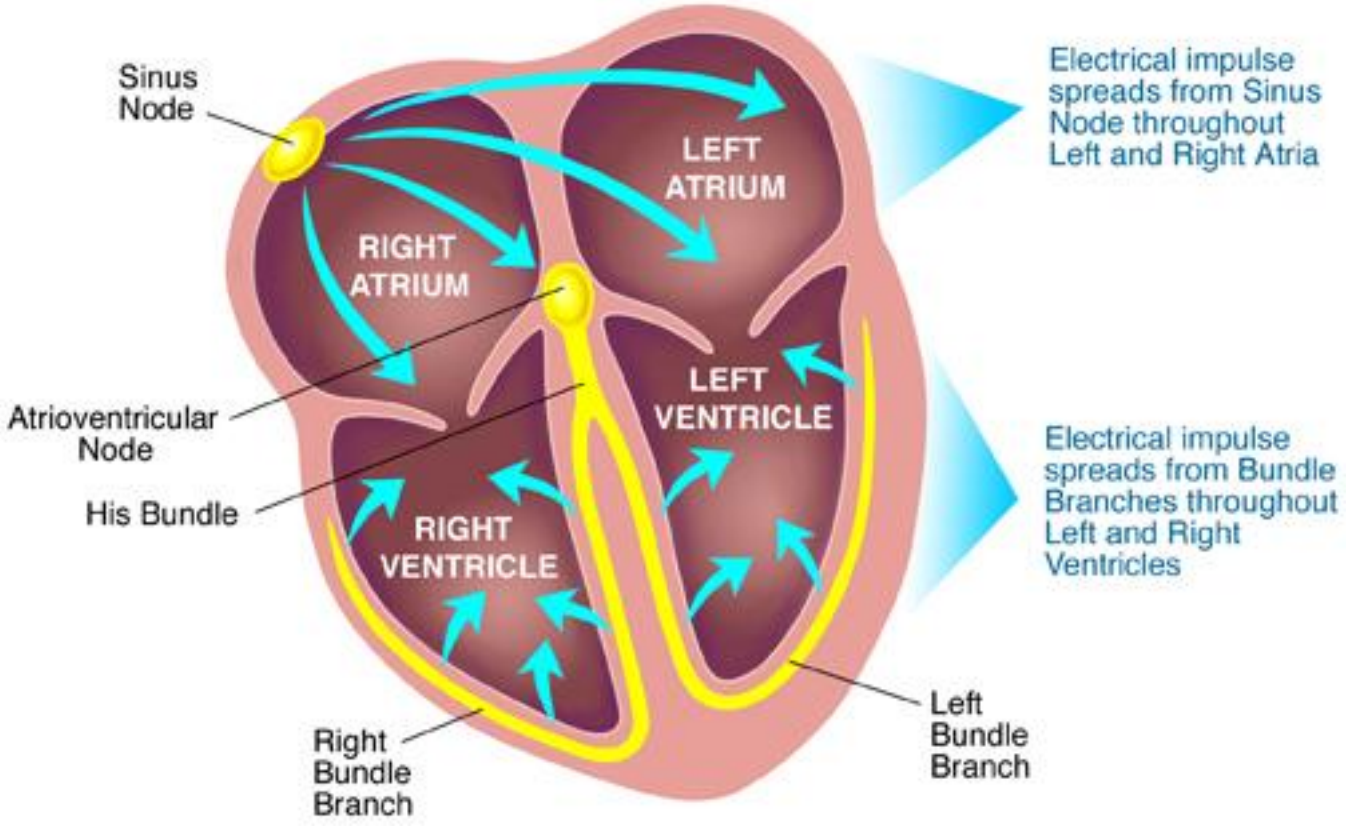
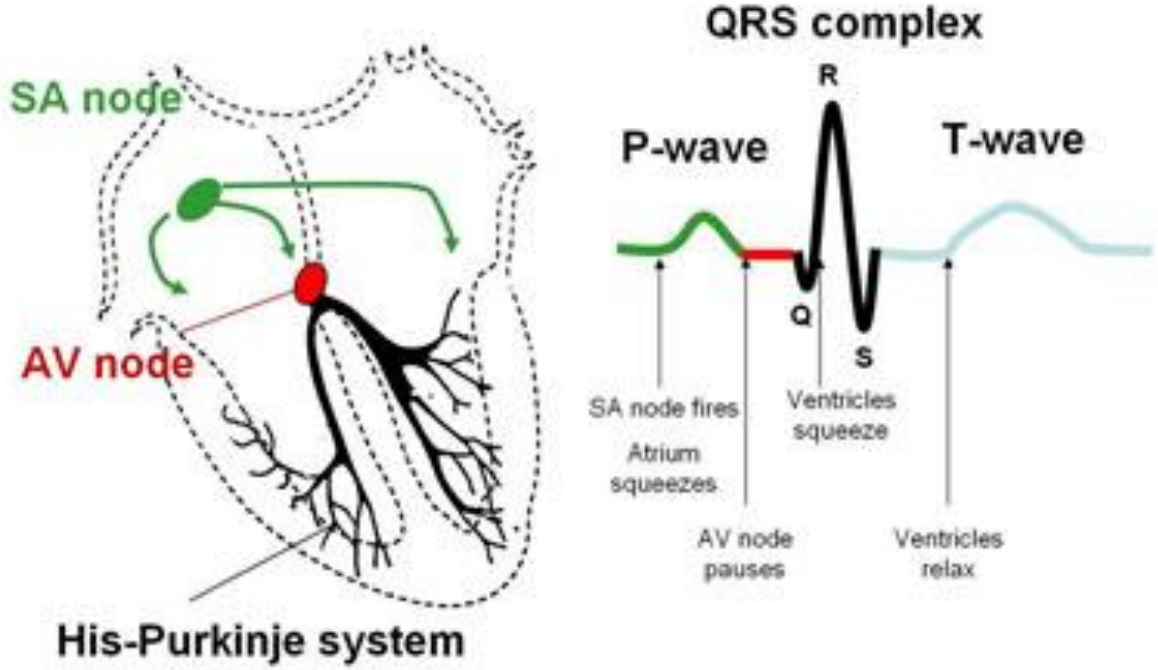
CARDIAC ARRHYTHMIAS

