APPENDIX A:

CLINICAL GUIDELINES

Assessment of Coronary Artery Disease by Cardiac Computed Tomography: A Scientific Statement From the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology

Matthew J. Budoff, Stephan Achenbach, Roger S. Blumenthal, J. Jeffrey Carr, Jonathan G. Goldin, Philip Greenland, Alan D. Guerci, Joao A.C. Lima, Daniel J. Rader, Geoffrey D. Rubin, Leslee J. Shaw and Susan E. Wiegers

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/cgi/content/full/114/16/1761

Subscriptions: Information about subscribing to Circulation is online at
http://circ.ahajournals.org/subscriptions/

Permissions: Permissions & Rights Desk, Lippincott Williams & Wilkins, a division of Wolters Kluwer Health, 351 West Camden Street, Baltimore, MD 21202-2436. Phone: 410-528-4050. Fax: 410-528-8550. E-mail:
journalpermissions@lww.com

Reprints: Information about reprints can be found online at
http://www.lww.com/reprints

AHA Scientific Statement

Assessment of Coronary Artery Disease by Cardiac Computed Tomography

A Scientific Statement From the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology

Matthew J. Budoff, MD, FAHA; Stephan Achenbach, MD; Roger S. Blumenthal, MD, FAHA;
J. Jeffrey Carr, MD, MSCE; Jonathan G. Goldin, MD, PhD; Philip Greenland, MD, FAHA;
Alan D. Guerci, MD; Joao A.C. Lima, MD, FAHA; Daniel J. Rader, MD, FAHA;
Geoffrey D. Rubin, MD; Leslee J. Shaw, PhD; Susan E. Wiegers, MD

TABLE OF CONTENTS

Executive Summary

This scientific statement reviews the scientific data for cardiac computed tomography (CT) related to imaging of coronary artery disease (CAD) and atherosclerosis. Cardiac CT is a CT imaging technique that accounts for cardiac motion, typically through the use of ECG gating. The utility

(*Circulation.* **2006;114:1761-1791.**)

© 2006 American Heart Association, Inc.

Circulation is available at http://www.circulationaha.org   **DOI: 10.1161/CIRCULATIONAHA.106.178458**

and limitations of generations of cardiac CT systems are reviewed in this statement with emphasis on CT measurement of CAD and coronary artery calcified plaque (CACP) and noncalcified plaque. Successive generations of CT technology have been applied to cardiac imaging beginning in the early 1980s with conventional CT, electron beam CT (EBCT) in 1987, and multidetector CT (MDCT) in 1999. Compared with other imaging modalities, cardiac CT has undergone an accelerated progression in imaging capabilities over the past decade, and this is expected to continue for the foreseeable future. As a result, the diagnostic capabilities at times have preceded the critical evaluation of clinical application. In this statement, the American Heart Association (AHA) Writing Group evaluates the available data for the application of cardiac CT for CAD.

Cardiac CT uses natural contrast within subjects (utilizing the different brightness of fat, tissue, contrast, and air). Noncontrast CT is a low-radiation exposure technique and, even without premedication or intravenous contrast, can determine the presence or absence of CACP in <10 minutes. The amount of CACP can be measured to provide a reasonable estimate of total coronary atheroma including calcified and noncalcified plaque. The data supporting detection of CACP as a measure of CAD are extensive. Imaging applications that detect CACP include conventional chest radiographs, cinefluoroscopy, conventional and helical CT, EBCT, and MDCT.

The majority of published studies have reported that the total amount of coronary calcium (usually expressed as the "Agatston score") predicts coronary disease events beyond standard risk factors. Although some registries used self-reported risk factor data, data from EBCT reports using measured risk factors demonstrate incremental risk stratification beyond the Framingham Risk Score (FRS). These studies demonstrate that CACP is both independent of and incremental with respect to traditional risk factors in the prediction of cardiac events. Data from Greenland et al[1] demonstrated that intermediate-risk patients with an elevated coronary artery calcium (CAC) score (intermediate FRS and CAC score >300) had an annual hard event rate of 2.8%, or a 10-year rate of 28%, and thus would be considered high risk. The best estimates of the relative risk (RR) from this study indicated that a CAC score >300 had a hazard ratio (HR) of about 4 compared with a score of 0. This would mean that the estimated risk in the intermediate-risk patient with a CAC score of 0 might be reduced by at least 2-fold while the risk of a person with a CAC score of 300+ would be increased by about 2-fold. Thus, the person with a high CAC score and intermediate FRS is now reclassified as high risk. CT information may then be used to guide primary prevention strategies, especially among individuals within the intermediate-risk category, in whom, as suggested by the AHA Prevention Conference V,[2] clinical decision-making is most uncertain. Individuals determined to be at intermediate risk of a cardiovascular disease (CVD) event on the basis of traditional risk factors may benefit from further characterization of their risk through measurement of their atherosclerotic burden with cardiac CT. This AHA Writing Group agrees with the statement from the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III): "In persons with multiple risk factors, high coronary calcium scores (eg, >75th percentile for age and sex) denote advanced coronary atherosclerosis and provide a rationale for intensified LDL-lowering therapy."[3] Guidelines and expert consensus documents[4] have extended the recommendation for use of coronary calcium measurements in clinically selected patients at intermediate risk for CAD (eg, those with a 10% to 20% 10-year FRS) to refining clinical risk predictions and to assessing whether more aggressive target values for lipid-lowering therapies are indicated for select patients.[5] Asymptomatic persons should be assessed for their cardiovascular risk with such tools as the FRS. Individuals found to be at low risk (<10% 10-year risk) or at high risk (>20% 10-year risk) do not benefit from coronary calcium assessment **(Class III, Level of Evidence: B)**. In clinically selected, intermediate-risk patients, it may be reasonable to measure the atherosclerosis burden using EBCT or MDCT to refine clinical risk prediction and to select patients for more aggressive target values for lipid-lowering therapies **(Class IIb, Level of Evidence: B).**

When cardiac CT is used for CACP assessment, the AHA Writing Group strongly recommends a low-dose technique using prospective ECG gating. Although alternative techniques may provide improved resolution or increased precision in measurement, data to support an enhanced predictive ability given the higher radiation exposure are limited. A minimum CT-system configuration of EBCT C150 or more up to date or MDCT 4 channel with 0.5-second gantry rotation or faster is recommended. Although virtually all of the prognostic and epidemiological data derived for CACP have been performed with EBCT, several large prospective trials have documented that cardiac CT (both MDCT and EBCT) measurements can be similarly applied across multiple centers with equally high levels of patient satisfaction and acceptance.

The utility of CACP in symptomatic patients has been widely studied and has been discussed in depth in a previous ACC/AHA statement,[4] as well as in the AHA Cardiac Imaging Committee scientific statement "The Role of Cardiac Imaging in the Clinical Evaluation of Women With Known or Suspected Coronary Artery Disease."[5] The test has been shown to have a predictive accuracy equivalent to alternative methods for diagnosing CAD. These studies may have been subject to referral bias, as a positive test may have been the rationale for subjecting the patient to the invasive angiogram. More comparison work between modalities is clearly needed. A positive cardiac CT examination in which any CACP is identified is nearly 100% specific for atheromatous coronary plaque. CACP can develop early in the course of subclinical atherosclerosis and can be identified histologically after fatty streak formation. CACP is present in the intima of both obstructive and nonobstructive lesions, and thus, the presence of calcified plaque on cardiac CT is not specific to an obstructive lesion. Studies using intracoronary ultrasound have documented a strong association between patterns of CACP and culprit lesions in the setting of acute coronary syndromes.

Cardiac CT studies correlating calcified plaque using EBCT technology and various methods of coronary angiography in more than 7600 symptomatic patients demonstrate negative predictive values of 96% to 100%, providing phy-

sicians with a high level of confidence that an individual without CACP (total calcium score=0) does not have obstructive angiographic CAD. The presence of CACP is extremely sensitive, albeit with reduced specificity, for diagnosing obstructive CAD (95% to 99%) in patients >40 years of age. A recent study of 1195 patients who underwent CACP measurement with EBCT and myocardial perfusion single photon emission CT (SPECT) assessment demonstrated that CACP was often present in the absence of myocardial perfusion scintigraphy (MPS) abnormalities (normal nuclear test) and that <2% of all patients with CACP <100 had positive MPS studies.[6] This is supported by other published reports and is synthesized in a recent appropriateness criteria statement from the American Society of Nuclear Cardiology and the American College of Cardiology.[7,8] CACP measured by cardiac CT has a high sensitivity and negative predictive power for obstructive CAD but markedly limited specificity. Because calcified plaque may be present in nonobstructive lesions, the presence of CACP in asymptomatic persons does not provide a rationale for revascularization but rather for risk factor modification and possible further functional assessment. Clinicians must understand that a positive calcium scan indicates atherosclerosis but most often no significant stenosis. With exceptions, high-risk calcium scores (such as an Agatston score ≥400) are associated with an increased frequency of perfusion ischemia and obstructive CAD. The absence of coronary calcium is most often associated with a normal nuclear test and no obstructive disease on angiography. Coronary calcium assessment may be reasonable for the assessment of symptomatic patients, especially in the setting of equivocal treadmill or functional testing **(Class IIb, Level of Evidence: B).** There are other situations when CAC assessment might be reasonable. CACP measurement may be considered in the symptomatic patient to determine the cause of cardiomyopathy **(Class IIb, Level of Evidence: B)**. Also, patients with chest pain with equivocal or normal ECGs and negative cardiac enzyme studies may be considered for CAC assessment **(Class IIb, Level of Evidence: B)**.

Coronary calcium assessment for diagnosis of atherosclerosis and obstructive disease and for risk stratification for future cardiac events has undergone significant validation over the past 20 years. CT angiography is a noninvasive technique, performed by either EBCT or MDCT, to evaluate the lumen and wall of the coronary artery. Especially in the context of ruling out stenosis in patients with low to intermediate pretest likelihood of disease, CT coronary angiography may develop into a clinically useful tool. CT coronary angiography is reasonable for the assessment of obstructive disease in symptomatic patients **(Class IIa, Level of Evidence: B).** Several small studies have assessed the value of EBCT and MDCT for detecting restenosis after stent placement. At this time, however, imaging of patients to follow up stent placement cannot be recommended **(Class III, Level of Evidence: C).**

Where MDCT is used for CT angiography, the AHA Writing Group currently recommends a minimum of 16-slice capability, submillimeter collimation, and 0.42-second gantry rotation with retrospective ECG gating. If EBCT is used, 1.5-mm slice thickness should be used. A limitation of EBCT relative to MDCT is its lower power, with EBCT limited to 63 or 100 milliamperes/second (mAs), depending on scanner generation, which becomes important in larger patients because image quality can be affected by noise. Another advantage of MDCT is thinner slice imaging, with section thickness as small as 0.5 mm, whereas EBCT is limited to 1.5 mm. An advantage of EBCT, however, is the lower radiation dose associated with this procedure (1.1 to 1.5 mSv), compared with MDCT angiography (5 to 13 mSv).[9,10] The use of both CT modalities to evaluate noncalcified plaque (NCP) is promising but premature. There are limited data on variability but none on the prognostic implications of CT angiography for NCP assessment or on the utility of these measures to track atherosclerosis or stenosis over time; therefore, their use for these purposes is not recommended **(Class III, Level of Evidence: C).**

CT technology is evolving rapidly, and these radiation dose estimates are likely to decrease with modification of the hardware and scanning protocols. The clinical relevance of the radiation dose that is administered with cardiac CT is unknown. However, higher radiation doses in general are associated with a small but defined increase in cancer risk later in life. The AHA Writing Group reviewing the available literature endorses the use of a prospective ECG trigger for measurement of CACP with a slice collimation of 2.5 to 3 mm for clinical practice. EBCT systems have an effective dose of 0.7 to 1 mSv (male) and 0.9 to 1.3 mSv (female), and MDCT systems have an effective dose of 1 to 1.5 mSv (male) and 1.1 to 1.9 mSv (female). Higher radiation exposures with retrospective gating for CACP assessment preclude its use for screening. Similarly, for CT angiography, the higher radiation doses (up to 1.5 mSv for EBCT and up to 13 mSv for MDCT) prohibit the use of this test as a screening tool for asymptomatic patients. CT coronary angiography is not recommended in asymptomatic persons for the assessment of occult CAD **(Class III, Level of Evidence: C)**.

The role of cardiac CT in measuring clinically or prognostically meaningful changes in calcified plaque over time and its correlation with other measures of coronary heart disease (CHD) is currently an area of intense investigation. Reductions in the test-to-test variability and improvements in the interreader reliability of the calcium score may allow for serial assessment of coronary calcium scores; however, more studies are required. It is difficult to justify the incremental population exposure to radiation and the cost associated with a repeat CT test to assess "change," until it is better understood what therapies may be of benefit and how clinicians should utilize this data in clinical practice. There is conflicting evidence as to whether vigorous cholesterol-lowering therapy with statins retards the rate of progression of CACP. The AHA Writing Group concluded that this potential use of cardiac CT will require additional validation before any recommendation. Serial imaging for assessment of progression of coronary calcification is not indicated at this time **(Class III, Level of Evidence: C)**.

Cardiac CT technology is rapidly evolving. On the basis of the substantial validation data, EBCT remains the reference standard for CACP measurement.[11] MDCT-64 is the current standard for coronary CT angiography and NCP characterization based on publications to date.[12] The trend for improved image

quality with cardiac CT is consistent. It is critical that the cardiac imaging scientific community continue to integrate evolving technological advances with best clinical practices for treatment and prevention of CVD.[7,13]

An area of ongoing clinical research is the application of hybrid positron emission tomography CT (PET-CT) and SPECT-CT scanners that are currently available. This research will allow for the acquisition of metabolic and/or perfusion information as well as anatomic data, including angiographic data and data on coronary calcification. The incremental benefit of hybrid imaging strategies will need to be demonstrated before clinical implementation, as radiation exposure may be significant with dual nuclear/CT imaging. At this time, there are no data supporting the use of hybrid scanning to assess cardiovascular risk or presence of obstructive disease (**Class III, Level of Evidence: C**).

In summary, cardiac CT has been demonstrated to provide quantitative measures of CACP and NCP. CACP, as determined by cardiac CT, documents the presence of coronary atherosclerosis, identifies individuals at elevated risk for myocardial infarction (MI) and CVD death, and adds significant predictive ability to the Framingham Score (an index of traditional CVD risk factors). Data suggest that cardiac CT may improve risk prediction, especially in individuals determined to be at intermediate risk according to the NCEP ATP III criteria and for whom decisions concerning prevention strategies may be altered based on the test results. The use of cardiac CT angiography for noninvasive assessment of lumen stenosis in symptomatic individuals has the potential to significantly alter the management of CAD and current diagnostic testing patterns. The assessment of progression of CACP and the detection of nonobstructive NCP by cardiac CT angiography warrant further investigation.

Introduction

The AHA has issued 2 prior statements on CAC scanning; one in 1996[14] and a second (in conjunction with the American College of Cardiology [ACC]) in 2000 specifically related to EBCT.[4] The AHA also sponsored the Prevention V Conference, which focused on the identification of the asymptomatic high-risk patient and discussed the potential role of CAC scanning.[2] In light of a rapidly evolving literature since the last ACC/AHA expert consensus statement (2000), the current statement will focus on new data available on using EBCT and MDCT to identify patients with coronary atherosclerosis defined by quantification of coronary artery calcification. EBCT is an especially fast form of x-ray imaging technology that can detect and measure calcium deposits in the coronary arteries.[5] The amount of calcium detected by EBCT is related to the amount of underlying coronary atherosclerosis. During the past decade, there has been a progressive increase in the clinical use of both EBCT and MDCT scanners to identify and quantify the amount of calcified plaque in the coronary arteries. This approach has generated much interest and scrutiny for several reasons. Although coronary calcification can be quantified and calcium scores can be related to the extent and severity of atherosclerotic disease and improving CHD risk prediction,

misuse or abuse of these methods as a broad-based "screening" tool has created considerable controversy.

Recently, CT scanners with subsecond image acquisition and MDCT (also referred to as multirow or multislice) capability have been studied and proposed as an alternative approach to EBCT for detecting coronary calcification owing to the greater availability of such CT scanners. This scientific statement will compare MDCT and EBCT and serve as a clinical update for the use of CACP in clinical decision-making regarding evaluations for CHD in the asymptomatic individual. Current evidence regarding noninvasive angiography using CT, as well as the future role of these techniques in monitoring atherosclerosis over time and in detecting NCP, will be reviewed.

1. Coronary Artery Calcification and Epidemiology of Coronary Calcium

Arterial calcium development is intimately associated with vascular injury and atherosclerotic plaque. CACP is an active process and can be seen at all stages of atherosclerotic plaque development.[15–17] The long-held notion of so-called "degenerative" calcification of the coronary arteries with aging is incorrect. Since Faber[18] noted in 1912 that Mönckeberg's calcific medial sclerosis did not occur in the coronary arteries, atherosclerosis is the only vascular disease known to be associated with coronary calcification.[4,11,14,19,20] Thus, CACP in the absence of luminal stenosis is not a "false-positive" result but rather evidence of coronary atherosclerosis.[20]

Coronary calcification is nearly ubiquitous in patients with documented CAD[21–23] and is strongly related to age, increasing dramatically after age 50 in men and after age 60 in women (Tables 1 and 2).[24,25] However, coronary plaque and its associated coronary calcification may have only a weak correlation with the extent of histopathologic stenosis.[26,27] The degree of encroachment on the vessel lumen by the atherosclerotic plaque is largely determined by individual variations in coronary artery remodeling. However, the presence of CACP is associated with atherosclerotic plaque size.[26]

Rumberger and colleagues[28,29] examined 13 autopsied hearts and compared measures of CACP using EBCT as compared with direct histological plaque areas and percent luminal stenosis. These studies determined that the total area of CACP quantified by EBCT is linearly and highly correlated ($r=0.90$) with the total area of histological coronary artery plaque. Although the total atherosclerotic plaque burden was tracked by the total calcium burden, not all plaques were found to be calcified, and the total calcium area was approximately 20% of the total atherosclerotic plaque area. Baumgart et al[30] and Schmermund et al[31] compared direct intracoronary ultrasound measures during angiography with EBCT scanning and confirmed a direct association, in vivo, of CACP score with localization and extent of atherosclerotic plaques.

The prevalence of CACP mirrors the prevalence of coronary atherosclerosis in both men and women.[32] The data show the following: (1) the prevalence of CACP increases from only a small percentage in the second decade of life to nearly 100% by the eighth decade in men and women; (2) the prevalence of CACP in women is similar to that in men who

TABLE 1. Descriptive Characteristics of the Total Electron Beam Tomographic CAC Scores in Asymptomatic Men and Women

Age, y	Men				Women			
		Total CAC Score				Total CAC Score		
	n	Mean	SD	Median	n	Mean	SD	Median
<40	3504	12	70	0.5	641	2	14	0
40–44	4238	27	120	1	1024	8	97	0
45–49	4940	57	175	3	1634	18	186	0
50–54	4825	121	305	16	2184	29	135	0.5
55–59	3472	203	411	49	1835	54	189	1
60–64	2288	350	972	113	1334	78	250	3
65–69	1209	464	731	180	731	147	338	24
70–74	540	665	921	309	436	225	515	55
>74	235	836	1053	473	174	258	507	75

Adapted from data presented in Hoff et al.[24]

are a decade younger; (3) the gender difference in prevalence with age is eliminated by approximately age 65 to 70, when the prevalence of coronary calcium in women is similar to that in men of the same age. The prevalence of CACP increases with age, paralleling the increased prevalence of coronary atherosclerosis with advancing age.

1.1. Calcium Detection Methods
This section will discuss methods related to CACP identification.

1.1.1. EBCT Methods
EBCT is a tomographic imaging device developed nearly 20 years ago specifically for cardiac imaging. Although the technique can quantify ventricular anatomy and function[33] as well as myocardial perfusion,[34] it is currently best known for defining and measuring CACP. Over the past decade, there have been more than 1000 articles published regarding EBCT and coronary artery imaging.

EBCT (also referred to as "EBT" and "Ultrafast-CT," General Electric, South San Francisco, Calif) uses unique technology enabling ultrafast scan acquisition times currently

of 50 ms, 100 ms, and multiples of 100 ms (up to 1.5 seconds) per slice (Table 3). There have been 3 iterations of EBCT systems since their clinical introduction in the early 1980s. The core imaging methods have remained unchanged, but there have been improvements in image acquisition; in data storage, manipulation, management, and display; and in spatial resolution. The original C-100 scanner was replaced in 1993 by the C-150, which was replaced by the C-300 in 2000. The current EBCT scanner, the "e-speed" (GE/Imatron, South San Francisco, Calif) was introduced in 2003. The e-speed is a multislice scanner and currently can perform a heart or body scan in half the total examination time required by the C-150 and C-300 scanners. In addition to the standard 50-ms and 100-ms scan modes common to all EBCT scanners, the e-speed is capable of high-resolution imaging speeds as fast as 50 ms. This very short acquisition time leads to fewer motion artifacts and improved contrast-to-noise ratios.[35]

EBCT uses a stationary multisource/split-detector combination coupled to a rotating electron beam and produces serial, contiguous, thin-section tomographic scans in syn-

TABLE 2. Electron Beam Tomographic CAC Score Percentiles for Men and Women Within Each Age Stratum

	Age, y								
	<40	40–44	45–49	50–54	55–59	60–64	65–69	70–74	>74
Men (25 251)	3504	4238	4940	4825	3472	2288	1209	540	235
25th Percentile	0	0	0	1	4	13	32	64	166
50th Percentile	1	1	3	15	48	113	180	310	473
75th Percentile	3	9	36	103	215	410	566	892	1071
90th Percentile	14	59	154	332	554	994	1299	1774	1982
Women (9995)	641	1024	1634	2184	1835	1334	731	438	174
25th Percentile	0	0	0	0	0	0	1	3	9
50th Percentile	0	0	0	0	1	3	24	62	75
75th Percentile	1	1	2	5	23	57	145	210	241
90th Percentile	3	4	22	65	121	193	410	631	709

Adapted from data presented in Hoff et al.[24]

TABLE 3. Basic Description of CT System Components

	EBCT	MDCT
Electron source (cathode)	Electron gun	Tungsten filament
Gantry	Fixed: Electron beam rapidly sweeps across tungsten rings	Rotates: Tube and opposing detectors rotate within gantry
Image reconstruction	Partial scan/filtered back-projection Sharp kernel	Partial scan/filtered back-projection Standard kernel
Beam current, mA	Fixed	User selectable
Exposure time for coronary calcium	50 or 100 ms (true prospective)	≥220 ms Dependent on gantry rotation speed and postprocessing
Gating for CT angiography	Prospective trigger	Retrospective gating
Exposure, mAs	Fixed mA × exposure time	User-selectable mA × exposure time
Heart rate limitations*	<110 bpm	<65 bpm
Best z-axis resolution	1.5 mm	0.5 mm

*Heart rate limitations based on the prevalence of studies with significant coronary motion.

chrony with the heart cycle. EBCT is distinguished by its use of a scanning electron beam rather than the traditional x-ray tube and mechanical rotation device used in current "spiral," single, and multiple-detector scanners. The electron beam is steered by an electromagnetic deflection system that sweeps it across the distant anode, a series of 4 fixed tungsten "target" rings. A stationary, single-level or dual-level arc of detectors lies in apposition to the tungsten target rings. In contrast, MDCT physically moves the x-ray tube in a circle about the patient; with EBCT, only the electron beam is moved.

Standardized methods for imaging, identification, and quantification of CAC using EBCT have been established.[4,36] The scanner is operated in the high-resolution, single-slice mode with continuous, nonoverlapping slices of 3-mm thickness and an acquisition time of 100 ms/tomogram.[37] Electrocardiographic triggering is done during end-systole or early diastole at a time determined from the continuous ECG tracing done during scanning.

Historically, the most common trigger time used is 80% of the R-R interval. However, this trigger occurs on or near the P wave during atrial systole, and the least cardiac motion among all heart rates occurs at 40% to 60% of the R-R interval.[38] Therefore, it has been demonstrated that the protocol of triggering at 80% of the R-R interval is not optimal for imaging of the coronary segments near the right or left atrium. Mao et al[39] compared 40% and 80% trigger delay (imaging during early compared with late diastole) and obtained an interscan variability of 11.5% versus 17.4%, respectively. For a more complete discussion on gating, see section 1.5.

1.1.2. MDCT Methods

The current generation of MDCT systems is capable of acquiring 4 to 64 sections of the heart simultaneously with ECG gating in either a prospective or retrospective mode. MDCT differs from single detector–row helical or spiral CT systems principally by the design of the detector arrays and data acquisition systems, which allows the detector arrays to be configured electronically to acquire multiple adjacent sections simultaneously (Table 3). In the current 16-row MDCT systems, 16 sections can be acquired at either 0.5- to 0.75-mm or 1- to 1.5-mm section widths, or 8 sections 2.5-mm thick.

In MDCT systems, like the preceding generation of single detector–row helical scanners, the x-ray photons are generated within a specialized x-ray tube mounted on a rotating gantry. The patient is centered within the bore of the gantry such that the array of detectors is positioned to record incident photons after they have traversed the patient. Within the x-ray tube, a tungsten filament allows the tube current to be increased (mA), which proportionately increases the number of x-ray photons for producing an image. This is a design difference with current generation EBCT systems, which use a fixed tube current.

MDCT systems have 2 principal modes of scanning, which depend on whether the patient on the CT couch is advanced in a step-wise fashion (axial, sequential, or conventional mode) or continuously moved at a fixed speed relative to the gantry rotation (helical or spiral mode). The axial mode is analogous to EBCT in using prospective ECG triggering at predetermined offset from the ECG-detected R wave and is the current mode for measuring coronary calcium at most centers using MDCT.

When prospective gating is performed, the temporal resolution of a helical or MDCT system is proportional to the gantry speed, which determines the time to complete one 360° rotation. To reconstruct each slice, data from a minimum of 180° plus the angle of the fan beam are required, typically approximately 220° of the total 360° rotation. Unless data from several consecutive heartbeats are combined, the temporal resolution is 257 ms for a 50-cm display FOV (field of view) when using a 16-row system with 0.42-second rotation. The newest 64-slice scanners now have rotation gantry speeds up to 330 ms.

1.2. Coronary Artery Calcified Plaque

Calcified plaque or calcified atheroma are the terms used in the AHA consensus paper on the definition of the advanced lesions of atherosclerosis (ie, AHA IVb lesion)—calcified plaque is a subcomponent of atheroma, not a surrogate measure.[40] CACP, as measured on cardiac CT, is defined as a hyperattenuating lesion above a threshold of 130

"Hounsfield Units" (HU) with an area ≥ 3 adjacent pixels (at least 1 mm^2).

There are currently 2 CT calcium scoring systems widely used: the original Agatston method and the "volume" score method. The Agatston score method involves multiplication of the calcium area by a number related to CT density and, in the presence of partial volume artifacts, can be variable. Also, the Agatston system was designed and is properly used only when the slice thickness of the scan is 3 mm. A calcium score is reported for a given coronary artery and for the entire coronary system; however, most research studies have reported data related to the summed or total "score" for the entire epicardial coronary system.

The Agatston scoring[37] scale is rule based: Calculate an area for all pixels above a threshold of 130 HU, do so every 3 mm (the slice thickness and spacing used by Agatston et al), and multiply it by a density factor. Partial volume effects lead to higher peak values for small lesions (but not for large ones). If the change in peak value happens to be such that it changes the density factor, then it can, theoretically, change the score by a factor of 4. The volume method of Callister et al[41] somewhat resolves the issue of slice thickness and spacing by computing a volume above threshold. The volume score is much less dependent on minor changes in slice thickness.

Current EBCT systems are now able to perform scanning at 1.5 mm, and the latest MDCT systems can provide slice thicknesses that are <1 mm. Use of thinner slices leads to higher radiation doses. In the future, a more universal scoring system may be possible that would be machine independent but, at present, data derived from MDCT should be compared with caution with those derived from EBCT. While the portability of the volume method is affected by the same issues that affect the Agatston method (slice thickness, calcium content), most studies demonstrate improved interscan reproducibility using volumetric scores for both MDCT and EBCT.

The calcium mass score has recently been reported. Basically, the mass score consists of integration of the signal for pixels above a given threshold. For a well-calibrated CT scanner, in the absence of noise, this integration (scaled by pixel volume) will give the total mineral content independent of slice thickness and spatial resolution. Although theoretically better for portability between scanners, this score has not yet undergone sufficient validation (autopsy, histology, outcomes, progression, or angiographic comparison), so its use clinically is premature.[42,43]

The retention of the Agatston score has been predicated on the availability of databases for these scores, which include the availability of outcome data so clinicians understand the significance of a certain score. Volume scores are similar, while mass scores tend to be much lower values for a given patient. Adoption of newer scoring methods will depend on the availability of similar risk stratification and outcome data. Data published by Rumberger et al[44] showed that the Agatston, volume, and mass scores, when applied properly, can provide similar characterization.

1.3. Speed/Temporal Resolution

Cardiac CT is dependent on having a high temporal resolution to minimize coronary motion–related imaging artifacts. Coupling rapid image acquisition with ECG gating makes it possible to acquire images in specific phases of the cardiac cycle. Studies have indicated that temporal resolutions of 19 ms would be needed to suppress all pulmonary and cardiac motion throughout the complete cardiac cycle.[45] Current-generation cardiac CT systems can create individual images at 50 to 100 ms (EBCT) and 83 to 210 ms (MDCT), a level of resolution that cannot totally eliminate coronary artery motion in all individuals.

Motion artifacts are especially prominent in the mid right coronary artery, where the ballistic movement of the vessel may be as much as 5 to 6 times its diameter during the twisting and torsion of the heart during the cardiac cycle. Blurring of cardiac structures secondary to coronary motion increases in systems with slower acquisition speeds. It should be noted that utilizing more detectors (ie, 4 versus 8 versus 16 versus 64 detector/channel systems) does not improve the temporal resolution of the images (the rotation speed of the scanners does not change) but reduces scan time (ie, breath-hold time) and section misregistration. Generally, the higher x-ray flux (mAs = tube current $\times$ scan time) and greater number and efficiency of x-ray detectors available with MDCT devices leads to images with better signal-to-noise ratio and higher spatial resolution when compared with current EBCT scanners.

1.4. Studies Comparing EBCT and MDCT for Calcium Scoring

Several studies comparing these modalities have been published. Becker et al[46] studied 100 patients comparing MDCT with EBCT and reported a variability of 32% between the 2 modalities. Knez et al[47] studied the diagnostic accuracy of MDCT compared with EBCT in 99 symptomatic male patients (60 ± 10 years). The mean variability between the MDCT- and EBCT-derived scores was 17%. The findings of extensive calcification and a good correlation over a large range of values do not fully address the need to measure CACP scores accurately and reproducibly in a given individual. These high correlations may not apply as well to a younger, "asymptomatic" population with generally much lower scores.[48]

Carr et al[49] found agreement could be further improved by calibration of the Agatston score to an external standard. It should be emphasized that the clinical value for CAC determination is to facilitate individual risk assessment, and thus scoring for a given individual should be as accurate as possible. In epidemiologic studies of CACP in broad population groups, measures by MDCT and EBCT may well provide important insight into the atherosclerotic process, a hypothesis currently under investigation in large, population-based studies (Multi-Ethnic Study of Atherosclerosis [MESA][50] and the Heinz Nixdorf RECALL study[51]).

1.5. Reproducibility and Validity of Calcium Scoring

A potential of these technologies is to estimate atherosclerosis burden and to track changes over time in order to assess

efficacy of therapy.[52] This ability to assess progression is dependent on the reproducibility of the technologies. With EBCT, the mean interscan variability, with improved methodology (early diastolic or end-systolic triggering) and hardware improvements available since 1997, has been shown to be approximately 15%, with interreader variability approximately 3% and intrareader variability <1%.[39,53–58] Achenbach demonstrated the median variability to be 5.7% using EBCT.[59]

The interscan variability in several early studies using noncardiac gated MDCT (dual slice) scanners was 32% to 43%.[60,61] The literature clearly supports the use of cardiac gating to improve the measurement of CACP. A study of 75 persons using 4-slice MDCT demonstrated a mean variability of 25% for overlapping images with volume scoring, as compared with 46% for Agatston scoring without overlap.[62] A study of 537 patients undergoing 2 studies on 4-slice MDCT with cardiac gating demonstrated a mean variability of 36% for volume scoring and 43% for Agatston scoring.[63] Other small studies demonstrated variabilities of 20% to 37% for Agatston scoring and 14% to 33% for volume scoring.[64–66]

The National Institutes of Health/National Heart, Lung, and Blood Institute MESA is a population-based study in which 6814 men and women 45 to 84 years of age and free of clinically apparent CVD were recruited from portions of 6 US communities. Cardiac CT (EBCT-C150 and MDCT-4) examinations for measuring CACP were performed during the baseline examination between July 2000 and August 2002 using a standardized protocol.[67] Dual scans were obtained in 3551 MESA participants on an EBCT-C150 and in 3190 participants on an MDCT-4-channel system to evaluate reproducibility of the CT systems for measuring CACP. Both systems were highly concordant on the paired scan series (96% EBCT and 96% MDCT) for the presence or absence of calcified plaque.[68] Chance corrected agreement for both technologies was high with an identical kappa statistic of 0.92. When the mean absolute rescan differences were compared, adjusted for body mass index and extent of CACP, no significant difference was seen between EBCT and MDCT-4 with absolute Agatston unit values (95% confidence intervals [CIs]) by scanner type for GE-Imatron C-150 (EBCT), Siemens Volume Zoom (MDCT-4), and GE LightSpeed Plus (MDCT-4) being 15.8 (15.1,16.6), 17.5 (16.5, 18.5) and 15.7 (14.5,17.1), respectively.

One important limitation of this study was the difference in methodologies used by the scanners. The triggers in this study used 80% gating for EBCT and 50% gating for MDCT.[67] Mao et al[56] demonstrated that the Agatston score variability with EBCT decreases from 24% to 15% with use of an early diastolic trigger rather than the 80% trigger employed in the MESA study ($P<0.05$). The measure of CACP volume in MESA had a mean relative difference of 18% with both technologies, and this 2% improvement as compared with the Agatston score was statistically significant. This improvement in reproducibility with the volume score is consistent with this measure not accounting for information related to plaque density (ie, calcium mass). The results from MESA

demonstrate good performance by both cardiac CT technologies with regard to presence, absence, and amount of CACP.

There has been some debate about using retrospective gating instead of prospective gating with MDCT to further improve reproducibility, despite the increased radiation exposure. Ohnesorge et al[69] studied 50 patients using retrospective gating, demonstrating mean variability of 23% (Agatston score) and 21% (volume score) when using nonoverlapping increments of 3 mm. A considerable reduction in rescan variability can be achieved by overlapping the slices obtained (Agatston 12%, volume 8%) with $P<0.01$. Considerably higher mean variability is present for the patient subgroup with low to mild calcification if image data with nonoverlapping increments are used (Agatston 42%, volume 34%). The radiation dose reported for this methodology was >2.6 mSv per patient, representing a 2-fold increase as compared with prospectively gated MDCT studies.

Van Hoe et al[65] evaluated 50 patients and reconstructed the retrospective datasets at 3 different time intervals to try to minimize interscan variability. The mean percentage interscan variability was 30±31% with the use of an image reconstruction window of 40%, 33±37% with use of an image reconstruction window of 50%, and 27±22% with use of the optimal image reconstruction window. The authors stated, "Although we used the same technique as that of Ohnesorge et al,[69] we found mean interscan variability values that were 2 to 3 times higher. No obvious explanation can be given for these striking differences."

Use of retrospective gating in an attempt to improve reproducibility with MDCT is associated with a higher radiation exposure, increased interreader variability, and markedly increased interpretation times. In 1 study of 30 patients, Agatston and volumetric scores were assessed by using 16-detector retrospectively gated MDCT.[70] For each patient, 10 datasets were created that were evenly spaced throughout the cardiac cycle. Nineteen (63%) of 30 patients could be assigned to >1 risk group depending on the reconstruction interval used to measure the calcium score. Agatston and volumetric scores both proved highly dependent on the reconstruction interval used (coefficient of variation ≤63%), even with the most advanced CT scanners. Accurate and reproducible quantification of coronary calcium using retrospective gating seems to require analysis of multiple reconstructions.

The AHA Writing Group proposes that the following minimum requirements be met in scanning for CAC[71]:

1. Use of an electron beam scanner or a 4-level (or greater) MDCT scanner
2. Cardiac gating
3. Prospective triggering for reducing radiation exposure
4. A gantry rotation of at least 500 ms
5. Reconstructed slice thickness of 2.5 to 3 mm to minimize radiation in asymptomatic persons (and to provide consistency with established results)
6. Early to mid-diastolic gating

1.6. Radiation Dose for Cardiac CT

CT uses x-rays, a form of ionizing radiation, to produce the information required for generating CT images. Although all

individuals are exposed to ionizing radiation from natural sources on a daily basis, healthcare professionals involved in medical imaging must understand the potential risks of a test and balance them against the potential benefits. This is particularly important for diagnostic tests that will be given to healthy individuals as part of a disease-screening or risk-stratification program. For healthcare professionals to effectively advise individuals, they must have an understanding of the exposure involved.

The FDA, in describing the radiation risks from CT screening,[72] used the following language:

In the field of radiation protection, it is commonly assumed that the risk for adverse health effects from cancer is proportional to the amount of radiation dose absorbed and the amount of dose depends on the type of x-ray examination. A CT examination with an effective dose of 10 millisieverts (abbreviated mSv; 1 mSv=1 mGy in the case of x-rays) may be associated with an increase in the possibility of fatal cancer of approximately 1 chance in 2000. This increase in the possibility of a fatal cancer from radiation can be compared with the natural incidence of fatal cancer in the US population, about 1 chance in 5. Nevertheless, this small increase in radiation-associated cancer risk for an individual can become a public health concern if large numbers of the population undergo increased numbers of CT procedures for screening purposes. It must be noted that there is uncertainty regarding the risk estimates for low levels of radiation exposure as commonly experienced in diagnostic radiology procedures. There are some authorities who question whether there is adequate evidence for a risk of cancer induction at low doses. However, this position has not been adopted by most authoritative bodies in the radiation protection and medical arenas.

Effective dose is an estimate of the dose to patients during an ionizing radiation procedure. It measures the total energy entered into the body and then takes into account the sensitivity of the organs irradiated. Although it has many limitations, it is often used to compare the dose from a CT examination or other examination using ionizing radiation to the background radiation a patient experiences in a year. Units are either millirem (mrem) or millisieverts (mSv); 100 mrem is 1 mSv. The estimated dose from chest x-ray is 0.04 to 0.10 mSv, and the average annual background radiation in the United States is 3 to 3.6 mSv.[10]

One drawback of MDCT as compared with EBCT is the higher radiation exposure to the patient (Table 4).[10,11,73–84] The x-ray photon flux expressed by the product of x-ray tube current and exposure time (mAs) is generally higher with MDCT. For example, 200 mA with 0.5-second exposure time yields 100 mAs in MDCT versus 614 mA (fixed tube current) with 0.1-second exposure time yields 61.4 mAs in EBCT.

Hunold et al[10] performed a study of radiation doses during cardiac examinations. Coronary calcium scanning was performed with EBCT and 4-level MDCT using prospective triggering to assess each patient's effective radiation expo-

sure, which was then compared with measurements made during cardiac catheterization. EBCT yielded effective doses of 1.0 and 1.3 mSv for men and women, whereas MDCT using 100 mA, 140 kV, and 500-ms rotation yielded 1.5 mSv for men and 1.8 mSv for women. Invasive coronary angiography yielded effective doses of 2.1 and 2.5 mSv for men and women, respectively.

When similar protocols using single-detector-row CT (SD CT) and MDCT were compared, MDCT resulted in a dose profile approximately 27% higher than that from SD CT in the plane of imaging (8.0 versus 6.3 mGy) and 69% higher adjacent to the plane of imaging (6.8 versus 4.0 mGy).[74] The individual doses to the kidneys, uterus, ovaries, and pelvic bone marrow were 92% to 180% higher with MDCT than with SD CT. The authors concluded, "With image noise constant between SD CT and MDCT, the radiation dose profile both inside and outside the plane of imaging was higher with MDCT than with SD CT. Organ dose also was higher with MDCT than with SD CT."

Because retrospective gating exposes the patient to significantly higher radiation, several techniques have been implemented to reduce those exposures. Mahnken et al[75] studied body-weight dosing (reducing the radiation exposure based on body size) and measured the mean of the effective radiation dose with and without dose modulation. The radiation dose for a calcium scan using MDCT was 4.44 mSv (range, 3.28 to 5.88 mSv) for women and 3.01 mSv (range, 2.52 to 4.18 mSv) for men, whereas with dose modulation, the mean of the calculated radiation dose was 3.34 mSv (range, 2.39 to 3.83 mSv) for women and 2.66 mSv (range, 2.09 to 3.53) for men.

1.6.1. Radiation Exposure During CT Angiography

MDCT angiography requires retrospective gating, associated with significantly greater radiation exposures, to acquire images. Radiation doses of cardiac MDCT scans reported in the literature vary a great deal depending on the scan parameter settings. The tube voltages in the published protocols vary from 120 to 140 kVp, and the tube currents vary from 150 to 600 mA.[76] In contrast, the scan settings of EBCT used for cardiac imaging were fixed, in the older technology, to 130 kVp, 630 mA, and 100-ms exposure time. These EBCT settings have been somewhat altered, however, by the newer e-Speed technology, with both higher kVp and mA potential (140 kVp, 1000 mA). Newer protocols for MDCT angiography allow for increased power utilization, with settings as high as 900 mA possible. These higher settings will further increase the radiation dose, which is an issue to be considered when performing these protocols.

Pitch is calculated as table speed divided by collimator width. A low pitch (low table speed) allows for overlapping data from adjacent detectors. Most commonly, physicians use a low table speed and thin collimation width, leading to a large number of very thin axial slices, which are of great value for imaging the heart with high resolution. The tradeoff for these overlapping images is a markedly higher radiation exposure.[76] These protocols are also responsible for substantial increases in radiation doses, especially for the MDCT

TABLE 4.   Radiation Doses With EBCT and MDCT Coronary Angiography

Author, Year[Reference]	EBCT Effective Dose	MDCT Prospective Trigger	MDCT Retrospective Gating	EBCT Angiography	MDCT Angiography	Cardiac Catheterization
Becker, 1999[46]	0.8 mSv		5.3 mSv			3.3 mSv
Ohnesorge, 2002[69]			3.0 mSv (m)			
			4.0 mSv (f)			
Cohnen, 2001[73]			2.8 mSv (m)			
			3.6 mSv (f)			
Jakobs, 2002[84]		2 mSv (m)				
		2.5 (f)				
		1 mSv (m)*				
		1.4 (f)*				
Hunold, 2003[10]	1 mSv (m)	1.5 mSv (m)	3 mSv (m)	1.5 mSv (m)	10.9 mSv (m)	2.1 mSv (m)
	1.3 mSv (f)	1.8 mSv (f)	3.6 mSv (f)	2.0 mSv (f)	13.0 mSv (f)	2.5 mSv (f)
Morin, 2003[9]	0.7 mSv	1.0 mSv	2.6–4.1 mSv	1.1 mSv	9.3–11.3 mSv	
Kopp, 2002[169]			7.6 mSv (m)			
			9.2 mSv (f)			
Achenbach, 2001[166]			6.7 mSv (m)			
			8.1 mSv (f)			
Flohr, 2003[77]		0.5 mSv (m)	1.9–2.2 mSv (m)		5.7–7.1 mSv (m)	
		0.8 mSv (f)	2.8–3.1 mSv (f)		8.5–10.5 mSv (f)	
			1-1.5 mSv (m)*		2.9–5 mSv (m)*	
			1.4–2 (f)*		4.2–7.4 mSv (f)*	
Trabold, 2003[78]			2.9 mSv (m)		8.1 mSv (m)	
			3.6 mSv (f)		10.9 mSv (f)	
			1.6 mSv (m)*		4.3 (m)*	
			2 mSv (f)*		5.6 (f)*	
Carr, 2000[49]	0.6 mSv (m)	0.9–1.5 mSv (m)	4.6 mSv (m)			
	0.7 mSv (f)	1.1–1.9 mSv (f)	5.6 mSv (f)			
Raff, 2005[80]					13 mSv (m)	
					18 mSv (f)	

(m) indicates male; (f), female.
*With dose modulation.

systems, with dose estimates of up to 11 to 13 mSv per study (Table 4).

Two studies have measured the radiation doses for CT angiography, comparing EBCT and 4-slice MDCT. The first reported EBCT angiography doses of 1.5 to 2.0 mSv, MDCT angiography doses of 8 to 13 mSv, and coronary angiography doses of 2.1 to 2.3 mSv, while the second reported EBCT angiography doses of 1.1 mSv and MDCT doses of 9.3 to 11.3 mSv.[9,10] Newer MDCT studies report that radiation doses are similar with 16-level multidetector scanners and higher with 64 MDCT.[77,78] Studies estimate radiation exposure for 16-row MDCT at 8.8 mSev for a 16×0.75-mm scan protocol with a pitch of 0.28 and power of 370 mA[79] and at 13 and 18 mSv (for men and women, respectively) with 64-row MDCT.[80] It should be noted that nuclear imaging has similar radiation exposure doses for cardiac studies (8 to 12 mSv).[81] Specifically, technetium studies are on the lower end of this spectrum (6 to 8 mSev on average), and thallium studies have been reported as high as 27 mSv.[82]

With the retrospective ECG-gating mode, scan data are acquired and available for the entire phase of the cardiac cycle. In most cases, however, the scan data used for image reconstruction are selected only during the diastolic phase. This implies that a high tube current is required only during the diastolic phase and that a low tube current is acceptable during the remaining cardiac phase. Modulating the tube current online with prospective ECG control (dose modulation) is reported to help reduce radiation exposure substantially without decreasing diagnostic image quality.[83,84]

For MDCT coronary angiography, dose modulation techniques reduce radiation exposures[84] and should be employed whenever possible. The effects of dose reduction are more pronounced for lower heart rates. Also, using the lowest necessary mA during each study will also help limit radiation exposures during these procedures. For MDCT, increased numbers of detectors allow for better collimation and spatial reconstructions. Having more of the heart visualized simultaneously will also allow for reductions in the contrast requirements and breathholding for the patient, further improving the methodology.

In summary, CT technology is evolving rapidly and radiation exposures are likely to be reduced with modification of

the hardware and scanning protocols. The clinical relevance of differences in radiation dose between different technologies is unknown, but most would agree that less radiation is better for patients than more radiation. The AHA Writing Group, reviewing the available literature, endorses the use of a prospective ECG trigger for measurement of CACP with a slice collimation of 1.5 to 3 mm for clinical practice. EBCT systems have an effective dose of 0.7 to 1 mSv (for men) and 0.9 to 1.3 mSv (for women), and MDCT systems have an effective dose of 1 to 1.5 mSv (for men) and 1 to 1.8 mSv (for women).[9,10,76] For CT angiography, the higher radiation doses suggest the need for greater forethought when using these tests, and use of these higher radiation exposure tests in asymptomatic persons for screening purposes is not currently recommended.

2. Clinical Utility of CACP Detection

This is the first time that the AHA evidence-based scoring system (see http://circ.ahajournals.org/manual/manual_IIstep6.shtml) has been incorporated into the AHA's evaluation of cardiac CT. The purpose of the scoring system is to assist the clinician in interpreting these recommendations and formulating treatment decisions. The system is based on both a classification of recommendations and the level of evidence. Each treatment recommendation has been assigned a class and a level of evidence. The use of this system should support but not supplant the clinician's decision making in the management of individual patients' cases.

Classification of Recommendations

- **Class I:** Conditions for which there is evidence, general agreement, or both that a given procedure or treatment is useful and effective.
- **Class II:** Conditions for which there is conflicting evidence, a divergence of opinion, or both about the usefulness/efficacy of a procedure or treatment.
  - **Class IIa:** Weight of evidence/opinion is in favor of usefulness/efficacy.
  - **Class IIb:** Usefulness/efficacy is less well established by evidence/opinion.
- **Class III:** Conditions for which there is evidence, general agreement, or both that the procedure/treatment is not useful/effective and in some cases may be harmful.

Level of Evidence

- **Level of Evidence A:** Data derived from multiple randomized clinical trials
- **Level of Evidence B:** Data derived from a single randomized trial or nonrandomized studies
- **Level of Evidence C:** Consensus opinion of experts

2.1. CT Coronary Calcium and Symptomatic Patients

The utility of measuring CAC in symptomatic patients has been widely studied and discussed in depth in a previous ACC/AHA statement,[4] as well as in the recent AHA Cardiac Imaging Committee consensus statement, "The Role of Cardiac Imaging in the Clinical Evaluation of Women With Known or Suspected Coronary Artery Disease."[5] A positive

TABLE 5.   Sensitivity and Specificity of Diagnostic Tests for Evaluation of CAD

	No. of Patients	Sensitivity, %	Specificity, %
Stress treadmill[85]	2456	52	71
Exercise SPECT[85,86]	4480	87	73
Stress echocardiography[85]	2637	85	77
EBCT calcium[22,23,89]	5730	85	75

EBCT study (indicating the presence of CACP) is nearly 100% specific for atheromatous coronary plaque[4,5,26–28] but is not highly specific for obstructive disease, as both obstructive and nonobstructive lesions have calcification present in the intima. The presence of CACP by EBCT is extremely sensitive, however, for obstructive (>50% luminal stenosis) CAD (95% to 99%).[4,5,20–22] This has led to much confusion over the interpretation of CACP as a diagnostic test.

A large multicenter study on EBCT for diagnosis of obstructive CAD in symptomatic persons (n=1851) found that the sensitivity and specificity of CACP were 96% and 40%, respectively.[22] However, increasing the cutpoint for calcification markedly improves the specificity. In this same study, increasing the CACP cutpoint to >80 decreased the sensitivity to 79%, while increasing the specificity to 72%. In another large study (n=1764) comparing CACP to angiographic disease, use of a CACP score >100 led to a sensitivity of 95% and a specificity of 79% for the detection of significant obstructive disease by angiography.[23] Summing these 2 large studies (n=3615) leads to a sensitivity of 85% with a specificity of 75%. In a meta-analysis of 44 studies, technetium stress was found to have a mean sensitivity of 87% and mean specificity of 64%,[85] similar to the results of CACP. Thus, CACP measurements have a similar accuracy to other commonly accepted modalities for diagnosis of obstructive CAD by angiography (Table 5). For all diagnostic accuracy literature, one must be concerned about posttest referral bias, whereby positive tests are the cause for the referral to the catheterization laboratory. If the test is allowed to be part of the referral pattern, the sensitivity will increase and the specificity will decrease. However, for the 3 studies of EBCT, imaging was performed after the patient was referred for an invasive angiogram. The reason for the low specificity with CAC testing is the presence of CAC in nonobstructive as well as obstructive lesions.

In direct comparison studies, EBCT coronary calcium has been shown to be comparable to nuclear exercise testing in the detection of obstructive CAD.[87,88] The accuracy of EBCT is not limited by concurrent medication, the patient's ability to exercise, baseline ECG abnormalities, or existing wall motion abnormalities. Patients whose studies prove negative would be less likely to undergo invasive angiography. More comparison work between modalities is clearly needed.

Data also support a complementary role for coronary calcium and MPS measurements. A recent study of 1195 patients who underwent CACP measurement and MPS assessment demonstrated that the presence of CACP was the most powerful predictor that a nuclear test would be positive for ischemia and that <2% of all patients with CACP <100

had positive MPS studies.[6] EBCT, owing to its high sensitivity for flow-limiting CAD, may be useful as a filter before angiography or stress nuclear imaging, with more caution in younger patients. Knez et al[89] studied 2115 consecutive symptomatic patients (n=1404 men, mean 62±19 years of age) with no prior diagnosis of CAD, finding CAC in more than 99% of patients with obstructive CAD. No calcium was found in 7 of 872 men (0.7%) and in 1 of 383 women (0.02%) who had significant luminal stenosis on coronary angiography. Seven of these 8 patients with missed obstructive disease and scores of 0 were <45 years of age.

Recent ACC/ASNC appropriateness criteria support that a low calcium score precludes the need for MPS assessment and a high score warrants further assessment.[8] These appropriateness criteria suggest nuclear testing may generally be inappropriate in patients with calcium scores <100, as the probability of obstruction or abnormal scan is very low. However, more recent evidence suggests that MPS may be indicated in patients with diabetes and those with a family history of CAD who have a calcium score <100.[90–92] For the remaining asymptomatic patients, a person with an Agatston score >400 may benefit from functional testing to detect occult ischemia. The use of functional testing is paramount in determining the need for revascularization, as functionally insignificant lesions do not benefit from revascularization.

CACP may also be considered in determining the etiology of cardiomyopathy (**Class IIb, Level of Evidence: B**). The clinical manifestations of patients with ischemic cardiomyopathy are often indistinguishable from those with primary dilated cardiomyopathy. One large study of 120 patients with heart failure of unknown etiology demonstrated that the presence of CACP was associated with 99% sensitivity for ischemic cardiomyopathy.[93] Another study demonstrated similarly high sensitivity using dual CT to differentiate ischemic from nonischemic cardiomyopathy.[94] Direct comparison studies have demonstrated this methodology to be more accurate than echocardiography and MPS techniques.[95,96] Additional comparative prognostic and diagnostic evidence is required to evaluate the role of CT as compared with conventional stress imaging techniques, as well as an assessment developing marginal cost-effectiveness models.

Another potential application of CACP relates to the triage of patients with chest pain. Three studies have documented that CACP is a rapid and efficient screening tool for patients admitted to the emergency department with chest pain and for whom ECG findings have been nonspecific.[97–99] These studies show sensitivities of 98% to 100% for identifying patients with acute MI and very low subsequent event rates for persons with negative test results. The high sensitivity and negative predictive value may allow early discharge of those patients with nondiagnostic ECG and negative CACP scans (score=0). Long-term follow-up of this cohort demonstrates patients without demonstrated CACP at the time of the emergency visit are at very low risk of subsequent events.[97]

Recommendation: Patients with chest pain with equivocal or normal ECGs and negative cardiac enzyme studies may be considered for CAC assessment (**Class IIb, Level of Evidence: B**).

For the symptomatic patient, exclusion of coronary calcium may be an effective filter before invasive diagnostic procedures or hospital admission. EBCT studies of more than 7600 symptomatic patients undergoing cardiac catheterization demonstrate negative predictive values of 96% to 100%, allowing physicians a high level of confidence that an individual with no coronary calcium (score=0) does not have obstructive angiographic disease.[21–23,89] Calcium scores <100 are associated with a very low probability (<2%) of abnormal perfusion on nuclear stress tests[6] and <1% probability of significant obstruction (>50% stenosis) on cardiac catheterization.[6,21–23,89] While models suggest this is a cost-effective algorithm, further testing and prospective analysis are required.[100,101]

Recommendation: Coronary calcium assessment may be reasonable for the assessment of symptomatic patients, especially in the setting of equivocal treadmill or functional testing (**Class IIb, Level of Evidence: B**).

