THESIS
for the degree of M.D.
on
BLOOD EXAMINATION FOR DIAGNOSTIC PURPOSES
WITH CASES AND COMMENTS

by

W. J. BURNS SELKIRK,
I propose in the following pages to consider the clinical examination of the blood, as far as possible in its practical application to the daily needs of general practice.

As members of the medical profession we are called, more than those who belong to other sections of the community, to the practice of the ancient virtue of chivalry. The dragons of our childhood's stories that claimed their annual toll take form and reality in such evil powers as Tuberculosis, Cancer, Plague, and the like; and we are called upon, whether as humble men of the rank, or as leaders of the front, to go forth as champions of them that are destined to affliction. We may find in the old chivalry, that knight's from whose shield Merlin blotted out the scroll "I follow fame."

"And made a Gardener putting in a graft,"

"With this for Motto "rather use than fame."

I have endeavoured as far as I have been able to make my study of the blood such as would provide equipment for use. I have been privileged in having in my work the guidance of Dr. Lovell Gulland and the majority of cases which I give later as illustrations were studied when I was his House Physician at Chalmers Hospital.
I am indebted to Dr. Gulland for the permission to use these.

Other cases given I have met in general practice while acting as locum tenens, and the remainder when House Surgeon at Hartlepool Hospital. For the permission to use these latter I am indebted to Dr. A. E. Morison.

The instruments I have used are Gowers Haemoglobinometer and the Thoma Zeiss Haemocytometer. The stains have been Jenner's, and Wright's modification of Jenner's, and the Iodine stain for the Glycogen reaction.

I have particularly preferred Jenner's stain as it is pre-eminently the stain of the general practitioner being easily and rapidly manipulated and the use of it readily modified according as one wished the eosin or the methylene blue to predominate. Where mainly an eosin stain was desired, as in determining the presence or absence of eosinophilia, the film was stained for 4 minutes and well washed in distilled water; where mainly a methylene blue effect was desired, as in looking for basophil granular degeneration of the red cells, or polychromasia, the film was stained 45 - 60 seconds, then dipped once, quickly, in distilled water and set on blotting paper to dry; where, as in the country, distilled water was not obtainable, an ordinary tap water was used though it alters the characters of the stain somewhat.

For/
For the Iodine Stain the following solution was used:

Iodine Gram I
Pot. Iod. Gram II
Ag Destill ad 100 cc.

To this sufficient Gum Acacia was added (about 5 Grammes) to make a thick syrup.

In examining films stained with this, attention was given to the stain only as it affected the polymorph leucocytes. The white and red cells are stained yellow. The reaction consists in a brown mottling or brown dots in the protoplasm outside the cell nucleus in the polymorph leucocytes. Extracellular brown dots are neglected. A fair number of polymorphs are examined before the reaction is pronounced negative.

In cases in which the reaction was only faint and doubtfully present recourse was had to a control film made from my own blood which gave no reaction, and a comparison of this with that of the patient removed all doubt as to the presence or absence of the reaction. After gaining familiarity with the reaction recourse to this control was no longer necessary. Further it was found advisable to pay attention to the following details of the technique: that the film be examined with an oil immersion lens and preferably a No.4 eyepiece, using an Abbe’s condenser/
condenser and the flat side of the reflecting mirror with the diaphragm fully open and good daylight or strong white artificial light.

The chemistry of the reaction is not properly known. It has been doubted whether the material which reacts is glycogen and iodophilia has been suggested as a better name for the property the leucocytes have of thus reacting. This difficulty fortunately does not affect the practical uses of the test.

From repeated examinations by different observers it has been determined that the glycogen reaction is absent in Scarlet Fever uncomplicated by suppuration, in uncomplicated Phthisis and Tubercular Abscess, in simple pleurisy whether dry or serous, in Tubercular Meningitis, in obstructed Hernia, in uncomplicated Rheumatic Fever or its ally Chorea, there being no pericarditis or endocarditis, in Rickets, Infantile Atrophy, Bronchitis, Whooping Cough, Asthma, Alcoholism, Nervous Diseases, Early Cancer and Lead Colic; while it is present in suppuration when the pus is not escaping freely, and of particular value is the fact that it is present in empyema; it is present in Gangrene of all kinds, in Pneumonia before the crisis, in Strangulated Hernia, in Septic and Cerebro Spinal Meningitis, in Endocarditis/
Endocarditis and Pericarditis, in Peritonitis, Acute Cystitis, in Severe Anaemias, in late Typhoid and occasionally in Miliary Tuberculosis. Its occurrence in these three is most probably due to a complicating secondary infection. In all except the late typhoid I have been able to confirm these observations by my own findings.

In an uncomplicated pneumonia the glycogen reaction should have disappeared in a matter of three days after the completion of the crisis. If it persists one suspects delayed resolution, developed or commencing empyema, or the reason may be a suppurative arthritis or in rare cases a pericarditis. From the above lists it is at once apparent that the test is of considerable value for diagnostic purposes. For instance in a previously simple Rheumatic Fever or Chorea its development would point to a developing endocarditis or Pericarditis, in a hitherto catarrhal Appendicitis its appearance would point to involvement of the peritoneum or the onset of suppuration, its presence in colic would exclude lead poisoning as a cause and so on in other instances.

It is particularly to be noted that the whole process of making the film staining and examining it under the microscope and deciding as to the presence or absence of the reaction does not take more than five minutes so that the test makes no unwarrantable encroachment on the time of the general practitioner.
I shall next consider the total and differential count of the leucocytes. Upon the whole I think I have got more help in ascertaining the absence of leucocytosis than from proving its presence. Its absence has frequently been a source of considerable comfort to me in cases which apart from blood examination would have given several days of anxiety.

There is no leucocytosis in health and the minor deviations from it such as what is commonly called a bilious attack or the cramp-like pains in the abdomen that accompany partial obstruction from severe constipation and simulate sometimes appendicitis, internal or external hernia, or even peritonitis. It is also absent in typhoid and paratyphoid infections, Malaria, uncomplicated Influenza, Measles, Rötheln, Mumps, Malta Fever, and in Cystitis, Tuberculosis including incipient Phthisis, Miliary Tubercle, Tubercular Peritonitis, Ostitis and Periostitis, Tubercular Pleurisy or Tubercular Pericarditis, but Tubercular Meningitis often shows a leucocytosis.

It is further to be remarked that leucocytosis is checked by antipyretic drugs but not by cold bathing, unless it is prolonged, so that from this one has a rational justification for avoiding these drugs and having recourse to the short cold bath or cold sponging instead.

