CASE REPORTS

Ventricular Origin of Bidirectional Tachycardia

Case Report of a Patient not Toxic from Digitalis

By John A. Kastor, M.D., and Bruce N. Goldreyer, M.D.

SUMMARY
Electrophysiological, hemodynamic and angiographic studies were performed on an 18 year old woman with asymptomatic recurrent bidirectional tachycardia of eight years' duration and mild hyperkalemic periodic paralysis. Multiple surface electrocardiograms and His bundle and right atrial electrograms revealed that 1) all beats during bidirectional tachycardia originated in the left ventricle; 2) the two forms of ventricular beats in the bidirectional tachycardia had qR patterns in lead V_1 (suggesting left ventricular origin) and axes in the frontal plane of -75° and +110°; 3) the interectopic interval of the two forms of QRS in bidirectional tachycardia was relatively fixed at 440-470 msec; 4) the runs of bidirectional tachycardia always terminated with the beat with right axis deviation; 5) fusion between sinus beats and one of the two forms of ventricular beats produced multiform complexes observed on the electrocardiogram; 6) the sinus beats showed T wave abnormalities and their A-H and H-V intervals were normal. Lidocaine and atrial pacing at an interval of 500 msec each separately suppressed the arrhythmia. Administration of potassium chloride to a blood level 5.4 mEq/liter and of procainamide did not affect the arrhythmia. Hemodynamic, left ventricular and coronary angiographic studies were normal. Bidirectional tachycardia in this case originated in the left ventricle and was unassociated with digitalis intoxication or demonstrable nonelectrical cardiac pathology.

Additional Indexing Words:
Hyperkalemic periodic paralysis  Aberrant conduction  His bundle electrogram

Bidirectional tachycardia (BT) is a rapid regular rhythm in which QRS morphology alternates so that the direction of the complexes is predominantly upward in one beat and downward in the next. The rate is usually between 140 and 180 beats/min; QRS duration may be equal to, less than or greater than 0.12 sec; a right bundle branch type form is seen in lead V_1; and the axes in the frontal plane of the two types of beats are approximately -60 to -80 degrees and +120 degrees.  

The origin of the disturbance has long been debated. Levy and Lewis first suggested from experiments in cats that two independently firing ventricular pacemakers were operating. In early clinical reports Schwensen concluded that the rhythm was ventricular in origin, whereas Felderbaum proposed that the locus might be in the atrioventricular (A-V) node or main bundle with alternating conduction through right and left bundle branches. A variation on the supraventricular concept was recently advanced by Rosenbaum, Elizari and Lazzari who observed that in each of their 13 patients the electrocardiograms showed the pattern of right bundle branch in the chest leads with alternation of the axis in the frontal plane from left to right. They concluded that bidirectional tachycardia is "... a supraventricular tachycardia with permanent aberrant conduction in the right bundle branch and alternant aberrant

From the Departments of Medicine, University of Pennsylvania School of Medicine and the Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

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Address for reprints: Dr. John A. Kastor, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

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Multiple lead surface electrocardiogram during bidirectional tachycardia. The arrhythmia is displayed simultaneously in leads V₁, V₂, V₃, aVR, aVL, aVF, V₄, V₅, and V₆. The initial beat is sinus followed by a ventricular beat and then a regular sinus rhythm. The latter could be aligned almost perfectly with lead V₁ appearance. Figure 1.

Figure 2. Unipolar chest leads during bidirectional tachycardia. Lead 2 is displayed with the 6 chest leads for 5 beats of bidirectional tachycardia and a sinus beat. Three separate recordings were combined to produce each figure with V₁, V₂, and V₆ used as references for each tracing. Paper speed 30 mm/sec. 

Clinical Report

D.S. is an 18-year-old woman whose physician first noted an irregular cardiac rhythm at age 16. The electrocardiogram taken then showed the same arrhythmia which has been consistently present to this time (Fig. 1 and 2). She was never aware of palpitations. Her physical examination revealed no abnormalities. Her heart rate usually ran between 120 and 130 beats per minute. The first heart sound was normal and the second was of normal intensity. The blood pressure was 120/60 mm Hg. Her chest X-ray was normal. No suppressed the arrhythmia completely although diphenylhydantoin decreased its frequency.

