SOME UNUSUAL BUT INSTRUCTIVE SURGICAL EMERGENCIES

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If the cause of acute symptoms is clear, proper treatment is begun without delay, including, if necessary, a well-placed incision. Only too often, however, the diagnosis is in doubt even in common conditions; and in uncommon ones the delays and difficulties are obviously increased.

The delays which occur are shown by the following eight cases, only two of which were admitted to hospital within twelve hours of the onset of the symptoms, two being delayed for two days, two for three days, one for seven days, and one for ten days. The difficulties in diagnosis are seen in the case reports which follow, and in the literature: for instance, in strangulation of the stomach in a diaphragmatic hernia operation is commonly delayed until gangrene has occurred (Warren, 1919), in biliary peritonitis a wrong diagnosis is usual (Fifield, 1926), and in strangulation of gut through a hole in the greater omentum a correct diagnosis has not been reported.

The following histories, with a brief discussion of the literature, are therefore presented in the hope that they may help in the diagnosis and treatment of future cases.

Strangulated Diaphragmatic Hernia

Case 1

A boy aged 16 was admitted on December 16, 1949, complaining of central abdominal pain, vomiting, and constipation for 10 days. His vomiting, which had occurred several times each day, consisted only of small amounts of greenish fluid; but his abdomen had become increasingly distended and he had passed neither faeces nor flatus for five days.

In June, 1949, he had had a similar attack lasting for three days, accompanied by pain in the left shoulder, but this attack had been mild and its history was not elicited until later. At the age of 16 months he had had a thoracotomy at the Radcliffe Infirmary for a left-sided empyema, and it was noted at the time that the tube was low and did not drain well. He had otherwise been in good health with normal appetite and bowel actions.

On examination (T. 98° F.—36.7° C.; P. 100; R. 20) his tongue was moist, his skin elastic, and he did not look ill, but his abdomen was very distended, with "ladder patterns" of bowel and visible peristalsis. There was a scar 2½ in. (6.25 cm.) long in the left ninth intercostal space in the posterior axillary line. No other abnormality was found in the chest or elsewhere.

A plain radiograph of the abdomen and chest showed gross small-bowel dilatation and fluid levels, the stomach below the diaphragm in its normal position, and a small loop of bowel with a fluid level within the chest, in a postero-lateral position.

Gastric suction and intravenous fluids were begun and the abdomen was opened through an upper left paramedian incision. It was found that the splenic flexure of the colon had entered a diaphragmatic hernia to the left of the oesophagus and was tightly held, but the exposure was inadequate because of the distended gut. The incision was therefore continued across the rectus and into the tenth intercostal space, and the diaphragm was divided as far as the hernia, the procedure being extrapleural. It was then seen that omentum as well as gut had herniated and that the omentum was adherent to the sac, which was not lined with peritoneum. The omentum was divided, the gut was reduced and found to be viable, and the diaphragm and wound were repaired with continuous sutures of braided nylon.

The boy's condition caused some anxiety for 48 hours, but then became satisfactory and he went home on the eighteenth post-operative day. A barium meal and enema two months later showed no recurrence of the hernia.

Case 2

A woman aged 36, five months pregnant with her first child, was admitted as an emergency case on February 1, 1950. She had complained for one week of pain in the left hypochondrium through to the back, made worse by deep breathing, together with regurgitation of her food and slight shortness of breath on exertion. There were no other symptoms, but she had suffered from occasional attacks of epigastric discomfort and flatulence after meals for some years.

On examination (T. 98° F.—36.7° C.; P. 102; R. 24) she looked well, and was not short of breath or cyanosed. Her trachea was to the right of the midline, the apex beat to the right of the sternum, and the left side of the chest moved less than the right. There was dullness at the left base and breath sounds were absent, but neither bowel sounds nor splashing were heard in the chest, and the abdomen showed no abnormality.

Radiographs of the chest showed the heart displaced to the right and a large translucent area in the left side of the chest which, at first sight, appeared to be a cyst of the lung; a barium meal, however, showed it to be stomach within the chest, but with the cardio-oesophageal junction and the pylorus in their normal positions. It is of interest to note that when the patient was lying flat and on her right side some air and barium passed on into the duodenum.

On February 6 there was some lower abdominal pain and pain in the left shoulder; regurgitation of food continued. Next day the patient was retaining no food, so that laparotomy was decided upon.

