STATE-OF-THE-ART REVIEW ARTICLE

Expert Review on the Prognostic Role of Echocardiography after Acute Myocardial Infarction

Maria Prastaro, MD, Elisabetta Pirozzi, MD, Nicola Gaibazzi, MD, Stefania Paolillo, MD, Ciro Santoro, MD, Gianluigi Savarese, MD, Maria Angela Losi, MD, Giovanni Esposito, MD, FESC, Pasquale Perrone Filardi, MD, FESC, Bruno Trimarco, MD, FESC, and Maurizio Galderisi, MD, FESC, *Naples and Parma, Italy*

Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide, placing a major economic and resource burden on public health systems. During hospitalization, all AMI patients should be evaluated with transthoracic echocardiography, a noninvasive, low-cost, and easily available bedside imaging tool that allows the detection of myocardial walls involved in the ischemic process, damage extent, functional consequences, and mechanical complications. Moreover, and more importantly, transthoracic echocardiography can provide information on short- and long-term outcomes after AMI. The purpose of this review is to clarify the role of standard and advanced echocardiographic parameters for an early identification of patients at high risk for developing adverse events and mortality after AMI. Standard echocardiography (in particular left ventricular ejection fraction, wall motion score index, and diastolic measurements including E velocity deceleration time and E/e′ ratio) proposes powerful parameters for risk stratification after AMI. Advanced echocardiographic technologies, in particular speckle-tracking–derived longitudinal strain, coronary flow velocity reserve, and myocardial contrast echocardiography (contrast defect index), can provide additional prognostic value beyond standard techniques. Therefore, echocardiography plays a fundamental role in predicting short- and long-term prognosis, and a more accurate risk stratification of patients may be useful to drive therapy and follow-up after AMI. Accordingly, a comprehensive echocardiography-based algorithm would be welcome for an early stratification of cardiovascular risk in patients experiencing AMI. (J Am Soc Echocardiogr 2017;30:431-43.)

Keywords: Acute myocardial infarction, Echocardiography, Doppler, Speckle-tracking echocardiography, Myocardial contrast agents, Real-time three-dimensional echocardiography

The use of transthoracic echocardiography in the coronary care unit has several advantages including easy availability and applicability at bedside, low cost, fast performance, and safety. In case of suspected acute myocardial infarction (AMI), echocardiography can identify regional wall motion abnormalities (WMAs), suggestive of myocardial ischemia or necrosis, and rule out alternative pathologies associated with chest pain such as acute aortic dissection, pericardial effusion, aortic valve stenosis, hypertrophic cardiomyopathy, or right ventricular (RV) dilation due to pulmonary embolism. Echocardiography also has an immediate impact on therapeutic decision making. In fact, it identifies patients with post-AMI mechanical complications who require life-saving surgical intervention. Diagnostic accuracy of standard echocardiography may be further improved by the application of advanced ultrasound technologies such as speckle-tracking echocardiography (STE), myocardial contrast echocardiography (MCE), and real-time three-dimensional (3D) echocardiography (RT3DE).[1] STE may even predict acute coronary occlusion in patients with non-ST-elevation myocardial infarction (NSTEMI) who do not present major electrocardiogram (ECG) abnormalities but urgently need revascularization.[2] Both American and European guidelines that deal with the management of NSTEMI and ST-elevation myocardial infarction (STEMI) recommend echocardiography as the elective cardiac imaging modality for identifying patients at high risk for adverse short-term (during hospitalization) and long-term (after hospital discharge) cardiovascular events, in particular reinfarction and death.[3-6] Accordingly, echocardiographic assessment of infarct size and left ventricular (LV) function at rest should be performed before discharge. Repeated assessment of LV ejection fraction (LVEF), also soon after discharge, is also needed in patients with life-threatening arrhythmias to select appropriate candidates for cardioverter defibrillator. In post-AMI patients (4–6 weeks after the acute event), pharmacological stress echocardiography (SE) is also considered as an alternative to myocardial perfusion scintigraphy to detect inducible myocardial ischemia and viability, two factors that further stratify prognosis and address management in this clinical setting.

From the Department of Advanced Biomedical Sciences, Division of Cardiology, "Federico II" University, Naples (M.P., E.P., S.P., C.S., G.S., M.A.L., G.E., P.P.F., B.T., M.G.); and Parma University Hospital, Parma (N.G.), Italy.

The first two authors should be considered the same in author order.

Reprint requests: Maurizio Galderisi, MD, FESC, Interdepartmental Laboratory of Cardiac Imaging, Federico II University Hospital, Via Pansini 5, Naples 80131, Italy (E-mail: *mgalderi@unina.it*).

0894-7317/$36.00

http://dx.doi.org/10.1016/j.echo.2017.01.020

Abbreviations

2D = Two-dimensional

3D = Three-dimensional

ACEi = Angiotensin-converting enzyme inhibitors

AF = Atrial fibrillation

AMI = Acute myocardial infarction

CDI = Contrast defect index

CFVR = Coronary flow velocity reserve

COSTAMI = Cost of Strategies after Myocardial Infarction

CTDI = Color tissue Doppler imaging

CV = Cardiovascular

DT = Deceleration time

ECG = Electrocardiogram

EDV = End-diastolic volume

EF = Ejection fraction

ESV = End-systolic volume

GLS = Global longitudinal strain

GLSr = Global longitudinal strain rate

HF = Heart failure

HR = Hazard ratio

ICD = Implantable cardioverter defibrillator

LA = Left atrial, atrium

LAVi = Left atrial volume index

LS = Longitudinal strain

LV = Left ventricular, ventricle

LVEF = Left ventricular ejection fraction

MAPSE = Mitral annular plane systolic excursion

MCE = Myocardial contrast echocardiography

MR = Mitral regurgitation

NSTEMI = Non-ST-elevation myocardial infarction

OR = Odds ratio

PASP = Pulmonary artery systolic pressure

PCI = Percutaneous coronary intervention

RFP = Restrictive filling pattern

RT3DE = Real-time three dimensional echocardiography

RV = Right ventricular, ventricle

RVFAC = Right ventricular fractional area change

RWT = Relative wall thickness

SCD = Sudden cardiac death

SE = Stress echocardiography

STE = Speckle-tracking echocardiography

STEMI = ST-elevation myocardial infarction

TAPSE = Tricuspid annular plane systolic excursion

TDI = Tissue Doppler imaging

WMA = Wall motion abnormality

WMSI = Wall motion score index

The prognostic value of several echo measurements after AMI has been documented. They include mainly two-dimensional (2D) and Doppler ultrasound but also advanced echocardiographic techniques. Nevertheless, their importance has not been completely underlined. In this review, we highlight the role and potentialities of both standard and advanced echocardiographic techniques in identifying high-risk patients and also propose a final comprehensive echo exam that is able to stratify the prognosis early after AMI (within 2 weeks after AMI).

STANDARD ECHOCARDIOGRAPHIC TECHNOLOGIES

Motion-Mode

Motion mode (M-mode) provides high temporal and spatial resolution of tissue motion along a single ultrasound beam. The assessment of LV mass and geometry is one of the most important clinical applications of M-mode. The prognostic role of LV mass has been shown after AMI. In 603 patients with AMI complicated by LV systolic dysfunction, heart failure (HF), or both, each 10-g/m^2 increase in LV mass index and each 0.1-unit increase in relative wall thickness (RWT) were independently associated with all-cause mortality, cardiovascular (CV) death, and HF hospitalization. Among LV geometric patterns, concentric LV hypertrophy showed a stronger association with CV mortality, HF, reinfarction, stroke, and sudden cardiac death (SCD).[7] M-mode can also provide information on LV longitudinal systolic function through the quantitation of atrioventricular plane displacement, since during cardiac systole, the atrioventricular plane moves towards the apex. In 271 patients with AMI, reduced mitral annular plane systolic excursion (MAPSE) was independently associated with all-cause mortality, HF hospitalization, reinfarction, and unstable angina; the incidence of death was 31.3% in patients with MAPSE <8 mm and 10.1% in those with MAPSE >8 mm.[8] In addition, tricuspid annular plane systolic excursion (TAPSE) was an independent predictor of mortality after AMI, with the incidence of death being 4% in the presence of TAPSE ≥20 mm, 9% with TAPSE = 16–19 mm, and 45% with TAPSE ≤15 mm.[9] However, M-mode has intrinsic limitations for assessing AMI patients. Mainly, the analysis of regional WMA is limited to only two walls (anterior septum and posterior wall), and no information is obtainable on evidence and magnitude of LV global systolic dysfunction.[10] Accordingly, M-mode echo is only an adjunct to other echocardiographic techniques after AMI.

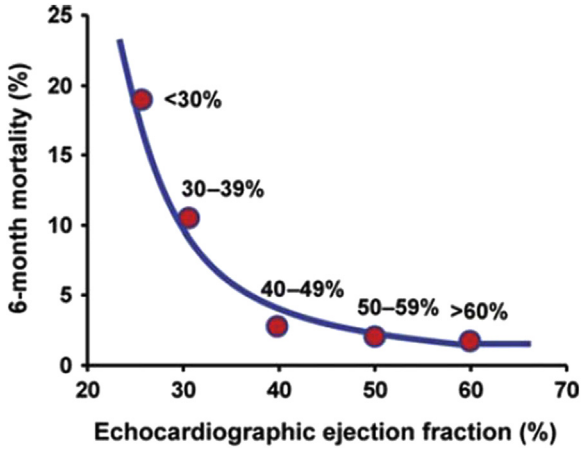

Figure 1 Hyperbolic inverse relation between LVEF and 6-month all-cause mortality after AMI. EF values drive patient management, with those > 40% being in the ischemia domain and those < 35% in the viability domain. (Modified from Volpi *et al.*[13]).

