CorSalud 2017 Apr-Jun;9(2):116-122

CorSalud

Cuban Society of Cardiology

Case Report

Cardiocentro Ernesto Che Guevara Santa Clara Cuba

# Singular electrical stories: Bidirectional ventricular tachycardia and other events

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*Este artículo también está disponible en español*

ARTICLE INFORMATION

Received: April 25, 2017
Accepted: May 30, 2017

**Competing interests**
The authors declare no competing interests

**Acronyms**
BVT: bidirectional ventricular tachycardia
LBBB: left bundle branch block
RBBB: right bundle branch block
VT: ventricular tachycardia

*On-Line* Versions:
Spanish - English

**ABSTRACT**
Bidirectional ventricular tachycardia is a rarely seen arrhythmia; resulting from a triggered activity alternatively originated in the left anterior and posterior fascicles with a beat-to-beat rightward alternation of the frontal plane axis, thus causing bidirectional appearance. Two patients with this atypical arrhythmia along with their electrocardiographic tracings are presented in this article.
*Key words:* Bidirectional ventricular tachycardia, Bigeminism, Late post-depolarization

## *Historias eléctricas singulares: Taquicardia ventricular bidireccional y otros eventos*

**RESUMEN**
*La taquicardia ventricular bidireccional es una arritmia que no se observa con frecuencia. Se produce como consecuencia de una actividad disparada que nace de manera alternativa en los fascículos anterior y posterior izquierdos, con un eje en el plano frontal que alterna de izquierda a derecha latido a latido y da lugar a la apariencia bidireccional. En este artículo se presentan dos pacientes con esta peculiar arritmia y se muestran los trazados electrocardiográficos.*
*Palabras clave: Taquicardia ventricular bidireccional, Bigeminismo, Posdespolarizaciones tardías*

## INTRODUCTION

Bidirectional ventricular tachycardia (BVT) is a rarely seen arrhythmia; resulting from a triggered activity alternatively originated in the left anterior and posterior fascicles with a beat-to-beat rightward alternation of the frontal plane axis, thus causing bidirectional appearance. BVT shows an alternating right and left bundle branch block pattern, where QRS axis shifts 180° from left to right with each alternate beat.

It is associated with digitalis toxicity, catecholaminergic polymorphic ventricular tachycardia (VT) and other clinical situations[1]. The mechanism of this bidirectional morphology has not been clearly identified, some refer to an alternating conduction of a single ventricular focus, a tachycardia

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dia arising from one focus and triggering another or a double ventricular foci (from the right and left apical portions of the heart). Such diverse morphology of the QRS in the same registry suggests that the focus of the arrhythmia varies. The Purkinje network has been suggested as the site of origin of BVT, with alternating firing from the right and left branches of the Purkinje fibers[2].

Two patients with this unusual arrhythmia are presented in this article.

## CASES REPORT

### Pacient 1

A 56-year-old man presented with symptoms where shortness of breath and edema of the lower extremity predominated.

Transthoracic echocardiogram showed: biatrial enlargement, hypertrophic cardiomyopathy involving the left ventricular inferior and posterobasal segment, posterior wall of 15-18 mm, normal septum, left ventricular asymmetric hypertrophy, decreased left ventricle ejection fraction with moderate ventricular dysfunction, and moderate-severe right ventricular dysfunction; as well as left pleural effusion that was evacuated but reproduced quickly. Coronary angiography was normal.

Patient was resuscitated from cardiac arrest resulting from ventricular fibrillation and received an implantable cardioverter-defibrillator. He has suffered two ICD shocks to date.

The patient was diagnosed with: atypical site-hypertrophic cardiomyopathy, and possible infiltrative or deposition disease was suspected.

Electrocardiographic tracings are shown in figures (**fig. 1**, **fig. 2** and **fig. 3**).

### Pacient 2

A 53-year-old woman, consumer of alcoholic beverages and smoker. She presented with faint and decay sensation. Echocardiogram and coronary angiography were normal, so structural heart disease cannot be demonstrable by conventional methods. Treatment was started with oral propranolol.

See electrocardiographic tracings in the following figures (**fig. 4**, **fig. 5**, **fig. 6**, **fig. 7** and **fig. 8**).

