INTRODUCTION
(Pennell 2010)

- Cardiac magnetic resonance imaging (MRI or CMR) provides high quality cardiovascular imaging without exposure to radiation. Quality imaging process requires patient ability to perform breath holding or regular free breathing, a regular rhythm, and absence of local implants that interfere with image or any implants that interfere with safety (Gerber 2018).

- Cardiac magnetic imaging (CMR) is often required when transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) elicit inadequate imaging data.

- Stress CMR for assessment of coronary artery disease (CAD) is performed pharmacologically either as:
  - Vasodilator perfusion imaging with gadolinium contrast
  - OR
  - Dobutamine inotropic wall motion (ventriculography)

- CMR is frequently competitive with Cardiac CT (Cardiac Computed Tomography) with respect to structural imaging (Warnes 2008; Baumgartner 2010; Pennell 2010).

<table>
<thead>
<tr>
<th>Modality</th>
<th>Cardiac CT</th>
<th>Cardiac MR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrast</td>
<td>Often required</td>
<td>Required for some tissue characterization studies, often unnecessary</td>
</tr>
<tr>
<td>Radiation*</td>
<td>Yes</td>
<td>None, advantage for young patients and those requiring frequent exams</td>
</tr>
<tr>
<td>Resolution</td>
<td>Higher spatial</td>
<td>Higher temporal</td>
</tr>
<tr>
<td>Flow</td>
<td>Not standard</td>
<td>Standard</td>
</tr>
<tr>
<td>Patient comfort</td>
<td>Easy</td>
<td>Claustrophobia issues</td>
</tr>
<tr>
<td>Ferromagnetic implants</td>
<td>No issue</td>
<td>Relative contraindication</td>
</tr>
<tr>
<td>Cost</td>
<td>Moderate to High</td>
<td>High</td>
</tr>
</tbody>
</table>
Some scenarios might provide more detail with low dose CT than with CMR, thereby overriding the radiation risk (Ohana 2015).

- **With respect to CAD evaluation**, since CMR is only pharmacologic (non-exercise stress), and stress echocardiography (SE) or myocardial perfusion imaging (MPI) provide similar information about CAD, with SE performed at lower cost:
  
  - Requests for stress CMR require **diversion** to exercise SE first, to exercise MPI second.

  **Exemptions** for the diversion to SE or exercise MPI:
  - If body habitus or marked obesity (e.g. BMI > 40) would interfere significantly with imaging with SE and MPI (Shah 2014)
  - Evaluation of young (< 55 years old) patients with documented complex CAD, who are likely to need frequent non-invasive coronary ischemia evaluation and/or frequent radiation exposure from other testing (Hirshfeld 2018).

- CMR can be used as an **alternative to required pharmacologic** MPI (Fihn 2012).

  Pharmacologic perfusion imaging is indicated over exercise perfusion imaging in the following (Askew 2018):
  - Inability to exercise safely (e.g. prohibitive comorbidity, severe valvular disease, provocation of serious arrhythmia with exercise, uncontrolled hypertension, with systolic BP > 180 or diastolic BP > 120)
  - Complete left bundle branch block (LBBB) or a V-paced rhythm (due to perfusion artifacts)

- CMR can also be performed as a dobutamine stress test **when vasodilator MPI would be contraindicated:** (Chareonthaitawee 2018; Henzlova 2016)
  - Pulmonary or allergic intolerance to adenosine and analogues, documented or anticipated
  - Dipyridamole within < 48 hours
  - Relative unsuitability due to:
    - Hypotension or bradyarrhythmia
    - Interfering medications: Theophylline/aminophylline, caffeine, or theobromine within the past 12-24 hours
    - Seizure disorder with potential for adenosine provocation

- CAD stenosis ≥ 50% is considered clinically significant or obstructive CAD. CAD and ischemic heart disease (IHD) mean the same thing. Hemodynamically or functionally significant CAD means the degree of stenosis is severe enough to cause ischemia. This is discussed in more detail in the Additional Information section (Fihn 2012; Wolk 2013; Montalescot 2013; Gerber 2018; Tobis 2007).
Indications for CMR
(Hendel 2006; Hundley 2010)

CMR in CAD
(Fihn 2012; Wolk 2013; Montalescot 2013)

- **Stable patients without known CAD** fall into 2 categories (Fihn 2012; Wolk 2013; Montalescot 2013):
  
  - **Asymptomatic**, for whom global risk of CAD events can be determined from coronary risk factors, using calculators available online (see part III in the Additional Information section).
  
  - **Symptomatic**, for whom we estimate the pretest probability that their chest-related symptoms are due to clinically significant (> 50%) CAD (below):

**The 3 Types of Chest Pain or Discomfort**

- **Typical Angina (Definite)** is defined as including all 3 characteristics:
  1) Substernal chest pain or discomfort with characteristic quality and duration
  2) Provoked by exertion or emotional stress
  3) Relieved by rest and/or nitroglycerine

- **Atypical Angina (Probable)** has only 2 of the above characteristics

- **Nonanginal Chest Pain/Discomfort** has only 0·1 of the above characteristics

- Once the type of chest pain has been established from the medical record, the Pretest Probability of CAD (meaning obstructive CAD defined as coronary arterial narrowing ≥ 50%) is estimated from the **Diamond Forrester Table** below, recognizing that in some cases multiple additional coronary risk factors could increase pretest probability (Wolk 2013):

