Oklahoma Heart Institute

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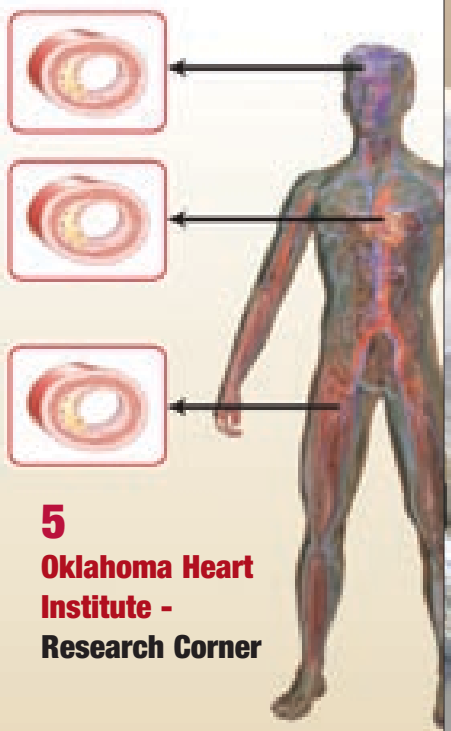
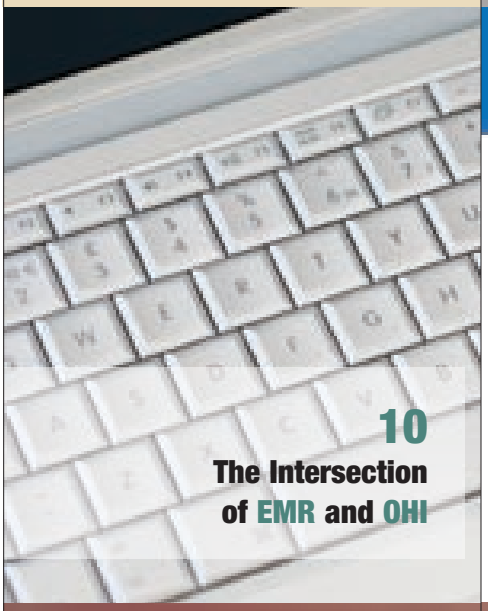
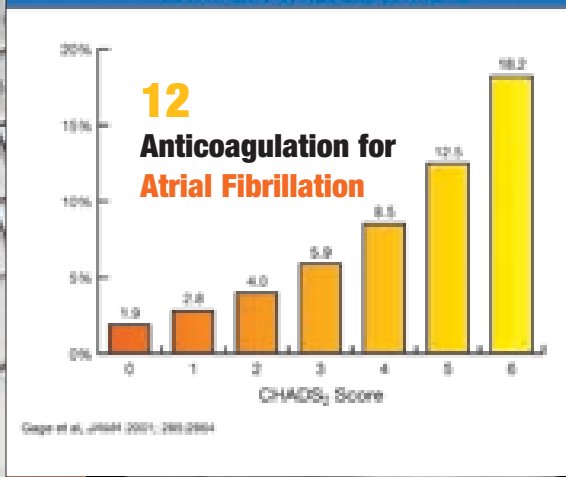
Oklahoma Heart Institute

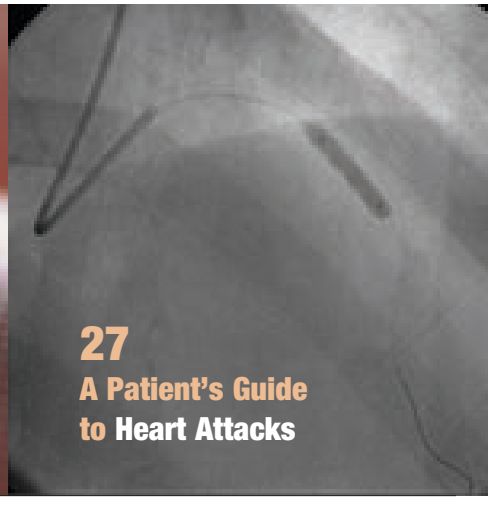
The *Oklahoma Heart Institute* magazine is mailed directly to referring physicians and other referring health care professionals in the Tulsa area and is also available in our patient waiting areas.

Cover photo of spring tulips in Tulsa by Rick Stiller









To Our Readers

THIS ISSUE OF THE Oklahoma Heart Magazine helps to clarify two common problems physicians frequently handle. Dr. Craig Cameron, an electrophysiologists at Oklahoma Heart Institute, nicely outlines the current guidelines for anti-coagulation for patients with atrial fibrillation. Dr. Frank Gaffney, who is co-director of the Oklahoma Heart Institute Pre-operative Clinic, reviews the recently presented studies addressing current guidelines on the use of pre-operative beta blockers for non-cardiac surgeries.

Also in this issue, a new section is added. Dr. Raj Chandwaney discusses the current management guidelines for acute myocardial infarction and has presented the information in a style so that physicians can give copies of the article to their patients as a reference to help them understand their care.

An article on EECP is presented to remind physicians of this effective, but often forgotten, treatment for refractory angina. Finally, the Research Corner presents some of the newer clinical trials that Oklahoma Heart is participating in which allow patients to have access to some of the newest therapies.

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

Sincerely,

Wayne N. Leimbach, Jr., MD

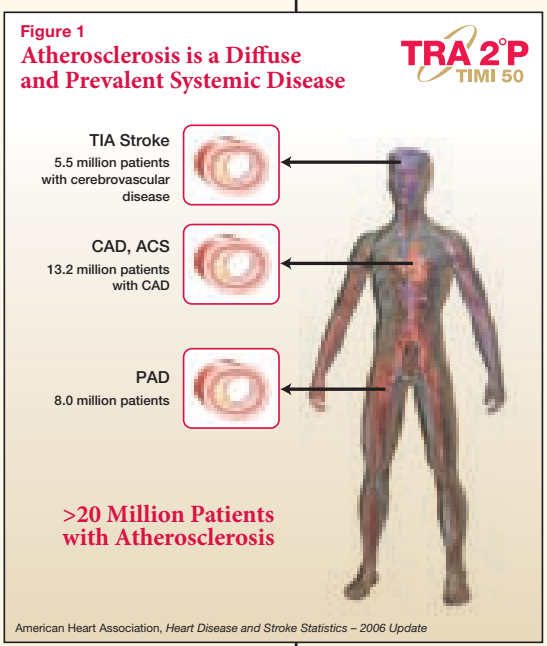
By Wayne N. Leimbach, Jr., MD, FACC, FSCAI, FCCP, FAHA

OKLAHOMA HEART INSTITUTE
Research Corner

THE FIELD OF CARDIOLOGY continues to be very dynamic with a large number of new treatment strategies being tested in randomized clinical trials. Oklahoma Heart Institute remains committed to clinical research. Through the Oklahoma Heart Research and Education Foundation, we are able to provide our patients access to newer, potentially superior, treatment options by participating in clinical research trials.

Two clinical trials that Oklahoma Heart is currently conducting examine whether thrombin receptor antagonists are superior to current antiplatelet therapy strategies when given to patients with acute coronary syndromes (TRA-CER Trial), or when given to patients with a history of atherosclerotic disease (TRA-2°P-TIMI-50 Trial).

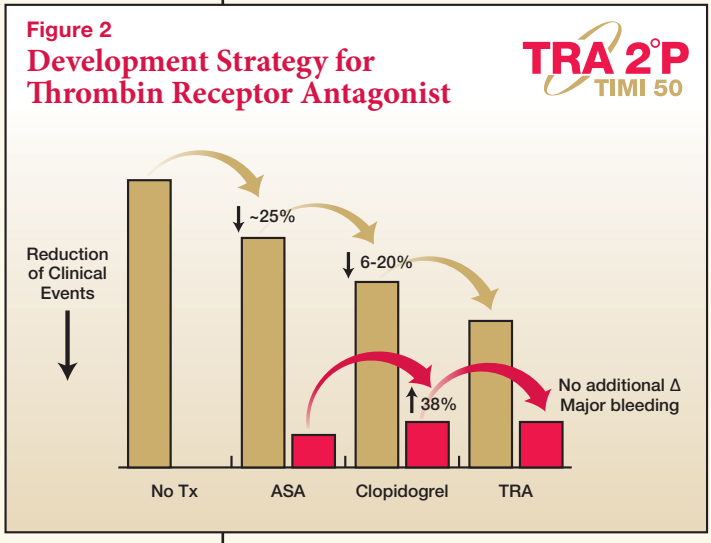
Figure 1
Atherosclerosis is a Diffuse and Prevalent Systemic Disease



Atherosclerosis is now known to be a diffuse systemic disease causing 5.5 million people to have cerebrovascular disease, 13.2 million people to have coronary artery disease and 8 million people to have peripheral vascular disease (Figure 1).

One year outcome data for 64,977 patients in the REACH Registry showed a 5.3 percent chance of cardiovascular death, myocardial infarction, or stroke, or the risk of hospitalization for an atherothrombosis event in patients with multiple risk factors for coronary artery disease. There was a 14.4 percent risk of the above cardiovascular events in those patients who already had established atherosclerotic vascular disease. If patients had more than one vascular bed involved with atherosclerotic vascular disease, there was a 22 percent risk of a cardiovascular event during the subsequent year. This data shows that patients with active atherosclerotic vascular disease remain at high risk of serious events over the subsequent years.

Figure 2
Development Strategy for Thrombin Receptor Antagonist

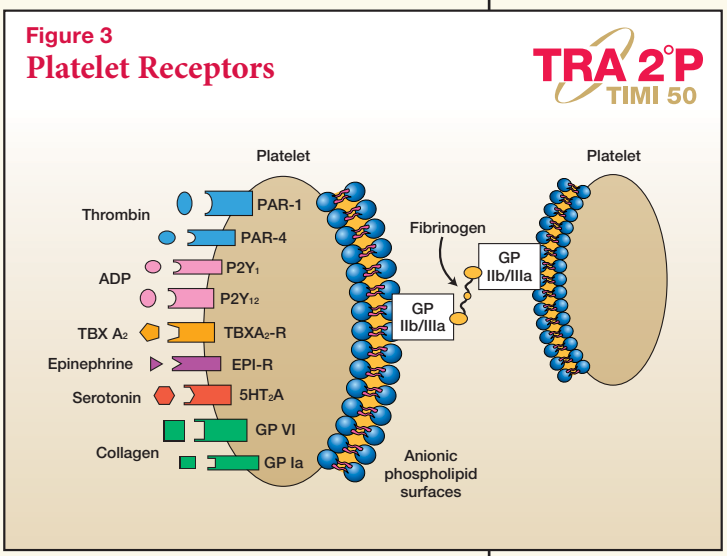


Many of these patients are treated with aspirin or a combination of aspirin and clopidogrel. Studies have shown that the use of aspirin reduces

the subsequent clinical events by 25 percent, compared to patients on no therapy (Figure 2). The introduction of clopidogrel on top of aspirin therapy provided an additional 6-20 percent reduction in clinical events. However, the addition of clopidogrel also resulted in an increase in bleeding complication rates. Newer preliminary studies suggest that thrombin receptor antagonists may provide additional reductions in clinical events without producing increases in bleeding complications.

