



Hypertension: Initial Work-up Critical to Successful Management



Clarence Shub, MD

Hypertension is a major risk factor for cardiac disease and stroke. The incidence of hypertension in the general population is approximately 33%, with increasing prevalence over the past few decades, especially in older patients. According to Clarence Shub, MD, a cardiologist and member of the hypertension section at Mayo Clinic in Rochester,

approximately two-thirds of individuals over the age of 60 years have hypertension, and the prevalence is even higher in African American and Hispanic patients. "Identifying hypertensive patients and appropriately treating their hypertension reduces the risk of cardiovascular complications and other end-organ damage," says Dr Shub. Evaluation of patients with hypertension should include the following objectives:

- To define the severity of hypertension, including the presence or absence of target organ damage.
- To determine the presence or absence of other risk factors of cardiovascular disease.
- To search for clues of secondary causes of hypertension.
- To confirm that hypertension is sustained by repeating measurements in the office and/or at home.

The medical history should include the known duration of hypertension, symptoms suggesting secondary causes of hypertension, and history of recent changes in weight, especially weight gain. A review of current drug treatment is important. Among drugs that may increase blood pressure are high-dose estrogens, adrenal steroids, nonsteroidal anti-inflammatory agents, nasal

decongestants, appetite suppressants, and cyclosporine. An assessment of smoking and dietary intake, including sodium, alcohol, saturated fat, and caffeine, should be done. Any history of over-the-counter medications, herbal remedies, analgesics, and illicit drugs (some of which may raise blood pressure or interfere with the effectiveness of antihypertensive therapy) is important. The response to and any adverse effects of previous antihypertensive therapy should be noted. Finally, psychosocial and environmental factors that may influence hypertension control and treatment compliance should be reviewed.

For proper blood pressure measurement, the patient should rest for 5 minutes and refrain from smoking or caffeine ingestion for 30 minutes before the measurement. The patient should be seated comfortably with feet on the floor, back and arms supported, with the arm used for blood pressure measurement positioned at the level of the heart. The clinician should use the appropriate cuff size for the patient; the cuff should encircle at least 80% of the upper arm. Two or more readings, separated by at least 2 minutes, should be averaged. Measuring blood pressure with the patient standing may be considered, especially in patients with a history of orthostatic dizziness or weakness. Blood pressure at the initial visit should be verified in the contralateral arm; if the values are different, the higher value should be used. A difference of 20 mm Hg or more between the 2 arms should arouse suspicion of arterial disease involving the subclavian, innominate, or brachial arteries. Disease in these arterial beds is most commonly caused by atherosclerosis, but fibromuscular dysplasia or arteritis also may be responsible.

The initial physical examination should include the following: assessment of height and weight; ophthalmoscopic examination (especially if hypertension is severe or of new onset); evaluation for carotid and femoral artery bruits, distended neck veins, and/or an enlarged thyroid gland; examination of the heart for

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abnormalities in rate and rhythm; and precordial palpation for the assessment of left ventricular hypertrophy. For proper palpation of precordial impulses, the patient should be examined in the supine, seated, upright, and left lateral decubitus positions. Examining the apical impulse with the patient in the sitting position may be the best method to appreciate subtle abnormalities of precordial motion. The normal apical impulse, usually generated by left ventricular contraction, occurs during early systole with an outward motion imparted to the chest wall. During mid and late systole, the left ventricle is diminishing in volume, and the apical impulse moves away from the chest wall. Thus, outward precordial apical motion occurring in late systole, referred to as a sustained impulse, is abnormal. The apical impulse of left ventricular hypertrophy without dilation is sustained and localized. It should not be displaced (the latter implying left ventricular dilation) but may be accompanied by a palpable presystolic outward movement, the A wave.

