# Best of the AHA Scientific Sessions 2007

*Highlights From the American Heart Association Scientific Sessions, November 4-7, 2007, Orlando, FL*

[Rev Cardiovasc Med. *2008;9(1):62-69*]



**Key words:** Myocardial infarction • Atrial fibrillation • Congestive heart failure • Cardiac resynchronization therapy • Percutaneous coronary intervention • Prasugrel • Clopidogrel • Multidetector spiral computed tomography

The American Heart Association (AHA) Scientific Sessions is the largest annual convention focused on cardiovascular (CV) diseases and stroke. The 2007 meeting featured more than 4000 abstracts describing original research. Here we discuss key studies on the utility of microvolt T-wave alternans testing for risk stratification after myocardial infarction (MI), rhythm control versus rate control among patients with heart failure and atrial fibrillation, use of resynchronization therapy in patients with a narrow QRS, prasugrel versus clopidogrel to prevent thrombotic complications of percutaneous coronary intervention (PCI), use of metoprolol in patients after noncardiac surgery, whether torcetrapib can help reduce CV events, and use of 64-slice multidetector spiral computed tomography in detecting significant coronary artery disease.

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## Microvolt T-Wave Alternans Testing for Risk Stratification of Post-MI Patients

Dr. Theodore Chow, Director of Electrophysiology Research for the Carl and Edyth Lindner Clinical Trials Center at the Ohio Heart and Vascular Center in Cincinnati, presented the results of the Microvolt T-Wave Alternans Testing for Risk Stratification of Post MI Patients (MASTER I) clinical trial.[1] The goal of the trial was to evaluate the utility of microvolt T-wave alternans (MTWA) testing in risk stratification for life-threatening ventricular tachyarrhythmic events among post-MI patients with impaired left ventricular systolic function undergoing implantable cardioverter defibrillator (ICD) implantation. Patients included in the study had a prior MI and a left ventricular ejection fraction (LVEF) of 30% or less. Since they were all to undergo ICD implantation, they met Multicenter Automatic Defibrillator Implantation Trial (MADIT) criteria. Patients were excluded if they had atrial fibrillation or flutter at the time of MTWA testing, an MI in the previous month, or a prior clinical sustained VT/VF, or if they had undergone revascularization in the previous 3 months or electrophysiological studies or MTWA testing in the previous year.

All patients underwent an MTWA assessment at baseline and were classified as MTWA negative or not normal (positive or indeterminant).

After testing, ICD implantation was performed.

The primary endpoint of life-threatening, ventricular tachyarrhythmic events occurred in 10.3% of the MTWA-negative group and 13.3% of the non-negative group ($P = .37$). Total mortality was lower in the MTWA-negative cohort compared with the non-negative cohort (6% vs 13%; $P = .02$). Efforts have been made to explain what appears to be an uncoupling between what was a significant ability to risk-stratify mortality, but not life-threatening tachyarrhythmic events. Included among the explanations is that in this trial, 90% of the events were ICD shocks, some of which may not have been for events that would have proved life threatening, and that the placement of the ICD itself may create a proarrhythmic milieu. The results of this study should be considered within the context of the entire clinical trial experience with MTWA.

## Rhythm Control Versus Rate Control

The Atrial Fibrillation and Congestive Heart Failure (AF-CHF) trial was designed to compare rhythm control with rate control among patients with heart failure and atrial fibrillation.[2] The study subjects had New York Heart Association (NYHA) class II to IV congestive heart failure and an LVEF of 35% or less, or they had NYHA class I congestive heart failure with prior hospitalization for heart failure or an ejection fraction of 25% or less and a documented clinically significant episode of atrial fibrillation within the past 6 months. The primary endpoint was CV mortality. Secondary endpoints included total mortality, stroke, worsening heart failure, and a composite of CV death, worsening congestive heart failure, and stroke. Patients were followed for a mean of 37 months. Among all patients, 31% had NYHA class III or IV heart failure, with a mean LVEF of 27%. Atrial fibrillation was chronic in 69% and paroxysmal in 31%.

There was no difference in the primary endpoint of CV death between the rhythm control and rate control groups (26.7% in the rhythm control group vs 25.2% in the rate control group; $P = .59$) (Figure 1). The rhythm and rate control groups also had no difference in the secondary endpoints, including total mortality (31.8% vs 32.9%; $P = .73$), stroke (2.6% vs 3.6%; $P = .32$), worsening heart failure (27.6% vs 30.8%; $P = .17$), or the composite of CV death, worsening congestive heart failure, or stroke (42.7% vs 45.8%; $P = .20$).

