PEDIATRIC

SUPRAVENTRICULAR

TACHYCARDIA

Pediatric Supraventricular Tachycardia

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Supraventricular tachycardia is the most common type of tachydysrhythmia in pediatrics, occurring in approximately 1 out of every 1,000 children. Therefore, it is important for the pediatric resident to have a good understanding of this disease. This review will provide a basic understanding of the pathophysiology, management strategies and clinical presentations of supraventricular tachycardia.

Normal conduction

To understand the different types of supraventricular tachycardia, you must understand the normal conduction system and its correlates on the surface electrocardiogram (PQRST). The "heartbeat" originates in the sinus node and propagates through the right and left atria producing a p-wave on the surface electrocardiogram. Because the atrium and ventricles are "insulated" from each other the impulse must travel across the AV node. This produces a delay of 0.1-0.2 seconds allowing the atrial contraction to fill the ventricles. This conduction delay that occurs at the AV node produces the PR interval on the ECG. The impulse then travels rapidly across the His-Purkinje system resulting in the simultaneous contraction of both ventricles producing the QRS complex. The ventricles then repolarize ("re-charge") producing the T-wave.

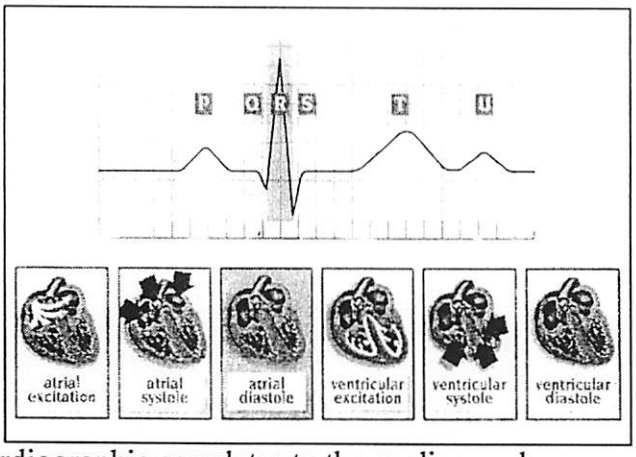


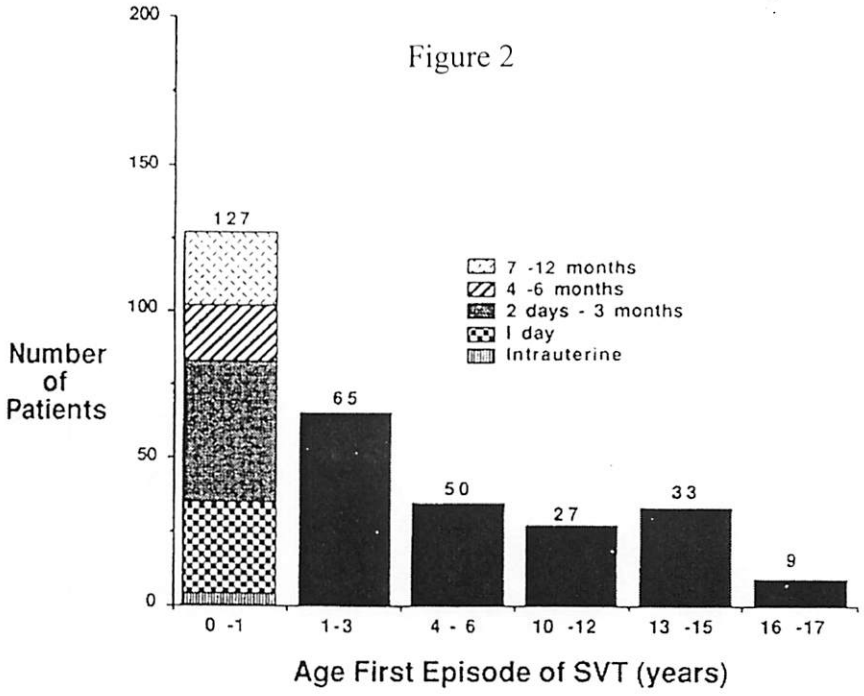
Figure 1: Electrocardiographic correlates to the cardiac cycle

