Original Communications.

CLINICAL MANIFESTATIONS OF TICK-BORNE RELAPSING FEVER WITH SPECIAL REFERENCE TO THE DISEASE IN CYPRUS

BY

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Epidemiological and entomological aspects of relapsing fever in Cyprus have been discussed previously by Gambles and Coghill (1948). In this communication the clinical features of the disease in the island are presented, followed by a discussion on the possibility of clinical differentiation of tick- from louse-borne forms as they occur throughout the world.

Records were collected of 35 military cases infected in Cyprus, which came under my care. 29 were Indians, 1 a Cypriot and 5 English. All had positive blood films.

Both forms of relapsing fever, perhaps especially the tick-borne, show considerable variation from case to case and locality to locality. The author's experience proved no exception to this and illustrative case histories are given.

The following examples are representative of what may be termed "straight-forward" cases:

Case 16.—Indian, Sepoy, aged 40. Admitted to 82 General Hospital (82 G.H.) on 22.6.42. Fever and headache without rigor, started on 17.6.42. There were generalized body and joint pains. He was still febrile on admission and a blood film contained spirochetes. He was given sulphostab 0.3 gramme intravenously (I.V.) and five days later 0.45 gramme. Discharged to unit on 30.6.42.

He relapsed, and was readmitted, on 8.7.42. He received no treatment and relapsed again with a rigor on 16.7.42, the day he was transferred to another hospital. While at this other hospital it is not known if he had any treatment, but there appear to have been no further relapses. Discharged to unit on 28.7.42.

On the same day he experienced his third relapse and was admitted to a staging section. A fourth relapse occurred on 2.8.42 when he was noted to be tender in the liver area. He had a fifth relapse on 10.8.42. A sixth occurred on 21.8.42 (T. 103° F.). He was admitted once more to 82 G.H. on 23.8.42, symptomless. On 1.9.42 he complained of pain over the spleen which was found enlarged and moderately tender. The liver was just palpable and not tender. The next day the patient shivered and his temperature rose to 102.4° F., constituting the seventh relapse. On this occasion the spleen was difficult to feel but he remained moderately tender in the area of that organ. In error treatment was not given.

Eighty-one days after the onset the patient went to Convalescent Depot (Con. Dep.) on 5.9.42, and thence to unit on 3.10.42. There were no further relapses.
Case 27.—U.K. Serjeant, aged 27. Admitted to 82 G.H. on 17.11.42. Onset on 15.11.42 with a feeling of cold (no shivering); headache; pain in the legs, back and loins; considerable nausea but no vomiting; a feeling of weakness; temperature 100° F. The next day he felt worse except for the nausea which was eased, and he developed a moderately severe continuous epigastric ache. Apart from fever (see chart) the only physical sign was moderate tenderness over the liver and in the epigastrium. A blood film was negative. There was general improvement as the fever diminished. A leucocyte count on 26.11.42 was normal (Table II). On 28.11.42 he complained of malaise; pain in the arms, severe backache and mild headache; he was rather prostrated; a blood film contained spirochetes as the temperature rose; sulphostab 0·6 gramme I.V. was given at the height of the fever. The next day he was very much improved. There were no symptoms by 30.11.42, but the following day he developed a tender point on a left lower rib giving rise to occasional shooting pains in this area for a day or two. Thereafter he was well until 12.12.42, when a lumbar ache appeared and he felt “very tired.” He slept little that night and the next day had severe backache and frontal headache, and mild pains in the legs. There was more fever (second relapse) and a blood film was again positive. His urine contained much increased quantities of urobilinogen (see chart). Novostab 0·6 gramme I.V. was given as the temperature was rising. Later he vomited twice. The next day he was much improved and became symptomless by 15.12.42. Novostab 0·6 gramme I.V. was repeated on 20.12.42 and 27.12.42. Discharged to unit on 21.12.42. There were no further relapses.

TEMPERATURE CHART. CASE 27.

Case 30.—Indian, Sepoy, aged 22. Admitted to 82 G.H. on 10.2.43. Onset on 9.2.43 with fever, malaise, headache and rigors. On admission there was further shivering but no other physical signs. Blood films were negative. The symptoms and temperature did not properly settle (see chart) but what was probably a true relapse occurred on 18.2.43, with the first positive blood film. The next day the temperature dropped and he had no symptoms, although the spleen was just palpable and the urine contained a great excess of urobilinogen. Novostab 0·3 gramme I.V. was given on 20.2.43; 0·6 gramme on 25.2.43, and on 4.3.43. He remained well until 9.3.43, when he had a frontal headache, severe at times, for two to three days; there was no fever. Thereafter he remained well and was discharged to Con. Dep. on 16.3.43. There were no further relapses.
Case 33.—Indian, Sepoy, aged 21. Onset of fever with a rigor on 7.2.43 and the next day was admitted to a staging section. There he had a cough with expectoration. The lungs contained numerous rhonchi and the spleen was felt. This attack subsided and he was admitted to 82 G.H. on 15.2.43 when his complaints were vague pain in the chest, persistent cough and malaise. He was afebrile (see chart). The first relapse started on 16.2.43; he had rigors and some back pain; a positive blood film was first obtained on 18.2.43; cough continued; he became moderately tender over the liver. Urinary urobilinogen was much increased on 19.2.43. He received 0.3 gramme novostab I.V. on 20.2.43 and 0.6 gramme on 25.2.43. The cough improved over a period of days and disappeared, but the tenderness in the right hypochondrium persisted although the liver was not felt. The patient felt weak. On 3.3.43 the tenderness spread to the left hypochondrium, but the spleen was not palpable. The next morning he received his third injection of novostab (0.6 gramme) in spite of which his temperature rose from normal to 102°F. that evening. He was moderately ill with this attack. There was pain across the front of the chest, and pains all over the body; and on 6.3.43 vomiting and occipital headache. This was his first complaint of headache. Bronchitis reappeared; his abdomen was moderately tender in umbilical and epigastric areas, and along both costal margins, with some abdominal wall resistance; no organs were felt. There was slight neck stiffness. He was greatly improved the next day. On 10.3.43 he complained only of painful muscles over the front of the chest and upper abdominal aching pain. The pectoral muscles remained moderately tender for some days; the upper non-muscular abdominal tenderness remained as before. The fundi were normal. On 13.3.43 the knee and ankle jerks, previously always brisk, were found to be equal and sluggish. Three days later, although symptomless, his upper abdominal tenderness had increased and the liver was now palpable one finger's breadth (f) below the costal margin. At this time there was a mild leucocytosis (Table II). On 18.3.43 his third relapse appeared, with headache. By 27.3.43 his tendon-jerks were brisk again, those at the knee being mildly exaggerated. A fourth relapse occurred on 28.3.43, with headache, vomiting, insomnia and rigors. There was a constant aching pain along both costal margins with
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tenderness as before. The spleen could now be felt 1½ f. below the costal margin which was considerably tender. Novostab 0·45 gramme was given as the temperature was on the second up-grade. Between this relapse and the next he became symptomless and the abdominal signs diminished to slight epigastric tenderness. The fifth relapse started on 9.4.43 with vomiting, epigastric pain and mild headache. During the night he passed 9 loose stools containing mucus but no blood. The abdominal signs were as in the previous relapse. Novostab, 0·6 gramme I.V. was given on 10.4.43 as the temperature was falling. The abdominal symptoms and signs slowly diminished and then began to reappear on 29.4.43; the liver was again felt on 1.5.43. At this time he looked and felt far from well, and had obviously lost a considerable amount of weight. The abdominal signs again diminished after 5.5.43 but a relatively symptomless sixth relapse occurred on 9.5.43. He became free of symptoms and signs on 13.5.43 and began to put on weight. Discharged to unit fit on 4.6.43, 118 days after the onset. There was no further fever.

TEMPERATURE CHART. CASE 33.

The following are histories of those cases with complications: Cases 18, 22 and 25 are examples of meningitis; Case 34 of encephalitis and Cases 10 and 32 of facial palsy. Cases 18, 22, 25 and 34 developed varying degrees of papilloedema during the course of their neurological complications.

Case 18.—Cypriot, Private, aged 20. Admitted to a surgical ward in 82 G.H. on 29.7.42 as a case of acute appendicitis. The previous day there had been a sudden onset of pain in the right abdomen. The pain was worst at a point above and to the right of the umbilicus, and was constant and aching in character. He felt tired and ill; there was nausea, fever without shivering, headache, dry cough and an aching pain down the length of the back. He had had no similar illness before. On admission he appeared flushed and ill. The surgeon noted that there was no abdominal resistance but the patient was very tender over McBurney's point. The liver was palpable 2 f. below the right costal margin on inspiration and considerably tender; the spleen was similarly enlarged 2 f., hard and not tender. The diagnosis of appendicitis was doubted.

During the next four days he was constipated; blood films were all negative; the temperature became normal by 31.7.42 (see chart); X-ray of lungs and liver areas showed nothing abnormal; 3 W.B.C. counts were normal (Table II).

He was transferred to a medical ward on 3.8.42 with a tentative diagnosis of amoebic hepatitis. At this time he felt better in himself, but the backache and abdominal pain remained fairly severe, the last being worse when lying on his right side. The abdominal tenderness was now most marked over an area a little above and to the right of the
Clinical Manifestations of Tick-borne Relapsing Fever

umbilicus, corresponding to where he felt the pain, but there was also moderate tenderness in the R.I.F. His liver remained the same. His condition was unchanged until 6.8.42 when the temperature rose again and he felt worse. A blood film now contained spirochaetes and he was given sulphostab 0·6 grammes i.v. The next day he felt better but he had a headache and vomited. By 8.8.42 his temperature was normal, his general condition much improved and the abdominal pain and tenderness began to go. Soon there was a marked improvement but mild abdominal tenderness persisted until 14.8.42 when a second dose of sulphostab, 0·6 grammes was given. A few days after this his back pain disappeared, his abdomen becoming free of pain and tenderness.