## COMMENT

BVT is characterized by an alternating beat-to-beat electrocardiogram QRS axis. It is a rare arrhythmia first described in 1922 associated with digitalis toxicity. This arrhythmia can also be seen in hypokale-

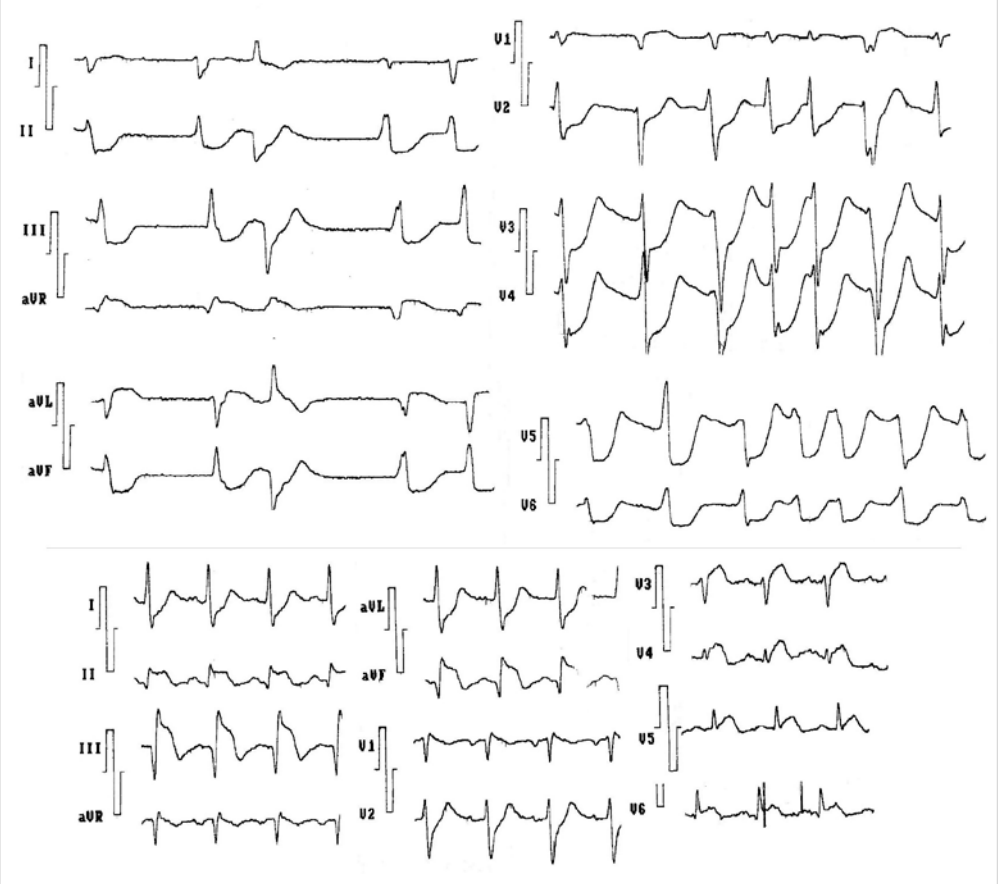


**Figure 1.** Patient 1, tracing N° 1. **Top:** no observed P wave, irregular R-R, QRS complexes with different morphology and ST-segment depression in $D_{II}$, $D_{III}$, aVF and $V_2$-$V_6$. **Bottom:** sinus rhythm, ST segment elevation in DII, DIII, aVF and $V_3$-$V_6$.

