Coronary CTA versus cardiac catheterization: Where do we stand today?

Invasive angiography excels at “lumenography,” but coronary CTA addresses the clinical questions that patients and physicians most want answered.

James K. Min, MD
Dr. Min is the Director of Cardiac CT, Weill Medical College of Cornell University, New York-Presbyterian Hospital, New York, NY.

If coronary computed tomographic angiography (CTA) were compared with invasive angiography on the basis of temporal and spatial resolution alone, coronary CTA would be forced to concede defeat. Whereas invasive angiography has a temporal resolution of just 33 msec, CTA has a temporal resolution of approximately 175 msec. Invasive angiography is superior to 16- and 64-slice coronary CTA in spatial resolution as well: 0.20 to 0.25 mm versus 0.4 to 0.8 mm, respectively.

CTA is capable of much more than “lumenography,” however, and its unique strengths are providing new insight into the evaluation of patients with suspected coronary artery disease (CAD). This article will discuss the role of coronary CTA in the evaluation of coronary plaque—not only obstructive plaque (Figure 1) but also nonobstructive plaque and plaque composition. Underscoring the discussion will be the concept of clinically relevant plaque and the potential of coronary CTA to guide diagnosis, treatment, and prognostication in a way that is meaningful to patients and physicians.

Obstructive plaque
During approximately the last 5 years, hundreds of papers have been published that compare coronary CTA and invasive angiography for the detection of CAD. A meta-analysis by van der Zaag-Loonen et al focused on just 15 studies, which were selected on the basis of study design, homogeneity of the patient population (including the absence of participants with known coronary disease or prior coronary interventions), and completeness and interpretability of published data.

The studies were conducted on 4- and 16-slice CT scanners and enrolled a total of 944 intermediate-risk patients. Disease prevalence averaged 59% (range 31% to 81%) and was lower in studies that used consecutive enrollment (52%), as compared with those that used more selective enrollment (72%; \( P = 0.008 \)).

All but 2 studies defined obstructive coronary disease as >50% stenosis on quantitative coronary angiography, resulting in a pooled sensitivity for obstructive stenosis of nearly 90% for coronary CTA. Studies conducted on 16-slice scanners reported more homogenous results than did those conducted on 4-slice CT scanners; similarly, studies that enrolled patients with a lower prevalence of disease reported more homogeneous findings than did those that enrolled patients with a higher prevalence of disease.

Whether the results of this meta-analysis are meaningful in clinical practice is open to question. As many as 28% of patients were excluded from its constituent studies on the basis of a high calcium score, a high heart rate, or other factors. In addition, up to 32% of coronary segments were considered unassessable, and, in many cases, these segments were not included in calculating the accuracy of coronary CTA. As a result, the authors concluded that contrast-enhanced multidetector CT using 4- or 16-slice scanners was not sufficiently sensitive to rule out coronary artery stenosis.

A recent analysis that included 64-slice CT was more optimistic. Stein et al found that the number of evaluable segments increased with the number of detector rows, such that 78% of segments were evaluable on a 4-slice CT scanner, 91% on a 16-slice CT scanner, and 100% on a 64-slice CT scanner. Moreover, a segment-based analysis of sensitivity and specificity for the detection of obstructive coronary disease showed a similar trend, with accuracy improving as the number of detector rows increased. Specifically, segment sensitivity improved from 83% on a 4-slice CT scanner to 88% on a 16-slice scanner and 94% on a 64-slice scanner. Similarly, segment specificity improved from 93% to 95% to 97%, respectively.

Rather than focusing on specific coronary segments, it is often useful to think of the patient in totality. A patient-based analysis by the same authors showed that 64-slice CT had a sensitivity and specificity of 100% for the detection of any significant coronary stenosis. By comparison, 4- and 16-slice CT had a 95% sensitivity and 84% specificity. Other studies of 64-slice CT confirm its high per-patient sensitivity (85% to 95%), specificity (96% to 98%), and negative-predictive value (93% to 100%).