He continued to complain of headache which became worse and on 23.8.42 was moderately severe. The pain was frontal and behind the eyes. The temperature was 99·6°F and the only sign was the enlarged spleen noted before. The next day there was a morning temperature of 99·8°F. On 25.8.42 he began vomiting. The headache was then severe and general; he was ill and drowsy; there was pain on moving his eyes and severe anorexia. There was marked neck rigidity but no Kernig's sign. The condition remained unchanged with persistent vomiting until 27.8.42 when the first lumbar puncture was performed, 7 c.c. clear colourless fluid being withdrawn (Table I). The next day there was no vomiting but occasional nausea and the same headache. For the first time there were now further signs in the C.N.S.; knee-jerks sluggish; all arm-jerks absent on the right. Fundi, moderate and equal; papilledema, most marked on the left. His vision deteriorated, but later recovered. Blood Kahn reaction negative. The condition was much the same on 29.8.42 when a second lumbar puncture was done. On this occasion 20 c.c. fluid (clear and colourless) were slowly withdrawn with coincident improvement of the headache. This was only slight the next day and the neck stiffness began to improve. The knee-jerks were now a little brisker. Fundi: very considerable oedema of the discs whose margins were almost invisible; vessels very congested. For the first time since 23.8.42 the temperature was 99°F.

He continued to improve until 3.9.42 when the headache became worse again. The

TEMPERATURE CHART. CASE 18.

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fundis remained the same except for the discs becoming slightly paler. A third lumbar puncture was performed, 15 c.c. clear colourless fluid being taken off. The Kahn reaction on the c.s.f. was negative. At this time there was mild vomiting but the headache was relieved by the lumbar puncture. He now began to run a low fever. The condition remained the same until 5.9.42 when novostab 0·6 gramme I.V. was given. Thereafter he slowly improved, gaining all the considerable amount of weight he had lost, and he made a complete recovery in every way. The spleen remained the same throughout. Discharged to Con. Dep. on 18.9.42. There was no further fever.

Case 22.—Indian, L./Naik, aged 20. Fever and headache first appeared on 2.8.42. He was admitted to another hospital on 5.8.42 and again had fever on 6.8.42 (first relapse). Blood films were negative and he was discharged on 8.8.42. The second relapse occurred on 12.8.42 for which he was readmitted to the same hospital. On this occasion it was noted that the liver was impalpable, but the spleen was felt one inch below the costal margin. Spirochetes were now found in a blood film. The next day a blood Kahn reaction was doubtfully positive and he received 0·3 gramme novostab, I.V.

He was transferred to 82 G.H. on 21.8.42, complaining of slight headache and pain in the back. The third relapse occurred on 26.8.42. On this occasion the blood film was negative. He was given novostab, 0·6 gramme I.V. during the fever, and his temperature became normal by 28.8.42. On 30.8.42 the liver was mildly enlarged, and the spleen moderately so and hard. On 1.9.42 there was more fever (fourth relapse) and headache. A blood film was negative and he was given novostab 0·6 gramme I.V. He improved the next day but on 3.9.42 complained of a severe general headache. There was now marked neck rigidity with mild fever (see chart). Fundi: mild papilloedema. A lumbar puncture was performed (Table I) and 4 c.c. clear colourless fluid withdrawn. The headache was almost completely relieved at the time by this operation. The same day he was given a further 0·6 gramme novostab, I.V. in error. On 6.9.42 he began to complain of aching pain in both sternomastoid muscles, which were tender. This continued, otherwise with general improvement, until 11.9.42 when he again had a severe

TEMPERATURE CHART. CASE 22.

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[Graph and chart information]

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DAY OF DISEASE.

Temperatur chart.
headache for twelve hours. The next day there were no symptoms and on 21.9.42 he was sent to Con. Dep. apparently well.

Two days later he had a headache and on 24.9.42 the fifth relapse occurred. A blood film was negative. He was given 0.6 gramme novostab, I.V. Mild pain and tenderness of the sternomastoid muscles recurred the next day, and persisted for some days. The temperature rapidly became normal after the novostab, but was 99.6° on 27.9.42 and 99° two days later. Thereafter he made a complete recovery, being discharged fit to his unit on 12.10.42. There was no further fever.

Case 25.—Indian, Sepoy, aged 22. Taken ill on 1.11.42 with fever, shivering, headache and lumbar pain. Admitted to another hospital on 3.11.42 when P. falciparum were seen in a blood film. Routine malaria treatment was started and he was transferred to 82 G.H. on 9.11.42. The next day there was more fever (see chart) and spirochætes were found in a blood film. Taking this as the primary attack of relapsing fever his first relapse was on 17.11.42 with the sudden onset of fever, headache and pains in the legs. This subsided, but on 21.11.42 he began to complain of frontal headache, worse at night; lumbar pain; mild leg pains. The abdomen was mildly tender in the L.I.F. and left hypochondrium but no organs were felt. The lumbar muscles were very tender on both sides. There was moderate neck stiffness but no Kernig and no other C.N.S. signs. There was further fever the next day and he vomited. On 25.11.42 he had a severe frontal and mild occipital headache. He was more tender in the L.I.F. The neck stiffness had increased. Fundi: mild papilloedema on the left. On lumbar puncture 12 c.c. clear colourless fluid were withdrawn under a pressure of 130 mm. of c.s.f. This was normal in every way, and contained no cells. The headache was somewhat relieved by this operation. The next day the third relapse occurred and for the first time novostab, 0.6 gramme I.V. was given, when the temperature was at its peak. Two days later he was improved but lumbar ache and tenderness, and moderate neck stiffness, persisted. The knee-jerks were now very sluggish, but equal, and the ankle-jerks mildly reduced; previously all had been brisk. The fundal appearances remained the same. Four days later there was further improvement and the tendon reflexes were normal again. The left disc did not become normal until 9.12.42. The blood Kahn was negative.
on 2.12.42. Mild lumbar pain and tenderness persisted, with occasional headache and pains in the thighs until 12.12.42 when the fourth relapse appeared with exacerbation of all the symptoms he had had back to 25.11.42. Neck stiffness reappeared and all the lower limb tendon reflexes were very sluggish. He was given 0.6 gramme novostab, I.V. Thereafter he slowly improved. The spleen was felt 1 f. down for the first time on 16.12.42. The knee and ankle jerks were normal on 26.12.42 but those at the knee were again very sluggish on 6.1.43 by which date he became free of symptoms. Discharged fit to unit on 11.1.43. There was no further fever.

Case 34.—Indian, Sepoy, aged 22. Onset on 7.2.43 of fever with malaise (see chart). Admitted to a staging section where he had signs of bronchitis. He complained of pain over the right chest in front, and vomited once. He had a headache most evenings until admitted to 82 G.H. on 15.2.43. The next day the first relapse occurred with a rigor and joint pains. The second relapse appeared on 24.2.43 when a positive blood film was obtained for the first time. He was given 0.3 gramme novostab, I.V. on 26.2.43 as the temperature was falling. At this time the liver edge could just be felt, and was moderately tender. The spleen, not previously palpable was enlarged 2 f. downwards, soft, but not tender. On 3.3.43 there was no abdominal tenderness and the spleen was smaller. He received novostab 0.6 gramme I.V. The third relapse occurred on 7.3.43 and fever continued for eight days. At first he complained only of headache and malaise but the knee-jerks, previously normal, were sluggish on 10.3.43. On this date he received novostab 0.6 gramme I.V. He became lethargic and a little drowsy with a severe headache, and on 13.3.43 the knee-jerks were absent and ankle-jerks a little diminished for the first time. At no stage was there neck stiffness. By 16.3.43 the leg reflexes were recovering but a mild degree of bilateral papilloedema had developed. There was no anaemia and the W.B.C.s were normal (Table II). The fourth and last relapse (very mild) occurred on 18.3.43. Thereafter he improved rapidly in every way. The spleen remained 1 f. palpable and became much firmer. On 24.3.43 there was a slight leucocytosis. Discharged fit to unit on 23.4.43. There was no further fever.
Case 10.—Indian, Sepoy, aged 20. Onset of fever on 31.5.42 and admitted to 82 G.H. on 4.6.42, still febrile. He complained of a "head cold" and sore throat but throat signs were minimal. He was tender in the epigastrium and the spleen was palpable 1 f. A blood film showed spirochætes. On the day of admission 0·3 grammes of sulphonamide was given I.V.; five days later 0·45 grammes and seven days after that 0·6 grammes. Discharged to unit on 16.6.42, no further fever having occurred since 5.6.42.

Thirty-one days after the end of the first attack he relapsed, with a rigor. He was readmitted to 82 G.H. on 8.7.42 and the blood film again contained spirochætes. He had a right Bell's Palsy which had appeared with the relapse of the fever. The paralysis was unilateral, complete, and of lower motor neurone type. He received no treatment.

On 5.8.42 his temperature rose again, with a rigor, and on this occasion *P. vivax* only were found in a blood film. This fever responded normally to routine malaria treatment. No further fever occurred. The facial paralysis had shown no sign of improvement by 26.8.42 (fifty-three days after its onset) when he had to be transferred to another hospital.

Case 32.—Indian, Havildar, aged 35. Admitted to 82 G.H. on 11.2.43. Onset on 8.2.43 of a dull aching pain in all his joints. Fever (see chart) began on 11.2.43 with rigor, malaise and anorexia. The spleen was enlarged 1 f. A blood film contained spirochætes on 12.2.43, but novostab, 0·3 grammes I.V., was not given until 15.2.43, when there were no symptoms, and was repeated on 22.2.43. On 24.2.43 the spleen remained as before, and the liver became transiently enlarged 1 f., but not tender. Novostab, 0·6 grammes was given on 1.3.43. He was afebrile and symptomless from 15.2.43 until 12.3.43 when the first relapse occurred with rigor and slight headache. The spleen was now enlarged 2 f. and rather hard. The knee-jerks, previously brisk, were now sluggish, but became normal again on 16.3.43. The second relapse appeared on 22.3.43. He had a rigor but no headache. Novostab 0·45 grammes was given on 23.3.43. The next day he complained of tightness and "uneasiness" in the abdomen, which was not distended but was a little tender along the right costal margin. The liver was not felt but the spleen remained the same size and was rather softer. Thereafter all symptoms and signs rapidly went and the spleen could not be felt on 6.4.43. He remained quite well until 22.4.43 when there was a fairly sudden onset of left facial

![Temperature Chart](image-url)
paralysis without fever. The palsy was complete and of lower motor neurone type. Recovery began slowly on 1.5.43. At lumbar puncture on 5.5.43 a few c.c. of clear colourless fluid were withdrawn (Table I). Recovery of the paralysis was sustained and

**Table I.**—The Findings in the Cerebrospinal Fluid in the Two Cases of Meningitis and One of Facial Palsy in Which They Were Abnormal.

