His bundle rhythms with retrograde conduction to the atria

S. Gavrilescu and C. Luca
From the First Medical Clinic, Institute of Medicine, Timişoara, Rumania

His bundle electrograms were repeatedly recorded in two patients with complex arrhythmias. In the first case the pacemaker was located in the lower part of the left bundle-branch, retrogradely activating the His bundle. Atrial standstill was associated with this mechanism. After four days, His bundle tachycardia occurred, with constant retrograde activation of the atria, and anterograde block. In the second patient, who had progressive degeneration of the conducting system, His bundle rhythm activated the ventricles with a 2:1 block. There was synchronization between the pacemaker located in the His bundle, and the atria. As the disease progressed, dissociation between atrial, His bundle, and ventricular rhythms developed.

Concealed His bundle automatic activity may explain some complex arrhythmias which would remain unsolved if the His bundle potentials were not recorded.

The interpretation of atioventricular nodal rhythms may be considerably aided by the use of His bundle electrograms. As a result some traditional concepts assuming the formation of automatic impulses in the AV junctional area have had to be modified. Damato and Lau (1969) have provided evidence that in some so-called nodal rhythms, the His bundle and not the AV node is the pacemaker site. In some upper nodal rhythms, Touboul, Clément, and Delahaye (1971) were able to show an H potential preceding a retrograde p wave, and a prolonged HV interval.

In this report the value of His bundle electrograms in the analysis of complex arrhythmias arising in the His bundle and the fascicular system is presented.

Methods

The technique of His bundle recordings used in our laboratory has been reported in previous communications (Pop et al., 1972; Gavrilescu et al., 1973). His bundle potentials were recorded employing a tripolar electrode catheter inserted percutaneously through the femoral vein. Pacing at the site of electrical activity was used to validate His bundle potentials. Recordings were made on a 6-channel direct-writing electrocardiograph (NEK-2, Zwönitz, East-Germany), at a paper speed of 100 mm/sec.

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Case reports

Case I

A 65-year-old woman had mitral valvular disease and chronic congestive heart failure. She was known to be in atrial fibrillation and was treated with digitalis. On admission, the electrocardiogram revealed nodal rhythm (rate 51 a minute) and no detectable atrial activity (Fig. 1). The QRS complexes were 0.08 sec in duration and showed right axis deviation (+120°).

His bundle electrograms were recorded on two separate occasions. The first recording (Fig. 2) showed atrial standstill and a regular ventricular activity with a cycle length of 1150 msec. These ventricular complexes were followed by H potentials which appeared at 90 msec from the beginning of the ventricular depolarization. At each third or fourth beat, a different type of ventricular activity, preceded by an H deflection and an HV interval of 50 msec, occurred at a cycle length of 850–950 msec. There was no difference on the surface electrocardiogram in the QRS complexes, whether preceded or followed by the H potentials.

The serum electrolytes were normal and digitalis toxicity was suspected. The patient was treated with bed rest and spironolactone. After four days the heart rate was about 90 a minute, and the rhythm slightly irregular. Irregular atrial activity could be seen on the surface electrocardiogram (Fig. 3).

The intra-atrial electrogram revealed an atrial tachycardia (cycle length 500 msec), with no constant relation to ventricular complexes (Fig. 4A). The second His bundle recording (Fig. 4B) showed H potentials.
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**FIG. 1** Case 1. The electrocardiogram on admission.

**FIG. 2** Case 2: His bundle electrogram (AV) and lead I (DI): first recording. There is no atrial activity. VH are fascicular beats with retrograde activation of the His bundle. HV is a His bundle beat. The diagrams show the proposed origin for each complex.

