INFANTILE SPLENIC ANÆMIA, WITH
NOTES OF TEN CASES.

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IN reading over the literature dealing with the anæmias of infancy one cannot but be impressed with the very great difference of opinion that exists regarding the etiology and pathogenesis of this group of diseases. Many of the writers, however, recognise that, when regarded from the point of view of their symptomatology, these anæmias may be grouped into several clinical types; though whenever the relationship of one type to another comes up for consideration disagreement is again at once apparent. Anæmia is a common symptom in childhood. It is a symptom which occurs in almost all the more chronic diseases and disorders of infancy, and it may be met with in every degree of severity. In a proportion of the cases there is enlargement of the spleen, especially in those which show the severer degrees of anæmia, though this is not always so. The size of the spleen, however, is a feature in anæmia to which much attention has been paid, and hence the anæmias of infancy have come to be divided, perhaps somewhat artificially, into two groups—namely, those with splenic enlargement and those without. Amongst the latter are classed most of the lesser forms of infantile anæmia. These are essentially secondary or symptomatic anæmias, secondary to a variety of causes, such as bad feeding, faulty assimilation, lack of fresh air, diarrhœa, intestinal worms, rickets, congenital syphilis, and repeated hæmorrhages. In such anæmias the red corpuscles average from 2,000,000 to 3,500,000 per cubic millimetre and the hæmoglobin index is low—from 0·6 to 0·7. A proportion of normoblasts are frequently to be found in stained films and most often there is some variation in the size and intensity of staining of the red cells. There is usually a slight leucocytosis—about 15,000 white corpuscles per cubic millimetre. The symptoms and blood picture will naturally depend on the severity of the anæmia, and this in turn on the severity of the conditions which produce it. The anæmia will be curable in so far as the conditions on which it depends are curable.

But it is with the anæmias associated with splenic enlargement that we are at present concerned, and of these three clinical types have been described, the chief point of distinction between them seeming to consist in the degree of leucocytosis that may happen to be present.

1. In the first type the blood picture is much the same as that in the splenic anæmia of the adult—that is, the white corpuscles are diminished instead of being increased in number and there is most often a relative lymphocytosis.

2. The second type differs from the above chiefly in that the leucopenia is replaced by a moderate leucocytosis of from 14,000 to 20,000 per cubic millimetre, and while some myelocytes are usually to be found in stained films the proportions of the various white cells are otherwise little altered. This is probably a severer variety of anæmia than Type 1, and it would seem to include most of the cases described under the term anæmia splenica infantum.

3. The third type is that described by von Jaksch under the name of anæmia infantum pseudoleukæmia. Here there is a still greater increase of white corpuscles—from 20,000 to over 100,000 per cubic millimetre. In a differential count there is almost always a definite proportion of marrow cells (up to 20 per cent.), and, generally, the variety of leucocytes is greater than is found in normal blood. Most often it is the lymphocyte that is present in largest numbers, but sometimes there is a very definite increase of the polynuclear cells.

Examination of the red blood corpuscles in these three types of splenic anæmia shows nothing distinctive; they have much the same characters as noted in the anæmias without splenic enlargement. The number averages about 2,500,000 per cubic millimetre, with a range of from 3,500,000 to less than 1,000,000 per cubic millimetre; and there is no recognised diminution corresponding to the increase of the white corpuscles in, for example, Types 2 and 3, other than that the severest anæmias are perhaps to be

met with in those cases with the most marked leucocytosis. There need then be but little diminution of red cells, but in almost all the cases the hæmoglobin index is low—0·5 to 0·6. Nucleated red corpuscles are present, usually in proportion to the severity of the anæmia, and of these a proportion may be megaloblasts. In all these anæmias there is considerable variation in the size of the red cells and sometimes in the intensity with which they take up acid stains; in the severer degrees of anæmia polychromatophilia is also met with. Poikilocytosis is not so commonly present as in the anæmias of the adult.

The etiology of infantile splenic anæmia is in dispute, but one may say without much fear of contradiction that the same disorders and diseases as cause an ordinary secondary anæmia (without enlarged spleen) are also to be found in those cases where splenic enlargement is present. Recovery may take place in any of the types of infantile splenic anæmia, and in this respect they differ from the splenic anæmia of the adult, where the patient lives on an average little more than three years after the onset of symptoms. In the child splenic anæmia may last for many years, but during that time there may be much debility and ill health. When recovery does take place it is usually a very slow process, many months' treatment being required before improvement is at all apparent. When death occurs in many of the cases it seems to depend on the development of a broncho-pneumonia. In none of the types is there any distinctive lesion to be found post mortem and in particular it is to be noted that in the so-called anæmia infantum pseudoleukæmia there are none of the lesions characteristic of leukæmia.

The following ten cases which I have collected together will serve to illustrate most of the points I have just mentioned. And here I would like to express my indebtedness to Dr. J. Lindsay Steven and Mr. James Battersby—to the former for permission to include Cases 3 and 7 and to the latter who permits me to add Case 2 to this series. I myself am responsible for the differential blood counts in all the cases and for the histological examination of the tissues in the four cases that died.

CASE 1.—A man, now aged 22 years, first came under my care as an out-patient at the Glasgow Royal Infirmary in 1905, but previous to that he had been at least three times in hospital suffering from anæmia. The enlargement of the spleen seemed to date from infancy, for when the child was nine months old the mother noticed a "lump" in the left side projecting below the costal margin. At this time, however, there were no definite symptoms of illness and the general health remained fairly good till after an attack of measles, when the child was four years old. After this the swelling in the abdomen seemed to get larger; when six years old the anæmia was so severe that he was admitted to the Children's Hospital and remained there for nine months. At that time it was noted that the blood "had the characters of the splenic anæmia of the adult rather than of the anæmia of infancy." For the next seven years the child remained fairly well and was able to attend school, but subsequent to that he suffered from weakness, breathlessness, and swelling of the feet. He had then to give up doing any form of work and was again (March, 1903) admitted to hospital. This time he was under treatment for three months and improved very greatly, gaining 10½ pounds in weight. But he was able to work for only six months, for the symptoms of anæmia appeared and he was again in hospital (March, 1904) for a period of two months. After this he worked for nine months, but in May, 1905, he was back to hospital suffering from his old complaint. For the past three years, however, he has enjoyed good health and has been able to do a full day's work as fireman on a steam engine. He now weighs 9 stones, and though thin is fairly well developed and fairly well nourished. He shows no rachitic deformities and no appearance of anæmia. The spleen during all these years of illness seems to have been greatly enlarged. In March, 1904, it extended "3¼ inches below the umbilicus," and in May, 1905, to "1½ inches from the symphysis pubis." At present (July, 1908) it extends downwards to the anterior superior spinous process of the ilium and forwards to half an inch from the middle line. At no time was the liver noted as being enlarged. The condition of the blood was as follows:—

