Oklahoma Heart Institute

VOLUME 7 | NUMBER 1 | SPRING 2012

Your Heart Matters

Reduce Your Heart Attack Risk
By Wayne N. Leimbach, MD

Is Your Leg Pain Peripheral Arterial Disease?
By Raj H. Chandwaney, MD

Heart Failure: Signs, Symptoms and Treatment Options
By Alan M. Kaneshige, MD

Sudden Cardiac Death The Dangers of Rapid Heart Rhythms
By Craig S. Cameron, MD

Varicose Veins: How to Find Relief
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The Beat Goes On: What You Need to Know About Atrial Fibrillation
By Gregory A. Cogert, MD

Every Woman's Greatest Health Risk
By Eugene J. Ichinose, MD

Spring Recipes Your Heart Will Love

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to our readers

Having an understanding of heart disease and vascular disease empowers people to optimize their health and prevent serious illness. In addition, understanding treatment options helps people navigate the complex health care system and achieve the best care available.

The current issue of Oklahoma Heart Institute magazine presents simple, yet effective, ways to reduce one's risk of a heart attack. For those wondering whether their leg pains are due to poor circulation, Dr. Chandwaney reviews the symptoms, diagnostic tests and treatment strategies related to peripheral artery disease. In addition, Dr. Robert Smith discusses varicose veins and symptomatic venous disease, another cause of painful legs.

Heart failure is the number one cause of admission to hospitals in the United States. Dr. Kaneshige, Director of the Heart Failure Clinic at Oklahoma Heart Institute, discusses the signs, symptoms and latest treatments for patients with heart failure.

Rhythm abnormalities can affect the quality of life, as well as cause sudden death. Doctors Cogert and Cameron, both electrophysiologists at Oklahoma Heart Institute, highlight the newest treatment strategies for two significant rhythm problems.

Finally, Dr. Ichinose discusses the issues concerning heart attack risks for women. In addition, delicious heart healthy recipes are provided to demonstrate that not all delicious foods are unhealthy.

We hope you enjoy the articles and welcome any comments or suggestions regarding the magazine content.

Sincerely,
Wayne N. Leimbach, Jr.
Publisher, Oklahoma Heart Institute Magazine

Heart Attack?

Ways to Reduce Your Risk

By Wayne N. Leimbach, MD, FACC, FSCAI, FCCP, FAHA

The disease that kills the most Americans each year is actually preventable in the majority of cases. It's heart disease, and it causes heart attacks in about 1.2 million people in the United States each year.

If heart disease is treatable for most people and can be diagnosed early, why are so many of us having heart attacks?

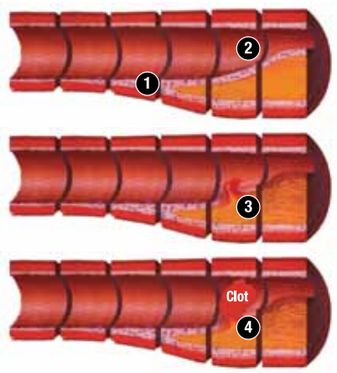
Many people don't know they are at risk. Others who actually know their risk factors don't treat them. Still others who know their risk factors do not seek testing to assess whether or not they already have significant heart disease that needs treating.

WAYS TO PREVENT A HEART ATTACK

Heart attacks can be prevented by treating the known risk factors that lead to your having one.

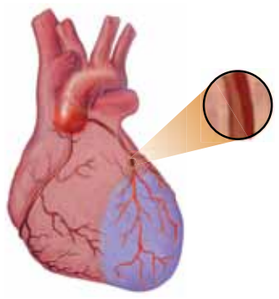
Figure 1

Atherosclerosis: The Risk of High Cholesterol



1. Initially, as atherosclerotic plaque builds up in the artery, the vessel wall stretches to maintain the vessel lumen.
2. Eventually, as the plaque builds up, the vessel lumen narrows.
3. Plaque rupture exposes the blood to the plaque contents and promotes formation of a blood clot.
4. If the blood clot that forms on the ruptured plaque is large enough to occlude the vessel lumen, then a heart attack occurs.

Figure 2



A heart attack occurs when a blood vessel to the heart muscle becomes totally blocked causing the heart muscle downstream from the blockage to die.

First, you should know your blood cholesterol and triglyceride levels (lipids).

Second, you should know your blood pressures and blood sugar. If these levels are elevated, blockages are probably being made in the blood vessels that provide blood supply to the heart.

We now know blockage formation can start as early as in the teenage years. The formation of blockages in the blood vessels to the heart can also be caused by smoking, chronic inflammation as measured by a blood test called the high sensitivity–CRP (hs-CRP), and a sedentary lifestyle.

Your risk of having a heart attack increases as blockages continue to develop in the blood vessels supplying the heart muscle. In most cases, blockage material (plaque) in the blood vessels to the heart muscle ruptures, and a blood clot forms on the ruptured plaque, causing the blood vessel to become completely blocked. This causes heart muscle to die. If a large amount of heart muscle dies, the person will die (Figure 1).

WAYS TO TREAT YOUR RISK FACTORS

Preventative cardiology is important. Here's why. If blockages in the blood vessels to the heart are never made, then blockages cannot rupture, and a blood clot cannot form on the ruptured plaque to block the blood supply to the heart muscle. Thus, a heart attack is prevented.

The goal, then, to preventing heart attacks is preventing blockage formation in the blood vessels to the heart.

You can do this by treating your major risk factors: high lipids (cholesterol and triglycerides), high blood pressures, high blood sugars, cigarette smoking, high hs-CRP (marker of inflammation), and sedentary lifestyle (Table 1).

KNOW YOUR NUMBERS

What are good cholesterol levels? For people at mild risk, LDL cholesterol (the bad cholesterol) should ideally be less than 130. For people with known blockages of the blood vessels and/or with diabetes, the LDL cholesterol levels should be definitely less than 100 and, if possible, less than 70 (Table 2).

What should your blood pressure be? Ideal blood pressures are less than 120/80. The goal for most people is blood pressures at rest consistently to be less than 140/90 (Table 3).

What should your blood sugars be? For most people fasting blood sugars should be less than 110, and ideally less than 100. The blood test called a high sensitivity-CRP is a marker of inflammation. It also indicates increased risk of blockage formation. The hs-CRP should be less than 2, and ideally less than 1.

Almost everyone's blood pressure, cholesterol levels and blood sugars can be normalized today.

WATCH YOUR DIET

You should also follow a low-cholesterol, low-saturated fat diet to reduce cholesterol levels. A low-sodium diet will help reduce blood pressures. A low simple carbohydrate diet helps reduce the risk of diabetes and lowers triglycerides. Weight loss in overweight patients significantly reduces the risk of diabetes. If your risk factors cannot be normalized with dietary changes and lifestyle modification, then medications should be used.

Studies have shown that if risk factors are normalized, not only can blockage formation be stopped, but also reversal of already existing blockages can occur.

Table 1

Major Treatable Risk Factors for Heart Attacks and Stroke

- High Blood Pressure
- High Lipids (Cholesterol & Triglyceride)
- High Blood Sugars (Diabetes Mellitus)
- Smoking
- High hs-CRP (Marker of inflammation)
- Sedentary Lifestyle
- Low HDL (Good Cholesterol)

Table 2

Cholesterol ATP-III Guidelines Quick Reference

LDL — Cholesterol ("Bad" Cholesterol)	
<70	Optimal if diabetic or CAD
<100	Optimal if any risk factors present
<130	Near optimal
130-159	Borderline high
160-189	High
≥190	Very high
Total Cholesterol	
<200	Desirable
HDL Cholesterol ("Good" Cholesterol)	
<40	Low (↑ risk)
≥60	Good (↓ risk)

Table 3

Blood Pressure Classifications

Blood Pressure Classification	Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)
Normal	<120	and <80
Pre-Hypertension	120-139	or 80-89
Stage 1 Hypertension	140-159	or 90-99
Stage 2 Hypertension	≥160	or ≥100

Table 4

Heart Attack Symptoms Can Include:

- Chest pain, discomfort, heaviness, tightness
- Indigestion symptoms not relieved by antacids
- Discomfort radiating to the left shoulder, arm, jaw, or back
- Discomfort or heaviness of the left arm or both arms
- Sweating, nausea, vomiting
- Extreme weakness or shortness of breath
- Rapid or irregular heart beats

TESTING.

For people who are having symptoms suggestive of heart disease (Table 4) or for people with multiple untreated risk factors, tests should be considered that screen for the presence of already severe blockages in the blood vessels to the heart. These include stress tests, calcium CT scans or CT angiograms. If severe blockages are present, they can be treated before a heart attack occurs.

HEART ATTACK INTERRUPTED

Finally, if you are having symptoms that may be those of a heart attack, call 911, so you can be taken immediately to the hospital.

If the diagnosis is confirmed at the hospital, the heart attack can be interrupted by emergency cardiac catheterization and balloon angioplasty and/or stenting. Most heart attacks can be interrupted, if the patient gets to the hospital early enough. This not only prevents death, but also minimizes the amount of damage done to the heart, so that the person can return to their normal lifestyle.

TAKING ACTION

It is important that we take an active role in our healthcare by knowing our risk factors for heart attacks, aggressively treating those risk factors and making sure symptoms suggestive of heart disease are evaluated.

Only in this way can you reduce your personal risks, thus reducing the number one cause of death in the United States.

Dr. Leimbach is a specialist in interventional cardiology, including cardiac catheterization, coronary angioplasty, percutaneous closure of PFOs & ASDs and related interventional procedures such as stents, atherectomy, laser, intravascular ultrasound imaging and direct PTCA for acute myocardial infarction.

Peripheral Arterial Disease

Is Your Leg Pain Related to Cardiovascular Disease?

By Raj H. Chandwaney, MD, FACC, FSCAI, FSVM

Are you having leg pain but don't know what's causing it? It could be lower extremity peripheral artery disease (PAD), which, actually, is very common. Just what is PAD?

Lower extremity PAD disease refers to the presence of plaque accumulation in the blood vessels that deliver blood to the feet. These blood vessels may include the distal abdominal aorta, iliac arteries, common femoral arteries, superficial femoral arteries, popliteal arteries, and/or infrapopliteal arteries (Figure 1). The plaque accumulation in these blood vessels is due to a disease called atherosclerosis. Atherosclerosis is also the disease that causes heart attacks when the plaque accumulates in the blood vessels that feed the heart. Atherosclerosis causes strokes if the plaque accumulates in the blood vessels that feed the brain.

PREVALENCE

Lower extremity PAD is present in 25-30% of people over age 70. The disease is also present in 25-30% of high-risk individuals over age 50. Individuals who have a history of diabetes or tobacco use are considered to be at high risk for developing lower extremity peripheral arterial disease.

SYMPTOMS

The classic symptom of lower extremity peripheral artery disease is called *claudication*, described as a tightness that occurs in the thighs or calves while walking. Interestingly, claudication only occurs in 10% of patients who have lower extremity peripheral arterial disease.

Atypical leg symptoms refer to any other type of leg discomfort that may occur in patients with lower extremity peripheral arterial disease. *Atypical leg symptoms* occur in 30% of patients with lower extremity peripheral arterial disease. Sixty percent of patients with lower extremity peripheral arterial disease are asymptomatic. Despite the fact that the majority of patients with lower extremity peripheral arterial disease are asymptomatic, it is very important to diagnose the disease in these asymptomatic individuals.

