

Arrhythmias and their Acute Management

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Shorofsky's Rules #1

- If it is fast and the patient is unstable:
SHOCK
 - Please make sure the patient is either unconscious or sedated
- If it is slow and the patient is symptomatic :
PACE
- Anything else you have time to think and call EP

Rule #2

- All wide complex tachycardias are VT
- If you think it is SVT with aberration, you are wrong (>95% of time)
- Hemodynamically stable presentation does not alter the above rule

Why are we afraid of electricity?



Electricity is your friend

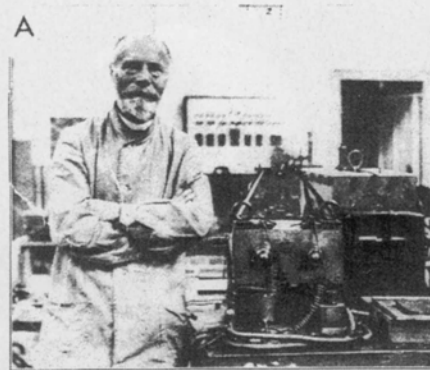
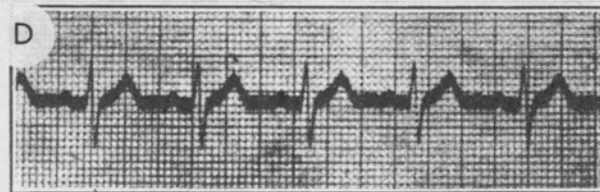
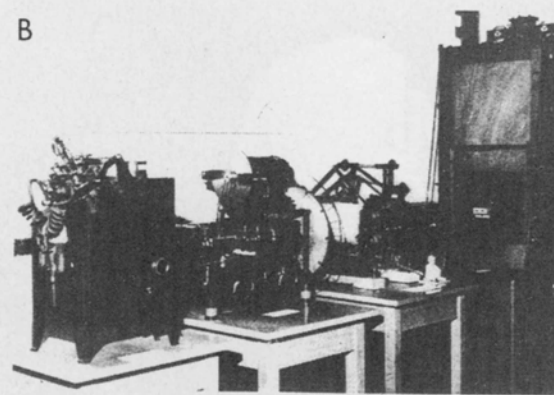


FIG. 1.—Professor W. Einthoven in his laboratory at Leiden with the original "string" galvanometer.



Initial Tachycardia Evaluation

- Patient hemodynamics
- Get a 12 lead if stable
- Determine whether narrow or wide QRS complex (you must look at more than one lead)
 - If it is wide - worry
 - If is is narrow – often can treat and send home