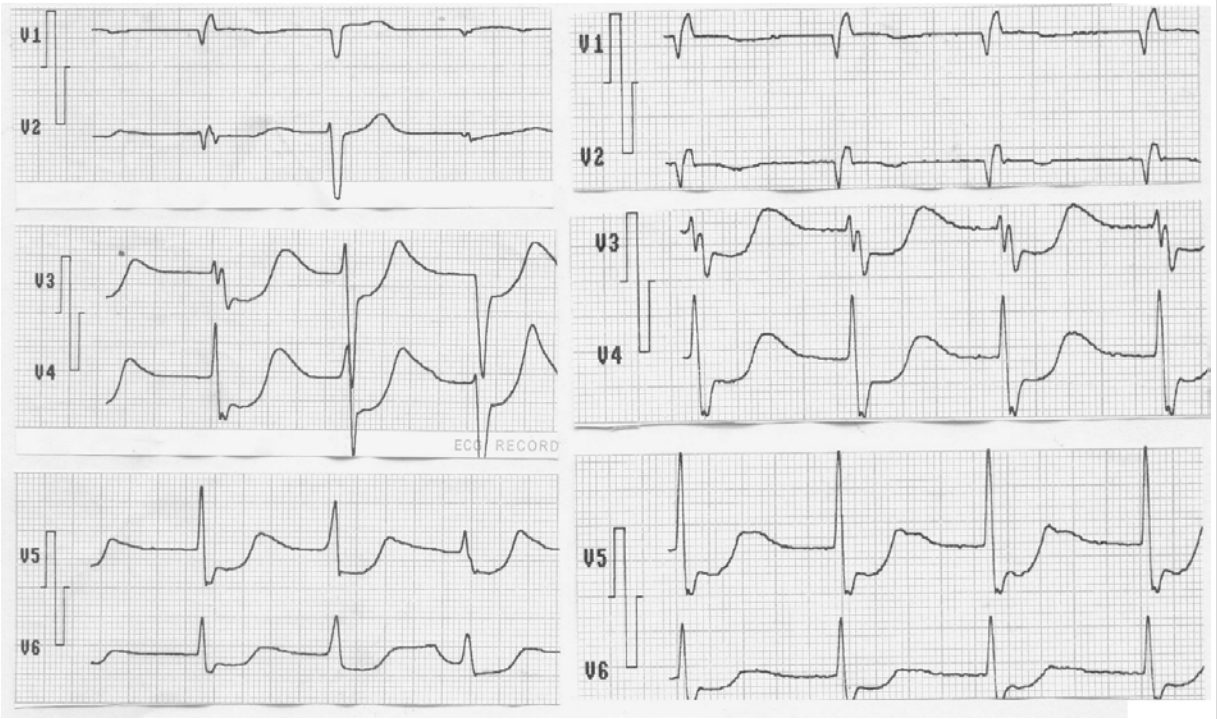


**Figure 2.** Patient 1, tracing N° 2. **Left:** no observed P wave, QRS complexes with different morphology and ST-segment depression up to 5 mm in $V_3$-$V_6$. **Right:** P wave absence, long QT interval, bimodal T-wave and ST-segment depression in $V_3$-$V_6$.

