Q-Wave Infarction Versus S-T Infarction

Nonspecificity of Electrocardiographic Criteria for Differentiating Transmural and Nontransmural Lesions

DAVID H. SPODICK, MD, DSc

Nonspecificity of the Electrocardiogram

For several decades cardiologists have considered abnormal Q waves synonymous with transmural myocardial infarction, as distinguished from infarcts characterized only by ST-T abnormalities, labelled "nontransmural" or "subendocardial." Curiously, journal reviewers have not vetoed these misnomers despite repeated marshalling of evidence demonstrating at autopsy that abnormal Q waves occur frequently with anatomically nontransmural infarction and may be absent with transmural infarcts.[1–3] This evidence first appeared about 40 years ago and has consistently been substantiated.[4–19] Indeed, early investigators who reported an electrocardiographic distinction between the 2 anatomic types of infarct failed to prove it on examination of their own illustrations: among 4 electrocardiograms illustrating nontransmural infarcts, 1 shows incomplete left bundle branch block and 3 clearly have pathologic Q waves[15]; among 6 subendocardial infarcts demonstrated pathologically, 2 electrocardiograms show incomplete left bundle branch block, 2 have abnormal (≥0.04 second) Q waves in lead aVF, and 2 were too poorly reproduced to decipher.[5] (This is especially surprising considering that illustrations invariably put the author's "best foot forward.") More recently, among 12 of 24 autopsied subjects with nontransmural infarcts, 10 had QRS changes, 8 with abnormal Q waves (including anatomically small infarcts); the other 12 patients had transmural infarcts, but 5 had no abnormal Q waves.[8] Another recent report confirms postmortem documentation of transmural infarcts with only ST-T changes.[11] In a series of 154 autopsy subjects, most of those with nontransmural infarction had typical QRS changes and many with transmural infarcts lacked pathologic Q waves.[7]

It is possible that some earlier work may have established an *idée fixe* in the mind of cardiologists, despite the early availability of a mass of contrary evidence.[4–6,17,19] Indeed, Wilson, a pioneer in cardiology, and his colleagues[19] demonstrated abnormal Q waves in nontransmural infarction both experimentally and clinically. The perceptive observations of Pipberger and Lopez[20] suggest that the erroneous criteria may have been reinforced by Prinzmetal's experiments of 1954, subsequently invalidated by the same investigator in 1957.

From the Department of Medicine, University of Massachusetts Medical School, and the Division of Cardiology, St. Vincent Hospital, Worcester, Massachusetts. Manuscript received October 12, 1982; revised manuscript received October 28, 1982, accepted November 22, 1982.

Address for reprints: David H. Spodick MD, Director of Cardiology, St. Vincent Hospital, 25 Winthrop Street, Worcester, Massachusetts 01604.

Conceptual Errors Inhibit Investigation

Whatever the reason for continued confusion regarding their anatomic extent, the pseudospecificity of Q and S-T myocardial infarctions remains a lasting myth. Indeed, investigators of the highest intellect and skill—despite explicitly acknowledging the material cited in References 1 to 3,[21]—write "regardless of the terms used. . . ,"[21] implying that slipshod terminology is unimportant. Such disregard by distinguished colleagues is disturbing. We are not dealing with minor matters such as "ECG" (English) versus "EKG" (Dutch and German). Arguments over terminology would be mindless quibbles if misleading labels did not perpetuate misleading concepts. False terminology does more than semantic damage: it does conceptual damage. If we continue to accept a Q infarct as dependably indicating a transmural lesion, we can inhibit consideration of what it really does mean. Indeed, do Q infarcts and S-T infarcts have distinctive gross anatomy? Given the ultimate nonspecificity of many electrocardiographic findings it should not be surprising that they overlap to such a degree that "transmural" and "nontransmural" are gross misnomers.

Q Infarcts Versus S-T Infarcts: Differences and Similarities

Although their gross anatomy cannot yet be predicted electrocardiographically, Q-wave and S-T infarcts differ clinically and physiologically. Prodromal symptoms are more frequent with S-T infarcts[22]; vomiting is more frequent with Q-wave infarcts.[18] Q-wave infarction implies more damage as measured by peak creatine phosphokinase levels,[23] with a greater tendency to undergo expansion.[34] Positron emission tomography[25] shows defects with homogeneous depression of tracer

TABLE I Q-Wave Infarction Versus S-T Infarction: Differences

	Q-Wave Infarct	S-T Infarct*
Fresh thrombosis	More	Less
Collateral vessels	Fewer	More
Damage		
↑Enzyme levels	Higher	Lower
↓Tracer concentration (PET)	Homogeneous	Unequal
LV wall abnormality,†	More	Less
improved by bypass	Less or none	More
Tendency to expand	More	Less
Prodromal symptoms	Less	More
Vomiting	More	Less
Atrioventricular and intraventricular blocks	More	Fewer
Congestive failure	More	Less
Early mortality	More	Less
Recurrent infarction	Less	More

* Without QRS abnormality. † Some studies differ.
PET = positron emission tomography.

