some complication making it necessary to call the physician, the patient usually calls at his office. I have, therefore, frequently found occasion to call at the home of patients when they were not expecting me, for the purpose of ascertaining how nearly they had been carrying out the instructions that had been given them. Such a visit will reveal the nature of the home environment and often explain why convalescence has not been more pronounced.

FACTORS AFFECTING CONVALESCENCE.

The important factors affecting convalescence are:

1. An early diagnosis.
2. An early abandonment of the habits of life, or the environment, by which the patient was surrounded during the development of the disease.
3. The immediate adoption and continued enforcement of rigid rules for healthful living.

The difficulties presented to the tuberculous convalescent in securing remunerative employment is a most important retarding factor. The establishment of remunerative industries in connection with the well-endowed sanatoria may do much toward overcoming the "bread" problem for the great middle class afflicted with tuberculosis.

TREATMENT DURING CONVALESCENCE.

The treatment of convalescence should be directed, first, with the view of correcting temporary symptoms; second, with the object of aiding body nutrition. It may be stated with emphasis that any form of treatment which tends to impair the digestion or assimilation of food, not only can do no good, but on the contrary does absolute harm.

Out-of-door life is pre-eminently the treatment for tuberculosis. Without open-air life convalescence is impossible. The climate which admits of the longest period of uninterrupted open-air life is best suited to the tuberculous patient. That region which admits of the greatest amount of outdoor life, with least discomfort, is the ideal climate. A cool, dry atmosphere is not only more agreeable but less enervating to patients than a hot, dry atmosphere. A tent, or tent-cottage, furnishing a good ventilation without exposing the patient to a draught, one capable of being adjusted to suit all conditions of the weather and all seasons of the year, furnishes the most rational mode of outdoor life, provides all the comforts that a patient requires, and is much less expensive than the indoor treatment.

A few practical points should be carefully noted in connection with the care during the convalescent stage:

Pure air and sunshine can avail but little if the patient is not provided with an abundance of suitable food.

The wisest advice to patients will avail nothing if they are not sufficiently impressed with the importance of proper living to put into practical operation the advice given them. Too often the rigid discipline is lacking in the home. It is chiefly in this respect that the sanatorium treatment surpasses the home treatment of tuberculosis.

CONCLUSIONS.

To know when health is restored to the tuberculous patient is both important and difficult. It is a well-known fact that foci of infection lie concealed in a latent state long after all subjective symptoms have disappeared. Tubercle bacilli may have disappeared from the sputum, or may never have appeared there, and yet there may be and often are concealed foci, active or inactive, which are by no means easy to detect.

To form a correct estimate of convalescence, one must form a composite picture of all the available clinical

evidence. If there is a gradual gain in weight extending over a comparatively long period; if the gain in weight is accompanied by a gain of strength; if the gain of weight and strength are permanent; further, if the cough, sputum and bacilli diminish, and finally disappear, and if this condition is maintained for a comparatively long period, a cure may be considered as practically established.

Finally, if it is evident from the clinical symptoms, physical signs or personal history, that the case was ever truly tuberculous, the conservative physician will use great caution in giving advice which will make it possible to lead his patient too near a danger.

It is safe to state that a person who has had tuberculosis, and who has been sufficiently fortunate to arrive at the advanced stage of convalescence, or even if he had been discharged as "cured," should always exercise care and should never venture too close or remain too long in the environment in which the trouble originally developed.

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THE DIFFERENTIAL LEUCOCYTE COUNT AS AN AID TO THE DIAGNOSIS OF FEVERS.*

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The factors influencing the movements of leucocytes are but imperfectly understood, hence a system of diagnosis based on a variation of the colorless elements in the peripheral blood is not without some hazard, yet it must be admitted that practically all we know of the physiology of the blood has been worked out in the clinical and pathologic laboratory. Stimulated by the reports to the Royal Society and observations of other workers in malarial districts, and feeling guilty of some negligence in this field in the past, I made random observations for some years which led to a systematic investigation of some 200 blood spreads with a view to ascertaining the diagnostic value of the malarial leucocyte variation.

Malarial blood has been found to vary within very wide limits, and the colorless cells show a percentage so nearly like that of typhoid fever, from which we have to differentiate most often, that it did not seem a very promising field, and I so stated at our meeting a year ago, yet, after eliminating the chief sources of error and using the clinical signs as collateral evidence, we can frequently make a positive diagnosis without searching for parasites or waiting for the appearance of the Widal reaction. This method is not to replace, but to supplement, other diagnostic methods.

It is the purpose of this paper to prove that the malarial leucocyte variation does not persist long after all the infection is eradicated, and that it should settle the controversy as to whether it is too sensitive to be used in a malarial district—in other words, that it is not to be thrown aside as of too general occurrence to be worth anything as a test.

In considering any leucocyte variation, it is necessary to have a reasonable idea as to whether this is due to the increase of the one variety or the diminution of another. Is the increase or diminution absolute or relative? What factors other than the disease in question influence it, and to what extent? The accompanying rather

*Read at the Fifty-fifth Annual Session of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. J. M. Anders, Frank Jones and W. S. Thayer.

cumbersome table is adduced as a guarantee, so to speak, as well as to enable any one to criticise the conclusions drawn. The idea that it might be of use as a test that could be made on dried films away from the bedside and in the absence of facilities for culture methods, and that blood films might be exploited in other ways than generally in vogue, is what attracted me in advance of the actual work.

I believe that the enormous increase in large mononuclear cells, especially the very large macrophages, is because phagocytosis is a distinct and easily recognizable phenomenon in malarial infection and is responsible for the occasion of the hibernation of the plasmodia in the interior. We can find pigmented mononuclears after all the parasites have disappeared from the peripheral circulation, but the relative increase of mononuclears persists even longer, and, what is of more importance, is present more uniformly.

An inspection of the tables will show that in malarial infection the large lymphocytes generally nearly equal and frequently outnumber the small, with a total relative increase of lymphocytes and proportionate decrease of polynuclears, except in the beginning of a paroxysm up to about the fastigium, and frequently still later, when there may be a decided absolute and relative increase in polynuclears. Frequently, even here, however, the white cells behave as they do during the afebrile stage. Sometimes there is a marked polynuclear increase without the high fever, but, so far as observed, this is not the case in recent infection, and we generally ascribe this to the effect of quinin, especially in what is called the post-malarial condition.¹ It has so far not been otherwise observed unless a complication exists. I am inclined to regard a polynuclear increase, except under the above conditions, as positive evidence of some other disease, excluding malaria if the large lymphocytes are not in excess of the small. In my table this state of affairs has been found to be due to sepsis, cancer, syphilis with active manifestation, hemorrhage, peritonitis, rheumatism and pneumonia. We would also expect to find it in epidemic meningitis, but no such case has presented itself.