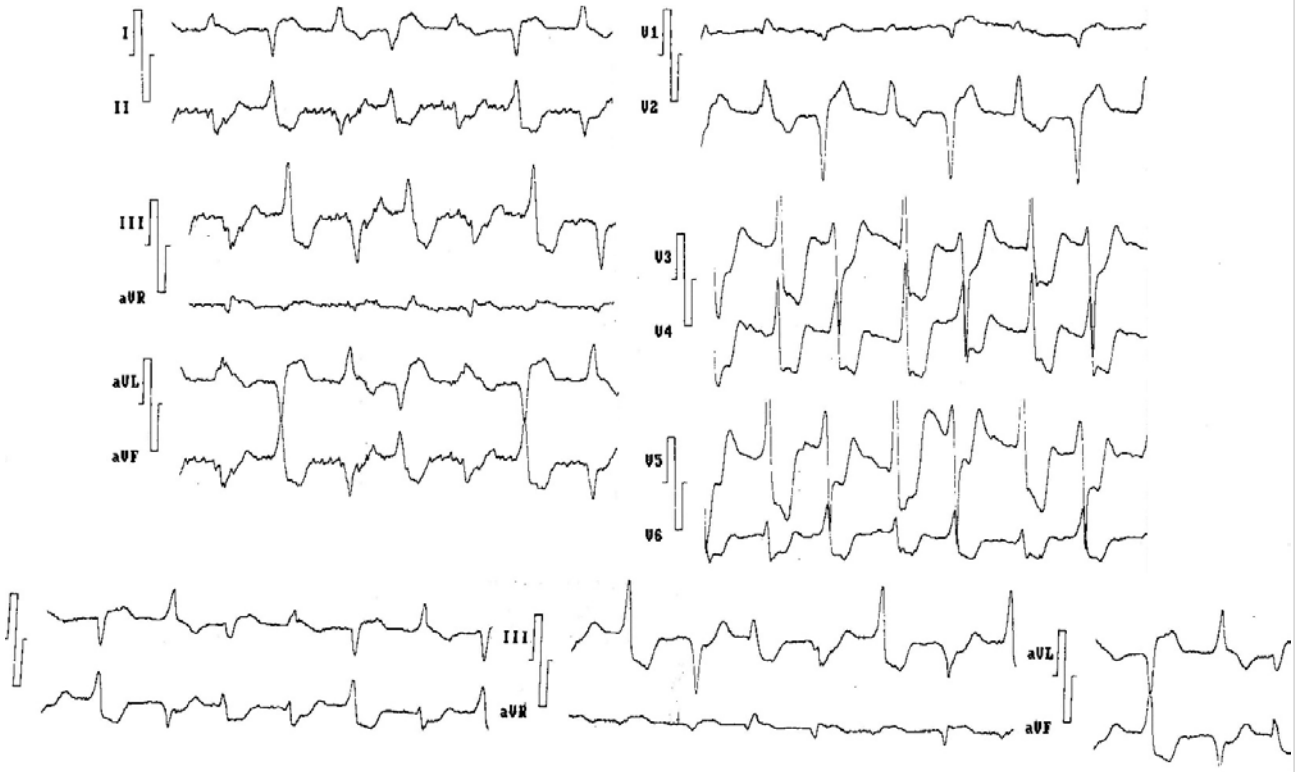


**Figure 3.** Patient 1, tracing N° 3. BVT, alternate positive and negative polarity of the QRS complex.

mic periodic parlaysis, Andersen-Tawil syndrome, fulminant myocarditis, catecholaminergic polymorphic VT, ischemic heart disease, arrhythmogenic right ventricular cardiomyopathy and several other conditions that predispose cardiac myocytes to late post-depolarization and triggered activity. The most characteristic pattern is right bundle branch block (RBBB) with an alternating frontal plane axis; but there are other patterns such as alternating RBBB and left bundle branch block or alternating QRS axis with a narrow QRS[3, 4]. There may be RBBB morphology without increased QRS if the two foci are in the anterior and posterior fascicles of the left bundle branch.

