The ECG diagnosis of myocardial infarction (MI) and ischemia in pacemaker patients can be challenging. Many of the criteria are insensitive, but the diagnosis can be made in a limited number of cases because of the high specificity of some of the criteria.

**Old myocardial infarction**

Box 1 outlines the difficulties in the diagnosis of MI, and Box 2 lists a number of signs of no value in the diagnosis of MI. Generally, when using the QRS complex, the sensitivity is low (25%) and the specificity is close to 100%. One cannot determine the age of the MI from the QRS complex.

**Anterior myocardial infarction**

**St-qR pattern**

Because the QRS complex during right ventricular (RV) pacing resembles (except for the initial forces) that of spontaneous left bundle branch block (LBBB), many of the criteria for the diagnosis of MI in LBBB also apply to MI during RV pacing [1–4]. RV pacing almost invariably masks a relatively small anteroseptal MI.

During RV pacing, as in LBBB, an extensive anteroseptal MI close to the stimulating electrode will alter the initial QRS vector, with forces pointing to the right because of unopposed activation of the RV. This causes (initial) q waves in leads I, aVL, V5, and V6, producing an St-qR pattern (Fig. 1). The abnormal q wave is usually 0.03 seconds or more, but a narrower one is also diagnostic.

Occasionally the St-qR complex is best seen in leads V2 to V4, and it may even be restricted to these leads. Finding the (initial) q wave may sometimes require placing the leads one intercostal space higher or perhaps lower. Ventricular fusion may cause pseudo-infarction patterns (Fig. 2).

The sensitivity of the St-qR pattern varies from 10% to 50% according to the way data are analyzed [5,6]. Patients who require temporary pacing in acute MI represent a preselected group with a large MI, so that the overall sensitivity is substantially lower than 50% in the patient population with implanted pacemakers. The specificity is virtually 100%.

**Late notching of the ascending S wave (Cabrera’s sign)**

As in LBBB, during RV pacing an extensive anterior MI may produce notching of the ascending limb of the S wave in the precordial leads usually V3 and V4—Cabrera’s sign ≥ 0.03 seconds and present in two leads (Fig. 3) [1]. The sign may occur together with the St-qR pattern in anterior MI (see Fig. 1). The sensitivity varies from 25% to 50% according to the size of the MI, but the specificity is close to 100% if notching is properly defined [1,5]. Interestingly, workers [7] that placed little diagnostic value on q waves, found a 57% sensitivity for Cabrera’s sign (0.04-second notching) in the diagnosis of extensive anterior MI. Box 3 outlines the causes of “false” Cabrera’s signs and the highly specific variants of Cabrera’s sign (Fig. 4).

**Inferior myocardial infarction**

The paced QRS complex is often unrevealing. During RV pacing in inferior MI diagnostic Qr,
QR, or qR complexes provide a sensitivity of 15% and specificity of 100% (Fig. 5) [1,5]. The St-qR pattern must not be confused with an overshoot of the QRS complex due to overshoot of massive ST elevation creating a diminutive terminal r wave or ventricular fusion (see Fig. 5). Cabrera’s sign in both leads III and aVF is very specific, but even less sensitive than its counterpart in anterior MI (S.S. Barold, unpublished observations).

Myocardial infarction at other sites

A posterior MI should shift the QRS forces anteriorly and produce a dominant R wave in the right V leads, but the diagnosis cannot be made during RV pacing because of the many causes of a dominant R wave in V₁. An RV MI could conceivably be reflected in V₃R with prominent ST elevation. Klein and colleagues [8] suggested that the diagnosis of RV infarction could be made when there is prominent ST elevation in lead V₄R in the first 24 hours, but such a change should be interpreted cautiously unless it is associated with obvious abnormalities suggestive of an acute inferior MI.

