INTRODUCTION AND EPIDEMIOLOGY — Supraventricular tachycardia (SVT) can be defined as an abnormally rapid heart rhythm originating above the ventricles, often (but not always) with a narrow QRS complex; it conventionally excludes atrial flutter and atrial fibrillation [1].

The prevalence of SVT is not well defined, but is estimated to be between one in 250 and 25,000 children [2,3]. The prevalence is much higher in critically ill children and adults with congenital or acquired heart disease treated in a pediatric cardiac intensive care unit. In one series of 789 admissions of 629 patients aged one day to 45 years, arrhythmias occurred in 29 percent [4]. The most common arrhythmias were nonsustained ventricular tachycardia and SVT, occurring in 18 and 13 percent of admissions, respectively.

The two most common forms of SVT in children are atrioventricular reentrant tachycardia (AVRT), including the Wolff-Parkinson-White (WPW) syndrome, and atrioventricular nodal reentrant tachycardia (AVNRT). The relative frequency of these arrhythmias was addressed in a report of 137 infants, children, and adolescents who underwent careful electrophysiologic testing. AVRT accounted for 73 percent and AVNRT for 13 percent (almost all cases of AVNRT occurred after two years of age) [5]. The remaining 14 percent were due to primary atrial tachycardias.

A similar distribution of causes was noted in another report of 346 patients (mean age 14 years) who had undergone radiofrequency catheter ablation for an arrhythmia [6]. AVRT and AVNRT accounted for 72 and 9 percent, respectively, with various other arrhythmias comprising the remaining 19 percent.

The majority of patients with SVT have structurally normal hearts. SVT also may be associated with congenital heart disease (CHD) or follow surgical repair of CHD. In the above series of 346 patients, 21 percent had CHD [6]. In this and other reports, CHD was more common in children with WPW syndrome than AVNRT (20 to 32 versus 7 percent) [6-8].

The management of SVT in children will be reviewed here. Two major issues will be addressed: acute management to terminate the arrhythmia, and chronic therapy to prevent recurrence. The clinical features of the different types of SVT are discussed separately. (See "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia").

ACUTE MANAGEMENT — Acute management of the child who presents in SVT can be a challenge because the exact mechanism of the tachycardia often is unknown. The treatment strategy depends upon the patient's presentation and clinical status (hemodynamically stable or unstable). The approach consists of initiating therapy while continuing to assess the patient's condition (show table 1). The following discussion is generally in agreement with the Pediatrics Advanced Life Support (PALS) guidelines published by the American Heart Association in collaboration with the International Liaison Committee on Resuscitation (show algorithm 1) [9].

An infant or child who presents with a tachyarrhythmia should have an immediate hemodynamic assessment and a 15 lead ECG (12 standard leads plus leads V3R and V4R [right sided leads analogous to V3 and V4 on the left] and V7 [left posterior axillary line at V4 level]). Continuous ECG monitoring during therapeutic maneuvers provides insight into the cause of tachycardia and helps in the planning of chronic therapy.

Hemodynamic assessment — The most important initial clinical determination to make in children presenting with a tachyarrhythmia is whether signs and symptoms related to the rapid heart rate are present. These include hypotension, heart failure, signs of shock, pallor, or decreased level of consciousness. Signs in infants may include irritability, tachypnea, and poor feeding.
Affected children can be asymptomatic except for palpitations, or may be unconscious with signs of cardiovascular collapse. The acute management depends whether the patient is hemodynamically stable or not (show table 1).

Hemodynamically unstable — Unstable patients with hemodynamic compromise, such as unconsciousness or shock with severe heart failure, require immediate termination of the tachyarrhythmia.

Cardioversion — Direct current cardioversion at 0.5 to 2.0 J/kg should be performed [9,10]. A narrow complex tachycardia should be converted in synchronous mode in which a shock is not delivered during the vulnerable repolarization period; this will avoid possible precipitation of ventricular fibrillation. (See "Basic principles and technique of cardioversion and defibrillation").

