AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
1999

Cardiac Dysrhythmias I: Recognition and Treatment of Tachycardia

This course presents a comprehensive overview of the pathophysiology, diagnosis, and management of the patient with dysrhythmia. Specifically, this part of the two-part series will focus on the systematic evaluation and management of tachycardias. The practical application of anatomy and physiology will be related to a broad range of electrophysiologic problems, including pre-excitation syndromes, atrial tachyarrhythmias, and ventricular dysrhythmias. Antidysrhythmic medications will also be discussed in depth.

- Systematically identify and treat the patient with a narrow or wide complex, regular or irregular tachycardia.
- Differentiate supraventricular tachycardia pathoanatomy from the 12-lead ECG, and distinguish ventricular tachycardia from wide complex supraventricular tachycardia.
- Explain the mechanism and interactions of antidysrhythmic medications.
- Discuss the controversies in the evaluation and management of tachycardia.

TU-73
Tuesday, October 12, 1999
9:00 AM - 10:55 AM
Room # N227
Las Vegas Convention Center

FACULTY

Steven A Pace, MD, FACEP

Clinical Assistant Professor, Department of Medicine (EM), University of Washington, Seattle, Washinton; Attending Physician, Department of Emergency Medicine, Madigan Army Medical Center, Fort Lewis, Washington; Associate Medical Director, Washington Poison Control Center; Staff, Tacoma, Emergency Care Physicians, Tacoma, Washington

Cardiac Dysrhythmias I:
Recognition and Treatment of Tachycardia

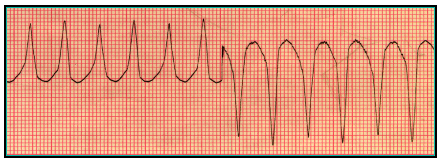
Objectives

- Evaluate and treat the patient with a narrow or wide complex tachycardia.
- Differentiate SVT pathoanatomy and distinguish VT from SVT with 12 lead ECG.
- Explain mechanisms and interactions of antidysrhythmic medications.
- Discuss controversies in tachycardia Rx.

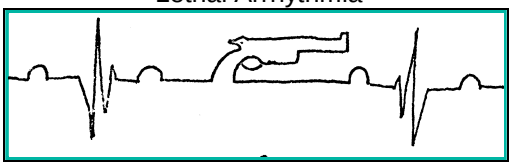
Outline

- General approach
- Clinical electrophysiology
- Supraventricular tachycardias
- Wide complex tachycardias

Case: Ann E - Cardiac Arrest



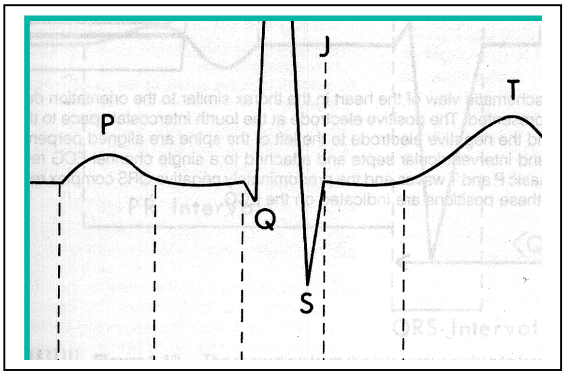
Lethal Arrhythmia



Intervals and Basic Physiology

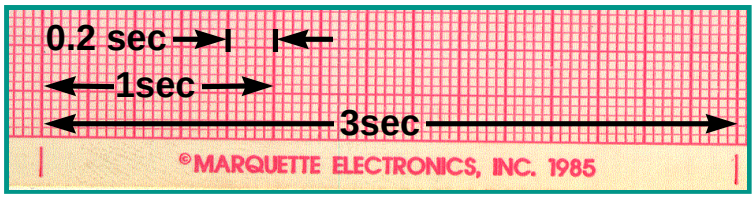
The ECG Tracing

- atrial depolarization - P
- rhythm and AV association - PR
- ventricular depolarization - QRS
- repolarization - ST, T, QT, U



The ECG Tracing

- One large box equals 0.2 second
- Five large boxes equal 1 second
- Rate = 60 divided by RR interval in sec
- Each hash mark represents 3 seconds



TACHYARRHYTHMIAS

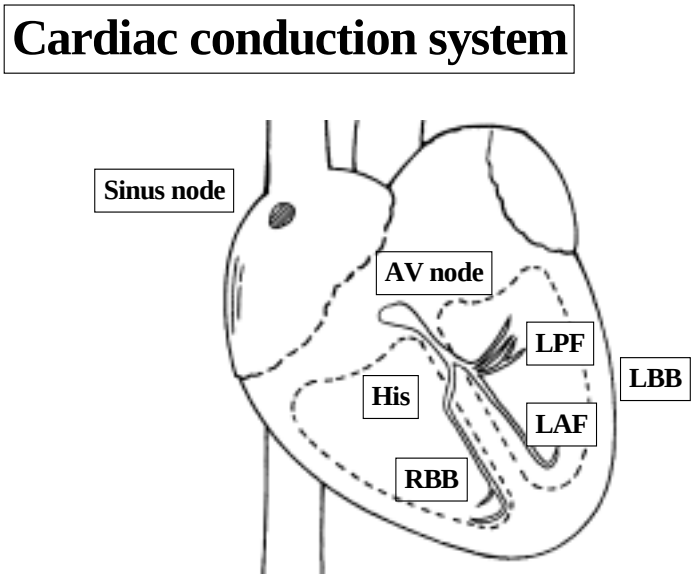
Introduction

Few things in emergency medicine are as acutely stressful to the emergency physician as the patient with a potentially malignant arrhythmia. This is our opportunity for a disaster, or a life saved. When dealing with patients with arrhythmias it is important to have a consistent, concise and simple approach. This approach should be one that can used in the heat of battle because, in general, your IQ drops at least 100 points under high stress conditions. Exact rhythm diagnosis is much less important than giving the correct therapy for a particular group of arrhythmias (or not giving the wrong therapy). By following some simple rules you can manage 99% of arrhythmias effectively.

Mechanisms for arrhythmia generation are interesting, but an expert knowledge is not required for good emergency department care. The section on mechanism of arrhythmias will therefore be brief and to the point. References are available for further reading. On the same note, memorizing the Vaugh-Williams classification may be fun for some people, but is it what you really need to know? I don't think so. As clinicians we do not look at a patient with an arrhythmia and think "I wonder if I will use a sodium channel blocker with or without propensity to increase the duration of phase 4." We need to know what drug to use when, its side effects, its contraindications, and what to do when things go bad or if the therapy doesn't work.

Anatomy and Physiology

Under normal circumstances the sinus node is the pacemaker for the heart. In the sinus node, the specialized cells spontaneously depolarize, reach threshold and discharge. Normal sinus rates are between 50 and 100 in most adults. The sinus node is not the only area of the heart that can generate a rhythm. Indeed, most areas of the heart can be pacemakers under the right conditions. If the sinus node fails to produce a rhythm then the HIS bundle, Purkinje system and ventricles may become the pacemakers. These alternate pacemakers are slower and less stable than the sinus node.



After an impulse starts in the sinus node, it travels down the atrium to the AV node. This atrial depolarization corresponds to the P wave on the EKG. The AV node is the electrical connection between the atrium and the ventricles. The AV node slows conduction and, in fact is protective against very rapid atrial rates producing very rapid ventricular rates. The normal slowing in the AV node (and HIS bundle) shows up on the ECG as the PR interval. After the impulse travels to the HIS bundle, it then breaks into the left and right bundle branches. This system is a specialized form of heart muscle that conducts very rapidly. Normal impulses that travel down this pathway thus produce a narrow QRS complex (the QRS complex reflects ventricular depolarization). Disease of the AV node causes heart blocks, disease of the Purkinje system produces bundle branch blocks or an intraventricular conduction delay.

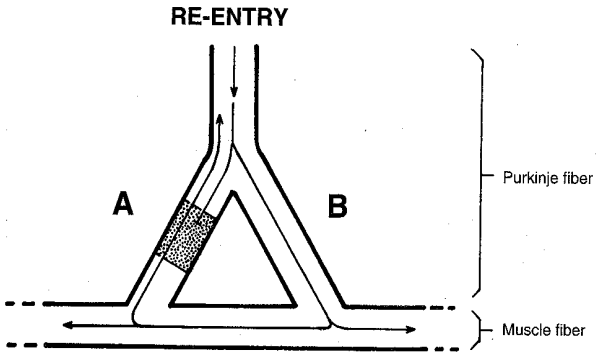
There are two major mechanisms of tachyarrhythmia generation:

Overview

1. *Disturbances of automaticity* - this is an increase or decrease in the rate of the spontaneous depolarization of cells, above their threshold. Enhanced automaticity can occur as a result of increased sympathetic tone (for example

you are getting chased by a tiger, have a fever, thyrotoxicosis, MI, have overdosed on digoxin, or are doing cocaine). When enhanced automaticity affects the sinus node, you get sinus tachycardia. When the pacemaker is from another site, other arrhythmias may develop. The bottom line on treatment is to reduce the stress that is causing the increased automaticity. For example, treat the fever, give Digibind or shoot the tiger. If this doesn't work, then the arrhythmia itself needs treatment. A number of modalities are available: a) drugs to reduce automaticity, b) overdrive pacing (to allow another part of the heart to take over as pacemaker function).

2. *Reentry* - this is a circus movement phenomenon in which an impulse runs in a circular movement and generates a tachyarrhythmia. The requirements of a reentry circuit are:
   - an available circuit.
   - unequal responsiveness of the two segments of the circuit.
   - slow conduction in one limb.
   - reexcitation of the initially blocked pathway.



Impulses travel down one limb, then back up the other and this cycle continues over again. The initial cause is a difference in conduction, combined with perhaps a premature beat, that results in one limb being refractory to the anterograde impulse but not to the retrograde impulse that arrives a little later on. Reentry can be at a microscopic level (i.e. in a small diseased part of the heart, say, after an MI) or on a macroscopic level (e.g. from atrium to ventricles over an accessory pathway).

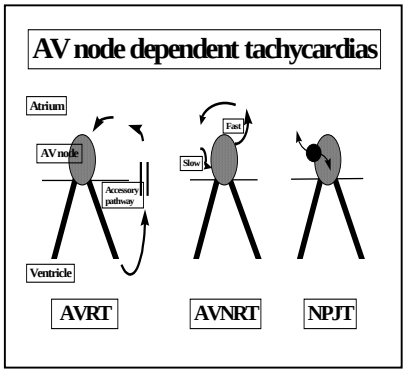
Glossary of Terms

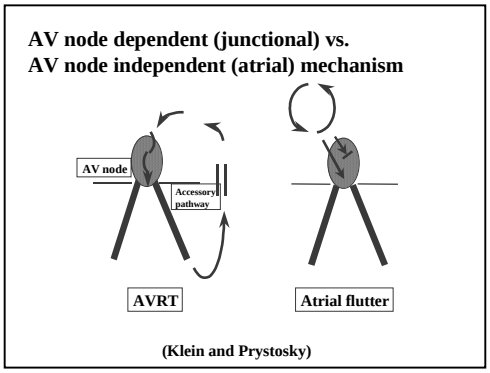
The language in this area of medicine can be confusing. What follows are some select definitions and explanations of terms that are frequently confused.

- *Aberrancy* = Aberrancy is a fancy term that means bundle branch block. It is most frequently used in conjunction with SVT which if there is aberrancy (bundle branch block) can look like ventricular tachycardia.
- *Arrhythmia vs. Dysrhythmia* = Great debates have occurred over which is the correct term to use. Both terms suggest an abnormality of rhythm, both terms are acceptable. Dissenters of the term arrhythmia suggest it means a lack of rhythm. Dr. Wellens notes that a lack of rhythm is not the meaning of the term in the Greek, arrhythmia can simply mean a disturbance of rhythm.
- *Supraventricular tachycardia (SVT)* = In the pure form a supraventricular rhythm simply means any rhythm that is generated above the level of the ventricles. This could be sinus rhythm, atrial fibrillation, PSVT (paroxysmal supraventricular tachycardia) etc. The more common usage of the term is for the more specific "PSVT" - a circus movement, or re-entry, tachycardia that most commonly occurs in the AV node or involves the AV as part of the circus and produces a narrow complex regular rhythm without P waves. For you physicists, this oscillation of this sustained electrical impulse is analogous to a harmonic electrical oscillation

Overview

- *AV node dependent tachycardias* **-** SVTs that require conduction through the AV node to maintain the tachycardia.
- *AVNRT* - atrioventricular nodal reentrant tachycardia.
- *AVRT* - atrioventricular reentrant tachycardia (via an accessory pathway).
- *AVNRT* and *AVRT* - majority of PSVTs. These tachycardias use a reentrant mechanism, and usually occur in younger patients without structural heart disease with a congenital substrate (dual AV nodal pathways or accessory pathway).
- *Circus Movement Tachycardia* = Another term that means reentry. Circus movement can occur on a microscopic level (eg within the AV mode to produce SVT or over an accessory pathway and produce some of the arrhythmia seen with WPW). Circus movement tachycardias are generally regular rhythms.





