Wolff Parkinson White Syndrome

A preexcitation syndrome resulting in a supraventricular tachycardia.

ANESTHETIC CONSIDERATIONS:

- Potential for acute SVT and hemodynamic compromise or collapse
    1. ACLS
    2. Defibrillation
    3. Global antiarrhythmics (procainamide, amiodarone may be useful)
- Need to avoid atrial fibrillation and/or atrial flutter
- Comorbidities – Ebstein's anomaly

ANESTHETIC GOALS:

1. Identify patients with WPW
2. Avoid any event (increased SNS activity) or drug (Adenosine, BB, CCB, Dig) that could enhance anterograde conduction of cardiac impulses through the accessory pathways
3. Reduce anxiety which may precipitate tachycardia

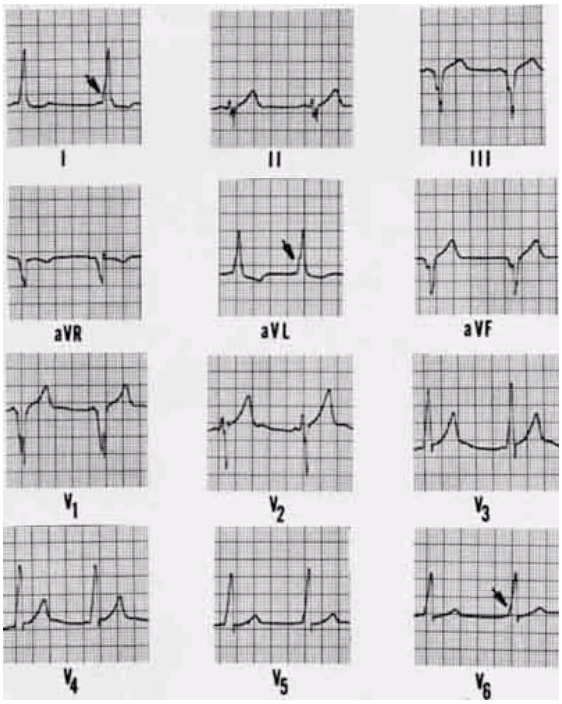
HISTORY (in addition to standard anesthetic history)

- Palpitations with or without associated dizziness, syncope, dyspnea, or angina
- Sudden cardiac death in close family member
- Previous diagnosis of and treatments for WPW
- Congenital heart disease or other cardiac co-morbid disease
- Cardiac dysrhythmias including AFib/Flutter
- Previous ablation procedures

PHYSICAL (in addition to standard anesthetic physical)

DICTATED PRIMARILY BY SURGICAL PROCEDURE AND CO-MORBID MEDICAL CONDITIONS

- **VITALS**
- **HEENT –** Examine airway for ease of intubation
- **RESP –** Auscultation for adventitial sounds
- **CVS** – Volume status, assess for tachydysrhythmia



INVESTIGATIONS

- **Labs**
    - Lytes, magnesium?
    - As dictated by the surgery: CBC, Lytes, BUN, Cr, Coags, Group and screen
- **Imaging**
    - As dictated by the surgery
- **Special**
    - EKG
        - Short PR interval – less than 120 msec during sinus rhythm
        - Widened QRS complex – duration > 120 msec with a slurred, slowly rising onset of the QRS in some leads (delta wave) and usually a normal terminal QRS portion
        - Secondary ST-T wave changes occur with T waves that usually are directed opposite to the major delta wave and QRS vectors

OPTIMIZATION

1. Continue any antidysrhythmia drugs the patient is on to maintain NSR
2. EP evaluation and ablation treatment if severely symptomatic
3. Ensure 5-lead EKG used
4. Consider cardioversion pads on patient
5. Have amiodarone and procainamide in room (if no AFib or Flutter) or Crash cart nearby

ANESTHETIC OPTIONS

6. Local
7. Regional
    - Need adequate sedation so as to reduce anxiety and SNS activity
8. General
    - Need adequate depth to prevent SNS activity

