Wenckebach phenomenon occurring in secondary syphilis

WINSTON E. INCE* AND BISRAM S. MAHABIR†

From the General Hospital, Port of Spain, Trinidad

Cardiac involvement in late syphilis is a relatively common occurrence and is a consequence of syphilitic aortitis, or rarely of gummatous lesions of the myocardium. Not so well known is the fact that cardiac manifestations in the form of abnormalities of the electrocardiogram may occur in early syphilis. Steiger and Edeiken (1948) recorded electrocardiographic abnormalities in 50 per cent. of a series of thirty cases, and in 42.5 per cent. of a further series of forty cases of early syphilis. Eisenberg, Davis, Wright, and Johnwick (1952) found that 38 per cent. of 29 patients with secondary syphilis showed abnormalities in the electrocardiogram before treatment, and one of these showed a conduction defect in the form of a prolonged P-R interval. Conduction defects were not observed in Steiger and Edeiken's series.

In this paper a case is reported of the Wenckebach phenomenon occurring in a patient with secondary syphilis. The discovery of this case prompted a further study of 49 consecutive cases of secondary syphilis. The patients were interviewed and questioned with regard to the presence or absence of cardiac symptoms, and a resting electrocardiogram was performed.

Case report

A 33-year-old unmarried negro mason was referred from the Chest Clinic on November 18, 1969, to the Cardiac Clinic of the General Hospital, Port of Spain. He had presented at the Chest Clinic on October 20, 1969, with the history that for the previous 2 months, to use his own words, 'my heart stays just so and starts to beat fast'. On the first occasion this occurred after walking home some distance from a funeral, but on the second occasion it occurred while standing talking to a neighbour, and was associated with faintness.

He was otherwise well and did not admit to any effort dyspnoea, chest pain, or ankle swelling. When seen at the Cardiac Clinic on November 18, 1969, he complained of a non-itching rash over the face, trunk, limbs, palms, and soles.

Examination

He was a fit-looking young man with a macular, slightly scaly rash over the trunk, forearms, palms, and soles. On the palms and soles the rash was pigmented (Fig. 1). The pulse was irregular, every fifth or sixth beat being dropped, and the rate was 44 per minute. The jugular venous pressure was normal. The heart was slightly enlarged with a left ventricular impulse 1 in. outside the mid-clavicular line. The first heart sound was of variable intensity. There were no murmurs. The radial pulses were equal, and the femoral, dorsalis pedis, and posterior tibial pulses were present. There was no lymphadenopathy, and the liver and spleen were not felt. There were no genital, buccal, or faucial lesions. The other systems and the optic fundi were normal.

FIG. 1 Lesions of secondary syphilis on palms of hands

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*Consultant Physician, General Hospital, Port of Spain, Trinidad.
Associate Lecturer in Medicine, University of the West Indies,
Eastern Caribbean Medical Scheme

†Consultant Venerologist to the Government of Trinidad and
Tobago, Specialist Medical Officer, Caribbean Medical Centre;
Physician, Dermatology Department of the General Hospitals, Port
of Spain and San Fernando, Trinidad

The ECG showed sinus bradycardia, rate 45 per minute, and a prolonged P-R interval with intermittent A-V block of the Wenckebach type (Fig. 2). The shortest P-R interval measured 0.20 sec., the longest 0.40 sec. Spiky T waves were present in V3 to V6 (Fig. 3).

FIG. 2 *Wenckebach phenomenon*

FIG. 3 *Wenckebach phenomenon with spiky T waves*

Laboratory investigations

Hb—13.9 g. per cent.; white blood count—5,000/c.mm (neutrophils 45 per cent., basophils 1 per cent., lymphocytes 48 per cent., monocytes 6 per cent.); erythrocyte sedimentation rate—25 mm./1st hr (Wintrobe); sickle cell test negative; serum electrolytes—sodium 140, potassium 4.5, chloride 95 mEq/L; blood VDRL reactive 1 in 32; chest x ray—slight cardiac enlargement, normal lung fields.

On November 25, 1969, he was referred to the Venereal Diseases Clinic where the diagnosis of secondary syphilis was confirmed. He was treated with one intramuscular injection of 2.4 m.u. benzathine penicillin G.

