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Technical Notes

Adenosine-stress dynamic myocardial volume perfusion imaging with second generation dual-source computed tomography: Concepts and first experiences

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Abstract. Recent research suggests that multidetector-row CT may have potential as a standalone modality for integrative imaging of coronary heart disease, including the assessment of the myocardial blood supply. However, the technical prerequisites for volumetric, time-resolved imaging of the passage of a contrast medium bolus through the myocardium have only been met with latest generation wide-detector CT scanners. Second-generation dual-source CT enables performing electrocardiographic (ECG)–synchronized dynamic myocardial perfusion imaging by a dedicated "shuttle" mode. With this acquisition mode, image data can be acquired during contrast medium infusion at 2 alternating table positions with the table shuttling back and forth between the 2 positions covering a 73-mm anatomic volume. We applied this acquisition technique for detecting differences in perfusion patterns between healthy and diseased myocardium and for quantifying myocardial blood flow under adenosine stress in 3 patients with coronary heart disease. According to our initial experience, the addition of adenosine stress volumetric dynamic CT perfusion to a cardiac CT protocol comprising coronary artery calcium quantification, prospectively ECG-triggered coronary CT angiography, and delayed acquisition appears promising for the comprehensive assessment of coronary artery luminal integrity, cardiac function, perfusion, and viability with a single modality.
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Introduction

Rapid evolution of multidetector-row computed tomography (MDCT) technology continues to enhance the role of this technique in the work-up of coronary artery disease. Recently, MDCT has been proposed as a stand-alone modality for integrative imaging of coronary heart disease,

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ie, the comprehensive assessment of cardiac anatomy, function, perfusion, and viability. Single-energy[1–3] and dual-energy CT techniques based on dual-source CT (DSCT) technology[4,5] have been proposed for this purpose. However, detection of myocardial perfusion defects by using the standard spiral acquisition mode of conventional MDCT relies on static image acquisition of the heart during contrast material infusion and cannot dynamically evaluate the time-resolved passage of a contrast medium bolus through the myocardium. This approach, therefore, cannot capture all phases of myocardial contrast kinetics and thus may provide only limited information about the extent of microvascular obstruction.

Recently, second-generation DSCT was introduced, which comprises two 128-section detectors that provide greater detector coverage compared with first-generation DSCT.[6,7] This system provides the ability of performing dynamic perfusion imaging by a dedicated "shuttle" mode that captures the passage of a contrast medium bolus through the myocardium. We describe the technical concepts of this method and our first clinical experience with this technique for performing dynamic contrast-enhanced myocardial perfusion imaging under pharmacologically induced stress.

Technical methods

In the shuttle mode of second-generation DSCT (SOMATOM Definition Flash;, Siemens Healthcare, Forchheim, Germany) the table shuttles back and forth during image acquisition between 2 adjacent anatomic positions with acceleration of 300 mm/s^2. This acquisition mode can also be performed in an electrocardiographic (ECG)–triggered fashion. Given a detector width of 38 mm, and a 10% overlap between both acquisition ranges, the anatomic coverage of this imaging technique is 73 mm. We applied this acquisition technique for the purpose of myocardial perfusion assessment during contrast medium infusion and under pharmacologic stress as a component of a comprehensive CT-based protocol for integrative imaging of cardiac structure, function, perfusion, and viability (Fig. 1).

Our initial experience is based on 3 consecutive subjects who had been referred for clinically indicated nuclear myocardial perfusion imaging with the use of single-photon emission CT (SPECT) for assessment of coronary artery disease. With approval by our institutional review board all 3 subjects underwent stress-perfusion and delayed enhancement cardiac CT as well as stress/rest perfusion and delayed enhancement magnetic resonance imaging (MRI). All imaging studies were performed within a week.

