Understanding the Heart

CT and MRI for Coronary Heart Disease

Gopi Kiran Reddy Sirineni, MD and Arthur E. Stillman, MD, PhD

Abstract: Methods of noninvasive evaluation of coronary artery disease—including multidetector row computed tomography, electron beam computed tomography, magnetic resonance imaging, and nuclear studies (single photon emission computed tomography, positron emission tomography)—are reviewed.

Key Words: coronary artery disease, computed tomography, magnetic resonance imaging

(*J Thorac Imaging* 2007;22:107–113)

Coronary atherosclerosis continues to be one of the great killers in the modern world. Coronary atherosclerosis and its associated conditions remain a severe strain on health care spending in developed economies.[1] Despite the advancements in diagnosis and treatment of coronary artery disease (CAD), associated mortality and morbidity continue to be high.

Invasive catheter coronary angiography is the reference standard for CAD. It is desirable to have a noninvasive test for CAD because of its cost and morbidity. Multidetector row computed tomography (MDCT), electron beam computed tomography (EBCT), magnetic resonance Imaging (MRI), nuclear studies (single photon emission computed tomography, positron emission tomography) all have roles for noninvasive evaluation of CAD.

Computed tomography of the heart was unimaginable until the advent of EBCT and more recently MDCT technology. This was because of the fast motion of the heart and small caliber of the coronary vessels. MDCT has relatively high spatial resolution and generates images of near isotropic quality. Although the temporal resolution of MDCT has not yet matched that of EBCT, acceptable image quality can be achieved with temporal resolution as low as 165 ms.[2–4] Advancements in MDCT technology (Dual source CT, 256 Slice MDCT) improve the temporal resolution thereby reducing the motion artifacts.[5–7] However, further advancements in terms of spatial and temporal resolution are required if MDCT is to match catheter angiography.

From the Division of Cardiothoracic Imaging, Department of Radiology, Emory University School of Medicine, 1364 Clifton Rd NE, Atlanta, GA, 30322.

Reprints: Arthur E. Stillman, MD, PhD, Division of Cardiothoracic Imaging, 1365 Clifton Rd NE, Suite AT 506, Atlanta, GA 30322 (e-mail: aestill@emory.edu).

EVALUATION OF ATHEROMATOUS PLAQUE BY MDCT

Catheter angiography provides accurate information about the lumen of the coronary artery but does not reliably detect noncritical disease because it lacks the ability to image the wall of the artery. Significant disease can exist in the coronary arterial wall even though the lumen is nonstenotic owing to positive remodeling of the wall. MDCT on the other hand provides information about the lumen and the wall as well. Theoretically differences in CT attenuation can be expected for lipid, fibrous, and calcified plaque because of the inherent difference in the respective plaque composition and studies have used this fact in an attempt to classify plaques by MDCT.[8,9] The sensitivity of detection of calcified plaque is very high; however, the sensitivity of detection of noncalcified plaque at present is between 50% and 80%. Lumen and plaque area measured on MDCT correlates well with the intravascular ultrasound (IVUS) measurements.[10] Although the ability of MDCT to characterize plaque still lacks the accuracy of IVUS, its noninvasive nature as compared with IVUS makes MDCT an appealing alternative.[9]

Calcified Plaque and Calcium Scoring

EBCT has been in use for the detection of coronary calcium for more than a decade now.[11] Wide availability of MDCT in comparison with EBCT and improvements in MDCT hardware resulted in MDCT evolving as the current prime modality of calcium scoring.

Coronary calcium scoring is being increasingly used for risk stratification of CAD in asymptomatic patients. Calcium scores show good correlation with the total coronary atherosclerotic plaque burden and point toward future adverse cardiac events like myocardial infarction and sudden cardiac death.[12] High absolute calcium scores in asymptomatic patients greatly increase the chance of the patient having unfavorable cardiac outcome in future. Hence high calcium scores currently form one of the triggers for institution of prophylactic medical therapy to prevent morbidity and mortality.[13] Data are lacking regarding the validity of calcium scores as a screening tool in isolation with other risk factors to predict future adverse cardiac events and also there is no consensus on

whether CAD risk can be lowered in patients with high calcium scores.

The calcium scoring system developed by Agatston et al[11] was originally intended for use with EBCT. The Agatston score was calculated from the coronary calcium that was greater than 130 HU in density and at least 2 contiguous pixels in area. This score has now been adapted for use with MDCT. In comparison with the Agatston score, other representations of calcium burden such as calcium volume and calcium mass have been shown to have better correlation with plaque burden and have low interscan and interobserver variability.[14] However, the Agatston score remains, currently, the primary way of reporting and interpreting the calcium burden.

