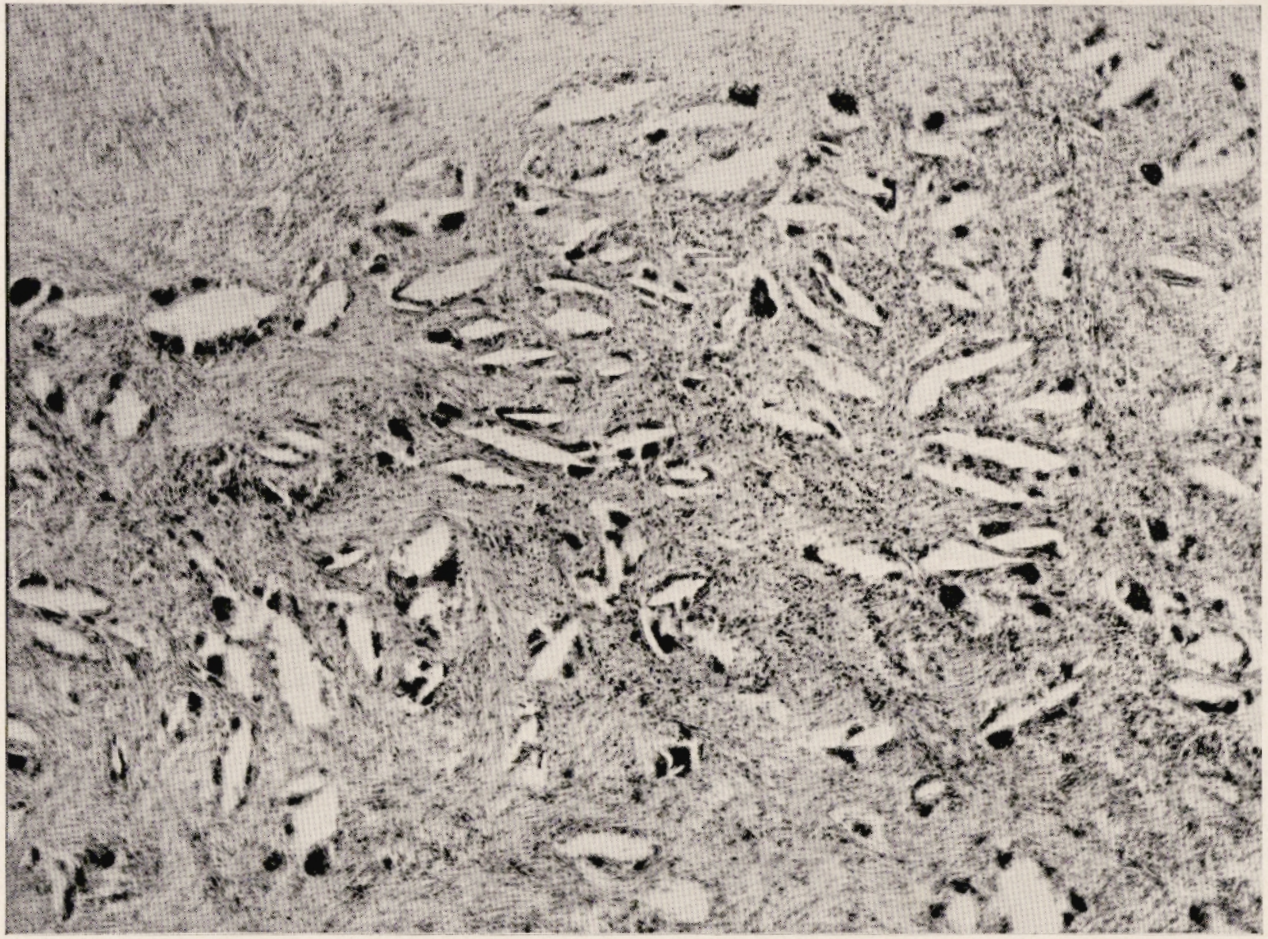


numbers around and in the capsule of the gland explains blockage of the flow of lymph to the glands and therefore the mechanism of the obstructive manifestation of the infection.

The most characteristic histological changes in filariasis are the preponderance of lymphocytes as compared with other cells and the ultimate development of fibrosis.

Local Changes:—Lymphatic glands in the vicinity of degenerating filariae showed great variation in histology. Frequently the capsule was thickened and contained dilated lymphatics and sclerosed blood vessels. In others there was also dilatation of the cortical sinuses and the lymphatics of the medulla. In more advanced cases the fibrous tissue of the medulla was considerably increased and invading the cortex, replaced in varying degrees the germ centers which were, in some instances, reduced to small islets of lymphoid tissue. In still other instances the structure within the capsule consisted of a large lymph varix surrounded by narrow bands of apparently normal lymphoid tissue.

Testicle, Epididymis and Cord:—When many living and some degenerated worms are present the cord is enlarged, soft and doughy. In the region of the individual worms there is often a localized enlargement which should be considered of clinical importance and removed in cases associated with hydrocele. Histologically, such tissues show considerable dilatation of lymphatics with moderate interstitial fibrosis. Under similar conditions the epididymis is enlarged, soft and irregular. Careful palpation in the living subject may reveal the presence of a cyst or, rarely, several cysts in the epididymis.