Supraventricular tachycardia

Supraventricular tachycardia (SVT) is differentiated from ventricular tachycardia in that the atrium and/or the AV node are essential for it to be sustained. Most SVTs have a "narrow" QRS that is identical to the baseline QRS reflecting conduction over the normal His Purkinje system. In some cases there may be preferential conduction, or more frequently a delay, in one of the bundle branches that produces a "wide QRS", mimicking ventricular tachycardia. Because the conduction of the bundle branches is dependent on the baseline rate, many SVTs begin with a wide QRS that then narrows to normal (Ashman's phenomenon).

Pearl: All SVTs do not have a narrow QRS complex and all ventricular tachycardias do not have a wide QRS complex (especially in infants).

Figure 2



Patients with supraventricular tachycardia present in a bimodal distribution. SVT most commonly occurs between 0-3 months of age with the second most common age of diagnosis between 1-3 years of age. SVT in the infant may have several manifestations from by feeding intolerance to congestive heart failure while the older verbal child might complain of their heart "beeping" fast.

There are three types of SVT with approximately 16 different sub-types. For illustrative purposes we will discuss the two most common types: focal and reentrant.

Focal supraventricular tachycardia

Focal supraventricular tachycardia arises from an ectopic site or "focus" within the atrium or AV node. Like the sinus node, an ectopic focus can generate its own heartbeat that speeds up ("warms up") and slows down ("cools down"). In general, focal tachycardias are not as fast as reentrant tachycardias, only occasionally exceeding 200 bpm. Focal tachycardias are considered to be "chronic" tachycardias because they are slower and may go unrecognized resulting in congestive heart failure. The two most common sub-types are (1) atrial ectopic tachycardia and (2) junctional ectopic tachycardia.

Atrial ectopic tachycardia

Atrial ectopic tachycardia (AET or EAT) may arise anywhere in the atrium. Some of the more common sites include the pulmonary veins, atrial appendage, crista terminalis and prior suture sites. In older patients, AET may be seen with chronic pulmonary disease. Usually, AET occurs as a result of a single focus; however, some patients may have a multifocal tachycardia. In general, single site atrial ectopic tachycardia is best approached with transcatheter radiofrequency ablation that has a *cure rate* well over 90% in most centers. In the past it was felt that multifocal tachycardia could only be treated with medications; however, recent data suggests that multifocal tachycardia may represent a single focus with different exit points within the atrium. Thus, multifocal atrial tachycardia has also been successfully approached with ablation.

Electrocardiographically, patients with AET have a narrow QRS tachycardia with a p-wave preceding all QRS complexes. Usually the ectopic p-wave will be noticeably different on a 12- or 15-lead electrocardiogram. In some cases there will be 2-to-1 or greater AV block. In patients with multifocal tachycardia there are at least 3-5 different p-waves detected on an electrocardiogram.

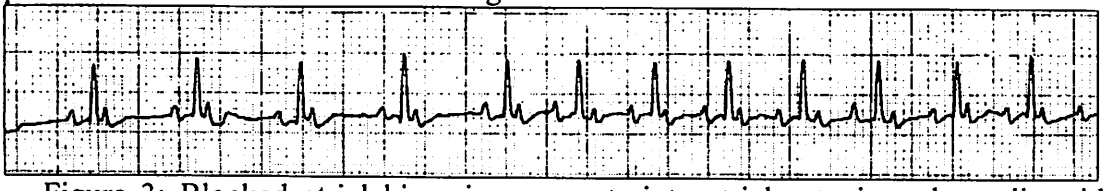


Figure 3: Blocked atrial bigeminy converts into atrial ectopic tachycardia with 2:1 conduction.

