typhoid arthritis and typhoid arthritis proper, as there were no other pyæmic symptoms present in the case, and there was no evidence of osteitis preceding or accompanying the arthritis. Keen, who introduced it, has used the term rheumatic typhoid arthritis rather vaguely. There is, so far as we know, nothing to prevent a patient suffering from enteric fever suffering from rheumatism also; but considering that we now recognise scarlatinal rheumatism as having nothing to do with true rheumatism, we should be very careful in calling cases which present a passing resemblance to rheumatism, "rheumatic typhoid arthritis." This is well seen by the fact that Keen and Dreschfeld in referring to Freyhaus's³ case consider it to be one of rheumatic typhoid arthritis and typhoid arthritis proper, respectively, and I should be slow to call this case one of rheumatic typhoid arthritis unless there were more than mere pain and swelling of the joint to justify it. Against the rheumatic nature of this joint affection there are the following facts: the boy had not suffered from any manifestation of rheumatism previously and none of his near relations had so suffered; the joint affection was almost wholly confined to one joint; none of the commoner complications of rheumatism, such as endocarditis, were present, and lastly the affection became suppurative. It might be argued that the affection became suppurative on account of the previous aspiration, but the fluid in the joint did not become purulent until almost a fortnight after the first aspiration, and every precaution was taken in aspirating.

The diagnosis of typhoid arthritis proper thus presents itself, and despite the later bacteriological examination this diagnosis must be held. The joint which is most frequently attacked in typhoid arthritis is the hip, and after this the other joints of the lower limb. In Keen's 84 cases the lower extremity was affected in 70, the upper in 17, and both extremities in 7 cases. The most frequent result of typhoid arthritis is spontaneous dislocation owing to the gradual lengthening of the ligaments through the distension of the joint. It was to prevent this result that the joint was at first aspirated. Suppurative arthritis is a rare result.

The bacteriological examinations in this case are of some interest. All our present information and ideas of enteric fever would point to the bacillus typhosus as being the cause of typhoid arthritis proper. It is quite in accordance, however, with the published records of bacteriological examinations to find that in this case at one time a negative result was got, while at another pyogenic organisms alone were found. Too few of these cases have been examined however, to enable us to arrive at any result.

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ON ANÆMIA IN TYPHOID FEVER.

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THE blood in typhoid fever has been studied by many observers and some of its interesting characteristics are now regarded as definite features of the disease. Thus it is well known that the anæmia of typhoid fever differs from the majority of secondary anæmias in the fact that while it presents in uncomplicated cases no evidence of leucocytosis there is generally a considerable increase in the proportion of large mononuclear leucocytes.¹

It is also agreed amongst bacteriologists that in typhoid fever there occurs a great increase in the bacterial fermentation of the intestinal contents. In the anæmia which occurs in a fair proportion of cases of typhoid fever we have, therefore, an opportunity of investigating the relation between the bacterial fermentation with absorption of toxins, and the modifications which occur in the blood of these cases. The bearing of this investigation on the various well-known theories of the primary anæmias need not be enlarged upon here.²

The following case of typhoid fever illustrates these peculiarities in a striking manner, and a consideration of the

observations made in this case seems to throw light on some of the interesting features of the disease.

History.—S. T., aged 30, a cattle dealer, was admitted to the fever wards of the Royal Victoria Hospital, Belfast, under the care of Professor Lindsay. He had a history of previous good health, and was described as a "full-blooded man." His only illnesses were an attack of acute pneumonia and some rheumatic attacks. Before admission he had some diarrhoea and epistaxis, and had been ill for about ten days. The fever ran a rather protracted course, and he was altogether 87 days in hospital. A few of the more important details copied from the clinical notes will be sufficient for our present purpose. On admission (Nov. 26th, 1900) having been ill about ten days, his pulse was 98, and his temperature 102°. He had slight cough, diarrhoea, with fullness of the abdomen, but no pain or gurgling in the right iliac fossa. There were a few spots, and the tongue was furred. The heart sounds were normal; there was some bronchitis; the spleen was enlarged, and the patient was somewhat incoherent in his answers to questions. On November 29th he had epistaxis. On December 8th there was slight hæmorrhage from the bowels. On December 12th there was hæmorrhage from the bowels fairly large in amount. On December 23rd phlebitis was discovered. The temperature for a few days before this had been about 101° at night. It now gradually rose to 104° (this rise was no doubt due to the phlebitis). On January 1st, 1901, the temperature was normal, and remained so for four or five days, and then began to rise again. On January 20th abscesses were discovered about the rectum, and incised. The temperature previous to this treatment was 103°. On January 27th the patient had a rigor; after this the temperature remained normal for some time, with very slight rises. He was noted to have an anæmic appearance. On February 16th small abscesses were found in the axilla, and opened. On February 20th the patient left the hospital. The temperature was subnormal. He was still somewhat anæmic looking, but otherwise was much improved.