When the condition is of long standing and especially when there are many degenerated worms resulting in obliteration of many lymphatic vessels, fibrosis of the tissues marks the dilatation of lymphatic vessels. Nevertheless in sections a considerable number of dilated lymph vessels may be present in the fibrous matrix. In these cases nodules in the cord are generally hard and may be of almost a cartilaginous consistency. Cysts of the epididymis in these cases may reach considerable dimensions and are not uncommonly mistaken for hydroceles. In some, the contents of the cyst,

especially following hemorrhage, tend to become solidified. In three cases when fluid was not withdrawn following tapping of a supposed hydrocele, the condition turned out to be a large cyst of the epididymis. Such large cysts have generally very thick hyalinized capsules. From study of some twenty cases the sequence of changes resulting in cyst of the epididymis where *W. bancrofti* are more commonly found, seems to be, dilatation of the lymphatic vessels, hypertrophy following dilatation, the development of definite varices, further thickening of the walls of the vessels with collapse of others and finally, formation of the cyst. In most of the cases of cyst observed in these studies hydrocele was not present. The testicle was flattened or elongated and the visceral and parietal layers of the tunica vaginalis were in contact. In a few cases however hydroceles were present. Parasites in greater or lesser numbers are generally present in the immediate vicinity of the cyst wall. The changes in the blood vessels show a relation to the duration of the foregoing conditions and the number of worms present independent of the age of the patient. Marked thickening in some cases, almost occlusion of the blood vessels in others, has been observed in comparatively young people showing advanced genital filariasis but with no evidence of generalized arteriosclerosis. Even when the spermatic cord and epididymis are extensively involved in filarial pathology the testicles are rarely enlarged in the absence of some other intercurrent infection.

Hydroceles in Puerto Rico exhibit every variation from small translucent bladders of fluid to dense, thick-walled, non-fluctuating sacs containing large deposits of calcium. In most of the cases which were sectioned calcified parasites were found either in the spermatic cord or upper part of the epididymis and more commonly in both situations.

Observations on Parasites in Sections:—Since immature worms were only rarely found in the tissues examined it seems possible that the parasites reach maturity within a short time after entry into the human host. The sex of all the worms was observed except when fragmentation in instances of advanced calcification made this difficult. Unfortunately, however, complete records of sex were not made

in the earlier examinations. In 169 instances however where such records were made there were 96 females and 73 male parasites.

Of 162 mature *W. bancrofti*, living or dead, found in the external genitalia of the male, 38 were in the spermatic cord, 111 in the epididymis and at its junction with the testis and 13 in the testis proper. The parasites in the testis were generally placed immediately inside the tunica albuginea but a few were found in the middle of the organ. All these parasites were in the connective tissues between the seminiferous tubules and in no instance were the tubules affected. In 13 cases in which *W. bancrofti* were found in the male external genitalia, spermatogenesis was active in nine. Of the remaining four cases two were lepers, while in a third necrosis, surrounded by massive fibrosis in the epididymis, was probably responsible for atrophy of the testis. The absence of spermatogenesis in the fourth case cannot be explained. In none of these four cases was syphilis excluded. The findings suggest that in the majority of males infected with filariae, the presence of the parasite does not interfere with fecundity.

In no case were parasites found in the tubules of the epididymis. There were however in a few instances calcified concretions which might be mistaken for parasites.

In five cases in which the testicle, epididymis and spermatic cord of both sides were available for study, out of a total of 80 parasites discovered, 33 were on the right side and 47 were on the left.

In four cases (Table 3 Cases 1, 6, 7, & 13) six, two, five and four recently living female *W. bancrofti* were found. In each of these cases the intra-uterine development of ova and microfilariae was identical. Extended microfilariae filled the vagina and a varying extent (for different cases) of the anterior part of the uterine tubes; behind them coiled embryos were found while posteriorly ova in varying degrees of differentiation were discovered.

Microfilariae in the Tissues:—In Case 1 (Table 3) microfilariae were found in lymphatics of the connective tissue of the testis, in the tunica albuginea and at the reflection of the tunica vaginalis of the hydrocele sac. Living and dead

embryos were present (Fig. 20). They were also found in granulation tissue formed along the endothelial lining of the lymphatics in these situations. At the angles of the hydrocele large numbers of calcified microfilariae were found in granulation tissue which consisted of lymphocytes, plasma cells, giant cells and fibroblasts. In this case also microfilariae, living or in varying stages of degeneration, were found in the afferent lymphatics, cortical sinuses and along the trebeculae of the inguinal glands (Figs. 21, 22). In these glands there were many small areas of necrosis, especially in the neighborhood of some of the calcified embryos. No embryos were found in the efferent lymphatic vessels. In this case and in another since studied where the larger viscera were available, microfilariae were numerous in the kidney. In this organ the largest numbers were found in the glomeruli (Fig 23), less commonly between the tubules while they were rare in the pyramids. In two cases (one of which was an accidental death) who died about midnight, microfilariae were scanty in any large vessel but were fairly common in small capillaries, especially in loose areolar tissue between muscle fibers or surrounding organs. They were much more numerous in these situations than in the capillaries of the skin. They were not very numerous in the vessels of the lungs.

It is worth noticing that of the four cases in which living female *W. bancrofti* were found, microfilariae were only discovered in the blood vessels of two, despite the fact that hundreds of sections were studied. It seems probable, therefore, that in some instances the embryos produced by the female do not eventually reach the blood stream.

HEMATOLOGY

Throughout these studies uniform methods were used in making blood examinations. Hemoglobin was estimated by the Sahli method. The total red and white blood cell counts were made with the Thoma Zeiss apparatus. The differential leucocyte count was based on the percentages of two hundred cells; it was made usually between the hours of ten and eleven thirty in the morning in order to eliminate the possibility of a fallacy arising from increased lympho-

embryos were present (Fig. 20). They were also found in granulation tissue formed along the endothelial lining of the lymphatics in these situations. At the angles of the hydrocele large numbers of calcified microfilariae were found in granulation tissue which consisted of lymphocytes, plasma cells, giant cells and fibroblasts. In this case also microfilariae, living or in varying stages of degeneration, were found in the afferent lymphatics, cortical sinuses and along the trabeculae of the inguinal glands (Figs. 21, 22). In these glands there were many small areas of necrosis, especially in the neighborhood of some of the calcified embryos. No embryos were found in the efferent lymphatic vessels. In this case and in another since studied where the larger viscera were available, microfilariae were numerous in the kidney. In this organ the largest numbers were found in the glomeruli (Fig 23), less commonly between the tubules while they were rare in the pyramids. In two cases (one of which was an accidental death) who died about midnight, microfilariae were scanty in any large vessel but were fairly common in small capillaries, especially in loose areolar tissue between muscle fibers or surrounding organs. They were much more numerous in these situations than in the capillaries of the skin. They were not very numerous in the vessels of the lungs.