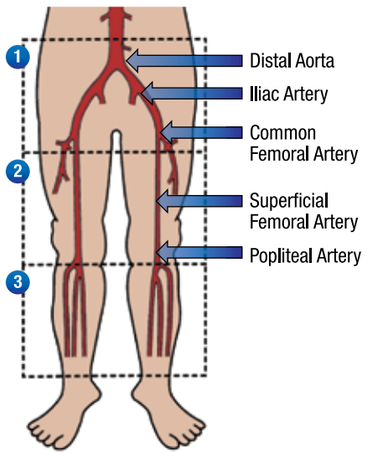
MORTALITY

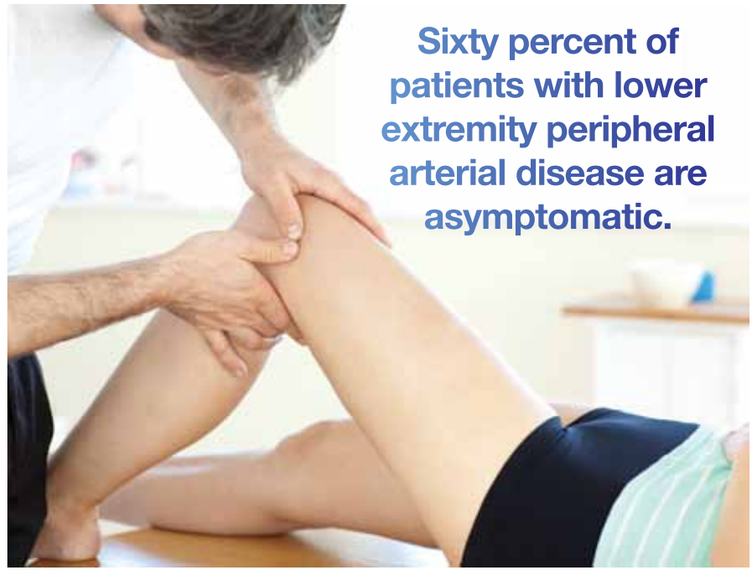
Mortality rates are four times greater amongst individuals with lower extremity peripheral arterial disease compared to individuals without the disease. The increased risk of death is equally present in lower extremity peripheral arterial disease patients with or without symptoms. The five-year mortality rate for patients with lower extremity peripheral arterial disease is 25% (one of four patients with the disease are dead in 5 years if not treated).

Seventy-five percent of the deaths that occur in patients with lower extremity peripheral arterial disease are cardiovascular deaths (predominantly heart attack and stroke). Patients with lower extremity peripheral arterial disease are at high risk for heart attack and stroke because individuals with plaque accumulation in the lower extremity arteries are very likely to have plaque accumulation in the arteries that feed the heart and brain. Atherosclerosis is a systemic disease. This means it is usually present throughout the body rather than in just one area of the body.

Figure 1

Blood vessels that provide blood supply to the feet





Sixty percent of patients with lower extremity peripheral arterial disease are asymptomatic.

RISK FACTORS

The risk factors for lower extremity peripheral arterial disease are similar to the risk factors for heart attack and stroke. These risk factors include: tobacco use, diabetes, high blood pressure, high cholesterol, family history of atherosclerosis, and advanced age. For unclear reasons, tobacco use and diabetes carry a much higher risk for lower extremity peripheral arterial disease than the other risk factors. The risk of developing lower extremity peripheral arterial disease is equal amongst men and women. Certain races are at higher risk for developing lower extremity peripheral arterial disease, such as African Americans.

DIAGNOSIS

The diagnosis of lower extremity peripheral arterial disease is readily established with the use of the Ankle-Brachial Index (ABI). The ABI is the perfect screening test because it is safe, cheap, accurate, and readily available. The ABI can be measured with the use of a simple hand-held Doppler by obtaining the systolic blood pressure in the ankle and brachial (arm) arteries. In my opinion, the ABI is the ideal test to establish the diagnosis of atherosclerosis, in asymptomatic individuals. Experts suggest performing a screening ABI on all individuals over age 70, and high-risk individuals over age 50. Screening ABIs facilitate the early diagnosis of systemic atherosclerosis in asymptomatic individuals.

When patients have symptoms that are concerning for lower extremity peripheral arterial disease, more sophisticated diagnostic tests may be required to diagnose and treat the patient's symptoms. These diagnostic tests include duplex ultrasound, magnetic resonance angiography, CT angiography, and invasive angiography.

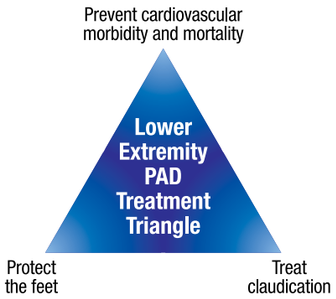
TREATMENT

The treatment of patients with lower extremity peripheral arterial disease is best summarized with the *Lower Extremity PAD Treatment Triangle,* which is demonstrated in Figure 2. The most important priority in the care of patients with lower extremity peripheral arterial disease involves addressing the high risk of cardiovascular mortality. This priority is emphasized on the *Lower Extremity PAD Treatment Triangle* by the placement of this priority at the top of the triangle. Strategies used to address the high risk of cardiovascular mortality in patients with lower extremity peripheral arterial disease include:

- Smoking cessation
- Antiplatelet therapy (aspirin or clopidogrel)
- Cholesterol control
- Hypertension control

Figure 2

Treatment



- Diabetes control
- Therapeutic Lifestyle Changes (heart healthy diet, routine exercise, weight loss)
- Flu shot annually

The next priority to be addressed in patients with lower extremity peripheral arterial disease is protecting the feet from amputation.

The final priority to be addressed in patients with lower extremity peripheral arterial disease is the treatment of leg pain that may be due to claudication. This priority is demonstrated on the *Lower Extremity PAD Treatment Triangle* located at the right lower corner of the triangle. Claudication symptoms can be treated with exercise rehabilitation, pharmacologic therapy, and/or revascularization.

Formalized exercise rehabilitation programs lasting 3 to 6 months have been proven to increase patient walking distances 100-150%. Rehabilitation sessions typically last 35 to 60 minutes. Patients are instructed to walk at an intensity that causes pain in 3 to 5 minutes, followed by rest until pain resolution, followed by walking again.

Pharmacologic therapy for claudication involves the prescription of cilostazol at a dose of 100 mg twice daily. Cilostazol has been proven to increase patient walking distances by 50%. Cilostazol has limited use with many lower extremity peripheral arterial disease patients because of common side effects that include headache, diarrhea, dizziness, and palpitations. Also noteworthy is that cilostazol has a black box warning contraindicating its use in patients with a history of congestive heart failure.

Finally, revascularization (restoration of blood flow) may be required in patients with lower extremity peripheral arterial disease who do not achieve adequate symptom relief with exercise rehabilitation or pharmacologic therapy. Revascularization strategies include endovascular techniques (balloon angioplasty, stents, atherectomy, or laser) and surgical techniques (bypass surgery).

CONCLUSIONS

Lower extremity peripheral arterial disease is a disease with a high prevalence. Most patients with lower extremity peripheral arterial disease are asymptomatic or have atypical symptoms. Patients with lower extremity peripheral arterial disease have significantly higher mortality rates (25% five year mortality) compared to the general population. Patients with lower extremity peripheral arterial disease die of cardiovascular diseases such as heart attack and stroke. Lower extremity peripheral arterial disease can easily be diagnosed in most patients with a cheap, safe, and simple screening test in the office (ABI). Treatment of all patients with lower extremity peripheral arterial disease should *primarily focus on lowering their risk of cardiovascular death.* Patients with lower extremity peripheral arterial disease require routine foot exams and diligent foot care. Some patients with lower extremity peripheral arterial disease will require treatment to improve claudication symptoms (exercise, pharmacologic, and/or revascularization). A smaller minority of patients will require revascularization to treat critical limb ischemia or acute limb ischemia.

Raj. H. Chandwaney is an interventional cardiologist with expertise in cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures. In addition to board certifications in cardiovascular disease and interventional cardiology, Dr. Chandwaney is also board certified in endovascular medicine by the American Board of Vascular Medicine.

The diagnosis of lower extremity peripheral arterial disease is readily established with the use of the Ankle-Brachial Index (ABI). It is safe, cheap, accurate, and readily available at Oklahoma Heart Institute.

Heart Failure:

Signs, Symptoms and Treatment Options

By Alan M. Kaneshige, MD, FACC, FASE

Heart failure (HF) is a major healthcare issue for the United States. Because of the aging population, HF is one of the three cardiac conditions increasing in prevalence, the others being degenerative valvular heart disease and atrial fibrillation. There are 5.8 million patients with symptomatic HF in the United States and an estimated 23 million patients worldwide. Patients between the ages of 40 to 80 have a one in five chance for developing HF during their lifetime. Approximately 670,000 patients will be diagnosed with HF this year and 283,000 deaths will be attributed to HF. Acute decompensated HF will account for 1.1 million hospital admissions. In 2010, total HF costs amounted to $39.2 billion of which $21 billion was for hospitalizations alone.

To emphasize the severity of HF, it is believed that 20% of patients will die within one year of their diagnosis.

The most common cause of heart failure today is coronary artery disease.

Risk Factors that Contribute to the Development of Heart Failure

- Cigarette smoking
- Diabetes
- Obesity
- Obstructive sleep apnea

WHAT IS HEART FAILURE?

Heart failure is a cardiac syndrome, a collection of symptoms that occurs when the heart is not able to meet the metabolic needs of the body. In most cases, HF occurs when the heart is not able to pump enough blood to meet the demands of the body (reduced ejection fraction).

Normal hearts will pump out about 60% of the blood that fills the left ventricle with each heartbeat (ejection fraction 60%). Hearts with ejection fractions of less than 50% are considered impaired.

About half of HF cases are due to diastolic dysfunction, the inability of the left ventricle to fill with blood due to increased wall stiffness. In this condition, symptoms of HF occur even with normal pump function. Because of the inability of the heart to keep up with the demands of the organ systems, powerful neurohormones such as epinephrine, norepinephrine, endothelin, and aldosterone are released to maintain adequate perfusion. Persistently high levels of these neurohormones cause progressive cardiac dysfunction and ultimate failure.

WHAT CAUSES HEART FAILURE?

The causes of HF are numerous. The most common cause of HF today is coronary artery disease. Other common causes of HF include hypertension and valvular heart disease, particularly degenerative aortic and mitral valve disease.

Risk factors that contribute to the development of HF include cigarette smoking, diabetes, and obesity and obstructive sleep apnea. Prevention of HF requires early detection and treatment of these conditions.

WHAT ARE THE SYMPTOMS?

The most common symptom caused by HF is shortness of breath (dyspnea). A patient with HF will initially notice dyspnea with exertion. The effort required to cause dyspnea becomes less as HF progresses. Dyspnea may eventually occur at rest. Fatigue is another common symptom associated with HF.

Over time, the HF patient will need to keep his or her head elevated when lying down in order to breathe comfortably and not feel smothered. This symptom is known as orthopnea. The patient may suddenly awaken at night from a sound sleep with a sense of breathlessness, even panic, as lung congestion occurs because of HF. That sensation is known as paroxysmal nocturnal dyspnea.

With later stages of HF, patients will notice discomfort in the right upper quadrant of their abdomen due to swelling of the liver, leading to nausea, anorexia, and the feeling of fullness before consuming a complete meal. Abdominal bloating and lower extremity swelling are late signs of HF as these represent fluid retention and dysfunction of the right ventricle. Another sign of HF is the onset of palpitations and dysrhythmias and may lead to loss of consciousness (syncope) and possibly sudden death.

HOW IS HEART FAILURE TREATED?

Treatment for HF is based on a multi-level approach involving lifestyle changes, education, medical therapy, and close follow-up with specialists, as well as a HF specialty clinic.

Lifestyle Changes

Lifestyle changes are important to the successful treatment of HF. Adjustments include sodium/salt restriction to two grams or less a day, diet, regular exercise, and weight reduction. Diet therapy encompasses the American Heart Association low fat diet, the Mediterranean diet, and the DASH diet as ways to maintain adequate nutrition. Regular exercise, such as moderate-paced walking 30 minutes a day, five days a week, prevents deconditioning and weight gain.