Mechanisms of Arrhythmias

- **Reentry**

- AVNRT, AVRT, Atrial Flutter, atrial tachycardia, most VT, sinus node reentry

- **Enhanced automaticity**

- Sinus tachycardia, junctional rhythms, idioventricular rhythms

- **Triggered Arrhythmias**

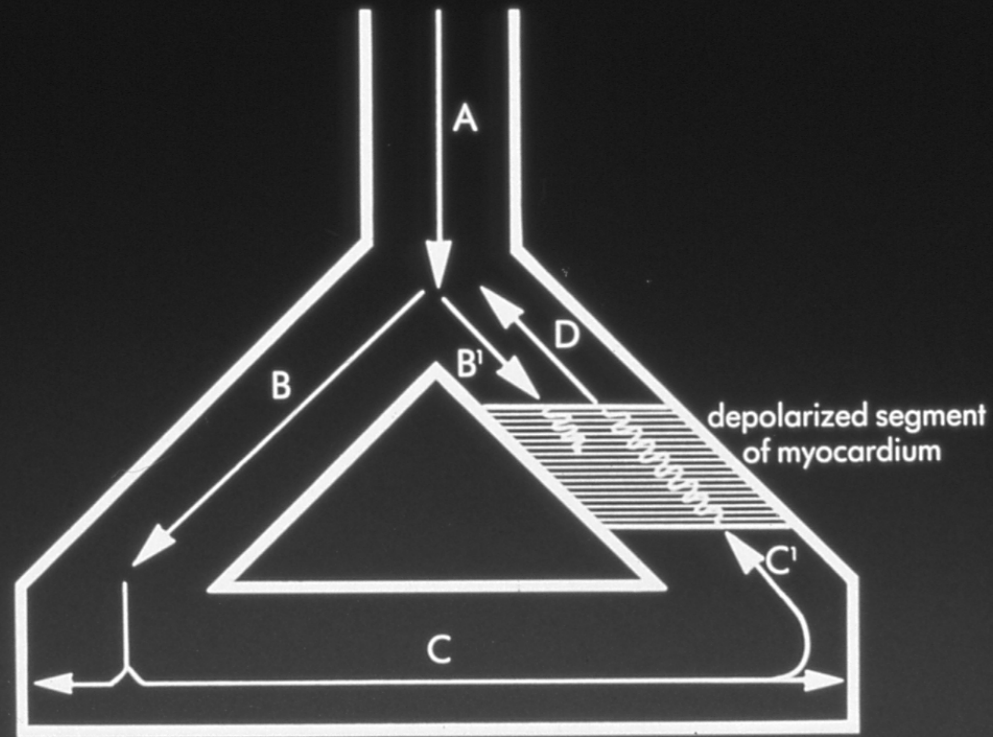
- Polymorphic VT/Torsade de Pointes- EAD
- Digoxin toxicity - DAD

