TREATMENT OF TACHYARRHYTHMIAS

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INTRODUCTION

New information published since 1992 prompted a critical reevaluation of recommendations for treatment of arrhythmias with 2 goals: (1) to develop new recommendations for treatment of common tachyarrhythmias based on clinical evidence of efficacy and (2) to develop common algorithms for treatment of such arrhythmias in children and adults insofar as available evidence and pathophysiologic considerations allow. Because of the emergent nature of treatment, evidence evaluation was restricted to parenteral antiarrhythmic medications, including drugs not yet approved for use in the United States (NAUS) but available elsewhere.

Topic 1: Hemodynamically Stable Wide (Broad)-Complex Tachycardia

OVERVIEW

“Hemodynamically stable wide (broad)-complex tachycardia” implies the presence of a regular tachycardia (exceeding the expected limits of sinus tachycardia at rest, ie, more than 120 beats/min in adults) with uniform (monomorphic) QRS configuration of 120 ms or greater duration without signs or symptoms of impaired consciousness or tissue hypoperfusion. Also implied is the presence of clinical stability sufficient to allow diagnosis of the rhythm (or transport to a facility where such a diagnosis can be made) and a blood pressure reserve sufficient to permit pharmacologic intervention and the absence of symptoms suggesting the need for immediate termination using electrical cardioversion.
The differential diagnosis of wide (broad)-complex tachycardia includes (1) ventricular tachycardia (VT); (2) supraventricular tachycardia (SVT) with aberrancy (intra-ventricular conduction delay), including sinus tachycardia (both sinoatrial reentrant and inappropriate automatic sinus tachycardia), intraatrial tachycardia (ectopic or reentrant), atrial flutter with fixed atrioventricular (AV) block, AV nodal reentry tachycardia (AVNRT), and junctional tachycardia; and (3) accessory pathway–mediated tachycardia, including preexcited sinus tachycardia (both sinoatrial reentrant and inappropriate automatic sinus tachycardia), preexcited intraatrial tachycardia (ectopic or reentrant), preexcited atrial flutter, and AV reentry tachycardia (AVRT) (orthodromic reentry tachycardia with aberrancy, antidromic reentry tachycardia, and Mahaim tachycardia).

1992 GUIDELINES

“Lidocaine is recommended as the first agent to use for VT plus all wide-complex tachyarrhythmias not known with certainty to be supraventricular in origin…. If tachycardia persists after a loading dose of lidocaine, adenosine should follow.” (JAMA. 1992;268:2225.) The 1992 guidelines recommend that procainamide, bretylium, and electrical cardioversion be used after adenosine. Wide (broad)-complex tachycardia in children is presumed to be VT. Lidocaine and DC-synchronized cardioversion are recommended therapies.

NEW SCIENCE

Evidence published since 1992 supports the following recommendations for treatment of hemodynamically stable wide (broad)-complex tachycardia: (1) When circumstances and expertise allow, determine whether the wide (broad)-complex tachycardia is of supraventricular or ventricular origin before starting treatment. The origin of the tachycardia can be determined by evaluating the ECG (obtain an esophageal ECG if readily available) and such clinical characteristics as the patient’s age, history of structural heart disease, and presence (known or suspected) of an accessory pathway. Wide (broad)-complex tachycardia in adults with a history of structural heart disease is much more likely to be due to VT than SVT with aberrancy. (2) Treat electrophysiologically confirmed or strongly suspected SVT with aberrancy or VT according to the appropriate treatment algorithm. (3) When electrical cardioversion is not feasible, desirable, or successful and empiric therapy is believed to be necessary, use drugs with activity against both supraventricular and ventricular tachyarrhythmias.

EVALUATION AND DEBATE

The origin (ventricular or supraventricular) of more than half of the wide (broad)-complex tachycardias occurring in adults is misdiagnosed by initial care providers (most often by misdiagnosing VT as SVT with aberrancy), and patients are subsequently treated inappropriately.1,2 The diagnostic use of the 12-lead ECG in the differentiation of SVT from VT is supported by 6 case series of good quality (level of evidence [LOE] 5).1,3-7 Unfortunately, 12-lead electrocardiography is not universally available outside the hospital, and the complex rules of rhythm discrimination pertaining to QRS morphology can be difficult to teach, learn, remember, and reproducibly apply.2,8-12 AV dissociation, defined as the loss of a 1-to-1 relationship between atrial electrical activity (P waves) and ventricular response (QRS complexes), is a less sensitive but more specific and intuitive criterion for determining the presence of VT. A number of case series of fair-to-good quality (LOE 5) suggest that the esophageal-lead ECG, which amplifies P waves, improving their recognition among a wide (broad)-complex tachycardia, is a useful and effective means of discriminating ventricular from supraventricular arrhythmias that requires minimal skill and little time and poses virtually no risk of complications.13-16

Lidocaine is neither an effective nor appropriate therapy for SVT. The use of lidocaine as a “first-line” agent for VT or a wide (broad)-complex arrhythmia of uncertain origin is not supported by available evidence. Although lidocaine can suppress ventricular arrhythmias associated with acute myocardial ischemia and infarction,17 its routine (prophylactic) use in patients with these conditions has been associated with higher mortality and has since been abandoned.18-21 Two studies of fair-to-good quality (LOE 5) suggest that lidocaine is relatively ineffective for termination of hemodynamically stable VT.22,23 and 2 studies of good quality (LOE 1) found it to be less effective against VT than intravenous (IV) procainamide24 or IV sotalol.25 Adenosine, whose principal effect is to slow AV nodal conduction, is ineffective for common forms of ventricular arrhythmia and preexcited atrial arrhythmias.26-30 Although the vasodilatory effects of adenosine are short lived, worsened hypotension has been reported in patients with marginally compensated blood pressure who were given adenosine for VT.28 Adenosine also may theoretically cause angina, bronchospasm, proarrhythmia, and acceleration of accessory pathway conduction.28
Antiarrhythmic agents such as procainamide and amiodarone are effective in treating a broad variety of arrhythmias, including supraventricular arrhythmias, supraventricular arrhythmias mediated by an accessory pathway, and VT. The efficacy of procainamide for termination of VT is supported by 1 study of good quality (LOE 2) comparing procainamide and lidocaine and 2 case series of fair quality (LOE 5). The efficacy of procainamide against SVT, including its ability to alter conduction across an accessory pathway, is supported by results extrapolated from 12 studies of fair-to-good quality (LOE 7) that evaluated mainly treatment of atrial fibrillation and flutter.

The efficacy of amiodarone against SVT, including alteration of accessory pathway conduction, is supported by results extrapolated from 30 studies of fair-to-good quality (LOE 7) examining principally patients with atrial fibrillation and flutter. Results extrapolated from 12 studies of fair-to-good quality (LOE 7) support the efficacy of amiodarone against hemodynamically unstable VT and fibrillation, although the drug has not been studied specifically for pharmacologic termination of hemodynamically stable VT. Both procainamide and amiodarone have vasodilatory and negative inotropic properties that can result in hemodynamic destabilization. These effects seem to be dependent on dosage and rate of administration; IV amiodarone may be better tolerated hemodynamically than procainamide or bretylium.