Avoiding nonsteroidal anti-inflammatory medications (NSAIDs) is important as these medications, both prescription and non-prescription, contribute to salt retention, swelling, and kidney dysfunction, all poorly tolerated by the HF patient. Advanced HF patients will even have to follow a fluid restriction of two liters or less a day to avoid fluid retention and progression of symptoms.

Medical Therapy

Medical therapy for the HF patients is a cornerstone for maintaining quality of life and to increase lifespan, as well as slow disease progression. Diuretics mobilize salt and water from the body. Angiotensin converting enzyme inhibitors (ACE inhibitors), angiotensin receptor blockers (ARBs), beta-blockers, and aldosterone inhibitors block the effects of harmful neurohormones that lead to HF progression. Physicians try to maximize these medications to approach the dosages that have shown benefit in large clinical trials.

Advanced Therapies

When HF patients show progression of symptoms and failure to respond to conventional lifestyle and medical treatments, more advanced therapy can be instituted. For some patients, biventricular pacing devices improve heart function. Ultrafiltration is utilized in volume-overloaded HF patients not responding to diuretic therapy. With ultrafiltration, fluid can be removed at a controlled rate so as not to significantly impact kidney function or blood pressure.

Surgical treatment for advanced HF includes high-risk coronary artery bypass surgery to attempt to improve pump function. For HF patients with severe mitral regurgitation, mitral valve repair can reshape the failing left ventricle and improve symptoms. Left ventricular assist devices (LVADs) are now approved for select refractory end-stage HF patients for prolonging life without the possibility of transplant (called destination therapy). These LVADs are also used to bridge advanced HF patients to transplant.

The ultimate surgical therapy at this time is a cardiac transplant. Because of limited organ supply, this option is only available to very select, advanced, end-stage HF patients. The totally artificial heart remains investigational.

Lifestyle Changes

- Sodium restrictions to 2 grams a day or less
- Blood pressure control
- Smoking cessation
- Avoidance of alcohol
- Diet and weight reduction
- Exercise 30 minutes daily, five days-a-week
- Avoidance of nonsteroidal anti-inflammatory medications (Naproxen, Ibuprofen, Meloxicam)

Medical Therapy For Heart Failure

- Diuretics
  - Furosemide (Lasix)
  - Bumetanide (Bumex)
  - Torsemide (Demadex)
- Beta Blockers
  - Carvedilol (Coreg)
  - Metoprolol Succinate (Toprol XL)
  - Bisoprolol (Zebeta)
- Aldosterone Inhibitors
  - Spironolactone (Aldactone)
  - Eplerenone (Inspra)
- Angiotensin Converting Enzyme Inhibitors (ACE Inhibitors)
  - Captopril (Capoten)
  - Enalapril (Vasotec)
  - Fosinopril (Monopril)
  - Lisinopril (Prinivil, Zestril)
  - Quinapril (Accupril)
- Angiotensin Receptor Blockers (ARBs)
  - Candasartan (Atacand)
  - Valsartan (Diovan)

CONCLUSION

Heart failure is a costly and growing problem for our healthcare system. Aggressive medical therapy, lifestyle changes, and education, as well as close medical following are key features to effective treatment. Advanced treatment is available to patients with progressive disease. A combined effort is needed to adequately provide a good quality of life and survival of the HF patient.

It is believed that 20% of patients will die within one year of their heart failure diagnosis.

Alan M. Kaneshige is a noninvasive cardiologist with expertise in adult, stress and transesophageal echocardiography. He is Chief of the section of Cardiology at Oklahoma Heart Institute Hospital, where he is Director of the Congestive Heart Failure C.A.R.E. Center and Adolescent and Adult Congenital Heart Clinic.

Sudden Cardiac Death: The Dangers of Rapid Heart Rhythms

Understanding Ventricular Tachycardia and Premature Ventricular Complexes

By Craig S. Cameron, MD, FCC, FACC

Sudden cardiac death is the leading cause of death in the United States. It accounts for more deaths than stroke, lung cancer, and breast cancer combined. When the normal electrical activation of the heart is interrupted by a rapid heart rhythm in the bottom chamber of the heart (ventricular tachycardia), sudden cardiac death occurs. Symptoms of ventricular tachycardia, VT, may include palpitations, light-headedness, dizziness, loss of consciousness, or seizures. VT is diagnosed using an electrocardiogram or heart monitor, and may indicate significant underlying heart disease.

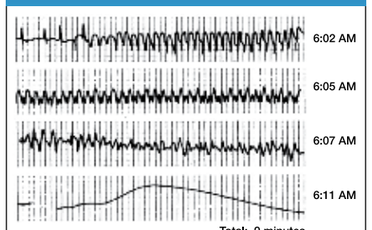
Individuals with a prior heart attack and reduced "pump function," are at particularly high risk for sudden cardiac death from VT. For this reason, patients with VT usually require additional cardiac testing to assess the risk of sudden death and to determine treatment options. Such testing may include an echocardiogram, stress test, cardiac MRI, cardiac CT, or heart catheterization.

IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS (ICDS)

The cornerstone of treatment for VT is to electrically shock the heart and reset its electrical activity. Unfortunately, most cases of sudden cardiac death progress too rapidly to allow sufficient time for emergency medical services to arrive and administer a life-saving shock (Figure 1). Thus, the implantable cardioverter-defibrillator (ICD) was developed and has revolutionized the prevention of sudden cardiac death. Current ICD models are capable of diagnosing and treating VT within seconds. While ICDs are highly effective at treating VT and saving lives, they do not address underlying heart disease and thus do not prevent VT from occurring. Furthermore, ICD shocks are painful and may result in reduced quality of life and psychological stress. ICD patients who have frequent VT often require either medications or a procedure called catheter ablation to reduce the burden of VT and likelihood of receiving electrical shocks.

Figure 1

Onset of Ventricular Tachycardia with Rapid Progression to Sudden Cardia Death
Patient Dies within 9 Minutes



ANTIARRHYTHMIC MEDICATIONS

Antiarrhythmic medications have demonstrated limited benefit for VT. Such "heart rhythm medications," have not been shown to prevent sudden death and are only modestly effective at reducing VT burden and preventing ICD shocks. Additionally, antiarrhythmic medical therapy is often limited by drug interactions and extensive side effects. In some cases, antiarrhythmic medications may actually cause VT! Alternatively, catheter ablation is rapidly becoming a suitable and often superior therapy for preventing ICD shocks.

CATHETER ABLATION

Catheter ablation of VT is a minimally invasive procedure performed by an electrophysiologist ("heart rhythm doctor") at specialized centers across the country, including Oklahoma Heart Institute.

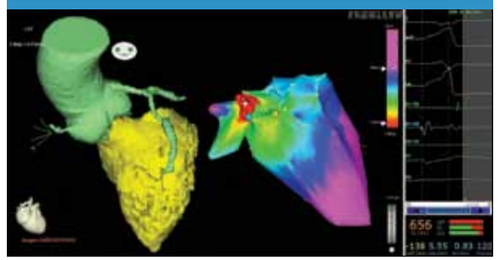
It is performed in the electrophysiology procedure room under sedation or general anesthesia using a variety of tools which include X-ray fluoroscopy, intracardiac ultrasound, and 3-dimensional mapping systems. Small catheters are introduced from blood vessels in the groin and positioned within the heart. These catheters are then used to record the heart's electrical activity and determine the source for the VT. Once identified, the abnormal tissues in the heart are targeted for radiofrequency ablation to eliminate VT.

The procedure generally lasts for 3-6 hours depending upon the number of abnormal electrical circuits identified and how easily they are eradicated. Overall, catheter ablation successfully reduces VT burden by more than 75%, thereby reducing ICD shocks. Despite the critically ill nature of many patients, VT ablation is generally well tolerated with a 1.5% risk of major complications.

Patients without heart disease who have VT are said to have "idiopathic VT." Such patients are usually considered low-risk for sudden cardiac death and treatment is thus directed at controlling individual symptoms. Within this group of patients, isolated "extra beats," from the bottom chamber of the heart called premature ventricular complexes (PVCs) are typically more common than actual VT. PVCs may cause palpitations, fatigue, shortness of breath, and lightheadedness. Individuals with very frequent PVCs (>10-20% of total beats on a 24 hour heart monitor) are at risk for developing a PVC-induced cardiomyopathy or "weakening of the heart's pump function." Thus, in addition to assessment of cardiac structure, such patients should also be considered for heart monitoring to quantitate their PVC burden. In some patients, lifestyle modification (e.g., caffeine reduction) is effective at reducing symptoms. Others require additional therapy with medications or catheter ablation.

Figure 2

3-Dimensional image integration of CT scan (left), electroanatomic data (middle), and local electrogram (right) to localize focal ventricular tachycardia to the left coronary cusp of the aorta. On the electroanatomic map, the earliest activation is in red progressing to late activation in purple. Successful ablation locations (maroon dots) are shown.



As with VT in structural heart disease, antiarrhythmic medications have limited efficacy but may be useful in some patients. Idiopathic PVCs/VT tends to be a focal problem arising from predictable locations within the heart. As such, many of these patients are excellent candidates for catheter ablation. In the absence of significant heart disease, the success rate of catheter ablation for frequent PVCs or VT approaches 90%. Catheter ablation is therefore very effective at treating patient symptoms from idiopathic PVCs/VT. Also, weakening of the heart from

Continued on p. 17

Oklahoma Heart Institute

Services of Oklahoma Heart Institute

Interventional Cardiology

- Cardiac Catheterization
- Coronary Angioplasty
- Multivessel Angioplasty and Stenting
- Atherectomy
- Rotablator Atherectomy
- Thrombolytic Therapy
- Coronary Stents
- Carotid Stenting
- Fractional Flow Reserve
- Intravascular Ultrasound
- Myocardial Biopsy
- Pericardiocentesis
- Peripheral Angioplasty
- Peripheral Stents
- Percutaneous ASD Closures
- Percutaneous PFO Closures
- Impella Circulatory Support
- Therapeutic Hypothermia for Cardiac Arrest Patients
- Venous Ablation

Noninvasive Cardiology

- CT Angiography
- CT Heart Scan
- Cardiac and Vascular Screening Services
- Nuclear Cardiology
- Echo and Doppler Studies
- Nuclear and Echocardiographic Exercise and Pharmacological Stress Testing
- Retinal Imaging
- Thyroid Ultrasound
- Transesophageal Echocardiography, Arterial Venous Peripheral Vascular Imaging and Doppler Studies
- Peripheral Arterial Doppler and Duplex Imaging
- Cardiovascular Magnetic Resonance Imaging
- External Counterpulsation (ECP) Therapy
- Transcranial Doppler
- Aquapheresis Therapy

Electrophysiology

- Electrophysiology Studies
- Ablation Therapy
- Pacemaker Implantation
- Pacemaker and Lead Extraction
- Pacemaker Programming
- Pacemaker Monitoring and Clinic
- Implantable Cardioverter Defibrillator (ICD) Replacement
- ICD and Hardware Removal
- ICD Programming
- ICD Monitoring and Clinic
- Holter Monitoring and Interpretation
- 30 Day Cardiac Event Monitors
- Implantation and Interpretation of Long-Term Heart Monitors
- Signal Averaged EKGs and Interpretation
- Head Up Tilt Testing and Interpretation
- Direct Current Cardioversion
- Antiarrhythmic Drug Loading and Monitoring

Metabolic Disorders

- Diabetes
- Thyroid
- Hypertension
- Other Endocrine Problems

Specialty Clinics

- Advanced Center for Atrial Fibrillation
- Dysrhythmia and Pacer Clinic
- Hypertension Clinic
- Adolescent and Adult Congenital Heart Clinic
- Lipid and Wellness Clinic
- Heart Failure Clinic
- Same Day Appointment Clinic
- Pre-Operative Clinic
- Center for the Treatment of Venous Disease
- Sleep Care

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Board certified in Internal Medicine, Cardiovascular Disease and Interventional Cardiology

Robert C. Sonnenschein, MD, FACC, ASE, RVT

Dr. Sonnenschein specializes in echocardiography and noninvasive peripheral vascular imaging. He is past Director of Peripheral Vascular Ultrasound Imaging at Hillcrest Medical Center and Oklahoma Heart Institute and serves as Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. He completed his Cardiology Fellowship at the State University of New York Upstate Medical Center in Syracuse, where he also completed his Internal Medicine Internship and Residency programs. Dr. Sonnenschein received his medical degree from Rush Medical College in Chicago and his Bachelor of Arts degree from the University of Pennsylvania.

