European Heart Journal – Cardiovascular Imaging (2015) **16**, 147–153
doi:10.1093/ehjci/jeu299

POSITION PAPER

Appropriateness criteria for cardiovascular imaging use in heart failure: report of literature review

Madalina Garbi[1], Theresa McDonagh[1], Bernard Cosyns[2], Chiara Bucciarelli-Ducci[3], Thor Edvardsen[4], Anastasia Kitsiou[5], Koen Nieman[6], and Patrizio Lancellotti[7,8]* On behalf of the EACVI Imaging Task Force

[1]King's Health Partners, King's College Hospital NHS Foundation Trust, London, UK; [2]Universitair Ziekenhuis van Brussel, CHVZ and ICMI Laboratory, CHIREC, Brussels, Belgium; [3]Bristol NIHR Cardiovascular Biomedical Research Unit (BRU), Bristol Heart Institute, University Hospitals Bristol NHS Foundation Trust, University of Bristol, Bristol, UK; [4]Department of Cardiology, Oslo University Hospital, Rikshospitalet and University of Oslo, Norway; [5]Cardiology Department, Sismanoglio Hospital, Athens, Greece; [6]Departments of Cardiology and Radiology, Erasmus MC, Rotterdam, The Netherlands; [7]GIGA Cardiovascular Sciences, Heart Valve Clinic, Department of Cardiology, University of Liège Hospital, University Hospital SartTilman, Liège, Belgium; and [8]GVM Care and Research, Bologna, Italy

Received 17 November 2014; accepted after revision 24 November 2014; online publish-ahead-of-print 30 December 2014

The Imaging Task Force appointed by the European Society of Cardiology (ESC) and the European Association of Cardiovascular Imaging (EACVI) identified the need to develop appropriateness criteria for the use of cardiovascular imaging in heart failure as a result of continuously increasing demand for imaging in diagnosis, definition of aetiology, follow-up, and treatment planning. This article presents the report of literature review performed in order to inform the process of definition of clinical indications and to aid the decisions of the appropriateness criteria voting panel. The report is structured according to identified common heart failure clinical scenarios.

Keywords Heart failure • Appropriateness criteria • Imaging • Echocardiography • Cardiac magnetic resonance • Cardiac computed tomography • Nuclear cardiology

Introduction

In the 'European Society of Cardiology (ESC) Guidelines for the diagnosis and treatment of acute and chronic heart failure (HF)',[1] HF is defined as a syndrome consisting of symptoms and signs resulting from an abnormality of cardiac structure and/or function. The symptoms are non-specific and the signs can be absent in patients receiving diuretics, so the demonstration of existence of an abnormality of cardiac structure and/or function is essential for HF diagnosis. Providing information on cardiac structure and function, imaging has an important role not only in HF diagnosis, but also in definition of HF aetiology, in follow-up, and in treatment planning.

Echocardiography represents the first-line cardiovascular imaging (CVI) modality for the assessment of patients with HF. Cardiac magnetic resonance (CMR), single photon emission computed tomography (SPECT), positron emission tomography (PET), and cardiac computed tomography (CCT) complement echocardiography or represent an alternative to it in the case of suboptimal acoustic window.

The demand for CVI in HF is constantly increasing as a result of continuously evolving technology, diversification of indications, and rise in HF prevalence, partially due to better life expectancy and higher HF prevalence in the elderly.[2,3] The increasing demand necessitates appropriateness criteria for CVI use in HF to assist decision-making.

A literature review was performed in order to inform the process of definition of clinical indications for CVI use in the most common HF clinical scenarios. The present report is structured according to these clinical scenarios.

HF clinical scenarios

The clinical scenarios belong to the following three categories.

Diagnosis

First diagnosis of HF

Echocardiography is recommended as the CVI modality of choice for first assessment in suspected HF because of its wide availability,

* Corresponding author. Tel: +32 4 366 71 94; Fax: +32 4 366 71 95, Email: plancellotti@chu.ulg.ac.be

bed-side portability, accuracy, safety, and low cost together with its large evidence base in all clinical scenarios[4–13] and main disadvantage only is the need for an acoustic window.[1] Echocardiography at rest, using 2D, 3D, contrast, spectral, and colour flow Doppler and Doppler Myocardial Imaging (DMI) provides information both regarding cardiac structure (wall dimensions, cavity volumes, and geometry, structural abnormalities of valves, pericardial thickness, or effusion) and function (global and regional function of the left and right ventricle, diastolic function, valvular function, and haemodynamics).[4–18] The increasing prevalence of HF with preserved left ventricular ejection fraction (LVEF)[19] enhances the importance of echocardiography in HF diagnosis, as the main diastolic dysfunction assessment and grading modality. About 50% of patients diagnosed with HF have normal LVEF.[19] Echocardiography can assess all diastolic function related parameters[20] while CMR can also assess some of them.[1] Although not often, radionuclide angiography can be used for systolic and diastolic function assessment.[21] Furthermore, systolic and diastolic function can be assessed by gated SPECT[22] at the time of myocardial perfusion assessment.

The initial HF diagnosis can be complemented by other imaging modalities, particularly by CMR,[1,23–28] which provides gold standard assessment of LV/RV volumes, EF, and cardiac mass, as well as providing non-invasive in vivo myocardial tissue characterization; arrhythmias can limit the precision of CMR LV volumes/EF assessment and other disadvantages of CMR are represented by claustrophobia, overt renal failure (relative contraindications), and the presence of magnetic resonance imaging (MRI)-conditional devices, and cerebral metallic clips (absolute contraindications).[1]

Transoesophageal echocardiography (TOE) can be used for LV assessment in the case of poor transthoracic window, if CMR is not available or not applicable (ventilated patient or contraindications). TOE assessment of valves (particularly mitral or prosthetic valves) can add important information, being essential in suspected endocarditis induced valvular abnormality resulting in HF. Also, left atrial appendage thrombus exclusion by TOE may be needed in HF patients with atrial fibrillation prior to cardioversion.

Symptomatic patient

Echocardiography is recommended as the first CVI modality of choice in patients with symptoms suggestive of HF.

In symptomatic patients with emergency presentation, echocardiography is recommended immediately in the case of haemodynamic compromise and early during hospitalization in the other cases. TOE may be necessary in suspected endocarditis or if the transthoracic window is poor, particularly in mechanically ventilated patients.