Contrast Use in Cardiac CTA Applications

SUPPLEMENT TO APPLIED RADIOLoGY® www.appliedradiology.com

December 2006
One of the best studies of 64-detector-row CT, reported by Raff et al., examined 70 patients scheduled to undergo cardiac catheterization, evaluating the diagnostic accuracy of coronary CTA by coronary segment, coronary artery, and patient. Patients were excluded from the study for atrial fibrillation, but not for high heart rate, obesity, or coronary calcification. All vessels were included in the analysis, including those with a diameter <1.5 mm. Coronary stenosis was defined as >50% luminal narrowing.

The sensitivity and specificity of 64-slice CT for significant coronary stenosis by segment was found to be 95% and 86%, respectively; by artery, 92% and 91%; and by patient, 90% and 95%.* The study also examined the effect of calcification on the diagnostic accuracy of coronary CTA. In patients with an Agatston score of 0 to 100, sensitivity was 94%; specificity was 95%; positive predictive value was 94%; and negative predictive value was 95%. In patients with an Agatston score >400, those numbers fell to 93%, 67%, 93%, and 67%, respectively.

In addition, the study confirmed clinical observations that obesity reduces the diagnostic accuracy of coronary CTA. In patients with a body mass index <25, sensitivity, specificity, positive predictive value, and negative predictive value...
were all 100%. In patients with a body mass index >30, those indicators fell to 90%, 86%, 91%, and 86%, respectively. This finding is intuitive: The larger the body mass, the greater the attenuation of photons within the body, and the fewer photons reaching the detector array.

Heart rate had a substantial effect on scan quality as well. In patients with a heart rate <70 bpm, sensitivity was 97%; specificity, 95%; positive predictive value, 97%; and negative predictive value, 95%. By comparison, in patients with a heart rate>70 bpm, sensitivity was 88%; specificity, 71%; positive predictive value, 78%; and negative predictive value, 83%.

In summary, many of the studies that have compared cardiac catheterization and coronary CTA have enrolled only small numbers of highly selected patients. It is clear, however, from well-designed studies and meta-analyses that current-generation CT scanners can detect obstructive coronary plaque with high accuracy. Even with older generation scanners, the posttest probability of disease decreases substantially with a negative CT angiogram. Scan quality and interpretation are hindered by high heart rates, obesity, and coronary artery calcification.

**Any plaque**

There are numerous methods to diagnose atherosclerosis, including detection of aortic and carotid plaque by magnetic resonance imaging, measurement of carotid intimal-medial thickness by ultrasound, coronary calcium scoring by CT, and measurement of the ankle-brachial index.
The best noninvasive test for the diagnosis of coronary artery disease is coronary CTA. Still, the diagnostic accuracy of this imaging technique for the detection of any plaque is far lower than for the detection of obstructive plaque. Achenbach et al reported a sensitivity and specificity for the detection of any plaque of 82% and 88%, respectively. Soft plaque was detected with a sensitivity of 78% and a specificity of 87%, while calcified plaque was detected with a sensitivity and specificity of 84% each.

The detection of any coronary plaque will improve with advances in spatial and temporal resolution. In addition, it will be important to better manage high- and low-contrast resolution, including determining optimal iodine dose, introducing new contrast materials that specifically target atherosclerotic plaque, and refining contrast materials and workstation techniques that enable better differentiation of the arterial wall, coronary plaque, and surrounding fat.

**Plaque composition**

The assessment of plaque composition by coronary CTA is an exciting area of focus. As shown in Figure 2, marked heterogeneity characterizes coronary plaque. Stenoses may be mild, moderate, or severe, and may be composed of calcified, noncalcified, or mixed plaque. CT easily distinguishes between these 3 categories of plaque. A study by Leber et al, conducted on a 16-slice CT scanner, showed that soft plaque had an average density of 49 HU, intermediate or mixed plaque had an average density of 91 HU, and calcified plaque had an average density of 391 HU. However, CT is less successful in differentiating the components of soft plaque—lipid, fibrous, and/or thrombotic tissues, and water—as the HU densities of these tissues tend to overlap.

