Anatomy and Terminology for the Interpretation and Reporting of Cardiac MDCT: Part 2, CT Angiography, Cardiac Function Assessment, and Noncoronary and Extracardiac Findings

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OBJECTIVE. In this part 2 of our two-part article, we aim to describe the evaluation and interpretation of coronary CTA, cardiac structures, and cardiac functional assessment using an approach similar to the reporting of catheter angiography and echocardiography.

CONCLUSION. The superb resolution and the isotropic image data acquisition of ECG-gated coronary CT angiography (CTA) enable evaluation of the coronary arteries and cardiac anatomy in any selected plane. The consistent use of standard terminology when reporting cardiac CT findings is part of a structured and comprehensive coronary CTA report.

ECG gating and recent improvements in MDCT technology have made possible excellent imaging of the coronary arteries [1]. The reported performance of coronary CTA using the current 64-MSCT scanners approaches that of invasive coronary angiography. Coronary CTA evaluates both the coronary artery wall and the lumen in a quick, noninvasive, relatively inexpensive, and robust way. As in conventional catheter angiography of most other vascular systems in the human body, it is clear that the extent of coronary artery disease (CAD) may be underappreciated with invasive coronary angiography, which views only the lumen of the arteries, in comparison with intravascular sonography and coronary CTA [2, 3]. Coronary CTA can both visualize and determine the extent of CAD in patients with normal traditional noninvasive tests, suggesting a role for screening if aggressive disease management can reduce the rate of cardiac events and cardiac mortality. It can also reduce the number of diagnostic cardiac catheterization procedures in symptomatic patients and patients with abnormal echocardiography and myocardial perfusion study results. With improved spatial and temporal resolution in the future, coronary CTA has the potential to quantify and monitor atherosclerotic plaque, particularly noncalcified and fatty or vulnerable plaque, providing a CT phenotype of an individual’s coronary arteries for targeted, patient-specific risk factor management and potential pharmaceutical strategies.

Although invasive coronary angiography and intravascular sonography provide direct evaluation of the coronary artery lumen and wall, respectively, other diagnostic tests provide predominantly indirect information about the presence of significant CAD based on the presence of calcifications (electron-beam CT), wall motion, or perfusion abnormalities (cardiac stress imaging). In addition, although risk factor assessment and management is the primary method for identifying and reducing cardiovascular risk, many patients presenting with acute coronary events are at a low risk for CAD on the basis of traditional criteria. It is important to note the high prevalence of atherosclerosis, even in relatively young asymptomatic individuals. For example, in one intravascular sonography study, one in six, or 17%, of asymptomatic individuals in the age range of 12–19 years had a greater than 0.5-mm intimal thickness of the coronary arteries, indicating the presence of preclinical CAD [4]. Given the prevalence of known CAD, let alone undiagnosed and asymptomatic CAD, interest continues in exploring the most cost-effective strategies to both screen for and diagnose CAD [5, 6].

Depending on the coronary CTA technique, cardiac function assessment may be possible. In retrospective ECG gating, dynamic information throughout the cardiac cycle is collected to facilitate cardiac functional assessment, both in a qualitative and a
Quantitative fashion. Typically, functional evaluation of both the left ventricle (LV) and the aortic valve is performed in our routine clinical practice. Similar principles may be extended to assess other cardiac chambers and valves [7–11]. Also, attempts have been made to perform myocardial perfusion assessment using CT [12–16]. We recommend that physicians refer to the appropriateness criteria outlined by the American College of Radiology (ACR) and the American College of Cardiology (ACC) as a guideline to indications for performing coronary CTA [17].

Awareness of the findings and standard descriptions pertaining to coronary CTA, plaque morphology, characterization, bypass graft assessment, other cardiac structures, and cardiac function may help in creating a structured cardiac CT report. In this article, we aim to provide a basic foundation for evaluating coronary CTA findings, non-coronary cardiac structures, and cardiac function assessment.

Coronary CTA of Acquired Coronary Artery Disease

Viewing Methods

The purpose of coronary CTA in the setting of suspected or known CAD is to determine the presence and severity of luminal narrowing and to evaluate lesion morphology. Distal portions of the coronary artery smaller than 2 mm in diameter cannot be evaluated because of the limits of spatial and temporal resolution of current coronary CTA data sets. Initial assessment is made using the axial source images followed by the postprocessed images. The origins of the vessels are first evaluated, followed by evaluation of each coronary artery from its respective ostium to its most distal extent. Various postprocessed images, such as short-axis views, curved reformations, long-axis extended views of the lumen, and 3D surface-shaded volume-rendered images, can be obtained to evaluate individual coronary arteries in various projections.