Evaluation of Noncalcified Plaque

Unlike calcium scoring, identification of noncalcified plaque requires the presence of contrast in the vessel lumen. On MDCT, plaque having a typical attenuation value less than 130 HU or according to some authors[15] attenuation values less than the attenuation of the contrast in the vessel lumen can be considered as noncalcified plaque.

Coronary atherosclerosis manifests as a spectrum of disease that undergoes change according to time. Histologic classification exists to describe these changes and the relevance of the changes to likely adverse events.[16,17] Studies have attempted to classify the atherosclerotic lesion by MDCT on similar lines.[18–20] Remodeling Index (RI) is defined as the ratio between cross-sectional area of the entire vessel at the site of maximum narrowing and the average of distal and proximal reference vessel cross-sectional areas. Measurements of RI can be performed on MDCT. A higher RI is associated with increased incidence of future adverse coronary events, but there is no definite cut-off value to identify the vulnerable patients.[15] On MDCT, the typical attenuation values of the lipid rich plaques and the noncalcified fibrous plaques are 50 to 70 HU and 90 to 116 HU, respectively.[18–20] The calcified plaques usually have attenuation values above 130 HU. Studies have reported mean attenuation values of calcified plaque around 390 HU.[19] There is a substantial overlap between the attenuation values of different types of plaque to reliably classify them on the basis of attenuation differences alone and also the thin fibrous cap of the vulnerable plaque cannot be resolved at the current spatial resolution of MDCT.[9,18–20]

Innovative techniques for the detection of plaque composition such as fractal textural analysis and wavelet analysis may help in the characterization of plaque.[21–23]

Obstructive CAD

The diagnostic accuracy of MDCT to detect significant coronary artery stenosis has been increasing with increase in the number of detector rows (4-slice to 16-slice to 64-slice currently) and faster gantry rotation.[24,25] Most studies in the past showed a moderate predictive value for detecting stenosis on per segment analysis but excluded unevaluable segments for arriving at the results.[26,27] Few studies that evaluated the performance of MDCT on a per patient basis reported high positive predictive values (PPVs) for the detection of stenosis on MDCT.[28,29] A recent study using a 64-slice MDCT reported a high PPV of 93% on per patient basis.[25] Another study, similarly, showed a PPV of 98% but about 8% of the total segments were unevaluable owing to motion, calcification, or stents.[30] Most of the studies, however, report a high negative predictive value of 95% to 98%.[25,30,31]

Despite the advancements in technology, many limitations in MDCT imaging of coronaries still exist. Motion is probably one of the most important factors affecting the image quality of coronary CT angiogram. Imaging patients with heart rates above 65 bpm and patients with arrhythmias is currently a challenge. Artifacts from vessel wall calcifications and stents make the visualization of adjacent lumen difficult. Visualization of vessel lumen on CTA is relatively better in patients with diffuse calcification when compared with those with clumpy calcification. Some institutions perform a "check" calcium scoring acquisition before CTA and discontinue the scan if a high calcium score is noted. However, no consensus exists regarding the cut-off value of calcium score beyond which CTA is not performed. Variable contrast enhancement can also be problematic.

MDCT VERSUS CATHETER ANGIOGRAPHY

Catheter angiography is not only more accurate for the diagnosis of CAD than MDCT, it also guides patient management. This latter attribute has yet to be addressed in studies using MDCT. Certainly if MDCT is to replace diagnostic coronary angiography, it must also be able to triage medical therapy, percutaneous interventions, and coronary bypass surgery. Some of the shortcomings of MDCT may be overcome to a certain degree by integrating MDCT with other imaging modalities like PET, MRI, and SPECT to gather complimentary information.

MYOCARDIAL VIABILITY ON MDCT

Apart from the morphologic information of the heart, MDCT can also provide information regarding the status of myocardial perfusion.[32–34] Myocardial defects can be categorized according to the enhancement patterns as early defect (ED), residual defect (RD), and late enhancement.[34] These are analogous to descriptions on MR perfusion imaging of the myocardium. ED is noted as dark area having significantly lower attenuation than the normally enhancing myocardium in the early phase (30 to 60 s) after contrast injection. ED indicates decrease in myocardial blood flow in the area. RD is noted as a dark zone with low attenuation with respect to the surrounding hyperenhanced myocardium in the delayed phase of imaging (5 to 10 min after initial contrast injection). This represents the necrotic myocardium with no chance for recovery. Late enhancement is the hyperenhanced region in delayed phase of imaging

usually noted surrounding RD. This hyperenhancement might predict the functional recovery on reperfusion.[34] Although many animal models were studied, very few human studies exist in literature. The main reasons for this slow progress on MDCT viability imaging are the increased radiation dose and contrast volume associated with perfusion imaging. MRI showing the same information with no radiation effects and relatively safer contrast has made MRI the choice in this regard. Some perfusion information can also be inferred from routine MDCT angiography.[35]

