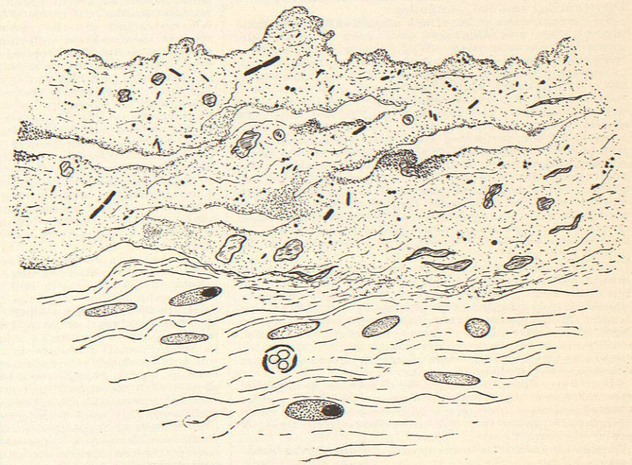

FIG. 1676.—Dysentery Due to Bacillus Dysenteriæ. Bacilli and cocci in superficial necrotic layer. (Methylene blue staining.) Enlarged 500 times. (Section from a specimen in the Army Medical Museum.)

20,556, or 6.32 per cent. of the total mortality; in 1860, 10,468, or 2.65 per cent.; in 1870, 7,912 or 1.60 per cent.; and in 1880, out of a total death rate of 756,893, there were 10,825 deaths from dysentery.

The death rate from dysentery in Europe is from six to eight per cent. (Hirsch) of the number attacked. In the

garrisons in France there were 7 deaths in 589 cases, or 1.18 per cent. Cases of a higher degree of severity last for from three to four weeks, although complete recovery does not take place for two or three weeks later. In epidemic dysentery death may occur in three or four days, or at the end of the second week, or later.

In individual cases the prognosis depends upon the previous state of health, intercurrent diseases, as scurvy, malaria, etc., and recent residence in the country. Unfavorable symptoms are delirium, distress in the epigastrium, dyspnœa, vomiting, lowering of the body temperature, hiccough, cyanosis, cold skin and extremities, with feeble, rapid pulse, offensive thin or bloody discharges, disappearance of pain with involuntary stools, aphthæ, erysipelas, gangrene of lung, ulcer of the cornea, or suppression of the urine.

In epidemics in temperate climates, the mortality varies from 7 to 15 per cent. From 1841 to 1846, in the epidemics which prevailed in France, one-tenth of those attacked died. In 1836, 1837, and 1838, 25 per cent. died, and in 1857 one out of every five died.

In tropical epidemic dysentery, the mortality is from 20 to 30 per cent. In Bombay it is 9 per cent.; in Hong-Kong, 20 per cent. A mortality of 35 or 40 per cent., and even one of from 60 to 80 per cent. has been reached. In the Manila cases previously referred to, the mortality was 9.4 per cent.

Treatment.—The preventive treatment of dysentery consists in avoiding all the known causes, and in living under the best hygienic influences. Measures should be taken looking to the complete drainage of moist soil, the procuring of pure drinking-water, the complete disinfection and removal of all excreta, and the prevention of overcrowding and bad ventilation.

In the treatment of the attack attention should be paid to ventilation and cleanliness, and to the hygienic rules governing the sick-room. With the earliest symptoms the patient should be put to bed (and in a single bed, for greater convenience). The bed covering should be warm, and even in warm weather a blanket should be used, with a view of preventing chilling of the surface by diurnal variations of temperature. The feet especially should be kept warm.

Sponging the surface of the body with cool or tepid water, with vinegar, or with alcohol gives comfort by removing the sensation of burning heat in the skin. The anal region should be carefully and constantly cleansed with a warm disinfectant solution, and anointed with vaseline or other ointment. Hot hip baths have been used with advantage in relieving the tenesmus of dysentery. Hot poultices to the abdomen, or hot fomentations covered with oiled silk, give some comfort to the patient.

The diet best suited to dysentery is one which is digested, as far as possible, in the stomach, and which has but little waste; therefore concentrated broths, or milk, should be given in small quantities at intervals of two hours. Milk may be given pure or diluted with Vichy water, lime water, barley water, or rice water. In the earlier stages of severer cases with high fever, milk should be given in very small quantities, from one to four drachms, every half-hour or hour.

To relieve thirst, mucilaginous drinks, as gum-arabic water, flaxseed tea, the white of egg drink, are advisable. Orange or grape juice is not objectionable, and in cases of scorbutic taint fruit juice, or even fresh fruit, is positively curative.

Alcohol is called for in many cases to sustain the feeble pulse.

Diet alone, or combined with rest, will, in many cases of dysentery, bring about a cure in a few days. In severer cases, although we have no specific, yet drugs are of service in relieving pain and in shortening the attack.

Laxatives.—The laxative method appears so rational that it has gained a firm foothold in popular favor; it is only liable to lose ground by being supported as a "method" and not as an aid to a combined effort to disinfect the intestinal canal by removing contents that irritate mechanically and through chemical decomposition.

