ECG DIAGNOSIS

In this tracing it is very difficult to identify P waves. Is very hard to see any P wave. We observe very broad, regular, monomorphic and bizarre QRS complexes with HR ≈ 125bpm. If this ventricular event is interpreted as a Monomorphic VT and it has the QRS axis near + 120º with LBBB-like pattern the focus is located on RVOT. Idiopathic VT originating from RVOT in patients without structural heart diseases is generally considered benign. Organic MVT from the RVOT are rare and observed in ARVD, an orphan disease/cardiomyopathy. It is not frequent MVT from RVOT with organic substrate. So much ischemic cardiomyopathy as non-ischemic idiopathic dilated cardiomyopathy have not MVT from RVOT.

This case is unfortunately a terminal patient with severe CHF, hipotension and prerenal uremia, BUN/Creatine ratio 20:1, hyperkalemia(8.4 mEq/L) and hypocalcemia whose initial ECG showed a sinoventricular rhythm, permanent atrial paralysis(1) or junctional tachycardia with widening of the QRS complexes and sine-wave pattern. The ECG pattern is very similar to Spodick case report(2).

Sinoventricular rhythm implies preserved SA node function with conduction of impulses to the A-V junction without generalized atrial muscle excitation. Impulse propagation in such cases is presumably via specialized internodal tracts. In this present case, synchronized but localized activation from an area of the right atrium preceded each QRS, without generalized atrial depolarization. These recordings are offered as further evidence for the clinical occurrence of sinoventricular rhythm in humans.

The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded).

Another recent described cause of absent-P wave is hyperkalemia, associated with hypocalcemia and hypomagnesemia. This electrolytic imbalance combination resulting in (1):

1. **Peaking T waves**
2. **Prominent U waves**
3. **Prolongation of the descending limb of the T wave producing an overlapped with the next cycle P wave: "tee-pee" sign.**

Additionally, the QRS pattern has atypical Complete Left Bundle Branch Block pattern or paradoxical Lepeschkin type. Severe hyperkalemia is characterized by diffuse QRS complexes widening, similar to left or right bundle branch block, associated to anterior or posterior fascicular block by extreme shift of SAQRS in the FP to left or right. This QRS complex widening is differentiated of genuine branch blocks, because in them, the delay is final or middle, while in hyperpotasemia is always global or diffuse.

ECG DIAGNOSIS

SINOVENTRICULAR RHYTHM: Sinus command

COMPLETE LEFT BUNDLE BRANCH BLOCK (CLBBB) PARADOXICAL CLBBB LEPESCHKIN TYPE (1). It is characterized by extreme deviation of QRS axis to the right beyond + 90°. This variant is lesser than 1% of all cases of CLBBB.

CAUSES THAT DETERMINE PARADOXICAL CLBBB OR CLBBB WITH EXTREME DEVIATION TO THE RIGHT

• Fascicular LBBB (LAFB + LPFB) with a higher degree of block in the Posteroinferior Fascicle related to Left Anterior Fascicle.

• CLBBB associated to lateral infarction (free wall of the left ventricle)

• CLBBB associated to right ventricular hypertrophy (CLBBB+ RVH)

• CLBBB with accidental exchange of limb electrodes.

CLASSIFICATION OF CLBBB ACCORDING TO ELECTRICAL AXIS OF QRS COMPLEX IN THE FRONTAL PLANE

1. QRS axis not deviated: between - 30º and + 60º (≈ 65% to 70% of cases)
2. QRS axis with extreme deviation to the left: beyond - 30º (≈25% of cases)
3. QRS axis deviated to the right: between + 60º and + 90º (≈ 3.5 a 5% of cases)
4. QRS axis with extreme deviation to the right: beyond + 90º (< than 1% of cases). It is named "paradoxical type of Lepeschkin".

• Normal or left-axis deviation of SÂQRS, the orientation of the latter ranging from + 90º to - 30º in the frontal plane reference frame. A normal axis or left-axis deviation are noted with equal frequency in CLBBB(1).

• CLBBB is usually associated with normal or left axis deviation. Rarely the ECG shows an CLBBB with changing QRS morphology and changing axis deviation.

• Atrial fibrillation can to produce intermittent right axis deviation in the presence of CLBBB(2).

• A tachycardia with CLBBB morphology and right axis deviation points to the diagnosis of VT. Conversely, any supraventricular tachycardia with LBBB is typically associated with a normal or leftward QRS axis(3).

Types of CLBBB according to SAQRS in the frontal plane (percentage of axis location).
LBBB AND RIGHT AXIS DEVIATION
FREQUENCY, CAUSES, AND CONSEQUENCES

Right axis deviation with CLBBB is a rare combination. From a database of 636,000 ECGs Chiders et al (1) report a series of 36 patients with this combination.

• The majority of subjects had dilated cardiomyopathy with biventricular enlargement. Right axis deviation with CLBBB is a marker of congestive cardiomyopathy(2).
• LBBB was fixed in 21 of 36 cases. It was freshly acquired, episodic, intermittent, or physiologic in 15 of 36.
• The right axis deviation was episodic in 30 of 36. It was fixed and concurrent with LBBB in only 2 cases, and never episodically concurrent.
• Reported for the first time here were 4 of 36 cases in which the combination of LBBB and right axis deviation was elicited with atrial premature impulses as a rare form of QRS aberration.
• In one case where the combination was intermittent, a clear relationship with freshly acquired intermittent left posterior fascicular block was demonstrated. The possible relationship of the deviation with variable degrees of right ventricular overload is discussed.
• Review of patients from the literature since 1950 until 1985 indicates that the uncommon combination of LBBB and right axis deviation is a marker of severe myocardial disease, especially primary congestive cardiomyopathy. The mechanism of production of this ECG pattern appears to be diffuse conduction system involvement in advanced myocardial disease

FASCICULAR LBBB (LAFB + LPFB) WITH A HIGHER DEGREE OF BLOCK IN THE POSTERIOR INFERIOR DIVISION OR FASCICLE

Outline that explains CLBBB with axis to the right, secondary to divisional block: complete LPFB associated to incomplete LAFB.
• The intermittent form of CLBBB with right axis deviation is suggestive of fascicular LBBB: incomplete Left Anterior Fascicular Block (LAFB) associated with complete Left Posteriorinferior Fascicular Block (LFPB) (1).

