The last three years have seen revitalization, growth and success for the Vanderbilt advanced heart failure and transplant programs. Since 2008, there have been 41 new adult heart transplant recipients and 24 pediatric recipients. Cumulative patient survival rates have exceeded 97%.

The transplant program is recognized as a Blue Distinction Center for Transplant and is in the LifeTrac Select Transplant Network, making our services available to a wider range of patients. As part of this network, patients are referred to Vanderbilt as a center of excellence for cardiac transplantation.

The Advanced Congestive Heart Failure program provides state of the art consultation and longitudinal management of our sickest patients with failing hearts. Patients should be referred at the time of first hospitalization for heart failure, with the development of New York Heart Class III or for optimization of care in difficult to treat heart failure patients. A cardiac-oncology clinic has been established, addressing the issues of chemotherapy-induced cardiomyopathy. The metabolic management of these complex patients is enhanced by vital support from faculty endocrinologists within the Cardiovascular Division. Critical to the optimal care of these patients has been the recruitment of committed nurse practitioners, nurses, financial coordinators, and a social worker, all focused on maximizing heart failure and transplant successes.

Enduring success requires training the next generation of leaders in heart failure and transplant cardiology. Vanderbilt has established an advanced heart failure/transplant fellowship program, which now provides training, leadership development and guidance of new trainees. This endeavor has already manifested tremendous successes, with multiple abstracts and papers presented over the last two years, as well as two young investigator awards from the American Heart Association in 2009 and the American College of Cardiology in 2010. (Continued on page 3)
Controversies swirl in the field of cardiology, but important questions continue to be addressed through ongoing research. At the American College of Cardiology Annual Scientific Sessions in March, multiple important studies were reported with valuable clinical insights for us as practitioners. Several of these seem particularly relevant to our readership.

Should we perform early angiography in our patients presenting with acute coronary syndromes? In patients with acute coronary syndromes (ACS), routine invasive therapy (coronary angiography +/- revascularization as indicated) proved superior to selective invasive therapy (e.g., prompted by recurrent ischemia) in a meta-analysis of ~5,000 patients from three clinical trials: RITA-3, FRISC-II, and ICTUS. At five years there was a 19% reduction in death and myocardial infarction with the strategy of routine invasive therapy. This new data strengthens the current recommendations for early invasive evaluation of patients presenting with ACS. [Fox KA. Long-term outcome of a routine versus selective invasive strategy in patients with non ST elevation acute coronary syndrome: first meta-analysis of five-year outcomes based on individual patient data (FIR Trial Collaboration)].

How long should we continue dual antiplatelet therapy (DAPT) with aspirin (ASA) and clopidogrel (Plavix) in patients receiving a drug eluting stent (DES)? Researchers presented combined results from two trials addressing this question: ZEST-LATE and REAL-LATE. In these studies 2,700 patients free of adverse cardiovascular complications one year out from DES were randomized to continuing DAPT or to ASA alone at 100-200 mg/d. At two years, the composite endpoint of cardiac death and myocardial infarction (MI) was 1.8% with DAPT and 1.2% with ASA alone (p=NS). There was no significant difference in revascularization rates, and the composite of death, MI, and stroke was higher with DAPT (3.2% vs 1.8%, p=0.051). There are many limitations to this study, including the fact that ~6% of those randomized to ASA alone actually took clopidogrel as well. Many ongoing studies are evaluating this important question. At present the consensus guidelines suggest at least 12 months of DAPT after DES placement. [Park SJ. Optimal duration of dual antiplatelet therapy after drug-eluting stents implantation: a randomized, multi-center trial].

How aggressively should we treat systolic blood pressure (SBP) in type II diabetic patients at high risk for cardiovascular disease (CVD) complications? The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7) currently recommends antihypertensive medication at SBP ≥ 130 mm Hg in diabetic patients with a goal of lowering to < 130 mm Hg. The ACCORD BP Trial enrolled 4,733 patients with type II diabetes (HbA1c 7.5-11% ) at high risk for CVD events (prior event, subclinical CAD, or ≥2 additional CVD risk factors) and SBP >130. Patients were randomized to Intensive Therapy (treatment SBP goal of <120 mmHg) versus Standard Therapy (<140 mmHg). At 4.7 years of follow-up, there was no difference in major adverse clinical events (1.9% vs 2.1% ). While observational data suggests that lower SBP is beneficial, treating with medication to achieve a SBP <130 mmHg in this high risk population may not provide additional benefit. One preliminary expert panel reaction has suggested perhaps treating to a SBP ~130 mmHg, but not adding additional medications for stricter control due to lack of demonstrated efficacy and increased cost. More aggressive nonpharmacologic lifestyle modification has been emphasized. [Cushman WC, ACCORD Study Group. Effects of intensive blood pressure control on cardiovascular events in type 2 diabetes mellitus: the Action of Control Cardiovascular Risk in Diabetes (ACCORD) Blood Pressure Trial].
Vanderbilt Heart Focus: Heart Failure and Cardiac Transplant