CT assessment started with single heartbeat CT calcium scoring with the use of the following parameters: 2 × 128 × 0.6-mm sections, 280-millisecond gantry rotation time, 120-kV tube potential, and 73 mAs per rotation tube current time product. Subsequently, prospectively ECG-triggered coronary CT angiography was performed. Contrast medium enhancement was achieved with a triphasic injection protocol with injection of 70 mL of pure, undiluted iodinated contrast material (Iopromide, Ultravist 370 mg/mL; Bayer-Schering Pharma AG, Berlin, Germany) followed by a constant volume of 50 mL of a 70%:30% saline-to-contrast medium mixture and 30 mL of pure saline, all injected at 6 mL/s through an 18G intravenous antecubital catheter with a dual-syringe injector (Stellant D; Medrad, Indianola, PA). The study acquisition delay time was estimated by injection of a 15-mL contrast medium test bolus at 6 mL/s, followed by 50 mL of saline. The actual delay time was calculated as the time of peak contrast medium attenuation in a region of interest in the ascending aorta plus 4 seconds. For prospectively ECG-triggered coronary CT angiography, acquisition parameters were 2 × 128 × 0.6-mm sections, 280-millisecond gantry rotation time, 120-kV tube potential, and 320 mAs per rotation tube current time product. Acquisition was craniocaudal from above the origin of the coronary arteries to below the dome of the diaphragm. Adaptive prospective ECG-triggering was used with the full radiation dose window set at 70% of the R-R interval in 2 patients with heart rates $\leq$70 beats/min, and 40% of the R-R interval in 1 patient with a heart rate of $>$70 beats/min. Reduced dose (25% of the nominal tube current) was applied between 30% and 90% of the R-R interval to obtain functional information. Coronary artery evaluation data sets were reconstructed with the use of 0.75-mm section thickness and 0.3-mm reconstruction increment at 40% or 70% R-R interval, depending on the heart rate. An additional reconstruction

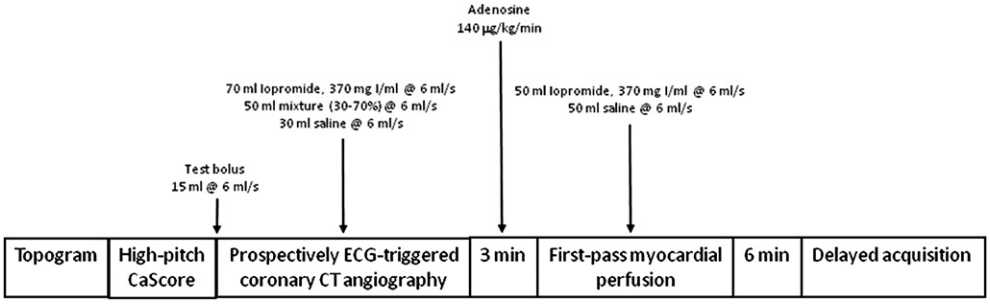


Figure 1 CT acquisition protocol.

was performed during systole at 250 milliseconds after the R peak to plan the coverage range for the myocardial perfusion acquisition.

Myocardial perfusion imaging was performed in the shuttle mode during peak adenosine (140 μg/kg/min Adenoscan; Astellas Pharma Inc, Tokyo, Japan) stress. Data were acquired during end systole (250 milliseconds after the R peak). The anatomic extension of the scan range was planned, based on the systolic reconstruction of the previously performed coronary CT angiography such as to encompass most of the myocardium, starting at the anterior and superior portions of the left ventricle and extending to the inferior wall along the 73-mm coverage. The study range for the volumetric myocardial perfusion data acquisition was planned by the CT technologist, without knowledge of the SPECT or cardiac MRI results. Image acquisition parameters were 100-kV tube voltage and 300 mAs. The image acquisition sequence was initiated 4 seconds before the arrival of the contrast medium bolus front as determined by the initial test bolus injection to ensure baseline acquisition of noncontrast images before the onset of first-pass perfusion. Myocardial perfusion studies were contrast medium enhanced with 50 mL of contrast medium, followed by 50 mL of saline, injected at 6 mL/s. Including test bolus acquisition and coronary CT angiography, each patient thus received a total volume of 150 mL of contrast medium and 135 mL of saline. Studies were obtained during end-inspiration with a standardized acquisition time of 30 seconds (Fig. 2). If patients could not hold their breath for 30 seconds, they were instructed to slowly release their breath and to continue breathing shallowly. Images were reconstructed with 3-mm slice width every 2 mm with a medium sharpness convolution algorithm.

Finally, delayed enhancement studies were performed 6 minutes after perfusion imaging with the use of a regular prospectively ECG-triggered mode with image acquisition at 70% of the R-R interval at 80 kV and 320 mAs.

Cardiac MRI acquisition protocol

Cardiac MRI studies were performed on a 1.5-T system (Magnetom Avanto; Siemens, Erlangen, Germany). Stress perfusion MRI was performed during peak adenosine stress with the use of steady-state free precession (SSFP; TrueFISP; Siemens) perfusion sequences. Three short axis sections representative of basal, mid, and apical myocardial portions were acquired. Ten minutes after stress perfusion, rest perfusion images were obtained with the same acquisition protocol. Contrast enhancement during stress and rest perfusion MRI was achieved with gadopentetate dimeglumine (0.1 mmol/kg total; Magnevist; Bayer-Schering, Berlin, Germany), injected at 4 mL/s. In addition, functional analysis was performed with retrospectively ECG-gated 8-mm slice thickness cine loops in short- and long-axis views with the use of a SSFP sequence. Finally, delayed enhancement studies were obtained with the use of a phase-sensitive inversion-recovery gradient echo SSFP sequence 15 minutes after contrast medium administration.

