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CARDIOVASCULAR UPDATE

CLINICAL CARDIOLOGY AND CARDIOVASCULAR SURGERY NEWS

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Inside This Issue

New Chair of the Division of Cardiovascular Diseases at Mayo Clinic in Arizona

Win-Kuang Shen, MD

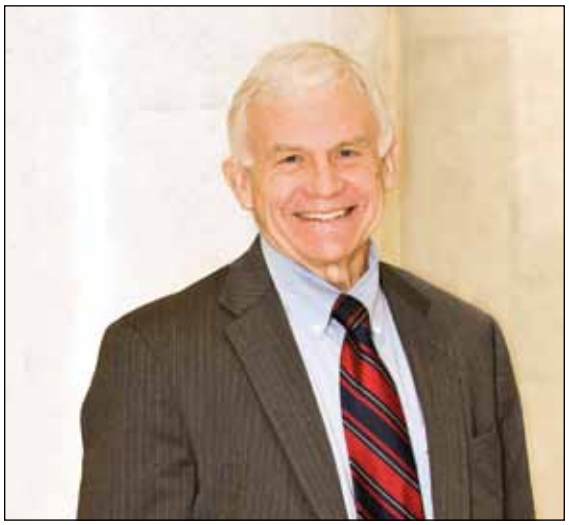
Win-Kuang Shen, MD, professor of medicine at the College of Medicine, Mayo Clinic, has been named the chair of the Division of Cardiovascular Diseases at Mayo Clinic in Arizona. Dr Shen graduated from New York Medical College in 1983 and completed postgraduate training in internal medicine, cardiology, and electrophysiology at Mayo Clinic and Duke University. He has a strong interest in medical education, previously serving as associate program director and clinical research director for the Mayo Clinic Internal Medicine Program, and has received multiple teaching awards from medical students, residents, and fellows. He is board certified by the American Board of Internal Medicine, with subspecialty boards in cardiology and electrophysiology.

Dr Shen has served on the American Heart Association's Basic Science Council and Council on Clinical Cardiology and the scientific program committee for the Heart Rhythm Society and the American Heart Association. He also has served on guideline writing committees jointly held by national and international societies. He has authored and coauthored more than 160 original scientific publications in peer-reviewed journals, including *Circulation Research*, *American Journal of Physiology*, *New England Journal of Medicine*, *Annals of Internal Medicine*, *JAMA, Circulation*, and *JACC*. He has written more than 30 book chapters and has coedited 2 books. His research interests include atrial fibrillation, mechanisms of syncope, and cellular electrophysiology. He has been very active also in international cardiology efforts. He has been regularly involved in international scientific programs providing educational opportunities for colleagues from many countries. In addition to his research and educational endeavors, Dr Shen continues to maintain an active clinical practice in cardiac electrophysiology involving ablation therapy for complex arrhythmias and device implantation.

ANNOUNCEMENT

Mayo Clinic's new network relationship with UnitedHealthcare gives UnitedHealthcare commercial plan customers in-network access to Mayo Clinic physicians and hospitals beginning November 1, 2010. The new network relationship covers all Mayo group practices and hospitals in Arizona, Florida, and Minnesota. More information is available at http://www.mayoclinic.org/news2010-rst/5993.html.

CREST Results Demonstrate Carotid Artery Stenting Is Effective and Safe



David R. Holmes Jr, MD

Carotid endarterectomy (CEA) has long been the standard treatment for symptomatic or hemodynamically significant carotid artery stenosis. The introduction of carotid artery stenting (CAS) has provided another, sometimes controversial option in the treatment of carotid artery disease. The debate is the result of the design of prior studies, lack of good controlled data, and issues of low operator stenting experience, all of which may have contributed to the potential for complications and error.