Conflicting views on the diagnosis of myocardial infarction of uncertain age

Kochiadakis and colleagues [9] studied ECG patterns of ventricular pacing in 45 patients with old MI and 26 controls (without angiographic evidence of coronary artery disease) during temporary RV apical at the time of routine cardiac catheterization (Fig. 6). In 15 of the 26 controls, a Q wave was observed in leads I, aVL, or V₆. However, it was not specified whether the Q waves were part of a qR (Qr) or a QS complex (their Fig. 1E shows a QS complex). This differentiation is important because a QS complex carries no diagnostic value during RV pacing in any of the

<table>
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<th>Box 1. Difficulties in the diagnosis of MI during ventricular pacing</th>
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<td>1. Large unipolar stimuli may obscure initial forces, cause a pseudo Q wave and false ST segment current of injury.</td>
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<td>2. QS complexes are of no diagnostic value. Only qR or Qr complexes may be diagnostically valuable.</td>
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<td>3. Fusion beats may cause a pseudoinfarction pattern (qR/Qr complex or notching of the upstroke of the S wave).</td>
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<td>4. Cabrera’s sign can be easily overdiagnosed.</td>
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<td>5. Retrograde P waves in the terminal part of the QRS complex may mimic Cabrera’s sign.</td>
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<td>6. Acute MI and ischemia may be difficult to differentiate.</td>
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<td>7. Differentiation of acute MI and old or indeterminate age MI may not be possible on the basis of abnormalities of the ST segment.</td>
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<td>8. Signs in the QRS complex are not useful for the diagnosis of acute MI.</td>
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<td>9. ST segment changes usually but not always indicate an acute process.</td>
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<td>10. Recording QRS signs of MI may require different sites of the left V leads such as a different intercostals space.</td>
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<td>11. Biventricular pacing can mask an MI pattern in the QRS complex evident during RV pacing.</td>
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<td>12. qR or Qr complexes are common during biventricular pacing and do not represent an MI.</td>
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<td>13. Cardiac memory. Repolarization ST-T wave abnormalities (mostly T wave inversion) in the spontaneous rhythm may be secondary to RV pacing per se and not related to ischemia or non-Q wave MI.</td>
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<td>14. QRS abnormalities have low sensitivity (but high specificity).</td>
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<td>15. Beware that not all the diagnostic criteria of MI in left bundle branch block are applicable during RV pacing.</td>
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<th>Box 2. QRS criteria of no value in diagnosis of MI</th>
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<td>• QS complexes V₁ to V₆</td>
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<td>• RS or terminal S wave in V₅ and V₆</td>
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<tr>
<td>• QS complexes in the inferior leads</td>
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<tr>
<td>• Slight notching of R waves</td>
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<td>• Slight upward slurring of the ascending limb of the S wave</td>
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QR, or qR complexes provide a sensitivity of 15% and specificity of 100% (Fig. 5) [1,5]. The St-qR pattern must not be confused with an overshoot of the QRS complex due to overshoot of massive ST elevation creating a diminutive terminal r wave or ventricular fusion (see Fig. 5). Cabrera’s sign in both leads III and aVF is very specific, but even less sensitive than its counterpart in anterior MI (S.S. Barold, unpublished observations).
standard 12 leads (QS complexes can be normal in leads I, II, III, aVF, V₅, and V₆). A well-positioned lead at the RV apex rarely generates a qR complex in lead I, and in our experience never produces a qR complex in V₅ and V₆ in the absence of an MI. It is also possible that in the study of Kochiadakis and colleagues [9], the pacing catheter in some of the controls might never produce a qR complex in V₅ and V₆.

Fig. 1. Twelve-lead ECG showing old anteroseptal myocardial infarction during unipolar DDD pacing in a patient with complete AV block. The ventricular stimulus does not obscure or contribute to the qR pattern in leads I, aVL, and V₆. Leads V₂ to V₄ show Cabrera’s sign and a variant in lead V₅. The lack of an underlying rhythm because of complete AV block excluded the presence of ventricular fusion.

Fig. 2. Twelve-lead ECG showing ventricular fusion related to spontaneous atrioventricular conduction. The pattern simulates myocardial infarction during DDD pacing (atrial sensing-ventricular pacing) in a patient with sick sinus syndrome, relatively normal AV conduction, and no evidence of coronary artery disease. The spontaneous ECG showed a normal QRS pattern. Note the QR complexes in leads II, III, aVF, V₅, and V₆.
have been slightly displaced away from RV apex and produced qR ventricular complexes in leads I and aVL (but not V6) with preservation of superior axis deviation in the frontal plane. On this basis, we cannot accept the authors’ claim of the poor diagnostic accuracy and specificity of Q waves in the diagnosis of MI.

