7 QRS Morphologies of Difficult Interpretation

Agnese Fioranelli, Enrico Paolini, and Alessia Quaranta

7.1 Case 1: Hemiblocks

A preoperative ECG of a 48-year-old male affected by dyslipidemia, without any previous cardiological pathologies, was recorded. He complained of occasional palpitations (Fig. 7.1).

7.1.1 ECG Analysis

Regular rhythm at 67 bpm; every QRS is preceded by a P wave with normal axis, amplitude, and duration typical of a sinus rhythm; the atrioventricular conduction is at the upper limit (PR 200 ms); QRS duration is slightly prolonged (110 ms).

There is a left axis deviation −60° with poor R wave progression on the precordial leads and a delayed intrinsicoid deflection in aVL (60 ms), a terminal R wave in aVR, and a biphasic complex in V6. These findings suggest a left anterior fascicular block.

The QTc is slightly prolonged (Bazett = 465 ms).

A. Fioranelli · E. Paolini (✉) · A. Quaranta
Clinica di Cardiologia e Aritmologia,
Università Politecnica delle Marche,
Ancona, Italy

7.1.2 From ECG to Clinic

When a complete left axis deviation (≥ −30°) is recorded, there are usually two main causes:

(1) A simple left anterior fascicular block (or hemiblock, LAH) where the QRS complexes have an rS morphology
(2) Inferior or inferolateral myocardial infarction; with a possible Q wave in leads II, III, and aVF (5%)

Other possible clinical conditions coming together with a left axis deviation are:

(3) Left ventricular hypertrophy
(4) Ventricular preexcitation
(5) Pulmonary emphysema (COPD) (Table 7.1)

Some authors [1] further distinguished LAH in complete and incomplete; that may be apparent when premature beats are conducted with different degrees of LAH with a different QRS axis and S wave amplitude in II and III.

The electrocardiographic and vectorcardiographic expressions of LAH may have a high variability secondary to the fascicular speed and its anatomy (Fig. 7.2). It could be possibly more appropriate to substitute the term "block" with simply "delay" [2].

A. Capucci (ed.), *New Concepts in ECG Interpretation*,
https://doi.org/10.1007/978-3-319-91677-4_7

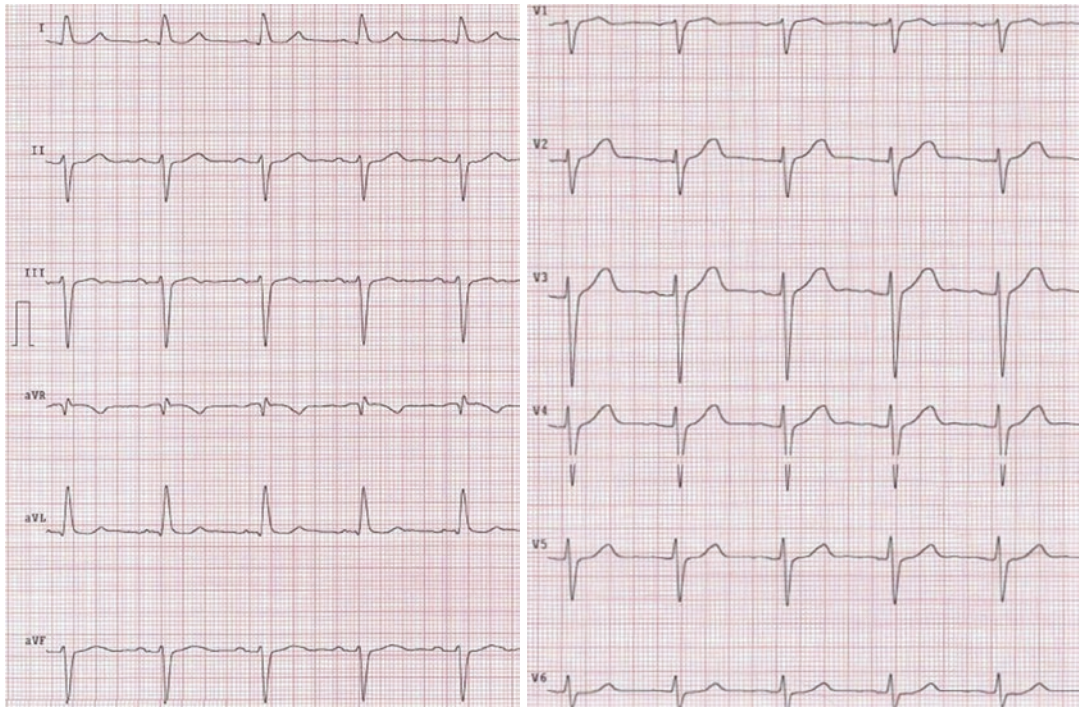

Fig. 7.1 Case 1 12-lead ECG

Table 7.1 Case 1 ECG features of LAH

ECG features of LAH	Additional findings
Left axis deviation of at least −30°[a]	Delayed intrinsicoid deflection (≥50 ms) in aVL
S wave II < III	Slurred R downstroke in I and/or aVL
qR complexes in aVL and I	Rs or rS without Q waves in V6[b]
rS complexes in III and aVF, and rS or RS in II	QRS duration ≤0.12 s
Secondary repolarization abnormalities of ST-T in I and aVL	r terminal wave in aVR

[a]In opinion of some authors, the lower limit should be set at -45° because of its specificity
[b]Or from V4–V6

Figure 7.3 reports a minor grade of left axis deviation (−30°) due to left anterior hemiblock. The AV conduction is at upper normal limit, and there is a slightly prolonged QTc. Are those figures clinically relevant features when compared to Fig. 7.1?

The prognostic value of a left anterior hemiblock depends on the clinical context. Previous studies [3, 4] that evaluated its prognostic significance in a general population with structurally normal hearts did not report any relationship with long-term mortality.

Biagini et al. [5] instead found that LAH is an independent predictor of total (1.5-fold) and cardiac (2.5-fold) mortality in a special patient population referred for a dobutamine stress test. Patients with LAH had a 10% higher incidence of ischemia at dobutamine stress echocardiography.

More recently, pre-existing isolated LAH was found to be associated with a higher incidence of permanent pacemaker implantation after a transaortic valve implantation (TAVI) [6].

However its natural history is benign since only 6% of LAH in people without apparent heart disease will evolve to complete left bundle branch block.

The latest European Guidelines [7] advise against preventive PMK implantation in asymptomatic patient with any bundle branch block (class 3, level of evidence B).

Fig. 7.2 Case 1 Diagrammatic sketches of the left-sided conduction system as observed in 20 normal hearts [taken from J.C.Demoulin and H.E. Kulbertus, 1972. Histopathological examination of concept of left hemiblock. Heart. 34(8) under concession of BMJ]

Possible misdiagnosis:

1. A right bundle branch block may mimic LAH; in that case, a possible wrong diagnosis of bifascicular block can be done.
2. A "standard lead" masquerading right bundle branch block coexisting with LAH.
3. A "precordial" masquerading right bundle branch block coexisting with LAH.

