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CLINICAL CARDIOLOGY AND CARDIOVASCULAR SURGERY NEWS

Treating AF With Antiarrhythmic Drug Therapy

When Rate Control Isn't Enough

Peter A. Brady, MD

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice, with approximately 1 in 4 persons over the age of 40 years destined to develop AF in their lifetime. Despite advances in understanding of AF and the emergence of catheter ablation, pharmacologic therapy remains central to the management of AF in most patients.

"In all patients presenting with AF, evaluation should focus on assessment of symptom severity, stroke risk, and exclusion of secondary causes of AF such as undiagnosed hyperthyroidism, cardiomyopathy, mitral valve disease, hypertension, and 'newer' risk factors such as obstructive sleep apnea," according to Peter A. Brady, MD, an electrophysiologist at Mayo Clinic in Rochester. "Patients with rapid ventricular response should be stabilized, initially with a rate control agent, and then begun on anticoagulant therapy while an assessment is made of the need for and safety of cardioversion and a long-term treatment strategy is formulated," he says.

The specific approach to long-term management is influenced by the clinical presentation and setting (eg, postoperative AF), the likelihood of recurrence, and the presence of comorbid conditions. When symptomatic recurrence is deemed likely, prophylactic therapy is warranted. Two broad strategies are control of the ventricular rate and anticoagulant therapy in at-risk individuals. Both strategies have been compared in 5 randomized controlled trials involving more than 5,000 patients.

Follow-up assessment of the adequacy of rate control is important in all patients. Current guidelines recommend a ventricular rate during AF of 60 to 80 beats per minute at rest and 90 to 115 beats per minute during exercise. On the basis of these definitions, rate control is possible in about 70% of patients taking β-blockers, 54% of patients taking calcium channel blockers (with or without digoxin), and 58% of patients taking digoxin alone.

Given the results of trials comparing antiarrhythmic drug (AAD) therapy with rate control alone, is a rhythm control strategy now obsolete? Certainly these trials demonstrated that a rate control strategy is both safe and effective. However, equivalence of rhythm control and rate control in terms of mortality should not be applied to all patients. "The AFFIRM study [Atrial Fibrillation Follow-up Investigation of Rhythm Management] and others enrolled older patients with 1 or more risk factors for stroke, most of whom had structural heart disease. In addition, most patients had few symptoms and were therefore deemed appropriate candidates for either rate control or rhythm control," says Dr Brady. "Thus, the patients enrolled in these studies would be expected to derive the least benefit from a rhythm control strategy and experience the greatest risk from AAD therapy."

Importantly, these trials did not include younger individuals, those without evidence of structural heart disease (so-called lone AF) who account for approximately 15% of AF patients, highly symptomatic individuals, or those in whom adequate rate control was not possible. Thus, a large proportion of patients with AF were not included in the available studies that examined rate control versus rhythm control strategies. On the basis of these limitations, clinical features favoring a rhythm control strategy rather than a rate control strategy are summarized in Table 1.

Agents in Vaughan-Williams classes I and III are used for suppression of AF (Table 2). AAD toxic effects are both cardiac (mainly proarrhythmia) and noncardiac. In most cases, AADs should be initiated at the lowest possible dose and titrated upward while

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the therapeutic effect is monitored. Measurement of serum drug levels is rarely helpful unless overdose or compliance is an issue.

Table 1. Rate Control Versus Sinus Rhythm

SINUS RHYTHM PREFERRED

- Symptoms despite good ventricular rate control
- Intolerance or inefficacy of rate control

RATE CONTROL AND ANTICOAGULATION PREFERRED

- Minimal or no symptoms during AF
- Structural heart disease (excluding tachycardia-induced cardiomyopathy)
- Candidate for long-term anticoagulant therapy

Less clear indications are "younger" patients or individuals who are not good candidates for anticoagulation or those who prefer attempt at "curative" approaches using catheter ablative therapy.

Class IC agents should be avoided in patients with structural heart disease, especially in the presence of myocardial ischemia or left ventricular dysfunction. Class IC agents may also slow atrial conduction sufficient to cause "organization" of the rhythm to atrial flutter and allow 1:1 impulse conduction via the atrioventricular (AV) node to the extent that the ventricular rate may exceed 200 beats per minute. This presents as a wide complex tachycardia, often with hemodynamic instability leading to an incorrect diagnosis of ventricular tachycardia, and is avoided with concomitant use of an AV nodal blocking agent.