Progress

After he was given the penicillin therapy, the palpitations did not recur. On December 2, 1969, the ECG still showed the Wenckebach phenomenon, but by December 30, 1969, 42 days after this finding was first observed, the ECG had reverted to regular sinus rhythm, rate 67 per

minute, with a P-R interval of 0.20 sec. (Fig. 4). The rash responded rapidly to the penicillin.

When he was re-examined on August 26, 1971, approximately 20 months after the original episode, he reported that he was well. No abnormal physical signs were noted, and the blood pressure was normal. The ECG showed sinus rhythm, rate 60 per minute, and a slightly prolonged P-R interval of 0.24 sec. (Fig. 5). Chest x ray (Fig. 6) still showed slight cardiomegaly. Blood VDRL, repeated on August 5, 1972, was non-reactive.

FIG. 4 *ECG showing disappearance of Wenckebach phenomenon*

FIG. 5 *ECG showing slightly prolonged P-R interval*

Study of 49 patients

49 consecutive patients with secondary syphilis were referred from the Venereal Diseases Clinic to the Cardiac Clinic where they were interviewed. 47 were of negro or mixed origin, and two were East Indian (both female). Their ages ranged from 14 to 56 yrs, 22 being male and 27 female (Table I).

History

All gave a history of exposure to venereal infection, and all had the characteristic lesions of secondary syphilis with reactive VDRL tests. None admitted to cardiac symptoms such as palpitations, chest pain, or effort dyspnoea.

FIG. 6 Chest x ray showing slight cardiac enlargement

TABLE I Age and sex distribution of 49 patients

Age group (yrs)	Sex	
	Male	Female
10-14	1	0
15-19	4	6
20-24	7	7
25-29	5	7
30-34	2	3
35-39	1	3
40-44	1	1
45-49	0	0
50-54	0	0
55-59	1	0
Total	22	27

Clinical examination

This was negative in 48 patients except for the presence of the skin lesions of secondary syphilis, and raised blood pressure (160/110, 150/110, and 140/100) in three cases. One patient (M. P.) was found to have an apical systolic murmur due to mitral incompetence.

Electrocardiograms

These were normal in 41 patients, including the three with raised blood pressure, and abnormal in eight (Table II), the incidence of abnormality being 16 per cent. Included among the normal ECGs were an ST elevation of the type commonly recorded in the negro in one case, and infantile T inversion in V1 to V3 in another. ST elevation in the

left precordial leads is a common finding in healthy negroes and has also been recorded in healthy Caucasians (Greene and Kelly, 1959; Grusin, 1954; Woods and Laurie, 1959; Fleishman and Gelfand, 1960). It is thought to be due to early ventricular repolarization.

Of the eight abnormal tracings (Table III), three showed non-specific ST-T changes, and four showed partial right bundle branch block. One tracing (of the patient with clinical evidence of mitral incompetence) showed left atrial hypertrophy. No tracing showed A-V nodal conduction disturbance.

TABLE II Details of eight patients with abnormal electrocardiograms

Patient	Sex	Age (yrs)	Blood pressure	Electrocardiogram
R.F.	M	18	160/70	Non-specific lowering of T in II and V6
A.B.	F	19	120/80	Partial RBBB
C.R.	M	16	110/70	ST depression in II, III, avF
M.B.	F	17	110/60	Partial RBBB
U.N.	F	27	120/50	Partial RBBB
W.P.	M	19	120/70	Partial RBBB
M.P.	M	24	150/75	L.A. hypertrophy (apical S.M.)
S.J.	F	39	120/80	Flat T V5 and V6, T inversion II, III, avF

TABLE III Types of abnormal electrocardiograms

Type	Male	Female
Non-specific ST-T changes	2 (16, 18) ^a	1 (39) ^a
Partial RBBB	1 (19)	3 (19, 17, 27)
Left atrial hypertrophy	1 (24)	

^aAge (yrs) in brackets

Discussion

Apart from involvement of bones and joints, eyes, liver, and nervous system, visceral manifestations of secondary syphilis are extremely rare. It is suggested that, in the case described in this paper, the Wenckebach phenomenon was produced by treponemal invasion of either the A-V node or its blood supply. The A-V nodal artery arises from the right coronary artery in 90 per cent. of cases and from the circumflex branch of the left coronary artery in the remaining 10 per cent. It is possible that endarteritis occurring during the secondary stage of the disease may involve the A-V nodal artery, resulting in impaired blood supply to this structure leading to impaired function. If this is so, it is conceivable that other small coronary vessels may become involved in the endarteritic process of secondary syphilis but, as far as is known, myocardial ischaemic syndromes are not encountered at this stage. This fact makes it unlikely that the A-V nodal dysfunction manifested in our patient had a vascular basis. It seems more likely that direct invasion of the A-V nodal structure had taken place.

