



New England Heart Institute Atrium

SUMMER 2008

A New Era of Medicine

Message from the Medical Director

Tremendous advances have been made in the last two decades in improving outcomes in patients with coronary artery disease. The pathophysiology of coronary artery disease is far better understood and effective preventive therapies have been identified. These include dietary optimization and exercise, although optimizing people's behaviors in areas such as obesity, smoking and exercise remains challenging. Today's medical therapy is also vastly superior for treating hypertension, dyslipidemia and diabetes. The result of these advancements in prevention and therapy of cardiovascular disease is reduced mortality from coronary artery disease.

Despite these advancements, however, we continue to see patients with significant heart disease. The incidence of patients with heart failure from systolic or diastolic dysfunction has increased and now occupies a significant position in the disease processes treated. Sudden cardiac death also continues to be a major health problem, with difficulty in prospectively delineating the vast population of people who experience sudden cardiac death. Many patients who could benefit from interventional therapies, such as an implantable cardioverter defibrillator, are not being referred for treatment. Articles in this issue of Atrium discuss these and other very important topics.

To discuss any of the prevention and treatment modalities offered by the New England Heart Institute, please call me at 603.663.6782. ♥

Louis I. Fink, MD, FACC, Medical Director

Despite dramatic advances in the primary prevention of heart disease, sudden cardiac arrest remains the most common cause of death in the U.S., claiming 450,000 lives a year. In fact, the annual mortality from stroke, lung cancer, breast cancer and HIV disease all combined remains lower than the sudden death rate from cardiac arrest. The dismal prognosis of malignant arrhythmias drives these sobering numbers. Americans suffering an out-of-hospital cardiac arrest have at best a 15% chance of reaching a hospital alive, and of those, half die before discharge.

Implanted cardioverter defibrillators (ICDs) have the potential to change all this. An ICD can rapidly terminate an episode of ventricular tachycardia or ventricular fibrillation better than 95% of the time, and in the appropriate patient population, can dramatically improve survival. Good data from large randomized prospective trials have already identified populations for whom ICDs have proven benefit, and the technology is widely available. Yet despite these compelling findings, many at risk for sudden death remain undiagnosed and untreated.

"Generally speaking, if someone has a persistent cardiomyopathy, with a left ventricular ejection fraction less than 35%, they are at high enough risk for sudden cardiac arrest that they ought to be a candidate for an implantable defibrillator," says Daniel M. Philbin, MD, FACC, electrophysiologist at the New England Heart Institute.



DANIEL M. PHILBIN
MD, FACC

"Population-based data show that the number of people in that group far exceeds the number of ICDs we implant, so this treatment appears to be significantly under-prescribed."

Better screening and primary prevention

Dr. Philbin suggests three reasons for the shortfall in the primary prevention of sudden cardiac arrest. First, some patients with low ejection fractions may be well compensated clinically, with few functional limitations, making treatment appear unwarranted.

Dr. Philbin suggests that this can create a false sense of security.

"Data from the MADIT and SCuDHFT trials imply that the risk of sudden death persists for years after the discovery of the cardiomyopathy," he states, "and in a subset of the Maastricht Circulatory Arrest registry, the median time from a myocardial infarction to a later sudden arrest was nine years. If your ejection fraction is low, your risk is high, no matter how long you've gotten away with it, and you need protection."

Media reports about defibrillator recalls may be another reason why some physicians and patients may have concerns about the effectiveness or safety of defibrillators. Dr. Philbin notes that defibrillators have been subject to field actions for a very low rate of failure to deliver therapy when called upon, and almost never for directly harming the patient. The underlying rhythm risk therefore remains the bigger problem.

"If a person has coronary artery disease and an ejection fraction of less than 35%,

Are We Neglecting An Epidemic? continued on page 4

New England Heart Institute



INSIDE

- Are We Neglecting An Epidemic? pg. 1
- Stress Cardiomyopathy Mirrors Myocardial Infarction pg. 2
- VATS Procedures Provide Definitive Patient Benefits pg. 2
- Role of Diastolic Dysfunction in Heart Failure pg. 3
- New EP Lab Enhances Interventional Procedures pg. 4

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Stress Cardiomyopathy Mirrors Myocardial Infarction

A postmenopausal woman complained of chest pain as she was being prepped for endoscopy. Her EKG demonstrated changes consistent with an acute myocardial infarction and she was consequently taken immediately to the catheterization laboratory at Catholic Medical Center. During catheterization, her coronary arteries appeared normal, but a left ventriculogram demonstrated lack of contraction of the entire left ventricular apex. The diastolic frame of the left ventriculogram had the configuration of a round pot with a long narrow neck. Although this patient first appeared to be experiencing a myocardial infarction, this diagnostic information led to the correct diagnosis of stress cardiomyopathy, notes interventional cardiologist Michael J. Hearne, MD, FACC.



