



Anti-arrhythmic therapy: Beyond the classes (I-IV)

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Owner:
 Patient: CHASSIE
 Breed: GERMAN WIRE HAIR
 Comment: EKG

Date: 5/17/2004
 Birth: 03/27/92
 Code:

Time: 11:37:49
 Weight (lb): 51.6
 Sex (M/F/S): F

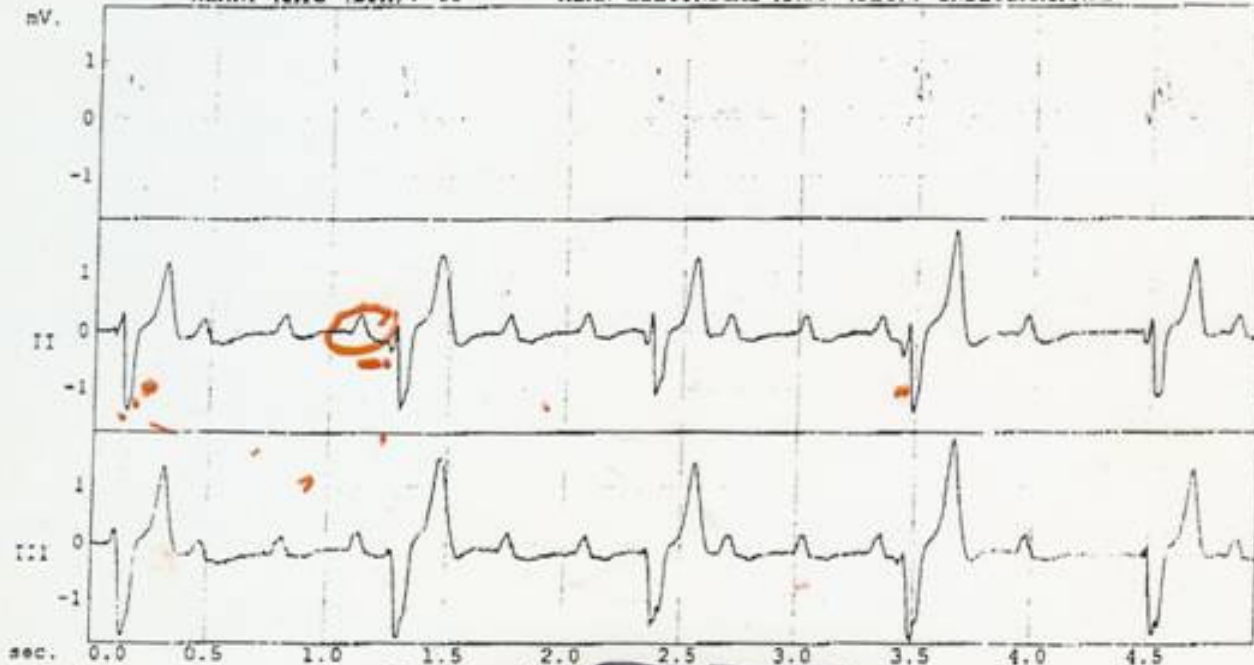
DIAGNOSES INDICATED

PVC's FOUND
 ACCELERATED IDIOVENTRICULAR RHYTHM
 MULTIFOCAL PVC's

	NORMALS	AVG	PARAMETER CHART													
			1	2	3	4	5	6	7	8	9	10				
P AMP	0.0-0.4mv			0.32	0.31	0.26										
P DUR	30-40ms			73	72	70										
P-R INT	60-130ms			153	277	110										
R AMP	0.5-3.0mv		0.32	0.10	0.32	0.13	1.75	0.20								
QRS DUR	20-60ms		103	102	93	107	110	105								
Q-T INT	150-250ms		285	287	292	232	390	288								
Q AMP	0.0-0.5mv		0.07	0.32	0.23	0.44		0.24								
S AMP	0.0-0.35mv		1.38	1.32	1.12	1.41	0.25	1.14								
T AMP	<25% R AMPmv		1.18	1.32	1.27	0.39	0.26	1.25								
R-R INT	ms	877		1167	1082	1105	195	838								

HEART RATE (BPM): 68

MEAN ELECTRICAL AXIS (DEG): INDETERMINATE



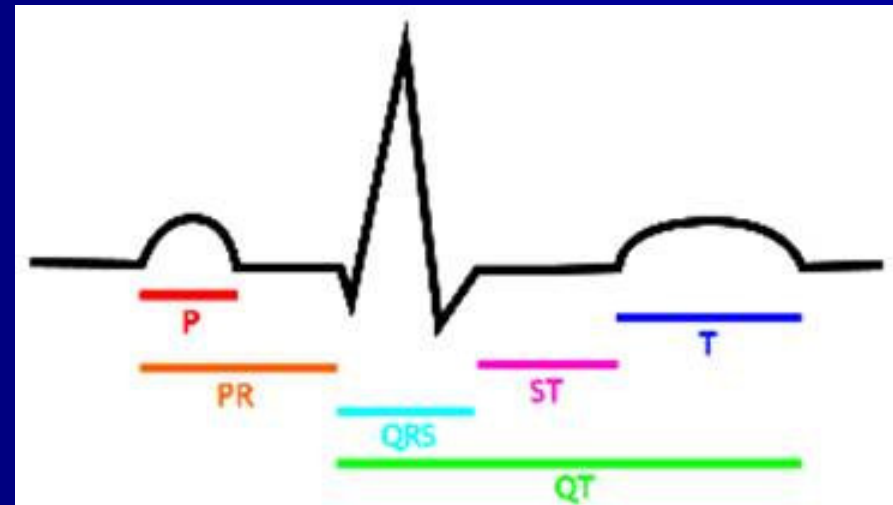
Approach to the ECG

- **Note the Basics**
- **Calculate the Heart RATE**
- **Regular or Irregular**
 - regularly irregular – Pattern
 - irregularly irregular – No pattern
- **Identify the P, QRS, and T waves**
- **P-wave for every QRS and QRS for every P-wave?**
 - Are they related?
- **Any early or late complexes?**
 - Supraventricular or Ventricular?
- **Diagnostic ECG**
 - Calculate mean electrical axis (MEA)
 - Measure waveforms and intervals
 - Measure for chamber enlargement
- **Proper diagnosis**
- **Proper treatment**

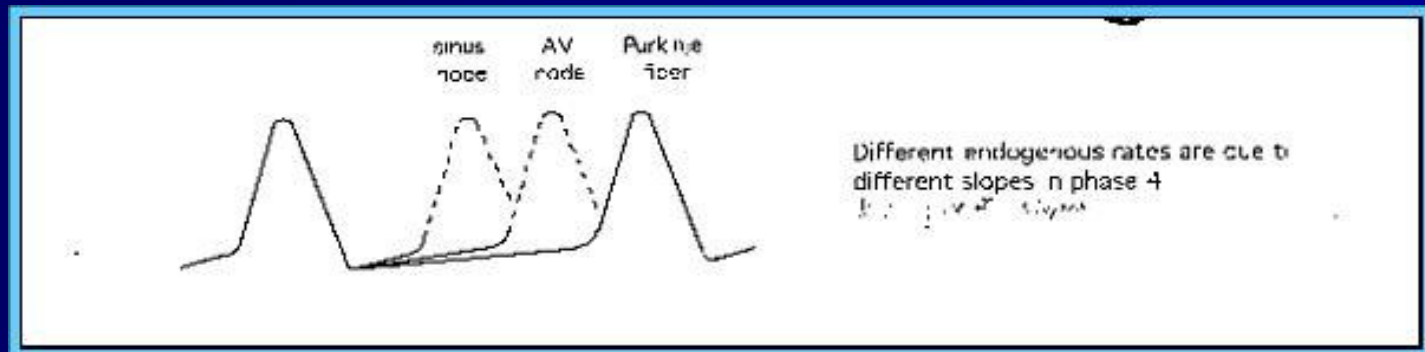
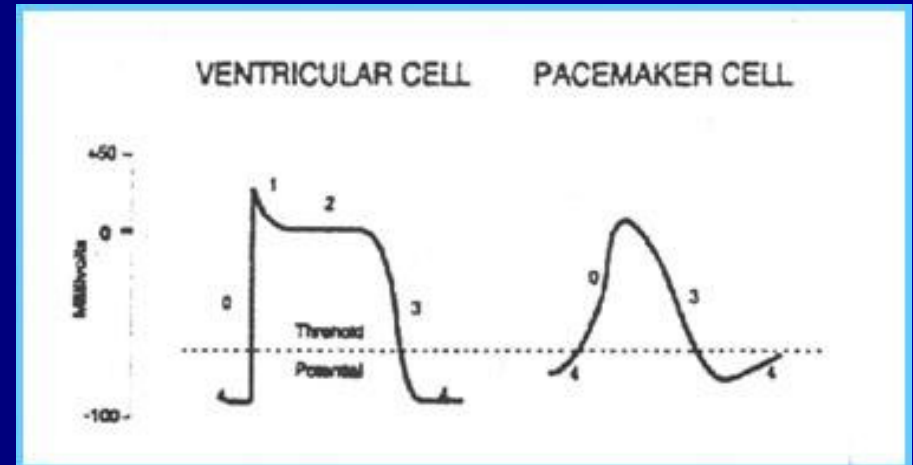
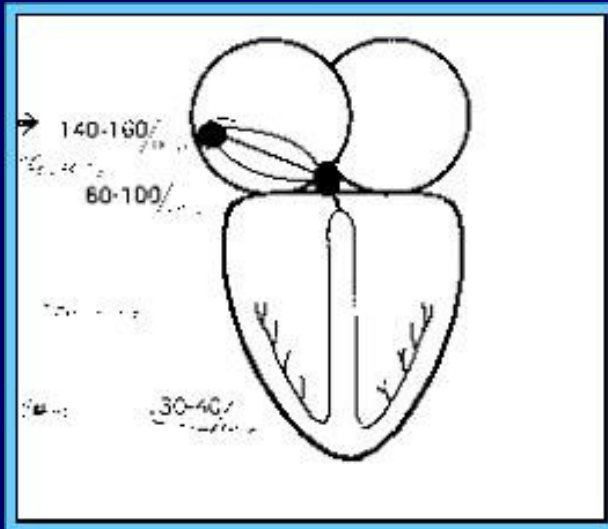
Heart rhythms