Bretylium has not been directly studied for treatment of wide (broad)-complex tachycardia of uncertain origin. Bretylium was developed initially as an antihypertensive agent; its use in patients with VT has been associated with hypotension, particularly when compared with amiodarone. IV sotalol (NAUS), IV propafenone (NAUS), and IV flecainide (NAUS) have not been studied specifically for treatment of wide (broad)-complex tachycardias. Nonetheless, all are effective against SVT, including atrial arrhythmias with or without preexcitation. IV sotalol is more effective against VT than IV lidocaine. IV flecainide and IV disopyramide effectively terminate hemodynamically stable VT; the use of IV propafenone in patients with VT has not been studied adequately. IV disopyramide is effective against atrial arrhythmias, but the incidence of adverse effects is high.

Wide (broad)-complex tachycardia in children

The definition of a wide (broad)-complex tachycardia in children is the same as in adults, except that the duration of the QRS configuration needs to be only greater than 100 ms. The electrophysiologic mechanisms of wide (broad)-complex tachycardia are heterogeneous, and the exact incidence of VT in children is unknown. Data on the efficacy of lidocaine, procainamide, and amiodarone in children with VT are extremely limited. Most data are from small patient series or case reports or are extrapolated from studies of adults (see above). Lidocaine is ineffective for treating SVT and atrial arrhythmias and is considerably less effective than procainamide or amiodarone for VT, but it can be administered rapidly and has few adverse hemodynamic consequences. Procainamide and amiodarone are highly effective for treatment of both SVT and VT. Of the 2 drugs, amiodarone is the most widely studied and seems to be reasonably effective and to have few adverse effects. Both amiodarone and procainamide must be given slowly, and both can cause hypotension. There is minimal published experience with the use of IV ibutilide (NAUS), IV flecainide (NAUS), or IV propafenone (NAUS) in children.

Adenosine, although very effective for SVT, is not useful for VT in children and may accelerate the heart rate, increasing the likelihood of serious hemodynamic compromise. Not all causes of VT respond to adenosine (eg, atrial flutter or fibrillation, particularly with a preexcited QRS complex). Verapamil remains a highly dangerous drug for patients with wide (broad)-complex tachycardia.

PROPOSED GUIDELINES

In adults and children first evaluate the ECG and patient characteristics to determine the origin of the tachycardia. Do not base assumptions about the mechanism solely on the hemodynamic status of the patient. Electrical cardioversion is an effective therapy for wide (broad)-complex tachycardia that avoids many of the potential complications resulting from the use of antiarrhythmic drugs but may not always be feasible, successful, or desirable.

When the origin of a wide (broad)-complex tachycardia cannot be determined by evaluation of the ECG or clinical characteristics of the patient, and electrical cardioversion is not feasible, desirable, or successful, administer IV procainamide, IV amiodarone, or IV sotalol. Lidocaine is less effective for termination of wide (broad)-complex tachycardia of uncertain origin in adults and children. Limited data suggest that IV flecainide or IV disopyramide may be effective against both SVT and stable VT in adults; no data for children are available. There is insufficient evidence to recommend the use of bretylium for wide (broad)-complex tachycardia, and this agent may cause hypotension. There is insufficient evidence to recommend the use of IV ibutilide (NAUS), IV flecainide (NAUS), IV propafenone...
(NAUS), or IV disopyramide (NAUS) in adults or children with wide (broad)-complex tachycardias of uncertain origin.

**Topic 2: Hemodynamically Stable (Monomorphic) VT**

**OVERVIEW**

“Hemodynamically stable VT” implies the presence of confirmed VT without clinical evidence of tissue hypoperfusion or symptoms that suggest the need for immediate termination using electrical cardioversion. Implicit in the definition of such a tachycardia is the presence of sufficient clinical stability and blood pressure reserve for pharmacologic intervention.

**1992 GUIDELINES**

The 1992 guidelines recommend the use of lidocaine, followed by procainamide, bretylium, and electrical cardioversion to treat hemodynamically stable VT.

The 1992 guidelines contain no specific recommendations for the treatment of hemodynamically stable VT in children. Existing recommendations for the treatment of unstable VT include administration of lidocaine (if vascular access is available) or electrical cardioversion. Bretylium is recommended for patients with arrhythmias that are resistant to or recur after initial treatment.

**NEW SCIENCE**

When electrical cardioversion is not feasible, desirable, or successful in a patient with hemodynamically stable VT, the use of IV procainamide, IV sotalol (NAUS), or IV amiodarone is preferred over the use of IV lidocaine or IV bretylium.

**EVALUATION AND DEBATE**

Although lidocaine can be administered rapidly with minimal effect on blood pressure, 2 studies of fair-to-good quality (LOE 5) and 2 studies of good quality (LOE 1) suggest that lidocaine is relatively ineffective for termination of VT\(^22,23\) and less effective against VT than IV procainamide\(^24\) or IV sotalol.\(^25\) The use of IV amiodarone is supported by results extrapolated from 12 studies of treatment of hemodynamically unstable VT and ventricular fibrillation (VF),\(^70,73-83\) but the drug has not been evaluated specifically for pharmacologic termination of hemodynamically stable VT. The use of bretylium is supported by results extrapolated from patients treated for cardiac arrest,\(^94-96\) but this drug results in a higher incidence of hypotension than amiodarone.\(^79\) Procainamide, sotalol, amiodarone, and bretylium cannot be given as rapidly as lidocaine, and all 4 may provoke or aggravate hypotension. Limited data on the efficacy of IV flecainide (NAUS) and IV disopyramide (NAUS) in hemodynamically stable VT induced in the electrophysiology laboratory\(^27\) are available, and the data for IV propafenone are insufficient to recommend this agent for such use.

**VT in children**

Since 1992, clinical researchers have presented data demonstrating the efficacy of IV amiodarone in the treatment of recurrent or refractory ventricular arrhythmias in children. These findings are consistent with those of several large-scale studies of adults. IV amiodarone is also helpful when a hemodynamically significant wide (broad)-QRS complex tachycardia of uncertain origin occurs because it can slow or terminate both supraventricular and ventricular arrhythmias.

Data on the efficacy of lidocaine, procainamide, and amiodarone in children are extremely limited. Most data are from small patient series or case reports.\(^73,88-92\) Procainamide is effective for a broad variety of arrhythmias,\(^97-99\) but there is little documentation of its efficacy in treating VT in children. There is no published experience with the use of IV sotalol, flecainide, or propafenone to treat VT in children. Much of current practice is based on results extrapolated from studies of adults (see above).