Because of the serious nature of potential cardiac and noncardiac toxic effects, careful monitoring of patients taking AADs is required.

Table 2. Most Commonly Used AADs for Suppression of AF

DRUG	ION BLOCK CHANNEL	COMMON INTERACTIONS	NONCARDIAC TOXICITY
Vaughan-Williams class I			
Quinidine	Na, K	↑QT; TdP; vagolytic effects; enhanced AV node conduction	Nausea; vomiting; diarrhea
Procainamide	Na, K	↑QT; TdP; ↑cimetidine and trimethoprim	Lupuslike syndrome; GI upset; agranulocytosis
Disopyramide	Na, K	TdP; exacerbation of CHF	Urinary retention; dry mouth; avoid with history of glaucoma
Flecainide	Na	Atrial flutter with 1:1 conduction; VT	CNS effects; dizziness; blurred vision
Propafenone	Na	Atrial flutter with 1:1 conduction; VT; ↑digoxin/warfarin	Metallic taste; wheezing; dizziness
Vaughan-Williams class III			
Sotalol	K	↑QT; TdP; bradycardia;	COPD or asthma exacerbation exclusive renal elimination
Dofetilide	K	↑QT; TdP; dosage based on creatinine clearance	
Amiodarone	K, Ca, Na	Bradycardia; TdP (rare); ↑warfarin/digoxin	Thyroid; liver; lung; optic neuritis (rare)

AV, atrioventricular; CHF, congestive heart failure; CNS, central nervous system; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal; VT ventricular tachycardia.

Class I Agents

Because sodium channel blocking agents slow conduction velocity, the drug tissue effect is best monitored with reference to the PR and QRS interval and duration. Class I drugs, especially class IC agents, flecainide and propafenone, exhibit use dependence because the degree of sodium channel blocking increases as the heart rate increases. Thus, increasing heart rate during a predismissal treadmill exercise test is a useful screening tool for proarrhythmic effect. In general, widening of the QRS interval should not exceed 150% of the pretreatment interval.

Class III Agents

Increased cardiac repolarization manifests as QT prolongation on the surface ECG. In most patients, the corrected QT interval should not exceed 520 ms during therapy with class III agents. The incidence of torsades de pointes (TdP) with use of class III agents is about 1% to 3% (Table 3). Unlike class I agents, class III drugs block potassium channels more effectively, increasing the likelihood of TdP at slower heart rates (reverse use dependence). In most cases, the risk of TdP increases with drug dosage, but in the case of quinidine, TdP is unrelated to serum drug levels and may occur after the first dose.

Amiodarone

Amiodarone has numerous cardiac and noncardiac toxic effects. Follow-up of patients prescribed amiodarone has not been standardized but should include periodic measurement of thyroid and hepatic function. Patients should be cautioned to avoid sun exposure. Because elimination of amiodarone is exclusively hepatic, it may be used without dose adjustment in patients with renal impairment or on dialysis.

The incidence of amiodarone pulmonary toxicity is about 0.5% to 1%. Although symptoms may develop acutely, most commonly presentation is insidious, with increasing cough and dyspnea, and is often difficult to distinguish from worsening congestive heart failure or pneumonia. A sensitive and specific finding is a decrease in the pulmonary diffusion capacity. One approach is to obtain a chest radiograph and pulmo-

Table 3. Risk Factors for TdP

PATIENT FACTORS

- Female
- Sinus node dysfunction leading to pauses after termination of AF
- $\downarrow K^+/Mg^{2+}$
- ↑Baseline QT
- Use of a class III agent

PHARMACOLOGIC FACTORS

- Concomitant use of drugs that interfere with metabolism of QT-prolonging agent
- Renal impairment of renally excreted agents that prolong QT (sotalol, procainamide, dofetilide)

nary function testing, including diffusion capacity, before initiation of amiodarone; these should be repeated annually or if new symptoms of cough or dyspnea develop.