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Dr. Lynch is a specialist trained in noninvasive and invasive cardiology with a special interest in the prevention of cardiovascular disease. He is former Chief of Cardiology at Hillcrest Medical Center, where he also has served as Chief of Medicine and President of the medical staff. Dr. Lynch is former Co-Director of the Lipid and Wellness Clinic at Oklahoma Heart Institute and Director of the Executive Health Program. Dr. Lynch is also a Clinical Assistant Professor at the University of Oklahoma College of Medicine – Tulsa. He completed his Cardiology Fellowship, as well as his Internal Medicine Internship and Residency, at the University of Oklahoma Health Sciences Center. Dr. Lynch received his medical degree from the University of Oklahoma School of Medicine and his Bachelor of Science degree from the University of Tulsa. Before establishing his practice in Tulsa, he served as Chief of Medicine at the U.S. Army Hospital, Bangkok, Thailand.

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James J. Nemec, MD, FACC

Dr. Nemec is a specialist in echocardiography, stress echocardiography and nuclear cardiology. He serves as Director of Nuclear Cardiology for Oklahoma Heart Institute. Dr. Nemec has served as Assistant Professor of Internal Medicine, Division of Cardiology, at Creighton University and as Assistant Professor, Department of Radiology, also at Creighton University. He completed his Clinical Cardiology Fellowship at the Cleveland Clinic Foundation and his Internal Medicine Internship and Residency at Creighton University. Dr. Nemec also completed a year of training in pathology at the University of Missouri, Columbia, MO. He received his medical degree from Creighton University, where he also received his Bachelor of Arts degree.

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Dr. Johnsen is an interventional cardiologist with expertise in cardiac catheterization, angioplasty and related interventional procedures, such as stents and atherectomy. He is Director of Cardiac Rehabilitation at Hillcrest Medical Center and Director of the Hillcrest Exercise and Lifestyle Programs. He completed his Clinical Cardiology Fellowship at the University of Oklahoma – Oklahoma City, where he then finished an extra year of dedicated training in interventional cardiology. He completed his Internal Medicine Internship and Residency training at the University of Oklahoma – Oklahoma City, where he also received his medical degree. Dr. Johnsen received his Bachelor of Science degree from Oklahoma State University.

Board certified in Internal Medicine, Cardiovascular Disease and Interventional Cardiology

Alan M. Kaneshige, MD, FACC, FASE

Dr. Kaneshige is a noninvasive cardiologist with expertise in adult echocardiography, stress echocardiography and transesophageal echocardiography. He is Chief of Cardiology at Oklahoma Heart Institute, where he is Director of the Congestive Heart Failure C.A.R.E. Center and the Adolescent and Adult Congenital Heart Clinic. He is past Chief of Cardiology at Hillcrest Medical Center. Dr. Kaneshige completed his Internal Medicine Internship and Residency at Creighton University School of Medicine, where he also received his medical degree. He received a Bachelor of Science in chemistry at Creighton University. Dr. Kaneshige completed his Clinical Cardiology fellowship at Creighton, where he also served as Chief Cardiology Fellow for two years. He completed an additional Cardiac Ultrasound Fellowship at the Mayo Clinic in Rochester. Dr. Kaneshige served as Assistant Professor of Medicine at Creighton University School of Medicine, where he was Director of the Noninvasive Cardiovascular Imaging and Hemodynamic Laboratory.

Board certified in Internal Medicine, Cardiovascular Disease, Adult and Transesophogeal Echocardiography

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Dr. Martin is a noninvasive cardiologist with specialty expertise in non-invasive imaging. He is Director of Cardiovascular Magnetic Resonance Imaging at Oklahoma Heart Institute and Hillcrest Medical Center. In addition, he is a Clinical Associate Professor of Medicine at the University of Oklahoma College of Medicine – Tulsa. Dr. Martin has specialty training in Nuclear Medicine, as well as additional training dedicated to Cardiovascular Magnetic Resonance Imaging. He completed his Cardiology Fellowship at the University of Alabama. Dr. Martin's Internal Medicine Internship and Residency training were performed at Temple University Hospital in Philadelphia. He received his medical degree from the Medical College of Ohio. Dr. Martin completed his Master of Science degree in mechanical engineering at the University of Cincinnati and his Bachelor of Science degree in physics at Xavier University. Dr. Martin is a founding member of the Society of Cardiovascular Magnetic Resonance and is an editorial board member of the Journal of Cardiovascular Magnetic Resonance.

Board certified in Internal Medicine and Cardiovascular Disease

Roger D. Des Prez, MD, FACC

Dr. Des Prez is a noninvasive cardiologist with specialty expertise in echocardiography, nuclear cardiology and cardiac computed tomography. He is Director of Cardiac Computed Tomography at Oklahoma Heart Institute Hospital, at Hillcrest Medical Center and Bailey Medical Center. Dr. Des Prez received his medical degree and Bachelor of Arts degree from Vanderbilt University. He completed his Residency in Internal Medicine and Pediatrics at University Hospital of Cleveland. Dr. Des Prez practiced for six years as an internist with the Indian Health Services in Gallup, NM. He returned to Vanderbilt University as a member of the Internal Medicine Faculty, at which time he also completed his cardiology training.

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Board certified in Internal Medicine, Endocrinology and Metabolic Diseases

David A. Sandler, MD, FACC, FHRS

Dr. Sandler is a cardiologist with subspecialty expertise in electrophysiology, complex ablation, and atrial fibrillation management. Dr. Sandler is Director of Electrophysiology at Oklahoma Heart Institute Hospital. He completed his Cardiac Electrophysiology Fellowship and his Cardiovascular Medicine Fellowship at New York University Medical Center, New York, NY. Dr. Sandler performed his Internal Medicine Internship and Residency at Mount Sinai Medical Center, New York, NY. He earned his medical degree from Georgetown University School of Medicine in Washington, DC. Dr. Sandler received his Bachelor of Arts degree at the University of Pennsylvania in Philadelphia.

Board certified in Internal Medicine, Cardiovascular Disease and Cardiac Electrophysiology

Raj H. Chandwaney, MD, FACC, FSCAI, FSVM

Dr. Chandwaney is an interventional cardiologist with expertise in cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures. Dr. Chandwaney is Director of the Chest Pain Center and Cardiology Telemetry Unit at Oklahoma Heart Institute Hospital. He completed his Clinical Cardiology Fellowship at Northwestern University Medical School in Chicago, IL., where he also completed an Interventional Cardiology Fellowship. Dr. Chandwaney's Internal Medicine Internship and Residency were performed at Baylor College of Medicine in Houston, TX. He received his medical degree from the University of Illinois at Chicago. Dr. Chandwaney completed his Master of Science degree at the University of Illinois at Urbana-Champaign, where he also received his Bachelor of Science degree.

Board certified in Internal Medicine, Cardiovascular Disease, Interventional Cardiology and Endovascular Medicine

D. Erik Aspenson, MD, FACE, FACP

Dr. Aspenson is a subspecialist in Endocrinology, Metabolism and Hypertension at Oklahoma Heart Institute, with expertise in diabetes, lipids, hypertension and thyroid diseases. He completed a fellowship in Endocrinology at Wilford Hall Medical Center, Lackland AFB, Texas. Dr. Aspenson's Internal Medicine Internship and Residency were completed at David Grant Medical Center, Travis AFB, California where he served as Chief Resident. He received his medical degree from the University of Oklahoma and his Bachelor of Science degree at Oklahoma State University.

Board certified in Internal Medicine, Endocrinology and Metabolic Diseases

Frank J. Gaffney, MD, FACC

Dr. Gaffney is an interventional and noninvasive cardiologist with subspecialty expertise in transesophageal echocardiography, nuclear cardiology, and coronary angiography. He completed his Cardiovascular Medicine Fellowship at Scott & White Memorial Hospital in Temple, Texas. Dr. Gaffney completed his Internal Medicine Internship and Residency at Brooke Army Medical Center in San Antonio. He then remained on staff at Scott & White Memorial Hospital for several years, before entering his Fellowship in Cardiovascular Medicine. Dr. Gaffney earned his medical degree from New York Medical College, Valhalla, New York, and he received his Bachelor of Arts degree at Hofstra University in Hempstead, New York.

Board certified in Internal Medicine, Cardiovascular Disease and Nuclear Cardiology

Eric G. Auerbach, MD, FACC

Dr. Auerbach is a general cardiologist who is particularly interested in preventative cardiology and cardiovascular risk reduction. He completed his cardiology fellowship at the University of Miami/Jackson Memorial Hospital in Miami, FL, following which he obtained additional subspecialty training in cardiovascular MRI, nuclear cardiology, and cardiac CT imaging. His areas of expertise also include echocardiograpgy, transesophageal echocardiograpgy, stress testing, and management of lipid disorders. Dr. Auerbach's Internal Medicine Internship and Residency were performed at the University of Miami/Jackson Memorial Hospital. He earned his medical degree at the University of Miami, Miami, FL, and his Bachelor of Arts degree at Princeton University, Princeton, NJ.

Board certified in Internal Medicine, Cardiovascular Disease, and Nuclear Cardiology

Kelly R. Flesner, MD

Dr. Flesner is a subspecialist in Endocrinology, Metabolism and Hypertension at Oklahoma Heart Institute, with expertise in diabetes, lipids, hypertension and thyroid diseases. Prior to joining Oklahoma Heart, she was at St. John Medical Center in Tulsa. She completed her fellowship in Endocrinology at the University of Texas at Galveston. Her Internal Medicine Internship and Residency were completed at the University of Texas in Houston, where she also received her medical degree. She earned her Bachelor of Science degree at Texas A&M University in College Station, TX.

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Robert L. Smith, Jr., MSc, MD, FACC, FSCAI

Dr. Smith specializes in interventional cardiology including cardiac catheterization, coronary angioplasty, and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound, and peripheral vascular interventional procedures. He completed an Interventional Cardiology Fellowship at the University of Florida College of Medicine in Jacksonville, FL. Dr. Smith performed his Clinical Cardiology Fellowship at Vanderbilt University School of Medicine in Nashville, TN and Tulane University School of Medicine in New Orleans. He received his medical degree from the University of Oklahoma College of Medicine in Oklahoma City and then completed his Internal Medicine Internship and Residency at Emory University School of Medicine in Atlanta, GA. Dr. Smith received his Bachelor of Arts, Bachelor of Science and Master of Science degrees at the University of Oklahoma in Norman, OK.

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Craig S. Cameron, MD, FACC

Dr. Cameron is a specialist in cardiac electrophysiology, including catheter ablation of arrhythmia, atrial fibrillation management, pacemakers, implantable defibrillators, and cardiac resynchronization devices. He completed his Cardiac Electrophysiology Fellowship and his Cardiovascular Disease Fellowship at Baylor University Medical Center in Dallas, TX. Dr. Cameron's Internship and Internal Medicine Residency were performed at Baylor College of Medicine in Houston. He earned his medical degree from the University of Kansas School of Medicine in Kansas City, KS. Dr. Cameron received his Bachelor of Science degree at Pittsburg State University in Pittsburg, KS.

