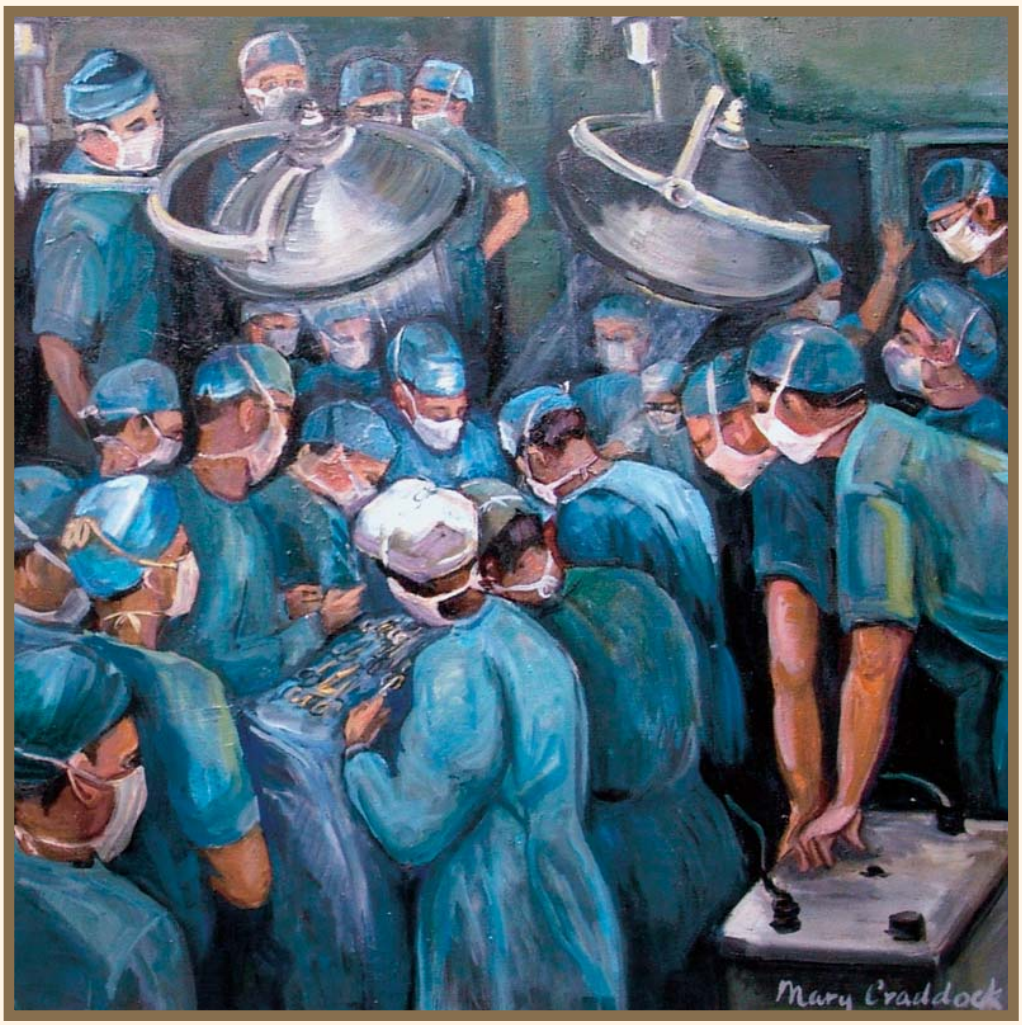
HeartWATCH

FALL 2007

A Newsletter Produced by the Texas Heart Institute



Texas Heart® Institute

at St. Luke's Episcopal Hospital

Emerging Predictors of Carotid Artery In-Stent Restenosis May Help Establish Treatment Guidelines

Abstract: Patients with carotid artery stents are at increased risk of restenosis if they have previously undergone a carotid endarterectomy or radiation therapy to the neck.