This patient had thus a rather protracted attack of typhoid fever complicated by some epistaxis and hæmorrhage from the bowels, and prolonged by a phlebitis and abscesses about the anus and in the axilla. The patient during the illness became considerably anæmic.

For the sake of clearness, the record of this case may be divided into six periods:

No. I.—Early Stage.

November 27th, 1900. Ill about twelve days. Red cells, 4,930,000; white cells, 5,300; hæmoglobin, 88 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	72.4
Small lymphocytes	20.0
Large mononuclear leucocytes	6.2
Transitional leucocytes	1.4

This was practically a normal blood, except that the white cells were somewhat diminished in number, and the large mononuclear leucocytes slightly increased.

No. II.—Anæmia Commencing; No Inflammatory Complications.

December 6th, 1900. Red cells, 3,000,000; white cells, 4,200; hæmoglobin, 67 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	51.2
Small lymphocytes	37.6
Large mononuclear leucocytes	8.4
Transitional leucocytes	2.8

The hæmoglobin and red blood corpuscles were now considerably diminished. In the differential count the lymphocytes and mononuclear leucocytes were increased in percentage.

No. III.—Anæmia persisting with Phlebitis.

December 20th, 1900. Red cells, 2,900,000; white cells, 4,600; hæmoglobin, 65 per cent.

Differential Count:

	Per Cent.
Polymorphonuclear leucocytes	66.0
Small lymphocytes	30.6
Large mononuclear leucocytes	3.4
Transitional leucocytes	0.2

Although the hæmoglobin was somewhat lower than in former observations, the leucocytes had risen somewhat, while the percentage of polymorphonuclear leucocytes had considerably increased. I was at first at a loss to understand the meaning of this alteration in the blood picture, as it was the reverse of what had been expected until Dr. Mandale Byers, the house-physician, on investigating this anomaly, discovered that the patient had developed a phlebitis of the left femoral vein. This explained also the rise of temperature which occurred at this time. It is, however, to be noted here that the number of leucocytes per c.mm., although greater than in the former count, was still much less than is usually found in an inflammatory condition. The increase, however, took the form which is observed in ordinary inflammation.

No. IV.—Anæmia Increasing; Phlebitis Disappearing.

January 2nd, 1901. Red cells, 2,800,000; white cells, 3,500; hæmoglobin, 60 per cent.

Differential count:

	Per cent.
Polymorphonuclear leucocytes	51.0
Small lymphocytes	40.0
Large mononuclear leucocytes	8.5
Eosinophile leucocytes	0.5

Here the hæmoglobin is still lower, while the white cells have somewhat decreased again. The percentage of polymorphonuclear leucocytes

has again begun to fall and the percentage of lymphocytes to rise. The effect of the inflammatory complication is evidently beginning to pass off. The temperature is now normal.

No. V.—Patient Profoundly Anæmic: Inflammation and Abscess Formation Present.

January 15th, 1901. Red cells, 3,070,000; white cells, 4,900; hæmoglobin, 54 per cent.

Differential count:

	Per cent.
Polymorphonuclear leucocytes ...	66.8
Small lymphocytes ...	25.8
Large mononuclear leucocytes ...	5.6
Transitional leucocytes ...	0.2
Eosinophile leucocytes ...	1.6

Here the hæmoglobin has reached its lowest point, and the enumeration of red cells shows that the blood is assuming a decidedly chlorotic type. The percentage of hæmoglobin per c.mm. is 54 per cent., that is, considerably lower than the percentage of red blood corpuscles, 61 per cent. The number of white cells, however, is somewhat higher than in the former case, and the differential count shows a greatly increased proportion of polymorphonuclear leucocytes. Here again I obtained a result which I did not expect, and as the temperature had again risen it was evident that inflammatory mischief was at work. Dr. Mandale Byers subsequently discovered some abscesses about the anus, which evidently accounted for the unexpected blood picture as well as the rise of temperature.