Upon opening the abdomen a localized collection of pus and barium was found around a perforation of the cardio-oesophageal junction at the point where it was compressed by the hernial orifice in the diaphragm. The perforation was repaired, a tube was put down to the area, and a jejunostomy was done, but her condition was unsatisfactory after the operation. She miscarried on February 10 and died on the 13th.

Post-mortem examination revealed a diaphragmatic hernia, about 3 in. (7.5 cm.) in diameter, to the left of the oesophagus, and through it had passed almost the whole stomach, the splenic flexure of the colon, and the spleen. The stomach and colon were tightly compressed by the margins of the hole in the diaphragm, and there was an area of necrosis of the stomach at the site of the perforation (presumably from pressure of the hernial margin) and some blackening of the colon where it passed through the ring. There was no peritoneal lining to the hernial sac, which occupied two-thirds of the left side of the chest.

Comment on Cases 1 and 2

In the diagnosis of Case 1 it was obvious that there was a low intestinal obstruction, but its origin was not at first clear, although the thoracotomy wound had been observed. That a diaphragmatic hernia might follow thoracotomy in infancy was not considered until the x-ray films had been seen; and the case illustrates not only the possibility of such a hernia but the value of a plain radiograph of the abdomen, including the lower chest, in all cases of intestinal obstruction.

The actual cause of the hernia must remain doubtful. Bryan (1921) and Harrington (1948) state that a diaphragmatic hernia may follow an empyema or the use of a drainage tube near the diaphragm, but quote no cases; nor has it been possible to find cases reported. It seems more probable that the diaphragm was injured at the time of operation, because it was noted that the tube was very low in the chest and never drained properly; and it is well known that injuries to the diaphragm heal badly. Indeed,

Guthrie (1855) stated that they never heal, although this has been shown to be incorrect (Greig, 1919; Meyer, 1919).

Case 2 illustrates one way in which a diaphragmatic hernia may present—that is, with repeated vomiting without other symptoms and with signs in the chest which are not obviously due to the presence of gut (Gaudier and Labré, 1918) and in which the x-ray findings at first sight suggest a pulmonary lesion or a raised diaphragm (Carter and Giuseffi, 1948).

I have been unable to find reports of any cases of intra-peritoneal perforation of the stomach at the diaphragmatic ring, presumably from pressure necrosis, but Gaudier and Labré record a case which ended fatally from repeated haematemeses and was found at necropsy to have bled from an ulcer. The ulcer was on the lesser curvature of the stomach, most of which had herniated through the diaphragm into the chest, and was at the site of the firm diaphragmatic ring and away from the usual ulcer sites; the authors considered it to be due to the pressure of the ring.

Those diaphragmatic herniae which come to the surgeon as emergencies (Astley Cooper, 1804; Guthrie, 1855; Gaudier and Labré, 1918; Warren, 1919; Bryan, 1921; Harrington, 1948; Carter and Giuseffi, 1948; Borrie and Foreman, 1948) are usually the result of penetrating wounds, but congenital herniae and those resulting from crushing injuries also strangulate. The injury is nearly always on the left side, and most of the traumatic herniae which strangulate do so within three years of wounding, but may do so many years later. Even those on the right may obstruct (Astley Cooper, 1804).

Most of these patients have had no warning of impending trouble and are in good health at the time of strangulation; but about one-third have had premonitory symptoms, and many of these are in poor condition from voluntary starvation because of pain with their meals and vomiting. These warning symptoms can be divided into four groups: (a) Abdominal, of which the chief are "indigestion," pain in the epigastrium and other regions, vomiting, and constipation; (b) respiratory, such as dyspnoea on exertion or after meals; (c) cardiac, such as palpitations and precordial pain; (d) recurrent fever. Many of these patients have been treated as malingerers or have had a previous operation without any relief of their symptoms.

The actual obstructive attack is usually brought on by straining, coughing, or a meal. It is heralded by colic or upper abdominal pain which may radiate to the shoulder, and by vomiting; and it is well to bear in mind that these cases may present in two ways—the one being as an ordinary intestinal obstruction (Case 1), and the other as a strangulation of the stomach characterized by frequent vomiting and a flat empty abdomen (Case 2). Other signs may be entirely absent (Case 1), or may consist of dullness and diminished movement of the left side of the chest, with an apex beat which is displaced to the right (Case 2), and perhaps bowel sounds or splashing in the thorax. If the strangulation has lasted long enough, as is common because of the difficulties in diagnosis, there will be a sero-sanguineous effusion in the chest cavity, sometimes in large quantities, with dyspnoea, cyanosis, and tachycardia, which are apt to lead to a diagnosis of a purely pulmonary lesion.