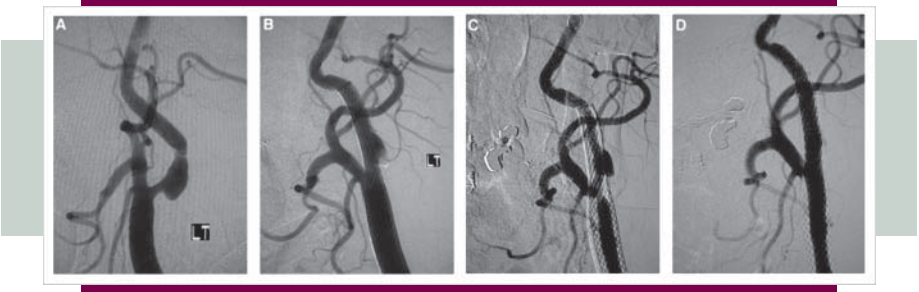
Percutaneous carotid artery stenting is a safe, effective, and, in some cases, superior alternative to carotid endarterectomy (CEA) for patients with symptomatic and asymptomatic carotid artery stenosis. Although restenosis is rare (incidence, 1.8% to 6.7%) after carotid artery stenting, there are no current guidelines for predicting or treating such restenosis. Thus, some researchers, including George A. Younis, MD, and Zvonimir Krajcer, MD, cardiologists at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI at SLEH), decided that more information was needed.

"To determine predictors of restenosis," says Dr. Younis, "we undertook a single-center retrospective study of 399 carotid stent procedures in 363 patients. We also wanted to review the clinical presentation of restenosis and evaluate the efficacy of endovascular techniques for its management" (*Cathet Cardiovasc Interv* 2007;69:673-82).

Their analysis revealed 2 possible predictors of restenosis after carotid artery stenting: a previous ipsilateral CEA and radiation therapy to the neck.

After a CEA, the risk of restenosis is highest during the first few years and decreases thereafter. Because a second CEA would pose a higher risk, carotid stent placement is considered a safe, effective alternative to reoperation. However, after placing stents in 57 carotid arteries with post-CEA restenosis, Drs. Younis and Krajcer found that in-stent restenosis occurred in 6 patients (10.5%), suggesting that patients with post-CEA restenosis are also at higher risk of in-stent restenosis.

The reported incidence of carotid disease in patients who have received radiotherapy to the neck is as high as 30%. Surgical treatment is difficult because of various radiotherapy complications, including radiation-induced arterial, periarterial, and cutaneous sclerosis; cranial nerve palsies; poor cutaneous healing; anastomotic rupture; and arterial infection. Carotid artery stenting is the preferred treatment for these patients; however, data collected by Drs. Younis and Krajcer suggest that postradiation patients also have a higher risk of carotid in-stent restenosis.



A) Angiography shows an 85% ulcerated proximal left internal carotid artery stenosis and a 50% to 60% ostial stenosis. **B)** Angiogram obtained after balloon angioplasty, placement of a 10×31-mm Wallstent (Boston Scientific, Natick, Mass), and postdilation with a 5×20-mm balloon. **C)** Angiogram obtained 1 year later shows at least an 80% in-stent stenosis. **D)** Angiogram obtained after predilation with a 5×20-mm cutting balloon (Boston Scientific) and placement of an 8×40-mm Precise stent (Cordis Corporation, Miami Lakes, Fla). Postdilation was performed with a 5×40-mm balloon. (From *Cathet Cardiovasc Intervent* 2007;69:673-82; with permission.)

"The most common mechanism of in-stent restenosis is myointimal hyperplasia with smooth muscle cell proliferation," says Dr. Younis. "Therefore, stent deployment in patients with preexisting, post-CEA hyperplasia or postradiation fibrosis may entail a higher rate of in-stent restenosis than stenting for de novo carotid artery atherosclerosis."

Unfortunately, the best options for managing carotid in-stent restenosis are not well defined, and surgical treatment can be especially challenging in these high-risk patients. In the THI at SLEH series of 363 patients, none required surgical treatment.

In the future, drug-eluting stents may be the most appropriate treatment for carotid in-stent restenosis in post-CEA and postradiation patients. The literature on coronary artery disease suggests that drug-eluting stents might decrease the incidence of restenosis in these patients. Another potential treatment includes the extended use of clopidogrel to prevent thrombotic complications.

