HeartWATCH

SUMMER 2003

A NEWSLETTER PRODUCED BY THE TEXAS HEART INSTITUTE

TEXAS HEART® INSTITUTE
at St. Luke's Episcopal Hospital

Surgical Ventricular Restoration Provides Innovative Treatment of Congestive Heart Failure

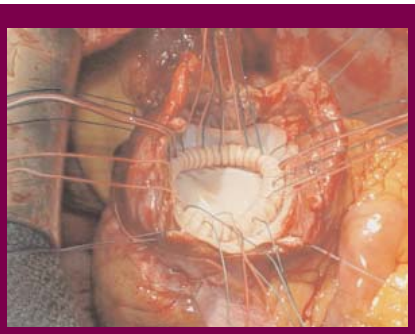
Abstract: After a myocardial infarction, deformation of the left ventricle can cause congestive heart failure, but a new surgical technique can restore normal function in some patients.

When a myocardial infarction (MI) damages an extensive area of the myocardium, the heart attempts to compensate by dilating the left ventricle, thinning and expanding the infarcted tissue, and enlarging the unaffected areas. These changes initially maintain the heart's normal stroke volume, but the extra workload and additional wall pressure continually exaggerate the deformation over time, often leading to congestive heart failure (CHF). Although medical therapy and cardiac assist devices can alleviate the symptoms of CHF, transplantation offers the only cure. However, the scarcity of donor hearts and the dangers of prolonged immunosuppressive therapy severely limit the number of transplants that can be performed. Therefore, researchers have attempted to develop alternative surgical treatments for CHF.

In the mid-1990s, Brazilian surgeon Randas Batista, M.D., proposed that excising a flap of myocardium could relieve the symptoms of CHF by returning the left ventricle to its normal size and shape, thereby restoring some of the heart's pumping capacity. This procedure, known as the Batista procedure or partial left ventriculectomy, has been performed in more than 1000 cases worldwide. Unfortunately, failure rates are high, ranging from 42% at 1 year to 74% at 3 years (*J Am Coll Cardiol* 2000;36:2098–2103).

More recently, an alternative procedure called ventricular restoration has evolved from techniques developed by Adib Jatene, M.D., at the University of São Paulo, Vincent Dor, M.D., at the Cardiothoracic Center of Monaco, and Denton A. Cooley, M.D., here at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI/SLEH). Like the Batista procedure, ventricular restoration is aimed at restoring a normal ventricular size and shape, but it does so by removing non-contractile myocardium rather than functioning muscle. The procedure can be completed with direct closure or with a patch such as the CorRestore™ device (Somanetics Corp., Troy, MI), which is made primarily of preserved bovine pericardium.

"Nearly 20% of patients with CHF will experience progressive dilatation of the left ventricle within 3 years of their initial MI," states Igor Gregoric, M.D., associate chief of Cardiopulmonary Transplantation at THI/SLEH. "For some of these patients, ventricular restoration will be more effective than the Batista procedure because the Batista method leaves behind the non-contractile segments of the myocardium, while ventricular restoration excludes mostly akinetic or dyskinetic tissue and restores the rest of the myocardium to a more physiologic shape."



A non-contractile segment of myocardium is replaced with a CorRestore™ patch, restoring the left ventricle to its original shape.

The ventricular restoration procedure has produced excellent results, adds Dr. Gregoric, citing a 3-year, 13-center study of 662 patients (*Semin Thorac Cardiovasc Surg* 2001; 13:448-458), of whom 89.4% survived for at least 3 years after the procedure. Furthermore, 88.7% of the survivors required no readmission for heart failure in the first 3 years.

Encouraged by these results, THI recently began a collaborative effort with Somanetics Corp. to train cardiothoracic surgeons in ventricular restoration with the CorRestore patch. In October 2002, THI hosted its first training session, which involved approximately 80 surgeons.

"Because nearly 5 million people in the United States have CHF, it's part of our mission to study, teach, and provide innovative treatments and technologies for this debilitating disease," says Ross M. Reul, M.D., director of Surgical Innovation at THI. "By becoming a center of excellence for teaching this type of intervention, we are renewing our commitment to the research and development of new cardiovascular surgery techniques." ●

For more information:

Dr. Igor Gregoric
832.355.3000
Dr. Ross M. Reul
832.355.5884

Clinical Trials Update

AbioCor On May 1, 2003, THI surgeons performed the 11th implantation worldwide of the AbioCor Implantable Replacement Heart (ABIOMED, Inc., Danvers, MA) in an ongoing FDA-sponsored multicenter clinical trial. The AbioCor has supported previous patients for an average of 6 months (range, 50–512 days).

