Bidirectional Tachycardia
A Sustained Form, not Related to Digitalis Intoxication, in an Adult without Apparent Cardiac Disease

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Summary
In this paper we report the first adult case of an "idiopathic" ventricular bidirectional tachycardia (BT), in a 57 year old woman. The tachycardia, at the time of our observation, was incessant in type and had a slightly irregular frequency of about 140 bpm. BT initiated and terminated abruptly, without any temporal relationship to the preceding RR interval, or the QRS morphology. The interval between the two alternating QRS patterns often varied over a wide range of values. The BT could be interrupted only by overdrive atrial and ventricular stimulation, but promptly reappeared as pacing was discontinued. Therapy with quinidine associated with propranolol was effective on a long term trial. The vectorcardiographic analysis and the electrophysiologic investigation demonstrated a ventricular origin of the BT, localizing its site of origin to common myocardial tissue, probably near the two left hemifascicles. Our data could not elucidate the electrogenic mechanism of this ventricular arrhythmia, because of its chaotic behavior.

Additional Indexing Words:
Bidirectional tachycardia  Ventricular tachycardia

Bidirectional tachycardia (BT), is an uncommon rhythm disturbance, the origin and mechanism of which are still disputed.\textsuperscript{1,3,5–8} In the majority of cases it occurs as a manifestation of digitalis toxicity, particularly in the presence of severe myocardial disease. This report describes the clinical, electro-vectorcardiographic and electrophysiologic findings in a 57 year old woman with incessant tachycardia, not caused by digitalis or other drugs.

Case Report
Clinical history: A 57 year old woman was referred because of an incessant BT. The past cardiac history included 17 years of frequent paroxysmal...
episodes of palpitations, which had increased in frequency and duration in the last year, and had recently became incessant. The patient had neither syncopeal episodes, nor dizziness or heart failure.

Her blood pressure was 160/90. Laboratory data were all normal. Chest X-ray showed a cardiothoracic index of 0.50. The echocardiograms, 2D and M mode, revealed normal diameters and a slight reduction of the left ventricular contraction indexes. Previous therapy with verapamil, lignocaine, and mexiletine, had no effect on the BT, which was suppressed by propranolol 160 mg/day + quinidine 800 mg/day.

**Electrocardiogram (ECG):** The admission standard ECG showed a sinus rhythm, a PR of 0.18 sec and a QRS axis of -45 degrees. During BT, on the precordial leads there was a right bundle branch block (RBBB) pattern, with a beat-to-beat alternation in amplitude and duration. On the frontal leads, the QRS axis had a beat-to-beat alternation between -45 and +100 degrees. The BT had a slightly irregular frequency of 140 beats per minute; each QRS complex was followed by a retrograde P wave (Fig. 1).

Previous ECG (Fig. 2), recorded 14 years before, showed isolated monomorphic premature ventricular contractions with a morphology of RBBB with right axis deviation (RAD). Later on, couplets and monomorphic ventricular tachycardia were documented, and in the last 5 years, typical episodes of BT were detected.

The ECG analysis of 50 consecutive episodes of BT showed that: The beginning of the BT was not preceded by variations of the sinus node cycle.

![Fig. 1. Twelve lead ECG showing an episode of BT. The first beat is of sinus origin. The BT is characterized by alternation of the QRS axis on the frontal plane, and by alternation of the QRS amplitude on the horizontal plane.](image-url)
Fig. 2. Lead aVF. The figure shows short strips of the ECG recorded over a period of years. There is an evolution from a monomorphic to a bimorphic pattern with increasing severity.

length; the morphology of the first QRS showed in 48 out of 50 documented episodes a RBBB with left axis deviation (LAD), and a coupling interval to the preceding sinus QRS varying between 500 and 540 msec; in 2 cases, the initial QRS had a RBBB+RAD morphology and a coupling interval of 640 msec; the BT cycle length often varied; the interval between the RBBB+LAD pattern and the following QRS ranged from 460 to 560 msec; the termination of the BT was often spontaneous and the last QRS had either of the two morphologies described above in a random pattern. Sometimes the BT
stopped with a premature ventricular contraction.