2.2. CT Coronary Calcification and Clinical Outcomes in Asymptomatic Individuals

Calcification of the coronary arteries occurs in approximate proportion to the severity and extent of coronary atherosclerosis.[102] In a landmark study of atherosclerosis, persons dying of coronary disease were found to have 2-fold to 5-fold greater amounts of coronary calcification than age-matched controls dying accidentally or of other natural causes.[103] Eight studies have examined the prognostic accuracy of CACP score by EBCT.

The first and longest study of EBCT scanning of the coronary arteries, the South Bay Heart Watch study,[1,104–106] began in 1990 as a prospective study of the prognostic accuracy of cardiac fluoroscopy in 1461 asymptomatic, high-risk individuals. In 1992, 1289 study participants (mean age 66±8 years) underwent EBCT scanning. Although an early analysis revealed no incremental advantage of EBCT scanning over conventional risk factor assessment for hard coronary events,[104] long-term (median=7.0 years) follow-up has demonstrated that the CACP score adds predictive power beyond that of standard coronary risk factors and C-reactive protein.[1,105] In multivariable models, a CACP score >300 was highly statistically significant and independently predictive of fatal or nonfatal MI, compared with a score of 0 (HR=3.9, $P<0.001$). In this study, patients with an FRS of 16% to 20% and a CAC score ≥300 had an annual event rate of 2.8%. This patient group would therefore have the 10-year event rate ≥20% that indicates high risk by current NCEP criteria.

From a retrospective cohort study of 632 asymptomatic persons (mean age 52±9 years, mean follow-up =2.7 years), the annual rate of nonfatal MI or CHD death increased from 0.045% in the lowest quartile of calcium scores to 2.7% among subjects in the highest quartile of calcium scores (a 59-fold increase).[107] Thus, patients with high calcific plaque burden did exceed the high-risk threshold (>2% per year hard cardiac event rate). These investigators demonstrated that EBCT added incremental benefit over and above standard coronary risk factors for risk prediction.[108]

Another study of 1172 asymptomatic persons (mean age 53±11 years, follow-up=3.6 years) demonstrated that a calcium score >160 was highly predictive of nonfatal MI or CHD death with an elevated risk 23.3-fold higher for CACP scores >160 versus CACP <160.[109] This study did not measure risk factors but did multivariable analysis to adjust for self-reported cardiovascular risk factors.

Wong et al[110] reported on 3.3-year follow-up in 926 asymptomatic persons (mean age 54±10 years). The calcium score predicted events independently of age, gender, and other cardiovascular risk factors (risk-adjusted RR=8.8 for scores in the fourth versus first quartile). Kondos et al[111] reported 37-month follow-up in 5635 initially asymptomatic low-risk to intermediate-risk adults (mean age 51±9 years). While follow-up was only obtained in 64% of patients, multivariable modeling demonstrated that patients with scores >170 (top quartile of scores) had an RR for hard cardiac events of 7.24-fold (95% CI, 2.01 to 26.15) as compared with patients without CACP. Finally, in a larger cohort of 10 377 asymptomatic individuals undergoing cardiac risk factor evaluation and CACP measurement with EBCT, a study with a mean follow-up of 5.0 years[112] used a risk-adjusted model to show that CACP was an independent predictor of all-cause mortality ($P<0.001$).

Shemesh et al[113] reported on a 3.8-year follow-up of 446 hypertensive patients prospectively followed up after risk factor measurement and CACP. CACP (total coronary calcium score >0) independently predicted cardiovascular events with an odds ratio (OR) of 2.76 (95% CI 1.09 to 6.99, $P=0.032$). Of note, this was the first prognostic study with MDCT (using a dual-slice CT system).

A significant limitation to a number of the early studies, with the exception of the South Bay Heart Watch Study, is that they were retrospective and did not include measured risk factors. However, 6 recently reported prospective studies, all with measured risk factors, now demonstrate the independent and incremental prognostic value of CAC measurement over the FRS.

The St. Francis Heart Study is a prospective observational study of 4613 subjects (59±5 years of age) with 4.3 years of follow-up.[114] A calcium score >100 predicted cardiovascular events, all coronary events, and the sum of nonfatal MI or CHD death events with RR ratios ranging from 9.2 to 11.1. Of note in this prospective series, the calcium score predicted cardiovascular events independently of standard risk factors and high-sensitivity C-reactive protein ($P<0.004$). Additionally, the calcium score also had improved event classification when compared with the FRS (area under the ROC curve 0.79±0.03 versus 0.68±0.03, $P=0.0006$).

Similarly, in a younger cohort of asymptomatic persons, the Prospective Army Coronary Calcium (PACC) Project[115] reported 3-year mean follow-up in 2000 participants (mean age 43 years). Participants were evaluated with measured coronary risk variables and coronary calcium detected by EBCT. Coronary calcium was associated with an 11.8-fold increased risk for incident CHD ($P<0.002$) in a Cox model controlling for the FRS. Among those with CAC, the risk of coronary events increased incrementally across tertiles of coronary calcium severity (HR 4.3 per tertile). A family

history of premature CHD was also predictive of incident events. A major limitation of this study is that coronary events occurred in only 9 of the men who participated, with no events reported in women. Thus, the CIs around the RR estimates were rather large. The authors concluded, "In young, asymptomatic men, the presence of CAC provides substantial, cost-effective, independent prognostic value in predicting incident CHD that is incremental to measured coronary risk factors."

The Rotterdam Heart Study[116] investigated a general, asymptomatic population of 1795 elderly subjects. Participants who were followed up prospectively (mean age=71 years) had CAC and measured risk factors. During a mean follow-up of 3.3 years, 88 cardiovascular events, including 50 coronary events, occurred. The multivariable-adjusted RR of coronary events was 3.1 (95% CI, 1.2 to 7.9) for calcium scores of 101 to 400, 4.6 (95% CI, 1.8 to 11.8) for calcium scores of 401 to 1000, and 8.3 (95% CI, 3.3 to 21.1) for calcium scores >1000, respectively, compared with calcium scores of 0 to 100. Risk prediction based on the cardiovascular risk factors improved when coronary calcification was also taken into account.

The Cooper Clinic Study[117] included 10 746 adults who were 22 to 96 years of age and who were free of known CHD. During a mean follow-up of 3.5 years, 81 hard events (CHD death, nonfatal MI) occurred. Age-adjusted rates (per 1000 person-years) of hard events were computed according to 4 CAC categories: no detectable CAC and incremental sex-specific thirds of detectable CAC; these rates were, respectively, 0.4%, 1.5%, 4.8%, and 8.7% (trend $P<0.0001$) for men and 0.7%, 2.3%, 3.1%, and 6.3% (trend $P<0.02$) for women. The association between CAC and CHD events remained significant after adjustment for CHD risk factors. The results revealed a strong, graded association between CAC scores and incident CHD events among asymptomatic individuals free of known CHD at the time of EBCT scanning. The findings were consistent for men and women and held after adjustment for age and conventional CHD risk factors. CAC was associated with CHD events in persons with no baseline CHD risk factors and in younger (<40 years of age) and older (>65 years of age) study participants.

A Munich study determined the extent of CAC by MDCT in 924 patients (443 men, 481 women, 59.4±18.7 years of age).[118] During the 3-year follow-up period, the event rates for coronary revascularization (5.4%/year versus 2.9%/year), MI (3.8%/year versus 1.8%/year), and cardiac death (2.1%/year versus 1.0%/year) in patients with volume scores above the 75th percentile were significantly higher compared with the total study group. Correspondingly, the volume scores in patients with revascularization (397±187), MI (412±176), and cardiac death (422±184) were significantly higher compared with patients without cardiovascular events (218±167). In addition, no cardiovascular events occurred in patients with scores of 0. In this study, 44 of 50 (88%) of MIs occurred in patients with scores in the top 25th percentile, and a receiving operator characteristic (ROC) curve demonstrated that the calcium score outperformed both Prospective Cardiovascular Münster (PROCAM) study and FRS

($P<0.0001$), where 36% and 34% of MIs occurred in the high-risk cohorts, respectively.

From a synthesis of both retrospective and prospective cohort studies, there appears to be a directly proportional relationship between CHD risk and the extent of CAC, as measured by the Agatston score. According to a meta-analysis by Pletcher et al,[119] the risk of major CHD events increased 2.1-fold and 10-fold for scores ranging from 1 to 100 and >400, respectively, as compared with scores of 0. This relationship has been established when predicting all-cause mortality, cardiovascular events, CHD death or nonfatal MI, and overall CHD events. When estimating all-cause mortality, researchers report the independent prognostic value of the coronary calcium score for diabetics and smokers, including specific outcome evaluations in women.[92,120,121]

A study demonstrated the risk stratification in uncomplicated type 2 diabetes in a prospective evaluation of coronary artery calcium and MPS.[91,92] Established risk factors and CAC scores were prospectively measured in 510 asymptomatic individuals with type 2 diabetes (mean age 53 ± 8 years, 61% men) without prior cardiovascular disease. MPS was performed in all subjects with CAC >100 Agatston units (AU) (n=127) and a random sample of the remaining patients with CAC ≤100 AU (n=53). Twenty events occurred (2 coronary deaths, 9 nonfatal MIs, 3 acute coronary syndromes, 3 nonhemorrhagic strokes, and 3 late revascularizations) during a median follow-up of 2.2 years (25th to 75th percentile=1.9 to 2.5 years). Multivariable logistic regression analysis showed that CAC score was the only predictor of myocardial perfusion abnormality ($P<0.001$). In the multivariable model, the CAC score and extent of myocardial ischemia were the only independent predictors of outcome ($P<0.0001$). ROC analysis demonstrated that CAC predicted cardiovascular events with the best area under the curve (0.92), significantly better than the United Kingdom Prospective Diabetes Study Risk Score (0.74) and the FRS (0.60, $P<0.0001$). The RR of a cardiovascular event for a CAC score of 101 to 400 was 10.1 and increased to 58.1 for scores >1000 ($P<0.0001$). The RR for ischemic burden was 5.5 for 1% to 5% burden, increasing to 12.3 for an ischemic burden >5% ($P<0.0001$). No cardiac events or perfusion abnormalities occurred in subjects with CAC ≤10 AU up until 2 years of follow-up. CAC and MPS findings were synergistic for the prediction of short-term cardiovascular events. The authors concluded that subclinical atherosclerosis measured by CAC imaging is superior to the established cardiovascular risk factors for predicting silent myocardial ischemia and short-term outcomes in patients with type 2 diabetes.

2.3. Limitations

The potential risk stratification (whether with CAC or other tests) first requires calculation of the Framingham risk. For example, a 45-year-old man with a total cholesterol of 225 mg/dL, an HDL cholesterol of 45 mg/dL, and systolic blood pressure of 140 mm Hg has a 10-year risk of 4% if he is not a smoker. If the same individual has a systolic blood pressure of 160 mm Hg, his 10-year risk is still only 5%. The article by Greenland et al[1] demonstrated the futility of calcium scanning when the Framingham risk is <10%. In that study, when the FRS was 0% to 9%, there was no increased risk with a CACS ≥301.[1] Similar data are available from the St. Francis Heart Study.[114] Thus, the risk stratification of individuals at low risk for CHD (<10% risk in 10 years) will not change with CACS testing, a conclusion further supported by the US Preventive Services Task Force.[20]

Modifying this case illustrates how noninvasive testing could influence patient treatment in an intermediate-risk patient. Consider the same patient 5 years later, this 50-year-old asymptomatic man who does not smoke, who has a blood pressure level of 140/85 mm Hg (treated), total cholesterol of 225 mg/dL, and HDL cholesterol of 45 mg/dL. This man's risk now falls into the "intermediate" zone with a 10% risk. If further testing were done with EBCT and a coronary calcium score >169 were found, the physician would be able to reassign him to a higher risk category (at least 20% in 10 years) and justifiably proceed more aggressively to reduce his risk factors.[122] Data from Greenland et al[1] demonstrated that intermediate-risk patients with an elevated CAC score (intermediate FRS and CAC >300) had an annual hard event rate of 2.8%, or a 10-year rate of 28%, and thus would be considered high risk. The best estimates of RR from this study demonstrated that a CAC score >300 had an HR of about 4 compared with a score of 0. This would mean that the estimated risk in the intermediate patient with a CAC score of 0 might be reduced by at least 2-fold, while the risk of a person with a CAC score of 300+ would be increased by about 2-fold. Thus, the person with high CAC and intermediate FRS is now reclassified as high risk. If the calcium score were 0 or very low, the patient's posttest risk assessment would be reduced.

Two of the largest studies with measured risk factors demonstrate a posttest probability of events of approximately 0.1% per year for persons without CACP present. Taylor et al[115] prospectively followed 3000 persons (mean age 43 years) for 3 years. CHD events occurred in only 2 of 1263 participants without CAC (event rate 0.16%; $P<0.0001$). Thus, a negative scan was associated with a 0.05% per year risk of events. In another large prospective, cohort study, 4903 asymptomatic persons 50 to 70 years of age were assessed: Only 8 of 1504 persons (0.5%) with scores of 0 had a coronary event over the next 4.3 years, with an annual event rate of only 0.1%. Two small prospective studies demonstrated no events in persons with scores of 0 over 2 to 3 years of follow-up.[91,92] The longest studies performed to date demonstrate that there is still a possible risk of MI or death associated with a negative (0) scan. In this study, 14 events occurred among 316 persons with scores of 0 at baseline over the subsequent median follow-up of 7 years (annual event rate 0.6%, 10-year risk 6.3%).[1]

EBCT is one of many contenders in a crowded field of emerging CAD risk-assessment tools. For example, other noninvasive modalities (eg, carotid intima-media thickness) and blood tests (eg, homocysteine and C-reactive protein) are under investigation with the aim of improving our ability to risk-stratify patients. Clinicians require high standards for assessing the value of new medical therapies and devices; evidence-based methods for evaluating screening strategies

TABLE 6.   Characteristics and Risk Ratio for Follow-Up Studies Using EBCT

Author	No.	Mean Age, y	Follow-Up Duration, y	Calcium Score Cutoff	Comparative Group for RR Calculation	Risk Factor Assessment	Relative Risk Ratio
EBCT studies in symptomatic cohorts							
Georgiou[98]	192	53	4.2	Median*	Below median	Measured	13.1
Detrano[123]	491	57	2.5	Top quartile	Bottom quartile	Self-reported	10.8
Keelan[124]	288	56	6.9	Median (>480)	Below median	Measured	3.2
Moehlenkamp[125]	150	63	5	CACP >1000	No CACP	Measured	2.5
EBCT studies in asymptomatic populations							
Arad[109]	1173	53	3.6	CACP >160	CACP <160	Self-reported	20.2
Detrano[104]	1196	66	3.4	CACP >44	CACP <44	Measured	2.3
Park[105] (subset of Detrano[104])	967	67	6.4	CACP >142.1	CACP <3.7	Measured	4.9
Raggi[107]	632	52	2.7	Top quartile†	Lowest quartile	Self-reported	13
Shemesh[113]	446	64	3.8	CACP >0	CACP=0	Measured	2.8
Wong[110]	926	54	3.3	Top quartile	Lowest quartile	Self-reported	8.8
Arad[114]	4613	59	4.3	CACP ≥100	CACP <100	Measured	9.2
Kondos[111]	5635	51	3.1	CACP	No CACP	Self-reported	3.86 (men) 1.53‡ (women)
Greenland[1]	1312	66	7.0	CACP >300	No CACP	Measured	3.9
Shaw[112]	10 377	53	5	CACP 401-1000	CACP ≤10	Self-reported	6.2§
Taylor[115]	2000	43	3	CACP	No CACP	Measured	11.8
LaMonte[117]	10 746	54	3.5	CACP top third	No CACP	Measured	8.7 (men) 6.3 (women)
Vliegenthart[116]	1795	71	3.3	>1000	0-100	Measured	8.1
Becker[118]	924	60	3	Top quartile (75th percentile)	Total study group	Measured	7.3

Duplicate series: Detrano, Park, and Greenland.
CACP indicates coronary artery calcium score; RR, relative risk ratio.
*Using age- and gender-matched cohorts, representing top quartile.
†Using age- and gender-matched cohorts, representing the top quintile.
‡After multivariate analysis, $P<0.05$ for men, $P=$ not significant for women.
§End point was all-cause mortality.

are important, as they ultimately dictate downstream testing, treatments, and costs. Current data support the benefit of CACP as a diagnostic test for particular patient populations in terms of diagnostic efficacy, acceptable safety, and affordability; however, further studies are warranted.

Despite the high quality of risk-stratification evidence, evidence is not available that screening with EBCT improves clinical outcomes by reducing mortality or morbidity from CAD (see section 2.5). In addition, cost-effectiveness models for the use of CACP are currently limited, since no study has demonstrated that EBCT reduces healthcare costs. The evidence does suggest that widespread and routine EBCT screening is unlikely to benefit low-risk or high-risk patients. Few patients with a low pretest probability of CAD will see their risk levels change enough to lead to changes in medical management. Patients with high pretest probabilities or diabetes are essentially at CAD-equivalent risk regardless of calcium score, and treatment of risk factors rather than screening would be more appropriate. There will be an expected decrease in efficacy of this test in older patients

(men >70 years of age or women >75 years of age), as atherosclerosis is more widespread in the elderly.

2.4. Recommendations of Professional Societies

In 2000, the ACC/AHA acknowledged the potential of coronary calcium to predict major coronary events.[4] However, due to the mixed data available at the time, routine scanning was not recommended. Subsequently, additional data have been published to strengthen the conclusion that CAC affords incremental risk prognostication (Table 6). The AHA Prevention Conference V concluded that "selected patients" could have CACP testing if initially found to be at intermediate risk (Table 7).[2] That committee agreed with a perspective paper[126] supporting the use of EBCT for risk stratification in intermediate-risk patients, which stated: "Recent work suggests that . . .EBCT. . . can also improve risk prediction in intermediate-risk patients."[109] Thus, with a prior probability of a coronary event in the intermediate range (≥6% in 10 years but ≤20% in 10 years), a calcium score of >100 would yield a posttest probability >2% per year in the

TABLE 7. Interpretation and Recommendations for CT Heart Scanning and CACP Scoring

1.	A negative test (score=0) makes the presence of atherosclerotic plaque, including unstable or vulnerable plaque, highly unlikely.
2.	A negative test (score=0) makes the presence of significant luminal obstructive disease highly unlikely (negative predictive power by EBCT on the order of 95% to 99%).
3.	A negative test is consistent with a low risk (0.1% per year) of a cardiovascular event in the next 2 to 5 years.
4.	A positive test (CAC >0) confirms the presence of a coronary atherosclerotic plaque.
5.	The greater the amount of coronary calcium, the greater the atherosclerotic burden in men and women, irrespective of age.
6.	The total amount of coronary calcium correlates best with the total amount of atherosclerotic plaque, although the true "atherosclerotic burden" is underestimated.
7.	A high calcium score (an Agatston score >100) is consistent with a high risk of a cardiac event within the next 2 to 5 years (>2% annual risk).
8.	Coronary artery calcium measurement can improve risk prediction in conventional intermediate-risk patients, and CACP scanning should be considered in individuals at intermediate risk for a coronary event (1.0% per year to 2.0% per year) for clinical decision-making with regard to refinement of risk assessment.
9.	Decisions for further testing (such as stress testing or cardiac catheterization) beyond assistance in risk stratification in patients with a positive CACP score cannot be made on the basis of coronary calcium scores alone, as calcium score correlates poorly with stenosis severity in a given individual and should be based upon clinical history and other conventional clinical criteria.

Adapted from ACC/AHA expert consensus document on EBCT for the diagnosis and prognosis of CAD.[4]

majority of patients, within the range of a CHD risk equivalent population and within a level requiring secondary prevention strategies.

The NCEP ATP III[3] supports the conclusions of the Prevention Conference V[2] and the ACC/AHA report[4] that high coronary calcium scores confirm an increased risk for future cardiac events, stating:

> Measurement of coronary calcium is an option for advanced risk assessment in appropriately selected persons. In persons with multiple risk factors, high coronary calcium scores (eg, >75th percentile for age and sex) denote advanced coronary atherosclerosis and provide a rationale for intensified LDL-lowering therapy. Moreover, measurement of coronary calcium is promising for older persons in whom the traditional risk factors lose some of their predictive power.

The European Cardiovascular Guidelines state, "The resulting calcium score is an important parameter to detect asymptomatic individuals at high risk for future CVD events, independent of the traditional risk factors."[126]

The Society of Atherosclerosis Imaging published guidelines for calcium scanning, with class I indications including (1) initial diagnostic test in ambulatory adults <65 years of age with atypical chest symptoms, in the absence of established cardiovascular disease; (2) supplementary diagnostic test in adults <65 years of age with indeterminate stress test results; and (3) emergency department evaluation of men <50 years of age and women <60 years of age with chest pain and normal or nondiagnostic ECGs. Class IIa recommendations include use for screening intermediate-risk patients and for assisting physicians in decision-making regarding initiation or change of drug therapy for cholesterol abnormalities in patients without established CVD.[127]

The 2004 AHA statement "Evidence-Based Guidelines for Cardiovascular Disease Prevention in Women"[128] deemed coronary calcification to be evidence of intermediate risk (10% to 20% 10-year risk) with a caveat that some patients with subclinical CVD will have >20% 10-year CHD risk and should be elevated to the high-risk category. The US Preven-

tive Services Task Force recommends against CACP scanning for either the presence of severe coronary artery stenosis or for prediction of CHD events in patients with no CHD symptoms or risk factors, specifically the low-risk patient.[20] This Task Force recommended neither for nor against scanning of intermediate-risk or high-risk patients.

A 2005 AHA scientific statement on cardiac imaging in women addressed the data on CACP[5]: "Given the evolving literature. . . current data indicate that CAD risk stratification is possible. Specifically, low CACP scores are associated with a low adverse event risk, and high CACP scores are associated with a worse event-free survival." This guideline included a recommendation to measure atherosclerosis burden using cardiac CT in clinically selected intermediate– CAD risk patients (eg, those with a 10% to 20% Framingham 10-year risk estimate) to refine clinical risk prediction and to select patients for more aggressive target values for lipid-lowering therapies.[5] Some guidelines (eg, AHA and NCEP ATP III) define intermediate risk as 10% to 20%, while others such as the Bethesda Conference define it as 6% to 20%.[129] A new ACC Clinical Expert Consensus Document for the recommended use of CACP is currently being drafted.

2.5. Utilizing Coronary Calcium Measure to Improve Outcomes

Ideally, there should be evidence that a strategy of refining risk assessments is associated with improved clinical outcomes compared with conventional risk prediction. No study has definitively demonstrated that screening with EBCT improves clinical outcomes by reducing mortality or morbidity from CAD. One study failed to show a significant effect of statins on outcomes when calcium scores were high ($P=0.08$).[130] This study was a double-blind, placebo-controlled, randomized clinical trial of atorvastatin 20 mg daily, vitamin C 1 g daily, and vitamin E (alpha-tocopherol) 1000 U daily versus matching placebos in 1005 asymptomatic, apparently healthy men and women 50 to 70 years of age with coronary calcium scores at or above the 80th percentile for age and gender. All study participants also received

aspirin, 81 mg daily. Mean duration of treatment was 4.3 years.

Treatment reduced low-density lipoprotein cholesterol by 39.1% to 43.4% ($P<0.0001$) and triglycerides by 11.2% to 17.0% ($P\leq0.02$), while reducing clinical end points by 30% (6.9% versus 9.9%, $P=0.08$). Event rates were related to baseline calcium score (prespecified analysis) and have been reduced in a subgroup of participants with baseline calcium score >400 (8.7% versus 15.0%, $P=0.046$ [42% reduction, not a prespecified analysis]). The magnitude of the risk reductions was similar to those in studies published to date of the same cholesterol-lowering agent in primary prevention cohorts.[131]

This study sample, however, was too small to detect a benefit given the surprisingly low overall rate of cardiovascular end points in asymptomatic patients. The authors of the trial acknowledged several limitations. Firstly, the power analysis was based on an event reduction with both statin and antioxidants, and thus, when antioxidants failed to reduce events, the study was significantly underpowered. Furthermore, all patients received aspirin, so this may have also reduced the primary end point in both placebo and treatment groups. However, a large, well-done randomized trial has been performed, yielding similarly negative results.

The 30% reduction in the primary end point of this study is similar to the reduction of atherosclerotic CVD (ASCVD) events seen in other large randomized clinical trials of statins, a class of drugs with unquestionable efficacy in this application.[132] This study had power of just 0.61 to detect a 30% reduction in events. Treatment reduced all CAD events by 28% ($P=0.13$), the sum of nonfatal MI and CHD death by 44% ($P=0.14$), and all ASCVD events occurring >90 days after initiation of therapy by 33% ($P=0.07$).

The definition of high risk on CAC was chosen based on age-based and gender-based cohorts rather than atherosclerosis burden (ie, absolute CAC scores). This was recommended as a risk-stratification technique in the NCEP ATP III report[3] but was later shown to be not as robust a predictor of risk as absolute plaque burden.[131] Thus, the authors established lower calcium score thresholds in younger persons than in older ones. Later evidence revealed that this was incorrect; that is, age was not a significant determinant of events. As a result of this error, the study population contained substantial numbers of low-risk individuals in whom treatment had little effect. Conversely, in 469 subjects with calcium scores >400, treatment reduced the event rate by 42% ($P=0.046$).

Thus, while outcome studies demonstrating that measurement of CAC leads to improved outcomes remains an ideal, the practicality of performing such a study is challenging.

Recommendations: It may be reasonable to measure atherosclerosis burden using EBCT or MDCT in clinically selected intermediate–CAD risk patients (eg, those with a 10% to 20% Framingham 10-year risk estimate) to refine clinical risk prediction and to select patients for more aggressive target values for lipid-lowering therapies (**Class IIb, Level of Evidence: B**).

It is important to recognize that widespread and routine EBCT screening is unlikely to benefit low-risk or high-risk patients. Few patients with low pretest probability of CAD will change risk levels enough as a result of the screening to require changes in medical management. Patients with high pretest probabilities or diabetes are essentially at CAD-equivalent risk regardless of calcium score, and treatment of risk factors would be more appropriate than screening.[133] While several studies demonstrated incremental prognostic ability of CAC in diabetes,[91,92,121] patients with diabetes should be treated for secondary prevention before risk stratification. Furthermore, prior studies have limited generalizability owing to a lack of ethnic diversity in their patient populations.

Recommendation: Low-risk (<10% 10-year risk) and high-risk (>20% 10-year risk) patients do not benefit from CAC measurement (**Class III, Level of Evidence: B**).

2.6. Limitations of the Use of Coronary Calcium for Detecting Obstructive Disease in Asymptomatic Persons

The NCEP ATP III full report states:

> The goal of improved risk assessment is a more selective approach to the use of noninvasive cardiovascular studies and of preventive interventions such as lipid lowering, aspirin, or further blood pressure reduction. It must be understood clearly that an abnormal noninvasive test result in an intermediate-risk, asymptomatic person should be interpreted as a predictor for a future cardiovascular event and not as a mandate for diagnosis of the presence or absence of angiographic CAD.[3]

Because the purpose of CACP screening is to detect subclinical atherosclerosis rather than severe stenoses, the data show that invasive procedures should be reserved for symptomatic patients with inducible ischemia. There is limited information showing benefit of revascularization in terms of prolongation or quality of life in asymptomatic patients.[134–136] To avoid inappropriate or unnecessary follow-up testing or invasive therapeutic procedures in patients who undergo EBCT or MDCT, the clinician should determine a priori that the goal of such noninvasive testing is to refine prognostic assessment and then employ, or not, well-proven preventive interventions based on test outcome.

Recommendation: It is not recommended to use CACP measure in asymptomatic persons to establish the presence of obstructive disease for subsequent revascularization (**Class III, Level of Evidence: C**).

3. Future Directions

3.1. Tracking Progression of Subclinical Atherosclerosis

A proposed use of CACP measurement is to track atherosclerotic changes over time using serial measurements. Before implementation, there are several important questions that need to be answered in regard to rescanning: What incremental change needs to occur between 2 scans for the clinician to be certain, with 95% confidence, that an apparent change is due to a change in the patient? Are there any data showing how often this actually occurs in patients who are reimaged after a year? These questions were answered by 2 studies of

patients with dual scans. The first was a trial of 1376 asymptomatic research participants,[58] not selected because they were at high risk for CAD, who were examined for the quantity of CAC with dual scan runs using EBCT. With these data, 95% limits of agreement were established and used to evaluate differences between scan runs performed approximately 3.5 years apart in 81 participants. Of those 81 participants, 59 (73%) had no apparent change in CAC between the 2 examinations, 21 (26%) had large increases suggesting progression of CAC, and 1 (1%) had a large decrease suggesting regression of CAC. Another study was conducted to develop a model for determining the smallest statistically significant change in the CAC score between serial measurements in a given subject.[137] The study consisted of 2217 pairs of repeated EBCT coronary calcium scans acquired in quick succession. The study evaluated the relationship between the interscan variability and the magnitude of the calcium score, formulating 95% repeatability coefficient equations for the Agatston and volumetric CAC scores. By examining repeatability of quantitative EBCT measurements of CAC as a function of the magnitude of the calcium score, Sevrukov and colleagues[137] developed a model to determine the smallest statistically significant change between serial measurements in a given subject.

Several studies have shown that serial EBCT scanning can be utilized to follow the evolution of CACP and aortic valve calcification.[138–142] Obviously, a noninvasive tool with which sequential testing could be performed safely and reliably would be highly desirable, and in this light, CACP could become a very useful marker of disease progression. There are a number of methodological considerations that are required for the evaluation of sequential imaging. A recent review of the relevant methodologies has recently been published by Taylor et al.[143] Currently, there are only 4 randomized, controlled trials evaluating CACP progression,[130,142,144,145] and these types of studies have not yet been reported with MDCT. The theoretical ability of statin therapy to slow or reverse CACP has demonstrated mixed results in the available literature, raising some doubts about using this tool for tracking progression of atherosclerosis.

There are only 4 published studies of outcomes related to CACP progression. The first study demonstrated, in 817 persons, that EBCT-measured progression was the strongest predictor of cardiac events.[146,147] This observational study suggests that continued accumulation of CACP in asymptomatic individuals is associated with increased risk of MI. A second study measured the change in CACP in 495 asymptomatic persons who underwent sequential EBCT scanning.[146] Statins were initiated in all patients after their initial EBCT scan. MI was reported in 49 patients during a follow-up of 3.2 ± 0.7 years. Interestingly, mean LDL level did not differ between patients experiencing an MI as compared with those who were event free (118 ± 25 mg/dL versus 122 ± 30 mg/dL, MI versus no MI).

On average, MI subjects demonstrated an annual rate of CACP change of $42\pm23\%$; event-free subjects showed a $17\pm25\%$ yearly change ($P=0.0001$). The associated relative risk for acute MI for patients exhibiting >15% CACP progression was elevated 17.2-fold (95% CI 4.1 to 71.2)

when compared with those without CACP progression ($P<0.0001$). In a Cox proportional hazard model, the follow-up score ($P=0.034$) as well as a score change >15% per year ($P<0.001$) were independent predictors of time to MI. Thus, from this and other reports, we have learned that the baseline score is a determinant of the rate of change even while it provides information for risk-assessment purposes. Patients with higher baseline scores generally exhibit more progression of CACP scores over time. Thus, the baseline score, rate of change, and also the patient's residual risk on the second scan are important determinants of the risk for future adverse cardiovascular events.

The CACP score increases by 15% to 20% annually, with greater increases being associated with increased incidence of MI.[52,146] A prospective study using EBCT to measure progression of CACP has just been reported. This prospective observational study evaluated 4613 asymptomatic persons 50 to 70 years of age with EBCT scanning of the coronary arteries at baseline and again at 2 years, with follow-up for 4.3 years.[114] The study demonstrated that the median (interquartile range) calcium score increased by 4 (95% CI 0 to 38) units from baseline to the year 2 scan in subjects who did not sustain a coronary event at any time during the study. In contrast, median (interquartile range) calcium scores increased by 247 (95% CI 40 to 471) units between the baseline and 2-year examinations in 49 subjects who experienced a first coronary disease event after the year 2 scan ($P<0.0001$). Multiple logistic regression demonstrated only age ($P<0.03$), male gender ($P<0.04$), LDL cholesterol ($P<0.01$), HDL cholesterol ($P<0.04$), and 2-year change in calcium score ($P<0.0001$) were significantly associated with subsequent CAD events. Increasing calcium scores were most strongly related to coronary events in this clinical study, similar to the results reported by observational studies.

However, effective treatment based on an increasing score in patients is still unclear. While several small observational studies indicated that vigorous cholesterol lowering retards the rate of progression of CACP,[138–141] a recently published, large randomized clinical trial showed that a combination of atorvastatin 20 mg, vitamin C, and vitamin E had no effect on progression of CACP at 4 years ($P=0.80$).[130] In this study, baseline coronary calcium score was higher in individuals who sustained ASCVD events (581) than in those who did not (361) ($P<0.0001$). The coronary calcium score also increased more from the baseline examination to the 2-year examination in those who subsequently experienced ASCVD events than in those who remained event free (256 ± 430 versus 120 ± 286, $P<0.01$).

In multivariable analysis, including standard CAD risk factors, C-reactive protein, and the baseline coronary calcium score, only the calcium score was significantly associated with disease events ($P<0.0001$). The change in calcium score, which was highly correlated with the baseline calcium score, did not predict events after adjustment for these variables. The failure of change in calcium score to predict ASCVD events seems to be a function of available statistical power, as the analysis was restricted to subjects who experienced a first event after the year 2 follow-up scan (n=34). Whether these results are unique to the drug combination

employed or would be observed with any cholesterol-lowering therapy is unknown. Continued progression of CACP appears to be an independent risk factor for future events, but future studies are needed. Despite this information, it is difficult to justify the incremental population exposure to radiation and the cost associated with a repeat CT test to assess "change" until it is better understood what therapies may be of benefit and how clinicians should utilize this data in clinical practice.

Several large observational studies, such as MESA (utilizing both EBCT and MDCT)[50] and RECALL (using EBCT),[51] are currently under way to also assess the prognostic value of increasing CACP burden in population-based samples. Genetic studies measuring calcified plaque with MDCT, such as the NHLBI's Family Heart Study-SCAN are also ongoing and will utilize the vascular calcium phenotype as a means of identifying genes related to atherosclerosis and CVD.

Recommendation: Serial imaging for assessment of progression of coronary calcification is not indicated at this time (**Class III, Level of Evidence: B**).

3.2. Hybrid Nuclear/CT Imaging

Currently available and an area of ongoing clinical research is the application of hybrid PET-CT and SPECT-CT scanners. This hybrid technology will allow for the acquisition of metabolic and/or perfusion information as well as anatomic data, including angiographic data and data on coronary calcification. Hybrid imaging currently remains a research tool with ongoing problems with image registration. Despite this, several recent reports using serial imaging have noted a high rate of coronary calcium in patients with normal perfusion SPECT. From the Berman series in 1119 patients with normal MPS, 20% had coronary calcium scores (CCS) in the range of 400 to 999 and 11% had CCS ≥1000.[6]

From a recent smaller series of 200 patients in whom SPECT was negative for ischemia, 17.5% of patients had CCS ≥100.[148] These data highlight the underlying, unaddressed risk faced by patients with normal SPECT results who nevertheless have a significant atherosclerotic disease burden. The future application of hybrid or serial imaging strategies will allow for a more precise delineation of anatomic and physiological components in a single test.

Two additional reports have been published on the use of hybrid scanning to assess cardiovascular risk or the present utility of SPECT imaging post-CT measurement of coronary calcium. The first report, on obstructive disease, was a large prospective series of 510 asymptomatic patients with type 2 diabetes. Patients with a calcium score >100 underwent SPECT imaging. A random sample of patients with a calcium score <100 also underwent SPECT. This report indicated that diabetic patients with a calcium score >100 had an increased frequency of abnormal perfusion defects. The rate of abnormal stress perfusion findings ranged from 23% to 71% for those with calcium scores >100 to >1000. These data are important, as they reveal that for individuals with diabetes a higher rate of abnormal SPECT findings is noted for a lower calcium score threshold of >100, as compared with >400 for an unselected patient series. Similar findings of calcium scores >100, associated with an elevated rate of perfusion abnormalities, were recently reported for patients with a family history of premature CHD.[90]

Recommendation: The incremental benefit of hybrid imaging strategies will need to be demonstrated before clinical implementation, as radiation exposure may be significant with dual nuclear/CT imaging. Therefore, hybrid nuclear/CT imaging is not recommended (**Class III, Level of Evidence: C**).

3.3. Contrast-Enhanced CT of the Coronary Arteries

When higher resolution image acquisition protocols (thinner slice collimation, higher x-ray tube current) are combined with intravenous injection of contrast agent, EBCT and MDCT permit visualization of the coronary artery lumen, coronary atherosclerotic plaque, and coronary stenoses. The small dimensions of coronary arteries, plaque, and stenoses make imaging by CT quite difficult. Also, the contrast that can be achieved between the vessel lumen, atherosclerotic plaque and vessel wall, and the surrounding structures is lower than that of coronary calcium versus the surrounding tissue. Thus, image acquisition protocols have to be tailored for maximum resolution, and image quality is, on the one hand, more critical but, on the other hand, not as stable as when coronary calcification alone is assessed.

Tremendous progress regarding spatial resolution, temporal resolution, and image noise has been made with the development from 4- to 16- and 64-row MDCT scanners, and their ability to visualize the coronary lumen and coronary atherosclerotic plaque has substantially improved over the past several years. This development is ongoing. At the moment, the use of EBCT, or MDCT equipment with at least 16 slices, submillimeter rotation speed, and rotation times below 500 ms has to be considered a prerequisite for contrast-enhanced coronary imaging since, in the published studies, data for this equipment were substantially more reliable than for previous scanner generations and because 16-detector scanners are now widely available (although no direct comparisons to previous scanner generations have been published).

3.3.1. Electron Beam CT

When EBCT is performed to visualize the coronary lumen, approximately 160 mL of contrast agent is injected intravenously. Atropine is sometimes used to increase the heart rate, since one image is acquired in each cardiac cycle and faster heart rates will thus decrease the overall scan and breathhold time. Sublingual nitrates are usually given to improve image quality. First comparisons between EBCT and invasive coronary angiography starting in the mid-1990s demonstrated the feasibility of stenosis detection.[149–161] Adequate patient selection, careful scan protocols, and careful evaluation of images resulted in a sensitivity to coronary artery stenoses in the proximal and mid segments of the coronary arteries of between 74% and 92%, with specificities of 71% to 95%. However, the limited spatial resolution and long scan time (requiring breathholds of up to 40 seconds) led to image artifacts.

In the early studies using 80% triggering, 11% to 35% of all coronary arteries had to be excluded from evaluation because of severe calcification or motion artifacts.[53] Early EBCT studies suffered from limited spatial resolution of the EBCT scanner (owing to fixed image collimation of 3.0 mm) and use of late diastolic triggering (80% of the R-R interval). More recent studies, with use of end-systolic triggering (where coronary motion is reduced) and 1.5-mm slice thickness, reduce the noninterpretability of coronary segments to 5% to 9% (the remaining noninterpretable segments are almost exclusively the result of dense calcifications) and reveal sensitivities of 90% to 91%, specificities of 93% to 94%, and high negative predictive values (≥96%).[160,161] Limitations that still exist with EBCT include the inability to increase the tube current (which leads to limitations concerning image noise), and the limited availability of the scanner has prevented more widespread evaluation and application of EBCT for detection of coronary stenoses.

3.3.2. Multidetector CT

Initial studies with 4-detector systems demonstrated the ability of mechanical CT scanners to visualize the coronary arteries.[162–173] However, spatial and temporal resolution were still limited and resulting artifacts precluded image evaluation regarding the presence of hemodynamically significant stenoses in a high percentage of cases (up to 32%). It was recognized that severe calcifications were the most frequent reason for impaired evaluability, owing to partial volume effects which are a consequence of limited spatial resolution. With the introduction of 16-detector systems that combined submillimeter collimation with faster gantry rotation times, image quality in coronary CT angiography became more stable. Several studies with inclusion of 22 to 149 individuals showed that with the further development of scanner technology, robustness and accuracy for detecting and ruling out hemodynamically relevant coronary artery stenoses increased substantially.[174–189] Sensitivities ranging from 72% to 98%, as well as specificities from 86% to 98%, have been reported for the detection of coronary artery stenosis (Table 8).

Studies are now being reported that use 64-detector MDCT. The increased collimation width and greater number of slices obtained allow for shorter examination times by reducing both the breathhold and contrast requirements. The acquisition speeds are not much faster than 16-detector scanners, with the fastest gantry rotation currently at 330 ms. Several single-center studies of 64-row MDCT results have been reported (Table 8).

Leschka et al[12] reported CT angiography in 67 patients with suspected CAD and compared the results with invasive coronary angiography. None of the coronary segments needed to be excluded from analysis. CT correctly identified all 20 patients having no significant stenosis on invasive angiography. Overall sensitivity for classifying stenoses was 94%, specificity was 97%, positive predictive value was 87%, and negative predictive value was 99%. Leber et al[190] studied 59 patients with stable angina pectoris. In 55 of 59 patients, 64-slice CT enabled the visualization of the entire coronary tree with diagnostic image quality. Sensitivity for the detection of stenosis <50%, stenosis >50%, and stenosis >75%

was 79%, 73%, and 80%, respectively, and specificity was 97%.

Raff et al[80] studied 70 consecutive patients undergoing elective invasive coronary angiography. Patients were excluded for atrial fibrillation. Specificity, sensitivity, and positive and negative predictive values for the presence of significant stenoses were by artery (n=279), 91%, 92%, 80%, and 97%, respectively; by patient (n=70), 95%, 90%, 93%, and 93%, respectively. Subset analysis confirms that patients with calcium scores >400, obesity (body mass index >30 kg/m^2), and heart rates >70 bpm remain a challenge to diagnose. Several additional studies confirmed sensitivities between 95% and 99% and specificities between 93% and 96% for the detection of coronary artery stenoses by 64-slice CT.[191–193]

For all MDCT scanner generations, including 64-row CT, it has been convincingly shown that low heart rates significantly improve image quality and evaluability.[194–197] In addition, the effectiveness of algorithms that modulate the x-ray tube current in synchronization with the patient's ECG to reduce the radiation exposure is higher for lower heart rates.[83,84] Therefore, low heart rates (preferably below 60 bpm) are desirable for MDCT imaging of the coronary arteries, and short-acting β-blockade is often used before scanning. Because of the need for retrospective gating for MDCT angiography, atrial fibrillation and other irregular heart rhythms remain a contraindication.

In a meta-analysis comparing CT angiography to magnetic resonance angiography (MRA),[198] a comparison of sensitivity revealed higher diagnostic accuracy for MDCT (weighted [by the proportional sample size] average: 82%, 95% CI 79% to 90%) when compared with MRA (weighted average: 75%, 95% CI 60% to 84%, $P=0.029$). In this meta-analysis, there was a significant difference that was also observed for the weighted specificity, which was 95% (95% CI 94% to 96%) for MDCT and 87% (95% CI 85% to 88%) for MRA ($P<0.05$). A significantly higher odds ratio (11.5-fold) for the presence of significant stenosis (≥50% diameter stenosis) was observed for MDCT as compared with MRA (6.6-fold) ($P<0.0001$). It is important to note that this report demonstrated improved specificity for MDCT, when compared with MRA, in populations with a lower disease prevalence ($P=0.022$).

Finally, a recent meta-analysis by Stein et al[199] reported the diagnostic accuracy by a patient and segmental analysis. Stein and colleagues also performed a subset analysis for CT results that were read while blinded to the invasive angiographic results. These authors noted that the average sensitivity and specificity values were 95% and 84% for 4-slice CT and increased to 100% for 64-slice CT. Additional analyses by these authors revealed a higher (on average) sensitivity for proximal (90%) stenosis when compared with distal (79%) segments. Diagnostic specificity values were ≥90% for proximal, mid, and distal segments. These authors also noted a high diagnostic accuracy for both 16-slice and 64-slice CT for detecting ≥50% stenosis in the left main coronary artery. Diagnostic sensitivity measurements were similarly high for 16-slice and 64-slice CT for detection of stenosis in the left anterior descending (16-slice=90%, 64-slice=95%), right

TABLE 8.   Results of Contrast-Enhanced EBCT and MDCT for the Detection of Coronary Stenoses

Author	Technology	n	Rate of Unevaluable Segments, %	Sensitivity, %	Specificity, %	Negative Predictive Value, %	Remarks
Reports using EBCT							
Nakanishi[150]	EBCT	37	…	74	95	93	Per-artery analysis
Schmermund[151]	EBCT	28	12	83	91	96	Per-segment analysis, mid and proximal segments
Reddy[152]	EBCT	23	…	88	79	94	Per-artery analysis
Rensing[153]	EBCT	37	19	77	94	95	Per-segment analysis, mid and proximal segments
Achenbach[154]	EBCT	125	25	92	94	98	Per-artery analysis, proximal and mid segments
Budoff[155]	EBCT	52	11	78	71	91	Per-artery analysis
Achenbach[156]	EBCT	36	20	92	91	92	Per-segment analysis, proximal and mid segments
Leber[157]	EBCT	87	24	87	93	97	Per-segment analysis, proximal and mid segments
Ropers[158]	EBCT	118	24	90	66	96	
Nikolaou[159]	EBCT	20	11	85	77	95	Per-segment analysis, proximal and mid segments
Lu[160]	EBCT	53	35	69	82	93	Per-artery analysis—80% trigger
Lu[161]	EBCT	80	9	91	94	97	Per-artery analysis—end-systolic trigger
Budoff[161]	EBCT	86	5	90	93	96	Per-artery analysis, 1.5-mm collimation
Reports using 4-slice CT							
Nieman[165]	4-Slice MDCT	31	27	81	97	97	Per-segment analysis, proximal and mid segments
Achenbach[166]	4-Slice MDCT	64	32	91	84	98	Per-artery analysis, all segments >2.0 mm
Knez[167]	4-Slice MDCT	42	6	78	98	97	Per-artery analysis, proximal and mid segments
Vogl[168]	4-Slice MDCT	64	…	75	99	…	Per-segment analysis, 15 segments
Kopp[169]	4-Slice MDCT	102	15	86–93	96–97	98–99	Per-segment analysis, 10 segments
Nieman[170]	4-Slice MDCT	53	30	82	93	97	Per-segment analysis, all segments >2.0 mm
Becker[171]	4-Slice MDCT	28	11	78	71	97	Per-segment analysis, 7 segments
Morgan-Hughes[172]	4-Slice MDCT	30	32	72	86	93	Per-segment analysis, proximal and mid segments
Sato[173]	4-Slice MDCT	54	5	94	97	97	Per-artery analysis, all segments >2.0 mm
Reports using 8-slice CT							
Maruyama[174]	8-Slice MDCT	25	26	73	73	96	Per-segment analysis, all segments
Reports using 16-slice CT							
Nieman[175]	16-Slice CT	59	7	95	86	97	Per-artery analysis, all segments >2.0 mm
Ropers[176]	16-Slice CT	77	12	93	92	97	Per-artery analysis, all segments >1.5 mm
Kuettner[177]	16-Slice CT	58	…	72–98	97–98	97–100	Per-segment analysis, all of 13 segments (in patients with Agatston score <1000)
Mollet[178]	16-Slice CT	128	…	92	95	98	Per-segment analysis, all segments >2.0 mm
Martuscelli[179]	16-Slice CT	64	16	89	98	98	Per-segment analysis, all segments >1.5 mm
Hoffmann[180]	16-Slice CT	33	…	63–89	95–96	96–97	Per-segment analysis, all of 17 segments (proximal and mid segments)
Kuettner[181]	16-Slice CT	72	7	82	98	97	Per-segment analysis, all of 13 segments
Mollet[182]	16-Slice CT	51	…	95	98	99	Per-segment analysis, all segments >1.5 mm
Schuijf[183]	16-Slice CT	45	6	98	97	100	Per-segment analysis, all segments
Morgan-Hughes[184]	16-Slice CT	58	2–37	83–89	97–98	97–99	Per-segment analysis, all of 15 segments (in patients with Agatston score <400)
Hoffmann[185]	16-Slice CT	103	6	95	98	99	Per-segment analysis, all segments >1.5 mm
Achenbach[189]	16-Slice CT	50	4	94	96	99	Per-segment analysis, all segments >1.5 mm
Aviram[188]	16-Slice CT	22	…	86	98	98	Per-segment analysis, all segments >1.5 mm
Kaiser[187]	16-Slice CT	149	23	30	91	83	Per-artery analysis, all segments
Fine[186]	16-Slice CT	50	2	87	97	98	Per-artery analysis, all segments >1.5 mm
Reports using 64-slice CT							
Leschka[12]	64-Slice CT	67	…	94	97	99	Per-segment analysis, all segments >1.5 mm
Raff[80]	64-Slice CT	70	12	86	95	98	Per-segment analysis, all segments
Leber[190]	64-Slice CT	59	…	73–88	97	99	Per-segment analysis, all segments
Mollet[191]	64-Slice CT	52	2	99	95	99	Per-segment analysis, all segments
Ropers[192]	64-Slice CT	82	4	95	93	99	Per-artery analysis, all segments >1.5 mm
Fine[193]	64-Slice CT	66	6	95	96	95	Per-artery analysis, all arteries >1.5 mm
Available meta-analysis							
Schuijf[198]	4-Slice CT	569	22	80	94	…	Per-segment analysis
	8-Slice CT	50	21	80	98	…	
	16-Slice CT	681	4	88	96	…	
	64-Slice CT	396	4	92	95	…	
Stein[199]	4-Slice CT	89	5–18	95	84	…	Per-patient (blinded) analysis
	16-Slice CT	448	0–21	95	84	…	
	64-Slice CT	67	0	100	100	…	

coronary artery (16-slice=91%, 64-slice=93%), and left circumflex coronary arteries (16-slice=82%, 64-slice=94%). Diagnostic specificity values were similar across the arteries but higher for 64-slice (range=92% to 100%) as compared with 16-slice CT (84% to 100%).

3.4. CT Angiography Applications in a Clinical Context

3.4.1. Suspected CAD
The studies that have evaluated the accuracy of EBCT and MDCT "coronary angiography" for the assessment of coronary artery stenoses have been relatively small (up to 149 individuals). They recruited somewhat selected patients (eg, excluding patients with acute coronary syndromes or atrial fibrillation), and all studies have been validated against invasive coronary angiography as a gold standard. No outcomes-based analyses that made further clinical management dependent on the EBCT or MDCT result have been published. However, all studies have convincingly demonstrated a very high negative predictive value of CT coronary angiography (see Table 8). Thus, a "normal" CT coronary angiogram allows the clinician to rule out the presence of hemodynamically relevant coronary artery stenoses with a high degree of reliability. When considering whether to refer a patient for EBCT or MDCT, clinicians must weigh the relative advantages of other testing methods such as exercise testing or stress imaging. The choice of testing will be determined by both local expertise in a given hospital as well as by the patient's specific clinical history. Functional information demonstrating the physiological significance of coronary lesions is still paramount for decision-making related to revascularization.

In a clinical context, the high negative predictive value may be useful for obviating the need for invasive coronary angiography in patients whose symptoms or abnormal stress test results make it necessary to rule out the presence of coronary artery stenoses. Especially if symptoms, age, and gender suggest a low to intermediate probability of hemodynamically relevant stenoses,[200] ruling out hemodynamically relevant stenoses by CT coronary angiography may be clinically useful and may help avoid invasive angiography. CT coronary angiography is reasonable for the assessment of obstructive disease in symptomatic patients **(Class IIa, Level of Evidence: B).**

Use of CT angiography in asymptomatic persons as a screening test for atherosclerosis (noncalcific plaque) is not recommended **(Class III, Level of Evidence: C).**

3.4.2. Follow-Up of Percutaneous Coronary Intervention
Several smaller studies have assessed the value of EBCT and MDCT to detect restenosis after stent placement. With EBCT, 4-detector MDCT, and 16-detector MDCT, artifacts caused by the stent material prevented, in many cases, adequate visualization of the coronary lumen within the stent. Thus, in-stent restenosis could not be reliably detected in most cases.[201–206] The ability to visualize in-stent restenosis depends on stent design and material, stent size, and scanner technology.[207–209] Thus, further studies may prove that a certain combination of stent type and scanner technology may

permit the detection of in-stent restenosis. In a first study performed by 64-slice CT, sensitivity for detection of in-stent restenosis was 83%, but only 8 stenoses were present in the overall study group.[210] Thus, based on current data, imaging of patients to follow up stent placement cannot be recommended **(Class III, Level of Evidence: C).**

3.4.3. Follow-Up After Bypass Surgery
Numerous studies have shown that EBCT and MDCT permit assessment of coronary bypass graft occlusion and patency with high accuracy. In most studies, the accuracy to detect bypass occlusion approached 100%.[211–226] Clinically, however, it might be reasonable in most cases to not only assess the patency of the bypass graft but also the presence of coronary stenoses in the course of the bypass graft or at the anastomotic site, as well as in the native coronary artery system **(Class IIb, Level of Evidence: C).** This is more difficult, owing to the smaller caliber of these vessels, the presence of artifacts caused by metal clips, and the often pronounced coronary calcification. Recent data suggest a high sensitivity for both coronary stenosis as well as assessment of bypass patency versus occlusion. A study of 52 patients using 16-detector MDCT demonstrated 99.4% assessibility of grafts, with a sensitivity and specificity of 100% (54/54) for occlusion and 96% sensitivity and 100% specificity for detecting high-grade stenoses in patent grafts.[227] Although more data are necessary, newer scanners may have the spatial resolution to overcome some of the earlier problems with graft assessment.

3.4.4. Anomalous Coronary Arteries
The presence of anomalous coronary arteries can be a differential diagnosis in patients with suspected coronary disease, chest pain, or syncope. The detailed assessment of anomalous coronary arteries concerning their origin and course can be difficult with invasive coronary angiography. The 3-dimensional nature of CT coronary angiography datasets allows for an exact analysis of anomalous coronary arteries. Both for EBCT and MDCT, numerous case reports suggest and several authors have investigated series of patients and could demonstrate that the analysis of coronary anomalies is straightforward and exact.[228–233] As opposed to magnetic resonance imaging, which also permits the analysis of coronary anomalies in tomographic images, CT requires radiation and a contrast agent. However, the high resolution of the datasets (permitting analysis even of small details) and the speed of image acquisition make it reasonable to use CT as one of the first-choice imaging modalities in the workup of known and suspected coronary anomalies **(Class IIa, Level of Evidence: C).**

3.5. Assessment of NCP
In addition to identifying lesions with significant luminal narrowing, there is also interest in visualizing and characterizing coronary artery plaques beyond the mere assessment of calcium. Some plaques may be at increased risk for erosion or rupture even when such lesions are not associated with a significant degree of luminal stenosis. These so-called unstable plaques are thought to play a role in the development of acute coronary ischemic events. It has been observed that

unstable plaques are generally higher in lipid content, and the use of cross-sectional imaging may be helpful in characterizing plaque composition.

