
Functional Improvement in Stunned Myocardium

The report by Ito and colleagues constitutes an advantage 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 echocardiography and the data from quantitative echocardiographical assessment of regional contraction abnormalities 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 abnormal 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).

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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 infarction. In our study, we initially aimed to clarify the relation between the size of the risk area and that of the segment showing abnormal (dyskinesis or akinesis) contraction in patients with acute anterior myocardial infarction. We used MCE for the delineation of the risk area. We delineated the risk area as an area showing no contrast enhancement during contrast injection into right or left coronary artery before coronary reflow in the apical long-axis view. However, no contrast enhancement was observed in this echo view during contrast injection into the right coronary artery because of poor development of collateral channels that come from the right coronary artery. Thus, we only used MCE images during contrast injection into the left coronary artery in this study, and our results, indeed, indicate that the lengths of segments showing abnormal contraction before coronary reflow (AS) coincide with those of segments with no contrast perfusion before coronary reflow (CD).

In cases of well-developed collateral channels that come from the right coronary artery, if the risk area were determined only by the injection into the left coronary artery, the lengths of this anatomical risk area would show discordance with those of the abnormal contraction segments (AS). In such cases, the injection into both left and right coronary arteries should be mandatory for the determination of functional risk area.

We believe that the determination of area that is supplied by the occluded coronary artery (“anatomical” risk area) still has another clinical value. “Anatomical” risk area can be delineated in the apical long-axis view during contrast injection only into the left coronary artery in patients with acute anterior infarction. Using this approach, we can assess the beneficial effect of coronary reflow on the salvage of myocardium in the areas supplied by occluded coronary artery.

Therefore, we consider that AS indicates the size of functional risk area, which is not always coincident with the size of “anatomical” risk area. Although functional risk area may be determined by the analysis of wall motion abnormalities, “anatomical” risk area should be obtained only by MCE with contrast injection into the infarct-related artery before coronary reflow.

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In Search of the Optimized Excimer Laser Angioplasty System

Our report on excimer laser-induced arterial wall damage is accompanied by an Editorial Comment by Litvack and colleagues, who base their description of excimer laser ablation on in vitro studies 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 laser 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. In vivo, it produces a 0.35-mm zone of collateral thermal necrosis. In standard histology, such thermal necrosis becomes fully evident only after 1-day survival following the intervention.
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