A Simplified Approach to Tachyarrhythmias

General Approach

- Treat the patient not the rhythm.
- Assess patient status: chest pain, dyspnea, CHF, hypotension, LOC
- Is dysrhythmia primary or secondary?
- Is the patient stable or unstable?

Electrical Cardioversion

- Paddle placement
  - AP best
  - Avoid implanted devices
- Electrode skin interface
- Synchronized
- Paper recording
- Energy levels
  - PSVT - 50 Joules
  - Atrial fibrillation - 100-200 Joules
  - Ventricular tachycardia - 100 Joules
- During exhalation

Overview

Notes

General Approach

1. *Plan on being stupid* - see introduction
2. *IV, O2, monitor, airway equipment, and defibrillator to bedside*. **Always prepare for the worst - as it often happens!!** Even arrhythmias that appear benign and stable can turn bad. All antiarrhythmic therapy is proarrhythmic, so your treatment may make the patient crash. In the end, the treatment of just about all bad arrhythmias is electricity. Be ready for rapid defibrillation/cardioversion and intubation.
3. *Is the patient stable or unstable?* Unstable patients are those with: ischemia chest pain, hypotension, altered mental status, poor perfusion, and pulmonary edema. **Patient "stability" is a continuum** from clearly doing just fine, to the patient in full arrest. Stability is also relative to what therapeutic options are available, for example, a distressed patient with PSVT may be stable enough to receive adenosine while preparing for cardioversion. In general, unstable patients receive cardioversion or defibrillation, though in some cases a trial of chemical therapy maybe tried first. (It is important to note that up to 70% of patients with tachyarrhythmias will complain of chest pain that sounds ischemic. The vast majority of these people will not have MI's and really are not "unstable". If the patient has a reasonable chance of underlying coronary artery disease then take the chest pain seriously and act appropriately. If they are young and healthy, a little chest pain is of much less concern).
4. *Is the rate regular or irregular?* Generally this is easily answered by a cursory look at the ECG. At very rapid rates the distinction can be a little difficult. In addition, the exact definition of what constitutes irregular is not clear. Fortunately in most cases the categorization is clear. The grouping of rhythms in this way is important because irregular rhythms are almost always generated above the AV-node and are treated with blockade of the AV node. **Ventricular tachycardia is very rarely irregular** except during the first few beats.
5. *Are the QRS complexes narrow or wide?* In an adult > 0.12 msec (or 3 small squares) is considered "wide". Usually, wide complex rhythms are much greater than 0.12msec. In a child < 8 years old, wide = >0.08 msec. In general, the widest QRS complex in the 12 lead ECG is the one to measure. Again, the nice thing is that it is usually clear if the rhythm is narrow or wide. Wide rhythms occur for two main reasons: 1) there is a block of the normal conduction pathway (bundle branch block), or 2) the rhythm starts in the ventricle and does not use the normal conduction pathway. **It is always safest to assume that a wide complex rhythm is coming from the ventricles and represents ventricular tachycardia.**
6. *Are there P waves?* Sometimes you really have to look. Try a different lead if you are not sure. If you see P waves, are they followed by QRS complexes in a regular manner. In addition look for (atrial) flutter waves: regular saw-tooth looking deflections of the ECG baseline. If there are distinct P waves look to see if the P waves are the same shape (morphology). In multifocal atrial tachycardia (MAT), there are at least three different P waves, and the rhythm is irregular. In reality**, the most important thing is to determine if the rhythm is sinus or not.** The difference in therapies between stable atrial fibrillation, atrial flutter and MAT is not very great and misdiagnosis between these groups is usually of little consequence in the acute setting. (In unstable MAT cardioversion is not helpful, in this situation it is important to differentiate MAT from atrial fibrillation or flutter).

Overview

Differential Diagnosis of Tachycardia

What is heart rate? Is QRS wide or narrow? Is rhythm regular or irregular?

	Narrow	Wide
Regular	sinus tachycardia reentrant SVT atrial flutter	ventricular tachycardia supraventricular tachycardia with bundle branch block, WPW, IVCD, or aberrant conduction
Irregular	atrial fibrillation atrial flutter with variable block atrial tachycardia with variable block multi-focal atrial tachycardia	atrial fibrillation with bundle branch block, WPW, IVCD, or aberrant conduction torsades de pointes

Tachycardia Therapy

	Narrow	Wide
Regular	adenosine calcium channel blockers beta adrenergic blockers	Lidocaine magnesium procainamide amiodarone
Irregular	calcium channel blockers beta adrenergic blockers digoxin procainamide Ibutilide amiodarone	calcium channel blockers beta adrenergic blockers digoxin procainamide magnesium ibutilide amiodarone

Tachycardia Therapy - Therapeutic Options

Treat underlying cause
- Sinus tachycardia
- Myocardial ischemia, hypoxia, hypotension
- Drug toxicity

Cardioversion
- VF - defibrillation 200, 300, 360 Joules
- VT - 100 Joules
- Atrial fibrillation - 50 Joules
- Atrial flutter - 25 Joules
- Re-entrant SVT - 25-50 Joules

Vagal maneuvers
- Valsalva
- Ice packs
- Carotid sinus massage
- Depresses sinus and AV nodes

Adenosine
- Depresses sinus and AV nodes
- AV nodal re-entry tachycardia
- May help with diagnosis of atrial fibrillation

Beta blockers
- propranolol
- esmolol
- metoprolol

Verapamil and Diltiazem
- Slows AV node conduction
- Terminate AV nodal re-entry tachycardia
- Control ventricular rate in atrial fibrillation
- Negative inotropes

Magnesium
- recurrent VF/VT if magnesium deficient
- torsades de pointes
- also slows AV node conduction

Lidocaine
- Ventricular fibrillation
- Ventricular tachycardia
- Ventricular ectopy during AMI

Procainamide
- Ventricular tachycardia
- Atrial fibrillation with accessory pathway
- Not with QT prolongation or torsades

Digoxin
- Sinus node slowing
- Depresses AV nodal conduction
- Control ventricular rate in atrial fibrillation

Amiodarone
- VT, ventricular (or atrial)

Overview

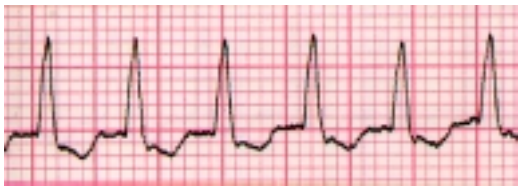
Regular, narrow complex tachycardia,

Diagnostic possibilities include (but not limited to):

- PSVT

- Atrial flutter with consistent block (i.e. not variable block), especially if ventricular rate is 150/min
- Orthodromic WPW
- Sinus tachycardia

Case 1: Regular Narrow Complex
47 year old female presents with 2 hours of palpitations and one brief episode of light-headedness. PMH reveals NIDDM and she uses glyburide. No other symptoms. She arrives by ambulance after two doses (12 mg) of adenosine with HR=220 and BP=143/90. She appears comfortable and stable.



Therapy for Stable Patients:
All these arrhythmias can be treated in a very similar manner, with blockade of the AV node. In the case of PSVT and antidromic WPW, this will break the circuit and stop the arrhythmia. In atrial flutter or atrial fibrillation the AV node block will not stop the arrhythmia, as the rhythm is generated in the atrium (see later section), but it will produce a block in the AV node which will make the diagnosis obvious. The intial treatment choice is **adenosine**. Verapamil is also very effective, as are diltiazem and beta-blockers. Adenosine is generally preferred, because it has a very short half-life and is less likely to cause prolonged hypotension (since it is rapidly metabolized in about 10 seconds). The calcium channel blockers are also very effective, but verapamil is a negative inotrope and vasodilator that can result in hypotension. This effect is reduced by pre-treating with calcium (eg. 10 mls of 10% calcium gluconate or 5 mls of calcium chloride).

Suggested Approach
Adenosine: 6mg IV push rapid

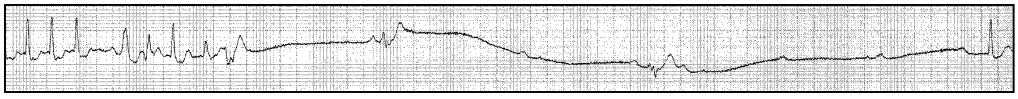
Adenosine: 12 mg IV push rapid

Verapamil: 2mg/min at a dose of 5 to 10mg
(repeat to no more than a total of 20mg and consider calcium pre-treatment)

Alternatives:
dltiazem, esmolol, metoprolol, or propranolol

If the patient is unstable or becomes so then cardiovert. The energy level for cardioversion is slightly different for each rhythm in this group. Simplistically, start at 50 J and go up by 50 J with each attempt until the patient has cardioverted.

In Case 1, above, the patient was treated once more with adenosine (12 mg) presuming that a very rapid push may be effective and produced transient bradycardia quickly reverting to sinus rhythm.



PSVT

Adenosine vs. Verapamil for PSVT:
In the stable patient with PSVT there has been controversy about the use of adenosine versus verapamil for therapy. Both are very effective. The pro's and cons are listed below. Ultimately it comes down to personable preference.

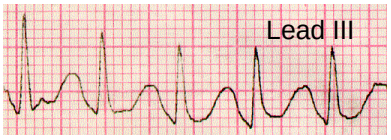
Pro Adenosine Arguments	Pro Verapamil Arguments
Ultrashort acting - less chance of sustained hypotension than verapamil	Cheap!
"Safe" if given to VT (verapamil definitely not safe in VT)	Effective - less recurrence than with adenosine
	No chest pain (as occurs with adenosine treatment)

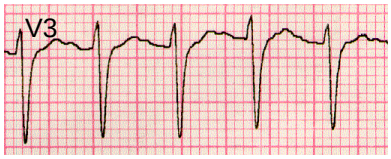
Adenosine Drug Interactions

- The effect of adenosine is potentiated in the face of: Tegretol and Persantin and especially in patients with heart transplants! In these patients start at a lower dose (eg 1mg and double with each dose).
- In patients taking methylxanthines adenosine may not be effective and verapamil is the preferred agent.

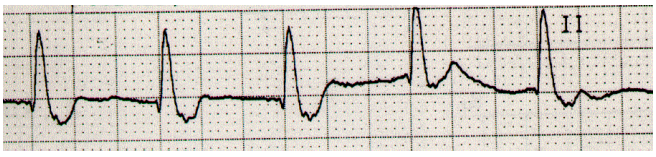
ST Segment Depression Associated with PSVT:
Many patients with PSVT develop ST segment changes (most commonly ST segment depression). This raises concern for ischemia. In general PSVT is a very rare cause of ischemia that leads to infarction (Imrie et al Can J Cardiol 6(8)323-6 1990). These ST segment changes are either repolarization changes or otherwise insignificant in the majority of patients. In elderly patients or those with a high probability of ischemic heart disease these ST segment changes may represent real ischemia and the patients should be observed and ruled out for an MI. Symptoms such as chest pain or dyspnea will help identify these cases.

Case 2: Regular Narrow Complex
A 26 year old, previously healthy, mother of three is brought by ambulance for a sudden deterioration in mentation at home after a 3 day gastroenteritis type illness. She has no chronic medical problems. With a HR=180 and BP=75/47 she appears pale, diaphoretic, morbidly ill and confused being unable to provide much history.





She had been given adenosine pre-hospital and while preparing for cardioversion in ED she was given one more dose of adenosine resulting in a transient slowing of the rhythm.



PSVT

Therapy for Unstable Patients:
Unstable patients frequently require cardioversion. Before cardioverting ask a few important questions:

- Is this person really unstable (see approach to arrhythmias)?
- Will the cardioversion be successful?
- (In patients with chronic atrial fibrillation cardioversion may not be successful. Certainly in this situation atrial contraction will not occur for days to week and cardioversion is really only a form of rate control (and a painful form at that!)
- Can I sedate the patient?

