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.

Revised 2022 (Resolution 36)*

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 care[1]. 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 considering 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 variables such as the condition of the patient, limitations of available resources, or advances in knowledge or technology after publication of this document. However, a practitioner who employs an approach substantially different from the guidance in this document may consider documenting in the patient record information sufficient to explain the approach taken.

The practice of medicine involves the science, and 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 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 831 N.W.2d 826 (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 computed tomography (CT) and magnetic resonance imaging (MRI) are important noninvasive methods for the assessment of ischemic and nonischemic cardiomyopathies, pericardial disease, cardiac masses, and valvular 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 practice parameters from the ACR have provided practitioners with the educational tools to perform cardiac CT and MR, CTA, and MRA. This parameter deals with the quantitative aspects of CT and MR for cardiovascular imaging.

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 (PC-MRI)
6. Characterization of cardiac morphology and function
7. Assessment of pressure gradients across focal vessel or valvar stenosis using PC-MRI
8. Assessment of volume of myocardial infarction (MI) in ischemic heart disease
9. Assessment of volume of resting and stress-induced hypoperfused myocardium with perfusion imaging
10. Assessment of myocardial tissue in nonischemic cardiomyopathy

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. SPECIFICATIONS OF THE EXAMINATION

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 the evaluation of cardiovascular morphology and function. There are 2 techniques for synchronizing the electrocardiogram (ECG) signal with the cardiac cycle: prospective and retrospective [3].

    Prospective cardiac gating 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, optimizing visualization of a vascular structure and, as in the case of coronary CTA, reducing patient exposure to ionizing radiation.

    Retrospective cardiac gating acquires data throughout the cardiac cycle over multiple beats. This technique is more prone to arrhythmia-related artifacts, although this effect is reduced with arrhythmia software and the latest generation scanners due to improved temporal resolution.

b. Measurements:

i. Distance and cross-sectional diameter measurements

Many cardiovascular imagers use standard multiplanar imaging and assess individual vessels using multiplanar reconstructions (MPR) perpendicular to the vessel axis in orthogonal planes. This is typically done in an interactive fashion with various segments of the vessels being evaluated sequentially for areas of plaque and stenosis. Alternatively, a widely used approach for morphological evaluation and measurement is to use curved multiplanar reconstructions (curved MPR), derived from the volumetric data set. 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 from curved MPR views. Pitfalls include inaccurate placement of the centerline by automated software that can cause artifactual stenoses. This most often occurs in small vessels such as the coronary arteries or calf vessels. On MRA, gradient nonlinearity can cause in-plane and out-of-plane image distortion that leads to incorrect vessel measurements.

Cross-sectional diameter measurements can be performed using a curved MPR. If an area of dilatation or stenosis is suspected, the area can be quantified using reference measurements from adjacent closest normal vessel or within a 1 cm distance. The diameter of the abnormal segment is divided by the reference normal diameter to arrive at a percentage of stenosis or dilatation (percentage stenosis or dilatation equals abnormal segment [millimeters] 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 one or more reference normal areas because of diffuse calcified and noncalcified plaque. If only one 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.

ii. Cross-sectional area, volume, and angle measurements

Stenosis: Stenosis of the arteries is far more common than dilation and is usually due to negative remodeling and atherosclerosis but can also be secondary to other causes such as vasculitis, dissection, or congenital. Measurements of stenoses are done from the inner wall to the opposite inner wall of the vessel lumen. 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 small vessels, such as the coronary arteries and anterior tibial artery. MRA is used less frequently for small vessels to characterize stenosis and dilation because of its spatial resolution. An advantage of MRI is that calcification is not readily visible, and therefore, blooming effects seen with CTA do not impact MRA. For this reason, MRA can be an alternative in severe vascular calcification.

Atherosclerosis: When atherosclerotic plaque is present, its precise anatomic location, severity, and length of stenosis should be 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 (peripheral or visceral) vessels, limitations in spatial resolution may preclude accurate use of percentage reduction, and qualitative analysis is used (mild, moderate, or severe). Coronary CTA has its own standardized grading system, with CAD-RADS being the most widely used: 0% (no visible stenosis), 1% to 24% (minimal stenosis), 25% to 49% (mild stenosis), 50% to 69% (moderate stenosis), 70% to 99% (severe stenosis), and 100% (occluded) [4]. In addition, with CTA, the plaque characteristics are typically reported as noncalcified, calcified, or partially calcified.

Fractional flow reserve by CT (FFRct) is an increasingly used and FDA-approved clinical tool that complements the coronary CTA source data by providing a calculated flow assessment of the coronary arteries. In selected cases of intermediate stenosis, FFRct can improve specificity of coronary CTA and markedly improve clinical decision making. FFRct values should be evaluated in the clinical context and

categorized as follows: I. >0.8, stenosis: not hemodynamically significant; II. 0.80 to 0.76, stenosis: borderline hemodynamically significant; and III. ≤0.75, stenosis: hemodynamically significant. Borderline hemodynamically significant stenosis needs further risk stratification [5].

Vasculitis: MRA and CTA have the unique advantage of not only evaluating for luminal narrowing but also allowing direct visualization of the vessel wall. MRI typically provides superior soft tissue contrast that can aid the detection of mural inflammation and edema [6] 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. An abnormally thickened artery wall may indicate the presence of vasculitis. In general, the aortic wall should be no thicker than approximately 2 mm, although it differs by age and sex [7].

Dissection: It is important to recognize and report:

- The classification of aortic dissection based on location and extent of the flap
- Aortic size (length and width) and the size of true and false lumen
- Dilatation of the aortic diameter and further extension of the dissection flap
- 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

Pulmonary veins (PVs): PV stenosis is a well-known complication of ablation procedure that has been widely used to treat atrial fibrillation (AF) and usually ablates the atrial myocardium inside the PVs to disconnect an abnormal interaction with the left atrium (LA) [8]. CT of PVs has been the most commonly used modality to detect postprocedure stenoses, but MRI is alternatively used. Because the PV size varies throughout the cardiac cycle and the difference between maximum and minimum diameter is 15% ± 8% [8], ECG-gated CTA acquisitions are preferred with images evaluated during late systole. Preprocedural CTA or MRA for the cross-sectional measurement of PV ostia is beneficial for selecting the optimal circular catheter. Furthermore, because 38% of patients with AF have variant anatomy of PVs, evaluating the number and location of PVs is useful in ascertaining that all PV orifices are evaluated during the procedure.