MRI OF HEART

MRI of heart can provide useful information about morphology of coronary atherosclerosis, myocardial function, and myocardial viability. Unlike MDCT, MRI does not involve ionizing radiation and the toxicity associated with MR (gadolinium) contrast is low. This makes MRI a safer modality in comparison with either MDCT or SPECT. Unlike MDCT, MRI typically involves acquisition of multiple datasets with different sequences or parameters for a single investigation to be complete and can be time consuming.

Coronary MR Angiography

As with CT, respiratory and cardiac motion is the major limiting factor for performing a successful MRI of the heart. Echocardiography (ECG) gating is used to compensate for the cardiac motion and the respiratory motion is minified by either breath hold or by navigator gating with free breathing. The fast gradient echo 2-dimensional sequences that were used previously are now replaced by 3-dimensional volumetric acquisitions of the images.[36] The steady state free precession (SSFP) sequences are currently popular for imaging the coronaries as they produce high signal to noise ratio with good contrast and spatial resolution of the coronaries.[37] The volume targeted sequences in which the major arteries are images in a thin slab are also currently used.

There is no agreement in the existing literature over the reliability of MRI to detect stenotic lesions largely due to the variety of techniques. One study reported a moderate sensitivity and specificity of 82% and 91% in detecting significant stenosis.[36] However, 13% of the patients were excluded for varying reasons from the analysis, thereby limiting its application to a real clinical setting. Another recent study evaluating the SSFP MR coronary angiography reported a low sensitivity and specificity of 32% to 65% and 53% to 73%, respectively for the detection of coronary stenosis and had a high percentage of unevaluable arteries.[37] Low spatial resolution coupled with poor reliability in the detection of stenosis limit the use of MR angiography in comparison with MDCT for coronaries.

Coronary Plaque Assessment by MRI

Atherosclerotic plaque imaging in large caliber arteries like aorta and carotid has been of interest for a long time. Cardiac motion, respiratory motion, the deep location of coronary arteries with respect to surface coils and low spatial resolution limit the use of MR for the evaluation of coronary plaque. Typically double inversion recovery black blood images are used for the investigation of plaque structure and composition.[38] Respiratory compensation is either by breath hold or navigator gating and ECG triggering is used for cardiac motion compensation.[39] The traditional 2-dimensional imaging sequence produces an in plane resolution of 0.5 mm but has a slice thickness of 2 mm. Usually, cross-sectional images of the plaque are obtained. Newer 3-dimensional volumetric acquisitions[40,41] are being used to produce a near isotropic voxels and hence plaque can be assessed in multiple planes using postprocessing. Reliability in characterizing coronary plaque is relatively poor in comparison with IVUS. Newer contrast agents such a Gadoflourine and molecular imaging with contrast tagged fibrin-specific molecules may improve the identification and characterization of plaque in future.[42,43] Novel techniques like intravascular MRI could provide better understanding of atherosclerotic plaque in future.[44]

Cardiac Function on MRI

Gradient echo sequences are used to acquire the cine images in the short axis or long axis. SSFP sequences are also alternatively used because of their shorter acquisition times and higher spatial resolution.[45] Cardiac motion is captured over multiple phases of the cardiac cycle. Assessment of ventricular function is considered to be highly accurate and is considered to be the gold standard for the calculation of ventricular volumetric parameters such as left ventricular (LV) ejection fraction, end diastolic volume, stroke volume, and LV mass.[46] Contrast administration is not required if the imaging is solely performed for ventricular functional assessment. Depending on the pulse sequence used, administration of contrast may improve image quality.

Analysis of LV wall motion may be performed from these cine images. Regional hypokinesia, dyskinesia of ventricular wall segments can be identified and assessment can be made of the arterial territory involved and the probable site of the lesion can be inferred. The high reproducibility has made this a reference standard for cardiac chamber size and function.[47]

MYOCARDIAL VIABILITY ON MRI

MR Perfusion Imaging

Myocardial perfusion can be assessed in a number of ways. Sequences that provide intrinsic contrast such as blood oxygen level dependent can be used without the need for gadolinium contrast to provide the information regarding the myocardial perfusion.[48] However, the low signal to noise ratio limits its widespread use. Dynamic imaging using gadolinium agents is the method of choice at present for the MRI of ventricular perfusion.

