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pressure monitoring and echocardiographic assessment of hemodynamically unstable patients, could critical care clinicians be persuaded to choose the least invasive technique. With increasing numbers of echocardiographers (cardiologists or other specialists with adequate training in echocardiography) involved in critical care management, this choice could start a new trend, eventually leading to reduced costs. Furthermore, as smaller and less expensive ultrasound units in conjunction with smaller probes become available, continuous monitoring (e.g., of the short axis of the left and right ventricles) should be possible. Thorough, lengthy training in echocardiography for all critical care providers would further improve critical patient care.

JAN POELAERT, MD, PHD
Department of Intensive Care
University Hospital
de Pintelaan 185
B9000 Ghent, Belgium

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Reply

We thank Poelaert for his interest in our report. We agree that transesophageal echocardiography frequently provides important information that is not evident from pulmonary artery catheterization. We are interested and encouraged to learn that his study demonstrated a similar management change rate after transesophageal echocardiography in patients with pulmonary artery catheters.

We also agree with his suggestion that transesophageal echocardiography may obviate the need for pulmonary artery catheterization in a significant number of patients with hypotension. His recommendation that hemodynamically unstable patients receive the less invasive procedure before right heart catheterization is reasonable if the transesophageal echocardiogram is readily available. However, demonstrating conclusively that this strategy is optimal may prove difficult. To prove that there is no clinically significant difference in outcome with the noninvasive strategy would require a very large randomized trial.

We agree that cost of therapy may prove the deciding factor. In today's cost-conscious market, cost-effectiveness will have to be demonstrated before either a one-time transesophageal echocardiogram or continuous monitoring can be widely recommended.

PAUL HEIDENREICH, MD
ELYSE FOSTER, MD, FACC
NEAL COHEN, MD
School of Medicine
University of California–San Francisco
San Francisco, California 94143

Q Wave and Non-Q Wave Myocardial Infarction After Thrombolysis

Matetzky et al. (1) recently reported the results of a study of 150 patients with acute myocardial infarction who received thrombolytic therapy. The authors found that 80% of patients had a Q wave and 20% a non-Q wave infarction on the 24-h electrocardiogram (ECG); no significant differences were noted between these groups with regard to either in-hospital clinical course or long-term prognosis. In contrast, predischarge ECG analysis revealed that 72% of patients had a Q wave and 28% a non-Q wave infarction. This predischarge ECG stratification was a more useful prognostic descriptor in that a trend toward lower 2-year mortality was seen among the non-Q wave group, despite a higher incidence of reinfarction and revascularization during this time period.

The differences in prognostic information conveyed by a predischarge rather than a 24-h ECG in this study appears to relate to a "crossover" from one group to the other, with the disappearance of pathologic Q waves in 18 patients and the subsequent development of Q waves in 7 patients during the hospital period. Surprisingly, the authors do not comment on a previously published study (2) that also describes the evolution and prognostic importance of Q waves after thrombolytic therapy. In contrast, this latter study concluded that the development of Q waves beyond the 24-h window after thrombolytic therapy to the time of hospital discharge was infrequent (1.5% of 201 patients). Further, the early and 1-year prognosis among the non-Q and Q wave groups was similar (2).

Clearly, the timing of ECG stratification after thrombolytic therapy is an important factor in the interpretation of the prognostic utility of Q waves versus non-Q waves. Indeed, it is difficult to reconcile the apparent differences in Q wave evolution and subsequent prognosis after thrombolysis seen in the few contemporary published reports. The incidence of non-Q waves ranges from as low as 13% to 15% (2,3) to as high as 43% (4,5), with a few of the large international thrombolytic trials suggesting that the occurrence of non-Q wave myocardial infarction after thrombolysis is between 20% and 30% (6–8). Even the subsequent prognosis of non-Q waves appears to vary dramatically. Aguirre et al. (8) described a trend toward a higher 1-year reinfarction rate but a similar mortality rate among the non-Q wave group compared with the Q wave group in a secondary analysis of the Thrombolysis in Myocardial Infarction (TIMI) II trial. Tajer et al. (6) described a significantly higher 6-month reinfarction and mortality rates among non-Q wave myocardial infarction hospital survivors in the Tissue Plasminogen Activator Versus Streptokinase Trial (TPASK). Barbagelata et al. (7) described a significantly lower 30-day and 1-year mortality rate among the non-Q wave group in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries (GUSTO) trial.