It is worth noticing that of the four cases in which living female *W. bancrofti* were found, microfilariae were only discovered in the blood vessels of two, despite the fact that hundreds of sections were studied. It seems probable, therefore, that in some instances the embryos produced by the female do not eventually reach the blood stream.

FIG. 20. Microfilariae in lymphatic vessel.

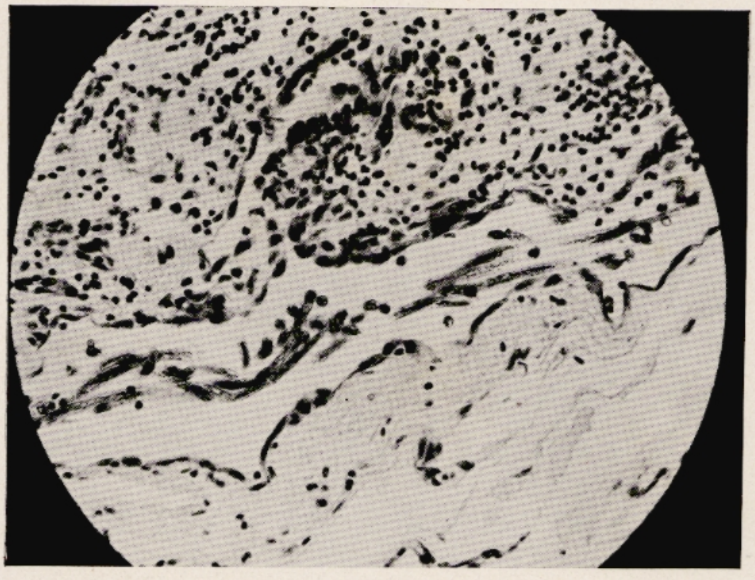
GRABADO 20. *Microfilarias en un vaso linfático.*

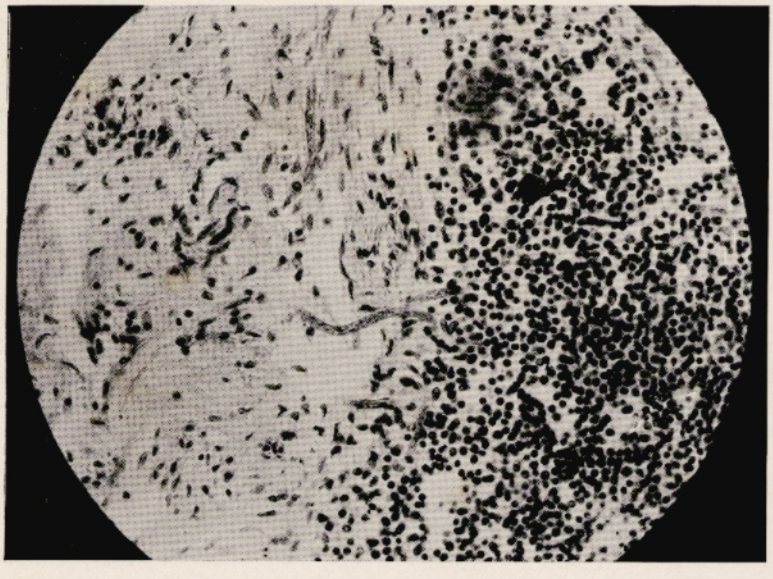
FIG. 21. Right inguinal glands. Microfilariae leaving the afferents for the inguinals.

GRABADO 21. *Ganglio inguinal derecho. Microfilarias saliendo de los vasos aferentes y pasando a los inguinales.*

HEMATOLOGY

Throughout these studies uniform methods were used in making blood [illegible] white blood cell counts were made with the Thoma Zeiss apparatus. The differential leucocyte count was based on the percentages of two hundred cells; it was made usually between the hours of ten and eleven thirty in the morning in order to eliminate the possibility of a fallacy arising from increased lympho-





