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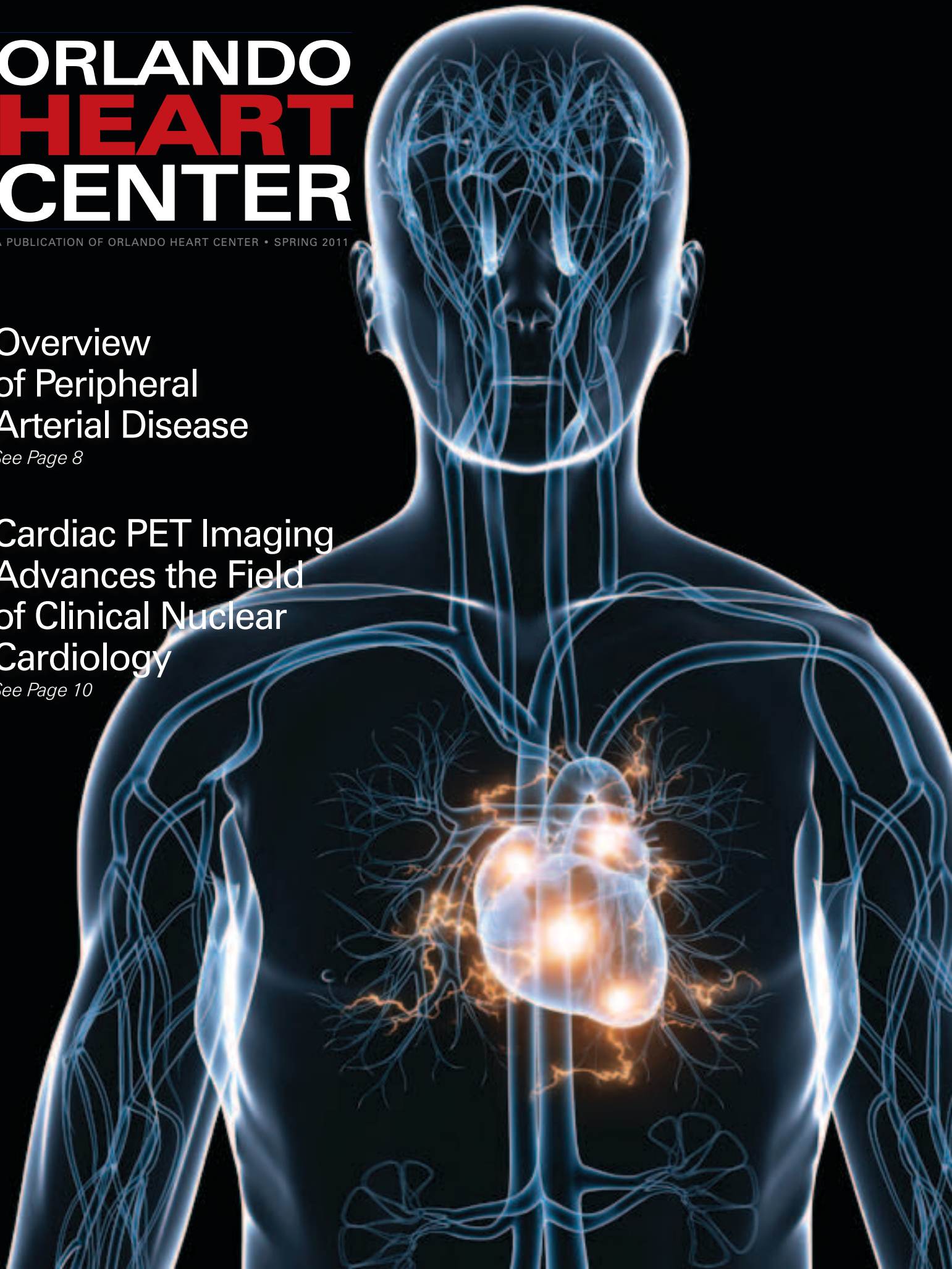
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Dear Reader,



With 22 cardiologists at four convenient locations throughout Central Florida, Orlando Heart Center offers a full spectrum of cardiovascular services and focuses on providing cost-effective, individualized, high-quality care. Since our inception in 1968, our philosophy has been to treat each patient in a customized, personal manner, using the latest technological advances available. In every aspect of our practice — from prevention to diagnosis to treatment — we remain committed to excellence.

As part of this commitment, our triage doctor service adds an added level of support to the referring physicians we serve. Through this service, we ensure that referring physicians can always reach one of the physicians in our downtown office to look at a test or answer any questions, even if the patient is not ours.

As the current managing partner, I am proud that our physicians have earned a solid reputation for quality cardiac care in the community. We are especially honored that so many physicians, nurses, hospital staff members and even other cardiologists choose Orlando Heart Center to care for themselves and their families. In fact, when someone is looking for a cardiologist and doesn't have a personal referral, I often recommend that he or she call the hospital nearest him or her that cares for cardiac patients and ask the hospital staff members where they take their families for cardiac care.

This magazine highlights our physicians' expertise and is also designed to educate you about various aspects of cardiovascular care, including feature articles on:

- Peripheral Arterial Disease
- Cardiac PET Imaging
- Syncope
- Pulmonary Hypertension
- Women and Heart Disease
- Carotid-Artery Stenting

Please feel free to contact our physicians with any comments or questions. We would be happy to discuss these topics further at any time. You can also visit our website at www.orlandoheart.com to learn more about our practice and access links to additional informative articles and sites.

As our mission statement reads, "The Orlando Heart Center physicians and associates are dedicated to providing the highest quality cardiovascular services to our patients and the growing Central Florida community. We promise to demonstrate a commitment to excellence, a caring attitude and social responsibility in all our endeavors."

Thank you for allowing us to care for your patients.

Mark A. Steiner, M.D., President/Managing Partner

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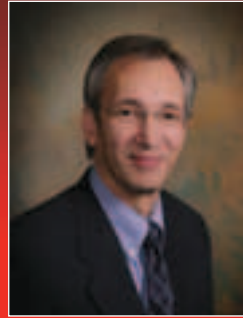
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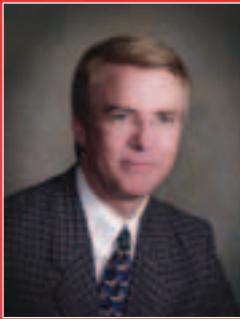
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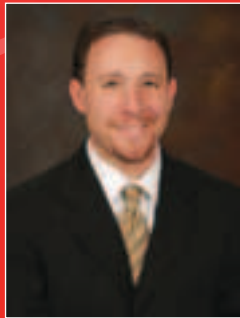
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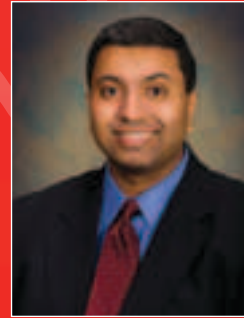
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Overview of Peripheral Arterial Disease



by Mark A. Steiner, M.D.