Dofetilide

Dofetilide is a "pure" class III agent, recently approved for treatment of AF. Hospital admission for initiation requires either 3 days or 12 hours after conversion to normal rhythm (whichever is greater). Verapamil, cimetidine, trimethoprim, ketoconazole, and prochlorperazine should be avoided or withdrawn, and hypokalemia should be treated (serum potassium level ≥ 4.0 mEq/L). If the baseline corrected QT interval is longer than 440 ms (500 ms in patients with intraventricular conduction delay), dofetilide cannot be used. Initial drug dosing is then based on calculated creatinine clearance and subsequently on changes in QT interval. One advantage of dofetilide is favorable mortality data in patients after myocardial infarction, and those with a history of congestive heart failure show a neutral effect of dofetilide on mortality. Thus, dofetilide is useful in patients with AF and a history of myocardial infarction or congestive heart failure. Table 4 summarizes optimal AAD management of AF.

Initiation of AAD Therapy

The decision to initiate AAD therapy in the hospital versus the outpatient setting is determined largely by weighing risk versus cost and the inconvenience of inpatient drug loading. In the case of dofetilide, in-hospital initiation is imperative. Outpatient loading and termination of AF should generally be avoided in patients with evidence of sinus node dysfunction, AV conduction disturbance, bundle branch block, structural heart disease, and QT prolongation. Unsuspected sinus node dysfunction, aggravated by use of AADs (especially class IC agents), may prolong sinus node recovery sufficient to cause syncope and injury on termination of AF. If outpatient loading of propafenone or flecainide, which worsen AV node and His-Purkinje conduction, is planned, cardioversion to restore sinus rhythm in hospital allows assessment of the impact of the drug at AF termination. Because of the QT-prolonging effects of quinidine, procainamide, and disopyramide, in-hospital initiation is recommended.

Outpatient initiation of sotalol is considered safe if the baseline QT interval is less than 450 ms in the absence of renal dysfunction and risk factors for TdP. Despite prolonging the QT interval, amiodarone is only rarely associated with TdP and is commonly initiated in the ambulatory setting, even in patients with persistent AF, provided significant sinus node dysfunction has been excluded and AV nodal blocking agents have been discontinued or the dosage has been reduced to avoid severe AV block.

"In general, in patients who are in sinus rhythm without evidence of sinus node disease, ventricular systolic dysfunction, myocardial ischemia, or baseline QT prolongation, outpatient initiation of AAD is safe," says Dr Brady.

Summary

- An initial strategy of rate control combined with anticoagulant therapy is safe and effective for most patients.
- The goal of long-term rate control is reduction of symptoms and prevention of tachycardia-induced cardiomyopathy.
- Rhythm control is preferable in symptomatic patients intolerant of or despite rate control.
- Patients taking AADs require careful monitoring.

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Table 4. Summary of Drug Choice in Patients With AF

DRUG	LONE AF	CHF, CAD	CAD (NORMAL EF)	RENAL FAILURE
First line	Flecainide; propafenone	Dofetilide; amiodarone	Sotalol	Amiodarone
Second line	Sotalol; procainamide; disopyramide; amiodarone			Propafenone
Avoid		Flecainide; propafenone	Flecainide; propafenone	Sotalol; procainamide; dofetilide

CAD, coronary artery disease; CHF, congestive heart failure; EF, ejection fraction.

Thoracic Aortic Aneurysmal Disease

New Imaging, New Treatments

Timothy M. Sullivan, MD, Kenton J. Zehr, MD, Thoralf M. Sundt III, MD

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The widespread application of high-resolution advanced imaging technologies has increased recognition of thoracic aortic disease, including degenerative aneurysmal disease as well as acute aortic syndromes. "Genetic causes such as Marfan syndrome or Ehlers-Danlos syndrome are well recognized, but relatively uncommon causes of aortic disease," according to Thoralf M. Sundt III, MD, a cardiovascular surgeon at Mayo Clinic in Rochester. "Most aneurysms of the descending thoracic and thoracoabdominal aorta are caused by nonspecific degenerative disease associated with long-standing hypertension and atherosclerosis, although awareness of familial thoracic aortic disease has increased, and 3 genetic loci have been mapped."

Aneurysmal disease may involve any or all segments of the thoracic aorta—the root, the ascending aorta, the arch, or the descending thoracic and thoracoabdominal components. Marfan syndrome most often causes aortic root aneurysms. A less widely recognized but far more common cause of dilation of the ascending aorta and root is bicuspid aortic valve disease.

