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Pharmacologic Management of Arrhythmias

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Abstract. The role of antiarrhythmic drugs in the management of children with arrhythmias has changed due to the rapid development of radiofrequency ablation. Moreover, the release of new drugs and a better insight into the electrophysiologic mechanisms of arrhythmias have changed former patterns of drug management. However, because of lack of controlled trials, arrhythmia management in pediatrics is still mainly based on clinical studies and individual experience. Within these limitations, I attempt to give practical recommendations for the management of the different arrhythmias seen in children based on the latest developments in this area.

Key words: Antiarrhythmic drugs — Pediatrics — Supraventricular tachycardia — Ventricular tachycardia

The role of antiarrhythmic drugs in the management of arrhythmias in children is changing, particularly because of the rapid development of radiofrequency (RF) catheter ablation for supraventricular tachycardia (SVT) as well as for atrial flutter and idiopathic ventricular tachycardia (VT). Other developments are the release in recent years of new antiarrhythmic drugs and the remarkable increase in the understanding of the electrophysiological substrate of the arrhythmia and its markers on the surface electrocardiogram (ECG). Therefore, antiarrhythmic drugs may now, more than in the past, be chosen according to the mechanism of the specific arrhythmia. However, the choice of an antiarrhythmic drug is still mainly based on individual experience and clinical studies. In contrast with adult cardiology, there are no controlled trials in children to guide therapy. Much of what we know of effects—proarrhythmia or side effects—comes from adult studies and is surely not always applicable to growing children with normal hearts or congenital heart disease.

Within these limitations, I try to give some practical recommendations for the pharmacologic management of arrhythmias in children based on the knowledge of newer drugs and the mechanism of the arrhythmia.

Treatment Options

In the decision of how to treat, the risk and the burden of the arrhythmia should be carefully weighed against the risk and the burden of drug treatment.

No Treatment

In children without preexcitation and with a structurally normal heart, paroxysms of SVT are annoying but not risky. In this category of patients, if the mechanism of SVT is explained to child and parents if they are reassured about the risk and vagal maneuvers are taught, many children with sporadic SVT and their families prefer no drug therapy and do well. Children with frequent premature beats do not need treatment and some children with asymptomatic idiopathic VT also do well without any treatment.

Nonpharmacologic Treatment

In recurrent or incessant SVT and in some patients with idiopathic VT, drug treatment may only function as a bridge to RF ablation at a safer age. In children with congenital heart disease, correction of abnormal hemodynamics and/or pacing may be an important adjunctive therapy to antiarrhythmic drugs. In acute termination of an arrhythmia D/C cardioversion or transesophageal pacing may be safer than intravenous drugs.

Periodic Treatment

Some children with sporadic but long-lasting SVT may be treated through one oral dose of a drug at the onset of the tachycardia. SVT induced by playing sports may be managed by one dose of a beta-blocker before the game.

Chronic Treatment

Only a few arrhythmias need life-long treatment: long QT syndrome, arrhythmogenic right ventricular dyspla-

Table 1. Vaughn Williams antiarrhythmic drug classification

Class	Action	Agents
I	Sodium channel blockers	
A	Prolong action potential duration	Quinidine, procainamide, disopyramide
B	No change or shortening of action potential duration	Lidocaine, mexiletine, phenytoin
C	Mild prolongation of action potential duration	Flecainide, encainide, propafenone,
II	Beta-adrenergic blockers	Propranolol, atenolol, metoprolol, nadolol
III	Potassium channel blockers Prolong action potential duration	Amiodarone, sotalol, ibutilide
IV	Calcium channel blockers	Verapamil, diltiazem
Others		Adenosine, digoxin

sia, catecholaminergic polymorphic VT, and arrhythmias after surgery for congenital heart disease or in children with cardiomyopathy. The disadvantages of chronic treatment are the incidence of side effects in the long term and the lack of compliance in teenagers. The manifestation of arrhythmias in children changes as they mature and the arrhythmia may even disappear. Thus, drug treatment should be stopped after some years in order to determine if the tendency to arrhythmia occurrence has resolved, except of course in patients at risk.

Pharmacology

It is beyond the scope of this article to cover all pharmacologic aspects [26]. However, I discuss two aspects: the risk of proarrhythmia and the interaction of drugs. The classification of antiarrhythmic drugs as described by Vaughn and Williams is used here (Table 1).

