TICK-BORNE RELAPSING FEVER IN SOMALILAND
WITH SPECIAL REFERENCE TO THE BLOOD
SEDIMENTATION RATE

BY


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This paper has been compiled from investigations carried out in Somaliland during the years 1941 to 1945.

Tick-borne relapsing fever in Somaliland differs from the louse-borne infection of the Abyssinian highlands in being far less severe and in having relapses of shorter duration. There was not a single fatality out of the 42 cases treated; this series included 4 Europeans, 31 Somalis and 7 East Africans. Out of 140 pyrexial attacks, the average duration was 3.1 days, the course of an attack varying in individual cases from 1 to 17 days. The interval between relapses also varied greatly, from 2 to 63 days, the average being 8.6 days. The average number of attacks per patient was four, but one patient, a European, had as many as 13 bouts of pyrexia, and was in hospital for a total of 137 days (over four months) before he was fit for discharge.

The disease was definitely more severe in the European cases than in the Somalis and East Africans. One European developed the complication of iritis while another suffered from cerebral symptoms with intractable headache and vomiting, neck rigidity and positive Kernig's sign. The latter patient, whose symptoms were relieved by lumbar puncture, showed a raised cerebrospinal pressure with an excess of cells and protein in his fluid.

A total of eight lumbar punctures was performed in this series, 5 of the patients suffering from neck rigidity and showing Kernig's sign. The C.S.F. protein was increased in 5 of the cases (4 of whom were suffering from neck rigidity) and there was a cobweb clot on standing in one case. Spirochaetes were not seen in films of the C.S.F., but might have been demonstrated by animal inoculation.

Complications included the following: iritis in 3 cases; basal pneumonia in 2; dysentery of the bacillary type (not responding to succinyl sulphathiazole) in 2; diarrhoea in 3 others; acute nephritis in one; hepatitis with jaundice.
in 1; herpes of the right auricle in 1; left cervical neuritis in 1; and uni-
lateral otitis media in 1.

An interesting case of Jacksonian epilepsy was seen. The patient was a Somali. It
is unfortunate that, as no spirochætes were recovered from his blood, the diagnosis could
not be proved, but his relapsing type of pyrexia suggested that he was suffering from
relapsing fever. The cerebrospinal fluid showed no abnormality and was not increased
in pressure. Each fit commenced in the right hand and then spread rapidly up the arm
and thence simultaneously to the right side of his face and lower extremity. The left side
of the body was unaffected. There was no unconsciousness. The attacks rapidly succeeded
one another and persisted with diminishing severity for fourteen days. Between the spasms
there was a flaccid upper motor neurone paralysis. After two weeks the fits, which had
diminished in number under phenobarbitone, ceased altogether. The power came back
in his right limbs, and within twenty-four days of his admission the abdominal reflexes
had returned and the plantar responses had become flexor. He was discharged walking
with a slight limp after eight weeks in hospital.

The Wasserman reaction is said to be positive in about 20 per cent of cases
(Manson-Bahr, 1945; Heilman and Herrell, 1943), but out of 8 cases examined
in my series the Kahn test was negative in all. The majority of these patients
were natives of British Somaliland who, being strict Mohammedans, rarely
suffered from syphilis. These findings are in agreement with those of Garnham
et al. (1947) who, during their investigations on a louse-borne epidemic in
Kenya, found the Kahn reaction positive in only 3 out of 36 specimens of blood
examined at different stages of the disease; they state that the positives were
probably due to syphilis or yaws.

There is usually said to be a leucocytosis in this disease. Out of 26 leucocyte
counts performed during the attack, the average total count was 7,795 (varying
between 3,000 and 14,000), and the average neutrophil percentage 60 (varying
between 45 per cent and 90 per cent). The neutrophil count varied from
1,740 to 10,920, the average being 4,389. Only 3 out of 18 cases had counts
over 6,000 per c.mm. In this series, therefore, a neutrophilia was the exception
rather than the rule.

There was commonly a secondary anæmia, the average red cell count being
4,140,000 per c.mm., the haemoglobin 71 per cent (Sahli) and the colour
index 0·84.

