ago, Professor Osler published four new cases in August, 1903,⁷ and other cases have been recorded in America and on the continent. In Dr. Russell's case the spleen weighed 48 ounces, in Dr. Weber's 23 ounces, but in two of Professor Osler's it was not demonstrably enlarged.

The nature of these cases is so far obscure. Dr. Weber found red transformation of the marrow of the left humerus, the left femur, and the left tibia. The marrow contained numerous nucleated cells representing myelocytes, eosinophile myelocytes, erythroblasts, and large endothelial cells and giant cells, and thus appeared to consist largely of tissue engaged in the formation of red blood corpuscles. And he regards the condition as a disease primarily of the bone marrow. But Dr. Russell had seen his patient with an enlarged spleen at least six years before the condition of cyanosis developed. In both these cases the spleen appeared to be only engorged and not to present any indication of being primarily diseased.

CHLOROMA.

I may still more briefly allude to a condition in which the spleen is involved—namely, that of chloroma. In this rare disease, in which lymphoid tumours of a green colour occur throughout the body, the blood presents the characteristics of a lymphocythæmia or lymphæmia and the spleen is enlarged by infiltration with lymphoid deposits or lymphocytes. The relation between chloroma and lymphatic leukaemia is believed to be very close but there remains no doubt a great deal of work to be done on both these conditions.

PRIMARY AND SECONDARY ANÆMIAS.

The attempt to distinguish between primary and secondary anæmias seems here to fail completely. The recognised primary anæmias are: (a) chlorosis and (b) pernicious anæmia. Among the secondary anæmias must be classed: (a) splenic anæmia, (b) infantile splenic anæmia (von Jaksch's), and (c) the anæmia of leukaemia. But after we have been searching into the pathology of these diseases, what essential difference do we see between these groups? In one of the primary anæmias, pernicious anæmia, a large number of facts have been collected which at least suggest, if they do no more, that some kind of toxic or infective process is at work which is not any obvious defect of food, or loss of blood, or continuous vital strain, or primary disease of any organ charged with blood formation; for although the spleen is sometimes enlarged it is not so constantly; it is often indistinguishable from that of health, and, moreover, the share of the spleen in the formation of the blood is at least doubtful. On the other hand, in splenic anæmia of adults the only explanation which at all satisfactorily introduces the spleen as a part of the causation regards the splenic lesion not as the primary condition but as an intermediate factor between absorption of toxins from the intestines and the morbid condition of the blood.

Secondly, in infantile splenic anæmia intestinal poisoning is again rather widely accepted as the essential cause and the splenic enlargement is looked upon as rather an accident than otherwise, so that in a great number of cases very little is said of the condition of the spleen beyond the fact that it presents an enlargement which is very different in different cases.

With regard to leukaemia the early impressions seem to have been twofold: one that the anæmia was due to the disease of the spleen or glands or marrow, whichever one or two or three were involved; the other that the extreme leucocytosis either necessitated a smaller number of red corpuscles as a matter of crowding out, or that the undue proportion of leucocytes represented an imperfect condition of the blood, of which the red corpuscles were the highest development. But with regard to the spleen it is almost certain that the primary lesion does not lie there, nor even in lymphatic leukaemia is it certain that the cause lies in the glands.

Thus we have reason to believe that we can see some cause in an anæmia regarded as primary and that in a form regarded as secondary the cause does not really lie in the organs originally thought to be primarily at fault. Indeed, primary is almost as unsatisfactory a group as idiopathic. It is only valid so long as our ignorance lasts or till fresh facts come before us to advance our knowledge. It is, indeed, no question of causeful or causeless cases; it is only the question of whether the essentials of the blood are

diminished by destructive hæmolysis or by failure of production—i.e., an hæmogenesis or deficient hæmogenesis. Or, thirdly, whether the deficiency of essentials is only relative or due to an increased quantity of plasma.⁸ Hæmorrhage is a coarse hæmolysis; pernicious anæmia is a hæmolysis in detail. Starvation is a coarse an hæmogenesis; various processes of disease are an hæmogenesis in detail. It is for us to find out what in each case are the causes of hæmolysis or an hæmogenesis. Of hæmolysis we have other examples in chlorate of potassium poisoning and in paroxysmal hæmoglobinuria. As a cause of the hæmolysis of pernicious anæmia we are led to look to some infective process, peculiar in this—that it alone, if it is a single toxin, should produce a hæmolysis while so many others do the reverse. Of an hæmogenesis or deficient hæmogenesis we have examples in nearly all the cases where anæmia follows upon prolonged infective disease. It may be very difficult to estimate the share which is to be attributed to actual deficiency in food supplied and that which other processes may have. But there are enough instances of anæmia occurring in cases when the food is not necessarily much diminished to make it probable that if the processes of splenic anæmia in children, splenic anæmia in adults, and chlorosis are due to some kind of unbottled poison inhibiting the process of blood formation, the same is taking place to account for the anæmia of syphilis, rheumatism, phthisis, empyema, prolonged sepsis from whatever cause, Bright's disease, and many other allied conditions.

