Coexistent Wenckebach Phenomenon in the Distal Branches of the Specialized Conduction System*

Samuel Sclarovsky, M.D.; Boris Strasberg, M.D.; and Jacob Agmon, M.D.

A patient in whom Wenckebach phenomenon was observed to coexist in the three distal branches of the specialized ventricular conduction system, with an intermittent trifascicular block producing a second degree A-V block, is described. Surface electrocardiogram demonstrated the presence of this phenomenon in the right bundle branch (RBB) and the anterior division of the left bundle branch (LBB). Evidence of the presence of Wenckebach phenomenon in the remaining fascicle was provided by His bundle recording.

It is now known that the Wenckebach phenomenon does not occur exclusively at the A-V node junction. In recent years, there has been increasing evidence of the appearance of the Wenckebach phenomenon both above and below the A-V node. In 1969, Rosenbaum et al. reported electrocardiographic evidence of this phenomenon in the bundle branches and since the introduction of His bundle electrography, evidence has also been obtained of the occurrence of the phenomenon within the atria,5-8 as well as the specialized ventricular conduction system. Kretz and DaRuds9 presented experimental evidence for the occurrence of Wenckebach phenomena in the anterior and posterior divisions of the left bundle and since then there has been accumulating clinical evidence for the appearance of this phenomenon in the posterior division of the left bundle.5-8

The purpose of this communication is to describe a patient in whom the Wenckebach phenomenon was demonstrated in the three branches of the distal conduction system.

CASE REPORT

A 76-year-old man with a history of myocardial infarction was admitted for investigation because of two episodes of syncope. On admission, he was in good general condition with auscultatory findings of mild aortic stenosis and regurgitation. Blood pressure was 140/80 mm Hg and pulse rate 80/min. The electrocardiogram showed normal sinus rhythm with a right bundle branch block (RBBB) and left anterior hemiblock pattern (LAHB). Laboratory findings, including serum enzymes SGOT and LDH, were within normal limits.

*From the Israel and Ione Massada Center for Heart Diseases, Intensive Coronary Care Unit and Institute for Cardiac Rehabilitation, Beilinson Medical Center, Petah Tikva, Israel.

Reprint requests: Dr. Agmon, Beilinson Medical Center, Petah-Tikva, Israel

FIGURE 1. Top to bottom, leads 1, 2 and 3. Lead 2 shows an increased left axis deviation (electrical axis of -10°, -30°, -45°, -60°) due to advancing left anterior hemiblock. There is a gradual widening of the QRS due to probable simultaneous RBBB.

Shortly after admission, 2nd degree A-V block was observed, and for this reason a His bundle electrogram (using Scherlag's technique9) and three-channel ECG were recorded.

RESULTS

Figures 1 and 2 give representative sections taken from continuous recording with a three-channel ECG.

FIGURE 2. Top to bottom, aVR, aVL, aVF. Lead aVR shows a progressive widening of the QRS (0.08 sec, 0.10 sec, 0.12 sec) due to a RBBB.
Figure 1 represents leads 1, 2, 3. The first beat shows a normal conduction pattern with an electrical axis of $-10^\circ$. The three following beats show a progressive left axis deviation due to an advancing left anterior hemiblock (LAHB) with an electrical axis of $-30^\circ$, $-45^\circ$, and $-60^\circ$ respectively. The P-R interval remains constant at 0.18 sec and there is an increasing widening of the QRS: 0.08 – 0.10 sec and 0.12 sec respectively. This widening of the QRS is due to probable simultaneous RBBB. Figure 2 represents leads aVR, aVL, aVF. Of note in lead aVR is the progressive widening of the QRS due to the last vector which is the result of the RBBB. Figure 3 shows the results of the His bundle study performed and a simultaneous recording of lead 2 with a three-channel ECG. There is a spontaneous sinus rhythm with a second degree A-V block. Leads 2 and V1 show a normal conduction pattern after the dropped beat followed by a complete LAHB and RBBB pattern up to the next dropped beat. The His bundle strip demonstrates the presence of the A-V block below the His deflection and measurement of the HV interval shows a progressive increase (40-90-100 msec) up to the dropped beat. It is of interest that there is no change in QRS morphology between beats 2 and 3, and 6 and 7.

