intensity of the neuritis forms no accurate guide to a knowledge of the nature or the size of the new growth, and its usefulness in localization is comparatively small.

The condition known as albuminuric retinitis is of more value in prognosis than from a diagnostic point of view, and this on account of two things: (1) The same clinical picture is sometimes present without the kidneys being affected, and (2) when it is present in renal disease there are usually other evidences of this affection.

As regards life-prognosis, however, the detection of albuminuric retinitis is most important. Amongst the poorer classes this retinal complication means that the end of life is drawing very near, and comparatively few survive more than a year. Among the well to do, where every care in diet, clothing, and attention to general hygiene can be exercised, the prognosis is not so bad, but even among these the probability of life does not exceed eighteen months to two years. The only exceptions to this unfavourable prognosis are when the retinitis accompanies acute nephritis, such as the post-scarlatinal, or when it occurs in connexion with the albuminuria of pregnancy.

There is one other ophthalmoscopic change which I must mention, and its significance must be recognized more and more as the importance of the early detection and proper treatment of general arterio-sclerosis is accepted by our profession. In the retina we have the unique privilege of being able to observe with care and accuracy these minute arteries which are so often the first to tell the tale of vascular degeneration, and the successive alterations in retinal vessels have now been followed and described with sufficient detail to enable us, in any given case, to give a shrewd guess concerning the stage at which the degeneration has arrived. That by this means we can predicate with an approach to certainty the condition of small arteries in other more vitally important tissues is no longer in dispute, and it is by this observation that we are often first able to diagnose granular kidney disease, and, at one late stage of the retinal arterial change, to anticipate with more than mere probability the imminence of grave evidences of cerebral vascular disease.

I have purposely given no verbal description of these ophthalmoscopic changes, for I feel strongly that such details by themselves are apt to be misleading, and at best fall far short of the actual pictures presented by the diseases in question. It is to instances of the affections mentioned that I would refer you, and I can assure you they are not uncommon.

It may be, Gentlemen, that I have appeared to you in these remarks to have unduly magnified the importance of one special branch of your work. I know that you have many points to think of and many facts to bear constantly in mind. My aim has been, not to add to these, but to present for your consideration a few clinical lessons gleaned from ophthalmology which may be of some service to you, and, if you think well of them, to induce you to take advantage of the opportunities this ophthalmic department will undoubtedly afford.

FOREIGN STUDENTS IN GERMAN UNIVERSITIES.—The number of matriculated foreign students on the books of the universities of Germany during the last winter semester was 4,151, being 9.2 per cent. of the total number of students. These foreign students were distributed among the several universities as follows: Heidelberg, 1,189 (16.1 per cent.); Leipzig, 662 (14.8 per cent.); Jena, 186 (4.6 per cent.); Berlin, 1,189 (14.5 per cent.); Königsberg, 134 (11.8 per cent.); Halle, 254 (11.3 per cent.); Freiburg, 164 (9.4 per cent.); Göttingen, 160 (9.2 per cent.); Munich, 416 (8.9 per cent.); Giessen, 84 (7.7 per cent.); Strassburg, 96 (5.8 per cent.); Greifswald, 43 (5.2 per cent.); Würzburg, 67 (4.7 per cent.); Marburg, 60 (3.9 per cent.); Tübingen, 59 (3.9 per cent.); Breslau, 77 (3.9 per cent.); Bonn, 88 (2.9 per cent.); Erlangen, 28 (2.6 per cent.); Rostock, 13 (2 per cent.); Kiel, 11 (1.4 per cent.); Münster, 11 (0.7 per cent.). Of the foreigners, 1,890 were from Russia, 681 from Austria-Hungary, 341 from Switzerland, 144 from Great Britain, 139 from Bulgaria, 83 from Roumania, 61 from Servia, 58 from France, 57 from Holland, 53 from Luxemburg, 47 from Greece, 40 from Turkey, 33 from Italy, 32 from Sweden and Norway, 23 from Spain, 19 from Belgium, 9 from Portugal, 5 from Denmark, 1 from Montenegro. In addition to these America sent 302 students, Asia 113, Africa 13, and Australia 11.

ACUTE COLITIS AND ULCERATIVE COLITIS.*

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THERE are few diseases so grave as acute colitis of which our knowledge is so deficient, and I think, therefore, the records of the following cases will be of interest:

CASE I.

A. W., a youth of 19, who had enjoyed good health except for rheumatic attacks, commenced on July 14th, 1894, to have diarrhoea, soon becoming severe; the motions were liquid and slimy, and there was pain after defaecation, but no tenesmus. The diarrhoea increased, the bowels being open eight times a day, and on August 13th he gave up work, melaena coming on, which increased till half a pint "like treacle," he says, passed with each motion, and on August 20th even more. He came into St. Mary's Hospital on August 23rd; he had vomited twice before admission. The tongue was quite clean. There was tenderness over the whole abdomen; the transverse colon was distended, and the abdominal muscles were kept tense. There was pain in the left iliac fossa before and after defaecation. The stools were loose, dark, and foul-smelling, with much mucus and no blood. He was ordered bismuth carbonate and opium, but continued passing seven stools a day, and soon experienced much pain over the sigmoid flexure and right iliac fossa. Occasional slight melaena till the 26th, when a clot passed the size of a crown piece.