We find, then, that when we have a differential leucocyte count of over 75 per cent. of polynuclears in a presumably malarial patient, in which the large lymphocytes outnumber the small, and during the fever stage, we have a condition which might not be due to the *Plasmodium malariae*; but if due to it, we have a blood in which the parasites would be easily found. If we have a leucocyte count showing upward of 15 large lymphocytes and less than 60 polynuclears, in the absence of an adenopathy and prior to the third week of a fever, neither the presence of the malarial organism nor the absence of a Widal reaction is needed for a diagnosis. In influenza, measles, syphilis, tuberculosis and in typhoid fever after the disease is well established, the count sometimes approaches that of malaria. The leucocyte count is of especial value in determining when the infection has been eradicated in chronic cases, and here it frequently is the only means of diagnosis at our command. In latent malaria, and cachexia, especially when there is no fever, it is the rule to find only 50 per cent. polynuclears, the large lymphocytes being frequently the most numerous element.

In typhoid fever, according to the tables of Thayer, who records nearly 200 counts, the large lymphocytes also increase; but they do not, in my experience, reach

the great size and number found in malaria, and what is more practical, the diagnosis of typhoid fever should be well established after the second week, when this leucocyte confusion is most marked. When the onset of a typhoid is complicated by the lighting up of a latent malaria, the malarial feature overshadows the typhoid; but even here, if in the absence of baths, quinin therapy or other disturbing factor, the polynuclears rise and fall markedly with the fever, typhoid is very probably absent, and the therapeutic test eliminates both the fever and leucocyte variation due to the malaria.

When the diagnosis lies between malaria, typhoid or sepsis, the differential count more certainly decides the question than the absolute leucocyte count or the Widal reaction; the latter is especially fallacious in these cases. (See Table 3, Cases 2, 7, 14 and 31.) In one of these cases (14) there was a pseudo-reaction with Widal, and there appeared to be only a question of typhoid or malaria, but the differential leucocyte count at once excluded both and pointed the way to the diagnosis of malignant endocarditis. In another case, of which the record was accidentally lost, we had to differentiate between a typhoidal pneumonia, pulmonary typhoid, or a mixed infection, when the enormous polynuclear increase, the presence of only 2 per cent. of large lymphocytes positively excluded both typhoid and malaria, although there was marked tympany and abdominal pain. Subsequent events proved that it was a pure lobar pneumonia.

Syphilitic cases seem to show sensitiveness of the leucocyte proportion to the character of the lesion; in adenopathy the large lymphocytes increase, and in bone and skin lesions the eosinophiles appear increased, and when suppuration occurs we have the increase of the total number of white cells, with great preponderance of the polynuclears.

A number of counts in tubercular cases had to be thrown out because the data accompanying the films did not admit of proper classification as to fever, adenopathy, sepsis, etc.

I beg leave now to refer to the tables. Under clinical diagnosis, the preliminary clinical diagnosis is meant, the corrected diagnosis appearing in the last column. Stain No. 1 is fixation in alcohol and ether and staining in eosin and hematoxylin. No. 2, same fixation, stained with eosin and methylene blue in two steps. No. 3, same fixation, stained with eosin and polychrome methylene blue in two steps. Stain 4 is Goldhorn's, which fixes and double stains in one operation, but it has been found desirable to faintly restrain with Loeffler's alkaline methylene blue. No. 5 refers to Ehrlich's method, and No. 6 to Jenner's, which has been found to answer best in inexperienced hands, but does not approach the modified Goldhorn in brilliancy of detail and fidelity of results. The basophile and neutrophile granulation are particularly well brought out. One of the precautions in the Goldhorn method needs to be mentioned, especially if it is desired to restrain with blue, and that is to dilute the stain with good wood alcohol and give it double time, as else the leucocytes may be observed to "get up and leave" when examined under water to see if the contrast is good. There is, unfortunately, a personal factor in the separation of the mononuclear cells, and for this reason I have indicated the name of the observer by an initial letter in one column. I was particularly fortunate in securing the services of Dr. A. G. Love, whose patient and conscientious work represents nearly half of the counts, and almost all of these especially made for this paper. Where the number of cells

1. Memphis Med. Monthly, December, 1901.

TABLE I. WITH LEUCOCYTIC VARIATION—MALARIAL CASES.