Cardioversion in children may be performed with pediatric electrode paddles (surface area 21 cm2). However, the use of adult electrode paddles (surface area 83 cm2) results in lower transthoracic impedance and therefore higher current flow, which may be preferable in children over 10 kg [11].

In general, the child should be given adequate sedation or general anesthesia before the procedure [12,13]. Rarely, a child is too critically ill to delay the procedure for administration of general anesthesia.

Diagnostic evaluation — The diagnostic evaluation of a child with a tachyarrhythmia is discussed in detail separately. The following findings support the presence of an SVT [9]:

- History incompatible with sinus tachycardia
- P waves absent or abnormal
- Heart rate does not vary with activity
- The presence of abrupt changes in heart rate
- Rate usually >220 beats/min in infants and >180 beats/min in children

Establishing the diagnosis of the two main causes of SVT, AVRT and AVNRT, is discussed separately. (See "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia", section on Diagnosis).

Vagal maneuvers — In children who have mild or no symptoms, vagal maneuvers should be attempted while supplies and personnel are assembled to proceed to medical therapy, if needed. These maneuvers should be performed while the ECG is continuously monitored. The electrocardiographic pattern seen during termination of the tachycardia can help determine its mechanism.

The vagal maneuver usually used in infants or young children is application of a bag filled with ice and cold water over the face for 15 to 30 seconds. This elicits the diving reflex, frequently interrupting the arrhythmia [14]. This maneuver is successful in 30 to 60 percent of cases [10,15,16].

Another method that may be successful in infants is rectal stimulation using a thermometer. In older children, bearing down (Valsalva maneuver) for 15 to 20 seconds provides vagal stimulation.

Carotid massage and orbital pressure should not be performed in children.

Antiarrhythmic drugs — If the vagal maneuver does not convert SVT that is hemodynamically stable to normal rhythm, an intravenous catheter should be placed for the administration of antiarrhythmic drugs. The most commonly used pharmacologic agent for acute management of SVT is adenosine; procainamide and amiodarone are sometimes given for tachycardia that is refractory to adenosine [9].

Digoxin is not usually used because of the delay in achieving therapeutic levels and the narrow therapeutic margin with the risk of serious toxicity [10]. In addition, digoxin should not be given if WPW is suspected, since it may potentiate accessory pathway conduction.
Adenosine — Adenosine is considered the drug of choice for acute medical conversion of SVT (show algorithm 1 and show table 1 and show figure 1) [9,17,18]. Adenosine interacts with A1 receptors on the surface of cardiac cells; the resulting effects include slowing of the sinus rate and an increase in the AV nodal conduction delay [19]. This interrupts the reentrant circuit of tachycardias that require the AV node for reentry, which account for the great majority of cases of SVT in children [5,6].

For intravenous adenosine administration, the patient should be supine and should have ECG and blood pressure monitoring. The drug is administered by rapid intravenous injection over one to two seconds at a site as close to the central circulation as possible. This is followed immediately by a 5 mL normal saline flush to speed delivery to the heart, which is needed because the drug is rapidly metabolized to an inactive form.

The usual initial dose is 0.1 mg/kg; if no response is seen within two minutes, the dose is doubled [20,21]. An alternative regimen consists of an initial bolus of 0.05 mg/kg; if no response is seen within two minutes, the dose is increased by 0.05 mg/kg increments every two minutes until termination of the arrhythmia or a maximum dose of 0.25 to 0.35 mg/kg or 12 mg is given.

The average effective dose is approximately 0.13 mg/kg [22]. However, because adenosine is metabolized by an enzyme on the red cell surface, a higher dose is required when given through a peripheral compared to a central vein [9].

Adenosine terminates 80 to 95 percent of episodes of AVRT, which accounts for almost three-quarters of episodes of SVT [5,6], and approximately 75 percent of episodes due to other causes of SVT [20,22,23]. Early recurrence of the SVT after termination occurs in 25 to 30 percent of cases [20,22].