"Obviously," says Dr. Younis, "further trials are needed so that we can better assess factors that may predict carotid stent restenosis and determine whether stent design, stent size, or deployment technique affects restenosis in post-CEA and postradiation patients. Until these factors are clarified, patients in these high-risk subgroups will continue to require careful observation." ●

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Endovascular Treatment of Thoracic Aortic Disease Is Beneficial for Selected Patients

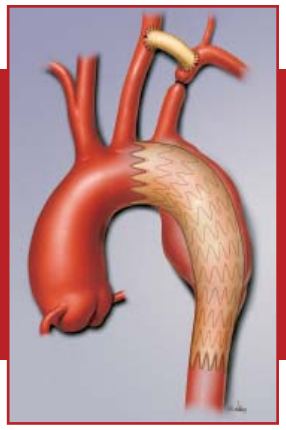
Abstract: Despite its potential complications, endovascular repair may be the best treatment approach for certain types of aneurysms and dissections of the thoracic aorta.

Because of the risk of rupture, aortic aneurysms and dissections often require surgical repair. When these lesions occur in the thoracic aorta, reparative operations are highly invasive and necessitate aortic clamping. Such procedures are particularly risky for patients with limited physiologic reserve.

For these reasons, considerable effort has been made to develop endovascular interventions for thoracic aortic disease. Among the surgeons at the forefront of this effort is Joseph S. Coselli, MD, chief of Adult Cardiac Surgery at the Texas Heart Institute at St. Luke's Episcopal Hospital. Dr. Coselli has written more than 100 journal articles and book chapters on aortic surgery, and he coauthored the Society of Thoracic Surgeons' "Guidelines for Credentialing of Practitioners to Perform Endovascular Stent-Grafting of the Thoracic Aorta" (*J Thorac Cardiovasc Surg* 2006;131:530-2).

"The decision to perform an endovascular versus an open repair is based mainly on 2 factors: the patient's physiologic reserve and the location and complexity of the disease," says Dr. Coselli. "Endovascular repair requires less physiologic reserve than open repair because the endovascular approach is much less invasive and doesn't necessitate aortic clamping. On the other hand, an endovascular repair may not work if the diseased aortic segment is too complex anatomically. For example, a purely endovascular approach often works well in the descending thoracic aorta: because this segment has few branch vessels, a simple tube graft can be deployed inside it without blocking blood flow to important arteries. In contrast, the thoracoabdominal aorta has many branch vessels, so a tube graft placed there must have multiple fenestrations, branches, or both, to allow blood flow to these vessels. Positioning such a graft correctly is difficult via a purely endovascular approach, and the graft usually has to be custom-made for the patient. This can take weeks, so endovascular treatment of thoracoabdominal aortic disease remains rare."

Endovascular aortic repairs are also associated with a number of potential complications.



An endovascular stent in the descending portion of the thoracic aorta. A bypass from the left common carotid artery maintains blood flow to the left subclavian artery, the origin of which is covered by the stent. (Image created by Scott Weldon for Baylor College of Medicine. Used with permission.)

Some of these occur at the access site—usually the femoral artery—where the puncture wound could eventually cause pseudoaneurysms, thrombosis, and even arterial rupture.

Complications can also occur in the aorta itself. For example, if the seal becomes insufficient between the endograft and the aorta, an endoleak can develop, allowing blood to flow to the dissecting or aneurysmal portion of the aorta. Without additional intervention, such an endoleak can lead to potentially fatal rupture. Also, during endovascular repairs of the descending aorta, the endograft (especially one with bare springs) can tear a portion of the aortic arch, necessitating emergency surgical repair.

Ischemic complications are also possible with endovascular repair. Fragile thrombus and atheroma lining the aortic lumen can become dislodged during the procedure and can embolize to the brain, gastrointestinal tract, kidneys, or limbs. Covering the brachiocephalic or left subclavian artery with the stent-graft—which is commonly done to lengthen the landing zone for the device—can cause cerebral malperfusion that can lead to a stroke. Similarly, stent-graft occlusion of intercostal arteries can result in immediate or delayed paraplegia.

Although there are potential complications associated with endovascular aortic repair, their frequency and severity are comparable to or less than those associated with open surgery in patients whose aortic disease and anatomy are amenable to endovascular treatment. As a result, endovascular approaches to aortic disease are gaining in popularity.