CONCLUSION.

In looking back on these "disorders of the spleen," of which my review has been so fragmentary and imperfect, one is brought to the conclusion that the spleen is more sinned against than sinning; that it is rarely responsible for the lesions which it suffers or for the complaints with which it is associated; that in the various infective disorders, in splenic anæmia, and in infantile anæmia it is probably poisoned from without; that in the different forms of leukaemia it is overcharged with the excess of leucocytes; and that only in splenic anæmia as yet is the charge seriously made against it, that itself having been poisoned from the bowel it subsequently inhibits by fresh production of poisons the formation of the blood. It is in these diseases that good results have sometimes been obtained by splenectomy, though it must be admitted that death has sometimes taken place shortly after the operation. In the other cases the primary condition must be treated, and in so far as that can be controlled or will spontaneously recover the enlargement of the spleen may be expected to subside.

In spleno-medullary leukaemia considerable reductions in size of the enlarged spleen have occurred under arsenic and sometimes under inhalations of oxygen, but a relapse appears in this disease to be almost inevitable. The spleen has also been extirpated for malarial enlargement, and it is recorded⁹ that of six cases operated on four recovered.

I have, Mr. President and Gentlemen, to express to you my gratitude for your kind and considerate attention during these three lectures, which again I cannot help thinking will have fallen very far short of what you have a right to expect.

A CASE OF PERFORATED GASTRIC ULCER IN A BOY AGED 13 YEARS; DISEASED APPENDIX; OPERATION; RECOVERY.

BY W. WATSON CHEYNE, F.R.S., F.R.C.S. ENG.,
PROFESSOR OF CLINICAL SURGERY, KING'S COLLEGE;

AND

HAYDOCK WILBE, M.D. DURH., L.R.C.P. LOND.,
M.R.C.S. ENG.

ON March 16th, 1904, the patient, a boy, aged 13 years, who had previously been perfectly well, complained of some stomach-ache during the afternoon when returning from school. In the evening this became worse and he asked to be allowed to go early to bed; his mother put a hot water bottle over his stomach which relieved him in a few minutes. He got up on the morning of March 17th much better and

⁷ American Journal of the Medical Sciences

⁸ Lorrain Smith: Transactions of the Pathological Society, 1900, p. 311.

⁹ Scheube: Warm Countries (English translation), p. 169.

practically felt well except for a little abdominal pain when stooping; the exact position of the pain was not well defined. He, however, asked to be allowed not to go to school. In the morning he had some Eno's fruit salt and ate a very good dinner that day. About an hour after dinner he began to complain of severe abdominal pain and very soon became seriously ill. Dr. Wilbe was sent for and saw him between three and four o'clock in the afternoon and found him complaining of violent pain in the abdomen which he could not localise very precisely but he placed his hand towards the right side and about the umbilicus. He found him very collapsed, with a pulse of about 130, with marked rigidity of the abdomen and tenderness over the whole abdomen. The symptoms were especially marked towards the right side and upper part of the abdomen. There was no vomiting. The temperature was normal. Mr. Watson Cheyne saw the patient at 8.30 in the evening. At that time the boy looked extremely ill; there was no abdominal respiration; the *alæ nasi* were moving; there were marked rigidity of the whole abdomen and general tenderness but no abdominal distension. The pain and rigidity were not well localised but were on the whole most marked towards the upper part on the right side. The abdomen was tympanitic everywhere but there was no distinct obliteration of the liver dulness. There had been no vomiting. The temperature was normal. The conclusion came to was that there had been a perforation of some abdominal organ, and from the general history and symptoms pointing more towards the right side and the probability at that age that the trouble was of the form of a fulminating appendicitis an incision was in the first instance made over the seat of the appendix. On exposing the peritoneum it at once bulged out in the wound and on puncturing it gas whistled out. On enlarging the opening the small intestines were forced out, partly by the gas and partly as the result of the great rigidity of the abdominal walls, which had not become relaxed under the anæsthetic. Some thin whitish fluid also came out of the abdomen. For the most part the intestines were normal in appearance but on some of the coils of the small intestine, which came apparently from the left side of the abdomen, there were some flakes of lymph. The pelvis and lower part of the abdomen were well irrigated with salt solution till the fluid returned clear, and the intestines were then, gradually but with considerable difficulty, returned to the abdominal cavity. The cæcum and appendix were now pulled out. The appendix was long, thickened, a good deal reddened, and contained a large concretion; but there was no perforation of the appendix nor of the cæcum in the neighbourhood. The appendix was removed in the usual manner.