KASTOR, GOLDNER

We have recently had the opportunity to test this hypothesis in a patient with hyperkalemic periodic paralysis associated with hyperkalemia as a result of this same arrhythmia. Electrocardiogram during hyperkalaemia showed that all ectopic beats originated in the left ventricle and not in supraventricular junctional tissues.
Arrhythmic drugs because of their real or presumed side effects and because she was virtually asymptomatic despite the constant presence of the cardiac arrhythmia. Only two potentially serious events have occurred. Several years ago she became lightheaded on one occasion without losing consciousness during a severe respiratory infection and at another time lost consciousness for several seconds upon leaving a crowded warm room. Since her pulse was not taken on either occasion, we cannot establish the relation of these events to her arrhythmia. While observed by us for the past 12 months, no similar episodes have been reported by the patient who is currently attending college and leading an active life.

The manifestations of her hyperkalemic periodic paralysis were first noticed at 18 months of age when she was observed to “drag her legs.” The weakness was never severe and did not interfere with normal growth and activities. The patient takes acetazolamide 500–750 mg/day resulting in a noticeable decrease in her attacks of weakness. A detailed evaluation of this aspect of her problem was undertaken in 1969 and has recently been reported.5

With development of techniques for specialized intracardiac recording and stimulation, a full electrophysiological and hemodynamic investigation seemed indicated to characterize more accurately the nature of the arrhythmia and to discover the presence of any associated cardiac structural abnormalities. The procedure was discussed in detail with the patient and her parents who readily gave informed consent, and the study was conducted on June 23, 1972.

Methods

The patient was brought to the laboratory in the fasting state. A #6 tripolar electrode catheter was introduced percutaneously into the right femoral vein and positioned under fluoroscopic and electrocardiographic guidance to record deflections from the bundle of His.6 The signal was filtered from 40 to 500 Hz on an Electronics for Medicine DR-16 amplifier-recorder. Simultaneous surface electrocardiographic signals from multiple leads were recorded. A bipolar electrode catheter was positioned via the right median basilic vein for right atrial pacing.

The internal and surface electrocardiographic signals were recorded during the study on a Phillips 7-channel tape recorder at 33 in/sec. Time markers at 10 msec intervals were produced by a specially constructed impulse generator and recorded on tape with the electrocardiographic signals. The tape signals were later transferred to photographic paper for analysis.

The right median basilic vein was also used for hemodynamic measurements and for the administration of drugs. Left heart catheterization and coronary angiography were conducted by retrograde catheterization through the right brachial artery. Cardiac output was measured by indocyanine green dye which was injected into the pulmonary artery and sampled from the left brachial artery.

Electrophysiological and Pharmacological Observations

Sinus beats. Although most of the ventricular complexes were fully or partially ectopic, normally conducted sinus beats were observed in each electrocardiographic lead. The characteristics of these beats are listed in table 1. Atrioventricular conduction was normal with both A-H and H-V intervals within the normal range (fig. 3). The His bundle deflection itself was about 28 msec in width. During atrial pacing at a rate of 125 beats/min, which suppressed the ectopic activity, the A-H interval was prolonged to 130 msec but the H-V interval did not change. Refractory periods of the A-V node could not be determined because of

Table 1

Electrophysiological Characteristics of Sinus Beats and the Two Beats of Bidirectional Tachycardia

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Sinus beats</th>
<th>VPB-1</th>
<th>VPB-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-R interval (msec)</td>
<td>160</td>
<td>——</td>
<td>——</td>
</tr>
<tr>
<td>A-H interval</td>
<td>115</td>
<td>——</td>
<td>——</td>
</tr>
<tr>
<td>H-V interval</td>
<td>45</td>
<td>——</td>
<td>——</td>
</tr>
<tr>
<td>QRS duration</td>
<td>70</td>
<td>130</td>
<td>110</td>
</tr>
<tr>
<td>Axis—frontal plane</td>
<td>+75°</td>
<td>—75°</td>
<td>+110°</td>
</tr>
<tr>
<td>VPB-VPB interval (msec)</td>
<td>440-470</td>
<td>440-470</td>
<td></td>
</tr>
</tbody>
</table>