The/
The number of the leucocytes is more or less subnormal in those diseases not showing a leucocytosis and is absolutely diminished in starvation and malnutrition, in Spleenic Anaemia and in the Severe anaemias of rickets and Syphilis and that following Haemorrhage. There is also this leucopenia in uncomplicated Pernicious Anaemia, though even with complications comparatively little increase results, but where there is a nephritis and albuminuria the leucocyte count may run to 10,000 or 15,000 and some writers have been led by this to say that the anaemia of Bright's Disease may simulate in all respects Pernicious Anaemia which in my opinion is a mistaken view of the condition. One of my cases illustrates this complication. Another of the cases shows a leucocytosis due to lactation and disappearing with its cessation.

I shall now give some of the cases in which I have used the Glycogen reaction and the leucocyte count.

THE BLOOD IN RHEUMATIC FEVER AND ITS COMPLICATIONS.

Rita Oddie, 8 years of age, was seen by me on 11th Feb. 1906 in the Out Patient Department of Chalmers Hospital. Her complaint was of stiffness of the jaw and fingers and pains in the head. She looked pale and in ill health and was admitted to the Hospital.

The/
The family and previous personal history does not bear upon the point at issue.

On examination in Hospital the patient was noticed to close the eyes spasmodically every minute or so and there were occasional slight jerking movements of the limbs.

In the Respiratory System nothing abnormal was noted. In the Circulatory system the pulse was regular and the rate 100, there was no enlargement or dilatation of the heart, all the sounds of the heart were good and there were no murmurs or pericardial friction.

The Temperature was normal.

The Blood examination showed:-

R.B.C's 4,840,000
W.B.C's 8,000
Hb. 60%
Cl. .64

A differential count of 300 leucocytes showed:-

Polymorphs 62%
Lymphocytes 28%
Eosinophils 11%
Mast cells 1%

The cause of the Eosinophilia was not apparent and in a count three days later it was gone and did not reappear.

The/
The case was diagnosed and treated as one of Chorea with Simple Anaemia.

The heart was carefully examined twice a day, and the blood tested daily for the glycogen reaction. On the 7th day after admission a positive glycogen reaction was obtained for the first time and there was a concurrent rise of the temperature to 100°. The Leucocyte count was 11,000, but there was no change of the heart sounds on auscultation. The temperature returned to normal the following day and did not rise above 99° during the ensuing 3½ weeks of the patients stay in Hospital. Two days later a faint systolic murmur appeared and so the rise of temperature and the appearance of the glycogen reaction was shown to be due to a slight endocarditis. The patient having been absolutely at rest in bed and receiving Iron and Arsenic and having no murmur from her slight anaemia on admission, no other condition could account for the development of the murmur which was still present on dismissal when the Haemoglobin had risen to 80%. The Glycogen Reaction disappeared after 17 days and was absent on dismissal.

A similar case is that of Dinah Booth, aged 7, admitted to Hartlepool's Hospital 10th December 1907, suffering from well marked Chorea. There were no cardiac/
cardiac murmurs or pericardial friction on admission and no glycogen Reaction nor any other affection than the Chorea. The patient was treated with Sod. Salicyl and Sod. Bicarb. and absolute rest in bed. She improved markedly but after 18 days in Hospital developed a Glycogen Reaction and not till three days later did the cardiac murmur develope.

Slightly different is the case of Archie Heriot, aged 4 admitted to Chalmers Hospital 18th November 1906. His mother complained that he was pale, had no appetite, was very cross and had a sore knee. On examination the left knee was found to be slightly swollen and painful and tender. The tonsils were very large but not inflamed. The Temperature was 100.2° and the pulse rate 130. Nothing abnormal could be discovered in the cardiac or any other system except a simple anaemia of the blood the examination of it giving

<table>
<thead>
<tr>
<th>R.B.C's</th>
<th>3,400,000</th>
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<tr>
<td>W.B.C.</td>
<td>7,000</td>
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<tr>
<td>Hb</td>
<td>54%</td>
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<tr>
<td>CI</td>
<td>.8</td>
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<tr>
<td>Polymorphs</td>
<td>68%</td>
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<tr>
<td>Lymphocytes</td>
<td>28%</td>
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<tr>
<td>Eosinophils</td>
<td>3%</td>
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<tr>
<td>Mast Cells</td>
<td>1%</td>
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No Glycogen Reaction.
The ultimate diagnosis in this case lay between Rheumatic Fever affecting the joint and Osteomyelitis. If it were Rheumatic Fever then as there were no complications discoverable there would be no Glycogen Reaction whereas if the reaction were present the case would be osteomyelitis unless a cardiac murmur could be found in 2 or 3 days. I consider this an important use of the test as I know of two cases, though I am not allowed to use them, in which osteomyelitis affecting the ankle was treated as a Rheumatic Fever Joint and in both cases the leg had to be amputated, the error being discovered too late.

This case was diagnosed and treated as Rheumatic Fever and simple anaemia with resulting improvement of the joint. But three days after admission the patient developed a Glycogen Reaction, a mitral systolic murmur and a leucocytosis of 17,000 a pretty severe endocarditis had developed. The Leucocytosis and the Glycogen Reaction remained present for close upon a month.

On 21st December 1906 the Blood Count showed

\[
\begin{align*}
\text{R.B.C.} & \quad 4,200,000 \\
\text{Hb} & \quad 86\% \\
\text{W.B.C.} & \quad 9,000 \\
\text{No Glycogen Reaction.} & \\
\end{align*}
\]

On 4th January 1907 the Surgeon removed the enlarged tonsils, the patient had no trouble from the operation and went/
and went home on the 10th there being still a slight mitral systolic murmur present but no symptoms from it and in our opinion all active endocarditis was absolutely gone.

These cases illustrate the use of the Glycogen Reaction as a control for where one knows of its presence one will be on the lookout for a cause such as endocarditis or pericarditis; its use in excluding osteomyelitis, a matter of no small satisfaction to an anxious practitioner, and, in my opinion, it may be used to determine when such a process as endocarditis, with which knowledge one may pronounce the patient so far convalescent and increase his liberty of action.

The cases also suggest that mild forms of endocarditis may readily pass unnoticed in childhood possibly not being brought under medical care at all. The resulting lesion may be latent till middle age or so, when failure of compensation may occur and no history pointing to previous heart affection be obtainable the original inflammation being unknown to or forgotten by the patient and relatives.

I desire here to state that I do not consider blood examinations as supplanting other methods of clinical examination and observation but regard them as entirely auxiliary but taking a worthy place in that/
that collection of all possible evidence on which alone a sound supportable judgment can be placed.

THE GLYCOGEN REACTION AND LEUCOCYTOSIS
IN PNEUMONIA AND EMPYEMA.