## Cardiac Resynchronization Therapy

The Resynchronization Therapy in Patients with Narrow QRS (RethinQ) trial was designed to evaluate the effectiveness of cardiac resynchronization therapy (CRT) among patients with heart failure and a narrow QRS complex who received an implantable cardioverter defibrillator (ICD).[3,4] Patients in this trial had an approved indication for implantation of an ICD, NYHA class III heart failure and an LVEF of 35% or less while on an optimal medical regimen, evidence of mechanical dyssynchrony as measured by echocardiography using tissue Doppler imaging or M-mode, and a QRS duration of less than 130 msec. Some of the exclusion criteria included patients who had a standard bradycardic indication for pacing; previous treatment with CRT; continuous atrial fibrillation within 1 year prior to enrollment or cardioversion for AF in the past month; symptomatic chronic obstructive pulmonary disease; recent MI, unstable angina, or cardiac revascularization in the previous 40 days; a cerebrovascular accident or transient ischemic attack within the previous 3 months; and a severe musculoskeletal disorder.

Approximately 2 weeks after ICD implantation, patients were randomized in a double-blind manner to receive or not receive CRT. Mean follow-up was for 6 months.

The primary endpoint, an increase in peak oxygen consumption at or exceeding 1.0 mL/kg of body weight per minute, did not significantly differ between the CRT group and the control group (46% vs 41%; $P$ = NS) (Figure 2). There was also no difference in change in quality of life scores (median −8 vs −7; $P = .91$), change in 6-minute walking test

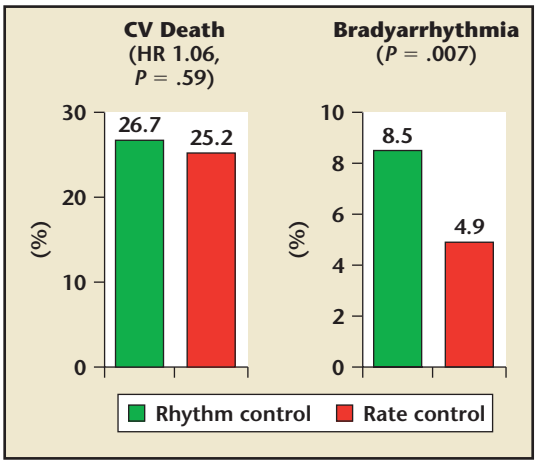



**Figure 1.** *The AF-CHF trial was designed to compare rhythm control with rate control among patients with heart failure and atrial fibrillation. There was no difference in CV death. Bradyarrhythmias were increased in the rhythm control group. AF-CHF, Atrial Fibrillation and Congestive Heart Failure; CV, cardiovascular; HR, hazard ratio. Data from Roy D.[2] Adapted with permission from Cardiosource.*

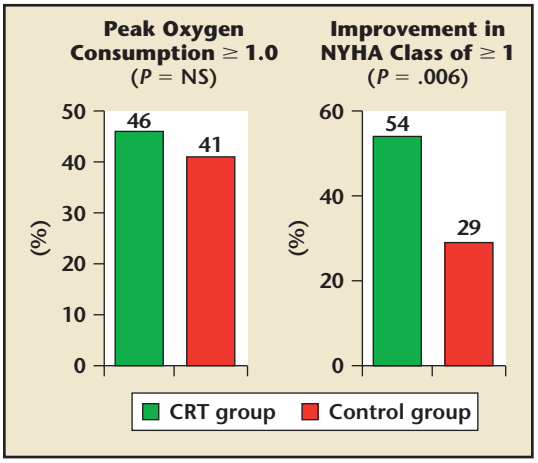



**Figure 2.** *In the RethinQ trial, CRT did not significantly affect the primary endpoint, an increase in peak oxygen consumption at or exceeding 1.0 mL/kg of body weight per minute. An improvement in NYHA classification of at least 1 class was more common in the CRT group than the control group. RethinQ, Resynchronization Therapy in Patients with Narrow QRS; CRT, cardiac resynchronization therapy; NYHA, New York Heart Association. Data from Beshai JF et al.[4] Adapted with permission from Cardiosource.*

(median 26 vs 6; $P = .23$), or change in ejection fraction (median, 1.2 vs 2.0; $P = .83$). Interestingly, despite the lack of change in peak oxygen consumption, an improvement in NYHA classification of at least 1 class was more common in the CRT group than the control group (54% vs 29%; $P = .006$). Among the subgroup of patients with a QRS interval at or exceeding 120 msec, peak oxygen consumption increased in the CRT group compared with the control group ($P = .02$), but it did not differ in the subgroup of patients with a QRS interval of less than 120 msec ($P = .45$).

