

CARDIOVASCULAR MEDICINE

Myocardial blood volume and the amount of viable myocardium early after mechanical reperfusion of acute myocardial infarction: prospective study using venous contrast echocardiography

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Background: Myocardial capillary perfusion is a prerequisite of myocellular viability after reperfusion of acute myocardial infarction. It was hypothesised that the magnitude of myocardial capillary perfusion, assessed by transmural signal intensity in venous contrast echocardiography as a corollary of the blood volume of myocardial capillaries, and the amount of viable myocardium, represented by differential levels of contractile function two weeks after reperfusion, are correlated.

Objectives: To evaluate the role of venous contrast echocardiography for the identification of viable myocardium in patients with acute myocardial infarction early after successful mechanical reperfusion.

Methods: 60 patients with a first acute myocardial infarction underwent venous contrast echocardiography several hours after successful mechanical reperfusion (median time interval 190 min.). The relative transmural videointensity (median (25th, 75th percentiles)) of akinetic segments was determined. After two weeks, contractile function was re-evaluated at rest and during dobutamine infusion if segments without functional recovery were present.

Results: Relative videointensity early after reperfusion differed significantly between functional groups after two weeks: normokinesia (88% (77%, 100%)), hypokinesia (74% (54%, 99%)), and akinesia with (61% (48%, 76%)) and without contractile reserve (31% (22%, 46%)). Relative videointensity and contractile function were significantly correlated ($r = -0.67$). The diagnostic accuracy of relative videointensity $> 50\%$ for prediction of contractility of initially akinetic segments at rest or during dobutamine was 82% ($\chi^2 = 76.2$, $p < 0.001$).

Conclusions: Early after successful mechanical reperfusion of acute myocardial infarction, the magnitude of capillary perfusion in the perfusion territory of an infarct related artery is correlated with the amount of viable myocardium. Quantitative venous contrast echocardiography can be used for accurate identification of viable myocardium.

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The aim of mechanical reperfusion in patients with acute myocardial infarction is to restore blood flow through the infarct related artery to limit myocellular necrosis and ultimately preserve myocardial contractile function, which is the most powerful predictor of survival after acute myocardial infarction.¹ The success of reperfusion, however, is indicated by perfusion of myocardial capillaries rather than simply patency of the infarct related artery.²⁻³ Myocardial contrast echocardiography can be used to assess myocardial capillary perfusion.⁴⁻⁵ The signal from the myocardium following an intravenous injection of microbubbles has been shown to be a corollary of the blood volume of myocardial capillaries and thus functional capillary integrity.⁶ Identification of viable myocardium after reperfusion following acute myocardial infarction, through contrast enhancement after intracoronary injection of microbubbles, was, however, limited in most studies by low specificity for the binary prediction of recovery of resting contractile function.⁷⁻⁹ We expected that the more physiological intramyocardial distribution of the contrast agent after venous administration would result in a precise image of differential degrees of myocardial microvascular damage. We therefore hypothesised that measuring the transmural contrast signal from the myocardium would correlate with the extent of myocardial viability and functional recovery after mechanical reperfusion of acute myocardial infarction. Since endocardial necrosis may not be associated with recovery of resting contractile function,¹⁰ we assessed myocardial viability by measuring contractile function during do-

butamine infusion in patients without functional recovery. Furthermore, because reactive hyperaemia may mask the degree of capillary damage immediately after reperfusion,¹¹ we measured contrast intensity several hours following reperfusion when the hyperaemic response had abated.

METHODS

Patient population

This prospective study enrolled patients with first acute myocardial infarction admitted to the hospital within 24 hours after onset of chest pain. Acute myocardial infarction was diagnosed in the presence of two of the following criteria: typical anginal chest pain lasting ≥ 20 minutes; presumed new ST elevation of ≥ 0.1 mV in two or more contiguous limb ECG leads or ≥ 0.2 mV in two or more contiguous precordial leads; and increased creatine kinase to two or more times the upper reference limit with a concomitant rise in creatine kinase MB isoenzyme. Exclusion criteria were inability to give informed consent, cardiogenic shock, and inadequate echocardiographic window for assessment of wall thickening in any segment of the apical four and two chamber views. All patients underwent successful mechanical reperfusion, which was defined as the presence of TIMI (thrombolysis in myocardial infarction) grade 3 flow immediately and two weeks after the intervention.

Study protocol

Two dimensional and venous myocardial contrast echocardiography were performed approximately three hours after

Table 1 Patient characteristics

Age, median years (25th, 75th centiles)	58 (51, 64)
Women, n (%)	9 (15)
Mechanical reperfusion, n (%)	
Stent	46 (77)
Percutaneous transluminal coronary angioplasty	14 (23)
Time from symptom onset to reperfusion, median hours (25th, 75th centiles)	8 (3, 17)
Peak creatine kinase, median IU (25th, 75th centiles)	825 (426, 1410)
Infarct related artery, n (%)	
Left anterior descending coronary artery	29 (48)
Right coronary artery	25 (42)
Left circumflex coronary artery	6 (10)
Multivessel disease, n (%)	36 (60)
Q wave infarction, n (%)	49 (82)
Arterial hypertension, n (%)	34 (57)
Hypercholesterolaemia, n (%)	39 (65)
Diabetes mellitus, n (%)	10 (17)
Smoking, n (%)	39 (65)
β Blocker medication at follow up, n (%)	57 (95)

mechanical reperfusion (median (25th, 75th centiles) 190 minutes (160 minutes, 242 minutes)). At follow up after two weeks, coronary angiography and both echocardiographic studies were repeated. Patients with akinetic segments at follow up underwent dobutamine stress echocardiography. The study protocol was approved by our institutional review board and patients gave written informed consent.

Venous myocardial contrast echocardiography

Three milliliters of Optison (Mallinckrodt Medical Imaging, Dublin, Ireland) was diluted with 27 ml 0.9% sodium chloride. A volumetric pump (Perfusor fm, Braun Melsungen, Melsungen, Germany) was used for intravenous infusion at 200 ml/h and adequate mixing of the solution was maintained by careful shaking of the pump. A phased array echocardiographic system (Sonos 5500, Agilent Technologies, Palo Alto, California, USA) was used. Harmonic (1.8/3.6 MHz) and intermittent imaging (every seventh cardiac cycle, ECG triggered to end systole) were applied.^{12 13} The maximum power output and dynamic range of the system (60 dB) were used and the most linear postprocessing mode (A) was selected. A dual focus was used and placed in the upper and lower third of the imaging sector. Overall gains and time gain compensation were adjusted to minimise myocardial tissue noise but still to allow myocardial contours to be seen. Machine settings were adjusted before each study and kept constant throughout. Imaging was performed using apical four and two chamber views and all images were recorded on S-VHS videotape. Contrast images were digitally processed off line as previously described.⁶ Mean videointensity was measured in each of the

six segments in the apical four and two chamber views. To account for differences in echogeneity between patients, the relative videointensity of a segment was calculated as the ratio between the videointensity of the segment and the brightest normokinetic segment of the same view. This parameter has been recently clinically validated with quantitative radionuclide single photon emission computed tomography data.¹⁴ Variability of relative videointensity of normokinetic segments early after reperfusion was 9% (1%, 13%). Intraobserver and interobserver variability of relative videointensity of segments were 5% (1.5%, 8.5%) and 7.5% (1.5%, 15%), respectively. Quantitative venous myocardial contrast echocardiography was performed by experienced investigators blinded to all clinical, echocardiographic, and angiographic data.