The next case I wish to submit is that of a nurse, aged 33, admitted as a private patient to Chalmers Hospital on 20th March 1907.

She had enjoyed excellent health till March 19th when, while nursing an Influenza case, she had a sudden onset of weakness and malaise. She went on with the nursing but on the morning of the 20th was seized with acute pain in the front of the chest on both sides and in the right side behind accompanied by Dyspnoea. She had to stop work and was brought to the hospital in an ambulance. On admission her temperature was 103.2°, the pulse rate 130, the respiration 60. She was in great distress, particularly from the acute stabbing pain in front and behind accompanying each respiration. Further clinical examination revealed the presence of double Pneumonia and widespread pleurisy.

The pulse was regular and of fair tension, there were no heart murmurs or pericardial friction, the heart was not dilated nor displaced. There was a trace of albumen in the urine.

The Leucocyte Count on admission and succeeding days was:

22nd March 22,000

Glycogen Reaction in a few Polymorphs.
23rd March 28,000
Glycogen Reaction in more Leucocytes.

24th March 22,000
Glycogen Reaction about same.

25th March 40,000 and Glycogen Reaction in most of the Leucocytes and large brown staining granules in some.

26th March 39,000 Glycogen Reaction as before

30th March 41,000 Glycogen Reaction less marked.

4th April 20,000 & Good Glycogen Reaction.

11th April 19,000 " " " "

16th April 28,000 " " " "

18th April 19,000 " " " "

Despite the apparent critical condition of the patient, the increasing leucocytosis and Glycogen Reaction of the first eight days enabled a good prognosis to be given, a matter of great comfort to the five medical men interested in the case, to the relatives and friends and to the patient herself and giving that great essential, encouragement, to all concerned to spare no efforts to obtain ultimate success.

On the 24th there was a pseudo crisis and again on the 28th. Thereafter till 19th April the temperature swung between normal and 102°.

On the 28th owing to the character of the dulness it/
it was decided to explore the right side of the chest. This was done 1\(\frac{1}{3}\)" below the lower angle of the scapula and clear fluid obtained but apparently there was only a thin layer present. Three drops of the fluid were dried on a cover glass and stained. On examination this showed some polymorph leucocytes and numerous pneumococci. From the number of pneumococci present it was expected that pus would ultimately develop.

On the 30th the right side of the chest was oza aspirated and ten, of somewhat opaque fluid removed. No more could be obtained.

On April 11th on aspirating the right side again below the scapula 6 oz. of pus were got. The left side of the chest was by this time resonant and apparently well cleared up. The cardiac dulness now extended 1\(\frac{1}{4}\)" outside the left mammillary line and 1\(\frac{1}{4}\)" to right of the sternum but the apparent apex beat was in the 5th space 1\(\frac{1}{2}\)" internal to the mammillary line. It was judged that the heart was probably somewhat dilated and pushed to the right.

On April 15th aspiration was repeated below the scapula with the largest canula and only \(\frac{1}{2}\) oz. pus obtained. The canula was moved about and the lung surface felt close to the chest wall. No better result was obtained in the axillary region which was explored.

The/
The pulse rate varied now from 100 to 130 and the patient was not gaining strength and a considerable leucocytosis and Glycogen Reaction were being maintained indicating that there was an accumulation of pus which we had not been able to strike, the surface layer not being sufficient to account for the continued raised temperature and the pulse rate and general condition of the patient. The considerable maintained leucocytosis decided against a complicating phthisis being the cause of the symptoms. It was decided to ask the Surgeon to explore for an interlobar empyema. Mr. Stiles on 19th April removed under chloroform three inches of the 8th rib opened the pleura and with his finger succeeded in reaching an interlobar empyema from which one pint of pus was evacuated.

Thereafter the temperature gradually fell to normal and the pulse rate to 90 and the Leucocytosis and Glycogen Reaction slowly decreased. Pus continued to discharge till May 16th. The Leucocytosis then was 10,850 and the Glycogen Reaction almost gone and the patient making an uninterrupted convalescence. On June 13th when the patient left hospital, the Leucocytosis was 9,000 and No Glycogen Reaction was present. She is now doing full nurse's duty. In this case the continued leucocytosis and Glycogen Reaction/
Reaction was of the greatest help in justifying our submitting the patient despite her weakened state to the risks of operation and chloroform. The good result of the operation proved that the interpretation put upon the continued leucocytosis and Glycogen Reaction was so far correct. To have confidence in such an interpretation is of course of much more importance in general practice than in hospital practice where the patient is under a continual observation and can receive necessary treatment without delay.

Another case is that of John Barrett, aged 22, admitted to Chalmers Hospital, 13th February 1907, as a case of Right Monolobar Pneumonia. The crisis occurred on the 15th the temperature falling from 103.6° to normal. Three days later the leucocytosis was still 23,000 and Glycogen Reaction still present so that the development of Empyema was feared. The Right base was explored and turbid fluid obtained showing numerous polymorph leucocytes and a few Pneumococci on examination. The fewness of the Pneumococci made the prognosis as to empyema good and the cells being polymorphs ruled out a complicating tubercular pleurisy. On 22nd February the leucocytosis was 10,500 and the Glycogen Reaction had disappeared, and the patient made a good recovery so that,
so that, in this case, though there was a complication, it turned out to be a very mild one.

**LYMPHOCYTOSIS OF BLOOD AND PLEURAL FLUID IN PULMONARY TUBERCULOSIS.**

The following case illustrated the value of examination of the blood and pleural fluid.

Wm. Mc.Lean aged 40, was admitted to Chalmers Hospital on March 20th 1907. He complained of a chill 13 days previously. He said he had no acute pain but his body was sore all over and he got a little breathless on exertion. He thought he had Influenza. He said he had had Influenza three times before. He had had no other illnesses since early childhood. He had not had a cough previously for the last twelve months at least but had one during this attack. He looked somewhat thin, but said he had always been so and did not think he had been losing weight.

On examination there was stony dulness on the right side of the chest from the base to the spine of the scapula and well up into the axilla.

No friction or crepitations were heard anywhere and only one rhonchus which was in the region of the 3rd Left rib anteriorly. There was also dulness at the left apex and hyper-resonance in the subclavicular region. The pulse rate was 86 and Temperature swinging from 99° - 101°.

A/
A large effusion in the right pleura was diagnosed and XLVI oz. (46) were aspirated on the 21st March. Some of this I centrifuged and the deposit showed a great preponderance of mononuclear leucocytes and no organisms.

The blood examination showed:-

W.B.C's 5400 with relative lymphocytosis;

No Glycogen Reaction.

The fluid reaccumulated and on the 27th XXXIV cunces were withdrawn and this showed a similar excess of lymphocytes.

No T.B. were found in the Sputum.