We now know that platelets have multiple receptors by which they can be activated to start the process of thrombosis (Figure 3). Aspirin blocks the thromboxane A_2 pathways. Clopidogrel inhibits both the ADP and collagen induced platelet aggregation pathways. Selective thrombin receptor antagonists have been developed which have additive effects to aspirin and ADP antagonists. What is special about the thrombin receptor antagonists is that they are not associated with a significant increase in major or minor bleeding rates. Therefore, the direct thrombin receptor antagonists provide an effective interruption of an important pathway of platelet aggravation which should lead to a strong potential for efficacy. In addition, they do not interfere with thrombin mediated formation of fibrin or collagen induced platelet activation, which should provide important safety properties to this drug.

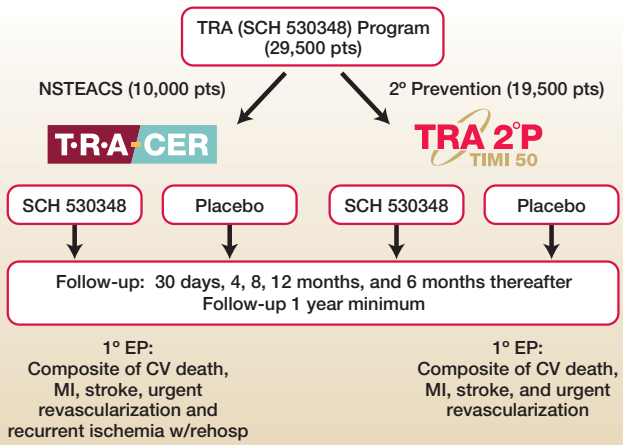
Figure 3
Platelet Receptors



The TRA-CER Clinical Trial will evaluate 10,000 patients presenting with a non ST segment elevation acute coronary syndrome. All patients will be treated with standard therapy of aspirin and clopidogrel. Patients will then be randomized to receive either placebo or the thrombin receptor antagonist SCH530348. Patients will then be followed for a minimum of one year. The primary end point will be a composite of cardiovascular death, MI, stroke, urgent revascularization, and recurrent ischemia with re-hospitalization.

The second trial (TRA-2°P-TIMI-50) looks at the use of thrombin receptor antagonists for secondary prevention. 19,500 patients with documented coronary disease or peripheral vascular disease will be treated with current standard therapy using anti-platelet agents and lipid modification agents. The patients will then be randomized to receive either the thrombin receptor antagonist or placebo and will be followed for a minimum of one year, and every six months after the first year, for the duration of the study. The primary end point will be cardiovascular death, myocardial infarction, stroke, or urgent coronary revascularization (Figure 4).

Figure 4
TRA (SCH 530348) Program



Since preliminary data suggests that thrombin receptor antagonists will provide additional efficacy to standard anti-platelet therapy without significantly increasing the risk of bleeding, these studies will provide our patients with a chance at receiving potentially superior therapy. In addition, as always, knowledge will be gained from these studies in regards to optimal treatment strategies and the natural history of atherosclerotic vascular disease states.

The SATURN Trial

Another interest in cardiology is whether the benefits of statin therapy for the prevention of progression of atherosclerotic vascular disease is just related to the LDL cholesterol lowering effects of these drugs or due to other mechanisms (pleiotropic effects). More specifically, there is the question of whether all the statins are equally beneficial for any given achieved LDL cholesterol level (class effect), or if there are definite benefits of one drug over the others because of additional potential mechanisms of action.

The statins are now the most widely used prescription medicine for the treatment of hypercholesterolemia. HMG-CoA reductase inhibitors have been associated not only with reductions in LDL levels, but also with substantial reduction of coronary events. Data from the statin trials indicates that reductions in the risk of coronary heart disease events are proportional to the reductions in LDL cholesterol levels. Angiographic trials have demonstrated that lowering LDL

cholesterol also slows the progression of coronary atherosclerosis. Previous studies have suggested that at least a 40 percent reduction in LDL cholesterol is needed to arrest progression of the atherosclerotic process. This would suggest that very aggressive lipid lowering is required to markedly reduce coronary heart disease risk. Therefore, it is possible that the more potent HMG-CoA reductase inhibitors would provide better clinical results when used at higher doses. However, clinical trials have also suggested that the statins may have other effects that are beneficial in preventing the progression of atherosclerotic vascular disease. The pleiotropic effects may not be the same for each of the different statins.

The SATURN Trial (Study of Coronary Atheroma by Intravascular Ultrasound: Effect of Rusuvastatin versus Atorvastatin) is a trial comparing the efficacy of two currently approved potent HMG-CoA reductase inhibitors. The study uses the technique of intravascular ultrasound (IVUS) to directly visualize the atheroma in the vessels' walls detected by coronary angiography. Atheroma volume can be measured with a high degree of sensitivity with the intravascular ultrasound technique. Previous studies using IVUS have demonstrated differences between statins with different lipid lowering effects. This study will evaluate whether 104 weeks of treatment with either rusuvastatin 40mg. or atorvastatin 80mg. differ in their effects on coronary artery atheroma burden, as assessed by changes in the percent atheroma volume and in the total atheroma volume, as measured by intravascular ultrasound imaging. Participation in this study provides our patients with free, already clinically approved, medications for at least a two year period. They also will receive a free follow-up coronary angiogram and intravascular ultrasound study to let them know if their medical therapy is actually stabilizing their coronary artery disease. This and similar studies will help define whether physicians and patients need to be more particular in their choice of cholesterol lowering medications or whether simply measuring the amount of LDL cholesterol lowering is adequate to guide effective therapy.

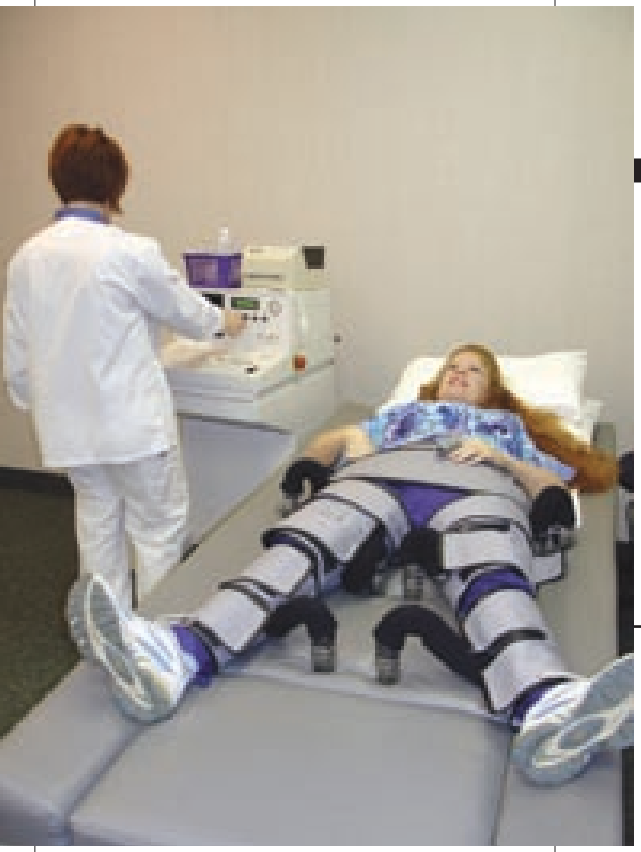
Physicians who have patients who would be interested in participating in either of these trials can contact one of the physicians at Oklahoma Heart Institute or contact one of the research nurses involved in the study.

Dr. Leimbach is a subspecialist in interventional cardiology, including cardiac catheterization, coronary angioplasty, and related interventional procedures such as stents, atherectomy, laser, intravascular ultrasound imaging, and direct PTCA for acute myocardial infarction.

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

EFFECTIVE NONINVASIVE THERAPY *FOR THE REFRACTORY ANGINA PATIENT:* EECP

Enhanced external counterpulsation (EECP) represents an effective noninvasive technique for the treatment of patients with symptomatic angina. Unfortunately, it is frequently forgotten by physicians as a treatment option for their patients.



Despite the use of optimal medical therapy, there are an estimated 300,000 to 900,000 patients in the United States who have refractory angina pectoris. These people are not candidates for standard revascularization procedures, such as angioplasty, coronary stenting or coronary artery bypass graft surgery. Their lifestyles are limited by anginal episodes that occur with daily tasks such as climbing stairs, mowing their lawn, or walking from parking lots to their work places or entertainment venues. The impact of symptomatic angina on quality of life has been shown to be significant. Rather than allowing these patients to restrict their activities and continue to worry about their condition, prescribing EECP treatments provides an effective and safe therapy with demonstrated 70-80 percent clinical response rates, which have been sustained up to five years.

Unfortunately, EECP is frequently forgotten by physicians as a treatment option for their patients.

FIGURE 1

POTENTIAL MECHANISMS OF ACTION OF EECP

1. Effects on Coronary Collateral Blood Flow
2. Improved Coronary Endothelial Function
3. Increased Vascular Growth Factors
4. Peripheral Effects – Increase Oxygen Update (V_{02})

Experimental work on counterpulsation techniques dates back almost 50 years. During the 1980s and 1990s, open label studies with enhanced external counterpulsation reported objective and subjective benefits in patients with angina. Since then, there have been numerous randomized and non-randomized clinical trials showing positive responses among patients treated with EECP therapy.

TECHNIQUE

The technique of EECP therapy consists of electrocardiogram-gated rapid, sequential compression of the lower extremities taking place during diastole followed by simultaneous decompression during systole. These actions produce hemodynamic effects similar to that of an intra-aortic balloon pump. However, unlike the intra-aortic balloon pump, EECP therapy also increases venous return.

Cuffs resembling oversized blood pressure cuffs are placed on the calves, the lower thighs, and the upper thighs including the buttocks. These cuffs inflate rapidly and sequentially via computer interpreted electrocardiogram signals. The cuffs are inflated starting from the lower calves and proceeding upward to the buttocks. During this sequential inflation, blood is forced back to the heart. In addition, arterial blood is forced back to the proximal arterial system, increasing blood flow to the coronary arteries. Just before the next heartbeat, all three cuffs simultaneously deflate, significantly reducing the workload of the heart. This is achieved because the vascular bed in the lower extremities is relatively empty of blood when

the cuffs are deflated. This significantly lowers the resistance to the ejection of blood by the heart.

The inflation and deflation activity is monitored by the help of finger plethysmograms and coordinated with a microprocessor. The end result is that there is the creation of a significant pressure wave that increases peak diastolic pressure and also reduces systolic pressure and systemic vascular resistance to the general benefit of the vascular system. A typical treatment course consists of 35 outpatient treatments administered as one hour per day over seven weeks.