Coronary angiography has traditionally served as the principal imaging modality to evaluate CAD. However, both necropsy and coronary intravascular ultrasound (IVUS) studies have consistently shown that angiographically "normal" coronary artery segments may contain a significant amount of atherosclerotic plaque and that coronary angiography consistently underestimates the amount of coronary atherosclerosis.[234,235] Furthermore, previous angiographic studies have shown that most MIs result from the rupture of a vulnerable plaque in the absence of a significant luminal stenosis. These rupture-prone plaques, which are 7 times more likely to cause disruption than the more severe, extensive plaques, are not visible on 2-dimensional x-ray angiography.[236,237]

Improved spatial and temporal image acquisition with submillimeter slice collimation has facilitated atherosclerotic plaque detection with MDCT. Plaque with density below the vessel contrast is defined as noncalcified plaque. Conversely, structures with densities above the adjacent vessel lumen are considered calcified.[238] Some studies have defined 3 levels of plaque: "soft" plaque, presumably lipid laden with lower densities, intermediate or presumably fibrous plaques, and calcific or high-density plaques.

Recent contrast-enhanced MDCT studies have shown that noninvasive scanning permits accurate detection and differentiation of coronary plaque when compared with IVUS. There have been 6 main studies reported comparing CT technology with IVUS in the detection of lipid-rich and fibrous atheroma, with both MDCT (4 studies) and EBCT (2 studies).[239–243]

Sensitivities for NCP (hypoechoic, lipid-rich) detection by MDCT ranged from 53% to 92%, with the sample size ranging from 14 to 37 patients. In one of the more robust MDCT studies, which evaluated 875 segments, sensitivity for hypoechoic, hyperechoic, and calcific plaques was 78%, 78%, and 95%, respectively. Specificity was a respectable 92%.[239] As expected, the sensitivities for detecting calcific atheroma were relatively higher than for noncalcific plaque in these studies: approximately 88% to 95%. Although the sample sizes are relatively small, they do demonstrate diag-

nostic accuracy in characterizing noncalcific atheroma, with some difficulty differentiating lipid-laden and fibrous components.

Quantification of coronary atherosclerotic plaque burden by CT technology is currently unsatisfactory.[244] In the study by Achenbach et al,[239] MDCT substantially underestimated plaque volume per segment as compared with IVUS (24 ± 35 mm^3 versus 43 ± 60 mm^3, $P<0.001$).

In another comparison between MDCT and IVUS, plaque areas showed moderate correlation ($r=0.55$) between the 2 methods, with a significant tendency toward overestimation by MDCT (8.3 ± 4.8 mm^3 versus 7.3 ± 3.1 mm^3, $P<0.001$).[245] The limitations of NCP detection may be much more significant than a limited sensitivity or underestimation of plaque burden. The reproducibility of the measure has not been reported. There is no prognostic information to determine whether NCP adds any information on top of risk factors, angiographic disease severity, or calcified plaque, and it is not recommended **(Class III, Level of Evidence: C).** Finally, this procedure requires both contrast administration and radiation exposure, and the risks may outweigh the benefit in individual patients. All of this will need to be studied before NCP detection by CT becomes a clinical tool.

Conclusion

EBCT has undergone a 20-year period of testing for reliability and validity and is now established as a useful technique in identifying individuals with or at risk for CHD. MDCT is a promising tool for coronary calcium scoring while additional studies evaluating progression, reproducibility, and outcomes are currently under way. Radiation doses, reproducibility, and validation studies must be taken into account when choosing a cardiac CT study. Serial coronary calcium scans to noninvasively assess progression rates of coronary calcium and CT angiography to assess NCP are now starting to be reported, but the data are premature at this time. The most promising use of these technologies is calcium scoring for risk assessment of the asymptomatic individual, whereby elevated calcium scores may trigger more vigorous application of both lifestyle and/or pharmacological therapies targeted to lower cardiovascular risk and CT angiography to rule out the presence of coronary stenoses in certain subsets of symptomatic patients.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Matthew J. Budoff	Harbor-UCLA Medical Center	None	None	General Electric	None	None	None
Stephan Achenbach	University of Erlangen, Germany	Siemens	None	None	None	None	None
Roger S. Blumenthal	Johns Hopkins Hospital	Merck, Pfizer, Wyeth, AstraZeneca, KOS	None	Merck, Pfizer, Wyeth, AstraZeneca, KOS	None	None	None
J. Jeffrey Carr	Wake Forest University	None	None	None	None	None	None
Jonathan G. Goldin	UCLA	(1) Multi-Ethnic Study of Atherosclerosis (MESA) NIH: N01-HC-95160. Role: Co-Principal Investigator (2) Multi-Ethnic Study of Atherosclerosis (MESA) Family Study NIH: 1 R01-HL-071051. Role: Investigator	None	None	None	Siemens Medical Systems Role: Advisory Board	None
Philip Greenland	Northwestern University	None	None	None	None	None	None
Alan D. Guerci	St. Francis Hospital	None	None	None	None	None	None
Joao A.C. Lima	Johns Hopkins Hospital	Toshiba, Merck	None	Novartis, Toshiba, General Electric	None	General Electric	None
Daniel J. Rader	University of Pennsylvania	None	None	None	None	None	None
Geoffrey D. Rubin	Stanford University	None	None	None	None	None	None
Leslee J. Shaw	Cedars-Sinai Medical Center	General Electric (on pharmaceutical side, not CT)	None	None	None	None	None
Susan E. Wiegers	University of Pennsylvania	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Kenneth L. Baughman	Brigham and Women's Hospital	None	None	None	None	None	None
Melvin Clouse	Harvard Medical Faculty Physicians—Beth Israel Deaconess Hospital	Core 64 Sponsor-Toshiba	National Heart, Lung, and Blood Institute	Spiral CT conferences in SnowMass, Colo, and Orlando, Fla	None	None	None
Gregg C. Fonarow	University of California, Los Angeles	GlaxoSmithKline, Medtronic, Johnson&Johnson	Pfizer	Pfizer, Bristol-Myers Squibb, Merck, Schering-Plough, KOS, GlaxoSmithKline, Medtronic, Johnson&Johnson	None	Pfizer, Bristol-Myers Squibb, Merck, Schering-Plough, GlaxoSmithKline, Johnson&Johnson, Guidant	None
Harvey Hecht	Lenox Hill Hospital	Philips	None	None	None	None	None
Thomas Lee	Partners Healthcare System	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit.

References

1. 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;291:210–215.
2. Smith SC, Greenland P, Grundy SM. AHA conference proceedings. Prevention Conference V: beyond secondary prevention: identifying the high-risk patient for primary prevention. Executive summary. *Circulation.* 2000;101:111–116.
3. National Institutes of Health, National Heart, Lung, and Blood Institute. *Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Final Report.* NIH Publication No. 02-5215. Washington DC: US Department of Health and Human Services; 2002.
4. O'Rourke RA, Brundage BH, Froelicher VF, Greenland P, Grundy SM, Hachamovitch R, Pohost GM, Shaw LJ, Weintraub WS, Winters WL Jr. American College of Cardiology/American Heart Association expert consensus document on electron-beam computed tomography for the diagnosis and prognosis of coronary artery disease. *Circulation.* 2000; 102:126–140.
5. Mieres JH, Shaw LJ, Arai A, Budoff MJ, Flamm SD, Hundley WG, Marwick TH, Mosca L, Patel AR, Quinones MA, Redberg RF, Taubert KA, Taylor AJ, Thomas GS, Wenger NK; Cardiac Imaging Committee, Council on Clinical Cardiology, and the Cardiovascular Imaging and

Intervention Committee, Council on Cardiovascular Radiology and Intervention, American Heart Association. Role of noninvasive testing in the clinical evaluation of women with suspected coronary artery disease: consensus statement from the Cardiac Imaging Committee, Council on Clinical Cardiology, and the Cardiovascular Imaging and Intervention Committee, Council on Cardiovascular Radiology and Intervention, American Heart Association. *Circulation*. 2005;111: 682–696.

6. Berman DS, Wong ND, Gransar H, Miranda-Peats R, Dahlbeck J, Hayes SW, Friedman JD, Kang X, Polk D, Hachamovitch R, Shaw L, Rozanski A. Relationship between stress-induced myocardial ischemia and atherosclerosis measured by coronary calcium tomography. *J Am Coll Cardiol*. 2004;44:923–930.

7. Shaw LJ, Berman DS, Bax JJ, Brown KA, Cohen MC, Hendel RC, Mahmarian JJ, Williams KA, Ziffer JA. Computed tomographic imaging within nuclear cardiology [information statement]. *J Nucl Cardiol*. 2005;12:131–142.

8. Brindis RG, Douglas PS, Hendel RC, Peterson ED, Wolk MJ, Allen JM, Patel MR, Raskin IE, Bateman TM, Cerqueira MD, Gibbons RJ, Gillam LD, Gillespie JA, Iskandrian AE, Jerome SD, Krumholz HM, Messer JV, Spertus JA, Stowers SA; American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American Society of Nuclear Cardiology; American Heart Association. ACCF/ASNC appropriateness criteria for single-photon emission computed tomography myocardial perfusion imaging (SPECT MPI): a report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group and the American Society of Nuclear Cardiology, endorsed by the American Heart Association [published correction appears in *J Am Coll Cardiol*. 2005;46:2148–2150]. *J Am Coll Cardiol*. 2005;46:1587–1605.

9. Morin RL, Gerber TC, McCollough CH. Radiation dose in computed tomography of the heart. *Circulation*. 2003;107:917–922.

10. Hunold P, Vogt FM, Schmermund A, Debatin JF, Kerkhoff G, Budde T, Erbel R, Ewen K, Barkhausen J. Radiation exposure during cardiac CT: effective doses at multi-detector row CT and electron-beam CT. *Radiology*. 2003;226:145–152.

11. Nasir K, Budoff MJ, Post WS, Fishman EK, Mahesh M, Lima JA, Blumenthal RS. Electron beam CT versus helical CT scans of coronary arteries: current utility and future directions. *Am Heart J*. 2003;146: 949–977.

12. Leschka S, Alkadhi H, Plass A, Desbiolles L, Grunenfelder J, Marincek B, Wildermuth S. Accuracy of MSCT coronary angiography with 64-slice technology: first experience. *Eur Heart J*. 2005;26:1482–1487.

13. Budoff MJ, Cohen MC, Garcia M, Hodgson JMcB, Hundley WG, Lima JA, Manning WJ, Pohost GM, Raggi PM, Rodgers GP, Rumberger JA, Taylor AJ, Creager MA, Hirshfeld JW Jr, Lorell BH, Merli G, Rodgers GP, Tracy CM, Weitz HH; American College of Cardiology Foundation; American Heart Association; American College of Physicians Task Force on Clinical Competence and Training; American Society of Echocardiography; American Society of Nuclear Cardiology; Society of Atherosclerosis Imaging; Society for Cardiovascular Angiography and Interventions. ACCF/AHA clinical competence statement on cardiac imaging with computed tomography and magnetic resonance: a report of the American College of Cardiology Foundation/American Heart Association/American College of Physicians Task Force on Clinical Competence and Training. *J Am Coll Cardiol*. 2005;46:383–402.

14. Wexler L, Brundage B, Crouse J, Detrano R, Fuster V, Maddahi J, Rumberger J, Stanford W, White R, Taubert K. Coronary artery calcification: pathophysiology, epidemiology, imaging methods, and clinical implications: a statement for health professionals from the American Heart Association. *Circulation*. 1996;94:1175–1192.

15. Bostrom K, Watson KE, Horn S, Wortham C, Herman IM, Demer LL. Bone morphogenetic protein expression in human atherosclerotic lesions. *J Clin Invest*. 1993;91:1800–1809.

16. Hirota S, Imakita M, Kohri K, Ito A, Morii E, Adachi S, Kim HM, Kitamura Y, Yutani C, Nomura S. Expression of osteopontin messenger RNA by macrophages in atherosclerotic plaques: a possible association with calcification. *Am J Pathol*. 1993;143:1003–1008.

17. Shanahan CM, Cary NR, Metcalfe JC, Weissberg PL. High expression of genes for calcification-regulating proteins in human atherosclerotic plaque. *J Clin Invest*. 1994;93:2393–2402.

18. Faber A. Die Arteriosklerose. In: Aschoff L, et al, eds. *Pathologische Anatomie; ein lehrbuch für studierende und ärzte*. Jena, Germany: G. Fischer; 1912:49–56.

19. Blankenhorn DH, Stern D. Calcification of the coronary arteries. *Am J Roentgenol Radium Ther Nucl Med*. 1959;81:772–777.

20. US Preventive Services Task Force. Screening for Coronary Heart Disease. Available at: http://www.ahcpr.gov/clinic/uspstf/uspsacad.htm. Accessed July 20, 2006.

21. Budoff MJ, Georgiou D, Brody A, Agatston AS, Kennedy J, Wolfkiel C, Stanford W, Shields P, Lewis RJ, Janowitz WR, Rich S, Brundage BH. Ultrafast computed tomography as a diagnostic modality in the detection of coronary artery disease: a multicenter study. *Circulation*. 1996;93: 898–904.

22. Budoff MJ, Diamond GA, Raggi P, Arad Y, Guerci AD, Callister TQ, Berman D. Continuous probabilistic prediction of angiographically significant coronary artery disease using electron beam tomography. *Circulation*. 2002;105:1791–1796.

23. Haberl R, Becker A, Leber A, Knez A, Becker C, Lang C, Bruning R, Reiser M, Steinbeck G. Correlation of coronary calcification and angiographically documented stenoses in patients with suspected coronary artery disease: results of 1,764 patients. *J Am Coll Cardiol*. 2001;37: 451–457.

24. Hoff JA, Chomka EV, Krainik AJ, Daviglus M, Rich S, Kondos GT. Age and gender distributions of coronary artery calcium detected by electron beam tomography in 35,246 adults. *Am J Cardiol*. 2001;87: 1335–1339.

25. Uretsky B, Rifkin R, Sharma S, Reddy PS. Value of fluoroscopy in the detection of coronary stenosis: influence of age, sex, and number of vessels calcified on diagnostic efficacy. *Am Heart J*. 1988;115:323–333.

26. Sangiorgi G, Rumberger JA, Severson A, Edwards WD, Gregoire J, Fitzpatrick LA, Schwartz RS. Arterial calcification and not lumen stenosis is highly correlated with atherosclerotic plaque burden in humans: a histologic study of 723 coronary artery segments using nondecalcifying methodology. *J Am Coll Cardiol*. 1998;31:126–133.

27. Simons DB, Schwartz RS, Edwards WD, Sheedy PF, Breen JF, Rumberger JA. Noninvasive definition of anatomic coronary artery disease by ultrafast computed tomographic scanning: a quantitative pathologic comparison study. *J Am Coll Cardiol*. 1992;20:1118–1126.

28. Rumberger JA, Schwartz RS, Simons DB, Sheedy PF 3rd, Edwards WD, Fitzpatrick LA. Relation of coronary calcium determined by electron beam computed tomography and lumen narrowing determined by autopsy. *Am J Cardiol*. 1994;73:1169–1173.

29. Rumberger JA, Simons DB, Fitzpatrick LA, Sheedy PF, Schwartz RS. Coronary artery calcium area by electron-beam computed tomography and coronary atherosclerotic plaque area: a histopathologic correlative study. *Circulation*. 1995;92:2157–2162.

30. Baumgart D, Schmermund A, Goerge G, Haude M, Ge J, Adamzik M, Sehnert C, Altmaier K, Groenemeyer D, Seibel R, Erbel R. Comparison of electron beam computed tomography with intracoronary ultrasound and coronary angiography for detection of coronary atherosclerosis. *J Am Coll Cardiol*. 1997;30:57–64.

31. Schmermund A, Baumgart D, Gorge G, Seibel R, Gronemeyer D, Ge J, Haude M, Rumberger J, Erbel R. Coronary artery calcium in acute coronary syndromes: a comparative study of electron-beam computed tomography, coronary angiography, and intracoronary ultrasound in survivors of acute myocardial infarction and unstable angina. *Circulation*. 1997;96:1461–1469.

32. Janowitz WR, Agatston AS, Kaplan G, Viamonte M Jr. Differences in prevalence and extent of coronary artery calcium detected by ultrafast computed tomography in asymptomatic men and women. *Am J Cardiol*. 1993;72:247–254.

33. Rumberger JA, Sheedy PF, Breen JF. Use of ultrafast (cine) x-ray computed tomography in cardiac and cardiovascular imaging. In: Giuliani ER, Gersh BJ, McGoon MD, Hayes DL, Schaff HF, eds. *Mayo Clinic Practice of Cardiology*. 3rd ed. St. Louis, Mo: Mosby; 1996: 303–324.

34. Bell MR, Lerman LO, Rumberger JA. Validation of minimally invasive measurement of myocardial perfusion using electron beam computed tomography and application in human volunteers. *Heart*. 1999;81: 628–635.

35. Budoff MJ, Shinbane JS, Oudiz RJ, Child J, Carson S, Chau A, Tseng P, Gao Y, Mao S. Comparison of coronary artery calcium screening image quality between C-150 and e-Speed electron beam scanners. *Acad Radiol*. 2005;12:309–312.

36. Rumberger JA, Brundage BH, Rader DJ, Kondos G. Electron beam computed tomographic coronary calcium scanning: a review and guidelines for use in asymptomatic individuals [published correction in *Mayo Clinic Proc*. 1999;74:538]. *Mayo Clinic Proc*. 1999;74:243–252.

37. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol*. 1990;15:827–832.

38. Mao S, Lu B, Oudiz RJ, Bakhsheshi H, Liu SC, Budoff MJ. Coronary artery motion in electron beam tomography. *J Comput Assist Tomogr*. 2000;24:253–258.

39. Mao SS, Bakhsheshi H, Lu B, Liu SC, Oudiz RJ, Budoff MJ. Effect of electrocardiogram triggering on reproducibility of coronary artery calcium scoring. *Radiology*. 2001;220:707–711.

40. Stary HC, Chandler AB, Dinsmore RE, Fuster V, Glagov S, Insull W Jr, Josenfeld ME, Schwartz CJ, Wagner WD, Wissler RW. A definition of advanced types of atherosclerotic lesions and a histological classification of atherosclerosis: a report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. *Circulation*. 1995;92:1355–1374.

41. Callister TQ, Cooil B, Raya SP, Lippolis NJ, Russo DJ, Raggi P. Coronary artery disease: improved reproducibility of calcium scoring with electron-beam CT volumetric method. *Radiology*. 1998;208:807–814.

42. Ferencik M, Ferullo A, Achenbach S, Abbara S, Chan RC, Booth SL, Brady TJ, Hoffmann U. Coronary calcium quantification using various calibration phantoms and scoring thresholds. *Invest Radiol*. 2003;38:559–566.

43. Hong C, Becker CR, Schoepf UJ, Ohnesorge B, Bruening R, Reiser MF. Coronary artery calcium: absolute quantification in nonenhanced and contrast-enhanced multi-detector row CT studies. *Radiology*. 2002;223:474–480.

44. Rumberger JA, Kaufman L. A rosetta stone for coronary calcium risk stratification: agatston, volume and mass scores in 11,490 individuals. *AJR Am J Roentgenol*. 2003;181:743–748.

45. Ritchie CJ, Godwin JD, Crawford CR, Stanford W, Anno H, Kim Y. Minimum scan speeds for suppression of motion artifacts in CT. *Radiology*. 1992;185:37–42.

46. Becker CR, Kleffel T, Crispin A, Knez A, Young J, Schoepf UJ, Haberl R, Reiser MF. Coronary artery calcium measurement: agreement of multirow detector and electron beam CT. *AJR Am J Roentgenol*. 2001;176:1295–1298.

47. Knez A, Becker CR, Becker A, Leber A, White C, Reiser M, Steinbeck G. Determination of coronary calcium with multi-slice spiral computed tomography: a comparative study with electron-beam CT. *Int J Cardiovasc Imaging*. 2002;18:295–303.

48. Budoff MJ, Mao S, Zalace CP, Bakhsheshi H, Oudiz RJ. Comparison of spiral and electron beam tomography in the evaluation of coronary calcification in asymptomatic persons. *Int J Cardiol*. 2001;77:181–188.

49. Carr JJ, Crouse JR, D'Agostino RB, Peterson NP, Burke GL. Evaluation of a subsecond gated helical CT for quantification of coronary artery calcium and comparison with electron beam CT. *AJR Am J Roentgenol*. 2000;174:915–921.

50. Bild DE, Bluemke DA, Burke GL, Detrano R, Diez Roux AV, Folsom AR, Greenland P, Jacob DR Jr, Kronmal R, Liu K, Nelson JC, O'Leary D, Saad MF, Shea S, Szklo M, Tracy RP. Multi-ethnic study of atherosclerosis: objectives and design. *Am J Epidemiol*. 2002;156:871–881.

51. Schmermund A, Mohlenkamp S, Stang A, et al. Assessment of clinically silent atherosclerotic disease and established and novel risk factors for predicting myocardial infarction and cardiac death in healthy middle-aged subjects: rationale and design of the Heinz Nixdorf RECALL Study: risk factors, evaluation of coronary calcium and lifestyle. *Am Heart J*. 2002;144:212–218.

52. Budoff MJ, Raggi P. Coronary artery disease progression assessed by electron-beam computed tomography. *Am J Cardiol*. 2001;88(2A):46E–50E.

53. Mao S, Budoff MJ, Bin L, Liu SC. Optimal ECG trigger point in electron-beam volumetric studies: three methods for minimizing motion artifacts. *Acad Rad*. 2001;8:1107–1115.

54. Lu B, Mao SS, Zhuang N, Bakhsheshi H, Yamamoto H, Takasu J, Liu SC, Budoff MJ. Coronary artery motion during the cardiac cycle and optimal ECG triggering for coronary artery imaging. *Invest Radiol*. 2001;36:250–256.

55. Callister TQ, Cooil B, Raya SP, Lippolis NJ, Russo DJ, Raggi P. Coronary artery disease: improved reproducibility of calcium scoring with electron-beam CT volumetric method. *Radiology*. 1998;208:807–814.

56. Mao SS, Budoff MJ, Bakhsheshi H, Liu SCK. Improved reproducibility of coronary artery calcium scoring by electron beam tomography with a new ECG trigger method. *Invest Radiol*. 2001;36:363–367.

57. Lu B, Zhuang N, Mao SS, Child J, Carson S, Bakhsheshi H, Budoff MJ. EKG-triggered CT data acquisition to reduce variability in coronary arterial calcium score. *Radiology*. 2002;224:838–844.

58. Bielak LF, Sheedy PF, Peyser PA. Coronary artery calcification measured at electron-beam CT: agreement in dual scan runs and change over time. *Radiology*. 2001;218:224–229.

59. Achenbach S, Ropers D, Mohlenkamp S, Schmermund A, Muschiol G, Groth J, Kusus M, Regenfus M, Daniel WG, Erbel R, Moshage W. Variability of repeated coronary calcium measurements by electron beam tomography. *Am J Cardiol*. 2001;87:210–213,A8.

60. Shemesh J, Tenenbaum A, Kopecky KK, Apter S, Rozenman J, Itzchak Y, Motro M. Coronary calcium measurements by double helical computed tomography: using the average instead of peak density algorithm improves reproducibility. *Invest Radiol*. 1997;32:503–506.

61. Qanadli SD, Mesurolle B, Aegerter P, Joseph T, Oliva VL, Guertin MC, Dubourq O, Fauchet M, Goeau-Brissonniere OA, Lacombe P. Volumetric quantification of coronary artery calcifications using dual-slice spiral CT scanner: improved reproducibility of measurements with 180 degrees linear interpolation algorithm. *J Comput Assist Tomogr*. 2001;25:278–286.

62. Mahnken AH, Wildberger JE, Sinha AM, Flohr T, Truong HT, Krombach GA, Gunther RW. Variation of the coronary calcium score depending on image reconstruction interval and scoring algorithm. *Invest Radiol*. 2002;37:496–502.

63. Daniell AL, Wong ND, Friedman JD, et al. Reproducibility of coronary calcium measurements from multidetector computed tomography. *J Am Coll Cardiol*. 2003;41(suppl A):456–457.

64. Hong C, Bae KT, Pilgram TK. Coronary artery calcium: accuracy and reproducibility of measurements with multi-detector row CT-assessment of effects of different thresholds and quantification methods. *Radiology*. 2003;227:795–801.

65. Van Hoe LR, De Meerleer KG, Leyman PP, Vanhoenacker PK. Coronary artery calcium scoring using ECG-gated multidetector CT: effect of individually optimized image-reconstruction windows on image quality and measurement reproducibility. *AJR Am J Roentgenol*. 2003;181:1093–1100.

66. Takahashi N, Bae KT. Quantification of coronary artery calcium with multi-detector row CT: assessing interscan variability with different tube currents pilot study. *Radiology*. 2003;228:101–106.

67. Carr JJ, Nelson JC, Wong ND, McNitt-Gray M, Arad Y, Jacobs DR Jr, Sidney S, Bild DE, Williams OD, Detrano RC. Calcified coronary artery plaque measurement with cardiac CT in population-based studies: standardized protocol of Multi-Ethnic Study of Atherosclerosis (MESA) and Coronary Artery Risk Development in Young Adults (CARDIA) study. *Radiology*. 2005;234:35–43.

68. Detrano RC, Anderson M, Nelson J, Wong ND, Carr JJ, McNitt-Gray M, Bild DE. Coronary calcium measurements: effect of CT scanner type and calcium measure on rescan reproducibility–MESA study. *Radiology*. 2005;236:477–484.

69. Ohnesorge B, Flohr T, Fischbach R, Kopp AF, Knez A, Schroder S, Schopf UJ, Drispin A, Lotz E, Reiser MF, Becker CR. Reproducibility of coronary calcium quantification in repeat examinations with retrospectively ECG-gated multisection spiral CT. *Eur Radiol*. 2002;12:1532–1540.

70. Schlosser T, Hunold P, Schmermund A, Kuhl H, Waltering KU, Debatin JF, Barkhausen J. Coronary artery calcium score: influence of reconstruction interval at 16-detector row CT with retrospective electrocardiographic gating. *Radiology*. 2004;233:586–589.

71. Achenbach S, Daniel WG, Moshage W. Recommendations for standardization of EBT and MSCT scanning. *Herz*. 2001;26:273–277.

72. US Food and Drug Administration, Center for Devices and Radiological Health. Whole body scanning using computed tomography (CT): what are the radiation risks from CT? Available at: http://www.fda.gov/cdrh/ct/risks.html. Accessed July 20, 2006.

73. Cohnen M, Poll L, Puttmann C, Ewen K, Modder U. Radiation exposure in multi-slice CT of the heart [published correction in *Rofo*. 2001; 173:521]. *Rofo*. 2001;178:295–299.

74. Thomton FJ, Paulson EK, Yoshizumi TT, Frush DP, Nelson RC. Single versus multi-detector row CT: comparison of radiation doses and dose profiles. *Acad Radiol*. 2003;10:379–385.

75. Mahnken AH, Wildberger JE, Simon J, Koos R, Flohr TG, Schaller S, Gunther RW. Detection of coronary calcifications: feasibility of dose reduction with a body weight-adapted examination protocol. *AJR Am J Roentgenol*. 2003;181:533–538.

76. Budoff MJ, Achenbach S, Duerinckx A. Clinical utility of computed tomography and magnetic resonance techniques for noninvasive coronary angiography. *J Am Coll Cardiol*. 2003;42:1867–1878.

77. Flohr TG, Schoepf UJ, Kuettner A, Halliburton S, Bruder H, Suess C, Schmidt B, Hofmann L, Yucel EK, Schaller S, Ohnesorge BM. Advances in cardiac imaging with 16-section CT systems. *Acad Radiol*. 2003;10:386–401.

78. Trabold T, Buchgeister M, Kuttner A, Heuschmid M, Kopp AF, Schroder S, Claussen CD. Estimation of radiation exposure in 16-detector row computed tomography of the heart with retrospective ECG-gating. *Rofo*. 2003;175:1051–1055.

79. Bae KT, Hong C, Whiting BR. Radiation dose in multidetector row computed tomography cardiac imaging. *J Magn Reson Imaging*. 2004;19:859–863.

80. Raff GL, Gallagher MJ, O'Neill WW, Goldstein JA. Diagnostic accuracy of noninvasive coronary angiography using 64-slice spiral computed tomography. *J Am Coll Cardiol*. 2005;46:552–557.

81. Gomez-Palacios M, Terron JA, Dominguez P, Vera DR, Osuna RF. Radiation doses in the surroundings of patients undergoing nuclear medicine diagnostic studies. *Health Phys*. 2005;89(2 suppl):S27–S34.

82. Picano E. Economic and biological costs of cardiac imaging. *Cardiovasc Ultrasound*. 2005;3:13.

83. Poll LW, Cohnen M, Brachten S, Ewen K, Modder U. Dose reduction in multi-slice CT of the heart by use of ECG-controlled tube current modulation ("ECG pulsing"): phantom measurements. *Rofo*. 2002;174:1500–1505.

84. Jakobs TF, Becker CR, Ohnesorge B, Flohr T, Suess C, Schoepf UJ, Reiser MF. Multislice helical CT of the heart with retrospective ECG gating: reduction of radiation exposure by ECG-controlled tube current modulation. *Eur Radiol*. 2002;12:1081–1086.

85. Fleischmann KE, Hunink MG, Kuntz KM, Douglas PS. Exercise echocardiography or exercise SPECT imaging? A meta-analysis of diagnostic test performance. *J Nucl Cardiol*. 2002;9:133–134.

86. Klocke FJ, Baird MG, Lorell BH, Bateman TM, Messer JV, Berman DS, O'Gara PT, Carabello BA, Russell RO Jr, Cerqueira MD, St John Sutton MG, DeMaria AN, Udelson JE, Kennedy JW, Verani MS, Williams KA, Antman EM, Smith SC Jr, Alpert JS, Gregoratos G, Anderson JL, Hiratzka LF, Faxon DP, Hunt SA, Fuster V, Jacobs AK, Gibbons RJ, Russell RO; American College of Cardiology; American Heart Association; American Society for Nuclear Cardiology. ACC/AHA/ASNC guidelines for the clinical use of cardiac radionuclide imaging–executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASNC Committee to Revise the 1995 Guidelines for the Clinical Use of Cardiac Radionuclide Imaging). *J Am Coll Cardiol*. 2003;42:1318–1333.

87. Schmermund A, Denktas AE, Rumberger JA, Christian TF, Sheedy PF 2nd, Bailey KR, Schwartz RS. Independent and incremental value of coronary artery calcium for predicting the extent of angiographic coronary artery disease: comparison with cardiac risk factors and radionuclide perfusion imaging. *J Am Coll Cardiol*. 1999;34:777–786.

88. Shavelle DM, Budoff MJ, Lamont DH, Shavelle RM, Kennedy JM, Brundage BH. Exercise testing and electron beam computed tomography in the evaluation of coronary artery disease. *J Am Coll Cardiol*. 2000;36:32–38.

89. Knez A, Becker A, Leber A, White C, Becker CR, Reiser MF, Steinbeck G, Boekstegers P. Relation of coronary calcium scores by electron beam tomography to obstructive disease in 2,115 symptomatic patients. *Am J Cardiol*. 2004;93:1150–1152.

90. Blumenthal RS, Becker DM, Yanek LR, Moy TF, Michos ED, Fishman EK, Becker LC. Comparison of coronary calcium and stress myocardial perfusion imaging in apparently healthy siblings of individuals with premature coronary artery disease. *Am J Cardiol*. 2006;97:328–333.

91. Anand DV, Lim E, Hopkins D, Corder R, Shaw LJ, Sharp P, Lipkin D, Lahiri A. Risk stratification in uncomplicated type 2 diabetes: prospective evaluation of the combined use of coronary artery calcium imaging and selective myocardial perfusion scintigraphy. *Eur Heart J*. 2006;27:713–721.

92. Shaw LJ, Raggi P, Callister TQ, Berman DS. Prognostic value of coronary artery calcium screening in asymptomatic smokers and non-smokers. *Eur Heart J*. 2006;27:968–975.

93. Budoff MJ, Shavelle DM, Lamont DH, Kim HT, Akinwale P, Kennedy JM, Brundage BH. Usefulness of electron beam computed tomography scanning for distinguishing ischemic from nonischemic cardiomyopathy. *J Am Coll Cardiol*. 1998;32:1173–1178.

94. Shemesh J, Tenenbaum A, Fisman EZ, Har-Zahav Y, Rath S, Apter S, Itzchak Y, Motro M. Coronary calcium as a reliable tool for differentiating ischemic from nonischemic cardiomyopathy. *Am J Cardiol*. 1996;77:191–194.

95. Le T, Ko JY, Kim HT, Akinwale P, Budoff MJ. Comparison of echocardiography and electron beam tomography in differentiating the etiology of heart failure. *Clin Cardiol*. 2000;23:417–420.

96. Budoff MJ, Jacob B, Rasouli ML, Yu D, Chang RS, Shavelle DM. Comparison of electron beam computed tomography and technetium stress testing in differentiating cause of dilated versus ischemic cardiomyopathy. *J Comput Assist Tomogr*. 2005;29:699–703.

97. Laudon DA, Vukov LF, Breen JF, Rumberger JA, Wollan PC, Sheedy PF 2nd. Use of electron-beam computed tomography in the evaluation of chest pain patients in the emergency department. *Ann Emerg Med*. 1999;33:15–21.

98. Georgiou D, Budoff MJ, Kaufer E, Kennedy JM, Lu B, Brundage BH. Screening patients with chest pain in the emergency department using electron beam tomography: a follow-up study. *J Am Coll Cardiol*. 2001;38:105–110.

99. McLaughlin VV, Balogh T, Rich S. Utility of electron beam computed tomography to stratify patients presenting to the emergency room with chest pain. *Am J Cardiol*. 1999;84:327–328.

100. Shaw LJ, Raggi P, Berman DS, Callister TQ. Cost effectiveness of screening for cardiovascular disease with measures of coronary calcium. *Prog Cardiovasc Dis*. 2003;46:171–184.

101. Rumberger JA, Behrenbeck T, Breen JF, Sheedy PF 2nd. Coronary calcification by electron beam computed tomography and obstructive coronary artery disease: a model for costs and effectiveness of diagnosis as compared with conventional cardiac testing methods. *J Am Coll Cardiol*. 1999;33:453–462.

102. Rifkin RD, Parisi AF, Folland E. Coronary calcification in the diagnosis of coronary artery disease. *Am J Cardiol*. 1979;44:141–147.

103. Eggen DA, Strong JP, McGill HC. Coronary calcification: relationship to clinically significant coronary lesions and race, sex and topographic distribution. *Circulation*. 1965;32:948–955.

104. Detrano RC, Wong ND, Doherty TM, Shavelle RM, Tang W, Ginzton LE, Budoff MJ, Narahara KA. Coronary calcium does not accurately predict near-term future coronary events in high-risk adults [published correction in *Circulation*. 2000;101:1355]. *Circulation*. 1999;99:2633–2638.

105. Park R, Detrano R, Xiang M, Fu P, Ibrahim Y, LaBree L, Azen S. Combined use of computed tomography coronary calcium scores and C-reactive protein levels in predicting cardiovascular events in nondiabetic individuals. *Circulation*. 2002;106:2073–2077.

106. Budoff MJ. Computed tomography. In: *Cardiac CT Imaging*. New York, NY: Springer-Verlag; 2006.

107. Raggi P, Callister TQ, Cooil B, He ZX, Lippolis NJ, Russo DJ, Zelinger A, Mahmarian JJ. Identification of patients at increased risk of first unheralded acute myocardial infarction by electron-beam computed tomography. *Circulation*. 2000;101:850–855.

108. Raggi P, Cooil B, Callister TQ. Use of electron beam tomography data to develop models for prediction of hard coronary events. *Am Heart J*. 2001;141:375–382.

109. Arad Y, Spadaro L, Goodman K, Newstein D, Guerci AD. Prediction of coronary events with electron beam computed tomography. *J Am Coll Cardiol*. 2000;36:1253–1260.

110. Wong ND, Hsu JC, Detrano RC, Diamond G, Eisenberg H, Gardin JM. Coronary artery calcium evaluation by electron beam computed tomography and its relation to new cardiovascular events. *Am J Cardiol*. 2000;86:495–498.

111. Kondos GT, Hoff JA, Sevrukov A, Daviglus ML, Garside DB, Devries SS, Chomka EV, Liu K. Electron-beam tomography coronary artery calcium and cardiac events: a 37-month follow-up of 5635 initially asymptomatic low- to intermediate-risk adults. *Circulation*. 2003;107:2571–2576.

112. Shaw LJ, Raggi P, Schisterman E, Berman DS, Callister TQ. Prognostic value of cardiac risk factors and coronary artery calcium screening for all-cause mortality. *Radiology*. 2003;228:826–833.

113. Shemesh J, Morag-Koren N, Goldbourt U, Grossman E, Tenenbaum A, Fisman EZ, Apter S, Itzchak Y, Motro M. Coronary calcium by spiral computed tomography predicts cardiovascular events in high-risk hypertensive patients. *J Hypertens*. 2004;22:605–610.

114. Arad Y, Goodman KJ, Roth M, Newstein D, Guerci AD. Coronary calcification, coronary risk factors, and atherosclerotic cardiovascular

disease events. The St. Francis Heart Study. *J Am Coll Cardiol*. 2005; 46:158–165.

115. Taylor AJ, Bindeman J, Feuerstein I, Cao F, Brazaitis M, O'Malley PG. Coronary calcium independently predicts incident premature coronary heart disease over measured cardiovascular risk factors: mean three-year outcomes in the Prospective Army Coronary Calcium (PACC) project. *J Am Coll Cardiol*. 2005;46:807–814.

116. Vliegenthart R, Oudkerk M, Hofman A, Oei HH, van Dijck W, van Rooij FJ, Witteman JC. Coronary calcification improves cardiovascular risk prediction in the elderly. *Circulation*. 2005;112:572–577.

117. LaMonte MJ, FitzGerald SJ, Church TS, Barlow CE, Radford NB, Levine BD, Pippin JJ, Gibbons LW, Blair SN, Nichaman MZ. Coronary artery calcium score and coronary heart disease events in a large cohort of asymptomatic men and women. *Am J Epidemiol*. 2005;162:421–429.

118. Becker A, Knez A, Becker C, Leber A, Anthopounou L, Boekstegers P, Steinbeck G. Prediction of serious cardiovascular events by determining coronary artery calcification measured by multi-slice computed tomography [in German]. *Dtsch Med Wochenschr*. 2005;130: 2433–2438.

119. Pletcher MJ, Tice JA, Pignone M, Browner WS. Using the coronary artery calcium score to predict coronary heart disease events: a systematic review and meta-analysis. *Arch Intern Med*. 2004;164: 1285–1292.

120. Raggi P, Shaw LJ, Berman DS, Callister TQ. Gender-based differences in the prognostic value of coronary calcification. *J Womens Health*. 2004;13:273–283.

121. Raggi P, Shaw LJ, Berman DS, Callister TQ. Prognostic value of coronary artery calcium screening in subjects with and without diabetes. *J Am Coll Cardiol*. 2004;43:1663–1669.

122. Greenland P, Smith SC Jr, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. *Circulation*. 2001;104: 1863–1867.

123. Detrano R, Hsiai T, Wang S, Puentes G, Fallovollita J, Shields P, Stanford W, Wolfkiel C, Georgiou D, Budoff M, Reed J. Prognostic value of coronary calcification and angiographic stenoses in patients undergoing coronary angiography. *J Am Coll Cardiol*. 1996;27: 285–290.

124. Keelan PC, Bielak LF, Ashai K, Jamjoum LS, Denktas AE, Rumberger JA, Sheedy PF II, Peyser PA, Schwartz RS. Long-term prognostic value of coronary calcification detected by electron-beam computed tomography in patients undergoing coronary angiography. *Circulation*. 2001;104:412–417.

125. Mohlenkamp S, Lehmann N, Schmermund A, Pump H, Moebus S, Baumgart D, Seibel R, Gronemeyer DH, Jockel KH, Erbel R. Prognostic value of extensive coronary calcium quantities in symptomatic males: a 5-year follow-up study. *Eur Heart J*. 2003;24:845–854.

126. De Backer G, Ambrosioni E, Borch-Johnsen K, Brotons C, Cifkova R, Dallongeville J, Ebrahim S, Faergeman O, Graham I, Mancia G, Manger Cats V, Orth-Gomer K, Perk J, Pyorala K, Rodicio JL, Sans S, Sansoy V, Sechtem U, Silber S, Thomsen T, Wood D; Third Joint Task Force of European and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. European guidelines on cardiovascular disease prevention in clinical practice. Third Joint Task Force of European and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. *Eur Heart J*. 2003;24:1601–1610.

127. Hecht HS. Practice guidelines for electron beam tomography: a report of the Society of Atherosclerosis Imaging. *Am J Cardiol*. 2000;86: 705–706.

128. Mosca L, Appel LJ, Benjamin EJ, Berra K, Chandra-Strobos N, Fabunmi RP, Grady D, Haan CK, Hayes SN, Judelson DR, Keenan NL, McBride P, Oparil S, Ouyang P, Oz MC, Mendelsohn ME, Pasternak RC, Pinn VW, Robertson RM, Schenck-Gustafsson K, Sila CA, Smith SC Jr, Sopko G, Taylor A, Walsh BW, Wenger NK, Williams CL; American Heart Association. Evidence-based guidelines for cardiovascular disease prevention in women. *Circulation*. 2004;109:672–693.

129. Taylor AJ, Bairey-Merz CN, Udelson JE. 34th Bethesda Conference: executive summary–can atherosclerosis imaging techniques improve the detection of patients at risk for ischemic heart disease? *J Am Coll Cardiol*. 2003;41:1860–1862.

130. Arad Y, Spadaro LA, Roth M, Newstein D, Guerci AD. Treatment of asymptomatic adults with elevated coronary calcium scores with atorvastatin, vitamin C, and vitamin E: the St. Francis Heart Study randomized clinical trial. *J Am Coll Cardiol*. 2005;46:166–172.

131. Wong ND, Budoff MJ, Pio J, Detrano RC. Coronary calcium and cardiovascular event risk: evaluation by age- and sex-specific quartiles. *Am Heart J*. 2002;143:456–459.

132. Sever PS, Dahlof B, Poulter NR, Wedel H, Beevers G, Caulfield M, Collins R, Kjeldsen SE, Kristinsson A, McInnes GT, Mehlsen J, Nieminen M, O'Brien E, Ostergren J; ASCOT investigators. Prevention of coronary and stroke events with atorvastatin in hypertensive patients who have average or lower-than-average cholesterol concentrations, in the Anglo-Scandinavian Cardiac Outcomes Trial–Lipid Lowering Arm (ASCOT-LLA): a multicentre randomised controlled trial. *Lancet*. 2003; 361:1149–1158.

133. Clouse ME. How useful is computed tomography for screening for coronary artery disease? Noninvasive screening for coronary artery disease with computed tomography is useful. *Circulation*. 2006;113: 125–146.

134. Pearson TA, Blair SN, Daniels SR, Eckel RH, Fair JM, Fortmann SP, Franklin BA, Goldstein LB, Greenland P, Grundy SM, Hong Y, Miller NH, Lauer RM, Ockene IS, Sacco RL, Sallis JF Jr, Smith SC Jr, Stone NJ, Taubert KA. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: Consensus Panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. American Heart Association Science Advisory and Coordinating Committee. *Circulation*. 2002;106: 388–391.

135. Eagle KA, Guyton RA, Davidoff R, Ewy GA, Fonger J, Gardner TJ, Gott JP, Herrmann HC, Marlow RA, Nugent W, O'Connor GT, Orszulak TA, Rieselbach RE, Winters WL, Yusuf S, Gibbons RJ, Alpert JS, Garson A Jr, Gregoratos G, Russell RO, Ryan TJ, Smith SC Jr. ACC/AHA guidelines for coronary artery bypass graft surgery: executive summary and recommendations: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1991 Guidelines for Coronary Artery Bypass Graft Surgery). *Circulation*. 1999;100: 1464–1480.

136. Gibbons RJ, Balady GJ, Bricker JT, Chaitman BR, Fletcher GF, Froelicher VF, Mark DB, McCallister BD, Mooss AN, O'Reilly MG, Winters WL Jr, Gibbons RJ, Antman EM, Alpert JS, Faxon DP, Fuster V, Gregoratos G, Hiratzka LF, Jacobs AK, Russell RO, Smith SC Jr; American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). ACC/AHA 2002 guideline update for exercise testing: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). *Circulation*. 2002;106:1883–1892.

137. Sevrukov AB, Bland JM, Kondos GT. Serial electron beam CT measurements of coronary artery calcium: has your patient's calcium score actually changed? *AJR Am J Roentgenol*. 2005;185:1546–1553.

138. Callister TQ, Raggi P, Cooil B, Lippolis NJ, Russo DJ. Effect of HMG-CoA reductase inhibitors on coronary artery disease as assessed by electron beam computed tomography. *N Engl J Med*. 1998;339: 1972–1978.

139. Budoff MJ, Lane KL, Bakhsheshi H, Mao S, Grassmann BO, Friedman BC, Brundage BH. Rates of progression of coronary calcium by electron beam tomography. *Am J Cardiol*. 2000;86:8–11.

140. Achenbach S, Ropers D, Pohle K, Leber A, Thilo C, Knez A, Menendez T, Maeffert R, Kusus M, Regenfus M, Bickel A, Haberl R, Steinbeck G, Moshage W, Daniel WG. Influence of lipid-lowering therapy on the progression of coronary artery calcification: a prospective evaluation. *Circulation*. 2002;106:1077–1082.

141. Shavelle DM, Takasu J, Budoff MJ, Mao SS, Zhao QX, O'Brien KD. HMG CoA reductase inhibitor (statin) and aortic valve calcium. *Lancet*. 2002;359:1125–1126.

142. Cowell SJ, Newby DE, Prescott RJ, Bloomfield P, Reid J, Northridge DB, Boon NA; Scottish Aortic Stenosis and Lipid Lowering Trial, Impact on Regression (SALTIRE) Investigators. A randomized trial of intensive lipid-lowering therapy in calcific aortic stenosis. *N Engl J Med*. 2005;352:2389–2397.

143. Taylor A, Shaw LJ, Fayad Z, O'Leary D, Nissen S, Rader D, Raggi P. Tracking atherosclerosis regression: a clinical tool in preventive cardiology. *Atherosclerosis*. 2005;180:1–10.

144. Budoff MJ, Takasu J, Flores FR, Niihara Y, Lu B, Lau BH, Rosen RT, Amagase H. Inhibiting progression of coronary calcification using aged garlic extract in patients receiving statin therapy: a preliminary study. *Prev Med*. 2004;39:985–991.

145. Raggi P, Davidson M, Callister TQ, Welty FK, Bachmann GA, Hecht H, Rumberger JA. Aggressive versus moderate lipid-lowering therapy in hypercholesterolemic postmenopausal women: Beyond Endorsed Lipid Lowering with EBT Scanning (BELLES). *Circulation*. 2005;112: 563–571.

146. Raggi P, Cooil B, Shaw L, Aboulhson J, Takasu J, Budoff M, Callister TQ. Progression of coronary calcification on serial electron beam tomography scanning is greater in patients with future myocardial infarction. *Am J Cardiol*. 2003;92:827–829.

147. Raggi P, Callister TQ, Shaw LJ. Progression of coronary artery calcium and risk of first myocardial infarction in patients receiving cholesterol-lowering therapy. *Arterioscler Thromb Vasc Biol*. 2004;24:1272–1277.

148. Thompson RC, McGhie AI, Moser KW, O'Keefe JH Jr, Stevens TL, House J, Fritsch N, Bateman TM. Clinical utility of coronary calcium scoring after nonischemic myocardial perfusion imaging. *J Nucl Cardiol*. 2005;12:392–400.

149. Moshage W, Achenbach S, Seese B, Bachmann K, Kirchgeorg M. Coronary artery stenoses: three-dimensional imaging with electrocardiographically triggered, contrast agent-enhanced, electron beam CT. *Radiology*. 1995;196:707–714.

150. Nakanishi T, Ito K, Imazu M, Yamakido M. Evaluation of coronary artery stenoses using electron-beam CT and multiplanar reformation. *J Comp Assist Tomogr*. 1997;21:121–127.

151. Schmermund A, Rensing BJ, Sheedy PF, Bell MR, Rumberger JA. Intravenous electron-beam computed tomographic coronary angiography for segmental analysis of coronary artery stenoses. *J Am Coll Cardiol*. 1998;31:1547–1554.

152. Reddy GP, Chernoff DM, Adams JR, Higgins CB. Coronary artery stenoses: assessment with contrast-enhanced electron-beam CT and axial reconstructions. *Radiology*. 1998;208:167–172.

153. Rensing BJ, Bongaerts A, van Geuns RJ, van Ooijen P, Oudkerk M, de Feyter PJ. Intravenous coronary angiography by electron beam computed tomography: a clinical evaluation. *Circulation*. 1998;98: 2509–2512.

154. Achenbach S, Moshage W, Ropers D, Nossen J, Daniel WG. Value of electron-beam computed tomography for the detection of high-grade coronary artery stenoses and occlusions. *N Engl J Med*. 1998;339: 1964–1971.

155. Budoff MJ, Oudiz RJ, Zalace CP, Baksheshi H, Goldberg SL, French WJ, Rami TG, Brundage BH. Intravenous three-dimensional coronary angiography using contrast-enhanced electron beam computed tomography. *Am J Cardiol*. 1999;83:840–845.

156. Achenbach S, Ropers D, Regenfus M, Muschiol G, Daniel WG, Moshage W. Contrast-enhanced electron beam computed tomography to analyze the coronary arteries in patients after acute myocardial infarction. *Heart*. 2000;84:489–493.

157. Leber AW, Knez A, Mukherjee R, White C, Huber A, Becker A, Becker CR, Reiser M, Haberl R, Steinbeck G. Usefulness of calcium scoring using electron beam computed tomography and noninvasive coronary angiography in patients with suspected coronary artery disease. *Am J Cardiol*. 2001;88:219–223.

158. Ropers D, Regenfus M, Stilianakis N, Birke S, Kessler W, Moshage W, Laub G, Daniel WG, Achenbach S. A direct comparison of noninvasive coronary angiography by electron beam tomography and navigator-echo-based magnetic resonance imaging for the detection of restenosis following coronary angioplasty. *Invest Radiol*. 2002;37:386–392.

159. Nikolaou K, Huber A, Knez A, Becker C, Bruening R, Reiser M. Intraindividual comparison of contrast-enhanced electron-beam computed tomography and navigator-echo-based magnetic resonance imaging for noninvasive coronary artery angiography. *Eur Radiol*. 2002; 12:1663–1671.

160. Lu B, Shavelle DM, Mao S, Chen L, Child J, Carson S, Budoff MJ. Improved accuracy of noninvasive electron beam coronary angiography. *Invest Radiol*. 2004;39:73–79.

161. Budoff MJ, Lu B, Shinbane JS, Chen L, Child J, Carson S, Mao S. Methodology for improved detection of coronary stenoses with computed tomographic angiography. *Am Heart J*. 2004;148:1085–1090.

162. Becker CR, Knez A, Leber A, Hong C, Treede H, Wildhirt S, Ohnesorge B, Flohr T, Schoepf UJ, Reiser MF. Initial experiences with multi-slice detector spiral CT in diagnosis of arteriosclerosis of coronary vessels [in German]. *Radiologe*. 2000;40:118–122.

163. Ohnesorge B, Flohr T, Becker C, Kopp AF, Schoepf UJ, Baum U, Knez A, Klingenbeck-Regn K, Reiser MF. Cardiac imaging by means of electrocardiographically gated multisection spiral CT: initial experience. *Radiology*. 2000;217:564–571.

164. Achenbach S, Ulzheimer S, Baum U, Kachelriess M, Ropers D, Giesler T, Bautz W, Daniel WG, Kalender WA, Moshage W. Noninvasive coronary angiography by retrospectively ECG-gated multislice spiral CT. *Circulation*. 2000;102:2823–2828.

165. Nieman K, Oudkerk M, Rensing BJ, van Ooijen P, Munne A, van Geuns RJ, de Feyter PJ. Coronary angiography with multi-slice computed tomography. *Lancet*. 2001;357:599–603.

166. Achenbach S, Giesler T, Ropers D, Ulzheimer S, Derlien H, Schulte C, Wenkel E, Moshage W, Bautz W, Daniel WG, Kalender WA, Baum U. Detection of coronary artery stenoses by contrast-enhanced, retrospectively electrocardiographically-gated, multi-slice spiral computed tomography. *Circulation*. 2001;103:2535–2538.

167. Knez A, Becker CR, Leber A, Ohnesorge B, Becker A, White C, Haberl R, Reiser MF, Steinbeck G. Usefulness of multislice spiral computed tomography angiography for determination of coronary artery stenoses. *Am J Cardiol*. 2001;88:1191–1194.

168. Vogl TJ, Abolmaali ND, Diebold T, Engelmann, Ay M, Dogan S, Wimmer-Greinecker G, Moritz A, Herzog C. Techniques for the detection of coronary atherosclerosis: multi-detector row CT coronary angiography. *Radiology*. 2002;223:212–220.

169. Kopp AF, Schroeder S, Kuettner A, Baumbach A, Georg C, Kuzo R, Heuschmid M, Ohnesorge B, Karsch KR, Claussen CD. Non-invasive coronary angiography with high resolution multidetector-row computed tomography: results in 102 patients. *Eur Heart J*. 2002;23:1714–1725.

170. Nieman K, Rensing BJ, van Geuns RJ, Munne A, Ligthart JM, Pattynama PM, Krestin GP, Serruys PW, de Feyter PJ. Usefulness of multislice computed tomography for detecting obstructive coronary artery disease. *Am J Cardiol*. 2002;89:913–918.

171. Becker CR, Knez A, Leber A, et al. Detection of coronary artery stenoses with multislice helical CT angiography. *J Comput Assist Tomogr*. 2002;26:750–755.

172. Morgan-Hughes GJ, Marshall AJ, Roobottom CA. Multislice computed tomographic coronary angiography: experience in a UK centre. *Clin Radiol*. 2003;58:378–383.

