Electrocardiography and Posterior Wall Infarction: Has the Enigma Been Solved?

To the Editor:

With great interest, we have read the article by Bayes de Luna[1] about the new electrocardiographic nomenclature for Q-wave myocardial infarctions. The article is very interesting in as much as it provides an electrocardiographic correlation with the magnetic resonance image, verified with gadolinium contrast. The

eternal dilemma about the nomenclature of different cardiologic segments based on surface electrocardiograms is not exclusive to the world of ischemic cardiopathies. Cosío et al[2] proposed new terminology to designate the localization of the accessory pathways as a function of their spatial anatomic location using new technologies, like magnetic resonance.

This brings us back to the complexity of the electrocardiogram—a basic diagnostic tool of incalculable value—which are simple to obtain but still present numerous enigmas in their interpretation. As Bayés de Luna says,[1] in the light of experience, "don't believe in immovable, lifelong dogmas." So, interpreting electrocardiograms is subject to innumerable factors of spatial resolution: cardiac obliquity, verticalization, or horizontalization of the central axis, thoracic conformation, precise electrode positioning, etc. Occasionally, even in patients with apparently normal ECGs, we discover a Brugada type I pattern by positioning the electrodes in the second intercostal space.[3]

In Figure 1, we present an ECG that, analyzed in detail, is very similar to the one presented at the end of Bayés de Luna's article: predominant qR in leads on the limbs, and V_5, V_6, together with a marked RS morphology in V_1 (90 ms in duration) with ST and negative T elevation in the same lead (as in the ECG presented by Bayés de Luna). A noteworthy difference (apart from the fact that our ECG presents a sinus arrhythmia), is that the ECG presented by Bayés de Luna portrays an rSr' pattern and ST rectification in III, which in principle would not be justified by ischemia given that, as the author explains, the AMI in the ECG under study is "lateral, without compromising the inferior wall" (as is borne out by the sagittal magnetic resonance image). Our ECG corresponds to a man aged 24 years, asymptomatic and without cardiovascular risk factors, referred to our cardiology clinic from primary care for respiratory sinus arrhythmia. Despite the curious V_1 morphology, the patient was slightly built, 1.79 m tall, 80 kg in weight, and presented no pectus excavatum- or pectus carinatum-type chest deformities. Electrodes were correctly positioned, the chest x-ray showed no obvious cardiac apex deviation, and echocardiograms were normal (including wall thickening and segment contractility). Logically, our patient was not suspected
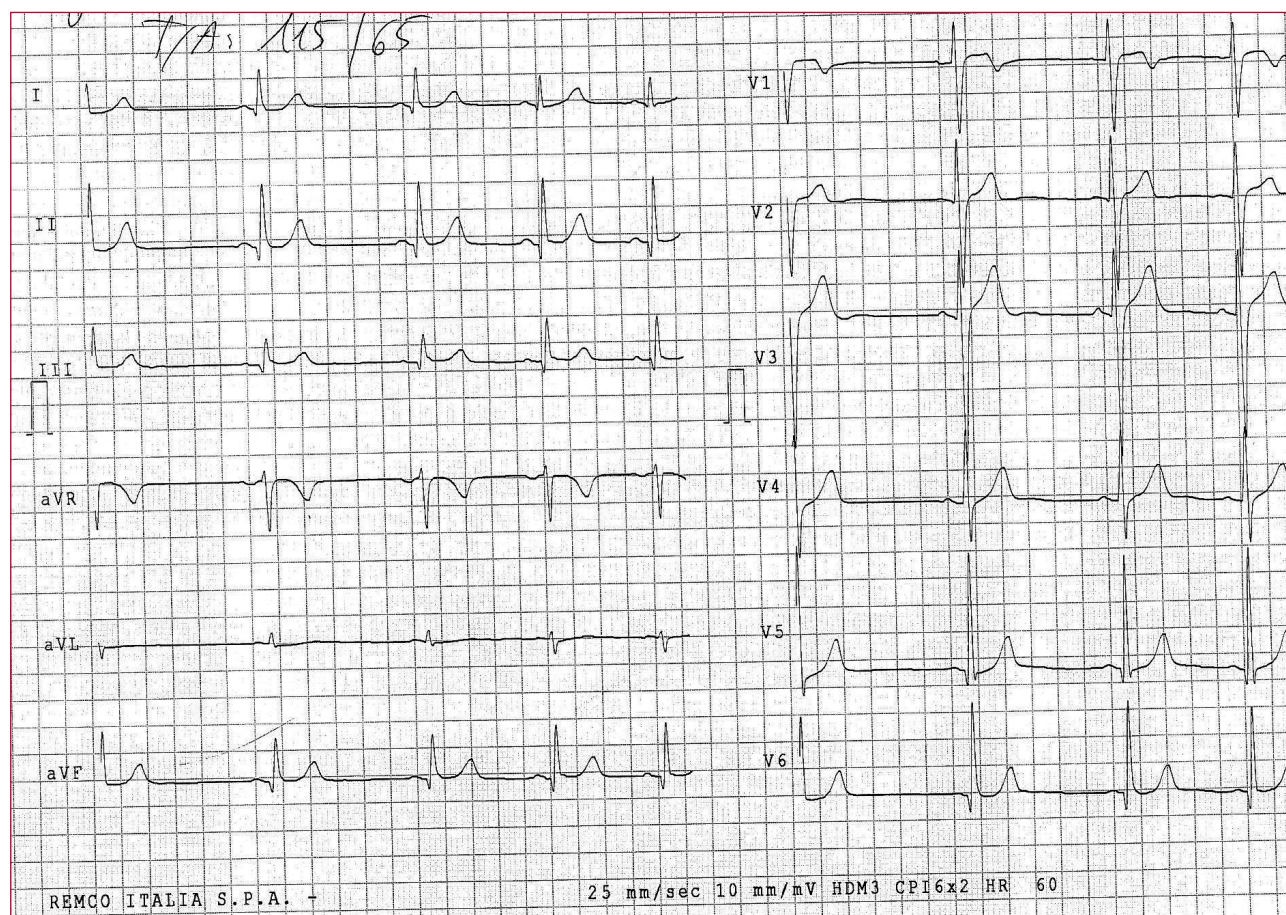