Date.	Red corpuscles.	White corpuscles.	Hæmoglobin.
	Per c.mm.	Per c.mm.	
April, 1903	 3,860,000	 3,500	 "Greatly reduced."
April 5th, 1904	 2,960,000	 3,100	 —
„ 27th, „	 3,200,000	 2,420	 —
May 16th, 1905	 3,500,000	 6,875	 35 per cent.
June 3rd, „	 4,045,000	 5,600	 35 „ „
June 26th „	 4,680,000	 5,000	 50 „ „
July 12th, 1908	 5,500,000	 8,550	 100 „ „

On examining stained films there was found in 1903 a marked poikilocytosis with considerable variation in the size of the various corpuscles. The red cells stained very faintly, many being only represented by a rim of oxyphile stain at the periphery of the disc. In May, 1905, there was a more moderate poikilocytosis but variation in the size and intensity of staining of the individual red cells; there was only a very small proportion of normoblasts. In July, 1908, the films showed the

red cell normal in size and staining reaction. Of the white corpuscles the following are the proportions:—

	Poly-nuclears.	Small lymphocytes.	Hyaline and transitionals.	Eosinophiles.
	Per cent.	Per cent.	Per cent.	Per cent.
April 27th, 1904	50	40	10	—
May 16th, 1905	55	33	10	2
July 12th, 1908	61	34	3	2

CASE 2.—This patient, a female, first came under my observation in January, 1905, when she was two years of age. She seemed to have been a healthy child up to the age of 16 months when she became fretful, disinclined to play, and generally out of sorts. For eight months she had remained in that condition. Shortly after the onset of these symptoms the abdomen was noted to be swollen, and a medical man who was consulted said that the spleen was enlarged. There never had been diarrhœa and only very occasionally any vomiting. The father, mother, two brothers, and two sisters were alive and well. Another brother died, aged seven years, after suffering for three years

child's condition was greatly improved, the lips and cheeks being well coloured, but the liver and spleen were as large as formerly. She was seen again last summer (June, 1908)—i.e., after an interval of over three years. The blood was then practically normal though there was still a slight leucopenia. The count was as follows: red corpuscles, 4,000,000 per cubic millimetre; white corpuscles, 4687 per cubic millimetre; and hæmoglobin, 95 per cent. The differential estimation of the white cells was: polynuclears, 45 per cent.; lymphocytes, 47 per cent.; hyaline cells, 4 per cent.; eosinophiles, 2 per cent.; and mast cells, 2 per cent. There were no marrow cells. The red corpuscles were of uniform shape and staining, but there was some variation in the size of the individual cells. There were no nucleated red cells. The child is now a picture of health, fat, well nourished, with rosy cheeks and red lips. But the edge of the liver is palpable two inches below the right costal margin and the spleen extends downwards to one inch below the level of the umbilicus.

CASE 3.—This patient, aged 21 months, was first seen on May 5th, 1905, and died five days later with areas of pneumonic consolidation in both lungs. No satisfactory history of the illness could be obtained in this case. On examination the child was seen to be greatly undersized

Table Showing Ten Cases of Infantile Splenic Anæmia.

No. of case.	Age.	Date.	No. of red corpuscles per c.mm.	Hæmoglobin percentage.	No. of white corpuscles per c.mm.	Percentage of polynuclears.	Percentage of lymphocytes.	Percentage of hyalines and transitionals.	Percentage of myelocytes.	Percentage of eosinophiles.	No. of normoblasts per 100 W.B.C.	No. of megaloblasts per 100 W.B.C.
1*	6 years.	1892.	—	—	—	—	—	—	—	—	—	—
17	"	April, 1903.	3,860,000	"Greatly reduced."	3,500	—	—	—	—	—	—	—
18	"	April 5th, 1904.	2,960,000	—	3,100	—	—	—	—	—	—	—
	"	" 27th, "	3,200,000	—	2,420	50	40	10	—	—	—	—
19	"	May 16th, 1905.	3,500,000	35	6,875	55	33	10	—	2	Small proportion.	—
	"	June 26th, "	4,680,000	50	5,000	—	—	—	—	—		—
22	"	July 12th, 1908.	5,500,000	100	8,550	61	34	3	—	2		0
2†	2	Jan. 25th, 1905.	3,900,000	35	2,800	58	34	7	0	1	0	0
	"	Feb. 12th, "	3,500,000	38	7,000	—	—	—	—	—	—	—
	5	June, 1908.	4,000,000	95	4,687	45	47	4	0	2	0	0
3‡	1½	May, 1905.	—	—	11,000	35	50	14	0	1	3	0
4§	1½	" 1905.	1,700,000	—	13,400	32.5	54	13.5	0	—	4	0
5	1½	May 3rd, 1905.	3,800,000	—	12,000	64	24	12	0	—	3	0
	"	" 19th, "	3,695,000	37	19,000	—	—	—	—	—	—	—
	5	July 2nd, 1908.	4,800,000	56	8,222	41.6	48.8	8	0.4	1.2	2	—
6	1½	June 1st, 1907.	1,650,000	25	18,312	31	44	11	8	5	7	3
	"	" 18th, "	2,130,000	20	15,900	—	—	—	—	—	—	—
	"	July 4th, "	1,700,000	20	12,792	—	—	—	—	—	—	—
	"	August 6th, "	3,000,000	15	9,330	—	—	—	—	—	—	—
	"	Sept. 4th, "	2,764,000	25	—	32	50	9	8	1	1	—
	"	Oct. 3rd, "	3,500,000	40	5,000	46	44	7	2	1	1	—
	"	March 16th, 1908.	4,290,000	86	6,400	—	—	—	—	—	—	—
7¶	1½	April, 1905.	1,600,000	—	60,000	34.5	44	8	9.5	4	2	2
8**	1½	July 3rd, 1908.	4,800,000	85	22,500	35	49	7.5	5.5	3	—	—
	"	" 10th, "	5,100,000	95	28,125	—	—	—	—	—	—	—
	"	" 17th, "	4,487,000	75	30,837	38	41.5	5.3	7.4	7.2	1	1
	"	" 24th, "	4,472,000	70	30,000	—	—	—	—	—	—	—
9††	1½	July 19th, 1908.	4,648,000	70	28,225	22	65	4	5	5	1	1
10‡‡	½	April, 1907.	2,088,000	18	95,144	14	66	11	6	2	59	3