(Continued from page 1)

Vanderbilt has also developed multiple surgical and interventional programs which have contributed to improved outcomes in patients with advanced heart failure. For very high risk patients with ischemia complicating advanced heart failure, we have utilized short-term ventricular support in the catheterization laboratory with Tandem Heart and the Impella systems to facilitate successful percutaneous coronary revascularization procedures. A robust surgical program utilizing minimally invasive mitral valve replacement has also allowed us to relieve mitral regurgitation in advanced heart failure patients who might not otherwise be approachable surgically. Intermediate term hemodynamic support with the Thoratec Centri-Mag ventricular assist device and axillary intra-aortic balloon pumps has also been successful in bridging patients to recovery or cardiac transplant over days to weeks. We also now offer the Thoratec Heart Mate II as long-term ventricular support, with the potential to be used as “destination therapy.”

A dedicated team of medical and surgical transplant physicians ensures optimal care when transplant is required. Clinical follow-up after successful transplantation is evolving. We are now using peripheral blood molecular gene expression profile to monitor for rejection in place of more invasive techniques. This has been a successful tool for surveillance monitoring and is preferred by patients as indicated by our longitudinal Quality of Life Survey.

Clinical and basic science research opportunities are immense for staff and patients. Stem cell therapy for patients with chronic ischemic cardiomyopathy and newer pharmaceutical trials are ongoing. We also have an active cardiovascular inherited disease program evaluating genetic cardiomyopathies. Finally, we have recently established a heart failure registry database to support clinical research designed to improve our patients’ outcomes.

We also understand that responsive longitudinal care is a critical component of this program. We have established numerous outreach clinics in Middle and East Tennessee and Southern Kentucky over the past three years. We are providing patient support groups and CME opportunities locally and regionally to health care providers on all aspects of advanced heart failure and transplant care. These clinics provide advanced heart failure consults and post cardiac transplant care with ongoing education to health care providers.

The advanced heart failure/cardiac transplant team will continue to expand our repertoire of therapeutic interventions for our patients. Our commitment to basic, clinical and translational cardiovascular medicine will facilitate far reaching opportunities for our patients and for physicians in training for generations to come. The more we know and share with our patients, the better recovery platform we can offer for those impaired by this condition.

Contact Information:
Heart Failure and Cardiac Surgery:
(615) 343-9188

Cardiac Transplant Services:
866-748-1494, extension 6-3500

Vanderbilt Heart Failure and Transplant Outreach Locations:
East Tennessee Heart Consultants, Knoxville, Tenn.
East Tennessee State University Heart, Johnson City, Tenn.
Vanderbilt Heart, Lebanon, Tenn.
and Murfreesboro, Tenn.

Advanced Heart Failure/Cardiac Transplant Physicians and Nurses
Thomas DiSalvo, M.D., Daniel Lenihan, M.D., Allen Nafilani, M.D., Doug Sawyer, M.D., Ph.D., Mark Wigger, M.D., and Connie Lewis, N.P.

Cardiac Transplant Surgery Physicians
John G. Byrne, M.D., Steve Hoff, M.D., and Rashid Ahmad, M.D.

Endocrinology Physicians
Jeffrey Boord, M.D.
Atherosclerosis imaging, and cardiac computed tomography (CT) in particular, has rapidly evolved over the past several years, primarily in response to the needs for accurate non-invasive coronary angiography and early identification of subclinical coronary artery disease (CAD), allowing for early aggressive modification of risk in those at increased risk for future cardiovascular events (CVE).