Dilatation (ectasia, aneurysm): Dilatation of the arteries is often due to positive remodeling and atherosclerosis, although multiple other causes exist, including vasculitis, connective tissue diseases, and trauma.

An aneurysm is defined by dilatation of an artery to greater than 1.5 times the diameter of the adjacent normal segment. Measurements of an aneurysm's cross-sectional area can be calculated from longitudinal straightened MPR and are generally done from one outer wall to the opposite outer wall (from the adventitial side of the vessel wall). Angle measurement are very helpful for follow-up of thoracoabdominal aortic aneurysms and iliac artery aneurysms after endovascular repair [9]. Mentioning how we measured in the report helps consistency with future or prior comparison studies.
.

In baseline and follow-up imaging studies, it is helpful to make aortic measurement at conventional locations to facilitate comparison. It is typical to make double-oblique short-axis measurements at the following locations:

- Aortic annulus (if valve replacement is being considered)
- Sinuses of Valsalva
- Sinotubular junction
- Ascending thoracic aorta at the level of the right pulmonary artery
- Transverse aortic arch between the left common carotid and subclavian artery origins
- Aortic isthmus (site adjacent to the ductus ligament insertion)
- Descending thoracic aorta at the level of the right pulmonary artery
- Diaphragmatic hiatus
- Celiac plexus and/or superior mesenteric artery origin

- Renal artery origin
- Infrarenal abdominal aorta midway between renal artery origins and the aortic bifurcation
- Aortic bifurcation
- Common iliac arteries

c. Attenuation (region-of-interest characterization)

Attenuation measurement of the arterial wall can be obtained for diagnosis of intramural hematoma, acute hemorrhage within intraluminal (or mural) thrombus, and atherosclerotic plaques. On CT, a region of interest (ROI) is placed on the arterial wall, and a Hounsfield unit (HU) measurement is obtained that represents an average pixel value. On MRI, hyperintensity on intrinsic T1-weighted sequences can also be used to depict methemoglobin in the acute and subacute hematoma.

When there is suspicion of intramural hematoma, a crescent-shaped high attenuation in the aortic wall can be seen in noncontrast study representing acute hemorrhage, but it is important to repeat the measurements after intravenous (IV) contrast because intramural hematoma does not enhance [10]. Within the intraluminal thrombus, the presence of focal hyperdensity (CT) or hyperintensity on intrinsic T1-weighted sequence (MR) may suggest acute hemorrhage and potential impending rupture. Also, a fissure or dissection within the intraluminal thrombus may predict a higher risk of rupture. In both intramural hematoma and intraluminal thrombus, the extent (both length and width) and branch vessel involvement should be noted.

In atherosclerotic plaques, optimally, it should be possible to categorize as primarily calcified, fibrous, or fatty in density. Because it is difficult to confidently differentiate fibrous and fatty atherosclerotic plaques in small vessels, the plaques can also be characterized as calcified, noncalcified, or partially calcified [11].

The presence of high-risk features in the coronary artery plaques, such as low density (HU < 30), positive remodeling, spotty calcification, or napkin ring signs, is associated with acute coronary syndrome [12].

2. Velocity and flow quantification: PC-MRI

Velocity and flow quantification with MRI are achieved using phase-contrast imaging [13-15]. 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.

The most important parameter for PC-MRI is the velocity encoding variable (V_{enc}). The 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 and sensitivity of the flow measurement. Velocity flow is measured by accurately drawing an ROI that includes the entire lumen of the vessel being evaluated [16]. 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.

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, 4-D flow) [17]. The 4D-flow CMR data can be co-registered with cine images and displayed with color coded velocity information. This overlay allows visualization of complex flow patterns associated with cardiovascular disease. Time-resolved contrast-enhanced MRA may be helpful for identifying collaterals and the presence of flow reversal [18,19].

Most current noninvasive angiographic techniques rely solely on the morphologic assessment of the vasculature. Phase-contrast MRA assesses the hemodynamic consequences of an arterial lesion. Phase-contrast 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 of velocity, volume, rate, and direction.

Pressure gradients across an arterial stenosis are used to determine its hemodynamic significance and therapy. Peak flow velocity is determined on PC-MRI. 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.

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 [SV]). 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.

In cardiac imaging, phase-contrast MR can be used for functional assessment of flow through the aortic and pulmonic valve. A unique evaluation in patients with suspected or known congenital heart disease for a left-to-right shunt is to use the pulmonary (Qp) to systemic (Qs) blood flow ratio (Qp/Qs ratio) [20,21]. 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 (ie, 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 a 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 (ie, 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 PC-MRI performed perpendicular to both the main pulmonary artery and the ascending thoracic aorta. In patients with suspected systemic to pulmonary collateral flow, the pulmonary flow can be estimated using the pulmonary venous return, and the systemic flow can be estimated using the caval return. The degree of systemic-to-pulmonary collateral flow affects immediate postoperative outcomes and can be intervened upon prior to surgery [22-25].

In addition, phase-contrast MR can quantify the volume of mitral valve regurgitation. The most frequent method to quantify the mitral regurgitation volume is left ventricular SV minus aortic forward flow in phase-contrast [26,27].

3. Quantitative techniques specific to cardiac MRI and CT

Transthoracic echocardiography remains the primary screening tool for evaluating cardiac morphology and function [28]. However, evaluation with echocardiography relies on operator skill, and variability in scanning technique may contribute to intra- and interobserver variation [29]. Such variation is notably higher with echocardiography than with MRI. The IV administration of contrast agents enables the determination of myocardial perfusion and myocardial delayed enhancement on MRI and more recently on CT.