Recently, our group investigated whether the identification of plaque composition and distribution by coronary CTA would enhance the prediction of clinically significant CAD, which was defined as >50% stenosis in any major epicardial vessel on invasive coronary angiography. The study involved

---

**FIGURE 4.** This illustration depicts a system to assist in determining the clinical significance of plaque burden seen on coronary CT angiography. The coronary arteries are divided into 16 segments and assigned points that describe the severity of stenosis (0 = none, 1 = mild, 2 = moderate; 3 = severe). The segment stenosis score is the sum of the stenosis scores in all 16 segments, for a maximum possible score of 48. The segment involvement score is the sum of the number of segments with plaque, for a maximum possible score of 16.
133 consecutive patients referred for both 16-slice coronary CTA and invasive coronary angiography. The mean age of the study group was 56 years, and 59% were male. Coronary CT angiograms were evaluated for plaque composition, distribution, and severity by investigators blinded to the results of invasive coronary angiography. Plaque composition was defined as soft (>75% soft), calcified (>75% calcified), or mixed (<75% soft and <75% calcified). Plaque distribution was defined as focal (≤3 discrete sites) or diffuse (>3 sites, or ≤3 sites with continuous plaque encompassing more than one third of the vessel). Plaque severity was defined as none, mild (<25% stenosis), moderate (25% to 50% stenosis), or severe (>50% stenosis).

The study showed that patients with clinically significant CAD on quantitative coronary angiography had significantly more mixed plaque than those without clinically significant CAD (76% versus 46%; P = 0.02). The prevalence of other types of plaque was equivalent in the 2 groups. Similarly, more patients with clinically significant CAD had diffuse plaque when compared with those without CAD (76% versus 20%; P < 0.001).

Examination of the odds ratios associated with plaque composition showed that patients with mixed plaque were nearly 4 times as likely to have significant coronary stenoses. A similar odds ratio was observed for diffuse plaque. Of patients with both mixed and diffuse plaque, nearly 92% had clinically significant CAD.

A study by Hoffmann et al compared the plaque composition of culprit lesions in patients with acute coronary syndromes (ACS) to that of stable lesions in the same patients and in patients with chronic stable angina. These authors found that noncalcified plaque was far more prevalent in the culprit lesions of patients with acute coronary syndromes when compared with the stable lesions in patients with ACS and in patients with stable angina (100%, 62%, and 77%, respectively).

A study by Hausleiter et al also demonstrates the importance of noncalcified plaque. In this study, 161 consecutive patients at intermediate risk for CAD underwent 64-slice coronary CT. The majority of noncalcified plaques resulted in luminal narrowing of <50%. However, patients with noncalcified plaque exhibited significantly higher levels of total cholesterol (231 mg/dL versus 202 mg/dL; P = 0.003), low-density-lipoprotein cholesterol (140 mg/dL versus 121 mg/dL; P = 0.008), and C-reactive protein (1.7 mg/L versus 1.0 mg/L, P = 0.045), when compared with patients with calcified plaque. Future studies will likely confirm that plaque characteristics and composition are independently associated with clinically significant CAD.

**Clinically relevant plaque**

Although the detection of obstructive and nonobstructive plaque and the assessment of plaque composition are important, it is the ability to use that information in the diagnosis and treatment of patients that truly matters. Cardiologists have traditionally used nuclear stress testing, invasive coronary angiography, and, more recently, coronary CTA to detect >70% stenosis of the coronary arteries. However, most myocardial infarctions (MIs) result from low-grade stenoses. In fact, 86% of people have a <70% stenosis prior to MI, and 4 of every 10 individuals who experience either MI or sudden cardiac death have no prior warning symptoms.

Figure 3 shows the diagnostic testing results of a man with atypical chest pain. He underwent nuclear stress testing, exercising >10 minutes on the Bruce protocol. Myocardial perfusion was found to be normal and the patient’s prognosis was judged to be extremely favorable. Because of persistent chest pain, he underwent coronary CTA, which demonstrated soft, calcified, and mixed plaque in the left anterior descending (LAD), left circumflex, and right coronary arteries. These findings raise the question: What is such a patient’s true risk? How should we interpret plaque burden?

To answer such questions, our group conducted a study of 1127 patients who were older than 45 years who underwent multidetector coronary CTA after presenting with symptoms that included pain, tightness, and pressure in the chest; dyspnea; and palpitations. The follow-up period averaged approximately 15 months. The primary endpoint of the study was all-cause mortality.