Distinguishing between coronary artery calcium scoring and coronary CT angiography

There are two varieties of “cardiac CT.” Coronary artery calcium scoring (CACS) is a relatively low-radiation (radiation exposure: approximately 1-2 mSv), noncontrasted CT of the chest with dedicated evaluation of the coronary arteries. CACS is used as a screening test in asymptomatic patients in whom it has been extensively studied for risk stratification. In the absence of contrast, CACS is limited to visualization of calcified coronary artery plaque. Quantification of coronary calcium burden using an Agatston score is a helpful prognostic indicator for risk of future CVE (see Figure 1). By comparison, coronary CT angiography (cCTA) is a contrasted CT examination of the chest (radiation exposure: approximately 8-12 mSv) which enables visualization of coronary arteries and reliable exclusion of coronary artery stenosis, especially in patients with chest pain and a low to intermediate pretest likelihood of significant CAD. The use of contrast with cCTA allows detection of both calcified and noncalcified atherosclerotic plaque (see Figure 2). These distinctions between CACS and cCTA are important for clinical application of these imaging modalities. CACS is a screening, risk prediction tool whose high sensitivity renders it most useful for ruling in the presence of subclinical atherosclerosis in asymptomatic patients, whereas cCTA is a diagnostic modality whose high negative predictive value renders it most effective for ruling out obstructive CAD in symptomatic patients.

Understanding these distinctions, the purpose of this article is to discuss the clinical utility of CACS for cardiovascular risk prediction in the asymptomatic patient. That is, how may we appropriately use CACS to rule in subclinical atherosclerosis and therefore identify patients in whom more aggressive preventive lifestyle and medical strategies are likely to favorably influence long-term cardiovascular risk?
Coronary artery calcium scoring and risk prediction

CACS demonstrates independent and incremental prognostic value over traditional risk factors for the prediction of all-cause mortality and cardiovascular events.1-7 Along with the emergence of novel serum biomarkers, coronary artery calcium scoring has improved the ability to detect subclinical atherosclerosis and, more importantly, to appropriately risk stratify patients and invoke aggressive preventive lifestyle and medical strategies accordingly.

The ACCF/AHA 2007 Expert Consensus Document on CACS compiled data from multiple trials comparing CACS and CV outcomes.8 From this data, a quantitative relative risk (RR) ratio scale based on coronary calcium density as measured by Hounsfield units (HU) was proposed, reflecting an incremental relationship in which higher coronary artery calcium scores are associated with higher event rates (CHD death or MI risk, over a 3-to-5-year period) and higher RR ratios. Using a calcium score of zero as the referent, the RR ratio for a calcium score of 1-100 was 1.9 (95% CI 1.3-2.8, p = 0.001). For calcium scores of 100-399, 400-999, and ≥1000, the RR ratios were 4.3, 7.2, and 10.8, respectively (p < 0.0001 for all).8

Similarly, Wayhs reported that a calcium score of >1000 on a screening CACS in asymptomatic individuals portended a very high risk (30%) of MI or death at 4-36 months.9 This risk appeared to be greater than the risk associated with a severe perfusion abnormality on nuclear stress testing (annualized event rate of 25% vs. 7.4%, respectively; p < 0.0001). At the other end of the spectrum, even the presence of minimal coronary artery calcium (Agatston score: 1-10) doubles 10-year mortality risk, according to a study by Blaha et al.10

The absence of coronary calcification, however, does not necessarily exclude obstructive coronary stenosis. A recent study by Gottlieb demonstrated that among patients with a coronary calcium score of zero, almost 20% had at least one ≥50% non-calcified coronary stenosis at the time of cardiac catheterization within 30 days of CACS.11

Which patients are appropriate candidates for CACS screening?

In their summary recommendations, the authors of the ACCF/AHA Expert Consensus Document on Coronary Artery Calcium Scoring specifically addressed the role of CACS in asymptomatic patients with low, intermediate and high CHD risk (10-year event risks of <10%, 10-20%, and >20%, respectively). In intermediate CHD risk patients, CACS was considered reasonable, given that available evidence demonstrates incremental risk prediction information in this group. Evidence of coronary calcium would result in reclassification of these patients to a higher risk status, warranting modification of patient management. In high-risk patients, CACS was not recommended, as these patients are already judged to be candidates for intensive risk reduction (Continued on page 6)
Coronary Artery Calcium Scoring
(Continued from page 5)

therapy and a high score would not change clinical management. In low-risk patients, CACS was considered similar to "population screening," and CACS was therefore not recommended. 8