Peripheral Arterial Disease (PAD) refers to any pathologic process causing obstruction to blood flow in the arteries exclusive of the coronary and cerebral vessels. PAD is a common, but often undiagnosed, disease, which limits the functionality of the patient and carries risk of early vascular mortality. PAD affects approximately 20 percent of adults older than 55.¹ About half of all people with PAD are asymptomatic. The prevalence of PAD increases with age, prolonged exposure to smoking, hypertension and diabetes.

The most practical and reliable marker for estimating the prevalence of this disease is an ankle brachial index (ABI).² The ABI is the ratio of the ankle systolic blood pressure and the higher of the two brachial systolic pressures. An ABI of less than 0.90 is up to 95 percent sensitive in detecting angiogram-positive PAD.³

About one-fifth of people with PAD have typical symptoms of intermittent

ized by ongoing atherogenesis in other vascular beds and a high rate of mortality (approximately 25 percent to 30 percent within five years for patients with symptomatic PAD), due mainly to stroke and myocardial infarction (MI).⁵

The diagnosis of PAD begins with an accurate history and physical examination. Comprehensive imaging of the peripheral vasculature has traditionally been possible only with catheter-based

dominant modifiable risk factor for PAD; a dose-dependent relationship is present between smoking and severity of PAD.⁶ In patients with stable intermittent claudication, exercise significantly improves maximal walking time and overall walking ability. Multiple trials have shown substantial benefit in decreasing the incidence of new claudication and the progression of PVD with aggressive cholesterol and diabetes control.^{7,8}

Contrary to prior belief, beta-adrenergic antagonist drugs do not worsen intermittent claudication in patients with PAD. Treatment with cilostazol (Pletal) has been demonstrated to improve maximum and pain-free walking distance.⁹ A variety of strategies to stimulate new collateral channels in peripheral ischemia, such as the use of growth factors and autologous bone marrow cells, is being evaluated.

Aspirin therapy reduces the risk of MI, stroke or cardiovascular death by about one-quarter in patients with PVD.¹⁰ Aspirin has not been shown to improve claudication, but it delays the rate of progression, reduces the need for intervention and reduces graft failure in patients with prior revascularization procedures. Clopidogrel (Plavix) has been shown to reduce the risk of MI, stroke or cardiovascular death compared with aspirin. The greatest benefit was evident in the subgroup of patients with PVD.¹¹

Revascularization remains a mainstay of therapy when medical treatments fail to resolve or improve claudication symptoms. As the endovascular techniques and equipment have improved, fewer patients are requiring vascular surgery



lower-limb claudication, "rest pain," ulceration or gangrene, and another one-third have atypical exertional leg symptoms.⁴ Among individuals with asymptomatic PAD, about 5 percent to 10 percent develop symptoms of PAD over five years. Although PAD is characterized by a low rate of local symptoms and complications, it is also character-

ized by ongoing atherogenesis in other vascular beds and a high rate of mortality (approximately 25 percent to 30 percent within five years for patients with symptomatic PAD), due mainly to stroke and myocardial infarction (MI).⁵

Treatment of PAD involves risk-factor modification, exercise, medications and revascularization. Smoking is the

for revascularization. Endovascular therapies have progressed due to improvements in balloon technologies but also due to alternative modalities, including stenting, cryoplasty and athrectomy.

The self-expanding nitinol stents are resistant to compression and are ideally suited for areas such as the superficial femoral and popliteal arteries. Stent grafts also may be useful for aneurysmal disease, given their unique ability to exclude the vessel wall from the lumen. There are no approved peripheral drug-eluting stents, but studies are ongoing. Cryoplasty is a technique that combines balloon angioplasty and cold therapy by use of liquid nitrous oxide. A variety of athrectomy and laser systems have seen increasing use.

Aggressive diagnosis, prompt medical treatment and the use of newer endovascular techniques have led to improved outcomes and decreased amputation rates in patients with PVD. Risk-factor modification remains a mainstay of therapy in these patients due to their high cardiac mortality. At Orlando Heart Center, we use the latest techniques available for the diagnosis and

treatment of your patients with peripheral vascular disease.

References

1. Hooi JD, Kester AD, Stoffers HE, Overdijk MM, van Ree JW, Knottnerus JA. Incidence of and risk factors for asymptomatic peripheral arterial disease: a longitudinal study. *Am J Epidemiol.* 2001;153:666-672.
2. Faglia E, Caravaggi C, Marchetti R, et al. Screening for peripheral vascular disease by mean of the ankle-brachial index in newly diagnosed type 2 diabetic patients. *Diabet Med.* 2005;22:1310-1314.
3. Dormandy JA, Rutherford RB; TASC working group. Management of peripheral arterial disease (PAD). *J Vasc Surg.* 2000;31:S1-S296.
4. McDermott MM, Mehta S, Greenland P. Exertional leg symptoms other than intermittent claudication are common in peripheral arterial disease. *Arch Intern Med.* 199;159:387-392.
5. Criqui M, Langer R, Fronek A, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med.* 1992;326:381-386.
6. Willigendael EM, Teijink JAW, Bartelink ML, et al. Influence of smoking on incidence and prevalence of peripheral arterial disease. *J Vasc Surg.* 2004;40:1158-1165.
7. Pedersen TR, Kjekshus J, Pyorala K, et al. Effect of simvastatin on ischemic signs and symptoms in the Scandinavian Simvastatin Survival Study (4S). *Am J Cardiol.* 1998;81:333-335.
8. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional

- treatment and risk of complications in patients with type 2 diabetes. *Lancet.* 1998;352:837-853.
9. Thompson PD, Zimet R, Forber WP, Zhang P. Meta-analysis of results from eight randomized, placebo controlled trials on the effects of cilostazol on patients with intermittent claudication. *Am J Cardiol.* 2002;90:1314-1319.
 10. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ.* 2002;324:71-86.
 11. CAPRIE Steering Committee. A randomized, blinded, trail of clopidogrel versus aspirin in patients at risk of ischemic events. *Lancet.* 1996;348:1329-1339.

Dr. Mark Steiner joined Orlando Heart Center in August 2001 and is practicing at our downtown Gore Street office. Dr. Steiner completed his undergraduate degree at the University of Virginia in 1990. He received his M.D. from Washington University in St. Louis in 1994 and remained there for his internal medicine training. He completed his cardiology fellowship and interventional cardiology training at Emory University in Atlanta.

Dr. Steiner is board-certified by the American Board of Internal Medicine in internal medicine, cardiovascular disease and interventional cardiology. He is a member of the American Medical Association.

Dr. Steiner is involved in clinical research in Orlando Heart Center's active research program. His interests include both cardiac and peripheral vascular evaluation and intervention. He can be contacted at (407) 650-1300.