Two dimensional and dobutamine stress echocardiography

Tissue harmonic imaging (2.1/4.2 MHz) was used to assess wall thickening at rest and during dobutamine stress echocardiography (incremental doses of intravenous dobutamine were 5, 10, 20, 30, and 40 g/kg/min at three minute intervals). Single systoles obtained at rest and before each dobutamine increment from the apical four and two chamber views were stored digitally for review in a quad screen format. The entire study was also continuously recorded on S-VHS videotape. Wall thickening was assessed by two experienced readers blinded to clinical, angiographic, and contrast echo data. In cases of disagreement, the wall motion score was resolved by consensus of opinion. Each of the same segments used for videointensity measurements was assigned a score (1 = normokinesia, 2 = hypokinesia, 3 = akinesia, 4 = dyskinesia). Recovery of contractile function of a formerly dyskinetic or akinetic segment was defined as an improvement to at least hypokinesia at rest. Contractile reserve was defined as improvement to at least hypokinesia during dobutamine infusion. Wall motion score index was defined as the mean score of the 12 segments of a patient.

Statistical analysis

Nominal variables are expressed as counts and percentages. Differences between groups were assessed with a two sided χ^2 test. Continuous variables are presented as median (with the 25th and 75th centiles) and, as appropriate, Kruskal-Wallis and Mann-Whitney tests were applied for group comparisons. α Correction using the method of Marcus and colleagues¹⁵ was used. Spearman correlation coefficient (*r*) is given. Sensitivity and specificity of relative videointensities for prediction of functional status at the two week follow up was calculated. Areas under the resulting receiver operator characteristic curves were compared with the Wilcoxon signed rank test. The Youden index (sensitivity + specificity – 100%) was derived to assess the optimal cut off value. Differences between groups

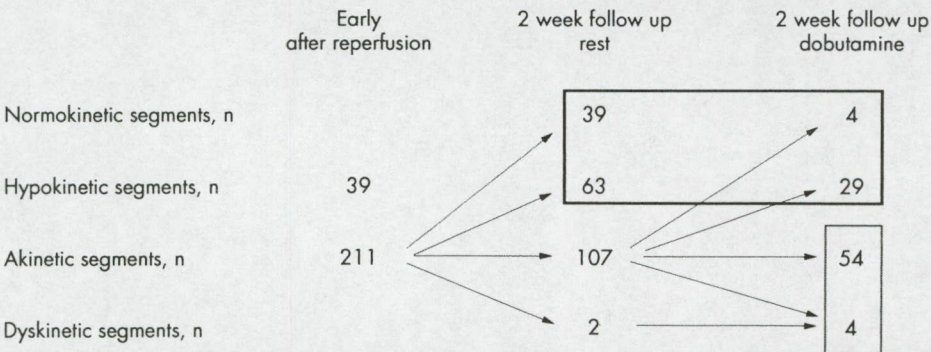


Figure 1 Segmental wall thickening early after reperfusion and after two weeks at rest and during dobutamine infusion. In seven patients (18 akinetic segments at follow up), dobutamine was not infused for clinical reasons. Thick frame: either recovery of contractile function or presence of contractile reserve; thin frame: neither recovery of contractile function nor presence of contractile reserve.

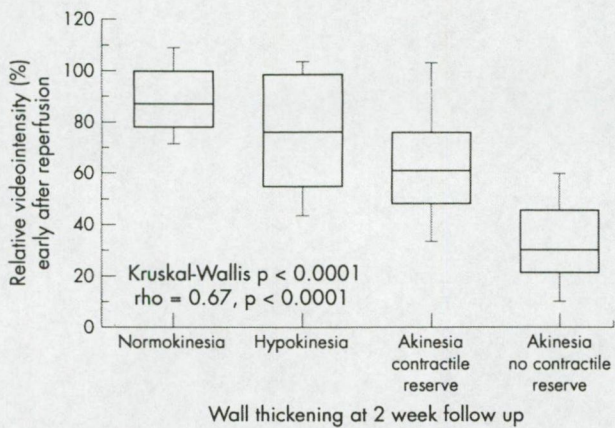


Figure 2 Relative videointensity of akinetic segments early after reperfusion is significantly different between differential functional groups as observed at two weeks' follow up.

were considered significant at $p < 0.05$. SPSS 10.0 (SPSS Inc, Chicago, Illinois, USA) was used as the statistical software package.

RESULTS

Patient characteristics

Seventy four patients were enrolled in the study. In six patients, inadequate image quality prevented quantification of contrast echocardiography. Six patients could not be followed up (one died, three underwent emergency coronary artery bypass grafting, and two refused to participate). The infarct related artery did not show TIMI 3 flow at follow up in two patients. Table 1 lists the demographic, clinical, and angiographic characteristics of the remaining 60 patients. In seven patients, dobutamine stress echocardiography was not performed at two week follow up for clinical reasons (congestive heart failure in two, ventricular tachycardia in one, atrial flutter in one, chest pain in one, and by patient request in two).

Relation between relative videointensity early after reperfusion and differential levels of contractile function at follow up

The vast majority of myocardial segments (211 of 720) in the territory of the infarct related artery were akinetic at the time of postreperfusion echocardiography. Fig 1 summarises

sequential changes in contractile function of initially akinetic segments at rest and during dobutamine at the two week follow up.

We specifically looked at the relation between relative videointensity of initially akinetic segments and contractile function after two weeks. Relative videointensity was higher (83% (66%, 99%)) in segments with functional recovery (normokinesia, $n = 39$; hypokinesia, $n = 63$) than in those that remained akinetic or dyskinetic (relative videointensity 40% (28%, 57%), $p < 0.0001$). In the functional recovery group, relative videointensity was significantly different between segments with recovery to normokinesia and those with recovery to hypokinesia (88% (77%, 100%) v 74% (54%, 99%), respectively, $p < 0.001$; fig 2). In segments without functional recovery, relative videointensity was significantly higher in those with contractile reserve ($n = 33$) than in segments without contractile reserve ($n = 58$; 61% (48%, 76%) v 31% (22%, 46%), respectively, $p < 0.001$; fig 2). Relative videointensity of segments with functional recovery to hypokinesia was significantly higher than that of segments without functional recovery but with a contractile reserve present ($p < 0.001$). Relative videointensity early after reperfusion and contractile function at the two week follow up were significantly correlated ($r = -0.67$).

Prediction of segmental contractile function with contrast echocardiography

Fig 3 depicts examples of differential relative videointensities within akinetic myocardium of patients early after successful mechanical reperfusion of a first acute myocardial infarction. Fig 4A (curve A) depicts the receiver operator characteristic curve for contrast echocardiographic prediction of myocardial segments with recovery of resting contractile function or presence of contractile reserve at the two week follow up. Curve B depicts the receiver operator characteristic curve for prediction of recovery of resting contractile function alone. The curves differ only slightly, albeit significantly ($p < 0.001$). Fig 4B shows the Youden index derived from curve A, which plateaus between 42% and 62%. In light of intraobserver and interobserver variabilities of relative myocardial videointensity of 5% and 7.5%, respectively, a cut off of 50% relative videointensity for prediction of recovery of contractile function or presence of contractile reserve by contrast echocardiography was determined. The power of this criterion for early discrimination between segments with and those without