SPECT acquisition protocol

Rest/stress SPECT myocardial perfusion imaging was performed after intravenous administration of Tetrofosmin (99mTechnetium) in a 1-day protocol. An activity of 370 MBq at rest and 1110 MBq immediately after peak stress was injected. Ergometric stress testing was performed with the use of the Bruce treadmill protocol. A triple-head camera system (Vertex 60+, collimator VXGP; Philips, Cleveland, OH) with attenuation correction was used for ECG-gated data acquisition. Short- and long-axis images were reconstructed.

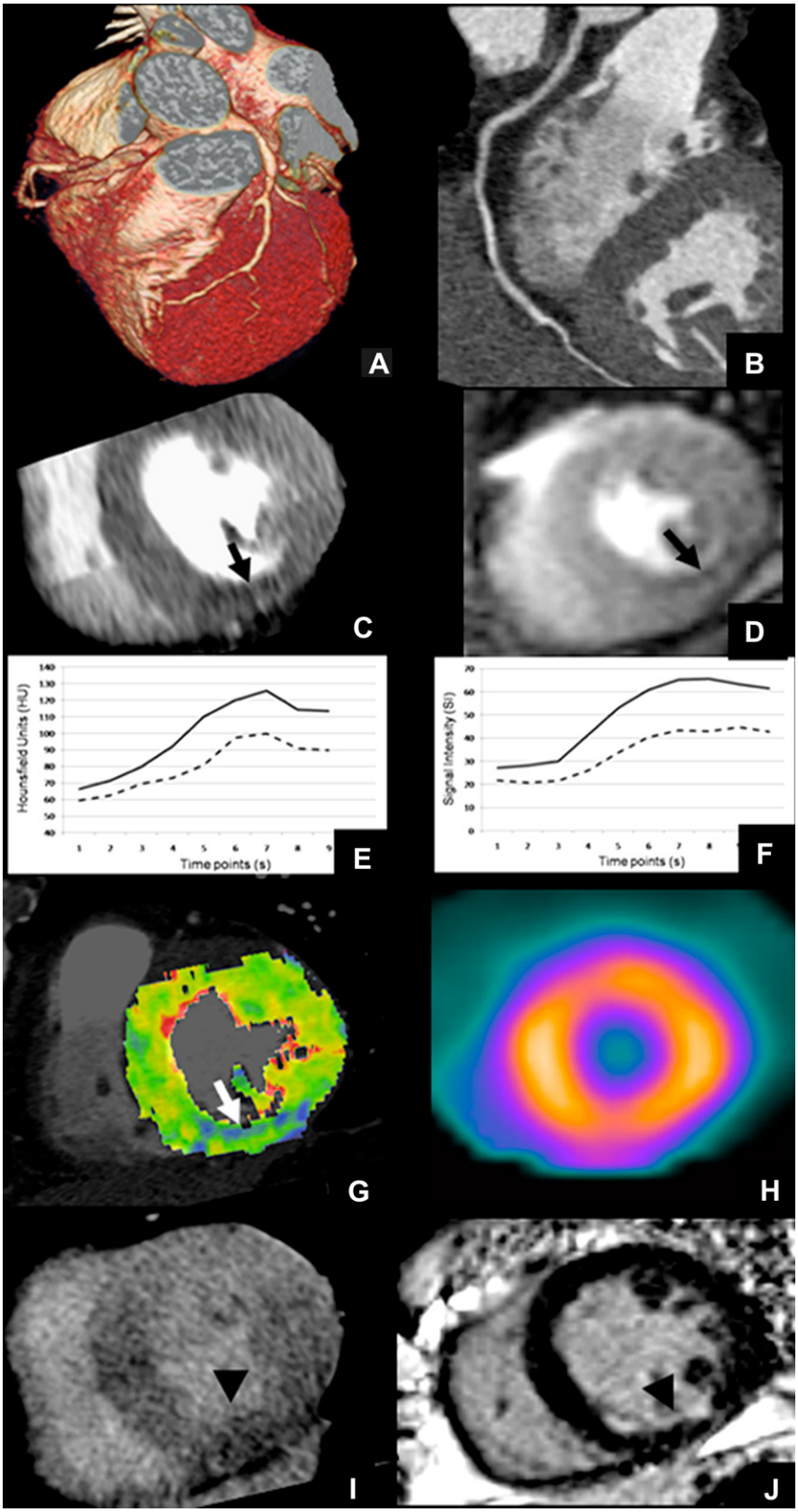
Perfusion data analysis

All myocardial perfusion imaging studies were evaluated with the 16-segment American Heart Association model.[8] Rest/stress SPECT and MRI studies were interpreted for fixed and reversible perfusion defects. Dynamic stress CT perfusion and MRI studies were interpreted in conjunction with delayed enhancement CT and MRI viability scans. On both CT and MRI, homogeneously perfused myocardium during adenosine stress that did not show enhancement on delayed images was classified as normal myocardium. Reversible ischemia was diagnosed when myocardial hypoperfusion visually lasted >6 heartbeats under adenosine stress and showed no enhancement on delayed scans. Myocardial perfusion defects were classified as fixed if the hypoperfusion lasted >6 heartbeats under adenosine stress and viability scans showed delayed enhancement.[9]

Semiquantitative perfusion analysis was performed on 10-mm slice thickness short-axis multiplanar reformats of the stress cardiac CT acquisitions representative of basal, mid, and apical portions of the left ventricular myocardium and on the 3 short-axis sections acquired at stress perfusion MRI. A commercially available software tool (Argus; Siemens) was used to calculate the "myocardial-to-left ventricular upslope index"; this is the upslope of the signal intensity over time curve from unenhanced myocardium to maximum signal intensity during the myocardial first pass of the contrast agent.[10] This index was normalized to blood pool signal intensity curves.[10,11]

Case 1

A 56-year-old man with history of inferior myocardial infarction and right coronary artery angioplasty 8 years earlier presented with recurrent atypical chest pain. Despite the history of remote myocardial infarction, stress/rest SPECT was interpreted as normal, without perfusion defects. Stress-perfusion and delayed enhancement cardiac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Hounsfield Units (HU)