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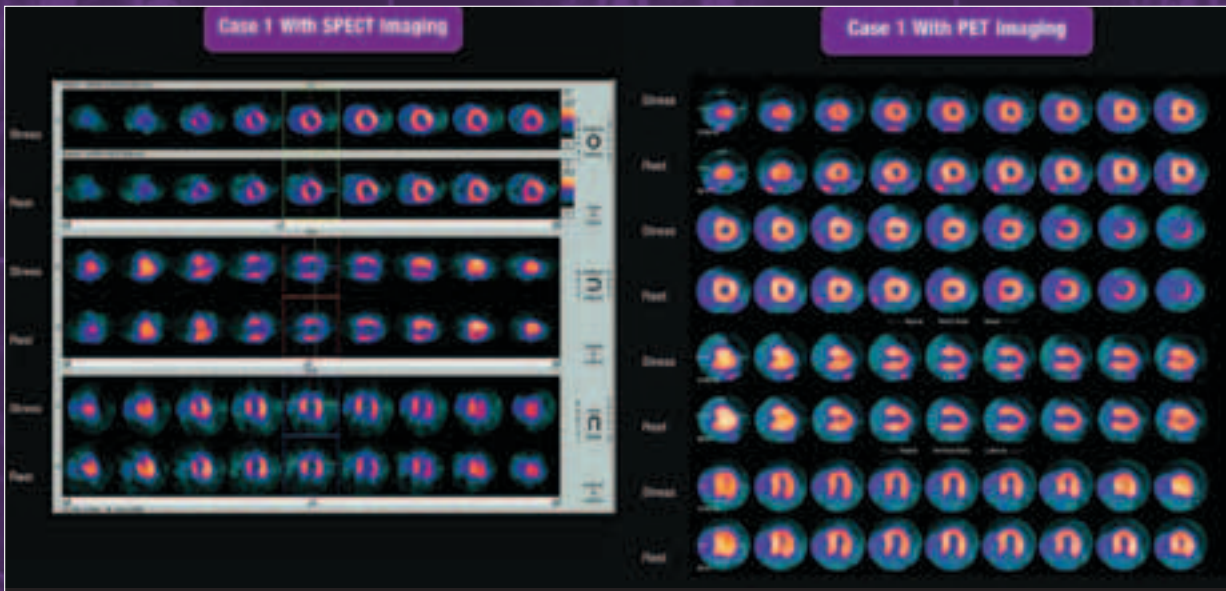
CARDIAC PET IMAGING ADVANCES THE FIELD OF CLINICAL NUCLEAR CARDIOLOGY



by Israel J. Mantecon, M.D.

Nuclear cardiology has evolved as a clinical sub-specialty during the past three decades. Its roots date back even further to the work of Blumgart and Prinzmetal and, most notably, Hal Anger for developing the first practical widely used high-resolution dynamic imaging device, the Anger camera.¹ Additional studies assisted in development of clinically useful techniques, which included first past studies and equilibrium blood pool gating to assess cardiac performance (ERNA).^{2,3,4} In addition, myocardial perfusion imaging was introduced using thallium-201, a lower-energy radionuclide that was a

major breakthrough in the development of nuclear cardiology as a viable clinical discipline.⁵ The technetium-based agents followed and provided a more optimal situation for tomography imaging, employing a single photon emission cardiac tomography (SPECT).⁶ More recently, positron emission tomography (PET) perfusion using rubidium-82 generators and pharmacological stress have received increasing attention.



No other imaging discipline has demonstrated the same rigorous and comprehensive approach to the assessment of noninvasive risk stratification that has been displayed by nuclear cardiology. In addition, as the discipline of nuclear cardiology matured, the American Society of Nuclear Cardiology (ASNC) was created and provided the field with a stature of a significant professional society. The *Journal of Nuclear Cardiology (JNC)* provided a vehicle and stimulus for development of the field.

The Certification Board of Nuclear Cardiology was created and is a well-established benchmark for the clinical practice of nuclear cardiology. Nuclear laboratory accreditation followed and established a serious emphasis on quality within the field.⁷

Nuclear stress tests are routinely performed either with exercise protocols or pharmacological drug effects, i.e., adenosine, dipyridamole, regadenosine and dobutamine. Exercise testing is the preferred modality, although pharmacological testing has been increasing yearly and is now used for more than 40 percent of patients studied.⁸ Pharmacological stress tests are reserved for patients with exercise limitations. Vasodilator stress testing is preferred in patients with left bundle branch block or electronically paced rhythms as well as recent myocardial infarction of less than 72 hours and abdominal aortic aneurysm. Dobutamine is used in patients with COPD and bronchospasm. Vasodilator agents like dipyridamole and adenosine cause a three to fivefold increase in myocardial blood inflow and both cause more increase in myocardial blood flow than exercise and dobutamine.⁹ Contraindication to pharmacological myocardial perfusion imaging is listed in Table 1. Myocardial perfusion protocols used with technetium-labeled agents can be same-day stress-rest, rest-stress or two day stress-rest. The thallium protocol is stress and four hour redistribution/reinjection. Dual-isotope protocols — i.e. rest/thallium, stress/technetium — carry a high radiation exposure to patients, and thus, more laboratories are eliminating this approach.



The major goal of noninvasive risk stratification in patients presenting with chest pain or known coronary artery disease (CAD) is the identification of high-risk sub-groups for subsequent cardiac death or nonfatal myocardial infarction who may benefit from early revascularization. In addition, low-risk patients can be spared unnecessary invasive evaluation. Prognosis for patients with suspected or known CAD depends on several variables, including the degree of left-ventricular dysfunction, the extent of CAD, the total myocardial ischemic burden and comorbidities.

A number of studies have been performed supporting myocardial perfusion images (MPI) primarily with SPECT technology in sub-groups of patients with specific issues:

1. Imaging in women – for instance, where now data exists in more than 8,000 women suggesting cardiac event rates in patients with normal stress myocardial perfusion study is less than 1 percent.¹⁰
2. Risk stratification of preoperative patient – MPI in stratifying patients at high risk, i.e. peripheral vascular disease or vascular surgery, and intermediate risk, i.e. carotid

TABLE 1

Contraindications to Pharmacologic Stress Testing

Contraindications to Dipyridamole or Adenosine

- Severe obstructive lung disease
- Second- or third-degree AV block without a functioning pacemaker
- Acute MI or unstable coronary syndrome (< 24 hours)
- Systolic blood pressure < 90 mm Hg
- Hypersensitivity to adenosine or dipyridamole
- Intake of xanthine-containing compounds within the previous 12 hours

Contraindications to Dobutamine

- Acute coronary syndrome (< 4 d)
- Severe aortic stenosis or hypertrophic obstructive cardiomyopathy
- Uncontrolled hypertension
- Uncontrolled atrial arrhythmias
- Uncontrolled heart failure
- Severe ventricular arrhythmias
- Large aortic aneurysms
- Narrow-angle glaucoma, myasthenia gravis, obstructive uropathy or obstructive gastrointestinal disorders

Elhendy A, Bax JJ, Poldermans D. Dobutamine stress myocardial perfusion imaging in coronary artery disease. J Nucl Med. 43:1634-1646, 2002.

- endarterectomy, orthopedic or prostate surgeries. Testing allows determination of which patients need to continue on to invasive procedures and those that can be optimized medically.
3. Known coronary artery disease patients – including those with percutaneous coronary intervention and coronary artery bypass surgery, and those requiring viability determinations.
 4. Patients with diabetes mellitus – identifying patients with silent ischemia and those at risk for myocardial infarction and sudden cardiac death.

