KALA-AZAR IN KENYA.

With Particular Reference to Baringo District.

By: - John Alexander McKinnon, M.R., Ch.B.,
Medical Officer,
Colonial Medical Service,
Kenya.
Contents.

Introduction.

PART I. REVIEW OF LITERATURE AND RECORDS.
1. REVIEW OF LITERATURE.
2. SUMMARY OF LITERATURE
3. REVIEW OF OFFICIAL RECORDS
4. SUMMARY OF RECORDS.

PART II. BARINGO - THE COUNTRY AND PEOPLE.

PART III. KALA-AZAR IN BARINGO AND ELGEYO-MARAKWET.
1. INTRODUCTION
2. PRESENT SERIES.
   a. Discovery and distribution.
   b. Disease Patterns.
   c. Sandfly Surveys.
   d. Animal Infections.
3. DISCUSSION
   a. General
   b. Tribal Incidence
   c. Distribution of cases
   d. Terrain
   e. Origin
   f. Transmission
4. CONCLUSIONS

PART IV. CLINICAL STUDIES.
1. INTRODUCTION.
   a. Predisposing Causes.
   b. Age Distribution.
   c. Sex Distribution.
   d. Occupation.
   e. Seasonal Incidence.
   f. Familial Occurrence.
   g. Contact Groups.
2. CLINICAL CHARACTERISTICS.
   a. Onset and Symptoms.
   b. Physical Signs.
   c. General Condition.
   d. Emaciation.
d. Splenomegaly and Hepatomegaly.
e. Lymphatic Glands.
f. Anaemia.
g. Pyrexia
h. Hair Changes.
i. Pigmentation
j. Oedema
k. Ulcers.
l. Respiratory Infection

3. CONCOMITANT DISEASE

4. COMPLICATIONS.
a. Respiratory Infections.
b. Dysentery.
c. Jaundice.
d. Haemorrhage.

5. LABORATORY INVESTIGATIONS.
a. Serum-aldehyde Test.
b. Spleen Puncture Examinations.
c. Blood Film Examinations.
d. Urine Examination.
e. Haematological Examinations.
f. Other laboratory investigations.

6. Differential Diagnosis.

7. Treatment.
a. General
b. East Africa.
c. Results of Treatment
   i. Deaths
   ii. Discharges.
   iii. Relapses.
   iv. Follow-up of discharged cases.

Summary.

Bibliography.

Acknowledgements.
APPENDICES.


INTRODUCTION.

In 1954 in the Baringo District of Kenya a case of kala-azar was confirmed. This was the first case to be found in the district and in the succeeding six months four more cases were discovered from widely separated areas and little interest was aroused. Shortly after the writer arrived in the area in April 1955 further cases were confirmed, all living in the same locality and it was decided to institute investigations to determine the location, epidemiology and extent of the disease.

Kala-azar has been increasing steadily in Kenya since the early years of the 1939-45 War, although little attention had been devoted to it in pre-war years. A review of the literature has been made and official records have been scrutinized and a picture of the present position regarding kala-azar in Kenya is presented. In addition, official records of adjoining territories have been reviewed and brief reference is made to the present position in these territories.

A total of 71 cases have been confirmed between October 1954 and the beginning of April 1957 and, of these, 68 were treated in the main hospital of Baringo District at Kabarnet. A clinical study of these cases has been made and is reported herein. The treatment is described and the results discussed.

The purport of this thesis is as follows:-

1. The discovery and definition of the new focus of kala-azar in the Baringo District of Kenya, the measures taken to discover the distribution of the disease, and its probable origin.
2. A description of the country and the people.

3. A clinical study of 68 cases treated in hospital.

PART I.

REVIEW OF LITERATURE.
Prior to 1942 it was not recognised that kala-azar occurred in any numbers in Kenya and the only recorded case in the literature is that described by Forbes (1933) who confirmed kala-azar in an adult male of the Elgeyo tribe who was treated in Eldoret Hospital.

In 1942 a number of authors referred to an outbreak of kala-azar among African troops serving on the delta of the Omo River. This river runs into the northern end of Lake Rudolf and although the delta lies in Ethiopia, this area has been, until recently, a "no-mans' land" and Turkana tribesmen from Kenya moved their herds there for grazing at certain periods of the year. Manson-Bahr (P.E.C) (1942) referred to 30 cases of kala-azar having been infected in the Omo River Delta. Elsdon-Dew (1942) referred briefly to an outbreak of kala-azar in the Lake Rudolf area, and Erasmus (1942) mentions 20 cases of kala-azar in four months, stating that this was the second commonest cause of splenomegaly, malaria being the first, in troops serving in the East African campaign. The clinical aspects of the disease were dealt with by Tobias (1942) describing acute and subacute kala-azar in presumably the same 20 patients. Cole (1942) also referred to these cases and later in 1942 Cole et al. described the 30 patients in Nyeri Hospital reported by Manson-Bahr (P.E.C). One of the Nyeri cases was not a soldier but an Arab from Garba Tulla in the Northern Frontier District of Kenya and was treated in 1940. In 1944 Cole gives a total of 60 cases treated, including the 30 previously described. Of these 29 cases were infected in the Omo River Delta in 1941: 8 in 1941 and 23 in 1942 came from units which had served in Kenya and as far north into Ethiopia as Addis Ababa. Others had been
as far north as Marsabit and 15 came from an Animal Transport Company which had spent three months en route from Addis Ababa to Nanyuki. Wright (1943) who reported 4 cases treated in Nairobi in 1942 stated that two of these came from the Machakos District which lies to the east of Nairobi, one was infected in Ethiopia and one was a male African of the Turkana tribe.

In 1943, Anderson reviewed all cases of kala-azar in the East African Forces, of which there were 136, all Africans, who contracted the disease between February 1941 and June 1943. He gives the distribution of these cases as follows:

<table>
<thead>
<tr>
<th>Table I. Kala-azar cases in East African Forces</th>
<th>Omo River Delta</th>
<th>Uaso Nyiro River (Kenya)</th>
<th>Addis Ababa - Nairobi Road</th>
<th>Southern Ethiopia</th>
<th>Sudan</th>
<th>Wajir (Kenya)</th>
<th>Garissa (Kenya)</th>
<th>Italian Somaliland</th>
<th>Unknown.</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>87</td>
<td>12</td>
<td>11</td>
<td>8</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>12</td>
<td>136</td>
</tr>
</tbody>
</table>

He states that Maclean visited the Omo River in September 1941 and found 10 cases in the Donyiro tribe. In 3 of these the splenic pulp contained Leishman-Donovan bodies.

The case mortality in the series reviewed by Anderson was 32%, the mean
6.

incubation period 13 weeks and the majority of cases were infected during the rains.

The next report of kala-azar in Kenya was that of Heisch (1947) who refers to the case of an officer of the King's African Rifles who died at Garba Tulla in 1924, the probable diagnosis being kala-azar. He added two proven and five possible cases to the literature and concluded that Sericho on the Uaso Nyiro River was an endemic focus.

Piers (1947) refers to a further six cases of kala-azar treated in Nairobi between 1943 and 1947. Of these 5 came from Machakos District. In 1949 Clark reported kala-azar in a boy of the Wakamba tribe who lived in Embu District near the boundary with Kitui District and referred to a previous case seen who had contracted the disease in Machakos District.

Following this, Fendall (1950) reported on 6 cases, all males, from Kitui District, seen in eleven months in 1948-49 and in 1951 he reports another 13 cases occurring in 1949-50 in a period of twelve months.

The literature on kala-azar in East Africa was reviewed in detail by Fendall (1952) and he summarized the position in Kenya then as being as follows:

A. Northern Frontier District.

1. (a) Focus at Sericho and other sources on the Uaso-Nyiro River. 3 proven cases and other probables.

(b) Archer's Post on the Uaso-Nyiro River: 12 cases.

(c) Garba Tulla. One proven case and one probable.

(d) Wajir. One positive case.

(e) Garissa. One positive case.
II. The Omo River Delta and Turkana District.

87 military cases.

10 cases in Donyiro Tribe.

1 case in Turkana tribe.

B. Ukambani (Machakos and Kitui Districts)

3 proven cases.

To these cases summarized by Fendall must be added one case from Elgeyo reported by Forbes (1933) and possibly the 20 cases reported by Kirk (1939) which occurred in Sudanese troops who spent 3 days at Lokitaung in Turkana in 1931. Lokitaung lies only 30 miles from the Omo River Delta.

In his 1952 paper Fendall gives a further 31 cases which occurred in Kitui District between October 1948 and June 1951. This new focus was centred on the Ngomeni location but he carried out surveys throughout the district. He described the clinical picture of the disease as seen and suggested that the disease was more amenable to treatment than the Sudan type of the disease and found stilbamidine to be the drug of choice provided adequate medical supervision is available.

In 1953 came reports of a marked increase in the number of patients suffering from kala-azar in the Kitui area. Heisch (1953) refers to a "violent outbreak" when giving some details of the types of sandfly found on a four day visit to Tseikuru in the affected area. These included Phlebotomus clydei, P. schwetzi and P. kirkii. Carswell (1953) gave details of the numbers involved with the clinical features and laboratory
findings. Thirteen cases had been diagnosed in 1951, 303 cases with 10 deaths in 1952 and there was a still sharper increase in 1953 when 563 cases with eighteen deaths were diagnosed in the first three months of the year. It should be noted that spleen puncture was not performed on all patients but Carswell states that the serum-aldehyde test was positive in all patients. He noted the marked apathy and mental depression seen in the patients and gave the history as being fever, weakness and enlargement of the spleen of one to six months duration.

Heisch (1964) in a report on the epidemiology of this outbreak reviews the distribution of kala-azar in Kenya and refers to 2,725 cases occurring in Kitui District between September 1952 and May 1954. 70% of these were males and 57% of the cases were in the 4-18 age group. He investigated the ecological picture and discussed the results of a sandfly survey and of a search for animal vectors. He regarded a new species of P. sergentomyia (provisionally designated P.(ser) sp. nov.2) as being the most likely vector of the disease, but was not able to incriminate a particular animal vector.

In 1956 Manson-Bahr and Heisch described the clinical features of 40 cases of kala-azar from the Kitui district of Kenya. The period when these cases were seen is not stated but the writers mention that the patients come from Tseikuru, the location affected most severely in the outbreak and where cases were still occurring in small numbers. The chief symptom was pain and discomfort beneath the left costal margin. Leishmania were present in the spleen and liver most commonly and sometimes
in the lymphatic glands and the bone marrow. They found the most effective treatment to be urea stilbamidine though a combined course of pentamidine and pentostam cured many patients. Unlike Fendall (1952) they found the disease around Kitui was very resistant to treatment. Heisch et al (1956) in further studies of the sandflies in Kitui now suggested P. martinii and P. vansomerenae as being the potential vectors.

In 1955 came the report of a new focus of kala-azar in Kenya in the Baringo District. This report, by McKinnon and Fendall, described eight cases occurring between October 1954 and July 1955 and a ninth case was included in an addendum. A further paper on this focus was added by McKinnon and Fendall (1956) who described 31 cases, including the nine previously reported, occurring in seventeen months between October 1954 and February 1956. Six further cases were mentioned in a footnote. It is intended to discuss these 40 cases and a further 31 cases in greater detail in this paper.

**SUMMARY**

Prior to 1942 there was only one report in the literature of kala-azar in Kenya.

In 1942 a number of authors described an outbreak of kala-azar among military personnel serving in the North-West corner of Kenya and there were further reports in 1944 of military cases occurring there, in the Northern Frontier District of Kenya and in adjoining territories.

A number of authors described certain cases, few in number, occurring in the Northern Frontier District, in Machakos District, and in Kitui
District and the focus in Kitui District was described and demarcated in 1952.

The sharp outbreak of kala-azar in Kitui District, which commenced in 1952 has been reported by several writers and in 1955 a new focus of the disease in Baringo District was first described.
REVIEW OF OFFICIAL RECORDS.
Kenya:

When Fendall (1952) reviewed the position as to kala-azar in Kenya he found, from the Annual Reports of the Medical Dept., Kenya, that between the years 1911 and 1938 inclusive a total of 9 cases were reported. The annual reports for the war years, 1939 to 1945 were published in abridged form and no information is available on the number of kala-azar cases treated. Following the war, in the period 1945-49 a total of 63 patients were reported, the numbers rising every year from 4 in 1945 to 22 in 1949. It should be noted that these figures are for hospitals only and include only in-patients.

Fendall then searched through the annual reports of individual hospitals and found a total of 110 cases reported between 1940 and 1950: 39 cases during the war years 1940-45 and 76 cases in the post-war period.

These cases were distributed as follows, the table showing the hospitals in which the patients were diagnosed.
### Table 2. Kala-azar cases in Kenya 1940 - 1950.

<table>
<thead>
<tr>
<th>Northern Frontier Province</th>
<th>In-Patients</th>
<th>Out-patients</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wajir</td>
<td>32</td>
<td>2</td>
<td>34</td>
</tr>
<tr>
<td>Garissa</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Lodwar</td>
<td>2</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Lokitaung</td>
<td>6</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Kabarnet</td>
<td>2</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Ukambani</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Machakos</td>
<td>4</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Kitui</td>
<td>23</td>
<td>-</td>
<td>23</td>
</tr>
<tr>
<td>Central Province</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nairobi</td>
<td>25</td>
<td>-</td>
<td>25</td>
</tr>
<tr>
<td>Kiambu</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Fort Hall</td>
<td>3</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Coast Province</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mombasa</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Kilifi</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>105</td>
<td>5</td>
<td>110</td>
</tr>
</tbody>
</table>

Fendall states that these records are correct in so far as he could determine. The writer, while stationed in Kabarnet searched the in-patient records for the 2 cases reported above but no trace could be found and these may be a simple error of transcription.

He then goes on to relate these cases to the then known foci of the
disease and where it was possible to trace the history of the patient found that most were from these known foci i.e. in the Northern Province, along the Uaso-Nyiro River and in the far north of Turkana District in relation to the Omo River delta. In Ukambani the cases came from Machakos and from Kitui.

Between 1950 and 1956, both years inclusive, a further 945 cases were reported in the Annual Reports of the Medical Department, Kenya, as follows:

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1950</td>
<td>24</td>
<td>6</td>
</tr>
<tr>
<td>1951</td>
<td>24</td>
<td>1</td>
</tr>
<tr>
<td>1952</td>
<td>100</td>
<td>10</td>
</tr>
<tr>
<td>1953</td>
<td>372</td>
<td>48</td>
</tr>
<tr>
<td>1954</td>
<td>152</td>
<td>16</td>
</tr>
<tr>
<td>1955</td>
<td>143</td>
<td>7</td>
</tr>
<tr>
<td>1956</td>
<td>130</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 3.

It will be seen that the tendency for the number of cases to rise every year continued with marked increases in 1952, again in 1953 and after this falling to a level much higher than that of pre-1952.

This was due almost entirely to the epidemic of kala-azar in the Kitui district and reference was made to the Annual Reports of the Medical Officer of Health for Kitui District for the years from 1952 to 1956, both years inclusive.

In 1952 the M.O.H. reported that in 1951 a total of 13 cases had been
reported in the district but that in the latter part of 1952 the African dresser at Tseikuru in the northern part of the district reported having seen large numbers of patients suffering from kala-azar. Attention was directed to this area and as a result 80 cases of kala-azar were treated in Kitui Hospital with seven deaths and 317 probable cases of kala-azar at Tseikuru were reported.

Since Kitui Hospital lay about 100 miles from this part of the district, it was decided to build two treatment centres in the northern area at Tseikuru and Nuu. These treatment centres consisted of temporary ward buildings erected with mud and wattle walls and thatched roofs, with attached kitchens and sanitary annexes.

In 1953 the M.O.H. reported a total of 2,142 cases with 90 deaths treated in Kitui hospital and the treatment centres. Most of the deaths were due to pneumonia complicating kala-azar. Of the total number 311 were treated in hospital at Kitui, with 40 deaths. During this year a routine treatment with pentostam and pentamidine was commenced. In the section on laboratory work there are only 33 splenic punctures with Leishman Donavan bodies reported, and it appears that the diagnosis of kala-azar was being made on clinical grounds and a positive serum aldehyde reaction. By 1954 the number of cases of kala-azar was lower with a total of 795 patients and 14 deaths. Of these, 89 were treated in Kitui hospital with two deaths. 15 positive spleen puctures were reported.
In 1955 for the first time the cases reported are divided into new cases and relapses (Pasquals, personal communication). There were 177 new cases altogether and 55 relapses but no figures for deaths are available.

The total number of cases reported in 1956 was 322 of which number 21 were treated at Kitui with one death. The diagnosis at the treatment centres was still being made on clinical grounds and a positive serum aldehyde test. Some doubt as to the accuracy of the diagnosis seems to have been raised since the M.O.H. refers to a survey of patients at a treatment centre when only 10% of spleen puctions contained Leishman-Donavan bodies. However, in the statistics relating to laboratory work at Kitui, 87 spleen puctions were positive for Leishman-Donavan bodies during the year.

Thus in the six years from 1951 to 1956, both years inclusive, a total of 3,846 cases with 152 deaths was reported from Kitui District. In addition 55 relapses were reported.

The discrepancy between the figures from Kitui District and Kenya as a whole is explained by the fact that the treatment centres were not graded as hospitals, and the colony figures include only in-patients in hospital. Kala-azar is not detailed separately in the out-patients returns but is included under the heading of 'Other infectious and parasitic diseases', when these returns are being prepared at the various hospitals for transmission to Medical Headquarters.

Returning to the annual figures for kala-azar in Kenya, a number of cases were reported from hospitals outside the known endemic foci but in
practically all cases, these were traced back as having become infected in these foci and no new localities were incriminated until 1954 when two cases were reported in the annual return from Kabarnet Hospital. 22 cases were reported in 1955 as being treated in this hospital and 40 cases in 1956.

Adjoining Territories to Kenya.

Fendall in 1952 summarized the position in adjoining territories and stated that indigenous kala-azar appeared to be non-existent in British East Africa apart from Kenya. He quoted Stephenson (1940) as stating that there are probably few areas in the Sudan where the sporadic form does not occur and certainly the whole of the eastern land frontier is notorious for kala-azar.

Sporadic cases probably occurred in Ethiopia and the Western and Southern borders were strongly suspected.

With a view to establishing the present position the Annual Medical Reports of the Sudan, Uganda and Tanganyika were examined.

Sudan.

In the Sudan 638 cases with 50 deaths in 1951/52, 613 cases with 53 deaths in 1952/53, 895 cases with 72 deaths in 1953/54 and 1,106 cases with 59 deaths in 1954/55 were reported.

When the records for the Sudan are examined from 1941 onwards it is evident that the disease is increasing. The annual averages over 3 year periods were as follows:-
Table 4.

Kala-azar in the Sudan 1941-1955.

<table>
<thead>
<tr>
<th>Years</th>
<th>Annual average</th>
<th>1941-43</th>
<th>1944-46</th>
<th>1947-51</th>
<th>1951-54</th>
<th>1954-55</th>
<th>363.7 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1941-43</td>
<td>1944-46</td>
<td>1947-51</td>
<td>1951-54</td>
<td>1954-55</td>
<td>363.7 cases</td>
</tr>
<tr>
<td>&quot;</td>
<td></td>
<td>1941-43</td>
<td>1944-46</td>
<td>1947-51</td>
<td>1951-54</td>
<td>1954-55</td>
<td>363.7 cases</td>
</tr>
<tr>
<td>*</td>
<td></td>
<td>1941-43</td>
<td>1944-46</td>
<td>1947-51</td>
<td>1951-54</td>
<td>1954-55</td>
<td>363.7 cases</td>
</tr>
<tr>
<td>&quot;</td>
<td></td>
<td>1941-43</td>
<td>1944-46</td>
<td>1947-51</td>
<td>1951-54</td>
<td>1954-55</td>
<td>363.7 cases</td>
</tr>
<tr>
<td>**</td>
<td></td>
<td>1941-43</td>
<td>1944-46</td>
<td>1947-51</td>
<td>1951-54</td>
<td>1954-55</td>
<td>363.7 cases</td>
</tr>
</tbody>
</table>

* 3½ year period.
** 1 year period.

Uganda.

Prior to 1951, kala-azar had not been reported in Uganda. However, in 1951 2 male cases were reported, but no cases were reported in 1952-55 inclusive. The writer, when stationed at Kapenguria in 1957, treated 4 cases infected in Karasuk which is administered by Uganda and a further two cases from Kromoja District have been treated at Moroto hospital (Vaizey: personal communication).