Proarrhythmia is mostly seen in class IA, IC, and III drugs. The challenge is to identify the child at risk [27]. Class IC drugs are known to create more proarrhythmia in children with structural heart disease. Torsade de pointes by class IA and III drugs is more often found in patients with hypokalemia, hypomagnesemia, bradycardia, a longer baseline QT duration, a higher plasma concentration of the drug (e.g., *d,l*-sotalol), and concomitant use of QT prolonging drugs (erythromycin, antihistamines, etc.).

Some antiarrhythmic drugs influence each other's plasma level (Table 2). Less well recognized is the influence of other drugs on the metabolism of the antiarrhythmic agent [1]. For example, through the cytochrome P450 enzyme system, the metabolism of lidocaine, flecainide, verapamil, propranolol, and propafenone is inhibited by concomitant use of cimetidine. On the other hand, the metabolism is induced by phenobarbital, phenytoin, and rifampin. Food products such as grapefruit juice may also substantially increase the drug levels of most antiarrhythmic drugs.

Monitoring

Drugs that carry the risk of development of proarrhythmia are initiated in a monitored hospital setting, especially in children with structural heart disease. At baseline and during treatment an ECG should be analyzed (PR, QRS, and QT duration), as should an echocardiogram (ventricular function) and electrolyte balance (K^+, Mg^{2+}). Plasma levels may be checked if appropriate for the drug or to prevent toxic values. A steady state is often reached after four or five times the drug's half-life. Proarrhythmia is frequently, but not exclusively, seen in the early period of treatment. Because of the (reverse) use dependence of many drugs (effect dependent on heart rate), Holter monitoring and an exercise test should be performed to judge the maximal effect of the drug during bradycardia (e.g., *d,l*-sotalol) or tachycardia (e.g., class IC).

Antiarrhythmic drugs

Adenosine

Action. Adenosine is an endogenous purine nucleoside with a direct effect on potassium conductance and an indirect antiadrenergic action [4, 32]. This results in sinus node and atrioventricular (AV) node depression and shortening of atrial refractoriness.

Clinical Use. Because of its extremely short half-life of 7 to 10 seconds, adenosine is the drug of choice for termination of paroxysmal SVT. Atrial tachycardia and idiopathic VT caused by triggered activity may also terminate after injection of adenosine. Adenosine terminates SVT in 90% to 100%, but SVT reinitiates in 25% to 30%.

Side Effects. All side effects are of short duration. Patients may develop hypotension, facial flushing, chest discomfort, or bronchospasm. Sinus arrest, premature atrial contractions and premature ventricular contractions

Table 2. Current antiarrhythmic drugs

Drug	Dose oral/day	Doses/day	Dose i.v.	Side effects	LV function	ECG	Half-life	Interactions
Class IA								
Procainamide	15–50 mg/kg	3×	L: <1 year 7 mg/kg, >1 year 15 mg/kg (1 hour) M: 20–80 μg/kg/min	Lupus, nausea	⇓	PR⇑, QRS⇔, QT⇑ AV block Torsade de pointes	3–4 hours 6–7 hours NAPA	Cimetidine, amiodarone, trimethoprim
Class IB								
Lidocaine			L: 1 mg/kg max. 3× M: 0.5–1.5 mg/kg/hour	Seizures, apnea	⇒		2 hours	Propranolol cimetidine
Mexiletine	6–15 mg/kg	3×		Nausea	⇒		12 hours	Antacids cimetidine
Class IC								
Propafenone	200–600 mg/m^2 <15kg: 10–20 mg/kg >15kg: 7–15 mg/kg	3×	L: 1 mg/kg (10 min) M: 4–7 μg/kg/min	Blurred vision, nausea	⇓	PR⇑, QRS⇔AV block SVT and VT acceleration	5–7 hours 10–30 hours (slow metabolizer)	Digoxin, beta-blockers, Ca antagonist
Class II								
Propranolol	1–3 mg/kg	3–4×		Asthma, fatigue, nightmares, Raynaud	⇓	PR⇑, bradycardia, AV block	3–6 hours	Cimetidine, quinidine, propafenone
Atenolol	1–2 mg/kg	1–2×		Idem	⇓	PR⇑, bradycardia, AV block	8–10 hours	Cimetidine quinidine, propafenone
Esmolol			L: 500 μg/kg M: 50–200 μg/kg/min	Hypotension	⇓	PR⇑, bradycardia, AV block	7–10 minutes	Cimetidine, quinidine, propafenone
Class III								
d,l-Sotalol	2–8 mg/kg 90–200 mg/m^2	2×	0.5–1.5 mg/kg (10 min)	Fatigue, dizziness, dyspnea	⇓	PR⇑, QT⇑, AV block, bradycardia, torsade de pointes	10–20 hours	
Amiodarone	L: 10–20 mg/kg M: 2–5 mg/kg	2× 1×	L: 5 mg/kg (30 minutes) M: 10–15 mg/kg/day	Skin, Cornea, thyroid, lungs	⇒	PR⇑, QRS⇔, QT⇑, bradycardia, torsade de pointes, AV block	3–15 weeks	Digoxin, warfarin, class I, II, IV
Ibutilide			10 μg/kg (10 minutes)			QT⇑, bradycardia, torsade de pointes	6–9 hours	
Class IV								
Verapamil	4–8 mg/kg	3×	0.1–0.15 mg/kg (2–5 minutes) Not in infants	Oral: hypotension, constipation	⇓	PR⇑, bradycardia, AV block	3–5 hours	Beta-blockers, digoxin, midazolam
Miscellaneous								
Adenosine			50–250 μg/kg	Hypotension, flushing, chest pain, bronchospasm		PACs, PVCs, sinus arrest, A fibrillation, PR⇑	7–10 seconds	Disopyramide, theophylline