The blood sedimentation rate is so commonly employed nowadays as a
sign of recovery from active disease, especially in rheumatic fever and tuber-
culosis, that I was tempted to investigate whether it might be of value as an
index of complete recovery or of the susceptibility to future relapses. The
 technique employed in Somaliland was that of Westergren. It was not possible
to correct for anæmia, neither is such a correction entirely reliable, since the
correction is based upon dilution of the blood which is not strictly comparable
to clinical anæmia (Davis, 1946), and since no allowance is made for the natural
power of anæmic blood to compensate for its tendency to rapid sedimentation
(McFarlane and O’Brien, 1946). Moreover, the Westergren is less susceptible
than the Wintrobe method to the effects of anæmia (Davis, 1946).

One hundred and fifty-nine B.S.R. examinations were performed, including
113 tests on Somalis, 27 on East Africans and 19 on Europeans. The B.S.R. taken during attacks varied greatly (from 4 to 110 mm.), the average of 19 examinations being 61.7 mm. in 1 hour (49.2 in the Somalis, 78 in the East Africans, and 58 in the Europeans). (Tables I and II.)

**Table I.**—Average B.S.R. During Attacks

<table>
<thead>
<tr>
<th></th>
<th>Average B.S.R.</th>
<th>Number of tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>58.0 mm.</td>
<td>2</td>
</tr>
<tr>
<td>East African</td>
<td>78.0</td>
<td>5</td>
</tr>
<tr>
<td>Somali</td>
<td>49.2</td>
<td>12</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>61.7 mm.</strong></td>
<td><strong>19</strong></td>
</tr>
</tbody>
</table>

**Table II.**—Average B.S.R. for Each Day of Attack

<table>
<thead>
<tr>
<th>Day of attack</th>
<th>Average B.S.R.</th>
<th>Number of tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>79.8 mm.</td>
<td>5</td>
</tr>
<tr>
<td>2nd</td>
<td>65.3</td>
<td>6</td>
</tr>
<tr>
<td>3rd</td>
<td>42.0</td>
<td>2</td>
</tr>
<tr>
<td>4th</td>
<td>4.0</td>
<td>1</td>
</tr>
<tr>
<td>5th</td>
<td>30.0</td>
<td>2</td>
</tr>
<tr>
<td>6th</td>
<td>110.0</td>
<td>1</td>
</tr>
<tr>
<td>11th</td>
<td>6.0</td>
<td>1</td>
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An attempt was made to ascertain whether the B.S.R. dropped to normal between attacks or whether it could be used as an index of prognosis in the prediction of further relapses. Examinations were therefore made at intervals for several weeks after the last bout of temperature. The results are depicted in Table III.

**Table III.**—Average B.S.R. After Attacks

<table>
<thead>
<tr>
<th></th>
<th>East African</th>
<th>European</th>
<th>Somali</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weeks</td>
<td>Average B.S.R.</td>
<td>Number of tests</td>
<td>Average B.S.R.</td>
<td>Number of tests</td>
</tr>
<tr>
<td>1st</td>
<td>40.9</td>
<td>10</td>
<td>38.7</td>
<td>9</td>
</tr>
<tr>
<td>2nd</td>
<td>35.7</td>
<td>6</td>
<td>28.2</td>
<td>5</td>
</tr>
<tr>
<td>3rd</td>
<td>51.0</td>
<td>6</td>
<td>13.5</td>
<td>2</td>
</tr>
<tr>
<td>4th</td>
<td>—</td>
<td>—</td>
<td>30.0</td>
<td>1</td>
</tr>
<tr>
<td>5th</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>6th</td>
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<td>7th</td>
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<td>8th</td>
<td>—</td>
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<td>—</td>
</tr>
<tr>
<td>9th</td>
<td>—</td>
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</table>

As shown in the table, there is a tendency for the figures to diminish after the fourth week, but there was still a tendency to a raised level even after two months. There was, of course, great variation in individual figures. A few cases dropped permanently to normal after a few weeks, as far as could be calculated from estimations over a period of three to four weeks. An interesting point
about these tests was the marked and sudden variations in B.S.R. without any corresponding rise of temperature or change in the patient's clinical condition. For instance, one patient showed a rise from 22 to 90 mm. during the three weeks after his last attack, in spite of the fact that he was feeling perfectly well and was discharged to his unit after four weeks. Another dropped to nil in three weeks, rose to 80 in four weeks, and again dropped to 9 in five weeks, without any rise of temperature or change in his general health. A third fell to one in two days, rose to 96 in eight days, and again fell to 4 in twenty-four days. One patient had a B.S.R. of 112 mm. ten days after his second attack and six days before his last rise of temperature to only 99°; he felt very fit at the time of these tests. One had a very high rate for three weeks after his third attack, rising to a maximum of 132 mm. four days before his last relapse, in spite of the fact that he was feeling quite well in the intervals between his pyrexial bouts.

