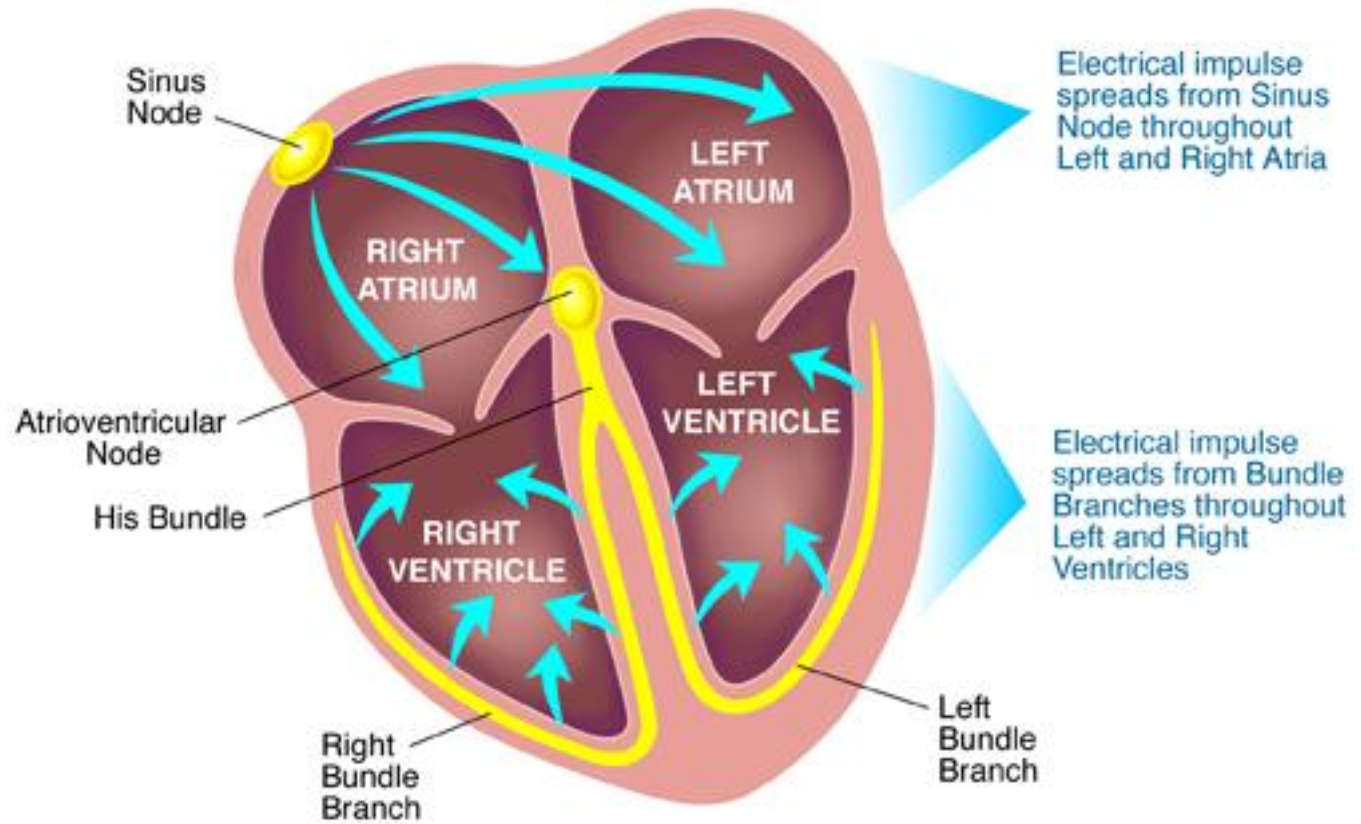


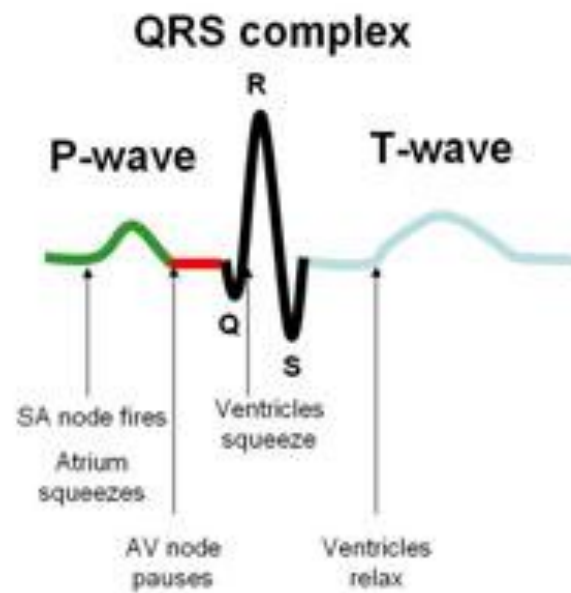
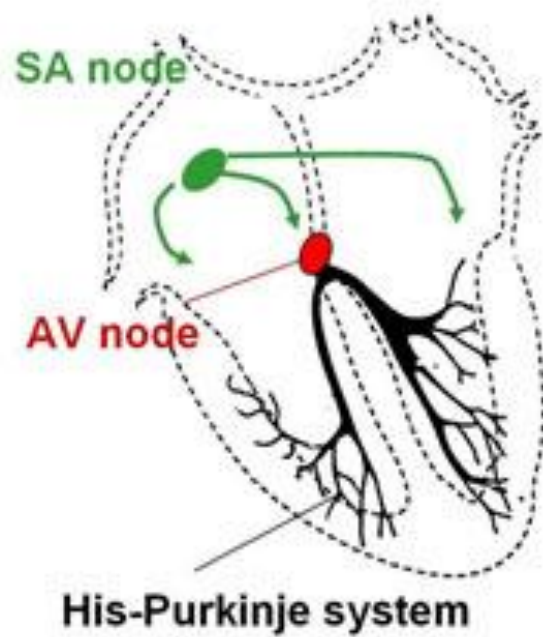
CARDIAC ARRHYTHMIAS

Sergey Yalonetsky, MD

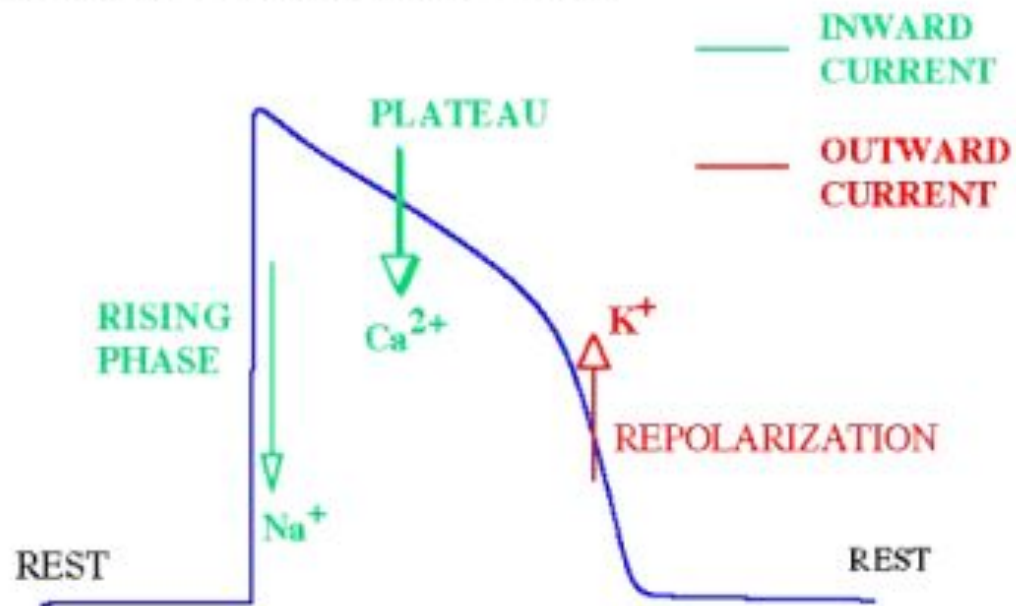


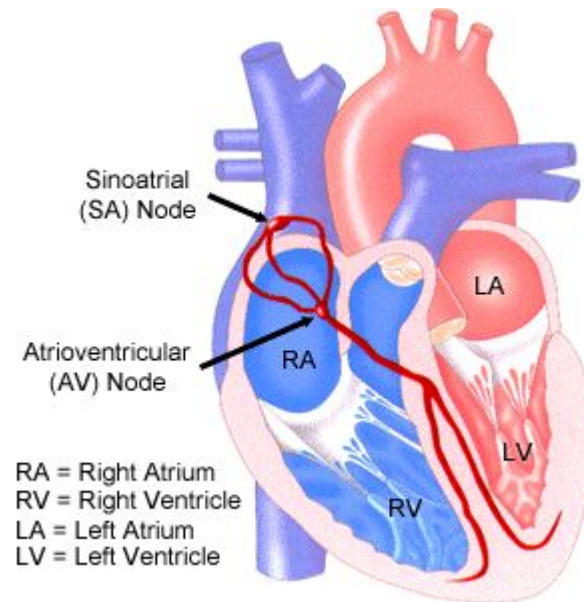
© Ron Leishman * www.ClipartOf.com/437774





CARDIAC ACTION POTENTIAL





Normal Sinus Rhythm



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

Normal Sinus Rhythm



Heart
Rate

Rhythm

P Wave

PR interval
(in seconds)

QRS
(in seconds)

60-100 bpm

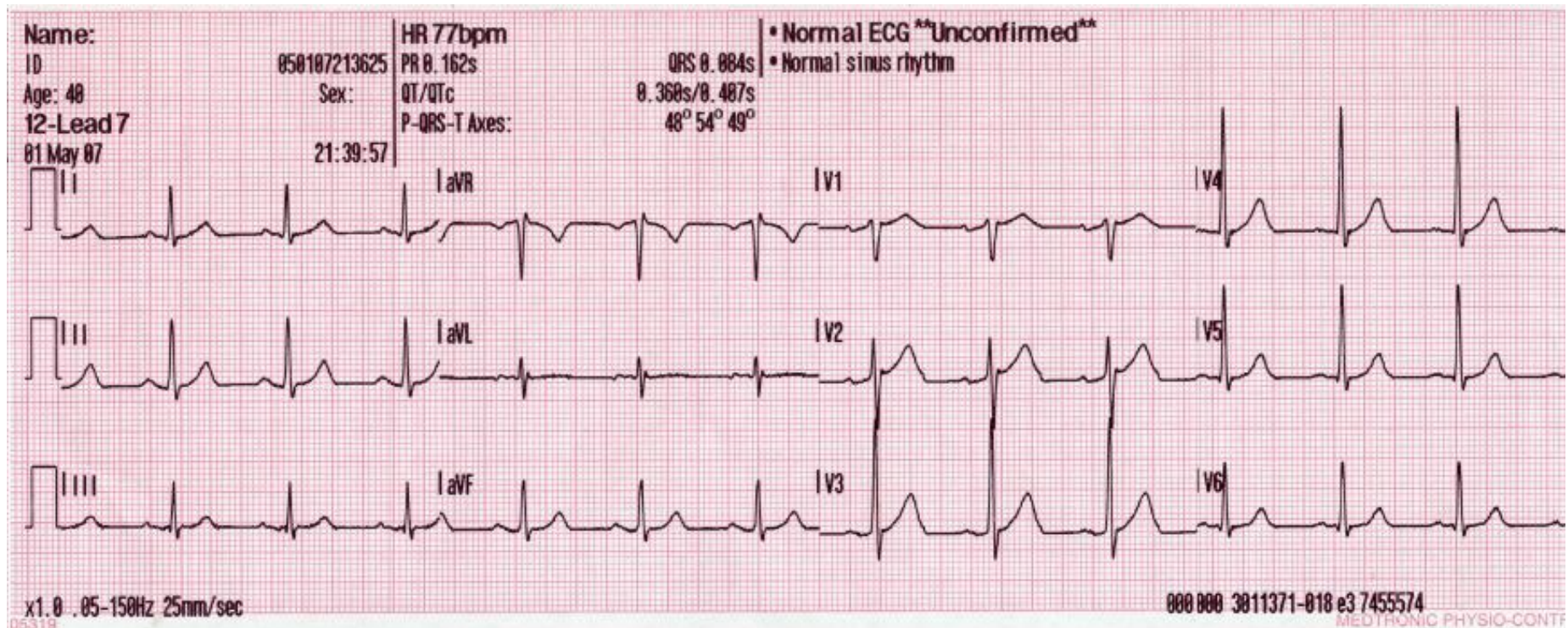
Regular

Before each
QRS, identical

.12 to .20

<.12

Normal Sinus rhythm



Classification

- Tachyarrhythmia:
 - Supraventricular
 - Ventricular
 - Bradycardia
-

APB or PAC

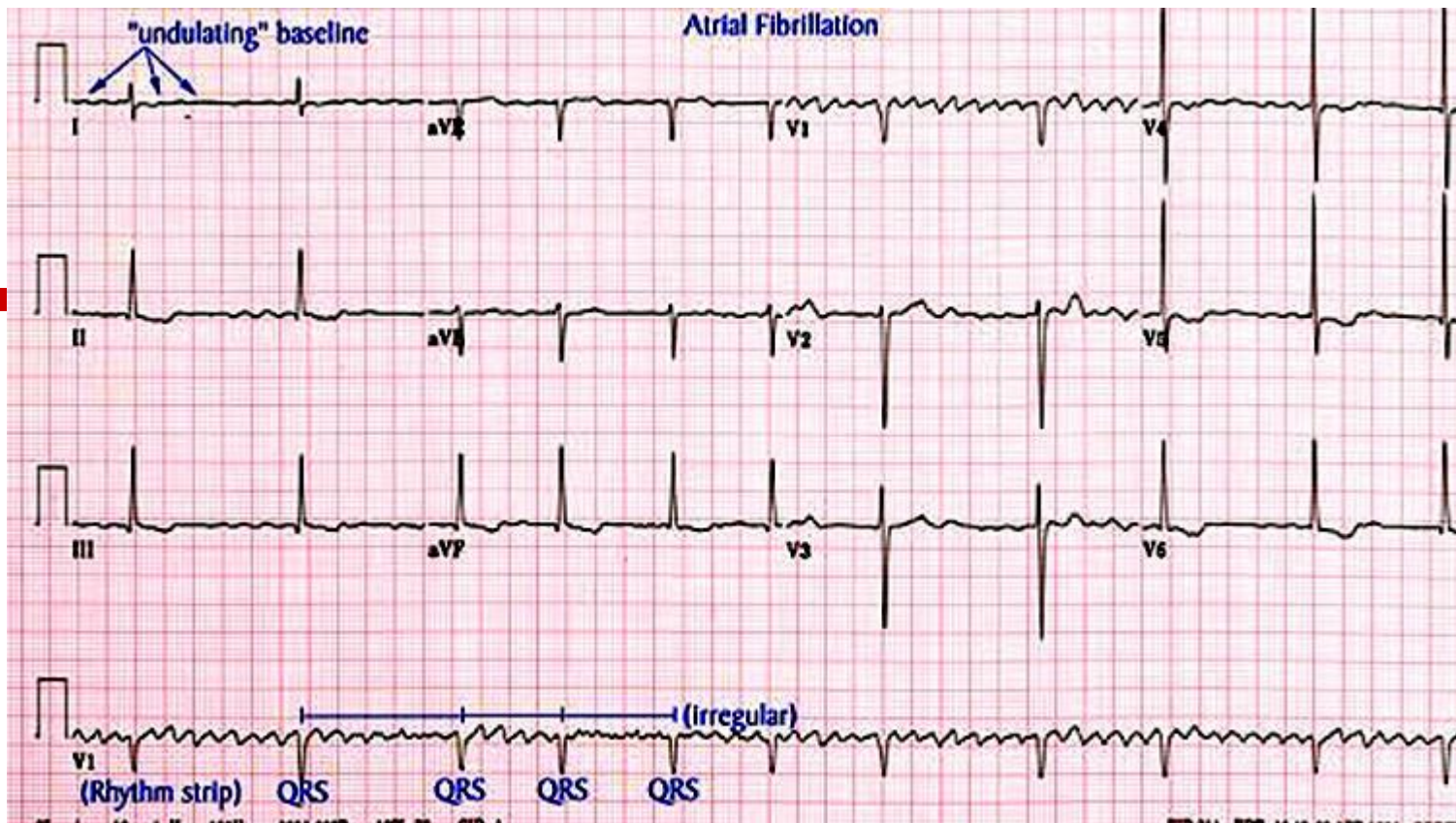
Premature Atrial Contraction • Isolated PAC's: Occur Single



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden	<.20	<.12

Atrial Fibrillation

- The most common arrhythmia in clinical practice
 - Frequency increases with age
-



Irregularly irregular rhythm
No P waves
F waves

Mechanism

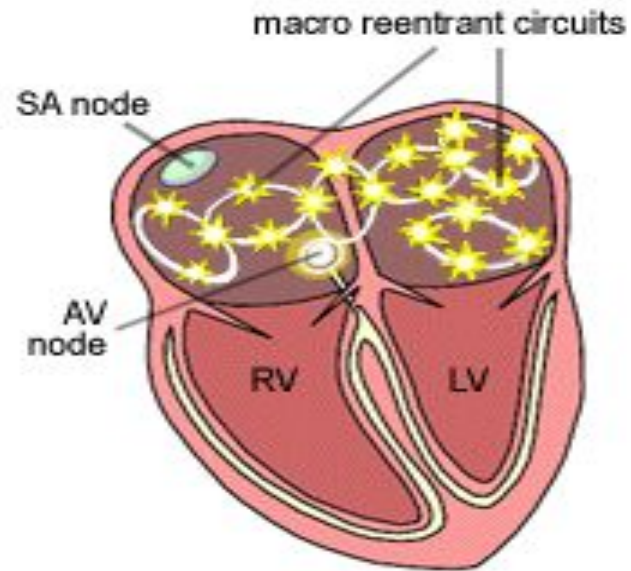
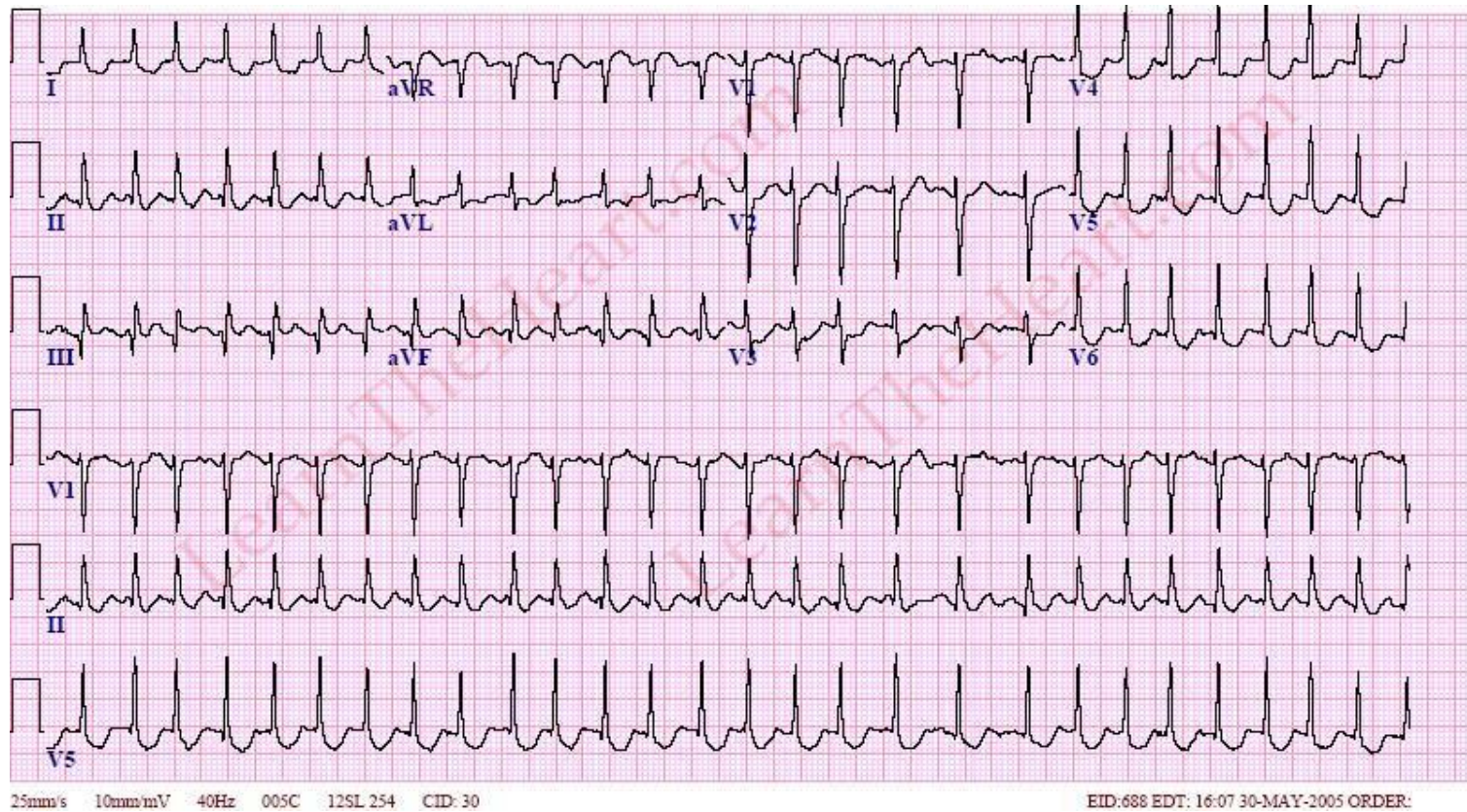


Illustration depicting macro reentrant circuit ("wavelet") activity during a run of complex atrial fibrillation (AF)

Most common causes

- Valvular heart disease: (MS,MR)
 - LV hypertrophy (HTN, other cause)
 - Cardiomyopathy
 - Thyrotoxicosis
 - Alcohol (“holiday heart”)
 - Atrial septal defect
 - Lone AF (structurally normal heart)
-

Rapid AF



Consequences of Atrial Fibrillation

Hemodynamic

- loss of synchronous atrial mechanical activity
- irregularity of ventricular response
- inappropriately rapid heart rate

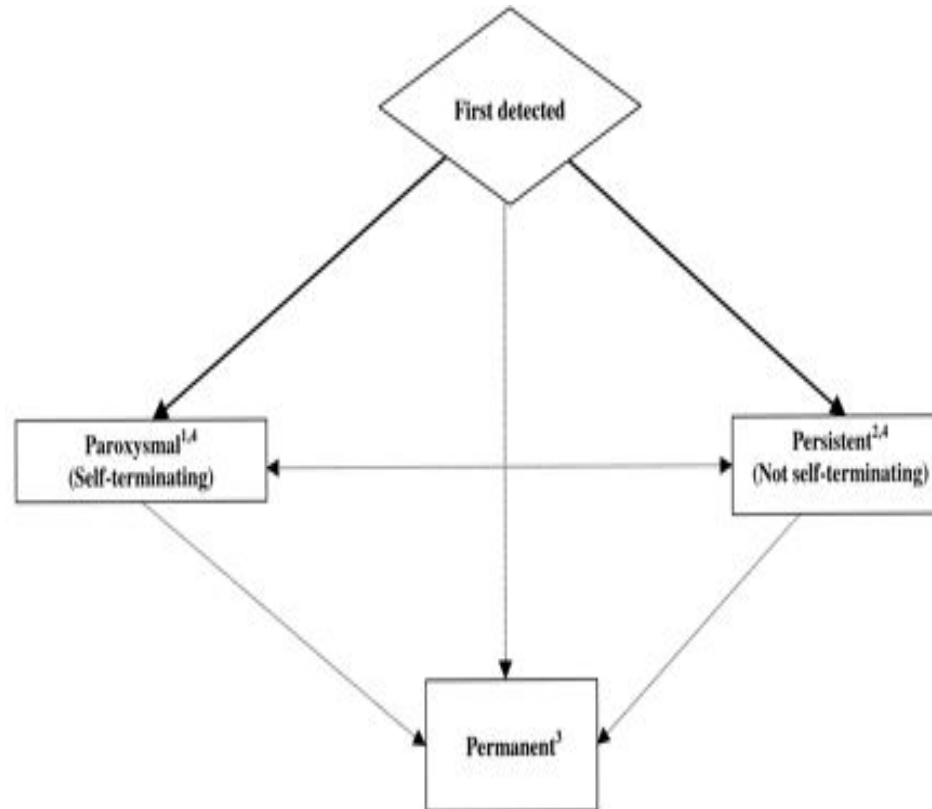
Myocardial – persistently rapid rate can lead to:

- atrial cardiomyopathy
- dilated ventricular cardiomyopathy

Thromboembolism

- ischemic stroke and systemic arterial occlusion attributed to LA and LAA thrombus

Classification



Treatment options

- 1. Rhythm control – restoration and maintenance of sinus rhythm
- 2. Rate control

Prevention of Thromboembolism !