Narrow Complex Tachycardias

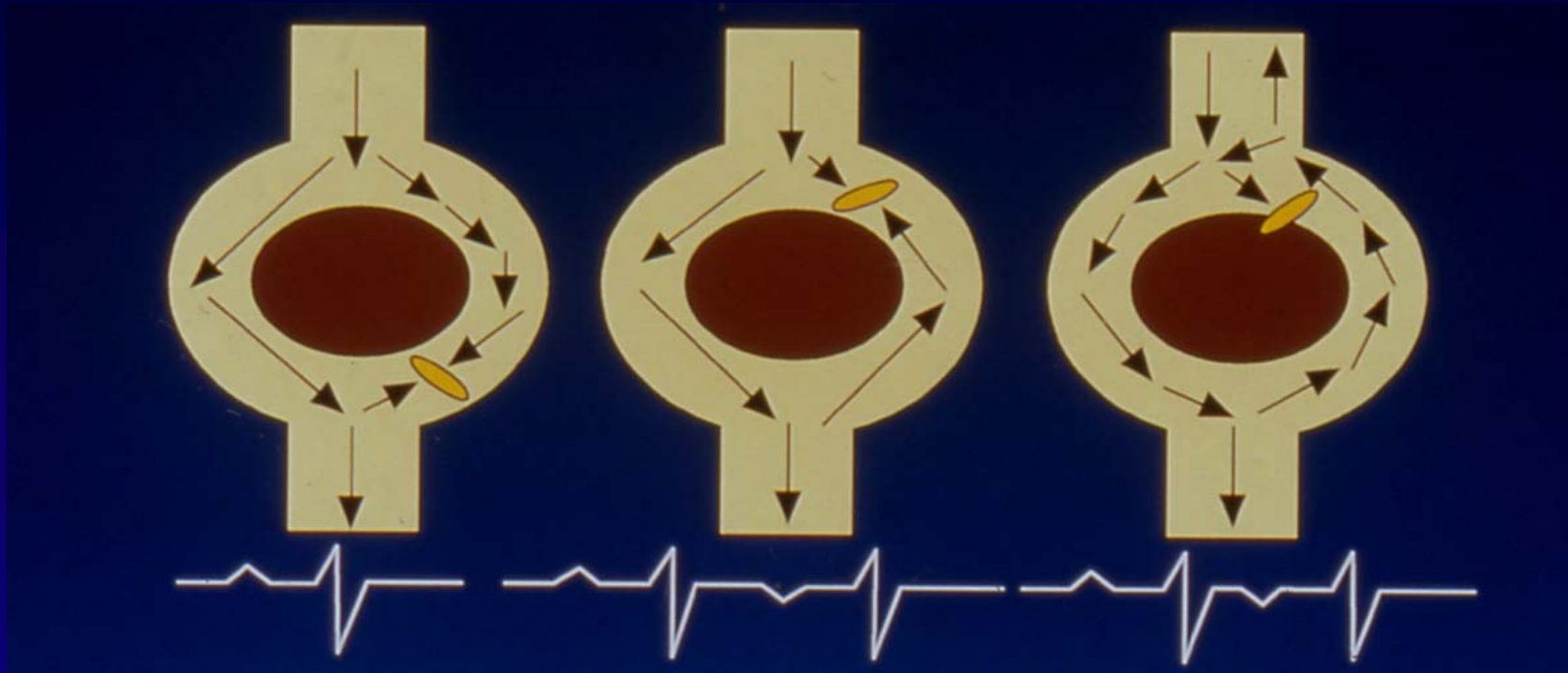
Differential Diagnosis

- Sinus Node
 - appropriate sinus tachycardia
 - inappropriate sinus tachycardia
 - sinus node reentry
- Atrial Arrhythmias
 - atrial tachycardia
 - atrial flutter
 - atrial fibrillation
- AV ring
 - junctional tachycardia
 - AV nodal reentry
 - AVRT (bypass tract, WPW)

Reentry

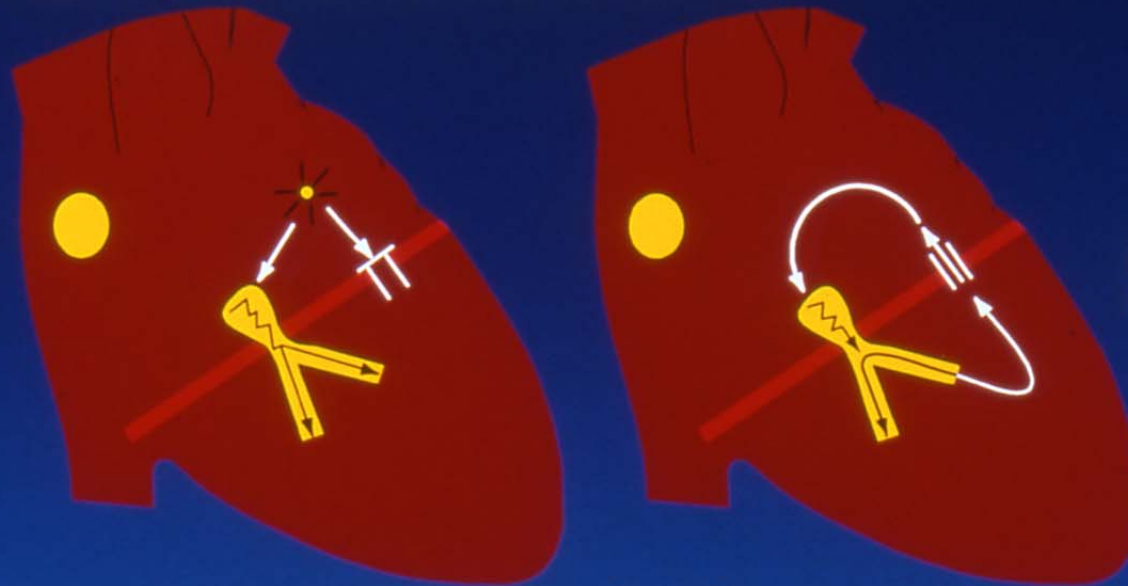


AV Nodal Reentry



WPW

A **tachycardia** initiates via a circus movement or bypass-tract-mediated macroreentry.