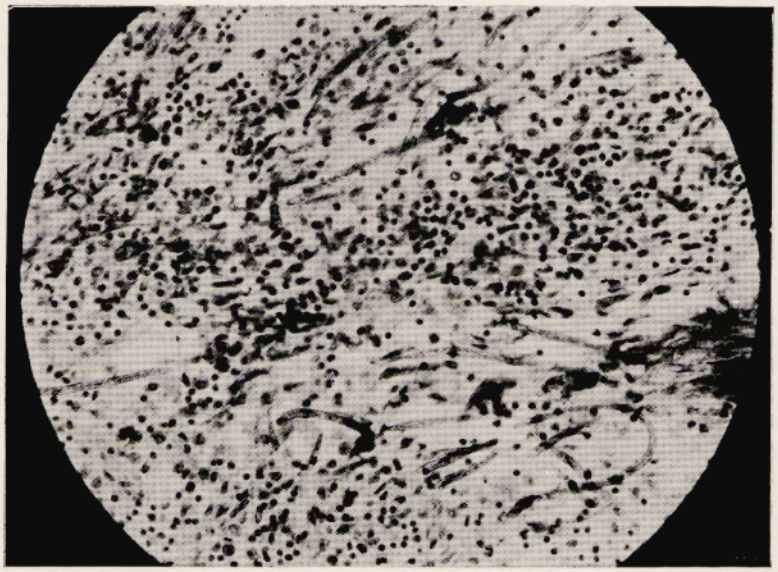
FILARIASIS O'CONNOR AND HULSE

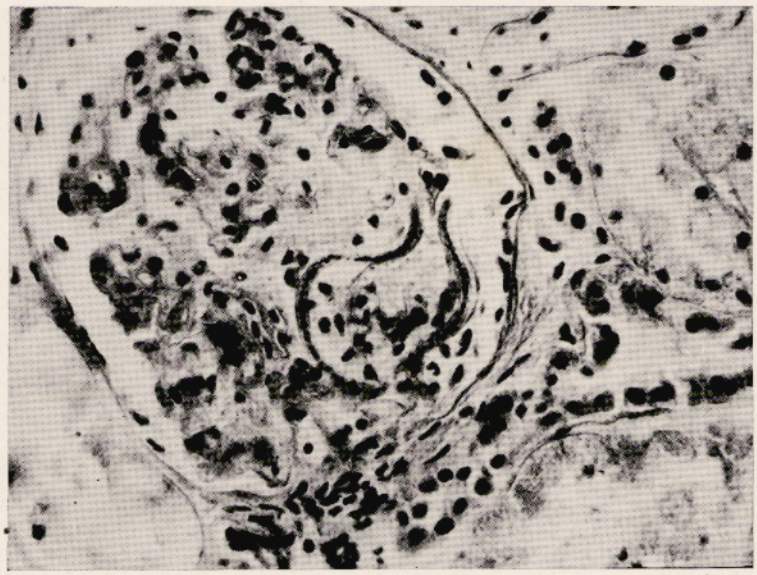
FIG. 22. Right inguinal glands. Microfilariae degenerating in sinuses along the trebeculae.

GRABADO 22. *Ganglio inguinal derecho. Microfilarias en proceso de degeneración, situadas en los espacios entre las trabéculas.*

FIG. 23. Microfilariae in the glomerulus of the kidney.

GRABADO 23. *Microfilarias en los glomérulos del riñón.*





FILARIASIS O'CONNOR AND HULSE

cytosis in association with digestion. Schilling's [27] estimate of the relative percentage of the different leucocytes normally present in the blood was used as a basis, namely:

Polymorphonuclear leucocytes	67.0 (54–72)
Lymphocytes	23.0 (21–35)
Eosinophils	3.0 (2–4)
Monocytes (large mononuclears, transitional forms)	6.0 (4–8)
Basophils or mast cells	0.5 (0–1)

The findings in these studies were similar to those of Rose [28], namely, that neither the hemoglobin index nor the total red and white cell counts are affected in uncomplicated infection by *W. bancrofti.* Interesting variations in the differential leucocyte count were observed however, in the different phases of filarial infection.

DIFFERENTIAL LEUCOCYTE COUNTS ON PERSONS AT THE TIME SUFFERING FROM ACUTE INFLAMMATORY ATTACKS.—In the blood of forty-six persons with clinical manifestations of filariasis, but no acute inflammatory symptoms, the percentage of polymorphonuclear leucocytes was below normal. The percentage of lymphocytes was high however, the average for the group being 39.9 per cent. If the four cases of chyluria are excluded from the group, the average percentage of lymphocytes in the group is 40.5. Taking 8 per cent as the upper limit of normality for eosinophil cells, only sixteen of the forty-four patients showed eosinophilia, and all of these, with two exceptions, were under forty years of age; 46 per cent were under thirty years of age.

Chyluria Cases:—The elimination of the four chyluria cases from this group seems indicated because it has been generally observed that while chyluria is present the lymphocytes are noticeably decreased in number. This is to be expected in a condition which drains the channels along which these cells progress from the source of production to the blood. Remlinger [29] cites two cases of chyluria in which the differential blood counts were: polymorphonuclears 19 per cent in one, 17 per cent in the other; lymphocytes 3 per cent in both; eosinophils 70 and 75 per cent; large mononuclears 8 and 5 per cent. In a series of eight cases, four from Puerto Rico and four from other countries, which were studied by

us in New York, the total leucocyte and differential counts were as follows:

TABLE 4.—*Differential Leucocyte Count in Eight Patients With Chyluria*

Patient	Leucocytes	Polymorphonuclears (percent)	Lymphocytes (percent)	Eosinophils (percent)	Large Mononuclears (percent)	Mast Cells (percent)
A. P.	7,000	86.5	9.5	1.5	2.0	0.5
C. C.	5,760	51.[illegible]	42.0	[illegible].0	4.0	0
J. H.*	7,200	48.5	23.0	26.0	2.5	0
A. D.	6,120	69.0	18.0	12.0	0	1.0
B. F.	4,160	79.0	11.0	4.0	5.0	1.0
R. D.	5,760	78.0	17.0	2.0	3.0	0
R. C.*	9,320	69.5	19.5	7.0	3.5	0.5
F. E.	7,640	83.0	13.0	1.0	3.0	0

*Microfilariae in both the blood and the urine.

As in other series, the eosinophil cells are in excess in some of these cases, but this is by no means the rule. In one case the lymphocytes are high; in another they are normal; but in the majority they are distinctly reduced.

DIFFERENTIAL LEUCOCYTE COUNTS ON SEVEN PERSONS WITH MICROFILARIAE IN THE BLOOD BUT NO CLINICAL MANIFESTATIONS OF FILARIASIS.—The number of persons in this group, Table 5, is not sufficient to justify conclusions, but the uniformly high lymphocyte incidence in this as compared with that of the group in Table 4 indicates the importance of further studies in persons of various ages. It is not surprising that most of the persons are young, since it is in youth that the microfilarial incidence is the highest whether with or without symptoms.