Vagal stimulation caused by adenosine is brief and self-limited. The onset of effect is almost immediate, and always within 30 seconds of the infusion. The half-life is less than 5 to 10 seconds. The effects of adenosine are diminished by methylxanthines such as caffeine or theophylline.

Side effects, including flushing, nausea, vomiting, vague feeling of discomfort, chest pain, and dyspnea are not uncommon with adenosine, but usually resolve rapidly [20,22-25]. Serious side effects, such as arrhythmias, are rare [23]. Adenosine can precipitate atrial fibrillation, although this usually terminates spontaneously [22,26]. In a series of 38 children with SVT, significant complications occurred in four: atrial fibrillation, ventricular tachycardia, apnea, and one minute of asystole [22].

A particular concern is that, in patients with WPW, AF can degenerate into ventricular fibrillation. As a result, caution should be used when giving adenosine if WPW is a possible mechanism, and emergency resuscitation equipment should be available [26]. Adenosine should be avoided in patients with a wide QRS complex tachycardia since it can provoke severe hemodynamic deterioration in those who have ventricular tachycardia rather than an SVT.

Bronchospasm has been reported with adenosine in patients with asthma [27,28], but is not common in other children [23]. The mechanism is uncertain but may be related to stimulation or enhancement of mast cell-derived mediators [27].

Verapamil — Verapamil is an effective acute therapy to slow AV nodal conduction and terminate the arrhythmia in older children with SVT. Verapamil is administered as an intravenous infusion in a dose of 0.1 to 0.3 mg/kg with a maximum dose of 10 mg.

Adenosine is preferred for first-line therapy because of its very short duration of action. In addition, there are a number of settings in which verapamil should not be used:

- In infants less than one year of age because it may cause apnea, hypotension, bradycardia, and cardiovascular collapse [29,30]. The mechanism of this complication may be a poorly developed sarcoplasmic reticulum in infants, so that myocardial contractility depends solely on calcium channels.
- In children with heart failure [10,29].
In children suspected to have WPW, since it may potentiate conduction down an antegrade conducting pathway and provoke ventricular fibrillation [10].
In children with a wide QRS complex tachycardia since it can provoke severe hemodynamic deterioration in those who have ventricular tachycardia rather than an SVT [31].

For these reasons, the Pediatric ALS guidelines discourage the use of verapamil [9].

Procainamide — SVT that is refractory to adenosine may respond to procainamide. Procainamide is a class IA antiarrhythmic drug that acts primarily by inhibiting phase 0 (sodium-dependent) depolarization and slows atrial conduction. Unlike adenosine and verapamil, procainamide acts by slowing conduction within the myocardium itself, rather than by blocking reentry at the AV node. As a result, procainamide may be used safely in patients with WPW without the risk of provoking accessory pathway conduction.

Procainamide is administered intravenously. In patients under one year of age, a loading dose of 7 to 10 mg/kg is given over 30 to 45 minutes. In older children, the loading dose is 15 mg/kg. This is followed by a continuous infusion of procainamide starting at 40 to 50 mcg/kg per minute. Plasma levels should be measured four hours after completion of the loading dose, during the maintenance infusion.

Negative inotropic effects may occur following the administration of procainamide. In addition, procainamide can prolong the QT interval and therefore should not be given with other drugs that have the same effect.

Amiodarone — Amiodarone is a class III antiarrhythmic agent. The intravenous preparation, which is used for the acute therapy of SVT, prolongs the refractory period of the AV node and, to a lesser degree, the duration of the action potential and the refractory period of both atrial and ventricular myocardium [32]. It is usually used for SVT that is refractory to other agents (adenosine, procainamide) [33] and, like procainamide, it can be used safely in patients with WPW. (See "Clinical use of amiodarone").

