

The American College of Radiology, with more than 30,000 members, is the principal organization of radiologists, radiation oncologists, and clinical medical physicists in the United States. The College is a nonprofit professional society whose primary purposes are to advance the science of radiology, improve radiologic services to the patient, study the socioeconomic aspects of the practice of radiology, and encourage continuing education for radiologists, radiation oncologists, medical physicists, and persons practicing in allied professional fields.

The American College of Radiology will periodically define new practice parameters and technical standards for radiologic practice to help advance the science of radiology and to improve the quality of service to patients throughout the United States. Existing practice parameters and technical standards will be reviewed for revision or renewal, as appropriate, on their fifth anniversary or sooner, if indicated.

Each practice parameter and technical standard, representing a policy statement by the College, has undergone a thorough consensus process in which it has been subjected to extensive review and approval. The practice parameters and technical standards recognize that the safe and effective use of diagnostic and therapeutic radiology requires specific training, skills, and techniques, as described in each document. Reproduction or modification of the published practice parameter and technical standard by those entities not providing these services is not authorized.

2017 (Resolution 21)*

ACR–NASCI–SPR PRACTICE PARAMETER FOR THE PERFORMANCE OF QUANTIFICATION OF CARDIOVASCULAR COMPUTED TOMOGRAPHY (CT) AND MAGNETIC RESONANCE IMAGING (MRI)

PREAMBLE

This document is an educational tool designed to assist practitioners in providing appropriate radiologic care for patients. Practice Parameters and Technical Standards are not inflexible rules or requirements of practice and are not intended, nor should they be used, to establish a legal standard of care¹. For these reasons and those set forth below, the American College of Radiology and our collaborating medical specialty societies caution against the use of these documents in litigation in which the clinical decisions of a practitioner are called into question.

The ultimate judgment regarding the propriety of any specific procedure or course of action must be made by the practitioner in light of all the circumstances presented. Thus, an approach that differs from the guidance in this document, standing alone, does not necessarily imply that the approach was below the standard of care. To the contrary, a conscientious practitioner may responsibly adopt a course of action different from that set forth in this document when, in the reasonable judgment of the practitioner, such course of action is indicated by the condition of the patient, limitations of available resources, or advances in knowledge or technology subsequent to publication of this document. However, a practitioner who employs an approach substantially different from the guidance in this document is advised to document in the patient record information sufficient to explain the approach taken.

The practice of medicine involves not only the science, but also the art of dealing with the prevention, diagnosis, alleviation, and treatment of disease. The variety and complexity of human conditions make it impossible to always reach the most appropriate diagnosis or to predict with certainty a particular response to treatment. Therefore, it should be recognized that adherence to the guidance in this document will not assure an accurate diagnosis or a successful outcome. All that should be expected is that the practitioner will follow a reasonable course of action based on current knowledge, available resources, and the needs of the patient to deliver effective and safe medical care. The sole purpose of this document is to assist practitioners in achieving this objective.

¹ *Iowa Medical Society and Iowa Society of Anesthesiologists v. Iowa Board of Nursing*, ___ N.W.2d ___ (Iowa 2013) Iowa Supreme Court refuses to find that the *ACR Technical Standard for Management of the Use of Radiation in Fluoroscopic Procedures* (Revised 2008) sets a national standard for who may perform fluoroscopic procedures in light of the standard's stated purpose that ACR standards are educational tools and not intended to establish a legal standard of care. See also, *Stanley v. McCarver*, 63 P.3d 1076 (Ariz. App. 2003) where in a concurring opinion the Court stated that "published standards or guidelines of specialty medical organizations are useful in determining the duty owed or the standard of care applicable in a given situation" even though ACR standards themselves do not establish the standard of care.

I. INTRODUCTION

This practice parameter was revised collaboratively by the American College of Radiology (ACR), the North American Society for Cardiovascular Imaging (NASCI), and the Society for Pediatric Radiology (SPR).

Cardiac magnetic resonance imaging (MRI) and computed tomography (CT) are important noninvasive methods for the assessment of ischemic and nonischemic cardiomyopathies, pericardial disease, cardiac masses, and congenital heart disease. In addition, CT angiography (CTA) and MR angiography (MRA) are well-established noninvasive cross-sectional imaging methods for the detection and assessment of vascular anatomy and a variety of vascular pathologies.

Previous published practice parameters from the ACR have provided practitioners with the educational tools to perform MRA, CTA, and cardiac MR and CT imaging. However, with continued improvements in the fidelity of advanced CT and MRI scanners and increasingly available advanced imaging methods, there is a clear need for new guidelines on the quantitative aspects of CT and MRI for cardiovascular imaging.

Given the rapid development of quantitative cardiovascular CT and MRI, it is anticipated that future versions of this document will evolve as advanced quantification methods are widely adopted into clinical practice.

II. INDICATIONS

Indications for quantification of CT and MRI include, but are not limited to, the following quantitative applications:

1. Characterization and grading of vascular stenosis
2. Measurement of vessel wall thickness
3. Characterization of aneurysmal disease
4. Evaluation of vascular morphology prior to surgical intervention
5. Flow measurement with phase contrast MRI
6. Flow characterization with contrast enhanced time-resolved MRA
7. Characterization of myocardial morphology and function
8. Assessment of pressure gradients across focal stenosis using phase contrast MRI
9. Assessment of volume of myocardial infarction in ischemic heart disease
10. Assessment of volume of hypoperfused myocardium with perfusion imaging
11. Extent of myocardial fibrosis/infiltration in nonischemic cardiomyopathy for assessment of risk of fatal arrhythmias

III. QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

See the [ACR Practice Parameter for Performing and Interpreting Magnetic Resonance Imaging \(MRI\)](#) [1] and the [ACR Practice Parameter for Performance and Interpreting Diagnostic Computed Tomography \(CT\)](#) [2].

IV. GENERAL ASPECTS OF THE PROCEDURE

A. General Aspects of Quantitative Cardiovascular Imaging with CTA and MRA

1. Morphological evaluation
 - a. Cardiac gating
Proper cardiac gating of CT and MR imaging is critical for the generation of diagnostic images for evaluation of cardiovascular morphology and function. There are 2 techniques for synchronizing the electrocardiogram (ECG) signal with the cardiac cycle: prospective and retrospective [3,4].

With prospective cardiac gating, the acquisition is triggered by the R wave and is paused for image acquisition during a specific phase of the cardiac cycle. This has the advantage of offering selective imaging during a specific cardiac phase to reduce motion blurring, optimize visualization of a vascular structure, and, as in the case of coronary CTA, reduce patient exposure to ionizing radiation.

Retrospective gating acquires data throughout the cardiac cycle so that no portion of the cardiac cycle is excluded. This technique is more sensitive to arrhythmia, although with current arrhythmia software, this effect can be minimized.

b. Distance measurements

For CTA and MRA, a widely used approach for morphological evaluation and measurement is to use curved planar images derived from the volumetric data set. One option is delineating the centerline of the vessel to create a curved planar reconstruction of the arterial segment in question. Vendor software allows deconvolution of the vessel, permitting a curved planar view that can be displayed in both cross-sectional and longitudinal projections. An accurate cross-sectional diameter and area measurement of the normal vessel can be obtained using this projection. Measurements of coronary artery diameter can be obtained within a precision of approximately 0.6 mm on CTA and to approximately 1 mm on MRA. Length measurements can be derived curved planar on multiplanar reformatted views.

Pitfalls include inaccurate placement of the centerline by automated software. This most often occurs in small vessels such as the coronary arteries or calf vessels. A centerline that is eccentric or incorporates mural calcification or thrombus does not accurately represent the lumen of the vessel. Artifactual stenoses may be produced by an improper centerline. Thus, it is important that the centerline be verified by an experienced observer to avoid this pitfall. On MRA, gradient nonlinearity can cause in-plane and out-of-plane image distortion that leads to incorrect vessel measurements.

Alternatively, many cardiovascular imagers use standard multiplanar imaging and assess the individual vessels using multiplanar reformats (MPR) perpendicular to the vessel axis in orthogonal planes. This is typically done in an interactive fashion with various segments of the arteries being evaluated sequentially for areas of plaque and stenosis.

c. Cross sectional diameter measurements

Cross sectional diameter measurements are performed using an MPR perpendicular to the vessel axis in orthogonal planes. If an area of dilatation or stenosis is suspected, the area can be quantified using reference measurements from adjacent normal vessel sections. A common practice is to compare luminal diameter that is deemed normal by taking measurements 1 cm proximal to the abnormal vessel section and another measurement 1 cm distal to the stenosis or dilatation on the longitudinal straightened curved planar images. The average diameter of these 2 measurements is used as the reference normal diameter of the vessel. The diameter of the abnormal segment is divided by the reference normal diameter to arrive at a percentage of stenosis or dilatation (percent stenosis or dilatation equals abnormal segment [mm] referenced to the normal segment). Workstation software is available to automate this calculation or it may be calculated manually. In practice, it may be difficult to confidently identify 1 or more reference normal areas because of diffuse calcified and noncalcified plaque. If only 1 reference normal area can be defined (either proximal or distal), this area can be used as a single reference segment with the caveat that it may slightly overestimate or underestimate the true extent of the stenosis or dilatation.

Dilatation of the arteries is often due to positive remodeling and atherosclerosis, although multiple other causes exist, including vasculitis and trauma. In general, an aneurysm is defined by dilatation of an artery to greater than 1.5 times the diameter of the adjacent normal segment. Stenosis is far more common and is usually due to negative remodeling and atherosclerosis but can also be secondary to other causes such as vasculitis and dissection. Because of the limited spatial resolution

(approximately 0.35 mm^3) for CTA of at least 64-slice [5,6], 9 to 10 voxels typically span the entire diameter of a proximal coronary artery lumen, for example. Each pixel represents approximately 10% of the luminal diameter. Thus, overly precise reporting of stenoses is often not appropriate. Generally, a percentage range is used. A typical spectrum might include a stenosis grading of less than 25%, 25% to 50%, 50% to 75%, or greater than 75% [7]. Alternatively, more recent guidelines for coronary CTA suggest the following grading system: 0% (no visible stenosis), 1% to 24% (minimal stenosis), 25% to 49% (mild stenosis), 50% to 69% (moderate stenosis), 70% to 99% (severe stenosis), 100% (occluded) [8,9]. Standardized reporting of coronary CTA with corresponding recommendations is in development and may provide a framework for a further study [10].

In evaluating stenoses on CTA, it is important to distinguish calcification from the opacified lumen to properly define the stenosis and minimize the effects of blooming artifacts. This can be particularly problematic in the evaluation of the anterior tibial artery. It is also a common problem in the interpretation of a coronary CTA. The most common, simple solution is to use a lower window center setting and a wider window width. Most vendors provide software with preset window and level settings that optimize evaluation of calcified arteries. It may also be useful to assess the extent of calcification in both the longitudinal and transverse curved planar reconstructions. MRA is used less frequently to characterize dilation and stenosis because of its spatial resolution. However, an approach similar to that described above can be used with MRA. Calcification is not readily visible on MRI, and therefore blooming effects seen with CTA do not impact MRA. For this reason MRA is often used in peripheral vascular disease, particularly in the setting of severe vascular calcification.

d. Cross sectional area, volume and angle measurements

Measurements of an aneurysm's cross-sectional area can be calculated from longitudinal straightened curved planar reconstructed images using the techniques described in section IV.A.1.c above, and many vendors provide this software. Nevertheless, it is not commonly used because most studies of the accuracy of CTA have correlated it with quantitative catheter-based angiography, which relies on unidimensional measurements. Volume and angle measurement are not commonly performed for CTA and MRA but are very helpful for follow-up of thoracoabdominal aortic aneurysms, iliac artery aneurysm after endovascular repair [11], and endovascular repair.

e. Region of interest characterization (CT only)

Attenuation measurements of the arterial wall can be obtained for plaque characterization. Generally, a region of interest (ROI) is placed on the wall and a Hounsfield unit (HU) measurement is obtained that represents an average pixel value. This measurement can be performed on unenhanced gated CT study, often a calcium-scoring examination, and it has also been attempted with CTA. Optimally, it should be possible to categorize plaques as primarily calcified, fibrous, or fatty in density. Although studies correlating CTA with intravascular ultrasound have shown some ability to distinguish among these 3 plaque densities, in clinical practice it is often difficult to confidently differentiate fibrous and fatty plaques. This limitation likely arises from partial volume averaging and variability of HU measurements among different vendors. Thus, plaque is generally characterized as calcified, noncalcified, or mixed. It is also important to quantify HU measurements in the aorta when they are elevated. Crescent-shaped high attenuation in the aortic wall is seen in intramural hematoma [12], but it is important to repeat the measurements after IV contrast is administered because intramural hematoma does not enhance, and an alternative diagnosis (eg, vasculitis) should be sought if there is enhancement [13].

2. Velocity and flow quantification: Phase Contrast MRI

Velocity and flow quantification with MRI are achieved using phase contrast imaging [14,15]. Phase contrast MRI (PC-MRI) exploits the fact that moving tissue (ie, blood) acquires a phase shift in the presence of velocity encoding gradients. This phase shift is directly proportional to the velocity of the blood as it moves through a magnetic field. With PC-MRI, 2 measurements are typically acquired: the first

with a positive bipolar gradient, the second with a negative bipolar gradient. The resultant image is a subtracted phase map image. Signal from stationary tissue is eliminated, while the only signal that remains originates from moving tissue (ie, blood) and is directly proportional to its velocity.

PC-MRI can be implemented as a breath-hold technique or with free-breathing [16,17]. Breath-holding may be preferred within the thorax or abdomen because of the effects of respiratory motion, but it has been suggested that breath-held PC-MRI may underestimate some measurements, such as pulmonary regurgitation [18], compared to PC-MRI obtained during free breathing. Prospective cardiac gating may be preferred with breath-held PC-MRI because of its more consistent acquisition, although either cardiac gating approach is acceptable. The principal drawback of breath-holding is the restricted acquisition time, which may compromise temporal resolution. Free-breathing PC-MRI permits a longer acquisition, which allows higher temporal resolution, although image quality may be reduced because of respiratory motion artifact. Free breathing PC-MRI may be preferable in uncooperative patients or in the pediatric population. Real time PC-MRI can be used as an alternative, if available.

The most important parameter for PC-MRI is the velocity encoding variable (V_{enc}). V_{enc} is generally given in cm/sec and is the highest and lowest detectable velocity measured by that PC-MRI pulse sequence. The closer the V_{enc} is to the actual velocity, the more accurate the measurement. If the V_{enc} is lower than the maximum velocity being measured, then aliasing will occur. If the V_{enc} is significantly higher than the actual velocity, then signal intensity is reduced and the noise floor is relatively higher, which may reduce the accuracy of the flow measurement. Since V_{enc} is inversely proportional to the amplitude of the magnetic gradient, the lower the velocity being measured and the higher the gradient strength required.