The temperature became normal on 30th March and remained so and no more fluid had gathered. After the withdrawal of the fluid a few crepitations could be heard at the base of the Right Lung.

From the examination of the chest and blood and the absence of the Glycogen Reaction the large number of lymphocytes in the pleural fluid a diagnosis of tuberculosis of the lungs was made and on April 3rd the patient was sent to the country. I do not think such a diagnosis could have been justified apart from the examination of the blood and pleural fluid without which one would have had to have the patient under observation for a considerably longer period.

A/
A similar case is that of Margaret Scott, age 41, admitted to Chalmers Hospital 7th December 1906. She complained of loss of flesh and weakness. While in hospital her temperature ran from 99°-102° being generally at 100°. The pulse rate from 110 to 126. There were dull areas in her chest but no signs of active disease and no fluid at the bases on exploration though there was dulness there.

Blood Examination showed

7th December  R.B.C.  2,900,000
            W.B.C.  2500
            Hb.    50
            CI     .86

Relative small celled Lymphocytosis, but no unusual preponderance of lymphocytes.

10th December  W.B.C.  3500
17th December  W.B.C.  4400

Polymorphs 65%
Small Lymphocytes 30%
Large 3.5%
Eosinophils 1.5%

The Red cells showed granular degeneration and polychromasia.

There was no Widal's reaction. There was no evidence of Exophthalmic Goitre or of any special blood disease other than simple anaemia and from the leucopenia anaemia loss of flesh, temperature and pulse/
pulse rate a diagnosis of tuberculosis was made though no other signs or symptoms of active disease could be obtained and there was no expectoration. The patient became noisy at night and peculiar in her conduct during the day and was send home on 31st December. She was however still kept under observation and 6 weeks later developed an enlargement of the Liver and Spleen and Ascites and shortly afterwards died.

A P.M. was obtained and a condition of general tuberculosis found. Thus the importance attached to the leucopenia as, in the absence of other satisfactory explanation, indicating tuberculosis was justified.

**BLOOD EXAMINATION IN DIAGNOSIS OF LYMPHADENOMA.**

A somewhat similar condition of the blood was seen in John Martin, a labourer, aged 35 admitted to Chalmers Hospital on 27th October 1906. He complained of attacks of shivering and malaise and of constipation. He said that except slight blood poisoning five years ago he had had no illness previous to admission.

For the first time in May 1906 he had shivering fits and felt ill for two days. This recurred at intervals of three or four weeks till he was admitted with this attack in October which he says is the worst he has had.

Re/
He was a man of large build and well developed and locked in excellent health.

The only things calling for any note in the examination of the patient were the presence of a firm slightly elastic swelling just above the Left Poupart's ligament, fairly movable and lying just under the skin, and the size of a hen's egg, and another similar but rather larger, felt with difficulty in the left hypogastric and lumbar regions. There were no other abnormal swellings palpable in the body and during the course of the illness no more appeared. The patient said he had not noticed the swelling in the groin till I drew his attention to it. Both testicles were descended.

The temperature on admission was 101.4° and the pulse rate 84.

The examination showed:-

28th October R.B.C's 4,800,000
W.B.C's 6,250
Hb. 85%
Cl - .9
Polymorphs. 62.3%
Large Lymphocytes 25.7%
Small " 10%
Basophils 2%

4th November/
4th November R.B.C's 4,500,000
W.B.C's 9,800
Hb. 75%
CI .8
Polymorphs 57.5%
Large Lymphocytes 26.5%
Small " 10.8%
Eosinophils 6.2%

The day following admission the Temperature fell to normal and remained so. The patient was treated for his severe constipation and 10 days later left Hospital feeling quite well he said.

He was readmitted on 29th January 1907. He said he had had several attacks of shivering since leaving hospital and was feeling weaker. His condition was the same as on previous admission but he was 3 lbs heavier in weight. Constipation was very bad again and he required to take Gr. XX Pil. col. et Hyoscy. to get a motion.

The temperature was 103° and pulse 112. For the rest of the illness the temperature varied between normal and 104°. There was, as a rule, a daily swing of about three degrees. The pulse rate fell to 80 but about the middle of March began to increase and ultimately reached 130.

The Blood Examination showed

30th January/
30th January  R.B.C.  4,410,000
W.B.C.       4,600
Hb.          75%
Large Mononuclears  47%
Small "          3%
Polymorphs.    50%

This condition of the blood was maintained till the middle of March when a rapid decline of Red cells and Haemoglobin began.

Up to the last fortnight of the illness the patient maintained his weight being on 11th April 10 st. 8½ lbs.

The Blood Examination on March 18th showed:-

R.B.C's.   3,110,000
W.B.C's.   4,000
Hb.        50%
Cl.        8
Polymorphs. 60%
Large Lymphocytes  33%
Small Lymphocytes  5%
Eosinophils    1%
Mast Cells     1%

The examination of a film showed a simple anaemia as far as the red cells were affected.

On 17th April/
On 17th April

R.B.C's 1,910,000
W.B.C's 3,200
Hb. 38%

On 21st April

R.B.C's 1,500,000
W.B.C's 3,700
Hb. 27%
Polymorphs 55%
Large Lymphocytes 42%
Small Lymphocytes 6%
Mast Cells 2%

Thereafter the heart rapidly failed and the patient died on 27th April, 1907.

The diagnosis was considered to be between Malaria, Typhoid Fever, General Tuberculosis, and Lymphadenoma. No benefit resulted from the administration of Quinine. No organisms could be found in the blood. The patient had never been abroad.

The blood was examined on three occasions for Widal and Paratyphoid reactions at the Royal College of Physicians Laboratory and the results were negative.

I frequently examined the urine for the Diazo Reaction. It was always negative. No abnormal constituents were at any time detected in the urine.

No affection of the lungs or other viscera could be detected.
be detected till towards the end of March when the heart sounds began to get weak and a faint mitral systolic murmur could be heard. Crepitations then appeared at the bases of the lungs. The spleen was now slightly enlarged.

On 26th February and on 10th March the Ophthalmos-Tuberculin test was negative. The body weight was well maintained throughout.

Although the blood examination was not in itself sufficient to base the diagnosis upon in conjunction with other clinical methods and observations it enabled one to make a diagnosis of Lymphadenoma. The points it was based upon being the ague-like attacks, the persistent weakness, the exclusion of other probable diseases, the character of the enlarged gland, the other abdominal swelling and finally the peculiar lymphocyte condition of the blood, a condition noted by other observers such as Da Costa and Cabot in a minority of their cases of Lymphadenoma.

The Post Mortem examination was made by Dr. Dickson on 29th April, the body having been injected with formalin shortly after death.