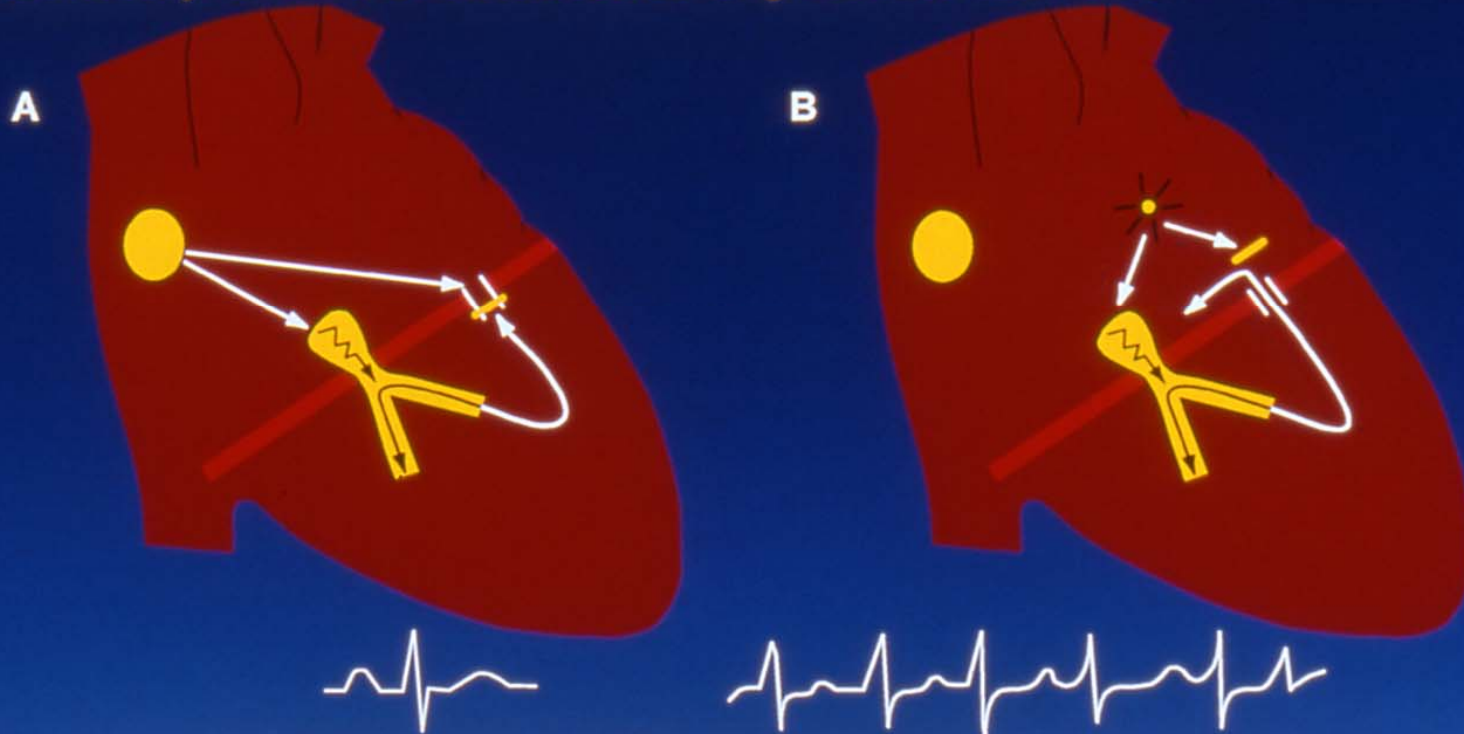


Normal P-R interval
and no delta wave



Concealed Bypass Tract

Concealed Pathway (no “delta wave” and not capable of antegrade conduction) occurs in about 30% of patients with SVT.