"As the population ages, endovascular repair is likely to become the standard treatment for noncomplex disease in parts of the aorta that are well suited to endografting," says Dr. Coselli. "For other types of aortic disease, combined surgical and endovascular procedures are showing considerable promise as a treatment option for patients with a limited physiologic reserve." ●

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Contents

Inflammation Contributes to the Delicate Dance of Apoptosis in the Failing Heart

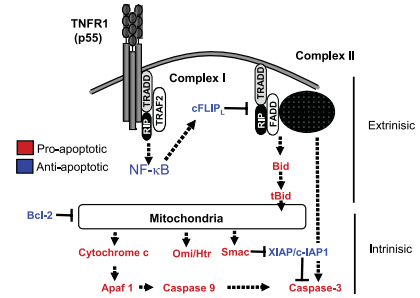
Abstract: Tumor necrosis factor activates multiple pathways leading to cardiomyocyte apoptosis and cardiac remodeling.

The programmed death, or apoptosis, of cardiomyocytes contributes to cardiac remodeling and progressive heart failure. Two independent pathways lead to apoptosis: (1) an extrinsic pathway initiated by the binding of ligands to death receptors on the cell surface and (2) an intrinsic pathway controlled by the intracellular release of death proteins from the mitochondria. A multitude of molecules, receptors, and cytokines interact to regulate the delicate balance of life and death in a cell (*see Figure*).

Contributing to the nascent understanding of apoptosis in heart disease, Douglas L. Mann, MD, chief of Cardiology at the Texas Heart Institute at St. Luke's Episcopal Hospital and director of the Winters Center for Heart Research at Baylor College of Medicine, has spent the last several years unraveling the complex role of an inflammatory mediator, tumor necrosis factor (TNF), in programmed cell death.

Induced in the failing heart, TNF activates both pro-life and pro-death (apoptosis) pathways in cardiomyocytes. In a mouse model characterized by cardiac-restricted overexpression of TNF, Dr. Mann's group showed that increased apoptosis of cardiomyocytes correlates with progressive thinning of the left ventricular wall, a classic component of adverse cardiac remodeling (*Am J Physiol Heart Circ Physiol* 2004;287: H1303-11). As heart failure progressed, cardiomyocyte apoptosis in TNF-overexpressed mice increased. There was also a decrease in Bcl-2, a protective, antiapoptotic protein that suppresses cell death.

"Although long heralded as the kiss of death, TNF may act in a more subtle, complex manner in eliciting apoptosis," says Dr. Mann. "During the transition to heart failure, cardiomyocytes become fragile and increasingly susceptible to death. Our results suggest that this fragility results from the progressive loss of cytoprotective proteins during sustained inflammation. Furthermore, our study shows that one consequence of the loss of cytoprotective proteins is the concomitant activation of multiple cell death pathways, which tips the delicate balance in favor of cell death."



Proapoptotic and antiapoptotic proteins that govern the extrinsic and intrinsic cell death pathways. The cytoprotective protein Bcl-2 prevents activation of the intrinsic (mitochondrial) cell death pathway, whereas the cytoprotective protein cFLIPL (flice inhibitory protein) prevents activation of complex II and, hence, activation of the extrinsic cell death pathway.

To delineate the role of cytoprotective mechanisms in cardiomyocyte apoptosis, Dr. Mann's group used a specific genetic approach—the development of bitransgenic mice with cardiac-restricted overexpression of Bcl-2 and TNF—to inhibit programmed cell death (*J Clin Invest* 2007;117:2692-701). Cardiac overexpression of Bcl-2 reduced TNF-induced apoptosis and prevented thinning of the ventricular wall. Furthermore, these researchers showed that Bcl-2 may help fend off cell death by blocking the release of proapoptotic proteins from the mitochondria, a key step in initiating the intrinsic pathway. However, Bcl-2 did not completely eliminate apoptosis of cardiomyocytes, and subsequent studies showed that the extrinsic apoptotic pathway, which is independent of Bcl-2, may be activated by sustained exposure to TNF.

The progressive loss of myocytes from programmed cell death contributes significantly to adverse cardiac remodeling. Because apoptosis is governed by an interplay of both positive and negative factors, myocardial recovery may occur if cells are given sufficient time for protective responses to prevail.