<table>
<thead>
<tr>
<th>Meningitis: or Facial Pressure</th>
<th>W. B. C.</th>
</tr>
</thead>
<tbody>
<tr>
<td>(M.) Palsy: in mm. C.S.F.</td>
<td>Total %</td>
</tr>
<tr>
<td>------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>18 M.</td>
<td>+</td>
</tr>
<tr>
<td>400+</td>
<td>26</td>
</tr>
<tr>
<td>150</td>
<td>11</td>
</tr>
<tr>
<td>22 M. \ 70</td>
<td>+</td>
</tr>
<tr>
<td>32 F.P. \ 0</td>
<td>20</td>
</tr>
<tr>
<td>+ +</td>
<td></td>
</tr>
<tr>
<td>Glob. + +</td>
<td>= normal.</td>
</tr>
<tr>
<td>Alb. + +</td>
<td>Laboratory unable to estimate.</td>
</tr>
</tbody>
</table>

soon complete by 16.6.43, when he was discharged to his unit. On 4.5.43 he began to have pot. iod. gr. xx t.d.s.; on 5.5.43 bismostab 0·2 grammes I.M. every seven days for six doses; and tryparsamide 1·5 grammes I.V. on 6.5.43 and 13.5.43, and 2·5 grammes on 20.5.43 and every seven days for four doses (total 13 grammes). This treatment was recommended for such cases by the Consultant Neurologist, M.E.F. Improvement began three days before treatment started.

The following notes are of cases which showed relatively unusual features in one way or another:

**Case 4.**—Indian, Sweeper, aged 25. Admitted to 82 G.H. on 25.5.42. There was sudden onset of fever on 21.5.42 with headache, pain in the back of the neck, joint and muscle pains, and severe pain in the right hypochondrium and epigastrium. On admission there were petechiae on the trunk, his liver was enlarged 2 f. and very tender, there was considerable epigastric tenderness, and his spleen was enlarged and soft but not tender. For some days the patient passed tarry stools and there was vomiting but no haematemesis. Headache was severe. Fever lasting two days occurred every three to four days (total of three relapses) and blood films did not become positive until the third relapse. A blood count (Table II) was done on the first day of the third relapse when he was given sulphostab, 0·3 grammes I.V. He received 0·45 grammes and 0·6 grammes at five- and seven-day intervals. Discharged fit to unit on 25.6.42. There was no further fever.

**Case 19.**—Indian, Havildar, aged 35. Sudden onset of fever on 29.7.42, with considerable malaise, chill and vomiting. Admitted to another hospital on 1.8.42 and transferred to 82 G.H. on 10.8.42. For the next sixteen days his condition remained unchanged. He ran an irregular fever, never higher than 101° F., with periods of one to three days with no rise of temperature. He felt ill during this time and complained of a pain in the left hypochondrium which had started shortly before the original fever.
There was a loose cough with purulent sputum and much recent loss of weight. There were mild signs of bronchitis and the spleen was enlarged and soft. A W.B.C. count was normal (Table II). Periodic blood films and spuita examinations were negative and an X-ray of the chest showed normal lung fields. On 26.8.42 there was a sudden rise of temperature to 103° with increased malaise, chill and considerable vomiting. He looked ill. A blood film showed spirochetes and he was given 0.6 gramme novostab, I.V. The next day he felt very much better and the fever rapidly subsided, but he complained of a persistent pain in the left knee and calf, with mild tenderness in these parts. On 3.9.42 the spleen was smaller but still mildly painful and tender. Discharged to Con. Dep. on 4.9.42. He was seen again on 25.9.42 when he had put on much weight and appeared quite fit. There was no further fever.

**Case 35.—U.K., Driver, aged 33.** He awoke on 14.4.43 “feeling like the morning after.” Later he felt weak, began to shiver, developed a very severe headache and became very thirsty. He was admitted to another hospital the same day (see chart). The next day a positive blood film was obtained and he was given 0.6 gramme sulphostab, I.V. He had a severe general headache for three days during which time he felt very ill, couldn’t sleep and had a persistent bizarre sensation “as though there were two of me.” On 18.4.43 his fundi were normal. Transferred without symptoms or signs to 82 C.H. on 22.4.43.

A single relapse occurred on 29.4.43 with malaise and headache. His head was “going round” but there was no true vertigo. Novostab, 0.6 gramme I.V. was given at noon when a blood film was negative; one hour later there were relatively numerous spirochetes in a blood film. During the night he vomited about twelve times with some griping abdominal pain. For the first time there was mild lower and moderate upper abdominal tenderness. Vomiting continued all day with severe general headache (30.4.43) and he became fairly ill. He was restless, had a flushed face, couldn’t sleep and again had the bizarre sensation previously mentioned. There was mild neck stiffness. He passed a good night after being given morphine and was very much improved the next day.

### Table II. White Blood Cell Counts in Cases Where They Were Performed

<table>
<thead>
<tr>
<th>Case</th>
<th>Date</th>
<th>Febrile Treated (F.)</th>
<th>Afebrile Untreated (A.F.)</th>
<th>Differential W.B.C. count (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>or Meta-</td>
<td>or</td>
<td>Metamyelo- Myelo- Plasma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>or</td>
<td>or</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>20.5.42</td>
<td>F. U.T.</td>
<td>8,800</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>21.5.42</td>
<td>A.F. U.T.</td>
<td>9,000</td>
<td>46</td>
</tr>
<tr>
<td>4</td>
<td>6.6.42</td>
<td>F. U.T.</td>
<td>7,000</td>
<td>73</td>
</tr>
<tr>
<td>6</td>
<td>30.5.42</td>
<td>F. U.T.</td>
<td>8,000</td>
<td>73</td>
</tr>
<tr>
<td>7</td>
<td>27.5.42</td>
<td>F. U.T.</td>
<td>7,800</td>
<td>78</td>
</tr>
<tr>
<td>9</td>
<td>2.6.42</td>
<td>F. T.</td>
<td>9,000</td>
<td>54</td>
</tr>
<tr>
<td>18</td>
<td>30.7.42</td>
<td>F. U.T.</td>
<td>8,000</td>
<td>60</td>
</tr>
<tr>
<td>19</td>
<td>13.8.42</td>
<td>A.F. U.T.</td>
<td>10,000</td>
<td>72</td>
</tr>
<tr>
<td>27</td>
<td>26.11.42</td>
<td>F. U.T.</td>
<td>9,800</td>
<td>79</td>
</tr>
<tr>
<td>33</td>
<td>16.3.43</td>
<td>A.F. T.</td>
<td>17,200</td>
<td>66</td>
</tr>
<tr>
<td>34</td>
<td>17.3.43</td>
<td>A.F. T.</td>
<td>10,000</td>
<td>60</td>
</tr>
<tr>
<td>24.3.43</td>
<td>A.F. T.</td>
<td>12,000</td>
<td>62·6</td>
<td>24·7</td>
</tr>
</tbody>
</table>

Note: The values are rounded off to the nearest unit.
Thereafter there was rapid improvement in every way. He was given novostab, 0.6 gramme I.V. on 6.5.43 and on 13.5.43. Discharged to Con. Dep. on 17.5.43 and to unit on 3.6.43. There was no further fever.

The following is a summary of the symptoms and signs recorded in the present series of 35 cases. Absolute numbers are given, with percentages in brackets. The figures and remarks in square brackets refer to the 12 Cyprus cases reported by Wood and Dixon (1945):

**General and Unclassified Symptoms and Signs**

Maximum number of relapses, 7 [4]; cases with no relapses, 2 [2]; duration of attacks, 1–8 days [1–3]; intervals between attacks, treated and untreated, 1–39 days [1–38]; headache, 22 (62.8 per cent) [11 (91.5 per cent)]; vomiting, 11 (31.4 per cent); nausea alone, 4 (11.4 per cent) [1 (8.4 per cent)]; constipation, 5 (14.3 per cent); shivering, 21 (60 per cent) [8 (66.6 per cent)]; sweating, 1 (2.9 per cent) [1 (8.4 per cent)]; feeling of coldness, 2 (5.7 per cent); malaise, 10 (28.6 per cent) [3 (25 per cent)]; anorexia (in fever), 5 (14.3 per cent) [1 (8.4 per cent)]; tiredness, 2 (5.7 per cent); weakness, 9 (25.7 per cent); insomnia, 4 (11.4 per cent); palpitation, 1 (2.9 per cent); "hangover," 1 (2.9 per cent); thirst, 1 (2.9 per cent); sense of “duality” (Case 35), 1 (2.9 per cent); giddiness, 1 (2.9 per cent) [2 (16.7 per cent)] petechiae, 2 (5.7 per cent); melena, 1 (2.9 per cent); face flushed, 2 (5.7 per cent); loss of weight, 5 (14.3 per cent); anaemia, 2 (5.7 per cent); leucocytosis, 2 (of 11 examined) (Table II); herpes simplex, 1 (2.9 per cent) [1 (8.4 per cent)]; prolonged irregular fever, 1 (2.9 per cent) [2 (16.7 per cent)]; aching behind eyes
Clinical Manifestations of Tick-borne Relapsing Fever

[2 (16.7 per cent)]; blood Kahn reaction, one doubtful in 4 tests [11 tests all negative]. In many cases succeeding relapses, if uncomplicated, tended to become shorter and less severe.