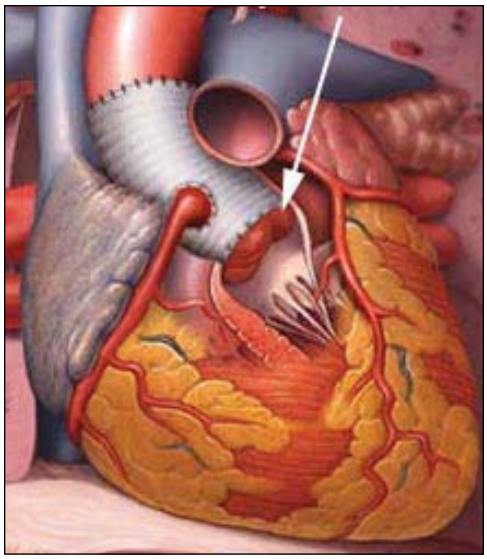


Figure 1. Diagram of graft placement in the valve-sparing ascending aortic aneurysm repair. The coronary arteries are reimplanted. The arrow indicates the aortic valve.

Bicuspid Aortic Valves

Bicuspid aortic valves are present in up to 1% of the general population, making it the most common congenital cardiac anomaly. It is also the underlying pathology in one-third to one-half of all patients treated with aortic valve replacement today. In addition, up to one-third of these individuals have dilation of the aortic root, placing them at risk of rupture and dissection. The cause of this dilation is unclear, with dispute over the relative importance of hemodynamic and genetic factors. "Importantly, even patients with normally functioning bicuspid valves may have severe root and ascending aortic dilation, and that dilation may progress even after successful aortic valve replacement," says Dr Sundt.

Accordingly, echocardiographic evaluation of patients with bicuspid aortic valves should include examination of the diameter of the aortic root and of the ascending aorta as well. In some individuals, the aortic dilation itself may be sufficient to warrant surgical intervention, even if the degree of valvular stenosis or regurgitation does not. Routine evaluation of prosthetic valve function after aortic valve replacement for bicuspid disease should also include imaging of the ascending aorta.

Giant Cell Arteritis

Up to 10% of patients with giant cell arteritis (GCA) may develop aortic aneurysm, most commonly of the ascending aorta and arch; up to half of these patients have aortic aneurysms elsewhere as well. Patients with GCA should have a baseline imaging study of their aorta performed, and surveillance imaging should be considered.

Indications for Operations

Once aneurysmal enlargement of the aorta has been identified, there are generally accepted guidelines for surgical intervention based on diameter. The law of Laplace dictates that wall tension increases with the radius of a tube. Accordingly, the risk of rupture has been shown to increase with the radius of the aorta. Precise measurement of the true diameter therefore is critical. Because axial imaging may not present a true cross section of a tortuous aorta, the shortest dimension in any given image is the most reliable measurement, with the important caveat that a truly cylindrical portion of the aorta must be measured. An important exception to this rule is saccular or asymmetric aneurysms. In patients with these aneurysms, 3-dimensional reconstruction after image acquisition is particularly useful in assessing aortic size and is routine in the Mayo practice.

The critical diameter at which the risk of rupture increases most differs for the ascending and descending thoracic aorta. In the ascending segment, the risk of a rupture or dissection increases most dramatically at a diameter of 6 cm. To avoid overlooking patients destined for dissection or rupture, Mayo cardiovascular surgeons generally recommend surgical intervention at a diameter of 5.5 cm. Among patients at lower operative risk, including younger patients, and

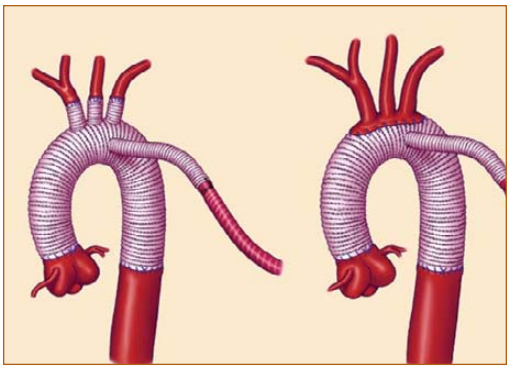
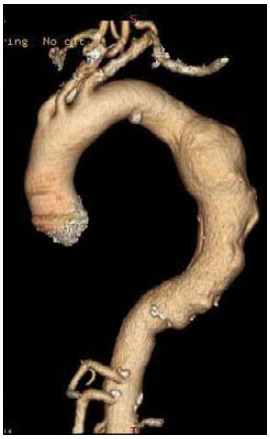


Figure 2. Surgical repair of the aortic arch. Left, total arch replacement. Right, partial replacement.



