Supraventricular Origin of Bidirectional Tachycardia

Report of a Case

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SUMMARY
We have confirmed a supraventricular origin of bidirectional tachycardia with simultaneous standard electrocardiograms and a His bundle electrogram. The patient had sinus rhythm, variable trifascicular block and atrioventricular block below the His bundle. Ventricular excitation varied among three patterns which included right bundle branch block with right axis deviation (RBBB-RAD), right bundle branch block with left axis deviation (RBBB-LAD), and right axis deviation without right bundle branch block (RAD). Sinus beats with alternating patterns of RBBB-RAD and RBBB-LAD resulted in bidirectional tachycardia as did sinus beats with alternating patterns of RBBB-LAD and RAD. Our observations prove that bidirectional tachycardia may be initiated by a supraventricular pacemaker and that the distinguishing bidirectional quality of this arrhythmia need not always occur with constant right bundle branch block.

Additional Indexing Words:
Trifascicular block
His bundle

The mechanism of bidirectional tachycardia has remained controversial since its first description in 1922. A ventricular origin of this arrhythmia has recently been proven with data collected in our laboratory and that of others. A supraventricular origin of bidirectional tachycardia has also been suggested.

Normal ventricular activation requires ordered excitation over three intraventricular pathways, namely, the right bundle branch, anterior division of the left bundle branch, and posterior division of the left bundle branch. Abnormalities of ventricular excitation are commonly attributed to disturbed conduction in at least one of these pathways. The patient herein reported presented with intrinsic abnormal conduction in each of the three major pathways, a disorder commonly referred to as trifascicular block. The pathways of prolonged or blocked conduction changed intermittently and at times alternated from beat to beat, thereby resulting in an electrocardiographic pattern of bidirectional tachycardia with rightward and leftward axis shifts in the frontal plane. Alternating rightward and leftward axis shifts were associated with inconstant right bundle branch block. In addition, intermittent atrioventricular block was recorded.

In a discussion of the mechanism of bidirectional tachycardia (BT) in 1969, Rosenbaum, Elizari, and Lazzari stated, “Because it is a trifascicular block, some variants in the regular pattern of BT are to be expected. For instance should the conduction impairment become more important in the three fascicles, A-V conduction disturbances ought to occur... In addition, should the conduction disturbances be less important within the right bundle branch, the beats of BT might occur without RBBB... Although we cannot exemplify these situations, the hypothesis predicts them, and we hope they will be seen in future cases when looked for.”

The documentation of a sinus node origin of a bidirectional tachycardia which was also characterized by trifascicular block, inconstant right bundle branch block, and intermittent high grade atrioventricular block support the hypothesis and prediction of Rosenbaum and co-workers.

Clinical Summary
A 77-year-old male was admitted to the Beth Israel Hospital with the acute onset of intermittent atrioventricular heart block, fever, and jaundice. The past cardiac history included coronary artery disease with varying degrees of expression such as angina pectoris six years prior to admission, prolonged myocardial ischemia five years prior to admission, and a subendocardial myocardial infarction four years prior to admission. Relevant past cardiac history also included intermittent mild hypertension, which had not
required therapy for six months. The patient was not taking any medications at the time of admission.

Physical examination revealed a jaundiced man. The blood pressure was 180/100 mm Hg. The apical pulse varied from 90 to 140 beats/min and was intermittently irregular. The respiratory rate was 18 per minute. The oral temperature was 102.4°. Examination of the neck revealed a slight increase in jugular venous distension. Cardiac examination revealed a palpable point of maximal impulse in the fifth intercostal space, 2 cm lateral to the midclavicular line. There were no murmurs or thrills. The bases of the lungs had bilateral inspiratory rales. The abdomen was soft and nontender. The liver was palpable 6 cm below the right costal margin. The extremities did not have evidence of edema or phlebitis.

Laboratory data revealed hematocrit, 45%; white blood count, 16,500/mm³; blood urea nitrogen, 20 mg/100 ml; sugar, 120 mg/100 ml; sodium 146 mEq/L; K, 3.5 mEq/L; Cl 120 mEq/L; and CO₂ 20 mEq/L. Total bilirubin 4.4 mg/100 ml with a direct fraction, 1.0 mg/100 ml; alkaline phosphatase, 51.5 King Armstrong Units. The chest X-ray revealed cardiomegaly and pulmonary venous redistribution to the upper lobe vessels.