Tanganyika.

No kala-azar is reported in Tanganyika until 1953 when 3 cases with one death were found. In 1954, 2 cases were reported and seven in 1955.
SUMMARY OF RECORDS.
In Kenya kala-azar has been increasing steadily since the war years and reached a peak in 1953. Since then the level has dropped but is still higher than it was prior to 1952 when there was a sharp rise due to an epidemic of the disease in the Kitui District, an already established focus. In 1954 the first reports were received of a new focus of the disease in the Upper Rift Valley.

Other foci of the disease existed on the Uaso-Nyiro River in the Northern Frontier District with sporadic cases occurring throughout the district, this being not unexpected in view of the nomadic life of the inhabitants and another on the delta of the Omo River at the northern end of Lake Rudolf.

The last known focus was in the Machakos District between Machakos and Kitui.

The extent of the new focus in the Baringo District of the Upper Rift Valley is revealed and defined in this paper.
PART II.

BARINGO - THE COUNTRY AND PEOPLE.
Baringo District (see Map 2) lies in the Rift Valley Province of Kenya, and here the walls of the Great Rift Valley, which extends from the Dead Sea in Israel in the North to Lake Nyasa in the South, run almost parallel some 40 miles apart for about 70 miles. The valley cuts through a plateau which lies at an average altitude of 8,000 feet, the floor of the valley being at an average altitude of 4,000 feet. Rising from the floor of the Valley are the Kamasia Hills which run north and south parallel to the walls of the valley, and swing round to the west at their southern end to join the western wall about 40 miles to the north of Nakuru. The Kamasia Hills are of the same geological formation as the enclosing escarpments of Laikipia on the east and Elgeyo on the west and were considered by Gregory (1896) to have been originally part of the valley floor and raised by some later volcanic upheaval than that which originally formed the valley. The lesser valley enclosed by the Elgeyo Escarpment and the Kamasia Hills is the Kerio Valley.

Baringo District is a diamond-shaped area of 3,511 square miles and lies entirely within the Rift Valley. The southern tip lies on the Equator and the distance from north to south is about 100 miles. The north–south axis of the district is Longitude 36 E, the eastern boundary being the Laikipia Escarpment and the western, the Kerio River which flows from north to south through the Kerio Valley. The district varies in width but at its maximum is about 36 miles.

To the west of the Kerio River is Elgeyo-Marakwet District which
lies partly in the Rift and partly on the plateau, the major feature of the district being the Elgeyo Escarpment which bisects the district from north to south.

The floor of the main part of the Rift Valley falls away to the north, the average height in the southern part of Baringo being 4,500 feet, falling to 2,500 feet at Karpeddo and Kollosia in the north.

The Kamasia Hills rise to a height of between 7,000 and 8,000 feet with occasional peaks rising to 8,200 feet. They fall away to the north into the plains which stretch across Turkana to Lake Rudolf.

There are two major lakes in the valley floor. Lake Baringo, discovered by Thomson in 1883, lies between Kamasia Hills and the Laikipia Escarpment, and is about 12 miles long by 8 miles wide. The lake is shallow throughout and there is no outlet.

The other large area of water is Lake Hannington, which was discovered by the bishop of that name in 1885. This lake is hidden by a fold of hills at the foot of the Laikipia Escarpment, with steep rocky shores, the water having a very high soda content and being, therefore, undrinkable.

The rivers of Baringo District, with two exceptions, all drain into Lake Baringo. The Kerio rises in the foothills of the Elgeyo Escarpment at the southern end of the Kerio Valley and, after receiving several tributaries which flow down the Elgeyo Escarpment, flows north into Lake Rudolf, a distance of 200 miles. During the dry season, however, it dries up at varying distances before reaching the lake. The other exception is a small stream, the Subukia or Sandai, which rises on the Laikipia Escarpment and flows into Lake Hannington.
The Elgeyo Escarpment from Chebloch Bridge.

The climate also varies according to the altitude. In the Kamasia, the day temperatures range between 30 and 40°F, with night temperatures between 70 and 80°F.

Soil varies much throughout the district. The Kamasia hills which, 50 years ago, were covered in primary forest, still contain pockets of forest island, although with passage of the years and the loss of the forest...
The remainder of the rivers flow into Lake Baringo, the only permanent stream being the Perkerra which rises in the Kamasia Hills to the north of Eldama Ravine. Their other rivers are intermittent and many only flow for a few weeks during the wet season. The flow of the Perkerra has dropped to as little as 3 cusecs in the dry season.

The rainfall of the district varies with the altitude. In the Kamasia Hills the average rainfall over the last forty years has been 50 inches per annum, in the low country this falls to 25 inches per annum in the south and 10-15 inches in the north. The average in the plateau country of Elgeyo-Marakwet is 40-50 inches and in the Kerio Valley 25-30 inches. The bulk of this rain falls in the period May to September. These "long rains" are followed in most years by the "short rains" in December and January, but these rains not uncommonly fail in the low country. The short rains are followed by the dry season which lasts for four months until the onset of the long rains. The growing season for vegetation, grasses and crops is from June to November, but except in sheltered pockets in the hills and near water, everything dries up after the short rains.

The climate also varies according to the altitude. In the Kamasia Hills the day temperatures range between 60 and 80 F., with night temperatures between 70 and 80 F.

Soil varies much throughout the district. The Kamasia Hills which, 50 years ago, were covered in primary forest, still contain pockets of forest loam, although with passage of the years and the loss of the forest
cover, much of this loam is being eroded and bare rock left. In the low country to the north and east the land consists of alluvial fertile plains with bare volcanic hills rising out of the plain. These plains are covered with grasses after the long rains but in the dry season are usually bare of grass and the only vegetation is acacia thorn bush.

Round the shores of Lake Baringo, especially to the south and south-east are rich alluvial plains which consist of forest soil washed down from the Kamasia Hills and Laikipia Escarpment. In this area, at Marigat, is being developed the Perkerra Irrigation Scheme, in which, initially, 3,000 acres of land will be irrigated from the Perkerra River. In the Kerio Valley is found a gray forest loam. The remainder of the district consists of a red laterite soil, which, over most of the area, is being eroded down to bare rock.

This soil erosion is continuing and is a major problem in the district. The early European explorers, Thomson (1885), Peters (1891) Von Hohnel (1894) and Gregory (1896), all of whom passed through the district, describe the country around lake Baringo as being a fertile savannah, abounding in game of all kinds, and Count Teleki, the discoverer of Lake Rudolf, is described by Von Hohnel as having killed enough game to feed his caravan of one hundred and fifty men over a period of three months. Now the numbers of sheep, goats and cattle are increasing due to control of animal diseases, and this, with the faulty agricultural methods of the people, has resulted in the disappearance of the grass cover. Consequently every shower of rain carries off the soil to leave an eroded rocky surface on which grows only a low thorn scrub.
In the hills are found the remains of the primary forest, consisting of indigenous hardwoods, podocarpus, and cedar. These are found mainly above the 6,000 feet line along with kikuyu grass, clover and bracken. Below this level is found leleshwa bush, acacia thorns and various perennial and annual grasses. These decrease with altitude until the rocky eroded thorn scrub is reached.

In the Kerio Valley the eastern or Baringo side is dryer than the Elgeyo-Marakwet side but in the main the country is not so badly eroded as that on the eastern side of the Kamasia Hills and there is more vegetation. This is partly due to the fact that much of the northern part of the valley is infested with tsetse fly, and although there is no human trypanosomiasis, or more correctly, none has been reported, the bovine form does exist there and consequently the numbers of stock are low.

THE PEOPLE.

The peoples of Baringo and Elgeyo-Marakwet Districts are all of Nilo-Hamitic stock. The Tugen of Baringo and Elgeyo and Marakwet form, with the Kipsigis and the Nandi, the Nandi or Kalenjin group of tribes and it is generally accepted that they came from the north about 100-150 years ago. They are more closely related to the Masai (Hamites) than to the Turkana, their present northern neighbours, who are Nilotic. The Suk, although in appearance and customs similar to the Turkana, are, in fact, more akin to the Tugen and their language has been described as bearing the same relationship to the Tugen language as Chaucerian English bears to modern English.
The Njems are a small offshoot of the Masai tribe.

| Population Figures. (1948 Census) |  
|-----------------------------|---|
| Tugen                      | 61,500 |
| Njems                      | 3,423  |
| East Suk                   | 6,598  |
| Elgeyo                     | 33,588 |
| Marakwet                   | 29,685 |

Table 5. Population Figures for the tribes of Baringo and Elgeyo-Marakwet.

The Tugen.

The Tugen occupy the Kamasia Hills throughout their length, the floor of the Rift Valley to the south of Lake Baringo, and the Baringo side of the Kerio Valley. They are reputed to have come from the Cherangani Hills in Marakwet round about 1860 A.D., moving into their present country. For a time a section lived on the Laikipia Plateau, intermixing and intermarrying with the Masai, before being driven back into South Baringo.

The tribe is now divided into two main sections. The Arror section live in the Kamasia Hills and the Kerio Valley. They are agriculturalists and, until the advent of British administration, practised shifting cultivation, burning the forest to clear new patches of ground when the old was exhausted. Now this is forbidden and slowly they are adopting more modern methods of agriculture. Their diet consists mainly of maize and posho, a maize flour, with occasionally millet and wild vegetables from the forest. Milk from their few cows is an occasional item in the diet and infrequently they slaughter a sheep or goat for meat.

It is unusual for a cow to be killed for meat, since these, as in so...
many parts of Africa, represent wealth and currency.

The Samorr Tugen live in the floor of the Rift Valley to the south of Lake Baringo, and are pastoralists, owning large herds of cattle, sheep and goats. They practise practically no cultivation, and like the Masai, their diet consists of blood and milk products from their stock. They eat much more meat than the Arror Tugen.

The Tugen are animists and believe in a Supreme Being - 'Assista', also in ancestral spirits. One interesting feature of the Tugen is that until recently they did not demand 'bride price', unlike the great majority of the Kenya tribes. 'Bride price' is paid by the prospective bridegroom to the father of his wife and usually consists of a number of cows, the number depending on the value of the bride.

In build the Tugen tend to be short in stature, the average being about 5'6" and slight in build, as are most hill people. They are so far not affected by European civilisation to any great extent, and tend to view any innovations with suspicion. This does not apply to those in South Baringo where there has been more contact with European farmers and with the more progressive Kikuyu tribe employed as farm labour by the European.

The Njemps.

The Njemps are a small tribe of Masai stock and are the remnants of clans defeated in a civil war among the Masai, which lasted for several years in the 1840s. The defeated clans settled at Taveta on the slopes of Mount Kilimanjaro, and on the Njemps' Flats to the south of Lake
Baringo. They were forced by the loss of their stock in the wars to become agriculturalists, and practised irrigation of their crops from the rivers. Until the railway to Uganda was built at the beginning of this century, all caravans, including those of the slave and ivory traders and the early European explorers, bought cereals from the Taveta and the Njemps, before traversing the dangerous Masai country which lay between these tribes. Both the Taveta and the Njemps were known as the Wa-Kwafi (traders or hucksters) by the caravan people.

The use of irrigation by the Njemps is diminishing although they still practise it on a small scale. They have again become a pastoral people and as such live on blood and milk products, supplemented by the produce of their irrigated fields.

The Suk.

The East Suk inhabit the northern part of Baringo District, living entirely on the plains to the north of Lake Baringo and in the northern part of the Kerio Valley. They are a section of the Suk or Pokwot tribe, which is now split into three sections - the Karasuk in Uganda, the West Suk, who live in the hills at the northern end of the Elgeyo Escarpment, and the East Suk living in Baringo. They were originally an agricultural people, who took advantage of the weakened state of the Samburu tribe living to the east, following a series of Masai raids, to become raiders themselves, thus acquiring stock. The East Suk are now completely nomadic and wander through their country in search of grass and water for their stock. They have acquired many of the customs and most of the dress of the Turkana,
but are, in fact, more closely related to the Kalenjin group of tribes. They are a tall, slim people whose diet is almost entirely of blood and milk products, the main food being a form of soured and curdled milk similar to yoghurt.

The Marakwet.

A number of the cases of kala-azar reported in this series are of the Marakwet tribe, and these are all from the Kerio Valley on the western side of the Kerio River. The Marakwet inhabit the northern part of Elgeyo-Marakwet District and form part of the Kalenjin group of tribes. The language, customs, and appearance are similar to those of the Tugen. There is, however, one important difference between the Tugen of the Kerio Valley and the Marakwet, in that the latter tribe have, in the northern part of their territory in the Kerio Valley, a well-developed irrigation system, fed by permanent streams which flow over the top of the escarpment down into the Kerio River. They are thus agriculturalists, producing in most years a surplus of grain and cereal crops, which they sell to the Turkana tribe. There is no such irrigation system on the Baringo side of the river, due to the lack of any permanent water, and, since, due to the presence of tsetse fly, the Tugen have no stock, they live near the starvation level.

Administration and Medical facilities.

Baringo, once an important halting place on the caravan route to Uganda, was bypassed by the Uganda Railway when it was built at the end of the last century, with consequent diminution in the importance of the
River crossing near Chini-ya-Milima.
district. The first government administrative headquarters were built at Eldama Ravine, which remained the provincial headquarters of the Rift Valley Province until 1928. In 1904 a station was established at Makutano on the slopes of the Laikipia Escarpment and here there was built a six-bedded hospital of mud and wattle with a thatched roof. This was staffed by two native dressers. In 1911 the administrative headquarters of the district were moved to Kabarnet in the Kamasia Hills, where it has remained since. Again a small hospital of six beds was built. In 1924, the District Commissioner reported in his Annual Report for the Baringo District for 1923, that medical facilities in the district could be called non-existent, consisting as they did of only one aged native dresser and two ruined huts.

However, in 1930, a European medical officer was posted to Kabarnet and a new permanent hospital of 24 beds built. A dispensary was built at Marigat and after a three-month safari the medical officer made a report on the district from a medical viewpoint.

In 1932, presumably because of the international trade depression, and the consequent need for economy in government expenditure, the medical officer was withdrawn, and until 1944 the hospital and district medical services were run by Indian assistant surgeons. Further dispensaries had been built at Nginyang, Emening, Maji Moto and Pokorr, these consisting of mud and wattle huts, staffed by native dressers. After 1944, Kabarnet was run by an African Hospital Assistant who was responsible for the whole district until 1951, when a permanent dispensary was built at Eldama.
Ravine and a Hospital Assistant placed in charge there, responsible for the southern part of the district. Further temporary dispensaries were built by the African District Council from their funds, at Tenges, Makutano, Kaptiony, Poi, and Kisanana.

In October 1953, a European medical officer was posted to Kabarnet, for duties as Medical Officer of Health of Baringo and Elgeyo-Marakwet Districts. A further ward with twenty-eight beds was built at Kabarnet, bringing the total number of beds there to fifty-two, and included in his charge was Tambach Hospital in Elgeyo with forty-six beds.

This officer was replaced by the writer in April 1955, who remained there until the end of 1956. It soon became evident that, owing to the financial circumstances of the African District Council which body is responsible for district services, it could not be hoped to provide any major expansion of the medical services in the district by building further dispensaries. Funds were not available for permanent buildings, and owing to the low salaries paid, the standard of dresser staff in the existing dispensaries was low. The average density of the population was 20 persons to the square mile, and the people lived in small family groups, in the case of the Suk, being almost completely nomadic. It was decided, therefore, that the best way to develop the medical service was by means of the mobile health unit which could visit the many small markets and trading centres in the district on a regular programme, the programme being
varied as circumstances required. In July 1955, the first mobile health unit was established in South Baringo with Eldama Ravine as its base, and a second based on Kabarnet commenced working in North Baringo in January 1956.

These mobile health units consisted of a suitable vehicle, in this case the four-wheel drive Landrover truck, and a staff comprising an African Hospital Assistant, a Health Assistant, a dresser and when possible, an African midwife. The programme for the unit was sent out to chiefs, headmen and traders, and also to government officials working in the district, some time in advance and they were asked to spread this information among the people of their area. When the unit arrived at a particular centre, the waiting crowd, which included relatives and those who came from curiosity, as well as the sick, were given a talk on some subject of health importance by the health assistant, and following this the sick were seen by the hospital assistant in the dispensary, if such existed, or in any other suitable building. While this was being done, the health assistant inspected the local market or trading centre, and was also available to give more particular advice to patients and their relatives on the direction of the hospital assistant. When the midwife was with the team, she would do ante-natal examinations and also child welfare clinics.

These units became very popular with the people of Baringo District who obviously gained confidence in them, due to the higher standard of
diagnosis and treatment provided, as compared to the old dispensaries, and within a very short time, the total number of patients being seen by the two units was three times as many as those being dealt with in all the dispensaries. Whenever possible the writer, who was in sole charge of the two districts and who had no other European staff, medical or nursing, joined the units on their visits, both to provide more skilled attention and to check on the working of the teams.
PART III

KALA-AZAR IN BARINGO AND ELGEYO-MARAKWET.
HISTORICAL.
The first recorded case of kala-azar from this area was that of Forbes (1933) who reported finding the disease in an adult male of the Elgeyo tribe who was treated in the Native Civil Hospital at Eldoret. No details of where this man lived are given in the paper and it is not known whether the disease had been contracted in the Kerio Valley or even elsewhere in Kenya.

Heisch (1954) refers to an African monkey trapper who developed kala-azar several months after visiting the Kerio Valley, Crescent Island in Lake Naivasha and several parts of the Kenya Coast. This man, who was, and still is, an employee of the Division of Insect-Borne Diseases of the Kenya Medical Department has been seen by the writer and states that he spent his time in the Kerio Valley at Rimo on the Elgeyo-Marakwet side of the river from which locality come Cases Numbers 52 and 53 in this series.

Fendall (1952) records two cases of kala-azar in the Annual In-Patients' Returns of Diseases from Baringo District Hospital, Kabarnet in 1943. The In-Patient Registers for this year at Kabarnet were searched by the writer and no trace of these was discovered. It is felt that the record of these cases was a typographical error during the preparation of the Annual Returns.

PRESENT SERIES.

Apart from these cases there is no record of kala-azar in this area until Mathews' observations in 1954. In October of that year he confirmed pathologically a case of kala-azar in a male child of the
Tugen tribe from Maji Moto near Lake Naivasha. He visited the
dispensary there and examined a number of local people and performed
a number of spleen punctures without discovering any further cases of
the disease.

Between October 1954 and April 1955, when the writer took over
medical charge of the area from Mathews, a further four cases of kala-
azar had been confirmed. These came from widely separated parts of
the district (see Table 7) two from the Marigat area, one from Poi
in the Kerio Valley and one from Nginyang some 45 miles to the north
of Marigat. Although the disease had not been reported before little
interest was aroused, but when, by the early part of July, a further
three cases were confirmed and these all came from the area between
Chini ya Milima and Marigat it was decided to institute medical surveys
to discover the extent of the problem.

In the latter part of July a tour of the district was made by the
writer as part of his routine duties and a total of 1,102 patients
were examined. This tour covered all the southern part of the district,
the area around Lake Baringo, and Poi in the Kerio Valley and six
suspected cases of kala-azar were discovered. At this time, he had
little experience of the technique of spleen puncture and this was not
performed on these cases in the field. These cases all reported to
Kabarnet in the succeeding months and five were confirmed pathologically
as suffering from kala-azar. The results of this initial medical
survey of the district are given in Table 6.
<table>
<thead>
<tr>
<th>PLACE</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
<th>Malaria</th>
<th>Dysentery</th>
<th>Bronchitis</th>
<th>Pneumonia</th>
<th>Tuberculosis</th>
<th>Syphilis</th>
<th>Gonorrhoea</th>
<th>Rheumatism</th>
<th>Trachoma</th>
<th>Otitis</th>
<th>Diarrhoea</th>
<th>Constipation</th>
<th>Intestinal</th>
<th>Worms</th>
<th>Abscess</th>
<th>Scabies</th>
<th>Ulcers</th>
<th>Tonsilitis</th>
<th>Suspected</th>
<th>kala-azar</th>
<th>Others</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>1,102</td>
<td>531</td>
<td>571</td>
<td>35</td>
<td>66</td>
<td>97</td>
<td>102</td>
<td>147</td>
<td>97</td>
<td>48</td>
<td>145</td>
<td>39</td>
<td>47</td>
<td>20</td>
<td>38</td>
<td>38</td>
<td>12</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>104</td>
</tr>
<tr>
<td>Malu Molo</td>
<td>8</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lolo</td>
<td>8</td>
<td>3</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ruraidi</td>
<td>5</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metemwen</td>
<td>6</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chaperen</td>
<td>7</td>
<td>2</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>53</td>
<td>50</td>
<td>27</td>
<td>19</td>
<td>13</td>
<td>14</td>
<td>27</td>
<td>12</td>
<td>99</td>
<td>47</td>
<td>49</td>
<td>73</td>
<td>49</td>
<td>107</td>
<td></td>
<td>107</td>
<td>99</td>
<td>117</td>
<td>99</td>
<td>107</td>
<td>104</td>
<td>104</td>
<td>104</td>
<td>104</td>
</tr>
</tbody>
</table>
In August 1955 the first survey specifically arranged to ascertain the extent of kala-azar in the district was held at Marigat.