It was somewhat surprising to see no improvement in peak oxygen consumption in the patients with a QRS interval of less than 120 msec, even though they did have evidence of dyssynchrony on echocardiography and were much more likely to have improvement in the NYHA grade for heart failure. Of interest would be longer term data that would give insight into whether one could prevent progression of heart failure with CRT in this patient population.

## Prasugrel Versus Clopidogrel for PCI Patients

Results from the Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel (TRITON-TIMI 38) were presented by Dr. Elliott Antman from Harvard Medical School in Boston, MA.[5,6] The trial was designed to compare treatment with the thienopyridine prasugrel versus clopidogrel among patients undergoing planned PCI for an acute coronary syndrome. Inclusion criteria were ischemic symptoms that lasted for at least 10 minutes within 72 hours of randomization, Thrombolysis In Myocardial Infarction (TIMI) risk score of 3 or greater and either ST-segment deviation of at least 1 mm or an elevated cardiac biomarker of necrosis for ST-elevation myocardial infarction (STEMI) patients, and symptom onset within 12 hours of randomization if the primary PCI was planned or within 14 days if the patient was receiving medical treatment for STEMI. Patients were excluded if they had an increased bleeding risk, anemia, thrombocytopenia, intracranial pathology, or used any thienopyridine within 5 days of presentation.

Patients were randomized in a double-blind manner to a 60-mg loading dose of prasugrel followed by a 10-mg maintenance dose or a 300-mg bolus of clopidogrel followed by a 75-mg maintenance dose. The maintenance doses were continued for 6 to 15 months. The primary endpoints were CV death, MI, or stroke. Secondary endpoints were the primary endpoint at 30 and 90 days; stent thrombosis; CV death, MI, stroke, or rehospitalization due to a cardiac ischemic event; and CV death, MI, stroke, or urgent target vessel revascularization.

The primary endpoint of CV death, MI, or stroke was significantly lower in the prasugrel group compared with the clopidogrel group (9.9% vs 12.1%; $P < .001$) (Figure 3). The efficacy benefit was observed by 3 days (4.7% in the prasugrel group vs 5.6% in the clopidogrel group; $P = .01$) and from 3 days to end of follow-up (5.6% in the prasugrel

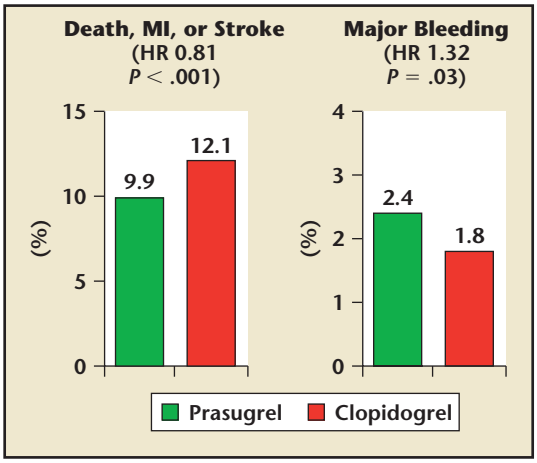



**Figure 3.** *In TRITON-TIMI 38, the primary endpoint of CV death, MI, or stroke was significantly lower in the prasugrel group compared with the clopidogrel group. Major bleeding was higher in the prasugrel group. TRITON-TIMI 38, Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel; CV, cardiovascular; MI, myocardial infarction; HR, hazard ratio. Data from Wiviott SD.[6] Adapted with permission from Cardiosource.*

group vs 6.9% in the clopidogrel group; $P = .01$). Definite or probable stent thrombosis occurred less frequently in the prasugrel group than in the clopidogrel group ($P < .001$).

The safety endpoint of TIMI major non–coronary-artery bypass grafting (CABG) bleeding was higher with prasugrel compared with clopidogrel (2.4% vs 1.8%; $P = .03$). CABG-related TIMI major bleeding was also increased with prasugrel (13.4% vs 3.2%; $P < .001$). There was no difference in mortality between the groups. The net clinical benefit endpoint, a composite of death, MI, stroke, or TIMI major bleeding, favored prasugrel (12.2% vs 13.9%; $P = .004$).