Shortly after admission the patient had a transvenous pacemaker placed through a transfemoral venous approach at which time a His bundle study was obtained. A permanent transvenous pacemaker (Cordis-Ectocor) was implanted on the twelfth hospital day.

The fever and jaundice were the result of cholelithiasis which had sporadically caused symptoms over a five year period. The abdominal X-ray demonstrated calcified densities in the right upper quadrant which were compatible with gall stones. Parenteral ampicillin was instituted for treatment of cholecystitis. Cholecystectomy and common duct exploration were performed on the seventh hospital day. The patient was discharged in satisfactory condition on the eighteenth hospital day.

Electrocardiograms

Selected samples of the preadmission electrocardiograms

![Figure 1](image)

Varied patterns of ventricular conduction noted prior to the appearance of trifascicular block. 8-24-67 left bundle branch block; 9-27-67, incomplete left bundle branch block; 3-13-72 right bundle branch block, axis “95°.”

![Figure 2](image)

Patterns of right bundle branch block right axis deviation (RBBB-RAD) and right bundle branch block left axis deviation (RBBB-LAD). A) Transition from RBBB-RAD to RBBB-LAD. B) Bidirectional tachycardia of sinus origin composed of alternating RBBB-RAD and RBBB-LAD complexes. The arrow indicates a break in the sequence.

![Figure 3](image)

Two patterns of bidirectional tachycardia of sinus origin. Initially bidirectional tachycardia is composed of alternating RBBB-RAD and RBBB-LAD complexes. There is a sudden nonconducted P wave (arrow) between two RBBB-RAD complexes after which the bidirectional tachycardia is composed of alternating RBBB-LAD and RAD complexes. A-V block (arrow) enhances identification of P waves.
times, the pattern of bidirectional tachycardia during sinus rhythm consisted of a beat-to-beat alternation between RBBB-LAD and RAD (fig. 3). Each period of bidirectional tachycardia was brief, with none lasting more than several minutes.

The His bundle study was performed by the technique of Scherlag et al.\textsuperscript{8} and was validated by His bundle pacing\textsuperscript{9, 10} which perpetuated an excitation pattern of RBBB-LAD and which preserved a pacing stimulus — V interval which was equal to the H-V interval. Graphs were recorded on a multichannel Electronics for Medicine Oscilloscopic Recorder. His bundle study defined the infra His location of A-V block (fig. 4), confirmed the supraventricular origin of the three patterns of ventricular excitation, and defined the A-V node (A-H interval) and His-Purkinje conduction time (H-V interval) of each type of ventricular excitation (fig. 5). The characteristics of the three patterns of intraventricular conduction are summarized in table 1. The patterns of RAD and RBBB-RAD had the same A-V node conduction time of 137 msec (normal A-H time = 50-120 msec) and His-Purkinje conduction time of 68 msec (normal H-V time = 35-50 msec). The RBBB-LAD pattern also had an A-V node conduction time of 137 msec but had a longer His-Purkinje (95 msec), which was reflected (in the standard electrocardiogram) by a slightly longer P-R interval.

**Discussion**

Bidirectional tachycardia has been a well established, although infrequently observed entity in man since its first description by Schwensen in 1922.\textsuperscript{1} A clear understanding of this arrhythmia has not yet emerged. The two explanations that have received the most attention are a supraventricular origin with aberrant ventricular conduction, and alternatively, ectopic impulse information within the left ventricle.

Indisputable evidence of a left ventricular focus of bidirectional ventricular tachycardia has recently been reported.\textsuperscript{2-4} However, a supraventricular origin of bidirectional tachycardia has been supported by reports with inconclusive electrocardiographic data\textsuperscript{11-13} or tenuous relationships such as the occasional cessation of bidirectional tachycardia with vagal maneuvers\textsuperscript{14-16}.

Rosenbaum et al.\textsuperscript{5} advanced a thesis, unsupported by conclusive example, that bidirectional tachycardia is of supraventricular origin with continuous aberrant conduction in a pathological right bundle branch and alternating right and left axis shifts that result from alternate aberrant conduction in two pathologically damaged divisions of the left bundle branch system. From their personal experience and review of the literature, Rosenbaum and co-workers\textsuperscript{5} believed that
Table 1

Patterns of Intraventricular Conduction

<table>
<thead>
<tr>
<th>QRS Pattern</th>
<th>QRS Duration (msec)</th>
<th>A-H (msec)</th>
<th>H-V (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBBB-RAD</td>
<td>165</td>
<td>137</td>
<td>68</td>
</tr>
<tr>
<td>RBBB-LAD</td>
<td>165</td>
<td>137</td>
<td>95</td>
</tr>
<tr>
<td>RAD</td>
<td>199</td>
<td>137</td>
<td>68</td>
</tr>
</tbody>
</table>

Table 1: Patterns of Intraventricular Conduction

all abnormal bidirectional beats had a right bundle branch configuration. However, as previously noted in the introduction to this paper, they also believed that the pattern of tachycardia could predictably vary.