Comparison of the morphology of the surface electrocardiogram (leads 1, 2 and V1) and His bundle electrogram of a sinus beat with each of the two types of ventricular beats in the bidirectional tachycardia. Each was recorded at the equivalent of 200 mm/sec. Note that both the ectopic complexes with frontal plane axis of −75° and of +110° are ventricular in origin with QRS beginning before any deflection resembling a His spike is recorded. The appearance of V1 in both ventricular beats is diphasic and the initial deflection is opposite to the r wave of the sinus beat. Such morphology favors ventricular ectopic origin over aberrancy from examination of the standard electrocardiogram alone. Time markers = 10 msec.
interference by the ectopic beats. The Q-T interval was normal.

Ventricular beat 2 (VPB-2) had an axis in the frontal plane of $-75^\circ$ with a diphasic predominantly positive qR pattern in V1 (figs. 1 and 3). His deflections did not precede the ventricular depolarizations and were presumably buried within them.

Ventricular beat 2 (VPB-2) had an axis in the frontal plane of about $+110^\circ$, predominantly positive deflections in the right chest leads (qR pattern) and large S waves in V5 and V6 like VPB-1 (figs. 1, 2, 3). Again no His bundle deflection was seen in relation to these beats.

Fusion beats. The QRS morphology of some beats was a mixture of the sinus and ectopic beats (fig. 4). In such cases the intracardiac records revealed the presence of fusion beats; P waves preceded slightly abnormal QRS complexes with normal A-H and shortened or "negative" H-V intervals.

Relationships between beats. The intervals between sinus beats and the initiating beats of bidirectional tachycardia and between VPB-1 and VPB-2 in the tachycardia were 440-470 msec (fig. 5). All beats occurred beyond the vulnerable phase of the previous beats. Although the runs of bidirectional tachycardia began either with beats of VPB type 1 or 2 or with fusion beats, the final beat was always VPB-2 (right axis deviation). No change of coupling intervals heralded the conclusion of a burst of tachycardia.

Suppression with atrial pacing. Atrial pacing was established at different fixed rates and the effect on the arrhythmia was observed. As the atrial rate approached the coupling interval between ectopic beats the incidence of abnormal beats decreased. The longest paced atrial cycle length (slowest rate) which produced complete suppression was 500 msec (120 beats/min). Following release of atrial pacing the arrhythmia was absent for 44 sec whereupon it returned in typical fashion.

Suppression with lidocaine: A 50 mg bolus intravenous injection of lidocaine produced compete although temporary suppression of all ectopic beats.

Hemodynamic and Angiographic Observations

All hemodynamic measurements were within the normal range and no evidence of an intracardiac shunt was demonstrated (table 2). Left ventricular cine angiography was normal. The coronary arteries were angiographically normal with a dominant right coronary circulation.

Discussion

Origin of the Tachycardia

The principal question debated in reports on bidirectional tachycardia is: does the arrhythmia

Figure 5

A burst of bidirectional tachycardia with three simultaneously recorded standard leads and His bundle electrogram. The interectopic interval varies slightly within a narrow range of 450-470 msec. The two atrial beats (A) do not conduct to the ventricles or influence significantly the morphology of the ectopic beats. The first A complex before ventricular beat 3 occurs too close to the beginning of the ectopic beat to reach the ventricles before they are depolarized from the ectopic focus. The A complex after beat 5 probably blocks in the A-V node because of retrograde penetration of the node by the VPB. Although unlikely, this A complex could result from retrograde activation of the atria. A sinus beat with the normal A-H and H-V intervals is seen after the tachycardia spontaneously stops. Paper speed of 100 mm/sec; time markers = 10 msec intervals.
BIDIRECTIONAL TACHYCARDIA