In the first case, the anterior direction of the terminal forces is missing, so an rSr' complex in precordial leads may be absent, and an rs or rS complex appears in II.

A close look at Fig. 7.4 reveals that S2 > S3 and there is a little s wave in aVL; those patterns are not typical of a simple LAH.

In the second and third cases, a right bundle branch block is masquerading by a left anterior hemiblock (respectively, Figs. 7.5 and 7.6):

- In the standard leads masquerading, there aren't S delayed waves in I and aVL.
- In the precordial masquerading, there are rSR' complexes in right precordial leads.

7.1.3 Left Posterior Hemiblock

Left posterior hemiblock is a rare finding.

Its electrocardiographic diagnosis is mainly made after a right ventricular pathology exclusion.

In a long-limbed patient, for instance, this diagnosis could be overdone since a right axis deviation could be due simply to the vertical heart position, in the absence of a true conduction delay.

In Fig. 7.7, there is an example of an ECG of a 31-year-old female: she was referred to the cardiologist for clinical evaluation before starting a physical training at the gym.

She was completely asymptomatic.

7.1.4 ECG Analysis

Narrow complex regular tachycardia; every QRS is preceded by a P wave with normal axis (+75°), high amplitude (0.25 mV in lead II), and normal duration typically of sinus origin. HR is 102 bpm; atrioventricular conduction is normal (PR = 180 ms); there is a small PR segment depression.

