In 1980, Pipberger and Lopez (1) published an excellent review entitled "‘Silent’ Subendocardial Infarcts: Fact or Fiction." They noted that the attempt to distinguish transmural from subendocardial infarcts on the basis of presence or absence of Q waves was not justified by any clinical, pathologic or experimental data. The authors observed that this common misconception arose from a single flawed study, later repudiated by the original investigator and by every credible study since that time. They ended on a despairing note, commenting that this absurdity was so deeply embedded in cardiologic folklore that it would probably never be extirpated.

Two years later, the authors' worst fears seem to have been realized. Publications continued to base conclusions on this imaginary distinction (2-4) and references to "subendocardial" and "transmural" infarcts, electrocardiographically delineated, are common among cardiologists and clinicians generally. At the recent (1982) scientific sessions of the American College of Cardiology, nine presentations (5-13) were concerned with this imaginary distinction or mentioned it prominently.

The concern transcends semantics; physicians persist in drawing therapeutic and pathologic conclusions from the presence or absence of Q waves, quite possibly to the detriment of the patient. In an era of scarce research dollars, it is unconscionable to waste time and resources to study a distinction that has no basis in fact. This paper, therefore, presents a brief review of the history of this misconception and its subsequent dissemination throughout the practice of cardiology.

Early Views

Wilson and associates (14) made the earliest well grounded statement about the relation of the pathologic Q wave to subendocardial infarction: "When the subendocardial layers of muscle are for the most part dead or incapable of responding to the cardiac impulse, and the outer layers of muscle are relatively normal, the embryonic RR arises above the baseline and becomes a true R deflection. The QRS complex then consists of an abnormally large Q wave, followed by a small R and often by an S deflection."

This view was not challenged until Prinzmetal et al. (15) studied 7 dogs with subendocardial infarction produced by coronary ligation and 12 dogs with subendocardial necrosis produced by electrical injury. The conclusions drawn from this limited number of studies were unusually dogmatic and their application to clinical electrocardiography was breathtaking. The investigators reported that the subendocardial region was electrically "silent" and that the ventricular complex recorded over a normal heart was found to result from activation of the outer ventricular layers and was not significantly affected by subendocardial depolarization. "Pure subendocardial infarcts do not significantly alter the depolarization complex, and since the inner ventricular myocardium appears to have little or no effect on the electrocardiogram, clinical electrocardiography is of limited value in the diagnosis of myocardial disease which does not involve the superficial layers of the heart." The investigators made no attempt to reconcile their findings with those of Wilson et al.

It is astonishing that in 1957, the same investigators partially repudiated this study, using more sophisticated methods, discovering that the subendocardium was not electrically "silent" but actually took part in the generation of the surface electrocardiogram (16). It is one of the oddest quirks of modern cardiology that the 1954 study (15) was accepted as definitive and the later study, which corrected some of the errors in the earlier work, remained largely unnoticed.

Q Waves in Subendocardial Infarction

Two concurrent lines of investigation and publication developed, each apparently oblivious of the other, though both were adequately reported. On the one hand, a number
of well designed experimental and clinical pathologic studies demonstrated beyond question that presence or absence of pathologic Q waves had no relation to the subendocardial or transmural character of myocardial infarcts. On the other hand, many competent investigators pursued the "difference" between subendocardial and transmural infarcts in terms of prognosis, therapeutic implications, acute and chronic course and so forth, using the presence or absence of Q waves as the only means of differentiation. It was as though electrocardiographic investigators splint at that time—one group following well planned studies leading to a clear-cut conclusion, and the other pursuing an ignis fatuus.

Durrer et al. (17) studied electrocardiographic correlates in dogs after ligation of coronary arteries. Both epicardial and intramural potentials were determined and meticulous postmortem correlations were carried out. These workers observed that "even the smallest subendocardial scar present in our series with the largest diameter of nearly one centimeter and intramural extension of less than one-fourth of the thickness of the left ventricular wall resulted in the occurrence of abnormal Q waves in unipolar epicardial complexes . . . we found no evidence for the existence of a silent zone . . . the main reason for the abnormal Q wave in subendocardial infarction is the loss of voltage."

Two studies by Abildskov and co-workers (18,19) again demonstrated clearly that both experimental and clinical subendocardial infarcts were often accompanied by pathologic Q waves. The authors commented that "in the present study only two of the lesions were not transmural (on clinical pathologic correlation) but both resulted in changes attributed to localized tissue loss, including the occurrence of pathologic Q waves. This is comparable to previous experimental findings where non-transmural lesions also resulted in pathologic Q waves." Cook and co-workers (20,21) contributed two clinical-pathologic studies, demonstrating that large nontransmural infarcts generated Q waves identical to those thought to characterize "transmural" infarcts. The authors speculated that "the change from a subendocardial to a transmural type of electrographic pattern occurs when an infarct involves somewhat more than the inner half of the thickness of the ventricular wall." The investigators also noted that there were QRS changes of some type in all five necropsy-proved large subendocardial infarcts, thus refuting the postulate that the subendocardium is "electrically silent."

