Chronic Myocardial Infarction
Where Is It Located?

ECG CHALLENGE
This ECG was recorded during a routine checkup of a 77-year-old man who had a heart attack 8 months earlier. He was in the countryside when he felt constrictive chest pain lasting ≈2 hours but was not hospitalized until 20 hours later. A coronary angiogram was performed the following day. Based on ECG demonstrated in Figure 1, where is the culprit lesion? Where was the myocardial infarction (MI) located?

Please turn the page to read the diagnosis.

Figure 1. The 12-lead ECG (see text for description).
RESPONSE TO ECG CHALLENGE

This ECG shows sinus rhythm with left-axis deviation in the frontal plane (left anterior hemiblock) with notches in the S wave, a broad R>S in V1, and Rs with progressively decreased voltage until V6. In frontal plane leads, the voltage was low in all leads, and the T wave was flat in I leads and left precordial leads and negative in VL. No repolarization abnormalities are seen in other leads.

Until recently, these ECG findings had been considered clear examples of a posterior MI because of the presence of a prominent and wide R wave in V1 that was thought to represent the mirror image of the Q wave in the inferior leads.

However, ECG and contrast-enhanced cardiac magnetic resonance correlations have demonstrated the end of this ECG dogma. In fact, a prominent R wave in V1 was shown to be due to lateral, not posterior, MI. This case shows an isolated lateral MI because no Q waves were displayed in the inferior leads. The presence of associated left anterior hemiblock with a slurred S wave in the inferior leads may be explained by a perinfarction block (or focal block).

A lateral MI originates an opposite electric vector that is directed to V1, which explains the presence of a prominent R wave in this lead (Figure 2).

This case supports the 2 criteria we previously published: R/S ≥ 1 and r in V1 ≥ 3 mm with a high specificity (100%) but a lower sensitivity for lateral MI. This indicates that although the presence of this pattern in a patient with a previous MI ensures the lateral location of the MI, many other patients with lateral MI, particularly when involving basal areas, may not present with a prominent R wave in V1. Conversely, if the necrotic area involves the inferobasal segment of the inferior wall (old posterior wall), the vector of an existing necrosis will be directed to V3 to V6 rather than to V1. In fact, because the activation wave arrives to this zone after 30 milliseconds, the necrosis of this zone cannot generate a Q wave (necrosis vector). In our case, the contrast-enhanced cardiac magnetic resonance (Figure 3) clearly shows that the necrotic area was located in the lateral wall and not in the inferobasal segment of the inferior wall.

The correct diagnosis of MI location concerns not only an academic interest but also the size of the MI in cases of lateral location, as in our case, which can be larger than an MI located in the inferobasal segment.

As an example, we present a case of inferior MI that involved the inferior wall including the inferobasal area (called posterior in the past) (Figure 4). The ECG showed pathological Q waves in the inferior leads, but the V1 presented a normal QRS morphology, and the cardiac magnetic resonance depicted the anatomic location of the necrotic area.

DISCLOSURES
None.

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**FOOTNOTES**

*Circulation* is available at http://circ.ahajournals.org.

**REFERENCES**


Chronic Myocardial Infarction: Where Is It Located?
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Circulation. 2017;136:966-968
doi: 10.1161/CIRCULATIONAHA.117.029565
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circ.ahajournals.org/content/136/10/966

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