Other sub-groups of patients benefiting from MPI include congestive heart failure, cardiomyopathy, cardiotoxic chemotherapy, renal failure, etc.¹¹

TABLE 2

Appropriate Patients Referred for PET MPI

- Pharmacologic stress
- Larger body habitus, especially women (attenuation artifacts)
- Prior poor-quality or non-diagnostic SPECT
- Obese patients
- Urgent work-ins

Even though SPECT MPI has been the mainstay of noninvasive cardiovascular testing for the last few decades, recent data suggests that positron emission tomography (PET) offers additional superior technology in subgroups of patients.

Bateman et al described in the *Journal of Nuclear Cardiology* (April 2006; 13:24-33) in a large population of matched pharmacological stress patients that myocardial perfusion (PET) was superior to SPECT in image quality (79 percent versus 62 percent), interpretive certainty (96 percent versus 81 percent) and diagnostic accuracy (89 percent versus 79 percent), as well as increased identification of multivessel coronary artery disease.¹² There are several potential advantages of PET MPI leading to these results, including higher spatial resolution, greater counting efficiencies and robust attenuation correction. Similar results were obtained in a meta-analysis reported in 2,442 patient studies from 1977 to 2007.¹³ At present, PET imaging's primary limitation is requirement for pharmacological testing. Appropriate patient population for PET testing is listed in Table 2. An example is shown in Figure 1 of a SPECT study with resolution of attenuation artifact following PET MPI. In addition, it is a faster study, completed in 45 minutes and with less radiation exposure than some SPECT protocols.

With today's health care environment of cost containment, a recent study compared PET MPI to SPECT MPI and looked at

downstream cost, including invasive procedures, utilization, cost and clinical outcomes. In patients matched for pretest likelihood of CAD, the study concluded PET MPI in patients with intermediate risk for CAD resulted in a greater than 50 percent reduction in invasive arteriography and CABG and a 30 percent cost savings with excellent clinical outcomes at one year compared to conventional SPECT.¹⁴

The field of clinical nuclear cardiology continues to grow and expand its role in the practice of cardiovascular diseases. SPECT and PET MPI will likely continue contributing to clinical outcomes and with respect to cost containment.

References

1. Anger HO, Van Dyke DC, Gottschalk A, et al. *The scintillation camera in diagnosis and research.* Nucleonics. 1949; 139:617.
2. Pryor DD, Harrell FE, Lee KI, et al. *Prognostic indicators from radionuclide angiography in medically treated patients with coronary artery disease.* Am J Cardiol. 1984;53:18.
3. Strauss HW, Zaret BL, Hurley PJ, et al. *A scintiphotographic method for measuring left ventricular ejection fraction in man without cardiac catheterization.* Am J Cardiol. 1971; 28:575.
4. Zaret BL, Strauss HW, Hurley PJ, et al. *A noninvasive scintiphotographic method for detecting regional ventricular dysfunction in man.* N Engl J Med. 1971; 284:1165.
5. Lebowitz E, Greene MW, Bradley-Moore P, et al. *201TI for medical use.* J Nucl Med. 1973; 14:421.
6. Zaret BL, Rigo P, Wackers FJ, et al. *Myocardial perfusion imaging with 99m-Tc Tetrofosmin: comparison to 201-Tl imaging and coronary angiography in a phase III multicenter trial.* Circulation. 1995; 91:313.
7. Iskandrian A, Garcia E (2008). *Nuclear Cardiac Imaging (4th edition).* 1, 3-8.
8. Hendel RC, Jamil T, Glover DK. *Pharmacologic stress testing: New methods and new agents.* J Nucl Cardiol. 2003; 10:197-204.
9. Beller GA, Zaret BL. *Contributions of nuclear cardiology to diagnosis and prognosis of patients with coronary artery disease.* Circulation. 2000; 101:1456-1479.
10. Cacciabaudo JH, Hachamovitch R. *Stress myocardial perfusion SPECT I women: Is it the cornerstone of the noninvasive evaluation?* J Nucl Med. 1998; 39(5):756-759.
11. Zaret B, Beller G. *Clinical Nuclear Cardiology (3rd edition).* 19-24.
12. Bateman TM, Heller GV, McGhie AI, et al. *Diagnostic accuracy of rest/stress ECG-gated Rb-82 myocardial perfusion PET: Comparison with ECG-gated Tc-99m sestamibi SPECT.* J Nucl Cardiol. 2006; 13(1).
13. Nandalur KR, Dwamena BA, Choudhri AF, et al. *Diagnostic performance of positron emission tomography in the detection of coronary artery disease: A meta-analysis.* Academic Radiology. 2008; 15(4).
14. Merhige ME, Breen WJ, Shelton V, et al. *Impact of myocardial perfusion imaging with PET and 82Rb on downstream invasive procedure utilization, costs, and outcomes in coronary disease management.* J Nucl Med. 2007; 48:1069-1076.

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Since 1994, Dr. Mantecon has been practicing at Orlando Heart Center's Sand Lake office. He can be contacted at (407) 370-5800.