Table 2

<table>
<thead>
<tr>
<th>Location</th>
<th>Pressures</th>
<th>Oxygen saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricle</td>
<td>105/11</td>
<td></td>
</tr>
<tr>
<td>Right brachial artery</td>
<td>105/70</td>
<td></td>
</tr>
<tr>
<td>Pulmonary capillary wedge</td>
<td>(11)†</td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>21/10</td>
<td>83%</td>
</tr>
<tr>
<td>High right ventricle</td>
<td></td>
<td>81%</td>
</tr>
<tr>
<td>Mid right ventricle</td>
<td>21/6</td>
<td>80%</td>
</tr>
<tr>
<td>Low right atrium</td>
<td>(5)†</td>
<td>78%</td>
</tr>
<tr>
<td>High right atrium</td>
<td></td>
<td>81%</td>
</tr>
<tr>
<td>Superior vena cava</td>
<td></td>
<td>79%</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td></td>
<td>87%</td>
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*Cardiac index 3.0 liters/min/M²; stroke volume index 33cc/M²; pulmonary vascular resistance index 1.0 units.
†Mean pressures in parentheses.

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originates in the ventricles, in the A-V junctional tissues or in the atria? The distinction is always difficult to make from examination of the surface electrocardiogram alone. Even careful documentation of the relationship between atrial and ventricular activity with esophageal or atrial electrograms may not settle the issue. Impulses originating in either the ventricles or the His bundle region may or may not control the atria depending upon A-V nodal refractoriness.

His bundle electrocardiography provides a technique which can solve this problem. Beats originating above or in the immediate vicinity of the His bundle will appear with His deflections preceding intraventricular depolarization. Beats of ventricular origin depolarize the bundle of His by retrograde activation. The His deflection occurs during inscription of the QRS and is usually obscured by the large electrical forces generated by the depolarizing ventricular myocardium.

In the case reported here, His bundle deflections were clearly seen during sinus beats but no His deflections preceded the beats of bidirectional tachycardia. This provided presumptive evidence that the beats were ventricular in origin. Further support of the ventricular origin theory was given by the presence of fusion beats. Such complexes are frequently cited as evidence for ventricular beating from surface records alone. Fusion beats are not commonly observed in the reported cases of bidirectional tachycardia, and therefore this important clue establishing the ventricular origin of the ectopic beats is not available. The reason for the lack of fusion beats may be that most patients with bidirectional tachycardia are toxic from digitalis, and the drug blocks transmission of the supraventricular beats through the A-V node. Our patient was not taking digitalis and had normal A-V nodal antegrade conduction.

The His bundle deflection is slightly wider than has been reported in most normal patients. The possibility cannot be dismissed, therefore, that some conduction disturbance is present within the bundle of His itself, since a prolonged His deflection may indicate such abnormality. We are uncertain whether, in fact, prolongation of the His deflection does carry such connotation. However, if this were true, then retrograde block within the His bundle could explain the absence of ventriculo-atrial conduction during the tachycardia.

The records also suggest that the ectopic beats begin in the left and not the right ventricle. The consistently positive deflections in lead V₁ during bidirectional tachycardia imply that the impulses travel toward the free wall of the right ventricle. The morphology of the beats in the horizontal plane leads is similar to that seen with left ventricular pacing. Other workers have also concluded that VPBs with this form originate in the left ventricle. The findings from direct epicardial mapping of the origin of ventricular beats in dogs support our belief that we can predict the approximate location of the ectopic focus from surface leads.

Our studies also demonstrate that the origin of the ectopic beats during bidirectional tachycardia is not in the proximal bundle branches fascicles. Such beats, according to recent work by Massumi, Ertem and Vera, can be identified by H-V intervals which are shorter than normal with the His bundle discharged retrograde in such cases. The focus of fascicular beats is presumably so close to the His bundle that the His deflection occurs and can be detected before it is obscured by the large signal of ventricular myocardial depolarization. In our case of bidirectional tachycardia, the time of His deflection during the ectopic beats could not be determined and therefore a more precise localization was not possible.