The results of the Carotid Revascularization Endarterectomy vs Stenting Trial (CREST) were published in the past year (*New England Journal of Medicine*, July 1, 2010). This multicenter trial enrolled 2,502 patients at 108 centers in the United States and 9 in Canada. Centers were required to have a multidisciplinary team consisting of a neurologist, an interventionist, a surgeon, and a research coordinator. Selection criteria were carefully documented and were in strict compliance. The interventionists were certified on the basis of their carotid stenting results, participation in hands-on training, and participation in a lead-in phase of training. The surgeons also had to document their experience. "These requirements satisfied some of the concerns and issues related to uneven operator experience in the prior 2 trials," according to David R. Holmes Jr, MD, an interventional cardiologist at Mayo Clinic in Rochester, Minnesota.

This large trial included both symptomatic (53%) and asymptomatic (47%) patients with carotid artery stenosis. The eligibility criteria used were stenosis of 50% or more at the time of angiography, 70% or more at the time of duplex ultrasonography, or 70% or more on CT angiography or magnetic resonance angiography. Criteria were expanded during the course of the study to include asymptomatic patients. Patient exclusion criteria included prior stroke severe enough to confound the assessment of study end points or another potential cause for stroke such as atrial fibrillation or the presence of unstable angina. Of note, the patients could be randomized on the basis of ultrasonography criteria. This stipulation had important implications; some patients who were randomly assigned to carotid stenting at the time of intervention were subsequently found to have anatomic characteristics that made them not suitable for carotid stenting, such as the lack of a distal landing zone for the embolic protection device. However, using intention-to-treat methods, data from these patients were carried forward as though they continued to be enrolled in the stenting arm of the study. Patients were randomly assigned to either conventional endarterectomy or carotid stenting with a relatively early-generation stent and a distal embolic protection filter.

The primary end point was a composite of any stroke, myocardial infarction, or death during the periprocedural period or an ipsilateral stroke within 4 years of randomization; there was no significant difference in this primary end point between CAS and CEA (7.2% vs 6.8%; P=.51). During the periprocedural period, the incidence of the primary end point was also similar; however, there were differences in specific individual end points. While there was no difference in death (0.7% with CAS, 0.3% with CEA), stroke, which was typically minor, occurred more frequently in CAS (4.1% vs 2.3% in CEA; P=.01), while myocardial infarction occurred more frequently with CEA (2.3% vs 1.1% in CAS; P=.03). There was a marked imbalance in cranial nerve palsy, which occurred in 4.7% of CEA patients vs 0.3% of CAS patients. After the periprocedural period, ipsilateral stroke was similarly low in both treatment arms (2.1% with CAS and 2.4% with CEA).

The 4-year rate of stroke or death in the CAS group was 6.4% vs 4.7% in the CEA group. In patients who were symptomatic, the respective rates were 8.0% vs 6.4% (P=.14); in asymptomatic patients, those rates were 4.5% vs 2.7% (P=.07).

The CREST results document that CAS and CEA in experienced hands are associated with similar rates of primary composite outcome. Procedural stroke, myocardial infarction, or death and subsequent ipsilateral stroke among patients with either symptomatic or asymptomatic carotid stenosis were not significantly different between patients treated with stenting and those treated with endarterectomy. There was a differ-

ence in the individual end points, with more CAS patients having stroke, which was typically minor, while more CEA patients had myocardial infarction and cranial nerve palsy.

Accordingly, the authors concluded that carotid revascularization performed by highly qualified surgeons and interventionists is effective and safe and that the low absolute risk of recurrent stroke suggests that both CAS and CEA are clinically durable.

"Subsequently, there have been other important developments to be considered. The concern about periprocedural embolism has been addressed using new proximal protection devices, which have drastically reduced the incidence of embolic strokes associated with stenting," says Dr Holmes. "The trial results indicate that the therapeutic choice should be individualized on the basis of anatomic considerations, comorbid conditions, and patient choice."

