

CARDIOVASCULAR UPDATE

CLINICAL CARDIOLOGY AND CARDIOVASCULAR SURGERY NEWS



“Common Sense” Helps Patients Through Diet Maze



Gerald T. Gau, MD

The prevalence of obesity and type 2 diabetes mellitus in both children and adults is approaching epidemic proportions in the United States. At present, two thirds of US adults are overweight (body mass index >25 kg/m²), and 30% are frankly obese (BMI >30 kg/m²); 8% are diabetic, and 24% have the metabolic

syndrome. About 15% of children are also obese. “Data from the Centers for Disease Control and Prevention show that the number of deaths attributable to poor diet and physical inactivity rose 33% during the past decade, and these will soon overtake tobacco as the leading preventable cause for death,” according to Gerald T. Gau, MD, a cardiologist at the Mayo Clinic Cardiovascular Health Clinic in Rochester. “The economic cost to our nation is immense and growing daily.”

Countless diet books recommend ways to curb caloric intake and lose weight. At one extreme of “the diet pendulum” is the very low-fat diet promulgated by Dr Dean Ornish, and at the other is the potentially high-fat, carbohydrate-restricted diet developed by Dr Robert C. Atkins. The Ornish diet is a very low-fat vegetarian diet. With long-term adherence, this diet achieves weight loss, decreases cardiovascular events, and improves blood pressure and lipid profiles. However, the Ornish diet is difficult for people to follow, is too low in fat, and does not contain adequate essential fatty acids. Until recently, it excluded fish oils, but this has now been altered by Dr Ornish.

The Pritikin diet is similar in many ways to the Ornish diet but has a wider variety of foods, including fish and chicken. The Pritikin plan decreases weight, improves lipid profiles, and reduces blood pressure,

cardiac events, and strokes. The high-fiber content, with its resultant gas production, and rigidity of the diet are not well tolerated by many in the long term.

The Atkins diet is at the opposite extreme with high fat and protein intake and carbohydrate restriction. This diet, like the Ornish diet, has no caloric restriction. The weight loss early in the Atkins diet is caused by water loss and ketosis, with resultant decreased appetite and subsequent further decrease in caloric intake. Liver and muscle glycogen declines with carbohydrate restriction, and metabolism shifts to burning fat. Vitamin and mineral supplements are needed because of the scant intake of fruits and vegetables.

The possible cardiovascular benefits of the Atkins diet have been much debated in the medical literature. One long-term study compared the Atkins diet with low-fat diets over 1 year. The results demonstrated that, with the Atkins diet, homocysteine, C-reactive protein, and lipoprotein (a) values all increased. This study also showed that, with a high-fat diet, the LDL cholesterol and triglyceride levels increased, the HDL cholesterol level declined, and the ratio of total cholesterol to HDL cholesterol became abnormal. These results suggest that the Atkins diet is not beneficial in the long run. Initial weight loss is easier with this diet, but eventually weight returns to baseline as the diet is not easily sustained.

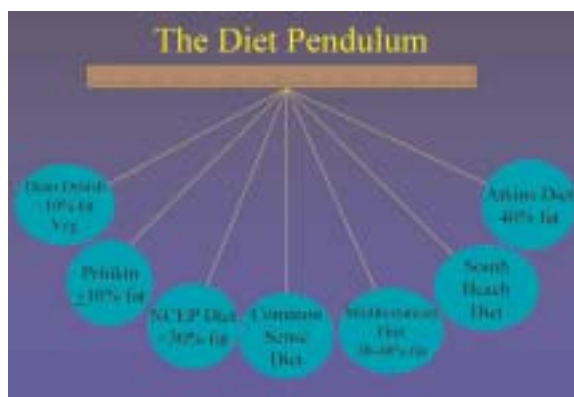
Other medical concerns relate to calciuria, renal stones, decreased bone mass, hepatic disease, and long-term increased atherogenicity. “We do not have a

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recommendation for the role of a modified carbohydrate-restricted Atkins diet for long-term weight reduction, and I worry about the long-term cardiovascular risk, as suggested by several published studies," says Dr Gau.

The South Beach diet is a carbohydrate-restricted diet, but unlike the Atkins diet, its content is not high fat and high protein. The diet is similar to the diet described by Dr Jean G. Dumesnil in Canada 20 years ago, and it is effective. When this progressive diet reaches its maintenance program, it is similar to the better-tasting Mediterranean diet. The South Beach diet leads to weight loss and does help people learn how to control their energy intake.

The most rational diet is the American Heart Association (AHA) "common sense" diet, in the middle of the dietary pendulum. This diet is calorie restricted with an important exercise component. The common sense diet includes fruits, vegetables, and complex carbohydrates in limited quantities, with small portions of lean meat, chicken, and fish.

