JOINT DISCUSSION No. 1

Section of Epidemiology and State Medicine with Section of Tropical Diseases and Parasitology

Chairman—Surgeon-Captain SHELDON F. DUDLEY, O.B.E., R.N. (President of the Section of Epidemiology and State Medicine).

[*November* 15, 1935]

DISCUSSION ON THE MALARIA EPIDEMIC IN CEYLON 1934-1935

OPENING PAPERS

1.—R. Briercliffe, O.B.E., M.B., M.R.C.P., and Sir Weldon Dalrymple-Champneys, Bart., M.D., F.R.C.P.

[Sir WELDON DALRYMPLE-CHAMPNEYS, before reading the joint paper prepared by Dr. Briercliffe and himself, expressed his deep regret, which he felt sure was shared by everyone present, that the recrudescence of malaria in Ceylon had prevented Dr. Briercliffe from being with them that night. Sir Weldon said he could testify as an eye-witness, to the splendid way in which Dr. Briercliffe had handled this unprecedented epidemic in very difficult circumstances and in the face of much hostile criticism and misrepresentation.]

INTRODUCTION

The epidemic of malaria in Ceylon, which started towards the end of 1934 and was followed by a large secondary wave starting in April 1935, attracted world-wide attention as being the most serious visitation of this disease on record in Ceylon. Though severe epidemics of malaria in Ceylon were recorded as far back as the middle of the seventeenth century, and at intervals ever since, scientific investigation has hitherto been concentrated almost entirely upon the *endemic* features of the disease and Government control has been focused upon centres of high endemicity. *Epidemic* malaria has consequently received little attention until the present disastrous outbreak, which is likely to have prolonged and far-reaching effects upon the welfare of the country. These circumstances, together with certain unusual features which will be referred to in the course of this paper, make the recent epidemic of particular interest to malariologists, medical officers working in tropical countries, and epidemiologists in general.

Moreover, the misleading and inaccurate accounts which appeared in the lay press of many countries (for instance, on one occasion a report of 5,000 deaths in a particular district became 50,000 in an English newspaper and had swollen to 500,000 by the time the same news appeared in the U.S.A.), render it particularly desirable that a clear account of the chief events and circumstances should be given to the medical profession.

CEYLON AND ITS CLIMATE

The island of Ceylon lies between 5° 55′ and 9° 50′ north latitude, that is to say very near the equator. It is 25,332 square miles in extent and is shaped roughly like the skull of a sheep standing on its occipital process with the lower jaw missing (fig. 1). The population of rather more than five and a half millions consists

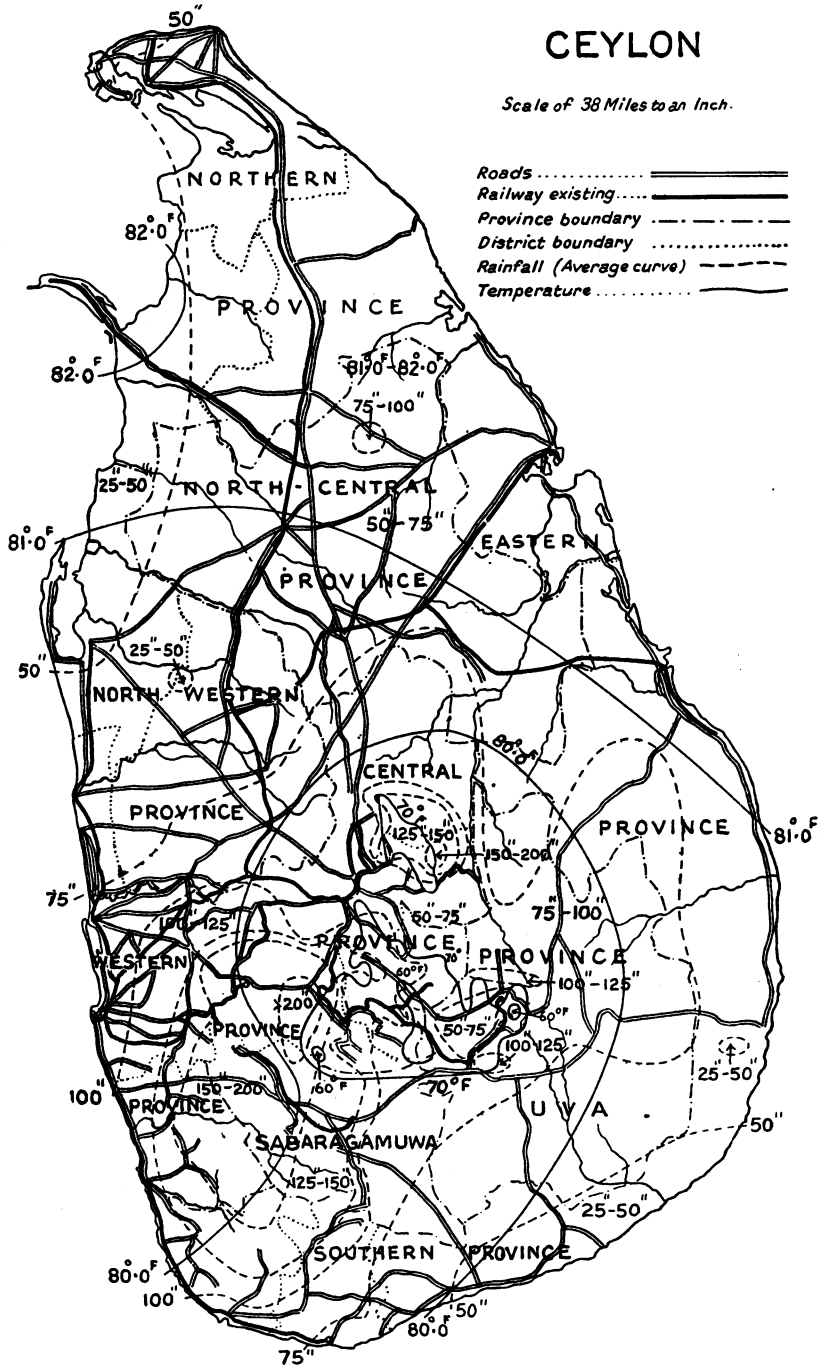
CEYLON
Scale of 38 Miles to an Inch.
Roads
Railway existing
Province boundary
District boundary
Rainfall (Average curve)
Temperature
NORTHERN PROVINCE
NORTH-CENTRAL PROVINCE
EASTERN PROVINCE
NORTH-WESTERN PROVINCE
CENTRAL PROVINCE
WESTERN PROVINCE
SABARAGAMUWA PROVINCE
SOUTHERN PROVINCE
UVA PROVINCE
50"
82·0° F
81·0° F
81·0-82·0°
75"-100"
25"-50"
50"-75"
125"-150"
150"-200"
100"-125"
80·0° F
70° F
60° F

FIG. 1.

of a number of different races and is very unevenly distributed (this will be referred to later). Malaria is endemic or hyperendemic over about three-quarters of the island and a general idea of its endemic distribution is given by fig. 2 which is based upon the examination of over 50,000 children for splenic enlargement. The deeper the shading here, the higher the endemicity. As will be seen from the map, the south-west quadrant is a comparatively non-malarious area in normal years and it was this area which was chiefly affected by the present epidemic.

The chief factor governing malaria in Ceylon is rainfall (fig. 3). The south-west quadrant, on account of the mass of mountains in the south-central part of the island, receives much more rain than other parts of Ceylon and the rain is distributed throughout the whole year, that is to say during the periods both of the south-west monsoon from April to September and of the north-east monsoon from October to March. On account of its abundant rainfall and the absence of long periods of drought the south-west quadrant is known as the "wet zone." It is the richest part of Ceylon agriculturally, and the healthiest and so it supports the larger part of the population (fig. 4).

The remaining three-quarters of Ceylon receive their rain mostly at the beginning of the north-east monsoon, October, November, and December being the wet months. The rains of the south-west monsoon are scanty in this area and on account of the long period of comparative drought during this monsoon the northern half and south-east quarter are known as the "dry zones."

Briefly, the sequence of events leading up to the epidemic was as follows: In 1934 from April onwards the rainfall was generally deficient owing to a partial failure of the south-west monsoon. The rainfall during the south-west monsoon of 1934 compared with the average rainfall for the same period (taken over a period of normal years) is shown in fig. 5, the different hatchings indicating the different degrees of excess or deficit.

Over the greater part of the wet zone the rainfall readings for July, August, and September, were so low as to constitute a record. In this zone only in the south was the rainfall for the year up to the average, though there was a considerable deficit during July, August, and September. Owing, however, to the higher rainfall here from May to September, a much greater deficit is required to produce a drought and cessation of the flow of rivers (the Gin Ganga in the Galle District and the Nilwala Ganga in the Matara District) than in the north of the wet zone and consequently no actual drought occurred, nor was there any malaria here.

The effect of the unprecedented drought on the state of the rivers in the wet zone was of fundamental importance in connexion with the epidemic. Normally there is a good flow of water down these rivers the whole year round which keeps them flushed and prevents anopheline breeding. But during the drought the flow was greatly reduced and in the upper and middle reaches it became a mere trickle leading to the formation of pools of water in the rocky or sandy beds of five of the main rivers and their larger tributaries (fig. 6).

Figs. 7 and 8 show the same part of the Mahaweli Ganga before and after rainfall in the upper catchment area 24 miles above this point.

These pools in the river beds provided ideal breeding places for *Anopheles culicifacies*, the only one of the eighteen known species of anopheles in Ceylon (including *A. varuna* and *A. maculatus*) which has been proved to be a carrier of malaria in the island.

