Echocardiography in coronary artery disease

Regina Votavová a, Anna Linhartová b, Josef Kořínek a, Josef Marek a, Aleš Linhart a, *

a 2nd Department of Medicine – Department of Cardiovascular Medicine, General University Hospital in Prague, First Medical Faculty, Charles University, Prague, Czech Republic
b Faculty of Arts, Charles University in Prague, Czech Republic

ARTICLE INFO

Article history:
Received 25 August 2015
Received in revised form
11 September 2015
Accepted 12 September 2015
Available online 17 October 2015

Keywords:
Echocardiography
Myocardial function
Coronary artery disease
Ischemic mitral regurgitation

ABSTRACT

Coronary artery disease (CAD) is one of the major causes of morbidity and mortality. Imaging techniques represent the key method for disease extent and severity assessment and evaluation of hemodynamic complications. In skilled hands the method provides useful information for clinical management and prognosis assessment. Complex evaluation brings information about global and regional myocardial function, myocardial viability, ischemic mitral regurgitation, and about development of complications such as left ventricular thrombus formation, myocardial rupture and pericardial effusion. The main drawback of echocardiography is the limited echogenicity of many patients and its undeniable operator-dependence. However, the possibility of bringing the echocardiographic imaging to the bedside of our patients makes the method essential and its knowledge indispensable for all cardiologists.

© 2015 The Czech Society of Cardiology. Published by Elsevier Sp.z.o.o. All rights reserved.

Contents

Introduction .......................................................... e409
Evaluation of the regional systolic function .......................................................... e409
The evaluation of global systolic function .......................................................... e411
Evaluation of left ventricular filling .......................................................... e412
Ischemic mitral regurgitation .......................................................... e413
Complications of acute myocardial infarction .......................................................... e416
Conflict of interest .......................................................... e417
Ethical statement .......................................................... e417
Acknowledgements .......................................................... e417
References .......................................................... e417

* Corresponding author at: 2nd Department of Medicine – Department of Cardiovascular Medicine, General University Hospital in Prague, First Faculty of Medicine, Charles University in Prague, U Nemocnice 2, 128 08 Prague 2, Czech Republic. Tel.: +420 2 24962634; fax: +420 2 24912154.
E-mail address: alinh@lf1.cuni.cz (A. Linhart).
http://dx.doi.org/10.1016/j.crvasa.2015.09.006
0010-8650/© 2015 The Czech Society of Cardiology. Published by Elsevier Sp.z.o.o. All rights reserved.
Introduction

Coronary artery disease (CAD) is one of the major causes of morbidity and mortality. Imaging techniques represent the key method for disease extent and severity assessment and evaluation of hemodynamic complications. Echocardiography is one of the most useful imaging methods due to its availability, ease of use, price, capacity to serve as bedside technique and repeatability. In this review we summarize fundamental principles of echocardiographic imaging recommended for the assessment of patients presenting with both acute and chronic manifestations of CAD.

Evaluation of the regional systolic function

Major consequences of ischemia include an impairment of regional systolic contractility. If severe ischemia persists myocardial necrosis develops followed by scarring which affects the regional function permanently. The regional myocardial function is usually assessed only visually by evaluating wall thickening and endocardial motion of myocardial segments. It is widely recognized that myocardial movements may be caused by adjacent segment tethering or overall LV displacement. It seems therefore preferable that regional deformation should be analyzed by using methods that are at least partially independent of tethering such as speckle tracking though keeping in mind that even the deformation may be passive [1].

The usual practice is a grading of regional myocardial function depending on the quality of contraction. The recommended scoring based on the current guidelines is as follows: (1) for normal or hyperkinetic, (2) for hypokinetic (reduced thickening), (3) akinetic (absent or negligible thickening), and (4) dyskinetic (systolic thinning or stretching or aneurysm). An aneurysm is characterized by a focal dilatation and thinning (remodeling) of the myocardial wall with either akinetic or dyskinetic systolic deformation (Fig. 1). In the past, a separate grade 5 was assigned to aneurysm [2].

An approach is still being kept by many echocardiographic laboratories. Based on the segmental motion evaluation a Wall motion score index (WMS) can be calculated as a sum of all scores divided by the number of visualized segments.