True short- and long-axis coronary artery images are created, respectively, as images perpendicular and parallel to the center line of the artery. The center line of the artery is automatically identified by most commercially available coronary CTA software and can be edited to reflect the true center line of the artery if it is incorrect. The window width and level are adjusted to suit reader preference. Three-dimensional volume-rendered images help to instantly review the epicardial surface anatomy of the coronary circulation; coronary artery dominance; presence of coronary stents; number and course of coronary artery bypass grafts (CABGs), their origin, and anastomosis to the run-off vessel; the presence of collateral vessels; and coronary artery anatomic variations and anomalies.

It is important to be cognizant of artifacts and pitfalls encountered on coronary CTA examinations, such as respiratory motion, cardiac pulsation artifacts, artifacts caused by beat variability, ventricular ectopic beats, blooming artifacts from densely calcified plaque, and misregistration artifacts. Many artifacts can be prevented by appropriate patient preparation and postprocessing using data sets in different phases of the R-R interval. The ECG editing capability available with some vendors is a useful tool for improving a potentially noninterpretable study to a diagnostic test.

Although purely or partly calcified plaques are easily visible on coronary CTA because of high contrast resolution, plaques that have no coronary artery calcifications—that is, noncalcified plaques—can be difficult to appreciate, particularly when they are small. The attenuation difference between the arterial wall and the surrounding perivascular fat can help in identification of subtle noncalcified plaques. Plaque-related wall thickening may also be apparent when comparing with either the opposite coronary wall or the adjacent normal arterial segment. Abrupt change in the lumen diameter due to plaques can also be appreciated when scrolling through the artery in the short-axis plane.

Coronary Artery Stenosis and Remodeling

Atherosclerosis in humans begins at birth and progresses in a nonlinear and largely unpredictable manner, with plaque disruption and thrombus formation resulting in an acute coronary syndrome. Significant obstructive CAD lesions are predominantly located in the proximal and mid coronary artery segments [18]. The prevalence of ostial involvement is low; in one series, 2.7% of 8,509 patients who underwent CABG surgery had ostial stenosis of 50% or more [19]. The severity of stenosis on coronary CTA is generally described as diameter stenosis rather than area stenosis, as shown on Figure 1 and detailed in Table 1. Arterial diameter reductions of 50%, 70%, and 90% correspond to area reductions of 75%, 90%, and 99%, respectively (Fig. 2). The critical narrowing of a lesion (A) may be estimated as the percentage of ratio between 1 minus actual (B) lumen diameter and the expected (C) lumen diameter—A% = 1 − (B / C)—as illustrated in Figure 3. Because coronary arteries progressively taper, the expected diameter of the lumen at the level of the lesion is determined as an average of the normal arterial segment proximal to it and the distal arterial lumen equidistant from the lesion. Reported ranges of 64-MDCT coronary CTA sensitivity, specificity, negative predictive value, and arterial segments that cannot be evaluated are 73–99%, 93–97%, 95–99%, and 2–12%, respectively [17]. CAD assessment using 64-MDCT scanners is significantly better and more robust than with earlier generations of MDCT scanners [20]. A systematic literature review concluded that the average sensitivity and specificity for diagnosing significant CAD (≥ 50% stenosis) were 95% and 84% with 4-MDCT, 95% and 84% with 16-MDCT, and 100% and 94% with 64-MDCT scanners [20].

Arterial remodeling was described by Glagov et al. [21] as positive or negative. Positive remodeling refers to exophytic growth of the artery wall at the site of coronary plaque, which results in unimpressive luminal narrowing even in the presence of significant CAD (Fig. 4). Negative remodeling refers to endophytic growth of plaque, which results in lumen decrease [22]. Positive remodeling is better appreciated with intravascular sonography and coronary CTA than with invasive coronary angiography. Positive remodeling may have a significant impact in stenosis estimation. The ratio between the lumen diameter (edges of the contrast column) and the artery wall-to-wall (edges of vascular adventitia) diameter may spuriously overestimate the luminal narrowing.

<table>
<thead>
<tr>
<th>Degree of Stenosis</th>
<th>Diameter Reduction (%)</th>
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<tbody>
<tr>
<td>Normal</td>
<td>None</td>
</tr>
<tr>
<td>Minimal</td>
<td>1–25</td>
</tr>
<tr>
<td>Mild</td>
<td>26–50</td>
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<tr>
<td>Moderate</td>
<td>51–70</td>
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<tr>
<td>Severe</td>
<td>71–99</td>
</tr>
<tr>
<td>Occlusion</td>
<td>100</td>
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TABLE 1: Severity of Coronary Artery Stenosis on CT Angiography
To avoid this, actual stenosis should be estimated as the ratio between the lumen diameter and the expected lumen diameter—that is, the mean lumen diameter of normal proximal and distal arteries equidistant from the lesion. Arterial remodeling assessment using coronary CTA appears to be reliable within the spatial resolution limits of the technology, and comparable to intravascular sonography [23]. Positively remodeled lesions are more prone to rupture even when they are only moderately stenotic [24]. Compensatory arterial enlargement is more common in patients with unstable than in those with stable angina; unstable angina is also associated with increased coronary artery distensibility [25].