"Our findings attest to the complexity of cell death pathways of cardiomyocytes in heart disease," says Dr. Mann. "One specific molecule, such as TNF, does not drive apoptosis. Rather, long-term exposure to TNF, as seen in inflammatory responses to injury in the heart, may render myocytes susceptible to death by progressively depleting protective proteins that prevent activation of both the intrinsic and extrinsic pathways. As heart failure progresses, cells are precariously balanced between life and death, and the outcome depends on how the cell interprets the cocktail of extracellular signals. Understanding the integration of these signals will help us develop improved therapies for patients with sustained cardiac injury." ●

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Texas Heart Institute at St. Luke's Episcopal Hospital Is Ranked Among Nation's Top 10 Heart Centers

For the 17th consecutive year, *U.S. News & World Report*'s annual guide to "America's Best Hospitals" has ranked the Texas Heart Institute at St. Luke's Episcopal Hospital (THI at SLEH) among the top 10 heart centers in the United States. "Since its founding 45 years ago, our institution has maintained a strong commitment to excellence in research, education, and patient care. To receive this honor consistently since the inception of 'America's Best Hospitals' is extremely gratifying," says Denton A. Cooley, MD, founder, president, and surgeon-in-chief of THI, and chief of Cardiovascular Surgery at SLEH. The Texas Heart Institute at SLEH is the only heart center in the Southwest to be listed among the top 10 in its category by this survey.

A Mannose-Binding Lectin Gene Haplotype Predicts Myocardial Infarction After Coronary Artery Bypass Surgery

Abstract: A specific combination of polymorphisms, or "haplotype," in the mannose-binding lectin gene is a novel independent predictor of heart attack after coronary artery bypass surgery in white patients.

Complement, an integral part of our innate immunity, is an important mediator of myocardial injury. Another essential component of the innate immune system is mannose-binding lectin (MBL), which activates the lectin complement pathway by binding to carbohydrate groups on the surfaces of microorganisms and to innate molecules that mimic these groups. Although reduced serum levels of MBL may increase the risk of infection in children and immunocompromised individuals, recent evidence suggests that increased MBL levels may mediate cardiovascular injury (*Circulation* 2004;109:471-5).

Levels of MBL, an acute phase reactant, increase in response to injury, including surgery. However, the extent and severity of surgical inflammation may be governed by a patient's genetic makeup, or genotype. For example, common genetic variations, or polymorphisms, in the human MBL gene (*MBL2*) have been shown to regulate both MBL function and serum levels.

In collaboration with Harvard Medical School and the National Cancer Institute, researchers at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI at SLEH) conducted a prospective, longitudinal study of 978 white patients undergoing coronary artery bypass graft (CABG) surgery at Brigham & Women's Hospital and THI at SLEH (*Circulation* 2007;116(11 Suppl): I106-12). These researchers looked at all possible *MBL2* haplotypes (combinations of inherited *MBL2* polymorphisms) to determine whether certain haplotypes independently predict an increased risk of postoperative myocardial infarction.

The lead author of the study, Charles D. Collard, MD, professor, Baylor College of Medicine Division of Cardiovascular Anesthesiology and THI at SLEH, says, "Ours is one of the largest perioperative genomics studies to date. Thus, we have substantial statistical power to identify genes that predict adverse outcomes, such as myocardial infarction, after cardiac surgery."

The researchers found that white patients who had a particular *MLB2* haplotype had a significantly higher incidence of myocardial infarction after CABG surgery than did white patients without this genetic combination (38% vs. 10%, respectively; $P<0.007$). Moreover, this *MBL2* polymorphism was an independent predictor of postoperative myocardial infarction, even after the researchers adjusted for patient demographics, medications, and perioperative risk factors (adjusted odds ratio: 3.97; 95% confidence interval: 1.30–12.07). Although the mechanism that underlies this increased risk of infarction has not yet been established, this information may be useful in improving perioperative risk stratification.

> *"Ours is one of the largest perioperative genomics studies to date. Thus, we have substantial statistical power to identify genes that predict adverse outcomes, such as myocardial infarction, after cardiac surgery."*

"We have shown that patients with this polymorphism are at increased risk of heart attack after CABG surgery, but we cannot necessarily establish causality between this genotype and clinical outcome. Furthermore, we don't yet know whether this association applies to nonwhite patients or to other types of surgery," cautions Dr. Collard. "The fact that MBL has a dichotomous nature and can recognize both foreign and self molecules suggests that the clinical setting may determine whether MBL is protective or detrimental."

To further define the mechanism involved in this association between genotype and outcome, Dr. Collard plans to study perioperative MBL levels in patients with and without this high-risk polymorphism.