A perfusion of data has been published regarding the Mediterranean diet and the use of omega-3 fatty acids, as well as the common sense and National Cholesterol Education Program (NCEP) diets. The AHA has recommended adding omega-3 fatty acids in the form of fish oil capsules to the daily regimens of all patients with documented heart disease and those at high risk. Olive oil and canola oil are the recommended cooking oils.

This Mediterranean diet plan has produced a striking decrease in cardiovascular risk with decreased cardiovascular mortality and sudden cardiovascular

death. A recent study of the diet of the Greek people demonstrated that the cardiovascular benefit closely followed the degree of adherence to this diet. "My own strong preference is the Mediterranean diet because of the palatability and effectiveness of this diet in long-term weight control and cardiovascular disease protection," says Dr Gau.

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Dr Gau's Diet Conclusions

- Ultra-low-fat diets (Ornish and Pritikin) are poorly tolerated.
- The NCEP Step I diet is largely ineffective.
- The NCEP Step II diet, which is low fat, has features of the Mediterranean diet, and decreases saturated fat with more monounsaturated and omega-3 fatty acids, is effective.
- The Atkins diet affords weight loss with some risk, but it is not useful in the long term.
- The common sense diet works.
- The carbohydrate-restricted South Beach diet works for many and adopts a Mediterranean-type diet in the weight maintenance phase.
- The Mediterranean diet is ideal; it is better tasting and is proven heart protective with increased longevity.
- Diet without exercise does not achieve the goal of healthy weight loss.



Iftikhar J. Kullo, MD

Early Atherosclerosis Clinic Uses Novel Markers for Finer Coronary Risk Assessment

Despite remarkable advances in treatment, coronary heart disease (CHD) remains the leading cause of death in the United States. Patients may experience CHD events without warning. Many patients who would be considered low risk on the basis of conventional methods of risk stratification experience events. "Clearly, improved risk stratification could have a major effect on CHD burden by identifying patients who need aggressive preventive measures," says Iftikhar Kullo, MD, director of the Mayo Clinic Early Atherosclerosis Clinic in Rochester.

Conventional risk factors classify patients generally as being at low, intermediate, or high risk for CHD. However, such an approach lacks precision. Although most patients who have a CHD event have 1 or more of

the conventional risk factors, so do many patients who are asymptomatic. Moreover, risk stratification using conventional risk factors does not take into account family history of CHD, obesity, elevated triglycerides, and fasting hyperglycemia.

The mission of the Mayo Clinic Early Atherosclerosis Clinic is to perform comprehensive risk profiling for patients with early-onset CHD or a family history of early-onset CHD (Figure 1).

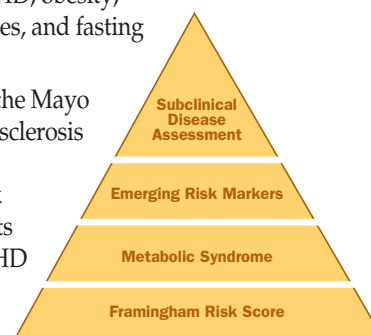


Figure 1. Risk prediction pyramid.

“In these patients, conventional risk algorithms do not perform well in explaining or predicting CHD events, and other ‘nonconventional’ risk factors may play a role,” says Dr Kullo.

Each patient seen in the Early Atherosclerosis Clinic undergoes comprehensive CHD risk profiling, including estimation of the 10-year probability of CHD. An individualized treatment plan is then developed in response to findings on the clinical evaluation and expanded testing:

- The 10-year probability of CHD is estimated using an equation derived from the Framingham Study. (Algorithm score sheets are available at www.nhlbi.nih.gov/about/framingham/riskabs.htm.)
- The metabolic syndrome is diagnosed when 3 of 5 criteria are met (Table 1). The metabolic syndrome is associated with doubling the CHD risk, and it appears to be an important risk factor for early-onset CHD.
- The conditional risk factors measured include homocysteine, fibrinogen, lipoprotein (a), LDL particle size, and C-reactive protein (Table 2). These new risk factors are referred to as “conditional” risk factors because elevated levels of these analytes may enhance the likelihood of a cardiovascular event when found in the presence of conventional risk factors. In most patients who develop CHD at a young age and in many with a family history of CHD, 1 or more of these factors is elevated, although it is not clear if they are causative agents or biomarkers of disease.
- Coronary artery calcium is measured by electron beam computed tomography in patients with family history of early-onset CHD (Figure 2). Presence of excess coronary calcium (based on age and sex) mandates aggressive treatment of risk factors.

