Functional Improvement in Stunned Myocardium

The report by Ito and colleagues1 constitutes an advancement in our capabilities of assessing the progress of functional recovery of postischemic myocardium in patients with acute anterior infarction, particularly because the monitoring of the degrees of residual mechanical impairment was done in the context of the extent of the initial ischemic area at risk. The authors should be commended for combining the information attained by myocardial contrast echocardiography2 and the data from quantitative echocardiographical assessment of regional contraction abnormalities3 in some patients who received thrombolysis and some others who were not given such therapy.

Although the invasive component of their study (intracoronary injection of contrast solution) constitutes an impediment for routine implementation of this monitoring technique to patients with acute anterior myocardial infarction, further research in this field is needed to investigate the possibility that the segment length of abnormal contraction prior to thrombolysis can be regarded as an index of the ischemic area at risk and that the acquisition of myocardial contrast echocardiographical data may not offer additional information. If such proof becomes available, one can dispense with the intracoronary injection of contrast medium, rendering the method clinically applicable.

I have only one criticism with this otherwise excellent study: Because, as the authors stated in “Results,” “the value for AS/CD before coronary reflow was 1.00±0.02, suggesting that lengths of segments showing normal contraction coincide with those of the contrast defect segment,” the essence of such important finding should have been included in the “message” of the article as part of the “clinical implications.” Instead, the authors have included in the last paragraph that the “initial infarct size” is of importance for assessment of therapeutic interventions. If they imply that a measure of such variable is the initial endocardial length of abnormal contraction (dyskinesis/akinesis) segment (AS), serial routine echocardiography may suffice; if they mean that “initial infarct size” is only provided by the initial contrast defect segment (CD) (which requires the intracoronary injection of contrast medium), this contradicts their findings (AS/CD=1.00±0.02).

John E. Madias, MD
Division of Cardiology
Mount Sinai Services
Elmhurst Hospital Center
Elmhurst, New York

References

Reply
We appreciate the comment by Dr Madias concerning the significance of determining the risk area with myocardial contrast echocardiography (MCE) in the acute stage of myocardial infarc-

In Search of the Optimized Excimer Laser Angioplasty System

Our report1 on excimer laser-induced arterial wall damage is accompanied by an Editorial Comment2 by Litvack and colleagues, who base their description of excimer laser ablation on in vitro studies3,4,5 from the early 1980s. For two reasons, those studies have provided a misleading picture of excimer laser coronary angioplasty (ELCA), a picture which in 1993 is persistently presented in brochures from the industry.

First, laser-tissue interaction of the 193-nm argon-fluoride excimer laser6 differs considerably from the fiber-delivered, pulse-stretched 308-nm xenon-chloride excimer laser used at present for ELCA. The latter ablates by a predominantly photothermal mechanism.7 In vivo, it produces a 0.35-mm zone of collateral thermal necrosis.8 In standard histology, such thermal necrosis becomes fully evident only after 1-day survival following the intervention.
In search of the optimized excimer laser angioplasty system.
C Borst, T G van Leeuwen and M J Post

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