Savage et al. (22) in 1976, on the basis of careful retrospective clinical-pathologic correlation, again demonstrated that a number of subendocardial or nontransmural infarcts were accompanied by pathologic Q waves. They commented that "the concept that infarcts will cause alterations in the QRS only when they are 'transmural' has not been validated by this study. Indeed, many of the patients in both the anterior and posterior groups with definitive Q waves had infarcts limited to the subendocardium."

Raunio et al. (23) studied postmortem examinations in 91 patients dying of myocardial infarction. In electrocardiograms taken 48 hours before death, QRS changes of infarction were noted in 53% of 15 patients with subendocardial infarcts and in 65% of 65 patients with transmural infarcts. In other words, about half the subendocardial infarcts actually generated pathologic Q waves, while about half the transmural infarcts did not. In terms of both accuracy and sensitivity, therefore, in this study, the Q wave of the surface electrocardiogram was useless. A clear line of experimental and pathologic evidence thus negated Prinzmetal's 1954 study and proved beyond any question that the presence or absence of pathologic Q waves had no relation to the transmural or subendocardial character of a myocardial infarct. So much for serious investigation.

Clinical and Prognostic Significance of Infarct With and Without Q Waves

Apparently disregarding all this evidence, a series of publications continued to appear over the years as investigators compared the clinical course and prognostic significance of infarcts with and without Q waves. In two studies in 1973, Scheinman and Abbott (24,25) compared "transmural versus non-transmural" infarcts in terms of clinical significance. They also compared patients with diagnostic (Q wave) and nondiagnostic (ST-T only) electrocardiograms in the presence of acute myocardial infarction. There was no difference in clinical course or prognosis of either group. Stimmel et al. (26) studied patients who were "considered to have undergone a subendocardial infarction" on the basis of the usual symptoms and ST-T changes without Q waves. They were surprised to note that a number of these patients did develop pathologic Q waves later in their hospital course and they concluded that the "subendocardial" infarct had progressed to a "transmural" infarct. Again, the presence or absence of Q waves was the only criterion for distinguishing the two types of infarct.

In 1975, Rigo et al. (27) compared "hemodynamic and prognostic findings in patients with transmural and non-transmural infarction," noting that "there was no significant difference in in-hospital mortality between those with transmural infarction and non-transmural infarction . . . the late mortality of those surviving their initial hospitalization was also not different between those with transmural infarction (18%) and non-transmural infarction (19%)."

Cannom et al. (28), studying non-Q wave infarcts, concluded that this "subendocardial" group had a worse prognosis than the "transmural" group, that is, those with Q wave infarcts. Vaisrub (29), commenting editorially on this study, seemed to be surprised that patients in the non-Q wave group should encounter a graver prognosis than those in the Q wave group of infarcts. Similar studies based on
this fancied differentiation appeared concurrently or subsequenly (4,30–35).

In these and other articles, the investigators unanimously reached the conclusion that there was no clinical difference between the two subgroups or, in some cases, decided that “subendocardial” infarcts implied a graver prognosis than the “transmural” variety. Without exception, they were comparing two completely random mixtures of subendocardial and true transmural infarcts; any supposed differences that emerged must have been the result of pure chance. Rarely in biologic science has there been such a striking example of the left hand not being aware of what the right hand was doing.

**Conclusions**

To summarize:

1) Abundant experimental and clinical-pathologic evidence now exists to establish permanently and irrefutably the fact that presence or absence of Q waves in the surface electrocardiogram does not permit distinction between transmural and subendocardial infarcts. In terms of both sensitivity and accuracy, the Q wave is a useless observation in this setting. (This, of course, must be distinguished from the unquestioned usefulness of the Q wave in diagnosing infarction as such.) Presence or absence of Q waves accompanying myocardial infarction does not delineate or differentiate any pathologic or clinical subset.

2) The use of the term “Q wave infarct” or “non-Q wave infarct,” which has emerged in a kind of half-way house of the intellect, should be avoided, since it implies a distinction between two varieties of infarction—a distinction that has no basis in pathologic fact.

3) Editors, research committees and formulators of electrocardiographic reading standards should use every effort to drop this misleading and potentially dangerous distinction, deeply embedded in cardiology, from the lexicon of electrocardiographic terminology.

**References**


