New Heart Wall Terminology and New Concepts to Explain the Ecg Patterns of MI with Q Waves Based on Correlations with Magnetic Resonance Imaging

Bayés de Luna, Antoni / Wagner, Galen / Zareba, Wojciech

How the Dogma That the Posterior Wall Existed and That a Posterior Infarct Originated an R Wave in V1 Through V2, the High Lateral Infarction Led to the QS Morphology in VL, and the Anteroseptal Infarct Generated a Q Wave in V1 Through V4Was Generated and Perpetuated

In 1940s, just after the implementation of the precordial leads, it was generally considered that there was an anterior infarct (Q in I and VL, and precordials), and a posterior one (Q in II, III, VF.(1) In the 1950s, some authors (2) indicated that the posterior infarction, which was the one that was in contact with the diaphragm, would be better called inferior or diaphragmatic. When Perloff (3) coined the concept of a strict posterior infarct to explain the RS morphology in V1 through V2, the expression was considered fortunate because it explained what was clear for this electrocardiographic patent, since this necrosis affected the basal part of the inferior wall that was thought to go always upwards and that, for this reason, was denominated the authentic or strict posterior wall. He was then correct to think that the infarct of what was called the strictly posterior wall originated a vector of necrosis that travelled from the back to the front and that was manifested by an RS in V1 through V2, an expression of the Q wave that was recorded in the back (Figure 1 below).