Amiodarone is administered intravenously, and a variety of regimens have been used. We typically recommend a bolus infusion of 5 mg/kg over 20 to 60 minutes. If there is no response, the bolus dose is repeated up to a total of 20 mg/kg. If the patient responds, this is followed by a continuous infusion of 10 to 15 mg/kg per day.

A number of small series have reported the efficacy of amiodarone in the acute treatment of SVT in children [33-36]. In a report of 40 children (mean age 5.4 years) who failed standard therapy, amiodarone successfully converted 16 of the 28 (57 percent) children with SVT to sinus rhythm [33]. An additional eight patients with junctional ectopic tachycardia had sufficient slowing of the rate to improve hemodynamic function.

In a report of 61 children (mean age 4.1 years), the response time was shorter and the overall success rate of conversion was greater in patients who received higher dosing regimens of amiodarone [36]. In this study, three loading dose regimens (low: 1 mg/kg, medium: 5 mg/kg, and high: 10 mg/kg) were used followed by three continuous infusion rates (low: 1, medium: 5, and high 10 mg/kg per day). Additional rescue bolus (up to five) could be administered 70 minutes after the loading dose. For the low, medium and high doses groups, the times to respond were 28.2, 2.6, and 2.1 hours, respectively; and successful conversions at 70 minutes were seen in 16, 25, and 41 percent of each respective group. At 48 hours, successful conversions were seen in 47, 80 and 73 percent of each respective group.

Side effects are relatively common. In one report of 31 episodes of tachyarrhythmias in children, adverse effects occurred in 18 (58 percent) but none required cessation of therapy [37].

In the first report of 40 patients discussed above [33], the most important side effects were mild hypotension in four and temporary pacing for bradycardia in one. No proarrhythmia was observed.
In the second report discussed above, 87 percent of patients reported side effects including hypotension, nausea, vomiting, atrioventricular block, and bradycardia [36]. There were too few of each type of adverse event to determine whether any of these were associated with amiodarone. Ten patients were withdrawn from the study because of significant adverse effects (hypotension, bradycardia, and atrioventricular block).

Many more patients treated with intravenous amiodarone were evaluated in a review of the published literature that was not limited to children or to patients with SVT [32]. The major acute side effect was hypotension. Proarrhythmia was noted in 2 to 3 percent; it is usually manifest as torsades de pointes but ventricular fibrillation can occur. Other cardiac side effects were less common. Intravenous therapy is not associated with the chronic side effects seen with prolonged oral therapy. (See "Major side effects of amiodarone").

Transesophageal pacing — Transesophageal pacing may be a useful adjunct to therapy for SVT in a child who is hemodynamically stable [10]. It can be used to determine the mechanism of and to terminate the tachycardia. The technique involves electrical cardiac stimulation until the tachyarrhythmia resolves or until other therapies can be initiated [10,38].

There are two major approaches to transesophageal pacing:

- Atrial overdrive pacing, in which atrial pacing commences at a rate slower than the tachyarrhythmia, with gradual acceleration
- Extrastimulation, in which the atrium is paced at a constant rate, with extra stimuli inserted at progressively shorter intervals.

In one report largely involving adults, AVRT and AVNRT were successfully terminated with transesophageal pacing in 62 of 63 patients [38]. However, transesophageal pacing requires specialized personnel and equipment. Sedation of the child may be necessary because the procedure can be quite uncomfortable. Thus, although useful, therapeutic pacing generally is considered only when the patient is hemodynamically stable [10].

Digoxin is not usually used because of the delay in achieving therapeutic levels and the narrow therapeutic margin with the risk of serious toxicity [10]. In addition, digoxin should not be given if WPW is suspected, since it may potentiate accessory pathway conduction.

