a good recovery. Her convalescence, though protracted, was very satisfactory, despite a small faecal fistula which persisted at the site of the pelvic drainage tube for about a month before closing spontaneously.

CASE II (S. C. C.)

Perforation of typhoid ulcers complicating pregnancy, with operation forty hours after onset: recovery.

L. H. A., aged 28, wife of a Chinese military officer, was admitted to hospital on November 18th, 1930, with the following history. She had been ill for twelve days, suffering from fever, headache, and abdominal discomfort. Following the ingestion of a herbal remedy recommended by a native "medicine man" she was seized with acute abdominal pain on the evening of November 16th. She vomited several times at the onset of the attack, and became very collapsed. The native practitioner treated her with morphine injections, but as her condition was rapidly becoming worse her relatives brought her to hospital at 6 p.m. on November 18th, forty hours after the onset of the acute pain, and, presumably, of the occurrence of perforation. The case was complicated by the fact that the patient was five months pregnant, but as it was obvious that peritonitis was present and that the patient was *in extremis*, I performed immediate laparotomy. On opening the peritoneum a quantity of gas escaped, with a lot of thin seropurulent fluid. The pregnant uterus filled the entire pelvis, and the inflamed and congested intestine, with considerable lymphatic exudation on the surface, appeared to occupy the entire abdomen. Two perforations were found on the free border of a coil of the ileum, about 12 inches from the caecum. They were about 2 inches apart, and were each closed with some difficulty, owing to the extreme friability of the bowel, by purse-string catgut sutures. Thorough irrigation of the entire peritoneal cavity was then carried out, using several pints of warm normal saline, and the abdomen was closed, three large tubes being left *in situ*, draining both flanks and the mid-line. The patient's condition was exceedingly grave for two days following operation, severe abdominal distension being present, but turpentine enemata caused the bowels to act on the second day, with considerable relief of symptoms. Matters were further complicated by the occurrence of a miscarriage on the third day following operation, a five-month foetus being expelled. The patient's condition however, did not seem to be greatly impaired by this mishap. On the eighth day following operation a severe attack of broncho-pneumonia developed, and for several days it was thought that this would prove a fatal complication. After several weeks' illness, however, the patient slowly recovered, and was discharged in good health from hospital eleven weeks after admission.

Consideration of these two histories makes it clear that it is never too late to operate in a case of perforated typhoid ulcer, as even the apparently hopeless patients (vide Case II) may possess amazing recuperative powers. An interesting and common feature to both cases was the thin, watery character of the peritoneal exudate, with flakes of yellowish-white lymph—not nearly so offensive as the exudate in many types of appendicular peritonitis. Unfortunately in Case I no bacteriological examination was made, but in Case II smear and culture showed staphylococci and Gram-negative bacilli.

Harte and Ashhurst,¹ in considering the bacteriology, have pointed out that the prognosis is graver if the streptococcus is the predominating organism, as, in cases in which the typhoid bacillus alone is the infecting cause, the peritonitis is of a relatively low-grade type, with a watery exudation and few adhesions. It is probable, therefore, that the successful results in these two cases were largely influenced by this factor.

CASE III (J. B. G. M.)

A Chinese casual labourer, aged 32, suffered since boyhood from a right inguinal hernia, which had latterly increased considerably in size, but, as he somewhat graphically put it, he could push his bowels back into his stomach when he lay down. On October 2nd, 1930, he was admitted to the Tongshan

Base Hospital as a case of strangulated inguinal hernia. The history was that for the preceding fortnight he had been feeling unwell, suffering from headache, some diarrhoea, fever, and swelling of his stomach. Several days before admission he had experienced a good deal of pain in his hernial swelling, and found that it could not be reduced. He used considerable force in attempting reduction on the day of admission, and the pain became exceedingly severe. The inguinal swelling was very tense, with the skin over it very red, and the patient's condition simulated intestinal obstruction due to strangulation of the inguinal hernia. Immediate operation was advised and agreed to. On incising the sac much purulent fluid escaped, and a coil of ileum, about 18 inches in length, was seen to be incarcerated. It was very swollen and congested, but obstructed rather than strangulated. A small round perforation was present on the free border of the coil, through which the extravasation of intestinal contents had occurred, and several ulcers could be palpated along the free border of the bowel. The perforation was closed by an invaginated purse-string suture, the intestinal loop carefully washed with warm saline solution, and the loop of bowel returned to just within the abdominal cavity. A large drainage tube was inserted through the neck of the sac down to the sutured perforation, and the patient returned to bed. A subsequent blood examination gave a strongly positive Widal reaction, and the later course of the case was that of typical typhoid fever. A radical cure was performed for the hernia two months later.