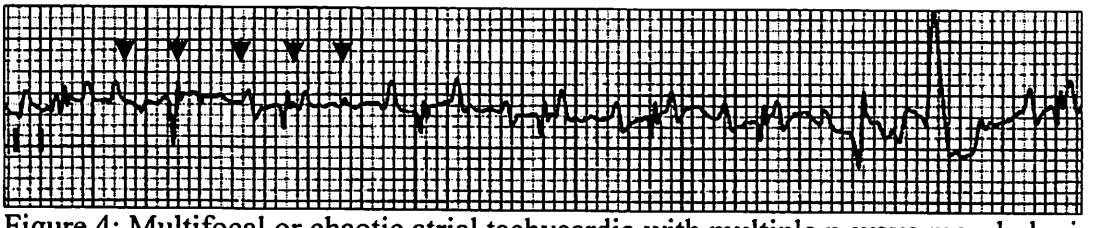


Figure 4: Multifocal or chaotic atrial tachycardia with multiple p-wave morphologies

Junctional ectopic tachycardia

Junctional ectopic tachycardia (JET) may be either congenital or postoperative. Congenital JET is a chronic tachycardia that may present with a tachycardia-induced cardiomyopathy in adolescents and teenagers. These patients may be approached with transcatheter radiofrequency ablation with an increased risk of AV node injury. Postoperative JET is commonly seen following congenital heart surgeries that require large muscle resection or VSD repair (e.g. with tetralogy of Fallot). Recent evidence suggests that post-op JET may be a result of hemorrhage or edema at or near the AV node. The ensuing inflammation results in junctional acceleration to rates as high as 200 bpm. Because the postoperative congenital heart is dependent upon the atrial contraction to fill the ventricle and maintain cardiac output, patients with post-op JET can develop hypotension and poor urine output prolonging the postoperative course. In these patients IV Amiodarone has been life saving.

Electrocardiographically, patients with JET have a narrow QRS tachycardia with no preceding p-wave. Because the impulse arises in the AV node, there may be retrograde conduction to the atrium producing a negative p-wave that follows the QRS complex. In other patients, there may be no retrograde AV node conduction resulting in V-A dissociation.

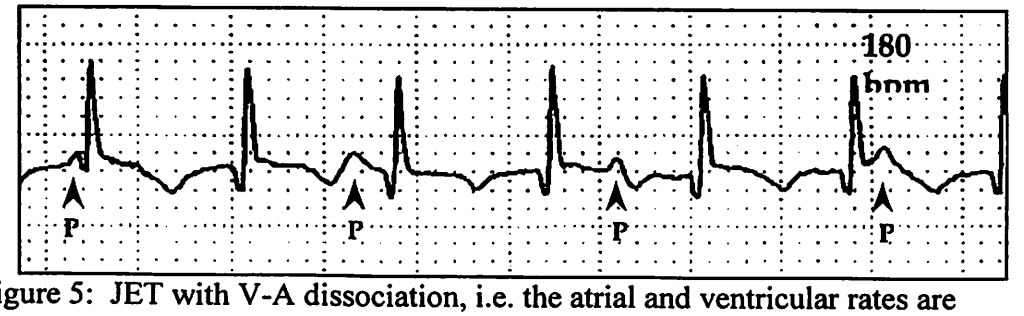


Figure 5: JET with V-A dissociation, i.e. the atrial and ventricular rates are independent from each other.

Reentrant tachycardias

Reentrant tachycardias involve reentry of an impulse around a circuit. Classically, the reentry circuit includes the atria, AV node, ventricles and an accessory pathway. Because the impulse travels from the atria to the ventricles and back to the atria again, it is said to be reciprocating. In ***orthodromic reciprocating tachycardia*** the impulse travels from the atria down the AV node to the ventricles and returns up an accessory pathway. In ***antidromic reciprocating tachycardia*** the impulse travels from the atria down an accessory pathway to the ventricles and up the AV node.

