

## Posterior Myocardial Infarction Is For Real

### To the Editor:

In the July issue of the *Revista Española de Cardiología*,<sup>1</sup> Bayés suggested a change in the terminology used to describe myocardial infarctions. Basing his arguments on a correlation between electrocardiography and magnetic resonance imaging (MRI),<sup>2,3</sup> this author rejects the classification put forward by Perloff<sup>4</sup> of greater curvature of the inferobasal left ventricular (LV) wall and attributes the posterior infarction pattern to what he calls the lateral wall, in keeping with a consensus document.<sup>5</sup>

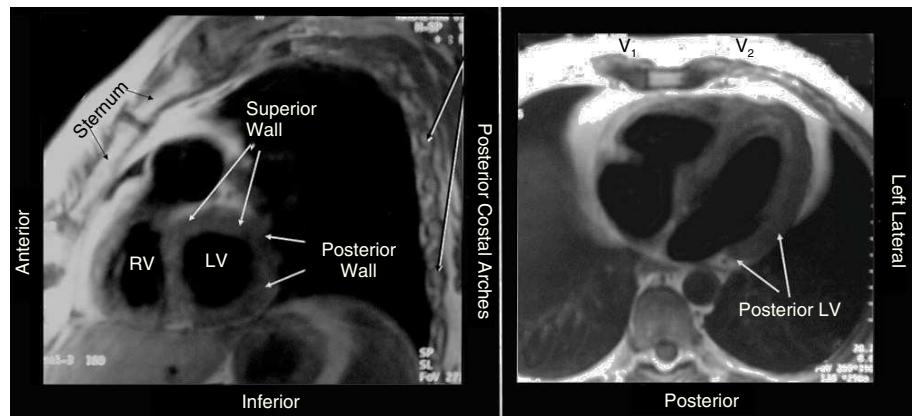
In 1999 a multidisciplinary group<sup>6</sup> called for the use of anatomically correct terminology for Wolff-Parkinson-White (WPW) syndrome and other arrhythmias. Using MRI and fluoroscopy, they showed that the lateral mitral annulus has a posterior position, near the spine, which explains why pre-excitation by accessory pathways in this area shifts the QRS vectors forward, with positive

QRS in  $V_1$  and  $V_2$ . Unfortunately, these observations were not taken into consideration by the working group that proposed a dissociated terminology for the anatomical position of the heart in 2002.<sup>5</sup> The left anterior oblique MRI views (Figure 1), including those of Bayés et al,<sup>1,2</sup> do not perfectly express the anteroposterior or mediolateral position, but do reflect the position with respect to the sternum, demonstrating a posterior position of the “lateral” basal segments next to the spine. Furthermore, computerized navigation reconstruction of the left ventricle allows models of the left ventricle to be created in its true position and shows infarction areas in a posterior position (Figure 2).

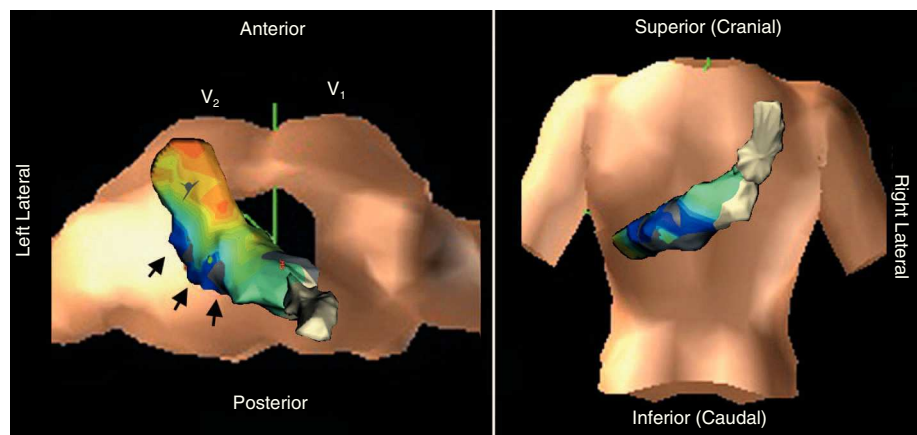
Another anomaly of the proposed nomenclature<sup>1,3,5</sup> is that it fails to recognize a superior wall, which is clearly evident on MRI (Figures 1 and 2). If we were to recognize this location as the site of what is now termed anterior infarctions, we would have a simple explanation of why they are recorded from the top (aVL) and why the ST is paradoxically depressed at the inferior leads.<sup>7</sup>

The use of topographically correct terminology may be of help to understanding the relationships between the anatomy and the diagnostic patterns in the ECG and

**Figure 1.** Left anterior (left) and transverse (right) oblique views of the heart showing the extremely posterior position of the basal portions of the lateral wall. The superior position of the segments known as “anterior” is also shown.