Normal Heart Cycle



Normal Pacemakers



Treatment of Arrhythmias

- Assess patient
 - hemodynamic compromise
- Underlying causes
 - trauma, electrolytes, extracardiac/ cardiac disease
 - drugs (α -2 agonists, Digoxin, antiarrhythmics)
- Understanding of arrhythmogenesis
 - automaticity, triggered phenomena, re-entry circuits
- Understanding of antiarrhythmic drugs
 - classification, pharmacokinetics/dynamics, side-effects

Anti-Arrhythmic Drugs

- Modified Vaughan-Williams Classification
 - Predominant electrophysiologic action
 - Class I - Sodium channel blockers
 - Class II – Beta adrenergic blockers
 - Class III- Potassium channel blockers
 - Class IV- Calcium channel blockers
- Others used to control the heart

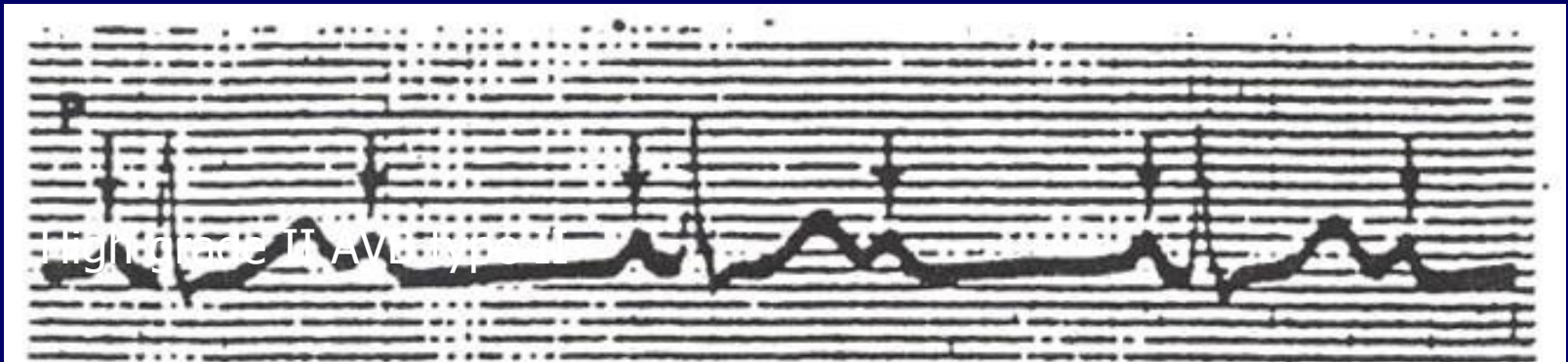
Ectopic Beats

- Originate **away** from the SINUS NODE
 - May have fast or slow rates
 - **Junctional (supraventricular)**
 - Typically normal, narrow QRS
 - **Ventricular**
 - Typically wide and bizarre QRS
- **Early – Premature**
 - Prior to the regular sinus beat
 - Compromise ventricular filling (Esp. High HR)
- **Late – Escape Beats**
 - Occur secondary to a long sinus pause or block
 - Subsidiary pacemaker – AUTOMATICITY
 - **LIFE SAVING RHYTHM !!!**

Selected Bradycardias

- A number of incidental findings
 - Most secondary to increased vagal tone
 - Electrolyte, endocrine abnormalities or drugs toxicities may be implicated
- More advanced disease
 - Associated with disease or destruction of SA or AV nodes
- Result from
 - Disorders of impulse conduction
 - Disorders of Impulse formation
 - Decreased Automaticity

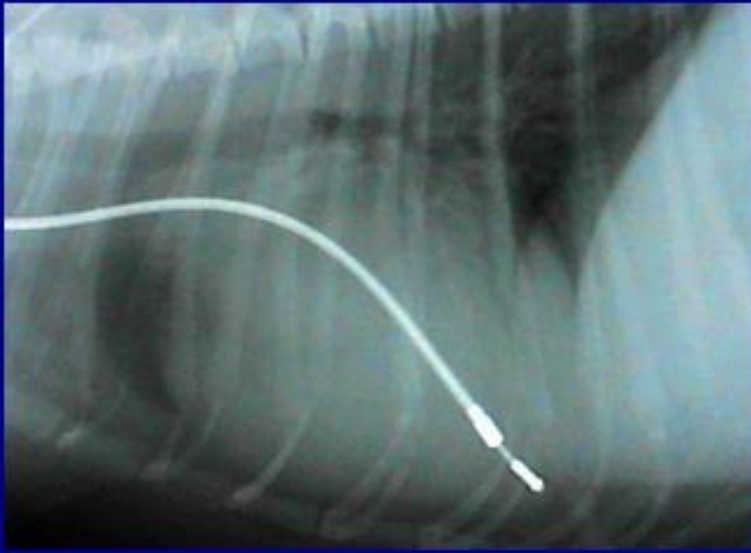
AV Node Diseases



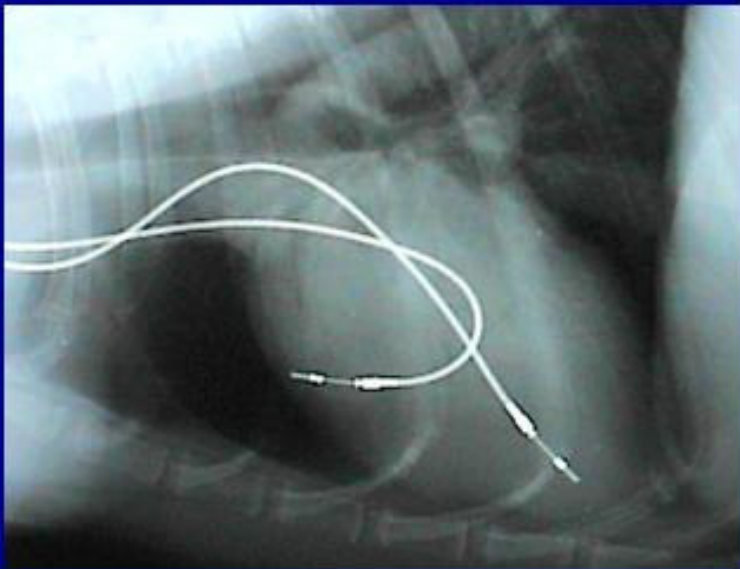
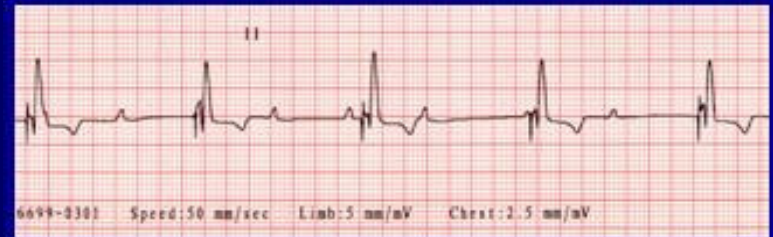
Escape Ectopic Beats

- LIFE SAVING RHYTHM
 - Don't treat with anti-arrhythmics
 - Typically slow rates
 - Junctional
 - Narrow QRS complex
 - Ventricular
 - Wide and Bizarre