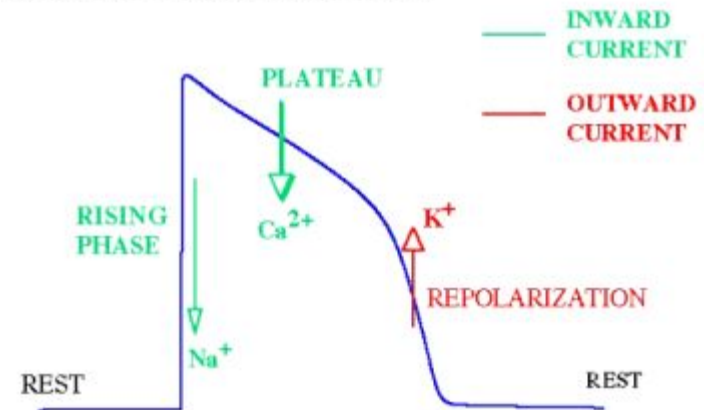
Williams Classification of Antyarrhythmic Drugs

□ **Class I-** blocking the fast Na channels:

IA – Reduce V_{max} and prolong action potential

- Quinidine
- Procainamide
- Disopiramide

CARDIAC ACTION POTENTIAL



IB : Do not reduce V_{max} and shorten action potential duration

- Lidocaine
- Phenytoin
- Mexiletine

IC: Reduce V_{max}

- Flecainide
 - Propafenon
-

-
- Class II – beta blockers
 - Class III – K channel blockers
 - Amiodaron
 - Sotalol
 - Bretylium
 - Class IV – Ca channel blockers
-

Cardioversion

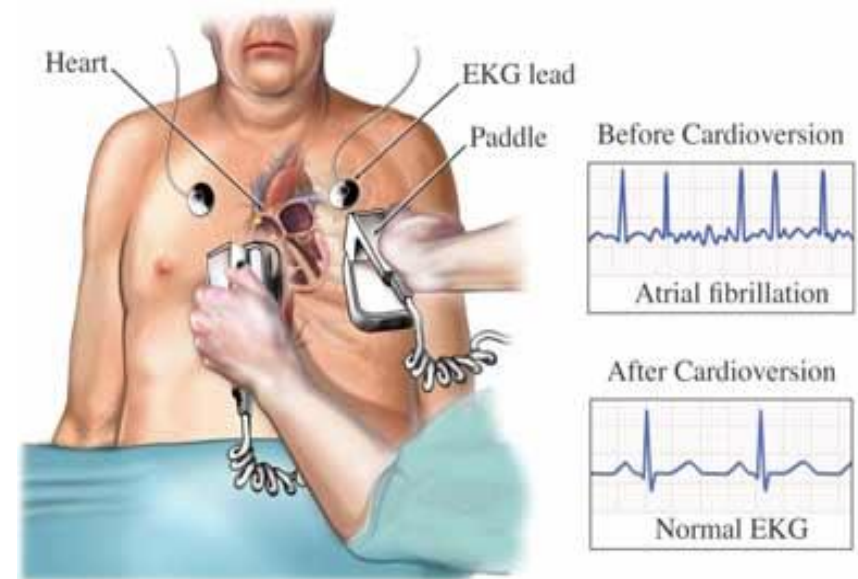
Pharmacological

- Propafenon
 - Amiodaron
 - Flecainide
-

Cardioversion

Electric

- In acute setting (hemodynamically unstable pt)
- In Chronic Setting
Elective cardioversion

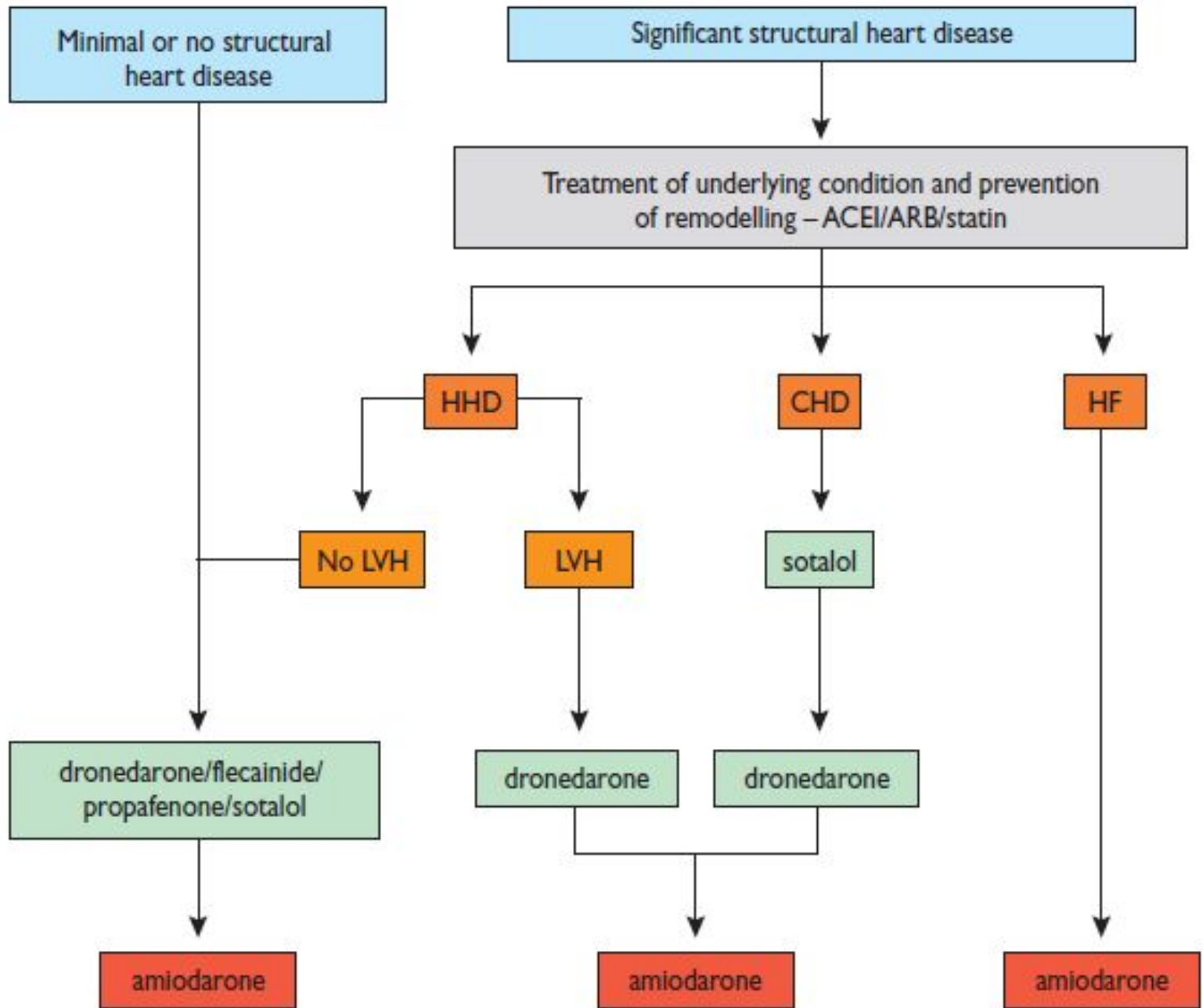


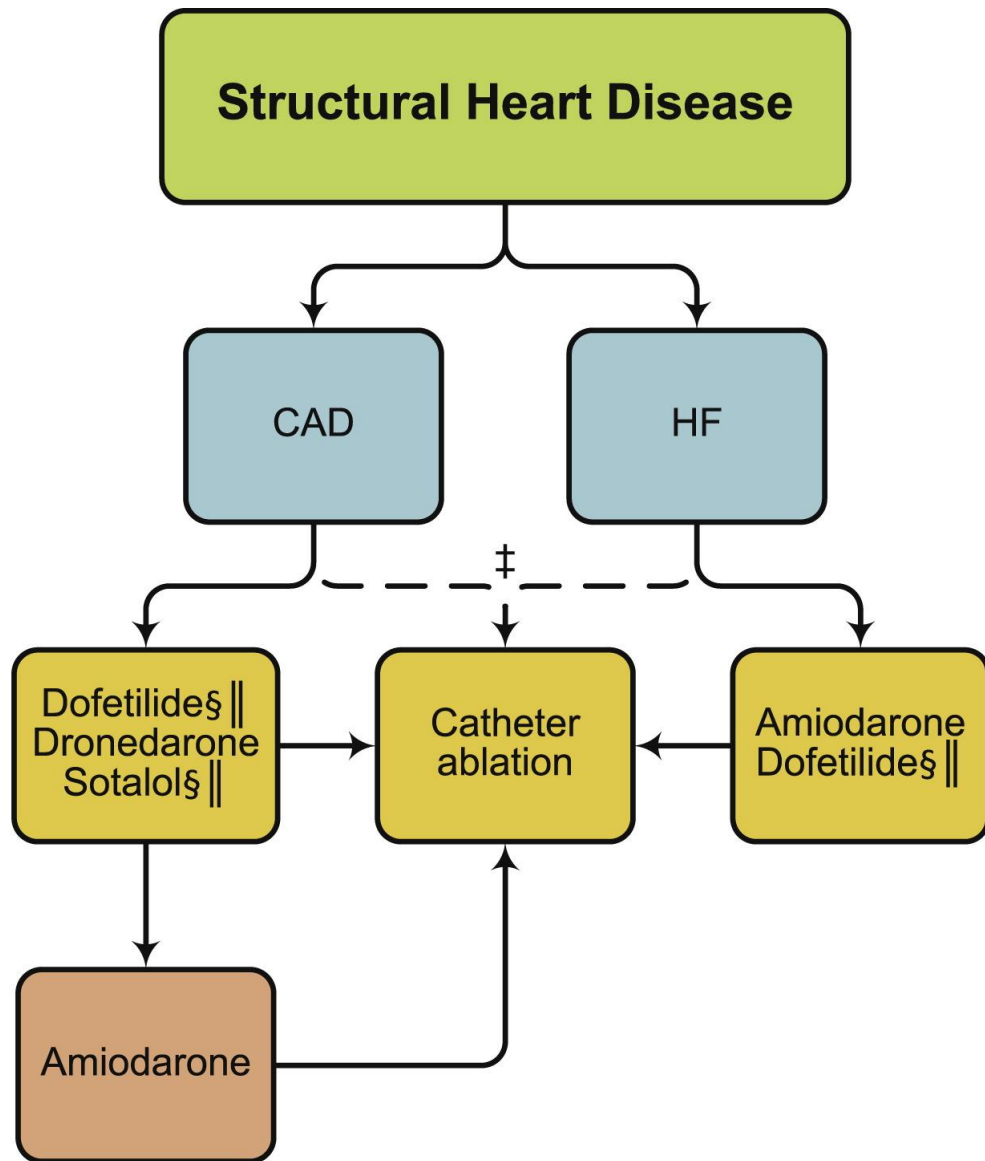
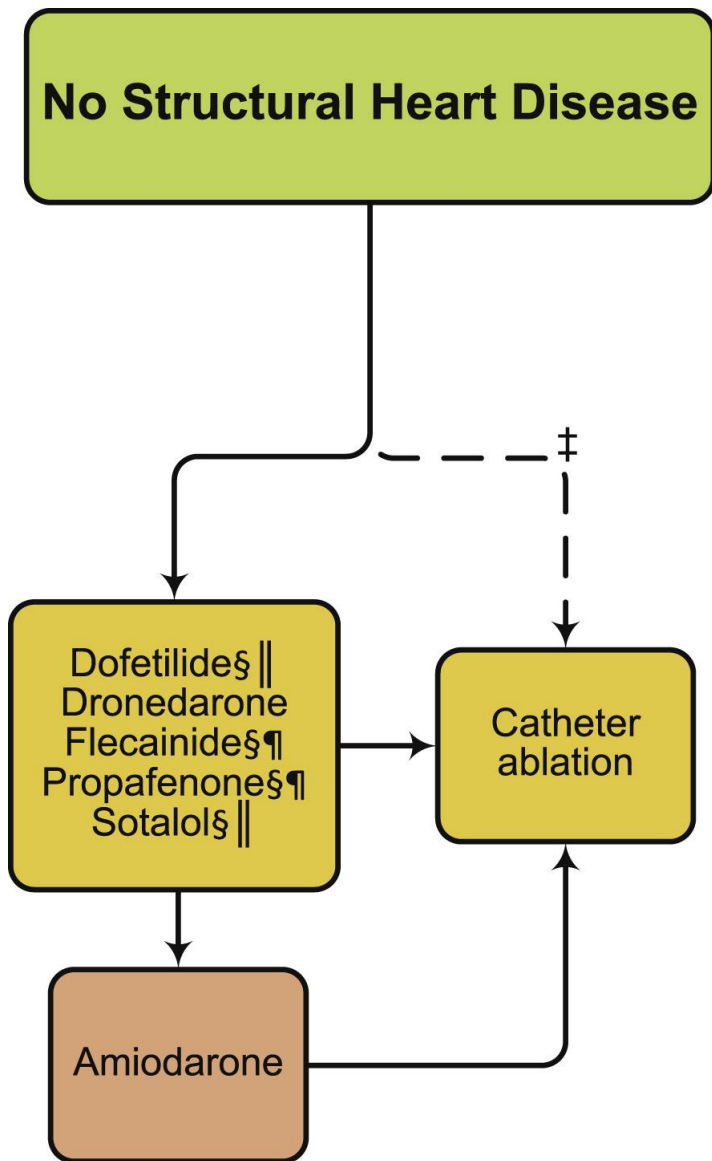
Predictors of successful cardioversion

- Short AF duration
 - Young age
 - Normal atrial size
 - No organic heart pathology
-

Maintenance of sinus rhythm

- Propafenon
 - Amiodaron
 - Dronedaron
 - Sotalol
 - Flecainide
-





Rate Control

- Acute setting – IV
 - Esmolol
 - Metoprolol
 - Verapamil
 - Diltiazem
 - Digoxin (HF)
 - Chronic setting – PO (the same drugs)
-

Table 3. Advantages and Disadvantages of Rate and Rhythm Control Strategies

Rate Control	Rhythm Control
Advantages	
Generally safe	Symptomatic improvement
Well tolerated	Hemodynamic improvement
Inexpensive	May reduce thromboembolic risk
	May allow discontinuation of anticoagulation
Disadvantages	
Incomplete symptom resolution	Proarrhythmic risk
Bradycardia	Extracardiac adverse effects
Life-long anticoagulation	Frequently ineffective
Cardiomyopathy if rate poorly controlled	Expensive

Attempt Rhythm Control First

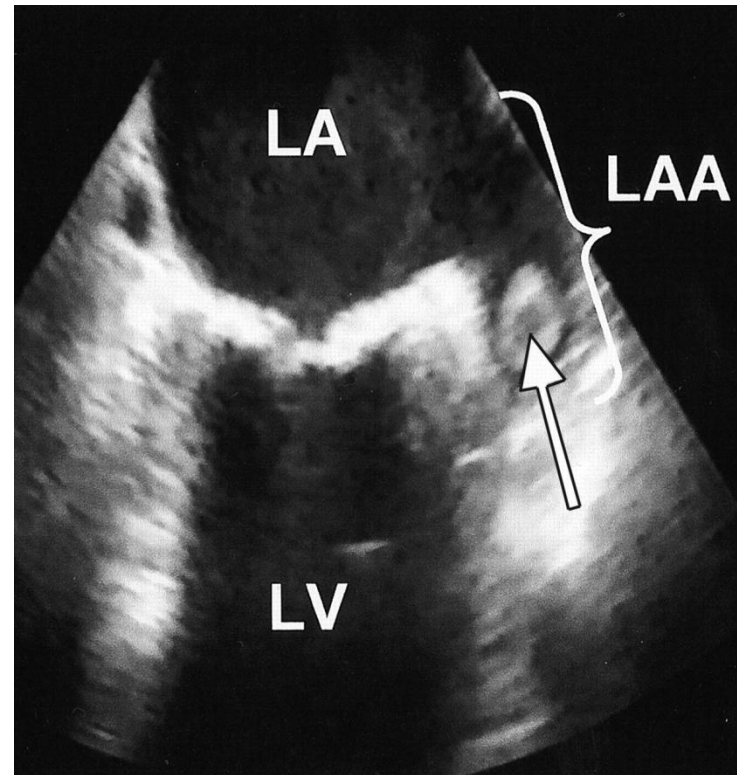
- Severe symptoms due to AF
 - Patients with CHF
 - Younger patients
 - Patients with lone AF
 - First episode of AF
-

Rate Control as First-Line Choice

Consider rate control as first-line therapy if:

- Patient is relatively asymptomatic
 - Older age group
 - Absence of CHF
 - Restoration of sinus rhythm is unlikely
 - AF present >12 months
 - LA dimension >6 cm
 - Proarrhythmic risk is high
-

Left Atrial Appendage



Anticoagulation

Table 5. Guidelines for Cardioversion of Atrial Fibrillation

Duration	Anticoagulation	
	Pre cardioversion	Post cardioversion
<24–36 hours	Not mandatory	Not mandatory
>24–36 hours	Three weeks therapeutic INR OR Initiate anticoagulation (heparin and/or warfarin), transesophageal echocardiogram negative for atrial thrombus	Four weeks therapeutic INR

INR = international normalized ratio.

CHADS₂ score

CHADS₂ criteria	Points	Stroke risk score	Recommended therapy
Previous stroke or TIA	2	High 2–6	Warfarin (INR 2–3)
Age ≥ 75 years	1		
Hypertension	1	Moderate 1	Warfarin or aspirin
Diabetes mellitus	1		
Heart failure	1		
		Low 0	Aspirin 100–300 mg daily

Scoring Differences Between CHADS₂ and CHA₂DS₂-VASc

Risk Factor	CHADS ₂	CHA ₂ DS ₂ -VASc
	(Maximum score, 6)	(Maximum score, 9)
	Points	Points
Congestive heart failure	1	1
Hypertension	1	1
Diabetes	1	1
Vascular disease	N/A	1
Age 65-74	N/A	1
Age ≥75	1	2
Female sex	N/A	1
Previous stroke/TIA	2	2

N/A – not applicable

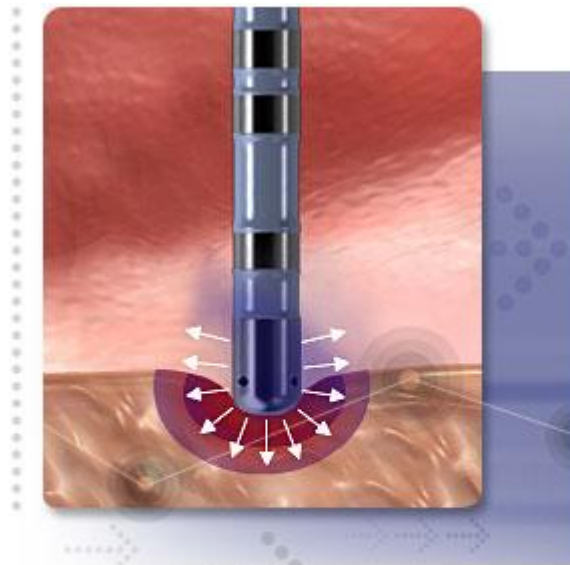
Novel Oral Anticoagulants

- Dabigatran (Pradaxa)- direct oral thrombin inhibitor
 - Rivaroxaban (Xarelto)- direct oral factor Xa inhibitor
 - Apixaban (Eliquis) - direct oral factor Xa inhibitor
-

Invasive AF treatment



RF ablation

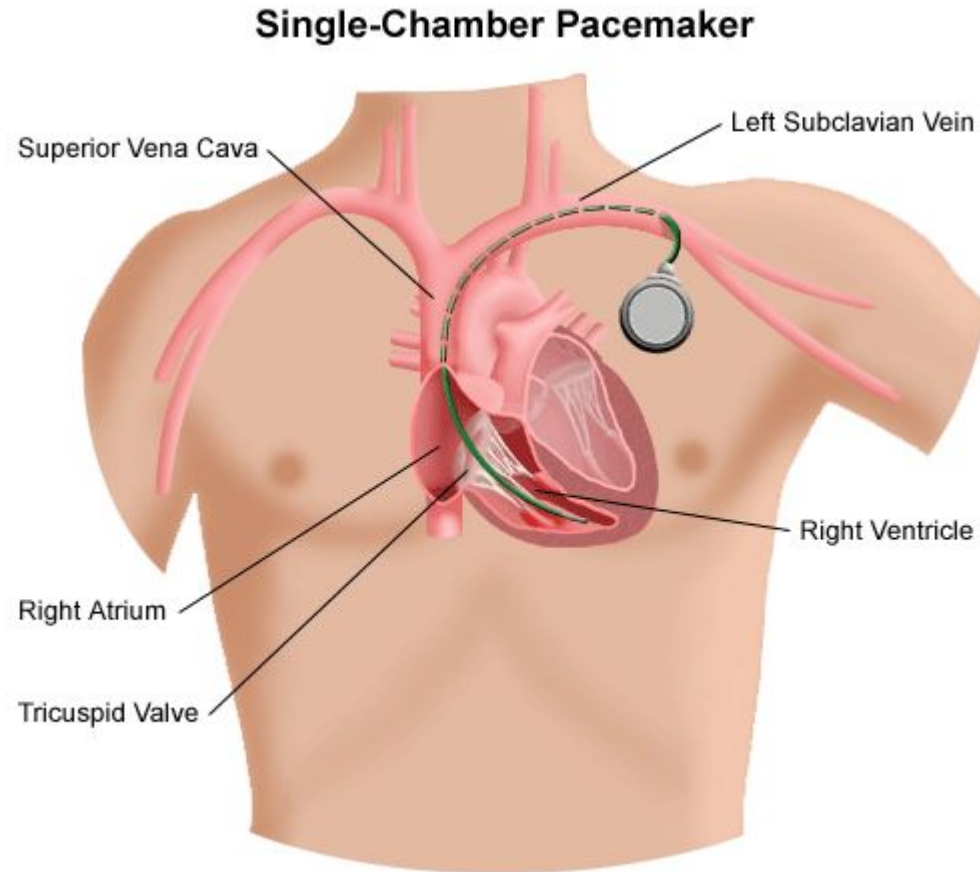


The catheter tip delivers bursts of high-energy waves that destroy the abnormal areas.