The information from comprehensive risk-factor profiling is helpful in making decisions regarding treatment of conventional risk factors, particularly about initiating statin therapy and determining target LDL cholesterol levels. Treatment of conditional risk factors can also be considered, such as niacin for elevated

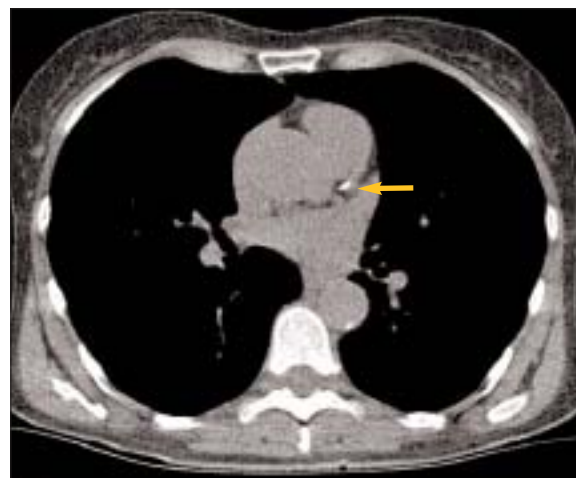


Figure 2. Coronary artery calcium scan showing coronary artery calcium (arrow) in an asymptomatic 54-year-old man with a family history of CHD.

lipoprotein (a) and folic acid for hyperhomocysteinemia. Delineation of these risk factors may also help increase patients’ motivation to make lifestyle changes.

“For example, treatment with a statin can be initiated in asymptomatic subjects on the basis of their family history, presence of excess coronary artery calcium, or abnormal levels of 1 or more conditional risk factors,” says Dr Kullo. “Statins reduce CHD risk, and treatment targeted to those asymptomatic patients who are at increased risk is likely to be cost-effective.”

Patients most likely to benefit from consultation in the Early Atherosclerosis Clinic are those with a family history of early-onset CHD and those aged 55 years or younger who have a history of CHD (including myocardial infarction, angina, or percutaneous coronary intervention) or peripheral atherosclerotic vascular disease.

Patients for whom consultation is less suitable are those older than 55 years and those already considered to be at high risk because of the presence of multiple conventional risk factors.

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**TABLE 1
 Risk Factors and Defining Levels for the Metabolic Syndrome**

Abdominal obesity (waist circumference)
Men >40 inches
Women >35 inches
Triglycerides ≥150 mg/dL
HDL cholesterol
Men <40 mg/dL
Women <50 mg/dL
Blood pressure ≥130/≥85 mm Hg
Fasting glucose ≥110 mg/dL

**TABLE 2
 Comparison of Risk Factors for CHD Screening**

Conventional risk factors	Conditional risk factors	Predisposing conditions
Cigarette smoking	Elevated homocysteine	Overweight/obesity
Elevated blood pressure	Elevated fibrinogen	Physical inactivity
Elevated serum cholesterol	Elevated lipoprotein (a)	Family history of early-onset CHD
Low HDL cholesterol	Small LDL particle size	Socioeconomic factors
Diabetes mellitus	Elevated C-reactive protein	Behavioral factors
	Elevated triglycerides	Insulin resistance

Early, Accurate Diagnosis of Acute Aortic Syndromes Guides Treatment

Surgery, Stents, Medication Appropriate Based on Nature, Location of Lesions



Thoralf M. Sundt III, MD
Peter C. Spittell, MD

Aortic dissection is 1 of 3 related life-threatening entities to which the phrase “acute aortic syndrome” is collectively applied. This spectrum of disease comprises classic aortic dissection, intramural hematoma (IMH), and penetrating atherosclerotic ulcer (PAU).

Advances in imaging technology that permit remarkable resolution have revealed this spectrum of disease where previously only acute dissection was recognized. “All are potentially rapidly lethal conditions for which the risks of surgical intervention are also considerable,” according to Thoralf M. Sundt III, MD, a cardiovascular surgeon at Mayo Clinic in Rochester. “Current data suggest, however, that the natural history of each may differ to some degree, making their accurate diagnosis clinically relevant.”

Classic Aortic Dissection

Classic aortic dissection is associated with degenerative changes of the media, commonly termed “cystic medial degeneration” (Figure 1); this condition may occur secondary to chronic hypertension, genetic factors (such as Marfan syndrome and familial syndromes of aneurysm and

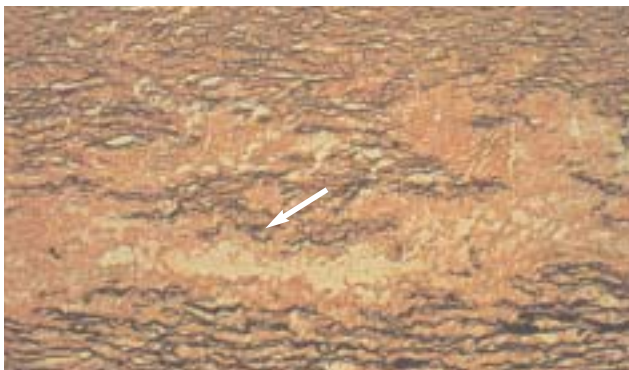


Figure 1. Cystic medial necrosis (arrow) of aortic wall.

dissection), or the interplay of both. Bicuspid aortic valve disease is also associated with an increased risk of aneurysm and dissection. Intriguingly, this risk is independent of the functional performance of

the bicuspid valve and may reflect an inherent abnormality of the aortic wall. Indeed, even after successful aortic valve replacement, patients with a history of bicuspid aortic valve remain at increased risk of dissection. Dissection may occur as a complication of pregnancy and may be associated with coarctation of the aorta.