The organisation of this and the succeeding surveys in the northern part of the district was as described hereafter. With the co-operation of the Administration, advance notice was sent to all local chiefs and headmen in the area. This notice gave the date and place at which the medical officer would attend and it was emphasised that all men, women and children, including those who had no complaints about their health, should attend and it was the responsibility of chiefs and headmen to ensure that this news was spread throughout their areas. The notices were sent also to African and Asian traders, and to all government officials in the locality to be visited. The survey team consisted of the medical officer, a laboratory assistant, a hospital assistant and one or two dressers. Equipment for spleen puncture and microscopic examinations was carried and also quantities of drugs and dressings for the treatment of the sick.

At the appointed place any suitable building such as a school or dispensary was used for the examinations but if such did not exist then a sheltered spot in the shade was selected and canvas screens erected. All the people were then examined by the medical officer for splenic enlargement and if this was found they were checked, other signs of kala-azar such as emaciation, anaemia, hair changes and glandular enlargement being looked for. If regarded as suspected kala-azar they were told to wait nearby and the assistance of the chief, headmen or
tribal police constable enlisted to ensure that they did not abscond. Those not suspected of suffering from kala-azar were then free to depart, but if complaining of sickness were examined by the hospital assistant and either treatment was given by him or they were held for examination by the medical officer.

After this initial assessment of all those present had been completed the suspected kala-azar patients were seen again. Medical histories were taken and more detailed and thorough examinations carried out. This normally resulted in a reduction in the number of suspects and then permission for spleen puncture was obtained from those remaining. Premedication was given and the laboratory assistant prepared the equipment for spleen puncture while the medical officer examined the patients referred to him by the hospital assistant.

When these had been dealt with, spleen punctures were then carried out by the procedure described later in this paper and preparations from the splenic pulp so obtained was stained and examined for the presence of Leishman-Donovan bodies. All patients were made to lie flat for three to four hours following the spleen puncture and as far as the writer is aware no adverse sequelae followed the performance of this procedure under these conditions.

All cases found to have Leishman-Donovan bodies in their spleens, and all doubtful cases were transported back to hospital and those who were negative were, after spending the night nearby, allowed to return to their houses the following day.
It was realized that the examination and treatment of patients suffering from other diseases was a complicating feature of the surveys but this was considered to be desirable in order to gain the confidence of the people, many of whom had little or no experience of European doctors and also in order to build up a picture of the pattern of disease in the district, which had been rather neglected from the medical point of view until October 1953 as described in Part II of this paper.

Some 230 people were seen at Marigat and by this time a total of sixteen cases had been confirmed. In addition to the centres mentioned before there had been two cases from Yatia in the foothills of the Kamasia range ten miles southwest of Nginyang; one case from Kisanana immediately to the south of Lake Hannington, one from Tot on the Marakwet side of the Kerio River, one from Kaptiony on the Baringo side of the Kerio and one from Sibillo in the Kamasia foothills.

The picture which was, therefore, developing was that cases were occurring in the eastern foothills of the Kamasia Hills with the greater number located in the Marigat-Chini ya Milima area. In addition three cases came from the northern end of the Kerio Valley.

Between the end of August 1955 and the middle of November four further cases were confirmed. These were all patients seen by the medical officer on his tour of the district in July and who had been told to report to hospital.
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Sex</th>
<th>Age</th>
<th>Date of Birth</th>
<th>Date of Onset</th>
<th>Location</th>
<th>Contact No.</th>
<th>Case Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C.C. Tugen</td>
<td>M</td>
<td>53</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Yatia</td>
<td>Contact No. 19</td>
<td>Advanced case, Died</td>
</tr>
<tr>
<td>2</td>
<td>K.C. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Marigat</td>
<td>Contact No. 18</td>
<td>Died on admission</td>
</tr>
<tr>
<td>3</td>
<td>K.C. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Relapsed.</td>
</tr>
<tr>
<td>4</td>
<td>C.K. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Died on admission</td>
</tr>
<tr>
<td>5</td>
<td>C.K. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Relapsed.</td>
</tr>
<tr>
<td>6</td>
<td>C.K. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Died on admission</td>
</tr>
<tr>
<td>7</td>
<td>C.K. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Relapsed.</td>
</tr>
<tr>
<td>8</td>
<td>C.K. Tugen</td>
<td>M</td>
<td>33</td>
<td>25.1.56</td>
<td>16.7.56</td>
<td>Chini ya Kilima</td>
<td>Contact No. 6, 7, 12</td>
<td>Died on admission</td>
</tr>
</tbody>
</table>
In the latter part of November 1955 a further kala-azar survey was made, this time in East Suk. As the roads, or rather the tracks, in this part of the district were very poor, water and food scarce, and the people suspicious of Europeans, the medical party joined forces with a tax collecting team under a district officer of the Administration who were able to make available an extra vehicle to transport petrol, water and food for the combined teams.

The route taken was north through the Kerio Valley to Kolloa, across the northern end of the Kamasia Hills to Nginyang, then north again to Akoret, which consists of one dilapidated mud and wattle shop, south past Nginyang into the eastern foothills of the Kamasia range, then east round the northern end of Lake Baringo to Tangulbei. The final place visited was Churo on the Laikipia Escarpment where many of the Suk had moved in search of grazing for their stock. The tour occupied fourteen days and 2,067 people were seen. 23 spleen puctures were done and six cases of kala-azar confirmed.

By the end of 1955, twenty-six cases of kala-azar had been diagnosed, all confirmed by spleen pucture, their distribution being as follows:-

(a) In the eastern foothills of the Kamasia Range:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marigat</td>
<td>8</td>
</tr>
<tr>
<td>Sibillo</td>
<td>2</td>
</tr>
<tr>
<td>Yatia</td>
<td>4</td>
</tr>
<tr>
<td>Nginyang</td>
<td>5</td>
</tr>
<tr>
<td>Akoret</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>20</strong></td>
</tr>
</tbody>
</table>
(b) In the Keris Valley:

Kaptumy

Kinyach

Tot

1

1

1

Total 4

c) In the Lake Hemington area:

Naji Nogo

Kiranana

Tot

1

1

Total 2

Grand Total 6

It was seen that these findings confirmed the earlier findings that the disease was being discovered along a north-south line running through the Komasla eastern side of the range.

Near Ngaratugu.
(b) In the Kerio Valley:

Kaptony 2
Kinyach 1
Tot 1  Total 4

(c) In the Lake Hannington area:

Maji Moto 1
Kisanana 1  Total 2

Grand Total 26

It was seen that these findings confirmed the earlier findings that the disease was being discovered along a north-south line running through the Kamasia foothills on the eastern side of the range. In addition a number of cases had occurred in the northern part of the Kerio Valley. No cases had occurred at an altitude greater than 4,500 feet and the individuals were from pastoral peoples.

A further kala-azar survey was carried out in January 1956 at Chini ya Milima on the road from Kabarnet to Marigat, some seven miles from the latter place. Chini ya Milima is a Swahili phrase which is translated as "under the hills" and this describes its situation at the point where the road, having wound through the hills from Kabarnet and down steep mountain slopes, debouches onto the plains which stretch across the floor of the Rift Valley to the Laikipia Escarpment. Some 320 people were examined on this survey and a further two case of kala-azar discovered.

The results of these surveys over the six months from August 1955
to January 1956 are summarized in Table 8 and it will be seen that some three thousand of the total population of 71,500 people in the district had been examined and nine cases of kala-azar discovered. In addition, some 1,100 people had been seen by the writer on his initial tour of the district and a further five confirmed cases discovered.

The tours carried out specifically as kala-azar surveys were difficult to organize due to the difficulties of communication in the district and consequent troubles in ensuring that the maximum number of the local people arrived on the specified day. The distances to be travelled on foot over difficult country by these people were considerable, being in some cases as much as twenty miles and there was a tendency for the healthy and the very ill to stay away. Hospital staff were limited and their diversion to work on the surveys lowered the standard of medical care available at Kabarnet Hospital.

On the other hand the mobile health units were now well established and increasing numbers of patients and relatives were arriving at the various centres visited on the programme of the units. The unit for South Baringo, based on Eldama Ravine, had commenced work in January 1955 and had treated 11,331 patients in the year. One case of kala-azar was discovered by this unit. The North Baringo unit was based on Kabarnet and between August 1955, when it commenced operations, and the end of the year, 2,771 patients were examined and treated, of whom ten were proven kala-azar patients.
### Table 8

**Kala-Azar Surveys - Baringo District**

<table>
<thead>
<tr>
<th>Date</th>
<th>Place</th>
<th>Tribe</th>
<th>Tugen</th>
<th>East Suk</th>
<th>Somali</th>
<th>Turkana</th>
<th>Njamps</th>
<th>Kala-azar Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>25.8.55</td>
<td>Marigat</td>
<td></td>
<td>100</td>
<td>1</td>
<td>-</td>
<td>31</td>
<td>549</td>
<td>1</td>
</tr>
<tr>
<td>15.11.55</td>
<td>Kinyach</td>
<td></td>
<td>337</td>
<td>5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>16.11.55</td>
<td>Kolloa</td>
<td></td>
<td>-</td>
<td>71</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18.11.55</td>
<td>Nginyang</td>
<td></td>
<td>-</td>
<td>650</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>20.11.55</td>
<td>Akoret</td>
<td></td>
<td>-</td>
<td>45</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>21.11.55</td>
<td>Karpeddo</td>
<td></td>
<td>-</td>
<td>345</td>
<td>7</td>
<td>19</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>23.11.55</td>
<td>Yatiya</td>
<td></td>
<td>163</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>25.11.55</td>
<td>Loruk</td>
<td></td>
<td>89</td>
<td>24</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>26.11.55</td>
<td>Tangulbei</td>
<td></td>
<td>-</td>
<td>150</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>27.11.55</td>
<td>Churo</td>
<td></td>
<td>-</td>
<td>162</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11.1.56</td>
<td>Chini ya</td>
<td></td>
<td>324</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Lilima</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Totals</td>
<td></td>
<td></td>
<td>1013</td>
<td>1453</td>
<td>7</td>
<td>51</td>
<td>549</td>
<td>9</td>
</tr>
</tbody>
</table>

**Grand Total**: 3072
The limiting factors in the length of time that each unit could spend out in the district were the need for considerable work on vehicle maintenance and the strain of working under arduous living conditions on tour. Thus the maximum number of days on tour was limited to about seventeen days in the month and when at base the medical staff did routine work in the hospital.

A further factor arising at this time in January 1956 was that it was proposed to institute a smallpox vaccination campaign in Baringo District. No comprehensive vaccination against smallpox of the population had been carried out in the district prior to this and the disease had been reported in Ethiopia in the preceding months. The African, even in the primitive tribes such as those of Baringo, is much impressed by a "sindano" or injection, which, no matter for what therapeutic reason it is given by the doctor, will cure all ailments, and confer strength and, more important, fertility on the recipient. The vaccination programme was to be carried out by the mobile units on their routine tours and it was confidently expected that this would attract considerable numbers of healthy individuals who would not have turned out for a kala-azar survey. In the event this proved correct and some 35,000 people were vaccinated during 1956.

After consideration of the above factors it was decided, therefore, that no further surveys for the specific purpose of discovering kala-azar should be carried out and that the area covered by the mobile health units was sufficiently widespread to pick up most of the kala-azar cases in the district. A mobile health unit based on Tambach commenced work in the
Eroded thorn bush near Nginyang.

Reported to the unit only to be vaccinated. It is realized that many of the individuals would have been seen on several occasions but it is probable that between 10% and 15% of the population has been missed.

The value of using the mobile health units in the search for kala-azar is shown by the numbers of cases found by these units. Of a total of 11 cases described here 9 were discovered by kala-azar survey. 4 were reported to hospital at Kabarnet, Teghach or Kibwezi Kaying and 2 were discovered by the mobile health units.

Between the 1st of January 1956 and the 30th of June 1956, one new case of kala-azar was confirmed, bringing the total to 17 and the cases were located as follows.
high country of Elgeyo and on the Elgeyo-Marakwet side of the Kerio Valley in January 1956 and thus there was medical cover for all the low country in this part of the Rift Valley. As mentioned before, the writer, in his capacity as medical officer in charge of the area, had made a practice of travelling with the mobile units periodically to supervise their work and this was now carried out at more frequent intervals, and on these occasions all the patients were seen by him personally.

Excluding the patients seen on the initial tour of the district in July 1955, some 17,000 patients were examined by the writer on kala-azar surveys and on tour with the mobile health units. The total patients seen by the three mobile units from January 1955 to December 1956 number approximately 57,500 in the district of Baringo and Elgeyo-Marakwet with their total population of 135,000. Of this number 14,300 people had reported to the units only to be vaccinated. It is realised that many of the individuals would have been seen on several occasions but it is probable that between 10% and 15% of the population has been surveyed.

The value of using the mobile health units in the search for kala-azar is shown by the numbers of cases found by these units. Of the total of 71 cases described here 9 were discovered by kala-azar surveys, 17 reported to hospital at Kabarnet, Tambach or Eldama Ravine and 45 were discovered by the mobile health units.

Between the 1st of January 1956 and the 30th of June, twenty-one new cases of kala-azar were confirmed, bringing the total to 47 and the cases were located as follows:-
(a) In the Eastern foothills of the Kamasia Range:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marigat - Chini ya Milima</td>
<td>12</td>
</tr>
<tr>
<td>Sibillo</td>
<td>2</td>
</tr>
<tr>
<td>Yatia</td>
<td>7</td>
</tr>
<tr>
<td>Ngaratugu</td>
<td>5</td>
</tr>
<tr>
<td>Nginyang</td>
<td>5</td>
</tr>
<tr>
<td>Akoret</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>32</strong></td>
</tr>
</tbody>
</table>

(b) In the Kerio Valley:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kapluk</td>
<td>2</td>
</tr>
<tr>
<td>Kaptiony</td>
<td>3</td>
</tr>
<tr>
<td>Kinyach</td>
<td>3</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>9</strong></td>
</tr>
</tbody>
</table>

(c) In the Lake Hannington area:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logumugum</td>
<td>1</td>
</tr>
<tr>
<td>Loboi</td>
<td>1</td>
</tr>
<tr>
<td>Maji Moto</td>
<td>1</td>
</tr>
<tr>
<td>Kisanana</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>4</strong></td>
</tr>
</tbody>
</table>

(d) North of Lake Baringo:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tangulbei</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2</strong></td>
</tr>
</tbody>
</table>

The disease was remaining confined, in the main, to the areas previously affected. In the Kamasia foothills a new focus had been discovered at Ngaratugu which lies to the south of Yatia and in the Kerio Valley there were two cases at Kapluk, south of Kaptiony. Outside
these areas there were two cases from Loboi and Logumugum. The latter place is only six miles in a direct line from Marigat trading centre, the major market in the area and an administrative sub-station, and the patient may well have become infected on a visit to Marigat. In the country to the north of Lake Baringo there had been one case at Tangulbei. This patient stated that he had not been out of this area but on the other hand, the grazing near Tangulbei is good and many of the Suk from the Nginyang area move to Tangulbei with their stock during the dry season. The case from Turkana District had lived at Loiya, about 30 miles north of Kollosia at the northern tip of Beringo District.

In the succeeding six months a further 19 cases were confirmed and in the next 3 months, up till 2 April 1957, five more cases, bringing the total of confirmed cases in the area to 71. These 71 cases were distributed as follows:-

(a) In the eastern foothills of the Kamasia Range:

<table>
<thead>
<tr>
<th>Location</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marigat-Chini ya Milima</td>
<td>16</td>
</tr>
<tr>
<td>Ndau</td>
<td>1</td>
</tr>
<tr>
<td>Sibillo</td>
<td>4</td>
</tr>
<tr>
<td>Yatia</td>
<td>7</td>
</tr>
<tr>
<td>Ngaratugu</td>
<td>7</td>
</tr>
<tr>
<td>Nginyang</td>
<td>9</td>
</tr>
<tr>
<td>Akoret</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>45</strong></td>
</tr>
</tbody>
</table>
(b) In the Kerio Valley:—

<table>
<thead>
<tr>
<th>Village</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chebloch</td>
<td>1</td>
</tr>
<tr>
<td>Kapluk</td>
<td>3</td>
</tr>
<tr>
<td>Kaptony</td>
<td>4</td>
</tr>
<tr>
<td>Kinyach</td>
<td>3</td>
</tr>
<tr>
<td>Rimo</td>
<td>2</td>
</tr>
<tr>
<td><strong>Tot</strong></td>
<td><strong>2</strong></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>15</strong></td>
</tr>
</tbody>
</table>

(c) In the Lake Hannington area:—

<table>
<thead>
<tr>
<th>Village</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logumugum</td>
<td>1</td>
</tr>
<tr>
<td>Loboi</td>
<td>1</td>
</tr>
<tr>
<td>Radad</td>
<td>1</td>
</tr>
<tr>
<td>Maji Moto</td>
<td>3</td>
</tr>
<tr>
<td>Kisanana</td>
<td>1</td>
</tr>
<tr>
<td><strong>Tot</strong></td>
<td><strong>15</strong></td>
</tr>
</tbody>
</table>

(d) North of Lake Baringo:—

<table>
<thead>
<tr>
<th>Village</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tangulbei</td>
<td>3</td>
</tr>
<tr>
<td>Loiya, Turkanan District</td>
<td>1</td>
</tr>
<tr>
<td><strong>Tot</strong></td>
<td><strong>4</strong></td>
</tr>
</tbody>
</table>

**Grand Total 71**

Thus although the greater number of cases came from the Kamasia foothills (63.3%) and the Kerio Valley (21.1%) the disease was being found in other centres in the Lake Hannington area.

Of the new centres involved, Ndari lies between Chini ya Milima and Sibillo, Chebloch at the point where the road from Kabarnet to Tambach crosses the Kerio River, and Rimo lies at the foot of the Elgeyo Escarpment and is probably the place where the case reported by Heisch (1954) was infected. Radad lies to the west of Lake Hannington on the main road
During the course of his work with the mobile health units, the writer examined 17,371 patients. Table 9 gives the diagnoses of these patients. Since by the middle of 1956 the units visited 47 different centres in the districts, the table has been simplified by grouping the results from these centres by geographical areas. Thus the heading "Kamasia Hills" includes all centres in the Kamasia range at an altitude of over 5,000 feet and the "Kerio Valley" all centres between the Kamasia Hills and the foot of the Elgaye Escarpment. "High Country Elgaye Marakat" describes all centres to the west of the Elgaye Escarpment at an average altitude. "Kerio Valley" the area between the Kerio Milsa and the Kerio River. Marigat road looking over Hadad.
from Nakuru to Marigat.

Disease Patterns.

During the course of his work with the mobile health units the writer examined 17,391 patients. Table 9 gives the diagnoses of these patients. Since by the middle of 1956 the units visited 47 different centres in the districts, the table has been simplified by grouping the results from these centres by geographical areas. Thus the heading "Kamasia Hills" includes all centres in the Kamasia range at an altitude of over 5,000 feet and the "Kerio Valley" all centres between the Kamasia Hills and the foot of the Elgeyo Escarpment. "High Country Elgeyo Marakwet" describes all centres to the west of the Elgeyo Escarpment at an average altitude of 7,500' and 9,000' and "Baringo Valley" the area between the Kamasia Hills and the foot of the Laikipia Escarpment on the eastern boundary of Baringo District.