Dynamic First Pass Imaging

Blood flow through the myocardial microcirculation can be evaluated by following the first pass of contrast through the myocardium and mapping the temporal change in intensity or enhancement of myocardium either visually or using complex analysis algorithms. Gradient echo recalled sequences, echo planar imaging and SSFP have been used for dynamic first pass imaging.[49] These imaging sequences are typically 2-dimensional with an in plane resolution of 3 mm. This relatively high resolution in comparison with SPECT allows reliable detection of subendocardial defects.[50]

Perfusion imaging is performed in a resting phase and under a pharmacologic stress (adenosine or dypiramidole). Resting perfusion imaging has low sensitivity in the detection of CAD, though underperfused segments on rest images correlate with nonviable tissue.[51] A resting perfusion defect after an acute myocardial infarction correlates to the severity of the LV functional impairment. Stress perfusion is used to detect significant CAD.[52] The myocardial perfusion reserve is determined by separate scans at stress and at rest.[53] The patients with CAD are identified by the abnormal perfusion reserve. Improvement in perfusion reserve can be used to monitor the success of revascularization.[54] The status of myocardial perfusion is estimated for each of the segments in a 17-segment model[55] of the LV myocardium either by software (quantitative) or by subjective visual interpretation.

In a study by Al-Saadi et al,[53] signal intensity versus time curves were used to calculate myocardial perfusion reserve for each of the segments assigned to the coronary arteries. Perfusion reserve of 1.5 was used as a cut-off value to diagnose a significant stenosis $\geq 75\%$. They reported sensitivity, specificity, and accuracy of 90%, 83%, and 87%, respectively using coronary angiograms as the reference standard. Most studies evaluating the efficacy of various MRI techniques for detecting significant CAD rely on visual interpretation to characterize perfusion defect.[51,52] Perfusion defects are considered to be present if persistent delay in enhancement was noted in at least 3 consecutive temporal images and at least in 2 contiguous images.[51] The rest perfusion study alone gave a sensitivity of 43%, specificity of 89%, and accuracy of 71% for the detection of significant CAD. Stress perfusion alone had a higher sensitivity of 81% and a higher accuracy of 87%.[51] Stress perfusion alone was noted to be more valuable in detecting CAD than rest perfusion images; however, there was no substantial difference when a combined stress and rest images were used when compared with stress alone.[51,52] Nevertheless, rest studies are of value particularly in defining areas with artifact. Better diagnostic accuracy has been reported combining delayed enhancement with stress perfusion.[51,52]

Perfusion studies can identify microvascular dysfunction when there is no apparent hemodynamically significant stenosis in patients with chest pain classified as syndrome X.[56]

Microvascular Obstruction

Microvascular obstruction presents a persistent hypoenhancement over 1 to 3 minutes in the first pass images and indicates poor prognosis after revascularization of the culprit lesion.[57] This lack of perfusion in the reperfused lesion owing to microvascular dysfunction is known as no-reflow phenomenon and presents as a persistent subendocardial defect on the first pass images.

Delayed Hyperenhancement

Most of the gadolinium contrast washes out of the normal myocardium in about 15 minutes. The contrast reaches equilibrium within the fluid compartments of the body. The contrast is predominantly present in the extracellular fluid compartment of the normal myocardium. However, in the infarcted myocardium the extracellular fluid space is increased and the contrast permeates into the injured cell because of membrane damage. Also, the contrast washes out slowly from the infarcted muscle. These effects result in infarcted myocardium appearing hyperenhanced with respect to the rest of normal myocardium.

Inversion recovery gradient echo sequences are used to evaluate for delayed enhancement in the myocardium 10 to 15 minutes after a bolus administration of contrast. Three-dimensional volumetric acquisition with parallel imaging may be performed to cover the entire ventricular myocardium within a single breath hold.[58]

The area of hyperenhancement correlates with the size and location of the infarcted region. Infarction of the myocardium usually begins first in the endocardium and progresses transmurally to the epicardium because the blood supply is from the epicardium toward the endocardium. Because of the high in plane resolution of the inversion recovery sequence, the transmural extent of the hyperenhancement can suggest the extent of necrosis of the myocardium. The transmural extent can be used as a predictor for functional improvement after revascularization. Lower functional recovery can be expected in the segment having a greater transmural hyperenhancement.[59] Because of the higher spatial resolution of MRI when compared with SPECT, smaller subendocardial infarctions can be picked up with greater reliability.