Apart from conditions due to heart failure the following were found.

The colon and small intestine were anaemic and atrophied, which is probably the explanation of the persistent constipation.
The Retroperitoneal glands were enlarged and formed a mass down the middle line about 10 inches long and three inches broad reaching from the diaphragm to slightly below the scaral promontory and forming a matted mass; the individual glands varying from a pea to a bantam's egg in size. There were some enlarged glands down the Left Iliac vein. The largest gland was in the left iliac region just above Poupart's Ligament and about the size of a Tangerine orange. The spleen was enlarged. The Liver was also enlarged and its surface showed numerous small white spots. On section it showed a slight iron reaction after prolonged use of the reagents.

Microscopic examination of the glands by Dr. Dickson showed them to be Lymphadenomatous.

Such an obscure case as this indicates the value of blood examination as a guiding post to the direction of one's search. Search in other possible directions having proved unavailing its indication was considered reliable and in the end justified. Apart from the blood examination the enlarged gland in the groin and an indefinite swelling in the abdomen would not have justified a diagnosis of Lymphadenoma.

LEUCOCYTOSIS IN RELATION TO SCARLET FEVER.

My next case is that of Emma Major, aged 19, admitted to Chalmers Hospital 11th January 1907 with a/
a history of attacks of recurrent abdominal pain. While in hospital however, a complication arose. The temperature on admission was 101.2° and pulse 94. There was constipation. On the following day the 12th the patient received Gr. III of Pil. Col. et Hyosc. and a soap and water enema. The Temperature fell to 99.2°. The pulse was 88. On the 13th the temperature was 101° and pulse 86 and a bright scarlatiniform rash developed on the neck and spread downwards over the rest of the body. The patient complained of nausea and headache. The tongue was coated. The throat was unaffected. The pulse rate and the throat condition were against a diagnosis of Scarlet Fever. But as is well known this disease may be indefinite in type and it is just such cases that bother one very much in general practice. For instance I have been confronted with just such a case in a girl of 9, having, in addition, a rapid pulse and living with her parents and these parents conducting a dairy farm.

Now in Scarlet Fever there is a leucocytosis at an early stage, sometimes even before the eruption appears, the White Blood Corpuscles numbering as a rule from 15,000 to 30,000 and the number declining with the fall of temperature.

Bearing/
Bearing this in mind I examined the blood of Emma Major and found:

- R.B.C's 4,850,000
- Hb. 70%
- Cl. .7
- W.B.C's 5,200

No Glycogen Reaction.

This justified the exclusion of Scarlet Fever which was a great relief to one's mind and enabled us to regard the rash as in all probability due to the enema. The case was watched for three weeks and gave no further signs of Scarlet Fever.

**THE LEUCOCYTE COUNT IN INFLUENZA AND IN SEPTICAEMIA.**

Somewhat analogous is the case of a puerperal woman, during an epidemic of Influenza.

While acting as locum tenens at Hawick I had under my charge a patient whom my principal had attended in confinement. The case had been one of complete placenta praevia and had caused much anxiety. She progressed well till the 5th day after delivery when the Temperature rose to 103.2° and the pulse rate was 110 and the patient complained of headache and nausea. The lochia were quite healthy but a good deal less than on the day before. One feared developing sepsis. Unfortunately I had no facility for making a blood examination. After two more days the Temperature and pulse fell to normal and the lochia increased/
increased again. The attack had been Influenzal. A blood examination in such a case would I am sure have saved one much worry and unnecessary visits as there is no leucocytosis in uncomplicated Influenza but it is distinct in sepsis unless the poisoning is extremely profound.

**THE BLOOD IN EXOPHTHALMIC GOITRE.**

I now desire to submit some blood examinations in cases of Exophthalmic Goitre.

Mary Middlemass, aged 13, was admitted to Chalmers Hospital on 24th October 1906. She had well marked exophthalmic Goitre. The Blood examination showed:--

<table>
<thead>
<tr>
<th>Date</th>
<th>R.B.C's</th>
<th>W.B.C's</th>
<th>Hb.</th>
<th>CI.</th>
<th>Polymorphs</th>
<th>Lymphocytes</th>
<th>Eosinophils</th>
<th>Basophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>28th October</td>
<td>5,500,000</td>
<td>10,000</td>
<td>90%</td>
<td>.81</td>
<td>55.5%</td>
<td>41.3%</td>
<td>1.5%</td>
<td>1.2%</td>
</tr>
<tr>
<td>7th November</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>9,500</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11th December</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
11th December

W.B.C's 9,200
Polymorphs 47%
Lymphocytes 52%
Eosinophils 1%

Mrs. Hall was admitted to Chalmers Hospital
7th January 1907.

She had a rapid pulse and fine tremors of the hands, no exophthalmos and an almost imperceptible enlargement of the Thyroid Gland.

The Blood examination showed:-
10th January  W.B.C's 5200
Polymorphs 58%
Lymphocytes 40%
Eosinophils 1.5%
Basophils .5%

20th January
W.B.C's 7000
Polymorphs 52%
Lymphocytes 45%
Eosinophils 1.6%
Basophils 1.4%

The significance of such a lymphocytosis in this disease has not yet been determined. Nor can much diagnostic value be claimed for it, yet in early or atypical cases it may be regarded as so far confirmatory.
confirmatory.

In a case as yet undiagnosed or diagnosed as another condition the finding of such a blood condition would call for a careful scrutiny for other signs of Exophthalmic Goitre.

**LEUCOCYTOSIS INDICATING COLLECTION OF PUS.**

The following two cases were under my care while acting as locum tenens. Both of them caused me much worry. They were both unsuccessfully treated. In my opinion comparatively good results might have been obtained had I then been in the habit of making blood examinations.

The first is that of Mrs. Munro, aged 65, at Perth. I was called to her in June 1906. She complained of a swelling of the neck. Stiffness of the neck and great pain at the site of the swelling and radiating upwards over the head. She had also great weakness. On examination a hard brawny undefined swelling was felt on the right side of the neck and occupying the posterior triangle. The Temperature was $100^\circ$ and while the patient was under my care it was sometimes normal and sometimes running to about $100^\circ$ especially at night. The patient was stout and the heart sounds were weak but there were no murmurs. There was pretty bad cystitis and some bronchitis, either of which might have caused the raised temperature.

There/
There was no fluctuation in the swelling. The pain was at times excruciating and morphia was frequently called for.

The swelling had been noticed about five weeks previously causing some pain and the condition had been treated by the family circle.