No.	Physician	Sex	Clinical Diagnosis	Stage of Illness	Temperature When Taken	Number of Red Cells	Number of White Cells	No. of White Cells Counted	Small Lym.	Large Lym.	Polymorpho- nuclear leu- cocytes	Eosinophiles	Basophiles	Myelocytes	Parasites	By Whom Counted	Stain	Widal Test	Remarks and Corrected Diagnosis
1-17	Average of 17 cases	17 ca	cases of Blackwater fever	by Christophers	and Steph														{ Reports to Royal Society, 1900 and 1901.
18-61	Average of 44 cases	44 ca	cases presumably malarial	by Christophers	and Steph														{ Includes some cases in which leucocyte variation and absence of other disease were the only known factors.
62-79	Average of 28 cases	28 ca	cases of malarial fever previously recorded	by Krauss															{ Had taken quinin: no more given.
80	Krauss	24 f.	malarial fever	2 weeks	none	3,640,000	6,470	200	10.4	23.3	65.3	3.5	1.0	none	not in all cases	C.S.	1		{ Cinchonized; recovered.
81	Krauss	19 m.	typhoid fever	1 week	100°	2,234,000	10,100	300	18.5	14.5	63.5	3.5	1.0	none	no reaction	C.S.	1		{ Had taken quinin: no more given.
82	Krauss	24 f.	same case	2 weeks	103°	4,208,000	3,508	400	14.3	18.4	64.8	2.5	1.0	none	no reaction	K	2		{ Cinchonized; recovered.
83	Rice	39 f.	continued fever	3 weeks	103°			400	6.8	31.0	64.0	1.0	1.0	none	no reaction	K	3		{ Later parasites; cinchonized; recovered.
84	Sutherland	22 m.	malarial hemoglobinuria	5 days	fever	2,342,000	10,200	200	8.0	28.5	53	8	1.0	none	no reaction	K	3		{ Hemoglobinemia; recovered.
85	Harris	16 m.	malarial hemoglobinuria	3 days	102°	1,984,000	6,970	400	10.2	21.0	67.0	2.0	0.2	none	no reaction	K	3		{ No quinin; suppression 3 days; recovery.
86	Krauss	30 m.	malarial hemoglobinuria	2 days	10°	2,162,000	11,400	500	13.0	18.8	68.8	2.0	0.2	none	no reaction	K	3		{ Quinin; recovery (colored boy).
87	Krauss	30 m.	malarial hemoglobinuria	3 days	104°	3,825,000	6,890	400	13.5	23.1	53.3	1.3	1.0	small hyaline	no reaction	K	3		{ Fever and quinin before attack; recovery.
88	Krauss	22 f.	malarial hemoglobinuria	1 day	103°	1,128,000	3,130	200	18.5	30	57.5	0.5	0.5	small hyaline	no reaction	K	3		{ Parasites night before; no quinin; recovery.
89	Sutherland	12 m.	malarial hemoglobinuria	2 days	102°	3,146,000	7,600	300	12.5	17.6	68.3	1.3	0.3	none	no reaction	K	3		{ Suppression third day; urine flowed sixth day; hicough; cinchonized 7 p.m. sixth day; died midnight.
90	Krauss	52 m.	malarial hemoglobinuria	1 day	102°			270	12.2	39.6	45.5	2.6	0.2	few	no reaction	K	4		{ Had been heavily cinchonized when hemoglobinuria appeared; recovered.
91	Stanley	46 m.	malarial hemoglobinuria	3 days	103°	1,432,000	12,084	600	9.0	6.6	82.8	2.0	0.2	1.0 very many	no reaction	K	4		{ Quinin; recovery; 5 days.
92	Stanley	46 m.	malarial hemoglobinuria	6 days	102°	2,234,000	6,720	500	10.2	12.8	76.2	0.4	0.2	0.2 none	no reaction	K	4		{ No quinin; suppression 3 days; recovery.
93	Stanley	46 m.	malarial hemoglobinuria	2 days	102°	1,820,000	6,720	300	10.66	29.66	57.3	2.3	0.8	tertian	no reaction	K	4		{ Quinin; recovery (colored boy).
94	Krauss	30 m.	malarial hemoglobinuria	2 days	100°	2,170,000	4,533	524	7.3	30.7	60.3	76	1.1	tertian	no reaction	K	4		{ Parasites night before; no quinin; recovery.
95	Krauss	30 m.	malarial hemoglobinuria	3 days	100°			350	11.8	33.9	53.5	0.8	0.2	tertian	no reaction	K	4		{ Suppression third day; urine flowed sixth day; hicough; cinchonized 7 p.m. sixth day; died midnight.
96	Krauss	22 m.	typhoid fever	2 weeks	104°	2,170,000	4,533	550	7.6	18.1	73	0.9	0.2	tertian	no reaction	K	4		{ Had been heavily cinchonized when hemoglobinuria appeared; recovered.
97	Jones	18 m.	malarial fever	2 weeks	fever			200	14.5	22.5	58.5	3	1.0	many tertian	no reaction	L	4		{ Quinin; recovery; 5 days.
98	Jones	18 m.	malarial fever	1 week	fever			200	14.5	22.5	58.5	3	1.0	many tertian	no reaction	L	4		{ Quinin; recovery; 5 days.
99	Jones	18 m.	malarial fever	1 week	fever			200	14.5	22.5	58.5	3	1.0	many tertian	no reaction	L	4		{ Quinin; recovery; 5 days.
100	Krauss	26 m.	malarial cachexia	some weeks	100°	2,492,000	2,789	400	25.2	30.0	39.8	3.0		many tertian	no reaction	L	4		{ Spleen 12 x 13 inches; had hematuria 2 weeks previous; parasites in splenic blood; every leucocyte on spread counted.
101	Jones	24 f.	malarial fever	2 weeks	103°			400	7.0	11.25	81.5	25		small tertian	no reaction	L	4		{ Taken 6 hours after chill; had taken quinin.
102	Krauss	20 m.	malarial cachexia	some weeks	100°	3,020,000	2,168	310	13.2	27.4	56.4	2.9	0.9	small tertian	no reaction	L	4		{ Spleen enlarged since last August.
103	Jones	18 m.	malarial fever	1 week	104°	2,310,000	6,500	400	8.75	14.0	70.0	1.0	0.2	small tertian	no reaction	L	4		{ Had taken quinin 2 days every 3 hours.
104	Krauss	27 m.	malaise	1 week	none			250	22.6	24.0	50.0	3.4		few	no reaction	K	4		{ Felt "bilious" for 1 week.
105	Jones	40 m.	feels well	3 days	none			250	16.4	29.0	71.6	4		many tertian	no reaction	K	4		{ Not included in totals (to show control).
106	Jones	40 m.	malarial fever	3 days	fever			400	11.0	16.0	66.7	6.3		many tertian	no reaction	L	4		{ Chill on 26th and 28th.
107	Jones	40 m.	slight fever	3 days	fever			230	15.2	19.1	65.6			many tertian	no reaction	L	4		{ Chills on 3 consecutive days.
108	Jones	40 m.	malarial fever	1 week	fever			240	9.6	17.0	72.0	0.9	0.5	many tertian	no reaction	L	4		{ Chills every other day; no quinin.
109	Jones	24 f.	malarial fever	3 days	fever			500	9.4	14.6	75.0	0.4	0.6	many tertian	no reaction	L	4		{ Chill 6 hours ago.
110	Krauss	30 m.	congestive malarial fever	6 days	05°			600	11.3	28.3	59.2	1.2		very many tertian	no reaction	K	4		{ Cyanosed; finger blood black; 3 chills before admittance; quinin hypodermically; recovery.
111	Krauss	29 m.	malarial fever	3 weeks	100°	3,265,000	8,770	250	27.2	25.6	44.8	2.4		estivos	no reaction	L	4		{ Enlarged spleen.
112	Krauss	42 m.	typhoid fever	3 weeks	101°			400	12.0	19.0	65.0	4		many estivos	no reaction	L	4		{ Like typhoid, but decidedly malarial next morning; broad tongue; quinin; recovery.
113	Turner	28 m.	typhoid fever	3 days	101°			300	9.0	21.8	68.3	0.9		estivos	no reaction	L	4		{ Diagnosed malaria; quinin; recovery.
114	Jones	28 m.	malarial fever	3 days	101°			300	7.3	17.6	75.0			estivos	no reaction	L	4		{ Had taken large doses quinin in last 24 hrs.
115	Jones	45 m.	"backache"	few days	101°			200	11.5	15.5	63.5	9.5		estivos	no reaction	L	4		{ Claimed backache from "overwork;" admitted having chill on 10th.
116	Jones	50 m.	chills	some time	101°			400	15.0	28.6	54.0	3.3		estivos	no reaction	L	4		{ Chills every other day.
117	Krauss	50 m.	malarial fever	some time	101°			500	36.6	24.6	37.0	1.4	0.4	estivos	no reaction	L	4		{ Very weak; enlarged spleen; recovery slow.
118	Krauss	45 m.	malarial fever	some weeks	100°			130	24.6	33.1	43.3	4.6	0.3	many estivos	no reaction	K	4		{ Had been at Tampico, Mexico; home on sick leave; is up and about.
119	Krauss	45 m.	malarial fever	some weeks	100°			300	11.0	21.3	60.6	0.3	0.3	many estivos	no reaction	K	4		{ Parasites, intracellular and extracellular.
120	Jones	30 f.	malarial fever	some weeks	101°			300	11.0	21.3	60.6	0.3	0.3	many estivos	no reaction	K	4		{ Parasites, intracellular and extracellular.
121	Jones	30 f.	malarial fever	some weeks	101°			300	11.0	21.3	60.6	0.3	0.3	many estivos	no reaction	K	4		{ Symptoms indefinite.
122	Jones	10 f.	fever	some time	fever			300	38.6	23.3	36.0	1.6	0.3	many	no reaction	L	4		{ Many crescents.
123	Jones	8 f.	malaria	some time	fever			200	31.0	23.4	44.5	1.5		many	no reaction	L	4		{ Had taken quinin; nearly well.
124	Jones	28 f.	malaria	some time	fever			400	20.2	18.2	59.0	2.6		many	no reaction	L	4		{ Had taken quinin; nearly well.
125	Jones	28 f.	malaria	some time	fever			210	18.3	14.1	59.6	8		many	no reaction	L	4		{ Many crescents and intracellular parasites.
126	Jones	28 f.	malaria	some time	fever			300	17.3	33.0	49.6	3.6	0.5	many	no reaction	L	4		{ Came from Miss. Co., Ark.; had taken quinin; nearly well.
127	Jones	28 f.	latent malaria	some time	fever			300	20.6	14.6	61.0	3.6	0.5	many	no reaction	L	4		{ Came from Miss. Co., Ark.; had taken quinin; nearly well.
128	Jones	28 f.	latent malaria	some time	fever			200	12.5	14.0	72.5	0.4	0.5	many	no reaction	L	4		{ Came from Miss. Co., Ark.; had taken quinin; nearly well.
129	Jones	28 f.	malaria	some time	fever			500	11.4	20.8	66.8	1.6	0.4	many	no reaction	L	4		{ Came from Miss. Co., Ark.; had taken quinin; nearly well.
130	Krauss	50 m.	fever	3 weeks	100°			300	10.3	30.6	58.6	0.3		few	no reaction	K	4		{ Pleuritic friction sound; fear tuberculosis.
131	Jones	10 m.	same patient	104°	104°	3,800,000	2,260	100	11.0	12.0	77.0			none	no reaction	L	4		{ Dullness left axillary line.
132	Jones	10 m.	same patient	103°	103°			225	34.2	44.0	21.8			few	no reaction	L	4		{ Consolidation left lower lobe; recovery.
133	Jones	10 m.	same patient	104°	104°			300	8.3	23.0	68.0	3.0	0.3	none	no reaction	K	4		{ Chills every other day.
134	Jones	10 m.	same patient	103°	103°			400	5.2	6.4	86.4	1.5	0.5	none	no reaction	K	4		{ Chills every other day.