On the 27th he complained of sudden severe pain in the splenic region; the spleen was not enlarged, and probably the pain arose in the splenic flexure of the colon. On the 28th there was much more blood in the stools and some pus. Up to the 29th he had only vomited twice, but he now commenced to return everything that was swallowed, and even enemata were not retained; his general condition was much worse, with prostration. On the 31st there was great pain and tenderness, especially over the left lumbar and iliac regions; bowels open, but merely mucus passed.

On September 1st, the abdomen, previously distended, became retracted; vomiting persisted, and on the 3rd hiccough came on; his pulse, previously only 84, ran up to 128 per minute on the 6th, and he died of syncope on the 8th, the hiccough having persisted from the 3rd until death; the temperature had been subnormal throughout. All along there had been some bladder strangury. *Post mortem*, on the outer surface of the colon numerous dusky patches of congestion; on opening it, it was seen to be intensely congested throughout, but especially the sigmoid flexure, and numerous ulcers were found in the caecum, ascending, transverse, and descending colon; in the sigmoid flexure the greater part of the surface was denuded of mucous membrane, the ulcers being only separated by narrow bridges of mucosa, which was quite shreddy. The upper half of the rectum showed ulcers, but the lower none; nor were there any in the small intestine or in the stomach; no peritonitis.

CASE II.

C. J., aged 48, a plumber, on December 7th, 1905, began to suffer from vomiting with abdominal pain and tenderness especially marked over the caecum and descending colon and most severe at night; there was much flatulent distension and slight melaena. After lying at home for three weeks under medical treatment, during the whole of which time he had hiccough and pyrexia, he came into St. Mary's Hospital on January 1st, 1906. He said that he had been perfectly well up to the date of his illness, which he at first attributed to some beefsteak pudding he had eaten; his doctor had thought it was lead colic.

On admission he lay on his back, with sunken eyes, groaning loudly, and incessantly coughing; he retched and vomited up most of the liquids given him; the colon was somewhat but not greatly distended; there was some tenderness over the sigmoid region and also at a point over the position of the hepatic flexure. His breath smelt almost gangrenous and his pulse could hardly be felt nor his heart sounds heard; he was clear in mind; the urine contained bile but no albumen; his temperature was 100° on admission, but fell to 96° on the next day, when he died of collapse.

The necropsy showed the whole of the descending colon to be in a state of sloughy ulceration exposing the muscular fibres; there was no perforation and no peritonitis.

CASE III.

L. M., a lady's maid, was attacked on July 17th, 1888, with severe lumbar pain, feeling of severe illness and diarrhoea. She had been previously in excellent health, had eaten nothing unusual, but had got wet in a thunderstorm the day before; there was nausea but no vomiting. She took to bed at once under medical treatment, but the diarrhoea increased, with occasional griping in the left hypochondriac region; there was no headache or epistaxis. She came into St. Mary's Hospital

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on August 11th with flushed face and hectic look, slight moist fur on the tongue; motions relaxed, offensive, light-coloured, semi-liquid; abdomen everywhere resonant, left side more full and distended than the right; pulse 120, soft. Her temperature during the next week ranged from 98° to 102.6° daily; the colon became markedly distended; there was general but not extreme tenderness over the abdomen, most marked over the position of the ileo-caecal valve. Rectal examination revealed nothing abnormal, but the passage of a rectal tube gave such pain as not to be repeated. The abdomen, though always distended, was soft and doughy; diarrhoea was very intractable, the motions being most offensive, but there was never any melaena. Pyrexia persisted, about 101° to 102° each day. She took liquid food well, but she sweated freely and gradually emaciated.

On August 24th—that is, the twenty-seventh day of illness—the abdominal pain and distension increased and her downward progress became more rapid. On the 26th she vomited once, purpuric spots appeared and oedum albicans in the mouth. The mind remained perfectly clear throughout, but she died of exhaustion on August 28th, after an illness of forty-one days.

The necropsy showed morbid conditions entirely confined to the colon, which in its whole length was thinned to an extraordinary degree; it was very pale and absolutely dried up; this desiccation was most striking; it was honey-combed by perforations, and where the wall was not perforated it was nearly destroyed. The perforations had thin, shreddy, or sharp-out edges, but no thickening or attempt at repair—in fact, were irregular circular holes about the size of a threepenny piece. There was no trace of lymph or peritonitis or adhesion, but in the peritoneal cavity was a good deal of putty-like, pale gluey, decolorized faecal matter which had slowly exuded through the perforations, and had evidently been in the peritoneal cavity some, if not many, days. There was most of this about the descending colon. There was no lymph exudation and no peritonitis and no effusion of blood.

CASE IV.

E. T., a servant girl, aged 18, always had good health till July 24th, 1906, when pains came on in the abdomen, much increased about half an hour after taking food; diarrhoea followed on the 26th and next day melaena; there was severe frontal headache and occasional vomiting. On the 26th she took to bed, and on July 30th came into

Chart for Case IV (E.T.).

