His Bundle Electrocardiography during Bidirectional Tachycardia

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SUMMARY

His bundle electrocardiography in a patient with a digitalis-induced bidirectional tachycardia revealed absence of His spike preceding earliest onset of ventricular activation during the bidirectional tachycardia. However, His potential preceded ventricular activation of normal complexes at a constant H-V interval of 35 msec. The presence of fusion QRS complexes, capture QRS complexes, the analysis of cycle lengths, and response to carotid sinus massage all favor a ventricular origin for this tachycardia in the patient presented.

Additional Indexing Words:
Digitalis toxicity  Aberrancy  Ventricular tachycardia

BiDIRECTIONAL tachycardia is an uncommon rhythm disturbance, characterized by QRS complexes with a right bundle branch block, alternating polarity in the frontal plane, and a regular rhythm. It is most often seen in a setting of digitalis intoxication and significant myocardial disease. However, since the first description in man by Schwensen in 1922,1 the mechanism and site of origin for this arrhythmia has remained controversial. Whereas earlier reports2-4 favored a ventricular locus, subsequent authors including Scherf and Bornemann5 and Castellanos7 have suggested that several different etiologic mechanisms are possible. More recently, Rosenbaum, Elizari and Lazzari6 proposed that bidirectional tachycardia has a supraventricular origin and is actually a form of functional trifascicular block: right bundle branch block, and alternating left anterior and left posterior hemiblock. This paper reports a patient with bidirectional tachycardia who was studied by His bundle electrocardiography.6 In this instance the absence of His bundle activity preceding the earliest onset of ventricular activation strongly favored a ventricular origin for the ectopic rhythm.

Report of a Case

A 69 year old Caucasian man was admitted to the hospital in a semicomatose state, having suffered a cerebral vascular accident. Physical examination revealed blood pressure 130/80 mm Hg, an irregular apical pulse of approximately 160/min, temperature 98.8°F and respirations 30-40/min. No jugular venous distention was present and there were no carotid bruises. Bi-basalar rales were noted. Cardiac examination demonstrated the apical impulse to be 14 cm lateral to the midsternal line in the 5th intercostal space; an S4 gallop was audible at the apex. The liver was not enlarged; 2+ pretibial and ankle edema were present. Neurological examination revealed a left hemiparesis with a left central facial palsy and dystarthric speech.

The following laboratory data were normal: hemoglobin, hematocrit, serum electrolytes, blood urea nitrogen (BUN), PBI and T3. The blood sugar was 113 mg/100 ml. Arterial blood gas analysis revealed pH 7.49, PO2 66 mm Hg, and PCO2 33 mm Hg. Chest X-ray demonstrated cardiomegaly, pulmonary venous congestion, and a small right pleural effusion. Initial ECG showed atrial fibrillation with an irregular ventricular response of 160/min and nonspecific ST-T wave changes. During the first 6 hours following admission, the patient received a total of 1.25 mg of digoxin intravenously, and by the following morning, he was noted to have a bidirectional tachycardia at a rate of 170/min (fig. 1). Digitalis therapy was discontinued. Family members now revealed that he had been taking 0.15 mg of digitoxin daily for the past several months. Immunoassay determination for digitoxin10 and digoxin11 on blood drawn at that time subsequently revealed the levels to be 22 ng/ml and 6.4 ng/ml, respectively. Although these figures may be somewhat elevated by cross reactivity between the two digitalis preparations, the serum digoxin level is compatible with the clinical impression of digitalis intoxication.12

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Figure 1

Twelve lead ECG recorded immediately prior to His bundle study revealed a bidirectional tachycardia at a rate of 170/min. Mean frontal plane QRS axis alternated between -60° and +130° with successive beats, and all complexes demonstrated a RBBB pattern in V1. Paper speed 25mm/sec.