Many of the measurement standards used for clinical cardiac CT and MRI are derived from those of echocardiography [30]. It is important to note that specific thresholds of measurement for healthy individuals vary based on body habitus, race, sex, and age [31-36]. Moreover, imaging technique itself can result in differences in measurement. For example, the actual pulse sequence used for cardiac MRI (ie, fast gradient echo versus steady state free precession) may affect left ventricular measurements [37-39], although field strength (1.5T versus 3T) does not appear to have any significant influence [40-42].

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. The IV

administration of contrast agents enables the determination of myocardial perfusion and myocardial late gadolinium enhancement (LGE) on MRI.

a. Myocardium

i. Wall thickness:

Myocardial wall thickness is traditionally measured on end-diastolic images. In healthy adults, end-diastolic left ventricular thickness is typically between 6 and 12 mm [43]. To minimize volume averaging effects, image acquisition is typically performed in a plane perpendicular to the wall being measured. For the left ventricle (LV), 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 views. The basal anteroseptal segment is best evaluated on a 3-chamber view.

ii. Myocardial mass (left ventricular mass):

The myocardial mass of the LV can be determined by measuring end-diastolic LV myocardial volume and multiplying this by the specific gravity of myocardium (1.05 g/mL) [33].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 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 exclude the left and right ventricular outflow tracts, although others draw endocardial contours up to the aortic and pulmonic valve planes. The planimetry measurement between endocardial and epicardial tracings represents the myocardial area. 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 end-diastolic volume (EDV) or end-systolic volume (ESV) measurements for most examinations.

The exclusion of the papillary muscles reduces postprocessing time requirements by obviating a separate trace of the papillary muscles [44]. 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 [45]. 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 (EF) [45].

Because of the variety in the method of measurements that existed among readers, it is recommended that these methods of tracing in any specific lab be clear and similar, at least among cardiac imagers in one lab for the purpose of comparison and follow-up of their patients. The cardiac imager may follow the major society guidelines for the various methods of measurement [46].

iii. Myocardial segmentation and nomenclature:

Since 2002, the American Heart Association [47] has recommended 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 the apical cap to compose the total 17 segments of the LV.

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

b. Cardiac chambers**:**

i. Ventricles:

Ventricular internal diameter and volumes can be measured linearly using short and long axis dimensions or through chamber tracing, respectively. In cardiac CT, the ventricular volume can only be accurately calculated in retrospective gated studies including sufficient phases. When quantifying the LV using 2-D linear measurements, the LV's internal diameters are measured in systole and diastole in the basal to mid-cavity from the endocardium of the anteroseptal wall to the endocardium of the inferolateral wall [48]. 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 [49]. Left ventricular volume calculations can often be made quickly using artificial intelligence postprocessing software for automated determination of chamber contours [50]. These values can be indexed to body surface area (BSA) or to calculate the ratio of right ventricle (RV) to LV size as an assessment of RV enlargement [51,52].

ii. Atria:

The normal measurements of the LA and right atrium (RA) are dependent upon the modality used to assess volumes. Echocardiographic standards using 2-D biplane measurements generally underestimate volumes. Limited data exists on the standardization of normative values [53-57]. End-systolic measurements should be performed for both LA and RA linear and volumetric measurements. LA linear measurements are typically performed in the anterior-posterior (or left ventricular outflow tract) view, while RA linear measurements are performed on the 4-chamber view. For LA volumetric measurements, the pulmonary veins should be excluded. Cardiac MR is considered the gold standard for atrial volumetric measurements and function [58,59]. Cardiac MR–derived biplane measurements also have a good correlation with full-volume methods. Cardiac CT is considered more accurate than 2-D echocardiography, and volumes correlate well with MRI [60].

c. Myocardial function:

i. Ventricular function:

The evaluation of cardiac function can provide valuable prognostic information on ischemic heart disease. The EF predicts outcome better than the number of vessels involved [61], and prognosis after MI is closely related to the degree of LV contractile dysfunction [62].

Volumetric and EF measurements by MRI and CT have been shown to be very comparable [50,63]. An individual physician, or by consensus an imaging laboratory, should establish a convention by which endocardial and 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.

Ventricular EF is defined by the following equation [32,48,49]: EF [%] = 100 × [EDV – ESV] / EDV. EDV and ESV are determined using the modified Simpson method by drawing endocardial tracings (as described above), preferably 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.

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) and stroke volume index (SVI= SV/ BSA)
- Ejection fraction (EF [%] = 100 × [EDV – ESV]/ EDV)
- Cardiac output (CO = SV × heart rate)

- Cardiac index (CI = CO / body surface area (BSA) = SV x 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 to BSA and/or body mass index is often helpful clinically to account from differences in patient habitus and size.

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 [64]. In the 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 [65-67]. 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 thoracic aorta is of normal size [65-67]. 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 [68]. 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 [66,69].

Acute 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 [65,70]. The ratio of the RV to LV diameters is an accurate sign for RV dysfunction [70]. Other signs have been described, including bowing of the interventricular septum and reflux of contrast medium into the inferior vena cava. The sizes of the azygous vein, superior vena cava, and pulmonary artery are also indirect measures of right heart dysfunction and pulmonary hypertension. Mean pulmonary artery (PA) pressure correlates linearly with main PA diameter [71], and a PA diameter greater than 30 mm indicates a PA pressure greater than 20 mm Hg [72].

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. Wall motion can be visually assessed during systole as normal, hypokinetic (decreased wall motion), hyperkinetic (increased wall motion), akinetic (no wall motion), or dyskinetic (paradoxical motion or reversal of wall motion) ie, aneurysm). Atrioventricular dyssynchrony occurs when the timing between atrial and ventricular contractions does not favor forward flow. Interventricular desynchrony occurs with a timing difference between the ventricles, and intraventricular desynchrony occurs when the sequence of activation and relaxation of segments within the LC or RV are abnormal.

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 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 [73,74].

d. Myocardial perfusion:

Among cross-sectional imaging modalities, myocardial perfusion imaging is most commonly performed with MRI, but recently, CT is increasingly used because of the advancements of the last generation CT scanners that decrease radiation exposure and scan time [75].