A. culicifacies selects shallow pools of still clear water exposed to sunlight in which to lay its eggs. Such pools are shown in fig. 9, a picture of the Deduru Oya. You will notice on the right the hoof prints of wild elephant. *A. culicifacies* prefers pools in the bed of a wide river to those in a narrow stream, because the latter are usually shaded by the vegetation on the banks, as is shown in fig. 10, a photograph of a section of the Kelani Ganga which proved negative for

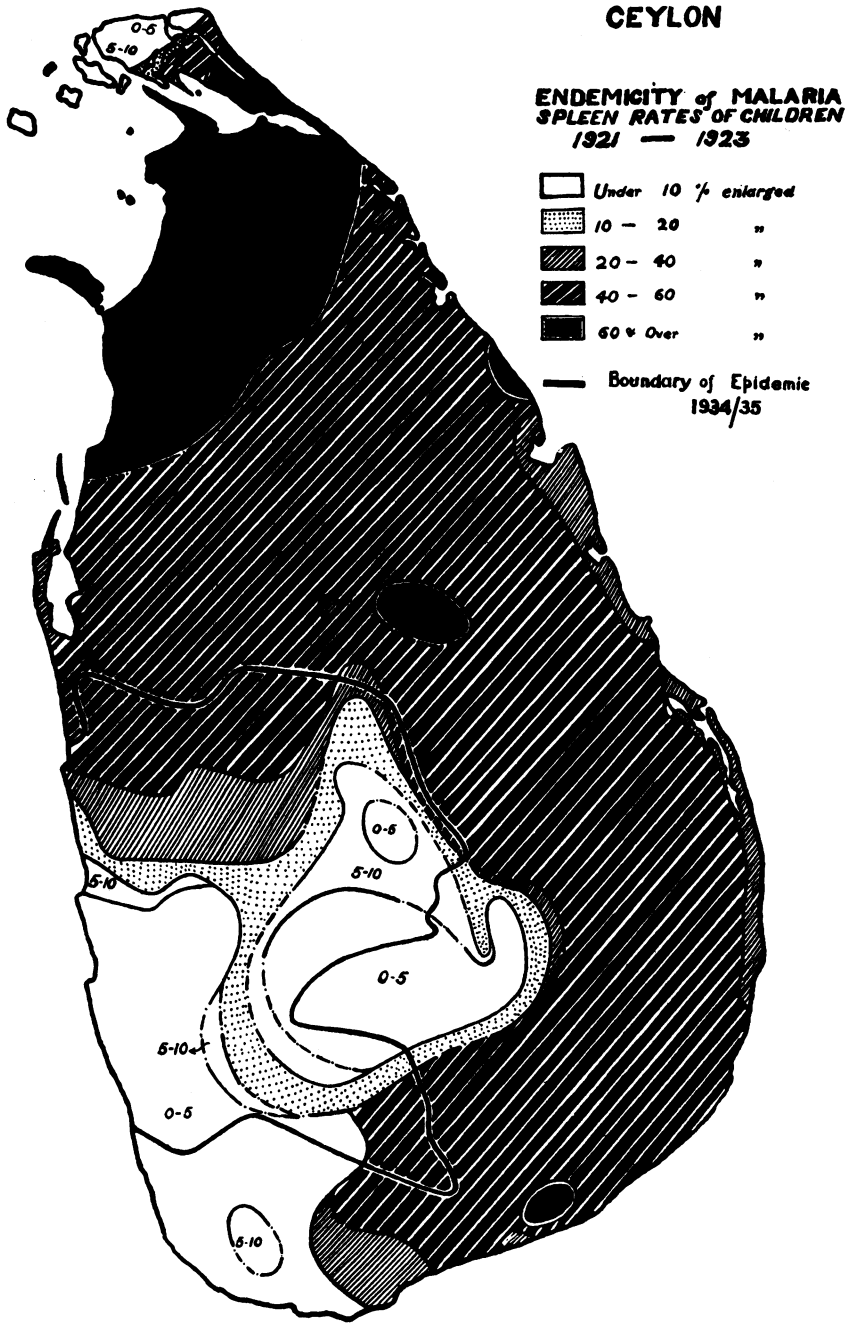
CEYLON
ENDEMICITY of MALARIA
SPLEEN RATES OF CHILDREN
1921 — 1923
Under 10 % enlarged
10 — 20 "
20 — 40 "
40 — 60 "
60 % Over "
Boundary of Epidemic
1934/35
0-5
5-10
0-5
5-10
0-5
5-10
0-5
5-10

FIG. 2.

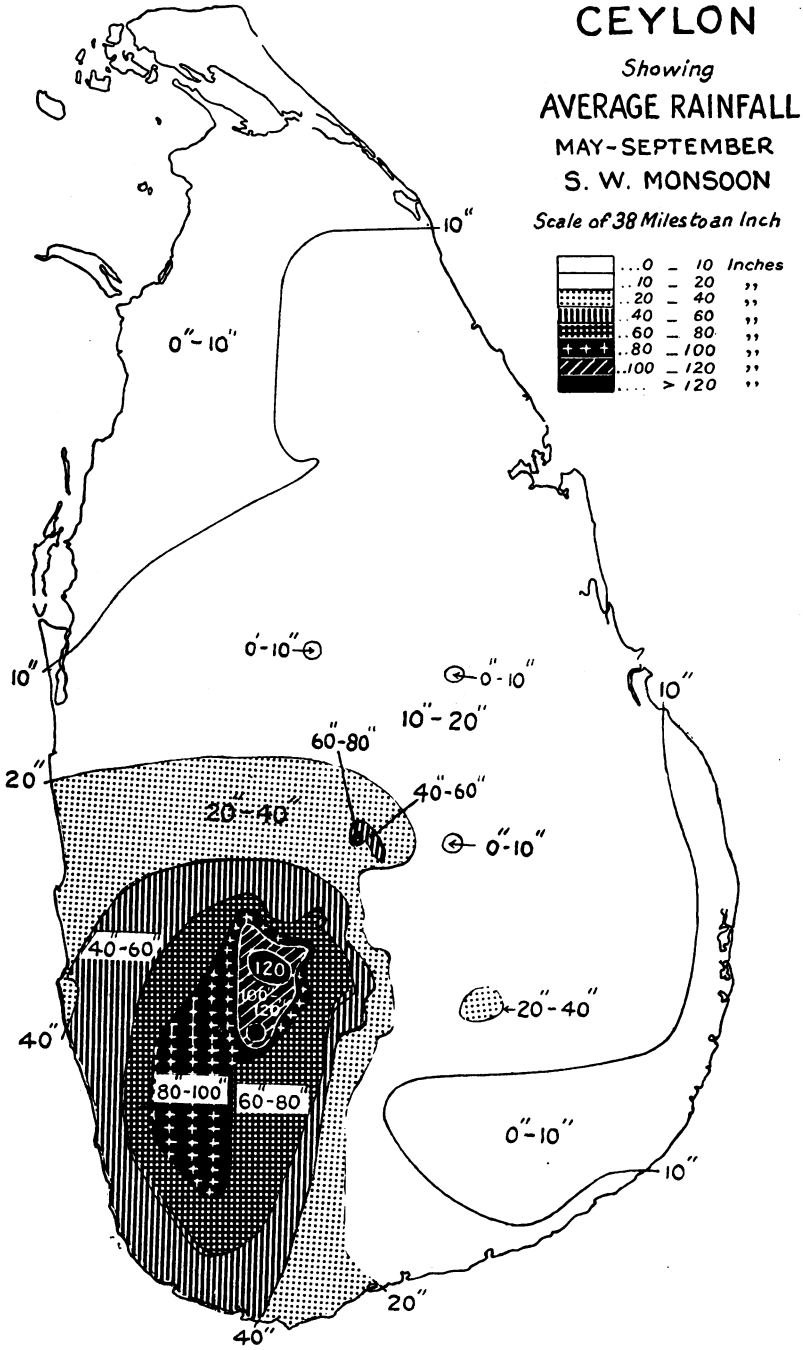
CEYLON
Showing
AVERAGE RAINFALL
MAY-SEPTEMBER
S. W. MONSOON
Scale of 38 Miles to an Inch
0 - 10 Inches
10 - 20 ,,
20 - 40 ,,
40 - 60 ,,
60 - 80 ,,
80 - 100 ,,
100 - 120 ,,
> 120 ,,
10"
0"-10"
0'-10"
0"-10"
10"-20"
10"
60"-80"
20"
20"-40"
40"-60"
0"-10"
40"-60"
120
100
80
40"
80-100"
60-80"
20"-40"
0"-10"
10"
20"
40"

FIG. 3.

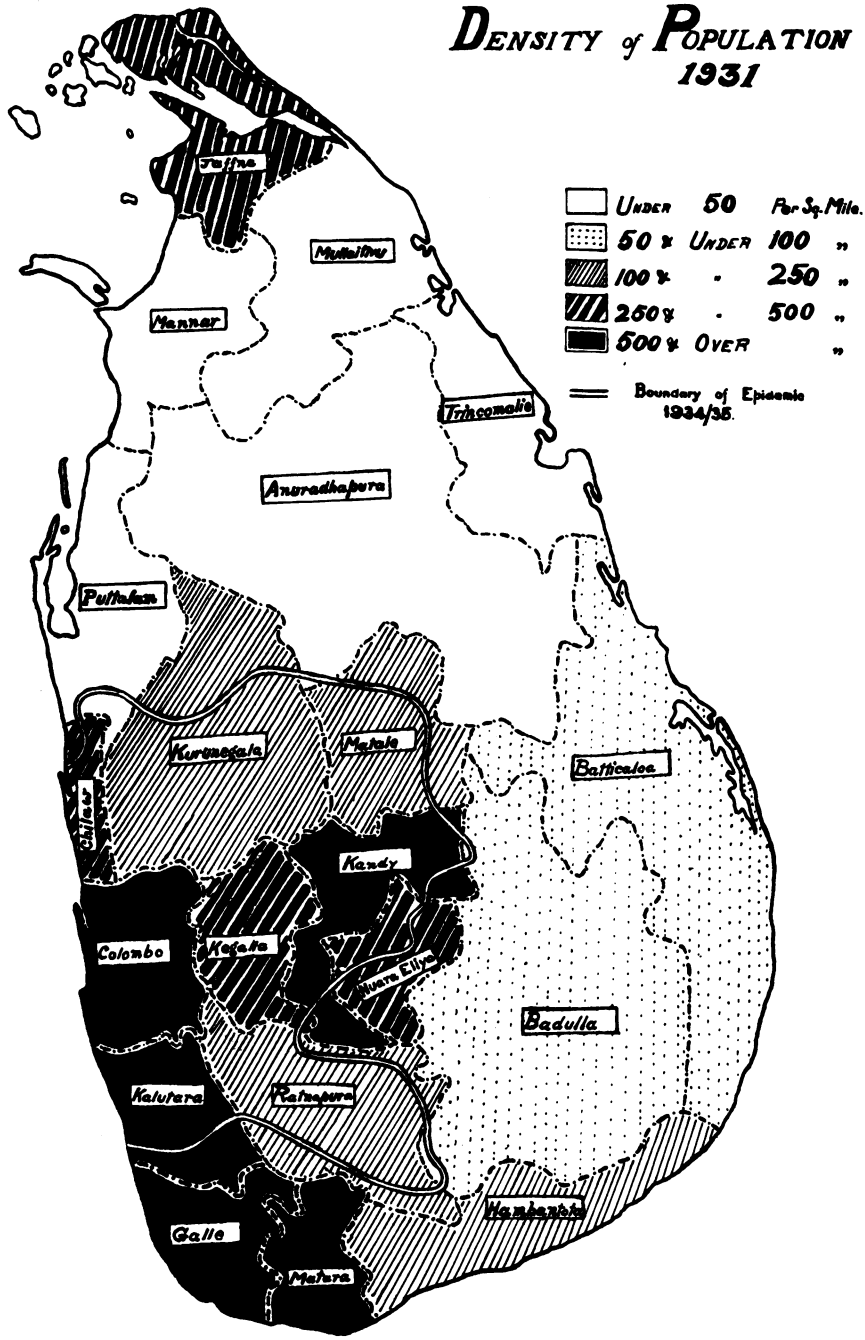
DENSITY of POPULATION
1931
Under 50 Per Sq. Mile.
50 % Under 100 "
100 % . 250 "
250 % . 500 "
500 % Over "
Boundary of Epidemic 1934/35.
Jaffna
Mullaitivu
Mannar
Trincomalie
Anuradhapura
Puttalam
Kurunegala
Matale
Batticaloa
Chilaw
Kandy
Colombo
Kegalle
Nuwara Eliya
Badulla
Kalutara
Ratnapura
Hambantota
Galle
Matara