Single Chamber System-VVI



Dual Chamber System-DDD

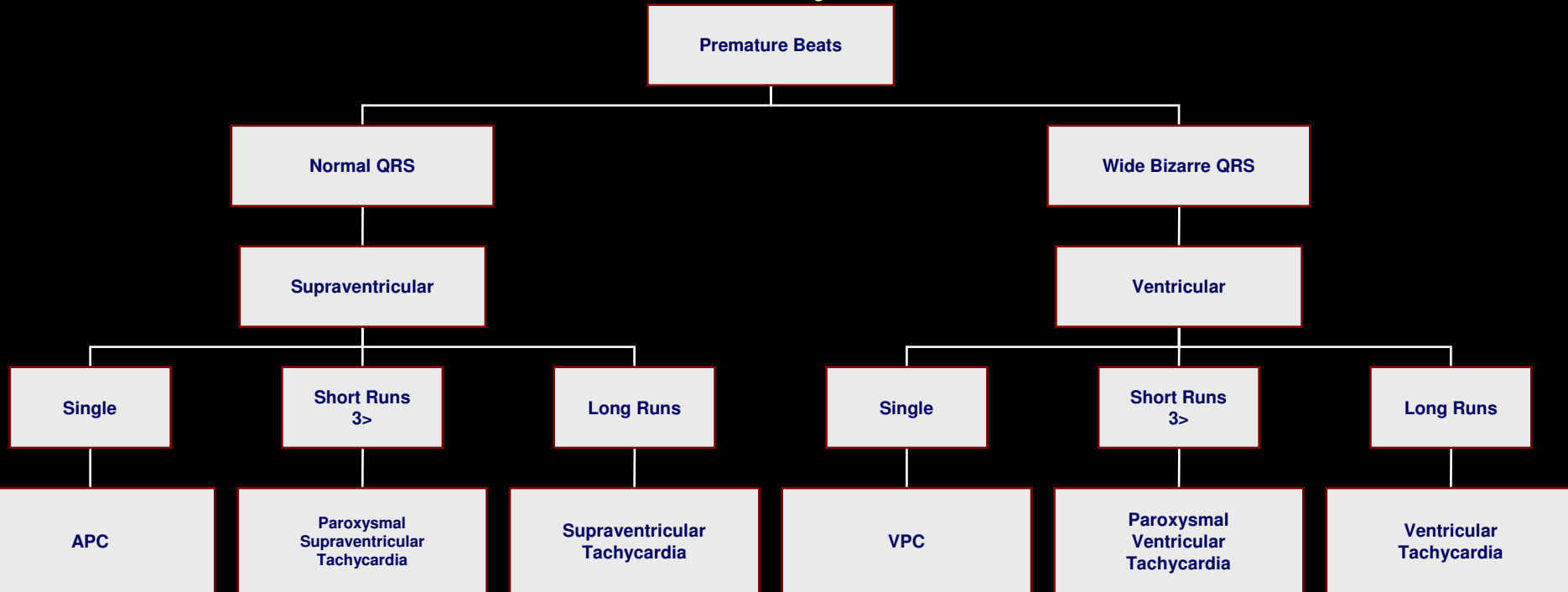


Tachyarrhythmias

- Disorders of impulse conduction
 - Lead to tachyarrhythmias
 - Re-entry phenomenon
- Disorders of Impulse formation
 - Enhanced or abnormal Automaticity
 - Triggered Activity
- Results in premature complexes on ECG

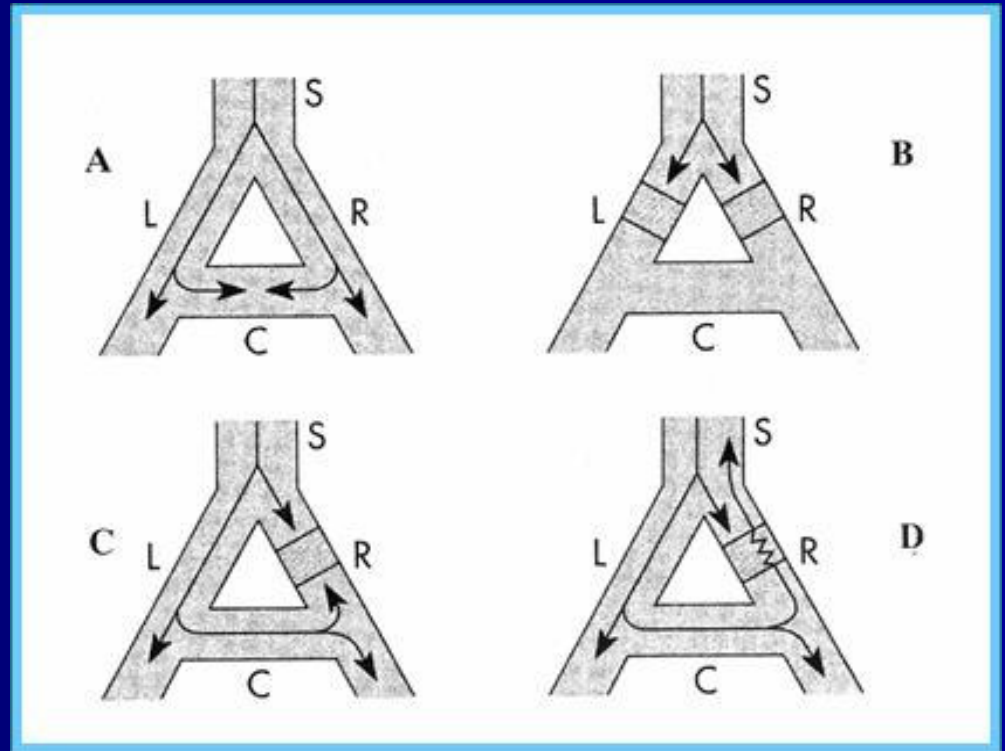
Premature Ectopics

Premature Beats Summary

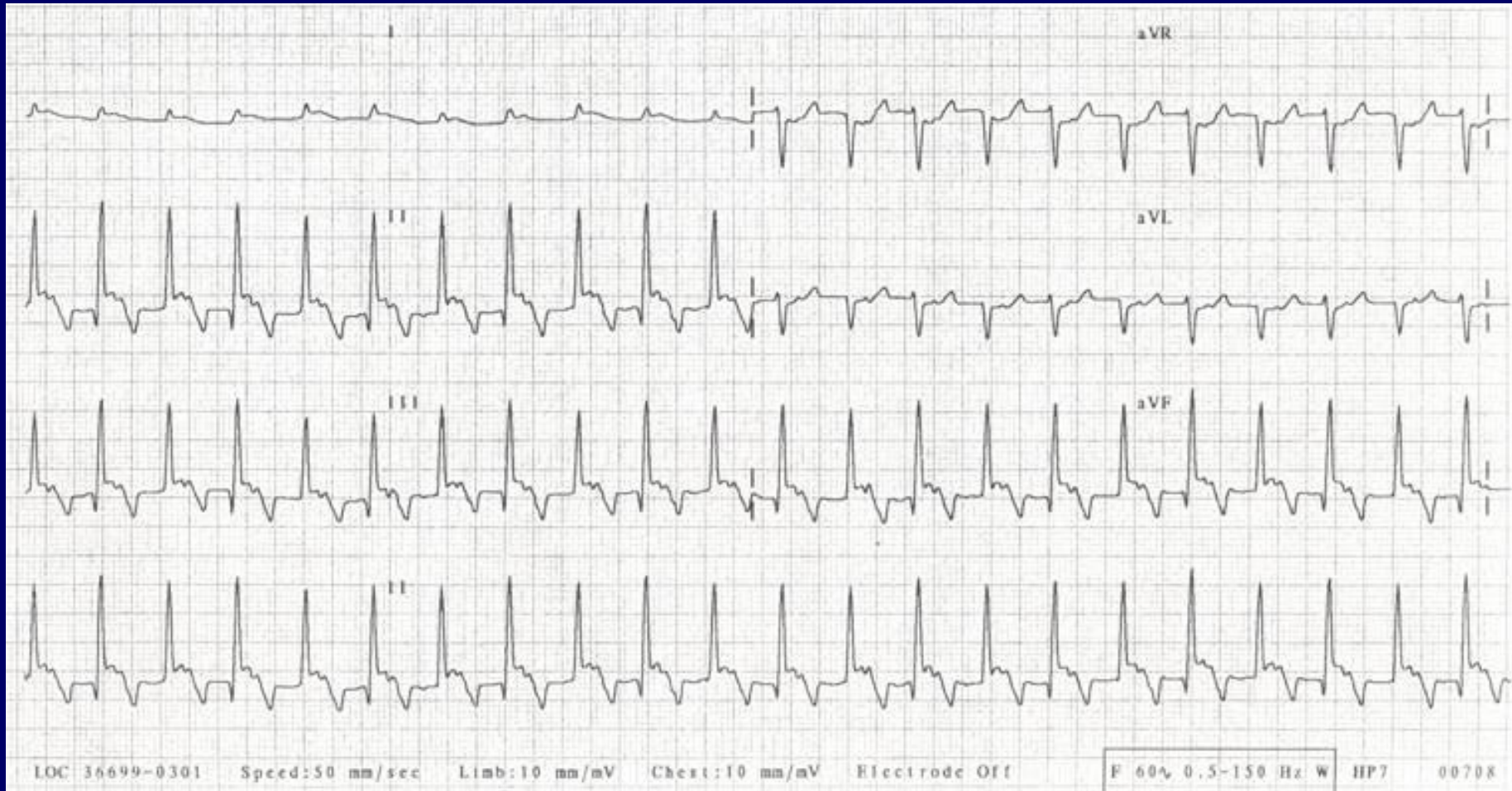


Re-Entry

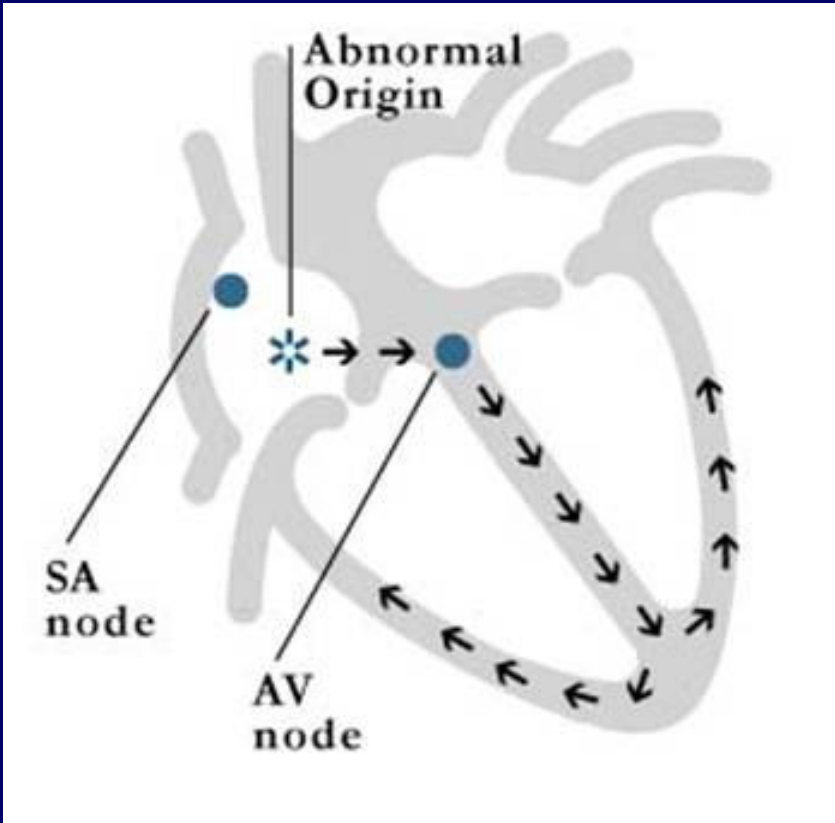
- Unidirectional block
- $ERP < \text{propagation time in loop}$
- Most common cause of arrhythmias