* The blood had the characters of the splenic anæmia of the adult. † Died. Nothing distinctive post mortem. ‡ Died. Nothing distinctive post mortem. § Died. Nothing distinctive post mortem. || Died. Infiltration of mononuclear cells into liver and kidneys. ¶ Still under treatment. ** Under observation. †† Under observation. ‡‡ Died. Infiltration of mononuclear cells into liver and kidneys. † Complete recovery. ‡ Made complete recovery. § Died.

with vomiting and an enlarged spleen; he died during an attack of vomiting. On examining the child she was seen to be well nourished, but the skin and mucous membranes were extremely pale. Most of the teeth were present and in a good state of preservation. There were no signs of rickets. The heart and lungs were normal. The abdomen was distended and both liver and spleen were greatly enlarged, the latter extending to the anterior superior spinous process, the former almost to the level of the umbilicus. The examination of the blood was as follows:—

	Red cells.	White cells.	Hæmoglobin.
	Per c.mm.	Per c.mm.	Per cent.
Jan 25th, 1905	3,900,000	2800	35
Feb. 12th "	3,500,000	7000	38

Of the white cells 58 per cent. were polynuclears, 34 per cent. lymphocytes, 7 per cent. hyaline and transitionals, and 1 per cent. eosinophiles. There were no nucleated reds and no marrow cells. The red corpuscles showed a moderate poikilocytosis, with considerable variation in the intensity of staining and size of the individual cells, some of the cells seeming large enough to be regarded as megalocytes and others quite as small as microcytes. After two months' treatment the

and in a state of extreme emaciation. The pallor of the skin and mucous membranes was very marked. The anterior fontanelle was widely open and there was some beading of the ribs, but no other signs of rickets. There were eight teeth in fair preservation. The abdomen was enlarged and both liver and spleen were readily palpable below the costal margin, the spleen extending to within three-quarters of an inch of the anterior superior spinous process of the ilium. The axillary, inguinal, and popliteal glands were palpable but not very definitely enlarged. The temperature remained normal for the five days the child was under observation. There were 11,000 white corpuscles per cubic millimetre, made up as follows: polynuclears, 35 per cent.; lymphocytes, 50 per cent.; hyaline and transitionals, 14 per cent.; and eosinophiles, 1 per cent. There were three normoblasts to 100 white cells. A count of the red cells was not made in this case, but it must have been very low judging from the pallor of the skin and mucous membranes. Films showed a moderate poikilocytosis but considerable variation in the size of the red cells, many being much smaller and a few rather larger than normal. There was also considerable variation in the staining reaction of the red cells, some being very pale and others taking the stain with considerable intensity. Neither megaloblasts nor marrow cells were to be found.

At the post-mortem examination the heart showed no signs of valvular or other disease. In the lungs there were patches of pneumonic consolidation in the right lower and left upper and lower lobes. The liver (7½ ounces) was congested, as was also the spleen (1 ounce). In the right kidney there were a number of wedge-shaped hæmorrhages suggesting infarctions. The mesenteric glands were slightly increased in size. The bone marrow of the right forearm bones was red and semi-fluid. On microscopic examination there was seen to be but slight fatty change in the liver cells and no definite increase of the connective tissue in the portal areas. The spleen pulp was much congested and possibly there was a slight fibrosis but this was not very definite. Except for marked congestion the kidneys seemed healthy. The consolidated areas of the lung presented the appearances of a fairly typical broncho-pneumonia. The heart muscle seemed healthy. The thymus gland showed considerable increase of the interlobar and interlobular connective tissue but there was no proper differentiation into cortex and medulla; the gland seemed encroached on by this fibrous tissue, but the medulla seemed to have suffered more thereby than the cortex. The bone marrow presented much the same appearances as in Case 7. There was, however, even a larger proportion of lymphocytes throughout the section and the aggregations of lymphocytes were also well marked. The prevailing cell nevertheless was the finely granular marrow cell. The number of giant cells was probably less than in the case just referred to.

CASE 4.—A girl, aged 14 months, was only under observation for 48 hours when she died with a pneumonic condition of the lungs. She was a fairly well nourished child, but there were signs of rickets in the curving of the bones of the legs as well as in the beading of the ends of the ribs. The skin and mucous membranes were exceedingly pale. There was no history obtained regarding the mode of nourishment of the child. Both liver and spleen were palpable below the costal margins. Examination of the blood showed the red corpuscles to number 1,700,000 per cubic millimetre and the white corpuscles 13,400 per cubic millimetre. Of the white cells 32.5 per cent. were polynuclears, 54 per cent. lymphocytes, and 13.5 per cent. hyaline and transitional cells. There were four normoblasts to 100 white cells, but neither marrow cells nor megaloblasts were found. The red corpuscles showed slight poikilocytosis and a certain amount of variation in the size of the individual cells, but few were large enough to be classed as megalocytes. There was slight variation in the intensity with which the cells stained with eosin, some being very pale and others quite up to the normal standard.

At the post-mortem examination the upper and middle lobes of the right lung were found to be consolidated, and there were several areas of consolidation scattered through the upper two-thirds of the left upper lobe. Both lower lobes were congested. The heart was pale but with no appearance of mottling. The valve segments were normal. The liver (1½ ounces) was congested and showed cloudy swelling. The spleen (1½ ounces) was enlarged and congested. The kidneys were pale. The thymus was also pale but of about normal size. The stomach and intestines, beyond some congestion of their mucous membranes, showed no abnormality. The bone marrow of the right femur was red. On microscopic examination the liver cells presented the appearance of slight fatty degeneration, but there was no definite increase of the connective tissue in the portal areas. In the spleen there seemed a slight fibrosis and the Malpighian bodies were about normal in size. The kidneys were normal. The heart muscle, stained with osmic acid, showed a slight fatty change. The consolidated lung had the characters of a fairly typical broncho-pneumonia. The mesenteric glands gave the appearance of considerable activity, for there was no proper differentiation into cortex and medulla and the sinuses were somewhat over-run with lymphocytes. In the thymus gland there was considerable increase of the interlobar and interlobular connective tissue. But while in this respect there seemed to be an atrophy of the gland, the parenchyma, on the other hand, showed no proper differentiation into cortex and medulla and so suggested an over activity of the gland. The concentric corpuscles of Hassall seemed larger and more numerous than normal. The marrow had much the same characters as in Case 7. There seemed, however, to be a larger proportion of lymphocytes and in places these cells were collected together to form little masses, giving something of the appearance of the Malpighian bodies in the spleen. The number of giant cells, too, seemed greater than in Case 7.