In symptomatic patients with elective presentation, in the absence of cardiac history, echocardiography is recommended in the case of elevated natriuretic peptide.

Asymptomatic (screening)

Screening first-degree relatives of cardiomyopathy patients, we can identify individuals with cardiomyopathy and subclinical or clinical HF. Echocardiography is usually the only imaging modality used for screening.[29,30] CMR may be necessary, for example, to exclude or confirm arrhythmogenic right ventricular cardiomyopathy or to identify infiltration. Serial testing is necessary in the absence of genetic exclusion of disease or in genotype-positive but phenotype-negative individuals. The screening timing and follow-up intervals have been described by the ESC Working Group on Myocardial and Pericardial Diseases.[29,30]

Patients on cardiotoxic chemotherapy often undergo periodic LV systolic function screening, despite lack of HF symptoms or signs. The same CVI modality should be used at follow-up to ensure comparability. A baseline echocardiogram is performed before commencing treatment to assess LV systolic and diastolic function, the valves and unexpected abnormalities. Echocardiography, with standardized image acquisition and analysis, provides reliable, reproducible, and cost-effective follow-up of systolic function in the majority of patients. Endocardial border delineation can be improved with contrast administration when needed. 3D echocardiography improves accuracy and DMI and speckle tracking increase sensitivity for subclinical cardiotoxicity detection.[31] Radionuclide LV angiography with multi-gated acquisition (MUGA) provides accurate and reproducible LV function assessment at the cost of ionizing radiation exposure.[32] CMR provides a radiation-free alternative but the high cost makes it difficult to justify CMR follow-up for patients on cardiotoxic drugs, considering the current low incidence of LV systolic dysfunction as a result of practice changes in oncology.[33] Following gadolinium-contrast administration though, CMR can identify myocardial fibrosis in cancer survivors.[34]

The high incidence of HF in patients with diabetes justifies screening for asymptomatic LV systolic dysfunction in this population[1] with or without BNP screening first, particularly in the case of associated risk factors for CAD, advanced age, hypertension, proteinuria, and retinopathy.[35] Echocardiography is again the CVI modality of choice for screening, but an alternative modality can be used in the case of poor acoustic window or need for additional information.

Asymptomatic patients in whom a murmur has been detected or with ECG abnormalities suggesting a primary or secondary underlying cardiomyopathy that could lead to HF should also be investigated.

Patient with cardiac history

(i) History of myocardial infarction.
(ii) History of structural heart disease.

Echocardiography is recommended in patients with a cardiac history of myocardial infarction or structural heart disease without prior natriuretic peptide check in the case of elective presentation with symptoms suggestive of HF.

Diagnosis of HF aetiology

Diagnosis of aetiology begins with echocardiography, which may suffice for this purpose (echocardiography at rest with or without stress echocardiography). Other CVI modalities may complement echocardiography, depending on their availability, affordability, diagnostic profile, contraindications, and associated risk.[1]

Diagnosis of ischaemic aetiology

Echocardiography may suffice for HF ischaemic aetiology diagnosis in the case of existence of regional wall motion abnormalities with thinned and highly echogenic (old, calcified) scar of myocardial infarction. Systolic dyssynchrony may mislead, giving false appearance of regional wall motion abnormality. Furthermore, a severely dilated LV with severely reduced global LV systolic function may have

apparent regional distribution of systolic dysfunction due to more pronounced hypokinesia without or with thinning in some areas.

In symptomatic patients with emergency presentation, initial existence of systolic dysfunction, usually with coronary artery territory distribution, with subsequent normalization, suggests ischaemic HF aetiology. This transient systolic dysfunction is due to myocardial stunning following an episode of acute ischaemia,[36] phenomenon which can persist for a few hours. Myocardial stunning may occur in both symptomatic and silent ischaemia and it can provoke ischaemic mitral regurgitation of similar transient nature. When performed late, echocardiography may miss these transient abnormalities. Strain assessment with echocardiography may detect ischaemia induced abnormal regional myocardial deformation in HF due to a non-ST-elevation acute coronary syndrome.[37]

Echocardiography at rest can be complemented by stress echocardiography, CMR and perfusion CMR, SPECT, CT coronary angiography or PET.

Stress echocardiography can be used to reveal inducible ischaemia as reason for an episode of unexplained acute pulmonary oedema. It can be also used detect viability,[13] by demonstrating existence of inotropic reserve with an increase in regional (in a coronary artery territory) and global LV systolic function. A low dose dobutamine infusion protocol with prolonged (5 min) stages is used for this purpose. Inotropic reserve is recruited in the case the systolic dysfunction is due to myocardial hibernation,[38–41] a phenomenon due to chronic post-ischaemic dysfunction resulting from cumulative stunning.[36] Inotropic reserve is also recruited in the case the systolic dysfunction is due to non-transmural infarct. Continuous improvement in systolic function throughout the test is observed in the case of non-ischaemic aetiology of systolic dysfunction. A bi-phasic response can be observed in the case of existence of flow limiting coronary disease, with drop in regional systolic function after an initial increase. Only in the case of bi-phasic response demonstration does stress echocardiography add information regarding ischaemic aetiology to the information provided by rest echocardiography.

Depending on availability and local expertise, CMR can be used for coronary artery disease (CAD) diagnosis in HF patients and for viability assessment.[40–44] Since regional wall motion abnormalities can also be found in non-ischaemic cardiomyopathies, the specificity of cine-imaging alone for CAD detection is limited.[45] Late gadolinium enhancement (LGE) assessment post contrast injection may help the differential diagnosis based on the distinct distribution patterns of contrast in ischaemic and non-ischaemic cardiomyopathies.[46] Subendocardial or transmural LGE is present in most ischaemic HF patients (sensitivity 86%).[47] CMR has good performance in CAD diagnosis.[48,49] It can predict reversibility of wall thinning due to myocardial hibernation rather than necrosis.[42] LGE CMR predicts systolic function recovery following revascularisation.[50,51] T_1 mapping is a recently developed CMR technique, which allows the detection of interstitial myocardial fibrosis, an early marker of disease that has an evolving role in HF.[52] CMR can be used for detection of inducible ischaemia, either by detecting inducible regional wall motion abnormalities or by assessing myocardial perfusion. Inducible regional wall motion abnormalities assessed with dobutamine stress CMR, similarly to dobutamine stress echocardiography, carries an increased risk in HF patients with severely reduced LV systolic function and high likelihood of proximal coronary disease. Myocardial perfusion assessment is performed with vasodilator stress (usually adenosine)[53] so it is safe for use even in suspected acute coronary syndromes.[54]