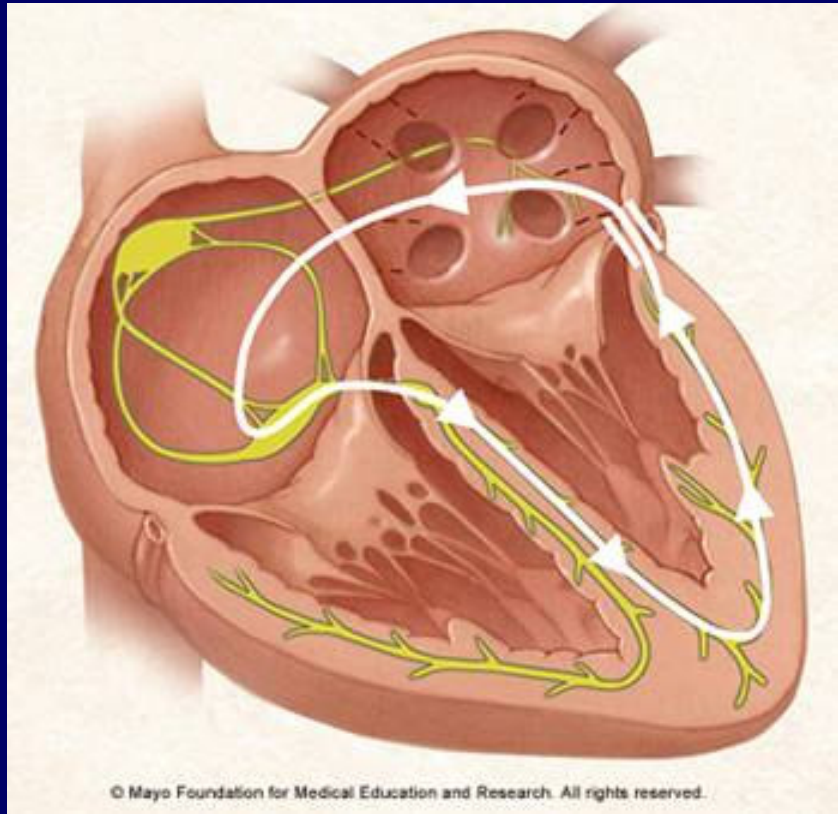
Supraventricular Tachycardia



Mechanisms of SVT



AV node no part of circuit



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AV node part of circuit

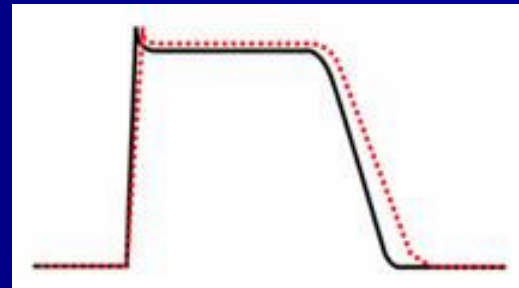
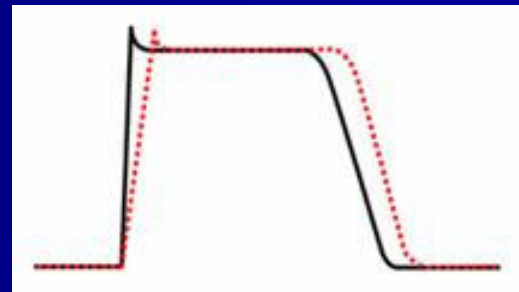
Treatment of SVT

- Vagal Maneuvers

- Increase vagal tone to AV node
- Slow ventricular response
- May break SVT rhythm using nodal pathway

- Class Ia or Class III

- Procainamide or Sotalol

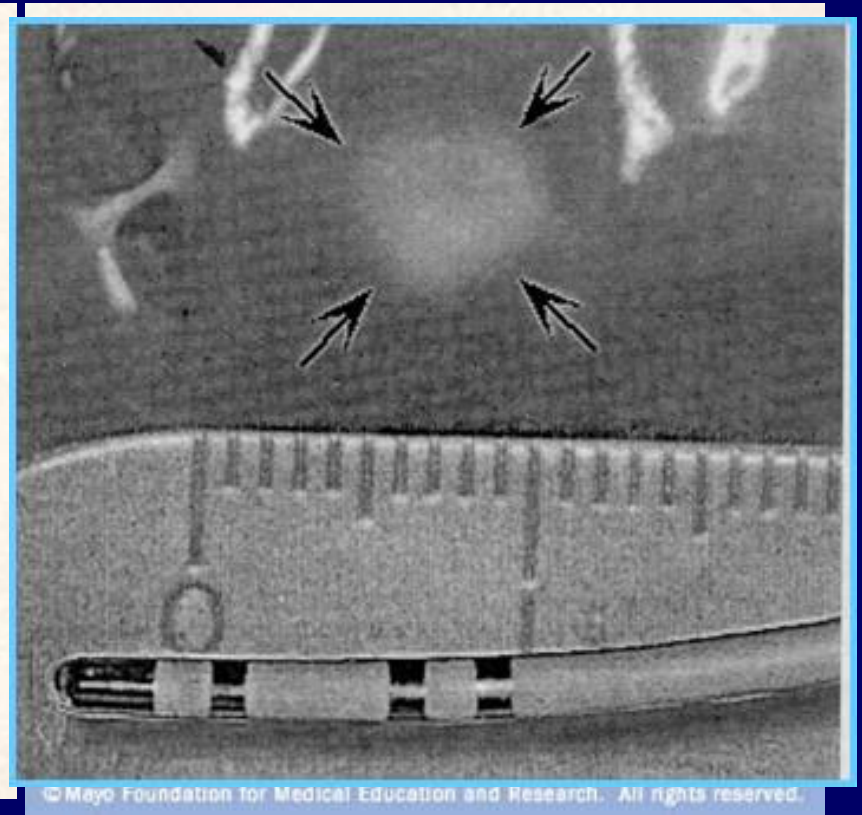
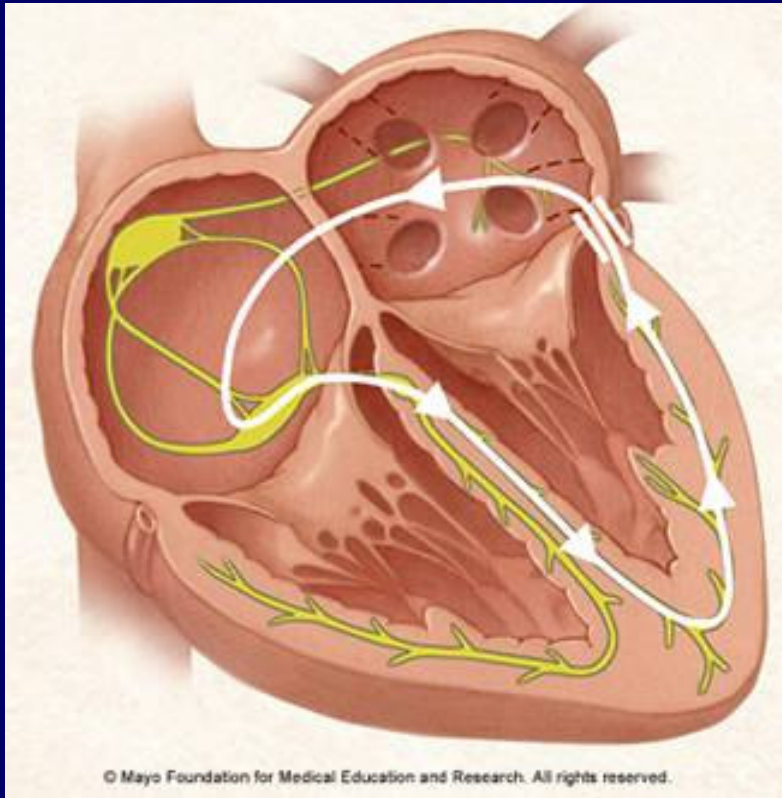


Response to VM or Class Ia



- Blocks a P wave
- At least that the AV node is involved
- May indicate accessory pathway
 - Ventricular pre-excitation
 - Short PR interval