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Gender</th>
<th>Typical/Definite Angina Pectoris</th>
<th>Atypical/Probable Angina Pectoris</th>
<th>Nonanginal Chest Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;=39</td>
<td>Men</td>
<td>Intermediate</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>Intermediate</td>
<td>Very low</td>
<td>Very low</td>
</tr>
<tr>
<td>40–49</td>
<td>Men</td>
<td>High</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>Intermediate</td>
<td>Low</td>
<td>Very low</td>
</tr>
<tr>
<td>50–59</td>
<td>Men</td>
<td>High</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>Intermediate</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>&gt;=60</td>
<td>Men</td>
<td>High</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>High</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
</tbody>
</table>
o **Very low**: < 5% pretest probability of CAD, usually not requiring stress evaluation (Fihn 2012)

o **Low**: 5-10% pretest probability of CAD

o **Intermediate**: 10% - 90% pretest probability of CAD

o **High**: > 90% pretest probability of CAD

### Indications for Cardiac Magnetic Resonance (CMR)
(Hendel 2006; Fuisz 2018)

<table>
<thead>
<tr>
<th>Use of CMR in CAD</th>
<th>(Fihn 2012; Wolk 2013; Montalescot 2013; Askew 2018; Hendel 2006 )</th>
</tr>
</thead>
</table>

#### Suspected CAD
(Without known history of CAD)

**CMR available as an alternative to appropriate vasodilator MPI**

1. **Symptomatic patients without known CAD**
   - Low pretest probability who are unable to exercise
   - Intermediate pretest probability
   - High pre-test probability
   - Repeat testing in patient with recurrent symptomatic presentation and negative result over 2 years ago
   - Repeat testing in patient with new or worse symptoms and negative result at least one year ago

2. **Asymptomatic patients without known CAD**:
   - Previously unevaluated electrocardiography (ECG) evidence of possible myocardial ischemia such as substantial ischemic ST segment or T wave abnormalities
   - Previously unevaluated pathologic Q waves or wall motion abnormality (evidence of prior myocardial infarction)
   - Unevaluated complete left bundle branch block in patients at intermediate to high global risk
   - Following radiation therapy to the anterior or left chest, at 5 years post inception of radiation and every 5 years thereafter (Lancellotti 2013)

3. **Incomplete or inconclusive CAD evaluation, within the past 2 years without known CAD**
   - Exercise stress ECG with low risk Duke treadmill score, but patient’s current symptoms indicate an intermediate or high pretest probability, which should include stress imaging
   - Exercise stress ECG with intermediate Duke treadmill score
   - Inconclusive/borderline CCTA (e.g. 40-70% lesions)
   - An indeterminate (equivocal, borderline, or discordant) evaluation by prior stress imaging (SE or MPI) within the past 2 years, for whom a noninvasive approach is preferable to proceeding to invasive coronary arteriography (e.g. unclear symptoms, ECG and imaging discordant, etc., but patient has severe contrast allergy, CKD, etc.)

#### Known Major Vessel CAD
CMR available as an alternative to appropriate vasodilator MPI
(Patel 2017)

- Validated concern for a previous acute coronary syndrome without subsequent invasive or noninvasive coronary evaluation
- Follow up MPI at 2-year intervals is approvable, if it will affect consideration of coronary revascularization (initial or additional), in patients with one of the following:
- History of silent ischemia with severe unrevascularized CAD, and revascularization could be feasible (Deedwania 2018)
- History of severe unrevascularized major multivessel CAD, and revascularization could be feasible
- Ejection fraction <= 40% with severe unrevascularized CAD, and revascularization could be feasible

- Ischemia assessment following inconclusive findings of invasive coronary arteriography or CCTA, for the purpose of assessing extent of ischemia and need for additional medical, interventional, or surgical therapy.
- For myocardial viability assessment with reduced LVEF <= 50% to assist with decisions regarding coronary revascularization, even when MPI or SE have been inconclusive in that regard (Patel 2013; Yancy 2013)
- New or worsening symptoms of ischemia in the absence of an acute coronary syndrome, unless the most current stress imaging study would warrant invasive coronary arteriography instead (e.g. History of high risk stress test without subsequent invasive coronary arteriography might warrant invasive coronary angiography) (Patel 2012)
- De novo HF, who have known CAD, even without angina, unless the patient is not eligible for revascularization of any kind, or unless invasive coronary arteriography is immediately planned (Yancy 2013)