The remodeling index \((RI)\) of a lesion may be reported as a ratio between the area (including both plaque and vessel lumen) at the site of maximal \((x)\) luminal narrowing and the mean \((y)\) area of the proximal and distal reference sites: \(RI = x / y\) (Fig. 5). Inflammation, coronary artery calcification, and medial thinning are the primary determinants of positive remodeling, which appears to be a feature of plaque instability [26].

**Plaque Characterization, Morphology, and Coronary Collateral Circulation**

Various techniques are being explored to characterize coronary plaques and their impact on clinical decision making [27]. Intravascular sonography and invasive coronary angiography have been used to differentiate coronary plaques [28], as has coronary CTA [29–32]. Plaques can most simply be described on coronary CTA as noncalcified, calcified, or mixed. Characterization of noncalcified plaques using coronary CTA seems feasible based on the attenuation values of the plaque composition for larger and therefore more proximal plaques [31, 33]. However, coronary CTA has not yet reliably and reproducibly been shown to differentiate among noncalcified plaques that are fibrotic versus fatty; greater spatial resolution will be required for this application. In patients with stable angina, intravascular sonography estimates that approximately one third have vulnerable plaques [34] that are usually noncalcified, with a focal lipid core in the coronary artery wall covered by a thin fibrous cap along the endoluminal surface. The thin fibrous cap is usually less than 65 \(\mu m\) thick and therefore not detectable on coronary CTA, making differentiation of vulnerable from nonvulnerable plaque on coronary CTA difficult [35].

In native coronary arteries, plaque rupture usually occurs in the proximal left anterior descending artery (LAD), proximal and distal right coronary artery (RCA), and anywhere along the left circumflex (LCX) artery [36]. Knowledge of lesion morphology and the status of the distal vasculature is necessary to devise the correct coronary intervention, either a percutaneous coronary interventional procedure or bypass graft surgery. Hence, it may be a good clinical practice to adequately describe lesion characteristics, such as those mentioned in Appendix 1 and Figures 4 and 6. The artery segment distal to a lesion can be described in regard to its size, degree of luminal opacification, presence or absence of disease, and collateral vessels. Regarding lesion characteristics, lesions can be classified as low-, moderate-, or high-risk for coronary intervention, according to the criteria of the American College of Cardiology (ACC)/American Heart Association (AHA) task force on lesion morphology [37], which were subsequently modified by some researchers [38]. Based on fewer lesion characteristics, simplification of the original ACC/AHA classification for predicting the success of percutaneous coronary intervention and complications of coronary lesions was proposed by the Society for Cardiovascular Angiography and Interventions, which grades lesions as 1 to 4 [39] (Fig. 7).

Based on appearances on invasive coronary angiography, 22 specific coronary collateral circulation pathways (Appendix 2) are possible [40]. Collateral circulation and the direction of blood flow with collaterals are more readily appreciated on invasive coronary angiography than on coronary CTA. Collateral circulation may mean that the circulation of one or more coronary arteries is in jeopardy. “Intercoronary” artery collateral vessel refers to vascular channels originating...
Reporting of Cardiac MDCT

Fig. 2—CT angiography of coronary artery stenosis.
A, 45-year-old woman with family history of coronary artery disease and hyperlipidemia who presented with chest pain. Long-axis reformation image of proximal left anterior descending artery shows focal noncalcified plaque (arrow) resulting in minimal luminal stenosis.
B, 65-year-old man with chronic stable angina. Short-axis reformation image shows partly calcified focal plaque (straight arrow) resulting in moderate luminal narrowing (curved arrow) of mid left circumflex artery segment.
C, 66-year-old man with chest pain and abnormal myocardial perfusion imaging. Short-axis reformation image shows partly calcified plaque in mid right coronary artery (straight arrow), resulting in severe luminal narrowing (curved arrow).
D, 75-year-old woman with chronic stable angina. Curved planar reformation image shows long-segment complete luminal occlusion (arrow) of proximal and mid right coronary artery segments. Opacification of distal right coronary artery is presumably due to blood flow through small intercoronary collateral vasculature.

Fig. 3—69-year-old man with chest pain and negative myocardial perfusion test. Visual estimation of curved planar reformation image at site of maximum stenosis (curved arrows) in left circumflex artery in relation to equidistant normal proximal (arrowheads) and distal (straight arrows) reference sites suggests mild stenosis. Semiautomated stenosis quantification tool also quantifies diameter stenosis of 25% (1 – 2.8 / 3.75 mm) and area stenosis of 45% (1 – 6.5 / 11.65 mm²).

from two coronary arteries, and “intracoronary” (bridging) collateral vessel refers to the dilated adventitial arteries that bypass an occlusion in the same coronary artery.