Two-Dimensional Echocardiography

The assessment of cardiac chamber size and function by 2D echocardiography provides very important prognostic information after AMI. The most used parameter is LVEF, which is obtained as the percent ratio between stroke volume and end-diastolic volume (EDV). LVEF has a very well established short-term[11] and long-term[12] prognostic value in this clinical setting. Worthy of note, the LVEF mortality curve after AMI exhibits a typical hyperbolic increasing with an upturn in mortality occurring at values < 40% (Figure 1).[13] In 417 patients with AMI, an LVEF < 40% was an independent predictor of the combined endpoint of death, congestive HF, and recurrent AMI (odds ratio [OR] = 3.82;

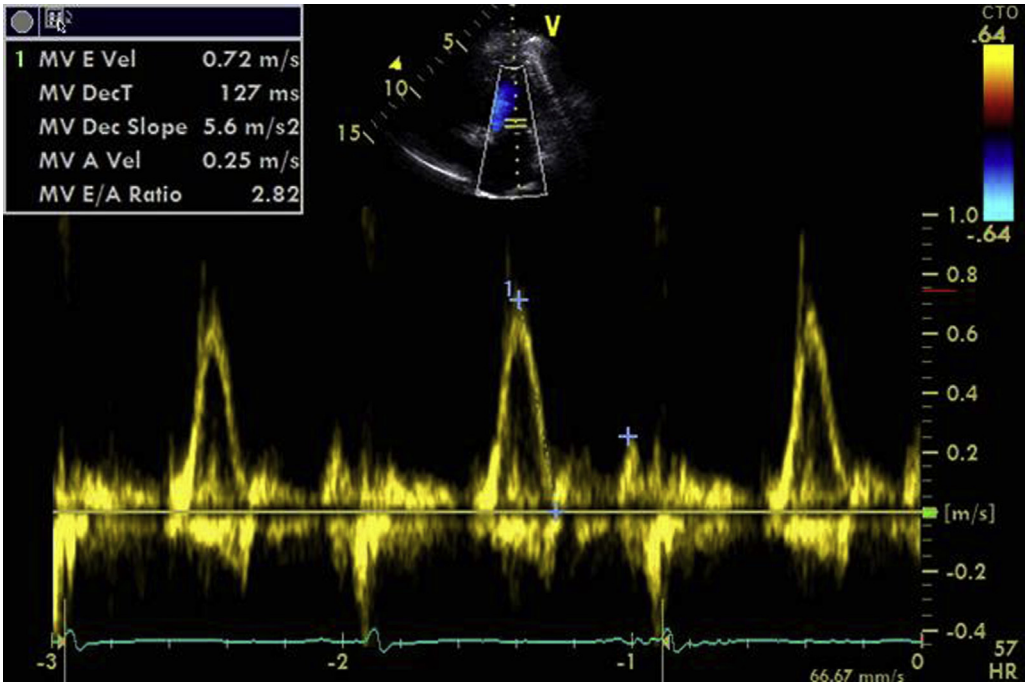

Figure 2 Sample of transmitral RFP in a patient after anterior AMI. Transmitral E/A ratio is 2.82, and E velocity DT is 127 msec. *MV*, mitral valve.

95% CI, 2.15–6.87) at 30 days after AMI.[11] In a large prospective cohort study,[12] 4,122 patients with AMI treated with primary percutaneous coronary intervention (PCI) underwent assessment of LVEF by invasive ventriculography, echocardiography, or radionuclide ventriculography before hospital discharge and were followed up for about 4 years. Patients with LVEF ≤30% and LVEF between >30% and ≤40% had an increased risk for SCD (hazard ratio [HR] = 5.99; 95% CI, 2.73–13.14; $P < .001$; HR = 3.37; 95% CI, 1.74–6.50; $P < .001$, respectively) and all-cause mortality (HR = 3.85; 95% CI, 2.96–5.00; $P < .001$; HR = 2.06; 95% CI, 1.66–2.57; $P < .001$, respectively) compared with patients with LVEF > 40%. Also, LV internal chamber volumes have a recognized prognostic value. In the GISSI-3 trial, predischarge LV EDV and end-systolic volume (ESV) predicted the incidence of the composite endpoint of 6-month mortality and nonfatal HF after AMI. At multivariate analysis, adjusted relative risks of the composite endpoint for an increase of 10 mL of LV EDV and ESV were, respectively, 1.16 (95% CI, 1.12–1.20) and 1.34 (95% CI, 1.28–1.41).[14] LV regional systolic function has been also proposed for early risk stratification after AMI. Because of its recognized strong inverse relation with LVEF, it is expected that the wall motion score index (WMSI) could predict outcome after AMI. Accordingly, in 144 patients with a first AMI treated with thrombolytic therapy, a predischarge resting WMSI ≥ 1.50 was superior to LVEF ≤ 40% to identify patients who had post-AMI cardiac death, unstable angina, nonfatal reinfarction, and HF.[15] In 767 AMI patients, WMSI was an independent predictor of death and HF hospitalization, whereas LVEF was not.[16] This discrepancy may be explained by the fact that compensatory hyperkinesia of normal myocardial segments may mask the infarction severity and LVEF may therefore underestimate the true amount of myocardial damage after AMI. In addition, RV systolic function provides strong prognostic information in AMI patients treated with PCI. RV fractional area change (RVFAC)—but not TAPSE—independently predicted all-cause mortality, HF hospitalization, or reinfarction; the risk of adverse cardiac events was two-fold increased in patients with RVFAC < 32% compared with those with RVFAC ≥ 32%.[17] The importance of left atrial (LA) dilation for risk stratification of AMI patients has been also investigated. It may occur after AMI in response to either volume (due to a mitral regurgitation [MR], in particular after inferior AMI)[18] or pressure overload (increased LV filling pressures). LA dilation can be accurately detected by the LA volume index (LAVi), whose use is strongly encouraged by American Society of Echocardiography and the European Association of Cardiovascular Imaging recommendations on chamber quantification.[19] A LAVi > 32 mL/m^2 determined within 48 hours after AMI was predictor of all-cause of mortality after AMI.[20] Conversely, in a study by Sakaguchi *et al.*[21] LAVi did not show a prognostic role early after AMI (within 2 days after hospitalization), but LAVi at the time of discharge (average of 16 days after admission) and the difference in LAVi between discharge and admission were both independent predictors of the composite endpoint of cardiac death or hospitalization due to HF. The different results of these two studies can be explained by different endpoints and by the fact that patients with prior AMI, moderate/severe MR, and atrial fibrillation (AF) were excluded in Sakaguchi *et al.*'s study but not in the study by Beinart *et al.*[20] It makes sense that LAVi size detected several days after AMI might predict outcome better than that assessed early, LA dilation being a marker of a long time duration increase of LA pressure. Worthy of note, LA distensibility ([difference of maximal and minimal LA volume]/minimal volume × 100) was also an independent predictor of in-hospital mortality (HR = 2.37 for LA distensibility ≤ 60%; 95% CI, 1.108–5.079; $P = .026$) in 521 post-AMI patients.[22] To the best of our knowledge, no information is available on the possible prognostic value of LA dilation prior to AMI, a factor that should not be underestimated in clinical practice.

Standard Doppler

Standard Doppler of mitral valve, a robust and very feasible technique, provides important prognostic indexes after AMI. LV

restrictive filling pattern (RFP), detected early after AMI, is a recognized predictor of adverse outcome (Figure 2) regardless of LV systolic function. In a study of Temporelli *et al.*, the reversibility of RFP (RFP = E velocity deceleration time [DT] < 130 msec) at predischarge of 571 AMI patients not undergoing revascularization procedures was associated with a more favorable late outcome, whereas its persistence was the most powerful independent predictor of late mortality. In this study, a DT at admission < 130 msec was a strong prognosticator of LV remodeling and survival.[23] In a meta-analysis of 12 prospective studies, including 3,396 post-AMI patients, RFP was confirmed as a predictor of all-cause mortality independently on LVEF, ESV, and Killip HF class.[24] The detection of an LV RFP pattern and a short DT in patients with low LVEF is enough to identify high LV filling pressures and stratify prognosis after AMI, without the need of extending the diastolic evaluation to other parameters. Also, the Tei index may be useful to distinguish post-AMI high-risk patients. In 64 patients evaluated at hospital admission, a Tei index > 0.45 was an independent predictor of HF.[25] Several studies[26,27] demonstrated a significant independent association between the presence of MR and adverse events after AMI. In 1,036 STEMI patients treated by primary PCI, moderate to severe MR (graded according to initial color flow Doppler followed by quantitative parameters) was an independent predictor of death (adjusted HR = 3.1; 95% CI, 1.34–7.2) and HF (adjusted HR = 3.3; 95% CI, 1.16–9.4).[28] In another study[29] moderate/severe MR (graded by the effective regurgitant orifice area) independently predicted subsequent HF. Functional MR was an independent prognosticator in both ischemic and nonischemic dilated cardiomyopathy.[30] The increase of pulmonary artery systolic pressure (PASP) may be a hemodynamic consequence of AMI by pathologic mechanisms such as ischemic MR and LV systolic and diastolic dysfunction. PASP can be determined noninvasively by adding the value of estimated right atrial pressure (size and respiratory reactivity of inferior cava) to the maximal retrograde gradient of tricuspid regurgitation. An estimated elevated PASP was found to be associated with CV morbidity and mortality in this context. Møller *et al.*[31] enrolled 536 AMI patients, excluding those with significant pulmonary and mitral valve stenosis or a history of primary pulmonary hypertension, who underwent echo within 2 days after AMI. At multivariate analysis, PASP degree was correlated with age, LV diastolic function, MR, and WMSI. Each 10-mmHg increase in PASP, each grade increase of LV diastolic dysfunction, and evidence of moderate/severe RV dilatation were independent predictors of all-cause mortality. In another study,[32] a PASP > 35 mmHg at hospital admission was predictive of a combined endpoint of all-cause death and death from HF (adjusted HR = 1.83; 95% CI, 1.31–2.57, $P < .0001$). All these data suggest that post-AMI consequences on pulmonary circulation may have greater prognostic implications than LV systolic dysfunction itself.

Spectral Pulse Wave Tissue Doppler Imaging

Tissue Doppler imaging (TDI) uses Doppler principles to quantify the higher-amplitude, lower-velocity signals of myocardial tissue motion along the axis of Doppler ultrasound beam. TDI has two different modalities: spectral pulse wave TDI, which can be performed during a standard echo-Doppler exam, and color TDI (CTDI), which is obtainable offline, by a post hoc elaboration. Spectral pulsed wave TDI is used to measure peak (instantaneous) myocardial velocities and is well suited to study LV longitudinal function. It has high temporal resolution but does not permit simultaneous analysis of different myocardial segments. The ratio of early transmitral flow velocity to spectral pulsed wave TDI-derived early diastolic velocity of the mitral annulus (E/e′ ratio) correlates accurately with LV filling pressures degree and has been shown to be an accurate predictor of prognosis after AMI. A post-AMI E/e′ ratio > 15 appears to be an independent predictor of all-cause mortality (relative risk = 4.8; 95% CI, 2.1-10.8, $P = .0002$).[33] The E/e′ ratio is also strongly promoted by the American Society of Echocardiography and the European Association of Cardiovascular Imaging recommendations on diastolic function in relation to its high feasibility.[34] It should therefore be used to estimate LV filling pressure degree in AMI patients with impaired or normal LVEF when the transmitral pattern is nonrestrictive and increased LV filling pressures cannot be diagnosed by the simple transmitral flow assessment.[34] This kind of estimation can be valuable to drive therapy, in particular to administrate loop diuretics and avoid pulmonary edema.

