XX. DYSENTERY.

Definition.—Under this clinical term several different forms of intestinal flux are described, which are characterized by frequent stools, and in the acute stage are accompanied by tormina and tenesmus. Anatomically there are inflammation and usually ulceration of the large bowel.

Etiology.—Dysentery is one of the four great epidemic diseases of the world. In the tropics it destroys more lives than cholera, and it has been more fatal to armies than powder and shot.

While especially severe in the tropics, sporadic cases constantly occur in more temperate climates, and under favoring circumstances epidemics are found even in the more northern countries, such as Canada and Norway. It has become less frequent of late years, owing to improved sanitary conditions. The statistics of the Montreal General Hospital, for the twenty years ending May 1, 1889, show a remarkable decrease in the disease. In the decade ending May, 1879, 150 cases were admitted; whereas in the last ten years there have been only 31 cases admitted. There has been a similar decrease at the Pennsylvania Hospital.

In the Southern cities of this country dysentery is more prevalent; even when not epidemic, sporadic cases are common. In Baltimore it prevails every summer, and has on several occasions been epidemic.

Epidemics of dysentery have occurred in the United States for more than a century, and Woodward has collected the data which show the various outbreaks. Perhaps the most serious was that which prevailed in various localities from 1847 to 1856. During the war of secession the disease existed to an alarming extent in both armies. According to Woodward's report,* there were in the Federal service in all 259,071 cases of acute and 28,451 cases of chronic dysentery. Probably a considerable proportion of the 182,586 cases of chronic diarrhœa should also come in this category. The decennial census reports since 1850 show a progressive decrease in the total number of deaths from this disease. It prevails most extensively in the summer and autumn. Sudden changes of temperature appear more harmful than variations in moisture. The effluvia from decomposing animal matter have been thought by some to predispose to or even to cause the disease. That dysenteric affections are more frequent in malarial localities has long been known, and is probably connected with external conditions favoring their development. With reference to the influence of drinking-water, Woodward is doubtless correct in stating that the effects of dissolved mineral matters have been greatly exaggerated. On the other hand, from the days of the old Greek physicians, it has been held that the impurities in the stagnant water of marshy districts and

* Medical and Surgical History of the War of the Rebellion, Medical, vol. ii; the most exhaustive treatise extant on intestinal fluxes—an enduring monument to the industry and ability of the author.

ponds may give rise to diarrhœa and dysentery. Here, however, it is probably not the vegetable impurities which are directly causative, but the organic matter renders the water a more favorable medium for the development of organisms which may cause disease.

Dyspeptic conditions, particularly those caused by the ingestion of bad food and unripe fruit, seem to predispose to the disease. Great stress has been laid by German authorities on the importance of constipation as a causal factor in dysentery.

Dysentery occurs at all ages. There is no race immunity. The contagiousness of the disease is doubtful. The experience of the civil war is decidedly against it, but the possibility, as with typhoid fever, must be acknowledged.

Clinical Forms.—(*a*) **Acute Catarrhal Dysentery.**—This may occur sporadically or endemically, and is the variety most frequently found in temperate climates.

Morbid Anatomy.—The lesions are confined to the large bowel, and sometimes the ileum also is involved. The mucous membrane is injected, swollen, and often covered with tenacious blood-stained mucus. The most striking feature is the enlargement of the solitary follicles, which stand out prominently from the mucous membrane. In very acute forms, as in children, the picture is that of an acute follicular colitis. In more protracted cases the follicles suppurate or are capped with an area of necrotic tissue. In other instances the sloughs have separated and the entire colon presents numerous ulcers, most of which have developed from the follicles, and others have resulted from necrosis and sloughing of the intervening tissue.

Symptoms.—There may be preliminary dyspepsia or slight pains in the abdomen. Chills are rare. Diarrhœa is the most constant initial symptom, and at first is not painful. Usually within thirty-six hours the characteristic features of the disease develop—abdominal pain of a colicky, griping character, frequent stools, which are passed with straining and tenesmus; the constitutional disturbance is variable, and in mild cases may be slight. The temperature range is not high, but at the outset the fever may rise to 102° or 103°. The tongue is furred and moist, and as the disease progresses becomes red and glazed. Nausea and vomiting may be present, but as a rule the patient retains nourishment. The constant desire to go to stool and the straining or tenesmus are the most distressing symptoms. The abdomen may be flat and hard. The thirst is often excessive. The stools in this variety of dysentery have the following characters: During the first twenty-four or forty-eight hours they consist of more or less clear mucus and blood mixed with small fæcal scybala. After this they become purely gelatinous and bloody, and are small and frequent, from fifteen to two hundred in twenty-four hours, according to the severity of the case. About the end of the first week the mucus becomes opaque, the proportion of blood diminishes, and grayish or brownish shreddy material

appears in the stools, which become gradually reduced in frequency. Some of the stools at this time may be wholly composed of a greenish pultaceous material and mucus. As the disease subsides, fæcal matter again appears in the stools, increasing in amount until fully formed fæces are passed, containing no mucus or blood. Microscopical examination of the glairy bloody stools shows red blood-corpuscles, few or many leucocytes, and constantly large, swollen, round or oval epithelioid cells, containing fat-drops and vacuoles. Bacteria are scarce; occasionally the *cercomonas intestinalis* is seen in large numbers.