ANESTHETIC SETUP

9. **Equipment**
    - Standard equipment
    - Defibrillator / Crash cart
10. **Monitors**
    - Standard CAS monitors + 5 lead EKG
    - Adequate IV access (for adenosine if required)
    - Invasive monitors only if required by surgery or other co-morbidities
11. **Drugs**
    - Standard drugs including resuscitative drugs (phenylephrine and ephedrine)
    - Special drugs
        - Procainamide (20mg/min infusion until arrhythmia suppressed, hypotension ensues, QRS prolonged by 50%, or a total of 17mg/kg has been given

- Amiodarone may be useful

ANESTHETIC MANAGEMENT

- **Induction**
  - Consider anxiolysis pre-operatively
  - As dictated by surgery and pt's other co-morbidities
  - Avoid Ketamine and Pancuronium given sympathomimetic activity
- **Maintenance**
  - As dictated by surgery and pt's other co-morbidities
  - Consider having cardio-version pads on pt
  - Ensure adequate depth of anesthesia, so as to avoid SNS activation
  - Avoid abrupt increases in desflurane levels (sympathetic discharge)
- **Emergence**
  - As dictated by surgery and pt's other co-morbidities
  - Avoid sympathetic stimulation
- **Disposition**
  - As dictated by surgery and pt's other co-morbidities UNLESS pt has had episode of SVT in which case they should be in as monitored bed

POTENTIAL COMPLICATIONS

- Hemodynamic compromise or collapse from:
  - AV Re-entry Tachycardia (AVRT)
  - AFib/Flutter with uncontrolled conduction through accessory pathway

PATHOPHYSIOLOGY

- Reported prevalence averages about 1.5 per 1,000 (0.15%)
  - Higher in relatives of patients who have Wolff–Parkinson–White syndrome
- Acquisition of an accessory pathway is probably congenital
  - Left-sided pathways are more common than right-sided pathways
- Found in all age groups
  - Higher prevalence in men and a decrease in prevalence with age
- Most adults with pre-excitation syndrome have normal hearts, although a variety of acquired and congenital cardiac defects have been reported to be associated
  - Most commonly Ebstein's anomaly
- Prognosis is excellent for those without tachycardia or an associated cardiac abnormality
- In patients with tachycardia sudden death does occur rarely
- Treatment:
  - In the patient without tachycardia, no therapy is indicated
  - For the patient with recurrent episodes of tachycardia
    - Pharmacologic – Adenosine, global antiarrhythmic
    - Electrical – DC cardioversion
    - Surgical therapy – ablation of accessory pathway
- ACLS and WPW:
  - WPW only a worry when the accessory pathway conducts down (Atrium → Ventricle) AND when the patient has AFIB or AFlutter
  - In this situation, there is a risk that the rapid atrial rate can be conducted into the ventricle and result in hemodynamic collapse from VFIB/Flutter
    - AV node maximal conduction rate = ~ 220 – Pt's age
    - Accessory pathway maximal conduction rate = NO MAXIMUM
  - In this situation, you should avoid AV nodal blocking drugs [A(adenosine)B(beta blocker)C(calcium channel blocker)D(digoxin)] as this may result in the impulses being transmitted down the accessory pathway
  - Safe to use global antiarrhythmics or cardioversion:
    - Amiodarone
    - Procainamide (may get transient increase in conduction through AV node before blocking down)

Background

Epidemiology

-accessory AV nodal pathways estimated to be present in 0.1-0.3% (Stoelting)

Pathophysiology

-congenital
-extra nodal accessory pathway between atria and ventricles (called Bundle of Kent)
-WPW = pre-excitation and tachyarrhythmias
-AVRT (Atrioventricular re-entry tachycardia) accounts for 95% of arrhythmias in WPW
-orthodromic AVRT- re-entrant impulse travels from atria to ventricles over AV node and from V to A over accessory pathway
-antidromic AVRT (10%)- A to V over accessory, V to A over AV node
-low prevalence of coexisting structural heart disease in WPW patients