New Antithrombotic Released for Stroke Prophylaxis in Atrial Fibrillation

Robert D. McBane, MD, Waldemar E. Wysokinski, MD, PhD

Atrial fibrillation is a major source of morbidity and mortality in the United States and increases the risk of ischemic stroke 5-fold, causing more than 200,000 events annually. Warfarin therapy reduces this risk by 64%. Yet warfarin therapy can be difficult to manage because of high variability within and between patients, its narrow therapeutic range, and the interaction with diet and medications. Warfarin use requires lifelong, frequent assessment of each patient's international normalized ratio (INR) for dose adjustment and is associated with increases in the risk of major hemorrhage, particularly in elderly patients. Collectively, these variables limit clinicians' enthusiasm for warfarin initiation in atrial fibrillation patients. Currently, only 50% of patients with atrial fibrillation who would benefit from warfarin therapy receive it, and the discontinuation rates are high. At 1 year, more than 25% of patients stop warfarin despite an ongoing indication for this drug. For these reasons, the prospect of antithrombotic prophylaxis strategies requiring neither monitoring nor dose adjustment is particularly attractive.

Recently, an oral direct reversible thrombin inhibitor, dabigatran, was approved by the US Food and Drug Administration (FDA) for the indication of stroke prevention in patients with nonvalvular atrial fibrillation. This drug is formulated as a prodrug and is metabolized to the active compound on ingestion. The time to peak anticoagulant effect is about 1 hour, and the drug half-life is about 15 hours, with 80% of elimination through the kidney. Dabigatran has been approved in 2 tablet strengths, 150 mg and 75 mg, given twice daily. Because of the drug's renal clearance, the 150-mg dose is indicated for patients with reasonable kidney function (creatinine clearance exceeding 30 mL/min). For patients with limited renal function (creatinine clearance of 15-30 mL/min), the 75-mg tablet strength is recommended (although this dose is untested in clinical trials). The drug should not be used in patients with more advanced kidney disease. "At currently available prices, dabigatran is approximately 3 times more expensive than warfarin plus INR monitoring (assuming 5 mg/day warfarin dosing with once-monthly INR monitoring)," according to Waldemar E. Wysokinski, MD, PhD, a cardiologist in the Gonda Vascular Center at Mayo Clinic in Rochester, Minnesota.

Dabigatran can be thought of as a second-generation oral direct thrombin inhibitor. Ximelagatran was a first-generation agent, which, though effective, did not receive FDA approval, largely stemming from concerns of liver toxicity. Compared with warfarin and enoxaparin, dabigatran has no greater risk of liver function test abnormality, defined as alanine aminotransferase levels greater than 3 times the upper limit of normal. At this

point, there is no recommendation regarding liver function test monitoring for patients receiving this drug.

The Randomized Evaluation of Long-term Anticoagulant Therapy (RE-LY) trial assessed the efficacy and safety of dabigatran compared with warfarin in 18,113 patients with nonvalvular atrial fibrillation. This was a noninferiority randomized trial design, open label with respect to warfarin and blinded with respect to 2 doses of dabigatran (110 and 150 mg twice daily). To qualify for trial participation, patients had to have nonvalvular atrial fibrillation plus at least 1 risk factor, including a prior thromboembolic event, an ejection fraction less than 40%, heart failure, or age of 75 years or older. Younger patients aged 65 to 74 years were eligible to participate if they had diabetes mellitus, coronary artery disease, or hypertension.

After a mean follow-up of 30 months, participants randomly assigned to receive dabigatran, 150 mg twice daily, experienced a significantly lower incidence of a primary end point (stroke or systemic embolism) compared with dabigatran, 110 mg twice daily, and warfarin: 1.1%/year vs 1.5%/year vs 1.7%/year. This improvement did not come at a cost of major bleeding, which was similar for patients receiving 150 mg of dabigatran and warfarin patients: 3.1%/year vs 3.4%/year. Life-threatening bleeding was also similar between these 2 groups: 1.5%/year vs 1.9%/year. Hemorrhagic stroke was in fact significantly lower in patients receiving 150 mg of dabigatran compared with those receiving warfarin: 0.1%/year vs 0.4%/year.

In a post hoc analysis, warfarin-treated patients were divided by treatment center on the basis of the time in the therapeutic INR range (above and below the 67% median). Hazard ratios were constructed to compare patients receiving warfarin and dabigatran. For patients receiving inadequate INR management from participating centers by this definition, the hazard was superior for dabigatran-treated patients for rate of embolism, major hemorrhage, and mortality compared with warfarin-treated patients. In contrast, for patients receiving INR management from participating centers above this median INR adequacy, there was no difference with respect to these outcomes. The average time within the therapeutic INR range observed in patients treated with warfarin worldwide is below the 67% median achieved in the RE-LY trial.