As it was evident that there must be some graver lesion than the condition of the appendix to account for the state of matters and as practically the whole of the small intestine had been seen, and as also there was no faecal material present in the abdominal cavity, a second incision was now made over the stomach. At first nothing seemed to be amiss. The body of the stomach and the pyloric end seemed perfectly healthy; the region of the duodenum was examined; the stomach was turned over and the posterior surface palpated and still no perforation was found. On further examination of the anterior surface high up towards the cardiac end some turbid fluid and gas bubbled up. After wiping that away and directing a good light into the cavity high up under the diaphragm, a round perforation was found on the anterior surface of the stomach about an inch from the cardiac end which would have admitted a small slate pencil. From this perforation gastric juice and gas were escaping. There was no thickening around; it was a clean punched-out hole and all the structures in the neighbourhood seemed perfectly healthy. The edges of the ulcer were inverted with a row of catgut sutures and a second row was then put outside. The peritoneal cavity in the neighbourhood was thoroughly sponged out and the whole cavity again washed out with salt solution. A drainage tube was passed down towards the seat of the perforation, another towards the left loin, and another (from the lower wound) towards the pelvis. The rest of the wounds were stitched up. The boy stood the operation very well and his pulse after it was 120. On the night of March 18th the wounds were dressed. There had been very little discharge. The abdomen was very much distended. The pulse was 130 and the temperature was 99.2° F. There was a good deal of tenderness everywhere. No flatus had been passed. There was very little sickness. The boy had not slept and

was somewhat excited. On the whole he looked rather better than he did on the previous day. He had been having rectal feeding with sips of water by the mouth. On the 19th the abdomen was still in much the same condition but the patient did not seem any worse than he was on the 18th. The pulse and temperature were the same. There was less tenderness at the lower part of the abdomen and there had been no vomiting. A little flatus had been passed but not enough to give him relief. Enemata had been given but without any result. At our visit in the evening we gave him an enema again and evacuated some faeces and flatus which relieved him very much. The drainage tubes were pulled out and the greater portion cut off, leaving only a small part projecting into the peritoneal cavity. There was very little discharge. On the 20th the patient was distinctly better. The abdomen was not so tense but still very much distended. He had passed a little flatus and slept a little during the night. The tubes were left out. There was very little discharge. He began to take small doses of salines and salol. On the 22nd the patient had a good action of the bowels in the afternoon and the abdomen was much less distended. He was very much better. On the 23rd feeding by the mouth was begun. From this date the boy went on uninterruptedly well. Solid food was allowed on April 1st. The wounds had completely healed on the 13th. The patient was allowed up for the first time on the 14th and went out in a bath chair. On the 25th the boy was in high spirits and had apparently perfectly recovered.

The interest of this case lies, in the first place, in the occurrence of a perforating gastric ulcer in a boy, aged 13 years. We can find no record of perforation of the stomach in a male at such an early age. In this case the ulcer was typically a peptic ulcer. Gastric ulcers have been found in younger children but apparently only of a tuberculous character. Thus Dr. G. F. Still¹ recorded five examples of tuberculous ulcers of the stomach in children, in three of which histological and bacteriological examination had been made with positive results. In all cases the ulcers were small. The first was a child, aged three and a half years, who was admitted with vomiting and ascites due to tuberculous peritonitis; the second was a child, aged ten months, suffering from cough and tuberculous meningitis, and, as shown after death, from generalised tuberculosis; the third was a case of tuberculous peritonitis; the fourth was a boy, aged nine years, who was also affected with tuberculous peritonitis; and the fifth was a child, aged two years, suffering from the same disease. The author had only found five cases out of 226 necropsies. There were no clinical symptoms associated with them. In one of the five cases perforation had occurred but extravasation had been prevented by adhesions. In the above case there was no sign whatever of tuberculosis either about the stomach or the abdomen generally nor was there any reason for suspecting it. As to the cause of the ulcer in this boy we cannot make any suggestion and there were no previous symptoms whatever leading to a suspicion that anything was wrong with the stomach.

The second point of interest is that, partly as the result of the symptoms pointing somewhat towards the appendix and partly on account of the great rarity of the actual condition, the appendix was in the first place cut down upon and a *diseased appendix* was found. The condition of the boy was so extremely bad that one was very much tempted to hope that that might be the cause of the whole trouble; but the presence of free gas in the abdomen made it absolutely certain that perforation of a hollow viscus other than the appendix must have taken place, and the absence of faecal material in the abdominal cavity and the fact that practically the whole of the small intestines had passed under review when they were forced out of the wound led us to assume that this perforation must have taken place either in the stomach or duodenum.

The third point of interest is the situation of the ulcer. It was situated so high up and was so difficult of access that its presence was nearly overlooked. Before it was found the whole of the lower part of the stomach had been examined, the duodenum had been investigated, the stomach had been turned up and the posterior part searched, and had it not been that we were convinced that there must be a perforation, and that a further search was made towards the upper part of the stomach, the condition might have been overlooked.

¹ Brit. Med. Jour., May 6th, 1899.