173. Sato Y, Matsumoto N, Kato M, Inoue F, Horie T, Kusama J, Yoshimura A, Imazeki T, Fukui T, Furuhashi S, Takahashi M, Kanmatsuse K. Noninvasive assessment of coronary artery disease by multislice spiral computed tomography using a new retrospectively ECG-gated image reconstruction technique. *Circ J*. 2003;67:401–405.

174. Maruyama T, Yoshizumi T, Tamura R, Takashima S, Toyoshima H, Konishi I, Yamashita S, Yamasaki K. Comparison of visibility and diagnostic capability of noninvasive coronary angiography by eight-slice multidetector-row computed tomography versus conventional coronary angiography. *Am J Cardiol*. 2004;93:537–542.

175. Nieman K, Cademartiri F, Lemos PA, Raaijmakers R, Pattynama PM, de Feyter PJ. Reliable noninvasive coronary angiography with fast submillimeter multislice spiral computed tomography. *Circulation*. 2002;106: 2051–2054.

176. Ropers D, Baum U, Pohle K, Anders K, Ulzheimer S, Ohnesorge B, Schlundt C, Bautz W, Daniel WG, Achenbach S. Detection of coronary artery stenoses with thin-slice multi-detector row spiral computed tomography and multiplanar reconstruction. *Circulation*. 2003;107: 664–666.

177. Kuettner A, Trabold T, Schroeder S, Feyer A, Beck T, Brueckner A, Heuschmid M, Burgstahler C, Kopp AF, Claussen CD. Noninvasive detection of coronary lesions using 16-detector multislice spiral computed tomography technology: initial clinical results. *J Am Coll Cardiol*. 2004;44:1230–1237.

178. Mollet NR, Cademartiri F, Nieman K, Saia F, Lemos PA, McFadden EP, Pattynama PM, Serruys PW, Krestin GP, de Feyter PJ. Multislice spiral computed tomography coronary angiography in patients with stable angina pectoris. *J Am Coll Cardiol*. 2004;43:2265–2270.

179. Martuscelli E, Romagnoli A, D'Eliseo A, Razzini C, Tomassini M, Sperandio M, Simonetti G, Romeo F. Accuracy of thin-slice computed tomography in the detection of coronary stenoses. *Eur Heart J*. 2004; 25:1043–1048.

180. Hoffmann U, Moselewski F, Cury RC, Ferencik M, Jank IK, Diaz LJ, Abbara S, Brady TJ, Achenbach S. Predictive value of 16-slice multidetector spiral computed tomography to detect significant obstructive coronary artery disease in patients at high risk for coronary disease: patient versus segment-based analysis. *Circulation*. 2004;110: 2638–2643.

181. Kuettner A, Beck T, Drosch T, Kettering K, Heuschmid M, Burgstahler C, Claussen CD, Kopp AF, Schroeder S. Diagnostic accuracy of non-invasive coronary imaging using 16-detector slice spiral computed

tomography with 188 ms temporal resolution. *J Am Coll Cardiol*. 2005;45:123–127.

182. Mollet NR, Cademartiri F, Krestin GP, McFadden EP, Arampatzis CA, Serruys PW, de Feyter PJ. Improved diagnostic accuracy with 16-row multi-slice computed tomography coronary angiography. *J Am Coll Cardiol*. 2005;45:128–132.

183. Schuijf JD, Bax JJ, Salm LP, Jukema JW, Lamb HJ, van der Wall EE, de Roos A, Noninvasive coronary imaging and assessment of left ventricular function using 16-slice computed tomography. *Am J Cardiol*. 2005;95:571–574.

184. Morgan-Hughes GJ, Roobottom CA, Owens PE, Marshall AJ. Highly accurate coronary angiography with submillimetre, 16 slice computed tomography. *Heart*. 2005;91:308–313.

185. Hoffmann MH, Shi H, Schmitz BL, Schmid FT, Lieberknecht M, Schulze R, Ludwig B, Kroschel U, Jahnke N, Haerer W, Brambs HJ, Aschoff AJ. Noninvasive coronary angiography with multislice computed tomography [published correction in *JAMA*. 2005;293: 2531–2533]. *JAMA*. 2005;293:2471–2478.

186. Fine JJ, Hopkins CB, Hall PA, Delphia RE, Attebery TW, Newton FC. Noninvasive coronary angiography: agreement of multi-slice spiral computed tomography and selective catheter angiography. *Int J Cardiovasc Imaging*. 2004;20:549–552.

187. Kaiser C, Bremerich J, Haller S, Brunner-La Rocca HP, Bongartz G, Pfisterer M, Buser P. Limited diagnostic yield of non-invasive coronary angiography by 16-slice multidetector spiral computed tomography in routine patients referred for evaluation of coronary artery disease. *Eur Heart J*. 2005;26:1987–1992.

188. Aviram G, Finkelstein A, Herz I, Lessick J, Miller H, Graif M, Keren G. Clinical value of 16-slice multi-detector CT compared to invasive coronary angiography. *Int J Cardiovasc Intervent*. 2005;7:21–28.

189. Achenbach S, Ropers D, Pohle FK, Raaz D, von Erffa J, Yilmaz A, Muschiol G, Daniel WG. Detection of coronary artery stenoses using multi-detector CT with 16 x 0.75 collimation and 375 ms rotation. *Eur Heart J*. 2005;26:1978–1986.

190. Leber AW, Knez A, von Ziegler F, Becker A, Nikolaou K, Paul S, Wintersperger B, Reiser M, Becker CR, Steinbeck G, Boekstegers P. Quantification of obstructive and nonobstructive coronary lesions by 64-slice computed tomography: a comparative study with quantitative coronary angiography and intravascular ultrasound. *J Am Coll Cardiol*. 2005;46:147–154.

191. Mollet NR, Cademartiri F, van Mieghem CA, Runza G, McFadden EP, Baks T, Serruys PW, Krestin GP, de Feyter PJ. High-resolution spiral computed tomography coronary angiography in patients referred for diagnostic conventional coronary angiography. *Circulation*. 2005;112: 2318–2323.

192. Ropers D, Rixe J, Anders K, Küttner A, Baum U, Bautz W, Daniel WG, Achenbach S. Usefulness of multidetector row computed tomography with 64- x 0.6-mm collimation and 330-ms rotation for the noninvasive detection of significant coronary artery stenoses. *Am J Cardiol*. 2006; 97:343–348.

193. Fine JJ, Hopkins CB, Ruff N, Newton FC. Comparison of accuracy of 64-slice cardiovascular computed tomography with coronary angiography in patients with suspected coronary artery disease. *Am J Cardiol*. 2006;97:173–174.

194. Giesler T, Baum U, Ropers D, Ulzheimer S, Wenkel E, Mennicke M, Bautz W, Kalender WA, Daniel WG, Achenbach S. Noninvasive visualization of coronary arteries using contrast-enhanced multidetector CT: influence of heart rate on image quality and stenosis detection. *AJR Am J Roentgenol*. 2002;179:911–916.

195. Schroeder S, Kopp AF, Kuettner A, Burgstahler C, Herdeg C, Heuschmid M, Baumbach A, Claussen CD, Karsch KR, Seipel L. Influence of heart rate on vessel visibility in noninvasive coronary angiography using new multislice computed tomography: experience in 94 patients. *Clin Imaging*. 2002;26:106–111.

196. Hoffmann MH, Shi H, Manzke R, Schmid FT, De Vries L, Grass M, Brambs HJ, Aschoff AJ. Noninvasive coronary angiography with 16-detector row CT: effect of heart rate. *Radiology*. 2005;234:86–97.

197. Herzog C, Abolmaali N, Balzer JO, Baunach S, Ackermann H, Dogan S, Britten MB, Vogl TJ. Heart-rate-adapted image reconstruction in multidetector-row cardiac CT: influence of physiological and technical prerequisite on image quality. *Eur Radiol*. 2002;12:2670–2678.

198. Schuijf JD, Bax JJ, Shaw LJ, de Roos A, Lamb HJ, van der Wall EE, Wijns W. Meta-analysis of comparative diagnostic performance of magnetic resonance imaging and multislice computed tomography for noninvasive coronary angiography. *Am Heart J*. 2006;151:404–411.

199. Stein PD, Beemath A, Kayali F, Skaf E, Sanchez J, Olson RE. Multidetector computed tomography for the diagnosis of coronary artery disease: a systematic review. *Am J Med*. 2006;119:203–216.

200. Gibbons RJ, Chatterjee K, Daley J, Douglas JS, Fihn SD, Gardin JM, Grunwald MA, Levy D, Lytle BW, O'Rourke RA, Schafer WP, Williams SV. ACC/AHA/ACP-ASIM guidelines for the management of patients with chronic stable angina: executive summary and recommendations: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Chronic Stable Angina). *Circulation*. 1999;99: 2829–2848.

201. Pump H, Mohlenkamp S, Sehnert CA, Schimpf SS, Schmidt A, Erbel R, Gronemeyer DH, Seibel RM. Coronary arterial stent patency: assessment with electron-beam CT. *Radiology*. 2000;214:447–452.

202. Gilard M, Cornily JC, Rioufol G, Finet G, Pennec PY, Mansourati J, Blanc JJ, Boschat J. Noninvasive assessment of left main coronary stent patency with 16-slice computed tomography. *Am J Cardiol*. 2005;95: 110–112.

203. Hong C, Chrysant GS, Woodard PK, Bae KT. Coronary artery stent patency assessed with in-stent contrast enhancement measured at multidetector row CT angiography: initial experience. *Radiology*. 2004;233: 286–291.

204. Knollmann FD, Moller J, Gebert A, Bethge C, Felix R. Assessment of coronary artery stent patency by electron-beam CT. *Eur Radiol*. 2004; 14:1341–1347.

205. Schuijf JD, Bax JJ, Jukema JW, Lamb HJ, Warda HM, Vliegen HW, de Roos A, van der Wall EE. Feasibility of assessment of coronary stent patency using 16-slice computed tomography. *Am J Cardiol*. 2004;94: 427–430.

206. Kruger S, Mahnken AH, Sinha AM, Borghans A, Dedden K, Hoffmann R, Hanrath P. Multislice spiral computed tomography for the detection of coronary stent restenosis and patency. *Int J Cardiol*. 89:167–172.

207. Mahnken AH, Buecker A, Wildberger JE, Ruebben A, Stanzel S, Vogt F, Gunther RW, Blindt R. Coronary artery stents in multislice computed tomography: in vitro artifact evaluation. *Invest Radiol*. 2004;39:27–33.

208. Maintz D, Seifarth H, Flohr T, Kramer S, Wichter T, Heindel W, Fischbach R. Improved coronary artery stent visualization and in-stent stenosis detection using 16-slice computed-tomography and dedicated image reconstruction technique. *Invest Radiol*. 2003;38:790–795.

209. Mahnken AH, Seyfarth T, Flohr T, Herzog C, Stahl J, Stanzel S, Kuettner A, Wildberger JE, Gunther RW. Flat-panel detector computed tomography for the assessment of coronary artery stents: phantom study in comparison with 16-slice spiral computed tomography. *Invest Radiol*. 2005;40:8–13.

210. Cademartiri F, Mollet N, Lemos PA, Pugliese F, Baks T, McFadden EP, Krestin GP, de Feyter PJ. Usefulness of multislice computed tomographic coronary angiography to assess in-stent restenosis. *Am J Cardiol*. 2005;96:799–802.

211. Stanford W, Brundage BH, MacMillan R, Bateman TM, Eldredge WJ, Chomka EV, Lipton MJ, White CF, Wilson RF, Johnson MR, Marcus ML. Sensitivity and specificity of assessing coronary bypass graft patency with ultrafast computed tomography: results of a multicenter study. *J Am Coll Cardiol*. 1988;12:1–7.

212. Knez A, von Smekal A, Haberl R, Spiegl F, Reichart B, Reiser M, Steinbeck G. The value of ultrafast computerized tomography in detection of the patency of coronary bypasses [in German]. *Z Kardiol*. 1996;85:629–634.

213. Achenbach S, Moshage W, Ropers D, Nossen J, Bachmann K. Noninvasive, three-dimensional visualization of coronary artery bypass grafts by electron beam tomography. *Am J Cardiol*. 1997;79:856–861.

214. Ha JW, Cho SY, Shim WH, Chung N, Jang Y, Lee HM, Choe KO, Chung WJ, Choi SH, Yoo KJ, Kang MS. Noninvasive evaluation of coronary artery bypass graft patency using three-dimensional angiography obtained with contrast-enhanced electron beam CT. *AJR Am J Roentgenol*. 1999;172:1055–1059.

215. Marano R, Storto ML, Maddestra N, Bonomo L. Non-invasive assessment of coronary artery bypass graft with retrospectively ECG-gated four-row multi-detector spiral computed tomography. *Eur Radiol*. 2004;14:1353–1362.

216. Lu B, Dai RP, Zhuang N, Budoff MJ. Noninvasive assessment of coronary artery bypass graft patency and flow characteristics by electron-beam tomography. *J Invasive Cardiol*. 2002;14:19–24.

217. Hoshi T, Yamauchi T, Kanauchi T, Konno M, Imai K, Suwa J, Onoguchi K, Hashimoto K, Horie T. Three-dimensional computed

tomography angiography of coronary artery bypass graft with electron beam tomography [in Japanese]. *J Cardiol*. 2001;38:197–202.

218. Engelmann MG. von Smekal A, Knez A, Kurzinger E, Huehns TY, Hofling B, Reiser M. Accuracy of spiral computed tomography for identifying arterial and venous coronary graft patency. *Am J Cardiol*. 1997;80:569–574.

219. Burgstahler C, Kuettner A, Kopp AF, Herdeg C, Martensen J, Claussen CD, Schroeder S. Non-invasive evaluation of coronary artery bypass grafts using multi-slice computed tomography: initial clinical experience. *Int J Cardiol*. 2003;90:275–280.

220. Rossi R, Chiurlia E, Ratti C, Ligabue G, Romagnoli R, Modena MG. Noninvasive assessment of coronary artery bypass graft patency by multislice computed tomography. *Ital Heart J*. 2004;5:36–41.

221. Gurevitch J, Gaspar T, Orlov B, Amar R, Dvir D, Peled N, Aravot DJ. Noninvasive evaluation of arterial grafts with newly released multidetector computed tomography. *Ann Thorac Surg*. 2003;76:1523–1527.

222. Ropers D, Ulzheimer S, Wenkel E, Baum U, Giesler T, Derlien H, Moshage W, Bautz WA, Daniel WG, Kalender WA, Achenbach S. Investigation of aortocoronary bypass grafts by multislice spiral computed tomography with electrocardiographic-gated image reconstruction. *Am J Cardiol*. 2001;88:792–795.

223. Nieman K, Pattynama PM, Rensing BJ, Van Geuns RJ, De Feyter PJ. Evaluation of patients after coronary artery bypass surgery: CT angiographic assessment of grafts and coronary arteries. *Radiology*. 2003;229: 749–756.

224. Martuscelli E, Romagnoli A, D'Eliseo A, Tomassini M, Razzini C, Sperandio M, Simonetti G, Romeo F, Mehta J. Evaluation of venous and arterial conduit patency by 16-slice spiral computed tomography. *Circulation*. 2004;110:3234–3238.

225. Schlosser T, Konorza T, Hunold P, Kuhl H, Schmermund A, Barkhausen J. Noninvasive visualization of coronary artery bypass grafts using 16-detector row tomography. *J Am Coll Cardiol*. 2004;44:1224–1229.

226. Anders K, Baum U, Schmid M, Ropers D, Schmid A, Pohle K, Daniel WG, Bautz W, Achenbach S. Coronary artery bypass graft (CABG) patency: assessment with high-resolution submillimeter 16-slice multidetector-row computed tomography (MDCT) versus coronary angiography. *Eur J Radiol*. 2006;57:336–344.

227. Chiurlia E, Menozzi M, Ratti C, Romagnoli R, Modena MG. Follow-up of coronary artery bypass graft patency by multislice computed tomography. *Am J Cardiol*. 2005;95:1094–1097.

228. Ropers D, Moshage W, Daniel WG, Jessl J, Gottwik M, Achenbach S. Visualization of coronary artery anomalies and their course by contrast-enhanced electron beam tomography and three-dimensional reconstruction. *Am J Cardiol*. 2001;87:193–197.

229. Yoshimura N, Hamada S, Takamiya M, Kuribayashi S, Kimura K. Coronary artery anomalies with a shunt: evaluation with electron-beam CT. *J Comput Assist Tomogr*. 1998;22:682–686.

230. Deibler AR, Kuzo RS, Vohringer M, Page EE, Safford RE, Patron JN, Lane GE, Morin RL, Gerber TC. Imaging of congenital coronary anomalies with multislice computed tomography. *Mayo Clin Proc*. 2004;79:1017–1023.

231. Lessick J, Kumar G, Beyar R, Lorber A, Engel A. Anomalous origin of a posterior descending artery from the right pulmonary artery: report of a rare case diagnosed by multidetector computed tomography angiography. *J Comput Assist Tomogr*. 2004;28:857–859.

232. Sato Y, Inoue F, Matsumoto N, Tani S, Takayama T, Yoda S, Kunimasa T, Ishii N, Uchiyama T, Saito S, Tanaka H, Furuhashi S, Takahashi M, Koyama Y. Detection of anomalous origins of the coronary artery by means of multislice computed tomography. *Circ J*. 2005;69:320–324.

233. Schmid M, Achenbach S, Ludwig J, Baum U, Anders K, Pohle K, Daniel WG, Ropers D. Visualization of coronary artery anomalies by contrast-enhanced multi-detector row spiral computed tomography. *Int J Cardiol*. 2006;111:430–435.

234. Roberts W, Jones AA, Nissen SE. Coronary intravascular ultrasound: implications for A. Quantitation of coronary arterial narrowing at necropsy in sudden coronary death. *Am J Cardiol*. 1979;44:39–44.

235. Mintz GS, Painter JA, Pichard AD, Kent KM, Satler LF, Popma JJ. Chuang UC, Bucher TA, Sokolowicz LE, Leon MB. Atherosclerosis in angiographically "normal" coronary artery reference segments: an intravascular ultrasound study with clinical correlations. *J Am Coll Cardiol*. 1995;25:1479–1485.

236. Little WC, Constantinescu M, Applegate RJ, Kutcher MA, Burrows MT, Kahl FR, Santamore WP. Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild-to-moderate coronary artery disease? *Circulation*. 1988;78:1157–1166.

237. Falk E, Shah PK, Fuster V. Coronary plaque disruption. *Circulation*. 1995;92:657–671.

238. Leber AW, Knez A, Becker A, von Ziegler F, Nikolaou K, Rist C, Reiser M, White C, Steinbeck G, Boekstegers P. Accuracy of multidetector spiral computed tomography in identifying and differentiating the composition of coronary atherosclerotic plaques: a comparative study with intracoronary ultrasound. *J Am Coll Cardiol*. 2004;43:1241–1247.

239. Achenbach S, Moselewski F, Ropers D, Ferencik M, Hoffmann U, MacNeill B, Pohle K, Baum U, Anders K, Jang IK, Daniel WG, Brady TJ. Detection of calcified and noncalcified coronary atherosclerotic plaque by contrast-enhanced, submillimeter multidetector spiral computed tomography: a segment-based comparison with intravascular ultrasound. *Circulation*. 2004;109:14–17.

240. Schoenhagen P, Tuzcu EM, Stillman AE, Moliterno DJ, Halliburton SS, Kuzmiak SA, Kasper JM, Magyar WA, Lieber ML, Nissen SE, White RD. Non-invasive assessment of plaque morphology and remodeling in mildly stenotic coronary segments: comparison of 16-slice computed tomography and intravascular ultrasound. *Coron Artery Dis*. 2003;14: 459–462.

241. Schroeder S, Kopp AF, Baumbach A, Meisner C, Kuettner A, Georg C, Ohnesorge B, Herdeg C, Claussen CD, Karsch KR. Noninvasive detection and evaluation of atherosclerotic coronary plaques with multislice computed tomography. *J Am Coll Cardiol*. 2001;37:1430–1435.

242. Baumgart D, Schmermund A, Goerge G, Haude M, Ge J, Adamzik M, Sehnert C, Altmaier K, Groenemeyer D, Seibel R, Erbel R. Comparison of electron beam computed tomography with intracoronary ultrasound and coronary angiography for detection of coronary atherosclerosis. *J Am Coll Cardiol*. 1997;30:57–64.

243. Rasouli ML, Shavelle DM, French WJ, McKay CR, Budoff MJ. Assessment of coronary plaque morphology by contrast-enhanced computed tomographic angiography: comparison with intravascular ultrasound. *Coron Artery Dis*. 2006;17:359–364.

244. Budoff MJ. Prevalence of soft plaque detection with computed tomography. *J Am Coll Cardiol*. 2006;48:319–321.

245. Moselewski F, Ropers D, Pohle K, Hoffmann U, Ferencik M, Chan RC, Cury RC, Abbara S, Jang IK, Brady TJ, Daniel WG, Achenbach S. Comparison of measurement of cross-sectional coronary atherosclerotic plaque and vessel areas by 16-slice multidetector computed tomography versus intravascular ultrasound. *Am J Cardiol*. 2004;94:1294–1297.

ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR APPROPRIATENESS CRITERIA

ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 Appropriateness Criteria for Cardiac Computed Tomography and Cardiac Magnetic Resonance Imaging*

A Report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology

CCT/CMR WRITING GROUP

Robert C. Hendel, MD, FACC
Manesh R. Patel, MD

Christopher M. Kramer, MD, FACC†
Michael Poon, MD, FACC‡

TECHNICAL PANEL MEMBERS

Robert C. Hendel, MD, FACC, *Moderator*§

James C. Carr, MD, BCH, BAO‖
Nancy A. Gerstad, MD
Linda D. Gillam, MD, FACC
John McB. Hodgson, MD, FSCAI, FACC¶
Raymond J. Kim, MD, FACC
Christopher M. Kramer, MD, FACC†
John R. Lesser, MD, FACC

Edward T. Martin, MD, FACC, FACP
Joseph V. Messer, MD, MACC, FSCAI
Rita F. Redberg, MD, MSc, FACC**
Geoffrey D. Rubin, MD, FSCBTMR††
John S. Rumsfeld, MD, PhD, FACC
Allen J. Taylor, MD, FACC
Wm. Guy Weigold, MD, FACC‡

Pamela K. Woodard, MD‡‡

†Society for Cardiovascular Magnetic Resonance Official Representative; ‡Society of Cardiovascular Computed Tomography Official Representative; §American Society of Nuclear Cardiology Official Representative; ‖Society of Interventional Radiology Official Representative; ¶Society for Cardiovascular Angiography and Interventions Official Representative; **American Heart Association Official Representative; ††American College of Radiology Official Representative; ‡‡North American Society for Cardiac Imaging Official Representative.

ACCF APPROPRIATENESS CRITERIA WORKING GROUP

Ralph G. Brindis, MD, MPH, FACC, *Chair*

Robert C. Hendel, MD, FACC
Pamela S. Douglas, MD, MACC
Eric D. Peterson, MD, FACC

Michael J. Wolk, MD, MACC
Joseph M. Allen, MA
Manesh R. Patel, MD

TABLE OF CONTENTS

ABSTRACT

Under the auspices of the American College of Cardiology Foundation (ACCF) together with key specialty and subspecialty societies, appropriateness reviews were conducted for 2 relatively new clinical cardiac imaging modalities, cardiac computed tomography (CCT) and cardiac magnetic resonance (CMR) imaging. The reviews assessed the risks and benefits of the imaging tests for several indications or clinical scenarios and scored them based on a scale of 1 to 9, where the upper range (7 to 9) implies that the test is generally acceptable and is a reasonable approach, and the lower range (1 to 3) implies that the test is generally not acceptable and is not a reasonable approach. The mid-range (4 to 6) indicates an uncertain clinical scenario. The indications for these reviews were drawn from common applications or anticipated uses, as few clinical practice guidelines currently exist for these techniques. These indications were reviewed by an independent group of clinicians and modified by the Working Group, and then panelists rated the indications based on the ACCF Methodology for Evaluating the Appropriateness of Cardiovascular Imaging, which blends scientific evidence and practice experience. A modified Delphi technique was used to obtain first and second round ratings of clinical indications after the panelists were provided with a set of literature reviews, evidence tables, and seminal references. The final ratings were evenly distributed among the 3 categories of appropriateness for both CCT and CMR. Use of tests for structure and function and for diagnosis in symptomatic, intermediate coronary artery disease (CAD) risk patients was deemed appropriate, while repeat testing and general screening uses were viewed less favorably. It is anticipated that these results will have a significant impact on physician decision making and performance, reimbursement policy, and future research directions.

PREFACE

The following paper combines the second and third reports in an ongoing series of technical documents that critically

and systematically create, review, and categorize appropriateness criteria for cardiovascular diagnostic tests and procedures utilized by physicians caring for patients with cardiovascular diseases. The ACCF believes that a careful blending of a broad range of clinical experience and available evidence-based information can help guide a more efficient and equitable allocation of health care resources in imaging. The ultimate objective of these reviews is to improve patient care and health outcomes in a cost-effective manner based on current understanding of the limits of the imaging modalities examined, without constraining the crucial role of physician judgment in the face of diverse clinical presentations and varying patient characteristics. Although there are a limited number of studies available to evaluate the techniques examined in these reports, the appropriateness criteria hopefully can serve as initial guides for the responsible use of CCT and CMR and related resources. Our approach is not to diminish the acknowledged ambiguity of clinical decision making for certain patients by statistical means or consensus techniques, but to recognize that real differences in clinical opinion can exist for particular patient presentation, especially in an evolving field with limited evidence. Such differences are grounds for more research and for even more careful deliberation on the proper care for each indication and patient. These reports will need to be updated more frequently than most policy statements as further data and information are gained about their use. Not ordering a test when it would be otherwise considered appropriate may be the correct clinical decision, and is a judgment call based on the individual characteristics of patients and their particular clinical scenarios. Likewise, ordering a test for an indication deemed inappropriate may be the correct clinical pathway if supported by mitigating characteristics of the patient that could justify this approach.

This work was not possible without the dedicated work of the Technical Panel, composed of clinician experts, some with special background in cardiac imaging and others with impeccable credentials in general cardiovascular medicine, health services research, and health plan administration. This diversity in backgrounds of the Technical Panel as shown in Appendix C made for a wide range of scoring for many of the indications. It is much easier to "game" or "bias" the scoring process by limiting panel membership solely to specialists of the particular procedure being evaluated for appropriateness. Such specialists would have a natural tendency to rate each indication higher than non-specialists in a given test or procedure. Thus, it is with gratitude that we applaud the Technical Panel, a professional group with a wide range of skills and insights, for a considered and thorough deliberation of the merits of each test for every indication.

Special mention and thanks are due to Elliott Antman, MD, FACC; Ronald Peshock, MD, FACC; Gregory Thomas, MD, FACC; and Samuel Wann, MD, FACC, for reviewing the draft indications; to Joe Allen, who continually drove the process forward; and to ACCF Past President

Pamela Douglas, MD, MACC, for her insight and leadership.

Robert Hendel, MD, FACC
Moderator, CCT/CMR Technical Panel

Ralph Brindis, MD, MPH, FACC
Chair, Appropriateness Criteria Working Group

INTRODUCTION

Rapid technological advances and new clinical applications in cardiovascular imaging technology, coupled with increasing therapeutic options for cardiovascular disease, have led to explosive growth in cardiovascular imaging. In fact, diagnostic imaging services reimbursed under Medicare's physician fee schedule grew more rapidly than any other type of physician service from 1999 to 2003 (1). During this time, the armamentarium of non-invasive diagnostic tools has expanded with innovations in contrast agents; molecular radionuclide imaging; perfusion echocardiography; CT for coronary angiography, cardiac structure and morphology, and calcium scoring and CMR for myocardial structure, function, and viability. These advances present new opportunities for physicians to utilize non-invasive techniques to gain important information about the condition of their patients. However, in the case of CCT and CMR, both tests are relatively expensive technologies, especially with regards to imaging equipment. Additionally, the potential for uncontrolled utilization and stimulation of downstream testing and treatment such as unwarranted coronary revascularization has raised substantial concern from government and private payers as well as clinical thought leaders of evidenced-based cardiovascular medicine. As each of these imaging modalities becomes clinically available, the health care community needs to understand how to incorporate these advances into acceptable clinical care.

Both CCT and CMR have been recognized as having a number of potential uses and advantages over existing technology. Coronary calcium scoring performed with either electron beam CT or multidetector row CT is one application that has gained some acceptance, despite the lack of reimbursement from most payers. Still, there has been, to date, little expert consensus regarding for whom this method is of clinical benefit. Computed tomographic angiography, while very promising with regard to the detection of coronary stenoses, definition of "soft plaque," assessment of left ventricular function and congenital coronary anomalies, and evaluation of cardiac structures, has limited data supporting its use for many clinical applications, especially with regard to its role within patient care algorithms. Cardiac magnetic resonance imaging, although continuing to demonstrate clinical utility, has been used primarily in specialized centers and, until recently, has had its major role in clinical research evaluating myocardial viability and cardiac structure and function. Cardiac magnetic resonance also has been found useful in the evaluation of ischemic heart disease with vasodilator stress perfusion imaging and dobutamine stress function imaging.

In an effort to respond to the need for the rational use of these newer imaging techniques, CCT and CMR, the ACCF, in conjunction with the societies listed on this report, undertook a process to determine the appropriateness of selected indications for these rapidly evolving cardiovascular imaging procedures. The Appropriateness Criteria Project was initiated to support the delivery of quality cardiovascular care and to ensure the effective use of diagnostic imaging tools, and it is an ongoing effort by ACCF to rigorously examine the appropriateness of all established imaging modalities.

METHODS

A detailed description of the methods used for ranking of the clinical indications is outlined in Appendices A and B and also more generally can be found in a previous publication entitled, "ACCF Proposed Method for Evaluating the Appropriateness of Cardiovascular Imaging" (2). Briefly, this process blends scientific evidence and practice experience by engaging a Technical Panel in a modified Delphi exercise. The Technical Panel was purposely balanced with a diverse set of individuals who ranged from imaging specialists within the CCT and CMR community including cardiologists and radiologists to referring physicians, health services researchers, and a medical director from a private payer. The panel members are highlighted in Appendix C.

The 39 CCT and 33 CMR indications that were rated are thought to encompass the majority of cases referred for CCT and CMR, respectively. They were constructed by several experts within the field and were modified slightly based on discussions of the Working Group, indication reviewers, and the panelists who rated the indications. Although not comprehensive, they are characteristic of contemporary practice. They include symptomatic patients stratified by pre-test probability of disease, asymptomatic patients based on Framingham risk, and patient presentation for assessment of structure and function, including coronary artery anomalies (3–7).

A reference list of key publications within the fields of CCT and CMR was provided to the raters. Additionally, evidence tables for various applications, as well as factual summaries of the potential uses of the test were distributed to the raters (online Appendix C and D at www.acc.org). Care was given to provide objective, non-biased information.

The panelists were asked to assess whether the use of CCT and CMR for various indications was appropriate, uncertain, or inappropriate. In rating each indication, the panel was provided the following definition of appropriateness:

An appropriate imaging study is one in which the expected incremental information, combined with clinical judgment, exceeds the expected negative consequences* by a sufficiently wide margin for a specific indication that the procedure is generally considered acceptable care and a reasonable approach for the indication.

*Negative consequences include the risks of the procedure (i.e., radiation or contrast exposure) and the downstream impact of poor test performance such as delay in diagnosis (false negatives) or inappropriate diagnosis (false positives).

The Technical Panel scored each indication as follows:

Score 7 to 9

Appropriate test for specific indication (test is generally acceptable and is a reasonable approach for the indication).

Score 4 to 6

Uncertain for specific indication (test may be generally acceptable and may be a reasonable approach for the indication). (Uncertainty also implies that more research and/or patient information is needed to classify the indication definitively.)

Score 1 to 3

Inappropriate test for that indication (test is not generally acceptable and is not a reasonable approach for the indication).

RESULTS OF RATINGS

The final ratings for CCT (Tables 1 to 8) and CMR (Tables 12 to 17) are listed by indication sequentially, by purpose and clinical scenario, as obtained from the second round rating sheets submitted by each panelist. In addition, Tables 9 to 11 and 18 to 20 arrange the indications into 3 main scoring categories (appropriate [median score of 7 to 9], uncertain [median score of 4 to 6], and inappropriate [median score of 1 to 3]) for CCT and CMR, respectively. Other tables, including documentation of the mean absolute deviation from the median and level of agreement for each indication, are found in the online Appendices A and B at www.acc.org. Abbreviations used in the tables and the text of this report are listed below.

Abbreviations

ACS = acute coronary syndromes
CABG = coronary artery bypass grafting surgery
CAD = coronary artery disease
CCT = cardiac computed tomography
CHD = coronary heart disease
CMR = cardiac magnetic resonance imaging
CT = computed tomography
EBCT = electron beam computed tomography
ECG = electrocardiogram
HF = heart failure
ICD-9 = International Classification of Diseases-9th Revision
LCD = local coverage determination
METs = estimated metabolic equivalents of exercise
MI = myocardial infarction
MPI = myocardial perfusion imaging
NSTEMI = non−ST-segment elevation myocardial infarction
PCI = percutaneous coronary intervention
SPECT MPI = single-photon emission computed tomography myocardial perfusion imaging
STEMI = ST-segment elevation myocardial infarction
TEE = transesophageal echocardiography

CCT APPROPRIATENESS CRITERIA (BY INDICATION)

Assume the logical operator between each variable listed for an indication is "**AND**" unless otherwise noted (e.g., Low pre-test probability of CAD **AND** No ECG changes and serial enzymes negative).

Table 1. Detection of CAD: Symptomatic

Indication		Appropriateness Criteria (Median Score)
Evaluation of Chest Pain Syndrome (Use of CT Angiogram)		
1.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	U (5)
2.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	A (7)
3.	• High pre-test probability of CAD	I (2)
Evaluation of Intra-Cardiac Structures (Use of CT Angiogram)		
4.	• Evaluation of suspected coronary anomalies	A (9)
Acute Chest Pain (Use of CT Angiogram)		
5.	• Low pre-test probability of CAD • No ECG changes and serial enzymes negative	U (5)
6.	• Intermediate pre-test probability of CAD • No ECG changes and serial enzymes negative	A (7)
7.	• High pre-test probability of CAD • No ECG changes and serial enzymes negative	U (6)
8.	• High pre-test probability of CAD • ECG—ST-segment elevation and/or positive cardiac enzymes	I (1)
9.	• "Triple rule out"—exclude obstructive CAD, aortic dissection, and pulmonary embolism • Intermediate pre-test probability for one of the above • ECG—no ST-segment elevation and initial enzymes negative	U (4)

Table 2. Detection of CAD: Asymptomatic (Without Chest Pain Syndrome)

Indication		Appropriateness Criteria (Median Score)
Asymptomatic (Use of CT Angiogram)		
10.	• Low CHD risk (Framingham risk criteria)	I (1)
11.	• Moderate CHD risk (Framingham)	I (2)
12.	• High CHD risk (Framingham)	U (4)

Table 3. Risk Assessment: General Population

Indication		Appropriateness Criteria (Median Score)
Asymptomatic (Calcium Scoring)		
13.	• Low CHD risk (Framingham)	I (1)
14.	• Moderate CHD risk (Framingham)	U (6)
15.	• High CHD risk (Framingham)	U (5)

Table 4. Detection of CAD With Prior Test Results

Indication		Appropriateness Criteria (Median Score)
Evaluation of Chest Pain Syndrome (Use of CT Angiogram)		
16.	• Uninterpretable or equivocal stress test (exercise, perfusion, or stress echo)	A (8)
17.	• Evidence of moderate to severe ischemia on stress test (exercise, perfusion, or stress echo)	I (2)

Table 5. Risk Assessment With Prior Test Results

Indication		Appropriateness Criteria (Median Score)
Asymptomatic (Calcium Scoring)		
18.	• Prior calcium score within previous 5 years	I (1)
Asymptomatic (Use of CT Angiogram)		
19.	• High CHD risk (Framingham) • Within 2 years prior cardiac CT angiogram or invasive angiogram without significant obstructive disease	I (2)
20.	• High CHD risk (Framingham) • Prior calcium score greater than or equal to 400	I (3)

Table 6. Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery

Indication		Appropriateness Criteria (Median Score)
Low-Risk Surgery (Use of CT Angiogram)		
21.	• Intermediate perioperative risk	I (1)
Intermediate- or High-Risk Surgery (Use of CT Angiogram)		
22.	• Intermediate perioperative risk	U (4)

Table 7. Detection of CAD: Post-Revascularization (PCI or CABG)

Indication		Appropriateness Criteria (Median Score)
Evaluation of Chest Pain Syndrome (Use of CT Angiogram)		
23.	• Evaluation of bypass grafts and coronary anatomy	U (6)
24.	• History of percutaneous revascularization with stents	U (5)
Asymptomatic (Use of CT Angiogram)		
25.	• Evaluation of bypass grafts and coronary anatomy • Less than 5 years after CABG	I (2)
26.	• Evaluation of bypass grafts and coronary anatomy • Greater than or equal to 5 years after CABG	I (3)
27.	• Evaluation for in-stent restenosis and coronary anatomy after PCI	I (2)

Table 8. Structure and Function

Indication		Appropriateness Criteria (Median Score)
Morphology (Use of CT Angiogram)		
28.	• Assessment of complex congenital heart disease including anomalies of coronary circulation, great vessels, and cardiac chambers and valves	A (7)
29.	• Evaluation of coronary arteries in patients with new onset heart failure to assess etiology	A (7)
Evaluation of Ventricular and Valvular Function (Use of CT Angiogram)		
30.	• Evaluation of LV function following myocardial infarction OR in heart failure patients	I (3)
31.	• Evaluation of LV function following myocardial infarction OR in heart failure patients • Patients with technically limited images from echocardiogram	U (5)
32.	• Characterization of native and prosthetic cardiac valves • Patients with technically limited images from echocardiogram, MRI, or TEE	U (5)
Evaluation of Intra- and Extra-Cardiac Structures (Use of Cardiac CT)		
33.	• Evaluation of cardiac mass (suspected tumor or thrombus) • Patients with technically limited images from echocardiogram, MRI, or TEE	A (8)
34.	• Evaluation of pericardial conditions (pericardial mass, constrictive pericarditis, or complications of cardiac surgery) • Patients with technically limited images from echocardiogram, MRI, or TEE	A (8)
35.	• Evaluation of pulmonary vein anatomy prior to invasive radiofrequency ablation for atrial fibrillation	A (8)
36.	• Noninvasive coronary vein mapping prior to placement of biventricular pacemaker	A (8)
37.	• Noninvasive coronary arterial mapping, including internal mammary artery prior to repeat cardiac surgical revascularization	A (8)
Evaluation of Aortic and Pulmonary Disease (Use of CT Angiogram*)		
38.	• Evaluation of suspected aortic dissection or thoracic aortic aneurysm	A (9)
39.	• Evaluation of suspected pulmonary embolism	A (9)

*Non-gated, CT angiogram which has a sufficiently large field of view for these specific indications.

CCT APPROPRIATENESS CRITERIA (BY APPROPRIATENESS CATEGORY)

Table 9. Inappropriate Indications (Median Score 1–3)

Indication		Appropriateness Criteria (Median Score)
**Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)**	
3.	• High pre-test probability of CAD	**I (2)**
**Detection of CAD: Symptomatic—Acute Chest Pain (Use of CT Angiogram)**	
8.	• High pre-test probability of CAD • ECG—ST-segment elevation and/or positive cardiac enzymes	**I (1)**
**Detection of CAD: Asymptomatic (Without Chest Pain Syndrome)—Asymptomatic (Use of CT Angiogram)**	
10.	• Low CHD risk (Framingham risk criteria)	**I (1)**
11.	• Moderate CHD risk (Framingham)	**I (2)**
**Risk Assessment: General Population—Asymptomatic (Calcium Scoring)**	
13.	• Low CHD risk (Framingham)	**I (1)**
**Detection of CAD With Prior Test Results—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)**	
17.	• Evidence of moderate to severe ischemia on stress test (exercise, perfusion, or stress echo)	**I (2)**
**Risk Assessment With Prior Test Results—Asymptomatic (Calcium Scoring)**	
18.	• Prior calcium score within previous 5 years	**I (1)**
**Risk Assessment With Prior Test Results—Asymptomatic (Use of CT Angiogram)**	
19.	• High CHD risk (Framingham) • Within 2 years prior cardiac CT angiogram or invasive angiogram without significant obstructive disease	**I (2)**
20.	• High CHD risk (Framingham) • Prior calcium score greater than or equal to 400	**I (3)**
**Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery—Low-Risk Surgery (Use of CT Angiogram)**	
21.	• Intermediate perioperative risk	**I (1)**
**Detection of CAD: Post-Revascularization (PCI or CABG)—Asymptomatic (Use of CT Angiogram)**	
25.	• Evaluation of bypass grafts and coronary anatomy • Less than 5 years after CABG	**I (2)**
26.	• Evaluation of bypass grafts and coronary anatomy • Greater than or equal to 5 years after CABG	**I (3)**
27.	• Evaluation for in-stent restenosis and coronary anatomy after PCI	**I (2)**
**Structure and Function—Evaluation of Ventricular and Valvular Function (Use of CT Angiogram)**	
30.	• Evaluation of LV function following myocardial infarction OR in heart failure patients	**I (3)**

Table 10. Appropriate Indications (Median Score 7–9)

Indication		Appropriateness Criteria (Median Score)
**Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)**
2.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	A (7)
**Detection of CAD: Symptomatic—Evaluation of Intra-Cardiac Structures (Use of CT Angiogram)**
4.	• Evaluation of suspected coronary anomalies	A (9)
**Detection of CAD: Symptomatic—Acute Chest Pain (Use of CT Angiogram)**
6.	• Intermediate pre-test probability of CAD • No ECG changes and serial enzymes negative	A (7)
**Detection of CAD With Prior Test Results—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)**
16.	• Uninterpretable or equivocal stress test (exercise, perfusion, or stress echo)	A (8)
**Structure and Function—Morphology (Use of CT Angiogram)**
28.	• Assessment of complex congenital heart disease including anomalies of coronary circulation, great vessels, and cardiac chambers and valves	A (7)
29.	• Evaluation of coronary arteries in patients with new onset heart failure to assess etiology	A (7)
**Structure and Function—Evaluation of Intra- and Extra-Cardiac Structures (Use of Cardiac CT)**
33.	• Evaluation of cardiac mass (suspected tumor or thrombus) • Patients with technically limited images from echocardiogram, MRI, or TEE	A (8)
34.	• Evaluation of pericardial conditions (pericardial mass, constrictive pericarditis, or complications of cardiac surgery) • Patients with technically limited images from echocardiogram, MRI, or TEE	A (8)
35.	• Evaluation of pulmonary vein anatomy prior to invasive radiofrequency ablation for atrial fibrillation	A (8)
36.	• Noninvasive coronary vein mapping prior to placement of biventricular pacemaker	A (8)
37.	• Noninvasive coronary arterial mapping, including internal mammary artery prior to repeat cardiac surgical revascularization	A (8)
**Structure and Function—Evaluation of Aortic and Pulmonary Disease (Use of CT Angiogram*)**
38.	• Evaluation of suspected aortic dissection or thoracic aortic aneurysm	A (9)
39.	• Evaluation of suspected pulmonary embolism	A (9)

*Non-gated, CT angiogram which has a sufficiently large field of view for these specific indications.

Table 11. Uncertain Indications (Median Score 4−6)

Indication		Appropriateness Criteria (Median Score)
Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)		
1.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	U (5)
Detection of CAD: Symptomatic—Acute Chest Pain (Use of CT Angiogram)		
5.	• Low pre-test probability of CAD • No ECG changes and serial enzymes negative	U (5)
7.	• High pre-test probability of CAD • No ECG changes and serial enzymes negative	U (6)
9.	• "Triple rule out"—exclude obstructive CAD, aortic dissection, and pulmonary embolism • Intermediate pre-test probability for one of the above • ECG—no ST-segment elevation and initial enzymes negative	U (4)
Detection of CAD: Asymptomatic (Without Chest Pain Syndrome)—Asymptomatic (Use of CT Angiogram)		
12.	• High CHD risk (Framingham)	U (4)
Risk Assessment: General Population—Asymptomatic (Calcium Scoring)		
14.	• Moderate CHD risk (Framingham)	U (6)
15.	• High CHD risk (Framingham)	U (5)
Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery—Intermediate or High Risk Surgery (Use of CT Angiogram)		
22.	• Intermediate perioperative risk	U (4)
Detection of CAD: Post-Revascularization (PCI or CABG)—Evaluation of Chest Pain Syndrome (Use of CT Angiogram)		
23.	• Evaluation of bypass grafts and coronary anatomy	U (6)
24.	• History of percutaneous revascularization with stents	U (5)
Structure and Function—Evaluation of Ventricular and Valvular Function (Use of CT Angiogram)		
31.	• Evaluation of LV function following myocardial infarction OR in heart failure patients • Patients with technically limited images from echocardiogram	U (5)
32.	• Characterization of native and prosthetic cardiac valves • Patients with technically limited images from echocardiogram, MRI, or TEE	U (5)

CMR APPROPRIATENESS CRITERIA (BY INDICATION)

Assume the logical operator between each variable listed for an indication is **"AND"** unless otherwise noted (e.g., Low pre-test probability of CAD **AND** No ECG changes and serial enzymes negative).

Table 12. Detection of CAD: Symptomatic

Indication		Appropriateness Criteria (Median Score)
Evaluation of Chest Pain Syndrome (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
1.	• Low pre-test probability of CAD • ECG interpretable AND able to exercise	I (2)
2.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	U (4)
3.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	A (7)
4.	• High pre-test probability of CAD	U (5)
Evaluation of Chest Pain Syndrome (Use of MR Coronary Angiography)		
5.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	I (2)
6.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	I (2)
7.	• High pre-test probability of CAD	I (1)
Evaluation of Intra-Cardiac Structures (Use of MR Coronary Angiography)		
8.	• Evaluation of suspected coronary anomalies	A (8)
Acute Chest Pain (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
9.	• Intermediate pre-test probability of CAD • No ECG changes and serial cardiac enzymes negative	U (6)
10.	• High pre-test probability of CAD • ECG—ST-segment elevation and/or positive cardiac enzymes	I (1)

Table 13. Risk Assessment With Prior Test Results (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)

Indication		Appropriateness Criteria (Median Score)
11.	• Normal prior stress test (exercise, nuclear, echo, MRI) • High CHD risk (Framingham) • Within 1 year of prior stress test	I (2)
12.	• Equivocal stress test (exercise, stress SPECT, or stress echo) • Intermediate CHD risk (Framingham)	U (6)
13.	• Coronary angiography (catheterization or CT) • Stenosis of unclear significance	A (7)

Table 14. Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery

Indication		Appropriateness Criteria (Median Score)
Low-Risk Surgery (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
14.	• Intermediate perioperative risk predictor	**I (2)**
Intermediate- or High-Risk Surgery (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
15.	• Intermediate perioperative risk predictor	**U (6)**

Table 15. Detection of CAD: Post-Revascularization (PCI or CABG)

Indication		Appropriateness Criteria (Median Score)
Evaluation of Chest Pain Syndrome (Use of MR Coronary Angiography)		
16.	• Evaluation of bypass grafts	**I (2)**
17.	• History of percutaneous revascularization with stents	**I (1)**

Table 16. Structure and Function

Indication		Appropriateness Criteria (Median Score)
Evaluation of Ventricular and Valvular Function Procedures may include LV/RV mass and volumes, MR angiography, quantification of valvular disease, and delayed contrast enhancement		
18.	• Assessment of complex congenital heart disease including anomalies of coronary circulation, great vessels, and cardiac chambers and valves • Procedures may include LV/RV mass and volumes, MR angiography, quantification of valvular disease, and contrast enhancement	**A (9)**
19.	• Evaluation of LV function following myocardial infarction OR in heart failure patients	**U (6)**
20.	• Evaluation of LV function following myocardial infarction OR in heart failure patients • Patients with technically limited images from echocardiogram	**A (8)**
21.	• Quantification of LV function • Discordant information that is clinically significant from prior tests	**A (8)**
22.	• Evaluation of specific cardiomyopathies (infiltrative [amyloid, sarcoid], HCM, or due to cardiotoxic therapies) • Use of delayed enhancement	**A (8)**
23.	• Characterization of native and prosthetic cardiac valves—including planimetry of stenotic disease and quantification of regurgitant disease • Patients with technically limited images from echocardiogram or TEE	**A (8)**
24.	• Evaluation for arrythmogenic right ventricular cardiomyopathy (ARVC) • Patients presenting with syncope or ventricular arrhythmia	**A (9)**
25.	• Evaluation of myocarditis or myocardial infarction with normal coronary arteries • Positive cardiac enzymes without obstructive atherosclerosis on angiography	**A (8)**
Evaluation of Intra- and Extra-Cardiac Structures		
26.	• Evaluation of cardiac mass (suspected tumor or thrombus) • Use of contrast for perfusion and enhancement	**A (9)**
27.	• Evaluation of pericardial conditions (pericardial mass, constrictive pericarditis)	**A (8)**
28.	• Evaluation for aortic dissection	**A (8)**
29.	• Evaluation of pulmonary veins prior to radiofrequency ablation for atrial fibrillation • Left atrial and pulmonary venous anatomy including dimensions of veins for mapping purposes	**A (8)**

Table 17. Detection of Myocardial Scar and Viability

Indication		Appropriateness Criteria (Median Score)
Evaluation of Myocardial Scar (Use of Late Gadolinium Enhancement)		
30.	• To determine the location and extent of myocardial necrosis including 'no reflow' regions • Post-acute myocardial infarction	**A (7)**
31.	• To detect post PCI myocardial necrosis	**U (4)**
32.	• To determine viability prior to revascularization • Establish likelihood of recovery of function with revascularization (PCI or CABG) or medical therapy	**A (9)**
33.	• To determine viability prior to revascularization • Viability assessment by SPECT or dobutamine echo has provided "equivocal or indeterminate" results	**A (9)**

CMR APPROPRIATENESS CRITERIA (BY APPROPRIATENESS CATEGORY)

Table 18. Inappropriate Indications (Median Score 1–3)

Indication		Appropriateness Criteria (Median Score)
Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
1.	• Low pre-test probability of CAD • ECG interpretable AND able to exercise	**I (2)**
Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of MR Coronary Angiography)		
5.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	**I (2)**
6.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	**I (2)**
7.	• High pre-test probability of CAD	**I (1)**
Detection of CAD: Symptomatic—Acute Chest Pain (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
10.	• High pre-test probability of CAD • ECG—ST-segment elevation and/or positive cardiac enzymes	**I (1)**
Risk Assessment With Prior Test Results (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
11.	• Normal prior stress test (exercise, nuclear, echo, MRI) • High CHD risk (Framingham) • Within 1 year of prior stress test	**I (2)**
Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery—Low Risk Surgery (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
14.	• Intermediate perioperative risk predictor	**I (2)**
Detection of CAD: Post-Revascularization (PCI or CABG)—Evaluation of Chest Pain Syndrome (Use of MR Coronary Angiography)		
16.	• Evaluation of bypass grafts	**I (2)**
17.	• History of percutaneous revascularization with stents	**I (1)**

Table 19. Appropriate Indications (Median Score 7–9)

Indication		Appropriateness Criteria (Median Score)
**Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)**		
3.	• Intermediate pre-test probability of CAD • ECG uninterpretable OR unable to exercise	A (7)
**Detection of CAD: Symptomatic—Evaluation of Intra-Cardiac Structures (Use of MR Coronary Angiography)**		
8.	• Evaluation of suspected coronary anomalies	A (8)
**Risk Assessment With Prior Test Results (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)**		
13.	• Coronary angiography (catheterization or CT) • Stenosis of unclear significance	A (7)
**Structure and Function—Evaluation of Ventricular and Valvular Function** Procedures may include LV/RV mass and volumes, MR angiography, quantification of valvular disease, and delayed contrast enhancement		
18.	• Assessment of complex congenital heart disease including anomalies of coronary circulation, great vessels, and cardiac chambers and valves • Procedures may include LV/RV mass and volumes, MR angiography, quantification of valvular disease, and contrast enhancement	A (9)
20.	• Evaluation of LV function following myocardial infarction OR in heart failure patients • Patients with technically limited images from echocardiogram	A (8)
21.	• Quantification of LV function • Discordant information that is clinically significant from prior tests	A (8)
22.	• Evaluation of specific cardiomyopathies (infiltrative [amyloid, sarcoid], HCM, or due to cardiotoxic therapies) • Use of delayed enhancement	A (8)
23.	• Characterization of native and prosthetic cardiac valves—including planimetry of stenotic disease and quantification of regurgitant disease • Patients with technically limited images from echocardiogram or TEE	A (8)
24.	• Evaluation for arrythmogenic right ventricular cardiomyopathy (ARVC) • Patients presenting with syncope or ventricular arrhythmia	A (9)
25.	• Evaluation of myocarditis or myocardial infarction with normal coronary arteries • Positive cardiac enzymes without obstructive atherosclerosis on angiography	A (8)
**Structure and Function—Evaluation of Intra- and Extra-Cardiac Structures**		
26.	• Evaluation of cardiac mass (suspected tumor or thrombus) • Use of contrast for perfusion and enhancement	A (9)
27.	• Evaluation of pericardial conditions (pericardial mass, constrictive pericarditis)	A (8)
28.	• Evaluation for aortic dissection	A (8)
29.	• Evaluation of pulmonary veins prior to radiofrequency ablation for atrial fibrillation • Left atrial and pulmonary venous anatomy including dimensions of veins for mapping purposes	A (8)
**Detection of Myocardial Scar and Viability—Evaluation of Myocardial Scar (Use of Late Gadolinium Enhancement)**		
30.	• To determine the location, and extent of myocardial necrosis including 'no reflow' regions • Post acute myocardial infarction	A (7)
32.	• To determine viability prior to revascularization • Establish likelihood of recovery of function with revascularization (PCI or CABG) or medical therapy	A (9)
33.	• To determine viability prior to revascularization • Viability assessment by SPECT or dobutamine echo has provided "equivocal or indeterminate" results	A (9)

Table 20. Uncertain Indications (Median Score 4−6)

Indication		Appropriateness Criteria (Median Score)
Detection of CAD: Symptomatic—Evaluation of Chest Pain Syndrome (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
2.	• Intermediate pre-test probability of CAD • ECG interpretable AND able to exercise	U (4)
4.	• High pre-test probability of CAD	U (5)
Detection of CAD: Symptomatic—Acute Chest Pain (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
9.	• Intermediate pre-test probability of CAD • No ECG changes and serial cardiac enzymes negative	U (6)
Risk Assessment With Prior Test Results (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
12.	• Equivocal stress test (exercise, stress SPECT, or stress echo) • Intermediate CHD risk (Framingham)	U (6)
Risk Assessment: Preoperative Evaluation for Non-Cardiac Surgery—Intermediate or High Risk Surgery (Use of Vasodilator Perfusion CMR or Dobutamine Stress Function CMR)		
15.	• Intermediate perioperative risk predictor	U (6)
Structure and Function—Evaluation of Ventricular and Valvular Function Procedures may include LV/RV mass and volumes, MR angiography, quantification of valvular disease, and delayed contrast enhancement		
19.	• Evaluation of LV function following myocardial infarction OR in heart failure patients	U (6)
Evaluation of Myocardial Scar (Use of Late Gadolinium Enhancement)		
31.	• To detect post PCI myocardial necrosis	U (4)

DISCUSSION

The indications contained in this report were selected to cover a wide variety of clinical presentations. They are based on common patient presentations such as symptoms suggestive of ischemia, multiple cardiac risk factors in an asymptomatic individual, and specific scenarios with indices of high clinical suspicion that are further stratified based on factors such as clinical risk, prior test results, and the interval since prior testing. The purpose of this approach is to delineate the possible value of CCT or CMR for a physician faced with everyday patient scenarios. The indications do not correspond directly to International Classification of Diseases-9th Revision (ICD-9) codes, as they convey more information than usually found in the ICD-9 classification system. Some correlation with previous model local coverage determination (LCD) documents is purposeful, but the indications are designed to provide further guidance within the categories outlined in the model LCD for ordering physicians. It is recognized that not all categories within an LCD or for ICD-9 codes are represented.