FIG. 4.

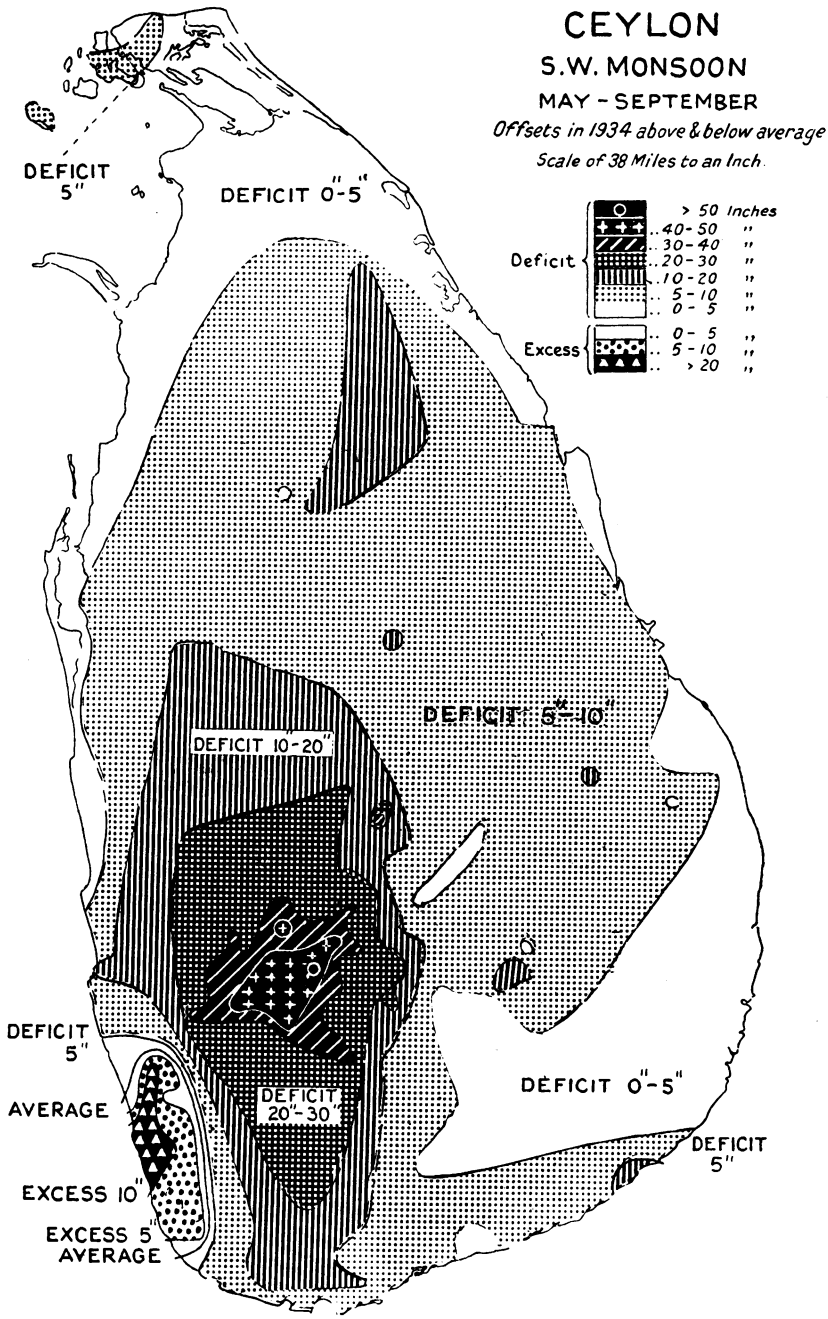
CEYLON
S.W. MONSOON
MAY - SEPTEMBER
Offsets in 1934 above & below average
Scale of 38 Miles to an Inch
Deficit
> 50 Inches
40-50 "
30-40 "
20-30 "
10-20 "
5-10 "
0-5 "
Excess
0-5 "
5-10 "
> 20 "
DEFICIT 5"
DEFICIT 0"-5"
DEFICIT 10-20"
DEFICIT 5"-10"
DEFICIT 5"
AVERAGE
EXCESS 10"
EXCESS 5"
AVERAGE
DEFICIT 20"-30"
DEFICIT 0"-5"
DEFICIT 5"

FIG. 5.

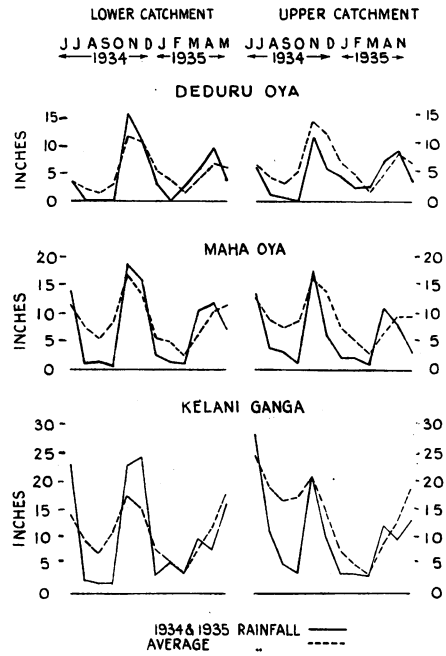

FIG. 6.

FIG. 7.—Portion of the Mahaweli Ganga during drought showing a large number of pools.

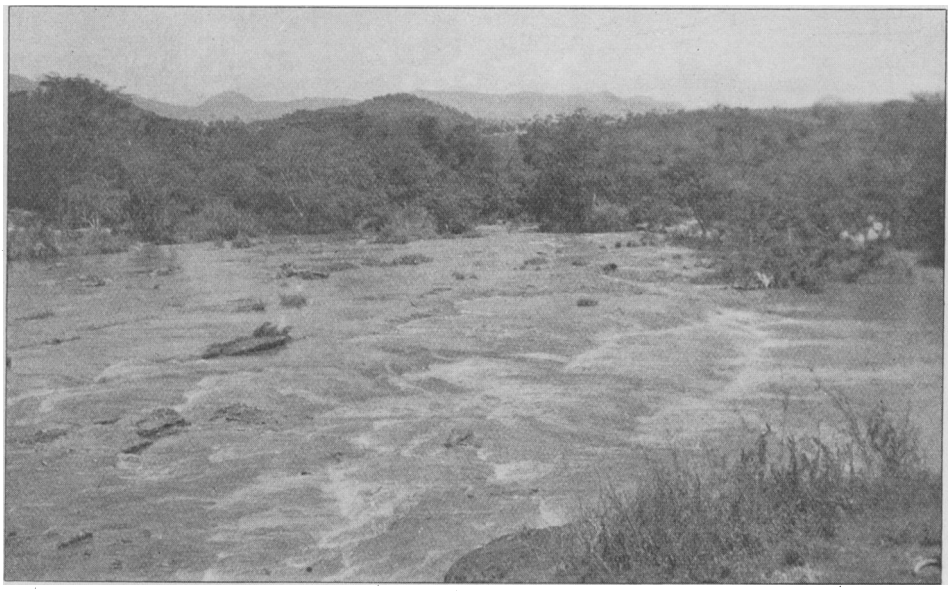

FIG. 8.—The same portion of the Mahaweli Ganga, as in fig. 7, showing the pools flushed out by rain in the upper catchment area.