St. Mary's Hospital, a well-nourished girl, cheerful, joking, and very anxious for solid food, though the temperature was 104° and she vomited three times on the day of admission. There was severe diarrhoea of offensive liquid dark motions containing much bile and very unlike typhoid motions; there was little abdominal distension, but some tenderness at a spot on the left side corresponding to McBurney's point on the right; the pulse was 90, of good tone. Below the inferior angle of the left scapula there was a considerable area of almost complete percussion dullness with absence of vocal fremitus, vocal resonance and breath sounds, but an exploring needle showed absence of any pleural effusion. The day after admission she passed a small blood clot; during the next few days the pyrexia was of a marked intermittent type, rising each evening to 104° or more and falling each morning to 99°, as shown in the annexed chart; the case resembled typhoid fever in some respects and tubercle of abdomen and lung in others. The character of the stools, the very early melaena and the intermittent pyrexia, however, excluded typhoid, and the stationary nature of the lung condition taken with the high fever made tubercle improbable. The blood showed marked leucocytosis at each examination—47,000 white corpuscles per c.mm.—and on August 2nd slight melaena recurred and colitis was diagnosed; the intermittent pyrexia persisted, and the patient, though not feeling ill but being in good spirits and continually asking for solid food, gradually lost flesh; diarrhoea was most intractable. On August 28th she got sudden pain in the left leg and tenderness over the middle of the tibia with puffiness of the foot. Slight diarrhoea and melaena continued, and death took place on August 28th. At the necropsy the whole length of the large intestine when slit open showed a continuous granulating surface with in several places superficial ulceration. The wall of the colon, sigmoid flexure, and rectum were greatly thinned and atrophied, but there was no perforation; there was no peritonitis and no adhesions. The left posterior tibial and popliteal arteries contained a continuous column of *antemortem* clot. There were two infarcts in the base of the left lung which had caused the area of percussion dullness and the other signs which had been observed during life in the left chest.

CASE V.

A medical man, aged 45, was attacked by symptoms of colitis: diarrhoea with mucus and blood in the motions, pyrexia and pain over the sigmoid region. He came into a nursing home in London, where he gradually improved, and in some weeks returned to his country practice very incompletely recovered. About two months later he was attacked by severe dyspnoea, shivers, and pyrexia. I saw him in the country and found extensive percussion dullness and weakened air entry at the base of the right lung; this and his repeated shivering attacks suggested empyema, and a few days later Mr. Pepper saw him with me. On the morning of our visit he had been attacked with pain in the loin and passed urine containing albumen and a considerable quantity of blood; an exploring needle negative fluid in the chest, and it appeared certain he had embolisms in the lung and kidney. After a long illness he recovered, but remained intensely anaemic and of waxy appearance during many months of convalescence at the seaside.

Two years later the diagnosis of embolism was verified, for he began to pass blood in the urine, and Mr. Pepper exposed the left kidney, and on incising it found broken-down infarcts with discoloured blood. He recovered after this, but some two years later died of nephritis.

CASE VI.

A. B., my clinical clerk, while in perfect health was attacked in June, 1892, by lumbar pains and by diarrhoea so severe that he was thought to have "English cholera"; this continued, and two or three days later he came into St. Mary's Hospital under my care; temperature was 101°; pain about the sigmoid region with diarrhoea of offensive stools containing much mucus. There was distension of the colon and it was evident that he had acute colitis; I could find no cause to account for it, but he had, shortly before the illness began, drunk freely of cold water while playing cricket on a very hot day. Sir William Broadbent attended him with me throughout a very severe illness. Blood had appeared in the motions three days after his admission to hospital and the motions were horribly offensive. There was rather acute pain over the sigmoid region and pyrexia of moderate degree, 101° to 102°. The pain over the left side of the abdomen gradually lessened, but for some weeks there succeeded attacks of acute pain over other parts of the colon, each time with other evidences of fresh colitis. He became greatly emaciated and very weak, but clear in mind except for some little nocturnal wandering. At the end of five weeks the symptoms began to subside and he eventually perfectly recovered from an illness which brought him to the point of death, and he is now, seventeen years afterwards, occupying a distinguished position in the profession. He was treated throughout his illness by small doses of calomel and opium internally and by antiseptic and astringent enemata.

CASE VII.

F. H., a youth of 17, after getting wet through on May 25th, 1906, did not feel very well, and on the 30th got acute pain in the abdomen and in the sacral region; he kept his bed, vomit-

Chart for Case VII (F. H.)

ing up everything except a little milk and tea. He was admitted into St. Mary's Hospital on June 4th. There was considerable abdominal pain and marked tenderness over the descending colon; the bowels were relaxed and the motions very offensive, containing much mucus but no blood; there was little tympanites; pulse, 98 per minute; frontal headache of some days' duration; mind quite clear; temperature 104.4°. During the next twelve days—that is, till June 16th, the temperature was 99° or 100° each morning, rising every evening to 103°, and on the 14th and 15th to 104°. He took food fairly well without vomiting; constipation alternated with diarrhoea, and disinfectant enemata were given on every second day. After the 16th the temperature gradually fell, as seen by the chart, the diarrhoea lessened, and the patient gradually got well.

CASE VIII.