Despite decades of research involving animals and clinical subjects, the actual mechanism by which EECP produces the clinical benefits are not known. There are four potential mechanisms of action for EECP (Figure 1), including the development of new functional collateral vessels. This may be related to increasing levels of nitric oxide and decreased endothelin-1 levels. In addition, stabilization of coronary endothelial function has been proposed as a potential mechanism of action for EECP therapy. Studies have demonstrated increased vascular growth factor levels after EECP therapy in humans. This raises the question as to whether arteriogenesis and angiogenesis may be providing improved vascularization to ischemic myocardium. Finally, the acute effects of EECP therapy on oxygen uptake has been demonstrated in adults with symptomatic coronary artery disease compared to healthy volunteer subjects getting sham therapy. The increase in oxygen uptake (V_{O2}) would be similar to what is seen with a training effect of exercise. EECP therapy has been demonstrated in clinical trials to improve exercise tolerance in stable angina patients.

There are now several non-randomized and randomized trials which show consistent positive clinical response among patients with refractory angina who are treated with EECP therapy (Figure 2). The benefits that have been demonstrated in clinical trials include a reduction of angina, reduction in nitroglycerin use, increased exercise tolerance, favorable psychosocial effects, enhanced quality of life as measured by questionnaires, prolongation of the time to exercise-induced ST-segment depression, and finally resolution of myocardial perfusion defects on perfusion imaging studies.

Although most of the clinical trials on EECP were not double-blind trials, the MUST-EECP trial was a randomized double-blind sham controlled trial. This trial did demonstrate clinical benefit of EECP therapy in patients with chronic stable angina and positive exercise stress tests. In this trial, 139 patients with documented coronary ischemia and Canadian Cardiovascular Society Classes I, II, and III angina were randomized to hemodynamically inactive counterpulsation with EECP versus active counterpulsation. Patients in the active EECP therapy group showed a statistically significant increase in time to exercise-induced ST-segment depression compared with sham and baseline. In addition, there was a statistically significant decrease in the frequency of angina episodes when compared with the sham and baseline levels. Exercise duration increased significantly in both groups; however, the increase was greater in the active EECP group compared to the sham EECP group.

Follow up data from the International EECP Patient Registry and the EECP Clinical Consortium have demonstrated that clinical benefits may be maintained up to five years in patients with a favorable initial clinical response. It should be emphasized that the clinical trials have demonstrated 70-80 percent clinical response rates in patients receiving EECP.

EECP IN ANGINA WITH LEFT VENTRICULAR DYSFUNCTION

Initially there was concern about using EECP therapy in anginal patients with significant left ventricular dysfunction. There was apprehension that the increased venous return from the EECP therapy could precipitate pulmonary edema in patients with severe left ventricular dysfunction. However, several studies have now been performed in patients with angina and significant left ventricular dysfunction, and the outcomes in the angina with significant left ventricular dysfunction patients have been positive, with the patients demonstrating significant reductions in nitroglycerin use, significant improvement in quality of life scores, and improvement in anginal class for the majority of the patients. Subgroup analysis of the International EECP Patient Registry showed that patients with symptomatic angina and left ventricular dysfunction did well and did not demonstrate adverse events because of the EECP therapy.

More recently, a large controlled study of EECP therapy in patients with stable heart failure and left ventricular dysfunction was undertaken. The study was called the Peach Trial (Prospective Evaluation of EECP and Heart Failure). This trial looked at the use of EECP therapy for heart failure and not for the treatment of angina. In this trial, the EECP therapy improved exercise tolerance, quality of life, and New York Heart Association functional class. Although this trial by itself does not confirm the utility of EECP therapy for the treatment of heart failure, it does support the previous studies showing the safety of EECP therapy in patients with significant left ventricular dysfunction.

Although EECP therapy has proven to be very safe in patients with angina with or without significant left ventricular dysfunction, there are some contraindica-

FIGURE 2

BENEFITS SEEN IN TRIALS ON EECP

1. Reduction of Angina
2. Reduction in Nitroglycerin Use
3. Increase Exercise Tolerance
4. Favorable Psychosocial Effects
5. Enhanced Quality of Life
6. Prolongation of the Time to Exercise-Induced ST-Segment Depression
7. Resolution of Myocardial Perfusion Defects

tions to the use of EECP therapy, listed in Figure 3. These include patients who have a coagulopathy with an INR of greater than 2.5, patients with significant dysrhythmias that interfere with the triggering mechanisms for the EECP, people who have moderate to severe aortic insufficiency, patients with severe hypertension, patients with aortic aneurysms, patients with significant venous thrombotic vascular disease, and patients with severe chronic obstructive pulmonary disease. EECP should also be avoided in patients who are within two weeks of cardiac catheterization, due to the risk of producing bleeding complications.

EECP therapy is not for everyone, but it has been demonstrated to be a very safe and effective therapy for patients with symptomatic angina. It is noninvasive. It is beneficial in both patients with class I and II symptomatic angina, as well as patients with severe disabling angina. Whereas in the past, patients often had to travel to large university clinical programs to have EECP, it is now widely available throughout the United States.

Additional information about EECP can be obtained by calling Oklahoma Heart Institute at (918) 592-0299.

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FIGURE 3

CONTRAINDICATIONS

- Coagulopathy with an INR (International Normalized Ratio) of prothrombin time ≥ 2.5
- Arrhythmias that may interfere with triggering of EECP (Enhanced External Counterpulsation) system (uncontrolled atrial flibrillation, flutter, and very frequent premature ventricular contractions)
- Within 2 weeks after cardiac catherization or atrial punction (risk of bleeding at femoral puncture site)
- Decompensated heart failure
- Moderate to severe aortic insufficiency (regurgitation would prevent diastolic augmentation)
- Severe peripheral arterial disease (reduced vascular volume and muscle mass may prevent effective Counterpulsation, increased risk of thromboembolism)
- Severe hypertension ≥ 180/110 mm Hg (the augmented diastolic pressure may exceed safe limit)
- Aortic aneurysm (≥5 mm) or dissection (diastolic pressure augmentation may be deleterious)
- Pregnancy or women of childbearing age (effects of EECP therapy on fetus have not been studied)
- Venous disease (phlebitis, varicose veins, stasis ulcers, prior or current deep vein thrombosis or pulmonary embolism)
- Severe chronic obstructive pulmonary disease (no safety data in pulmonary hypertension)

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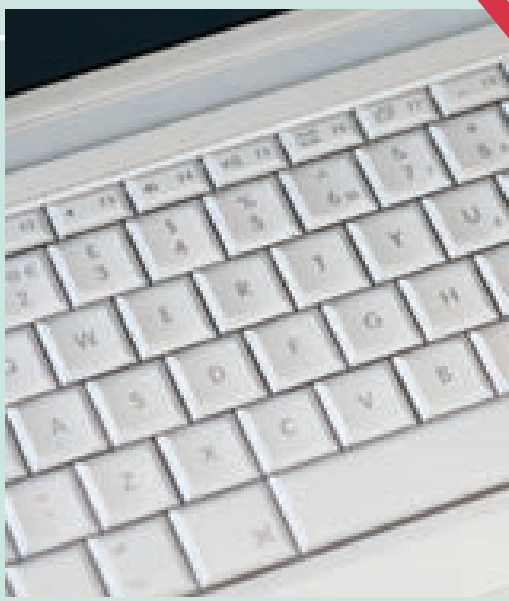


What's the connection between EMR (Electronic Medical Record) and this quote?

The opinion "...that it will ever come into general use notwithstanding its value is extremely doubtful; there is even something ludicrous in the picture of physicians using this device" (The Times-London). Well, none directly; the comment was made in the 1860s and concerns the dubious utility of the…stethoscope.

The technological challenge of this early 21st century may prove to be Electronic Medical Record. Many different products exist, and more are being developed. The commonalities among them are their capabilities to retrieve, manage, transact, and document patient care and all related components.

Oklahoma Heart Institute decided in 2006 to make the leap and join the ranks of early adopters of EMR technology. Ten years earlier, Oklahoma Heart adopted a scanning solution that produced an electronic storage system for patient records. Not interactive, not searchable, but devoid of paper charts and their maintenance. It enabled record sharing between two Tulsa office sites and set the tone for corporate adoption of computer-based documentation.

After reviewing the most dynamic systems available, GEMMS One was chosen for implementation. GEMMS One is unique in that it is designed specifically for cardiolo-

By Harriet Vaughan, RN, MS

THE INTERSECTION OF EMR AND OHI

gists; over sixty cardiology practices have made GEMMS One their EMR of choice. GEMMS One is also the system of choice for a variety of other types of clinical practice groups.

Having a robust EMR at your fingertips gives the practice tools to automate more processes, make information more available at the point of care or need, and create standards to ensure that quality is a major focus.

The GEMMS One system supports these components in a single database:

- Clinical documentation – including medications, lab, clinic visits, specialty clinics
- Messaging, ordering, tasking
- Scheduling
- Demographics/insurance/pre-certification
- Lab testing interfaces
- Billing/electronic remittance/collections
- Reporting – financial and clinical information

It is a point-of-service system that fully supports wireless data collection and documentation. Oklahoma Heart utilizes wireless tablet computers and a variety of desktop computers. Clinic encounters are gathered in the exam room and finished with the insertion of typed components to complete the documentation. Patient records are available 24 hours a day via local or remote access. Oklahoma Heart is using GEMMS One in both of their Tulsa locations and all outreach clinics.

What was the process of implementation like? It depends on whom you ask. It was complex, challenging, and frustrating at times; however, everyone survived, and every Oklahoma Heart team member, including all physicians, is using the system. We began in October 2006 with hardware and software implementation. Preparation for appointment and demographic conversion began in January 2007. Creating clinical records and abstracting from previous information began in January (and will continue until all previously seen patients have an appointment). Staff training occurred in February and March. We began using the appointment component of the system in late February and quickly added more components including billing. Clinical staff began using the system in March. Physician training began in late March and continued through April.

Our intent was to use the product 'right out of the box' in order to learn before we began any extensive customization. Our Physician Protocol Committee was instrumental in determining the customization projects to be tackled and provided the physician perspective we needed. An EKG interface was the first project. This was followed by an electronic interface with office lab equipment. A second interface with an external lab is in process now. This permitted lab ordering to be pushed from GEMMS and lab results to be pushed back to GEMMS from the lab management system. Appointment reminders and test result posting were other interfaces created. Custom flow sheets were developed to accommodate unique requirements of our endocrinology division.

We opted to participate in the Medicare PQRI program and utilized tools that GEMMS provided to track five cardiology-related quality indicators. Again, there was a learning curve to be able to remember to utilize the tools and to complete the quality indicator billing with the level of service billing. The alternative of creating a paper tracking device and marrying that to a claim would have required extra staff and would have delayed billing. We likely could not have considered participating in the PQRI reporting without the assistance of an EMR.