Abdominal Symptoms and Signs

Loin pain, 2 (5.7 per cent); epigastric pain, 5 (14.3 per cent); right hypochondrium pain, 4 (11.4 per cent); left hypochondrium pain, 3 (8.6 per cent); vague general abdominal pain, 4 (11.4 per cent); R.I.F. pain, 1 (2.9 per cent); “tight, uneasy” abdomen, 1 (2.9 per cent); abdominal colic, 1 (2.9 per cent); spleen enlarged, 21 (60 per cent); spleen tender, 8 (22.9 per cent); liver enlarged, 5 (14.3 per cent); liver tender, 14 (40 per cent); increased urinary urobilin or urobilinogen, 7 (11 tested) [6 (7 tested)]; epigastric tenderness, 8 (22.9 per cent); R.I.F. tenderness, 2 (5.7 per cent); umbilical tenderness, 1 (2.9 per cent); diarrhoea, 2 (5.7 per cent); mucus in stools, 2 (5.7 per cent); blood in stools, 1 (2.9 per cent); jaundice [1 (8.4 per cent)]; urine: some cases had mild albuminuria during pyrexia.

Joint and Muscle Symptoms and Signs

Backache, 11 (31.4 per cent) [3 (25 per cent)]; pain at back of neck, 7 (20 per cent); general aches and pains, 18 (51.5 per cent); stiff back, 1 (2.9 per cent); vague chest pain, 6 (17.1 per cent); joint effusions, 1 (2.9 per cent); tender muscles, 5 (14.3 per cent); tender rib, 1 (2.9 per cent); pains in shoulders [1 (8.4 per cent)]; pains in limbs [1 (8.4 per cent)].

Respiratory Tract Symptoms and Signs

Coryza, 2 (5.7 per cent); sore throat, 1 (2.9 per cent); cough, 6 (17.1 per cent); sputum, 4 (11.4 per cent); difficulty in breathing, 1 (2.9 per cent); lung rhonchi, 4 (11.4 per cent).

Neurological and Ocular Complications

Meningitis, 3 (8.6 per cent) [4 (33.3 per cent)]; facial palsy, 2 (5.7 per cent); encephalitis, 1 (2.9 per cent); papilledema, 4 (11.4 per cent) [5 (41.5 per cent)]; choroiditis [1 (8.4 per cent)]; weakness of ocular accommodation [1 (8.4 per cent)]; deterioration of vision, 1 (2.9 per cent).

Total cases with neurological complications, 6 (17.1 per cent) [4 (33 per cent)].

Symptoms and Signs Present with Neurological Complications

Headache, 4; vomiting, 1; painful eye movements, 1; drowsy or lethargic, 2; neck rigidity, 3; knee-jerks sluggish, 4; ankle-jerks sluggish, 1; arm-jerks absent, 1; abdominal reflexes sluggish, 1; restlessness, 1; abnormal c.s.f., 3 [4] (Table IV).

Other Points

Incubation period, 7–15 days [8–10]; effect of organic arsenicals in treatment, poor [poor]; period of incapacity, 13–131 days [20–111]; spirochætes in peripheral blood, nearly always scanty, never profuse.
Complicating Diseases


The commonest manifestations met with in this series were:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>62.8</td>
</tr>
<tr>
<td>Shivering</td>
<td>60.0</td>
</tr>
<tr>
<td>Splenic enlargement</td>
<td>51.5</td>
</tr>
<tr>
<td>General aches and pains</td>
<td>40.0</td>
</tr>
<tr>
<td>Liver tenderness</td>
<td>31.4</td>
</tr>
<tr>
<td>Vomiting</td>
<td>31.4</td>
</tr>
<tr>
<td>Backache</td>
<td>28.6</td>
</tr>
<tr>
<td>Malaise</td>
<td>25.7</td>
</tr>
<tr>
<td>Weakness</td>
<td>22.9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain at back of neck</td>
<td>20.0</td>
</tr>
<tr>
<td>Vague chest pain</td>
<td>17.1</td>
</tr>
<tr>
<td>Cough</td>
<td>14.3</td>
</tr>
<tr>
<td>Anorexia</td>
<td>14.3</td>
</tr>
<tr>
<td>Loss of weight</td>
<td>14.3</td>
</tr>
<tr>
<td>Constipation</td>
<td>14.3</td>
</tr>
<tr>
<td>Epigastric pain</td>
<td>14.3</td>
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<tr>
<td>Liver enlargement</td>
<td>14.3</td>
</tr>
<tr>
<td>Tender muscles</td>
<td>14.3</td>
</tr>
<tr>
<td>Increase of urinary</td>
<td>14.3</td>
</tr>
<tr>
<td>Urobilin 7 of 11 cases</td>
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</tbody>
</table>

COMPARISON OF THE CLINICAL MANIFESTATIONS OF TICK- AND LOUSE-BORNE RELAPSING FEVERS

The purpose of this discussion is partly to amplify the findings so far reported and partly to examine the possibilities of a clinical differentiation of the two forms of the disease. Few accounts of relapsing fever attempt such a formulation except on a somewhat superficial plane. This analysis makes no attempt to be comprehensive and is referred chiefly to what was found in our cases. Examination of the literature reveals that there are not a great many instances of writers giving detailed attention to the clinical aspect of the condition and all too often reliable evidence as to the vector is lacking. The need for investigation into the nature of the transmitting agent has been stressed by Coghill and Gambles (1948). For this, guinea-pig inoculation and search for the vector in the field are essential.

In only eight of the papers quoted in the following discussion were the vectors known with complete or almost complete certainty. In all the other papers consulted varying degrees of evidence are available by which it might be possible to adduce the transmitting agent. Many articles in which it is impossible to decide what the vector might have had to be ignored. For the purposes of tabulation the letters “T” and “L” are applied after the papers quoted, to signify “Tick-borne” or “Louse-borne.” Unqualified letters are used only after the eight papers noted above. Where the vector is less certainly proved, but where there is reasonable probability, “?T” and “?L” are used, and where there is greater uncertainty “??T” and “??L.” In the last two categories it is difficult to judge degrees of certainty without begging the question and only a few points such as site, season and incidence of infection, can sometimes be used for differentiation. This is far from being a satisfactory basis for an attempt to classify clinical manifestations, additionally so as some of the outbreaks recorded may have contained cases of both types. However, even a tentative effort in this direction may help to put the diseases on a firmer clinical footing.
Maximum number of relapses.—A perusal of the literature reveals what is generally appreciated, that more relapse may be expected with the tick-borne (T-B) form. More than 4 are very rarely seen with the louse-borne (L-B) type where one or two are the usual maxima. 3 or more are common if not usual with the former and the maximum recorded appears to be 20 by Kassirsky (1933) (?T). 17 are reported by Bulmer (1943) (?T), 13 by Adler et al. (1937) (T), 12 by Briggs (1935) (?T), 11 by Manson and Thornton (1919) (?T) and Lawrence and Terrell (1942) (T), 9 by Cooper (1942) (?T). In our series the maximum recorded was 7 in one patient, and for the rest one had 6, one 5, three 4, five 3, nine 2, thirteen 1 and two had none. However, all these were treated for the most part relatively early in the disease. The figures for relapses before treatment are: one had 4, two had 3, eight had 2, fourteen had 1 and ten had none.

Absence of relapses, treated or untreated, is much commoner in the L-B form. This may be a point of importance in diagnosis, especially at the onset of an epidemic.

Duration of attacks.—Untreated these are generally longer with the L-B type, varying from two to ten days in different series, and treatment appears to exert more effect in lowering the temperature. The paroxysms, treated or untreated, are usually briefer with the other form and vary from as few as three hours to five or seven days. Occasionally prolonged irregular fever is noted in T-B cases. This may last up to one month, be almost typhoid-like, remittent-intermittent or grossly irregular in which case the fever may not necessarily be high. Examples of the first are mentioned by Ordman and Jones (1940) (?T) and Marques (1944) (?T); of the second by Adler et al. (1937) (T) and Coghill et al. (1947) (?T); and of the third by Drake-Brockman (1914) (?T), Harold (1920) (?T) and Wood and Dixon (1945) (T). Our Case 19 belongs to the last group. These types of prolonged fever are an interesting manifestation and contrast with the classical fever of the disease, leading to much difficulty in diagnosis. They seldom occur in L-B relapsing fever although Chung and Chang (1939) (?L) noted irregular pyrexia sometimes in children.

Headache is a very common symptom and equally frequent in both forms of the disease.

Vomiting appears to be relatively uncommon in most reports of T-B relapsing fever in the absence of meningitis, although it occurred in 31 per cent of cases in the present series and Ross and Milne (1904) (?T) describe it as a prominent symptom and Briggs (1935) (?T) records it in 74 per cent of his 53 cases. In the other conditions vomiting is reported up to high percentages by Sinton (1921) (?L), Selwyn-Clarke et al. (1923) (?L), Beveridge (1928) (?L), Chung and Chang (1939) (?L) and Benhamou et al. (1946) (?L). Nausea alone is occasionally noted in both forms.

Constipation is recorded by some observers as a notable symptom, perhaps more common in the L-B cases. McCulloch (1925) (?L) found it in 79 per cent of 300 cases and Chung and Chang (1939) (?L) in 47 per cent of 337.
Shivering (rigor) with the onset of the paroxysm seems to be a variable symptom. Its rarity or absence is specifically noted by some authors—Browse (1912) (??T) and Newcomb (1920) (??L). It is a common symptom in many series.

Sweating of an especially profuse nature has been commented on by some authors, notably Briggs (1935) (??T) (82 per cent of 53 cases), Chung and Chang (1939) (??L) (27 per cent of 337 cases) and Cooper (1942) (??T) (62 per cent of 63 cases).

Malaise is specifically mentioned by only a few authors. However, there is no doubt that during the paroxysm many patients feel very ill.

Tiredness, weakness and debility are mentioned as being notable only in the T-B type. They are symptoms which may be related to the length of the illness.

Insomnia is a symptom not often noted. It is mentioned by Sinton (1921) (??L), Magee (1942) (??T) and Cooper (1942) (??T) (11 per cent).

Giddiness has been reported from time to time, mostly in the T-B form. It was common in the cases of Selwyn-Clarke et al. (1923) (??L) and the incidence in Cooper’s (1942) (??T) series was 24 per cent. The single complaint of giddiness recorded in the present author’s cases was in an Englishman and it was not a true vertigo. “Giddiness” is a usual complaint by Indians when ill from any cause.