“The classic presentation of acute aortic dissection is the sudden onset of severe pain (often migratory) in the anterior chest, back, or abdomen (70% to 80% of patients) and hypertension (60% to 80% of patients),” says Mayo Clinic cardiologist Peter C. Spittell, MD. Dissections

involving the ascending aorta may cause acute aortic regurgitation, coronary malperfusion, or pericardial tamponade. Findings on physical examination may include an aortic murmur, pulse deficits, and neurologic changes. Syncope in association with aortic dissection occurs when rupture into the pericardial space occurs, producing cardiac tamponade. Congestive heart failure is usually attributable to severe aortic regurgitation. Acute myocardial infarction (most commonly inferior infarction caused by right coronary artery ostial dissection) and pericarditis are additional cardiac presentations.

Natural history studies have suggested that approximately 40% of those experiencing acute dissection die immediately, and 70% of the remainder succumb to the disease within the first 24 hours (a mortality rate of 1% to 3% per hour). The diagnosis of dissection is most commonly made by computed tomography, although transesophageal echocardiography and magnetic resonance imaging play an important role today as well. “Aortography, the ‘gold standard’ just a decade ago, has been relegated to a minor role today by these noninvasive techniques,” says Dr Spittell.

The diagnosis of dissection can only be made, however, if aortic dissection is included in the differential diagnosis of any patient with sudden-onset chest pain. In a patient with a catastrophic presentation, a history of systemic hypertension, and unexplained physical findings of vascular origin (especially in the presence of chest or back pain and an aortic murmur), aortic dissection should always be included in the differential diagnosis, and an appropriate screening test should be performed emergently.

Aortic dissections may be classified according to either the DeBakey scheme or the simpler and more popular Stanford classification (Figure 2). The immediate management of aortic dissection is β -blockade and additional antihypertensive control as required. Acute dissections involving the ascending aorta (Stanford type A) should be considered for emergent surgery. “Although the in-hospital mortality rate associated with surgical repair of type A dissection is approximately 20% to 25%, that of nonoperative treatment is roughly twice that,” says Dr Sundt. “Consequently, the standard of care for type A dissection is surgical in most cases.”

Patients with acute type B dissection involving the descending thoracic aorta are, in most centers, treated medically with aggressive antihypertensive therapy. Surgical intervention is reserved for those experiencing refractory or recurrent pain, active leak, or a malperfusion syndrome. The in-hospital mortality rate for nonoperative

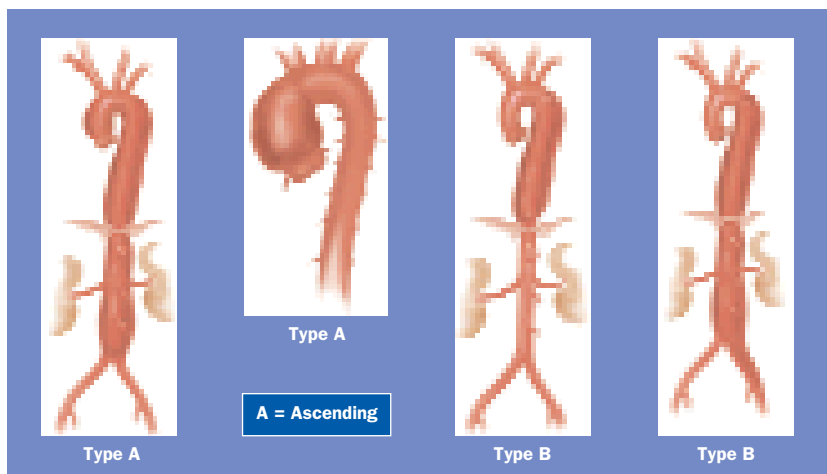


Figure 2. Stanford classification of aortic dissection. Type A dissections involve the aortic root and arch.

treatment is approximately 10% and that for surgery is 3 times higher. In the current era of endovascular interventions, complications of type B aortic dissection can often be treated with catheter fenestration and stenting of distal vessels to correct malperfusion. When long-term pharmacologic therapy is used for type B aortic dissection, indications for surgery include development of saccular aneurysm, increasing aortic diameter, or symptoms related to chronic dissection. The use of stent grafts in descending thoracic aortic dissection is in its infancy and cannot be considered the standard of care.