The physical examination also includes assessment of clicks, murmurs, and third and fourth heart sounds; examination of the lungs for crackles or bronchospasm; examination of the abdomen for bruits, masses, or abnormal aortic pulsation suggesting abdominal aneurysm; and finally, examination of the extremities for diminished peripheral arterial pulsations, including assessment of radial-femoral pulse delay, suggesting possible aortic coarctation. The latter finding would be especially important in a young patient with hypertension. In addition, the presence of peripheral edema should be noted.

Routine laboratory tests recommended before initiating therapy consist of testing to determine the presence of target organ damage and other cardiovascular risk factors. These include urinalysis, complete blood cell count, blood chemistry (potassium, sodium, creatinine, fasting glucose, and serum lipids), and 12-lead electrocardiography.

Optional tests, depending on clinical circumstances, include determination of creatinine clearance, microalbuminuria, 24-hour urinary protein, blood calcium, uric acid, glycosylated hemoglo-

bin, and thyroid-stimulating hormone. Echocardiography can be considered for assessment of left ventricular function, left ventricular mass, and left atrial size, as well as other features that might suggest diastolic dysfunction, especially in a dyspneic patient. In selected circumstances, assessment of plasma renin and aldosterone activity can be obtained if there is a question of primary aldosteronism and imaging of the renal arteries if renal artery stenosis is suspected (eg, magnetic resonance angiography or renal ultrasonography). Renal artery stenosis (Figure) should be considered in an older patient with resistant hypertension, any hypertensive patient with progressive renal insufficiency, or a patient with a history of “flash” pulmonary edema.

Additional diagnostic procedures may be indicated to seek secondary causes of hypertension, especially in patients with onset of hypertension at the extremes of age or when the severity of hypertension suggests secondary causes. Secondary causes of hypertension also should be considered when blood pressure responds poorly to drug therapy; when the patient has a history of previously well-controlled hypertension and blood pressures begin to increase without other explanation; in a patient with advanced stages of hypertension; or in a patient with sudden onset of hypertension.

Secondary causes of hypertension are rare, but potentially correctable, and therefore they are important to recognize. Labile hypertension or paroxysms of hypertension accompanied by headache, palpitations, pallor, or perspiration suggest pheochromocytoma. Abdominal bruits are neither sensitive nor specific for renal artery stenosis, but those that lateralize to the flank or have a diastolic component suggest renovascular disease (Figure); truncal obesity with purple striae suggests Cushing syndrome. A history of snoring (with or without daytime somnolence) raises the possibility of obstructive sleep apnea, a recently recognized, important, and potentially correctable cause of hypertension.

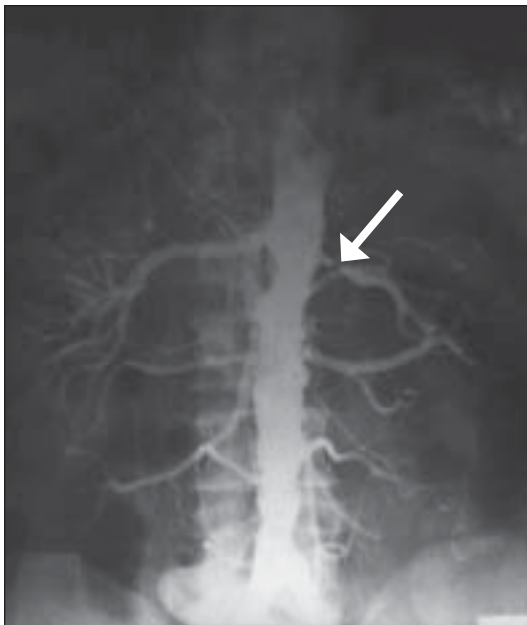
Clues from initial laboratory tests include unprovoked and otherwise unexplained hypokalemia (suggesting primary aldosteronism), hypercalcemia (suggesting hyperparathyroidism), and elevated creatinine or abnormal urinalysis (suggesting renal parenchymal disease).

