Neonatal Supraventricular Tachycardia (SVT)

Harinder R. Singh, MD,* Swati Garekar, MD,* Michael L. Epstein, MD,† Thomas L’Ecuyer, MD‡

Case Report
A male neonate born at 31 weeks’ gestation had a history of fetal supraventricular tachycardia (SVT) detected at 28 weeks’ gestation, with no hydropic changes noticed on ultrasonography. The mother was started on enteral digoxin with instructions for weekly follow-up. On her first return visit at 31 weeks’ gestation, ultrasonography revealed hydropic changes and persistent SVT. An emergency cesarean section was performed, and the infant was found to have hydrops and respiratory distress, with a heart rate of 270 beats/min. The patient was intubated and mechanically ventilated and received a dose of intravenous (IV) adenosine, resulting in a transient decrease in heart rate to 120 beats/min before increasing back to 260 to 270 beats/min. SVT persisted despite institution of an esmolol drip at 500 mcg/kg per minute, and cardioversion was performed when hemodynamic instability developed. Recurrent SVT, associated with hypotension, developed within 45 minutes of successful cardioversion. The patient was given a loading dose of amiodarone, followed by cardioversion, continuous amiodarone, and dobutamine infusion. Echocardiography revealed normal anatomy with moderately reduced function. The patient remained in sinus rhythm and was weaned to oral amiodarone after 1 week of infusion. An echocardiogram revealed normal function. The patient was discharged from the hospital receiving 3 mg/kg per day of oral amiodarone.

Introduction
SVT is the most common symptomatic arrhythmia in childhood that can be a recurrent and persistent condition. The quoted incidence of SVT in children of 1 in 25,000 is based on an estimate made in 1967, (1) but with a higher index of suspicion and better methods of detection, it now is estimated to be 1 in 100 for children of all ages and 1 in 200 to 250 for neonates. (2) We report a case series of seven patients admitted to our neonatal nursery recently who had a diagnosis of SVT and review the pathophysiology and acute and chronic management of this condition in neonates.

Table 1 summarizes the clinical presentations and hospital courses of the seven neonates in this report. The heart rate during SVT ranged from 230 to 340 beats/min. Three neonates had Wolff-Parkinson White (WPW) syndrome, and one neonate presented with atrial flutter. Six patients had normal cardiac anatomy. SVT was diagnosed prenatally in three neonates, and they exhibited hydrops at birth and tachyarrhythmia-induced decreased cardiac function. Electrical cardioversion was attempted and successful in all three infants. Because of refractoriness to initial pharmacologic management and decreased cardiac function, amiodarone was used for chronic maintenance therapy.