The x-ray findings may consist of a heart displaced to the right, obvious bowel or stomach in the chest, or an apparently raised diaphragm or of appearances as in Case 2. If the effusion is big enough it may mask the underlying

lesion. And, finally, the patient may be so ill that an x-ray examination is not attempted.

Treatment is operative. Conservative measures always result in death, as is so well described by Guthrie; but even with operation, unless undertaken very early, the prognosis is poor.

The incision can be abdominal (Harrington advises one parallel to the costal margin for the left side), which is most satisfactory if the bowel is not distended and if the liver is mobilized by division of the left triangular ligament; it can be thoracic, which is necessary on the right and good on the left because the contents of the hernia are usually adherent to the margins of the opening in the diaphragm or to the praecordium or chest wall, although it has the disadvantage, particularly if resection of gut is needed, of opening the pleura (but it is necessary to remember that the pleural cavity will usually communicate with the abdomen when the gut is withdrawn, whatever incision is used); or it may be abdomino-thoracic, which gives good exposure and can remain extrapleural, as in Case 1.

Biliary Peritonitis

Case 3

A man aged 59 had on the morning of December 7, 1948, an attack which his doctor diagnosed as acute cholecystitis. He was admitted on the evening of the 9th with a temperature of 101° F. (38.3° C.), pulse 120, and respirations 30 a minute. He was ill, with a dry tongue and lax skin and an obvious generalized peritonitis, which was thought to be arising from a ruptured empyema of the gall-bladder, although there were no localizing signs. He was not jaundiced.

Gastric suction, intravenous fluids, and chemotherapy were begun, and the abdomen was opened when his condition was a little better. The gut was found to be reddened and distended and there was much free bile in the peritoneal cavity. The gall-bladder, cystic duct, and common bile duct were thick and oedematous as far as the foramen of Winslow, where there was a hole 1 cm. in diameter in the common duct; below this the duct felt normal. No stones were found in the gall-bladder or ducts, or lying free; nor was there any localization of the perforation or of the bile by adhesions or omentum. The gall-bladder, which contained bile of normal appearance, and the common duct were drained.

Progress was not satisfactory, and on December 18 an upper intestinal fistula developed. On December 24 his abdomen burst. The leaking bowel presented and was found to be jejunum; this was repaired. On January 8 the wound burst and was again repaired, and on January 17 the patient died.

Case 4

A woman aged 62 was admitted as an emergency on February 24, 1949 (T. 97.4° F.—36.3° C.; P. 70; R. 20). Her history and physical findings were suggestive of acute cholecystitis of some 12 hours' duration, and she had suffered for some years from "indigestion" and flatulence after meals, especially with fatty foods.

Six hours later her pain was more severe and was generalized, as was her tenderness, and there was some distension of the abdomen; but her general condition, pulse, temperature, and respiration rate were unchanged. She clearly had a generalized peritonitis, but the normal temperature and pulse and the lack of toxicity made its origin doubtful, as it was not realized at the time that a biliary peritonitis can present in this way.

Her abdomen was opened and was found to contain much normal-looking bile, of which 20 oz. (568 ml.) was aspirated. There were a few adhesions between the gall-bladder and duodenum, but a close examination of the biliary system revealed no other abnormality. No site of biliary leak could be found.

Drainage of the gall-bladder was decided upon, and it seemed reasonable to do this internally rather than externally. A cholecyst-jejunostomy was therefore done.

Her post-operative course was uneventful and she was discharged on the seventeenth post-operative day. She has remained well up to the time of writing—more than a year later.

Case 5

A man aged 79 was admitted as an emergency on October 4, 1949 (T. 99° F.—37.2° C.; P. 80; R. 25), with an obvious generalized peritonitis and faint jaundice. His pain had begun 48 hours and his jaundice seven to eight days before admission. He was cyanosed, with gross venous congestion, and had a history of precordial pain with effort for some years, and breathlessness and swelling of the feet for one year. There was no past history suggestive of cholecystitis.