Atrial fibrillation

-is potentially life-threatening arrhythmia in WPW patients
-10-30% of patients with WPW have AF (UTD)
-if accessory pathway has short anterograde refractory period, then rapid repetitive conduction to ventricles during AF can result in rapid ventricular response with subsequent degeneration to VF
-rapid AVRT may contribute to initiation of AF

Sudden Cardiac Death

-annual incidence of sudden cardiac death 0.15-0.4%

Risk factors for cardiac arrest:

- -shortest pre-excited R-R interval < 250ms during AF
- -history of symptomatic tachycardia
- -multiple accessory pathways
- -Ebstein's anomaly

-Lower risk:

- -intermittent pre-excitation
- -loss of pre-excitation with procainamide
- -inducibility of AVRT or AF during EP study

Antiarrhythmics and AV node

-alter conduction through AV node

- -verapamil, beta blockers, digoxin, adenosine

-alter conduction through atrium, ventricle, or accessory pathway

- Class IA: quinidine, procainamide, disopyramide
- Class IC: flecainide, propafenone
- Class III: ibutilide, sotalol, amiodarone

-lidocaine- has been associated with deterioration of WPW into VF and sudden cardiac death

Acute Treatment for Orthodromic (narrow QRS)

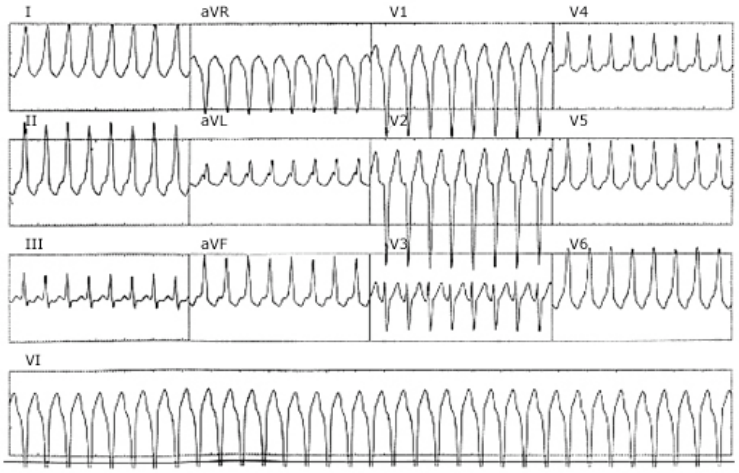
-AV node is site of antegrade conduction- therapies that lengthen AV nodal refractoriness and depress its conduction can terminate and prevent the tachycardia

-If unstable: synchronized cardioversion

-If stable:

- 1st line: vagal maneouvers, verapamil (5mg IV q3min up to 15mg), adenosine (6-12 mg IV bolus with flush)
- 2nd line: procainamide, BB, digoxin, amiodarone

(prolongs the refractoriness of all cardiac tissue)



AVRT

Acute treatment for Antidromic (pre-excited, wide QRS) tachycardia

-accessory pathway is site of antegrade conduction

-ensure not VT

-Unstable- synchronized cardioversion

-Stable:

- -Procainamide- drug of choice
  - -depresses conduction and prolongs refractoriness in atrial and ventricular myocardium, accessory pathways, and the His-Purkinje system, while having no effect or causing slight shortening of AV nodal refractory period
- -avoid- adenosine, BB, CCB, digoxin