B.S.R. estimations were also made before relapses (Table IV) in order to find out whether there was any tendency for a rise to occur as the next relapse became more imminent. The average was 40·9 mm. one week, 46·5 mm. two weeks, and 41·5 mm. three weeks, before the attack. There was, therefore, no tendency for the B.S.R. to rise as the time approached for the next relapse.

One is tempted, after studying the B.S.R. figures, which remain raised for weeks after all symptoms have subsided, to conclude that, even after all clinical signs have disappeared, there still remains a latent infection in the body. Such a view is supported by the long interval which may sometimes occur between relapses (sixty-three days in one case), and by the fact that spirochaetes have been isolated from the brains of guinea-pigs fourteen months after primary inoculation (Manson-Bahr, 1945), and by the insidious manner in which the organism is transmitted by the tick through its ova even to the third generation, without the necessity for any intermediary host.

It appears that the spirochaete of relapsing fever resembles that of syphilis in its characteristic property of remaining latent in the body for long periods without causing symptoms. I had one patient who developed a bout of relapsing fever (confirmed by the discovery of spirochaetes in his blood) exactly one year after his previous attack; one wonders whether the second attack was a fresh infection or a very late relapse of his original illness. Another feature common
to the spirochaetes of relapsing fever and syphilis is the neurotropic character and the tendency to latent infections in the nervous system. Garnham et al. (1947), working in East Africa, found that neurotrophism was rare in the louse-borne spirochete, although invariably present in the tick-borne variety. An interesting feature of the B.S.R. figures is the curious way in which the B.S.R. fluctuates without any corresponding clinical signs of activity. This phenomenon suggests that fluctuation may occur in the intervals of the infection quite independently of the clinical features as indicated by relapses of pyrexia.

Treatment.—There have been so many reports about the value, or lack of value, of arsenical therapy that, at the suggestion of Brigadier E. R. Cullinan, I decided to investigate its therapeutic action, employing untreated controls taken at random. These tests were performed at Mandera, in Somaliland, and at Diredawa, in Abyssinia, the patients including Europeans, East Africans and Somalis. Neosalvarsan 0.6 gramme was given intravenously at the onset of the first relapse, the primary attack being untreated on account of the difficulty of seeing the patient at the onset of his disease. 20 patients were treated and 14 employed as untreated controls. The following were the statistics drawn from 91 attacks of pyrexia amongst the treated cases and 49 amongst the untreated:

Of the 20 treated cases, 18 relapsed after arsenical therapy.
It must be concluded that at Mandera and Diredawa arsenical therapy cannot be claimed to have had any therapeutic value.

At Borama, in British Somaliland, on the other hand, and at Urso, in Abyssinia, 4 Free French Somalis were treated without a single subsequent relapse. 5 patients were also treated at Yatta, in Kenya, 4 of whom responded without a recurrence and 1 had a single extremely mild relapse. Quin and Perkins (1946), working among African troops in East Africa, observed little benefit from N.A.B. injections, the 80 cases treated exhibiting the same average of two relapses as the 49 controls.

<table>
<thead>
<tr>
<th>Table V.—Therapeutic Effect of Intravenous Arsenic in Tick-borne Relapsing Fever at Mandera and Diredawa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Average</strong></td>
</tr>
<tr>
<td>number of attacks</td>
</tr>
<tr>
<td>from onset</td>
</tr>
<tr>
<td>Treated cases</td>
</tr>
<tr>
<td>Untreated cases</td>
</tr>
</tbody>
</table>

It therefore appears that, in certain localities, arsenic did produce benefit, while in other districts it had no effect whatsoever. The explanation probably lies in the fact that certain strains of tick-borne spirochaetes have become arsenic-resistant, possibly through previous arsenical treatment of these strains, just as strains of gonococci become sulphonamide-resistant in Italy and elsewhere.
A similar theory probably explains the differing views with regard to the value of arsenic in the louse-borne disease for, whereas the author (1942) found this drug to be of great value in the Soddu district of Abyssinia, and Garnham, Davis, Heisch and Timms (1947) found it of benefit in the Kenya disease, Wolman (1944) claimed that it had no effect in Addis Ababa.