Jarvik 2000 Two patients—a man and a woman—on the transplant waiting list at THI/SLEH were recently discharged home after receiving the Jarvik 2000 axial-flow left ventricular assist device. These patients are the first to be sent home from here with the device. Both are part of an ongoing clinical trial of the pump as a temporary bridge to transplantation. The trial, which began in April 2000, now includes 56 patients at several U.S. and European centers (31 patients at THI/SLEH).

Emerging Evidence Links Influenza and Coronary Artery Disease

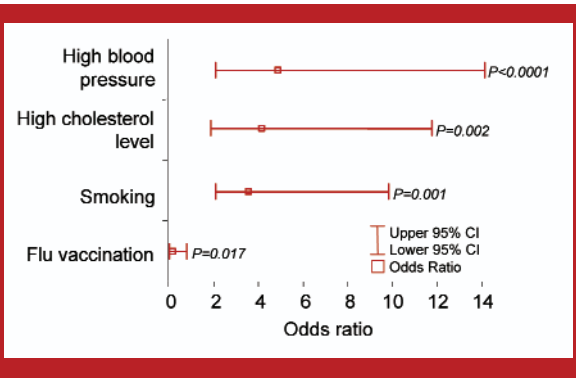
Abstract: In elderly patients and those with heart disease, researchers are finding increasing evidence that influenza vaccination may reduce the risk of hospitalization for heart disease and stroke.

Medical breakthroughs often rely on the discovery of a link between seemingly unrelated processes or conditions. Such a breakthrough is currently gaining momentum with regard to influenza and coronary artery disease (CAD). Although CAD has traditionally been regarded as a degenerative disease, infection and inflammation appear to play an important role in atherogenesis. In fact, some researchers believe that acute respiratory infections may increase the risk of a myocardial infarction. This theory was recently strengthened by a large observational study involving more than 386,000 subjects aged 65 or older (*N Engl J Med* 2003;348:1322–1332). Those who had received a flu shot had fewer hospitalizations for heart disease and stroke and a lower all-cause mortality during influenza seasons.

These findings came as no surprise to S. Ward Casscells, M.D., and Mohammad Madjid, M.D., who have been pioneering similar research at the Texas Heart Institute (THI). Dr. Casscells is associate director of Basic Cardiology Research, and Dr. Madjid is a research associate at THI. "After seeing numerous patients in whom an upper respiratory infection was soon followed by a heart attack," says Dr. Casscells, "we found that this pattern is seen in up to 35% of cases described in the literature." With Morteza Naghavi, M.D. (co-director of THI's Center for Vulnerable Plaque Research), Drs. Casscells and Madjid performed a case-control study of 218 patients with CAD (*Circulation* 2000;102:3039–3045). "Influenza vaccination was associated with a 67% reduction in myocardial infarctions during the same flu season," says Dr. Madjid.

Flu patterns may account for the high incidence of myocardial infarctions in the winter months. If one allows for a lag time of about 2 weeks, CAD-related deaths tend to parallel deaths related to flu and pneumonia (*J Infect Dis* 1982;146:313–321).

"The flu virus causes the release of cytokines and endogenous catecholamines, platelet aggregation, endothelial dysfunction, altered plasma viscosity, and tachycardia," explains Dr. Madjid. "More important, by weakening inflamed (vulnerable) plaque, the flu can cause thrombosis, followed by a myocardial infarction."



Odds ratios for various risk factors for myocardial infarction. (Adapted from *Circulation* 2000;102:3039–3045.)

The emerging field of influenza and CAD is attracting cardiovascular specialists, infectious disease experts, virologists, epidemiologists, and public health personnel. On April 26, 2003, leading representatives of these disciplines convened at THI for the First Symposium on Influenza and Cardiovascular Disease: Science, Practice, and Policy, co-chaired by Drs. Casscells and Madjid. This event presented the most recent evidence concerning the apparent link between influenza and CAD and sought new methods for encouraging high-risk patients to be vaccinated.