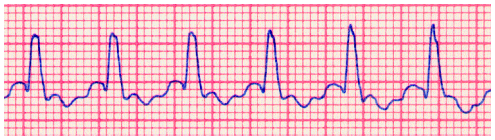
Sedation for Cardioversion
It is important at least to consider sedation of patients that are being cardioverted. This can be a very painful process. Unfortunately many of these patients are being cardioverted because they are unstable and the use of sedation may complicate already poor cardiac function.

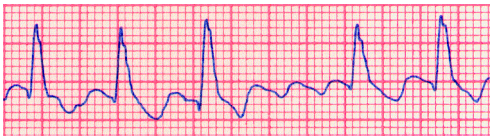
- Deep sedation
- Drugs
  - etomidate
  - propofol
  - barbiturates
  - benzodiazepines
  - opiates

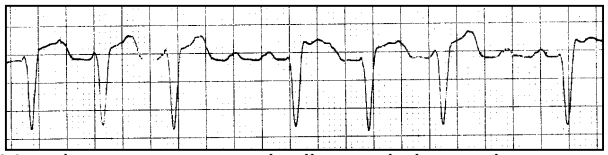
Note: everyone has their own preference, there is no right answer. A short-acting barbiturate is the preference of many physicians. All sedatives and analgesics potentially will drop blood pressure.

Case 2: Therapy
She was sedated with methohexital and cardioverted 3 times (100, 200, 300 J) with no change in her tachycardia. After about 30 minutes in ED, and after 1500 cc of NS her heart rate had decreased to 150 with discernible P waves representing a sinus tachycardia. A rectal temperature was noted to be 105.5^0 F and she was treated for sepsis but expired 24 hours later. This case represents a misdiagnosis of sinus tachycardia.

Atrial Flutter


72 yo male with palpitations


Adenosine reveals flutter waves

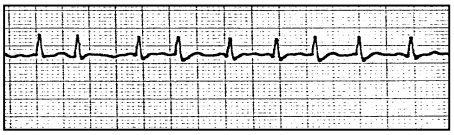

Vagal maneuver reveals diagnosis in another example

PSVT

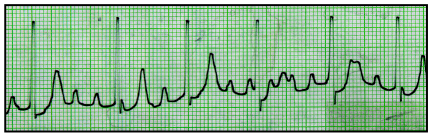
Irregular, narrow complex tachycardia:

Diagnostic possibilities include:

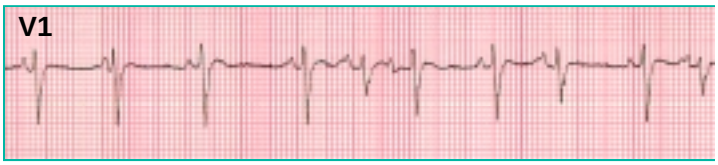
- Atrial fibrillation



- Atrial flutter with variable block



- MAT (if you don't look closely to find the differing P waves)



Therapy for Stable Patients:
Irregular rhythms without P waves are almost always atrial fibrillation or flutter with a variable block. **The aim of treatment, in the stable patient, in this circumstance is not to convert the rhythm but to slow the ventricular rate.** This is achieved by blocking the AV node.

A Suggested Approach

Diltiazem: 5mg slow IV and repeat every 5 mins to a total of 50 to 60mg if required.
(Consider pre-treatment with calcium as for verapamil.)

To continue to control the rate, a drip can be run at 5-10 mg/hour

Alternate Strategies

Verapamil: Same dose as for PSVT

Digoxin: 0.5mg IV or PO and 0.25mg 4 hours later
(very slow onset of action)

Others: magnesium, esmolol

Therapy for Unstable Patient:
This can present a real problem. Patients with chronic atrial fibrillation, structural heart disease, and cardiomyopathy will often fail cardioversion making restoration of sinus rhythm unattainable. If the patient is "really unstable" then cardioversion is required but may be unsuccessful. Start at 100 Joules and increase as required. The problem is that many patients with atrial fibrillation have big floppy hearts that do not cardiovert to a sinus rhythm easily, or remain in sinus rhythm if they convert. In addition, if the patient has been in atrial fibrillation for more than 2-3 days, cardioversion can cast off an atrial clot and cause a stroke. For these reasons **cardioversion is frequently the last resort.** An emerging therapy is magnesium bolus followed by an infusion. Give 2 to 4 grams over 5 to 10 minutes and follow with an infusion of 1-2 grams per hour. This has been associated with a reduction in heart rate and conversion to sinus rhythm.

Atrial Fibrillation Atrial Fibrillation
Overview

- Most common sustained dysrhythmia seen in ED
- Mortality at least doubles compared to age-matched controls

Therapeutic Principles

- Rate control
- Restoration of sinus rhythm
- Prevent thromboembolism

Pathophysiology

- Rapid, chaotic atrial quivering at a rate of 350-600 cycles per second
- Causes rapid, irregular, ventricular response rates decreasing cardiac output
- Stagnating blood forms clots

Etiologies

- Acute
  - "holiday heart" or "lone" fibrillation
  - thyrotoxicosis
  - drug toxicity
  - WPW
- Chronic
  - hypertension
  - ischemic cardiomyopathy
  - valvular disease
  - cor pulmonale
  - CHF

Presentations

- Palpitations
- Dyspnea
- Syncope
- Chest pain
- Thromboembolism

Therapy

- Unstable
  - electrical cardioversion
- Stable

  Rate Control
  - Calcium antagonists
  - verapamil
  - diltiazem
  - Beta blockers
  - metoprolol
  - propranolol
  - Magnesium
  - Digoxin
  - Amiodarone

  Restore Sinus Rhythm
  - Procainamide
  - Propafenone
  - Amiodarone
  - Ibutilide
  - Amiodarone

  But >50% spontaneously convert within 24^0

Atrial Fibrillation

Therapy of atrial fibrillation

- Stable
  Prevent thromboembolism in chronic atrial fibrillation - anticoagulation
  Warfarin decreases stroke rate by 68% with 1.3% bleeds and aspirin decreases stroke rate by 21% with a little less bleeding (1.0%). *(Gorelick JAMA 1999*) Warfarin is indicated in patients with:
  - Valvular disease
  - Age > 75
  - Non-valvular with risk factors
  - *CHF, HTN, DM, previous TIA or stroke*

  Warfarin or aspirin is indicated in patients: *(Arch Int Med 1997)*
  - Age 65 -75 without risk factors
  - Aspirin
  - Age < 65 with no risk factors

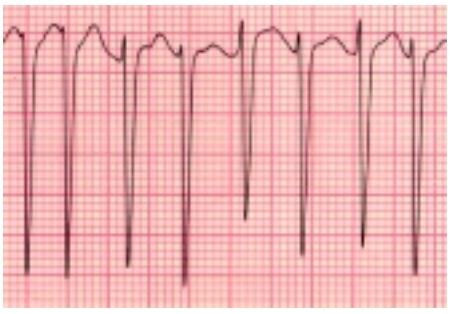
  Prevent thromboembolism in acute atrial fibrillation - anticoagulation
  20% of patients show clot by TEE if $<72^0$ duration and 30% develop clot by TEE after NSR due to "atrial stunning." The duration of atrial fibrillation does not correlate well with symptoms. *(Prince Am Heart J 1995 and Fatkin JACC 1994)* Admission is warranted for patients with significant chest pain, ECG ischemia, CHF, hypotension, and TIA or those who need anticoagulation.

  Out-Patient (*Mulcahy, AEM 1996*)
  - retrospective review
  - 216 admitted, 143 "medically justified," and only 3 of remaining 73 could not be identified in ED

  Out-Patient (*Michael, Ann EM 1999)*
  - retrospective review
  - 289 patients with 280 discharged
  - Cardioversion performed
    - 180 chemical (50% success)
    - 80 electrical (89% success)

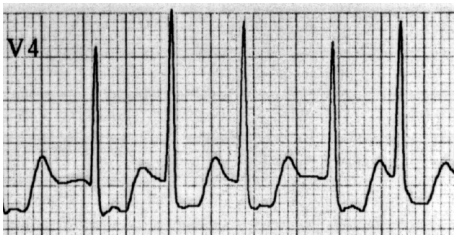
To summarize, out-patient management is an option for those patients whose rate is controlled, have no CAD risk factors, who may be treated with aspirin or LMWH and can be followed up soon.

Atrial Fibrillation - Case 1
72 year old male with CAD and CHF presents by ambulance with 1^0 severe CP and SOB. He appears ill; pale, diaphoretic, 2-3 word dyspnea with HR=200, BP=96/70, and RR=40. He is treated by electrical cardioversion with sedation but fails treatment after 3 shocks. Rate control is now necessary and diltiazem is administered. The patient expires 6^0 later due to CHF.



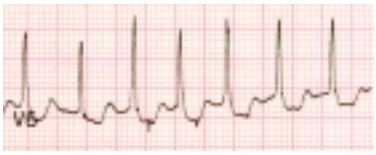
Atrial Fibrillation - Case 2

- 80 year old female with CAD presents with 1^0 severe CP radiating to neck. She appears ill and in severe pain, with HR=160, BP=93/71, RR=18, and pulse oximetrty 98%. This is an unstable patient with limited medication options who undergoes electrical cardioversion (100, 200, 300 J). She awoke with BP=160/100 and HR=140 and is next treated with diltiazem. HR decreases to 100, ischemia resolve, and she "ruled-in" with small CK-MB rise.



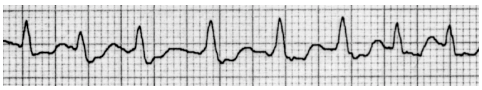
Atrial Fibrillation

Atrial Fibrillation - Case 3



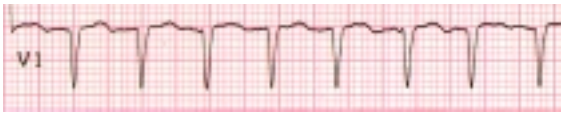
68 year old female with no PMH presents 2^0 of a "tightness in the chest … like something is stuck." She appears comfortable and stable with HR=180, BP=183/90, RR=20, and pulse oximetrty 98%. Is the patient stable or unstable? She is treated with diltiazem, magnesium, and NTG with minimal response. Her pain worsens and she is electrically cardioverted after 45' in ED with restoration of NSR. She is treated with heparin.

Atrial Fibrillation - Case 4



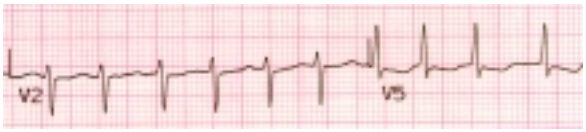
67 year old male with CAD and 3^0 CP. The patient appears comfortable and hemodynamically stable. He is treated with diltiazem and NTG.

Atrial Fibrillation - Case 5



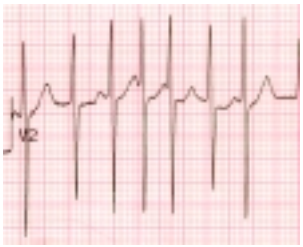
68 year old male with history of paroxysmal atrial fibrillation and HTN presents with palpitations. He takes atenolol and coumadin. He is hemodynamically stable and appears comfortable. He is treated with IV metoprolol to decrease heart rate to 90. His atenolol is increased to 100mg daily and the patient is discharged.

Atrial Fibrillation - Case 6



38 year old male soldier with several hours of palpitations and no PMH. He is stable with HR=150, BP=160/90, and RR=30. What treatment is necessary? He was sedated with fenatnyl and midazolam and electrically cardioverted. Perhaps beta-blocker therapy and out-patient follow-up would be safer.

Atrial Fibrillation - Case 7



26 year old female with CC of confusion and difficulty speaking. On exam she is alert and oriented but has trouble finding words and communicating. HR=130, BP=172/100, RR=24, T=99.6^0 Thyroid is enlarged. TSH is undetectable. She is treated with propranolol (inhibits conversion of T_4 - T_3) and PTU. CT scan a stroke in the left frontal cortex and she is given heparin.

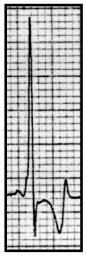
Wide Complex, Irregular Tachycardia

Diagnostic possibilities include:

- Atrial fibrillation with bundle branch block
- Atrial flutter with variable AV block and bundle branch block
- Wolf-Parkinson-White Syndrome and atrial fibrillation (please see the section on specific arrhythmias)
- Polymorphic VT or Torsades

Treatment is as for narrow complex irregular tachycardias (except in the case of atrial fibrillation and WPW and polymorphic VT). Cardioversion should not be attempted in a stable patient with atrial fibrillation (unless the possibility of WPW exists) unless one is sure the patient does not have an atrial thrombus that may dislodge and cause a stroke. This may be impossible to decide clinically. Therefore, 3 weeks of anticoagulation may be required (or, at the very least a transesophageal echo).