i.v., intravenous; LV, left ventricular; L, loading dose; M, maintenance dose; PAC, premature atrial contraction; PVC, premature ventricular contraction; ⇑, prolonged; ⇔, widened; ⇓, depressed; ⇒, no influence.

as well as atrial fibrillation may develop. Rebound increase of sympathetic activity may increase the rate of the reinitiated tachycardia or enhance AV conduction in atrial flutter, causing hemodynamic instability. Adenosine may only be given if resuscitation equipment is present.

Interaction. Dipyridamole and diazepam inhibit the metabolism of adenosine, so the dose should be reduced. Conversely, theophylline and caffeine decrease the action of adenosine, forcing a higher dose. There is a report of ventricular fibrillation after a dose of adenosine in a neonate with a concealed Wolff–Parkinson–White (WPW) syndrome treated with digoxin [18]. Therefore, the physician giving adenosine should be ready to manage other arrhythmias that may result from adenosine use.

Dose. 50 to 100 μg/kg i.v.; if the initial dose is ineffective repeat the dose every 2 minutes with increments of 50 μg/kg up to a maximal dose of 250 μg/kg (maximum dose for adults: 12 mg). Because of the short life, the injection is given as a rapid bolus followed by saline flush via a central vein.

Amiodarone

Action. Amiodarone is a class III drug with its main electrophysiological effect being the prolongation of the refractory period of all cardiac cells [19, 21]. Additionally, it has class IB, II, and IV effects. Oral amiodarone is one of the rare antiarrhythmic drugs that does not typically depress cardiac function. It is also rare in its prolonged action, with a half-life of 3 to 15 weeks. The clinical effect is only detectable several days after the start of oral treatment. The metabolite desethylamiodarone is also electrophysiologically active. Plasma levels correlate poorly with efficacy.

Clinical Use. Amiodarone is very effective in most supra- and ventricular arrhythmias. Because of its important side effects, it is mostly reserved for drug refractory or life-threatening arrhythmias. However, if used for short term and at a low dose, amiodarone is safe and effective. Intravenous amiodarone has been used successfully in postoperative arrhythmias as well as junctional ectopic tachycardia (JET) and VT [25]. Chronic therapy with amiodarone may be chosen in postoperative intraatrial reentrant tachycardias or VT in an abnormal heart (cardiomyopathy and myocarditis).

Side Effects. These are less frequent than in adults but still range from 8% to 33% in pediatric series. Younger than the age of 10 years side effects occur in 4% and older than the age of 10 years up to 44%. Systemic effects are corneal deposits, photosensitivity, skin pigmentation, hypo- and hyperthyroidism, and pulmonary interstitial fibrosis. Sinus bradycardia, atrioventricular block, and torsade de pointes may occur. Short-term use and low dose minimize the side effects. If long-term treatment is necessary in older children, one has to consider the high incidence of side effects of amiodarone when children mature [8]. Intravenous amiodarone may cause hypotension, which may be markedly reduced by the use of the aqueous form of amiodarone (study under way).