Narrow Complex Tachycardias

Diagnosis

- Primarily based on ECG
- Initiations and terminations are very useful
- Carotid sinus massage or Valsalva may terminate or block one beat
- Adenosine is often useful

Narrow Complex Tachycardias

Treatment

- **Depends on the arrhythmia**
 - Sinus tachycardia- treat the underlying cause
 - Reentrant arrhythmias
 - Adenosine or carotid massage may terminate
 - Afib/atrial flutter
 - **Anticoagulate**
 - Control ventricular response rate
 - consider timing of cardioversion

PSVT - Acute Treatment

- Vagal maneuvers
- Adenosine
- Cardioversion

Adenosine

- Endogenous nucleoside
- Metabolized in every cell
- $T_{1/2} = 0.6-1.5$ sec
- Reversed by methylxanthines
- Potentiated by dipyrimidole
- Must be administered by a rapid IV bolus into a large bore IV followed by a large flush

Adenosine (cont.)

- Hemodynamics
 - Slight hypertension followed by hypotension due to vasodilatation
 - AV block and possible sinus arrest
 - Very short-lived (<10 sec)
- Other Effects
 - Flushing, chest discomfort, bronchospasm

Adenosine Indications

- Termination of reentrant PSVT utilizing the AV node
- Diagnosis of SVT
- Diagnosis of wide complex tachycardia if SVT is suspected and the patient is hemodynamically stable

Narrow Complex Tachycardias

Medication

- **Rate control**
 - Beta-blockers
 - Ca channel blockers
 - Digoxin - only as last resort
- **Long-term management**
 - Beta-blockers or Ca channel blocker
 - once a day drug
 - Ablation

PSVT - Drugs

- Advantages
 - high efficacy
 - low initial cost
 - not invasive
- Disadvantages
 - High long term cost
 - palliative
 - adverse effects

PSVT- Treatment

- If recurrent arrhythmia, simple once a day drug treatment with a calcium channel blocker or beta-blocker
- If fail above or patient does not want to take drugs, RF ablation prior to using an anti-arrhythmic medication

Atrial Flutter - Drugs

- Type 1 and Type 3 anti-arrhythmics have been used with limited success
- Disadvantages
 - anti-arrhythmics often have pro-arrhythmic effects
 - AV nodal blocking agents often have to be given at large dosages to control the ventricular rate. When the patient converts, they can be severely bradycardic.
 - patient must remain anticoagulated if atrial flutter is not eliminated

Atrial Flutter - Ablation

- First reported in 1992
- Create a complete line of bi-directional block between the tricuspid annulus and the IVC
- High success rate
- Does not eliminate atrial fibrillation if the patient is predisposed to this arrhythmia

Atrial Fibrillation

- The most common arrhythmia
 - Prevalence
 - 1-3% of patients in their 60s
 - 5-7% of patients in their 70s
 - 10% of patients in their 80s
- Most common risk for stroke (rr=5) and patients can be stratified according to their risk for stroke

AF - Overall Strategy

- Rate control
 - relieve symptoms related to rapid ventricular rate
 - does not eliminate need for anticoagulation
- Rhythm control
 - anti-arrhythmic drugs may have more morbidity than the arrhythmia
 - ablation techniques are becoming very useful
 - surgical techniques are improving
- Anticoagulation

Wide Complex Tachycardia

- Hemodynamically unstable - Shock, shock
- If stable - get a 12 lead
- Medications
 - Amiodarone
 - 150mg bolus, 1mg/min x 6 hours, then 0.5mg/min, a second bolus can be given
 - Procainamide
 - not used much but is useful if have both VT and SVT in the same patient or acutely terminating VT

Wide Complex Tachycardia

Medication (cont.)