The segmentation of the left ventricular cavity may vary between imaging techniques and echocardiographic laboratories. A 16-segments model was proposed both for WMS calculations at basal conditions and for evaluation of stress echocardiography (Fig. 2). However, the recent guidelines suggested to use a 17-segments model where the 17th segment is represented by the “apical cap”. Therefore the apical segment is now divided into five segments instead of four used in the past. This approach is recommended only for perfusion studies as it allows a better comparison with other imaging modalities as single photon emission tomography (SPECT), positron emission tomography (PET) or cardiac magnetic resonance (CMRI) [1]. However, this segmentation should not be used for regional motion assessment and deformation (strain) studies where the 16-segments model is still the method of choice due to the fact that endocardial excursion and thickening of the apical cap are imperceptible.

The use of deformation imaging, nowadays mostly using the strain and strain rate derived from speckle tracking, should allow less subjective evaluation of myocardial contraction as compared to simple visual assessment. The speckle tracking echocardiography replaced techniques based on tissue Doppler imaging (TDI) originally used for myocardial motion and

![Fig. 1](image1.png)  
**Fig. 1** – Aneurysm of the inferior wall (arrows) visualized by transthoracic echocardiography. LV = left ventricle, LA = left atrium.

![Fig. 2](image2.png)  
**Fig. 2** – The classical segmentation of the left ventricular walls using 16-segments. Ao = aorta, LA = left atrium, IVS = interventricular septum (for mid and basal portion often labeled as inferoseptum), ANT = anterior wall, POST = posterior or inferolateral wall, INF = inferior wall, LAT = lateral or anterolateral wall.  
Adapted from Ref. [2].
deformation assessment. As TDI is deriving the strain and strain rate data from tissue velocities it is affected by passive translational movements and tethering of the myocardial segments, making the discrimination of akinetic segments difficult. Another phenomenon complicating the TDI-based evaluation is the fact that myocardial velocities are decreasing from base to the apex. Moreover, TDI as all Doppler-based techniques suffers from angle dependence making the evaluation of the angulated segments nearly impossible [3].

The strain and strain rate derived from two-dimensional (2D) echocardiography are based on computer algorithms tracking the movement of so-called “speckles” – clusters of natural acoustic markers generated within the myocardium by an interaction with ultrasonic waves. These techniques are independent of the ultrasound beam propagation angle and allow evaluation of longitudinal (from apical views), radial (from short-axis and apical views), and circumferential (from short-axis views) strains. Nevertheless, the translational movements of the heart may still induce an error when the heart shifts out of the imaging plane during the cardiac cycle. The reproducibility is better with 2D than Doppler techniques. It should be kept in mind that the technique requires higher frame rates (50–70/s) than those that are used in routine imaging and a good image quality is needed for accurate tracking [3].

In the normal myocardium strains and strain rates are nearly homogeneously distributed and even subtle changes may suggest myocardial contractile impairment. Several studies proposed that particularly the longitudinal strain decrease correlates with the presence of decreased coronary perfusion in segments that appear visually normal (Fig. 3) [4].

The deformation imaging allows identification of a presence of longitudinal shortening or radial thickening of the myocardium after aortic valve closure, the so-called post-systolic shortening or thickening (PSS), or tardokinesis (Fig. 3). Small extent of PSS may occur even under physiological conditions in some LV segments and myocardial layers. However, mode pronounced PSS is suggestive of ischemia or scarring of the myocardium. Its development during a stress test is considered as an indicator of regional ischemia. The deformation analysis may be also useful in evaluating the myocardial viability as stunned myocardium displays reduced systolic strain rates that improve with low-dose dobutamine infusion. There is an ongoing debate whether the appearance of PSS suggests scarring or whether it may be an active phenomenon implicating viability [5].

Unfortunately, regional deformation measurements may vary depending on the myocardial region being investigated, the measurement methodology used, and a sample volume definition. Moreover, although the patterns of strain and strain rate curves may be similar, the quantitative strain measurements derived from the software used by different ultrasound machine manufacturers may vary and are not directly comparable. Therefore in spite of the theoretical advantages over the visual assessments, the universally applicable normative data for different myocardial segments are not recommended [1].

Fig. 3 – 2D speckle tracking on the apical two-chamber view showing a significant decrease of longitudinal strain within the basal inferior wall (yellow segment, arrow) with a clear post-systolic deformation (arrow) on the yellow curve from the same segment.
The evaluation of global systolic function