V_{enc} is most commonly encoded in a single direction during a PC-MRI acquisition (ie, unidirectional PC-MRI). The direction of the V_{enc} variable can be altered depending on what is being measured and this will determine slice prescription. In-plane PC-MRI is where the V_{enc} direction is encoded within the plane of the image, either anterior-posterior direction, left-right direction, or superior-inferior direction. In-plane PC-MRI is useful for determining flow direction such as when characterizing the eccentricity of an aortic regurgitant jet on a 3-chamber cardiac orientation. Through-plane PC-MRI is where the V_{enc} is encoded through the plane of the slice. This technique is commonly used for measuring velocity and flow, and it is important that the through plane imaging slices be directly orthogonal to the flow being measured. V_{enc} can be also encoded in 3 directions (x, y, and z) during a single acquisition (ie, tridirectional PC-MRI) [19]. Since more time is needed to acquire the additional directions, imaging times are long and therefore temporal resolution may be compromised with a breath-hold acquisition.

a. Direction

In its most basic application, PC-MRI can be used to visualize flow direction. This can be achieved with in-plane PC-MRI where the imaging slice is chosen to match the region of interest. Precise selection of V_{enc} is less important for this application as long as the V_{enc} is above the peak velocity being measured. Accurate slice orientation is essential, especially in regions where position may be affected by respiratory variations (eg, in a portal venous system [20]).

b. Velocity

Through-plane PC-MRI is used for accurate measurement of velocity [21]. It is essential that the through-plane slice be positioned directly orthogonal to the flow so that the true velocity is being measured and not a vector component of the true velocity. In-plane PC-MRI may be useful for planning the setup of the through-plane slice. For example, a sagittal oblique in-plane PC-MRI of a thoracic aortic coarctation will depict the direction of the high velocity jet inferior to the stenosis so that the through plane slice can be prescribed directly orthogonal to the flow jet. Preliminary in-plane PC-MRI has the added advantage of providing an assessment of actual velocity so that the V_{enc} can be increased on the through-plane slice if aliasing occurs. In order to ensure true orthogonal positioning, the through-plane slice should or must be set up from at least 2 different orientations. In-plane velocity measurements can also be used for estimation of peak velocity [22] but care must be taken to

ensure that the imaging plane is indeed aligned with the direction of the flow, which often may be eccentric and/or moving, as in the case of a valve. Velocity is measured by drawing a ROI that includes the entire lumen of the vessel being evaluated. Peak velocity is the pixel with the highest signal intensity in the direction of interest within the ROI. Average velocity represents the average of all the pixels within the ROI. Pressure gradients (mmHg) across a focal stenosis can be estimated using the modified Bernoulli equation, $P=4V^2$, where V is the peak velocity in m/sec. If the peak velocity is measured at multiple points along a vessel, such as above and below a coarctation, then the pressure gradient between those points can be estimated [23]. It is important to note that the modified Bernoulli equation does not apply for long segment stenoses. In some instances, highly turbulent flow may result in intravoxel dephasing that results in the absence of signal, which results in inaccurate peak flow determination using PC-MRI. Finally, assessment of the shape of the velocity-time curve may be helpful in conditions where there is dampened flow, such as in pulmonary hypertension.

c. Flow

Blood flow can be calculated from the velocities measured by PC-MRI. It is optimal to acquire the velocity measurements directly orthogonal to the direction of flow; therefore, using in-plane PC-MRI to set up the through-plane slice is very helpful. For correct calculation of flow, the ROI needs to be accurately drawn within the flow region since the ROI area will determine the final flow value [24]. It is essential that spatial resolution is set to match the vessel of interest. If spatial resolution is too low, flow and velocity will be underestimated because of partial volume effects. Similarly, temporal resolution (ie, time per frame) must be adequate for measuring flow in the vessel of interest. For example, high flow vessels such as the thoracic aorta require higher temporal resolution.

3. Time resolved angiography

a. Technical aspects

Time-resolved angiography refers to rapid frame rate angiography where images are acquired per unit of time such that sequential filling and draining of vascular structures can be assessed. Time-resolved angiography can be carried out using either MRI or CT. Time-resolved MRA (TR-MRA) refers to ultrafast MRA in which 3-dimensional (3-D) data sets are acquired every 1 to 3 seconds. In order to speed up the acquisition, conventional MRA is implemented with acceleration strategies such as parallel imaging or view sharing (ie, TRICKS, TWIST) [25-30]. If TR-MRA is implemented as a 2-D acquisition, then frame rates of several images per second can be achieved. Time-resolved angiography with CT usually involves acquiring a single slice or stack of slices (with multi-detector CT) every second as a contrast bolus is injected and is the preferred method for bolus timing with CT.

b. Applications

TR-MRA, in its most basic use, can be used as a bolus timing acquisition for measuring contrast transit times for conventional MRA. TR-MRA can also be used to visualize, in real-time, the passage of a bolus of contrast through different portions of the circulation. For example, TR-MRA may be the method of choice for imaging the pulmonary vasculature because it depicts sequential filling of pulmonary arteries, pulmonary veins, and thoracic aorta, which has particular utility for assessing congenital heart disease or aortic dissection. The passage of a contrast bolus can also be quantified by placing ROIs in different vessels to measure its time to peak enhancement. For example, the absolute transit time between the pulmonary trunk and thoracic aorta is elevated in conditions such as pulmonary hypertension and congestive heart failure [31-33]. Similarly, relative contrast transit times between different vascular territories can be expressed as ratios. Contrast transit times between the left heart and right heart can be calculated in order to better characterize intra-cardiac and extra-cardiac shunts.

4. Quantitative techniques specific to cardiac MRI and CT

Echocardiography, notably transthoracic echocardiography, remains the primary screening tool for evaluating cardiac morphology and function [34]. However, evaluation with echocardiography relies on operator skill, and variability in scanning technique may contribute to intraobserver and interobserver variation [35]. Such variation is notably higher with echocardiography than with MRI [36].

Cardiac-gated MRI and CT can provide images of the heart chambers throughout the entire cardiac cycle; thereby enabling quantitative measurement of myocardial wall thickness and mass, chamber sizes, and myocardial function that are similar and arguably more reproducible than that achieved by transthoracic echocardiography [37,38]. Moreover, the intravenous administration of contrast agents enables the determination of myocardial perfusion and myocardial delayed enhancement on MRI and more recently on CT [39].

Many of the measurement standards used for clinical cardiac CT and MRI are derived from those of echocardiography [34]. It is important to note that specific thresholds of measurement for healthy individuals vary based on body habitus [40-42], race [40-42], gender [40-46], and age [45,47,48]. Moreover, imaging technique itself can result in differences in measurement. For example, the actual pulse sequence used for cardiac MRI (eg, fast gradient echo versus steady state free precession [49-51]) may affect left ventricular measurements, although field strength (1.5T versus 3T) does not appear to have any significant influence [50].

a. Myocardium

i. Wall thickness

Myocardial wall thickness is traditionally measured on end-diastolic images. End diastole can be defined at the onset of the P-wave but is preferably defined as the frame after mitral valve closure or the frame in the cardiac cycle in which the cardiac dimension is largest. In healthy adults, end-diastolic left ventricular thickness is typically between 6 and 12 mm [34,45]. To minimize volume averaging effects, image acquisition is typically performed in a plane perpendicular to the wall being measured. For the left ventricle this is typically performed on short axis images. Special regions such as the apex are better suited for evaluation on 2-chamber and 4-chamber long-axis views. The basal anteroseptal segment is best evaluated on a 3-chamber view.

ii. Myocardial mass (left ventricular mass)

The myocardial mass of the left ventricle (LV) can be determined by measuring end-diastolic LV myocardial volume and multiplying this by the specific gravity of myocardium (1.05 g/ml) [41]. The myocardial volume of the LV can be determined by summing the area of the myocardium from a stack of images that covers the entirety of the LV and multiplying this by the thickness of each slice (and slice gap if present). The difference between endocardial and epicardial tracings represents myocardium. The area of the myocardium can be calculated by subtracting the area of the LV's chamber (endocardial tracing) from the area of the LV (epicardial tracing). Note that the papillary muscles are typically excluded from the endocardial border (ie, included within the volume of the chamber) as exclusion of the papillary muscles reduces postprocessing time requirements by obviating a separate trace of the papillary muscles [52]. However, in some specific cases, such as in patients with hypertrophic cardiomyopathy, it may be useful to perform an additional trace of the papillary muscles and include their mass in the LV myocardial volume [52]. In hypertrophic cardiomyopathy, the papillary muscles are relatively larger, and their exclusion would underestimate overall myocardial mass as well as overestimate the LV diastolic volume and underestimate the LV ejection fraction [53].

b. Cardiac chambers

i. Ventricular volumes can be measured linearly using short and long axis dimensions but are more commonly measured in terms of volume. When quantifying the LV using 2-dimensional (2-D) linear measurements, the LV's internal diameters are measured from the endocardium of the anteroseptum to the endocardium of the inferolateral wall at the midventricular level. Left and right ventricular volumes can be best measured using a modified Simpson method whereby the ventricular chamber volume is determined by the sum of the endocardial area multiplied by the slice distance using short axis or long axis images [54-56]. CT provides added flexibility for postprocessing in that ventricular volume calculations can be made quickly using the volumetric data [57] and using advanced region growing post-processing software based on density for fast, accurate determination of chamber contours [58]. It is often helpful to index these values to body surface area (BSA) or to calculate the ratio of right ventricle (RV) to LV size as an assessment of RV enlargement. LV and RV measurements can be important particularly in the growing number of adult patients with congenital heart disease who require lifelong CT and/or MRI surveillance [59,60].

ii. Atria

There are few CT and MRI studies reporting normal left and right atrial measurements. Echocardiographic standards, however, suggest that the normal left atrial anterior-posterior dimension is less than 4.0 cm during end-systole and that the normal minor axis (ie, transverse) right atrial dimension is less than 4.5 cm [34]. However, the atria, especially the right atrium, are often oblong or unusually shaped, making specific diameter measurements less useful as a determination of enlargement. However, atrioventricular valvular dysfunction (eg, mitral or tricuspid insufficiency or stenosis) will often be present with atrial enlargement.

c. Myocardial function

i. Ventricular ejection fraction

Ventricular ejection fraction (EF) is defined by the following equation [41,54,55]: $EF[\%] = 100 \times [EDV - ESV]/EDV$, in which EDV is end-diastolic volume and ESV is end-systolic volume. EDV and ESV are determined using the modified Simpson method described above by drawing endocardial tracings on short axis slices of the heart, from the atrioventricular valve plane (base of the heart) to the apex, at end-diastole and end-systole. Because the length of the ventricle is shorter at end-systole than in end-diastole, it is often necessary to trace an endocardial contour on an additional end-diastolic slice.

There is variability in how endocardial contours are drawn. Whether one includes or excludes the papillary muscles and ventricular trabeculae from the blood pool volume is a matter of choice. In normal patients or in those with coronary artery disease it has been shown that inclusion or exclusion of the papillary muscles in ventricular volume measurements has no significant difference in EDV or ESV measurements [52]. Inclusion or exclusion of the papillary muscles may result in clinically relevant differences in EDV and ESV values in patients with specific pathologies [61] such as hypertrophic cardiomyopathy.

Volumetric and EF measurements by MRI and CT have been shown to be very comparable [58,62]. An individual physician, or by consensus an imaging laboratory, should establish a convention by which epicardial contours will be drawn in all patients. By establishing this standard, one will have confidence in the accuracy, reproducibility, and stability of functional measurements when measuring cardiac function in patients returning for repeat examinations.

The ventricular chambers are bounded by the atrioventricular valves (ie, mitral or tricuspid valves) and the ventriculoarterial valves (ie, aortic or pulmonic valves). The atrioventricular valve

plane defines the base of the ventricular chamber and is therefore a well-defined boundary of the ventricle. The decision of how much of the ventricular outflow tract to include (ie, how close to the ventriculoarterial valve each endocardial contour tracing extends) varies. Some investigators include and others exclude the left and right ventricular outflow tracts, although others draw endocardial contours up to the aortic and pulmonic valve planes.

In addition to EDV and ESV, the following functional parameters are easily calculated from the same short axis image data after drawing endocardial contours:

- Stroke volume ($SV = EDV - ESV$)
- Ejection fraction ($EF[\%] = 100 \times [EDV - ESV]/EDV$)
- Cardiac output ($CO = SV \times \text{heart rate}$)
- Cardiac index ($CI = CO / \text{body surface area (BSA)} = SV \cdot \text{heart rate} / BSA$)
- Myocardial mass (grams), which is determined when epicardial borders are drawn on end-systolic slices in addition to the endocardial contours
- End-diastolic volume index ($EDVI = EDV/BSA$)
- End-systolic volume index ($ESVI = ESV/BSA$)

Indexing of measurements (eg, cardiac output, cardiac index (CI): myocardial mass, myocardial mass index, end-diastolic volume, end-diastolic volume index, or end-systolic volume index) to body surface area and/or body mass index (BMI) is often helpful clinically to account from differences in patient habitus and size.

ii Wall motion

Although there are a variety of methods for quantitative assessment of wall motion, the visual assessment of cine images remains the standard for wall motion assessment [63]. Wall motion can be visually assessed during systole as normal, hypokinetic (decreased wall motion), akinetic (no wall motion), or dyskinetic (paradoxical motion or reversal of wall motion, eg. aneurysm). In some circumstances it may be helpful to further subdivide hypokinesis into mild, moderate, and severe hypokinesis.

Assessment of myocardial wall motion can be performed during rest. For the assessment of patients with suspected coronary artery disease, however, wall motion assessment during pharmacologic stress using an inotropic medication (eg, dobutamine) is often helpful as significant coronary disease may not be demonstrated in the resting state. For stress wall motion assessments, regional wall motion during stress is compared with resting wall motion, typically on a segment-by-segment basis. Recent meta-analyses and large reviews have shown favorable performance of MRI to detect significant CAD compared to cardiac stress scintigraphy [64-66].

d. Myocardial perfusion

Myocardial perfusion imaging is most commonly performed with MRI [64], but more recently CT has shown promise as well, particularly with dual-energy technique [67-71]. Myocardial perfusion imaging is most typically performed during administration of a pharmacologic vasodilator stress agent (eg, adenosine, dipyridamole, or, more recently, regadenoson [72]), and enhancement of myocardium using rapid T1-weighted images is evaluated over time. This assessment is typically performed using a series of rapidly acquired short axis T1-weighted images that enables visual assessment of regional differences in enhancement. Enhancement of each myocardial region reflects perfusion of specific coronary arterial vascular territories. Similar to stress wall motion evaluation, a meta-analysis has shown stress myocardial perfusion MRI to have a high sensitivity (90%) and specificity (81%) for detecting significant coronary artery disease ($\geq 50\%$ arterial diameter stenosis).

e. Myocardial delayed enhancement imaging

Myocardial delayed enhancement (MDE) also called late gadolinium enhancement, delayed gadolinium enhancement, or delayed contrast enhancement imaging—is a useful tool for assessing myocardial tissue [73,74]. Imaging is typically performed using MRI 10 to 20 minutes following the intravenous injection of a gadolinium-chelate contrast agent (eg, 0.2 mmol/kg cumulative dose) in short axis views and often in supplemental long-axis and/or 4-chamber views. On delayed imaging, abnormal regions of myocardium appear brighter than adjacent normal myocardium and are therefore often termed “hyperenhancement.” The underlying mechanisms for hyper-enhancement are varied and not fully understood but reflect the relative faster washout of contrast in normal myocardium and prolonged retention of contrast in the abnormal tissue.