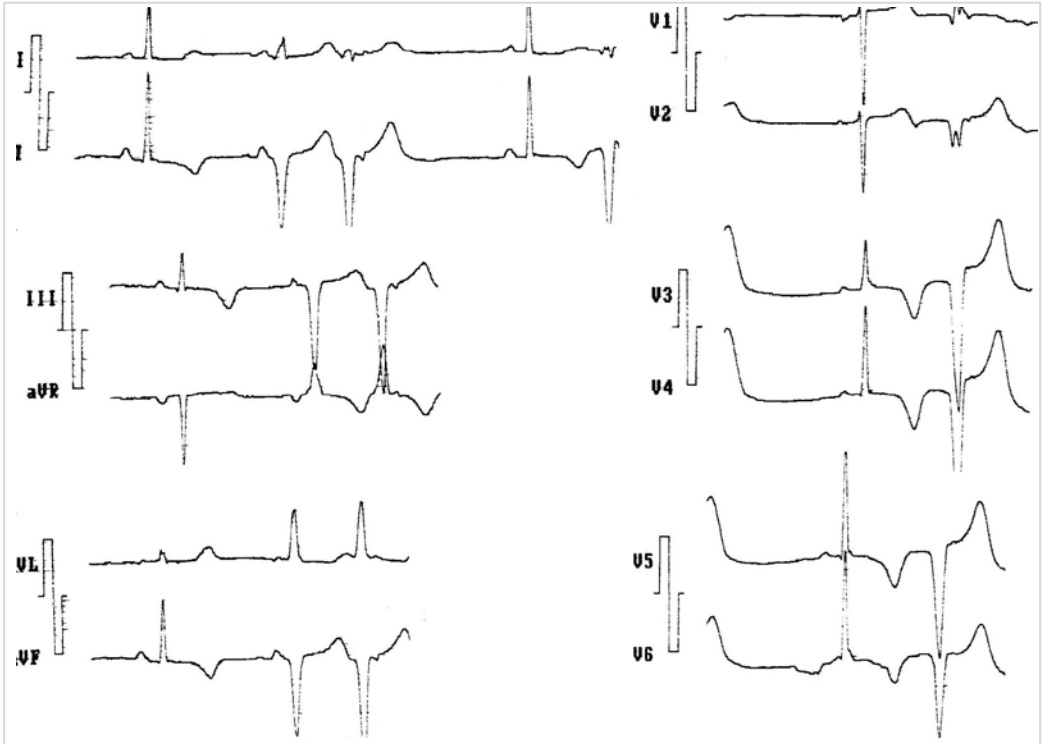


**Figure 4.** Patient 2, tracing N° 1. Presence of negative T wave in $D_{II}$, $D_{III}$, aVF and precordial leads; incessant arrhythmia, first sinus complex followed by ventricular complexes with atrioventricular and ventriculo-atrial dissociation.

BVT presents with atrioventricular dissociation and a repetitive pattern of alternate ventricular activation, with left and right axis deviations usually in association with RBBB. Such mechanism is controversial[4].

Its common precursors are premature ventricular contractions and ventricular bigeminy. Baher *et al.*[3] propose a simple ping-pong mechanism called reciprocating bigeminy in order to explain the alternating pattern observed in BVT, a mechanism that would produce the characteristic electrocardiogram and its degeneration to polymorphic VT if additional sites develop bigeminy[3].

BVT has a ventricular origin with conduction system involvement, although, altered automaticity, reentry, and triggered activity have been proposed as possible mechanisms, mainly occurring in the His-Purkinje system with alternating reciprocal activetion of the right and left conduction system[4]. When there are two anatomically separate foci, BVT can be converted into monomorphic through ablation of one of the ectopic focus.

Evidence from human and animal studies attributes BVT to alternating ectopic foci originating from the distal His-Purkinje system in the left and right

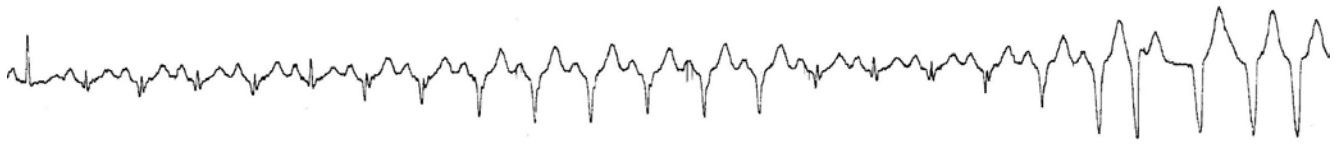

**Figure 5.** Patient 2, tracing N° 2. First sinus complex followed by bursts of fusion complexes and dissociated ventricular tachycardia, again fusion and dissociated tachycardia. Different morphologies of the QRS and irregular R-R.