A recent study by Preis evaluated the percentage of intermediate risk patients among more than 3,500 participants in the Framingham Heart Study Offspring and Third Generation cohort participants who could potentially be reclassified as having high CHD risk based on the presence of a high CACS (as defined by ≥90th age- and gender-specific percentiles or absolute modified Agatston score of ≥100 HU). Of the intermediate risk patients, 22% had CACS ≥90th percentile and 39% had CACS >100 and were thus eligible for reclassification as high CHD risk. Interestingly, prevalence of CHD risk factors did not differ between intermediate risk participants with and without high CACS. 12

How do coronary artery calcium scores impact clinical management?

There are no current guidelines with regard to tailoring medical management based on coronary artery calcium score; however, it is widely accepted that patients with documented asymptomatic subclinical atherosclerosis should be medically managed similar to those with symptomatic CAD, in whom aspirin and statin therapy are strongly advocated. In particular, statin therapy is recommended not only for LDL-lowering, but also for cholesterol-independent, pleiotropic effects, such as atherosclerotic plaque stabilization, improvement of endothelial function, and reduction of oxidative stress and inflammation. In patients with subclinical atherosclerosis, the importance of therapeutic lifestyle changes, including heart-healthy dietary adherence and regular exercise must be counseled and reinforced. 13 Finally, strict management of other cardiovascular risk factors, using targets for those with established CAD, should be employed, such as conservative lipid (e.g. LDL <100, preferably <70) and blood pressure targets (<130/80 mm Hg). 14-15

Can CACS be used as a complementary tool with functional imaging?

CACS can be thought of as an anatomic (or perhaps quantitative) imaging modality, whereas single photon emission computed tomography myocardial perfusion imaging (SPECT MPI) is a functional cardiovascular imaging modality. Both testing modalities are imprecise in cardiovascular risk stratification. Patients with a normal stress MPI result still have a low but well-defined annual risk of cardiac death and/or MI, and most patients with a moderate or severe CACS result do not develop a subsequent cardiac event.

Current ACCF/ASNC Appropriateness Criteria recommend SPECT MPI to assess for ischemia in asymptomatic subjects with a severe (Agatston score ≥400) coronary artery calcium score. 16 A recent study by Chang et al. evaluated whether integration of CACS results with stress SPECT MPI results could improve cardiovascular risk prediction in a large cohort (>1000) of generally asymptomatic patients without clinically apparent CAD. 17 Not surprisingly, the percentage of subjects with abnormal SPECT MPI results (p<0.001) and those with a large stress-induced total (≥15%) and ischemic (≥10%) LV perfusion defect size (p<0.001) increased with increasing CACS severity. However, in subjects with a normal SPECT MPI result, CACS added incremental prognostic information, with a 3.55-fold relative increase (median follow-up: 6.9 years) for any cardiac event (2.75-fold for death/MI) when the CACS was severe (>400). 17 These findings demonstrate that although a normal SPECT MPI result predicts excellent short-term, event-free survival, long-term outcome is significantly worse if CACS is severe (>400), suggesting a complementary role for CACS testing among patients with a normal SPECT MPI result. Doing so could help identify those at long-term risk for cardiac events, in whom an earlier focus on aggressive risk factor modification and other medical therapeutic measures may be beneficial.

How often should coronary calcium scoring be performed?

A recent study by Min identified the predictors of conversion from a normal to abnormal CACS during serial CACS scans over five years, in an effort to determine the "warranty period" of a normal CACS. 18 Among individuals with initial CACS of zero, conversion to CACS >0 occurred at low frequency (25.1%) before four years. Interestingly, progression to CACS >0 was associated with age, diabetes, and smoking (p <0.01 for each). 18 No clinical predictors mandated earlier repeat CACS scanning, however. In fact, there is no data currently supporting the use of serial CACS in those with an initial CACS of zero at an interval of less than five years. Further, one could argue that if an initial CACS demonstrates evidence of coronary
In summary, coronary artery calcium scoring is an important noninvasive diagnostic tool that adds independent and incremental prognostic value over traditional risk factors for the prediction of all-cause mortality and cardiovascular events, and can be effectively used to rule in the presence of subclinical atherosclerosis in asymptomatic patients, leading to early aggressive modification of risk in those at high risk for future cardiovascular events.