SPECT can be used to detect ischaemia and viability, providing diagnostic and prognostic information.[1,55–58] Thallium-201 (^{201}Tl) and technetium-99m (^{99m}Tc) labelled tracers (sestamibi and tetrofosmin) are commonly used. Exposure to ionizing radiation, relatively low spatial resolution and attenuation artefacts are disadvantages of SPECT.[36] Nevertheless, there is extensive evidence that SPECT can predict global and regional systolic function improvement after revascularisation.[36] The CAD diagnosis relies on existence of flow heterogeneity and as such, SPECT should be used with caution for diagnosis of HF aetiology because of the likelihood of falsely negative results in balanced ischaemia with uniform radioisotope uptake encountered in 5–10% of patients with three vessels coronary disease.[59]

PET or PET–CT can be used for assessment of both ischaemia and viability.[1] PET has superior spatial resolution compared with SPECT. Ischaemia can be evaluated using perfusion tracers like Rubidium-82, Nitrogen-13 ammonia, or Oxygen-15 water. For viability detection, F-18-fluorodeoxyglucose (FDG) is most commonly used. Among viability imaging modalities, FDG PET has the highest sensitivity and is regarded as the gold standard.[60,61] PET can predict improvement in HF symptoms, functional status, quality of life[62,63] global, and regional systolic function post revascularisation.[36] Limitations are the need for a cyclotron on site[36] when very short half-life radiotracers are used, the high cost and the exposure to radiation.[1]

CT calcium scoring and CT coronary angiography can be used in HF patients[1,64] mainly for CAD diagnosis, having the advantage of their non-invasive nature. The absence of coronary calcium on CT virtually excludes CAD as cause the of HF. CT coronary angiography can reliably rule out CAD, being most effective in patients with a relative low probability of ischaemic HF aetiology.[65,66] Disadvantages are the exposure to radiation and the potential contrast nephrotoxicity of CT coronary angiography often relevant in patients with renal impairment at the time of HF diagnosis. Limited data and current practical challenges prevent recommendation of late-enhancement CT for viability detection.[67]

Diagnosis of non-ischaemic aetiology

Echocardiography is the first test of choice and often sufficient for non-ischaemic HF aetiology diagnosis (valve disease, cardiac tumours, pericardial disease, congenital heart disease, non-ischaemic LV systolic dysfunction, or LV diastolic dysfunction).

Valve disease can be identified on the initial echocardiogram performed to investigate HF symptoms and signs. The degree of valvular abnormality may be enough to explain HF (e.g. severe mitral regurgitation or moderate mixed aortic and mitral valve disease). Further assessment with stress echocardiography is needed if the degree of valvular abnormality at rest does not justify HF. Stress echocardiography with supine bicycle exercise is needed in moderate mitral regurgitation to detect a dynamic component (increase in regurgitation severity on exertion). Changes in pulmonary artery pressure during exercise have prognostic and therapeutic importance. For assessment of mitral stenosis, both supine bicycle exercise and dobutamine stress echo can be appropriate. With exercise, increase in mean transvalvular gradient to >15 mmHg and increase in systolic pulmonary artery pressure (SPAP) to >60 mmHg suggest severe mitral stenosis. With dobutamine, increase in mean gradient to >18 mmHg suggests severe

mitral stenosis; SPAP changes are not interpretable. In the case of suspected severe paradoxical low flow aortic stenosis, both supine bicycle exercise echo and low dose dobutamine stress echo can be appropriate. In low flow low gradient aortic stenosis with reduced LV EF, low dose dobutamine stress echo is the classical investigation of choice, for assessment of both flow reserve and valve compliance to flow. Nevertheless, usually patients can exercise enough to trigger sufficient myocardial recruitment and flow increase at low workload for severity of disease diagnosis. When existence of flow reserve and significant exertion induced rise in transvalvular gradient with or without concomitant increase in functional valve area are demonstrated, supine bicycle exercise echocardiography can be diagnostic. In the case of a negative result for existence of flow reserve or in the case of suboptimal exercise, low dose dobutamine stress echo should be performed. CMR is the second line imaging modality in valve disease, and sometimes it can complement echocardiography.[68–71] CMR provides information regarding the surrounding anatomy (great vessels), accurate quantification of ventricular volumes and regurgitations, as well as assessment of myocardial fibrosis, which carries prognostic information.[72]

Diagnosis of cardiac tumours or of cardiac involvement by tumour as reason for HF can be made by echocardiography. CMR may complement the echocardiography diagnosis.[1]

The diagnosis of pericardial disease as reason for HF can be made by echocardiography. Cardiac CT or CMR can complement the diagnosis.[1]

Even if echocardiography can provide a first diagnosis of congenital heart disease and can usually suffice for a complete comprehensive evaluation, CMR is the CVI modality of choice in complex congenital heart disease.[1,73]

Non-ischaemic LV systolic dysfunction is diagnosed by exclusion of CAD, described in the 'Diagnosis of ischaemic aetiology' section.

Diastolic dysfunction is diagnosed as reason for HF in the absence of LV systolic dysfunction, valvular abnormality or pulmonary hypertension which to explain the HF symptoms and signs. Diastolic dysfunction is usually diagnosed by echocardiography. Morphologic correlates of diastolic dysfunction (LV hypertrophy and left atrial dilatation[17]) can be diagnosed by echocardiography or CMR. Serial studies of morphologic correlates (cardiac mass, left atrial volume) for follow-up of disease progression or regression can be performed more accurately with CMR.[14] Furthermore, CMR provides tissue characterization, detecting infiltration, or inflammation.[14] Functional correlates of diastolic dysfunction—left atrial function and pulmonary artery systolic and diastolic pressure[17]—can be diagnosed by echocardiography or invasive cardiac catheterization. LV relaxation, filling, diastolic distensibility, and diastolic stiffness are assessed with cardiac catheterisation.[14] Echocardiography can provide a comprehensive complete diastolic function study. In the case of inconclusive diastolic parameters at rest and no other diagnostic structural cardiac abnormality, exercise echocardiography, ideally with a supine bicycle, can be used for diagnosis.[17] CMR can provide many filling parameters similar to echocardiography[14] but it is not used routinely due to the need for specific image acquisition and lengthy analysis. Radionuclide angiography can assess all parameters of diastolic function, but its use is limited nowadays.[21] Diastolic function can also be assessed by myocardial perfusion gated SPECT.[22]