*135 Jones	16 m.	malaria fever.	some weeks	none	200	25.0	22.0	13.0	many	L	4	no reaction	Few dumb chills; ward case.
*136 Krauss	27 m.	latent malaria.	some weeks	fever.	500	26.0	17.8	54.4	few	K	4	no reaction	No chills; malaria exposure for some weeks.
*137 Jones	1 m.	latent malaria.	1 week	fever.	400	10.0	12.5	77.0	0.2	L	4	no reaction	
*138 Jones	1 m.	latent malaria.	1 week	fever.	400	10.0	13.2	77.0	0.2	L	4	no reaction	Trachoma; headache; bilious.
*139 Jones	1 m.	latent malaria.	1 week	fever.	400	10.0	13.2	77.0	0.2	L	4	no reaction	Malaise; coated tongue; quinin.
*140 Krauss	24 m.	latent malaria.	1 week	fever.	300	19.6	33.0	66.7	0.5	L	4	no reaction	Take ill in Africa; small (boss).
*141 Krauss	30 m.	latent malaria.	3 weeks	fever.	270	9.1	18.1	72.8	0.5	L	4	no reaction	Chills; malaria; enlarged spleen; no parasites in spleen blood.
*142 Krauss	30 f.	latent malaria.	6 weeks	100°	820	24.4	27.8	47.8	0.5	L	4	no reaction	Recovery on quinin.
*143 Jones	45 f.	"fever"	1 week	fever.	200	13.0	16.0	71.0	0.5	L	4	no reaction	Had large doses of quinin.
*144 Krauss	2 m.	malaria fever.	2 weeks	fever.	300	3.6	11.3	84.3	0.6	L	4	no reaction	
*145 Jones	3 m.	chills.	3 days	none.	500	23.3	19.0	54.0	0.3	L	4	no reaction	Found 2 parasites in 2 hours.
*146 Jones	6 m.	chills daily	3 weeks	fever.	500	16.3	29.7	53.4	0.3	L	4	no reaction	
*147 Jones	1 m.	chills	1 week	fever.	300	18.0	42.6	59.0	0.3	L	4	no reaction	
*148 Jones	1 m.	chills	1 week	fever.	400	7.8	15.5	76.2	0.2	L	4	no reaction	
*149 Jones	1 m.	chills	1 week	fever.	200	26.0	15.0	56.5	0.5	L	4	no reaction	
*150 Krauss	21 m.	malaria cachexia	3 weeks	102°	400	8.8	14.2	75.5	1.2	L	4	no reaction	Very large spleen last year; crescents and many half grown.
*151 Jones	30 m.	chills.	some weeks	none.	300	19.0	45.0	55.6	0.4	L	4	no reaction	Very cachectic.
*152 Turner	30 m.	continued fever	2 weeks	100°	200	12.0	28.0	59.0	0.5	L	4	no reaction	Malaria; confirmed by quinin.
*153 Jones	1 m.	continued fever	1 week	100°	300	36.3	22.3	41.3	1.5	L	4	no reaction	Malaria; confirmed by quinin.
*154 Jones	1 m.	malaise	1 week	100°	200	8.0	19.0	72.5	1.5	L	4	no reaction	Has taken quinin.
*155 Krauss	23 m.	malaria cachexia	5 weeks	100°	300	14.0	26.3	57.6	2.0	L	4	no reaction	Third attack; spleen 8 x 10 inches.
*156 Jones	3 m.	chills	some time	chill.	300	13.5	20.5	63.5	2.0	L	4	no reaction	
*157 Jones	3 m.	chills	3 weeks	chill.	300	9.3	17.3	71.0	3.0	L	4	no reaction	Has chill now; tertian.
*158 Jones	3 m.	chills	3 weeks	chill.	300	12.4	22.6	61.0	1.0	L	4	no reaction	
*159 Krauss	46 m.	latent malaria	8 weeks	none	300	35.6	21.3	41.3	1.6	L	4	no reaction	Desultory cinchonization.
*160 Krauss	46 m.	malaria infection	8 weeks	none	300	27.6	29.0	43.3	0.3	L	4	no reaction	Desultory treatment; 2 weeks.
*161 Holder	31 f.	fever	some weeks	none	200	20.0	21.0	58.7	0.3	L	4	no reaction	
*162 Jones	31 f.	chills	some weeks	none	210	31.4	21.9	45.2	1.5	L	4	no reaction	
*163 Jones	31 m.	chills	some weeks	fever.	400	9.8	15.0	75.2	0.5	L	4	no reaction	
*164 Jones	31 m.	malaria	some weeks	none	400	25.0	24.0	51.0	0.5	L	4	no reaction	(Only one parasite found; diagnosed on blood count.)
*165 Braun	26 m.	malaria hemoglobinuria	3 days	fever	300	9.3	16.3	74.3	0.5	L	4	no reaction	Whites counted from "ret" pipette.
*166 Sutherland	26 m.	malaria hemoglobinuria	4 days	101°	300	39.0	23.6	37.4	0.5	L	4	no reaction	Had chills for some weeks.
*167 Jones	6 m.	tertian malaria	1 week	102°	400	11.5	16.5	72.0	0.5	L	4	no reaction	
*168 Jones	6 m.	tertian malaria	1 week	102°	400	11.5	16.5	72.0	0.5	L	4	no reaction	
*169 Krauss	24 f.	malaise	few days	none	200	17.6	43.0	39.3	0.5	L	4	no reaction	Very slight infection.
*170 Jones	40 m.	fever	2 weeks	fever.	220	22.2	12.2	65.4	0.5	L	4	no reaction	Sent for diagnosis; possible sepsis; quinin; recovery.
*171 Krauss	28 m.	fever	5 weeks	103°	200	7.5	20.0	68.0	4.5	L	4	no reaction	
*172 Jones	a. m.	malaria	some weeks	fever	300	6.0	12.3	81.7	0.5	L	4	no reaction	Sporulation; gametocytes.
*173 Jones	a. m.	chills	some weeks	none	600	7.6	9.3	83.0	0.5	L	4	no reaction	
*174 Krauss	32 f.	debility	some weeks	none	300	28.0	41.0	30.3	0.6	L	4	no reaction	History chills some weeks; enlarged spleen.
*175 Jones	38 m.	malaria fever	1 week	103°	200	13.3	23.6	57.7	5.4	L	4	no reaction	Loss of flesh; malaise; quinin and arsenic; recovery.
*176 Krauss	38 m.	malaria fever	1 week	103°	200	4.6	19.0	74.3	1.6	L	4	no reaction	Double infection; spleen very much enlarged
*177 Turner	38 m.	malaria fever	6 weeks	101°	120	12.5	17.5	68.3	1.66	L	4	no reaction	Cinchonism marked.
*178 Jones	a. m.	malaria	2 weeks	none	300	9.0	10.33	80.6	0.5	L	4	no reaction	Had no chills; fever subcontinuous.
*179 Krauss	25 m.	malaise	2 weeks	100°	200	24.5	40.0	32.5	3.0	L	4	no reaction	Anemic and aching.
*180 Jones	25 m.	malaise	2 weeks	none	170	23.5	16.4	58.8	1.2	L	4	no reaction	Rigid abdomen; pain region spleen; abdomen retracted; crescents and intracapsular bodies.
*181 Jones	36 m.	fever	some time	none	130	33.1	29.2	36.2	1.6	L	4	no reaction	No treatment.
*182 Krauss	36 m.	malaise	1 week	none	400	27.2	25.2	47.6	0.5	L	4	no reaction	Bilious; feels badly; cinchonized.
*183 Jones	36 m.	malaise	1 week	none	800	11.0	15.6	72.3	1.0	L	4	no reaction	Sick some weeks; no chills.
*184 Jones	36 m.	fever	some time	fever.	400	27.2	25.2	47.5	0.5	L	4	no reaction	
*185 Goltman	36 m.	fever	some time	fever.	200	4.5	15.0	78.0	2.0	L	4	no reaction	
*186 Moore	36 m.	malarial fever	1 week	fever.	200	4.5	15.0	78.0	2.0	L	4	no reaction	
*187 Jones	36 m.	malarial fever	3 weeks	fever.	320	23.1	31.5	39.7	0.6	L	4	no reaction	
*188 Jones	36 m.	chills	2 days	none	300	9.5	16.7	70.5	3.2	L	4	no reaction	
*189 Jones	26 m.	chill last night	2 weeks	103°	200	3.0	12.0	84.6	0.3	L	4	no reaction	
*190 Bramwood	f.	malaria hemoglobinuria	2 weeks	fever.	300	6.0	14.0	80.0	0.5	L	4	no reaction	Enlarged spleen; lived in bottom for years; mitral murmur; casts; albumin; no fever; no quinin.
*191 Holder	a. m.	malaria cachexia	indefinite	none	200	8.0	38.0	52.0	2.0	L	4	no reaction	
*192 Shelby	6 m.	continued fever	2 weeks	103°	500	7.4	28.5	63.8	0.2	L	4	no reaction	Quinin hypod.; recovery; diagnosis on leucocyte count.
*193 Goltman	1 1/2 m.	fever	1 week	103°	400	2.2	17.0	80.8	0.5	L	4	no reaction	Plethoric; bronzed; history of repeated fevers; fragmented leucocytes; quinin; recovery.
*194 Goltman	21 m.	suspected hepatic abscess	indefinite	100°	300	12.3	27.6	58.3	1.3	L	4	no reaction	No quinin; ordered comp. quinin pills.
*195 Krauss	25 m.	malaise	1 week	none	300	10.3	13.6	74.0	1.0	L	4	no reaction	
*196 Jones	25 m.	malaise	long time	none	400	13.7	18.7	67.3	3.5	L	4	no reaction	
*197 Jones	b. m.	malaria cachexia	indefinite	none	200	10.0	24.0	60.0	6.0	L	4	no reaction	