E. K., a retired naval officer, was quite well till December 16th, 1893, when after dinner he felt a little sick; next morning he vomited again twice, and felt so ill he had to lie down. After this he remained in bed, with slight pyrexia, and with hiccough, which never left him night or day; he vomited all food given him, and there was retention of urine. On December 26th I saw him with Dr. Payne of Storrington; I could find nothing objective, except some distension and tenderness over the descending colon. I thought the attack was due to bacterial poisoning, as he had taken some scalloped fish just before his attack. He gradually got well, but on May 21st, 1899, he got an exactly similar attack, vomiting, and hiccough, with melaena, diarrhoea, and great pain and tenderness over the descending colon, and pyrexia; his symptoms continued till June 11th, and he gradually recovered. I think there is little doubt this was a case of colitis and not of bacterial poisoning.

CASE IX.

E. W., a girl of 13, after suffering for a fortnight with severe abdominal pains, diarrhoea and pyrexia, came into St. Mary's Hospital on October 15th, 1903. The temperature during the ten days following remained at 102° to 103°. There was slight tympanites and mucous diarrhoea. She gradually got well, and the case was regarded as one of possibly typhoid fever, though the severe abdominal pain rendered this very doubtful.

On February 8th, 1905, she came again into the hospital with similar symptoms—abdominal pains, diarrhoea, and continued pyrexia. The abdominal pain was severe, there was tenderness over the descending colon, and mucus with the dejecta. Her blood count showed marked leucocytosis, 18,950 white corpuscles per c.mm.

The symptoms persisted for some twenty-one days, with fairly clean tongue, no vomiting, but persistent pyrexia, the maximum temperature being 103° and the minimum 102° on the 9th and 10th; then 100° to 103° till the 18th, then fell to 102° on the 18th and 19th; and fell to 99° on the 19th, almost by crisis.

The clean tongue, absence of splenic enlargement or of spots, the history of the previous attack, the leucocytosis, and the acute pain, excluded typhoid fever, and I think there is little doubt that the two illnesses were attacks of colitis.

CASE X.

Nurse L. was quite well till the night of October 18th, when she vomited three times while on night duty, and had pains in the abdomen but no diarrhoea. She took to her bed on the morning of October 19th, when I saw her with Dr. Bird, the Resident Medical Superintendent of St. Mary's Hospital; she had vomited twice; there was headache and diarrhoea; temperature 101°. There was tenderness and distension over the colon, and much mucus and a very large quantity of blood in the motions, which were offensive and frequent. She was ordered calomel and opium three times a day, and sanitas enemata.

On the night of the 21st there was a good deal of abdominal pain and diarrhoea, but less blood. There was marked tenderness over the hepatic flexure. Dr. Spillbury's blood examination showed white blood corpuscles 14,000 per c.mm.; polynuclear leucocytes 68 per cent.; large mononuclear 12 per cent.; small polynuclear corpuscles 20 per cent.

She gradually improved, the temperature falling, and in about three weeks was well, though very anaemic and weak.

CASE XI.

I may add notes of a case still under my observation, in which a gentleman of 51 years of age, after suffering pains in the abdomen for over a year, suddenly developed, in March, 1906, a very acute attack of colitis with severe diarrhoea and melaena; from this he soon recovered. But in September, 1906, a similar attack occurred with pyrexia, but slight melaena, diarrhoea, and severe abdominal pain, with phantom tumour in the sigmoid region; hiccough soon succeeded and a severe rigor, threatening fatal collapse. An exploratory operation showed nothing abnormal except pallor and dryness of the colon. Terrible and exhausting hiccough for ten days threatened the patient's life, after which recovery slowly ensued; but early in January the colitis recurred in a very acute form with return of hiccough, symptoms of intestinal obstruction suddenly came on, and death was averted only by opening the caecum.

Addendum.—For the five months since this paper was read acute colitis has persisted with pyrexia, occasional rigors, and signs of pulmonary embolism; and it is only now, after seven months of acute illness, that gradually diminishing symptoms of colitis allow some slight hopes to be entertained of recovery.

CASE XII.

In another case, seen in consultation with Dr. Wiggin early this year, a lady was attacked with severe acute colitis, accompanied by melaena, which resisted all medicinal treatment and became so severe that, as a last resort, the caecum was opened; the melaena gradually ceased, and, after many weeks of colitis and intermittent pyrexia, recovery ensued, but three or four weeks later a relapse occurred with evidences of embolism in the lungs. Ultimately the patient got quite well.

REMARKS.

Records of other cases which have come under my observation might be added, but these sufficiently illustrate the main features of the disease to which I wish to draw attention; no attempt will be made here to give a complete account of the disease.

First as to the *Onset*: (1) Very acute cases may commence by severe pains in the back and limbs, like variola or influenza or lumbago; this back pain may precede other symptoms by two or three days; there may be accompanying fever and chills. (2) More commonly the first symptoms indicate some abdominal disorder—acute abdominal pain or diarrhoea or melaena—either of which may be profuse; vomiting, too, is common, and headache, and there may be pain on defaecation. (3) The onset may be gradual, with headache, diarrhoea, and pyrexia simulating typhoid fever. The distinguishing points between the two will be discussed later.

The *tongue* is usually, in the early stages, thickly coated with white fur, which soon clears off, and throughout the course even of very severe cases the tongue may remain small, moist, and clean; the dried up, brown tongue of typhoid is seldom seen, and the clean, moist tongue is quite a characteristic of the disease.

The *gums* may become sore, ulcerated, or spongy, and in bad cases acute pharyngitis may occur. In one case a membrane of *oidium albicans* formed on the palate.