Invasive AF management

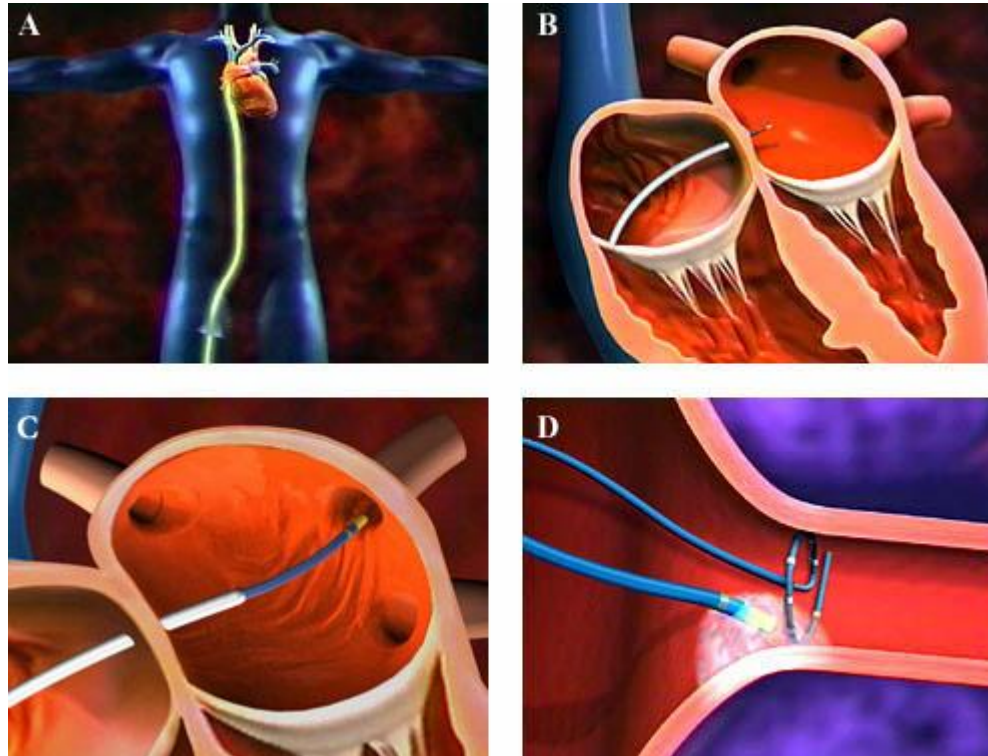
- Rate control

“Ablate and pace” –
A-v nodal ablation
& Permanent
pacemaker

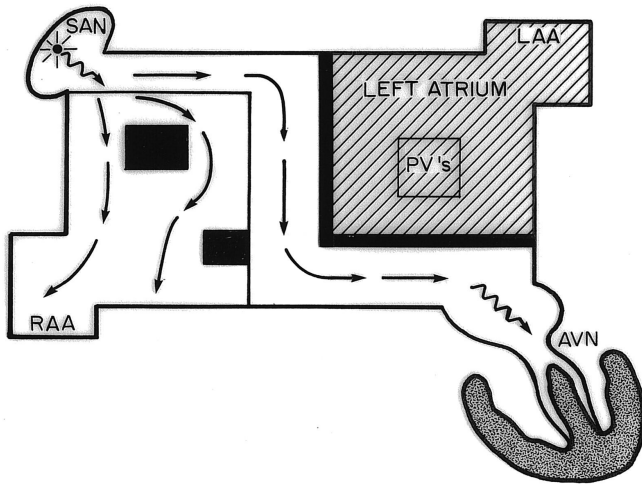


Pulmonary Venous Isolation

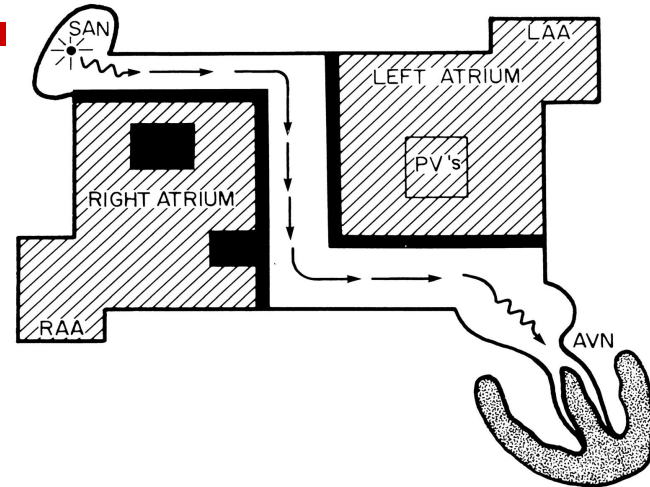
- For recurrent paroxysmal AF



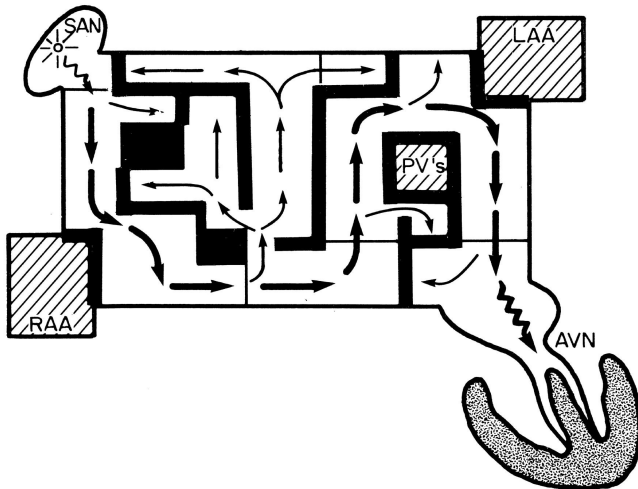
Cox-Maze Procedure



Left Atrial Isolation (1980)



Corridor Procedure (1985)



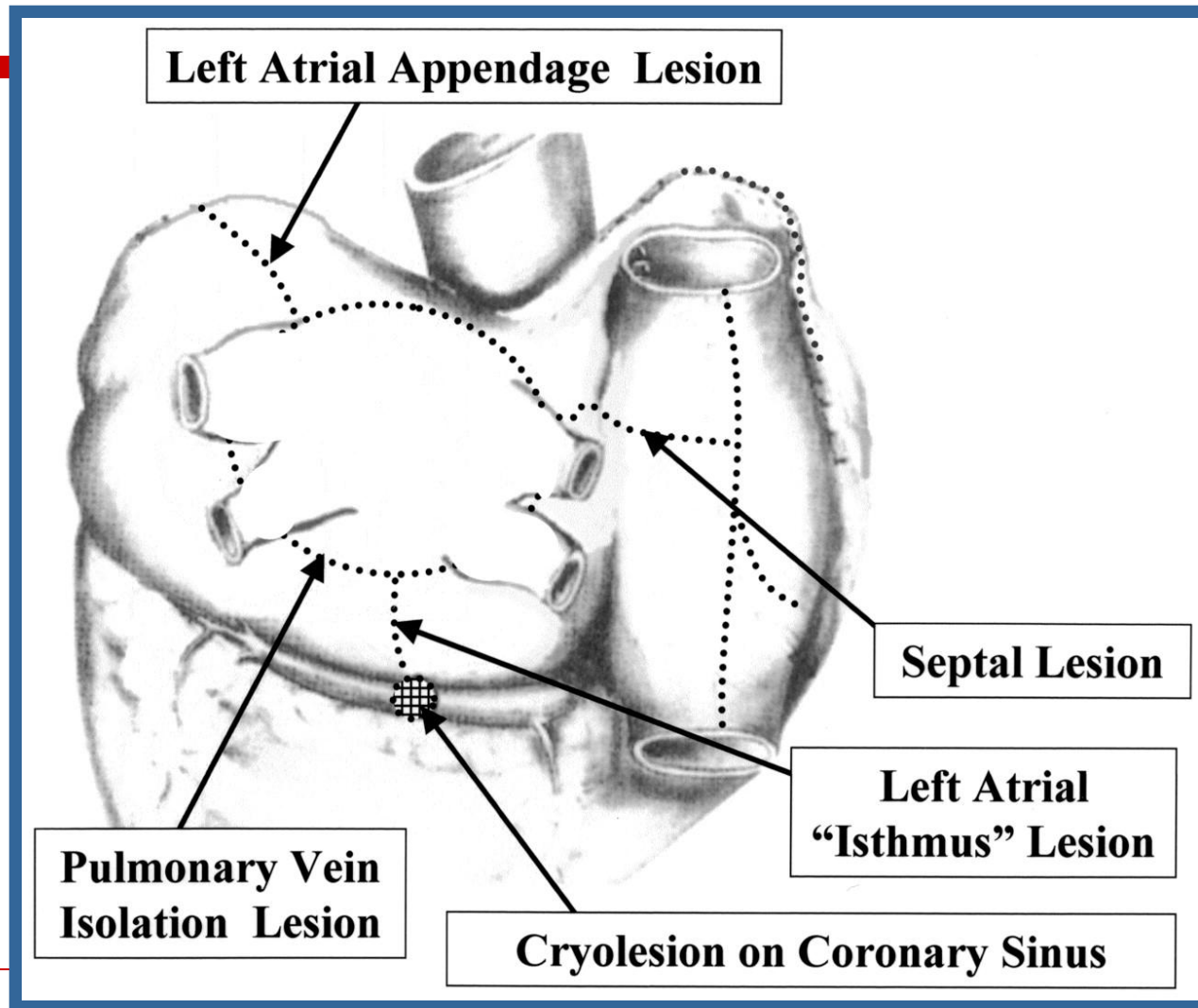
Maze Procedure (1987)

Pathway from the SA to AV Node

Disrupt Macro-reentrant Circuits

Allow Activation of All Atrial Tissue

Maze

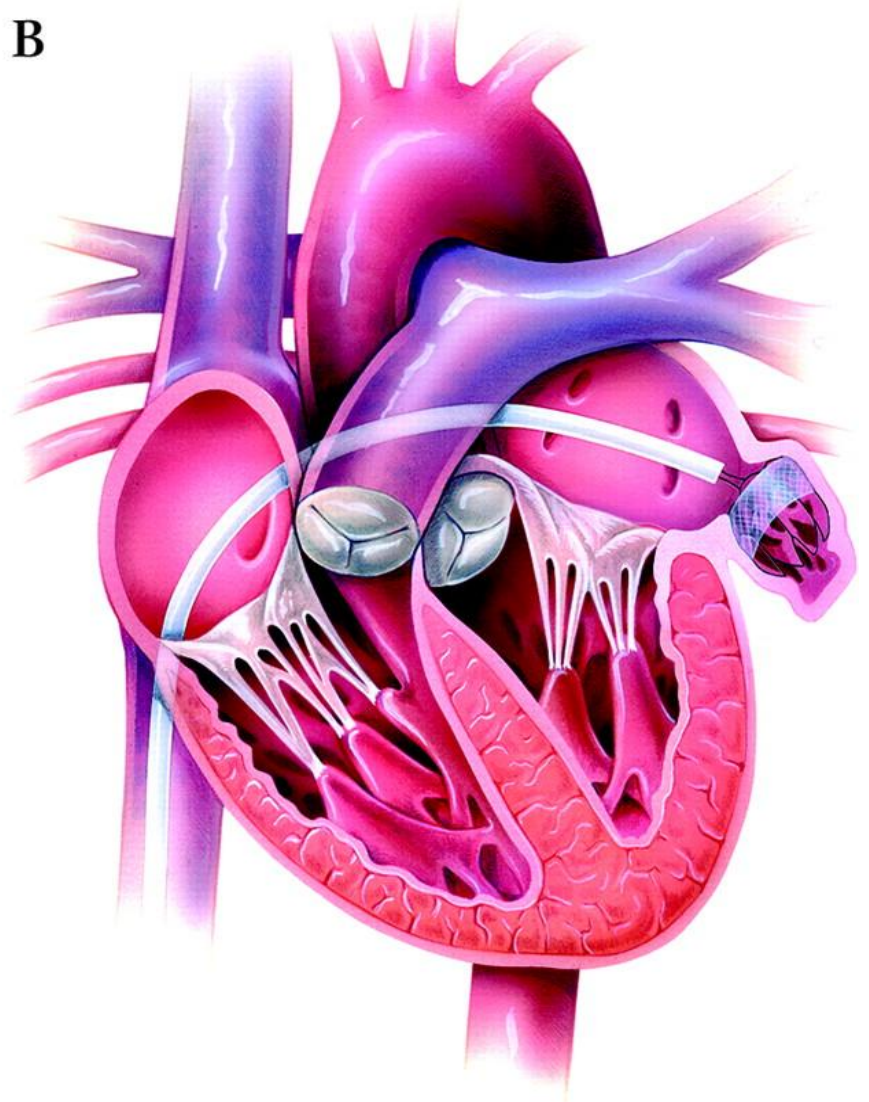


LA appendage closure

A

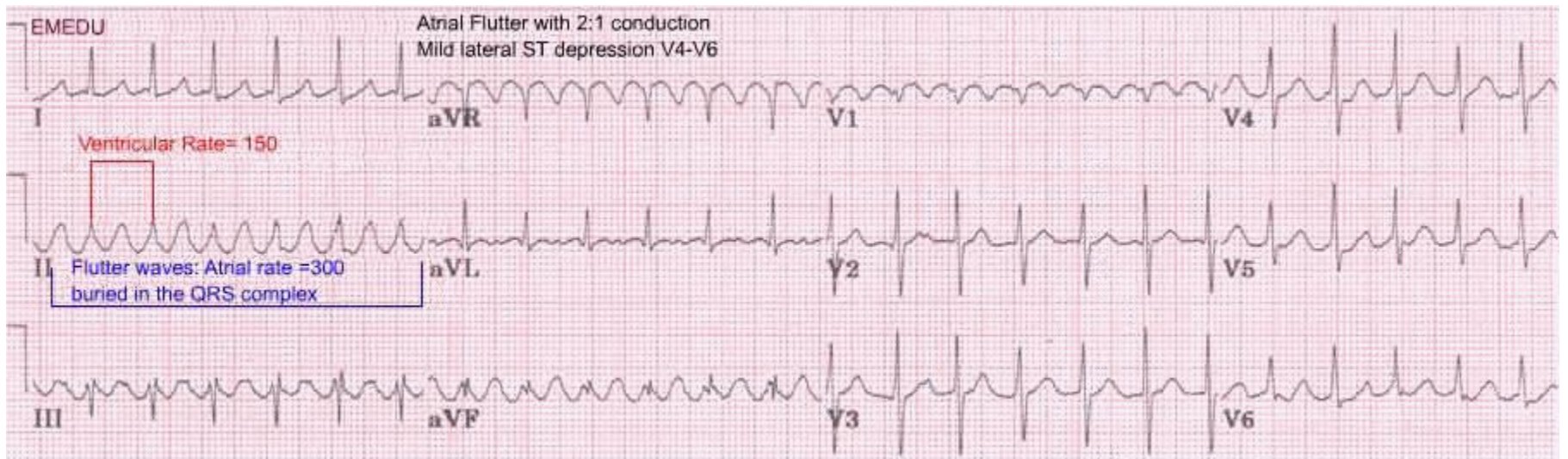


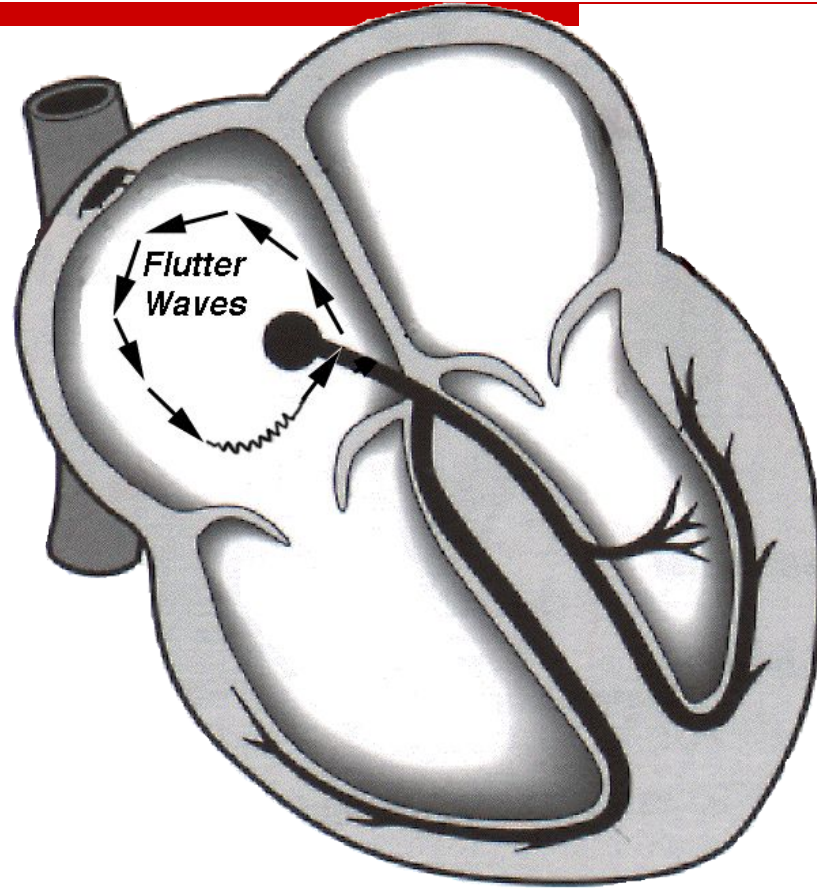
B



Atrial flutter





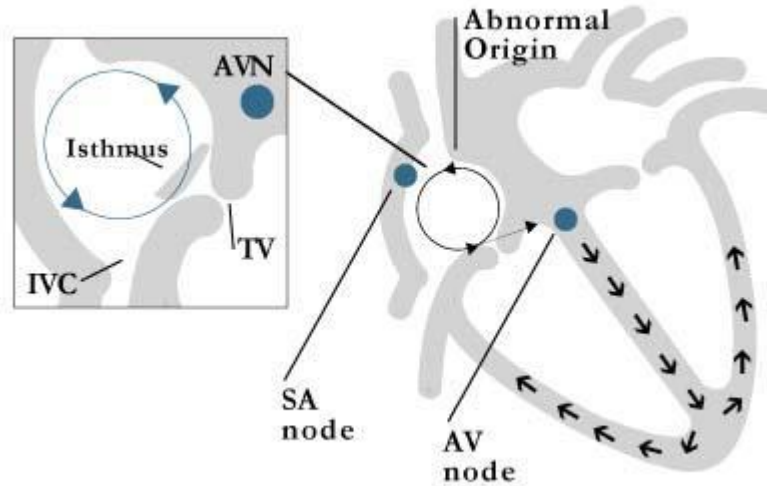


Management

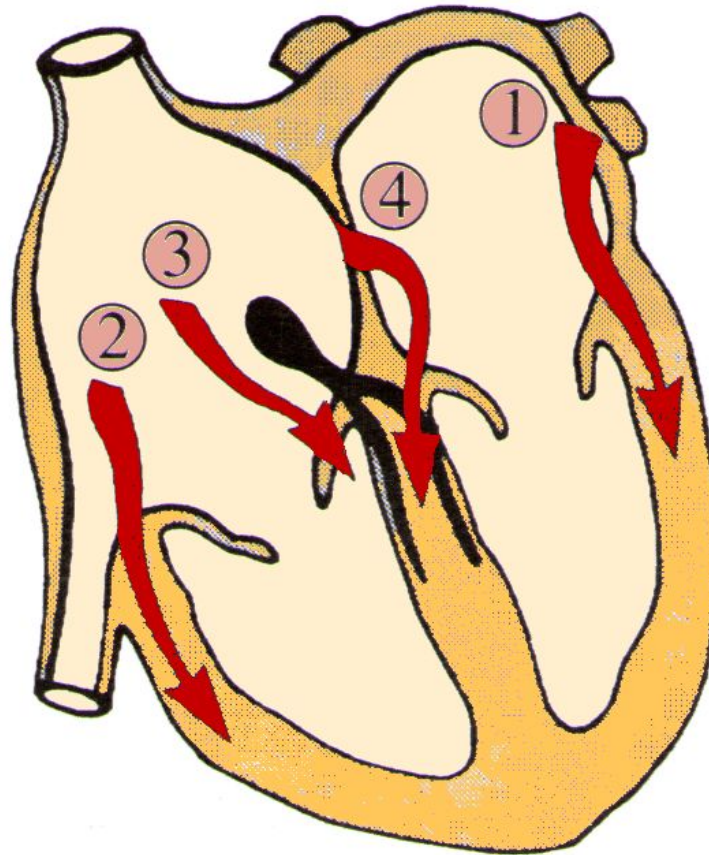
- Electric Cardioversion
 - Slowing Ventricular rate
 - Beta Blockers
 - Ca Channel blocker
 - Digoxin
 - Propafenon or Flecainaide
-

Prevention

□ Isthmus ablation

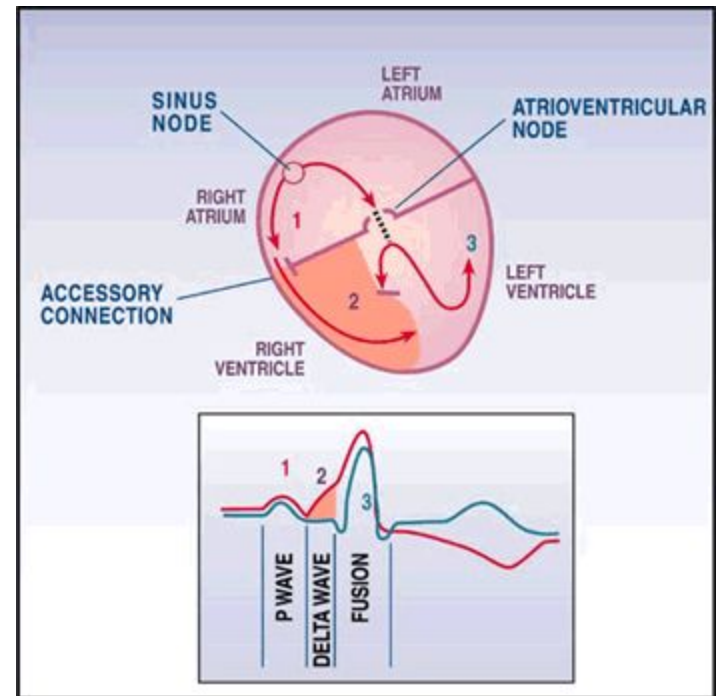


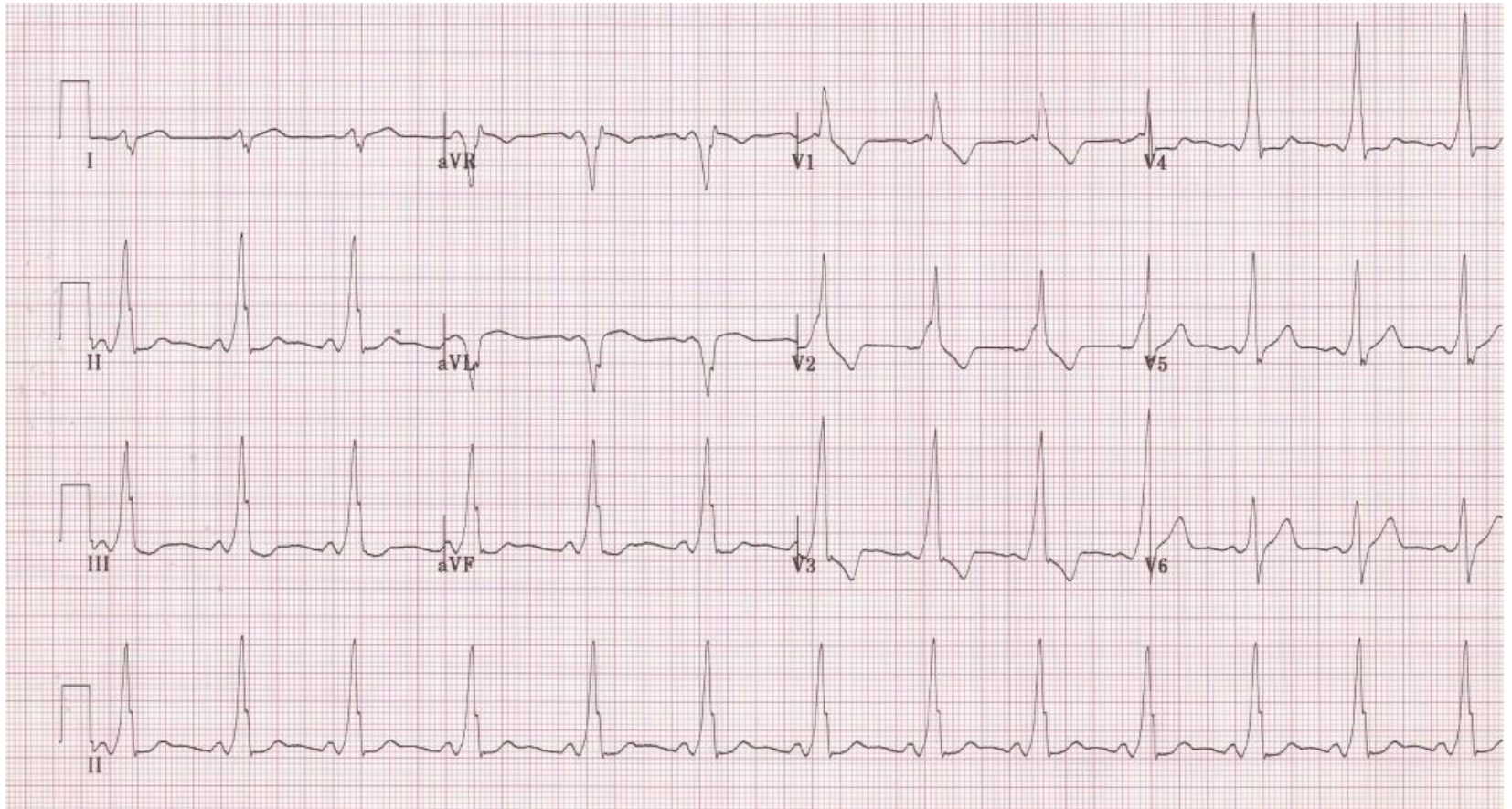
Preexcitation – WPW syndrome (accessory pathway)