ADVANCED ECHOCARDIOGRAPHIC TECHNOLOGIES

Color Tissue Doppler Imaging

CTDI quantifies mean velocities (cm/sec) of a given myocardial segment with the possibility of evaluating multiple structures and segments in a single view. The prognostic value of CTDI has been demonstrated after AMI. In 391 STEMI patients treated with primary PCI, CTDI-derived longitudinal systolic (s′), early diastolic (e′), and late diastolic (a′) myocardial velocities were measured at six mitral annular sites and averaged to provide global estimates. At multivariate analysis, the sum of global s′, e′, and a′ velocities (optimal cutoff point = 17.2 cm/sec) was independently associated with the composite endpoint of all-cause mortality, new AMI, and HF hospitalization.[35] In another study, myocardial performance index assessed by CTDI through the mitral leaflet was an independent predictor of a combined outcome including death, HF hospitalization, or new AMI (optimal cutoff point = 0.52).[36] The major weakness of both spectral pulse wave TDI and CTDI is represented by their angle dependency, common to all Doppler-based technologies. Moreover, TDI is not independent on heart translational motion and is influenced from tethering of adjacent segments. This limitation may be overcome by TDI-derived strain imaging that provides quantitative information on deformation (strain) and rate of deformation (strain rate) of a given myocardial segment or overall myocardium (=global strain). In a study of Sjøli *et al.*,[37] TDI-derived global longitudinal strain (GLS) < −15.6% and LVEF <44% were both independent predictors of cardiac death, reinfarction, HF hospitalization, unstable angina, or life-threatening arrhythmia after AMI. Nevertheless, Doppler-derived strain is strongly limited by technical problems, mainly the need of a high frame rate during images acquisition, vulnerability to signal noise, poor spatial resolution, and angle dependence.[38] These limitations reduce the feasibility and reproducibility of this technique, which has never been introduced in routine clinical practice.

Speckle-Tracking Echocardiography

Two-dimensional STE is based on the quantitative analysis of spatial dislocation (tracking) of acoustic markers ("speckles") generated by the interaction between the ultrasound beam and myocardial fibers during the cardiac cycle. Two-dimensional STE overcomes both the angle dependency and signal-to-noise ratio of CTDI-derived strain imaging.[39] It has a recognized better reproducibility than TDI-derived strain.[40] Two-dimensional STE allows a functional evaluation of all myocardial deformations, longitudinal strain (LS) being a marker of subendocardial

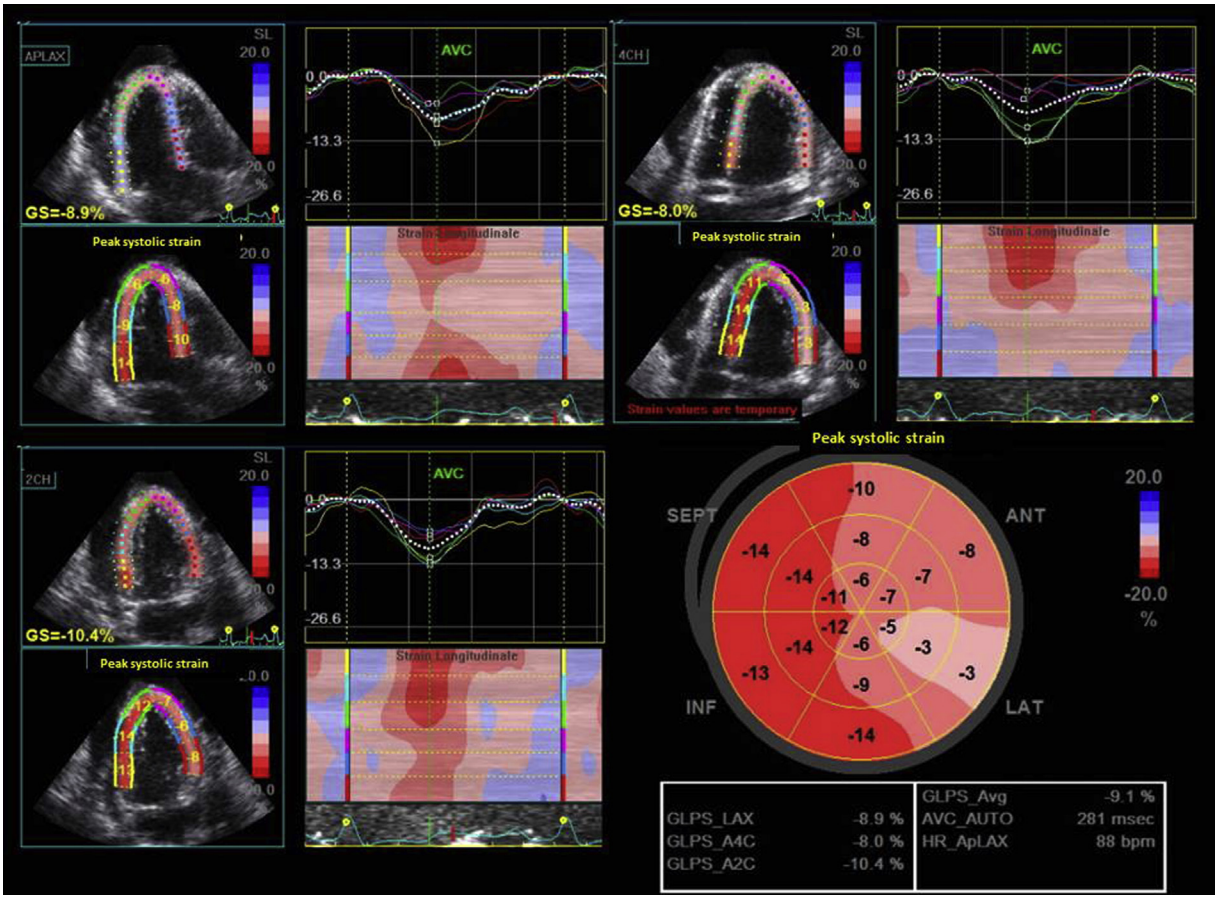

Figure 3 Global and regional LS assessed by 2D STE after anterior AMI. The bull's-eye representation (*right bottom*) depicts a clear involvement of anterior septum and anterior and lateral walls, and the systolic GLS average is −9.1%, a cutoff point value prognostically validated in this clinical setting. In the *left section* of each view (apical long axis and four chamber on the top and two chamber at the *left bottom*) is shown the color representation of STE on 2D imaging and quantitation of peak regional strain values (normally negative) referring to six myocardial regions. In the *right upper section* of each view regional color systolic curves of systolic strain are marked, while the dotted white line corresponds to average strain (GS). In the *right lower section*, qualitative color M-mode strain representation referring to the six consecutive myocardial segments is shown: at the *bottom*, LV basal right segments; in the *central part*, LV apex; at the *top*, LV basal segments (*yellow color*); *dark red* indicates normal strain; *light red*, reduced strain; and *pink color*, strongly impaired or even absent systolic strain. *ANT*, anterior; *AVC*, atrioventricular contraction; *GLPS*, global lateral pulse strain; *GS*, global strain; *INF*, inferior; *LAT*, lateral; *SEPT*, septum.

longitudinal shortening (expressed in negative value), midwall circumferential strain (again negative values), and radial strain (positive values). Notably, subendocardial fibers are more prone to ischemic damage and early altered during acute ischemia, a characteristic that can be efficiently utilized in acute coronary syndromes.[39] Two-dimensional STE is also very feasible, relatively operator independent, and much more reproducible than 2D LVEF,[41,42] another point of strength in this clinical setting. The prognostic value of 2D STE-derived longitudinal function after AMI has been described in recent studies. In 50 anterior AMI patients treated with PCI, systolic LS derived from seven segments of left anterior descending artery territory was an independent predictor of death and HF.[43] In 659 AMI patients treated by primary PCI, systolic GLS and LS rate of the infarct zone were independently associated with all-cause mortality. In particular, at univariate analysis, patients with systolic GLS $> -15.1\%$ and systolic GLS rate (GLSr) > -1.06 sec^{-1} showed HRs of 4.5 (95% CI, 2.1–9.7) and 4.4 (95% CI, 2.0–9.5), respectively, for all-cause mortality.[44] In another study, a reduced systolic GLS predicted HF during hospitalization for AMI also in the presence of normal LVEF.[45] These data suggest that we should pay particular attention to HF symptoms in the first phases of AMI in patients when GLS is impaired. GLS has shown an incremental value beyond traditional echo parameters for risk stratification after AMI. Ersbøll *et al.*[46] found that impaired systolic GLS—but not reduced LVEF (<40%)—was a powerful independent predictor of a composite endpoint of all-cause of mortality and HF hospitalization and cardiac death. A systolic GLS $\geq -14\%$ was associated with a three-fold increase in risk for the combined endpoint of all-cause mortality and HF hospitalization (HR = 3.21; 95% CI, 1.82–5.67; $P < .001$). In a study of 576 AMI patients treated by primary PCI, systolic GLS and WMSI were independently associated with the composite endpoint of all-cause mortality, reinfarction, hospitalization, HF, or stroke within 1 year, whereas LVEF and ESV did not enter the model.[47] In particular, patients with systolic GLS $\geq -10\%$ experienced more adverse events than patients with systolic GLS $\leq -15\%$ (HR = 4.6; 95% CI, 2.8–7.7; $P < .001$). The latest American guidelines[3,4] suggest

administering angiotensin-converting enzyme inhibitors (ACEi) within the first 24 hours in patients with anterior STEMI, HF, or LVEF ≤40% and with NSTEMI and LVEF ≤40%, hypertension, diabetes, or stable chronic kidney disease. Aldosterone antagonist should be given to patients with STEMI or NSTEMI who are already receiving ACEi and beta blockers and who have LVEF ≤40% and either HF symptoms or diabetes.[3,4] On the basis of the above-mentioned evidence, GLS can identify patients who might benefit from antiremodeling therapy independently on LVEF and even represent a selection criterion for future clinical trials. In another study of Ersbøll *et al.*[48] on 988 AMI patients, systolic GLS (HR = 1.24; 95% CI, 1.10–1.40; $P = .0004$) and mechanical dispersion (=SD of average time from ECG-derived peak R to peak systolic LS [HR = 10 msec = 1.15; 95% CI, 1.01–1.31; $P = .031$]) were both associated with risk of ventricular arrhythmias and SCD after AMI at multivariate analysis; these associations remained significant in subgroups of patients with LVEF < 35% and > 35%.[48] The impact of mechanical dispersion on risk prediction on ventricular arrhythmias after AMI has been confirmed by Haugaa and coworkers.[49] Prediction and prevention of SCD early after AMI represent a clinical challenge, and the Defibrillator IN Acute Myocardial Infarction Trial failed to demonstrate an overall survival advantage of implantable cardioverter defibrillator (ICD) implantation within 40 days in AMI patients with in-hospital LVEF ≤35%.[49] Accordingly, both European and American guidelines for management of ventricular arrhythmias suggest reevaluating LVEF (6–12 weeks and 40 days or more after AMI, respectively) to identify candidates for ICD.[4,50] In this clinical contest, the study of Ersbøll *et al.*[48] suggests possible additional information on LS to LVEF in the early AMI phase for identifying more accurately patients to be referred to ICD.[51] On the grounds of all these studies, in the setting of AMI patients, systolic GLS (Figure 3) should be integrated into the assessment of LVEF and WMSI, which are operator dependent and poorly reproducible.