Board certified in Internal Medicine, Cardiovascular Disease and Cardiac Electrophysiology

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Dr. Ichinose specializes in interventional cardiology including cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures. He completed his Interventional and Clinical Cardiology Fellowships and his Internal Medicine Residency at the University of Massachusetts Memorial Health Care Center in Worcester, MA. Dr. Ichinose received his medical degree from Louisiana State University in New Orleans. He earned his Bachelor of Science degree from Texas Christian University in Fort Worth, TX.

Board certified in Internal Medicine, Cardiovascular Disease, Interventional Cardiology and Nuclear Cardiology

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Dr. Bruns is a specialist in Endocrinology, Diabetes and Metabolism at Oklahoma Heart Institute, with expertise in diabetes, thyroid disease (including thyroid cancer) and polycystic ovary syndrome. She completed her Internal Medicine Internship and Residency and Endocrinology Fellowship at the University of Wisconsin Hospital and Clinics in Madison, WI. Dr. Bruns earned her medical degree from Saint Louis University School of Medicine in St. Louis, MO and her Bachelor of Arts and Bachelor of Science degrees in biology from Truman State University in Kirksville, MO. Prior to joining Oklahoma Heart Institute, Dr. Bruns worked as a clinical endocrinologist at the Dean Clinic in Madison, Wisconsin.

Board certified in Internal Medicine, Endocrinology and Metabolic Diseases

Gregory A. Cogert, MD, FACC, FHRS

Dr. Cogert is a cardiologist who specializes in electrophysiology, including catheter ablation of arrhythmia, as well as the implantation and management of cardiac pacemakers, defibrillators, and cardiac resynchronization devices. He completed his Cardiac Electrophysiology Fellowship at Mayo Clinic in Rochester, MN and his Cardiovascular Fellowship at Cedars-Sinai Medical Center in Los Angeles, CA. Dr. Cogert's Internal Medicine Internship and Residency were completed at UCLA Medical Center in Los Angeles. He earned his medical degree from the University of California in Irvine and received his Bachelor of Science degree in microbiology and molecular genetics from the University of California in Los Angeles.

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Dr. Tulloch is a noninvasive cardiologist with expertise in adult echocardiography, peripheral vascular imaging, nuclear cardiology, cardiac computed tomography and MRI. Dr. Tulloch is Director of the Cardiac and Vascular Ultrasound Department of Hillcrest Medical Center's Cardiovascular Diagnostics. He completed his Cardiovascular Fellowship at the University of Kansas Medical Center in Kansas City, KS. Dr. Tulloch's Internal Medicine Internship and Residency also were completed at the University of Kansas Medical Center. He earned his medical degree from Ross University School of Medicine in New Brunswick, NJ and received his Bachelor of Science degree in biology from Avila University in Kansas City, MO.

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Dr. Duda is a specialist in Endocrinology, Diabetes and Metabolism at Oklahoma Heart Institute, with expertise in diabetes, lipids, hypertension and thyroid diseases. He completed his Fellowship in Endocrinology and Metabolism at the Mayo Graduate School of Medicine, where he also completed his Residency in Internal Medicine. Dr. Duda received his medical degree from Northwestern University School of Medicine in Chicago, IL. He earned his Bachelor of Science degree from Benedictine University in Lisle, IL.

Board certified in Internal Medicine, Endocrinology, Diabetes and Metabolism, Clinical Lipidology, Clinical Hypertension, Clinical Bone Densitometry and Thyroid Ultrasonography

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Dr/ Davies is a hospital-based cardiologist who provides continuity of care for patients admitted to Oklahoma Heart Institute – Hospital. He completed a Clinical Cardiology Fellowship and additional training in nuclear cardiology at the Medical College of Virginia, where he also completed his Internal Medicine and Residency programs. Dr. Davies received his medical degree from Johns Hopkins University School of Medicine in Baltimore.

Board Certified in Internal Medicine, Cardiovascular Disease, Nuclear Cardiology and Cardiovascular Computed Tomography Angiography

Kevin L. Lewis, MD

Dr. Lewis is a sleep specialist and a leading researcher and expert on the diagnosis and treatment of sleep disorders. He is Director of Sleep Care Services for Oklahoma Heart Institute, as well as Medical Director of Oklahoma Heart Institute Sleep Care of Hillcrest Medical Center. Dr. Lewis completed Fellowship training in Sleep Care, Pulmonary, and Critical Care at the University of Missouri Hospitals and Clinics in Columbia and the University of Kentucky Medical Center in Lexington. He completed his Internal Medicine Residency programs at the University of Nebraska Medical Center in Omaha and the Oklahoma University College of Medicine in Tulsa. Dr. Lewis earned his medical degree from the University of Texas Health Science Center in San Antonio.

Board certified in Internal Medicine, Pulmonary Diseases, Critical Care and Sleep Medicine.

Neil Agrawal, MD

Dr. Agrawal is a noninvasive cardiology specialist with expertise in adult echocardiography, nuclear cardiology, cardiac computed tomography and MRI. He completed his Cardiovascular Fellowship at the University of Vermont. Dr. Agrawal's Internal Medicine Internship and Residency were completed at the University of Louisville, and he earned his medical degree from St. George's University in Granada, West Indies. Dr. Agrawal completed his Bachelor of Science degree in biochemistry at the University of Texas at Austin.

Board certified in Internal Medicine

Kamran I. Muhammad, MD, FACC

Dr. Muhammad is a subspecialist in interventional cardiology with expertise in cardiac catheterization, coronary intervention (including angioplasty, stent placement, atherectomy, intravascular ultrasound), peripheral vascular intervention (including carotid intervention) as well as interventional therapies for structural heart disease, including PFO, ASD and valvular disease. In addition to his clinical experience, Dr. Muhammad has authored many peer-reviewed articles and textbook chapters on important cardiology topics.

Dr. Muhammad completed his Interventional Cardiology Fellowship at the Cleveland Clinic in Cleveland, Ohio, which included an additional year of dedicated training in peripheral vascular and structural cardiac intervention. His Clinical Cardiology Fellowship was also conducted at the Cleveland Clinic. Dr. Muhammad completed his Internal Medicine Internship and Residency at Yale University in New Haven, Connecticut, where he was selected and served as Chief Resident. He earned his medical degree from the University of Massachusetts Medical School in Worcester, Massachusetts. Dr. Muhammad earned his Bachelor of Science degree in computer science from the University of Massachusetts in Amherst, Massachusetts.

Board certified in Internal Medicine, Cardiovascular Disease, Nuclear Cardiology and Interventional Cardiology

Morakod Lim, MD

Dr. Lim is an interventional and noninvasive cardiologist with subspecialty expertise in cardiac catheterization, angioplasty, stents and atherectomy, as well as echocardiography, nuclear cardiology and coronary angiography. He completed his Interventional Cardiology Fellowship at the University of Medicine and Dentistry of New Jersey/Robert Wood Johnson Medical School in New Brunswick, NJ. His Clinical Cardiology Fellowship was conducted at the Albert Einstein College of Medicine in the Bronx, NY.

Dr. Lim completed his Internal Medicine Internship and Residency at Loma Linda University in Loma Linda, CA. He earned his medical degree from the Stony Brook School of Medicine in Stony Brook, NY. Dr. Lim received his Bachelor of Science degree in physics at New York University in New York, NY.

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Kala Mehta, MD, FACC

Dr. Mehta specializes in diagnostic and interventional cardiology, including cardiac catheterization, peripheral angiography, cardioversion, cardiac nuclear studies and echocardiograms.

Dr. Mehta is chair of the Claremore Hospital Physician Leadership Group, and serves on the hospital's Medical Executive Committee.

Dr. Mehta completed her Fellowship in Cardiology at the Oklahoma University Health Sciences Center, Oklahoma City, OK. She also completed her Internal Medicine Internship and Residency at Loyola University and Veterans Health Administration, Hines, IL. She was selected as Chief Resident for Internal Medicine at Smt. N.H.L. Municipal Medical College Gujarat University, Ahmadabad, India, where she also earned her medical and undergraduate degrees.

Board certified in Internal Medicine and Cardiovascular Disease

Arash Karnama, DO, FACC

Dr. Karnama is a specialist in interventional cardiology, including cardiac catheterization, coronary intervention, nuclear cardiology, echocardiography (TEE/TTE), cardioversion, peripheral angiography, peripheral intervention, carotid angiography, intravascular ultrasound, atherectomy, and PTCA/stenting for acute myocardial infarction.

Dr. Karnama completed his Interventional and Clinical Cardiology Fellowships at Oklahoma State University Medical Center and his Internal Medicine Internship and Residency at the Penn State Milton S. Hershey Medical Center in Hershey, PA. Dr. Karnama received his medical degree from Des Moines University in Des Moines, IA and his Bachelor of Arts degree from the University of Iowa in Iowa City.

Board certified in Internal Medicine, Interventional Cardiology, Cardiovascular Disease, Nuclear Cardiology, and Cardiovascular Computed Tomography

Victor Y. Cheng, MD, FACC, FSCCT

Dr. Cheng joins Oklahoma Heart Institute after serving as cardiology faculty at Cedars-Sinai Medical Center and assistant professor at the University of California in Los Angeles for the past four years. He is a specialist in noninvasive heart and vascular imaging, particularly in cardiac computed tomography (CT), a topic on which he has published numerous original research publications addressing quality, clinical use, and novel applications.

Dr. Cheng's training included a Clinical Cardiology Fellowship and Advanced Cardiac Imaging Fellowship at Cedars-Sinai Medical Center, and an Internal Medicine Internship and Residency at the University of California in San Francisco. Dr. Cheng received his medical degree from Northwestern University in Chicago, IL and his Bachelor of Science degree from Northwestern University in Evanston, IL.

Board certified in Internal Medicine, Cardiovascular Disease, Nuclear Cardiology, Echocardiography and Cardiovascular Computed Tomography

Varicose Veins: How to Find Relief

By Robert L. Smith, MD, MSc, FACC, FSCAI

If you are plagued by painful varicose veins, heavy, swollen, discolored legs, or any form of venous disease read on to learn more about venous insufficiency and the treatment options available at the OHI Vein Clinic

In 2009, Oklahoma Heart Institute began performing catheter ablations of incompetent saphenous veins, which are the underlying cause of painful, ropy varicose veins, as well as most other forms of symptomatic venous insufficiency. Since then, we have successfully treated hundreds of incompetent veins, and have expanded our practice to include adjunctive therapies like microsurgical phlebectomy (surgical removal of varicose veins through tiny incisions) and foam sclerotherapy of varicose veins and spider veins.

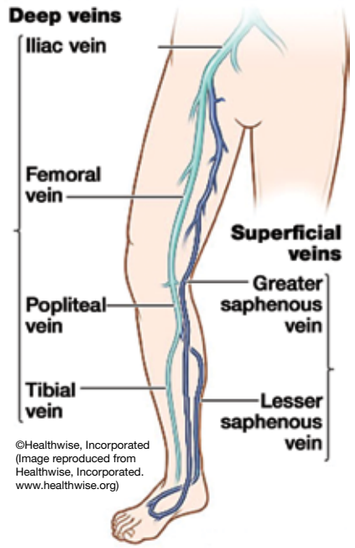
Patient satisfaction after treatment of venous insufficiency at Oklahoma Heart has been remarkably high, and our team of vein specialists has grown to include nurses, nurse practitioners, ultrasound technicians, and physicians alike. Recently, we added a brand new, state-of-the-art vein treatment center at our OHI SouthPointe office (92nd and Mingo).

