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New ECG classification of Q-wave myocardial infarctions based on correlations with cardiac magnetic resonance

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Based on Myers' anatomopathological studies from 1940s [1–3] it is believed that the determination of the location of the Q wave myocardial infarction (MI) in electrocardiogram (ECG) was the following: Q in V1–V2-septal MI; in V3–V4 anterior MI; in V5–V6 low lateral MI; in I–VL high lateral MI; in II, III, VF inferior MI. In 1960-ties, Perloff [4] introduced the concept of a "strict" posterior infarction to explain RS morphology in V1–V2, this concept was accepted as it was assumed that a clear explanation was found to describe the above mentioned ECG pattern. It was believed that in case of necrosis affecting basal part of an inferior wall, this part was bending upwards and was considered as a "true" or "strict" posterior wall. An infarction of this wall resulted in a necrosis vector directed forward that explained a tall R wave in V1–V2 as a mirror-image of a Q wave recorded in posterior leads. Therefore, it was correct to think that an infarction of above mentioned "strict" posterior wall, produced a necrosis vector directed forward, manifested as RS morphology in V1–V2. In case of an infarct involving only a mid- and apical part of the wall leaning on the diaphragm, the true inferior wall, a Q wave in II, III, and VF was identified. The infarction that involved inferior and posterior wall corresponded to an inferoposterior infarction on ECG (Q in II, III, VF + RS in V1–V2).

As early as in 1956, Dunn et al. [5] documented by anatomopathological correlation that the R wave in V1 should be explained more by a lateral than a posterior infarction. Modern imaging techniques as isotopes [6] and magnetic resonance [7] are the source of data documenting that an infarct producing a tall R wave in V1 is a lateral and not posterior one. Nevertheless, in all textbooks (including ours), task forces and guidelines it was described that posterior infarction is manifested by a tall and broad R wave in V1. ECG experts did not question Perloff's theory with his saggital section of the heart showing that the location of an infarct in a posterior wall produces a necrosis vector directed forward resulting in the R wave in V1.

Regarding lateral MI, the Mexican School defined an ECG pattern of pathologic Q seen in VL and sometimes in I as a high lateral infarct while the presence of this pattern in V5–V6 is defined as a low lateral infarct [8–10].

For years there were scientific doubts regarding the validity of the established electrocardiographic — anatomic correlation. The reasons for these doubts might include potential influence of lead placement, which could alter the ECG morphology and contribute to false positive or false negative R waves or Q waves. Secondly, there was not clear convincing anatomical evidence that the basal part of the inferior (or so called diaphragmatic) wall is surely directed upwards and thus, becoming truly posterior.

Contrast enhanced cardiac magnetic resonance (CE-CMR) is a modern and reliable method for identification and localization of myocardial necrosis. We studied the ECG-CMR correlation in a series of patients with Q-wave myocardial infarction [11, 12]. It was found that in over two thirds of the cases, the posterior wall of myocardium could not be identified, and that the basal part of the inferior wall was lying on the diaphragm as a simple continuation of the rest of the wall. We evaluated cases in which exclusive or predominant posterior MI was present according to gadolinium enhancement in Segments 4

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and 10 of the AHA Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging [13]. We observed that in these cases, on the contrary of the expected, the QRS morphology in V1 was rS instead of RS. The CE-CMR images in horizontal axial sections may explain these discrepancies because they confirmed that the heart is oriented obliquely in postero-anterior and right-to-left direction, and not located strictly in postero-anterior direction [14] as presented by the pathologists who study the excised heart. Thus, in the case of inferobasal segment infarction, previously known as posterior wall MI, the necrosis vector is directed towards V3 and does not produce change in R in V1 being masked by RS morphology normally seen in V3.

Furthermore, it should be recognized that for years not only anatomical but also electrophysiological error has existed. Durrer et al. [15] demonstrated that the myocardial zones which correspond to the posterior wall (now named Segment 4 or inferobasal), depolarize after 40 ms when the recording of normal QRS complex has been initiated and thus, cannot originate Q wave (or R wave as a mirror-image in V1–V2); although, in such case, a distortion of the second part of QRS complex and/or voltage reduction could be recorded [16].