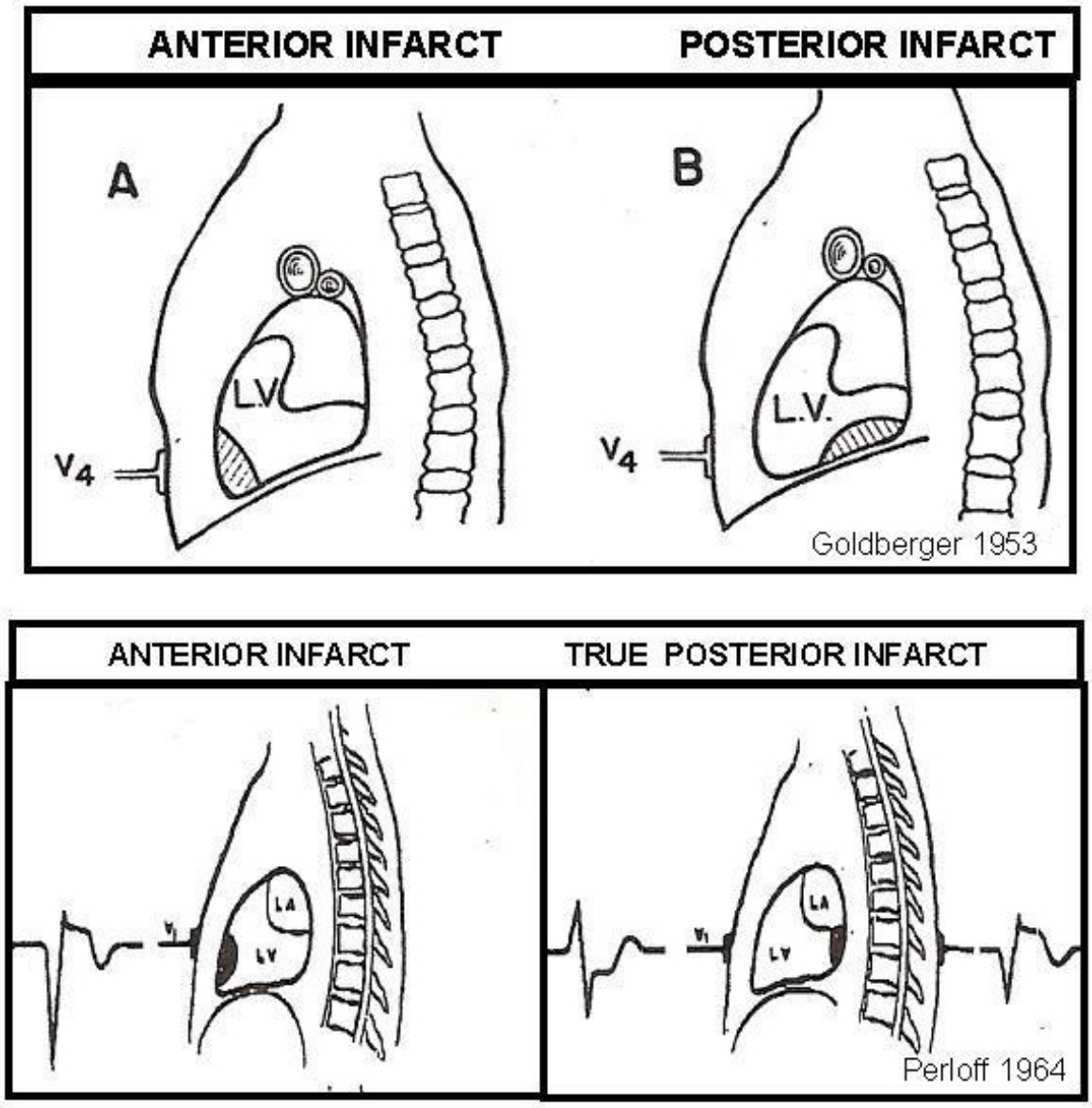


Fig. 1. Above: The concept of anterior and posterior infarction according to Goldberger (1953). Below:The concept of anterior and true or strict posterior infarction is shown according to Perloff (1964). The other part of the wall that lies on the diaphragm to be named inferior .

Naturally, if the infarct only occupied the medial and apical part of the wall that rests on the diaphragm, the authentic inferior wall originated a Q wave in II, III, VF that was considered as an expression of an inferior wall infarction and, therefore, the infarct occupied the inferior wall and the authentically posterior corresponded to an inferoposterior infarct (Q in II, III, VF + RS in V1 through V2).

In spite of the fact that in the past some authors (4) manifested that the anatomopathological correlation that the R on V1 was more easily explained by a lateral infarct than a posterior one, their studies had no echo or were obscured by the prestige of Perloff and the journal in which he published his paper, as well as by the enormous capacity that humans have to transmit wrong information when we believe it to be an article of faith. Even recently there has been evidence, in an age of radionuclide imaging studies (5) and magnetic resonance,(6,7) that the infarct that originates a R wave in V1 is lateral and not posterior. In spite of that, in all of the textbooks, including ours, task forces and guidelines (8 -15) have taken for granted the fact that the posterior infarction was the lesion that generated a tall R wave in V1. It was never questioned whether Perloff could be wrong, probably because their research had never been cast in doubt. All of the electrocardiographists had assimilated and copied in different forms his sagital slicing of the heart (fig. 1 below) in which the localization of the posterior wall infarction originated a necrosis vector that goes from baqck to forward and gave place to the R wave on V1.

In a similar manner, during the 1950s, the idea arose that a high lateral infarction originated changes in I and VL (QR, QS, r with a negative T) without an apparent modification in the precordial leads. The Mexican school vigorously defended this concept of high lateral infarction when the pattern image with a Q (QS – QR) was seen, especially in VL and sometimes in I, and of a low lateral infarction when it was seen especially in V5 through V6. (2,16) The fact that I, VL, and V5 through V6 are high and low lateral leads, respectively, facilitated this correlation. This concept was also perpetuated in most cardiology and ECG books, ours included.

Finally, the name anteroseptal infarction given to cases with a Q wave from V1 to V4 has been questioned for a long time (17,18). Q waves in V1 through V2 only appear when the middle and lower parts of the septum are affected and different imaging techniques, starting by echocardiography (17) and magnetic resonance, (19,20) have demonstrated that in the case of a Q wave further tan V2 the affected zone is clearly apical, with more or less affection of the anterior, septal, and inferior walls according to the length of the anterior descending artery. Because of this, the most appropriate name for us is apical/anterior (21).