Stress perfusion cardiac MR is performed during administration of a pharmacologic vasodilator stress agent

(eg, adenosine, dipyridamole, regadenoson) and concurrent imaging of myocardium enhancement using short axis rapid T1-weighted images. These stress first pass perfusion images are then compared with perfusion images acquired at rest (second pass perfusion), enabling a visual assessment of regional differences in the myocardial enhancement at stress and at rest. Focal areas with inducible myocardial ischemia after pharmacologic stress agent show decreased or lack of perfusion at stress (darker) compared with at rest (enhanced), whereas areas of chronic myocardial infarct show decreased or lack of perfusion at stress and at rest [73,74,76,77].

e. LGE:

LGE is used to depict myocardium focal necrosis, fibrosis/scarring, or infiltration.

Abnormal regions of myocardium appear brighter than adjacent normal myocardium in LGE and are therefore also termed "hyperenhancement." The underlying mechanisms for LGE reflect the relative faster washout of contrast in normal myocardium compared with prolonged retention of contrast in the abnormal tissue due to enlargement of the extracellular space [78,79]. Imaging is typically performed approximately 10 minutes following gadolinium-chelate contrast agent injection in short axis, 2-, 3-, and 4-chamber views.

LGE is seen in both acute and chronic MI [80]. In MI, the LGE begins in the subendocardial region, as this represents the end-vessel or "at risk" territory of the myocardium as coronary arteries originates from the epicardial surface of the heart and branches dive deep into the subepicardium, mesocardium, and ultimately into capillaries at the subendocardium.

The segmental transmurality of the hyperenhancement has been shown to correlate with the likelihood for functional improvement following a coronary revascularization procedure. Transmurality of LGE is best characterized in quartiles, as less than 0% to 25%, 26% to 50%, 51% to 75%, and 76% to 100% of the myocardial thickness [81]. 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%) [82,83].

More recently, the use of CT for myocardial delayed enhancement 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 an independent predictor of mortality [84]. A volume greater than 2.8 cm^3 is associated with inducibility of ventricular tachycardia [83,85]. Software quantification for delayed enhancement volume is possible using manual and automated thresholding techniques. Although there is no consensus regarding the technique of quantification of LGE, and the quantification depends greatly upon the method used, results using 6 SDs above the threshold and full width half maximum methods had no difference between visual assessments. All other thresholding techniques resulted in significant differences of LGE volumes for patients with hypertrophic cardiomyopathy [86].

T1-mapping and extracellular volume fraction (ECV) mapping cardiac MR ACR-NASCI-SPR Practice Parameter for the Performance and Interpretation of Cardiac Magnetic Resonance Imaging (MRI) [87]:

Native T1 relaxation time (ie, native mapping) and ECV differences, in normal and focal or diffuse fibrotic myocardium, may be used to detect and quantify myocardial disease, which may not be as evident using other MR sequences. These techniques may be particularly helpful for identifying diffuse myocardial processes such as diffuse myocardial fibrosis in hypertrophic cardiomyopathy, muscular dystrophy, and cardiac amyloidosis [88].

The ECV can be calculated using the values from myocardium and blood, before and after injection of contrast, and the patient's hematocrit [89]. T1 mapping can also be helpful in determining intrinsic myocardial disease in patients who can otherwise not receive IV contrast. There is a large amount of variability between vendors and MRI scanner models for normal T1 values based upon sequence options, and field strengths; thus, it is incumbent upon each site to determine their normal range of T1 values locally,

by performing quality control using a standardized phantom [90,91].

f. T2-weighted and T2 mapping sequences cardiac MR:
Water in the myocardium causes longer T2 relaxation times and increased signal intensity. High signal intensity on T2-weighted and abnormal values on T2 mapping sequences are the result of myocardial inflammation or edema frequently seen with myocarditis, MI, and cardiomyopathies such as amyloidosis. In STIR, the extent of high T2-signal intensity in ischemia-associated myocardial edema reflects the area of risk that may include regions of reversible injury as well [92]. T2 mapping normal values varies with the strength of the magnetic field and has been described at 1.5 T as 52.18 ± 3.4 ms and at 3T as 45.1 ms [93,94].

g. Coronary artery calcium scoring:

Calcium-scoring images are acquired with noncontrast ECG-gated CT to optimally visualize and quantify calcified plaque. High "calcium scores" are associated with an increased risk of MI, and a calcium score of 0 has a very low but nonzero risk of a major adverse cardiac event [95,96].

Coronary calcium scores were first reported more than 20 years ago by Agatston et al [97,98] using electron-beam CT whereby coronary calcium lesions with 3 adjacent pixels of >130 HU were assessed using an ROI. 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 in the coronary system.

Two other methods for measuring coronary calcium are the volume score and the mass score. The volume score reflects the volume of calcium above the threshold; the mass score uses a phantom to calibrate the mass (milligram) 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.

h. Pericardial disease**:**

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, or calcified 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.

CT and MR images can be used to measure the pericardial thickness in which normal is 1.2 mm ± 0.5 mm, and abnormal thickness is defined as a thickness ≥3 mm [99-102]. 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 of pericardial constriction. Contrast enhancement is an additional qualitative finding associated with abnormal pericardium.

A fluid collection with attenuation close to that of water (approximately 20 HU) is likely to be a simple effusion, but attenuation measurements greater than that may suggest malignancy, hemopericardium (HU ≥35), purulent exudate, or effusion associated with hypothyroidism [103]. MR can also characterize pericardial fluid, although qualitatively, with the use of multiple pulse sequences; hemorrhagic effusion is characterized by high signal on intrinsic T1-weighted SE images and low intensity on gradient echo (GRE) cine images. Another important feature of CT is its ability to detect pericardial calcifications, a finding that may be indicative of constrictive pericarditis. MRI is also helpful for the evaluation of pericardial adhesion and constriction with tagging sequence and cine techniques to detect ventricular coupling.

i. Transcatheter Aortic Valve Replacement (TAVR):

In high surgical risk patients with severe aortic stenosis, TAVR has demonstrated long-term results comparable to open surgical repair [104,105]. Preprocedural imaging evaluation should include noncontrast and contrast cardiac-gated evaluation and measurement of the following intracardiac and aortic structures [106].