Neonatal Tachycardia
Neonatal tachycardia is a common problem, either representing arrhythmia or sinus tachycardia. Sinus tachycardia can occur at a rate of 180 to 250 beats/min. The criteria that may help to identify sinus tachycardia are a normal P-wave axis (upright P waves in leads I and aVf), gradual onset and termination, variable rate, presence of a possible secondary
<table>
<thead>
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<th>Postnatal Age of Presentation/Gestational Age At Birth</th>
<th>Clinical Features</th>
<th>Prenatal Diagnosis/Management</th>
<th>Max Heart Rate (beats/min)</th>
<th>Mechanism of Tachycardia</th>
<th>Results of Echocardiography</th>
<th>Sequence of Treatment Termination</th>
<th>Maintenance (Enteral)</th>
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<tr>
<td>1. 2 d/31 wk</td>
<td>Hydrops</td>
<td>Fetal SVT (28 wk)/Digoxin</td>
<td>270</td>
<td>Pre-excitation/atrioventricular re-entry tachycardia (AVRT)</td>
<td>Normal anatomy/decreased function</td>
<td>Adenosine/Esmolol/Cardioversion</td>
<td>Amiodarone</td>
</tr>
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<td></td>
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<td></td>
<td></td>
<td>Adenosine (transient) Amiodarone/Cardioversion</td>
<td>Amiodarone</td>
<td></td>
</tr>
<tr>
<td>2. 1 d/34 wk</td>
<td>Hydrops</td>
<td>Fetal SVT (30 wk)/Digoxin</td>
<td>260</td>
<td>Pre-excitation/AVRT</td>
<td>Normal anatomy/decreased function</td>
<td>Adenosine/Procainamide/Digoxin/Propranolol/Esmolol/Cardioversion</td>
<td>Amiodarone</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Adenosine (transient) Cardioversion Amiodarone/Digoxin</td>
<td>Amiodarone</td>
<td></td>
</tr>
<tr>
<td>3. 1 d/term</td>
<td>Hydrops</td>
<td>Presumed fetal SVT (hydrops at birth)/-</td>
<td>340</td>
<td>Pre-excitation/AVRT</td>
<td>Normal anatomy/decreased function</td>
<td>Adenosine/Esmolol/Cardioversion</td>
<td>Cardioversion Amiodarone</td>
</tr>
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<td></td>
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<td></td>
<td>Adenosine (transient) Cardioversion Amiodarone</td>
<td>Amiodarone</td>
<td></td>
</tr>
<tr>
<td>4. 1 d/35 wk</td>
<td>SVT on cardiorespiratory (CR) monitoring</td>
<td>-/-</td>
<td>230</td>
<td>Atrial flutter</td>
<td>Normal anatomy/Normal function</td>
<td>Adenosine/Esmolol</td>
<td>Amiodarone</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>Adenosine (transient) Cardioversion Amiodarone</td>
<td>Amiodarone</td>
<td></td>
</tr>
<tr>
<td>5. 1 d/28 wk</td>
<td>SVT on CR monitoring</td>
<td>-/-</td>
<td>320</td>
<td>No pre-excitation/AVRT</td>
<td>Normal anatomy/Normal function</td>
<td>Ice pack/Digoxin (oral)/Propranolol (oral)</td>
<td>Adenosine Digoxin (oral)/Propranolol (oral)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ice pack</td>
<td>Amiodarone</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>6. 3 d/term</td>
<td>SVT on CR monitoring</td>
<td>-/-</td>
<td>280</td>
<td>No pre-excitation/AVRT</td>
<td>Normal anatomy/Normal function</td>
<td>Ice pack</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>7. 6 d/term</td>
<td>SVT</td>
<td>Congenital heart defect</td>
<td>250</td>
<td>No pre-excitation/AVRT</td>
<td>Normal function</td>
<td>Ice pack</td>
<td>Adenosine</td>
</tr>
</tbody>
</table>

Table 1. Overview of Neonates Who Had Supraventricular Tachycardia (SVT)
cause (eg, fever, sepsis, catecholamine excess), and no response to maneuvers used to convert SVT to normal sinus rhythm.

SVT is a tachyarrhythmia originating proximal to the bundle of His. The typical infant who has SVT has a regular R-R interval, with rates often greater than 230 beats/min and commonly 260 to 300 beats/min.

The normal cardiac impulse originates in the sinus node and travels antegrade through the atria to the atrioventricular (AV) node, where the impulse is delayed and is then propagated through the His bundle into the right and left bundle branches. Normal conduction uses the AV node as the only connection between atria and the ventricles. The most common type of SVT in infancy and childhood, representing approximately 70% of SVT, results from a re-entry circuit between the atria and the ventricles and is called atrioventricular re-entry tachycardia (AVRT) (Fig. 1) (Table 2). In this circuit, the AV node generally forms the antegrade pathway, and an accessory connection between the ventricle and the atria serves as the retrograde pathway for conduction of impulses. This is referred to as orthodromic AV reciprocating tachycardia. In some patients, the accessory connection can serve as the antegrade pathway, and the AV node or a second accessory pathway serves as the retrograde pathway. This is referred to as antidromic (antegrade) AV reciprocating tachycardia. WPW syndrome is SVT with evidence of pre-excitation. It is identified from a surface electrocardiogram (ECG) in sinus rhythm by a short P-R interval and a “delta wave.” The short P-R interval in WPW syndrome represents impulse conduction antegrade through the accessory pathway without AV nodal delay. The delta wave is caused by fusion of ventricular complexes resulting from a portion of ventricular myocardium initially depolarized by antegrade conduction through the accessory pathway along with depolarization of the rest of the myocardium through the normal pathway. WPW syndrome comprises 12% to 56% of AVRTs. (2)(3)(4)(5)(6)(7)(8)(9) Patients 1 and 2 from Table 1 showed evidence of WPW syndrome.