Because the patient tolerated the tachycardia well without evidence of further cardiovascular decompensation, it was decided to perform His bundle electrocardiography. Tracings were recorded on a multichannel oscillographic photographic recorder (Electronics for Medicine DR8) at filter settings between 40 Hz and 500 Hz and paper speeds of 50 mm/sec or 100 mm/sec. We used a tripolar His catheter (USCI #5655) with ring electrodes 2 mm wide which were separated by 10 mm (distal and middle rings) and 6 mm (middle and proximal rings).

Results

A right atrial electrogram confirmed the presence of atrial fibrillation (not shown). The bidirectional tachycardia recurred intermittently for a few systoles as well as for long paroxysms (fig. 2). On occasion, intermediate forms of QRS distortion appeared, and were considered to be fusion complexes. Ventricular depolarizations which occurred early and had a normal QRS contour were thought to represent capture of ventricular activation by an atrial impulse (fig. 2).

During normal ventricular conduction, when the bidirectional tachycardia terminated for brief periods, a His potential preceded each ventricular electrogram at an H-V interval of 35 msec (fig. 3). Each time the bidirectional tachycardia occurred, a His spike no longer anteceded the onset of ventricular depolarization. In some of the anomalous complexes a His spike could be recorded just prior to the inscription of the local ventricular electrogram, but after the onset of the QRS complex (fig. 3). Despite the presence of digitalis excess, the ventricular response to the fibrillating atria was rapid during periods when the bidirectional tachycardia transiently ceased.

If ventricular aberration of a supraventricular impulse, owing to functional bundle branch block, were the cause of bidirectional tachycardia, then an analysis of cycle lengths should reveal aberrant conduction present after shorter cycles, and normal conduction following longer cycles. Figure 3 illustrates that, in fact, the opposite occurred. Longer cycles were terminated by QRS complexes which had a right bundle branch block and alternating polarity. These anomalous systoles were not preceded by a His potential, while a normal QRS initiated by a His spike terminated shorter cycles. During supraventricular origin with functional aberration, one would expect the fourth and fifth QRS complexes to be normal, and the other QRS complexes to be aberrant. Therefore, cycle length analysis provided additional evidence against supraventricular origin with functional aberrancy of intraventricular conduction.

The bidirectional tachycardia was not always precisely regular; the rate ranged between 170/min and 200/min. On four occasions when the rate fell to 150/min or below, the alternating polarity of the axis in the frontal plane ceased, and all complexes maintained left axis deviation and right bundle branch block (fig. 4).

The onset of the bidirectional tachycardia, following at least two normal complexes, was recorded 150 times. The coupling interval between the last normal QRS and initiating QRS of the bidirectional tachycardia ranged between 350-500 msec. The first QRS of the bidirectional tachycardia showed left anterior hemiblock on 125 occasions, and normal axis on 25 occasions.

During the study, carotid sinus massage consistently decreased the ventricular response to the atrial fibrillation, whereas carotid sinus massage
Figure 2

Fusion and capture complexes during bidirectional tachycardia. Second and ninth complexes demonstrated intermediate degrees of QRS distortion in the surface ECG, and were preceded by a His deflection at a short H-V time (fusion QRS complexes, F). The last systole occurred early, had a normal QRS morphology, and was preceded by a His potential with a normal H-V interval (capture, C). Note first QRS complex had a right bundle branch block with a normal frontal axis, probably representing equal or no conduction delay in both fascicles of the left bundle branch. Irregular deflections in baseline may be artifact due to slight catheter movement or, more probably, represent impulses recorded intermittently from the fibrillating atria. II, standard surface ECG lead II; V_1, standard surface ECG lead V_1; BHE_1, BHE_2, simultaneously recorded bipolar bundle of His electrograms; H, His potentials. Paper speed 50mm/sec.