Wolff-Parkinson-White Syndrome

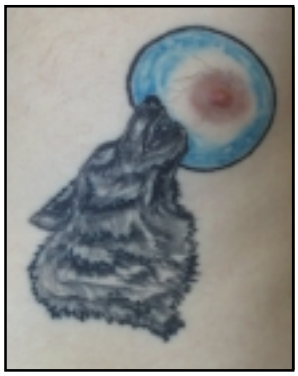


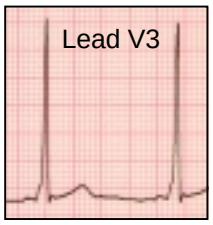
EKG pattern - variable fusion of ventricular depolarization via the accessory pathway (responsible for the short PR and delta wave) and the His Purkinje system.

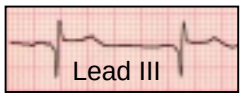
- Short PR interval
- Wide QRS
- Delta wave
- Associated ST - T changes
- Paroxysmal tachycardias

WPW Case 1

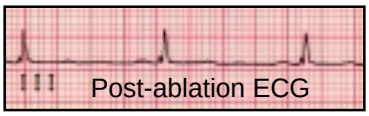
30 year old male soldier with exertional syncope, twice during last week and twice 2 years ago.
Examination is normal except for a remarkable dermatologic clue to diagnosis.



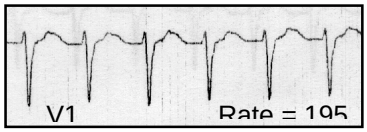




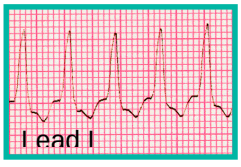
Notice “pseudo-infarction” pattern in inferior lead.

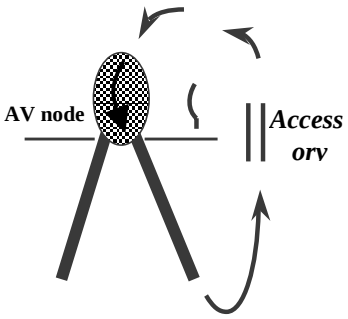


Orthodromic AVRT - most common arrhythmia in WPW.
EKG shows classic PSVT - regular narrow complex tachycardia, P waves buried in ST segment.
Circuit - down AV node and His-Purkinje system, up via accessory pathway.
Syncope - usually secondary to peripheral vasodilatation (via mechanism of neurocardiogenic syncope) as opposed to rate of tachycardia.
Therapy – vagal maneuver, adenosine, verapamil, beta-blocker

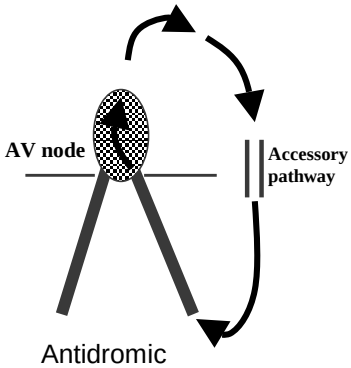


Antidromic atrio-ventricular tachycardia (AVRT)
wide complex regular tachycardia looking like ventricular tachycardia
lidocaine, procainamide, cardioversion
(vagal, adenosine, beta blocker)





Orthodromic AVRT

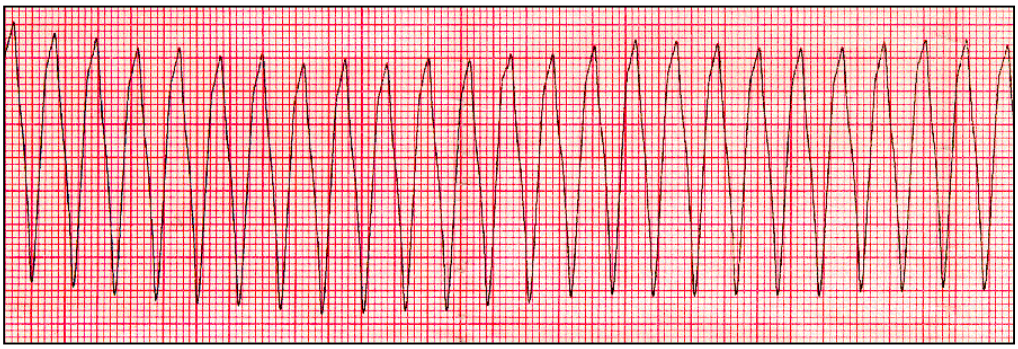


Antidromic AVRT

WPW

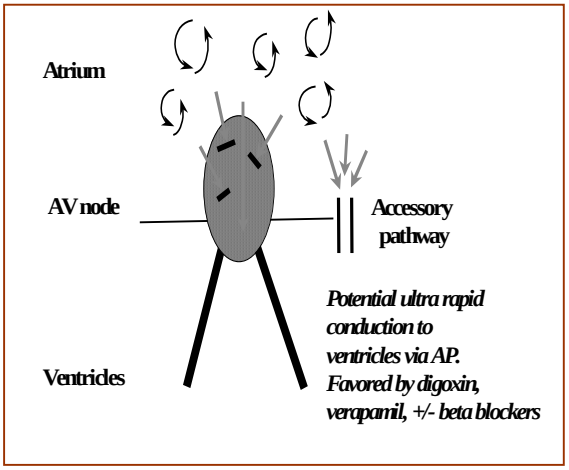
WPW Case 2

History - 17 year old female presents acutely ill and diaphoretic 30 minutes after developing near-syncope and palpitations. She has no chronic medical problems nor any previous syncopal episodes or cardiac difficulties. On exam she appears ill and uncomfortable with HR= 240 and BP= 70 systolic. ECG reveals:



ECG - A rapid, wide complex tachycardia which appears regular but may represent atrial fibrillation as RR intervals are very short. This patient is demonstrating conduction over an accessory pathway capable of extremely rapid conduction and she is at risk for hemodynamic deterioration.

Therapy - She underwent emergent cardioversion after sedation with methohexital and sinus rhythm was restored.

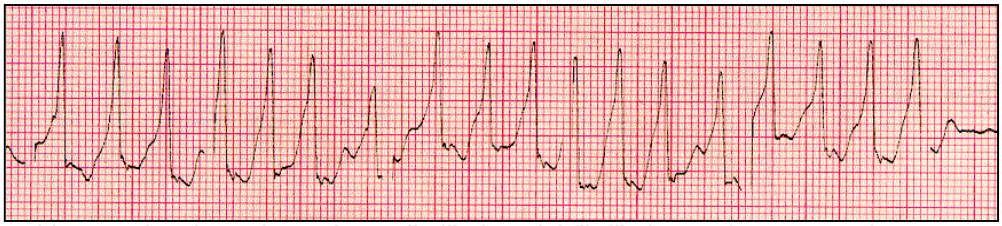


WPW and atrial fibrillation - second most common arrhythmia in WPW.
- Extremely rapid conduction via AP possible and can precipitate ventricular fibrillation and sudden cardiac death. Look for RR intervals <250 ms. These very short intervals represent rapid conduction over AP and these patients are at highest risk for degeneration to mortal ventricular tachydysrhythmias.
- AP conduction during atrial fibrillation favored by digoxin, calcium antagonists, adenosine and +/- beta blockade.
- In general, these agents should be avoided in patients with WPW syndrome. (Adenosine is indicated for narrow complex AVRT).
- Acute treatment.
  - Unstable - D.C. synchronized cardioversion.
  - Stable - intravenous procainamide slows atrial rate, may cardiovert; also increases AV node conduction.
- Chronic treatment.
  - Radiofrequency catheter ablation.
  - Class IA, IC, or III antiarrhythmic therapy.

WPW

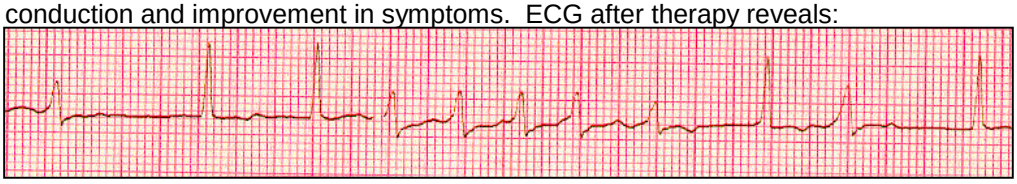
WPW Case 3

History - 30 year old male presents with mild light-headedness and palpitations. On one previous occasion he experienced syncope and was told he had WPW. He has had no further difficulties and takes no medicines. He denies chest pain or other symptoms. On exam he appears alert and comfortable with HR=180 and BP=146/88. ECG reveals:



wide complex, irregular tachycardia likely atrial fibrillation and WPW syndrome

Therapy - The patient was treated with a procainamide infusion resulting slowed conduction and improvement in symptoms. ECG after therapy reveals:

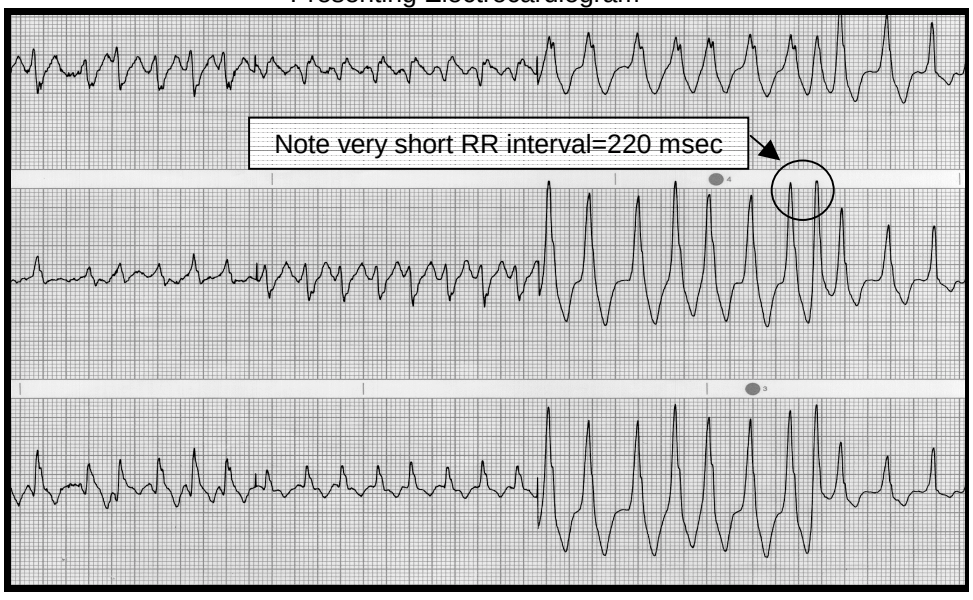


Follow-up - A few months later the patient underwent cardiac catheritization and electrophysiologic testing resulting in radiofrequency catheter ablation of his accessory pathway.

WPW Case 4

31 yo female nurse presents with palpitations and a history of WPW sydnrome currently taking no medicines. She appears alert, comfortable in no distress with HR=184, BP=144/95, RR=16, T=97.6. Further exam reveals: Neck - no JVD, Chest - clear, Heart - irregular, rapid, no murmur, Hct = 44%.

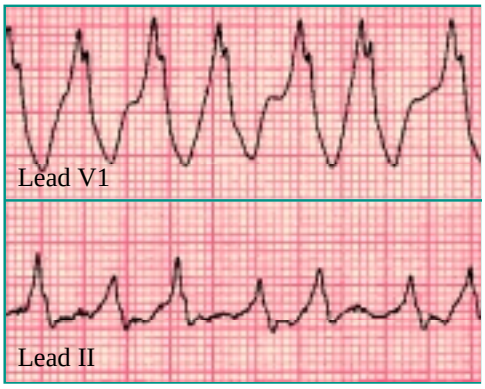
Presenting Electrocardiogram



WPW

WPW Case 4 - ED Course:

Procainamide IV infusion to total of 250 mg over 30 minutes was aborted due to hypotension. Blood pressure dropped to 90 systolic and rhythm became more irregular, wide, and bizarre. She did not tolerate procainamide and appeared more ill.
After sedation the patient was cardioverted at 50 Joules without success followed by 100 Joules which resulted in ventricular fibrillation. Defibrillation was successful and patient awoke in ED. She was referred for EP studies and underwent surgical ablation in 1986. She continues to work in the NICU.