“Before the definite diagnosis of hypertension is established, the blood pressure should be measured on at least 2 occasions after the original determination, unless the initial levels are markedly elevated, for example, higher than 180/110 mm Hg, in which case, the diagnosis should be considered as established and therapy initiated promptly,” says Dr Shub. Individuals with initially elevated blood pressures

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Surgical repair of the aortic arch in a patient with total renal artery stenosis. Arrow denotes focal stenosis of the left renal artery at angiography.

who become normotensive on follow-up evaluation should not be ignored, because they have a tendency to develop persistent hypertension in the future. Follow-up at regular intervals between 6 months and 1 year is recommended.

In general, cardiovascular risk increases as blood pressure rises. Elevation of systolic blood pressure is a more reliable and important prognostic factor than elevated diastolic pressure, especially in men. An exception is individuals less than 50 years old in whom diastolic blood pressure is a reliable risk predictor. In addition, increased pulse pressure, which reflects decreased arterial compliance in older patients, appears to be an even better index of cardiovascular risk.

Future *Cardiovascular Update* articles will feature specific hypertensive syndromes and their treatment. For additional resources, please see the *Cardiovascular Update* Web site.

UNDER THE STETHOSCOPE

by Clarence Shub, MD

Physical Examination in Hypertensive Patients

- Ophthalmoscopic examination is not diagnostic in the majority of patients with mild or moderate hypertension, but it is useful in patients with severe hypertension.
- An S4 is common in hypertensive patients (50%-70% incidence), especially in older patients and when left ventricular hypertrophy is present.
- An S3 is unusual in hypertensive patients, and when present, it suggests left ventricular systolic failure.
- An abdominal systolic bruit may be heard in patients with renal artery stenosis, but sensitivity and specificity of the bruit as an indicator of renal artery stenosis are poor.

Heart Failure Management: New Devices and Leads Increase Resynchronization Benefit



Robert F. Rea, MD

Cardiac resynchronization therapy (CRT), used adjunctively with appropriate medical therapy, provides additional improvement in congestive heart failure symptoms in many patients. In some studies, CRT has improved mortality, especially when combined with an implantable cardioverter defibrillator (ICD). “For patients to realize the potential benefits of this therapy, however, the surgical procedure must be low risk and the delivery site of left ventricular (LV) pacing stable, anatomically appropriate, and free from extracardiac stimulation,” says Robert F. Rea, MD, a cardiac electrophysiologist and head of the implantable device section of the Heart Rhythm Services at Mayo Clinic in Rochester. “In addition, the pulse generator must incorporate unique timing algorithms to maximize the percentage of time that LV pacing is delivered, even in the face of irregular underlying native rhythms.”

LV Lead Implantation

Early in the cardiac resynchronization era, standard

right ventricular (RV) pacing leads were placed in coronary veins, usually via coronary sinus guide catheters. With the subsequent development of dedicated LV pacing leads, implant times decreased by about 38%, and successful LV lead placement was achieved in more than 90% of patients. This success was offset by an LV lead dislodgement rate of 8% and by the incidence of extracardiac stimulation (typically phrenic nerve), resulting in lead repositioning in 2% and device reprogramming in 0.5% of patients.

Over-the-wire LV lead models are generally of smaller caliber and can be threaded over an angioplasty guide wire into smaller and more distal tributaries of the coronary venous system. Overall, successful and timely LV lead implantation is achieved in 95% of patients. And, as hoped with the over-the-wire technology, the dislodgement rate of 3.4% is significantly lower than the rate with larger stylet-driven leads (8%). Extracardiac stimulation, however, requires repositioning in 0.8% or reprogramming (or turning off the LV output) in 8.8% of patients.

The contrasting successes and limitations of these 2 types of leads illustrate an important consideration in LV lead design. More proximally located, larger-

diameter leads may dislodge more frequently, but as they do not pass close to the more distally located phrenic nerve, they produce extracardiac stimulation less frequently.