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Figure 2

MRI (Fig. 2) showed preserved left ventricular function with chronic inferior subendocardial infarction. No peri-infarct ischemia was noted. Coronary CT angiography showed mild luminal irregularities in the right coronary artery, without significant obstructive disease. Stress first-pass myocardial perfusion imaging with CT showed an inferior subendocardial perfusion defect, as confirmed by MRI, but not seen on SPECT. Total dose length product (DLP) from all 4 cardiac CT studies was 1177 mGy cm (calcium score, 33 mGy cm; coronary CT angiogram, 254 mGy cm; dynamic perfusion, 795 mGy cm; delayed acquisition, 95 mGy cm).

Case 2

A 64 year-old man was admitted with shoulder pain suspicious for cardiac origin. The patient had a remote history of inferolateral myocardial infarction 13 years earlier treated with right coronary artery angioplasty. Stress/rest SPECT showed inferolateral fixed defect encompassing 25% of the left ventricular myocardium with peri-infarct ischemia. Stress-perfusion and delayed enhancement cardiac MRI (Fig. 3) showed inferolateral akinesis, inferior/inferoseptal perfusion defects, and delayed enhancement, representing chronic transmural myocardial infarction but no peri-infarct ischemia. Coronary CT angiography showed proximal ectasia with distal occlusion of the right coronary artery and inferolateral akinesis. Stress first-pass myocardial perfusion and delayed enhancement imaging with CT showed perfusion defects of the inferior, inferoseptal, and inferolateral segments and no peri-infarct ischemia. Total DLP from all 4 cardiac CT studies was 1127 mGy cm (calcium score, 28 mGy cm; coronary CT angiogram, 398 mGy cm; dynamic perfusion, 604 mGy cm; delayed acquisition, 97 mGy cm).

Case 3

A 70-year-old man with history of previous coronary artery bypass surgery (left internal mammary artery to left anterior descending coronary artery, radial arterial graft to circumflex coronary artery, and saphenous vein graft to distal right coronary artery) and prior right coronary artery stent placement was admitted for occasional episodes of chest pain suggestive of cardiac origin. Stress/rest SPECT was performed, which showed anterior ischemia. Cine-MRI sequences showed anterior and septal hypokinesis. Stress perfusion MRI (Fig. 4) showed perfusion defect of the anterior and septal mid-ventricular segments. Viability imaging showed delayed enhancement, representing chronic