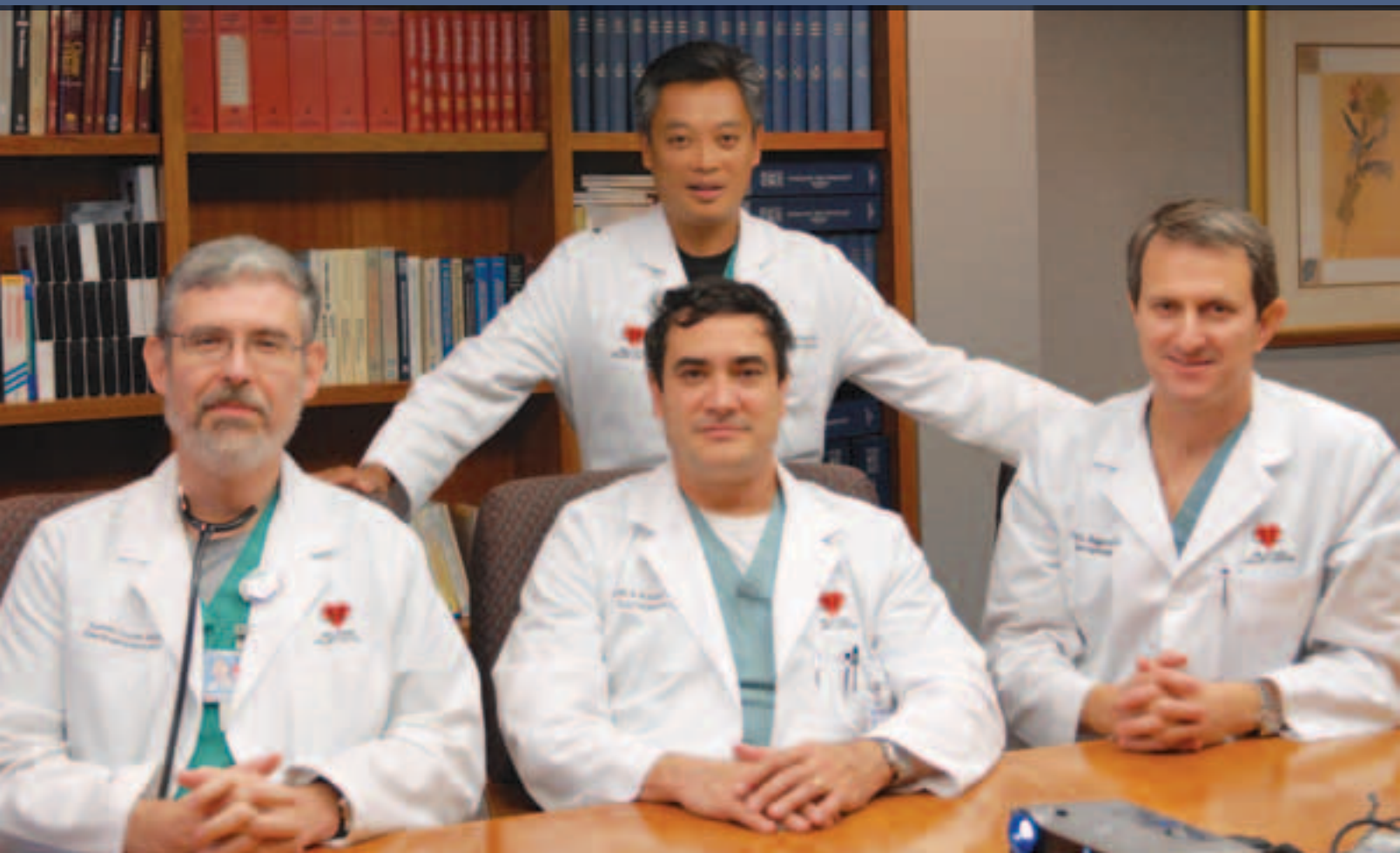
Q&A WITH THE PHYSICIANS OF CAA: SYNCOPE



What is Syncope, and How Common is This Problem?

Syncope is a transient loss of consciousness and postural tone, characterized by unresponsiveness, followed by spontaneous recovery without specific resuscitative measures. This is generally caused by a sudden and global drop in cerebral blood flow. This is a common clinical problem whose origin can often be traced to a cardiovascular event.

An analysis of 822 participants with syncope in the Framingham Heart Study reported an age-adjusted incidence of 7.2/1,000 person years.¹ A vasovagal reaction accounted for 21.2 percent of the syncopal events. Syncope is generally triggered by standing or an emotional reaction. Other causes included cardiac in 9.5 percent, orthostasis in 9.4 percent, medication in 6.8 percent and stroke in 4.1 percent. No cause was identified in 36.6 percent of cases. There is a bimodal incidence in the general population with the peaks affecting teenagers and the elderly (i.e. > 70 years old).



Estimated Prevalence of Syncope:

- Children before age 18 years old – 15 percent
- Young athletes – 6.2 percent
- Military population ages 17 to 27 years old – 25 percent
- Men and women ages 40-49 years old – 19 percent over 10 years
- Elderly ages > 70 years old – 23 percent over 10 years

According to data collected from 1992 to 2000 among emergency departments (ED) throughout the country by the Centers for Disease Control & Prevention and the National Center for Health Statistics, syncope was responsible for 7.75 percent of all ED visits. That translates to 740,000 ED

underlying structural heart disease, such as previous myocardial infarction or cardiomyopathy, but may also include supraventricular tachycardias and bradyarrhythmias.

Where Do We Begin?

The evaluation and treatment of syncope has evolved through the years with advancement in our understanding of the pathophysiology of its many causes. The main reason to evaluate patients with syncope is to determine whether the patient is at increased risk of death, especially in those with underlying heart disease in whom syncope can be a precursor for sudden cardiac death. In most patients, the reasons for the syncope can be determined from a meticulous history and physical exam. This process should

The mortality in those with structural heart disease and syncope is extraordinarily high and approaches 50 percent at five years in those who are untreated. We call this “Cardiac Cancer.” This is both a high-risk and a high-profile scenario. This is an example where appropriate intervention can save lives. For this reason, very rapidly evaluating patients felt to possibly have ischemia or myopathy is so important. Those who have known heart disease, risk factors or symptoms of angina or congestive heart failure, as well as those over 40, would merit at least an expedited, if not an inpatient, workup.

Many studies, the best of which is the Framingham study, have documented the low cardiovascular mortality in those without intrinsic cardiac abnormalities. Syncope in the patient with known heart disease should be an alarming symptom. It merits that the physician carefully assess the situation. The consequences of a mistake are quite high. Once determined to be a low-risk individual without cardiac disease, neurocardiogenic syncope becomes the most likely etiology.

Many studies, the best of which is the Framingham study, have documented the low cardiovascular mortality in those without intrinsic cardiac abnormalities. Syncope in the patient with known heart disease should be an alarming symptom. It merits that the physician carefully assess the situation.

visits annually. The overall admission rate for patients presenting with syncope is 32 percent; for patients with known heart disease, the admission rate is even higher — 66 percent.²

What Causes Syncope?

In the general population, the most common cause of syncope is vasovagal or “the common faint,” followed by primary arrhythmias. Etiologies can differ among different age groups. For example, young patients are most likely to have a neurally mediated form of syncope, conversion or psychiatric reactions and primary arrhythmias like long QT & Wolff-Parkinson-White syndromes. In the middle-age and elderly populations, neurally mediated forms of syncope may also occur but are triggered by deglutition, micturition, defecation or cough. Orthostatic hypotension due to autonomic dysfunction or medications should be evaluated. Cardiovascular causes include outlet obstructions such as aortic stenosis or pulmonary embolism. Arrhythmic causes are generally related to

include assessment of medications, especially anti-arrhythmic agents, which can cause pro-arrhythmias, as well as anti-hypertensives (i.e. diuretics). Observations by eyewitnesses can be particularly helpful. Whereas tonic-clonic seizure-like activity can be seen both with neurologic and cardiac causes of syncope, neurally mediated forms of syncope are often followed by fatigue, weakness and nausea. The absence of prodromal symptoms is suggestive of a cardiac arrhythmia.

The initial evaluation does not focus on electrical issues. During the physical exam, it is important to evaluate for orthostasis; carotid bruits; and signs of LV dysfunction, pulmonary hypertension and valvular heart disease. A good history and physical, echocardiogram and stress testing help to rapidly subdivide the high-risk from the low-risk individual. This is a crucial step. The EKG can be a useful adjunct in evaluating ischemic heart disease, structural heart disease, Wolff-Parkinson-White syndrome, long QT syndrome and Brugada syndrome.

Which Patients Require EP Studies?

An EP study is a direct assessment of the patient’s conduction system and screens for inducible arrhythmias. It is an outpatient hospital procedure that is done with mild sedation and takes approximately an hour. It is especially helpful in those whom a vagal etiology is felt to be a less likely cause of syncope. It can pinpoint numerous abnormalities that can be treated that cause loss of consciousness. These range from abnormalities of impulse formation or conduction, which would cause bradycardia, to inducible ventricular arrhythmias. EP testing, though invasive, is done via venous access, uses no dye and is very low-risk, even in very high-risk patients. After an evaluation of LV function and a screen for ischemia, it is an early tool in the evaluation of syncope for those with cardiac disease. Again, the entities we are looking for are very high-risk and treatable so patients’ lives can be protected from sudden death secondary to ventricular tachycardia. The mortality from outpatient cardiac arrest is in the 95 percent range. Identification of these individuals before this happens is critical.