Newer LV Lead Designs

The ability to pass leads more distally and in smaller and angulated vein tributaries has provided operators with more options in terms of lead placement. This flexibility was recognized as increasingly important as a substantial number of patients failed to derive clinical benefit from earlier CRT systems. Early acute hemodynamic studies using epicardial LV lead placement suggested that the mid-lateral to posterolateral LV provided the best hemodynamic results. Subsequent studies using tissue-Doppler measurements of myocardial contraction have shown that highly targeted LV lead placement in regions of greatest dyssynchronous contraction may be required for optimal clinical effect. “It is recognized that prolongation of the QRS interval, a criterion for enrollment in all major randomized trials, might be a relatively blunt instrument for discerning potential responders to CRT,” says Dr Rea. These observations have led to an increasingly more critical approach to the positioning of the LV lead.

Because coronary vein leads do not have myocardial trabeculae, many manufacturers have relied on shaping the distal portion of the lead for fixation. Current lead models include preformed tips—the curves in the S-shaped tip wedge against opposite walls of the vessel, and the helical lead tips “bunch up” in the vein when the wire is removed. Increased lead diameters have conferred

additional endovascular stability. The development of bipolar LV leads and independently programmable RV and LV outputs have provided a number of approaches to minimize the likelihood of extracardiac stimulation.

Features of New CRT Devices

Programmable V-V Timing. Available in CRT pacemakers and now being released in CRT defibrillators is the ability to deliver pacing impulses to the RV and LV sequentially rather than

simultaneously. In a small observational study, improved tissue-Doppler indices of resynchronization and echocardiographic estimates of ejection fraction have been demonstrated with RV-LV timing offset, with some suggestion that the etiology of heart failure may play a role in choice of RV versus LV preactivation. In another study, stroke volume determined from the aortic velocity time integral was maximized by changing RV-LV impulse timing. With V-V delays of ± 40 ms, stroke volume was maximized at discharge. At 3-month follow-up, the optimized V-V timing was nearly the same in 27 of 34 patients, indicating relative stability of the initial programming; however, there was no discernible change in clinical status between patients who did and did not undergo V-V timing optimization in this small sample. The exact mechanism by which this effect is achieved is unclear but likely reflects optimization of left atrial-LV activation time. Further study is warranted, especially with deliberate pacing of the right atrium, as interatrial conduction times vary with spontaneous and paced atrial rhythms depending on atrial lead location.

Maximized Biventricular Pacing. Typically the goal of standard pacers and ICDs is to minimize the use of pacing, which improves battery longevity. In the case of DDD/R pacers and ICDs, there is an increasing trend specifically to minimize the amount of time the RV is paced so as to minimize device-related ventricular dyssynchrony. With CRT devices, the opposite is the case: any situation that results in less than about 90% delivery of synchronizing pacing (biventricular or LV) can be deleterious.

Ventricular ectopic beats can potentially interfere with biventricular pacing by simple inhibition of pacing output, as seen in VVI/DDD pacers and ICDs. In many CRT devices, however, a sensed ventricular event can initiate right, left, or biventricular pacing outputs in an attempt to maintain synchronous ventricular activation. Ventricular ectopic beats (premature ventricular contractions [PVCs]) may interfere with the delivery of CRT. In a standard pacemaker, a PVC initiates extension of the postventricular atrial refractory period (PVARP) so as to minimize the possibility of a pacemaker-mediated tachycardia. This may bring the next sensed atrial event inside the PVARP. This refractory atrial event is not tracked, and a conducted rather than paced ventricular event may occur. This could initiate another PVARP that encompasses the next atrial event, triggering another conducted ventricular event, and so on. To circumvent this problem, in many CRT devices after a programmed number of ventricular sensed events that follow atrial refractory sensed events, the PVARP is shortened to allow atrial tracking and biventricular pacing to resume.