The cornerstone of surgical repair of type A dissection is graft replacement of the ascending aorta. This is most often performed using profound hypothermia and circulatory arrest. Accordingly, the risks of renal dysfunction and respiratory failure are real, especially among the elderly. Aortic regurgitation caused by proximal extension of the dissection is frequently repaired, although root replacement may be required and is recommended in patients with Marfan syndrome or other defined connective tissue abnormalities. The distal extent of repair may be at the level of the innominate artery or may include the underside of the aortic arch (“hemi-arch replacement”). Surgical repair of type B dissection is also most often performed with circulatory arrest and entails replacement of the upper portion of the thoracic aorta and a variable amount of the distal descending thoracic aorta.

Intramural Hematoma

The second entity in the spectrum of disease is IMH, long considered a forme fruste of aortic dissection, representing about 5% of acute aortic syndromes. IMH is thought to be caused by rupture of the vasa vasora in the presence of cystic medial necrosis, although this theory remains unproven. The presenting signs and symptoms of IMH are identical to those of dissection, although malperfusion does not typically complicate

this condition. The diagnosis is most readily made by CT scanning (Figure 3). IMH typically has thickening of the wall in the entire circumference of the aorta with no intraluminal flap, as may be seen in dissection. Displacement of intimal calcification from the outer wall, if present, is a helpful clue.

The natural history of IMH is a matter of some debate. Most specialists in aortic disease recommend a treatment algorithm identical to that for acute dissection, with IMH type A treated surgically and type B treated medically. Recent studies suggest, however, that IMH type A with little dilation of the ascending aorta may be managed expectantly. This view is being debated, and at this time, surgical referral for evaluation is certainly appropriate. It is clear that long-term β -blocker therapy has a profoundly salutary effect on these individuals.

Penetrating Atherosclerotic Ulcer

PAU has been recognized with increasing frequency in the last quarter-century as a distinct disease process. Such ulcers occur in patients with advanced atherosclerotic disease of the thoracic aorta when an atherosclerotic plaque undergoes ulceration and penetrates the internal elastic lamina. “PAU typically occurs in the very elderly and, in contrast to dissection, which occurs most often in the ascending aorta, usually arises in the descending thoracic aorta,” says Dr Spittell.

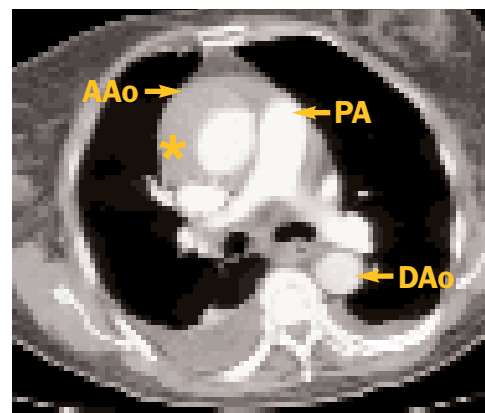


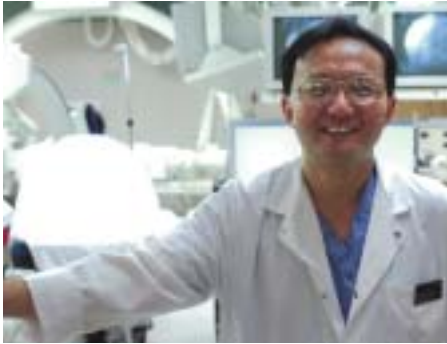
Figure 3. Thoracic CT scan of type A IMH. The asterisk indicates hemorrhage into the aortic wall. AAo, ascending aorta; DAo, descending aorta; PA, pulmonary artery.

The management of PAU is even more controversial than that for IMH. All agree that patients with PAU are at high risk and that long-term survival is poor. One school of thought concludes that an aggressive surgical approach should be undertaken. “The alternate school, of which we at Mayo Clinic are proponents, argues that the operative risks are sufficiently high that a trial of medical therapy with antihypertensives is preferable as a first step,” says Dr Sundt. Late mortality appears to be more a reflection of the comorbid conditions in these patients than the aggressiveness of the lesion itself.

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Inducible Arrhythmias May Cue for Aggressive WPW Treatment



Win-Kuang Shen, MD

Wolff-Parkinson-White (WPW) syndrome was first described by Drs Louis Wolff, John Parkinson, and Paul Dudley White in 1930. It was characterized as a potential cause of supraventricular tachyarrhythmias and, rarely, ventricular fibrillation (VF) and sudden cardiac death (SCD).

Approximately 2 per 1,000 persons in the general population are affected, but not all develop arrhythmias.

