Multidetector high-speed computed tomography angiography (CTA) has been much heralded as a major diagnostic innovation of many cardiovascular systems, including the coronary and peripheral circulation. It has changed the diagnosis and management of all these cardiovascular systems. Limitations in resolution of the CT scanner (usually to 1-mm vessel size) reduce the number of vessels that are visible and the exact determination of the degree of stenosis, which would be greater on traditional angiographic studies. The detection of anomalies of the coronary arteries is important in diagnosing and treating potentially dangerous variations. The information provided by the multislice CT scan provides useful information on the origin and course of the anomalous vessel and its relationship with the great vessels before surgical or eventual coronary intervention. Variations that occur in less than 1% of the general population may be considered abnormal or anomalies.1

**MATERIALS AND METHODS**

A Philips 64-slice CT scanner (Philips Medical Systems, Best, The Netherlands) was placed in our institution in February 2007. We retrospectively reviewed the 452 coronary patients who have undergone CTA since February in an outpatient setting.

The protocol for coronary CTA includes .90-mm slice thickness cuts obtained at .45-mm intervals with a tracking marker placed upon the descending thoracic aorta. Usually, 75 mL of nonionic contrast media is injected at 5 mL/s through a large-bore, intravenous access in the antecubital fossa. Reconstructions of the raw data were performed using the Philips, as well as the TeraRecon (Tokyo, Japan) workstations. Serial axial images, as well as the reconstructed multiplanar and maximum intensity projections, were used primarily for diagnostic purposes. Three-dimensional volume rendering was also used to help diagnose patients.

Patients were referred primarily from both cardiologists and internists. Most patients had a history of coronary artery disease, peripheral vascular disease, or known carotid disease. Indications for coronary CTA included:

- Symptomatic patients with an equivocal stress test
- Filter for low-risk patients prior to coronary angiography
- Known or suspected coronary artery anomalies
- Assessment of coronary artery bypass graft patency and location

Exclusion criteria included abnormal renal function as measured by blood urea nitrogen and creatinine, as well as glomerular filtration rate calculations. Patients with elevated creatinine levels of 1.6 or greater and who required a scan were brought back at a later date (with hydration orders and possible acetylcysteine) and, if at that time their secondary labs were within normal range, they underwent scanning. If their results were still elevated, they were sent for nephrology consult.2 Once the scan was completed, patients were observed for 30 to 60 minutes and then discharged. Adverse events were monitored and treated accordingly. Physicians and technologists then reconstructed the images, evaluated the anatomy, and made the appropriate reports.

**Diagnosis of Coronary Artery Anomalies**

Sixty-four-slice CT angiography has allowed for new diagnosis and management of coronary anomalies.

**BY MICHAEL H. WHOLEY, MD; WILLIAM C. L. WU, MD, FACC; ART TONTIPLAPHOL, MD; MICHAEL GONZALES, MD; CHUN TAN, MD, FACC; AND IAN NOWAK, CRT**
RESULTS

Of the 1,300 patients who underwent CTA of the coronary arteries, 12 were adult patients with congenital heart disease, eight were treated surgically, and two had coronary anomalies. One of the cases, a tetralogy of Fallot, had an anomalous takeoff of the left coronary artery off the right sinus, with a course between the aorta and the right ventricle outflow track. The other was an Ebstein’s anomaly with right coronary artery (RCA) fistula to the bronchial artery. There were 10 other cases with anomalous coronary variants in asymptomatic patients.

Below is a description of the findings of the anomalies, including a review of normal CTA anatomy of the coronary arteries.

NORMAL ANATOMY

Right Coronary Artery

The RCA most commonly originates from the right sinus of Valsalva and continues along in the atrioventricular groove to the crux of the heart where it forms the posterior descending and posterolateral arteries.

Figure 1. Three-dimensional volume rendering from multidetector CT from an inferior oblique view shows the origin and course of a normal RCA that continues along in the atrioventricular groove to the crux of the heart where it forms the posterior descending and posterolateral arteries.

Figure 3. In a curved maximum-intensity projection, the RCA originates off the left sinus of Valsalva with the left main and courses between the aorta and the right ventricle infundibulum. This is a dangerous course because it is associated with a slit-like ostium due to a 170º takeoff and because of a stretching of the affected vessel that compromises blood flow at the ostium of the vessel.

from the proximal RCA in approximately 50% of patients. The conus artery or infundibular branch is the first ventricular branch of the proximal RCA and may have its own ostium 50% of the time from the RCA. The conus artery goes anteriorly around the right ventricular conus and ends in three short branches that may provide collaterals to the left anterior descending (LAD) artery.

Distally, the RCA provides nutrient branches to the right ventricular free wall, extending to the acute margin of the heart. The distal extent of the RCA varies and may extend posteriorly as far as the obtuse margin of the heart. In 90% of patients, the RCA supplies the posterior descending coronary artery branch at the crux of the heart, which supplies the atrioventricular (AV) node and the posterior aspect of the interventricular septum. A posterolateral branch of the RCA provides blood supply to the basal-most portion of the posterolateral left ventricular wall.