There is often a horribly offensive *odour of the breath*, a heavy musk-like sickening odour, which may permeate the whole sick room, and is very different from the characteristic odour of typhoid fever.

The *appetite and gastric digestion* are preserved in a remarkable degree. The patient may enjoy and digest food well throughout a serious, even a fatal, attack of colitis. In one case the patient asked for solid food up to almost the day of her death. *Vomiting* is rarely present except at the very onset of the disease, when it may be repeated, and severe.

Chart for Case IX (E. W.)

Abdominal Pain.—At the onset there may be severe colicky, or cramp-like pains, and in all acute cases there is a feeling of soreness or "gnawing inside," and frequently in addition paroxysms of cramp-like pain in the situation of parts of the colon which seem to be in a condition of spasm, or in which active peristaltic contraction is traceable through the abdominal wall. These paroxysms may be very severe, and persist with intensity dependent on the severity of the colitis. The position of the pain in colitis may change from time to time quite suddenly, sometimes from involvement of fresh tracts of colon by inflammation, sometimes from spasm of alternating parts of the colon. When the rectum is affected there may be pain on defaecation.

Still, on the whole, colitis is not a very painful disease, and very intense, even fatal, ulcerative colitis may be unaccompanied by pain from first to last.

Tenderness is usually present over the part of the colon affected, most often the descending or sigmoid colon, and often a more widespread hyperaesthesia of the abdominal wall.

Abdominal Distension.—The distended outline of the part of the colon affected—most usually the descending colon—is often discernible; the distension is not usually extreme, nor is there usually any general tympanites, and the abdomen, so far as I have seen, remains soft and yielding, and never becomes tense and drumlike, as it may do in typhoid fever; one part of the intestine may be distended and compressible, while another part may be tense and spasmodically contracted, with severe cramp-like pain. Moreover, these conditions may suddenly change, a portion of intestine that is spasmodically contracted and tense becoming quite soft an hour later; Dr. Langenhagen¹ observes that "this spasm is not in contradistinction to atony, the two conditions succeeding one another with great facility"; the abdominal muscles may become tensely contracted over spasmodically-contracted lengths of colon, producing a prominent swelling liable to be taken for abscess or solid tumour; in fact, a "phantom tumour"; such tumours produced by spasmodic contraction of "lengths" of intestine have been described by Laënnec under other conditions, such as dynamic pulsation of the abdominal aorta.

Diarrhoea is usually an early symptom, the motions being frequent, liquid, containing mucus, and maybe blood, and being horribly offensive; they are usually dark in colour and contain much bile, very unlike typhoid motions; the mucus is passed in shreds or membranes, or filaments or pellets, and is often blood-stained; melaena may appear at the very commencement of the disease, and certainly may do so before any ulceration has occurred; it may be very profuse and bright red in colour; more often it is passed as red-stained fluid with the motion. The melaena may intermit and recur with other evidences of recurrent colitis. Considerable-sized thick sloughs may be passed with the stools. Although there is in colitis usually passage of loose frequent stools, there is a tendency to retention of faecal matter in the intestine, probably from atony of the bowels, especially in the more chronic cases.

The circulatory and respiratory organs are singularly little affected in the course of colitis; the pulse does not greatly quicken and it keeps its tone and strength, except of course when a fatal end is approaching; there appears much less tendency to congestion of lungs, etc., in colitis than in other long-continued febrile maladies. There is, however, a great tendency to severe anaemia in protracted cases, partly no doubt from melaena. The anaemia after recovery may be most intense and long lasting. And leucocytosis was observed so frequently in my cases that I

cannot but regard it as a feature of the disease, and one useful in the diagnosis.

Nervous System.—There is not the tendency to gradual prostration with dulling of the mental faculties, hebetude, or stupor, as seen in typhoid; on the contrary, the alertness and clearness of mind, and the freedom from delirium are most marked, and the patient's mental condition is no criterion of the seriousness of his illness—he may be cheerful and hopeful in the most serious and fatal cases; one patient joked and made inquiries as to how long it would be before she was well almost to the day of death. Sleeplessness is often troublesome, and headache may be present at the onset of the disease.

Hiccough in the course of acute intra-abdominal disease is usually significant of gangrene, and may be so in ulcerative colitis. But in this disease it frequently occurs without gangrene, as shown by the ultimate recovery of the patient, or, if he die, by *post-mortem* examination. Such gangrene is probably a reflex symptom or a result of toxæmia. But, whatever its cause, hiccough occurs in the course of ulcerative colitis so frequently that it is almost to be regarded as a symptom of the disease. And its severity, frequency, and persistency for many days or weeks constitute it in itself a very dangerous symptom, tending to exhaust the patient by want of food and sleep to the point of death, or to death itself.

Urinary Symptoms.—There is occasionally some strangury or difficulty in micturition, but the urine is usually of good quantity and normal in composition, though often containing bile pigment.

General Symptoms.—The temperature in acute colitis is usually raised, though not to a very high range—101° or 102° as a rule; it may rise higher in some cases, not necessarily the most severe. As a rule, the pyrexia is of the continued type, with but slight variations, but it may take on a remittent or intermittent type, as exemplified by Case v, where the temperature, normal each morning, rose every evening to 104°; many other cases have shown that the temperature in colitis may be of this intermittent character, which is worthy of note, as in Murchison's well-known enumeration of the causes of intermittent pyrexia ulcerative colitis is not included.