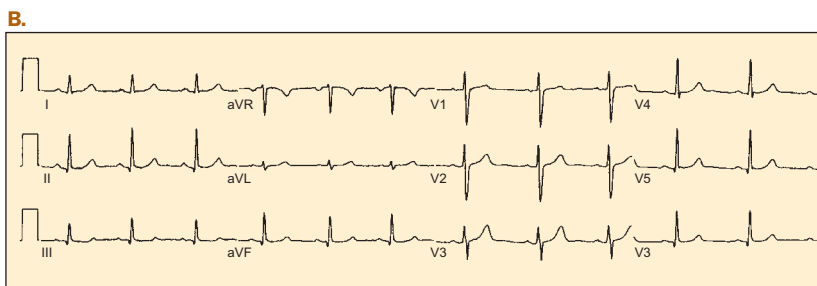
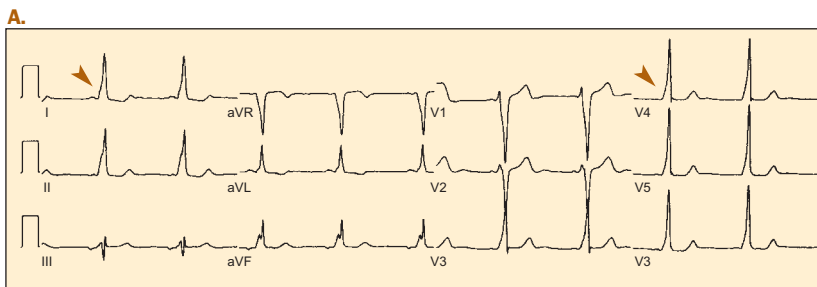
“Predicting which patients are at risk for SCD is one of the most difficult aspects of WPW syndrome,” according to Win-Kuang Shen, MD, of the Mayo Clinic Heart Rhythm Service. “Patients with symptomatic arrhythmias or characteristics that place them at high risk for SCD should be offered radiofrequency ablation (RFA).”

Attempts have been made to determine risk factors associated with SCD. Noninvasive markers such as intermittent loss of preexcitation on electrocardiography (ECG), sudden loss of preexcitation during exercise, or loss of preexcitation after class I antiarrhythmic drugs have been associated with poor conduction over the accessory pathway (AP) and low risk of SCD. A short preexcited R-R interval (<250 ms) during sustained atrial fibrillation (AF) induced by electrophysiologic (EP) study has been found universally in patients who were resuscitated from SCD and therefore is suggested to be a marker of increased risk of SCD. All markers are limited by their lack of positive predictive values because of low event rates in asymptomatic patients. Guidelines suggested that a routine invasive approach is not recommended in asymptomatic patients with WPW syndrome.

For example, the ECG in Figure 1A is from an 18-year-old woman who was evaluated for frequent palpitations during exercise. Her symptoms were nonparoxysmal and suggested sinus tachycardia. All subsequent rhythm strips from an event monitor showed sinus tachycardia. An extensive review of goals and risks of therapeutic options for asymptomatic WPW syndrome (including the increased risk of heart block during RFA because of the presumed anteroseptal location of the AP) led to the decision not to treat but to observe her condition.

Several years later, the patient complained of recurrent palpitations. An event recorder transmitted strips that indicated sinus tachycardia. When she returned for a consultation, a repeat ECG (Figure 1B) showed that preexcitation was no longer present. “She was reassured that her condition was not life-threatening, and elective evaluation for sinus tachycardia and possible orthostatic intolerance was recommended if her symptoms persisted,” says Dr Shen. The loss of preexcitation on subsequent ECGs was 17% in a study of WPW patients in Olmsted County, Minnesota. The patient described here raises a number of questions about WPW syndrome.

Figure 1. 12-lead ECGs from an 18-year-old patient with WPW syndrome. A, PR, 80 ms; QRS, 160 ms. Arrows indicates delta (preexcitation) waves. B, PR, 140 ms; QRS, 90 ms. The delta wave is no longer present.



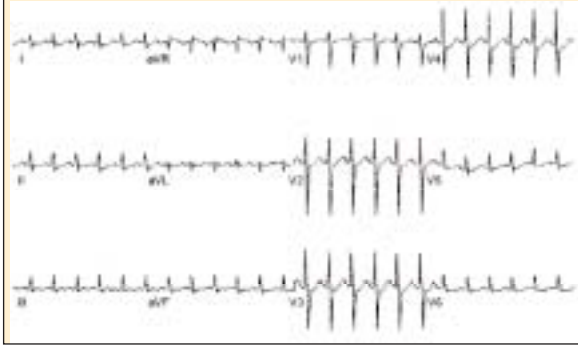
What are the rhythm disturbances and associated concerns in patients with WPW syndrome?

Three types of rhythm disturbances are seen most frequently in patients with WPW syndrome. *Orthodromic reentrant tachycardia* (Figure 2A) uses the normal conduction pathway (atrioventricular node and His-Purkinje conduction) in the anterograde direction and AP in the retrograde direction. *Antidromic reentrant tachycardia* (Figure 2B) uses the AP in the anterograde direction and the normal conduction pathway in the retrograde direction. *AF with variable atrioventricular conduction* (manifested by variable preexcited QRS complexes) is shown in Figure 2C. Degeneration of atrioventricular reentrant tachycardia into AF with rapid ventricular response and then VF is believed to be the mechanism underlying SCD in patients with WPW syndrome.

