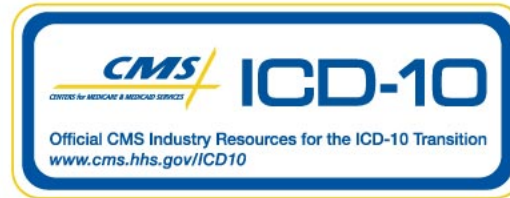


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Q-Wave Versus Non-Q Wave Myocardial Infarction: A Meaningless Distinction

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The concept of the NQMI as a discrete clinicopathologic entity has gained widespread acceptance. It is commonly believed that the NQMI is somehow less than “complete,” an unstable phenomenon with an increased potential for further acute ischemic insult in the postinfarct period. In 1988 the joint ACC-AHA Committee on Myocardial Infarction grouped NQMI together with unstable angina in terms of management and prognosis and this association seems to be widely accepted (1). In fact, unstable angina and non-Q wave MI are almost universally combined as an entity or continuum in current literature (2,3).

The pragmatic consequences of this concept have been far-reaching: because of the supposed hazard of further ischemic insult in the immediate postinfarct period, NQMI has commonly been thought to constitute an indication for invasive study and possible revascularization. The 1988 American Heart Association/American College of Cardiology Joint Task Force Report cited above (1) defined NQMI *with or without symptoms* as a “Class I” or “definite” indication for diagnostic coronary angiography. While the 1996 ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction (4) cautioned that invasive study following NQMI was not justified by evidence (category IIb), there is little question that the earlier recommendation is applied in the majority of cases today (5,6).

The character of the NQMI therefore emerges as a major issue in both theoretical and practical terms justifying a critical review of the data in the field. The results of such a review are presented here; they may be briefly summarized as follows:

1. Every appropriately designed study comparing the postinfarct history of QMI and NQMI has demonstrated that the post-MI course of the two categories is essentially identical in terms of untoward events and complications. None of these studies has validated the concept of the NQMI as an “unstable” entity. Nine such

studies have been reported and the results are summarized below.

2. Every study alleging the “unstable” character of the NQMI has suffered from one or more major flaws in protocol that essentially negate the findings.
3. Three prospective studies investigating conservative versus invasive management of NQMI have shown no benefit from invasive management. One retrospective study reached the same conclusion.

These statements are sufficiently at variance with common perception to warrant detailed exposition; it is as well to begin by tracing the origins of today's widespread beliefs.

The pathologic Q wave and myocardial infarction: persistence of an experimental error. Until 1954 the Q wave was recognized simply as a marker of myocardial infarction; it was not thought to designate any particular subset. In that year Prinzmetal et al. (7), using needle electrodes, thought they demonstrated that the subendocardium was electrically “silent” and did not enter into the genesis of depolarization. They concluded that only transmural infarcts could deform the forces of depolarization, thus producing a pathologic Q. No other valid pathologic or experimental evidence has ever been produced to support this concept.

It is truly astonishing to find that these same workers repudiated their earlier results in 1957 (8) admitting errors of method and concluding that there was no reason to suppose that subendocardial infarcts could not generate pathologic Q waves.

Although the second study was published in the *American Heart Journal*, the medical profession accepted the findings of the original flawed study and ignored or were unaware of the retraction. The Q wave-transmural infarct association appears in every cardiologic text of the period; the notion persisted for a generation (9–12).

It is even more remarkable that every cardiac pathologist who studied the problem in this thirty year period found no association between the transmural or subendocardial character of infarction and the presence or absence of Q waves (13–18). Consistently, they reported that about half of all subendocardial infarcts are accompanied by Q waves while half of transmural infarcts are not.

This surprising divergence of pathologic fact and clinical concept persisted until three articles published between

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1980-83 (19-21) reviewed the pathologic data and pointed out that the Q wave-transmural infarct association did not exist, a fact now universally accepted and recorded in all standard texts.

The clinical character of the NQMI: current concepts.