Although recent years have brought about a greater awareness of the importance of diagnosing and treating peripheral arterial disease, pathology of the venous system has received little attention. Peripheral venous disease was first mentioned in the medical literature in 500 BC, when Hippocrates described an association between leg ulcers and varicose veins[1]. Despite longstanding recognition, understanding of issues surrounding venous hypertension and resultant venous incompetence has lagged.

Peripheral venous disease is four to five times more common than peripheral arterial disease[1], and the clinical presentation of lower extremity venous incompetence spans a broad clinical spectrum from telangiectasias (spider veins) to long-standing and recalcitrant venous stasis ulcers. This spectrum of disease manifests from cosmetically displeasing lesions on the legs to threats of serious secondary illnesses, such as deep venous thrombosis (DVT), pulmonary embolism (PE), and infection from chronic ulceration. Venous disease has a hereditary component, and children of patients with varicose veins are more likely to develop problems related to venous reflux[2]. Occupations characterized by inactivity, such as standing or sitting for long periods of time, also place patients at risk[3].

Until recently, the treatment of varicose veins and venous incompetence consisted mainly of surgical "vein stripping" procedures, during which large veins from the superficial collecting system of the legs are ligated and/or removed under general anesthesia. These surgical procedures, while effective, are associated with the risks of general anesthesia, infection of surgical wounds, long recovery times, and considerable patient discomfort. Perhaps for these reasons, surgical treatment of venous disease has occupied a rather small niche, and many patients have chosen to live with the condition rather than opt for surgery.

Figure 1
Veins of the Deep and Superficial Venous Systems of the Lower Extremity



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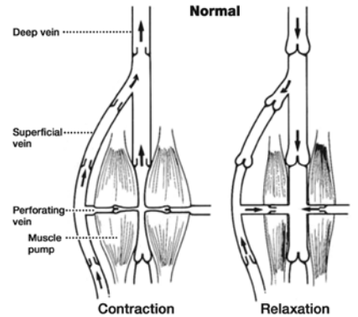
During the last ten years, outpatient, catheter based treatments have revolutionized how this disease is treated.

In order to understand the physiology of venous reflux disease, one must understand that there are two distinct venous systems in the lower extremities: The deep venous system is comprised of veins that run parallel to the arteries. These veins are surrounded by muscle and encased in connective tissue, providing them with good mechanical support. The deep veins are responsible for returning >95% of the venous blood from the lower extremity back to the heart.

The superficial system, comprised primarily of the saphenous veins, lies just beneath the skin and is without the mechanical support of the deep system. These superficial veins collect blood from surrounding tissues and, in a healthy system, route blood into the deep system so that it may be returned to the heart (Figure 1). It is typically incompetence of the superficial system that gives rise to the symptoms and physical manifestations of venous reflux disease.

It is widely accepted that most varicose veins and other problems from venous reflux relate to incompetence of the largest superficial vein; the greater saphenous vein. In some cases, the small saphenous vein (previously referred to as the lesser saphenous vein) is also involved. These superficial veins are located about a centimeter beneath the skin and should be less than 3mm in diameter in a healthy patient, though they can dilate to greater than 10mm in a patient with venous reflux disease. Unlike the deep venous system, where contraction of the leg muscles plays an important role in "pumping" the venous blood back to the heart, the return of venous blood in the superficial systems is passive, and depends entirely upon a series of one-way valves that aid the flow of the superficially collected blood on its route back to the deep venous system. Once in the deep system, venous blood is actively propelled against gravity back to the heart, primarily through the action of the calf muscles (Figure 2). In a healthy system, the superficially collected blood enters the deep venous system at two critical anatomic locations: The small saphenous vein deposits blood into the deep system at the sapheno-popliteal junc-

Figure 2
Flow of venous blood from the deep system to the heart is aided by the pumping of the calf muscles, which actively propel blood against gravity with each contraction.



(Image reproduced from "Chronic Venous Insufficiency" ASUM Ultrasound Bulletin, 2004 August. 7:4 14-21)

tion (near the knee) and the greater saphenous vein deposits blood into the deep system at the sapheno-femoral junction (near the groin). If the series of one-way valves in the superficial veins become incompetent (Figure 3), the superficial system becomes congested, and the flow of blood into the deep system (and subsequently toward the heart) stalls, resulting in swelling, pain, and other clinical sequelae.

When valvular function in the superficial system becomes sufficiently compromised, deoxygenated venous blood flows backwards, *from the deep system into the superficial system,* at these critical anatomic points, resulting in a blind loop, where deoxygenated blood cycles endlessly from the deep system to the superficial system, back to the deep system, and so forth, never making it back to the heart.

Common clinical manifestations of these events are swelling, heaviness, throbbing, pain, varicosities (bulging tributaries of the overloaded superficial veins), and skin changes that occur as a result of the endless loop of oxygen and nutrient poor blood circulating throughout the lower extremity network of veins. At the extreme, refractory and painful venous ulcers develop.

Although incompetence of the one-way valves can occur at any point in the network, the most common site of incompetence is the sapheno-femoral junction (Figure 4). When incompetence of the sapheno-femoral junction occurs, venous blood from the deep system, en route to the heart, is diverted from the (deep) femoral vein back into the (superficial) greater saphenous vein. This is typically the starting point of the pathologic cycle, and causes the greater saphenous vein to become increasingly engorged, setting off a cascade of events where superficial valves, unaided by the pumping function of the leg muscles, fail in a top to bottom fashion until clinical manifestations develop. This is why surgical removal of a diseased greater saphenous vein has proven effective in the treatment of venous reflux disease: the most common site of retrograde flow from the deep to superficial system is eliminated and the blood has nowhere to go but up (and back to the heart).

Experience with surgical removal of the greater saphenous vein goes back many decades. During the last several years, less invasive alternatives to surgical vein stripping procedures have arisen, and have targeted treatment of greater saphenous and small saphenous veins. Endovenous laser ablation and radiofrequency ablation of diseased greater and small saphenous veins have largely replaced the more painful surgical procedures. In several trials, these minimally invasive procedures, which can be performed on an outpatient basis, have proven to be as effective as the surgical alternatives[7,8,9,10]. While laser endovenous ablation of the greater saphenous vein and radio-frequency ablation of the greater saphenous vein are both as effective as surgery, radiofrequency ablation causes the least amount of patient discomfort, and is emerging as the minimally invasive procedure of choice[11,12].

During radiofrequency ablation of the greater saphenous vein, a tiny incision is made near the knee (Figure 5). Under ultrasound guidance, the greater saphenous vein is entered and a radiofrequency ablation catheter is advanced from the knee to the sapheno-femoral junction near the groin. Radiofrequency waves are then applied to the vein, causing the vein to fibrose and permanently close (Figure 6). In a matter of minutes, the vein is ablated, and the same functional outcome of the more painful surgical treatments is achieved. Although the benefit to the patients is the same as the surgical approach, patients may have this procedure performed on an outpatient basis (often in about 15 minutes) and will walk out of the office afterward, often experiencing dramatic results in hours to days after the procedure. When done properly, radiofrequency ablation of the saphenous veins is associated with little pain, speedy recovery times, and lasting results[11,12]. Complications, which include infection, paresthesias, DVT/PE, skin burns, and lymphedema, are exceedingly rare[11,12].

Several clinical scenarios may lead to the recommendation of minimally invasive saphenous vein ablation, but the presence of reflux must be confirmed by ultrasound prior to consideration of any treatment. Ultrasound mapping studies are performed in order to rule out deep venous thrombosis in the deep venous system and to evaluate for dilation and reflux in the superficial system. Prior to minimally invasive saphenous vein ablation, conservative measures should be tried. These include compression with graded elastic stockings, leg elevation, exercise, and symptomatic pain management with NSAIDS. If the patient demonstrably fails a 3-6 month trial of conservative therapy, they may be considered for minimally invasive saphenous vein ablation. In cases where isolated saphenous vein ablation fails to entirely treat the problem, a host of adjunctive therapies, such as stab phlebectomy and sclerotherapy, are available.

Venous reflux disease results in painful and cosmetically displeasing derangements of normal venous anatomy. At the extreme, it leads to recalcitrant venous ulcers, which can themselves cause secondary health issues. In the modern era, this common disease can be effectively treated with a minimally invasive, office-based procedure, with pleasing results (Figure 7). Radiofrequency saphenous vein ablation results in less discomfort and risk to the patient than surgical alternatives, and is proven to have lasting benefit. If you are interested in learning more about the treatment of varicose veins and other forms of venous disease, please call for an evaluation in the OHI vein clinic. 918-592-0999.

Dr. Smith specializes in interventional cardiology including cardiac catheterization, coronary angioplasty, and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound, and peripheral vascular interventional procedures.

Figure 3
Normal and Incompetent Venous Valves

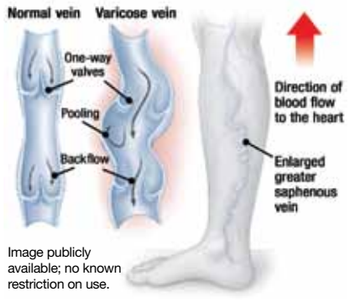


Image publicly available; no known restriction on use.

Figure 4

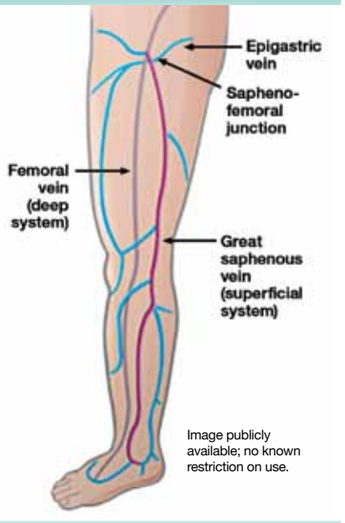
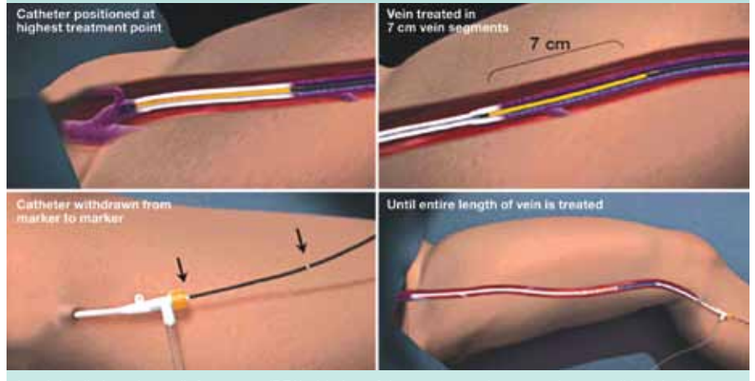


Image publicly available; no known restriction on use.

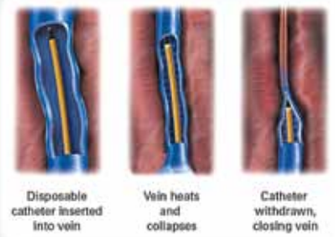
The most common site of venous incompetence is the sapheno-femoral junction, where blood from the deep system is diverted from its route back to the heart and flows into the superficial system in a retrograde fashion, resulting in a blind loop, where oxygen and nutrient poor blood cycles endlessly from the deep system into the superficial system and back again, never returning to the heart.