Coronary Artery Bypass Grafts and Stents

Usually the left internal mammary artery (LIMA) and saphenous vein grafts are used for CABG; less commonly, other vessels such as gastroepiploic or radial arteries are used [41] (Fig. 8A). Arterial grafts have better long-term patency than saphenous vein grafts. Graft type, origin, course, and anastomosis may be reported, and unusual CABG anatomy (such as T-shaped grafts) and their anastomoses may be described. CABG-related complications, such as stenosis, thrombosis, aneurysm, anastomotic stricture or dehiscence, twisting, and opacification of native coronary artery distal to the anastomosis, may be described (Figs.
Graft false aneurysms are commonly located near anastomoses, whereas true aneurysms are located in the body of the CABG [41]. False aneurysms develop because of weakening at the site of valves with saphenous vein grafts or vessel injury during surgery. Graft aneurysms are better characterized for the presence of calcification, dissection and thrombosis, mass effect, rupture, and fistulization into the adjacent organs. Abnormalities in the feeding vessel near the origin of a CABG (ascending aorta for saphenous vein grafts or subclavian arteries for LIMA grafts) may also be documented. Graft evaluation with coronary CTA has high sensitivity and specificity for stenosis and occlusion, as reported in a recent meta-analysis [42].

Coronary stents are described by location, diameter, length, and patency (Fig. 9). Stent descriptions—such as complete thrombosis; in-stent restenosis; excluded side branches; single, overlapping, or tandem stents; and contrast runoff in the distal coronary artery segment—may help to explain the pathophysiology of the stent-related abnormality. Edges of the stents can be assessed for neointimal hyperplasia. Coronary artery segments adjacent to stents may need to be evaluated for dissection and aneurysm formation. In-stent contrast attenuation values may be higher than values in the proximal and distal coronary segments because of partial volume averaging effects from high-attenuation stent struts [43]. Hence, the presence of low-attenuation areas in these stents may be due to in-stent thrombus. However, these need to be differentiated from strut-related streak artifacts. Caution should be used in evaluating stents because many variables make them difficult to evaluate, including blooming artifact related to stent struts and calcification in stents that can obscure plaques. A study reports that coronary CTA has moderate sensitivity and high negative predictive values for detecting in-stent restenosis [44].

**Noncoronary Cardiovascular Findings**

**Pericardium**

The pericardial cavity is lined by the 1- to 2-mm-thick fibrous visceral and parietal pericardium and contains a minimal amount of physiologic fluid. The CT appearances of oblique and transverse pericardial sinuses may be mistaken for mediastinal masses such as enlarged lymph nodes or bronchogenic cysts [45]. Pericardial effusions may be described for their size, for the presence or absence of septations, and for high-attenuation material that suggests hemothorax; they may also be described by the presence of gas, calcification, pericardial nodules or masses, and a pericardial drainage window or catheter. Cardiac tamponade may be suspected when there is inward bowing of the anterior wall of the right ventricle (RV) or lack of RV expansion on cine imaging.

**Cardiac Chambers**

The upper and inferior margins of the heart are called the "acute" margins and the left heart border is called the "obtuse" margin, hence the terms “acute” and “obtuse” marginal coronary artery branches. Normal LV myocardial thickness (measured at end-diastole either across the ventricular septum or the posterior LV wall) is 0.6–1 cm in men and 0.6–0.9 cm in women. LV wall thickness measurements are commonly used in routine clinical practice to detect LV hypertrophy. The ranges of mild, moderate, and severe hypertrophy are 1.1–1.3 cm, 1.4–1.6 cm, and 1.7 cm or more for men and 1–1.2 cm, 1.3–1.5 cm, and 1.6 cm or more for women, respectively [46].
On CT, the LV myocardial mass can be measured by summing the volume of muscle between the endocardial and epicardial contours; this is time-consuming and is not used in routine clinical practice. Abnormal myocardial enhancement with IV contrast administration, focal thinning, calcification, fat replacement, or aneurysm formation due to a prior myocardial infarction scar may be described. In our current clinical practice, myocardial perfusion study and viability assessment as done in cardiac MRI are not routinely performed using coronary CTA. Some reports have shown that delayed myocardial enhancement on CT correlates with MRI and nuclear medicine studies and is predictive of infarct size [12–16].