**PROPOSED GUIDELINES**

Electrical cardioversion remains a highly effective and recommended therapy for hemodynamically stable VT. When electrical cardioversion is not feasible, desirable, or successful in adults, use IV procainamide (Class IIa), IV sotalol (Class IIa NAUS). IV amiodarone (Class IIb) or lidocaine (Class IIb). If the patient exhibits clinical signs of impaired left ventricular (LV) function (ejection fraction <40% or congestive heart failure [CHF]), use amiodarone (Class IIb) or lidocaine (Class IIb), followed by synchronized cardioversion if unsuccessful. In children, a more cautious approach to the treatment of hemodynamically stable VT is warranted. If pharmacologic therapy is undertaken, amiodarone (Class IIb) is preferred, with procainamide or lidocaine as alternative agents. There is insufficient evidence to recommend the use of
bretylium, IV sotalol, IV flecainide, or IV propafenone to treat VT in children. Apart from these differences in drug classification, adults and children with VT can be treated similarly.

**Topic 3: Polymorphic VT**

**OVERVIEW**

Polymorphic VT refers to VT with beat-to-beat variations in QRS complex morphology, including torsade de pointes, which is defined as a bradycardia (pause)-dependent polymorphic VT usually in the setting of QT prolongation, and polymorphic VT in the absence of clinical evidence of torsade de pointes (ie, normal QT interval and no evidence of pause dependence).

**1992 GUIDELINES**

The 1992 guidelines contain no recommendations for treatment of polymorphic VT. The 1992 guidelines recommend use of magnesium for patients with pulseless VT in whom torsade de pointes is suspected.

**NEW SCIENCE**

There are limited data on treatment of polymorphic VT with or without suspected torsade de pointes. Hemodynamically unstable polymorphic VT should be treated according to the VF/pulseless VT algorithm. Hemodynamically stable polymorphic VT should be treated on the basis of whether torsade de pointes is a suspected mechanism.

**EVALUATION AND DEBATE**

Data from case reports (LOE 5) suggest that polymorphic VT attributable to torsade de pointes should be treated with immediate discontinuation of potentially offending medications with QT-prolonging properties, correction of electrolytes, and administration of IV magnesium, temporary atrial or ventricular pacing with or without adjunctive β-blockade (based on data extrapolated from studies of long-term management of torsade de pointes), or isoproterenol (as a prelude to temporary pacing in patients without ischemic contraindications or in whom temporary pacing is not feasible). Extrapolation from studies evaluating antiischemic interventions in patients with acute myocardial infarction (LOE 7) suggest that β-blockers (and perhaps other antiischemic measures) may be beneficial against ventricular arrhythmias in this setting. Lidocaine may also be more effective than other agents in patients with acute myocardial ischemia. Amiodarone, procainamide, bretylium, or sotalol may also be helpful, based on results extrapolated from studies of their efficacy against VT.

**EVALUATION AND DEBATE**

Results extrapolated from 1 preventative study of familial polymorphic VT not associated with torsade de pointes or acute myocardial ischemia suggest that treatment with β-blockers is effective. The use of IV propafenone, IV flecainide, IV disopyramide (NAUS), and IV ibutilide in the treatment of polymorphic VT has not been studied, although IV ibutilide has been associated with provocation of polymorphic VT and thus may be contraindicated in its treatment.

**Polymorphic VT in children**

Data on treatment of polymorphic VT in children are mainly from studies of long QT syndrome, in which pacing with or without adjunctive β-blockers is reportedly an effective prophylactic measure (LOE 7, extrapolated evidence). No information on treatment of polymorphic VT in the absence of torsade de pointes in children is available; recommendations are based on data extrapolated from studies of VF and VT (LOE 7).

**PROPOSED GUIDELINES**

Electrical cardioversion remains a highly effective and recommended therapy for VT regardless of its morphology. Treat patients with hemodynamically unstable polymorphic VT according to the VF/pulseless VT algorithm, being careful to watch for signs of torsade de pointes or acute myocardial ischemia. In adults or children with polymorphic VT believed to be due to torsade de pointes, immediately discontinue the use of potentially offending QT-prolonging drugs (and avoid the use of such drugs during treatment), correct electrolyte imbalances, administer IV magnesium, begin temporary pacing with or without adjunctive β-blocker therapy (based on results extrapolated from studies of long-term treatment of patients with long QT syndrome), or administer isoproterenol (if not contraindicated because of the patient’s age or the presence of myocardial ischemia, as a temporizing measure until placement of a temporary pacemaker, or in patients in whom temporary pacing is not feasible). The use of antiarrhythmic medications, particularly those with QT-pro-
longing properties, is not recommended and may be harmful. When torsade de pointes is not believed to be operative but provocation by ischemia is suspected, anti-ischemic measures may be used; electrolyte imbalances should also be corrected. Potentially effective parenteral antiarrhythmic drugs in adults and in children include lidocaine, IV amiodarone, procainamide, bretylium, sotalol, β-blockers, or phenytoin. IV flecainide (NAUS), propafenone (NAUS), and disopyramide (NAUS) have not been sufficiently studied; IV ibutilide is not recommended. All medications for treating torsade de pointes in the final guidelines were classified as an Indeterminate Class recommendation.

Topic 4: VF and Pulseless VT

OVERVIEW

The highly emergent nature of shock-refractory pulseless VT or VF requires that the initial agent selected for therapy have the greatest demonstrated efficacy. To date, the only studied measures of efficacy of treatment with antiarrhythmic drugs pertain to short-term outcome and not necessarily long-term survival. The efficacy and recommendations for the use of antiarrhythmic agents are therefore based on such surrogate measures of outcome, which may or may not represent their actual impact on survival after cardiac arrest.

1992 GUIDELINES

The 1992 guidelines recommend the use of antiarrhythmic medications (lidocaine, followed by bretylium, procainamide, and magnesium sulfate when hypomagnesemia or torsade de pointes is suspected) if epinephrine and 4 precordial shocks (3 administered before and a fourth after receipt of epinephrine) do not restore a perfusing rhythm.

The 1992 guidelines provided only cursory recommendations for the use of drugs to treat children with shock-refractory VF or recurrent hypotensive VT. Bretylium was recommended if defibrillation and lidocaine did not terminate VF in a pediatric patient.

NEW SCIENCE

After repeated administration of precordial shocks and epinephrine, IV amiodarone is an acceptable agent for improving the short-term success of resuscitation and restoring spontaneous circulation in patients with shock-refractory cardiac arrest due to VF or pulseless VT. Lidocaine, bretylium, magnesium (for suspected torsade de pointes or patients in hypomagnesemic states), and procainamide are alternative treatments with less supportive evidence for their efficacy in cardiac arrest at the present time.