MICHAEL J. HEARNE
MD, FACC

This condition, originally dubbed Takotsubo cardiomyopathy after a Japanese octopus-trapping pot with this distinctive shape, is also known as apical ballooning syndrome or by its more colloquial name, “broken heart syndrome.” Intense emotional stress, such as a death or traumatic event, or physical stress, such as a stroke, seizure or operative procedure, is associated with stress cardiomyopathy. The syndrome is most frequently seen in postmenopausal women, although some younger women and men can present with this condition.

Diagnostics reveal syndrome

Stress cardiomyopathy, which causes transient severe heart muscle weakness, may initially be indistinguishable from myocardial infarction. Patients may present with chest pain, dyspnea, syncope, hypotension, congestive heart failure or a combination of these symptoms. Many of these patients can also exhibit ST segment elevation or diffuse T wave inversion.

The diagnosis of stress cardiomyopathy is typically made in the cardiac catheterization laboratory. Patients exhibiting little or no evidence of occlusive coronary artery disease and who have the typical

left ventricular wall motion abnormality are most likely experiencing stress cardiomyopathy. “It is a somewhat unusual contraction abnormality because it involves the entire apex and usually one artery does not supply all the myocardium affected, so several coronary artery distributions are involved,” explains Dr. Hearne. “The diagnosis should be considered in patients presenting with chest pain following an extremely stressful situation, primarily psychological but also physiological. The presenting symptoms, the modest enzyme elevation, the lack of significant coronary artery disease and the typical left ventricular contraction abnormality are all indicative of stress cardiomyopathy.”

Causes and treatment undetermined

Optimal treatment of stress cardiomyopathy is as yet undetermined. Typically, a patient is treated initially with ACE inhibitor and beta blocker therapy, although no definitive therapy exists other than the tincture of time, notes Dr. Hearne. Within a few days or weeks, a patient’s left ventricular contraction abnormality almost always returns to normal, evidenced by a follow-up echocardiogram. Because heart muscle weakness caused by the syndrome is temporary, a patient’s long-term prognosis is excellent.

The etiology of stress cardiomyopathy is not fully understood. Potential etiologies include epicardial coronary artery spasm, coronary microvascular spasm, and catecholamine-induced myocardial stunning, as many patients with this syndrome have elevated levels of plasma catecholamines.

“Physicians should be aware of this syndrome, especially if, following acute emotional or physical stress, a postmenopausal woman presents with chest pain and other findings suggestive of an acute myocardial infarction,” says Dr. Hearne. “It is important to make the proper diagnosis in this situation so that unnecessary procedures such as percutaneous intervention or bypass surgery are not performed and the patient can be treated appropriately.”

For more information on stress cardiomyopathy, contact Dr. Hearne at 603.669.0413. ❖

VATS Procedures Provide Definitive Patient Benefits

The cardiothoracic surgeons at Catholic Medical Center have been performing video-assisted thoracoscopic surgery (VATS) procedures for a decade. David C. Charlesworth, MD, FACS, advanced the use of this procedure further when he performed the first VATS lobectomy in Manchester in mid-February. Minimally invasive VATS procedures provide patients with particular advantages over open procedures, including smaller incisions, reduced postoperative pain and faster recovery.

“All three of our cardiothoracic surgeons are committed to performing VATS procedures, including the VATS lobectomy, because



DAVID C. CHARLESWORTH
MD, FACS

of the benefits these procedures provide to patients,” says Dr. Charlesworth. “Our default position is to perform VATS for virtually any lung or chest procedure, if possible, as it is ideal medical care.”

To read a case study about the first VATS lobectomy performed in the area, search “Atrium” at neheartinstitute.org. For more information on VATS procedures, contact Dr. Charlesworth at 603.663.6340. ❖

Role of Diastolic Dysfunction in Heart Failure

Historically, when patients presented with symptoms of fluid overload consistent with congestive heart failure (CHF), physicians assumed that the most likely cause was systolic dysfunction. Over the past 10 years, however, as our understanding of diastolic dysfunction has grown, it has become clear that the cause of heart failure in an increasing number of these patients is due to abnormalities in diastolic function. In fact, for patients presenting with CHF in their 70's, up to 50% are found to have diastolic CHF. This changing etiology of heart failure requires physicians to be more aware of diastolic dysfunction to correctly diagnose and manage its manifestations.