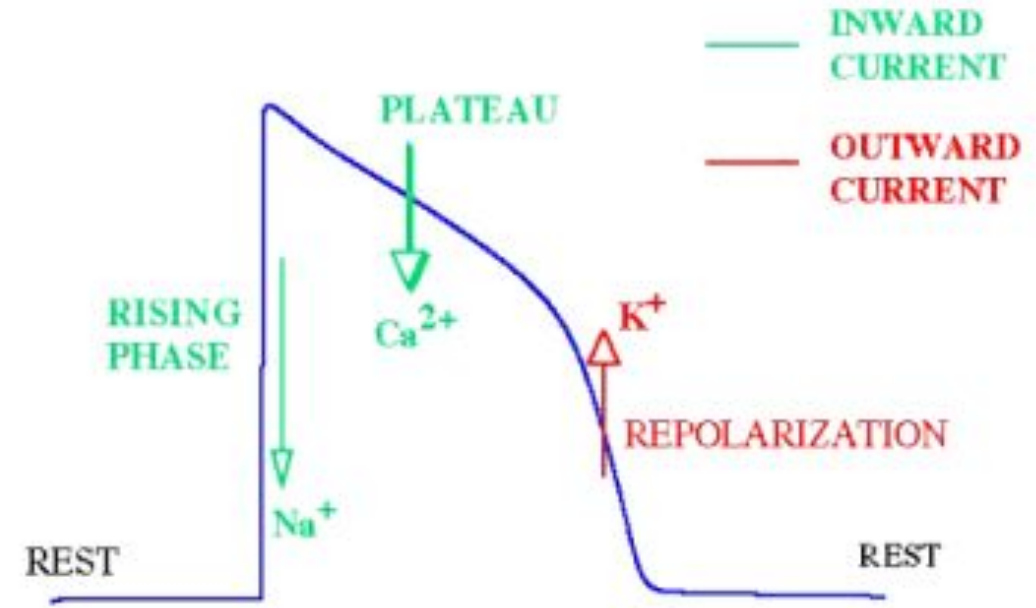
Sergey Yalonetsky, MD

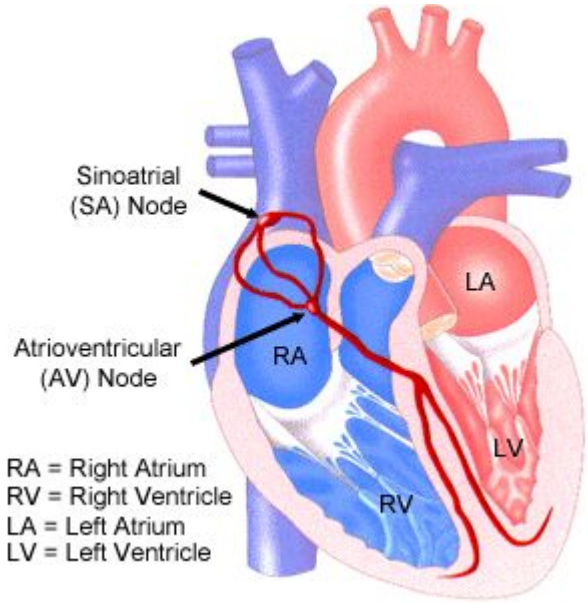

BIO
MATH
TRIG
© Ron Leishman * www.ClipartOf.com/437774


Sinus Node
LEFT ATRIUM
RIGHT ATRIUM
Electrical impulse spreads from Sinus Node throughout Left and Right Atria
Atrioventricular Node
His Bundle
LEFT VENTRICLE
RIGHT VENTRICLE