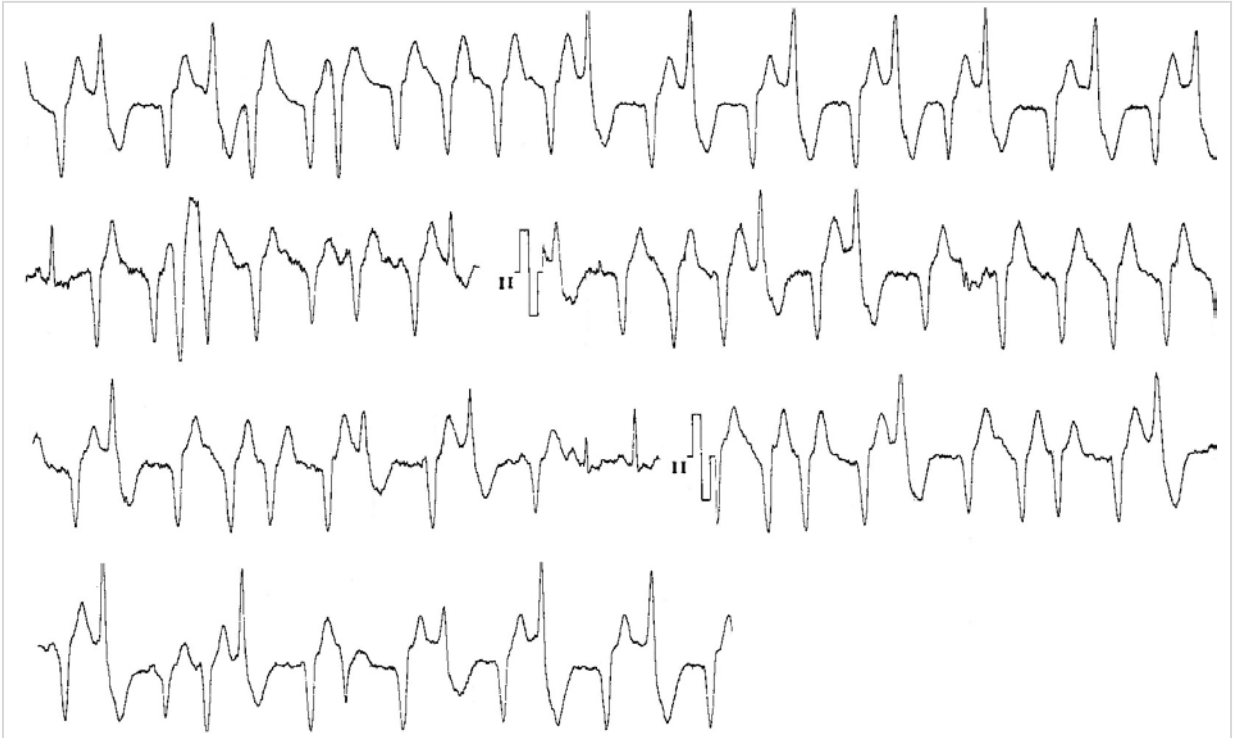


**Figure 6.** Patient 2, tracing N° 3. Irregular ventricular tachycardia, different QRS complexes. Irregular R-R. Sometimes bidirectional ventricular tachycardia.