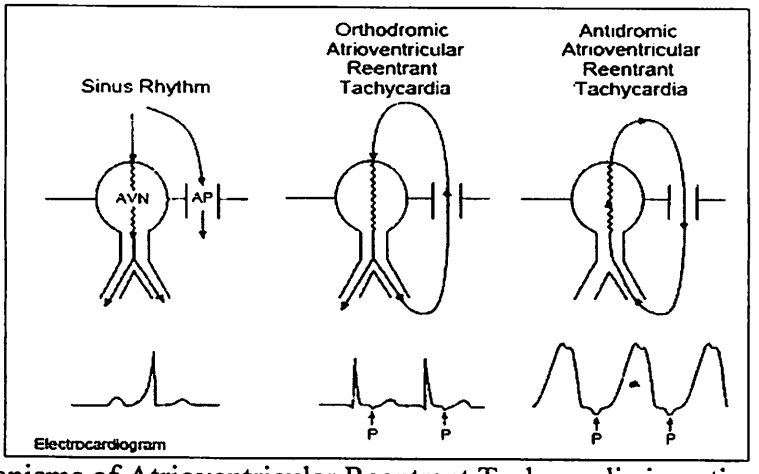


Figure 6: Mechanisms of Atrioventricular Reentrant Tachycardia in patients with WPW

Orthodromic reciprocating tachycardias

Wolff-Parkinson-White syndrome (WPW)

The most recognized type of accessory-pathway mediated tachycardia is that seen with Wolff-Parkinson-White syndrome (WPW). In WPW, the baseline electrocardiogram demonstrates a widened QRS indicating preexcitation of the ventricle where the accessory pathway inserts. WPW constitutes one of several types of preexcitation syndromes. The syndrome was first described in 1930 and consists of

a) short PR interval

b) slurred upstroke of the initial QRS complex (delta wave), giving a pseudo-bundle branch block pattern in sinus rhythm

c) occurrence of paroxysmal (sudden onset) tachycardias

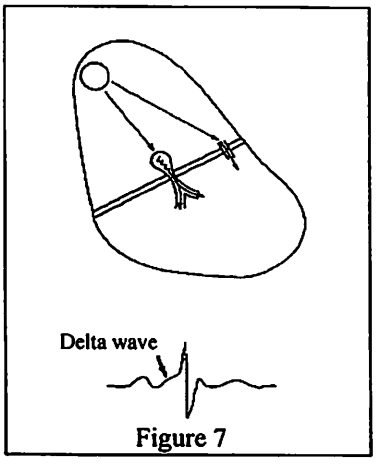


Figure 7

Permanent Junctional Reciprocating Tachycardia (PJRT)

PJRT is classically a slower narrow QRS tachycardia that occurs during childhood and is often refractory to medical therapy. It is caused by a slowly conducting accessory pathway that allows only retrograde (upward) conduction. Because there is no antegrade (downward) conduction as in WPW, there is no preexcitation on the surface ECG. The slow conduction of this accessory pathway allows for AV node recovery such that every heartbeat that returns up the accessory pathway can reenter down the AV node resulting in an incessant (or permanent) tachycardia.

The ECG features of PJRT are:
–Narrow QRS complex
–Retrograde 'P' wave in the inferior leads (II, II, aVF)
–Long R-P interval (slow conduction from the ventricle (R) to atrium (P))

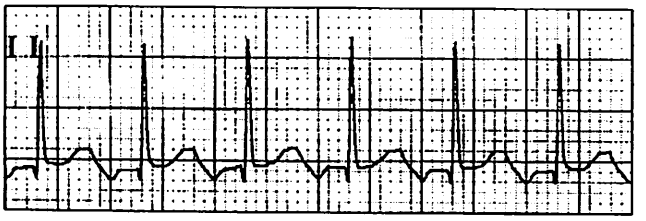
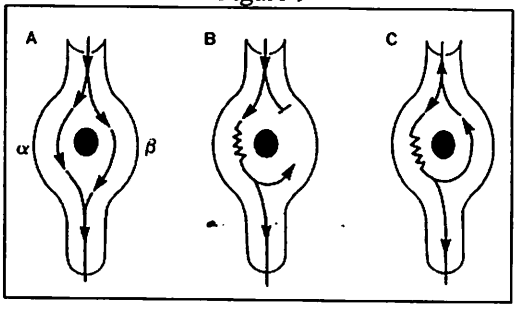


Figure 8: PJRT with large negative inferior P-waves

Atrioventricular node reentrant tachycardia (AVNRT)

Figure 9



In AVNRT the anatomic substrate or abnormality is the presence of dual AV node pathways (designated α and β or slow and fast, respectively) each with slightly different conduction properties (**A**). A premature atrial contraction blocks in the fast pathway and conducts down the slow pathway (**B**). The fast pathway recovers and permits retrograde conduction (reentry) often initiating tachycardia (**C**). During AVNRT, the atria are depolarized retrograde simultaneously with antegrade ventricular depolarization so that the retrograde P waves are buried in the QRS complex. Sometimes, they are just visible as part of the terminal QRS complex. Blocking AV node conduction by changing autonomic tone or using pharmacologic agents will terminate the tachycardia.