Figure 5
The VNUS Closure Procedure
Using the ClosureFAST™ Catheter



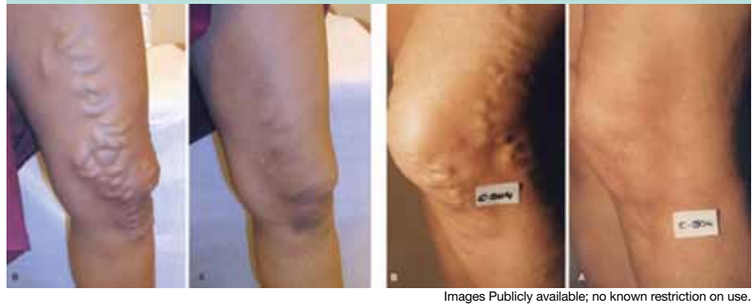
In minimally invasive radiofrequency (RFA) ablation of the greater saphenous vein, a small incision is made near the knee (Lower left panel), and the RFA catheter is advanced to a level just distal to the sapheno-femoral junction (upper left panel). Radiofrequency waves are then applied in a segmental fashion, treating 7 centimeters of the vein at a time. Pullback of the catheter is performed and the treatment is repeated until the entire length of the desired segment of vein is treated (right panels).
(Image provided by VNUS Medical Technologies, Inc.)

Figure 6



Using a radiofrequency ablation catheter, the diseased vein is cannulated and energy is applied within, resulting in fibrotic obliteration of the vein. The result is functionally identical to surgical "vein-stripping" procedures, during which the vein is surgically removed under general anesthesia.
(Image provided by VNUS Medical Technologies, Inc.)

Figure 7
Images from Before and After RFA Ablation of the Greater Saphenous Vein



Images Publicly available; no known restriction on use.

Continued from p. 10

a PVC-induced cardiomyopathy may be reversed by successfully ablating the culprit PVC. Figure 2 demonstrates how three-dimensional mapping is used to localize a focal source of idiopathic VT arising from the left coronary cusp of the aorta. On the electroanatomic map (middle), the earliest site is red and progresses to late in purple. Successful ablation locations (maroon dots) are shown.

In conclusion, sudden cardiac death is the leading cause of death in the United States and occurs as a result of ventricular tachycardia. Patients with VT should be assessed for underlying heart disease and risk of sudden death. ICDs are highly effective at preventing sudden death in at-risk individuals but do not address the underlying cause of VT. Thus, many patients with ICDs will ultimately require additional treatment to prevent ICD shocks. Likewise, many patients with idiopathic PVCs/VT may require treatment to address symptoms, or to prevent or reverse weakening of the heart's pump function. Medications are often not effective or well tolerated in treating PVCs/VT. Thus, catheter ablation of PVCs/VT is offered at highly specialized centers such as the Oklahoma Heart Institute. Ablation is a safe and effective option to eliminate or significantly reduce VT in the majority of patients and should be considered early in the course of this disorder.

Dr. Cameron is a specialist in cardiac electrophysiology, including catheter ablation of arrhythmia, atrial fibrillation management, pacemakers, implantable defibrillators, and cardiac resynchronization devices.

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The Beat Goes On

What You Need to Know About Atrial Fibrillation

By Gregory A. Cogert, MD, FACC, FHRS

WHAT IS ATRIAL FIBRILLATION?

Atrial fibrillation (AF) is the most common heart rhythm problem in America, with over 4 million people who carry the diagnosis of AF and many more yet to be diagnosed. So, just what is AF?

Normally, every beat of the heart is initiated by the upper chamber (atrium) contracting. This atrial impulse facilitates the flow of blood to the ventricle, which in turn pumps blood to the body. In addition to keeping the blood flowing normally, the atrium sets the heart rate, going faster during periods of stress or exercise and slower during rest. With atrial fibrillation, there is continuous chaotic electrical activity in the atrium with no atrial contraction and no atrial control of the heart rate (Figure 1). The loss of normal atrial blood flow can result in clotting of blood in the heart. The loss of heart rate control results in an erratic heart rate that can often be dangerously fast or slow.

Atrial fibrillation can result in a dramatic reduction in quality of life, physical condition, mental health, social functioning as well as cause congestive heart failure, stroke, dementia, and death.

WHO IS AT RISK FOR ATRIAL FIBRILLATION?

There is an increasing incidence with age and it is estimated that 25% of adults over 40 will develop AF during their lifetime. The most common risk factors for AF are age, high blood pressure, obesity, and obstructive sleep apnea. Patients with any chronic medical problem are at an increased risk for AF, especially problems of the heart, lungs, kidney, thyroid, and diabetes.

HOW IS ATRIAL FIBRILLATION TREATED?

Preventing Stroke

The first step in the treatment of AF is to evaluate the risk of stroke and initiate a treatment plan to minimize that risk. There are 5 classic risk factors for stroke in AF. They are the "CHADS risk factors"

C = Congestive Heart Failure
H= Hypertension
A= Age over 75 years old
D= Diabetes
S= prior Stroke or TIA

The risk for stroke in AF with none of these risk factors is under 2% whereas in the presence of all 5 the annual stroke rate approaches 20%. Stroke risk is also increased in women, patients over 65 years old, and the presence of vascular disease.

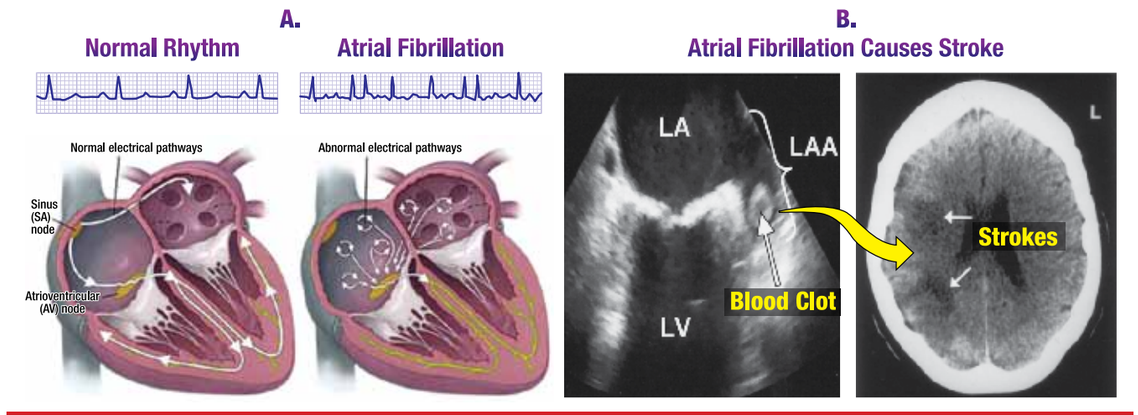
There are currently three approved anticoagulant medications (blood thinners) used to minimize stroke in AF.

Warfarin: Blocks the liver's production of clotting factors. Warfarin was the only option prior to 2010. Warfarin is a once daily medication that is affordable. An individual's dose is highly variable and frequent blood tests are required to confirm the correct dosing. Negatives include multiple food and drug interactions resulting in frequent dose changes and blood tests.

Dabigatran: Direct Thrombin Inhibitor. Approved by the FDA October 2010. In a large research trial was found to be superior to warfarin. If kidney function is stable, the dosing is reliable and no blood tests are required. There are significantly less food and drug interactions than warfarin. It is more expensive and there are less longterm safety data than warfarin. Negatives include the cost, twice daily dosing, and 10% of patients do not tolerate due to stomach irritation.

Rivaroxaban: Clotting factor (Xa) inhibitor. Approved for treatment of AF November 2011. Similar to dabigatran with stable dosing and minimal food and drug interactions negating the need for frequent blood tests in patients with stable kidney function. Cost is similar to dabigatran. In the large research trial that led to approval, its effectiveness was found to be equivalent to warfarin (as opposed to superiority seen with dabigatran). Advantages include once daily dosing and an improved side effect profile.

Figure 1



Treating the Symptoms

There are two strategies to minimize symptoms of AF: the "rate control" and "rhythm control" strategies (Figure 2). The goal of the rate strategy is to keep the heart rate within a normal range while permitting the atria to remain in fibrillation. The goal of the rhythm strategy is to maintain normal atrial function.

The advantage of the rate control strategy is the ease with which it is employed. If the heart is too fast, slowing medications are given. If the heart is too slow, a pacemaker is inserted to speed it up. If the medications are not effective or not tolerated, a simple ablation of the heart's electrical connection from the atrium to ventricle (AV node) is performed making the heart dependent on the pacemaker to beat.

The main disadvantage to the rate control strategy is the commitment to AF. Often symptoms continue despite rate control due to the absence of atrial contraction and the loss of a physiologic heart rate control. This strategy is generally pursued in elderly, sedentary patients with a long history of atrial fibrillation and minimal symptoms.

The advantage of the rhythm strategy is that, when successful, it restores normal heart function. Rhythm control is obtained by medications, catheter ablation, or heart surgery. In its early stages, AF tends to be intermittent (paroxysmal). If it progresses to become persistent, an electrical shock (cardioversion) is often required to regain normal rhythm. The longer a patient remains in AF, the greater likelihood they will become permanently in AF.

Catheter ablation involves electrically isolating the pulmonary veins in the left atrium that trigger AF. Prompted by research trials of over 7000 patients undergoing ablation showing superiority to medication, the Heart Rhythm Society, American College of Cardiology, and American Heart Association published the 2011 AF guidelines giving ablation a class I recommendation for the first line treatment of many patients with AF[1]. Through 2011, the Oklahoma Heart Institute physicians have performed over 350 AF ablations. Although research data is mounting that ablation can reduce the risk of stroke, congestive heart failure, and dementia, ablation is currently restricted to patients with symptomatic AF pending validation of this data.

The main disadvantage of the rhythm control strategy is the time and expense required to eliminate AF. The single procedure success rate without medication is 70%. Often additive medications or additional procedures are required to maintain normal rhythm.

SUMMARY

Atrial Fibrillation is the most common heart rhythm problem in America. It can decrease quality of life and cause congestive heart failure, stroke, dementia, and death. Risk factors for AF include older age, hypertension, and obesity. The first step in management is to minimize stroke risk. The second step is to minimize symptoms. Ablation of AF is superior to medical therapy and recently received a class 1 recommended for the first line treatment of AF.

Dr. Cogert is a cardiologist who specializes in electrophysiology, including catheter ablation of arrhythmia, as well as the implantation and management of cardiac pacemakers, defibrillators, and cardiac resynchronization devices.

References

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Figures

Figure 1. A. Comparison of normal rhythm and AF. With AF, there is continuous chaotic electrical activity in the atrium with no atrial contraction and no atrial control of the heart rate. B. Ultrasound of a heart with atrial fibrillation. The loss of atrial contraction leads to stasis of blood in the left atrium (LA) and left atrial appendage (LAA) resulting in the blood clot shown. CT scan of the brain shows two strokes cause by atrial fibrillation.

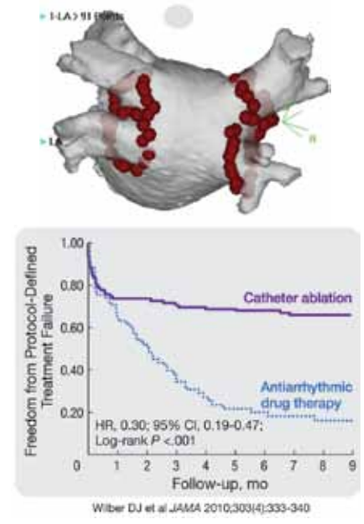
Figure 2. A. List of medications commonly used for rate and rhythm control of AF. B. Catheter ablation electrically isolates the pulmonary veins that trigger AF. Research has shown clear superiority of ablation over medications for rhythm control. C. If rhythm and rate control fail, an AV node ablation can be performed leaving the patient in AF forever with a pacemaker placed to define the heart rate.