Interaction. Amiodarone interacts with the metabolism of many drugs, resulting in an increase in plasma level of digoxin, warfarin, procainamide, quinidine, disopyramide, flecainide, phenytoin, beta-blockers, and calcium blockers.

Dose: Oral loading dose, 10 to 20 mg/kg/day in two doses; maintenance dose, 2 to 5 mg/kg/day in one dose. Intravenous loading dose, 5 mg/kg over 30 minutes; maintenance dose, 10 to 15 mg/kg/day.

Ibutilide

Action. In contrast with *d,l*-sotalol and amiodarone, ibutilide is a pure class III drug which acts predominantly by prolonging myocardial action potential duration [5]. The oral bioavailability is very low, so ibutilide can only be used intravenously. It is mainly excreted in the urine.

Clinical Use. Ibutilide is mainly used for conversion of atrial flutter and fibrillation. It is more effective in atrial flutter than in atrial fibrillation and its effect is superior to that of *d,l*-sotalol or procainamide. Clinical experience in adults is limited and efficacy and safety in children have not been established. Theoretically, it might be a good drug for conversion of direct and late postoperative atrial flutter. It should be administered in a monitored setting such as an intensive care unit.

Side Effects. Ibutilide causes polymorphic VT in 4% to 8% due to its effect on QT duration. This risk seems to be higher for pure class III drugs, such as ibutilide, dofetilide, and *d*-sotalol than for non-pure class III drugs such as *d,l*-sotalol and amiodarone. Bradycardia, heart failure, hypokalemia, and prolonged QT duration elevate the risk of developing torsade de pointes. Ibutilide has minimal hemodynamic effects, but no studies have been performed in hemodynamically unstable patients.

Interaction. None important. Use cautiously or avoid use in patients pretreated with class III drugs.

Dose. Intravenous 0.01 mg/kg over 10 minutes. This dose may be repeated once.

Propafenone

Action. Propafenone is a class IC drug [9, 11]. Its electrophysiological effects are comparable with those of flecainide except for its beta-blocking and weak calcium channel-blocking properties. It prolongs the refractory periods of atrium and ventricle; it slows the conduction

over AV node and accessory pathways and depresses automaticity. Propafenone is metabolized by the liver, but there is a nonlinearity of dose and plasma level, with the risk of accumulation at higher doses. Therefore, there is also no clear relationship between drug level and efficacy.

Clinical Use. Propafenone is effective in atrioventricular reentrant tachycardia (AVRT) due to an accessory connection, or atrioventricular nodal reentrant tachycardia (AVNRT), but it is also very effective in more rare and more difficult to treat tachycardias such as atrial ectopic tachycardia (AET), permanent junctional reciprocating tachycardia (PJRT), postoperative JET, and chaotic atrial tachycardia [7].

Side Effects. The systemic side effects are mainly blurred vision and gastrointestinal complaints and are dose related. The electrophysiological side effects are of more importance. Among children of the European Retrospective Multicenter Study, 5 of 772 (0.6%) experienced cardiac arrest or sudden death and 1.9% developed proarrhythmia [10]. The electrophysiological adverse effects were significantly more common in children with structural heart disease. However, these numbers are superior to those of other IC drugs such as encainide and flecainide. Propafenone treatment should be started in a monitored hospital setting in children with structural heart disease.

Interaction. Propafenone elevates the plasma level of digoxin, increases the effect of beta-blocking drugs, and intensifies the negative inotrope effect of calcium antagonists.

Dose. Oral, 200 to 600 mg/m^2 or <15 kg: 10 to 20 mg/kg; >15 kg: 7 to 15 mg/kg in three doses. Intravenous: 1.0 mg/kg over 10 minutes; maintenance dose 4 to 7 μg/kg/min.

d,l-Sotalol

Action. d,l-Sotalol is a nonselective beta-blocking drug with additional class III properties [22]. It therefore combines the beta-blocking effects on SA and AV node with prolongation of the action potential duration and lengthening of the refractory periods of all cardiac muscle. The beta-blocking effect dominates at a low dose and the class III effect at a high dose. The drug is mainly excreted by the urine. The plasma half-life is 10 to 20 hours with a peak plasma concentration at 2 to 4 hours. Therapeutic drug level (peak) is 0.8 to 5 mg/L.