ROBERT C. CAPODILUPO
MD, FACC

If a patient presents with dyspnea, rales, lower extremity swelling and a chest x-ray consistent with pulmonary congestion, a diagnosis of CHF becomes part of the differential, says Robert C. Capodilupo, MD, FACC, director of the Congestive Heart Failure Clinic at the New England Heart Institute. To determine the cause of heart failure, cardiologists typically perform an echocardiogram. If the echo demonstrates a normal ejection fraction, diastolic heart failure moves up higher on the differential diagnosis.

To more accurately describe this condition, a growing number of cardiologists suggest that diastolic heart failure should be labeled “heart failure with a preserved ejection fraction.” Systolic dysfunction, on the other hand, is heart failure with an inefficient heart squeeze or “bad pump.” It is important to note that the term diastolic dysfunction and diastolic heart failure with a preserved ejection fraction are not synonymous; the former refers to asymptomatic abnormalities in cardiac filling, whereas the latter refers to patients who present with symptoms of heart failure and have a normal left ventricular ejection fraction.

Diagnosing diastolic dysfunction

One reason for the increase in diastolic dysfunction as patients age is believed to be the chronicity of disease, explains Dr. Capodilupo. As people age, their vasculature becomes increasingly stiff and less responsive; a similar phenomenon occurs in the left ventricle. Diastolic dysfunction is a failure of the heart to relax, causing the heart to fill to a less than optimal volume, subsequently reducing the volume of blood ejected.

To determine the severity of a patient's diastolic dysfunction, cardiologists in the echocardiography lab examine the flow pattern of the mitral valve and tissue Doppler imaging of the mitral annulus.

The presence of left ventricular hypertrophy, left atrial size and mitral annular motion are also assessed.

“With diastolic dysfunction, the stiff left ventricle requires higher than usual pressures to fill it to a normal volume,” explains Dr. Capodilupo. “When the mitral valve opens, much of that pressure is transmitted back to the left atrium, leading to left atrial dilatation, as the left atrium is not used to being a high-pressure chamber.”

Left atrial dilatation may therefore be one of the first indications that the patient has significant disease, including heart failure with a preserved ejection fraction, hypertension or cardiovascular disease. Left atrial dilatation may function as a barometer for cardiovascular disease, a “HbA1c” for the chronicity of disease, says Dr. Capodilupo. These patients should be treated aggressively.


Treating diastolic dysfunction

Although multiple clinical trials have formed the foundation of evidence-based medicine for the treatment of systolic dysfunction (those with a left ventricular ejection fraction of less than 35 to 40%)—including ACE inhibitors, angiotensin receptor blockers (ARBs), aldosterone antagonists and beta blockers—no randomized clinical trial data are available for the treatment of diastolic dysfunction, adding to the challenge of managing these patients. As with systolic dysfunction, potentially reversible causes of diastolic heart failure (see box) should be ruled out. If the etiology of diastolic heart failure is felt to be due to left ventricular stiffness, diuretics are a principle course of treatment to manage the resultant volume overload.

“We want to keep a patient's filling pressures low enough to not cause pulmonary edema, yet high enough to maintain an adequate cardiac output as these patients are preload dependent,” explains Dr. Capodilupo. “In early diastolic dysfunction, grades 1 and 2, beta blockers and calcium channel blockers are important to decrease the heart rate, giving ample time for the left ventricle to fill. However, in more advanced diastolic dysfunction, grades 3 and 4, left ventricle filling might be complete by mid-diastole, and prolonging diastasis may be detrimental, leading to issues such as diastolic mitral regurgitation.”

Given the relative lack of data in the treatment of this condition, research trials focusing on diastolic heart failure have been increasing. Possible pharmacologic targets for treatment include endothelin receptor antagonists, as well as phosphodiesterase (PD5A) inhibitors. The New England Heart Institute's Research Department participated in one such trial examining the role of endothelin antagonists and now is a participating center for the RELAX trial, a randomized placebo-controlled trial conducted by the National Institutes of Health looking specifically at the use of PD5A antagonists in patients who suffer from diastolic heart failure. Dr. Capodilupo is the principal investigator for the RELAX trial at NEHI.

