Electrocardiographic Changes in Leptospirosis

MALCOLM PARSONS,* M.A., M.R.C.P.

The finding of electrocardiographic (E.C.G.) abnormalities in patients with leptospirosis who show clinical evidence of cardiac involvement is well recognized. In a review of the subject Sodeman and Killough (1951) reported the unexpected discovery of abnormalities in three patients without such clinical signs, and expressed the opinion that “an active interest in the [cardiovascular] system is necessary to establish involvement.” The fact that an apparently normal E.C.G. taken in the first week of the illness might give such evidence if compared with a later record supported their thesis. Moreover, Mackay-Dick and Robinson (1957) subsequently demonstrated transient minor changes in the QRS complex in two out of six consecutive patients with uncomplicated leptospirosis.

Apart from this small series, no systematic search for latent cardiac involvement in leptospirosis has been made. In an attempt to remedy this, the present paper deals only with patients in whom there was no clinical evidence of heart disease. A comparable group of patients with malaria was also studied to find whether the changes observed were specific or associated with the response to fever.

Patients Investigated and Methods

This paper concerns 25 soldiers suffering from leptospirosis, whose ages ranged from 17 to 34 years. Eleven were New Zealanders, six Gurkhas, four Australians, three English, and one American. In each case the clinical diagnosis was later confirmed by a diagnostic rise in the antibody titre in a haemolytic test for leptospirosis done on paired sera. The series includes all patients with this disease admitted to the British Military Hospital, Kamunting, Malaya, during an 18-month period in 1960–2 and who had at least two E.C.G.s done during the illness.

When first examined, none of these patients had clinical evidence of congestive cardiac failure, cardiac enlargement, pericarditis, valvular disease, hypertension, or hypotension. None had an arrhythmia, although most had a tachycardia. Chest x-ray films, taken routinely on admission, confirmed the normal heart size.

The first E.C.G. record was taken during the first week of the clinical illness in 84%, and during the first three days in 40%. The second record was taken about eight days later, when the patient was afebrile. In all but three cases the first E.C.G. was taken before penicillin treatment was started.

For comparison, a second series of E.C.G.s was done on 18 febrile soldiers shown by clinical evidence, microscopical demonstration of the parasite, and response to therapy to have malaria. The E.C.G. records were taken in the same way during the febrile stage, and about a week later.

E.C.G. in Patients with Leptospirosis

P, QRS, and T Waves.—Significant abnormalities were found mainly in the R, S, and T waves. The P and Q waves were normal and unchanged throughout. The R wave was less than 5 mm. tall in the most favourable standard lead, and in V4 or V7 in a third of all first records. The QRS complex was less than 7 mm. tall in the most favourable standard lead in these cases. Apart from the occasional failure of a small R, to increase in size, the R waves throughout all second records were within the accepted limits of the normal range. Nearly all patients showed an increase in the size of the R waves in standard and chest leads as the fever abated. The greatest increase in size in the standard leads was usually in lead II, and the average of the greatest increases was 4.6 mm. The greatest increase in the chest leads was in V4, and the average 6.5 mm. (Table I). Forty-four per cent. of patients showed an increase of 3 mm. or more in the height of the R wave in two standard leads, and 36% an increase of 3 mm. or more in two chest leads. The S wave decreased in size in a standard lead in two-thirds of the patients as the fever abated. The decrease averaged 1.7 mm., and was usually seen in lead I or II. S1 and S2 also decreased in size, but S1 usually showed an increase. The T wave in leads I and II was less than 1.5 mm. tall in 72% of all first records, and T4 or T5 was reduced in size in 52%, T5 being inverted in 28%. During recovery there was a general increase in the size of the T wave in all standard and chest leads. The change was most marked in leads I, II, and V4 (Table II).

Serial E.C.G.s showed that by the eleventh day of the clinical illness the record had returned to normal, and no further major changes occurred.

Other Abnormalities.—Elevation of the S-T segment by about 1 mm. occurs in 20% of second records, especially in leads I and V7 after the disappearance of an S wave. The pulse rate increased at the rate of 6 beats/minute/degree Fahrenheit. Occasionalventricular extrasystoles were seen in two first records, and one patient developed atrial fibrillation late in his illness. The P–R interval was normal throughout, with the
exception of one reading of 0.22 sec on a first record. There
was no consistent change during recovery. The QRS complex
rarely lasted longer than 0.1 sec. and never longer than 0.12 sec.
It varied little with temperature. One set of records showed
left and one right bundle-branch block throughout. The Q-Tc
interval often exceeded 0.42 sec. in both sets of records. It did
not show any consistent change during recovery.

E.C.G. in Patients with Malaria

Identical changes occurred in patients with malaria. The R
waves were abnormally small in all leads in about 30% of first
records, and increased in height in nearly 90%, during recovery.
The change was best seen in leads II and V4, and was of the
order of 5 mm. (Table I). Simultaneously I, II, V4, and V7
decreased in height.

The T wave was abnormally flat in standard leads in 72%
of the first records, and T was flat or inverted in 39%. Nearly
all returned to normal, and 80% of patients showed an
increase in the height of the T wave of the order of 2 to 3 mm.
during recovery, especially in leads I, II, and V4 (Table II).

The P and Q waves were normal and unchanged throughout.
The P-R and QRS intervals tended to shorten in the febrile
stage, but were never abnormal. The pulse rate increased by
6 beats/minute/degree Fahrenheit.