EVALUATION AND DEBATE

The use of lidocaine for shock-refractory, hemodynamically unstable ventricular arrhythmias is supported by 1 historical comparative trial (n = 1,360 patients) of fair-to-good quality (LOE 4) that demonstrated improved resuscitation and survival to hospital admission, 5 animal studies of fair quality (LOE 6), and results extrapolated from 1 historical study of the use for suppression of premature ventricular contractions and prevention of VF after acute myocardial infarction. One trial of fair quality (LOE 3) showed no statistically significant differences in outcome between victims of cardiac arrest who received lidocaine and patients in a historical control group who received no antiarrhythmic medications, and 2 randomized studies (LOE 2) comparing lidocaine and bretylium demonstrated no difference in outcome. Studies opposing the use of lidocaine include 1 randomized study of good quality (LOE 1) comparing amiodarone and lidocaine that showed a greater likelihood of successful resuscitation with use of amiodarone, and 1 randomized study of good quality (LOE 2) comparing lidocaine and epinephrine that demonstrated a higher incidence of asystole with the use of lidocaine and no difference in return of spontaneous circulation. 1 retrospective, uncontrolled trial of good quality (LOE 4) suggesting that lidocaine reduced the short-term success of resuscitation, numerous animal studies (LOE 6) demonstrating a lower rate of short-term survival after administration of lidocaine and elevation in the defibrillation threshold after treatment, and meta-analyses and recent retrospective cohort studies suggesting increased mortality with the prophylactic use of lidocaine in patients with acute myocardial infarction.

Support for the use of bretylium in patients who have had a cardiac arrest comes from 1 randomized trial of fair quality (LOE 2) demonstrating a marginally significant benefit in patients treated for VF or asystole (no benefit in those treated for VF alone) who had been provided with only basic life support before arrival in the emergency department and 2 randomized (LOE 2) trials comparing lidocaine and bretylium in patients treated for cardiac arrest that demonstrated no difference in outcome between the
2 agents. Studies opposing the use of bretylium for ventricular tachyarrhythmias include 1 cohort study (LOE 4) suggesting worsened short-term outcome in recipients of bretylium, 1,18 1 comparative study of good quality (LOE 2) of IV amiodarone demonstrating a higher incidence of hypotension with bretylium, 79 1 study of fair quality (LOE 3) demonstrating significant hypotension after administration of bretylium during induced VT, 127 1 of good quality (LOE 6) suggesting hemodynamic deterioration after treatment with bretylium. 128

The use of procainamide in patients in overt cardiac arrest is supported by a retrospective comparison study involving only 20 patients. 129 The dosage and manner of procainamide administration in this study were not specified. Typically, procainamide is recommended to be given as a relative slow infusion (maximum rate of 50 mg/min), which is an impractical consideration during pulseless cardiac arrest. Bolus dosing of the drug has not been studied, and available data suggest such rapid rates of administration may be toxic. 84

The use of magnesium in patients with torsade de pointes is supported by 2 uncontrolled case series of fair quality (LOE 5). 103,130 Apart from such specific indications, the routine use of magnesium during resuscitation is not supported by 2 randomized controlled trials that demonstrated no difference in outcome, 1 of which identified a potential for a higher incidence of hypotension from routine use of magnesium. Magnesium has been associated with improved neurologic outcome in survivors of cardiac arrest 131,132 and requires further study for this indication.

The use of IV amiodarone in patients with VF/pulseless VT is supported by 1 randomized, placebo-controlled trial of excellent quality (LOE 1) demonstrating an improvement in survival to hospital, 83 1 randomized trial of excellent quality (LOE 2) identifying similar overall efficacy (by intention to treat) but less hypotension than bretylium, 79 1 small randomized trial of fair-to-good quality (LOE 1) showing greater efficacy than lidocaine, 116 3 case series (LOE 5), 75,76,133 and 1 animal study (LOE 6) demonstrating greater benefit of treatment with amiodarone and lidocaine than treatment with lidocaine alone. 128 Studies providing opposing evidence include 1 animal study of good quality (LOE 6) suggesting that amiodarone, like lidocaine, also increases the defibrillation threshold, 134 although this has not been a consistent observation. 135 No studies of the use of IV sotalol (NAUS), IV flecainide (NAUS), IV propafenone (NAUS), IV disopyramide (NAUS), or IV ibutilide in patients in cardiac arrest have been published.

On the basis of discussions at the Evidence Evaluation and Guidelines 2000 conferences, scientists believe that evidence supports the use of IV amiodarone following epinephrine to treat shock-refractory cardiac arrest due to VF or pulseless VT (Class IIb). A higher classification was not considered justified at this time because the only endpoint that has been studied with an adequate sample size is restoration of spontaneous circulation and improved early survival to hospital discharge. Lidocaine is still considered to be an acceptable antiarrhythmic agent for use in treating patients with VF/pulseless VT, but it is now considered to be an Indeterminate Class recommendation because there have been no placebo-controlled randomized trials of lidocaine’s efficacy. Magnesium sulfate is recommended (Class IIb) only for polymorphic VT (torsade de pointes) or VF/pulseless VT accompanied by suspected or known hypomagnesemia. Procainamide is acceptable (Class IIb) for intermittent/recurrent VF/VT but not recommended because its prolonged administration time is usually unsuitable for cardiac arrest.

**VF and pulseless VT in children**

Shock-refractory VT or VF is a rare problem in pediatric resuscitation. Although use of amiodarone has been evaluated in children, the studies are primarily small case series (<40 patients). 66,73,91-93 Results of 4 open-label clinical studies of the use of IV amiodarone for treatment of recurrent monomorphic VT or VF in children (who received amiodarone for severe arrhythmias but not pulseless VT or VF) have been reported, 1 of good quality and 3 of fair quality, with 1 study of level 3 methodology and 3 studies with level 5 methodology and design. Minimal evidence supporting the use of lidocaine or procainamide in pediatric patients with sustained or shock-refractory VT or VF has been published. Data on other frequently used or potential antiarrhythmic medications, including bretylium, magnesium, IV ibutilide, IV sotalol (NAUS), IV flecainide (NAUS), IV propafenone (NAUS), and IV disopyramide (NAUS), in pediatric resuscitation are even more limited or nonexistent. 97 As in adults, improved long-term survival has not been documented for any pharmacologic intervention given for cardiac arrest in children.

**Proposed Guidelines**

Along with epinephrine, IV amiodarone is an acceptable pharmacologic agent for improving the short-term success of resuscitation and restoring spontaneous circulation in adults with shock-refractory cardiac arrest due to VF or
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pulseless VT. Lidocaine, magnesium (for suspected torsades de pointes or hypomagnesemic states), and procainamide are alternative treatments with less supportive evidence for their efficacy in cardiac arrest at the present time. Procainamide must be further studied under conditions of cardiac arrest that require bolus drug administration, and at present is recommended only in patients with perfusing rhythms at the time of its infusion (ie, for intermittent VT/VF). Parenteral disopyramide, flecainide, propafenone, sotalol, IV ibutilide, and dofetilide have not been formally studied for efficacy in cardiac arrest. Bretylium is no longer recommended because of limited evidence for its efficacy, high incidence of hypotension, and shortage of raw material for its continued manufacture. Antiarrhythmic agents acceptable for use in adults are also acceptable treatment of shock-refractory VT or VF in children, but the data documenting their effectiveness and safety in children are more limited (evidence is extrapolated from studies of adults).