"We want to find out how this haplotype affects serum MBL levels and whether MBL levels predict a heart attack," says Dr. Collard. "Furthermore, we have developed an anti-MBL monoclonal antibody that we can use in preclinical models of myocardial infarction. We hope that these efforts will lead to the development of novel strategies for preventing complement-mediated myocardial injury in humans." ●

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Texas Heart Institute at St. Luke's Episcopal Hospital Participates in Collaborative Thoracic Aortic Research

The Baylor College of Medicine (BCM) division of Cardiothoracic Surgery, The University of Texas Medical Branch at Galveston, and The University of Texas Medical School at Houston (UTMSH) are collaborating to use an $11.6 million grant from the National Heart, Lung, and Blood Institute to study thoracic aortic disease. This 5-year, interinstitutional award was used to create the Specialized Center for Clinically Oriented Research in Thoracic Aortic Aneurysms and Dissections. Directed by Dr. Diana Milewicz (UTMSH) and codirected by Dr. Joseph Coselli (BCM and the Texas Heart Institute at St. Luke's Episcopal Hospital) and Dr. Hazim Safi (UTMSH), the research team will identify genetic determinants of and biomarkers for aortic dissections.

Stem Cells Fuse With Host Cardiomyocytes in Myogenesis

Abstract: The fusion of human CD34+ cells with resident mouse cardiomyocytes involves cell surface adhesion molecules and reprograms newly formed cells to divide.

Cell-based therapies have been developed to address the central pathophysiologic mechanism that underlies heart failure—the loss of functional cardiomyocytes. Adult stem cells can develop into a variety of cell types, including cardiomyocytes. However, the nature of this transformation is not well understood. In studies of different types of stem cells in various experimental systems, 2 mechanisms have emerged as front-runners: direct differentiation of donor stem cells into cardiomyocytes (transdifferentiation) and fusion of donor cells with host cardiomyocytes.

The fate of stem cells injected into the heart has been the focus of extensive research by Edward T.H. Yeh, MD, professor and chair of the department of Cardiology at The University of Texas (UT) M. D. Anderson Cancer Center and staff cardiologist at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI at SLEH), and by James T. Willerson, MD, president-elect and medical director of THI at SLEH and president of the UT Health Science Center at Houston. Using a model of myocardial infarction in severe combined immunodeficiency (SCID) mice, Drs. Yeh and Willerson's research team showed that both fusion and transdifferentiation account for the transformation of human peripheral blood CD34+ cells, which contain progenitor/stem cells, into cardiomyocytes in the injured heart (*Circulation* 2004;110:3803-7).

"Although transformation occurred via both mechanisms, most of the newly formed cardiomyocytes resulted from fusion between transplanted stem cells and host cardiomyocytes," says Dr. Willerson. "However, the mechanisms involved in cell fusion and the consequences of this transformation are poorly understood."

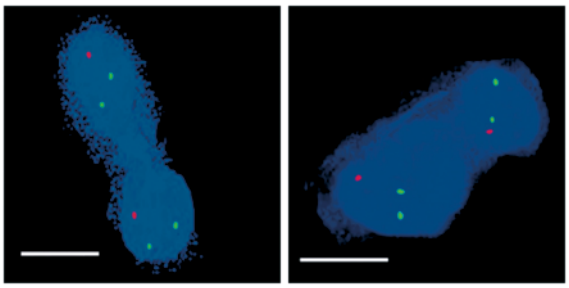
To examine the details of cell fusion, these researchers developed an in vitro model showing spontaneous fusion between human CD34+ cells and mouse cardiomyocytes (*Circ Res* 2007;100:693-702). The rate of fusion increased during periods of hypoxia and after treatment with proinflammatory cytokines; both of these conditions characterize the postinfarction milieu. Thus, local environmental factors seen after myocardial injury promote cell fusion.

Because fusion requires close contact between cells, the most likely mediators are cell adhesion molecules, which function in cell recruitment and trafficking during inflammation. The THI at SLEH researchers showed that pretreating cells with antibodies to vascular cell adhesion molecule-1 (VCAM-1) and α4β1 blocked the in vitro fusion of human progenitor cells with mouse cardiomyocytes. Taking their studies a step further, the group then assessed the effect of antibody treatment in vivo.