### Special Diagnostic Conditions, Requiring Coronary Evaluation

<table>
<thead>
<tr>
<th>CMR available as an alternative to appropriate vasodilator MPI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>•</strong> Newly diagnosed systolic or diastolic heart failure, especially with symptoms or signs of ischemia AND without invasive coronary angiography immediately planned (Yancy 2013; Patel 2013; Fihn 2012)</td>
</tr>
<tr>
<td><strong>•</strong> Newly found wall motion abnormality (Colucci 2018a)</td>
</tr>
<tr>
<td><strong>•</strong> Ventricular arrhythmias</td>
</tr>
<tr>
<td>o Sustained VT &gt;100 bpm, VF, or exercise induced VT, when invasive coronary arteriography is not the initially required test (Al-Khatib 2018 in press)</td>
</tr>
<tr>
<td>o Nonsustained VT, multiple episodes, each ≥3 beats at ≥100 bpm, without known cause or associated cardiac pathology, when an exercise ECG has shown an intermediate risk Duke Score or exercise ECG could not be performed (Zimetbaum 2018)</td>
</tr>
<tr>
<td>o Frequent PVCs ≥ 30/hour, or any PVC on a 12 lead ECG, without known cause or associated cardiac pathology, when an exercise ECG has shown an intermediate risk Duke Score OR an exercise ECG is not feasible due to inability to exercise or due to an uninterpretable ECG (Cha 2012; Manolis 2018)</td>
</tr>
<tr>
<td><strong>•</strong> Prior to Class IC antiarrhythmic drug initiation in intermediate and high global risk patients (see global risk calculators in Additional Information Section) (Kumar 2018)</td>
</tr>
<tr>
<td><strong>•</strong> Assessment of hemodynamic significance of one of the following previously documented conditions (also see Congenital Heart section below) (Anagnostopoulos 2004):</td>
</tr>
<tr>
<td>o Anomalous coronary arteries (Grani 2017; Kilner 2010)</td>
</tr>
<tr>
<td>o Muscle bridging of coronary artery (perform with exercise stress) (Sorajja 2018)</td>
</tr>
<tr>
<td>o Coronary aneurysms in Kawasaki’s disease (Newburger 2018)</td>
</tr>
</tbody>
</table>
### Congenital Heart Disease

**Warnes 2008; Baumgartner 2010; Kilner 2010; Orwat 2014; Wiant 2009**

- For evaluation of anomalous thoracic arteriovenous vessels, such as TGA (Cohen 2016).
- Further assessment of complex adult congenital heart disease after confirmation by initial echocardiography (TTE and/or TEE), to answer remaining clinically relevant questions with the exception that:
  - Echocardiography is preferable to CMR for the identification of patent foramen ovale, structural abnormalities of valve leaflets, and their suspensory apparatus, and CMR should generally not be required (Douglas 2011)
- When TTE and/or TEE has been or would be insufficient for clinical management, for the choice between CMR and CT, several aspects must be considered including radiation exposure, resolution required, sum of information required, its impact upon management, the presence of a pacemaker/ICD, or other implants and patient claustrophobia. Sample indications include:
  - Quantification of right ventricular (RV) volumes and ejection fraction (tetralogy of Fallot, systemic RV, and tricuspid regurgitation) CMR is preferred over CT (Haddad 2008; Dupont 200; Benza 2008).
  - Evaluation of the RV outflow tract and RV-PA conduits (CMR or CT).
  - Quantification of pulmonic regurgitation (PR) (CMR, not CT).
  - Quantification of shunts by measurements of flow in the ascending aorta and pulmonary trunk (CMR, not CT).
  - Evaluation of the entire aorta (aneurysm, dissection, intramural hematoma, Loeys-Dietz, Ehlers-Danlos, or confirmed genetic mutation known to predispose to aortic aneurysm and dissection) (CMR or CT initially, with annual CMR (MR) for Loeys-Dietz, Ehlers Danlos; multiple options for Marfan’s, Turner’s; see Aortic Pathology section below) (Hiratzka 2010).
  - Evaluation of pulmonary arteries (stenosis and aneurysms) and the aorta (coarctation) (CMR or CT).
  - Evaluation of systemic and pulmonary veins (anomalous connection, obstruction, etc.) (CMR or CT).
  - Aorto-pulmonary collaterals and arteriovenous malformations (either, but CT is superior to CMR for spatial resolution).
  - Identification of coronary anomalies and CAD (CCTA better than CMR, if no other CMR data required) (also see coronary section above - Special Diagnostic Conditions)
  - Evaluation of intra- and extra-cardiac masses (CMR or CT).
  - Quantification of myocardial (muscle) mass (CMR or CT).
  - Detection and quantification of myocardial fibrosis/scar (gadolinium late enhancement) (CMR, not CT).
  - Tissue characterization (fibrosis, fat, iron etc.) (CMR, not CT).
  - Assessment of right ventricular morphology in arrhythmogenic right ventricular dysplasia/cardiomyopathy, based upon reason for suspicion, of which examples are:
    - Nonsustained VT
    - Syncope
    - ECG abnormality: Prolonged S wave upstroke, epsilon waves, or right precordial T wave inversions (> 14 yr old) in the absence of complete right bundle branch block
    - First degree relative with phenotype or genotype of ARVD/C (either, but CMR is superior to CT) (Marcus 2010; McKenna 2018; te Riele 2015).
Valvular (Doherty 2017; Baumgartner 2017; Nishimura 2014; Ordovas 2008)

- Both TTE and TTE images are inadequate or not feasible for evaluation of possible valvular heart disease due to patient characteristics.
- Severe tricuspid regurgitation and suboptimal TTE images, for assessment of RV systolic function and systolic and diastolic volumes

- In patients with MR, when TTE and TEE (if able) show:
  - Moderate or severe MR, but images are suboptimal for assessment of MR severity, left ventricular function, and/or systolic and diastolic volumes
  - OR
  - Severity of the MR that is discordant with the clinical assessment

- In patients with AR, when TTE shows:
  - Moderate or severe AR, but images are suboptimal for assessment of AR severity, left ventricular function, and/or systolic and diastolic volumes
  - OR
  - Severity of the AR that is discordant with the clinical assessment