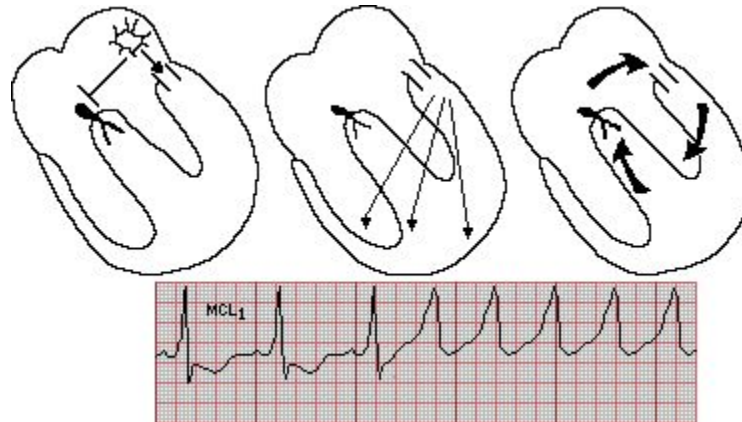
AVRT

- Short PR (<120 msec)
- Wide QRS with delta wave
- ST-T Changes

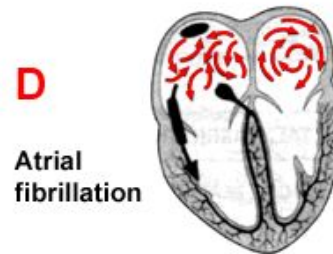
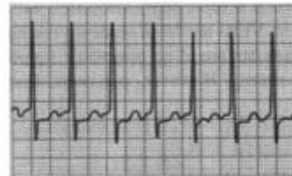
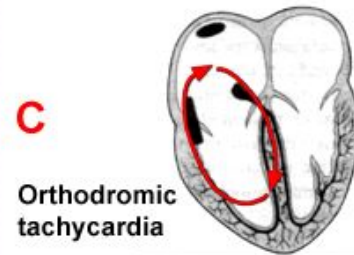
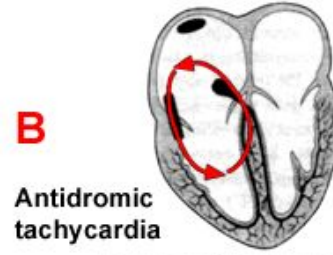
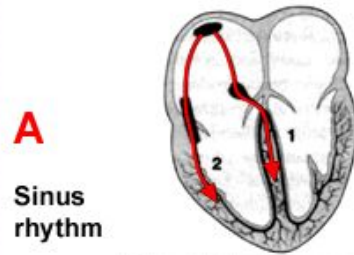




AVRT



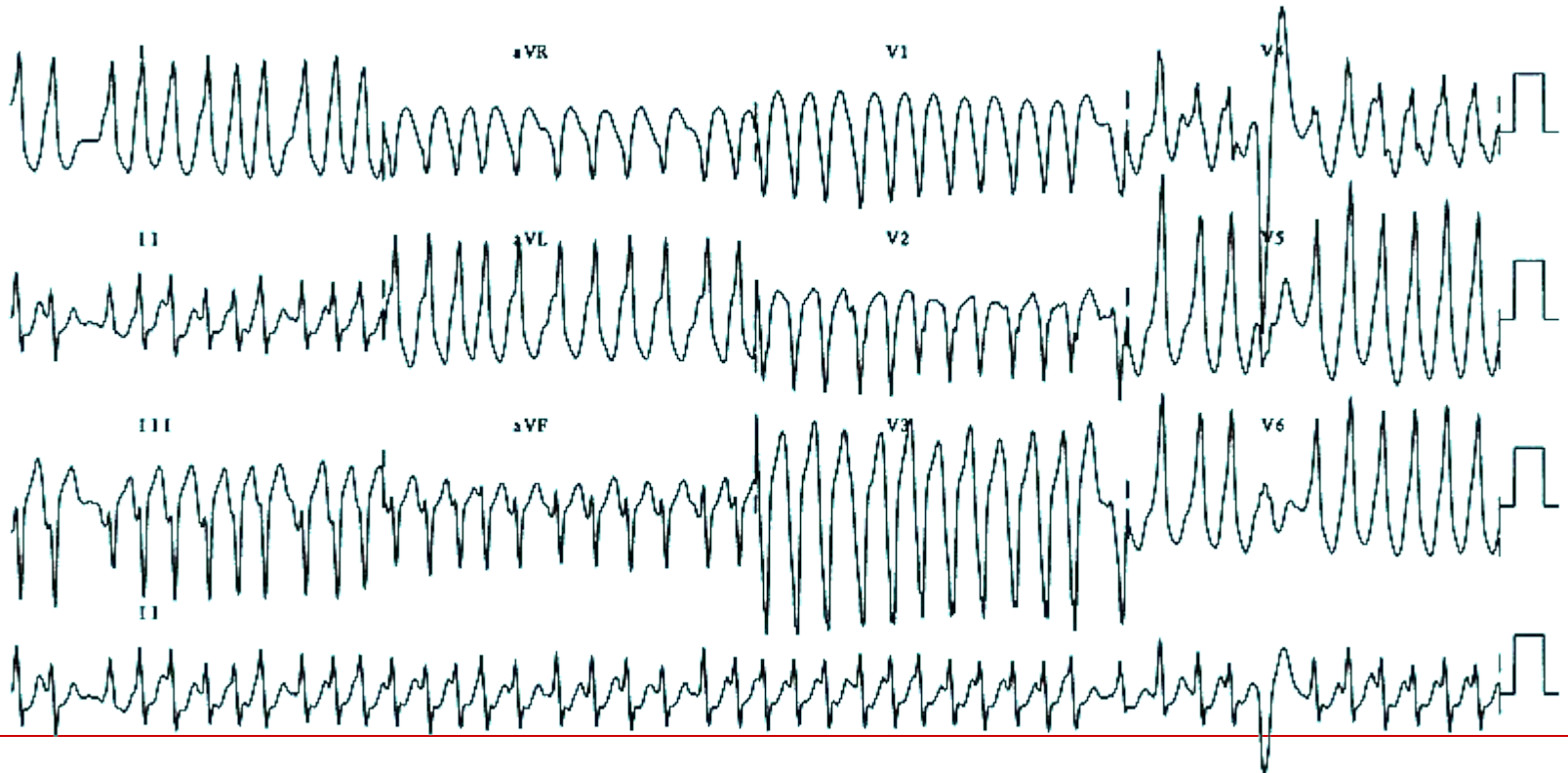
AVRT



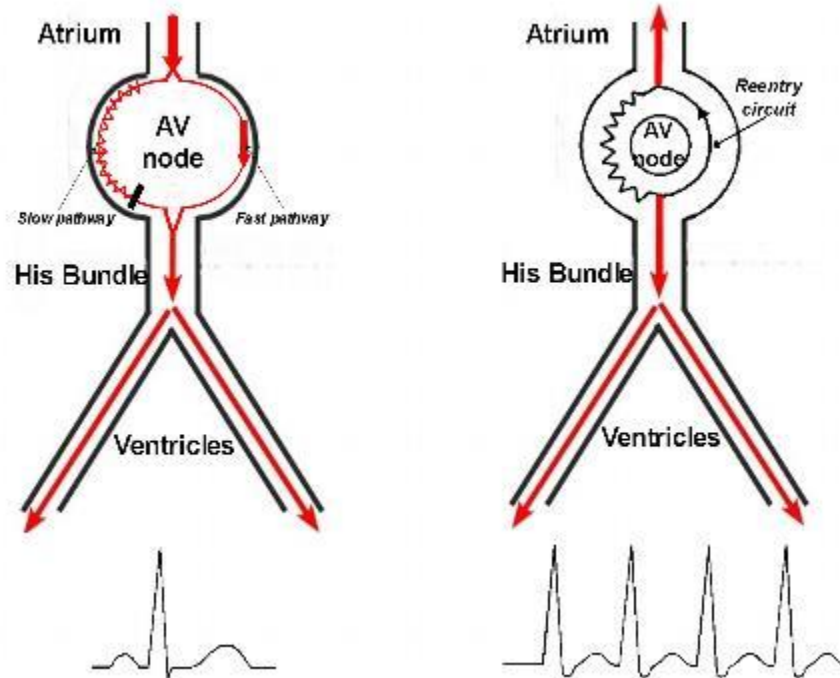
Treatment

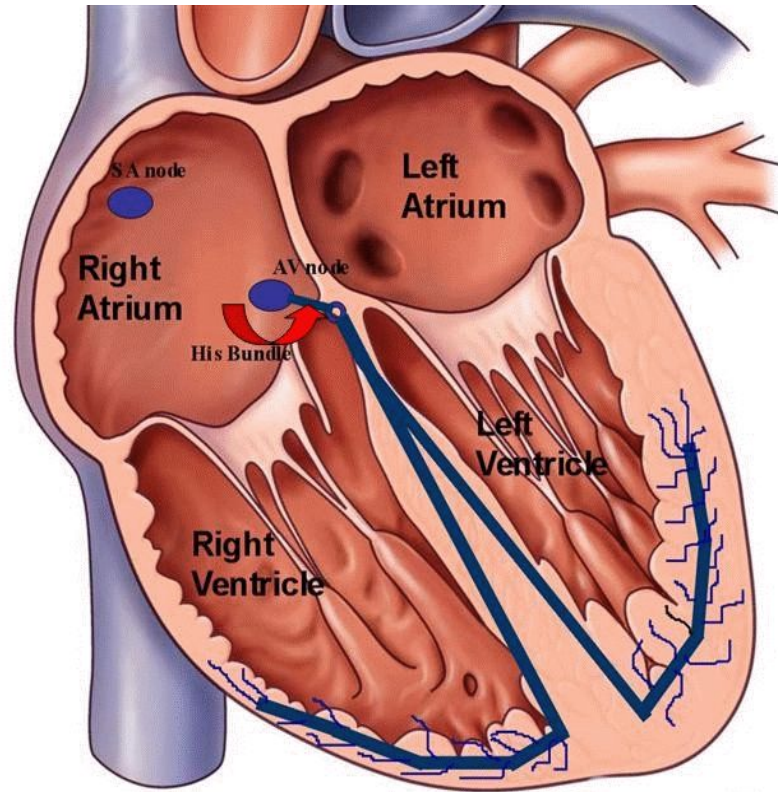
- Acute treatment:
 - Wide complex – Procainamide
 - DC Shock
 - Narrow complex – Verapamil,
 - Beta Blockers
 - Preventive treatment : accessory pathway ablation
-

□ AF with WPW – high risk of VF

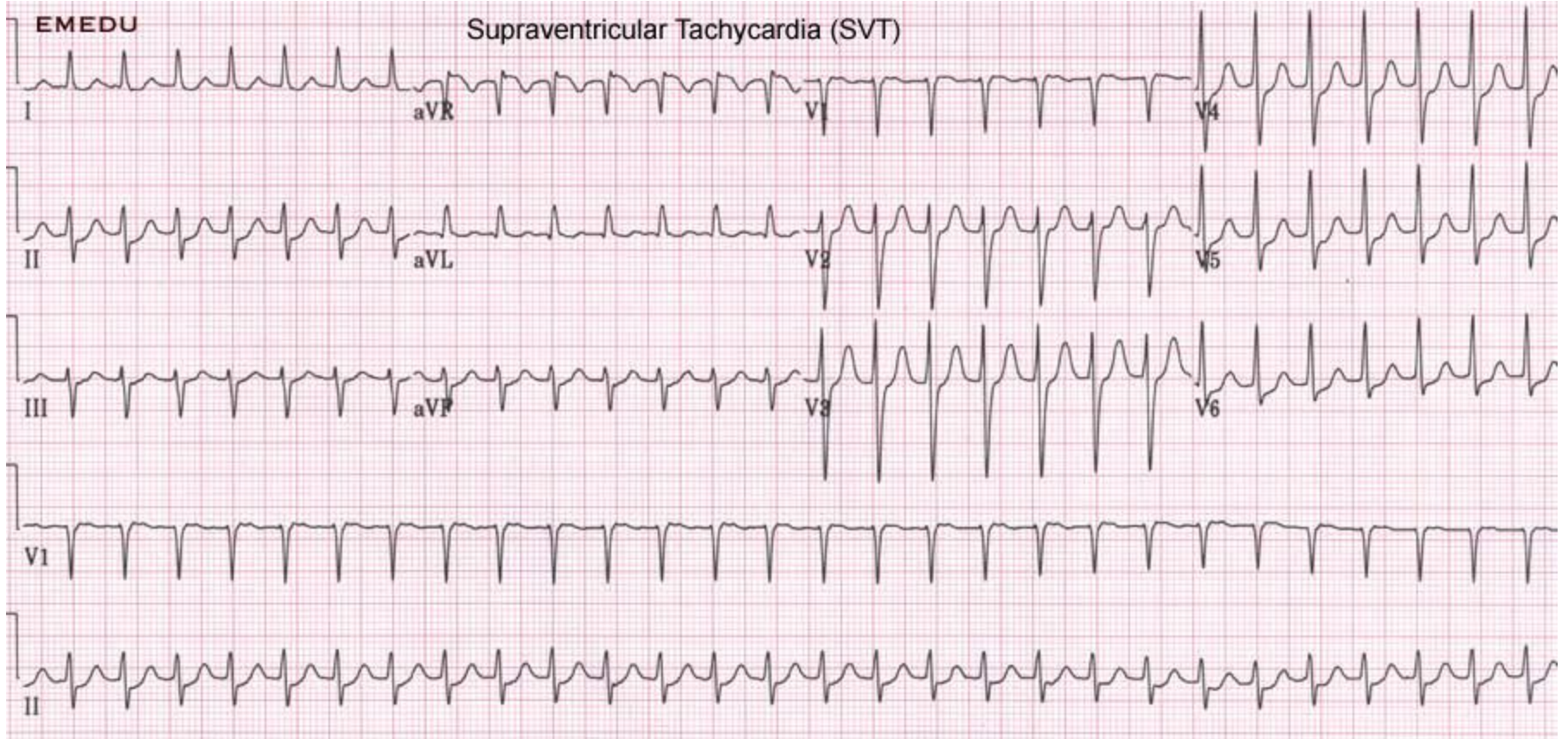


Double A-V nodal physiology





AVNRT



Management of narrow complex SVT

- If unstable – DC shock
 - If Stable :
 1. Vagal maneuvers
 2. Adenosin
 3. Verapamil
-

Preventive treatment

- Drugs
- EPS

Ventricular Arrhythmias

Ventricular premature beats

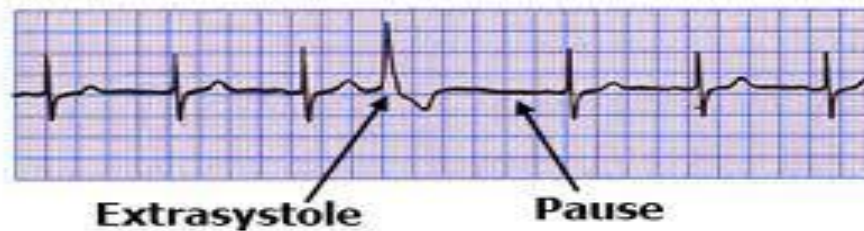
Ventricular premature complexes

- premature occurrence of a QRS complex that is abnormal in shape and has a duration usually exceeding the dominant QRS complex, generally longer than 120 milliseconds.
- The T wave is usually large and opposite in direction to the major deflection of the QRS.
- The QRS complex is not preceded by a premature P wave



Compensatory pause

Ventricular Extrasystole

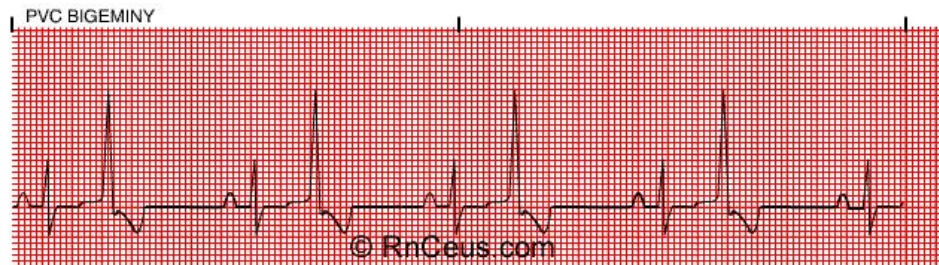
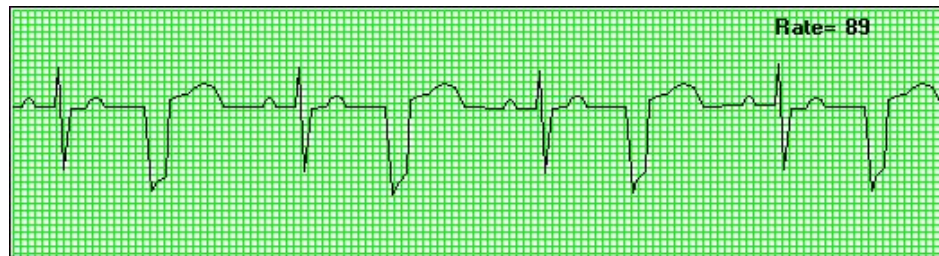


Interpolated PVC

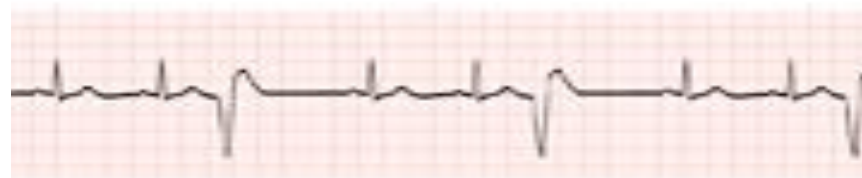
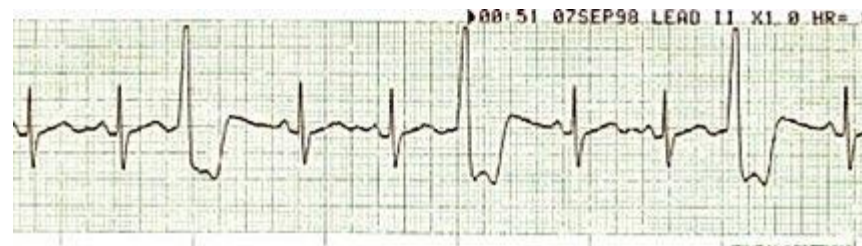


Note that there is no compensatory pause with an interpolated PVC.

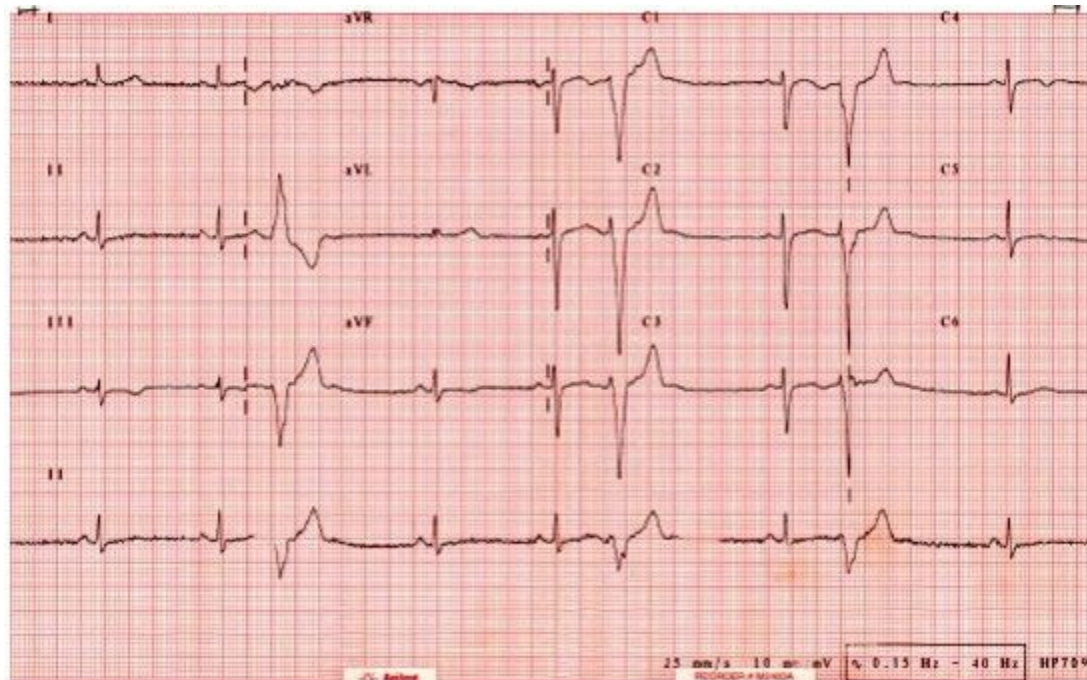
Bigeminy



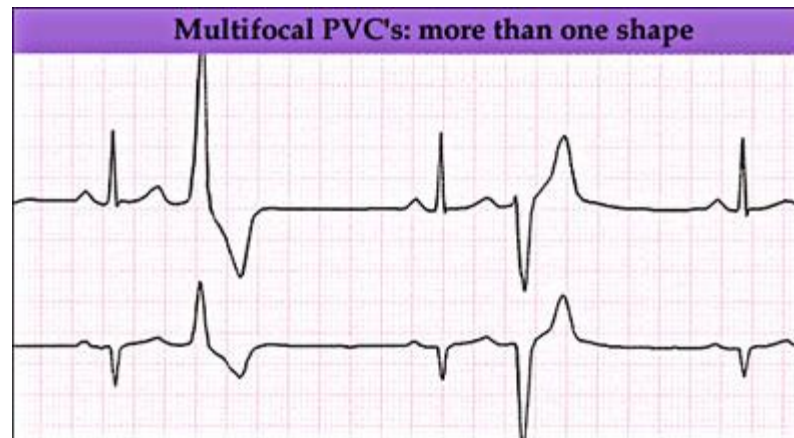
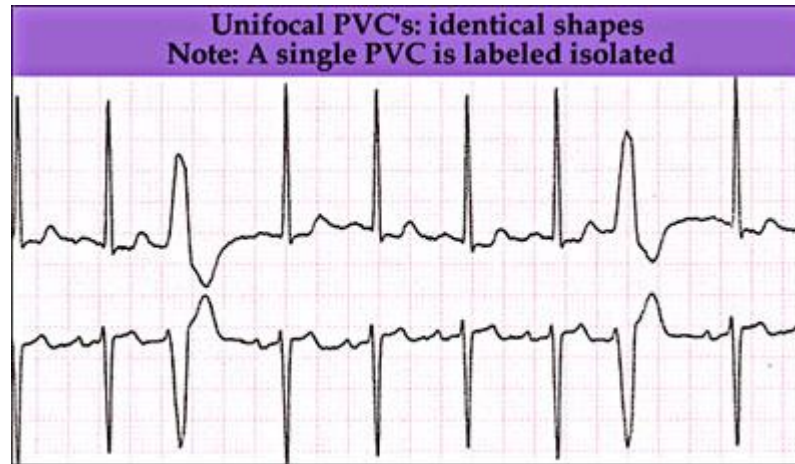
Trigeminy



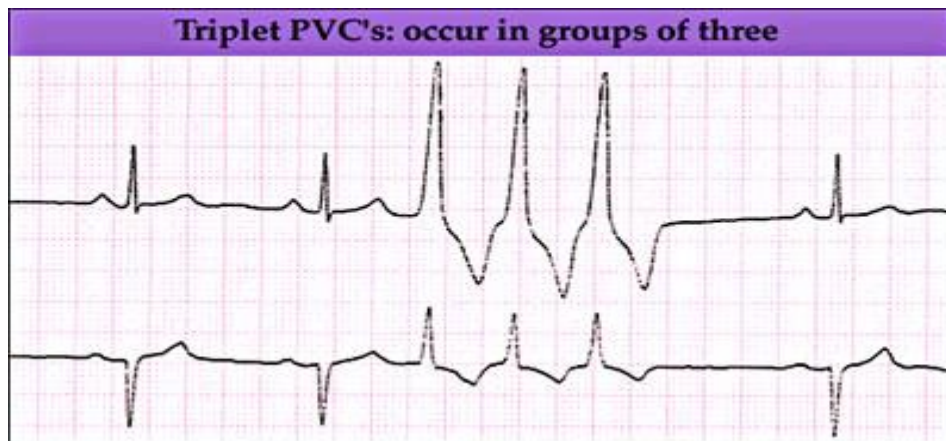
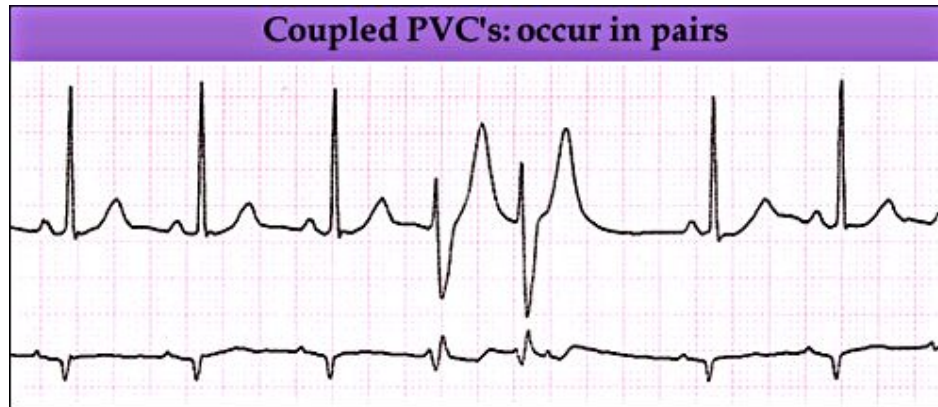
VPB's