The electrocardiographic alterations found in our patient have not been described previously. They represent an indistinct example of a supraventricular (sino-atrial) origin of bidirectional tachycardia in which the bidirectional pattern resulted from beat-to-beat alteration in abnormal trifascicular conduction. Previous reports have recorded only intermittent change rather than beat-to-beat alternation in the context of a sinus mechanism and trifascicular block.17-18 The electrical events illustrated in our report evidence trifascicular delay and block as follows: A past history of bilateral bundle branch block with first degree block (fig. 1), intermittent right and left axis shifts during RBBB (fig. 2), beat-to-beat right and left axis shifts during bidirectional tachycardia (figs. 2, 3), documented A-V block below the His bundle (fig. 4), and the presence of prolonged His-Purkinje conduction time (H-V) during patterns of RBBB-LAD and RBBB-RAD (fig. 5). The abnormally prolonged conduction times of the left anterior and left posterior fascicles were characterized by His bundle study. When two conducting pathways are blocked or delayed such as in RBBB-RAD and RBBB-LAD, the H-V time represents conduction time in the remaining least affected pathway.19-22 During RBBB-RAD, the His-Purkinje conduction time of 68 msec in our patient indicated abnormally prolonged conduction in the left anterior division. When the pattern abruptly changes to RBBB-LAD, the even longer conduction time (95 msec) of the posterior division was unmasked.

It is of interest to speculate on the nature of intraventricular conduction during RAD. There have been some examples of block in the left anterior fascicle that obscured simultaneous right bundle branch block.23 These co-existing abnormalities are identified, in part, by a QRS duration which is unusually wide for uncomplicated block of the left anterior fascicle. Our pattern of RAD is extremely wide for pure block of the left posterior fascicle. Perhaps the unusual RAD pattern resulted from co-existing delays or blocks of the left posterior fascicle and the right bundle branch, with abnormal conduction in the posterior fascicle being far more pronounced than abnormal conduction in the right bundle branch.

The subject of this report had a clinical course which was unlike that of most patients with bidirectional tachycardia. The common clinical features of bidirectional tachycardia have recently been reviewed.5, 8 This arrhythmia occurs in the setting of severe cardiac disease. Atrial fibrillation is present in 44% of cases, digitalis was administered in 82% of cases and in many patients was implicated as the agent which produced the arrhythmia. Bidirectional tachycardia has a poor prognosis. Seventy percent of patients die within days and only 15% of patients survive more than 12 months. In contrast, our patient had enough cardiac reserve to sustain him through the stress of sepsis and major surgery. He continues to do well more than one year following permanent pacemaker placement and as such is following the course of the majority of patients that have progressive intraventricular conduction disturbances which result in high grade atrio-ventricular block.

Rate-dependent functional atrioventricular block within the His-Purkinje system has been noted in man.24 In our patient the intermittent and alternating changes in ventricular conduction did not appear to be rate related. Changes in intraventricular conduction were noted within a range of 93 and 134 beats per minute. It must be emphasized that our observations occurred in the setting of trifascicular block, a condition in which the sudden blocking and unblocking of pathways is poorly understood. Sudden alteration of bundle branch conduction has occurred within a narrow range of heart rates,25, 26 over a wide range of heart rates,27 and without relation to heart rate (fig. 5).28 The demonstration that oxygen could reverse bundle branch block29 has bolstered the view that anoxia can hamper conduction in pathways that are capable of conduction only under the most favorable of circumstances. It is possible that our patient’s ischemic heart disease, variable heart rate, and fluctuating fever all combined to upset the balance of favorable conditions in pathways with marginal conduction capabilities.

Our data establishes a firm support for a supraventricular origin of bidirectional tachycardia. The evidence also fulfills the predictions that right bundle branch block pattern need not be a constant requirement of bidirectional tachycardia of supraventricular origin and that A-V block may co-exist with bidirectional tachycardia.

Acknowledgment

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