CLINICAL CASES WITH ELEPHANTIASIS OR WITH A HISTORY OF RECURRENCES OF LYMPHANGITIS OR ADENITIS, OBSERVED DURING THE INFLAMMATORY REACTION, THROUGHOUT CONVALESCENCE AND FOR A VARYING TIME THEREAFTER.—Rose [28] presented a series of tables indicating leucocyte response during and after inflammatory reactions in filariasis. In Table 1 of his series were listed ten cases showing lymphocyte response at the expense of the polymorphonuclear cells; in Table II were six which he described as "cases showing increase of polymorphonuclears at the expense of the lymphocytes." In only one instance were the former cells increased above the normal count, but the lymphocytes were in all cases definitely

below the normal. In one case in this group there was an abscess in process of formation; one was a case of lymphangitis lasting three weeks; while a third was a case of lymphangitis with abscess formation, and free discharge of pus. Table III showed two cases in which the balance between polymorphonuclear cells and lymphocytes seemed to be held. On the basis of his observations he concluded:

> The first result of the onset of an attack is a relative increase in the polymorphonuclear cells.
>
> Favorable progress is accompanied by a relative increase in the lymphocytes apparently at the expense mainly of the neutrophil cells.
>
> A relative increase in lymphocytes is also found in subacute and chronic cases.
>
> A relative increase in the polymorphonuclears is found, as might be expected, in abscess formation, and also in all cases at and before the climax of the attack and in severe cases for some days after subsidence.

TABLE 5.—*Blood Picture of Persons With Microfilariae But Without Symptoms*

Patient	Age	Sex	Hemoglobin (Per cent)	Total Red Blood Cells	Total White Blood Cells	Polymorphonuclears (percent)	Lymphocytes (percent)	Eosinophils (percent)	Large Mononuclears (percent)
M. R.	14	F	80	3,400,000	8,440	37.5	50.0	11.5	1.0
L. C.	6	F	75	5,290,000	5,440	38.0	55.0	6.5	0.5
A. C.	8	F	60	4,300,000	7,720	30.0	64.0	6.0	0
S. M.	12	F	80	4,590,000	9,760	51.0	42.0	7.0	0
J. G.	13	F	78	4,750,000	7,500	23.5	66.5	8.0	2.0
E. C.	55	M	85	4,200,000	10,440	38.5	46.5	15.0	0
A. R.	14	F	85	4,660,000	9,560	50.5	45.0	3.0	1.5

Findings in the present investigation are in agreement with these views. Fourteen cases were carefully studied, beginning as soon as possible after the onset of the inflammatory attack and continuing as long as possible after convalescence had been established; in all the blood was examined daily for at least a week. Some of the results are recorded in Charts XI, XII, and XIII.

The degree of leucocytosis usually shows some relation to the severity of the attack, but there may be fairly high leucocytosis with comparatively mild constitutional disturbance. Yet the leucocytes do not normally reach the numbers usually associated with conditions due to septic organisms. The leucocytosis diminishes rapidly and suddenly in some cases.

Chart XI

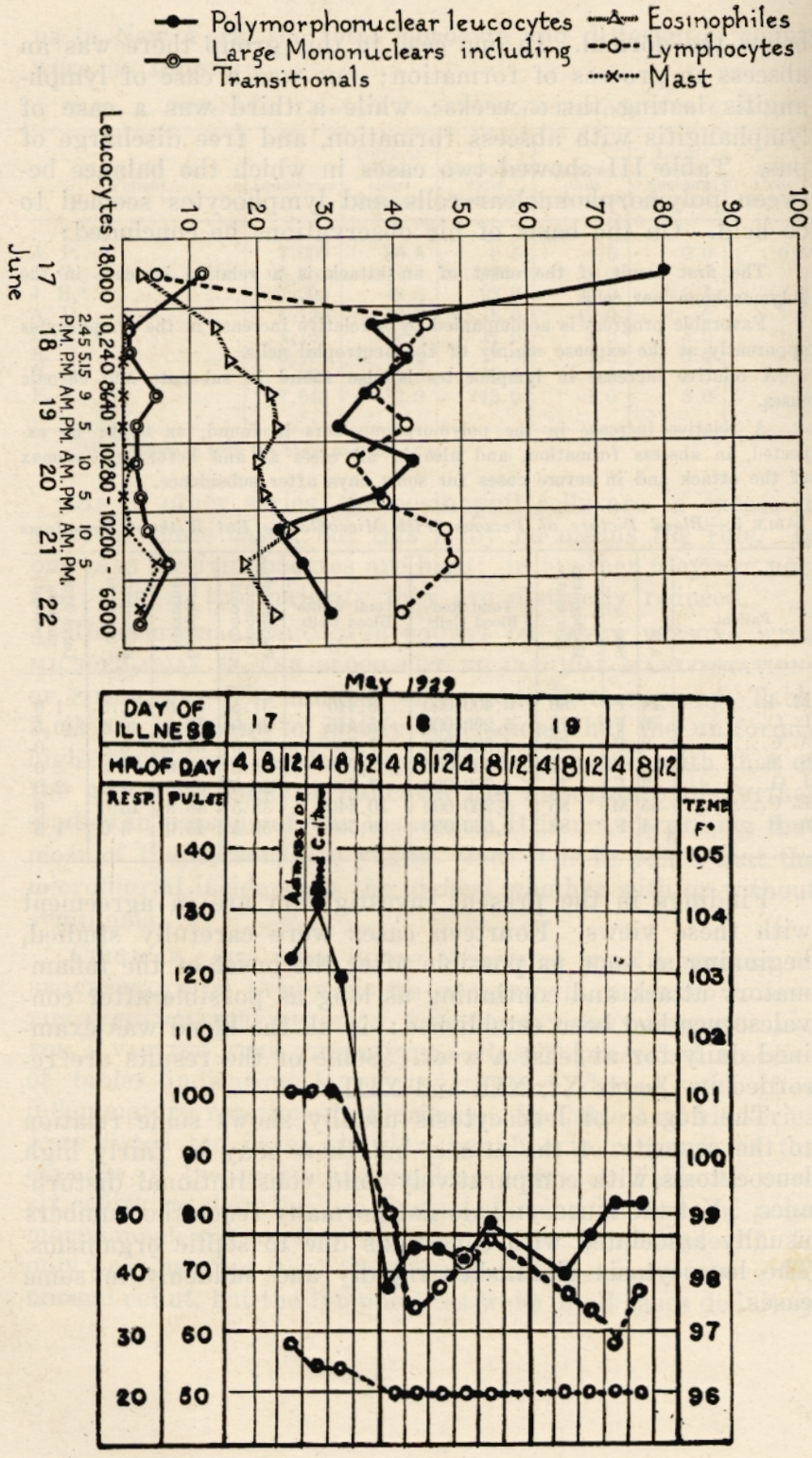
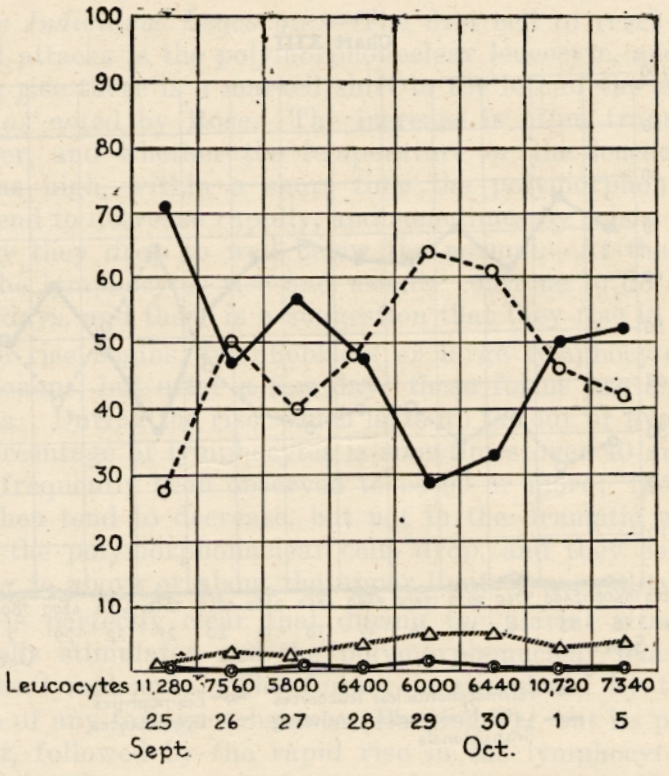