Of this total of 17,391 patients, 10,336 patients lived in the areas in which kala-azar was found i.e. the Baringo Valley and the Kerio Valley, and from Table 9 it will be seen that, perhaps, surprisingly, the greatest number of patients seen were suffering from respiratory infections. These numbered 2,657 and second in importance was malaria with 2,060 cases. Trachoma and conjunctivitis patients numbered 1,396, this disease being found mainly in the northern half of the Kerio Valley and in East Suk. These diseases accounted for 6,107 (59%) of all patients seen. Other disease of importance were scabies (615 cases), diseases of bones, joints and muscles (605 cases) composed mainly of
ill-defined complaints such as fibrositis, rheumatic pains and arthritis, constipation (366) and dyspepsia (282). The more correct diagnosis for the last two groups might well be that of curiosity, although the African is notoriously impressed by the effects of purgatives, the more powerful the better. Blindness was a common complaint (275 cases) this being due in the main to the late sequelae of trachoma although senile cataracts were not uncommon. Infantile gastro-enteritis was common (245 cases) and it is the writer's opinion that this, with malaria, accounts for most of the high infant mortality rate. There were 208 cases of chronic ulcer and 203 cases of gonorrhoea.
### Table 9: Medical Survey - Baringo and Elgeyo-Marakwet

<table>
<thead>
<tr>
<th>Condition</th>
<th>Kamasia Hills</th>
<th>Kerio Valley</th>
<th>High Country Elgeyo Marakwet</th>
<th>Baringo Valley</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory Tuberculosis</strong></td>
<td>22</td>
<td>1</td>
<td>—</td>
<td>5</td>
<td>28</td>
</tr>
<tr>
<td><strong>Other Tuberculosis</strong></td>
<td>4</td>
<td>1</td>
<td>—</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td><strong>Syphilis</strong></td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>25</td>
<td>36</td>
</tr>
<tr>
<td><strong>Gonorrhoea</strong></td>
<td>16</td>
<td>15</td>
<td>9</td>
<td>188</td>
<td>228</td>
</tr>
<tr>
<td><strong>Acute Dysentery</strong></td>
<td>115</td>
<td>18</td>
<td>—</td>
<td>19</td>
<td>138</td>
</tr>
<tr>
<td><strong>Amoebic Dysentery</strong></td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Whooping Cough</strong></td>
<td>27</td>
<td>1</td>
<td>4</td>
<td>46</td>
<td>78</td>
</tr>
<tr>
<td><strong>Meningitis</strong></td>
<td>14</td>
<td>3</td>
<td>6</td>
<td>4</td>
<td>27</td>
</tr>
<tr>
<td><strong>Measles</strong></td>
<td>70</td>
<td>15</td>
<td>1</td>
<td>14</td>
<td>100</td>
</tr>
<tr>
<td><strong>Chicken Pox</strong></td>
<td>9</td>
<td>—</td>
<td>—</td>
<td>8</td>
<td>17</td>
</tr>
<tr>
<td><strong>Infectious Hepatitis</strong></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td><strong>Diphtheria</strong></td>
<td>160</td>
<td>25</td>
<td>3</td>
<td>972</td>
<td>1291</td>
</tr>
<tr>
<td><strong>Malaria</strong></td>
<td>271</td>
<td>354</td>
<td>44</td>
<td>1676</td>
<td>2375</td>
</tr>
<tr>
<td><strong>Tapeworm</strong></td>
<td>15</td>
<td>—</td>
<td>—</td>
<td>3</td>
<td>19</td>
</tr>
<tr>
<td><strong>Ascariasis</strong></td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Tinea</strong></td>
<td>18</td>
<td>10</td>
<td>—</td>
<td>15</td>
<td>43</td>
</tr>
<tr>
<td><strong>Scabies</strong></td>
<td>583</td>
<td>21</td>
<td>23</td>
<td>404</td>
<td>1221</td>
</tr>
<tr>
<td><strong>Other Infections and Parasitic Diseases</strong></td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td><strong>Post-Kala-azar</strong></td>
<td>4</td>
<td>10</td>
<td>—</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td><strong>Asthma</strong></td>
<td>22</td>
<td>3</td>
<td>—</td>
<td>19</td>
<td>44</td>
</tr>
<tr>
<td><strong>Kwashiorkor</strong></td>
<td>24</td>
<td>2</td>
<td>1</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td><strong>Anaemia</strong></td>
<td>113</td>
<td>43</td>
<td>7</td>
<td>33</td>
<td>256</td>
</tr>
<tr>
<td><strong>Other Allergic, Metabolic &amp; Blood Diseases</strong></td>
<td>17</td>
<td>4</td>
<td>5</td>
<td>21</td>
<td>47</td>
</tr>
<tr>
<td><strong>Mental Disorder</strong></td>
<td>9</td>
<td>1</td>
<td>—</td>
<td>16</td>
<td>26</td>
</tr>
<tr>
<td><strong>Epilepsy</strong></td>
<td>21</td>
<td>12</td>
<td>1</td>
<td>43</td>
<td>77</td>
</tr>
<tr>
<td><strong>Other Diseases of Nervous &amp; Sense Organs</strong></td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td><strong>Conjunctivitis</strong></td>
<td>58</td>
<td>63</td>
<td>—</td>
<td>139</td>
<td>326</td>
</tr>
<tr>
<td><strong>Blindness</strong></td>
<td>48</td>
<td>64</td>
<td>—</td>
<td>211</td>
<td>323</td>
</tr>
<tr>
<td><strong>Other Diseases of Eye</strong></td>
<td>36</td>
<td>11</td>
<td>1</td>
<td>72</td>
<td>120</td>
</tr>
<tr>
<td><strong>Diseases of the Ear</strong></td>
<td>47</td>
<td>13</td>
<td>1</td>
<td>102</td>
<td>163</td>
</tr>
<tr>
<td><strong>Cardiac Failure</strong></td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>9</td>
<td>18</td>
</tr>
<tr>
<td><strong>Pneumonia</strong></td>
<td>103</td>
<td>13</td>
<td>24</td>
<td>106</td>
<td>246</td>
</tr>
<tr>
<td><strong>Other Diseases of Respiratory System</strong></td>
<td>2638</td>
<td>613</td>
<td>25</td>
<td>1919</td>
<td>5195</td>
</tr>
<tr>
<td><strong>Dental Caries</strong></td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>14</td>
<td>23</td>
</tr>
<tr>
<td><strong>Ophthalmia</strong></td>
<td>277</td>
<td>118</td>
<td>10</td>
<td>164</td>
<td>569</td>
</tr>
<tr>
<td><strong>Deformity</strong></td>
<td>11</td>
<td>4</td>
<td>2</td>
<td>21</td>
<td>38</td>
</tr>
<tr>
<td><strong>Gastro-enteritis, (Infantile)</strong></td>
<td>121</td>
<td>34</td>
<td>6</td>
<td>203</td>
<td>370</td>
</tr>
<tr>
<td><strong>Gastro-enteritis</strong></td>
<td>61</td>
<td>11</td>
<td>13</td>
<td>23</td>
<td>114</td>
</tr>
<tr>
<td><strong>Constipation</strong></td>
<td>423</td>
<td>122</td>
<td>4</td>
<td>244</td>
<td>793</td>
</tr>
<tr>
<td><strong>Diseases of Male Genital Organs</strong></td>
<td>17</td>
<td>4</td>
<td>11</td>
<td>21</td>
<td>53</td>
</tr>
<tr>
<td><strong>Sterility (Female)</strong></td>
<td>41</td>
<td>21</td>
<td>1</td>
<td>59</td>
<td>122</td>
</tr>
<tr>
<td><strong>Cystitis</strong></td>
<td>17</td>
<td>8</td>
<td>—</td>
<td>7</td>
<td>32</td>
</tr>
<tr>
<td><strong>Diseases of Uterus &amp; Female Genital Organs</strong></td>
<td>23</td>
<td>21</td>
<td>3</td>
<td>16</td>
<td>63</td>
</tr>
<tr>
<td><strong>Normal Pregnancy</strong></td>
<td>52</td>
<td>5</td>
<td>10</td>
<td>50</td>
<td>117</td>
</tr>
<tr>
<td><strong>Abortion</strong></td>
<td>72</td>
<td>13</td>
<td>6</td>
<td>33</td>
<td>136</td>
</tr>
<tr>
<td><strong>Boils and Infections of Skin &amp; Subcutaneous Tissues</strong></td>
<td>35</td>
<td>6</td>
<td>—</td>
<td>28</td>
<td>63</td>
</tr>
<tr>
<td><strong>Chronic Ulcers</strong></td>
<td>380</td>
<td>149</td>
<td>5</td>
<td>59</td>
<td>593</td>
</tr>
<tr>
<td><strong>Other Diseases of Skin</strong></td>
<td>37</td>
<td>9</td>
<td>3</td>
<td>22</td>
<td>71</td>
</tr>
<tr>
<td><strong>Diseases of Bones, Joints &amp; Muscles</strong></td>
<td>549</td>
<td>131</td>
<td>42</td>
<td>474</td>
<td>1196</td>
</tr>
<tr>
<td><strong>Fractures &amp; Dislocations</strong></td>
<td>21</td>
<td>4</td>
<td>—</td>
<td>30</td>
<td>55</td>
</tr>
<tr>
<td><strong>Sprains</strong></td>
<td>81</td>
<td>11</td>
<td>3</td>
<td>36</td>
<td>201</td>
</tr>
<tr>
<td><strong>Foreign Bodies</strong></td>
<td>4</td>
<td>2</td>
<td>—</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td><strong>Burns &amp; Scalds</strong></td>
<td>19</td>
<td>3</td>
<td>3</td>
<td>20</td>
<td>45</td>
</tr>
<tr>
<td><strong>Other Injuries &amp; Wounds</strong></td>
<td>11</td>
<td>22</td>
<td>13</td>
<td>63</td>
<td>115</td>
</tr>
</tbody>
</table>

TOTAL: 6757, 2494, 238, 7842, 17391
Sandfly Surveys.

It is generally accepted that the sandfly is the proven vector of kala-azar and it was, therefore, considered necessary after the first eight cases of kala-azar had been confirmed, to enlist the aid of the specialized entomological organisation in the Kenya Medical Department. This organisation, the Division of Insect-Borne Diseases, was much extended on work in the kala-azar area of Kitui District and it has not yet proved possible for a full survey of the district to be undertaken. However, an entomological team was established at Marigat in September 1955 and this team has remained there since then, covering the area between Marigat and Chini ya Milima, the major focus of kala-azar. In addition, a field officer of this organisation spent a month in the Kerio Valley in 1956, catching small animals which might be intermediate hosts of the disease.

Reports establish that sandflies abound in these areas and the writer has found sandflies in most of the affected centres elsewhere in the district, but also in centres from which kala-azar has not as yet been reported.

No detailed reports have yet been made on the bionomics of the disease although Heisch et al (1956) in a report on the sandflies found in Baringo and Kitui districts states that although P. garnhami was at first considered the most likely vector in Kitui, the types now under suspicion are P. Wansomerenae and P. Martinii, and the latter type has also been found at Marigat. In earlier reports on kala-azar in Kitui, Heisch
reported that very few sandflies were found biting inside native huts and that they were found in large numbers in termite hills, in tree holes and in clefts in the rock of river beds. This is apparently also the case in Baringo as reported to the writer by the survey teams. *P.congolensis*, which was reported by Fendall (1952) as being found in Kitui and by Kirk and Lewis (1947) in kala-azar areas in the Sudan, has not been found in Baringo. Kirk and Lewis (1947) found that sandflies bred in holes, burrows and cracks in the ground and such an environment is commonly found in the low country of Baringo. Sufficient moisture would certainly be found in the dried river beds where the inhabitants obtain water for themselves and their stock by digging holes in the sand.

A possible reason for the failure to find sandflies biting inside the native huts and, in fact, to find them in any numbers in these huts may be the type of hut construction. These are usually round in shape with walls made of wooden poles and branches, and thatched roofs. In the hills the chinks between the poles are filled in with mud but in the low country suitable clay for this is very scarce and plastering of the walls is uncommon. Thus there are no dark narrow cracks, the huts are much brighter inside than the normal African hut and when the wind rises for an hour or so at sunset it blows strongly through the interior. Under these conditions it seems unlikely that sandflies would be found in large numbers.

Animal Infections.

It has been suggested that animal reservoirs of the disease may
Chini-ya-Milima.

As mentioned before, a field officer of the Ministry of Agriculture spent a month in the Kanto Valley to check existing potential tick vectors and also investigated the animals against ticks. No evidence of the findings have yet been recorded.

In his work in the district, the officer observed for increased cases of ticks and enlisted the aid of the veterinary officer and his veterinary scouts. The results of this search were negative and it was accepted that there is as yet no positive evidence of an infective reservoir.
exist, mainly in the dog, but investigations in Kenya have so far failed to incriminate any animal. Fendall (1952) suggested that there was no positive evidence of such in Kitui District although the position had not been adequately investigated. However, Heisch (1954) discussing the later large outbreak of kala-azar in the same district commented on the fact that this followed on an intensive anti-rabies campaign and there was a possibility that sandflies, normally feeding on the dogs and jackals, which had been killed in large numbers, had been thereby diverted to feeding on man. He examined gland smears from over 100 dogs and spleen smears from 30 jackals and found these to be negative for Leishman-Donovan bodies. In addition he examined numerous small animals, mainly rodents, including 330 gerbils and 30 mongooses, with negative results. Two hamsters inoculated with the pooled organs of gerbils and mongooses became infected with Leishmania, but he though that cross infection may have occurred in the laboratory.

As mentioned before, a field officer of the Division of Insect-Borne Diseases spent a month in the Kerio Valley in 1956 catching potential animal vectors and also investigated the position around Marigat. No results of the findings have yet been published.

In his work in the district, the writer searched for diseased dogs and jackals and enlisted the aid of the veterinary officer and his African veterinary scouts. The results of this search were negative and it must be accepted that there is as yet no positive evidence of an animal reservoir.
DISCUSSION.
Prior to the discovery of the first case in October 1954 at Maji Moto there were no firm suspicions that kala-azar was to be found in this part of Kenya although it was known that the disease occurred in the Omo River Delta at the northern end of Lake Rudolf some 300 miles to the north, and along the Uaso Nyiro River 150 miles to the east. It is probable that the case reported by Forbes (1933) in an Elgeyo tribesman was infected in the Kerio Valley and he suggests that as Indian troops had been used in Elgeyo District in the early years of the 20th Century, the disease may have been introduced by them. His description of Elgeyo as a "hot wet district" is not, however, strictly applicable to the Kerio Valley even during the rains. The African monkey trapper mentioned by Heisch (1954) was almost certainly infected at Rimo in the Kerio Valley, now a known focus of the disease.

The records of Kabarnet Hospital which date back to 1930 and those for Tambach Hospital, dating back to 1934, were searched for mention of kala-azar without result. In addition the hospitals at Eldoret and Nakuru were asked as to whether their records included any cases of kala-azar coming from Baringo or Elgeyo-Marakwet. Again the results were negative.

The European medical officer who was stationed in the district in 1930 spent three months on tour in the district and established a dispensary at Marigat, to reach which he had to pass through the largest of the present foci of the disease. In those days no road existed and his tour was made on foot. In his report on medical work in the district
he makes no mention of kala-azar although he includes the majority of the diseases found now.

The elders of the Tugen, Njemps and Suk tribes were questioned in detail about the disease. They were familiar with the course of kala-azar and were able to differentiate it from chronic malaria. According to them the disease always resulted in death in two to three years after onset. When asked about the length of time for which the disease had been present in the district they were more vague but the impression received from most was that it had first been seen about 10-15 years before. Supporting this view that the disease is of relatively recent introduction is that there is no specific word for the disease in either the Tugen, Suk or Njemps language although most of the other disease have specific titles. The name given to kala-azar at present can be translated as 'enlarged spleen' and could be used for any condition e.g. chronic malaria, giving rise to this sign.

Thus the cases now reported are the first 'proven' cases coming from this area. All were confirmed by the presence of Leishman-Donovan bodies in splenic smears and all doubtful cases have been excluded from the series.
Tribal Incidence.

<table>
<thead>
<tr>
<th>Tribe</th>
<th>Population</th>
<th>Kala-azar cases</th>
<th>Rate per 1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tugen</td>
<td>61,500</td>
<td>53</td>
<td>.86</td>
</tr>
<tr>
<td>Njemps</td>
<td>3,423</td>
<td>1</td>
<td>.29</td>
</tr>
<tr>
<td>East Suk</td>
<td>6,598</td>
<td>13</td>
<td>1.97</td>
</tr>
<tr>
<td>Elgeyo</td>
<td>33,588</td>
<td>1</td>
<td>.03</td>
</tr>
<tr>
<td>Marakwet</td>
<td>29,685</td>
<td>1</td>
<td>.03</td>
</tr>
<tr>
<td>Turkana</td>
<td>100,000 (estimated)</td>
<td>1</td>
<td>.01</td>
</tr>
</tbody>
</table>

From this it would appear that the incidence is much higher in the East Suk tribe than in the others. However, this tribe lives entirely in the low country and the foothills of the Kamasia range, whilst the Tugen, Elgeyo, Marakwet and Turkana inhabit the hill country in addition to the low country. The Njemps occupy the flat alluvial plains of the south of Lake Baringo, a country without hills.

Thus to be more accurate in assessing tribal incidence it would be better to assess this on the populations actually living in the country at an altitude of below 4,500 feet, since no cases of kala-azar have occurred at altitudes greater than this. However, this is not possible since the smallest geographical unit for which census figures are available is the location. This term is applied to administrative subdivisions of a district. In Baringo and Elgeyo-Marakwet the boundaries of the locations are such that these include in all but some of the Suk locations, and the Njemps location, both high and low country. Thus it is not possible to find population figures for the low country alone and it is
possible that the incidence in all tribes might be more nearly equal if only the population at risk were included.

**Distribution.**

This has been described already and it will be seen from reference to Table 7 and Map 2 that the disease is confined in the main to the eastern foothills of the Kamasia range, to the foothills of the Kamasias in the Kerio Valley, and to the broken country around Lake Hannington. No case has occurred at an altitude greater than 4,500 feet and the individual foci are restricted in area. Tracing of the individual cases to their homes established that they all lived within short distances of each other and near the centre on the map to which they have been related. These centres consist of a number of small shops, never more than six or seven and only the owners of the shops and their staff are normally resident in the centre. They are all situated on rivers which contain water during the rains and from the beds of which water can be obtained during the dry season by digging holes in the sand. The people tend to be concentrated round these centres, the intervening areas being very sparsely inhabited, and very badly eroded with practically no vegetation even during the rains.

No kala-azar has yet been found along the base of the Laikipia Escarpment with the exception of the cases at Tangulbei. The country is very similar to that of the Kamasia foothills. The cases in the Lake Hannington area are more scattered but it is likely that the same spread may be seen here as with the other centres.
The cases in the Kerio Valley are distributed mainly on the Baringo side of the River Kerio, there being 11 on this side and only 4 on the Elgeyo side.

The focus of the disease in the Marigat-Chini ya Milima area is important in view of the development of the Perkerra Irrigation Scheme mentioned in Part II of this paper. This scheme is intended to allow the resettlement of Tugen from the hills where agricultural land is scarce. These Tugen are resistant to the idea of moving into the low country which, they say with justification, is unhealthy and since three of the kala-azar patients live in the area now under development, the prospect of these hill Tugen becoming infected with kala-azar if they settle on the irrigation scheme has increased their reluctance to move.

Terrain.

This has already been described generally in Part II and reference to Map 2 will show that all the foci are located in the low-lying and drier country below 4,500 feet. This country is broken with steep rocky hills several hundred feet in height rising out of an eroded red laterite plain. The country is cut up by many small watercourses which contain water only during the rains and are dry for most of the year. The annual rainfall is low, averaging twenty inches and the climate hot, day temperatures rising to 90 - 100 F during the dry season. The vegetation consists almost entirely of acacia thorn scrub with annual grasses which last for only a few weeks after the end of the rainy season except in the
immediate vicinity of the watercourses. Practically no agriculture is practised and the people are pastoralists, owning cows, sheep and goats. The latter are particularly numerous and by their stripping of such vegetation as exists, contribute to the soil erosion. Game is uncommon now and the only wild animals to be seen are small buck, gerbils, ground squirrels, lizards and a few colonies of baboon.

This description applied to the eastern and western foothills of the Kamasias, to the foot of the Kaikipia Escarpment and the Lake Hannington region.

The Elgeyo-Marakwet side of the Kerio Valley is, however, much better watered by permanent streams which rise at the top of the Elgeyo Escarpment and flow into the Kerio. There is much more vegetation and permanent grass and the presence of tsetse fly has prevented this from being overgrazed with consequent soil erosion. Between Tot and Chesegon there are quite extensive areas irrigated by furrows which tap the rivers high on the escarpment walls and run down the steep slopes, carried across sheer rock faces in conduits made of tree trunks. The people who built these irrigation channels were not the present Marakwet and are reputed to have been the Sirikwa, one of the vanished tribes of Kenya. In this irrigated area are grown cereal crops - maize and millet in the main - and pawpaws, bananas and mangoes have been introduced in recent years.