**Discussion**

In 1925, Scherf and Shookhoff reported experimental evidence of the occurrence of the Wenckebach phenomenon in the bundle branches of dogs. Friedberg and Schamroth presented electrocardiographic evidence of the phenomenon in the left bundle in a single patient and in the same year, Rosenbaum et al. reported two such cases, one in which the Wenckebach phenomenon was demonstrated in the right bundle and the other in the left bundle. The advent of His bundle electrography has made it easier to obtain more exact evidence of the Wenckebach phenomenon. Narula and Samet presented a case occurring within the His bundle area and another occurring distal to the HB. Ranganathan et al. in 1972 found that the Wenckebach phenomenon may occur in the contralateral bundle or in the corresponding Purkinje system in patients suffering from bundle branch block. Kretz and DaRuds experimentally demonstrated the appearance of the Wenckebach phenomenon in the anterior and posterior divisions of the left bundle branches of the canine heart. There have also been recent reports of the Wenckebach phenomenon in the posterior division of the LBB.

In our patient, the ECG on admission showed a pattern of RBBB and LAHB with a normal P-R interval, but shortly afterwards a 2nd degree A-V block was observed. Continuous recording with a 3-channel ECG showed a Wenckebach phenomenon in the RBB as evidenced by gradual prolongation and change in the morphology of the QRS from normal conduction to an incomplete and then to a complete RBBB pattern (Fig 2). The same phenomenon was observed in the anterior division of the LBB, as evidenced by a gradual increase in the left axis deviation, beginning with a normal conduction pattern (electrical axis $-30^\circ$, $-45^\circ$, $-60^\circ$) (Fig 1).

In the His bundle recording (Fig 3), the first beat following the dropped one conducts with a normal pat-
tern and normal HV interval (40 msec). Afterwards, there is a complete RBBB and LAHB pattern with an HV interval of 90 msec, followed by an additional increase in the HV interval (100 msec) with no further change in the other two bundles, the RBB or the anterior division of the LBB. The next beat is a dropped beat. It cannot be affirmed that the increase in the HV interval from 40 to 90 msec is due to a Wenckebach phenomenon in the posterior division of the LBB since there are simultaneous delays in the two remaining fascicles. However, the further prolongation of the HV interval from 90 to 100 msec, while the remaining bundles already show a complete block pattern, clearly demonstrates an increase in conduction time within an infra-Hisian area, most probably the posterior division of the LBB. The change in morphology of the QRS in leads 2 and V1 from the first to second beat (after the dropped beat) is due to Wenckebach phenomenon in the RBB and anterior division of the LBB.

In conclusion, these findings demonstrate a Wenckebach phenomenon coexisting in the three distal branches of the specialized ventricular conduction system.

REFERENCES


**Survival of a Patient with Pancytopenia and Disseminated Coagulation Associated with Miliary Tuberculosis**

Michael J. Rosenberg, M.D., and Larry W. Rumans, M.D.

A 56-year-old man with histologically and bacteriologically proved disseminated tuberculosis in association with pancytopenia responded to antituberculosis chemotherapy with bacteriologic cure of his tuberculosis and concomitant resolution of the pancytopenia. This association has been generally believed to have a nearly 100 percent mortality. In addition, the patient developed laboratory evidence of disseminated intravascular coagulation (DIC). The single and simultaneous occurrence of these two hematologic abnormalities is extremely rare. A number of factors possibly relating to the development of pancytopenia and DIC in conjunction with miliary tuberculosis are briefly discussed.

The association between disseminated tuberculosis and severe hematologic abnormalities has been long recognized. Pancytopenia in particular, rare even in disseminated tuberculosis, has proven a grave prognostic indicator. Disseminated intravascular coagulation (DIC) in conjunction with miliary tuberculosis has only rarely been reported and in none of the previous cases has there been an associated pancytopenia. We report the first patient with pancytopenia and DIC believed to be secondary to disseminated tuberculosis. Our patient recovered on therapy with isoniazid, ethambutol, and rifampin.

**CASE REPORT**

A 56-year-old black man with a history of alcoholism was brought to the emergency room by his landlady, who reported that he had been lying in bed incontinent and obtunded for the previous two weeks. When last seen two years before this admission, laboratory studies included normal prothrombin activity, hematocrit, white blood cell count and differential, platelet count, serum protein, albumin, bilirubin, and alkaline phosphatase, but the erythrocyte sedimentation rate was 15 mm/hour (Westergren, normal to 10), LDH of 243 IU/L (normal 90-220), and SGOT of 46 IU/L (normal 7-40). Chest radiographs obtained at that time demonstrated apical pleural scarring and a calcified cervical lymph node.

The admission examination revealed a cachectic, obtunded black man who was disoriented to time and place and responded inappropriately. There was no evidence of acute ethanol intoxication. Blood pressure was 126/80 mm Hg and

*From the Department of Medicine and the Division of Infectious Diseases, Santa Clara Valley Medical Center and Stanford University Medical Center, San Jose and Stanford, California.

Reprint requests: Dr. Rosenberg, New England Sinai Hospital, Stoughton, Massachusetts 02072

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