Patients receiving dabigatran do not require anticoagulant monitoring. In some circumstances, however, coagulation testing may be performed or required. Dabigatran has been shown to prolong both the activated partial thromboplastin time (aPTT) and prothrombin time. The aPTT will be about twice the control value within 1 hour of ingestion. At 12 hours after ingestion, when the next dose is taken (on a regimen of twice-daily dosing), the aPTT will still be about 1.5 times the basal values. Previous studies showed that coagulation parameters closely followed drug concentrations. Single, orally administered doses (10-400 mg) led to rapid, dose-dependent increases in aPTT, mean INR, thrombin time, and ecarin clotting time, with the maximum anticoagulant effect occurring at the maximum plasma dabigatran concentration. The aPTT may be helpful in assessing the circulating dabigatran levels in a patient for whom an invasive procedure is needed. In general, dabigatran administration should be stopped for 4 or 5 half-lives (60-75 hours) before an invasive procedure to ensure that all drug has been eliminated. Whereas dabigatran is a small-molecule direct thrombin inhibitor, there is no antidote or reversing agent. Particularly for patients undergoing surgical procedures with a high risk of major bleeding, this is an important management point to consider.

"For patients treated with dabigatran who have a major bleeding event, health care providers have several treatment options," according to Robert D. McBane, MD, a cardiologist in the Gonda Vascular Center. First, it is important to recall that major bleeding events are rare; between 1.3% and 2% have been reported in the large randomized trials published to date. The second and perhaps more important point is to support the patient with red blood cell transfusion, fluids, and pressure support as needed. Equally important is the identification and correction of all sources of bleeding, which may include endoscopy, surgery, and interventional radiology (angiography and coiling) where indicated. Third, whereas dabigatran is a small molecule, it is to some degree (60%) dialyzable. Fourth, some have advocated the use of additional hemostatic agents, including FEIBA (factor VIII inhibitor bypass activity used for patients with factor VIII inhibitors), prothrombin complex concentrates, and recombinant factor VII when needed. These agents should be used with caution and only when absolutely needed as they undoubtedly swing the hemostatic pendulum toward thrombosis. It is important to recall that patients receiving dabigatran are doing so for an antithrombotic indication.

In summary, dabigatran represents a new era of anticoagulant therapy. For the indication of nonvalvular atrial fibrillation, dabigatran is a reasonable alternative to warfarin therapy. "Its use should be restricted to the FDA-approved indication of nonvalvular atrial fibrillation," says Dr McBane. Ideal candidates for this medication include those patients whose warfarin is difficult to manage and who have widely fluctuating INR values, those for whom good anticoagulation management is not available, and those living remotely. For those patients with ready access to good anticoagulant management, dabigatran and warfarin are likely equivalent therapies.

Management Guidelines for Acute Aortic Dissection and the International Registry of Acute Dissection

Thoralf M. Sundt III, MD

Jae K. Oh, MD

Cardiovascular Surgery

Hartzell V. Schaff, MD, ***Director***
Harold M. Burkhart, MD
Richard C. Daly, MD
Joseph A. Dearani, MD
Kevin L. Greason, MD
Lyle D. Joyce, MD, PhD
Soon J. Park, MD
Thoralf M. Sundt III, MD
Rakesh M. Suri, MD, DPhil

Acute dissection is the most common fatal aortic catastrophe. Although abdominal aortic aneurysms occur more frequently than thoracic aortic dissections, they less often present with rupture and, when they do so, are less often fatal than ruptured thoracic aortic dissections. The incidence of aortic dissection is estimated at 10 to 15 per 100,000 adults in the United States annually. Accordingly, while thoracic aortic dissections are uncommon, their malignant course makes them an important cause of cardiovascular morbidity and mortality.