The diagnosis lay between a deep seated abscess and a tumour pressing upon the cervical nerves and possibly involving the cervical vertebrae. From a general view of the patient's condition I considered that she could not survive a severe operation but would benefit much if at operation the condition turned out to be a deep abscess. The patient was strongly against operation and from the hardness of the swelling I believed the condition to be a tumour, so that I could not urge operation with the promise of a successful result. So palliative drugs and poulticing for the pain were resorted to. Three weeks later I left the patient unimproved and decidedly weaker, the swelling being larger but otherwise unaltered in character. Her own doctor now continued the same treatment till two weeks later slight fluctuation was made out and permission then obtained for a small incision under local anaesthesia. This was made and a large quantity of pus escaped. The patient was however by this time very exhausted and died from heart failure a week later. I feel sure that a blood examination when the patient was first seen/
seen would have revealed a leucocytosis and a Glycogen Reaction. From a leucocytosis one would have excluded the tumour diagnosis in this case and the cystitis being comparatively mild would not have accounted for it. So one would have been justified in strongly urging even demanding operation with promise of a beneficial result while the patient had still strength to profit by it.

The second case is that of a boy at Dumfries who, coming in contact with a revolving saw-wheel, received a cut on his knee, a piece of the patella being sawn off and the knee joint slightly opened. I advised his removal to Hospital where he could get more constant care and observation. His mother refused and pressed me to treat him at home. I consented. Despite all care the joint suppurated and to all appearance the pus was discharging freely. The temperature was swinging up to 101° and the pulse was gradually increasing in rate but I was misled by the apparent free discharge of the pus. Ultimately, as the patient continued to deteriorate, I demanded his removal to hospital to have the joint freely opened and frequently flushed. This was done and at the operation a large pocket of pus was found extending upwards between the femur and the muscles. Even after free incisions the patient failed to improve and/
and the leg had to be amputated to check the blood poisoning. Thereafter the patient slowly recovered. I am convinced that blood examinations would have revealed a distinct persistent leucocytosis indicating that there was not a free discharge of all the pus so that one would have insisted on the joint being freely laid open at a much earlier date and thereby quite probably the leg would have been saved.

Of course had one had more experience one might have surmised earlier what was occurring but on the other hand the blood examination would have taken the place of the experience which is apt to be of value, as in this case, in proportion to its painfulness.

**BLOOD EXAMINATION REVEALING OBSCURE PERNICIOUS ANAEMIA.**

There is one blood disease in particular which is apt to be elusive, escaping diagnosis much more frequently, I think, than is generally realised. This disease is Pernicious Anaemia.

The reasons of its elusiveness are found in its pathology and the fact that a lemon tint of the skin, which is frequently only a late manifestation, has come to be associated with the disease as a necessary feature of it. The disease in the great majority of cases can be and should be diagnosed long before the development of the lemon yellow tint.

For the pathology of the disease I accept that given by Hektoen and Riesman in their System of Pathology/
Pathology, with one reservation. I would emphasise these points of it

(a) High colour index with marked obliscythaemia and large average size of red cells,
(b) Sclerosis of the posterior and lateral columns of the spinal cord, accounting for nerve symptoms.
(c) Atrophy of the gastric and intestinal mucous membrane accounting for dyspepsia and diarrhoea.

I take exception to their statement that there is no leucocytosis.

Of cases complicated by Bright's Disease some have a leucocytosis and these cases besides having a blood picture typical of Pernicious Anaemia also show post mortem the typical iron reaction in the liver. So that I hold it is much more rational to regard these cases as Pernicious Anaemia plus Bright's Disease than as cases of Bright's Disease producing an anaemia indistinguishable from Pernicious Anaemia as some writers do and so encourage an error in treatment, the neglect of Arsenic.

Lactation during the disease may also produce a leucocytosis as shown in one of my cases.

And again writers, for example Osler, exclude from true Pernicious Anaemia those anaemias resembling it otherwise, but developing in pregnancy and parturition or accompanied by atrophy of the stomach or dead Bothriocephalus Latum in the intestine.

The Atrophy of the stomach may be accepted as
and necessarily part of the pathology of the disease and not the cause of the blood condition. Pregnancy and Parturition may merely emphasise a per-existent Pernicious Anaemia, for undoubtedly many existing ones are unrecognised, and, indeed, in interval periods are unrecognisable by our present methods.

The following are two counts of cases which had previously been in Hospital with very marked Pernicious Anaemia and had come up to report themselves. On examination I found

1st Case

- R.B.C's: 6,000,000
- Hb.: 100%
- Cl.: .83
- W.B.C's: 6200
- Polymorphs: 48.5%
- Lymphocytes: 49.7%
- Eosinophils: 1.7%
- Basophils: 1%

2nd Case

- R.B.C's: 4,980,000
- Hb.: 100%
- W.B.C's: 10,600

Bothriocephalus Latuus certainly produces an anaemia in all respects similar to that we know as Pernicious Anaemia and yet absolutely curable by the expulsion of the worm. This but encourages the hope and supports/
supports the belief that a cure may yet be found for what we regard as true Pernicious Anaemia.

We know so far that a lipoid substance can be obtained from the decomposing proglottides of the Bothriocephalus Latus which experimentally produces a Pernicious Anaemia, and that haemolyzing substances can be obtained from the intestinal mucosa which atrophies as we know, in Pernicious Anaemia. So that the atrophy and absorption of part of this mucosa may have a very direct bearing upon the production of the Pernicious Anaemia.

A colour index of 1 or higher with Red blood Corpuscles 3,500,000 per cmm. or fewer is practically diagnostic of Pernicious Anaemia. When the Red Blood Corpuscles are more numerous the high colour index is not necessarily conclusive. With the high colour index and Red Blood Corpuscles 3,500,000 or less one finds other signs of Pernicious Anaemia. Thus the Red Blood Corpuscles are of an average large size and vary both in size and shape, the circular shape being considerably departed from in favour of the oval, very much more so than in a secondary anaemia with a similar number of red cells. There may be megaloblasts but these seem to disappear from the peripheral blood when the patient is confined to bed. As a rule there is Leucopenia. Symptoms corresponding to/
to other pathological features are generally present. Thus there may be ulcers of the mouth, atonic dyspepsia, diarrhoea, numbness and tingling affecting particularly the extremities, or ataxia, or the complaint may be simply of weakness. The complexion may be excellently red till late in the disease or it may be muddy instead of having the typical lemon tint. Thus it can easily be understood that the more familiar symptoms such as the dyspepsia, the diarrhoea, or the nerve effects hold the attention of the Physician to the exclusion of the underlying blood condition. That this is so some of my cases show. It is also curious that in reckoning the numbers of patients admitted to during 1906 Chalmers Hospital under the headings of the various diseases the first place is held by Pneumonia and Pernicious Anaemia an equal number having been admitted under each of these headings.