Unifocal & Multifocal



Couplet & Triplet



Causes

- LV false tendons,
 - infection
 - in ischemic or inflamed myocardium,
 - hypoxia,
 - Anesthesia or
 - surgery.
 - Medications
 - electrolyte imbalance,
 - tension states,
 - myocardial stretch,
 - excessive use of tobacco, caffeine, or alcohol.
-

Complex Ventricular Arrhythmia

- **Nonsustained ventricular tachycardia (VT)**
 - ♥ **Monomorphic**
 - ♥ **Polymorphic**
 - **Sustained VT**
 - ♥ **Monomorphic**
 - ♥ **Polymorphic**
 - **Torsades de pointes**
 - **Ventricular fibrillation**
-

VT

Definition:

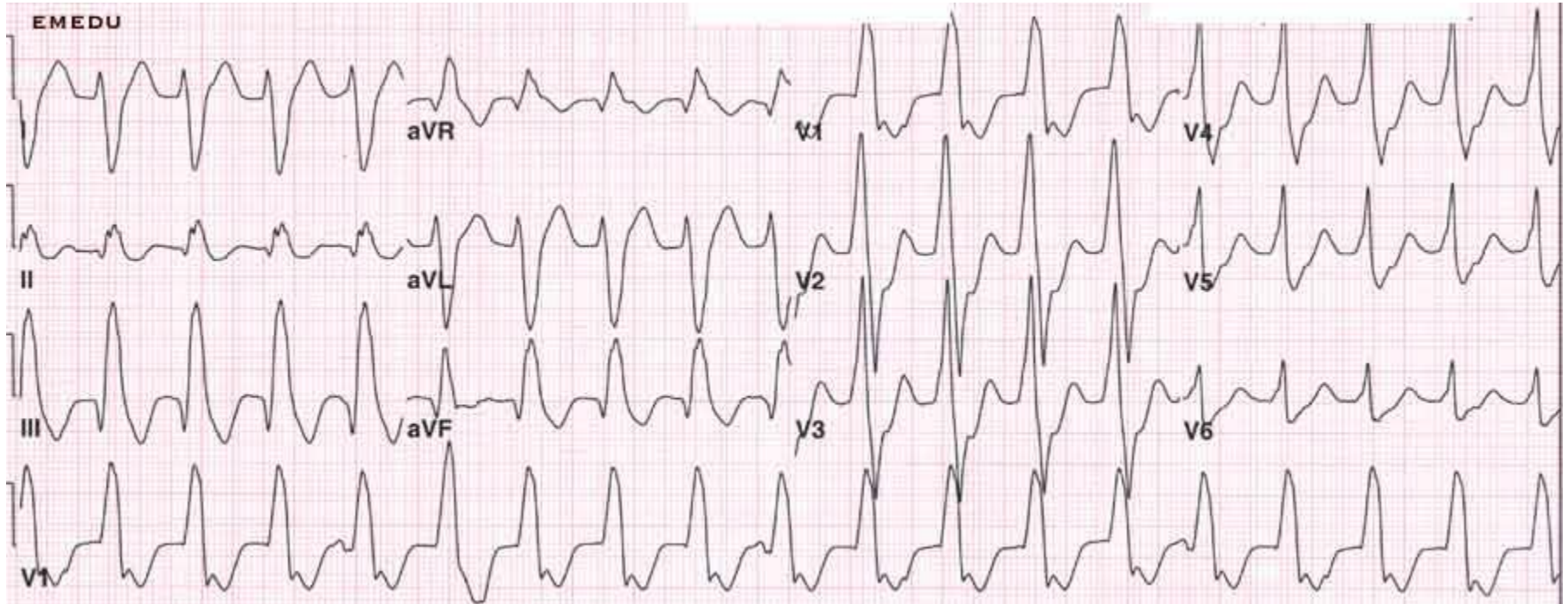
Ventricular tachycardia consist of at least three consecutive QRS complexes originating from the ventricles and recurring at a rapid rate (> 100 bpm).

Sustained ventricular tachycardia is arbitrarily defined as lasting ≥ 30 seconds.

The rhythm is generally regular or slightly irregular.



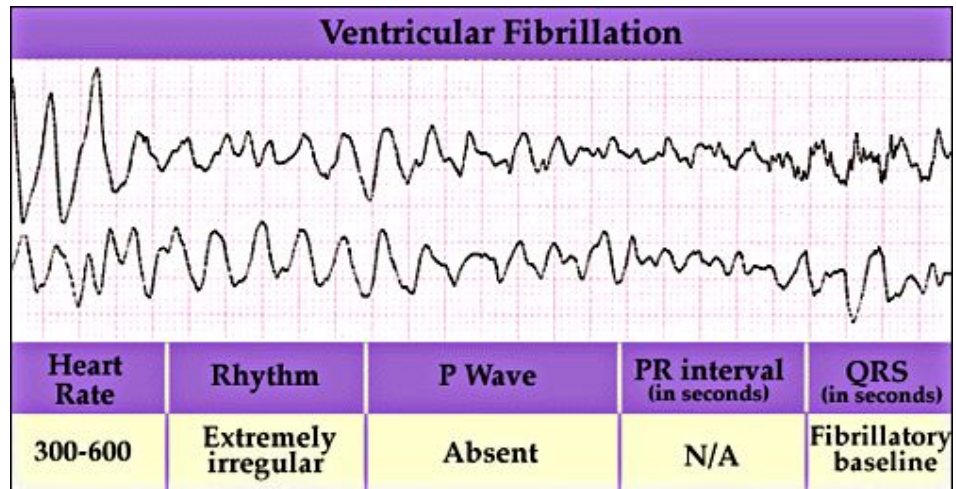
VT - monomorphic



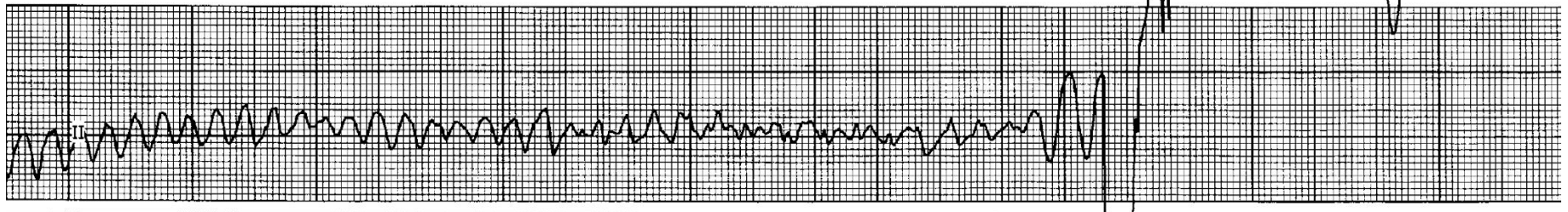
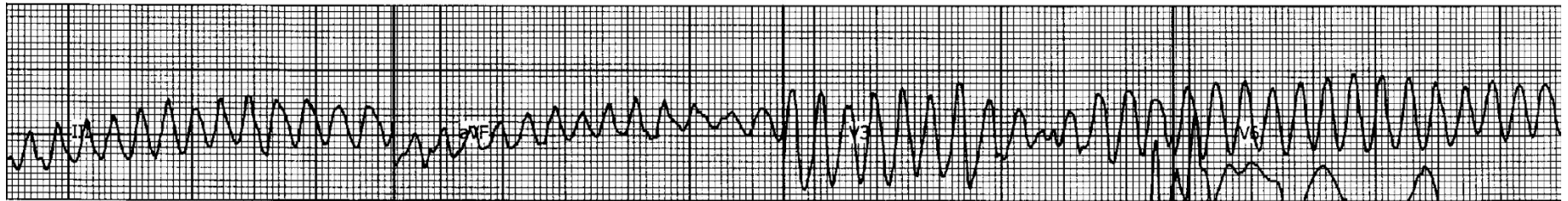
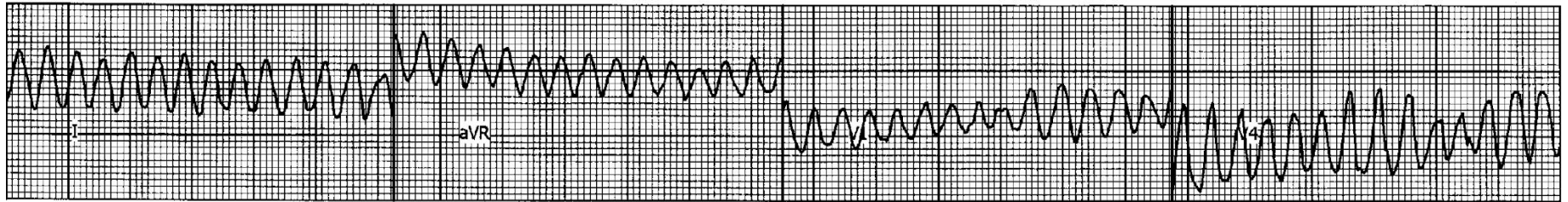
Sustained Polymorphic VT



VF



VF with Defibrillation (12-lead ECG)



Causes

- Chronic coronary heart disease
- Heart failure
- Congenital heart disease
- Neurological disorders
- Structurally normal hearts
- Sudden infant death syndrome
- Cardiomyopathies
 - ♥ Dilated cardiomyopathy
 - ♥ Hypertrophic cardiomyopathy
 - ♥ Arrhythmogenic right ventricular (RV) cardiomyopathy

Mechanisms of Sudden Cardiac Death

- Ventricular fibrillation - 62.4%
- Bradyarrhythmias (including advanced AV block and asystole) - 16.5%
- Torsades de pointes - 12.7%
- Primary VT - 8.3%

VA management

- Acute
 - Chronic (secondary prevention)
-

Sustained VT

□ **Hemodynamically stable:**

- Amiodaron
- Lidocain
- Procainamide

If pharmacotherapy ineffective – DC shock
(synchronized)

Ventricular pacing

□ **Hemodynamically unstable – Immediate DC shock**

Polymorphic VT

- Polymorphic VT with long QT –
Torsades de pointes
Treatment – Mg , Pacing
 - Polymorphic VT w/o long QT
Antyarrhythmic drugs
-

CPR Quality

- Push hard (≥ 2 inches [5 cm]) and fast (≥ 100 /min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
 - If $PETCO_2 < 10$ mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
 - If relaxation phase (diastolic) pressure < 20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in $PETCO_2$ (typically ≥ 40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

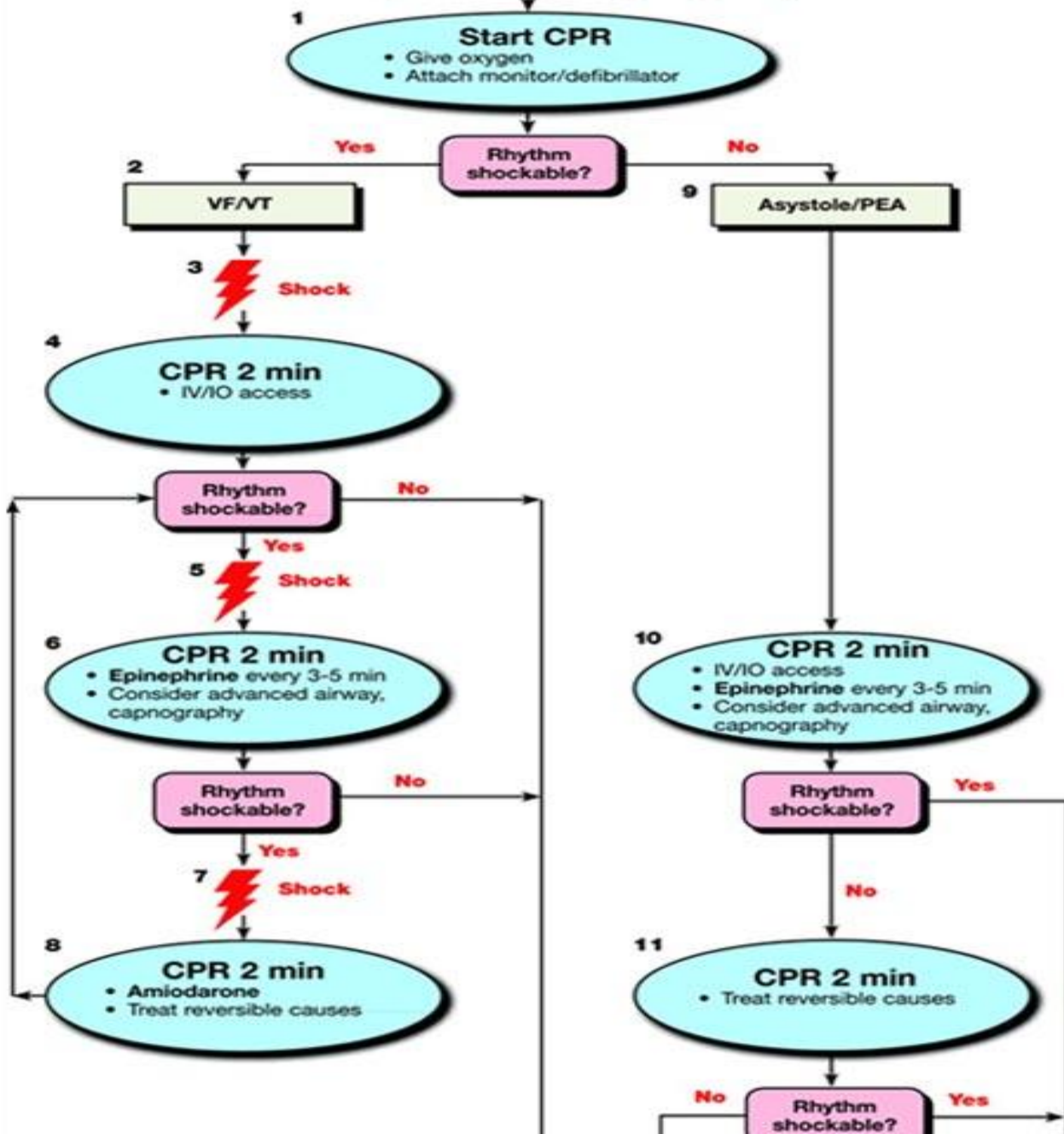
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway

- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Adult Cardiac Arrest

Shout for Help/Activate Emergency Response



Chronic Management (secondary prevention)

Evaluation

- Rest ECG
 - Exercise test
 - Ambulatory ECG
 - Imaging (LV function, CMP, Valves etc...)
 - EPS
-

Treatment of the underlying disease

- Revascularisation
 - Valve surgery
 - CHD repair
-

Non-antiarrhythmic Drugs

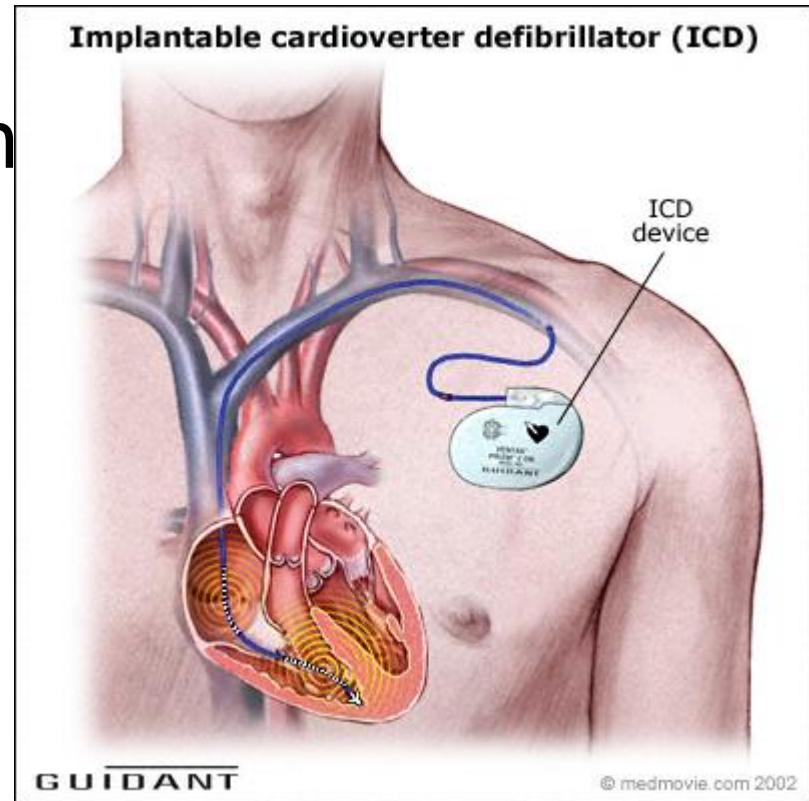
- ♥ Electrolytes: Mg & K
- ♥ ACE inhibitors,
- ♥ Antithrombotic and antiplatelet agents
- ♥ Statins

Antiarrhythmic drugs

- Antiarrhythmic drugs (except for BB) should not be used as *primary* preventive therapy of VA and the prevention of SCD
-

Invasive treatment

- AICD
- EPS with ablation
- Surgical ablation

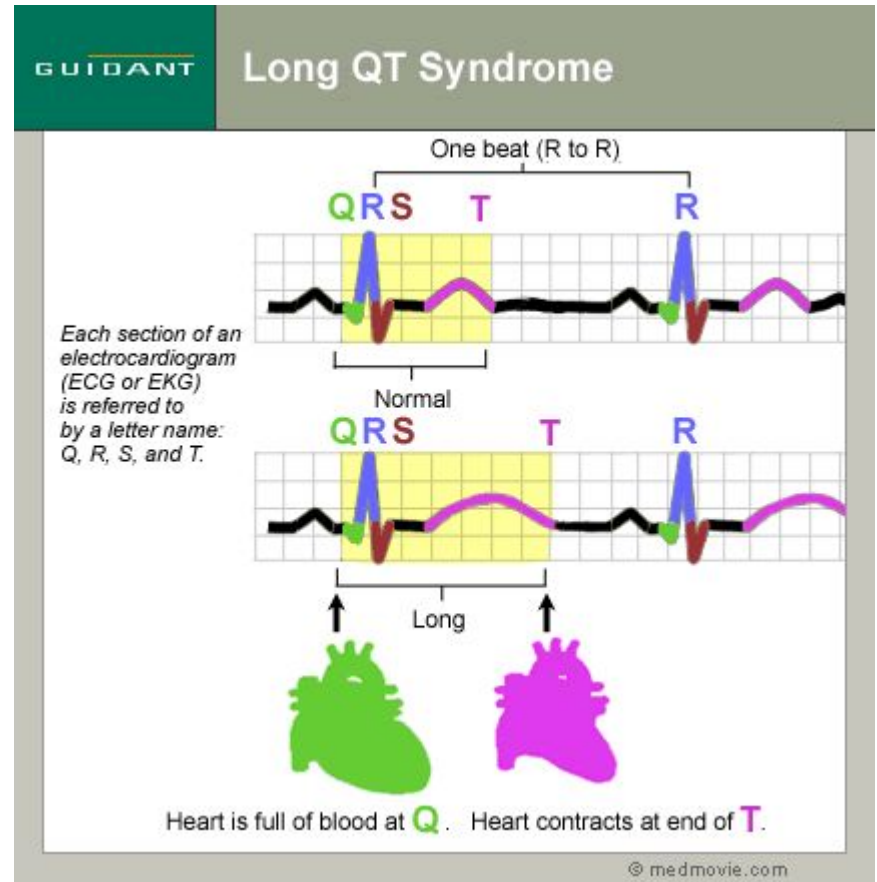


AICD for primary prevention of SCD

- 1. Post MI
 - LVEF < 30%
 - LVEF 30-35%, NYHA II-III
 - LVEF 30-40%, NSVT, positive EP
 - 2. Non ischemic CMP
 - LVEF < 30%
-

Long QT syndrome

1. Congenital (family)
2. Acquired:
 - Electrolyte anomalies – K, Mg
 - Drug induced
 - Antiarrhythmics
 - Tricyclic antidepressants
 - Antihistamines
 - CNS lesions



Torsades de Pointes



outline looks like a party streamer



Long QT syndrome treatment

□ Acute

1. Remove the precipitating factor
 2. Mg IV
 3. Pacing
 4. Isoproterenol
 5. IB antiarrhythmic
-

Long QT syndrome treatment

- **Chronic** – for congenital long QT
 1. Beta blockers
 2. AICD
-

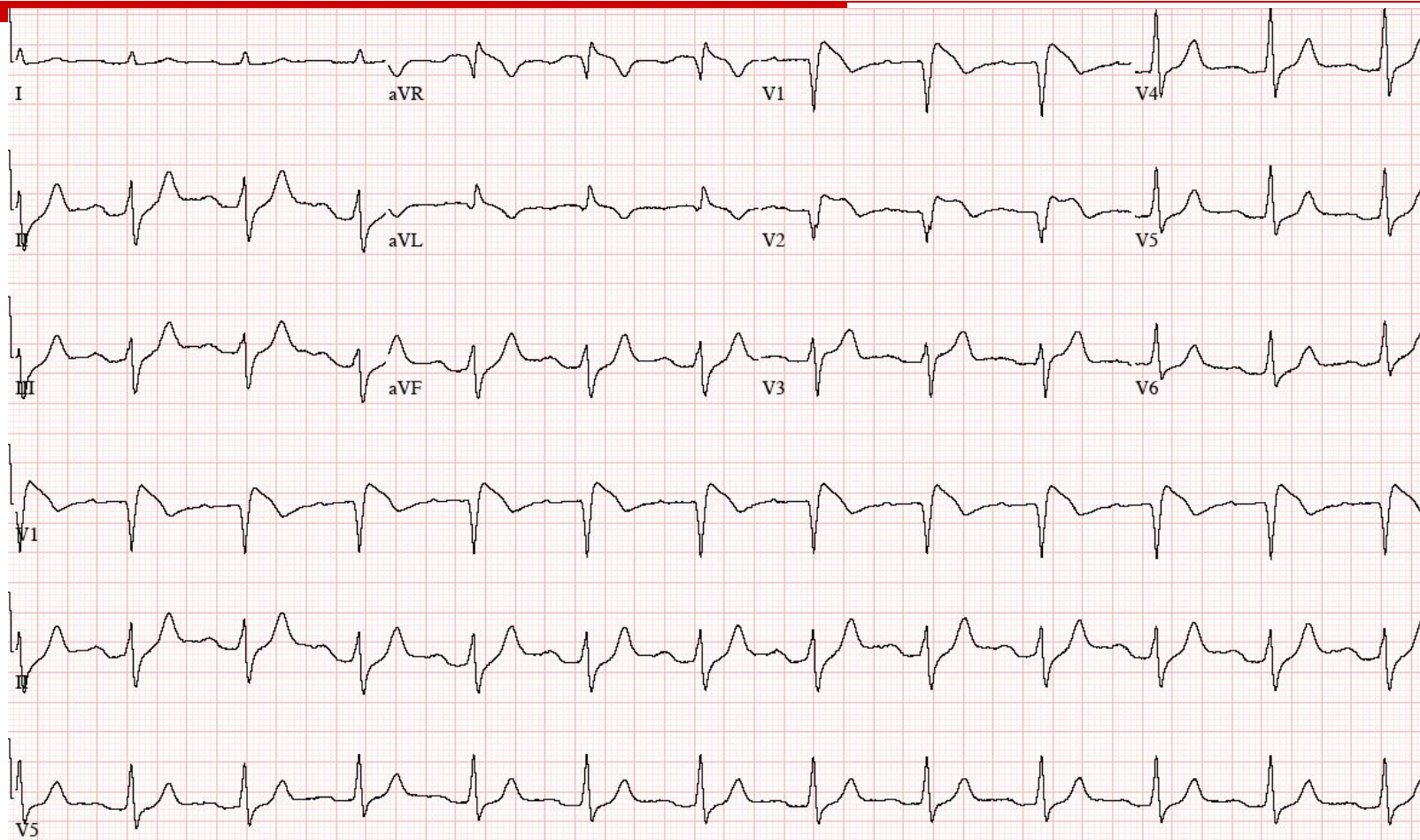
The Brugada Syndrome

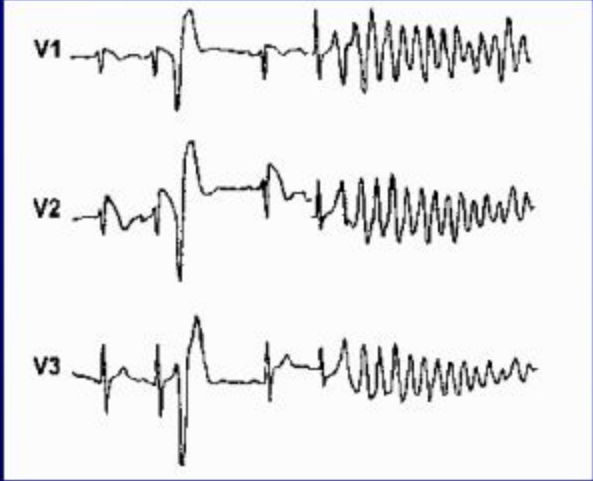
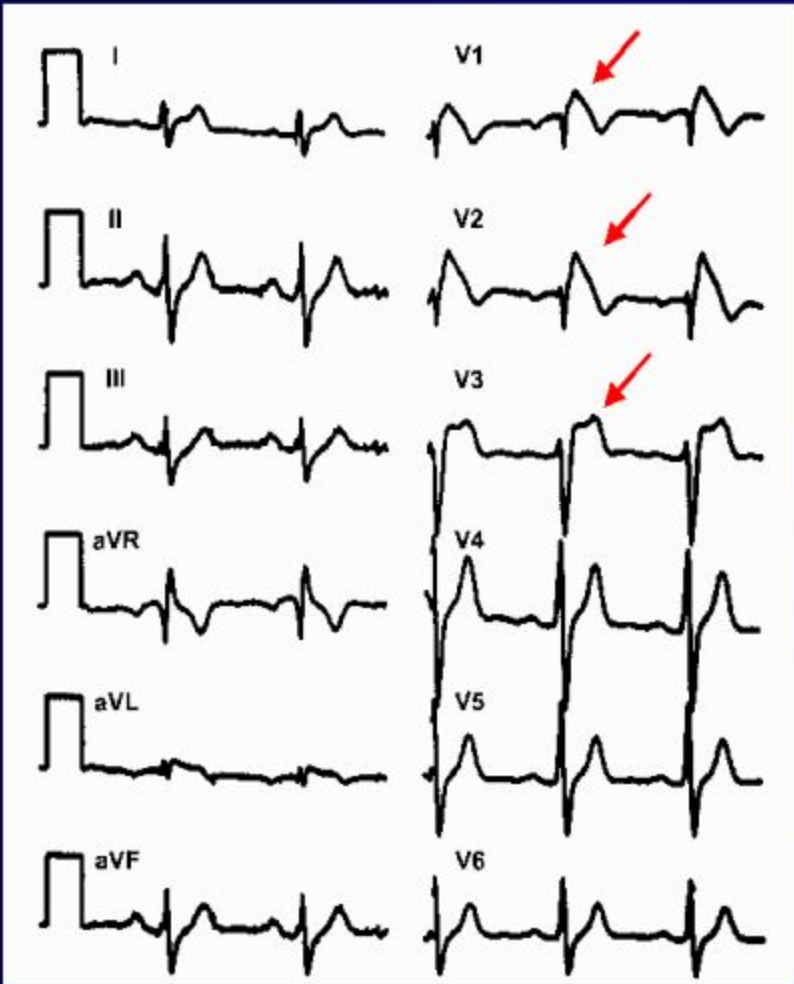
Definition