Hyperenhancement on MDE imaging was initially reported in the setting of myocardial infarction in which infarcted or nonviable myocardium is hyperenhanced [73,74]. Hyperenhancement typically begins in the subendocardial region, as this represents the end-vessel or “at risk” territory of the myocardium as coronary arteries originate from the epicardial surface of the heart and dive deep into the subepicardium, mesocardium, and ultimately into the subendocardium.

Hyperenhancement of myocardial infarction is seen in both acute and chronic myocardial infarction. The segmental transmural extent of the hyperenhancement has been shown to correlate with the likelihood for functional improvement following a coronary revascularization procedure. Transmurality of hyperenhancement is best characterized in quartiles, as less than 0% to 25%, 26% to 50%, 51% to 75%, or 76% to 100%. The likelihood of benefit from a revascularization procedure is high if there is little or no hyperenhancement (ie, entirely viable myocardium) and very low if there is transmural enhancement (100%). Generally, myocardial segments with less than 50% hyperenhancement on MDE will benefit from a coronary revascularization procedure since they retain sufficient viable myocardium to respond favorably to revascularization efforts [74].

More recently, the use of CT for MDE imaging has shown promise for myocardial characterization, notably for identification of myocardial scar, a known potential substrate for ventricular arrhythmia, the most concerning being ventricular tachycardia, which is associated with increased risk for sudden cardiac death [75,76].

f. T1-mapping and Extracellular volume fraction mapping MRI

Native T1 relaxation time (ie, T1-mapping) and extracellular volume fraction (ECV) differences in normal and fibrotic myocardium may be used to detect and quantify myocardial disease such as myocardial infarction and nonischemic cardiomyopathies. These techniques may be particularly helpful for identifying diffuse myocardial processes, such as diffuse myocardial fibrosis, which may not be evident using other MR methods. Initial experience with these novel T1 mapping techniques suggest the potential for these techniques to reliably image diffuse myocardial disease and may allow earlier detection and perhaps treatment of myocardial disease, enabling earlier treatment [77-80]. Evaluation of the cellular and extracellular interstitial compartments of the myocardium may be prognostically important [81].

g. Myocardial segmentation and nomenclature

In 2002, the American Heart Association [82] suggested a standard reporting nomenclature for cardiac imaging studies (nuclear medicine, echocardiography, MRI, and CT) that is based on a 17-segment heart model in which the myocardial segments are defined by their location relative to the long axis (basal, mid, or apical) and circumferential location at each location. There are 6 segments (anterior, anteroseptal, inferoseptal, inferior, inferolateral, and anterolateral) at both the basal and

midventricular levels, 4 segments (anterior, septal, inferior, and lateral) at the apical level, and a single apex region to compose the total 17 segments of the left ventricle.

This segmental nomenclature is intended for regional descriptions of cardiac wall motion, myocardial perfusion, and myocardial delayed enhancement.

h. Calcium scoring

Coronary calcium scores were first reported over 20 years ago by Agatston et al. [83] using electron-beam CT whereby coronary calcium lesions with >130 Hounsfield units (HU) were assessed using a region of interest. The area of each calcified coronary lesion was then multiplied by a weighting factor based on the peak HU measured within the lesion (weighting factor = 1: 130 to 199 HU; weighting factor = 2: 200 to 299; weighting factor = 3: HU; 300 to 399 HU; weighting factor = 4: ≥ 400 HU). The Agatston score is achieved by adding all the calcium scores for each region.

Two other methods for measuring coronary calcium are the volume score and the mass score [84]. The volume score reflects the volume of calcium above the threshold; the mass score uses a phantom to calibrate the mass (mg) of coronary calcium above the threshold. In a large cohort study of 11,490 individuals, the Agatston, volume, and mass scores were found to be equally accurate for calcium scoring, and no single method was deemed superior in terms of reproducibility of results from consecutive scans in a patient [84].

B. Applications of Quantitative Imaging to Specific Disease Entities

1. Aneurysm (primarily aorta): CTA and MRA

a. Initial diagnosis and description

Location, involved anatomy, size, morphology/configuration, volume, and attenuation (ROI) of mural thrombus

The location and involvement of the ascending thoracic aorta, transverse arch, descending thoracic aorta, and juxtarenal and infrarenal abdominal aorta will determine potential repair, and their respective diameters should be reported. The largest diameter is the most important one to be reported. The aneurysm diameter can only be estimated from axial images. When there is angulation in the vessel, measurements from MPRs orthogonal to the arterial axis are the most accurate. CPR with centerline tracing as denoted for coronary artery assessment can similarly be performed for aortic measurements. If workstations for MPRs are not available, then measurements of the vessel perpendicular to the axis of the vessel (ie, shortest in plane diameter) can be made from the axial images but are not preferred. It may also be useful to describe or report aneurysm length. If the length of the aneurysm is reported, it should be clearly identified as the length to avoid confusion with the diameter of the aneurysm. The aneurysm morphology (fusiform, saccular, “onion” or “tulip” bulb from effaced sinotubular junction) as well as mural thrombus volume should be evaluated and described. A fissure or dissection within the intraluminal thrombus may predict a higher risk of rupture [85]. Hyperdense (CT) or hyperintense (MR) thrombus within the intraluminal thrombus may suggest acute hemorrhage within the thrombus and may suggest impending rupture [86-88].

b. Surveillance

i. Role of noncontrast CT and MRI for surveillance of aneurysmal disease

In the surveillance of an aortic aneurysm, the diameter and rate of growth of the diameter should be reported. They can be evaluated on noncontrast or contrast enhanced imaging. MR or CT can be used; however, the spatial resolution of CT and the standardization between different CT

scanners have generally led to CT becoming the standard surveillance test, particularly in older patients. In younger patients or patients with small aneurysms, an ultrasound examination may be used with the understanding that if there is growth of the aneurysm, a CT or MRI scan can be obtained in order to best assess the precise size and characteristics of the aneurysm prior to treatment. There is no consensus algorithm for the surveillance of patients with aortic aneurysms.

ii. In baseline and follow-up imaging studies it is helpful to make aortic measurement at conventional locations in order to facilitate comparison. It is typical to make measurements at the following locations:

- Aortic annulus
- Sinuses of Valsalva at the level of the left main coronary artery origin from the left sinus
- Sinotubular junction
- Ascending aorta at the level of the right pulmonary artery
- Transverse aortic arch between the left common carotid and subclavian artery origins
- Aortic isthmus (site of the ductus ligament insertion)
- Descending aorta at the level of the right pulmonary artery
- Diaphragmatic hiatus
- Celiac plexus and/or superior mesenteric artery origin
- Renal artery origin
- Infrarenal aorta midway between renal artery origins and the aortic bifurcation
- Aortic bifurcation
- Common iliac artery diameters

c. Presurgical planning

i. Endostent and open repair

An aortic aneurysm may be repaired either in an open surgical fashion where a graft replaces the aneurysm or in an endovascular fashion where an endostent is deployed to exclude the aneurysm lumen. A number of cases may require a hybrid technique where both techniques are used.

ii. Location, size, volume, angles, areas, access via femoral/iliac arteries, etc

The evaluation for and endovascular treatment of aortic aneurysms requires several important measurements and observations. The lengths of the nondilated aorta proximal and distal to the aneurysm are termed the proximal neck and distal neck of the aneurysm, respectively. The diameter and length of the proximal and distal neck determine the possibility and long term success of an endovascular repair. The angulation, quality of the aneurysm neck (calcification, thrombus), and relationship to nearby branches from the aorta are also factors involved in an endovascular repair.

For a descending thoracic aortic aneurysm, the distance between the left subclavian artery or left common carotid artery to the beginning of the aneurysm determines the proximal neck length. The distance between the distal aspect of the descending thoracic aortic aneurysm and the visceral vessels defines the distal neck. In an abdominal aortic aneurysm (AAA), the extent of the aneurysm into the iliac vessels determines the length and distal diameter of the bifurcated grafts used in endovascular abdominal aneurysm repair (EVAR). The length from the proximal neck to the aortic bifurcation is also important for stent placement planning. These lengths can be estimated on axial imaging using table position, but a centerline measurement is preferred and considered the most accurate method. The centerline measurement is based on the true perpendicular vessel center acquired from the double oblique MPR technique. Endovascular repair may require the delivery of large devices from the femoral approach into the aorta. The diameter, tortuosity, and degree of calcification of the iliac and femoral vessels will usually predict the successful delivery of the graft devices.

d. Postsurgical monitoring guidelines

In contrast to patients who undergo surgical repair of an aortic aneurysm and may receive a single follow-up scan, patients who have undergone endovascular aneurysm repair with endografts require lifelong monitoring. There are no established guidelines for surveillance imaging post endovascular repair. Most patients receive a CT examination with intravenous contrast media to assess the aorta and graft and the possibility for endoleaks within the first 3 months after the repair. Endoleaks represent arterial flow into the aneurysm sac [89]. If there is enlargement of the endosac (excluded aortic lumen) from an endoleak, the aneurysm remains at risk of rupture. Therefore, aneurysm diameter measurements and possible increase in sac diameter must be reported. Changes in endosac volume, however, may be a more sensitive measure of sac enlargement [11]. Sac volumes as well as sac diameters may be reported on noncontrast imaging and may be helpful to identify an enlarging sac or shrinking sac before there are changes in sac diameter [11].

e. Other sites of aneurysmal disease

i. Popliteal

Popliteal artery aneurysms as well as a number of peripheral aneurysms may not only be a risk for rupture but may also serve as a source of thrombi and subsequent distal embolization. The description of popliteal artery aneurysm should include not only the diameter and length of the aneurysm but also the presence and amount of thrombus within the aneurysm, as well as the patency of the distal (ie, tibial) vessels at risk of embolization.

ii. Renal, splenic, mesenteric, great vessels, upper extremities

The size and location of an aneurysm, number of inflow and outflow vessels, and the amount of tissue perfused by the vessel are important in the determination as to when and how the aneurysm should be repaired or excluded. Pseudoaneurysms are associated with a higher risk of rupture.

2. Dissection, intramural hematomas, penetrating ulcer (primarily aorta): CTA and MRA

Penetrating atherosclerotic ulcers, intramural hematomas, and aortic dissections are closely related diagnoses discussed with the term “acute aortic syndromes”. CTA is more commonly used in the acute setting because of its availability and faster image acquisition, although MRA examinations are common particularly in surveillance and follow up of these patients. The use of noncontrast CT prior to a contrast enhanced study is essential for the diagnosis of intramural hematoma.

a. Initial diagnosis and description

i. Location, involved anatomy, size, volume ROI in IMH (CT)

The classification of aortic dissections into Stanford Type A (involving the thoracic aorta proximal to the left subclavian origin) or Type B (involving only the thoracic aorta distal to the left subclavian vessel origin) should be reported. Dilatation of the aortic diameter and further extension of the dissection flap are important to recognize and report. Additionally, the location and number of fenestrations as well as the relative size and density of the false and true lumen may be helpful in determining the possible need for treatment. The extent of a penetrating ulcer and possible involvement into nearby branches should be reported. Aortic size and size of true and false lumen should be reported.

The noncontrast acquisition allows depiction of the hyperdensity of the acute hemorrhage within the wall of the vessel. T1-weighted MRI sequences can also be used to depict methemoglobin in the acute and subacute intramural hematoma. With intramural hematoma and dissection the extent of hematoma (both length and width) and possible branch vessel involvement should be

noted. Imaging will document the existence of vessel rupture. In aortic dissection, the diameter and flow within the true and false lumen should be reported.

ii. Involvement of end organs (eg, renal and mesenteric arteries)

The patients should be evaluated for possible end organ malperfusion, as this finding may necessitate urgent therapy.

b. Surveillance

Surveillance of patients with known high risk conditions associated with thoracic aortic dilatation and dissection require meticulous evaluation with MRA and CTA. These patients require centerline diameter measurements at the aortic annulus, sinus of Valsalva, sinotubular ridge, ascending aorta, and other involved areas.

c. Presurgical planning

Vessel diameters and treatment length must be quantitated. These will help determine if an endovascular repair can be performed and the diameter of the grafts needed. The amount of angulation of the arch, length from the arch vessels (left subclavian and left carotid artery) and from the visceral vessels, and the status of the vertebral arteries should be reported. Possible sites of endovascular access, including subclavian arteries and common femoral and iliac arteries, should be assessed.

d. Postsurgical monitoring

Early after endovascular repair, CTA is most commonly used to determine the presence of endoleak as well as possible complications such as stent migration or fracture. Generally, lifelong annual CTA scans are needed to assess changes in the aortic diameter after repair. MRA is less commonly used because of its limited direct visualization of stent grafts, but it is an excellent alternative in patients with contraindications to CTA.

3. Atherosclerotic stenotic disease: CTA and MRA

a. Location, extent (length), severity (stenosis grading)

Atherosclerosis is a progressive systemic disease characterized by accumulation of lipid, fibrous tissue, and occasionally hemorrhages in the large arteries. Clinical manifestations are primarily due to ischemia related to stenotic disease or from rupture of aneurysms or emboli from associated in situ thrombus. CT and MR accurately depict the location, severity, and length of arterial stenoses or aneurysms. Quantitative evaluation of the stenoses is heavily dependent on the spatial resolution of the CT or MR technique used. Spatial resolution determines the level of detail that can be evaluated and the accuracy of quantitative measurements.

When atherosclerotic plaque is present, its precise anatomic location should be described and the severity and length of stenosis reported. The severity of stenosis is graded as a percentage of diameter reduction; the diameter of the stenotic segment is divided by an adjacent normal diameter to determine the percentage of stenosis (or dilatation). However, in smaller vessels, limitations in spatial resolution may preclude accurate use of percentage reduction, and qualitative analysis is used (mild, moderate, or severe). In smaller vessels, such as the infrapopliteal arteries of the leg, calcified atherosclerotic plaque may also cause artifactual narrowing of the apparent residual lumen because of blooming and beam hardening on CT; this should be taken into account during stenosis determination in order to avoid overestimating the degree of diameter reduction. MRA may be the preferred

imaging modality in such patients. The length from the beginning to the distal most aspect of a stenosis should be described; this will influence the choice of potential intervention.

b. Typical sites of disease

i. Renal, mesenteric, aorto-iliac-femoral, runoff

Renal artery atherosclerosis leads to renal failure and renovascular hypertension. Aortic or proximal renal artery plaques are the usual culprit when atherosclerosis causes renal failure, whereas stenosis of the proximal or more distal main renal artery or its branches leads to hypertension [90]. Both CTA and MRA have high sensitivity and specificity for depicting atherosclerotic narrowing of the entire renal artery and often the segmental branches [91,92].

Mesenteric occlusive disease is frequently due to atherosclerosis of the celiac axis, superior mesenteric artery, and inferior mesenteric artery. Accurate detection of proximal mesenteric arterial stenosis is possible with both CTA and MRA, and precise description of the site, length, and diameter reduction should be reported.

The abdominal aorta is a common site of atherosclerosis. The infrarenal aorta is generally considered aneurysmal if it is 3 cm or greater in diameter, “ectatic” if it is between 2 and 3 cm in diameter [93], and considered stenotic if the lumen is less than 1 cm. Imaging studies are important in determining the aneurysm size, detecting the involvement of branch vessels, and depicting any associated significant stenoses involving the abdominal visceral or extremities. Preoperative imaging for potential endovascular repair (EVAR) of AAA is based on aneurysm morphology and access vessel size and patency [94]. After stent placement, imaging is used to monitor aneurysm diameter and volume, detect and classify endoleaks, and evaluate morphologic details of the stent graft [95].