CASE 5.—A girl, aged 20 months, was seen by me several times during the month of May, 1905. The child was born at full time and at birth was apparently quite healthy. The mother, however, had not been robust during the later months of the pregnancy. There had been two miscarriages after the birth of the first child but there were no appearances of syphilis in any of the children. There were two other children, the eldest being four years old and strong and healthy; the youngest was a babe of one month. The food for the first five weeks had consisted entirely of cow's milk. Then Savory and Moore's food had been added, and from the third month onwards oat-flour porridge. The child remained fairly healthy till she was eight months old, but from that time she had ceased to thrive and had been getting progressively paler and weaker. Growth seemed at a standstill and the abdomen had been increasing in size. She had been taking her food fairly well at this time, and only during the last two months had there been any vomiting, and this not oftener than three times a week. On one occasion there was some clotted blood in the vomited matter and on another occasion there was a not very copious bleeding from the nose. There had been a good deal of sweating about the head, especially at night, for some months past. On examination the child was seen to be undersized for her age. The skin and mucous membranes were exceeding pale. The anterior fontanelle was wide open and there was slight enlargement of the ends of the long bones, but little other rachitic deformity. There were five teeth in all, one of which was considerably decayed. The abdomen was protuberant. The liver was palpable below the costal margin, while the spleen extended almost to the crest of the ilium and forwards to within one and a half inches of the umbilicus. The heart and lungs were normal. The examination of the blood was as follows:—May 3rd, 1905, red corpuscles, 3,800,000 per cubic millimetre; and white corpuscles, 12,000 per cubic millimetre. May 19th, red corpuscles, 3,695,000 per cubic millimetre; white corpuscles, 19,000 per cubic millimetre; and hæmoglobin, 37 per cent. A differential count made on May 5th gave these proportions: polynuclears, 64 per cent.; lymphocytes, 24 per cent.; and large hyalines and transitionals, 12 per cent. There were three normoblasts to 100 white cells. Films showed a moderate poikilocytosis but a considerable variation in the size of the red cells,

many being much smaller than normal and some larger, though almost none sufficiently large to be regarded as megalocytes. There were no megaloblasts and no marrow cells found in the films. There was considerable variation in the intensity with which the red cells stained, some being darker and others much fainter than normal. Polychromatophilia was not in evidence.

This child was seen again on July 2nd, 1908. She was then five years old and stout and well nourished, but small for her age, being not so tall as her sister who is two years younger. There were now definite signs of rickets in the presence of marked genu valgum. The mucous membranes were still rather pale. The child, the mother said, was pale and very delicate till three years old. Since then she had been fairly robust and took her food well. The spleen was still much enlarged, extending downwards to ½ inch from the level of the umbilicus. The liver was not palpable. Examination of the blood (July 2nd, 1908) gave the following result:—Red corpuscles, 4,800,000 per cubic millimetre; white corpuscles, 8222 per cubic millimetre; and hæmoglobin, 56 per cent. Films showed a moderate poikilocytosis with some variation in the size and intensity of staining of the individual cells; as a rule they stained much too faintly. There were two normoblasts to 100 white cells. The proportions of the white corpuscles were as follows:—Polynuclear cells, 41.6 per cent.; lymphocytes, 48.8 per cent.; hyaline and transitionals, 8.0 per cent.; eosinophiles, 1.2 per cent.; and marrow cells, 0.4 per cent.

CASE 6.—A girl, aged 14 months, was under treatment in my wards of the Glasgow Royal Infirmary from May 31st to Oct. 14th, 1907. The child was born at full time and the labour was normal in every respect, but she was a very small child at birth. She was fed on the breast for the first fortnight and after that on cow's milk (undiluted), though she seems to have had in addition the "run of the house." There had never been diarrhoea, but the mother said there had been a slight cough almost since birth. It is not certain when the anæmia first appeared, but it had only been noted for about a fortnight before admission to hospital. The father and mother were healthy. There were two other children, the first was stillborn and the other, very small at birth, only lived one week. The mother had had no miscarriages. On admission the child seemed very small for her age; she weighed 15 pounds. The skin and mucous membranes were exceedingly pale, and there was a slight purpuric rash on the back and abdomen. There were definite signs of rickets in the presence of enlargement of the ends of the ribs and of the long bones of the arms and legs. The anterior fontanelle was still unclosed. The abdomen was enlarged and both liver and spleen were palpable below the costal arch, the former extending one inch below the right margin and the spleen as far down as the crest of the ilium. There was no evidence of enlargement of lymphatic glands. There were some small moist râles at the base of the right lung behind, but otherwise the lungs seemed healthy. The heart was normal. Examination of the blood gave the following results:—

	Red corpuscles.	Hæmoglobin.
June 1st, 1907	1,650,000 per c.mm.	25 per cent.
June 18th, "	2,130,000 " "	20 " "
July 4th, "	1,700,000 " "	20 " "
August 6th, "	3,000,000 " "	15 " "
Sept. 4th, "	2,764,000 " "	35 " "
Oct. 3rd, "	3,500,000 " "	40 " "
March 16th, 1908	4,290,000 " "	86 " "

Stained films were examined on June 4th, Sept. 19th, and Oct. 11th. On each occasion there was a moderate poikilocytosis, with considerable variation in the size of the individual cells as well as in their staining reaction; a proportion stained a leaden tint (polychromatophilia). On June 4th there were 10 nucleated red cells to 100 white cells and of the former three might be regarded as megaloblasts. On each of the other two occasions there was 1 normoblast to 100 white corpuscles.

White corpuscles.

June 1st, 1907	14,976 per c.mm.	July 19th, 1907	14,500 per c.mm.
" 2nd "	15,900 "	Aug. 6th "	9,330 "
" 3rd "	18,312 "	Oct. 3rd "	5,000 "
" 18th "	15,900 "	March 16th, 1908	6,400 "
July 4th "	12,792 "		

The proportions of the various white cells were as follows:—

	June 4th.	Sept. 19th.	Oct. 11th.
	Per cent.	Per cent.	Per cent.
Polynuclears	31	32	46
Large hyalines and transitionals...	11	9	7
Small lymphocytes	44	50	44
Marrow cells	8	8	2
Eosinophile cells	5	1	1
Mast cells	1	0	0

During the four and a half months the child was in hospital the improvement, though considerable, was exceedingly slow. The gain in weight in that time was only 3 pounds, but the child looked healthier and the muscles felt firmer, and this in spite of the fact that the feeding was most liberal (three pints of milk and the juice of half a pound of raw beef in the 24 hours). The spleen, too, had lessened considerably in size, reaching on Oct. 14th to rather above the level of the umbilicus. The liver at this time extended about half an inch below the right costal margin.