The appropriateness criteria for CCT and CMR are 2 separate reports and were not developed in a way that can provide comparative information about the utility of one test versus the other. Although the same panel ranked the indications for both CCT and CMR, members of the Technical Panel were asked specifically NOT to comparatively rank each of these imaging procedures, but instead to consider each test on its own merits. As such, the scores and

the conclusions about appropriateness also should not be compared with the prior report for appropriateness for single-photon emission computed tomography myocardial perfusion imaging (SPECT MPI) (8) or to those soon to be written for other imaging procedures, such as echocardiography.

For the 39 indications for CCT, 13 were found to be appropriate, and 12 were uncertain. Fourteen of these indications were felt to be inappropriate reasons for CT test performance. There was great variability in scores for the uncertain category, suggesting markedly differing opinions. However, there was substantial agreement as defined by RAND (9) for a panel this size for the categories labeled as either appropriate or inappropriate, with 77% and 86%, respectively, showing agreement. Cardiac computed tomography was considered reasonable for a number of scenarios beyond assessments of structure and function, but still over 40% of the indications were for this area.

For CMR, 17 of the 33 indications were ranked as appropriate, with another 7 being uncertain. Nine scenarios were considered to be inappropriate reasons for magnetic resonance test performance. Similar to the indications for CCT, uncertain scenarios showed wider dispersion of scores than those for indications at either end of the spectrum. Agreement, as defined for a panel this size by RAND (9), was present for 82% of the appropriate indications and 89% for those felt to be inappropriate. Two-thirds of the appropriate and uncertain indications for CMR were related to

assessment of structure and function. These results support the strengths of CMR as a tool for defining the etiology of complex patient presentations where the clinical suspicion is high. The scores for other uses reflect the evolving nature of the capabilities of the test.

The indications contained in this report are not exhaustive. For example, the use of CCT or CMR for the non-invasive evaluation of coronary arteries before non-coronary cardiac surgery was not listed as an indication, although this may be an evolving application. Additionally, there may be medical reasons that would preclude the application of the appropriateness criteria to a specific patient, and clinician judgment should be used at all times in the application of these criteria. Furthermore, the local availability or quality of equipment or personnel may influence the selection of appropriate imaging procedures. Appropriateness criteria, in other words, are not substitutes for sound clinical judgment and practice experience with each patient and clinical presentation. For example, the rating of an indication as inappropriate should not preclude a provider from performing CCT or CMR procedures when there are patient- and condition-specific data to support that decision. Conversely, not doing a study that is deemed appropriate may be the correct decision in light of unique patient, clinical, and other relevant information.

The category of "uncertain" was discussed at length by the Technical Panel and the Working Group. The consensus of the Panel was that this intermediate level of appropriateness should be labeled "uncertain," as either critical data were lacking or significant differences of opinion exist among Panel members regarding the value of the method for that particular indication. The categorization of a particular indication as uncertain should serve as a nidus for additional information and research so as to formulate a definitive level of appropriateness.

The primary objective of this report is to provide guidance regarding the perceived suitability of CCT and CMR for diverse clinical scenarios. As with the Appropriateness Criteria for SPECT MPI (8), consensus among the raters was desirable, but achievement of complete agreement within this diverse panel would have been artificial and not necessarily of clinical value. Two rounds of rating with intervening discussion did lead to some consensus. However, further attempts to drive consensus might have artificially diluted true differences in opinion among panelists. This is especially true for both CCT and CMR, as these are still emerging clinical imaging modalities with an evolving evidence base.

The appropriateness criteria in these reports are expected to be useful for clinicians, health care facilities, and third-party payers in the delivery of quality cardiovascular imaging. For example, individual clinicians could use the ratings as a supportive decision or educational tool when ordering a test or providing a referral to another qualified physician. The criteria also may be used to respond to a referring physician who has ordered a test for an inappropriate

indication. Facilities and payers can use the criteria either prospectively in the design of protocols and pre-authorization procedures or retrospectively for quality reports. It is hoped that payers will use this document as the basis for their own strategies to ensure that their members receive quality, but cost-effective, cardiovascular care.

When used for accountability, appropriateness criteria should be used in conjunction with systems that support quality improvement. Prospective pre-authorization procedures, for example, may be used most effectively once a retrospective review has identified a pattern of potential inappropriate use. Because the criteria are based on up-to-date scientific evidence and the deliberations of the Technical Panel, they can be used to help resolve future reimbursement cases or appeals but should not be applied to cases completed before issuance of this report.

The linking of indications rated as generally acceptable practice with analysis of related patient outcomes, and a review of what is "necessary" care, will improve understanding of regional variations in imaging and the potential for ensuring the equitable and efficient allocation of resources for diagnostic studies. Further exploration of the indications that are rated as "uncertain" will generate new empirical research and the data required to further define the appropriateness of CCT and CMR. Finally, periodic assessment and updating of the indications and criteria will be required as new data and field experience become available.

APPENDIX A: METHODS

Panel Selection

An initial list of potential Technical Panel members was generated based on a call for nominations issued to all relevant stakeholders. Panel members were selected by the Working Group in a manner that ensured an appropriate balance with respect to expertise in the specific modality, academic versus private practice, health services research, and specialty training.

Development of Indications

The process for creating a robust set of indications involved consulting current literature, previously published statements, and model local coverage determination documents. The indications capture the majority of scenarios faced by cardiologists or referring physicians, but are not meant to be inclusive of all potential indications for which CCT or CMR imaging studies may be performed. Review was done by the Working Group, including additional comments from external reviewers. As a result of the meeting of the Technical Panel before the second round of rating, a few of the indications were clarified and modified. A final set of indications comprised the list of possible clinical scenarios that were rated for appropriateness by the panelists and compiled for this report.

General Assumptions

All indications for CCT and CMR were considered with the following important assumptions:

1. All indications should first be evaluated based on the available medical literature. In many cases, studies are reflections of the capabilities and limitations of the test but provide minimal information about the role of the test in clinical decision making. Appropriateness criteria development requires determination of a <u>reasonable</u> course of action for clinical decision making based on a risk/benefit trade-off as determined by individual patient indications.
2. Cost **SHOULD** be considered **implicitly** in the appropriateness determination.
3. Risks, such as radiation exposure and contrast adverse effects, should be considered.
4. Additional factors may be considered **implicitly** in the appropriateness determination including the impact of the image on clinical decision making when combined with clinical judgment.
5. For each indication, the panelists' ratings should reflect whether the test is reasonable for the patient according to the appropriateness definition, **not whether the test is better or worse than another**. It also should not consider issues of local availability or skill for any modality or variation in equipment. It should be assumed that the imaging procedure will be performed in accordance with best practice, using appropriate equipment and techniques.
6. Specific comparisons with previous sets of appropriateness criteria should **not** be made.
7. **All techniques** are assumed to be performed in an optimal fashion, using appropriate equipment and protocols.
8. The test is assumed to be performed by a qualified individual in a facility that is proficient in the imaging technique.

Assumptions for CCT only:

1. Cardiac computed tomography imaging equipment and personnel are available that have the minimal technical capabilities required for the indication (the number of detector rows, spatial and temporal resolution, and acquisition protocols).
2. Indications for CT angiography assume that calcium scoring also may be obtained for that indication.
3. Calcium scoring is assumed to be performed by EBCT or multislice CT.
4. Unless specifically noted, use of the test to determine non-cardiac etiologies for an indication is not considered.
5. For CT angiography, patients are assumed not to present with any of the following:
   a. Irregular rhythm (e.g., atrial fibrillation/flutter, frequent irregular premature ventricular contractions or premature atrial contractions, and high grade heart block);

b. Very obese patients, body mass index greater than 40 kg/m²;
   c. Renal insufficiency, creatinine greater than 1.8 mg/dL;
   d. Heart rate greater than 70 beats/min refractory to heart-rate-lowering agents (e.g., a combination of beta-blocker and calcium-channel blocker);
   e. Metallic interference (e.g., surgical clips, pacemaker, and/or defibrillator wires, or tissue expander.

6. For CT angiography, patients must be able to:
   a. Hold still;
   b. Follow breathing instruction;
   c. Take nitroglycerin (for performing coronary CT angiography only);
   d. Take iodine in spite of steroid prep for contrast allergy;
   e. Lift both arms above the shoulders.

Note: Any patient presenting with the characteristics listed in 5 and 6 above is assumed to be excluded from the indications for scoring purposes.

Assumptions for CMR only:

1. Cardiac magnetic resonance imaging equipment and personnel are available that have the minimal technical capabilities required for the indication.
2. Images are obtained with at least a 1.5-T magnet using standard sequences provided by the current vendors.
3. Use of gadolinium contrast is assumed for studies involving perfusion, angiograms, and contrast enhancement.
4. Patients are assumed not to present with general CMR imaging contraindications examples of which include:
   a. severe claustrophobia;
   b. specific metallic contraindications such as pacemakers, defibrillators, and certain aneurysm clips.

Note: Studies are ongoing with regards to pacemakers and implantable defibrillators. In April 2005, the Food and Drug Administration approved magnetic resonance imaging studies immediately after implantation of sirolimus- and paclitaxel-eluting stents, which is now reflected in the respective package instructions for use.

Rating Process

The Technical Panel was instructed to follow the process outlined in the article previously published by the College entitled, "ACCF Proposed Method for Evaluating the Appropriateness of Cardiovascular Imaging" (2). The appropriateness method combines expert clinical judgment with the scientific literature in evaluating the benefits and risks of medical procedures. Ratings of the net benefits and risks of performing medical procedures for a comprehensive array of potential patient indications or scenarios are obtained from a multidisciplinary panel of expert clinicians. Each panel member has equal weight in producing the final result, and the method does not force consensus.

The rating process includes a modified Delphi process involving 2 rounds of ratings and an intervening face-to-face meeting. The first round of ratings was completed individually with no interaction among panel members. The panel was then convened for a face-to-face meeting that was facilitated by a moderator. The goal of the meeting was to focus discussion on indications for which the first round scores of the panel were widely divergent. The objective of the meeting was to allow all views to be heard. The second round ratings were conducted individually subsequent to the face-to-face meeting. The second round ratings were used to determine the final appropriateness score based on the median score for each indication.

At the face-to-face meeting, each panelist received a personalized rating form that indicated his/her rating for each indication and the distribution of ratings of other members of the panel, but without personal identification. In addition, the moderator received a summary rating form with similar information (including panelist identification), along with other statistics that measured the level of agreement among panel members. A measure of the level of disagreement was applied to each score after both the first and second round scoring was completed. This project employed the BIOMED Concerted Action on Appropriateness definition for a panel size of 14 to 16. As defined in the RAND/UCLA manual (9) upon which the ACCF ratings method is based, the BIOMED rule for agreement (+) is that no more than 4 panelists rate the indication outside the 3-point region containing the median; for disagreement (−), at least 5 panelists rate in each extreme rating region (i.e., 1 to 3 and 7 to 9). Measures of agreement and the dispersion of ratings (mean absolute deviation from the median) may highlight areas where definitions are not clear or ratings are inconsistent, where panelist perceptions of the "average" patient may differ, or where various specialty groups or individual panelists may have differences of clinical opinion. In cases of obvious disagreement or outlier scores, the indication was highlighted in a summary table and identification of the outlier raters brought to the attention of the moderator. This information was used by the moderator to guide the panel's discussion.

Relationships With Industry

The College and its partnering organizations rigorously avoid any actual, perceived, or potential conflicts of interest that might arise as a result of an outside relationship or personal interest of a member of the Technical Panel. Specifically, all panelists are asked to provide disclosure statements of all relationships that might be perceived as real or potential conflicts of interest. These statements were reviewed by the Appropriateness Criteria Working Group, discussed with all members of the Technical Panel at the face-to-face meeting, and updated and reviewed as necessary. A table of disclosures by each Technical Panel and Oversight Working Group member can be found in Appendix D.

Literature Review

The Technical Panel members were asked to refer to the literature summary, evidence tables, and reference list provided for each modality when completing their ratings (online Appendix C and D at www.acc.org). A paper recently published on clinical indications for CMR (10) also was provided. Lastly, they were given the previously published materials pertaining to the appropriateness criteria work (2,8).

APPENDIX B: DEFINITIONS AND PROCESSES FOR DETERMINING LIKELIHOOD OF DISEASE AND RISK

Determining Pre-Test Probability of CAD

Chest Pain Syndrome: Any constellation of symptoms that the physician feels may represent a complaint consistent with obstructive CAD. Examples of such symptoms include, but are not exclusive to: chest pain, chest tightness, burning, dyspnea, shoulder pain, and jaw pain.

Pre-Test Probability of CAD: Once the physician determines the presence of symptoms that may represent obstructive CAD (chest pain syndrome present), then the pre-test probability of CAD should be determined.

Although there are several methods for determining pre-test probability of CAD (3,4), the method assumed for this report is a modification of a literature review (5) recommended by the American College of Cardiology/American Heart Association (ACC/AHA) 2002 Guideline Update for Exercise Testing (11) and ACC/AHA 2002 Guideline Update for Management of Patients with Chronic Stable Angina (12). The reader should refer to the definitions of angina and Table B1.

Angina: As defined by the ACC/AHA 2002 Guideline Update on Exercise Testing (11):

- *Typical* Angina (Definite): **1)** Substernal chest pain or discomfort that is **2)** provoked by exertion or emotional stress and **3)** relieved by rest and/or nitroglycerin (6).
- *Atypical* Angina (Probable): Chest pain or discomfort that **lacks one** of the characteristics of definite or typical angina (6).
- **Non-Anginal Chest Pain:** Chest pain or discomfort that **meets one or none** of the typical angina characteristics.

Determining Pre-Test Risk
Assessment for Risk Stratification

Risk Assessment The rating sheets on risk assessment include indications in patients with suspected CAD. This assessment is particularly valuable in the setting of asymptomatic individuals.

It is assumed that clinicians will use imaging studies in addition to standard methods of risk assessment as presented in the ACC/AHA Scientific Statement: Assessment of Cardiovascular Risk by Use of Multiple-Risk-Factor Assessment Equations (7), see Tables B2 and B3. Numerous discussions of the Framingham Risk Score calculation can be found online

Table B1. Pre-Test Probability of CAD by Age, Gender, and Symptoms*

Age (yrs)	Gender	Typical/Definite Angina Pectoris	Atypical/Probable Angina Pectoris	Nonanginal Chest Pain	Asymptomatic
30–39	Men	Intermediate	Intermediate	Low	Very low
	Women	Intermediate	Very low	Very low	Very low
40–49	Men	High	Intermediate	Intermediate	Low
	Women	Intermediate	Low	Very low	Very low
50–59	Men	High	Intermediate	Intermediate	Low
	Women	Intermediate	Intermediate	Low	Very low
60–69	Men	High	Intermediate	Intermediate	Low
	Women	High	Intermediate	Intermediate	Low

High: Greater than 90% pre-test probability; Intermediate: Between 10% and 90% pre-test probability; Low: Between 5% and 10% pre-test probability; Very Low: Less than 5% pre-test probability. *No data exist for patients less than 30 years or greater than 69 years, but it can be assumed that prevalence of CAD increases with age. In a few cases, patients with ages at the extremes of the decades listed may have probabilities slightly outside the high or low range.
    Reproduced with permission from ACC/AHA 2002 Guideline Update for Exercise Testing (11).

including at the National Heart, Lung, and Blood Institute Web site: http://www.nhlbi.nih.gov/about/framingham/riskabs.htm).

Coronary Heart Disease (CHD) Risk

- **CHD Risk—Low**
  Defined by the age-specific risk level that is below average. In general, low risk will correlate with a 10-year absolute CHD risk less than 10%.

- **CHD Risk—Moderate**
  Defined by the age-specific risk level that is average or above average. In general, moderate risk will correlate with a 10-year absolute CHD risk between 10% and 20%.

- **CHD Risk—High**
  Defined as the presence of diabetes mellitus or the 10-year absolute CHD risk of greater than 20%.

Evaluating Perioperative Risk for Non-Cardiac Surgery

Method for Determining Perioperative Risk Perioperative risk was determined for this report using a "Stepwise Approach to Preoperative Cardiac Assessment," found in the ACC/AHA 2002 Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery (13). Based on that algorithm, once it is determined that the patient does not require urgent surgery, and that there has not been revascularization within the last 5 years, the clinician should determine the patient's perioperative risk predictors (see definitions in the following text). If major

Table B2. Men: 10-Year CHD Risk According to Framingham Risk Score

Age (Low-risk level)*	30–34 (2%)	35–39 (3%)	40–44 (3%)	45–49 (4%)	50–54 (5%)	55–59 (7%)	60–64 (8%)	65–69 (10%)	70–74 (13%)	Absolute Risk Total CHD‡	Absolute Risk§ Hard CHD§
Points†											
0	1.0									2%	2%
1	1.5	1.0	1.0							3%	2%
2	2.0	1.3	1.3	1.0						4%	3%
3	2.5	1.7	1.7	1.3	1.0					5%	4%
4	3.5	2.3	2.3	1.8	1.4	1.0				7%	5%
5	4.0	2.6	2.6	2.0	1.6	1.1	1.0			8%	6%
6	5.0	3.3	3.3	2.5	2.0	1.4	1.3	1.0		10%	7%
7	6.5	4.3	4.3	3.3	2.6	1.9	1.6	1.3	1.0	13%	9%
8	8.0	5.3	5.3	4.0	3.2	2.3	2.0	1.6	1.2	16%	13%
9	10.0	6.7	6.7	5.0	4.0	2.9	2.5	2.0	1.5	20%	16%
10	12.5	8.3	8.3	6.3	5.0	3.6	3.1	2.5	1.9	25%	20%
11	15.5	10.3	10.3	7.8	6.1	4.4	3.9	3.1	2.3	31%	25%
12	18.5	12.3	12.3	9.3	7.4	5.2	4.6	3.7	2.8	37%	30%
13	22.5	15.0	15.0	11.3	9.0	6.4	5.6	4.5	3.5	45%	35%
>14	26.5	>17.7	>17.7	>13.3	>10.6	>7.6	>6.6	>5.3	>4.1	>53%	>45%

*Low-risk level is defined in the Framingham Report as the risk of coronary heart disease (CHD) at any age for a non-smoker, non-diabetic, with blood pressure less than 120/80 mmHg, total cholesterol of 160–199 mg/dL, LDL-C 100 to 129 mg/dL, and HDL-C greater than or equal to 45 mg/dL in men and greater than or equal to 55 mg/dL in women. †Points = number of points estimated from ACC/AHA Scientific Statement: Assessment of Cardiovascular Risk by Use of Multiple-Risk-Factor Assessment Equations, Table 4 (7). ‡Total Coronary Heart Disease (Total CHD) includes angina pectoris, recognized and unrecognized myocardial infarction, unstable angina, and CHD deaths. §Hard CHD includes all of the total CHD events except for angina pectoris. Reprinted with permission from Grundy SM, Pasternak R, Greenland P, et al. ACC/AHA scientific statement: assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. J Am Coll Cardiol 1999;34: 1348–59 (7).

Table B3. Women: 10-Year CHD Risk According to Framingham Risk Score

Age (Low-risk level)*	40–44 (2%)	45–49 (3%)	50–54 (5%)	55–59 (7%)	60–64 (8%)	65–69 (8%)	70–74 (8%)	Absolute Risk Total CHD‡	Absolute Risk Hard CHD§
Points†									
0	1.0							2%	1%
1	1.0							2%	1%
2	1.5	1.0						3%	2%
3	1.5	1.0						3%	2%
4	2.0	1.3						4%	2%
5	2.0	1.3						4%	2%
6	2.5	1.7	1.0					5%	2%
7	3.0	2.0	1.2					6%	3%
8	3.5	2.3	1.4	1.0				7%	3%
9	4.0	2.7	1.6	1.1	1.0	1.0	1.0	8%	3%
10	5.0	3.3	2.0	1.4	1.3	1.3	1.3	10%	4%
11	5.5	3.7	2.2	1.6	1.4	1.4	1.4	11%	7%
12	6.5	4.3	2.6	1.9	1.6	1.6	1.6	13%	8%
13	7.5	5.0	3.0	2.1	1.9	1.9	1.9	15%	11%
14	9.0	6.0	3.6	2.6	2.3	2.3	2.3	18%	13%
15	10.0	6.7	4.0	2.9	2.5	2.5	2.5	20%	15%
16	12.0	8.0	4.8	3.4	3.0	3.0	3.0	24%	18%
≥17	>13.5	>9.0	>5.4	>3.9	5.4	5.4	5.4	>27%	>20%

*Low-risk level is defined in the Framingham Report as the risk of coronary heart disease (CHD) at any age for a non-smoker, non-diabetic, with blood pressure less than 120/80 mmHg, total cholesterol of 160−199 mg/dL, LDL-C 100 to 129 mg/dL, and HDL-C greater than or equal to 45 mg/dL in men and greater than or equal to 55 mg/dL in women. †Points = number of points estimated from ACC/AHA Scientific Statement: Assessment of Cardiovascular Risk by Use of Multiple-Risk-Factor Assessment Equations, Table 4 (7). ‡Total Coronary Heart Disease (Total CHD) includes angina pectoris, recognized and unrecognized myocardial infarction, unstable angina, and CHD deaths. §Hard CHD includes all of the total CHD events except for angina pectoris. Reprinted with permission from Grundy SM, Pasternak R, Greenland P, et al. ACC/AHA scientific statement: assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. J Am Coll Cardiol 1999;34: 1348−59 (7).

risk predictors are present, coronary angiography and the postponement or cancellation of non-cardiac surgery should be considered. Once perioperative risk predictors are assessed based on the algorithm, then the surgical risk and patient's functional status should be used to establish the need for non-invasive testing.

Perioperative Risk Predictors*

- **Major risk predictors**
  Unstable coronary syndromes, decompensated heart failure (HF), significant arrhythmias, and severe valve disease.
- **Intermediate risk predictors**
  Mild angina, prior myocardial infarction (MI), compensated or prior HF, diabetes, or renal insufficiency.
- **Minor risk predictors**
  Advanced age, abnormal electrocardiogram (ECG), rhythm other than sinus, low functional capacity, history of cerebrovascular accident, and uncontrolled hypertension.

Surgical Risk Categories*

- **High-Risk Surgery—cardiac death or MI greater than 5%**
  Emergent major operations (particularly in the elderly), aortic and peripheral vascular surgery, prolonged surgical procedures associated with large fluid shifts and/or blood loss.

- **Intermediate-Risk Surgery—cardiac death or MI = 1% to 5%**
  Carotid endarterectomy, head and neck surgery, surgery of the chest or abdomen, orthopedic surgery, prostate surgery.
- **Low-Risk Surgery—cardiac death or MI less than 1%**
  Endoscopic procedures, superficial procedures, cataract surgery, breast surgery.

As defined by the ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation of Non-Cardiac Surgery (13).

ECG—Uninterpretable

Refers to ECGs with resting ST-segment depression (greater than or equal to 0.10 mV), complete left bundle-branch block, pre-excitation (Wolf-Parkinson-White syndrome), or paced rhythm.

APPENDIX C: ACCF APPROPRIATENESS CRITERIA WORKING GROUP AND TECHNICAL PANEL

CCT/CMR Writing Group

Robert C. Hendel, MD, FACC, Moderator of the Technical Panel, Midwest Heart Specialists, Fox River Grove, IL.

Manesh R. Patel, MD, Assistant Professor of Medicine, Division of Cardiology, Duke University Medical Center, Durham, NC.

Christopher M. Kramer, MD, FACC, Professor of Radiology and Medicine, Director, Cardiac MRI, University of Virginia Health System, Charlottesville, VA.

Michael Poon, MD, FACC, President-Elect, SCCT, Associate Professor of Medicine (Cardiology), Mount Sinai School of Medicine, New York, NY; Chief of Cardiology, Cabrini Medical Center, New York, NY.

Technical Panel

James C. Carr, MB, BCh, BAO, Assistant Professor, Departments of Radiology and Cardiology, Northwestern University Medical School, Chicago, IL.

Nancy A. Gerstad, MD, Medical Director, Medical & Credentialing Policy, WellPoint, Inc., Chicago, IL.

Linda D. Gillam, MD, FACC, Professor of Medicine (Clinical), University of Connecticut School of Medicine, Hartford, CT; Director, Echocardiography Lab, Hartford Hospital, Hartford, CT.

John McB. Hodgson, MD, FSCAI, FACC, Professor of Medicine and Chief of Academic Cardiology, Department of Internal Medicine, St. Joseph's Hospital & Medical Center, Phoenix, AZ.

Raymond J. Kim, MD, FACC, Associate Professor of Medicine and Co-Director, Duke Cardiovascular Magnetic Resonance Center (MRI), Duke University Medical Center, Durham, NC.

Christopher M. Kramer, MD, FACC, Professor of Radiology and Medicine, Director, Cardiac MRI, University of Virginia Health System, Charlottesville, VA.

John R. Lesser, MD, FACC, Director of Cardiac CT and MRI, Minneapolis Heart Institute, Minneapolis, MN.

Edward T. Martin, MD, FACC, FACP, Director, Cardiovascular MRI, Oklahoma Heart Institute, Tulsa, OK; Clinical Associate Professor of Medicine, University of Oklahoma, Tulsa, OK.

Joseph V. Messer, MD, MACC, FSCAI, Professor of Medicine, Rush University Medical Center, Chicago, IL; Associates in Cardiology, Ltd., Chicago, IL.

Rita F. Redberg, MD, MSc, FACC, Professor of Medicine, Division of Cardiology, UC San Francisco School of Medicine, San Francisco, CA.

Geoffrey D. Rubin, MD, FSCBTMR, Professor of Radiology, Chief of Cardiovascular Imaging, Associate Dean for Clinical Affairs, Stanford University School of Medicine, Stanford, CA.

John S. Rumsfeld, MD, PhD, FACC, Staff Cardiologist, Denver VAMC, Denver, CO; Associate Professor of Medicine, University of Colorado Health Sciences Center, Denver, CO; Chief Science Officer, ACC-NCDR, Bethesda, MD.

Allen J. Taylor, MD, FACC, Professor of Medicine, USUHS, Chief, United States Army Cardiology Service, Walter Reed Army Medical Center, Washington, DC.

Wm. Guy Weigold, MD, Director, Cardiac CT, Washington Hospital Center Division of Cardiovascular Disease, Washington, DC.

Pamela K. Woodard, MD, Associate Professor, Cardiovascular Imaging Laboratory, Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, MO.

ACCF Appropriateness Criteria Working Group

Ralph Brindis, MD, FACC, Chair, Regional Senior Advisor for Cardiovascular Diseases, Oakland Kaiser Medical Center, Oakland, California; Clinical Professor of Medicine, University of California at San Francisco, San Francisco, CA.

Pamela Douglas, MD, MACC, Immediate Past President, ACC; Ursula Geller Professor of Research and Chief, Cardiovascular Disease, Duke University Medical Center, Durham, NC.

Robert Hendel, MD, FACC, Moderator of the Technical Panel, Midwest Heart Specialists, Fox River Grove, IL.

Eric Peterson, MD, FACC, Associate Professor of Medicine and Director, Cardiovascular Outcomes, Duke University Medical Center, Durham, NC.

Michael J. Wolk, MD, MACC, Past President, ACC; Clinical Professor of Medicine, Weill-Cornell Medical School, New York, NY.

Joseph M. Allen, MA, Director, TRIP (Translating Research into Practice), American College of Cardiology, Bethesda, MD.

Manesh R. Patel, MD, Assistant Professor of Medicine, Division of Cardiology, Duke University Medical Center, Durham, NC.

APPENDIX D: RELATIONSHIPS WITH INDUSTRY

Table D1. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR Appropriateness Criteria Writing Group, Technical Panel, Working Group, and Indication Reviewers (In Alphabetical Order)

Committee Member	Research Grant	Speakers Bureau/Honoraries/ Expert Witness	Stock Ownership	Board of Directors	Consultant/Advisory Board/ Steering Committee
CCT/CMR Appropriateness Criteria Writing Group					
Dr. Robert C. Hendel	• Astellas Healthcare • GE Healthcare • Cornatus Genetics	• Bristol-Myers Squibb			• GE Healthcare • CV Therapeutics
Dr. Chirstopher Kramer	• Siemens Medical Solutions • Novartis Healthcare • Astellas Healthcare	• GE Healthcare			• GE Healthcare
Dr. Manesh R. Patel	None	None	None	None	None
Dr. Michael Poon	• Siemens Medical Solutions	None	None	None	• Siemens Medical Solutions
CCT/CMR Appropriateness Criteria Technical Panel					
Dr. James Carr	None	None	None	None	None
Dr. Nancy Gerstand			• WellPoint, Inc.		
Dr. Linda Gillam	• Acusphere • Philips • Bristol-Myers Squibb	• Bristol-Myers Squibb • Medtronic	None	None	None
Dr. John Hodgson	• GE Healthcare	• GE Healthcare			
Dr. Raymond Kim	• Siemens Medical Solutions				• Mallinckrodt
Dr. Chirstopher Kramer	• Siemens Medical Solutions • Novartis Healthcare • Astellas Healthcare	• GE Healthcare			• GE Healthcare
Dr. John Lesser	None	• Siemens Medical Systems	None	None	• Vital Images, Inc.
Dr. Edward Martin	• Guidant Corporation	• GE Healthcare			• Guidant Corporation • GE Healthcare
Dr. Joseph Messer	None	None	None	None	None
Dr. Rita Redberg	None	None	None	None	• Medicare Carrier Advisory Board
Dr. Geoffrey Rubin	• Siemens Medical Solutions • GE Healthcare • Bracco Diagnostics	None	None	None	• Biosense-Webster Inc. • Boston Scientific • Bracco Diagnostics • GE Healthcare • MED Institute, Inc. • Siemens Medical Solutions • United Healthcare • CV Therapeutics
Dr. John S. Rumsfeld					
Dr. Allen Taylor	None	None	None	None	None
Dr. Wm. Guy Weigold		• Phillips Medical Systems			
Dr. Pamela Woodard	• GE Healthcare • Siemens Medical Systems	• GE Healthcare	None	None	• TycoHealthcare/Mallinckrodt • GE Healthcare
CCT/CMR Appropriateness Criteria Working Group					
Joseph M. Allen	None	None	None	None	None
Dr. Ralph G. Brindis	None	None	None	None	None
Dr. Robert C. Hendel	• Astellas Healthcare • GE Healthcare • Cornatus Genetics	• Bristol-Myers Squibb			• GE Healthcare
Dr. Pamela S. Douglas					• GE Healthcare
Dr. Manesh R. Patel	None	None	None	None	None
Dr. Eric D. Peterson	• Millennium Pharmaceuticals • Schering Plough • BMS/Sanofi				
Dr. Michael J. Wolk	None	None	None	None	None
CCT/CMR Appropriateness Criteria Indication Reviewers					
Dr. Elliott Antman	None	None	None	None	None
Dr. Ronald Peshock					
Dr. Gregory Thomas	• CV Therapeutics • BMS Medical Imaging	• Astellas Healthcare	• CardioCura		
Dr. Samuel Wann			• The Wisconsin Heart Hospital Inc. • CardioCura		

REFERENCES

1. Medicare Payment Advisory Commission (MedPAC). Report to the Congress: growth in the volume of physician services. December 2004. Available at http://www.medpac.gov/publications/congressional_reports/December04_PhysVolume.pdf.

2. Patel MR, Spertus JA, Brindis RG, et al. ACCF proposed method for evaluating the appropriateness of cardiovascular imaging. J Am Coll Cardiol 2005;46:1606–13.

3. Morise AP, Haddad WJ, Beckner D. Development and validation of a clinical score to estimate the probability of coronary artery disease in men and women presenting with suspected coronary disease. Am J Med 1997;102:350–6.

4. Pryor DB, Harrell FE Jr., Lee KL, et al. Estimating the likelihood of significant coronary artery disease. Am J Med 1983;75:771–80.

5. Diamond GA, Forrester JS. Analysis of probability as an aid in the clinical diagnosis of coronary-artery disease. N Engl J Med 1979;300:1350–8.

6. Diamond GA. A clinically relevant classification of chest discomfort. J Am Coll Cardiol 1983;1:574–5.

7. Grundy SM, Pasternak R, Greenland P, et al. ACC/AHA scientific statement: assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. J Am Coll Cardiol 1999;34:1348–59.

8. Brindis RG, Douglas PS, Hendel RC, et al. ACCF/ASNC appropriateness criteria for single-photon emission computed tomography myocardial perfusion imaging (SPECT MPI): a report of the American College of Cardiology Foundation Strategic Directions Committee Appropriateness Criteria Working Group and the American Society of Nuclear Cardiology. J Am Coll Cardiol 2005;46:1587–605.

9. Fitch K, Bernstein SJ, Aquilar MD, et al. The RAND/UCLA Appropriateness Method User's Manual. Santa Monica, CA: The RAND Corporation, 2001.

10. Pennell DJ, Sechtem UP, Higgins CB, et al. Clinical indications for cardiovascular magnetic resonance (CMR): Consensus Panel report. Eur Heart J 2004;25:1940–65.

11. Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA 2002 guideline update for exercise testing: summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). J Am Coll Cardiol 2002;40:1531–40.

12. Gibbons RJ, Abrams J, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina—summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Chronic Stable Angina). Circulation 2003;107:149–58.

13. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). Circulation 2002;105:1257–67.

American College of Radiology
ACR Appropriateness Criteria®

<u>**Clinical Condition:**</u>　　　　　　**Chronic Chest Pain—Suspected Cardiac Origin**

Radiologic Procedure	Rating	Comments	RRL*
X-ray chest	9	Helpful to exclude a noncardiac cause for chest pain.	Min
NUC myocardial perfusion scan stress	9	Effective for evaluating myocardial perfusion.	High
US echocardiography transthoracic stress	7	If coronary arteries are normal, and concern involves structural heart disease.	None
US echocardiography transthoracic resting	7	Can be used to demonstrate LV regional dysfunction due to ischemia and excellent for regional wall motion abnormalities.	None
CTA heart	7	Can be used to noninvasively visualize the coronary arteries. Excellent to assess coronary disease with multidetector scanners. May be useful in low-risk population but has not been studied in this population.	High
INV angiography coronary	7	The definitive test for establishing the diagnosis and directing treatment if clinical suspicion of CAD is high, or if there is an abnormal noninvasive imaging test.	IP
MRI heart resting (function and delayed enhancement)	6	Can be used to noninvasively evaluate LV regional dysfunction and areas of prior MI.	None
PET heart stress	6	Especially for patients who may not be optimal for conventional nuclear imaging (ie, obese patients).	High
CT heart calcium scoring	5	Negative test highly accurate in excluding CAD. Indicated in appropriate population where a pretest probability of zero calcium score is high.	Low
MRI heart stress (wall motion and perfusion)	5	Stress studies should only be performed at sites with appropriate expertise and equipment, due to safety concerns.	None
CT chest with contrast	4	Could be used to establish a noncardiac cause for chest pain. Possible utility in aortic dissection and potential pulmonary abnormalities.	Med
US gall bladder	3	Only if complete cardiac workup is negative. Can be used to exclude a noncardiac cause for chest pain.	None
MRA coronary arteries	2	May be indicated in patients unable to receive iodinated contrast, at sites with extensive expertise.	None
<u>**Rating Scale:**</u> **1=Least appropriate, 9=Most appropriate**			***Relative Radiation Level**

CHRONIC CHEST PAIN—SUSPECTED CARDIAC ORIGIN

Expert Panel on Cardiac Imaging: David S. Gerson, MD[1]; Frank J. Rybicki, MD, PhD[2]; E. Kent Yucel, MD[3]; Arfa Khan, MD[4]; Linda B. Haramati, MD[5]; Vincent B. Ho, MD[6]; Anna Rozenshtein, MD[7]; U. Joseph Schoepf, MD[8]; William Stanford, MD[9]; Pamela K. Woodard, MD[10]; Michael Jaff, MD.[11]

Summary of Literature Review

Chronic chest pain of suspected cardiac origin is usually a consequence of myocardial ischemia. This is usually caused by fixed stenosis (atherosclerotic plaques), coronary spasm, microvascular disease, or a combination of the three. Chest pain of cardiac ischemic origin represents an imbalance between myocardial oxygen demand and coronary blood flow, and chronic pain can occur in patients with normal coronary arterial caliber for whom the primary cardiac pathology is extracoronary, (eg, aortic stenosis, hypertrophic cardiomyopathy). Nonischemic cardiac pain may be caused by pericarditis. While the syndrome of exertional angina pectoris is nearly always diagnostic for chronic coronary arterial disease, other extracardiac etiologies should be considered, especially for nonexertional or atypical chest pain, such as esophageal reflux and spasm, biliary disease, costosternal syndrome, and cervical radiculitis.

In patients with chronic chest pain, imaging has a major role in determining and documenting the presence, extent, and severity of myocardial ischemia and/or the presence, site, and severity of obstructive coronary lesions. Imaging findings are an important factor in determining the course of management of patients with suspected chronic myocardial ischemia in order to determine those patients best suited for medical therapy, angioplasty/stenting, or surgery. Imaging is also necessary to evaluate left ventricular function because ejection fraction and end systolic volume are important in predicting the long-term prognosis and likely benefit from various therapeutic options. Imaging studies are also required to demonstrate abnormalities such as aortic stenosis and hypertrophic cardiomyopathy, which can produce angina in the absence of coronary obstructive disease.

The historically established imaging studies that may be used in evaluating suspected chronic myocardial ischemia are chest radiography, radionuclide myocardial perfusion imaging and ventriculography with and without stress; and catheter-based coronary angiography, and left ventriculography. Stress echocardiography (echo) and computed tomography (CT), both electron beam and multidetector CT (MDCT), have made significant progress in the evaluation of ischemic heart disease. Positron emission tomography (PET) is also now available for this purpose. Cardiac magnetic resonance imaging (MRI), while making significant headway in the diagnosis of infarction, is less widely used for stress-induced ischemia. In those patients who do not present with signs classic for angina pectoris, or in those patients who do not respond as expected to standard management, the exclusion of noncardiac causes of chronic chest pain require the use of additional studies, including esophagography, upper gastrointestinal series, and biliary imaging with ultrasound (US).

Chest Radiography
The chest radiograph is an inexpensive test that can rapidly demonstrate many noncardiac causes of chronic chest pain, including a variety of diseases of the mediastinum, pleura, or lung. It may also provide qualitative information about left ventricular function as reflected in cardiac size and pulmonary venous status. However, radiography can neither establish nor exclude chronic ischemic heart disease. It is relatively insensitive for detecting coronary arterial calcification. Also, fluoroscopy cannot reliably detect coronary artery disease (CAD) [1].

Radionuclide Imaging
Stress myocardial perfusion imaging demonstrates relative myocardial perfusion defects, indicating the presence of myocardial ischemia. For this reason, it is considered an important first line study in the evaluation of patients with chronic chest pain. The territory of the perfusion defect identifies the likely culprit coronary artery and can sometimes distinguish between significant single-vessel and multi-vessel coronary arterial obstruction(s) [2-11]. The rest and redistribution perfusion scans demonstrate reversibility (ischemia) or irreversibility (infarction) of the perfusion defect. Technetium 99^m sestamibi has been shown to be more specific for ischemia when compared to thallium [10]. In a meta-analysis of 20 published studies including 488 patients studied with technetium 99m sestamibi, sensitivity and specificity were calculated to be 81% and 66% respectively with positive and negative predictive values of 71% and 77% respectively for detecting hibernating myocardium [3]. Limitations of stress

[1]Research Author, Mount Sinai Medical Center, Miami Beach, Fla; [2]Principal Author, Brigham and Women's Hospital, Boston, Mass; [3]Co-Chair, Boston VA Healthcare System, West Roxbury, Mass; [4]Co-Chair, Long Island Jewish Medical Center, New Hyde Park, NY; [5]Albert Einstein College of Medicine, Montefiore Medical Center, Bronx, NY; [6]Uniformed Services University, Bethesda, Md; [7]Columbia Presbyterian Medical Center, New York, NY; [8]Medical University of South Carolina, Charleston, SC; [9]University of Iowa Hospitals & Clinics, Iowa City, Iowa; [10]Mallinckrodt Institute of Radiology, Saint Louis, Mo; [11]Massachusetts General Hospital, Boston, Mass, American College of Cardiology.
Reprint requests to: Department of Quality & Safety, American College of Radiology, 1891 Preston White Drive, Reston, VA 20191-4397.

myocardial perfusion imaging are its relatively high cost, difficulties with interpretation (especially in women), and difficulties imaging obese patients.

Stress radionuclide ventriculography (RNV) consists of measurement of the ejection fraction and assessment of regional wall motion at rest and at the peak of stress. This technique can be used to identify patients with "balanced" 3-vessel disease, which can be missed in perfusion studies and for differentiating attenuation artifacts from infarcts [8], although CT is becoming increasingly useful for these indications. Wall motion abnormalities and ejection fraction have been shown to be independent predictors of the extent of CAD [12,13]. However, stress myocardial perfusion scintigraphy is generally the preferred method for identifying regional ischemia, and stress RNV is not usually necessary if an adequate perfusion study has been obtained. In the presence of a positive perfusion study, the stress RNV is superfluous.

In patients with typical angina (high pretest likelihood of disease), stress perfusion or RNV studies are useful for estimating the extent (single-vessel versus multi-vessel disease) and severity of coronary stenosis, which has relevance for prognosis, choice among therapeutic options, and advisability of performing coronary arteriography. In patients with atypical angina, stress perfusion imaging is useful for determining whether myocardial ischemia is the etiology.

Positron Emission Tomography
Myocardial PET imaging with ^{82}Rb, fluro-deoxy glucose (FDG), and ^{13}N is now reimbursable by the Center for Medicaid and Medicare Services, underscoring recent technology advances. The coincidence detection method used in PET imaging allows for reliable correction of the problems associated with nonuniform attenuation of photons in the chest and for differences between men and women [14]. In a meta-analysis of 8 studies with 791 patients evaluated for CAD by PET, a combined sensitivity and specificity were determined to be 93% and 92%, respectively [15]. In the same article, three studies comparing Tl-201 single-proton-emission computed tomography (SPECT) and Rb-82 or NH3 PET were analyzed, and the overall accuracy of PET was 91%, compared to 81% for Tl-201 SPECT. It also may be the case that the sensitivity of PET can be increased when it is performed with CT [16].

Echocardiography
Stress 2-dimensional (2-D) echo is increasingly used for patients with suspected regional wall motion abnormalities produced by regional ischemia, in part because of the ubiquity of 2-D echocardiography. Technical limitations associated with exercise stress can be overcome by using pharmacological (dobutamine)

stress. A recent meta-analysis of 44 studies indicated that stress echocardiography has a similar sensitivity to stress SPECT (85% and 87%, respectively) with a higher specificity (77% vs 64%) [17]. This technique is limited by the fact that it sometimes yields nondiagnostic results and that suboptimal definition of some regions of the left ventricle can lead to subjective interpretation Resting echocardiography can be useful if pericardial effusion or valvular or chamber abnormalities are suspected.

Transesophageal echocardiography is generally not indicated for evaluating chronic angina. The expense of this study does not justify its use in this setting. Although it is sometimes used for assessing aortic pathology (eg, dissection, aneurysm, and penetrating ulcer) in patients with chronic chest pain, CT and MRI are less invasive and simpler to perform.

Computed Tomography
Electron beam (ultrafast) CT (EBCT) can detect the presence and severity of calcification, a sign of coronary atherosclerosis [18-22]. EBCT is very sensitive for significant atherosclerotic disease, but many coronary lesions are eccentric and do not decrease the luminal diameter; therefore the presence of calcification is not specific for stenosis. A meta-analysis of 9 studies and 1662 subjects calculated a pooled sensitivity and specificity of 92.3% and 51.2%, respectively, for ≥50% stenosis [23]. The absence of coronary arterial calcification (CAC) in patients with chronic angina makes significant coronary obstructive disease unlikely (less than 1% [24]) but does not exclude it. Similarly, the presence of 3-vessel disease and/or extensive calcification (eg, a high calcium score) confers a high likelihood of coronary obstructive disease, but it does not confirm the diagnosis.

Because of the limitations described above, at present no CT vendor manufactures commercial EBCT units, and support for units currently in use is becoming scarce. Research focused on the relative equivalence of EBCT and MDCT with submillimeter spatial resolution and high temporal resolution has demonstrated agreement between coronary calcium scores [25-27], despite early reports of poor correlation with older CT technology [28].

Calcium scoring (noncontrast ECG-gated MDCT) is controversial. On one hand, the test is relatively inexpensive, and absence of coronary calcification is useful evidence against myocardial ischemia [18]. In a large study of 10,377 subjects it has been shown that coronary calcium score provides independent incremental information in addition to traditional risk factors in the prediction of all cause mortality [29]. On the other hand, patients who present with chronic chest pain of suspected cardiac origin are typically older, with a significant

proportion over 60 years old. Because coronary calcium is so prevalent in this population, a "positive" score, even in the upper quartiles, cannot be used as strong evidence of myocardial ischemia.

There is also significantly greater use of coronary CT angiography (CTA) (specifically, contrast enhanced ECG-gated MDCT) to evaluate for CAD. Over the past 5 years, CT vendors have increased the number of detectors (from 4 to 64 and, with experimental human results, up to 256), improved the spatial resolution to submillimeter, and decreased the temporal resolution to approximately 0.1 second. While these improvements have not equaled catheter-based coronary angiography, recent studies have shown a high sensitivity of MDCT for treatable stenoses of the coronary arteries [30-32]. Using present technology, the major strength of coronary CTA is its high negative predictive value (in comparison with the positive predictive value), and thus it suffers the same limitations as calcium scoring. It should be noted that the utility of coronary CTA becomes limited in more elderly patients (ie, those with a high burden of calcium) who have a pretest probability of CAD. Namely, the population of patients who present with chronic chest pain typically have CAD, and thus excluding a hemodynamically significant stenosis may be challenging. In patients who are younger and who have a lower pretest probability of CAD, coronary CTA can exclude a coronary etiology of chronic chest pain. Moreover, CT can exclude 3-vessel disease potentially missed by nuclear imaging (eg, so-called "balanced" ischemia) in patients with a high clinical suspicion of CAD.

There are other indications for which CT is the imaging test of choice, specifically aortic disease (aortic dissection, penetrating aortic ulcer, etc) and pulmonary embolism. CT has the advantage that it detects, with high specificity, a large number of extracardiac diagnoses.

Magnetic Resonance Imaging
Use of MRI for evaluating cardiac anatomy, valvular disease, certain cardiomyopathies, viability, and cardiac function continues to evolve. Protocols for measuring myocardial perfusion and angiography of the pulmonary and systemic vessels have matured significantly in the past few years. Magnetic resonance angiography (MRA) of the coronary arteries is still problematic due to their small size and incessant motion tied to the respiratory and cardiac cycles. At this time, MRA should be limited to sites with extensive experience and appropriate hardware and software to exclude disease in the proximal coronary arteries. At present, only CTA can noninvasively visualize coronary arteries on a routine basis.

MRI myocardial perfusion can be used to assess for significant CAD. First pass perfusion, rest perfusion, and stress perfusion protocols have been developed and validated; these are equivalent to and in some cases reported superior to SPECT [33-35]. High-dose dobutamine stress cardiac MRI has also been used in patients with poor acoustic windows which would have otherwise limited the utility of stress echocardiography [36] and has been shown to have a higher diagnostic accuracy than dobutamine stress echocardiography [37]. However, MRI is difficult to use, as most patients with pacemakers or implanted cardiac defibrillators are prohibited from obtaining a study and some other patients are too claustrophobic to tolerate an examination that routinely requires up to 60 minutes. While MRI is significantly more expensive than other studies that provide similar information, it can be used as a problem-solving tool for patients who can benefit from the high image contrast inherent in the myocardium and blood interface.

Invasive Techniques
Catheter-based angiography remains the coronary imaging modality with the highest spatial and temporal resolution. Thus, despite the fact that only projection images are obtained (as opposed to 3D volumes in CT), catheter-based angiography is considered by most to be the "gold-standard" for depicting the anatomy and the severity of obstructive CAD and other coronary arterial abnormalities (such as spasm) [38]. Moreover, it is needed to guide transluminal interventions. There is no general agreement regarding its use in patients with angina, but it is clearly not indicated in all patients who present with chronic chest pain. There is evidence that this test may be over utilized [39].

There remains agreement that catheter-based angiography is indicated in patients in whom angina is not adequately managed by vigorous medical therapy and in those in whom left main stenosis or severe multivessel disease is suggested by results of nuclear perfusion imaging. Left ventricular catheterization and left ventriculography are generally indicated, but not always necessary, to define ventricular function in patients with angina. In many patients, left ventricular function can be evaluated adequately using noninvasive studies (echocardiography and RNV).

Other Studies
Neither ultrasound nor nuclear imaging of the biliary system is usually indicated in patients who present with typical angina. However, patients who fall under the category of "chronic chest pain" can have a variety of diagnoses, and intermittent biliary obstruction from a gallstone can mimic intermittent pain from CAD. With respect to the "chronic" patient, a similar argument can be made for gastroesophageal reflux, and a fluoroscopy-based esophagram with or without an upper GI study, or

endoscopic evaluation of the esophagus, can be obtained when symptoms are not classic for pain of a cardiac origin, or when the patient does not respond to standard therapy.

Summary

The defined approach to evaluation of the patient with chronic chest pain of probable cardiac origin is supported by a substantial body of literature. For patients with 1) a classic history and physical examination and 2) expected response to moderate medical therapy, no imaging study may be needed. Otherwise, stress nuclear imaging is used as a front-line modality to establish the diagnosis and assess the severity of myocardial ischemia. Based on the results of nuclear perfusion and/or clinical response to medical therapy, the next procedure is usually coronary angiography, with or without cardiac catheterization, and/or left ventriculography. Given the underlying prevalence of CAD in this patient population, the substitution of newer examinations (eg, CT and stress echocardiography) is promising but at present is not justified by current data; this outlook could change based on results of comparative studies and cost analysis.

References

1. Margolis JR, Chen JT, Kong Y, Peter RH, Behar VS, Kisslo JA. The diagnostic and prognostic significance of coronary artery calcification. A report of 800 cases. *Radiology* 1980; 137(3):609-616.
2. Basu S, Senior R, Dore C, Lahiri A. Value of thallium-201 imaging in detecting adverse cardiac events after myocardial infarction and thrombolysis: a follow up of 100 consecutive patients. *Bmj* 1996; 313(7061):844-848.
3. Bax JJ, Poldermans D, Elhendy A, Boersma E, Rahimtoola SH. Sensitivity, specificity, and predictive accuracies of various noninvasive techniques for detecting hibernating myocardium. *Curr Probl Cardiol* 2001; 26(2):147-186.
4. Beller GA. Diagnostic accuracy of thallium-201 myocardial perfusion imaging. *Circulation* 1991; 84(3 Suppl):I1-6.
5. Christian TF, Miller TD, Bailey KR, Gibbons RJ. Noninvasive identification of severe coronary artery disease using exercise tomographic thallium-201 imaging. *Am J Cardiol* 1992; 70(1):14-20.
6. Gibbons RJ. Rest and exercise radionuclide angiography for diagnosis in chronic ischemic heart disease. *Circulation* 1991; 84(3 Suppl):I93-99.
7. Giri S, Shaw LJ, Murthy DR, et al. Impact of diabetes on the risk stratification using stress single-photon emission computed tomography myocardial perfusion imaging in patients with symptoms suggestive of coronary artery disease. *Circulation* 2002; 105(1):32-40.
8. Meine TJ, Hanson MW, Borges-Neto S. The additive value of combined assessment of myocardial perfusion and ventricular function studies. *J Nucl Med* 2004; 45(10):1721-1724.
9. Soman P, Taillefer R, DePuey EG, Udelson JE, Lahiri A. Enhanced detection of reversible perfusion defects by Tc-99m sestamibi compared to Tc-99m tetrofosmin during vasodilator stress SPECT imaging in mild-to-moderate coronary artery disease. *J Am Coll Cardiol* 2001; 37(2):458-462.
10. Taillefer R, DePuey EG, Udelson JE, Beller GA, Latour Y, Reeves F. Comparative diagnostic accuracy of Tl-201 and Tc-99m sestamibi SPECT imaging (perfusion and ECG-gated SPECT) in detecting coronary artery disease in women. *J Am Coll Cardiol* 1997; 29(1):69-77.
11. Vanzetto G, Ormezzano O, Fagret D, Comet M, Denis B, Machecourt J. Long-term additive prognostic value of thallium-201 myocardial perfusion imaging over clinical and exercise stress test in low to intermediate risk patients: study in 1137 patients with 6-year follow-up. *Circulation* 1999; 100(14):1521-1527.
12. Borges-Neto S, Shaw LJ, Kesler KL, et al. Prediction of severe coronary artery disease by combined rest and exercise radionuclide angiocardiography and tomographic perfusion imaging with technetium 99m-labeled sestamibi: a comparison with clinical and electrocardiographic data. *J Nucl Cardiol* 1997; 4(3):189-194.
13. Palmas W, Friedman JD, Diamond GA, Silber H, Kiat H, Berman DS. Incremental value of simultaneous assessment of myocardial function and perfusion with technetium-99m sestamibi for prediction of extent of coronary artery disease. *J Am Coll Cardiol* 1995; 25(5):1024-1031.
14. Eisner R, Patterson R. Differences between women and men in the heterogenecity of myocardial perfusion images: SPECT, Tl-201, Tc-99m sestamibi, and PET Rb-82. *J Nucl Cardiol* 1997; 1(Part 2):S104.
15. Machac J. Cardiac positron emission tomography imaging. *Semin Nucl Med* 2005; 35(1):17-36.
16. Namdar M, Hany TF, Koepfli P, et al. Integrated PET/CT for the assessment of coronary artery disease: a feasibility study. *J Nucl Med* 2005; 46(6):930-935.
17. Fleischmann KE, Hunink MG, Kuntz KM, Douglas PS. Exercise echocardiography or exercise SPECT imaging? A meta-analysis of diagnostic test performance. *Jama* 1998; 280(10):913-920.
18. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M, Jr., Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol* 1990; 15(4):827-832.
19. Agatston AS, Janowitz WR, Kaplan G, Gasso J, Hildner F, Viamonte M, Jr. Ultrafast computed tomography-detected coronary calcium reflects the angiographic extent of coronary arterial atherosclerosis. *Am J Cardiol* 1994; 74(12):1272-1274.
20. Breen JF, Sheedy PF, 2nd, Schwartz RS, et al. Coronary artery calcification detected with ultrafast CT as an indication of coronary artery disease. *Radiology* 1992; 185(2):435-439.
21. Fallavollita JA, Brody AS, Bunnell IL, Kumar K, Canty JM, Jr. Fast computed tomography detection of coronary calcification in the diagnosis of coronary artery disease. Comparison with angiography in patients < 50 years old. *Circulation* 1994; 89(1):285-290.
22. Wong ND, Vo A, Abrahamson D, Tobis JM, Eisenberg H, Detrano RC. Detection of coronary artery calcium by ultrafast computed tomography and its relation to clinical evidence of coronary artery disease. *Am J Cardiol* 1994; 73(4):223-227.
23. Nallamothu BK, Saint S, Bielak LF, et al. Electron-beam computed tomography in the diagnosis of coronary artery disease: a meta-analysis. *Arch Intern Med* 2001; 161(6):833-838.
24. Haberl R, Becker A, Leber A, et al. Correlation of coronary calcification and angiographically documented stenoses in patients with suspected coronary artery disease: results of 1,764 patients. *J Am Coll Cardiol* 2001; 37(2):451-457.
25. Carr JJ, Crouse JR, 3rd, Goff DC, Jr., D'Agostino RB, Jr., Peterson NP, Burke GL. Evaluation of subsecond gated helical CT for quantification of coronary artery calcium and comparison with electron beam CT. *AJR Am J Roentgenol* 2000; 174(4):915-921.
26. Detrano RC, Anderson M, Nelson J, et al. Coronary calcium measurements: effect of CT scanner type and calcium measure on rescan reproducibility--MESA study. *Radiology* 2005; 236(2):477-484.
27. Stanford W, Thompson BH, Burns TL, Heery SD, Burr MC. Coronary artery calcium quantification at multi-detector row helical CT versus electron-beam CT. *Radiology* 2004; 230(2):397-402.
28. Becker CR, Jakobs TF, Aydemir S, et al. Helical and single-slice conventional CT versus electron beam CT for the quantification of

coronary artery calcification. *AJR Am J Roentgenol* 2000; 174(2):543-547.