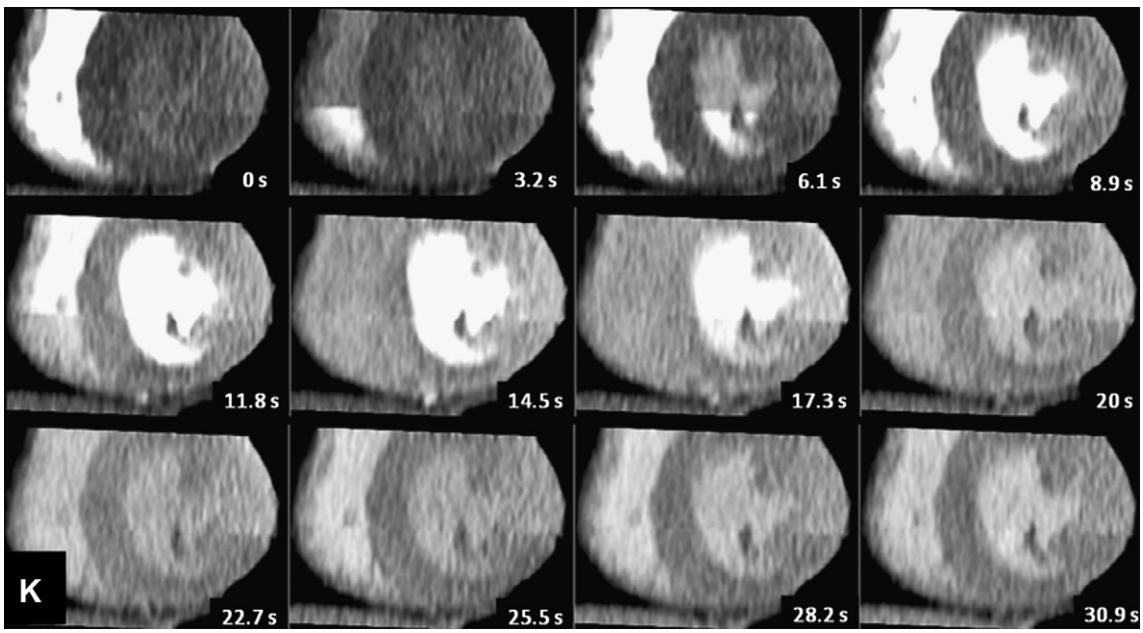

Figure 2 (*continued*). A 56-year-old man with past history of inferior myocardial infarction and right coronary artery angioplasty. Volume-rendered coronary CT angiography (**A**). Curved multiplanar reformat of the right coronary artery (**B**) shows mild luminal irregularities without significant obstructive disease. Visual assessment of stress dynamic CT perfusion (**C**) and stress MRI (**D**) show hypoperfusion in the inferior wall (*arrows*). Semiquantitative analysis shows comparable myocardial perfusion curves for CT (**E**) and MRI (**F**) with decreased perfusion in the abnormal inferior myocardium (*dashed lines*) compared with normal-appearing anterior myocardium (*solid lines*). Absolute myocardial blood flow (MBF) images (**G**) show the perfusion defects visualized during first-pass perfusion (*arrow*) but not detected on SPECT (**H**), which correspond to chronic inferior myocardial infarction (*arrowheads*), confirmed on delayed enhancement CT (**I**) and MRI (**J**). (**K**) Shown are short-axis mid-ventricular reformats across 12 time points (030.9 seconds) of the dynamic contrast-enhanced myocardial volume CT perfusion imaging sequence which form the underlying source data for performing CT myocardial perfusion analysis (**C, E, G**).