In this case the hernia was undoubtedly rendered irreducible by the inflammatory swelling and infiltration of the Peyer's patches in the herniated bowel, as a consequence of typhoid. It was indeed a lucky coincidence that the perforation occurred in the hernial sac rather than in the general peritoneal cavity, although it is quite probable that perforation was induced by the patient's own attempts at reduction of the inflamed bowel.

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TUBERCULOUS PERITONITIS SIMULATING COELIAC DISEASE

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In view of the interest aroused in coeliac disease by the excellent review of Leonard Parsons,¹ and by the theory put forward by Ryle² that the essential cause is an obstruction of the lacteals by previous inflammation, it seems opportune to record a case recently under my care in the Miller General Hospital, which closely mimicked coeliac disease, and in which extensive tuberculous peritonitis was found at necropsy.

CASE HISTORY

Mary O. was first referred to me by Dr. Rogerson of Bexley Heath, on account of diarrhoea, abdominal enlargement, and wasting. In this family the seven other children were healthy. At birth she was a full-term baby weighing 6 lb., and she was breast-fed. She remained well till two months before I first saw her, when, at the age of 2 years and 4 months, she began to get periodic attacks of diarrhoea with occasional pale stools, associated with loss of weight, feverish turns, whining, abdominal enlargement, and occasionally prolapse of the rectum. At no time was there any

abdominal pain or vomiting. On examination she was evidently small for her age. Her weight at the age of 2½ years was 17½ lb., and her height 30 inches (average normal 33 inches). She was very wasted, the limbs being much more so than the abdomen and face. There was but little subcutaneous fat anywhere; there was marked wasting of the

gluteal region, so that the prominence of the buttocks had almost disappeared and the skin hung in folds. There was definite loss of tone in the muscles, producing a dorsal kyphosis. In contrast to this wasting the abdomen was enlarged (girth at umbilicus 18 inches). There were no signs of free fluid, but gaseous distension was so marked that coils of gut could occasionally be seen through the abdominal wall. Palpation did not give the characteristic doughy feel of tuberculous peritonitis, and careful examination did not reveal any enlarged or palpable abdominal glands. Rectal examination was also negative. The sclerotics were of rather light blue colour, with the particularly brilliant reflection from them so often associated with hepatic disorders. The bony skeleton showed some evidence of rickets, as the radial epiphyses were enlarged, the tibiae rather bowed, the head bossed, and there was beading of

the ribs. She was also evidently anaemic.

There were anything up to five stools a day, the average being two to three. They were large, bulky, very offensive and pale, with evident excess of fat and some excess of mucus. This was confirmed by microscopical examination, which showed a large excess of split fat to be present. The stools were alkaline (pH 8.0). Cultivations showed the presence of *B. coli* and *Streptococcus faecalis* only. X-ray examination of the abdomen failed to reveal any opacities suggestive of tuberculous peritonitis. A blood count showed haemoglobin 50 per cent.; red cells, 4,700,000 per c.mm.; colour index 0.5; white cells, 10,800 per c.mm. During the whole of her time in hospital she ran an irregular pyrexia; some days the temperature was normal, while at other times the rectal temperature would rise to 104°. For periods of two to three days, with the larger rises of temperature, she was fretful, the abdominal distension became much more marked, and the stools more offensive, copious, and still more fatty. In between these bouts the condition would settle down to the quiescent phase. She also exhibited the mental characteristics of cases of coeliac disease. During her brighter periods she looked precocious in proportion to her size, and took an intelligent interest in what was being said about her, although saying very little herself. Her appetite was capricious.