- Aortic valve calcium score
- Presence and severity of calcifications in the annulus and sub-annular region
- Left cardiac chambers and left atrial appendage (LAA) for thrombus
- Alignment of the LV outlet tract (LVOT)
- Dimensions (perimeter, maximum and minimum diameters, and area) of the aortic annulus at the maximum aortic valve opening, typically during systole.
- Width of the aortic sinus (cusp to commissure distance), number of cusps (tricuspid or bicuspid)
- Coronary ostia height from the annulus
- Width and height of sinotubular junction
- Width and tortuosity of ascending and descending thoracic aorta, and abdominal aorta
- Vascular access (subclavian arteries, common and external iliac arteries, and common femoral arteries): minimal luminal diameter, tortuosity, and extend and distribution of atherosclerotic disease
- Incidental noncardiovascular findings

j. Transmitral Valve Replacement (TMVR):

Preprocedural evaluation of the left ventricular outflow track for TMVR is increasingly used for predicting post procedural neo-LVOT stenosis with balloon expandable valves. Postprocessing allows simulation of the percutaneous valve in position using CAD. When the predicted neo-LVOT surface area is $\leq$1.9 cm^2, the result is 100% sensitivity and 96.8% specificity for predicting TMVR-induced LVOT obstruction of >10 mm Hg [107].

k. Pre- and postimplantation LAA closure device imaging:

LAA occlusion is a reasonable alternative to long-term anticoagulation therapy for patients with AF to prevent stroke [108,109].

Preprocedure imaging is performed to assess LAA measurements (length, width, and orifice size/area) for size optimization of the closure device; LAA shape, size, and relationship with adjacent structures; presence of LAA thrombus, which is a contraindication for device occlusion; LA volume; and interatrial septal abnormalities (patent foramen ovale and septal defects [110], lipomatous hypertrophy, and aneurysm). When there is an LAA filling defect in the arterial phase, it is important to differentiate between slow flow (or mixed contrast-blood flow) and thrombus. The thrombus tends to be well defined and hypodense (<100 HU) and persistent in the delayed phase (acquired 60 sec after the arterial phase); on the contrary, slow flow is more ill-defined with heterogeneous attenuation and disappears in the delayed phase. Postprocedure imaging is performed for device surveillance to assess atrial-side device thrombus, residual leak, device embolization and position, and pericardial effusion [111,112].

V. DOCUMENTATION

Reporting should be in accordance with the ACR Practice Parameter for Communication of Diagnostic Imaging Findings [113].

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.

Equipment performance monitoring should be in accordance with the ACR–AAPM Technical Standard for Diagnostic Medical Physics Performance Monitoring of Computed Tomography (CT) Equipment [114] or ACR–AAPM Technical Standard for Diagnostic Medical Physics Performance Monitoring of Magnetic Resonance Imaging (MRI) Equipment [115], as appropriate.

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 Quality Control & Improvement, Safety, Infection Control, and Patient Education* on the ACR website (https://www.acr.org/Advocacy-and-Economics/ACR-Position-Statements/Quality-Control-and-Improvement).

ACKNOWLEDGEMENTS

This practice parameter was revised according to the process described under the heading *The Process for Developing ACR Practice Parameters and Technical Standards* on the ACR website (https://www.acr.org/Clinical-Resources/Practice-Parameters-and-Technical-Standards) 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.

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

ACR
Larissa Braga Casaburi, MD, MPH, MHA, Co-Chair
Andrew L. Rivard, MD, Co-Chair
Klaus Hagspiel, MD
Beverley Newman, MD
Ashley Prosper, MD

NASCI
Dhiraj Baruah, MD
Diana Litmanovich, MD
Markus S. Renno, MD, MPH
Quynh A. Truong, MD, MPH

SPR
Marcos Ferreira Botelho, MD
Maryam Ghadimi Mahani, MD
Ramkumar Krishnamurthy, PhD
Christopher Lam, MD
Evan Zucker, MD

Committee on Body Imaging – Cardiovascular
(ACR Committee responsible for sponsoring the draft through the process)

Klaus Hagspiel, MD, Chair
Lucia Flors Blasco, MD, PhD
Larissa Braga Casaburi, MD, MPH, MHA
Yoo Jin Lee, BS, MS, MD
Ashley Prosper, MD
Steven S. Raman, MD
Andrew L. Rivard, MD
Phillip M. Young, MD

Committee on Practice Parameters – Pediatric Radiology
(ACR Committee responsible for sponsoring the draft through the process)

Terry L. Levin, MD, FACR, Chair
John B. Amodio, MD, FACR
Jesse Berman, MD
Tara M. Catanzano, MB, BCh
Harris L. Cohen, MD, FACR
Jane Sun Kim, MD
Jennifer A. Knight, MD
Jessica Kurian, MD
Matthew P. Lungren, MD, MPH
Helen R. Nadel, MD

Committee on Practice Parameters – Pediatric Radiology

Kassa Darge, MD, PhD
Dorothy L. Gilbertson-Dahdal, MD
Lauren P. Golding, MD
Safwan S. Halabi, MD
Jason Higgins, DO
Erica Poletto, MD
Richard B. Towbin, MD, FACR
Andrew T. Trout, MD
Esben S. Vogelius, MD

Andrew B. Rosenkrantz, MD, Chair, Commission on Body Imaging
Richard A. Barth, MD, FACR, Chair, Commission on Pediatric Radiology
David B. Larson, MD, MBA, Chair, Commission on Quality and Safety
Mary S. Newell, MD, FACR, Chair, Committee on Practice Parameters and Technical Standards

Comments Reconciliation Committee

Jamaal Benjamin, MD, PhD– CSC Chair
Juan C. Batlle, MD, MBA– CSC Co-Chair
Richard A. Barth, MD, FACR
Dhiraj Baruah, MD
Marcos Ferreira Botelho, MD
Larissa Braga Casaburi, MD, MPH, MHA
Timothy A. Crummy, MD, FACR
Samuel A. Einstein, PhD
Maryam Ghadimi-Mahani, MD
Klaus Hagspiel, MD
Amy L. Kotsenas, MD, FACR
Ramkumar Krishnamurthy, PhD
Christopher Lam, MD
David B. Larson, MD, MBA
Paul A. Larson, MD, FACR
Terry L. Levin, MD, FACR
Diana Litmanovich, MD
Mary S. Newell, MD, FACR
Beverley Newman, MD
Ashley Prosper, MD
Markus S. Renno, MD, MPH
Andrew L. Rivard, MD
Andrew B. Rosenkrantz, MD
Lisa Michele Fox Thain, MD
Quynh A. Truong, MD
Evan Zucker, MD

REFERENCES

1. American College of Radiology. ACR practice parameter for performing and interpreting magnetic resonance imaging (MRI). Available at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Perf-Interpret.pdf. Accessed July 12, 2021.