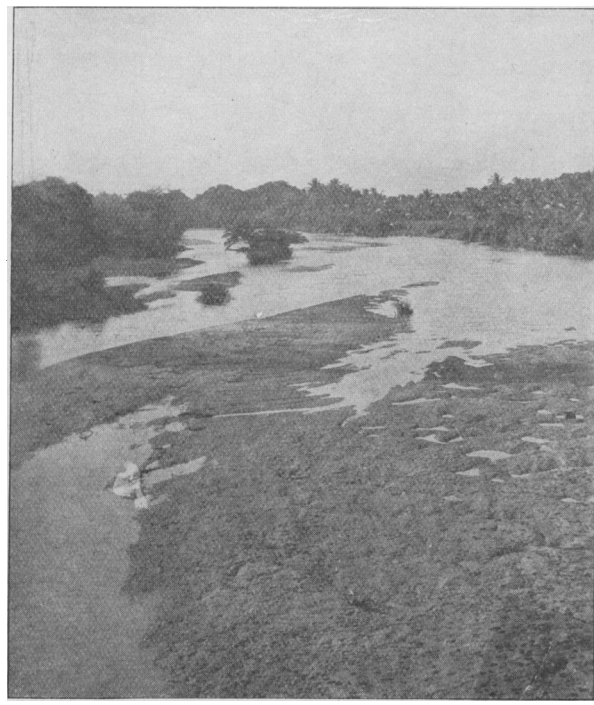

FIG. 9.—Portion of the Deduru Oya showing pools of clear water, exposed to sunlight which provide ideal breeding places for *Anopheles culicifacies*. On the right are seen the hoof-prints of wild elephant.

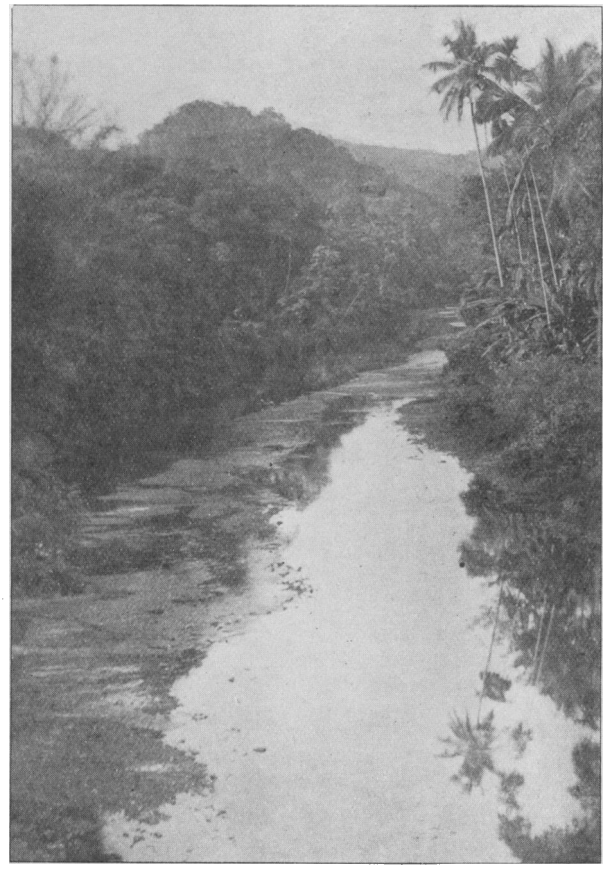

FIG. 10.—Section of the Kelani Ganga, shaded by vegetation, which proved negative for *Anopheles culicifacies*.

A. culicifacies. Shallow wells, rock pools in quarries, borrow pits by the side of roads and railways and brick pits are subsidiary breeding places of this mosquito, but they were not of major importance during the epidemic.

The natural home of *A. culicifacies* is in the dry zones, because suitable conditions for its propagation exist there. In the wet zone it is normally found only in small numbers, but in July, August, and September 1934, conditions over a large part of the wet zone approximated to those in the dry zones, and several hundred miles of the beds of five of the main rivers and their tributaries were providing unusually favourable conditions for the propagation of *A. culicifacies*.

The adult *A. culicifacies* is a difficult mosquito to find, but during the early stages of the epidemic the entomological assistants obtained it from the houses of villages near the five rivers of the epidemic area in numbers never previously met with in the wet zone. Thus of 5,063 anophelines collected in dwellings from November onwards, 88·5% were *A. culicifacies* and 8% were *A. subpictus*, a harmless mosquito. The remaining 2·8 per cent. consisted of five different species of anopheles.

There is no scarcity of gametocyte carriers in Ceylon, since in any year it is rare to get less than 400,000 persons seeking treatment for malaria at the Government

dispensaries, and in a year of high malarial incidence (such as 1930) this number may be doubled. Ample opportunity existed therefore for *A. culicifacies* to become infected, and high infection rates were recorded. Thus in December 12·9% of the *culicifacies* dissected contained oöcysts, or sporozoites or both. In certain localities infection rates as high as 19% were obtained.

With regard to the parasite, throughout the epidemic benign tertian infections (*Plasmodium vivax*) predominated, the relative proportions of the three parasites found in the positive blood films examined from November 1934 to March 1935 being *P. vivax* 62·2%, *P. falciparum* 36·7%, and *P. malariæ* 1·1%. The proportion of malignant tertian infections increased, however, from 24·7% in November to 43·4% in January, and then fell to 28·0% in March.

(As an example of the extraordinary statements made in some newspapers, it may be mentioned that early in the epidemic a reporter cabled that a green caterpillar pest had appeared in the Kurunegala district, and thousands were being buried. In the American newspaper the word caterpillar was omitted, and several requests were received by the medical department from American malariologists for specimens of the "green malarial parasite"!)

Malaria broke out in epidemic form in the following river basins from north to south: Dedura Oya, Maha Oya, Kelani Ganga and Kalu Ganga (fig. 11). These four rivers lie on the western side of the watershed; on the eastern side part of the basin of the Mahaweli Ganga was involved. In each river basin the villages first and worst affected were near the banks of the main river or its larger tributaries.

The area affected by the epidemic (*see* fig. 4) is about 5,800 square miles in extent, and has a population of approximately 3,100,000 persons. Thus the epidemic involved nearly a quarter of the island's area, containing more than half its people. In the south of the wet zone (Galle district) neither *culicifacies* larvæ nor adult *culicifacies* were found. The rivers were flowing as usual, and malaria did not occur.

Course of the Epidemic

The abnormalities of the rainfall during the third quarter of the year had given rise to the expectation of an increased malaria prevalence and of localized outbreaks towards the end of the year, so that when villages near the Maha Oya showed a rising incidence of malaria in October the provincial surgeons had already been warned to expect malaria by a memorandum written by Dr. K. J. Rustomjee, Superintendent of the Anti-Malaria Campaigns.

But the overwhelming epidemic which broke out with extreme suddenness during the latter half of November and early December was not anticipated, and was indeed unprecedented.

First the valleys of the Maha Oya and Deduru Oya were invaded (*see* fig. 11), and it was in these river basins that the epidemic was most intense and took the greatest toll of life. Then parts of the Mahaweli Ganga basin were quickly involved, and early in December the Kelani Ganga and the Kalu Ganga basins.

The explosive nature of the onset was at first the characteristic feature of the epidemic. For example, the lower two-thirds of the Deduru Oya valley lie in the dry zone, and autumnal outbreaks of malaria occur every two or three years, but the rise in the number of patients takes place gradually. The epidemic, however, burst with amazing suddenness in the Deduru Oya basin, which in the course of a week was completely involved.

The lower reaches of the rivers and the coast were the last parts to be invaded. South of the mouth of the Maha Oya the thickly populated coastal region, with its large towns, is normally free from malaria, and this region escaped comparatively lightly from the epidemic. Generally conditions were worst in the upper and middle reaches of the rivers, and so the inland districts suffered more than the corresponding coastal districts.

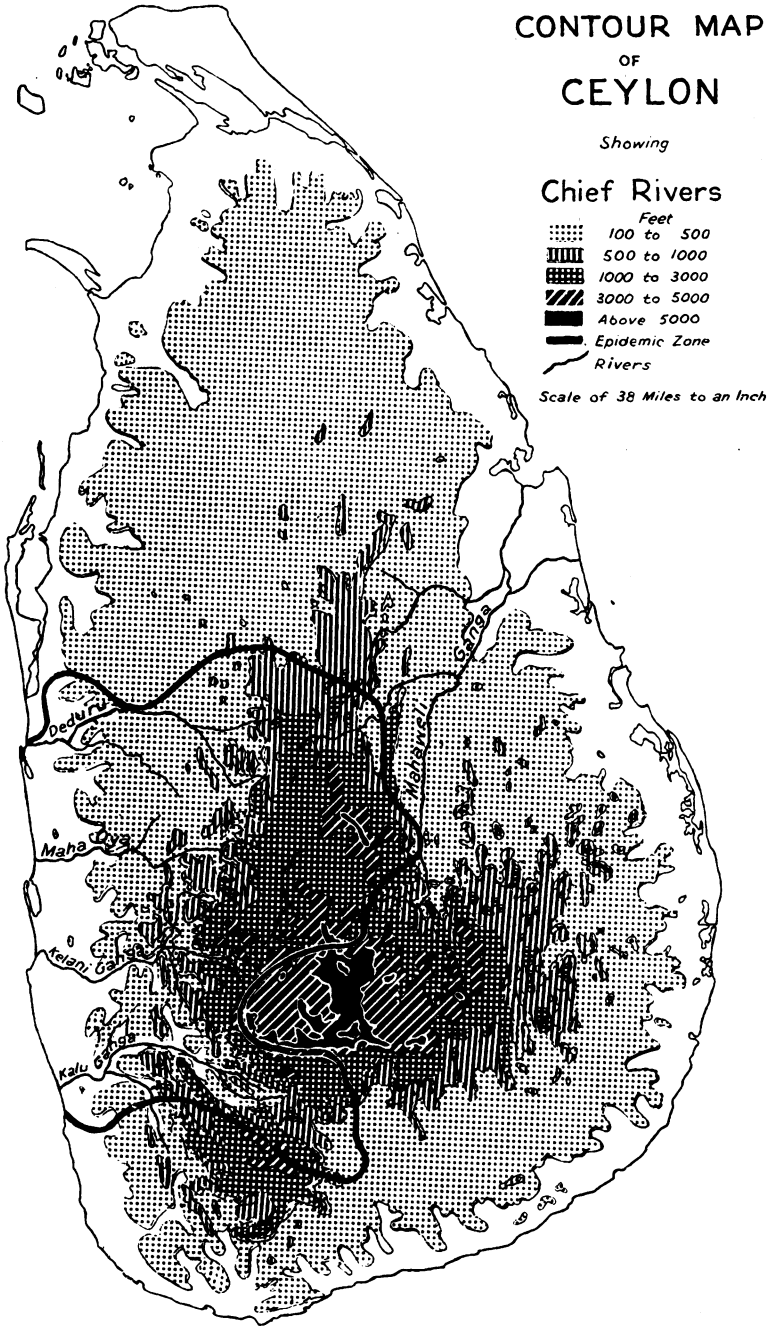
CONTOUR MAP
OF
CEYLON
Showing
Chief Rivers
Feet
100 to 500
500 to 1000
1000 to 3000
3000 to 5000
Above 5000
Epidemic Zone
Rivers
Scale of 38 Miles to an Inch
Deduru Oya
Maha Oya
Kelani Ganga
Kalu Ganga
Mahaweli Ganga

FIG. 11

Judging by dispensary attendances (fig. 12) the epidemic reached its peak about December 22. On that day there were 62,000 attendances at the Government hospitals, dispensaries, and temporary treatment centres in the epidemic area. It was estimated that by the middle of December about half a million people in the epidemic area had contracted malaria.