- Pre TAVR assessment of aortic annular size and shape and/or the aortic dimensions, when the patient cannot undergo cardiac CT (Otto 2017)
- Prior to transcatheter mitral valve interventions, when TTE and TEE result in uncertain assessment of the severity of mitral regurgitation (Wunderlich 2018)
- Patients with bicuspid aortic valve and aortic dilation > 4.0 cm require annual imaging with CT, MRI, or echo. (Echo is required when is can evaluate the full extent of pathology under surveillance.) This would increase to biannual (twice-yearly) imaging in the event of any one of the additional conditions: diameter > 4.5 cm, rapid rate of change 0.5 cm/yr, or a family history of a first degree relative with aortic dissection. Initial imaging with first 6 month re-evaluation for rate of expansion is appropriate.
- Characterization of bioprosthetic valve if suspected clinically significant valvular dysfunction and inadequate images from TTE and TEE.

Myocardial & Heart Failure
(Patel 2013, Yancy 2013)

- Evaluation of LV function following myocardial infarction OR in heart failure patients, when TTE (even with contrast agents) or MUGA, have been inadequate or discordant with prior information. (Montalescot 2013)
- For management of patients requiring cardiotoxic chemotherapy, with any ONE of the following: (Plana 2014) (See Cardio-Oncology section under Additional Information.)
  - TTE has been inadequate, unreliable, or discordant with prior information.
  - Candidacy for cardiotoxic chemotherapy is questionable due to borderline left ventricular dysfunction on other imaging
  - Discontinuation of cardiotoxic chemotherapy on the basis of a decline in left ventricular dysfunction is being considered
- Left ventricular function assessment at baseline prior to initiation of radiation to the anterior or left chest, at 5 years post initiation, and every 5 years thereafter, when TTE has been inadequate. (Lancellotti 2013)
- Diagnosis and monitoring of specific infiltrative cardiomyopathies, amyloidosis, sarcoidosis, hemochromatosis, endomyocardial fibrosis (Pereira 2018, Ordovas 2008)
• Assisting with assessment of sudden cardiac arrest/death in patients with non ischemic cardiomyopathy, if it will affect decision making with respect to management of the risk for sudden cardiac arrest/death (e.g. ICD implantation) (Al-Khatib 2017, Kuruvilla 2014, Halliday 2017)

• In a patient suspected of cardiac sarcoid, evaluation of possible diffuse inflammation noted on 18-FDG PET, in order to guide therapy (Vita 2018)

• Diagnosis of acute myocarditis, with suspicion based upon new, unexplained findings such as any one of:
  o Rise in troponin not clearly due to acute myocardial infarction
  o Change in ECG suggesting acute myocardial injury or pericarditis, without evident myocardial infarction, often with arrhythmia
  o Abnormal systolic function
when the results could alter management (Friedrich 2013, Kinderman 2012, Cooper 2018, Ordovas 2008)

• In cardiomyopathy (Ordovas 2008)
  o For detailed assessment of hypertrophic cardiomyopathy, when TTE is inadequate for diagnosis, management or operative planning, or when tissue characterization (fibrosis quantitation) will impact indications for ICD (Maron 2012, Maron 2014, Al-Khatib 2017)
  o For confirming a diagnosis of ischemic cardiomyopathy when it will make a difference in clinical management (Fuisz 2016, bColucci 2018)

• Evaluation of first degree relatives with strong family history of cardiomyopathy, when TTE (even with contrast) was inadequate.

**Evaluation of Intra- and Extra-Cardiac Structures**

• Suspected cardiac mass, paravalvular abscess, suspected tumor or non-valvular thrombus (CT for valvular thrombus), or potential cardiac source of emboli, when TTE and TEE images are inadequate (Doherty 2017, Pennell 2010, Baumgartner 2017, Nishimura 2014, Kassop 2014, Ordovas 2008, Sexton 2018)

• In suspected infective endocarditis with moderate to high pretest probability (i.e. staph bacteremia, fungemia, prosthetic heart valve, or intracardiac device), when TTE is inadequate and TEE cannot be performed (Doherty 2017)

• Detailed evaluation of a known cardiac mass (tumor), non-valvular thrombus (CT for valvular thrombus), paravalvular abscess (most often previously noted on echocardiography) (Doherty 2017, Pennell 2010, Baumgartner 2017, Nishimura 2014, Kassop 2014, Ordovas 2008)

• When TTE and/or TEE are inadequate, evaluation of pericardial conditions (pericardial mass, constrictive pericarditis, constriction versus restrictive cardiomyopathy) (CT superior for calcium assessment) (Klein 2013, Pennell 2010, Ordovas 2008)

• Evaluation of left atrium and pulmonary veins prior to radiofrequency ablation for atrial fibrillation, including dimensions of veins for mapping purposes. (Ohana 2015, Figtree 2011)

• Assessment of left ventricular pseudoaneurysm, when TTE was inadequate and/or left ventriculography was not performed with cardiac catheterization or was inadequate. (Shapira 2018)