The underlying cause of aortic dissection is medial degeneration. This may be secondary to inherited connective tissue diseases such as Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome, or any of a family of thoracic aortic aneurysm and dissection syndromes. More commonly, medial degeneration occurs secondary to the ravages of hypertension over time. Tobacco use accelerates the process. Dissections also occur more commonly among patients with aortic dilation as increasing aortic diameter increases wall tension and the mechanical stress placed on the aortic tissues. Rarely, dissections occur during pregnancy, most often among individuals with connective tissue disorders. Dissections tend to occur somewhat earlier in men (peak incidence in their 50s to 60s) than in women (peak incidence in their 60s to 70s).

Thoracic dissections are classified anatomically as Stanford type A if the ascending aorta is involved, and Stanford type B if the dissection is confined to the descending thoracic or thoracoabdominal aorta (Figure). The associated natural history of type A and type B dissections is markedly different: if treated nonoperatively, the mortality rate during the index hospitalization for type A dissections may be as high as 80%, while that for surgical treatment is 10% to 25%. The preferred treatment algorithm is clear: acute type A dissections are treated surgically on an urgent or emergent basis except under unusual circumstances (such as advanced age or comorbid conditions such as acute stroke).

The preferred treatment of type B dissections is more controversial, particularly today in the era of endovascular stent grafts. Historically, the mortality rate associated with medical treatment of type B dissections (approximately 10%) has been clearly less than that for open surgical repair (approximately 30%). Accordingly, the preferred treatment for a patient with type B dissection is aggressive blood pressure control. But when, then, is intervention indicated?

Our understanding of the clinical presentation and outcome of acute aortic dissection has been greatly enhanced through the efforts of the International Registry of Acute Aortic Dissection (IRAD). The IRAD database was established in 1996 by a group of interested investigators, including Thoralf M. Sundt III, MD, a cardiovascular surgeon at Mayo Clinic in Rochester, Minnesota, and Jae K. Oh, MD, a cardiologist at Mayo Clinic in Rochester, who recognized that there was a paucity of information about this disease. The database initially included 12 large referral centers in 6 countries and has now grown to include 24 centers in 12 countries. A recent publication from this group reviewed the impact of refractory pain and persistent hypertension on outcome and, by inference, their role as indications for surgical intervention among patients with type B dissection. In their series of 365 patients with uncomplicated acute type B dissection—type B dissection without rupture or malperfusion—69 demonstrated refractory pain or refractory hypertension despite best medical therapy. While the overall in-hospital mortality was 6.5% for all 365 patients with type B dissection, it was dramatically higher among the 69 patients demonstrating refractory pain or hypertension (17.4% vs 4% for the remainder; $P<.001$). Furthermore, among the 69 patients with refractory pain or hypertension, the mortality rate among those in whom no intervention was undertaken was 35.6% and was significantly higher than the mortality rate for those treated surgically (20%) or endovascularly (3.7%). "These data support the notion that recurrent pain and refractory hypertension should encourage a more aggressive interventional approach to patients with type B dissection," says Dr Sundt. The data are further suggestive, although not definitive proof, of a role for endovascular stent grafts in this subset of patients. It must be clearly stated, however, that endovascular stent grafts are currently approved by the US Food and Drug