The results of this trial confirm the ability of prasugrel in the doses used to be superior to clopidogrel in the doses used to prevent thrombotic complications of PCI in acute coronary syndrome patients. This benefit may be partly related to prasugrel's ability to inhibit platelet function in patients who are clopidogrel-resistant. The increase in major bleeding may indicate a need to be cautious in using prasugrel in patients who may be more highly predisposed to bleeding complications.

[Norman E. Lepor, MD, FACC, FAHA, FSCAI]

## Perioperative Cardiac Events in Patients Undergoing Noncardiac Surgery

The Canadian Institutes of Health sponsored the Perioperative Ischemic Evaluation (POISE) trial of 8351 patients, ages 45 years and older, who were undergoing noncardiac surgery and were expected to stay in the hospital longer than 24 hours. Subjects were recruited from 188 centers in 23 countries.[7,8] They had a history of coronary or peripheral vascular disease, stroke, or heart failure within 3 years of randomization. Subjects received either 100 mg of controlled release (CR) metoprolol or placebo 2 to 4 hours before and 0 to 6 hours after surgery. Daily doses of 200 mg metoprolol CR or placebo were continued for 30 days.

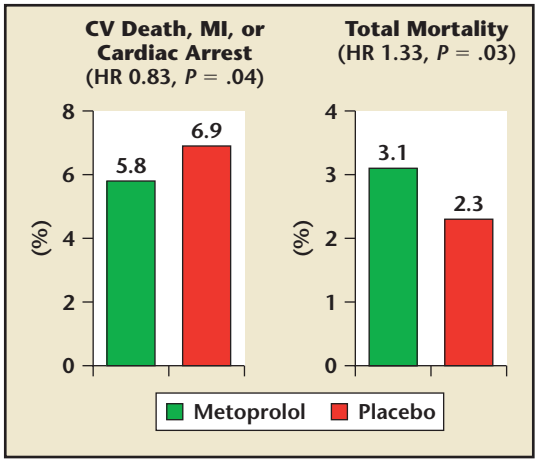



**Figure 4.** *Primary results of the POISE trial. POISE, Perioperative Ischemic Evaluation; CV, cardiovascular; MI, myocardial infarction; HR, hazard ratio. Data from Devereaux PJ et al.[7] Adapted with permission from Cardiosource.*

The primary outcome—the composite of CV death, nonfatal MI, and nonfatal cardiac arrest—occurred in 5.8% of the metoprolol group and 6.9% of the placebo group ($P$ = NS) (Figure 4). In an analysis of the trial, Devereaux and colleagues[7] proposed that for every 1000 patients treated, metoprolol CR would prevent 15 MIs, 3 cardiac revascularizations, and 7 new cases of atrial fibrillation. However, the drug would cause 8 excess deaths, 5 strokes, 53 cases of hypotension, and 42 cases of bradycardia. Thus, the POISE trial is in agreement with Diabetic Postoperative Mortality and Morbidity (DIPOM), Metoprolol after Vascular Surgery (MaVS), and Perioperative Beta-Blockade (POBBLE) as a quartet of large, randomized, placebo-controlled trials that have failed to demonstrate a benefit to beta-blockade in the reduction of perioperative cardiac events in subjects undergoing noncardiac surgery.[9-11] Almost certainly, the 2007 American College of Cardiology/AHA Guidelines on the perioperative management of cardiac patients will need to be revised based on these trials.[12]

## High-Density Lipoprotein Cholesterol Elevation and Reduction in CV Events

Final results of the Investigation of the Lipid Level Management to Understand its Impact in Atherosclerotic Events (ILLUMINATE) and Investigation of Lipid Level Management Using Coronary Ultrasound to Assess Reduction of Atherosclerosis by CETP Inhibition and HDL Elevation (ILLUSTRATE) trials were presented at the AHA 2007 Scientific Sessions[13,14] and published in 2007 in the *New England Journal of Medicine*.[15,16] New data from ILLUMINATE regarding torcetrapib, a cholesteryl ester transfer protein (CETP) inhibitor that increases high-density lipoprotein cholesterol (HDL-C), have shown that the drug probably activated the renin-angiotensin system and stimulated aldosterone secretion (Table 1). This action contributed to the development of hypertension and worked to increase CV events in the torcetrapib group. The ILLUMINATE trial was terminated prematurely in December 2006 because of an increased risk of death and cardiac events in patients receiving torcetrapib (Table 2).