**Figure 2.** Virtual anatomical reconstruction of the left ventricle (Navx®) superimposed on the torso image, showing the actual anatomical position (not the size). The infarct area (gray, blue, and violet) indicated with arrows has a posterior position.



other examinations. In another publication,<sup>8</sup> we have shown how this facilitates an understanding of normal and abnormal ECGs.

In short, the problem with the ECG is that it remains faithful to the anatomy and tells us that the left ventricle has posterior and superior segments. Our effort to ignore this reality is the actual problem.

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## Response

### To the Editor:

First of all, I would like to thank Dr García Cosío for his interest in our work on the ECG-MRI correlation,<sup>1-3</sup> which we summarized in an editorial published in the

*Revista Española de Cardiología* last October.<sup>4</sup> In response to his letter, I would like to make the following comments.

For more than 40 years,<sup>5</sup> the true posterior wall of the heart has been considered the portion corresponding to the inferobasal part of the inferior wall. This area was believed to curve upward. An infarction therein causes a necrosis vector running from back to front, which explains the R-wave in  $V_1$  that was the key electrocardiographic criterion of posterior infarction. Nonetheless, magnetic resonance imaging has shown that<sup>1-3</sup>: *a)* curvature of the inferobasal part of the inferior wall is uncommon (about 20%); *b)* even though a posterior wall might exist, we have proven that infarction of this area does not originate an R-wave in  $V_1$  because the necrosis vector runs toward  $V_3$ - $V_4$ , and not toward  $V_1$ ; *c)* in contrast, with the same correlation, when necrosis occurs in the anterior or posterior parts of the lateral wall (particularly middle and low), the necrosis vector runs toward  $V_1$  and explains the R at this lead; and *d)* following the work done by Durrer,<sup>6</sup> necrosis of the inferobasal part of the inferior wall, classically called the posterior wall, cannot cause Q-wave in the leads on the back or R-wave in  $V_1$  because this area depolarizes after 30 to 40 ms. Therefore, infarction of the so-called posterior wall may naturally exist, but does not cause the R-wave in  $V_1$  that is observed in some lateral infarctions.

Dr García Cosío states that “computerized navigation reconstruction of the left ventricle allows models of the left ventricle to be created in its true position and shows infarction areas in a posterior position.” The area that he marks with the arrows in his Figure 2 as a posterior infarction corresponds to the posterior part of the lateral wall. The true posterior wall, if it exists, would be located to the right of the arrows (segment 4 of the Cerqueira classification<sup>6</sup>). Often, the entire lateral wall is the part affected when there is R-wave at  $V_1$ , and not just the posterior part of the wall. What Dr Cosío points out as the posterior wall in his Figure 1 on the left is actually the lateral wall, which is opposite to the septal wall, and what he indicates as the posterior left ventricle on the right side of the figure is the posterior part of this lateral aspect, but not actually a posterior wall. The posterior wall of the heart from the time of Perloff has been considered the basal-most part of the inferior aspect.

In summary, I would like to express appreciation for the letter because it has allowed me to point out several aspects that were perhaps unclear and are summarized below: *a)* the title may cause confusion if our studies are understood to indicate that there is no posterior myocardial infarction; the fact is that there is no posterior wall—considering as such the part of the inferior wall that curves upward—; if the posterior wall existed and were infarcted, it would not produce high R-wave in  $V_1$  for the reasons we explain in our editorial; and *b)* R-wave in  $V_1$  is due to infarction of the lateral wall, the wall opposite the septal wall which, naturally,

has an anterior portion and another posterior portion, although R-wave in V<sub>1</sub> is often seen when both parts of the lateral wall are necrotic.

Antonio Bayés de Luna

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