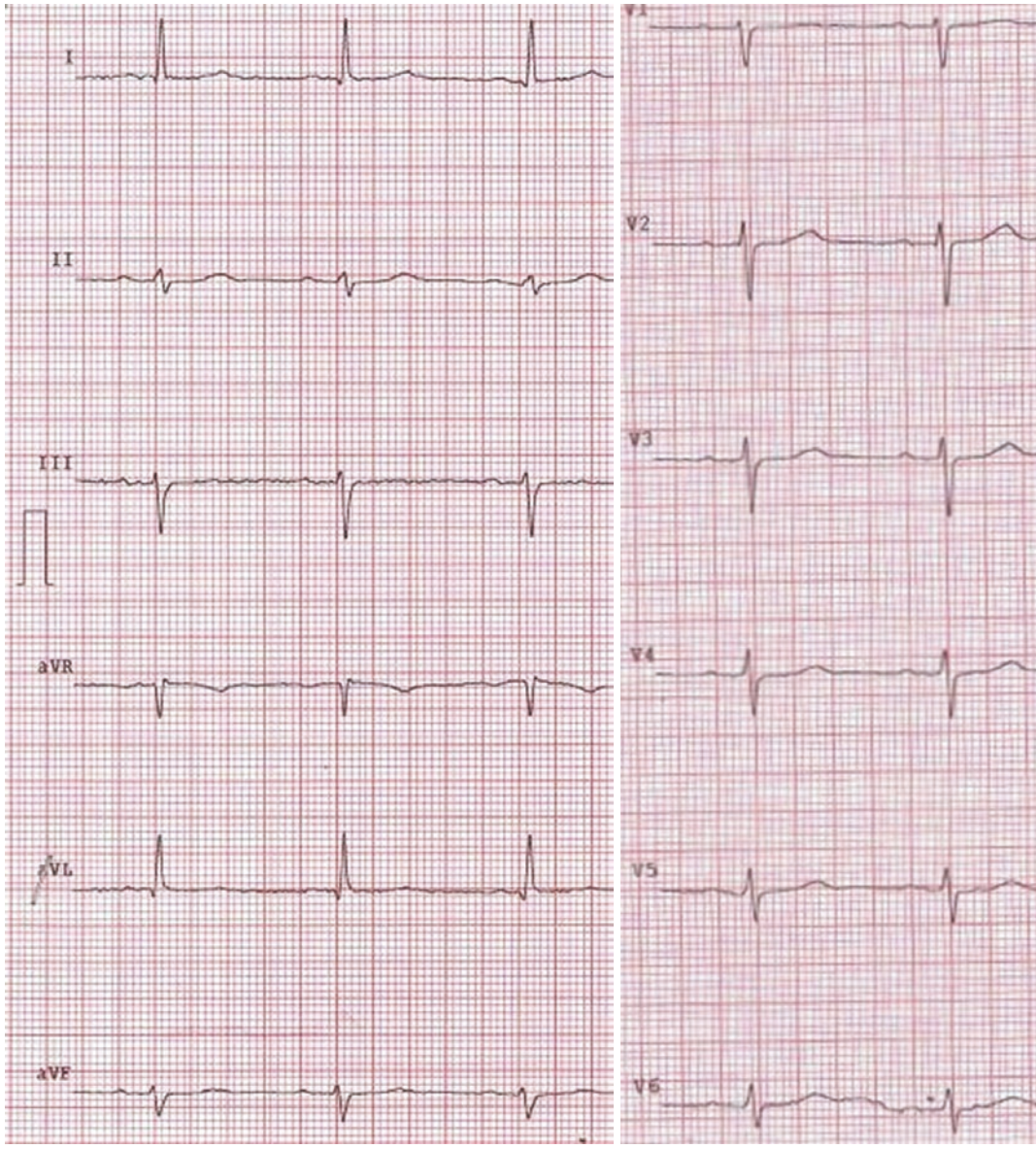

Fig. 7.3 Case 1 12-lead ECG

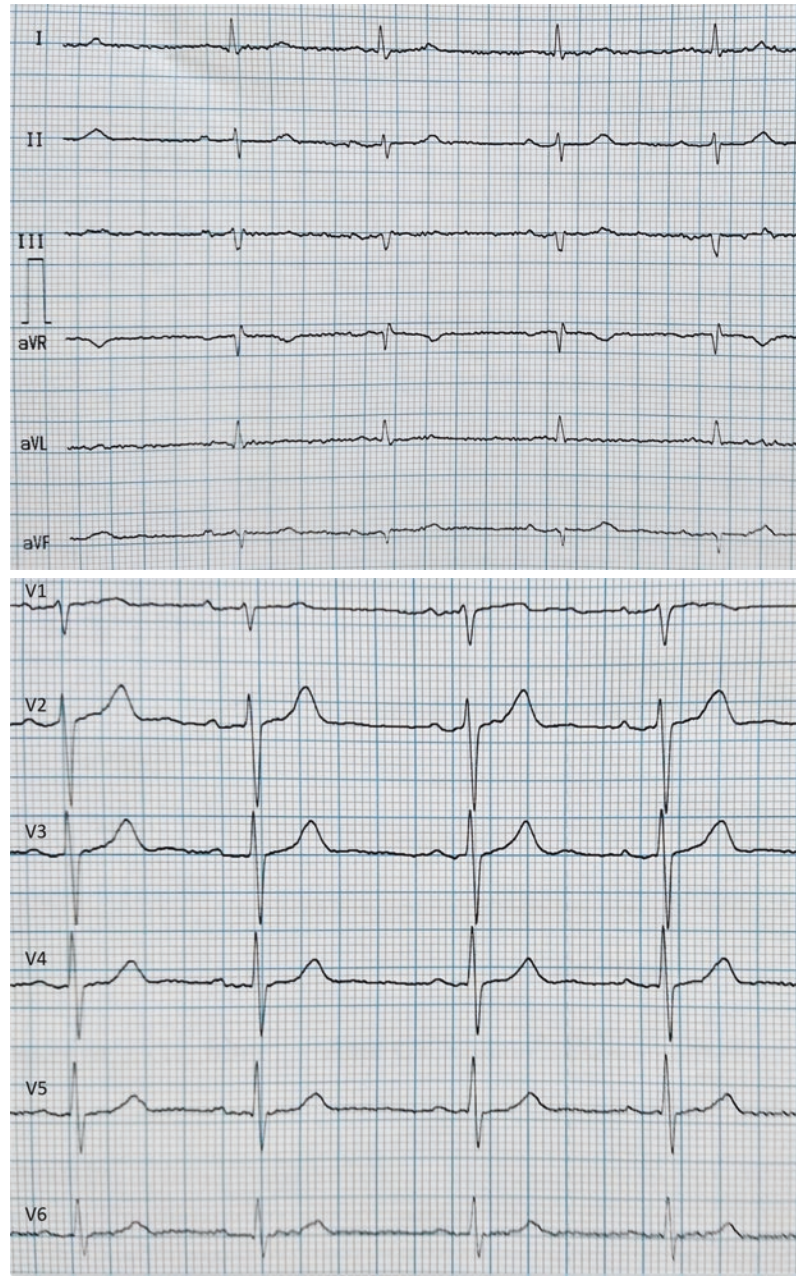

Fig. 7.4 Case 1 12-lead ECG

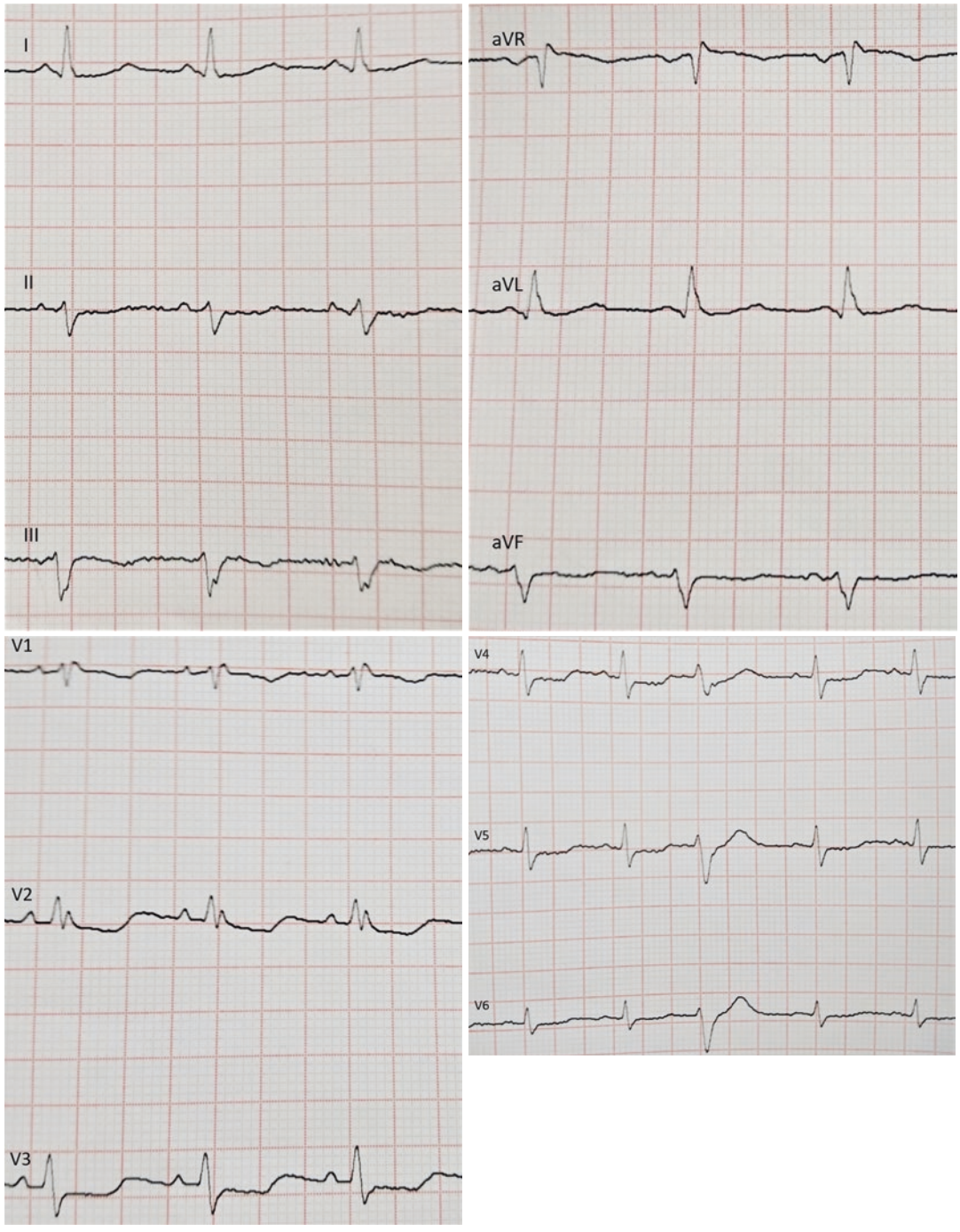

Fig. 7.5 Case 1 12-lead ECG

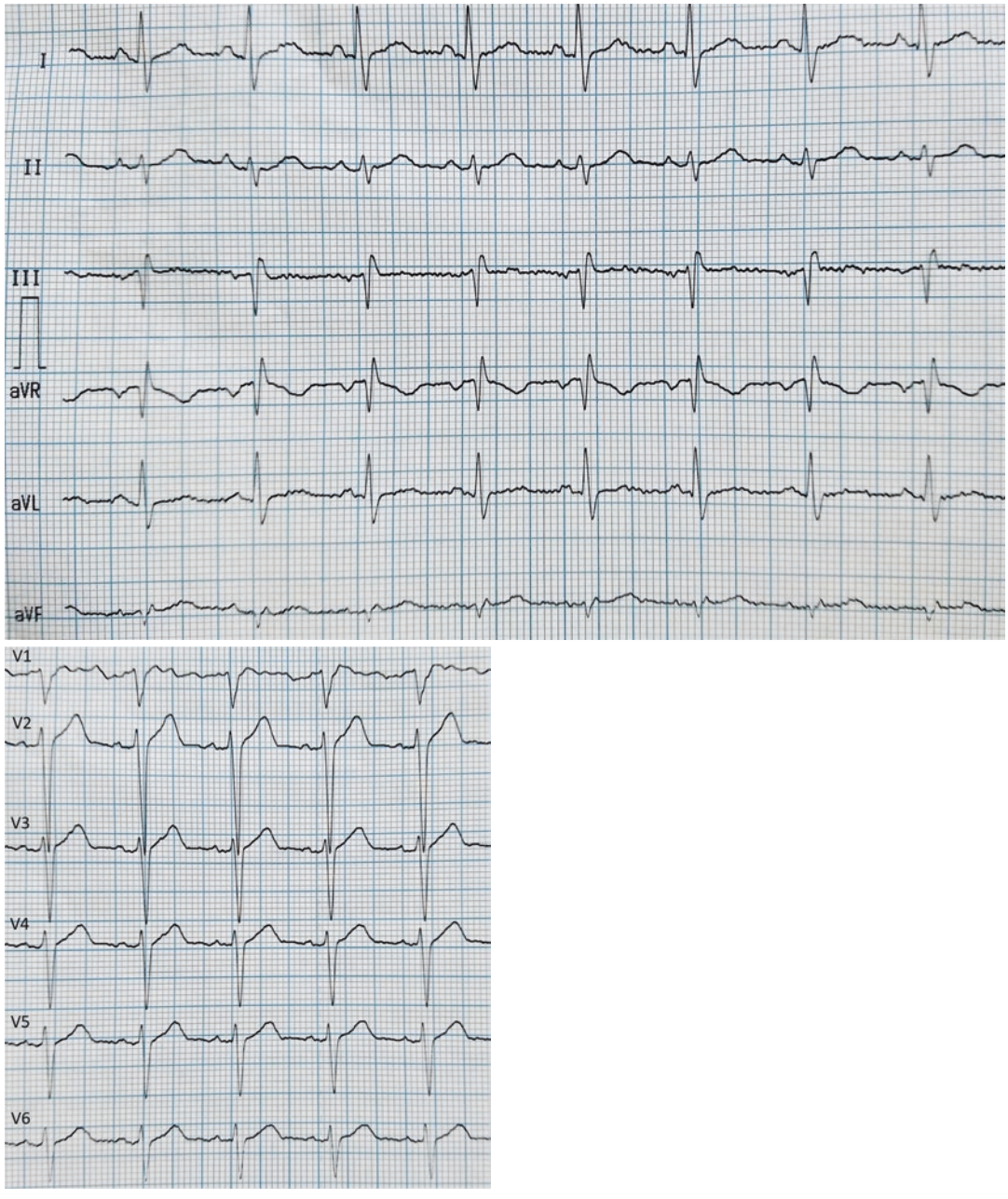

Fig. 7.6 Case 1 12-lead ECG

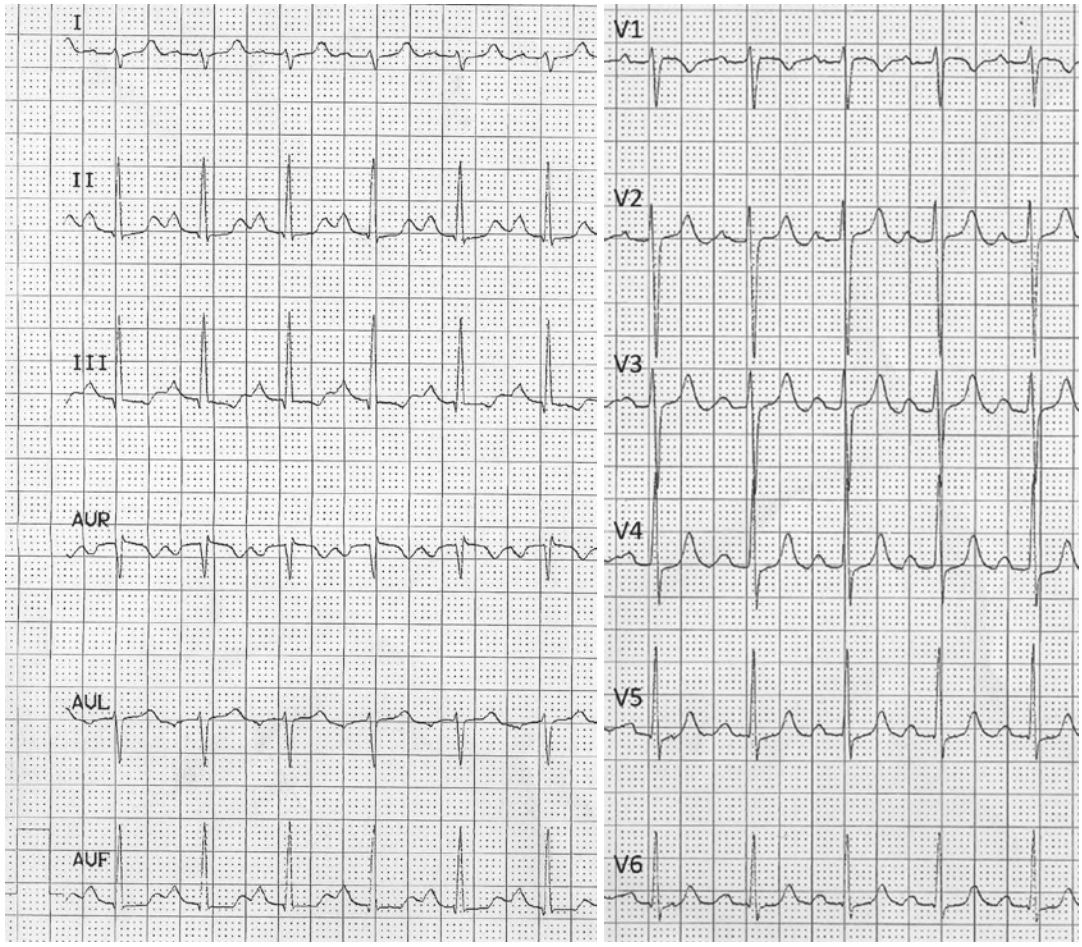

Fig. 7.7 Case 1 12-lead ECG

QRS duration is normal, with a right axis deviation (+115°).

There are diffuse ST-T alterations: flat ST segment with biphasic T waves in III and aVF, symmetrical in V2-V5.

QTc is normal (430 ms).

In summary, the ECG shows sinus tachycardia, normal AV conduction, right axis deviation secondary to left posterior fascicular hemiblock, nonspecific repolarization abnormalities, and normal QTc.

7.1.5 From ECG to Clinic

In the absence of any pathology, an echocardiogram was also recorded and confirmed a normal right ventricular morphology, dimensions, and function and normal pulmonary artery dimensions and gradients; there was only a mild mitral regurgitation due to anterior leaflet prolapse.

A pectus excavatum was present; that anatomical pattern by changing the normal topography of the chest/heart could influence on the axis deviation and thus question the diagnosis of left posterior hemiblock.

The diagnostic reported classical signs of this rare conduction disturbance are:

- Right axis deviation (>90°)
- qR complexes in II, III, and aVF
- ST-T inversion in the same leads
- Q wave absence in V6
- aVF intrinsicoid deflection delay slower than in V6
- QRS duration ≤120 ms

We believe that this reported ECG is a normal variant in a young woman with mild mitral valve prolapse and without other abnormal findings.

The diagnosis of left posterior hemiblock remains a conundrum, which is possible to solve only after an exclusion criteria work-up.