Hæmorrhage.—A variety of types of bleeding, usually infrequent, are recorded in the literature, most kinds being much commoner in L-B than in T-B relapsing fever. Epistaxis is the commonest hæmorrhagic manifestation and is recorded among others by Manson and Thornton (1919) (??T) (4 in 1,500 cases), Sinton (1921) (??L) (29 per cent of 31 cases), McCulloch (1925) (??L) (17 per cent of over 300 cases), Chung and Chang (1939) (??L) (27 per cent), Ordman and Jones (1940) (??T) (frequently in 1,800 cases) and Robinson (1942) (??L) (12 per cent of 340 cases). Hæmatoxymesis is not often mentioned; Medulla (1934) (??T) had one case in a small series of 4, and Chung and Chang (1939) (??L) noted it in 0·3 per cent of their series of 337. Melæna is only rarely recorded; it occurred in 1·5 per cent of the last mentioned series but these authors occasionally noted frank red blood in the stools of other patients, a manifestation also noted by Wright and Harold (1920) (??T), Ordman and Jones (1940) (??T) and Marques (1944) (??T). Hæmoptysis is rare; Charters (1942) (??L) had one instance in 32 cases, and it was also seen by Wright and Harold (1920) (??T) and Roy (1921) (??L). Hæmorrhage from and into mucus membranes (gums, conjunctivæ, etc.) is uncommon but is noted by Manson and Thornton (1919) (??T), Sinton (1921) (??L), Ordman and Jones (1940) (??T) and Robinson (1942) (??L) (12 per cent). Petechiæ are a little more commonly reported, notably by Briggs (1935) (??T) (4 per cent), Chung and Chang (1939) (??L) (35 per cent), Robinson (1942) (??L) (8 per cent) and Cooper (1942) (??T) (8 per cent). Occasional echymoses were recorded by Lebert (1875) (??L). Speaking of T-B relapsing fever Rogers and Megaw (1944) state that the “rash may be hæmorrhagic in severe cases.” No rash other than petechiæ was observed in any of our cases (admittedly most were dark-skinned) and those with petechiae
were not severe cases. *Hæmaturia* has been recorded a few times, notably by Chung and Chang (1939) (?L) (11 per cent), but in most cases the blood is only found on microscopical examination of the urine and frank hæmaturia must be rare; it has been seen coming from the bladder by Manson and Thornton (1919) (?T). *Uterine hæmorrhage* is recorded as a rare event by Lebert (1875) (?L). *Subarachnoid hæmorrhage* was found in one case by Dewar and Walmsley (1945) (?T) and unspecified *cerebral hæmorrhage* is mentioned by Robinson (1942) (?L) and Manson and Thornton (1919) (?T). Occasionally one case may show numerous bleeding manifestations; such a patient is that of Dewar and Walmsley (1945) (?T). Benhamou et al. (1946) (?L) report many different kinds of hæmorrhage among their 3,800 cases.

Little appears to have been done to determine the cause of hæmorrhage in relapsing fever. Aggregations of spirochaetes in the capillaries was a theory put forward by Manson and Thornton (1919) (?T) and Charters (1942) (?L) to explain the focal manifestations of relapsing fever (compare malignant tertian malaria). Wail (1922) found evidence of capillary endothelium damage of a specific nature. Adler and Ashbel (1937) (T) postulate capillary damage due to spirochaetes escaping through capillary walls, a phenomenon they have observed. One author (Robinson (1942) (?L)) found that the prothrombin time was increased in all the cases tested (probably from liver damage), and that vitamin K helped to stop hæmorrhage.

*Flushing of the face*, noted in two of our cases does not appear to be a usual sign and no reference to it has been found in the papers consulted. *Pallor of the skin* was remarked on by Cooper (1942) (?T).

*Loss of weight* is a manifestation noted by few, but is commented on by the following, some of whom remark on it as a striking effect of the disease: Harold (1920) (?T), Beveridge (1928) (?L), Medulla (1934 and 1935) (?T) (3 of 7 cases), Cooper (1942) (?T) (28.5 per cent) and Magee (1942) (?T).

*Anaemia* appears to be rare, and almost never severe. Most references to it concern cases which are possibly or probably T-B; this may be fortuitous, but if not is probably due to the greater length of this illness. Anaemia is mentioned as occurring sometimes by Lebert (1875) (?L), McCulloch (1925) (?L) and Briggs (1935) (?T); as occurring occasionally by Browse (1912) (?T), Adler et al. (1937) (T) and Chung and Chang (1939) (?L). Calwell (1920) (?T) states that he found anaemia frequently in cases in Palestine.

*Leucocytosis* is recorded by some authors, usually during the fever. On the whole it seems commoner in the L-B than the T-B form. Sawtschenko and Melkich (1901) (?L), Manson and Thornton (1919) (?T); Sachs (1934) (?T), Briggs (1935) (?T) and Adler et al. (1937) (T) found it in some of their cases, usually slight. Calwell (1920) (?T), Sinton (1921) (?L), McCulloch (1925) (?L) and Lawrence and Terrell (1942) (T) report it as fairly common, while Chung and Chang (1939) (?L) found it in 60 per cent of their series. Respiratory complications, if marked, cause an increased leucocytosis according to Manson and Thornton, 1919 (?T) and Chung and Chang (1939) (?L). In our series the blood leucocyte counts showed nothing very unusual. Two had a moderate monocytosis which is considered a fairly common finding in re-
N. F. Coghill

Relapsing fever. (Manson and Thornton, 1919 (?T); Calwell, 1920 (?T); Sinton, 1921 (?L); McCulloch, 1925 (?L); Lawrence and Terrell, 1942 (T).

Herpes simplex (usually labialis) is not often remarked on in the literature and seems less common than in malaria or pneumonia. It occurred in 3.9 per cent of Chung and Chang's (1939) (?L) cases, was recorded once by Drake-Brockman (1914) (?T) and occasionally by Dutton and Todd (1905b) (?T) and Sergent (1938) (T). However Briggs (1935) (?T) noted it in 14 per cent of 53 cases. It seems more common in T-B than in L-B relapsing fever.

Succeeding relapses have been found by some to become milder and shorter. (Briggs, 1935 (?T) and Magee, 1942 (?T)). This was also recorded by Kemp et al. (1935) (?T) in some cases although in others the earlier succeeding relapses became more severe.

Sense of Duality.—Our patient (Case 35) had the bizarre feeling that there were “two of him” during the initial attack and again in the single relapse he sustained. The patient was an intelligent witness but found difficulty in adequate description of the symptom. Marques (1944) (?T), among others, has noted what he terms “complex psyche phenomena.”

Abdominal Symptoms and Signs

We found abdominal pain and especially tenderness fairly commonly in one situation or another, particularly over liver and spleen. Many others have recorded somewhat similar findings.

Pain or pressure in the epigastrium was noted by Beveridge (1928) (?L) occasionally, by Medulla (1934) (?T) in 3 or 4 cases, by Burns (1936) (?T) in 1 case, by Adler et al. (1937) (T) in 1 of 45 cases, by Chung and Chang (1939) (?L) in 9.5 per cent of cases, frequently by Magee (1942) (?T) and often by Benhamou et al. (1946) (?L).

Splenic pain has been reported as an occasional occurrence by Manson and Thornton (1919) (?T), Wright and Harold (1920) (?T) and Sinton (1921) (?L); in 3 of 45 cases by Bergsma (1929) (?L); and as a fairly frequent manifestation by Ordman and Jones (1940) (?T).

Hepatic pain was recorded relatively often by Lebert (1875) (?L), Manson and Thornton (1919) (?T), Calwell (1920) (?T) and Sinton (1921) (?L).

Loin pain was noted by Cooper (1942) (?T) in 11 per cent of cases.

General abdominal pain has been reported by Lebert (1875) (?L) and Marques (1944) (?T), in 19 per cent of cases by Chung and Chang (1939) (?L), in 33 per cent of cases by Cooper (1942) (?T) and by Dewar and Walmsley (1945) (?T) in their single case.

Abdominal rigidity was noted sometimes by Charters (1942) (?L). In our Case 18 where abdominal pain was conspicuous there was no increase in abdominal wall resistance.

Epigastric Tenderness.—In our cases this may sometimes have been no more than an extension of the liver tenderness; in only one was it limited to the epigastrium. This sign was occasionally found in the series of Beveridge (1928) (?L).
General abdominal tenderness has been reported by Roy (1921) (?L), Sergent et al. (1933) (T) and Cooper (1942) (?T) (11 per cent of cases).

Iliac fossa tenderness was noted in one case by Coghill et al. (1947) (?T). Although different series vary greatly in the incidence of the above symptoms and signs it seems likely that they occur about as frequently in L-B as in T-B relapsing fever, and they are sometimes common. Delamare (1930) has drawn particular attention to these features of the disease but unfortunately does not state to which type he is referring. He describes a patient rather similar to our Case 18, who presented as a surgical emergency and whose abdomen was opened for appendicectomy.

These abdominal manifestations are mentioned, but not enlarged upon, by Rogers and Megaw (1944), Manson-Bahr (1945), Strong (1945) and Fairley (1946).

Splenic Tenderness.—This is recorded by many authors and is a common finding, although its incidence appears to vary widely. It was sometimes found by Kemp et al. (1935) (?T), often by Drake-Brockman (1914) (?T), Sinton (1921) (?L) and Ordman and Jones (1940) (?T), and in 3 of 18 cases by Browne (1912) (?T), in 13 per cent of cases by McCulloch (1925) (?L), in 20 per cent by Bergsma (1929) (?L), in 6 of 337 by Chung and Chang (1939) (?L), in 41 per cent of 32 by Charters (1942) (?L), in 17 per cent by Cooper (1942) (?T) and in 44 per cent by Robinson (1942) (?L).