- Lidocaine
 - Give 1mg/kg bolus (about 100mg) and hang drip. Give 0.5mg/kg bolus 10 min later
- Magnesium
 - very useful for polymorphic VT , little down-side; 1-5 grams IV
- Beta-blockers
 - Useful for VT storm

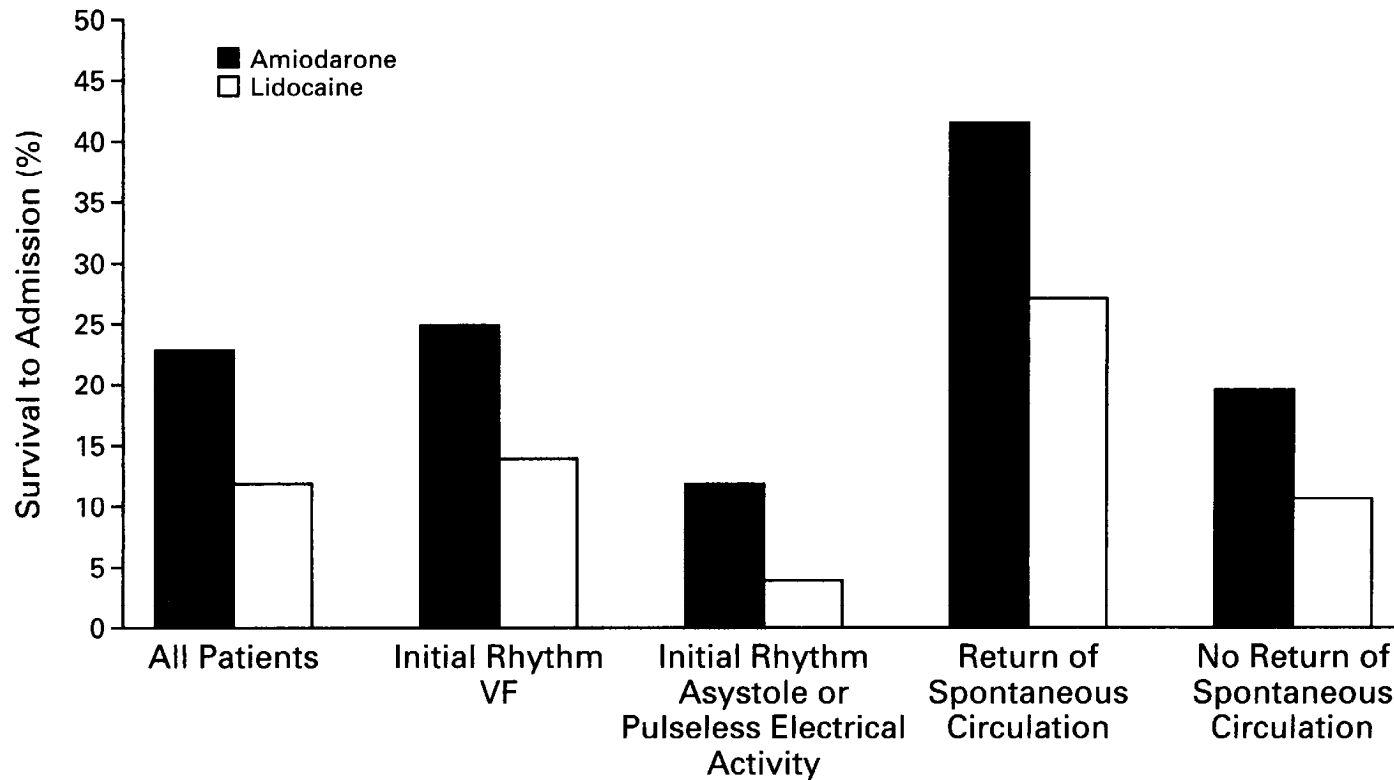
Amiodarone

- Originally designed as an anti-anginal
- Type I, II, III, IV properties
- $T_{1/2} = 10$ to 107 days (mean 52)
- Active metabolites
- Toxicity
 - Bradycardia, tachy-arrhythmias, increases DFT
 - Pneumonitis- 1-5%, decrease DLCO, ARDS very rare
 - Hypo- or hyperthyroid, Transaminitis, peripheral neuropathy

IV Amiodarone

- Indications – life-threatening ventricular arrhythmias
- Given as 150 mg bolus followed by and infusion of 1 mg/min for 6 hours then 0.5 mg/min.
- Repeat boluses can be given
- Hypotension – due to the detergent (TWEEN)

Amiodarone vs Lidocaine



No. SURVIVING/TOTAL No.