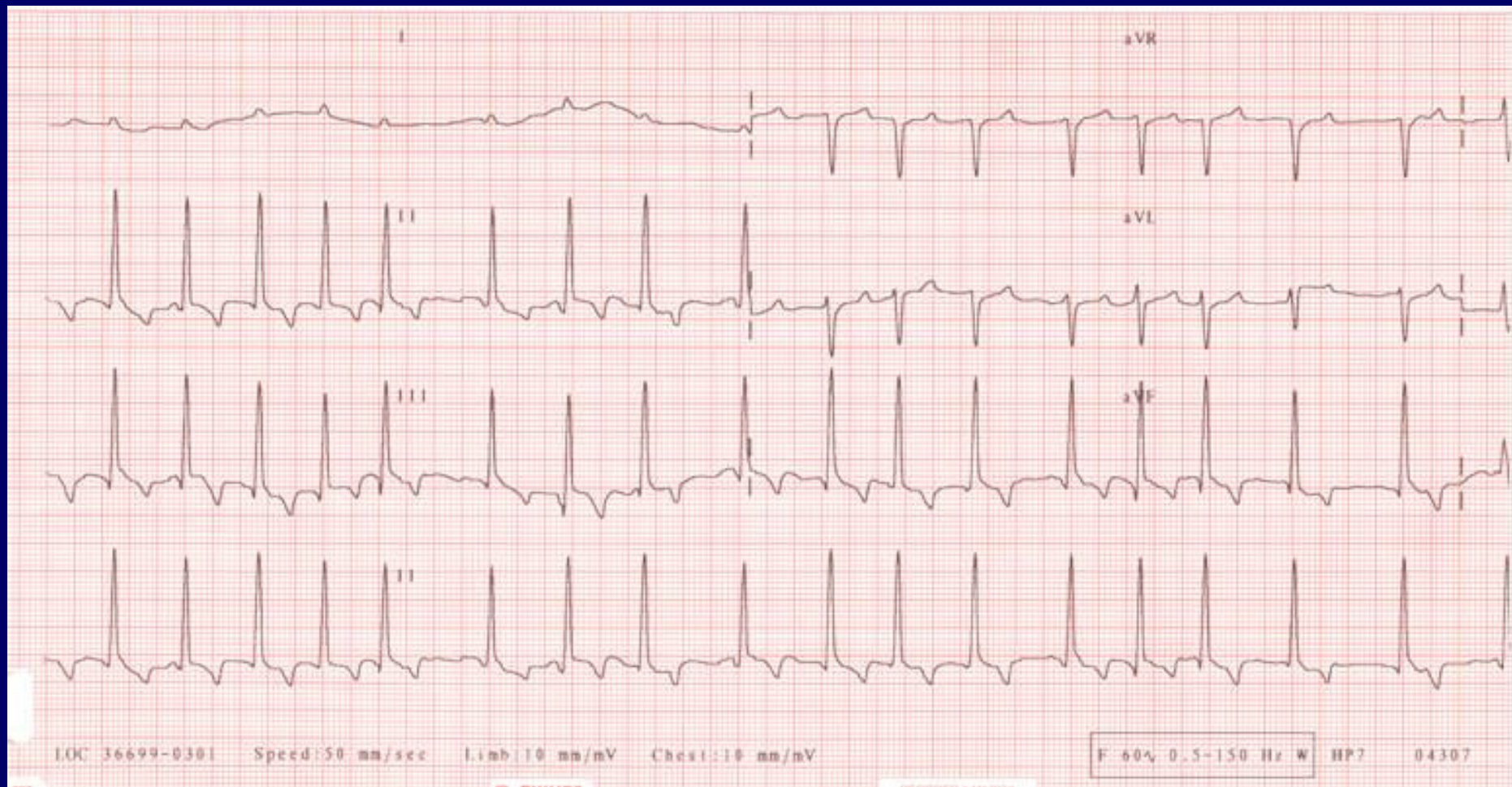
Electrophysiologic Study



Chronic Therapy

- Chronic oral Sotalol therapy
 - Prolongs AP of atrial myocytes to break circuit
- Slowing AV node conduction
 - Calcium channel blockers
 - Diltiazem
 - Beta blockers
 - Atenolol
 - Digoxin

Atrial Fibrillation

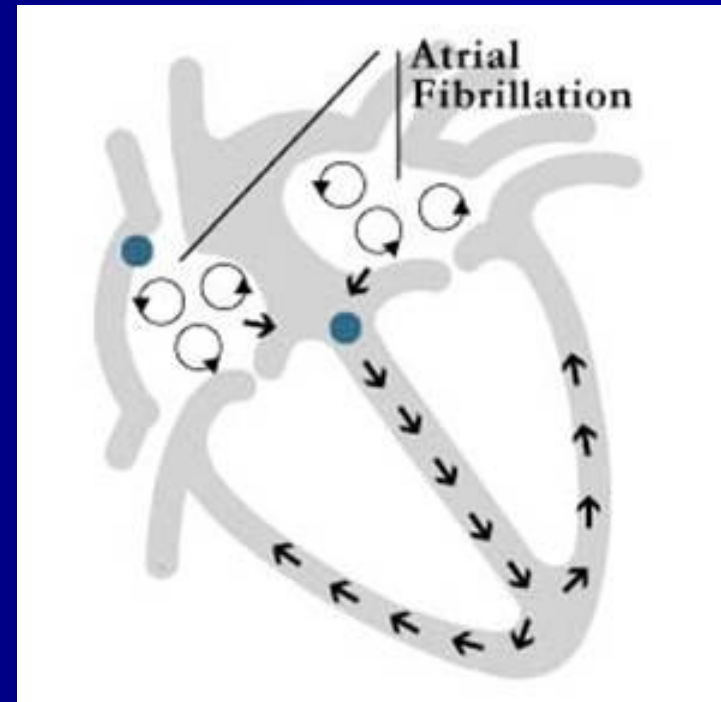


Atrial Fibrillation

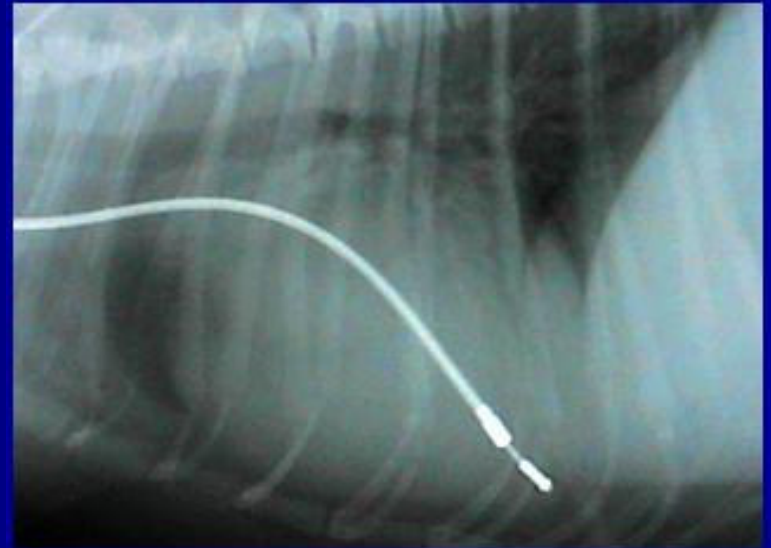
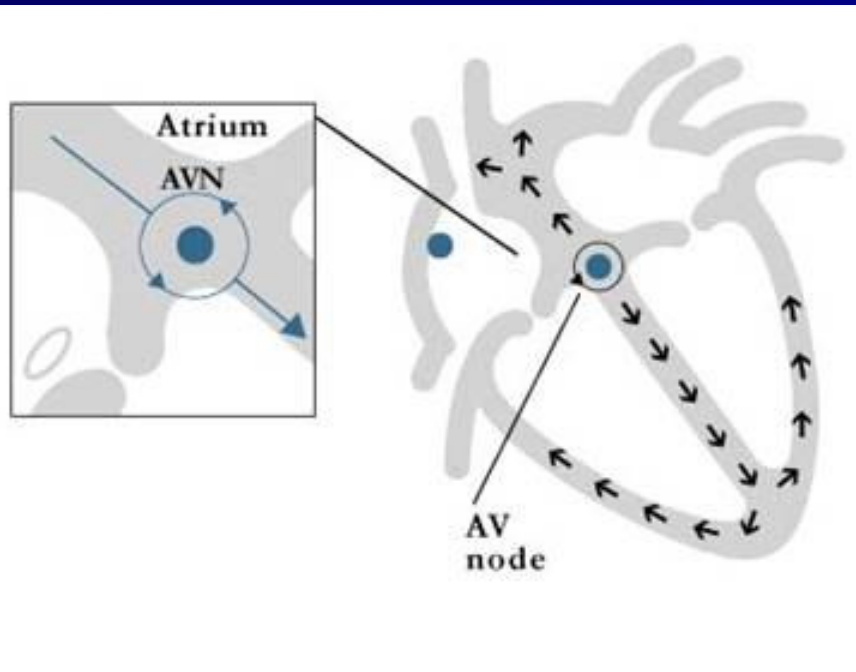
- Classically no discernable P waves
- Very irregular ventricular response
- Often associated with underlying heart disease
 - Leads to numerous self sustaining micro-reentrant circuits

Atrial Fibrillation Therapy

- Convert to Sinus rhythm
 - Class Ia or Class III
 - Quinidine
 - Amiodarone
 - Cardioversion
- Aimed at slowing ventricular response rate
 - AV nodal drugs
 - CCB- Diltiazem
 - B-blockers- Atenolol
 - Digoxin