Splenic Enlargement.—This is reported by many; good examples are Lebert (1875) (?L) (often), Dutton and Todd (1905a) (?T) (fairly often), Newcombe (1920) (?L) and Sinton (1921) (?L) (each 30 per cent of cases), Mackie (1927) (?T) (often), Beveridge (1928) (?L) (52 per cent), Briggs (1935) (?T) (33 per cent), Chung and Chang (1939) (?L) (69 per cent), Cooper (1942) (?T) (30 per cent), Robinson (1942) (?L) (89 per cent), Charters (1942) (?L) (59.5 per cent), Bulmer (1943) (?T) (usual) and Marques (1944) (?T) (71 per cent). Considerable variation is seen in different series of cases. Marzinowsky (1927) comparing European (probably L-B) relapsing fever with Persian (presumably T-B) stated that the spleen is enlarged and easily palpable in the former but very hard and not always felt in the latter.

Among our own cases 60 per cent had mildly enlarged spleens; of these all but two were Indians and one of the remainder was a Cypriot. Splenic enlargement due to chronic malaria, however, is not as common as this among Indian adults, and under observation many of these spleens were softer than would have been expected if they were malarial, and the enlargement frequently diminished or disappeared along with the tenderness, in convalescence.

Perisplenitis and splenic infarcts are mentioned by Ordman and Jones (1940) (?T), Robinson (1942) (?L) and Lebert (1875) (?L) (infarcts only) as uncommon findings.

Liver tenderness has been noted among others by Lebert (1875) (?L) (often), Drake-Brockman (1914) (?T) (often), Manson and Thornton (1919) (?T) (sometimes), Sinton (1921) (?L) (often), Beveridge (1928) (?L) (2 per cent of cases), Katz (1930) (?T) (all of 38 cases), Chung and Chang (1939) (?L) (several cases), Cooper (1942) (?T) (79 per cent) and Charters (1942) (?L) (72 per cent).
Liver enlargement has been recorded by many; chief examples are: Lebert (1875) (?L) (often), Drake-Brockman (1914) (?T) (often), Manson and Thornton (1919) (?T) (often), Sinton (1921) (?L) (93.5 per cent), Beveridge (1928) (?L) (44 per cent), Kemp et al. (1935) (?T) (sometimes), Briggs (1935) (?T) (14 per cent), Chung and Chang (1939) (?L) (41 per cent), Ordman and Jones (1940) (?T) (sometimes), Charters (1942) (?L) (72 per cent), Cooper (1942) (?T) (13 per cent) and Lawrence and Terrell (1942) (T) (sometimes). Others have noted that as with the spleen the enlargement may diminish during the apyrexial periods (Benhamou et al., 1946 (?L)).

Jaundice is an important manifestation which is fairly common. It has been reported principally by Manson and Thornton (1919) (?T) (sometimes), Calwell (1920) (?T) (slight in 25 per cent, severe in one), Wright and Harold (1920) (?T) (up to 5 per cent), Sinton (1921) (?L) (48 per cent), McCulloch (1925) (?L) (about 80 per cent), Beveridge (1928) (?L) (20 per cent), Briggs (1935) (?T) (18 per cent), Chung and Chang (1939) (?L) (29 per cent), Ordman and Jones (1940) (?T) (sometimes), Robinson (1942) (?L) (72 per cent), Charters (1942) (?L) (16 per cent), Cooper (1942) (?T) (10 per cent) and Benhamou et al. (1946) (?L) (about two-thirds of 3,800 cases).

Taking the hepatic and splenic signs together it appears probable that liver tenderness, enlargement and jaundice are rather commoner in L-B than T-B relapsing fever, but that splenic tenderness and enlargement are about equal in the two forms. However, the work of Russell (1932) in the Gold Coast on what must have been L-B relapsing fever, and evidence she brings forward from the literature, makes it probable that splenic lesions in this type may be more severe than those usually found in the T-B form. As long ago as 1875 Lebert (?L), quoting others, described severe changes in the spleen similar to those found by Russell. There is less frequent opportunity to examine the spleens post-mortem in T-B relapsing fever with its lower mortality so that material is not as plentiful in this form. However Manson and Thornton (1919) (?T) did find almost completely necrotic spleens at autopsy. This compares with Ordman and Jones (1940) (?T) who described only a surface fibrinous splenic exudate, and Dewar and Walmsley (1945) (?T) who discovered little evidence of disease in the spleen of their single mortal case.

In our own series of cases liver enlargement was found in 14 per cent. This agrees well with the incidence of this sign usually found in T-B relapsing fever and contrasts with that in the L-B type where it is often much higher. None of our cases became jaundiced although from the testimony of Mr. Mogabgab (quoted by Gambles and Coghill, 1948) relapsing fever in Cyprus, if untreated, may give rise to this manifestation, and one of Wood and Dixon's (1945) (T) cases developed slight icterus.

Diarrhoea is sometimes a frequent finding but is often rare or absent. When present it often occurs with the crisis. It was noted by Ross and Milne (1904) (?T) and is otherwise mentioned chiefly by Calwell (1920) (?T) (occasionally), McCulloch (1925) (?L) (21 per cent), Beveridge (1928) (?L) (47 per cent), Chung and Chang (1939) (?L) (14 per cent) and Cooper (1942) (?T) (11 per cent). It appears to be commoner in the L-B type. The presence of excess mucus in
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The stools has been reported once by Adler et al. (1937) (T) and as occurring sometimes by Ordman and Jones (1940) (?T) and Marques (1944) (?T).

**Urinary Urobilin (or Urobilinogen).—**Coskinas (1914) (?L) was the first to describe in relapsing fever an increase in the urinary urobilin, without the presence of bile pigments or salts. Lafforgue (1914) (?T) confirmed this finding in cases in Tunis. In some of our cases the urine was tested for an increase in urobilin and/or urobilinogen when Dixon (1943) (T) communicated his results to us.

The test used for urobilin was Schlesinger's, with alcoholic zinc acetate; that for urobilinogen was with Ehrlich's reagent (p-dimethylaminobenzaldehyde). In all cases the urine was obtained during the late morning or in the afternoon, and was tested immediately after voiding in the case of urobilinogen, or after standing some hours in daylight or sunlight for urobilin. In the Ehrlich test the urine was diluted one in ten before addition of the reagent. This was found to be the best test. Used according to the method of Stitt et al. (1938) it is capable of a greater degree of quantitative accuracy than is Schlesinger's test. The greater number of negatives among our cases compared with those of Wood and Dixon (1945) (T) and Dixon (1943) (T) is accounted for by the fact that the first cases we tested were at a late stage in their illness. The urinary increase of urobilin may not occur with each attack as Dixon found. It may occur during an apyrexial period; but this is so uncommon that the test cannot be used to forecast a relapse.

The presence of urobilin in the urine in greater amounts than normal was presumed by Dixon (1943) (T) to indicate some degree of liver damage. Although in the occasional case there seemed to be some relation between the presence and degree of hepatic tenderness and enlargement, and the quantities of urobilin in the urine, generally speaking no constant correlation could be made out (see Temperature Charts). It must not be forgotten that an increase in urinary urobilin is common during fevers and may be considerable although short-lived in conditions such as sandfly fever and especially malaria. In these, however, the increase is related to the fever. The fact that in relapsing fever it may (rarely) not be so might be taken as additional evidence of liver damage by the spirochætes.

Although such manifestations as hepatic pain, tenderness and enlargement, jaundice and urobilinuria point to the presence of liver damage in this disease there are not often convincing records of its presence, degree or nature. Manson and Thornton (1919) (?T) speak of the liver at post-mortem as considerably enlarged, soft and showing "a marked degree of early toxic change." Kennedy (1920) describes the findings in two fatal cases (no evidence as to vector). He found, among other things, great destruction of the liver cells, more in the central part of the lobule than the periphery. Ordman and Jones (1940) (?T) make no mention of the liver in discussing their autopsy material. In the well-described severe case of Dewar and Walmsley (1945) (?T) histological evidence of liver damage was slight.

Using these tests for urobilin and urobilinogen in urine there was no evidence that in our cases any liver damage arose from the administration of arsenicals.
Joint and Muscle Symptoms and Signs

Backache and back pains may be prominent symptoms which are recorded by a number of writers, including Lebert (1875) (?L), Drake-Brockman (1914) (?T), Manson and Thornton (1919) (?T), McCulloch (1925) (?L) (45 per cent), Kemp et al. (1935) (?T), Davis et al. (1941) (T), Cooper (1942) (?T) (44·5 per cent) and Magee (1942) (?T).

Chest pain is mentioned by Manson and Thornton (1919) (?T), Sinton (1921) (?L), Chung and Chang (1939) (?L) (5 per cent) and Cooper (1942) (?T) (11 per cent).

Painful and tender muscles were sometimes noted by Lebert (1875) (?L), Drake-Brockman (1914) (?T), Roy (1921) (?L) and Kassirsky (1933) (?T). In our cases the sternomastoids were most often affected. The tenderness was sometimes considerable and lasted continuously for several weeks. The pectoral muscles were affected in one.

Joint pains are less common than limb or body pains but have been noted with fair frequency in some series. Joint effusions were seen in one of our cases (knees) who, however, gave a history of previous attacks of rheumatic fever and who had mitral stenosis. In this instance it was difficult to be certain of the aetiology of the arthritis. Roy (1921) (?L) and Dixon (1943) (T) each describe a case with joint effusions apparently due to relapsing fever.

All the manifestations in this category have probably much the same incidence in both forms of relapsing fever.

Respiratory Tract Symptoms and Signs

It is possible that these would be better termed "complications." In our own series it is difficult to know how much attention should be paid to them. The lung signs were not a striking feature as they have been in some series and were usually no more than are commonly met with in sandfly fever, malaria and typhoid, during the febrile period. Pulmonary complications such as dyspnœa, lobar or bronchopneumonia, and pleurisy seem relatively unusual and to be found equally with the L–B or T–B forms. However, a cough, presumably indicative of a mild acute bronchitis, has been found in considerable numbers in some series. Calwell (1920) (?L) noted it in 14·5 per cent of 69 cases in Egypt, Wright and Harold (1920) (?T) in 68 per cent, Roy (1921) (?L) in 50 per cent, Chung and Chang (1939) (?L) in 41 per cent and Cooper (1942) (?T) in 21 per cent. The incidence of lobar pneumonia has been of the order of 3 in 337 cases of Chung and Chang (1939) (?L) and 4 in 63 cases of Cooper (1942) (?T); and bronchopneumonia was seen in many cases of Lagrange (1927) (?L) and in 5 per cent of cases of Chung and Chang (1939) (?L). Dyspnœa is sometimes a notable feature and may be severe.