• Rarely the ECG shows an CLBBB with changing QRS morphology and changing axis deviation.

• The intermittent positive aspect of the neglected lead aVR indicates an intermittent right axis deviation in the presence of complete LBBB. The phenomenon is observed only when the QRS axis is beyond + 120°.

• An additional LPFB accompanying predivisional LBBB is the possible explanation.

• Permanent atrial fibrillation and LBBB with intermittent left axis deviation or intermittent right axis deviation is possible by alternating fascicular or divisional block(2).


When electrocardiography was starting, Wilson postulated that the S wave of $V_6$ in the LBBB associated to lateral infarction was due to the sensing by the exploring electrode of $V_6$ of intracavitary potential of the LV (RS): it is called the “electric window” of Wilson. Today we know that the afferent limb is dislocated to right of the efferent limb.
THE AFFERENT LIMB IS DISLOCATED TO THE RIGHT OF THE EFFERENT LIMB ON HP

LBBB + FREE WALL MYOCARDIAL INFARCTION

ECG/VCG CORRELATION HORIZONTAL PLANE

Wide S wave

Cabrera’s sign

NOTCH
CABRERA’S SIGN: LBBB COMPLICATED WITH ANTERIOR MI

Notch of 50 ms in the ascending ramp of S wave of V₃ and V₄. It is seen more often with MI than without (anterior more often than inferior), and the left axis increased its sensitivity.


The appearance of the S wave in V6 in LBBB associated to lateral MI, is due to a dislocation to the right of the Z line of the afferent limb of QRS loop and not to the sensing of the intracavitary potential. The fact that the S wave appears broadened (>40 ms) and with a notch, reinforces this position.

Comparison of vectorcardiographic QRS/T loops in the horizontal plane in isolated LBBB and associated to free wall infarction. The QRS loop in isolated LBBB has a morphology in "eight" and is found in the left posterior quadrant and its main rotation is clockwise. There is middle final delay. The T loop is heading to the front and the right in the right anterior quadrant, with a QRS/T angle near 180°. In the QRS loop of LBBB complicated with LV free wall infarction, the main rotation is inverted (counterclockwise) with the afferent branch located to the right from the efferent one. Additionally, a dislocation of the QRS loop is observed to the right, with half to the left and half to the right: R=S in V6 with broad S wave in V6 and a duration ≥ 40 ms. The T loop is usually located in the left anterior quadrant.
DIFFERENTIAL DIAGNOSIS OF QRS LOOP IN HP IN ISOLATED CLBBB AND CLBBB ASSOCIATED WITH RVH
QRS PATTERN IN LEFT PRECORDIAL LEADS V5-V6: rS PATTERN
Name: ASC; Gender: Male; Age: 54 y.; Ethnic group: White; Weight: 86Kg; Height: 1.68 m; Biotype: Endomorph;

Date: 04/03/2003; Medication in use: Enalapril 10 mg 2X + Atenolol 50 mg + Chlortalidone 12.5 mg.

Clinical diagnosis: Hypertensive heart disease + aortic insufficiency by aortic cause.
Echo diagnosis: Moderate concentric hypertrophy: septum 13 mm and posterior wall 14 mm. Moderate aortic insufficiency.
ECG diagnosis: SR; HR: 72 bpm; SAP: +60°; SAQRS: +110°; QRSD: 165 ms; DI and aVL = rS; DIII = qR; RIII > RII.
CONCLUSION: 1) CLBBB; 2) LPFB (Left Posterior Fascicular Block).

Typical example of an ECG with SAQRS deviated to the right by associated left posterior fascicular block.
Which is the electrocardiographic foundation for CLBBB diagnosis?

- In the presence of supraventricular command (sinus rhythm) the duration of the QRS complex >120 ms (in this case 165 ms);

- QRS complexes totally or predominantly negative in right precordial leads;

- T wave opposite to QRS complex with an angle near the 180°.
COMMENTS

Which is the electrocardiographic foundation for LPFB diagnosis?

- SAQRS deviated to the right in clinical absence of RVE, verticalized heart or lateral infarction;
- QRS complexes of the rS type in DI and aVL;
- Complexes of the qR type in inferior leads;
- R wave of DIII > than R wave of DII;
- There are references in literature to aortic insufficiency by regurgitant jet, which thrown on the postero-inferior wall may cause LPFB. On the other hand, the CLBBB has as its most frequent cause high blood pressure.

An accurate diagnosis of LPFB must obligatorily be clinical and electrocardiographic, as in this case, in which in an obese, endomorph, hypertensive patient, the SAQRS is in +115°.

Typical example of an ECG with SAQRS deviated to the right by associated left posterior fascicular block.
ECG/VCG correlation in the frontal plane of CLBBB with axis deviated to the right in the FP by LPFB.
ECG/VCG correlation in the horizontal plane of CLBBB with axis deviated to the right in the FP by LPFB.