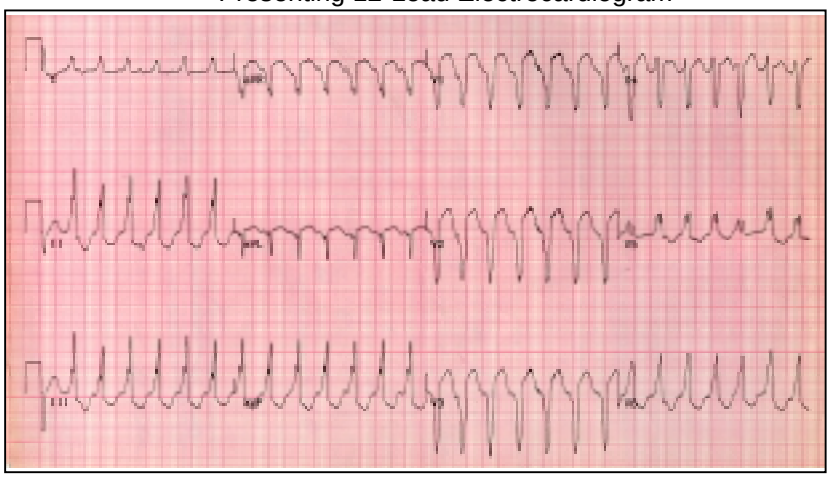


Wide Complex Tachycardia

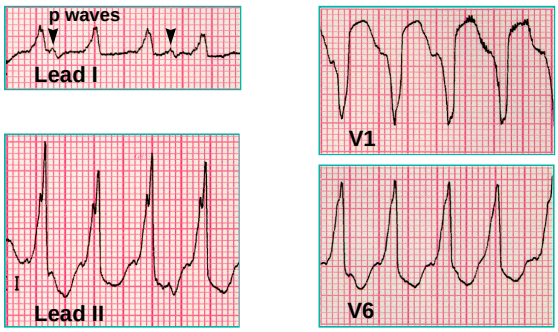
WCT – Case 1

30 year old male trisomy 23 presents with abdominal pain. He has a history of congenital heart disease (tetralogy of fallot) and recurrent VT having failed multiple antiarhythmic agents, currently on no medicines. On examination he appears pale, diaphoretic and ill writhing in pain; HR=160, P=96/70.

Presenting 12-Lead Electrocardiogram



ED Course:
Ventricular tachycardia is the diagnosis. Atrioventricular dissociation (Lead I) is diagnostic. The patient was initially given lidocaine with no change in status (as might be expected - his problem is not ischemia). He was then sedated and cardioverted.



Wide Complex Tachycardia

Differential Diagnosis of Wide Complex Tachycardia
(QRS>120 msec and heart rate >120 beats/min)

- Ventricular tachycardia
- Supraventricular tachycardia with abnormally wide QRS complexes

SVT may be:	*Wide QRS complexes may be due to:*
sinus tachycardia	aberrant ventricular conduction
SA nodal reentrant tachycardia	preexisting bundle branch block
atrial flutter with 2:1 conduction	preexisting non-specific IVCD
automatic atrial tachycardia	antegrade conduction through bypass tract (WPW)
intraatrial reentrant tachycardia	antegrade conduction over atriofasicular or nodoventricular tract
AV nodal reentrant tachycardia	
automatic AV junctional tachycardia	
AV reentrant tach using bypass tract	

A regular wide complex tachycardia should be considered to be VT until clearly proven otherwise. Therapeutic decision making is safer using this assumption. It is difficult (if not impossible) in many (or most) circumstances to differentiate VT from SVT with aberrancy *(Herbert ME Ann Emerg Med 1996).* Indeed, it is may be dangerous to do so if SVT is mistakenly diagnosed and treated with verapamil. VT is treated with **magnesium** or **lidocaine** and (if this fails) **procainamide**. Both magnesium and procainamide are safe, and sometimes effective in SVT. Adenosine is useful if you are not sure of the diagnosis, since it is probably safe in VT, and will convert SVT. On the other hand, *giving verapamil to VT may cause cardiac arrest* in some patients.

WCT History and Examination
History favors ventricular tachycardia if:
prior MI or CABG, CHF, recent angina, antidysrhythmic therapy
Akhtar (1991) found 87/89 patients with a history of MI and a WCT had VT as the etiology of the WCT
Examination favors VT if cannon "A-waves" are present. Hemodynamic status is irrelevant.
Overall, when treating a patient with a regular wide complex tachycardia the diagnosis is most often VT and therapy directed towards VT (lidocaine, cardioversion) will be the safest approach.

WCT ECG Diagnosis
There are numerous publications and criteria for differentiating a VT from wide complex SVT. Wellen's (1978) criteria are the most well known (QRS duration > 140 msec, left axis deviation, certain QRS morphologic characteristics, AV dissociation). However, the usefulness of this approach was lacking because there is no heirarchy to the presence of some findings but not others (except for AV dissociation). More recently, Brugada (1991) offered an approach using sequential criteria that establish the diagnosis in an algorithmic form. A yes answer to a question diagnoses VT and terminates further use of the algorithm.
Brugada reported 554 episodes of tachycardia. (384 VT, 170 SVT) (sensitivity = 99%, specificity = 95%)

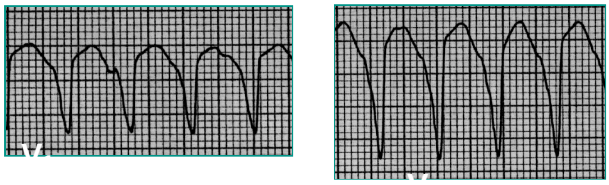
Brugada Criteria for VT

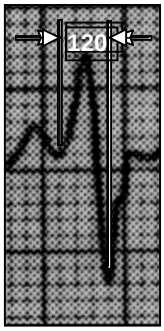
1. Absence of RS complex in all precordial leads? (yes = VT)
2. R to S interval > 100 msec in one precordial lead? (yes = VT)
3. AV dissociation present? (yes = VT)
4. Morphology criteria present both in precordial leads V_{1-2} and V_6 (yes = VT)

Wide Complex Tachycardia

Brugada Criteria for diagnosis of WCT

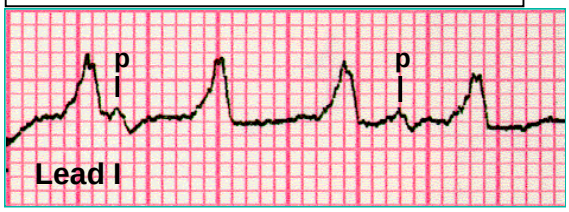
1. Absence of RS complex in all precordial leads? (yes = VT)



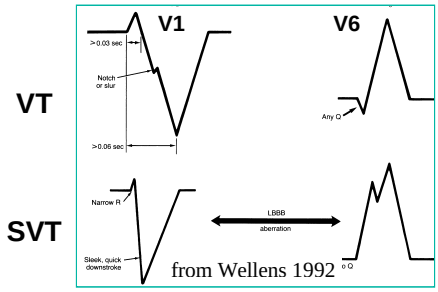


2. R to S interval > 100 msec in one precordial

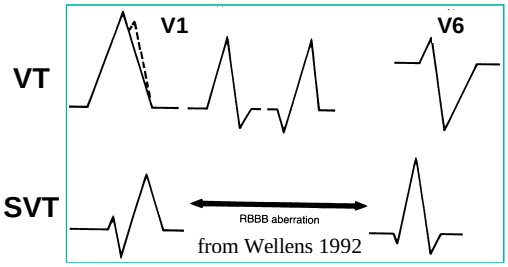
3. AV dissociation present? (yes = VT)



4. Morphology criteria present both in precordial leads V_{1-2} and V_6 (yes = VT)

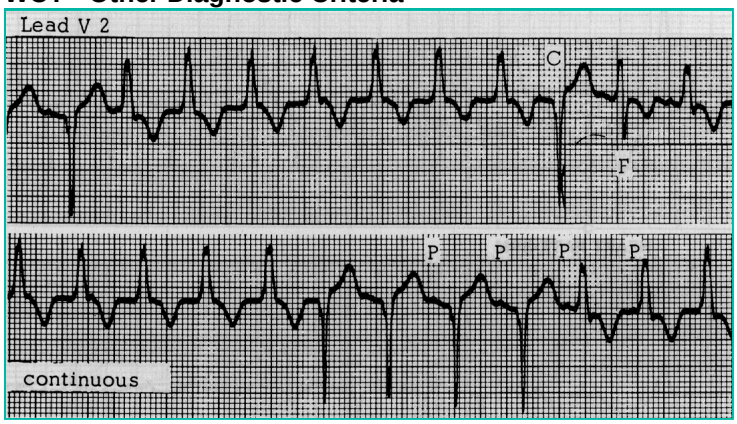


Left Bundle Branch Block Configuration



Right Bundle Branch Block Configuration

WCT - Other Diagnostic Criteria



from Chou 1996

C represents a capture beat where a normal, but early, QRS complex appears during VT indicating a supraventricular origin of this one complex thus confirming the diagnosis of VT.

F is a fusion beat which also indicates VT as impulse formation is partially occurring in the ventricle. Ventricular capture of an atrial impuls occurs about the same time as a spontaneous ventricular depolarization occurs resulting in a QRS complex that is intermediate in configuration. P indicates the presence of P waves.

Therapy of Wide Complex Tachycardia

Suggested Approach for Stable Patient
If SVT is considered likely, i.e. younger patient with no heart disease,
(The best predictor of VT is underlying structural or ischemic heart disease.)
then use **adenosine** 6 mg and repeat at 12 mg

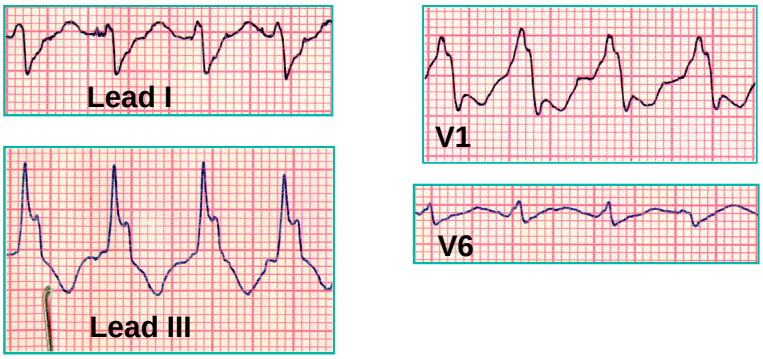
magnesium 2-4 grams over 2-4 minutes, then an infusion of 1-2 gm/hr
(ACLS says use lidocaine 100 mg IV, repeat in 10 min if no response. However, lidocaine is only about 20% effective at converting VT and Mg may be better.)

procainamide 17 mg/kg slow IV
(Do not infuse at greater than 20 mg/min, stop for hypotension, stop for QRS widening of >50% initial value)

Cardioversion 100 J initially and go up from there.
There are other alternatives, but by this point the patient has been in the rhythm for a long time and it's time to get aggressive before condition deteriorates.

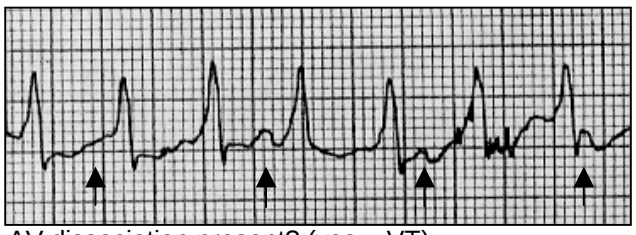
For unstable patients: cardioversion at 50-100 Joules is the best therapy. If the rate is very rapid the defibrillator may not sense to synchronize and unsynchronized mode may be required. **Again, regular wide complex tachycardia is VT until proven otherwise…calcium channel blockers should not be used!**

WCT – Case 2
75 yo male with hx of CAD presents with several days of weakness, increasing exertional angina, and some dyspnea with mild leg swelling. On exam is alert, stable, comfortable with HR=140, BP=130/70, in mild CHF.



ECG demonstrates VT by R to S interval > 100 msec.
Therapy with lidocaine and 2 grams of magnesium abolished the tachycardia.

WCT - Case 3
92 yo female with 2 days of weakness. History of hypertension only. Meds include HCTZ and levothyroxine. On examination she appears confused, pale with HR=150, BP=90.