Clinical Use. Oral *d,l*-sotalol is effective in the prevention of Supraventricular tachycardia (SVT), not only AVRT or AVNRT but also the more difficult forms such as PJRT. It is also very effective in the prevention of atrial flutter, for example, in patients after extensive atrial surgery [2]. In adults it is also used for VT, but the experience in children is very limited. The same applies for the use of intravenous *d,l*-sotalol. *d,l*-Sotalol passes the placental barrier freely and has been used successfully for treatment of fetal SVT in a small series of patients [28].

Side Effects. Fatigue, dizziness, and dyspnea are the main side effects. Proarrythmia occurred in Pfammatter et al.'s [23] study in 7 of 71 patients—mainly bradycardia and in one case torsade de pointes. Female gender, ventricular arrhythmias, congestive heart failure, and a high *d,l*-sotalol dose increase the risk of the development of torsade de pointes in adults [16]. Proarrhythmia mostly develops in the first days of treatment. Therefore, *d,l*-sotalol should be initiated in a monitored hospital setting, especially in children with structurally heart disease.

Interaction. There are no important interactions with other drugs. Of course, it should not be combined with other class III drugs; side effects may be exacerbated with concurrent beta-blocker use. Hypokalemia and a low magnesium level increase the risk of developing proarrhythmia.

Dose. Oral, 2 to 8 mg/kg/day in two doses or 90 to 200 mg/m^2. Intravenous, 0.5 to 1.5 mg/kg over 10 minutes.

Types of Arrhythmias

Paroxysmal Supraventricular Tachycardia

The mechanism of SVT should be determined before treatment using the 12-lead ECG and/or esophageal leads during tachycardia. After conversion to sinus rhythm, the ECG should be checked for the presence of preexcitation.

Termination. If vagal maneuvers including ice application fail, adenosine is the drug of choice. The very short half-life of adenosine favors its use for acute termination. Verapamil is also very effective (80–95%) but may cause severe hemodynamic deterioration in infants and in patients with heart failure and is therefore contraindicated in this group.

Prevention. Many drugs that affect the electrophysiological properties of the AV node or the accessory connection may be used for prevention of recurrences, such as beta-blockers, verapamil, digoxin, propafenone, and *d,l*-sotalol. Digoxin and verapamil should not be used in WPW syndrome due to the risk of accelerating antegrade conduction over the accessory connection, resulting in a fast conduction of atrial fibrillation and potentially triggering ventricular fibrillation. In WPW a beta-blocker is the drug of choice. In AVNRT, digoxin combined with a beta-blocker or verapamil is a good first choice. Amiodarone is effective, but it should be reserved for drug refractory cases or for short-term use, such as in neonatal

SVT if beta-blockers fail. Drug refractory cases are extremely rare and, in the era of radiofrequency (RF) ablation, intervention should be considered, at least in children older than 4 or 5 years of age.

Incessant Supraventricular Tachycardia

The mechanism of incessant SVT is usually AET, PJRT, or, less often, chaotic atrial tachycardia. Propafenone and *d,l*-sotalol are effective in two thirds of children with AET [14]. Amiodarone may be used in refractory cases. The same applies to PJRT, although less data are available. If there is no spontaneous resolution of the tachycardia, drugs only serve as a bridge to RF ablation at an older age.

Junctional Ectopic Tachycardia

Postoperative JET is a life-threatening arrhythmia. In addition to hypothermia and atrial pacing, antiarrhythmic drugs are essential to overcome the critical first days after surgery. Procainamide, amiodarone, and propafenone have proven effective [7, 25, 31]. In congenital JET, amiodarone has also proven very effective (78%), as has propafenone [20, 30]. Other drugs were ineffective in both types of JET.

Atrial Flutter

Termination. Termination of atrial flutter is mostly achieved by electrical means either by DC cardioversion or by transesophageal pacing, and it is the preferred treatment for neonates [15]. If the patient is hemodynamically stable, ibutilide or procainamide (risk of hypotension) may be given intravenously to convert atrial flutter to sinus rhythm. Conversion may also be achieved orally with *d,l*-sotalol, propafenone, or a class IA drug such as quinidine.