As veterans of an EMR implementation will tell you, "it's not just software." The opportunity for change is improving workflows and patterns of behavior that have been created through edict or tradition. Having a robust EMR at your fingertips gives the practice tools to automate more processes, make information more available at the point of care or need, and create standards to ensure that quality is a major focus. More thorough knowledge of the EMR (way beyond initial training) probably best precedes extensive customization. Otherwise, we are only paving the cow paths.

I believe that implementing GEMMS One has prepared us for future requirements such as e-prescribing. As states or payors require e-prescribing, GEMMS has components in place to be able to accommodate e-prescribing requirements – such as formulary checking, electronic prescription routing, and subscription drug updating. We are further customizing the system to better support our diagnostic areas and specialty clinics. Another plan directly benefits our patients as we roll out GEMMS My Record, a portable and personal medical record.

At less than one year, we are still making modifications; this will continue. The implementation of GEMMS One was largely successful, and we will persist to fully use the features and advantages of the system.

Harriet Vaughan is Director of Research and Information Services at Oklahoma Heart Institute and is the project director for the GEMMS EMR implementation. She is a registered nurse and has worked as a clinical nurse specialist, research coordinator, cardiology services director and case management/disease management director. The combination of healthcare and information services is a logical progression, and Harriet is very hands-on with the IT support of Oklahoma Heart. Her tool bag consists of clinical knowledge, systems analysis and intervention, and an orange bucket of cables, monitoring devices, climbing gear, pliers, and screwdrivers.

By Craig S. Cameron, MD

ANTICOAGULATION IN ATRIAL FIBRILLATION

INTRODUCTION: Atrial fibrillation (AF) is one of the most common arrhythmias in adults, affecting over 2.8 million Americans. Thromboembolism is a major source of morbidity in patients with AF, making anticoagulation an integral part of therapy. Despite the importance of anticoagulation in AF, there continues to be confusion and controversy surrounding its use.

RISK STRATIFICATION FOR CHRONIC ANTICOAGULATION

As with many other forms of therapy, the decision regarding chronic anticoagulation for AF should be individualized on the basis of patient risk.

The most recent American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) guidelines for the management of patients with AF[1] classifies risk factors for thromboembolism as "high," "moderate," or "weaker" risk. Aspirin is recommended for individuals without risk factors. Warfarin (goal international normalized ratio [INR] 2.0-3.0) is recommended for patients who possess any high risk factor or > 2 moderate risk factors. Individuals with a single moderate risk factor may receive either aspirin or warfarin (Figure 1).

Several similar thromboembolic risk stratification algorithms exist and have led to much confusion. One commonly used scheme in nonvalvular AF (mitral stenosis and prosthetic heart valves excluded) is the so-called $CHADS_2$ scoring system[2]. CHADS is an acronym which stands for Congestive heart failure, Hypertension, Age > 75 years, Diabetes mellitus, and Stroke (prior stroke, transient ischemic attack, or systemic embolus) (Figure 2). As such, the $CHADS_2$ score is easy to remember and use at the bedside. Each risk factor in the $CHADS_2$ score receives a single point, except for stroke which receives two. Annual stroke risk correlates with the $CHADS_2$ score in a more continuous fashion (Figure 3) than with other risk stratification schemes. Comparable to the ACC/AHA/ESC scheme, $CHADS_2$ categorizes patients as low, moderate, and high risk. Aspirin is recommended for low risk ($CHADS_2$ score = 0) patients. High risk patients ($CHADS_2$ score ≥ 2) should receive warfarin. Moderate risk individuals ($CHADS_2$ score = 1) may receive either aspirin or warfarin. One analysis compared $CHADS_2$ to four other risk stratification schemes in a cohort of patients with atrial fibrillation taking aspirin[3]. The five schemes divided the same cohort very differently, and $CHADS_2$ better identified the high risk group (annual risk > 5 percent) than the other algorithms.

Figure 1
Anticoagulation Recommendations

Risk Category	Risk Factors	Treatment
High	History of stroke, transient ischemic attack, mitral stenosis, prosthetic heart valve, 2 or more moderate risk factors	Warfarin to target INR 2.5 (range 2.0–3.0)
Moderate	Age ≥ 75, hypertension, heart failure, LVEF ≤ 35%, diabetes	Aspirin or warfarin to target INR 2.0–3.0
All others		Aspirin 81–325 mg/day

Weak risk factors: female gender, CAD, age 65–75, thyrotoxicosis

ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation 2006

THE CONTROVERSIES

WELL-CONTROLLED HYPERTENSION

Whether or not well-controlled hypertension is truly a moderate risk factor for thromboembolism is a source of debate. This controversy originates in part from a comparison of the warfarin-treated groups in the SPORTIF III[4] and SPORTIF V[5] trials. A lower stroke risk was observed in SPORTIF V, which had a lower average systolic blood pressure at entry. Studies with higher entry blood pressure have, in general, had higher stroke risk in the anticoagulation-treated arms. Additionally, many of the trials that identified hypertension as a risk factor are older with higher average blood pressure at entry, raising concerns about their relevance with respect to contemporary definitions of hypertension and blood pressure treatment goals.

PAROXYSMAL VS. PERSISTENT OR PERMANENT ATRIAL FIBRILLATION

The current guideline recommendation is that paroxysmal AF be considered to impart the same thromboembolic risk as persistent or permanent AF. When written, this recommendation was based upon weak data and engendered controversy. Recently, the largest trial of oral anticoagulation in nonvalvular AF[6] was published, demonstrating equivalent thromboembolic risk for paroxysmal and sustained AF, despite initially lower $CHADS_2$ scores in the paroxysmal AF group (Figure 4). From this study, no conclusions can be drawn regarding the amount of paroxysmal AF (AF burden) required to produce a thromboembolic risk equal to that of persistent or permanent AF. Two large prospective clinical trials[7,8] are currently underway utilizing device telemetry to address this question.

WARFARIN IN THE ELDERLY

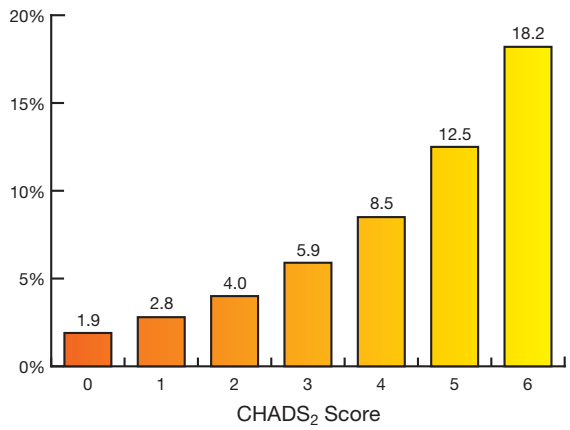
It has long been believed that elderly patients taking warfarin are at increased risk of bleeding. This is especially unfortunate since elderly patients are also generally at increased risk of stroke and may derive the most potential benefit from oral anticoagulation. A large cohort study[9] found a significantly higher rate of major hemorrhage among patients > 80 years of age than previously reported. The guidelines suggest using a lower target INR in the elderly. However, it may be preferable to use the same goal INR, monitoring it more closely.

Figure 2
Anticoagulation: Estimating Risk of Ischemic Stroke

$CHADS_2$

Congestive Heart Failure
Hypertension
Age > 75
Diabetes
Stroke or TIA (**2** points)

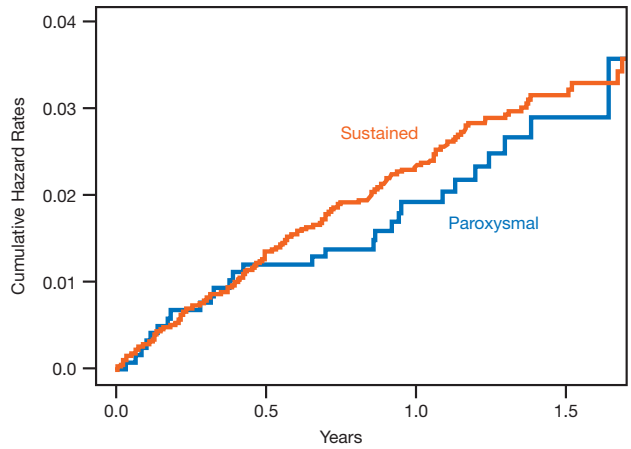
Figure 3
Annual Risk of Stroke



Gage et al, *JAMA* 2001; 285:2864

Despite the importance of anticoagulation in AF, there continues to be confusion and controversy surrounding its use.

Figure 4
Risk of Stroke
Paroxysmal vs. Sustained



Hohnloser SH et al, *J Am Coll Cardiol* 2007; 50:2156

SPECIAL CIRCUMSTANCES: CARDIOVERSION AND LEFT ATRIAL CATHETER ABLATION

CARDIOVERSION

Based upon observational data, the risk of stroke and systemic embolus is increased around the time of cardioversion. As such, anticoagulation is recommended for those undergoing cardioversion when the duration of AF is > 48 hours or unknown. There are two acceptable approaches. Patients may either receive warfarin at a therapeutic INR for three weeks or undergo transesophageal echocardiogram to rule out left atrial thrombus before sinus rhythm is restored. With either approach, patients must be anticoagulated for a minimum of four weeks following cardioversion. Bridging with unfractionated or low molecular weight heparin should be used as needed for subtherapeutic INRs around the time of cardioversion. The guidelines make no distinction between electrical and pharmacologic cardioversion.

RADIOFREQUENCY ABLATION IN THE LEFT ATRIUM

No randomized controlled trials have examined anticoagulation in individuals undergoing left atrial ablation procedures. However, thromboembolism is a recognized periprocedural complication. So it is customary for these patients to receive oral anticoagulation for a period of time before the ablation, unfractionated heparin during the procedure and then resume

warfarin for a period of time following ablation[10]. In high risk patients (CHADS^2 score > 3), there is no evidence that warfarin can be safely discontinued following a successful ablation procedure[11].

CONCLUSIONS

The decision to anticoagulate a patient with AF is based upon an individual's estimated thromboembolic risk. The ACC/AHA/ESC treatment guidelines and $CHADS_2$ scoring system serve as important tools in assessing this risk and prescribing appropriate chronic therapy. For the time being, clinicians should consider paroxysmal AF to confer the same thromboembolic risk as persistent or permanent AF. Elderly patients taking warfarin are at increased risk of bleeding and may require more careful monitoring of INR. Patients undergoing cardioversion (pharmacologic or electrical) and left atrial radiofrequency ablation procedures possess a high thromboembolic risk and should receive periprocedural anticoagulation regardless of their estimated risk by guideline or $CHADS_2$ criteria.