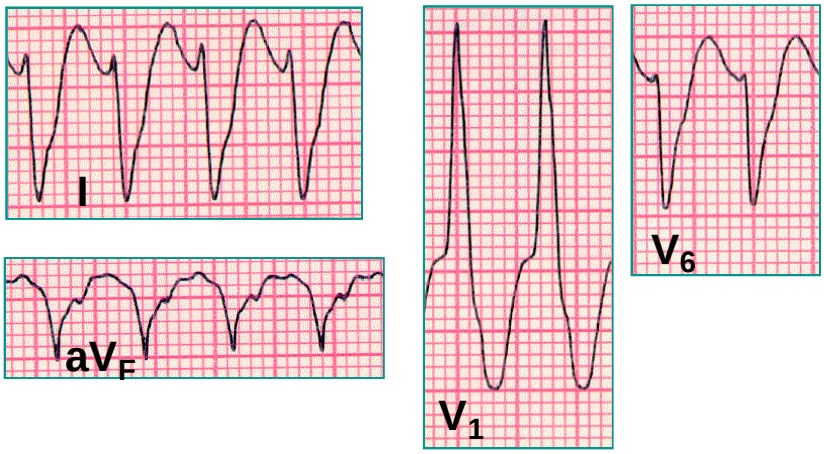

AV dissociation present? (yes = VT)

Wide Complex Tachycardia

She was initially treated with lidocaine, sedated, and rapidly cardioverted because she was hemodynamically unstable.

WCT – Case 4

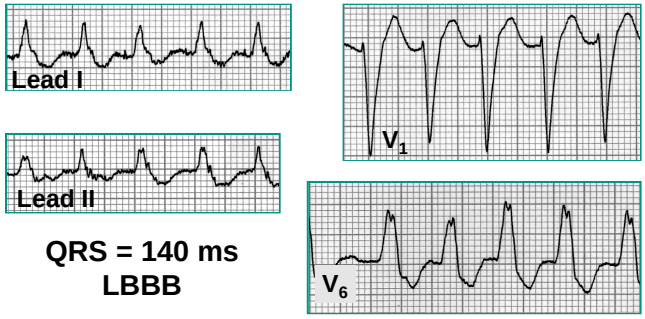
78 yo male with one hour of chest pain and a history of CAD. After "fender-bender" MVA and discussion with police symptoms developed. Meds include procainamide and NTG. On examination he apeears mildly ill with a rapid, regular heart rate at 200 beats per min and BP = 180/100.



ECG demonstrates ventricular tachycardia with RBBB configuration meeting both morphology criteria for VT (large, monophasic R in V_1 and rS in V_6.
Lidocaine bolus did not succeed and because of patient's level of distress with chest pain, in addition to his history of CAD, he was deemed unstable, sedated, and cardioverted.

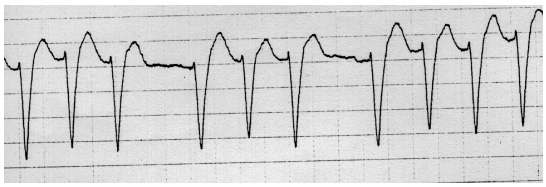
WCT - Case 5

82 yo female with an hour of chest pain and a rhistory of CAD. On exam she appears comfortable with HR=160, BP=189/110. An old ECG reveals pre-existing LBBB. The current tracing does not meet morphology criteria for VT.

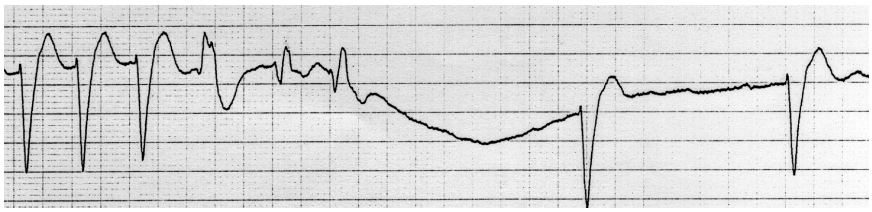


QRS = 140 ms
LBBB

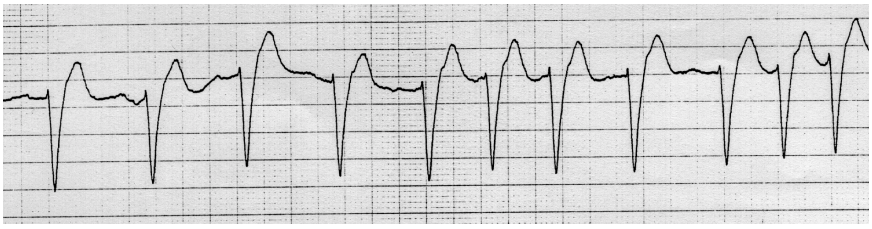
ED Course:
A valsalva maneuver momentarily interrupted the tachycardia.



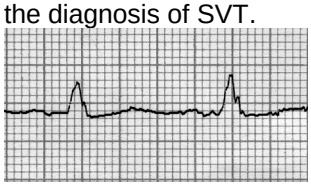
Adenosine briefly restored sinus rhythm.



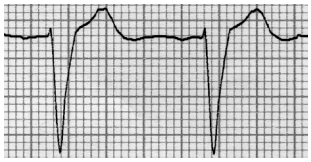
But tachycardia recurred.



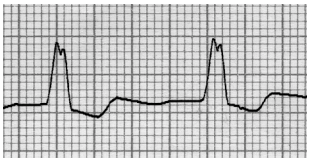
While preparing to treat the patient with beta blocker (esmolol) she spontaneously converted to sinus rhythm with the same QRS morphology (LBBB) thus confirming the diagnosis of SVT.



Lead II



V_1



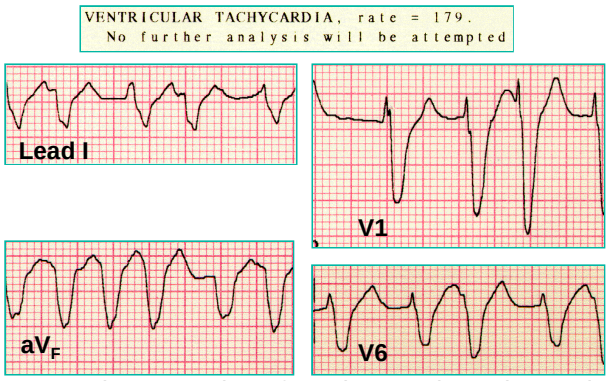
V_6

Outcome:

The patient was admitted and ruled in for AMI with CK-MB = 45 ng/ml. She underwent coronary angiography which revealed obstructive disease similar to that seen on previous study several years earlier. Her myocardial necrosis was rate related and due to a fixed lesion.

WCT - Case 6

82 yo male with 36 hour of dyspnea and history of CAD and CHF. meds include digoxin, nitrates, furosemide and ACE inhibitor. On exam he appears stable and comfortable with HR=180 and BP=145/82.

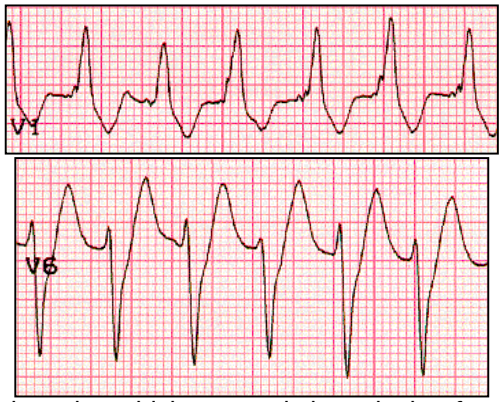


Despite the computer interpretation of VT the very irregular RR intervals demonstrate atrial fibrillation. When atrial fibrillation is very rapid it may be difficult to discern the irregularity causing the errant diagnosis of VT.

Case 7

34 year old male presents with one hour of palpitations and a history of tachycardia. He was discharged from the Army after 2 syncopal episodes and an electrophysiologic study that did not provoke the tachycardia. His presumptive diagnosis was idiopathic VT initially treated with beta blockade which he is not using now. He has had no symptoms in six years. On examination he appears mildly sweaty but otherwise stable with HR=160 and BP=162/90.

ECG reveals a wide complex tachycardia that meets Brugada criteria for RBBB configuration with monophasic R in V_1 and rS in V_6.



Therapy began with adenosine which restored sinus rhythm for a few minutes but complex atrial and ventricular ectopy ensued and the tachycardia recurred. Furhter therapy included magnesium, lidocain, and procainamide. After one to two hours on a procainamide infusion a normal sinus rhythm persisted.

He later underwent EP study. The presumptive diagnosis was idiopathic left ventricular VT which sometimes responds to adenosine. However, AVNRT was induced along with RBBB and LAFB aberrancy. The nodal tissue was ablated. The patient experienced a TIA (homonymous hemianopsia) believed to be catheterization related.

This case demonstrates a false positive in Brugada's scheme in addition to the difficulties encountered in diagnosis of WCT. However, the therapeutic algorithm we have discussed was appropriate and beneficial, nonetheless.

Wide Complex Tachycardia – Summary

Approach to Patient

Treat the patient not the rhythm. Assess the patient for chest pain or dyspnea, pulmonary edema, hypotension, or decreased mentation. Is dysrhythmia primary or secondary? 80-85% of all WCT is VT ECG criteria for diagnosing VT are difficult to remember and interpret.

Therapy includes magnesium, lidocaine, adenosine, procainamide, sedation and cardioversion.

Pitfalls include assuming SVT, treating with verapamil, delaying cardioversion, and treating with a type Ia antidysrhythmic in a patient on chronic therapy.

Appendix

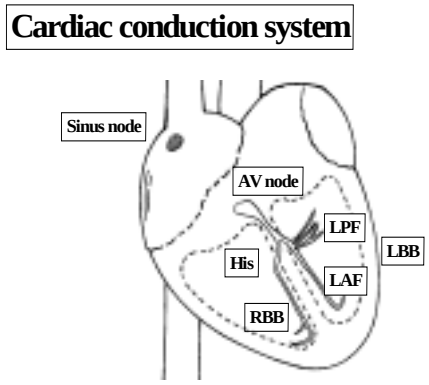
Clinical Electrophysiology

Conduction system of the heart:

Sinus node - located in high right atrium. Exhibits automaticity - can be suppressed with over drive pacing.

AV node - Exhibits automaticity (as escape pacemaker rhythm) and decremental conduction. In normals, only connection between atrium and ventricle.Conduction speed ~ 1/20 of His-Purkinje system, ~1/3 of atrial or ventricular muscle.

His-Purkinje system - rapidly conducting tissue that divides into right and left bundles; left bundle further subdivides into anterior and posterior fascicles.



Vascular supply to conduction system:

Sinus node - supplied by right coronary artery ~ 55%, left circumflex ~ 45%.

AV node - from RCA in ~ 95% of patients (dominant or co-dominant systems); from LCFX in ~ 15% patients (dominant or co-dominant).

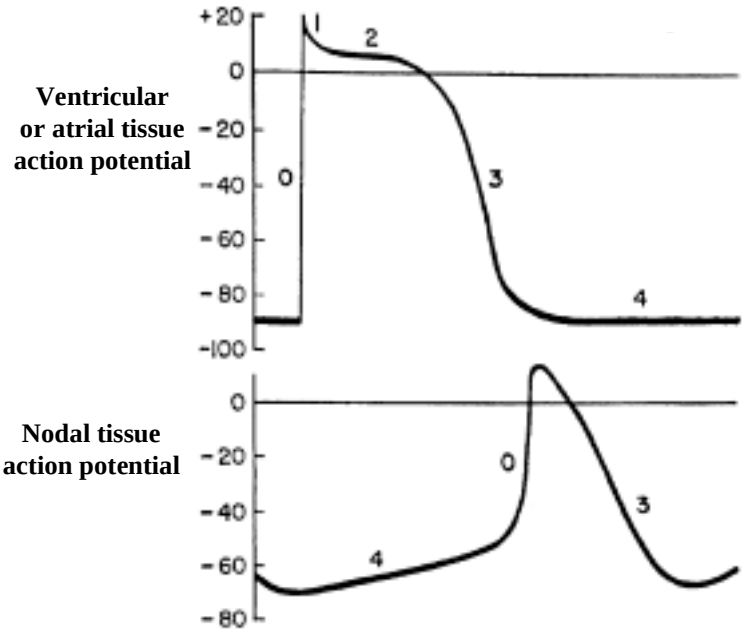
His bundle - receives blood supply from both LAD and RCA.