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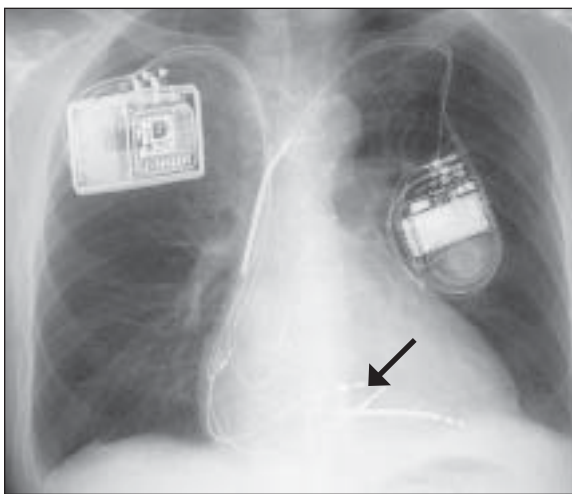


Figure 1. Posteroanterior chest radiograph showing a left-sided ICD and a right-sided CCM pulse generator (investigational device). This patient had advanced congestive heart failure but a narrow QRS complex and hence did not qualify for a standard CRT device. The arrow indicates a septal lead that delivers a subthreshold stimulation pulse.

Atrial fibrillation is common in CRT recipients, and ventricular rates above the programmed rate could inhibit delivery of resynchronization therapy. In some patients, it may be necessary to prescribe atrioventricular nodal blocking drugs or to ablate the conduction system and thus enable the device to assume control of the ventricles. There are, however, pacing algorithms in current devices that attempt to pace the ventricles slightly faster than the

conducted rate. The average ventricular response rate during a mode switch for atrial fibrillation is calculated over a short time interval. The pacing rate is then adjusted upward until conducted beats are eliminated. When this is achieved, the pacing rate is decreased slightly until conducted beats reemerge. The rate is then adjusted upward slightly once again. With this iterative algorithm, the pacemaker may assume substantial control of the ventricles, even during relatively rapidly conducted atrial fibrillation, and helps to ensure consistent delivery of CRT.

Cardiac Contractility Modulation. In isolated heart muscle, subthreshold stimuli increase the force of contraction, an effect likely dependent on calcium ion flux. Whereas available devices for resynchronization depend on leads that pace the left ventricle, with the cardiac contractility modulation (CCM) device, subthreshold stimulation pulses are delivered to the septal aspect of the right ventricle. Two right ventricular leads are placed; one is directed into the septum for delivery of the CCM pulses and the other is located a distance away for sensing. Pulses of substantial amplitude and duration are delivered during the absolute refractory period in order to avoid local capture (Figure 2). Because this approach results in a large current drain, the device is programmed to deliver this therapy for a limited number of hours each day. It can be delivered via LV epicardial leads but results in considerable patient discomfort, likely as a result of stimulation of epicardial nociceptors with the large-amplitude, long-duration pulses. “Despite the fact that the impulses are delivered to the septum, it appears that global LV function is improved, and preliminary data from investigational implants in Europe are encouraging,” says Dr Rea. In the United States, the device is in its early investigational phase.

For additional information about device protocols at Mayo Clinic in Rochester, please see the *Cardiovascular Update* Web site. The next issue of *Cardiovascular Update* will review current indications for ICD implantation.

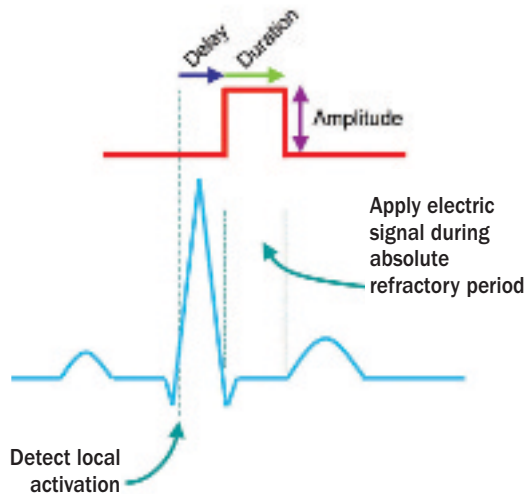


Figure 2. Diagram of the CCM pulse. A high-amplitude, long-duration pulse is applied in the absolute refractory period to stimulate Ca^{++} ion release from sarcoplasmic reticulum and enhance contractility.