The second type of SVT is AV nodal re-entry tachycardia (AVNRT) (Fig. 2) (Table 2), which represents

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**Table 2. Electrocardiographic (ECG) Features of SVT**

<table>
<thead>
<tr>
<th>Type of SVT</th>
<th>Baseline ECG</th>
<th>QRS Complex During VT</th>
<th>P Wave During Tachycardia</th>
<th>P-R interval and R-P interval</th>
<th>Effect of Adenosine</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVRT</td>
<td>Delta wave may be present</td>
<td>Usually narrow</td>
<td>Inverted P wave may be seen following the QRS complex</td>
<td>P-R interval is longer than R-P interval</td>
<td>Termination</td>
</tr>
<tr>
<td>AVNRT</td>
<td>Normal</td>
<td>Narrow</td>
<td>Often not seen in typical AVNRT; seen after QRS complex in “fast-slow” (atypical) AVNRT</td>
<td>Fast-slow AVNRT: R-P interval is longer than P-R interval</td>
<td>Termination</td>
</tr>
<tr>
<td>AT</td>
<td>Normal</td>
<td>Narrow</td>
<td>Flutter or ectopic P waves may be seen</td>
<td>R-P interval is longer than P-R interval</td>
<td>Usually will not terminate; may “uncover” atrial rhythm</td>
</tr>
</tbody>
</table>

AVRT = atrioventricular re-entry tachycardia, AVNRT = atrioventricular nodal re-entry tachycardia, AT = atrial tachycardia
about 13% of SVTs. The tachycardia circuit also involves dual pathways, but both are situated within or near the AV node. Typical AVNRT conducts in an antegrade direction slowly down to the ventricles through one pathway and has a rapid retrograde conduction via the other pathway. It is, therefore, called “slow-fast” AVNRT. On the ECG, this manifests as a QRS complex followed by a T wave, with the P wave often not visible (concealed in the QRS complex). Atypical AVNRT has a comparatively slower retrograde conduction, so the P wave is visible as an inverted complex after the QRS. This is called “fast-slow” AVNRT. Other rarer forms are “slow-slow” AVNRT and “fast-fast” AVNRT.

The third most common type of SVT is atrial tachycardia (AT), comprising approximately 14% of SVTs (Table 2). (3) AT includes atrial flutter and atrial ectopic tachycardia (AET). Atrial flutter results from a re-entry circuit within the atrial muscle, with flutter wave rates as high as 400 to 500 beats/min. AET is due to ectopic atrial foci that display abnormal automaticity. These foci are reflected by an abnormal P-wave axis during tachycardia. The atrial rates range from mild tachycardia to 300 beats/min. The onset of AET is characterized by gradual acceleration of the atrial rate (ramping up) to maximum. AET does not employ re-entry. The AV node is not a part of the circuit in atrial flutter or atrial re-entry tachycardia. The ventricular rate is dependent on conduction of the atrial impulse through the AV node. Therefore, it can be as high as the atrial rate, with 1:1 conduction through the AV node or, more often, lower, with variable degrees of AV conduction.

Of particular importance in the management of SVT is the fact that both AVRT and AVNRT require conduction through the AV node as a part of the circuit to maintain tachycardia (Figs. 1 and 2). Natural history studies in patients who have SVT have demonstrated that approximately 70% of infants lose SVT inducibility by 1 year of age, and clinical recurrences are uncommon. (10)

**Clinical Features**

Presenting features of SVT in neonates can include fetal SVT, unexplained nonimmune hydrops in the newborn (patients 1, 2, and 3 from Table 1), sudden cardiovascular collapse, incidental detection during cardiorespiratory monitoring (patients 4, 5, and 6 from Table 1), or symptomatic heart failure developing over a few hours to days. A cardiac cause is found in about 9% of nonimmune hydrops, 60% of which is due to fetal SVT. (11) SVT can occur in patients who have underlying congenital cardiac defects, especially Ebstein anomaly and L-transposition of the great arteries. Congenital heart disease is reported in about 6.5% to 37% of neonates who have SVT. (2)(3)(12) Patient 7 in Table 1 was diagnosed with transposition of the great arteries and developed SVT awaiting cardiac surgery. SVT has been reported as the initial presenting feature of cardiac involvement in patients who have tuberous sclerosis with cardiac rhabdomyomas. (13)(14)(15)(16)

**ECG Features**

As noted previously, the typical neonate who has SVT has a regular R-R interval, with rates greater than 230 beats/min and usually 260 to 300 beats/min. P waves are discernible in only 60% of cases and usually differ morphologically from those present in sinus rhythm. They often are inverted and are seen just after the QRS complex. Wide QRS complex is seen in patients who have pre-existing or rate-dependent bundle branch block and in antegrade AVRT (Fig. 1). Nevertheless, additional consideration should be given to ventricular tachycardia in all patients who have wide-complex tachycardia (Fig. 4), and treatment should be directed accordingly. A delta wave (Fig. 5) and a short P-R interval seen on the baseline ECG in sinus rhythm in a neonate who has SVT makes the diagnosis of WPW syndrome.