No. VI.—Patient Convalescent: Inflammation Subsiding.

February 19th, 1901. Red cells, 3,870,000; white cells, 3,000; hæmoglobin, 66 per cent.

Differential count:

	Per Cent.
Polymorphonuclear leucocytes ...	50.4
Small lymphocytes ...	37.0
Large mononuclear leucocytes ...	9.0
Transitional leucocytes ...	3.2
Eosinophile leucocytes ...	0.4

This determination was made the day before the patient left the hospital, and it bears a remarkable resemblance to the second determination. The only difference is that the blood has become somewhat more chlorotic in type.

In order to understand the variations in hæmoglobin, and the meaning to be attached to the variations in the numbers of the different forms of white corpuscles, it was necessary to obtain an estimate of the volume of this patient's blood and of the total amount of hæmoglobin. Dr. Lorrain Smith carried out this estimation by the CO method, with the following result:

Volume ...	9 c.cm. per 100 grams body weight
Oxygen capacity ...	1.1 c.cm. per 100 grams body weight
This result may be compared with the normal:	
Volume ...	4.6 c.cm. per 100 grams body weight
Oxygen capacity ...	0.83 c.cm. per 100 grams body weight

It is to be remembered that this patient was described as a "full-blooded" man when in health, and he has therefore a somewhat large amount of hæmoglobin per unit weight. It is clear from this observation that in this case the toxins of the typhoid bacillus had no hæmolytic power, and that the loss of hæmoglobin per c.mm. was merely the effect of an increase in the plasma. This conclusion will become clear also from other evidence.

Before discussing the observations in detail, however, it may be noted that the anæmia of typhoid fever tends to become of the chlorotic type. For instance, in the last determination the red corpuscles were 3,870,000 per c.mm. (or 77 per cent. of the normal number), while the hæmoglobin was 66 per cent. The percentage of lymphocytes was also considerably higher than normal, while the total volume of blood was nearly doubled. This, as Dr. Lorrain Smith³ has shown, is the typical blood picture of chlorosis. It is interesting in this relation to note the tendency which has been observed for cases of typhoid anæmia to assume such a character that they are afterwards described as chlorosis following typhoid fever. I was asked by Professor Lindsay to examine the blood of a female patient under his charge in the hospital.

The following is the result:

February 28th, 1901. M. M., aged 18: Red cells, 300,000; white cells, 3,200; hæmoglobin, 37 per cent.

Differential count:

	Per cent.
Polymorphonuclear leucocytes ...	55.8
Small leucocytes ...	33.8
Large mononuclear leucocytes ...	3.6
Transitional leucocytes ...	3.0
Eosinophile leucocytes ...	3.8

The examination of the volume and total hæmoglobin by the CO method (this determination was made some time after the above observations were taken when the patient was considerably improved by treatment)

gave the following result showing that this case was one of typical chlorosis:

Oxygen capacity per 100 grams body weight...	0.86 c.cm.
Volume of blood per 100 grams body weight...	7.7 c.cm.

The interesting point is that about a year before this patient had typhoid fever and since then she had felt weak and suffered from breathlessness. The typhoid fever here seemed to have been followed by the chlorotic condition. Ollivier⁴ mentions a similar case. This group of cases is of interest, not merely in regard to the pathology of typhoid fever, but also in so far as it furnishes a suggestion as to the etiology of this chlorotic condition of the blood.

Comparative Table of Blood Determinations.

Stage.	Leucocytes per c.mm.	Polymorphonuclears per c.mm.	Lymphocytes per c.mm.	*Percentage of Large Mononuclear Leucocytes.	Percentage of Hæmoglobin.
Early stage ...	5,330	3,817	1,463	7.6 %	88 %
Anæmia commencing ...	3,200	1,636	1,561	11.2 %	67 %
Anæmia and phlebitis ...	4,600	3,036	1,564	3.6 %	65 %
Anæmia and phlebitis disappearing ...	3,500	1,785	1,697	8.5 %	60 %
Profound anæmia; inflammatory complications...	4,900	3,273	1,548	5.8 %	54 %
Patient convalescent; inflammation subsiding...	3,000	1,512	1,476	12.2 %	66 %

* The large mononuclear and transitional leucocytes are here estimated together.