**Table 1**
**Off-Target Adverse Physiologic Effects of Torcetrapib in ILLUMINATE**

In patients receiving torcetrapib/atorvastatin (but not in those receiving atorvastatin alone), there was a significant:

- Increase in blood pressure
- Reduction in serum potassium
- Increase in serum bicarbonate
- Increase in serum sodium
- Increase in serum aldosterone

These changes are consistent with activation of the renin-angiotensin-aldosterone system.

ILLUMINATE, Investigation of the Lipid Level Management to Understand its Impact in Atherosclerotic Events. Data from Barter PJ et al.[15]

ILLUMINATE was a randomized, double-blind study involving 15,067 patients at high CV risk who were randomized to either torcetrapib plus atorvastatin or atorvastatin alone. At 12 months, in patients who received torcetrapib, there was an increase of 72.1% in HDL-C and a decrease of 24.9% in low-density lipoprotein cholesterol (LDL-C) from baseline ($P < .001$ for both). However, torcetrapib was associated with an increase of 5.4 mm Hg in systolic blood pressure, a decrease in serum potassium, and increases in serum sodium, bicarbonate, and aldosterone ($P < .001$ for all comparisons). There was also an increased risk of CV events (hazard ratio, 1.25; 95% confidence interval [CI], 1.09-1.44; $P = .001$) and death from any cause (hazard ratio, 1.58; 95% CI, 1.14-2.19; $P = .006$). Post hoc analyses showed an increased risk of death in patients treated with torcetrapib whose reduction in potassium or increase in bicarbonate was greater than the median change. Importantly, those patients who achieved the highest HDL-C incurred the lowest rate of cardiac events in a strong, graded, inverse relationship (Figure 5).

**Table 2**
**Increased Cardiovascular Events With Torcetrapib in ILLUMINATE**

| | Atorvastatin (n = 59) | Torcetrapib/Atorvastatin (n = 93) |
|---|---|---|
| **Any cardiovascular death** | 35 | 49 |
| Sudden cardiac death | 25 | 26 |
| Fatal MI (not procedure-related) | 6 | 8 |
| Fatal stroke | | |
| Hemorrhagic | 0 | 4 |
| Ischemic | 0 | 2 |
| Embolic | 0 | 0 |
| Other cardiac death | 1 | 4 |
| Heart failure | 1 | 2 |
| Other vascular death/ procedure-related MI | 2 | 3 |
| **Any noncardiovascular death** | 20 | 40 |
| Cancer | 14 | 24 |
| Infection | 0 | 9 |
| Other noncardiovascular | 2 | 4 |
| Trauma/suicide/homicide | 4 | 3 |
| **Reason unknown** | 4 | 4 |

ILLUMINATE, Investigation of the Lipid Level Management to Understand its Impact in Atherosclerotic Events; MI, myocardial infarction. Adapted with permission from Barter PJ et al.[15] 

The ILLUSTRATE trial recruited a total of 1188 patients with coronary disease and treated them with atorvastatin to reduce levels of LDL-C cholesterol to less than 100 mg/dL. Patients were then randomly assigned to receive either atorvastatin monotherapy or atorvastatin plus 60 mg of torcetrapib daily. After 24 months, disease progression was measured by repeated intravascular ultrasonography. There was a 61% relative increase in HDL-C with torcetrapib; however, treatment was associated with an increase in systolic blood pressure of 4.6 mm Hg. The percent atheroma volume (the primary efficacy measure) increased by 0.19% in the atorvastatin-only group and by 0.12% in the torcetrapib-atorvastatin group ($P = .72$). A secondary measure, the change in normalized atheroma volume, showed a small, favorable effect for torcetrapib ($P = .02$), but there was no significant difference in the change in atheroma volume for the most diseased vessel segments.