In order to answer such questions, we have devised a system to aid in interpreting the clinical significance of plaque burden on coronary CTA, first dividing the coronary arteries into 16 arbitrary segments based on a modified American Heart Association coronary tree model (Figure 4). We then devised a point system to describe the severity of stenosis, with 0 signifying no stenosis; 1, mild stenosis; 2, moderate stenosis; and 3, severe stenosis. The segment stenosis score was determined by summing the stenosis scores in all 16 segments, for a maximum possible score of 48. The segment involvement score was determined by summing the number of segments with plaque, for a maximum possible score of 16. We are now attempting to study the effects of such plaque burden on over 1000 individuals who underwent multidetector coronary CTA after presenting with chest symptoms that include pain, tightness and pressure in the chest, as well as dyspnea and palpitations.

**Conclusion**

Without question, invasive coronary angiography is superior to coronary CTA for the detection of luminal stenosis. But in 2006, coronary CTA can offer far more than mere lumengraphy, instead identifying clinically relevant disease and providing important clues as to a patient’s true risk of myocardial infarction and cardiac death. In short, coronary CTA addresses the 3 questions that patients and physicians most want answered: 1) Are there stenoses of the coronary arteries? Coronary CTA clearly has the diagnostic accuracy to identify obstructive lesions as well as high-risk nonobstructive lesions. 2) If there are stenoses of the coronary arteries, what is the best treatment strategy? Coronary CTA can assist in determining whether a patient should undergo revascularization or medical therapy.
3) Whatever the treatment approach, what is the prognosis? The evidence of the prognostic capabilities of CTA is beginning to accumulate and will certainly grow over time.

REFERENCES

Discussion

ELLIOT K. FISHMAN, MD: Thanks very much, Jim. That was some terrific data. I would like to start the discussion in terms of cardiac cath versus coronary CTA. In your practice, how do you triage patients? Have you seen any changes in the number of caths performed now that you’re using 64-slice CT?

JAMES K. MIN, MD: The way we triage coronary CT is to limit it to individuals at low-to-intermediate risk of coronary heart disease. If I have a high enough pretest probability that that person has significant coronary disease, I see no reason not to proceed directly to invasive coronary angiography. Cath has a low complication rate and it’s pretty routine. So, I don’t have any problem sending a patient to the cath lab. In terms of what coronary CTA does to cath lab volume, I think it will increase it more than anything. Certainly, it will increase the intervention, for better or worse. I think that in the short-term future, it will increase diagnostic angiograms as well.

What I think it will ultimately decrease is nuclear SPECT volume more than anything. I would love to hear other people’s perspectives on this. But if we can find a way to combine perfusion with CT, in particular myocardial perfusion, then I think it’s going to dramatically reduce nuclear volume.

STEPHAN ACHENBACH, MD: At our institution, cardiac CTA has decreased the number of purely diagnostic caths a little bit. It has definitely increased the share of interventions in the cath lab, in that a higher percentage of people who have caths are those who then need interventions. I would guess that we are replacing roughly 8% to 10% of our diagnostic caths with CTA. Overall, in our institution, I think approximately 8% to 10% of people who are sent for CTA, and who would have had an invasive angiogram if they didn’t have CT, now have a CT and do not go on to angiography. But it’s very hard to say how much of the change is the influence of the CT scan, because cath lab volumes change all the time for other reasons.

MATTHEW BUDOFF, MD: I’d like to reinforce that use of CTA as the front end of the cath lab, as a filter, as Stephan described. We use CTA in our county hospital with a pre-cath clinic. We triage some of the patients who were going to get cath to CTA, and when the CTA is normal to some reasonable diagnostic eye, then they don’t go on for invasive cath. So those patients definitely would have gotten a cath if CTA had not been available at our institution. So in that capacity, CTA lowers the cath volume.