What is the Role of Tilt-Table Testing in the Evaluation of Syncope?

A tilt-table test is a helpful aid in the evaluation of recurrent neurocardiogenic (formerly termed vasovagal) syncope. It employs a bed able to lift the patient from a supine position to an 80-degree upright position. The patient is secured to the bed with the help of several wide straps, and a foot board provides additional support. An intravenous line is the most invasive aspect of this test. Protocols are varied, but most employ upright posture for 10 to 20 minutes at baseline and a similar amount of time after drug stimulation. The purpose of this is to accumulate the majority of the blood volume in the leg veins, therefore mimicking a hypovolemic state. The decrease in venous return activates the sympathetic nervous system in an effort to maintain an adequate cardiac output. Drug stimulation consists of increased sympathetic stimulation with isoproterenol or a further increase in venous pooling by using sublingual nitroglycerin, a potent venodilator. The final objective is to trigger a parasympathetic reflex, which results in bradycardia and/or hypotension — which, in this extreme situation, results in syncope or near-syncope from decreased cerebral perfusion. Once the patient's clinical scenario is replicated, the table is immediately lowered, resulting in restoration of cerebral blood flow by gravity. The

test is exceedingly safe and is usually performed in an outpatient or office setting.

The test's usefulness depends on the replication of the patient's symptoms surrounding recurrent syncopal events. It is less important to achieve actual syncope than to recreate the typical symptoms that accompany syncope. Very often, patients will report diaphoresis, nausea, dyspnea, flushing, clamminess and other typical symptoms. These vary widely from patient to patient but are recognizable to the patient if they have happened to him/her before, even before bradycardia and hypotension can be detected. Syncope can be induced in many patients who have never before had it; therefore, a test is only "positive" if it also replicates the patient's symptoms. The main utility of the test is verifying that symptoms can be replicated and correspond to this relatively benign condition. A "negative" test is of little value, as it does not exclude the presence of syncope — it just tells you that, on that given instance, you were unable to produce the clinical syndrome. It is for this reason that the test cannot be used to "clear" a patient following a syncopal event. Only clinical observation on therapy, showing a prolonged absence of syncopal events, is of significance.

When Should Driving Restrictions Be Lifted?

The recommendation of restricting a patient's driving privileges because the patient may be a danger to himself or to others on the roadway is reasonable but can be a very sensitive subject. Many times it affects our patients' livelihood and quality of life. Restriction times vary from state to state but generally range from three to six months of being syncope-free after therapy has been instituted. Data gathered to formulate the latest European Guidelines would suggest that the risk for a motor vehicle accident due to syncope is low, and there is no evidence that waiting for three asymptomatic months will guarantee no further attacks. However, this is the latest consensus. Restrictions will also differ based on the vehicle and the type of driving (i.e. commercial versus private).

References

1. Soteriades ES, Evans JC, Larson MG et. al., *Incidence and prognosis of syncope*, NEJM 2002; 347:878-885.
2. Sun BC, Emanuel JA, Camargo CA Jr., *Acad Emergency Med* 2004;11:1029-1034.
3. Sorajja, D, *Cardiosource Review Journal* 2006;15: 12-15.

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PULMONARY HYPERTENSION

The Other High Blood Pressure



by James H. Tarver III, M.D.

Pulmonary Arterial Hypertension (PAH) is a rare disorder of the pulmonary vasculature that results in increasing resistance to blood flow through the lungs. It is defined as a mean pulmonary artery pressure greater than 25 mmHg at rest or greater than 30 mm of mercury with exercise. Because its symptoms mimic those of other diseases, and because standard testing in this situation is often unrevealing, the time from the onset of symptoms to diagnosis can reach up to two years. Unfortunately, pulmonary arterial hypertension is a life-threatening disease with a very high mortality if left untreated.

Formerly classified as either primary or secondary, the World Health Organization (WHO) developed a new classification system in 1998, to aid in the appropriate diagnosis and treatment of pulmonary hypertension.

Symptoms typically include dyspnea and decreases in exercise capacity. EKG and CXR abnormalities, lower-extremity edema, ascites, chest discomfort and syncope are late findings.

Severity of disease is graded based on the New York Heart Association classes for congestive heart failure. Greater severity of symptoms correlates with greater mortality.

Diagnosis of PAH requires a high clinical suspicion. Cardiac echocardiography is the screening test of choice and may reveal elevations in pulmonary artery systolic pressure, right-sided chamber enlargement and tricuspid regurgitation. It is also helpful in evaluating for left-sided cardiac disease, which may be contributing to the PAH. Additional evaluation should include a thorough history and physical examination, laboratory screening for connective tissue diseases, thyroid disorders, blood dyscrasias and hypercoagulable states, VQ scanning, pulmonary function testing with measurement of diffusing capacity and oxygenation, screening for portal hypertension, EKG and CXR. Additionally, high-resolution CT of the chest may also be helpful to exclude pulmonary fibrosis and mediastinal abnormalities; a sleep study may identify patients with sleep disorders. An echo bubble study or transesophageal echo can non-invasively detect congenital cardiac abnormalities. Functional testing in the form of a six-minute walk is helpful to determine the severity of the patient's disease and to follow the results of therapy. Prognostically, walking less than 350 meters predicts a poorer long-term prognosis.



All patients with suspected pulmonary hypertension should undergo cardiac catheterization. Echocardiography alone is inadequate to diagnose pulmonary hypertension, as it sometimes over or underestimates the severity of the disease. Catheterization also provides additional prognostic data, such as right-atrial pressure and cardiac output, which are essential in diagnosis and management. Catheterization is also used to exclude left-sided heart disease, which, if present, could complicate or increase the risks associated with standard therapies for pulmonary hypertension. Pulmonary vasodilator testing should be performed in most patients. While vaso-reactivity is present in only 10 percent to 15 percent of patients, these patients often respond well to treatment with supra-therapeutic doses of calcium channel blockers. Vaso-reactivity testing should only be performed with the short-acting, pulmonary selective agents epoprostenol, adenosine or nitric oxide. Catheterization in these patients should only be performed by clinicians experienced in the diagnosis and treatment of pulmonary hypertension.



Rapid advances in the understanding and treatment of pulmonary hypertension have occurred over the last several years. Epoprostenol (Flolan), the first available treatment for PAH, was approved as recently as 1995. While extremely expensive and complicated to use, its introduction has vastly improved the prognosis of patients with PAH. Because of its extremely short duration of action, it must be infused continuously via an indwelling intravenous catheter. Two other prostanoid compounds are now available. Treprostinil (Remodulin) improves on Flolan with a longer duration of action and is available for both intravenous and subcutaneous continuous infusion. Iloprost (Ventavis) is an inhaled prostanoid, which is administered via a nebulizer six to eight times daily.

The endothelin receptor antagonists (ERAs), bosentan (Tracleer) and ambrisentan (Letairis), as well as Sildenafil (Revatio), are oral therapies that improve symptoms and six-minute walk distance. ERAs are contraindicated in patients with hepatic disease and during pregnancy. Monthly hepatic enzyme monitoring is required. While patients with more severe symptoms (Class 4 and some Class 3) should be treated with prostanoid therapy, those with milder therapies can often be treated successfully orally.