Despite the admitted absence of any pathological distinction, the concept that NQMI is in some essential quality different from QMI has persisted. It is now alleged that early mortality is lower with NQMI whereas one-year or later mortality of the two types is identical. It is further alleged that the reason for the "catchup" in mortality is the "unstable" state of the NQMI, predictive of increased risk of acute events in the year following infarction (22-23). Before reviewing the studies affirming or denying this concept, it is essential to define an acceptable protocol of study.

Q wave vs non-Q wave MI: an acceptable protocol for comparative study. To assess the Q wave as an independent marker of mortality or morbidity, any study should correct for the following variables.

1. First infarct versus subsequent infarcts. Subsequent infarcts pose a much higher risk than first infarcts: early and late mortality after reinfarction have been found to increase by factors of 1.9 to 3.0 respectively in two studies (24,25) and "prior myocardial infarction" emerges as a major risk factor in all studies of morbidity and mortality of myocardial infarction (26,27). To compare morbidity and mortality in groups containing varying numbers of first and subsequent infarcts in terms of mortality and morbidity is therefore an exercise in confusion. This confusion is compounded by the fact that new Q waves are relatively uncommon in subsequent infarcts; they appear about half as often as in initial infarcts (28). Thus many subsequent infarcts with higher risk will be grouped with the non-Q group with additionally confounding results.
2. Stratification by age. Age is one of the most important determinants of morbidity and mortality; stratification by decades would be reasonable.
3. Q wave versus depolarization abnormality. The process of infarction alters depolarization by a number of mechanisms, e.g., dispersion, slow conduction and localized block. These forces change the surface QRS in a number of ways: to suppose that they can only cause negative deflections, or Q waves, is electrocardiographically naive. It is equally naive to confine attention to the initial 40 msec forces, since at least 8 to 10% of all infarcts involve the base of the left ventricle, a region depolarized during the middle or terminal vectors of the QRS. A tall R in the right precordium and localized R wave diminution are two obvious and accepted "Q wave equivalents" but there are others. QRS alterations correlated with infarction in a number of studies include R/S changes (29), acute frontal-plane right axis deviation (30), new left axis deviation, low voltage and QRS notching (31), precor-

dial QRS notching (32), initial and terminal QRS notching (33), high-frequency notching in orthogonal leads (34) and abnormally narrow precordial R waves (35). A rational approach therefore, should include all acute deformities of depolarization documented to correlate with infarction, not simply those derangements of initial forces that happen to produce a negative deflection in the surface ECG. Summing up the findings of the studies listed above in quantitative terms, it is conservative to estimate that approximately half of all infarcts without Q waves will manifest Q wave-equivalent distortions of depolarization. If Q waves alone were used as markers, about half of the cases listed as "S-T-T only" would be incorrectly classified. The only scientifically valid basis for comparison would be "depolarization abnormality" versus "repolarization abnormality only."

4. Location of infarct. Most investigators have reported a significantly higher mortality and morbidity with anterior as opposed to inferior infarcts: any valid comparison should be corrected for infarct location.
5. Enzymatic infarct size. There is a rough but useful correlation between enzyme release and infarct size, especially when thrombolytic agents are not employed (36). Estimation of size based on enzyme release should be included in any comparative study of prognosis of infarction.

Studies comparing first Q with first non-Q MI. (Table 1)

Nine studies have engaged this critical variable; other listed elements were addressed in varying degree.

1. Stone et al. (37) studied 471 patients with first myocardial infarcts with a mean follow-up of 30.8 months. The infarcts were stratified as Q or non-Q, anterior or inferior. They were also grouped by quartiles of enzymatic infarct size. The authors concluded that "there was no increased rate of reinfarction or mortality in hospital survivors with non-Q compared with those with Q wave infarction and total cardiac mortality was the same." There were no differences in late outcome between patients with Q versus those with non-Q wave infarction and there was no "catch-up" phenomenon observed, i.e., no increased incidence of late infarction and late fatality in patients with initial non-Q infarction. They also noted an increased mortality and morbidity with anterior as compared with inferior infarction, even when corrected for enzyme size, and they found that Q wave infarcts were on average larger than non-Q. The mean ages of patients in each group were the same, the tall R V1 was the only "Q-equivalent" deflection recognized.
2. Nicod et al. (38) reported in-hospital and 1-year mortality in 2,024 patients stratified by age and previous myocardial infarction. These investigators found that, in patients younger than 70 years of age, "there was no difference in the incidence of major events, such as in-hospital infarct extension, recurrent myocardial infarction after discharge or coronary bypass surgery in the year

