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LEUCOCYTOSIS.

BY

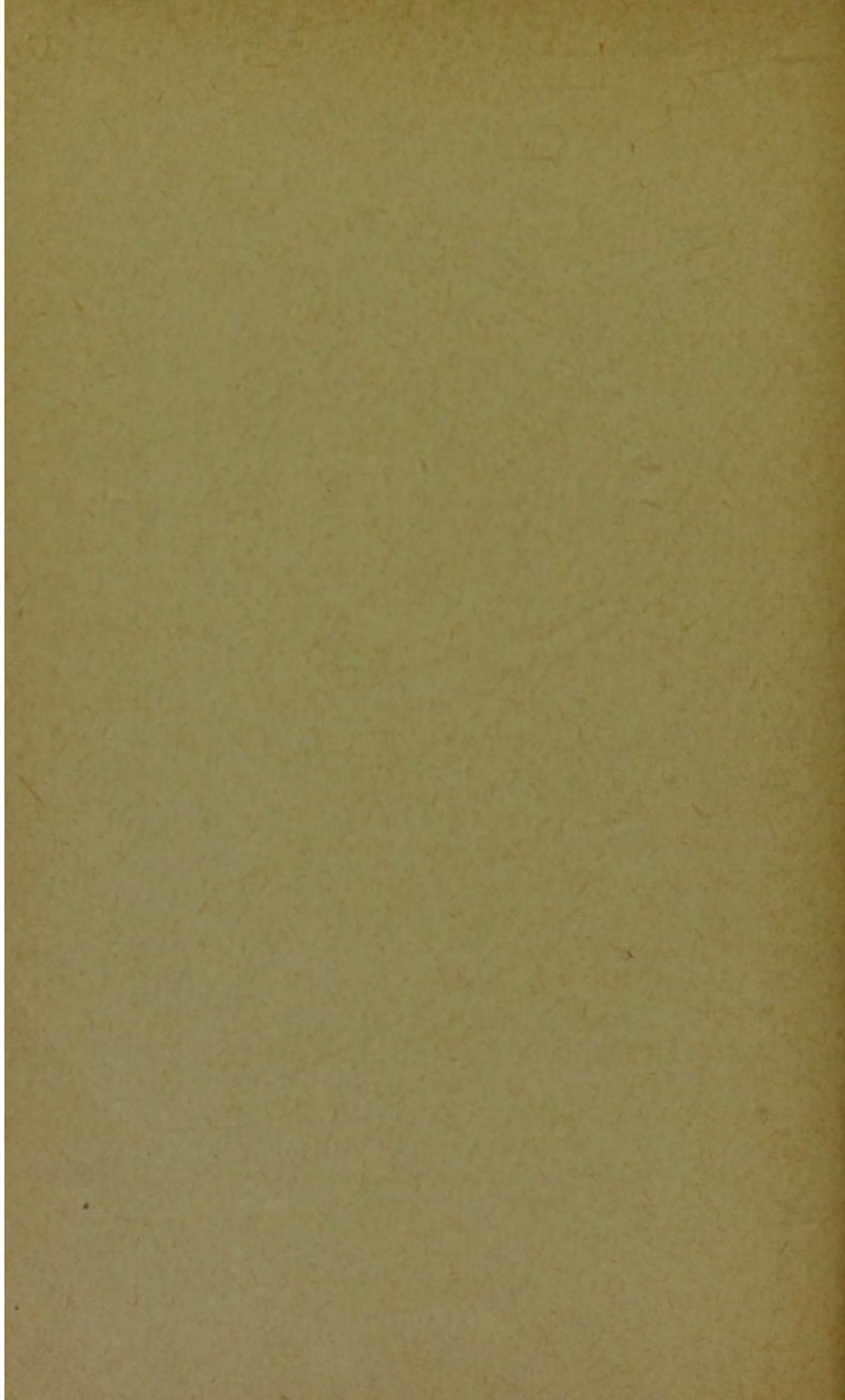
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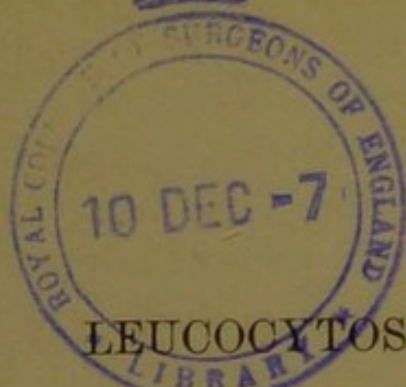
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PERHAPS there is no one factor within the realm of clinical medicine that appeals to the clinician with greater force than that of leucocytosis; for, through the excess, defect, or perversion of the leucocyte, we often provide ourselves with a key whereby many obscure pathological problems may be solved.