**Aortic Pathology:**
Echo is required when is can evaluate the full extent of pathology under surveillance.

- CT, MR, or echo can be used for screening and follow up, with CT and MR preferred for imaging beyond the proximal ascending thoracic aorta (see table below for top normal sizes).
  - Screening first degree relatives of individuals with a history of thoracic aortic aneurysm (defined as ≥ 50% above top normal) or dissection or an associated high risk mutation for thoracic aneurysm in common.
  - Screening second degree relative of a patient with thoracic aortic aneurysm (defined as ≥ 50% above top normal), when the first degree relative has aortic dilation, aneurysm, or dissection.
  - Six month follow up after initial finding of a dilated thoracic aorta, for assessment of rate of change.
  - Annual follow up of enlarged thoracic aorta that is above top normal for age, gender, and size up to 4.4 cm.
  - Biannual (twice/yr) follow up of enlarged aortic root ≥ 4.5 (> 4.5 cm with bicuspid aortic valve) cm or showing growth rate ≥ 0.5 cm/year.
An aneurysm is defined as ≥ 50% greater than top normal. (Cikach 2018; Hiratzka 2010)

- Marfan’s patients require annual imaging with CT, MRI (avoids radiation, especially when frequent evaluation required), or echo when it can evaluate the full extent of pathology, with increase to biannual (twice-yearly) when diameter reaches 4.5 cm or when expansions is > 0.5 cm/yr. (Complete aortic annual CMR is recommended for annual evaluation Loeys-Dietz, Ehlers-Danlos, and certain other noted genetic mutations, wherein surgical intervention is recommended at 4.2 cm.)

- Turner’s syndrome patients should undergo imaging (CT, MRI - avoids radiation, especially when frequent evaluation required, or echo (when it can evaluate the full extent of pathology), of the heart and aorta for evidence of dilatation of the ascending thoracic aorta, and with normal imaging and no risk factors for aortic dissection, repeat imaging should be performed every 5-10 years or if otherwise indicated. If the aorta is enlarged, appropriate follow up imaging should be done according to size, as noted above. With a bicuspid aortic valve, the recommendation below applies.
• Patients with bicuspid aortic valve and aortic dilation > 4.0 cm require annual imaging with CT, MRI, or echo. (Echo is required when is can evaluate the full extent of pathology under surveillance.) This would increase to biannual (twice-yearly) imaging in the event of any one of the additional conditions: diameter > 4.5 cm, rapid rate of change 0.5 cm/yr, or a family history of a first degree relative with aortic dissection. Initial imaging with first 6 month re-evaluation for rate of expansion is appropriate.

• CMR can be used for the diagnosis and surveillance of aortitis (Bhave 2018).

• Any interval increase > 3 mm on echo should be validated by CT or CMR. (Baumgartner 2014).

• When higher resolution measurement is required for determining an indication or surgery, CT appear slightly better (Baumgartner 2014).

• Computed tomographic imaging or magnetic resonance imaging of the thoracic aorta is reasonable after a Type A or B aortic dissection or after prophylactic repair of the aortic root/ascending aorta.

• Computed tomographic imaging or magnetic resonance imaging of the aorta is reasonable at 1, 3, 6, and 12 months post un-operated dissection or intramural hematoma, penetrating atherosclerotic aortic ulcer, and if stable, annually thereafter so that any threatening enlargement can be detected in a timely fashion.

• Postoperative surveillance recommendations are taken from the 2010 ACC Thoracic Aortic Disease Guideline: See Table below (Hiratzka 2010).
### Table 17. Suggested Follow-Up of Aortic Pathologies After Repair or Treatment

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Interval</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute dissection</td>
<td>Before discharge, 1 mo, 6 mo, yearly</td>
<td>CT or MR, chest plus abdomen TTE</td>
</tr>
<tr>
<td>Chronn dissection</td>
<td>Before discharge, 1 y, 2 to 3 y</td>
<td>CT or MR, chest plus abdomen TTE</td>
</tr>
<tr>
<td>Aortic root repair</td>
<td>Before discharge, yearly</td>
<td>TTE</td>
</tr>
<tr>
<td>AVR plus ascending</td>
<td>Before discharge, yearly</td>
<td>TTE</td>
</tr>
<tr>
<td>Aortic arch</td>
<td>Before discharge, 1 y, 2 to 3 y</td>
<td>CT or MR, chest plus abdomen</td>
</tr>
<tr>
<td>Thoracic aortic stent</td>
<td>Before discharge, 1 mo, 2 mo, 6 mo, yearly or 30 days*</td>
<td>CXR, CT, chest plus abdomen</td>
</tr>
<tr>
<td>Acute IMH/PAU</td>
<td>Before discharge, 1 mo, 3 mo, 6 mo, yearly</td>
<td>CT or MR, chest plus abdomen</td>
</tr>
</tbody>
</table>

*US Food and Drug Administration stent graft studies usually required before discharge or at 30-day CT scan to detect endovascular leaks. If there is concern about a leak, a predischarge study is recommended; however, the risk of renal injury should be borne in mind. All patients should be receiving beta blockers after surgery or medically managed aortic dissection, if tolerated. Adapted from Erbel et al (539).

AVR indicates aortic valve replacement; CT, computed tomographic imaging; CXR, chest x-ray; IMH, intramural hematoma; MR, magnetic resonance imaging; PAU, penetrating atherosclerotic ulcer; and TTE, transthoracic echocardiography.

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CT and MR preferred for imaging beyond the proximal ascending thoracic aorta.