Demonstration That Often the Posterior Wall Does Not Exist and That the R Wave in V1 Owes Itself to a Lateral Infarction and the QS Morphology in VL Is Due to a Medial-Anterior Infarction

We have had our doubts about the anatomopathological correlation in electrocardiography for years (22-24) fundamentally for 2 reasons: *a)* because the placing of the precordial electrodes has changed slightly over time, and in the same patient the morphology of the ECG can be changed by moving the precordial leads making the Q wave appear and disappear; the presence of Q in V5

and, in consequence, we can no longer perform the diagnosis of a lateral infarction. We can, therefore, turn a lateral infarction into an anterior one to our liking, and vicecersa; and *b)* because we could not see clearly, when verifying with the anatomy atlas,(25) that the whole basal part of the wall that was evidently resting on the diaphragm, named diaphragmatic or inferior, was directed upwards in a true manner and authentically became posterior.

We know of the reliability of magnetic resonance imaging (MRI) with gadolinium contrast (CE-CMR) to identify and locate the presence of an infarct, (26,27). Therefore we decided, to study the ECG-MRI correlation in cases of Q wave infarction. We immediately proved in a sagital view of the heart that in more than two-thirds of the cases the posterior wall did not exist, because the basal part of the inferior wall was simply a continuation of said wall in the same direction (fig 2).

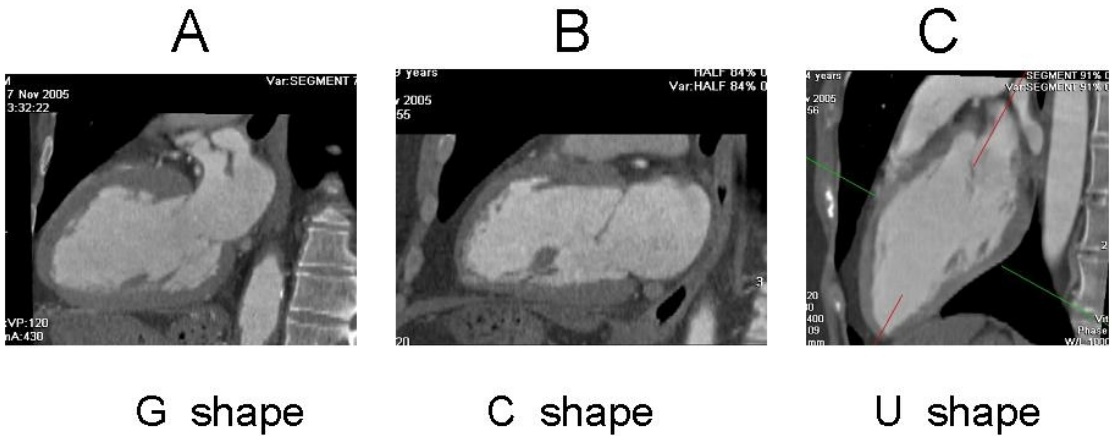


Fig. 2. Sagittal-oblique view in case of normal-body-build subject (A) (G shape). In a man with horizontal heart (B) (C shape) and in a very lean subject (C) (U shape). We have found that the inferior wall does not bend upward in C shape (two-third of the cases), and only in very lean individuals with U shape, the largest part of the wall is posterior (5% of the cases) (C).

That was in agreement with the diagrams observed in most anatomy books (25). We then correlated the cases that theoretically corresponded to an inferior infarction exclusively, because they had an uptake of gadolinium in segment 4 that, according to the Expert Committee of the

American Heart Association (AHA),(28) corresponded to what previously was known as the exclusive posterior wall, and we were able to prove that in V1, contrary to what was expected, there was an rS morphology instead of RS. The images in MRI, in the horizontal plane, gave us the solution because it allowed us to prove that the heart was not situated in an exclusive posteroianterior sense (Figure 3A), following what is said by anatomists that study the heart out of the thorax, the form of Saint Valentine (29) but presented an rS morphology instead of an RS one.