**Vectorcardiogram (VCG):** Both loops corresponding to the two QRS morphologies excluded an intraventricular conduction disturbance, namely RBBB associated with a left anterior hemiblock (LAH), or with a left posterior hemiblock (LPH). The initial forces (first 40 msec) presented a marked slurring and a left anterosuperior direction with the first morphology (LAD), and a left anteroinferior direction with the second one (RAD) (Fig. 3). These loops, characterized by an initial slurring, and their subsequent morphologies, closely resemble 2 Wolff-Parkinson-White patterns due to a left anterosuperior paraseptal, or to a left posteroinferior paraseptal bundle. A reasonable explanation for these analogies could be based upon the origin and/or by an initial spread of the BT being located in common myocardial tissue with a subsequent partial invasion of the two left hemifascicles.

**His bundle electrocardiogram (HBE):** Basic intervals were PA = 35, AH = 100, HV = 45 msec. The first beat of the BT was a fusion beat, while during the BT the His potential was recorded after the onset of the QRS. 1:1 retrograde conduction was present with a VA of 150 msec (Fig. 4). Mapping of the right ventricle documented that the earliest electrical activity during BT was detectable on the right intraventricular septum, near the His potential, with a subsequent invasion of the apex (QRS with LAD), and of the infundibulum (QRS with RAD), respectively (Fig. 5).

Vagal maneuvers did not affect the BT. The arrhythmia was suppressed by both atrial and ventricular overdrive pacing at a cycle length of 400 msec. Paired or coupled extrastimuli, continuous underdrive or overdrive pacing at different sites and with different cycle lengths, neither induced different arrhythmias, nor caused deterioration of the former.

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![Fig. 4. The intracavitary ECG during an episode of BT shows on the left a sinus rhythm followed by AV dissociation due to the appearance of the BT. The Hisian deflection shifts gradually behind QRS. HBE = His bundle electrocardiogram; MRA = middle right atrium; HRA = high right atrium.](image)
DISCUSSION

Most of the reports concerning cases of BT deal with a strong association between this arrhythmia and digitalis intoxication.1,3 There are only 7 reported cases of apparently “idiopathic” BT, all in people younger than 20 years.2,4,9 In 4 of them, the BT was paroxysmal, and stress related, in 3 it was often incessant. Three had syncope. Two died suddenly (they were brothers), and the pathologic study in one revealed a fatty and mononuclear
infiltration of both ventricles and the conduction tissue. The follow up of the 5 other patients is not reported.

In our patient, the symptoms started only in her forties, and only when she was 53 years old was a typical BT documented. The arrhythmia was always well tolerated, and only in the last month did the patient complain of discomfort, in relation to the high heart rate.

From an electrocardiographic aspect, the BT demonstrated peculiar behavior. Initially it had a monomorphic pattern and progressive severity, isolated premature ventricular contractions, couplets and sustained tachycardia. The appearance of a second morphology was documented only 12 years later.

Neither the first nor the second focus had a constant relationship to the other. Other characteristics of the BT included initiation randomly with either morphology, and various late diastolic coupling intervals without modification of the preceding sinus node cycle length. Its ending had a similarly chaotic behavior.

The HBE results did not contribute to the interpretation of the mechanism of the arrhythmia. A re-entry mechanism could be ruled out as response to pacing was inconsistent with this hypothesis. The overdrive suppression of the BT, both from the atria and the ventricle, and the late diastolic initiation of the arrhythmia (predominantly by one of the two QRS morphologies), could be consistent with an automatic focus situated close to the two hemifascicles and conducted to the ventricle through two different pathways, but this hypothesis is not supported by the chaotic relationship of the two QRS patterns. A third possibility could be the presence of two distinct automatic foci, with some irregular modulation between them, but the confirmation of this hypothesis is extremely difficult.

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REFERENCES