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LETTERS TO THE EDITOR

Transmural Versus Q Wave Infarction

Mukharji et al. (1) perpetuate a myth that by now should have been dispelled. They attempt to define "transmural" myocardial infarcts on the basis of the presence of "new Q waves at least 40 ms wide and 0.2 mV deep."

I should like to refer these authors to my article (2) and one by Spodick (3) in this Journal. Spodick and I, after an exhaustive review of all available pathologic and correlated data, concluded, as had previous investigators (4), that there is no basis for the assumption that the presence of a pathologic Q wave indicates a transmural infarct, or that the absence of such a Q wave is correlated with a "subendocardial" abnormality. Exhaustive electrocardiographic and pathologic correlations by the best investigators in the world have shown that there is a random association between the transmural or nontransmural character of an infarct and the presence or absence of pathologic Q waves. Spodick, Pipberger and I all pointed out that this misconception is based on the study of 12 dogs by Prinzmetal, which was later acknowledged to be faulty and which was repudiated by the investigator himself. The astonishing fact is that this hoary myth continues to plague cardiology and causes a considerable waste of time, effort and money by investigators.

Even the smallest subendocardial infarct is quite likely to produce a pathologic Q wave, while very large transmural infarcts have no more than a 50% likelihood of producing such a deflection. The association between pathologic Q waves and the transmural or nontransmural character of an infarct is a random one; the investigator might as well flip a nickel and describe the infarct as "Indian head" or "buffalo" in character. It would have just as much relation to the actual abnormality.

Why are some infarcts accompanied by Q waves and others not? Nobody knows, and the subject is certainly worthy of study. What is known is that the two categories of "Q wave infarcts" and "non Q wave infarcts" each include a random mixture of every kind of pathologic feature known to accompany acute myocardial infarction; namely, transmural, subendocardial, patchy, confluent, large or small.

One is not surprised to see myths and superstition assume a life of their own in the realm of folklore and legend, but they certainly have no place in modern medical science.

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1. Mukharji J, Murray S, Lewis SE, et al. Is anterior ST depression with acute transmural inferior infarction due to posterior infarction? A vectorcardiographic and scintigraphic study. J Am Coll Cardiol 1984;4:28–34.
2. Phibbs B. "Transmural" versus "subendocardial" myocardial infarction: an electrocardiographic myth. J Am Coll Cardiol 1983;1:561–4.
3. Spodick DH. Q-wave infarction versus S-T infarction—nonspecificity of electrocardiographic criteria for differentiating transmural and nontransmural lesions. Am J Cardiol 1983;51:913–5.
4. Pipberger HV, Lopez EA. "Silent" subendocardial infarcts: fact or fiction? Am Heart J 1980;100:597–9.

Reply

We are entirely in agreement with the informed and valid discussion by Phibbs. However, our manuscript was not aimed at examining the relation between the pathologic transmurality of infarcts and the electrocardiographic markers thereof. Neither do we wish to suggest that Q waves on an electrocardiogram necessarily have any pathologic implication. Our study was designed to examine the relation between inferior infarction and anterior ST depression. This should be clear from a cursory examination of the text. In identifying patients with inferior infarction, we chose the presence of Q waves in the inferior leads on electrocardiograms. We did so because, although Q waves do not necessarily connote transmurality, they are nevertheless the most specific electrocardiographic markers of myocardial necrosis. Thus, we seem to be guilty of semantic license in using the terms "Q wave infarction" and "transmural infarction" interchangeably. Such license is blessed by years of traditional acceptance in the cardiology literature. Since the term "transmural" is more familiar and less cumbersome to most readers than "Q wave infarction," the only myth that we take credit for perpetuating is that "a rose by any other name smells as sweet."

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Intimal Flap Prolapse in Aortic Dissection

The report of Sraow et al. (1) appears to be essentially a duplicate of our previous report in the *British Heart Journal* (2) except that we emphasized the M-mode findings as we felt that M-mode equipment was more readily available to the practicing physician. Inspection of their Figure 1 suggests, in fact, that the last aortic valve echo may show the reduplication we reported. Their Figure 2 duplicates our Figures 2 and 3, except that the flap prolapsed

into the left ventricular outflow tract, while in our case, the flap fell onto the coapted aortic valve leaflets.

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1. Sraow J, Desser K, Benchimol A, DeSa'Neto A, Peebles S. Two-dimensional echocardiographic recognition of an aortic intimal flap prolapsing into the left ventricular outflow tract. J Am Coll Cardiol 1984;4:180–2.
2. Cohen I, Wharton T. "Duplication" of aortic cusp: new M-mode echocardiographic sign of intimal tear in aortic dissection. Br Heart J 1982;47:173–6.

Reply

1. Inspection of our Figure 1 indicates that the M-mode aortic echo may show the duplication reported by Cohen and Wharton. Duplication of aortic structures in patients with dissection has been described before and was not the cardinal feature of our report.

2. At the time of their publication (1982), M-mode equipment may have been more readily available. Currently, the proper application of echocardiography for the definitive diagnosis of aortic dissection mandates both M-mode and two-dimensional echocardiographic studies in conjunction. Interestingly enough, the genesis of M-mode duplication was explicated by a two-dimensional echocardiogram (Fig. 2) in their case report.

3. Our Figure 2 does not "duplicate" their Figures 2 and 3 since the intimal flap in our study prolapsed into the left ventricle. This prolapse was in fact the primary description in our report. Prolapse of a clearly defined flap may be more specific for the diagnosis of aortic dissection than isolated duplication of aortic root structures on an M-mode examination. Further study will ascertain the relative specificity of these two findings for the identification of dissection.

4. If Cohen and Wharton had demonstrated prolapse of an intimal flap on the two-dimensional ultrasonic study in a patient with nonspecific findings on a computed axial tomographic study of the thorax and no identifiable false lumen on aortic angiography then the two papers might be construed as duplication. Such was not the case. The exception of prolapse defies the term "duplicate" in this context.

5. The importance of the case report by Cohen and Wharton is hereby acknowledged. Intimal flap prolapse represents a new and important finding for the diagnosis of aortic dissection, a feature that was not described in their report.

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Correction

Because of a printer's error, Figure 3, which appeared on page 1190 in the article by Sasayama et al. (Three-Dimensional Analysis of Regional Myocardial Function in Response to Nitroglycerin in Patients With Coronary Artery Disease. J Am Coll Cardiol 1984;3:1187–96), is incorrect. The figure that should have appeared was reproduced on page 200 of the July 1984 issue of the Journal (J Am Coll Cardiol 1984;4:200), but was printed upside down.