Cardiac Disease in Cancer Patients: More Common Than Realized

By Daniel J. Lenihan M.D., professor of Clinical Medicine

There is tremendous overlap in the risk factor profiles of cardiology patients and patients being treated for cancer. Fortunately, advances in cancer care have yielded striking reductions in mortality in a host of malignancies, such that the five-year survival rate of all cancers has increased dramatically, from 50% in the 1970s to 66% in the late 1990s. However, as survival rates have increased, so has recognition that these survivors face cardiac complications that have a major impact on quality of life.

Furthermore, the complexity of cancer therapeutics continues to rise, with many specialized molecular markers of uncontrolled cancer growth being targeted. Not surprisingly, such treatments may have at least transient important negative effects on the metabolically active cardiovascular tissues. As a result of these dynamics, it is imperative for cardiovascular specialists to have an understanding of the prevention, identification and treatment of cardiac disease in cancer patients in order to optimize clinical outcomes.

Cardiologists broadly recognize the potential for anthracyclines or trastuzumab to induce cardiomyopathy. However, for us to provide optimal care to these complex patients we must recognize that there are actually many other cardiac complications to consider (see inset). For instance, significant rhythm disturbances including atrial fibrillation, are common during cancer treatment and chemotherapy. Monitoring the QT interval is a required method to detect early toxicity with certain chemotherapeutic regimens. Agents such as arsenic trioxide can have a profound effect on the QT interval or can result in bradycardia, requiring a permanent pacemaker. Cardiac masses, either thrombus or tumor, are frequently detected on imaging, such as echocardiography and MRI (figures 1 and 2, respectively), and present challenging clinical decisions that require an interdisciplinary approach. Cancer patients’ propensity for systemic thrombi, including pulmonary emboli and myocardial infarction, even in the face of thrombocytopenia, requires careful anticoagulation while balancing the risk of bleeding. Cardiac valvular structures can also be involved, such as tricuspid regurgitation commonly seen with carcinoid syndrome. Pericardial disease is a major consideration, sometimes progressing to life-threatening cardiac tamponade. The vasculature in general is greatly affected by systemic therapy. Radiation therapy is known to promote fibrosis and accelerated atherosclerosis in treated areas. The effect of newer cancer therapies intended to be anti-angiogenic by limiting blood supply to the tumor is likely to have remote vascular effects that may be only partially recognized at present. Several reports have demonstrated severe hypertension, heart failure and other arterial thrombotic events during anti-vascular growth factor and anti-angiogenic based therapy.

Common Cardiac Problems Encountered During Cancer Therapy

- Arrhythmias (Atrial Fibrillation, Bradycardia)
- ECG Abnormalities (QT Prolongation, ST changes)
- Thrombi (arterial and venous)
- Myocardial Infarction
- Cardiomyopathy and Heart Failure (Chemotherapy-related, Stress Cardiomyopathy)
- Valvular disease (Tricuspid Regurgitation, Endocarditis)
- Pericardial Effusion and Tamponade
- Hypertension (Especially related to Anti-angiogenic therapies)
- Masses (tumors, either metastatic or primary, or intracardiac thrombi)
- Vascular compromise (radiation related or accelerated atherosclerosis)
The optimal management of cardiac disease in cancer patients clearly needs to emphasize prevention, detection and treatment. The history of cardiac toxicity in cancer patients during a variety of therapies is often difficult to establish definitively. This is because there are often changing definitions of exactly what is considered cardiac toxicity and it is unclear how strongly the surrogate marker (typically left ventricular ejection fraction [LVEF] measured by echocardiography or nuclear imaging) used to detect this damage is related to the actual timing of chemotherapy. Several recent studies have utilized a sophisticated cardiac biomarker approach to carefully identify patients developing cardiac toxicity. It is possible that this strategy will be superior to serial LVEF measurements for the early detection of cardiac toxicity to allow optimal preventive measures for the development of cardiac disease. There is a multicenter study being initiated to test this strategy during anthracycline chemotherapy to establish whether this approach is advantageous. Most importantly, if toxicity is identified, this trial will help document the cardiac outcomes with different cardiology-based treatment choices to guide future consensus recommendations. At this time, affected patients are treated using typical medications that are known to be effective in preventing and optimally treating heart failure. Unfortunately, the strategy for using cardiac based biomarkers to detect cardiac toxicity early has not been widely adopted for a host of reasons, including difficulty in establishing the exact timing for when the serum should be drawn and a changing definition of toxicity across all of the studies.