I think though, the tendency is to use CTA in some capacity for screening. It can even be used in the next generation of patients who probably wouldn’t quite qualify for a cath, but since we now have a safer, cheaper, easier cath, we can do it anyway. Then you find obstructive disease, now you’re cathing those patients. So, in a real-world practice, you’ll see a slight increase in cath volume. But the cost offset is so great, that I still think that CTA will prove to be cost saving, even if it does slightly increase the cath volume. You can definitely get rid of the problem that we have in the United States of the high rate of normal caths. Although it’s safe, patients’ pocket books are not as safe as they are. If a diagnostic cath costs upwards of $10,000, the patient’s 20% co-pay is significant. When you look at it as an economic burden, it’s going to be a huge savings to the patient, as well as to the institution, to use CTA judiciously as the front end of the cath lab.

SAMUEL WANN, MD, MACC: I think nuclear volume is more likely to be reduced by coronary CTA than cath lab volume. The other big cost savings for us is in the emergency room. Reducing the length of a patient’s stay in the ER and not admitting patients with normal coronary CTA to the hospital is a very popular strategy both with the patients and with the hospital administration, because it reduces cost. I’m not sure we’ll reduce the cath lab volume, but I think the caths that are done will be more appropriate and will lead to better care. I would be careful not to promise that we’re going to
reduce cath lab volume. That may be a false promise, but I certainly think we can take care of patients more efficiently and more safely by using CT.

**FISHMAN:** I do think CTA will truly affect the volume of nuclear studies, particularly from the ER perspective. I think that’s where the biggest drop will be. I think at cath time, you’ll lose some patients or gain some patients. It will just be a different segment of population. At Hopkins, we do 7 to 10 nuclear studies in the morning—and the morning drags on to about 5 in the afternoon for us. The rate of negative studies is 92%. So, we’re starting to use cardiac CT. If the ER people have a negative CT study and the EKG is negative, we send the patient home.

Ella Kazerooni has presented some of her work in Michigan, and she is working on an article. They were looking at their cardiac acute care center and they have looked at cost savings in an ER. If you go to a CT-based model, rather than a traditional model, it could save up to $3 million per year. So, I think those numbers are important, particularly as many of us around the table here deal with payers who do not want to reimburse for cardiac CT. If we had the ability to say that it does provide a cost savings, besides better patient care, I think there might be more interest in paying for cardiac CT.

**ACHENBACH:** Yes. There’s tremendous potential in the ER applications, but there is also a tremendous lack of data at the present moment. This is probably the most promising application, but, except for 2 flimsy abstracts, we really don’t have a lot of data.

**FISHMAN:** There are 2 published articles on use in the ER. But I think you’ll see a lot more of those. The big problem is the control of doing those studies. The group from Michigan managed to do both a nuclear study and a CT on all the patients. They didn’t change the management initially and I think that will be critical. There’s a problem, I think, for all of us, with the reimbursement. Many of the payers say the same thing as you showed—that there are not enough data and that the number of patients who have actually been studied is small.

Those patients are a subselected population, you know, so the outcomes are predictable in some ways. The question is, how are the outcomes going to change in low-risk patients? It’s critical to really have that data. If there are roughly 600 to 900 patients that keep getting quoted—and that’s been difficult. Medicare was against reimbursing for it because the studies weren’t done on their older population. A private insurer referred to the Duke evaluation, which wasn’t very positive. So one of the things that is critical, in terms of what we all do or other people do, is getting data in the literature to really show data in larger numbers of patients.

In the data you showed, which are very impressive on plaque composition, you mentioned a couple of conclusions. Just to summarize, what are your best conclusions from some of the work you’re doing in terms of plaque composition?

**MIN:** Very simply stated, mixed plaque bothers me a lot. I think that we will find that this is much more predictive of adverse outcomes or ischemia. Calcified plaque bothers me very little, soft plaque bothers me somewhat, and mixed plaque bothers me more. It seems to be more stenotic and it looks angrier, more irritable.

**BUDOFF:** I thought I’d heard about some of the analysis in your joint work that showed that mixed plaque had worse outcomes. So there might be something in the evolution of the analyses coming from your data pool.