Topic 5: Paroxysmal Supraventricular Tachycardia

OVERVIEW

“Paroxysmal supraventricular tachycardia” (PSVT) implies the presence of a regular tachycardia (exceeding the expected limits of sinus tachycardia at rest, ie, >120 beats/min) of abrupt onset and termination, with or without discernible P waves that is known to be of supraventricular origin (QRS complex <100 ms or if wide [broad], bundle branch aberrancy is known to be present). PSVT may include AVNRT or AVRT mediated by a concealed or manifest accessory pathway.

1992 GUIDELINES

The 1992 guidelines recommended that PSVT be treated with vagal maneuvers, followed by adenosine and thereafter treated on the basis of the duration of the QRS complex (wide [broad]- versus narrow-complex tachycardia) and blood pressure.

NEW SCIENCE

Since 1992, new parenteral drugs for treatment of PSVT have become available, and a new algorithm for treatment of wide (broad)-complex tachycardia of uncertain origin has been proposed. Great caution should be exercised when administering previously and newly recommended parenteral medications in patients with PSVT and significantly impaired LV function.

EVALUATION AND DEBATE

Initial use of vagal maneuvers and IV adenosine in all patients with PSVT and no contraindications remains supported by evidence evaluated for the 1992 guidelines. In patients with preserved LV function, the use of calcium channel blockers (verapamil and diltiazem) and β-blockers (esmolol and metoprolol) remains supported by previous evidence. Digoxin is a time-honored drug for treatment of PSVT, but indirect evidence from studies of its use for treatment of atrial fibrillation and flutter suggests that it has a slower onset of action and is less potent than alternative drugs.

In addition to procainamide, newly available parenteral antiarrhythmic agents for treatment of PSVT refractory to vagal maneuvers, adenosine, and AV nodal–blocking agents include amiodarone, propafenone (NAUS), flecainide (NAUS), and sotalol (NAUS). The use of IV procaainamide for PSVT (in the presence or absence of an accessory pathway) is supported by 1 study of good quality (LOE 2) showing its comparability with IV amiodarone61 and results extrapolated from numerous studies (LOE 7) related primarily to its use for conversion of atrial fibrillation or alteration of conduction in accessory pathways. The use of IV amiodarone is supported by at least 8 studies of good quality (LOE 5) specific to AVNRT or accessory pathway–mediated AVRT45,49,52,55,66,71,73,92 (3 of these studies66,73,92 were conducted in children), 2 studies (LOE 2) showing comparability with procaainamide61 and magnesium,136 and 1 study (LOE 2) showing less efficacy than propafenone50. The use of IV flecainide for PSVT is supported by evidence from 1 study (LOE 7) demonstrating an effect on slowing or blocking accessory pathway conduction similar to that of propafenone137, and 1 study (LOE 5) specific to AVNRT or AVRT.138 The use of IV propafenone is supported by results extrapolated from 2 studies (LOE 7) examining its effects on accessory pathway conduction139 and comparing IV propafenone with IV flecainide137 and 5 studies evaluating treatment of PSVT due to AVNRT and AVRT (2 with an LOE of 140,141 and 3 with an LOE of 5142-143). The use of IV sotalol to treat PSVT due to AVNRT and AVRT is supported by 2 trials of excellent quality (LOE 1)144,145 and data extrapolated from 1 study (LOE 7) demonstrating its slowing effects on accessory pathway conduction.146 No data related to the use of parenteral bretylium, ibutilide, or IV disopyramide (NAUS) for treat-
ment of PSVT have been published, although ibutilide and disopyramide are known to be effective against atrial fibrillation and flutter. Virtually all antiarrhythmic agents, except digoxin, can decrease blood pressure and cause hypotension. In addition, primary antiarrhythmic agents (eg, amiodarone, procainamide, sotalol, flecainide, propafenone, ibutilide, and disopyramide) have the potential for proarrhythmic effects, including provocation of life-threatening ventricular arrhythmias. Therefore, antiarrhythmic agents should be considered only when AV nodal–blocking agents or electrical cardioversion is not feasible, desirable, or successful.

If LV function is significantly impaired (ie, there is clinical evidence of CHF or moderately to severely reduced LV ejection fraction) in patients with PSVT, exercise caution in administering drugs with negative inotropic effects (eg, verapamil, β-blockers, parenteral bretylium, ibutilide (NAUS), sotalol (NAUS), flecainide (NAUS), or disopyramide (NAUS)) in children. Significant life-threatening side effects have been reported in infants, so use of verapamil should be limited to children older than 1 year.

Several of the newer antiarrhythmics have been studied in children, but the data are much more limited than in adults and many of the recommendations are based on results extrapolated from studies of adults. IV amiodarone was reported to be reasonably effective in 3 studies (LOE 5). The use of IV propafenone is supported by 1 pediatric study (LOE 5). No experience with use of parenteral bretylium, ibutilide (NAUS), sotalol (NAUS), flecainide (NAUS), or disopyramide (NAUS) in children has been published. Because these agents cause significant hypotension and have potentially life-threatening proarrhythmic effects, their use should be considered only when AV nodal–blocking agents or electrical cardioversion is not feasible, recommended, or successful.

PROPOSED GUIDELINES

If no contraindications are present, initially treat all patients with PSVT with vagal maneuvers or adenosine. If LV function is preserved, calcium channel blockers (verapamil or diltiazem), β-blockers, or digoxin may be used. Strong consideration should be given to electrical cardioversion when AV nodal–blocking agents are unable to terminate PSVT. When electrical cardioversion is not feasible, desirable, or successful and AV nodal–blocking agents do not terminate a persistent or recurrent PSVT, antiarrhythmic agents (ie, parenteral procainamide, amiodarone, flecainide [NAUS], propafenone [NAUS], or sotalol [NAUS]) may be used, although the proarrhythmic potential of these drugs makes them less desirable alternatives than AV nodal–blocking drugs. In patients with significantly impaired LV function, avoid the use of verapamil, β-blockers, parenteral bretylium, ibutilide, propafenone, and sotalol and instead use digoxin, amiodarone, diltiazem, or DC cardioversion.

Available data support a similar approach to PSVT in children, although many of the pharmacologic recommendations are based on data extrapolated from studies of adults. Vagal maneuvers (Class IIa for facial ice or ice water application, Class IIb for other vagal maneuvers
such as carotid sinus massage or Valsalva) should be tried first. If unsuccessful, adenosine is recommended (Class IIa) for children. Verapamil should not be used to treat SVT in infants because refractory hypotension and cardiac arrest have been reported (Class III). Its use in children is discouraged because it may cause hypotension and myocardial depression. When used in older children, verapamil is infused in a dosage of 0.1 mg/kg. Procainamide and amiodarone are alternative agents for use in children with SVT and stable hemodynamics (Class IIb) but they should not be used concurrently with agents that may prolong the QT interval. Amiodarone and procainamide generally should not be administered together because they both prolong the QT interval.