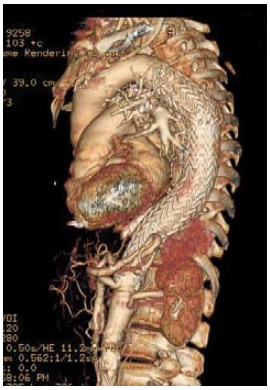


Figure 3. 3-Dimensional reconstruction of descending aortic aneurysm. Before (top) and after (bottom) placement of an endovascular stent.

those known to be at high risk of rupture or dissection such as those with Marfan syndrome or bicuspid aortic valve, earlier intervention is recommended.

"We often intervene earlier and will recommend proceeding with surgery when the diameter of the aneurysm is 5 cm or less in these patients," according to Kenton J. Zehr, MD, a cardiovascular surgeon at Mayo Clinic in Rochester. "Dissection does occur, albeit it at a low incidence, even at this dimension."

Special Consideration for Ascending Aorta

A special case can also be made for intervention on the ascending aorta when valvular stenosis or incompetence is present. Certainly, if the patient is undergoing open cardiac surgery for other reasons, the aorta may be replaced at a diameter as narrow as 4.5 cm. In the case of aortic regurgitation, which may be due to geometric distortion of the root by displacement of the valve commissures at the level of the sinotubular junction, intervention to repair the aneurysm may render the valve competent. In such a case, an argument can be made for early surgery before damage has occurred to the valve leaflets themselves (Figure 1). Experience with such valve-sparing root replacements is growing, and the results are encouraging. While the long-term durability of the native valve leaflets resuspended within a Dacron graft remains unknown, the patient may avoid the need for anticoagulation for at least some time. This technique has found particular application among patients with Marfan syndrome, although it is also applicable in the setting of root dilation secondary to GCA or other arteritides that affect the aortic wall but not the leaflets. In experienced hands, these procedures can be performed at low operative risk.

Aortic Arch Aneurysms

Criteria for treating aortic arch aneurysms are less well defined; however, from a practical standpoint, criteria similar to those for the ascending aorta are applied. The risks of surgery are somewhat higher because blood flow to the brain must be interrupted to perform these procedures. Aneurysmal dilation of the aortic arch can be repaired at low risk using profound hypothermia and circulatory arrest. Circulatory arrest intervals may be minimized and stroke risk reduced through the use of selective antegrade cerebral perfusion and other techniques used at centers with specialized expertise. Repair of the ascending aorta, arch, and much of the descending thoracic aorta can also be undertaken now in a single procedure at acceptable risk (Figure 2). "Using current techniques, many individuals whose condition was thought previously to be inoperable can be offered surgical repair with reasonable expectations of a good result and return to normal functional status," says Dr Sundt.

Special Consideration for Descending Aorta

The criteria for intervention on the descending aorta are somewhat different, because the risk for rupture or dissection of the descending thoracic aorta does not increase dramatically until its diameter is approximately 7 cm. This may be attributable to differences in hemodynamic stress to which this segment of the arterial tree is subjected. Accordingly, surgical intervention in this segment is usually recommended when the diameter reaches 6 to 6.5 cm, depending again on operative risk. Reconstructions involving the descending thoracic and thoracoabdominal aorta generally require circulatory support in the form of profound hypothermic circulatory arrest, partial femorofemoral bypass, or left heart bypass. These and other adjuncts have reduced the risk of paraplegia, the most dreaded of complication after thoracoabdominal aortic repair. Results have been demonstrated to vary with surgical experience; however, with these adjuncts in experienced hands, extensive repairs can be accomplished at acceptable operative risk.