(Table above from Hiratzka 2010)
ADDITIONAL INFORMATION
I. General

Scenarios for which approval CMR is generally not approvable:

- For any combination imaging study
- For same imaging tests less than six weeks part unless specific guideline criteria states otherwise, e.g. evaluation of cardiac sarcoid with MR subsequent to PET (Vita 2018)
- For different imaging tests, such as CTA and CMR, of same anatomical structure less than six weeks apart without high level review to evaluate for medical necessity.
- For re-imaging of repeat or poor quality study

Request for a follow-up study - A follow-up study may be needed to help evaluate a patient’s progress after treatment, procedure, intervention or surgery. Documentation requires a medical reason that clearly indicates why additional imaging is needed for the type and area(s) requested.

Cardiac Tumors (Kassop 2014; Ordovas 2008)
MRI is the modality of choice to evaluate cardiac tumors due to its high contrast resolution and multiplanar capability which allows for optimal evaluation of myocardial infiltration, pericardial involvement and extra-cardiac vascular structures within and beyond the thorax. It is also useful in the differentiation of benign and malignant cardiac tumors and in differentiating thrombi from cardiac tumors.

CMR Safety (Chernoff 2018; Russo 2017; Nazarian 2017; Indik 2017; Brignole 2013)
Since many cardiac patients have cardiac implanted electrical devices (CIEDSs), the risk of CMR to the patient and the device must be weighed against the benefit to the patient, in terms of clinical value in optimal management.

Many newer CIEDs are ‘MR conditional’ for thoracic scanning, some only for non-thoracic scanning, and some for both. With adherence to manufacturer’s recommendations and precautions with respect to programming and patient/device monitoring, MR conditional CIEDs do permit safe MR scanning, with a limited amount of data available specific to cardiac MR.

The older ‘non MR conditional’ devices are often amendable to MR scanning at field strength ≤ 1.5 Tesla. However, the presence of a CIED is still generally considered a strong relative contraindication to routine MR examination, and therefore, MR imaging in patients with non-MRI-conditional permanent pacemakers or ICD should be undertaken only if no alternative diagnostic test is available and the potential benefit to the patient clearly outweighs the potential risks. Such an approach warrants informed patient consent, and the scanning protocol requires on site imaging and device management expertise.

Additional non-conditional device materials include combinations of components (even if individually conditional) from various manufacturers that have not been specifically tested together for conditional labeling. Other examples of non-conditional components include epicardial leads, abandoned leads, fractured leads, or an active non cardiac device.
II. ECG Stress Test Alone versus Stress Testing with Imaging

Prominent scenarios suitable for an ECG stress test WITHOUT imaging (i.e. exercise treadmill ECG test) are inferred from the guidelines presented above, often (but not always) requiring that the patient can exercise for at least 3 minutes of Bruce protocol with achievement of near maximal heart rate AND has an interpretable ECG for ischemia during exercise (Wolk 2013):

- The (symptomatic) low pretest probability patient who is able to exercise and has an interpretable ECG
- The (asymptomatic) high global risk patient who is able to exercise and has an interpretable ECG
- The patient who is under evaluation for exercise induced arrhythmia (or long QT interval evaluation when the resting QTc is normal), and coronary artery disease is not suspected (Al-Khatib 2017)
- The patient who requires an entrance stress test ECG for a cardiac rehab program or for an exercise prescription.

Duke Exercise ECG Treadmill Score calculates risk from ECG treadmill alone:

- The equation for calculating the Duke treadmill score (DTS) is: DTS = exercise time in minutes - (5 x ST deviation in mm or 0.1 mV increments) - (4 x exercise angina score), with angina score being 0 = none, 1 = non-limiting, and 2 = exercise-limiting.
- The score typically ranges from -25 to +15. These values correspond to low-risk (with a score of > +5), intermediate risk (with scores ranging from - 10 to + 4), and high-risk (with a score of ≤ -11) categories.

An uninterpretable baseline ECG includes (Fihn 2012):

- Abnormalities of ST segment depression of 0.1 mV (1 mm with conventional calibration) or more
- Ischemic looking T wave inversions of at least 0.25 mV (2.5 mm with conventional calibration)
- ECG findings of probable or definite LVH, WPW, a ventricular paced rhythm, or left bundle branch block
- Digitalis use or hypokalemia
- Resting HR under 50 bpm on a beta blocker and an anticipated suboptimal workload (e.g. rate-pressure product less than 20-25K) could render inconclusive result
- Prior false positive stress ECG

III. Global Risk of Cardiovascular Disease
Global risk of CAD is defined as the probability of manifesting cardiovascular disease over the next 10 years and refers to asymptomatic patients without known cardiovascular disease. It should be determined using one of the risk calculators below. A high risk is considered greater than a 20% risk of a cardiovascular event over the ensuing 10 years. High global risk by itself generally lacks scientific support as an indication for stress imaging (Douglas 2018). There are rare exemptions, such as patients requiring a I-C antiarrhythmic drug, who might require coronary risk stratification prior to initiation of the drug, when global risk is moderate or high.

- **CAD Risk—Low**
  10-year absolute coronary or cardiovascular risk less than 10%.

- **CAD Risk—Moderate**
  10-year absolute coronary or cardiovascular risk between 10% and 20%.

- **CAD Risk—High**
  10-year absolute coronary or cardiovascular risk of greater than 20%.