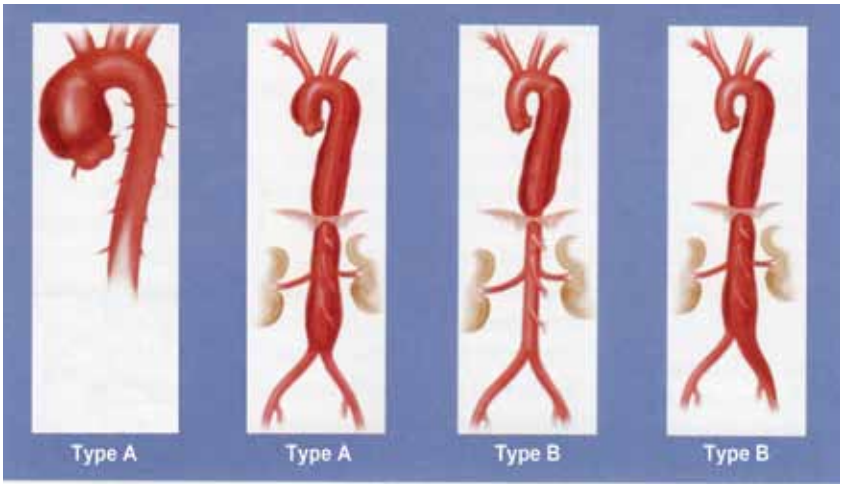


Figure. Stanford classification of aortic dissection. Both types may extend below the diaphragm.

Administration for the treatment of aneurysmal disease, but not dissection.

Of equal interest in this analysis was the outcome of the patients with uncomplicated type B dissection who did not experience refractory pain or hypertension. The mortality rate among medically treated patients was 1.5%. This is of particular note as interest has risen in the possible role of endovascular stent grafting among patients with uncomplicated dissection in the hope of preventing late complications. Clearly, any intervention, be it surgical or endovascular, must be accomplished among these patients without incremental increase in this risk.

Finally, it is important to remember that aortic dissection is a chronic condition. "Once a patient has experienced aortic dissection, whether type A or type B, he or she should be followed carefully with aggressive control of blood pressure and serial imaging studies to observe for aneurysmal dilation of the injured aorta," says Dr Oh. All too often, patients with acute dissection are lost to follow-up after an initial surgical repair or successful nonoperative hospitalization at the time of the acute event. As many as 30% of patients with dissection of the descending thoracic and thoracoabdominal aorta ultimately demonstrate expansion of the aorta sufficient to warrant consideration of surgical intervention.

For additional information on the IRAD registry, please see the Web site, http://www.iradonline.org/.

Interested in receiving cardiovascular updates on patient care, research, and education electronically? To sign up, go to http://www.mayoclinic.org/publications/medicalprofs-enews.html

RECOGNITION

Leslie T. Cooper Jr, MD, is the new director of the Gonda Vascular Center at Mayo Clinic in Rochester, Minnesota. Dr Cooper has been a member of the Mayo Clinic staff since 1997 and has served as the director of the Section of Vascular Medicine since 2008. He is a professor of medicine at the College of Medicine, Mayo Clinic, and is board certified by the American Board of Internal Medicine, with subspecialty boards in cardiology and vascular medicine. Dr Cooper also performs research in myocarditis, currently funded by 3 National Institutes of Health grants, and has served as president and chairman of the board of the Myocarditis Foundation.

RECOGNITION

Virend K. Somers, MD, PhD, has received the Mayo Clinic Department of Medicine Landmark Contribution to the Literature Award. Veronique L. Roger, MD, has received the Department of Medicine Outstanding Investigator Award. Both are members of the Division of Cardiovascular Diseases.

IN THE NEWS

Healthy Living Rochester

CardioVision 2020, originally established in 1999 as a community-based program aimed at reducing the burden of cardiovascular disease in Olmsted County, Minnesota, recently underwent a facelift and update to reflect its widening efforts to help improve the health of people in the Rochester, Minnesota, area. This new effort has occurred with the help of a number of groups, including Stratis Health, which supplied a community health grant to help fund the project, and other groups from the Rochester area, including CardioVision 2020, Active Living Rochester, Mayo Clinic's Dan Abraham Healthy Living Center, Olmsted County Public Health Services, Rochester-Olmsted Planning, Rochester Public Works, and MLT Group Advertising & Marketing.

Under the direction of Randal J. Thomas, MD, director of the Cardiovascular Health Clinic at Mayo Clinic in Rochester, Healthy Living Rochester is aimed at implementing local activities and policies that promote physical activity, healthy nutrition, and tobacco-free living for people in the Rochester area. In addition, activities and policies are promoted to improve the identification, treatment, and control of cardiovascular risk factors and to help promote the optimal provision of preventive care to people with known cardiovascular disease.