Acute treatment for pre-excited Atrial Fibrillation

-Unstable- DC cardioversion

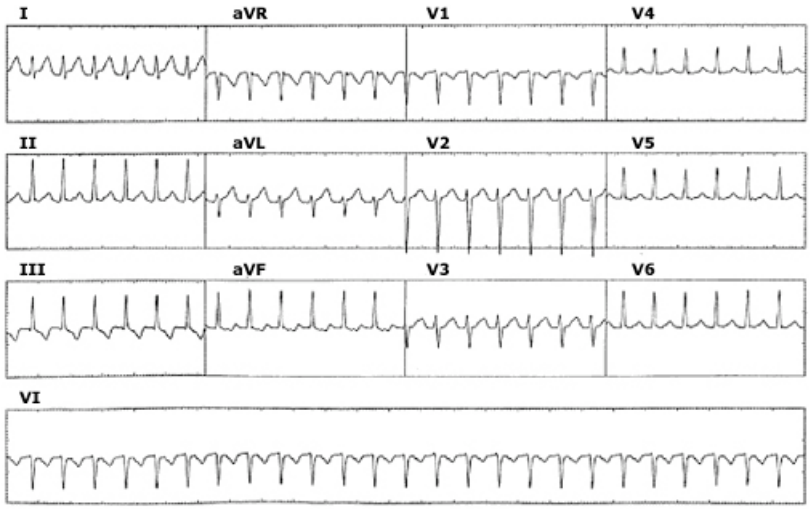
-IV procainamide 20mg/min IV infusion until arrhythmia suppressed, hypotension ensues, QRS prolonged by 50% of original duration or total of 17mg/kg has been given

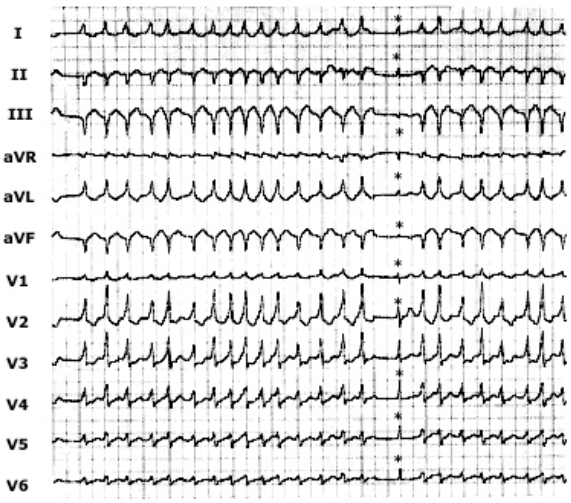
- -drug of choice

-amiodarone may be useful

-AV nodal blocking drugs contraindicated- BB, digoxin, verapamil, adenosine

- -can precipitate ventricular fibrillation





Chronic treatment for pre-excitation

-if asymptomatic and low risk for sudden cardiac death- no treatment

Radiofrequency ablation

-therapy of choice for those with pre-excitation + symptoms,
-95% effective
-Risks: 0-0.2% mortality, 2% risk of major complication

Pharmacotherapy- prophylactic or prn

-for patients that are not candidates for RFA
-may need guidance from EP study to choose correct agent based on properties of reentrant circuit- ie. Sometimes verapamil, BB are appropriate therapy
-alter conduction through AV node
    -verapamil, beta blockers, digoxin
-alter conduction through atrium, ventricle, or accessory pathway
    Class IA: quinidine, procainamide, disopyramide
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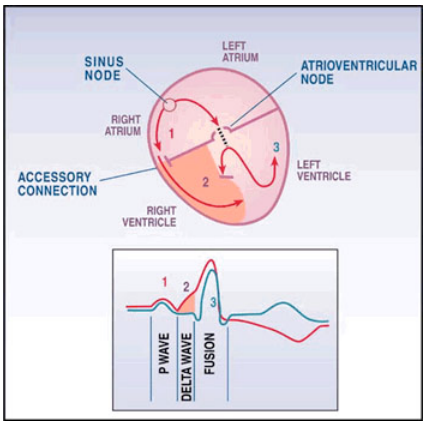

RESOURCES:

- Stoelting, Anesthesia and Co-existing Disease, 4th Ed., pgs. 68 – 89
- Humes, Kelley's Textbook of Internal Medicine, 4th Ed., Chapter 76
- ACLS: Principles and Practice