Chamber morphology may be evaluated along both the long and the short axes of the LV (Fig. 10). Horizontal long axis and vertical long axis are known respectively as four-chamber and two-chamber views. The atrioventricular valves and the LV apex are aligned in plane to form the ideal horizontal long-axis and vertical long-axis evaluation planes. The short-axis viewing plane is the true axial view of the LV, which could be further divided into basal, mid chamber, and apical short-axis views. The three-chamber view is the view in which the LA, LV, and
Lesions

Lesion A (> 85% success during PCI)
- Discrete (< 1 cm long)
- Little or no calcification
- Concentric
- < Total occlusion
- Readily accessible
- Not ostial in location
- Nonangulated segment < 45°
- No major branch involvement
- Smooth contour
- No thrombus

Lesion B (60–80% success during PCI)
- Tubular (1–2 cm long)
- Moderate to heavy calcification
- Eccentric
- Total occlusion < 3 months old
- Moderately tortuous proximal segment
- Ostial location
- Moderately angulated segment 45–90°
- Bifurcation lesion needs double guidewires
- Irregular contour
- Some thrombus

Lesion C (< 60% success during PCI)
- Diffuse (> 2 cm)
- Excessive tortuosity of proximal segment
- Extremely angulated segments > 90°
- Total occlusion > 3 months old
- Degenerated vein grafts, friable lesions
- Inability to protect major side branches

Non C lesion

Type 1 (Patent)
Type 3 (Occluded)

C lesion

Type 2 (Patent)
Type 4 (Occluded)

Fig. 7—Coronary artery lesion classifications from American College of Cardiology/American Heart Association (ACC/AHA) [37] and Society for Cardiovascular Angiography and Interventions (SCAI) [39]. PCI = percutaneous coronary intervention.

Fig. 8—Coronary artery bypass grafts (CABGs).
A, 64-year-old man with history of CABG for multivessel coronary artery disease. Volume-rendered left anterior oblique image of heart shows panoramic view of these bypass grafts. Curved arrow indicates left internal mammary artery perfusing distal left anterior descending (LAD) artery, straight arrow indicates saphenous vein graft perfusing left circumflex artery, and arrowhead points to saphenous vein graft perfusing obtuse marginal artery.
B, 55-year-old man with prior CABG who underwent coronary CT angiography to assess saphenous vein graft patency. Curved planar reformation of saphenous vein graft anastomosed to distal LAD shows occluded coronary artery stent (arrow) in distal portions of graft.
C, 58-year-old woman with prior CABG surgery. Left anterior oblique view of volume-rendered image of right heart shows saphenous vein graft to LAD (arrowhead). Saphenous vein graft perfusing distal right coronary artery (straight arrow) has focal aneurysmal dilatation (curved arrow) at its proximal anastomosis with ascending aorta.
RV are seen with both the mitral valve and the aortic valve in the viewing plane.

\textit{Atria}

The volume of the right atrium (RA) chamber is minimally larger than that of the left (LA) (75–80 mL vs 55–65 mL) (Fig. 11). At its posterosuperior aspect, the RA communicates with the superior vena cava, and at its posteroinferior margin it communicates with the inferior vena cava (IVC). The IVC opening is guarded by a semilunaris valve known as the eustachian valve. IVC size at inspiration is reported to potentially correlate with cardiac function [47, 48]. The exterior surface of the RA has a groove known as the “sulcus terminalis,” which separates the vena cavae from true RA chamber. The

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\textbf{Fig. 9—Coronary artery stents.}

A, 55-year-old man with history of coronary artery stent insertion for focal severe luminal stenosis. Short-axis reformation and long-axis reformation images of coronary CT angiography show widely patent coronary stent (curved arrows) and excluded origin of marginal branch (straight arrows). B, 67-year-old man with history of coronary stent insertion in left anterior descending artery (LAD). Curved planar reformation image of LAD shows in-stent thrombosis (arrow) resulting in mild diffuse luminal disease.

\textbf{Fig. 10—Traditional cardiac views in 50-year-old man.}

A, Three-chamber view shows right ventricle (RV), left atrium (LA), and left ventricle (LV). Note mitral (straight arrows) and aortic (AO) (curved arrow) valves in plane. B, Short-axis view of RV and LV shows anterior (arrow) and posterior (arrowhead) LV papillary muscles. C, Two-chamber (vertical long-axis) view of LA and LV shows arrows pointing to mitral valve. D, Four-chamber (horizontal long-axis) view shows all four cardiac chambers with tricuspid (curved arrows) and mitral (straight arrows) valves in plane.
corresponding ridge on the interior surface of the RA is the crista terminalis. The right atrial appendage (RAA) is a broad-based structure located anterior to and continuous with the RA. Pectinate muscles inside the RA should not be confused with filling defects such as tumor or thrombus. An arbitrary boundary between the RAA and RA is formed by the crista terminalis and sulcus terminalis. The LA is posteriorly located and receives oxygenated blood from the lungs through the pulmonary veins. Approximately 80% of the population has four pulmonary veins: a superior and an inferior pulmonary vein on both the right and the left. The right upper and middle lobe lung parenchyma drain via the right superior pulmonary veins, the left upper lobe and lingula via the left superior pulmonary veins, and the lower lobes each through their respective inferior pulmonary veins. The morphology of pulmonary veins and their ostial size are important for planning ablation procedures to treat atrial arrhythmias [49, 50]. Variability in pulmonary vein branching, ostial size, and the distance to the first tributary of each may be described in studies performed specifically for ablation mapping [49, 50]. The left atrial appendage is a long narrow chamber with a narrow base and tubular chamber configuration that contains pectinate muscles; the latter should not be mistaken for thrombus or tumor.