Delayed hyperenhancement is not specific for myocardial infarction. Conditions such as myocarditis, tumor invasion, and cardiomyopathy can show delayed hyperenhancement. Clinical setting, pattern of enhancement, and other findings such as regional wall motion abnormalities, are needed to assign the noted hyperenhancement to myocardial infarction.[49]

Wall motion abnormalities can be caused either by true infarction or by myocardial stunning or hibernation. Absence of delayed hyperenhancement points to the presence of viable myocardium and predicts functional recovery.[59]

Dobutamine Stress Testing by MRI

Important information regarding viability can be obtained evaluation of regional wall motion under

pharmacologic stress. Cine MRI can be performed with infusion of dobutamine to increase the contractility of the myocardium thereby stressing the myocardium. This is analogous to the stress echocardiogram.

The acquisition sequences are similar to those acquired at rest. Gradient recalled echo or SSFP sequences are used with ECG gating and breath hold. At least 3 to 4 short axis, a horizontal and a vertical long axis views are used to cover all the coronary territories. The 17-segment model of the LV is generally used to record and interpret the findings.[55]

Several criteria have been proposed for interpretation of the stress MRI. A resting wall thickness > 5.5 mm or stress-induced systolic wall thickening of at least 1 mm was considered by some as the cut-off for diagnosing viability.[60] A more commonly used definition of viability on stress MRI is an induced response of 2 mm or more in a previously dysfunctional segment. Others use a baseline grading of wall motion (0 to 4 grade scale, 0 = normal and 4 = dyskinesia) and consider the myocardium to be viable if there is an improvement in wall motion of at least 1 grade.[61] In patients with poor acoustic windows to perform a stress echocardiogram, MRI is a good replacement. To improve accuracy, MR tissue tagging has been proposed.[62]

IMAGE FUSION AND COREGISTRATION

MRI provides accurate physiologic data regarding the myocardium. The representation of this data is commonly made on a 17-segment model that assigns each segment to corresponding major coronary arteries. However, this is an arbitrary assignment and may not hold well in all cases because of varied coronary anatomy. This can be important for presurgical decision making. Setser et al[63] used the segmented coronary tree and LV contours from MDCT to coregister with MRI. A total of 17% of the myocardial segments differed in their assignment. Apical lateral, midanterolateral, and apical inferior segments were discordant in most cases. In their study, surgical planning was improved in 83% of the patients because of the additional information provided by the coregistration.

Image fusion and coregistration has been attempted with other combinations of imaging modalities that provide functional and structural information about CAD separately. These include coregistration of SPECT and catheter angiography, SPECT and MDCT coronary angiography.[64,65] Integrated cardiac PET-CT uses hybrid hardware to acquire the structural and functional information in the same sitting.[66]

The functional significance of stenosis can be estimated in a limited way by invasive angiography. For this reason, fractional flow reserve may be performed to gain information regarding the significance of the stenosis.[67] MDCT coronary angiography, optimally, can be expected to perform no better than invasive angiography. Thus the combination of anatomy with functional measures can be expected to be important.

INTEGRATED PET-CT

The 2 imaging modalities each have certain limitations. The diagnostic performance of MDCT decreases substantially in the presence of stents, calcification, high heart rates, and irregular rhythms. Evaluation of distal smaller caliber coronary vasculature cannot be accurately evaluated at present by MDCT. And importantly no data regarding the functional significance of lesions detected on MDCT exist in the current literature to guide further patient management. MDCT does not provide information regarding the perfusion status of the microvascular bed and status of collateral circulation and tends to overestimate the degree of stenosis. Conversely, PET underestimates the true anatomic extent of atherosclerosis in subclinical patients but identifies the critically underperfused territory.[66,68] Stress perfusion imaging by PET provides excellent information regarding the severity of CAD and takes into account the collateral circulation and true physiologic effect of atherosclerosis on myocardial microvascular perfusion. Balanced hypoperfusion from multivessel disease can be a problem. On integration, the complimentary nature of these modalities is expected to minimize their respective individual shortcomings.

Thus PET-CT can provide accurate diagnostic information even in patients with suboptimal MDCT situations and also identify extensive subclinical CAD apart from guiding management with excellent anatomic and physiologic information regarding the culprit lesion.[66,69] However, further studies are needed to establish PET-CT as a single study alternative for evaluation of CAD.