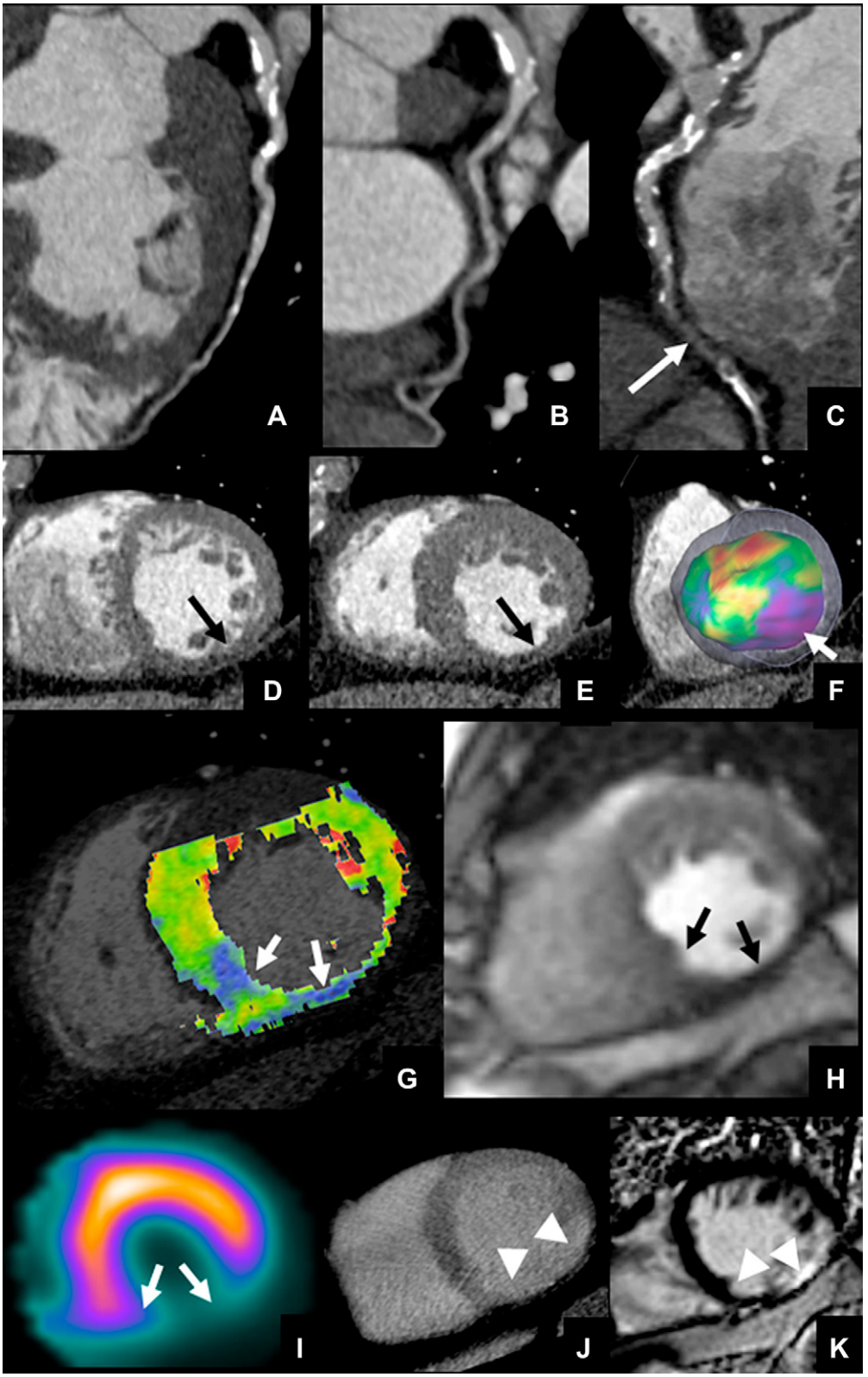

Figure 3 A 64-year-old man with past history of inferolateral myocardial infarction. Curved multiplanar reformats of the left anterior descending (**A**), circumflex (**B**), and right (**C**) coronary arteries. Coronary CT angiography shows distal right coronary artery occlusion (*arrow* in **C**). Functional analysis shows inferolateral akinesis (*arrows* in **D**, **E**, and **F**). First-pass stress perfusion CT (**G**) shows inferior and inferoseptal perfusion defect (*arrows*), confirmed on MRI (**H**) and SPECT (**I**). This perfusion defect corresponds to known chronic infarction, as confirmed by delayed enhancement CT (**J**) and MRI (**K**) (*arrowheads*).