One remarkable point was noticeable in two of my cases of colitis that, while the skin was cool to the feel and the temperature normal or but slightly raised in the axilla, it was raised 1½° or 2° higher in the mouth; this difference in axillary and mouth temperature may be marked all through the course of colitis.

Rigors may occur in colitis, sometimes severe, and without any subsequent indications of local complication; they appear due to toxic absorption. Similar rigors occur during the course of typhoid fever.

Emaciation and Debility are less rapid than might be expected in so grave a pyrexial disease; probably this is owing to food being well taken, and to the digestive functions of the stomach and small intestine being unimpaired; the skin may be dry, or there may be profuse sweats; in long lasting cases there is a tendency to bedsores.

Complications and Sequelae.

Perforation may occur, though, considering the severity of the ulceration, it is remarkably infrequent, partly, no doubt, because the wall of the colon is much thicker than that of the small intestine; there may be multiple perforations, and even considerable sloughing of the wall of the colon. Evidently, if multiple perforations are found *post mortem*, perforation is not rapidly fatal, and in Case III there was a large accumulation of dried, putty-like faecal matter in the abdominal cavity, which must

have gradually found its way through perforations formed long before death occurred.

It is noteworthy that peritonitis does not necessarily result from perforation from colitis, and that *perforation may occur without any symptom whatever to denote it*, and may be survived a considerable time. Other writers have noted this; thus, in Lazarus Barlow's case,² there were many perforations, but "nothing to lead to a suspicion of the existence of the lesions during life," and in Sharkey's case³ "there had been no indications during life of perforations" which were found *post mortem* in the caecum and sigmoid colon. Possibly the bell sound heard widely over the abdomen might indicate it.

Embolism.—In E. T.'s case there were signs of consolidation in the base of the right lung, the nature of which was difficult of diagnosis; the autopsy showed it to be due to embolic infarctions in the lung, and in this case there was also embolic blocking of the anterior tibial artery; in Case v there were symptoms during life of embolism in the lung, liver, and kidney, and two years after the infarcts in the latter were shown by operation; in two other cases there have been evidence of sudden embolisms in the lungs during convalescence followed by physical signs of local pneumonia, pleurisy, or collapse of lung. Hale White, too, mentions a case fatal with all the signs of pulmonary embolism.

There appears to be, therefore, in colitis a marked tendency to embolism in distant organs, usually after the acute stage of colitis has subsided.

Relapses.—The whole course of the disease tends to consist in a series of repeated attacks of inflammation of the colon. But apart from this there is a tendency to relapse after apparent convalescence and even recovery; one patient after a month's continuous improvement, and when he seemed practically well, got a second attack worse than the first. And I have several times met with patients in whom acute colitis recurred after much longer intervals.

Anaemia of very intense and lasting nature is apt to follow on colitis; the leucocytosis of colitis has been already alluded to.

Local After-Effects of Colitis.—In the condition in which large ulcers small in number occur in the colon contraction may occur on healing with serious after-results, in one case a subdiaphragmatic abscess. But when recovery ensues after acute ulcerative colitis there need be no necessary contraction or after-symptoms; in one case the patient has been quite free from any intestinal trouble for seventeen years since the attack of very severe ulcerative colitis.

Causation.

No doubt there are many varieties of colitis, and the cause may not be the same in all cases. In some cases it has been clearly due to drinking of impure water, and in some cases has been set up by poisonous food. Possibly preserved and tinned foods may set it up, or even the substances used as preservatives to foods. Sir Almroth Wright was good enough to investigate the blood and faeces in one case of mine, but *B. coli* and streptococcus were alone detectable, and the specific organism of colitis, if such there be, is as yet undiscovered.

Morbid Anatomy.

Innumerable varieties of acute colitis have been described, but this subdivision affords little assistance in the study of the disease. The condition in which one or two or three large ulcers may be found in the colon, comparable to the perforating ulcers of stomach or duodenum, is a condition quite distinct from acute ulcerative colitis of which this paper treats.

In acute colitis the mucous membrane becomes intensely hyperaemic and gorged with blood; no doubt the congestion affects also the deeper coats of the intestine, and its whole wall may be much thickened, with turgidity of the mucous membrane and ready effusion of blood without any ulceration. The turgidity of the mucous membrane subsides considerably after death, but is sometimes observable during life in cases in which the colon has been opened by operation. It can also be seen through the sigmoidoscope (which must be used sparingly in colitis); the condition seems to be what in old works is called "phlegmonous inflammation." Usually only part of the colon is attacked, most commonly the descending or sigmoid colon, but any part, and sometimes the whole length of the colon, may be attacked at once, or may get gradually

involved by continuous extension, or by successive attacks of independent parts. This is shown *post mortem* sometimes, tracts of ulceration being seen in the colon with intervening lengths of healthy mucous membrane.