Very close follow-up and vigilance for progression of symptoms is essential, as most patients will progress, even with therapy.

An echo bubble study or transesophageal echo can non-invasively detect congenital cardiac abnormalities. Functional testing in the form of a six-minute walk is helpful to determine the severity of the patient's disease and to follow the results of therapy. Prognostically, walking less than 350 meters predicts a poorer long-term prognosis.

For patients who fail medical therapy, lung transplantation should be considered. Dramatic improvements in symptoms, right heart function and survival are accomplished with lung transplantation; however long-term morbidity from obliterative bronchiolitis is substantial. Surgical thromboendarterectomy is a very effective therapy in patients with chronic thromboembolic pulmonary hypertension (CTEPH). This is available in only a few centers around the world.

PAH is a rare but increasingly recognized disease for which significant improvements in treatment have recently become available. Diagnosis requires a high index of suspicion, and diagnosis and treatment should be undertaken by physicians intimately familiar with the disease.

Dr. James H. Tarver III was awarded a U.S. Navy Health Professions Scholarship in 1984 and received his bachelor of arts and doctorate of medicine degrees from Boston University in 1988 through the six-year Combined BA/MD program. He completed his residency in internal medicine at National Naval Medical Center and his cardiology fellowship at National Naval Medical Center and Walter Reed Army Medical Center in 1999. During that time, he pursued his interest in pulmonary hypertension by completing additional training in this discipline at Johns Hopkins University.

Dr. Tarver has served as a general medical officer aboard the aircraft carrier USS Enterprise and as a staff internist at Pensacola Naval Hospital. Before joining Orlando Heart Center, he held positions as a staff cardiologist at National Naval Medical Center and Naval Medical Center Portsmouth, where he was active in training internal medicine residents and cardiology fellows. He is board-certified in internal medicine and cardiology and holds a clinical appointment as an assistant professor of medicine at the Uniformed Services University of the Health Sciences.

Dr. Tarver has been recognized by the American College of Physicians for his academic pursuits and is a recipient of the Navy Achievement Medal, the Navy Commendation Medal and the Joint Services Achievement Medal.

Dr. Tarver practices at Orlando Heart Center's downtown location at 60 West Gore Street. He can be contacted at (407) 650-1300.

WOMEN AND HEART DISEASE



by Linda E. Jaffe, M.D.

Cardiovascular disease (CVD) remains the number-one killer of women, surpassing all cancers combined. In many countries, including the United States, more women than men die

every year of CVD, and overall outcomes for women with coronary disease are worse than for men. In particular, 38 percent of women (versus 24 percent of men) die within one year of their first coronary event, reminding us of the need for aggressive risk modification and early disease detection in women.



The primary risk factors for women, as for men, include hypertension, diabetes mellitus, dyslipidemia, smoking and family history of early coronary artery disease. The NCEP has recognized the postmenopausal state as a risk factor for CVD in women, assigning it the same weight as male sex for men. The incidence of **hypertension** approaches 80 percent for women above age 70. **Diabetes** is a much more potent risk factor for women than men, increasing the risk of CVD three to sevenfold (versus two to threefold in men). It negates the protective effect of gender and doubles the risk of a second MI in

women. **Dyslipidemia** is a significant risk factor for both men and women, but in women, a low HDL is more predictive than an elevated LDL for CVD, and elevated triglycerides appear to be a more potent risk factor. **Smoking** has been associated with half of all coronary events in women, and even minimal use increases coronary risk. Fortunately, risk returns to baseline after two to three years, and thus, smoking cessation remains critical in the management of cardiac risk in women. **Family history** remains a non-modifiable risk factor but increases the importance of aggressive management

of other risk factors. More recently, chronic kidney disease and peripheral vascular disease have been cited as risk factors for CAD. Secondary risk factors include obesity, increasing weight within the "normal" range and metabolic syndrome (abdominal obesity, glucose intolerance, hypertension and elevated triglycerides with a low HDL). A sedentary lifestyle is also associated with increased cardiac risk.

In 2004, the AHA published evidence-based guidelines for CVD prevention in women, and these guidelines were updated in 2007. Assessment of level

of risk was fundamental to subsequent recommendations for intervention, and based on the 10-year absolute risk of CVD, patients were divided into high (> 20 percent), intermediate (10 percent to 20 percent) and low (< 10 percent) risk groups. A Framingham risk calculator has been published and can be found online at <http://hp2010.nhlbi.nih.gov/atpiii/calculator.asp?usertype=prof>. The more updated guidelines assess risk more generally, based on clinical findings. The "high-risk" group includes established CVD, known cerebrovascular disease, peripheral arterial disease, abdominal aortic aneurysm, end-stage or chronic renal disease, diabetes mellitus or a 10-year Framingham global risk of > 20 percent. Patients considered "at risk" are those with one or more of the risk factors described above, evidence of subclinical vascular disease (e.g. coronary calcification), poor exercise capacity on treadmill testing and/or abnormal heart-rate recovery after stress testing. The guidelines emphasize lifestyle interventions, which include a low-fat diet; the addition of omega-3 fatty acids, particularly in women with high triglycerides; physical activity with a goal of 30 minutes a day; maintenance of appropriate weight; smoking cessation; cardiac rehabilitation following acute cardiac events; and screening for depression. Additional recommendations include an ideal BP goal of < 120/80; treatment of dyslipidemia, preferably with statins, to achieve an optimal LDL determined by risk category; and subsequent intervention as needed to achieve an HDL > 50 and triglyceride levels less than 150. Diabetes should be treated to attain a HBA1c level < 7 percent.

The issue of when to use aspirin has sparked some interest in recent years. Clearly, aspirin at a dose of 75 to 325 mg/d is recommended in all high-risk women, including every diabetic. Patients who are truly intolerant to aspirin should be considered for clopidogrel therapy. The use of aspirin in low-risk women has been controver-



sial. As recently as 2004, aspirin was not considered appropriate therapy in low-risk women, although a beneficial effect on the risk of ischemic stroke had been demonstrated. More recently, the recommendation has been to use low-dose aspirin (81 mg daily or 100 mg every other day) in all women with a 10-year Framingham risk score of 6 percent to 10 percent. Additionally, in women 65 years of age or older, aspirin therapy should be considered if blood pressure is controlled and benefit for ischemic stroke and MI prevention is likely to outweigh risk of GI bleeding and hemorrhagic stroke. In women under 65, aspirin appears beneficial for ischemic stroke prevention, but not for MI prevention.

The consideration of hormone replacement therapy (HRT) has undergone much change. Although it is known that LDL and triglyceride levels increase after menopause and HDL decreases concurrently, and we know that these changes correlate with increased CV risk, HRT has not been shown to offset this risk. Whereas multiple studies have failed to show a net benefit on CV outcomes with HRT, more recent analyses have suggested that early treatment in the perimenopausal years may have modest benefit. Recent recommendations support limited use of HRT as needed for perimenopausal relief for the shortest time period and at the lowest dose possible, beginning soon after onset of symptoms. There

remains no role for HRT as preventive therapy for CVD.