Cardiomyopathies can be first diagnosed as a result of presentation with HF symptoms and signs. Usually, echocardiography alone makes the diagnosis.[30] CMR may be needed, for example to diagnose arrhythmogenic right ventricular cardiomyopathy or to confirm an echocardiographic diagnosis by identifying inflammation or infiltration with tissue characterisation.[74,75] Myocardial inflammation and oedema are diagnosed with T_2 weighted CMR imaging, whereas myocardial infiltration and fibrosis are diagnosed with T_1 weighted CMR imaging post contrast (LGE). Non-ischaemic LGE patterns include mid-wall, epicardial, and patchy. For example, mid-wall LGE can be detected in dilated cardiomyopathy (DCM)[75] albeit it cannot indicate the underlying aetiology (idiopathic DCM vs. DCM secondary to myocarditis).[47] Amyloidosis can have a characteristic diffuse LGE, or a zebra pattern LGE coupled with a typical dark myocardial cavity due to very abnormal contrast kinetics.[76]

Treatment planning

Imaging plays a central role in HF treatment planning, being used for risk stratification and to predict treatment benefit.

Revascularization

Coronary revascularization may improve LV systolic function in ischaemic HF, by reversing systolic dysfunction due to hibernation rather than infarction. The benefit depends not only on the existence of regional viability, but also on the existence of appropriate revascularization targets in the respective coronary arteries and on the degree of LV remodelling.

HF and angina symptoms

Based on evidence, revascularization is recommended in patients with angina and HF due to ischaemic LV systolic dysfunction.[77] In the case of severe proximal CAD, the ESC recommends surgical revascularization regardless of any other considerations.[77] Concomitant surgical ventricular reconstruction may be necessary when the LV end-systolic volume index is >60 mL/m^2 and there is transmural scar in the LAD (left anterior descending artery) territory. Percutaneous coronary intervention may replace CABG in the case of suitable coronary anatomy and existence of viability in the respective territory of distribution.[39,77] Evidently, treatment planning implies imaging based assessment of end-systolic volume and viability. As detailed in the 'Diagnosis of ischaemic aetiology', echocardiography can provide this assessment, and so does CMR.[40,41] SPECT or PET can be used to detect viability.[41] In the case of angina symptoms, HF patients will have invasive coronary angiography performed for CAD diagnosis, making usually unnecessary the detection of inducible ischaemia and consequently reducing the risk of a dobutamine stress echocardiography viability test.

HF without angina symptoms

In the absence of angina, revascularization planning is strictly based on existence of viability, increasing further the significance of the above discussed considerations.[77] The Surgical Treatment for Ischaemic Heart Failure (STICH) Trial[78] found lower rates of death from cardiovascular causes and of hospitalization for cardiovascular causes in HF patients treated with CABG revascularization. Nevertheless, a substudy of the STICH Trial,[79] despite finding greater likelihood of survival in patients with viability, found no differential survival benefit of revascularization based on diagnosed viability. However, many criticisms accompanied this sub-study, including the fact that

viability imaging was offered at the physicians' discretion, rather than randomization.

LV aneurysmectomy is recommended at the time of surgical revascularisation[77] and in this regard, the definition of the transition zone in between the scarred and not scarred myocardium is of major significance. LGE CMR or transthoracic echocardiography can be performed before the procedure[80] while TOE can be performed intraoperatively. Echocardiography has the advantage of concomitantly assessing the mitral valve for need of repair, based on assessment at rest or during exercise. CMR has the advantage of accurate assessment of volumes and better definition of the transition zone and it represents the gold standard for patient selection for aneurysmectomy.[80]

Contrast echocardiography, CMR,[81] or CCT can all assess the existence of mural thrombus prior to surgery.

No symptoms

In the absence of symptoms, detection of inducible ischaemia should complement the above discussed investigations. Considerations discussed in the 'Diagnosis of ischaemic aetiology' are appropriate in this regard.

Device therapy

Accurate assessment of LV EF is essential for deciding on device therapy.[82] Echocardiography is the first CVI modality of choice for LV EF assessment. 2D echocardiography can be used to assess LV EF with the bi-plane method, with or without the help of contrast to improve endocardial delineation. 3D echocardiography provides accurate and reproducible LV EF assessment. CMR provides gold standard assessment of LV EF and can be used in patients with poor acoustic window. Gated SPECT can be also used in patients with poor acoustic window and CMR contraindications.

Implantable cardioverter defibrillator planning

LV EF assessment with or without assessment of existence of ischaemic aetiology of HF are necessary for implantable cardioverter defibrillator (ICD) implantation planning. Echocardiography is the first CVI modality. Accurate LV EF assessment is essential for decision-making. CMR, MUGA, or gated SPECT can be used for LV EF assessment in patients with poor acoustic window. Considerations extensively presented in the 'Diagnosis of ischaemic aetiology' are relevant for aetiology assessment. There is emerging evidence that LGE CMR can predict ventricular arrhythmic events in ischaemic cardiomyopathy.[83,84]

Cardiac resynchronization therapy (CRT) planning

A complete echocardiographic assessment is recommended[85] before considering CRT. As for ICD planning, accurate EF assessment is essential and can be performed with echocardiography, CMR, MUGA, or gated SPECT. Viability assessment may be needed in the case of myocardial infarct involving potential LV lead placement areas (basal infero-lateral or lateral wall). CMR has been used to guide LV lead placement avoiding areas of transmural infarct[86] and to predict clinical outcome in CRT patients based on assessment of the right ventricle.[87,88]

Cardiac CT can visualize the coronary veins non-invasively if pre-procedural planning of LV lead placement is needed.

There is a wealth of echocardiography research performed in the field of dyssynchrony,[89–95] using DMI derived techniques, speckle tracking, 3D regional volume curves analysis, and even M-mode. Nevertheless, based on currently existent evidence, echocardiographic dyssynchrony assessment should not be used to exclude patients from CRT.[85] Similarly, gated SPECT phase analysis,[96] gated PET/CT,[97] and CMR techniques[53,98] have been studied for the assessment of dyssynchrony. In the absence of relevant randomized control trials though, no CVI assessment of dyssynchrony should be used for clinical decision-making.[99]

LV assist device

A complete echocardiographic assessment is needed before considering an LV assist device. Accurate assessment of LV EF is essential[82] for device treatment timing.