Discussion

Examination of E.C.G.s taken in the acute stage of leptos-
pirosis, and comparison of them with others taken during
recovery, showed that 90% of first records were abnormal.
The chief abnormalities were a tachycardia, small QRS com-
plexes, an increase in the size of the S wave in leads I and V7,
and flat or inverted T waves (Fig. 1). The age and nationality
of the patient, the duration of the illness, and the height of the
fever did not influence these changes, which disappeared during
the first 10 days, leaving only a slight S-T elevation in leads I
and V7 on some records taken during convalescence.

Similar changes have been described or depicted before.
Plate I in the article by Mackay-Dick and Robinson (1957)
shows an increase in the height of R, the disappearance of S, a
slight S-T elevation, and a marked increase in the height of the
T waves. The relative slowness of the pulse in proportion to
the fever (a rise of 6 instead of the usual 7 to 10 beats/minute/
dergee Fahrenheit) was noted by Dawson and Hume (1916-17).
On the other hand, no evidence of a consistent change in the
P-R and Q-T intervals was found, and, for obvious reasons,
there was no evidence of the arrhythmias or pericarditis, descrip-
tions of which hitherto dominated the literature. The
results confirm the predictions of Hume and Szekely (1944)
and of Sodeman and Killough (1951) that cardiographic evidence
of cardiac involvement may be found in the absence of physical
signs, especially if serial records are examined.

Although the changes described occur regularly in leptos-
piosis, they are not peculiar to this disease. Examination of
serial E.C.G. records from 18 otherwise healthy soldiers with
malaria contracted under similar operational conditions revealed
changes almost identical in nature, frequency, and degree.
Seventy-eight per cent. of first records were outside the strict
limits of normality, and nearly all altered during the recovery
process (Tables I and II; Fig. 2).

Among the common factors possibly responsible for the
identical E.C.G. changes found in the two illnesses, fever is the
most obvious. It is therefore interesting that similar changes
have been reported in patients rendered febrile artificially by
means of a hot-air cabinet. These included diminution in
the height of the R waves, especially in lead II, an increase in
the height of S, and depression of T, with slight elevation
of the S-T segment in leads II and III during recovery
(Knies, 1941). Such changes have been attributed to acute
diffuse coronary insufficiency caused by tachycardia and
sympathetic stimulation (Lepeschkin, 1951). In febrile infec-
tions the exudation of plasma or cellular infiltrates between
the muscle fibres may further interfere with oxygen diffusion.

If it is accepted that identical changes occur in these two
groups of patients, and that similar changes occur in artificially
induced fever, it cannot be said that the abnormalities found on
Peripheral Sensorimotor Neuropathy Associated with a Localized Myeloma

D. I. RUSHTON,* M.B., CH.B.

Neurological manifestations in diseases of the reticuloendothelial system are not uncommon and are said to occur in 15-50% of cases (Aita, 1962). The vast majority of these cases show neurological symptoms as a result of direct invasion or compression of the nervous tissues. However, in a small number of cases neurological manifestations are severe and yet there is no direct involvement of the nervous system. These may be similar to those syndromes associated with carcinoma.

Victor et al. (1958) presented five cases of sensorimotor neuropathy associated with myeloma, in three of which post-mortem examination revealed no evidence of invasion of the nervous system. Barron et al. (1960) described three cases in which local infiltrations of the peripheral nerves by plasma cells led to demyelination of the adjacent nerve fibres. Other cases of neuropathy associated with myeloma have been reported by Hassan and Yousef (1959) and Boudin et al. (1961), and the condition has been noted by Oslerman (1959).

The case reported below is believed to be a case of the type described by Victor et al. (1958).

Case History

A man aged 45 first noticed coldness and numbness of his feet in January 1962. This was dismissed as being due to the cold weather. However, the condition progressed and paraesthesiae of his lower limbs developed below the knees. In July he had marked difficulty in walking and experienced numbness of his hands.

In August he was admitted to the Midland Nerve Hospital for investigation. At this time he had great difficulty in walking, paraesthesiae of the hands and feet, and increasing clumsiness when using his hands.

Clinical examination revealed extensive neurological manifestations in an otherwise normal male. These were: The upper limbs showed loss of power, areflexia, impaired pinprick and vibration sense, and loss of touch sensation below the elbows. The lower limbs showed gross loss of power, areflexia, absent joint sense, and loss of pinprick and vibration sense below the costal margin. The plantar response was absent. These findings were symmetrical.

After admission his condition slowly progressed, and while in hospital he developed deep-vein thrombosis of his calf; this led to pulmonary embolus, from which he made a satisfactory recovery with the aid of anticoagulant therapy.

On 6 September a bony lesion of D 11 was first noted on an x-ray film; it was thought to be myeloma, but no other confirmation of this diagnosis was obtained.

The neuropathy was provisionally diagnosed as a form of Guillain-Barré syndrome, and steroid therapy was begun. This led to a slight temporary improvement in the power of his legs; but it was not maintained, and the symptoms and progression of the disease continued.

In May 1963 he was transferred to Selly Oak Hospital. At this time he had paraesthesiae of all limbs, intermittent dysesthesia, areflexia, gross impairment of sensation, and severe attacks of pain in the lower limbs. Muscular weakness was severe. The fundi showed blurring of the disks. On exercise he became very dyspnoeic, probably as a result of involvement of his respiratory muscles. Gradually over the next few months his condition deteriorated and he developed gangrene of the right fifth toe. By August severe sensorimotor neuropathy of his upper limbs, shoulders, and lower limbs was evident. The extremities were cyanosed and the gangrene of the right foot was progressing.

* Department of Pathology, Selly Oak Hospital, Birmingham. Now Research Fellow, Department of Morbid Anatomy, Children's Hospital, Birmingham.