The large remaining question concerned the impact of a markedly elevated HDL-C on CV events, despite the increase in blood pressure. In both studies, patients with the largest HDL-C increases showed benefits with the drug. Although torcetrapib development has been halted, these findings justify the continued development of other CETP inhibitors, including anacetrapib, which does not elevate blood pressure.[17] At

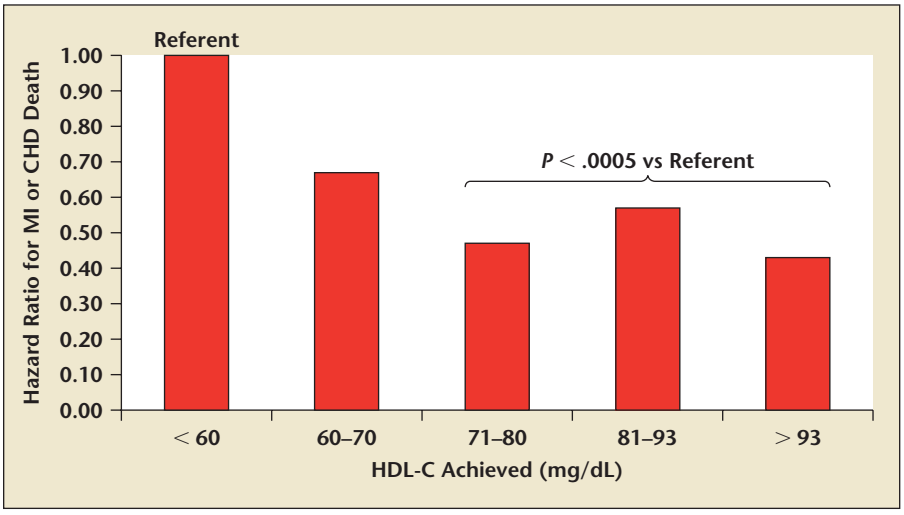



**Figure 5.** *Cardiovascular events stratified by HDL-C level achieved in ILLUMINATE. ILLUMINATE, Investigation of the Lipid Level Management to Understand its Impact in Atherosclerotic Events; HDL-C, high-density lipoprotein cholesterol; MI, myocardial infarction; CHD, coronary heart disease. Adapted with permission from Barter PJ et al.*[13]

the AHA presentation, Dr. Philip Barter (Heart Research Institute, Camperdown, Sydney, NSW, Australia) stated, "There have been concerns voiced that the HDL-C produced by CETP inhibitors may be dysfunctional in some way. But our results, along with new data from the ILLUSTRATE study, are not consistent with that idea. Rather, they are supportive of other in vitro studies that suggest the HDL formed by these drugs is functional." He added, "The observation that torcetrapib increases aldosterone levels is very exciting. If this off-target toxicity was not there, the clinical outcome results may have been very different. Several other CETP inhibitors in early development do not appear to have this effect on aldosterone, which gives us hope that this class of drugs may still be successful."[13]
[Peter A. McCullough, MD, MPH, FACC, FACP, FCCP, FAHA]

## Multidetector Computed Tomography

Coronary angiography remains the gold standard for evaluating the coronary anatomy to detect the presence and degree of coronary stenoses, and the feasibility (and specific type) of revascularization. However, newer imaging modalities capable of diagnosing coronary disease continue to advance, in part in an effort to avoid the inherent risks associated with invasive procedures. Multidetector computed tomography (MDCT) has enabled detailed imaging of the coronary arteries, and a few previous single-center studies have reported good sensitivity and negative predictive value of this technique in excluding coronary disease in low- to moderate-risk patients.

The purpose of the Coronary Evaluation Using Multi-detector Spiral Computed Tomography Angiography Using 64 Detectors (CORE-64) study was to determine the diagnostic accuracy of 64-slice MDCT in detecting significant coronary artery disease as compared with quantitative coronary angiography (QCA).[18] Accordingly, the study evaluated 405 patients older than 40 years undergoing coronary angiography for known or suspected coronary disease at 9 centers in 7 countries. Electrocardiogram-gated, contrast-enhanced, 64-slice MDCT (0.5 mm slice thickness) was performed within 30 days prior to the invasive coronary angiogram, and those patients with Agatston calcium scores of less than 600 (n = 316) underwent MDCT angiography. All segments without coronary stents greater than 1.5 mm were analyzed and assessed visually and quantitatively for stenoses by both methods. Significant stenosis was defined as greater than or equal to 50% by QCA. Diagnostic accuracy was assessed by the area under the curve (AUC) generated by the receiver operating characteristics (ROC), the latter obtained by plotting sensitivity versus 1 minus specificity for each distinct threshold of the diagnostic test that is measured on a continuous scale. The area of the ROC represents the diagnostic ability of the test. The primary endpoint of the study was diagnostic accuracy of MDCT angiography for identifying significant stenoses compared with QCA. Secondary endpoints included per vessel diagnostic accuracy and diagnostic ability to predict need for revascularization between the 2 imaging modalities. Clinical endpoints were determined at 30 days and 6 months.