Follow-up

Planned follow-up

HF follow-up

Echocardiography is the first imaging modality of choice for follow-up of HF progression or regression on medical treatment or in patients treated with CRT. CMR can be used for follow-up when acoustic window is suboptimal and in the absence of an implanted non-MRI-conditional device (which prohibits the use of CMR).

CRT follow-up

Based on current evidence, echocardiography follow-up[85] for assessment of LV volumes and EF together with assessment of functional mitral regurgitation and pulmonary hypertension is recommended. Echocardiography based CRT optimization may be useful but it is not strongly recommended[85] based on current evidence.

New symptoms

A complete echocardiogram is necessary in the case new symptoms develop. Findings may justify involvement of an alternative CVI modality, mirroring already described scenarios.

Conflict of interest: none declared.

References

1. McMurray JJV, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K *et al.* ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012. *Eur Heart J* 2012;**33**:1787–847.
2. Mosterd A, Hoes AW. Clinical epidemiology of heart failure. *Heart* 2007;**93**: 1137–46.
3. Bui A, Horwich T, Fonarow G. Epidemiology and risk profile of heart failure. *Nat Rev Cardiol.* 2011;**8**:30–41.
4. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K *et al.* Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography,a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;**23**:685–713.
5. Dokainish H, Nguyen JS, Bobek J, Goswami R, Lakkis NM. Assessment of the American Society of Echocardiography-European Association of Echocardiography guidelines for diastolic function in patients with depressed ejection fraction: an echocardiographic and invasive haemodynamic study. *Eur J Echocardiogr* 2011;**12**:857–64.
6. Kirkpatrick JN, Vannan MA, Narula J, Lang RM. Echocardiography in heart failure: applications, utility, and new horizons. *J Am Coll Cardiol* 2007;**50**:381–96.
7. Lancellotti P, Tribouilloy C, Hagendorff A, Popescu BA, Edvardsen T, Pierard LA *et al.* On behalf of the European Association of Cardiovascular Imaging: Recommendations for the Assessment of Native Valvular Regurgitation: An Executive Revised

