chospasm and edema of the bronchial wall. The problem of plugs is compounded by the invariably present tachypnea, resulting in respiratory loss of fluids. Dehydration produces tenacious, inspissated plugs, which are difficult to remove via the cough mechanism. Messer et al., in a large autopsy series, reported a 97 percent incidence of abnormal bronchial contents (mucous, purulent exudate, or both) in 35 patients dying in status asthmaticus. Mucous plugs in the bronchi and focal areas of collapse were prominent features in another 20 cases of fatal status asthmaticus. In the present case, mucoid material was found at the four bronchoscopies.

The degree of atelectasis is of interest in the present case, since reports of complete atelectasis of a lung are rare in the literature. However, there is no reason to believe that the mechanism responsible for the massive collapse in the present case is qualitatively different from the more frequent lobar or segmental atelectasis.

Treatment in the present case was directed primarily to re-expansion of the right lung. In cases of segmental or lobar atelectasis, conservative management with fluids, chest percussion, administration of bronchodilators and antimicrobials, and intermittent positive pressure breathing, is usually successful. When these measures fail, bronchoscopy with removal of obstructing secretions is indicated. In this patient, bronchoscopy was performed at an early point in the hospitalization because of extensive atelectasis and was repeated until satisfactory expansion of the lung was appreciated.

Careful and persistent suctioning and irrigation of the bronchi were performed during bronchoscopy, with improvement in the postbronchoscopic roentgenogram and clinical condition. As judged by pulmonary function studies, chest roentgenogram, lung scan, arterial blood gas determinations, and clinical status, this patient essentially made a complete recovery. Early and multiple bronchoscopies are indicated in patients with massive asthmatic atelectasis to re-expand the lung and prevent lung abscess and bronchiectasis. Multiple bronchoscopies in the present case preserved a functioning lung that otherwise might have remained collapsed, with resultant tissue destruction.

REFERENCES


Wenckebach Periods in Posterior Inferior Division of Left Bundle

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A case of right bundle branch and left anterior superior division block-bifascicular block, developing into intermittent complete heart block-trifascicular block is described. Atrial stimulation produced Wenckebach periods. Mobitz type II conduction defect, with block below the His-bundle area and at the AV node and complete heart block. His-bundle electrography documented the Wenckebach's phenomenon as occurring below the His bundle in the only conducting fascicle, post inferior division of the left bundle. During atrial stimulation a phenomenon of concealed conduction into the infra-His conduction system was also noted. Implantation of a permanent cardiac pacemaker abolished symptoms of syncope.

Wenckebach's phenomenon was thought to be exclusively a property of AV functional tissue. Its occurrence at the AV node has been confirmed by the use of His bundle electrographic studies. Bundle branches, on the other hand, have not been commonly known to show Wenckebach's phenomenon. Scattered reports describe such an occurrence, and support it by clinical electrocardiograms only. Although experimentally produced, and confirmed by direct recordings by Scherf and Shookhoff in animals in 1925, Wenckebach's phenomenon has been directly demonstrated to occur in bundle branches only in recent rare reports. This communication will describe a patient in whom His bundle electrography confirmed that Wenckebach's phenomenon was occurring in the posterior inferior division of the left bundle, and further that the right bundle branch and left anterior superior division block progressed to complete heart block, necessitating the implantation of a permanent pacemaker.

*From the Cardiology Division, Howard University Medical Service, D.C. General Hospital, Washington, D.C.*
A 58-year-old man was found unconscious on the street and brought to District of Columbia General Hospital on Sept. 30, 1971. After his recovery, he gave a history of “black-out spells,” in 1965. He was free of symptoms for the next three years, after which he suffered from syncopal episodes at least once a month. These episodes occurred without warning and were rarely associated with a generalized type of seizure. He had been drinking alcohol rather excessively during this period. He admitted smoking one pack of cigarettes a day for 20 years. The rest of the history was unremarkable.