“As practitioners, we have all become well aware of the impact of left ventricular systolic dysfunction on patients' morbidity and mortality,” notes Dr. Capodilupo. “As we delve deeper into the etiology of CHF, it is now recognized that an increasing number of those afflicted suffer from an abnormality of diastolic function, and the proper identification of these patients will hopefully lead to new and more effective treatments.”

For more information on diastolic dysfunction and diastolic heart failure, contact Dr. Capodilupo at 603.669.0413. 

Causes of Diastolic Dysfunction*

- Bradycardia
- Arrhythmia
- Constrictive pericarditis
- Valvular disease
- Infiltrative disorders (amyloid, hemochromatosis)
- Pericardial effusion
- Ischemia

If all other causes of diastolic dysfunction have been ruled out, left ventricle stiffness is the assumed cause. **partial list*

New EP Lab Enhances Interventional Procedures


The recent completion of a fully digital electrophysiology laboratory at the New England Heart Institute is allowing electrophysiologists to perform complex interventional procedures with a higher level of efficiency and reduced radiation exposure. The updated EP lab's new state-of-the-art digital fluoroscopy provides high resolution digital imaging that gives electrophysiologists a more well-defined view as they perform intra cardiac procedures.

"A major benefit of this new fluoroscopy is the reduced radiation dose for patients, as well as for technicians and physicians," says Connor J. Haugh, MD, FACC, director of the NEHI Electrophysiology Laboratory.



CONNOR J. HAUGH
MD, FACC

"We are able to get improved image quality more efficiently, with a reduction in radiation of up to 60 percent, a reduction that is especially important with complex cases that can take six to seven hours to complete."

To read more on the updated EP Lab, search "Atrium" at neheartinstitute.org. For more information on digital fluoroscopy, contact Dr. Haugh at 603.669.0413. 

Are We Neglecting An Epidemic? *continued from pg. 1*

a defibrillator will reduce his or her likelihood of dying by 30% over the first five years after the implant," he adds. "That reduction is so dramatic, and the likelihood of a problem with the ICD is so trivial, that the possibility of harming the patient is far offset by the likelihood that the device will save his or her life."

Lastly, some patients at high risk for sudden death may not be identified in a primary care setting because of under-screening. Dr. Philbin and other electrophysiologists recommend that every patient who has coronary artery disease or has had a myocardial infarction or congestive heart failure should have an ejection fraction measurement by cardiac echo. "Unless a patient has another limiting diagnosis or is very elderly, an ejection fraction of less than 35% should prompt us to proffer him or her the protection of an implantable defibrillator," he explains.

ICD implantation and monitoring


Arrhythmia specialists implant ICDs transvenously in an electrophysiology laboratory while the patient is under conscious sedation. A 2 cm incision is made in the shoulder area to access the cephalic or subclavian vein, with up to three leads advanced under fluoroscopy into the heart chambers. If the walls of the left ventricle are uncoordinated, the third lead is placed via the heart's vein system to resynchronize the ventricle, which improves heart failure symptoms such as dyspnea. The back ends of the leads are placed in the device, which is then inserted subcutaneously near the clavicle. The electrophysiologist tests the implanted defibrillator by inducing a ventricular arrhythmia in the patient and

ensuring the device can sense the arrhythmia and appropriately shock the heart into sinus rhythm. Most patients return home one day after the procedure.

Following implantation of the ICD, many patients receive a device for their telephones to allow NEHI medical staff to remotely monitor the defibrillator. The system relays the device's battery level, indicates if the patient has experienced any arrhythmia events, downloads data collected from inside the heart during an event, and supplies information on shocks delivered by the device and why the shocks were administered.

"The New England Heart Institute maintains the only fully staffed electrophysiology laboratory in southern New Hampshire, so we do a large volume of both screening for sudden cardiac arrest, implantation of a defibrillator if appropriate, and maintenance of care for the device," states Dr. Philbin. "By monitoring devices remotely, we can oversee the care of patients wherever they live, allowing us to take appropriate action from here."

This can include sending patients to a local hospital, arranging a prompt outpatient evaluation at the New England Heart Institute, or assuring them by phone that they are okay. "It's particularly important in a place with a geographically dispersed population," adds Dr. Philbin, "because we can assist primary physicians throughout New Hampshire and the region without forcing patients to travel for hours to retrieve the device's data."

For more information on the diagnosis and treatment of patients at risk for sudden cardiac arrest, contact Dr. Philbin at 603.669.0413. 

We'd like to hear from you

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We welcome your comments about this issue of *Atrium* and encourage your ideas about future stories. Please contact us at catholicmedicalcenter.org or send e-mail to gwinslowpine@cmc-nh.org.

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