Summary from the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2013;**14**:611–44.
8. Lancellotti P, Moura L, Pierard LA, Agricola E, Popescu BA, Tribouilloy C *et al.* European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 2: mitral and tricuspid regurgitation (native valve disease). *Eur J Echocardiogr* 2010;**11**:307–32.
9. Lancellotti P, Tribouilloy C, Hagendorff A, Moura L, Popescu BA, Agricola E *et al.* European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 1: aortic and pulmonary regurgitation (native valve disease). *Eur J Echocardiogr* 2010;**11**:223–44.
10. Popescu BA, Stefanidis A, Nihoyannopoulos P, Fox KF, Ray S, Cardim N *et al.* Updated standards and processes for accreditation of echocardiographic laboratories from The European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging.* 2014;**15**:717–27.
11. Popescu BA, Andrade MJ, Badano LP, Fox KF, Flachskampf FA, Lancellotti P *et al.* European Association of Echocardiography recommendations for training, competence, and quality improvement in echocardiography. *Eur J Echocardiogr* 2009;**10**:893–905.
12. Nagueh SF, Bhatt R, Vivo RP, Krim SR, Sarvari SI, Russell K *et al.* Echocardiographic evaluation of hemodynamics inpatients with decompensated systolic heart failure. *Circ Cardiovasc Imaging* 2011;**4**:220–7.
13. Sicari R, Nihoyannopoulos P, Evangelista A, Kasprzak J, Lancellotti P, Poldermans D *et al.* Stress echocardiography expert consensus statement: European Association of Echocardiography (EAE) (a registered branch of the ESC). *Eur J Echocardiogr* 2008;**9**: 415–37.
14. Paulus WJ, Tschope C, Sanderson JE, Rusconi C, Flachskampf FA, Rademakers FE *et al.* How to diagnose diastolic heart failure: a consensus statement on the diagnosis of heart failure with normal left ventricular ejection fraction by the Heart Failure and Echocardiography Associations of the European Society of Cardiology. *Eur Heart J* 2007;**28**:2539–50.
15. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA *et al.* Recommendations for chamber quantification. *Eur J Echocardiogr* 2006;**7**:79–108.
16. Lang RM, Badano LP, Tsang W, Adams DH, Agricola E, Buck T *et al.* EAE/ASE Recommendations for image acquisition and display using three-dimensional echocardiography. *Eur Heart J Cardiovasc Imaging* 2012;**13**:1–46.
17. Nagueh SF, Appleton CP, Gillebert TC, Adams DH, Agricola E, Buck T *et al.* Recommendations for the evaluation of left ventricular diastolic function by echocardiography. *Eur J Echocardiogr* 2009;**10**:165–93.
18. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quinones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. *J Am Coll Cardiol* 1997;**30**:1527–33.
19. Owan TE, Hodge DO, Herges RM, Jacobsen SJ, Roger VL, Redfield MM. Trends in prevalence and outcome of heart failure with preserved ejection fraction. *N Engl J Med* 2006;**355**:251–9.
20. Melenovsky V, Borlaug BA, Rosen B, Hay I, Ferruci L, Morell CH *et al.* Cardiovascular Features of heart failure with preserved ejection fraction versus nonfailing hypertensive left ventricular hypertrophy in the Urban Baltimore Community. *J Am Coll Cardiol.* 2007;**49**:198–207.
21. Bonow RO. Radionuclide angiographic evaluation of left ventricular diastolic function. *Circulation.* 1991;**84**:I208–15.
22. Abidov A, Germano G, Hachamovitch R, Slomka P, Berman DS. Gated SPECT in assessment of regional and global left ventricular function: an update. *J Nucl Cardiol.* 2013;**20**:1118–43.
23. Schwitter J. Extending the frontiers of cardiac magnetic resonance. *Circulation* 2008; **118**:109–12.
24. Leong DP, De Pasquale CG, Selvanayagam JB. Heart failure with normal ejection fraction: the complementary roles of echocardiography and CMR imaging. *JACC Cardiovasc Imaging* 2010;**3**:409–20.
25. Raman SV, Simonetti OP. The CMR examination in heart failure. *Heart Fail Clin* 2009; **5**:283–300. v.
26. Charoenpanichkit C, Hundley WG. The 20-year evolution of dobutamine stress cardiovascular magnetic resonance. *J Cardiovasc Magn Reson* 2010;**26**:12–59.
27. Gulati A, Ismail TF, Jabbour A, Alpendurada F, Guha K, Ismail NA *et al.* The prevalence and prognostic significance of right ventricular systolic dysfunction in nonischemic dilated cardiomyopathy. *Circulation.* 2013;**128**:1623–33.
28. Masci PG, Francone M, Desmet W, Ganame J, Todiere G, Donato R *et al.* Right ventricular ischemic injury in patients with acute ST-segment elevation myocardial infarction: characterization with cardiovascular magnetic resonance. *Circulation.* 2010;**122**:1405–12.
29. Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, Charron P *et al.* 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy. The Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J* 2014;**35**:2733–79.
30. Charron P, Arad M, Arbustini E, Basso C, Bilinska Z, Elliott P *et al.* Genetic counselling and testing in cardiomyopathies: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2010;**31**:2715–28.
31. Tocchetti CG, Ragone G, Coppola C, Basso C, Bilinska Z, Elliott P *et al.* Detection, monitoring, and management of trastuzumab-induced left ventricular dysfunction: an actual challenge. *Eur J of Heart Failure* 2012;**14**:130–7.
32. Jones AL, Barlow M, Barrett-Lee PJ, Canney PA, Gilmour IM, Robb SD *et al.* Management of cardiac health in trastuzumab-treated patients with breast cancer: updated United Kingdom National Cancer Research Institute recommendations for monitoring. *Br J Cancer* 2009;**100**:684–92.
33. Hamirani YS, Jiji R, Salerno M, Wong A, Loffler A, Brenin CM *et al.* Delineation of anthracyclines and Herceptin induced cardiotoxicity using contrast enhanced CMR. *J Cardiovasc Magn Reson* 2014;**16**:P278.
34. Ylänen K, Poutanen T, Savikurki-Heikkilä P, Rinta-Kiikka I, Eerola A, Vettenranta K. Cardiac magnetic resonance imaging in the evaluation of the late effects of anthracyclines among long-term survivors of childhood cancer. *J Am Coll Cardiol.* 2013;**61**: 1539–47.
35. MacDonald MR, Petrie MC, Hawkins NM, Petrie JR, Fisher M, McKelvie R *et al.* Diabetes, left ventricular systolic dysfunction, and chronic heart failure. *Eur Heart J* 2008; **29**:1224–40.
36. Camici PG, Prasad SK, Rimoldi OE. Stunning, hibernation and assessment of myocardial viability. *Circulation.* 2008;**117**:103–14.
37. 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.
38. Vanoverschelde JLJ, Pasquet A, Gerber B, Melin JA. Pathophysiology of myocardial hibernation. Implications for the use of dobutamine echocardiography to identify myocardial viability. *Heart* 1999;**82**:III1–7.
39. Afridi I, Kleiman NS, Raizner AE, Zoghbi WA. Dobutamine echocardiography in myocardial hibernation. optimal dose and accuracy in predicting recovery of ventricular function after coronary angioplasty. *Circulation.* 1995;**91**:663–70.
40. Camici PG, Prasad SK, Rimoldi OE. Contemporary reviews in cardiovascular medicine. stunning, hibernation, and assessment of myocardial viability. *Circulation.* 2008; **117**:103–14.
41. Underwood R, Bax JJ, vom Dahl J, Henein MY, van Rossum AC, Schwarz ER *et al.* Imaging techniques for the assessment of myocardial hibernation. Report of a Study Group of the European Society of Cardiology. *Eur Heart J* 2004;**25**:815–36.
42. John AS, Dreyfus GD, Pennell DJ. Images in cardiovascular medicine. Reversible wall thinning in hibernation predicted by cardiovascular magnetic resonance. *Circulation.* 2005;**111**:e24–5.
43. Bucciarelli-Ducci C, Wu E, Lee DC, Holly TA, Klocke FJ, Bonow RO. Contrast-enhanced cardiac magnetic resonance in the evaluation of myocardial infarction and myocardial viability in patients with ischemic heart disease. *Curr Probl Cardiol.* 2006;**31**:128–68.
44. Schuster A, Morton G, Chiribiri A, Perera D, Vanoverschelde JL, Nagel E. Imaging in the management of ischemic cardiomyopathy: special focus on magnetic resonance. *J Am Coll Cardiol.* 2012;**59**:359–70.
45. Marcu CB, Nijveldt R, Beek AM, Van Rossum AC. Delayed contrast enhancement magnetic resonance imaging for the assessment of cardiac disease. *Heart Lung Circ* 2007;**16**:70–8.
46. Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT *et al.* International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol.* 2009;**53**:1475–87.
47. Valle-Munoz A, Estornell-Erill J, Soriano-Navarro CJ, Holmvang G, Alakija P, Cooper LT *et al.* Late gadolinium enhancement-cardiovascular magnetic resonance identified coronary artery disease as the aetiology of left ventricular dysfunction in acute new-onset congestive heart failure. *Eur J Echocardiogr* 2009;**10**:968–74.
48. Greenwood JP, Maredia N, Younger JF, Holmvang G, Alakija P, Cooper LT *et al.* Cardiovascular magnetic resonance and single-photon emission computed tomography for diagnosis of coronary heart disease (CE-MARC): a prospective trial. *Lancet* 2012;**379**:453–60.
49. Schwitter J, Wacker CM, Wilke N, Holmvang G, Alakija P, Cooper LT *et al.* Superior diagnostic performance of perfusion-cardiovascular magnetic resonance versus SPECT to detect coronary artery disease: The secondary endpoints of the multicentre multivendor MR-IMPACT II (Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary Artery Disease Trial). *J Cardiovasc Magn Reson* 2012;**14**: 1–10.
50. Kim RJ, Wu E, Rafael A, Holmvang G, Alakija P, Cooper LT *et al.* The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med* 2000;**343**:1445–53.
51. Bondarenko O, Beek AM, Twisk JW, Visser CA, van Rossum AC. Time course of functional recovery after revascularization of hibernating myocardium: a contrast-enhanced cardiovascular magnetic resonance study. *Eur Heart J* 2008;**29**:2000–5.
52. Baksi AJ, Pennell DJ. T1 mapping in heart failure: from technique to prognosis, toward altering outcome. *Circ Cardiovasc Imaging.* 2013;**6**:861–3.