Antidromic Reciprocating Tachycardias

Mahaim (Atrio-fascicular) Tachycardia

The most common type of antidromic reciprocating tachycardia is that seen with a Mahaim or ***atrio-fascicular*** fiber. This is a special fiber that permits conduction only in the antegrade (downward) direction producing a wide QRS preexcited tachycardia. This type of tachycardia is very difficult to distinguish from ventricular tachycardia and in the presence of hypotension should be treated aggressively.

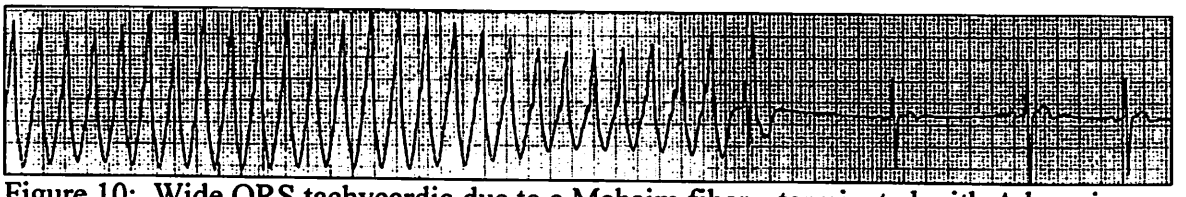


Figure 10: Wide QRS tachycardia due to a Mahaim fiber-- terminated with Adenosine.

Intra-atrial reentry tachycardias (IART)

Atrial flutter (AFL)

Intra-atrial reentry tachycardias comprise a group of tachycardias in which the reentrant circuit is confined to the atrium. In the case of atrial flutter, the circuit reenters around the tricuspid valve annulus in either a counterclockwise ("typical" atrial flutter) or clockwise ("atypical" atrial flutter) direction. These ***macroreentrant*** (large circuit) flutters give the classic saw-tooth pattern that is commonly seen. In patients who have undergone surgery for repair of a congenital heart disease there may be an atrial scar that permits reentry. This is usually associated with a lower amplitude p-wave and a slower rate of tachycardia.

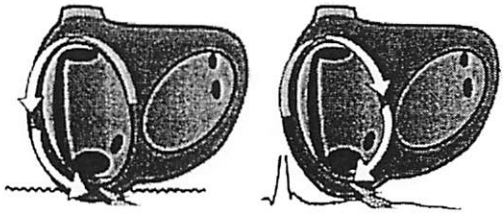


Figure 11: Typical and Atypical atrial flutter with counterclockwise and clockwise circuits, respectively.

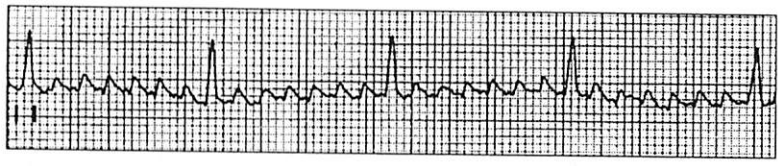


Figure 12: Typical atrial flutter with 7-to-1 AV block (one p-wave is buried in the QRS complex). The atrial rate is 500 bpm while the ventricular rate is 65 bpm (medication effect). Note the saw-tooth pattern consistent with a large circuit of reentry (macroreentry).