"Cell fusion was blocked in vivo in the injured mouse heart by antibodies to the α4β1/VCAM-1 adhesion molecule pair but not by antibodies to vascular endothelial growth factor. This finding indicates the specific biologic relevance of adhesion molecules in fusion," says Dr. Yeh. "However, defining the molecular aspects of cell fusion is not the whole picture. Most clinical studies of stem cell therapy show improvements in cardiac function. So the bigger question is, how does the fusion of stem cells with resident cardiomyocytes translate into clinical benefits in a patient with heart disease? In other words, what are the consequences of cell fusion?"

After fused cells are obtained from the hearts of stem cell–transplanted SCID mice, most newly formed cardiomyocytes express cyclin B, a cell cycle marker, and incorporate 5-bromodeoxyuridine, a label for actively dividing cells.

"Our results indicate that cell fusion promotes reentry of these new cardiomyocytes into the cell cycle, ultimately leading to cell proliferation. This amplification of fused cells may help repair damaged myocardium," says Dr. Willerson. "Thus, the clinical benefit of stem cell therapy may be attributed, at least in part, to myogenesis resulting from the fusion of stem cells with host cardiomyocytes." ●



Nuclear division in a fused nucleus that contains both human (red) and mouse X (green) chromosomes. This photograph clearly shows that both cell fusion and cell division are important mechanisms in the generation of new heart muscle. (From *Circulation Res* 2007; 100:693-702; with permission.)

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Transvenous Left Ventricular Pacemaker Lead Placement Can Be Successful in Patients With Tricuspid Valve Prostheses

Abstract: Patients with tricuspid valve prostheses who need pacemaker therapy may now be able to avoid open surgery for pacemaker lead insertion.

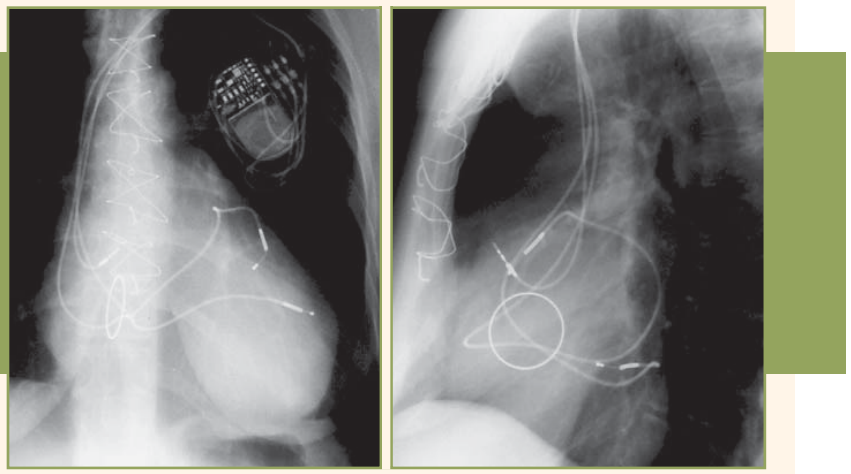
Traditionally, placement of ventricular pacemaker leads involved direct epicardial implantation via a thoracotomy incision with the aid of general anesthesia. Beginning in the mid 1960s, however, advances in lead technology allowed endocardial pacing leads to be inserted transvenously. This safer, less invasive approach quickly became the preferred method for lead implantation.

Nevertheless, transvenous lead insertion is contraindicated for some patients, particularly those who have undergone previous tricuspid valve replacement. In such patients, inserting an endocardial lead into the right ventricle across a tilting-disc prosthesis or a bioprosthesis may lead to acute valve failure.

"The need for ventricular pacing is fairly common after tricuspid valve replacement," says J. Alberto Lopez, MD, a cardiologist at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI at SLEH), who has a special interest in electrophysiologic disorders of the heart. "For instance, permanent pacing is often needed for patients with Ebstein's anomaly, a congenital malformation of the tricuspid valve. Most of these patients have undergone tricuspid valve repair or replacement."

Dr. Lopez and his colleague D. Richard Leachman, MD, recently treated a 62-year-old woman with Ebstein's anomaly and a tricuspid valve bioprosthesis who needed a permanent atrioventricular pacemaker because of highly symptomatic sinus node dysfunction and atrioventricular block.

"To avoid inducing further right ventricular dyssynchrony," says Dr. Lopez, "we placed 1 transvenous bipolar lead in the patient's anterior cardiac vein. This allowed us to place the lead as close to the right ventricular myocardium as possible from the coronary sinus. The lead stimulated the basal interventricular septum and outflow tract early and improved right atrial and right ventricular timing. The second bipolar lead was placed in the posterolateral coronary vein to preserve left intraventricular synchrony and to increase safety in case of anterior lead dislodgement and loss of atrial capture."