"Although flu shots are advisable for everyone aged 50 or older, only 66% of Americans older than 65 received a flu shot in 2001, and only 27% of patients with heart disease received one," says Dr. Madjid. "Unfortunately, physicians do not always promote the flu shot and may not even recognize all of its benefits."

According to Dr. Casscells, "Vaccination probably reduces the risk of heart attack, stroke, or sudden death by 20% or 25%. It might save 36,000 to 91,000 Americans from CAD-related death in addition to those saved from other flu-related causes of mortality. Looking at it another way, statin therapy, which saves many lives, costs about $1000 per patient annually. By spending an additional $12 per patient per year on vaccination, even more lives might be saved."

The suspected link between influenza and CAD has not been unequivocally proven, so prospective, randomized trials are needed.

"Meanwhile," says Dr. Madjid, "because flu shots are safe, effective, inexpensive, and recommended anyway, the goal is to increase their use in high-risk patients, particularly elderly persons and those with heart disease." ●

For more information:

Dr. Mohammad Madjid
713.500.6373
Dr. S. Ward Casscells
713.500.6545

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Researchers Continue to Clarify Myocardial Cell Survival in the Failing Heart

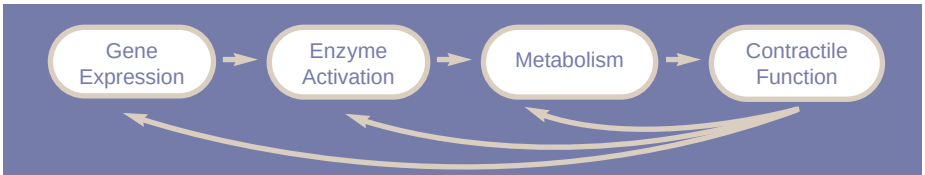
Abstract: Examination of myocardial cell survival in the failing heart may soon reveal useful metabolic and genetic targets for heart failure therapy.

The heart has a ferocious appetite for energy. To maintain its ability to pump oxygenated blood throughout the body, the heart uses oxygen and the most efficient metabolic substrates in the coronary circulation as fuel. The normal heart metabolizes mainly fatty acids. The stressed heart, however, reverts to a fetal metabolic pattern in which glucose and glycogens become the main energy source. The more available glycogens are, the more tolerant of stress (e.g., ischemia) the heart becomes while trying to protect itself against programmed cell death.

"Over the last few decades, this metabolic picture has been confirmed by laboratory, animal, and clinical studies showing that the myocardial metabolism maintains a delicate balance between contractile function and cardiac gene expression," says Heinrich Taegtmeyer, M.D., D.Phil., a research scientist at the Texas Heart Institute (THI) and co-director of the Division of Cardiology at The University of Texas Medical School at Houston. "However, this balance can easily be thrown off by stresses such as the ischemia that occurs after cardiopulmonary bypass or during acute coronary syndromes."

Ischemia hinders normal cardiac metabolism by starving the myocardium of oxygen, thus slowing aerobic metabolism and accelerating anaerobic metabolism. The myocardial cells respond by reactivating a fetal program of cardiac gene expression and entering an energy-sparing metabolic state that favors maintenance of viability over efficient contraction. The end result is increased glucose oxidation and glycogen resynthesis, which promote a return to normal contractile function.

This survival response, says Dr. Taegtmeyer, can be seen at work in stunned myocardium (e.g., in cases of reperfusion after hypothermic ischemic arrest or revascularization of an occluded coronary artery), hibernating myocardium (e.g., in cases of ischemic cardiomyopathy), and ischemically preconditioned myocardium (e.g., in cases of unstable angina in which short periods of ischemia appear to protect against the damage otherwise caused later by longer periods of ischemia).



Proposed interactive links between contractile function, gene expression, enzyme activation, and metabolism in the myocardium.

Armed with this knowledge, researchers are continuing to unravel the many biochemical and physiologic pathways involved in myocardial cell survival and to seek clinical correlates. For instance, since the late 1980s, researchers at THI/SLEH have periodically studied the outcomes of cardiac surgery patients who receive postoperative metabolic support with a solution of glucose, insulin, and potassium (GIK).

"The rationale for using GIK is clear, especially when systemic and myocardial energy reserves are so depleted that the function of all the vital organs is threatened," says Dr. Taegtmeyer. "Insulin restores ion gradients, stimulates potassium reuptake, and promotes glucose uptake for energy production."