Penicillin was administered to 2 patients with tick-borne relapsing fever, both Europeans, in the dosage of 15,000 units every three hours for six days. One of the patients, whose treatment commenced during the sixth attack, developed seven subsequent relapses, while the other, treated during his fifth attack, suffered no further pyrexia.

The general impression was that penicillin, in the dose given, was ineffective in preventing relapses. This would be expected in view of the results of animal experiments, for the effective dose in mice is 400,000 units per kg. administered in divided doses every three hours over a period of forty-eight hours. This dose approximated closely to the toxic dose for the particular preparation employed. Applying these results to man, the curative dose would be 25,000,000 units for a man of 60 kg. (Eagle, Magnuson and Musselman, 1944).

It must, however, be admitted that the first patient appeared to obtain symptomatic benefit. He had been ill for thirty-nine days with a very severe persistent headache. He suffered from attacks of vomiting and, although his Kernig's sign was negative and neck rigidity was absent, his cerebrospinal fluid was under increased pressure and contained 46 mg. protein per 100 c.c. The cell count was 9 per c.mm., the chloride 770 mg. and the sugar 50 mg. per 100 c.c. Lumbar puncture relieved his headache for twenty-four hours, after which it returned with its previous intensity. Penicillin was started during his sixth relapse. His headache began to improve twenty-four hours after the commencement of the penicillin and had disappeared by the end of the six days' course. The interval between relapses, which had previously not exceeded eight days, was extended to twenty-two days before his next bout of pyrexia. In view of the natural tendency for this disease to diminish in severity during its course, it is impossible to form any definite conclusion on these isolated cases. At the present time opinion with regard to the value of penicillin in human relapsing fever is divided. Workers on the tick-borne variety, for example, have had little success; they include Merskey (1947), who observed no improvement in 2 cases treated with under 1,000,000 units in Cullinan, Transvaal; Pospelova-Strom and Tiburskaya (1946), who, employing a dosage of 23,000 and 50,000 units on the second and third days respectively of the primary attack, were unable to prevent a relapse in an Iranian strain of spirochaete; and Muwazi, who administered four-hourly intramuscular injections, up to a total dosage of 900,000 units in forty-eight hours, to 29 patients in Uganda, without apparent benefit. Better results have, however, been recorded in louse-borne relapsing fever by Ingraham and Lapenta (1946), who cured 52 cases in Egypt with 25,000 units of penicillin every three hours, and by Ling (1947), who was able to prevent relapse in 2 cases of Chinese relapsing fever by doses of 960,000 and 1,780,000 units. Streptomycin holds
out more hope of success, and has already proved of greater value than penicillin in the eradication of residual cerebral infection in mice infected with *Treponema duttoni* (Levaditi and Vaisman, 1947).

A Somali with iritis, and spirochaetes in his blood, was treated with two-hourly instillation of penicillin, 2,000 units per c.c., into his conjunctival sac, and improved within twenty-four hours. It is, of course, impossible to form an estimate of the value of local penicillin from the examination of a single case.

**SUMMARY**

(1) An account of the clinical features and complications of tick-borne relapsing fever in Somaliland has been given.

(2) The Kahn test was negative in the 8 cases examined.

(3) In this series, neutrophilia was the exception rather than the rule.

(4) B.S.R. estimations were made during, and for several weeks after, attacks. There was a tendency to a raised rate for several weeks after an attack, and to fluctuation in the B.S.R. without any corresponding clinical signs of activity.

(5) It is believed that a latent infection persists in the body (possibly the brain) for a considerable period after all clinical signs have disappeared. An analogy is drawn to the latency and neruroporic character of *Treponema pallidum*.

(6) Arsenical therapy was of benefit in some areas, while proving of no value whatsoever in other districts, the explanation probably being the occurrence of arsenic-resistant strains of spirochaetes in certain localities.

(7) Penicillin did not prevent relapses.

**REFERENCES**


London: Cassell & Co.