TABLE II Q-Wave Infarction Versus S-T Infarction: Similarities

Both either transmural or nontransmural
Number of diseased vessels
Degree of occlusion throughout coronary arteries
Ejection fraction*
Hypotension
Ventricular arrhythmia*
Late (>2 year) mortality

* Some studies differ.

accumulation in all Q-wave infarcts as compared with unequal tracer distribution in 23 of 24 S-T infarcts, with tomographic infarct size greater and residual tracer more depressed in Q-wave infarcts.[28] Two-dimensional echocardiograms showed more (100%) regional wall abnormality during Q-wave than S-T infarction (86%).[26,27] Although total mortality may equalize over 5 to 10 years in the 2 groups, early mortality both in the hospital and after discharge is greater with Q-wave infarcts, whereas recurrent infarction and late mortality are much greater with S-T infarcts.[28,30]

Although the number of diseased coronary arteries and the degree of occlusion throughout the system appear equal in both types,[23] fresh coronary thrombosis may be slightly[31] to moderately[10] more frequent with Q-wave infarction. Collateral vessels were demonstrated in 93% of all patients with S-T infarct and 78% with Q-wave infarct who had postinfarction angina, but in only 35% of patients with Q-wave infarct and no subsequent angina.[31] Coronary bypass tends to improve wall motion in S-T infarcts but only in Q-wave infarcts in which transmural fibrosis is absent.[32,33] Some investigators found reinfarction, angina, and late ventricular arrhythmias to occur equally in Q-wave and S-T infarcts, but more akinesis in patients with anterior Q-wave than with anterior S-T infarcts.[34] Yet, other investigators report comparable ejection fractions and wall motion abnormalities.[23] Finally, in a carefully matched, although retrospective, series S-T infarcts produced less congestive failure, fewer intraventricular conduction defects, and fewer atrial tachyarrhythmias; hypotension and ventricular arrhythmias occurred equally.[21] Yet, the same study also reported a statistically equal occurrence of pericarditis.[21] Because meticulous postmortem examination shows that in fresh infarction acute pericarditis always indicates transmural lesions,[10] those non-Q–wave infarcts with pericarditis, although labelled "nontransmural,"[21] certainly represented transmural infarction—more evidence of the electrocardiogram's anatomic nonspecificity.

Subdivisions Within Q Infarctions and S-T Infarctions

Investigations pertinent to this analysis correlated anatomically transmural and nontransmural infarcts with electrocardiographically Q-wave and S-T infarcts, irrespective of regional distribution.[4–19] Fresh S-T infarcts usually occur with a normal QRS, but may also occur in patients with previous infarction, ventricular hypertrophy, preexcitation, or conduction disturbances producing QRS changes that mask or cancel processes producing new Q waves. Although most Q-wave infarcts are predominantly either inferior or anterior, it is not established whether either location is more likely to be transmural or nontransmural. Yet, location may have clinical importance: overall mortality in some series is worse with anterior infarcts, perhaps because of their tendency to be larger than inferior infarcts.[35,36] In contrast, in the AMIS Study (4,524 participants) total mortality did not differ among infarct locations, including patients who lost abnormal Q waves.[37] Yet, ventricular dysfunction tends to be worse with anterior Q waves.[38,39] Finally, both bundle branch block and significant myocardial failure are more frequent with anterior Q-wave infarcts, whereas sinus bradycardia, atrioventricular block, and atrial arrhythmias are more frequent with inferior Q-wave infarcts.[21]

Q-Wave Infarcts Versus S-T Infarcts

Tables I and II list the similarities and the many differences between Q-wave and S-T infarction. Q-wave infarcts include posterior infarction appearing as a new anterior R wave.

Conclusions: The unfortunate practice of mislabelling Q-wave infarction as "transmural" and S-T infarction as "nontransmural" does not correspond to the pathologic evidence. Q-wave infarcts and S-T infarcts differ clinically, physiologically, and prognostically but cannot be anatomically differentiated by electrocardiography. As with a variety of other loose or erroneous terminologies, the absence of precise labelling interferes with clarity of concept and may inhibit further investigation.[40] When a myocardial infarct is characterized strictly by electrocardiography, it should be described by the only accurate terminology—Q-wave infarct or S-T infarct. Confucious tells us:

> If language is not used rightly, then what is said is not what is meant. If what is said is not what is meant, then that which ought to be done is left undone.

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