Furthermore, Kochiadakis and colleagues [9] published an ECG example of Cabrera’s sign (their Fig. 1A), but the tracing showed unimpressive slight slurring (with a rapid upward deflection—dv/dt or slope) of the ascending limb of the S wave (see Fig. 6). In our experience, this pattern is commonly seen during uncomplicated RV apical pacing. A true Cabrera’s sign is more prominent, with a markedly different dv/dt beyond the notch, making the sign unmistakable as seen in Figs. 1 and 3. We believe that the ECG in their Fig. 1B [4] showing Chapman’s sign (notching with minimal slurring of the upstroke of the R wave) is also consistent with uncomplicated RV apical pacing (see Fig. 6).

Another group [7] has claimed that Q waves (qR or Qr complexes were not specified) in leads I, aVL, or V6 are not diagnostically useful, but their conclusions are also questionable because of problematic methodology: (1) the number

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**Box 3. Cabrera’s sign**

**Specific Cabrera variants**
- Small, narrow r wave deforming the terminal QRS.
- Series of tiny notches giving a serrated appearance along the ascending S wave.
- Similar series of late notches on QRS during epicardial pacing.

Notches are probably due to a gross derangement of intraventricular conduction.

**False Cabrera’s signs**
- Slight notching of the ascending S wave in V leads is normal during RV apical pacing. It is usually confined to 1 lead, shows a sharp upward direction on the S wave and usually <0.03 seconds; no shelflike or downward notch typical of true Cabrera’s sign.
- Ventricular fusion beats.
- Early retrograde P waves deforming the late part of the QRS complex.

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Fig. 3. Twelve-lead ECG showing Cabrera’s sign during VVI pacing in a patient with an old extensive anterior myocardial infarction. Note the typical notching of the S wave in leads V4 to V6. There is no qR pattern.
Fig. 4. Cabrera Variants. (A, B) There are small and narrow terminal R waves in leads V₂ and V₃, respectively, during ventricular pacing. (C) Series of tiny notches representing gross derangement of intraventricular conduction during ventricular pacing in a patient with an extensive anterior myocardial infarction. (*From* Barold SS, Falkoff MD, Ong LS, et al. Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In: Barold SS, editor. Modern cardiac pacing. Mt Kisco [NY]: Futura; 1985; with permission.)

Fig. 5. Ventricular pacing during acute inferior wall myocardial infarction showing a qR pattern in leads II, III, and aVF associated with ST segment elevation. The R wave in the inferior leads is substantial and, therefore, not due to an overshoot of the QRS complex by marked ST-segment elevation. (*Reproduced from* Barold SS, Ong LS, Banner RL. Diagnosis of inferior wall myocardial infarction during right ventricular right apical pacing. Chest 1976;69:232–5; with permission.)
of “abnormal” patients with Q waves only in the two frontal plane leads and not in V6 was not specified. (2) The protocol called for a LBBB pattern with left axis deviation (more negative than −30 degrees). Normal subjects might have been included in the “abnormal” group because pacing lead somewhat away from the RV apex can cause left-axis deviation with q waves in I and aVL in the absence of MI.

Based on the above arguments, we believe that the findings of Kachiadakis and colleagues [4] and Kindwall and colleagues [5] are questionable and probably not valid.