A 12-lead ECG should be performed during tachycardia and after sinus rhythm is achieved, but this should not delay treatment. Once the patient is stable, an echocardiogram should be performed to evaluate ventricular function, identify the presence of congenital heart disease, and look for cardiac rhabdomyomas. The response
to adenosine can facilitate an accurate diagnosis of SVT as well as restore sinus rhythm in a significant percent of cases. As shown in Figure 6, adenosine can terminate SVT promptly if the AV node is a part of the circuit.

**Acute Management of Neonatal SVT**

SVT is a relatively common tachyarrhythmia in the neonatal intensive care unit. It may be recurrent or occasionally persistent, but rarely is it life-threatening. Acute termination of SVT is critical in patients who develop signs and symptoms of hemodynamic instability, including lethargy, pallor, poor perfusion, hypotension, acidosis, and signs of cardiac failure. Immediate restoration of sinus rhythm is achieved best by cardioversion (patients 1, 2, and 3 in Table 1). If IV access is available, adenosine may be tried while preparing for cardioversion.

Cardioversion refers to an electrical energy discharge that is synchronized with the large R (or S) wave of the QRS complex. Synchronization with the early part of the QRS complex avoids energy delivery in the early phase of repolarization when ventricular fibrillation can be induced. Transient delivery of electrical current causes a momentary depolarization of most cardiac cells. This allows the sinus node to resume normal pacemaker activity. The key components in preparing the patient are intravenous access, airway management equipment, sedative drugs, and a converter/defibrillator monitoring device. Lack of IV access or sedation should not delay cardioversion in the critically ill neonate. For neonates, a single paddle or patch is placed immediately to the right of the sternum, and the other paddle/patch is placed between the tip of the left scapula and the spine to place as much heart mass as possible between the two paddles/patches. If a paddle is used, conductive gel should be applied to ensure good contact because skin can conduct away a significant portion of the current. The patches are pre-gelled. The paddles should be applied firmly against the chest wall to avoid arcing and skin burns. An initial synchronized shock of 0.5 J/kg is recommended. In subsequent attempts, the energy is doubled. Even under ideal circumstances, only 10% to 30% of the total current reaches the heart. If conversion to sinus rhythm is not achieved after two attempts, drug therapy should be initiated before attempting cardioversion again. Although cardioversion can restore sinus rhythm, it cannot maintain sinus rhythm. Therefore, it is always prudent to have a pharmacologic agent available to maintain sinus rhythm after conversion.

Adenosine can be administered if a neonate is hemo-
dynamically stable and can be provided to the unstable neonate in whom IV access has been established while preparing for cardioversion. Adenosine is administered as a rapid IV bolus followed by a saline flush in doses of 0.05 to 0.2 mg/kg, with continuous ECG monitoring and availability of cardiopulmonary resuscitation equipment. Adenosine is successful in terminating SVT in about 42% to 86% of cases (Fig. 7). (4)(17)(18)(19) In our case series, adenosine successfully terminated SVT in four of the six neonates (66%) in whom it was used. It produces AV block (may appear as a brief episode of asystole), thereby disrupting one limb of the re-entry circuit. The advantages of adenosine are its very short half-life (10 to 15 sec) and the onset of efficacy (within 10 to 20 sec). Administration can be repeated immediately if required, and the drug has no negative inotropic effect. The disadvantage is immediate recurrence of tachycardia in approximately 30% of cases. Adenosine can cause flushing, bronchospasm, and transient new rhythms at the time of conversion, including premature ventricular contractions, premature atrial contractions, sinus bradycardia, tachycardia, as well as varying degrees of AV nodal block. (20)(21)

For patients who are hemodynamically stable, a more graded approach is appropriate. Initially, vagal maneuvers, which are simple, quick, and safe to perform, can be attempted. Placement of an ice pack on the face and nose for 5 to 10 seconds at a time is used commonly. This maneuver initiates a “diving reflex” that has been described in certain species of animals in which diving into cold water induces a reflex peripheral vasoconstriction accompanied by a profound vagal discharge. (22) The afferent pathway of the reflex is the trigeminal nerve endings around the nose and mouth, and the efferent pathway is the vagus nerve. In humans, bradycardia also is observed with these maneuvers. Success rates vary from 63% to 96%. (2)(5)(18)(23) In our case series, an ice pack was used in three neonates and was successful in terminating SVT in only one (33%).