Course of the Disease.—The milder cases run a course, as Flint has shown, of about eight days; severer ones rarely terminate within four weeks. Recovery may be imperfect, and the affection occasionally becomes chronic. In this form the complications are not numerous; peritonitis and liver abscess are extremely rare. Except in young children, a majority of the cases terminate favorably.

(*b*) **Tropical Dysentery—Amœbic Dysentery.**—This form of intestinal flux is characterized by irregular diarrhœa and the constant presence in the stools of the *amœba coli* (Lösch), *amœba dysenteriæ* (Councilman and Lafleur). It is this variety which prevails extensively in the tropical and subtropical regions, and which proves so fatal in epidemic form. The amœba is a unicellular, protoplasmic, motile organism, from ten to twenty micromillimetres in diameter, consisting of a clear outer zone, ectosarc, and a granular inner zone, endosarc, containing a nucleus and one or more vacuoles. It was first described by Lambl in 1859, and subsequently by Lösch, who considered it the cause of the disease. In the endemic dysentery of Egypt, Kartulis, in 1883, found these amœbæ constantly in the stools, in the intestines, and in the liver abscesses. He was afterward enabled to cultivate them in straw infusion, and to produce the disease artificially in cats and dogs. In 1890 I reported a case of dysentery with abscess of the liver originating in Panama, in which the amœbæ were found in the stools and in the pus from the abscess; and lately Councilman and Lafleur* have described the clinical features and anatomical lesions in a series of cases of this form of dysentery in my wards. Dock, in Galveston, has demonstrated their presence in a number of cases, and Musser has found them in Philadelphia. The disease is very common in tropical and subtropical countries. It is, however, found more or less widely distributed throughout Europe and North America. The sources of infection are not known, but it seems probable that one of them is drinking-water.

Morbid Anatomy.—The lesions are found in the large intestine, sometimes in the lower portion of the ileum. Abscess of the liver is a common sequence. Perforation into the right lung is not infrequent.

Intestines.—The lesions consist of ulceration, produced by preceding

* Johns Hopkins Hospital Reports, vol. ii.

infiltration, general or local, of the submucosa, the general infiltration being due to an œdematous condition, the local to multiplication of the fixed cells of the tissue. In the earliest stage these local infiltrations appear as hemispherical elevations above the general level of the mucosa. The mucous membrane over these soon becomes necrotic and is cast off, exposing the infiltrated submucous tissue as a grayish-yellow gelatinous mass, which at first forms the floor of the ulcer, but is subsequently cast off as a slough.

The individual ulcers are round, oval, or irregular, with infiltrated, undermined edges. The visible aperture is often small compared to the loss of tissue beneath it, the ulcers undermining the mucosa, coalescing, and forming sinuous tracts bridged over by apparently normal mucous membrane. According to the stage at which the lesions are observed, the floor of the ulcer may be formed by the submucous, the muscular, or the serous coat of the intestine. The ulceration may affect the whole or some portion only of the large intestine, particularly the cæcum, the hepatic and sigmoid flexures, and the rectum. In severe cases the whole of the intestine is much thickened and riddled with ulcers, with only here and there islands of intact mucous membrane.

The disease advances by progressive infiltration of the connective-tissue layers of the intestine, which produces necrosis of the overlying structures. Thus, in severe cases there may be in different parts of the bowel sloughing *en masse* of the mucosa or of the muscularis, and the same process is observed, but not so conspicuously, in the less severe forms.

In some cases a secondary diphtheritic inflammation complicates the original lesions.

Healing takes place by the gradual formation of fibrous tissue in the floor and at the edges of the ulcers, which may ultimately result in partial and irregular strictures of the bowel.

Microscopical examination shows a notable absence of the products of purulent inflammation. In the infiltrated tissues polynuclear leucocytes are seldom found, and never constitute purulent collections. On the other hand, there is proliferation of the fixed connective-tissue cells. Amœbæ are found more or less abundantly in the tissues at the base of and around the ulcers, in the lymphatic spaces, and occasionally in the blood-vessels.