In the iliac and lower extremity arteries, atherosclerosis may lead to claudication or limb threatening ischemia. Depiction of the anatomic location, length, and severity of stenosis is critical in determining if medical management, intervention, or surgery is best.

c. Other sites: great vessels, subclavian, carotids

The thoracic aorta may become aneurysmal secondary to extensive atherosclerosis, connective tissue disease, aortitis, dissection, or poststenotic changes. Accurate short-axis measurement of the aortic diameter is determined using multiplanar techniques as diameters determined on axial images may be inaccurate. The presence of aortic atheromata, ulceration, intramural hematoma, and dissection can all be accurately depicted and described using current cross-sectional techniques. Atherosclerosis of the proximal internal carotid artery leads to cerebrovascular ischemia and stroke. Ultrasound, CTA, and contrast-enhanced MRA (CE-MRA) are all highly sensitive for detecting internal carotid artery stenosis. Depiction of a stenosis with a diameter reduction of 70% to 99% is most commonly used for intervention.

d. Role of phase contrast MRI

i. Visualization of flow reversal, waveforms (tardus-parvus), etc

Most current CT and MR angiographic techniques rely solely on the morphologic assessment of the vasculature. Phase contrast (PC) MR angiography assesses the hemodynamic consequences of an arterial lesion. PC flow quantification is a valuable, versatile tool in the noninvasive evaluation of flow characteristics within almost any vascular bed. It accurately depicts quantitative flow profiles depicting velocity, volume, rate, and direction. PC imaging can depict a tardus-parvus

phenomenon distal to a high-grade stenosis, often adding specificity to other MR angiographic methods.

- ii Hemodynamic significant stenosis (eg, renal artery MRA with signal dropout)
The hemodynamic significance of a stenosis can be assessed using a phase contrast MR flow profile, which may depict a delay or loss of the early systolic peak or a signal void [96]. A signal dropout on PC MRA is seen when a stenosis is hemodynamically significant because of the presence of turbulent flow and intravoxel dephasing resulting from a broad spectrum of intravoxel velocities [97]. Cine phase-contrast MRI flow quantification techniques in combination with contrast-enhanced MRA can accurately detect and determine the degree of renal artery stenosis [96,98].
- iii. Estimation of pressure gradients
Pressure gradients across an arterial stenosis are used to determine its hemodynamic significance and therapy. Peak flow velocity is determined on phase contrast MR imaging. Pressure gradients across short/focal stenosis can then be approximated using a modified Bernoulli equation, $\Delta P = 4V^2$, where ΔP is the peak pressure gradient in millimeters of mercury and V is the peak blood flow velocity in meters per second.

4. Embolic disease: CTA and MRA

a. Pulmonary embolus (acute)

Acute pulmonary embolism (PE) increases the pulmonary arterial pressure, which may progress to right heart failure and circulatory collapse. Right ventricular dysfunction is a marker for adverse outcome in patients with acute PE [99,100]. The ratio of the right ventricle (RV) to left ventricle (LV) diameters is an accurate sign for RV dysfunction [101,102]. Other signs have been described, including bowing of the interventricular septum and reflux of contrast medium into the inferior vena cava (IVC). The sizes of the azygous vein, superior vena cava, and pulmonary artery are also indirect measures of right heart dysfunction and pulmonary hypertension [103].

b. Pulmonary embolus (chronic)—see the [ACR–NASCI–SPR Practice Parameter for the Performance and Interpretation of Cardiac Magnetic Resonance Imaging \(MRI\)](#) [104].

5. Vasculitides (infectious and inflammatory): MRA and CTA

MRA and CTA are excellent methods to evaluate the presence, severity, and extent of vasculitides such as, but not limited to:

- Takayasu arteritis
- Giant cell arteritis
- Infectious arteritis
- Kawasaki disease
- Autoimmune vasculitis (eg, Lupus, Behçet syndrome)
- Phakomatoses (eg, neurofibromatosis) [105,106]

MRA and CTA are cross-sectional methods that have the unique advantage of not only evaluating for luminal narrowing but also allowing direct visualization of the vessel wall. In general, direct visualization of vasculitis with CTA and MRA is limited to processes involving large vessels such as the aorta and its branches. Vasculitis of medium and small vessels may be more challenging related to the spatial resolution of these imaging methods, and evaluation of these entities may be indirect, related to tissue damage caused by the vasculitis.

a. Location, extent, and severity of luminal narrowing and/or aneurysmal dilatation

i. Stenosis grading (stenotic disease)

Luminal narrowing/stenosis is an important sequela of large vessel vasculitis and is responsible for a large percentage of morbidity related to vasculitis. Quantification of stenosis in vasculitis is identical to that performed for atherosclerotic disease, and details are described above. As with all stenotic disease, the location, severity, and length of the stenosis are important to report.

ii. Diameter and/or cross sectional area (aneurysmal disease)

Aneurysmal dilatation is another major complication of vasculitis, leading to potential rupture (eg, luetic vasculitis of the ascending aorta in syphilis) or formation of thrombus with subsequent embolization (eg, Kawasaki disease). Quantitative evaluation of aneurysmal dilatation associated with vasculitis is identical to that for aneurysmal disease, providing a description of the location, length, cross-sectional diameter, or area measured from orthogonal multiplanar reconstruction (MPR). In addition, it may be helpful in some situations to measure the volume of the aneurysm using 3-D segmentation software for longitudinal observation.

iii. Wall thickness

In addition to quantifying the luminal dimensions, CTA and MRA are uniquely positioned to visualize the vessel wall and therefore quantify the thickness. An abnormally thickened artery may indicate the presence of vasculitis. In general, the aorta should be no thicker than approximately 2 mm, although it can vary up to 4 mm [107]. Longitudinal tracking of wall thickening may be a useful marker of disease activity [108,109], although the definitions of abnormal wall thickness are not precise. When measuring the wall thickness, it is important to use orthogonal MPR measurements to obtain a slice perpendicular to the vessel wall to ensure accurate measurements by minimizing partial volume effects that may cause wall thickness to be overestimated. Both MRI and CT are excellent methods to visualize vessel walls. MRI methods typically provide superior soft-tissue contrast that can aid the detection of mural inflammation and edema [106] and aid in more precise delineation of the vessel wall boundaries, although CTA generally has higher spatial resolution and the benefit of shorter examination times.

b. Role of phase contrast MRI for flow reversal (eg, subclavian steal in great vessel disease)

In stenotic disease, particularly Takayasu arteritis and giant cell arteritis, severe narrowing or occlusion of the great vessels produces altered flow patterns that can result in symptomatic conditions such as subclavian steal. Cardiac gated phase contrast MRI performed in the axial plane is a useful means to visualize flow direction and also quantifies flow reversal in the vertebral arteries. In some cases, it may be helpful to perform maneuvers such as arm exercises of the affected side to elicit steal phenomenon.

6. Fibromuscular dysplasia

Fibromuscular dysplasia (FMD) is a relatively common nonatherosclerotic vascular disease that affects the intima or media of large and medium arteries, including, but not limited to:

- Renal arteries
- Internal carotid arteries
- Iliac arteries
- Vertebral arteries
- Mesenteric arteries

a. Morphology

The morphology of FMD is highly varied, ranging from focal stenoses to long tubular stenoses to the classic “string of beads” appearance. FMD is associated with the development of aneurysms and

dissections of the affected vessels. Quantification of stenosis can be performed just as with other forms of stenotic disease. In addition, the presence of webs, particularly in the string of beads configuration, may make identification and grading of hemodynamically significant stenoses challenging [110]. For these reasons, CTA may be the preferable modality if FMD is suspected, as it has higher spatial resolution than MRA, although both methods provide an excellent noninvasive means for evaluating renal artery stenosis [111,112]. However, few direct comparisons in the setting of FMD have been made [112].

b. Phase contrast for turbulence / hemodynamically significant stenosis

3-D phase contrast (PC) MRA is commonly used to evaluate stenoses for hemodynamic significance. As discussed above, Grist et al demonstrated that signal dropout on PC-MRA images at the site of a hemodynamically significant stenosis may be a useful method to distinguish mild to moderate narrowing from more severe disease because of the dephasing of signal within a voxel that occurs in the presence of turbulent flow [97]. Further, Prince et al first demonstrated the ability of 3-D PC MRA to predict functional recovery after revascularization [113].

c. Phase contrast for pressure gradients

2-D phase contrast, like ultrasound Doppler, can be used to measure the peak velocity across a focal stenosis. Using the Bernoulli approximation (also known as the modified Bernoulli equation), the pressure gradient across a focal stenosis can be approximated as: ΔP (mmHg) $\approx 4V^2$, where V =maximum velocity (m/s). This approximation is not valid over long segment stenoses.

7. Vascular malformations: MRA and CTA

Vascular malformations are complex entities with a spectrum of abnormalities, including parenchymal arteriovenous malformations (AVMs), venous angiomas, cavernous angiomas, and capillary telangiectasias. In addition to characterizing the qualitative features of vascular malformations (eg, presence of nidus, draining, veins), MRA and CTA can be used for quantitative assessment of these entities.

a. Location, extent, size

The location with respect to adjacent anatomy and the extent and size of a vascular malformation should be reported.

b. Other quantitative aspects of morphology, size of feeding/draining vessels

In AVMs large draining veins are often identified. Their diameter (measured with MPR) and potential length may be helpful information for the treating physician.

c. Use of time resolved imaging, bolus passage time

Time resolved contrast-enhanced MR imaging methods (eg, TRICKS, TWIST, CENTRA) [30] may offer relative estimates of transit times of small boluses of injected gadolinium based contrast agents (GBCAs) to help characterize vascular malformations. Higher temporal resolution techniques are under development. The precise utility of transit time is not well defined at this time.

8. Venous disease: MRA and CTA

CTA and MRA in the delayed phase (for contrast enhanced imaging) or non-contrast enhanced MRA using time of flight methods are excellent methods to evaluate for the presence of deep venous thrombosis in the lower extremities and pelvis [114,115].

a. May-Thurner syndrome

May-Thurner syndrome typically occurs in young women presenting with left lower extremity deep vein thrombosis (DVT) and is caused by compression of the left common iliac vein as it passes between the lumbar spine posteriorly and (typically) the right common iliac artery anteriorly.

i. Morphology of venous stenosis

In addition to the presence of clot, patients with left lower extremity DVT should undergo evaluation of the left common iliac vein with high resolution CTA or MRA acquired in the delayed phase. Orthogonal MPRs visualizing the iliac vein at the narrowest point should be performed. The area of narrowing is typically ribbon-like, and measurements of the major and minor axis of the vessel cross-section should be provided. In some cases the vein may be occluded.

ii. Time resolved MRA for venous collaterals, flow reversal, etc.

Time resolved contrast enhanced MRA (TRICKS, TWIST, CENTRA) [30] may be helpful for identifying venous collaterals and the presence of flow reversal. The use of phase contrast MRA for quantitative assessment of venous narrowing for measuring pressure gradients in May-Thurner syndrome is not well established, although it holds promise.

9. Acquired cardiac disease: MRI/MRA, CTA

a. Ischemic disease

i. Function and morphology (primarily LV, but also RV)

The evaluation of cardiac function can provide valuable prognostic information on ischemic heart disease. The ejection fraction (EF) predicts outcome better than the number of vessels involved [116], and prognosis after myocardial infarction (MI) is closely related to the degree of LV contractile dysfunction [117]. Regional ventricular dysfunction (thinning of wall, decreased systolic wall thickening, abnormal wall motion, or the presence of LV thrombus) is also a good indicator of acute/chronic ischemia [118]. Based on these data, quantitative measures of ventricular function should be performed by short axes direct planimetry when ECG gating is used for image acquisition. For cardiac CT, imaging is often performed with prospective ECG gating to limit patient radiation exposure. When this acquisition strategy is used, quantitative measures will not be available.

Myocardial perfusion imaging is another important method using myocardial blood flow (MBF) or coronary flow reserve (CFR), detecting multivessel disease that is sometimes not obvious in qualitative imaging [119]. Although quantification has been studied, at present image interpretation is primarily subjective. The main target is LV in most IHD patients, but RV evaluation is also important, especially in inferior wall ischemia/infarction. Later generation multidetector CT scanners with faster scan time and thinner slices are now used for research purposes in this field, with encouraging preliminary data.

ii. Presence and extent of scar/infarct, T2 signal in acute MI

Myocardial delayed contrast enhancement (MDE) imaging using either gadolinium (MR) or iodine (CT) indicates irreversible injury [120]. At present, these metrics are used on a quartile basis with specific cutoffs of 50% delayed enhancement [74]. In more extensive myocardial infarctions, microvascular obstruction (a region of “no re-flow”) may be seen as a dark subendocardially-based inner core of the myocardial infarction surrounded by hyperenhancement or the larger myocardial infarct territory. Ischemia-associated myocardial edema shows high signal on T2-weighted imaging. The extent of high T2-signal reflects the area of risk [121-123]

that may include regions of reversible injury as well. Myocardium with potentially reversible injury (myocardial salvage area) is represented by the difference between the entire high T2-signal area and the MDE area determined from MR images. This is routinely performed subjectively. T1- and T2-mapping MRI are emerging techniques that are showing promise for further myocardial characterization and may be of particular value for not only myocardial infarction but diffuse myocardial involvement as can be seen in many cardiomyopathies.

iii. Complications, eg, valvular related abnormalities

Several complications are associated with acute myocardial infarction (MI), including papillary muscle injury, ventricular septal defect (VSD), contained rupture, and pericarditis or Dressler's syndrome, which can be detected by CT or MR imaging [124]. Papillary muscle involvement is known to cause mitral valve regurgitation. Quantification of valve regurgitant volume/fraction by MR and evaluation of pericardium are discussed below.

iv. Coronary artery calcium scoring (CT) for risk assessment

Calcium scoring images are acquired with noncontrast ECG gated CT to optimally visualize and quantify calcified plaque [83]. High "calcium scores" are associated with an increased risk of MI [125], and a calcium score of 0 has a very low but nonzero risk of a major adverse cardiac event [126].

b. Nonischemic cardiomyopathy and infiltrative disease

i. Function and morphology

There are several nonischemic cardiomyopathies: hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), and restrictive cardiomyopathy. Restrictive cardiomyopathy usually occurs secondary to infiltration of the myocardium, amyloidosis, myocardial fibrosis (after open heart surgery), radiation, sarcoidosis, or endomyocardial eosinophilia. Nonischemic cardiomyopathy usually has an alteration in the ventricular function, leading to heart failure. In addition to EF, the LV myocardial mass (myocardial volume \times myocardial density) is a useful parameter to assess nonischemic cardiomyopathy; LV mass and myocardial wall thickening correlate independently with prognosis [118]. Generalized or regional wall-motion abnormalities also occur in DCM and HCM. The metrics for function and morphology follow those described in section IV.A. Native T1-mapping and extracellular volume fraction (ECV) mapping using MRI are promising techniques for identification of diffuse myocardial disease that may not be readily apparent on standard perfusion or MDE MR imaging.