“Syncopal episodes and/or sudden death in patients with a structurally normal heart and a characteristic electrocardiogram displaying a pattern resembling right bundle branch block with an ST segment elevation in leads V1 to V3”

Brugada et al. Circ 1992

Brugada syndrome

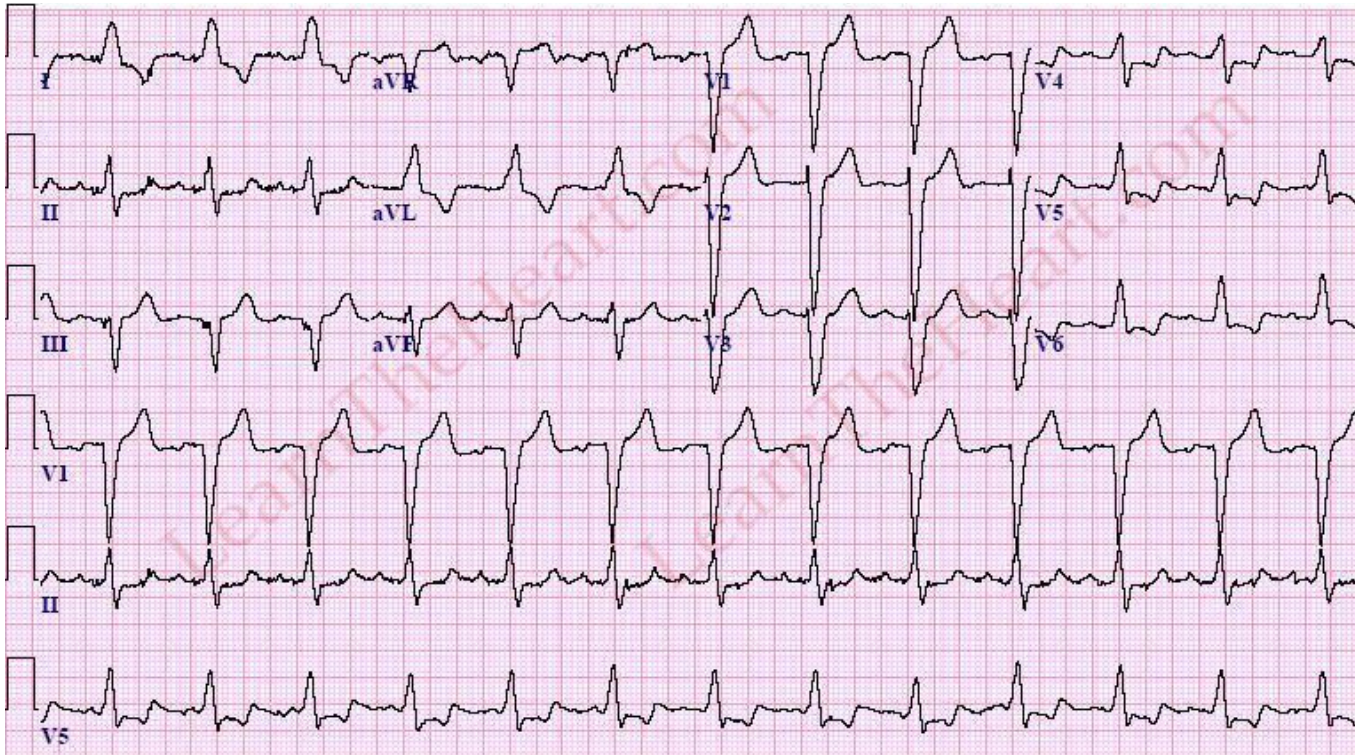




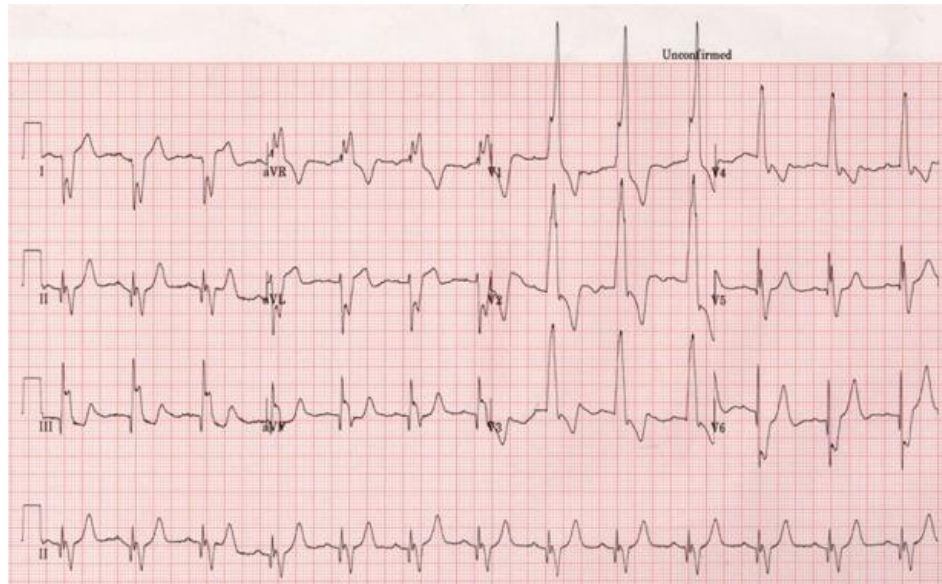
- **Typical presentation**

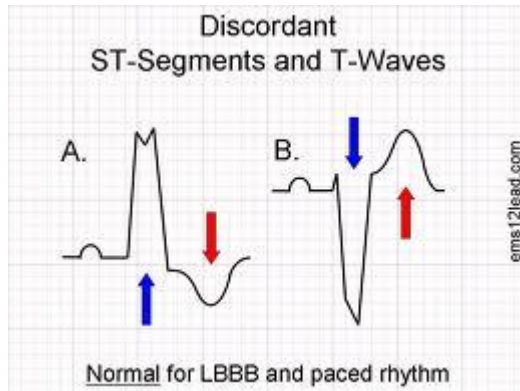
Middle-aged male with the typical ECG pattern, no structural heart disease, recovered from sudden cardiac death due to VF and with a previous history of syncopal episodes due to self-terminating rapid polymorphic VT.

CLBBB



CRBBB





**Right bundle branch block
characteristics**



"Wide Complex Tachycardia"

□ **VT**

□ **SVT with**

Preexistent BBB

Rate dependent
BBB

Preexcitation

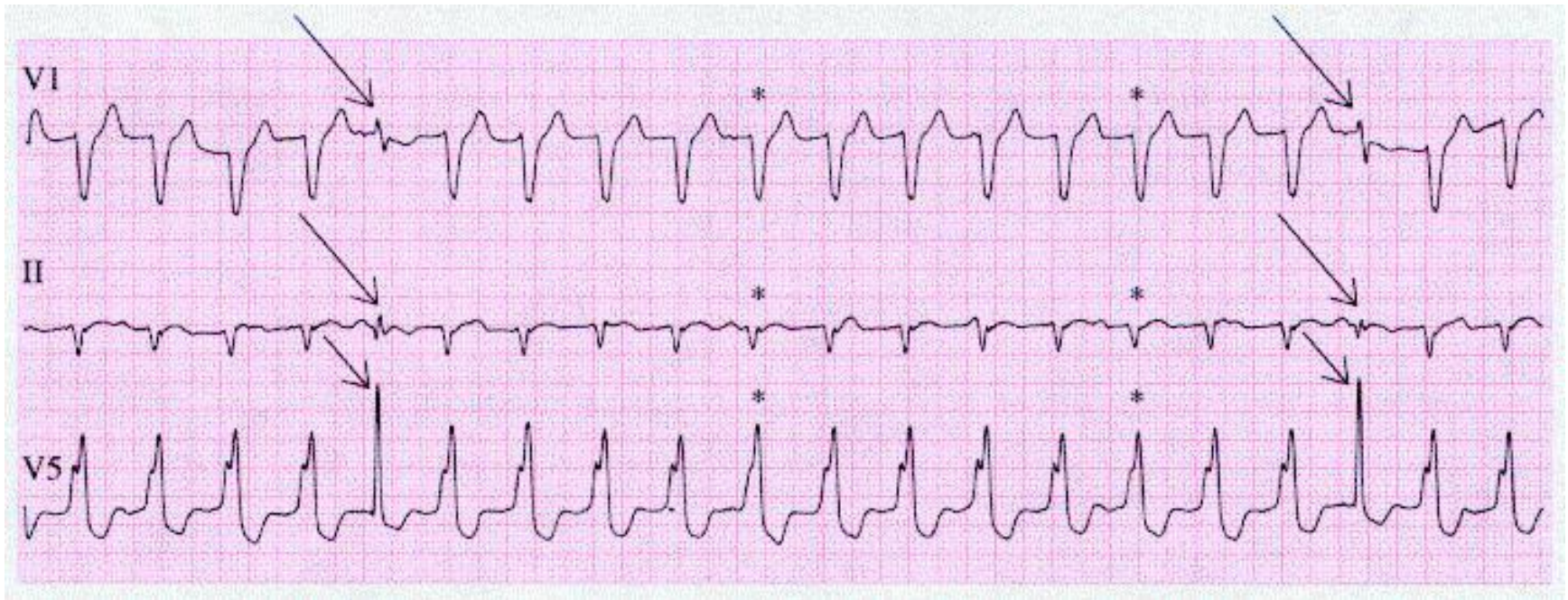


Wide QRS Irregular Tachycardia: Atrial Fibrillation with antidromic conduction in patient with accessory pathway – Not VT



Futures favoring VT

- 1) **AV Dissociation**
 - 2) **QRS > 0.14**
 - 3) **QRS Axis between – 90 & - 180 degrees**
 - 4) **Positive QRS deflection in all precordial leads**
 - 5) **LBBB morphology with rightward QRS axis**
 - 6) **Capture beats, fusion beats**
 - 7) **QRS morphology identical to PVC's during sinus rhythm**
-

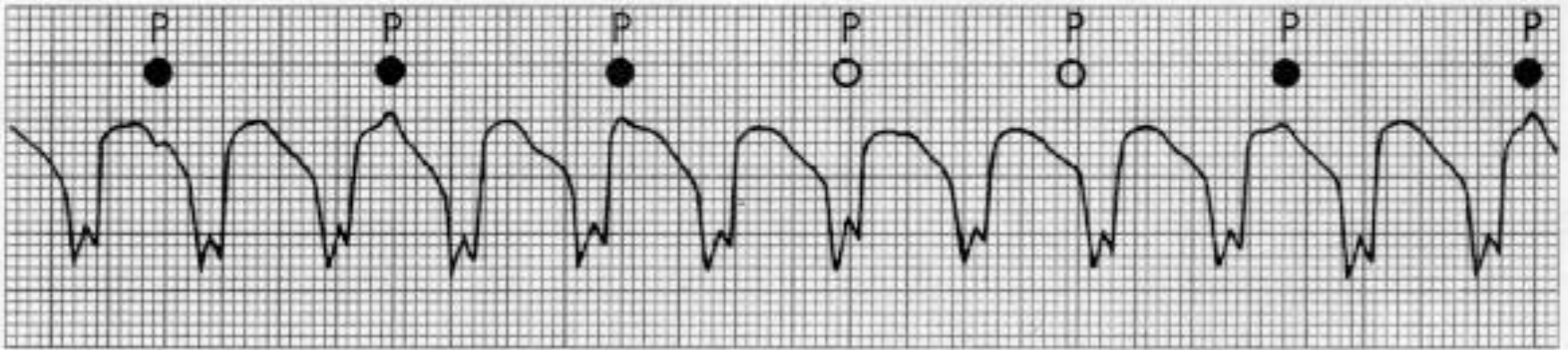


Fusion and Capture Beats

A three-lead rhythm strip from a 62-year-old man who presented with acute shortness of breath 2 months after an inferior-posterior MI. *Arrows* indicate capture beats and *asterisks* indicate fusion beats.

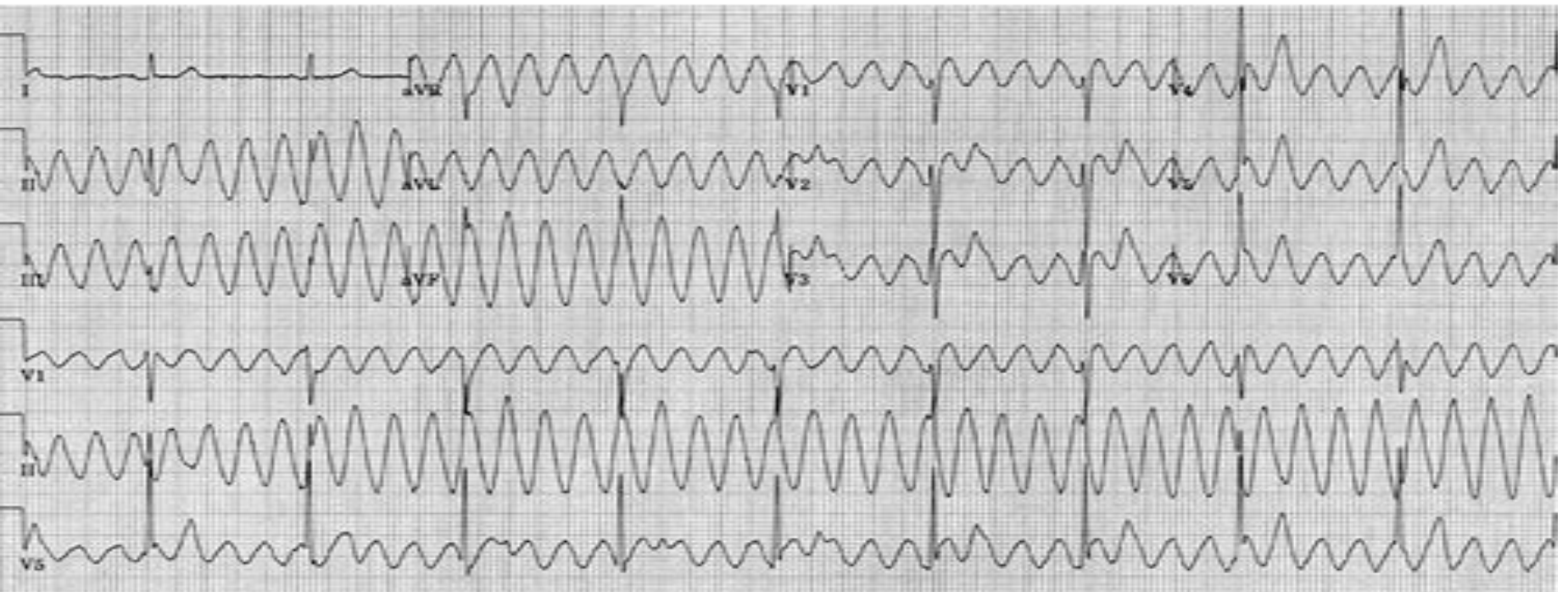
Ventricular Tachycardia: AV Dissociation

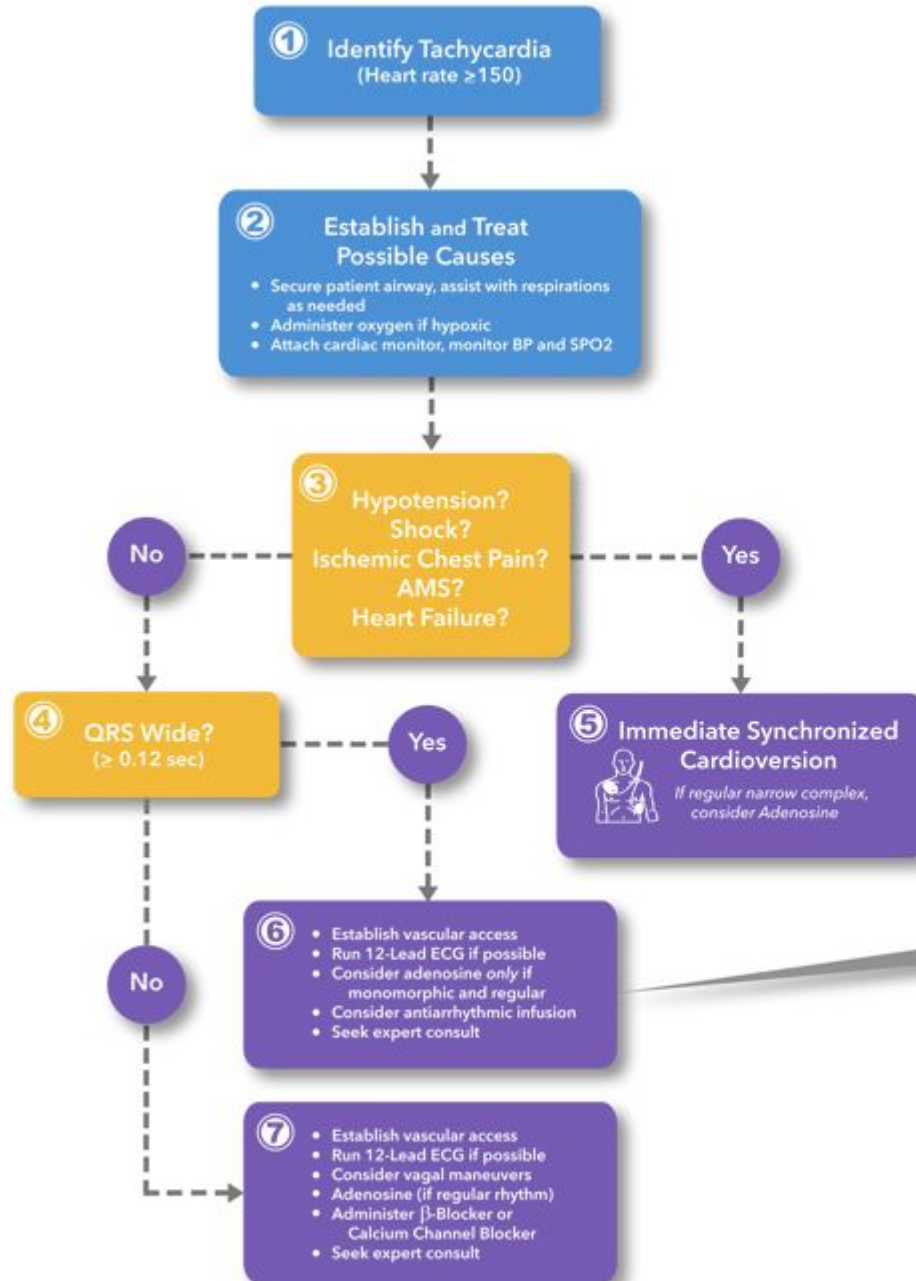
Monitor



Sustained monomorphic ventricular tachycardia with atrioventricular (AV) dissociation. Note the independence of the atrial (sinus) rate (75 per minute) and ventricular (QRS) rate (140 per minute).

?