Origin.

It may be suggested that, in view of Forbes' (1933) discovery of the disease in a man of the Elgeyo tribe that the disease has been endemic in
the area for many years, and that the discovery of over 70 cases in 2\(\frac{1}{2}\) years has been due to the presence of a medical officer. But it is not certain from the report of the Elgeyo case that this man had been infected in the Kerio Valley. Also Tambach Hospital has been in existence for almost thirty years and being only 26 miles from Eldoret by road, the practice has always been for all difficult cases to be referred to Eldoret where a European medical officer was stationed. It would be expected that if the disease was endemic at that time more cases would have followed the first. Again, a scattered and sporadic incidence of cases throughout the district would have been expected when the first cases of this present series were discovered. Instead cases were found in extending on a north-south axis in the eastern foothills of the Kamasia Hills and in the northern half of the Kerio Valley, while no cases were seen along the base of the Laikipia Escarpment in country with no apparent major differences to the foothills of the Kamasias. It was more than a year after the first cases that cases were found in this area which had in the interim period been visited regularly by the mobile health units.

The medical officer who made a medical survey of the district in 1930 left a record of his findings in which it is evident that the survey was carried out carefully and the diagnoses were detailed. He passed through at least one of the present foci but he did not find kala-azar.

During the 1939-45 War the East African Forces suffered from several outbreaks of kala-azar whilst operating in known endemic localities such as the Omo River delta and the Uaso-Nyiro River.
The Tugen have always provided a small number of recruits for the Army and the Police and according to records at Kabarnet approximately 1,000 men were on military service during the war. It is possible that Tugen served in the units in which kala-azar occurred but in view of Fendall's (1952) report that Army records were consulted without success when trying to ascertain whether Wakamba service-men had been infected in the same outbreaks, since the information required was not available, no attempt was made to trace Tugen service-men. It may well be that soldiers on leave or on discharge introduced the disease in Baringo District and this might explain the views of tribal elders that kala-azar had only been seen in the district for 10-15 years. However, if this is correct it is difficult to explain the finding that the disease is confined to a north-south axis in the district.

Although Lake Turkana lies to the north of Baringo the main supply axis to the forces operating in the south-west corner of Abyssinia ran through Eldoret and Kitale on the plateau west of the Rift Valley and it seems unlikely that troop movements account for the introduction of the disease.

In the writer's opinion it is most likely that the disease has been introduced into this area by the movement of infected members of the Turkana tribe. This tribe, which inhabits Turkana District to the north of Baringo (see Map I) is completely pastoral and family groups will travel from one boundary of the district to the other in a very short period of time. The writer knows of one family consisting of husband, two wives, and five children, all under the age of ten who covered a distance of 250 miles in two weeks. Although the Omo River delta is now
within the boundaries of Ethiopia, the Turkana still graze their herds in
the delta area and there is a recognised trade route running from the
Omo along the western shore of Lake Rudolf, south along the course of
the Kerio River then into Baringo. The route then runs along the
foothills of the Kamasia Hills as far as Marigat. At Marigat, for
many years, stock sales have been held and of recent years the Turkana
have commenced bringing their sheep and goats here for sale. A branch
of the route runs into the northern end of the Kerio Valley where the
Turkana are now buying cereals from the cultivators of the irrigated plots
between Tot and Chesegon. Further, within the past ten years, Turkana
tribesmen have been finding employment as cattle-herds on the European
ranches which lie on the plateau above the Laikipia Escarpment.
To reach this cattle ranching country, the Turkana travel on, south
from Marigat to Lake Hannington, and there climb the escarpment through
various passes. Reference to Table 8 which gives the results of the
kala-azar surveys shows that some 30 Turkana were examined at Marigat
in August 1955. These Turkana were on their way to Laikipia where the
men hoped to obtain employment and had come from the Ferguson's Gulf region
of Lake Rudolf.

The north-west corner of Turkana District was first incriminated
as an endemic focus of kala-azar by Kirk (1939) who reported 20 cases in
a patrol of the Sudan Defence Force which visited Lokitaung. Tobias
(1942) reported on 20 cases of kala-azar occurring in African troops of
the Kings African Rifles recently stationed near Lokitaung on the Omo
River. This area was also incriminated by Cole et al (1942).
Wright (1943) reported 4 cases of kala-azar treated in Nairobi in 1942. These included one male Turkana who was infected in his home district.

One Turkana (Case No. 38) is included in the present series, and since leaving Baringo District the writer has treated three Turkana suffering from pathologically confirmed kala-azar.

It is, therefore, the contention that the disease has been introduced from Turkana District, a known endemic focus of the disease, by movement of infected members of the tribe. The disease has occurred along a route used by the Turkana people in the last 10-15 years. That the disease is of recent introduction is supported by its distribution, and it is only recently beginning to spread out from the foci along the stock route; by the evidence of the local elders; and by the lack of a specific name for the disease in the local languages.

Transmission.

It is generally accepted that the disease is transmitted by the sandfly although the finding of leishmania in stools and rectal smears and in nasal smears suggests that a direct person-to-person transmission may be possible. The writer is now aware of any work on the life history of the parasite between introduction into the body and its discovery in the reticulo-endothelial system and whether infection may follow inhalation or ingestion of the parasite.

An interesting suggestion as to transmission is that of Lamborn (1956) who postulates that kala-azar may be transmitted by certain blood sucking flies of the muscid variety, more especially the cutaneous forms of leishmaniasis but possibly also the visceral forms. He discusses
the habits of these flies including those which are definitely anthropophilic and feed prior to oviposition on fresh faeces. Faeces may contain leishmania and Thomson and Lamborn (1934) showed that living leptomonads in culture are freely ingested by M. sorbeus and are excreted in viable form. Archibald and Mansour (1937) examined houseflies without finding leishmania but did not apparently examine any blood-sucking flies.

The following observations on the disease as seen in Baringo are made:

1. Sandflies are found throughout the district.

2. The full range of species found has not yet been reported but P. martinii on which suspicion has now fallen as the potential vector in Kitui has also been found at Marigat (Heisch et al 1956).

3. That in the endemic foci a number of patients have been in contact with each other (see Table 7) Archibald and Mansour (1937) noted the importance of contact in transmission of the disease.

4. That all the people affected are pastoralists and cattle owners. Blood sucking flies are often found in association with cattle. McCombe Young (1914) noted that in his cases in India 76.3% kept cattle.

5. The spread of the disease has been slow and so far there has been no tendency to epidemic spread.
PART IV

CLINICAL STUDIES.
INTRODUCTION

This part is based on the case reports of all confirmed cases of kala-azar admitted to Kabarnet Hospital between October 1954 and April 1957. During this period a total of 68 cases were treated and are now described.
Predisposing Causes.

It has been suggested by various authors that certain factors may serve as predisposing causes for an outbreak of kala-azar and that kala-azar infection may remain latent in the individual without any clinical signs or symptoms until activated by these factors. Corkill (1943) (a), discussing an outbreak of kala-azar in Nubian troops of the Sudan Defence Force, reports that an outbreak of kala-azar, on two occasions, followed severe battle experience. On the first occasion the peak of numbers affected was not related to the normal peak in the nearest place for which records were available. He suggests that this activation may have been caused by high adrenalin output following stress. He then suggested in another paper (1943) (b), that malaria is also an activating cause as are cold and high energy output. Napier and Krishnan (1931) also show this relationship between malaria and kala-azar and state that all areas in India where kala-azar is endemic are also malarious areas. Smith and Ahmed (1941) support this view, since there is no seasonal incidence in sandfly prevalence in Bihar, India.

Cold weather is incriminated by Archibald and Mansour (1937) and by Napier and Das Gupta (1931).

Corkill (1949) examined the position further and suggests that other diseases in which the common factors are acute onset, fever and infection, resulting in increased metabolism, increased protein metabolism and fatigue. He notes that the diets of many people in endemic areas in the Sudan is low in animal protein. The influenza pandemic of 1918 was suggested by
Young (1929) as predisposing to a kala-azar epidemic in 1918 in Assam.

Kirk (1939) discussing the disease in the Sudan notes that kala-azar is rural in distribution.

There are no urban communities in Baringo and thus the disease is entirely rural in distribution. The diet of the majority of the patients was composed of meat and blood and milk products and was therefore high in animal protein. However, malaria is endemic throughout the low country of Baringo and Elgeyo-Marakwet, having its maximum incidence during the rains.

In 1955, the rains, although in total quantity not much below the annual average, were badly distributed in that very little fell in June. Thus crops already planted in April and May withered and died as did the new grass. The rains of July, August and September then ran off the ground because of the lack of vegetation to hold it in the soil. Thus stock were in poor condition and milk and blood products scarce and normal grain supplements also in short supply. Although no frank malnutrition was seen it was considered that famine relief measures might have to be instituted before the 1956 crops were harvested. In the event, this was not necessary but it is interesting to note that although the first kala-azar surveys were done in 1955 and the mobile health units also commenced their work in this year, the number of patients who reported the onset of the disease as being in 1956 (28) was much greater than the similar figure for 1955 (10).

There was no major outbreak of infectious disease in the Baringo
area during the years 1953-1956 but it is not possible to say whether there had, in fact, been increases in the incidence of the infectious diseases since prior to 1953, in the absence of a medical officer, the only records from the district, excluding the hospital statistics, were those of the dispensaries. These diagnoses, being made by ungraded African dressers, are not to be relied upon.

Age Distribution.

Table II. Age Distribution.

<table>
<thead>
<tr>
<th>Age-in years.</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>3</td>
</tr>
<tr>
<td>5-9</td>
<td>23</td>
</tr>
<tr>
<td>10-14</td>
<td>20</td>
</tr>
<tr>
<td>15-19</td>
<td>5</td>
</tr>
<tr>
<td>20-24</td>
<td>13</td>
</tr>
<tr>
<td>25-29</td>
<td>3</td>
</tr>
<tr>
<td>30-34</td>
<td>2</td>
</tr>
<tr>
<td>35-39</td>
<td>2</td>
</tr>
</tbody>
</table>

From the above table it is seen that 64.9% of the cases seen were in the 0-14 age group, 25.3% in the 15-24 years age group and 9.9% in the 25-39 age group. The youngest cases were 3 years and the oldest 39 years. This finding that the disease affects mainly young children and young adults is in conformity with other reports of the disease in Kenya. Fendall (1952) reported 52% in children and 19% in young adults in 31 cases from Kitui District, Heisch (1954) 57% in the 4-18 years age group in a series of 2,725 cases from Kitui District and Heisch and Manson-
Bahr (1956) reported that "nearly all were in the 6-20 age group" in a further series of 40 cases from the same district. Heisch (1947) reporting on kala-azar in the Northern Province of Kenya found 57.3% of cases in the 4-18 years age group. McKinnon and Fendall (1956) reporting on 34 cases of the disease in Baringo District (these cases are included in this series) found 65% in children, 23% in young adults and 12% in adults.

In the Sudan, Kirk (1939) found that the disease affected mainly those in late childhood and again (1956) he reports that in the Northern Sudan 30% were in the 0-15 age group and 33% in the 16-25 age group. At Kapoeta in the Southern Sudan, which is not far north of the Kenya border, these figures change to 61% in the 0-15 age group and 20.4% in the 16-25 years age group. It appears that the age distribution in the Southern Sudan is similar to that in Kenya.

Napier (1925) found 45% of the cases between the ages of 8 and 20 years in Calcutta and he and Das Gupta (1931) report 71% of cases under the age of 20 years. Again, Napier and Krishnan (1933) found in Madras that 83% of cases were under the age of 25 years.

From this it would appear that the findings in the present series are similar to those already reported in E. Africa and that the E. African age distribution is much the same as that in India, the disease affecting mainly children and young adults. This is confirmed by Heisch (1954) who quotes a personal communication on this point from Dr. L.E. Napier.

It should be noted, however, that the age distribution in the Mediterranean form of the disease is very different. Adler and Theodore
(1926) reported that in Catania 88% of cases were under the age of 4 years and in Pittaluga 90% below that age. Debono (1947) states that in Malta 93% of patients were below the age of 5 years. Thus the disease in the Mediterranean affects infants predominantly.

**Sex Distribution.**

**Table 12. Sex Distribution.**

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-14</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td>15-24</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>25-39</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>45</strong></td>
<td><strong>26</strong></td>
</tr>
</tbody>
</table>

Of the 71 cases of kala-azar confirmed, 63.4% were male and 36.6% female. These figures are in agreement with other series reported from Kenya. Fendall (1952) reports 80% in males, Heisch (1954) 70% in males, Heisch and Manson-Bahr (1956) 77.5% males and McKinnon and Fendall (1956) 71%.

From the Annual Medical Reports of the Medical Officer of Health, Kitui District, Kenya, for the years 1953, 1954, 1955 and 1956 the sex incidence of kala-azar cases reported during these years were given as:

- 1953: 68.5% Male.
- 1954: 73% Male.
- 1955: 76.8% Male.
- 1956: 66% Male.

Kirk (1939) writing on kala-azar in the Sudan stated that males were affected more frequently than females, the difference being less marked in
young children.

In India Napier (1926) reported that 76% of kala-azar cases seen in the Calcutta School of Tropical Medicine were males, and of cases seen in Assam 70% were males.

Thus there seems to be little difference in the sex distribution of kala-azar in the Sudan, India and Kenya.

When the sex distribution in the 0-11 years age group is examined it is seen that 60.9% were in males. This does not agree with Kirk's (1939) findings in the Sudan as mentioned above. Fendall (1952) found that the sex distribution in the 0-15 years age group was 75% male.

This difference between the sexes may be only an apparent one in that in an area such as Baringo and Elgeyo-Marakwet where the services of a medical officer have been available for a short time only, the women and girls tend to be much more conservative than the men in coming forward for medical treatment. McKinnon and Fendall (1956) reporting on the first 34 cases of this series, found 74.1% males while in the full series of 71 cases only 63.8% were males. This may be coincidental but, on the other hand, may well be due to an increase of confidence among the women as successfully treated cases began to return to their homes from hospital, with the result that they came forward more readily.

Napier, in the discussion on Heisch's paper on Studies in Leishmaniasis in East Africa (1954) stated that the preponderance of male patients disappeared when patients were sought out in villages by house-to-house visits. This, no doubt, referred to a paper (Napier and Das Gupta 1931)
reporting a series of 367 cases in a five year survey in an endemic area, when 52.7% were male and 47.3% female.

**Occupation.**

Kirk (1956) in the Sudan noted that the disease was rural rather than urban and was found mainly in those whose work entailed long periods in open country. Anderson (1943) reviewed 136 cases of kala-azar in the E. African Forces and found that 87 had occurred in the Omo River Delta the north end of Lake Rudolf, 12 on the Uaso Nyiro River and 11 in an Animal Transport unit which took three months to travel from Addis Ababa to Nairobi. In all these cases the troops were on active service conditions which would entail living in the open in country which is very poorly covered with vegetation and thinly populated. Similarly Kirk (1939) records the case of a Sudanese patrol which spent some time on a trip from Kapoeta into Northern Turkana, spending 3 days at Lakitaung, which lies only 30 miles from the Omo River Delta. Of this patrol 20 men (27% of the party) developed kala-azar after their return.

In a sandfly survey in the Kitui District of Kenya Heisch (1954) found no evidence of sandflies biting man in huts or other buildings with the exception of *P. kirkii* which has not been incriminated as a factor. He visited villages at night and saw no sandflies feeding on children or adults. He suggests that most infections are not contracted in huts but out in the bush.

From the tables of age and sex distribution (11 and 12) it is seen that over the age of 14 years there are 17 cases in males and 8 cases in females. In the tribes of Baringo and Elgeyo-Marakwet District,
the people who live in the low country where cases of kala-azar are found (see Map 2) are pastoral and very little cultivation is done. Consequently, the women of these tribes, who in agricultural tribes would so most of the work in the fields, instead spend most of their time in their homes as do the very young children. However, from the age of 5 years or so both boys and girls are employed in herding the stock (cattle, sheep and goats) and spend all day in the open, returning often just before dusk and covering fairly extensive areas in search of water and grass. Similarly the men herd the stock and travel long distances to meet their friends. The average age of girls at marriage is 16-18 years and after marriage their activities would be confined to the environs of their homes.

Thus it may well be that these variations in sex and age distribution seen in this series can be explained by the occupational differences in the groups. One interesting case is No. 35 in the list of patients (Table 7) who is a stock trader by occupation, and although his home is in the hills, spends much time visiting various centres in the low country to buy stock. He became ill in November 1955 and had spent three months from July to September 1955 at Ngaratugu in the low country before returning to his home at Kabarnet. He has, therefore, been shown on the Distribution Map (Map No. 2) as having contracted the disease at Ngaratugu.
Figure 1: Seasonal Incidence

Month of onset of disease

Rainfall - Mean monthly readings at Marigat & Nginyang
Seasonal Incidence.

Figure 1 has been drawn up utilizing rainfall figures obtained from two recording stations in the low country of Baringo, at Marigat and at Nginyang. From February 1954 daily recordings were made by the European staff of the Perkerra Irrigation Scheme at Marigat, prior to this, records were kept by the African dresser at Marigat Dispensary. The readings at Nginyang were made by the dresser at Nginyang Dispensary. Since Marigat and Nginyang are situated at the southern and northern ends of the area from which the majority of the cases in this series have come, it was decided to average these figures to give a picture of the rainfall in the low country. It will be seen that the wet season lasts from April to August, the short rains having failed every year except at the end of 1955.

All patients were questioned as to the length of their illness at admission and their answers were used to produce the nomogram shown. It should be realized that their answers may be inaccurate for the following reasons. Firstly, the African's concept of time is very vague and without the fixing of the length of time by some event known to the questioner, such as the visit of a Government official, the figures may well be wildly inaccurate. Many patients stated initially that they had only been ill for a period of weeks when it was evident from their clinical condition that this was not so.

Secondly, since many of the patients were children, unless a parent or older relative was available to give the history, it was extremely
difficult to obtain any information whatsoever.

To further complicate the position, one tribe, the Suk, when using the word which is normally translated into English as "year" in fact mean a period of between seven months and five months which is related to the wet and dry seasons.

However, it is felt that the information as recorded in Table 7 and Figure 1 is reasonably accurate since much time was spent on this point, patients being submitted to detailed questioning by senior African members of the staff belonging to the local tribes.

From Figure 1 it will be seen that while there is no firm relationship between rainfall and onset of disease, 30 cases (42%) occurred during the months April to August and if those occurring in January 1956, which was a wet month, are added this becomes 35 of the cases (49%) occurring during the rains.

Heisch (1954) shows 1,301 of his 2,725 cases i.e. 47.7% as reporting during the rains but these figures are, of course, not comparable.

Napier and Das Gupta (1931) state that the majority of cases occur during the cold weather in India and Kirk (1939) in the Sudan reported that the risk of infection was greatest during the rains. Cole (1944) reporting on kala-azar in the East African Forces found that the highest rate of infection was during the four months March to June i.e. during the rains. Similarly Fendall (1952) found that the onset of the disease was during and immediately after the rains.
Corkill (1949) also reports that the seasonal peaks in India and the Sudan occur in the cold season. In this connection it may be assumed that the cold season is also the wet season, such being the case in Kenya.

Thus the position in this series is similar to that reported in the Sudan, in India and elsewhere in Kenya.

**Familial Incidence.**

Familial incidence of kala-azar has been reported as being common in the Indian form of the disease, but uncommon in the Mediterranean form by Adler and Theodore (1931).

Kirk (1939) found that it occurred in the Sudan but stated that there might be a long gap between cases occurring in the same family. In Fendall's (1952) series of 31 cases he found one case of familial incidence. This was two stepbrothers, living in the same house, who developed kala-azar.

McKinnon and Fendall (1956) reporting on the first 34 cases of the present series, noted one case of familial incidence, to wit, Case No. 13, a child admitted in August 1955 and his father Case No. 30, who was admitted in February 1956. The probable onset of the disease in the child was August 1954 and in the father, November, 1955, three months after the admission of the child to hospital. To this must now be added two more examples of familial incidence. The first is Case No. 8 and Case No. 50 who were sisters. Case No. 8, a girl of 7, was admitted in July 1955 and her sister, aged 3 was admitted in August 1956. The onset of the disease in Case No. 8 was probably in April 1955 and in her younger sister in June.
1956, that is, seven months after the discharge of Case No. 8 from hospital. Her sister had been seen again at the hospital in April 1956 when the aldehyde test was negative and no Leishman-Donovan bodies were seen on examination of the sternal marrow. It seems unlikely that she was the source of infection for her younger sister. The remaining case of two members of the same family are Case No. 40, a girl of 9 who was admitted in March 1956 and Case No. 66, her brother, aged 11 who was admitted in November 1956. The onset of the disease in Case No. 40 was probably in February 1956 and in Case No. 66, in September 1956, that is, five months after his sister was admitted to hospital.