7.2 Case 2: Pathological and Physiological Q Waves

In Table 7.2, the common conditions that favor Q waves on the surface ECG are reported [8–10].

In Wolff-Parkinson-White (WPW) preexcitation a pseudo-infarction pattern may be a common finding in up to 70% of patients. That is due to negative delta waves in the inferior/anterior leads ("pseudo-Q waves") or to a prominent R wave in V1–3 (mimicking a posterior infarction).

Inferior lead pseudo-infarct Q waves are a common finding in the Wolff-Parkinson-White (WPW) syndrome.

In a retrospective study [11], pseudo-infarct Q waves in the inferior leads were associated with positive or isoelectric T waves in 47 of 50 examples (94%).

Table 7.2 Features of Case 2 pathological and physiological Q waves

Pathological Q waves
Physiologic and positional effects: *dextrocardia, rightward mediastinal shift in left pneumothorax, pectus excavatum, COPD, corrected transposition of the great vessels, congenital absence of the left pericardium*
Ventricular enlargement
Altered ventricular conduction: *left bundle branch block, WPW*
Misplacement of chest lead electrodes
Stress-induced (Tako-Tsubo) cardiomyopathy
Myocardial ischemia (without infarction)
Sign of previous myocardial infarction: *pathological Q waves are 25% or more of the height of the partner R wave, and they are greater than 40 msec in width and greater than 2 mm in depth*
Physiological Q waves
Physiologic activation of the ventricles begins at the left side of the interventricular septum: small "septal" Q waves occur in the lateral leads
QS complex can appear in aVL with a vertical axis and in leads III and aVF with a horizontal axis
Q wave in lead III may be positional and a normal finding

This characteristic Q wave-T wave vector discordance may be related to a nonhomogeneous ventricular activation.

A further cause of pseudo-infarct pattern could be left or right ventricular enlargement [12].

Q waves in these settings may reflect a variety of mechanisms, including a change in the balance of early ventricular depolarization forces and altered cardiac geometry and position; in fact, slow R wave progression in the precordial leads is commonly observed either in left ventricular hypertrophy or in right ventricular overload.

The acute pulmonary embolism develops the classic S1Q3T3 pattern of McGinn and White, but this sign is neither sensitive nor specific for pulmonary embolism (Fig. 7.8).

A prominent Q wave (usually as part of a QR complex) in lead aVF is also reported in this condition.

It was found that an acute right ventricular overload itself does not necessarily cause any pathologic Q wave in lead II [13].

7.2.1 ECG Analysis

Figure 7.9 shows a sinus tachycardia (HR 105 bpm) with PR interval of 160 ms, QRS axis of −15°, normal intraventricular conduction, 1 mm ST depression in lateral leads, biphasic T waves in leads I and II, and flattened T waves in precordial leads V3–V6. There is a classic pattern S1Q3T3.

The polarity of T waves may be useful in the differential diagnosis between hypertrophic cardiomyopathy and previous myocardial infarction.

T wave will be usually upright in an ECG of a patient with Q waves secondary to hypertrophy.

In case of myocardial infarction, prominent Q waves may be associated either with upright or also inverted T waves.

Figure 7.9 represents an equivocal ECG: there is a sinus rhythm at 55 HR, the QRS axis is −15°, and there is normal AV and IV conduction and nonspecific repolarization pattern.

There are clear Q waves in the inferior leads that could be related to previous infarction.

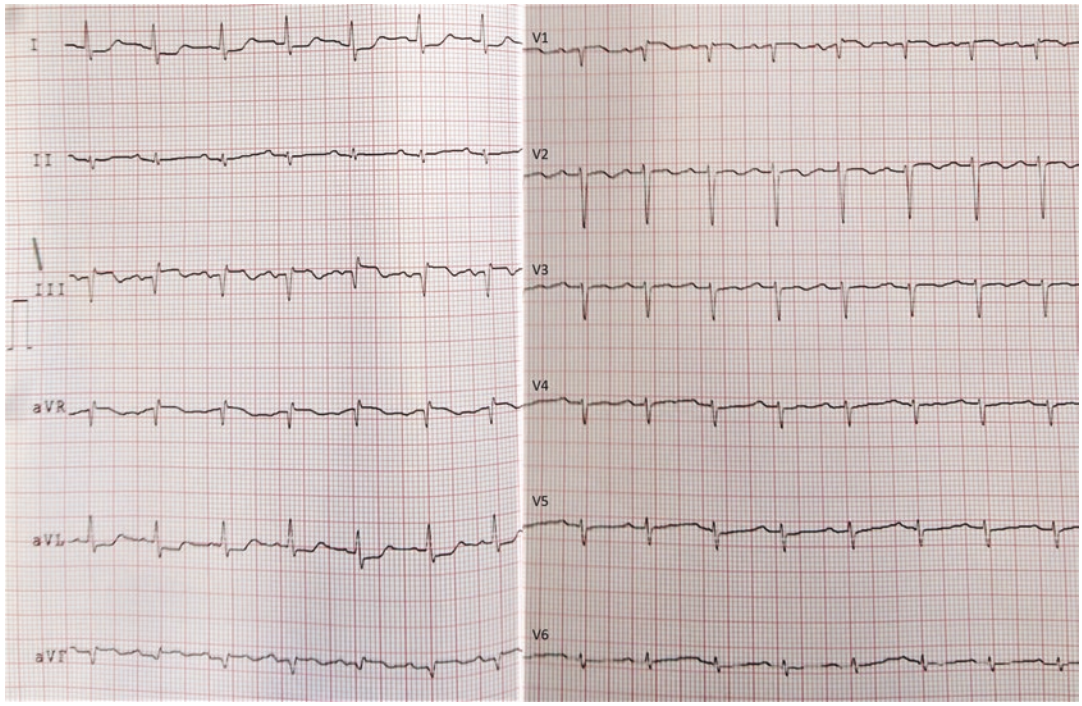

Fig. 7.8 Case 2 12-lead ECG

In Fig. 7.10 is shown the same ECG repeated during a deep inspiration.

7.2.2 ECG Analysis

Sinus rhythm at 60 bpm; QRS axis is −15°; normal atrioventricular and intraventricular conduction; Q wave in lead III with an r appearance in aVF (look at the arrow).

7.2.3 From ECG to Practice

Bodenheimer et al. [14] studied the effect of deep inspiration on Q waves in leads III and aVF in 31 patients by comparing with their cardiac catheterization results.

They found that the phasic inspiration can significantly influence Q wave duration in leads III and aVF, regardless of the presence or absence of significant associated coronary artery disease and asynergy. When that happens, those Q waves have no relevant values.

Nanni et al. [15] evaluated the diagnostic accuracy of electrocardiographic inferior Q wave persistence during inspiration by comparing with echocardiographic segmental wall motion abnormalities and using cardiac magnetic resonance as the gold standard.

They prospectively enrolled 50 apparently healthy subjects with inferior Q waves on routine electrocardiogram and high atherosclerotic risk profile.