CONCLUSIONS

Noninvasive modalities like MDCT, EBCT, MRI, and PET-CT provide important diagnostic information in the assessment of CAD. However, no single modality gives all the necessary information needed for patient management for the spectrum of CAD. As these noninvasive modalities are complimentary to each other with some providing structural detail and others functional data, a combined approach may be a reliable noninvasive alternative to catheter angiography in future. Coregistration of SPECT and MDCT or MRI and MDCT images may be a useful combination. Alternatively, integrated hardware such as PET-CT allows a combined acquisition in a single sitting. With further advances in hardware and software, these noninvasive imaging modalities can be expected to make a significant contribution toward diagnosing and understanding CAD.

REFERENCES

1. Fenter TC, Naslund MJ, Shah MB, et al. The cost of treating the 10 most prevalent diseases in men 50 years of age or older. *Am J Manag Care*. 2006;12(4 suppl):S90–S98.
2. Kalender WA. X-ray computed tomography. *Phys Med Biol*. 2006;51:R29–R43.
3. Klingenbeck-Regn K, Schaller S, Flohr T, et al. Subsecond multislice computed tomography: basics and applications. *Eur J Radiol*. 1999;31:110–124.

4. Nikolaou K, Flohr T, Knez A, et al. Advances in cardiac CT imaging: 64-slice scanner. *Int J Cardiovasc Imaging*. 2004;20: 535–540.
5. Achenbach S, Ropers D, Kuettner A, et al. Contrast-enhanced coronary artery visualization by dual-source computed tomography—initial experience. *Eur J Radiol*. 2006;57:331–335.
6. Flohr TG, McCollough CH, Bruder H, et al. First performance evaluation of a dual-source CT (DSCT) system. *Eur Radiol*. 2006; 16:256–268.
7. Johnson TR, Nikolaou K, Wintersperger BJ, et al. Dual-source CT cardiac imaging: initial experience. *Eur Radiol*. 2006;16:1409–1415.
8. Gertz SD, Cherukuri P, Bodmann BG, et al. Usefulness of multidetector computed tomography for noninvasive evaluation of coronary arteries in asymptomatic patients. *Am J Cardiol*. 2006; 97:287–293.
9. Pohle K, Achenbach S, Macneill B, et al. Characterization of non-calcified coronary atherosclerotic plaque by multi-detector row CT: comparison to IVUS. *Atherosclerosis*. 2006.
10. 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.
11. Agatston AS, Janowitz WR, Hildner FJ, et al. Quantification of coronary artery calcium using ultrafast computed tomography. *J Am Coll Cardiol*. 1990;15:827–832.
12. Raggi P, Callister TQ, Cooil B, et al. Identification of patients at increased risk of first unheralded acute myocardial infarction by electron-beam computed tomography. *Circulation*. 2000;101: 850–855.
13. O'Rourke RA, Brundage BH, Froelicher VF, et al. 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.
14. 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.
15. Hoffmann U, Moselewski F, Nieman K, et al. Noninvasive assessment of plaque morphology and composition in culprit and stable lesions in acute coronary syndrome and stable lesions in stable angina by multidetector computed tomography. *J Am Coll Cardiol*. 2006;47:1655–1662.
16. Stary HC, Chandler AB, Dinsmore RE, et al. 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.
17. Thompson BH, Stanford W. Imaging of coronary calcification by computed tomography. *J Magn Reson Imaging*. 2004;19:720–733.
18. Becker CR, Nikolaou K, Muders M, et al. Ex vivo coronary atherosclerotic plaque characterization with multi-detector-row CT. *Eur Radiol*. 2003;13:2094–2098.
19. Carrascosa PM, Capunay CM, Garcia-Merletti P, et al. Characterization of coronary atherosclerotic plaques by multidetector computed tomography. *Am J Cardiol*. 2006;97:598–602.
20. 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.
21. Angheloiu GO, Arendt JT, Muller MG, et al. Intrinsic fluorescence and diffuse reflectance spectroscopy identify superficial foam cells in coronary plaques prone to erosion. *Arterioscler Thromb Vasc Biol*. 2006;26:1594–1600.