The study results were presented by Julie M. Miller, MD (Johns Hopkins Hospital, Baltimore, MD) on behalf of the investigators.[18] Among the study cohort, 74% of the subjects were men, the median age was 59 years, and the median body mass index was 27. The median and mean calcium scores were 80 and 140.3, respectively, and the prevalence of significant coronary disease by QCA was 56%. Due to technical and protocol limitations, 291 patients with 868 vessels and 3782 segments were included in the final analysis. All

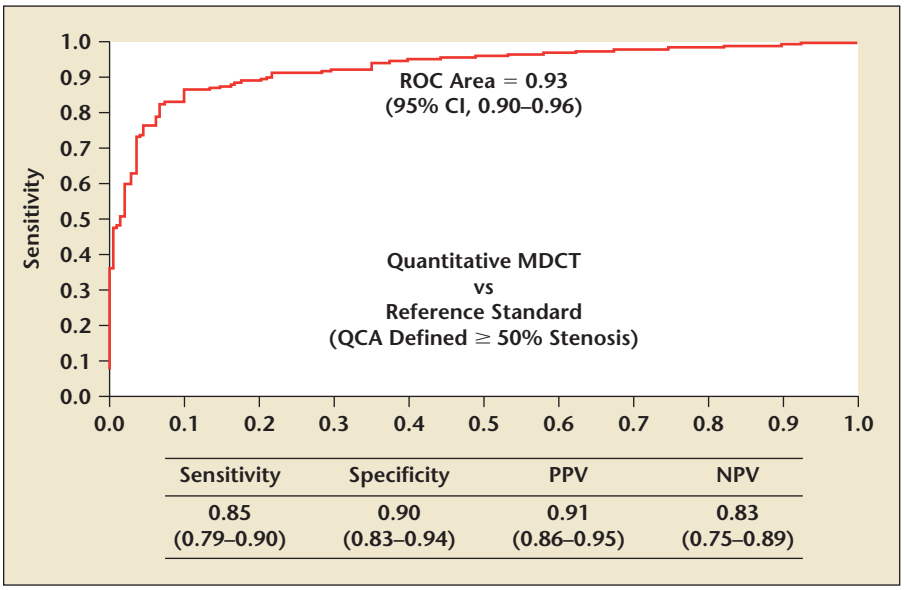



**Figure 6.** *In the CORE-64 study, MDCT had good diagnostic accuracy for detecting significant luminal stenoses as compared with QCA. CORE-64, Coronary Evaluation Using Multi-detector Spiral Computed Tomography Angiography Using 64 Detectors; MDCT, multidetector computed tomography; QCA, quantitative coronary angiography; CI, confidence interval; NPV, negative predictive value; PPV, positive predictive value; ROC, receiver operating characteristics. Adapted with permission from Miller JM.*[18]

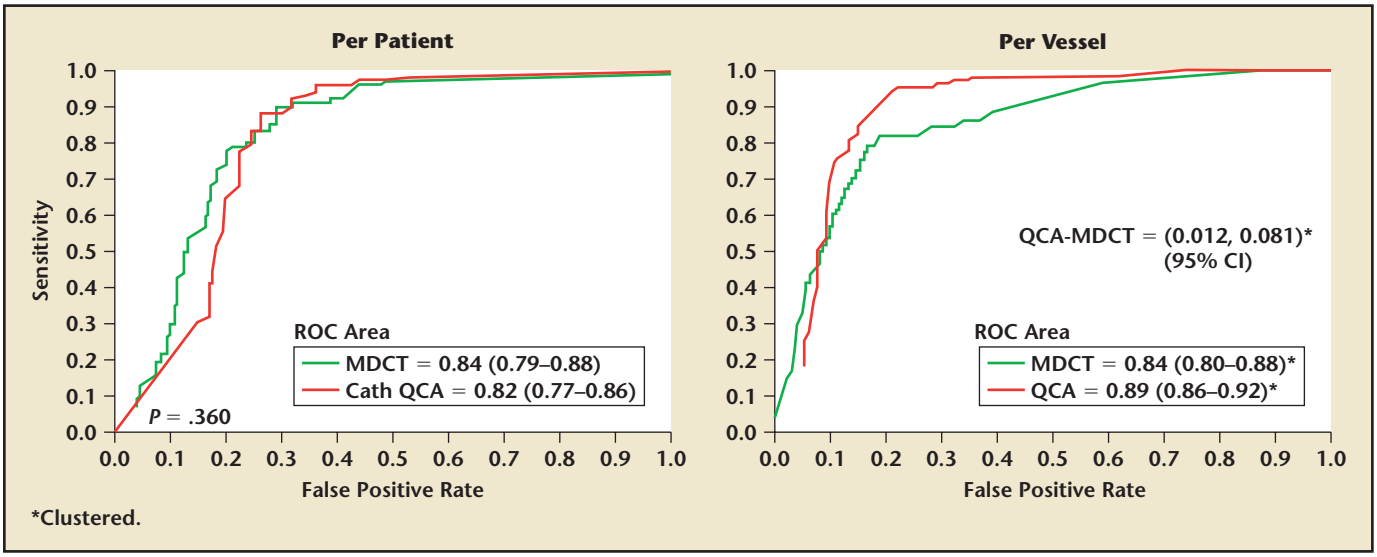
segments were evaluable in 97% of patients, and 98% of vessels and 95% of segments were evaluable.