Nevertheless, he notes, there is understandable reluctance to use GIK in patients suffering from stunning of the heart after cardiopulmonary bypass. Some clinicians worry that hyperglycemia and hypokalemia will impair organ function or that the presence of excess lactate may cause acidosis and inhibit energy production. However, Dr. Taegtmeyer says that adverse effects have not been seen on follow-up examination of GIK recipients. Instead, GIK significantly reduces the morbidity and mortality attributable to cardiogenic shock after hypothermic ischemic arrest for coronary artery bypass grafting.

Myocardial cell survival programs also appear to play a role in other types of heart failure. For example, in response to recent reports that contractility of the hypertrophic human heart improves after mechanical ventricular unloading, Dr. Taegtmeyer and coworkers asked whether this improvement might be due to metabolic reactivation of the fetal cardiac gene program.

"Interestingly," he says, "we found that both pathologic overloading (i.e., hypertrophy) and mechanical unloading push the myocardium into an adaptive pattern of fetal gene expression."

By uncovering the metabolic underpinnings of heart failure, Dr. Taegtmeyer's group and other researchers may soon be able to identify useful metabolic and genetic targets for treating the stressed and failing heart. ●

For more information:

Dr. Heinrich Taegtmeyer

713.500.6569

Transplant Recipient Marks 20th Anniversary

On May 1, 2003, one of the longest-surviving heart transplant recipients in the world returned to THI/SLEH for an annual follow-up visit. The visit marked the 20th anniversary of 67-year-old Charles Washington's transplant. Under the care of cardiologist Edward K. Massin, M.D., and transplant surgeon O.H. Frazier, M.D., Mr. Washington was able to return to work within 6 months of surgery and has never had a rejection episode or transplant coronary artery disease.

Contrast-enhanced Magnetic Resonance Imaging May Help Clinicians Recognize Hibernating Myocardium

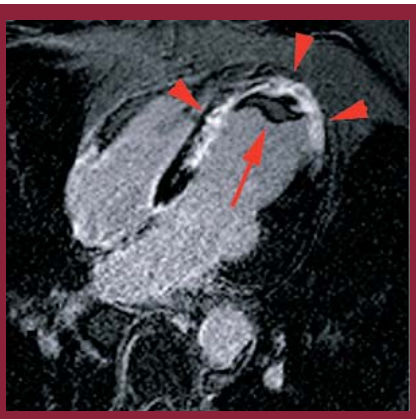
Abstract: An international study may confirm the utility of contrast-enhanced magnetic resonance imaging for identifying viable ischemic myocardium that is likely to recover function after revascularization.

Led by researchers at the Texas Heart Institute (THI) at St. Luke's Episcopal Hospital (SLEH), 15 experienced cardiac magnetic resonance imaging (MRI) centers will participate in a 200-patient clinical trial to determine whether contrast-enhanced MRI can predict which dysfunctional regions of the left ventricular myocardium will recover after coronary angioplasty (with or without stent placement) or coronary artery bypass grafting.

In delayed enhancement MRI (DE-MRI), patients receive an intravenous injection of gadolinium before undergoing an inversion-recovery, T1-weighted, gradient-echo imaging sequence. DE-MRI allows clinicians to distinguish between hibernating (potentially recoverable) ischemic heart tissue and scarred or irreversibly damaged tissue. Unlike scarred myocardium, hibernating myocardium can regain healthy function after revascularization. Positron-emission tomography (PET), single-photon-emission computed tomography (SPECT), and dobutamine echocardiography are currently used to assess the presence of viable myocardium, but these methods all have limitations and can produce artifacts that reduce diagnostic accuracy.

"Differentiating reversible from nonreversible myocardial injury is critical before patients undergo a revascularization procedure; it helps predict not only improved left ventricular function but also survival," says Scott D. Flamm, M.D., director of Magnetic Resonance Imaging and Cardiovascular Magnetic Resonance Imaging Research at THI/SLEH and the study's principal investigator. "Noninvasive methods for assessing myocardial viability frequently require radiation exposure, pharmacologic stressors, or both. DE-MRI requires no radiation and can be performed while the patient is at rest."

According to Dr. Flamm, DE-MRI also improves spatial resolution, so clinicians can better determine the transmural extent of irreversible myocardial injury. In contrast, the lower spatial resolution of conventional radionuclide techniques frequently reduces the interpretation of viability to a binary response—the "all-or-none phenomenon."