Amiodarone 41/80

Lidocaine 20/167

35/141

19/134

4/34

1/27

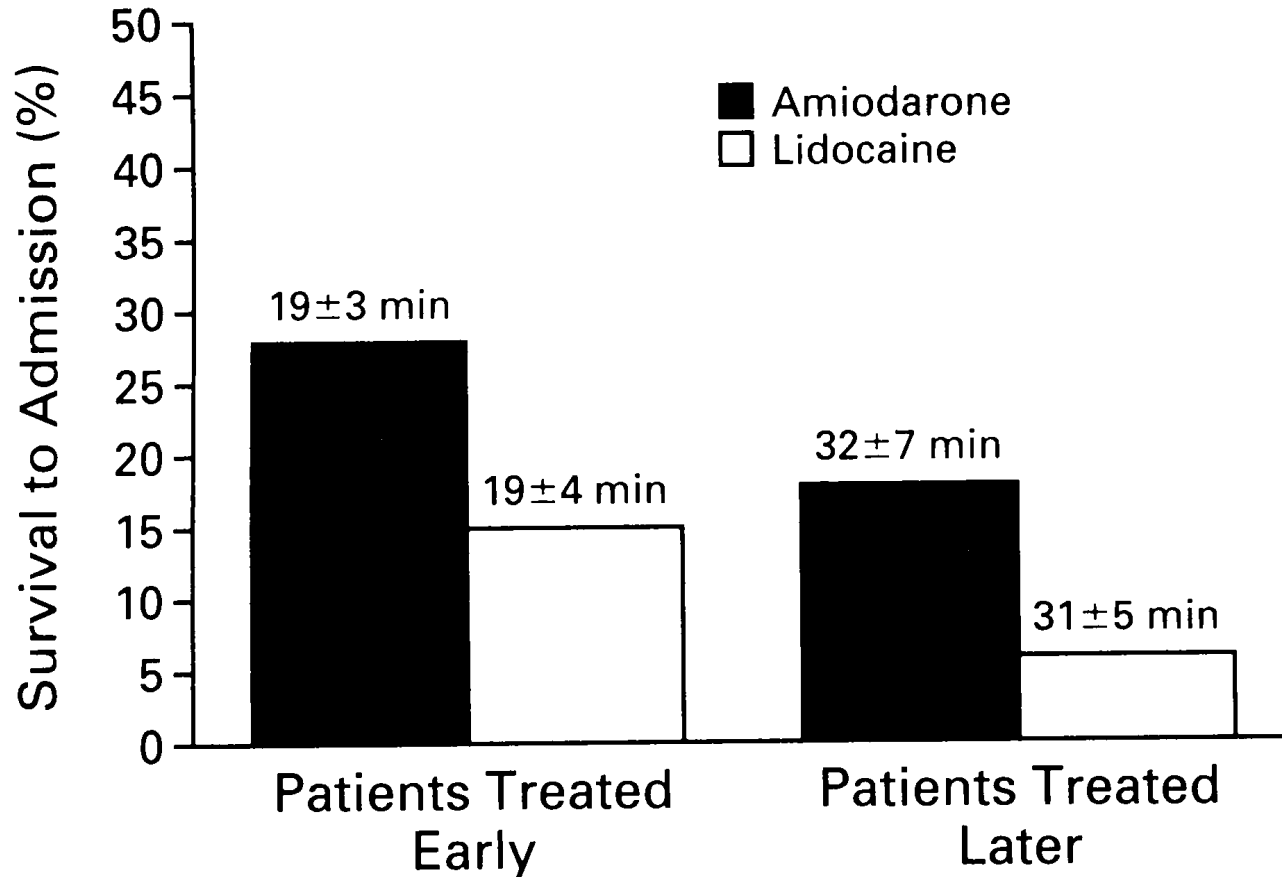
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3/11