On opening the abdomen free normal-looking bile was found and some 25 oz. (710 ml.) sucked out. The gall-bladder was red, thickened, and distended, with one or two blackened areas but no perforation. No site of leakage was found, but the examination was cursory because of the patient's general condition. The gall-bladder, which contained bile normal in appearance and no stones, was drained and a second tube put into the region of the common duct.

The patient's cardiac condition remained precarious, and he died on the fifth post-operative day from heart failure.

Necropsy revealed no perforations in the biliary system; but there were several stones in the common bile duct, and gross myocardial degeneration.

Comment on Cases 3-5

Free escape of bile into the peritoneal cavity from perforation of an inflamed gall-bladder or bile duct (Case 3) is not only rare but a most serious affair. McWilliams (1912) found it to occur in only 0.9% of over 3,000 operations on the biliary system. In 90% of these the gall-bladder had perforated, and half of the patients died, but of those operated upon within 12 hours, with drainage of the gall-bladder and its site or with cholecystectomy, only 15% perished. The diagnosis of these cases can be most difficult, as is shown by 28 cases from the London Hospital, reported by Fifield (1926), of which 4 were diagnosed as intestinal obstruction, 3 as perforated peptic ulcer, 2 as high appendix abscess, and only 9 as acute cholecystitis; the rest were described as peritonitis of unknown origin.

In contrast to the severity of an infected biliary peritonitis is the escape of sterile bile as the result of an injury. Numerous cases have been reported and discussed (Ricketts, 1905; Wangenstein, 1926; Mentzner, 1934). These patients survive in good health for days or weeks, but they finally all die unless the bile is removed by frequent tapings or operation.

To complete the discussion of biliary peritonitis from an obvious source, it is of interest to mention post-operative leakage. Such a leak is usually harmless, but it can be a most serious matter. Cases described by Douglas and Grey Turner (1940) and Lewis (1940) were characterized by much post-operative pain in the right hypochondrium, followed, some 12 hours later, by profound shock. Lewis's case recovered rapidly when the collection of bile was drained; the other did not rally enough for laparotomy, and died.

Free bile in the peritoneal cavity without a perforation of the gall-bladder or bile ducts (Cases 4 and 5) is well recognized but is a rare and interesting phenomenon, and Case 4 is of particular interest because of the normal appearance of the biliary system (except for a few adhesions), and because the diagnosis could have been made from the rapid onset of peritoneal irritation and intestinal distension, without a rise in temperature and without the signs associated with a bacterial peritonitis.

Both cases are of interest in that they seem to support the view of Buchanan (1918) and Stenson (1937), based on a review of some 40 cases from the literature, that biliary peritonitis cannot be explained on any one theory. Thus Clairmont and von Haberer (1910) showed that ligation of the common duct in dogs will cause a biliary peritonitis without a perforation, and described a case following obstruction of the common duct in man. They suggested that the rise of pressure and distension may allow filtration of bile from an apparently normal gall-bladder in some unexplained way; but in Case 4 no obstruction was found, and certainly no distension of the gall-bladder or ducts. Blad (1917) suggested that, where there is obstruction of the ampulla of Vater, there may be a reflux of activated pancreatic juice into the gall-bladder, producing a digestion necrosis and permeability of its wall to bile, but without rupture (this can be produced experimentally in dogs). This theory would account very well for Case 5, but does not seem applicable to Case 4.

In some cases there may be a small perforation which cannot be found, especially if it is retroperitoneal (Nauwerck and Lübke, 1913; Buchanan, 1918; Cope, 1925). This is suggested by a case of Sohn's (quoted by Stenson, 1937) in which a needle was put into the common bile duct to locate it during an operation unconnected with the biliary system. The field was dry at the end of the operation, but the patient subsequently died of a biliary peritonitis. At necropsy the hole in the common duct was found only with the greatest difficulty. This theory might account for both cases, but it is difficult to believe that a considerable escape of bile would occur without at least some distension of the gall-bladder.

Finally, the leak may have occurred from rupture of a small duct on the surface of the liver (Buchanan, 1918; Smith, 1926), but no abnormality of the liver was seen, and the good result following cholecyst-enterostomy in Case 4 suggests that the leak was from the main bile duct system. Whether anastomosis of the gall-bladder to jejunum was a good idea remains to be seen, but the post-operative course was excellent, the patient has remained well for a year, and reports from the Mayo Clinic (Walters, Gray, and Priestly, 1948) and others (Peterson and Cole, 1948) suggest that cholecyst-enterostomy is a satisfactory operation.