Discussion

Although the ventricular origin for bidirectional tachycardia has been suggested previously on the basis of observations made from surface electrocardiography, our report documents His bundle activity during such a tachycardia. The absence of a His deflection preceding the earliest onset of ventricular activation during the bidirectional tachycardia, and the presence of a His deflection preceding the normal QRS complexes conducted from the fibrillating atria, clearly place the origin of the rhythm distal to the His bundle recording site. Since we did not attempt a pacing confirmation of the recording site, an objection could be raised that the His catheter electrode actually recorded only a right bundle potential. Right bundle activation would not be expected to precede the ventricular electrogram during the right bundle branch block that accompanied the bidirectional tachycardia. However, when simultaneously recording between two different pairs of bipolar leads, it would be unlikely to record only a right bundle branch deflection without also recording a second deflection indicating His activation. This is particularly true since we repeatedly explored the area near the His bundle by recording from numerous catheter positions to register the best possible His potential. A second deflection representing right bundle activation was not seen. Once an adequate His spike was recorded, the catheter was not disturbed. A second objection might be that catheter movement prevented us from recording a His potential during the bidirectional tachycardia. This appears even more unlikely since the catheter position remained stable throughout the procedure. We consistently recorded a His potential during normal ventricular conduction and failed to record a His potential during abnormal ventricular conduction.
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The first three complexes were conducted from the fibrillating atria with a His-to-ventricle (H-V) time of 35 msec. Apparent variations in H-V interval were due to changes in the recording of ventricular septal depolarization. The H-V interval in the normal complexes was constant when measured to the onset of QRS in leads II and V_1. The fourth and fifth complexes demonstrated a RBBB pattern in V_1 and alternating QRS polarity in lead II characteristic of bidirectional tachycardia. A His spike preceded the ventricular electrogram of the fifth systole, but occurred after the earliest onset of ventricular activation as recorded in leads II and V_1, indicating that His depolarization was most likely retrograde. His deflection was probably buried within the inscription of the local ventricular electrogram of the fourth QRS. Note that anomalous QRS complexes terminated longer intervals, while normal QRS complexes terminated shorter intervals. These groupings are incompatible with ventricular aberration of a supraventricular impulse. Numbers indicate cycle lengths in msec. Conventions as in figure 2. Paper speed 100 mm/sec.

Despite cycle length changes for both rhythms. In addition, during some of the anomalous complexes we recorded a retrograde His potential which occurred after the onset of the QRS but prior to the inscription of the local ventricular electrogram, thus proving that the catheter was adequately positioned.

Since each ventricular complex during the bidirectional tachycardia had a right bundle branch block pattern, and was not preceded by a His deflection, it would appear that this arrhythmia originated in the left side of the ventricular conducting system. Furthermore, since the QRS axis alternated between -60° and +130° in successive...

Figure 3

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Figure 4

Persistent left anterior hemiblock and RBBB when the ventricular rate was about 150/min. Next to last QRS complex is a fusion (F). Conventions as in figure 2. Paper speed 100 mm/sec.
beats, during rates greater than 150/min, a focus in the main left bundle branch with alternating functional left anterior hemiblock and left posterior hemiblock would seem most likely. When the rate slowed to less than 150/min, the left axis remained constant, suggesting uninterrupted conduction down the posterior fascicle and persistent block in the anterior fascicle. In addition, the initiating QRS complex of the bidirectional tachycardia followed a pause in the normal rhythm and demonstrated right bundle block and left anterior hemiblock over 80% of the time. These observations are consistent with data suggesting that the posterior division of the left bundle branch exhibits a shorter refractory period duration than the anterior division of the left bundle branch.8

One of the several alternative suggestions has postulated two separate ventricular foci, one focus located in the posterior division of the left bundle producing a left anterior hemiblock pattern and the other focus located in the anterior division of the left bundle producing a left posterior hemiblock pattern.13 The fairly regular cycle lengths and loss of the posterior hemiblock pattern when the rate slowed makes this explanation unlikely.

Our study does not establish a ventricular etiology for all cases of bidirectional tachycardia; and indeed, bidirectional tachycardia may only be a descriptive term for a heterogeneous group of cardiac arrhythmias which have a similar electrocardiographic configuration. However, we do feel that we have demonstrated that the ectopic locus in the patient herein reported was distal to the bundle of His, and the work of others14,15 further suggests that a ventricular origin for bidirectional tachycardia may not be uncommon.

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