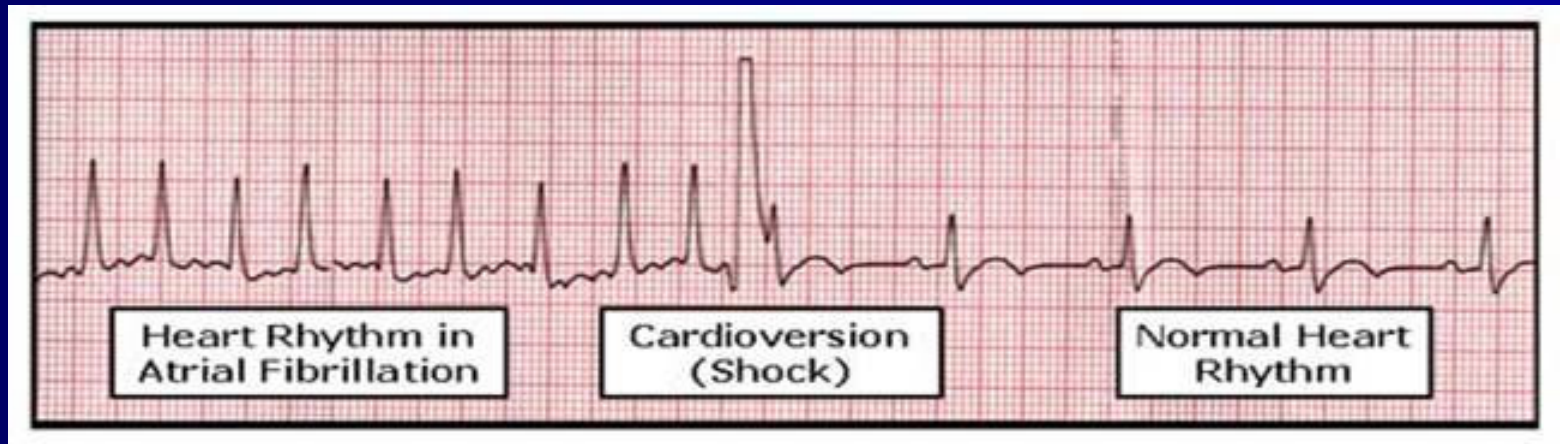


Advanced Therapy

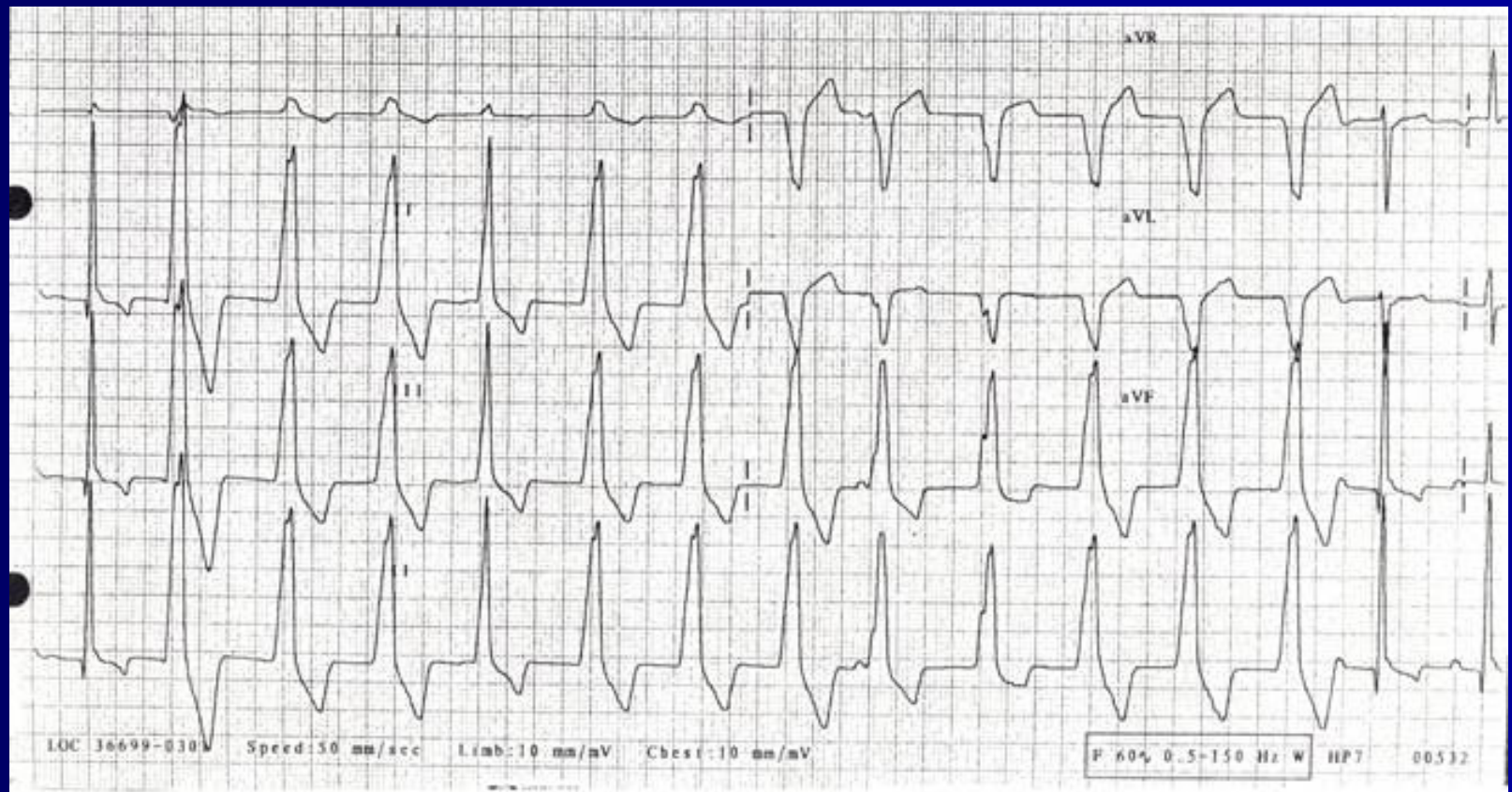


Advanced Therapy

- Biphasic Cardioversion
 - Appropriately timed stimulus breaks rhythm
- Bright J et al: Convert 92% AF patients

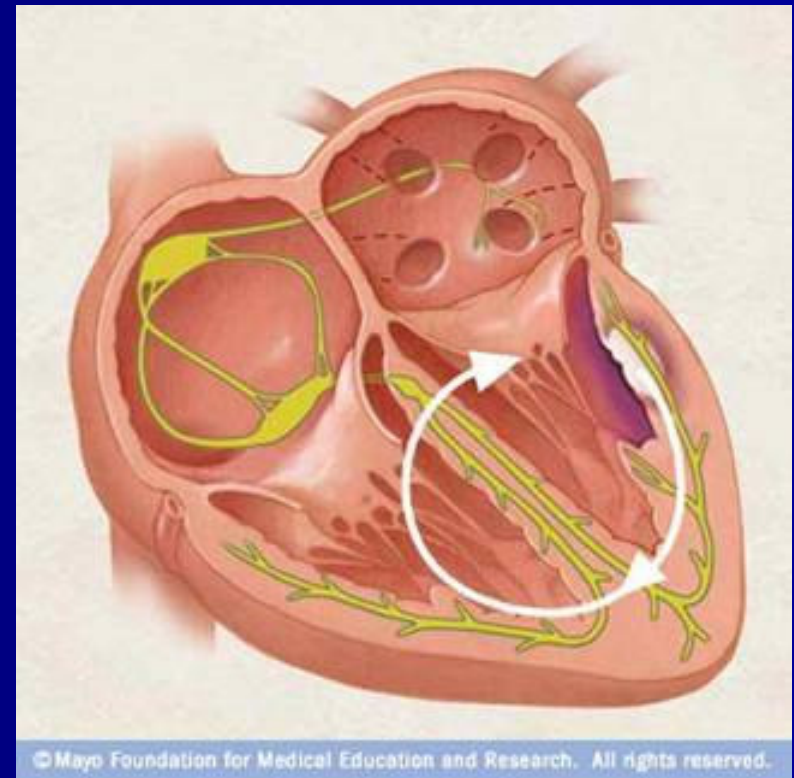


Ventricular Tachycardia



Ventricular tachyarrhythmias

- Originates from ectopic focus in ventricle
- Significantly compromises hemodynamics
- Detrimental to myocardium



Ventricular Origin

- Ventricular Premature Complexes
 - Unifocal – Holter to assess for quantity
 - Multifocal –
 - Animal stable – Oral medications
 - Class Ia – procainamide
 - Class Ib- Mexiletine +/- Class II- Atenolol
 - Class III - Sotalol
 - Tachycardia, Unstable Multifocal, or Fibrillation
 - Class Ia – Parentally – IV Procainimide
 - Class Ib – Parentally – IV Lidocaine
 - Treat the underlying cause as a large % are extracardiac causes

Specific Anti-Arrhythmic Uses

Sinus tachycardia

No treatment necessary, treat underlying cause

Sinus bradycardia

No treatment usually necessary, treat underlying cause

Sinus pause or Sinus block

No treatment necessary if infrequent or absence of clinical signs

Oral anticholinergics can be tried, pacemaker

Sinus arrest

Oral anticholinergics can be tried, pacemaker implantation is preferred treatment

1AVB

No treatment necessary, treat underlying cause

2AVB type I

No treatment necessary, treat underlying cause

2AVB type II

Low-grade

Treatment is not usually necessary, monitor ECG

High-grade

Anticholinergics can be tried but usually Unsuccessful

Pacemaker implantation is preferred treatment

3AVB

Pacemaker implantation

Ventricular Pre-excitation

No treatment necessary if no SVT present

SVT can be treated with Procainamide (accessory pathway), β -blocker (atenolol) (AV node), Ca-channel blocker (diltiazem) (AV node), Class III antiarrhythmic (Sotalol) (accessory pathway, AV node)

refractory SVT can be treated with RF ablation of the accessory pathway

Atrial premature complexes-

Treatment not usually necessary for individual ectopics, treat underlying cause (ie CHF)

Supraventricular tachycardia-
(Atrial/junctional)

Most commonly use drugs that prolong AV nodal conduction (digoxin, CCB, β -blocker) due to underlying cardiac disease

Class Ia drugs (Procainamide, Quinidine) or Class III drugs (Sotalol) combined with AV nodal drugs can sometimes terminate underlying mechanism

Atrial fibrillation

most commonly secondary to underlying organic heart disease where ventricular response rate is primary goal (digoxin, CCB, β -blocker) to slow AV nodal conduction

without underlying cardiac disease, conversion to sinus rhythm can be attempted (Quinidine, +/- digoxin, DC cardioversion)