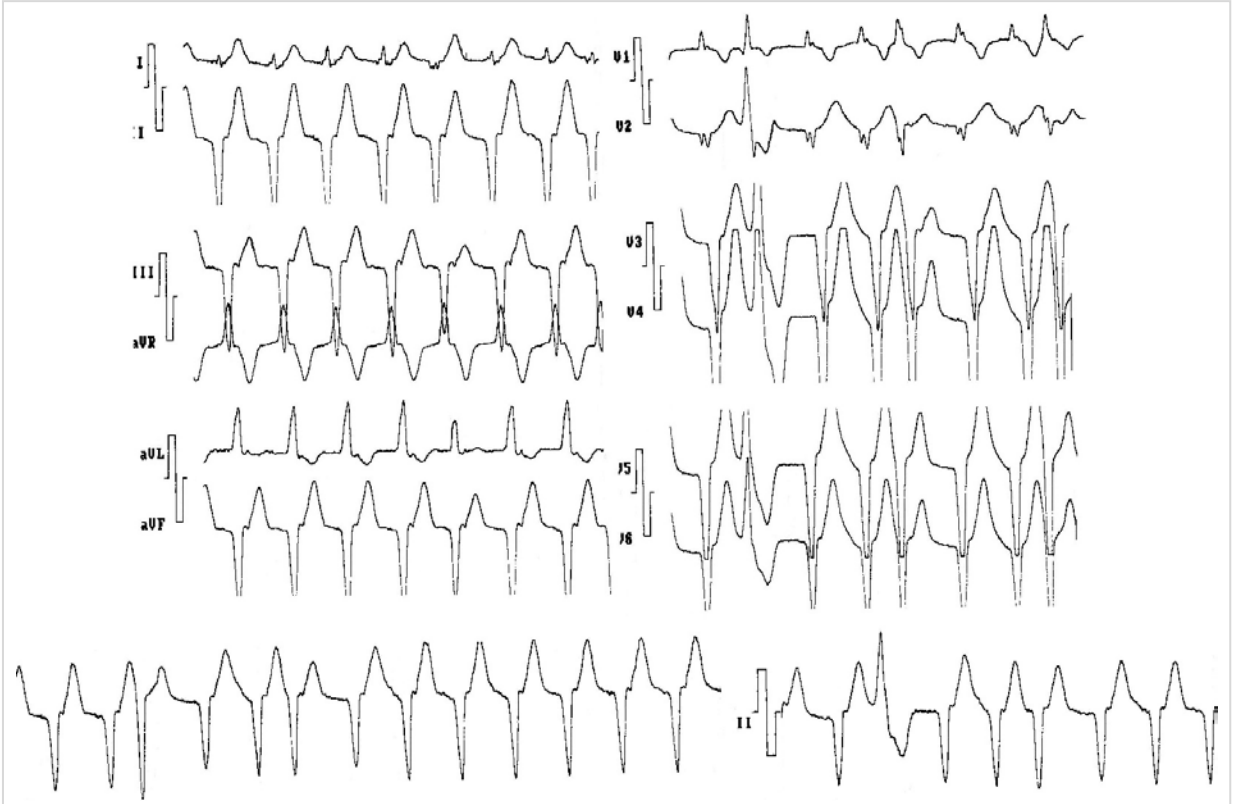


**Figure 7.** Patient 2, tracing N° 4. Sustained ventricular tachycardia, right bundle branch block morphology with left axis, different forms of QRS, occasionally irregular and $V_2$-$V_6$ concordance.

ventricle respectively[3].

When the heart rate exceeds the threshold for bigeminy at the first site in the His-Purkinje system, ventricular bigeminy is developed, causing the heart rate to accelerate and exceed the threshold for bigeminy in a second site. The triggered beat from the first site induces a triggered beat from the second site; it reciprocates and induces a triggered beat from the first site, and so forth. Reciprocating bigeminy from the two sites would produce BVT while three or more would produce polymorphic VT. The ventricular mechanisms include a single focus in the proximal His or branches showing alternating left fascicular block with single or double foci in the dis-