ii. Extent of delayed enhancement for staging/prognosis

Although MDE imaging is used more often in detecting MI, scar quantification on MDE images may also play an important role in determining prognosis and risk assessment for nonischemic cardiomyopathy patients. Hyper-enhancement is often detected in the myocardium of HCM patients [127] in a characteristically patchy midwall distribution in hypertrophied areas, although DCM patients often show linear midwall striae. A higher percentage of MDE on MR in HCM patients is known to be associated with ventricular tachycardia and fatal arrhythmias [128]. Subjective assessment is routine.

iii. Complications (valvular disease, subaortic stenosis)

HCM is known to cause subaortic stenosis outflow obstruction because of the septum hypertrophy and systolic anterior motion (SAM) of the anterior leaflet of the mitral valve. MR imaging can potentially quantify pressure gradients or valve area using a phase contrast acquisition, which is discussed in the valvular disease section below.

c. Valvular disease

i. Cross sectional area (CT and MRI)

Valve area measurements in patients with aortic stenosis greatly affect treatment strategies and predict prognosis. On MR images, the valve area is usually calculated indirectly by measuring the time-velocity integrals at the valve and at an adjacent site with an easily measurable diameter (for example, the aortic outflow tract) and then assuming conservation of flow. Several studies have also tested the direct measurement of valve areas by MR cine or phase-contrast sequences through the valve plane [129,130]. However, when measuring valve planimetry directly, CT with cardiac ECG gating allows excellent visualization of valve structure and thus is frequently used in clinical settings. When using either MR or CT for measuring valve planimetry, at least 30 cardiac phases should be imaged or reconstructed in order to most accurately identify end systole, or the time at which the aortic valve orifice is most open.

ii. Detection of insufficiency and stenosis

MR enables quantitative analysis of valvular disease, consisting of calculation of regurgitant volume and fraction in patients with regurgitant valves and measurement of peak or time average velocities and pressure gradients in patients with stenotic valves [129]. CT usually detects valve stenosis itself or poststenotic dilatation with direct planimetry but does not greatly contribute to the diagnosis of valve insufficiency.

iii. Phase-contrast MR: pressure gradients, regurgitant fractions

Phase-contrast MR sequences can be used for both flow quantification for valvular insufficiency and peak and average velocities quantification for valvular stenosis. Aortic insufficiency is usually graded by regurgitant volume (volume of regurgitant flow across the valve per heartbeat) or regurgitant fraction (regurgitant volume divided by forward stroke volume). Quantification of stenotic valves measures peak and average velocities across the valve on phase-contrast images. These velocities are converted into pressure gradients with the modified Bernoulli equation: $\Delta P = 4V^2$ (as described above). A mean gradient greater than 50 mm Hg or peak velocity greater than 4.5 m/sec is defined as severe aortic stenosis.

iv. Effect on heart (chamber enlargement) or great vessels (poststenotic dilatation)

The pathophysiology of aortic stenosis involves obstruction of LV outflow, which leads to elevated LV pressures and LV hypertrophy. Arterial stenosis also causes poststenotic dilatation, a dilation of the vessel 1 to 3 cm distal to the area of stenosis. In contrast, aortic insufficiency involves volume overload of the left ventricle, resulting in LV dilatation. CT or MR imaging can directly demonstrate and measure LV hypertrophy, LV dilatation, and poststenotic dilatation of the ascending aorta.

v. Presurgical Planning

In high surgical risk patients with severe aortic stenosis (AS), transcatheter aortic valve replacement (TAVR) has demonstrated long-term results comparable to open surgical repair [131-134]. In a meta-analysis of 344 studies and 872 participants who had undergone previous CABG, TAVR patients had shorter hospital stays and performed similarly to surgical valve replacement patients in mid-term all-cause cardiovascular mortality [135]. Pre-procedural imaging evaluation should include cardiac-gated evaluation and measurement of the following intracardiac and aortic structures: LV cavity for thrombus, alignment of the LVOT, dimensions of the aortic valve annulus, distance of the coronary ostia to the aortic valve plane, length of the aortic cusp, width of the aortic sinus, sinotubular junction and ascending aorta. Additionally, the width of the descending thoracic aorta, abdominal aorta, and iliofemoral arteries should be measured and evaluated for extensive atherosclerotic disease or tortuosity. Most studies have utilized contrast-enhanced cardiac CT, although CMR may have a role, given the high prevalence of renal dysfunction in TAVR patients [136].

d. Diastolic dysfunction/heart failure

i. Function/morphology

Heart failure is characterized by any structural or functional cardiac disorder that impairs the ability of ventricles to fill with or eject blood. Therefore, for a final diagnosis of heart failure, the evaluation of systolic and/or diastolic dysfunction is required. As described above, MR imaging can quantify LV volume and ejection fraction (EF), or assess wall motion and be used for both diagnosis and monitoring. Myocardial perfusion imaging determines whether coronary artery disease contributes to the development of heart failure. Delayed enhancement (DE) imaging can also be used for heart failure assessment; the extent of DE predicts the response to beta-blocker therapy [137].

ii. Role of phase contrast (E/A reversal)

The E/A ratio is the ratio of early to late (“atrial”) diastolic filling velocity of the ventricle and can rapidly detect abnormal diastolic function. Although the normal E/A ratio is greater than 1, impaired relaxation of the ventricle decreases early diastolic filling and results in a reduced or reversed E/A ratio, eg, E/A ratio less than 1. E/A ratio is usually measured by echocardiography but can also be acquired with phase-contrast MRI by calculating transmitral (or transtricuspid) velocity.

e. Pericardial disease

i. Morphology and function

Many disease processes can affect the pericardium, including inflammation, infection, neoplasm, trauma, primary myocardial disease, and congenital disease. Imaging can provide morphologic evaluation of the pericardium, such as thickened/enhanced pericardium, presence of pericardial fluid, and chamber sizes (eg, atrial and ventricular size). Imaging usually targets the direct visualization of thickened/enhanced pericardium or the analysis of ventricular function. For example, in patients with constrictive pericarditis, a leftward bounce (or flattening) of the interventricular septum can often be identified on early diastolic images, best noted on short-axis cine views. This occurs secondary to pericardial constriction of diastolic ventricular filling and an increase in ventricular pressure. In constrictive pericarditis, the elevation in right ventricular pressure results in the paradoxical leftward motion (ie, bounce) of the interventricular septum during early diastole. On occasion, the septal bounce can also be seen during inspiratory phases of a free breathing cine acquisition secondary to the augmentation of systemic venous return that occurs during inspiration [138].

ii. Pericardial thickness and enhancement

CT and MRI provide excellent visualization of the pericardium and can lend support to the diagnosis of pericardial disease. Regarding constrictive pericarditis, the CT and MR images can be used to directly measure pericardial thickening greater than 4 mm. This metric can be used with a subjective assessment of narrow, tubular deformation of the ventricles with a straightened or sigmoid-shaped interventricular septum to support the diagnosis [139]. Contrast enhancement is an additional qualitative finding associated with abnormal pericardium.

iii. ROI analysis for hemopericardium, calcium (CT)

ROI CT attenuation measurements characterize pericardial fluid about 40 to 60 HU. A fluid collection with attenuation close to that of water is likely to be a simple effusion, but attenuation greater than that of water suggests malignancy, hemopericardium, purulent exudate, or effusion associated with hypothyroidism [140]. MR can also characterize pericardial fluid, although qualitatively, with the use of multiple pulse sequences; hemorrhagic effusion is characterized by high signal on T1-weighted SE images and low intensity on gradient echo (GRE) cine images

[141]. Another important feature of CT is its ability to detect pericardial calcifications, a finding indicative of constrictive pericarditis. Assessment of constrictive physiology with MRI or CT requires ECG gating.

f. Pulmonary veins preablation, postablation for atrial fibrillation and pulmonary vein stenosis

- i. Preprocedure measurements (cross sectional diameters, length, number of veins, anatomy [especially variants])

In atrial fibrillation (AF) patients, atrial myocardium tissue is more often present in the pulmonary veins (PVs) and the atrial myocardium in the PVs has more severe discontinuity, hypertrophy, and fibrosis [142]. Catheter ablation has been widely used to treat AF and usually ablates the atrial myocardium inside the PVs to disconnect an abnormal interaction with left atrium. Preprocedural CTA or MRA for cross-sectional measurement of PV ostia is beneficial for selecting the optimal circular catheter [143]. Furthermore, because 38% of AF patients have variant anatomy of PVs [144], evaluating the number and location of PVs is useful in ascertaining that all PV orifices are evaluated during the procedure [144].

- ii. Postprocedure stenoses

A well-known complication of catheter ablation is PV stenosis. CT has been the most commonly used modality to detect post-procedure stenoses, but MRI can be used as well. Because the PV size varies throughout the cardiac cycle and the difference between maximum and minimum diameter is $15\% \pm 8\%$ [145], ECG-gated CTA acquisitions are preferred.

g. Pulmonary arterial hypertension

- i. Primary or secondary

Pulmonary arterial hypertension (PAH) is a condition characterized by increased pulmonary arterial pressure. In the conventional classification, it is divided into 2 main categories: 1) primary PAH (not caused by any other disease or condition); and 2) secondary PAH (caused by another underlying condition), including lung diseases (eg, COPD, interstitial lung diseases), heart diseases (eg, congestive heart failure, congenital heart disease, mitral stenosis), chronic thromboembolic diseases (eg, pulmonary embolism), HIV infection, or medications. Secondary PAH is much more common than primary PAH.

- ii. Right ventricle function

Increased pulmonary arterial pressure causes an increased workload of the RV, leading to RV hypertrophy with subsequent dilatation and right heart failure. MR and CT have been increasingly used for imaging the RV, as well as for the LV, but protocol should be carefully adjusted to accurately visualize the more complex shape of the RV [118]. In case of acute pulmonary embolism (PE), the chest CT measures the RV/LV diameter ratio and uses greater than 0.9 to predict 30-day mortality and major complications [99,100]. A ratio of main pulmonary artery diameter to the ascending aorta diameter of greater than 1 can be reliably used to detect pulmonary hypertension in adult patients with cardiopulmonary diseases if the ascending aorta is of normal size [146]. In pediatric patients, a ratio of the main pulmonary artery diameter to the ascending aorta diameter of greater than 1.3 may suggest pulmonary hypertension. In addition to morphological assessment, MR imaging can easily measure EF of both ventricles and LV end-diastolic volume, which are significantly decreased in patients with PAH [147,148].

- iii. Pulmonary artery morphology (diameters, cross sectional areas)

Mean pulmonary artery (PA) pressure correlates linearly with main PA diameter [149], and a PA diameter greater than 30 mm indicates a PA pressure greater than 20 mm Hg [150]. However,

several studies failed to demonstrate that main PA diameter predicts increased mortality or indicates severity of acute PE [151,152].

iv. Assessment of clot burden with chronic thromboembolic disease

The presence, location, and degree of obstruction of arterial clots can be scored according to several different scoring systems. Qanadli and Mastora [153,154] use CT pulmonary angiography to quantify acute PE severity. However, PA clot load scores usually do not take into account clots located in small peripheral PAs and the current literature shows some discrepancies regarding the association between the clot burden and immediate outcome. For example, although reports of the score proposed by Qanadli suggest that it is a significant predictor of death [152], others reported the clot scores to be a poor predictor of mortality [151]. In general, clot burden in CTPA is not reported.

v. Assessment of valve function in PAH (morphology, flow, pressure gradients)

Mitral valve stenosis can cause PAH. On the other hand, PAH can cause dilatation of the pulmonic valve ring and then results in pulmonic valve regurgitation. Assessment of mitral valve stenosis or pulmonic valve regurgitation can be performed on phase-contrast sequences for quantitative velocity and flow measurement using the methods previously described.

10. Congenital cardiac disease (vascular and cardiac): MRI, CT

a. Cardiac function

Cardiac-gated CT [155-163] and MRI [159,163-167] are useful for the evaluation of patients with suspected or known congenital heart disease (CHD). As with other conditions, both cardiac-gated CT and MRI can provide quantitative measurements of the various chamber sizes and function, notably chamber volumes, myocardial mass, and ejection fractions for the left and right ventricles using standard quantitative tools outlined previously in section IV.A.4. Valvular function can also be assessed as detailed previously in section IV.B.9.c. For example, CT and MRI are useful for the postoperative assessment of repaired tetralogy of Fallot, although MRI is the preferred modality unless there is a contraindication to MRI [159,166]. In this case, CT and MRI can provide functional assessment of ventricular volumes and ejection fractions. Pulmonic insufficiency and pulmonic stenosis can also be assessed using cine phase contrast MRI performed perpendicular to the main pulmonary artery. These data provide essential functional information, especially of the RV, for determining proper timing for pulmonic valve replacement in patients with corrected or uncorrected tetralogy of Fallot.

b. Vessel assessment

Arterial (eg, thoracic aorta) and venous structures (eg, pulmonary veins) are also well evaluated using CT angiography [155,157-163] or MR angiography [163,168-172]. For example, both CT and MRI have been shown to provide comparable diagnostic evaluation of aortic narrowing in children with coarctation of the aorta [163]. MRI has the added benefit of allowing blood flow analysis using velocity-encoded cine phase contrast MRI that can measure peak velocity across a juxtaductal aortic narrowing to estimate the pressure gradient across the aortic coarctation using the modified Bernoulli equation [168]. Time-resolved MR angiography [173,174] can be particularly helpful when evaluating the presence of anomalous and/or postsurgical vascular connections in patients with CHD.

CT angiography of the cardiopulmonary structures is often a very informative method of examination. Elimination of retrospective ECG-gating allows one to decrease the radiation dose to the patient. Prospective ECG triggered studies can allow for anatomic imaging with reduced cardiac motion artifacts and with radiation dose equivalent to non-gated studies.

c. Pulmonary-to-systemic shunt (Qp/Qs ratio)

A unique evaluation in patients with suspected or known CHD is the assessment for a left-to-right shunt using the pulmonary (Qp) to systemic (Qs) blood flow ratio (Qp/Qs ratio) [175,176]. This measures the volume of blood flow between the pulmonary (ie, right heart) and systemic (ie, left heart) circulations. In healthy individuals, the blood flow is equal and the resultant Qp/Qs ratio is 1. In patients with an underlying left-to-right shunt lesion (eg, atrial septal defect, ventricular septal defect, or partial anomalous pulmonary venous return), there is shunting of blood from the left to the right heart and Qp/Qs ratio greater than 1. When the Qp/Qs ratio is less than 1, this represents right to left shunting. Symptomatic patients often present when the shunting becomes moderate (ie, Qp/Qs >1.5) or large (eg, Qp/Qs >2.2). The Qp/Qs ratio is most commonly measured using MRI. It can be determined by comparing the measured flow over the cardiac cycle on cine phase-contrast MRI performed perpendicular to both the main pulmonary artery (Qp) and the ascending thoracic aorta (Qs). In patients with suspected systemic to pulmonary collateral flow, the pulmonary flow (Qp) can be estimated using the pulmonary venous return and the systemic flow (Qs) can be estimated using the caval return [177].