Delayed enhanced magnetic resonance imaging of left ventricle, showing a large myocardial infarction of the left anterior descending coronary artery distribution (arrowheads) and a thrombus in the left ventricular cavity (arrow).

The proposed trial will include patients with chronic ischemic myocardial damage and impaired wall motion (as documented by echocardiography, left ventriculography, or MRI) who are scheduled for a revascularization procedure within 30 days of the baseline MRI. Patients will undergo DE-MRI and cine wall-motion imaging immediately before revascularization and 4 months afterwards to confirm the functional recovery of viable myocardium. Patients will serve as their own internal controls, thus providing the most clinically relevant end points of functional recovery after intervention. The trial is expected to last 12 months.

"A strong set of investigators from the United States, Europe, South Korea, and Thailand has come together for this important trial," says Dr. Flamm. "It is also the second multicenter trial this group has performed—the first resulted in a *New England Journal of Medicine* report in 2001 about our experience with coronary MR angiography in ischemic heart disease. We hope this new study will have a similar impact."

Recent small studies have shown a close correlation between areas of enhancement on DE-MRI and chronically ischemic, irreversibly damaged myocardium, establishing that DE-MRI is comparable to the current gold standard, PET. Dr. Flamm hopes this larger study will confirm DE-MRI's utility in determining myocardial viability and will further validate the technique.

"This is a novel, exciting way to image the heart," says Dr. Flamm. "It can provide so much information about cardiac morphology and function—and now myocardial viability—all in a short period, without radiation or stress agents. We believe DE-MRI will likely become the new gold standard for determining myocardial viability. This will mean improved diagnosis and prognosis, a faster diagnostic workup, and, inevitably, better care for patients with heart disease." ●

For more information:

Dr. Scott D. Flamm

832.355.4201

Carotid Endarterectomy at THI/SLEH

Carotid endarterectomy (CEA) to remove occlusive plaque from the carotid artery can relieve severe stenosis. However, because stroke-causing emboli may be released during CEA, some clinicians advocate carotid angioplasty plus stenting instead. Recently, THI/SLEH researchers addressed the safety of CEA by analyzing the in-hospital incidence of stroke and death in a series of 1614 CEAs performed here between 1993 and 2002. Only 21 patients (1.3%) had a postoperative stroke; only 6 patients (0.37%) died, including 2 in the stroke group.

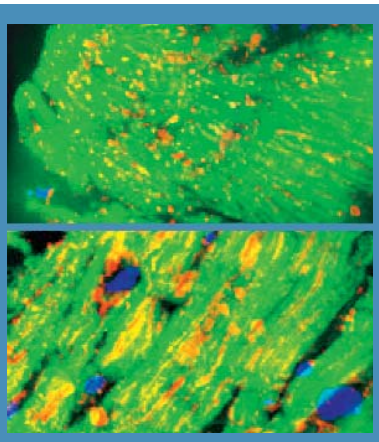
Unloading with a Left Ventricular Assist Device May Allow Adrenergic Receptor Recovery

Abstract: In patients with chronic heart failure, unloading the heart with a left ventricular assist device may give myocardial β-adrenergic receptors an opportunity to recover.

Chronic heart failure can begin with a variety of insults to the myocardial tissue, resulting in ischemia that progressively worsens over time. In the early stages of the disease, ventricular contractility can be maintained by increased adrenergic stimulation and other compensatory mechanisms. With time, however, these mechanisms become less and less effective as myocardial cells deteriorate and β-adrenergic receptors become desensitized or disappear. Once the circulation becomes inadequate and symptoms begin to occur, physicians may prescribe inotropic medications, but these agents also decrease in effectiveness as the loss or desensitization of β-adrenergic receptors continues. Eventually, it may become necessary to implant a left ventricular assist device (LVAD) to augment the heart's outflow.

The LVAD's immediate survival benefits have been documented for more than 30 years. However, a recent collaborative study by researchers at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI/SLEH) and The University of Texas Health Science Center at Houston (UTHSCH) has revealed that LVAD use may have another important effect: giving β-adrenergic receptors in the myocardium an opportunity to recover.