The interatrial septum is a thin structure of variable thickness that varies both within a patient and among patients. Along the RA surface of the septum is a 1- to 2-cm round or oval craterlike area, the fossa ovalis, where the foramen ovale was located in utero. This crater is covered by a thin portion of the interatrial septum along the LA surface. During adult life, there is normally no communication between the RA and LA. The prominent oval rim of the fossa ovalis is known as the limbus ovalis, which is deficient in its inferior rim. The remaining interatrial septum is the thick muscular portion, about 1–2 cm thick. The amount of invaginating fat in the interatrial septum may vary, and in some patients is extensive. The latter is known as lipomatous hypertrophy of the interatrial septum, which could be misdiagnosed as a cardiac mass [51].

The coronary sinus is a short venous segment that runs in the posterior aortic valve groove and opens into the RA medial to the IVC orifice, with the thebesian valve at its orifice. Most of the cardiac venous return enters the RA through the coronary sinus except venous return from the anterior part of the heart. Great, middle, and small cardiac veins can be recognized and evaluated for anatomy, variations, and abnormalities and described, particularly when examinations are performed for anatomic mapping before placement of a biventricular pacemaker. Conventional anatomic and functional classification of coronary sinuses is nicely described by Singh et al. [52], who propose a segmental classification system of the coronary venous anatomy based on their epicardial location.

**Figures**

**Fig. 11**—Atria and appendages in 74-year-old woman.

A, Horizontal long-axis view shows all four cardiac chambers. Arrow points to membranous portion of interatrial septum (fossa ovalis).

B, Curved arrow indicates sulcus terminalis and arrowhead points to crista terminalis. Thin pectinate muscles (straight arrow) in right atrial appendage should not be mistaken for thrombus.

C, Arrow denotes pectinate muscles in left atrial appendage.

**Fig. 12**—52-year-old woman with prior myocardial infarction of inferior wall who developed congestive heart failure. Cardiac CT reveals large left ventricular aneurysm (curved arrows) and significant amount of intraluminal thrombus. Straight arrow indicates anterior mitral valve leaflet, arrowhead indicates posterior leaflet.

**Ventricles**

The RV inflow tract is known as a “sinus,” and the RV outflow tract (RVOT) is known as the “infundibulum.” The RVOT–pulmonary artery inflow tract is also known as the “conus arteriosus,” from which the pulmonary artery arises. The three components of the septal portion of the RV are the inflow tract, the trabecular wall (which typifies the internal appearance of RV), and the portion of the septum that contributes to the RVOT. The RVOT is formed by three components, the conal septum, the septal band division, and the trabecular septum; the conal septum is clinically significant because it can be malpositioned in patients with congenital
disorders. Morphologic characteristics are useful in recognizing the anatomic RV in the context of congenital cardiac diseases such as transposition of the great vessels. Lateral to the conal septum, the parietal extension of the infundibular septum and the infundibular fold constitutes the crista supraventricularis. Ventricular septal defects commonly occur in the area between the sinus and the RVOT. The tricuspid valve is a trileaflet valve that separates the RV from the RA. RV papillary muscles connect the RV myocardium to the caudal aspects of the tricuspid valve via chordae tendinea.

The anterior leaflet is the largest leaflet and is notched; the posterior leaflet is smaller and scalloped. The medial leaflet is known as the “septal leaflet,” which usually attaches to the membranous and muscular portions of the interventricular septum. The moderator band is a characteristic structure in the RV apex and is a thick muscular strip that connects the base of the anterior papillary muscle and the interventricular septum. The LV cavity is somewhat narrower and longer than the RV, measuring 7.5 cm in length and 4.5 cm in width. It is divided into a large sinus portion and a small LV outflow tract (LVOT). The inflow portion has the two-leaflet mitral valve, and the LVOT has the aortic valve. Unlike the RV, the inflow and LVOT are closely related. Near the LV apex, the interventricular septum has fine trabeculations. Two papillary muscles, the anterolateral and posteromedial, are connected to the anterior and posterior mitral valve leaflets through chordae tendinea. The papillary muscles vary in size and morphology. They may appear masslike in cross section, particularly during systole when the myocardium thickens [53]. The triangular anterior leaflet is the larger leaflet; the scalloped posterior leaflet is the smaller (Figs. 10A, 10C, 10D, and 12).