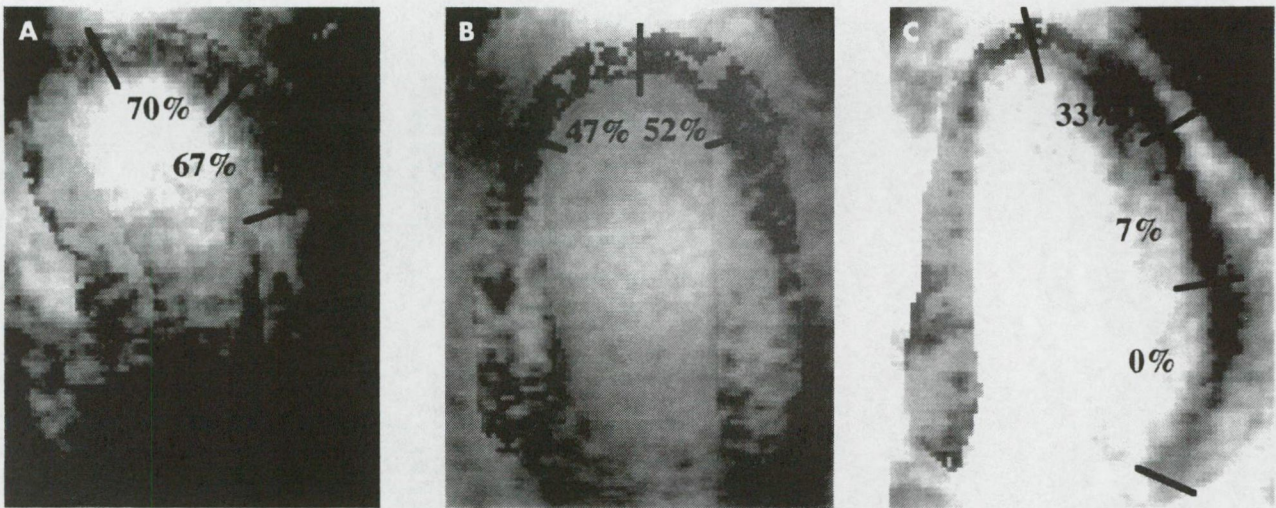


Figure 3 Relative videointensity of akinetic segments of three patients in the two chamber view early after reperfusion (A) with functional recovery at rest at two weeks' follow up, (B) without functional recovery at rest but presence of contractile reserve at two weeks' follow up, and (C) with neither functional recovery at rest nor presence of contractile reserve at two weeks' follow up.

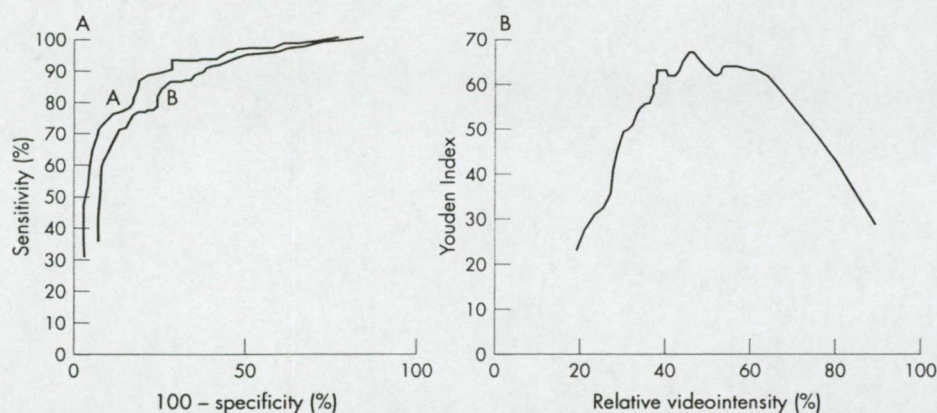


Figure 4 Receiver operator characteristic curves for identification of functional recovery or presence of contractile reserve (curve A) or functional recovery alone (curve B) by contrast echocardiography. (B) The Youden index (sensitivity + specificity - 100) of different relative videointensities for the prediction of functional recovery or presence of contractile reserve at two weeks' follow up.

contractility at rest or during dobutamine infusion at two weeks' follow up is characterised by a $\chi^2 = 76.2$ (df = 1, $p < 0.001$). Diagnostic accuracy was 82% (sensitivity 83%, specificity 81%).

Prediction of recovery of global left ventricular function with contrast echocardiography

We then compared the wall motion score index of two groups of patients early after reperfusion and at the two week follow up: $\geq 50\%$ (group 1, $n = 23$) and $< 50\%$ (group 2, $n = 37$) of initially akinetic segments contrast positive (relative videointensity $\geq 50\%$). Clinical and angiographic characteristics were not different between group 1 and 2 except for the presence of non-Q wave acute myocardial infarction (nine in group 1 v none in group 2, $p < 0.01$). Early after reperfusion, the wall motion score index was not different between group 1 and group 2 (1.5 (1.3, 1.9) v 1.8 (1.3, 2.0), respectively). At follow up, the wall motion score index was significantly lower in group 1 than in group 2 (1.3 (1.1, 1.5) v 1.5 (1.3, 1.8), respectively, $p = 0.002$). Improvement of the wall motion score index between early postreperfusion and follow up was significant only in group 1 ($p < 0.0001$, fig 5).

Relative videointensity early after reperfusion and at two weeks' follow up

No significant difference was found between relative videointensity early after reperfusion (60% (35%, 87%)) and at two weeks' follow up (73% (37%, 91%)).

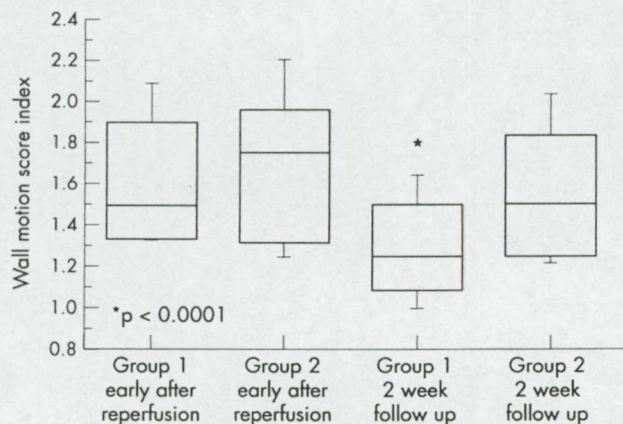


Figure 5 Wall motion score index early after reperfusion and at follow up in patients with $\geq 50\%$ (group 1) and $< 50\%$ (group 2) contrast positive (relative videointensity $\geq 50\%$) segments of initially akinetic myocardium.

DISCUSSION

The present study had two main findings in patients with a first acute myocardial infarction, early after successful mechanical reperfusion once reactive hyperaemia has abated. Firstly, there is a correlation between the blood volume of myocardial capillaries, as assessed by transmural signal in venous contrast echocardiography, and the amount of viable myocardium within the perfusion territory of the infarct related artery. Secondly, quantitative venous contrast echocardiography can be used early after reperfusion to predict accurately not only recovery of contractile function at rest but also the presence of contractile reserve without functional recovery.

Pathophysiology of myocardial perfusion and contractile function early after reperfusion of acute myocardial infarction

Structural capillary damage accompanying myocellular necrosis,¹⁶ complex cellular interactions including release of inflammatory and vasoconstrictive mediators from ischaemic myocardium and blood cells triggering endothelial adhesion and aggregation of neutrophils¹⁷ and platelets,¹⁸ and distal embolisation of platelet aggregates from the infarct causing ruptured atherosclerotic plaque¹⁹ are thought to contribute to myocardial "no reflow" after reperfusion of a previously occluded coronary artery. Measuring reduced blood volume of myocardial capillaries, associated with "no reflow", throughout the thickness of a myocardial segment as in the present study can account for transmural heterogeneity of these processes and therefore can accurately quantify capillary perfusion of a myocardial segment.

The pathophysiology of myocardial contractile function after mechanical reperfusion in acute myocardial infarction is heterogeneous and may, in addition to myocardial stunning, include various amounts of myocardial necrosis or recurrent ischaemia. Early after reperfusion, conventional echocardiographic measures of contractile function cannot be used to differentiate the underlying pathophysiology of akinetic myocardium. Later, once stunning has abated, myocardium with only minor transmural extent of necrosis will show functional recovery. The persistence of akinesia, however, is not synonymous with transmural myocardial necrosis. Evaluation of myocardial contractile function at rest allows evaluation of only the inner (endocardial) third of the myocardium: if there is no or only minor necrosis the myocardium is normokinetic or hypokinetic but subendocardial necrosis results in akinesia.¹⁰ Akinesia, therefore, occurs irrespective of the amount of vital tissue in the mid and epicardial portions of the myocardium. Although these portions contribute little to contraction at rest, they can be recruited to produce contractile reserve of akinetic myocardium during dobutamine

stimulation.²⁰ The four functional classes differentiated at the two week follow up in the present study can therefore be regarded as functional corollaries of differential degrees of transmural myocardial necrosis.