**FIG. 3** Case 1. Lead V2 showing the irregular atrial activity, four days after the recordings shown in Fig. 1 and 2.
constantly preceding atrial activity by 50 msec. An exception is the second complex (VHA) from Fig. 4B which shows ventricular depolarization 90 msec before an H deflection; this was followed 50 msec later by an atrial activation. This electrogram resembles those recorded four days previously (Fig. 2). The third and fourth complexes (HAV) show H deflections 50 msec before atrial activation, and preceding ventricular depolarization by 100 and 130 msec. A His-Purkinje-system block of Wenckebach type can be assumed for the third, fourth, and fifth (HAV, HAV, HA) complexes shown in Fig. 4B. This is supported by the appearance of a wide QRS complex on the surface electrocardiogram associated with the prolongation of the HV interval from 100 to 130 msec, demonstrating the slowing of anterograde conduction along the bundle-branches. The sixth complex (VHA) is a fusion beat between fascicular and His bundle rhythm.

In this patient, who probably suffered from digitalis toxicity and who had atrial standstill, the pacemaker was initially located in the lower part of the left bundle-branch, activating the His bundle retrogradely at a slow rate. His bundle premature beats were also seen. The second recording showed His bundle tachycardia with retrograde activation of the atria and anterograde block of Wenckebach type. Escape beats from the left divisional pacemaker were also observed.

Case 2
A 32-year-old man with hypertrophic cardiomyopathy developed spontaneous atrioventricular block. Fig. 5 shows His bundle rhythm with a cycle length of 900 msec and 2:1 block. The ventricular complexes are preceded by an H deflection and a prolonged HV interval (140 msec) in the conducted beats. The QRS complexes show a left bundle-branch block pattern (0.13 sec). Atrial activity (A) shows the same cycle length (900 msec) and synchronization of H and A depolarization by 'accrochage.' The injection of 0.25 mg orciprenaline, a beta-adrenergic stimulating drug, shortened the cycle length of the His bundle rhythm to 60 msec. The anterograde block was unchanged, but retrograde activation of the atria occurred 60 msec after each H deflection (Fig. 6). The retrograde conduction to the atria is demonstrated by the negative P' waves in lead II and III, in contrast with the positive P waves seen in Fig. 5.

A further shortening of the cycle length of the His bundle rhythm (520 msec) under the effect of orciprenaline was associated with an increase of anterograde block to 3:1, while H depolarizations and atrial activity were incompletely dissociated (Fig. 7). There were isolated H potentials, blocked anterogradely and retrogradely, as well as retrogradely-conducted H deflections (HA) and His bundle echoes (HVH'). Isolated capture beats (AHV) were also recorded.
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**Fig. 5** Case 2. His bundle electrogram (HBEG) and leads I, II, and III. The His bundle rhythm shows a 2:1 block and an HV interval of 140 msec in the conducted beats. The H deflections are followed at 50 msec by atrial activity (A). 'Accrochage' is the proposed mechanism for explaining synchronization (see text for details). The P wave is positive in leads I, II, and III.

**Fig. 6** Case 2. His bundle recording after the injection of orciprenaline. The cycle length of His bundle rhythm shortens from 900 msec (Fig. 5) to 600 msec. The H deflections are followed at 60 msec, by retrograde P' waves, which are negative in the peripheral leads.
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FIG. 7  Case 2. A further shortening of the cycle length of His bundle rhythm, increases anterograde block to 3:1. The relations between His bundle activity, atrial, and ventricular beats are shown on the upper diagram. There are anterograde conducted, retrograde conducted, and blocked H potentials. AHV is an isolated capture beat. HVH' is a His bundle echo.

Three months later a second recording showed complete heart block with idioventricular rhythm (cycle length: 1410 msec, QRS duration: 0.14 sec) with retrograde activation of the His bundle. The VH' interval was 100 msec. There was complete dissociation between atrial and ventricular activity, and isolated blocked H potentials were also present (Fig. 8).

To summarize, in this patient with progressive disease of the AV conducting system the pacemaker was located in the His bundle activating the ventricles with a 2:1 block. Synchronization between His bundle depolarization and atrial activity could be observed: by 'accrochage' at a longer cycle length, and, under the influence of orciprenaline, by retrograde conduction. With the progress of the disease idioventricular rhythm appeared, with retrograde activation of the bundle of His. Complete dissociation between atria and ventricles occurred. Isolated, blocked H deflections were also present.