It was first assumed that the slight cardiomegaly in our patient was due to the bradycardia, but 20 months later the heart was still enlarged, although the cardiac rate had risen to 60 per minute. This finding suggests the possibility that focal lesions occurred throughout the myocardium. Penicillin therapy did not bring about any diminution in cardiac size, although the reversion of the reactive blood VDRL suggested cure of the disease. It is also possible that the slight cardiac enlargement was unrelated to the syphilis. The symptom with which the patient presented suggested some form of paroxysmal tachycardia, but the opportunity for taking an ECG tracing during an attack did not present itself. The paroxysmal tachycardis could conceivably be a manifestation of secondary syphilitic myocarditis.

It is much more common for cardiovascular syphilis to manifest itself during the tertiary stage of the disease. The lesions characteristically involve the proximal aorta and the coronary ostia, producing aortic aneurysms, aortic incompetence, and myocardial ischaemic syndromes. Gummata may appear in any organ, and the heart is no exception. Gummatus myocarditis has been described (Leach, 1960; Ramgam, Bhagwat, and Bhandari, 1960). Atrio-ventricular block due to gumma has also been well documented (Ramamoorthy, Sahiar, and Golwalla, 1962; Soscia, Fusco, and Grace, 1964). The disturbance of A-V nodal function described in this paper is interesting in that it provides evidence of A-V nodal involvement as early as the secondary stage of the disease, so establishing the basis for gummatous development later.

The relative infrequency of disordered A-V nodal function in secondary syphilis is indicated by the absence of A-V nodal conduction disturbance in the electrocardiograms of the 49 consecutive patients studied. Of the 49 patients, eight (16 per cent.) had abnormal electrocardiograms. This figure included four patients with partial right bundle branch block. It is well known that partial right bundle branch block is a fairly common finding in healthy persons, and its presence does not necessarily indicate organic heart disease. Bundle branch block was not described in the series reported by Steiger and Edeiken (1948) and Eisenberg and others (1952). Included also among the abnormal electrocardiograms was that of one patient with left atrial hypertrophy due to mitral regurgitation. ST and T wave abnormalities occurred in only three patients (6 per cent.) compared with 42.5 per cent. in the series studied by Steiger and Edeiken (1948).

Summary

A case is described in which A-V nodal dysfunction

in the form of the Wenckebach phenomenon occurred in a patient suffering from secondary syphilis. It was associated with a slight degree of cardiac enlargement. The patient presented with palpitations which resolved with antisyphilitic therapy, but the cardiac enlargement persisted. Electrocardiograms carried out on a further 49 consecutive patients with secondary syphilis showed no evidence of A-V nodal conduction defect. Abnormal electrocardiograms were found in eight (16 per cent.) of the series, and in three (6 per cent.) the abnormality consisted of ST segment and T wave changes.

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Phénomène de Wenckebach survenu au cours d'une syphilis secondaire

SOMMAIRE

On rapporte un cas de trouble auriculo-ventriculaire nodal, se manifestant sous forme du phénomène de Wenckebach, chez un malade atteint de syphilis secondaire. Il existait en outre une légère hypertrophie cardiaque. Le malade accusait des palpitations qui disparurent par le traitement antisyphilitique alors que l'hypertrophie cardiaque persistait. Des électrocardiogrammes pratiqués ensuite sur une série consécutive de 49 syphilitiques secondaires ne montra pas de défaut de la conduction nodale auriculo-ventriculaire. L'électrocardiogramme fut trouvé anormal chez 8 (16 pour cent) des sujets de cette série, et chez 3 (6 pour cent) l'anomalie portait sur le segment ST avec modifications de l'onde T.