2. American College of Radiology. ACR practice parameter for performance and interpreting diagnostic computed tomography (CT). Available at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CT-Perf-Interpret.pdf. Accessed July 12, 2021.

3. Cody DD, Mahesh M. Technologic Advances in Multidetector CT with a Focus on Cardiac Imaging. RadioGraphics 2007;27:1829-37.

4. 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.

5. Nørgaard BL, Fairbairn TA, Safian RD, et al. Coronary CT Angiography-derived Fractional Flow Reserve Testing in Patients with Stable Coronary Artery Disease: Recommendations on Interpretation and Reporting. Radiology: Cardiothoracic Imaging 2019;1:e190050.

6. Matsunaga N, Hayashi K, Sakamoto I, et al. Takayasu arteritis: MR manifestations and diagnosis of acute and chronic phase. J Magn Reson Imaging 1998;8:406-14.

7. Rosero EB, Peshock RM, Khera A, Clagett P, Lo H, Timaran CH. Sex, race, and age distributions of mean aortic wall thickness in a multiethnic population-based sample. J Vasc Surg 2011;53:950-7.

8. 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;152:974 e1-6.

9. Bryce Y, Rogoff P, Romanelli D, Reichle R. Endovascular Repair of Abdominal Aortic Aneurysms: Vascular Anatomy, Device Selection, Procedure, and Procedure-specific Complications. RadioGraphics 2015;35:593-615.

10. Buckley O, Rybicki FJ, Gerson DS, et al. Imaging features of intramural hematoma of the aorta. The

international journal of cardiovascular imaging 2010;26:65-76.
11. Saremi F, Achenbach S. Coronary plaque characterization using CT. AJR Am J Roentgenol 2015;204:W249-60.
12. Pflederer T, Marwan M, Schepis T, et al. Characterization of culprit lesions in acute coronary syndromes using coronary dual-source CT angiography. Atherosclerosis 2010;211:437-44.
13. Lotz J, Meier C, Leppert A, Galanski M. Cardiovascular flow measurement with phase-contrast MR imaging: basic facts and implementation. Radiographics 2002;22:651-71.
14. 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;198:344-50.
15. Srichai MB, Lim RP, Wong S, Lee VS. Cardiovascular Applications of Phase-Contrast MRI. American Journal of Roentgenology 2009;192:662-75.
16. Carr JC, Finn JP. MR imaging of the thoracic aorta. Magn Reson Imaging Clin N Am 2003;11:135-48.
17. 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;46:317-25.
18. 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;33:225-31.
19. Korosec FR, Frayne R, Grist TM, Mistretta CA. Time-resolved contrast-enhanced 3D MR angiography. Magn Reson Med 1996;36:345-51.
20. 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;108:1355-61.
21. 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;22:895-905; discussion 05-6.
22. 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;2:405-11.
23. Grosse-Wortmann L, Drolet C, Dragulescu A, et al. Aortopulmonary collateral flow volume affects early postoperative outcome after Fontan completion: a multimodality study. The Journal of thoracic and cardiovascular surgery 2012;144:1329-36.
24. Odenwald T, Quail MA, Giardini A, et al. Systemic to pulmonary collateral blood flow influences early outcomes following the total cavopulmonary connection. Heart 2012;98:934-40.
25. Glatz AC, Rome JJ, Small AJ, et al. Systemic-to-pulmonary collateral flow, as measured by cardiac magnetic resonance imaging, is associated with acute post-Fontan clinical outcomes. Circ Cardiovasc Imaging 2012;5:218-25.
26. Garg P, Swift AJ, Zhong L, et al. Assessment of mitral valve regurgitation by cardiovascular magnetic resonance imaging. Nature reviews. Cardiology 2020;17:298-312.
27. Myerson SG, d'Arcy J, Christiansen JP, et al. Determination of Clinical Outcome in Mitral Regurgitation With Cardiovascular Magnetic Resonance Quantification. Circulation 2016;133:2287-96.
28. Douglas PS, Garcia MJ, Haines DE, et al. ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR 2011 Appropriate Use Criteria for Echocardiography. A Report of the American College of Cardiology Foundation Appropriate Use Criteria Task Force, American Society of Echocardiography, American Heart Association, American Society of Nuclear Cardiology, Heart Failure Society of America, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, Society of Critical Care Medicine, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance American College of Chest Physicians. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography 2011;24:229-67.
29. 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;90:29-34.
30. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by

echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Journal of the American Society of Echocardiography : official publication of the American Society of Echocardiography 2015;28:1-39.e14.
31. 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:S357-65.
32. 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;39:1055-60.
33. 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;1:782-6.
34. 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;1:7-21.
35. 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;2:191-8.
36. Lorenz CH. The range of normal values of cardiovascular structures in infants, children, and adolescents measured by magnetic resonance imaging. Pediatr Cardiol 2000;21:37-46.
37. Hori Y, Yamada N, Higashi M, Hirai N, Nakatani S. Rapid evaluation of right and left ventricular function and mass using real-time true-FISP cine MR imaging without breath-hold: comparison with segmented true-FISP cine MR imaging with breath-hold. J Cardiovasc Magn Reson 2003;5:439-50.
38. Papavassiliu T, Kühl HP, Schröder M, et al. Effect of endocardial trabeculae on left ventricular measurements and measurement reproducibility at cardiovascular MR imaging. Radiology 2005;236:57-64.
39. Winter MM, Bernink FJ, Groenink M, et al. Evaluating the systemic right ventricle by CMR: the importance of consistent and reproducible delineation of the cavity. J Cardiovasc Magn Reson 2008;10:40.
40. 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;223:789-97.
41. 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;24:312-8.
42. 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;17:323-9.
43. 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;10:438-42.
44. 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;6:9-16.
45. 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;30:1197-202.
46. Schulz-Menger J, Bluemke DA, Bremerich J, et al. Standardized image interpretation and post processing in cardiovascular magnetic resonance: Society for Cardiovascular Magnetic Resonance (SCMR) Board of Trustees Task Force on Standardized Post Processing. Journal of Cardiovascular Magnetic Resonance 2013;15:35.
47. 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;105:539-42.
48. 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;16:551-9.
49. 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.
50. 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;67:1956-64.
51. Radiology ACo. ACR Appropriateness Criteria®: Known or Suspected Congenital Heart Disease in the Adult. Available at: https://acsearch.acr.org/docs/69355/Narrative/. Accessed November 2, 2021.
52. Bellenger NG, Grothues F, Smith GC, Pennell DJ. Quantification of right and left ventricular function by cardiovascular magnetic resonance. Herz 2000;25:392-9.
53. Kawel-Boehm N, Hetzel SJ, Ambale-Venkatesh B, et al. Reference ranges ("normal values") for cardiovascular magnetic resonance (CMR) in adults and children: 2020 update. J Cardiovasc Magn Reson 2020;22:87.
54. Mahabadi AA, Samy B, Seneviratne SK, et al. Quantitative assessment of left atrial volume by electrocardiographic-gated contrast-enhanced multidetector computed tomography. Journal of cardiovascular computed tomography 2009;3:80-87.
55. Stolzmann P, Scheffel H, Leschka S, et al. Reference values for quantitative left ventricular and left atrial measurements in cardiac computed tomography. European Radiology 2008;18:1625-34.
56. Stolzmann P, Scheffel H, Trindade PT, et al. Left Ventricular and Left Atrial Dimensions and Volumes: Comparison Between Dual-Source CT and Echocardiography. Investigative Radiology 2008;43:284-89.
57. Stojanovska J, Cronin P, Patel S, et al. Reference Normal Absolute and Indexed Values From ECG-Gated MDCT: Left Atrial Volume, Function, and Diameter. American Journal of Roentgenology 2011;197:631-37.
58. Boyd AC, Thomas L. Left atrial volumes: two-dimensional, three-dimensional, cardiac magnetic resonance and computed tomography measurements. Current opinion in cardiology 2014;29:408-16.
59. Whitlock M, Garg A, Gelow J, Jacobson T, Broberg C. Comparison of left and right atrial volume by echocardiography versus cardiac magnetic resonance imaging using the area-length method. Am J Cardiol 2010;106:1345-50.
60. Kühl JT, Lønborg J, Fuchs A, et al. Assessment of left atrial volume and function: a comparative study between echocardiography, magnetic resonance imaging and multi slice computed tomography. The international journal of cardiovascular imaging 2012;28:1061-71.
61. 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.
62. 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;36:1985-91.
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;8:427-33.
64. 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.
65. 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;26:587-91.
66. 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;239:884-91.
67. Truong QA, Bhatia HS, Szymonifka J, et al. A four-tier classification system of pulmonary artery metrics on computed tomography for the diagnosis and prognosis of pulmonary hypertension. Journal of cardiovascular computed tomography 2018;12:60-66.
68. Caro-Domínguez 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;46:1378-83.
69. Kuriyama K, Gamsu G, Stern RG, Cann CE, Herfkens RJ, Brundage BH. CT-determined pulmonary artery diameters in predicting pulmonary hypertension. Invest Radiol 1984;19:16-22.
70. Boxt LM. MR imaging of pulmonary hypertension and right ventricular dysfunction. Magn Reson Imaging Clin N Am 1996;4:307-25.
71. 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;235:798-803.
72. 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;13:29-35.
73. Schwitter J, Wacker CM, Wilke N, et al. MR-IMPACT II: Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary artery disease Trial: perfusion-cardiac magnetic resonance vs. single-photon emission computed tomography for the detection of coronary artery disease: a comparative multicentre, multivendor trial. Eur Heart J 2013;34:775-81.
74. Schwitter J, Wacker CM, van Rossum AC, et al. MR-IMPACT: comparison of perfusion-cardiac magnetic resonance with single-photon emission computed tomography for the detection of coronary artery disease in a multicentre, multivendor, randomized trial. Eur Heart J 2008;29:480-9.
75. Patel AR, Bamberg F, Branch K, et al. Society of cardiovascular computed tomography expert consensus document on myocardial computed tomography perfusion imaging. Journal of cardiovascular computed tomography 2020;14:87-100.
76. 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;22:1881-95.
77. 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;17:27-37.
78. Kim RJ, Chen E-L, Lima JoAC, Judd RM. Myocardial Gd-DTPA Kinetics Determine MRI Contrast Enhancement and Reflect the Extent and Severity of Myocardial Injury After Acute Reperfused Infarction. Circulation 1996;94:3318-26.
79. Wong TC, Piehler K, Meier CG, et al. Association between extracellular matrix expansion quantified by cardiovascular magnetic resonance and short-term mortality. Circulation 2012;126:1206-16.
80. 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;343:1445-53.
81. Gibbons RJ, Valeti US, Araoz PA, Jaffe AS. The quantification of infarct size. Journal of the American College of Cardiology 2004;44:1533-42.
82. Kwon DH, Asamoto L, Popovic ZB, et al. Infarct characterization and quantification by delayed enhancement cardiac magnetic resonance imaging is a powerful independent and incremental predictor of mortality in patients with advanced ischemic cardiomyopathy. Circ Cardiovasc Imaging 2014;7:796-804.
83. Ghannam M, Siontis KC, Cochet H, et al. Risk stratification in patients with nonischemic cardiomyopathy and ventricular arrhythmias based on quantification of intramural delayed enhancement on cardiac magnetic resonance imaging. Journal of cardiovascular electrophysiology 2020;31:1762-69.
84. 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;9:822-32.
85. 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;24:617-25.
86. Spiewak M, Malek LA, Misko J, et al. Comparison of different quantification methods of late gadolinium enhancement in patients with hypertrophic cardiomyopathy. Eur J Radiol 2010;74:e149-53.
87. American College of Radiology. ACR–NASCI–SPR Practice Parameter for the Performance of Cardiac Magnetic Resonance Imaging (MRI). Available at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Cardiac.pdf. Accessed November 2, 2021.
88. Karamitsos TD, Piechnik SK, Banypersad SM, et al. Noncontrast T1 mapping for the diagnosis of cardiac amyloidosis. JACC Cardiovasc Imaging 2013;6:488-97.
89. Haaf P, Garg P, Messroghli DR, Broadbent DA, Greenwood JP, Plein S. Cardiac T1 Mapping and Extracellular Volume (ECV) in clinical practice: a comprehensive review. J Cardiovasc Magn Reson 2016;18:89.
90. Captur G, Bhandari A, Brühl R, et al. T1 mapping performance and measurement repeatability: results from the multi-national T1 mapping standardization phantom program (T1MES). Journal of Cardiovascular Magnetic Resonance 2020;22:31.
91. Messroghli DR, Moon JC, Ferreira VM, et al. Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2* and extracellular volume: A consensus statement by the Society for