My first case is that of William Wilson, aged 53, whom I saw at Moniaive, Dumfriesshire at the end of March 1906. The patient was a shepherd living in a very secluded part of the hills. He had always been a healthy man till January 1906 when he began to complain of weakness and unaccustomed breathlessness in climbing the hills. He consulted a doctor and particularly asked if he would be fit for the heavy work/
work of the lambing time in the late spring. The doctor said he would and that he only required a tonic which was prescribed. The patient in April happening to be in Moniaive consulted me with the same symptoms, his master particularly desiring to know what work he was fit for as he had charge of a large and remote sheep farm. As a result of a thorough examination of the patient the only abnormalities to be discovered were a muddy complexion and slight enlargement of the Liver. The urine was normal. A blood film showed poikilocytosis of some of the red cells which were on an average large and mostly somewhat oval in shape and some megaloblasts one of which showed mitosis. The patient had travelled 17 miles that morning which I think may account for the prevalence of the megaloblasts. They disappeared after a few days rest in bed in Hospital. A diagnosis of Pernicious Anaemia was made from the blood film and master and man informed of the poor prognosis as to work and arrangements made to send the patient to Chalmers Hospital where the diagnosis was further confirmed by full examination of the blood.

John Lennen was admitted to Chalmers Hospital 4th March 1907 and was under my care there. He complained of breathlessness on exertion and great weakness.
These had been present more or less for six months and he had had medicines for them. His work consisted of fitting gas pipes in houses and connecting them in the streets in this way he frequently inhaled gas and particularly he remembers one occasion shortly before this illness began when he had breathed gas for six hours a day for a fortnight, while working at a leak in the street. He felt sick but kept on working. Towards the end of the fortnight vomiting began even water being rejected. This has been present off and on since.

The roof of the mouth was sore and on examination was seen to be very red and glazed. The face was rather puffy there was no oedema elsewhere, the cheeks were somewhat red and the rest of the complexion muddy.

There had been attacks of numbness in the hands. The patient sways markedly when standing with the feet together and the eyes shut. The urine shows specific gravity 1013, no sugar, a little albumen present, some granular casts.

The blood count showed

4th March  
R.B.C's 1,710,000
Hb. 58%  
Cl. 1.1
W.B.C's 10,000

This/
This illustrates the combination of Pernicious Anaemia, Nephritis and a comparatively high leucocyte count.
A test breakfast revealed absence of Hydrochloric Acid which is the rule in Pernicious Anaemia and points to a farinaceous diet as most suitable.

The blood improved showing on 13th March

<table>
<thead>
<tr>
<th>R.B.C's</th>
<th>2,170,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb.</td>
<td>43%</td>
</tr>
<tr>
<td>Cl.</td>
<td>1.0</td>
</tr>
<tr>
<td>W.B.C.</td>
<td>9,800</td>
</tr>
</tbody>
</table>

and on 23rd March

<table>
<thead>
<tr>
<th>R.B.C's</th>
<th>2,200,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb.</td>
<td>54%</td>
</tr>
<tr>
<td>Cl.</td>
<td>1.2</td>
</tr>
<tr>
<td>W.B.C.</td>
<td>8600</td>
</tr>
</tbody>
</table>

The patient however was noticed to be more and more inclined to be drowsy and in spite of all effort on our part passed into a uraemic coma without uraemic fits and died on 31st March.

Unfortunately we could not obtain a post mortem examination. I think there may be some connection between Pernicious Anaemia and the inhalation of coal gas. I have known four other cases of Pernicious Anaemia, but not under my care, in which the inhalation of/
of gas figured prominently in the history; none of these had nephritis, all made a good temporary recovery under treatment. They were all males. I am inclined to think that for uncomplicated cases with a history of coal gas inhalation the prognosis for temporary recovery is comparatively good. But I hold that one would not be justified in saying that the inhalation of coal gas may produce an anaemia in all respects similar to Pernicious Anaemia yet not to be placed under that disease.

A case showing well the elusive nature of Pernicious Anaemia is that of a private patient, aged 55 brought up to Chalmers Hospital by Dr. McPherson on 9th January 1907 for consultation. She had a history of recurrent very intractable attacks of diarrhoea during the past two years. There was also some loss of strength. The diarrhoea had been treated as such with apparent temporary successes. Its recurrence had raised a fear of malignant disease of the intestines. The patient had never been abroad. Her complexion was particularly good and did not suggest anaemia of any kind. All her organs and systems were clinically examined and no pathological condition elicited till I examined the blood which showed the following:-

<table>
<thead>
<tr>
<th>R.B.C.'s</th>
<th>1,870,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb.</td>
<td>58%</td>
</tr>
<tr>
<td>CI.</td>
<td>1.57</td>
</tr>
<tr>
<td>W.B.C.'s</td>
<td>5000</td>
</tr>
</tbody>
</table>
The red cells were of large average size and showed great variation of shape and the microcytes were few. There were present granular degeneration and polychromasia of the red cells. The case was one of Pernicious Anaemia and had been through the hands of several doctors who had failed to discover the true condition through not examining the blood though it is well known that attacks of diarrhoea are of common occurrence in the course of Pernicious Anaemia. These are not due to arsenic for they occur in cases that have never been treated with it.

The following case first showed noticeable symptoms of the disease during pregnancy. The patient Mrs. Arthur, aged 28, was admitted to Chalmers Hospital 18th March 1907. Eighteen months previously and one month before parturition she had an illness being affected by a swollen tongue, sore mouth, weakness and diarrhoea. At the birth she lost a good deal of blood and was in bed for four months after when the illness passed off. Four months later, two months before parturition and nine months before admission the tongue again became sore and developed ulcers. No sooner did one of these heal than a fresh one developed. The mucous membrane of the cheek became also similarly involved. On admission to hospital two/
two ulcers were present on the tongue and one on the cheek. For the nine months previous to admission there have been recurrent attacks of diarrhoea. There has been persistent weakness and dyspepsia. This second attack developed two months before parturition. The patient looked somewhat anaemic but the cheeks had a fair red hue. There was a faint mitral Systolic murmur and some dyspnoea on exertion. A full examination revealed nothing else of note. The patient on coming to hospital was still suckling her child. Her blood was examined for the first time as an outpatient at hospital on March 9th she having been sent for persistent weakness and recurrent diarrhoea.

The examination showed:

R.B.C's  2,710,000
Hb.     64%
Cl.     1.2
W.B.C.  12,600

The red cells were of large average size and of great variation in shape tending chiefly to the oval. There were no megaloblasts or normoblasts. The leucocytosis was accounted for by the lactation. The patient had been treated by iron outside without benefit. She was ordered to cease lactation and was admitted to hospital on the 18th March. She was treated/
treated in hospital for Pernicious Anaemia receiving Arsenic, Farinaceous Diet, a daily saline douche per rectum and absolute rest in bed. Lactation was stopped.