Contractile reserve of akinetic myocardium is clinically relevant because it has been shown to protect against ventricular dilatation and development of heart failure.²¹ Our data indicate that the continuum between myocardial "no reflow" and "reflow", imaged with venous contrast echocardiography and quantified with its transmural signal intensity, gives an estimate of the extent of necrosis in a myocardial segment as soon as three hours after reperfusion. This result is in line with the findings of previous animal studies comparing the extent of myocardial contrast enhancement after venous contrast injection with myocardial necrosis by using triphenyltetrazolium chloride staining.^{22, 23} With respect to the relevance of the spatial extent of contrast enhancement for subsequent recovery of global left ventricular function, our findings confirm the results of Porter and colleagues²⁴ and Lepper and associates.²⁵ These first two studies of the use of venous contrast echocardiography in patients after reperfusion of acute myocardial infarction used a semiquantitative score for the assessment of myocardial contrast enhancement in the risk area 48²⁴ and 24 hours²⁵ after reperfusion.

Prediction of functional recovery or presence of contractile reserve with venous contrast echocardiography early after reperfusion

This is the first study to define a quantitative signal threshold for the prediction of myocardial contractile function in venous contrast echocardiography early after mechanical reperfusion of acute myocardial infarction. Several investigators have attempted to predict recovery of contractile function early after reperfusion treatment in acute myocardial infarction using semiquantitative assessment of myocardial contrast enhancement with intracoronary injection of microbubbles. Depending on the strictness of criteria for the positive contrast effect, sensitivities for prediction of subsequent functional recovery were found between 66%⁷ and 100%.⁸ Specificity, however, was only between 18%⁹ and 60%⁷ in most studies. We found higher diagnostic accuracy of venous contrast echocardiography for prediction of recovery of contractile function or the presence of contractile reserve after acute myocardial infarction. This finding cannot be attributed solely to our consideration of contractile reserve, in distinction to previous studies with intracoronary contrast injection, because our receiver operator curves using functional recovery alone (fig 4A, curve B) or both variables (curve A) differ only slightly.

Several additional factors may contribute to the improved diagnostic accuracy. Firstly, intracoronary bolus injection with a hand held syringe used in previous studies may force contrast material into the dependent microvasculature. Therefore, lack of opacification may be observed only in the myocardium with the most seriously damaged microvessels, whereas it may still be seen in areas with moderate microvascular damage. Venous infusion, in distinction, may result in more physiological myocardial distribution of microbubbles and provide a better representation of differential degrees of microvascular damage. Secondly, the diagnostic accuracy of the prediction of functional recovery depends on the definition of the threshold for positive contrast effect. It is difficult to quantify the magnitude of the contrast effect after poorly standardised intracoronary bolus injection of sonicated radio-opaque dye. Therefore, semiquantitative assessment has been used in most intracoronary studies. However, defining a threshold is less accurate with a framework of only a few scores than with the quantitative approach used in the present study. Thirdly, myocardial contrast enhancement was evaluated 10–16 minutes after successful reperfusion in previous intracoronary studies while reactive hyperaemia might have

been present, likely causing an overestimation of contrast effect within the infarct bed.¹¹ Venous contrast echocardiography was performed later (approximately three hours after reperfusion) in the present study and relative videointensity was even slightly higher after two weeks. It is therefore unlikely that relative videointensity measurements were affected by reactive hyperaemia. Our data therefore favour a time interval of approximately three hours between mechanical reperfusion of acute myocardial infarction and contrast echocardiographic assessment of myocardial viability.

Swinburn and colleagues²⁶ recently reported a sensitivity of 62% of venous contrast echocardiography scores obtained approximately five days after acute myocardial infarction for binary prediction of recovery of resting contractile function of myocardial segments. Apart from differences in methods and the sole consideration of functional recovery at rest, however, undefined coronary anatomy at the time of contrast echocardiography and at follow up may have prevented unequivocal interpretation of the association between an early contrast score and contractile function at follow up in their study.²⁶

Study limitations

Because of anisotropy, heterogeneous beam profile, and attenuation, the contrast signal intensity of differential myocardial segments is subject to variability independent of the volume of myocardial microvasculature (9% (1%, 13%) in normokinetic segments in the present study), which might have prevented a closer correlation between relative myocardial videointensity early after reperfusion and the amount of viable myocardium. A slight divergence of imaging planes between initial and follow up studies may have attenuated this correlation as well. Although imaging planes were carefully chosen, this typical source of inaccuracy of serial echocardiography cannot be completely eliminated.

Advantages of intermittent harmonic imaging, used in the present study, are a broad dynamic range and high signal amplitude caused by microbubble disruption with high mechanical index imaging. On the other hand, off line image processing is mandatory because the tissue component of the harmonic signal is considerable. Recent advances in this technique including multiple pulse transmission, reduction of myocardial tissue signal by modified transmit and receive frequencies, and colour coding of raw image data may, however, obviate the need for time consuming off line processing in the future. Recently developed autocorrelation based imaging technology offers alternative echocardiographic modalities (power Doppler, pulse inversion, or power modulation technique) for venous contrast echocardiography. Image analysis is facilitated by on line background subtraction, and real time venous myocardial contrast echocardiography has become available. The range of myocardial blood volumes detectable with these techniques, characterised by a narrow dynamic range compared with harmonic imaging, however, needs to be defined.

We used a measure of myocardial blood volume to characterise myocardial perfusion. There are first reports that myocardial blood flow velocity assessed by analysis of increasing pulsing intervals may be a more robust parameter for the quantitative analysis of venous myocardial contrast echocardiography.⁶

Diagnostic accuracy found for prediction of contractile myocardium by venous myocardial contrast echocardiography in the present study may be too optimistic since the underlying contrast intensity cut off value was determined in the same patient population.

Although raw signal intensity data were not used in the present study, despite theoretical superiority, the validity of videodensitometric data was shown in a head to head comparison with digital raw data.²⁷

The contractile response to dobutamine could have been attenuated by β blocker treatment, which was not discontinued in the present study. Therefore, although we used high

doses of dobutamine, we may have incorrectly classified patients as having no contractile reserve.

Clinical implications and perspective

Our data imply that myocardial capillary perfusion characterised by venous contrast echocardiography is a sensitive and meaningful marker for the evaluation of the success of reperfusion treatment in patients with acute myocardial infarction. Determination of TIMI myocardial perfusion grade,³ Doppler flow wire,^{25,28} nuclear myocardial perfusion imaging,²⁹ and magnetic resonance imaging³⁰ have been used to assess myocardial microvasculature after acute myocardial infarction. Each of these techniques has its specific strengths and limitations, but none of them has been implemented in routine diagnostic investigation of patients with acute myocardial infarction because they are either invasive or not readily available in most hospitals. Venous myocardial contrast echocardiography used in the present study, in distinction, is a bedside technique and has the potential to be widely available. Furthermore, the application of pharmacological stress, so far needed for echocardiographic assessment of myocardial viability, is not required. In light of increasing numbers of reperfusion strategies for patients with acute coronary syndromes and existing highly effective treatment modalities, a versatile tool for rational and cost effective comparison of efficacy differences between them is important and may guide the planning for large scale clinical mortality trials.

Based on the present data, it also appears reasonable to evaluate the role of venous contrast echocardiography for risk stratification after acute myocardial infarction in future studies.

Analysis of time intensity curves based on signals from incremental imaging intervals or real time registration of signals using low mechanical index imaging has the potential for evaluating myocardial microvascular flow velocity and blood flow.⁶ This may provide further insight in to the process of recovery of myocardial microvascular and contractile function after reperfusion of acute myocardial infarction.