If the case is seen early and if the stools are small, consisting of blood and mucus, the treatment can well be begun by a saline laxative, as magnesium sulphate, sodium sulphate, potassium and sodium tartrate, or by castor oil, the dose to be repeated until a distinct effect is produced. In certain cases, especially in children, calomel acts very well and is to be preferred. The presence of hard fæcal matter in the stools and the occurrence of watery fæcal movements are evidences that the laxative has acted sufficiently.

As purgation increases peristaltic movement and irritates the mucous surface, it must, to a certain extent, be harmful in all cases, and is not to be advised if the stools are copious and fluid or if there is much preliminary diarrhœa. The question of repeating the purgative requires some judgment, and it is here that the belief in purgatives as a "method" may do much harm. At the end of a week or later a saline laxative should be given if, with subsidence of the inflammation, there are no natural fæcal movements.

Opium.—The pain and the frequent stools suggest the frequent use of opium, and in moderate doses this drug is of service; the objections to it are the danger of arresting peristalsis completely and causing an accumulation in the bowel of matters that are much better expelled. It is in cases with large, liquid, putrid stools that opiates do positive harm by arresting the discharges. The indications for opium are intense abdominal pain, sleeplessness from pain, or very frequent stools. Dover's powder, deodorized tincture of opium, and morphine hypodermically, are the best forms of administration. For many reasons the last is to be preferred, the doses being sufficient to subdue great suffering and to keep the nervous system in comparative repose. But any effect approaching narcotism should be most carefully avoided by giving small doses at first and at safe intervals afterward.

There is very little to be said in approbation of any other medicine given by the mouth. The treatment by ipecacuanha retains its place in the books, but the Anglo-Indian method with large doses has never had a firm place in the practice of this country, and even its warmest supporters seem to be losing confidence in its value. Small doses of the drug are spoken of favorably, but there is no reason to think that they have any curative effect. The same may be said of the numerous drugs suggested from time to time. Astringents are without effect, and are harmful because they derange digestion. Bismuth in large doses may prove of use, and in any event does no harm.

Antiseptics given by the mouth, although not so efficacious as when injected into the lower bowel, may be employed to supplement their more direct action.

The value of calomel in the treatment of dysentery no doubt resides in its antiseptic properties, and the time-honored combination with Dover's powder has some rational support; but to be of any service the drug should be given in small doses and kept up, in this combination, for several days. Corrosive sublimate in small doses, from gr. $\frac{1}{12}$ to gr. $\frac{1}{9}$ (0.00054–0.0011) every hour, is employed with the same object in view.

Other intestinal antiseptics can be much more readily and thoroughly used by injection and irrigation.

Treatment by Suppositories and by Rectal and Colon Injection and Irrigation.—The use of suppositories containing opiates and astringents, although at one time a common practice, is much less in favor than formerly. It is not desirable to limit to too great an extent the tendency of the rectum to expel its contents. The disease cannot be cured by putting a stop to dysenteric discharges, and therefore suppositories used for this purpose are not to be recommended. After the action of a purgative an opiate suppository may arrest too free purgation and keep the bowel at rest with benefit; after irrigation, also, advantages may, upon the same principle, follow the introduction of a suppository, but the doses of opium should never be large. In attempting to control tenesmus the temptation is frequently to give too large doses, which induce narcotism and which involve danger.

These same rules hold against following the once popular method of using injections of starch water and laudanum, which may be of service with the limitations just mentioned, but as a routine treatment the plan is not to be advised.

The injection into the bowels of large quantities of warm or cold water has many advocates. The sedative effect of water of a high or low temperature upon an inflamed mucous membrane is of undoubted service in dysentery. The only objection is to be found in the danger of overdistending the inflamed gut. Irrigation, by cleansing the ulcerated surfaces, washing away the decomposing contents of the bowel, and destroying bacterial life, gives promise of accomplishing a great deal more than would the mere injection of fluids into the bowel.

Cool or hot water may be used for irrigation, but if an antiseptic agent be added to the water there is an additional benefit.

Antiseptic Irrigation may be practised with solutions of mercuric chloride 1 to 5,000, quinine 1 to 5,000 or 1 to 2,000, salicylic acid, tannin, thymol, carbolic acid, sulphocarbolate of zinc, boracic acid, etc. The use of bichloride solution must always be attended with a certain amount of danger, and precautions should be taken to secure a free exit.

