Electrophysiological features of complete AV block within the His bundle

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The site of block in 3 patients with complete heart block was localized within the His bundle with the aid of the His bundle electrogram. Two patients had normal QRS morphology while the third had a pattern of right bundle-branch block. Split His bundle potentials, i.e. H and H’ denoting the activity of the proximal and distal His bundle segments, were seen in all 3 patients. Atrial pacing in 2 patients produced a gradual increase in the AH interval, consistent with normal AV nodal function. In one of these, His bundle pacing was also successful and produced QRS complexes similar to the spontaneous beats. Intravenous atropine was used in 2 patients. In one it did not increase the heart rate, while in the other the heart rate increased slightly. It is concluded that heart block may occur within the main His bundle and that the surface electrocardiogram is of no use in predicting the site of block in such patients. Response to intravenous atropine may be inadequate or absent.

The technique of His bundle electrography introduced by Scherlag et al. (1969) has made possible the localization of the site of delay in various atrioventricular conduction abnormalities. Block occurring within the His bundle was initially described by Narula and Samet (1970). Two types of His bundle potentials, i.e. H and H’ denoting the activity of the proximal and distal His bundle segments, were recorded in such patients. More recently, several other workers have described patients with various conduction abnormalities localized within the His bundle (Gupta et al., 1972a; Rosen et al., 1971, 1972; Schuilenberg and Durrer, 1972). The present report describes three cases of complete AV block within the His bundle. The results of His bundle pacing and the response to atropine are discussed.

Electrophysiological studies
The His bundle electrograms were recorded by the method described by Scherlag et al. (1969). A size 6, bipolar electrode wire with interelectrode distance of 1 cm was passed percutaneously via the right femoral vein and advanced to the tricuspid valve. Electrograms were recorded on an Electronics for Medicine¹ multi-channel recorder at a frequency response of 40–500 Hz and paper speeds of 75 and 150 mm/sec. One or more leads of the peripheral electrocardiogram were recorded simultaneously. Atrial pacing was performed by a second bipolar wire which was positioned against the lateral wall of the right atrium.

The following measurements were made. The AH interval which included AV nodal conduction time was measured from the onset of the atrial depolarization (A) to the onset of the proximal His bundle deflection (H). The H’Q interval which represents conduction time in the distal His bundle segment and bundle-branches was measured from the onset of the distal His bundle deflection (H’) to the onset of the ventricular depolarization.

Case reports
Case 1 A 74-year-old Cuban-born woman was seen at the cardiac clinic because of episodes of dizziness of several months’ duration. Six months before this visit, the patient was noted to have a slow pulse rate by her physician. Physical examination revealed a regular pulse rate of 39 a minute. Blood pressure was 190/90 mmHg in both arms. A 12-lead electrocardiogram on admission (Fig. 1) showed complete AV block with an atrial rate of 93 a minute and a ventricular rate of 39 a minute. The ventricular complexes were narrow and measured 0.06 sec in duration. The His bundle electrograms were recorded during temporary pacemaker insertion. Fig. 2 shows the His bundle electrograms of this patient with a lead II electrocardiogram. Complete AV block is evident. Each atrial depolarization (A) is followed by a biphasic proximal His bundle potential (H) at an AH interval of 95 msec. Every ventricular depolarization is preceded by a separate biphasic spike labelled H’ at an

¹ Electronics for Medicine, Inc., White Plains, New York.
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Case 2 This 87-year-old white woman was admitted to the hospital with a history of recent dizziness. The patient also had several syncopal episodes two days before the admission. On examination the pulse rate was 40 a minute and the blood pressure was 124/80 mmHg. A 12-lead electrocardiogram recorded on admission (Fig. 3) showed complete AV block with narrow QRS complexes. A temporary transvenous pacemaker was inserted and His bundle recordings were obtained. Right atrial pacing at increasing rates increased the AH interval but complete AV block persisted. His bundle pacing was unsuccessful. Intravenous atropine (1 mg) increased the atrial rate from 85 to 105 a minute, but the ventricular rate remained unchanged.

Case 3 This 85-year-old white man was admitted because of multiple syncopal episodes over a three-month period. A year before this admission the electrocardiogram had shown 2:1 AV block with a QRS pattern of right bundle-branch block. Electrocardiogram on this admission (Fig. 5) showed complete heart block with an atrial rate of 83 a minute and a ventricular rate of 34 a minute. The idioventricular complexes showed a pattern consistent with right bundle-branch block. Intravenous atropine (2mg) increased the ventricular rate from 34 to 39 a minute. The His bundle electrograms were recorded during the pacemaker insertion. Baseline His bundle electrograms are shown in Panel I of Fig. 6. Each atrial depolarization (A) is followed by an H at an AH interval of 90 msec. Every ventricular depolarization is preceded by an H' at an H'Q interval of 45 msec. No fixed relation exists between H and H'. The effect of atrial pacing at a rate of 145 a minute is shown in Panel II of Fig. 6. The AH interval has increased to 140 msec and the complete heart block persists.

In Fig. 7A and 7B the results of His bundle pacing are shown. Pacing was achieved by the catheter that initially recorded the His bundle electrogram. The atrial pacing catheter was used to record the right atrial electrogram (RAE). The His bundle pacing was performed at a fixed rate of 75 a minute. In Fig. 7A and 7B each QRS complex is preceded by a pacer impulse (PI) at a PIQ interval of 45 msec, which is similar to the H'Q interval recorded during spontaneous rhythm. In addition, QRS morphology of the paced beats is similar to the idioventricular complexes. This supports the validity of the His bundle pacing. Two pacer impulses, i.e. last in Fig. 7A and the one before last in Fig. 7B, are not followed by any ventricular activity (this will be discussed further).