Polymorphonuclear leucocytes
Large Mononuclears including Transitionals
Eosinophiles
Lymphocytes
Mast
Leucocytes
10 20 30 40 50 60 70 80 90 100
June
17 18 19 20 21 22
18,000 10,240 8640 - 10280 - 10200 - 6800
2 4 5 5 9 5 10 5 10 5
P.M. P.M. A.M. P.M. A.M. P.M. A.M. P.M.
May 1929
DAY OF ILLNESS
17 18 19
HR. OF DAY
4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12 4 8 12
RESP.
PULSE
TEMP. F°
140 130 120 110 100 90 80 70 60 50
50 40 30 20
105 104 103 102 101 100 99 98 97 96

Chart XII


100
90
80
70
60
50
40
30
20
10
Leucocytes 11,280 7560 5800 6400 6000 6440 10,720 7340
25 26 27 28 29 30 1 5
Sept. Oct.
Polymorphonuclear leucocytes
Large Mononuclears including Transitionals
Eosinophiles
Lymphocytes

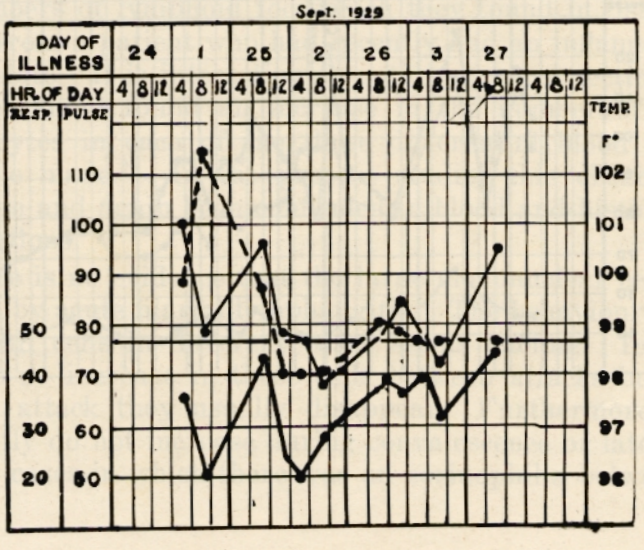
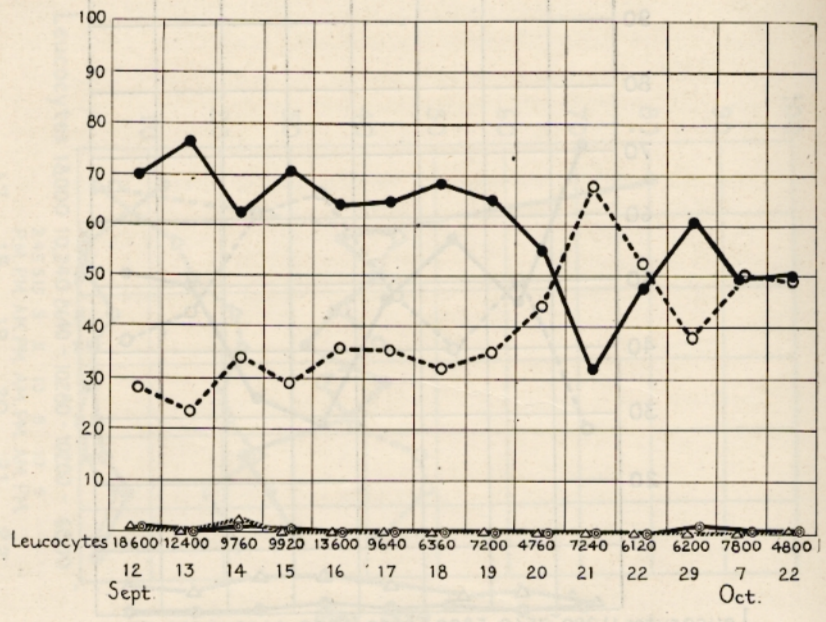
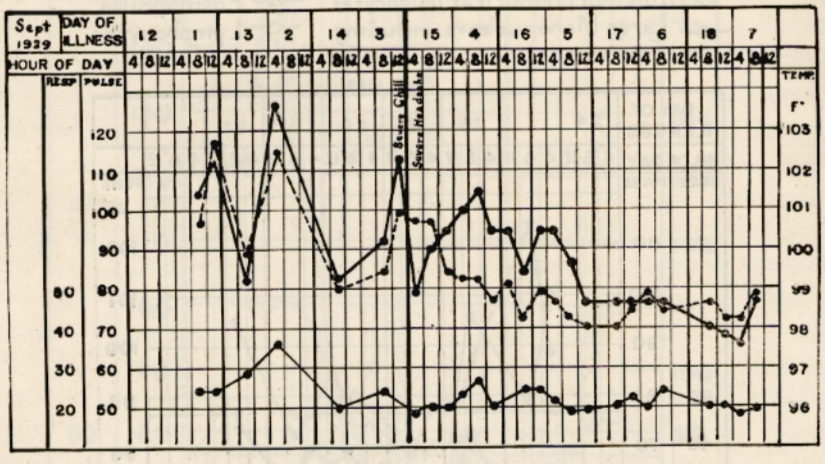