Topic 6: Atrial Tachycardia (Ectopic Atrial Tachycardia and Multifocal Atrial Tachycardia)

OVERVIEW

Ectopic atrial tachycardia and multifocal atrial tachycardia represent a complex range of atrial arrhythmias that are often “secondary phenomena” triggered by the patient’s underlying disease. When an obvious trigger can be identified, effort should be made to correct the underlying abnormality.

1992 GUIDELINES

The 1992 guidelines do not specify recommendations for treatment of atrial tachycardias apart from atrial fibrillation and atrial flutter.

NEW SCIENCE

Existing evidence suggests that automatic atrial tachycardias are due to increased automaticity and require a different treatment approach than other reentrant supraventricular arrhythmias (ie, PSVT, atrial fibrillation, and atrial flutter).

EVALUATION AND DEBATE

Atrial tachycardia can be confirmed by use of either vagal maneuvers or adenosine to demonstrate AV block without disruption of the atrial arrhythmia. Unlike arrhythmias whose mechanisms are due to reentry, automatic rhythms

(i.e., ectopic atrial tachycardia and multifocal atrial tachycardia), like sinus tachycardia, do not respond to electrical cardioversion. Many of these arrhythmias are secondary phenomena, requiring supportive measures and treatment of precipitating causes. Multifocal atrial tachycardia typically occurs in patients with severe illness, most commonly chronic obstructive pulmonary disease. In patients with preserved LV function, limited data (LOE 5) suggest that β or calcium channel blockers (verapamil or diltiazem) may improve rate control by enhancing AV block or converting the arrhythmia to normal sinus rhythm; digoxin may slow heart rate but will not terminate an ectopic atrial arrhythmia (LOE 5). The use of amiodarone for ectopic or multifocal atrial tachycardia is supported by 3 studies (LOE 5); flecainide, by 2 studies (LOE 5); and propafenone, by 2 pediatric studies (LOE 5). No studies on the use of bretylium, sotalol, or disopyramide have been published. Quinidine, procainamide, and phenytoin have not been found to provide benefit (LOE 5).

Atrial tachycardia in children

For treatment of atrial tachycardia in children, several studies (LOE 5) support an approach similar to that used in adults. Such arrhythmias in children are often observed as a late complication of surgical correction of congenital heart disease. The arrhythmias tend to be chronic and often do not require emergency therapy. The approach to diagnosis in children is comparable to that used in adults.

PROPOSED GUIDELINES

The presence of ectopic or multifocal atrial tachycardia can be confirmed by a 12-lead ECG (demonstrating an unusual P wave axis or P waves with >2 configurations) or diagnostic vagal maneuvers or adenosine (if necessary) to demonstrate AV block without disruption of the atrial arrhythmia. Remember that adenosine may promote bronchospasm and may be harmful to patients with reactive airways disease. Electrical cardioversion is ineffective for treatment of automatic arrhythmias and should not be used to terminate automatic ectopic or multifocal atrial tachycardia. In patients with preserved LV function, acceptable treatments include calcium channel blockers, β-blockers, digoxin, amiodarone, IV flecainide (NAUS), and IV propafenone (NAUS). Insufficient data are available to recommend the use of procainamide, bretylium, disopyramide, ibutilide, and phenytoin. In patients with
impaired LV function, in whom drugs with significant negative inotropic properties (eg, verapamil, β-blockers, flecainide, and propafenone) are contraindicated, use digoxin, diltiazem, or amiodarone. Recommendations for treatment of atrial tachycardia in children are based on data extrapolated from studies of adults.

**Topic 7: Atrial Fibrillation and Flutter**

**OVERVIEW**

Atrial fibrillation is the result of chaotic atrial depolarization from multiple areas of reentry within the atria. The ventricular rate in atrial fibrillation is irregularly irregular, with no organized P wave activity preceding QRS complexes. Atrial flutter is the result of a single reentry circuit within the right atrium. P waves in atrial flutter have a characteristic “sawtooth” appearance, usually at a rate of 300 beats/min. Atrial fibrillation and flutter are treated similarly with respect to anticoagulation, control of ventricular rate, and restoration of normal sinus rhythm.

**1992 GUIDELINES**

The 1992 guidelines and tachycardia treatment algorithm provided only cursory recommendations for treatment of atrial fibrillation or flutter (“Consider diltiazem, β-blockers, verapamil, digoxin, procainamide, quinidine, anticoagulants.” JAMA. 1992;268:2223) and no specific recommendations for treatment of stable atrial flutter or fibrillation in children.

**NEW SCIENCE**

New evidence has become available since 1992 to allow more specific recommendations for treatment of acute atrial fibrillation or flutter. Pharmacologic rate control, anticoagulation, and cardioversion of patients with preserved or significantly impaired LV function and with preexcited atrial fibrillation or flutter are addressed.

**EVALUATION AND DEBATE**

Ventricular rate control, assessment of the need for anticoagulation, and restoration of sinus rhythm are major goals in the management of atrial fibrillation. It is also essential to investigate and correct reversible and underlying causes of these arrhythmias, including hypoxemia, ischemia, anemia, hypertension, mitral regurgitation, thyrotoxicosis, hypokalemia, hypomagnesemia, and other toxic and metabolic causes.

In patients with atrial fibrillation or flutter, preserved LV function, and a heart rate 120 beats/min or greater, evidence published before and since 1992 supports initial efforts at obtaining rate control with diltiazem (6 studies of fair-to-excellent quality, LOE 2,162-167 and 2 studies of fair-to-good quality, LOE 5168,169), β-blockers (6 studies of fair quality, LOE 5,170-175 and 1 study of good quality, LOE 2176), verapamil (6 studies of good-to-excellent quality, LOE 2,164,177-181 and 3 studies of fair-to-good quality, LOE 5182-184), or digoxin (1 study of good quality, LOE 2185). Available evidence (from both placebo-controlled and comparative studies of β and calcium channel blockers) suggests that digoxin is the least potent agent and has the slowest onset of action of the available pharmacologic options for ventricular rate control. New evidence suggests that IV amiodarone is also effective for rate control if efforts to convert atrial fibrillation to sinus rhythm are unsuccessful (LOE 1),186 in patients resistant to conventional heart rate control measures (LOE 5),54 or in combination with digoxin (LOE 2).47 Amiodarone can convert atrial fibrillation to sinus rhythm, and its use for rate control should be weighed against this possible conversion risk, particularly in patients who are not adequately anticoagulated.