Discussion

The existence of His bundle rhythms as well as the localization of ectopic activity within the bundle of His and its fascicles have been well documented (Damato and Lau, 1969; Massumi 1970; Massumi, Ertem, and Vera, 1972). Spontaneous His bundle extrasystoles conducted retrogradely to the atria, simulating nonconducted atrial premature beats or atrioventricular block, were described in the dog (Damato, Lau, and Bobb, 1971) and in man (Cannom et al., 1972; Eugster et al., 1973). The presence of narrow or wide QRS complexes during such rhythms can be explained by the site of impulse formation, aberrant conduction, and the speed of conduction in the fascicular system.

In the cases now reported, His bundle rhythm occurred under different circumstances. In the first patient there was a depression of the automatic activity, probably due to digitalis toxicity. Atrial standstill and left lower fascicular rhythm with slow retrograde activation of the His bundle were present on admission. After some days an enhancement of His bundle automatic activity took place. His bundle tachycardia occurred (120 a minute) with anterograde block and retrograde activation of the atria. It is possible that in some cases where the atrial activity returns to 'sinus rhythm' after long standing atrial fibrillation (Saint-Pierre and Perrin, 1973) there is in fact a concealed His bundle rhythm with retrograde activation of the atria.

The second case had progressive determination of the conducting system. The ventricles were driven for a time by a pacemaker arising in the His bundle. This mechanism was described by Narula et al. (1970). The importance of our recordings resides in the synchronization between H potentials and atrial activity. The mechanisms of synchronization of atria and ventricles have been studied in clinical (Marriott, 1956) and experimental (Levy and Edelstein, 1970) conditions. As far as we know, this has not yet been demonstrated between His bundle and atrial activity.

The mechanism of synchronization between His bundle and atrial depolarizations was related to
FIG. 8 Case 2. His bundle electrogram (HBEG) and leads I, II, III, second recording. Idio-ventricular rhythm with retrograde activation of the His bundle (VH') is the dominant mechanism. A: atrial activity, H: His bundle depolarizations (see text for details).

the cycle length of the His bundle rhythm. At a longer cycle length ‘accrochage’ was assumed to be the mechanism, while at shorter cycle lengths, under the influence of orciprenaline, retrograde activation of the atria took place.

On the basis of work with amphibian hearts Segers (1946) called ‘accrochage’ the process of synchronization which occurred when independent fragments of myocardium were placed in close contact. An electrical influence exerted by one tissue on the other may be responsible for this phenomenon. It is different from the capture of the heart chamber by retrograde conduction.

An alternative explanation for the two types of P waves and HP relations found in our patient can be the activation of different ventriculoatrial pathways (Waldo et al., 1970).

The retrograde conduction from the ventricles to the atria is an infrequent event during atioventricular block (Scherf, Cohen, and Orphanos, 1964). Gupta and Haft (1972) studied ventriculoatrial conduction in a patient with complete heart block, using His bundle electrography. It was concluded that retrograde conduction could occur if the block was beyond the bundle of His and the AV node was intact. The retrograde pathway was supposed to be by muscular conduction. It was also suggested that retrograde P' waves were not the result of an impulse reaching the atria through the ventricular specialized system, but were a consequence of mechanical or electrotonic effects of ventricular contraction on a latent atrial focus (Mirowski and Tabatznik, 1970). However, our findings suggest that the ventricular impulses pass through the His bundle during its pathway towards the atria (Castillo and Castellanos, 1971).

The effect of orciprenaline, a drug that enhances the atioventricular conduction, on the synchronization of His bundle and atrial activity is also of interest. Under its influence ‘accrochage’ was replaced by retrograde conduction. It is possible that the drug influenced the anterograde and retrograde pathways in different positions.

Concealed His bundle activity may explain some complex dysrhythmias which would remain unsolved if His bundle electrograms were not performed.

References
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Requests for reprints to Professor S. Gavrilescu, str. Feuerbach 10, Timişoara, Rumania.