For the next few weeks there was a fairly rapid drop in the number of patients and from the end of January the numbers declined gradually until the second week of April. During this period new infections and reinfections were getting fewer, but it was estimated that nearly one and a half million persons contracted malaria

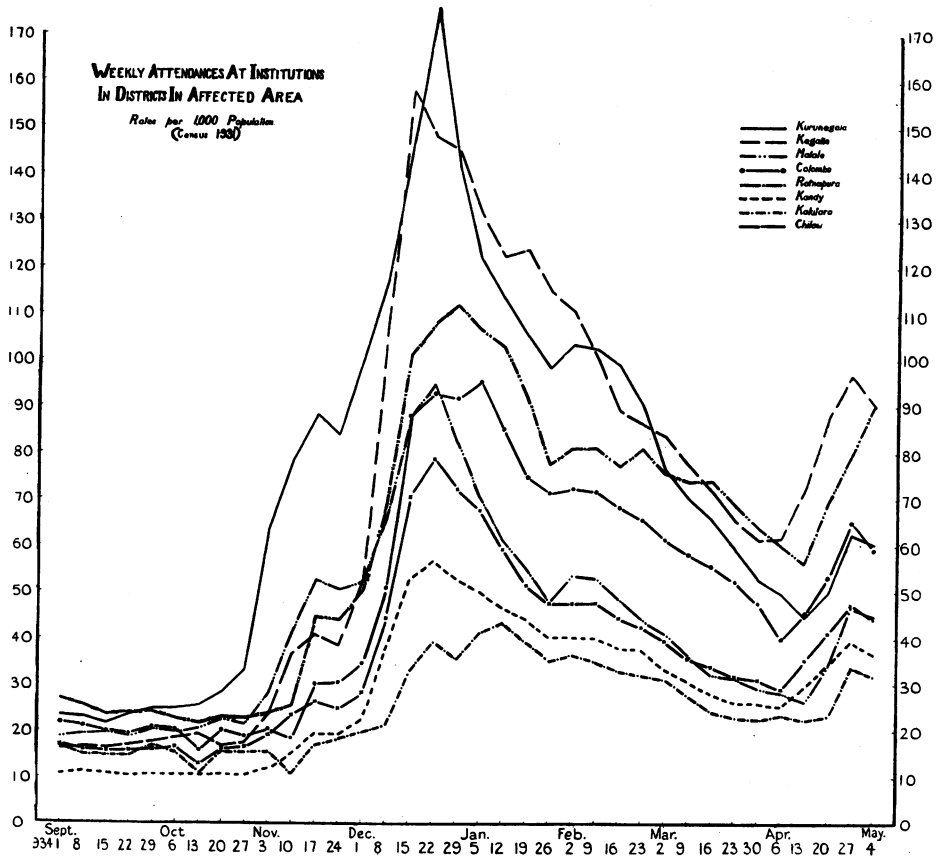

FIG. 12.

during the period October to April. Large numbers of patients suffering from relapses and from post-malarial debility and œdema continued to attend the dispensaries and treatment centres.

During the six months, November 1934 to April 1935, 92,556 deaths from all causes took place in the epidemic area (fig. 13). The average number of deaths for the same period of the previous four years was 33,412. The excess of 59,144 may be attributed directly or indirectly to the epidemic.

Outside the epidemic area unhealthy conditions prevailed in most parts of Ceylon and in certain hyperendemic districts there was a much increased seasonal incidence of malaria.

The expected number of deaths for the non-epidemic areas for the six months ending April 30 was 26,724 and the actual number 37,175, an excess of 10,451 (fig. 14). January is normally the month of highest mortality and the heaviest mortality during the epidemic took place in this month, i.e. the month after *morbidity* had reached its peak. In the epidemic area the excess of deaths over the expected number was 1,770 in November, 8,195 in December, 20,594 in January, 13,619 in February, 8,470 in March, and 6,496 in April.

At the end of the first week of April a secondary wave of malaria started to sweep over the epidemic area. It was not unexpected.

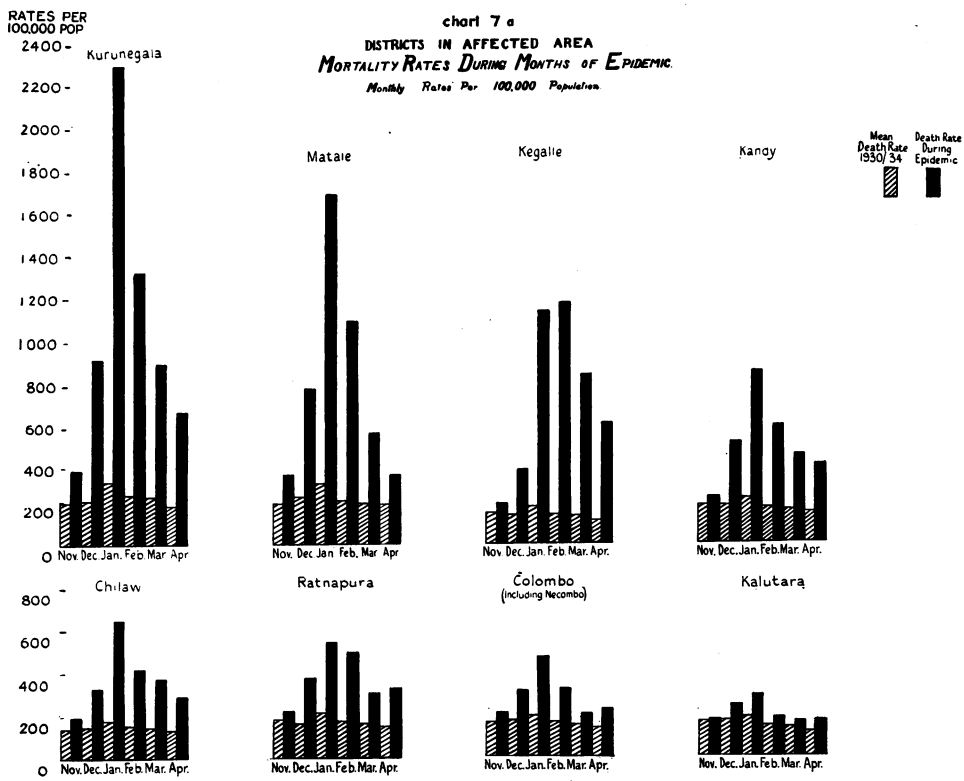

FIG. 13.

In a preliminary report on the epidemic dated January 10, 1935, it was stated :—

" In the dry zones of Ceylon where malaria is endemic or hyperendemic the seasonal increase starts in November after the first heavy rains of the north-east monsoon and continues till March. During 1934 on account of the lack of rain there had been less malaria than usual in many parts of the dry zones. In the wet zone, however, localized outbreaks of malaria when they occur usually follow deficient rainfall from the north-east monsoon and begin in April, consequent on pool formation in the rivers during the hot dry weather preceding the break of the south-west monsoon.

Therefore, while it is anticipated that the present epidemic will slowly subside during the next two or three months, it is possible that after March there will be a recrudescence which poor economic conditions may intensify."

The great epidemic differed from other malaria epidemics which have occurred in the wet zone because it started in October and not in April. When malaria occurs in the wet zone April to July is its usual period.

The failure of the south-west monsoon had been followed by a weak north-east monsoon and from December to the middle of March the river beds, particularly those of the Maha Oya and Mahaweli Ganga, continued to provide very favourable conditions for the breeding of *A. culicifacies*. The infection rate of this mosquito, which had fallen to 1·9% in February, was 3·4% in April, 7·2% in May and 5·5% in June. In July and August so few *culicifacies* could be caught that infection rates for these months could not be determined.

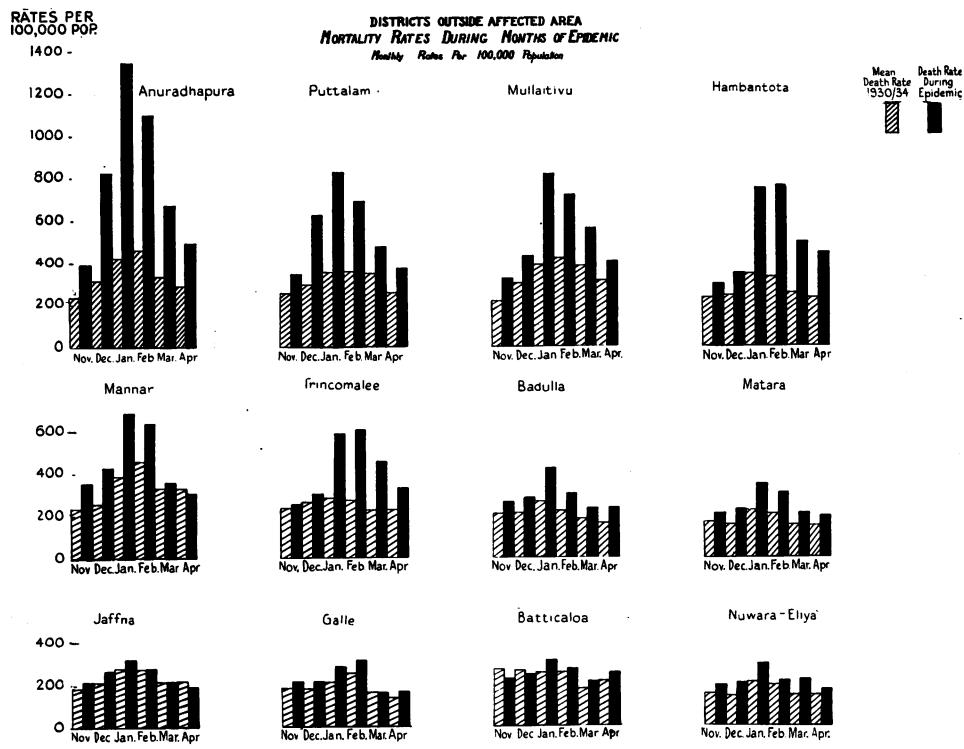

FIG. 14.