Recently, regional diastolic strain rate has been proposed as a further marker of elevated LV filling pressure degree. In a total of 1,048 post-AMI patients, after a median follow-up of 29 months, the ratio of transmitral E velocity to global diastolic strain rate (E/e′sr, optimal cutoff value >1.25) was superior to E/e′ ratio and independently associated with the composite endpoint of all-cause mortality, HF hospitalization, stroke, and new onset AF, and the prognostic value of E/e′ was driven by mortality (HR per 1-unit change, 2.52; 95% CI, 2.09–3.04; $P < .0001$) and HF admission (HR per 1-unit change, 2.79; 95% CI, 2.23–3.48; $P < .0001$).[52]

In addition, the assessment of RV myocardial function by 2D STE has shown a prognostic value after AMI. In 621 AMI patients treated with primary PCI, RV LS and RVFAC—but not TAPSE—independently predicted a composite endpoint including all-cause mortality, HF hospitalization, or reinfarction; in particular, the risk of adverse outcomes was increased three-fold in patients with RV LS ≥ −22.1% compared with patients with RV LS < −22.1%.[17] Two-dimensional STE can also provide a comprehensive LA assessment. LA function can be divided into three phases: in the first phase, as a reservoir, the LA stores pulmonary venous return during systole and isovolumic relaxation; in the second one, as conduit, the LA transfers blood passively into LV; in the third, as booster pump, it actively contracts during end diastole. During the reservoir phase, LA LS increases as a consequence of atrial filling, achieving a positive peak just before mitral valve opening, the so-called peak atrial LS. LA strain has been explored in 843 patients with AMI: at univariate analysis, peak atrial strain predicted HF hospitalization, but at multivariate analysis, LV systolic GLS and maximal LA volume removed this effect.[53] Accordingly, further experience is needed to eventually promote LA strain in this clinical setting.

Recently, a layer-specific analysis of myocardial deformation has allowed accurate discrimination of transmurality of myocardial infarction[54] and prediction of functional recovery after AMI.[55] However, further studies are needed to clarify the prognostic role of layer-specific strain analysis in patients with AMI.

Coronary Flow Velocity Pattern and Reserve

Doppler-derived coronary flow velocity reserve (CFVR) has also been proposed for early risk stratification in patients after AMI. Iwakura *et al.*[56] first demonstrated that coronary flow assessed by invasive Doppler guide wire allows the identification of the presence of coronary microvascular dysfunction after AMI. In particular, the coronary flow pattern in patients with no reflow was characterized by the appearance of systolic retrograde flow, diminished systolic anterograde flow, and rapid deceleration of diastolic flow after coronary intervention. CFVR can also be obtained noninvasively by transthoracic Doppler echo, which is easily available at bedside and not expensive.[57] In a small prospective study,[58] a DT of diastolic coronary flow velocity ≤600 msec, assessed by this technique 12–48 hours after PCI, was an independent predictor of subsequent HF (OR = 11.04; 95% CI, 1.43–85.11; $P = .021$). In 55 patients with first anterior STEMI successfully treated with primary PCI (thrombolysis in myocardial infarction flow grade 3), a CFVR < 1.7 determined <12 hours after symptoms onset independently predicted in-hospital cardiac events (in particular, HF [OR = 12; 95% CI, 1.4–93; $P < .02$]) as well as global and regional LV function recovery (OR = 23; 95% CI, 5–98; $P < .01$; and OR = 58; 95% CI, 2–73; $P = .01$, respectively).[59] This finding was interpreted by the authors as due to coronary microvascular dysfunction because of the documented patency of the epicardial coronary vessel. On the basis of these results, noninvasive detection of impaired coronary microcirculation, despite successful percutaneous treatment of the culprit lesion, could drive management of patients with low LVEF to prevent in-hospital HF: patients with CFVR > 1.7 should not be referred for prophylactic ICD implantation because of a high probability of LV function recovery.

Myocardial Contrast Echocardiography

MCE is a bedside technique that assesses myocardial perfusion using microbubbles and can be used to determine infarct size and hence myocardial viability; it is a powerful predictor of hard cardiac events after AMI. In a study by Lepper *et al.*, the size of persistent MCE perfusion defect after revascularization for AMI had a high predictive value for LV remodeling during a 4-week follow-up period.[60] In a study of Dwivedi *et al.*,[61] 95 patients underwent MCE within 7 ± 2 days after AMI, and residual myocardial viability was assessed through contrast defect index (CDI): this was calculated as the sum of the contrast scores (1 = homogeneous opacification; 2 = heterogeneous opacification; 3 = minimal/absent contrast opacification) of all LV interpretable segments divided by the number of LV segments analyzed. At multivariable analysis, elevated CDI, as a marker of lower residual myocardial viability, was a predictor of the composite endpoint of cardiac death and nonfatal AMI and of cardiac death, whereas LVEF and ESV did not achieve statistical significance. The optimal cutoff of CDI

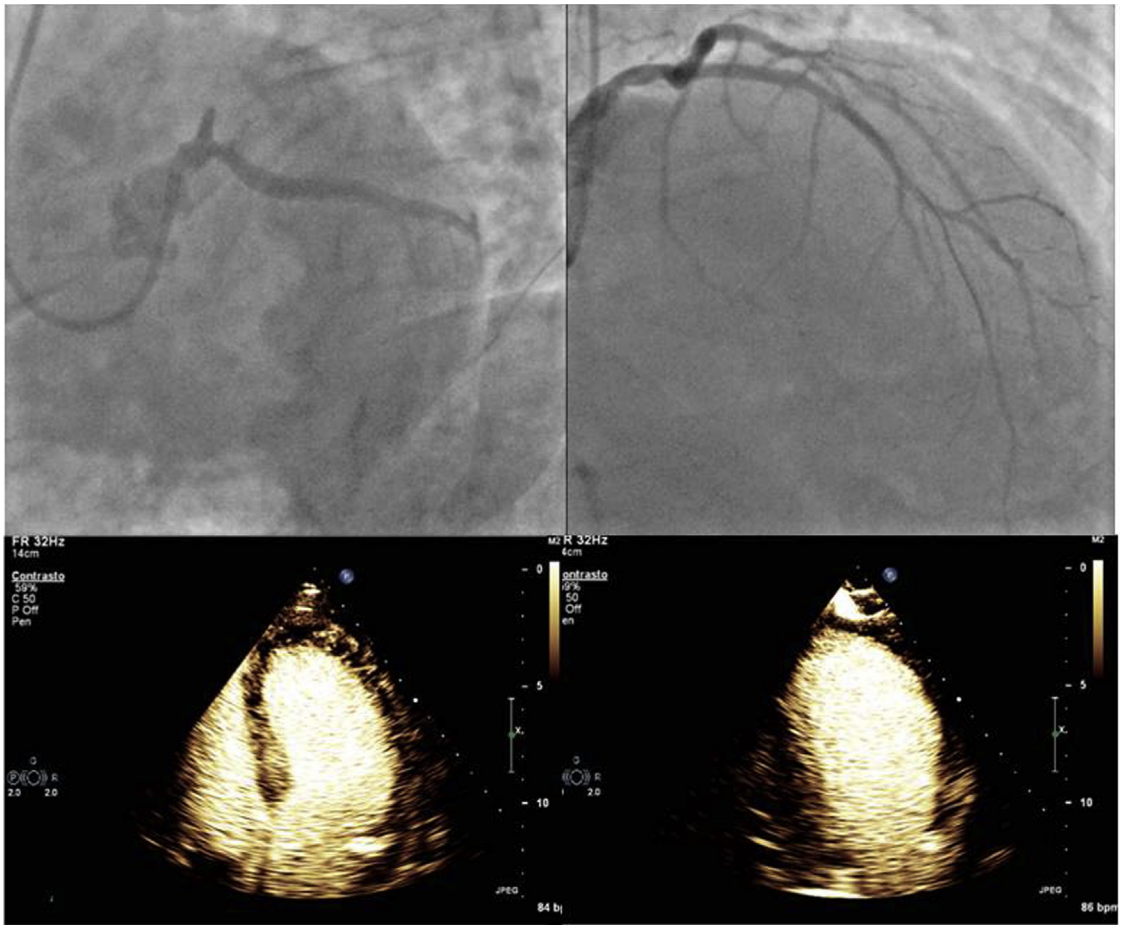

Figure 4 MCE performed after successful percutaneous transluminal coronary angioplasty. At the *top left*, the coronary angiography shows the total proximal left anterior descending (LAD) coronary artery occlusion before performing primary PCI. The *top right* shows the open artery after thromboaspiration and primary PCI with good final result (thrombolysis in myocardial infarction 3 flow). The *bottom left* shows an echocardiographic four-chamber view obtained soon after primary angioplasty with infusion of a 0.5-mL bolus of microbubble contrast media (SonoVue), using contrast-specific very-low mechanical index real-time imaging. It shows almost complete absence of tissue-level perfusion (black myocardium) in the apical and midapical segments (which were also akinetic) compared with normally perfused basal and non-LAD segments. The *bottom right* similarly shows the echocardiographic two-chamber view, which confirms the absence of myocardial perfusion in the mid and apical anterior segments.

was 1.86 for the composite endpoint of cardiac death and nonfatal AMI (sensitivity, 62%; specificity, 84%) and for cardiac death (sensitivity, 87%; specificity, 84%). In addition, the extent of microvascular damage, quantified by CDI, was the most important predictor of LV remodeling among patients with first successfully reperfused STEMI (thrombolysis in myocardial infarction flow grade 3). It is conceivable that LV function assessed early after AMI could overestimate the extent of irreversible infarct area because of myocardial stunning, while MCE—by assessing the infarct area through the absence/reduction of tissue-level perfusion—could provide a more realistic picture and stronger prognostic information, especially in patients with impaired LVEF.

Figure 4 shows a typical example of incremental usefulness of downstream tissue-level perfusion assessment compared with angiographic data. Real-time MCE clearly shows the absence of myocardial perfusion a few hours after primary PCI. MCE is able to detect the relevant percentage of patients (1/4 to 1/3) with a no-reflow phenomenon after AMI,[62,63] similar to cardiac magnetic resonance, but with lower cost and easy performance at the bedside in a coronary care unit. The application of MCE molecular imaging in acute coronary syndromes can be of potential interest. This technology is based on the use of microbubbles functionalized with ligand molecules that bind to molecular markers of disease. Since during acute myocardial ischemia/reperfusion an endothelial up-regulation of leukocyte adhesion molecules occurs and persists even after ischemia resolution (ischemic memory), microbubbles adhering to endothelial selectins might permit echo identification of recently ischemic myocardial territories and facilitate the diagnostic approach in patients with chest pain of undetermined origin.[64,65] Future investigations are needed to clarify the role of MCE molecular imaging in this clinical context.