Morphology of the Beats in Lead V₁

The most comprehensive review of bidirectional tachycardia in recent years was published by Rosenbaum, Elizari and Lazzari in 1969. Actually in that year the technique of His bundle electrocardiography in man was first reported and the conclusions of Rosenbaum, Elizari and Lazzari of course were based solely on observations of the surface electrocardiogram. They were the first to
demonstrate that in lead V₁ the deflections were always positively directed and had the appearance of right bundle branch block with the morphology of qR or rsR'. From the tracings presented in their paper we cannot be certain how often typical triphasic forms of right bundle branch block were present in V₁ or whether the beats often demonstrated the monophasic or diphasic contour more often found with ventricular ectopic beats. In our case the morphology of each beat of bidirectional tachycardia was diphasic or occasionally monophasic in lead V₁. Typically a small q wave preceded a wide tall R wave. The initial vector was thus opposite in direction to the r wave seen in lead V₁ during sinus beats. On the basis of such analysis from the surface electrocardiogram alone, one would conclude that the beats were more likely to be ventricular ectopic than aberrantly conducted from a supraventricular pacemaker. The His bundle records confirmed this assumption.

**Alternating Morphology**

Our study does not explain the electrocardiographic feature of bidirectional tachycardia which gives it distinction: the alternating morphology of QRS with the characteristic positive and negative deflections. In this case we could localize the origin of the arrhythmia to the left ventricle but could not conclude whether the beats originated from two separate foci or from a single focus with alternating routes of ventricular depolarization.

**Pacing and Pharmacological Interventions**

Suppression of the bidirectional tachycardia by atrial pacing does not help localize the ectopic beats since any lower focus whether in the A-V junction or the ventricles might be extinguished in this manner. A reentrant tachycardia using the A-V node is certainly unlikely since such an arrhythmia does not disappear during rapid atrial pacing and then reappear when the pacing is stopped. This type of response is more characteristic of an automatic ectopic focus. The effects of rapid atrial pacing on a sustained tachycardia can be used as one criterion to differentiate reentrant supraventricular from ectopic atrial tachycardias.

The suppressing effect of lidocaine favors ventricular origin of the tachycardia since this drug is relatively ineffective in terminating sustained supraventricular arrhythmias. The lack of clinical response to other anti-arrhythmic drugs, however, does not further define the nature of this arrhythmia. No drugs were administered to the point of toxicity, and blood level determinations were not obtained. It is likely that with higher doses of drugs such as procainamide the arrhythmia could be controlled. In view of the patient's own desires in this matter and her asymptomatic state for the past eight years, such an effort was not undertaken.

**Clinical Relationships**

Ectopic arrhythmias have been observed to occur with surprising frequency in patients with hyperkalemic periodic paralysis. It is tempting to relate dysfunction in skeletal and cardiac muscle in such cases, but with our current state of knowledge only speculation is possible.

The appearance of bidirectional tachycardia in this patient is highly unusual for several reasons. She is young, relatively asymptomatic, has normal heart function apart from the arrhythmia and is not receiving digitalis. Except for the absence of periodic paralysis, the case recently reported by Gault et al. may be relevant. The subject was a 16 year old girl with repetitive bouts of bidirectional tachycardia. Fusion beats were observed on the standard electrocardiogram, and speeding of the atrium by atropine or atrial pacing (rate of 110 beats/min) suppressed the arrhythmia. As with our patient, the disorder was not suppressed by high doses of antiarrhythmic drugs, and she died from ventricular arrhythmias possibly exacerbated by quinidine administration. Fatty and mononuclear cell infiltration in the A-V conduction system and main left bundle branch was seen at autopsy. The authors suggested that the arrhythmia may have originated in "the main left bundle branch proximal to its bifurcation."

Bidirectional tachycardia typically occurs in older people with severe myocardial disease, is thought characteristically to be produced by digitalis intoxication and carries a poor prognosis. It is of course possible that in such patients the origin of the arrhythmia may be different from our case despite apparent similarities from surface leads. However, if additional cases of bidirectional tachycardia studied with intracardiac electrode technique in patients with the more typical clinical syndrome should confirm the data reported here, it would seem reasonable that this arrhythmia be called "bidirectional ventricular tachycardia."

**Addendum**

Since submission of this manuscript, a patient has been reported with bidirectional tachycardia occurring in the presence of digitalis intoxication. Intracardiac records revealed that the origin of the abnormal beats...

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JOHN A. KASTOR and BRUCE N. GOLDREYER

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