Alina Grigore, M.D., director of Cardiovascular Anesthesia Echocardiography at THI/SLEH and assistant professor of Anesthesiology at UTHSCH, together with Roger Bick, Ph.D., associate professor of Pathology and Laboratory Medicine at UTHSCH, examined samples of myocardium taken from the apex of the left ventricle at the time of LVAD implantation and compared them with samples taken later at LVAD explantation, heart transplantation, or autopsy. Using fluorescence deconvolution microscopy, the researchers treated the myocardial samples with fluorescent prazosin and isoproterenol analogues that bind with α-adrenergic and β-adrenergic receptors, respectively. Used in combination with computerized microscopy, this labeling method makes it possible to examine the number and distribution of both receptor types in the myocardial tissue.



Fluorescent microscopic images of myocardial samples taken before (top) and after (bottom) LVAD implantation showing a dramatic increase in β-adrenergic receptor density with LVAD unloading.

"With LVAD unloading of the left ventricle, there was an increase in β-adrenergic receptor density and an improvement in receptor distribution," says Dr. Grigore.

Before LVAD implantation, she reports, the myocardia of 13 participants with chronic heart failure contained fewer β-adrenergic receptors than did the healthy myocardia of control subjects, and the receptors tended to clump together abnormally. After 1 to 15 months of LVAD support, however, receptor density increased substantially, nearly doubling in some cases. Furthermore, the receptors were more evenly distributed throughout the myocardium. These findings suggest that the innate compensatory mechanisms that gradually collapse as chronic heart failure progresses may become useful again after a period of ventricular unloading with an LVAD.

The investigators also found a small but significant change in the number and distribution of α-adrenergic receptors. Although the clinical significance of this change was not clear, fluorescent microscopic images of both types of receptors showed that the myocardial tissue itself may have benefited from unloading with an LVAD.

"The change in distribution suggests that the myocytes themselves had recovered somewhat," Dr. Grigore states. "In the samples taken before LVAD implantation, the myofibrils looked disorganized and damaged, with a shortened, hypercontracted appearance. But after LVAD unloading, the myofibrils were longer and more parallel; they looked healthier."

These findings underscore the value of LVAD implantation in giving the heart time to rest and regain some of its functional capacity, thereby helping patients resume a normal lifestyle and reducing the need for cardiac transplantation. The results also suggest that fluorescence microscopy could become a useful tool for predicting the clinical outcome after LVAD implantation. ●

For more information:

Dr. Alina Grigore

832.355.2666

FDA Approves Sirolimus-Coated Stents

On April 24, 2003, the FDA approved the use of sirolimus-coated stents (Cypher; Cordis Corp., Miami Lakes, FL) to reduce restenosis after angioplasty. This approval was based mainly on the SIRIUS trial, which compared coated and uncoated stents in roughly 1100 patients at THI and other U.S. institutions. Nine months after receiving the drug-coated stents, patients had less restenosis, less need for repeat stenting, and fewer adverse outcomes (repeat angioplasty, coronary artery bypass surgery, heart attack, and death). Though more costly than bare stents, sirolimus-coated stents should be more beneficial.

Design Changes Have Improved the Reliability of the HeartMate Pulsatile Blood Pump

Abstract: Changing the design of the HeartMate pulsatile blood pump has improved outcomes for patients who receive this device as a treatment for severe, chronic heart failure.

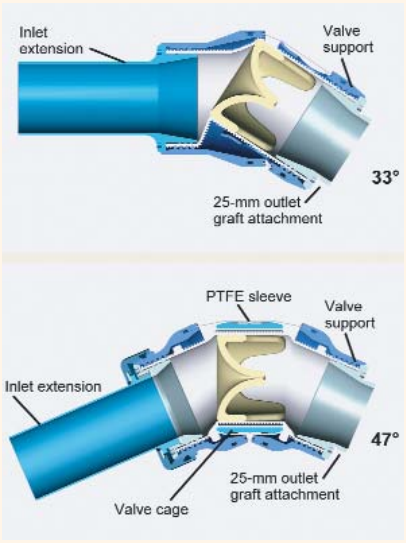
The first implantation of the electrically powered HeartMate pulsatile blood pump occurred at the Texas Heart Institute at St. Luke's Episcopal Hospital (THI/SLEH) in 1991. The patient lived for 505 days after implantation and was the first to be discharged from the hospital with such a device. A large man with type O blood, he would probably have had to wait 600 days for a transplant (40% of patients die while awaiting a transplant). Although he died of a stroke on day 505 after abruptly discontinuing warfarin anticoagulation, examination of his pump revealed pristine internal components (lining and valves) and no blood clots. This and other successes encouraged additional transplant centers to use the HeartMate as a bridge to transplantation.