Neurological Complications

These, of which there is a considerable variety, are nearly always met with much more commonly in the T–B disease. Their incidence in any unselected group of cases is usually low. As Scott (1944) (?T) points out, however, certain localities may give rise to greater numbers, for example about 20 per cent in
cases from the Egyptian western desert and around Tobruk. Taking our cases with those of Wood and Dixon (1945) (T) there were 10 with neurological complications among a total of 47 which gives an incidence in Cyprus of 21 per cent. This figure is probably unwarrantably high through partial selection. They may appear at any time in the course of the illness up to eight weeks or more from the onset (Cooper, 1942 (?T) and Scott, 1944 (?T)) and are often late (Manson and Thornton, 1919 (?T)). Of the two commonest manifestations meningitis normally appears earlier than cranial nerve lesions.

Meningitis.—Early accounts of this complication are given by Coskinas (1914) and Ardin-Delteil et al. (1914), the former possibly L-B and the latter possibly T-B. It was seen in a “fair proportion” of cases by Bulmer (1943) (?T), in one case by Chung and Chang (1939) (?L), in at least 3 of about 40 cases by Hawking (1941) (?T), in 5 out of 5 patients by Sautet (1941) (T), in one by Magee (1942) (?T), in 7 of 63 by Cooper (1942) (?T), in a few cases by Benhamou et al. (1946) (?L), and in 2 of 4 by Coghill et al. (1947) (?T). Collected cases are reported by Scott (1944) (?T) and McAlpine (1946) (?T). “Pachymeningitis” is recorded by Lebert (1875) (?L) in a few cases, and by Robinson (1942) (?L) in 31 of 340 cases. Scott (1944) (?T) and others (Consultant in Tropical Medicine, M.E.F., 1943a (?T)) found meningitis might recur persistently. Such a course of the illness, although by no means infrequent is not a general rule in the Middle East or elsewhere as has been suggested by McAlpine (1946) (?T).

There is nearly always a leucocytic pleocytosis in the c.s.f. which, however, seldom rises above 2,000 per c.mm., the majority of the cells being lymphocytes. Hawking (1941) (?T) found a mild increase in W.B.C. in the c.s.f. of 2 cases who had no clinical signs of meningitis and in 1 of whom spirochaetes were demonstrated in the c.s.f. Chung (1938) (?L) examined the c.s.f. of 26 patients and found a mild increase in cells (mostly lymphocytes) in some, but did not state if there was any clinical evidence of meningitis in these cases. The c.s.f. pressure is often increased and the protein variably so. The c.s.f. Wassermann reaction is sometimes positive as reported by Chung (1938) (?L) and others (Consultant Neurologist, M.E.F., 1942 (?T)).

Spirochaetes have been found in the c.s.f. from time to time, either by microscopy or animal inoculation. Such findings have been described by Soulé (1907) (?T-T), Hawking (1941) (?T) and Chung (1938) (?L) who quotes a number of similar instances from the literature. The Consultant in Tropical Medicine, M.E.F. (1943a) (?T) recorded a case where spirochaetes were found by guinea-pig inoculation two months after the completion of very adequate treatment; and the Consultant Neurologist, M.E.F. (1942) (?T) a case where they were found in the c.s.f. although they were never seen in the peripheral blood.

The signs of meningitis recorded in the literature, apart from abnormal cerebrospinal fluid, are photophobia, headache, vomiting, neck rigidity and, much less commonly, a positive Kernig’s sign. It is believed that headache may constitute the only sign of meningitis (“Notes on Nervous Diseases, etc., M.E.F.”, 1943 (?T)).

In two of the three cases here reported there was recurrence or exacerbation of the meningitis. In Case 18 the pressure was very high (over 400 mm. c.s.f.)
and in this patient papilloedema was marked and there was severe vomiting. One patient (Case 25) had clinical evidence of meningitis (severe headache, moderate neck stiffness, mild unilateral papilloedema) but a completely normal c.s.f. In two there was diminution in the strength, or absence, of the tendon reflexes during the course of the illness.

In view of what has been written about the neurotropism and multiplicity of different strains of spirochaetes it is interesting to record that our Case 18 and Wood and Dixon's (1945) (T) Case 10, both of whom had meningitis, were infected at the same site.

Cranial nerve palsies are the next most common neurological complication, and occur chiefly in the T-B form. They have been recorded among others by Manson and Thornton (1919) (?T) ("very common"), Beveridge (1928) (?L) (2 per cent), Kemp et al. (1935) (?T) (2 in 258 cases), Medulla (1935) (?T) (2 in 3 cases), Ordman and Jones (1940) (?T) (few cases), Consultant in Tropical Medicine, M.E.F. (1942a and b and 1943b) (?T) (several cases), Cooper (1942) (?T) (7 in 63 cases) and Scott (1944) (?T) (collected cases). The most commonly affected nerve is the VIIth, usually unilaterally. Ptosis and diplopia are reported by Manson and Thornton (1919) (?T), McCulloch (1925) (?L) (rare) and Scott (1944) (?T) (N.VI).Magee (1942) (?T) quotes a personal communication from Davis that permanent damage may result to the optic and auditory nerves, Rogers and Megaw (1944) state that facial paralysis may be permanent. In our 2 cases of facial palsy (lower motor neurone type) the onset was thirty-seven and seventy-four days respectively after the commencement of the relapsing fever. In one (Case 32) the c.s.f. thirteen days after the onset of the complication contained 20 W.B.C. per c.mm., but no other abnormality. One case recovered rapidly, perhaps hastened by treatment, the other showed no improvement over a period of observation of fifty-three days.

Neuritis (true peripheral), with pain, has been noted in both forms of the disease, but more often in the T-B variety, by Adler et al. (1937) (T), Magee (1942) (?T), Charters (1942) (?L), Cooper (1942) (?T) and Marques (1944) (?T).

Other, rare, neurological manifestations include hemiparesis and monoplegias (Ordman and Jones, 1940, and Scott, 1944); stupor (Lebert, 1875) (?L), Bergsma, 1929 (?L) and Coghill et al. (1947) (?T); convulsions (Chung and Chang, 1939 (?L)); epi leptiform state (Marques, 1944 (?T)); delirium and psychosis, mostly in the L-B form; aphasia (Manson and Thornton, 1919 (?T)); aphonia (McCulloch; 1925 (?L)); loss of sphincter control (Ordman and Jones, 1940 (?T), and Magee, 1942 (?T)); sensory changes (Cooper, 1942 (?T)); depression (Dutton and Todd, 1905a (?T)); radiculitis (Magee, 1942 (?T)); hiccup, usually uncommon, but seen in about 9 per cent of cases by McCulloch (1925) (?L); restlessness and irritability (Lebert, 1875 (?L), and Scott, 1944 (?T)); cerebellar signs have been noted from time to time and were recorded recently by the Consultant in Tropical Medicine, M.E.F. (1943b) (T) quoting a case reported by Major Mann from Cyprus; encephalitis is rarely described but was noted by the Consultant in Tropical Medicine, M.E.F. (1942b) (?T) in the last war and our Case 34 appears to be an example of this complication.
McAlpine (1946) (?)T provides a useful brief summary of the neurological findings in this disease in the Middle East during the last war. His 84 collected cases include some of ours. "Notes on Nervous Diseases, etc., M.E.F." (1943) (?)T gives a good description of these complications as they occurred in the same theatre. They are discussed under the headings: (a) Meningitis; (b) Facial palsy; (c) Meningo-encephalitis, acute and chronic; (d) Other Neurological Complications—(i) Cranial nerve palsies, (ii) Spinal cord and peripheral nerve lesions, (iii) Ocular signs; (e) C.S.F. Changes. The diagnosis and treatment are also discussed.

Ocular Complications

There is a variety of these and they are most commonly found in T-B form. Papilloedema occurs usually, but not necessarily with meningitis. In one of our patients (Case 25) there was mild unilateral papilloedema although the c.s.f. was completely normal; there was, however, clinical evidence of meningitis. This manifestation is not often recorded. It is mentioned once each by Adler et al. (1937) (T) and Coghill et al. (1947) (?)T and McAlpine (1946) (?)T states it was found in 11 of his 84 cases. Its occurrence was recorded by the Consultant in Tropical Medicine, M.E.F., in 1942b, and 1943a (?)T, and by Wood and Dixon (1945).

Iridocyclitis is sometimes seen. Elliott (1920) remarks that it has been commonly noticed in the T-B disease. Others who have recorded it have been Adler et al. (1937) (T): Consultant in Tropical Medicine, M.E.F. (1943b) who quotes 2 cases reported by Lt.-Col. E. Jones from Tunisia, both probably T-B; Hamilton (1943) (?)T; Marques (1944) (?)T; and Benhamou et al. (1946) (?)L.

The prognosis of this complication is said to be good by Adler et al. (1937) (T) and Hamilton (1943) (?)T. In our case of chronic iridocyclitis with little sign of improvement over many years it was the opinion of the ophthalmologist, Dr. J. G. Shelley, that the original cause was not relapsing fever although the latter might have caused an exacerbation of the condition.

Iritis is less rare. Elliott (1920) without reference to type states that it has been “observed with considerable frequency.” Drake-Brockman (1914) (?)T reports the condition in 4 cases, Manson and Thornton (1919) (?)T in 3 per cent of 1,500, McCulloch (1925) (?)L in 5 of about 300 cases and Mackie (1927) (?)T found it in 1 case.

Retinitis and choroiditis are rare (Lebert, 1875 (?)L); (Marques, 1944 (?)T and Benhamou et al., 1946 (?)L). Conjunctivitis is rare but suffused sclerae are not uncommon in some series. for example Chung and Chang (1939) (?)L noted it in 47 per cent. of their cases, and it has been recorded by Manson and Thornton (1919) (?)T, Sinton (1921) (?)L and Cooper (1942) (?)T.