In younger patients, MRI may be the preferred modality, particularly when functional assessment with CT would require retrospective ECG gating and relatively high radiation doses. Further, the use of time-resolved MRA and phase contrast MRI methods offer significant advantages whose relative importance will depend on the specific application.

V. DOCUMENTATION

Reporting should be in accordance with the [ACR Practice Parameter for Communication of Diagnostic Imaging Findings](#) [178].

VI. EQUIPMENT SPECIFICATIONS

The MRI equipment specifications and performance must meet all state and federal requirements. The requirements include, but are not limited to, specifications of maximum static magnetic strength, maximum rate of change of the magnetic field strength (dB/dt), maximum radiofrequency power deposition (specific absorption rate), and maximum acoustic noise levels.

VII. QUALITY CONTROL AND IMPROVEMENT, SAFETY, INFECTION CONTROL, AND PATIENT EDUCATION

Policies and procedures related to quality, patient education, infection control, and safety should be developed and implemented in accordance with the ACR Policy on Quality Control and Improvement, Safety, Infection Control, and Patient Education appearing under the heading *Position Statement on QC & Improvement, Safety, Infection Control, and Patient Education* on the ACR website (<http://www.acr.org/guidelines>).

ACKNOWLEDGEMENTS

This practice parameter was developed according to the process described under the heading *The Process for Developing ACR Practice Parameters and Technical Standards* on the ACR website (<http://www.acr.org/guidelines>) by the Committee on Body Imaging (Cardiovascular) of the ACR Commission on Body Imaging and the Committee on Practice Parameters – Pediatric Radiology of the ACR Commission on Pediatric Radiology in collaboration with the NASCI and SPR.

Collaborative Committee – members represent their societies in the initial and final revision of this practice parameter.

ACR

Vincent B. Ho, MD, MBA, Chair
 John P. Lichtenberger III, MD
 James C. Carr, MD
 Beverley Newman, MB, BCh, BSc, FACR

NASCI

Karen Ordovas, MD, MAS, FNASCI
 Akos Varga-Szemes, MD, PhD

SPR

Eric J. Hoggard, MD
 Karen Lyons, MD

Committee on Body Imaging (Cardiovascular)

(ACR Committee responsible for sponsoring the draft through the process)

Vincent B. Ho, MD, MBA, Chair
 David M. Biko, MD
 William R. Corse, DO
 James P. Earls, MD
 Travis S. Henry, MD
 Lynne M. Hurwitz, MD

Jonathan Keung, MD
 Jacobo Kirsch, MD
 Constantino S. Pena, MD
 Andrew L. Rivard, MD
 Alan H. Stolpen, MD, PhD, FACR
 Richard D. White, MD, FACR

Committee on Practice Parameters – Pediatric Radiology

(ACR Committee responsible for sponsoring the draft through the process)

Beverley Newman, MB, BCh, BSc, FACR, Chair
 Lorna P. Browne, MB, BCh
 Timothy J. Carmody, MD, FACR
 Brian D. Coley, MD, FACR
 Lee K. Collins, MD
 Monica S. Epelman, MD
 Lynn Ansley Fordham, MD, FACR
 Kerri A. Highmore, MD

Sue C. Kaste, DO
 Tal Laor, MD
 Terry L. Levin, MD
 Marguerite T. Parisi, MD, MS
 Sumit Pruthi, MBBS
 Nancy K. Rollins, MD
 Pallavi Sagar, MD

Lincoln L. Berland, MD, FACR, Chair, Commission on Body Imaging
 Marta Hernanz-Schulman, MD, FACR, Chair, Commission on Pediatric Radiology
 Jacqueline Anne Bello, MD, FACR, Chair, Commission on Quality and Safety
 Matthew S. Pollack, MD, FACR, Chair, Committee on Practice Parameters and Technical Standards

Comments Reconciliation Committee

Eric J. Stern, MD, Chair
 Greg N. Nicola, MD, Co-Chair
 Jacqueline A. Bello, MD, FACR
 Lincoln L. Berland, MD, FACR
 David M. Biko, MD
 James C. Carr, MD
 Marta Hernanz-Schulman, MD, FACR
 William T. Herrington, MD, FACR
 Vincent B. Ho, MD, MBA
 Eric J. Hoggard, MD
 Jonathan Keung, MD

Friedrich D. Knollman, MD, PhD
 Negar Knowles, MD
 Paul A. Larson, MD, FACR
 John P. Lichtenberg III, MD, MBA
 Karen Lyons, MD
 Beverley Newman, MB, BCh, BSc, FACR
 Karen Ordovas, MD, MAS, FNASCI
 Matthew S. Pollack, MD, FACR
 Timothy L. Swan, MD, FACR, FSIR
 Akos Varga-Szemes, MD, PhD

REFERENCES

1. American College of Radiology. ACR practice parameter for performing and interpreting magnetic resonance imaging (MRI). 2014; Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Perf-Interpret.pdf>. Accessed November 9, 2015.
2. American College of Radiology. ACR practice parameter for performance and interpreting diagnostic computed tomography (CT). 2011; Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CT-Perf-Interpret.pdf>. Accessed November 9, 2015.
3. Bluemke DA, Boxerman JL, Atalar E, McVeigh ER. Segmented K-space cine breath-hold cardiovascular MR imaging: Part 1. Principles and technique. *AJR Am J Roentgenol*. 1997;169(2):395-400.
4. Boxerman JL, Mosher TJ, McVeigh ER, Atalar E, Lima JA, Bluemke DA. Advanced MR imaging techniques for evaluation of the heart and great vessels. *Radiographics*. 1998;18(3):543-564.
5. Otero HJ, Steigner ML, Rybicki FJ. The "post-64" era of coronary CT angiography: understanding new technology from physical principles. *Radiologic clinics of North America*. 2009;47(1):79-90.
6. Rybicki FJ, Otero HJ, Steigner ML, et al. Initial evaluation of coronary images from 320-detector row computed tomography. *The international journal of cardiovascular imaging*. 2008;24(5):535-546.
7. Raff GL, Abidov A, Achenbach S, et al. SCCT guidelines for the interpretation and reporting of coronary computed tomographic angiography. *Journal of cardiovascular computed tomography*. 2009;3(2):122-136.
8. Hendel RC, Budoff MJ, Cardella JF, et al. ACC/AHA/ACR/ASE/ASNC/HRS/NASCI/RSNA/SAIP/SCAI/SCCT/SCMR/SIR 2008 Key Data Elements and Definitions for Cardiac Imaging A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Data Standards (Writing Committee to Develop Clinical Data Standards for Cardiac Imaging). *Journal of the American College of Cardiology*. 2009;53(1):91-124.
9. Stillman AE, Rubin GD, Teague SD, White RD, Woodard PK, Larson PA. Structured reporting: coronary CT angiography: a white paper from the American College of Radiology and the North American Society for Cardiovascular Imaging. *Journal of the American College of Radiology : JACR*. 2008;5(7):796-800.
10. Cury RC, Abbara S, Achenbach S, et al. CAD-RADS: Coronary Artery Disease - Reporting and Data System.: An Expert Consensus Document of the Society of Cardiovascular Computed Tomography (SCCT), the American College of Radiology (ACR) and the North American Society for Cardiovascular Imaging (NASCI). Endorsed by the American College of Cardiology. *Journal of the American College of Radiology : JACR*. 2016.
11. Bley TA, Chase PJ, Reeder SB, et al. Endovascular abdominal aortic aneurysm repair: nonenhanced volumetric CT for follow-up. *Radiology*. 2009;253(1):253-262.
12. Buckley O, Rybicki FJ, Gerson DS, et al. Imaging features of intramural hematoma of the aorta. *The international journal of cardiovascular imaging*. 2010;26(1):65-76.
13. Lu MT, Millstine J, Menard MT, Rybicki FJ, Viscomi S. Periaortic lymphoma as a mimic of posttraumatic intramural hematoma. *Emergency radiology*. 2006;13(1):35-38.
14. Lotz J, Meier C, Leppert A, Galanski M. Cardiovascular flow measurement with phase-contrast MR imaging: basic facts and implementation. *Radiographics*. 2002;22(3):651-671.
15. O'Donnell M. NMR blood flow imaging using multiecho, phase contrast sequences. *Med Phys*. 1985;12(1):59-64.
16. Ley S, Ley-Zaporozhan J, Kreitner KF, et al. MR flow measurements for assessment of the pulmonary, systemic and bronchosystemic circulation: impact of different ECG gating methods and breathing schema. *Eur J Radiol*. 2007;61(1):124-129.
17. Ley S, Unterhinninghofen R, Ley-Zaporozhan J, Schenk JP, Kauczor HU, Szabo G. Validation of magnetic resonance phase-contrast flow measurements in the main pulmonary artery and aorta using perivascular ultrasound in a large animal model. *Invest Radiol*. 2008;43(6):421-426.
18. Johansson B, Babu-Narayan SV, Kilner PJ. The effects of breath-holding on pulmonary regurgitation measured by cardiovascular magnetic resonance velocity mapping. *J Cardiovasc Magn Reson*. 2009;11:1.
19. Liu X, Weale P, Reiter G, et al. Breathhold time-resolved three-directional MR velocity mapping of aortic flow in patients after aortic valve-sparing surgery. *J Magn Reson Imaging*. 2009;29(3):569-575.
20. Clark TW, Culham JA. Cine phase-contrast MR to assess portal blood flow in a 10-year-old girl with abdominal aortic coarctation: a case report. *Pediatr Radiol*. 1998;28(8):602-604.

21. Lew CD, Alley MT, Bammer R, Spielman DM, Chan FP. Peak velocity and flow quantification validation for sensitivity-encoded phase-contrast MR imaging. *Acad Radiol.* 2007;14(3):258-269.
22. Hodnett PA, Glielmi CB, Davarpanah AH, et al. Inline directionally independent peak velocity evaluation reduces error in peak antegrade velocity estimation in patients referred for cardiac valvular assessment. *AJR Am J Roentgenol.* 2012;198(2):344-350.
23. Frydrychowicz A, Markl M, Hirtler D, et al. Aortic hemodynamics in patients with and without repair of aortic coarctation: in vivo analysis by 4D flow-sensitive magnetic resonance imaging. *Invest Radiol.* 2011;46(5):317-325.
24. Hope MD, Hope TA, Meadows AK, et al. Bicuspid aortic valve: four-dimensional MR evaluation of ascending aortic systolic flow patterns. *Radiology.* 2010;255(1):53-61.
25. Carr JC, Finn JP. MR imaging of the thoracic aorta. *Magn Reson Imaging Clin N Am.* 2003;11(1):135-148.
26. Carr JC, Laub G, Zheng J, Pereles FS, Finn JP. Time-resolved three-dimensional pulmonary MR angiography and perfusion imaging with ultrashort repetition time. *Acad Radiol.* 2002;9(12):1407-1418.
27. Carroll TJ. The emergence of time-resolved contrast-enhanced MR imaging for intracranial angiography. *AJNR Am J Neuroradiol.* 2002;23(3):346-348.
28. Cashen TA, Jeong H, Shah MK, et al. 4D radial contrast-enhanced MR angiography with sliding subtraction. *Magn Reson Med.* 2007;58(5):962-972.
29. Frydrychowicz A, Bley TA, Winterer JT, et al. Accelerated time-resolved 3D contrast-enhanced MR angiography at 3T: clinical experience in 31 patients. *MAGMA.* 2006;19(4):187-195.
30. Korosec FR, Frayne R, Grist TM, Mistretta CA. Time-resolved contrast-enhanced 3D MR angiography. *Magn Reson Med.* 1996;36(3):345-351.
31. Jeong HJ, Vakil P, Sheehan JJ, et al. Time-resolved magnetic resonance angiography: evaluation of intrapulmonary circulation parameters in pulmonary arterial hypertension. *J Magn Reson Imaging.* 2011;33(1):225-231.
32. Shors SM, Cotts WG, Pavlovic-Surjancev B, et al. Non-invasive cardiac evaluation in heart failure patients using magnetic resonance imaging: a feasibility study. *Heart Fail Rev.* 2005;10(4):265-273.
33. Shors SM, Cotts WG, Pavlovic-Surjancev B, Francois CJ, Gheorghiade M, Finn JP. Heart failure: evaluation of cardiopulmonary transit times with time-resolved MR angiography. *Radiology.* 2003;229(3):743-748.
34. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. *Eur J Echocardiogr.* 2006;7(2):79-108.
35. Gordon EP, Schnittger I, Fitzgerald PJ, Williams P, Popp RL. Reproducibility of left ventricular volumes by two-dimensional echocardiography. *Journal of the American College of Cardiology.* 1983;2(3):506-513.
36. Grothues F, Smith GC, Moon JC, et al. Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. *Am J Cardiol.* 2002;90(1):29-34.
37. Hunold P, Schlosser T, Vogt FM, et al. Myocardial late enhancement in contrast-enhanced cardiac MRI: distinction between infarction scar and non-infarction-related disease. *AJR Am J Roentgenol.* 2005;184(5):1420-1426.
38. Vogel-Claussen J, Rochitte CE, Wu KC, et al. Delayed enhancement MR imaging: utility in myocardial assessment. *Radiographics.* 2006;26(3):795-810.
39. Kim HW, Farzaneh-Far A, Kim RJ. Cardiovascular magnetic resonance in patients with myocardial infarction: current and emerging applications. *Journal of the American College of Cardiology.* 2009;55(1):1-16.
40. Brumback LC, Kronmal R, Heckbert SR, et al. Body size adjustments for left ventricular mass by cardiovascular magnetic resonance and their impact on left ventricular hypertrophy classification. *The international journal of cardiovascular imaging.* 2010;26(4):459-468.
41. Natori S, Lai S, Finn JP, et al. Cardiovascular function in multi-ethnic study of atherosclerosis: normal values by age, sex, and ethnicity. *AJR Am J Roentgenol.* 2006;186(6 Suppl 2):S357-365.
42. Salton CJ, Chuang ML, O'Donnell CJ, et al. Gender differences and normal left ventricular anatomy in an adult population free of hypertension. A cardiovascular magnetic resonance study of the Framingham Heart Study Offspring cohort. *Journal of the American College of Cardiology.* 2002;39(6):1055-1060.