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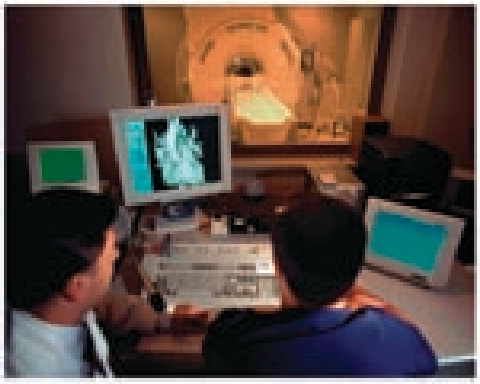

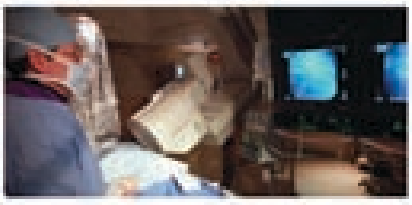

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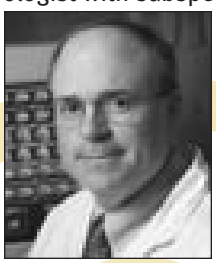

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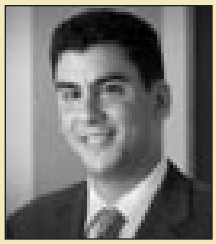

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By Frank J. Gaffney, MD, FACC

PERIOPERATIVE BETA BLOCKERS: FRIEND OR FOE?

The preoperative assessment of the patient heading to noncardiac surgery has always been a big part of the consultant work of a cardiologist. In fact, in 2007, OHI added a "Same Day" preoperative assessment clinic that gives a 24-hour turn around time for patients to be seen and tests to be done. This has been a tremendous benefit to surgeons and anesthesiologists, as well as primary care physicians and patients. Demand has been so great that we are planning to expand enrollment.

In 2007, we also saw an update of the ACC/AHA guidelines on perioperative cardiovascular evaluation and care for non-cardiac surgery. (see figure 1 on page 22) The big changes to the guidelines included a departure from segregating clinical risk factors into major, intermediate, and minor, with the incorporation of active cardiac conditions and the Revised Cardiac Risk Index (Lee TH Circulation 1999;100:1043-9). Functional capacity still plays a pivotal role in risk assessment with less emphasis on testing and a bigger role for heart rate control during the perioperative and postoperative period. It is this expanded role of the use of perioperative and postoperative beta blockers that has come under fire recently.

The first study that we need to discuss is the DIPOM trial. 921 patients not on a beta blocker with type II diabetes undergoing noncardiac surgery were randomized to receive placebo or 100mg/day of metoprolol (long acting) for a maximum of eight days. The primary end point was a composite of all-cause mortality, acute MI, unstable angina or congestive heart failure. After a median follow-up of 18 months, there was no significant difference in the occurrence of the primary end point. A closer look at this trial reveals that, if a patient could not take the medication orally, then five mg IV was given every six hours. Likewise, the remaining patients were dosed orally, which in the postoperative period certainly can lead to a variable rate of absorption of the drug. Critics of the study claim that this was a relatively low dose of beta blocker in this low risk population, and the findings were not unexpected.

More importantly, the POISE trial was presented at the AHA 2007 scientific sessions. According to a review published on the Web site www. theheart.org, (this is a great Web site for the cardiology junkie) POISE randomized 8351 patients

45 years or older undergoing noncardiac surgery with or at risk of atherosclerotic disease. Patients had to have a history of CAD, PAD, stroke or congestive heart failure within the last three years, be undergoing major vascular surgery, or have three of the following seven risk factors: undergoing high risk surgery, having a history of congestive heart failure, having DM, having renal insufficiency, being 70 years of age or older, having a history of TIA, or undergoing urgent/emergent surgery.

Patients were randomized to receive either metoprolol CR or placebo started two to four hours preoperatively and continued for 30 days. The dose of metoprolol given was 100mg in the preoperative period, 100mg in the six-hour postoperative period, 200mg 12 hours later and 200mg daily for 30 days. Doses were not titrated, and the drug was stopped only if the blood pressure dipped below 100 mmHg.

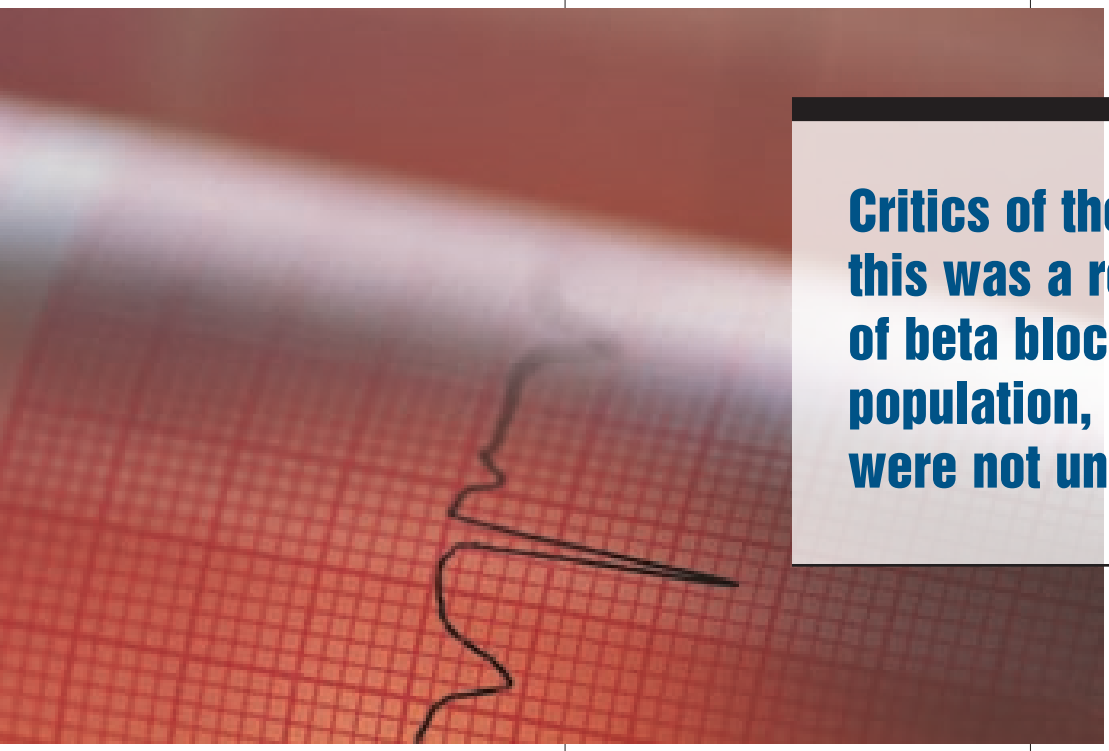
The primary outcome was a composite of cardiovascular death, nonfatal MI, and nonfatal cardiac arrest at 30 days after randomization. Secondary outcomes included total mortality, cardiovascular death, MI, cardiac revascularization, clinically significant atrial fibrillation, clinically significant bradycardia, clinically significant hypotension, and stroke.

Results showed a clear reduction in MI and a decrease in coronary revascularization and atrial fibrillation, but an increase in death and stroke, as well as an increase in hypotension and bradycardia.

There are some real problems with this study. First, we should understand that the POISE trial excluded patients already taking beta blockers. Stopping a beta blocker in someone who is going to surgery is absolutely the wrong thing to do. Second, there did not appear to be a period of titration of the dose of beta blocker before surgery. It seems very telling that there was an increase in stroke, hypotension, bradycardia and death, with stroke being the biggest predictor of death. These are the same conditions found when too high a dose of beta blocker is used in hypertensive or coronary artery disease patients who are not going to surgery. I can't ever remember a time that I titrated a patient's dose of beta blocker to 200mg a day in a 24 hour period who wasn't suffering from hypertensive urgency/emergency. Why anyone thought that this regimen would be safe, especially in the elderly or normotensive patient is not clear to me.

The guidelines for perioperative cardiovascular evaluation and care of the noncardiac surgical patient continue to be a valuable tool to assess the cardiovascular risk of patients going to surgery. Moreover, the strategy of using beta blockers or even clonidine to reduce the individual perioperative risk of our patients is still a viable option.

The two studies mentioned seem to set a reasonable floor and ceiling on the use of perioperative beta blockade, mainly that the low risk patient might not benefit from low dose beta blocker initiated at the time of surgery, and the high risk patient should undergo a careful deliberate up titration of beta blocker when at all possible, keeping in mind that one dose not fit all.

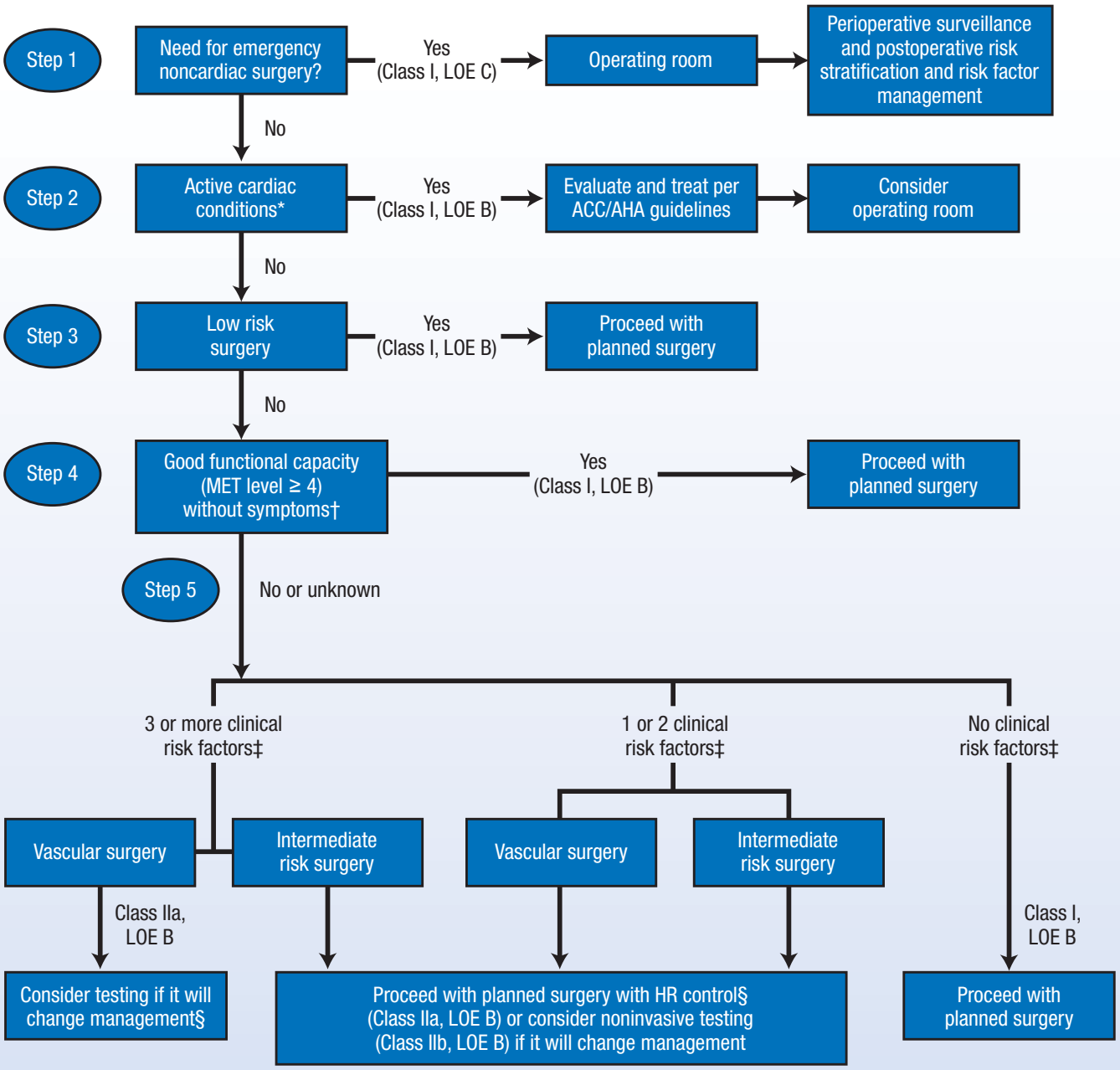


Critics of the study claim that this was a relatively low dose of beta blocker in this low risk population, and the findings were not unexpected.