53. Sohal M, Duckett SG, Zhuang X, Shi W, Ginks M, Shetty A *et al.* A prospective evaluation of cardiovascular magnetic resonance measures of dyssynchrony in the prediction of response to cardiac resynchronization therapy. *J Cardiovasc Magn Reson* 2014;**16**:58.
54. Lockie T, Nagel E, Redwood S, Plein S. Use of cardiovascular magnetic resonance imaging in acute coronary syndromes. *Circulation* 2009;**119**:1671–81.
55. Dilsizian V, Rocco TP, Freedman NM, Leon MB, Bonow RO. Enhanced detection of ischemic but viable myocardium by the reinjection of thallium after stress redistribution imaging. *N Engl J Med* 1990;**323**:141–6.
56. Kitsiou AN, Srinivasan G, Quyyumi AA, Summers RM, Bacharach SL, Dilsizian V. Stress-induced reversible and mild-to-moderate irreversible thallium defects: are they equally accurate for predicting recovery of regional left ventricular function after revascularization? *Circulation* 1998;**98**:501–8.
57. Udelson JE, Coleman PS, Metherall J, Pandian NG, Gomez AR, Griffith JL *et al.* Predicting recovery of severe regional ventricular dysfunction. Comparison of resting scintigraphy with 201Tl and 99mTc-sestamibi. *Circulation.* 1994;**89**:2552–61.
58. Senior R, Kaul S, Raval U, Lahiri A. Impact of revascularization and myocardial viability determined by nitrate-enhanced Tc-99m sestamibi and Tl-201 imaging on mortality and functional outcome in ischemic cardiomyopathy. *J Nucl Cardiol.* 2002;**9**:454–62.
59. Beller GA, Ragosta M. Decision making in multivessel coronary disease. *JACC Cardiovasc Interv* 2010;**3**:315–7.
60. Beanlands RS, Nichol G, Huszti E, Humen D, Racine N, Freeman M *et al.* F-18-fluorodeoxyglucose positron emission tomography imaging-assisted management of patients with severe left ventricular dysfunction and suspected coronary disease: a randomized, controlled trial (PARR-2). *J Am Coll Cardiol* 2007;**50**:2002–12.
61. Schinkel AF, Bax JJ, Poldermans D, Elhendy A, Ferrari R, Rahimtoola SH. Hibernating myocardium: diagnosis and patient outcomes. *Curr Probl Cardiol* 2007;**32**:375–410.
62. Di Carli MF, Asgarzadie F, Schelbert HR, Brunken RC, Laks H, Phelps ME *et al.* Quantitative relation between myocardial viability and improvement in heart failure symptoms after revascularization in patients with ischemic cardiomyopathy. *Circulation* 1995;**92**:3436–44.
63. Marwick TH, Zuchowski C, Lauer MS, Secknus MA, Williams J, Lytle BW. Functional status and quality of life in patients with heart failure undergoing coronary bypass surgery after assessment of myocardial viability. *J Am Coll Cardiol* 1999;**33**:750–8.
64. Nieman K, Cury RC, Ferencik M, Nomura CH, Abbara S, Hoffmann U *et al.* Differentiation of recent and chronic myocardial infarction by cardiac computed tomography. *Am J Cardiol.* 2006;**98**:303–8.
65. Ghostine S, Caussin C, Habis M, Habib Y, Clément C, Sigal-Cinqualbre *et al.* Non-invasive diagnosis of ischaemic heart failure using 64-slice computed tomography. *Eur Heart J.* 2008;**29**:2133–40.
66. Ten Kate GJ, Caliskan K, Dedic A, Meijboom WB, Neefjes LA, Manintveld OC *et al.* Computed tomography coronary imaging as a gatekeeper for invasive coronary angiography in patients with newly diagnosed heart failure of unknown aetiology. *Eur J Heart Fail.* 2013;**15**:1028–34.
67. Goetti R, Feuchtner G, Stolzmann P, Donati OF, Wieser M, Plass A *et al.* Delayed enhancement imaging of myocardial viability: low-dose high-pitch CT versus MRI. *Eur Radiol.* 2011;**21**:2091–9.
68. Myerson SG, d'Arcy J, Mohiaddin R, Greenwood JP, Karamitsos TD, Francis JM *et al.* Aortic regurgitation quantification using cardiovascular magnetic resonance: association with clinical outcome. *Circulation.* 2012;**126**:1452–60.
69. Chan KM, Wage R, Symmonds K, Rahman-Haley S, Mohiaddin RH, Firmin DN *et al.* Towards comprehensive assessment of mitral regurgitation using cardiovascular magnetic resonance. *J Cardiovasc Magn Reson.* 2008;**10**:61.
70. Barone-Rochette G, Piérard S, De Meester de Ravenstein C, Seldrum S, Melchior J, Maes F *et al.* Prognostic significance of LGE by CMR in aortic stenosis patients undergoing valve replacement. *J Am Coll Cardiol.* 2014;**64**:144–54.
71. Karamitsos TD, Myerson SG. The role of cardiovascular magnetic resonance in the evaluation of valve disease. *Prog Cardiovasc Dis.* 2011;**54**:276–86.
72. Bucciarelli-Ducci C, Azevedo CF. On fibrosis, prognosis, and the unique role of CMR: a paradigm shift from 'bright is dead' to 'bright is bad'. *J Am Coll Cardiol.* 2014;**64**:144–54.
73. Kilner PJ, Geva T, Kaemmerer H, Trindade PT, Schwitter J, Webb GD. Recommendations for cardiovascular magnetic resonance in adults with congenital heart disease from the respective working groups of the European Society of Cardiology. *Eur Heart J* 2010;**31**:794–805.
74. Schwitter J, Arai AE. Assessment of cardiac ischaemia and viability: role of cardiovascular magnetic resonance. *Eur Heart J* 2011;**32**:799–809.
75. Mahrholdt H, Wagner A, Judd RM, Sechtem U, Kim RJ. Delayed enhancement cardiovascular magnetic resonance assessment of non-ischaemic cardiomyopathies. *Eur Heart J.* 2005;**26**:1461–74.
76. Maceira AM, Joshi J, Prasad SK, Moon JC, Perugini E, Harding I *et al.* Cardiovascular magnetic resonance in cardiac amyloidosis. *Circulation.* 2005;**111**:186–93.
77. Wijns W, Kohl P, Danchin N, Di Mario C, Falk V, Folliguet T *et al.* Guidelines on myocardial revascularization The Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS) Developed with the special contribution of the European Association for Percutaneous Cardiovascular Interventions (EAPCI). *Eur Heart J* 2010;**31**:2501–55.