Lower Levels of B-Type Natriuretic Peptides Predict Short-term Mortality in End-Stage Heart Failure Patients Treated With Nesiritid

B-type natriuretic peptides (BNP/NT-proBNP) are released from the myocardium under conditions of chronic volume overload and are elevated in the plasma of patients with heart failure. Nesiritide (human recombinant BNP) has been approved by the US Food and Drug Administration for treatment of patients in acutely decompensated chronic heart failure. Investigators at Mayo Clinic in Rochester conducted a prospective study of 40 patients with severe chronic heart failure to evaluate whether biomarkers can be used to titrate therapy with nesiritide. This study demonstrated that, despite a good clinical response and a robust response in cyclic GMP levels (which is thought to be the effector of the therapeutic benefit), there were no marked reductions in the natriuretic peptide

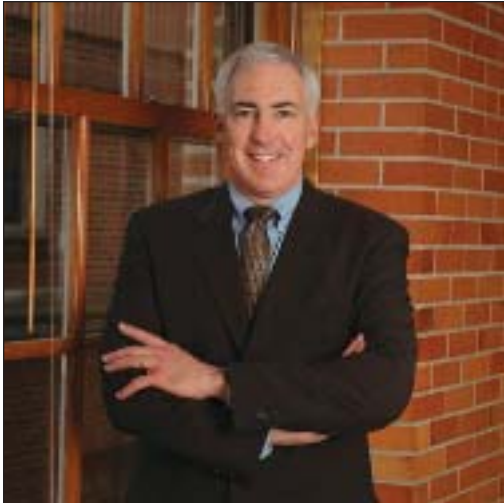
levels, suggesting that natriuretic peptide levels respond less rapidly than expected, making titration based on the values of these markers problematic.

Recently, there have been concerns about potential adverse effects of worsening renal function and death after nesiritide use in patients with heart failure. Analysis of the Mayo data suggest otherwise. “In contrast to most previous studies that have suggested that higher levels of BNP and NT-proBNP predict an adverse prognosis, we found exactly the opposite,” says Wayne L. Miller, MD, PhD, lead author of the study published in the September 15, 2005, issue of the *American Journal of Cardiology*. “Patients with poor short-term survival after hospital discharge had much lower blood levels of NT-proBNP and

BNP than those patients who survived long-term after discharge.”

The study does not define a mechanism for these effects. There may be an exhaustion of the endogenous production of cardiac natriuretic peptides in patients with advanced heart failure. The study's data could be interpreted as suggesting that more rather than less nesiritide might be of benefit in such heart failure patients. “If so, it may be that, rather than discarding nesiritide, we need to find ways to augment natriuretic peptides,” says Dr Miller. “The data underscore the complexity of the natriuretic peptide system and suggest that additional investigations are needed into both natriuretic peptide responses and how to optimize their use in patients with advanced heart failure.”

Is There a Role for Mitral Valve Repair in Heart Failure Patients?