**MIN:** Our data is a collaborative effort between myself, Tracy Callister, Leslee Shaw, and Daniel Berman.

**ACHENBACH:** I would like to comment that I personally don’t like the term “soft plaque” at all. Everybody uses it, because it’s so graphic. But we don’t know whether it’s soft or not. I think we should stick to the terms “calcified” or “noncalcified” plaque. That’s really all we know. Soft is a little too suggestive, so I really don’t like the term. After anyone trains at my place, they will never dare say “soft plaque” ever again.

**WANN:** Coming back to a previous discussion of how to report these out, I guess I’m pleased. I intuitively start out my dictation with an assessment of the overall plaque burden, and I use Dr. Achenbach’s terminology, saying something like “There is diffuse 3-vessel atherosclerotic coronary artery disease with calcified and noncalcified plaque involving the proximal vessels.” I try to get an overall, verbal qualitative description of what you’re trying to quantify. Then, I go on to further describe areas of narrowing, using terms like “nonobstructive” or “probably obstructive,” or “high grade, possibly occlusive.” I like to convey the perceived severity of obstruction, but I don’t like to get pinned down on numbers. What does the vessel look like? You’ve seen a picture of an almost normal coronary angiogram, and then a diffusely diseased right coronary artery with lots of plaque. I would not have described it as a high-percentage stenosis, but I would have conveyed to the clinician that there was a severe atherosclerotic disease in the right coronary artery. When you’re reporting out clinically, I presume you’re not giving a quantitative analysis. But do you include a description, as I do, of the general appearance of the artery?

**MIN:** Yes, we do. Usually, by segment, we’ll note all three of those things: non, mild, moderate, severe; noncalcified, mixed, or calcified; and focal or diffuse within arterial segment. Then there will be a conclusion statement that will indicate if there is a low, intermediate, or high overall coronary artery plaque burden.

**WANN:** Then I also give some assessment of flow obstruction.

**MIN:** Yes. So we note non, mild, moderate, or obstructive, and that way we can at least convey to them if we think that there’s something that should be cathed here. But we note the location of it, and they have to decide whether or not they want to cath it or not.

**ACHENBACH:** If you want to do a cath, what is the reason for it?

**MIN:** We cath with the anticipation that there will be a >70% stenosis.

**CHIP GILKESON, MD:** Do you anticipate that this kind of data is then going to change your lipid therapies?
Then with the follow-up scans, they will to look and quantify composition and change.  

**MIN:** We do not quantify the volume at the present time. I don’t think any of the software algorithms work. I think that the only way you could do it is to literally just take the segment and cut it up, and try to get the areas interpolated. But I think that estimating plaque volume is not that accurate right now. Clinically, nobody’s ever going to do it—just because it would just take too long, unless it were automated. In terms of looking at the overall plaque, I think all you can say is that it’s mild, moderate, or severe; or mild, moderate, or obstructive. Then 2 or 3 years later, we can try to rescan them. We haven’t even thought to do that yet and certainly will need a better, more accurate measurement of plaque stenosis or volume in order to consider it.

**FISHMAN:** As you commented, a lot of software vendors now sell packages that do plaque analysis. They create all sorts of numbers and data, and it comes in very nice colors. But it’s hard to say what it all means, and it comes in a quantification of numbers that probably, at this point, have no value. Time will tell.

**BUDOFF:** Even quantitative coronary angiography is poor at following plaque. I apologize to those who have made their career out of it, but it’s not an easy thing to do. Now we’re looking at something with much lower spatial resolution. You’re just not going to get that same quantitation to be able to reliably follow people serially, at least in an individual. Maybe in a group we can do that, as we have with carotid IMT, with coronary calcium, and with QCA. But, in an individual, I think it’s going to be challenging to the point of maybe not possible, at least with the current scanner configuration and the spatial resolution, which I think is most critical.

**ACHENBACH:** There’s a good paper out by Dr. Leber from Munich, looking at 64-slice CTA plaque quantification. The inter-observer variability in a very careful analysis was 37% for plaque volume. So, I mean, you don’t have to even think about a follow-up scan if there is 37% variability of 2 people looking at the same thing.

**BUDOFF:** In one of your earlier papers, you noted 53% plaque volume compared with being detectable compared with IVUS. So, clearly, we’re missing at least half the boat right there. So, you miss half of it and then you’re unsure about 37% of the half that you see. You’re in a pretty bad place, statistically.