Atrial fibrillation (AF)

Atrial fibrillation is the most common arrhythmia in the world; however, it is relatively uncommon in children with structurally normal hearts. There is a higher incidence among patients with structural abnormal hearts, e.g. Ebstein's anomaly, hypertrophic cardiomyopathy or Eisenmenger complex, and postoperative congenital heart disease, e.g. Fontan procedure, due to chronic atrial stretching. ***Paroxysmal*** (sudden-onset) atrial fibrillation may occur in patients with structurally normal hearts. Recent evidence has shown that these patients may have focal atrial tachycardia arising in the pulmonary veins that induce them into AF. Radiofrequency ablation of the atrial tachycardia can "cure" the AF.

Patients with WPW are also at an increased risk of developing atrial fibrillation. Atrial fibrillation in the presence of a normal conduction system is not usually a life-threatening arrhythmia, because the ventricles (pumps of the heart) are protected by the AV node. However, in patients with WPW and atrial fibrillation, conduction may be rapid across the accessory pathway potentially producing ventricular fibrillation and sudden cardiac death. Therefore, atrial fibrillation + WPW must be suspected in any wide QRS rhythm with an irregularly irregular rhythm. **The use of digoxin and verapamil are**

contraindicated in WPW because they can promote conduction across the accessory pathway increasing the risk of sudden death if the patient should develop AF.

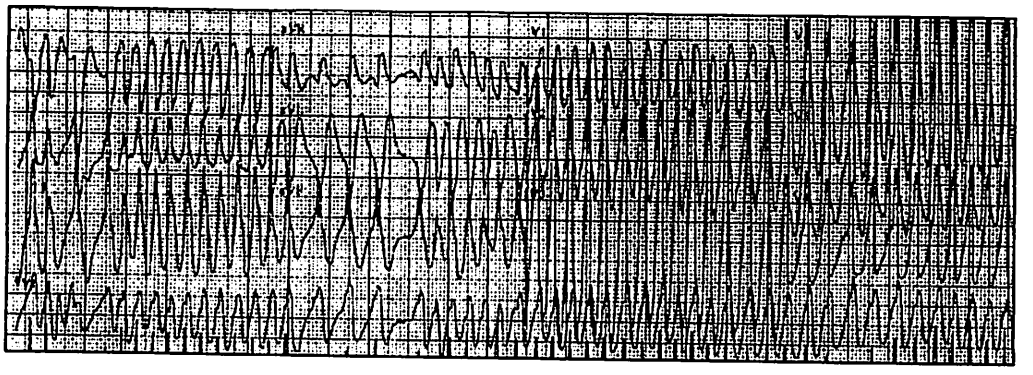
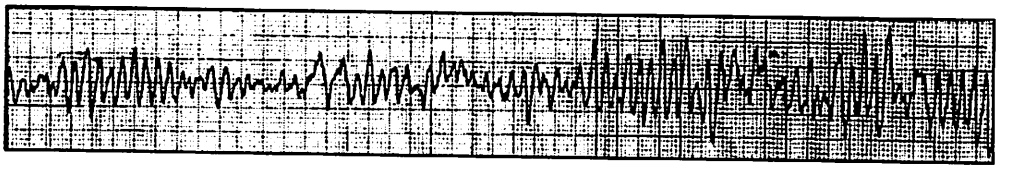


Figure 13 (above): Irregularly irregular wide QRS rhythm due to atrial fibrillation in the presence of WPW.

Figure 14 (below): Treatment of this arrhythmia with digoxin or verapamil could result in ventricular fibrillation and sudden cardiac death.



Acute Management of SVT

<u>Hemodynamically unstable (hypotension/poor perfusion/mental status changes)</u>

1. Synchronized DC cardioversion 0.5-2 Joules/kg
2. If IV present, Adenosine 0.1-0.3 mg/kg.
   Adenosine terminates SVT by producing AV block at the level of the AV node. Because Adenosine is metabolized by the red blood cells, it must be given by rapid IV push and its effect is usually transient (half-life 2-3 seconds). Adenosine is effective in most SVTs that use the AV node as an obligatory part of the circuit (AV reciprocating tachycardias and AV node reentry tachycardia). Because it also has effects on myocardium, it can cause termination of atrial ectopic tachycardias, as well as some ventricular tachycardias.
3. Other options
   a. IV Amiodarone 5 mg/kg over 15-30 minutes
   b. IV Procainamide 15 mg/kg over 15 minutes

Pearl: Adenosine and cardioversion produce acute termination of SVT. Because there has been no change in the substrate that caused the SVT originally, SVT may recur. This is an indication for longer-acting medications to maintain NSR.