The lesions in the *liver* are of two kinds: firstly, local necroses of the parenchyma, scattered throughout the liver and possibly due to the action of chemical products of the amœbæ; and, secondly, abscesses. These may be single or multiple. When single they are generally in the right lobe, either toward the convex surface near its diaphragmatic attachment, or on the concave surface in proximity to the bowel. Multiple abscesses are small and generally superficial. In an early stage the abscesses are grayish-yellow, with sharply defined contours, and contain a spongy necrotic material, with more or less fluid in its interstices. The larger abscesses have

ragged necrotic walls, and contain a more or less viscid, greenish-yellow or reddish-yellow purulent material mixed with blood and shreds of liver-tissue. The older abscesses have fibrous walls of a dense, almost cartilaginous toughness. A section of the abscess wall shows an inner necrotic zone, a middle zone in which there is great proliferation of the connective-tissue cells and compression and atrophy of the liver-cells, and an outer zone of intense hyperæmia. There is the same absence of purulent inflammation as in the intestine, except in those cases in which a secondary infection with pyogenic organisms has taken place. The material from the abscess cavity shows chiefly fatty and granular detritus, few cellular elements, and more or less numerous amœbæ. Amœbæ are also found in the abscess walls, chiefly in the inner necrotic zone. Cultures are usually sterile. Lesions in the lungs are seen when an abscess of the liver—as so frequently happens—points toward the diaphragm and extends by continuity through it into the lower lobe of the right lung. The gross and microscopical appearances are similar to those of the liver.

Symptoms.—The onset may be sudden, as in catarrhal dysentery, or gradual, beginning as a trifling and perhaps transient diarrhœa. In severe gangrenous cases the abrupt onset is more common. The subsequent course is a very irregular diarrhœa, marked by exacerbations and intermissions, and progressive loss of strength and flesh. There is moderate fever as a rule, but many cases are afebrile throughout the greater part of their course. Abdominal pain and tenesmus are frequently present at the onset, especially in severe cases, but may be entirely absent, and vomiting and nausea are only occasionally observed. The stools vary very much in frequency and appearance in different cases and at different periods in the same cases. They may be very frequent, bloody, and mucoid at the outset, as in catarrhal dysentery; but their main characteristic, when the disease is well established, is fluidity. From six to twelve yellowish-gray liquid stools, containing mucus and occasionally blood in varying proportions, are passed daily for weeks. Actively moving amœbæ are found in these stools, more abundantly during exacerbations of the diarrhœa, and disappear gradually as the stools become formed.

Abscess of the liver, and especially of the liver and lung, is a frequent and formidable complication. In India it occurs once in every four or five cases.

The duration of the disease in uncomplicated cases varies from six to twelve weeks. Recovery is tedious, owing to anæmia and muscular weakness, often delayed by relapses, and there is in all cases a constant tendency to chronicity. The mortality is much higher than in catarrhal dysentery. A fatal issue is due either to the initial gravity of the intestinal lesions, to exhaustion in prolonged cases, or to involvement of the liver.

(*c*) **Diphtheritic Dysentery.**—A form of colitis or entero-colitis in which areas of necrosis occur in the mucous membranes, which on separation leave ulcers. This occurs: (*a*) As a *primary* disease coming on acutely and sometimes proving fatal. In its milder grades the tops of the folds of the colon are capped with a thin, yellow exudate. In severer forms the colon is enormously enlarged, the walls are thickened, stiff, and infiltrated, and the mucosa, from the ileo-cæcal valve to the rectum, represented by a tough, yellowish material, in which on section no trace of the glandular elements can be seen. It is an extensive necrosis of the mucosa. There are cases in which this necrosis is superficial, involving only the upper layers of the mucous membrane; but in the most advanced forms it may be, as in the description by Rokitansky, "a black, rotten, friable, charred mass." The areas of necrosis may be more localized, and large sloughs are formed which may be a half to three fourths of an inch in thickness and extend to the serosa. There are instances in which this condition is confined to the lower portion of the large bowel. A sailor from the Mediterranean was admitted to the Montreal General Hospital under my care with symptoms resembling typhoid fever. The autopsy showed enormous sloughs in the rectum and in the sigmoid flexure, but scarcely any disease in the transverse or ascending colon. In cases which last for many weeks the sloughs separate and may be thrown off, sometimes in large tubular pieces.

(*b*) *Secondary Diphtheritic Dysentery.*—This occurs as a terminal event in many acute and chronic diseases. It is not infrequent in chronic heart affections, in Bright's disease, and in cachectic states generally. In acute diseases it is, as pointed out by Bristowe, most frequently associated with pneumonia. Anatomically there may be only a thin, superficial infiltration of the upper layer of the mucosa in localized regions, particularly along the ridges and folds of the colon, often extending into the ileum. In severer forms the entire mucosa may be involved and necrotic, sometimes having a rough, granular appearance. In the secondary colitis of pneumonia the exudation may be pseudo-membranous and form a firm, thin, white pellicle which seems to lie upon, not within, the mucous membrane.

Symptoms.—The clinical features of diphtheritic dysentery are very varied. In the acute *primary* cases the patient from the outset is often extremely ill, with high fever, great prostration, pain in the abdomen, and frequent discharges. Delirium may be early and the clinical features may closely resemble severe typhoid. I have, on more than one occasion, known this mistake to be made. The abdomen is distended and often tender. The discharges are frequent and diarrhœal in character, and tenesmus may not be a striking symptom. Blood and mucus may be found early, but are not such constant features as in the follicular disease. This primary form is very fatal, but the sloughs may separate and the condition become chronic. In the *secondary* form there may have been no symptoms to attract attention to the large bowel. In a majority of the cases the patient has a diarrhœa—three, four, or more movements in the