Sept. 1929
DAY OF ILLNESS
HR. OF DAY
RESP.
PULSE
TEMP F°
110
100
90
80
70
60
50
50
40
30
20
102
101
100
99
98
97
96

Chart XIII


100
90
80
70
60
50
40
30
20
10
Leucocytes 18600 12400 9760 9920 13600 9640 6360 7200 4760 7240 6120 6200 7800 4800
12 13 14 15 16 17 18 19 20 21 22 29 7 22
Sept.
Oct.
Polymorphonuclear leucocytes
Large Mononuclears including Transitionals
Eosinophiles
Lymphocytes


Sept 1929
DAY OF ILLNESS
HOUR OF DAY
RESP
PULSE
TEMP. F°
Severe Chill
Severe Headache

The Individual Leucocyte:—The first cell to react in all filarial attacks is the polymorphonuclear leucocyte, and during its rise there is a marked shift to the left of the Arneth count as noted by Rose. The increase is often transitory, however, and whether the temperature or the leucocytosis remains high, within a short time the polymorphonuclear cells tend to decrease rapidly, and sometimes by crisis. Frequently they drop to well below the normal. At the same time the lymphocytes rise and usually continue to do so for a few days, and there is a suggestion that they rise in steps. As the rise begins, lymphoblasts or large lymphocytes are conspicuous, but after a few days these forms are less numerous. During the rise, which lasts up to four or five days, the percentage of lymphocytes is sometimes over 40 and has not infrequently been observed to be 60 or more; the numbers then tend to decrease, but not in the dramatic way in which the polymorphonuclear cells drop, and they continue to vary to above or about the upper limit of normality.

It is perfectly clear that during the filarial attack the two cells stimulated are the polymorphonuclear leucocytes and the lymphocyte. The former is stimulated by the invasion of any foreign substance (Bunting [30]); but its prompt retreat, followed by the rapid rise in the lymphocytes, attended by the presence of young forms, suggests that the lymphocyte is essentially the cell stimulated by the poison responsible for the attack. Lothrop and Pratt [31] describing the numbers of lymphoid foci which they found in tissue removed from a patient who had recently had an inflammatory attack state "It is interesting to note in this connection that Dr. Steensland found an increase in the number of small lymphocytes in each of the three differential blood counts which he made." In the present studies this observation has again and again been confirmed in blood and histological examinations.

There is no evidence that the large mononuclear cells are affected by acute filarial lymphangitis. The behaviour of the eosinophil cells in different cases seems baffling. In some there is no increase in these cells between attacks but during the attack they usually disappear. Furthermore, they frequently do not increase during convalescence or later. In the few cases in which there was an eosinophilia between at-

tacks the behaviour of these cells was interesting; almost disappearing during the acute phase, they reappeared with or soon after the rise of the lymphocytes and gradually regained the preinflammatory level, or having reached this level, sometimes decreased slightly. No attempt is made to explain this phenomenon, but several possibilities suggest themselves. The eosinophil cell has probably a chemiotactic attraction for new infection by living worms, as analogy with ankylostomiasis, schistosomiasis and trichiniasis would suggest. It is possible that in filariasis eosinophilia may be an indication of the presence of living worms or, as suggested by Calvert, of a recent reinfection of living worms. It should be noted for instance that marked local eosinophilia is sometimes, though not always, found in the vicinity of degenerating worms. The absence of eosinophilia possibly may be due to the absence of living worms or to the presence of only calcified, calcifying or degenerating worms in which the process is well advanced. Again it should be emphasized that these views are not mentioned as conclusions but as suggestions for future investigations.

Occasionally, when recurrences of lymphangitis follow each other in rapid succession, so that the clinical picture almost suggests one protracted attack, the blood picture is altered. The leucocytosis remains high and the polymorphonuclear cells only gradually decrease with a simultaneous slow rise of lymphocytes. In one such case repeated cultures from the blood and local areas of inflammation failed to reveal the presence of bacteria.

When convalescence from one attack is interrupted by a recurrence a new leucocytosis develops, with a rise in the polymorphonuclear cells and a proportionate fall in the lymphocytes and the eosinophil cells. It was noted that in successive attacks in the same person the blood picture tended to be fairly similar with each recurrence.