According to current recommendations, global systolic functional parameters should be based on 2D or 3D echocardiographic imaging. The formulas using linear measurements, such as fractional shortening, Teichholz and Quinones methods, are generally not recommended [1]. Their use may be particularly misleading in CAD patients with regional functional contractility impairments. Although 3D echocardiography begins to change the clinical practice in many ways, the 2D approach is still prevailing. Left ventricular volumes should be measured from apical four and two chamber views. During scanning attention should be paid to maximize cavity areas to avoid LV foreshortening (Fig. 4). Endocardial border delineations may be improved by adapting focus number and position. In subjects with suboptimal LV visualization the use of echocardiographic contrast agents may be considered. The most commonly used method for 2D volume calculations is the Simpson’s bi-plane method of discs summation. When evaluating the cavity size, the body build of the patients should be always considered. Although criticized, the traditional approach of indexing to body surface area (BSA) is still recommended. Upper normal limits for two-dimensional echocardiographic end-diastolic volumes are proposed at 74 mL/m² for men and 61 mL/m² for women. In contrast to previous recommendations proposing a uniform lower limit for ejection fraction (EF) at 55%, current guidelines suggest that EF of <52% in a man and <54% in a women are suggestive of abnormal LV systolic function [1,2].

In addition to regional myocardial strain evaluation, a global longitudinal strain (GLS) can be measured. It has been shown that GLS is compromised in patients with coronary artery disease. The decrease in its absolute value has also prognostic implications for development of heart failure and death [1,3]. Therefore the GLS has been proposed as a useful index for evaluation of global myocardial function. The parameter is derived from standard apical views (four-chamber, two-chamber and long-axis view) analyzing and averaging peak longitudinal strain in all evaluable segments. A GLS ≥ -17.4% provided a reasonable sensitivity and specificity in identifying patients with CAD [6]. Unfortunately as for most strain indices, there is a disparity in results depending on the machine and software used. In addition, GLS calculation could be based on endocardial, midwall, or average deformation. Due to this confusion the current recommendations on

Fig. 4 – Bi-plane disc summation method (modified Simpson’s rule) for left ventricular volumes and ejection fraction calculations.
chamber quantification give only weak guidance indicating that normal values of GLS for a healthy individual should be around –20% and summarizing the literature in supplementary data tables. The attempts to standardize the measurements and algorithms are in progress, yet still far from reaching the clinical applications [7].

An occlusion of right coronary artery and in some cases of the left circumflex artery may lead to right ventricular (RV) myocardial infarction. RV involvement is rarely isolated and most often associates with inferior myocardial infarction (MI). Wall motion abnormalities constitute the most sensitive and specific echocardiographic findings in the RV infarction. The most common site of involvement is the posterior (inferior) wall, though anterior and lateral walls may be affected as well [8]. Abnormalities in wall motion may persist after evolution and normalization of the hemodynamic alterations of acute RV infarct. In some patients RV aneurysms may develop. In others a paradoxical interventricular septum motion becomes obvious [9].

The acute impairment of regional wall motion leads to a significant decrease of RV systolic performance as assessed by either tricuspid annular plane systolic excursion (TAPSE usually <16 mm), S wave velocity measured by pulsed wave TDI on the tricuspid annulus (usually <10 cm/s), or fractional area change (FAC <35%). The two-dimensional approach is recommended since TAPSE may be falsely low especially in patients after coronary bypass surgery. The endocardial tracings of the RV are performed at end-diastole and end-systole from apical four-chamber view. FAC is calculated as (end-diastolic area – end-systolic area)/end-diastolic area × 100 (Fig. 5). Normal values are above 35%. A decrease of RV contractility may be demonstrated also by using speckle tracking methods and analyzing RV deformation. In addition to RV dysfunction a cavity dilatation may be observed. RV infarction is often associated with rapidly developing diastolic dysfunction leading to vena cava dilatation and loss of respiratory variations (Fig. 6).

As compared to pulmonary embolism, the RV overload in acute right ventricular myocardial infarction does usually not induce interventricular septal displacement (D-shape of the LV); in case of tricuspid regurgitation the gradients are usually very low. Pulmonary embolism was referred to be associated with the so-called McConnell sign showing a decrease in free wall contraction with preserved apical function [10]. However, it was demonstrated that the sign is not specific and may be seen also in patients with RV myocardial infarction [11].

In addition to measurements of left and right regional and global ventricular function, echocardiography allows a reasonably precise evaluation of cardiac output. The most clinically accepted technique is combining the measurement of left ventricular outflow tract dimension (LVOT) (usually obtained in systole from the parasternal long axis view) and velocity time integral (VTI_{LVOT}) of the flow through LVOT measured from apical views (Fig. 7). Cardiac output is then calculated as \pi \times (LVOT^2/4) \times VTI_{LVOT} \times heart rate. This approach is very useful particularly in acute settings for evaluation of the overall hemodynamic situation.