Aorta, Pulmonary Arteries, and Pulmonary Veins

The aortic valve is a three-leaflet structure with three sinuses. The most common congenital abnormality of the aortic valve is an anatomically bicuspid valve [54]; there is also a rarer quadricuspid aortic valve. Partial or full fusion of two leaflets results in functional bicuspid morphology. Valve leaflet morphology, focal or diffuse leaflet calcification or thickening, vegetations, stenotic and regurgitant valve orifice areas, leaflet mobility, and the LVOT (measured just below the aortic valve) may be described in a cardiac CT report. An aortic valve stenotic orifice—narrowest area when the valve is fully open during LV systole; and the regurgitant orifice, when the valve fully closes during LV diastole. Aortic valve calcification may be quantified on unenhanced calcium scoring CT data sets—that is, shown to correlate with the transvalvular gradients and aortic valve area [55, 56]. The pulmonary valve is a trileaflet valve with right, left, and nonseptal cusps.

The thoracic aorta included in the displayed field of view may be evaluated for aneurysm and dissection, particularly for acute syndromes in the context of acute chest pain. Pulmonary artery filling defects and morphology, including hypertension, aneurysms, and stenosis, may be evaluated. It is not unusual to notice incidental pulmonary embolism on routine outpatient CT examinations, particularly in patients with malignancy [57].

Cardiac Functional Evaluation

The use of retrospective ECG gating for cardiac CT enables evaluation of wall motion at rest and the quantification of LV function without additional scanning time, radiation exposure, or contrast material. Newer prospective triggering methods allow coronary CTA to be performed with the x-ray beam on during end-diastole only at a substantially lower radiation exposure for the patient; however, because CT data are collected at end-diastole, quantitative and qualitative functional analyses cannot be performed. In routine clinical practice, functional analysis is most commonly done to evaluate the LV and aortic valve; if needed, the same principles and technique can be extrapolated for other cardiac chamber and mitral valve function assessment [7–11]. Precise evaluation of LV function is important for the diagnosis, therapy, and follow-up of patients with cardiac disease [58].

LV Function

Ventricular volumes at end-diastole and end-systole (EDV and ESV), ejection fraction, stroke volume, and cardiac output can be calculated. The endocardial borders traced on short-axis reformations selected at both end-diastole and end-systole are used to determine LV volume. The contours are automatically drawn to consistently include or exclude the papillary muscles in the LV volume and are manually adjusted as needed (Fig. 13). The most basal slice is the one just anterior to the atroventricular ring.

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**Fig. 13**—55-year-old man with suspected coronary artery disease. A and B, Short-axis reformations of left ventricle (LV) at end-diastole (A) and end-systole (B) with endocardial (red) and epicardial (green) contours. Note normal systolic symmetric wall thickening. C, LV volume curve obtained from retrospectively gated cardiac CT shows ejection fraction of 73%. EDV = end-diastolic volume, ESV = end-systolic volume, SV = stroke volume, EF = ejection fraction.
displaying myocardium in at least 50% of the perimeter, and the most apical slice is the last slice with a contrast-opacified lumen. LV volume calculation is made using Simpson’s rule: the sum of all cross-sectional areas $A$ multiplied by section thickness $S$, as follows [59]:

$$\text{LV vol} = \sum A \times S.$$ 

Ejection fraction, stroke volume, and cardiac output are derived from the ventricular volumes and are calculated as follows:

- Ejection fraction (%) = $(\text{EDV} - \text{ESV} / \text{EDV}) \times 100$,
- Stroke volume (mL) = EDV − ESV,
- Cardiac output (mL/min) = stroke volume $\times$ heart rate.

An LV volume curve and the global parameters of CT-derived LV function are depicted in Figure 13; they are generated when images of the heart are reconstructed at 10–20 partitions of the R-R interval. Several studies have compared 4-, 8-, and 16-MDCT-derived global function parameters with measurements from the current reference standard for cardiac function, MRI [60–64], as well as echocardiography [65, 66], cine-ventriculography [67–69], and SPECT [64]. CT has good agreement with MRI, slightly overestimating ESV and underestimating ejection fraction by 1–7% [59–64, 70]. This has been attributed to partial volume averaging resulting from the limited temporal resolution achieved by the current scanners. The use of faster scanners with improved temporal resolution is expected to improve agreement with MRI. Normal reference values for global LV function as used for MRI [71] are depicted in Table 2.

LV wall motion is assessed for endocardial excursion and the extent and timing of systolic LV myocardial thickening. Wall motion may be reported as normal, hypokinetic, akinetic, dyskinetic, or aneurysmal (diastolic LV deformation) [72] (Figs. 14, S15, and S16; supplemental cine images may be viewed at www.ajronline.org). Abnormal wall motion is related to loss of normal functional myocardium and insufficient myocardial perfusion and corresponds to diseased coronary artery territories. Regional function may be reported using the 17-segment model of the AHA [73], with six segments each in the basal and mid LV section, four segments in the apical section, and one segment representing the LV apex. Stress imaging, commonly used in echocardiography and MRI, is impractical with cardiac CT because of the radiation dose and the need for additional IV administration of iodinated contrast material [59]. Thus, only wall motion abnormalities
from high-grade lesions that lead to impaired perfusion even at rest or fixed abnormalities from prior myocardial infarctions with myocardial damage are usually detected.