From ten positive cardiac magnetic resonance subjects, eight showed persistence of inferior Q waves during inspiration, giving a sensitivity of 80% and a specificity of 95%.

The conclusion was that the inferior Q wave persistence during deep inspiration is a simple test with a high accuracy for the diagnosis of silent myocardial infarction, while the standard echocardiography result is less accurate.

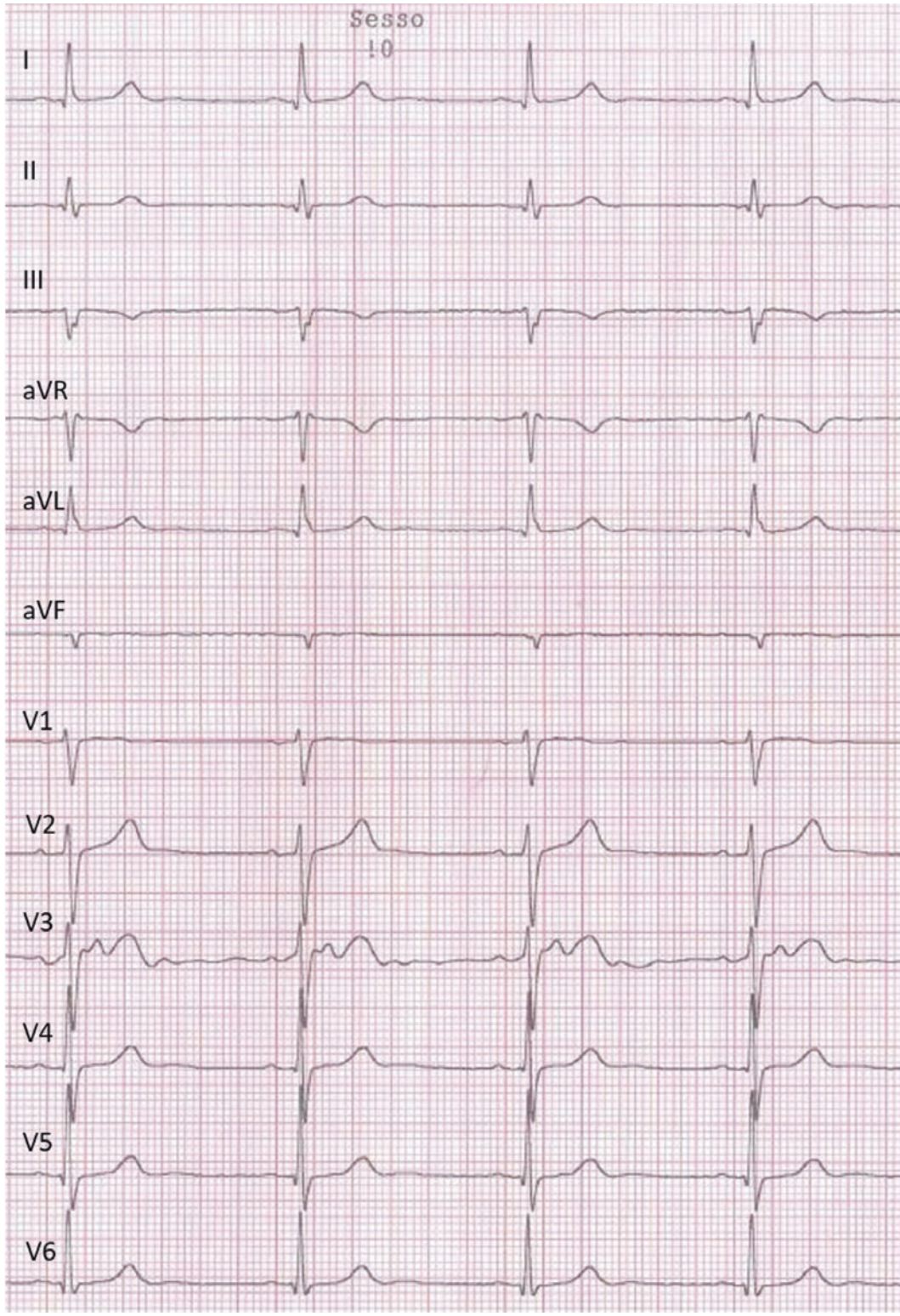

Fig. 7.9 Case 2 12-lead ECG

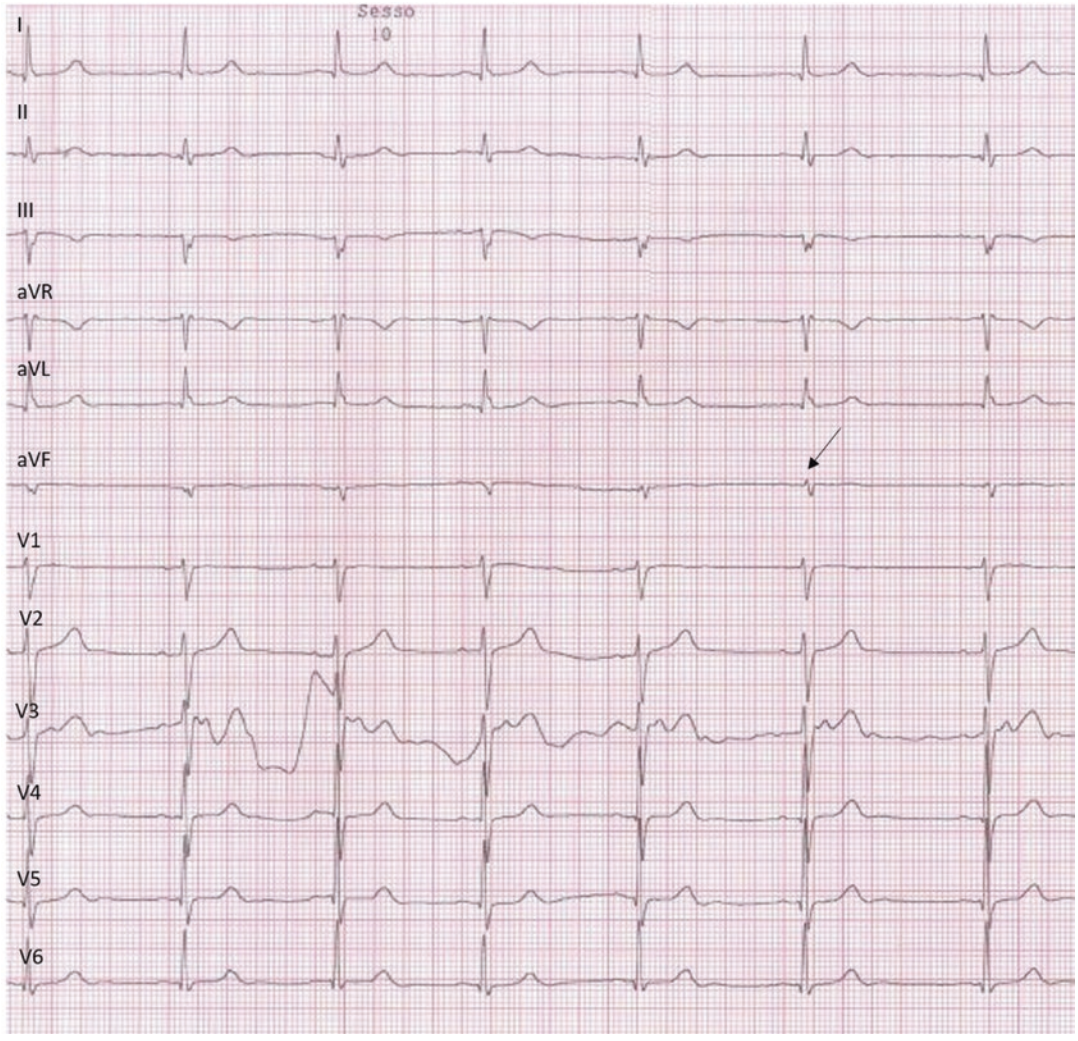

Fig. 7.10 Case 2 12-lead ECG

7.3 Case 3: rSr′ Complexes in the Precordial Leads

Male, 30 years old, without previous cardiovascular pathologies. Normal familiar medical history and physical examination; no echocardiographic signs of structural heart disease.

In Fig. 7.11 is shown his ECG:

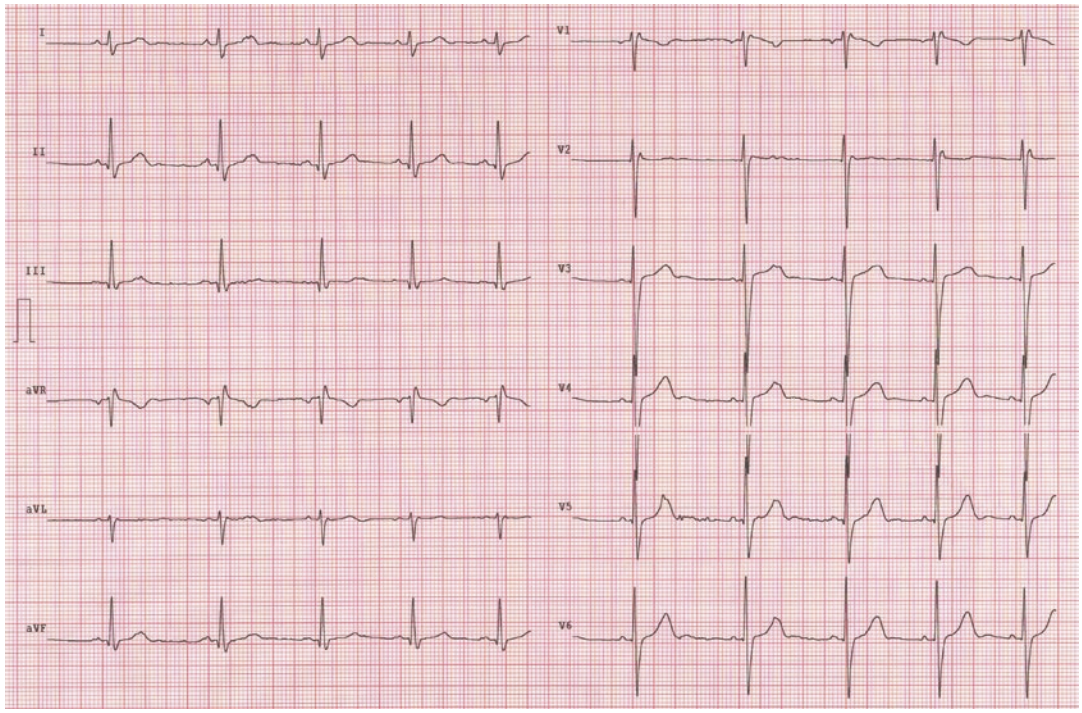

Fig. 7.11 Case 3 12-lead ECG

7.3.1 ECG Analysis

Regular rhythm is at 60 bpm; every QRS is preceded by a P wave with normal axis, amplitude, and duration, thus expression of sinus rhythm; the atrioventricular conduction is normal (PR 120 ms). QRS duration is normal (80 ms), showing rSR' morphology in the right precordial leads (V1-V2); QRS axis is vertical (+90°) with a clockwise QRS rotation on the longitudinal axis; there is an S wave in lead I and V6 and a wide terminal R wave in aVR.

QTc is normal (400 ms).

In summary, the ECG shows a sinus rhythm with normal atrioventricular conduction, incomplete right bundle branch block, and secondary repolarization abnormalities.

7.3.2 From ECG to Practice

Incomplete RBBB diagnosis is mainly QRS morphology related because the ventricular activation length could be in total poorly prolonged despite a significant delay already in the right ventricle [16, 18] (Table 7.3).

Table 7.3 Case 3 ECG features of RBBB

ECG diagnostic criteria [17]