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Az életképes myocardium megítélése közvetlenül az akut myocardialis infarctus reperfúziós terápiája után végzett vénás myocardialis kontrasztultrahanggal

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Bevezetés: Az akut myocardialis infarctus (AMI) reperfúziós terápiáját követően az epicardialis coronaria átjárhatósága mellett ép mikrovasculatura is szükséges a kontraktilis funkció későbbi javulásához. **Célkitűzés:** A szerzők sikeres reperfúziós terápián átesett AMI-os betegeken a microvasculatura állapota és a későbbi kontraktilis funkció közötti összefüggést vizsgálták szemikvantitatív intravénás myocardialis kontrasztultrahanggal (VMKE). **Módszer:** Hatvanhárom első AMI-on átesett betegen végeztek VMKE-t 3 órával a sikeres primer percutan transluminális coronaria-angioplastica (PTCA) után, és szemikvantitatív módon értékelték az akinetikus szegmentumok kontrasztfelvételét (1 = normális, 2 = kissé csökkent, 3 = jelentősen csökkent, 4 = kontrasztfelvétel nem észlelhető). A korábban akinetikus szegmenteket (n = 218) a 14. napon végzett nyugalmi ultrahangvizsgálat alapján különböző funkcionális csoportokba osztották: normokínézis, hipokínézis, akinézis. **Eredmények:** Közvetlenül a reperfúziós terápia után észlelt szemikvantitatív kontraszt score a későbbi három funkcionális csoportban szignifikánsan különbözött ($1,21 \pm 0,47$; $1,65 \pm 0,77$; $2,75 \pm 0,85$; $p < 0,01$). A nyugalmi funkciójavulást 79%-os diagnosztikus pontossággal lehetett VMKE-vel megítélni ($\chi^2 = 76,2$; $p < 0,001$). A globális falmozgáscore-index a kontraszt pozitív betegeknél (a kezdetben akinetikus szegmentek több mint felében 1-es vagy 2-es kontrasztscore) szignifikánsan javult az utánkövetésig ($1,607 \pm 0,30$ vs. $1,295 \pm 0,25$; $p < 0,001$), míg a kontraszt negatív betegeknél nem változott ($1,702 \pm 0,38$ vs. $1,603 \pm 0,33$; $p = \text{NS}$). **Következtetés:** AMI-on átesett betegeknél közvetlenül a reperfúziós terápia után végzett VMKE-gal kielégítő pontossággal lehet megjósolni a későbbi nyugalmi funkciójavulást és következtetni lehet az infarctus transmuralis kiterjedésének mértékére.

Kulcsszavak: myocardialis kontrasztultrahang, myocardialis infarctus, életképes myocardium, mikrocirkuláció

Detection of myocardial viability with venous contrast echocardiography immediately after reperfusion in patients with acute myocardial infarction. **Introduction:** After reperfusion therapy of acute myocardial infarction not only the patency of infarct related artery (IRA) but uncompromised myocardial perfusion are essential for recovery of myocardial contractile function. **Aim:** The authors sought to evaluate the relation between the status of myocardial microvasculature early after successful mechanical reperfusion therapy of AMI and contractile function at rest two weeks later. **Methods:** Sixty-three patients with first acute myocardial infarction underwent venous myocardial contrast echocardiography (VMCE) 3 hours after successful percutaneous coronary intervention. The myocardial contrast intensity of akinetic segments was evaluated according to a semiquantitative score (1 = normal; 2 = moderate contrast defect; 3 = serious contrast defect; 4 = no contrast at all). Two weeks later the resting contractile function of previously akinetic segments (n = 218) was re-evaluated. **Results:** The semiquantitative contrast score was significantly different between differential functional groups observed after two weeks: normokinesia (1.21 ± 0.47); hypokinesia (1.65 ± 0.77); akinesia (2.75 ± 0.85). Sensitivity and the specificity of semiquantitative venous myocardial contrast echocardiography for early prediction of functional recovery is 90% and 69%, respectively ($\chi^2 = 76.2$; $p < 0.001$). The global wall motion score index improved in contrast positive patients (more than 50% of initial akinetic segments show score 1 or 2) (1.607 ± 0.30 vs. 1.295 ± 0.25 ; $p < 0.001$), but did not change in the contrast negative patients (1.702 ± 0.38 vs. 1.603 ± 0.33 ; $p = \text{NS}$). **Conclusion:** Functional recovery after AMI can be predicted with VMCE immediately after successful reopening of IRA. The post-PTCA contrast intensity of an infarcted segment is closely related to its subsequent functional status.

Key words: myocardial contrast echocardiography, myocardial infarction, viable myocardium, microcirculation

Az akut myocardialis infarctus (AMI) korai terápiájában döntő az infarctus területét ellátó artéria (in-

Rövidítések: AMI = akut myocardialis infarctus; CK = kreatin-kináz; IRA = infarct related artery (infarctus területét ellátó artéria; PTCA = perkután transluminális koronáriaangioplastika; TIMI = Thrombolysis In Myocardial Infarction (thrombolysis myocardialis infarctusban); VMKE = vénás myocardialis kontraszt ultrahang; WMS = wall motion score index (falmozgás core index)

farct related artery, IRA) átjárhatóságának mielőbbi biztosítása. A reperfúziós kezelés során elért TIMI 3 (thrombolysis in myocardial infarction) áramlás esetén jobb a későbbi systolés pumpafunkció, mely az AMI betegek mortalitásának döntő meghatározója (14, 23). Ugyanakkor intracoronariásan adott kontrasztanyaggal végzett myocardialis kontraszt-echokardiográfiás vizsgálatokkal (MKE) igazolták,

hogyan az IRA TIMI 3 áramlás mellett az ép myocardialis perfúzió is szükséges feltétele a későbbi systolés kamrafunkció javulásának (9, 10). A TIMI 3 epicardialis coronariaáramlás melletti perfúzióki-
esét nevezük „no-reflow” jelenségnek (11). A fenti megfigyelést számos más és hasonló módszerrel végzett vizsgálat is megerősítette (5, 6, 15, 16, 18, 19).

Célunk közvetlenül a sikeres reperfúziós terápia után vénás myocardialis kontrasztultrahanggal (VMKE), szemikvantitatív módon megítélt microvascularis integritás és a későbbi szegmentális és globális myocardialis systolés funkció közötti összefüggés vizsgálata volt.

Módszer

Betegbeválasztás

Első 12 órán belüli AMI-os betegeket vizsgáltunk. Az időintervallum hosszabb is lehetett (24 óra), amennyiben a betegnek a felvételtől még anginás fájdalma volt. A betegek sikeres revascularizációs terápián (primer PTCA) estek át: TIMI 3 IRA áramlás közvetlen a PTCA után és a 14 napos ismételt koronarográfia során is. Az AMI diagnó-
isa a következő kritériumokon alapult:

- > 30 percig tartó típusos anginás fájdalom;
- felvételtől legalább 1 mm-es ST-eleváció, legalább két összetartozó EKG-elvezetésben;
- a kreatin kináz (CK) normális felső határértékének legalább kétszeresére való emelkedése, megfelelő MB-izoenzim-emelkedéssel a sorozatos enzimvizsgálatok során.

Kizárási kritérium volt:

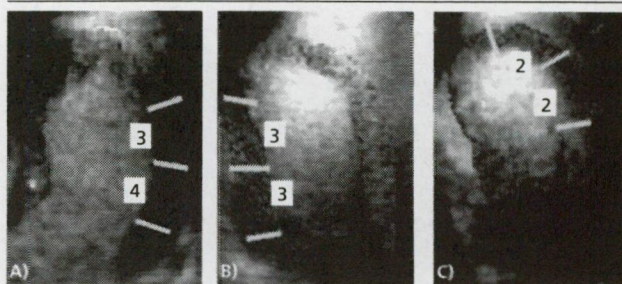
- nem megfelelő minőségű ultrahangvizsgálat kép;
- korábbi myocardialis infarctus az anamnézisben, praemenopausalis életkor;
- a beteg nem volt képes a beleegyező nyilatkozatot aláírni.

Vizsgálati protokoll

A VMKE-vizsgálat közvetlenül a sikeres primer PTCA után történt (1. nap). Kétdimenziós (2D) ultrahangfelvételt végeztünk közvetlenül a VMKE előtt és kéthetes utánkötés után (14. nap). A kéthetes kontrollvizsgálatkor ismételt coronaria-angiográfia történt. A vizsgálati protokollt a Technische Universität, München Etikai Bizottsága jóváhagyta, és minden részt vevő beteg aláírta a felvilágosítást tartalmazó beleegyező nyilatkozatot.