The newest approaches to descending thoracic aortic disease involve the application of endovascular stent graft technology, now commercially available (Figure 3). Leveraging the experience obtained in treatment of infrarenal aortic aneurysms with similar technologies, covered stent grafts are being applied in various special circumstances in the descending thoracic aorta, including discrete degenerative aneurysms, anastomotic pseudoaneurysms, and traumatic disruptions. "Although such minimally invasive approaches have obvious appeal, the long-term durability of such repairs is still uncertain," says Timothy M. Sullivan, MD, a vascular surgeon at Mayo Clinic in Rochester. "Lifetime surveillance of the treated segment with serial CT imaging is required." Accordingly, younger patients with long life expectancy and those believed to have low operative risk may be better served with conventional repair. There are also anatomic limitations, including the location of branch vessels that would be occluded by such a covered graft, and the need to find relatively normal diameter aorta above and below the aneurysm into which the graft may be positioned. All patients with thoracic aortic aneurysms are evaluated for possible endovascular repair, and all available options are discussed thoroughly.

Mayo Clinic Testing Nonsurgical Treatment for Mitral Regurgitation

Device May Help Symptomatic Patients Too Sick for Surgery

Charanjit S. Rihal, MD

One of the consequences of congestive heart failure is enlargement and dilation of the left ventricle, which in turn may produce functional mitral regurgitation (MR) caused by stretching and distortion of the mitral annulus. This type of MR is unlike mitral valve disease secondary to for example rheumatic valvular disease, where thickening and distortion of the valve leaflets occur. The hemodynamic consequences, however, are similar. Forward stroke volume is reduced, further compromising already impaired cardiac output. With isometric exercise, the regurgitant volume further increases, and forward stroke volume is even further reduced. The volume load imposed on the left ventricle leads to further dilation and thus even more MR. About 60% of the 5 million patients with congestive heart failure in the United States have hemodynamically significant MR.

Studies have demonstrated the impact of the degree of functional MR on mortality (Figure 1). "Surgical mitral annuloplasty improves cardiac output, ejection fraction, and New York Heart Association class," says Charanjit S. Rihal, MD, director of the Cardiac Catheterization Laboratory at Mayo Clinic in Rochester. "But for some patients, the surgical risk is prohibitive, because of either severely impaired cardiac reserve or other comorbid conditions."

A new, percutaneously delivered device may allow clinicians to reshape and tighten the mitral annulus, thus reducing or eliminating functional MR without sternotomy or cardiac bypass. The device (Carillon Mitral Contour System, Cardiac Dimensions Inc, Kirkland, Washington) is currently undergoing clinical evaluation. Eight medical centers in the United States, including Mayo Clinic, are participating in clinical trials. Dr Rihal is principal investigator for the study at Mayo Clinic.

Device placement involves obtaining jugular access, positioning the delivery catheter within the coronary sinus, and delivering the device. After the device is placed in the coronary sinus, a distal anchor is set to hold the position. Tension is applied to tighten the annulus; a proximal anchor is then set to hold the device in position (Figure 2). Finally, the catheter delivery system is removed.

The clinical study will evaluate the safety of this system; secondary end points are long-term safety, efficacy, hemodynamic characteristics, and function of the device. Patient inclusion criteria are dilated isch-

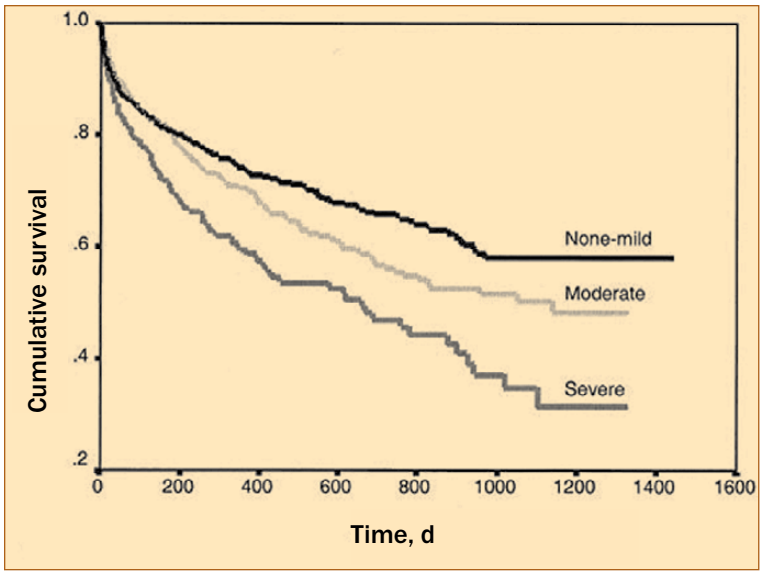


Figure 1. The impact of severity of mitral valve regurgitation on survival. *Reprinted with permission from the Koelling TM, et al. Am Heart J 2002;144(3):524-9.*