### Evaluation of left ventricular filling

Ischemia and CAD do not impair only systolic function but also affects diastolic function as well. Thus the recognition of diastolic dysfunction or worsening of diastolic function can be indicative of CAD presence at rest even without the systolic dysfunction or obvious wall motion abnormalities. Over the last two decades it became obvious that many patients have signs and symptoms of heart failure without a clear reduction in the LV EF. In addition, the presence of severe diastolic dysfunction was shown to be an independent prognostic factor for mortality of heart failure patients regardless the EF [12]. Echocardiography allows evaluation of LV filling using Doppler measurements of transmittal flow and pulsed wave TDI recordings of mitral annular movements. The traditional classification based on this approach is shown in Fig. 8. In cases of doubt, the evaluation should be completed by
analyzing pulmonary venous inflow pattern, using Valsalva maneuvers and considering the left atrial size and volume [13]. In acute settings, the changes of LV filling may heavily depend on loading conditions. However, in stable patients the flow pattern predicts the outcome. The prognosis of the patients depends also on LA volumes which should be preferred over linear atrial measurements and performed by using two perpendicular apical views applying either area-length or disc summation methods (Fig. 9) [14]. The measurements should be done in end-systole or on the frame preceding mitral valve opening. The tracing of LA is closed at the level of mitral annulus and LA appendage or pulmonary vein inflows are not included [1].

Ischemic mitral regurgitation

The pathophysiology of ischemic mitral regurgitation (IMR) is complex. In most cases the IMR develops as a consequence of myocardial infarction inducing LV cavity dilatation and systolic dysfunction. IMR is encountered in as many as 19% of MI survivors and predicts an unfavorable long-term prognosis. In most cases it is caused by maladaptive LV remodeling after the MI resulting in apical and posterior papillary muscle displacement. This leads to mitral leaflet tethering and to an inappropriate coaptation of the mitral valve leaflets. The cavity dilatation contributes by secondary mitral annular dilatation and loss of annular contraction. In addition, global LV dysfunction is associated with a decrease in closing forces necessary for the appropriate mitral valve closure. Mitral regurgitation, in turn, leads to LV volume overload resulting in further cavity dilatation which exacerbates the vicious circle by aggravating the mitral regurgitation. It should be noted that although majority of patients have normal mitral leaflets and subvalvular apparatus, in some cases organic changes such as leaflet thickening, shortening, annular calcification and other changes may also be present [15].

Echocardiography allows quantitation of IMR and detailed assessment of underlying mechanisms. The quantitation should not be based solely on semi-quantitative evaluation of regurgitant jet area. The measurement of vena contracta width seems more accurate. However, in IMR the jet orifice is often asymmetric and measurements from different views may substantially differ. A mean value of >8 mm obtained from several views seems to identify a severe mitral insufficiency regardless of the etiology (Fig. 10) [16].

A quantitative analysis based either on Doppler volumetric method or proximal isovelocity surface area principle (PISA) is preferable (Fig. 11). Both methods have several limitations. The
comparison of antegrade stroke volume measured at the level of LVOT to antegrade transmural flow serves for calculation of regurgitant volume. Unfortunately, the estimation of LVOT area is often inaccurate and mitral valve orifice has an elliptic shape dynamically changing throughout the diastole. Due to these limitations and its time consuming character the method is rarely used. On the other hand, a simple comparison of mitral to aortic VTI may be helpful. Severe MR should be suspected whenever their ratio exceeds 1.4 [17].

Unfortunately, the PISA method may lead to underestimation or overestimation of the regurgitant volume and orifice as well. First, PISA radius is changing during systole. Ideally PISA should be measured several times and averaged. However, this approach is only rarely used in clinical practice. The regurgitant orifice is seldom circular and therefore the PISA itself is seldom hemispheric. Most PISA resemble rather a hemiellipse. Since algorithms incorporated in the majority of echocardiographic machines are forcing the operators to trace PISA as a hemisphere, regurgitant flow is often underestimated. The most important drawback of PISA in IMR is the fact that many patients have extremely distorted and/or multiple regurgitant jets not suitable for PISA measurement. It should be noted that simple addition of several PISA volumes has not been validated. Last but not least, IMR may have a significant functional/dynamic component and its magnitude may then depend on loading conditions and patient’s blood pressure variations. In case of doubt exercise echocardiography using a dedicated stress table with semisupine bicycle ergometer is recommended. An increase of ERO of more than 13 mm² is associated with adverse outcomes [16,17].

Fig. 9 – Left atrial volume calculation using the bi-plane approach and a disc summation method.