Irrigation can be practised in the rectum or colon; in either case a soft-rubber tube is passed into the rectum or is gently and gradually forced upward through the sigmoid flexure. This is by no means an easy task, as the instrument so readily turns on itself; an occasional examination ought to be made with the finger to see if this has happened. Experiments on the cadaver show that the smaller-sized rectal and colon tubes do not make their way as readily as those of larger size. The habit of injecting water, as the tube progresses, favors twisting; progression is more easily made if the bowel is empty, as the mucus-covered wall guides the instrument in the proper direction. In many cases the colon cannot be reached, and there is danger of perforating the ulcerated bowel if the efforts are continued for too long a time. One must be content, therefore, to pass the tube into the sigmoid. The fluid is then allowed to flow in from a fountain syringe, or is thrown in by a Davidson; when from six to eight ounces have entered, the fluid is allowed to escape through the same tube. This process is repeated until a quart or more has been used, or until the water escapes perfectly clear. If the patient is in the dorsal position or on the left side, with the hips raised, gravity favors the entrance of the fluid. In most cases, and especially in bad cases, with putrescent fluid discharges, it is safer never to attempt to push the instrument beyond the rectum.

The frequency of the irrigation should vary with the number and character of the stools. Large, frequent, putrid stools call for more frequent cleansing of the bowel. If the discharges consist of small masses of blood-stained mucus there is less need of frequent irrigation and less benefit is likely to come from it. As a rule the benefit depends more upon the frequency of the treatment than upon the properties of the antiseptic employed. (See also article on *Enteroclysis.*)

Symptomatic treatment is required for sleeplessness, tormina, and tenesmus, and morphine, to be administered hypodermically, is the most satisfactory remedy. Small doses (gr. $\frac{1}{6}$ to $\frac{1}{4}$) only are safe and these can be repeated at intervals of from two to three hours until some effect is noticed, but great care should be taken to avoid narcotism, to which dysenteric patients are liable.

The strength of the heart can be sustained by strychnine and by nitroglycerin, when called for in emergencies.

Toward the end of the attack solids are harmful when given too early. These should be deferred until the stools have been normal for some days. Rest during convalescence favors a more rapid return of strength.

Great care to avoid exertion and imprudent eating during the convalescence from an acute dysentery, is the best way to prevent the establishment of the chronic forms. If there are evidences of the existence of unhealed ulcers patients should be kept at rest, on a simple diet, for a prolonged period. Colon irrigation with antiseptic solutions or with a solution of silver nitrate, five grains to the pint, is the most rational method of treatment. Sometimes a change of climate is of use.

The treatment of the acute and chronic forms of amœbic dysentery is not invariably satisfactory and often does not appear to have much influence upon the course of the disease. The patient should be kept in bed upon a restricted diet even in the absence of severe symptoms. Warm rectal irrigations with a 1 to 5,000 solution of quinine, which, as shown by Lösch, readily destroys amœbæ outside of the body, should be given two or three times a day, and the patient should attempt to retain the fluid for ten or fifteen minutes. The strength of the solution should be gradually increased to 1 to 500. The corrosive sublimate solution or a solution of nitrate of silver, thirty grains to the quart, may be substituted, but neither of these is so efficient as the quinine, which being absorbed by the tissues undoubtedly destroys the amœbæ embedded in the deeper layers of the intestine.

An antitoxin prepared with Shiga's bacillus by Kitasato in Tokyo is being extensively used in Japan for the cure of bacillary dysentery. No statistics are as yet obtainable, but it is claimed that the results are second only to those of the antitoxin treatment of diphtheria.

William W. Johnston.

DYSIDROSIS. See *Pompholyx.*

DYSMENORRHŒA.—The term dysmenorrhœa (from the inseparable particle δύς, with difficulty, μήν, a month, and ῥέω, I flow) is used in its strict etymological sense, that of painful menstruation; but it should be applied only to menstruation accompanied by severe pain, as distinguished from the moderate aching usually attendant upon the function. Dysmenorrhœa is commonly considered to be only a symptom, and not a disease, and that view is doubtless true of the generality of cases; but in many instances no structural pathological condition can be discovered to account for it, and it may then be looked upon as a neurosis, or perhaps as an expression of the rheumatic diathesis.

Almost all sytematic writers divide dysmenorrhœa into several varieties, founding their division upon what they conceive to be its varying pathology; but a few authors will have it that the affection is necessarily due to obstruction to the escape of blood from the uterus. Those who admit a number of forms of dysmenorrhœa follow a nomenclature intended to express various pathological states, such as the neuralgic, the spasmodic, the congestive, the inflammatory, the membranous, the obstructive, etc.; while others employ names designed to indicate the seat of the fundamental morbid condition, such as constitutional, ovarian, uterine, etc. Both classes of writers enumerate the signs and symptoms by which, as they maintain, the particular forms of the disease may be diagnosticated. Practically, these diagnostic points are not much to be relied upon, and we can only say with certainty: 1. That women who, so far as we can discover, are in perfect health in other respects, both constitutionally and locally, suffer from dysmenorrhœa; although it must be admitted that the great majority of sufferers show evidence of a depraved constitutional state. 2. That women of every sort of systemic ill health escape this ailment. 3. That the affection is found associated with every abnormity of the sexual apparatus. 4. That, except positive occlusion of the uterine canal, there is no condition of the parts concerned that invariably gives rise to it. It will be seen from all this that the relations between dysmenorrhœa and its causes are very diverse and but imperfectly understood, that no single theory of its causation will apply in all cases, and that no one of the nosological systems covers the ground satisfactorily