Coronary arteries almost always arise normally from the “facing” sinuses of Valsalva on either side of this point of commissural contact. Coronary arteries do not normally arise from “nonfacing” or most-distant sinus; however, variations in coronary anatomy are common.

Figure 2. Three-dimensional volume rendering from multidetector CT from an inferior oblique view shows the origin and course of a normal left anterior descending along the interventricular groove with a diagonal D1 branch. The left atrial appendage covers the left main. Also shown is the circumflex artery coursing along the left atrioventricular groove with its obtuse marginal branches.
Left Coronary Artery

Left main. The left sinus of Valsalva is the origin of the left main coronary artery. The left main does not taper and usually extends approximately 1 cm before bifurcating into the LAD artery and left circumflex artery.4 Frequently, there may be a trifurcation including the intermediate artery or ramus medians (Figure 2). The ramus coronary artery provides blood supply to the anterior left ventricular free wall. Occasionally, the left main will be absent, and the LAD and left circumflex originate separately off the left sinus of Valsalva.4

Left anterior descending artery. The LAD lies in the interventricular groove and varies in its length from a small segment to a vessel that communicates with the left PDA. The LAD provides septal branches that usually originate off the LAD in a 90º angle to provide nutrient flow to the interventricular septum. The LAD also provides epicardial branches over the anterolateral wall, which are called diagonal branches and are numbered 1 through 3 (or more).4 The LAD can end at the apex of the heart or can continue to the inferior septum.4 The termination of the LAD characteristically looks like an inverted “Y.”4

Left circumflex artery. The left circumflex artery lies in the left atrioventricular groove alongside the great cardiac vein. The left circumflex major branches are called left circumflex marginal arteries and are numbered first, second, third, and so on. Because the obtuse heart border includes the inferior and left side of the heart, these are referred to as obtuse marginal branches.

CORONARY ANOMALIES

Coronary anomalies are defined morphologically in which variation can arise in the origin, course, or termination of the coronary arteries.4 These may be isolated anomalies or related to certain forms of congenital heart disease. Coronary anomalies may cause serious events that will be listed subsequently.

Angelini’s comprehensive review identified an incidence of coronary anomalies in 5.6% of consecutive patients undergoing angiographic study.6,7 Several large studies address the frequency of minor and major coronary anomalies in different subsets of patients by varying techniques and recording methods. The most common coronary variants were split RCA (1.23%) and ectopic origin of the RCA near the right aortic sinus (1.13%). Many coronary variations, such as intramural extension or myocardial bridging of the LAD, which occurs in 5% to 25% of patients, are so common, they are not considered an anomaly.3 The incidence of incidental coronary anomalies at autopsy includes a single coronary artery in .024% and coronary arterial fistulae in .2%.3 After hypertrophic cardiomyopathy, coronary artery abnormalities are the second most common cause of sudden death in young athletes.3

High takeoff of the left or right coronary ostia, defined as the location of the ostium of the left or right coronary artery more than 1 cm above the sinotubular junction, has been described.3

ANOMALIES OF THE RCA

Anomalies of the RCA and left coronary arteries include ectopic origins of either off the pulmonary artery. Origin of the LAD from the pulmonary artery may cause cardiac ischemia. Anomalies of the RCA may include a variation of its
origination from the left anterior sinus of Valsalva with anomalous course. The RCA may originate off the left sinus of Valsalva and pass several routes. The first route is referred to as type A, in which the RCA courses anterior to the aorta; it should not produce symptoms unless disease is present. If the RCA should course between the aorta and the right ventricle infundibulum, it is referred to as type B and is a dangerous course. This particular anomaly is often associated with a slit-like ostium and an obtuse takeoff of the proximal portion of the vessel. This combination may result in ischemia during exertion due to the stretching of the affected vessel that compromises blood flow at the ostium of the vessel. Type C is when the RCA courses though the septum. In type D cases, the anomalous RCA may course posteriorly along the atrioventricular groove or retrocardiac (Figure 4). Another course that we encountered is the passage of the anomalous right coronary coursing anterior to the right ventricular outflow tract (Figure 5).

Other anomalies of coronary termination include inadequate arteriolar/capillary ramifications and fistulas from RCA, left coronary artery, or infundibular ostium.
artery to the right ventricle, right atrium, coronary sinus, superior vena cava, pulmonary artery, pulmonary vein, left atrium, left ventricle, and multiple, right, and left ventricles (Figure 6).³

The left circumflex coronary artery may also originate from the distal RCA. In this case, the left circumflex coronary artery is merely a continuation of the RCA in the posterior atrioventricular groove.³

ANOMALIES OF THE LEFT CORONARY ARTERY

A common anomaly includes the left circumflex, which arises off the right sinus of Valsalva and passes behind the aorta in the left atrioventricular groove. When the left main originates from the right sinus of Valsalva, it may course the following manners:

