LETTERS TO THE EDITOR

Rescue Angioplasty After Failed Thrombolysis—High Mortality Despite Successful Procedure

Ross et al. (1) have presented the largest prospective and prespecified series to date of rescue angioplasty procedures, which is of considerable interest. One important point should be made. In the text they state the following:

“Patients with successful thrombolysis had the lowest 30-day mortality rate, followed by those with successful rescue PTCA and those in whom no rescue was attempted.”

It is hard to reconcile this statement with Figure 1, which depicts 30-day mortality rates in the successful rescue group and conservatively treated lytic failure group of 8.6% and 7.9% respectively.

One of the major controversies concerning rescue angioplasty is the distressingly high mortality rate following a failed rescue attempt. Unfortunately, the 30-day mortality after successful rescue angioplasty in this series is also uncomfortably high. At least two features of rescue angioplasty will be required before the strategy is universally accepted. First, failed rescue angioplasty should be a rare event. It is possible that the use of coronary artery stents, intraaortic balloon pumping and glycoprotein IIb/IIIa inhibitors will facilitate this. Second, the mortality rate following a successful attempt should be considerably lower than that of patients receiving conservative treatment for a persistently occluded IRA. Ross et al. (1) do not provide convincing evidence that the latter is true.

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Transfer delay for primary PTCA: Does it Influence Clinical Outcome?

In Liem et al.’s (1) article on the effect of transfer delay on infarct size and clinical outcome in patients who are referred for primary percutaneous transluminal coronary angioplasty (PTCA), they compared a cohort of 207 patients transferred from other hospitals with a matched group of 207 patients directly admitted to a PTCA center. The difference between the two cohorts in time delay from onset of symptoms to PTCA was a transfer delay of median 43 min, and time from symptom onset, infarct location, age, gender and Killip class were similar. Transfer delay was associated with a larger infarct size and lower ventricular function, albeit with similar patency rates. Despite these adverse effects on myocardial salvage, clinical outcome after six months has reported to be not adversely affected, i.e., 7% versus 6% mortality and 4% versus 3% nonfatal reinfarction. We object, however, to the authors’ conclusion that “6-month clinical outcome [was] not affected by this [transfer] delay.” Such a conclusion cannot be drawn from this study, because the sample size of 414 patients was too small. For instance, the hypothesis that a mortality difference between transferred and nontransferred patients would not exceed 2% from an expected 6% mortality rate would require more than 3,400 patients in each treatment group, when tested by a two-tailed chi-square test with alpha 0.05 and beta 0.10. The authors would be correct only when a difference in mortality of 10% was accepted as not clinically important, but such a difference is not considered ethically acceptable. Therefore, the adage “time is muscle” still holds for patients considered for primary PTCA, but a larger study should be conducted before conclusions can be drawn about the effects of transfer delay on clinical outcome in patients referred for primary PTCA.

A second item we wish to discuss is the time frame within which patients may still be transferred to a PTCA center. The possible benefit of myocardial salvage will decrease with increasing time, and additional delay by transfer of patients in this stage may not be desirable. It seems from this study that all patients were presented to the hospital within 3 h (Table 1). For this reason, myocardial salvage difference between the transferred and nontransferred was found to be large. It would be interesting if the authors could
provide some data on patients presented between 3 and 6 h following the onset of symptoms.

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REPLY
We thank Kok, Umans, and Arnold for their statistical calculations. Indeed, a large sample size would be required to prove that there is equivalence in clinical outcome between patients directly admitted compared to transferred patients. To demonstrate that the six-month mortality rate would not exceed more than 1% (6% vs. 7%) would require a sample size of even more than 25,000 patients! However, it was not our objective to prove that transferred patients have an identical clinical outcome compared with directly admitted patients. The “primary endpoints” of this analysis were variables such as ejection fraction, enzymatic infarct size and patency rate. For completeness, six-month mortality and reinfarct rates were reported, demonstrating that the delay was indeed not associated with a strong increase of adverse events. However, we emphasize the importance of the patency rate achieved in the referral patients for longterm survival (1,2), and therefore, our data are compatible with equivalence in clinical outcome, albeit without the statistical proof.

The second question regards data on the subgroup of patients admitted between 3 h and 6 h following onset of symptoms. As described in our article, one of the characteristics of referral patients is the short delay between onset of symptoms and admission. This is probably caused by the criteria used by referral cardiologists for the decision to transport the patient for primary angioplasty. In the study group, therefore, 43 patients presented between 3 h to 6 h following onset of symptoms. This subset of patients is too small to be separately evaluated.

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Seasonal Distribution of Myocardial Infarction and Seasonal Mood Changes
I read with interest the article by Spencer et al. on the seasonal distribution of acute myocardial infarction (1). I would like to offer some comments on this article.

Since ancient times, people have known about the seasonal changes in mood and behavior (2). Seasonal changes in mood were later described by Esquirol in 1845 (3) and by Kraepelin in 1921 (4). In 1984, the syndrome of “seasonal affective disorder,” a condition where depressions in fall and winter alternate with nondepressed periods in the spring and summer, was described (5). Sadness, anxiety, irritability, decreased activity, difficulties at work, social withdrawal, changes in appetite, decreased libido and changes in sleep are characteristic symptoms of winter depression. The degree to which seasonal changes influence mood, energy, sleep, appetite, food preference or the wish to socialize with other people has been called “seasonality” (6). Seasonality can manifest itself to a different degree in different individuals. Some people experience only very mild seasonal changes, and others are severely influenced. Surveys have shown that 25% of the general population in New York City and 27% of the general population in Montgomery County, Maryland, noted that seasonal changes were a problem in their lives (6,7). Recent studies have demonstrated that seasonal mood changes are related to the genetic factors (8). It means that people may have genetically determined sensitivity to seasons.

Psychological factors have a very considerable effect on the cardiovascular system (9–12). Many studies have documented increased cardiovascular morbidity and mortality in patients with depressive disorders. Depression has been implicated as an independent risk factor in the pathophysiologic progression of cardiovascular disease. Depression, stress, anger, anxiety and social isolation have been shown to substantially increase risk for myocardial infarction in patients diagnosed with coronary artery disease. Therefore, I suggest that seasonal mood changes may play a part in the observed seasonal differences in the incidence of myocardial infarction.

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