

Update in Nonpulmonary Critical Care

The Rapidly Changing Management of Cardiac Arrhythmias

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The management of cardiac arrhythmias has changed dramatically over the past decade with improved understanding of arrhythmia mechanisms and application of revolutionary therapies, including radiofrequency ablation and the implantable cardioverter defibrillator (ICD). The rapid advancement in arrhythmia management has been fueled by evidence-based medicine, which helps to clarify optimal approaches to management. This review will touch on the highlights of recent changes in our understanding of arrhythmia mechanisms and management.

ATRIAL TACHYARRHYTHMIAS

Atrial Fibrillation

Atrial fibrillation (AF) affects more than 1 million Americans, resulting in 75,000 cases of stroke each year. The incidence of AF increases with age with patients older than 65 years, having a 2–5% annual rate of developing AF (1). Controlled clinical trials (2, 3) have identified clinical risk factors for developing emboli in the presence of AF: (1) history of prior emboli, (2) hypertension, (3) history of congestive heart failure, (4) women over age 75, and (5) diabetes. In addition, two risk factors are identified by echocardiography: (1) left ventricular dysfunction ($EF < 40\%$), and (2) left atrial size greater than 2.5 cm^2 . Patients with no risk factors have a 1% embolic rate per year, those with one or two risk factors have a 6% embolic rate per year, and those with three or more risk factors have a 19% embolic rate per year. The following conclusions can be drawn concerning antithrombotic therapy: (1) patients without risk factors should receive aspirin 325 mg/d and (2) patients with one or more risk factors (including age > 75) should be anticoagulated with warfarin. Warfarin should be dosed to raise the INR (international normalized ratio) to values between 2 and 3. INR levels below 1.8 do not protect patients from thromboembolic complications.

When and if to cardiovert. The decision to cardiovert patients with AF should be individualized based on hemodynamic status and symptoms. Cardioversion is successful in 70–85% of patients, but AF recurs in 50–75% of the cases depending on antiarrhythmic therapy. The success of cardioversion is probably improved if the left atrial size is less than 60 mm in diameter, although data are conflicting (1). The cardioversion-related risk of embolization in patients who have been in AF for longer than 48 h is approximately 5.5%, but this risk falls to less than 1% if patients are anticoagulated according to the recommended guidelines. Since atrial flutter also predisposes patients to thromboembolic complications from cardioversion,

the same anticoagulation management is recommended for patients with atrial flutter (4).

Controlled trials have demonstrated that the success rate of transthoracic cardioversion for AF is enhanced by ibutilide (5). Pretreatment with ibutilide (1 gm IV) reduced the energy for cardioversion from $228 \pm 93 \text{ J}$ to $166 \pm 80 \text{ J}$ ($p < 0.001$) and improved the success rate from 72% to 100% compared to placebo. All patients who failed cardioversion while receiving placebo were successfully cardioverted after treatment with ibutilide. Ibutilide is a class III potassium channel blocker that must be used with caution in patients with severe systolic dysfunction and should not be used in patients with prolonged QT intervals. Because approximately 3% of patients who receive ibutilide develop torsades de pointes, they should be monitored for 4 h following administration of the drug.

On the basis of the results of prospective randomized clinical trials the following recommendations can be made regarding anticoagulation in conjunction with cardioversion (1) assuming that the patient is hemodynamically stable: (1) no anticoagulation is required if AF has been present for less than 48 h; (2) patients who had been in AF for longer than 48 h should receive anticoagulants for 3 wk before and at least 4 wk after cardioversion. The role for transesophageal echocardiography in establishing embolism risk and guiding anticoagulation decisions related to cardioversion has yet to be firmly established.

Maintaining sinus rhythm. The benefits of maintaining normal sinus rhythm include relief of symptoms, decreased risk of emboli, and decreased risk of warfarin-related complications. However, it is unclear if helping to maintain sinus rhythm improves survival. This is the focus of the AFFIRM (6) trial in which patients are randomized to anticoagulation plus maintenance of sinus rhythm with antiarrhythmic drugs versus anticoagulation and rate control alone. The results of the trial will be available in 2001. Antiarrhythmic drugs are effective in maintaining sinus rhythm for 1 yr in 50–65% of patients (1); amiodarone is the most effective of these. Unfortunately, antiarrhythmic drugs are also proarrhythmogenic. These drugs trigger life-threatening ventricular arrhythmias in 1 to 3.5% of patients with normal hearts and in up to 15% of patients with reduced ventricular function and/or a history of ventricular arrhythmias.