In 1995, a design change to the HeartMate by its maker (Thoratec Corporation, Pleasanton, CA), which was intended to facilitate placement, altered directional outflow from the pump.

"Within a year, we began to see inflow valve disruptions not present in the earlier model," says O. H. Frazier, M.D., director of Cardiovascular Surgical Research and Cardiopulmonary Transplantation at THI/SLEH.

The changed model was used in the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial, which compared maximal medical therapy in terminally ill heart failure patients ineligible for a transplant to HeartMate use as long-term (2-year) therapy. Although the final results of the REMATCH trial showed that the HeartMate offered a significant survival benefit over maximal medical therapy, there were a number of pump-related complications, especially inflow valve incompetence and aortic valve insufficiency.

"Some of the patients in the trial died; others needed to have their pumps replaced," says Dr. Frazier. "The increased outflow resistance related to the design change caused high pressures (up to 600 mm Hg) inside the pump, which resulted in inflow valve disruption and pump-dysfunction endocarditis, leading to the deaths of at least 12 patients."



Old (top) and new (bottom) inflow valve assemblies of the HeartMate blood pump. To reduce conduit deformation, articulation (bend and twist) has been redistributed by the split angle, and the valve has been completely contained within a protective cage. Extra support is given to the valve through the use of a tubular polytetrafluoroethylene (PTFE) sleeve.

In the REMATCH trial, complications increased such that investigators looked to the 1995 design change as a probable cause.

Dr. Frazier explains how the problems were corrected. "To avoid increasing outflow pressures, the proximal portion of the outflow graft was reinforced to prevent kinking and to protect the graft at its entrance into the thoracic cavity below the relatively immovable sternum."

"Also," he says, "we realized that the closed inlet valve in the HeartMate is subject to a high systolic pressure that a native aortic valve never faces, and that sustained closure of the natural aortic valve was causing commissural fusion, leaflet distortion, and, ultimately, central aortic valve insufficiency."

This, in turn, caused circular flow to develop within the pump, maximizing pump flow and accelerating pump failure. To overcome this problem, pump flow is now periodically reduced to allow the aortic valve to open naturally. At THI/SLEH, this is done at night while patients are sleeping. Engineers have also changed the pump's computer programming to an "opti-fill" system: if high outlet pressures develop, the ejected volume is automatically reduced.

Infection has always been the main complication of HeartMate implantation. Investigators determined that this problem was related to the pump's placement in the abdominal wall, where pump motion, blood collecting around the pocket, and contamination from the long surgical procedure increased the likelihood of infection. To combat this problem, the pump is now placed intraabdominally in an antibiotic-impregnated sheath, which separates the pump from the abdominal contents and minimizes infection. Dr. Frazier recently removed a pump placed in this manner; 2 years after implantation, it was perfectly clean, with no evidence of infection.

"Addressing the problems of inflow valve disruption, aortic insufficiency, and infection has significantly improved the outcomes for patients who receive these pumps as destination therapy," says Dr. Frazier. ●

For more information:

Dr. O. H. Frazier

832.355.3000

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Editorial Office 832.355.6630
jrichard@heart.thi.tmc.edu

For physician referrals,
call 1.800.872.9355

Cover: Artwork donated by Ann Criswell and Fran Fauntleroy for the Celebration of Hearts display in the Wallace D. Wilson Museum 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 Symposium

Texas Heart Institute Congestive Heart Failure Summit

November 6, 2003
Houston, Texas
Program Director: James T. Willerson, M.D.

For information about the CME activity listed above, please contact cme@heart.thi.tmc.edu or call 832.355.2157.

Selected Upcoming National and International Meetings

American Heart Association Scientific Sessions 2003

November 9–12, 2003
Orlando, Florida

Society of Thoracic Surgeons 40th Annual Meeting

January 26–28, 2004
San Antonio, Texas
Abstract submission deadline: August 18, 2003

American College of Cardiology 53rd Annual Scientific Session

March 7–10, 2004
New Orleans, Louisiana
Abstract submission begins: August 1, 2003
Abstract submission deadline: September 16, 2003

International Society for Heart and Lung Transplantation 24th Annual Meeting and Scientific Sessions

April 21–24, 2004
San Francisco, California

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