It does not seem possible to draw any firm conclusions from these cases. It may be that in the first instance, assuming an incubation period of 3-4 months (Anderson, 1943, Cole, 1944) the child (Case No. 13) would have been the source of infection of his father (Case No. 30). This, however, is unlikely in the second and third instances (Cases 8 and 50) and (Cases 40 and 66).

Contact Groups.

In contrast to the lack of familial incidence reported above, there was evidence of contact between 29 of the 71 cases (40.8%). These have been divided into eight groups from six localities. In each group each individual has been in contact with at least one other member of the group, and, in some cases, as will be seen in Table 7, up to four other members of the group. Contact is defined as having played with, worked alongside, or visited at one or other's home on a number of occasions.
Thus in the 17 cases which occurred in the Marigat-Chini ya Vilima area (see Map 2) there are two contact groups.

Group A consists of ten individuals, Cases No. 2, 6, 7, 12, 15, 20, 27, 28, 26 and 45 i.e. 10 cases.

Group F consists of the sisters, Cases No. 8 and 50 reported above.

In the Yatia area, from a total of seven cases, five fall into Contact Group B. This consists of Cases No. 9, 13, 19, 30 and 37. It should be noted that Cases 13 and 30 are father and son as reported above, the son being a contact of Case No. 9.

Group C includes two of the seven cases from Ngaratugu, Cases No. 31 and 34. Also from Ngaratugu is Group H which consists of the brother and sister, Cases 40 and 66, reported under Familial Incidence.

From Kapluk in the Kerio Valley, where three cases have been reported, two from Group E. These are Cases No. 44 and 48.

The last contact group is Group G. These are Cases 62, 63, and 64 from Nginyang forming three cases out of the nine from that area.

In order to facilitate consideration of these contact groups Figure 2 and Figure 3 have been drawn up for the two largest groups A and B.

Figure 2 shows the period of time between the onset of the disease as stated by the patient, and his or her admission to hospital for all the cases in Group A. Similarly Figure 3 shows this for Group B.

It is reasonable to draw the conclusion from these figures, that allowing for inaccuracies in the date of onset and the incubation period
that each case was in contact with another proven case of kala-azar before
the admission of that case to hospital for treatment. For example, on
reference to Table 7 it will be seen Case No. 6 stated that she had been
in contact with Cases No. 2, 7, 12 and 20 before admission to hospital,
and reference to Figure 2 shows that such could have been the case and that
thereby the infection may have been transmitted from patient to patient.
CONTACT GROUP A.
MARIGAT - CHINI YA MILIMA.

FIGURE SHOWING PERIOD OF TIME BETWEEN ONSET OF DISEASE AND ADMISSION TO HOSPITAL FOR EACH CASE IN GROUP.
Figure 3. Contact Group B

Yatia

Figure showing period of time between onset of disease and admission to hospital for each case in the group.
CLINICAL CHARACTERISTICS.
Onset and Symptoms.

The majority of the cases gave a history that the disease had commenced in a manner not unlike an attack of malaria in the semi-immune, with fever, headache, joint pains and slight enlargement of the spleen. In fact, many thought that it was malaria and only became suspicious that this was not the case when the spleen, instead of regressing, increased in size and recurrent attacks of fever continued. Enlargement of the spleen and its behaviour during malarial attacks is known by the Tugen and Suk people and they, therefore, knew that this disease was different to malaria. These symptoms were not acute and did not seem to be incapacitating to any great degree in the beginning, thus resembling more the semi-acute onset described by Tobias (1942). The average duration of symptoms before admission to hospital was 8.7 months with extremes of one month and two years.

Table 13. Duration of Disease before admission

<table>
<thead>
<tr>
<th>Duration of disease before admission</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 3 months</td>
<td>20</td>
</tr>
<tr>
<td>4-6 months</td>
<td>19</td>
</tr>
<tr>
<td>7-12 months</td>
<td>14</td>
</tr>
<tr>
<td>13-24 months</td>
<td>14</td>
</tr>
<tr>
<td>Unknown</td>
<td>4</td>
</tr>
</tbody>
</table>

These figures were examined in detail to see if there was any tendency for patients in any particular age group to report earlier for admission, but no significant differences were seen.

Fendall (1952) reported an average duration of symptoms before admission of seven months in his cases from Kitui, and with gradual onset of the
CASE NO. 52.

Male aged 13 yrs of the Elgeyo tribe.
Similarly Manson-Bahr and Heisch (1956) described a gradual onset in their series but with an average duration of symptoms of 3-4 months. It should be noted, however, that their cases were seen at the end of the Kitui epidemic of 1953 and 1954 when presumably the Wakamba people were familiar with the disease and its symptoms.

The presenting symptoms were as shown in the table below.

Table 14. Presenting symptoms.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Nos.</th>
<th>Symptoms</th>
<th>Nos.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of weight</td>
<td>59</td>
<td>Diarrhoea</td>
<td>13</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>67</td>
<td>Epistaxis</td>
<td>33</td>
</tr>
<tr>
<td>Enlarged Abdomen</td>
<td>53</td>
<td>Rigors</td>
<td>22</td>
</tr>
<tr>
<td>Fever</td>
<td>56</td>
<td>Jaundice</td>
<td>5</td>
</tr>
<tr>
<td>Cough</td>
<td>56</td>
<td>Pain in joints</td>
<td>36</td>
</tr>
<tr>
<td>Pain in splenic area</td>
<td>65</td>
<td>Pain in chest</td>
<td>41</td>
</tr>
<tr>
<td>Weakness</td>
<td>22</td>
<td>Bleeding from gums</td>
<td>21</td>
</tr>
<tr>
<td>Headache</td>
<td>43</td>
<td>Liver enlargement</td>
<td>17</td>
</tr>
<tr>
<td>Debility</td>
<td>2</td>
<td>Sore throat</td>
<td>17</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>16</td>
<td>Oedema</td>
<td>11</td>
</tr>
<tr>
<td>Increased appetite</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breathlessness on exertion</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain over liver</td>
<td>14</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The typical case was ambulant and complained of enlargement of the spleen, with pain in the splenic area, loss of weight, weakness, fever and a cough.

This is a similar picture to that described by Fendall (1952), Carswell
(1953) and Hanson-Bahr and Weiss (1956), all of whom were discussing cases in the Kitui district. No vomiting was complained of as reported by Cole (1941) and Cole et al. (1942) but his cases were acute in onset. The picture described now is more similar to that described by Tobias (1942) but the symptoms were more definite possibly owing to the longer duration between onset and admission to hospital.

Splenomegaly. The large number of patients complaining of enlargement of the spleen as a symptom is due to, as noted above, the familiarity of the peoples of Baringo District with the enlargement of this organ caused by malaria. It may well be also that it is a result of the propaganda spread by the writer through the medium of government officials, chiefs, headmen and personally at tribal gatherings.

Haemorrhage. Epistaxis and bleeding from the gums were complained of frequently. More than half of these patients stated that the bleeding from the nose and gums was an early symptom, occurring soon after the onset of the disease. It must have been, therefore, of fairly severe degree to have impressed itself on the memories of the patients. One case was admitted with a history of severe bleeding from the gums, this being the reason for seeking admission to hospital.

Weakness and breathlessness on exertion. These were only complained of in 22 and 18 cases. This was rather surprising in view of the loss of weight and anaemia which were marked signs on admission. Three of those complaining of these symptoms were also suffering from schistosomiasis with severe degrees of anaemia. Fendall (1952) also noted this point and this may be to the fact that nearly all the patients stated that they felt reasonably well.
CASE NO. 42.

Male aged 20 yrs of the Tugen tribe.
Appetite. 43 of the patients gave a history of their appetite having increased appreciably since the onset of the disease while only 16 stated that they had lost their appetites. Nearly all patients ate very well in hospital, managing to cope with the standard hospital diets and the supplements of extra meat, milk and vegetables that they were given. This interesting feature of kala-azar is noted in Manson's Tropical Diseases (14th Edition 1954) and was commented on by Fendall (1952).

Cough. This was complained of by 56 of the 71 patients and in 46 cases was accompanied by the production of sputum. Almost invariably these patients showed signs of bronchitis on examination. There were signs of compression of the base of the left lung or both lungs, probably due to splenomegaly and hepatomegaly and seven cases were diagnosed as suffering from broncho-pneumonia. The impression received was that these signs of respiratory involvement were more severe in the cases with longer histories of illness. A complaint of pain in the chest was also common in those suffering from coughs.

Fever. This was a common complaint but of the 56 patients who gave this as a symptom, only 22 also complained of rigors. It was the experience of the writer that the Tugen will often insist that they are suffering from fever when their oral temperatures are normal, and little value is to be placed on this as a symptom unless also supported by a history of sweating and shivering.

Oedema. Swelling of the ankles was complained of by eleven patients. In three cases this was severe and had spread up the legs but two of these
had a severe degree of albuminuria and on further questioning it was
established that the oedema was of much longer duration than the kala-
azar symptoms.

Jaundice. Two of the five patients complaining of jaundice were admitted
in extremis and the jaundice was of marked degree. Both of these
patients died within 48 hours of admission and it is though that a long
and extremely rough journey in a Landrover over rocky roads may have been
too severe a strain in their weak condition.

Physical signs.

The physical appearance of the majority of patients was such that
once the examiner had experience of several case of kala-azar it was
possible to make a provisional diagnosis almost immediately.

Characteristically the patient was a child or young adult of either
sex with marked wasting, especially of the limbs and the chest cage with
a protuberant abdomen, the picture described as the "pregnant skeleton".
The elbow and knee joints were prominent and the hair had lost its
normal lustre, being in advanced cases, fine, straight and grey in colour.
The skin was dull, having lost the normal gloss seen in the healthy
African, but was often pigmented on the anterior aspect of the lower leg
and dorsum of the forearms. There were trophic changes of the skin - the
"crazy-pavementing" also described in kwashiorkor - over the tibiae often.
A particularly striking feature in children was the look of apathy on the
face. This was very characteristic and it became possible to pick a kala-
azur case from amongst a crowd who had collected for treatment by the mobile
health unit or for the kala-azar surveys. In many parts of the district a European doctor had never been seen before and in some cases the children had never seen a European of any profession. The normal African child and even the sick child would usually show interest in the doctor and his equipment, and often would show fear. The kala-azar patient would be led forward by a relative or friend and would stand apathetically without evincing any reactions whatsoever. This apathy of the child suffering from kala-azar has also been described by Carswell (1953).

Examination of the patient revealed splenomagaly of varying degree, and hepatomegaly. Anaemia was evident in the pallor of the mucous membranes. There was pyrexia and increase in the pulse rate. Less often there would be seen adenitis, particularly of the axillary and epitrochlear glands and in younger children with a marked degree of splenomagaly there was a splaying of the lower ribs. The tongue was normally clean. There were signs of bronchitis, broncho-pneumonia or congestion of the lung bases in most cases. Oedema of the feet and ankles was seen less frequently as was injection of the conjunctvae.

This is a similar picture to the kala-azar patients described from Kitui district by Fendall (1952), Carswell (1953) and Manson-Bahr and Heisch (1956).

The following table shows the frequency with which the various physical signs occurred.
Table 15. Physical signs on admission.

<table>
<thead>
<tr>
<th>Physical Signs</th>
<th>Nos.</th>
<th>Physical Signs</th>
<th>Nos</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emaciation</td>
<td>55</td>
<td>Haemic murmurs</td>
<td>3</td>
</tr>
<tr>
<td>Spleenomegaly</td>
<td>68</td>
<td>Splaying of lower ribs</td>
<td>12</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>31</td>
<td>Oedema</td>
<td>17</td>
</tr>
<tr>
<td>Adenitis</td>
<td>50</td>
<td>Ulcer (noma)</td>
<td>8</td>
</tr>
<tr>
<td>Anaemia</td>
<td>55</td>
<td>Injection of Conjunctivae</td>
<td>15</td>
</tr>
<tr>
<td>Enlarged Abdomen</td>
<td>52</td>
<td>Jaundice</td>
<td>5</td>
</tr>
<tr>
<td>Pyrexia</td>
<td>52</td>
<td>Cheilosis</td>
<td>6</td>
</tr>
<tr>
<td>Hair changes</td>
<td>35</td>
<td>Concomitant disease</td>
<td>60</td>
</tr>
<tr>
<td>Pigmentation</td>
<td>16</td>
<td>Haemorrhage</td>
<td></td>
</tr>
</tbody>
</table>

General Condition.

In general the patients stated that they felt well and fit even when they were seen to be in poor general condition with severe emaciation, marked splenomegaly, anaemia of severe degree and with signs of bronchitis and respiratory infection. The children were often apathetic but even they would not stay in bed and instead would be found sitting out in the open or wandering around the hospital. For a patient to stay in bed usually indicated that there was either a concomitant or intercurrent disease or, in the absence of any signs of this, any desire to remain in bed was a sign of a bad prognosis. Practically all the patients were ambulant throughout their stay in hospital.

There was no evident correlation between the duration of symptoms, and the clinical condition. Of the 39 cases with a history of less than
six months the condition was graded as being "fair" in 19 patients, 4 as "good" and the remainder "poor". Where the duration of disease was more than 6 months or more, 12 of the 28 patients were in "fair" condition 6 as "good" and 10 as "poor". There was, however, a tendency for the younger children to be in fair or poor condition and for the adults to be in better condition. 5 patients were admitted in moribund condition and all died within five days of admission. They had been ill for 2,12, 17,19 and 24 months respectively, the patient with a two months history being a child of three.

Emaciation.

This was seen in 55 of the 68 patients for whom records are available. As described above, the wasting of the limbs and intercostal spaces was in marked contrast to the enlarged and protuberant abdomen. Splaying of the lower ribs was seen in 12 cases, all of whom were young children and was probably due to pressure on the ribs by the enlarged spleen and liver. The wasting was of severe degree in many cases and examples are given below.

Male child aged 3 yrs. 24 lbs.
Female child aged 6 yrs. 28 lbs.
Male child aged 9 yrs. 30 lbs.
Male child aged 12 yrs. 42 lbs.
Adult female aged 20 yrs. 5 st. 10 lbs.
Adult male aged 30 yrs. 7 st. 8 lbs.

Splenomegaly & Hepatomegaly.

All patients in this series were found to have enlarged spleens of
varying degree. This is in accordance with the findings of Kendall (1952) and Manson-Bahr and Heisch (1956). Cole (1944) found, however, that splenomegaly was not constant and tended to be a late sign.

The enlarged spleen was invariably firm and hard with a smooth surface. In about a third of the patients there was tenderness on pressure of the spleen. The spleen was usually mobile and did not appear to be adherent to the peritoneum, the abdominal wall being movable over the surface.

Enlargement of the spleen varied from an enlargement of 3-4 finger-breadths below the costal margin to a spleen occupying the major part of the abdominal cavity. Classification of the size of the spleen by Schuffner's method is shown below:

<table>
<thead>
<tr>
<th>Splenomegaly</th>
<th>Schuffner Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Class II</td>
</tr>
<tr>
<td>10</td>
<td>25</td>
</tr>
</tbody>
</table>

Thus ten patients had a spleen extending less than halfway between the costal margin and the umbilicus, twenty-five had spleens extending more than halfway to the umbilicus and thirty-three had spleens extending beyond the umbilicus.

The largest spleens, (3 in Class IV) were all seen in children under the age of 14 years. Case No. 1, aged 5 years, had a spleen extending 10 finger-breadths below the costal margin, Case No. 47, aged 8 years, a spleen enlarged 11 finger-breadths and Case No. 61, aged 13 years, a spleen enlarged 10 finger-breadths. There was a tendency for splenic
enlargement to be less marked in adults. Many of these gave a history of repeated attacks of malaria during their lives and a spleen already enlarged before the onset of the kala-azar. It is thought, therefore, that a degree of fibrosis of the spleen was present which prevented marked increase in size due to kala-azar.

There did not appear to be any correlation between the duration of the illness and the degree of splenic enlargement. One patient with a history of illness of only one month had a spleen enlarged to four finger-breadths below the costal margin whilst in eight cases who had been ill for two months the enlargement of the spleen varied between three finger-breadths and eight finger-breadths below the costal margin, with a mean of 5.6.

In seven patients who had been ill for two years, the degree of enlargement of the spleen ranged between four finger-breadths and nine finger-breadths with a mean of 6.

Thus there was a marked degree of enlargement of the spleen very shortly after the onset of the disease, a finding at variance with the suggestion of Cole (1944) that splenic enlargement occurred as a late sign.

The spleen regressed in size during treatment in all cases successfully treated and who received a full course of treatment. From these findings are excluded the patients who died (6) and those still under treatment at the time of writing (2). The reduction in size of the spleen was used as an index to the progress of the patient and in about 50% of the cases this regression commenced within three days of the
initiation of treatment. On discharge of the patients the spleen was impalpable in 24 patients, palpable only on deep inspiration in 11 patients, enlarged one finger-breadth in 16, and enlarged two finger-breadths below the costal margin in nine.

Hepatomegaly was not such a constant feature and was seen in 31 cases (45.6%). In addition this hepatomegaly was not of such marked degree as the splenomegaly as shown below.

**Distance below costal margin.**

<table>
<thead>
<tr>
<th>Distance below costal margin</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 finger-breadth</td>
<td>15</td>
</tr>
<tr>
<td>2 finger-breadth</td>
<td>8</td>
</tr>
<tr>
<td>3 finger-breadth</td>
<td>5</td>
</tr>
<tr>
<td>4 finger-breadth</td>
<td>3</td>
</tr>
</tbody>
</table>

There was a degree of correlation between the splenomegaly and the hepatomegaly, the most enlarged livers being found in patients with gross enlargement of the spleen.

With this splenomegaly and hepatomegaly there was enlargement of the abdomen. This was obvious in 52 cases and was probably due in part to loss of the tone of the musculature of the abdominal wall through weakness as well as the enlargement of the spleen and liver. The enlargement of the abdomen was more obvious in young children than in adults and in 12 of these children there was an outward splaying of the lower ribs of varying degree which would also appear to be due to the splenomegaly and hepatomegaly.
Lymphatic Glands.

Enlargement of lymphatic glands was an especially common feature and was seen in 50 of the 68 patients for whom records are available. The enlargement of the glands was not always symmetrical, one or other side being involved to a differing degree. The groups of glands involved were as follows:

- Sub-mental: 3
- Cervical: 17
- Axillary: 50
- Epitrochlear: 48
- Inguinal: 50

The enlargement was never of a marked degree and the glands were discrete, freely movable and shotty in texture.

Manson-Bahr and Heisch (1956) report that the axillary glands are rarely affected by chronic inflammation in the African and that enlargement of this group is significant.

The enlargement of the epitrochlear glands was also remarkably constant and the palpation of these was a standard feature when examining large numbers of possible kala-azar cases on field surveys. This enlargement was often more marked than that at other sites and was slow in responding to treatment.

Anaemia.

This was also a sign often seen. In 55 cases a clinical diagnosis of anaemia was made after examination of the mucous membranes. Every
degree from mild to severe was seen. Blood counts were performed on 56 cases on admission and the average haemoglobin level was 49.6% (Sahli) equivalent to approximately 7.4 grammes per cent. The range in haemoglobin levels was from the lowest figure of 35% (6.9 g. per cent) to the highest figure of 70% (12.9 g. per cent). The average haemoglobin level on discharge was 82% Sahli (approximately 13.89 g. per cent).

As noted above, in spite of the degree of anaemia found on examination, surprisingly few patients complained of breathlessness or other symptoms attributable to anaemia. In three cases a haemic systolic murmur at the apex was heard. The anaemia was an early feature in the disease and the more severe degrees of anaemia were seen in the more advanced cases. There did not seem to be any correlation between the severity of the anaemia and the duration of the disease.

Pyrexia.

All the patients exhibited pyrexia during the course of their stay in hospital, 52 of them being admitted with a raised temperature.