In Fig. 7A, each of the first three pacer impulses is followed by a ventricular activity and a retrograde atrial deflection marked rA. This suggests that the pacing catheter is able to pace the His bundle completely so that both antegrade and retrograde conduction could occur. A sinus P comes exactly at the time of the fourth pacer impulse. A QRS follows the fourth pacer impulse at 45 msec interval but there is no retrograde atrial
FIG. 3 Case 2. A 12-lead electrocardiogram with rhythm strip showing complete AV block. QRS complexes are narrow.

FIG. 4 Case 2. His bundle electrograms showing complete heart block and presence of split His bundle potentials. Paper speed 150 mm/sec. Time lines 1 sec apart.

FIG. 5 Case 3. A 12-lead electrocardiogram showing complete heart block. The QRS morphology is consistent with complete right bundle-branch block.
activity because of the refractoriness of the AV node due
to the earlier sinus P wave. The last impulse falls about
90 msec after a sinus P wave and is not followed by a
ventricular depolarization. This is possibly due to the
refractoriness of the proximal His bundle segment which
was depolarized by the earlier sinus P wave. This finding
suggests that in order to achieve His bundle pacing, both
segments of the His bundle had to be in a nonrefractory
state at the time of the pacer impulse.

In Fig. 7B, the first three atrial depolarizations are of
sinus origin whereas the last four represent retrograde
activation of the atria (rA). The first two pacer impulses
come at intervals of 230 and 290 msec from the preceding
sinus atrial depolarizations. Both pacer impulses are
followed by the ventricular activity but no retrograde
atrial activity. This suggests that the proximal His
bundle segment has recovered from the previous atrial
depolarization and is no longer refractory to the pacer
impulse, but the AV node is still refractory and hence
there is no retrograde conduction to the atria. The third
pacer impulse comes 350 msec after the third sinus A
and is followed by a ventricular depolarization as well
as a retrograde atrial activity. It is clear that the further
away the pacer impulse is from a sinus A, the better
the chance of its being conducted forward and backward.
The fifth impulse is followed by an rA, but no ventricu-
lar complex. There are two possibilities to explain this
beat. It is possible that the fifth pacer impulse instead
of depolarizing the whole His bundle, just depolarized
the AV node and proximal His bundle segment and
hence only the rA could occur. The other possibility is
that the fifth pacer impulse, in fact, depolarized the
whole of the His bundle and the antegrade conduction
showed a Mobitz type II block in the left bundle-branch.

**Discussion**

Recent studies using His bundle electrograms have
shown the site of block to be distal to the His
bundle potential in the majority of patients with
chronic complete heart block (Narula et al., 1970a;
Steiner et al., 1971). Heart block occurring within
the main His bundle was first described by Narula
and Samet (1970). They demonstrated the presence
of double or split His bundle potentials which
signify the electrical activity of the proximal and
distal segments of the His bundle. Recently, others
have reported cases with various degrees of block
within the His bundle (Gupta et al., 1972a; Rosen
et al., 1971; Rosen et al., 1972; Schuilenberg and
Durrer, 1972). Pathological confirmation of this
interesting electrophysiological entity is not avail-
able.
The 3 cases presented here are among the 21 cases of chronic complete heart block studied in our laboratory during the past 2 years. We have previously reported 2 patients with Mobitz type II AV block within the His bundle (Gupta, Lichstein, and Chadda, 1972b). Schuilenberg and Durrer (1972) reported 4 cases of block within the His bundle: 2 patients had Mobitz type II AV block while the other 2 had complete heart block. While most of the patients reported appear to have arteriosclerotic or degenerative heart disease, this type of block has also been found with traumatic heart disease (Rosen et al., 1972) and congenital heart disease (Rosen et al., 1971). These data suggest that block within the His bundle may not be as uncommon as previously considered.

Block within the His bundle cannot be distinguished electrocardiographically from block within the AV node. In either instance, if the block is complete, the QRS morphology is of a supraventricular type, unless a prior bundle-branch block is present (Case 3 in the present report and Case 4 of Schuilenberg and Durrer (1972)). Though we have noted only Mobitz type II patterns in our cases of second-degree AV block, Wenckebach patterns have also been described (Narula and Samet, 1970).

Atrial pacing at increasing rates progressively increases the AH interval and is one of the methods of His potential validation (Damato et al., 1969). His bundle pacing is another method which has been suggested (Narula, Scherlag, and Samet, 1970a), but is not recommended by others (Rosen et al., 1972). In our experience, continuous His bundle pacing is difficult to achieve and it invariably results in the
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stimulation of the adjacent myocardium. However, if one is successful in pacing the His bundle, this strongly supports the validity of the His bundle potential. A right bundle potential can be separated from the distal His bundle potential by the shorter RBQ interval and the position of the recording catheter.

Unlike patients with AV nodal block, patients with intra-His block do not increase their ventricular rate adequately after intravenous atropine administration (Narula and Samet, 1971; Rosen et al., 1971, 1972). This is possibly because of the lack of autonomic control on the distal His bundle segment. In 2 of our patients, where atropine was used, the heart rate either did not increase or increased slightly.

In conclusion, complete AV block within the His bundle can only be localized with the aid of the His bundle electrogram. The electrocardiogram is of little help since the QRS complexes have a supraventricular morphology. Atropine usually does not increase the heart rate.

Miss Nancy McNamara, R.N., assisted during the procedures.

References

Addendum
Since completion of this manuscript we have seen another patient with complete AV block within the His bundle. This patient had previously shown Mobitz type II block within the His bundle (Gupta et al., 1972b).

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