**Physical Examination**

On admission to the cardiac care unit, he was noted to be a well developed, poorly nourished, cooperative man in no distress. His blood pressure was 100/70 mm Hg and pulse rate 55/min. Examination findings of the head, neck, ear, nose, and throat were unremarkable. The lungs were clear to percussion and auscultation. The cardiac dullness was within normal limits. The S1 was thought to be regular in rhythm and intensity, while the S2 showed wide splitting. No murmur was heard. Examination of the abdomen revealed generalized tenderness and voluntary guarding. No pedal edema was noted. Neurologic examination was grossly within normal limits.

**Laboratory Findings**

Laboratory studies revealed the following: hemoglobin, 9.3 gm percent; hematocrit 28 percent; leukocytes 6,400 per cu mm; platelets 99,000/cu mm; and reticulocytes, 0.5-1.0 percent. The blood urea nitrogen value was 70 mg percent on admission but later became normal. Serum amylase was 225 units, glutamic oxalacetic transaminase 84 units, and lactate dehydrogenase 825 units. The serum protein level was 6.7 gm percent (albumin 2.2 gm percent), and the total bilirubin, 1.0 mg percent (direct fraction 0.35 percent). The chest x-ray film was unremarkable.

An electrocardiogram showed a normal sinus rhythm with a rate of 75/min, first-degree atrioventricular block, right bundle branch block and left axis deviation (Fig 1A). Three minutes later the patient was found to be in complete heart block, with an idioventricular or His bundle rhythm with aberrant conduction with a rate of 50/min (Fig 1B).

**Hospital Course**

Shortly after admission to the cardiac care unit, a temporary bipolar transvenous pacemaker catheter was inserted and pacing started with a demand AO pacemaker (Fig 1C).

On the third hospital day he was found to be predominantly in normal sinus rhythm. Conducted beats showed complete right bundle branch block, with a stable idioventricular or His bundle rhythm with aberrant conduction. The patient continued this rhythm, when a His bundle electrogram was recorded on the seventh day of hospitalization (Fig 2). A Biotronic permanent "R" wave triggered demand pacemaker was implanted on the eighth hospital day. The patient's subsequent hospital course was uneventful. He also underwent an uncomplicated transurethral prostatic resection for prostatic hypertrophy and symptoms of partial obstruction. He has been followed in the clinic since, and has been asymptomatic.

**Methods**

Leads 1, 2, His bundle electrogram (at a frequency of 120-200 cps) using a bipolar electrode catheter introduced percutaneously through the right femoral vein and V1 were recorded on Kodak photographic paper, running at a speed of 100 mm/sec and time lines marking .04 secs. Baseline recording with the patient in normal sinus rhythm was followed by atrial stimulation obtained by the use of a no. 6 bipolar pacemaker catheter introduced percutaneously through the right femoral vein and positioned in contact with the lateral middle wall of the right atrium. After determining the minimum rate at which the atrium could be captured, the atrial rate was increased by 10/min in a step-wise manner until complete heart block was obtained, and a recording made at each increment. A-H and H-V intervals were measured in milliseconds. The landmarks for the rest of the measurements have already been discussed.210

**Results**

Figure 2 shows the first two beats to be the control normal sinus beats with an A-H interval of 80 msec and an H-V of 100 msec. The P2 is the first captured atrial beat and conducts with a prolonged A-H interval, while
WENCKEBACH PERIODS IN POSTERIOR INFERIOR DIVISION OF LEFT BUNDLE

Figure 2. Top to bottom, leads 1, 2, His bundle electrogram, and lead V1. Distance between two time lines, 40 msec. His bundle potentials marked with arrow and H. Numbers above His bundle electrogram show A-H intervals and below it H-V intervals in milliseconds. P1 and P2 are control conducted beats before atrial stimulation. A-H interval of P3, first atrial paced beat 100 msec, while H-V remains unchanged at 95-100 msec. P3 conducts only up to His-bundle with A-H further lengthening to 115 msec—pseudo-Wenckebach type phenomenon at AV node, because A-A interval gradually shortens up to P4. 2:1 block between P5 and P7 may be form of Wenckebach block when H-V of P5 of 100 msec prolongs to infinity, then recovers to 100 msec in P7.