43. Lin FY, Devereux RB, Roman MJ, et al. Cardiac chamber volumes, function, and mass as determined by 64-multidetector row computed tomography: mean values among healthy adults free of hypertension and obesity. *JACC Cardiovasc Imaging*. 2008;1(6):782-786.
44. Lorenz CH, Walker ES, Morgan VL, Klein SS, Graham TP, Jr. Normal human right and left ventricular mass, systolic function, and gender differences by cine magnetic resonance imaging. *J Cardiovasc Magn Reson*. 1999;1(1):7-21.
45. Nikitin NP, Loh PH, de Silva R, et al. Left ventricular morphology, global and longitudinal function in normal older individuals: a cardiac magnetic resonance study. *Int J Cardiol*. 2006;108(1):76-83.
46. Sandstede J, Lipke C, Beer M, et al. Age- and gender-specific differences in left and right ventricular cardiac function and mass determined by cine magnetic resonance imaging. *Eur Radiol*. 2000;10(3):438-442.
47. Cheng S, Fernandes VR, Bluemke DA, McClelland RL, Kronmal RA, Lima JA. Age-related left ventricular remodeling and associated risk for cardiovascular outcomes: the Multi-Ethnic Study of Atherosclerosis. *Circ Cardiovasc Imaging*. 2009;2(3):191-198.
48. Lorenz CH. The range of normal values of cardiovascular structures in infants, children, and adolescents measured by magnetic resonance imaging. *Pediatr Cardiol*. 2000;21(1):37-46.
49. Alfakih K, Plein S, Thiele H, Jones T, Ridgway JP, Sivananthan MU. Normal human left and right ventricular dimensions for MRI as assessed by turbo gradient echo and steady-state free precession imaging sequences. *J Magn Reson Imaging*. 2003;17(3):323-329.
50. Hudsmith LE, Petersen SE, Tyler DJ, et al. Determination of cardiac volumes and mass with FLASH and SSFP cine sequences at 1.5 vs. 3 Tesla: a validation study. *J Magn Reson Imaging*. 2006;24(2):312-318.
51. Moon JC, Lorenz CH, Francis JM, Smith GC, Pennell DJ. Breath-hold FLASH and FISP cardiovascular MR imaging: left ventricular volume differences and reproducibility. *Radiology*. 2002;223(3):789-797.
52. Sievers B, Kirchberg S, Bakan A, Franken U, Trappe HJ. Impact of papillary muscles in ventricular volume and ejection fraction assessment by cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2004;6(1):9-16.
53. Han Y, Osborn EA, Maron MS, Manning WJ, Yeon SB. Impact of papillary and trabecular muscles on quantitative analyses of cardiac function in hypertrophic cardiomyopathy. *J Magn Reson Imaging*. 2009;30(5):1197-1202.
54. Bellenger NG, Grothues F, Smith GC, Pennell DJ. Quantification of right and left ventricular function by cardiovascular magnetic resonance. *Herz*. 2000;25(4):392-399.
55. Heuschmid M, Rothfuss JK, Schroeder S, et al. Assessment of left ventricular myocardial function using 16-slice multidetector-row computed tomography: comparison with magnetic resonance imaging and echocardiography. *Eur Radiol*. 2006;16(3):551-559.
56. Childs H, Ma L, Ma M, et al. Comparison of long and short axis quantification of left ventricular volume parameters by cardiovascular magnetic resonance, with ex-vivo validation. *J Cardiovasc Magn Reson*. 2011;13:40.
57. Juergens KU, Seifarth H, Maintz D, et al. MDCT determination of volume and function of the left ventricle: are short-axis image reformations necessary? *AJR Am J Roentgenol*. 2006;186(6 Suppl 2):S371-378.
58. Busch S, Johnson TR, Wintersperger BJ, et al. Quantitative assessment of left ventricular function with dual-source CT in comparison to cardiac magnetic resonance imaging: initial findings. *Eur Radiol*. 2008;18(3):570-575.
59. Gurvitz M, Burns KM, Brindis R, et al. Emerging Research Directions in Adult Congenital Heart Disease: A Report From an NHLBI/ACHA Working Group. *Journal of the American College of Cardiology*. 2016;67(16):1956-1964.
60. Radiology ACo. ACR Appropriateness Criteria®: Known or Suspected Congenital Heart Disease in the Adult. 2016; Available at: <https://acsearch.acr.org/docs/69355/Narrative/>. Accessed August 29, 2016.
61. Weinsaft JW, Cham MD, Janik M, et al. Left ventricular papillary muscles and trabeculae are significant determinants of cardiac MRI volumetric measurements: effects on clinical standards in patients with advanced systolic dysfunction. *Int J Cardiol*. 2008;126(3):359-365.
62. Palumbo A, Maffei E, Martini C, et al. Functional parameters of the left ventricle: comparison of cardiac MRI and cardiac CT in a large population. *La Radiologia medica*. 2010;115(5):702-713.

63. Caiani EG, Toledo E, MacEneaney P, et al. Automated interpretation of regional left ventricular wall motion from cardiac magnetic resonance images. *J Cardiovasc Magn Reson*. 2006;8(3):427-433.
64. Nandalur KR, Dwamena BA, Choudhri AF, Nandalur MR, Carlos RC. Diagnostic performance of stress cardiac magnetic resonance imaging in the detection of coronary artery disease: a meta-analysis. *Journal of the American College of Cardiology*. 2007;50(14):1343-1353.
65. Underwood SR, Anagnostopoulos C, Cerqueira M, et al. Myocardial perfusion scintigraphy: the evidence. *Eur J Nucl Med Mol Imaging*. 2004;31(2):261-291.
66. de Jong MC, Genders TS, van Geuns RJ, Moelker A, Hunink MG. Diagnostic performance of stress myocardial perfusion imaging for coronary artery disease: a systematic review and meta-analysis. *Eur Radiol*. 2012;22(9):1881-1895.
67. Okada DR, Ghoshhajra BB, Blankstein R, et al. Direct comparison of rest and adenosine stress myocardial perfusion CT with rest and stress SPECT. *J Nucl Cardiol*. 2010;17(1):27-37.
68. Ko SM, Choi JW, Hwang HK, Song MG, Shin JK, Chee HK. Diagnostic performance of combined noninvasive anatomic and functional assessment with dual-source CT and adenosine-induced stress dual-energy CT for detection of significant coronary stenosis. *AJR Am J Roentgenol*. 2012;198(3):512-520.
69. Jin KN, De Cecco CN, Caruso D, et al. Myocardial perfusion imaging with dual energy CT. *Eur J Radiol*. 2016.
70. Cannao PM, Schoepf UJ, Muscogiuri G, et al. Technical prerequisites and imaging protocols for dynamic and dual energy myocardial perfusion imaging. *Eur J Radiol*. 2015;84(12):2401-2410.
71. De Cecco CN, Harris BS, Schoepf UJ, et al. Incremental value of pharmacological stress cardiac dual-energy CT over coronary CT angiography alone for the assessment of coronary artery disease in a high-risk population. *AJR Am J Roentgenol*. 2014;203(1):W70-77.
72. Wang R, Yu W, Wang Y, et al. Incremental value of dual-energy CT to coronary CT angiography for the detection of significant coronary stenosis: comparison with quantitative coronary angiography and single photon emission computed tomography. *The international journal of cardiovascular imaging*. 2011;27(5):647-656.
73. Choi KM, Kim RJ, Gubernikoff G, Vargas JD, Parker M, Judd RM. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. *Circulation*. 2001;104(10):1101-1107.
74. Kim RJ, Wu E, Rafael A, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med*. 2000;343(20):1445-1453.
75. Watabe H, Sato A, Nishina H, et al. Enhancement patterns detected by multidetector computed tomography are associated with microvascular obstruction and left ventricular remodelling in patients with acute myocardial infarction. *Eur Heart J*. 2016;37(8):684-692.
76. Esposito A, Palmisano A, Antunes S, et al. Cardiac CT With Delayed Enhancement in the Characterization of Ventricular Tachycardia Structural Substrate: Relationship Between CT-Segmented Scar and Electro-Anatomic Mapping. *JACC Cardiovasc Imaging*. 2016;9(7):822-832.
77. Pedersen SF, Thrysoe SA, Robich MP, et al. Assessment of intramyocardial hemorrhage by T1-weighted cardiovascular magnetic resonance in reperfused acute myocardial infarction. *J Cardiovasc Magn Reson*. 2012;14:59.
78. Puntmann VO, D'Cruz D, Smith Z, et al. Native myocardial T1 mapping by cardiovascular magnetic resonance imaging in subclinical cardiomyopathy in patients with systemic lupus erythematosus. *Circ Cardiovasc Imaging*. 2013;6(2):295-301.
79. Karamitsos TD, Piechnik SK, Banyersad SM, et al. Noncontrast T1 mapping for the diagnosis of cardiac amyloidosis. *JACC Cardiovasc Imaging*. 2013;6(4):488-497.
80. Moon JC, Messroghli DR, Kellman P, et al. Myocardial T1 mapping and extracellular volume quantification: a Society for Cardiovascular Magnetic Resonance (SCMR) and CMR Working Group of the European Society of Cardiology consensus statement. *J Cardiovasc Magn Reson*. 2013;15:92.
81. Wong TC, Piehler K, Meier CG, et al. Association between extracellular matrix expansion quantified by cardiovascular magnetic resonance and short-term mortality. *Circulation*. 2012;126(10):1206-1216.
82. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation*. 2002;105(4):539-542.

83. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M, Jr., Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *Journal of the American College of Cardiology*. 1990;15(4):827-832.
84. Rumberger JA, Kaufman L. A rosetta stone for coronary calcium risk stratification: agatston, volume, and mass scores in 11,490 individuals. *AJR Am J Roentgenol*. 2003;181(3):743-748.
85. Polzer S, Gasser TC, Swedenborg J, Bursa J. The impact of intraluminal thrombus failure on the mechanical stress in the wall of abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg*. 2011;41(4):467-473.
86. Mehard WB, Heiken JP, Sicard GA. High-attenuating crescent in abdominal aortic aneurysm wall at CT: a sign of acute or impending rupture. *Radiology*. 1994;192(2):359-362.
87. Siegel CL, Cohan RH, Korobkin M, Alpern MB, Courneya DL, Leder RA. Abdominal aortic aneurysm morphology: CT features in patients with ruptured and nonruptured aneurysms. *AJR Am J Roentgenol*. 1994;163(5):1123-1129.
88. Labruto F, Blomqvist L, Swedenborg J. Imaging the intraluminal thrombus of abdominal aortic aneurysms: techniques, findings, and clinical implications. *Journal of vascular and interventional radiology : JVIR*. 2011;22(8):1069-1075; quiz 1075.
89. Bashir MR, Ferral H, Jacobs C, McCarthy W, Goldin M. Endoleaks after endovascular abdominal aortic aneurysm repair: management strategies according to CT findings. *AJR Am J Roentgenol*. 2009;192(4):W178-186.
90. Myers DI, Poole LJ, Imam K, Scheel PJ, Eustace JA. Renal artery stenosis by three-dimensional magnetic resonance angiography in type 2 diabetics with uncontrolled hypertension and chronic renal insufficiency: prevalence and effect on renal function. *Am J Kidney Dis*. 2003;41(2):351-359.
91. Bakker J, Beek FJ, Beutler JJ, et al. Renal artery stenosis and accessory renal arteries: accuracy of detection and visualization with gadolinium-enhanced breath-hold MR angiography. *Radiology*. 1998;207(2):497-504.
92. Christensson A. Renovascular disease and renal insufficiency--diagnosis and treatment. *Scand J Urol Nephrol*. 1999;33(6):400-405.
93. Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. Subcommittee on Reporting Standards for Arterial Aneurysms, Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery and North American Chapter, International Society for Cardiovascular Surgery. *J Vasc Surg*. 1991;13(3):452-458.
94. Truijers M, Resch T, Van Den Berg JC, Blankensteijn JD, Lonn L. Endovascular aneurysm repair: state-of-art imaging techniques for preoperative planning and surveillance. *J Cardiovasc Surg (Torino)*. 2009;50(4):423-438.
95. Geller SC. Imaging guidelines for abdominal aortic aneurysm repair with endovascular stent grafts. *Journal of vascular and interventional radiology : JVIR*. 2003;14(9 Pt 2):S263-264.
96. Schoenberg SO, Knopp MV, Bock M, et al. Renal artery stenosis: grading of hemodynamic changes with cine phase-contrast MR blood flow measurements. *Radiology*. 1997;203(1):45-53.
97. Grist TM. Magnetic resonance angiography of renal artery stenosis. *Am J Kidney Dis*. 1994;24(4):700-712.
98. De Cobelli F, Mellone R, Salvioni M, et al. Renal artery stenosis: value of screening with three-dimensional phase-contrast MR angiography with a phased-array multicoil. *Radiology*. 1996;201(3):697-703.
99. Quiroz R, Kucher N, Schoepf UJ, et al. Right ventricular enlargement on chest computed tomography: prognostic role in acute pulmonary embolism. *Circulation*. 2004;109(20):2401-2404.
100. Schoepf UJ, Kucher N, Kipfmüller F, Quiroz R, Costello P, Goldhaber SZ. Right ventricular enlargement on chest computed tomography: a predictor of early death in acute pulmonary embolism. *Circulation*. 2004;110(20):3276-3280.
101. Contractor S, Maldjian PD, Sharma VK, Gor DM. Role of helical CT in detecting right ventricular dysfunction secondary to acute pulmonary embolism. *J Comput Assist Tomogr*. 2002;26(4):587-591.
102. Reid JH, Murchison JT. Acute right ventricular dilatation: a new helical CT sign of massive pulmonary embolism. *Clin Radiol*. 1998;53(9):694-698.
103. Boxt LM. MR imaging of pulmonary hypertension and right ventricular dysfunction. *Magn Reson Imaging Clin N Am*. 1996;4(2):307-325.