• rSR' complex in the right precordial leads with R' wave usually higher than initial R wave in V1–V2
• Wide S waves in I, V5, and V6
• Wide terminal R wave in aVR
• QRS duration >120 ms and S waves duration >40 ms (complete RBBB)
• QRS duration <120 ms and S waves duration <40 ms (incomplete RBBB)

V1 QRS morphology depends on the amount of activation delay.

However, the increased conduction time can be related not only to a slower conduction velocity but also to an abnormal long right bundle branch or to a right ventricular outflow hypertrophy [19].

The QRS axis is usually normally oriented in RBBB, but there could be a mild right axis deviation or an undetermined axis.

A rSr' complex only in V1 (not extended to V2) could be often found in normal young people as a normal variant.

Some authors [20] identified the following features in the right precordial leads (V1, V2) that precede or accomplish the appearance of the rSr' as RBBB findings:

- Reduced S wave depth (100%)
- Inverted ratio of the S wave depth to SV1 > SV2 (93%)
- Slurred downstroke or upstroke of the S wave (27%)
- Prolonged QRS duration >0.10 s (73%)

The rSR' pattern is not associated with an increased risk of cardiovascular diseases and therefore may be considered as a benign finding [21].

The RBBB pattern could be confused so far with the "Brugada syndrome" since the last is characterized by a rSr' complex in lead V1 that resembles an incomplete RBBB.

Nevertheless, r' wave of the QRS complexes of Brugada pattern is not a sign of delayed activation of the right ventricle, but it is an expression of a repolarization abnormality with a consequent delayed initial repolarization, and it should be called more appropriately "J" wave [22].

Thus ST-segment elevation in Brugada and terminal r' wave are not synchronized with the extended S wave in leads I and V6.

Below (Fig. 7.12) there is an example of Brugada pattern.