Recent efforts have focused on achieving a consensus regarding important issues involving the overlap of cardiology and oncology, and on developing strategies to improve our collective knowledge in a clinically practical manner. We have developed the International CardiOncology Society, whose primary goal is to eliminate cardiac disease as a barrier to effective cancer therapy and to extend and improve our knowledge of these complex clinical settings. We are organizing our fourth Annual International Meeting to be held in Nashville, Oct. 7-9, in which the latest research, including developing basic and clinical studies, and a consensus document will be described and published. Several ongoing clinical trials, including a multicenter database and registry, are being organized as part of this concerned group of providers which will help to answer some of the burgeoning clinical conundrums faced by providers on a daily basis. (Continued on page 12)
Sleep-disordered breathing (SDB) affects at least 5% of the adult population and is associated with hypertension, cardiovascular disease and mortality. Typical symptoms include loud snoring and otherwise unexplained excessive sleepiness during waking hours including unintentionally falling asleep during normal activities (reading, watching TV, having a conversation). Witnessed cessation of breathing while asleep is another key feature of SDB. During sleep, episodes of airway collapse lead to intermittent hypoxia and its sequelae (increased blood pressure, inflammation, endothelial dysfunction, sympathetic activation), all of which can promote sustained hypertension, myocardial ischemia, and arrhythmias. Treatment of SDB with continuous positive airway pressure (CPAP) can favorably impact not only the symptoms of SDB itself, but also ameliorate its downstream consequences.

Over the past three decades, studies exploring the relationships between SDB and cardiac arrhythmias have shown that SDB increases the risk of atrial fibrillation (OR ~ 2-3) and ventricular arrhythmias (OR ~ 3-4), which may relate to the increased risk of sudden cardiac death (SCD) of those with SDB during overnight sleeping hours. However, whether the respiratory disturbances that are the hallmark of SDB are triggers for these arrhythmias is unknown.

Our group set out to investigate this question. Working with an interdisciplinary team of sleep physicians, biostatisticians, and cardiologists, we analyzed polysomnograms (sleep studies) from the Sleep Heart Health Study, a large multicenter longitudinal study of sleep-disordered breathing.

We identified participants who experienced discrete episodes of AF and/or non-sustained ventricular tachycardia (NSVT) during their polysomnogram. We then used the case-crossover design to determine whether respiratory disturbances can trigger paroxysmal AF or NSVT. The case-crossover method analyzes the risk of an event occurring in the context of a temporary exposure, with subjects serving as their own controls. In our study, the ‘events’ are arrhythmias (PAF and NSVT), the ‘exposures’ are respiratory disturbances (apneas or hypopneas), and the three ‘control’ (or ‘referent’) periods are derived from time spent in sinus rhythm (Figure 1).

Our study showed that the odds of an arrhythmia following a respiratory disturbance were nearly 18 times the odds of an arrhythmia occurring following normal breathing. These findings suggest a direct temporal link between respiratory disturbances and the development of PAF and NSVT.

Given our findings, it is possible that undetected and/or untreated SDB could contribute to the increasing prevalence of AF. Similarly, increased ventricular ectopy following apneas or hypopneas may lead to malignant arrhythmias that could explain the nocturnal predominance of sudden death in those with SDB.

Future research will focus on testing the hypotheses that pharmacotherapy and/or SDB treatment (with CPAP) can modify the SDB-arrhythmia relationships demonstrated in our work. Further studies are also needed to explore whether screening individuals with AF for SDB improves detection of sleep apnea and/or assists in the treatment of AF. It remains

Study Reinforces Link Between Sleep Apnea and Cardiac Arrhythmias

By Ken Monahan, M.D., assistant professor of Medicine

Our study showed that the odds of an arrhythmia following a respiratory disturbance were nearly 18 times the odds of an arrhythmia occurring following normal breathing. These findings suggest a direct temporal link between respiratory disturbances and the development of PAF and NSVT.
unclear to what extent those with SDB should undergo dedicated cardiac evaluation, but further investigation of electrocardiographic abnormalities that occur during a sleep study seems reasonable.


Cardiac Disease in Cancer Patients: More Common Than Realized

(Continued from page 9)