Herniation of Gut, with Strangulation, Through a Hole in the Greater Omentum

Case 6

A woman aged 86 was admitted on January 5, 1949, complaining of a constant pain in the epigastrium and right hypochondrium for six hours. She had vomited four times, and her bowels had not been opened for 24 hours, but she had been passing flatus.

Her temperature was 97.6° F. (36.4° C.), pulse 89, and respirations 20 a minute, and she appeared to be neither ill nor distressed. Upon examination of her abdomen a tender indefinite intra-abdominal lump, about 5 in. (12.5 cm.) in diameter, which moved on respiration and was freely mobile, was found to the right of the umbilicus. No other signs were observed.

On the 6th her pain had gone and there was no further vomiting, but the mass was much more distinct. On the 7th she had a recurrence of pain, which was now severe. There was no vomiting and her temperature was 98° F. (36.7° C.), but her pulse had risen to 130 and her respirations to 30 a minute. Her abdomen was very tender to the right of the umbilicus, and rigidity of the abdomen rendered the lump impalpable. There was no distension.

Laparotomy was decided upon, and the abdomen was opened through a right para-umbilical incision. The intestines were not dilated. It was found that the greater omentum was attached in the region of the right internal inguinal ring (there was no hernia), and ileum had passed and strangulated through a hole in the omentum at the level of the umbilicus, forming a rounded mass. The gut was gangrenous; it was resected and end-to-end repair done; the omentum was freed and the hole repaired.

Her post-operative course was uneventful and she was discharged on the fourteenth post-operative day.

Case 7

A woman aged 53 was admitted on December 7, 1949, complaining of vague abdominal discomfort for some days and, beginning 20 hours before admission, severe central abdominal colic and vomiting.

Some hours after the start of the severe pain she was seen by her doctor, who diagnosed acute cholecystitis and gave her $\frac{1}{2}$ gr. (16 mg.) of morphine and 1/100 gr. (0.65 mg.) of atropine. This relieved her for some hours, but the pain recurred. She was then given 50 mg. of pethidine every four hours until her admission.

On admission her temperature was 98° F., pulse 100, and respirations 20 a minute. She was not in pain, but had a furred tongue, and there was localized tenderness and guarding over an area about 6 in. (15 cm.) in diameter to the right of the umbilicus. It was thought that an indefinite lump could be felt in this area, but the guarding made it difficult to be sure. The abdomen was flat.

When the effects of the pethidine had worn off, her colic and vomiting began again and her tenderness became more generalized. Intestinal obstruction was diagnosed and the cause tentatively suggested.

On opening the abdomen, conditions identical with Case 6 were found (except that the bowel was viable). The contracted state of all the intestines was striking. The offending omentum was dealt with and her post-operative course was uneventful.

Case 8

A man aged 68 was admitted to the General Infirmary, Leeds, on July 2, 1950, complaining of generalized colicky abdominal pain and vomiting for three days. His bowels had not acted during this period, nor had he passed any flatus. He had noticed constant abdominal pain and tenderness for about 12 hours, as well as the more severe spasms of colic.

On examination the temperature was 98° F., pulse 80, and respirations 20. The patient looked ill and had a dry furred tongue. His abdomen did not move with respirations, and was very tender and rigid all over, with shifting dullness in the flanks.

Rectal examination revealed a tense fluctuant swelling in the recto-vesical pouch, which was considered to be distended gut. No other abnormal physical signs were found.

Aspiration of the stomach and intravenous therapy were begun and the abdomen was opened. Much free blood-stained fluid was found, together with a tense segment of lower ileum, which had strangulated through a hole in the edge of the greater omentum and was lying in the pelvis. It was gangrenous and was resected, and a side-to-side anastomosis was done. The rest of the small bowel was only slightly distended.

The patient's post-operative course was uneventful, and he was discharged to a convalescent home on July 10.