Ulceration is not a necessary part of acute colitis: it may be very intense, even fatal, without any ulceration; in other cases the ulcers may be very minute, as in Wilks and Moxon's case, in which "the whole internal surface of the colon presented a bright vascular soft red surface, with but a few minute points of ulceration." And as a rule the individual ulcers are not of large size, mostly only about the size of a sixpenny-piece, but they may be innumerable and coalescent, so that little of the mucous membrane remains except shreds or bridges between ulcerated parts of the bowel. The ulcers may be deep, destroying the coats of the intestine except the peritoneum and the colon, or lengths of it may be so thinned that they can be seen through, and the colon cannot be removed from the body without tearing it. Sometimes there is considerable thickening of the colonic wall. The ulcers have sharp-cut irregular edges, and tend to the circular form. The edges may be shreddy, and show often no attempt at repair.

There seems no definite dividing line between acute colitis with and without ulceration, and there is certainly a stage of acute colitis before ulceration occurs.

The colon is often quite fragile and rotten; the condition of the wall has been likened to that of sodden paper; in Case III the noticeable feature was the dryness of the colon; it was desiccated and fragile rather than sodden; in another case in which operation exposed the colon during life, its dry, pale condition afforded a great contrast to that of the glistening, moist small intestines.

Perforation is not common, but may produce irregular openings with ragged, shreddy edges, no thickening or adhesions and no signs of repair—indeed, mere holes in the gut; they may be multiple—in Case III there were six—and this fact and the presence of dried-up, putty-like faecal matter extruded from them into the abdominal cavity shows that perforation may be long survived. But the colon wall may be much more extensively destroyed than by such perforations; there may be *wide-spread sloughing of the colonic wall*, and there is no limit to the ravages of the disease; in Ormerod's case⁴ "the front wall of the transverse colon was so completely destroyed that the inner surface lay open and exposed; the caecum was perforated in several places; the only parts of the bowel that were not laid widely open were the ascending colon, splenic flexure, lower part of the sigmoid flexure, and the rectum."

Peritonitis or local abscess may occur, but in most *post-mortem* examinations there is a singular absence of signs of peritoneal inflammation.

The process of repair cannot be looked for in *post-mortem* evidence, but patients who recover after even very severe attacks, with all the evidences of deep ulceration, may be afterwards free from symptoms, from which it may be inferred cicatrization may occur without much contraction of intestine.

Course and Duration of the Disease.

No doubt many varieties of acute colitis exist, but it is not possible to give any clinical classification of them at present.

Some cases appear to run a definite course of about three weeks, after which the symptoms subside and recovery ensues, though relapse may occur. Other cases run a very rapid course, being soon fatal from exhaustion. And many run on indefinitely, with evidences of recurring attacks of inflammation of the colon, and end fatally after many months from asthenia or complications. In a case of Dr. Gee's, death occurred after eighteen months' illness.

Prognosis.

It is a grave and dangerous disease. Data as to its fatality are wanting, and in an individual case the prognosis must be based on the severity of the symptoms. Very severe cases may be recovered from.

Diagnosis.

The diagnosis of acute colitis usually presents little difficulty if it be borne in mind that such a disease as colitis exists.

When commencing with severe lumbar pain it may simulate influenza, variola, Bright's disease, or lumbago; when it begins with abdominal pain it has been taken for lead, biliary, or renal colic. When it comes on with diarrhoea it has been diagnosed as English cholera, and is

often set down as gastro-enteritis or English dysentery, sometimes as typhoid or paratyphoid; in all these cases the characteristic signs of colitis soon develop.

When it commences with profuse melaena there is usually a *s* mucus in the stools, pointing to acute colitis.

Greater difficulty in diagnosis arises when the disease comes on gradually with headache, diarrhoea, and pyrexia simulating typhoid fever; but in colitis the headache is less severe and persistent than in typhoid; the pyrexia does not increase in the same step-like manner, the tongue does not become dry, the abdominal detension is limited to the colonic area, the stools are different in character, the spleen is not enlarged, there is no cutaneous eruptions, and the breath has a characteristic odour. Melaena may occur in both diseases, but in colitis melaena may occur much earlier than the tenth day, but not in typhoid. Vidal's test may also assist the diagnosis.

Of all the distinguishing points, there is none, I think, so important as that in colitis there is often acute abdominal pain, which is unknown in typhoid, and there is leucocytosis.

Tuberculous abdominal disease, like colitis, leads to pyrexia, diarrhoea, and melaena, and if there chance to be the physical signs of consolidation of lung, as arose from an infarction in Case iv, the resemblance may be increased. But tuberculosis so acute as to simulate acute colitis will not fail to yield some more distinctive evidences of itself than those common to it and acute colitis.

Typhlitis and Appendicitis.—Typhlitis is colitis limited to the caecum, and it and appendicitis are distinguishable from acute colitis by the persistent limitation of the local manifestations to the right iliac region. Appendicitis is sometimes met with in association with colitis.

Treatment.

Treatment should be directed to lessening the amount of toxic substances in the colon, which may be readily absorbed by its ulcerated mucous membrane. Innumerable substances have been employed as intestinal antiseptics; probably but a small proportion of any of them, when given by the mouth, can exercise any *direct* effect so far down the intestine as the colon, but they may, nevertheless, be beneficial by their antiseptic effect on the contents of the small intestine before they pass into the colon. Of all the antiseptics none is so useful in acute colitis as mercury in some form—as blue pill, or grey powder, or calomel, combined, if it produce diarrhoea, with opium. Where the colon has been opened by operation the antiseptic powers of the mercury are very readily observable. It is surprising for how long—weeks or months—mercury may be taken in small doses in colitis without producing “mercurialism.” Possibly mercury has a beneficial effect in colitis in other ways than by its antiseptic power, and certainly appears to control the disease to some extent. Intestinal antiseptics may be assisted by antiseptic enemata, peroxide of hydrogen, or chlorinated liquors, or sanitas, added to the enema. But on no account must an enema be introduced with any force, or if it produce much pain. Weak solutions of nitrate of silver are useful in some cases.