Esmolol is a very useful pharmacologic agent in patients who experience recurrent or sustained SVT. This beta blocker has class II antiarrhythmic properties, an immediate onset of action, and a very short half-life of about 3 minutes. It is administered IV at a dose that can be titrated based on the response and can guide long-term management. (24)(25)(26) It acts by increasing the refractory period of the AV node, thus interrupting the re-entry circuit. It usually is administered as a loading dose of 100 to 500 mcg/kg over 1 minute fol-

Figure 6. Responses of narrow complex tachycardia to adenosine. IV=intravenous, AV=atrioventricular, VT=ventricular tachycardia, AT=atrial tachycardia, AVRT=atrioventricular re-entry tachycardia, AVNRT=atrioventricular node re-entry tachycardia, AET=atrial ectopic tachycardia. Adapted from ACC/AHA guidelines for the management of patients with supraventricular arrhythmias. Circulation. 2003;108:1871–1909. Copyright 2004 The American College of Cardiology Foundation and American Heart Association, Inc. Permission granted for one time use. Further reproduction is not permitted without permission of the ACC/AHA.

Figure 7. Termination of SVT with adenosine.
supraventricular tachycardia

The infusion should be stopped if the QRS
followed by a continuous infusion of 20 to 80 mcg/kg
every 5 to 10 minutes to a maximum of 15 mg/kg, and
procainamide. Procainamide can be administered IV in a
tachycardia. Therefore, it is recommended that the pa-
rate across the AV node, potentially causing ventricular
wave rate, but it may increase flutter wave conduction
atrial excitability, procainamide decreases the flutter
and depresses myocardial contractility. By decreasing
creases myocardial excitability and conduction velocity
has anticholinergic and local anesthetic effects. It de-
ate. Procainamide is a class IA antiarrhythmic agent that
rhythm, a class I antiarrhythmic agent may be appropri-
receptors and AV conduction and sinus node function.

Digoxin has been the drug of choice for neonatal SVT
in the past. Digoxin acts by decreasing conduction
through the AV node. This agent is avoided in patients
who have WPW syndrome because of their predisposi-
tion to develop atrial fibrillation, which may lead to
ventricular arrhythmias due to enhanced conduction
through the accessory pathway. We tend to use digoxin
in neonates who have SVT and poor cardiac function and
have not responded to beta blockade. Digoxin has an onset
of action within 5 to 30 minutes, with the maxi-
mum effects seen within 1 to 4 hours after parenteral
administration. After oral administration, the onset of
action is within 30 minutes to 2 hours, with maximum
effects seen after 2 to 8 hours. The elimination half-life is
61 to 170 hours in preterm neonates and 35 to 45 hours
in term neonates. The oral total digitalizing dose (TDD)
is 20 to 30 mcg/kg in preterm neonates and 25 to
35 mcg/kg in term neonates. The parenteral TDD is
75% of the oral TDD. The dose is reduced by 50% in
patients receiving concomitant amiodarone or patients
who have end-stage renal disease. Therapeutic serum
levels are 0.8 to 2 ng/mL. Toxicity may be seen with
concentrations higher than 2 ng/mL. Adverse effects
commonly seen with digoxin include nausea, vomiting,
heart block, and rhythm disturbances. Monitoring potas-
sium concentrations is crucial, especially in patients re-
ceiving concomitant diuretic therapy because hypokale-
ia can potentiate digoxin toxicity. (2)(28)(29)(30)

If beta blockade is ineffective in controlling the
rhythm, a class I antiarrhythmic agent may be appropri-
ate. Procainamide is a class IA antiarrhythmic agent that
has anticholinergic and local anesthetic effects. It de-
creases myocardial excitability and conduction velocity
and depresses myocardial contractility. By decreasing
atrial excitability, procainamide decreases the flutter
wave rate, but it may increase flutter wave conduction
rate across the AV node, potentially causing ventricular
tachycardia. Therefore, it is recommended that the pa-
tient be digitalized before receiving a loading dose of
procainamide. Procainamide can be administered IV in a
loading dose of 3 to 6 mg/kg over 5 minutes, repeated
every 5 to 10 minutes to a maximum of 15 mg/kg, and
followed by a continuous infusion of 20 to 80 mcg/kg
per minute. The infusion should be stopped if the QRS
complex widens to more than 125% of baseline. It is
important to monitor the patient for hypotension and
arrhythmias during the infusion. The enteral dose of
procainamide is 15 to 50 mg/kg per day divided every
3 to 6 hours. Procainamide is metabolized to the active
metabolite N-acetyl procainamide (NAPA). The half-life
of procainamide in infants is approximately 1.7 hours,
and the half-life of NAPA is approximately 6 hours.
Therapeutic concentrations of both procainamide and
NAPA are 4 to 10 mcg/mL. Other potential adverse
effects are lupus-like syndrome, nausea, vomiting,
diarrhea, and liver dysfunction. (31)

Flecainide is a class IC drug that exerts profound
effects on the accessory connection and the AV node,
which is unlike the action of the beta blockers adenosine
digoxin, which terminate SVT by blocking AV node
conduction only. Flecainide is administered at a dose of
2 mg/kg IV over 10 minutes during the acute phase
followed by a dose of 3 to 6 mg/kg per day orally in two
divided doses. (29)(32)(33)(34) The half-life in new-
borns is approximately 29 hours. Flecainide has a mod-
ate negative inotropic effect and can be proarrhythmic.