**Links to Global Cardiovascular Risk Calculators**

*Patients who have already manifested cardiovascular disease are already at high global risk and are not applicable to the calculators.

(D’Agostino 2008; Ridker 2007; McClelland 2015; Goff 2014)

<table>
<thead>
<tr>
<th>Risk Calculator</th>
<th>Link to Online Calculator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reynolds Risk Score</td>
<td><a href="http://www.reynoldsriskscore.org/">http://www.reynoldsriskscore.org/</a></td>
</tr>
<tr>
<td>Pooled Cohort Equation</td>
<td><a href="http://clincalc.com/Cardiology/ASCVD/PooledCohort.aspx?example">http://clincalc.com/Cardiology/ASCVD/PooledCohort.aspx?example</a></td>
</tr>
<tr>
<td>MESA Risk Calculator</td>
<td><a href="https://www.mesa-nhlbi.org/MESACHDRisk/MesaRiskScore/RiskScore.aspx">https://www.mesa-nhlbi.org/MESACHDRisk/MesaRiskScore/RiskScore.aspx</a></td>
</tr>
</tbody>
</table>
**IV. Definitions of Coronary Artery Disease**

(Fihn 2012; Montalescot 2013; Patel 2017; Mintz 2016; Tobis 2007)

1. Percentage stenosis refers to the reduction in diameter stenosis when angiography is the method and refers to cross sectional narrowing when IVUS (intravascular ultrasound) is the method of determination.

2. Coronary artery calcification is a marker of risk, as measured by Agatston score on coronary artery calcium imaging. It is not a diagnostic tool so much as it is a risk stratification tool. Its incorporation into global risk can be achieved by using the MESA risk calculator.

3. Stenoses ≥ 50% are considered obstructive coronary artery disease (also referred to as clinically significant), while stenoses ≤ 50% are considered nonobstructive coronary artery disease (Gerber 2018).

4. Ischemia-producing disease (also called hemodynamically or functionally significant disease, for which revascularization might be appropriate) generally implies at least one of the following:
   
   i. Suggested by percentage diameter stenosis ≥ 70% by angiography; borderline lesions are 40-70% (Fihn 2012; Tobis 2007)
   
   ii. For a left main artery, suggested by a percentage stenosis ≥ 50% or minimum lumen cross sectional area on IVUS ≤ 6 square mm (Fihn 2012; Mintz 2016)
   
   iii. FFR (fractional flow reserve) ≤ 0.80 for a major vessel (Mintz 2016)
   
   iv. Demonstrable ischemic findings on stress testing (ECG or stress imaging), that are at least mild in degree

5. A major vessel would be a coronary vessel that would typically be substantial enough for revascularization, if it were indicated. Lesser forms of coronary artery disease would be labeled as “limited” and not major (i.e. A 50% lesion in a tiny septal or modest size mid PDA would be limited obstructive coronary artery disease.)

6. Microvascular ischemic coronary artery disease, as might be described by a normal FFR (fractional flow reserve) above 0.80 with a reduced CFR (coronary flow reserve less than 2.5), has not otherwise been addressed in this manuscript, because it is very rarely an issue in compliance determinations. However, it would constitute a form of ischemic heart disease.

7. FFR (fractional flow reserve) is the distal to proximal pressure ratio across a coronary lesion during maximal hyperemia induced by either intravenous or intracoronary adenosine. Less than or equal to 0.80 is considered a significant reduction in coronary flow. Newer iterations such as iFR (instantaneous wave free ratio) might supersede basic FFR technology in the near future.

8. New technology is evolving that estimates FFR from CCTA images. This is covered under the separate NIA Guideline for FFR-CT.

**V. Anginal Equivalent**

Development of an anginal equivalent (e.g. shortness of breath, fatigue, or weakness) either with or without prior coronary revascularization should be based upon the documentation of reasons to suspect that symptoms other than chest discomfort are not due to other organ systems (e.g. dyspnea due to lung disease, fatigue due to anemia), by presentation of
clinical data such as respiratory rate, oximetry, lung exam, (as well as d-dimer, chest CT(A), and/or PFTs, when appropriate), and then incorporated into the evaluation of coronary artery disease as would chest discomfort. Syncope per se is not an anginal equivalent (Moya 2009; Shen 2017; Fihn 2012).

VI. Peripheral Arterial Disease/Cerebrovascular Disease

Arterial vascular disease below the renal arteries is generally referred to as peripheral arterial disease, when the ankle brachial index is <0.9 or there is at least 50% vessel diameter narrowing on ultrasound or angiography (Hussain 2018).

Cerebrovascular disease generally refers to a history of TIA (transient ischemic attack) or stroke, or cerebrovascular lesions that put the patient at considerable risk for stroke (Caplan 2018).

There is no evidence to demonstrate that screening all patients with peripheral arterial disease for asymptomatic atherosclerosis in other arterial beds improves clinical outcome. Intensive treatment of risk factors through guideline directed medical therapy is the principal method for preventing adverse cardiovascular ischemic events secondary to atherosclerotic disease in other arterial beds (Gerhard-Herman 2016).