It is to be noted here:

1. That the leucocytes per c.mm. are always below the normal, and show a gradual decrease in number, except in the two instances where an inflammatory complication intervened. That the inflammatory increase in leucocytes is not by any means so pronounced as we find even in slight cases of suppuration, for in such cases the leucocytes rise to 10,000 or 12,000 per c.mm. This interesting feature of the leucocytosis in the complications of typhoid fever has been pointed out by other observers, although no adequate explanation has as yet been offered.⁵ It is to be noted, however, that, although there is no very great increase in the number of the leucocytes per c.mm. in the inflammatory complications occurring in this case, yet the differential count is very materially altered. (For this reason it will be seen that the differential count is of much greater importance as a means of diagnosing the inflammatory complications of typhoid fever than the mere enumeration of the leucocytes.) This peculiarity of the blood of typhoid fever seems best explained by the hypothesis that the anæmia is the result of dilution of the blood with excess of plasma. Taking this into consideration, it seems evident that the leucocytes in typhoid fever react normally to inflammatory toxins, but that this reaction is to a large extent masked by the dilution of the blood.

2. The polymorphonuclear leucocytes in this table show a gradual decrease except where the inflammatory complications intervened. The fall of the polymorphonuclear leucocytes is roughly proportional to the decrease in hæmoglobin. Now, unless there is hæmolytic, the percentage of hæmoglobin may be taken as an index of the dilution of the blood. In this case there was no hæmolytic. If we take this fact into account, we see that the apparent fall of polymorphonuclear leucocytes was due to the passage into the blood of an excessive quantity of fluid which did not contain these corpuscles. Thus, leaving out the inflammatory periods, and allowing for the dilution, we find that the absolute number of polymorphonuclears remains, roughly speaking, stationary throughout.

3. The lymphocytes, taking all varieties together, come out practically the same in all cases. Their proportional increase is therefore due to the gradual "apparent" decrease of the polymorphonuclear elements. If we suppose the blood in this case diluted according to the percentage of hæmoglobin, their absolute numbers must have increased almost identically in proportion to the dilution. Ehrlich⁶ believes that the lymphocytes in the blood come from the lymph glands and lymphatic apparatus, and are increased only by an increased flow of lymph more or less mechanically. We would, therefore, from the results of these determinations infer that as

the lymph flows through the lymphatic apparatus it washes out, so to speak, 1,500 lymphocytes per c.mm. of lymph, so that in diluted blood the number of lymphocytes per c.mm. remains constant, while their proportion to the polymorphonuclears will rise. We have often determined this percentage increase of lymphocytes in cases of chlorosis, and found that it corresponds practically to the increase of volume as determined by the CO method. (It is not to be understood that the number of lymphocytes is constant in different individuals.)

4. The mononuclear leucocytes (Ehrlich)⁷ show a considerable increase in this case following the rule in typhoid fever, as has been pointed out by numerous observers.⁸ According to the determinations given in the table, the percentage of these elements increases with the percentage of ordinary lymphocytes and decreases with a decrease in the percentage of typical lymphocytes. This is a strong physiological proof that their affinities are with the ordinary lymphocytes, and not with the polymorphonuclear leucocytes, and from their staining reactions I do not think that any such hard-and-fast line of distinction as Ehrlich attempts to draw can be drawn between these cells and the typical lymphocytes.

5. In the foregoing differential counts the eosinophiles occur only in very small numbers.

From these observations it seems clear that during the course of this case there was considerable dilution of the blood, and, indeed, this seems the only possible explanation of these determinations. The view put forward by Ehrlich and others to account for the "apparent" decrease in the polymorphonuclear elements in typhoid fever is that of "negative chemiotaxis."⁹ Against this view I would urge the following objections:

1. The decrease of polymorphonuclear elements in typhoid bears no relation to the intensity of the toxæmia of the disease. Thus we have found patients moribund from typhoid toxæmia with their polymorphonuclear leucocytes scarcely diminished (their hæmoglobin was not much decreased); whereas if "negative chemiotaxis" were the true explanation, we should expect to get them reduced to a minimum in such cases.

2. The decrease in polymorphonuclear elements is proportional to the decrease in hæmoglobin when the number varies inversely as the amount of dilution. It has often been noticed that the so-called lymphocytosis, or proportional increase of lymphocytes, is more marked during the decline of the fever—that is, when the anæmia is more pronounced.