Comment on Cases 6-8

Of the 25 cases of strangulation of gut through a hole in the greater omentum (22 listed since 1840 and three described above) all but three presented as typical acute intestinal obstruction of small bowel (Martzloff, 1930; Sanes and Postoloff, 1944; Hindmarsh, 1949). Of the 12 case histories which were available, one (Curran, 1869) showed acute localized tenderness and hyperaesthesia over

the strangulated gut. Four cases (Lisanti, 1899; Sanes and Postoloff, 1944; Hindmarsh, 1949; and Case 7) presented with a tender palpable mass which was the involved bowel. Two cases had definite localized signs (tenderness and hyperaesthesia, Martzloff, 1930; a tender lump, Case 6), although neither presented as an intestinal obstruction. These signs would be expected from the pathology, for when the gut passes through the omentum it lies just behind, and is indeed held in contact with, the anterior abdominal wall, and when it becomes congested it should be palpable and must produce local irritation of the parietes. It should therefore be possible to suggest the cause of an acute intestinal obstruction of small bowel presenting with either of the above signs.

Considering Martzloff's case (where symptoms were present for at least nine days), Case 6, where a lump was present for at least two days before it finally strangulated, and Case 7, where the patient had had abdominal discomfort for some days before the final attack, it is evident that the bowel does not necessarily strangulate at once; but in none of the cases described could it have been long through the hole, for in none was it adherent to the omentum.

Summary

Two cases of strangulated diaphragmatic hernia are described. These illustrate the two ways in which a strangulated diaphragmatic hernia may present—that is, with or without premonitory symptoms and with or without signs in the chest, and as either a typical intestinal obstruction or as an obstruction of the stomach with a flat, empty abdomen.

Three cases of biliary peritonitis are recorded: one with a perforation in the common bile duct, and two with no demonstrable site for the biliary leak.

The theories of causation, and the treatment, are briefly discussed.

Three cases of herniation of gut, with strangulation, through a hole in the greater omentum, are reported. Such cases nearly always present as an acute strangulation of small bowel and often have localizing signs, so that a pre-operative diagnosis is possible.

Cases 1-7 were admitted to the Radcliffe Infirmary, Oxford, and Case 8 to the General Infirmary, Leeds. I would like to thank Dr. A. M. Cooke and Mr. Elliot Smith for allowing me to include Case 2, and Mr. D. A. Abernethy and Mr. H. W. Symons for permission to treat the remainder and publish this report.

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FAMILIAL CRISIS IN ACHOLURIC JAUNDICE

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There are on record at least seven instances of spherocytic anaemia in which the haemolytic crisis occurred in a number of members of a family within a short interval (Murray-Lyon, 1935; Scott, 1935; Dedichen, 1937; Damashek, 1941; Lyngar, 1942; Horne *et al.*, 1945; Owren, 1948). We should like to present a series of four cases which occurred in a family of seven.

Case 1

Thomas C., aged 32, a zinc sprayer, on August 12, 1948, became giddy, had the "shivers," and went to bed. The next morning he awoke perspiring. He got up, but soon after developed dizziness and nausea. On the 14th his doctor considered him to be a case of catarrhal jaundice. Next day the patient arose and nearly collapsed. He was then sent to the Smethwick Fever Hospital as a case of typhoid fever. When seen he complained of abdominal pain. He had noticed that on several occasions his urine was dark.

On examination his temperature was 101° F. (38.3° C.), and pulse 80. The conjunctivae had an icteric tinge, and the spleen was enlarged and tender. Blood culture revealed no growth. A blood count on August 16 showed: red cells, 2,700,000; haemoglobin, 55%; colour index, 1; white cells, 2,300 (polymorphonuclears 50%); packed cell volume, 24%; mean cell volume, 88 μ^3 ; mean corpuscular haemoglobin concentration, 33%. The urine contained no urobilin.

The patient was transferred to the General Hospital, Birmingham, on August 19 under Professor Thomson. He was symptom-free and slightly icteric; the spleen was two finger-breadths below the costal margin; and his knee-jerks were diminished. Otherwise no abnormal physical signs were found. On the day of admission his blood count was as follows: red cells, 1,900,000; haemoglobin, 40%; colour index, 1.06; white cells, 6,300 (neutrophil polymorphonuclears 53%, eosinophils 4%, basophils 1%, lymphocytes 28.5%, monocytes 11%, metamyelocytes 1.5%, Türk cells 1%); packed cell volume, 15%; mean cell volume, 80 μ^3 ; mean corpuscular haemoglobin concentration, 37%; the red cells showed anisocytosis and spherocytosis; reticulocytes, 0.1%—two days later they were 0.4%.