TABLE I. WITH LEUCOCYTIC VARIATION—MALARIAL CASES—Continued.

No.	Physician	Age	Sex	Clinical Diagnosis	Stage of Illness	Temperature When Blood Taken	Number of Red Cells	Number of White Cells	Small Lym-phocytes	Large Lym-phocytes	Polymorpho-nuclear leu-cocytes	Eosinophiles	Basophiles	Myelocytes	Parasites	By Whom Counted	Stain	Widal Test	Remarks and Corrected Diagnosis.
*198	Jones	40 m.	m.	malaria	1 week	102°		230	5.6	9.2	85.2	10.0			present	L	4		
*199	Jones	40 f.	m.	malaria cachexia				160	4.0	20.0	66.0	10.0			present	L	4		
*200	Jones	40 m.	m.	malaria	some weeks			200	11.0	30.5	58.5	2.5			present	L	4		
*201	Jones	40 m.	m.	chills				200	10.5	19.5	67.5				present	L	4		
*202	Jones	40 m.	m.	malaria	fever			300	5.5	10.5	84.0				present	L	4		
*203	Jones	40 m.	m.	malaria	fever			200	10.0	26.0	64.0				present	L	4		Chill yesterday.
*204	Kincaid	40 m.	m.	malaria	slight			300	18.3	14.3	67.0	0.3			present	K	4		Gradually rising fever, malaise.

Total mean of 204—Small lymphocytes, 14.8; large lymphocytes, 19.5; polymorphonuclear leucocytes, 63.7; eosinophiles, 2.0.
(Numbers with asterisk (*) were counted expressly for this study.) (The dagger (†) represents plus.)

TABLE II. MIXED CASES.