Right bundle - Receives blood supply from RCA

Left anterior fascicle - receives blood supply from LAD.

Left posterior fascicle - dual blood supply from LAD and RCA.

Action potentials.



Action potentials.

Atrial or ventricular myocyte - “fast sodium channels”.

Characterized by highly negative, flat baseline phase 4 potential (~ -90mV).
With stimulus sufficient to reach threshold, rapid depolarization occurs in phase 0, mediated by "fast" sodium channels. Class 1 agents block sodium channels, slowing conduction as mechanism of antiarrhythmic action.
Phase 1 - brief repolarization mediated by outward potassium current.
Phase 2 - plateau potential, mediated by the inflow of "slow" calcium current. Responsible for electromechanical coupling of the action potential to myocyte contraction.
Phase 3 repolarization is mediated by potassium channels. In early phase 3, cell is absolutely refractory; in late phase 3, the cell is relatively refractory. Prolongation of refractoriness characterizes the mechanism of action of Class III agents.

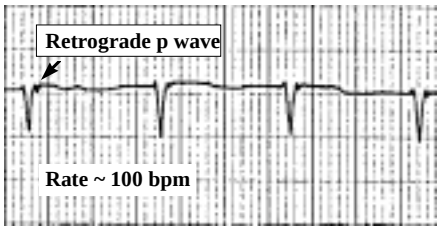
Nodal cells - "slow calcium channels"
Baseline potential less than myocyte (~ -65 mV), characterized by spontaneous decay of potential to threshold (automaticity).
Automaticity is increased by catecholamine stimulation, decreased by acetylcholine.
Phase 1 depolarization mediated via "slow" calcium channels - site of activity for calcium antagonists.

Mechanism of arrhythmias.

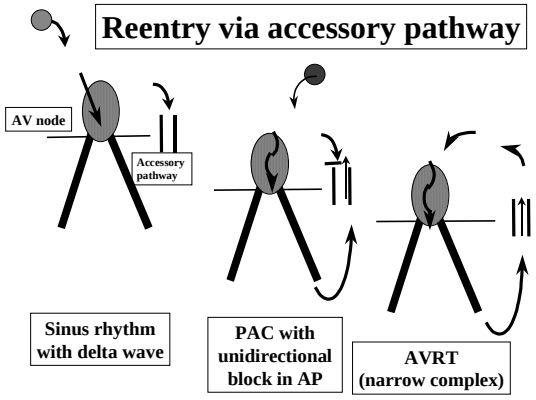
Automaticity.
Enhanced - increased firing of cells that normally exhibit automaticity, such as inappropriate sinus tachycardia or nonparoxysmal junctional tachycardia.
Abnormal - automatic firing of tissues that normally don't exhibit automaticity, such as MAT, AIVR.

Nonparoxysmal junctional tachycardia

Enhanced automaticity at AV node secondary to digoxin toxicity



Re-entry.
Common proximal and distal tissue.
Parallel pathways - one pathway with anterograde unidirectional block, alternate pathway with slowed conduction.
*AV node in PSVT - exhibits slowed conduction necessary for re-entry.

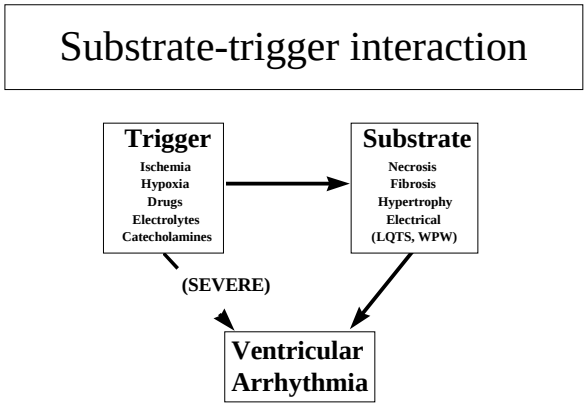


Triggered rhythms - result of early or late after depolarizations occurring as result of prolonged repolarization. (e.g. - Torsades).

Substrate - Trigger interaction of arrhythmias.

Appendix

Substrate - Structural or electrical condition necessary to allow arrhythmia to be manifest.
Common substrate abnormalities - (ventricular rhythms):
Necrosis or scar (MI)
Fibrosis (dilated cardiomyopathy).
Hypertrophy - LVH or HCM.
Electrical - WPW, Prolonged QT, or primary ventricular fibrillation.
Triggers - acute stimulus that induces arrhythmia. Common triggers include: Electrolyte, drug, catecholamines, hypoxia, ischemia.



Supraventricular Tachycardias

Definition of SVT: Diverse group of tachyarrhythmias that share their origin in the atrium, or require the AV node as a critical component of the arrhythmia.

Approaches.

Historical - Atrial fibrillation vs. PAT (paroxysmal atrial tachycardia), because all SVT was considered to originate in the atrium. Outdated term - misleading at best.
Catalog approach - Classifying arrhythmia as per mechanism or P to QRS relationship. Often requires electrophysiologic study to know exact mechanism.
PR < RP vs. PR > RP (e.g.):

Long RP - short PR	*Short RP - long PR*
Sinus node reentrant tachycardia	atrial tachycardia with first degree AV block
Atrial tachycardia	typical AVNRT
atypical AVNRT	orthodromic AVRT
AVRT with slowly conducting concealed bypass tract (e.g. Ebstein's)	
PJRT	

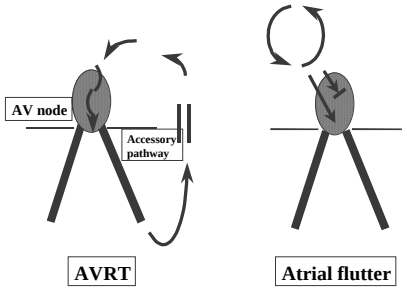
Practical approach (Prystowsky and Klein) - AV node dependent vs. AV node independent tachycardias.

Appendix

AV node dependent tachycardias - SVTs that require conduction through the AV node to maintain the tachycardia.
AVNRT - atrioventricular nodal reentrant tachycardia.

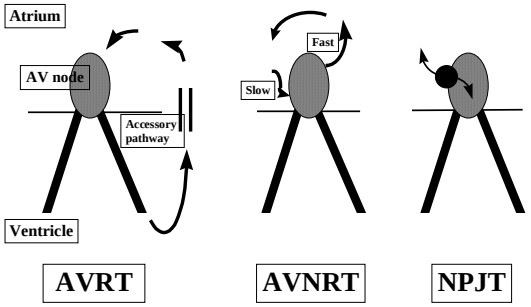
AVRT - atrioventricular reentrant tachycardia (via an accessory pathway).
AVNRT and AVRT - majority of PSVTs.

AV node dependent (junctional) vs.
AV node independent (atrial) mechanism



(Klein and Prystosky)

AV node dependent tachycardias



Reentrant mechanism.
Usually occur in younger patients without structural heart disease.
Congenital substrate (dual AV nodal pathways or accessory pathway).

PSVT and adenosine

Appendix

Nonparoxysmal junctional tachycardia (rate ~ 60 to 110) - enhanced automatic mechanism, due to ischemia or inflammation of junctional tissue. Causes a fall in baseline potential with phase 4 spontaneous depolarization.

Differential:
Digitalis toxicity.
Ischemia.
Myocarditis.
Post cardiac surgery.

AV node independent tachycardias - originate in atrium and atrial rhythm may be maintained despite block in AV node.

Most often occurs in patients with congenital or cardiopulmonary disease - an acquired substrate.

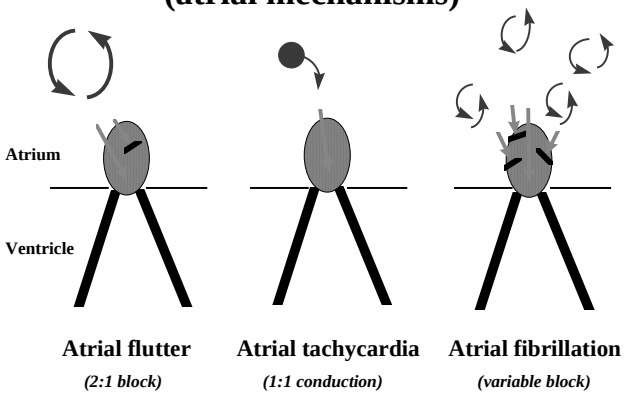
Irregular SVTs - almost always an AV node independent mechanism.

- Atrial fibrillation
- Multifocal atrial tachycardia.
- Atrial tachycardia with variable AV nodal conduction.

Regular SVTs.

- Automatic atrial tachycardia
- Reentrant atrial tachycardia.
- Atrial flutter
- Sino-atrial reentrant tachycardia.

AV node independent tachycardias (atrial mechanisms)



"First approximation to mechanism" (Wathen and Klein)

Patient history - congenital vs. acquired substrate.

P wave axis - superior to inferior, has to be atrial mechanism (AV node independent).

Response to CSM, val salva, or adenosine - diagnostic, often therapeutic.

- Sinus tachycardia - will transiently slow, but then speeds up due to reflex tachycardia.
- Automatic atrial tachycardia - Usually does not alter atrial mechanism (rarely will terminate). May increase level of AV block.
- Reentrant atrial mechanisms - atrial fibrillation, atrial flutter, reentrant atrial tachycardias -does not alter atrial mechanism, may increase AV block and unmask atrial activity.
- AV node dependent PSVT - often abruptly terminates.
- Wide complex tachycardia of unknown mechanism - may terminate and aide diagnosis if of supraventricular origin (see above), or do nothing to rhythm if VT. Effect of these maneuvers is transient, should not have adverse effects.
- Adenosine contraindicated in atrial fibrillation in WPW syndrome - may facilitate rapid conduction via AP into ventricle, may precipitate VF.

Appendix

Acute treatment of AV node dependent tachycardias.

Unstable - synchronized cardioversion.

Vagal maneuvers, CSM, or adenosine - to produce transient AV slowing or block.

Verapamil IV.

Chronic treatment of AV node dependent tachycardias.

Medical therapy (chronic, prn).

digoxin - delayed onset, often overwhelmed by catecholamine stimulation.
Beta blockade
Calcium antagonists (verapamil or diltiazem).

RF catheter ablation.

Highly successful for WPW, AVNRT; minimal morbidity, almost nil mortality.
Often treatment of choice in highly symptomatic individuals, or when medications do not control the arrhythmia or are poorly tolerated.

Treatment of AV node independent SVT - generally requires dual therapy:

Therapy (acute and chronic) targeted at AV node to slow ventricular response
AND
Therapy aimed at stabilizing atrium (Class I or III antiarrhythmic therapy).

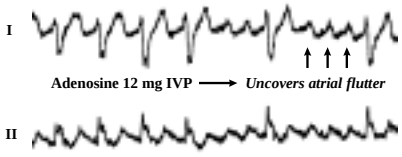
Class IA - slows atrial myocyte conduction and prolongs repolarization (QT interval). Also has some anticholinergic activity that may facilitate AV node conduction. May be problematic with atrial flutter (slows flutter rate, speeds AV node conduction -> may increase AV conduction to 1:1.)

Class IC - marked slowing of atrial and AV node conduction without change of QT interval. Avoid in structural heart disease or CAD due to significant risk of proarrhythmia.

Class III agents - prolong repolarization (and QT interval), extends atrial refractoriness. dl sotalol has additional beta blocker activity (~ 1/4 activity of same dose of propranolol). Amiodarone has Class I - IV activity, though Class III activity predominates.

Atrial slowing	*AV node slowing*	*Atrial and AV node activity*
Quinidine	Digitalis	Flecainide
Procainamide	Beta blockade	Propafenone
Disopyramide	Verapamil	dl-Sotalol
	Diltiazem	Amiodarone

SVT in patient with Epstein's anomaly



Appendix

Bibliography

General

ACLS Handbook 1994 American Heart Association

Akhtar M. *Cardiology Clinics* 1993; 11(1):1-198.

Chou T, Knilans TK. *Electrocardiography in Clinical Practice: adult and pediatric.* WB Saunders, Co 1996.

Davison R. *Electrocardiography in acute care medicine.* Mosby Year Book, Inc., 1995.

Domanovitis H, et al. Adenosine for the management of patients with tachycardias - a new protocol. Europ Heart J 1994:15:589-593

Hardman JG, Limbird LE. *Goodman and Gilman's: The pharmacologic basis of therapeutics (9th edition).* McGraw-Hill, 1996.