On dismissing the child from hospital the mother was given very definite instructions as regards the feeding of her child and as to the necessity of keeping her in the open air. And the result was eminently satisfactory, for in the next five months the improvement was very great indeed. The gain in weight at the end of that time was over 14 pounds, and the spleen had lessened so much in size that it extended to only three quarters of an inch below the costal margin; the lower edge of the liver was no longer palpable. The improvement in the condition of the blood has been noted.

CASE 7.—A girl, aged 13 months, was under observation from April 15th to 23rd, 1905, on which latter date she died. The child was fed on the

breast till 10 months old and she seems to have been in excellent health up to the end of her first year. But from that time onwards she became progressively paler and swellings appeared in the groins. Latterly she was so listless she could scarcely hold up her head and she seemed inclined to doze most of the time. There were two other children in the family, a boy, aged seven years, and a girl, aged four years, both alive and well. Another girl, when aged four years, and a boy, aged six months, had died from "measles and whooping-cough." On examination the child was seen to be fairly well nourished, but the pallor of the skin and mucous membranes was extreme. There was slight enlargement of the sterno-mastoid, submaxillary, axillary, and inguinal glands, the largest being of the size of a horsebean. The teeth were in good condition, but there was slight rachitic deformity and the anterior fontanelle was still unclosed. The abdomen was unduly prominent and both liver and spleen were palpable below the costal arch; the spleen extended down as far as the crest of the ilium. The heart and lungs were normal. The blood count was as follows: red corpuscles, 1,600,000 per cubic millimetre; and white corpuscles, 60,000 per cubic millimetre (four days later, 30,600). Of the white corpuscles 34.5 per cent. were polynuclears, 8 per cent. hyaline and transitionals, 44 per cent. lymphocytes, 9.5 per cent. marrow cells, and 4 per cent. eosinophiles. There were two normoblasts and two megalo-blasts to 100 white corpuscles. There was only a slight degree of poikilocytosis and only slight variation in the size of the individual cells. The intensity of the staining, however, varied considerably, some cells staining at the periphery only, while others stained well throughout. In the seven days the child lived while under observa-tion there was nothing fresh to note; the temperature kept normal and the bowels regular, the motions being normal in appearance.

On post-mortem examination the heart was seen to be pale but there was no mottling visible. The epicardium was studded with small ecchymoses. The valve segments were normal. The lungs were pale, excepting the lower lobe of the left lung which was oedematous and congested so that it would not float in water. The thymus was pale and very small. The liver (14½ ounces) was large and pale, with very evident fatty degeneration. The spleen (3½ ounces) was large and of a deep pink colour. The kidneys, suprarenals, and pancreas all seemed healthy. The stomach was of normal size and presented no ab-normality of its mucous membrane. In the intestines Peyer's patches as well as the solitary follicles were swollen, but there was no ulceration. The mesenteric glands were considerably enlarged, but the bronchial and mediastinal glands seemed normal in size. The bone marrow from the shaft of the femur was red in colour. On microscopic examination there was seen to be a very marked fatty degeneration of the liver cells, especially those at the centre of the lobules. There was also a slight increase of the connective tissue of the portal areas but confined to these areas and in no way amounting to a cirrhosis of the liver. There was only a slight hæmo-siderin deposit, confined almost entirely to the portal areas. The spleen showed a slight fibrosis but the Malpighian bodies were about normal in size. The heart and kidneys seemed normal. The mesen-teric glands had the appearances of a general hypertrophy, there being no proper differentiation into cortex and medulla, the sinuses, indeed, being overrun with lymphocytes. The bone marrow of the shaft of the femur was examined in sections and as films. In sections it was seen to be a dense cellular marrow with no appearance of fat cells. It had the characters of the leucoblastic rather than an erythroblastic marrow, the prevailing cell being the finely granular marrow cell, with but few aggregations of the lymphocytes. The nucleated red cells were normo-blasts and the proportion of giant cells was small.

CASES 8 AND 9.—These patients were twin sisters, aged 16 months. The former was admitted to my wards on June 30th, 1908, on account of swelling of the abdomen and general weakness; the latter I asked the mother to bring to the hospital for examination and I found her suffering with much the same symptoms as her sister. These two children were of the same size at birth, only the patient in Case 8 was rather the thinner of the two. For the first 11 months they were fed on the breast only and they seemed to thrive quite well, but at the end of three months it was apparent that the patient in Case 8 was not doing so well and not growing as large as the patient in Case 9. At 11 months the patient in Case 9 was weaned and fed on cow's milk and oatmeal flour. The patient in Case 8 also got the oatmeal but being delicate she was kept on the breast till she was about 15 months old. Neither seems to have got much into the open air but the patient in Case 8 much more than her sister. Both had measles when ten months old but neither seemed to have been specially ill. The mother dates the beginning of the illness in Case 8 from the age of 14 months, when the child became cross, restless, and was sleeping badly. It was at this time that the abdomen was noted to be swollen and it seemed painful on pressure. The swelling of the abdomen had increased since then and the child otherwise had emaciated somewhat. The bowels as a rule had been constipated and the only history of diarrhoea was for a period of about ten days during the convalescence from measles. There had been no diarrhoea in Case 9. The father and mother were alive and well. There were no other children. The mother had had no miscarriages. Both children were thin and small for their age, but Case 8 was distinctly the smaller of the two. She weighed 13½ pounds as against 18 pounds for her sister. In both there were signs of rickets in the presence of enlargement of the ends of the long bones. The anterior fontanelle was not closed in either. The mucous membranes were well coloured in both, and each had several teeth in good state of preservation. The spleen in Case 9 extended to the level of the umbilicus; in Case 8 to one inch below that point. The liver was not enlarged in either. Examination of the blood gave the following results:—

Case 8.		Red		White		Hæmoglobin.
		corpuscles.		corpuscles.		
		Per c.mm.		Per c.mm.		Per cent.
July 3rd, 1908		4,800,000		22,500		85
„ 10th, „		5,100,000		28,125		95
„ 17th, „		4,487,000		30,837		75
„ 24th, „		4,472,000		30,000		70
Case 9.						
July 19th, 1908		4,648,000		28,225		70

Films gave almost exactly the same picture in the two cases. There was but slight poikilocytosis but considerable variation in the size of the red cells, some being distinctly larger than normal and many much

smaller. Certain cells showed polychromatophilic staining, and in Case 9 pigmentary degeneration was seen in some of the cells. There were from two to three nucleated reds to 100 white corpuscles, and at least half of these nucleated reds were megaloblasts. The following were the proportions of the various white cells:—