31/156

17/156

Amiodarone vs Lidocaine



Amiodarone – Drug Interactions

- Digoxin – increases bioavailability
- Quinidine – increases concentrations
- Warfarin – increases effectiveness
- Beta-blockers – worsens bradycardias
- Ca channel blockers – worsens bradycardias and hypotension
- Dilantin – increases concentration

Wide Complex Tachycardia Chronic Treatment

- If no structural heart disease
 - Ablation often possible
- If structural heart disease or ablation not possible
 - Implantable cardioverter defibrillator (ICD)
 - Often need adjuvant drug therapy or ablation in addition

Modified Vaughan Williams

Class	Available	Investigational	Mechanisms
Ia	quinidine, procainamide disopyramide imipramine	ajmaline	decrease gNa decrease gK prolongs APD
Ib	lidocaine tocainide dilantin, morizicine mexilitine	aprinidine	decrease gNa increase gK ? shortens APD
Ic	flecainide propafenone		decrease gNa APD unaffected

Modified Vaughan Williams (cont.)

Class	Available	Investigational	Mechanisms
II	Beta-blockers		Beta-adrenergic blockade
III	Amiodarone Sotalol dofetilide	NAPA	Block gK Prolong APD
IV	Ca channel blockers		
V	Digoxin		

Sicilian Gambit

DRUG	CHANNELS						RECEPTORS				PUMPS
	Na			Ca	K	I _r	α	β	M ₂	P	Na/K ATPase
	Fast	Med	Slow								
Lidocaine	●										
Mexiletine	●										
Tocainide	●										
Moricizine	I										
Procainamide		A			●						
Disopyramide		A			●				●		
Quinidine		A			●		●		●		
Propafenone		A						●			
Flecainide			A		●						
Encainide			A								
Bepiridil	●				●	●					
Verapamil	●				●		●				
Diltiazem					●						
Bretium					●		○	○			
Sotalol					●			●			
Amiodarone	●				●	●	●	●			
Alinidine					●	●					
Nadolol								●			
Propranolol	●							●			
Atropine									●		
Adenosine										○	
Digoxin											●

Relative blocking potency

○ Low ● Moderate ● High

○ = Agonist

● = Agonist/Antag.

A = Activated state blocker

I = Inactivated state blocker

Arrhythmias amenable to Ablation

- SNRT
- Inappropriate sinus tachycardia
- Atrial tachycardias
- Atrial flutter
- AF – paroxysmal or persistent; or fast ventricular response rates
- AVNRT
- AVRT- bypass tracts
- VTs - in normal and diseased hearts

When to call EP

- EP is always available – 8-7801 or 8-6056
- Wide complex tachycardias
- Sustained supraventricular tachycardias
- Symptomatic bradycardias
- NSVT in patients with ischemic heart disease
- EF <30 and ischemic heart disease or EF <35 and non-ischemic cardiomyopathy
- Syncope (esp. with poor pumps)
- Afib
- Atrial flutter

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Rule #2

- All wide complex tachycardias are VT
- If you think it is SVT with aberration, you are wrong and it is VT (>95% of time)
- If you treat a wide complex tachycardia as an SVT, you may kill the patient
- Hemodynamically stable presentation does not alter the above rule

AF - Rate Control

- Drugs
 - calcium channel blockers
 - beta-blockers
 - digoxin
- Ablation
 - AV node ablation and placement of a permanent pacemaker - effective for symptom relief but not for decreasing mortality

AF - Rhythm Control

- Normal LV function -
 - Type 1 anti-arrhythmics - flecanide, propafenone, quinidine, procainamide
 - Type 3 anti-arrhythmics - sotalol, amiodarone
- Decreased LV function
 - amiodarone

AF - Rhythm Control

- Surgical
 - maze procedure
 - valve replacement with limited maze procedure
- Ablation
 - maze-like procedure
 - limited ablation: RA only, or in pulmonary vein for focal atrial fibrillation

Classification