Drugs used to treat patients with atrial fibrillation and significant LV dysfunction must have minimal negative inotropic properties or they may further compromise cardiovascular function. In the past IV β-adrenergic antagonists, calcium channel blockers, and antiarrhythmic agents, including procainamide, were widely used. We now know these therapies may harm patients who have atrial fibrillation, heart failure, and a rapid ventricular rate, although no large clinical trials have been performed to evaluate alternative therapies in such patients. In patients with clinical evidence of CHF, great caution should be exercised in the use of calcium channel and β-blockers because of their negative inotropic properties and because patients with CHF were generally excluded from efficacy trials. Evidence from 1 study of 37 patients with moderate-to-severe CHF (LOE 5)166 suggests that this risk may be less with diltiazem than with verapamil or β-blockers. Digoxin remains the only parenteral AV nodal–blocking drug with positive inotropic properties, but its usefulness is limited by its relative impotence and slow onset of action. One study of good-to-excellent quality (LOE 1),186 3 studies of good-to-excellent quality (LOE 2),47,48,59 and 2 studies of good quality (LOE 5)54,70 suggest that IV amiodarone is effective for rate control of
atrial fibrillation at “conventional doses” with no greater risk of conversion to sinus rhythm than digoxin or placebo, particularly in patients resistant to conventional heart rate control measures (LOE 5) or clinical shock (LOE 5). Other studies have found that IV amiodarone is effective for pharmacologic conversion of atrial fibrillation to sinus rhythm (see below); however, this agent should be used only within the first 48 hours of the onset of arrhythmia, only in patients who have been adequately anticoagulated or those in whom the possible thromboembolic risk from pharmacologic cardioversion is believed to be justified.

In patients with antegrade conduction of an AV accessory bypass tract (Wolff-Parkinson-White syndrome), extremely rapid ventricular responses may occur during atrial fibrillation. The refractory period of the antegrade accessory bypass tract, as opposed to that of the normal AV node, can be very short, resulting in ventricular rates of more than 300 beats/min. This extremely rapid, irregular ventricular rate may lead to degeneration to VT or VF. The anatomic substrate that supports high ventricular response rates is not sensitive to AV nodal–blocking drugs, β-blockers, or adenosine. In fact, digitalis, verapamil, diltiazem, and possibly IV β-blockers can cause a paradoxical increase in ventricular response rates during preexcited atrial fibrillation. A number of reasons for this increase in ventricular response have been suggested, including a decrease in the number of impulses conducted over the anatomic AV node, which provides for less concealed conduction to the ventricular insertion site of the bypass tract, thereby enhancing antegrade conduction via the accessory pathway from atrium to ventricle. Adenosine, calcium channel and β-blockers can also cause a fall in blood pressure, resulting in reflex adrenergic activation, which indirectly shortens the refractory period of the bypass tract. In addition, adenosine, possibly by its effect on shortening atrial refractoriness and by slowing AV nodal conduction, can cause acceleration of ventricular response rates and degeneration to VF. Thus, electrical cardioversion is usually preferred for such arrhythmias unless the duration of the arrhythmia or other clinical risk factors for thromboembolism (see below) suggest that immediate cardioversion is inadvisable or has proved ineffective in maintaining sinus rhythm.

When pharmacologic interventions do not provide adequate rate control, and conversion to sinus rhythm is unsuccessful, refer the patient for ablation of the His bundle and permanent pacing to avoid the downward spiral of tachycardia-induced cardiomyopathy. In patients with atrial fibrillation or flutter and rapid conduction across an accessory pathway, refer the patient for ablation of the accessory pathway.

In all instances of hemodynamically stable atrial fibrillation or flutter, conversion to normal sinus rhythm requires consideration of the duration of the arrhythmia. In patients with atrial fibrillation or flutter of 48 hours or less duration, anticoagulation is not required before cardioversion unless other risk factors necessitate it. When the duration of the arrhythmia is more than 48 hours or unknown, a minimum of 3 weeks of anticoagulation therapy before cardioversion and 4 weeks of anticoagulation therapy after successful cardioversion are recommended. If the presence of a left atrial thrombus is excluded by transesophageal echocardiography, patients who are adequately heparinized may undergo cardioversion, which should be followed by 4 weeks of anticoagulation therapy. Electrical cardioversion of atrial fibrillation or flutter to normal sinus rhythm remains supported by evidence evaluated for the 1992 guidelines and remains the technique of choice for cardioversion of patients with atrial fibrillation or flutter with preserved or significantly impaired LV function.
Once satisfactory rate control is achieved, a number of pharmacologic alternatives can be used for cardioversion (or to facilitate electrical cardioversion) of patients with preserved LV function, including parenteral ibutilide, procainamide, amiodarone, propafenone (NAUS), flecainide (NAUS), and sotalol (NAUS). The efficacy of all these agents depends on the duration of the arrhythmia; patients with atrial fibrillation or flutter of longer duration are generally less responsive to pharmacologic cardioversion than those in whom onset is more acute. The use of procainamide is supported by 1 study of excellent quality (LOE 1), 1,34 5 studies of good-to-excellent quality (LOE 2), 33,34,37,61,194 and 2 studies of fair quality (LOE 5), 38,39 Nevertheless, procainamide poses a risk of hypotension because of its vasodilatory properties. The efficacy of high-dose IV amiodarone therapy for pharmacologic conversion of atrial fibrillation to sinus rhythm was demonstrated by 2 studies of good quality (LOE 1), 53,186 4 studies of fair-to-good quality (LOE 2), 53,56,60,61 although it generally takes longer than propafenone or flecainide), and 4 studies of fair quality (LOE 5). 49,55,62,64 Other studies, including 3 with a LOE of 2,47,48,59 and 1 with a LOE of 5,51 found that conventional dosages of amiodarone are effective for rate control but not necessarily better than placebo for conversion of atrial fibrillation to sinus rhythm. The use of IV ibutilide for conversion of atrial fibrillation or flutter is supported by 3 studies of excellent quality (LOE 1),195-197 and 3 studies of good-to-excellent quality (LOE 2). 198-200 Ibutilide has also been shown to facilitate electrical cardioversion of atrial fibrillation (LOE 1)201 and antitachycardia pacing conversion of atrial flutter (LOE 2). 199 Ibutilide is associated with polymorphic VT in 3% to 8% of patients, particularly those with impaired LV function. The use of IV flecainide (NAUS) for cardioversion of atrial fibrillation or flutter is supported by 1 study with an LOE of 1,58 1 with an LOE of 2,202 and 2 with an LOE of 5. 138,203 The use of IV propafenone (NAUS) for cardioversion of atrial fibrillation or flutter is supported by 3 studies of good-to-excellent quality (LOE 1), 53,204,205 4 studies of good-to-excellent quality (LOE 2), 56,60,206,207 and 3 studies of fair quality (LOE 5). 208-210 The use of IV sotalol (NAUS) for cardioversion of atrial fibrillation or flutter is supported by 2 studies of fair quality (LOE 2), 202,211 1 of which demonstrated a high incidence of associated hypotension, and is not supported by 1 study of excellent quality (LOE 2). 145 The efficacy of IV disopyramide (NAUS) for atrial fibrillation was demonstrated by 1 study with an LOE of 5,86 1 study (LOE 2) of PSVT that included patients with atrial fibrillation and flutter. 109 and results extrapolated from 1 comparative study (LOE 2) in which disopyramide was used as the “default” treatment. 211 Data from a placebo-controlled study (LOE 1) of 96 patients suggest that IV dofetilide may also be an effective agent for pharmacologic conversion of atrial fibrillation or flutter, but this drug may pose a risk of torsade de pointes, which occurred in 3% of studied patients. 212

Patients with preexcited atrial fibrillation or flutter would be expected to be comparably responsive to antiarrhythmic agents used for pharmacologic cardioversion in the absence of an accessory pathway, although many of these agents have not been specifically evaluated in patients with preexcited arrhythmias (LOE 7).