Case 8.		Poly-nuclears.		Lympho-cytes.		Hyaline and transi-tionals.		Marrow cells.		Eosino-philés.	
		Per cent.		Per cent.		Per cent.		Per cent.		Per cent.	
July 3rd		35.0		49.0		7.5		5.5		3.0	
„ 17th		38.6		41.5		5.3		7.4		7.2	
Case 9.											
July 19th		22.0		65.0		4.0		5.0		5.0	

CASE 10.—A girl, aged seven weeks, was under observation for only six days, when she died on April 1st, 1907. She was a twin child and at birth seemed quite healthy but since then she had never thriven, for while to begin with she was the larger of the twins, on admission to hospital she weighed 4½ pounds, only half as much as the other child. She had been fed on breast milk and had always taken it well. She at no time had been unduly fretful. When four weeks old the mother noticed that the abdomen was considerably swollen and she thought it was due to flatulence. The bowels had always been rather loose but the motions invariably contained bile. There were six other children in the family. The first was stillborn at full time. The second was alive and very healthy. The third died, aged one year and ten months, from convulsions. The fourth was stillborn at seven months. The fifth died from congestion of the lungs when eight months old. The sixth also died from congestion of the lungs when a year old. The father was alcoholic and the mother had had a severe hæmorrhage during the third month of this last pregnancy, but there was nothing else of importance in the family history. On examining the child it was seen to be exceedingly small, puny, and ill nourished. The skin of the face and of the body generally was loose and wrinkled and had some-thing of the parchment-like appearance. There were no abrasions of the skin or mucous membrane visible, and no desquamation unless a little over the scalp. The abdomen was markedly distended. Both spleen and liver were greatly enlarged, the former extending to the crest of the ilium and the latter to an inch below the umbilicus. The heart and lungs seemed normal and there was no sign of hæmorrhage into the skin or from any mucous surface. The blood count was as follows: red corpuscles, 2,088,000 per cubic millimetre; white corpuscles (including nucleated reds), 95,144 per cubic millimetre; and hæmoglobin, 18 per cent. On examining films there were seen to be a moderate poikilocytosis and considerable variation in the size of the different red cells. A propor-tion were larger than normal—some as large as megalocytes, but more were nearer the size of the microcyte. There was also a large number of corpuscles showing all shades of polychromatophilic staining, and in some others there were spots of pigmentary degeneration. The out-standing feature of the film, however, was the very large number of nucleated red cells, these forming 39 per cent. of all the nucleated cells present. About 5 per cent. of these nucleated red corpuscles might be regarded as megaloblasts. The following were the proportions of the various white corpuscles: polynuclear cells, 14 per cent.; small lympho-cytes, 66 per cent.; hyaline and transitional cells, 11 per cent.; marrow cells, 6 per cent.; eosinophile cells, 2 per cent.; and mast cells, 1 per cent. There were 62 nucleated red cells to 100 white cells. There was nothing of special interest to note during the six days the child lived after admission to the ward. The bowels moved regularly and the motions were normal in appearance. The milk, however, was not well taken and death was apparently due to inanition.

At the post-mortem examination there was no definite evidence of rickets, syphilis, or tuberculosis. The heart was pale but otherwise normal. In the right lung there was an area of broncho-pneumonia along its posterior border and similar but smaller areas were seen scattered through the left lung. The liver (5½ ounces) was large, firm, of a yellowish colour, with patchy areas of congestion. The spleen (4½ ounces) was large, firm, and congested. The kidneys, adrenals, pancreas, and stomach were all pale but otherwise seemed healthy. There was no notable enlargement of any of the lymphatic glands. On microscopic examination the liver showed a fairly marked fatty degeneration of its parenchyma. There was a large excess of leucocytes in the blood and the capillaries were distended with blood. Scattered over the section were groups of leucocytes the large majority of which were mononuclear. The appearance generally was somewhat suggestive of what a syphilitic monocellular cirrhosis might be in an early stage of that disease, but the spirochæta pallida was not obtained on appropriate staining. There was no hæmosiderin reaction. In the spleen the pulp was congested and many of the sinuses were packed with white corpuscles. There was possibly a slight fibrosis and there seemed to be multiplication of the endothelial cells. The Malpighian bodies were not increased in size. The kidneys showed the same infiltration with round cells as the liver. The cells were mostly lymphocytes but polynuclear and eosinophile cells were also present. The infiltration was chiefly round the vessels and was very marked in places. The lymphatic glands were not examined microscopically. The lungs presented the characteristic appearance of broncho-pneumonia. The marrow was definitely leucoblastic, more so than in Cases 3, 4, and 7. It was not, however, a lymphoid marrow though it did contain a considerable proportion of lymphocytes. A number of the nucleated red corpuscles were megaloblasts and many of these, as well as of the normoblasts, showed a pigmentary degeneration of their plasma.

Of these 10 cases the first two may be regarded as examples of the first clinical type to which I have already referred. Case 1 is now 22 years of age and in good health, but from the age of four till he was 19 he had been always more or less anæmic and never in robust health, as shown by the number of times he was in hospital. The blood in this case was not examined in infancy, but from the sixth year onwards till three years ago there was a leucopenia every count that was made; now the blood is normal but the enlargement of the spleen persists. Case 2 also had a

leucopenia when under observation in 1905, and even now, when there is no anæmia and the child is apparently in the best of health, a leucopenia still exists. It may be here noted in passing that of the other cases in the one (Case 6) that made the best recovery the leucocytosis of 18,000 ultimately gave place to a leucopenia which, as may be seen, lasted for at least five months. In Case 5, which is now practically well, the leucocytes are about normal.

The second clinical type is represented by Cases 3, 4, 5, and 6—that is, cases with a leucocytosis of from 11,000 to 19,000; and the third type will include Cases 7, 8, 9, and possibly 10, all with a leucocytosis of over 25,000. This, however, is clearly a most unsatisfactory manner of grouping the cases, for the blood picture in Case 6 is much closer to that of Case 7 than to that of Cases 3 and 4, and yet for lack of a few thousand leucocytes it falls to be included with the latter. But at the same time one must admit that the blood of Cases 6, 7, 8, and 9 presents some notable differences from that of Cases 3, 4, and 5, and particularly in regard to the number of myelocytes and megaloblasts that were present in the former. Whether this is a point of any consequence may be considered later. As to Cases 8 and 9 it may be objected that they should not be included in this series, as examination of their blood shows no anæmia in either of them. But, on the other hand, the general aspect of the children, the enlarged spleen, the leucocytosis of 30,000, with the proportion of myelocytes and megaloblasts, associate them so closely with Cases 6 and 7 that it seemed of interest to add them to the list. They at least show that this form of leucocytosis is not dependent on the anæmia and that it may be produced by causes other than those that determine the diminution of red corpuscles.