All types of temperature were seen, intermittent, remittent, undulant, the step-like rises characteristic of early typhoid, evening rises as seen in tuberculosis, in fact, the main impression was that there was no single type of temperature chart which could be described as being characteristic of the disease. The double peaks in twenty-four hours which Manson-Bahr (Manson's Tropical Diseases) reports as occurring in 68% of cases, were seen, but were not such a constant feature as might be supposed from textbooks. In some cases there were periods of apyrexia lasting seven to ten days. All types might be seen in one patient.
An interesting feature is that 45 patients exhibited a pyrexial reaction on the institution of treatment similar to the Herxheimer reaction in syphilis. This was also reported by Kirk and Henry (1954) who considered that this exacerbation of fever was found when the disease process was active.

Hair Changes.

In the healthy African the hair is normally a glossy black, tightly curled and strong, and coarse and profuse in growth. In kala-azar the hair loses its lustre becoming dull and its tensile strength lessens so that it becomes straighter. As the disease advances the hair becomes finer and the colour changes to a dull gray, later becoming almost colourless. The hair becomes silky and straight resembling that of a baby. The picture is very similar to that seen in kwashiorkor except that in the latter condition the hair normally has a reddish tinge.

These hair changes were seen in 35 patients and they were more common in children than in adults. There was a correlation between the hair changes and the length of the disease and it seemed that the picture described above was only seen in cases who had been ill for four months or longer.

Similar changes are seen in the hair of the eyebrows, which becomes much finer and may disappear as the hair falls out.

Pigmentation.

The pigmentation which gives rise to the name kala-azar, the "black disease", is not so marked or so obvious in Africans as in Asians.
or Europeans, and only 16 patients in the series exhibited this sign.

It normally appeared as a smudgy discolouration of the skin, which was dry and coarse, on the extensor aspects of the arms and legs. The skin was gray in colour and, at first, this was thought to be due to dirt. More commonly seen were trophic changes of the skin over the anterior aspects of the lower legs. The skin was thin and shiny and broken up by irregular creases, the picture resembling a crazy pavement. This sign was seen in 42 African patients suffering from other conditions.

In six patients, small solitary circumscribed patches of pigmentation, in no case larger than one inch in diameter, were noted. In two cases they were seen on the back, in one case on the cheek and in three cases on the legs. Similar lesions were described by Manson-Bahr and Heisch (1956) who found Leishman-Donavan bodies in some. They considered that these lesions may be healed primary chancre following the bites of infected sandflies.

Cedema.

This was seen in 17 patients. As mentioned above in three cases this extended to the knees, and in the remainder involved only the feet and ankles. In two of the cases with the more marked signs the oedema had been present before the onset of the kala-azar and in both of these a moderate degree of albuminuria and granular casts were found on investigation of the urine. Albuminuria was seen in six cases in all and was mild to moderate in degree.
All patients with oedema were also suffering from anaemia as shown by low haemoglobin levels, in these cases ranging from 30% to 45% (Sahli), and their general condition was poor. On the other hand, several patients with haemoglobin levels as low as this did not exhibit oedema of the ankles.

**Ulcers.**

Ulcers of the cheeks and gums were seen in eight patients. These varied in severity and were seen in cases whose general condition was poor. In two cases the ulcers had penetrated the musculature of the cheek. The ulcers were normally situated opposite the molar teeth and were foul and sloughing with eroded edges. In two cases the patients died but in the remainder the ulcers improved with the patients' general condition after the institution of specific kala-azar therapy. Antibiotics were used in these cases and their use usually resulted in the ulcer becoming much cleaner and the disappearance of the sloughs, but no marked improvement was seen until the general condition improved. Specimens taken from the edges of the ulcers were examined for leishmania in all cases, but none were found. Kahn reactions were also negative.

A leucopenia was found in all eight patients, the total white cell count ranging between 2,500 w.b.c. per cubic millimetre to 4,000 w.b.c. per cubic millimetre.

It is considered that these ulcers could be classified as noma in advanced kala-azar.
Respiratory Infection.

A remarkable feature of this series was the large number of patients suffering from respiratory infections. 12 of the 68 patients (i.e. 61.8%) exhibited signs of this, varying from mild bronchitis to definite broncho-pneumonia. Seven cases were found to be suffering from broncho-pneumonia, 17 from severe bronchitis and 12 were diagnosed as suffering from chronic bronchitis with cough and muco-purelent sputum. The remainder had signs of congestion of the lungs at one or both bases.

This high percentage of respiratory infections is attributed to two main causes. These infections, as will be seen from reference to Table 9 which gives the diagnoses of some 17,000 patients seen by the mobile health unit teams, are a major cause of morbidity among the inhabitants of the low country of Baringo and of the Kerio Valley. Chronic bronchitis, therefore, is a common finding. This may well be due to the poor standard of housing and to the cold winds which often blow for short periods at sunset even during the hot season.

Thus it might be expected that a considerable number of kala-azar patients would suffer from respiratory infections but, in addition, there might well be a predisposing factor in the disease in that the increased intra abdominal pressure from splenomegaly and hepatomegaly would raise the diaphragm causing congestion of the bases with consequent impairment in respiratory ventilation.
CONCOMITANT DISEASE.
A number of patients were suffering from a second disease in addition to kala-azar. These were, in order of frequency:

- Malaria 16
- Trachoma 10
- Schistosomiasis 6
- Measles 3
- Ancylostomiasis 3
- Pyogenic infection 3
- Taeniasis 2

This total of 43 patients out of 68 indicates the need for care in examination and verifies the commonly held theory that, in the African, it is usual to find more than one pathological condition present at the same time.

Since malaria is endemic in the area from which the kala-azar cases come it was not unexpected that this would be found in a number of patients. The diagnosis was established by the routine blood examinations carried out on all patients after admission to hospital.

The discovery of schistosomiasis was interesting in that, with the exception of one case in a European which might have been contracted in the district, no bilharzia had been reported previously in Baringo District. All six cases came from the Kerio Valley and it was later confirmed that the infection had been contracted by bathing in a small stream running through Kaptiony which was found to contain cercaria.
Five patients complained of haematuria and since routine urine tests were done on admission the sixth case was brought to light. It is of interest to record that the schistosomiasis responded to the treatment given for the kala-azar and that no further treatment was necessary.

As remarked earlier, patients suffering from a concomitant disease or complication were more likely to stay in bed in hospital and tended to be more ill.
COMPLICATIONS.
Respiratory infections.

As described above, the greatest complicating feature was the high number of respiratory infections. This was seen so commonly in the series that it might be considered a normal feature of the disease in this area rather than as a complication. The seven patients with broncho-pneumonia were slow to respond to antibiotic therapy and gave rise to much anxiety, being seriously ill. Those twelve patients suffering from chronic bronchitis gave histories of respiratory trouble over a number of years, antedating the onset of the kala-azar, and although some improvement was seen in most cases, the signs of chronic bronchitis were still evident on discharge from hospital.

Dysentery.

Two patients developed diarrhoea with blood and mucus in the stools. Stool examinations were made on several occasions but no Leishman-Donavan bodies were found. Manson-Bahr (Manson’s Tropical Diseases) considers that dysenteric symptoms may be produced by intestinal lesions caused by leishmania or to superadded infection with amoebic or bacillary dysentery. No entamaeba histolytica were seen in the stools and laboratory facilities for stool culture were not available.

The dysenteric symptoms responded in both cases to routine therapy with succinyl sulphathiazole.

Jaundice.

Five patients were suffering from clinical jaundice as shown by icteric discoloration of the sclera and of the palms of the hands.
Two of these were severely jaundiced and, as noted above, died very shortly after admission. In the remaining three patients the jaundice was less severe. However, all jaundiced patients were in poor condition with severe weakness and emaciation and low haemoglobin levels. Splenomegaly and hepatomegaly were marked.

The jaundice was thought to be toxic in origin since, in the three surviving patients, while bile pigments were found in the urine, the faeces were normal in colour and no bile pigments found.

It was not possible to investigate this jaundice in detail since laboratory facilities were not available at Kabarnet hospital, but the hypothesis that it was toxic in origin is supported by accepted theory that the leishmania is essentially an invader of the reticuloendothelial system. The marked hepatomegaly in these five cases would indicate involvement of the liver. That cases of jaundice in areas where kala-azar is endemic should be investigated as suspect cases is suggested by MacKay-Dick (1945).

Haemorrhage.

Severe epistaxis occurred in three cases one of whom died, and one patient had a haematemesis shortly before death. In addition, as reported above two patients with dysenteric symptoms passed blood and mucus in the stools. Manson-Bahr and Heisch (1956) reported epistaxis in 17 of their 40 cases but no haemorrhage from other sites, and Cole (1944) found an increased tendency to haemorrhage in his cases which were acute in onset.
In marked distinction to the small number of patients who suffered from haemorrhage while in hospital were the large number of patients who gave a history of haemorrhage since the onset of the disease, 33 patients complained of epistaxis and 21 of bleeding from the gums. 14 patients suffering from both. This has already been commented on above.
LABORATORY INVESTIGATIONS.
Laboratory facilities at Kabarnet were limited and it was not possible to do serological investigations or bacterial culture. The postal services were subject to delay and mail normally was carried by an orderly on foot between Kabarnet and Tambach through the heat of the Kerio Valley. It was normal to receive reports from the provincial laboratory at Nakuru that specimens had been damaged in transit and that the investigation requested had not been possible.

Routine laboratory investigations on all suspect kala-azar patients were as follows:

a. Serum-aldehyde test (Napier and Bramachari).
b. Spleen puncture.
c. Blood films, thick and thin.
d. Urine.
e. Haematology.
Serum Aldehyde Test.

This test, also known as the "formol-gel" test, was carried out by Brahmachari and Napier's method as described in Manson's Tropical Diseases (Manson-Bahr 1954) and was positive in seventy cases in all. Sixty eight of these patients were treated in hospital and a further two (Cases No. 25 and 26) were diagnosed on a field survey but failed to report to hospital.

Of these cases, 51 gave a strong positive reaction within twenty minutes, the serum becoming jellified and opalescent in that period. 15 further cases gave positive reactions in under two hours and the remaining four within 6 hours.

Examination of the serum before discharge from hospital was done in 50 patients and of these 32 were still positive. This was probably due to the short lapse of time between the end of treatment and the discharge from hospital. Nine of the patients in whom the test was positive on discharge were seen three months later and all but one were then negative for the aldehyde test.

A positive result in this test, although useful when taken in conjunction with other signs and symptoms, did not always indicate that the individual was suffering from kala-azar. Positive results were obtained in fourteen cases of chronic malaria, in 3 cases of pulmonary tuberculosis, and in one case each of primary carcinoma of the liver, cirrhosis of the liver and bilharzia.

Spleen Puncture Examinations.

These were made regularly and routinely in all suspect cases of
kala-azar in hospital and also in the field (see above). Although described in most text-books of medicine as being a procedure not to be adopted lightly, the writer was able to train senior African staff to carry this out and no adverse sequelae or fatalities were reported either in hospital or in the field.

The procedure in hospital was as follows:—

In apprehensive patients, premedication, usually with barbiturates, was given one hour beforehand. The skin of the abdomen was thoroughly cleaned with cetrimide solution, allowed to dry and then painted with iodine, gentian violet or brilliant green. The puncture site was then infiltrated with local anaesthetic as far as the peritoneum. The patient was then told to hold his breath in full inspiration and the spleen fixed manually by an assistant. A sterile, dry hypodermic needle of the type used for intramuscular injections of oily penicillin, fitted to a 10 or 20 cc hypodermic syringe is inserted into the spleen. The piston of the syringe is withdrawn several times and there should be a marked degree of resistance. The aim is not to withdraw blood but splenic material and after the needle is withdrawn the piston of the syringe should be depressed slowly to extrude any blood, the contents of the needle then being blown on to a glass slide where the translucent masses of splenic pulp may be seen. Films are then spread and stained by Leishman's method. The puncture wound was sealed with collodion and the patient kept lying absolutely flat for 3-4 hours.

If, for any reason, it was necessary to repeat the spleen puncture, this
was not done for at least four days and a different puncture site selected.

As experience was gained it was found that in many cases the infiltration of the puncture site with local anaesthetic could be omitted in the less apprehensive patient.

Spleen punctures were positive for Leishman-Donavan bodies in all seventy patients on admission (this includes the two patients who failed to report for treatment in Hospital). In one case the spleen puncture was performed immediately after death.

It was difficult to get patients to accept spleen puncture on discharge and only twenty cases were so examined. No Leishman-Donavan bodies were found in any of these. Sternal marrow puncture was done on discharge in the later cases in the series, since in many cases the spleen was impalpable, and a further sixteen cases accepted this procedure. Again no Leishman-Donavan bodies were found.

**Blood film examinations.**

Routine blood films, thick and thin, were taken from 68 patients. Thick films were stained by Field's method and examined for malaria parasites. These were found in 16 cases, all the parasites being *Plasmodium falciparum.*

Thin films were examined for Leishman-Donavan bodies using Shortt's technique (Shortt et al 1927) but no parasites were seen even though repeated examinations were made of slides from eight patients with heavily infected spleens.
Manson-Bahr and Heisch (1956) failed to find parasites in the blood of 40 patients from the Kitui area in Kenya whereas Archibald and Mansour (1937) found parasites commonly in the blood of patients in the Kapoeta District but only on a few occasions in patients in the Fung area. However, in this area Henderson (1937) found parasites in 1% of his patients. The position is different with reference to Indian kala-azar. Knowles and Das Gupta (1921) saw parasites in the blood of 67% of their patients and Shortt et al (1927) in 98% of their cases.

Urine Examinations.

Routine examination of the urine revealed bile pigments in five cases who were jaundiced, but this was not marked. Albumen was found in small quantities (++) in three cases and in moderate quantity in a further three (+++). Two of these also had granular casts in the urine and it was thought that renal damage had preceded the onset of the kala-azar. This was confirmed by the patients' histories of cedema of the ankles and lower leg of several years duration.

The ova of Schistosoma haematobium were found in the urine of six patients. This has been commented on above. Cole (1944) reported albuminuria in 77.5% of his cases but these were acute cases. Fendall (1952) did not find that renal damage was a feature of the disease and the present findings are in conformity with this.
Haematological Examinations.

Table 16. Haematological Findings.

<table>
<thead>
<tr>
<th></th>
<th>On Admission 57 cases</th>
<th>On Discharge 40 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td><strong>Haemoglobin % (Sahli)</strong></td>
<td>49.6%</td>
<td>35% - 70%</td>
</tr>
<tr>
<td><strong>Red Blood cells</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(millions/cu mm)</td>
<td>3.19</td>
<td>1.77 - 5.23</td>
</tr>
<tr>
<td><strong>White Blood cells</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(/cu mm)</td>
<td>10,900</td>
<td>2,500 - 16,000</td>
</tr>
<tr>
<td><strong>Differential Count</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>W.B.C.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neutrophil polymorphs</td>
<td>65.6%</td>
<td>45% - 87%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>33.1%</td>
<td>11% - 55%</td>
</tr>
<tr>
<td>Basinophils</td>
<td>1.8%</td>
<td>0% - 7%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.5%</td>
<td>0% - 3%</td>
</tr>
</tbody>
</table>

Anaemia was recorded in every case in the series, the highest haemoglobin level recorded being 70% Sahli. and the lowest 35%. The anaemia was hypochromic and poikilocytic, thus there was an element of iron deficiency in its aetiology and it was not purely a secondary effect of invasion of the bone marrow by leishmania.

The remarkable feature of this series is the average white cell count of 10,900 per cubic millimetre. Manson-Bahr states (Manson's Tropical Disease 1954) that the total white cell count is below 3,000 per c.mm. in 95% of cases. Cole (1914) found an average count of 3,100 per c.mm in his
cases from the Kings African Rifles; Fendall (1952) gave an average count of 3,717 W.B.C. per cu.mm in his cases from Kitui. Carswell (1953) reporting in the early stages of the epidemic of kala-azar in Kitui district found that the white cell count was consistently low, being between 1,500 and 4,000 per c.mm. However, Manson-Bahr and Heisch (1956) found that, in 40 cases from Kitui, leucopenia was not invariably present and when present it took about three months to develop.

When the writer, on reviewing the records of the first fifteen cases in this series, noted that a leucocytosis, rather than a leucopenia, was being found in these cases, it was decided to check the work of the laboratory technician. The haematology of the next ten cases was done independently by the writer and the technician and spot checks made from time to time after this. In all cases the results were within the normal limits for independent examinations. It was, therefore, accepted that the results obtained was reasonably accurate.

Two suggestions are, therefore, put forward to account for this. As reported above, Manson-Bahr and Heisch (1956) found that the leucopenia takes about three months to develop. Twenty cases of the seventy one in this series were admitted to hospital with a history of illness of less than three months. The lowest white cell count seen in any of these patients was 5,500 per cu.mm.

Secondly, there was a high incidence of respiratory infection in the series. This would result in a leucocytosis as would also pyogenic infection. It has been suggested that the liability to such concomitant
infections in kala-azar is due to the leucopenia. However, chronic respiratory infections are, as reported above, common among the inhabitants of the low country of the Baringo area and it may well be that this chronic respiratory disease had lowered the resistance to infection by leishmania, and such patients were predisposed to the disease.

This failure to find the leucopenia, which is normally accepted as a diagnostic criterion of kala-azar, has also been reported by Dr. Murphy, working in the West Suk District which lies north-east of the Kerio Valley (see Map 2) in a personal communication. Kala-azar was first discovered in this district in mid-1956, and he has also found that there is a leucocytosis rather than a leucopenia and that the incidence of respiratory infections is high.

In view of this it is to be expected that the granulopenia and relative lymphocytosis normally found in kala-azar would not be present and this is confirmed by the figures given in the table above.

It is of interest to note, however, that in those patients with leucopenia, the relative increase in lymphocytes was found. Thus in one case with a total white count of 2,500 the lymphocytes were 50% of the total; in another with a total white count of 3,700 per c.mm. polymorhs were 11% and lymphocytes were 74%; and in a third case lymphocytes were 62% of a total white count of 2,900 per c.mm.

The haematological findings in 40 patients on discharge are also given above. The picture shows a general all-round improvement with increase in the haemoglobin values, increase in red cell counts and a
decrease in white cell counts as a result of treatment of the concomitant infections.

The average duration of time elapsing between the completion of treatment and discharge from hospital was three weeks.

Other Laboratory investigations.

Stool examinations were made in 34 cases. In two cases taenia saginata were found and blood and mucus in two patients with dysenteric symptoms. No leishmania were found in the stools although Manson-Bahr (Manson's Tropical Diseases) quotes the demonstration of these by Shortt and his colleagues.

Nasal smears were also examined in sixteen patients for leishmania but none were found.

The sputum was examined in twenty cases for acid-fast bacilli but all were negative and the Kahn test ten times, this also being negative.

The blood pressure was recorded in 30 cases. The average systolic pressure was 104 mm of mercury and the average diastolic pressure 66 mm. Manson-Bahr and Heisch (1956) also report this low blood pressure in their cases.
DIFFERENTIAL DIAGNOSIS.
Since a special effort was made to discover and treat as many cases of kala-azar in the district as possible, the staff of the hospital, dispensaries and mobile health units were given full descriptions of the disease, as were Government officers, who spent much time on tour in the area. In most cases, patients suffering from kala-azar were demonstrated to them.

The commonest disease causing confusion was undoubtedly chronic malaria, with concomitant respiratory infection. In these cases the lack of hair changes and the absence of enlarged axillary and epitrochlear glands were indications that the condition was not kala-azar and a negative formal-gel test was confirmatory evidence.

Two cases were though to be chronic malaria and respiratory tuberculosis when in fact they were suffering from kala-azar.

Chronic malaria caused some confusion but detailed questioning usually produced the answer that the spleen regressed in size between the pyrexial attacks. This did not occur in kala-azar, the spleen increasing in size steadily in almost all cases.

In the first patient with noma, tertiary syphilis producing gumma, and splenomegaly and hepatomegaly was considered. The formal-gel test was done in this case coincidentally and when this was discovered to be positive, a spleen puncture done. In the remaining cases with noma the probable diagnosis was immediately kala-azar although Kahn tests were done to exclude concomitant syphilis.
Cirrhosis of the liver and obstruction of the portal circulation was diagnosed in one case very early in the series before familiarity with kala-azar was gained. It was thought that the pyrexia observed was due to malaria but failure to respond to malaria therapy aroused suspicions as to the diagnosis.

Brucellosis as a differential diagnosis is reported by Fendall (1952) and Manson-Bahr and Heisch (1956) but this was extremely uncommon in Baringo District and was not a cause of confusion as it is in the Kitui District where this disease is relatively common.

Manson-Bahr (1954) states that Egyptian splenomegaly or mansonian schistosomiasis should be excluded but this disease has never been reported among the inhabitants of Baringo.