29. Shaw LJ, Raggi P, Schisterman E, Berman DS, Callister TQ. Prognostic value of cardiac risk factors and coronary artery calcium screening for all-cause mortality. *Radiology* 2003; 228(3):826-833.

30. Fine JJ, Hopkins CB, Hall PA, Delphia RE, Attebery TW, Newton FC. Noninvasive coronary angiography: agreement of multi-slice spiral computed tomography and selective catheter angiography. *Int J Cardiovasc Imaging* 2004; 20(6):549-552.

31. Nieman K, Cademartiri F, Lemos PA, Raaijmakers R, Pattynama PM, de Feyter PJ. Reliable noninvasive coronary angiography with fast submillimeter multislice spiral computed tomography. *Circulation* 2002; 106(16):2051-2054.

32. Ropers D, Baum U, Pohle K, et al. Detection of coronary artery stenoses with thin-slice multi-detector row spiral computed tomography and multiplanar reconstruction. *Circulation* 2003; 107(5):664-666.

33. Ibrahim T, Nekolla SG, Schreiber K, et al. Assessment of coronary flow reserve: comparison between contrast-enhanced magnetic resonance imaging and positron emission tomography. *J Am Coll Cardiol* 2002; 39(5):864-870.

34. Keijer JT, van Rossum AC, van Eenige MJ, et al. Magnetic resonance imaging of regional myocardial perfusion in patients with single-vessel coronary artery disease: quantitative comparison with (201)Thallium-SPECT and coronary angiography. *J Magn Reson Imaging* 2000; 11(6):607-615.

35. Wilke NM, Jerosch-Herold M, Zenovich A, Stillman AE. Magnetic resonance first-pass myocardial perfusion imaging: clinical validation and future applications. *J Magn Reson Imaging* 1999; 10(5):676-685.

36. Hundley WG, Hamilton CA, Thomas MS, et al. Utility of fast cine magnetic resonance imaging and display for the detection of myocardial ischemia in patients not well suited for second harmonic stress echocardiography. *Circulation* 1999; 100(16):1697-1702.

37. Nagel E, Lehmkuhl HB, Bocksch W, et al. Noninvasive diagnosis of ischemia-induced wall motion abnormalities with the use of high-dose dobutamine stress MRI: comparison with dobutamine stress echocardiography. *Circulation* 1999; 99(6):763-770.

38. Alderman EL, Corley SD, Fisher LD, et al. Five-year angiographic follow-up of factors associated with progression of coronary artery disease in the Coronary Artery Surgery Study (CASS). CASS Participating Investigators and Staff. *J Am Coll Cardiol* 1993; 22(4):1141-1154.

39. Graboys TB, Biegelsen B, Lampert S, Blatt CM, Lown B. Results of a second-opinion trial among patients recommended for coronary angiography. *JAMA* 1992; 268(18):2537-2540.

Consensus Update on the Appropriate Usage of Cardiac Computed Tomographic Angiography

Moderators: [a,§]Michael Poon, MD and [b,*]Geoffrey D. Rubin, MD

Panelists: [c]Stephan Achenbach, MD, [d]Tim W. Attebery, MD, [e]Daniel S. Berman, MD, [f]Thomas J. Brady, MD, [g]Jill E. Jacobs, MD, [h]Harvey S. Hecht, MD, [i]João A.C. Lima, MD, [j]Wm. Guy Weigold, MD

Introduction and Purpose

The consensus statement outlined here was formulated following a roundtable meeting among clinical experts in the fields of radiology and cardiology held in Miami, Florida, in June 2007. This group was gathered under the auspices of two key specialty societies supporting the field of computed tomographic angiography (CTA): the Society of Cardiovascular Computed Tomography (SCCT) and the North American Society for Cardiac Imaging (NASCI).

The purpose of the roundtable meeting was to produce an updated consensus on CTA's utility and appropriateness in everyday clinical practice among radiologists and cardiologists. This work does not represent a clinical guideline, rather, it serves as a follow-up consensus statement to the CTA component of the ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 Appropriateness Criteria for Cardiac Computed Tomography and Cardiac Magnetic Resonance Imaging, and the 2006 AHA Scientific Statement on the Assessment of Coronary Artery Disease by Cardiac Computed Tomography.

Specifically, this consensus statement aims to propose a unified approach for clinicians to adopt in their everyday practice with regard to CTA and its role in diagnosing and evaluating coronary artery disease. It offers updated information on this new, rapidly advancing technology for practitioners who want to incorporate this noninvasive imaging modality into their daily practice. A number of ongoing controversial topics are examined in this document, and a consensus opinion is provided on each

of them in the hopes of guiding practitioners toward more appropriate utilization of this new imaging technology. Whether coronary artery calcium (CAC) scoring should be done prior to CTA, and whether CAC is helpful in determining risk of future cardiovascular events in asymptomatic patients are two such topics covered here, as is the importance of contrast agent selection in CTA. This statement also offers an opinion on the usefulness of CTA in evaluating patients in various clinical scenarios: asymptomatic patients, symptomatic patients with suspected or known coronary artery disease (CAD), patients presenting with acute coronary syndromes in the emergency room (ER), and symptomatic patients with CAD who have undergone previous coronary artery stenting or coronary artery bypass graft surgery (CABG).

A. The Role of Coronary Artery Calcium (CAC) Measurement in Asymptomatic Patients

Consensus

1. CAC is useful in the detection of subclinical atherosclerosis in all ethnic groups.
2. CAC is most useful for risk stratification of patients with an intermediate Framingham risk of future cardiovascular events (10–20% 10-year risk), in whom a high-risk CAC score may prompt an increase in aggressive medical therapy.
3. As set forth by the SHAPE Task Force, CAC screening is recommended for all asymptomatic men 45–75 years of age and women 55–75 years of age who do not have very-low-risk characteristics (absence of any traditional cardiovascular risk factors) or a documented history of cardiovascular disease.
4. In general, CAC cannot at this time be recommended for general screening in unselected individuals or on the basis of self-referral, as it is of limited clinical value in patients at low risk for cardiovascular events (rate < 1.0% per year), and also cannot be used to exclude high-risk patients from medical therapy, even if their CAC score is zero.
5. Routine monitoring of CAC progression through the use of serial CT scanning cannot be recommended at this time.

B. The Role of Coronary CTA in Asymptomatic Patients

Consensus

1. CTA offers more information than calcium scanning regarding coronary atherosclerosis.
2. We do not have any data on how to and to what level we should treat patients with noncalcified plaques shown on CTA. This is true in general, but is especially relevant for patients with a zero calcium score.
3. Equally, no data exist that would support *not* treating a patient at high clinical risk who has a normal CTA.
4. Although CTA may contribute to refined risk assessment in certain subsets of the population, there are currently no clinical data to support its use or upon which to base therapeutic recommendations. Therefore, it is currently not recommended to use CTA for screening purposes.
5. In a small subset of patients with a very strong family history of premature CAD, *e.g.*, early or sudden death due to heart disease and multiple risk factors, CTA may have a role in early detection and prevention of CAD. However, there are no data to support this.
6. CTA for plaque characterization appears to be feasible in selected patients, but impractical in the general population, due to the high degree of variation in CTA image quality, and because whatever criteria are developed based on high-quality scans cannot be extended to low-quality scans.
7. Plaque volume may be determined with CTA and may be proportional to the degree of coronary risk. It is hypothesized that plaque rupture is associated with a larger plaque volume. The degree of positive remodeling is also associated with an increased risk of having a cardiac event. Involvement of more proximal segments probably carries higher risk than distal segments.
8. It would be useful to develop a system for quantifying noncalcified plaque.

C. The Role of Coronary CTA in Symptomatic Patients with Suspected CAD

Consensus

1. CTA is useful in ruling out the presence of significant coronary stenosis.
2. According to the National Cardiovascular Data Registry (NCDR) CATH-PCI, nearly 37% of cardiac catheterizations are normal and were generated from false-positive stress/stress perfusion tests. CTA is useful in the evaluation of symptomatic patients with equivocal or discordant results on a stress perfusion or wall motion study.
3. Many practitioners feel that any degree of stenosis due to calcified or noncalcified plaque on CTA should be treated aggressively with a statin and aspirin, although there are no data to support this. The goal of statin therapy in this setting, generally, is to decrease low-density lipoprotein (LDL) cholesterol to < 70 mg/dl.

4. Data in symptomatic patients are not available to predict the likelihood of future adverse cardiovascular events based on the presence of calcified or noncalcified plaques on CTA.
5. When compared to conventional angiography, CTA offers additional information regarding the vessel wall that may affect treatment strategy. However, data are not yet available to support this.
6. CTA has high negative predictive value, and the result may provide clinical guidance for a longer period of time than other noninvasive imaging modalities.
7. Physicians may use the calcium score and noncalcified plaque information from CAC and CTA to decide if aggressive lipid-lowering treatment is warranted.
8. CTA has great potential for prognosis, but currently we do not have good outcome data in support of the use of this technology in routine disease management.
9. Until data are available regarding the natural history of coronary arterial atherosclerosis progression on CTA imaging, this modality cannot be recommended for assessment of the effectiveness of medical therapy.
9. Because of the prevalence of false-positive CTA results, practitioners should avoid scanning very low-risk patients.
10. There is a tendency to overcall stenosis severity with CTA in non-expert hands.
11. Stress radionuclide perfusion imaging in the evaluation of symptomatic patients with known CAD or with a high likelihood of CAD is likely to remain an initial test of choice. Data are currently available supporting the use of functional information from stress myocardial perfusion imaging (MPI) in guiding decisions regarding revascularization, and no such data exist at this time for CTA. Furthermore, these patients frequently have extensive coronary calcification, reducing the ability of CTA to rule out significant obstruction with high confidence. Despite this, the Achilles heel of nuclear stress testing is the patient with three-vessel or left main disease, in which CTA may be more sensitive.
12. CTA is particularly useful in symptomatic patients with a low pretest likelihood of CAD, or in whom the pain is unlikely to be cardiac in origin.
13. It is recommended to lower the heart rate for CTA to < 60 beats/minute, for example, through premedication with beta blockers. In general, CTA should include the administration of nitroglycerin immediatelyy before the scan.
14. Technical limitations should be evaluated prior to considering CTA, *e.g.*, heart rate, body mass index (BMI), irregular rhythm, asthma, contrast allergy and renal insufficiency.
15. If CTA reveals stenosis between 50–70%, stress testing is the most appropriate next step before resorting to cardiac catheterization.
16. CTA may replace diagnostic catheterization in patients undergoing noncoronary cardiac surgery, *e.g.*, valve replacement or repair, cardiac tumor or repair of congenital heart disease.

D. The Role of CTA in Symptomatic Patients with Known CAD

Consensus

1. Patients with known CAD, *i.e.*, those with a very high pretest probability of having significant CAD and who are experiencing active chest pain, should be taken straight to the catheterization laboratory.
2. Patients with high pretest probability may not be the appropriate candidates for CTA because of a higher positive predictive value of stress MPI and other forms of stress functional testing.
5. CTA is not ready to replace diagnostic catheterization prior to CABG.
6. CTA may be useful in patients with known CAD to rule out progression of disease and presence of new disease in other vessel territories, though technical limitations exist in imaging patients with known CAD and prior angioplasty, stenting and/or CABG.

E. The Role of CTA in the Assessment of Acute Chest Pain in the Emergency Room (for Acute Coronary Syndrome)

Consensus

1. Only a small proportion of emergency room (ER) patients presenting with acute chest pain may be suitable for CTA evaluation due to many exclusionary criteria for the performance of CTA in the ER setting *(see the list of contraindications later in this section)*.
2. Unless medically contraindicated, or a very high-quality scanner is available, *i.e.*, with the fastest temporal resolution, the patient should be given a beta blocker in the ER to achieve a heart rate between 45–60 beats per minute in order to acquire the highest possible image quality. The quality standard used in the ER should be even higher than what is used for routine outpatient imaging.
3. Patients who would be considered for stress MPI or echocardiography to determine the nature of their chest pain may be suitable candidates for cardiac CTA in the ER.
4. Any patient who would be sent straight to the catheterization laboratory or to whom a thrombolytic agent would be administered, *e.g.*, a patient with positive cardiac enzymes or acute electrocardiographic (ECG) changes, should not have their evaluation and treatment delayed by first performing a cardiac CTA.
5. It is preferable to wait until morning than to perform a suboptimal cardiac CTA study during the night with suboptimal imaging staff or patient preparation.
6. Although it is generally not recommended to perform "triple rule-out" on a routine basis, it is appropriate to open up the field of view, if clinically indicated, to rule out additional important diagnoses. However, the primary goal is still to achieve the highest-quality coronary CTA study.

Contraindications for CTA in the emergency room:

1. Atrial fibrillation (except in expert hands, and with careful adjustment of acquisition parameters, reconstruction window, ECG editing, higher radiation exposure due to ECG pulsing off, and knowing when *not* to scan);
2. Grade III renal failure (estimated glomerular filtration rate < 60);
3. History of stents and bypass grafts;
4. Hypotensive or shock state;
5. Allergy to iodinated contrast agents;
6. Uncooperative patient.

F. The Role of CTA in the Assessment of Acute Chest Pain in the Emergency Room ("Triple Rule-Out" Protocol)

Consensus

1. The triple rule-out protocol is rarely needed for cardiac CTA in the ER.
2. Currently, it is neither technically suitable nor medically necessary to perform triple rule-out on a routine basis.
3. Optimal protocols for pulmonary embolism, coronary CTA and CTA for aortic dissection differ; a triple rule-out protocol would not be ideal for all three.
4. The consensus recommendation is to first risk-stratify the patient and then perform a specific CT protocol for a specific indication (*i.e.*, CT pulmonary angiogram with or without a CT venogram of lower extremities to rule out acute pulmonary embolism, thoracic and abdominal CTA to rule out acute dissection, and coronary CTA to rule out acute coronary syndrome).
5. The ultimate goal is to perform the highest-quality study for the specific indication, and to avoid a "shotgun" approach that compromises quality.

G. The Role of CTA in Symptomatic Patients with Known Coronary Artery Disease (Post-Coronary Stenting)

Consensus

1. Not all patients with stents are evaluable by CTA.
2. The evaluability of a stent is very much dependent on the size of the patient and the internal diameter of the stent (> 3 mm stents are more often evaluable).
3. Larger patients exhibit lower image quality due to increases in image noise and reductions in arterial opacification. The general upper limit of the patient's BMI should be 35–40 kg/m^2.
4. The positive predictive value of diagnosing in-stent restenosis using CTA is low. This may be improved by performing CTA on patients with a higher pretest likelihood based on their clinical symptoms.
5. Because they tend to be larger, left main stents are evaluable, but not common.

6. The minimum scanner requirement for evaluating coronary stents is ≥ 64-slice multidetector-row computed tomography (MDCT).
7. The presence of calcium in or around the stent can also negatively affect the evaluability of a coronary artery stent.
8. If symptoms are classic for angina, cardiac catheterization, not CTA, is the appropriate test to perform.

H. The Role of CTA in Symptomatic Patients with Known Coronary Artery Disease (Post-CABG)

Consensus

1. The challenge of CTA in post-CABG patients is the evaluation of the native vessels.
2. Depending on the nature and location of the grafts, assessment can be very difficult due to artifacts from the metallic surgical clips.
3. Motion also plays an important role in making it difficult to assess the distal anastomosis of a bypass graft, especially if there are surgical clips at or adjacent to the anastomosis.
4. Functional assessment using stress MPI or echocardiography may be more useful in this group of patients in general, and should be used first rather than CTA, because the question of graft patency is not as important as the functional significance of the grafts and the native vessels.
5. Exceptions include: reoperation bypass mapping of the previous bypass grafts, the setting of aortic dissection, extremely difficult catheterization, or patients who are high-risk for catheterization, *e.g.*, patients with Marfan's syndrome, or who had strokes from previous catheterization due to severe aortic atherosclerotic disease.
6. If CTA is necessary, it is extremely desirable to have documentation of the prior operation before performing the CTA study.
7. It is inappropriate to perform CTA in an asymptomatic patient post-CABG.
8. There is no consensus on the scan range in the presence of internal mammary artery grafts. Some experts have suggested starting from the subclavian artery, and others from just above the aortic arch.

I. Should a Calcium Score Be Obtained before Performing CTA?

Consensus

1. Calcium scoring (CAC) is applied for risk stratification of asymptomatic patients.
2. CTA is for evaluation of chronic or acute chest pain, and does not absolutely need to be combined with CAC.
3. Some centers use CAC to determine if they should proceed with the CTA study, *e.g.*, they establish a cutoff at a score of 1000, above which they will not proceed with the CTA.
4. CAC scoring should be based upon a single scan, not an average of the results of two scans.

5. Sometimes calcium is so dense that visually there is no need to proceed with the CTA, but this is scanner-dependent due to variation of artifact.
6. It may be sufficient for clinical purposes to estimate the amount of coronary calcium from the coronary CTA itself, and not perform a separate, unenhanced scan.

J. The Role of Contrast Agent Selection in CTA

Consensus

1. Contrast-induced nephropathy (CIN) is associated with poorer outcomes in patients with chronic renal dysfunction.
2. The major risk of CIN is preexisting renal dysfunction.
3. Most of the published studies on CIN involve intra-arterial use of contrast agents, most commonly in cardiac catheterization.
4. CIN is probably dose-dependent.
5. CIN does not seem to be dependent on iodine concentration.
6. Osmolality and iodine concentration are not necessarily directly related; *e.g.*, in the United States, the highest concentration approved for an iodinated contrast agent is 370 mgI/mL; iopamidol 370 is manufactured at that concentration, and yet has a relatively low osmolality.
7. When comparing high-osmolar to low-osmolar contrast agents, CIN may be related to osmolality.
8. Among low-osmolar contrast agents, including iso-osmolar agents, CIN rates may depend on the individual agent.
9. CIN may take 24 to 96 hours to manifest itself through a rise in the serum creatine level; monitoring of at-risk patients should be continued after scanning.
10. A delayed rash is a type IV hypersensitivity reaction to a drug (or contrast agent). It takes hours to days to manifest itself, resists treatment and may take weeks to resolve, similar to exposure to poison ivy. For patients who suffer such a delayed rash after CT scanning, the contrast agent may be the cause.
11. Urticaria (hives) is a type I hypersensitivity reaction that typically manifests itself immediately and resolves quickly, either with or without specific treatment. However, care should be taken to ensure that the patient does not suffer a more serious type I reaction such as throat edema, bronchospasm or shock.
12. As a direct effect, contrast media may cause vasodilation, leading to a decrease in blood pressure that may be associated with some pooling of contrast in major vessels. Data do not demonstrate a difference in heart rate response to iso-osmolar versus low-osmolar contrast agents.
14. The deep breath taken and held during contrast agent administration for coronary CTA could result in a Valsalva maneuver and delay entry of the contrast agent into the heart.
15. In general, the higher the iodine concentration, the better the quality of the CTA study due to improved vascular opacification.

References

1. The Role of CVCT in Imaging of Patients with Suspected CAD

ACCF/AHA 2007 Clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain. *Circulation* 2007;115;402–426.

Alderman EL, Corley SD, Fisher LD, et al. Five-year angiographic follow-up of factors associated with progression of coronary artery disease in the Coronary Artery Surgery Study (CASS). CASS participating investigators and staff. *J Am Coll Cardiol* 1993;22:1141–1154.

Berman DS, Hachamovitch R, Shaw LJ, et al. Roles of nuclear cardiology, cardiac computed tomography, and cardiac magnetic resonance: Assessment of patients with suspected coronary artery disease. *J Nuclear Med* 2006;47:774–782.

Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: A scientific statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation* 2006;114:1761–1791.

Budoff MJ, Georgiou D, Brody A, et al. Ultrafast computed tomography as a diagnostic modality in the detection of coronary artery disease: A multicenter study. *Circulation* 1996;93:898–904.

Budoff MJ, Diamond GA, Raggi P, et al. Continuous probabilistic prediction of angiographically significant coronary artery disease using electron beam tomography. *Circulation* 2002;105:1791–1796.

Georgiou D, Budoff MJ, Kaufer E, et al. Screening patients with chest pain in the emergency department using electron beam tomography: A follow-up study. *J Am Coll Cardiol* 2001;38:105–110.

Greenland P, Bonow RO, Brundage BH, et al. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain. *J Am Coll Cardiol* 2007;49:378–402. [copublished in the January 23, 2007 issue of *Circulation*].

Haberl R, Becker A, Leber A, et al. Correlation of coronary calcification and angiographically documented stenoses in patients with suspected coronary artery disease: Results of 1,764 patients. *J Am Coll Cardiol* 2001;37:451–457.

Hendel RC, Patel MR, Kramer CM, Poon M, Hendel RC, Carr JC, Gerstad NA, Gillam LD, Hodgson JM, Kim RJ, Kramer CM, Lesser JR, Martin ET, Messer JV, Redberg RF, Rubin GD, Rumsfeld JS, Taylor AJ, Weigold WG, Woodard PK, Brindis RG, Hendel RC, Douglas PS, Peterson ED, Wolk MJ, Allen JM, Patel MR; American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American College of Radiology; Society of Cardiovascular Computed Tomography; Society for Cardiovascular Magnetic Resonance; American Society of Nuclear Cardiology; North American Society for Cardiac Imaging; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: A report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. *J Am Coll Cardiol* 2006;48:1475–1497.

Knez A, Becker A, Leber A, et al. Relation of coronary calcium scores by electron beam tomography to obstructive disease in 2,115 symptomatic patients. *Am J Cardiol* 2004;93:1150–1152.

Blumenthal RS, Becker DM, Yanek LR, et al. Use of electron-beam computed tomography in the evaluation of chest pain patients in the emergency department. *Ann Emerg Med* 1999;33:15–21.

McLaughlin VV, Balogh T, Rich S. Utility of electron beam computed tomography to stratify patients presenting to the emergency room with chest pain. *Am J Cardiol* 1999;84:327–328.

Naghavi M, Falk E, Hecht HS, et al; SHAPE Task Force. From vulnerable plaque to vulnerable patient — Part III: Executive summary of the Screening for Heart Attac Prevention and Education (SHAPE) Task Force report. *Am J Cardiol* 2006;98(2A):2H–15H.

2. The Role of CVCT in Imaging of Patients with Known CAD

Alderman EL, Corley SD, Fisher LD, et al. Five-year angiographic follow-up of factors associated with progression of coronary artery disease in the Coronary Artery Surgery Study (CASS). CASS Participating investigators and staff. *J Am Coll Cardiol* 1993;22:1141–1154.

Berman DS, Hachamovitch R, Shaw LJ, et al. Roles of nuclear cardiology, cardiac computed tomography, and cardiac magnetic resonance: Assessment of patients with suspected coronary artery disease. *J Nuclear Med* 2006;47:74–82.

Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: A scientific statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation* 2006;114:1761–1791.

Hendel RC, Patel MR, Kramer CM, Poon M, Hendel RC, Carr JC, Gerstad NA, Gillam LD, Hodgson JM, Kim RJ, Kramer CM, Lesser JR, Martin ET, Messer JV, Redberg RF, Rubin GD, Rumsfeld JS, Taylor AJ, Weigold WG, Woodard PK, Brindis RG, Hendel RC, Douglas PS, Peterson ED, Wolk MJ, Allen JM, Patel MR; American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American College of Radiology; Society of Cardiovascular Computed Tomography; Society for Cardiovascular Magnetic Resonance; American Society of Nuclear Cardiology; North American Society for Cardiac Imaging; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: A report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. *J Am Coll Cardiol* 2006;48:1475–1497.

3. Imaging of Patients with Known or Suspected CAD: The Role of CVCT in Patients with Known or Suspected Vulnerable Plaque

Alderman EL, Corley SD, Fisher LD, et al. Five-year angiographic follow-up of factors associated with progression of coronary artery disease in the Coronary Artery Surgery Study (CASS). CASS participating investigators and staff. *J Am Coll Cardiol* 1993;22:1141–1154.

Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: A scientific statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation* 2006;114:1761–1791.

Dey D, Callister T, Slomka P, et al. Computer-aided detection and evaluation of lipid-rich plaque on noncontrast cardiac CT. *Am J Radiol* 2006;186:S407–S413.

Escolar E, Weigold G, Fuisz A, Weissman NJ. New imaging techniques for diagnosing coronary artery disease. *CMAJ* 2006;174:487–495.

Halliburton SS, Schoenhagen P, Nair A, et al. Contrast enhancement of coronary atherosclerotic plaque: A high-resolution, multidetector-row computed tomography study of pressure-perfused, human ex-vivo coronary arteries. *Coron Artery Dis* 2006;17:553–560.

Hausleiter J, Meyer T, Hadamitzky M, et al. Prevalence of noncalcified coronary plaques by 64-slice computed tomography in patients with an intermediate risk for significant coronary artery disease. *J Am Coll Cardiol* 2006;48:312–318.

Hendel RC, Patel MR, Kramer CM, Poon M, Hendel RC, Carr JC, Gerstad NA, Gillam LD, Hodgson JM, Kim RJ, Kramer CM, Lesser JR, Martin ET, Messer JV, Redberg RF, Rubin GD, Rumsfeld JS, Taylor AJ, Weigold WG, Woodard PK, Brindis RG, Hendel RC, Douglas PS, Peterson ED, Wolk MJ, Allen JM, Patel MR; American College of

C a rdiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American College of Radiology; Society of Cardiovascular Computed Tomography; Society for Cardiovascular Magnetic Resonance; American Society of Nuclear Cardiology; North American Society for Cardiac Imaging; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropria t eness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: a report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. *J Am Coll Cardiol* 2006;48:1475–1497.

Leber AW, Becker A, Knez A, et al. Accuracy of 64-slice computed tomography to classify and quantify plaque volumes in the proximal coronary system: A comparative study using intravascular ultrasound. *J Am Coll Cardiol* 2006;47:672–677.

Maintz D, Ozgun M, Hoffmeier A, et al. Selective coronary artery plaque visualization and differentiation by contrast-enhanced inversion recovery MRI. *Eur Heart J* 2006;27:1732–1736.

Naghavi M, Falk E, Hecht HS, et al for the SHAPE Task Force. From vulnerable plaque to vulnerable patient — Part III: Executive summary of the Screening for Heart Attack Prevention and Education (SHAPE) Task Force report. *Am J Cardiol* 2006;98:2H–15H.

Rasouli ML, Shavelle DM, French WJ, et al. Assessment of coronary plaque morphology by contrast-enhanced computed tomographic angiography: Comparison with intravascular ultrasound. *Coron Artery Dis* 2006;4:359–364.

4. The Role of CVCT in Evaluating Patients Presenting with Acute Chest Pain: The "Triple Rule-Out"

Chiles C, Carr J. Vascular diseases of the thorax: Evaluation with multidetector CT. *Radiology Clino N Am* 2005;43:543–69.

Hendel RC, Patel MR, Kramer CM, Poon M, Hendel RC, Carr JC, Gerstad NA, Gillam LD, Hodgson JM, Kim RJ, Kramer CM, Lesser JR, Martin ET, Messer JV, Redberg RF, Rubin GD, Rumsfeld JS, Taylor AJ, Weigold WG, Woodard PK, Brindis RG, Hendel RC, Douglas PS, Peterson ED, Wolk MJ, Allen JM, Patel MR; American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American College of Radiology; Society of Cardiovascular Computed Tomography; Society for Cardiovascular Magnetic Resonance; American Society of Nuclear Cardiology; North American Society for Cardiac Imaging; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: A report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. *J Am Coll Cardiol* 2006;48:1475–1497.

Leber AW, Knez A, Ziegler F, et al. Quantification of obstructive and nonobstructive coronary lesions by 64-slice computed tomography: A comparative study with quantitative coronary angiography and intravascular ultrasound. *J Am Coll Cardiol* 2005;46:147–155.

Raff G, Gallagher MJ, O'Neill WW, et al. Diagnostic accuracy of non-invasive coronary angiography using 64-slice spiral computed tomography. *J Am Coll Cardiol* 2005;6:552–557.

5. The Role of CVCT in Imaging of Patients with Preexisting CAD for Post-Revascularization Follow Up

Anders K, Baum U, Schmid M, et al. Coronary artery bypass graft (CABG) patency: Assessment with high-resolution submillimeter 16-slice multidetector-row computed tomography (MDCT) versus coronary angiography. *Eur J Radiol* 2006;57:336–344.

Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: A scientific Statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation* 2006;114:1761–1791.

Cademartiri F, Mollet N, Lemos PA, et al. Usefulness of multislice compu t ed tomographic coronary angiography to assess in-stent restenosis. *Am J Cardiol* 2005;96:799–802.

Chiurlia E, Menozzi M, Ratti C, et al. Follow-up of coronary artery bypass graft patency by multislice computed tomography. *Am J Cardiol* 2005;95:1094–1097.

Gilard M, Cornily JC, Rioufol G, et al. Noninvasive assessment of left main coronary stent patency with 16-slice computed tomography. *Am J Cardiol* 2005;95:110–112.

Gilard M, Cornily JC, Pennec PY, et al. Assessment of coronary artery stents by 16 slice computed tomography. *Heart* 2006;92:58–61.

Hendel RC, Patel MR, Kramer CM, Poon M, Hendel RC, Carr JC, Gerstad NA, Gillam LD, Hodgson JM, Kim RJ, Kramer CM, Lesser JR, Martin ET, Messer JV, Redberg RF, Rubin GD, Rumsfeld JS, Taylor AJ, Weigold WG, Woodard PK, Brindis RG, Hendel RC, Douglas PS, Peterson ED, Wolk MJ, Allen JM, Patel MR; American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group; American College of Radiology; Society of Cardiovascular Computed Tomography; Society for Cardiovascular Magnetic Resonance; American Society of Nuclear Cardiology; North American Society for Cardiac Imaging; Society for Cardiovascular Angiography and Interventions; Society of Interventional Radiology. ACCF/ACR/SCCT/SCMR/ASNC/NASCI/SCAI/SIR 2006 Appropriateness criteria for cardiac computed tomography and cardiac magnetic resonance imaging: A report of the American College of Cardiology Foundation Quality Strategic Directions Committee Appropriateness Criteria Working Group, American College of Radiology, Society of Cardiovascular Computed Tomography, Society for Cardiovascular Magnetic Resonance, American Society of Nuclear Cardiology, North American Society for Cardiac Imaging, Society for Cardiovascular Angiography and Interventions, and Society of Interventional Radiology. *J Am Coll Cardiol* 2006;48:1475–1497.

Hong C, Chrysant GS, Woodard PK, Bae KT. Coronary artery stent patency assessed with in-stent contrast enhancement measured at multidetector row CT angiography: Initial experience. *Radiology* 2004;233:286–291.

Knollmann FD, Moller J, Gebert A, et al. Assessment of coronary artery stent patency by electron-beam CT. *Eur Radiol* 2004;14:1341–1347.

Mahnken AH, Buecker A, Wildberger JE, et al. Coronary artery stents in multislice computed tomography: In vitro artifact evaluation. *Invest Radiol* 2004;39:27–33.

Mahnken AH, Seyfarth T, Flohr T, et al. Flat-panel detector computed tomography for the assessment of coronary artery stents: Phantom study in comparison with 16-slice spiral computed tomography. *Invest Radiol* 2005;40:8–13.

Marano R, Storto ML, Maddestra N, Bonomo L. Non-invasive assessment of coronary artery bypass graft with retrospectively ECG-gated four-row multi-detector spiral computed tomography. *Eur Radiol* 2004;14:1353–1362.

Martuscelli E, Romagnoli A, D'Eliseo A, Tomassini M, Razzini C, Sperandio M, Simonetti G, Romeo F, Mehta J. Evaluation of venous and arterial conduit patency by 16-slice spiral computed tomography. *Circulation* 2004;110:3234–3238.

Moore RKG, Sampson C, Mac Donald S, et al. Coronary artery bypass graft imaging using ECG-gated multislice computed tomography: Comparison with catheter angiography. *Clin Radiol* 2005;60:990–998.

Rossi R, Chiurlia E, Ratti C, et al. Noninvasive assessment of coronary artery bypass graft patency by multislice computed tomography. *Ital Heart J* 2004;5:36–41.

Schlosser T, Konorza T, Hunold P, et al. Noninvasive visualization of coronary artery bypass grafts using 16-detector row computed tomography. *J Am Coll Cardiol* 2004;44:1224–1229.

Schuijf JD, Bax JJ, Jukema JW, et al. Feasibility of assessment of coronary stent patency using 16-slice computed tomography. *Am J Cardiol* 2004;94:427–430.

6. Contrast Media Considerations

American College of Radiology. Manual on Contrast Media, version 5.0, 2004, Reston, Virginia, p. 13.

Aspelin P, Aubry P, Fransson SG, et al. Nephrotoxic effects in high-risk patients undergoing angiography. *N Engl J Med* 2003;348:491–499.

Barrett BJ, Carlisle EJ. Meta-analysis of the relative nephrotoxicity of high- and low-osmolality iodinated contrast media. *Radiology* 1993;188:171–178.

Barrett BJ, Katzberg RW, Thomsen HS, et al. Contrast-induced nephropathy in patients with chronic kidney disease undergoing computed tomography: A double-blind comparison of iodixanol and iopamidol. *Invest Radiol* 2006;41:815–821.

Becker CR. The use of contrast media in cardiac CT. *Appl Radiol* 2003;December:50–56.

Briguori C, Colombo A, Airoldi F, et al. Nephrotoxicity of low-osmolality versus iso-osmolality contrast agents: Impact of N-acetylcysteine. *Kidney Int* 2005;68:2250–2255.

Cademartiri F, Mollet NR, van der Lugt A, et al. Intravenous contrast material administration at helical 16-detector row CT coronary angiography: Effect of iodine concentration on vascular attenuation. *Radiology* 2005;236:661–665.

Choi CU, Rha SW, Suh SY, et al. Extensive exfoliative dermatitis induced by non-ionic contrast medium iodixanol (Visipaque) used during percutaneous coronary intervention. *Int J Cardiol* 2007;doi:10.1016/j.ijcard.2006.11.179

Ho AL, O'Malley ME, Tomlinson GA. AEs with universal use of iodixanol for CT: Comparison with iohexol. *J Comput Assist Tomogr* 2007;31:165–168.

Hosoya T, Yamguchi K, Akutsu T, et al. Delayed adverse reactions to iodinated contrast media and their risk factors. *Radiat Med* 2000;18:39–45.

Mehran R, et al. ICON: Ionic versus nonionic Contrast to Obviate worsening Nephropathy after angioplasty in chronic renal failure patients, presented at the Cardiovascular Research Foundation's annual Transcatheter Cardiovascular Therapeutics meeting, October 2006.

Schmid I, Dominique D, Pfammatter T, et al. Effects of non-ionic iodinated contrast media on patient heart rate and pressures during intra-cardiac or intra-arterial injection. *Int J Cardiol* 2007;118:389–396.

Mikkonen R, Kontkanen T, Kivisaari L. Acute and late adverse reactions to low-osmolal contrast media. *Acta Radiol* 1995;36:72–76.

Munechika H, Hiramatsu Y, Kudo S, et al. A prospective survey of delayed adverse reactions to iohexol in urography and computed tomography. *Eur Radiol* 2003;13:185–194.

Rudnick MR, Goldfarb S, Wexler L, et al. Nephrotoxicity of ionic and non-ionic contrast media in 1196 patients: A randomized trial. *Kidney Int* 1995;47:254–261.

Solomon R, Du Mouchel W. Contrast media and nephropathy: Finding from systematic analysis and Food and Drug Administration report of adverse effects. *Invest Radiol* 2006;41:651–660.

Solomon R, Natarajan M, Doucet S, et al. The Cardiac Angiography in REnally Impaired Patients (CARE) study: A randomized, double-blind trial of contrast-induced nephropathy (CIN) in high-risk patients. Presented at the Cardiovascular Research Foundation's annual Transcatheter Cardiovascular Therapies meeting, October 2006.

Sutton AGC, Finn P, Grech ED, et al. Early and late reactions after the use of iopamidol 340, ioxaglate 320, and iodixanol 320 in cardiac catheterization. *Am Heart J* 2001;141:677–683.

Webb JAW, Stacul F, Thomsen HS, et al. Late adverse reactions to intravascular iodinated contrast media. *Eur Radiol* 2003;13:181–184.

Yeung AC. Best of the TCT scientific sessions 2006: Highlights from the Transcatheter Cardiovascular Therapeutics Scientific Symposium, October 22–27, 2006, Washington, D.C. *Rev Cardiovasc Med* 2006;7:228–237.

ACCF/AHA CLINICAL COMPETENCE STATEMENT ON CARDIAC CT AND MR

ACCF/AHA Clinical Competence Statement on Cardiac Imaging With Computed Tomography and Magnetic Resonance

A Report of the American College of Cardiology Foundation/ American Heart Association/American College of Physicians Task Force on Clinical Competence and Training

Developed in Collaboration With the American Society of Echocardiography, American Society of Nuclear Cardiology, Society of Atherosclerosis Imaging, and the Society for Cardiovascular Angiography & Interventions

Endorsed by the Society of Cardiovascular Computed Tomography

WRITING COMMITTEE MEMBERS
MATTHEW J. BUDOFF, MD, FACC, FAHA, *Chair*

MYLAN C. COHEN, MD, MPH, FACC, FAHA†
MARIO J. GARCIA, MD, FACC‡
JOHN McB. HODGSON, MD, FSCAI††
W. GREGORY HUNDLEY, MD, FACC, FAHA*
JOAO A. C. LIMA, MD, FACC, FAHA

WARREN J. MANNING, MD, FACC, FAHA**
GERALD M. POHOST, MD, FACC, FAHA
PAOLO M. RAGGI, MD, FACC‡‡
GEORGE P. RODGERS, MD, FACC
JOHN A. RUMBERGER, MD, PhD, FACC

ALLEN J. TAYLOR, MD, FACC, FAHA

*AHA Representative, **SCMR Representative, †ASNC Representative, ††SCAI Representative, ‡ASE Representative, ‡‡SAI Representative

TASK FORCE MEMBERS
MARK A. CREAGER, MD, FACC, FAHA, *Chair*

JOHN W. HIRSHFELD, JR, MD, FACC, FAHA
BEVERLY H. LORELL, MD, FACC, FAHA*
GENO MERLI, MD, FACP

GEORGE P. RODGERS, MD, FACC
CYNTHIA M. TRACY, MD, FACC, FAHA
HOWARD H. WEITZ, MD, FACC, FACP

*Former Task Force member

TABLE OF CONTENTS

PREAMBLE

The granting of clinical staff privileges to physicians is a primary mechanism used by institutions to uphold the quality of care. The Joint Commission on Accreditation of Health Care Organizations requires that the granting of continuing medical staff privileges be based on assessments of applicants against professional criteria specified in the medical staff bylaws. Physicians themselves are thus charged with identifying the criteria that constitute professional competence and with evaluating their peers accordingly. Yet the process of evaluating physicians' knowledge and competence is often constrained by the evaluator's own knowledge and ability to elicit the appropriate information, problems compounded by the growing number of highly specialized procedures for which privileges are requested.

The American College of Cardiology Foundation/American Heart Association/American College of Physicians (ACCF/AHA/ACP) Task Force on Clinical Competence was formed in 1998 to develop recommendations for attaining and maintaining the cognitive and technical skills necessary for the competent performance of a specific cardiovascular service, procedure, or technology. These documents are evidence-based, and where evidence is not available, expert opinion is utilized to formulate recommendations. Indications and contraindications for specific services or procedures are not included in the scope of these

documents. Recommendations are intended to assist those who must judge the competence of cardiovascular health care providers entering practice for the first time and/or those who are in practice and undergo periodic review of their practice expertise. The assessment of competence is complex and multidimensional; therefore, isolated recommendations contained herein may not necessarily be sufficient or appropriate for judging overall competence.

The ACCF/AHA/ACP Task Force makes every effort to avoid any actual or potential conflicts of interest that might arise as a result of an outside relationship or a personal interest of a member of the ACCF/AHA/ACP Writing Committee. Specifically, all members of the Committee are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest relevant to the document topic. These changes are reviewed by the Committee and updated as changes occur. The relationship with industry information for the Writing Committee members is published in the appendix of this document.

Mark A. Creager, MD, FACC, FAHA
Chair, ACCF/AHA/ACP Task Force on
Clinical Competence and Training

INTRODUCTION

The disciplines of cardiac imaging using computed tomography (CT) and magnetic resonance imaging (MRI) define unique areas worthy of competence. Existence of multidisciplinary practitioners in the field, the complex nature of the imaging devices and anatomy, and the rapidly advancing uses of these modalities require credentialing guidelines for physicians in, hospital as well as private, outpatient settings. The guidelines are broad-based and applicable to cardiovascular practitioners from multiple medical backgrounds. This statement on clinical competence is designed to assist in the assessment of physicians' expertise in the ability to apply and interpret cardiovascular computed tomography (CCT) and cardiovascular magnetic resonance (CMR). The minimum education, training, experience, and cognitive skills necessary for the evaluation and interpretation of cardiac imaging using these newer approaches are specified. It is important to note that these are minimum training and experience requirements for the assessment of expertise in these approaches in the broadest sense. The specifications are applicable to most practice settings and can accommodate a number of ways in which physicians can substantiate expertise and competence in utility of either CCT or CMR.

Moreover, it is important to stress that competence levels for CCT and CMR are distinct and require separate training. This document specifically applies to cardiac applications of these two modalities. The official name for the discipline of magnetic resonance (MR) applied to the cardiovascular system per the Society for Cardiovascular Magnetic Resonance (SCMR) is "cardiovascular magnetic resonance" whether it is applied to the heart alone (includ-

ing the coronary arteries) or the heart and the peripheral blood vessels. Because of the complexities of the peripheral anatomy as well as the different methods of interpretation and acquisition, peripheral imaging using either modality is outside the scope of this document and will require separate attention and training.

The Writing Committee includes representatives from the American College of Cardiology (ACC), the American Heart Association (AHA), the American Society of Echocardiography (ASE), the American Society of Nuclear Cardiology (ASNC), the Society of Atherosclerosis Imaging (SAI), the Society for Cardiovascular Angiography and Interventions (SCAI), and the SCMR. Peer review included two official representatives from the ACC and AHA; organizational review was done by the ASE, ASNC, SCAI, Society of Cardiovascular Computed Tomography (SCCT), SCMR, and SAI, as well as 40 content reviewers. This document was approved for publication by the governing bodies of the ACC and AHA. In addition, the governing boards of the ASE, ASNC, SAI, SCAI, and SCCT have reviewed and formally endorsed this document.

Rationale for developing a competence statement. In this document, the term "cardiac disease" refers to acquired and congenital diseases of the heart muscle, valves, pericardium, coronary arteries and veins, pulmonary veins, and diseases of the thoracic aorta. Diseases of the pulmonary arteries (e.g., pulmonary embolism), peripheral vascular system, and carotid, renal, and intracranial vessels are outside the realm of this document. Furthermore, this document addresses other clinical imaging applications of both CCT and CMR. For CCT, anatomic, functional imaging, coronary calcium, non-calcified plaque assessment, and CCT use in congenital heart disease (CHD) will be included. For CMR, its use in anatomic, functional, and perfusion imaging, vasodilator or dobutamine stress imaging, viability, plaque assessment, valvular disease, and CHD will be discussed.

Coronary heart disease constitutes the most common cause of morbidity and mortality in Western society. Scientific advances have substantially increased the diagnostic capabilities of both CCT and CMR. Most cardiovascular and radiology programs do not provide formal post-training education in CCT and CMR, yet there is a strong need to establish competence guidelines for practicing physicians in these emerging fields. This document does not replace the Cardiovascular Medicine Core Cardiology Training (CO-CATS) document on CMR (1), which specifically addresses training requirements during cardiovascular fellowship, nor the recommendations made by the American College of Radiology (ACR) (2). This document is intended to be and is complementary to the SCMR statement regarding training requirements during fellowship and for practicing physicians (1,3) and to recommendations by the ACR (2). It must be understood that the SCMR guidelines, which require relatively more "in laboratory" training than the guidelines listed here, include the field of vascular imaging.

Whereas cardiologists, nuclear medicine specialists, and radiologists should possess core knowledge of cardiovascular physiology and imaging, it is unreasonable to expect the majority of such physicians to be fully conversant with all potential uses of CCT or CMR. Thus, there is a role for specialists who have more in-depth understanding of the utility and diagnostic capability of CCT and CMR.

Medical specialists trained in the distinct disciplines of cardiovascular medicine, radiology, and nuclear medicine are all involved in the imaging of cardiovascular diseases, albeit from differing perspectives. These perspectives, however, also share many common features, emphasizing the importance of a broadly based, multi-disciplinary approach for management. These specialist physicians also can be subdivided into those who have exposure or training in CCT and those who have exposure or training in CMR. Each of these subsets of physicians concerned with the care of the patient with cardiovascular disease must hold a specialized knowledge base that is applicable to one's particular imaging discipline. This document addresses the minimal knowledge base required for expertise, the education and training pathways available to acquire that expertise, and the requirements to maintain expertise for each of the two related disciplines that involve tomographic cardiac imaging with CCT and CMR. Accordingly, this document is presented in two major sections: 1) CCT, and 2) CMR. Each section describes the cognitive, clinical, and/or procedural skills required for expertise, the training necessary for achieving competence, and the means for maintaining that expertise and competence.

COMPUTED TOMOGRAPHY (CT)

Overview of X-Ray CT

"Computed tomography" is a generic term that can apply to several methods currently employed in the evaluation of cardiovascular diseases. The first discussion must be one of semantics in defining CT derived in a specific manner using X-ray information from multiple sites. From here forward, CT will refer to the latter method partly by tradition and mostly by convention.

The development of CT, resulting in widespread clinical use of CT scanning by the early 1980s, was a major breakthrough in clinical diagnosis. Imaging a thin axial cross-section of the body avoided superposition of three-dimensional (3D) structures onto a planar two-dimensional (2D) representation, as is the problem with conventional projection X-ray. The basic principle of CT is that a fan-shaped, thin X-ray beam passes through the body at many angles to allow for cross-sectional images. The corresponding X-ray transmission measurements are collected by a detector array. Information entering the detector array and X-ray beam itself is collimated to produce thin sections and avoid unnecessary photon scatter. The transmission measurements recorded by the detector array are digitized into picture elements (pixels) with known dimensions. The

gray-scale information contained in each individual pixel is reconstructed according to the attenuation of the X-ray beam along its path using a standardized technique termed "filtered back projection." Gray-scale values for pixels within the reconstructed tomogram are defined with reference to the value for water and are called "Hounsfield Units" (HU) (for the 1979 Nobel Prize winner, Sir Godfrey N. Hounsfield) or simply "CT numbers." Air attenuates the X-ray less than water, and bone attenuates it more than water, so that in a given patient, the HU may range from −1,000 HU (air) through 0 HU (water) to approximately +1,000 HU (bone cortex). A range of 2,000 gray-scale values represents densities of various hard and soft tissues within the body and between these two extreme limits.

The CT technology has significantly improved since its introduction into clinical practice in 1973. Current conventional scanners used for cardiac and cardiovascular imaging now employ either a rotating X-ray source with a circular, stationary detector array (spiral or helical CT) or a rotating electron beam (electron beam computed tomography [EBCT]). Continuous or step increments of the patient table using electron beam methods allow imaging at 50 to 100 ms or continuous scanning (spiral or helical CT or multi-detector computed tomography [MDCT]), allowing for image reconstruction windows now on the order of 200 to 400 ms with short inter-scan delay. Today, 64-slice MDCT scanners provide enhanced scan modes of temporal resolution as low as 165 ms, and in multi-sector mode a range of temporal resolution as low as 100 ms. Improved temporal resolution should lead to lower motion artifacts and possibly higher diagnostic rates. Reconstruction algorithms and multi-row detectors common to both current EBCT and spiral/helical CT have been implemented, enabling volumetric imaging, and multiple high-quality reconstructions of various volumes of interest can be done either prospectively or retrospectively, depending on the method.

Although the purpose of this statement is to provide an overview of the requirements of competence in current CCT and MRI technology, continued efforts will be required to maintain competence as additional technological improvements and modifications are made in CCT hardware and software.

Minimal knowledge and skills required for expertise in CCT. Table 1 lists common CCT procedures performed currently in many hospital-based inpatient and outpatient imaging centers and in some private imaging clinics.

Cognitive skills required to demonstrate competence in CCT are summarized in Table 2. Candidates for competence in CCT shall have completed a formal residency in general radiology or nuclear medicine or will have completed an Accreditation Council for Graduate Medical Education (ACGME)-approved cardiovascular fellowship. A thorough knowledge and understanding of cardiac and vascular anatomy is required. Because cardiology, nuclear medicine, and radiology training is very much involved with

T1

T2

Table 1. Classification of CCT Procedures

Cardiac:
- Static tomographic and 3D non-contrast and contrast-enhanced anatomy of the heart, heart chambers, and pericardium (electron beam tomography [EBT] and multi-detector computed tomography [MDCT])
- Dynamic contrast-enhanced assessment of left and right ventricular function (EBT and MDCT)
- Quantitative coronary artery calcium scoring and interpretation (EBT and MDCT)
- Performance and interpretation of tomographic and 3D contrast-enhanced CCT coronary angiography, including native and anomalous coronary vessels and coronary bypass grafts, aortic root, proximal pulmonary arteries, superior and inferior vena cavae, pulmonary veins (EBT and MDCT), and common congenital abnormalities involving the heart and central vasculature

Thoracic Aorta:
- Static tomographic and 3D non-contrast and contrast-enhanced anatomy of central vasculature (thoracic aorta) (EBT and MDCT)
- Performance and interpretation of tomographic and 3D contrast-enhanced CCT central vascular angiography including aortic arch and thoracic aorta (EBT and MDCT)

anatomic definition, this requirement should be met or would have been met by individuals completing an ACGME-approved cardiovascular fellowship, nuclear medicine residency, or general radiology residency. Likewise, characteristics of the heart in health and disease by traditional cardiac imaging methods (echocardiography, nuclear medicine, and angiography) will provide a significant background for application to CCT. These dynamic tomographic or projection imaging techniques of the heart are commonplace in formal cardiology training, so little additional instruction is required when interpreting dynamic CCT sequences of the heart for cardiologists (e.g., evaluat-

Table 2. Cognitive Skills Required for Competence in CCT

General:
- Knowledge of the physics of CT and radiation generation and exposure
- Knowledge of scanning principles and scanning modes for non-contrast and contrast-enhanced cardiac imaging using multi-detector and/or electron beam methods
- Knowledge of the principles of intravenous iodinated contrast administration for safe and optimal cardiac imaging
- Knowledge of recognition and treatment of adverse reactions to iodinated contrast
- Knowledge of the principles of image postprocessing and appropriate applications

Cardiac:
- Clinical knowledge of coronary heart disease and other cardiovascular diseases
- Knowledge of normal cardiac, coronary artery, and coronary venous anatomy, including associated pulmonary arterial and venous structures
- Knowledge of pathologic changes in cardiac and coronary artery anatomy due to acquired and congenital heart disease
- Basic knowledge in ECG to recognize artifacts and arrhythmias

Aorta:
- Knowledge of normal thoracic arterial anatomy
- Knowledge of pathologic changes in central arterial anatomy due to acquired and congenital vascular disease

ing ventricular function by watching the wall motion throughout a cardiac cycle). Cardiac physiology is also vital for CCT and CMR, and basic training should be part of both formal cardiology fellowship and radiology residency. Competence in peripheral CT is beyond the scope of this report. A brief overview of the technical aspects of CCT is included to facilitate understanding of the terms used in the subsequent sections of this report and is not intended to be comprehensive.