Dr. Gaffney is an invasive and noninvasive cardiologist with subspecialty expertise in transesophageal echocardiography.

Figure 1

Cardiac evaluation and care algorithm for noncardiac surgery based on active clinical conditions, known cardiovascular disease, or cardiac risk factors for patients 50 years of age or greater.



* See Table 2 for active clinical conditions include unstable coronary syndromes, unstable or severe angina, recent MI, decompensated HF, significant arrythmia, and severe valvular disease.

† See Table 3 for estimated MET level equivalent.

‡ Clinical risk factors include ischemic heart disease, compensated or prior HF, diabetes mellitus, renal insufficiency, and cerebrovascular disease.

§ Consider perioperative beta blockade (see Table 11) for populations in which this has been shown to reduce cardiac morbidity/mortality.

ACC/AHA:	American College of Cardiology/ American Heart Association
HR:	Heart Rate
LOE:	Level Of Evidence
MET:	Metabolic Equivalent

JACC Vol. 50, No. 17, 2007 Fleisher et al. e169
October 23, 2007:e159–241 ACC/AHA 2007 Perioperative Guidelines

Aerobic Gardening

by Terry Rudd

Lawn and Garden Athletes Find Physical Fitness in Their Own Backyards

It's an exercise craze with hordes of fervent followers and pages of weight-loss success stories. No, it's not the latest kickboxing style or abdominal exercise contraption peddled on television shopping channels.

It's aerobic gardening. And although the name may cause chuckles, it's also causing plenty of people to improve their physical and mental well-being—not to mention what it's done for their landscaping.

Aerobic gardening melds the drive for a slim stomach with the quest for a green thumb. Its appeal is growing daily, with thousands finding a faster route to fitness through gardening exercise.

There's even an annual National Gardening Exercise Day every June that is designed to promote the idea of yard work as "yard exercise." State garden clubs and thousands of gardeners across the country spend the day flexing their muscles and reshaping their outdoor surroundings.

Aerobic Gardening's Surprising Benefits

Couch potatoes, take heart. Despite the potentially ominous-sounding combination of exhausting exercise and tedious yard work, gardening exercise is something far less frightening. In fact, if done correctly and regularly, aerobic gardening can prove to be a enjoyable—and relatively painless—way to shed unwanted pounds while improving property values.

Forget about bench-pressing bushes or doing pull-ups from a hanging tree limb. And don't worry about acquiring a vast new wardrobe of fashionable workout clothing.

If you can pull a weed, you can participate in aerobic gardening. Gardening exercise incorporates activities familiar to anyone who has ever spent time behind a lawn rake or bending over a row of vegetables. And garden exercise chic includes everything from torn jeans and worn-out running shoes to threadbare sweatshirts. In fact, grungy is in when it comes to mixing garden exercise with garden fashion.

Gardening exercise probably won't transform an Archie Bunker body into an Arnold Schwarzenegger physique. But for those of us with a little more flab and a little less vigor than we'd like, putting in time at the "outdoor gym" can make a major difference in strength and stamina. Even moderate activity out in the yard and garden can improve cardiovascular health and boost psychological well-being.

Think there's no comparison between gardening exercise and more "legitimate" approaches to keeping physically fit?

Think again. Spreading fertilizer can burn as many calories as a moderate walking session, while mowing and raking the lawn can expend as much energy

as a session of aerobics or swimming laps in the pool. Trimming hedges or weed-eating a yard can match the benefits of a baseball or softball game, while pulling weeds and sacking grass and leaves can be as rewarding as a 9-hole game of golf – without the cart, that is. If you head out to seed your yard, you'll expend about as much energy as you would
in a bowling match. While chopping wood, tilling and shoveling are the most strenuous activities, even re-potting plants count in this style of exercise.

How to Become a Gardening Athlete:

Aerobic gardening's premier "guru" is probably Jeffrey Restuccio, author of the book *Fitness the Dynamic Gardening Way*. Restuccio has been a tireless promoter of the benefits of good gardening exercise technique. Among Restuccio's top tips for beginning aerobic gardeners and seasoned veterans alike are:

Ease into exercise:

Gardeners young and old need to take time to get their muscles ready for a workout

before ever grabbing a gardening rake. That means spending five to ten minutes before every gardening exercise session simply warming up and stretching. Hit the gardening playing field without giving your body a chance to warm up, and you risk plenty of sore muscles or even injury to your back or legs. Also, don't forget to us sunscreen and drink plenty of fluids.

Variety is the spice of life:

It's also the key to preventing injury and keeping a gardening workout from becoming a mind-numbing chore. Using a variety of gardening motions at a steady pace can keep heart rates up and interest from waning. Mix up mowing, weeding, pruning and digging, alternating between tasks every 15 minutes or so.

Bend right or ache wrong:

Before you jump into any gardening exercise program, master a few simple techniques. The chief exercise technique: don't bend from the back while raking, digging or hoeing. Seasoned gardening athletes know the best way to get the job done is by bending from the knees and using shoulders, arms and legs in a rocking motion. Switching between right-handed and left-handed stances also redistributes the strain of exercise and keeps repetitive activities from becoming harmful. Match gardening tool handles and positioning, too. Standing activities should only be done with long-handled tools, while shorter hand tools should be used only when kneeling or sitting.

Use a progressive approach:

Start out any aerobic gardening workout with light exercise, such as picking up branches, pruning or doing some light raking of leaves. Once your body is warmed up, move on to a higher rate of exercise for 20 minutes to 30 minutes. Keeping it going for 30 minutes or more will burn more fat.

Wind down the right way:

As with any exercise activity, an abrupt stop can lead to soreness. Take some time to cool down by finishing up with simple tasks or a relaxing walk. And remember to stretch again after a gardening workout.

Interested in finding more ways to trim your waistline while beautifying your garden? Check out Jeffrey Restuccio's web site at www.ritecode.com/aerobic-gardening.

Flavor Savers

Nothing's more disappointing than a meal that's had all the flavor cooked out of it. When grilling, lock in juices by searing steaks, chops, and burgers before cooking at the recommended recipe temperature.

Trim excess fat from steaks and chops, leaving only a scant 1/4-inch of fat, which is sufficient to flavor the meat. Less fat is a virtual guarantee against flare-ups and makes cleanup easier.

Grill with the lid down for perfectly cooked food every time.

To avoid piercing meats and losing their natural juices, use tongs rather than a fork for turning and handling them. (Forks are great for lifting large foods like roasts or whole poultry from the cooking grate after they're done cooking.) Use two spatulas for handling large, whole fish.

Perk up your favorite stand-bys with some new marinades and sauces. Marinades make meat, poultry, and fish more tender, juicy, and savory. Sauces add a little tang or sweetness to an ordinary meal.

Visit www.fabulousfoods.com/features/grilling.html for some great recipes to make your own grilling sauces and marinades.

By Raj H. Chandwaney, M.D., FACC, FSCAI

EDITOR'S NOTE: *Physicians can reproduce and give a copy of Dr. Chandwaney's article to their patients to help them understand heart attacks and their treatment.*

A PATIENT'S GUIDE TO Heart Attacks

Cardiovascular disease continues to be the leading killer in the United States. It accounted for 1 of every 2.8 deaths that occurred in 2005. Fifty-two percent of these cardiovascular deaths are attributable to coronary heart disease. In 2008, an estimated 770,000 Americans will have a new heart attack; 430,000 Americans will have a recurrent heart attack. About every 26 seconds, an American will have a coronary event, and every minute someone will die from one.

The purpose of this article is to educate the reader about heart attacks. There are five questions the article will address:

- What is a heart attack?
- What are the risk factors for heart attack?
- What can we do to prevent heart attacks?
- What are the symptoms of heart attack?
- What to do if someone is having a heart attack?

WHAT IS A HEART ATTACK?

A heart attack occurs when a blood vessel that feeds the heart abruptly closes. These blood vessels are called coronary arteries. They usually close because a plaque within them suddenly ruptures. After a plaque ruptures, a clot forms on top of the ruptured plaque. If the clot partially obstructs the coronary artery (see figure 1), this causes unstable angina, and/or a small heart attack, which is referred to as a non ST elevation myocardial infarction (NSTEMI). If the clot obstructs the coronary artery completely (figure 2), this causes a large or massive heart attack, which is referred to as a ST elevation myocardial infarction (STEMI). Small heart attacks usually do not cause much damage to the heart, but they may be a warning that a massive heart attack is coming soon. Large massive heart attacks can be life threatening.

After a coronary artery abruptly closes, the heart muscle, which is normally fed by that coronary artery, will be starved of blood and oxygen. If the coronary artery remains obstructed for too long, that part of the heart will die and eventually turn into scar tissue (figure 3).

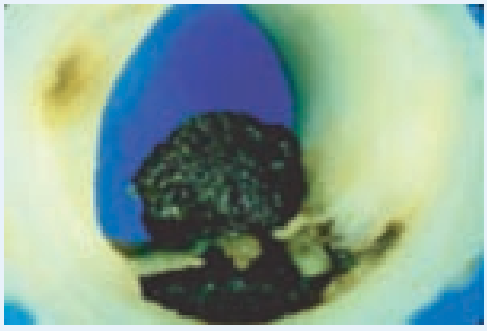


Figure 1 & 2: Autopsy specimen of a coronary artery with ruptured plaque. A clot has formed on top of the plaque totally obstructing the coronary artery.

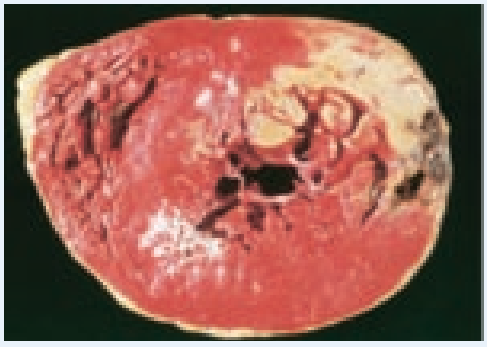


Figure 3: Autopsy specimen of a heart from a patient who suffered a large untreated heart attack. A large area of scar tissue is present in the right upper quadrant of the specimen.