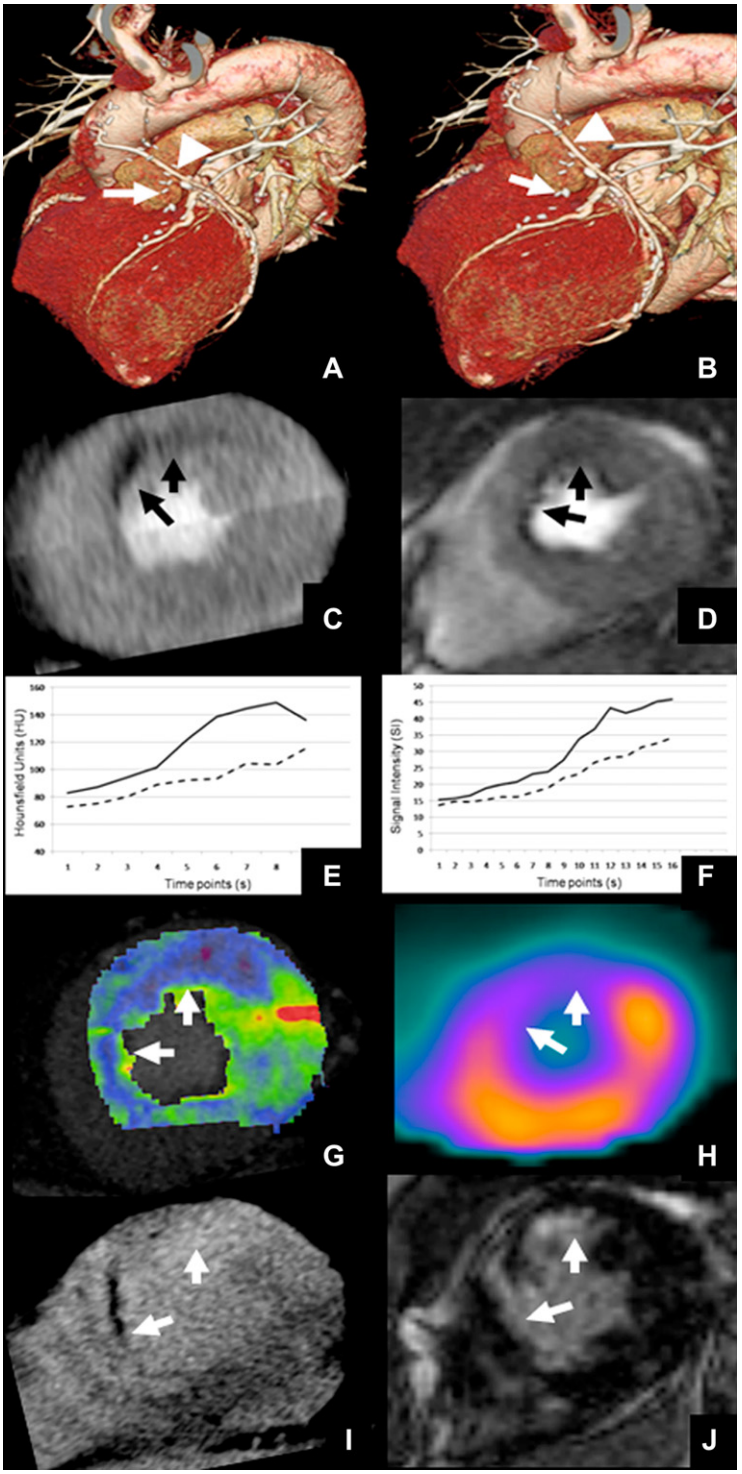

Figure 4 A 70-year-old man with history of previous coronary artery bypass surgery and prior right coronary artery stent placement. Volume-rendered display (**A, B**) of coronary CT angiography study shows occlusion of left internal mammary artery graft to left anterior descending coronary artery (*arrows*) with patent graft to circumflex coronary artery (*arrowheads*). First-pass stress perfusion CT (**C**) and MRI (**D**) show transmural perfusion defects of the anterior and septal mid-ventricular segments (*arrows*). Semiquantitative analysis shows analogous myocardial signal intensity upslope for CT (**E**) and MRI (**F**) in the hypoperfused anterior and septal myocardium (*dashed lines*) and remote normal-appearing myocardium (*solid lines*). Absolute MBF (**G**) and SPECT (**H**) also show the perfusion defects visualized during first-pass perfusion (*arrows*), which correspond to known chronic anterior and septal myocardial infarction, as established by delayed enhancement CT (**I**) and MRI (**J**) (*arrows*).

myocardial infarction of the anterior and septal myocardium. Coronary CT angiography of the entire thorax showed status post coronary artery bypass graft surgery with patent grafts to the posterior descending artery and circumflex coronary artery and occlusion of the left internal mammary artery to left anterior descending coronary artery graft. Stress first-pass myocardial perfusion imaging with CT showed perfusion defect of the anterior and septal mid-ventricular segments in agreement with stress MRI and SPECT studies. Total DLP from all 4 cardiac CT studies, including coronary CT angiography of the entire thorax, was 1463 mGy cm (calcium score, 24 mGy cm; coronary CT angiogram, 668 mGy cm; dynamic perfusion, 621 mGy cm; delayed acquisition, 150 mGy cm).

Discussion

We provide the first description of the technical feasibility of performing dynamic contrast-enhanced myocardial volume perfusion imaging with mechanical CT for detecting perfusion defects and myocardial infarction in patients with coronary heart disease. As shown in the cases presented here, this technique can show subendocardial infarction not seen on SPECT but confirmed by MRI and can detect ischemia in good correlation with stress-perfusion MRI and SPECT.