Propafenone is an alternate class IC agent that occa-
sionally is used in neonatal SVT, especially junctional
tachycardia. Its effects are similar to flecainide. An IV
dose of 1 to 2 mg/kg can be used for acute control of
SVT. The oral dose of propafenone is 8 to 10 mg/kg per
day divided in three to four equal doses, which may be
increased in increments of 2 mg/kg per day up to a
maximum of 20 mg/kg per day. Peak plasma concentra-
tions are reached after 2 to 3 hours, and steady-state
levels are achieved in 4 to 5 days. The elimination half-life
is approximately 5 to 8 hours. The adverse effects re-
ported with propafenone are negative inotropic effects,
proarrhythmic effects, sinus node dysfunction, blood
dyscrasias, and neurologic effects. (35)(36)(37)(38)

For patients who are unresponsive to the beta block-
ade or class I agents, a class III drug such as amiodarone
may be successful in terminating SVT. Amiodarone in-
hbits adrenergic stimulation, prolongs the action poten-
tial and refractory period in myocardial tissue, and de-
creases AV conduction and sinus node function. Amiodarone
is useful in patients who have SVT unre-
sponsive to beta blockers or class I agents or who have
depressed cardiac function. Amiodarone is started with
an IV loading dose of 5 mg/kg over 1 hour, followed by a
continuous infusion of 5 mcg/kg per minute, with dose
increases until either conversion of SVT or attainment of
a maximum of 15 mcg/kg per minute. The onset of
effect after parenteral administration is usually within
minutes, especially after a loading dose. Once SVT is
controlled with amiodarone infusion, the total daily dose can be converted into an oral dose that is weaned slowly to a maintenance dose of 3 to 5 mg/kg per day in two divided doses. The half-life of amiodarone is about 40 to 55 days. Amiodarone can cause new arrhythmias, hypothyroidism, liver dysfunction, pulmonary fibrosis, and corneal opacities. With the use of lower doses, such adverse effects are infrequent.

For cases of SVT that do not respond to single pharmacologic agents, a combination of different medications can be tried. Commonly used combinations include digoxin with propranolol (7) and digoxin with amiodarone. Various other combinations have been tried with high success rates. The reported success rate for the combination of amiodarone and propranolol is 80%, flecainide with amiodarone is 78%, and flecainide with sotalol is 100% in controlling single or multidrug refractory SVT. (52)(53)(54)

**Chronic Management of SVT**

Chronic medical treatment is appropriate for neonates who have hemodynamically significant SVT, frequent SVT requiring medical management, pre-excitation on ECG, or congenital cardiac defect. For neonates who have had self-terminating asymptomatic SVT with no evidence of pre-excitation on ECG and with normal cardiac anatomy, chronic medical treatment may be deferred.

A beta blocker such as propranolol is most appropriate for patients responding to esmolol or who have evidence of pre-excitation on ECG. Propranolol is a noncardioselective class II antiarrhythmic agent that has properties similar to esmolol. The starting dose of propranolol is 0.25 mg/kg every 6 to 8 hours and can be increased slowly to a maximum of 4 mg/kg per dose with close monitoring of heart rate, heart size, and contractility. Propranolol can cause bradycardia, hypotension, impaired myocardial contractility, hypoglycemia, and bronchospasm. It is contraindicated in patients who have poor cardiac function and those who have hyperactive airway disease. (27)

Patients who achieved control of SVT with class I or class III agents are treated with oral preparations of the same medications. Because 70% of neonates who have SVT lose inducibility by 1 year of age, most cardiologists treat for about 6 to 9 months, adjusting dosage for size, and subsequently allow the patient to outgrow the drug dose by about 1 year of age.

Figure 8 presents an algorithm for management of neonatal SVT that can be modified based on institutional preferences.