Intravénás myocardialis kontrasztultrahang

3 ml Optisont (Mallinckrodt Medical Imaging, Dublin, Ireland) 27 ml fiziológias sóoldattal hígítottunk és 200 ml/h infúziós sebességgel adagoltuk intravénásan. ATL HDI 5000-es vagy Agilent-Sonos 5500-as ultrahang készülékkel harmonikus módban (1,7/3,4 vagy 1,8/3,6 MHz), intermittáló képalkotást (minden 5. szív ciklusban EKG-triggerrel meghatározott végsystole) alkalmaztunk. Az így nyert csúcsi négy- és kétüregű metszeteket (metszetenként 6 szegmet) S-VHS videokazettára rögzítettük, majd a korábbiakban leírtak szerint off-line digitálisan feldolgoztuk és színekódoltuk (2, 24). A kapott színekódolt képeken az



1. ábra: (A) négyüregű kép, laterális AMI, a „sikeres” primer PTCA ellenére a laterális basális és középső szegmentekben jelentős kontraszthiány, az utánkötés során nincs funkcionális javulás. (B) kétüregű kép, inferior infarctus, az inferior basális, középső szegmentekben súlyos kontrasztkiesés, funkcionális javulás nincs. (C) kétüregű kép, anterior AMI, az anterior apicalis, középső szegmetben csak kissé károsodott a kontrasztfelvétel, az utánkötés során a két szegmet már normokinetikus volt

akinetikus szegmentek kontraszttelítettségét szemikvantitatív skála szerint osztályoztuk: 1 = normális, 2 = kissé csökkent, 3 = jelentősen csökkent, 4 = kontrasztfelvétel nem észlelhető (1. ábra). A szemikvantitatív kontrasztscore prediktív funkciójának vizsgálatok az 1-es és a 2-es kontrasztscore-ral rendelkező szegmenteket kontraszt pozitív, a 3 és 4 kontrasztscore-értékkel rendelkező szegmenteket kontraszt negatív szegmenteknek tekintettük. A betegenkénti értékelésnél kontraszt pozitívnak tekintettünk egy beteget, ha az akinetikus szegmentek legalább 50%-a kontraszt pozitív volt, ha ennél kevesebb, a beteg kontraszt negatív volt.

A felvételeket két, a szegmentális systolés funkciót nem ismerő, kontrasztultrahangban jártas vizsgáló (min. 100 korábbi vizsgálat) értékelte, eltérés esetén konszenzussal döntöttek az adott szegmet szemikvantitatív kontrasztscore-értékéről.

Kétdimenziós echokardiográfia

Felvételkor és az utánkötés során folyamatos harmonikus módot (2,1/4,2 MHz) használtuk a szegmentális falmozgászavar megítélésére. A falmozgást a fent említett négy- és kétüregű képeken elemeztük 12 szegmentes modellen. Az egyes szegmenteket szemikvantitatív módon osztályoztuk: 1 = normális, 2 = hipokinézis, 3 = akinézis. A globális systolés funkciót a falmozgásscindexszel (WMSI) jellemeztük (a 12 szegmet szemikvantitatív score-jának összege osztva 12-vel). A nyugalmi 2 és 4 üregű felvételeket folyamatosan videomagnóra rögzítettük. A felvételeket két független, a kontrasztfelvételeket nem ismerő vizsgáló értékelte, és amennyiben egy szegmet megítélésében eltérés volt, konszenzussal döntöttek. Funkcionális javulásként értékeltük, ha egy korábban akinetikus szegmentnél a 14. napos nyugalmi 2D felvételen hipokinézist vagy normokinézist észleltünk.

Statisztikai analízis

A nominális változókat tartalmazó csoportok közötti különbséget khi-négyzet (χ^2) teszttel vizsgáltuk. A számszerű változók átlagértékét adtuk meg standard deviációval. A csoportok összehasonlítására Kruskal-Wallis és Mann-Whitney-tesztet alkalmaztunk. A csoportok közötti különbséget szignifikánsnak tekintettük, amennyiben $p < 0,05$ volt.

1. táblázat: A vizsgált betegek klinikai és angiográfiás jellemzői

Életkor, medián [25., 75. percentil]	58 [51, 63,5]
Nők, (%)	9 (14)
Mechanikus reperfúzió, (%)	63 (100)
Stent, (%)	48 (76)
PTCA, (%)	15 (24)
Fájdalom kezdetétől a reperfúzióig eltelt idő (óra), medián [25., 75. percentil]	7,7 [3-17]
CK-maximum, medián (IU/l) [25., 75. percentil]	825 [426-1410]
IRA	
LAD, (%)	31 (49)
RCA, (%)	26 (41)
LCX, (%)	6 (10)
Többér betegség, (%)	36 (57)
Q-infarctus, (%)	51 (81)
Hypertonia, n (%)	34 (54)
Hypercholesterinaemia, (%)	39 (62)
Diabetes mellitus, (%)	10 (16)
Dohányos, (%)	39 (62)

PTCA = percutan transluminalis coronaria-angioplastica; CK = kreatinínáz; IRA = infarctus területét ellátó artéria; LAD = ramus descendens anterior; RCA = coronaria dextra; LCX = ramus circumflexus

Eredmények

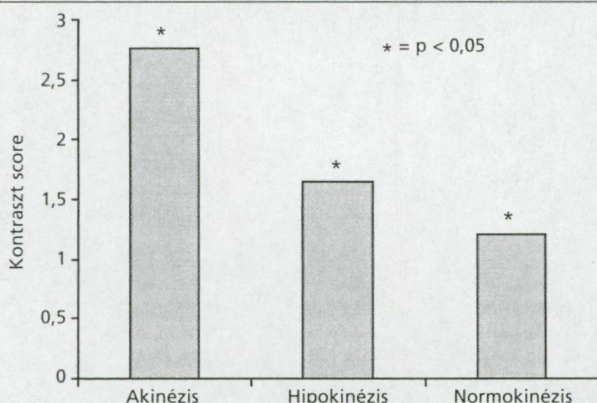
Betegek

A bevételi kritériumoknak 77 beteg felelt meg, akik közül 14 kizárásra került: 2 beteg az utánkövetési idő alatt meghalt, 1 beteg ismételt infarctust szenvedett, 4 betegnél az utánkövetés során TIMI 0-2 IRA áramlást észleltünk, 5 betegnél értékelésre alkalmatlan volt az ultrahangkép minősége, 2 betegnél nem történt meg a kontroll koronarográfia és így az IRA TIMI áramlás a 14. napon ismeretlen volt.

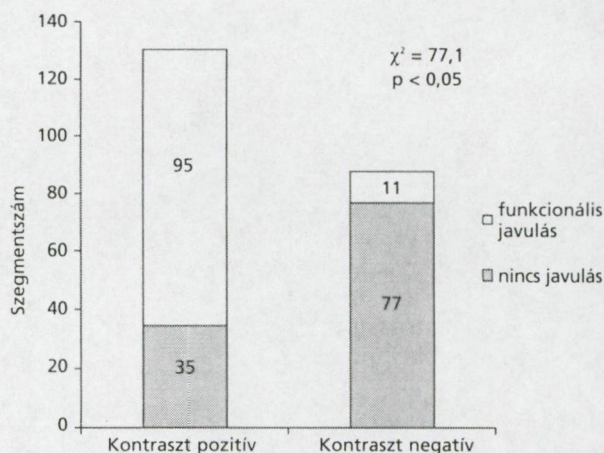
A vizsgálatban részt vevő 63 beteg klinikai és angiográfiás jellemzői az 1. táblázatban láthatók.