Coronary artery calcium quantification is now commonplace as a means of detecting coronary and peripheral vascular atherosclerotic disease, but will require specific CCT training in addition to traditional radiology residency, nuclear medicine residency, or cardiology fellowship training. A full discussion of computer workstation methods is beyond the scope of this document, but the candidate will be required to show competence in manipulation of the tomographic datasets.

Myocardial perfusion imaging can be performed using electron beam tomography (EBT) (4) and follows principles of first-pass kinetics and perfusion imaging by nuclear medicine methods; however, this application is not yet appropriately validated for routine use in cardiac CT. Because CCT is expected to undergo rapid technical evolution, current training requirements specifically cover noncontrast studies and contrast studies involving angiography and function, but not perfusion imaging. As this modality evolves and further matures, training requirements may change.

As many CCT studies are done before and after intravenous administration of iodinated contrast, a thorough understanding of contrast injection methods, adverse events and their treatments, and contrast kinetics in patients will be required. In particular, knowledge is needed in the methods of contrast-enhanced imaging of the pericardium, right ventricle (RV), right atrium, and superior and inferior vena cavae as well as imaging of the left heart, surrounding great vessels, and the central circulation.

CT physics and nature of radiation exposure. The physician will be required to demonstrate competence in the principles of CCT imaging using EBT and/or MDCT and tomographic imaging production. Candidates should receive didactic lectures from a qualified CT-trained physician and/or physicist on the basic physics of CT in general and of CCT in particular.

EBT. Electron beam tomography is a Food and Drug Administration (FDA)-approved body-imaging device developed over 20 years ago and is the only CT device specifically designed from inception for cardiac imaging. Since EBT first appeared in 1984, there has been significant validation for this approach for cardiac and body imaging, with imaging times as low as 50 ms. The EBT method is distinguished by its use of a scanning electron beam rather than a traditional X-ray tube and mechanical rotating device used in current "spiral" single and multiple detector scan-

ners. The electron beam (cathode) is steered by an electromagnetic deflection system that sweeps the beam across the distant anode, a series of fixed "target" rings. A stationary single or multi-level detector lies in apposition to the target rings. The technique can be used to quantify ventricular anatomy and global and regional function (5), for quantitation of coronary artery calcified plaque (6–8), noninvasive coronary angiography (9–12), and central and peripheral vascular anatomy and angiography. There have been three iterations for EBT since it was introduced clinically in the early 1980s. In addition to the standard 50-ms and 100-ms scan modes common to all EBT scanners, current generation units are capable of imaging speeds as fast as 33 ms per tomographic section, as well as multi-level image acquisition in the high resolution mode.

MDCT. Helical/spiral CT has undergone considerable changes in the past five years, from a single slice/detector to multiple slices/detectors. This modality employs a rotating X-ray source with a circular, stationary detector array. Continuous incrimination of the patient table has enabled continuous scanning (spiral or helical CT), allowing for image reconstruction windows on the order of 165 to 400 ms with shortened inter-scan delay. Reconstruction algorithms and multi-row detectors have been implemented, enabling volumetric imaging, and multiple high-quality reconstructions of various volumes of cardiovascular interest can be done in retrospect with even shorter image reconstruction windows (multi-sector reconstructions). Current generation MDCT systems are capable of acquiring data from 40 or 64 (and potentially greater) levels of the body simultaneously. Cardiac imaging is facilitated using electrocardiographic (ECG) gating in either a prospective or retrospective mode (11–13). The MDCTs differ from single-slice helical or spiral CT systems principally by the design of the detector arrays and data acquisition systems. The new design allows the detector arrays to be configured electronically to acquire multiple levels of various slice thickness simultaneously. Measurement of the true maximum (end-diastolic) and true minimum (end-systolic) volumes are more problematic with MDCT (as compared to EBT and especially CMR) owing to lower temporal resolution.

In MDCT systems, like the preceding generation of single-slice helical scanners, the X-ray photons are generated within a specialized X-ray tube mounted on a rotating gantry. The patient is centered within the bore of the gantry such that the array of detectors is positioned to record incident photons after traversing the patient. Within the X-ray tube a tungsten filament allows the tube current to be increased (in mA) which proportionately increases the number of X-ray photons for producing an image. This ability to vary the power is a substantial design difference with current generation EBT systems, which has only two mA settings (14). The attenuation data (after passing from the source, through the body, and incident on the detector

array) are recorded and transformed through a filtered back-projection into the CT image. This final step is common to both EBT and MDCT.

Radiation dose. The CCT utilizes X-rays, a form of ionizing radiation, to produce the information required for generating CCT images. Although ionizing radiation from natural sources is part of our daily existence, a role of health care professionals involved in medical imaging is to understand the potential risks of the test and balance those against the potential benefits. This is particularly true for diagnostic tests which are applied to healthy individuals as part of a disease-screening or risk-stratification program. Health care professionals must have an understanding of the exposure involved in CCT to effectively advise candidates for imaging.

Because of the dangers of ionizing radiation, a comprehensive understanding must be obtained in physics and radiation safety for anyone involved with CCT. Patient doses for CCT acquisition should be set at the lowest values that are consistent with satisfactory image quality. Most candidates will likely have had some didactic training regarding radiation physics during radiology residency, nuclear medicine residency, or cardiology fellowship. However, specific instructions in the need to keep radiation exposure to the patient to a minimum when performing CCT will be required.

In general, there are differences in radiation exposure depending on the examination performed and the CT method (EBT vs. MDCT). Adoption of the *effective dose* as a standard measure of dose allows comparability across the spectrum of medical and non-medical exposures. "The effective dose is, by definition, an estimate of the uniform, whole-body equivalent dose that would produce the same level of risk for adverse effects that results from the non-uniform partial body irradiation. The unit for the effective dose is the milliSievert (mSv)" (www.fda.gov/cdrh/ct/rqu.html). The typical radiation dose for calcium scanning using EBT is 0.5 to 0.7 mSv, increasing with 4- to 16-slice MDCT (prospective gating) to 0.8 to 1.5 mSv, and for MDCT (retrospective gating) is up to 6.2 mSv (13–17). Radiation dose exposure for coronary angiography is much higher. Using EBT coronary angiography yielded effective doses of 1.5 and 2.0 mSv for male and female patients, respectively (15). Effective doses delivered during 16-slice MDCT coronary angiography are reported to be 6.7 to 10.9 mSv for male patients and 8.1 to 13.0 mSv for female patients (15,16).

In MDCT coronary angiography, the dose can be reduced by 30% to 50% using ECG-controlled dose modulation techniques (18). For both EBT and MDCT, the radiation dose increases with thinner slices and more overlapping images (13). In comparison, routine conventional diagnostic X-ray coronary angiography is associated with effective doses of 2.1 and 2.5 mSv for male and female patients, respectively (15). Depending on the operator and the nature of the diagnostic procedure, the effective dose of X-ray coronary angiography can be significantly higher. Understanding the appropriate use of prospective triggering (EBT), prospective gating (MDCT), and retrospective gating (MDCT), especially given the patient radiation dose implications is important (9,13–16).

The current EBT configuration has two power (mA) settings and performs prospective triggering through only 210° of arc, so radiation dose is reduced, and there is limited opportunity either to increase or decrease radiation dose to the patient with varying protocols. However, this is not the case with MDCT angiography, which images through 360° of radiation exposure.

There are various choices with MDCT that can dramatically change the patient radiation dose during coronary computed tomography angiography (CTA). Because of rapid technical advances, scanning protocols for MDCT have not yet been standardized. Controversies about optimal tube current and voltage are ongoing. Dose (in mA) can be increased or decreased, and this is most often based upon the body habitus of the patient (16). Furthermore, slice thickness can be decreased. However, one 16-slice MDCT study that utilized high mA and 0.5-mm slice thickness for CTA (thus achieving nearly isotropic imaging) reported radiation doses as high as 24.2 mSv per patient (13). This can be reduced by using dose modulation (turning down the radiation exposure during parts of the cardiac cycle that imaging is not useful) during systolic cardiac phases; however, this is also dependent upon the patient's heart rate (17), and cannot be applied in all cases. Although the efficacy of dose modulation depends on the heart rate, it can theorectically be applied to any heart rate with the 64-detector CT scanner. Beta-blockade to achieve heart rates below 60 beats/min is still most often part of the MDCT angiogram (10,11,18,19).

CT laboratory requirements. Defining the specific requirements for a valid CCT laboratory is beyond the scope of this physician competence document. However, some general aspects of the appropriate CT environment can be considered. A continuous quality control (QC) program must be established for all CT units with the assistance of a qualified medical physicist and a Level 3-trained physician (training levels are described in the following text). The scanners must be staffed by qualified CT technologists with appropriate background and/or training in CCT imaging.

Several states require all CT operators to qualify for a state permit. A current permit should be held by all technologists and Level 2- and 3-trained physicians, when required by state law.

Training to achieve clinical competence in CCT (Table 3). The recommendations for all levels of training in the following text represent a cumulative experience, and it is expected that for many practicing clinicians the training will not be continuous. A summary of the training requirements is given in Table 3. Time spent at didactic continuing medical education courses specifically targeting CCT can contribute to the total time. Due to the advancement in the

T3

Table 3. Requirements for CCT Study Performance and Interpretation to Achieve Level 1, 2, and 3 Clinical Competence

	Cumulative Duration of Training	Minimum Number of Mentored Examinations Performed	Minimum Number of Mentored Examinations Interpreted
Level 1	4 weeks*	—	50†
Level 2—non-contrast	4 weeks*	50	150†
Level 2—contrast	8 weeks*	50	150†
Level 3	6 months*	100	300†

*This represents cumulative time spent interpreting, performing, and learning about CCT, and need not be a consecutive block of time, but at least 50% of the time should represent supervised laboratory experience. In-lab training time is defined as a minimum of 35 h/week. †The case load recommendations may include studies from an established teaching file, previous CCT cases, journals and/or textbooks, or electronic/on-line courses/CME.

sophistication and widespread availability of electronic training medias, the committee felt that some training can now be obtained outside the laboratory setting. However, for all Level 2 and 3 requirements, minimum time in a CCT laboratory is half of the time listed, with the other half garnered by supervised time, CT exposure and other courses, case studies, CD/DVD training, time at major medical meetings devoted to performance of CCT, or other relevant educational training activities, as a few examples. Several aspects of CCT can be learned from the general CT expert, including use of the workstation, tomographic imaging, and radiation physics, among others. The caseload recommendations may include studies from an established teaching file, previous CCT cases, and electronic/on-line experience or courses.

For all levels of competence, it is expected that the candidate will attend lectures on the basic concepts of CCT and include parallel self-study reading material. A basic understanding of CCT should be achieved, including the physics of CCT imaging, the basics of CCT scan performance, safety issues in CCT performance, post-processing methods, and the basics of CCT interpretation as compared with other cardiovascular imaging modalities, which include echocardiography, nuclear medicine, CMR, and invasive cardiac and peripheral X-ray angiography.

LEVEL 1 TRAINING. Level 1 is defined as the minimal introductory training for familiarity with CCT, but is not sufficient for independent interpretation of CCT images. The individual should have intensive exposure to the methods and the multiple applications of CCT for a period of at least four weeks. This should provide a basic background in CCT for the practice of adult cardiology or for general radiology. During this cumulative four-week experience, individuals should have been actively involved in CCT interpretation under the direction of a qualified (Level 2- or Level 3-trained) physician-mentor. There should be a mentored interpretative experience of at least 50 cases for all studies in which other cardiovascular imaging methods are also available; correlation with CCT findings and interpretation is strongly encouraged and should be included if possible. As much as possible, studies should consist of procedures outlined in Table 1. Independent performance of CCT is not required for Level 1, and the mentored interpretive experience may include studies from an estab-

lished teaching file or previous CCT cases and also the potential for CD/DVD and on-line training.

LEVEL 2 TRAINING. Level 2 is defined as the minimum recommended training for a physician to independently perform and interpret CCT. This is an extension of Level 1 training and is intended for individuals who wish to practice or be actively involved with CCT performance and interpretation.

COMPETENCE IN NON-CONTRAST CCT. For those physicians only interested in the ability to interpret non-contrast CT studies (the "heart scan"), there are separate requirements for non-contrast Level 2 training (Table 4). The successful candidate will demonstrate competence in analysis and interpretation of cardiac and proximal aorta calcification data. The acquisition, post-processing, and interpretative learning curve for this procedure is rapid, but competence must be defined. A specific requirement for the physician only credentialed to interpret non-contrast CCT studies will be training for a minimum of four weeks (including coursework, scientific meetings and continuing medical education [CME]/on-line training) with 150 cases interpreted, with a minimum of 50, which should be interpreted with a mentor.

COMPETENCE IN CONTRAST CCT. Physicians seeking Level 2 training inclusive of contrast and non-contrast studies will need to interpret 50 non-contrast cases with more time and cases specifically targeting contrast. Training in contrast and non-contrast CT may occur concomitantly.

The minimum requirement for the dual credentialing is eight weeks of cumulative experience in a program actively performing CCT examinations in a clinical environment. In-lab training time is defined as a minimum of 35 h/week. Twenty hours of didactic CME courses specifically targeting CCT can contribute to the total time. The variety of exposure should include as much as possible the list of studies outlined in Table 1.

During this training experience, each candidate should actively participate in CCT study interpretation under the direction of a qualified (preferably Level 3-trained) physician-mentor. Some supervision can be by an expert non-cardiac CT physician for some of the basics of CT/reformatting, workstation, radiation physics, and so forth. The candidate should be involved with the interpretation of at least 150 CCT examinations (with contrast enhance-

Table 4. Requirements for Level 2 and Level 3 Clinical Competence in CCT

	Level 2		Level 3
Initial Experience	● NON-CONTRAST REQUIREMENTS ● Board certification or eligibility, valid medical license, and completion of 4 weeks of training (to include coursework, scientific meetings, and courses/on-line training) ● AND 150 non-contrast CCT examinations (for at least 50 of these cases, the candidate must be physically present, and be involved in interpretation of the case) ● AND completion of 20 h of courses/lectures related to CT in general and/or CCT in particular	● FULL CCT REQUIREMENTS ● Board certification or eligibility, valid medical license, and completion of 8 weeks (cumulative) of training in CCT ● AND 150 contrast CCT examinations. For at least 50 of these cases, the candidate must be physically present, and be involved in the acquisition and interpretation of the case ● AND evaluation of 50 non-contrast studies ● AND completion of 20 h/lectures related to CT in general and/or CCT in particular	● Board certification or eligibility, valid medical license, and completion of 6 months (cumulative) of training in CCT, ● AND 300 contrast CCT examinations. For at least 100 of these cases, the candidate must be physically present, and be involved in the acquisition and interpretation of the case ● AND evaluation of 100 non-contrast studies ● AND completion of 40 h of courses/lectures related to CT in general and/or CCT in particular
Continuing Experience	50 non-contrast CCT exams conducted and interpreted per year	50 contrast CCT exams conducted and interpreted per year	100 contrast CCT exams conducted and interpreted per year
Continuing Education	20 h Category I every 36 months of CCT		40 h Category I every 36 months of CCT

ment). The candidate should be physically present and involved in the acquisition and interpretation of the case in at least 50 studies. Cases should reflect the broad range of anticipated pathology. Didactic studies should include advanced lectures, reading materials, and formal case presentations. These didactic studies should include information on the sensitivity, specificity, accuracy, utility, costs, advantages, and disadvantages of CCT as compared with other cardiovascular imaging modalities. Each physician should receive documented training from a CCT mentor and/or physicist on the basic physics of CT in general and on CCT in particular. Lectures will include discussions of anatomy, contrast administration and kinetics, and the principles of 3D imaging and post-processing. The physician should also receive training in principles of radiation protection, the hazards of radiation exposure to both patients and CT personnel, and appropriate post-procedure patient monitoring. Finally, the physician should be thoroughly acquainted with the many morphologic and pathophysiologic manifestations and artifacts demonstrated on CCT images.

A physician with Level 2 training should demonstrate clear understanding of the various types of CT scanners available for cardiovascular imaging (EBT and MDCT) and understand at a minimum the common issues related to imaging, post-processing, and scan interpretation, including:

- Important patient historical factors (indications and risk factors that might increase the likelihood of adverse reactions to contrast media, if applicable)
- Radiation exposure factors
- CT scan collimation (slice thickness)
- CT scan temporal resolution (scan time per slice)

- Table speed (pitch)
- Field of view
- Window and level view settings
- Algorithms used for reconstruction
- Contrast media
- Post-processing techniques and image manipulation on work stations
- Total radiation dose to the patient

LEVEL 3 TRAINING. Level 3 training represents the highest level of exposure/expertise that would enable an individual to serve as a director of an academic CCT section or director of an independent CCT facility or clinic. This individual would be directly responsible for QC and training of technologists and be a mentor to other physicians seeking such training. The minimum cumulative training period will be six months, to include all of the didactic requirements of Level 2 training as well as participation in CCT study interpretation under the direction of a qualified (Level 3-trained) physician-mentor. In-lab training time is defined as a minimum of 35 h/week. Level 3 candidates should be involved with interpretation of at least 100 non-contrast and 300 contrast CCT examinations. For at least 100 of these cases, the candidate must be physically present and be involved in the acquisition and interpretation of the case. Cases should reflect a broad range of pathology.

In addition to the recommendations for Level 1 and Level 2 training, Level 3 training should include active and ongoing participation in a basic research laboratory, clinical research, or graduate medical teach-

ing. This level also requires documented and continued clinical and educational experiences. Additionally, Level 3 CCT physicians should have appropriate knowledge of alternative imaging methods, including the use and indications for specialized procedures including echocardiography and vascular ultrasound, CMR, and nuclear medicine/positron emission tomography (PET) studies. A summary of the training requirements is given in Table 3.

Competence Considerations Unique to Specific Applications

Non-contrast cardiac CT including coronary artery calcium. Quantification of coronary artery (as well as central and peripheral artery) calcification has been established as a means to estimate atherosclerotic plaque burden. Calcification scores have been shown to provide individualized cardiovascular risk assessment independent of and incremental to conventional cardiovascular risk factors (6–8).

It is to be emphasized, however, that a study done primarily to quantify coronary artery calcification (EBT or MDCT) will require not only separating native coronary calcification from aortic and mitral valvular and pericardial calcification, but also defining the potential for gross cardiac chamber abnormalities as well as potential abnormalities of the pericardium. Proximate calcification and/or enlargement of the ascending aorta and the descending thoracic aorta, included in the heart in the imaging field, should also be recognized if imaged. Tables 3 and 4 discuss the recommended number of cases for proficiency for non-contrast studies (not included in the totals for contrast CT).

Physicians must demonstrate competence in analysis and interpretation of cardiac and proximate aorta calcification data. The learning curve for this procedure is rapid, but competence must be defined. For those physicians interested in full Level 2 competence (to include contrast studies), this non-contrast requirement can be done concomitantly with training for contrast studies. Specific requirements are outlined in Table 4.

Non-invasive coronary CT angiography (CTA). Performance and interpretation of CTA involving the intravenous administration of 60 to 140 ml of iodinated contrast during a prolonged breath-hold is significantly more challenging than coronary calcium assessment. Understanding the anatomy, which is learned most directly from conventional coronary arteriography, is vital to the applicant, and it is expected that all the candidates will have adequate understanding of the coronary distribution and anatomy. The tortuosity of the vessels and limited temporal resolution (MDCT) or spatial resolution (EBT) contributes to this difficulty. However, studies have demonstrated a high sensitivity and specificity in the major epicardial segments for the diagnosis of significant (greater than 50% diameter) obstructive coronary artery disease (CAD) as compared to conventional X-ray coronary angiography using EBT and 16-slice MDCT scanners (9–12,18,19). The successful candidate will demonstrate competence in analysis and interpretation of cardiovascular angiographic data. The post-processing and interpretive learning curve is not rapid, and much of the overall time spent training in CCT should thus be directed at non-invasive angiography training. This is necessary for clinicians to obtain both clinical expertise and to become technically competent in 2D and 3D rendering using a computer workstation. For clinicians to obtain expertise in performance of CTA, a majority of the cases must be directed at the performance of these contrast studies. Cases should reflect the broad range of coronary artery and bypass graft pathology.

CHD evaluation by CCT. Use of CCT is an important resource for the evaluation of known or suspected CHD in children and adults. Echocardiography and CMR are the most commonly used technologies for assessment of CHD. Cardiovascular computed tomography can provide accurate 3D assessment of the heart, offering additional clarity when findings are in question. The ability to assess CHD quickly allows for studies either without any sedation in most cases, or with markedly reduced sedation requirements, which is particularly useful in children. However, CCT requires exposure to ionizing radiation and iodinated contrast. The CCT technique has excellent spatial resolution, and can be utilized to assess the great vessels, including anomalies of the aorta, pulmonary artery, patent ductus arteriosus, pulmonic or tricuspid valve atresia, persistent left superior vena cava, anomalous pulmonary veins, assessment of intracardiac shunts, and the presence of bronchial collaterals.

A properly trained CCT practitioner at Level 2 should be able to determine the appropriate indications for echocardiography, CCT, or CMR in CHD assessment. This is especially true in the assessment of pediatric CHD patients, where the information gained from the CCT examination does not require the risks associated with the general anesthesia or conscious sedation required to perform CMR, but does expose the young patient to ionizing radiation (where radiation exposure may be more clinically important than in adults) and iodinated contrast.

A cardiologist, nuclear medicine specialist, or radiologist with Level 2 or Level 3 CCT competence should be capable of recognizing simple CHD. However, as with echocardiography, few adult cardiology or radiology training programs have a sufficient case load and case mix of complex CHD lesions to ensure an adequate level of training. Although those trained in CCT may be able to recognize the presence of a complex congenital lesion, most CCT programs will be unable to provide enough experience to trainees to develop the special skills necessary to evaluate complex CHD, post-surgical appearance, and post-surgical complications. Practitioners who wish to perform CCT for adult and pediatric CHD patients need special experience. A recommended case load (as part of the total number recommended for competence) for Level 2 is 25 cases; for Level 3 it is 50 cases, with an additional 20 cases annually to maintain competence.

Cardiac function and structure assessment by CCT. Electron beam tomography was initially validated for quantification of LV and RV global and regional systolic and diastolic function—demonstrating similarities to other methods. Lower temporal resolution MDCT data results in lower ejection fraction (EF) estimates than reference methods such as CMR. A thorough knowledge of the utilization of CCT reconstructions of short axis, long axis, and trans-axial imaging is necessary. These imaging planes have been standardized for multiple imaging methods (20).

The use of CCT has been demonstrated to be accurate for the measurement of RV and LV mass, volumes, and EF (5,21), and has been used to quantify calcified plaque on the aortic and mitral valve (22). However, it is inferior to specially sequenced CMR and echocardiography for assessing valvular function and volumes. For clinicians to obtain expertise in performance of CCT functional assessment, a minimum number of cases must be directed at the performance of these contrast studies. As part of the total cases necessary for Level 2 competence, at least 25 cases should be performed and 50 cases interpreted with mentorship. Cases should include assessment of the thoracic aorta.

Nuclear/CT hybrid devices. Hybrid devices are rapidly evolving to incorporate state-of-the-art, high-speed MDCT technology, along with the latest PET and single-photon emission computed tomography (SPECT) detector systems. Dual-modality imaging presents an opportunity to use a single piece of equipment for distinctly different purposes, such as the determination of perfusion, function, and metabolism (PET-SPECT) or coronary calcification and CTA. Therefore, a laboratory may use these hybrid devices for multiple purposes, thereby reducing space requirements compared to installation of two separate systems, and also reducing overall financial cost involved in the purchase of both CT and PET-SPECT cameras. There is the potential for a hybrid system to provide attenuation correction for SPECT, thereby further improving the diagnostic accuracy of more traditional radionuclide techniques.

Furthermore, the combination of SPECT-CT or PET-CT may provide an evaluation of coronary anatomy in the same setting as perfusion imaging. The functional imaging (PET or SPECT) combined with anatomic imaging (CT calcium or CTA) may also offer superior prognostic information, although this has not yet been demonstrated. Current hybrid systems available do not include state-of-the-art MDCT systems. As the CT hardware in hybrid systems becomes more robust, the feasibility of combining CTA, coronary calcium scoring, and SPECT- or PET-gated perfusion imaging will become a reality. Given that hybrid devices or sequential scintigraphic and CCT imaging will result in increased radiation exposure, expertise is needed to know in whom to apply these complementary techniques (23).

To facilitate the availability of new technology in all regions of the country, eventually including rural areas where there is frequently limited access to individuals trained in the latest technologies, consideration should be made to allow "cross-over" training for CT and nuclear medicine technologists. Similarly, "cross-over" may occur for physicians, including cardiology fellows, radiology residents, and nuclear medicine residents as well as for cardiologists, nuclear medicine physicians, and radiologists currently in practice. Each of these specialties incorporates radiation safety into their training programs. However, those trained in nuclear cardiology will require additional training in CCT detector physics and instrumentation, because individuals trained primarily in CCT will require additional time to learn physics and instrumentation of scintillation techniques and to review aspects of radionuclide handling and safety. Otherwise, requirements for the performance and interpretation of radionuclide, PET, and CCT studies should follow previously established guidelines and the recommendations set forth in this document.

PROOF OF TRAINING. Documentation of training can be achieved by means of letters or certificates from the director of a fellowship training program or from individuals who are Level 2 or Level 3 qualified in hospital-based or independent imaging centers or clinics. This documentation should state the dates of training and that the candidate has

Table 5. Documentation and Maintenance of Clinical Competence in CCT

Documentation of Competence	Training Guidelines	Proof of Competence
Training completed after July 1, 2008	Level 2 or Level 3 training as outlined	Letter of certification from training supervisor OR letter attesting to competence from Level 2- or 3-trained physician
Training completed before July 1, 2008	Level 2 training OR interpretation of at least 150 studies (in which 50 where the candidate is physically present, involved in the acquisition and interpretation of the case) and attendance in at least 20 h of devoted CCT classes	
	Level 3 training OR interpretation of at least 300 studies (in which 100 where the candidate is physically present, involved in the acquisition and interpretation of the case) and attendance in at least 40 h of classes devoted to CCT	
Maintenance of competence	Contrast CCT examinations per year be performed and interpreted: Level 2: 50 Level 3: 100	

T5

successfully achieved or surpassed each of the training elements (Levels 1, 2, and 3) (Table 5).

Practicing physicians can achieve appropriate training in CCT without enrolling in a formal CCT training program. However, the same prerequisite medical knowledge, medical training, and goals as outlined in the previous text are required. Directly working with a mentor on an ad-hoc basis is acceptable, but formalized time of cumulative exposure for Levels 1 to 3 competence will be maintained. Currently, a practicing physician seeking this training should have completed an ACGME-approved cardiovascular diseases fellowship, a general radiology residency, or a nuclear medicine residency.

Maintaining expertise in CCT. All individuals are required to provide evidence of continuing expertise in CCT. Individuals who are currently at Level 1 or Level 2 training can advance to Level 2 or Level 3 by pursuing independent advanced studies from either an academic center or an independent center specializing in CCT. No formal mechanisms currently exist regarding assurance of continuing competence. The field of CCT has expanded rapidly, and ongoing procedural and technical improvements are expected.

Maintenance of CCT expertise requires both ongoing continuing education and regular performance and interpretation of clinical and research CCT examinations. Physicians should periodically attend postgraduate courses and workshops that focus on clinical applications of CCT, especially those that emphasize new and evolving techniques and developments. In addition, physicians should seek to compare the quality, completeness, and results of their own examinations with those presented at scientific meetings and in professional publications. Level 2- and 3-trained individuals will be required to document and continue Category I CME in the area. The recommendations for maintaining Level 2 training is a minimum of 20 h of coursework devoted to CCT over a 3-year period of time. The recommendations for maintaining Level 3 training is a minimum of 40 h of coursework devoted to CCT over a 3-year period.

In conjunction with guidelines provided for other cardiovascular imaging modalities, it is recommended that at least 50 CCT examinations annually be performed and interpreted for those at Level 2 and 100 CCT examinations for those at Level 3 (Table 5).

Prior experience to qualify for Levels 2 and 3 clinical competence for CCT. It is expected that a substantial number of practitioners from multiple specialties have been performing these studies for some time before the creation of these guidelines. Thus, these practitioners can qualify for completion of Level 2 or Level 3 training by having achieved the minimum criteria set forth below by July 1, 2008, as well as board certification and completion of an ACGME-approved radiology or nuclear medicine residency or cardiology fellowship, or at least two months of formal training in CCT. Proof of competence can be obtained by a letter of attestation by a Level 3-trained physician who has overread studies, or by letters or certificates from the training supervisor (Table 5).

Level 2 training: substantive activities in CCT over the last 3 years, with documented involvement in the performance and interpretation of at least 150 studies (at least half with contrast enhancement), and at least 20 h of coursework devoted to CCT.

Level 3 training: activities in CCT to include directing a CCT laboratory with documented involvement in the performance and interpretation of at least 300 studies (at least half with contrast enhancement), accrual of at least 40 h of coursework devoted to CCT, and peer recognition to include at least one of the following: 1) faculty lecturer for at least two CME courses on the topic of CCT, 2) fellowship/residency teaching activities, or 3) three or more peer-reviewed publications in the area of CCT.

CMR IMAGING

Overview of CMR

Cardiovascular magnetic resonance is well established in clinical practice for the diagnosis and management of a wide spectrum of cardiovascular disease. Use of CMR represents the specialized application of MR to the cardiovascular system, employing specialized receiver coils, pulse sequences, and gating methods.

A brief overview of the technical aspects of CMR (24) is included to facilitate the understanding of the terms used in the subsequent sections of this report and is not intended to be comprehensive. A CMR scanner has six major components. The magnet, which is usually superconducting, produces the static magnetic field whose strength is measured in Tesla (e.g., 1.5-T or 3-T; 1.5-T is equivalent to 15,000 Gauss, and the Earth's magnetic field is approximately 0.5 Gauss). A stable, homogeneous field is required about the area of interest. Resistive gradient coils within the bore of the magnet produce the gradient fields, and the currents within these coils are driven by the gradient amplifiers. The performance of the gradient system determines the speed of the MR acquisition. A radiofrequency (RF) coil (antenna) is coupled to an RF amplifier to excite the patient's protons with RF pulses, and this (or another more localized surface coil) is coupled to the receiver to measure the resultant signal. A computer is required to control the scanner and generate the images, which are then displayed in static, dynamic (cine) modes. Post-processing tools are extensive and used both for quantitation and for image display.

Like echocardiography, MR is fundamentally safe and does not interfere with the electron shells involved in chemical binding (e.g., DNA) that can be altered by ionizing radiation methods such as X-rays or CT. The phenomenon of MR is restricted to atomic nuclei with unpaired spin and includes hydrogen, carbon, oxygen, sodium, potassium, and fluorine; these latter elements are rarely used for imaging in clinical practice owing to their

low abundance or sensitivity to RF stimulation. Phosphorus is used for clinical CMR spectroscopy, but the majority of clinical CMR imaging involves the hydrogen nucleus, which is abundant in water, fat, and muscle.

In the presence of an external magnetic field (primary magnet), the hydrogen nucleus acts like a small magnet, which can align itself parallel to an external magnetic field, processing about the field in a manner analogous to a spinning top in a gravitational field. The frequency of precession is 63 MHz for field strength of 1.5-T and is in the RF range. Hydrogen nuclei can be excited by radio waves only at this resonant frequency, which has the effect of rotating the net magnetization vector by an amount termed the "flip angle." After this excitation, the net magnetization vector precesses around the direction of the main field, returning to its former position (relaxation) during which energy is transmitted as a radio signal that can be detected by a receiver coil. The return of the net magnetization vector to equilibrium has two components. The vector component parallel to the main field relatively slowly returns to equilibrium by interacting with surrounding molecules, and this is known as "T1 relaxation." Recovery of the vector component transverse to the field is more rapid and is termed "T2 relaxation." The CMR images can be weighted to show the distribution of T1 or T2 (proton density). In order to localize the signals coming from the body, gradient magnetic fields are required that are switched on and off at appropriate times.

An MR pulse sequence is a combination of RF pulses and magnetic gradient field switches controlled by the scanning computer. For CMR, spin echo, gradient echo, steady-state free precession, phase velocity, and inversion recovery, pulse sequences are the most commonly used sequences. Spin echo sequences are routinely used for multi-slice anatomical imaging; rapidly moving blood is typically suppressed/dark while gradient echo and steady-state free precession sequences are used for physiological assessment of cardiac function though cine acquisitions, and rapidly moving blood is typically bright/white. Inversion recovery sequences are typically used in concert with MR contrast agents for infarction/viability imaging, where myocardium is purposefully nulled/black, infarct is bright/white, and blood is an intermediate/gray. Images may be performed with ECG gating/triggering (less preferred is peripheral pulse gating) and with respiratory suppression (breath holding or navigator gating). This reduces image artefacts.

As compared with CCT in which images are acquired in the axial plane and reconstructed in oblique orientations, with CMR, the data are often directly acquired in oblique imaging planes.

Some specialized sequences exist that have particular application for the cardiovascular system. For example, magnetic resonance angiography (MRA) is usually performed with 3D coverage of the vessel during a short breath-hold and after intravenous injection of a gadolinium-based contrast agent. Gadolinium has seven unpaired elec-

trons in its outer shell, and it hastens T1 relaxation, thereby increasing signal in the area of interest. Gadolinium alone is cytotoxic, but not if chelated with diethylenetriamine pentaacetic acid (DTPA). Gadolinium-chelate has similar pharmacokinetic properties to iodinated X-ray contrast but with minimal nephrotoxicity and anaphylaxis risk. Several FDA-approved gadolinium-DTPA preparations have been used for over a decade, and the safety profile is far more favorable than for iodinated contrast; however, it is not presently FDA approved for use in the heart.

Myocardial perfusion CMR follows the effect of a first pass of a bolus of intravenous gadolinium through multiple planes of the myocardium. Coronary MRI requires high spatial resolution. To characterize regional myocardial contraction, non-invasive "tagging" of the myocardium with a grid at end-diastole and subsequent cine acquisition to observe tag deformation allows for calculation of myocardial strain. Finally, velocity mapping is a sequence used to measure velocity and flow in blood vessels or within the heart (somewhat analogous to Doppler echocardiography) in which each pixel in the image displays the signal phase, which is encoded. Flow is calculated from the product of mean velocity, and the vessel area is measured throughout the cardiac cycle.

CMR safety. The CMR method is very safe for the cardiovascular patient, and no short- or long-term ill-effects have been demonstrated at current field strengths (less than 3-T). Claustrophobia is problematic in over 2% of patients (25). A very important safety issue for CMR is the prevention of ferromagnetic objects from entering the scanner area as these will become projectiles (attractive force accelerates as they approach the scanner). Common practice is to specifically check and verify that each medical device present in patients is MR safe. Metallic implants such as hip prostheses, mechanical heart valves, coronary stents, and sternal sutures present no hazard since the materials used are not ferromagnetic (although a local image artifact will result). Care is required in patients with cerebrovascular clips; however, specialist advice is needed for such patients. Patients with most pacemakers (implanted cardioverter-defibrillators [ICDs]) retained permanent transvenous pacemaker leads, and some other electronic implants (infusion or monitoring devices) should not be scanned, although some reports of success do exist in non–pacemaker-dependent patients who are carefully monitored during the procedure and have device interrogation before and after CMR (25,26).

Biological and clinical effects of CMR exposure. The biological effects of CMR exposure will be considered under the headings of attractive forces, heating, and stimulation. The presence of a large magnetic field will impart forces on all ferromagnetic materials. The RF field, which is used for excitation, can induce heating of tissue and implanted devices (particularly pacemaker leads or related devices). The RF power deposition (also known as specific absorption rate [SAR]) is actively monitored in accordance with FDA

standards. Finally, it is possible to stimulate sensitive tissues such as peripheral nerves owing to the rapidly changing gradient magnetic fields used to generate images. Myocardial stimulation has not been described with current hardware. Clinical CMR hardware has reached the limit for stimulation of peripheral nerves, and clinical imaging approaches are designed to avoid such stimulation.

CMR Laboratory Requirements

General considerations. Defining the specific requirements for a valid CMR laboratory is beyond the scope of this document, which is concerned about accreditation for physicians. However, some general aspects of the appropriate MRI environment can be considered.

A continuous QC program must be established for all CMR units with the assistance of a qualified medical physicist and a Level 3-trained physician coordinator (levels of training are described in the following text). The scanners must be staffed by CMR technologists with appropriate background and/or training in CMR. A qualified medical physicist should periodically check equipment calibration (signal-to-noise ratio levels and image quality factors). A safety program should be active, and should include controlled access to the CMR equipment and training programs in CMR safety for all personnel. In addition, patient safety should be assured through a well documented screening and evaluation program for implanted devices, administered by the CMR medical director with ongoing documentation, training, and evaluation by nursing personnel performing screening procedures.

MONITORING AND ANCILLARY HARDWARE. The scanner should have appropriate monitoring hardware for ECG rhythm, non-invasive blood pressure measurement, and pulse oximetry determination during CMR procedures. If stress cardiac studies are performed using high-dose dobutamine, real-time image reconstruction and display or an on-the-fly display of cine images allowing rapid assessment of global and regional function to monitor the signs of myocardial ischemia should be available. For stress myocardial perfusion studies, CMR-compatible infusion pumps are necessary for the infusion of adenosine and possibly dipyridamole (if administered in the CMR imaging room), and an appropriate patient monitoring system is required. Nursing and physician personnel should be trained in the administration, monitoring, and side effects of the pharmacologic stress agents used by the center. Some patients benefit from supplemental oxygen using low flow nasal prongs during the study. A CMR-compatible power injector is necessary for gadolinium contrast-enhanced myocardial perfusion studies and preferred for contrast-enhanced MRA procedures. The CMR personnel should be trained in the recognition and management of reactions to CMR contrast media, to sedatives, and to other drugs administered as part of the CMR procedure.

POST-PROCESSING AND DATA ANALYSIS. The CMR laboratory should have the capabilities for post-processing CMR data, including LV and RV function determination, analysis of myocardial perfusion images, cine review of myocardial function studies, and a full 3D processing and display system for MRA studies.

Clinical indications for CMR. A comprehensive review of the clinical indications for CMR is beyond the scope of this report. Interested readers are referred elsewhere (24). A brief overview of broad clinical indications is presented in the text. These are intended to serve as a general guide for CMR training to include a broad spectrum of pathologic cases inclusive of these indications.

CMR in ischemic heart disease: regional and global function, perfusion, viability, and coronary angiography. Because of its inherent 3D capabilities, high spatial and temporal resolution, and high contrast resolution, CMR is widely recognized as the "reference standard" for the quantitative assessment of RV and LV volumes, EF, mass, and regional ventricular function. The CMR tagging techniques are unique among all modalities for determination of myocardial strain.

Use of CMR is a highly accurate and reproducible noninvasive method for measuring EF, LV volumes, and LV mass, and also to assess LV structure and function (27). Usually bright blood gradient echo sequences, with 15 s of breath-hold, are used to cover the entire LV with short-axis views from the mitral plane. Also, the sample size needed to detect LV parameter changes in a clinical trial is far less than other imaging modalities; this markedly reduces the time and cost of patient care and pharmaceutical trials (28). Regional LV function can be measured as in echocardiography using visual assessment of endocardial motion but more frequently systolic wall thickening. Beek et al. (29) demonstrated the relationship between functional recovery and transmural necrosis using visual assessment of systolic endocardial motion and wall thickening. Previous studies used similar methods to document similar relationships both in the acute (30) and chronic (31) myocardial infarction (MI) settings.

Although physical exercise within the confines of the magnet is technically difficult, there have been extensive studies of pharmacologic CMR stress. Both beta-agonist (e.g., dobutamine) stress examinations for inducible wall motion abnormalities and stress vasodilator (e.g., adenosine) in combination with first passage of a small dose of gadolinium-DTPA for assessment of myocardial perfusion have been shown to be accurate for detecting CAD. As the ST-segment is distorted/uninterpretable in the CMR environment, close clinical patient monitoring is required. Recent progress in the diagnosis and treatment of CAD has intensified the need to differentiate viable from non-viable myocardium with accuracy and high spatial resolution. Use of CMR has been demonstrated to be an effective technique to assess myocardial viability (32). The development of inversion recovery gradient echo imaging techniques pro-

vided the ability to temporally distinguish perfusion abnormalities created by microvascular obstruction from myocardial hyper-enhancement secondary to myocardial necrosis (33,34). The prognostic value of contrast-enhanced MRI in patients with acute MI is well established (35). However, recent work on the specific utilization of this technique for the purpose of predicting local functional recovery after acute MI (29,30,36) has further expanded its potential utilization.

Acute and chronic MI can be detected with high accuracy and sensitivity using delayed enhancement CMR with an inversion recovery sequence. The inversion time is chosen to null/suppress normal myocardial signal with resultant bright signal in areas of fibrosis where gadolinium-DTPA will concentrate. Both the delayed-enhancement technique and low-dose dobutamine have been shown to have great utility for the assessment of myocardial viability among patients being considered for revascularization.

The application of CMR coronary angiography for the assessment of the course of anomalous coronary arteries and coronary artery bypass graft (CABG) patency is well established. The use of CMR coronary angiography for native vessel integrity has been shown to be feasible, especially for the proximal and mid-portions of the major coronary vessels, but it remains technically demanding in the branch vessels owing to their smaller size, tortuosity, complex 3D anatomy, and cardiac and respiratory motion artifacts (9,37,38). Using current 3D acquisitions and optimized sequences, good results for the exclusion of multivessel disease have been shown. Currently available intracoronary stents appear to be CMR "safe," but they can result in a localized signal artifact. Each new stent material requires evaluation for CMR safety.

CMR in non-ischemic cardiomyopathies. The non-ischemic cardiomyopathies include a variety of disorders in which the primary pathology directly involves the myocardium. Use of CMR is proving increasingly valuable in the identification and management of these conditions—including delineation of hypertrophy and fibrosis for hypertrophic cardiomyopathy, iron deposition in hemochromatosis, fatty or fibrous replacement in arrhythmogenic RV dysplasia, and myocarditis. The CMR technique is very effective for monitoring the severity of LV hypertrophy in hypertrophic cardiomyopathy and for the monitoring of ventricular volumes in dilated cardiomyopathy.

CMR in pericardial disease. Both CMR and CCT accurately define pericardial thickening and circumferential and loculated pericardial effusions. Although CCT has the advantage of pericardial calcium identification, CMR has the advantage of being able to depict and quantify the functional abnormalities, which may be associated with pericardial disease, and for demonstrating physiologic signs of ventricular interdependence with calcified pericardium.

CMR in valvular heart disease. Although echocardiography remains the preferred imaging modality for the routine determination of valve morphology and flow abnormalities,

CMR is starting to be utilized in the care of patients with regurgitant lesions. Valvular regurgitation is usually recognized as a signal void on cine CMR. A quantitative assessment of single-valve lesions can be obtained by calculating the regurgitant volume from the difference of RV and LV stroke volumes or the use of phase-velocity data from the ascending aorta and main pulmonary artery to calculate regurgitant volumes. Also, CMR has been shown to provide data for the estimation of gradients and areas in mitral and aortic stenosis.

CMR for CHD patients. The CMR technique is a very important resource for the evaluation of known or suspected CHD in children and adults. Assessment of CHD was one of the first clinical indications for the performance of CMR, and its utility in the assessment of CHD has grown with the development of CMR technology. Although echocardiography is often the initial imaging modality used in the assessment of CHD, CMR can provide accurate 3D assessment of cardiac structure and blood flow, especially valuable for patients with suboptimal acoustic windows. In addition, especially in adults, CMR may be a better method for assessing the great vessels and complex CHD.

A graduate of an ACGME-approved fellowship in cardiovascular diseases or residency in nuclear medicine or radiology should be able to determine the appropriate indications for CMR in CHD assessment, knowing whether to refer to CMR or another imaging modality. This is especially true in the assessment of pediatric CHD patients for whom echocardiography is not sufficient. The benefits of CMR in children must be balanced against the occasional requirement of deep sedation or general anesthesia.

Acquired Vascular Disease

Use of CMR is particularly helpful for vascular lumen imaging because of its ability to generate projectional MRA. These can be generated either without contrast (time-of-flight technique) or contrast-enhanced with intravenous gadolinium. Consequently, it is well suited for use in patients with contraindications to X-ray contrast due to allergy or renal insufficiency. In addition to angiography, the wide variety of soft tissue contrast available on CMR (proton density, T1, T2, lipid-saturation) can be applied to vascular imaging to assess features of vessel wall such as hematoma/thrombus, inflammation, and atherosclerotic plaque. In addition to morphologic imaging of blood vessels, phase-contrast imaging (velocity mapping) can be used to quantify blood flow. Vascular CMR is beyond the scope of this document.

Technical aspects of the CMR examination. A CMR physician must be skilled in all technical aspects of performance of the CMR examination. This includes a thorough knowledge of available pulse sequences and the indications for their use. Sequences the CMR physician must be familiar with include:

1. Spin echo and cine sequences (segmented K-space gradient echo, steady-state free precession) for assessment of function and anatomy.
2. Fast, spin echo and half Fourier spin echo, black-blood sequences, for assessment of anatomy.
3. Phase contrast sequences for calculation of blood flow, shunt ratios, valve regurgitant fraction, and gradient assessment.
4. Contrast-enhanced MRA techniques for assessment of great vessels.
5. Delayed hyperenhancement imaging, dobutamine wall motion stress, and vasodilator stress perfusion imaging, tagging, and post-processing.

The CMR physicians should be familiar with the various types of coils available for cardiac imaging, and how they can be used in their patient populations (pediatric vs. adult), as well as K-space acquisition (symmetric, asymmetric, centric), and methods of ECG gating. The latter is especially important as the placement of leads may vary from the norm in patients with CHD.

In addition, the CMR physician must be familiar with the use of gadolinium-based MRI contrast agents including their indications and adverse reactions. The CMR physician should also be well versed in the treatment of the contrast reactions, including hives, wheezing, hemodynamic responses, and anaphylaxis, although rare. A thorough knowledge of appropriate doses of treatment medication, depending upon one's patient population (adult vs. pediatric), is crucial.

Minimal Knowledge and Skills Required for CMR Expertise

The CMR training recommendations have been published by three societies: ACR, ACC, and SCMR (1–3). The ACC and SCMR recommendations directly address the goals of general training and are outlined by Task Force 12 of the COCATS-2 (1). The purpose of general training is to provide the practicing physician or resident/fellow in training with the working knowledge of CMR methods in order to facilitate patient care and management. It is recommended that all trainees in cardiovascular diseases and radiology have CMR exposure for at least one month or its equivalent when integrated with other activities during the practice of cardiovascular medicine or radiology. From a practical perspective, many clinicians in practice may not have access to CMR-enabled equipment or qualified (Level 2 or Level 3) mentors. It is recommended that candidates take this opportunity to supplement their education through lecture material, didactic reading, and journal or electronic media review. The committee reviewed guidelines set forth for other imaging modalities (for example, echocardiography, COCATS2) (1), and the caseload required reflects the need for increased time to learn the interpretation skills necessary but decreased physical training (for example, transducer time).

During general training in cardiovascular diseases and radiology, physicians should obtain a basic understanding of MR physics, which include the principles of image construction, T1 and T2 relaxation, measurements of blood flow, determinations of anatomy, image contrast, function, viability, myocardial perfusion, CMR contrast agents, and metabolism. Review of the indications and side effects of CMR contrast materials should occur along with exposure to proper receiver coil selection, methods of cardiac gating and triggering (e.g., ECG and peripheral pulse), respiratory motion suppression (e.g., breath-hold and navigators), and sources of image artifacts (e.g., motion, arrhythmias, and metal objects). Also, understanding of the contraindications to CMR, and the safety of devices within the MR environment, should be reviewed. Recognition of the sensitivity, specificity, diagnostic accuracy, costs, indications, and prognostic capability is to be accomplished in general training as this information is important for understanding the proper clinical use of CMR.

All cardiovascular and radiology trainees should actively participate in CMR interpretation under the supervision of a qualified (preferably Level 3) physician-mentor. Some supervision by an expert non-CMR physician can suffice for some of the basics of CMR, including workstation exposure, tomographic imaging training, and so on. Correlative sessions should be performed with other imaging modalities (echocardiography, nuclear cardiology, CCT, and invasive X-ray) as well as historical, physical examination, and laboratory and hemodynamic data. The types of procedures reviewed should include those directed toward assessments of the cardiovascular system, incorporating measures of structure, tissue characterization, function, myocardial perfusion, delayed hyperenhancement imaging, blood flow, plaque characterization, and angiography of the thoracic aorta and bypass grafts incorporating vessels within these territories. Procedures involving the use of intravenous contrast material should be included. A minimum of 50 such cases should be performed. This might include review and interpretation from an established teaching file of previous CMR cases or those administered from electronic media. Hands-on experience is not necessary for general training.

Formal training to achieve competence in CMR. The recommendations for all levels of training below represent a cumulative experience, and it is expected that for many practicing clinicians the training will not be continuous. Time spent at didactic CME courses that specifically target CMR can also contribute to the total time. Due to the advancement in the sophistication and widespread availability of electronic training medias, the committee felt that some training can now be obtained outside the laboratory setting. However, for all Level 2 and Level 3 requirements, minimum time in a laboratory supervised by a Level 2 or Level 3 CMR physician is half of the time listed, with the other half garnered by supervised time, CME and other courses, case studies, CD/DVD training, and time spent at

major medical meetings devoted to performance of CMR, to cite just a few examples. The caseload recommendations might include studies from an established teaching file, previous CMR cases or electronic/on-line CME. Although this is less "in-laboratory" time than that listed in both the COCATS and the SCMR published recommendations (1,3), these other documents also include training time for peripheral imaging.

For all levels of competence, it is expected that the candidate will attend lectures on the basic concepts of CMR and include parallel self-study reading material. A basic understanding of CMR should be achieved, including the physics of MRI in general and of CMR in particular. The content should include the basics of CMR scan performance, CMR safety issues, and basics of CMR interpretation as compared with other cardiovascular imaging modalities including echocardiography, nuclear medicine, CCT, and invasive cardiac X-ray angiography.

LEVEL 1 TRAINING. Level 1 is defined as the minimal introductory training for familiarity with CMR, but this exposure is not sufficient for independent interpretation of CMR images. The individual should have intensive exposure to the methods and the multiple applications of CMR for a period of at least four weeks. This should provide a basic background in CMR for the practice of adult cardiology or for general radiology.

During this cumulative four-week experience, individuals should have been actively involved in CMR interpretation under the direction of a qualified (Level 2- or Level 3-trained) physician-mentor. For all studies in which other cardiovascular imaging methods are also available, correlation with CMR findings and interpretation should be included. Studies should consist as much as possible within the range of pathologies outlined in the previous text. Independent performance of CMR is not required for Level 1 training, and the mentored interpretive experience of 50 cases may include studies from an established teaching file or previous CMR cases.

LEVEL 2 TRAINING. Level 2 training is defined as the minimum recommended instruction for a physician to independently perform and interpret CMR. This is an extension of Level 1 training and is intended for individuals who wish to practice or actively be involved with CMR performance and interpretation. The minimum requirement is three months of cumulative experience, with a minimum time in a program supervised by a Level 2 or Level 3 CMR physician over a period of six weeks, with the other six weeks garnered by supervised time, CME and other courses, case studies, CD/DVD training, time spent at major medical meetings devoted to performance of CMR, and so forth. In-lab training time is defined as a minimum of 35 h/week.

Didactic instruction as well as tested self-study should be administered in MR physics, MR applications and indications, and clinical interpretation. Didactic studies should

consist of more advanced lectures and reading materials as well as formal case presentations and should include the following:

1. Physics—trainees should receive didactic lectures from a CMR-trained physician and/or physicist on the basic physics of MR. Topics should include:
   a. Image formation, including K-space (implications of symmetric and asymmetric, spiral, radial K-space sampling) gradient echo imaging, spin echo imaging, echo planar imaging, fast spin echo imaging, and 2D and 3D imaging.
   b. Physics implicating patient safety, including energy deposition, specific absorption rate (SAR) limits and possible neurological effects, and heating and motion of metallic implants.
   c. Specialized imaging sequences, including flow and motion, phase imaging, time-of-flight, respiratory gating, contrast agents, and MR tagging.
   d. Hardware components, including basic elements of gradient coil design, receiver coils, and digital sampling.
2. Applications and Indications—didactic activities should include discussion of the sensitivity, specificity, accuracy, utility, costs, and disadvantages of all CMR techniques and applications. The following techniques should be covered in the didactic program:
   a. ECG and peripheral pulse gating and triggering, including timing of image acquisition within the R-R interval, motion artifacts and their effects upon image interpretation, velocity calculation, and other physiological quantifications.
   b. Respiratory motion suppression, including its uses and effects upon image interpretation.
   c. Stress pharmacologic agents and their application to CMR, including adequate monitoring under CMR performance and reversal of stress conditions.
   d. Imaging of structure and tissue characterization (T1, T2, spin echo imaging), tissue (inversion recovery, saturation recovery methods) and fat suppression.
   e. Imaging of ventricular function (cine and tagged cine MRI).
   f. Flow imaging (velocity-encoded techniques).
   g. First-pass perfusion and delayed contrast-enhancement imaging (gadolinium-enhanced techniques).
   h. Image processing for creating of angiographic images, velocity and flow calculation, function parameters (EF, myocardial mass, and so on)
   i. Clinical instruction—a Level 3 CMR mentor must be able to provide the trainee with instruction in adequate image interpretation. Topics addressed should include those listed above.

During this training experience, each candidate should actively participate in CMR study interpretation under the direction of a qualified (preferably Level 3-trained) physician-mentor. The candidate should be involved with

interpretation of at least 150 CMR examinations in which at least 50 necessitated the candidate being physically present and involved in the acquisition and interpretation of the case. Cases should reflect the broad range of anticipated pathology.

Each physician should receive didactic lectures from a CMR mentor and/or physicist on the basic physics of MR in general and on CMR in particular. Lectures should include discussions of anatomy, contrast administration and kinetics, CMR safety, and the principles of 3D imaging and post-processing. Finally, the physician should be thoroughly acquainted with the many morphologic and pathophysiologic manifestations and artifacts demonstrated on CMR images.

Table 6 provides a summary of the overall requirements for CMR Level 1 and Level 2 training.

LEVEL 3 TRAINING. Level 3 training represents the highest level of exposure/expertise that would enable an individual to serve as director of an academic CMR section or director of an independent CMR facility. This individual would be directly responsible for QC and training of technologists and to be a mentor to other physicians seeking such training. The minimum cumulative training period should be 12 months and include all the didactic requirements of Level 2 training as well as participation in CMR study interpretation under the direction of a qualified (Level 3-trained) physician-mentor. In-lab training time is defined as a minimum of 35 h/week. The candidate should be involved with interpretation of at least 300 CMR examinations in which at least 100 involved the candidate as the primary operator and interpreter. Cases should reflect the broad range of pathology expected in a CMR practice.