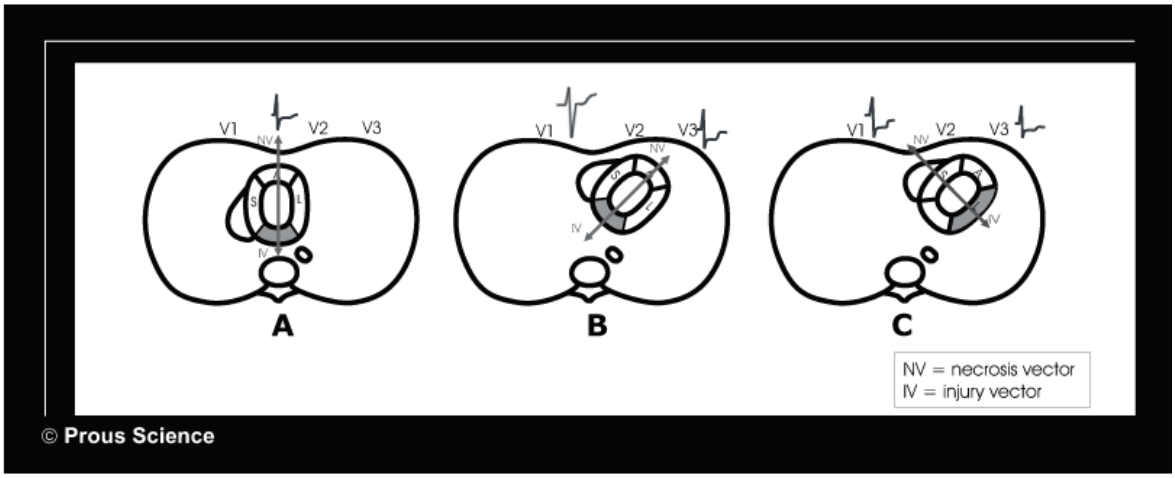
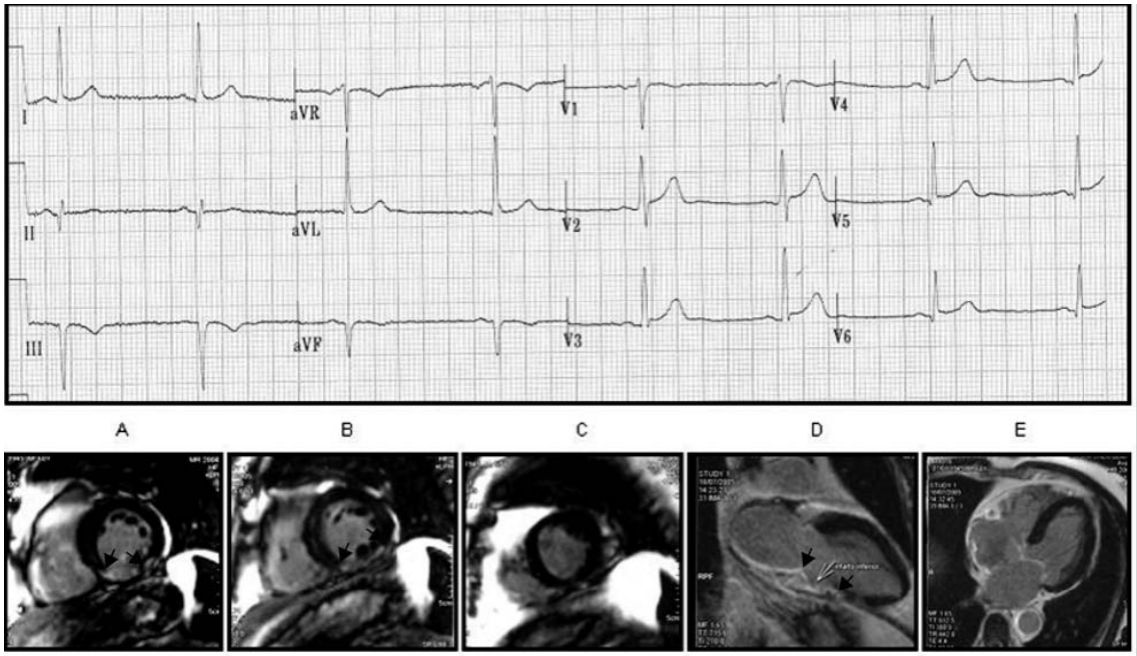


Fig. 3. A: the posterior wall (inferobasal) in its erroneously considered location. With this localization, the infarct vector in the inferior infarction (segments 4 and 10 in thin individuals) is directed towards V1 through V2 and explains the RS patent in said leads. B and C: real anatomical localization of the inferior wall (inferobasal) and lateral infarction. The infarction vector of the inferobasal and medial segments in thin individuals is directed to V3 through V4 and not toward V1, and can contribute to the RS patent that is seen in such leads. On the contrary, the infarct vector of the lateral wall is directed to V1 and explains the RS patent in this derivation.