In patients with impaired LV function, the negative inotropic effects of flecainide (LOE 5), 147 propafenone, sotalol, and procainamide and the proarrhythmic potential of ibutilide make these agents less desirable for pharmacologic cardioversion. IV amiodarone is the preferred agent in such patients, including patients with preexcited atrial fibrillation or flutter.

Efforts to slow the ventricular rate response or to convert atrial fibrillation to sinus rhythm can lead to profound bradycardia and even asystole, especially in patients with significant underlying conduction system disease or sick sinus syndrome. Consequently, it is advisable to have temporary pacing capability (transcutaneous or transvenous) or pharmacologic support (IV dopamine, isoproterenol, or atropine) available.

**Atrial flutter in children**

Atrial flutter is known to occur during the prenatal and neonatal periods in infants with a structurally normal heart. Although morbidity is common, the long-term prognosis is excellent. 213 Treatment of acute atrial fibrillation or flutter in children varies from DC cardioversion to digoxin, overdrive pacing, and administration of a Vaughan Williams classification IA drug. There are few published data on the use of other antiarrhythmic medications to treat this problem. In older children with congenital heart disease, atrial flutter or fibrillation often occurs after surgical repair. Typical examples include patients with D transposition of the great arteries who undergo an atrial switch operation to redirect the flow of systemic and pulmonic venous blood (Mustard or Senning procedure) and those with single-ventricle physiology after atrio pulmonary or cavopulmonary anastomoses (Fontan-type repair). Although numerous agents have been evaluated for long-term use in children with such conditions, there is little data on the use of pharmacologic agents for acute treatment; the data that are available are
largely extrapolated from studies of adults, as it is for newborns. Pertinent issues in children, as in adults, include impaired LV function and AV conduction.

PROPOSED GUIDELINES

Patients with hemodynamically unstable, rapid-response atrial fibrillation or flutter should be immediately electrically cardioverted regardless of the duration of the arrhythmia.

Pharmacologic rate control is the recommended initial therapy for stable, rapid atrial fibrillation or flutter (≥120 beats/min) regardless of its duration. Specific drug treatment should be selected on the basis of whether patients have evidence of impaired LV function or preexcitation (Wolff-Parkinson-White syndrome). Do not use adenosine to manage atrial fibrillation or flutter (it has an ultrashort duration of action).

In patients with preserved LV function, β-blockers (Class I) or calcium channel blockers are recommended for rate control. In patients with CHF, digoxin (Class IIb), diltiazem (Class IIb), or amiodarone (Class IIb) is recommended. In patients with preexcited atrial fibrillation or flutter, β-blockers, calcium channel blockers, adenosine, and digoxin are contraindicated for rate control (Class III); preferable agents for rate control are parenteral procainamide, amiodarone, propafenone (NAUS), flecainide (NAUS), or sotalol (NAUS) in patients without CHF, and amiodarone in patients with CHF. Evidence on the potential for pharmacologic conversion of atrial fibrillation by acute administration of antiarrhythmic agents (ie, parenteral procainamide, amiodarone, flecainide, propafenone, and sotalol) and the potential for these drugs to cause hypotension and thereby accelerate ventricular rate in patients with preexcited atrial fibrillation or flutter is controversial. This potential risk must be balanced against the benefit of the drugs.

Extrapolated data indicate that most antiarrhythmic agents used in adults can also be used in children. Verapamil is not recommended for infants.

Topic 8: Unusual Arrhythmias

OVERVIEW

Junctional tachycardia is defined as an automatic tachycardia originating in the AV node. P waves are either not seen during the tachycardia, or retrograde P waves may be inscribed shortly before or after the QRS complex.

JUNCTIONAL TACHYCARDIA: EVALUATION AND PROPOSED GUIDELINES

True junctional tachycardia is rare in adults. Apparent junctional tachycardia is most commonly misdiagnosed PSVT and should be treated according to the PSVT algorithm. True junctional tachycardia in adults is usually a manifestation of digitalis toxicity (which is best treated by withdrawal of digoxin) or exogenous catecholamine or theophylline toxicity (which is best treated by reduction or withdrawal of such infusions). If no apparent cause is found, symptomatic junctional tachycardia may respond to β or calcium channel blockers (this recommendation is based on data extrapolated from studies of the antisympa-
theic and nodal effects of such agents, LOE 7) or IV amiodarone (based on data extrapolated from pediatric studies, LOE 7).

In children, junctional tachycardia most commonly occurs after surgery. Available data (LOE 5) suggest it may respond best to IV amiodarone. Procainamide is reportedly useful when used with hypothermia.\textsuperscript{2,14} Studies of propafenone for postoperative junctional tachycardia in children suggest a significant incidence of hypotension.\textsuperscript{142} Electrical cardioversion is ineffective against automatic arrhythmias such as junctional tachycardia.

**INAPPROPRIATE SINUS TACHYCARDIA: OVERVIEW**

Sinus tachycardia is most commonly a reflex or compensatory response to an underlying condition such as pain, stress, anemia, fever, heart failure, thyrotoxicosis, infection, or exogenous catecholamine infusions and should not be treated without consideration of such primary causes. Inappropriate sinus tachycardia is defined as the presence of a rapid supraventricular rhythm of sinus origin without apparent explanation. Ectopic atrial tachycardia should be excluded by careful evaluation of the 12-lead ECG for P-wave configuration, performance of vagal maneuvers, or administration of adenosine.

**EVALUATION AND PROPOSED GUIDELINES**

If due to sinoatrial reentry, inappropriate reentrant sinus tachycardia may be treated similarly to PSVT. If due to an automatic mechanism, inappropriate sinus tachycardia should be treated with \( \beta \)-blockers (based on data extrapolated from studies of the antisym pathetic effects of such drugs, LOE 7). Electrical cardioversion is effective against reentrant sinoatrial tachycardia but not automatic forms of sinus tachycardia. Calcium channel blockers may also be of benefit (based on data extrapolated from studies of the negative chronotropic and dromotropic effects of such drugs on the sinus and AV nodes, LOE 7), as may amiodarone (based on its \( \beta \) and calcium channel–blocking properties). There is little published data on treatment of inappropriate tachycardia in children. Treatment of children follows the same principles used for treatment of adults and is based on data extrapolated from the limited studies conducted in adults.

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