Deterioration of vision was noted as a temporary phenomenon in our Case 18 who had severe papilloedema. This symptom is recorded once by Adler et al. (1937) (T).

Aural Complications

These are unusual. Lebert (1875) (?)L described tinnitus as an occasional finding, and Chung and Chang (1939) (?)L record this symptom with deafness
in 14 per cent of their cases. Deafness is also mentioned by Manson and Thornton (1919) (??T). These manifestations appear commoner in the L-B form of relapsing fever.

Some Other Features of Relapsing Fever

Allergic reactions at the sites of the bites are recorded by Adler et al. (1937) (T) and Cooper (1942) (??T) in some cases.

Sudden Onset.—Most are agreed that this is the usual manner in which both types of relapsing fever start, but some describe premonitory symptoms.

Blood Wassermann Reaction (W.R.).—This has been investigated by a few workers. Manson and Thornton (1919) (??T) found no positive W.R. in a series of cases. Fairley and Sullivan (1919) (??T) noted a few positive reactions among cases of relapsing fever. Roof (1922) (??L) examined 18 cases of whom 11 gave a positive reaction at some stage of the disease. Pai (1937) (??L) found about half of 13 cases with a positive W.R. No convincing evidence is produced, however, that some of these cases did not have syphilis. Chung (1938) (??L) reported negative W.R.s with positive C.S.F. reactions. However, this author quotes other work with a collaborator in which it was shown that blood W.R.s may be positive in Chinese relapsing fever. Murrell (1939) (T) records a boy of 7 years infected with Sp. novyi whose blood gave a positive W.R. Pai (1937) (??L) produces evidence that the blood W.R. is more likely to be positive than the Kahn in relapsing fever. However, Dixon (1943) (T) showed that the latter may be positive in this disease in Cyprus. In all cases the serological reactions are usually positive only in the febrile stages, but this is not invariable. The reactions soon become permanently negative with cure of the disease. This subject is well reviewed by Strong (1945).

Variability of the Symptoms and Signs.—This is a notable feature of relapsing fever, perhaps more especially of the T-B form: (1) in the same case in different paroxysms, particularly remarked on by Adler et al. (1937) (T); (2) from case to case in the same series, especially noted by the last author and by Cooper (1942) (??T) and Magee (1942) (??T) and (3) from outbreak to outbreak. This variability is evidently due to the spirochete affecting different organs predominantly in different cases.

Death.—Mortality varies much in different reported series, from nil up to 50 per cent. or more. It rarely exceeds 10 per cent. in the T-B form and is usually much lower. In the L-B type it is apt to be much higher, although even here it may sometimes be nil. Complications (chiefly respiratory) often account for most of the deaths (Chung and Chang 1939, (??L)). Mortality is influenced in both types by such factors as age, nutritional state and previous health. Nicolle (1932) remarks on the “extreme benignity” of T-B relapsing fever in French N. Africa. No deaths occurred among the 98 military cases of the disease known to have arisen in Cyprus between August, 1941 and April, 1943, and reported by Gambles and Coghill (1948), nor among any of the civilian cases mentioned by the same authors.

Incubation Period.—In most of our cases it was impossible to make even an approximate estimate of the incubation period. In three it was seven days or
less, in two nine or less, and in one each eleven, fourteen or fifteen days or less. In one there was proof that it was ten days. In two other cases not in this series it was less than eleven and fifteen days respectively. In the literature figures for L-B relapsing fever are scanty but do not seem to differ from those for T-B. The average incubation period appears to be about seven to twelve days with extremes of four to sixteen which are seldom exceeded.

**Effect of Organic Arsenicals.**—This will be discussed in a future publication (Coghill, in preparation).

**Numbers of Spirochætes in the Peripheral Blood.**—There is little doubt that in most cases of T-B relapsing fever spirochætes are scanty in the peripheral blood at all stages and may be extremely difficult to find. Conversely in the L-B form these organisms are usually very numerous and may be present in enormous numbers. It is possible that the numbers of circulating spirochætes bear some relation to the kind and degree of manifestations in both forms of the disease. Spirochætes may sometimes be found in the blood during afebrile periods, probably a commoner finding in the L-B form. Robinson (1942) (?L) recorded this phenomenon in 38 per cent. of cases. Cooper (1942) (?T), however, also noted it in 35 per cent. In the present cases it was often difficult to find spirochætes in blood films taken during the febrile stage and they were always in relatively small or scanty numbers.

**Period in Hospital, etc.**—The “Period of incapacity” is a not altogether satisfactory term which represents in this case either the period the patient was in hospital and convalescent depot, or the duration of his disease. After treatment there was sometimes long periods between relapses. During such asymptomatic periods patients may have been back at their units and able to work; these periods are included in the figures given under “incapacity.” 11 were “incapacitated” between thirteen and thirty days, 5 between thirty-one and fifty, 4 between fifty-one and sixty, 3 between sixty-one and seventy, 4 between seventy-one and eighty, 3 between eighty-one and ninety, 1 between ninety-one and one hundred, 1 between 101 and 110, 2 between 111 and 120 and one for 131 days. 15 (not quite half the cases) were therefore “incapacitated” for over sixty days (two months). Other writers record similar prolonged periods of “incapacity” which are usually longer in the T-B form. Cooper (1942) (?T) records such periods up to twenty-six weeks, and the patients reported by Sautet (1941) (T) were ill for five months. Although in T-B relapsing fever numbers are often small in relation to the total population at risk, this aspect is of some importance from the military point of view and stresses the need for improved treatment.

**Complicating Diseases**

Five cases of malaria (all Indian) among a series such as this would appear to be rather excessive. Others have suggested that there may be a relation between the two diseases, and that relapsing fever interferes in some way with the body’s premunity mechanisms, so favouring a relapse of the malaria in a patient who is chronically infected as are many Indian other ranks. There is little evidence to agree with the suggestion of Hamilton (1943) (?T) that malaria exerts any beneficial effect on relapsing fever, as it does on a tertiary stage of syphilis (G.P.I.).
CONCLUSIONS

The main difficulties in the clinical diagnosis of the relapsing fevers are:

1. The symptomatology in any given case in the first attack may not be unlike that of other quite different conditions.
2. There may be very few symptoms and signs in many cases.
3. There are, perhaps not unnaturally, considerable similarities between the two forms of the disease.
4. There is a lack of clinical uniformity.

Examination of a blood film is undoubtedly the best method for arriving at an early non-specific diagnosis of relapsing fever. The further clinical differentiation of relapsing fever into its two types presents greater difficulties. Those who have seen both at different times, such as Boyd (1919), Calwell (1920), Mackenzie (1920) and Woodcock (1920) remark on the clinical differences of the two conditions. Cunningham (1925) states: “There is no doubt that two definite clinical varieties of the disease exist, the louse-borne and the tick-borne.”

Nicolle (1932) makes an attempt at differentiation on clinical and other grounds. Rogers and Megaw (1944) also make an effort in this direction, but it is doubtful if some of the special features they attribute to L-B relapsing fever are in fact so specific to this form. They mention heart changes. These have been reported in accounts of cases of both kinds but evidence adduced that the heart is actually affected is nearly always scanty and unconvincing.

As a result of the present analysis of the literature it may be said that the following manifestations are much more common in T-B than L-B relapsing fever: Two or more relapses (untreated): spirochætes scanty in the peripheral blood; relatively short paroxysms of not more than five to seven days; prolonged irregular fever; vomiting chiefly with meningitis only; lethargy and weakness; giddiness (probably not true vertigo); loss of weight; debility; neurological complications of all kinds of which the commonest are lymphocytic meningitis and cranial nerve palsies; ocular complications, chiefly papilloœdema (mostly with meningitis) and iridocyclitis; allergic reactions at the sites of tick bites; poor therapeutic effect of organic arsenicals; illness relatively less severe; mortality usually very low (10 per cent or much less).

The following manifestations may be commoner in T-B than L-B relapsing fever: Variability of the symptoms and signs in the same case from paroxysm to paroxysm, and from case to case; mild anaemia; monocytosis; herpes simplex; iritis.

The following are much more common in L-B than T-B relapsing fever: Less than two relapses, often none; spirochætes numerous in the peripheral blood; relatively long paroxysms, maybe up to ten days; vomiting at any stage; hemorrhagic manifestations of all kinds; diarrhœa; jaundice, especially if severe; very fair therapeutic effect of organic arsenicals; illness relatively more severe; mortality often high (up to 50 per cent, sometimes higher).

The following may be commoner in L-B than T-B relapsing fever: Constipation; thirst; leucocytosis; hepatic tenderness and enlargement; aural complications, chiefly tinnitus and deafness.

These lists cannot, for the most part, be said to indicate striking differences
between the two forms of the disease. Some of the points are of feeble diagnostic value either because they are rare in any relapsing fever or common in other diseases. However the differences noted, such as they are, can be considered worth drawing attention to as a preliminary attempt to define the two conditions clinically and may perhaps stimulate further observation. It is interesting to speculate, for example, if there is any real basis for describing all relapsing fevers (L-B or T-B) in terms of the geographical location of the disease. What is particularly needed is careful differentiation of the spirochæte (T-B or L-B) in all types of clinical case and in different localities.

SUMMARY

(1) Representative histories are given from among 35 collected military cases infected in Cyprus. Various clinical aspects are commented upon and examples of "atypical" cases are reported.

(2) The symptoms and signs found in this series of cases are summarized.

(3) In Cyprus the chief complications were neurological and appeared in 21 per cent of cases. The present series included 3 cases of meningitis, 2 of facial palsy, 1 of encephalitis and 4 of papilledema.

(4) In nearly half the patients the disease lasted over two months.

(5) From an analysis of the manifestations in the present series and those recorded in the literature an attempt is made to differentiate tick-borne from louse-borne relapsing fever on clinical grounds. Lists of symptoms and signs are compiled which, from reports, seem commoner in one or other condition. Only partial success is achieved in this attempted differentiation. One of the greatest difficulties is the lack of evidence in most publications as to the vector. This point is stressed and a plea made that in future more attention be paid to this point so that clinical differentiation may be facilitated.

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