An interactive Web site has been launched that encourages people from throughout the Rochester area to participate and help promote healthy living. Resources on the Web site are aimed at helping people identify ways in which they can improve their health and implement strategies to do so. The Web site also invites people to submit their ideas and experiences regarding healthy living, videos with positive and upbeat messages about healthy living, favorite recipes, and healthy hot spots around town, among other things.

To view the new Web site and its resources and to find out more about Healthy Living Rochester and how to get involved, go to www.healthylivingrochester.org.

CLINICAL TRIALS

Prior studies have demonstrated that administration of autologous bone marrow–derived mononuclear cells improves cardiac function in patients after acute myocardial infarction; however, optimal timing of this treatment is unknown. Mayo Clinic in Rochester, Minnesota, is participating in multicenter clinical trials sponsored by the Cardiovascular Cell Therapy Research Network, funded by the National Heart, Lung, and Blood Institute of the National Institutes of Health, and designed to 1) evaluate effects of bone marrow–derived mononuclear cells on regional and global left ventricular function compared with placebo therapy in patients with acute anterior myocardial infarction and 2) assess whether effects of bone marrow–derived mononuclear cells on global and regional left ventricular function and safety are influenced by the time of administration.

TIME: Transplantation in Myocardial Infarction Evaluation Protocol: A Phase 2, Randomized, Controlled, Double-Blind Trial Evaluating the Effect of Timing on the Administration of Bone Marrow Mononuclear Cells (BMMNCs) vs Placebo in Patients With Acute Myocardial Infarction: Bone marrow harvest is conducted at either 3 or 7 days after acute myocardial infarction. The cells are processed and returned for intracoronary infusion within 12 hours.

Late TIME: A Phase 2, Randomized, Controlled, Double-Blind Pilot Trial Evaluating the Safety and Effect of Administration of Bone Marrow Mononuclear Cells (BMMNCs) 2 to 3 Weeks Following Acute Myocardial Infarction: Bone marrow harvest is conducted at either 2 or 3 weeks after acute myocardial infarction. The cells are processed and returned for intracoronary infusion within 12 hours.

Inclusion criteria include moderate to large anterior acute myocardial infarction, successful percutaneous coronary intervention of the left anterior descending coronary artery, and left ventricular ejection fraction of 45% or less by echocardiography. For more information about enrolling patients in either of these clinical trials, please contact study coordinator Kelly Flood, RN, at 507-255-9524 or principal investigator Amir Lerman, MD, at 507-255-6670.

Upcoming Courses

Continuing Medical Education, Mayo Clinic

To request additional information or to register, unless noted otherwise, please call 800-323-2688, e-mail cme@mayo.edu, or visit www.mayo.edu/cme.

Arrhythmias and the Heart
Jan 31-Feb 3, 2011, Big Island, HI

Frequently Encountered Ethical Dilemmas in the Community Practice
Feb 2-4, 2011, Rochester, MN

36th Annual Cardiovascular Conference at Snowbird
Feb 9-12, 2011, Snowbird, UT

Translating New Findings Into Clinical Practice: A San Antonio Breast Meeting Update and Saving the Hearts of Women: Evaluation, Management and Prevention of Cardiovascular Disease
Feb 11-12, 2011, Jacksonville, FL

Optimal Treatment Strategies for Advanced Heart Failure
Feb 11-12, 2011, Scottsdale, AZ

16th Annual Cardiology at Cancun
Feb 28-Mar 4, 2011, Cancun, Mexico

18th Annual Echocardiographic Workshop on 2-D and Doppler Echocardiography at Vail
Mar 7-10, 2011, Vail, CO

7th Annual Women's Health Update
Mar 10-12, 2011, Scottsdale, AZ

Cases in Valvular Heart Disease
Apr 2, 2011, New Orleans, LA

Adult Congenital Heart Disease
Apr 2, 2011, New Orleans, LA

Multimodality Imaging in Cardiovascular Disease
Apr 2, 2011, New Orleans, LA

Ventricular Assist Devices: Managing the Advanced Heart Failure Patient
Apr 2, 2011, New Orleans, LA