In addition to the recommendations for Level 1 and Level 2 training, Level 3 training should include active participation in an ongoing laboratory, clinical research, or teaching. This also requires continued and documented clinical and educational experiences. These requirements are listed in Table 7. Cooperation between radiology and cardiovascular disease colleagues is encouraged. Additionally, supervising CMR physicians should have appropriate knowledge of alternative imaging methods, including the use and indications for specialized procedures encompassing echocardiography and vascular ultrasound, CCT, and nuclear medicine/PET studies.

Special training in CHD requirements. A physician with Level 2 or Level 3 training in CMR should be capable of recognizing simple CHD. However, as with echocardiography (39), few adult cardiology training programs have a sufficient case load and case mix of complex lesions to ensure an adequate level of training. Although those trained in CMR may be able to recognize the presence of a complex congenital lesion, many MRI programs are unable to provide enough experience to trainees to develop the special skills necessary to evaluate complex CHD, post-surgical appearance, and post-surgical complications. Competence in performing and/or interpreting CMR in pediatric and adult patients with complex CHD requires the basic knowledge of MRI physics, instrumentation, anatomy, physiology, and pathology for CMR interpretation. Practitioners who wish to perform CMR in patient populations with adult and pediatric CHD need special experience. A recommended case load (as part of the total number recommended for competence) for Level 2 is 25 cases, and Level 3 is 50 cases, with additional 20 cases annually to maintain competence. Of note, case mix is an important aspect of the training experience.

The CMR physician must also be well trained in the following:

For the pediatric CHD patient:
1. Cardiac structure and physiology during growth and development from infancy to adulthood.
2. Spectrum of acquired heart disease in the pediatric age group.
3. Spectrum of surgical palliation and surgical repair of CHD and its manifestations on CMR.
4. Spectrum of catheter-based interventions for CHD and its manifestations in the CMR examination.
5. Indications for performance of CMR in the pediatric patient, including the risks of general anesthesia and conscious sedation in relationship to the benefit of diagnostic information obtained for a given purpose.

For the adult CHD patient:
1. Anatomic and physiologic spectrum of CHD and its manifestations in the adult.
2. Spectrum of surgical palliation and repair for CHD and its manifestations on CMR.
3. Spectrum of catheter-based interventions for CHD and its manifestations in the adult CMR.
4. Sequelae of surgical palliation and repair and catheter-based interventions and their presence and manifestation in CMR.

Table 6. Requirements for CMR Study Performance and Interpretation to Achieve Level 1, 2, and 3 Training

	Cumulative Duration of Training	Minimum Total Number of Mentored Examinations Performed	Minimum Number of Mentored Examinations Interpreted
Level 1	1 month	—	50†
Level 2	3 months*	50	150†
Level 3	1 year*	100	300†

*This represents cumulative time spent interpreting, performing, and learning about CMR, and need not be a consecutive block of time, but at least 50% of the time should represent supervised laboratory experience. This can include time spent at educational courses on the topic. Training time is defined as a minimum of 35 h/week. †The caseload recommendations may include studies from an established teaching file, previous CMR cases, and electronic/on-line CME.

Table 7. Requirements for Level 2 and Level 3 CMR Training Clinical Competence

	Level 2	Level 3
Initial experience	• Board certification or eligibility, valid medical license, and completion of a 3-month (cumulative) specialty residency or fellowship in CMR, AND • 150 CMR examinations in which 50 where the candidate is physically present, involved in the acquisition and interpretation of the case, AND • Completion of 30 h of courses related to MR in general and/or CMR in particular	• Board certification or eligibility, valid medical license, and completion of a 12-month (cumulative) specialty residency or fellowship in CMR, AND • 300 CMR examinations in which 100 where the candidate is physically present, involved in the acquisition and interpretation of the case, AND • Completion of 60 h of courses related to MR in general and/or CMR in particular
Continuing education	30 h of coursework every 36 months in CMR	60 h of coursework every 36 months in CMR

5. Impact of acquired heart disease on the physiology of the underlying congenital lesion.

PROOF OF TRAINING. Documentation of training can be achieved by means of letters or certificates from the director of a fellowship training program or from individuals who are Level 2 or Level 3 qualified in hospital-based or independent imaging centers or clinics (Table 8). This documentation should state the dates of training and that the candidate had successfully achieved or surpassed each of the training elements (Levels 1, 2, and 3).

Practicing physicians can achieve appropriate training in CMR without enrolling in a full-time formal training program. However, the same prerequisite medical knowledge, medical training, and goals as previously outlined are required. Directly working with a mentor on an ad-hoc basis is acceptable, but formalized time of cumulative exposure for Levels 1 through 3 competence will be maintained. Currently, a practicing physician seeking this training should have completed an ACGME-approved cardiovascular diseases fellowship, nuclear medicine or general radiology residency, and hold a valid license to practice.

Maintaining CMR expertise. Individuals, even after a formal fellowship training period, are required to continue maintenance of expertise in CMR. Those who are currently at Level 1 or Level 2 training can advance to the next level by pursuing independent advanced studies either from an academic center or an independent center specializing in CMR. No formal mechanisms currently exist regarding assurance of maintaining competence. The field of CMR has expanded rapidly, and ongoing procedural and technical improvements are expected.

Maintenance of CMR expertise requires both ongoing continuing education and regular performance and interpretation of clinical and research CMR examinations. Physicians should periodically attend postgraduate courses and workshops that focus on clinical applications of CMR, especially those that emphasize new and evolving techniques and developments. In addition, physicians should seek to compare the quality, completeness, and results of their own examinations with those presented at scientific meetings and in professional publications. Level 2- and Level 3-trained individuals will be required to document category I CME in the area. Recommended CME for maintaining Level 2 training is 30 h devoted to CMR over a 3-year period. Recommended CME for maintaining Level 3 training is 60 h devoted to CMR over a 3-year period. It is also recommended that at least 50 CMR examinations each year be performed and interpreted for those at Level 2 and at least 100 CMR examinations for those maintaining Level 3 training.

Prior experience to qualify for Levels 2 and 3 training for CMR. It is expected that a substantial number of practitioners from radiology, cardiology, and nuclear medicine have been performing CMR studies for some time prior to the creation of these guidelines. These practitioners can qualify for completion of Level 2 or Level 3 training by having achieved the minimum criteria set forth below by July 1, 2008, as well as board certification and completion of an ACGME-approved radiology residency or cardiology fellowship, or at least six months' formal training in CMR.

Level 2 training: there should be substantive activities in

Table 8. Documentation and Maintenance of Clinical Competence in CMR

Documentation of Competence	Training Guidelines	Proof of Competence
Training completed after July 1, 2008	Level 2 training	Letter or certification from training supervisor OR letter attesting to competence from Level 2- or 3-trained physician
Training completed before July 1, 2008	Level 2 training OR interpretation of at least 150 studies (in which 50 where the candidate is physically present, involved in the acquisition and interpretation of the case) and attendance in at least 30 h of devoted CME Level 3 training OR interpretation of at least 300 studies (in which 100 where the candidate is physically present, involved in the acquisition and interpretation of the case) and attendance in at least 60 h of devoted CME, and acknowledged teacher of CMR	
Maintenance of competence	Performance and/or interpretation of 50 (Level 2) or 100 (Level 3) cases/yr	

CMR over the last three years, with documented involvement in the performance and interpretation of the last three years.

Level 3 training: activities in CMR should include running a CMR laboratory, with documented involvement in the performance and interpretation of at least 300 CMR studies; attendance in at least 60 h of CME devoted to CMR, and being an acknowledged CMR instructor as a faculty member teaching CME courses on the topic; or CMR teaching activities with at least three published studies in the area of CMR. Proof of competence can be obtained by a letter of attestation by a Level 3 physician who has overread the studies or a letter or certificate from one's training supervisor (Table 8).

STAFF

American College of Cardiology
Christine W. McEntee, Chief Executive Officer
Lisa Bradfield, Senior Specialist, Clinical Policy & Documents
American Heart Association
M. Cass Wheeler, Chief Executive Officer
Kathryn A. Taubert, PhD, FAHA, Senior Science Advisor

REFERENCES

1. Pohost GM, Kim RJ, Kramer CM, Reichek N. Task Force 12: Training in Cardiovascular Magnetic Resonance. Available at: http://www.acc.org/clinical/training/COCATS2.pdf. Last update 2002.
2. Weinreb JC, Larson PA, Woodard PK, et al. American College of Radiology Clinical Practice Statement on noninvasive imaging. Radiology 2005;235:723–7.
3. Society for Cardiovascular Magnetic Resonance (SCMR) Clinical Practice Committee. Guidelines for credentialing in cardiovascular magnetic resonance (CMR). J Cardiovasc Magn Reson 2000;2:233–4.
4. Bell MR, Lerman LO, Rumberger JA. Validation of minimally invasive measurement of myocardial perfusion using electron beam computed tomography and application in human volunteers. Heart 1999;81:628–35.
5. Rumberger JA. Use of ultrafast (cine) X-ray computed tomography in cardiac and cardiovascular imaging. In: Giuliani ER, editor. Mayo Clinic Practice of Cardiology. St. Louis, MO: Mosby, 1996:303–24.
6. Arad Y, Spadaro LA, Goodman K, Newstein D, Guerci AD. Prediction of coronary events with electron beam computed tomography. J Am Coll Cardiol 2000;36:1253–60.
7. Kondos GT, Hoff JA, Sevrukov A, et al. Electron-beam tomography coronary artery calcium and cardiac events: a 37-month follow-up of 5,635 initially asymptomatic low- to intermediate-risk adults. Circulation 2003;107:2571–6.
8. Shaw LJ, Raggi P, Schisterman E, Berman DS, Callister TQ. Prognostic value of cardiac risk factors and coronary artery calcium screening for all-cause mortality. Radiology 2003;228:826–33.
9. Budoff MJ, Achenbach S, Duerinckx A. Clinical utility of computed tomography and magnetic resonance techniques for noninvasive coronary angiography. J Am Coll Cardiol 2003;42:1867–78.
10. Lu B, Shavelle DM, Mao S, et al. Improved accuracy of noninvasive electron beam coronary angiography. Invest Radiol 2004;39:73–9.
11. Ropers D, Baum U, Pohle K, et al. Detection of coronary artery stenoses with thin-slice multi-detector row spiral computed tomography and multiplanar reconstruction. Circulation 2003;107:664–6.
12. Hoffmann U, Moselewski F, Cury RC, et al. Predictive value of 16-slice multidetector spiral computed tomography to detect significant obstructive coronary artery disease in patients at high risk for coronary artery disease: patient-versus segment-based analysis. Circulation 2004;110:2638–43.
13. Leta R, Carreras F, Alomar X, et al. [Non-invasive coronary angiography with 16 multidetector-row spiral computed tomography: a comparative study with invasive coronary angiography]. Rev Esp Cardiol 2004; 7:217–24.
14. Nasir K, Budoff MJ, Post WS, et al. Electron beam CT versus helical CT scans for assessing coronary calcification: current utility and future directions. Am Heart J 2003;146:969–77.
15. Hunold P, Vogt FM, Schmermund A, et al. Radiation exposure during cardiac CT: effective doses at multi-detector row CT and electron-beam CT. Radiology 2003;226:145–52.
16. Morin RL, Gerber TC, McCollough CH. Radiation dose in computed tomography of the heart. Circulation 2003;107:917–22.
17. Jakobs TF, Becker CR, Ohnesorge B, et al. Multislice helical CT of the heart with retrospective ECG gating: reduction of radiation exposure by ECG-controlled tube current modulation. Eur Radiol 2002;12:1081–6.
18. Kuettner A, Beck T, Drosch T, et al. Diagnostic accuracy of noninvasive coronary imaging using 16-detector slice spiral computed tomography with 188 ms temporal resolution. J Am Coll Cardiol 2005;45:123–7.
19. Mollet NR, Cademartiri F, Krestin GP, et al. Improved diagnostic accuracy with 16-row multi-slice computed tomography coronary angiography. J Am Coll Cardiol 2005;45:128–32.
20. 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.
21. Baik HK, Budoff MJ, Lane KL, Bakhsheshi H, Brundage BH. Accurate measures of left ventricular ejection fraction using electron beam tomography: a comparison with radionuclide angiography, and cine angiography. Int J Card Imaging 2000;16:391–8.
22. Shavelle DM, Takasu J, Budoff MJ, Mao S, Zhao XQ, O'Brien KD. HMG CoA reductase inhibitor (statin) and aortic valve calcium. Lancet 2002;359:1125–6.
23. Benatar NA, Cronin BF, O'Doherty MJ. Radiation dose rates from patients undergoing PET: implications for technologists and waiting areas. Eur J Nucl Med 2000;27:583–9.
24. Pennell DJ, Sechtem UP, Higgins CB, et al. Clinical indications for cardiovascular magnetic resonance (CMR): Consensus Panel report. Eur Heart J 2004;25:1940–65.
25. Kuijpers D, Janssen CH, van Dijkman PR, Oudkerk M. Dobutamine stress MRI. Part I. Safety and feasibility of dobutamine cardiovascular magnetic resonance in patients suspected of myocardial ischemia. Eur Radiol 2004;14:1823–8.
26. Martin ET, Coman JA, Shellock FG, Pulling CC, Fair R, Jenkins K. Magnetic resonance imaging and cardiac pacemaker safety at 1.5-Tesla. J Am Coll Cardiol 2004;43:1315–24.
27. Task Force of the European Society of Cardiology, in collaboration with the Association of European Paediatric Cardiologists. The clinical role of magnetic resonance in cardiovascular disease. Eur Heart J 1998;19:19–39.
28. Semelka RC, Tomei E, Wagner S, et al. Normal left ventricular dimensions and function: interstudy reproducibility of measurements with cine MR imaging. Radiology 1990;174:763–8.
29. Beek AM, Kuhl HP, Bondarenko O, et al. Delayed contrast-enhanced magnetic resonance imaging for the prediction of regional functional improvement after acute myocardial infarction. J Am Coll Cardiol 2003;42:895–901.
30. Choi KM, Kim RJ, Gubernikoff G, Vargas JD, Parker M, Judd RM. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. Circulation 2001; 104:1101–7.
31. 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.
32. Goldman MR, Brady TJ, Pykett IL, et al. Quantification of experimental myocardial infarction using nuclear magnetic resonance imaging and paramagnetic ion contrast enhancement in excised canine hearts. Circulation 1982;66:1012–6.
33. Lima JA, Judd RM, Bazille A, Schulman SP, Atalar E, Zerhouni EA. Regional heterogeneity of human myocardial infarcts demonstrated by

contrast-enhanced MRI: potential mechanisms. Circulation 1995;92:1117–25.

34. Rochitte CE, Lima JA, Bluemke DA, et al. Magnitude and time course of microvascular obstruction and tissue injury after acute myocardial infarction. Circulation 1998;98:1006–14.

35. Wu KC, Zerhouni EA, Judd RM, et al. Prognostic significance of microvascular obstruction by magnetic resonance imaging in patients with acute myocardial infarction. Circulation 1998;97:765–72.

36. Gerber BL, Garot J, Bluemke DA, Wu KC, Lima JA. Accuracy of contrast-enhanced magnetic resonance imaging in predicting improvement of regional myocardial function in patients after acute myocardial infarction. Circulation 2002;106:1083–9.

37. Woodard PK, Li D, Haacke EM, et al. Detection of coronary stenoses on source and projection images using three-dimensional MR angiography with retrospective respiratory gating: preliminary experience. AJR Am J Roentgenol 1998;170:883–8.

38. Kim WY, Danias PG, Stuber M, et al. Coronary magnetic resonance angiography for the detection of coronary stenoses. N Engl J Med 2001;345:1863–9.

39. Quinones MA, Douglas PS, Foster E, et al. ACC/AHA clinical competence statement on echocardiography: a report of the American College of Cardiology/American Heart Association/American College of Physicians-American Society of Internal Medicine Task Force on Clinical Competence. J Am Coll Cardiol 2003;41:687–708.

Appendix. ACCF/AHA Writing Committee to Develop a Clinical Competence Statement on Cardiac Imaging With Computed Tomography and Magnetic Resonance—Relationships With Industry

Name	Consultant	Research Grant	Scientific Advisory Board	Speaker's Bureau	Steering Committee	Stock Holder
Dr. Matthew J. Budoff	None	None	None	● General Electric	None	None
Dr. Mylan C. Cohen	None	None	None	None	None	None
Dr. Mario J. Garcia	None	● Philips Medical Systems ● Bristol Myers Squibb	None	● GE Healthcare ● Bristol Myers Squibb	None	None
Dr. John McB. Hodgson	● Volcano Therapeutics	● Bristol Myers Squibb	● Volcano Therapeutics	● Medicines ● Guillford ● Radi ● Volcano Therapeutics		
Dr. W. Gregory Hundley	None	● Mallinckrodt ● Bracco Diagnostics	None	● Bracco Diagnostics ● Pfizer	None	None
Dr. Joao A.C. Lima	None	● Merck, Inc. ● Fujisawa Inc. ● Toshiba Inc.	● GEMS	● Toshiba Inc.	None	None
Dr. Warren J. Manning	● Amesham Health	● Philips Medical Systems	● Astra-Zeneca ● Bristol Myers Imaging	None	None	None
Dr. Gerald M. Pohost	● White Memorial Hospital	● GE Healthcare	None	● Takeda	None	None
Dr. Paolo M. Raggi	None	● Pfizer	None	None	None	None
Dr. George P. Rodgers	None	None	None	None	None	None
Dr. John A. Rumberger	None	None	None	● GE Healthcare	None	None
Dr. Allen J. Taylor	None	Kos	None	● Pfizer ● Kos ● Wyeth	None	None

This table represents the relationships of committee members with industry that were reported orally at the initial Writing Committee meeting and updated in conjunction with all meetings and conference calls of the Writing Committee during the document development process. It does not necessarily reflect relationships with industry at the time of publication.

ACR PRACTICE GUIDELINE FOR THE PERFORMANCE AND INTERPRETATION OF CARDIAC COMPUTED TOMOGRAPHY (CT)

PREAMBLE

These guidelines are an educational tool designed to assist practitioners in providing appropriate radiologic care for patients. They are not inflexible rules or requirements of practice and are not intended, nor should they be used, to establish a legal standard of care. For these reasons and those set forth below, the American College of Radiology cautions against the use of these guidelines 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 physician or medical physicist in light of all the circumstances presented. Thus, an approach that differs from the guidelines, 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 the guidelines when, in the reasonable judgment of the practitioner, such course of action is indicated by the condition of the patient, limitations on available resources, or advances in knowledge or technology subsequent to publication of the guidelines. However, a practitioner who employs an approach substantially different from these guidelines is advised to document in the patient record information sufficient to explain the approach taken.

The practice of medicine involves not only the science, but also the art of dealing with the prevention, diagnosis, alleviation, and treatment of disease. The variety and complexity of human conditions make it impossible to always reach the most appropriate diagnosis or to predict with certainty a particular response to treatment.

Therefore, it should be recognized that adherence to these guidelines will not assure an accurate diagnosis or a successful outcome. All that should be expected is that the practitioner will follow a reasonable course of action based on current knowledge, available resources, and the needs of the patient to deliver effective and safe medical care. The sole purpose of these guidelines is to assist practitioners in achieving this objective.

I. INTRODUCTION

Cardiac computed tomography (CT) is an evolving modality that includes a variety of examinations to assess the anatomy and pathology of the central great vessels and pericardium as well as the function of the heart and cardiac valves. CT is a proven and useful procedure for detecting and characterizing cardiac and pericardial masses and pericardial effusion. With the development of multidetector CT (MDCT) scanners with increasing number of detector rows, narrow section thickness, increasing scanner speed, ability for electrocardiogram (ECG) gating, and radiation dose modulation, CT can also assess the coronary arteries and veins and can evaluate cardiac function. This guideline attempts to maximize the probability of detecting cardiac abnormalities with cardiac CT.

Cardiac CT involves the exposure of patients to ionizing radiation and should only be performed under the supervision of a physician with the necessary training in radiation protection to optimize examination safety. Medical physicists and trained technical staff must be available.

Cardiac CT should be performed only for a valid medical indication and with the minimum radiation exposure that provides diagnostic image quality.

While important abnormalities of the heart and associated structures can be detected on chest CT performed for other reasons, these guidelines are written specifically for dedicated examinations designed to detect cardiac pathology.

For further information on CT imaging of other structures within the chest and of the noncardiac vasculature see the ACR Practice Guideline for the Performance of Computed Tomography (CT) for the Detection of Pulmonary Embolism in Adults, the ACR Practice Guideline for the Performance of Pediatric and Adult Thoracic Computed Tomography (CT), and the ACR Practice Guideline for the Performance and Interpretation of Computed Tomography Angiography (CTA).

II.   DEFINITIONS

A. Cardiac CT

Cardiac CT is a chest CT performed primarily for the morphologic evaluation of the cardiac chambers, valves, ventricular myocardium, coronary arteries and veins, aortic root, central pulmonary arteries and veins, and pericardium. However, noncardiac structures are included and must be evaluated.

B. Unenhanced Cardiac CT

Unenhanced cardiac CT is a dedicated chest CT performed primarily for evaluating cardiac calcification, i.e., the coronary arteries (coronary calcium scoring), cardiac valves, pericardium, and cardiac masses. ECG gating reduces motion artifact and is required for calcium detection, scoring, and localization.

C. Contrast-Enhanced Cardiac CT

1. Contrast-enhanced cardiac CT is performed after intravenous (IV) administration of iodinated contrast to optimize evaluation of the cardiac chambers, myocardium, valves, and pericardium.
2. CT coronary arteriography is performed to characterize the origin and course of the coronary arteries and/or bypass grafts and to assess stenosis, and/or atherosclerotic plaque formation.
3. CT cardiac venography is performed to assess the cardiac or pulmonary veins.

III.   INDICATIONS

Unenhanced ECG-gated cardiac CT may be indicated for detecting and quantifying coronary artery calcium ("calcium scoring"). While the role of coronary artery calcium scoring is currently being refined, data support its use for risk stratification and therapeutic decision making in select patients with intermediate risk for a significant ischemic cardiac event. An additional indication is the localization of myocardial and pericardial calcium.

Indications for contrast-enhanced cardiac CT include, but are not limited to, the diagnosis, characterization, and/or surveillance of:

1. Arterial and venous aneurysms.
2. Atherosclerotic disease.
3. Traumatic injuries of arteries and veins.
4. Arterial dissection and intramural hematoma.
5. Arterial and venous thromboembolism (also see the ACR Practice Guideline for the Performance of Computed Tomography (CT) for the Detection of Pulmonary Embolism in Adults).
6. Vascular congenital anomalies and variants.
7. Vascular interventions (percutaneous and surgical, e.g., angioplasty, coronary stenting, coronary bypass grafts [CABGs], ablation therapy for cardiac dysrhythmia, valve surgery, aortic root replacement, pacemaker placement planning,).
8. Vascular infection, vasculitis, and collagen vascular diseases.
9. Sequelae of ischemic coronary disease (myocardial scarring, ventricular aneurysms, thrombi).
10. Cardiac tumors and thrombi.
11. Pericardial diseases.
12. Cardiac functional evaluation, especially in patients in whom cardiac function may not be assessed by magnetic resonance imaging (automatic implantable defibrillator, pacemaker, general MRI contraindications, etc.) or echocardiography (e.g., poor acoustic window).

All imaging facilities should have policies and procedures to reasonably attempt to identify pregnant patients prior to the performance of any examinations involving ionizing radiation. If the patient is known to be pregnant, the potential radiation risk to the fetus and clinical benefits of the procedure should be considered before proceeding with the study. (1995, 2005 - ACR Resolution 1a)

IV.   QUALIFICATIONS AND RESPONSIBILITIES OF PERSONNEL

See the ACR Practice Guideline for Performing and Interpreting Diagnostic Computed Tomography (CT) and the ACR Practice Guideline for the Performance and Interpretation of CT Angiography (CTA) for physician qualifications to interpret general CT examinations and CTA, and for qualifications of the Qualified Medical Physicist and the Radiologic Technologist. The

requirements set forth below will become applicable by July 1, 2008.

A. Physician

The physician shall have the responsibility for all aspects of the study including, but not limited to, reviewing all indications for the examination, specifying the imaging sequences to be performed, specifying the methods of image reconstruction, specifying the use and dosage of contrast and pharmacologic agents, interpreting images, generating an official interpretation,[1] and assuring the quality of the images and the interpretation.

1. Physician with prior qualifications in general and/or thoracic CT interpretation.

    The radiologist or other physician who meets the qualifications of the ACR Practice Guideline for Performing and Interpreting Diagnostic Computed Tomography (CT) has substantial knowledge of radiation biology, the physics of CT scanning, the principles of CT image acquisition and postprocessing including use of diagnostic workstations, and the design of CT protocols including rate and timing of contrast administration. The physician also will have substantial experience in CT interpretation, including CT of extracardiac thoracic structures that will be included on the cardiac CT examination, and experience with CT angiography of other regions of the body. Some of these physicians will also have substantial experience in other methods of cardiac imaging, assessment of cardiac function, and/or experience specifically in cardiac CT. These physicians are qualified to interpret coronary artery calcium scoring based on their prior experience. However, in order to achieve competency in all aspects of cardiac CT imaging, many physicians will require additional education in cardiac anatomy, physiology, pathology, and/or cardiac CT imaging.

    The supervising and interpreting physician with prior qualifications in general and/or thoracic CT interpretation should also meet one of the following requirements:

    a. Training in cardiac CT in an Accreditation Council for Graduate Medical Education (ACGME) or an American Osteopathic Association (AOA) approved training program to include:
        i. Education in cardiac anatomy, physiology, pathology, and cardiac CT imaging for a time equivalent to at least 30 hours of CME.
                    and
        ii. The interpretation, reporting, and/or supervised review of at least 50 cardiac CT examinations in the last 36 months. Coronary artery calcium scoring does not qualify as meeting these requirements.
                    or
    b. Completion of at least 30 hours of Category I CME in cardiac imaging, including:
        i. Cardiac CT, anatomy, physiology, and/or pathology, or documented equivalent supervised experience[2] in a center actively performing cardiac CT.
                    and
        ii. The interpretation, reporting, and/or supervised review of at least 50 cardiac CT examinations in the last 36 months. Coronary artery calcium scoring does not qualify as meeting these requirements.

2. Physician who does not have prior qualifications in general and/or thoracic CT interpretation.

    The radiologist or other physician who does not meet the qualifications of the ACR Practice Guideline for Performing and Interpreting Diagnostic Computed Tomography (CT) or who meets these qualifications only for a specific anatomic area outside of the thorax requires more extensive training and experience in CT scanning with an emphasis on the thorax and specific experience in cardiac CT scanning. In addition to specific training in imaging interpretation, this training must include knowledge of the principles of CT image acquisition and postprocessing including use of diagnostic workstations and the design of CT protocols including rate and timing of contrast administration. The physician must also meet the same requirements, or document equivalent training, as those delineated in the ACR Practice Guideline for Performing and Interpreting Diagnostic Computed Tomography (CT) with

---

[1]The ACR Medical Legal Committee defines official interpretation as that written report (and any supplements or amendments thereto) that attach to the patient's permanent record. In healthcare facilities with a privilege delineation system, such a written report is prepared only by a qualified physician who has been granted specific delineated clinical privileges for that purpose by the facilities governing body upon the recommendation of the medical staff.

[2] Documented equivalent supervised experience is defined as supervision at a center where the proctoring physician meets these criteria to independently interpret cardiac CT.

regard to knowledge of the physics of CT scanning and radiation biology. Some physicians will also require additional education in cardiac anatomy, physiology, and pathology.

The supervising and interpreting physician without prior qualifications in general and/or thoracic CT interpretation should meet the following requirements:

a. Completion of sufficient training and experience to meet the qualifications of the ACR Practice Guideline for Performing and Interpreting Diagnostic Computed Tomography (CT). For a physician who assumes responsibilities for CT imaging exclusively in a specific anatomical area such as cardiac CT, this includes:
   i. Completion of an ACGME approved training program in the specialty practiced plus 200 hours of Category I CME in the performance and interpretation of CT in the subspecialty where CT reading occurs.

   and

   ii. Supervision, interpretation, and reporting of 500 cases, at least 100 of which must be a combination of thoracic CT or thoracic CT angiography during the past 36 months in a supervised situation. Coronary artery calcium scoring does not qualify as meeting these requirements.

   and
b. Included in the above, completion of at least 30 hours of Category I CME in cardiac imaging, including
   i. Cardiac CT, anatomy, physiology, and/or pathology, or documented equivalent supervised experience[3] in a center actively performing cardiac CT.

   and

   ii. The interpretation, reporting, and/or supervised review of at least 50 cardiac CT examinations in the last 36 months. Coronary artery calcium scoring does not qualify as meeting these requirements.

3. Administration of pharmacologic agents

Physicians administering pharmacologic agents as part of cardiac CT imaging should be knowledgeable about the administration, risks,

and contraindications of the pharmacologic agents used and should be capable of monitoring the patient throughout the procedure.

4. Maintenance of competence

All physicians performing cardiac CT examinations should demonstrate evidence of continuing competence in the interpretation and reporting of those examinations. If competence is assured primarily on the basis of continuing experience, performance and interpretation of a minimum of 75 examinations every 3 years is recommended in order to maintain the physician's skills.

5. Continuing medical education

The physician's continuing medical education should be in accordance with the ACR Practice Guideline for Continuing Medical Education (CME) of 150 hours of approved education every 3 years, and should include CME in cardiac CT as is appropriate to the physician's practice needs.

B. Qualified Medical Physicist

A Qualified Medical Physicist is an individual who is competent to practice independently one or more of the subfields in medical physics. The ACR considers that certification and continuing education in the appropriate subfield(s) demonstrate that an individual is competent to practice one or more of the subfields in medical physics, and to be a Qualified Medical Physicist. The ACR recommends that the individual be certified in the appropriate subfield(s) by the American Board of Radiology (ABR) or for MRI, by the American Board of Medical Physics (ABMP) in magnetic resonance imaging physics.

The appropriate subfields of medical physics for this guideline are Therapeutic Radiological Physics, Diagnostic Radiological Physics, Medical Nuclear Physics, and Radiological Physics.

The continuing education of a Qualified Medical Physicist should be in accordance with the ACR Practice Guideline for Continuing Medical Education (CME). (2006 - ACR Resolution 16g)

C. Registered Radiologist Assistant

A registered radiologist assistant is an advanced level radiographer who is certified and registered as a radiologist assistant by the American Registry of Radiologic Technologists (ARRT) after having successfully completed an advanced academic program

---

[3]Documented equivalent supervised experience is defined as supervision at a center where the proctoring physician meets these criteria to independently interpret cardiac CT.

encompassing an ACR/ASRT (American Society of Radiologic Technologists) radiologist assistant curriculum and a radiologist-directed clinical preceptorship. Under radiologist supervision, the radiologist assistant may perform patient assessment, patient management and selected examinations as delineated in the Joint Policy Statement of the ACR and the ASRT titled "Radiologist Assistant: Roles and Responsibilities" and as allowed by state law. The radiologist assistant transmits to the supervising radiologists those observations that have a bearing on diagnosis. Performance of diagnostic interpretations remains outside the scope of practice of the radiologist assistant. (2006 - ACR Resolution 34)

D.   Radiologic Technologist

The technologist should participate in assuring patient comfort and safety, in preparing and positioning the patient for the CT examination including proper positioning of the ECG leads, and in obtaining the CT data in a manner suitable for interpretation by the physician. The technologist's continuing education credits should include continuing education in cardiac CT performance as is appropriate to the technologist's practice needs. Basic life support (BLS) and automatic defibrillator (AED) training is recommended.

V.   SPECIFICATIONS OF THE CONTRAST-ENHANCED CARDIAC CT EXAMINATION

The written or electronic request for cardiac CT should provide sufficient information to demonstrate the medical necessity of the examination and allow for its proper performance and interpretation.

Documentation that satisfies medical necessity includes 1) signs and symptoms and/or 2) relevant history (including known diagnoses). Additional information regarding the specific reason for the examination or a provisional diagnosis would be helpful and may at times be needed to allow for the proper performance and interpretation of the examination.

The request for the examination must be originated by a physician or other appropriately licensed health care provider. The accompanying clinical information should be provided by a physician or other appropriately licensed health care provider familiar with the patient's clinical problem or question and consistent with the state scope of practice requirements. (2006 - ACR Resolution 35)

The supervising physician must have complete understanding of the indications, risks, and benefits of the examination, as well as alternative imaging procedures. The physician must be familiar with potential hazards associated with CT, including potential adverse reactions

to contrast media. The physician should be familiar with relevant ancillary studies that the patient may have undergone. The physician performing CT interpretation must have a clear understanding and knowledge of the anatomy and pathophysiology relevant to the CT examination.

Standard imaging protocols may be established and varied on a case-by-case basis when necessary. These protocols should be reviewed and updated periodically.

A.   Patient Selection and Preparation

The appropriate guidelines for patient selection for a contrast-enhanced cardiac CT examination will continue to evolve with the introduction of new scanner technology with higher temporal and spatial resolution. The availability of specific scanner technology (16 vs 64 MDCT for example) may impact patient selection for contrast-enhanced cardiac studies as the positive and negative predictive values will vary based on available hardware configurations. Patients scheduled for CT coronary arteriography must have adequate peripheral venous access and be able to cooperate with breath holding and the administration of medication as needed (i.e., beta blockers or nitroglycerin/nitrates). Patients referred for cardiac CT should be first evaluated by an appropriate health care provider knowledgeable of risk factors for cardiac and vascular disease.

Based on the results reported in recent publications, selection for CT coronary arteriography may include patients with:

1.   Unexplained or atypical chest pain when an aberrant origin of the coronary artery is considered possible.
2.   Unexplained or atypical chest pain with low to intermediate likelihood of coronary artery disease based on gender, age, and risk factors.
3.   Typical or atypical chest pain with normal or equivocal stress test and normal or equivocal ECG findings.
4.   Unexplained severe chest pain in the acute setting without a clinical history of coronary artery disease. Cardiac CT may be used as a rapid triage method to evaluate for the presence of coronary artery disease, and to exclude pulmonary embolism or aortic dissection.

Additional indications for coronary CT arteriography include patients with CABG surgery:

1.   With new or recurrent symptoms of chest pain or chest pain equivalent to confirm graft patency or detect graft stenoses.

2. Who are scheduled for additional cardiac surgery (e.g., aortic valve replacement) when preoperative definition of anatomic detail, including the bypass grafts, is critical.

Cardiac CT should be used selectively in patients with a high pretest probability of significant coronary artery disease based on clinical, laboratory or other imaging studies, including stress testing. These higher risk patients are more likely to need invasive coronary catheter studies and interventions. CT should be used with caution in patients with borderline or compromised renal function since if the patient requires an invasive procedure, the contrast load will be significantly increased by performing the CT which could result in even a greater chance of renal impairment.

Patients should have a liquid only diet for 3 hours and abstain from caffeine for at least 6 hours before the study. When a patient has a relative contraindication to the administration of IV iodinated contrast media, measures to reduce the possibility of contrast media reactions or nephrotoxicity should be followed as defined in the ACR Practice Guideline for the Use of Intravascular Contrast Media. A physician should also be available to treat adverse reactions to IV contrast media.

A 20-gauge or larger right antecubital IV catheter is the preferred administration route of iodinated contrast media for CT coronary arteriography. To minimize the risk of contrast media extravasations, all catheters used for cardiac CT angiography should first be tested with a rapidly injected bolus of sterile saline to insure that the venous access is secure and effective. Trained medical personnel should monitor the injection site for signs for IV extravasation. Departmental procedures for treating IV extravasations should be documented.

Because faster heart rates tend to degrade image quality, patients may need to be medicated with beta-blockers, unless contraindicated, prior to or during the cardiac CT arteriogram. Nitroglycerin/nitrates may also be administered in conjunction with the study. Physicians performing CT coronary arteriography should be knowledgeable of the administration, risks, and contraindications of these drugs. Blood pressure and heart rate should be monitored.

Patients suffering from anxiety or claustrophobia may require sedation or additional assistance. Administration of moderate or "conscious" sedation may enable achievement of the examination. If moderate sedation is necessary, refer to the ACR Practice Guideline for Adult Sedation/Analgesia or the ACR Practice Guideline for Pediatric Sedation/Analgesia.

B. Examination Technique

An initial unenhanced CT acquisition may be needed to depict calcification of the arteries, valves, pericardium, and myocardium to detect mural or extravascular hemorrhage or to localize an anatomic structure. The section thickness may vary but should not exceed 5 mm.

Because of substantial variations in the time required for an IV contrast media injection to reach the targeted vascular anatomy, an assessment of patient-specific circulation time is required in protocols that include the administration of IV contrast media. Circulation timing can be performed using two techniques:

1. Test bolus technique. IV injection of a small bolus (e.g., 10-15 ml) of contrast media at the rate and via the IV site to be used during the examination. Sequential stationary CT images are acquired at the anatomic level of interest during the test bolus. The timing of the contrast delivery and ensuing attenuation of the vascular lumen of interest are then plotted to create a time-density curve. The time of the peak of vascular enhancement is used to determine the scanning delay.

2. Bolus track and trigger technique. Following the initiation of the full dose of contrast media injection, automated triggering CT software monitors the attenuation within the cardiac structure of interest. The CT is automatically started when the enhancement in the monitored vessel or structure reaches a predetermined operator selected level.

A right arm injection is preferable to avoid artifacts from undiluted contrast media in the left brachiocephalic vein. A bolus of saline following the iodinated contrast media injection may reduce the volume of contrast media required to achieve adequate vascular opacification and reduce artifacts from high concentration of contrast media in the superior vena cava and right atrium. Contrast injection parameters should be modified on an individual patient basis whenever possible. The administration of iodinated contrast media for the contrast-enhanced cardiac CT should ideally be performed with a minimum flow rate of 3 ml per second in any patient weighing 50 or more kilograms. Higher flow rates of 5 ml per second or greater are frequently required for larger patients, and in general are required for shorter acquisition scan times. In children, contrast media dosing should be scaled by body weight with injection rate scaled similarly. Preferably the contrast is delivered via powered injection. The volume of contrast media should be selected in consideration of the patient's weight and comorbidities that might increase the risk of nephrotoxicity.

The contrast-enhanced cardiac CT acquisition should be performed with a section thickness of $\leq$ 1.50 mm depending on the cardiac structure to be assessed. Calcium scoring typically has been performed using 3 mm section thickness, but thinner sections may be used. The field of view (FOV) should span from below the tracheal carina through the apex of the heart. If the patient has had a CABG, the FOV should span from the top of the clavicular heads to the apex of the heart, to include the entire length of internal mammary grafts using breath holding and retrospective cardiac gating. Multisector reconstruction associated with lower pitch values may improve temporal resolution of the reconstructed images, depending on the heart rate and the CT scanner.

For CT coronary arteriography, the use of oral and/or IV beta-blockers, if not contraindicated, may be used during the scan, to obtain a stable heart rate of approximately 50-70 beats per minute. The scan data should be reconstructed at various phases of the cardiac cycle with overlapping sections at a maximum slice increment of 50% of the effective section thickness and a FOV of approximately 25 cm. Thin section reconstruction during the most optimal temporal window is recommended to improve conspicuity of the structures of interest. Thick section reconstructions that span the entire cardiac cycle can be performed to assess cardiac contractility. When recording to film, display settings of window width and level should be customized to clearly delineate the enhanced vascular lumen from mural calcification and myocardium.

Postprocessing of the cardiac CT data should be performed by physicians, registered radiology technologists, or other experienced personnel knowledgeable of cardiovascular anatomy and pathophysiology. The data may be formatted and presented using various display techniques, including multiplanar reformations (MPRs), maximum intensity projections (MIPs), 3D volume renderings (VR), 3D shaded surface displays, and/or 4D dynamic reconstructions.

Images are to be labeled with the following: a) patient identification, b) facility identification, c) examination date, and d) the side (right or left) of the anatomic site imaged. Postprocessed images should be recorded and archived in a manner similar to the source CT sections.

C. Interpretation

Cardiac CT data should be interpreted on a computer workstation that displays axial, reformatted, and postprocessed images. Interpretation of the CT coronary arteriogram includes assessment of intraluminal plaques, to include segmental vascular location, attenuation characteristics, and degree of luminal narrowing; vascular anomalies; and abnormalities of the cardiac chambers,

myocardium, and pericardium. Frequently, reconstruction from different phases of the cardiac cycle may be required to fully interpret the examination. For functional cardiac assessment, multiples phases should be examined. Interpretation of the noncardiac portion of the examination should include use of proper windowing and leveling for adequate visualization of the soft tissue, mediastinum, pulmonary, and bony portions of the chest. Comparison with previous chest CT images should be performed if appropriate.

VI.   DOCUMENTATION

Reporting should be in accordance with the ACR Practice Guideline for Communication of Diagnostic Imaging Findings. In addition to examining the cardiac structures of interest, the CT sections should be examined for extracardiac abnormalities that may have clinical relevance. These abnormalities should also be described in the formal report of the examination.

VII.   EQUIPMENT SPECIFICATIONS

For diagnostic quality cardiac CT, the CT scanner should meet or exceed the following specifications:

1. Contrast-enhanced cardiac CT by MDCT, including CT coronary arteriography, a scanner capable of achieving in-plane spatial resolution $\leq$ 0.5 x 0.5 mm axial, z-axis spatial resolution $\leq$ 1 mm longitudinal, and temporal resolution $\leq$ 0.25 sec.
2. Non-contrast-enhanced MDCT for coronary artery calcium scoring may be adequately performed on a scanner with a temporal resolution of $\leq$ 0.50 second using retrospectively gated volume-series acquisition or a prospectively triggered "stop and shoot" sequential acquisition.
3. Tube heat capacity that allows for a single $\geq$ 20 second acquisition.
4. Minimum section thickness: no greater than 3 mm; no greater than 1.5 mm for CT coronary arteriography.

To maximize the CT interpretation, any CT scanner used for cardiac CT must allow display and interpretation of the full 12 bits (from -1,000 to 3,095 Hounsfield Units) of attenuation information. Additionally the display FOV must be sufficient to assess the vasculature region of interest, the end-organ, and adjacent structures.

For adequate contrast-enhanced cardiac CT, including CT coronary arteriography, a power injector capable of delivering a programmed volume of a contrast agent at a steady flow rate of at least 3 cc per second for delivery of $\geq$ 300 mg of iodine/ml is necessary. A dual chambered

power injector is preferred if a saline flush will be administered immediately after the intravenous contrast material injection.

A workstation capable of creating straight or curved multiplanar reformations, maximum intensity projections, volume renderings that can be compared across multiple cardiac phases and 4D dynamic reconstructions should be available for coronary CTA and for other applications as appropriate.

Appropriate emergency equipment and medications must be immediately available to treat adverse reactions, an acute coronary syndrome, and cardiac arrest. The equipment and medications should be monitored for inventory and drug expiration dates on a regular basis.

VIII. RADIATION SAFETY IN IMAGING

Radiologists, medical physicists, radiologic technologists, and all supervising physicians have a responsibility to minimize radiation dose to individual patients, to staff, and to society as a whole, while maintaining the necessary diagnostic image quality. This is the concept "As Low as Reasonably Achievable (ALARA)".

Facilities, in consultation with the medical physicist, should have in place and should adhere to policies and procedures, in accordance with ALARA, to vary examination protocols to take into account patient body habitus, such as height and/or weight, body mass index or lateral width. The dose reduction devices that are available on imaging equipment should be active or manual techniques should be used to moderate the exposure while maintaining the necessary diagnostic image quality. Patient radiation doses should be periodically measured by a medical physicist in accordance with the appropriate ACR Technical Standard. (2006 - ACR Resolution 17)

IX. QUALITY CONTROL AND IMPROVEMENT, SAFETY, INFECTION CONTROL, AND PATIENT EDUCATION CONCERNS

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 Concerns appearing elsewhere in the ACR Practice Guidelines and Technical Standards book.

Equipment performance monitoring should be in accordance with the ACR Technical Standard for Medical Physics Performance Monitoring of Computed Tomography (CT) Equipment.

REFERENCES

1. Achenbach S, Giesler T, Ropers D, et al. Detection of coronary artery stenoses by contrast-enhanced, retrospectively electrocardiographically-gated, multi-slice spiral computed tomography. *Circulation* 2001;103:2535-2538.
2. Achenbach S, Ropers D, Hoffmann U, et al. Assessment of coronary remodeling in stenotic and nonstenotic coronary atherosclerotic lesions by

multidetector spiral computed tomography. *J Am Coll Cardiol* 2004;43:842-847.

3. Achenbach S, Ulzheimer S, Baum U, et al. Noninvasive coronary angiography by retrospectively ECG-gated multislice spiral CT. *Circulation* 2000;102:2823-2828.

4. ACR practice guideline for the performance and interpretation of CT angiography (CTA). In: *Practice Guidelines and Technical Standards*. Reston, Va: American College of Radiology; 2005:221-226.

5. ACR practice guideline for the performance of pediatric and adult thoracic computed tomography (CT). In: *Practice Guidelines and Technical Standards*. Reston, Va: American College of Radiology; 2005:183-187.

6. ACR practice guideline for the use of intravascular contrast media. In: *Practice Guidelines and Technical Standards*. Reston, Va: American College of Radiology; 2005:21-25.

7. Araoz PA, Mulvagh SL, Tazelaar HD, et al. CT and MR imaging of benign primary cardiac neoplasms with echocardiographic correlation. *Radiographics* 2000;20:303-1319.

8. Axel L. Assessment of pericardial disease by magnetic resonance and computed tomography. *J Magn Reson Imaging* 2004;19:816-826.

9. Boxt LM. Magnetic resonance and computed tomographic evaluation of congenital heart disease. *J Magn Reson Imaging* 2004;19:827-847.

10. Boxt LM, Lipton MJ, Kwong RY, et al. Computed tomography for assessment of cardiac chambers, valves, myocardium, and pericardium. *Cardiol Clin* 2003;21:561-585.

11. Datta, J, White CS, Gilkerson RC, et al. Anomalous coronary arteries in adults: depiction at multi-detector row CT angiography. *Radiology* 2005;235:812-818.

12. Desjardins B, Kazerooni EA. ECG-gated cardiac CT. *AJR* 2004;182:993-1010.

13. Detrano RC, Anderson M, Nelson J, et al. Coronary calcium measurements: effect of CT scanner type and calcium measure on rescan reproducibility – MESA study. *Radiology* 2005;236:477-484.

14. Flohr T, Ohnesorge B. Heart rate adaptive optimization of spatial and temporal resolution for electrocardiogram-gated multislice spiral CT of the heart. *J Comput Assist Tomogr* 2001;25:907-923.

15. Flohr T, Prokop M, Becker C, et al. A retrospectively ECG-gated multislice spiral CT scan and reconstruction technique with suppression of heart pulsation artifacts for cardio-thoracic imaging with extended volume coverage. *Eur Radiol* 2002;12:1497-1503.

16. Flohr T, Stierstorfer K, Raupach R, et al. Performance evaluation of a 64-slice CT system with z-flying focal spot. *Rofo* 2004;176:1803-1810.

17. Giesler T, Baum U, Ropers D, et al. Noninvasive visualization of coronary arteries using contrast-enhanced multidetector CT: influence of heart rate on image quality and stenosis detection. *AJR* 2002;179:911-916.

18. Halliburton SS, Stillman AE, Lieber M, et al. Potential clinical impact of variability in the measurement of coronary artery calcification with sequential MDCT. *AJR* 2005;184:643-648.

19. Hoffmann MH, Shi H, Manzke R, et al. Noninvasive coronary angiography with 16-detector row CT: effect of heart rate. *Radiology* 2005;234:86-97.

20. Hoffmann U, Moselewski F, Cury RC, et al. Predictive value of 16-slice multidetector spiral computed tomography to detect significant obstructive coronary artery disease in patients at high risk for coronary artery disease: patient versus segment-based analysis. *Circulation* 2004;110:2638-2643.

21. Hong C, Chrysant GS, Woodard PK, et al. Coronary artery stent patency assessed with in-stent contrast enhancement measured at multi-detector row CT angiography: initial experience. *Radiology* 2004;233:286-291.

22. Jakobs TF, Becker CR, Ohnesorge B, et al. Multislice helical CT of the heart with retrospective ECG gating: reduction of radiation exposure by ECG-controlled tube current modulation. *Eur Radiol* 2002;12:1081-1086.

23. Jongbloed MR, Dirksen MS, Bax JJ, et al. Atrial fibrillation: multi-detector row CT of pulmonary vein anatomy prior to radiofrequency catheter ablation: initial experience. *Radiology* 2005;234:702-709.

24. Kalra MK, Maher MM, Toth TL, et al. Techniques and applications of automatic tube current modulation for CT. *Radiology* 2004;233:649-657.

25. Knollmann FD, Moller J, Gebert A, et al. Assessment of coronary artery stent patency by electron-beam CT. *Eur Radiol* 2004;14:1341-1347.

26. Kopp AF, Schroeder S, Kuettner A, et al. Cornary arteries: retrospectively ECG-gated multi-detector row CT angiography with selective optimization of the image reconstruction window. *Radiology* 2001;221:683-688.

27. Koyama Y, Matsuoka H, Mochizuki T, et al. Assessment of reperfused acute myocardial infarction with two-phase contrast-enhanced helical CT: prediction of left ventricular function and wall thickness. *Radiology* 2005;235:804-811.

28. Koyama Y, Mochizuki T, Higaki J. Computed tomography assessment of myocardial perfusion, viability, and function. *J Magn Reson Imaging* 2004;19:800-815.

29. Kuettner A, Beck T, Drosch T, et al. Diagnostic accuracy of noninvasive coronary imaging using 16-detector slice spiral computed tomography with 188 ms temporal resolution. *J Am Coll Cardiol* 2005;45:123-127.

30. Lacomis JM, Wigginton W, Fuhrman C, et al. Multi-detector row CT of the left atrium and pulmonary veins before radio-frequency catheter ablation for artial fibrillation. *Radiographics* 2003;23:S35-S48.

31. Lawler LP, Ney D, Pannu HK, et al. Four-dimensional imaging of the heart based on near-isotropic MDCT data sets. *AJR* 2005;184:774-776.

32. Leber AW, Knez A, Becker A, et al. Accuracy of multidetector spiral computed tomography in identifying and differentiating the composition of coronary atherosclerotic plaques: a comparative study with intracoronary ultrasound. *J Am Coll Cardiol* 2004;43:1241-1247.

33. Leber AW, Knez A, von Ziegler F, et al. Quantification of obstructive and nonobstructive coronary lesions by 64-slice computed tomography: a comparative study with quantitative coronary angiography and intravascular ultrasound. *J Am Coll Cardiol* 2005;46:147-154.

34. Mahnken AH, Buecker A, Wildberger JE, et al. Coronary artery stents in multislice computed tomography: in vitro artifact evaluation. *Invest Radiol* 2004;39:27-33.

35. Mahnken AH, Seyfarth T, Flohr T, et al. Flat-panel detector computed tomography for the assessment of coronary artery stents: phantom study in comparison with 16-slice spiral computed tomography. *Invest Radiol* 2005;40:8-13.

36. Maintz D, Seifarth H, Flohr T, et al. Improved coronary artery stent visualization and in-stent stenosis detection using 16-slice computed tomography and dedicated image reconstruction technique. *Invest Radiol* 2003;38:790-795.

37. Mochizuki T, Hosoi S, Higashino H, et al. Assessment of coronary artery and cardiac function using multidetector CT. *Semin Ultrasound CT MR* 2004;25:99-112.

38. Mochizuki T, Murase K, Higashino H, et al. Two- and three-dimensional CT ventriculography: a new application of helical CT. *AJR* 2000;174:203-208.

39. Mollet NR, Cademartiri F, Nieman K, et al. Multislice spiral computed tomography coronary angiography in patients with stable angina pectoris. *J Am Coll Cardiol* 2004;43:2265-2270.

40. Moselewski F, Ropers D, Pohle K, et al. Comparison of measurement of cross-sectional coronary atherosclerotic plaque and vessel areas by 16-slice multidetctor computed tomography versus intravascular ultrasound. *Am J Cardiol* 2004;94:1294-1297.

41. Moser KW, Bateman TM, O'Keefe JH Jr, et al. Interscan variability of coronary artery calcium quantification using an electrocardiographically pulsed spiral computed tomographic protocol. *Am J Cardiol* 2004;93:1153-1155.

42. Nieman K, Cademartiri F, Lemos PA, et al. Reliable noninvasive coronary angiography with fast submillimeter spiral computed tomography. *Circulation* 2002; 106:2051-2054.

43. Nieman K, Oudkerk M, Rensing BJ, et al. Coronary angiography with multi-slice computed tomography. *Lancet* 2001;357:599-603.

44. Nieman K, Rensing BJ, van Geuns RJ, et al. Non-invasive coronary angiography with multislice spiral computed tomography: impact of heart rate. *Heart* 2002;88:470-474.

45. Pannu HK, Flohr TG, Corl FM, et al. Current concepts in multi-detector row CT evaluation of coronary arteries: principles, techniques and anatomy. *Radiographics* 2003;23:S111-S125.

46. Ropers D, Baum U, Pohle K, et al. Detection of coronary artery stenoses with thin-slice multi-detector row spiral computed tomography and multiplanar reconstruction. *Circulation* 2003;107:664-666.

47. Schlosser T, Konorza T, Hunold P, et al. Noninvasive visualization of coronary artery bypass grafts using 16-detector row computed tomography. *J Am Coll Cardiol* 2004; 44:1224-1229.

48. Schlosser T, Pagonidis K, Herborn CU, et al. Assessment of left ventricular parameters using 16-MDCT and new software for endocardial and epicardial border delineation. *AJR* 2005;184:765-773.

49. Schoenhagen P, Halliburton SS, Stillman AE, et al. Noninvasive imaging of coronary arteries: current and future role of multi-detector row CT. *Radiology* 2004;232:7-17.

50. Schoepf UJ, Becker C, Ohnesorge BM, et al. CT of coronary artery disease. *Radiology* 2004;232:18-37.

51. Schragin JG, Weissfeld JL, Edmundowicz D, et al. Non-cardiac findings on coronary electron beam computed tomography scanning. *J Thorac Imaging* 2004;19:82-86.

52. Schroeder S, Kopp AF, Baumbach A, et al. Noninvasive detection and evaluation of atherosclerotic coronary plaques with multislice computed tomography. *J Am Coll Cardiol* 2001;37:1430-1435.

53. White RD. MR and CT assessment for ischemic cardiac disease. *J Magn Reson Imaging* 2004;19:659-675.

54. Yamamuro M, Tadamura E, Kubo S, et al. Cardiac functional analysis with multi-detector row CT and segmental reconstruction algorithm: comparison with echocardiography, SPECT, and MR imaging. *Radiology* 2005;234:381-390.