Table 1. Studies Comparing First Q With First Non-Q MI: Summary of Variables Addressed and Outcome

	1st MI only	Age	Location	Enzymes	Q-equiv.	Outcome
Stone et al. (37)	+	+	+	+	+1/2	No diff.
Nicod et al. (38)	+	+	-	-	-	Increased mortality over 70 non-Q. No diff. under 70 in acute events.
Benhorin et al. (39)	+	+	+	-	+1/2	No diff.
TIMI (40)	+	+	-	-	-1/2	No diff.
Goodman et al. (41)	+	?	?	?	?	No diff.
Zareba et al. (42)	+	+	-	-	-	No diff.
Thanavaro et al. (43)	+	+	-	+	-	No diff.
Krone et al. (44)	+	+	+	-	-	No diff. under 60 Increased mortality non-Q over 60.
Welty et al. (45)	+	-	+	-	-	No diff.

The term "no diff." means there was no difference in incidence of acute events post MI, e.g., postinfarct angina, reinfarction, extension of infarct, unstable angina, also no difference in immediate or late mortality. "+1/2" = some but not all major Q wave equivalents were recognised, (usually tall R V1); "?" = the element was not clearly recorded in the study.

after discharge in the Q and non-Q group." They found an increased incidence of angina in the non-Q group at all ages and an increased 1-year mortality only in patients over 70 with non-Q infarction. In patients under 70, the presence or absence of Q waves served no discriminative function. This study did not correct for infarct size or location.

- Benhorin et al. (39) studied 757 patients with first myocardial infarcts in patients enrolled in the Multi-center Diltiazem Post-Infarction trial with an average follow-up of 1.4 years. They found no difference in the 533 Q wave infarcts and 227 non-Q wave infarcts in cardiac mortality or in nonfatal reinfarction at the end of one year or at total follow-up. They further found no difference in any aspect of outcome between anterior and inferior-posterior infarcts although they noted that anterior infarcts were apparently larger, with significantly more severe depression of ejection fraction.
- In 1995 the TIMI investigators reported the results of a study comparing the clinical course of Q and non-Q wave infarcts (40). These investigators studied 2,643 patients with first myocardial infarcts. The patients all received thrombolytic therapy and were then randomized to "conservative" or "invasive" protocols of treatment. At the end of a year the investigators compared the course of Q wave and non-Q wave infarcts with each protocol. They found no difference in early (21 day) or 1-year mortality between the two groups in either protocol: most significantly, there was no difference in the occurrence of adverse cardiac events between the two groups during a year of observation. Moreover, an invasive approach to postthrombolytic management conferred no benefit in either group as compared with a conservative protocol. Ventricular function was assessed and, again, patients with Q wave infarcts manifested significantly more depression of left ventricular function and an increased incidence of congestive heart failure.
- In 1996 Goodman et al. (41), as part of the Gusto-I project, compared first MIs on the basis of presence or absence of Q waves in a series of 1,830 patients, 555 with non-Q MI's and 1,275 with Q wave MIs. They found that "patients with non-Q wave MI had lower 30 day and 1-year mortality (1.5% vs. 4.5% p = 0.01 and 4.7% vs. 6.8% p = 0.04, respectively), but no increase in angina or interventions and similar quality of life when compared with patients with a Q wave MI." The age of the two groups was comparable, but it is not clear from the abstract whether other variables such as Q-wave equivalents were addressed.
- Zareba et al. (42) reported a comparison between Q and non-Q first infarcts (363 Q, 186 non-Q). After a mean follow-up of 23 months, there was no difference in cardiac events (unstable angina requiring hospitalization, nonfatal reinfarction or death from cardiac causes.) The occurrence of postinfarct angina was associated with increased risk of cardiac events in patients with both infarct types. In the non-Q group there was a borderline increase in risk of cardiac events in those patients manifesting postinfarct angina (p = 0.065). No other difference was documented.
- Thanavaro et al. (43) studied 745 first infarcts and found that enzyme level, not Q wave status, was the significant determinant of outcome. There was a higher overall mortality for the Q wave group, but, in the author's words, "regardless of the electrocardiographic changes of nontransmural (non-Q) or transmural (Q) myocardial infarction we found a similar morbidity and mortality with comparable peak SGOT levels." In other words, when stratified according to enzyme levels, there was no difference between the two groups. Again, Q wave infarcts were larger in this study. Infarcts were not stratified by location and Q wave equivalent deflections were not recognized.
- Krone et al. (44) followed 593 patients after first MI for an average of 4.7 years. They found that for the first two years, enzymes were predictive of mortality regardless of Q or non-Q classification. After two years they found an increased mortality in the non-Q group only in subjects