Three-Dimensional Echocardiography

RT3DE imaging is a major innovation of cardiac ultrasound. It can provide accurate and reliable measurements of chamber size, mass, geometry, and function. It also offers a comprehensive anatomic characterization of valvular abnormalities, improving diagnosis and

Table 1 Independent associations of conventional and advanced echocardiographic indexes assessed early after AMI with adverse outcomes

First author	Echo time	Index	Cutoff point	Mean duration of FU (months)	Adverse outcome
Verma[7]	Mean 5.0 ± 2.5 days after AMI	LVM RWT	>115 g/m^2 (M) >95 g/m^2 (F) >0.42	24.7	All-cause mortality; CV death; all-cause mortality + HF
Brand[8]	Mean 4.2 ± 6.4 days after AMI	MAPSE	<8 mm	21	All-cause mortality + HF + AMI + UA
Samad[9]	2–4 days after AMI	TAPSE	≤15 mm	24	All-cause mortality
Schwammenthal[11]	≤2 days of admission	LV EF	≤40%	1	All-cause mortality + AMI + HF
Nicolosi[14]	Median 9 days after AMI	LV EF LV EDV LV ESV		6	All-cause mortality + HF
Carluccio[15]	6–8 days after AMI	WMSI	≥1.5	18	Cardiac death Cardiac death + UA + AMI Cardiac death + UA + AMI + HF
Møller[16]	0–2 days after admission	WMSI		40	All-cause mortality; HF
Antoni[17]	≤2 days of admission	RVFAC RV strain*	<32% ≥−22%	24	All-cause mortality; HF All-cause mortality + AMI + HF
Beinart[20]	≤2 days of admission	LAVi	>32 mL/m^2	60	All-cause mortality
Sakaguchi[21]	Mean 16 days after admission	LAVi	>32 mL/m^2	26	Cardiac death + HF
Hsiao[22]	4.1–5.2 hours after symptom onset	LAd	≤60%		In-hospital mortality
Temporelli[23]	Mean 12 ± 5 days after confirmed AMI	DT	<13 msec	48	All-cause mortality
Poulsen[25]	≤1 hour of hospitalization	MPI†	>0.45		In-hospital HF
López-Pérez[28]	Median 4 days after PTCA	MR	Moderate or severe	33.6	All-cause mortality; HF; all-cause mortality + HF
Carrabba[29]	≤1 days of admission	MR	Moderate or severe	18	HF
Møller[31]	0–2 days after AMI	PASP		40	All-cause mortality
Mutlak[32]	Median 2 days after admission	PASP	>35 mmHg	12	HF
Hillis[33]	mean 1.6 ± 1.6 days after admission	E/e′	>15	13	All-cause mortality
Biering-Sørensen[35]	Median 2 days after PTCA	Global s′+e′+a′‡	<17.2 cm/sec	25	All-cause mortality + HF + AMI
Biering-Sørensen[36]	Median 2 days after admission	MPI§	≥0.52	25	All-cause mortality + HF + AMI
Sjøli[37]	Mean 2 hours after thrombolysis therapy and mean 10 ± 5 days after admission	GLS‖	>−15.6%	39.5	Cardiac death + AMI + HF + UA + life threatening arrhythmia
Park[43]	≤2 days after PTCA	LS of the infarct zone*		18.3	All-cause mortality + HF
Antoni[44]	≤2 days of admission	GLS*	≥−15.1%	21	All-cause of mortality; AMI + revascularization + HF All-cause mortality + AMI + revascularization + HF
Antoni[44]	≤2 days of admission	GLSr*	≥−1.06s^{-1}	21	AMI + revascularization + H; all-cause mortality + AMI + revascularization + HF
Ersbøll[45]	≤2 days of admission	GLS*			In-hospital HF
Ersbøll[46]	≤2 days of admission	GLS*	≥−14%	30	CV death; HF; all-cause mortality + HF
Munk[47]	≤1 day after PTCA	GLS*		24	All-cause mortality + stroke + AMI + HF
Ersbøll[48]	≤2 days of admission	GLS*		29.7	SCD + admission with VA + appropriate therapy from ICD

Ersbøll[52]	≤2 days of admission	E/e′sr*	>1.25	29	All-cause mortality; all-cause mortality + stroke + HF + new-onset AF
Ersbøll[53]	≤2 days of admission	GLS*		23	All-cause mortality + HF
Katayama[58]	0.5-2 days after PTCA	diastolic CFV DT	≤600 msec		In-hospital HF
Meimoun[59]	<1 day after PTCA	CFVR	<1.7		In-hospital Death + AMI + HF
Dwivedi[61]	Median 7 ± 2 days after AMI	CDI¶	>1.68	46	Cardiac death Cardiac death + AMI

CFV, Coronary flow velocity; *E/e′sr*, ratio of transmitral E velocity to global diastolic strain rate; *F*, female; *FU*, follow-up; *LAd*, left atrial distensibility; *LVM*, left ventricle mass; *M*, male; *MPI*, myocardial performance index; *PTCA*, percutaneous transluminal coronary angioplasty; *UA*, unstable angina; *VA*, ventricular arrhythmias.
Studies are in the same order as they are cited in the text.
*Assessed by STE.
†Assessed by standard Doppler.
‡Assessed by CTDI.
§Assessed by CTDI M-mode of mitral leaflet.
‖Assessed by TDI.
¶Assessed by MCE.

preoperative planning. RT3DE overcomes several limitations of 2D echo because it is more accurate for evaluating LV volumes and LVEF without the need of a geometric assumption and errors caused by possible foreshortened views.[66] To date, no clinical study has demonstrated any kind of association of RT3DE parameters with major adverse CV events after AMI. However, some data have shown the usefulness of an RT3DE-derived sphericity index calculated 6 days after the acute events in predicting subsequent LV remodeling.[67] In a recent study of 100 patients with successfully reperfused first AMI, 3D STE discriminated transmural from nontransmural extent of scar and predicted microvascular obstruction, using cardiac magnetic resonance as the gold standard. The best 3D strain component for detecting nonviable segments with microvascular obstruction was global area strain (area under the curve = 0.867),[68] a parameter that encompasses longitudinal and circumferential strain.[69] In another study including 160 patients with first AMI treated by primary PCI, global 3D LS provided incremental value over clinical and conventional echo variables in predicting global LV function recovery (the c-statistic improved from 0.64 to 0.71 to 0.84).[70]

Stress Echocardiography

SE performed early after AMI has been demonstrated to predict subsequent recovery of global[71] and regional[72] systolic function. The viability of two or more contiguous to infarct regions, by low-dose dobutamine SE—performed 3 days after AMI—was an independent predictor of 3-month LVEF improvement.[71] The viability of two or more contiguous to infarct segments at low-level exercise SE and at low-dose dobutamine—5 ± 2 days after AMI—predicted myocardial recovery at rest 1 month later.[72] Even 2D STE applied to low-dose dobutamine SE appears to be promising for determining the probability of LV functional recovery after AMI.[73,74] In particular, LS $< -14.92\%$ and LS rate < -1.30 sec^{-1} at low dobutamine predicted LV regional recovery with sensitivity of 86.0%, 87.0%, and 86.1% and specificity of 87.6%, 83.3%, and 86.1%, respectively.[73] SE also identifies AMI patients at increased risk of subsequent adverse events. In a large-scale multicenter prospective study,[75] 755 in-hospital patients underwent pharmacological SE with either dipyridamole or dobutamine and an exercise ECG 10 days from AMI. At multivariate analysis, peak stress WMSI was an independent predictor of all-cause mortality; WMSI difference between rest and peak stress was the most important predictor of the composite endpoint of death, nonfatal AMI, and rehospitalization for unstable angina. In the Cost of Strategies after Myocardial Infarction (COSTAMI) trial,[76] a pharmacological SE performed early after uncomplicated AMI followed by early discharge in case of negative test provided a similar clinical outcome but lower costs than delayed discharge followed by exercise ECG. In the COSTAMI II trial,[77] exercise SE early after AMI allowed risk stratification with similar costs to predischarge exercise ECG. The Viability-Guided Angioplasty after Acute Myocardial Infarction trial[78] demonstrated that in patients with sub-AMI, not treated by primary or rescue PCI, the detection of myocardial viability at low-dose dobutamine identified a subgroup to be referred to viability-guided PCI of infarct-related coronary artery.

A Comprehensive Echocardiographic Prognosticator

In the last 20 years, several studies have addressed the importance of transthoracic echocardiography for the prediction of risk for subsequent adverse events after AMI. Supplemental Tables 1 and 2

Figure 5 A proposed sequential multiparametric algorithm for early risk stratification of patients with AMI treated by PCI. In the *left part* of the figure are the parameters of the proposed algorithm; in the *middle*, their prognostic value; and on the *right*, their possible impact on decision making. Superscript numbers are reference citations. *ARB*, Angiotensin receptor blocker; *E/e'*, ratio of transmitral E velocity to early diastolic velocity of mitral annulus; *LVFP*, left ventricular filling pressures; *MRA*, mineralcorticoid receptor antagonist; *Pts*, patients; *RAAS*, renin-angiotensin-aldosterone system; *RVOT*, RV outflow tract; *WCD*, wearable cardioverter defibrillator.

summarize the characteristics of the study populations of studies using conventional echo indexes and advanced ultrasound technologies, respectively. Populations were similar for age, gender, and CV risk factors, but the therapeutic strategies for AMI were different. Obviously, in most studies dealing with advanced imaging, AMI patients were treated by myocardial revascularization, a setting where the degree of LV dysfunction is generally mild. Conversely, in historical studies using conventional echo, several patients followed a conservative strategy, with a consequent higher rate of LV dysfunction and HF. LVEF,[12] LAVi,[21] RVFAC,[17] MR degree,[28,29] PASP,[31] and E/e' ratio[33] emerge as the main conventional parameters in AMI patients treated by PCI. According to the scientific literature, morphologic and functional data can be very useful to predict complications during AMI hospitalization[22,25,45,58,59] and more delayed post-AMI CV events and mortality. Table 1 reports independent associations (by multiple linear regression analyses) of echo indexes with clinical outcomes, specifying the timing of echo and follow-up duration. In all of the studies, it was not possible to distinguish echo abnormalities preceding AMI from those caused by AMI because preacute event data were not available. In relation to the need for an early risk stratification for patients experiencing AMI, we propose an echo-based algorithm for AMI patients (Figure 5). Of interest, in studies showing incremental value of GLS[46] and RV strain[17] over conventional echo parameters (LVEF or RVFAC and TAPSE), the majority of patients enrolled were treated with PCI (79.1% and 100%, respectively) and had normal or mildly reduced LVEF. The strength of this approach is consistent with the possibility of identifying post-AMI patients with LVEF > 40%—who are not particularly considered by actual guidelines for more aggressive treatment (antiremodeling, antiarrhythmic therapy, etc.)—but who could nevertheless have an adverse prognosis. The main weakness of this approach corresponds to the limited availability of advanced echo and the need for physicians with advanced ultrasound competencies in coronary care units, the lack of well-defined prognostic cutoff points of novel echo parameters in relation to specific endpoints, and the absence of information on optimal timing of echo exam during and after hospitalization.