Neonates — Neonates with SVT are often symptomatic with a high proportion of cases presenting with some signs or symptoms of cardiac failure. In a case series of 109 patients from a single tertiary center treated over a 27 year span (1971 to 1997), medical management was acutely successful in most of the patients with conversion to sinus rhythm in over 90 percent of patients after 24 hours of diagnosis and in 99 percent of patients after one week [39]. In eight patients, there was spontaneous conversion to sinus rhythm, and vagal stimulation was successful in 18 of 41 cases, in which it was attempted. There were seven deaths, five due to cardiac failure, and two not related to SVT. Patients who required multiple drugs or more than six days to obtain arrhythmia control were more likely to have recurrent episodes of SVT requiring chronic therapy.

CHRONIC THERAPY — After the acute episode is terminated, a 12 lead ECG should be performed to look for evidence of WPW syndrome (the presence of a wide QRS complex with a "delta wave") (show ECG 1). In addition, an echocardiogram should be obtained to assess for structural heart disease, since SVT can be associated with congenital disorders (21 percent of patients referred for catheter ablation in one series) [6].

The chronic management of SVT includes several options, each with advantages and disadvantages. The specific approach should be tailored to the individual patient based upon age and severity of symptoms (show algorithm 2).

Expectant management — One approach to the chronic management of children with recurrent episodes of SVT is expectant, with no specific treatment [10]. In this approach, the patient and family are taught to recognize SVT and to terminate episodes using vagal maneuvers. The rationale for this strategy is that patients who present at less than five years of age have a high likelihood of
outgrowing their SVT and may not require chronic therapy that may be associated with side effects. In addition, digoxin, which is the most commonly used drug, may not be more effective than expectant management [40-42].

If this is the first episode of SVT and there is no evidence of ventricular dysfunction and no hemodynamic instability, we offer the parents the option of expectant management and no specific treatment. We monitor the child for at least 24 hours and teach the parents how to check the heart rate. Vagal maneuvers may include using an ice bag applied to the face, performing the Valsalva maneuver, assuming a head-down position, inducing the gag reflex, and other techniques [10]. (See "Vagal maneuvers" above).

In infants who cannot complain of palpitations, recurrences of SVT may not be promptly detected [10]. While having the parents check the heart rate may be effective, we are more likely to consider the use of medications in this situation.

Medications — Medical therapy is used in children who have frequent episodes of SVT or who become symptomatic during infrequent episodes. The objective of medical treatment is to prevent episodes of SVT or to lessen symptoms during a recurrence. Another form of treatment is the use of "pulsed" or "cocktail" therapy, in which the patient takes an antiarrhythmic drug only during an episode of SVT in order to terminate the arrhythmia [10,43,44], although we do not generally use this approach.

Specific characteristics may help identify those patients who require long-term therapy. In one study, infants with SVT and structurally normal hearts were classified as simple (completely controlled by one medication) or complex (recurrent episodes needing at least one medication change for control) [45]. Complex (recurrent) SVT was associated with young age at presentation (10 versus 50 days), ventricular dysfunction at presentation, and slow retrograde conduction properties of the accessory pathway. Gender, initial medication, duration of treatment, cycle length in SVT or sinus rhythm, and presence of preexcitation were similar between the groups.

The benefits of medication therapy remain uncertain due to the lack of controlled studies. For infants, retrospective and anecdotal data suggest that recurrence rates with drug therapy range from 40 to 80 percent [10]. Conclusions about whether some drugs are more effective than others are difficult to reach from the available reports.

First-line agents — Our first-line agents for SVT are digoxin and a beta blocker, largely because they avoid the more serious side effects that can be seen with other antiarrhythmic drugs. In one report of 101 infants (under age 12 months) treated with digoxin, a beta blocker, or both, 70 percent were free of recurrence while on these agents [46]. As noted above, however, digoxin has not been proven to have greater efficacy than expectant management [40,41].

In infants, we give digoxin (10 mcg/kg per day divided into two doses) and propranolol (2 to 4 mg/kg per day divided into four doses). In older children we use a longer acting beta blocker, atenolol (1 to 2 mg/kg per day).