**Fetal SVT**

SVT is the most common symptomatic fetal cardiac arrhythmia. Atrophicventricular re-entry tachycardia is the most common mechanism, occurring in more than 90% of fetal SVTs. Other, rare mechanisms for SVT in fetuses are atrial flutter or fibrillation, automatic tachycardia, and permanent junctional reciprocating tachycardia. In fetuses that have SVT, the characteristic heart rate is 240 to 260 beats/min. In fetuses that have atrial flutter, the characteristic atrial rate is 300 to 500 beats/min, with varying ventricular response rates. (55)

Ultrasoundography currently is the most commonly used diagnostic tool to analyze heart rhythm in a fetus. A detailed study includes definition of cardiac structure, rhythm, function, hemodynamics, and presence of fetal hydrops. Structural malformations of the heart are seen in up to 5% of fetuses that have SVT, which most frequently include Ebstein anomaly of the tricuspid valve, AV canal defect, hypoplastic left heart syndrome, or rhabdomyoma. (56)(57)
Fetal SVT is associated with a fetal and neonatal mortality rate of 8% to 30%. Preterm delivery of an infant who has hydrops and SVT is associated with high rates of morbidity and mortality. Supraventricular tachycardia is often refractory to digoxin therapy. (55) The therapeutic goal is rate control (in atrial flutter) or complete control of the arrhythmia. Prior to initiating therapy, a complete maternal history for arrhythmia and a baseline maternal ECG to rule out long QT syndrome should be obtained. Daily monitoring of maternal ECG for rate and rhythm, P-R interval, and QRS and QTc duration as well as fetal monitoring is important for all medications. (55) Therapy with antiarrhythmics is initiated using low doses, with gradual dose increases and careful monitoring of maternal and fetal drug response in an inpatient setting. Fetuses that have hydrops are at a higher risk of adverse drug effects than fetuses that do not have hydrops. A fetus treated for SVT usually requires therapy for at least 2 to 3 weeks after conversion to sinus rhythm because of a predilection for recurrence.

Digoxin is the most frequently used monotherapy in mothers whose fetuses do not have hydrops but do have either SVT or atrial flutter. (55)(56)(60) It is used at a higher-than-usual dose to maintain therapeutic concentrations because of increased clearance and shorter elimination half-life during pregnancy. (61) A maternal loading dose of 0.25 to 0.5 mg is administered IV every 6 to 8 hours during the first 24 hours for rapid digitalization (maximum, 2 mg/d). The enteral loading dose of digoxin is 0.25 to 1.5 mg, followed by a maintenance dose of 0.25 mg to 0.5 mg/d. (62) The digoxin cord blood concentration varies widely, from 40% to 90% of the maternal level. Placental transfer of digoxin is as low as 10% in fetuses that exhibit hydrops. The dose should be titrated to achieve a maternal serum level of 1 to 2 ng/mL. The effectiveness of digoxin monotherapy is 32% to 71% in fetuses that do not have hydrops and 10% to 20% in fetuses that have hydrops. (56)(57)(62)(63)

Amiodarone and flecainide are emerging as the second-line antiarrhythmic agents for fetal SVT that does not respond to digoxin therapy. (63)(64)(65)(66) Amiodarone is the most effective treatment for drug-refractory fetal tachycardia accompanied by hydrops. A maternal oral loading dose of 800 to 2,400 mg/d is administered for 1 to 7 days, with a single dose not exceeding 800 mg. Once sinus rhythm is obtained, the dose is decreased to the lowest effective dose necessary to sustain sinus rhythm, usually 200 to 400 mg/d. Chronic maintenance therapy is continued until hydrops resolves and tachycardia is quiescent for 2 to 3 weeks. The half-life of amiodarone is 50 to 60 days. Maternal thyroid and liver profiles should be monitored before and during amiodarone therapy. Fetal plasma concentrations of amiodarone vary from 10% to 50% of the maternal level. During chronic treatment, the desired therapeutic serum concentration is 0.5 to 2.5 mcg/mL. Amiodarone alone or in conjunction with digoxin is successful in restoring sinus rhythm in approximately 60% to 75% of fetuses that have SVT and 33% to 50% of fetuses that have atrial flutter. (45)(63)(65)(66)(67)