CONCLUSIONS

Transthoracic echocardiography plays a fundamental role in early risk stratification of patients with AMI. Standard echo-Doppler provides powerful prognosticators that are well validated in several

large clinical studies. More recently, advanced echocardiography—mainly 2DSTE and MCE—have been shown to predict long-term adverse outcomes and to provide additional information beyond conventional echo parameters. RT3DE has been also introduced as a promising technique, but its role for risk stratification after AMI is derived from few studies and needs further investigation on large population sample sizes and with longer follow-up periods. In general, echocardiographic findings that predict short-term adverse events (such as mechanical complications, early arrhythmias) may differ substantially from findings that predict long-term adverse outcomes such as systolic HF, death, and late arrhythmias. In an era where clinicians are continually pressed to work efficiently, advanced echo technique, in particular STE-derived systolic GLS, should be taken into account to stratify the CV risk profile and drive management in patients admitted to coronary care units for AMI better than conventional ultrasound assessment. The mild to moderate increase of time for a performance of a comprehensive echo exam could be largely balanced by the improvement of care efficiency and possible cost reduction corresponding to a decrease in rehospitalization.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.echo.2017.01.020.

REFERENCES

1. Lancellotti P, Price S, Edvardsen T, Cosyns B, Neskovic AN, Dulgheru R, et al. The use of echocardiography in acute cardiovascular care: recommendations of the European Association of Cardiovascular Imaging and the Acute Cardiovascular Care Association. Eur Heart J Cardiovasc Imaging 2015;16:119-46.
2. Grenne B, Eek C, Sjøli B, Dahlslett T, Uchto M, Hol PK, et al. Acute coronary occlusion in non-ST-elevation acute coronary syndrome: outcome and early identification by strain echocardiography. Heart 2010;96:1550-6.
3. Amsterdam EA, Wenger NK, Brindis RG, Casey DE Jr., Ganiats TG, Holmes DR Jr, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. J Am Coll Cardiol 2014;64:e139-228.
4. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr., Chung MK, de Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2013;127:e362-425.
5. Steg PG, James SK, Atar D, Badano LP, Blömstrom-Lundqvist C, Borger MA, et al., for the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology (ESC). ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J 2012;33:2569-619.
6. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, et al. 2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2016;37:267-315.
7. Verma A, Meris A, Skali H, Ghali JK, Arnold JM, Bourgoun M, et al. Prognostic implications of left ventricular mass and geometry following myocardial infarction: the VALIANT (VALsartan In Acute myocardial iNfarcTion) Echocardiographic Study. JACC Cardiovasc Imaging 2008;1:582-91.
8. Brand B, Rydberg E, Ericsson G, Gudmundsson P, Willenheimer R. Prognostication and risk stratification by assessment of left atrioventricular plane displacement in patients with myocardial infarction. Int J Cardiol 2002;83:35-41.
9. Samad BA, Alam M, Jensen-Urstad K. Prognostic impact of right ventricular involvement as assessed by tricuspid annular motion in patients with acute myocardial infarction. Am J Cardiol 2002;90:778-81.
10. Cameli M, Mondillo S, Solari M, Righini FM, Andrei V, Contaldi C, et al. Echocardiographic assessment of left ventricular systolic function: from ejection fraction to torsion. Heart Fail Rev 2016;21:77-94.
11. Schwammenthal E, Adler Y, Amichai K, Sagie A, Behar S, Hod H, et al. Prognostic value of global myocardial performance indices in acute myocardial infarction: comparison to measures of systolic and diastolic left ventricular function. Chest 2003;124:1645-51.
12. Shiga T, Hagiwara N, Ogawa H, Takagi A, Nagashima M, Yamauchi T, et al. Sudden cardiac death and left ventricular ejection fraction during long-term follow-up after acute myocardial infarction in the primary percutaneous coronary intervention era: results from the HIJAMI-II registry. Heart 2009;95:216-20.
13. Volpi A, De Vita C, Franzosi MG, Geraci E, Maggioni AP, Mauri F, et al. Determinants of 6-month mortality in survivors of myocardial infarction after thrombolysis. Results of the GISSI-2 data base. The Ad Hoc Working Group of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI)-2 Data Base. Circulation 1993;88:416-29.
14. Nicolosi GL, Latini R, Marino P, Maggioni AP, Barlera S, Franzosi MG, et al. The prognostic value of predischarge quantitative two-dimensional echocardiographic measurements and the effects of early lisinopril treatment on left ventricular structure and function after acute myocardial infarction in the GISSI-3 Trial. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico. Eur Heart J 1996;17:1646-56.
15. Carluccio E, Tommasi S, Bentivoglio M, Buccolieri M, Prosciutti L, Corea L, et al. Usefulness of the severity and extent of wall motion abnormalities as prognostic markers of an adverse outcome after a first myocardial infarction treated with thrombolytic therapy. Am J Cardiol 2000;85:411-5.
16. Møller JE, Hillis GS, Oh JK, Reeder GS, Gersh BJ, Pellikka PA. Wall motion score index and ejection fraction for risk stratification after acute myocardial infarction. Am Heart J 2006;415:419-25.
17. Antoni ML, Scherptong RW, Atary JZ, Boersma E, Holman ER, van der Wall EE, et al. Prognostic value of right ventricular function in patients after acute myocardial infarction treated with primary percutaneous coronary intervention. Circ Cardiovasc Imaging 2010;3:264-71.
18. Hong SJ, Kim SA, Hur J, Rhee SJ, Shim CY, Kim JS, et al. Mechanism of mitral regurgitation in the acute phase of inferior wall myocardial infarction: reduced closing force as a consequence of left ventricular systolic dysfunction in the presence of tethering as a determinant of mitral regurgitation. Circ J 2011;75:619-25.
19. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1-3914.
20. Beinart R, Boyko V, Schwammenthal E, Kuperstein R, Sagie A, Hod H, et al. Long-term prognostic significance of left atrial volume in acute myocardial infarction. J Am Coll Cardiol 2004;44:327-34.
21. Sakaguchi E, Yamada A, Sugimoto K, Ito Y, Shiino K, Takada K, et al. Prognostic value of left atrial volume index in patients with first acute myocardial infarction. Eur J Echocardiogr 2011;12:440-4.
22. Hsiao SH, Chiou KR, Porter TR, Huang WC, Lin SK, Kuo FY, et al. Left atrial parameters in the estimation of left ventricular filling pressure and prognosis in patients with acute coronary syndrome. Am J Cardiol 2011;107:1117-24.
23. Temporelli PL, Giannuzzi P, Nicolosi GL, Latini R, Franzosi MG, Gentile F, et al. Doppler-derived mitral deceleration time as a strong prognostic

marker of left ventricular remodeling and survival after acute myocardial infarction: results of the GISSI-3 echo substudy. J Am Coll Cardiol 2004; 43:1646-53.
24. Møller JE, Møller JE, Whalley GA, Dini FL, Doughty RN, Gamble GD, et al., Meta-Analysis Research Group in Echocardiography (MeRGE) AMI Collaborators. Independent prognostic importance of a restrictive left ventricular filling pattern after myocardial infarction: an individual patient meta-analysis: Meta-Analysis Research Group in Echocardiography Acute Myocardial Infarction. Circulation 2008;117:2591-8.
25. Poulsen SH, Jensen SE, Tei C, Seward JB, Egstrup K. Value of the Doppler index of myocardial performance in the early phase of acute myocardial infarction. J Am Soc Echocardiogr 2000;13:723-30.
26. Núñez-Gil IJ, Estrada I, Pérez de Isla L, Feltes G, De Agustín JA, Vivas D, et al. Functional mitral regurgitation after a first non-ST segment elevation acute coronary syndrome: very-long-term follow-up, prognosis and contribution to left ventricular enlargement and atrial fibrillation development. Heart 2013;99:1502-8.
27. Amigoni M, Meris A, Thune JJ, Mangalat D, Skali H, Bourgoun M, et al. Mitral regurgitation in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both: prognostic significance and relation to ventricular size and function. Eur Heart J 2007;23:326-33.
28. López-Pérez M, Estévez-Loureiro R, López-Sainz A, Couto-Mallón D, Soler-Martin MR, Bouzas-Mosquera A, et al. Long-term prognostic value of mitral regurgitation in patients with ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention. Am J Cardiol 2014;113:907-12.
29. Carrabba N, Parodi G, Valenti R, Shehu M, Migliorini A, Memisha G, et al. Clinical implications of early mitral regurgitation in patients with reperfused acute myocardial infarction. J Card Fail 2008;14:48-54.
30. Rossi A, Dini FL, Faggiano P, Agricola E, Cicoira M, Frattini S, et al. Independent prognostic value of functional mitral regurgitation in patients with heart failure. A quantitative analysis of 1256 patients with ischaemic and non-ischaemic dilated cardiomyopathy. Heart 2011;97: 1675-80.
31. Møller JE, Hillis GS, Oh JK, Pellikka PA. Prognostic importance of secondary pulmonary hypertension after acute myocardial infarction. Am J Cardiol 2005;96:199-203.
32. Mutlak D, Lessick J, Carasso S, Kapeliovich M, Dragu R, Hammerman H, et al. Utility of pulmonary hypertension for the prediction of heart failure following acute myocardial infarction. Am J Cardiol 2012;109:1254-9.
33. Hillis GS, Møller JE, Pellikka PA, Gersh BJ, Wright RS, Ommen SR, et al. Noninvasive estimation of left ventricular filling pressure by E/e' is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol 2004;43:360-7.
34. Nagueh SF, Appleton CP, Gillebert TC, Marino PN, Oh JK, Smiseth OA, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: An update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2016;29:277-314.
35. Biering-Sørensen T, Jensen JS, Pedersen S, Galatius S, Hoffmann S, Jensen MT, et al. Doppler tissue imaging is an independent predictor of outcome in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. J Am Soc Echocardiogr 2014;27:258-67.
36. Biering-Sørensen T, Mogelvang R, Søgaard P, Pedersen SH, Galatius S, Jørgensen PG, et al. Prognostic value of cardiac time intervals by tissue Doppler imaging M-mode in patients with acute ST-segment-elevation myocardial infarction treated with primary percutaneous coronary intervention. Circ Cardiovasc Imaging 2013;6:457-65.
37. Sjøli B, Grenne B, Smiseth OA, Edvardsen T, Brunvand H. The advantage of global strain compared to left ventricular ejection fraction to predict outcome after acute myocardial infarction. Echocardiography 2011;28: 556-63.
38. Marwick TH. Measurement of strain and strain rate by echocardiography: ready for prime time? J Am Coll Cardiol 2006;47:1313-27.
39. Mor-Avi V, Lang RM, Badano LP, Belohlavek M, Cardim NM, Derumeaux G, et al. Current and evolving echocardiographic techniques for the quantitative evaluation of cardiac mechanics: ASE/EAE consensus statement on methodology and indications endorsed by the Japanese Society of Echocardiography. J Am Soc Echocardiogr 2011;12:167-205.
40. Sjøli B, Ørn S, Grenne B, Ihlen H, Edvardsen T, Brunvand H. Diagnostic capability and reproducibility of strain by Doppler and by speckle tracking in patients with acute myocardial infarction. JACC Cardiovasc Imaging 2009;2:24-33.
41. Macron L, Lairez O, Nahum J, Berry M, Deal L, Deux JF, et al. Impact of acoustic window on accuracy of longitudinal global strain: a comparison study to cardiac magnetic resonance. Eur J Echocardiogr 2011;12:394-9.
42. Farsalinos KE, Daraban AM, Ünlü S, Thomas JD, Badano LP, Voigt JU. Head-to-head comparison of global longitudinal strain measurements among nine different vendors: the EACVI/ASE Inter-Vendor Comparison Study. J Am Soc Echocardiogr 2015;28:1171-11812.
43. Park YH, Kang SJ, Song JK, Lee EY, Song JM, Kang DH, et al. Prognostic value of longitudinal strain after primary reperfusion therapy in patients with anterior-wall acute myocardial infarction. J Am Soc Echocardiogr 2008;21:262-7.
44. Antoni ML, Mollema SA, Delgado V, Atary JZ, Borleffs CJ, Boersma E, et al. Prognostic importance of strain and strain rate after acute myocardial infarction. Eur Heart J 2010;31:1640-7.
45. Ersbøll M, Valeur N, Mogensen UM, Andersen MJ, Møller JE, Hassager C, et al. Relationship between left ventricular longitudinal deformation and clinical heart failure during admission for acute myocardial infarction: a two-dimensional speckle-tracking study. J Am Soc Echocardiogr 2012; 25:1280-9.
46. Ersbøll M, Valeur N, Mogensen UM, Andersen MJ, Møller JE, Velazquez EJ, et al. Prediction of all-cause mortality and heart failure admissions from global left ventricular longitudinal strain in patients with acute myocardial infarction and preserved left ventricular ejection fraction. J Am Coll Cardiol 2013;61:2365-73.
47. Munk K, Andersen NH, Terkelsen CJ, Bibby BM, Johnsen SP, Bøtker HE, et al. Global left ventricular longitudinal systolic strain for early risk assessment in patients with acute myocardial infarction treated with primary percutaneous intervention. J Am Soc Echocardiogr 2012;25:644-51.
48. Ersbøll M, Valeur N, Andersen MJ, Mogensen UM, Vinther M, Svendsen JH, et al. Early echocardiographic deformation analysis for the prediction of sudden cardiac death and life-threatening arrhythmias after myocardial infarction. JACC Cardiovasc Imaging 2013;6:851-60.
49. Haugaa KH, Grenne BL, Eek CH, Ersbøll M, Valeur N, Svendsen JH, et al. Strain echocardiography improves risk prediction of ventricular arrhythmias after myocardial infarction. JACC Cardiovasc Imaging 2013;6: 841-50.
50. Hohnloser SH, Kuck KH, Dorian P, Roberts RS, Hampton JR, Hatala R, et al. Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med 2004;351:2481-8.
51. Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: the Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 2015;36:2793-867.
52. Ersbøll M, Andersen MJ, Valeur N, Mogensen UM, Fahkri Y, Thune JJ, et al. Early diastolic strain rate in relation to systolic and diastolic function and prognosis in acute myocardial infarction: a two-dimensional speckle-tracking study. Eur Heart J 2014;35:648-56.
53. Ersbøll M, Andersen MJ, Valeur N, Mogensen UM, Waziri H, Møller JE, et al. The prognostic value of left atrial peak reservoir strain in acute myocardial infarction is dependent on left ventricular longitudinal function and left atrial size. Circ Cardiovasc Imaging 2013;6:26-33.
54. Becker M, Ocklenburg C, Altiok E, Füting A, Balzer J, Krombach G, et al. Impact of infarct transmurality on layer-specific impairment of myocardial function: a myocardial deformation imaging study. Eur Heart J 2009;30: 1467-76.