Digoxin should not be used in patients with WPW since it can accelerate conduction across the accessory pathway, particularly in patients who develop atrial fibrillation [10,47]. Side effects of beta blockers include hypotension, emotional disturbances and nightmares. (See "Major side effects of beta blockers").

As noted above, verapamil is contraindicated in children less than one year of age [29,30] and can cause accessory pathway acceleration and is not often used in WPW. (See "Verapamil" above).

Other drugs — The definitive therapy for SVT that does not respond to first-line therapy is radiofrequency ablation (RFA). However, because the risk of morbidity and mortality of this procedure is high in infants, pharmacologic therapy is usually attempted. Little information is available on the outcome of chronic medical treatment in older children, who are more often treated with RFA once their weight exceeds 15 kg [10].
Drugs that may be effective alone or in combination include flecainide, sotalol, and amiodarone [41,48-51]. The efficacy of these agents ranges from 20 to 100 percent. However, the results are difficult to interpret because of undocumented natural history, variations in practice, and lack of controlled trials. (See "Therapeutic use and major side effects of sotalol" and see "Clinical use of amiodarone").

The potential efficacy of these drugs is illustrated by the following observational studies:

Among 39 infants with SVT due to AV reentry, control was achieved in five of six treated with flecainide compared to 14 of 33 treated with digoxin [40]. In addition, flecainide controlled all 19 patients who did not respond to digoxin. The authors concluded that, compared to previous natural history studies, digoxin may have provided no benefit.

The efficacy of amiodarone was evaluated in 47 patients ranging in age from 23 weeks of gestation to 29 years; 24 had SVT. Treatment was judged effective in 32 (68 percent), but 11 of these required the addition of a second drug [51].

Among nine infants with tachyarrhythmias (including three with SVT) who had failed monotherapy with an average of four drugs (including flecainide and amiodarone), combined therapy with flecainide and amiodarone controlled the tachyarrhythmias in seven [47]. Similar findings were noted in another report in which the combination of flecainide and sotalol was effective in 10 infants with SVT who had failed treatment with at least two antiarrhythmic agents including either flecainide or sotalol alone [50]. No proarrhythmia occurred during treatment for five to 35 months.

The side effects of these drugs are more serious than those seen with digoxin and beta blockers. They include proarrhythmia, myocardial depression, and, with amiodarone, hypothyroidism and pulmonary fibrosis. (See "Major side effects of flecainide" and see "Therapeutic use and major side effects of sotalol" and see "Major side effects of amiodarone").

The use of combinations of drugs is variable from institution to institution as well as from patient to patient. Such an approach is reserved for patients for difficult to control arrhythmia and requires careful monitoring for proarrhythmic effects.

Radiofrequency ablation — Radiofrequency ablation (RFA), which was first reported as a successful alternative to drug therapy in children in 1991 [52], is the definitive therapy for SVT. In contrast, direct current ablation, which was employed prior to the development of RFA, was associated with a high incidence of complications and is no longer in clinical use [53,54].

Technique — RFA is performed as part of an invasive electrophysiology (EP) study. Sedation or general anesthesia is generally necessary to minimize discomfort and prevent patient movement during the procedure; general anesthesia may be used, especially in patients less than 10 to 12 years of age [10,55]. The study is performed in two stages:

First, transvenous catheter electrodes are inserted and manipulated for pacing and recording. The pattern of recordings obtained during normal sinus rhythm, in response to pacing, and during the SVT (if it can be induced) are used to make a definitive diagnosis of the mechanism of the SVT.

After the mechanism of SVT is determined, and the accessory pathway or AV nodal pathway is localized, a modified catheter is used that can deliver radiofrequency energy, which cauterizes the myocardial tissue and destroys the pathway. (See "Nonpharmacologic therapy of arrhythmias associated with the Wolff-Parkinson-White syndrome" and see "Catheter ablation for AV nodal reentrant tachycardia (junctional reciprocating tachycardia)").