No.	Physician	Age	Sex	Clinical Diagnosis	Stage of Illness	Temperature When Blood Taken	Number of Red Cells	Number of White Cells	Small Lym-phocytes	Large Lym-phocytes	Polymorpho-nuclear leu-cocytes	Eosinophiles	Basophiles	Myelocytes	Parasites	By Whom Counted	Stain	Widal Test	Remarks and Corrected Diagnosis.
1	Krauss	40 m.	m.	pernicious anemia			3,104,000	12,300	24.0	12.0	54.0			10.0	none	K	3		Megaloblasts, polikilocytes; history malar- ial fever, spleen enlarged.
2	Goltman	40 f.	m.	pernicious anemia			1,083,330	8,652	500	15.5	10.0	73.0	1.0	0.5	none	K	4		History chronic malaria; purpura; died.
*3	Turner	18 m.	m.	typhoid fever	4 weeks			502	22.1	11.2	66.0				none	L	4		Typical typhoid; widal not tried before.
*4				typhoid fever	5 weeks			411	19.3	32.3	48.4				many tertian	L	4		Remittent curve; diagnosis intermittent malaria.
*5	Krauss	15 m.	m.	typhoid fever	4 weeks			300	34.6	14.6	50.0	0.8			none	L	4		Typical picture; not tried before this test.
*6				typhoid fever	5 weeks	104°		441	8.6	12.0	79.4				many tertian	L	4		Melapse; intermittent malaria; perfora- tion; death.
7	Krauss	35 m.	f.	fever	6 weeks	fever		9,945	400	9.5	78.0	1.5	0.5		estivo-autumnal	L	4		Fever; liver enlarged upwards; cough; diagnosis hepatic abscess.
8	Krauss	1 1/4 m.	f.	fever	3 weeks	104°		?	32.0	17.4	50.4				none	K	4		Indigestion, malaise, quinin; recovery.
*9	Krauss	25 m.	m.	typhoid fever	3 weeks	104°		200	10.0	6.5	83.5				none	K	4		Chill before taking of blood.
*10	Krauss			typhoid fever	10 1/2	105°		210	23.3	20.4	56.2				estivo-autumnal	K	4		Septic curve; recovery uneventful.
*11	Krauss	22 m.	m.	inflammatory rheumatism	2 weeks	fever		600	11.6	9.0	78.3	0.8	0.2		few	K	4		Complicating malarial infection; quinin.
*12	Krauss			chills	3 days	100°		300	15.0	6.0	79.0				none	K	4		Ulcerated proctitis; colitis; (syphilis?)
*13	Jones	m.	m.	chills	3 days	none		600	23.3	19.2	54.3	3.3			many small	L	4		(Amt. leucocytosis and slight fever with no quinin; history taken to indicate pus; appendicitis later.
*14	Krauss	40 m.	m.	intestinal obstruction	3 days	fever		400	6.0	18.2	75.8				few	K	4		Malarial infection and syphilis.
*15	Jones	f.	f.	pain all over	3 weeks	fever		400	13.7	12.0	73.0	1.0	0.2		few	L	4		Double infection.
*16	Krauss	30 m.	m.	continued fever	3 weeks	fever		400	23.5	14.0	62.0	0.5			estivo-autumnal	L	4		Reported as malaria on this blood count;
*17	McCown	22 m.	m.	leukemia	some weeks	slight	4,025,000	5,323	300	16.3	17.3	64.3	1.0	1.0		none	K	4	fever some weeks; recovery.
*18	Jones	36 f.	f.	salpingitis		fever	4,870,000	15,500	500	7.0	9.4	83.4		0.2		present	L	4	Malarial organisms searched for and found on this leucocyte variation.
*19	Jones	25 f.	f.	syphilis				300	17.6	21.4	59.6	1.4			present	L	4		Headache every other day; diagnosis; syn- chills and malaria.
20	Baldwin	16 f.	f.	typhoid fever	5 weeks	102°		300	30.3	31.6	38.0				many estivos	L	4		Convalescent from typhoid in cottage un- screened; no bar; spleen enlarged; su- peradded malaria.
*21	Jones	m.	m.	hypertrophic cirrhosis		fever	3,600,000	19,100	300	8.6	13.6	77.6			many	L	4		History of syphilis also.
22	Goltman	20 m.	m.	fever	2 days	none		200	12.0	31.0	52.0	4.0	1.0		two groups	K	4		Decorative case; also crescents; much fever.
23	Shelby	10 m.	m.	slow fever	2 weeks	fever		400	17.2	15.0	67.5	0.2			5 large estivos	K	4		Double mixed infection.
24				fever	4 weeks	fever		250	25.0	10.8	63.0	1.0	0.2		3 small pigments	K	4		Old infection complicating convalescence.
25	Holder	20 f.	f.	latent appendicitis	6 weeks	none	4,878,000	5,460	400	21.5	24.7	52.8	1.0		present	K	6		Bitten by mosquitoes, 150 miles south of Memphis.
26	Blackburn	m.	m.	rheumatism	3 weeks	fever		300	10.0	8.6	81.3				present	K	6		Suspected typhoid; no quinin; no further typhoid symptoms.
*27	Jones			rheumatism	3 weeks	fever	2,614,000	7,400	15.5	15.5	62.5	6.5			present	L	4		Endocarditis.

Average per cent.—Small lymphocytes, 17.5; large lymphocytes, 15.8; polymorphonuclear leucocytes, 65.4; eosinophiles, 0.9; basophiles, 0.2; myelocytes, 0.4.
(Those marked with a * were taken expressly for this study.)

TABLE III. LEUCOCYTE COUNT—CONTROL CASES.