Heart attacks can be associated with life threatening rhythm problems. When parts of the heart don't get enough blood, the muscle becomes electrically irritable and serious rhythm abnormalities develop. Some types of arrhythmias are not compatible with life because the heart cannot pump blood efficiently. If a patient survives an untreated heart attack, they are left with a weakened heart that may predispose them to develop congestive heart failure and life threatening arrhythmias in the future. Patients with congestive heart failure experience shortness of breath because their lungs fill up with excess fluid when the weakened heart cannot pump blood efficiently.

WHAT ARE THE RISK FACTORS FOR HEART ATTACK?

There are five classic risk factors that physicians use to assess a patient's risk for heart attack:

- History of tobacco use
- High cholesterol
- High blood pressure
- Diabetes Mellitus
- Family history of atherosclerotic disease

In addition to the five risk factors listed above, the Framingham Risk Score is a tool which provides individuals with their calculated risk of having a heart attack over the next 10 years. It takes into account the individual's age, gender, total cholesterol, HDL cholesterol, blood pressure, history of hypertension, and smoking history. Individuals can calculate their own 10 year risk by using

the Framingham Risk Score calculator, which is available on the Worldwide Web *(Google search: Framingham Risk Score)*.

WHAT CAN WE DO TO PREVENT HEART ATTACKS?

Although the risk factors are highly prevalent in our society, there are things we can do to prevent heart attack. Therapeutic lifestyle changes (TLC) are the foundation of heart attack prevention. TLC starts with eating a low fat and low cholesterol diet. Some individuals may need to consult with a dietician, but a heart healthy diet can be summarized by the five principles listed below:

- Eat less red meat; eat more fish or chicken
- Always bake, broil, or grill (never fry!)
- Eat more vegetables in the form of salads and steamed vegetables (no french fries and no onion rings!)
- Eat more whole grains rather than bleached white rice or bread
- Eat a piece of fruit for dessert instead of cakes or cookies

In addition to eating a low fat, low cholesterol diet, regular exercise is also an important part of TLC. Achieving the fitness level of a competitive athlete is not required to experience the lifesaving benefits of exercise. A simple regimen that includes a brisk 30 minute walk performed five to six days each week has been shown to markedly reduce the risk for cardiovascular disease.

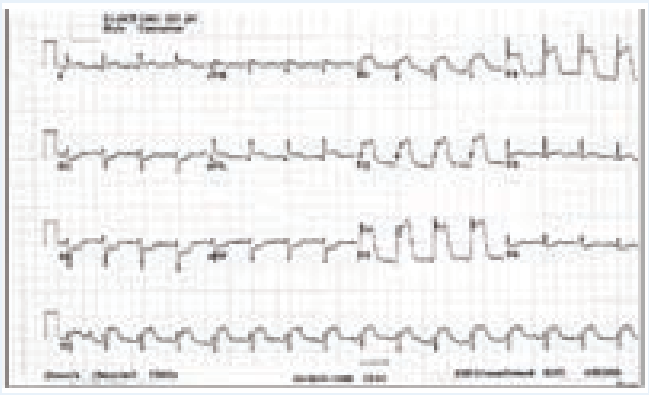


Figure 4: ECG from a patient experiencing a large heart attack (STEMI). Notice the obvious ST segment elevation in leads V2-V4.

Maintaining proper body weight is also essential. Obesity is highly prevalent in our society and contributes to the risk factors for heart attack. The body mass index (BMI) is used to estimate an individual's ideal body weight. It is calculated by dividing a person's body weight by their body surface area. Individuals can go to the Worldwide Web (www.nhlbisupport.com/bmi/) to calculate their own BMI. BMI results are categorized below:

- Healthy BMI=19-24
- Overweight BMI=25-29
- Obese BMI=30-39
- Severely Obese BMI>40

If an individual's BMI suggests that they are overweight, caloric intake should be reduced until a healthy BMI is achieved. Measurements of waist circumference have also proven to be a useful tool. Men need to maintain a waist circumference less than 40 inches. Women need to maintain a waist circumference less than 35 inches.

The final therapeutic lifestyle change that must occur to reduce an individual's risk of heart attack is cessation of all tobacco products.

Physicians can help their patients lower their risk of heart attack by monitoring and treating high cholesterol levels. The bad cholesterol is called LDL. Good cholesterol is called HDL. The LDL cholesterol is our primary target. A patient's LDL goal varies depending on their risk:

- <160 if 1 or fewer risk factors are present
- <130 if 2 or more risk factors are present and 10 year risk is <20%
- <70-100 if 2 or more risk factors are present and 10 year risk is ≥20%

Generally, if a patient's LDL cholesterol is not appropriate, a physician will initially recommend therapeutic lifestyle changes. If a patient cannot achieve the target LDL cholesterol after 6-12 weeks of therapeutic lifestyle changes, pharmacologic therapy should be initiated. Preferably, an HMG coenzyme A reductase inhibitor (also known as "statin") should be started. Physicians will need to monitor the fasting lipid profile periodically and may have to titrate statin doses to achieve the target LDL cholesterol level. After reaching a patient's target LDL cholesterol, secondary targets such as Non-HDL cholesterol (calculated as total cholesterol minus HDL), HDL cholesterol, and triglycerides may need to be addressed.

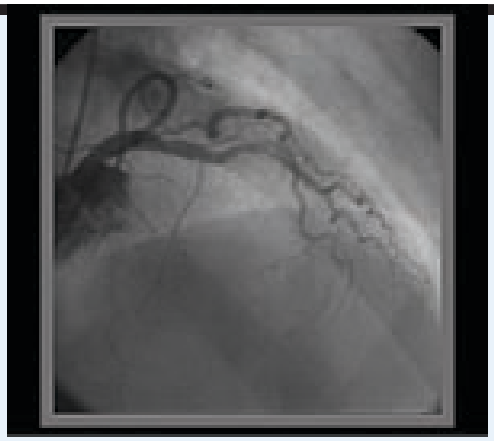


Figure 5: Cornary angiogram demonstrating an occluded left anterior descending coronary artery.

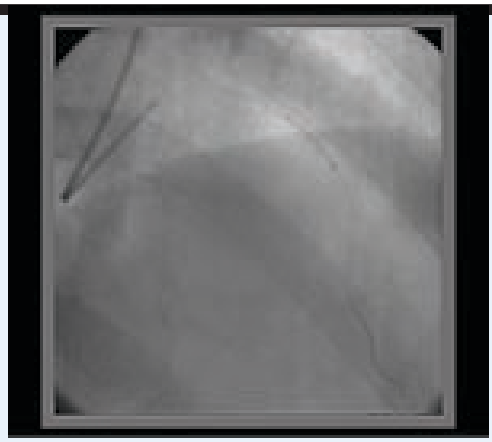


Figure 6: Image demonstrates placement of a thin guidewire across the blockage. A balloon tipped catheter with a premounted stent was advanced over the guidewire across the blockage.

If an individual's blood pressure is high, it must be aggressively treated to lower the risk for heart attack. For most people, ideal blood pressure is less than 140/90. For patients with diabetes, the ideal blood pressure is less than 130/85. If a physician documents high blood pressure readings on three separate measurements, blood pressure lowering medicines should be initiated.

Patients who have diabetes are at increased risk for heart attack. Diligent treatment is required to reduce their risk. In addition to targeting their LDL cholesterol to less than 70-100, and maintaining their blood pressure to less than 130/85, their blood sugars must be treated uncompromisingly. Fasting blood sugars should be maintained below 100 mg/dL and glycosylated hemoglobin A1C levels below 7.0.

The final recommendation that can be made to reduce an individual's risk of heart attack is aspirin administration. It is advised that individuals whose calculated 10 year risk of having a heart attack is greater than 10 percent be treated with aspirin on a daily basis as long as there are no contraindications. The ideal dose of aspirin to prevent heart attack is still controversial, based on scientific studies, but most physicians recommend a dose of 81 mg daily (one baby aspirin).

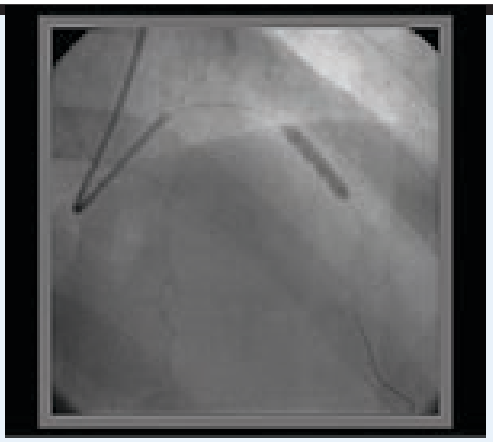


Figure 7: Image demonstrates inflated balloon to open the blockage and deploy the stent.

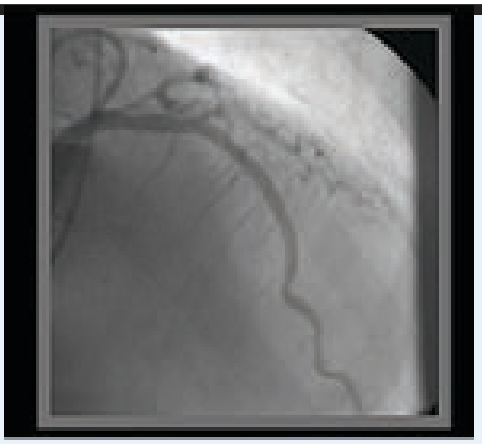


Figure 8: Final coronary angiogram demonstrates restored blood flow in the left anterior descending coronary artery after stent deployment.

WHAT ARE THE SYMPTOMS OF HEART ATTACK?

The typical symptoms of heart attack include chest pain, usually described as a pressure or tightness, that can travel toward the jaw or down the left arm. The chest pain is frequently associated with shortness of breath, nausea, and/or a cold sweat. Please be aware that many patients experience atypical symptoms while they are experiencing a heart attack. Women are more likely to experience atypical symptoms during a heart attack than men. Therefore, physicians should consider the diagnosis of heart attack or coronary artery disease in all patients presenting with the acute onset of chest pain or shortness of breath.

WHAT TO DO IF SOMEONE IS HAVING A HEART ATTACK?