She was given a considerably reduced fat diet in accordance with the usual plan in these cases; the protein was increased, and irradiated ergosterol and ultra-violet light were administered. To control the diarrhoea, silver nitrate and, later, kaolin were given. Under this regime the abdomen became smaller, the stools became much less offensive and firmer, and the fat largely disappeared. In spite of this her general condition did not improve. She slowly lost weight, became very anaemic, and continued to get periods of pyrexia. Later, streaks of blood and some pus appeared in the stools, in which starch granules and excess of meat fibres were present. Treatment with pancreatic ferments and bile salts combined with a blood transfusion were unavailing. Towards the end she became drowsy, and marked oedema of her face and legs occurred. When she died at the age of 2 years and 10 months her weight had fallen to 12 lb. 12 oz.

POST-MORTEM FINDINGS

Dr. W. Smith, pathologist to the Miller General Hospital, made the following observations. There was much wasting of the arms and gluteal regions of an emaciated child. Recent adhesions surrounded the whole of the right lung, in which small areas of consolidation were present in all lobes, more marked in the upper lobe; the left lung appeared normal. There were no obviously enlarged glands in the mediastinum;

the heart muscle was good, with slight right-sided dilatation; the liver showed some degree of venous engorgement. The intestines were matted together and adherent to a large mass of mesenteric glands involving the whole of the mesentery; caseation was present in many. Section of the glands revealed the typical appearances of tuberculous disease, with areas of caseation and giant cell systems. Similar changes, and also large areas of lymphocytic infiltration, were found on histological examination of the lungs.

COMMENTARY

This case showed all the classical symptoms and signs of coeliac disease. The patient was a girl (Still states that coeliac disease is nearly twice as common in girls as in boys), who developed the disorder at the age of 2 years and 4 months (Still gives the limit of age of onset in his series as 8 to 38 months). There was marked hypotonia and wasting, especially of the glutei—described as typical of coeliac disease by Reginald Miller. The stools were large, bulky, pale, mucoid, and offensive, and consisted largely of split fat in the form of soaps and fatty acids. The abdomen was much distended with gas. The mental state was typical of the coeliac child, and there was a tendency to rickety changes in the bones, a point stressed by Leonard Parsons. A variation in the patient's condition every few days is often found in coeliac disease, as are also the secondary anaemia and the rather low white cell count; and some oedema of the face and extremities is not unusual. An irregular pyrexia is often present in the diarrhoeic form of coeliac disease (R. Miller). The picture seemed complete, as there was no radiological evidence of diseased abdominal glands, yet at necropsy an extensive tuberculous peritonitis was found, without any calcification in the glands. The only points that were in favour of a diagnosis of tuberculous peritonitis in this case were the height of the pyrexia and the rather rapidly fatal termination of the disease.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

PERFORATION OF GASTRIC ULCER THREE AND A HALF YEARS AFTER GASTRO-ENTEROSTOMY FOR PERFORATING DUODENAL ULCER

This case seems to me to be of sufficient interest to put on record as an unusual sequel of gastro-enterostomy.

The patient, a male, first came under my care on July 28th, 1927, at which date he was 44 years of age. He had a small perforation of the anterior wall of the duodenum, and gave a six months' history of pain after meals. I sutured the perforation, and performed posterior gastro-enterostomy. Recovery was uneventful.

In reply to a follow-up inquiry in September, 1930, he wrote to the effect that he had been under his doctor recently with gastritis, but that the condition had cleared up.

On January 28th, 1931, he was again admitted to hospital with symptoms of perforation. Laparotomy showed the duodenum apparently normal, the gastro-enterostomy in perfect order, and an indurated ulcer of the lesser curvature of the stomach, proximal to the stoma, which had perforated. I repaired the perforation, removed the appendix, and drained the pelvis. Recovery was again rapid.

X-ray examination of the stomach about three weeks after the second operation showed that food passed very rapidly through the gastro-enterostomy opening.

The patient was a fairly healthy-looking man of spare build. Most of his teeth had been extracted. He was said to have a partiality for alcohol. He told me that he was