The diagnosis in Case 10 has been a matter of some difficulty. From the examination of the blood while the child was alive I was disposed to regard it as a case of von Jaksch's splenic anæmia, with a very primitive blood, depending in part on the age of the child, for she was only seven weeks old. But the infiltration of mononuclear cells into the liver and kidneys which was met with on microscopic examination suggested that the case should really be regarded as an early stage of lymphatic leukaemia. These infiltrations, especially in the liver, certainly presented much the appearance that one sees in congenital syphilitic cirrhosis of the liver, and hence we had to consider the possibility of the case being one of anæmia associated with, and perhaps dependent on, the syphilitic toxin. Staining for the spirochæta pallida has, however, been negative, and so I am disposed to regard the case as one of leukaemia. Professor Muir has been good enough to examine the sections in this case and he is of opinion that, from the histological point of view, it is really a leukaemia, in spite of the absence of any considerable lymphoid infiltration of the bone marrow or enlargement of lymphatic glands. But even if this Case 10 be a lymphatic leukaemia it is of interest to add it to this series, for clinically, at least, it had much the same characters as Cases 7, 8, and 9; and apart from the very large numbers of nucleated red corpuscles the proportions of the other nucleated cells were much the same as in these three cases. I do not wish by this to suggest that lymphatic leukaemia and von Jaksch's anæmia are but different phases of the same disease but simply to draw attention to a clinical resemblance between them as shown by the above cases.

In considering the etiology of infantile splenic anæmia the only aid we can extract from these ten cases is of an entirely negative kind, for there seems to be no constant factor in any number of them to which one could attribute the anæmia and splenic enlargement. In only one (Case 8) was there a history of diarrhoea, and this of not more than ten days' duration. A large proportion of the cases were fed on the breast, so that artificial feeding cannot be blamed for the illness in any considerable number of them. In Cases 1, 8, and 9 there was a history of measles, but with Case 1 the "lump" in the abdomen was noted before the onset of this fever. In Cases 8 and 9 there was apparently a family tendency to splenomegaly, as these children were twin sisters and both affected; also with Case 2 there was a history of another child having died with an enlarged spleen. In Case 3 the child was small and greatly emaciated and the anæmia was probably just a symptom of the marasmus, brought on possibly by unsuitable or insufficient food. In Cases 4, 5, 6,

and 7 there was no obvious cause for the anæmia unless rickets, signs of which were present in all of them as well as in Cases 3, 8, and 9. But in spite of the rickets there was no anæmia in Cases 8 and 9, and Dr. Leonard Findlay tells me that he has just completed an inquiry into the state of the blood in 30 cases of uncomplicated rickets and he finds that the number of red corpuscles and the percentage of hæmoglobin are distinctly above rather than below the reputed normal of infancy and as verified by him in 12 control examinations that he has made in healthy children. (The leucocytosis in the 30 rachitic cases ranged from 7000 to 18,000.) Rickets may therefore predispose to anæmia in the same way that it seems to predispose to a catarrhal condition of mucous membranes, but it cannot be held to be the exciting cause of the anæmia. We may say, then, that from the clinical point of view at least, these cases, as well as many others, support the view that there is no specific and constant factor at work in the production of infantile splenic anæmia. Neither does the histological examination of the tissues in the cases that died help us much as regards the etiology or pathogenesis of the anæmia. In neither liver nor spleen was there any change in any way distinctive, at least in Cases 3, 4, and 6, and in particular there was no evidence of any considerable deposit of iron pigment in either organ. The lymphatic glands showed signs of an undue activity but this may be met with in other conditions. The bone marrow was exceedingly cellular and perhaps more leucoblastic than normal, but that is as one expects it in any case with a leucocytosis. Finally, there was nothing to distinguish the tissues from cases (3 and 4) with a moderate leucocytosis from those of Case 7 with a leucocytosis of 60,000.

What, then, is the cause of infantile splenic anæmia? What is its pathogenesis? Is it a secondary anæmia, secondary to a variety of causes, or is it, or any of its varieties, a primary anæmia due to some specific toxin? In trying to answer these questions we have unfortunately little to go upon. If it is a primary anæmia we have no direct proof of it, for the toxin has not been isolated, neither has the disease been produced experimentally. Post-mortem examination shows no signs of the action of a specific toxin. The plea for the disease being a primary anæmia is therefore based on clinical grounds, on the clinical picture the cases present to us. Now in this picture the main features of a well-marked case are, in addition to the anæmia, the enlarged spleen, the considerable leucocytosis with its increased variety of leucocytes, and the presence of a notable proportion of normoblasts and megaloblasts. But are we justified in accepting such a group of signs as evidence that the anæmia is a primary anæmia? The enlargement of the spleen is a sign common to many diseases; it is met with in most of the infections, particularly those of any considerable duration. It depends chiefly upon a hyperplasia of the splenic pulp, but is also in part due to the accumulation of effete elements, such as bacteria, red corpuscles, and certain white corpuscles, which have been withdrawn from the circulation to the spleen where they undergo disintegration. The spleen in children seems to enlarge more readily than in the adult, doubtless because the conditions which determine the enlargement are more commonly met with in the former than with the latter. If, then, the enlarged spleen in infantile splenic anæmia has any special significance, it may be that there is an undue amount of foreign material circulating in the blood and that the spleen has hypertrophied in its efforts to deal with it.

It has been suggested that the enlarged spleen manufactures a special toxin which entering the circulation is responsible for the anæmia. We have, however, just seen that the enlarged spleen is more likely to be secondary to the anæmia than the cause of it, but even if a toxin were produced in the spleen as the result of disintegration of red and white corpuscles and other effete substances, one would expect such a toxin to be discharged into the portal circulation and that its toxic effect would first be felt by the liver and the blood corpuscles in the portal area. We have seen, however, that there is no evidence of an increased destruction of red corpuscles in the portal circulation and no evidence of the action of a toxin on the liver. If, then, the spleen does, as has been claimed for it, produce such poisonous substances we have no proof of it.