In children with structural heart disease it is urgent to terminate atrial flutter (or intra-atrial reentrant tachycardia) promptly due to the risk of clot formation and emboli. Furthermore, a prolonged period of an elevated heart rate may lead to hemodynamic deterioration. Another reason to try to regain sinus rhythm as soon as possible is the electrophysiological remodeling of the atria that is caused by prolonged atrial flutter. Remodeling is responsible for a high early recurrence rate. If the duration of atrial flutter is >48 hours or unknown, and the patient can tolerate persistent tachycardia hemodynamically (i.e., not a patient with single ventricle physiology), the patient should undergo anticoagulation therapy for 3 weeks while the atrioventricular conduction of atrial flutter is slowed by antiarrhythmic drugs. Anticoagulation should be continued for 4 weeks after successful cardioversion.

Prevention. The recurrence rate of atrial flutter in neonates is very low and chronic therapy is seldom necessary. On the other hand, late postoperative atrial flutter or intra-atrial reentry recur easily and chronic therapy is often indicated. Digoxin, beta-blockers, and calcium antagonists slow atrioventricular conduction but are ineffective in the prevention of atrial flutter. Class III drugs such as *d,l*-sotalol and amiodarone are effective in 70% to 80% of cases [2, 3, 29]; the same percentage is found after treatment with propafenone. The efficacy of class IA drugs is about 50%. The efficacy after long-term treatment is less for all drugs. If class IC drugs are used, the combination of an atrioventricular node-slowing drug should be considered to prevent 1:1 conduction of slow atrial flutter. In patients with diminished ventricular function, the risk of proarrhythmia while using IC drugs is higher. The side effects of amiodarone make the use of this drug for long-term treatment less attractive. Often, pacemaker implantation is needed to treat spontaneous or induced bradycardia, which enhances the development of atrial flutter.

Evaluation of hemodynamic abnormalities as a cause of atrial flutter is always indicated, and if positive a surgical intervention possibly combined with arrhythmia cryoablation should be performed. Radiofrequency ablation of the atrial reentry circuit(s) is another good option, although the recurrence rate after ablation is still high.

Ventricular Tachycardia

Termination. Vagal maneuvers may sometimes stop VT, making the effort worthwhile. If the patient is unstable, prompt DC cardioversion is performed. In the stable patient intravenous drug conversion may be tried. Lidocaine is a safe drug but not highly efficacious in terminating sustained VT. Procainamide and amiodarone are more effective in this setting. In torsade de pointes, intravenous magnesium sulfate is the drug of choice, followed by isoproterenol or right ventricular pacing. Idiopathic VT originating from the right ventricular outflow tract (left bundle branch block and inferior axis morphology) can often be terminated with adenosine [13]. Idiopathic VT arising from the left ventricle (right bundle branch block and left axis morphology) often responds well to intravenous verapamil [6].

Prevention. In decision making about prophylactic treatment of VT, two factors are of importance: the presence of symptoms and the prognosis of that particular type of VT. In the prognosis the presence or absence of structural heart disease plays a major role [24]. Table 3 gives suggestions for the use of antiarrhythmic drugs in the prevention of VT recurrence, taking into account the

Table 3. Medication for prevention of ventricular tachycardia in the structurally normal and abnormal heart

Type of ventricular tachycardia	Prophylactic treatment
Normal heart	
Accelerated idioventricular rhythm	No treatment
Idiopathic right ventricular outflow tract tachycardia	No treatment or beta-blocker Second choice: sotalol Consider: radiofrequency ablation
Idiopathic verapamil sensitive tachycardia (left ventricle)	No treatment or beta-blocker or verapamil Consider: radiofrequency ablation
Exercise-induced ventricular tachycardia (VT)	Beta-blocker
Cathecholaminergic polymorphic VT	Beta-blocker life-long [12]
Torsade de pointes in long QT syndrome	Beta-blocker
Abnormal heart	
After surgery for congenital heart disease	Correct hemodynamics Beta-blockers, sotalol, amiodarone
Myocarditis	Amiodarone
Dilated or hypertrophic cardiomyopathy	Amiodarone
Mitral valve prolapse	Beta-blocker
Arrhythmogenic right ventricular dysplasia	Sotalol, amiodarone, or combination [17]

absence or presence of structural heart defects and the specific type of ventricular arrhythmia or the specific type of heart disease.

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