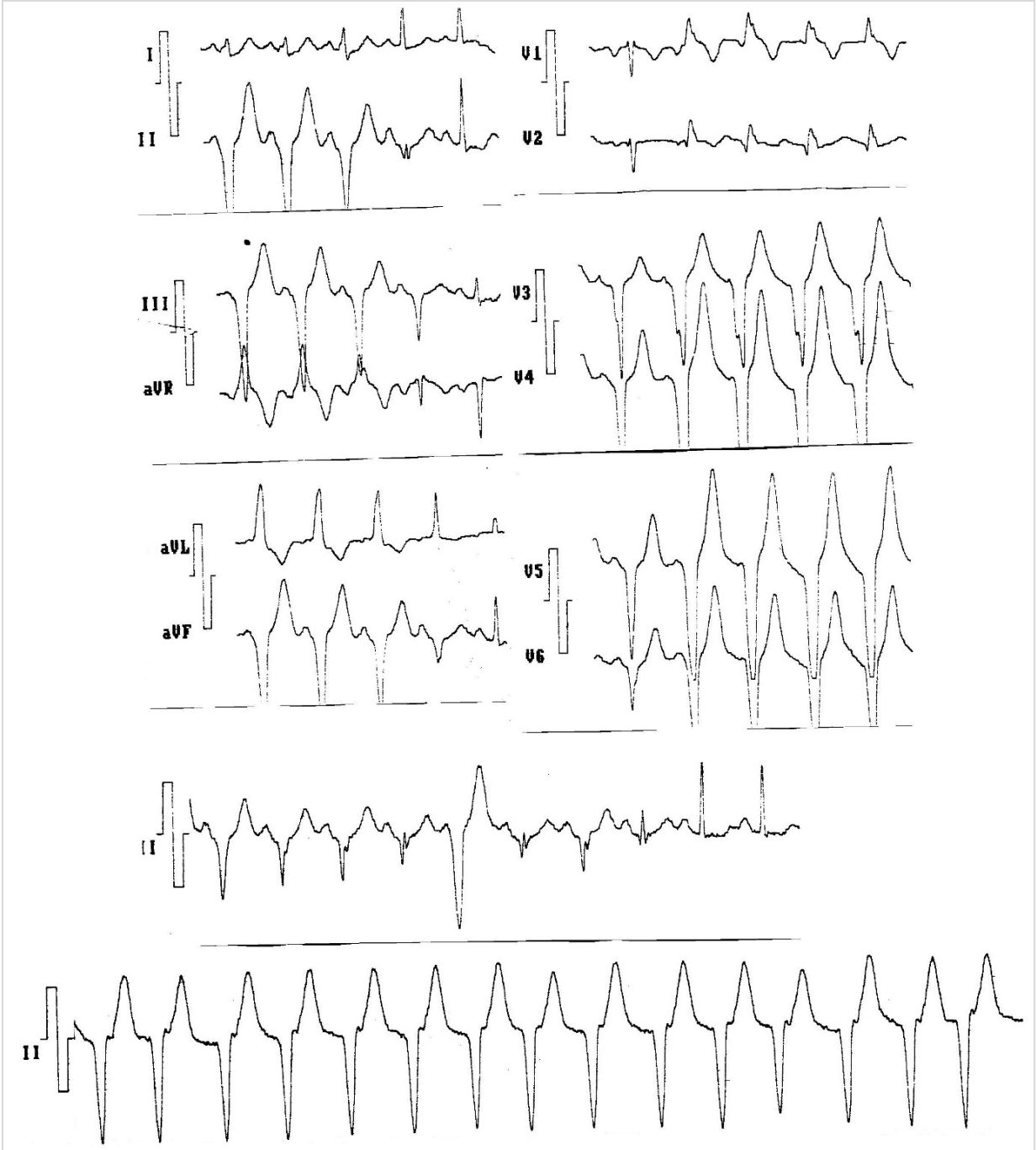


**Figure 8.** Patient 2, tracing N° 5. Ventricular tachycardia, sometimes fusion and sinus complexes.

tal His-Purkinje system. There is no obvious explanation as to why the fascicle or branch block may alternate during BTV when there is only one focus.

Usually concealed retrograde conduction perpetuates the fascicle or bundle branch block that initially develops conduction block. In the double foci, when none is protected by entrance block, the faster focus could overdrive the slower one, which results in a monomorphic VT rather than BVT[3,4].

In contrast to these complicated mechanisms reciprocating bigeminy clarifies the enigma of the QRS alternating morphology by a simple ping-pong mechanism where late post-depolarizations-induced triggered activity develops at different heart rate thresholds in different regions of the His-Purkinje system or ventricles, related to the cellular properties of late post-depolarizations that induce triggered activity. Producing a constant cycle length during the non-alternating BVT only requires the coupling interval of triggered beat to be similar at the two sites. Bigeminy models resulting from triggered activity due to late post-depolarizations are similar to any mechanism inducing ventricular bigeminy, which includes automaticity and reentry at more than one ventricular site. There are no strict requirements for the two bigeminal foci to be located in the His-Purkinje system or in opposite fascicles or in the ventricles. Two reciprocating triggered foci located at the the same ventricle/sites in the endocardium and epicardium can produce BVT by this mechanism even if QRS axis and morphology changes were different[3].

In humans, the most common BVT pattern during digitalis toxicity and catecholaminergic polymorphic VT is RBBB with alternating right and left axis deviation in relation to reciprocating ectopic foci located in the distal left anterior and posterior fascicles[3].

The full spectrum of arrhythmias associated with BVT, whether acquired or familial forms, can be related to the properties of late post-depolarizations-triggered arrhythmias. BVT originates when a second site develops ventricular bigeminy by reciprocal activation of the first site by the ping-pong mechanism. This explains ventricular bigeminy, polymorphic VT, monomorphic VT and degeneration into ventricular fibrillation[3].

Possible BVT electrocardiographic patterns according to the location of double ventricular foci with reciprocating bigeminy are described in the **Box**[3].

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**Box.** Possible electrocardiographic patterns for bidirectional ventricular tachycardia.

| | | |
|---|---|---|
| Left bundle branch block | - | Right bundle branch block |
| Right bundle branch block and left axis deviation | - | Right bundle branch block and right axis deviation |
| Left bundle branch block | - | Right bundle branch block and right axis deviation |
| Left bundle branch block | - | Right bundle branch block and left axis deviation |
| Left axis deviation | - | Right axis deviation (normal QRS) |