Atrial Fibrillation Syndrome
Apr 3, 2011, New Orleans, LA

Echocardiography in the Nation's Capital: Focus for the Physician
Apr 11-13, 2011, Arlington, VA

Echocardiography in the Nation's Capital: Focus for the Sonographer
Apr 14-16, 2011, Arlington, VA

Echo Fiesta: An In-depth Review of Adult Echocardiography for Sonographers and Physicians
Apr 26-29, 2011, San Antonio, TX

Echocardiography Review Course for Boards and Certification
May 7-10, 2011, Rochester, MN

Controversies in Cardiovascular Disease
May 21-22, 2011, St Paul, MN

Controversies in Women's Health
Jul 14-16, 2011, Wisconsin Dells, WI

25th Annual Echocardiographic Symposium at Vail: New Technologies, Live Scanning, and Clinical Decision Making
Jul 25-28, 2011, Vail, CO

Cardiology Update in Sedona
Aug 5-7, 2011, Sedona, AZ

Success With Failure: New Strategies for the Evaluation and Treatment of Congestive Heart Failure
Aug 7-10, 2011, Whistler, BC

Mayo Clinic International Vascular Symposium
Sep 9-11, 2011, Paris, France

Electrophysiology for Boards and Recertification
Sep 9-11, 2011, Rochester, MN

Interventional Cardiology Board Review
Sep 9-11, 2011, Rochester, MN

Mayo Clinic Nutrition in Health and Disease
Sep 15-16, 2011, Seattle, WA

Mayo Cardiovascular Review Course for Cardiology Boards and Recertification
Sep 17-22, 2011, Rochester, MN

Echocardiography for the Sonographer: Focus on Adult Echocardiography
Sep 18-20, 2011, Rochester, MN

27th Annual Echocardiography in Pediatric and Adult Congenital Heart Disease
Oct 9-12, 2011, Rochester, MN

Imaging Ventricular Function in Congenital and Acquired Heart Disease: From Doppler to Deformation: State of the Art 2011
Oct 13-14, 2011, Rochester, MN

Cases in Echocardiography: TEE, Doppler, and Stress: Interpretation and Clinical Decision Making for the Advanced Echocardiographer
Oct 19-22, 2011, Napa, CA

Coronary Artery Disease: Prevention, Detection, and Treatment
Oct 20-23, 2011, Las Vegas, NV

Thoracic Oncology for the Non-Oncologist
Nov 5, 2011, Scottsdale, AZ

Echo in Marco Island: Case-Based Approach
Dec 1-4, 2011, Marco Island, FL

Other Education Opportunities

Echo Hawaii
Jan 24-28, 2011, Big Island, Kona, HI
www.asecho.org
Phone: 919-297-7157; e-mail: abuff@asecho.org

24th Annual State-of-the-Art Echocardiography
Preconference: Feb 11, 2011
General Session: Feb 12-16, 2011, Scottsdale, AZ
Phone: 919-297-7171; e-mail: dlewis@asecho.org

American College of Cardiology ACC.11: 60th Annual Scientific Session and Expo
Apr 2-5, 2011, New Orleans, LA
Phone: 800-699-5113; e-mail: accregistration@jspargo.com

Heart Rhythm Society 32nd Annual Scientific Sessions
May 4-7, 2011, San Francisco, CA
Web: www.hrsonline.org

American Society of Echocardiography 22nd Annual Scientific Sessions
Jun 11-14, 2011, Montreal, Quebec
Web: www.asecho.org

Mayo Clinic Cardiovascular Update

Editor: Margaret A. Lloyd, MD, MBA

Editorial Board: Charanjit S. Rihal, MD, MBA, Hartzell V. Schaff, MD, Frank Cetta, MD, Gurpreet S. Sandhu, MD, PhD, Hugh M. Martin, Marjorie G. Durhman

Managing Editor: Jane C. Wiggs, MLA, ELS

Art Director: Marjorie G. Durhman

Photography: Amanda R. Durhman

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MAYO CLINIC

200 First Street SW
Rochester, Minnesota 55905
www.mayoclinic.org