Figure 1

of having transmural "posterior" (or lateral, in the new terminology) AMI.

While we agree with the proposed new terminology for Q-wave AMI,[4] as it seems a better reflection of the reality, we would point out that, although surface ECG continues to be the fundamental diagnostic tool, it stills holds undiscovered secrets.

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Response

To the Editor:

May I express my thanks to Dr Alarcón-Duque and his co-authors.

I would like to make it clear that the electrocardiographic criteria for Q wave myocardial infarction refer to patients with STEACS who, in the chronic phase, present tall Q or R waves in V_1. Thanks to the correlation with magnetic resonance, our study shows that in these patients the presence of Q waves of necrosis or equivalent images (R in V_1 with R/S >1, and/or R ≥40 ms in duration) enables us to locate the infarction. In Alarcón-Duque et al's example, R duration is <4 ms and the R/S relation is around 0.5. I seem to recall that an R/S relation ≥1 is the value which has 100% specificity for lateral infarction.

What we want to illustrate is that in a patient with myocardial infarction following STEACS, a tall R in V_1—above all if the R/S relation is ≥1 and R duration ≥40 ms—necrosis must be lateral and not posterior. It cannot be posterior because: *a)* the posterior wall (now segment 4 of the Cerqueira classification: inferobasal) often does not exist because segment 4 does not curve upwards; *b)* even if it existed, necrosis would not give a tall R in V_1, the mirror-image of the Q-wave of the leads on the back, because it is depolarized after 30-40 ms when the normal R wave has begun to register, meaning there cannot be Q on the back; and *c)* because, in any case, even if the necrosis vector existed, it would be directed towards V_3-V_4 not V_1-V_2 due to the oblique position of the heart in the chest.

Naturally, with a tall R wave in V_1, we need to rule out Wolf-Parkinson-White syndrome, right bundle branch block, and right ventricular growth. In their absence, never in normal individuals, duration of R is ≥40 ms and the R/S relation is >1. The ECG presented by Alarcón-Duque et al presents neither R ≥40 ms nor R/S ≥1.

I thank Alarcón-Duque et al for stating the need always to correlate the ECG with the symptoms. Furthermore, I would like to add that more information on these data can be found in some of our other studies,[1-4] in addition to those they quote.

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