A szegmentális funkció értékelése

A vizsgált 756 szegmentből 218 volt akinetikus az 1. napon. Ezen 218 eredetileg akinetikus szegment későbbi systolés funkcióját vizsgáltuk a 14. napon: 106 szegmentnél észleltünk nyugalmi javulást (40 normokinézis, 66 hipokinézis), 112 szegment akinetikus maradt. Ezen csoportokban közvetlenül a pri-



2. ábra: Szemikvantitatív kontrasztscore-értékek a különböző funkcionális csoportokban

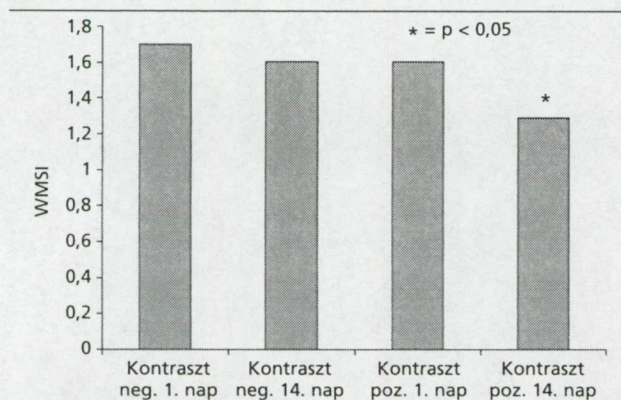


3. ábra: A késői szegmentális funkcionális javulás és a korai kontrasztfelvétel összefüggése

mer PTCA után észlelt szemikvantitatív kontrasztscore különböző volt: normokinézis: $1,205 (\pm 0,47)$, hipokinézis: $1,651 (\pm 0,77)$, akinézis: $2,75 (\pm 0,85)$, ($p < 0,05$) (2. ábra). A 130 kontraszt pozitív szegmentből 95 mutatott későbbi nyugalmi funkcionális javulást, míg 88 kontraszt negatív szegmentből csak 11. A fentiek alapján a szegmentális nyugalmi funkciójavulás előrejelzésében a közvetlenül a sikeres revascularisatio után végzett szemikvantitatív kontrasztultrahang szenzitivitása 90%, specificitása 69%, pozitív prediktív értéke 73%, negatív prediktív értéke 88%, diagnosztikus pontossága 79% ($\chi^2 = 77,1$; $p < 0,001$) volt (3. ábra).

A globális funkció értékelése

A 40 kontraszt pozitív és 23 kontraszt negatív betegnél az 1. napon nem volt szignifikáns különbség a WMSI-ben ($1,607 \pm 0,30$ vs. $1,702 \pm 0,38$). A 14. napon azonban a kontraszt pozitív betegeknél a WMSI kisebb volt, mint a kontraszt negatívoknál ($1,295 \pm 0,25$ vs. $1,603 \pm 0,33$; $p < 0,01$). Ugyanakkor a WMSI kontraszt pozitív betegeknél az utánkö-



4. ábra: A falmozgáscscore-index (WMSI) változása kontraszt pozitív és kontraszt negatív betegekben

vetésig szignifikánsan javult ellentétben kontraszt negatív betegekkel (4. ábra).

Megbeszélés

Főbb megállapításaink a következők:

- a közvetlenül az AMI reperfüziós terápiája után szemikvantitativ módon megbecsült myocardialis vérvolumen és az életképes myocardium mennyisége arányos egymással,
- a reperfüziós terápia utáni korai időszakban megfelelő pontossággal jósolható meg a későbbi nyugalmi systolés funkciójavulás.

Az életképes myocardium mennyiségének megbecslése

Az optimális eredményű (TIMI 3 IRA áramlás) reperfüziós terápia után észlelt systolés funkciózavar (akinezis) patofiziológiai okai eltérőek lehetnek: a különböző kiterjedésű necrosistól (no-reflow) a myocardialis stunningig. A fenti különböző prognózisú okok konvencionális ultrahangvizsgálattal az akut szakban nem különböztethetők meg egymástól. Az általunk bemutatott példákon jól elkülöníthető a definitív necrosis a stunningtól (1. ábra).

Az epicardialis coronaria sikeres revascularisatiója esetén a stunned myocardium nyugalmi systolés funkciója az esetek többségében 2 héten belül megjavul (8). Állatkísérletekben 20–30%-os, vagy annál nagyobb kiterjedésű subendocardialis necrosis esetén az érintett területet akinetikusnak találták, a kisebb kiterjedésű necrosis esetén az elhalt subendocardium arányától függően hipo- vagy normokinézist észleltek (12, 20). Az általunk alkalmazott módszerrel a fenti csoportokban szignifikánsan különböző kontrasztintenzitást észleltünk (2. ábra). A kontrasztintenzitás mértéke a megfelelően hosszú triggerintervallum esetén (például minden 5. végsystole) a myocardialis vérvolumen mennyiségével (myocardial blood volume, MBV) arányos (24). Ez arra utal, hogy közvetlenül a sikeres reperfüziós terápia után az MBV mennyisége fordítottan arányos a necrosis kiterjedésével.

A nyugalmi systolés funkciójavulás előrejezése (korábbi tanulmányok)

A korábban intracoronarián adott kontrasztal végzett tanulmányok hasonlóan jó szenzitivitással (66–100%), de lényegesen gyengébb specificitással (18–60%) becsülték meg az utánkövetés során észlelt nyugalmi funkciójavulást (3, 4, 13). Csak egy munkacsoport talált 100%-os szenzitivitást és 90%-os specificitást, nem minden esetben optimálisan revascularizált és nem olyan homogén betegcsoporton, mint a fenti munkacsoportok vagy az általunk vizsgált csoportok (1). Az általunk elért jobb specificitásnak több oka is lehet: 1. Az intracoronarián, bolusban adott kontraszt myocardialis mennyisége függ a bolus nehezen standardizálható sebességétől, és az egyszerre beadott nagyobb kontrasztmennyiség valószínűleg csak a súlyosan károsodott microvasculatura esetén mutatott defektust. Alacsonyabb kontrasztkoncentráció-tartományban a koncentráció-mért videointenzitás görbe meredekebb, mint az intracoronariás adagolásánál elért magasabb myocardialis koncentráció-tartományban (21). Az intravénásan folyamatos infúzióban adott kontrasztanyaggal egyenletes, de alacsonyabb myocardialis kontrasztkoncentrációt érünk el, és ebben a tartományban létrejövő kontrasztkülönbségeket érzékenyebben tükrözi a videointenzitás változása. 2. A korábbi tanulmányokban a vizsgálat jellegéből fakadóan a kontrasztultrahang közvetlenül a PTCA után (10–25 perc), még a katéteres laboratóriumban történt. Mi a vizsgálatot már az intenzív osztályon, a PTCA-t követő 180. ($\pm$ 24) percben végeztük. Így minden bizonnyal az állatkísérletekben észlelt (bár humán tanulmányokban eddig nem igazolt), a reperfüziós terápiát követően 2 óráig tartó reaktív hyperaemia (7, 22) nem befolyásolta vizsgálati eredményeinket, kevesebb álpozitív esetet találtunk, ami ugyancsak magyarázhatja az elért jobb specificitást.

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További részletek márkakereskedésünkben. *Induló ár: 1.2 motor, 3-ajtós modell. Akció: április 15 – augusztus 31. A képen látható modell felszereltsége eltér a Viva Plusz modelltől. Két doboz hűtött alkoholfmentes Buckler sörrel ajándékozunk meg a 2002. augusztus 1. és szeptember 30. között átadásra kerülő modellek tulajdonosait. Fogyasztás (vegyes): 6,2 l/100km, CO₂: 149 g/km.