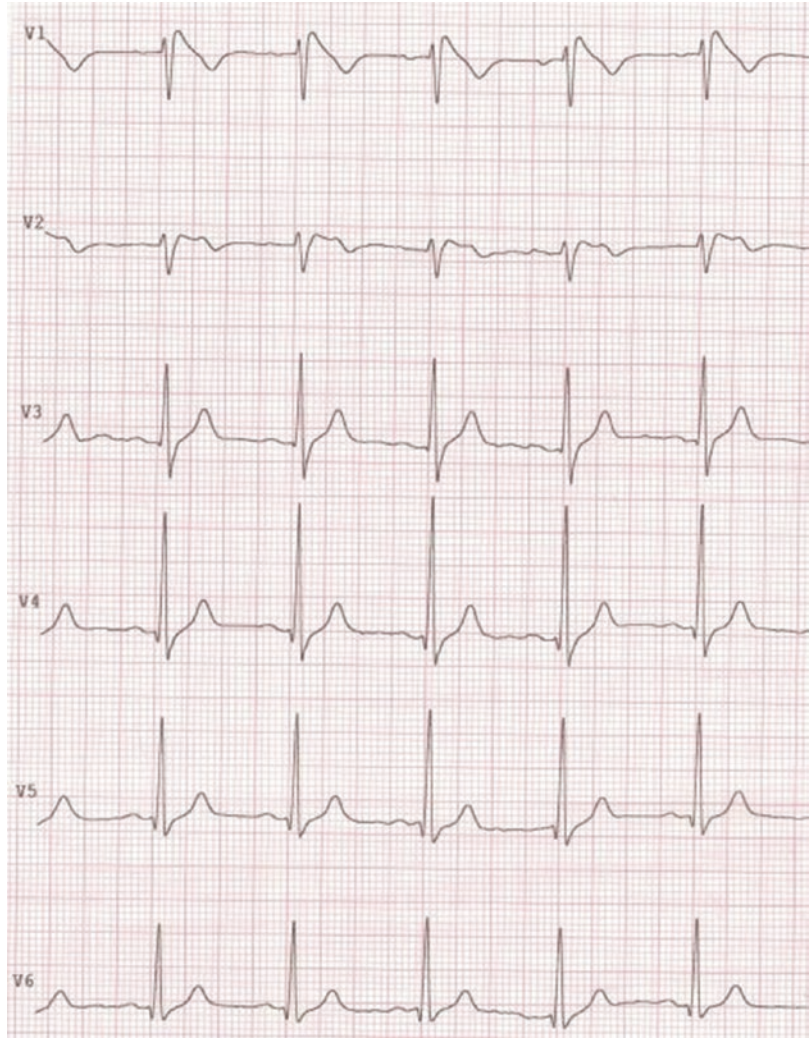

Fig. 7.12 Case 3 Chest lead ECG

7.3.3 ECG Analysis

Sinus rhythm with normal atrioventricular conduction; normal QRS duration of 120 ms with rSR' pattern in lead V1 and S wave in V6.

In V1 and V2, there are few features that can lead to the correct diagnosis:

- In lead V1, there is a J-point elevation of 3 mm and coved-type descending ST-segment elevation which merges into a negative T wave.
- In lead V2, there is J-point elevation (2 mm), a saddleback ST-segment elevation (1 mm in its terminal portion), and a biphasic T wave.

The V1 features are typically a Brugada type 1 pattern, whereas the V2 findings are typical of a Brugada type 2 pattern. The difference with an incomplete RBBB is striking.

This ECG belongs to a young man with a clear Brugada syndrome.

References

1. Elizari MV, Chiale PA. The electrocardiographic features of complete and partial left anterior and left posterior hemiblock. J Electrocardiol. 2012;45(5):528–35.
2. Piccolo E, Raviele A. L'emiblocco anteriore sinistro 40 anni dopo rosenbaum. Rel.16/2008 6-02-2008. 2008. Available from: http://atti.centrolottainfarto.org/anno/2008/pdf/Piccolo 16_2008.pdf. Last Access 2018 Jan 27.
3. Ostrander LD. Left axis deviation: prevalence, associated conditions, and prognosis. Ann Intern Med. 1971;75(1):23.
4. Yano K, Peskoe SM, Rhoads GG, et al. Left axis deviation and left anterior hemiblock among 8,000 Japanese-American men. Am J Cardiol. 1975;35(6):809–15.
5. Biagini E, Elhendy A, Schinkel AFL, et al. Prognostic significance of left anterior hemiblock in patients with suspected coronary artery disease. J Am Coll Cardiol. 2005;46(5):858–63.
6. Siontis GCM, Jüni P, Pilgrim T, et al. Predictors of permanent pacemaker implantation in patients with severe aortic stenosis undergoing TAVR: a meta-analysis. J Am Coll Cardiol. 2014;64(2):129–40.
7. Brignole M, Auricchio A, Baron-Esquivias G, et al. ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Europace. 2013;15(8):1070–118.
8. Goldberger AL, Goldberger ZD, Shvilkin AS. Goldberger's clinical electrocardiography: a simplified approach. 9th ed. Philadelphia: Elsevier; 2017.
9. Mirvis DM, Goldberger AL. Electrocardiography. In: Zipes DP, Libby P, Bonow RO, et al., editors. Braunwald's heart disease: a textbook of cardiovascular medicine. 11th ed. Philadelphia: W.B. Saunders; 2017.
10. Delewi R, Ijff G, van de Hoef TP, et al. Pathological Q waves in myocardial infarction in patients treated by primary PCI. JACC Cardiovasc Imaging. 2013;6:324.
11. Goldberger AL. Pseudo-infarct patterns in the Wolff-Parkinson-White syndrome: importance of Q wave-T wave vector discordance. J Electrocardiol. 1980;13(2):115–8.
12. Pirwitz MJ, Lange RA, Landau C, et al. Utility of the 12-lead electrocardiogram in identifying underlying coronary artery disease in patients with depressed left ventricular systolic function. Am J Cardiol. 1996;77:1289.
13. Kucher N, Walpoth N, Wustmann K, Noveanu M, et al. QR in V1-an EKG sign associated with right ventricular strain and adverse clinical outcome in pulmonary embolism. Eur Heart J. 2003;24(12):1113–9.
14. Bodenheimer MM, Banka VS, Helfant RH. Determination of lead III Q waves significance utility of deep inspiration. Arch Intern Med. 1977;137(4):437–9.
15. Nanni S, et al. Inferior Q waves in apparently healthy subjects: Should we take a deep breath? An electrocardiographic, echocardiographic and cardiac magnetic resonance study. J Electrocardiol. 2016;49(1):46–54.
16. Goldman MJ. Principles of clinical electrocardiography. Los Altos: Lange; 1988.
17. Agarwal AK, Venugopalan P. Right bundle branch block: varying electrocardiographic patterns. Aetiological correlation, mechanisms and electrophysiology. Int J Cardiol. 1999;71(1):33–9.
18. Schamroth L. The 12 lead electrocardiogram. Oxford: Blackwell; 1989.
19. Schamroth L, Myburgh DP, Schamroth CL. The early signs of right bundle branch block. Chest. 1985;87:180.
20. Mauric AT, Samani NJ, de Bono DP. When should we diagnose incomplete right bundle branch block? Eur Heart J. 1993;14(5):602–6.
21. O'Neal WT, Qureshi W, Li Y, et al. RSR' pattern and the risk of mortality in men and women free of cardiovascular disease. J Electrocardiol. 2015;48(3):430–3.
22. Bayés de Luna A. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. J Electrocardiol. 2012;45(5):433–42.