On a per vessel basis, MDCT had good diagnostic accuracy for detecting significant luminal stenoses (sensitivity = 0.85, specificity = 0.90, positive predictive value = 0.91, negative predictive value = 0.83, ROC AUC = 0.93) when compared with QCA (Figure 6). On a per vessel basis, the diagnostic accuracy was not as good (sensitivity = 0.76, specificity = 0.93, positive predictive value = 0.82, negative predictive value = 0.89, ROC AUC = 0.91) as that obtained with using MDCT on a per patient basis. MDCT angiography had a similar ability to identify patients who had been referred for percutaneous or surgical revascularization when compared with QCA (ROC AUC: MDCT 0.84 vs QCA 0.82; $P$ = .36) (Figure 7). The authors concluded that in those patients with suspected coronary disease and a calcium score of less than 600, the ability of MDCT angiography to assess the presence of significant coronary stenoses and identify patients likely to be referred for revascularization is similar to that of QCA. However, the diagnostic accuracy of MDCT angiography on a per patient basis was better than on a per vessel basis.

*Comment*

The results of CORE-64 add to the accumulating evidence supporting the ability of MDCT to predict significant coronary artery disease and the need for revascularization. However,

**Figure 7.** *In the CORE-64 study, MDCT angiography had similar ability to identify patients who had been referred for percutaneous or surgical revascularization when compared with QCA. CORE-64, Coronary Evaluation Using Multi-detector Spiral Computed Tomography Angiography Using 64 Detectors; MDCT, multidetector computed tomography; QCA, quantitative coronary angiography; ROC, receiver operating characteristics; Cath, catheterization. Adapted with permission from Miller JM.*[18]



the diagnostic accuracy of MDCT in patients with calcium scores greater than 600 and in patients with prior revascularization remains unknown. Given that the amounts of contrast used and radiation exposure are similar for both MDCT angiography and QCA, whether MDCT angiography will ultimately serve to duplicate QCA and add cost rather than eliminate an invasive procedure is unclear. Moreover, the impact of MDCT angiography on outcomes for patients with and without significant coronary disease will require further study. ■

[Alice K. Jacobs, MD, FACC, FAHA]

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## Main Points

- In a trial comparing rhythm control with rate control among patients with heart failure and atrial fibrillation, there was no difference in the primary endpoint of cardiovascular death or in secondary endpoints, including total mortality and stroke.
- Cardiac resynchronization therapy in patients with heart failure and a narrow QRS complex who received an implantable cardioverter defibrillator did not increase the number of patients with peak oxygen consumption at or exceeding 1.0 mL/kg of body weight per minute.
- In a trial comparing the thienopyridine prasugrel with clopidogrel among patients undergoing planned percutaneous coronary intervention for an acute coronary syndrome, the primary endpoint of cardiovascular death, myocardial infarction, or stroke was significantly lower in the prasugrel group compared with the clopidogrel group.
- Treatment with torcetrapib exerted a 61% relative increase in high-density lipoprotein cholesterol; however, it was also associated with an increase in systolic blood pressure of 4.6 mm Hg.
- In patients with suspected coronary disease and a calcium score of less than 600, the ability of 64-slice multidetector computed tomography angiography to assess the presence of significant coronary stenoses and identify patients likely to be referred for revascularization is similar to that of quantitative coronary angiography.