It must be mentioned here that, in addition to the seventy one cases described in this paper, twenty seven cases, who satisfied all the diagnostic criteria for kala-azar, with the exception of the finding of Leishman-Donovan bodies by spleen or sternal marrow puncture, were given specific kala-azar therapy in hospital. In twenty three of these cases the spleen diminished markedly in seze and there was considerable improvement in the patients' general condition. It is considered extremely probable that these patients were suffering from kala-azar but since Leishman-Donovan bodies were not demonstrated they have, therefore, been excluded from the series.
TREATMENT.
It is remarkable that while the clinical findings in kala-azar vary but little in the different major endemic areas, the response to treatment varies considerably. Adler (1947) suggests that there is a variation in the parasites which can be established by cultural characteristics, pathogenicity in the natural hosts and their behaviour in man and in sandflies. He also found the susceptibility to organic antimony compounds and aromatic diamidines and reports that infections caused by the parasite of Indian kala-azar are more easily eradicated than those caused by the Mediterranean form.

The first drug to be used successfully in the treatment of kala-azar was antimony tartrate. In India the results were very good with 88% recovery in an epidemic in 1921 (Rogers 1939) and 77% in another series reported by Sati (1941) where this was used alone and in conjunction with other antimonial drugs.

On the other hand, results in the Sudan were disappointing. Archibald (1923) had 13 deaths in 17 cases, and Stephenson (1940) a 78% death rate. Kirk (1947) considered that these differences were due to the parasite in the Sudanese form of kala-azar differing from that in the Indian form.

With the introduction of the aromatic diamidines more satisfactory results were obtained in the Sudan. Kirk and Sati (1939) had four deaths in 28 cases treated with Stilbamidine (4 : 4 diamido - stilbene) and reported favourably in a series of 43 some treated with stilbamidine and some with Pentamidine (4 : 4 diamidine diphenoxypentane) (Kirk and Sati 1943).

Again, however, the results in India tended to be better. Napier
et al (1942) reported a 90% cure rate in 101 patients with stilbamidine.

East Africa.

The first recorded series of cases in East Africa in which the treatment was discussed was that of Cole et al (1942) which was followed by a further report by Cole (1944) incorporating the results of the first. He used stilbamidine and later urea stibamine and came to the conclusion that urea stibamine was preferable in view of the unpleasant side effects when using stilbamidine. These toxic reactions were also described by Kirk and Henry (1944). These were a fall in blood pressure, with sweating, dizziness, nausea and vomiting. Kirk (1947) also describes sudden deaths in patients who are otherwise doing well and suggests that this may be due to a toxic effect of antimony on the myocardium. Of the 60 patients in the series published by Cole, 22 patients died. 15 of these deaths occurred among 29 patients in the 2/3rd Bn. Kings African Rifles who were suffering from an acute toxic form of the disease contracted on the Omo River. Heisch (1947) also used stilbamidine during the war, on two patients in the Northern Province of Kenya, one of whom was cured.

Fendall (1952) used stibophen and stilbamidine in two cases each. Those treated with stibophen, a trivalent antimony compound, did not respond but those who had received stilbamidine were discharged as cured. The remainder of his cases were treated with urea-stibamine (Brahmachari) (11 cases), pentamidine isethionate (13 cases) and combined therapy (2 cases). He concluded that the treatment of choice where adequate medical supervision was available was urea-stibamine but that pentamidine isethionate should be
used, in spite of the lower percentage of immediate cures, where adequate supervision was not available.

Carswell (1953) used combined therapy of pentamidine followed by urea-stibamine.

The Kitui District Annual Medical Report for the year 1952 records that urea-stibamine was used by itself but that the relapse rate was high. During the year the standard treatment became pentostam followed by pentamidine, and reference to the Annual Medical Reports for following years shows that this combined therapy was continued.

Manson-Bahr and Heisch (1956) discuss the various drugs used which were pentamidine, pentostam and urea-stibamine. They consider urea-stibamine the most effective but report that a combined course of pentostam and pentamidine cured many patients.

Present series.

A number of factors were taken into account when determining the type of treatment to be adopted.

Firstly, that response to treatment in kala-azar seems to vary according to the geographical area in which the disease occurs, in that the Indian and the Chinese types seem to respond better to treatment than the Mediterranean and Sudan form (Kirk 1947, Adler 1947). It was felt that the Kenya form was more likely to resemble the Sudan form in view of geographical location of the two countries.

Secondly, the only published records to treatment of kala-azar in Kenya at the time (1955) were those of Cole et al (1942) and Cole (1944)
which described an outbreak of acute toxic kala-azar, and Fendall (1952) in his description of 31 cases in Kitui. Carswell (1953) did not discuss treatment in detail. Fendall and Cole both considered urea-stibamine to be the treatment of choice where adequate medical supervision was available. It was known, however, that a combined course of treatment with pentostam and pentamidine had been used on a large scale in the Kitui epidemic since 1952, both in hospitals under medical supervision and in the kala-azar treatment centres which had been established in the northern area of Kitui District where the cases were most numerous. The treatment centres were staffed by African hospital assistants with periodic visits by a medical officer. No adverse reports about the combined therapy had been received.

Thirdly, the local medical arrangements existing in the area had to be taken into account. There was one medical officer for an area of 4,600 square miles and a population of some 138,000 people. Three hospitals, at Kabarnet, Tambach and Marigat Works Camp, and a large dispensary with beds at Eldema Ravine, had to be administered by the medical officer. At Marigat an irrigation scheme was being developed by labour from a Works Camp whose population varied between 1,100 and 2,000 people. Communications were poor and the average speed of travel on the roads in a four-wheel drive vehicle was ten to twelve miles in the hour. There were no other European staff, the medical staff consisting of unqualified African personnel.

This work, inevitably, entailed much travelling on the part of the medical officer, and long absences from Kabarnet hospital, in some cases, extending up to fourteen days, but, on average, the number of days away at
any one time was three or four. In a month the medical officer was absent from Kabarnet for an average of sixteen days.

Having taken all the above considerations into account it was decided that the treatment, whilst reasonably effective, should be simple and sufficiently non-toxic to allow of routine administration by the African staff, after full instruction in the method.

Urea-stibamine did not satisfy these criteria and it was decided to employ combined therapy with pentostam and pentamidine.

The routine course for an adult consisted of ten daily, intramuscular injections of pentamidine isethionate (4 ; 4 diamidine diphenoxypentane) totalling 2.0 grammes. Three days after the conclusion of this course, ten daily intravenous injections of pentostam (nitrogen glucoside of sodium p-amino phenyl stibinate) of 6 ccs (0.6 gm) each were given. These dosages were adjusted suitably for children. All intravenous injections were given with the patient lying flat and he was not allowed to get up for at least one hour.

No toxic effects were seen in any of the patients so treated, with the exception of a pyrexial reaction, with a sharp rise in temperature on the day on which treatment was commenced or on the following day. The temperature rose to a level of 102-104°F for a period of several hours before falling to the pre-existing level. There was also slight malaise and exacerbation of the existing symptoms but vomiting was seen in only two cases. This was not severe. This exacerbation of fever was also reported by Kirk and Henry (1944) and Fendall (1952) reports pyrexia in 6 of 11
patients treated with urea-stibamine but does not state at what stage of treatment the pyrexia occurred.

Results of treatment.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Deaths</td>
<td>6</td>
</tr>
<tr>
<td>Discharged from hospital</td>
<td>60</td>
</tr>
<tr>
<td>on completion of treatment</td>
<td></td>
</tr>
<tr>
<td>Treatment not yet completed</td>
<td>2</td>
</tr>
</tbody>
</table>

Deaths.

Six deaths occurred in hospital amongst the 71 cases. In addition, two cases diagnosed on a field survey failed to report to hospital and their fate is unknown. A rather dubious report was received about Case No. 42, who was reputed to have died suddenly at home six months after leaving hospital. Attempts to trace this case were unsuccessful since the family had moved from their home location with their stock in search of water and grass. From the description given to the writer it is possible that death - if it did occur - had been due to the delayed toxic effects of antimony as described by Kirk and Henry (1944).

Of the six deaths occurring in hospital, two were classified as hepatic failure complicating kala-azar, one as epistaxis complicating kala-azar, one as haematemesis complicating kala-azar and the remaining two were attributed directly and solely to kala-azar.

Case No. 29 was a male aged 20 years who had been ill for one year. He was an advanced case with marked jaundice and anaemia who had travelled about seven miles on foot to report to the mobile health unit and had then been transported 45 miles the following day over extremely rough roads
to hospital. He died two days after admission to hospital.

Case No. 50 was a girl aged ten who had been ill for two years. She had travelled ten miles on foot to the nearest road in order to get transport to hospital and was picked up by the writer on his way back from Eldama Ravine to Kabarnet. This child was extremely ill with marked emaciation, splenomegaly and hepatomegaly. There was gross clinical anaemia and jaundice and although much care was taken in driving the remaining forty miles to hospital her condition deteriorated during the journey and she died three hours after admission. In this case the spleen puncture, which had been delayed in view of her condition, was done immediately after death.

Case No. 1 a male child aged 5 years had been ill for eight months. He again was an advanced case with splenic enlargement to 10 finger-breadths below the costal margin and a haemoglobin of 50%. A full course of pentamidine had been given when the child had a single severe haematemesis and died.

Cases No. 16 a female aged 10 with a history of illness for eighteen months, was a severe case with marked splenomegaly and hepatomegaly and a low blood pressure of 90/60 mm of mercury. She had profuse bleeding from the nose which continued for two days in spite of treatment and death occurred on the third day.

Case No. 17 was a boy aged 8 who weighed 39 lbs on admission and had been ill for twenty months. The smear from the spleen puncture contained very large numbers of Leishman-Donovan bodies and his condition was advanced.
He died before treatment commenced.

Case No. 50 a girl aged 3 had been ill for only two months. The spleen was enlarged four finger-breathths, there was a moderate degree of emaciation and her condition was classified as moderately advanced on admission. There was no response to treatment in this case and her condition deteriorated steadily until death.

Discharges.

Sixty patients completed the full course of treatment before discharge from hospital. In all cases there was reduction in the size of the spleen, improvement in the blood picture, increase in weight, absence of fever and where spleen puncture or sternal marrow puncture was permitted, disappearance of the parasite. There was also considerable improvement in the general condition.

The reduction in the size of the spleen and improvement in the blood picture have already been commented on above.

The increase in weight was extremely variable and in some cases there was very little improvement. Examples are:

<table>
<thead>
<tr>
<th></th>
<th>On admission</th>
<th>On discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male child aged 7 years</td>
<td>37 lbs.</td>
<td>42 lbs.</td>
</tr>
<tr>
<td>Adult female aged 23 years</td>
<td>108 lbs.</td>
<td>110 lbs.</td>
</tr>
<tr>
<td>Male child aged 16 years</td>
<td>102 lbs.</td>
<td>108 lbs.</td>
</tr>
<tr>
<td>Female child aged 7 years</td>
<td>38 lbs.</td>
<td>41 lbs.</td>
</tr>
<tr>
<td>Male child aged 13 years</td>
<td>67 lbs.</td>
<td>74 lbs.</td>
</tr>
</tbody>
</table>
In these patients on admission there was a marked degree of splenomegaly and the spleens must have weighed quite considerable amounts. This reduction in the size of the spleen, which occurred in all of these cases, also reduced the weight of the spleen and there was a clinical impression that the body weight of some patients fell after treatment commenced, and increased later. It would seem, therefore, that although the total body weight increased but little in some patients, the real improvement was much greater.

The temperature fell to normal within ten days of the institution of treatment in 68% of the patients and was normal in all patients by the completion of treatment.

Spleen punctures were done on discharge in 20 patients and no Leishman-Donavan bodies were found. In addition, 16 patients who refused spleen puncture allowed sternal marrow punctures to be done. Again all specimens were negative.

Relapses.

There have been only two relapses known to the writer in the period covered by the paper.

One patient has relapsed twice after full courses of treatment. This is Case No. 6, a Tugen girl aged 15 years on first admission in April, 1955, who was a moderately advanced case with splenic enlargement of 7 finger-breaths, moderate anaemia, a formal-gel reaction classified as +++ and a spleen puncture positive for Leishman-Donavan bodies. She received a total of 1.5 grammes of pentamidine in ten daily injections and 50 ccs of
pentostam in ten daily injections. On discharge her general condition had improved and no Leishman-Donovan bodies were found on spleen puncture. She was re-admitted in October 1955 complaining of enlargement of the spleen, fever, and pain in the splenic area. On examination the spleen was enlarged 9 finger-breadths below the costal margin and Leishman-Donovan bodies were found in the spleen. As only pentamidine and penstostam were immediately available, a further course of treatment similar to the first was given. She was discharged in January clinically cured but refused permission for spleen puncture or sternal marrow puncture.

In July 1956 she again reported to hospital complaining of pyrexia, anaemia and pain in the splenic area. The spleen was found to be enlarged three finger-breadths and the formol-gel reaction was classified as +. She refused spleen puncture but agreed to sternal marrow puncture after persuasion. Scanty Leishman-Donovan bodies were found in the marrow smear. Since treatment with urea-stibamine presented problems of supervision and the patient was not willing to be transferred to the provincial headquarters for treatment there, it was decided, in view of the report by Kirk (1947) that cases had been treated with two or three courses of antimony without success, and a further course had then been effective, that she should again be given pentamidine and pentostam therapy. Sternal marrow puncture was negative for Leishman-Donovan bodies on discharge in September. She reported to the mobile health unit at Chini ya Milima in January 1957 and was then fit and well.
The other case which relapsed was Case No. 44, a girl aged 20 years, who was first admitted in May 1956 as a mild case of kala-azar. The spleen was enlarged four finger-breadths below the costal margin and Leishman-Donovan bodies were found in the spleen puncture smear. She received a total of 2 grammes of pentamidine and 60 ccs of pentostam and was discharged clinically cured after refusal to allow spleen puncture or sternal marrow puncture. The writer has since been informed that she was readmitted to hospital in July 1957 as a relapsed case of kala-azar but no further details have been forthcoming.

Follow-up of Discharged Cases.

When the original patients were treated at Kabarnet, they were given instructions on discharge that they should report back to the hospital in approximately six months time, even if they felt fit and had no complaints. In addition, letters were sent to the chief of the location from which the patient came. It soon became evident that it was unlikely that the patients would return to hospital for re-examination and, in fact, only seven patients did do so. It was decided, therefore, that it was much more likely that patients would be prepared to travel the much shorter distances to centres visited by the mobile health units and patients were instructed to report to these instead. If a patient did not report to the unit within eight months of discharge from hospital, the health assistant on the unit team visited the home of the patient, and brought them in to the unit for examination by the hospital assistant in charge of the team. The hospital assistants were all familiar with the disease
since their health unit activities were only part-time, the rest of their work being in hospital. They examined the patient with particular attention to the following points: general condition, presence or absence of splenomegaly, pyrexia and clinical anaemia, and a clinical assessment of body weight was made.

As a result of this, thirty three patients were seen by the mobile health unit staff and all were reported to be clinically cured. In addition, a further sixteen patients were reported by friends and acquaintances to be fit and well. Two patients (see above) had relapsed, seven had reported to hospital, thus of the sixty patients discharged from hospital only two were not examined after six months or news received of their condition. It is always difficult to trace individuals in a pastoral tribe who tend to move their homes in search of water and grazing for their stock, and some of the sixteen patients reported to be fit and well had, in fact, moved their homes.

Thus to recapitulate:

68 patients were admitted to hospital.

4 patients died before treatment was commenced and

2 died after the beginning of treatment. Two were still under treatment at the time of writing and sixty were discharged as clinically cured, thirty six of these having been examined for leishmania with negative results.

Of the sixty patients discharged from hospital, two were re-admitted having relapsed, seven were re-examined in hospital six months after
discharge, 33 were examined by mobile health units, fifteen were reported to be fit and well, one was reported to have died and two could not be traced.

Comment.

The results obtained in this series of cases indicate that combined therapy with pentostam and pentamidine is effective and can be used safely in the absence of continuous qualified medical supervision. Thus, the disease, as seen in Baringo appears to be less resistant to treatment than the Kitui form which Manson-Bahr and Heisch (1956) found very resistant. They state that 17 to 50 per cent of patients seen at Tseikuru in Kitui District in 1954 and 1955 had previously received a course of combined therapy. It should be noted, however, that their cases received 4 ccs of pentostam daily by intramuscular injection whereas, in this series, the daily dose of pentostam for an adult was 6 cc intravenously. In its response to treatment the disease as seen in Baringo would appear to resemble the Sudanese form which Manson-Bahr and Heisch report, on the authority of Kirk, as being quite easily cured by pentostam alone.
Kala-azar seems to be spreading in British East Africa and although five years ago it appeared to be confined to Kenya, has been reported now in Uganda and Tanganyika in small numbers. It is also tending to increase in the Sudan.

In Kenya, sporadic cases were reported until the outbreak of the Second World War in 1939. During the war years several outbreaks involving fairly large numbers occurred among East African troops in the northern areas of Kenya. These areas were in the north-east corner of Turkana on the delta of the Omo River, and on the Uaso Nyiro River in the Northern Frontier District.

In 1942 two new foci were revealed in the Machakos and Kitui districts of Ukambani. The numbers increased in the Kitui focus and in 1952 there was a sharp outbreak of the disease in this area with over 3,000 cases in the years 1952-54 inclusive.

This paper describes a further endemic focus of the disease in Baringo District which lies in the Rift Valley on the southern edge of the deserts of the Northern Province. The first cases were discovered in 1954 and 71 cases are now described, being diagnosed over a period of thirty months.

The origin of the disease in Baringo appears to be the movement of infected members of the Turkana tribe from the known focus in the north-east corner of Turkana District into Baringo to sell their stock and to buy cereal grains. The distribution of the disease in Baringo follows the routes taken by the Turkana.

The epidemiology of the disease is described and discussed and
information given on the general disease pattern in the area.

The clinical description of the disease as it occurred in Baringo district is given and from this it appears to resemble the Sudanese form of the disease. It was noted that there tended to be a leucocytosis rather than the leucopenia which is described as being characteristic of this disease. Possible reasons for this are the short duration of the illness in many patients and the high incidence of pre-existing respiratory infections. For various reasons described a standard course of treatment using pentamidine isethionate and pentostam was given. This treatment appears to be sufficiently safe and effective for use by medical staff in the absence of supervision by qualified medical officers. The results of treatment indicate that the cure rate was high.
BIBLIOGRAPHY.


" " (1931) Proc.R.Soc.B., cviii,447,494.


Annual Reports, Med Depts., Kenya, Uganda, Tanganyika, Sudan. (1951-1956)
Annual Reports, District Commissioners, Baringo District, (1944-1956)
Annual Reports, Medical Officer of Health, Kitui District, (1952-56)
Annual Reports, Kabarnet Hospital, (1930-1956)
Annual Reports, Tambach Hospital, (1932-56)


xxxvi,25.


GREGORY, J.W. (1896) "The Great Rift Valley"


HENNINGS, R.O (1951) "African Morning"


MANSON-BAHR, Sir Phillip H. (1954) Manson's Tropical Diseases (14th Ed)


" " (1956) Ibid. llix,208.

" (1926) Indian Med.Res.Mem. No.4
" " (1933) Indian med. Res.,xxi,155.

PETERS, C. (1891) "New Light on Darkest Africa."


THOMSON, J. (1885) "Through Masai land".


VON HOHNET, L. (1894) "The Discovery of Lakes Rudolf and Stephanie"


YOUNG, T.C.M. "Kala-azar in Assam".
ACKNOWLEDGEMENTS.

The work described in this paper has been performed during the course of routine duties as the sole medical officer in charge of Baringo and Elgeyo-Marakwet Districts. I wish to acknowledge the assistance of my wife and of Hospital Assistants William Kiptui Rotich and John Abong Kere, of Laboratory Assistant Paulo Wabuti Wamukya and other members of the African staff of Kabarnet Hospital. My thanks are due to Dr. N.R.E. Fendall, Assistant Director of Medical Services, Kenya, for his advice and encouragement, and to Dr. R.B. Heisch, Senior Parasitologist and other members of the staff of the Division of Insect-borne Diseases. I also wish to thank H.J. Simpson Esq., District Commissioner, Baringo and D.P.L. Chester, Esq., of the Agricultural Department, for their advice on the country and people of Baringo.

Table No. 6 is reproduced from the September 1955 issue of The Journal of Tropical Medicine and Hygiene and the editor's permission for this is acknowledged.