Therefore, with no doubts, necrosis of the previously named posterior wall (Segment 4) could not produce R wave in V1 for the following reasons:

— It is a zone of delayed depolarization that cannot result in Q waves.
— The posterior wall in general does not exist as in over two thirds of the cases it follows a strict alignment with respect to the other segments of the inferior wall. This is why the necrosis vector generated in this case is generally directed upwards and will originate Q waves in II, III and VF leads but will not result in a tall R wave in V1 lead.
— Even cases with extremely vertical heart position, where a great part of the inferior wall is authentically posterior, maintain their oblique position in the thorax. Therefore, not only inferobasal part but also medium part of inferior wall will be affected by an infarct. In these cases if a necrosis vector capable of producing Q wave is generated, this vector will be directed towards V3 and not V1. Therefore it may not explain the appearance of the R wave in V1.

On the contrary, in case of a lateral infarction affecting also basal part, particularly Segments 5 and 11 of the Cerqueira classification [13], the necrosis vectors will be directed towards V1 and will explain the presence of RS complex in this lead. We observed [11, 12] that RS morphology in V1 is very specific (100%) but not sensitive for the lateral infarction. It is well known that lateral infarcts with almost normal ECG or with qr or a small r in I, VL and V5–V6 leads exist. We could also demonstrate [11, 12] that infarctions caused by occlusion of the first diagonal artery sometimes resulted in low voltage QS morphology in VL occasionally with "qr" complexes in I lead but with no pathological Q wave in V6. This pattern did not correspond to high lateral infarction as was proposed as ECG dogma established for years, but was a result of a mid-anterior infarction. It is explained by a fact that the high lateral zone is supplied by LCx artery. Therefore, occlusion of a diagonal branch cannot result in a necrosis of this zone. On the other hand high basal lateral infarction, similarly as it happens with "old" posterior wall, presents delayed depolarization and does not produce pathological Q wave.

New classification of Q wave infarcts presented in Figure 1 was developed based on high concordance (88%) between ECG findings and CE-CMR imaging and consensus of experts in the field [17].

NAME	ECG PATTERN	INFARCTION AREA (CMR)
SEPTAL	Q in V1–V2	
MID-ANTERIOR	Q (qs or qr) in aVL and sometimes in I and/or V2–V3	
APICAL-ANTERIOR	Q in V1–V2 to V3–V6	
EXTENSIVE ANTERIOR	Q in V1–V2 to V4–V6, aVL and sometimes I	
LATERAL	RS in V1–V2 and/or Q wave in leads I, aVL, V6 and/or diminished R wave in V6	
INFERIOR	Q in II, III, aVF	

Figure 1. The ECG patterns of Q-wave myocardial infarction (MI) or Q-wave equivalents with the names given to MI and related infarction area documented by cardiac magnetic resonance (CMR). Reprinted with permission from reference [17].

We identified 4 ECG patterns of Q wave infarcts well correlated with 4 necrosed zones in the anteroseptal area:

— Septal infarct (subocclusion of the left descending anterior artery affecting septal branches), Q in V1–V2.
— Apico-anterior infarct (distal occlusion of LAD), Q in precordial leads in V1–V2 all the way to V3–V6.
— Extensive anterior infarct (proximal LAD occlusion) Q in precordial leads V1–V2 to V4–V6, aVL, and sometimes in lead I.
— Mid-anterior infarct (1st diagonal occlusion) QS in VL and sometimes Q in I, without pathological Q in V5–V6. Sometimes small "q" in V2–V3.

In the inferobasal zone we found 3 ECG patterns of Q wave infarcts:

— Inferior (RCA occlusion, sometimes distal LCx occlusion) Q in II, III, VF.
— Lateral (occlusion of LCx or its branches OM), RS in V1–V2 and/or q (qr, r) in I, VL, V5–V6.
— Infero lateral (occlusion of RCA or dominant LCx), with ECG signs of inferior and lateral infarct.

There is no doubt that medicine needs to benefit from new modalities (in this case cardiac magnetic resonance imaging) to verify some so called established concepts. The change in terminology of the location of myocardial infarction in ECG will require some time to be accepted in clinical medicine. The clinical significance of the changed terminology is underinvestigated and requires studies utilizing ECG, CMR, and angiographic findings as well as outcome studies relating new terminology to risk of cardiac events in postinfarction patients.

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