Although other methods are preferred for obtaining physiologic information, CT is unique in its ability to noninvasively interrogate the coronary arteries. Accordingly, recent developments in cardiac CT imaging have focused on the integrative assessment of coronary heart disease, with the goal of combining coronary artery stenosis detection with the assessment of the hemodynamic effect of lesions on cardiac function, perfusion, and viability with a single modality.[1–5,12] However, when applied for the detection of myocardial perfusion deficits, previous CT-based approaches merely provide a one-time static snapshot of the state of the myocardial blood supply during the time the respective portion of the myocardium is imaged, even when wide area detector scanners[3] are used. With narrower detector CT systems, more cranial portions of the heart are imaged during a different perfusion phase than the most caudal portions, because contrast material continues passing through the heart and the myocardium during spiral CT acquisition. Hence, these techniques may have limitations for showing the true hemodynamic significance of coronary artery stenosis on myocardial perfusion.[13]

Visualization of first-pass myocardial perfusion relies on time-resolved image acquisition during contrast medium infusion. Animal experiments have shown the feasibility of this concept with MDCT[14] and have shown potential for visual and semiquantitative assessment of first-pass myocardial perfusion, albeit image acquisition was restricted to a single transaxial anatomical level because of limitations in detector coverage. For this approach to be clinically viable in patients, greater volume coverage along the z-axis of the heart is desirable along with high temporal resolution, if the effect of pharmaceutical stress on myocardial perfusion is to be evaluated. These prerequisites have only recently been fulfilled with the introduction of broad detector array CT systems[3,15,16] and second-generation DSCT[6,7]; however, these instruments have hitherto not been applied for the purpose of time-resolved myocardial perfusion imaging in actual patients with coronary heart disease. With the technique introduced here, greater volume coverage, ie, 73 mm, along the z-axis of the heart is accomplished by acquiring perfusion data at 2 alternating table positions in ECG-triggered mode during end systole with the table shuttling back and forth between the 2 positions.

As shown in the cases presented here, dynamic first-pass myocardial perfusion CT may provide results comparable to MRI and can show subendocardial perfusion defects not seen on SPECT, thus overcoming one of the limitations of this latter test. Accordingly, CT dynamic first-pass perfusion imaging performed with the technique introduced here combines high spatial and temporal resolutions in a similar fashion as MRI and may allow obtaining comparable results for estimation of myocardial perfusion. The tissue kinetics of iodine contrast agents and gadolinium chelates are similar.[17] However, different from perfusion MRI with gadolinium-based contrast agents, there exists a linear relationship between myocardial contrast medium enhancement and iodine concentration, therefore potentially enabling the direct quantification of myocardial blood flow with CT.[14]

Our method has several limitations. This technical note describes our preliminary experience based on only 3 patients with known history of coronary heart disease, and much larger studies are needed to determine the clinical utility of our approach. Radiation exposure still remains of concern with dynamic myocardial first-pass CT perfusion imaging. However, according to our preliminary experience the total radiation exposure from all 4 CT acquisitions that we performed in each patient is within the range of values described for conventional retrospectively ECG-gated coronary CT angiography[18] and is equivalent to the radiation dose received during thallium SPECT imaging.[19] Should the routine clinical implementation of this technique materialize, radiation savings could be realized by decreasing the need for nuclear myocardial perfusion imaging and invasive coronary angiography, the combination of which is traditionally necessary to obtain comparable information. The total injected volume (150 mL) of iodinated contrast medium is higher than what has become routine with recent generation MDCT systems, but it is not excessive compared with traditional body CT applications. Further improvements in CT technology should aim at providing greater coverage along the z-axis to approach that of latest generation single-source CT systems.[15] Although our 73-mm coverage may suffice for obtaining perfusion data on all vascular territories of most hearts during systole,

this requires exact planning of the anatomic acquisition range, and changes in patient positioning or inspiration level can result in incomplete coverage of portions of the myocardium. Finally, similar to current trends in nuclear[20] and MRI myocardial perfusion imaging,[21] we performed stress perfusion CT only, without image acquisition during rest. Accordingly, based on stress CT imaging alone, we cannot differentiate between fixed and reversible perfusion defects. However, the differentiation between infarct and ischemia is enabled by the addition of delayed enhancement CT and possibly by performance of prospectively ECG-triggered coronary CT angiography before stress perfusion, which may serve as a surrogate in lieu of a dedicated rest perfusion acquisition.

Despite these limitations we demonstrate the feasibility of our CT-based approach for integrative imaging of coronary heart disease, enabling comprehensive assessment of coronary artery luminal integrity, cardiac function, perfusion, and viability with a single modality.

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