over 60; in the group under 60 there was still no recordable difference. No evidence of an "unstable" state in the year following infarction was recorded in this study. These workers did not stratify for infarct location and did not recognize Q wave equivalents.

9. Welty et al. (45) compared the course of Q and non-Q infarcts in patients who had undergone percutaneous angioplasty for postinfarct ischemia. They found no difference in the incidence of recurrent angina, repeat coronary angioplasty, coronary bypass surgery, reinfarction or death between the two groups after a mean follow-up period of 34 months. This study documented an increased risk of subsequent events with anterior compared with inferior infarction. Q wave equivalent deflections were not recognized.

It is interesting that two studies reported a more complicated late post-MI course only in the older non-Q group (over 70 in the study by Stone et al. cited above) (37). Benhorin et al. (39) suggested that this phenomenon might follow from the increased likelihood of previous "silent" myocardial damage in the older age group. The fact that subsequent infarcts do not usually generate Q waves is particularly relevant in this setting.

In brief, nine studies have been reported comparing the post-MI course of first QMI and NQMI. With the minor exception cited above, none found any difference in incidence of acute events in the year following infarction; all, in fact, refuted the concept of the non-QMI as an "unstable" phenomenon, subject to further acute ischemic insult.

Studies supporting the concept of the "unstable" NQMI.

Review of studies supporting the conventional "unstable non-Q" concept reveals one overriding error of protocol in the great majority, i.e., comparison of random mixtures of first and subsequent infarcts, always with a sizeable minority of subsequent infarcts. (In none of these studies is it specified whether the infarcts are second or third; the term "previous MI" is used in all tables and summaries.) (46-50). It is reemphasized that beside the two to three-fold increase in morbidity and mortality to be expected with second and third infarcts, these subsequent infarcts usually do not generate Q waves (less than half as often as first infarcts); obviously a large number of these infarcts will be included in the "non-Q" category with confounding results.

A significant recent study by Goodman et al. (51) was marred by the same investigational lapse, but is of great interest nevertheless. These investigators compared the course of Q and NQMIs following thrombolysis but the authors admitted that, "Patients with evidence of prior MI on the baseline ECG were included...". They did not comment on the number of such cases and did not distinguish them in any way from subjects suffering a first MI. Despite this problem, the findings of this study are significant. In-hospital, 30-day, 1-year and 2-year mortality were all lower in the non-Q group. (6.3% vs. 10.1%) The authors did not record any increased incidence of acute events in the

postinfarct period in the NQMI group as compared to QMI. Ventricular function was significantly better in the NQMI group with a median ejection fraction of 66% as compared to 57% following QMI. All these findings reinforce the data from earlier studies demonstrating that Q wave infarcts are, on average, larger than non-Q. The lack of any evidence of an "unstable" course in NQMI following thrombolysis corresponds to the TIMI findings described above (40).

Angiographic findings in Q and NQMI. A number of studies have documented identical incidence and degree of 1, 2 and 3-vessel disease with Q and non-Q MI (52,53). However, it has been claimed that early angiographic findings differ in Q and NQMI. One study (54) noted that total occlusion of the infarct-related vessel was present in 26%, 37% and 42% of patients studied, respectively, less than 24 h, 24-72 h, and 72-h to seven days after NQMI. The same group (55) documented total occlusion of infarct-related vessel in 87% of QMI's studied within four h and 65% in patients studied 12-24 h after onset of symptoms.