Acute myocardial infarction

Leads V1 to V3 sometimes show marked ST elevation during ventricular pacing in the absence of myocardial ischemia or infarction [10]. The diagnosis of myocardial ischemia or infarction should therefore be based on the new development of ST elevation. Sgarbossa and colleagues [11,12] recently reported the value of ST segment abnormalities in the diagnosis of acute MI during ventricular pacing and their high specificity. ST elevation ≥5 mm in predominantly negative QRS complexes is the best marker, with a sensitivity of 53% and specificity of 88%, and was the only criterion of statistical significance in their study (Figs. 7 and 8). Other less important ST changes with high specificity include ST depression = or > 1 mm in V1, V2, and V3 (sensitivity 29%, specificity 82%), and ST elevation ≥1 mm in leads with a concordant QRS polarity. ST depression concordant with the QRS complex may occur in leads V3 to V6 during uncomplicated RV pacing [11,12]. Patients who present with discordant ST elevation ≥5 mm have more severe coronary artery disease than other MI patients without such ST elevation [13,14]. Patients with an acute MI, the primary ST changes may persist as the MI becomes old. So-called primary T-wave abnormalities (concordant) are not diagnostically useful during RV pacing if they are not accompanied by primary ST abnormalities (Fig. 9) [11].
Fig. 7. Twelve-lead ECG showing acute inferolateral myocardial infarction during VVI pacing. There is obvious discordant ST-elevation in leads II, III, aVF, and V_{6} that meets the criterion of Sgarbossa and colleagues [11] for the diagnosis of acute infarction. (From Barold SS, Falkoff MD, Ong LS, et al. Normal and abnormal patterns of ventricular depolarization during cardiac pacing. In: Barold SS, editor. Modern cardiac pacing. Mt Kisco [NY]: Futura; 1985; with permission.)

Fig. 8. Twelve-lead ECG showing an acute anterior myocardial infarction during VVI pacing. There is marked ST-elevation in leads V_{1} to V_{5} that meets the criterion of Sgarbossa and colleagues [11] for the diagnosis of acute infarction. The ST-elevation drags the QRS complex upwards. Note the right superior frontal plane axis occasionally seen with right ventricular apical pacing.
Cardiac ischemia

Discordant ST elevation

Marked discordant ST elevation (>5 mm) during ventricular pacing, a recently described sign (with good specificity and moderate sensitivity) for the diagnosis of myocardial infarction [9], could also be used for the diagnosis of severe reversible transmural myocardial ischemia as recently reported in a case of anterior ischemia (Fig. 10) [15]. Two similar cases of ischemia with discordant ST elevation during ventricular pacing have been published [16,17]. Both affected the inferior wall. A report in the French literature [17] involved a temporary pacing lead in the RV in a patient who demonstrated transient but massive ST elevation of unspecified duration in the inferior leads during Prinzmetal’s angina, possibly superimposed on an inferior infarction of undetermined age. During these ischemic episodes, the ECG documented reversible second-degree type I (Wenckebach) atrioventricular block and reversible type I second-degree exit block from the pacemaker stimulus to the myocardium. The latter probably occurred because the tip of the lead was in direct contact with the area of severe transmural ischemia. The other case is less impressive because the patient had a unipolar VVI system (unclear degree of overshoot into the ST segment) and exhibited during chest pain of uncertain duration only about 5 mm of additional discordant ST elevation in a Holter recording with an unspecified lead [16]. Transient massive ST elevation (>10 mm) in paced beats and spontaneous beats in lead III was precipitated during an ergonovine-induced spasm of a dominant right coronary artery in the presence of otherwise normal coronary arteries angiographically [16]. In this patient, the associated ST elevation in spontaneously conducted beats diminished the diagnostic value of the changes during pacing.

Discordant ST abnormalities

ST depression in leads V1 and V2 is rarely normal, and should be considered abnormal and indicative of anterior or inferior MI or ischemia.

Exercise-induced ST changes

Exercise-induced ST abnormalities are in all likelihood nondiagnostic, as in complete LBBB.
The two cases reported by Diaz and colleagues [18] are questionable on the basis of the criteria of Sgarbossa and colleagues [11,12].

**Cardiac memory**

Abnormal depolarization causes altered repolarization. Cardiac memory refers to T-wave abnormalities that manifest on resumption of a normal ventricular activation pattern after a period of abnormal ventricular activation, such as ventricular pacing, transient LBBB, ventricular arrhythmias, or Wolf-Parkinson-White syndrome [19–22]. Pacing-induced T-wave inversion is usually localized to precordial and inferior leads. The direction of the T wave of the memory effect in sinus rhythm is typically in the same direction as the QRS complex. In other words, the T wave tracks the QRS vector of the abnormal impulse. Thus, inhibition of a pacemaker may