Fig. 10 – Measurement of vena contracta width from bi-plane orthogonal views obtained during transesophageal echocardiography.
Severity grading of IMR differs from organic mitral regurgitation. Severe IMR is defined by regurgitant volume >30 mL and effective regurgitant orifice (ERO) >20 mm². The reason for defining the threshold of severity on values that are twice lower as compared to organic mitral insufficiency is based on the evidence of adverse prognostic impact of IMR of such magnitude. Currently, there is an expert consensus that mitral valve surgery should be offered to patients with severe IMR undergoing surgical revascularization (class of recommendation I, level of evidence C) and may be considered as an isolated surgical procedure (class of recommendation IIb, level of evidence C). The indication of surgery in patients with moderate IMR (ERO >10 mm² but <20 mm²) is controversial [16-18].

A detailed assessment of mitral anatomy is recommended. First, it is important to determine regional contraction abnormalities of the LV, its dilatation (volume and diameter) and remodeling (eccentricity/sphericity index). IMR is characterized by an apical displacement of the coaptation point (Fig. 12) and an increased tenting area (Fig. 13). This is often due to an increasing distance between the posterior papillary muscle and the fibrous annulus (Fig. 14). In addition, mitral annulus enlarges and becomes disproportionate compared to the anterior leaflet length. Annular dilatation is present when the ratio between the annular diameter (measured from parasternal long axis in systole) to anterior mitral leaflet (measured in diastole) is exceeding 1.3 or in case the annulus exceeds 35 mm. Several other parameters have been suggested for evaluation of IMR mechanisms including leaflet angle measurements and posterior displacement of papillary muscles assessments. Before any indication of surgery it is important to determine the origin of the jet and its direction.

Fig. 11 – Measurement of regurgitant mitral flow volume and effective regurgitant orifice by using the PISA method. Of note, the variance signal of the color Doppler is switched off and zero shift method applied for the correct PISA radius measurement.

Fig. 12 – An example of ischemic mitral regurgitation (right panel) in a patient with severe systolic leaflet motion restriction documented by an increased coaptation distance (CD) from the mitral annular plane.
Based on these measurements, several parameters represent unfavorable characteristics for mitral valve repair and its subsequent durability. These include mitral anatomy – coaptation distance of more than 1 cm and tenting area exceeding 2.5–3 cm² and local or global LV remodeling (inter-papillary muscle distance >20 mm, posterior papillary muscle to fibrosa distance >40 mm, posterolateral wall akinesia end excessive LV dilatation (diameter >65 mm, sphericity index >0.7) [17].

Complications of acute myocardial infarction

Echocardiography is the method of choice for identifying acute or subacute complications of myocardial infarction. These may include papillary muscle rupture (Fig. 15), with severe acute mitral regurgitation, free wall rupture with tamponade or pseudoaneurysm formation (Fig. 16), or interventricular septum rupture (Fig. 17). In the era of effective anticoagulation and antiaggregation therapy a thrombus formation is relatively rare. However in a small subset of patients its formation may be observed on the endocardial layers affected by necrosis (Fig. 18). Last but not least, echocardiography is very helpful in identifying patients with pericarditis developing during the subacute phase of MI. Before the era of reperfusion presence of some pericardial effusion was reported to be as high as 28%. Currently only about 4% patients with acute MI treated by PCI have some degree of pericardial effusion (Fig. 19). The finding develops usually within 4 days from admission. Cardiac tamponade resulting from post-MI pericarditis is rare. However an effusion exceeding 10 mm in parasternal long axis was reported to be associated with some risk of free wall myocardial rupture [19,20].
In conclusion, echocardiography is an excellent tool for a complex and comprehensive evaluation of structural and hemodynamic changes induced by chronic or acute CAD. In skilled hands the method provides useful information for clinical management and prognosis assessment. The main drawback of echocardiography remains in the limited echogenicity of many patients and its undeniable operator-dependence. However, the possibility of bringing the echocardiographic imaging to the bedside of our patients makes the method essential and its knowledge indispensable for all cardiologists.

Conflict of interest

No conflict of interest.

Ethical statement

I declare, on behalf of all authors, that the research was conducted according to Declaration of Helsinki.

Acknowledgement

The study was supported by PRVOUK-P3S/LF1/5.

Funding body

None.

REFERENCES


[2] R.M. Lang, M. Bierig, R.B. Devereux, et al., Chamber Quantification Writing Group, American Society of Echocardiography’s Guidelines and Standards Committee; European Association of Echocardiography, Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology, Journal of the American Society of Echocardiography 18 (2005) 1440–1463.


12. J.J. McMurray, S. Adamopoulos, S.D. Anker, et al., ESC Committee for Practice Guidelines, ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC, European Heart Journal 33 (2012) 1787–1847.