The Donath-Landsteiner and acid haemolysin tests were negative. Agglutinations for leptospirae were negative. Coombs's test was negative. The patient was Group O rhesus-positive. Urine at this time showed large amounts of urobilinogen. The serum bilirubin was 0.7 mg. per 100 ml.; direct van

den Bergh negative; blood urea, 22 mg. per 100 ml.; Wassermann reaction doubtful; Kahn test negative.

On August 21, nine days after the onset of illness, a sternal puncture revealed:

		%	Normal
Polymorphonuclears	Neutrophil	28.00	20-50
	Eosinophil	1.50	0-4
	Basophil	0.25	0-1
Metamyelocytes	Neutrophil	22.50	2-8-12
	Eosinophil	0.50	0-2.5
	Basophil	—	—
Myelocytes	Neutrophil	15.50	2-8
	Eosinophil	—	0-1
	Basophil	—	—
Premyelocytes	—	0.25	0.5-5
Myeloblasts	—	—	0-2.5
Lymphocytes	—	7.00	5-20
Plasma cells	—	1.00	0-1
Reticulum cells	—	1.75	—
Normoblasts	Late	0.50	7-19
	Intermediate	2.25	4-15
	Early	11.75	—
Macronormoblasts	Late	0.25	—
	Intermediate	—	—
Proerythroblasts	—	6.75	0-4
Haemocytoblasts	—	—	0-1

Megakaryocytes were normal in number and appearance. The marrow showed some slight increased cellularity and a shift to the left of the erythroid and myeloid series.

On August 23 his blood count showed a marked change: red cells, 2,810,000; haemoglobin, 50%; colour index, 1.08; white cells, 16,100 (polymorphonuclears 52.5%, eosinophils 0.5%, lymphocytes 16.5%, monocytes 4.5%, premyelocytes 0.5%, myelocytes 4.0%, metamyelocytes 21.5%); reticulocytes, 16.2%; platelets, 439,000 per c.mm. A fragility test showed: commencing haemolysis, 0.60% saline (control, 0.40% saline); complete haemolysis, 0.32% saline (control, 0.32% saline).

By August 26 his reticulocytes were 25.2%, and on the 30th his haemoglobin had risen to 72%. A fragility test repeated several weeks later showed: commencing haemolysis, 0.64% saline (control, 0.44% saline); complete haemolysis, 0.44% saline (control, 0.32% saline).

Case 2

Thomas C., jun., a schoolboy aged 10, became ill with vomiting on August 1, eleven days before his father. He complained of headache and pain in the eyes, and was noticed to become rapidly pale. He was admitted to the fever hospital with a diagnosis of meningitis.

On examination he was extremely pale and had a temperature of 99.4° F. (37.4° C.). No other abnormal signs were found. The blood culture was sterile. His blood count five days after the beginning of the illness was as follows: red cells, 1,500,000; haemoglobin, 30%; colour index, 1; polymorphonuclears, 77%, with 3% myelocytes. He was transfused with two pints (1.14 litres) of blood, and his condition rapidly improved. The spleen was not palpable. He was transferred to the General Hospital on August 24.

On admission his blood count was: red cells, 3,910,000; haemoglobin, 76%; colour index, 0.97; white cells, 14,400 (neutrophil polymorphonuclears 75%, eosinophils 3%, lymphocytes 17.5%, monocytes 4.5%). Films showed anisocytosis, spherocytosis, and some polychromasia. Reticulocytes, 8.4%; platelets, 352,000 per c.mm. He was Group O, rhesus-positive. Coombs's test, the acid haemolysin test, Donath-Landsteiner test, and Wassermann reaction were negative. A fragility test showed: commencing haemolysis, 0.56% saline (control, 0.44% saline); complete haemolysis, 0.32% saline (control 0.28% saline).

The urine showed an excess of urobilin. Serum bilirubin was 1.1 mg. per 100 ml.; blood urea, 72 mg. per 100 ml.; and leptospiral agglutinations were negative. The sternal puncture showed increased cellularity. There was no other abnormality.

Cases 3 and 4

Case 3.—Valerie C., aged 6, first had abdominal pain and vomiting on August 10. She was admitted to the fever hospital on the 16th and found to be extremely pale, with a slight icteric tinge. Her temperature was normal. A blood count showed: red cells, 2,700,000; haemoglobin, 40%; colour index, 0.74;