Cardiovascular Magnetic Resonance (SCMR) endorsed by the European Association for Cardiovascular Imaging (EACVI). J Cardiovasc Magn Reson 2017;19:75.
92. 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;26:549-57.
93. Giri S, Shah S, Xue H, et al. Myocardial T_2 mapping with respiratory navigator and automatic nonrigid motion correction. Magnetic resonance in medicine 2012;68:1570-78.
94. von Knobelsdorff-Brenkenhoff F, Prothmann M, Dieringer MA, et al. Myocardial T1 and T2 mapping at 3 T: reference values, influencing factors and implications. J Cardiovasc Magn Reson 2013;15:53.
95. Church TS, Levine BD, McGuire DK, et al. Coronary artery calcium score, risk factors, and incident coronary heart disease events. Atherosclerosis 2007;190:224-31.
96. 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;291:210-5.
97. 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;41:467-73.
98. 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;15:827-32.
99. Hort W, Braeun H. [Studies on the dimensions, wall thickness and microscopic structure of the pericardium in normal and pathological conditions]. Archiv fur Kreislaufforschung 1962;38:1-22.
100. Sechtem U, Tscholakoff D, Higgins CB. MRI of the normal pericardium. AJR Am J Roentgenol 1986;147:239-44.
101. Kåsin JI, Kjellevand TO, Kjekshus J, Nesheim GB, Wagstaff A. CT examination of the pericardium and lungs in helicopter pilots exposed to vibration and noise. Aviation, space, and environmental medicine 2012;83:858-64.
102. Delille JP, Hernigou A, Sene V, et al. Maximal thickness of the normal human pericardium assessed by electron-beam computed tomography. Eur Radiol 1999;9:1183-9.
103. Wang ZJ, Reddy GP, Gotway MB, Yeh BM, Hetts SW, Higgins CB. CT and MR imaging of pericardial disease. Radiographics 2003;23 Spec No:S167-80.
104. Smith CR, Leon MB, Mack MJ, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. N Engl J Med 2011;364:2187-98.
105. Gopal A, Grayburn PA, Mack M, et al. Noncontrast 3D CMR imaging for aortic valve annulus sizing in TAVR. JACC Cardiovasc Imaging 2015;8:375-8.
106. Blanke P, Weir-McCall JR, Achenbach S, et al. Computed Tomography Imaging in the Context of Transcatheter Aortic Valve Implantation (TAVI)/Transcatheter Aortic Valve Replacement (TAVR). JACC: Cardiovascular Imaging 2019;12:1-24.
107. Wang DD, Eng MH, Greenbaum AB, et al. Validating a prediction modeling tool for left ventricular outflow tract (LVOT) obstruction after transcatheter mitral valve replacement (TMVR). Catheter Cardiovasc Interv 2018;92:379-87.
108. Reddy VY, Sievert H, Halperin J, et al. Percutaneous Left Atrial Appendage Closure vs Warfarin for Atrial Fibrillation: A Randomized Clinical Trial. JAMA 2014;312:1988-98.
109. Belgaid DR, Khan Z, Zaidi M, Hobbs A. Prospective randomized evaluation of the watchman left atrial appendage closure device in patients with atrial fibrillation versus long-term warfarin therapy: The PREVAIL trial. Int J Cardiol 2016;219:177-9.
110. Prosper A, Shinbane J, Maliglig A, Saremi F, Wilcox A, Lee C. Left Atrial Appendage Mechanical Exclusion: Procedural Planning Using Cardiovascular Computed Tomographic Angiography. J Thorac Imaging 2020;35:W107-w18.
111. Saw J, Fahmy P, DeJong P, et al. Cardiac CT angiography for device surveillance after endovascular left atrial appendage closure. European heart journal cardiovascular Imaging 2015;16:1198-206.
112. Rajiah P, Alkhouli M, Thaden J, Foley T, Williamson E, Ranganath P. Pre- and Postprocedural CT of Transcatheter Left Atrial Appendage Closure Devices. Radiographics 2021;41:680-98.
113. American College of Radiology. ACR practice parameter for communication of diagnostic imaging findings. Available at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CommunicationDiag.pdf. Accessed July 22, 2021.
114. American College of Radiology. ACR–AAPM Technical Standard for Diagnostic Medical Physics

Performance Monitoring of Computed Tomography (CT) Equipment. Availale at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/CT-Equip.pdf. Accessed January 21, 2022.

115. American College of Radiology. ACR–AAPM Technical Standard for Diagnostic Medical Physics Performance Monitoring of Magnetic Resonance Imaging (MRI) Equipment. Available at: https://www.acr.org/-/media/ACR/Files/Practice-Parameters/MR-Equip.pdf. Accessed January 21, 2022.

---

*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)
Revised 2022 (Resolution 36)
Amended 2023 (Resolution 2c)