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Fig. 10. Diagnosis of myocardial ischemia during ventricular pacing. Three representative panels of three-channel Holter recordings of lead V₁ on top and V₅ at the bottom, together with a special pacemaker channel in the middle displaying the pacemaker stimuli. The top control panel was recorded before chest pain. The second panel shows marked ST-elevation (>5 mm) in V₁ and to a lesser degree in V₅. The bottom panel was recorded about 3.5 minutes after the middle panel. The ST-elevation has partially resolved. (From Barold SS. Diagnosis of myocardial ischemia during ventricular pacing. Pacing Clin Electrophysiol 2000;23:1060–1; with permission.)
Fig. 11. Cardiac memory effect secondary to ventricular pacing recorded in the ECG of a patient with complete heart block from a lesion in the His bundle (confirmed by His bundle recordings). (Top) The tracing is normal except for the rhythm. (Bottom) Chest wall stimulation was performed to inhibit a VVI pacemaker implanted several months previously. There was no clinical evidence of heart disease apart from AV block. Note the striking T-wave inversions in leads II, III, aVF, and V₄ to V₆. (From Barold SS, Falkoff MD, Ong LS, et al. Electrocardiographic diagnosis of myocardial infarction during ventricular pacing. Cardiol Clin 1987;5:403–17; with permission.)
allow the emergence of the spontaneous rhythm with a diagnostic Q wave, but pacing per se may produce prominent repolarization abnormalities that do not represent ischemia, a non-ST elevation, or non-Q wave MI (Fig. 11) [19–22]. It may occur even after 1 minute of RV pacing in humans, with T-wave abnormalities visible after 20 minutes [23]. The marked repolarization abnormalities reach a steady state in a week with RV endocardial pacing at physiologic rates. The repolarization abnormalities related to cardiac memory persist when normal depolarization is restored, and they resolve completely in a month. The changes and their duration are proportional to the amount of delivered ventricular pacing [24]. Cardiac memory is associated with complex biochemical abnormalities. Angiotensin inhibitors and AT-1 receptor blockers attenuate the effects of short-term memory. Calcium blockers reduce the impact of short-term and long term-memory [25]. Long-term cardiac memory involves de novo protein synthesis [26].

Differentiation of cardiac memory from ischemia

Shvilkin and colleagues [27] recently reported that cardiac memory induced by RV pacing results in a distinctive T-vector pattern that allows discrimination from ischemic precordial T-wave inversions regardless of the coronary artery involved. T-wave axis, polarity, and amplitude on a 12-lead ECG during sinus rhythm were compared between cardiac memory and ischemic patients (Fig. 12). The cardiac memory group included 13 patients who were paced in the DDD mode with a short atrioventricular delay for 1 week after elective pacemaker implantation. The ischemic group consisted of 47 patients with precordial T-wave inversion identified among 228 consecutive patients undergoing percutaneous

![Circular histogram of frontal plane T-axes distribution in LAD, LCx, and CM groups. Solid bars indicate LAD; hatched bars, LCx; open bars, CM. Difference in T-vector axis between CM and LAD/LCx is statistically significant (P < 0.01). (From Shvilkin A, Ho KK, Rosen MR, et al. T-vector direction differentiates postpacing from ischemic T-wave inversion in precordial leads. Circulation 2005;111:969–74; with permission.)](image_url)
coronary intervention for an acute coronary syndrome. The combination of (1) positive T wave in aVL, (2) positive or isoelectric T wave in lead I, and (3) maximal precordial T wave inversion > T-wave inversion in lead III was 92% sensitive and 100% specific for cardiac memory, discriminating it from ischemic precordial T-wave inversion regardless of the coronary artery involved.

Summary

Electrocardiographic criteria involving the paced QRS complex are less sensitive but more specific than primary ST abnormalities for MI diagnosis during ventricular pacing. Although one cannot determine with certainty the age of an MI (hours, days, or even years), from a single ECG, the presence of primary ST-segment abnormalities strongly suggests the diagnosis of acute MI or severe ischemia and need for possible emergency revascularization. Patients with a history of chest pain and a nondiagnostic paced ECG should also be considered for emergency cardiac catheterization with a view to performing revascularization. A patient with the ECG shown in Fig. 13 should certainly be a candidate for this strategy.

References