Efficacy — The initial success rates for RF ablation in children can exceed 90 percent, although SVT can recur late after treatment. The following studies illustrate the range of published results:

The Pediatric Radiofrequency Catheter Ablation Registry prospectively evaluated the initial outcome of RF ablation in 481 infants and children with SVT (97 percent with structurally normal hearts) [56]. The procedure was immediately successful in 95 percent of cases with accessory pathways and 99 percent with AVNRT. At three years, the freedom from arrhythmia recurrence was 77 and 71 percent for accessory pathways and AVNRT, respectively [57].
In an analysis from a single center, 410 RFA procedures were performed in 346 children and young adults over a five-year period, almost all with SVT [6]. Repeat procedures were necessary for recurrent arrhythmia in 64 patients (18 percent). When repeat procedures were included, the final success rate of RFA was 90 percent (96 percent for AVRT and 83 percent for AVNRT). Over the five-year study period, initial procedural success rates increased from 80 to 100 percent, while recurrence rates declined from 20 to <5 percent.

Complications — Among the 4500 children evaluated in these two reports, there were a total of three deaths within two months of the procedure [6,57]. The total complication rates ranged from 1.2 to 3.2 percent. In the Registry data, the most common complications were AV block (25), cardiac perforation and/or pericardial effusion (24), brachial plexus injury (10), emboli (four CNS and four other), and pneumothorax (7) [57]. Risk factors for complications are patient age less than four years, patient weight <15 kg, and center inexperience with the procedure [57-59].

Indications — Indications for RFA are based upon an assessment of the benefits and risks and continue to evolve. Consensus guidelines on indications for RFA in children have been developed by the North American Society of Pacing and Electrophysiology (NASPE) [55]. There was consistent agreement and/or supportive data (class I indication) that RFA was beneficial in patients with SVT in the following situations:

WPW syndrome after an episode of aborted sudden cardiac death.
WPW at high risk for sudden death, which was defined as syncope with rapid conduction antegrade across the accessory pathway.
Recurrent or chronic SVT associated with ventricular dysfunction.

Other indications for RFA include patients older than 5 years of age with frequent symptomatic episodes of SVT who do not respond to antiarrhythmic therapy. In many cases, elective RFA is considered a preferred alternative to chronic antiarrhythmic therapy. However, the procedure should be avoided in patients younger than 5 years of age or 15 kg body weight because of their increased risk of complications. If the child is older than five years and weighs more than 15 kg, we offer the family the options of RFA or drug therapy (show algorithm 2).

The role of RFA in patients with asymptomatic WPW remains uncertain. (See "Asymptomatic WPW" below).

Cryoablation — Data are limited on the role of catheter-based cryoablation in children with SVT. An initial report showed that this modality can be safely performed in children as small as 20 kg and that it may be more effective for septal tachycardias due to AVNRT than those due to AVRT [60]. A comparison of cryoablation using a 4 mm cryocatheter and radiofrequency ablation in pediatric AVNRT found similar short-term efficacy (95 (cryo) versus 100 percent (RFA)) but higher recurrence rates in the cryoablation group (8 versus 2 percent) [61].

Surgical therapy — The success of RFA for the definitive treatment of SVT in children has led to a marked decline in the need for surgical intervention. Exceptions include the rare instances in which an arrhythmia is refractory to attempted RFA and those occasions in which another cardiac surgical procedure is planned as part of the patient's management (such as tricuspid valve repair for Ebstein's anomaly) [62-64].

ASYMPTOMATIC WPW — The best approach to management of asymptomatic children with the WPW syndrome remains uncertain. The risk of VF in asymptomatic adults is thought to be very low, ranging from almost zero to about 0.1 percent per year. However, the risk factors for sudden death in adults may not be entirely applicable to children with WPW. Of greatest concern are reports of patients in whom sudden cardiac death was the first presenting symptom of WPW. (See “Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia”, section on Ventricular fibrillation).