Richard C. Daly, MD

The presence of severe mitral regurgitation (MR), even with apparently normal left ventricular (LV) function, adversely affects the left ventricle, sometimes insidiously. If repair of severe MR is performed when the left ventricular ejection fraction (LVEF) falls below even 60%, the late outcome is poor, with a higher incidence of late death and congestive heart failure. If severe MR has a deleterious effect on the normal left ventricle, it seems reasonable to assume that the impaired left ventricle would also

be adversely affected. Indeed, a review of 91 patients with dilated cardiomyopathy showed that 3-year survival was 50% in the absence of MR, 26% with mild MR, and 17% with moderate to severe MR. These findings were statistically significant. “Whether the presence of MR truly affects outcome or whether it is a marker for more advanced myocardial disease remains indeterminate,” says Richard C. Daly, MD, a cardiovascular surgeon at Mayo Clinic in Rochester.

Recent enthusiasm for studying surgical repair of MR in patients with reduced LVEF has stemmed from understanding the prognostic value of early correction of MR in patients with normal LVEF and a desire to improve functional status in patients with congestive heart failure, as well as improvement in technique and surgical outcome for mitral valve

repair. The results of published studies have been mixed. Surgical series have shown improved functional status and reduced LV volume. However, a survival benefit has not been shown, and the selection of appropriate patients for surgical intervention remains a challenge. The anticipated potential for percutaneously placed mitral annuloplasty devices further complicates the issue.

Historically, physicians have been reluctant to consider surgical therapy for severe MR in the presence of reduced LVEF for a number of reasons, most of which are now considered inaccurate. It was postulated that the poor LV might benefit from a “pop-off” effect provided by the MR, effectively reducing LV afterload. Additionally, the risk of mitral valve repair in patients with poor LVEF was believed to be prohibitive because loss of the pop-off effect might further reduce LVEF. The concept that severe MR does not reduce afterload and may actually increase afterload represents a change in the understanding of the pathophysiology of the resulting cardiomyopathy.

Development of MR aggravates LV volume overload and may contribute to further LV dilation and subsequent worsening of MR—a cycle of progressive decline in LV function. Wall stress increases with the increasing diameter of the left ventricle, as described by the law of LaPlace. The increased wall stress increases oxygen consumption, reduces subendocardial coronary blood flow, and probably contributes to the progressive cycle of LV decline. At the myocyte level, increased wall stress is the equivalent of increased myocardial afterload. Thus, the volume overload caused by MR results in increased LV diameter and (at the myocyte level) increased afterload.

Several reports of surgical series have provided evidence of LV benefit from correction of severe

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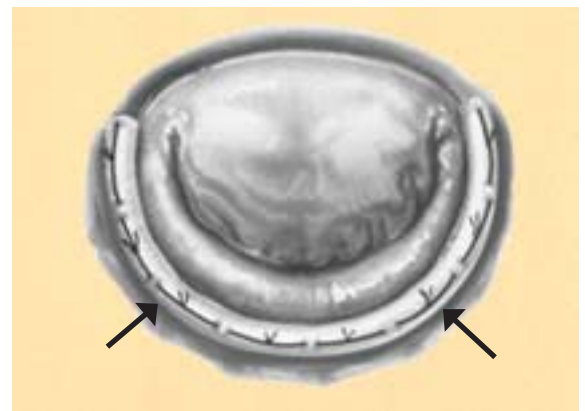
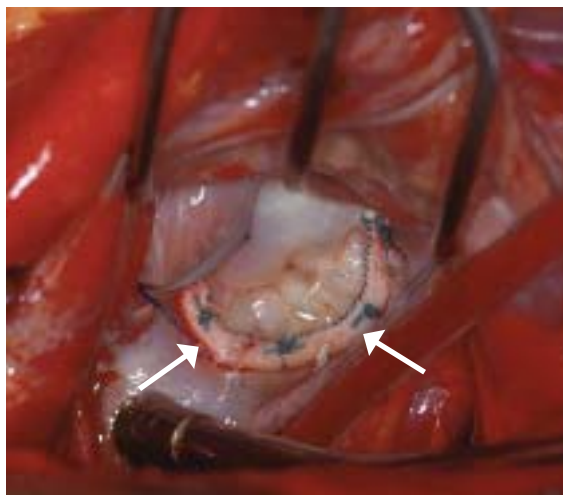


Figure 1. Left, Intraoperative photo after mitral valve repair. Right, Diagram of valve repair. Arrows at ring suture line.