22. de Paula AR Jr, Sathaiah S. Raman spectroscopy for diagnosis of atherosclerosis: a rapid analysis using neural networks. *Med Eng Phys*. 2005;27:237–244.
23. Raggi P, Thomas GS. Role of computed tomography and perfusion imaging in patients with known or suspected coronary artery disease. *J Nucl Cardiol*. 2006;13:170–175.
24. Schuijf JD, Bax JJ, Shaw LJ, et al. 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.
25. Raff GL, Gallagher MJ, O'Neill WW, et al. Diagnostic accuracy of noninvasive coronary angiography using 64-slice spiral computed tomography. *J Am Coll Cardiol*. 2005;46:552–557.
26. 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.
27. Schroeder S, Kuettner A, Leitritz M, et al. Reliability of differentiating human coronary plaque morphology using contrast-enhanced multislice spiral computed tomography: a comparison with histology. *J Comput Assist Tomogr*. 2004;28:449–454.
28. Nieman K, Cademartiri F, Lemos PA, et al. Reliable noninvasive coronary angiography with fast submillimeter multislice spiral computed tomography. *Circulation*. 2002;106:2051–2054.
29. Nieman K, Rensing BJ, van Geuns RJ, et al. Usefulness of multislice computed tomography for detecting obstructive coronary artery disease. *Am J Cardiol*. 2002;89:913–918.
30. Ehara M, Surmely JF, Kawai M, et al. Diagnostic accuracy of 64-slice computed tomography for detecting angiographically significant coronary artery stenosis in an unselected consecutive patient population: comparison with conventional invasive angiography. *Circ J*. 2006;70:564–571.
31. Nikolaou K, Knez A, Rist C, et al. Accuracy of 64-MDCT in the diagnosis of ischemic heart disease. *AJR Am J Roentgenol*. 2006; 187:111–117.
32. George RT, Silva C, Cordeiro MA, et al. Multidetector computed tomography myocardial perfusion imaging during adenosine stress. *J Am Coll Cardiol*. 2006;48:153–160.
33. Gerber BL, Belge B, Legros GJ, et al. Characterization of acute and chronic myocardial infarcts by multidetector computed tomography: comparison with contrast-enhanced magnetic resonance. *Circulation*. 2006;113:823–833.
34. Koyama Y, Mochizuki T, Higaki J. Computed tomography assessment of myocardial perfusion, viability, and function. *J Magn Reson Imaging*. 2004;19:800–815.
35. Nikolaou K, Sanz J, Poon M, et al. Assessment of myocardial perfusion and viability from routine contrast-enhanced 16-detector-row computed tomography of the heart: preliminary results. *Eur Radiol*. 2005;15:864–871.
36. Sakuma H, Ichikawa Y, Suzawa N, et al. Assessment of coronary arteries with total study time of less than 30 minutes by using whole-heart coronary MR angiography. *Radiology*. 2005;237: 316–321.
37. Dewey M, Teige F, Schnapauff D, et al. Combination of free-breathing and breathhold steady-state free precession magnetic resonance angiography for detection of coronary artery stenoses. *J Magn Reson Imaging*. 2006;23:674–681.
38. Fayad ZA, Fuster V, Fallon JT, et al. Noninvasive in vivo human coronary artery lumen and wall imaging using black-blood magnetic resonance imaging. *Circulation*. 2000;102:506–510.
39. Stuber M, Botnar RM, Danias PG, et al. Double-oblique free-breathing high resolution three-dimensional coronary magnetic resonance angiography. *J Am Coll Cardiol*. 1999;34:524–531.
40. Kim WY, Stuber M, Bornert P, et al. Three-dimensional black-blood cardiac magnetic resonance coronary vessel wall imaging detects positive arterial remodeling in patients with nonsignificant coronary artery disease. *Circulation*. 2002;106:296–299.
41. Botnar RM, Kim WY, Bornert P, et al. 3D coronary vessel wall imaging utilizing a local inversion technique with spiral image acquisition. *Magn Reson Med*. 2001;46:848–854.
42. Barkhausen J, Ebert W, Heyer C, et al. Detection of atherosclerotic plaque with Gadofluorine-enhanced magnetic resonance imaging. *Circulation*. 2003;108:605–609.
43. Botnar RM, Buecker A, Wiethoff AJ, et al. In vivo magnetic resonance imaging of coronary thrombosis using a fibrin-binding molecular magnetic resonance contrast agent. *Circulation*. 2004; 110:1463–1466.