- Type A: the left main may pass anterior to the right ventricular outflow tract;
- Type B: it may go between the pulmonary artery and the aorta (Note: this may cause cardiac ischemia because of the expansion of the aorta and pulmonary arteries during systole);
- Type C: rarely, the left coronary may assume an intramyocardial course through the crista supraventricularis and then continue as an LAD and circumflex (Figure 7);
- Type D: the left main may circle posteriorly behind the aorta to get to the left ventricle (Figure 8).⁵

One of the most common anomalies is the takeoff of the left circumflex from the right sinus with a course that goes behind the aorta, before supplying the usual circumflex territory.⁵

Otherwise normal coronary arteries may have an intramyocardial course (ie, myocardial bridge) (Figure 9). This particular abnormality involves a variable length of the vessel and is observed most commonly in the proximal portion of the LAD coronary artery.⁵

ROLE OF CONGENITAL HEART DEFECTS

In congenital heart defects, such as those that effect the positioning of the aorta and pulmonary outflow tracts, there is a higher incidence of coronary anomalies. Disorders such as tetralogy of Fallot, transposition of the great vessels, congenitally corrected transposition, double-outlet right ventricle, and single ventricle are all suspect for coronary artery anomalies.¹ In approximately 5% of the tetralogy of Fallot patients, the LAD originates off the right sinus of Valsalva and passes anteriorly across the right ventricle.¹ Hence, the vessel lies directly in the path of the surgical ventriculostomy. Other anomalies include fistulas between the coronary artery and the pulmonary and major aortopulmonary collaterals that take off in the descending aorta (Figure 10).³ Anomalous origin of the left coronary artery from the pulmonary artery is reported in <1% of the general population. This anomaly is responsible for 18% of all cases of congestive heart failure in children younger than 2 years.⁵

FISTULAS

Major epicardial coronary arteries may terminate abnormally into one of the cardiac chambers, the coronary sinus, or the pulmonary trunk and, thus, produce fistulas (Figure 11). Coronary artery fistulas are present in 0.002% of all patients with congenital heart disease.³ These fistulas can originate from the left coronary artery system (50% to 60%), right coronary artery system (30% to 40%), or both (2% to 5%).⁵ Most fistulas (90%) drain into the right heart.⁵ A coronary fistula may occur to any adjacent vascular bed.¹ Fistulas may connect to the four chambers, pulmonary arteries, and coronary sinus systemic arteries and veins. There are many complications associated with fistulas, such as infection, congestive heart failure, ischemia distal to the fistula, and arterial rupture.¹

Figure 10. Multiplanar image shows fistula communication from the sinoatrial branch off the RCA (arrow) with the right bronchial artery in this middle-aged patient with Eisenmenger’s syndrome (A). Selective right coronary catheterization reveals the sinoatrial branch to be enlarged and leading to the left upper lung in addition to adjacent and competing bronchial arteries (B).
Large coronary artery fistulas may result in right- or left-sided cardiac volume overload with or without symptoms of congestive heart failure. The hemodynamic effects of coronary artery fistulas depend on their site of drainage, diameter, and length. Drainage into the right heart produces right-to-left shunt with dilation of the right heart chambers and increase in pulmonary resistance. Eisenmenger’s syndrome has not been reported in association with such shunts. Drainage into the left heart produces left ventricular volume overload that may mimic aortic insufficiency clinically.

CONCLUSION
The advent of multislice CTA has ushered in a new era in the diagnosis and decision-making process for the management of coronary anomalies. Most coronary artery anomalies are clinically silent and do not affect the quality of life or lifespan of the affected individuals. Specific forms of anomaly, such as the origin of the left main coronary artery from the pulmonary trunk, the aberrant course of the arteries between the great vessels in association with anomalous and slit-like ostium, and large coronary artery fistulas, may be associated with sudden death, myocardial ischemia, or congestive heart failure. As expected, there is a higher incidence of anomalies in congenital heart disease. However, with modern medicine, many of the patients who had severe congenital heart disease conditions treated as children are now presenting today for diagnosis and management of adult-related diseases, such as atherosclerotic disease. Furthermore, the increased use of CTA scanning, as in this independent facility, is allowing a larger range of patients to be examined, resulting in several new anomalous pathways.

Michael H. Wholey, MD, is a cardiovascular radiologist with the Central Cardiovascular Institute of San Antonio in San Antonio, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Wholey may be reached at (210) 271-3203; wholey@uthscsa.edu.

William C. L. Wu, MD, FACC, is Chief, Central Cardiovascular Institute of San Antonio in San Antonio, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Wu may be reached at (210) 271-3203.

Art Tontiplaphol, MD, is with South Texas Cardiovascular. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Tontiplaphol may be reached at (210) 615-7734.

Michael Gonzales, MD, is with the Central Cardiovascular Institute of San Antonio in San Antonio, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Gonzales may be reached at (210) 271-3203.

Chun Tan, MD, FACC, is with the Central Cardiovascular Institute of San Antonio in San Antonio, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Tan may be reached at (210) 271-3203.

Ian Nowak, CRT, is with the Central Cardiovascular Institute of San Antonio in San Antonio, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Mr. Nowak may be reached at (210) 271-3203.