**JILL E. JACOBS, MD:** Not to mention the fact that when you do the follow-up, the technology is slightly different, or your technique is slightly different, or the patient’s slightly different.

**BUDOFF:** Or the reader’s different.

**JACOBS:** So how accurate is your follow-up actually going to be?

**WANN:** The magnitude of the change with the early trials, looking at intervention on vascular ultrasound is not great. It’s well within the variability of the measurement by CT. The plaque doesn’t just melt away and disappear, unfortunately.

**ACHENBACH:** We are currently running a scientific evaluation of looking at plaque volumes. This is not clinical; it is research. We limit ourselves to the left main and proximal because those are the image segments that have the highest image quality. In these segments, using two observers, we found (looking at it manually, as you described, it was very tedious) that we had 18% interobserver variability. This was even when limiting ourselves to the segments with the highest image quality. We are actually following patients in 12-month intervals. When we scanned patients twice in a 12-month interval, we found that 85% of these patients have had progression of plaque volume, and 15% had either stable plaque or regression of plaque volume. So there seems to be something going on and that we can measure, but we limit ourselves very strictly, not to the entire coronary tree, but only to the segments that have the highest image quality. There’s potential, but the technology is not there at the moment.

**BUDOFF:** The literature addresses evaluable segments. The insurance companies are telling us that you have to include all segments and cull all the ones that you couldn’t evaluate as false positives or false negatives, and then change the test characteristics dramatically. Clinically, if a test is diagnostic, then I can apply it to my patient population. If it’s non-diagnostic, then I wouldn’t. For example, if I have a nondiagnostic echocardiogram, I’m not going to try to take the best guess of the ejection fraction from that test. I’ll do something else if I can’t get good image quality. You’ve looked at the scientific literature pretty carefully. What do you think we should do with these nonevaluable segments? How important are they in our overall assessment of the technology?

**MIN:** Clinically, it’s not going to make that much difference. We took about 600 people who had CTs, looked at where the plaque was, and it was all proximal to distal. Everybody has proximal LAD plaque, and after that, it sort of tapers down. It’s more so in the proximal circumflex and the proximal right than it is in the distal vessel, which is more often the unevaluable segment of the vessel. But, that was all on 16-slice CT. With 64-slice CT, I would argue that in the vast majority of patients this will not happen. With 16-slice, there may have a good proportion of the vessels that you couldn’t evaluate. I think it’s going to be less of an issue with 64-slice.

**ACHENBACH:** We must not fool ourselves. When we think we see plaque on CT, it might often look like a plaque, but there might not be any. It could be just an artifact. Or, it can be the other way around—the artery might seem perfectly clear on the CT scan and you think you have super image quality. But if you actually push an IVUS catheter down the same vessel, you will see that there is plaque sitting there that you just don’t see on CT. The spatial resolution is 0.4 mm, and with IVUS you can look at the smaller plaques, and they may be 0.5 mm in thickness. How are you supposed to see that with CT?

So if you see something on CT, it’s likely that there is plaque. But, if you don’t see anything on CT, it does not necessarily mean that there’s no plaque. So you have to be very, very careful.
FISHMAN: In terms of what you mentioned in terms of payers, I think one of the issues is that the data from studies tend to focus on patient management rather than on segment management. I think you're almost asking too much, particularly when it comes down to it—you're managing patients and not worrying if you saw 14 or 16 segments. I think people focus on that, rather than focusing on the important management aspects.

BUDOFF: But the data have to be patient based, and I think the papers that are published without patient-based numbers are just poorly done, because we manage our patients. Actually, once I see a definitive stenosis, and I know that person is going to the cath lab, if I missed a second stenosis, I don't care, because I got the other one right, and they were cathed. My goal was not to make the final decision of surgery versus angioplasty. The decision was: Does this person need cath or not? I'm correct, from a patient-based management. That's what's really going to matter to the patients and for outcomes. So we have to keep it patient based and not segment based, as much as possible. Obviously it's nice to see that segment-based analysis, we're still in the low 90s with 64-slice CT, but patient-based management is going to be what's clinically important.