44. Larose E, Yeghiazarians Y, Libby P, et al. Characterization of human atherosclerotic plaques by intravascular magnetic resonance imaging. *Circulation*. 2005;112:2324–2331.
45. Carr JC, Simonetti O, Bundy J, et al. Cine MR angiography of the heart with segmented true fast imaging with steady-state precession. *Radiology*. 2001;219:828–834.
46. Alfakih K, Reid S, Jones T, et al. Assessment of ventricular function and mass by cardiac magnetic resonance imaging. *Eur Radiol*. 2004;14:1813–1822.
47. Bellenger NG, Davies LC, Francis JM, et al. Reduction in sample size for studies of remodeling in heart failure by the use of cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2000;2:271–278.
48. Egred M, Al-Mohammad A, Waiter GD, et al. Detection of scarred and viable myocardium using a new magnetic resonance imaging technique: blood oxygen level dependent (BOLD) MRI. *Heart*. 2003;89:738–744.
49. Edelman RR. Contrast-enhanced MR imaging of the heart: overview of the literature. *Radiology*. 2004;232:653–668.
50. Wagner A, Mahrholdt H, Holly TA, et al. Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: an imaging study. *Lancet*. 2003;361:374–379.
51. Cury RC, Cattani CA, Gabure LA, et al. Diagnostic performance of stress perfusion and delayed-enhancement MR imaging in patients with coronary artery disease. *Radiology*. 2006;240:39–45.
52. Klem I, Heitner JF, Shah DJ, et al. Improved detection of coronary artery disease by stress perfusion cardiovascular magnetic resonance with the use of delayed enhancement infarction imaging. *J Am Coll Cardiol*. 2006;47:1630–1638.
53. Al-Saadi N, Nagel E, Gross M, et al. Noninvasive detection of myocardial ischemia from perfusion reserve based on cardiovascular magnetic resonance. *Circulation*. 2000;101:1379–1383.
54. Al-Saadi N, Nagel E, Gross M, et al. Improvement of myocardial perfusion reserve early after coronary intervention: assessment with cardiac magnetic resonance imaging. *J Am Coll Cardiol*. 2000; 36:1557–1564.
55. 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;105:539–542.
56. Panting JR, Gatehouse PD, Yang GZ, et al. Abnormal subendocardial perfusion in cardiac syndrome X detected by cardiovascular magnetic resonance imaging. *N Engl J Med*. 2002;346: 1948–1953.
57. Ito H, Maruyama A, Iwakura K, et al. Clinical implications of the 'no reflow' phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. *Circulation*. 1996;93:223–228.
58. Dewey M, Laule M, Taupitz M, et al. Myocardial viability: assessment with three-dimensional MR imaging in pigs and patients. *Radiology*. 2006;239:703–709.
59. 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;343:1445–1453.
60. Baer FM, Voth E, LaRosee K, et al. Comparison of dobutamine transesophageal echocardiography and dobutamine magnetic resonance imaging for detection of residual myocardial viability. *Am J Cardiol*. 1996;78:415–419.
61. Schmidt A, Wu KC. MRI assessment of myocardial viability. *Semin Ultrasound CT MR*. 2006;27:11–19.
62. Sayad DE, Willett DL, Hundley WG, et al. Dobutamine magnetic resonance imaging with myocardial tagging quantitatively predicts improvement in regional function after revascularization. *Am J Cardiol*. 1998;82:1149–1151, A10.
63. Setser RM, O'TP, Smedira NG, et al. Coregistered MR imaging myocardial viability maps and multi-detector row CT coronary angiography displays for surgical revascularization planning: initial experience. *Radiology*. 2005;237:465–473.
64. Higashino H, Mochizuki T, Haraikawa T, et al. Image fusion of coronary tree and regional cardiac function image using multislice computed tomography. *Circ J*. 2006;70:105–109.
65. Nakaura T, Utsunomiya D, Shiraishi S, et al. Three-dimensional cardiac image fusion using new CT angiography and SPECT methods. *AJR Am J Roentgenol*. 2005;185:1554–1557.
66. Di Carli MF, Dorbala S, Hachamovitch R. Integrated cardiac PET-CT for the diagnosis and management of CAD. *J Nucl Cardiol*. 2006;13:139–144.
67. Pijls NH, De Bruyne B, Peels K, et al. Measurement of fractional flow reserve to assess the functional severity of coronary-artery stenoses. *N Engl J Med*. 1996;334:1703–1708.
68. Yoshinaga K, Katoh C, Noriyasu K, et al. Reduction of coronary flow reserve in areas with and without ischemia on stress perfusion imaging in patients with coronary artery disease: a study using oxygen 15-labeled water PET. *J Nucl Cardiol*. 2003;10:275–283.
69. Di Carli MF, Dorbala S. Integrated PET/CT for cardiac imaging. *Q J Nucl Med Mol Imaging*. 2006;50:44–52.

ERRATUM

In "Notes From the 2005 Annual Meeting of the Korean Society of Thoracic Radiology" (*J Thorac Imaging*. 2006;21:245–249), the authors' names are displayed incorrectly. The correct format for the authors' names is as follows:

Goo, Jin Mo; Im, Jung-Gi; Lee, Kyung Soo.

We regret the error.

1. Goo JM, Im JG, Lee KS. Notes From the 2005 Annual Meeting of the Korean Society of Thoracic Radiology. *J Thorac Imaging*. 2006;21:245–249.