The Blood examination now showed

19th March  R.B.C's  2,520,000
          Hb.     65%
          Cl.     1.3
          W.B.C.  8000.

The leucocytosis is gone, lactation having been stopped.

27th March  R.B.C's  2,720,000
          Hb.     80%
          Cl.     1.5
          W.B.C.  8800.

4th April   R.B.C's  3,030,000
          Hb.     82%
          Cl.     1.3
          W.B.C's  6000

She now felt considerably better and was digesting the farinaceous diet comfortably. The ulcers were healed. Owing to circumstances at home she left hospital.

Whether or not the relation to pregnancy in this case be regarded as a coincidence, all the signs and symptoms can be accounted for by the admitted pathology of Pernicious Anaemia and similar cases coming.
coming to Post Mortem examination have shown the actual pathological lesions. Until further light as to the cause proves or disproves our conclusion such a case undoubtedly belongs to the clinical and pathological entity known as Pernicious Anaemia. This consideration may also be extended to the Bothriocephalus Anaemia with the admittance that in it we have a known and removable mediate cause.

The next case Mrs. Grant, aged 57, illustrates nervous symptoms. She had a history of recurrent dyspepsia and slowly progressing weakness and dyspnoea of 6 years duration. She was seen by Dr. Gulland on 11th January 1906 and Pernicious Anaemia diagnosed from the general and blood condition. The blood had not been previously examined. She was admitted to hospital and left greatly improved on 21st February. She had no nervous symptoms at that time.

She was readmitted during my term at Chalmers Hospital on 24th January 1907.

The Blood examination showed:-

25th January R.B.C's 1,930,000

Hb. 48%

CI 1.28

W.B.C's 2800

She/
She had then the lemon complexion of the disease. She now complained of pains starting in the shoulders and running down the arms, starting in the hips and running down to the knees and feet; prickling feelings in the feet and when walking she feels as if there were large pads under the soles. The feet are also numb, Romberg's symptom is present. Such a condition, were the lemon complexion absent, might readily be assigned to an uncomplicated nerve affection, the underlying blood condition being overlooked and so an error of diagnosis committed.

Dr. Gulland, for instance, has published cases where diagnoses of peripheral neuritis and of aphasia had been made, the underlying condition of Pernicious Anaemia being found on Blood examination.

Later, while in hospital this patient was partly aphasic for two days, being only able to say 'yes' and 'no'.

The case did not respond much to treatment as the following counts show.

1st February  R.B.C's  2,100,000
               Hb  56%
               Cl  1.3
               W.B.C's  8200

5th February  R.B.C's  1,560,000
               Hb  56%
               Cl  1.7
               W.B.C's  2600
12th February  R.B.C.s  2,200,000  
            Hb.    60% 
            CI     1.36 
            W.B.C.s 3500  
18th February R.B.C.s  2,140,000  
            Hb.    60% 
            CI.    1.3 
            W.B.C.s 3000  

This patient and another similar case received in addition to other treatment three tumblers a day of milk prepared with fresh lactic acid bacilli cultures. This however had no apparent effect in these cases. The reaction of the faeces remained alkaline. Had the bacilli been established in the intestine one would have expected the reaction to become acid.

THE BLOOD IN HYDATID DISEASE OF THE LUNG.

I investigated the blood in the following case of Hydatid disease of the lung. Carrie Anderson, aged 9, was admitted to Hartlepool's Hospital on 1st January 1908. In the Liver and other organs, except the lungs nothing abnormal could be discovered. There was dulness, diminished breath sounds and slightly increased vocal resonance and fremitus at the base of the right lung. There were also a few crepitations there at the end of inspiration. The rest of the lungs was normal. The patient on four occasions after severe fits of coughing expectorated some filmy material/
material which on examination was found to be
typical laminated Hydatid ectocyst. Frequent search
failed to discover hooklets.
On 3rd January Blood examination showed:-

\[
\begin{align*}
R.B.C.s. & \quad 4,200,000 \\
Hb. & \quad 75\% \\
W.B.C.s. & \quad 12,000 \\
\text{Glycogen Reaction} & \text{well marked.}
\end{align*}
\]

Polymorphs 75\%
Large Lymphocytes 13\%
Small Lymphocytes 11\%
Basophils 1\%

26th January
Polymorphs 60\%
Large Lymphocytes 4\%
Small Lymphocytes 35\%
Basophils 1\%

29th January
Polymorphs 54\%
Large Lymphocytes 7.5\%
Small Lymphocytes 37\%
Eosinophils 1.5\%

In each count 400 leucocytes were counted.
The age of the child probably accounts for the
lymphocytosis. With the rupture of a Hydatid
cyst eosinophilia, as in this case, disappears and
its absence may be taken as showing that there are
not/
not other cysts as yet unruptured, and the progress of a case as regards fresh development can be in this way controlled from blood examination.

Such examples as I have given, all drawn from my own experience illustrate sufficiently to my mind, the value of blood examination in general practice. It is a powerful weapon upon occasion and the possessor of it, who has familiarised himself with its use, has tangible grounds for an added self-confidence and reliance. Precision in diagnosis to which it tends, economises the time of the Practitioner and of the patient and of others more remotely concerned. It supplies facts otherwise unobtainable. Sometimes it supplies the missing link, sometimes but one of several that we cannot find; and at times a link whose place in the chain we cannot assign. To discover a fact, but not its significance acts as a mental stimulus so that one is on the alert to establish connections with it, its reason and its effect. The mystery of the blood appeals to the imagination, it works a charm. By the expectation of discovery we are called to effort. We both pursue an art and utilise a science. We draw from our work the flavour of life.
BOOKS AND PAPERS CONSULTED.

I. Clinical Hematology. Da Costa.

II. The Clinical Examination of the blood. Cabot.

III. Clinical Pathology of the Blood. Ewing.

IV. The Diseases of the blood. Coles.


VI. The Principles and Practice of Medicine. Osler.


VIII. In the Practitioner. Feb. 1903


IX. In the British Medical Journal. 1907

    Anomalous Cases of Pernicious Anaemia
    Dr. Lovell Gulland.

X. Scottish Medical and Surgical Journal, 1902.

    Cyto-Diagnosis in Pleural Effusion.
    Dr. Lovell Gulland.

XI. The Glycogen Reaction in Blood, Its Pathological and Diagnostic Import. 1904. Dr. Lovell Gulland.

XII. British Medical Journal 1907.

    Albuminuria and Renal Disease.
    Dr. Rose Bradford.