All five river basins were affected by this secondary wave and everywhere a peak was reached about April 27. Then there was a decline which in the case of the Kelani Valley and Kalu Ganga has continued to the present time, but in the case of the Maha Oya, Deduru Oya and Mahaweli Ganga lasted only until May 18, after which these three river valleys suffered from a further and rather greater rise which reached a peak in the second week of June. Since then there has been a steady drop in cases. The greatest intensity of the secondary wave was in the Maha Oya Valley and parts of the Mahaweli Ganga basin, but in none of the five river basins were figures approximating to those of the previous December recorded.

During the secondary wave many persons who had not previously suffered from malaria were infected, certain villages which had hitherto escaped lightly or even completely were involved, and in the Mahaweli Ganga basin new areas were invaded.

But the most interesting extension of the epidemic was upwards. Until April infections at altitudes above 2,000 ft. were uncommon and above 2,500 ft. were very rare, but in May and June malaria broke out in villages on the banks of streams situated 3,500 to 4,000 ft. above sea-level. The possibility of infection being spread by *A. maculatus* at these high altitudes was considered, but investigation by Mr. Carter's entomological assistants failed to find this mosquito, while *culicifacies* larvæ were recovered from the streams and adult *culicifacies* were obtained from the houses in the affected villages. Previously at these elevations the larvæ of *culicifacies* had rarely been found and the adults never.

The disease generally was of a much milder character during the secondary wave than during the early months of the epidemic and deaths were not so numerous. Only in the Kandy District (Mahaweli Ganga basin) did this wave of malaria lead to an increase in the number of deaths. Thus in the Kandy District deaths from all causes rose from 2,147 in April to 2,447 in May, to 2,895 in June and to 3,324 in July. In other districts the secondary wave merely slowed down the rate at which deaths were decreasing.

In the whole of the epidemic areas there has been continued improvement since the second week of June, and conditions in most parts are now approximating to normal.

Outside the epidemic area the only part of Ceylon where malaria is causing any anxiety is in and around the town of Badulla in the Uva Province. Here there is a fairly extensive outbreak, but the disease is of a mild type.

The deaths for September were down to the expected figure in Colombo, Negombo, Kalutara, Chilaw and Ratnapura districts and only slightly in excess in the Kurunegala District, but in the Kandy, Matale and Kegalle Districts they were still double the usual number. In the districts outside the epidemic area the number of deaths was only very little in excess of the expected number.

Measures taken to Deal with the Epidemic

It will be convenient to consider first the anti-larval measures taken during this epidemic. These were started in January 1935, and consisted in the elimination of breeding places by canalization work in the beds of the main rivers in the epidemic area and their tributaries, and the oiling of pools in these rivers, and in quarries, brick pits, etc., in the neighbourhood of large villages.

It was suggested by critics at this time and later that the epidemic could have been brought to an early conclusion by widespread anti-larval measures of this kind, but the magnitude of the task may be judged from the photograph reproduced (fig. 15) and from the fact that carefully drawn maps and calculations carried out at the time showed that about 7,000 miles of water-courses would have had to be dealt with. The approximate distance covered by a labourer employed in this work was one and a half miles per day at an average cost of Rs. 7 to Rs. 12 per mile, according to the distance from rail head and oil depots. The cost would therefore have been quite out of proportion to the benefit to be expected, even if the trained personnel necessary for such work had been available, which was not the case. Nevertheless, a mobile organization, consisting of locally recruited labour under trained personnel, was rapidly built up and some 300 miles of the four main rivers and their tributaries were brought under control, oiling (fig. 15) being confined to areas in the neighbourhood of large villages where *A. culicifacies* larvæ were found to be prevalent. Moreover intensive anti-larval measures against *A. culicifacies* were taken in and around Colombo in order to protect the city from malaria and as there were no legal powers for carrying out such measures emergency regulations were drafted and issued in February under the title of "The Malaria (Epidemic) Regulations 1935."

Elephants proved useful for the clearing of obstructions in river beds.

Clinical Features of the Epidemic

A description of the clinical features of this epidemic would require a paper to itself, but it may be noted that most of the cases were of the simple febrile type and responded readily to treatment, but relapses were frequent. Blackwater fever was reported on only five occasions. Most of the deaths were attributable to dysentery and severe diarrhœas and to pneumonia, which were the most important complications met with. Post-malarial œdema (fig. 16) was common in the northern half of the epidemic area and occurred chiefly in children. It was regarded as a nutritional manifestation and the feeding of children at school was undertaken as a relief measure by the Government. Gangrene of the extremities also occurred occasionally (fig. 17).

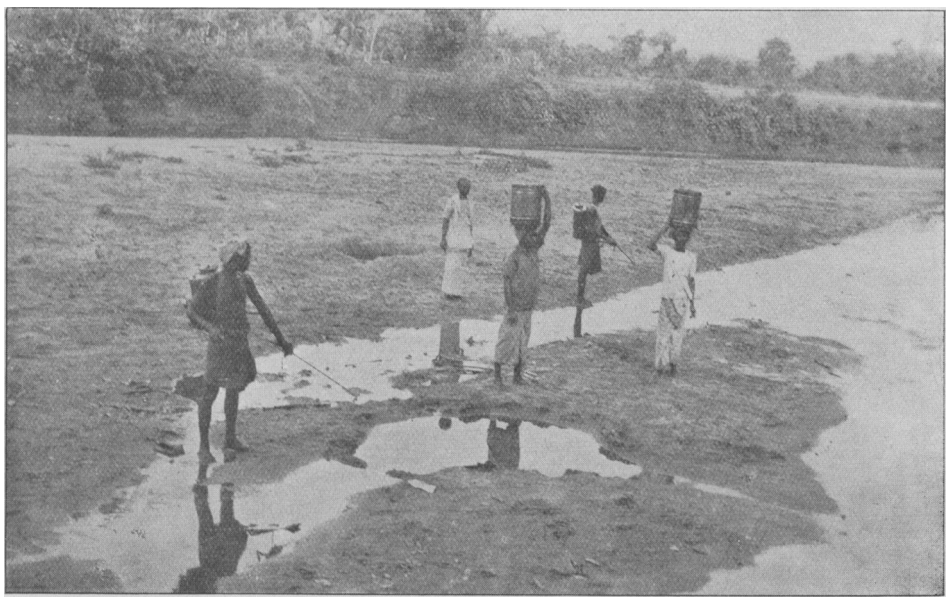

Fig. 15.—Anti-larval work in the epidemic area. Oiling pools in a river bed.

Treatment of Patients

The most important anti-malarial measure undertaken was the treatment of patients suffering from the disease. The standard treatment for adults was 7½ gr. of quinine sulphate or bisulphate in solution three times a day. This treatment had the great advantage of being safe in the hands of unqualified persons and rapidly reduced fever and relieved symptoms, but patients discontinued the use of the drug as soon as they were free from symptoms and there was consequently a high relapse rate. Plasmoquine and atebrin were frequently employed for the treatment of hospital in-patients, but were considered unsuitable for use at dispensaries and in the field where close medical supervision could not be exercised.

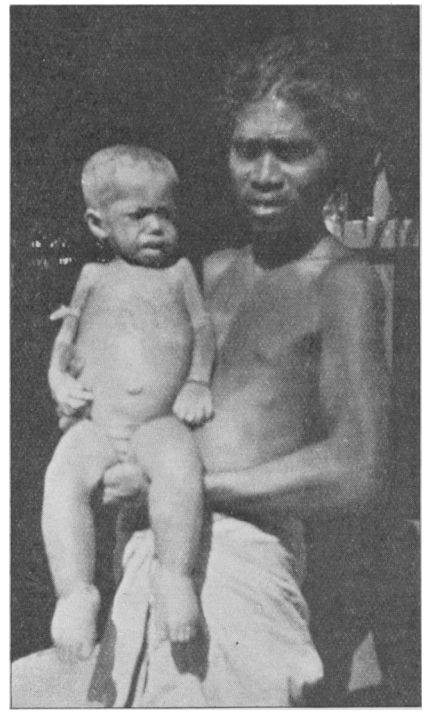

FIG. 16.—Child with post-malarial œdema.

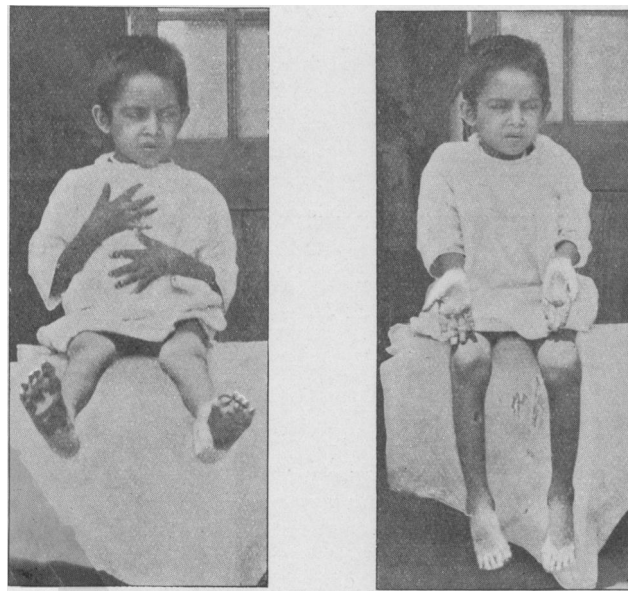

FIG. 17.—Gangrene of the extremities following malaria.