It is my intention in this paper to confine myself wholly to the practical aspect of this subject, not entering into the physiological characteristics of the various leucocytes with which you are all so familiar.

In the study of leucocytosis, as it appeals to the practical physician, we have to consider the normal numerical relationship of the different leucocytes, one to the other, the variation of this relationship as effected by infancy, digestion, exercise, massage, cold baths, pregnancy, and parturition. We have next to consider the pathological changes which may take place within the leucocytes, and the variation from the established normal relationship of one type to the other.

We are told that the normal proportions of the various leucocytes are as follows: The small uninuclear

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lymphocytes, from twenty to thirty per cent. The large uninuclear lymphocytes, from four to eight per cent. The polymorphonuclear leucocytes, from sixty to seventy per cent. The eosinophiles, from a half to four per cent. Mast cells, from a quarter to a half per cent. But this relationship is subject to physiological fluctuations—as in infancy the percentage of the small monomorphonuclear lymphocytes varies from forty to sixty per cent., while after a meal rich in proteids the count varies to thirty-three per cent., the increase being manifested proportionally in regard to the various leucocytes, except the eosinophiles, which are diminished. Pregnancy also causes a physiological leucocytosis, with a uniform increase of the various white cells, the average count being thirteen thousand to the cubic millimetre. Violent exercise, massage, cold baths, and parturition also cause physiological leucocytosis.

When considering physiological leucocytosis, we must always take into consideration that we have to deal with individuals, in no two of whom shall we find the processes of metabolism the same, so that what may be physiological with one may be pathological for the other.

En passant, I will here state that after a large number of counts I have found that a fairly accurate average of the normal relationship of the leucocytes may be computed as follows:

Two polymorphonuclear leucocytes to each microscopic field.

One small lymphocyte to every five fields.

One large lymphocyte to ten fields.

One eosinophile to twenty fields.

This estimate, to be of any practical value, necessitates counting the number found in at least twenty-five

fields, or, better, one hundred, and averaging the total amount, and then it will be found somewhat rude, and perhaps not wholly trustworthy; but I mention it, as I have often been asked by physicians how leucocytosis could be estimated without resorting to the fatiguing process of counting by means of a Thoma-Zeiss apparatus, and I think that for practical purposes some reliance may be placed upon the above-stated ratio. This, of course, applies to stained blood.

Pathological leucocytosis differs from the physiological from the fact that in place of a proportional increase of the different leucocytes we find a predominance of one particular kind. Thus we have a leucocytosis proper, in which the increase is manifested with regard to the polymorphonuclear cell; lymphocytosis, in which the small and large lymphocytes predominate; eosinophilia, in which the eosinophiles are proportionately increased; and, lastly, myelocytes, which are almost wholly confined to cases of pernicious anæmia and leucæmia, although occasionally found in grave anæmia, syphilis, and phthisis.

Nearly all the inflammatory and infectious diseases are characterized by leucocytosis, the principal exceptions being typhoid fever, measles, grippe, malaria, tuberculosis, and tuberculous affections.

I have selected the following cases that have fallen under my observation, as demonstrating the value of blood examinations in certain obscure conditions:

L. F., aged forty-seven years; occupation, machinist; was attacked April 23, 1897, with severe pains in the lower right quadrant of the abdomen, with extreme tenderness upon pressure, more marked directly over McBurney's point. He presented a history of having been

suffering from a slight indigestion for several days, although he informed me that he had not been constipated at any time during his indisposition. The pulse was rapid and weak, and the temperature 99° F. I prescribed an opiate to relieve the intense pain from which he was suffering, and directed that a full dose of magnesium sulphate be given, and also an enema. In the afternoon of the same day I found the temperature at 102° and the pulse 120, very weak and compressible. The tenderness was intense and more circumscribed at McBurney's point, and there were also retraction of the abdominal muscles and dullness upon percussion over the involved area. The enema, I was informed, had been effective, he having had a thorough movement. As I thought the case was one of appendicitis, I wished to verify the observations of others in regard to the accompanying leucocytosis; but, much to my surprise, I found a deficient leucocytosis, the examination of the urine at the same time revealing large quantities of indican and colon bacilli. In the afternoon my *confrère*, Dr. Wasse, saw the case with me, and confirmed my diagnosis of appendicitis, and, as all the symptoms were much aggravated at this time, we advised an immediate operation. But before sending for a surgeon we decided, from the result of the microscopic findings, to use a long rectal tube and make sure that there was no impaction. We did so, and were surprised to find our appendicitis transformed into a case of impacted colon.