When a patient subject to recurrent attacks of lymphangitis of short duration, has a new attack complicated by bacterial invasion with the development of abscess, the blood picture at first resembles in all essentials that observed in the cases already described. But with the introduction of septic organisms two things may happen. If this has taken place early the case does not end by crisis nor does the blood

picture change as in the ordinary attack; instead, there is rising leucocytosis with polymorphonuclear predominance, the condition typical of the purulent process. If infection occurs later in the attack or during convalescence the process is interrupted; the patient complains of local pain, becomes ill, with rising temperature, and shows a pyogenic blood picture. In those so-called abscess cases, in reality lymphocele, in which sterile abscess commonly is found, the blood picture differs in no essential from that of uncomplicated filarial lymphangitis.

RELATIONSHIP OF THE BLOOD PICTURE TO MICROFILARIAL PERIODICITY.—Gulland [32], studying the blood of filarial patients in relation to microfilarial periodicity, found that there was an evening eosinophilia, a constant high proportion of lymphocytes which did not show much variation and a percentage of polymorphonuclear cells, which never reached the normal of seventy. The relation between the polymorphonuclear cells and the lymphocytes remained fairly constant, and he concluded that both must have been increased absolutely to have produced the high evening counts of leucocytes. He also found that the eosinophil cells rose steadily from normal in the early morning, when no microfilariae were found, to great numbers just before midnight, when the embryos were most numerous.

In one of the two cases described by Calvert [33], however, lymphocytes were low, while in the other they only once reached 26 per cent. He found eosinophil cells most numerous between 9 a. m. and 5 p. m., when there were no embryos; between 5 and 11 p.m., when the embryos were increasing, the eosinophil cells were decreasing; while between 11 p. m. and 8 a. m., with embryos decreasing, he found the eosinophil increasing. In a second paper [33a] the same observer states that "leucocytosis and eosinophilia are cyclic, following by a few hours the periodicity of the embryos in the peripheral circulation."

The two studies (Charts VII & VIII) are notably similar, and the similarity is the more remarkable in that their microfilarial peaks were four hours apart. These various circumstances suggest the closest correlation between the blood changes and the different phases of periodicity and suggest that the blood changes while cyclic, as Calvert be-

lieved, roughly coincide with the presence of embryos in the peripheral circulation. Furthermore the polymorphonuclear cells were well below normal and the relation, as in Gulland's experience, between these cells and the lymphocytes was remarkably constant, thus confirming his belief that both cells are absolutely increased to produce leucocytosis during microfilarial periodicity.

TREATMENT AND PREVENTION OF FILARIASIS

The treatment of filariasis involves three factors: treatment of the inflammatory attacks, prevention of their recurrence, and treatment of the obstructive phenomena, especially elephantiasis.

Treatment of Acute Filarial Lymphangitis.—Magnesium or sodium sulphate taken at the first warning of an impending attack of filarial lymphangitis, or with the onset of primary pain, is usually of distinct benefit in that it modifies the severity of the inflammation and sometimes actually prevents its development. When possible, the patient should go to bed and remain there until convalescence is complete. The headache is generally relieved by aspirin in 5 or 10 grain doses. The patient is as a rule disinclined to eat, but he should be encouraged to take plenty of bland fluid to aid elimination by the kidneys. The affected limb should be raised and an icebag applied to the areas of most intense inflammation. When ice is not available, and provided there is no abrasion of the skin, cooling lotions, such as *lotio plumbi* may be applied on cloths. When a focal spot is definitely determined or where inflammatory reaction is intense an ethylchloride spray once a day is of great benefit and may shorten the attack, but should be stopped before definite frosting of the epidermis takes place. Any of the well known sedative lotions may be applied to limit the pruritis. While still in bed it is well to bandage the limb from below upward in order to reestablish the lymph supply with a view to preventing the onset of permanent swelling. The bandage should be retained for at least a week.

Prevention of the Recurrence of Lymphangitis.—No drug has been discovered that will prevent recurrences of filarial lymphangitis or adenitis. Tartar emetic and various arsenical compounds have been administered intravenously with-

out success. That these drugs should be ineffective when administered in this way is not surprising, since the parasites are not in the blood stream, and if degenerating are largely cut off from nutrition.

In the belief that this disease is largely caused by pyogenic bacteria, various investigators have claimed beneficial results from the administration of the streptococcal vaccines and serums and vaccines of other organisms. Observations made during the present studies indicate that such claims are not substantiated.

Recently an effort was made to treat lymphangitis by infiltration of the tissues in and around the focal spots with sulpharsphenamin. In the majority of cases attacks were postponed for from several months to a year or more by this treatment; but it is now clear that like other methods of therapy, this can not be considered curative. At the present time studies are being made with regard to determining the efficacy of small amounts of roentgen therapy applied locally over fairly large areas (Golden [34]). It is too early to evaluate results, but the indications following two years of experimentation are encouraging.

Removal to Another Climate.—Since earliest times the belief has been current that removal to a temperate climate will cause a cessation or modification of the inflammatory symptoms. Towne [35] (1726) wrote, "Let me therefore exhort all such whose circumstances will admit of it to leave the island at first approach of distemper and remove for some time to England". Hendy [14] was "very credibly informed that persons affected with glandular disease while at Barbadoes, when they go to reside at Tobago are never attacked with it" and he cites a case, in which the patient ceased to have the attacks during an eighteen months' sojourn in England. During the last century Waring [36] (1859), Day [37] (1860) and Fayrer [38] (1879), to mention only a few prominent observers, also described the beneficial effects of removal to cooler climates. So strong is the belief among Puerto Ricans that residence in the northern States will put an end to lymphangitis that in New York one finds a curious anomaly, namely, that in the Puerto Rican population there is probably a higher percentage of persons with a history of clinical filariasis or of persons with microfilariae in the