Journal of the American College of Cardiology
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Published by Elsevier Science Inc.

Vol. 35, No. 1, 1999
ISSN 0735-1097/99/$20.00

LETTERS TO THE EDITOR

Electrocardiogram in Myocardial Infarction: What Is Most Relevant?

Phibbs et al. (1) have recently published an interesting review article on studies comparing Q wave with non–Q wave myocardial infarction (MI). This classification of Q wave/non–Q wave gained widespread use in the prereperfusion era because the rather passive role of clinicians during the acute phase of infarction entailed awaiting Q wave development (or lack thereof) for outcome prediction in survivors. As Phibbs et al. indicated, the dichotomy of Q wave/non–Q wave is inaccurate. The "non Q" category has encompassed infarctions that have produced R-wave changes (i.e., posterior MI, decrease in R-wave amplitude) and are indeed Q wave equivalents. In addition, I believe that the main limitation of the Q/non Q dichotomy is that it erroneously polarized prognostic groups. Several authors have alerted that within the non–Q wave classification there were lumped together infarctions of the T type (which manifest in the electrocardiogram [ECG] only with T wave inversion) and of the ST type (which mainly manifest as ST segment depression) (2,3). The latter type often included patients with a previous infarction, and the underlying anatomy was usually left main occlusion or extensive coronary disease with patchy necrosis. A review of prethrombolytic studies would indicate that, from a prognostic viewpoint, most Q wave infarctions were between the T and the ST types of non–Q wave MI (4). Thus, comparisons of Q versus non–Q wave outcomes have been fraught with the problem that patients and control subjects were often included in the same study arm.

The value of the "T versus ST" classification deserves further evaluation in patients undergoing reperfusion. In a recent study we analyzed over 1,500 patients admitted to the hospital with ST segment elevation. Patients with a history of MI and Q wave equivalences were also included. In this "retrolective" analysis, the favorable prognostic significance of T wave inversion after thrombolysis was confirmed (5). When negative T waves were tested separately from non–Q waves, both variables were associated with similar 30-day survival rates. In a combined four-category plot, patients with negative T waves, but absence of Q waves (i.e., T type of non–Q wave MIs), were the most likely to survive at 30 days; patients in the opposite extreme (i.e., those without negative T waves and with Q wave MIs) were the least likely to survive. Other investigators have suggested that one possible reason for this outcome is a high prevalence of patent culprit coronary arteries (6). We also found that negative T waves were independent, powerful predictors of a nearly four times higher survival rate after adjusting for clinical variables and for new Q waves.

ST segment depression, by contrast, is known to predict cardiac events and death (7), and no benefit from thrombolysis has been shown in this group (8).

Whether or not the categorization "T type/ST type" is prospectively confirmed, the terms "Q wave" and "non–Q wave" should be redimensioned and used as one more ECG element to assist in prognostic stratification, rather than as polar categories.

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PII S0735-1097(99)00511-2

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REPLY

We appreciate Dr. Sgarbossa's kind comments about our review entitled "Q wave vs. non–Q wave myocardial infarction: a meaningless distinction." She points out, appropriately, the need to include "Q wave equivalent" deflections in any comparative study, but we would also like to reemphasize the overriding importance of comparing *first myocardial infarctions (MIs) only* in this type of study, because subsequent MIs have a much higher morbidity and mortality and usually do not generate Q waves. The main thrust of our review was that there is no basis for the notion that the non–Q wave MI is somehow "unstable," with an increased risk of post-MI acute events, and with this we are sure Dr. Sgarbossa agrees. In fact, we quoted a study from Sgarbossa's group (1) supporting this point of view in our review.

She quotes her own study of T wave polarity after MI, in combination with the presence or absence of Q waves, as a prognostic index in both the Q wave and non–Q wave categories. Because this report has appeared only in abstract so far, it is impossible to comment on the details of the protocol.