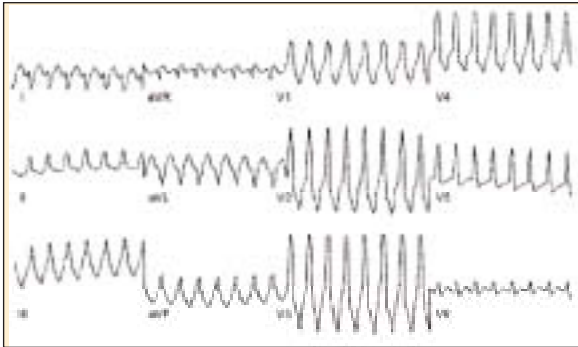
What is the best approach to treat patients with symptomatic WPW syndrome?

In multicenter studies completed in the mid 1990s, the overall success rate for RFA ranged from 90% to 93% and was highest when the AP was located in the left free wall ($\geq 95\%$) and lowest when the AP was located in the right free wall or posteroseptum (85%-90%). The success rates should be even higher today because mapping, catheters, and RFA delivery systems continue to improve. Risk of death has been estimated to be about 0.1%. Other major complications associated with RFA include

A.



B.



C.

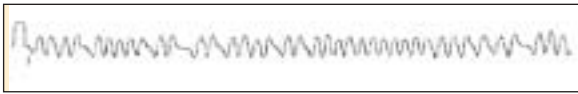


Figure 2. 12-lead ECGs. A, Orthodromic reentrant tachycardia. B, Antidromic reentrant tachycardia. C, Rhythm strip of AF with varying degrees of preexcitation.

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perforation or tamponade, pneumothorax, stroke, myocardial infarction, and heart block. The overall major complication rate for RFA has been estimated in the range of 1.8% to 4.4%. The recurrence rate (after initial successful ablation) of AP conduction is around 5%.

At Mayo Clinic in Rochester, 369 RFA procedures for APs were performed between 1997 and 2003; approximately 25% of these patients were referred to Mayo Clinic because of previously failed ablation attempts. The immediate success rate was 94%, with 1.2% major complications; no death has occurred.

According to Dr Shen, RFA is considered the treatment of choice in the following patients: 1) symptomatic patients with documented tachyarrhythmias; 2) patients who cannot tolerate or do not wish to take medications for symptomatic tachyarrhythmias; and 3) patients with high-risk occupations such as airline pilots, school bus drivers, and firefighters. A thorough review of the merits and concerns of pharmacologic and ablative therapy should be provided to the patient. The latest practice guidelines were published in 2003 (www.americanheart.org/downloadable/heart/1062186010820SVAFullTextGLfinal.pdf).

What is the latest round of debate on the approach to asymptomatic WPW syndrome?

Although the majority of patients who experience SCD have had previous episodes of symptoms or documented tachycardia, several studies have reported that SCD could be the first presenting symptom. Among highly selected patients with WPW syndrome who were resuscitated from SCD, up to 50% reported no prior symptoms. Among asymptomatic patients from population-based studies, up to 21% may develop atrioventricular reentrant tachycardia or AF during follow-up; the actual risk of SCD has been estimated up to 0.4% annually.

The “observational” approach in patients with asymptomatic WPW syndrome was questioned in 2

recent studies. The first study reported that 115 of 162 asymptomatic patients had no inducible arrhythmias during EP study; symptomatic arrhythmias developed in only 4 of these 115 patients during a mean follow-up of 38 months. Among 47 of 162 patients with inducible arrhythmias, 29 developed symptomatic arrhythmias, including 2 who were resuscitated after cardiac arrest and 1 who had SCD. Younger patients (<35 years), shorter AP anterograde refractory periods, and the presence of multiple APs were predictors of subsequent development of symptomatic arrhythmias.

In the second report, asymptomatic patients with inducible arrhythmias were randomly assigned to receive ablation (37 patients) versus observation (35 patients). The 5-year Kaplan-Meier estimates of the incidence of arrhythmia events were 7% versus 77% ($P<.001$). One patient from the control group who had VF arrest was resuscitated successfully.

Combining the results from these 2 studies, all 4 patients who experienced VF arrest were less than 30 years old and had multiple APs, inducible atrioventricular reentrant tachycardia triggering AF, and short preexcited PR interval during EP study. One of the 4 patients had VF arrest as the presenting symptom. The investigators suggested that “prophylactic” ablation should be considered in selected asymptomatic patients with WPW syndrome.