Figure 2

A. Rate and Rhythm Control Medications

Drug	Comments
Beta blocker (Metoprolol)	Rate control
Calcium channel blocker (Diltiazem)	Rate control
Digoxin	**Not recommended for rhythm control. May increase AF and death in absence of heart disease.**
Soduim channel blockers (Flecainide, Propafenone)	Requires absence of heart disease
Potassium channel blockers (Sotalol, Dofetilide)	Requires hospitalization to start
Dronedarone	Safe in most patients, less effective than Amiodarone
Amiodarone	Long term use associated with organ damage

B. Cathether Ablation for Rhythm Control



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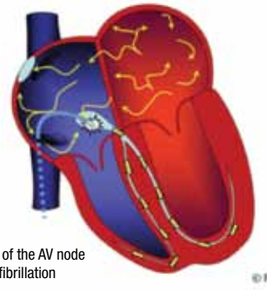
C. AV Node Ablation: Failure of Rhythm and Rate Control (Requires Prior Pacemaker Implant)



Ablation of the AV node in atrial fibrillation

Every Woman's Greatest Health Risk

Heart Disease is Different for You

By Eugene J. Ichinose, MD, FACC

Do you realize that more women die of cardiovascular disease than from stroke, lung cancer, chronic lung disease and breast cancer combined?

Regardless of race or ethnicity, cardiovascular disease is the leading cause of death among women, both in Oklahoma and nationwide. It accounts for nearly 500,000 deaths in the U.S. each year. Despite these sobering statistics, 45 percent of women fail to identify cardiovascular disease as their greatest health risk. [1]

HOW DO YOU DETERMINE YOUR RISK FOR CARDIOVASCULAR DISEASE?

In Table 2, medical history, lifestyle behavior and family history are indicators of early disease. Other conditions influence a woman's risk for heart disease and determine if a woman needs further screening tests to detect heart disease.

In the "high risk" group, there is a 19 percent chance that within 10 years a woman will experience a heart attack, stroke or die from heart disease.

In the "at risk" group, there is a 5.5 percent chance that within 10 years a woman will experience a heart attack, stroke or die from heart disease.

In the "optimal risk" group, there is a 2.2 percent chance that within 10 years a woman will experience a heart attack, stroke or die from heart disease.

In the "unclassified" group, there is 2.6 percent chance that within 10 years a woman will experience a heart attack, stroke or die from heart disease. Women in the unclassified group are without risk factors for heart disease. Because they do not maintain a healthy lifestyle, they are excluded from the optimal risk group.[2]

Obesity is defined as a condition of having a body mass index of greater than 30 (see BMI chart).

Evidence of atherosclerosis (coronary heart disease) can be determined by obtaining a screening test called a carotid ultrasound or coronary calcium score, which is available through the Oklahoma Heart screening program (918-592-0999).

The 10-year predicted cardiovascular disease (CVD) risk can be calculated using the Framingham Risk Score which you can find at http://hp2010.nhlbihin.net/ATPiii/calculator.asp?usertype=profand.

Pregnancy is a natural cardiovascular and metabolic stress test that may estimate a woman's lifetime risk for heart disease. Histories of preeclampsia will double the risk for subsequent ischemic heart disease, stroke and venous thromboembolic event over the five to 15 years after pregnancy. This may be an indication to carefully monitor and control risk factors during those years after pregnancy. [2]

All women should stop smoking and avoid second hand smoke. All women should also plan regular physical activity such as 30 minutes of brisk walking. For women who need to lose weight or sustain weight loss, a minimum of 60-90 minutes of moderately intense physical activity is recommended.

High blood pressure is a systolic blood pressure of greater than 140mmHg or diastolic blood pressure greater than 80mmHg. High blood pressure becomes more common in women over 65 years. The prevalence of hypertension in blacks in the United States is among the highest worldwide. It is especially high in black women at 44 percent.[2] Unfortunately, women tend to be under treated. Although, men continue to improve their rates of treatment and control, in the NHANE survey of 1999-2000, the treatment and control of hypertension in women has not changed.

Body Mass Index Table

BMI	Normal						Overweight					Obese																		Extreme Obesity																									
	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54																	
Height (in)	Body Weight (lb)																																																				
58	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258																	
59	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267																	
60	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276																	
61	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285																	
62	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295																	
63	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304																	
64	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314																	
65	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324																	
66	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334																	
67	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344																	
68	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354																	
69	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365																	
70	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376																	
71	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250	257	266	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386																	
72	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397																	
73	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265	272	280	288	295	302	310	318	325	333	340	348	355	363	371	378	386	393	401	408																	
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76	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443																	

Source: Adapted from Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Evidence Report.

Women more frequently experience non-classic symptoms on presentation of a heart attack.

Women should strive for a blood pressure of less than 120/80mmHg through lifestyle approaches such as weight control, increased physical activity, sodium restriction and increased consumption of fresh fruits, vegetables and low fat dairy products.

There is a frightening trend of increased body weight. Nearly two of every three U.S. women over 20 years old are now overweight or obese. This is a major contributor to the epidemic of type 2 diabetes mellitus now seen in more than 12 million women in the U.S. Type 2 diabetes mellitus greatly increases overall risk for heart attack and stroke.

Both lifestyle and medications should be used as indicated in women with diabetes to achieve a hemoglobin A one c (HbA1c) of less than seven percent if this can be accomplished without significant hypoglycemia.

During perimenopause, cholesterol and triglycerides become erratic, increasing by approximately 10 percent. HDL gradually declines after menopause. In the U.S., saturated fats come mainly from meat, seafood, poultry with skin, and whole-milk dairy products (cheese, milk, and ice cream). A few plant foods are also high in saturated fats, including coconut and coconut oil and palm oil. [4] The intake of saturated fat should be less than seven percent of total calories and cholesterol intake should be less than 200mg per day.

The use of hormone therapy and selective estrogen-receptor modulators should not be used for primary of secondary prevention of coronary heart disease. The use of vitamin supplements such as vitamin E, C, beta-carotene, folic acid with or without B6 and B12 have not been found helpful in preventing or treating coronary heart disease.

Women more frequently experience non-classic symptoms on presentation of a heart attack. Shortness of breath, nausea & vomiting, fatigue, sweating and arm or shoulder pain without chest pain occur more frequently in women than in men.

Table 2

Classification of CVD Risk in Women

Risk Status	Criteria
High risk (≥1 high-risk states)	Clinically manifest CHD Clinically manifest cerebrovascular disease Clinically manifest peripheral arterial disease Abdominal aortic aneurysm End-stage or chronic kidney disease Diabetes mellitus 10-y predicted CVD risk ≥10%
At risk (≥1 major-risk factors)	Cigarette smoking SBP ≥120 mm Hg, DBP ≥80 mm Hg, or treated hypertension Total cholesterol ≥200 mg/dL, HDL-C <50 mg/dL, or treated for dyslipidemia Obesity, particularly central adiposity Poor diet Physical inactivity Family history of premature CVD occurring in first-degree relatives in men <55 y of age or in women <65 y of age Metabolic syndrome Evidence of advanced subclinical atherosclerosis (e.g. coronary calcification, carotid plaque, or thickened IMT) Poor exercise capacity on treadmill test and/or abnormal heart rate recovery after stopping exercise Systemic autoimmune collagen-vascular disease (e.g. lupus or rheumatoid arthritis) History of preeclampsia, gestational diabetes, or pregnancy-induced hypertension
Ideal cardiovascular health (all of these)	Total cholesterol <200 mg/dL (untreated) BP <120/<80 mm Hg (untreated) Fasting blood glucose <100 mg/dL (untreated) Body mass index <25 kg/m^2 Abstinence from smoking Physical activity at goal for adults >20 y of age: ≥150 min/wk moderate intensity, ≥75 min/wk vigorous intensity, or combination Healthy (DASH-like) diet (see Appendix)

CVD indicates cardiovascular disease; CHD, coronary heart disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; IMT, intima-media thickness; BP, blood pressure; and DASH, Dietary Approaches to Stop Hypertension.

Based on a 2009 survey from the Center for Disease Control, Oklahoma remains in the top five states for the highest rate of heart attacks. 25 percent of the population of Oklahoma actively smokes, which is the third highest smoking rate. Oklahoma also placed third as the most sedentary state with 31 percent of the population not participating in any physical activity in the past 30 days. Tragically, Oklahoma had the greatest percent of people, 85 percent, consuming less than five servings of fruits and vegetables per day.

By following the above recommendations, you can begin to prevent heart disease from jeopardizing your health and longevity. Screening today could save your life tomorrow. Take time to take care of yourself.

Dr. Ichinose specializes in interventional cardiology including cardiac catheterization, coronary angioplasty and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound and peripheral vascular interventional procedures.

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Spring Recipes YOUR HEART WILL LOVE

Roasted Fish and Veggies with Quinoa and Pine Nuts

Serves 4

This easy, colorful meal is roasted in a parchment-paper-lined pan to avoid the need for added oil. We garnish the finished dish with cilantro and pine nuts, but a combination of basil and almonds or walnuts and parsley would be delicious as well.

- 2 carrots, cut into 2-inch sticks
- 1 large yellow bell pepper, cut into strips
- 1 1/2 cups grape tomatoes
- 1 red onion, halved and thinly sliced
- 3 garlic cloves, finely chopped
- 1/4 teaspoon fine sea salt, divided
- 4 (3-ounce) pieces mild white fish fillet such as tilapia, sole or flounder
- Zest and juice of 1 lemon
- 1 cup quinoa, cooked according to package directions
- 3 tablespoons chopped cilantro
- 3 tablespoons toasted pine nuts, chopped

Preheat oven to 400°F and line a 9- x 13-inch baking pan with parchment paper. Place carrots, bell pepper, tomatoes, onion, garlic and 1/4 teaspoon salt in the pan and toss. Bake in the top third of the oven until vegetables are tender, about 25 minutes. Stir vegetables, arrange fish on top and sprinkle with remaining 1/4 teaspoon salt, lemon zest and juice and bake until fish is just cooked through, 15 to 20 more minutes. Transfer fish and vegetables to plates or a platter along with the quinoa and sprinkle with cilantro and pine nuts.

NUTRITION Per serving: 330 calories (80 from fat), 9g total fat, 1g saturated fat, 45mg cholesterol, 220mg sodium, 40g total carbohydrate (6g dietary fiber, 8g sugar), 26g protein

Creamy Curried Cauliflower Soup

Serves 4

Curry powder and almond milk conspire here with the cauliflower to produce a full-flavored blended soup that's creamy on the palate without including any actual cream. The toasted sunflower seeds on top are a beautiful touch both floating in the bowl and crunching in the mouth.

- 3 cup raw sunflower kernels
- 3 1/2 cups unsweetened almond milk, divided
- 5 teaspoons mild curry powder, divided
- 1 cup chopped yellow onion
- 3 cloves garlic, chopped
- 5 cups (about 1 pound) cauliflower florets

Preheat oven to 350ºF. In a medium bowl, toss sunflower kernels with 1 teaspoon almond-milk and 1 teaspoon curry powder. Spread out on a small parchment paper-lined baking sheet and bake, tossing once or twice, until toasted and fragrant, 6 to 8 minutes; set aside. Meanwhile, heat 5 cup almond milk in a large pot over medium heat. Add onion and garlic and cook, stirring occasionally, until soft, about 10 minutes. Add cauliflower, remaining 4 teaspoons curry powder and almond milk, cover and simmer until cauliflower is very tender, about 40 minutes. Working in batches, carefully purée in a blender until smooth. Transfer to bowls, garnish with sunflower seeds and serve.

NUTRITION Per serving: 140 calories (60 from fat), 7g total fat, 0.5g saturated fat, 0mg cholesterol, 200mg sodium, 16g total carbohydrate (6g dietary fiber, 5g sugar), 6g proteinVegetarian, Wheat Free, Gluten Free, Vegan, High Fiber, Dairy FreeRomantic

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The Health Starts Here recipe data base is on the Whole Foods website (www.wholefoods.com), where you'll find recipes for soups, stews, bean dishes, vegetables, salads, and desserts that not only taste delicious, but also will nourish you with nutrients dense foods.

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