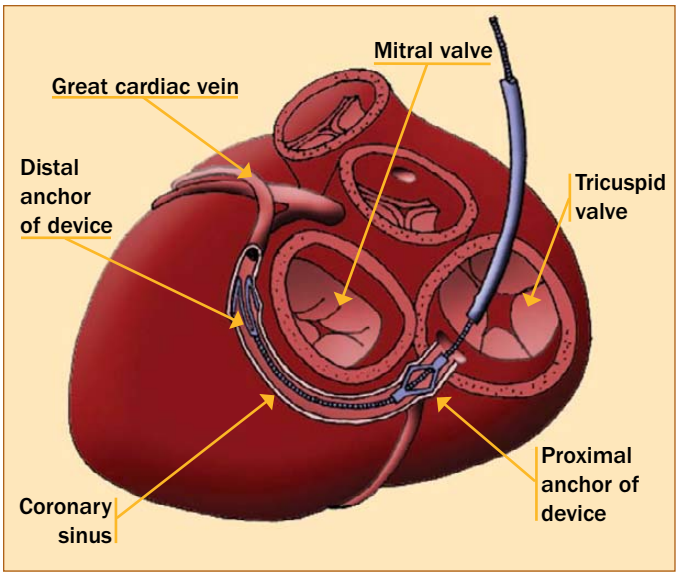


Figure 2. Diagram of deployed device in the coronary sinus. *Reprinted with permission from Carillon Mitral Contour System, Cardiac Dimensions Inc, Kirkland, Washington.*

emic or nonischemic cardiomyopathy, 3+ or 4+ functional MR, and left ventricular ejection fraction less than 40%, and the patient must be a surgical candidate in the event device-related complications occur. Exclusion criteria are recent myocardial infarction, foreign body (such as pacing lead) in the coronary sinus, chronic atrial fibrillation, and renal failure.

Safety considerations will be evaluated, including the effects of the device on coronary perfusion in adjacent coronary artery segments, tissue erosion, positional stability of the device, thrombogenicity, and device fatigue failures. Preclinical canine and swine studies suggest the device safely reduces the annulus diameter. "The inconsistent anatomy of the coronary sinus and the range of deformation seen in the mitral annulus in patients with dilated left ventricles indicate that this device will not be appropriate for every patient," says Dr Rihal. "However, we hope that the device will provide a therapeutic option for symptomatic patients for whom surgery is not an option."

Subsequent trials are anticipated to determine which patients and which conditions are optimal for the percutaneous approach to mitral repair. For further information regarding this trial, please see the *Cardiovascular Update* Web site, or contact the study coordinator at 507-255-4502.

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UNDER THE STETHOSCOPE

by Clarence Shub, MD

Mitral Regurgitation

- Systolic murmur of mitral regurgitation (MR) is usually pansystolic.
- Systolic murmur of severe chronic MR due to organic causes, eg, mitral prolapse, is usually loud (grade 3-4 or louder).
- Systolic murmur of functional MR, eg, due to left ventricular systolic dysfunction, is variable and may be unimpressive or even absent despite significant MR, especially in the presence of low cardiac output states.
- Apical S3 and/or mid diastolic flow murmur are specific, but insensitive signs of severe MR.
- Systolic murmur of "posterior mitral leaflet syndrome" can be well transmitted to the aortic area and confused with aortic stenosis; palpation of the carotid pulse helps to differentiate the two.
- The systolic murmur of "anterior mitral leaflet syndrome" is transmitted posteriorly and can be heard along the thoracic spine.

IN THE NEWS

Bariatric Surgery Is Safe and Effective for Obese Patients With Coronary Disease

Recent data from myocardial infarction surveillance studies suggest that obesity is the most prevalent cardiovascular risk factor in patients with myocardial infarction and that its prevalence in developed countries continues to increase. Joint guidelines of the American Heart Association/American College of Cardiology for the management of patients with coronary artery disease (CAD) recommend weight loss for patients with body mass index (BMI) higher than 25 kg/m^2.