Sick Sinus Syndrome

care should be taken to document the cause of symptoms (ie bradycardia from sinus arrest or supraventricular tachycardias)

oral anticholinergics can be used, often ineffective, exhibit significant side-effects, can exacerbate SVT's

AV nodal drugs (digoxin, CCB, β -blocker) are effective for SVT's but will exacerbate the sinus pause/block/arrest

permanent pacemaker is preferred treatment, refractory SVT's can then be safely treated with AV nodal drugs (digoxin, CCB, β -blocker)

Ventricular premature complex

treat if risk for worsening arrhythmia

- >20/min
- multiforme/multifocal
- R on T's

IV Lidocaine bolus, if conversion use continuous infusion

IV Procainamide bolus if Lidocaine is ineffective (Procainamide can be used first), continuous infusion

oral/IM Procainamide, oral Class Ib (Mexiletine), oral/IM Quinidine if stable

Ventricular tachycardia

attempt to identify underlying cause

if hemodynamically compromised/life-threatening:

IV Lidocaine bolus, if conversion use continuous infusion

IV Procainamide bolus if Lidocaine is ineffective (Procainamide can be used first), continuous infusion

IV Quinidine, IV Bretylium, DC cardioversion are alternative therapies for refractory life-threatening ventricular tachycardia

Ventricular fibrillation

DC cardioversion, lidocaine and epinephrine

Accelerated Idioventricular rhythm

treatment usually not necessary

inherent overdrive suppression when sinus rate increases

if multifocal or hemodynamically compromised, treat as ventricular tachycardia

Ventricular escape rhythm

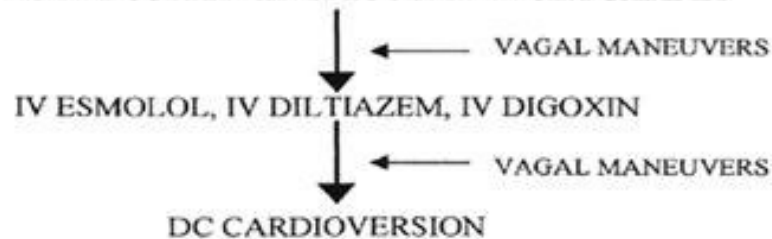
!!DO NOT TREAT!!

permanent pacemaker implantation to relieve symptoms from the bradycardia (ie 3AVB)

Atrial Standstill

pacemaker implantation

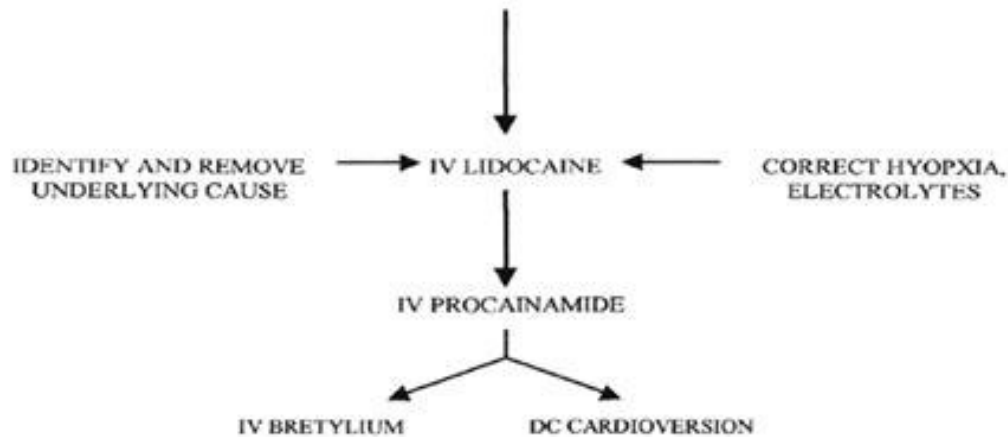
EMERGENT SUPRAVENTRICULAR TACHYCARDIA



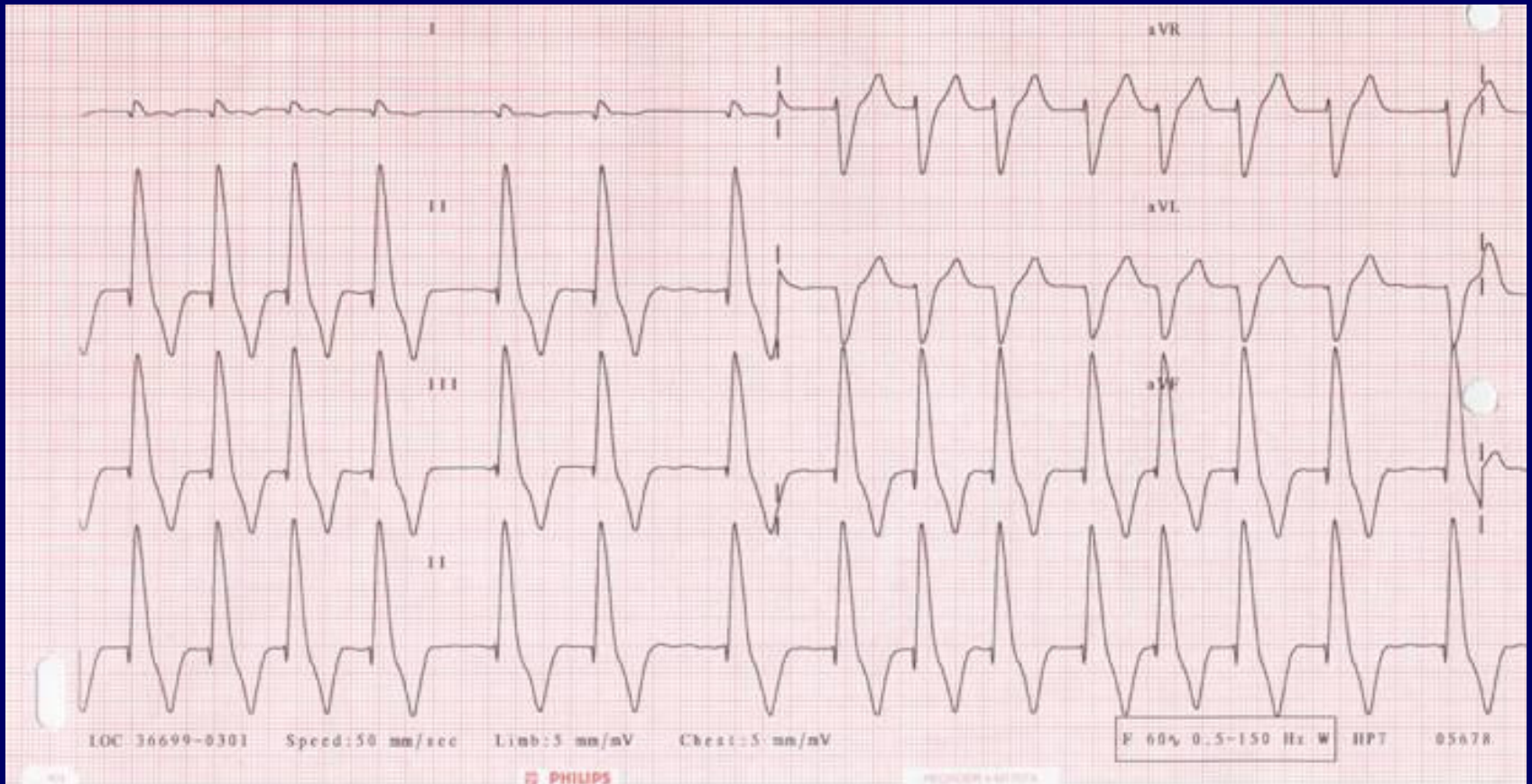
NON-EMERGENT SUPRAVENTRICULAR TACHYCARDIA



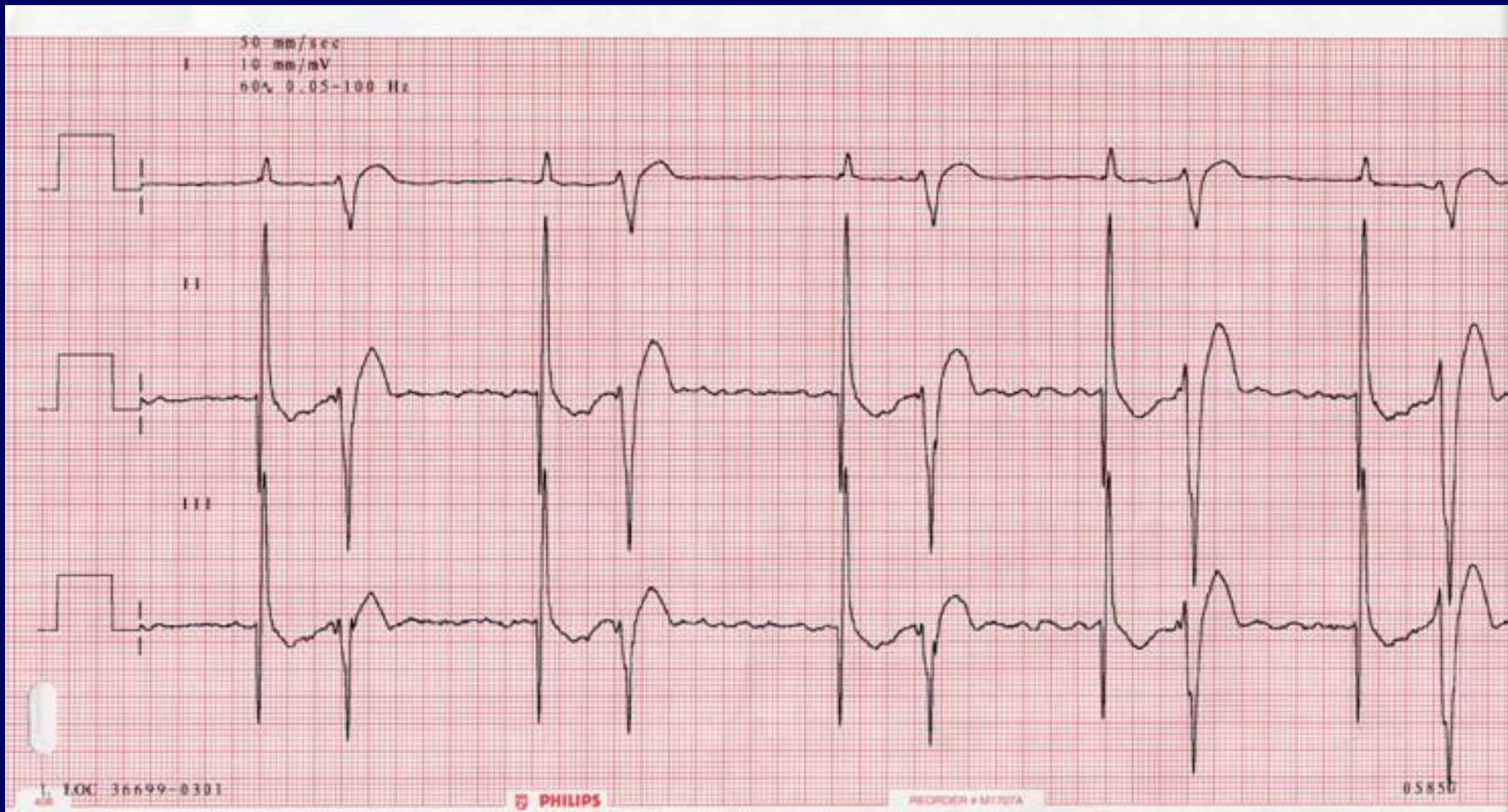
LIFE-THREATENING VENTRICULAR TACHYCARDIA