The possible sources of error in these and similar studies include all those listed above with a major source of error inherent in the practice of limiting ECG observation to simple Q waves while ignoring other acute changes in depolarization or "Q-wave equivalents." These studies, in fact, did not even recognize such obvious Q wave equivalent deflections as the tall R-V1 of posterior infarction. Thus, it can be reasonably predicted that a sizeable proportion of the "non-Q" infarcts in these and other studies in fact belonged in the "Q-wave" group. Even overlooking this confounding error, these studies document only a percentage difference in total occlusion between the two groups with substantial overlap, (e.g., difference 22% between Q and non-Q, 12-24 h after onset of symptoms). These figures do not permit a precise distinction between the two groups.

Invasive versus noninvasive management of NQMI.

Four studies have addressed the benefit of invasive versus noninvasive management of NQMI. The first was the TIMI study described above which found no benefit from invasive management as compared with noninvasive following either QMI or NQMI postthrombolysis (40).

The second was a smaller study by the TIMI III B group in which 476 NQMI subjects were randomized to invasive or noninvasive management with documentation of 6-week outcomes. There was no difference in survival or incidence of acute events between the two groups. This study, however, was limited to short-term outcomes and left open the possibility that some difference between the two groups might emerge later (56).

The VANQWISH project carried on by Boden et al. appears to be definitive (57). Nine-hundred twenty patients were entered in a randomized, prospective study comparing the results of invasive versus conservative management following NQMI. Death and recurrent MI were assessed during a 12-44 month follow-up (mean, 2.5 years). There

was no benefit from aggressive invasive management and in fact the authors concluded that the patients so treated "might be harmed." Editorial comment (58) amplified and expanded this view.

A retrospective study by Ghazzal et al. of 403 NQMI patients found no statistical difference in one-year and five-year survival between groups managed conservatively or invasively (59). In brief, all studies to date have demonstrated no benefit of invasive as compared with conservative management of NQMI, with or without thrombolysis.

Of tangential interest only are recent studies of troponin "leak" as a marker of acute myocardial ischemia (60-62). Without exception, these studies have uncritically accepted the common association of non-Q MI with crescendo angina under the heading of an "acute coronary syndrome" without citing any basis for such a classification and have limited investigation to this specious grouping. An elevated troponin-I assay was shown to have a statistical association with subsequent mortality and morbidity but in none of these studies was there a comparison of troponin-I levels in QMI as compared to NQMI. Further, these studies have provided no comparison of clinical outcome of QMI versus NQMI and hence have no bearing on the issues raised here. While troponin appears to be a useful addition to the enzyme armamentarium and may provide more prognostic information than CPK, these articles are of interest here chiefly because they illustrate to what an extent the concept of the "unstable" NQMI has dominated current cardiologic perception.

Summary. The whole subject can thus be summed up in two statements.

1. Every appropriately designed study comparing first Q and NQMI's has found no difference in post-MI course of the two categories and no foundation for the common notion that the NQMI is a uniquely "unstable" entity, to be classed with unstable angina in terms of prognosis and management. Nine such studies have been published. On the other hand, all studies alleging the "unstable" character of the NQMI have been invalidated by major flaws, chief among them the comparison of undifferentiated mixtures of first and subsequent infarcts with widely differing mortality and morbidity. This confusion is further compounded by the fact that subsequent infarcts generate Q waves less than half as often as first infarcts.
2. All current studies indicate that there is no benefit to an invasive as compared with a conservative protocol for management of NQMI. Since the characterization of an infarct as "non-Q" conveys no therapeutic implications, the classification becomes irrelevant and should be discarded.

Two quotations sum the whole matter succinctly. Moss (63) commented that "The Q-wave versus non-Q-wave categorization does not provide sufficient sensitivity, speci-

ficity, or predictive accuracy about the subsequent clinical course of patients with a first myocardial infarction to use it as reliable data in the clinical decision-making process." Surawicz (64) put the matter even more concisely: "... a non-Q wave MI is not a unique entity: rather it is a smaller and less extensive MI."

In a word, the magnitude of a myocardial infarction should be judged on anatomical and functional considerations rather than on the designation of Q wave versus non-Q wave infarction.

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