Chest roentgenogram in the posteroanterior and lateral projections shows the atrial lead at the interatrial septum (left) and 2 leads in the coronary sinus with bipolar electrodes in the anterior cardiac vein and a posterolateral branch (right). (From *Ann Thorac Surg* 2007;83:1183-5; with permission.)

In addition, an atrial septal lead was placed to control atrial pacing. Tissue Doppler imaging and the Doppler-derived stroke volume were used to adjust interventricular and atrioventricular timing.

This approach preserved prosthetic valve function, offered back-up ventricular pacing in case of anterior lead failure, and maintained atrioventricular and interventricular synchrony. A report of this case recently appeared in the *Annals of Thoracic Surgery* (2007;83:1183-5).

In a related case, Dr. Lopez and another colleague, Roberto Lufschanowski, MD, implanted a permanent pacemaker in a 56-year-old woman with bradycardia related to atrioventricular block. Fifteen years earlier, the patient had undergone a tricuspid valve replacement for rheumatic heart disease. As in the previous case, Drs. Lopez and Lufschanowski inserted 1 lead into the anterior cardiac vein and the other lead into the posterolateral coronary vein.

Dr. Lopez believes that this is the first case in which multiple left ventricular leads have been inserted through the coronary sinus tributaries to prevent pacemaker-induced dyssynchrony and, possibly, to optimize the stroke volume and resulting cardiac output.

"This method allowed us to avoid interventricular delay and to preserve left ventricular synchrony in a patient with symptomatic high-grade atrioventricular block," he comments. "In addition to having a safety benefit, the redundant ventricular pacing may reduce pacemaker-induced ventricular remodeling and its related hemodynamic complications."

By using an innovative approach in these 2 cases, Dr. Lopez and his colleagues spared the patients an open surgical procedure, with its heightened morbidity and mortality. "At THI at SLEH," Dr. Lopez concludes, "we are constantly seeking new ways to benefit not only our own patients but also patients elsewhere." ●

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Cover: Open Heart Era, 1962; detail of oil painting by Mary Cooley Craddock, commissioned by Denton A. Cooley, MD, for the Fifth Floor Lobby of the Texas Heart Institute at St. Luke's Episcopal Hospital —The Denton A. Cooley Building.

Calendar of Events

Texas Heart Institute Continuing Medical Education Symposia

Denton A. Cooley Cardiovascular Surgical Society 15th International Symposium
October 25–27, 2007 • Houston, Texas
Program Director: James Livesay, MD
For more information, visit www.cooleysociety.com

Cardiac Arrhythmia Symposium
February 16, 2008 • Houston, Texas
Program Director: Ali Massumi, MD

Adult Congenital Heart Disease Symposium
March 1, 2008 • Houston, Texas
Program Director: Wayne Franklin, MD

Eighth Texas Update in Cardiovascular Advancements
July 25–26, 2008 • Houston, Texas
Program Director: James T. Willerson, MD

Selected Upcoming National and International Meetings

American College of Surgeons 93rd Annual Clinical Congress
October 7–11, 2007 • New Orleans, Louisiana

American Heart Association Scientific Sessions 2007
November 4–7, 2007 • Orlando, Florida

Society of Thoracic Surgeons 44th Annual Meeting
January 28–30, 2008 • Fort Lauderdale, Florida

American College of Cardiology 57th Annual Scientific Sessions
March 28–April 1, 2008 • Chicago, Illinois

International Society for Heart and Lung Transplantation 28th Annual Meeting and Scientific Sessions
April 9–12, 2008 • Boston, Massachusetts

American Surgical Association 128th Annual Meeting
April 24–26, 2008 • New York, New York
Abstract submission ends: November 30, 2007

For information about the Texas Heart Institute CME activities listed above, please e-mail cme@heart.thi.tmc.edu or call 832.355.2157. To view selected CME presentations and other physician resources online, visit cme.texasheart.org.

For 17 consecutive years, the Texas Heart Institute at St. Luke's Episcopal Hospital has been ranked among the top 10 heart centers in the United States by *U.S. News & World Report*'s annual guide to "America's Best Hospitals."

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