the H-V remains unchanged. In the second captured beat, P4 A-H is further prolonged to 115 msec, but conducts only up to the His bundle. The A-H interval gradually prolongs from 80-115 msec. It may be noted that A-A interval also decreases from P3 to P4. Thus, this Wenckebach type of block occurring at the AV node is not a true Wenckebach's phenomenon. This is further apparent by the fact that an A-H interval of P5 fails to shorten. The P5 may be termed as a blocked premature atrial beat. Between the P3 and P4, the patient develops a Mobitz II block of 2:1. This is also compatible with 2:1 Wenckebach's phenomenon of a posterior inferior division of the left bundle, since that was the only fascicle conducting below the His bundle. Atrial pacing, as shown in Figure 3, produces a Mobitz II 2:1 block between the P3 and P5. The P4 is blocked in the AV node, and not below the His bundle area, as seen in Figure 2.

Continuous atrial pacing produced yet another kind of infra-His block, as is seen in Figure 4. All the P waves are conducted up to His bundle, with a rather unchanging A-H of 80 msec. The P5 fails to conduct beyond the His bundle. The manner in which this occurs is evident from the preceding three beats. From a stable H-V of 125-130 msec in beats one and two, there occurs a gradual prolongation of the H-V to 170 msec for the P6, and the P7, gets blocked in the infra-His area, a Wenckebach's phenomenon in the posterior inferior division of left bundle, since the anterior division of left bundle and right bundle are already blocked, and the absence of split His bundle deflections rules out block in the distal His bundle area. Following the blocked P5, the posterior

Figure 3. Leads 1, 2, His bundle electrogram and V1. P1-P3 conduct with an unchanging A-H and H-V interval 80 msec. P4 unexpectedly is dropped—Mobitz II phenomenon but block resides in AV node, rather than area below His bundle.

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The infra-His block may reside anywhere between the His bundle (recordable), but a little beyond it (not recordable), although not all, owing to the impulse travels beyond the His bundle, but only partially, thus fails to produce a QRS, and consequently there was no deflection recorded after the His potentials. This thesis is borne out by the His bundle and Purkinje fibers. This infra-His block may represent a form of Wenckebach's phenomenon where the H-V prolongs from 120 msec to infinite, and a dropped beat ensues. If this were true, then an H-V of the P2 would recover to 120 msec, or less. The same phenomenon can also be explained by supposing that the impulse travels beyond the His bundle, but only partially, thus fails to produce a QRS, and consequently there was no deflection recorded after the His potentials. This thesis is borne out by the H-V measurement of 140 msec for the P3. Since the atrial rate and A-H interval remain unchanged in the preceding beats, there are only two possibilities by which the H-V could have further prolonged from 120 msec. The first may be that the H-V changed due to autonomic influences. A second explanation would be that the P3 not only conducted up to the His bundle (recordable), but a little beyond it (not recordable), although not all the way to the Purkinje fibers, thus failing to produce ventricular depolarization. This incomplete and concealed penetration to the infra-His conduction system is responsible for a further prolongation of the H-V by 20 msec for the next conducted P wave. The latter explanation seems more plausible, since we know that the autonomic system has little influence over the infra-His conduction.

From the P3 to the P20 the patient goes into complete heart block, with an idioventricular rhythm of 38/min. The QRS configuration is similar to one seen in Figure 1-B. During this time the A-H remains constant at 120-130 msec, and the site of block is thus localized in the infra-His region. The P20 and P22 are conducted with a prolonged A-H and H-V interval. The P21 is blocked below the His-bundle.