3. It may further be noted that in typhoid generally, although there seem to be exceptional cases, the eosinophiles are also decreased.¹⁰ This also corresponds to a dilution of the blood, and is against the theory of negative chemiotaxis, as Ehrlich¹¹ has shown that substances which repel the polymorphonuclear elements usually act positively on the eosinophile.

This tendency to dilution of the blood in typhoid fever may be due to increased activity in the lymphatic apparatus of the alimentary canal. (The lymphocytosis which occurs as the result of digestion may have a similar explanation. In many cases of typhoid fever this tendency to anæmia is not by any means marked, but it may be noted that diarrhoea and sweating, etc., often serve to counteract this process by concentrating the blood. Again, it is only in exceptional cases that a profound anæmia follows typhoid, and perhaps this may be due to the fact that typhoid is a disease of healthy adult life, and that therefore the compensatory power of the system is generally good.

I have already pointed out the similarity between the blood picture of chlorosis and typhoid anæmia, and it is interesting to note that these are both diseases in which large hæmorrhages are common. In typhoid epistaxis and hæmorrhage from the bowels are commoner than in any other fever, while in chlorosis gastric ulcer with severe hæmorrhage is a common complication. In these conditions the blood pictures are similar, and in both these anæmias the blood seems to be much diluted. I may mention that the ulcers which occur in these conditions can hardly be the sole factors in producing the hæmorrhages, as there are many other cases of ulceration of the bowels in which hæmorrhage is comparatively rare.

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SIMPLE CONTINUED FEVER IN SOUTH AFRICA.

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DURING this campaign one has met with a variety of continued fever which is not malarial, and certainly not enteric. It is rather sudden in its onset, and the temperature may be from 102° to 104° at the end of the first day. There is usually a good deal of headache and considerable disturbance of the gastro-intestinal tract as shown by the thickly-coated tongue. There is more often constipation than diarrhoea. At first there is very considerable difficulty in being certain that the case is not one of enteric fever, and I have frequently seen typical typhoid tongues at the end of the second day. The duration of the fever is very variable, from 2 to 7 or 8 days, the average being 4 days, and there is very rapid convalescence.

While the 3rd Cavalry Brigade was at Bloemfontein last year waiting for the general advance, we had a good many cases of this sort, and we thought that very possibly the state of the camping ground and crowded condition of the tents, which was brought about by the rains, had something to do with its causation, and so dubbed it camp fever. While at the dynamite factory early in June last I met one of the leading Johannesburg practitioners, and spoke to him about this new kind of fever, which was puzzling me. He told me that I might be perfectly certain that every case of continued fever which we met with in this country, not malarial, was enteric. From my subsequent experience I am quite unable to accept this statement.

During the past three months I have kept notes of these cases of fever. It has frequently happened that, owing to trekking away from the railway line and the difficulties in getting rid of the sick by convoys, I have had to carry many of the men along. No fewer than 16 have been sent back to duty, and the longest period which any of these men has been detained for has been eight days. Some of these men have been allowed to ride with the baggage waggons for two days after discharge, but the bulk of them have gone straight back to their ordinary cavalry duties and field rations. That they were able to do this shows, I think, quite clearly that none of them could have been suffering from even the mildest form of enteric. During the same period 33 cases were transferred to other hospitals as simple continued fever owing to opportunities having occurred for transfer, and I do not think there was any suspicion of enteric fever in any of them.

It would be very interesting to have the experience of some who are doing duty in the general hospitals as to the number of those who are transferred as simple continued fever and are found subsequently to be suffering from undoubted enteric fever. I am aware that great exception is taken (chiefly by the civil surgeons) to the diagnosis of simple continued fever, but I think it difficult at present to diagnose these cases otherwise.

As to treatment, I have found 5 grs. of calomel with 10 grs. of phenacetin to relieve headache, followed by quinine when the fever goes, the most efficacious.

The strength of the column I have been serving with during this period has never been above 500 men.

WE regret to have to announce the unexpected death of Dr. Gibson, of Randalstown, which took place at the residence of his cousin, Dr. William Gibson, Belfast. Dr. Gibson had been ailing for a couple of weeks, and was staying with his cousin, when acute pneumonia suddenly set in, and he died on the morning of May 29th, after barely four days' serious illness. Dr. Gibson was a young man of great promise; he had been a highly-prized house-surgeon in the Belfast Union Infirmary, with its immense field for gaining experience, and had lately acquired a large practice in Randalstown. In both places he succeeded in gaining hosts of friends and well-wishers. He leaves a young widow.