It was in an oblique posteroanterior and right-left direction (Figures 3B y C). Therefore, in the case of an infarct of the previously named posterior wall, now and according to the AHA consensus, inferobasal segment of the inferior wall, the necrosis vector headed towards V3, showing no increase in the R wave in V1 and being masked in the RS morphology that normally already exists in V3 (Figures 3B and 4).



Fig, 4

Fig. 4. An example of an inferior myocardial infarction with compromising segments 4 and 10 (A and D), and an rS morphology in V1. There is no compromise of the lateral (E) or septal wall.

In contrast, the lateral infarction shows a vector of necrosis that explains the tall R wave in V1 (Figures 34C and 5).

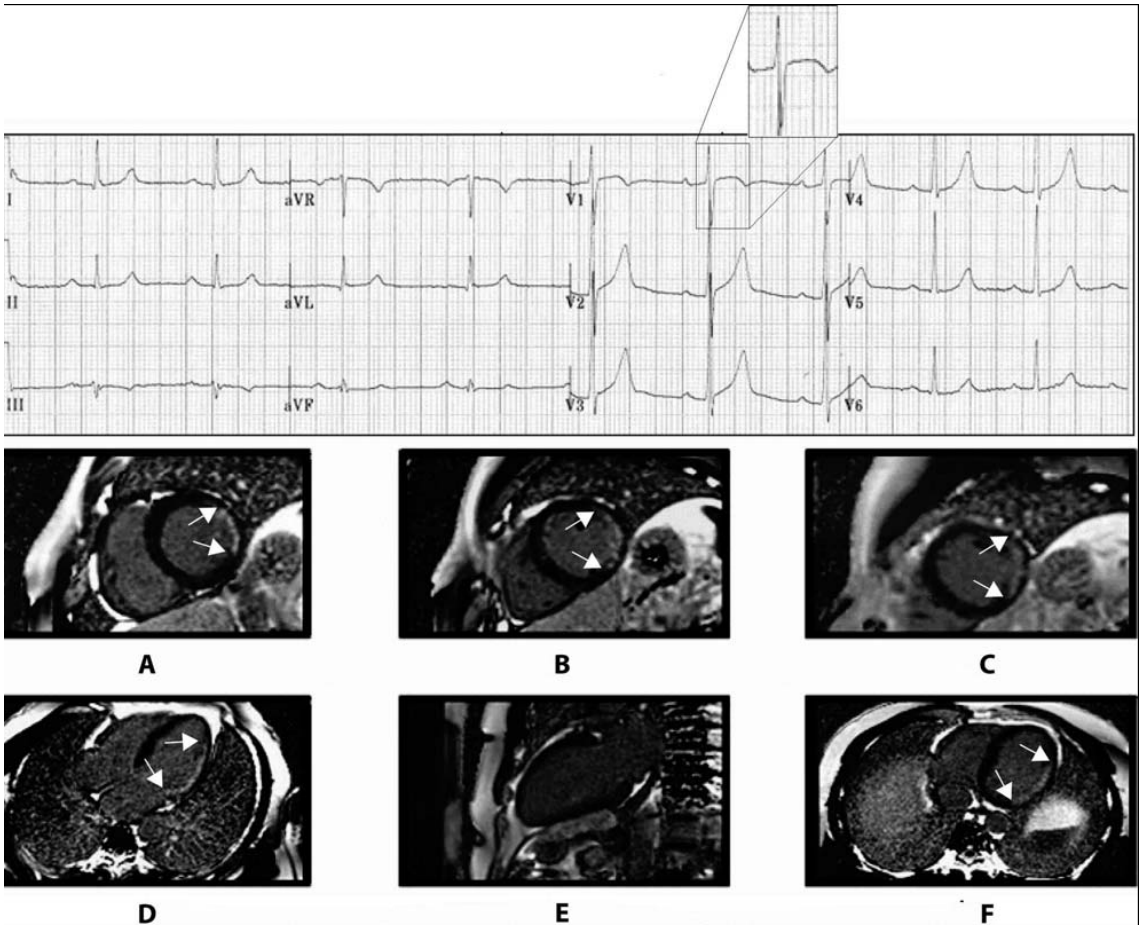


Fig. 5.