Results of further acceleration of the atrial pacer to 137/min are seen in Figure 5. The opening beat has an A-H and H-V of 120 and 120 msec, respectively. The P2 shows evidence of conduction up to the His bundle area only. The infra-His block may reside anywhere between the His bundle and Purkinje fibers. This infra-His block may represent a form of 2:1 Wenckebach's phenomenon where the H-V prolongs from 120 msec to infinite, and a dropped beat ensues. If this were true, then an H-V of the P3 would recover to 120 msec, or less. The same phenomenon can also be explained by supposing that the impulse travels beyond the His bundle, but only partially, thus fails to produce a QRS, and consequently there was no deflection recorded after the His potentials. This thesis is borne out by the H-V measurement of 140 msec for the P3. Since the atrial rate and A-H interval remain unchanged in the preceding beats, there are only two possibilities by which the H-V could have further prolonged from 120 msec. The first may be that the H-V changed due to autonomic influences. A second explanation would be that the P2 not only conducted up to the His bundle (recordable), but a little beyond it (not recordable), although not all the way to the Purkinje fibers, thus failing to produce ventricular depolarization. This incomplete and concealed penetration to the infra-His conduction system is responsible for a further prolongation of the H-V by 20 msec for the next conducted P wave. The latter explanation seems more plausible, since we know that the autonomic system has little influence over the infra-His conduction.

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**FIGURE 4. Top to bottom, leads 1, 2, His bundle electrogram and lead V1. All P waves are result of atrial stimulation. A-H remains constant from P1 to P8. H-V progressively lengthens from 125 msec in P3 to 170 msec in P4, followed by failure of conduction of P5 below His bundle after which H-V recovers to 125 msec in P9-4:3 Wenckebach block of posterior inferior division since right bundle and left superior division are already not conducting.**

**Discussion**

Scherf and Shookhoff were able to demonstrate directly the occurrence of Wenckebach's phenomenon in bundle branches under experimental conditions in dogs. With the advent of His bundle electrography, it was possible for the first time to study, document and localize various conduction abnormalities along the AV node-His axis in an intact human heart. Conduction through the infra-His pathway, ie, from His through the three fascicles, could be studied through the H-V interval. Rosenbaum's group have convincingly shown clinical electrocardiograms revealing Wenckebach's phenomenon in right and left bundles. The hallmark of all the electrocardiograms was the progressive development of complete bundle branch block. Our case is unique, in that from the conventional electrocardiograms it would have been passed as showing Wenckebach's periods of 4:3 in the AV node (Fig 3), because there is a gradual prolongation in the P-R interval from 290 msec to 330 msec before the P6 fails to conduct. Throughout the tracing the QRS remains constant, thus establishing one more pattern of fascicular Wenckebach's phenomenon, in that if, in a setting of bifascicular block the P-R interval lengthens, culminating in a nonconducted P wave, the condition may be compatible both with the Wenckebach's periods in the AV node and/or in the remaining conducting fascicle. The present case also documents concealed conduction phenomenon in the posterior inferior division of the left bundle. Figure 4 shows the P7, conducting with an A-H of 120 and H-V of 120 msec. The P3 is documented to have conducted to the area of His bundle, at least. It might also have conducted for an undetermined distance below the His bundle, but short of producing ventricular depolarization. This incomplete infra-His conduction is not recordable by the present method of His bundle electrography. This would cause a prolonged recovery period of the fascicle, which would be manifested by lengthening of the H-V interval for the next conducted beat. With the rate of atrial stimulation remaining unchanged, a further prolongation of the H-V from 120 to 140 msec for the P7...
fascicular system and presents rather an uncommon complication of complete heart block in a patient with right bundle branch block and left anterior superior division block, in that in an unselected series with this pattern the risk of development of complete heart block is only 10 percent. The absence of "black-out spells" after the implantation of a permanent pacemaker makes it reasonable to assume that the history of those "black-out spells" was due to development of intermittent complete heart block.

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