Hemodynamically stable SVT

1. Adenosine
2. Digoxin
    a. Total digitalizing dose is age- and weight-dependent, but 10 mcg/kg IV is a safe first dose.
    b. Check electrolytes
    c. Contraindicated if suspect WPW/Afib
3. Amiodarone
    a. 5 mg/kg IV over 15-30 minutes
    b. Check electrolytes
    c. IV $CaCl_2$ for hypotension
4. Procainamide
    a. 15 mg/kg IV over 15 minutes
5. Initiate oral chronic therapy

Chronic management of SVT

1. Digoxin
2. Propranolol
    a. 3-4 mg/kg divided q 6-8 hours
3. Flecainide
    a. 50-200 mg/m^2/day divided bid
    b. Check electrolytes
    c. Trough level just prior to 6^{th} dose. Must be initiated during inpatient monitoring
    d. Absorption is hindered by milk products, so must adjust dose if taken off formula/milk
4. Sotalol
    a. 75-200 mg/m2/day
    b. Check electrolytes
    c. May prolong corrected QT interval; may produce torsades de pointes
    d. Must be initiated during inpatient monitoring
5. Amiodarone
    a. 10 mg/kg divided bid load for 2-4 weeks, maintenance 5 mg/kg q day
    b. Needs baseline LFTs, TFTs, CBC, PFTs (older pts)
6. Verapamil
    a. 3 mg/kg divided tid
7. Transcatheter radiofrequency ablation
    a. >95% cure rate
    b. Low complication risk
    c. Low recurrence risk
    d. Same-day procedure

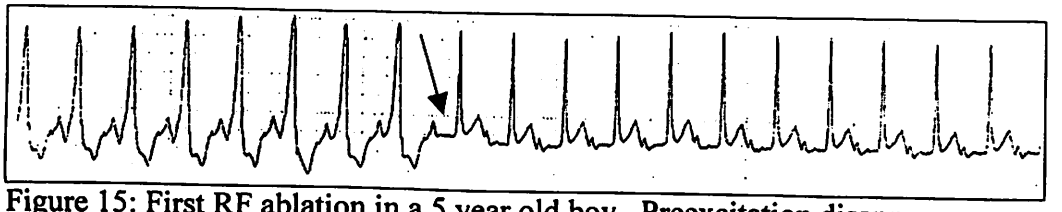


Figure 15: First RF ablation in a 5 year old boy. Preexcitation disappears.

Pearl: Pharmacologic or electrical cardioversion of SVT may produce a hemodynamically unstable dysrhythmia, e.g. ventricular fibrillation or severe bradycardia. Don't be caught unaware!! With electrical cardioversion, use electrode patches if possible so that you can concentrate on the post-cardioversion arrhythmia. With pharmacologic cardioversion, always have a defibrillator near-by--- just in case.

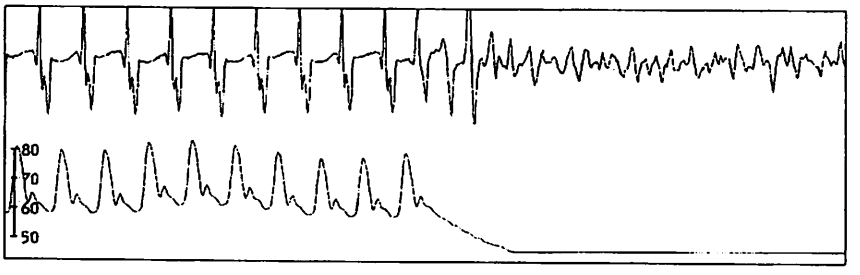


Figure 16: Ventricular fibrillation following Adenosine to terminate SVT (200 bpm) in an infant. Note the loss of blood pressure with the onset of VF.

References

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