55. Altiok E, Tiemann S, Becker M, Koos R, Zwicker C, Schroeder J, et al. Myocardial deformation imaging by two-dimensional speckle-tracking echocardiography for prediction of global and segmental functional changes after acute myocardial infarction: a comparison with late gadolinium enhancement cardiac magnetic resonance. J Am Soc Echocardiogr 2014;27:249-57.
56. Iwakura K, Ito H, Takiuchi S, Taniyama Y, Nakatsuchi Y, Negoro S, et al. Alternation in the coronary blood flow velocity pattern in patients with no reflow and reperfused acute myocardial infarction. Circulation 1996; 94:1269-75.
57. Meimoun P, Tribouilloy C. Non-invasive assessment of coronary flow and coronary flow reserve by transthoracic Doppler echocardiography: a magic tool for the real world. Eur J Echocardiogr 2008;9:449-57.
58. Katayama M, Yamamuro A, Ueda Y, Tamita K, Yamabe K, Ibuki M, et al. Coronary flow velocity pattern assessed noninvasively by transthoracic color Doppler echocardiography serves as a predictor of adverse cardiac events and left ventricular remodeling in patients with acute myocardial infarction. J Am Soc Echocardiogr 2006;19:335-40.
59. Meimoun P, Malaquin D, Benali T, Boulanger J, Zemir H, Sayah S, et al. Non-invasive coronary flow reserve after successful primary angioplasty for acute anterior myocardial infarction is an independent predictor of left ventricular recovery and in-hospital cardiac events. J Am Soc Echocardiogr 2009;22:1071-9.
60. Lepper W, Kamp O, Vanoverschelde JL, Franke A, Sieswerda GT, Pasquet A, et al. Intravenous myocardial contrast echocardiography predicts left ventricular remodeling in patients with acute myocardial infarction. J Am Soc Echocardiogr 2002;15:849-56.
61. Dwivedi G, Janardhanan R, Hayat SA, Swinburn JM, Senior R. Prognostic value of myocardial viability detected by myocardial contrast echocardiography early after acute myocardial infarction. J Am Coll Cardiol 2007;50:327-34.
62. Galiuto L, Garramone B, Scarà A, Rebuzzi AG, Crea F, La Torre G, et al. The extent of microvascular damage during myocardial contrast echocardiography is superior to other known indexes of post-infarct reperfusion in predicting left ventricular remodeling. J Am Coll Cardiol 2008;51:552-9.
63. Dwivedi G, Janardhanan R, Hayat SA, Lim TK, Greaves K, Senior R. Relationship between myocardial perfusion with myocardial contrast echocardiography and function early after acute myocardial infarction for the prediction of late recovery of function. Int J Cardiol 2010;140:169-74.
64. Villanueva FS, Lu E, Bowry S, Kilic S, Tom E, Wang J, et al. Myocardial ischemic memory imaging with molecular echocardiography. Circulation 2007;115:345-52.
65. Davidson BP, Kaufmann BA, Belcik JT, Xie A, Qi Y, Lindner JR. Detection of antecedent myocardial ischemia with multiselectin molecular imaging. J Am Coll Cardiol 2012;60:1690-7.
66. Mor-Avi V, Sugeng L, Lang RM. Real-time 3-dimensional echocardiography: an integral component of the routine echocardiographic examination in adult patients? Circulation 2009;119:314-29.
67. Mannaerts HF, van der Heide JA, Kamp O, Stoel MG, Twisk J, Visser CA. Early identification of left ventricular remodelling after myocardial infarction, assessed by transthoracic 3D echocardiography. Eur Heart J 2004;25:680-7.
68. Huttin O, Zhang L, Lemarié J, Mandry D, Juillière Y, Lemoine S, et al. Global and regional myocardial deformation mechanics of microvascular obstruction in acute myocardial infarction: a three dimensional speckle-tracking imaging study. Int J Cardiovasc Imaging 2015;31: 1337-46.
69. Galderisi M, Esposito R, Schiano-Lomoriello V, Santoro A, Ippolito R, Schiattarella P, et al. Correlates of global area strain in native hypertensive patients: a three-dimensional speckle-tracking echocardiography study. Eur Heart J Cardiovasc Imaging 2012;13:730-8.
70. Abate E, Hoogslag GE, Antoni ML, Nucifora G, Delgado V, Holman ER, et al. Value of three-dimensional speckle-tracking longitudinal strain for predicting improvement of left ventricular function after acute myocardial infarction. Am J Cardiol 2012;110:961-7.
71. Nijland F, Kamp O, Verhorst PM, de Voogt WG, Visser CA. Early prediction of improvement in ejection fraction after acute myocardial infarction using low dose dobutamine echocardiography. Heart 2002;88:592-6.
72. Hoffer EP, Dewé W, Celentano C, Piérard LA. Low-level exercise echocardiography detects contractile reserve and predicts reversible dysfunction after acute myocardial infarction: comparison with low-dose dobutamine echocardiography. J Am Coll Cardiol 1999;34:989-97.
73. Gong L, Li D, Chen J, Wang X, Xu T, Li W, et al. Assessment of myocardial viability in patients with acute myocardial infarction by two-dimensional speckle tracking echocardiography combined with low-dose dobutamine stress echocardiography. Int J Cardiovasc Imaging 2013;29:1017-28.
74. Iwakura K, Okamura A, Koyama Y, Date M, Higuchi Y, Inoue K, et al. Automated assessment of myocardial viability after acute myocardial infarction by global longitudinal peak strain on low-dose dobutamine stress echocardiography. Circ J 2010;74:2158-65.
75. Sicari R, Landi P, Picano E, Pirelli S, Chiarandà G, Previtali M, et al. Exercise- electrocardiography and/or pharmacological stress echocardiography for non-invasive risk stratification early after uncomplicated myocardial infarction. A prospective international large scale multicentre study. Eur Heart J 2002;23:1030-7.
76. Desideri A, Fioretti PM, Cortigiani L, Gregori D, Coletta C, Vigna C, et al. Cost of strategies after myocardial infarction (COSTAMI): a multicentre, international, randomized trial for cost-effective discharge after uncomplicated myocardial infarction. Eur Heart J 2003;24:1630-9.
77. Desideri A, Fioretti PM, Cortigiani L, Trocino G, Astarita C, Gregori D, et al. Pre-discharge stress echocardiography and exercise ECG for risk stratification after uncomplicated acute myocardial infarction: results of the COSTAMI-II (cost of strategies after myocardial infarction) trial. Heart 2005;91:146-51.
78. van Loon RB, Veen G, Baur LH, Kamp O, Bronzwaer JG, Twisk JW, et al. Improved clinical outcome after invasive management of patients with recent myocardial infarction and proven myocardial viability: primary results of a randomized controlled trial (VIAMI-trial). Trials 2012; 13:1.