An alternative to antiarrhythmic drug therapy for maintaining sinus rhythm is the surgical Maze procedure (7). This procedure involves the ligation of the appendage of both atria followed by the placement of incisions in locations that prevent the development of AF. The procedure is indicated in patients who fail to improve on medications and have intolerable symptoms. It is an open-heart surgical technique with an 80–90% chance of success and a 2% risk of complications including needing a pacemaker. Trials are ongoing to evaluate the efficacy of a number of pacing techniques designed to maintain a normal sinus rhythm. These include pacing in two atrial

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locations, antitachycardia pacing, and low-energy cardioversion of the atria.

Recent reports (8) have indicated that the onset of AF may be caused by premature atrial complexes arising in one of the pulmonary veins. Atrial tissue may extend one or more centimeters into the vein and can give rise to ectopic atrial beats that can initiate sustained episodes of AF. This mechanism may account for 40% of AF episodes in patients with normal hearts and should be suspected when AF is consistently initiated by a premature atrial complex of one morphology. Unless normal sinus rhythm is reestablished early by cardioversion or by pharmacologic means the atrial myocardium remodels, promoting the development of chronic AF. Ablation of atrial tissue in pulmonary veins requires placing one or more catheters in the left atrium using a transseptal approach. At present this approach is successful in 30% of patients with venous foci and restores efficacy of antiarrhythmic therapy in an additional 30% of patients whose AF had been refractory to drug treatment before the ablation (8).

Rate control. Attempts to keep the resting heart rate below 90 and the heart rate below 120 during a brisk walk are extremely important in preventing tachycardia-induced cardiomyopathy (1). In a recently published controlled clinical trial (9) the combination of digoxin plus atenolol was most effective in controlling heart rate of patients with rapid AF. Lesser rate control was achieved with digoxin plus diltiazem, atenolol alone, diltiazem alone, while digoxin alone was least effective.

Ablation of the atrioventricular (AV) conduction system and the placement of a permanent pacemaker is indicated for patients with AF in whom AV nodal-blocking drugs are ineffective or produce unacceptable side effects (10). The success rate of radiofrequency ablation is 98%. Long-term anticoagulation is usually required since the atria will continue to fibrillate. Eighty-five percent of patients report an improved quality of life and increased exercise performance following AV nodal ablation.

Atrial fibrillation after cardiac and thoracic surgery. Postoperative AF increases the risk for stroke, delays hospital discharge, prolongs ICU stay, and increases hospital costs (11). Ibutilide has been demonstrated to successfully terminate AF in 50% of patients following cardiac surgery whereas placebo converted 20% of patients. It is interesting to note that torsades de pointes occurred in 1.8% of patients receiving ibutilide and in 1.2% of patients receiving placebo (12). A metaanalysis (13) showed that beta blockers reduce the occurrence of AF whereas verapamil and digoxin had no effect. Beta blockers should be continued in the postoperative period to help reduce the risk of AF. The effectiveness of amiodarone in preventing AF following cardiac surgery has been evaluated in two studies (14, 15). Daud and associates (14) started administering amiodarone to patients 7 d before surgery at a dose of 600 mg daily and continued it at a dose of 200 mg daily until dismissal. Compared with placebo patients on amiodarone had a reduced risk of AF (42% versus 23%), shorter hospital stays (7.9 d versus 6.5 d), and lower hospital cost. Guarineri and associates (15) started administering intravenous amiodarone immediately following surgery (1 g/d) and continued treatment for 2 d. Compared to placebo, there were significantly fewer episodes of AF (47% versus 35%), but the length of hospitalization was similar.

Atrial Flutter

New insight into the anatomy of the "flutter circuit" has led to significant advances in management of this rhythm disorder (16). The atrial flutter circuit goes around the tricuspid valve with activation up the atrial septum, across the dome of the

atrium, and down the lateral atrial wall. Conduction is slowed near the isthmus of tissue between the posterior tricuspid valve annulus and the inferior vena cava, allowing the circuit to be maintained, producing "counterclockwise isthmus-dependent atrial flutter," typical of type 1 atrial flutter. This mechanism gives rise to the typical ECG findings with flutter waves that are negative in II, III, and aVF. Atypical atrial flutter has positive flutter waves in the inferior ECG leads and can be caused by a reverse of the usual flutter circuit or by a circuit around incisional scars such as those that occur following an atriotomy or patch placement for repair of an atrial septal defect.

Ibutilide successfully converts 50% of patients with atrial flutter while procainamide converts 20% (17). Antiarrhythmic drugs are typically not helpful in preventing atrial flutter. Catheter ablation is now recommended early in the management of patients with atrial flutter. This involves placing a line of conduction block across the isthmus between the posterior tricuspid valve annulus and the inferior vena cava. The initial success rate is 85–90%. Patients with normal ventricular function and no history of AF have a 95% chance of being free of recurrence long-term whereas patients with a history of AF and reduced ventricular function have a 75% recurrence rate of AF (18).