The failure of conventional techniques to successfully treat severe obesity has sparked an interest in surgical strategies. Studies of unselected patients undergoing bariatric surgery have demonstrated marked and long-lasting weight loss, suggesting that this approach may be especially valuable in patients with CAD, particularly those with multiple cardiovascular risk factors. However, the safety and efficacy of bariatric surgery in patients with documented CAD have not been determined, even though these patients are the most likely to benefit from surgical weight reduction. Mayo Clinic Rochester cardiologist Francisco Lopez-Jimenez, MD, is the lead author of a study published in the September 2005 issue of ***Mayo Clinic Proceedings***, which assessed the perioperative risk for cardiovascular events (including mortality) in patients undergoing bariatric surgery and the impact of this procedure on cardiovascular risk factors.

The rate of major in-hospital cardiovascular complications and mortality was compared between 52 patients with and 507 without documented CAD. The efficacy of bariatric surgery was measured by changes in body weight and cardiovascular risk factors at follow-up.

No perioperative deaths occurred in either group. The rate of cardiovascular complications was relatively low in both groups and no different than in other types of abdominal surgery. Both groups of patients had statistically significant weight loss and improvements in cardiovascular risk factors such as hypertension, glycemic indices, and lipid profiles. Six patients had a positive preoperative stress test along with a postoperative stress test; 4 of these patients had a reduction in the extension or severity of stress-induced ischemia. "This report suggests that bariatric surgery is a safe alternative approach for weight loss in patients with severe obesity and a history of CAD, who have been unsuccessful in losing weight and maintaining this weight loss," says Dr Lopez-Jimenez. "Additionally, this study further demonstrates that bariatric surgery induces major improvement in several metabolic risk factors linked to cardiovascular disease."

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Jan 30-Feb 3, 2006, Big Island, Hawaii
Phone: 800-283-6296 or 507-266-0677; fax: 507-538-0146; e-mail:cvcme@mayo.edu

Cardiology at Cancun 2006: Integrating Advances in Imaging Into Clinical Practice (Echo, CMR, MSCT, PET)
Feb 27-Mar 3, 2006, Cancun, Mexico
Phone: 800-283-6296 or 507-266-0677; fax: 507-538-0146; e-mail:cvcme@mayo.edu

19th Annual State-of-the-Art Echocardiography
Feb 19-23, 2006, Phoenix, Ariz

Congenital Echo Workshop: Review of Common Congenital Cardiac Lesions in the Adult
Feb 21, 2006, Phoenix, Ariz

13th Annual Echocardiographic Workshop on 2-D and Doppler Echocardiography at Vail
Mar 6-9, 2006, Vail, Colo

3rd Annual Team Echocardiography: The Heart of Cardiovascular Medicine
Apr 20-23, 2006, Hilton Head Island, SC
Phone: 336-716-4505; fax: 336-716-2447; e-mail: cvcme@mayo.edu

American Society of Echocardiography 17th Annual Scientific Sessions
Jun 3-7, 2006, Baltimore, Md

American College of Cardiology Programs

To register or for information about programs, visit www.acc.org ("Programs") or call the ACC Resource Center at 800-253-4636, ext 694.

ACC.06: American College of Cardiology 55th Annual Scientific Session
Mar 11-14, 2006, Atlanta, Ga

Other Educational Opportunities

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

Heart Rhythm Society 27th Annual Meeting
May 17-20, 2006, Boston, Mass
Phone: 202-464-3400; Web site: www.HRSonline.org.

A. Jamil Tajik, MD, has been elected a fellow in the International Academy of Cardiovascular Sciences. Dr Tajik recently transferred from Rochester to Mayo Clinic Arizona.

Questions for *Cardiovascular Update*
Send us an e-mail, CVUpdate@mayo.edu

NEW FEATURE

This issue of *Cardiovascular Update* debuts a new feature,

UNDER THE STETHOSCOPE
by Clarence Shub, MD,

Mayo Clinic Rochester cardiologist. Dr Shub joined Mayo Clinic in 1978 and in his career has been a national leader in medical education at all levels of training. His activities range from introduction of the cardiac patient simulator ("Harvey") to development of computer-assisted medical teaching programs. He has received numerous teaching awards, including the Mayo Clinic Henry S. Plummer Distinguished Physician Award. Each issue Dr Shub will summarize key physical examination findings associated with one of the issue's featured topics. We hope you find **UNDER THE STETHOSCOPE** helpful in your clinical practice.

Mayo Clinic **Cardiovascular Update**

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