Fig. 5. An example of a lateral myocardial infarction with an RS morphology in V1 but without a q in V5 through V6. The magnetic resonance images (A-F) show a compromise of the lateral wall (A-D and F) without a compromise of the inferior wall (E). The sagital section (E) shows an inferior wall that is not compromised. Lateral compromise is evident in all other sections.

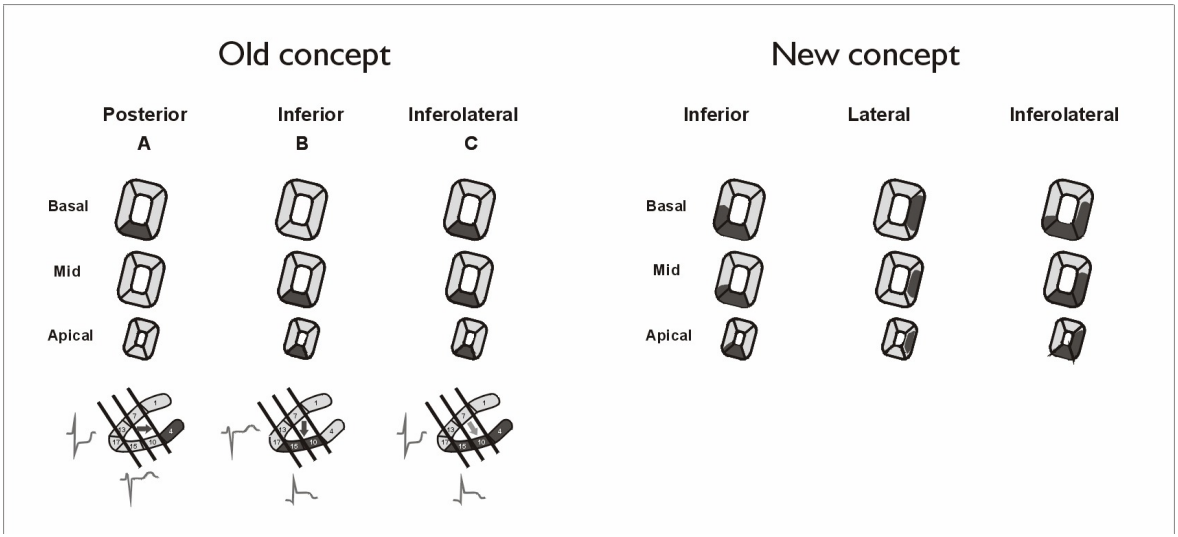


Figure 6 shows the differences between the classic concept and the new concept of types of myocardial infarcton of the inferolateral area.

Fig. 6. The classical concept of the myocardial infarction classification due to the occlusion of the right coronary artery or the circumflex artery (inferolateral zone) is shown here. The basal segment of the wall in contact with the diaphragm (inferior wall) was considered as an upward curve and this portion of the wall was named the posterior wall. Because it was believed that the posterior walinfarction explained the presence of SR in V1 (an equivalent to the Q wave) the myocardial infarction of the inferolateral zone was divided in groups: inferior (Q in II, III. and VF), posterior (RS V1, V2), and lateral (pathologic Q in laterals leads). In infarctions involving 2 or 3 of these areas it receives the corresponding name (inferoposterior, inferolateral, posterolateral). To the left the area involved in the case of the inferior, posterior, and inferoposterior infarction is shown, with ECG patterns in a chronic phase. To the right: with the newly exposed concept, the RS patent in V1 is explained by a lateral myocardial infarction and the infarction of the inferobasal segment of the inferior wall (classically called the posterior wall) does not generate a Q wave because it is a zone of delayed depolarization. In this way, inferolateral zone infarcts are divided in 3 groups: inferior (Q in II, III, and VF), lateral (RS V1 and/or pathologic Q waves in lateral leads), and inferolateral (both patterns).