Aortic Valve Function

With ECG-gated MDCT, aortic valve leaflet motion can be evaluated dynamically. This trileaflet valve opens during systole with effacement of the sinuses and closes during diastole with complete coaptation of the leaflets (Fig. S17). When evaluating a native aortic valve, note the number of leaflets (Figs. S18 and S19), valve stenosis (Fig. S19), regurgitation (Fig. S20; supplemental images are available at www.ajronline.org), the presence or absence of calcification, and valve vegetation. Cine images are useful to evaluate the precise architecture of the valve and the patterns of commissural fusion [74]. Incomplete coaptation of the leaflets may represent valve dysfunction, and the diastolic regurgitant aortic valve orifice area is reported to correlate well in grading known aortic incompetence as defined by transesophageal echocardiography [75].

Noncardiovascular Findings

Noncardiac findings on coronary CT can be seen in 25–61% of patients, of which 5–41% may be significant or potentially significant findings [76–81], similar to earlier reports of calcium scoring electron-beam CT examinations [78, 80, 82]. A patient with absolutely normal coronary arteries could have a potentially life-threatening finding in the thorax accounting for chest pain, such as acute pulmonary embolism, acute aortic syndrome, or a relatively benign finding such as a large hiatal hernia. Few studies have reported the rate of incidental findings of lung cancer first detected on coronary CT [76, 77]. It is therefore important to evaluate all the structures surrounding the heart and included in the acquisition. A systematic approach to analyzing the noncoronary findings is helpful; whether a field of view is reconstructed for the heart only or for the entire width of the thorax, findings in the imaged field of view may have a major impact on patient management.

Conclusion

Cardiac CT is increasingly integrated in clinical practice for elective and emergent evaluation of suspected CAD. In this article, we have reviewed our understanding of CAD, and the usefulness of coronary CTA in the diagnosis and characterization of CAD. We have illustrated accepted descriptions relevant to coronary artery disorders, cardiac anatomy, and cardiac functional assessment that may be useful to interpret cardiac CT examinations, with the aim of providing a comprehensive cardiac CT report that is concise, structured, and informative about the question posed by the nonradiology physician colleague.

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**Reporting of Cardiac MDCT**

Appendices appear on next page
### APPENDIX 1: Potential Descriptions of Characteristics of Native Coronary Artery Disease

**Lesion Characteristics**
- Califications (minimal, moderate, severe)
- Length
- Eccentricity
- Ostial involvement
- Ulceration
- Irregularity
- Tortuosity
- Vessel wall remodeling
- Intraplaque hypodensity
- Thrombus or embolus
- Acute or chronic occlusion
- Bifurcating lesions
- Tandem lesions
- Coronary artery aneurysm

### APPENDIX 2: Coronary Collateral Circulation Pathways

#### Right Coronary Artery Occlusion
- Left anterior descending (LAD) artery to posterior descending artery (PDA) via ventricular septal branches
- Distal left circumflex (LCX) artery to distal right coronary artery (RCA)
- Obtuse marginal (OM) to posterior left ventricular (LV) branch of RCA
- Proximal acute marginal (AM) artery or conus branch to distal AM
- Kugel’s artery: proximal RCA or left main (LM) coronary artery to atrioventricular (AV) nodal branch
- Distal LAD to PDA
- Distal LCX or its left atrial (LA) branch to AV node branch
- AM to PDA
- Sinoatrial node branch to LA circumflex branch, then to distal RCA
- Right ventricular (RV) branch of LAD to AM

#### Left Anterior Descending Artery Occlusion
- AM to LAD
- Proximal ventricular septal branch of LAD to distal septal branch
- OM to LAD
- Conus branch of the RCA to LAD
- Diagonal branch to distal LAD
- PDA to LAD via apex
- PDA to LAD via the septal branches

#### Left Circumflex Artery Occlusion
- LA LCX branch to distal LCX
- Proximal OM to distal OM
- Diagonal artery to OM
- Distal RCA to distal LCX
- Posterior LV branch of the RCA to OM

Adapted from [40].

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- A data supplement for this article can be viewed in the online version of the article at: www.ajronline.org.
- This article is available for CME credit. See www.arrs.org for more information.
- The reader’s attention is directed to part 1 of this article, titled “Anatomy and Technology for the Interpretation and Reporting of Cardiac MDCT: Part 1, Structured Report, Coronary Calcium Screening, and Coronary Artery Anatomy,” which appears on page 574.