A girl, aged 12, who was critically ill with pneumonia complicating malaria was quite intolerant of quinine but responded rapidly to atebrin.

Treatment centres (fig. 19) were started in every group of badly affected villages and by the third week of December there were 690 such centres in addition to the 38 permanent hospitals and 80 central dispensaries in the epidemic area. No patient had to go more than a mile and a half for treatment. Moreover 136 estates threw open their dispensaries for the treatment of village patients and were supplied by the Government with quinine. Other voluntary agencies also arranged treatment. At the height of the epidemic there were upwards of 60,000 out-patient attendances a day at the Medical Department's institutions in the epidemic area and during the six months November to April some 33,000 lb. of quinine were issued. In view of reports to the contrary which appeared at the time, it should be noted that the Medical Department never ran short of quinine.

FIG. 18.—Interior of the boys' school at Biyagama converted into a temporary malaria hospital.

The 38 Government hospitals in the epidemic area have beds for 4,000 in-patients and accommodation for 1,000 more was provided by erecting temporary wards and arranging for the reception of malaria patients in infectious diseases hospitals and elsewhere. The congestion in the hospital wards was most acute towards the end of February when more than 3,500 patients were under treatment in the permanent hospitals. Twenty-two temporary hospitals, with accommodation for 1,300 patients were opened by the Department during January, February and the beginning of March for the treatment of malaria. They were usually housed in school buildings and an example is shown in fig. 18.

The usual method of transporting a patient to hospital was in a bullock-cart, but the loan of buses, lorries and motor cars helped greatly in this work.

Very serious cases and those complicated by dysentery or pneumonia were transferred to the nearest permanent hospital. In addition voluntary agencies and private individuals opened 43 hospitals and convalescent homes which were visited by officers of the Department, which also supplied them with drugs. Certain standards and conditions were laid down for these temporary hospitals.

By the end of August conditions had so far improved that all except two of the Government's temporary hospitals had been closed, but in the Kandy District of

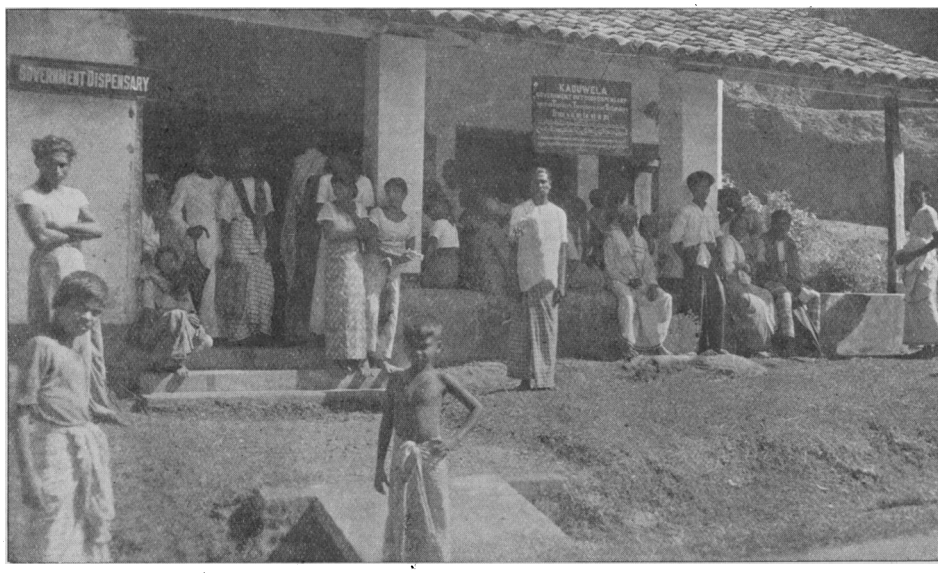

FIG. 19.—Malaria treatment centre at a Government dispensary.

the Central Province six additional temporary hospitals were opened during the secondary wave of malaria and are still functioning.

We should like to pay a tribute here to the excellent work done by the medical officers and apothecaries of the Medical Department throughout the epidemic.

[In conclusion Sir Weldon showed a short portion of the cinematograph film which he had taken of the epidemic.]

II.—V. B. Wigglesworth, M.D.

ANOPHELES CULICIFACIES AND RURAL MALARIA

At the present day it is probably denied by no one that in the genesis of epidemics of malaria many factors are at work. The degree of immunity in the population, their state of nourishment, the species and perhaps the "strain" of parasite, must all influence the occurrence and the character of an epidemic. But, equally, it cannot be denied that an appropriate mosquito in the necessary abundance is a *sine qua non* of epidemic, just as much as it is of endemic malaria. And although I

would not support an exclusively "mosquito theory" of the Ceylon epidemic as opposed to a theory taking all these factors into account, I need make no apology for confining my remarks to the mosquito factor.

As we have already heard, *A. culicifacies* is the proved carrier in Ceylon. The two other species that have been suspect are *maculatus* and *varuna*: and there are many interesting questions about them which we could well discuss. But I propose to speak only about *culicifacies*.

The Habits of *A. culicifacies*

This mosquito is well known as being the chief malaria carrier of the Indian region; and I think it is relevant to the problem of Ceylon to consider the behaviour of *culicifacies* elsewhere.

In the tea gardens of Assam during the monsoon, which is the season of malaria transmission, *culicifacies* is not a common species. The rivers and streams which it might otherwise favour are continually flushed out by the abundant rain. In the dry season the water in the rivers is reduced, and in the clear water pools that remain there are myriads of *culicifacies* larvæ. But this is also the cold-weather season; malaria transmission does not take place and *culicifacies* in this region is therefore a harmless species. As Ramsay has firmly established, *A. minimus* breeding in grassy-edged streams is the carrier of malaria.

Conditions are somewhat similar in the Anamallai Hills in South India. *Culicifacies* occurs in the rivers and streams through the estates, but during the short transmission season, from March to early June, it does not thrive there; its numbers are small and it is of no importance as a carrier of malaria. As recently proved by Measham, the carrier in this so-called "Valley of Death" is *A. fluviatilis*, a close ally of *varuna*.

In the Punjab things are very different. Throughout that area there are spots where, every year, *culicifacies* finds suitable breeding places: seepages from large irrigation canals, the small canals themselves, or pools in the bed of derelict channels. The towns and villages which lie close to these prolific and perennial breeding places are centres of endemic malaria, which occurs every year during the warm moist weeks of the monsoon. These places have much in common with the endemic areas of the dry zone of Ceylon: they too contain ruined cities. From time to time, in years of exceptional rainfall and flooding, when innumerable temporary breeding places are created, wide tracts of the Punjab become infested with *culicifacies*, and if these conditions develop after an interval during which the people have lost their immunity to malaria, devastating epidemics result. There are differences in points of detail between these Punjab epidemics and those of Ceylon. But this they have in common: the coincidence of an abundance of *culicifacies* and a non-immune state in the population.

The chief difference between these two areas is, of course, that excessive rainfall favours *culicifacies* in the Punjab, excessive drought in Ceylon. But there is nothing unusual about drought favouring *culicifacies*. In the hilly districts of the Madras Agency, for example, some of the most malarious country in India, the malaria season begins when the rivers dry up and contain residual pools. It was very striking to turn from the empty Maha Oya in Ceylon, as I did last December, and a few days later to see the same phenomenon in Central India—not as something exceptional, but as an annual event in a region of hyperendemic malaria.

Those examples illustrate what are probably the main breeding places of *culicifacies* in large numbers. But this species maintains itself also in small streams and nullahs, in irrigation wells and domestic wells, quarry pits, brick pits and borrow pits, and ornamental waters.

THE FUTURE OF MALARIA CONTROL IN RURAL AREAS

How is *culicifacies* in Ceylon to be combated? With a good organization the larger towns ought to be able to protect themselves effectively from malaria. But what about the innumerable villages in the area that is subject to these epidemics? Now I must confess that the three spots that I visited in the East where I felt least hopeful about anti-mosquito measures in rural areas were the three places—the Punjab, the Madras Agency, and Ceylon—where *culicifacies* was the chief malaria carrier.

Rural malaria is a notoriously difficult problem. But it so happens that in some regions the occurrence of malaria is intimately associated with the method of agriculture. In the flat coastal lands of Malaya, for instance, efficient agriculture necessitates clean drains and tide gates. These measures incidentally eliminate the two dangerous species of mosquito—*umbrosus* and *ludlowi*. Therefore good agriculture automatically eliminates malaria—a fortunate circumstance for that part of Malaya. And in Java the Dutch have been able to modify the native methods of fish culture so that conditions are made unfavourable for the malaria carrier, *ludlowi*, and they are attempting to do the same for rice cultivation.

It is along these lines, it seems to me, that the best hope of control for rural malaria lies—the modification of agricultural practice in such a way that the dangerous species of Anopheles are automatically eliminated. Such a policy, to be successful, will demand, of course, an intimate knowledge of the bionomics of the mosquito in question.

But in the south-west quadrant of Ceylon and in Central India the abundance of *culicifacies* bears comparatively little relation to agriculture. The drying rivers are (for the most part at any rate) a natural phenomenon, not a creation of man. And another point about *culicifacies* has recently come into prominence. It was noted in Ceylon during the epidemic that *culicifacies* larvæ occurred not only in the marginal pools, but also in the main bodies of water in the rivers. That means that the larvæ must be carried down stream, and however well one stretch of river is controlled, fresh larvæ will be continually brought down stream to it. Recently Sinton and Majid put a boom of muslin across a 60 foot stream flowing through a controlled area in the Punjab, and caught 4,000 *culicifacies* larvæ in an hour. And when I was with Mr. Senior-White in Central India we had no difficulty in showing that the same thing was happening there.

Clearly, the control of *culicifacies* in rural districts where it is breeding largely in natural waters, is going to be a formidable problem. But I came away from the East with the feeling that before we turn defeatist we want to know a great deal more about *culicifacies* and its ways. We want, for instance, to be able to define in precise physical terms how it selects its breeding places, to understand exactly those conditions which lead to its appearance in vast numbers, and to know the effect of climate on the duration of its adult life. We know that *culicifacies* is one of those species which feeds with equal readiness on man and animals; can its choice as between these hosts be influenced by the construction of the house or the disposition of cattle around it? To what extent do infected females of *culicifacies* remain sheltering in houses? In other words, is the destruction of mosquitoes in houses likely to prove an effective measure? The impression of most workers with tropical species is that it is unlikely to be of much value. But in view of recent successes claimed in South Africa and Antigua, we may have to modify this opinion.