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be related to streptococci, such as polyarteritis nodosa and Henoch-Schönlein purpura, are encountered in FMF more commonly than in the general population.² In addition acute poststreptococcal glomerulonephritis has been characterised as a type of renal involvement,³ and RHD has been reported⁴ to be more frequent in patients with FMF than in the normal population. Moreover, increased antistreptolysin O titres have been reported in FMF patients.⁴ This is the proof of greater antibody response against streptococci in patients with FMF. Exaggerated antistreptococcal antibody response is also a characteristic feature of RF. Although RF can follow any group A β haemolytic streptococcus pharyngitis, it was demonstrated that children who developed RF had an exaggerated antibody response to streptococcal antigens when they were compared with children who had recovered from a streptococcal upper respiratory tract infection.⁵

Increased RF or RHD incidence in relatives of our patients strongly supports previous evidence suggesting a familial predisposition in RF. There is a strong consensus that RF represents an abnormal host immune response in genetically predisposed individuals to those streptococcal antigens cross reactive with heart, brain, and joint tissues.¹ Considering the basic defect of FMF, which we propose to be the impaired control of the immune response, we speculate that even bearing only one MEFV mutation might directly or indirectly

result in immune hyperreactivity against streptococcal antigens that leads to development of RF. Although a predisposition to RF is more likely to be multifactorial in origin, or may be polygenic, this is the first study implicating the possible role of the MEFV gene in the pathogenesis of RF.

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IMAGES IN CARDIOLOGY.....

Left ventricular pseudoaneurysm: a mechanical complication of acute myocardial infarction

A 51 year old man was admitted with exertional dyspnoea and a grade 1 to-and-fro heart murmur. He had undergone coronary artery bypass graft surgery for severe three vessel coronary artery disease nine months before. Three weeks before admission, he suffered an acute Q wave lateral myocardial infarction which was treated conservatively. Chest x ray on admission showed a pronounced increase of the cardiac silhouette and signs of pulmonary congestion. Echocardiography was performed and revealed a large echo-free space adjacent to the lateral wall of the left ventricle (below left, asterisk) which extended from the apex

to the mitral annulus (LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle). A small myocardial rupture (below centre) was identified in the basal lateral left ventricular wall, and colour Doppler imaging showed bidirectional shunting of blood between the left ventricle and the echo-free space (below right), enabling a diagnosis of left ventricular pseudoaneurysm to be made. Moderate mitral regurgitation was also present.

During subsequent heart surgery, the pseudoaneurysm was incised, its fibrous wall was resected, and the ruptured left ventricular myocardium was sutured. The mitral valve

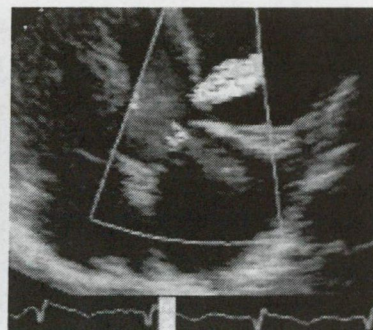
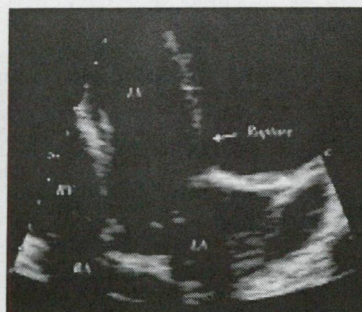
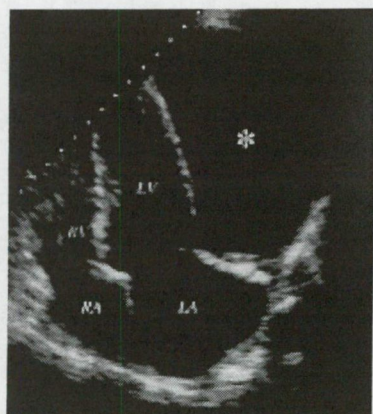
needed to be replaced with a mechanical prosthesis. The vein graft to the left circumflex coronary artery was occluded, but because of the small size and distal occlusion of the native vessel, no redo bypass surgery was performed. The patient's postoperative hospital course was uneventful and he was discharged two weeks later.

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Penetrating Atherosclerotic Ulcer of the Aortic Arch

Christian Firschke, MD; Marek Orban, MD; Peter Andrásy, MD;
Rüdiger Lange, MD; Albert Schömig, MD

A 62-year-old man was referred to our hospital with a 2-day history of moderate continuous interscapular pain. The patient had a history of arterial hypertension and coronary artery disease treated with bypass surgery in 1990. His medication consisted of aspirin, a β -blocker, and an ACE inhibitor. His physical examination was unremarkable except for a blood pressure of 150/95 mm Hg. Acute coronary syndrome was ruled out. A contrast-enhanced CT scan showed a narrow-necked, contrast-filled outpouching of the aortic arch (Figure 1, *) embedded in a mass of lower contrast uptake (Figure 1, arrows). The dimensions of the ascending and descending aorta were normal. On transesophageal echocardiography, marked atherosclerosis of the descending aorta was seen. The anterior wall of the aortic arch could not be delineated unequivocally. An ulcerlike crater (Figure 2, *) with a diameter of $\approx 2 \times 2.5$ cm was part of the anterior margin of the aortic arch (Movie I) and was surrounded by material of varying echolucence with color Doppler signals outside the lumen

of the aorta (Figure 3, arrows; Movie II). On the basis of these findings, perforation of the aortic arch with periaortic hematoma was assumed and was suspected to have been caused by atherosclerotic degeneration of the aortic wall. Urgent surgery was performed, and intraoperatively, extensive atherosclerotic ulceration was found in the aortic arch. The anterior part of the aortic wall in that region was virtually nonexistent, and a functional lumen had been fashioned by semiliquid thrombotic material surrounding the aorta, held together by numerous adhesions. The aortic arch was replaced with a 30-mm Hemashield prosthesis, and the supra-aortic vessels were reimplanted. The patient's postoperative course was uneventful, and he was discharged after 10 days.

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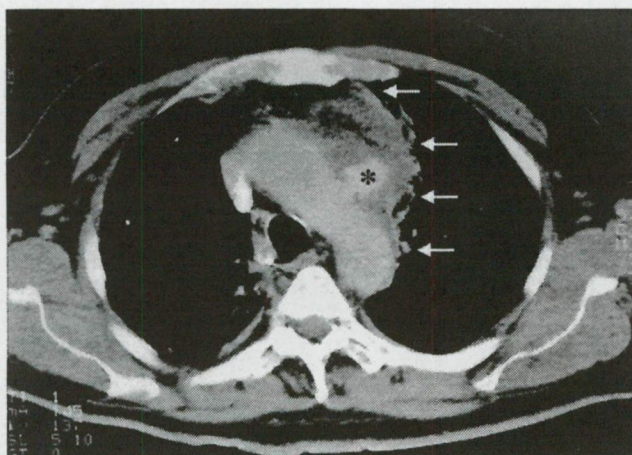


Figure 1. Contrast-enhanced CT scan of the aortic arch.



Figure 2. Transesophageal echocardiogram of the aortic arch.

From Deutsches Herzzentrum, Technische Universität München, Germany.

Movies are available in the online-only Data Supplement at <http://www.circulationaha.org>.

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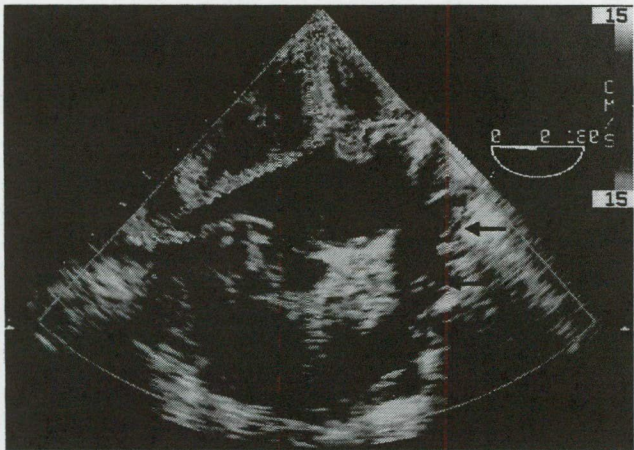


Figure 3. Color Doppler on transesophageal echocardiography (same image plane as in Figure 2).