The considerable leucocytosis met with in the majority of the cases of infantile splenic anæmia is another sign to which

much importance has been attached. But on looking over the proportions of the various kinds of leucocytes in cases such as 6, 7, 8, and 9, where there was a leucocytosis of 20,000 or more, one is struck with how nearly these percentages correspond to the normal, always remembering that in the infant the proportions of the different types of leucocyte differ somewhat from those in the adult.¹ The leucocytosis then in these cases affects all the different types of white corpuscles more or less uniformly, and it must mean that the various tissues where these corpuscles are produced are also more or less uniformly affected. It is questionable if chemiotaxis has much to do with the production of this sort of leucocytosis. We know that the polynuclear cells will increase greatly in number in response to certain chemiotaxic substances, but the same cannot be said for the lymphocyte. Ehrlich holds that a lymphocytosis is produced by increased flow of lymph through lymphatic glands and other lymphoid structures, and possibly irritants in the lymph increase this flow. Neither does the marrow cell, as far as is known, enter the blood in response to any phagocytic or chemiotaxic demand; indeed, its association in the same cases with normoblasts and megaloblasts looks as if the passage into the circulation of all these cells were a passive rather than an active one. The appearance of nucleated red corpuscles in the blood in cases such as Cases 8 and 9 cannot well be due to an increased demand for red corpuscles, seeing that the blood count shows the red corpuscles to be above rather than below normal. If we say, then, that there is a disturbance of the blood-forming organs so that an increased number of leucocytes (and other cells) pass into the circulation, we go as far as we may in dealing with the leucocytosis of infantile splenic anæmia. The blood-forming organs in infants seem to be readily disturbed by disease, and hence we may meet with myelocytes and nucleated red corpuscles in their blood in a variety of morbid conditions. But why there should be a leucocytosis in certain of these anæmias and a leucopenia in others is not the least obvious. It may be a coincidence, but in Cases 1 and 2, the only ones without leucocytosis, there was no appearance of rickets, while in all the others (excepting Case 10) signs of rickets were undoubtedly present. Now, in rickets there is always, to begin with at least, a hyperæmia at the line of ossification. Is it possible that this hyperæmia also involves the marrow of the bone and so accounts for the "disturbance" in this tissue above referred to?

This reading of the signs of infantile anæmia that I have given leads me, then, to the conclusion that the condition is not a primary but rather a secondary anæmia, and in the main due to a functional disorder of the blood-forming organs. Such a disorder, we may say, depends chiefly on a debility of these hæmopoietic tissues, a debility perhaps inherited but depending (in common with other tissues) for its production chiefly on such faults as lack of suitable food and fresh air, but at times determined by the toxins of measles, syphilis, and other infections. If a number of children are ill-fed all their tissues suffer more or less, but usually one tissue more than another, the tissue most affected varying in different children. Hence with some children the debility affects chiefly the blood-forming organs. The same with adults. With some insufficient food or fresh air at once determines anæmia, while with others it is another disorder that shows itself. That these infantile anæmias are of this nature is shown by the results of treatment. Give the infant suitable food and lots of fresh air and, provided no complication such as pneumonia occurs, a cure may be confidently looked for in time. The cure may be slow, but then it is to be remembered that one is perhaps treating a child half its normal weight and with its tissues wasted in a corresponding manner; it cannot but take time to effect a cure in such cases. In speaking of infantile splenic anæmia in this way one includes the three types already referred to—namely (1) the cases with leucopenia; (2) those with moderate leucocytosis; and (3) cases grouped as von Jaksch's splenic anæmia; and one regards them all as secondary anæmias and as simply different phases of the same disease.

Glasgow.

¹ In infancy the following may be taken as the proportions of the different kinds of white corpuscles met with normally: polynuclears, 28 to 40 per cent.; lymphocytes, 50 to 70 per cent.; large mononuclears and transitionals, 6 to 14 per cent.; and eosinophiles, 0.5 to 10 per cent.

Clinical Notes : MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

A RUPTURED TUBAL GESTATION WITH TWIN FŒTUSES.

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F.R.C.S. EDIN.

A WOMAN, aged 21 years, was seen by a medical practitioner as she complained of abdominal pain and a feeling of faintness. She was told that she was anæmic and an iron tonic prescribed for her. She stayed in bed during the next four days but on the evening of the fourth she came downstairs and "dragged about" her work. Two days after this partial resumption of her duties she went out in the evening for a short walk but on returning home was attacked by severe abdominal pain and fainted while going upstairs, falling and cutting her forehead badly. She remained in bed all the next day and on the next—i.e., some nine days after the original attack—she became so much worse that I was asked to see her. I found her in bed moaning and restless and it was at once obvious that some very grave peritoneal disaster had overtaken her. The patient was absolutely blanched, even her lips and gums were colourless. The flickering pulse was almost uncountable (I made it somewhere between 160 and 170 to the minute), the abdomen was distended, dull all over to percussion, and so tender that one could make nothing out bimanually. There had been a little sickness but there was no history of digestive disturbances suggesting the probability of her symptoms being due to a perforated gastric ulcer, nor was there any balance of evidence in favour of appendicular trouble. There was, however, a history of some three or four months' amenorrhœa. I asked her if she had any reason to suspect that she might be pregnant and after some hesitation she admitted that she believed herself to be three or four months in the family way. I therefore looked upon the case as one of ruptured ectopic gestation and sent for assistance. On the arrival of the operating surgeon (I do not wish to give names) he opened the abdomen which was full of blood and ligatured and removed a ruptured tubal sac with twin fœtuses attached. The patient was infused with saline solution after the operation and there was some little sickness for a day or two but her recovery was uninterrupted.

Taking the symptoms in conjunction with the undoubted existence of pregnancy I think that I was justified in diagnosing a ruptured extra-uterine conception, though there had been none of the usual irregular hæmorrhages, no colicky pains, and no extrusion—neither at the time of the rupture nor since—of anything like a decidual membrane. I think that probably the course of the case was that there was a partial rupture with a small opening which was responsible for the initial symptoms, but with rest in bed this extremely limited rent became temporarily sealed, but that her resumption of work and the exertion of walking precipitated the complete rupture of the sac, and that the fortunate issue of the case was due to the promptitude with which surgical measures were adopted.

AN UNUSUAL SEQUEL TO PYLORECTOMY.

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THE patient, a man, aged 59 years, was admitted to the Sheffield Royal Infirmary on July 23rd, 1908. His history pointed to malignant disease of the stomach and an exploratory laparotomy revealed the presence of a cancerous mass occupying the pyloric end of the stomach and spreading along its lesser curvature almost to the œsophageal opening. The growth was excised in the usual way, an anastomosis being effected between the first part of the jejunum and what was left of the stomach.

The patient made a good recovery from the operation and