These are only a few of the many questions that require an answer, and to my mind our best hope lies in an intimate study of the bionomics of *culicifacies*—a study equal in refinement to that expended by taxonomists upon the species and races of mosquitoes. The usefulness of such taxonomic work has often been

questioned, but it has proved of inestimable value, and I firmly believe that if the same scientific acumen were brought to bear, in the field, upon the natural history of mosquito species, we should discover, perhaps when we least expected it, the weak links in their ecological armour and new methods of breaking the chain of malaria transmission.

Colonel C. A. Gill said that, having recently returned from Ceylon, where he acted for a period of five months as expert adviser on malaria to the Ceylon Government, he was glad to have an opportunity of testifying to the extremely efficient arrangements made by Dr. R. Briercliffe, the Director of Medical and Sanitary Services, Ceylon, and the Ceylon Medical Department to cope with an unexpected and unprecedented emergency.

With remarkable celerity the whole of the medical resources of the island were mobilized, the services of all available private practitioners, dispensers, and even medical students being requisitioned, whilst steps were immediately taken to enlarge existing hospitals, and to open temporary hospitals, dispensaries, and " treatment centres " wherever necessary.

One thing that impressed him greatly was the immediate switching over of all available personnel of the Public Health Branch of the Medical Department and other specialists to assist in the treatment of the sick; this procedure would not have been possible in India, where the Medical and Public Health Departments were distinct. The policy followed in Ceylon—which he felt strongly was the right one—was that, after an epidemic had broken out, the treatment of the sick should be taken over fully and promptly by the Medical Department, and not, as in India, left to the Public Health Department to deal with, with such assistance as it could get from the Medical Department.

Other salient features of the medical scheme were the provision of free kitchens at dispensaries, the supply to hospitals and dispensaries of milk products for issue on medical grounds to children, and, on the administrative side, the establishment of an intelligence service, composed of sanitary inspectors and school teachers, to keep the department in touch with the conditions prevailing in villages, the institution of weekly reports from all hospitals and dispensaries, and the holding periodically of departmental conferences, under the chairmanship of the head of the department, at which the position was reviewed and future plans were discussed.

The Medical Department was subjected to much undeserved criticism in the press of Ceylon, but he thought that the manner in which this epidemic had been fought was not only highly creditable to the Medical Department, but was probably without precedent in the history of civil medical administration. From the epidemiological point of view the epidemic presented many unusual features, such, for example, as its association with drought and not excessive rainfall, and the occurrence of two waves of mortality instead of one, as in India.

Colonel S. P. James said that the mode of action of some of the factors causing the epidemic might possibly be explained by the results obtained in the laboratory study of induced malaria which had been conducted in England during the last ten years in connexion with the practice of malaria therapy. For example it was understood that prior to the epidemic the population at risk in the affected area was almost entirely without any immunity to malaria. Why was this lack of immunity an important factor in causing the epidemic? According to the results of laboratory work at Horton the answer was that in non-immune persons suffering from malaria, gametocytes appeared in the blood more frequently and in far greater numbers than in immune persons. The less the degree of immunity of the population

at risk the greater the number of cases with gametocytes in the peripheral blood, the greater the number of gametocytes in each case, and therefore the greater the number of cases which were "good infectors of anopheles." When anopheles fed upon these patients their stomachs became crowded with from 300 to 800 oöcysts, with the result that enough sporozoites were produced to infect many people and to keep the insect infective throughout its life. But when anopheles fed upon malaria patients possessing some immunity, the number of oöcysts found on their stomachs seldom exceeded ten, with the result that the sporozoites produced were all used up in biting two or three people and the mosquito remained infective for only a very few days. Thus, even a few heavily infected mosquitoes might cause more cases of malaria than many lightly infected ones. Lack of immunity in the population at risk was the first requirement for the production of heavily infected mosquitoes and therefore it was an important factor in the causation of an epidemic.

Dr. W. Schulemann (Elberfeld) said that the opening papers had not only greatly interested him, but had considerably helped him to get a better understanding of the state of affairs during the Ceylon epidemic. He quite agreed with Colonel Gill and Dr. Briercliffe that in an island-wide scheme for the control of a malaria epidemic—especially an epidemic of such an extent and with so many complications—the methods to be employed must be arranged to suit the circumstances and the local conditions. He fully realized the enormous work involved and the great difficulties that had to be overcome. It was therefore quite clear to him that the well-known routine methods had usually to be employed, and that there was little time to test new methods. This applied also to treatment, especially to the administration of so-called atebrin musonat, which was used for the first time during the epidemic in Ceylon. In the overcrowded hospitals the overworked physicians could not make complete observations, so that only a general impression could be gained. He therefore considered it inadvisable to make any criticism but he wished to express his personal opinion and especially to make suggestions for further work.

He agreed that the conclusions drawn by Simeons from his experimental results published in the *Indian Medical Gazette*, should be received with some reserve. The theoretical reasoning of Simeons that in peroral treatment with atebrin a period of three days would be lost until "the liver became saturated with atebrin" was not in accordance with the facts, and therefore he must disagree with it. This opinion of Simeons had been correctly criticized in an editorial notice which appeared in the *Indian Medical Gazette*.

Further, the practical conclusions arrived at by Simeons appeared to him to be too far-fetched. In order to attain a therapeutic effect it was necessary to have a certain concentration of an effective drug in the plasma and tissues, but the supposition that, by rapid production of a special high concentration of a drug in the body, it was generally possible to carry out a "therapia sterilisans magna" could not be confirmed with malarial remedies in animal experiments, and from practical experience they knew that it was not even possible in the case of salvarsan. It was a well-known fact that under certain circumstances injection treatment of malaria was indicated, if, for instance, owing to the serious condition of the patient, a very rapid effect on the parasites, and thus also on the symptoms of the acute infection was necessary. This applied both to quinine and atebrin. On the other hand, the peroral treatment was generally to be preferred to injection treatment and this peroral route should be especially chosen to prevent relapses. The findings in this direction made by Fernando, Hoole and de Silva agreed with the results obtained by Field of Kuala Lumpur.

He fully agreed with Dr. Briercliffe and Colonel Gill that it was impossible to draw any definite conclusions from the large comparative hospital experiment between atebrin musonat and quinine to which Dr. Briercliffe referred in his report. The same might be said of the field experiments carried out in Ceylon. Although very great care was exercised, an actual comparison of the results mentioned appeared to him to be impossible, since injection treatment with atebrin musonat alone was compared with injection treatment with quinine, which was, however, subsequently followed by quinine given by mouth.

Summarizing the information he had gained he had come to the conclusion that it was unfortunate to recommend the application of methods which had not been fully worked out at a time when the medical staff was overburdened with work. He entirely agreed with Dr. Briercliffe and Colonel Gill when, during the epidemic in Ceylon, they had recommended the use of atebrin musonat only in those cases in which favourable conditions for carrying out these experiments were provided.

He believed, however, that it was indispensable to carry out these experiments, since in suitable dosage he was of the opinion that owing to better local tolerance, more rapid absorption and action, atebrin musonat had many advantages.

He was sorry to note, on the other hand, that the exaggerated idea of using atebrin musonat as a panacea in all cases had caused the peroral administration of atebrin, which was so well founded, to be relegated into the background. It was generally known that a five to seven days' treatment with 0·3 grm. atebrin administered daily by mouth (especially when immediately afterwards daily doses of 0·02 grm. plasmoquine was given for three to four days) was not only able to reduce the relapse rate to a greater extent, but that also the gametocytes of *P. falciparum* were destroyed or damaged to such an extent that they were no longer able to infect mosquitoes. These facts were of particular importance both therapeutically and epidemiologically. If, in addition to the combined atebrin-plasmoquine therapy described above, it had been possible to administer 0·2 grm. atebrin on two days of the week and 0·02 to 0·03 grm. plasmoquine on one of the days on which atebrin had been given, they might have expected from recent practical experience, such as had been reported to them especially from Sardinia and North Africa, that relapses would have been diminished, and fresh infections of patients and mosquitoes, which were important from an epidemiological standpoint, would have been considerably reduced.

This full scheme would have to be considered in those places where intensive treatment and supervision was possible, or where the population was not a floating one, so that the advantages of such treatment, from an epidemiological point of view, might fully display themselves.

Where those conditions did not exist, or where there was a floating population, in his (the speaker's) opinion it would be advisable to administer atebrin only according to the work done, for example, by Dr. Green, at Kuala Lumpur.

He therefore hoped that when further experiments on these simple lines were undertaken in Ceylon, generalizations from both sides could be avoided. Further progress would then be made to the mutual satisfaction of the patient and the physician.

Dr. Kikuth (Elberfeld) who was prevented from reading a paper by lack of time, had prepared a few remarks from the point of view of the laboratory worker.

Experiments on animals had shown that the treatment of malaria by injection of atebrin musonat might hasten the therapeutic effect, but that the eventual cure could be achieved just as completely with atebrin by mouth.

Practical experience had indicated that injections of atebrin musonat were better tolerated than injections of quinine bihydrochloride, but he was unable to

commend Simeons' practice of injecting the total daily quantity of atebrin musonat at one dose. Intravenous injections, both of atebrin musonat and quinine were superfluous and dangerous. The limits of intramuscular dosage with atebrin should be defined by clinicians, and also a list of contraindications drawn up.

The question of mental derangement following the use of atebrin was a difficult one. In spite of the wide use of atebrin throughout the world, such reports had been received only from Malaya and Ceylon—13 cases were mentioned in Dr. Briercliffe's report. Possibly special circumstances might be concerned. In the Ceylon epidemic there was an unusually large number of cerebral complications of malaria (8 to 12%), the general condition of the patients under treatment was poor, and severe ankylostoma infections were often present. Further investigation of this problem was necessary.