Synchronized Cardioversion Starting Doses:

- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (Not synchronized)

Adenosine

6 mg rapid IV push,
follow with NS flush
2nd Dose: 12 mg



Antiarrhythmic Infusions (Stable Wide-Complex)



Amiodarone:

150 mg over 10 min
Repeat as necessary if VT recurs

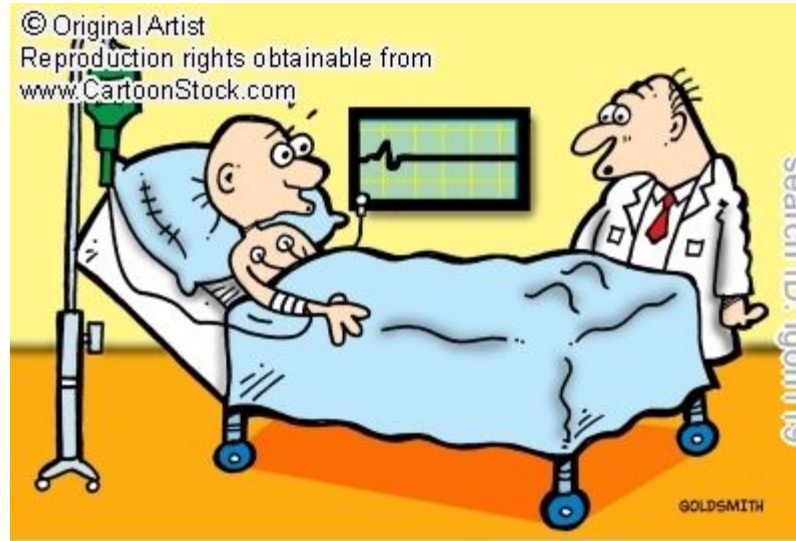
Procainamide:

20-50 mg/min until arrhythmia is suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose of 17 mg/kg is reached

Sotalol:

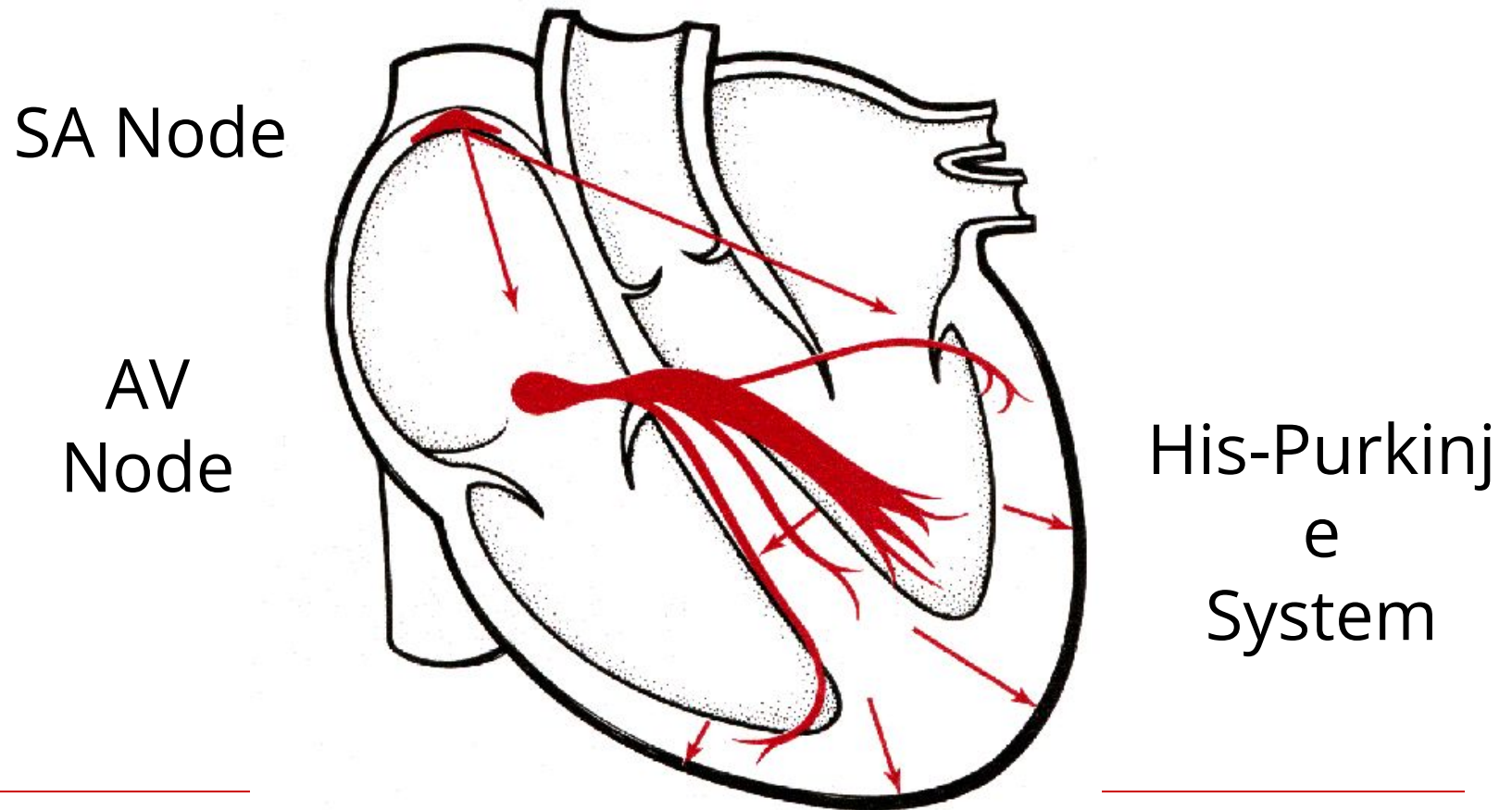
100 mg (1.5 mg/kg) over 5 min

Atrioventricular Conduction Disturbances and Bradyarrhythmias



IF THE ECG ISN'T BROKEN THEN WE HAVE PROBLEM

Sites of Disturbances in Impulse Formation or Conduction Leading to Bradyarrhythmias



Pacemaker Hierarchy (Dominant vs Subsidiary/Escape Pacemakers)

Intrinsic Rate of Firing

**60-100
min⁻¹**

**SA
Node**
(Atria+)

AV Junction
AVN/His=)
(Bundle

**40-60
min⁻¹**

Ventricles
Distal Purkinje =)
(System

**30-40
min⁻¹**

AV Block

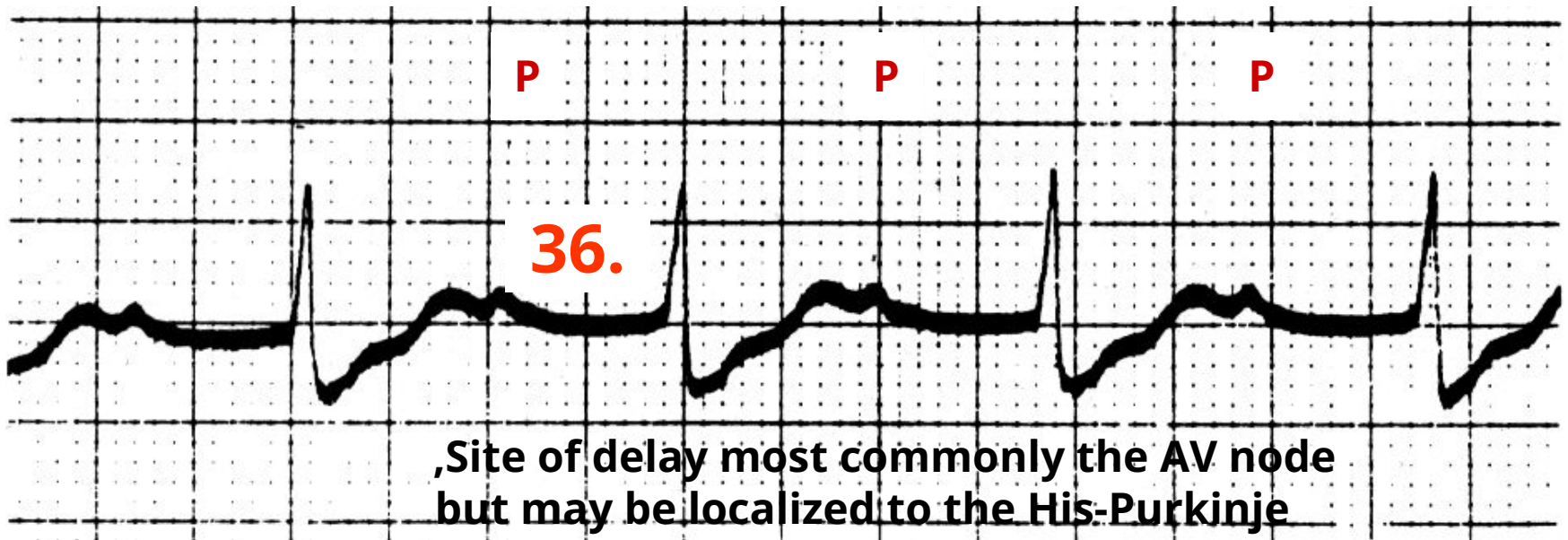
AV Block - Definitions

- **First Degree:** Prolonged conduction time
 - **Second Degree:** Intermittent non-conduction
 - **Third Degree:** Persistent non-conduction
-

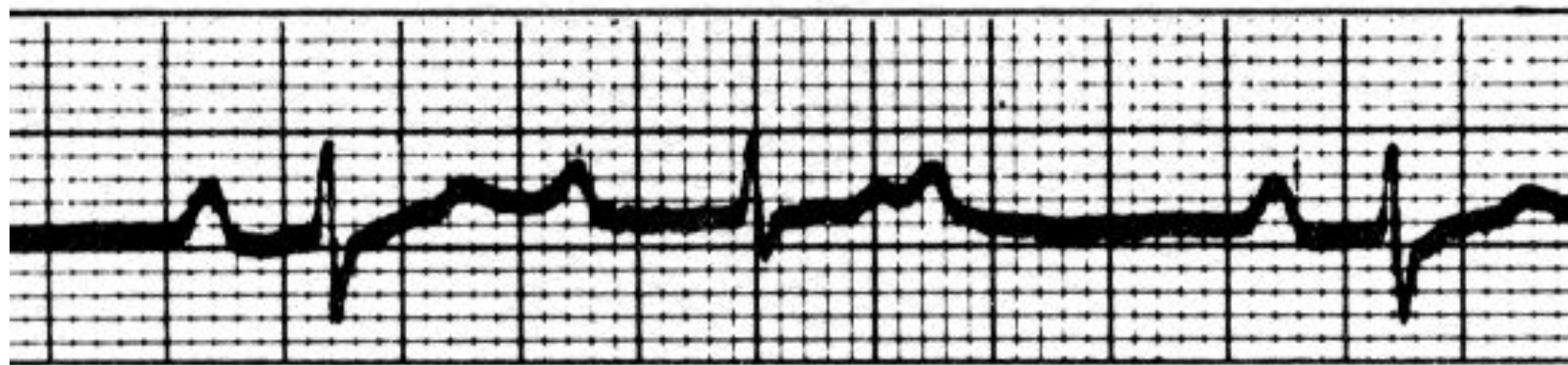
First Degree AV Block

PR > .20 sec [1 big)
(box]

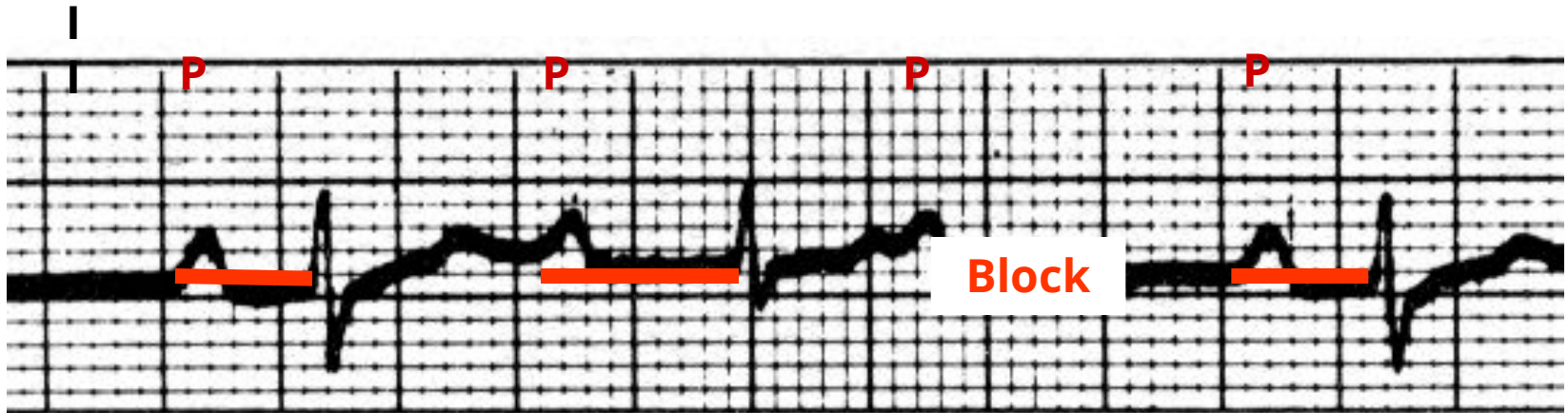
II



,Site of delay most commonly the AV node
but may be localized to the His-Purkinje
system

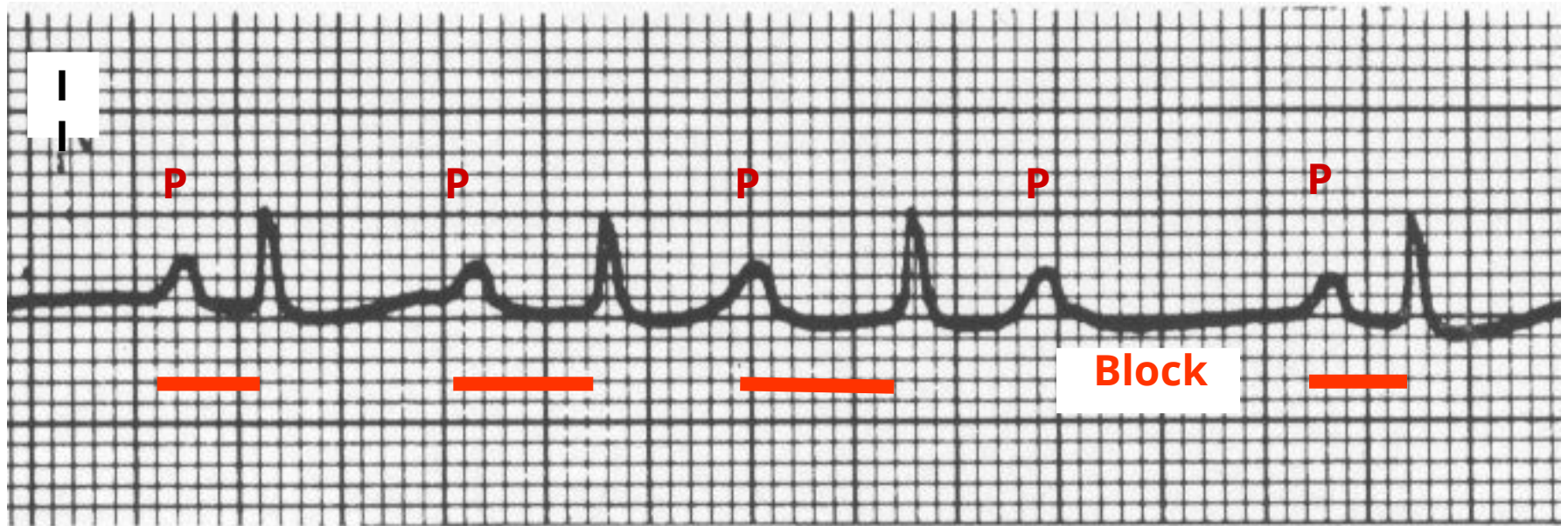


Second Degree AV Block - Type I (Wenkebach or Mobitz) (I Block)



- ;Example of 3:2 conduction ratio •
- Note PR \uparrow prior to block and \downarrow post-block •
- Characteristic of *AV nodal* site of block •

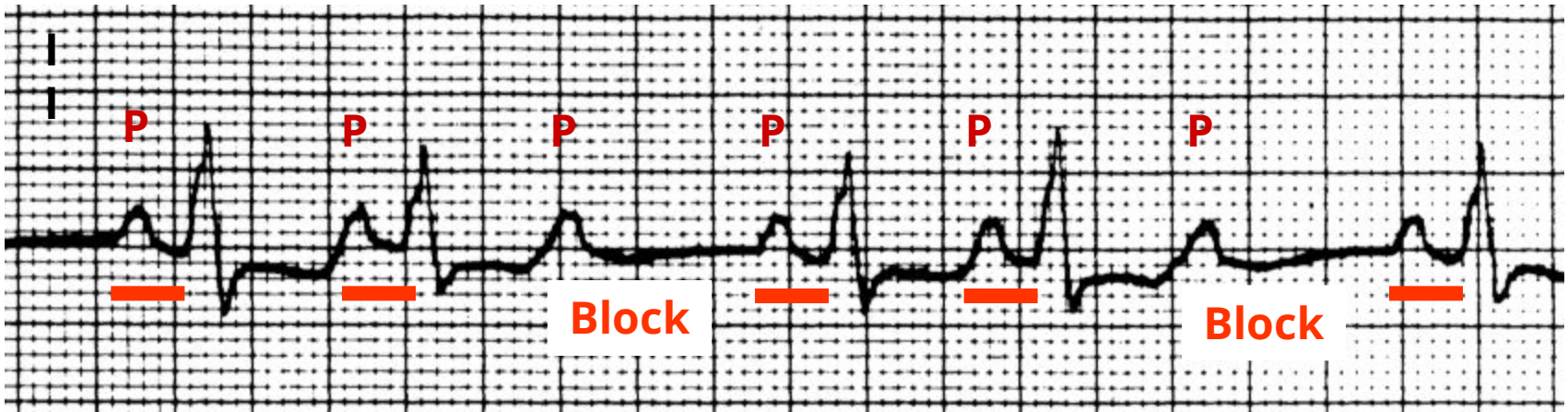
Second Degree AV Block - Type I (Wenkebach or Mobitz I Block)



conduction ratio 4:3 •
Note first RR *longer* than second RR •



Second Degree AV Block - Type II (Mobitz II)

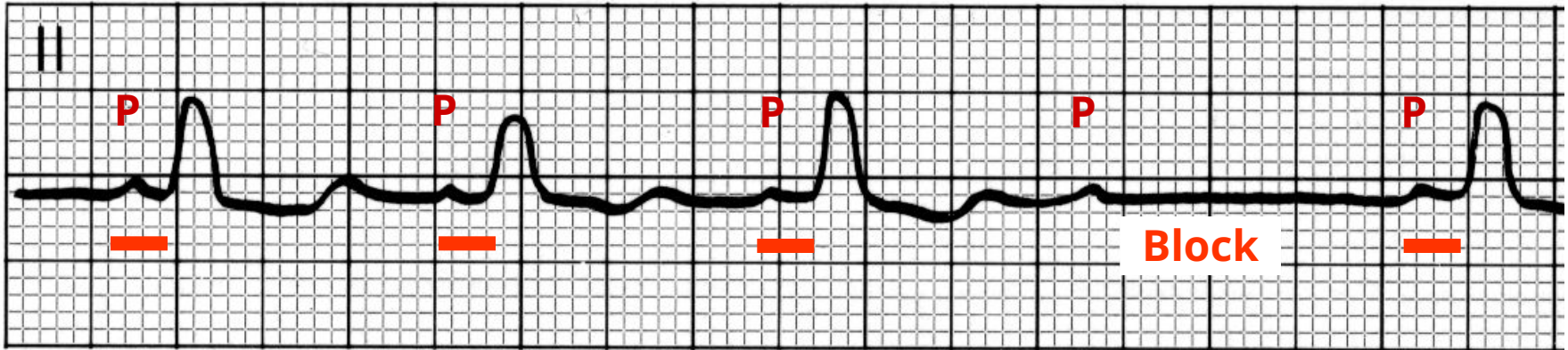


• Example of 3:2 conduction ratio •

• Note *fixed* PR for all conducted beats •

• Characteristic of *His-Purkinje system* site of block •

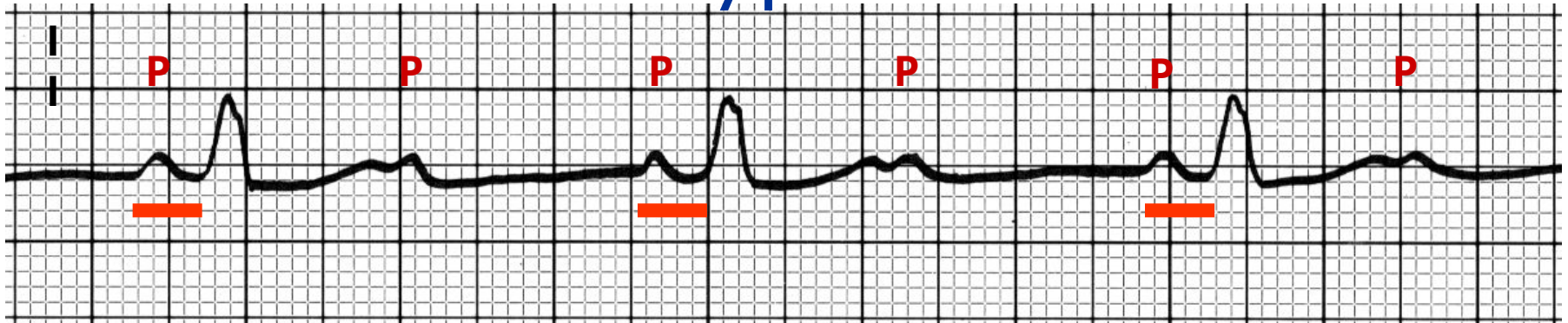
Second Degree AV Block - Type II



conduction 4:3
ratio

Second Degree AV 2:1 - Block

Type I or
?Type II



?Is site of block within the *AV node* or *His-Purkinje System*

EKG/Clinical Clues* to site of 2:1 Second Degree AV block

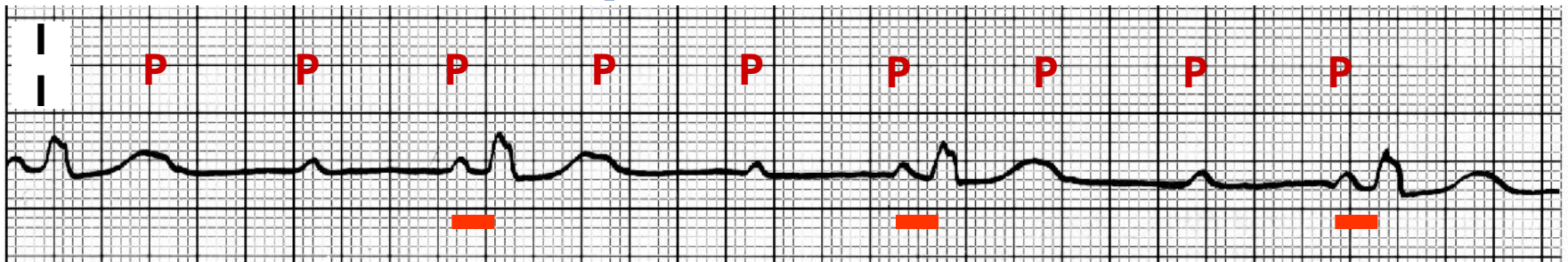
Favoring AV Node

- QRS *narrow*
- Improves with exercise (catecholamine-facilitated conduction)
- Observed in setting of increased vagal tone (e.g., sleep) or AV nodal depressant drugs

Favoring His-Purkinje System

- QRS *wide* (BBB patterns)
 - Unchanged (possibly even precipitated) during exercise
 - May improve with heart rate slowing during increased vagal tone
-

Advanced Second Degree AV Block (Block of ≥ 2 Consecutive) (P Waves



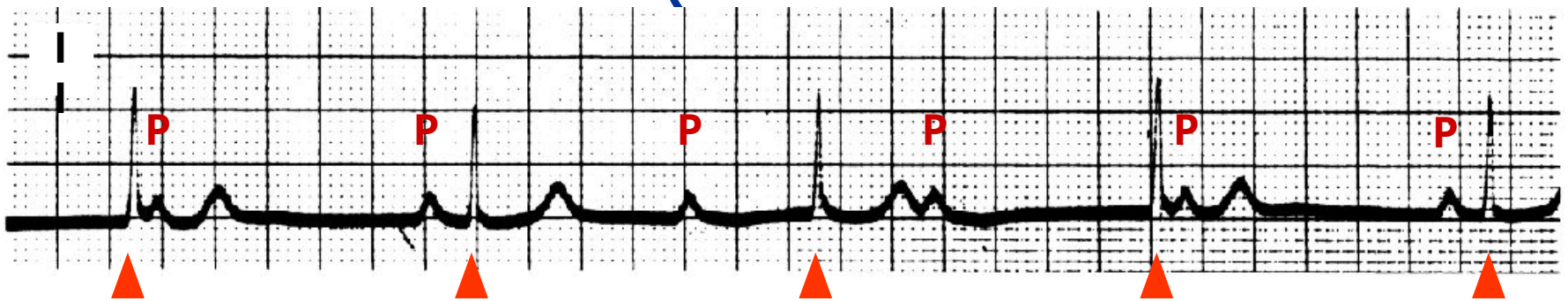
**conduction ratio, with ventricular rate in the 3:1
30's**

Site of AV Block vs. Escape Rhythm

- **AV Node:** Junctional or ventricular
 - **His-Purkinje System:** Ventricular
-



Third Degree AV Block (Complete Heart) (Block



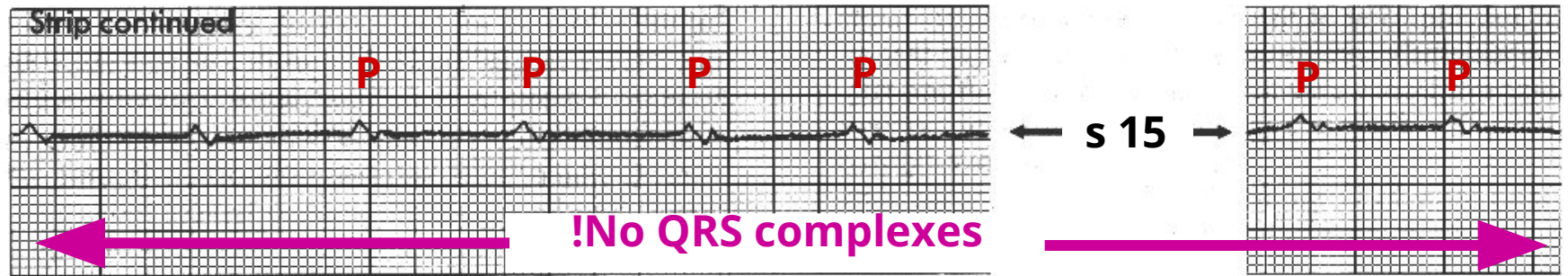
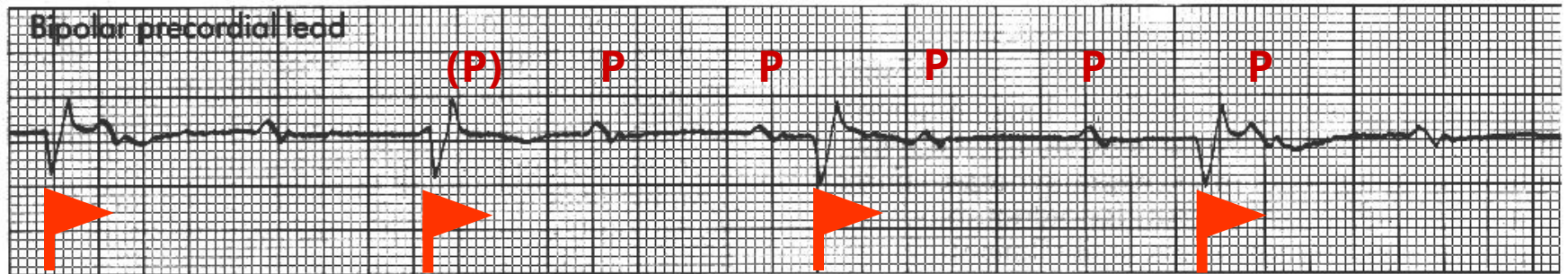
P waves at 60 beats/min •