No.	Physician	Sex	Clinical Diagnosis	Stage of Illness in Years, Months and Weeks	Temperature When Taken	Number of Red Blood Corpuscles	Number of White Cells	No. of White Cells Counted	Small Lym.	Large Lym.	Polymorpho-nuclear leucocytes	Eosinophiles	Basophiles	Myelocytes	Parasites	By Whom Counted	Stain	Widal Test	Remarks and Corrected Diagnosis.
1	Smyth	30 m.	typhoid fever.	3 weeks	102°	3,484,000	7,600 cover glass leucocy-tosis	300	20.3	8.3	69.3	1.60.3	...	...	none	K...	3	no reaction	Spinal myelitis.
2	Goltman	m.	malaria or typhoid	...	...	...	...	300	6.3	2.3	91.0	0.3	...	...	none	K...	3	no reaction	Malignant endocarditis.
3	Goltman	f.	anemia	...	...	416,000	...	200	12.0	1.5	65.5	13.0.2.0	6.0	none	none	K...	3	...	{ Polkioleocytes; megaloblasts purpura; per-nicious anemia; death. { Leukemia; marked purpura; spleen en-larged; skin, lemon yellow; hemorrhage { nose, stomach and bowels.
4	Saunders	16 f.	leukemia	6 weeks	slight	347,000	56,600	280	46.4	16.1	37.1	0.40.4	...	...	none	K...	3	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
5	...	...	...	9 weeks	slight	...	11,200	400	31.2	7.2	47.0	13.0.1.6	...	...	none	K...	5.2	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
6	Hall	20 m.	leukemia	6 weeks	slight	3,808,000	17,300	500	21.0	4.5	73.0	1.00.5	4.0	none	none	K...	3	no reaction	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
7	Mickie	44 m.	gallstones	3 years	slight	4,294,000	6,200	300	22.3	6.6	69.0	2.0	...	...	none	K...	4	no action	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 8	Jones	35 f.	asthma	...	...	...	...	500	22.6	21.6	37.6	17.60.3	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 9	Jones	40 f.	syphilis (sec.)	...	...	3,105,000	8,190	400	22.7	10.3	58.7	7.50.8	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 10	Krauss	41 f.	malaria	...	none	3,458,000	6,550	550	20.1	8.9	70.9	0.1	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 11	Jones	f.	rheumatism	...	fever	2,328,800	10,420	520	13.5	7.2	76.5	2.30.5	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 12	Black	30 m.	typhoid fever	3 weeks	102°	...	...	200	19.2	8.8	72.0	...	...	...	none	L...	4	positive	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 13	Turner	30 m.	typhoid fever	2 weeks	103°	...	...	200	20.5	23.0	56.5	...	...	...	none	L...	4	positive	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 14	Venn	56 m.	typhoid fever	2 weeks	102°	...	18,000	500	12.4	4.2	81.2	2.0.2	...	...	none	K...	4	pseudo	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 15	Jones	31 m.	acting	...	...	...	...	400	14.4	7.2	78.2	0.2	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 16	Jones	34 m.	marasmus	some weeks	...	6,000,000	9,600	100	30.0	19.0	51.0	...	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
17	Krauss	28 m.	sepsis	some months	...	...	14,040	?	9.2	12.7	73.9	2.31.1	0.5	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
18	Krauss	25 m.	adenopathy	indefinite	none	6,420,000	15,680	300	14.3	12.0	72.6	1.0	...	...	none	K...	4	positive	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
19	Krauss	30 m.	suspected syphilis	2 months	none	7,800	...	400	8.24	10.5	80.24	0.80.24	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
20	Krauss	35 m.	nodule liver	indefinite	99°	3,988,400	9,450	400	15.0	14.5	69.0	1.00.5	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
21	Krauss	60 m.	blood poison	indefinite	none	3,860,000	11,520	200	11.0	18.0	68.0	2.01.0	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
22	Krauss	47 m.	pneumonia	2 weeks	101°	4,120,000	12,600	400	16.7	15.3	66.7	1.3	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
23	Krauss	20 m.	syphilis	6 weeks	none	3,982,000	12,170	400	14.8	8.4	74.2	2.6	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
24	Krauss	30 m.	syphilis	8 weeks	none	4,561,000	6,430	300	18.0	24.0	49.0	4.02.0	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
25	Goltman	7 m.	measles	...	fever	...	...	300	13.6	16.4	66.3	3.00.7	...	...	none	K...	6	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
26	Goltman	3 f.	exposed measles	...	103°	...	...	200	7.5	9.5	81.5	2.00.5	...	...	none	K...	6	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
27	Venn	60 m.	gastric cancer	long time	100°	1,265,400	5,760	500	7.4	7.8	83.8	0.80.2	...	...	none	K...	6	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 28	Jones	m.	enlarged lymphatics	...	...	3,290,000	30,000	500	4.6	6.4	89.0	...	...	...	none	K...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 29	Holder	m.	pyemia	3 weeks	fever	3,030,000	16,000	200	14.5	5.0	80.5	...	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
* 30	Jones	m.	ankylostomiasis	...	fever	2,820,000	10,000	200	10.0	5.6	73.4	11.0	...	...	none	L...	4	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.
31	Krauss	30 m.	tubercular peritonitis	6 months	101°	3,678,400	10,340	400	13.0	12.3	72.0	2.5	...	...	none	K...	6	...	{ Enlarged glands; fever; diagnosis; Hodg-kin's disease. { History colic; operation; adhesions; death. { Hypertrophied heart; granular casts; diag-nosis, renal asthma.

Total 31 controls—Small lymphocytes, 14.26; large lymphocytes, 8.10; eosinophiles, 74.49; polymorphonuclear leucocytes, 0.40; myelocytes, 0.05.
Normal average—Small lymphocytes, 20; large lymphocytes, 8; eosinophiles, 1.5; basophiles, 0.5.
(The dagger (†) represents plus.)

SUMMARY:

Small Lym-phocytes	Large Lym-phocytes	Polymorpho-nuclear	All Others
15.4	21.6	61.3	1.7
14.8	19.5	63.7	2.0
10.0	17.0	70.0	...
17.0	23.0	49.0	...
21.5	29.7	46.4	1.9
18.0	15.0	64.0	1.5
14.3	8.1	74.0	2.8
12.9	12.4	74.0	0.5
14.6	13.4	70.9	0.8
21.5	11.6	66.3	0.3
20.1	14.4	65.0	0.4

Mean of last 125 cases.
Total mean.
Thirty cases of malaria with over 102° fever.
Ten cases during apyrexia.
Twenty-five cases malarial cachexia (temperature under 101°).
Twenty-seven cases disease plus malarial infection.
Thirty-one control cases, presumably without malaria.
Thayer's cases typhoid fever:
First week.
Second week.
Third week.
Fourth week.

counted is under 300, it was due to the scarcity of cells in the spread, the whole film having been gone over with a mechanical stage.

In referring again to Table 3 we find three cases very probably malarial (8, 16, 24). They were positively exposed to it in proper season, in surroundings which precluded protection, although no parasites were found on the spreads. Of the balance, 11 cases had over 10 per cent. of large lymphocytes, and only 5 of these reached the obligate 14 per cent. of Christophers and Stephens. These were:

	Per cent.
One suspected secondary syphilis, with ulcers....	18
One hepatic gumma with peritonitis.....	14
One leukanemia.....	16.1
One grippal pneumonia.....	15
One typhoid fever, third week.....	23

With the exception of the latter, they all had a marked leucocytosis, none had marked fever when film was taken, and the only question unanswered and unanswerable was, whether they had an associated malarial infection.

CONCLUSIONS.

1. It is not so much the absolute increased per cent. of large lymphocytes which is diagnostic of malarial infection as the relative increase over the small lymphocytes.

2. In cases of malarial infection without much fever and without quinin history, the polynuclears are markedly diminished and the large lymphocytes very much increased in proportion.

3. In the absence of an adenopathy, possibly also of influenza and measles, the above finding is positive evidence of present or very recent malarial infection.

4. During the rise of a malarial fever, or as a result of quinin therapy, the polynuclear leucocytes may reach 80 per cent., but in that event the large lymphocytes exceed in number the small ones.

5. In malarial hemoglobinuria any of the various blood findings characteristic of malarial fever may be present in any stage, and there is no evidence now at hand to explain this.

6. Malarial and typhoid fevers can be more positively differentiated from fevers of sepsis, pneumonia, rheumatism, malignant tumors, etc., by the differential leucocyte count than by the hemocytometer, and it can be done on dried films, away from the bedside.

7. Malarial infection makes a strong impress on the leucocytic relation, no matter with what other disease it is associated.

8. It is not safe to rule out typhoid fever when the malarial variation is found, but, as it calls for anti-malarial medication, the therapeutic test may decide before a Widal reaction would appear.

9. In a gradually developing fever, the absence of marked increase in large lymphocytes or polynuclears during the first week justifies a preliminary diagnosis of typhoid fever.

DISCUSSION.

DR. RICHARD CABOT, Boston—What criterion is used in the classification of blood cells for a large and what for a small mononuclear? No two men agree, and no two men are likely to observe the same rules in regard to this point, and this uncertainty makes the results of Dr. Krauss and the English school less available. At the same time I am influenced toward the conviction that what he has given us will be of value in confirming the diagnosis of malaria. I am not convinced that it will take the place of the direct observation of malarial parasite, because the same amount of work required to demonstrate the percentage of large mononuclear cells would usually suffice to demonstrate the parasite itself. What has most in-

terested me in the paper is its tendency to prove the central importance of the examination of the film specimen; this implies that the count of the red cells is less important, and in many cases is practically of little value. I seldom count the red cells. What we should count is the white corpuscles, and especially note their differential relation to each other. If we know what actually can be obtained from the film specimen (dried on glass and stained) we know all that will give information of value as regards the diagnosis in four-fifths of the cases; nothing more in many cases could be learned from an actual count of the cells.