I have carefully examined the blood in five cases of pneumonia, and in four of them there was a distinct leucocytosis, while in the fifth, where the pneumonia followed measles, there was deficient leucocytosis. In these cases I found that those marked by a distinct increase of the polymorphonuclear leucocytes did the best. One of these cases with leucocytosis was unusually severe, and in this case the increase was made manifest in regard to the old adult neutrophites—*i. e.*, the cells

were large, the nuclei were fragmented and irregularly stained, the cell walls broken in places—in fact, many cells presented themselves as a shapeless mass of broken nuclei and granules, the cell wall seeming to have disappeared. With the first indication of a change for the better in this case, the cells became more distinct, nuclei clear and deeply stained, cell wall distinct, and the cells themselves very small, with well-defined outlines—in fact, they had the appearance of young and normal cells, although somewhat smaller than those usually found in normal blood.

The case with deficient leucocytosis was a very severe one. The polymorphonuclear cells were of immense size, some of them being nearly as large as a myelocyte; they were badly stained, with indistinct cell wall and fragmented nuclei. In this case I detected an appreciable change after the exhibition of nuclein, the cells increasing in number, and being much smaller and more distinct. Another peculiar condition I observed in this case was that with the accompanying degenerated condition of the leucocyte, a careful examination of the urine revealed albumin, granular casts, a distinctly acid reaction, and innumerable colon bacilli, together with a marked diazo reaction.

In three other cases of pneumonia, presenting the so-called typhoid condition, I have found the colon bacilli in large numbers, but in this case I made a bouillon culture and was surprised to obtain a marked reaction with diluted blood, following the technique of the Widal-Johnston reaction. There was complete agglutination in less than a minute. Further investigations of this bacillus proved it to be of the para-colon type described by Widal.

With the reappearance of the young and vigorous neutrophiles all the above conditions disappeared.

My experience with typhoid fever has been very limited, as I have made blood examinations in but two cases, and in both there was deficient leucocytosis, the evolution of the leucocyte being similar to that found in pneumonia.

In tuberculosis I have never been able to verify the observations of Dr. Holmes, of Denver, in regard to the early diagnosis of phthisis by means of degenerative changes within the leucocyte. This may be, and doubtless is, owing to my deficient technique in the preparation of blood smears and staining. In advanced cases I have always found the peculiar degenerative changes he describes, but never in the incipency, before well-marked septicæmia became manifest; but I have in two instances found myelocytes, as mentioned by both Holmes and Cabot.

One of these cases is somewhat peculiar, and with your permission I shall report it:

Millie V., aged fifteen years, had been ill two weeks when I saw her. Her family history was satisfactory, and her previous health had been excellent. She had been under the care of a physician whose diagnosis was typhoid fever. I found her having a morning temperature varying but little from 102° F., while that of the evening was 104°. The pulse varied from 110 to 120, the tongue was dry, and the face flushed; there were also ileo-cæcal gurgling and diarrhœa. The spleen was distinctly palpable; there was a hacking cough, jerky respiration, and a prolonged expiratory murmur over both apices, but no dullness upon percussion. There were no rose-colored spots, but there was a low, muttering delirium. Examination of the urine revealed neither albumin nor casts, but a distinct diazo reaction. Widal-Johnson reaction negative, after many trials. Exam-

ination of the blood revealed red cells normal, deficient leucocytes, the predominant variety of leucocytes being the large uninuclear lymphocyte, and now and then, but by no means frequently, a large myelocyte. This, with the negative Widal-Johnston reaction, led me to think of acute tuberculosis, which was confirmed several days later when a specimen of sputum could be procured.

I am now treating a case which to me is very interesting from its illustration of the clinical significance of lymphocytosis.

Matie R., aged seventeen years, came to Baldwinsville three weeks ago from New York. The patient was a well and healthy girl up to the age of sixteen years, when she began to suffer from very severe headaches. The menses, which had been regular since the age of fourteen, at that time became scanty and irregular. The pain was referred mostly to the eyes and back part of the head. After a time she became unable to walk alone, as she could not coordinate muscular movement, and there was also an absence of patellar reflex. There was at times distortion of the left side of the face, and there was persistent vomiting, although the tongue was perfectly clean and moist. She had lost fifteen pounds during the past month. There was hectic fever, the temperature averaging 100° F. in the afternoon. I noticed that the pupil of the left eye refused to respond to light, while the right seemed normal. There was no cough or expectoration, and a careful examination revealed no chest lesion. An examination of the blood revealed a slight anæmia and a marked lymphocytosis, the small uninuclear lymphocytes predominating. This, together with the loss of vision of the left eye, led me to think of hereditary syphilis. I wrote to Dr. Hirshfield, of New York, who had examined her eyes, and he informed me that she had chorioiditis, the result, he thought, of hereditary syphilis. Both parents stoutly denied ever having had syphilis, but, nevertheless, under large doses of sodium iodide, she is rapidly improving.