Atrial Fibrillation



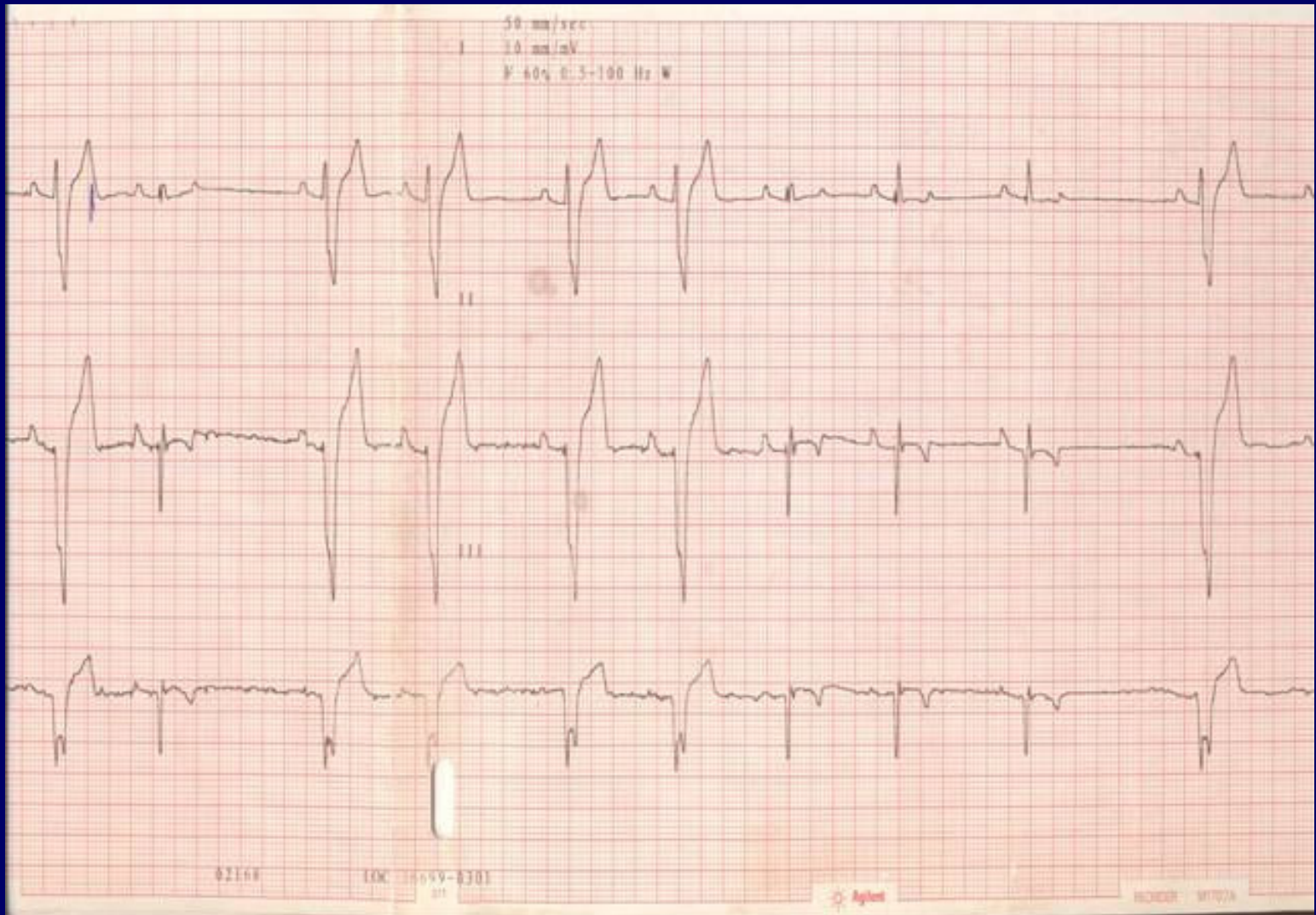
Atrial Fib + Ventricular Bigeminy



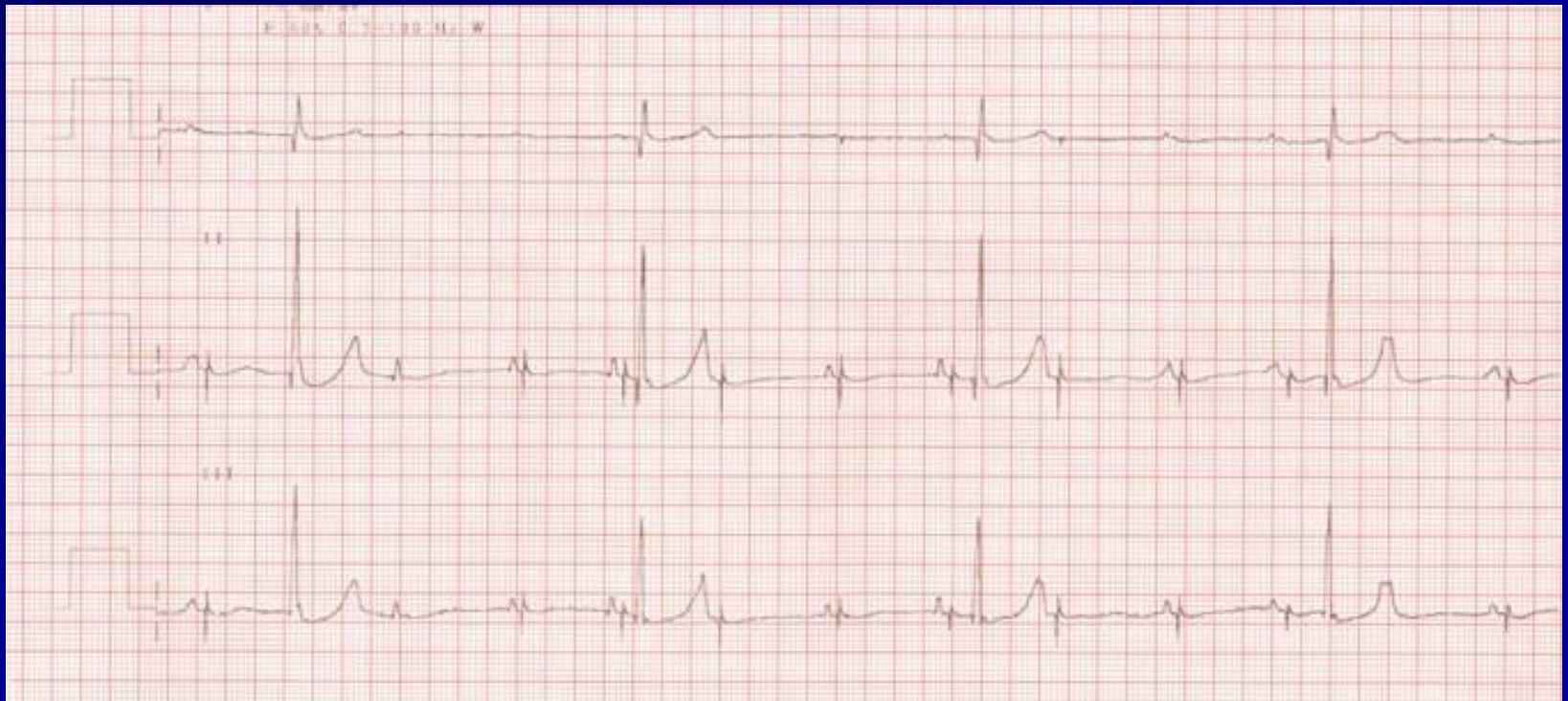
Heart Cycle

- Heart Cycle
 - Thinkwell

Int. RBBB (Rate Dependent)



3AVB, Ventricular Escape Pacemaker Non-Capture



Ventricular Tach. - Multiform



Electrophysiologic Study