DR. ROBERT N. WILLSON, Philadelphia—Unless I have misunderstood his statement I think that there is an error in the assertion of Dr. Krauss that a high percentage of polymorphonuclear cells in the blood excludes malaria and typhoid fever from the diagnosis. I have personally had the privilege of studying a large number of cases of both typhoid fever and malaria with reference to the blood picture, and in common with many other observers have repeatedly found a high percentage of these cells in the presence of complicating conditions. In such cases, unless this fact is remembered, the count will hinder rather than help toward a proper diagnosis. One particular case which I have reported was that of a former member of this Association, a physician who died of perforation of the bowel in typhoid fever. In this instance the polymorphonuclear percentage remained almost constantly about 9 per cent., and was highest at the time when the total leucocyte count was at its lowest point (6,400). In one or two other cases of typhoid perforation, proved by autopsy, there was the same occurrence. One case seen recently, however, was of typhoid complicated by cystitis, in which there was a polymorphonuclear count of only 54 per cent., in spite of the presence of quantities of pus in the bladder and urine. With regard to the statement made by both Drs. Cabot and Krauss that in certain cases the malarial organisms failed of discovery because quinin had been given to the patient, it is interesting to note that in the Philadelphia Hospital I have studied a number of cases in which the parasite could not be discovered after a very careful search until after quinin had been given. This experience was shared by a number of other observers in the same hospital, and has extended over several years time.

DR. C. R. GRANDY, Norfolk, Va.—It often happens in my section of the country that the people will take quinin before coming to the physician for a diagnosis, and, therefore, in many of these cases a blood examination is made more difficult. I wish Dr. Krauss would state distinctly if he has found an increase of large lymphocytes in such cases.

DR. W. E. ROBERTSON, Philadelphia—In the consideration of the differential leucocyte count, the influence of age is an important factor. As is well known, the younger the child the more pronounced the leucocytosis. Then too, up to the fifth year of life there is a marked lymphocytosis, the polynuclears being proportionately deficient.

DR. WILLIAM KRAUSS—The difference between a large and a small mononuclear cell is an arbitrary one. The best way is to class all the cells the size of a polynuclear cell as small unless the protoplasmic margin is relatively large and contains scattered neutrophile granules, which stamps the cell as a "large" one. In refutation of Dr. Cabot's contention, English observers have found the leucocyte count a great saving of time in differentiating latent malaria and when plasmodia are absent in peripheral blood. If you make a slide spread a differential leucocyte count can be made in 20 minutes at the far edge of the film, whereas it might take two hours' patient search to find a parasite, and, even if found, it furnishes no information as to the possible presence of a complication. The polynuclears may be increased in malaria, but the relation of the lymphocytes is not interfered with, and when found at the height of a fever, the polynuclear increase points rather definitely to presence of malaria and absence of typhoid. The best evidence of the real value of this diagnostic measure is that in the tables, in which the diagnosis had to be bolstered up by all the collateral evidence available in order to prove my point—it was frequently the sole sign post to a correct diagnosis. The

answer to Dr. Willson's question appears in the tables. In answer to Dr. Grandy I would say that here it was that I found my best field; in persons cinchonized, the question: Should they not be further cinchonized? is best answered by the malarial lymphocyte variation, as it is more sensitive than the presence of plasmodia or pigmented leucocytes. As to Dr. Robertson's question, in children over one year old the variation is usually reliable.

PHYSIOLOGIC OPTICS.

NEW EXPERIMENTS FOR DEMONSTRATION AND NEW APPARATUS.*

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When it was proposed to establish a laboratory course in the physiology of vision, the head of the department of ophthalmology was requested to state what he wished students to know in this field when they presented themselves in his department for clinical work on the eye. The reply was in effect this:

Before the student can enter on the clinical study of the eye he should be acquainted with: the anatomy of the conjunctiva; the lachrymal apparatus, including the drainage and the extrinsic muscles; the circulation, extrinsic and intrinsic; the nerve supply of the muscles, the mechanism of muscle action, the gross and minute anatomy of the coats of the eyeball; the refractive media, the retinal circulation, and the ciliary apparatus. He should thoroughly understand the functions of all the parts studied. The student can not hope to succeed in clinical ophthalmology unless he has a clear understanding of the refractive media of the eye from an optical standpoint: the indices of refraction; the law of lenses and their influence on the path of a ray of light; in short, the dioptric system of the eye. He must understand the mechanism of accommodation and be able to determine its range; he must be able to outline the field of indirect vision for form and color, and to map the distribution of the retinal vessels through ophthalmoscopic examination of models and of the eyes of animals and of normal man.

In the development of a course to cover the required ground, several new appliances and methods have been devised to facilitate the laboratory instruction, and I take this opportunity to make a brief description of the more important of them together with diagrams showing their structure.

In these optical appliances three requirements are met: accuracy, simplicity and economy.

TO DETERMINE THE INDICES OF REFRACTION OF WATER AND OF GLASS.

A very convenient and sufficiently exact apparatus for making the required determination may be easily made as follows:

Take a carpenter's tri-square, constructed wholly of iron, from the angle x (Fig. 1), where the graduated limb joins the body, measure off centimeters on the inner surface of the body and cut them in with a file.

Locate on the inner edge of the graduated limb any point as y , 6 to 9 centimeters from the point. With files remove about one-half centimeter of the edge as indicated in figure, cutting deeply at z , so as to leave a slender point at y , as indicated.

Drill a hole in the inner surface of the body at o ; fit and drive a heavy brass or iron wire into this; sharpen the upper end of the wire. The length of the wire above the body must be two or three centimeters, greater

than the distance xy . Bend the point over so that distance op shall equal xy .

Place the instrument in the water pan; fill the pan, so adjusting it that both points p and y will just fail to touch the water, for the surface of the water at y must be absolutely plane. If the points touch it the surface will not be plane.

Bring a small rule, r , into position and clamp it to the limb of the instrument by means of heavy serre-fine forceps. So adjust the rule that as one sights along its upper edge the points a , y and z seem to lie in the same straight line. Lift the apparatus out of the water and lay it on the table, taking care not to disturb the adjustment.

With dividers measure the distance from the point y to line z . This is the radius. Determine the point where the circumference would cut the upper surface of the rule, say point b .

From this point determine the perpendicular distance to the edge of the limb at c .

Fig. 1.

The line cyx is a normal to the surface of the water at the point y . The angle i is the angle of incidence; the angle r is the angle of refraction. Imagine a circle whose center is at y and circumference passes through b and z . The line bc is the sine of the angle of incidence. The line xz is the sine of the angle of refraction.

DETERMINE THE RATIO OF SINE I TO SINE R.

In the same manner determine the ratio of these angles when the rule is also adjusted as to bring ayz or $ay4$ apparently in one straight line.

If the instrument has been carefully constructed, and if the determination has been made with sufficient care, the ratio, $\text{sine } i : \text{sine } r$, will be found to be practically equal to the ratio, $\text{sine } i : \text{sine } r$; furthermore, the ratio for any substance such as water, is found to be constant. This constant ratio is called the index of refraction, and is conventionally represented by μ .

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