In addition, it must be recognized that during decades we have made an anatomical as well as electrophysiological mistake. Durrer et al (30) demonstrated that the zones that corresponded to the previously called posterior wall, now inferobasal or segment 4, are depolarized after 30- 40 ms, and therefore cannot originate a Q wave Q (or an R wave as a mirror image in V1 through V2) because the QRS complex has already started to be recorded. In any case, a modification of the second half of the QRS complex will be recorded as a distortion of QRS (31) and/or a reduction in its voltage. We have found that the RS morphology in V1 is very specific for lateral infarct (100%),

though not too sensitive, because it is well known that lateral infarcts often occur with an almost normal ECG or with qr or a small r in I, VL and V5 through V6.

We also were able to prove (19,20) that, in the case of an infarct secondary to the occlusion of the first diagonal artery, a low voltage QS or QR was often recorded in VL with an occasional “qr” in I but without a pathological Q in V6. Therefore this pattern was not due to a high lateral infarct that was the ECG dogma that had been established for decades, but to a medial anterior infarct (occlusion of diagonal) (Figure 7).

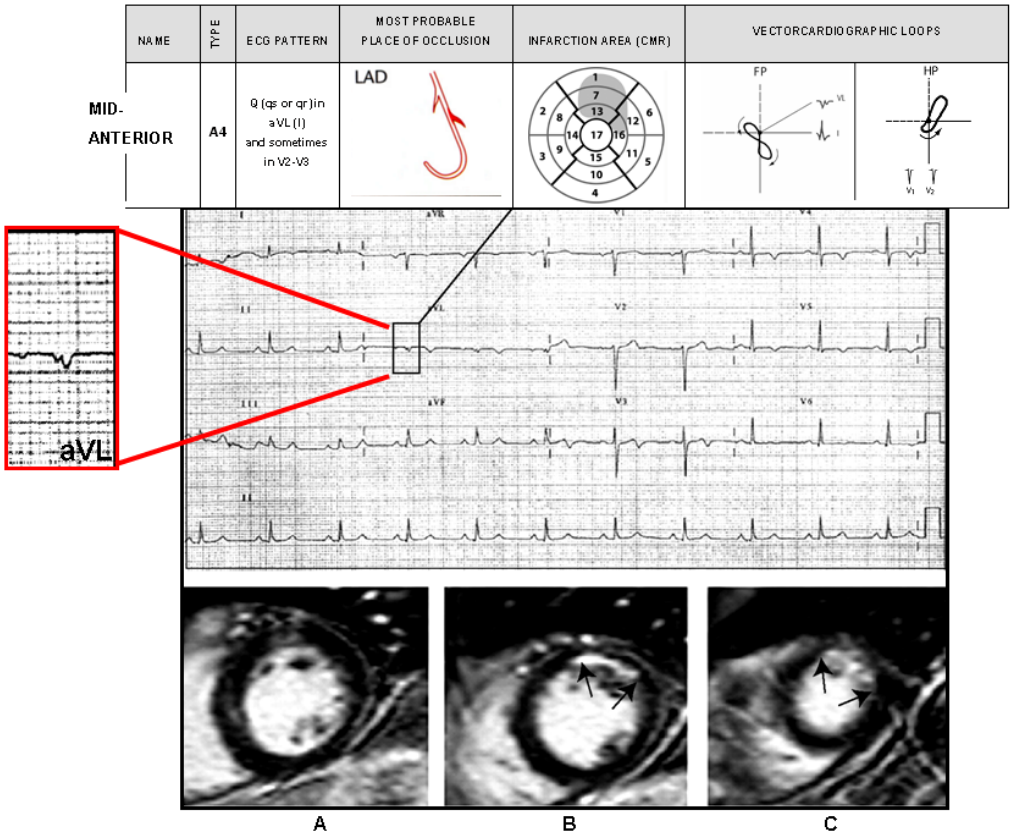


Fig. 7. Example of mid-anterior MI (QS in VL without Q in V5-V6), most probable place of occlusion, CE-CMR area and the VCG loop in this case. CE-CMR images show mid-low-anterior and lateral wall involvement (B-3) but not involvement of basal part (A).

That is explained because the high lateral zone is perfused by the circumflex artery and, therefore, the occlusion of the diagonal cannot produce its necrosis. On the other hand, the basal lateral infarct, as occurs in the infarction of the previously named posterior wall, cannot originate a Q wave due to delayed depolarization of this area.

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