VII. Imaging Surveillance for Cardiotoxic Chemotherapy
(Plana 2014; Zamorano 2016; Maleszewski 2018; Herrmann 2014)

**TTE is the method of choice** for the evaluation of patients before, during, and after cancer therapy. Ideally accuracy prefers that 3D and global longitudinal strain (GLS) are part of the exam, and serum troponin (Tn) should also be measured. However, GLS and Tn might not have been performed, in which case determinations might need to be made with LVEF only. *Serum troponin (Tn) and GLS abnormalities constitute an abnormal assessment of LV function, because their abnormalities frequently herald an imminent fall in LVEF* (Plana 2014; Zamorano 2016).

**CMR** is recommended when TTE has been unreliable and/or candidacy for cardiotoxic chemotherapy based upon LVEF is questionable. MUGA can also be considered when TTE is inadequate and CMR is not available (Plana 2014).

**MUGA** is accurate and reproducible, but lacks information about pericardium and valves, incurs repeated radiation exposure, and is inaccurate during an irregular cardiac rhythm (Plana 2014).

**Surveillance guidelines** are somewhat complex, possibly beyond the scope of this guideline, especially in patients with additional risk factors for LV dysfunction (Herrmann 2014). As with all guidelines, adequate information for complex decisions might be impractical to acquire. However, if the reader requires more rigorous recommendations, they are summarized concisely in the table below. Necessity determinations might not require strict adherence to this table at this time, but it is here to serve as a helpful reference for the reader, if desired.
**TTE Surveillance Strategy for Cardiotoxic Chemotherapy (Optional Information)**
(Plana 2014; Herrmann 2014; Zamorano 2016; Maleszewski 2018)

<table>
<thead>
<tr>
<th>Suspected/Detected LV Status at Baseline, During, or After Completion of Therapy</th>
<th>Type I Anthracyclines: Doxorubicin, Epirubicin, Idarubicin Mitoxantrone (Asnani 2018)</th>
<th>Type II Trastuzumab, Labatinib, Pertuzumab, Sorafenib, Sunitinib, Bevacizumab, Bortezomib **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal: EF is ≥ 55%, troponin is negative, and global longitudinal strain (GLS) &gt; lower limit of normal*</td>
<td>Normal assessment: Assess after a cumulative dose &gt; 200mg/M² (or its anthracycline equivalent) and prior to each additional 50 mg/M², and at completion of therapy, and 6 months later, and for cumulative dose &gt; 300 mg/M² include assessment at 1 year and at 5 years post completion of therapy. (Zamorano 2016)</td>
<td>Normal assessment: Assess every 3 months during therapy and at 6 months post completion of therapy</td>
</tr>
<tr>
<td>Abnormal: any one of:</td>
<td>Abnormal assessment: Assess after every cycle, and reassess for verification 2-3 weeks later if a drop in LV function has been detected/suspected: assess 6 months post completion of therapy, followed by reassessment every 6 months until stable, and for cumulative dose &gt; 300 mg/M² include assessment at 1 year and 5 years post completion of therapy. (Zamorano 2016)</td>
<td>Abnormal assessment: Assess after every cycle, and reassess for verification 2-3 weeks later if a drop in LV function has been detected /suspected: assess 6 months post completion of therapy, and if still not stable reassess every 6 months until stable.</td>
</tr>
<tr>
<td>- GLS reduced ≥ 10-15% below normal (about 20 is normal*, labs vary)</td>
<td></td>
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<tr>
<td>- Troponin positive</td>
<td></td>
<td></td>
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<tr>
<td>- LVEF started &lt; 55%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- During therapy LVEF drops below 55% AND ≥ 5 points for a symptomatic/≥10 points for an asymptomatic patient. (Maleszewski 2018)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* GLS of (negative) 20 is generally normal, but individual labs vary (Collier 2017).
** Imatinib, rarely cardiotoxic, does not require surveillance of LV function (Floyd 2018).
### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ARVD/C</td>
<td>Arrhythmogenic right ventricular dysplasia/cardiomyopathy</td>
</tr>
<tr>
<td>CABG</td>
<td>Coronary artery bypass grafting surgery</td>
</tr>
<tr>
<td>CAD</td>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>CCT</td>
<td>Cardiac CT</td>
</tr>
<tr>
<td>CCTA</td>
<td>Coronary CT angiography</td>
</tr>
<tr>
<td>CHF</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>CMR</td>
<td>Cardiac magnetic resonance (imaging)</td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
<tr>
<td>CTA</td>
<td>Computed tomographic angiography</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>GLS</td>
<td>Global longitudinal strain (measure of left ventricular function)</td>
</tr>
<tr>
<td>HF</td>
<td>Heart failure</td>
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<tr>
<td>LBBB</td>
<td>Left bundle-branch block</td>
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<tr>
<td>LV</td>
<td>Left ventricular</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>MPI</td>
<td>Myocardial perfusion imaging</td>
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<tr>
<td>MR(I)</td>
<td>Magnetic resonance (imaging)</td>
</tr>
<tr>
<td>PCI</td>
<td>Percutaneous coronary intervention</td>
</tr>
<tr>
<td>PET</td>
<td>Positron emission tomography</td>
</tr>
<tr>
<td>RNA</td>
<td>Radionuclide angiography</td>
</tr>
<tr>
<td>RV</td>
<td>Right ventricle</td>
</tr>
<tr>
<td>SE</td>
<td>Stress echocardiography</td>
</tr>
<tr>
<td>Tn</td>
<td>Troponin</td>
</tr>
<tr>
<td>TR</td>
<td>Tricuspid regurgitation</td>
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</table>
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