Were only first MIs included in the study? An outcome study based on two variables can be very tricky, as any statistician will attest, but the results may well be significant.

Dr. Sgarbossa comments on several other studies addressing the value of ST segment depression and T wave inversion as prognostic indexes. This element was not included in our review, because we were concerned only with the presence or absence of depolarization abnormality as a clinical marker. The studies cited by Sgarbossa, suggesting that the type of S-T-T deformity may contribute important prognostic information, are all based on

assessment of only non–Q-wave MIs, which dates from a period when the non–Q wave MI was assumed to be a valid subset with unique characteristics. It is to be hoped that this misconception has been permanently put to rest and, further, that investigations of outcome after MI will avoid the egregious error of combining random mixtures of first and subsequent infarcts.

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PII S0735-1097(99)00512-4

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Does Flow Reserve Match Contractility?

I have read with great interest the report by Barillà et al. (1). The data reported are intriguing because, to the best of my knowledge, this is the first report indicating that the restoration of regional contractility during low dose dobutamine administration may occur despite different perfusion patterns, depending on the presence or absence of collateral filling.

Let me raise an issue not addressed in the Discussion of Barillà's article. I definitely agree with Bonow (2) that the increase in flow in patients with collateral filling is expected, because the drop of pressure beyond the fixed obstruction can increase the flow, despite the coronary driving pressure's being unchanged. The no-measurable-flow response in patients without collateral channels can also be expected. In fact, why should the flow increase through a stenosis or an occlusion? Irrespective of flow regimen, the authors (1) noted an amelioration in contractility of dysfunctional myocardium—one that was still present at 2-methoxy-isobutyl-isonitrile (MIBI) administration and during the time allowed for it to distribute to the myocardium (i.e., up to 8 min), I presume, because no mention was ever made to subsequent deterioration of wall motion. This is an astonishingly long time, which would more appropriately define the response to low dose dobutamine of stunned myocardium (but this was not the case, as indicated by the low sestamibi uptake). It seems inconceivable that such a prolonged increase in contractility may occur in the absence of an adequate increase in blood flow, the situation being absolutely different from the postextrasystolic potentiation of contractility, when myocytes burn their energy stores all in one go. By contrast, the increase in contractility of ischemic but viable myocardium at low dose dobutamine is a short-lived phenomenon: it may begin at very low dosage (as low as 2.5 μg/kg body weight per min, in our experience) and usually fades away at 10 μg/kg per min, seldom at 20 μg/kg per min. In patients with very severe coronary stenosis or coronary occlusion without collateral blood filling, a biphasic response to dobutamine should be expected at a dosage even lower than that at which the authors injected technetium-99m sestamibi. Given this, as well as the notion of the ischemic cascade (3,4), I make the point that Barillà et al. (1) described an intermediate phase of the biphasic response phenomenon—that is, the time when the flow reserve is exhausted, but wall contractility has not yet deteriorated in response to forthcoming or ongoing ischemia, or both. I suggest that this possibility is whispered to the reader.

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PII S0735-1097(99)00519-7

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REPLY

We are grateful to Dr. Barletta for his comments. Obviously, the findings we have reported constitute an intermediate step of a biphasic response phenomenon, as stated by Dr. Barletta. However, a 5-min step protocol for low dose dobutamine echocardiography is common (1,2), and a biphasic response (i.e., wall motion improvement followed by worsening) is rarely observed at low doses of 5 to 10 μg/kg body weight per min (3). Nevertheless, no change in wall motion and thickening occurred during the 3 min after tracer injection, even when we used 10 μg/kg per min of dobutamine.

We also wish to emphasize that our study was not intended to describe the behavior of inotropic contractile reserve during low dose dobutamine infusion, but it was aimed at investigating the pathophysiologic and clinical implications of the presumed mismatch between perfusion and contractility in areas with severely hypoperfused viable myocardium.

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PII S0735-1097(99)00520-3

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