QRS complexes (junctional escape rhythm) at 45 beats/min •

Atrial and ventricular activity are completely *unrelated* •

Junctional escape rhythm suggests *AV nodal* site of block •

Unreliability of Ventricular Escape Rhythm in Third Degree AV Block







Causes of NON-Physiologic AV Block

- Ischemic heart disease, cardiomyopathy and degenerative changes
 - Drugs that depress AV conduction
 - AV Node: digoxin, beta blockers, calcium channel blockers, amiodarone
 - His-Purkinje System: Antiarrhythmic drugs that depress the inward sodium current
 - Myocardial infection, infiltration (e.g., tumor)
 - Trauma (e.g., surgery; therapeutic ablation)
 - Congenital abnormalities
-

Sinus Bradycardia

Sinus Bradycardia



P wave upright in leads I and II, just as in normal sinus rhythm

Causes of Sinus Bradycardia

- ❑ Increased vagal tone
 - ❑ Drugs: beta blockers, calcium channel blockers, amiodarone, digoxin (indirect effect)
 - ❑ Myocardial ischemia/infarction
 - ❑ Hypothyroidism
 - ❑ “Sick sinus syndrome” - degenerative/fibrotic atrial process
-

Sequence of P Wave Generation

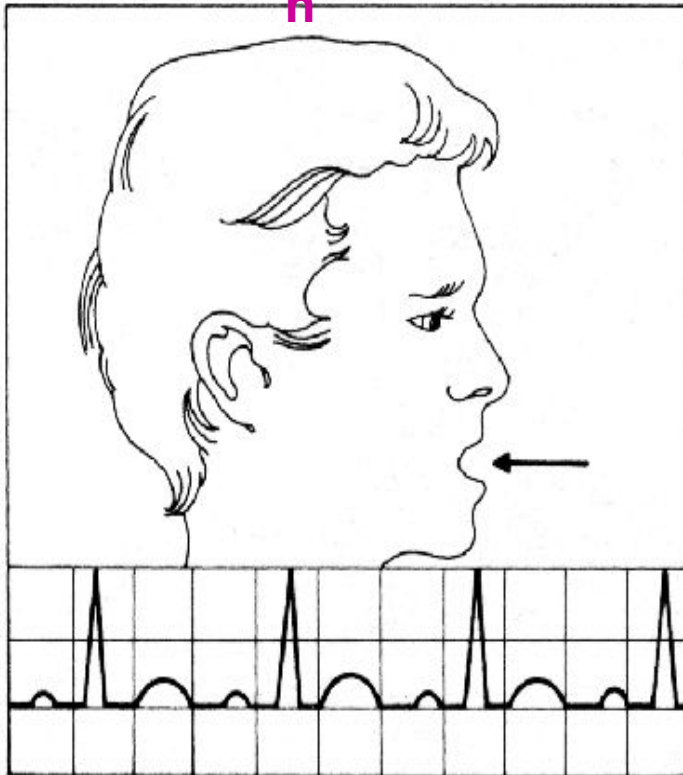


Non-visible process on the EKG

Sinus Arrhythmia

Inspiratio

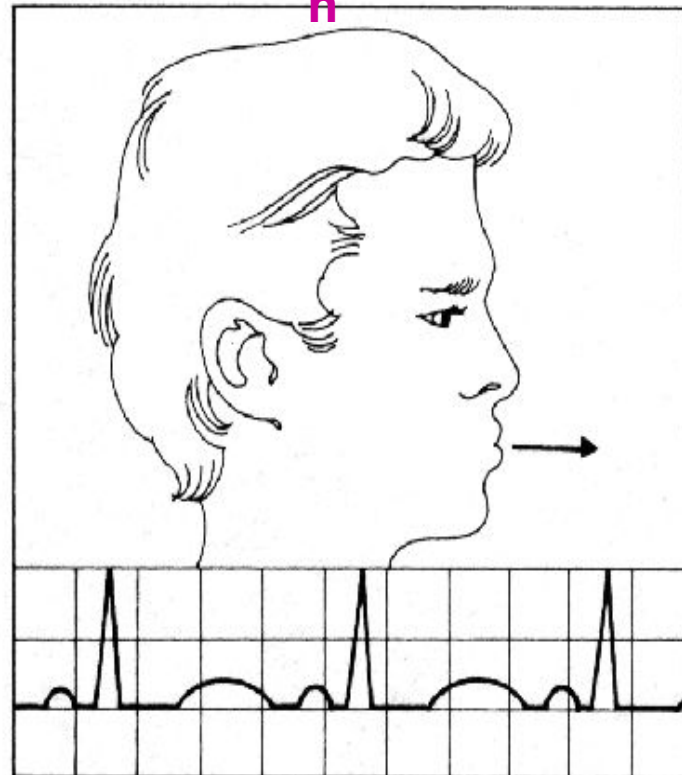
n



SA nodal
acceleration

Expiratio

n

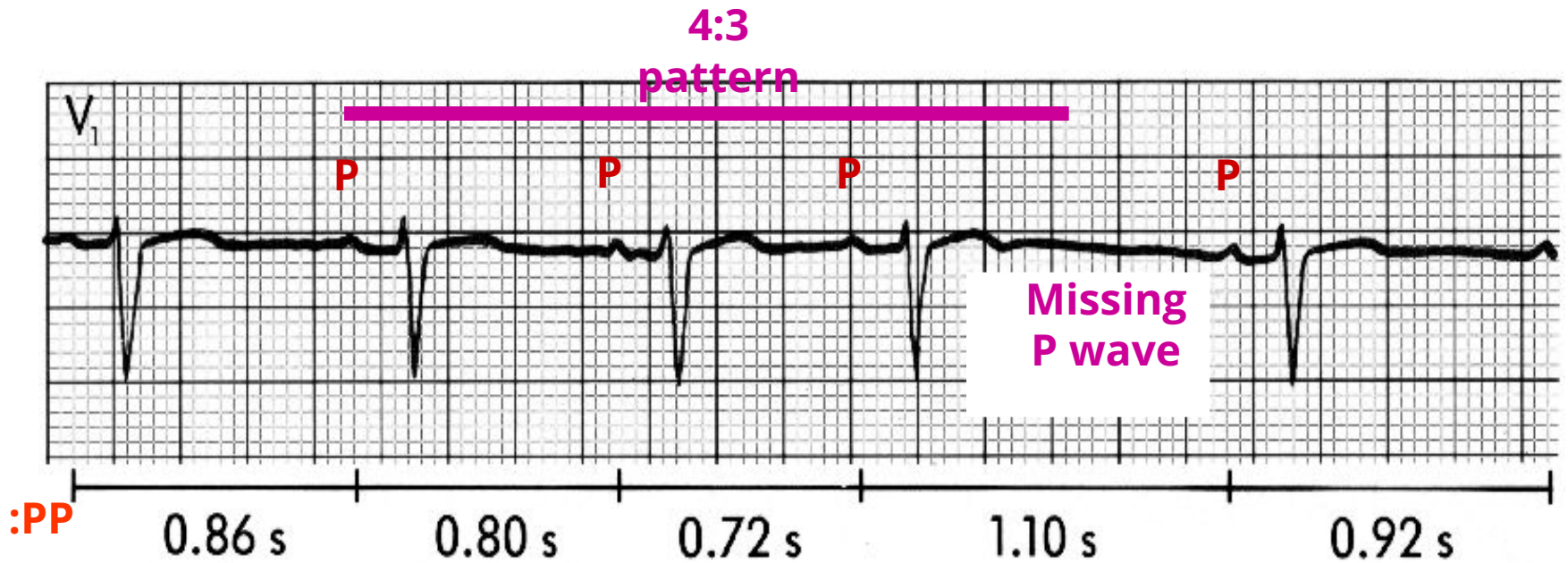


SA nodal
deceleration

Sinoatrial (SA) Exit Block - Definitions

- ❑ **First Degree:** Prolonged SA conduction time (non-detectable on EKG; no missing P waves)
 - ❑ **Second Degree:** Intermittent non-conduction (intermittent absence of P waves)
 - ❑ **Third Degree:** Persistent non-conduction (complete absence of P waves; escape rhythms only)
-

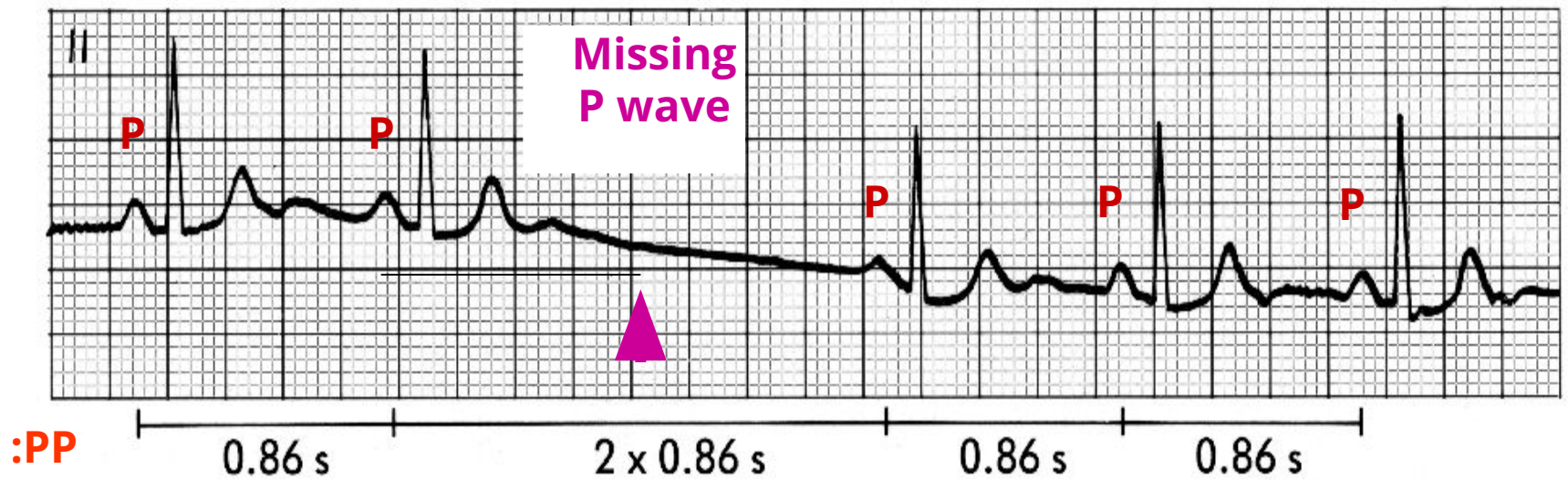
Second Degree SA Exit Block - Type I (Wenkebach)



PP intervals shorten prior to
block

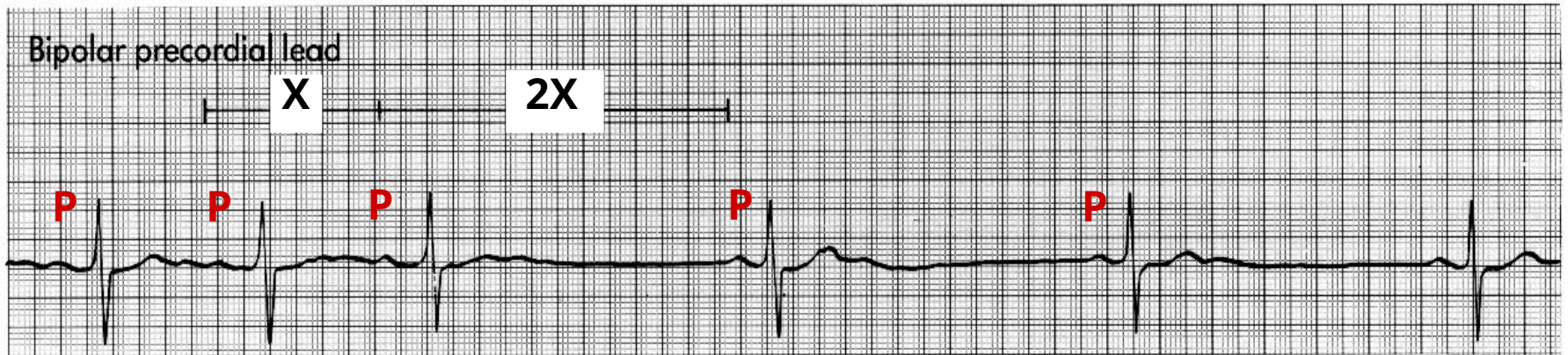
Note unaffected, *fixed* PR
intervals

Second Degree SA Exit Block - Type II

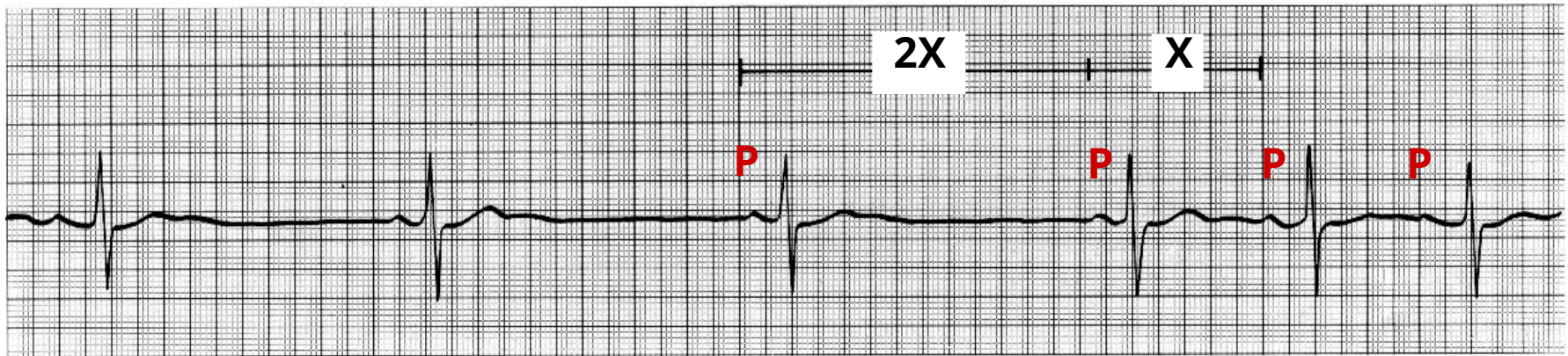


One P wave abruptly “drops out” on time

2:1 SA Exit Block (Every Other P wave is “Dropped”)

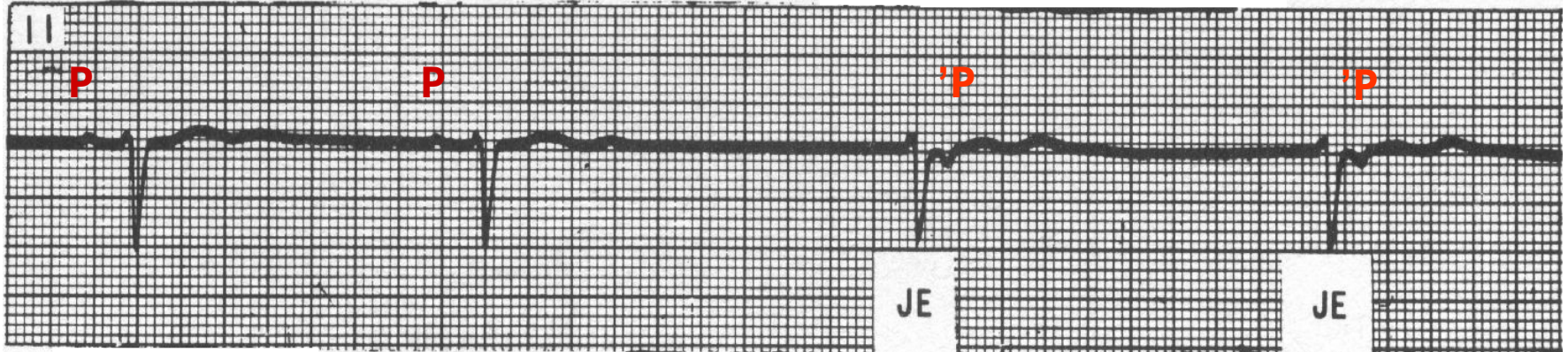


Atrial rate is abruptly cut in half



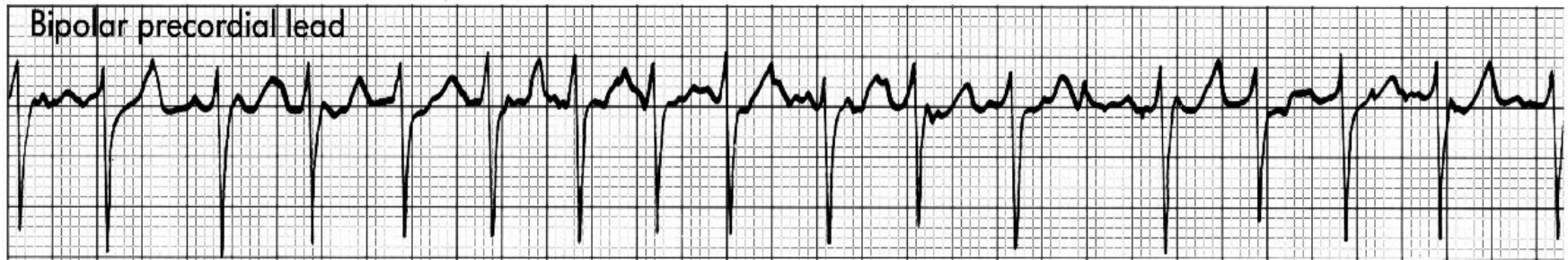
Resolution of block

Sinus Arrest

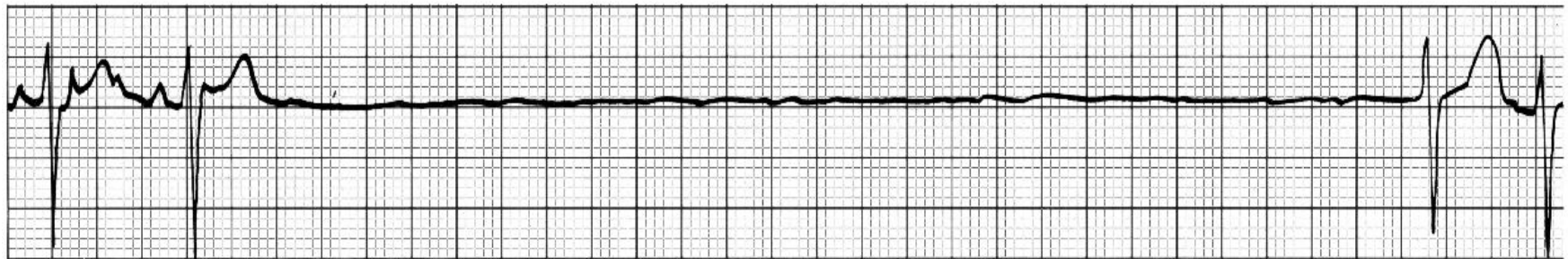


Sinus bradycardia → Sinus arrest rhythm → Slow junctional escape
with retrograde p)
(waves

Tachycardia-Bradycardia Syndrome ("Form of "Sick Sinus)



**Atrial
Flutter** →



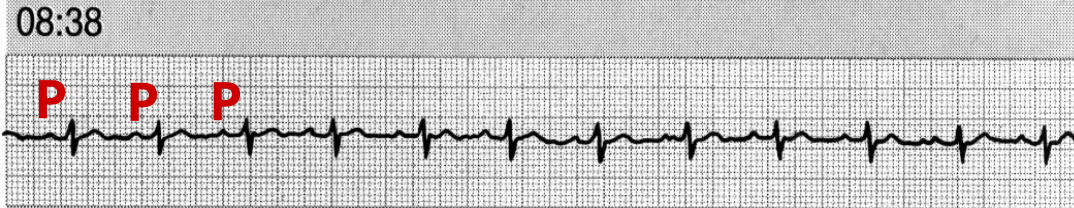
**Atrial
Flutter
terminates**

**Sinus
arrest**

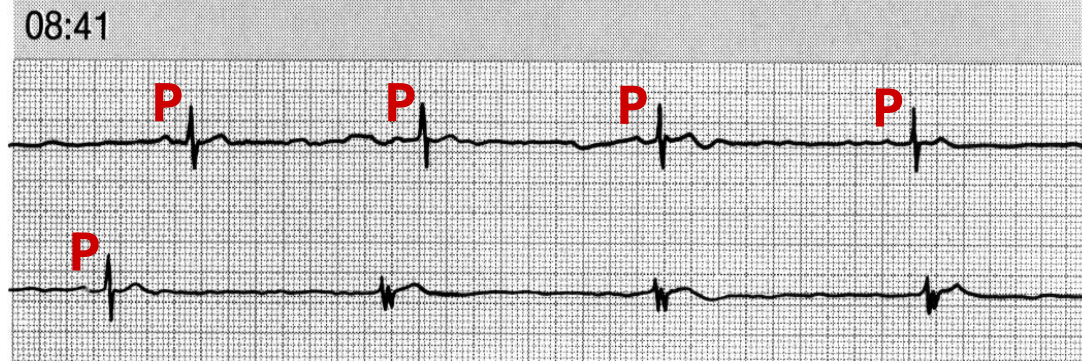
**Junctional
escape
(tardy)**

Sinus Arrest → Asystole

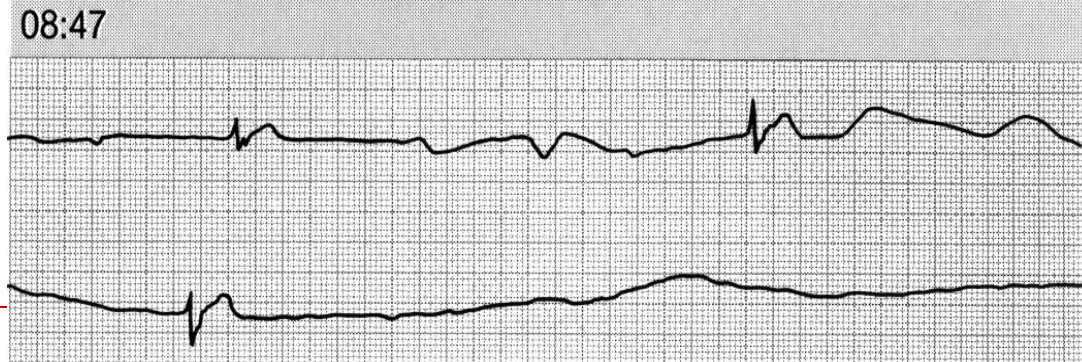
Sinus rhythm



.Sinus brady
Sinus →
arrest
V. escape →
rhythm



.Failure of V
escape
rhythm
Asystole →



Causes of SA Exit Block and Sinus Pauses/Arrest

- ❑ Increased vagal tone (very intense for sinus arrest)
 - ❑ Drugs: beta blockers, calcium channel blockers, amiodarone, digoxin (indirect effect)
 - ❑ Myocardial ischemia/infarction
 - ❑ Sick sinus syndrome
 - ❑ Sequela of open heart surgery
-

Sick Sinus Syndrome

- (1) persistent spontaneous sinus bradycardia not caused by drugs and inappropriate for the physiologic circumstance;
 - (2) sinus arrest or exit block
 - (3) combinations of SA and AV conduction disturbances
 - (4) alternation of paroxysms of rapid regular or irregular atrial tachyarrhythmias and periods of slow atrial and ventricular rates (bradycardia-tachycardia syndrome)
-

Adult Bradycardia (With Pulse)