Conclusions

These recent data have renewed interest in and debate on how best to approach asymptomatic patients with WPW syndrome. According to Dr Shen, the new data suggest that 1) approximately a third of asymptomatic patients have inducible arrhythmias during EP study; 2) the risk of noninducible patients’ developing symptomatic arrhythmias (approximately 4%) or SCD (0%) is very low; 3) the risk of inducible patients’ developing symptomatic arrhythmias is high (77% in 5 years); 4) VF arrest occurred in 4 of 82 patients who had inducible arrhythmias; and 5) variables that predict development of symptomatic arrhythmias (including VF arrest) are younger age, shorter AP anterograde refractory periods, multiple APs, and inducible atrioventricular reentrant tachycardia or AF.

The most recent practice guidelines suggest that EP study and RFA in asymptomatic patients with WPW syndrome is a class IIa indication with level B evidence. “Physicians could consider a strategy of offering a diagnostic EP study to asymptomatic patients younger than 30 years old; ‘prophylactic’ ablation for prevention of future symptomatic arrhythmias and possible VF arrest can be considered if any of the predictors of developing symptomatic arrhythmias are present,” says Dr Shen.

Upcoming Courses

CONTINUING MEDICAL EDUCATION, MAYO CLINIC

Internal Medicine Review for Nurse Practitioners and Physician Assistants
 Sep 16-17, 2004, Rochester, Minn
 Info: 800-323-2688, 507-284-2509, cme@mayo.edu

Perspectives in Women's Health: Sleep Disorders in Women [interactive satellite program]
 Sep 23, 2004, Rochester, Minn
 Info: 866-869-4069, womenshealth@mayo.edu, www.mayo.edu/womenshealth

Echocardiography for the Sonographer
 Sep 26-28, 2004, Rochester, Minn
 Info: 800-288-6296, 507-266-0677, cvcme@mayo.edu



Mandeep Singh, MD, recipient of 2004 American College of Cardiology William W. Parmley Young Author Achievement Award. Dr Singh and his colleagues validated a system of clinical variables that predict before angiography the risk of future cardiac events.



Michael D. McGoon, MD, received the Pulmonary Hypertension Association Outstanding Physician Award at the Sixth International Pulmonary Hypertension Conference.



Completing the cardiothoracic surgery fellowship in July 2004 are **Juan A. Crestanello, MD (left)**, and **Subrato J. Deb (right)**. **Joseph A. Dearani, MD (center)**, is the program director. Dr Crestanello has joined the Department of Surgery at Ohio State University and Dr Deb is now located at the National Naval Medical Center, Bethesda, Maryland.

9th Annual Mayo Cardiovascular Review Course for Cardiology Boards and Recertification

Oct 2-7, 2004, Rochester, Minn
 Info: cme@mayo.edu, www.mayo.edu/cardiologyreview

20th Annual Echocardiography in Pediatric and Adult Congenital Heart Disease

Oct 3-6, 2004, Rochester, Minn
 Info: 507-266-6703, 507-284-0536, echocme@mayo.edu

7th Annual Mayo Clinic Internal Medicine Update: Sedona 2004

Oct 7-10 and Oct 21-24, 2004, Sedona, Ariz
 Info: 480-301-4580; mcs.cme@mayo.edu

AMERICAN COLLEGE OF CARDIOLOGY PROGRAMS

To register or for information about programs, visit www.acc.org or call the ACC Resource Center at 800-253-4636, ext 694. Outside the United States and Canada, call 301-897-2694 or fax request to 301-897-9745.

Cases in Echocardiography: TEE, Doppler, and Stress: Interpretation and Clinical Decision Making for the Advanced Echocardiographer

Oct 28-30, 2004, Seattle, Wash
 Directed by: Rick A. Nishimura, MD, FACC; Fletcher A. Miller, Jr, MD, FACC

Echo Hawaii 2005

Jan 24-28, 2005, Kohala Coast, Hawaii
 Directed by: A. Jamil Tajik, MD, FACC; James B. Seward, MD, FACC

Cardiology at Cancun

Feb 14-18, 2005, Cancun, Mexico
 Directed by: A. Jamil Tajik, MD, FACC; Guy S. Reeder, MD, FACC



OTHER EDUCATION OPPORTUNITIES

Nutrition in Health and Disease

Sep 23-24, 2004, St Paul, Minn
 Info: 800-323-2688, 507-284-2509, cme@mayo.edu

Genomics in Clinical Practice: What You and Your Patients Need to Know

Oct 11-12, 2004, Rochester, Minn
 Info: 800-323-2688, 507-284-2509, cme@mayo.edu

Heart Rhythm Society Advanced Ablation Course

Oct 18-20, 2004, Chicago, Ill
 Info: 508-647-0100, www.naspe.org/aac04, info@naspe.org

Mayo Clinic Nicotine Dependence Conference

Oct 24-27, 2004, Rochester, Minn
 Info: 800-323-2688, 507-284-2509, cme@mayo.edu

Women's Health 2004

Oct 28-30, 2004, Phoenix, Ariz
 Info: 480-301-4580; mcs.cme@mayo.edu

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