MR in patients with severely reduced LV function. LV end-diastolic volume was reduced (by as much as 26% in 1 study), LVEF improved (by about 9 percentage points), and functional class improved from class 3-4 to class 1-2 in most patients. Five-year survival was 52% to 78% and correlated with the severity of LV dysfunction.

One retrospective review attempted to evaluate survival benefit of mitral valve repair in patients with

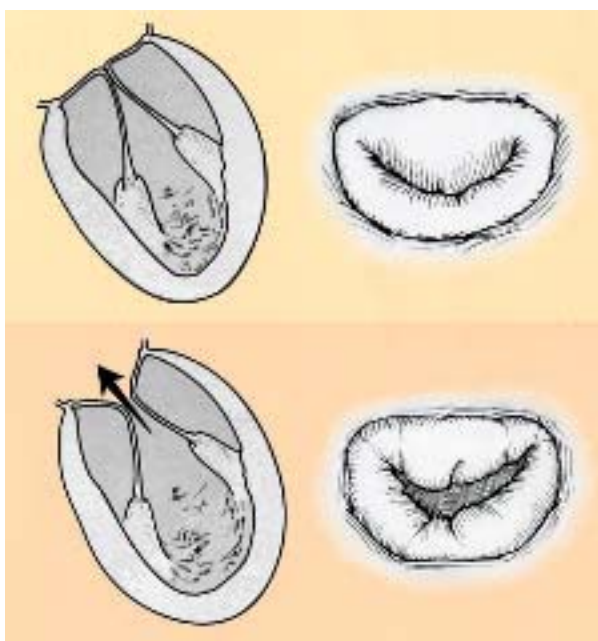


Figure 2. Top, Normal LV size with normal apposition of the mitral valve leaflets. Bottom, Dilated left ventricle with functional MR. The dilated valve ring does not allow valve leaflets to occlude the mitral orifice during systole.

poor LV function (ejection fraction less than 30%). No survival advantage was noted for patients who had repair of MR compared with those treated medically. “In the absence of data showing a prognostic benefit, but with other data showing symptomatic improvement and reasonable medium-term survival, the prudent selection of patients for surgical intervention is imperative,” says Dr Daly.

Operative mortality in recent series has been low: 2.3% to 4.8%. However, certain comorbid conditions increase risk substantially. Contraindications probably include severe disease in other organ systems, severe peripheral or cerebrovascular disease, inoperable coronary artery disease, inotrope dependence, severe pulmonary hypertension, decompensated congestive heart failure (including very low serum sodium), and advanced cachexia. Risk is also increased by age, previous cardiac surgery (especially coronary artery bypass grafting), degree of LV dilation, and extent of LV dysfunction.

Most studies that have focused on mitral valve repair in the presence of poor LV function have combined dilated and ischemic cardiomyopathy pathologies. In general, patients with ischemic disease have a poorer outcome without surgery compared with those with dilated cardiomyopathy, but they also have higher surgical mortality (closer to 9%). In addition, mitral valve repair may be more challenging technically in patients with ischemic disease because of asymmetric LV dilation and papillary muscle displacement.

“There is probably a symptomatic benefit to correcting severe MR in patients with poor LV function,” says Dr Daly. “A survival benefit has not been proven: thus, careful patient selection is important to minimize surgical risk.”

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Recipients of the Rochester, Minnesota, Mayor's Medal of Honor included Lawrence J. Sinak, MD (left), and Hugh C. Smith, MD (right). Dr Sinak and his wife, Anne Black-Sinak, were honored for outstanding contributions to the arts and culture. Dr Smith was honored for outstanding business and industrial leadership. Dr Smith retired as chair of Mayo Clinic Rochester on January 1, 2006.

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