Flecainide is a sodium channel blocker (class 1C) that has been used effectively in fetal SVT, both as monotherapy and in combination with digoxin. Flecainide should not be used as monotherapy in atrial flutter because it can increase the ventricular rate. Maternal dosing is initiated at 100 mg enterally twice a day. The dose can be increased to 300 mg/d in two to three divided doses. The half-life is 14 hours, and the time to reach 90% of a steady-state plasma concentration is approximately three times the half-life. The fetomaternal plasma ratio of flecainide varies from 50% to 80%. The therapeutic flecainide plasma concentration is 0.4 to 0.8 mcg/mL, and careful monitoring of values is required to decrease the incidence of proarrhythmic effects in the mother and fetus. Other maternal adverse events include dizziness, flushing, nausea, vomiting, and visual disturbances. Flecainide has negative inotropic effects and should be avoided when SVT is accompanied by structural heart disease or depressed myocardial contractility. Overall, the reported conversion rate to sinus rhythm has been 90% to 100% for fetuses that do not have hydrops with SVT and 60% to 85% for fetuses that do have hydrops. (62)(63)(64)(68)(69)

Sotalol has both beta-blocking and class III antiarrhythmic properties. Because of its potent antiarrhythmic effect and excellent placental transfer, sotalol is effective for fetal SVT, particularly in atrial flutter. Its half-life varies from 9 to 12 hours. Sotalol accumulates in amniotic fluid but not in the fetus. Maternal sotalol concentrations increase linearly with increasing dose. The fetomaternal plasma ratio of sotalol is nearly 1, indicating complete transplacental passage of sotalol. The oral maternal dose should be initiated at 80 mg twice daily and increased in incremental doses to a maximum of 160 mg three times daily. The therapeutic plasma concentration ranges from 1.27 to 1.63 mcg/L, but the maternal concentration does not correlate with clinical efficacy. Maternal ECG monitoring for prolongation of QTc interval...
is warranted during treatment with sotalol. Sotalol can cause temporary adverse effects in mothers, including nausea, dizziness, and fatigue, and can be proarrhythmic in fetuses. (70) Sotalol monotherapy is effective in converting 80% to 85% of fetuses that have atrial flutter and 60% to 70% of fetuses that have SVT to sinus rhythm. In fetuses that exhibit hydrops, the conversion rate is 40% to 60% as monotherapy and 80% in combination with digoxin. (63)(70)(71)(72)

Summary
SVT, which can be recurrent and persistent, is the most common symptomatic arrhythmia. The most common type of SVT in infancy results from a re-entry circuit between the atria and the ventricles. SVT inducibility is lost in 70% of infants by 1 year of age. Twelve-lead ECG during and after SVT along with the response to adenosine can facilitate accurate diagnosis. SVT can have varying clinical presentations, ranging from incidental detection to hemodynamic collapse. Hemodynamic instability warrants immediate restoration to sinus rhythm, best achieved by synchronized electrical cardioversion. Medical management of SVT consists of a trial of vagal maneuvers, adenosine, and medications to maintain sinus rhythm such as beta blockers and class I or class III antiarrhythmic medications. For neonates who have hemodynamically significant SVT, frequent SVT requiring medical management, pre-excitation on ECG, or congenital cardiac defect, chronic medical treatment is appropriate.

References


51. Etheridge SP, Craig JE, Compton SJ. Amiodarone is safe and highly effective treatment for supraventricular tachycardia in infants. Am Heart J. 2001;141:105–110


**NeoReviews Quiz**

6. Supraventricular tachycardia (SVT), a tachyarrhythmia that originates proximal to the bundle of His, can occur in infants who have underlying congenital heart defects. Of the following, the most common congenital heart disease that predisposes to SVT is:

A. Coarctation of the aorta.
B. Ebstein anomaly of the tricuspid valve.
C. Pulmonary atresia with intact ventricular septum.
D. Tetralogy of Fallot.
E. Total anomalous pulmonary venous return.

7. A term newborn has recurrent episodes of tachycardia that began at 12 hours of age. During one such episode, the heart rate is 280 beats/min. Electrocardiography reveals inverted P waves, most of which follow QRS complexes; normal QRS pattern; and P–R intervals that exceed R–P intervals. The infant is hemodynamically stable, has no echocardiographic evidence of congenital heart disease, and has no indwelling vascular catheters. Vagal maneuvers and intravenous adenosine are unsuccessful in restoring normal heart rhythm. Of the following, the next pharmacologic agent recommended, according to the algorithm for management of neonatal supraventricular tachycardia, is:

A. Amiodarone.
B. Digoxin.
C. Esmolol.
D. Procaainamide.
E. Propafenone.

8. In neonatal cases of supraventricular tachycardia nonresponsive to a single pharmacologic agent, a combination of medications may be tried. Of the following, the combination of medications most successful in controlling refractory supraventricular tachycardias is:

A. Amiodarone and propranolol.
B. Digoxin and amiodarone.
C. Digoxin and propranolol.
D. Flecainide and amiodarone.
E. Flecainide and sotalol.