78. Velazquez EJ, Lee KL, Deja MA, Jain A, Sopko G, Marchenko A *et al.* for the STICH Investigators. Coronary-Artery Bypass Surgery in Patients with Left Ventricular Dysfunction. *N Engl J Med* 2011;**364**:1607–16.
79. Bonow RO, Maurer G, Lee KL, Holly TA, Binkley PF, Desvigne-Nickens P *et al.* for the STICH Trial Investigators. Myocardial Viability and survival in ischemic left ventricular dysfunction. *N Engl J Med* 2011;**364**:1617–25.
80. Castelvecchio S, Menicanti L. Left ventricular reconstruction: update to left ventricular aneurysm/reshaping techniques. *Multimedia Manual of Cardiothoracic Surgery* 2013; doi: 10.1093/mmcts/mmt002.
81. Weinsaft JW, Kim HW, Shah DJ, Klem I, Crowley AL, Brosnan R *et al.* Detection of left ventricular thrombus by delayed-enhancement cardiovascular magnetic resonance prevalence and markers in patients with systolic dysfunction. *J Am Coll Cardiol.* 2008; **52**:148–57.
82. Dickstein K, Vardas PE, Auricchio A, Daubert JC, Linde C, McMurray J *et al.* 2010 Focused Update of ESC Guidelines on device therapy in heart failure. An update of the 2008 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure and the 2007 ESC guidelines for cardiac and resynchronization therapy Developed with the special contribution of the Heart Failure Association and the European Heart Rhythm Association. *Eur Heart J* 2010;**31**:2677–87.
83. Scott PA, Rosengarten JA, Curzen NP, Morgan JM. Late gadolinium enhancement cardiac magnetic resonance imaging for the prediction of ventricular tachyarrhythmic events: a meta-analysis. *Eur J Heart Fail.* 2013;**15**:1019–27.
84. Roes SD, Borleffs CJ, van der Geest RJ, Westenberg JJ, Marsan NA, Kaandorp TA *et al.* Infarct tissue heterogeneity assessed with contrast-enhanced MRI predicts spontaneous ventricular arrhythmia in patients with ischemic cardiomyopathy and implantable cardioverter-defibrillator. *Circ Cardiovasc Imaging.* 2009;**2**:183–90.
85. Daubert JC, Saxon L, Adamson PB, Auricchio A, Berger RD, Beshai JF *et al.* 2012 EHRA/HRS expert consensus statement on cardiac resynchronization therapy in heart failure: implant and follow-up recommendations and management. *Europace* 2012;**14**:1236–86.
86. Bleeker GB, Kaandorp TA, Lamb HJ, Boersma E, Steendijk P, de Roos A *et al.* Effect of posterolateral scar tissue on clinical and echocardiographic improvement after cardiac resynchronization therapy. *Circulation.* 2006;**113**:969–76.
87. Alpendurada F, Guha K, Sharma R, Ismail TF, Clifford A, Banya W *et al.* Right ventricular dysfunction is a predictor of non-response and clinical outcome following cardiac resynchronization therapy. *J Cardiovasc Magn Reson.* 2011;**13**:68.
88. Leyva F, Taylor RJ, Foley PW, Umar F, Mulligan LJ, Patel K *et al.* Left ventricular midwall fibrosis as a predictor of mortality and morbidity after cardiac resynchronization therapy in patients with nonischemic cardiomyopathy. *J Am Coll Cardiol.* 2012;**60**: 1659–67.
89. Delgado V, Bax JJ. Assessment of systolic dyssynchrony for cardiac resynchronization therapy is clinically useful. *Circulation.* 2011;**123**:640–55.
90. Cleland JG, Daubert JC, Erdmann E, Freemantle N, Gras D, Kappenberger L *et al.* The effect of cardiac resynchronization on morbidity and mortality in heart failure. *N Engl J Med.* 2005;**352**:1539–49.
91. Bax JJ, Bleeker GB, Marwick TH, Molhoek SG, Boersma E, Steendijk P *et al.* Left ventricular dyssynchrony predicts response and prognosis after cardiac resynchronization therapy. *J Am Coll Cardiol.* 2004;**44**:1834–40.
92. Van Bommel RJ, Ypenburg C, Borleffs CJ, Delgado V, Marsan NA, Bertini M *et al.* Value of tissue Doppler echocardiography in predicting response to cardiac resynchronization therapy in patients with heart failure. *Am J Cardiol.* 2010;**105**:1153–8.
93. Yu CM, Fung WH, Lin H, Zhang Q, Sanderson JE, Lau CP. Predictors of left ventricular reverse remodeling after cardiac resynchronization therapy for heart failure secondary to idiopathic dilated or ischemic cardiomyopathy. *Am J Cardiol.* 2003;**91**:684–8.
94. Chung ES, Leon AR, Tavazzi L, Sun JP, Nihoyannopoulos P, Merlino J *et al.* Results of the Predictors of Response to CRT (PROSPECT) trial. *Circulation.* 2008;**117**:2608–16.
95. Bleeker GB, Bax JJ, Fung JW, van der Wall EE, Zhang Q, Schalij MJ *et al.* Clinical versus echocardiographic parameters to assess response to cardiac resynchronization therapy. *Am J Cardiol.* 2006;**97**:260–3.
96. Soman P, Chen J. Left ventricular dyssynchrony assessment using myocardial single-photon emission CT. *Semin Nucl Med.* 2014;**44**:314–9.
97. Wang L, Wei HX, Yang MF, Guo J, Wang JF, Fang W *et al.* Phase analysis by gated F-18 FDG PET/CT for left ventricular dyssynchrony assessment: a comparison with gated Tc-99 m sestamibi SPECT. *Ann Nucl Med.* 2013;**27**:325–34.
98. Andersson LG, Wu KC, Wieslander B, Loring Z, Frank TF, Maynard C *et al.* Left ventricular mechanical dyssynchrony by cardiac magnetic resonance is greater in patients with strict vs nonstrict electrocardiogram criteria for left bundle-branch block. *Am Heart J.* 2013;**165**:956–63.
99. Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA *et al.* 2013 ESCGuidelines on cardiac pacing and cardiac resynchronization therapy. The Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). *Eur Heart J* 2013;**34**:2281–329.