Supraventricular Tachycardia

Catheter ablation is now used early in the management of patients with supraventricular tachycardia caused by an accessory pathway (either manifest or concealed Wolff-Parkinson-White syndrome), reentry within the AV node, or atrial tachycardia (19). Electrophysiologic mapping of AV reentry circuits have demonstrated that the fast pathway is within the compact AV node, while the slow pathway may be as far as 2–3 cm posterior to the compact AV node. This remote location makes it possible to ablate the slow pathway with only a 1% risk of creating a complete heart block. The success rate of radiofrequency ablation is 95% for patients with an accessory pathway or AV node reentry and approximately 80% for patients with atrial tachycardia.

VENTRICULAR ARRHYTHMIAS

Refractory Ventricular Tachycardia and Fibrillation

Early after acute myocardial infarction and at times after coronary bypass grafting, patients may develop recurrent bursts of rapid, polymorphic ventricular tachycardia (VT) that are unresponsive to lidocaine and procainamide. In such situations, treatment options include: (1) intravenous amiodarone, (2) bretylium, (3) overdrive pacing, (4) intraaortic balloon pump, and (5) coronary revascularization. The cornerstone of therapy is usually revascularization to prevent ischemia. Intravenous amiodarone can be quite effective and is now often used in patients in whom lidocaine and procainamide are ineffective. Patients who develop VT or ventricular fibrillation 48 h after a myocardial infarction in spite of the absence of recurrent ischemia are at high risk for subsequent death. These patients need electrophysiologic evaluation and possible placement of an ICD (20).

Nonsustained Ventricular Tachycardia

Patients with asymptomatic nonsustained VT and otherwise normal left ventricular function (ejection fraction > 40%) are at low risk for developing sudden death or serious ventricular arrhythmias and do not require antiarrhythmic therapy. In contrast, patients with ischemic cardiomyopathy (ejection fraction < 40%) and asymptomatic nonsustained VT are at increased risk for sudden death and require further evaluation.

The MADIT (21) and MUSTT (22) trials have demonstrated that patients with low LV ejection fractions and inducible VT during electrophysiologic testing have improved long-term survival when they are treated with an ICD (3 yr mortality \approx 20%) as compared to conventional treatment with antiarrhythmic medications (3 yr mortality \approx 40%).

Patients with dilated (nonischemic) cardiomyopathy and asymptomatic nonsustained VT are also at increased risk for sudden death despite adequate treatment of their heart failure; however, electrophysiologic testing has not been shown to be of benefit for risk stratification. The use of amiodarone has been demonstrated to improve survival in the GESICA trial in which amiodarone was associated with a 2-yr survival rate of 60% compared to 40% of patients receiving placebo (23). The survival advantage was primarily in patients with a resting heart rate greater than 90 beats/min.

Survivors of Out-of-Hospital Cardiac Arrest and Episodes of Hemodynamically Significant Ventricular Tachycardia

The AVID (24) trial evaluated patients who survived cardiac arrest, sustained VT with syncope, or sustained VT with ejection fraction less than 40%. Patients were randomized either to ICD or to standard treatment with antiarrhythmic drugs, which included either empiric amiodarone or sotalol, guided by electrophysiologic testing. One thousand sixteen patients were randomized with death at 18 mo occurring in 16% of the ICD patients compared to 24% of patients who were receiving the antiarrhythmic drugs ($p < 0.02$).

The widespread use of the ICD has been propelled by the dramatic change in size, ease of implantation, and functionality of the device: (1) the size has been reduced from over 200 cc³ to approximately 35 cc³, (2) implantation mortality is reduced from 5% to less than 1%, (3) device longevity has increased from 1.5 to 5–7 yr, (4) capabilities include dual-chamber rate responsive pacing in addition to antitachycardia pacing and defibrillation therapies, (5) the algorithms to differentiate between supraventricular and ventricular tachycardia have been improved, (6) implantation using a prepectoral device with one or two transvenous leads usually allows hospital dismissal within 24 h, and (7) device memory to assess device function has been markedly improved.

Specific Mechanisms of Ventricular Tachycardia Amenable to Catheter Therapy

Bundle branch reentrant VT. This is perhaps the easiest of all monomorphic VT rhythms to ablate using catheter techniques (25). It is a reentrant tachycardia with activation preceding antegrade over the right bundle, transeptally to the left ventricular apex, returning retrogradely over the left bundle system, and then connecting back again at the junction of the bundles to proceed once again antegrade over the right bundle. The critical portions of the circuit include the left and right bundles as well as the ventricular septum. It is estimated that 5% of patients with coronary artery disease and up to 40% of patients with dilated cardiomyopathy will have this circuit as the mechanism for their clinical monomorphic VT. Thus, a typical patient presenting with bundle branch reentry VT would have (1) dilated cardiomyopathy, (2) intraventricular conduction delay (IVCD) or left bundle branch block, (3) cardiac arrest or syncope, (4) prolonged His to ventricle (HV) interval at the time of electrophysiologic testing, and (5) inducible VT with left bundle branch block/left axis morphology. The conduction system is critical to maintenance of the circuit, and the tachycardia can be cured with ablation of the right bundle branch in 99% of patients. This particular rhythm should be kept in

mind in any patient who has the clinical profile of syncope, dilated cardiomyopathy, and conduction disease.