If an individual is experiencing symptoms that may be caused by a heart attack for more than five minutes, they should call 911. Many heart attack patients make the mistake of enduring their symptoms at home far too long before seeking medical attention. As time progresses, the amount of heart muscle that is irreversibly damaged will increase; and the amount of heart muscle that is salvageable with restoration of blood flow will decrease. During a heart attack, time is muscle, and every minute counts! Another common mistake made by many heart attack patients is that they try to drive themselves to the hospital or they ask a family member to drive them. Emergency medical service personnel who are activated by a 911 phone call are capable of transporting heart attack patients to the hospital more quickly and safely than the patient or family members. For these reasons, an individual who is experiencing symptoms that may be caused by a heart attack for more than five minutes should call 911, fast! While waiting for the ambulance to arrive, the patient can chew and swallow four baby aspirin (81 mg) or one adult aspirin (325 mg).

Upon arrival to an emergency room, patients with heart attack symptoms need to be assessed promptly. Initial assessment needs to include an electrocardiogram (ECG). National guidelines recommend that patients presenting with heart attack symptoms have an ECG performed and reviewed by a physician within ten minutes of hospital arrival. Patients who are experiencing a large heart attack (STEMI) will have an obvious abnormality on their ECG which demonstrates at least 1 mm of ST segment elevation in two contiguous leads (figure 4).

If an ECG reveals that a patient is experiencing a large heart attack (STEMI), the patient needs to be treated as soon as possible to restore blood flow to the obstructed coronary artery. The patient's chance of surviving the heart attack is improved with quick restoration of blood flow. There are two common methods of restoring blood flow through an acutely obstructed coronary artery.

The first method involves the intravenous administration of a class of medications called thrombolytics. Thrombolytics dissolve clots inside the body. Due to widespread availability, administering thrombolytics to treat patients with large heart attacks (STEMI) is considered the standard of care for the majority of hospitals in the United States. National guidelines recommend that if thrombolytic therapy is the chosen treatment strategy, it be administered to patients with STEMI within 30 minutes of patient arrival to the hospital. Unfortunately, the thrombolytic treatment strategy is imperfect. Thrombolytics are only 50-60 percent effective at restoring blood flow in the acutely obstructed coronary artery. They are also associated with a one percent chance of major intracranial bleeding that usually results in a massive stroke or death.

The preferred alternative treatment strategy to restore coronary blood flow involves a technique called percutaneous coronary intervention (PCI). PCI requires that a patient be taken to a cardiac catheterization laboratory emergently. A long thin tube called a catheter is advanced into the groin area, through the femoral artery all the way up to the heart. Contrast dye is injected directly into the blood vessels that feed the heart to obtain x-ray pictures of the coronary arteries. These pictures are called a coronary angiogram, and they will show the location of the acutely obstructed coronary artery (figure 5). Next, a thin guidewire is advanced across the blockage. A balloon tipped catheter with a premounted stent is then advanced over the guidewire across the blockage (figure 6). The balloon is inflated to open the blockage and deploy (figure 7) a mesh stainless steel tube (called a stent) that creates a scaffold to keep the blood vessel open (figure 8).

PCI is the preferred treatment strategy because it is more than 95 percent effective at restoring blood flow in an acutely obstructed coronary artery. Also, the one percent risk of massive intracranial bleeding that occurs with the thrombolytic strategy is not present with PCI. Unfortunately, the majority of hospitals in the United States do not have cardiac catheterization laboratories capable of performing PCI. Furthermore, national heart attack guidelines recommend that if the PCI strategy is chosen to treat patients with large heart attacks (STEMI), that it be performed with a *Door to Balloon Time* less than 90 minutes. *Door to Balloon Time* refers to the time that passes from the patient's arrival at the hospital to the moment that the balloon is inflated in the obstructed coronary artery.

Interestingly, a recently published study that includes data from over 400 PCI capable hospitals in the United States demonstrates that the average *door to balloon time* in the best performing quartile of hospitals is more than 90 minutes (figure 9). The doctors at Oklahoma Heart Institute are dedicated to providing their heart attack patients with the finest care possible. With Oklahoma Heart Institute's leadership, our flagship hospital, Hillcrest Medical Center, has created a critical

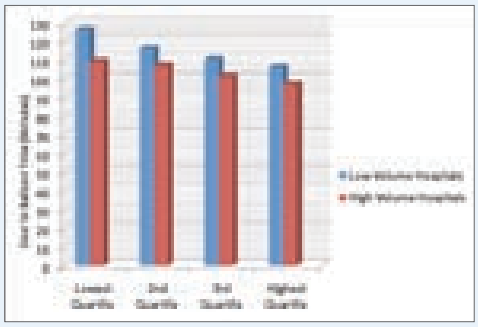


Figure 9: Data published from the National Registry of Myocardial Infarction revealing that the average door to balloon time in the best performing quartile of hospitals is more than 90 minutes in STEMI patients.

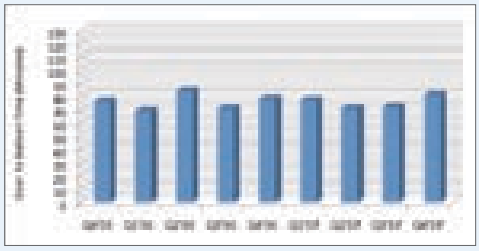


Figure 10: Median *Door to Balloon Times* have consistently been below 90 minutes over the last nine quarters in STEMI patients treated at Hillcrest Medical Center since the doctors at Oklahoma Heart Institute created a critical pathway heart attack protocol which is constantly scrutinized by a quality improvement initiative.

pathway heart attack protocol which is constantly scrutinized by a quality improvement initiative. Through these efforts, we have maintained a median *Door to Balloon Time* of less than 90 minutes consistently over the last nine quarters (figure 10). The doctors at Oklahoma Heart Institute are also embarking on a heart attack transfer protocol that would allow us to treat patients who present to rural hospitals with a large heart attack (STEMI) with timely emergent PCI rather than exposing them to the risk of massive intracranial bleeding associated with the thrombolytic therapy they would otherwise receive at the rural hospital.

CONCLUSIONS

Heart disease continues to be the leading killer in the United States. A large number of heart disease related deaths are due to heart attack. A heart attack occurs because a coronary artery becomes acutely obstructed, which results in heart muscle starved for blood and oxygen. An individual can reduce his or her risk of heart attack with preventive strategies that include therapeutic lifestyle changes, cessation of all tobacco products, cholesterol management, blood pressure control, aggressive diabetes treatment, and aspirin therapy. If an individual suspects they are having a heart attack, they need to call 911 immediately! Remember, time is muscle, and every minute counts.

Dr. Chandwaney is an interventional cardiologist at Oklahoma Heart Institute with expertise in cardiac catheterization, coronary angioplasty, and related interventional procedures such as coronary stents, atherectomy, intravascular ultrasound, and peripheral vascular interventional procedures. He is Medical Director of the Hillcrest Chest Pain Center and President-Elect of the American Heart Association-Tulsa Chapter. Dr. Chandwaney is Board Certified in Internal Medicine, Cardiovascular Disease, Interventional Cardiology, and Endovascular Medicine.

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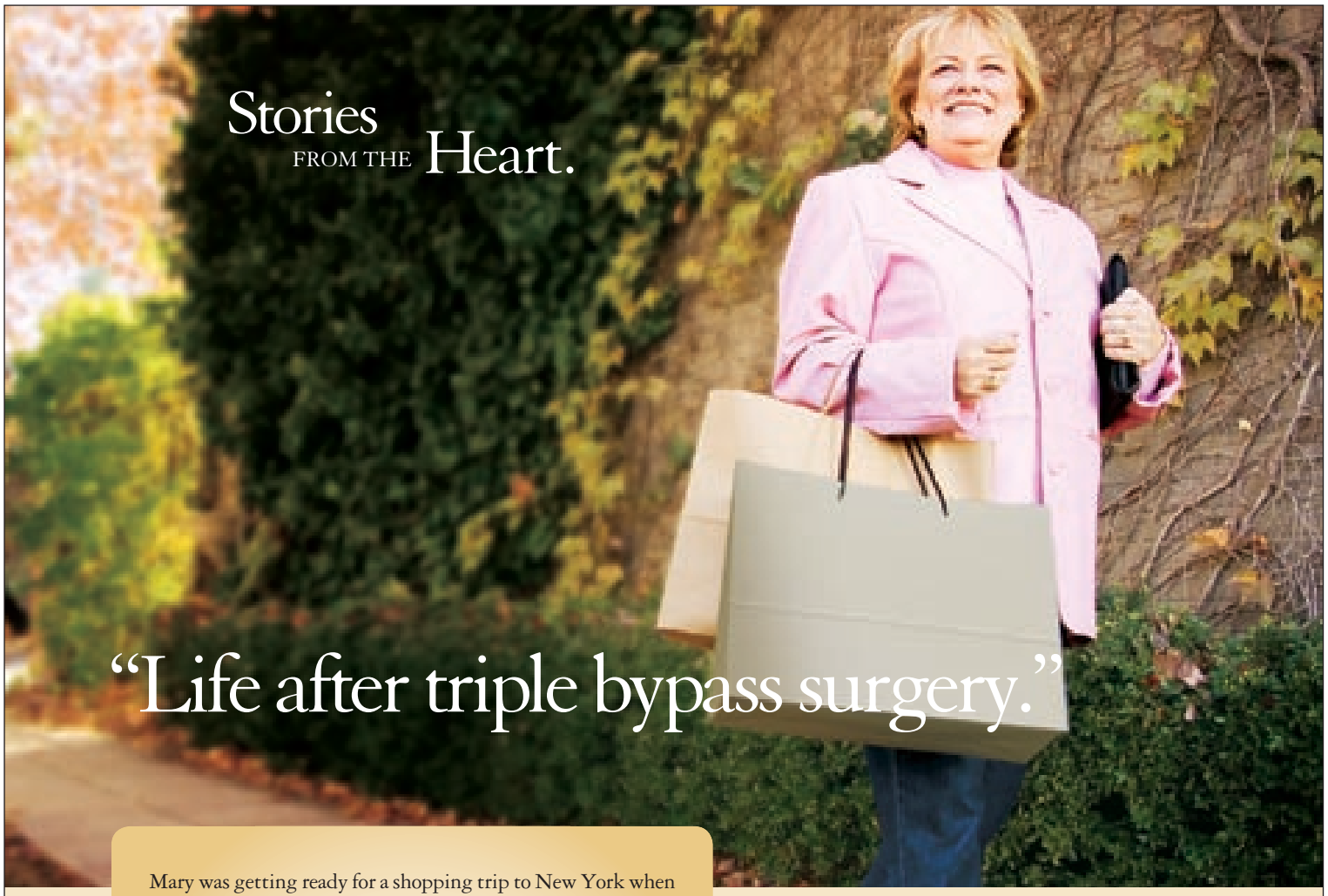

Mary was getting ready for a shopping trip to New York when she experienced shortness of breath. • Mary is a diabetic and decided it would be smart to see a doctor before she left. She was immediately referred to Hillcrest Medical Center where she had triple by-pass surgery. • Just a few months after surgery and with the support from the Cardiac Rehab Team, Mary can't believe how good she feels. • She has lost weight, no longer gets short of breath and is ready to begin hiking and biking. • Right after her shopping trip to New York, that is.

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