Noninvasive risk stratification may be useful in asymptomatic children with WPW. Ambulatory monitoring and exercise testing have been demonstrated to have predictive value; intermittent loss of preexcitation, including loss of preexcitation with exercise, is associated with a decreased risk of
sudden death. (See "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia", sections on Ambulatory monitoring and Exercise testing).

Electrophysiologic evaluation of accessory pathway conduction properties may be necessary to determine which children with WPW are at high risk. This concept was suggested in a report of 60 children undergoing surgery for WPW, 10 of whom had a history of cardiac arrest [65]. The conduction characteristics of the accessory pathway (the shortest preexcited RR interval <220 msec) appeared to be a more sensitive risk factor than clinical symptoms for sudden death, as it occurred in all of the children with cardiac arrest. (See "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia", section on Ventricular fibrillation).

RECOMMENDATIONS — The treatment strategy for SVT depends upon the patient's presentation and clinical status.

Acute treatment — An infant or child who presents in the nursery or to the emergency department in SVT should have a 15 lead ECG performed while a hemodynamic assessment is made. If the child is hemodynamically unstable, synchronized DC cardioversion with 0.5 J/kg should be performed. (See "Cardioversion" above).

If the child is hemodynamically stable, vagal maneuvers should be attempted to terminate the tachycardia (show table 1). For an infant, this consists of the application of an ice bag to the face for 15 to 30 seconds. In an older child, the vagal maneuver should consist of bearing down (Valsalva maneuver) for 15 to 20 seconds. A continuous ECG rhythm strip should be obtained during the maneuver.

If the vagal maneuver does not convert SVT to normal rhythm, an intravenous catheter should be placed. Adenosine should be administered in an initial bolus dose of 0.1 mg/kg, followed by a rapid saline flush. If the rhythm does not convert with the initial dose, adenosine is repeated with increases to a maximum dose of 0.25 to 0.35 mg/kg or a total dose of 12 mg.

Chronic therapy — After the acute episode is terminated, a 12 lead ECG and echocardiogram should be performed to look for evidence of WPW syndrome and structural heart disease. If this is the first episode of SVT and there is no evidence of ventricular dysfunction and no hemodynamic instability, we offer the parents the option of expectant management with no specific treatment. We monitor the child for at least 24 hours and teach the parents how to check the heart rate. The parents (and child, if able) are taught vagal maneuvers (such as ice bag or Valsalva) to assist in self-termination of SVT if it recurs.

If the child has frequent episodes of tachycardia or is symptomatic, we initiate drug therapy. Our first-line regimen in infants is digoxin (10 mcg/kg per day divided into two doses) plus propranolol (2 to 4 mg/kg per day divided into four doses). In older children we use atenolol (1 to 2 mg/kg per day), a longer acting beta blocker. We do not treat patients with WPW syndrome with digoxin.

If the child is older than five years and weighs more than 15 kg, we offer the family the option of RFA rather than drug therapy (show algorithm 2).

WPW without symptoms — In 2003, the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee task force did not recommend routine EP evaluation of asymptomatic patients with WPW [66]. However, these guidelines acknowledge that the role of EP testing in asymptomatic patients remains controversial and that the potential benefit of identifying high-risk patients who may benefit from catheter ablation must be balanced against the adverse events associated with the procedures. Given case reports of patients in whom sudden cardiac death was the first presenting symptom of WPW, a majority of pediatric electrophysiologists perform EP studies in asymptomatic children [67].

We evaluate children with WPW syndrome who are asymptomatic with noninvasive risk stratification via Holter monitor and exercise testing. Those with persistent preexcitation are considered to be at high risk. High risk patients over the age of five years are offered EP study and possible RFA. Patients
under the age of five years do not undergo routine EP testing, both because of the increased risk of complications and because preexcitation in infants often disappears at least transiently as they get older. (See "Complications" above and see "Supraventricular tachycardia in children: AV reentrant tachycardia (including WPW) and AV nodal reentrant tachycardia", section on Natural history).

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REFERENCES