104. American College of Radiology. ACR–NASCI–SPR practice parameter for the performance of cardiac magnetic resonance imaging (MRI). 2014; Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Cardiac.pdf>. Accessed November 17, 2015.
105. Khandelwal N, Kalra N, Garg MK, et al. Multidetector CT angiography in Takayasu arteritis. *Eur J Radiol*. 2011;77(2):369-374.
106. Nastri MV, Baptista LP, Baroni RH, et al. Gadolinium-enhanced three-dimensional MR angiography of Takayasu arteritis. *Radiographics*. 2004;24(3):773-786.
107. Li AE, Kamel I, Rando F, et al. Using MRI to assess aortic wall thickness in the multiethnic study of atherosclerosis: distribution by race, sex, and age. *AJR Am J Roentgenol*. 2004;182(3):593-597.
108. Atalay MK, Bluemke DA. Magnetic resonance imaging of large vessel vasculitis. *Curr Opin Rheumatol*. 2001;13(1):41-47.
109. Matsunaga N, Hayashi K, Sakamoto I, et al. Takayasu arteritis: MR manifestations and diagnosis of acute and chronic phase. *J Magn Reson Imaging*. 1998;8(2):406-414.
110. Leiner T, Michaely H. Advances in contrast-enhanced MR angiography of the renal arteries. *Magn Reson Imaging Clin N Am*. 2008;16(4):561-572, vii.
111. Eklof H, Ahlstrom H, Magnusson A, et al. A prospective comparison of duplex ultrasonography, captopril renography, MRA, and CTA in assessing renal artery stenosis. *Acta Radiol*. 2006;47(8):764-774.
112. Rountas C, Vlychou M, Vassiou K, et al. Imaging modalities for renal artery stenosis in suspected renovascular hypertension: prospective intraindividual comparison of color Doppler US, CT angiography, GD-enhanced MR angiography, and digital subtraction angiography. *Ren Fail*. 2007;29(3):295-302.
113. Prince MR, Schoenberg SO, Ward JS, Londy FJ, Wakefield TW, Stanley JC. Hemodynamically significant atherosclerotic renal artery stenosis: MR angiographic features. *Radiology*. 1997;205(1):128-136.
114. Oguzkurt L, Tercan F, Pourbagher MA, Kizilkilic O, Turkoz R, Boyvat F. Computed tomography findings in 10 cases of iliac vein compression (May-Thurner) syndrome. *Eur J Radiol*. 2005;55(3):421-425.
115. Wolpert LM, Rahmani O, Stein B, Gallagher JJ, Drezner AD. Magnetic resonance venography in the diagnosis and management of May-Thurner syndrome. *Vasc Endovascular Surg*. 2002;36(1):51-57.
116. Mock MB, Ringqvist I, Fisher LD, et al. Survival of medically treated patients in the coronary artery surgery study (CASS) registry. *Circulation*. 1982;66(3):562-568.
117. Sanz G, Castaner A, Betriu A, et al. Determinants of prognosis in survivors of myocardial infarction: a prospective clinical angiographic study. *N Engl J Med*. 1982;306(18):1065-1070.
118. Savino G, Zwerner P, Herzog C, et al. CT of cardiac function. *J Thorac Imaging*. 2007;22(1):86-100.
119. Attili AK, Schuster A, Nagel E, Reiber JH, van der Geest RJ. Quantification in cardiac MRI: advances in image acquisition and processing. *The international journal of cardiovascular imaging*. 2010;26 Suppl 1:27-40.
120. Fieno DS, Kim RJ, Chen EL, Lomasney JW, Klocke FJ, Judd RM. Contrast-enhanced magnetic resonance imaging of myocardium at risk: distinction between reversible and irreversible injury throughout infarct healing. *Journal of the American College of Cardiology*. 2000;36(6):1985-1991.
121. Tilak GS, Hsu LY, Hoyt RF Jr, Arai AE, Aletras AH. In vivo T2-weighted magnetic resonance imaging can accurately determine the ischemic area at risk for 2-day-old nonreperfused myocardial infarction. *Invest Radiol*. 2008;43:7-15.
122. Croisille P, Kim HW, Kim RJ. Controversies in cardiovascular MR imaging: T2-weighted imaging should not be used to delineate the area at risk in ischemic myocardial injury. *Radiology*. 2012;265(1):12-22.
123. Kim HW, Van Assche L, Jennings RB, et al. Relationship of T2-Weighted MRI Myocardial Hyperintensity and the Ischemic Area-At-Risk. *Circulation research*. 2015;117(3):254-265.
124. Hombach V, Grebe O, Merkle N, et al. Sequelae of acute myocardial infarction regarding cardiac structure and function and their prognostic significance as assessed by magnetic resonance imaging. *Eur Heart J*. 2005;26(6):549-557.
125. Greenland P, LaBree L, Azen SP, Doherty TM, Detrano RC. Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals. *JAMA*. 2004;291(2):210-215.
126. Church TS, Levine BD, McGuire DK, et al. Coronary artery calcium score, risk factors, and incident coronary heart disease events. *Atherosclerosis*. 2007;190(1):224-231.
127. Yamada M, Teraoka K, Kawade M, Hirano M, Yamashina A. Frequency and distribution of late gadolinium enhancement in magnetic resonance imaging of patients with apical hypertrophic

- cardiomyopathy and patients with asymmetrical hypertrophic cardiomyopathy: a comparative study. *The international journal of cardiovascular imaging*. 2009;25 Suppl 1:131-138.
128. Kwon DH, Setser RM, Popovic ZB, et al. Association of myocardial fibrosis, electrocardiography and ventricular tachyarrhythmia in hypertrophic cardiomyopathy: a delayed contrast enhanced MRI study. *The international journal of cardiovascular imaging*. 2008;24(6):617-625.
 129. Glockner JF, Johnston DL, McGee KP. Evaluation of cardiac valvular disease with MR imaging: qualitative and quantitative techniques. *Radiographics*. 2003;23(1):e9.
 130. Strohm O, Schulz-Menger J, Hanlein D, Dietz R, Friedrich MG. Magnetic resonance planimetry of the vena contracta as a new approach to assessment of stenotic heart valves: an in vitro study. *J Magn Reson Imaging*. 2001;14(1):31-34.
 131. Mack MJ, Leon MB, Smith CR, et al. 5-year outcomes of transcatheter aortic valve replacement or surgical aortic valve replacement for high surgical risk patients with aortic stenosis (PARTNER 1): a randomised controlled trial. *Lancet*. 2015;385(9986):2477-2484.
 132. Barbanti M, Petronio AS, Ettori F, et al. 5-Year Outcomes After Transcatheter Aortic Valve Implantation With CoreValve Prosthesis. *JACC. Cardiovascular interventions*. 2015;8(8):1084-1091.
 133. Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. *N Engl J Med*. 2010;363(17):1597-1607.
 134. Smith CR, Leon MB, Mack MJ, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. *N Engl J Med*. 2011;364(23):2187-2198.
 135. Alberts MJ, Peacock WF, Fields LE, et al. Association between once- and twice-daily direct oral anticoagulant adherence in nonvalvular atrial fibrillation patients and rates of ischemic stroke. *Int J Cardiol*. 2016;215:11-13.
 136. Gopal A, Grayburn PA, Mack M, et al. Noncontrast 3D CMR imaging for aortic valve annulus sizing in TAVR. *JACC Cardiovasc Imaging*. 2015;8(3):375-378.
 137. Bello D, Shah DJ, Farah GM, et al. Gadolinium cardiovascular magnetic resonance predicts reversible myocardial dysfunction and remodeling in patients with heart failure undergoing beta-blocker therapy. *Circulation*. 2003;108(16):1945-1953.
 138. Bogaert J, Francone M. Cardiovascular magnetic resonance in pericardial diseases. *J Cardiovasc Magn Reson*. 2009;11:14.
 139. Sengupta PP, Eleid MF, Khandheria BK. Constrictive pericarditis. *Circ J*. 2008;72(10):1555-1562.
 140. Tomoda H, Hoshi ai M, Furuya H, et al. Evaluation of pericardial effusion with computed tomography. *Am Heart J*. 1980;99(6):701-706.
 141. Mulvagh SL, Rokey R, Vick GW, 3rd, Johnston DL. Usefulness of nuclear magnetic resonance imaging for evaluation of pericardial effusions, and comparison with two-dimensional echocardiography. *Am J Cardiol*. 1989;64(16):1002-1009.
 142. Hassink RJ, Aretz HT, Ruskin J, Keane D. Morphology of atrial myocardium in human pulmonary veins: a postmortem analysis in patients with and without atrial fibrillation. *Journal of the American College of Cardiology*. 2003;42(6):1108-1114.
 143. Ghaye B, Szapiro D, Dacher JN, et al. Percutaneous ablation for atrial fibrillation: the role of cross-sectional imaging. *Radiographics*. 2003;23 Spec No:S19-33; discussion S48-50.
 144. Kato R, Lickfett L, Meininger G, et al. Pulmonary vein anatomy in patients undergoing catheter ablation of atrial fibrillation: lessons learned by use of magnetic resonance imaging. *Circulation*. 2003;107(15):2004-2010.
 145. Hauser TH, Yeon SB, Kissinger KV, Josephson ME, Manning WJ. Variation in pulmonary vein size during the cardiac cycle: implications for non-electrocardiogram-gated imaging. *Am Heart J*. 2006;152(5):974 e971-976.
 146. Ng CS, Wells AU, Padley SP. A CT sign of chronic pulmonary arterial hypertension: the ratio of main pulmonary artery to aortic diameter. *J Thorac Imaging*. 1999;14(4):270-278.
 147. Boxt LM, Katz J, Kolb T, Czegledy FP, Barst RJ. Direct quantitation of right and left ventricular volumes with nuclear magnetic resonance imaging in patients with primary pulmonary hypertension. *Journal of the American College of Cardiology*. 1992;19(7):1508-1515.

148. Caro-Dominguez P, Compton G, Humpl T, Manson DE. Pulmonary arterial hypertension in children: diagnosis using ratio of main pulmonary artery to ascending aorta diameter as determined by multi-detector computed tomography. *Pediatr Radiol*. 2016;46(10):1378-1383.
149. Frank H, Globits S, Glogar D, Neuhold A, Kneussl M, Mlczoch J. Detection and quantification of pulmonary artery hypertension with MR imaging: results in 23 patients. *AJR Am J Roentgenol*. 1993;161(1):27-31.
150. Kuriyama K, Gamsu G, Stern RG, Cann CE, Herfkens RJ, Brundage BH. CT-determined pulmonary artery diameters in predicting pulmonary hypertension. *Invest Radiol*. 1984;19(1):16-22.
151. Ghaye B, Ghuysen A, Willems V, et al. Severe pulmonary embolism: pulmonary artery clot load scores and cardiovascular parameters as predictors of mortality. *Radiology*. 2006;239(3):884-891.
152. van der Meer RW, Pattynama PM, van Strijen MJ, et al. Right ventricular dysfunction and pulmonary obstruction index at helical CT: prediction of clinical outcome during 3-month follow-up in patients with acute pulmonary embolism. *Radiology*. 2005;235(3):798-803.
153. Mastora I, Remy-Jardin M, Masson P, et al. Severity of acute pulmonary embolism: evaluation of a new spiral CT angiographic score in correlation with echocardiographic data. *Eur Radiol*. 2003;13(1):29-35.
154. Qanadli SD, El Hajjam M, Vieillard-Baron A, et al. New CT index to quantify arterial obstruction in pulmonary embolism: comparison with angiographic index and echocardiography. *AJR Am J Roentgenol*. 2001;176(6):1415-1420.
155. Cook SC, Dyke PC, 2nd, Raman SV. Management of adults with congenital heart disease with cardiovascular computed tomography. *Journal of cardiovascular computed tomography*. 2008;2(1):12-22.
156. Goo HW, Park IS, Ko JK, Kim YH, Seo DM, Park JJ. Computed tomography for the diagnosis of congenital heart disease in pediatric and adult patients. *The international journal of cardiovascular imaging*. 2005;21(2-3):347-365; discussion 367.
157. Haramati LB, Glickstein JS, Issenberg HJ, Haramati N, Crooke GA. MR imaging and CT of vascular anomalies and connections in patients with congenital heart disease: significance in surgical planning. *Radiographics*. 2002;22(2):337-347; discussion 348-339.
158. Haramati LB, Moche IE, Rivera VT, et al. Computed tomography of partial anomalous pulmonary venous connection in adults. *J Comput Assist Tomogr*. 2003;27(5):743-749.
159. Leschka S, Oechslin E, Husmann L, et al. Pre- and postoperative evaluation of congenital heart disease in children and adults with 64-section CT. *Radiographics*. 2007;27(3):829-846.
160. Memisoglu E, Hobikoglu G, Tepe MS, Norgaz T, Bilsel T. Congenital coronary anomalies in adults: comparison of anatomic course visualization by catheter angiography and electron beam CT. *Catheter Cardiovasc Interv*. 2005;66(1):34-42.
161. Ropers D, Moshage W, Daniel WG, Jessl J, Gottwik M, Achenbach S. Visualization of coronary artery anomalies and their anatomic course by contrast-enhanced electron beam tomography and three-dimensional reconstruction. *Am J Cardiol*. 2001;87(2):193-197.
162. Schmitt R, Froehner S, Brunn J, et al. Congenital anomalies of the coronary arteries: imaging with contrast-enhanced, multidetector computed tomography. *Eur Radiol*. 2005;15(6):1110-1121.
163. Fogel MA, Pawlowski TW, Harris MA, et al. Comparison and usefulness of cardiac magnetic resonance versus computed tomography in infants six months of age or younger with aortic arch anomalies without deep sedation or anesthesia. *Am J Cardiol*. 2011;108(1):120-125.
164. Dodge-Khatami A, Tulevski, II, Bennink GB, et al. Comparable systemic ventricular function in healthy adults and patients with unoperated congenitally corrected transposition using MRI dobutamine stress testing. *Ann Thorac Surg*. 2002;73(6):1759-1764.
165. Fogel MA, Hubbard A, Weinberg PM. A simplified approach for assessment of intracardiac baffles and extracardiac conduits in congenital heart surgery with two- and three-dimensional magnetic resonance imaging. *Am Heart J*. 2001;142(6):1028-1036.
166. Oosterhof T, van Straten A, Vliegen HW, et al. Preoperative thresholds for pulmonary valve replacement in patients with corrected tetralogy of Fallot using cardiovascular magnetic resonance. *Circulation*. 2007;116(5):545-551.
167. Sampson C, Martinez J, Rees S, Somerville J, Underwood R, Longmore D. Evaluation of Fontan's operation by magnetic resonance imaging. *Am J Cardiol*. 1990;65(11):819-821.

168. Didier D, Saint-Martin C, Lapierre C, et al. Coarctation of the aorta: pre and postoperative evaluation with MRI and MR angiography; correlation with echocardiography and surgery. *The international journal of cardiovascular imaging*. 2006;22(3-4):457-475.
169. Geva T, Greil GF, Marshall AC, Landzberg M, Powell AJ. Gadolinium-enhanced 3-dimensional magnetic resonance angiography of pulmonary blood supply in patients with complex pulmonary stenosis or atresia: comparison with x-ray angiography. *Circulation*. 2002;106(4):473-478.
170. Greil GF, Powell AJ, Gildein HP, Geva T. Gadolinium-enhanced three-dimensional magnetic resonance angiography of pulmonary and systemic venous anomalies. *Journal of the American College of Cardiology*. 2002;39(2):335-341.
171. McConnell MV, Ganz P, Selwyn AP, Li W, Edelman RR, Manning WJ. Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. *Circulation*. 1995;92(11):3158-3162.
172. Prasad SK, Soukias N, Hornung T, et al. Role of magnetic resonance angiography in the diagnosis of major aortopulmonary collateral arteries and partial anomalous pulmonary venous drainage. *Circulation*. 2004;109(2):207-214.
173. Fenchel M, Saleh R, Dinh H, et al. Juvenile and adult congenital heart disease: time-resolved 3D contrast-enhanced MR angiography. *Radiology*. 2007;244(2):399-410.
174. Mohrs OK, Petersen SE, Voigtlaender T, et al. Time-resolved contrast-enhanced MR angiography of the thorax in adults with congenital heart disease. *AJR Am J Roentgenol*. 2006;187(4):1107-1114.
175. Beerbaum P, Korperich H, Gieseke J, Barth P, Peuster M, Meyer H. Rapid left-to-right shunt quantification in children by phase-contrast magnetic resonance imaging combined with sensitivity encoding (SENSE). *Circulation*. 2003;108(11):1355-1361.
176. Varaprasathan GA, Araoz PA, Higgins CB, Reddy GP. Quantification of flow dynamics in congenital heart disease: applications of velocity-encoded cine MR imaging. *Radiographics*. 2002;22(4):895-905; discussion 905-896.
177. Whitehead KK, Gillespie MJ, Harris MA, Fogel MA, Rome JJ. Noninvasive quantification of systemic-to-pulmonary collateral flow: a major source of inefficiency in patients with superior cavopulmonary connections. *Circ Cardiovasc Imaging*. 2009;2(5):405-411.
178. American College of Radiology. ACR practice parameter for communication of diagnostic imaging findings. 2011; Available at: <https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CommunicationDiag.pdf>. Accessed November 9, 2015.

*Practice parameters and technical standards are published annually with an effective date of October 1 in the year in which amended, revised or approved by the ACR Council. For practice parameters and technical standards published before 1999, the effective date was January 1 following the year in which the practice parameter or technical standard was amended, revised, or approved by the ACR Council.

Development Chronology for this Practice Parameter

2012 (Resolution 14)

Amended 2014 (Resolution 39)

Revised 2017 (Resolution 21)