Evidence-Based Guidelines for CVD Prevention in Women

J Am Coll Cardiol. 2004 Mar 3;43(5):900-21. Circulation. 2007 Mar 20;115(11):1481-501.

Dr. Linda Jaffe received her bachelor of arts degree in psychology at the State University of New York at Albany in December 1977. Dr. Jaffe then attended the University of North Carolina at Chapel Hill, where she received her doctorate in medicine in May 1984. Her post-doctoral training included an internship and residency in internal medicine at the Columbia Presbyterian Medical Center in New York, which she completed in 1987. After finishing her residency program in internal medicine, Dr. Jaffe completed a fellowship in cardiovascular disease at the Long Island Jewish Medical Center in June 1990.

Dr. Jaffe returned to Columbia Presbyterian Medical Center after her fellowship to become director of the Cardiology Diagnostic Center at the Allen Pavilion. She held an appointment as assistant professor of medicine at Columbia University and was an active member of the teaching service in the Department of Medicine. Dr. Jaffe is board-certified in internal medicine and cardiovascular diseases. Dr. Jaffe is also certified in nuclear cardiology.

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CAROTID-ARTERY STENTING

A PROMISING NEW TREATMENT FOR CAROTID ARTERY DISEASE



by Deepak P. Vivek, M.D.

The standard practice for the treatment of significant carotid artery disease has been carotid endarterectomy

(CEA). CEA has been proven to reduce stroke in those patients with recent stroke symptoms and > 50 percent diameter stenosis (NASCET trial) of the carotid artery or those patients without symptoms and > 60 percent stenosis of the carotid artery (ACAS trial). However, these trials of CEA only included carefully selected patients with carotid disease and excluded patients with significant comorbidities. Moreover, the results of the landmark NASCET¹ and ACAS² trials have not been reproduced reliably in community practice where surgical expertise varies. Therefore, a less-invasive endovascular technique, such as carotid-artery stenting with embolic protection, has recently been studied as alternative in patients felt to be at elevated risk for CEA.



The recently published SAPHIRE³ study found that carotid stenting with embolic protection provided equivalent, if not better, outcomes in these high-risk patients. Based on this study and other carotid stent registries, the FDA has approved carotid stenting for patients felt to be at high risk for CEA. Stenting is indicated for high-risk patients who are asymptomatic and have > 80 percent stenosis or who are symptomatic and have > 50 percent stenosis. However, Medicare has only approved reimbursement for high-risk symptomatic patients with > 70 percent stenosis unless the patient has been enrolled in a clinical trial.

What constitutes a "high-risk" patient for CEA? Patients can be designated as elevated risk for CEA for surgi-

cal considerations or due to medical comorbidities. For example, surgical factors that would place a patient at high risk for CEA include: previous radiation therapy to the neck or previous head and neck surgery, restenosis after previous CEA, contralateral carotid occlusion, high or very low carotid bifurcation, laryngeal nerve palsy, severe tandem stenoses or presence of a tracheal stoma. Significant medical comorbidities such as recent MI (< 30 days), class III-IV congestive heart failure, significant LV dysfunction (EF < 35 percent), severe COPD and advanced age (> 80 years) are medical factors that would increase a patient's risk for CEA.

Embolic protection devices are now used routinely in all carotid stenting

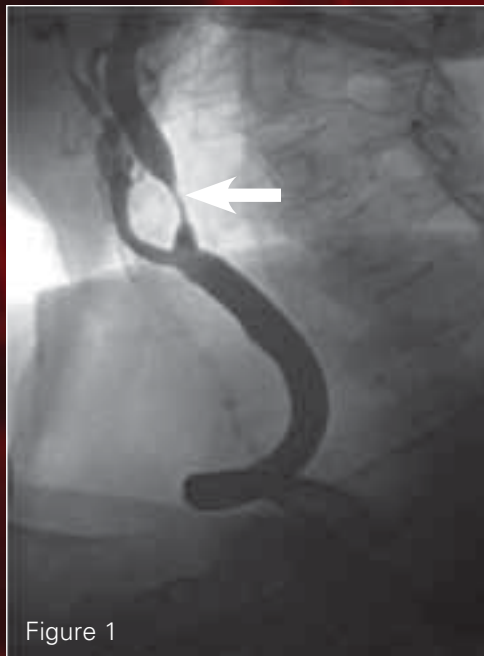


Figure 1



Figure 2

procedures. These devices are essentially small filters that are placed distal to the carotid stenosis during the interventional procedure. These devices help to capture debris that may be liberated during the angioplasty and stenting procedure. The filter protection device is then removed at the end of the procedure. The stents utilized for an interventional carotid procedure are composed of nitinol, a highly flexible metal alloy. As for any stenting procedure, all patients need to tolerate both aspirin and clopidogrel with clopidogrel needed for a minimum of one month post-procedure.

CASE STUDY

A 64-year-old male with active tobacco use, a history of head and neck carcinoma s/p radiation therapy, and head and neck surgery five years ago presents for evaluation of carotid bruit. The patient also had a laryngeal nerve palsy related to his previous head and neck surgery. A carotid duplex was performed, which demonstrates a severe 80 percent to 99 percent right internal carotid-artery stenosis with mild left internal carotid disease. The patient's history of radiation therapy increases the risk of neurological complications with CEA. Therefore, carotid stenting was thought to be a more suitable alter-

native. The patient underwent a carotid angiogram, which demonstrated a severe 95 percent stenosis of the RICA (figure 1). We performed a successful carotid artery stenting procedure with marked improvement in lumen diameter and no periprocedural neurological events (figure 2). The patient has now done well in follow-up without any TIA/stroke symptoms.

Orlando Heart Center provides comprehensive vascular testing, including non-invasive imaging and invasive angiography and carotid stent placement. We have access to the latest clinical trials in carotid stenting, allowing patients across the spectrum of cardiovascular risk to be treated. Please contact our office at (407) 650-1300 for further information.

References

1. NASCET Investigators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *NEJM* 1991;325:445-53.
2. ACAS Investigators. Endarterectomy for asymptomatic carotid artery stenosis. *JAMA* 1995;273:1421-8.
3. Yadav et al. Protected carotid-artery stenting versus endarterectomy in high-risk patients. *NEJM* 2004;351:1493-501.

Dr. Deepak P. Vivek attended the University of North Carolina School of Medicine, where he earned his bachelor of arts degree in chemistry in 1993 and a doctorate in medicine in 1997. His post-doctoral training included an internship and residency program in internal medicine at the University of Washington, which he completed in 2000. In June 2003, Dr. Vivek completed a fellowship in cardiovascular diseases at the Cleveland Clinic Foundation. In 2005, Dr. Vivek completed a fellowship at the Cleveland Clinic Foundation in interventional cardiology, specializing in coronary, peripheral and carotid intervention.

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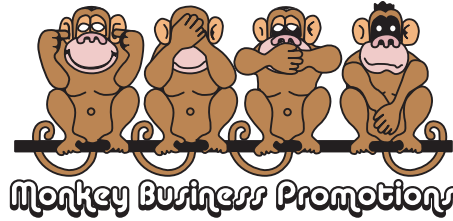
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