I would appreciate some comment by Matetzky et al. on these issues, particularly because four of the coauthors of this recent publication were also coauthors of the earlier and apparently conflicting analysis (2) of postthrombolytic Q wave evolution and prognosis, and they failed to mention this latter study in their current discussion and conclusions regarding the timing and value of Q wave/non-Q wave dichotomization in the thrombolytic era.

SHAUN GOODMAN, MD, FRCPC
Division of Cardiology
St. Michael's Hospital
30 Bond Street
Toronto, Ontario, Canada M5B 1W8

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Reply

We appreciate the comments made by Goodman with regard to our recent study and would like to clarify some points mentioned by him. Goodman suggests a discrepancy between our findings and those of Eisenberg et al. (1). However, there are a few important differences between the two studies.

Eisenberg et al. (1) classified the patients according to Q wave appearance (on admission, 0 to 3 h, 3 to 24 h and 24 h to discharge and patients with non-Q wave myocardial infarction) and showed the importance of the time of Q wave appearance after thrombolytic therapy. Matetzky et al. classified the patients according to eventual electrocardiographic (ECG) pattern at two time points: 24 h and before discharge and demonstrated Q wave regression in a substantial number of patients with a Q wave myocardial infarction early (24-h ECG) after thrombolytic therapy and late Q wave appearance in a few patients.

Although the proportion of patients with a non-Q wave myocardial infarction at the end of the first 24 h in the study of Eisenberg et al. (1) (16%) was similar to that in the study of Matetzky et al. (2) (20%), it was substantially smaller than that on the discharge ECG (28%) in the Matetzky et al. study, where the dynamic changes in the Q waves throughout the hospital period were manifested. Thus, the two studies looked at the same phenomenon but from a different standpoint.

Eisenberg et al. (1) did not compare, as did Matetzky et al. (2), patients with a Q wave and non-Q wave myocardial infarction, but grouped together patients with Q wave appearance within 3 h ("early group") and those with Q wave appearance beyond the first 3 h and with non-Q wave myocardial infarction ("delayed group") and compared these two groups.

Contrary to the remarks of Goodman with regard to the study of Eisenberg et al. that "the early and 1-year prognosis among the non-Q and Q wave groups was similar," when the patients in the study of Eisenberg et al. are grouped on the basis of the presence or absence of Q waves, important differences existed, resembling the differences that Matetzky et al. had shown. Patients with a Q wave myocardial infarction compared with those with a non-Q wave myocardial infarction had higher peak creatine kinase levels (1,251 to 1,081 vs. 661), a higher incidence of heart failure (13% vs. 3%) and a higher in-hospital mortality rate.

Goodman points toward another potential discrepancy between the two studies—the rate of Q wave appearance after the first 24 h: 1.5% in the study of Eisenberg et al. versus 5% in the study of Matetzky et al. However, whereas Eisenberg et al. reported the rate of at least one new Q wave appearance, Matetzky et al. reported the rate of patients moving from non-Q wave to Q wave myocardial infarction, which is something different. Moreover, this discrepancy might represent a higher rate of late reocclusion among the patients of Matetzky et al., where more successful thrombolysis might be anticipated as a result of earlier thrombolytic therapy (within 4 vs. 6 h) and administration of recombinant tissue-type plasminogen activator to all patients compared with treatment with streptokinase in 27% of the patients in the study by Eisenberg et al.

In conclusion, the two studies are complementary rather than contradictory, and both add important information to the published data:

1. The timing of Q wave appearance after thrombolytic therapy carries important prognostic information independent of the later natural history of the Q waves.

2. In postthrombolytic patients, the dichotomization for Q and non-Q waves is still important for risk stratification and should be determined according to the discharge ECG.

We thank Goodman for commenting on our report and hope that we have clarified the points raised in his letter.

SHLOMI MATETZKY, MD
GABRIEL I. BARABASH, MD
BABETH RABINOWITZ, MD, FACC
SHMUEL RATH, MD
YEDAEL HAR ZAHAV, MD
OREN AGRANAT, MD
ELIEZER KAPLINSKY, MD, FACC
HANOCH HOD, MD, FACC
The Heart Institute
Sheba Medical Center
Tel-Hashomer, 52621 Israel

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