Right ventricular outflow tract VT. This condition has also been termed repetitive monomorphic VT (26). This rhythm occurs in the background of a structurally normal heart and is typically diagnosed in young men or women. Right ventricular outflow tract VT often begins with a fusion complex and can be associated with repetitive bursts of a nonsustained VT before the sustained tachycardia appears. In over half of all patients, tachycardia cannot be induced with programmed electrical stimulation since the rhythm is autonomously mediated and not caused by myocardial reentry. Calcium channel blockers, beta blockers, and class I antiarrhythmic drugs have been used with some success. The prognosis is generally excellent, although patients who do improve on simple beta or calcium channel blocker therapy should be referred for electrophysiologic testing, and if VT is inducible, radiofrequency catheter ablation should be performed. In some patients in whom sustained VT cannot be induced, nonsustained ventricular ectopy can be used as a target to guide ablation. The success rate with ablation of VT from the right ventricular outflow tract is approximately 90%. The tachycardia is sensitive to adenosine but should not be confused with a supraventricular tachyarrhythmia (27). Typical patients have (1) left bundle branch block, right axis morphology VT, (2) VT that terminates with adenosine, and (3) an otherwise normal heart.

Idiopathic left ventricular tachycardia. This condition has a focal origin in the septum or apical portion of the left ventricle (26, 27). This form of tachycardia often originates in patients experiencing states of high catecholamine drive such as exercise or emotion and is sensitive to verapamil, resulting in misdiagnosis as a supraventricular tachycardia. It is probably caused by micro reentry within the Purkinje network of the left ventricle, is associated with a normal ventricle, and often responds to treatment with a calcium channel blocker or beta blocker. Should these drugs not be effective or not tolerated, the rhythm can be treated with radiofrequency ablation with a success rate of 90%. Typical patients with idiopathic left ventricular tachycardia have (1) right bundle branch block, left axis deviation morphology VT, (2) VT that terminates with verapamil, and (3) an otherwise normal heart.

Long QT Syndrome

The following features are observed clinically in long QT syndrome: (1) QT prolongation with T- and U-wave abnormality, (2) QTc greater than 0.46 seconds, (3) recurrent syncope caused by torsades de pointes, (4) spells misdiagnosed as seizure or vasovagal syncope, and (5) sudden death. Chromosome abnormalities have been identified that affect the sodium and potassium channels with differing phenotypic expression. For example, a defect on chromosome 3 involving the SCN5A gene affects the sodium channel with QT interval shortening during exercise and bradycardia-dependent QT prolongation and torsades de pointes. In contrast, a defect on chromosome 7 involving the HERG gene affects the potassium channel, with the QT interval prolonging during exercise and patients presenting with torsades de pointes caused by adrenergic stress and, frequently, pauses associated with ectopy. The prognosis after the first episode of syncope is 20% mortality at 1 yr and 50% mortality within 10 yr. The mortality can be reduced to 3–4% with therapy. Beta blockade prevents new syncope in 75% of patients, and recurrent syncope can be treated with pacing to prevent pause or bradycardia-dependent syncope. The ICD is increasingly used in this group of patients at high risk for sudden death (28, 29).

Lidocaine Versus Procainamide for the Treatment of Ventricular Tachycardia

A study by Wellens and associates (30) demonstrated that procainamide was superior to lidocaine for the treatment of patients presenting with sustained monomorphic VT in the absence of acute myocardial infarction or myocardial ischemia. Such patients have a reentrant circuit caused by a fixed scar. Procainamide effectively terminated tachycardia in 80% of patients and lidocaine terminated tachycardia in 20% of patients. Lidocaine should be used for the treatment of polymorphic VT occurring in the first 24–48 h after myocardial infarction and in other clinical situations associated with acute ischemia. Intravenous procainamide should be used for the treatment of recurrent monomorphic VT.

CONCLUSIONS

Our understanding of the treatment, mechanism, and prevention of cardiac arrhythmias has advanced substantially over the last several years. The advent of radiofrequency catheter ablation has revolutionized the treatment of patients with cardiac arrhythmias, allowing for cure of the arrhythmia. Patient populations who benefit from the ICD have been identified for both primary prevention in patients at high risk for developing a life-threatening arrhythmia and secondary prevention in patients who have already presented with sudden cardiac death or hemodynamically significant VT. It is anticipated that similar advances will allow improved treatment of patients with AF, which is now the most common cardiac arrhythmia problem.

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