Diarrhoea may be checked by enemata with or without opium, or by bismuth salicylate, etc., given by mouth. As a rule, even though several liquid motions are passed daily, there is a tendency to retention of faeces in the bowel, probably owing to an atonic condition of the colon; and this requires occasional enemata. Purgatives, even simple ones such as castor oil or salines, may greatly aggravate the symptoms. But, after the acute stage, gentle laxatives, such as part of a sulphur lozenge, which is also a good antiseptic, are allowable.

Melaena may become so severe as to be a grave danger; enemata of adrenalin control this best, but in one case an opening of the caecum was made, and stopped the melaena after all other measures failed.

Hiccough.—Of the numerous drugs and modes of treatment tried, opium is the only one which has any effect at all, enabling the patient to get some sleep, but even it has little effect on the hiccough.

The Nutrition of the patient must be carefully attended to, as the illness tends to be long and exhausting. Liquid foods seem best, and a mixed diet is advisable. The tendency to anaemia must be anticipated, and a little fruit or lime juice is useful. Alcohol, ether, gumbul, and

other diffusible stimulants may be required. In one case in which the inflammation was intense and phlegmonous I gave large doses of perchloride of iron with seeming benefit.

Future investigations may provide effective treatment by “vaccines,” but cases of colitis recorded in which this has been tried are not yet sufficiently numerous to give definite conclusions.

Operative Measures.

Colotomy or caecostomy have been performed, sometimes for the purpose of promoting recovery by diverting the passage of faeces from the part affected, sometimes in order to lessen dangerous haemorrhage, and in one case under my own observation it had to be done to avert the dangers of acute obstruction. Colotomy has been thought to cut short the disease, and very possibly it may do so sometimes, but this cannot be assured, and in one case acute colitis continued for months after performance of caecostomy; in another a bad relapse occurred while there was still an opening in the caecum. When ordinary measures fail in arresting melaena, opening the colon may be efficacious. In one case the patient's life was in extreme danger from haemorrhage, which greatly lessened after opening the caecum.

REFERENCES.

- ¹ *Muco-membranous Enterocolitis*, p. 31. ² *Path. Soc. Trans.*, 1892, p. 72.
³ *Ibid.*, vol. xlvii, p. 109. ⁴ *Ibid.*, 1889, p. 109.

CASE OF TUBERCULOUS MENINGITIS IN BOY TREATED WITH TUBERCULIN: RECOVERY: RECURRENCE AND DEATH.

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I MERELY record the following case as I am not aware that any similar case has been published, and in such a hopeless disease as this one is glad of any line of treatment offering any improvement in the prognosis.

I have put down the notes of this case as they were entered in my notebook during my visits. They are not very complete, as I did not feel very confident that any benefit would result from tuberculin treatment.

J. M., aged 8½ years. No tuberculosis in family. Suffered at Christmas from sore throat, which was probably diphtherial, as a younger sister was at the same time suffering from diphtheria. The throat symptoms cleared up after an injection of 2,000 units of antitoxin. A week after the injection he began to complain, and was feeling so ill that during the first week of the present year he voluntarily took to bed, complaining of feeling tired. The pulse was then about 120, and occasionally missed a beat, and I attributed his feeble condition to his late throat trouble. No cardiac lesion could be made out and there was no temperature. He did not at first complain of headache, and even when he first vomited I thought he had just upset his stomach as the Christmas and New Year festivities were delayed by the diphtheria in the family. Head symptoms, however, gradually developed; persistent vomiting; fever up to 101° at night, headache, fast pulse, and aversion to light; the abdomen was retracted. He gradually got worse, though the temperature and pulse seemed to improve, and by January 20th the headaches were confined to the frontal and temporal regions—mostly frontal. There was occasionally paresis of left side, including face and leg, but more especially of arm, which at times became completely paralysed. The pulse-rate was now 48, weak and irregular; temperature in the mouth 97.9°. The headache was paroxysmal in character—bad yesterday, January 19th, less severe to-day. Appetite good.

January 21st. Left side still paretic, but arm now completely paralysed; sensation in arm apparently unaffected, but tested only by touch. Later in the day I saw him, in consultation with a colleague specially skilled in children's diseases, and he agreed that the disease was probably tuberculous meningitis, and gave the parents a bad prognosis. With his concurrence, I resolved to try tuberculin, and gave 100 mg. of Messrs. Meisters and Lucius's T.R. The pulse was now 60, irregular; and temperature in the mouth 96°.

January 22nd. Very bad headache during the night. Pulse 58, temperature 97°. Leg still moved voluntarily; complained of pain in left side over the lower ribs.

January 23rd. Slightly less headache; pulse 68, regular. Gave for headache a powder containing sod. salicyl. gr. ii, phenacetin and caffeine aa gr. j.

January 24th. Had taken two of the powders, and headache was a little better. Had slept for three hours.

January 28th. Has improved during the last four days.