APPENDIX

Supplemental Table 1 Baseline characteristics of the study population of studies assessing prognostic value of conventional echocardiographic indexes after AMI

First author (echo parameter)	No. of Pts	Age	Male	pMI	pHF	HT	SH	DM	HL	STEMI	NSTEMI	ANT	INF	LAT	TT	PTCA	CABG
Verma[7] (LVM, RWT)	603	ND	411 (68.1)	168 (27.8)	96 (15.9)	339 (56.2)	ND	137 (22.7)	ND	ND	ND	358 (59.3)	200 (33.1)	ND	233 (38.6)	106 (17.6)	10 (1.6)
Brand[8] (MAPSE)	271	69.1 (11.8)	188 (69.4)	83 (30.9)	20 (7.5)	86 (31.9)	82 (30.5)	56 (20.7)	ND	ND	ND	107 (41.5)	ND	ND	71 (26.5)	ND	ND
Samad[9] (TAPSE)	194	64 (10)	143 (73.8)	0 (0)	ND	62 (31.9)	ND	26 (13.4)	ND	ND	ND	93 (47.9)	101 (52.1)	0 (0)	149 (76.8)	ND	ND
Schwammenthal[11] (LV EF)	417	ND	369 (88.5)	48 (11.5)	10 (2.3)	87 (20.8)	ND	55 (13.1)	ND	ND	ND	90 (21.6)	93 (22.3)	ND	ND	ND	ND
Shiga[12] (LVEF)	4,122	66.4 (11.9)	3,039 (73.7)	573 (13.9)	ND	2,352 (57)	2,348 (56.9)	1,437 (34.8)	1,737 (42.1)	ND	ND	ND	ND	ND	ND	3208 (77.8)	151 (3.7)
Nicolosi[14] (LV EF, EDV, ESV)	8,606	ND	6,946 (80.7)	2,238 (13.2)	ND	4,951 (29.2)	ND	2,543 (15)	ND	ND	ND	2,512 (29.2)	3,029 (35.2)	ND	6273 (72.9)	ND	ND
Carluccio[15] (WMSI)	144	ND	110 (76.4)	0 (0)	0 (0)	70 (48.6)	75 (52)	25 (17.3)	66 (45.8)	ND	ND	80 (55.6)	47 (32.6)	17 (11.8)	144 (100)	ND	ND
Møller[16] (WMSI)	767	73	572 (62)	169 (22)	ND	417 (54)	ND	176 (23)	ND	376 (49)	391 (51)	346 (47)	ND	ND	143 (19)	340* (44)	340* (44)
Antoni[17] (RVFAC)	621	60 (12)	486 (78)	45 (7)	N/S	190 (31)	313 (51)	61 (10)	125 (20)	ND	ND	ND	217 (35)	ND	0 (0)	621 (100)	ND
Beinart[20] (LAVi)	395	62 (12)	311 (78.8)	82 (20.7)	ND	160 (40.5)	147 (37)	91 (23)	137 (34.6)	ND	ND	160 (40.5)	ND	ND	173 (43.7)	159 (40.2)	26 (6.5)
Sakaguchi[21] (LAVi)	205	65 (ND)	173 (84.4)	0 (0)	ND	128 (62.4)	118 (57.6)	71 (34.6)	103 (50.2)	124 (60.5)	81 (39.5)	113 (55.1)	ND	ND	ND	165 (80.5)	10 (4.9)
Hsiao[22] (LAd)	521	65 (14)	414 (79.5)	33 (6.3)	ND	316 (60.7)	271 (52)	188 (36.1)	ND	393 (75.5)	128 (24.5)	240 (46.1)	134 (25.7)	19 (3.6)	0 (0)	ND	ND
Temporelli[23] (DT)	571	61 (11)	471 (82)	63 (11)	ND	162 (28)	ND	83 (14)	ND	ND	ND	174 (30)	216 (38)	ND	405 (71)	ND	ND
Møller[24] (RFP)	3,396	64	2,512 (74)	397 (11.6)	ND	757 (22.2)	743 (21.8)	349 (10.2)	229 (6.7)	ND	ND	1213 (35.7)	ND	ND	ND	ND	ND
Poulsen[25] (MPI†)	64	62 (10)	48 (75)	ND	ND	12 (19)	ND	5 (8)	ND	ND	ND	24 (38)	ND	ND	44 (69)	ND	ND
López-Pérez[28] (MR)	1,036	ND	828 (79.9)	76 (7.3)	ND	470 (45.3)	411 (39.6)	172 (16.6)	410 (39.5)	1,036 (100)	0 (0)	448 (43)	518 (50)	ND	0 (0)	1036 (100)	ND
Carrabba[29] (MR)	184	64.4 (12)	143 (78)	ND	ND	71 (39)	ND	32 (17)	52 (28)	184 (100)	(0)	89 (48)	ND	ND	0 (0)	184 (100)	ND
Møller[31] (PASP)	536	ND	296 (52.2)	126 (23.5)	ND	286 (53.3)	233 (43.4)	130 (24.2)	ND	245 (45.7)	291 (54.3)	247 (46)	ND	ND	88 (16,4)	321* (59.8)	321* (59.8)
Mutlak[32] (PASP)	1,054	ND	750 (71)	243 (23)	0 (0)	578 (54.8)	199 (18.8)	305 (28.9)	ND	872 (82.7)	182 (17.3)	461 (43.7)	ND	ND	ND	512* (48.5)	512* (48.5)
Hillis[33] (E/e′)	250	68 (13)	155 (62)	50 (20)	ND	142 (57)	57 (23)	56 (22)	104 (42)	120 (48)	130 (52)	106 (42)	ND	ND	39 (16)	160 (64)	18 (7)

ANT, Anterior myocardial infarction; *CABG*, coronary artery bypass grafting; *DM*, diabetes mellitus; *E/e′*, ratio of transmitral E velocity to early diastolic velocity of the mitral annulus; *HL*, hyperlipidemia; *HT*, hypertension; *INF*, inferior myocardial infarction; *LAd*, left atrial distensibility; *LAT*, lateral myocardial infarction; *LVM*, left ventricle mass; *MPI*, myocardial performance index; *ND*, not determined; *pHF*, prior heart failure; *pMI*, prior myocardial infarction; *PTCA*, percutaneous transluminal coronary angioplasty; *PTS*, patients; *SH*, smoke habit; *TT*, thrombolytic therapy.

Studies are in the same order in which they are cited in the text. Data are expressed as number (*n*) and percentage (%) and as years and SD.

*PTCA and CABG were counted together.

†Assessed by standard Doppler.

Supplemental Table 2 Baseline characteristics of study population of studies assessing advanced echo indexes for risk stratification after AMI

First author (echo parameter)	No. of Pts	Age	Male	pMI	pHF	HT	SH	DM	HL	STEMI	NSTEMI	ANT	INF	LAT	TT	PTCA	CABG
Antoni[17] (RV strain*)	621	60 (12)	486 (78)	45 (7)	ND	190 (31)	313 (51)	61 (10)	125 (20)	ND	ND	ND	217 (35)	ND	0 (0)	621 (100)	ND
Biering-Sørensen[35] (s′+e′+a′†)	373	ND	281 (75)	16 (4)	ND	120 (32.1)	193 (51.7)	32 (8.5)	29 (7.7)	373 (100)	0 (0)	178 (47.7)	151 (40.4)	41 (11)	0 (0)	373 (100)	ND
Biering-Sørensen[36] (MPI‡)	386	ND	289 (74.8)	17 (4.4)	ND	124 (32.1)	196 (50.7)	32 (8.2)	31 (8)	386 (100)	0 (0)	ND	ND	ND	0 (0)	386 (100)	ND
Sjøli[37] (GLS§)	77	64 (12)	59 (77)	0 (0)	0 (0)	26 (34)	39 (51)	6 (8)	ND	77 (100)	0 (0)	35 (46)	42 (54)	0 (0)	77 (100)	66 (85.7)	7 (9)
Park[43] (LS of infarct zone *)	50	56 (13)	41 (82)	0 (0)	ND	24 (48)	25 (50)	12 (24)	11 (22)	ND	ND	50 (100)	0 (0)	0 (0)	6 (12)	50 (100)	ND
Antoni[44] (GLS*)	659	60 (12)	517 (79)	4.9 (7.4)	ND	203 (31)	331 (50)	60 (10)	131 (20)	ND	ND	311 (48)	ND	ND	0 (0)	659 (100)	ND
Ersbøll[45] (GLS*)	548	63.2 (11.7)	393 (71.6)	ND	33 (6)	253 (46.2)	368 (66.7)	75 (13.7)	ND	370 (67.5)	178 (32.5)	ND	ND	ND	103 (18.8)	436 (79.6)	44 (8.1)
Ersbøll[46] (GLS*)	849	61.9 (12)	616 (72.5)	89 (10.4)	28 (3.2)	373 (43.9)	588 (69.2)	100 (11.7)	ND	576 (67.8)	272 (32)	ND	ND	ND	ND	672 (79.1)	63 (7.4)
Munk[47] (GLS*)	576	ND	446 (77.4)	71 (12.3)	ND	195 (33.8)	323 (56)	59 (10.2)	ND	576 (100)	0 (0)	246 (41.6)	ND	ND	0 (0)	576 (100)	ND
Ersbøll[48] (GLS*)	988	62.6 (12.1)	714 (72.2)	122 (12.3)	ND	454 (45.9)	ND	129 (13)	ND	677 (68.5)	331 (31.5)	ND	ND	ND	ND	779 (78.8)	83 (8.4)
Ersbøll[42] (E/e′sr*)	1048	63 (12)	766 (73)	132 (12.5)	ND	490 (46.7)	728 (69.4)	143 (13.6)	ND	711 (67.8)	337 (32.2)	ND	ND	ND	ND	818 (78)	92 (8.7)
Ersbøll[53] (PALS*)	843	62.1 (11.8)	612 (72.5)	102 (12)	41 (4.8)	370 (43.8)	578 (68.5)	107 (12.6)	ND	583 (69.2)	260 (30.8)	ND	ND	ND	ND	671 (79.6)	72 (8.5)
Katayama[58] (diastolic CFV DT)	59	ND	39 (66)	0 (0)	ND	28 (47.4)	27 (45.7)	22 (37.2)	35 (59.3)	ND	ND	59 (100)	0 (0)	0 (0)	55 (93.2)	59 (100)	ND
Meimoun[59] (CFVR)	51	59 (13)	34 (66.6)	ND	ND	20 (39.2)	22 (43.1)	17 (33.3)	27 (52.9)	51 (100)	0 (0)	ND	ND	ND	0 (0)	51 (100)	ND
Dwivedi[61] (CDI‖)	95	61 (10.8)	72 (76)	0 (0)	ND	31 (32)	40 (42)	25 (26)	42 (44)	95 (100)	0 (0)	76 (71)	ND	ND	86 (87)	65¶ (68)	65¶ (68)

ANT, Anterior myocardial infarction; *CABG*, coronary artery bypass grafting; *CFV*, coronary flow velocity; *DM*, diabetes mellitus; *E/e′*sr, ratio of transmitral E velocity to global diastolic strain rate; *HL*, hyperlipidemia; *HT*, hypertension; *INF*, inferior myocardial infarction; *LAT*, lateral myocardial infarction; *MPI*, myocardial performance index; *ND*, not determined; *PALS*, peak atrial longitudinal strain; *pHF*, prior heart failure; *pMI*, prior myocardial infarction; *PTCA*, percutaneous transluminal coronary angioplasty; *Pts*, patients; *s′*, longitudinal systolic myocardial velocity; *SH*, smoke habit; *TT*, thrombolytic therapy.

Studies are in the same order in which they are cited in the text. Data are expressed as number (*n*) and percentage (%) and as years and SD.

*Assessed by STE.

†Assessed by CTDI.

‡Assessed by color tissue Doppler M-mode of mitral leaflet.

§Assessed by TDI.

‖Assessed by MCE.

¶PTCA and CABG were counted together.