Marriott HJL, Boudereau Conover MH. *Advanced Concepts in Arrhythmias*. Mosby 1983

Votey SR, Hoffman JR. *Tachyarrhythmias* Chapter 133. The Clinical Practice of Emergency Medicine, 2nd Ed Edited by Harwood-Nuss et al. Lippincott-Raven Publishers

Wagner GS. *Practical electrocardiography (9th edition).* Williams and Wilkins, 1994.

Wellens HJ, Conover MB. *The ECG in clinical decision making.* WB Saunders, Co., 1992.

Zimetbaum P and Josephson ME. Evaluation of patients with palpitations. NEJM 1998; 338(19):1369-1373.

Supraventricular Tachycardia and Clinical Electrophysiology

Camm AJ, Garratt CJ: Adenosine and supraventricular tachycardia. *NEJM*, 1991:325:1621-1629.

Caterine MR, Yoeger DM, Spencer KT et al. Effect of electrode position and Gel-application technique on predicted transcardic current during transthoracic defibrillation. Ann Emerg Med 1997;29:588-595

Cummins RO, ed.: Essentials of ACLS. *Textbook of Advanced Cardiac Life Support*. AHA. 1994, pp. 1-1 - 1-77.

Ellenbogen KA et al.: Efficacy of intravenous ibutilide for rapid termination of atrial fibrillation and atrial flutter: a dose-response study. *JACC.* 1996;28:130-6.

Furlong R, Farber P, Schrank K, et. al. Intravenous adenosine as first-line prehospital management of narrow complex tachycardias by EMS personnel without direct physician control. Am J Emerg Med 1995; 13:383-388.

Gilman JK, Naccarelli GV: Sudden cardiac death. In O'Rourke RA (ed): *Current Problems in Cardiology*, St Louis, Mosby Year Book, Inc., 1992, XVII, pp 695-778.

Haines DE, DiMarco JP: Current therapy for supraventricular tachycardia. In O'Rourke RA (ed): *Current Problems in Cardiology*, St Louis, Mosby Year Book, Inc., 1992, XVII, pp 411-477.

Huagui GL, Morillo CA, Zardini M, et al. Effect of Adenosine Triphosphate on Antidromic Tachycardia. J Am Coll Cardiol 1994:24:728-31

Imrie JR, Yee R, Klein GJ, et al. Incidence and clinical significance of ST segment depression in supraventricular tachycardia. Can J Cardiol 1990;6(8):323-326

McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest by verapamil in patients with WPW syndrome. *Ann Intern Med*. 1986;104:791-794.

Nelsen SD, Kou WH, Annesley T, et al Significance of ST segment depression during paroxysmal supraventricular tachycardia. J Am Coll Cardiol 1988;12:383-7

NHLBI AFFIRM Investigators: Atrial Fibrillation Follow-up investigation of rhythm management - the AFFIRM study design. *Am J of Cardiology*, 1997;79:1198-1202.

Bibliography

NHLBI Working Group on Atrial Fibrillation: Atrial Fibrillation: Current understandings and research imperatives. *JACC* 1993;22:1830-4.

Obrien JF, Tremml GP, and Falk J. Intravenous calcium chloride in the conversion of paroxysmal supraventricular tachycardia to normal sinus rhythm. Am J Emerg Med 1996; 14:50-52.

Oren JW, Beckman KJ, McClelland JH, Wang X, Lazzara R, and Jackman WM: A functional approach to the preexcitation syndromes. *Cardiology Clinics*, 1993;11:121-150.

Prystowsky EN, et al., Management of patients with atrial fibrillation. *Circulation.* 1996;93:1262-1277.

Prystowsky EN, Klein GJ, *Cardiac Arrhythmias - An integrated approach for the clinician*. New

York, MrGraw-Hill, Inc., 1994.

Stewart RB, Bardy GH, Green HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med*. 1986;104:791-794.

Takayanagi K, Hoshi H, Shimizu M, et al Pronounced ST-segment depression during paroxysmal supraventricular tachycardia. Jpn Heart J 34:269-278 1993

Wathen MS, Klein GJ, Yee R, and Natale A: Classification and terminology of supraventricular tachycardia: diagnosis and management of the atrial tachycardias. *Cardiology Clinics*, 1993;11:109-120.

Atrial Fibrillation

Atrial Fibrillation Investigators. The efficacy aspirin in patients with atrial fibrillation: analysis of pooled data from three randomized trials. Arch Int Med 1997; 157:1237-1240.

Capucci A, Lenzi T, Boriani G, et al. Effectiveness of loading oral flecainide for converting recent onset atrial fibrillation to sinus rhythm in patients without organic heart disease or with only systemic hypertension. Am J Cardiol 1992:70:69-72

Donovan KD, Dobb GJ, Coombs LJ, et al. Reversion of recent-onset atrial fibrillation to sinus rhythm by intravenous flecainide. Am J Cardiol 1991:67:137-141

Donovan KD, Power BM, Hockings BEF, et al. Intravenous flecainide verus amiodarone for recent-onset atrial fibrillation. Am J Cardiol 1995:75:693-697

Figa FH, Gow RM, Hamilton RM, et al. Clinical efficacy and safety of intravenous amiodarone in infants and children. An J Cardiol 1994:74:573-577

Fatkin. Detection of thrombus by transesophageal echocardiography. JACC 1994; 23:307.

Gorelick PB, et. al. Prevention of a first stroke. A review of guidelines and a multidisciplinary consensus statement from the national stroke association. JAMA 1999; 281(12):1112-1120.

Herbert ME, Votey SR. New Treatments for Atrial Fibrillation. West Med 1996 Jan; 164(11):63

Madrid AH, Moro C, Marin-Huerta E, et al. Comparison of flecainide and procainamide in cardioversion of atrial fibrillation. Europ Heart J 1993; 14:1127-1131.

Mulcahy B, Coates WC, Henneman PL, et. al. New onset atrial fibrillation: whwen is admission medically justified. Acad Emerg Med 1996; 3:114-119.

Michael JA, Stiell IG, Agarwal S, et. al. Cardioversion of paroxysmal atrial fibrillation in the emergency department. Annals EM; 33(4):379-387.

Prince LR, et. al. Transesophageal electrocardiographic guidance in patients with atrial fibrillation. Am Heart J 1995; 129:1024-1029.

Bibliography

Cardioversion and Pacing

Falk RH, Ngal ST. External cardiac pacing: Influence of electrode placement on pacing threshold. Crit care Med 1986;14:931-932

Kerber RE, Jensen SR, Grayzel J, et al Elective cardioversion : influence of paddle-electrode location and size on success rates and emergency requirements. N Eng J Med 1981;305:656-662

Olson CM, Jastremski MS, Smith RW, et al External cardiac pacing for out of hospital bradyasystolic arrest. Am J Emerg Med 1985;3:129-131

Wolff-Parkinson-White Syndrome

Afridi I et al. Termination of supraventricular tachycardia with intravenous adenosine in a pregnant women with Wolf-Parkinson-White syndrome. Obstet Gynecol 1992:80:481-3

Atie J, Brugada B, Brugada J, et al. Clinical and electrophysiologic characteristics of patients with antidromic circus movement tachycardia in the Wolf-Parkinson-White syndrome. Am J Cardiol 1990:1082-1091

Garrat CJ, Griffith MJ, O'Nunain S, et al. Effects of intravenous adenosine on anterograde refractoriness of accessory atrioventricular connections. Circulation 1991:84:1962-1968

McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest in patients with Wolf-Parkinson-White Syndrome. Ann Int Med 1986:104:791-794

Mulla N, Karpawich PP. Ventricular fibrillation following adenosine therapy for supraventricular tachycardia in a neonate with concealed WPW syndrome treated with digoxin. *Ped Emerg Care* 1995; 11(4):238-9.

Perrot B, et al. Effect of adenosine triphosphate on the accessory pathways. Eurp Heart J 1984:5:382-393Rankin AC, et al. Acceleration of ventricular response to atrial flutter after intravenous adenosine. Br Heart J 1993:69:263-265

Timmermans C, Smeets, J, Rodriguez, L, et al. Aborted sudden death in the Wolf-Parkinson-White syndrome. Am J Cardiol 1995:76:492-494

Tsuchioka Y, Karakawa S, Nagata K, et al. The role of the accessory pathway in the onset of atrial fibrillation in the Wolf-Parkinson-White syndrome. Jpn Circ 1994:58:95-99

Wide Complex Tachycardia

Akhtar M, Sherasa M, Jazayeri M, et. al. Wide QRS tachycardia: reappraisal of a common problem. *Ann Intern Med* 1988; 109:905-912.

Brugada P, Brugada J, Mont L, et al. A new approach to the differential diagnosis of a regulat tachycardia with a wide QRS complex. *Circulation* 1991; 83(5):1649-1659.

Desai AD, Chun S, Sung R. The role of intravenous amiodarone in the management of cardiac dysrhythmias. *Ann Intern Med* 1997; 127(4):294-303.

Griffith MJ, et al. Adenosine in the diagnosis of broad complex tachycardia. Lancet March1988:672-675

Helmy I, Herre JM, Gee G, et al. Use of intravenous amiodarone for the emergency treatment of life threatening ventricular arrhythmias. J Am Coll Cardiol 1988:12:1015-22

Herbert ME, Votey SR. Adenosine in Wide Complex Tachycardia. Ann Emerg Med 1997 Jan 1997;29:172-174

Herbert ME, Votey SR, Morgan, MT, Cameron P, Dziukas LJ. Failure to Agree on the ECG Diagnosis of VT. Ann Emerg Med Jan 1996; 27:35-38

Hohnloser SH, Meinertz T, Dammbacher T, et al. Electrocardiographic and antiarrhythmic effects of intravenous amiodarone: Results of a prospective, placebo-controlled study. Am Heart J 1991:121:89-95

Illkhanipour K, Berrol R, Yealy DM. Therapeutic and diagnostic efficacy of adenosine in wide-complex tachycardia. Ann Emerg Med August 1993:22:1360-1364

Isner JM, Roberts WC, Heymsfield SB, et al. Anorexia nervosa and sudden death. *Annals of Internal Medicine* 1985; 102:49-52.

Bibliography

Lee KL, Tai Y-T: Adenosine in wide complex tachycardia: Potential Pitfalls in Diagnostic Value. Ann Emerg Med October 1994:24:741-747

Levine JH, Massumi A, Scheinman MM, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. J Am Coll Cardiol 1996:27:67-75

Lopez JA, Lufschanowski R, Massumi A. Transesophageal electrocardiography and adensoine in the diagnosis of wide complex tachycardia. Tex Heart J 1994:21:130-131

Middlekauff HR, Stevenson WG, Saxon LA. Amiodarone and torsades de pointes in patients with advanced heart failure. Am J Cardiol 1995:76:499-502

Moss AJ, Schwartz PJ, Crampton RS, et al. The long QT syndrome: a prospective international study. *Circulation* 1985; 71(1):17-21.

Ochi Rp, Golderberg IF, Almquist A, at al. Intravenous amiodarone for the rapid treatment of life threatening ventricular arrhythmias in critically ill patients with coronary artery disease. Am j Cardiol 1989:64:599-603

Rankin AC, Oldroyd KG, Chong E, et al. Value and limitations of adenosine in the diagnosis and treatment of narrow and broad complex tachycardia. Br Heart J 1989:62:195-203

Rinne C, Sharma AD, Klien GJ, et al. Comparative effects of adenosine triphosphate on the acessoy pathway and atrioventricular nodal conduction. Am Heart J 1988:115:1042

Schutzenberger W, Leisch F, Kerschner K, et al. Clinical efficacy of intravenous amiodarone in the short term treatment of recurrent sustained ventricular tachycardia and ventricular fibrillation. Br Heart J 1989;62:367-71

Sharama AD, Klein GJ, Yee RY. Intravenous adenosine triphosphate during wide QRS complex tachycardia: Safety, therapeutic efficacy and diagnostic utility. Am J Med April 1990:88:337-343

Slade AK, Garratt CJ. Proarrhythmic effect of adenosine in a patient with atrial flutter. Br Heart J 1993:70:91-92

Stewart RB, Brady GH, Greene HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med* 1986;104:766-771.

Wilber DJ, Baerman J, Olshansky B, et al. Adenosine-sensitive ventricular tachycardia: Clinical characteristics and response to catheter ablation. Circulation 1993:87:126-134

Williams ML, et al. Intravenous amiodarone during prolonged resuscitation from cardiac arrest. Ann Intern Med 1989:110:839

Wellens HJJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *The American Journal of Medicine* 1978; 64:27-32.

Bibliography