Electrical impulse spreads from Bundle Branches throughout Left and Right Ventricles
Left Bundle Branch
Right Bundle Branch

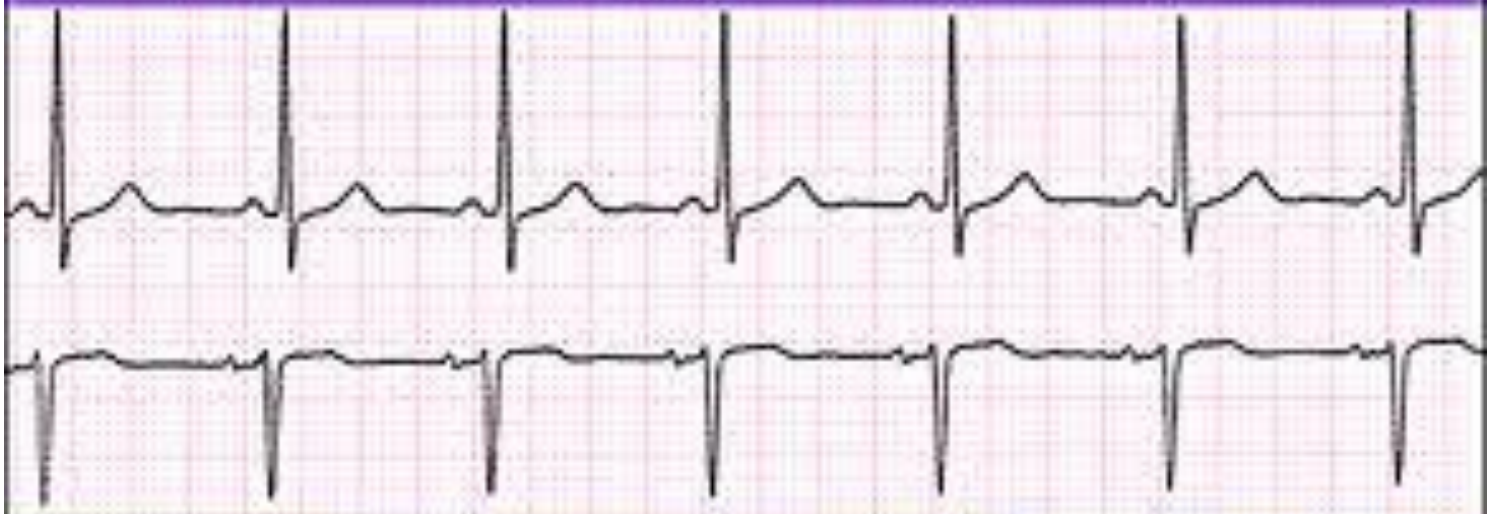

SA node
AV node
His-Purkinje system
QRS complex
R
P-wave
T-wave
Q
S
SA node fires
Atrium squeezes
AV node pauses
Ventricles squeeze
Ventricles relax

CARDIAC ACTION POTENTIAL



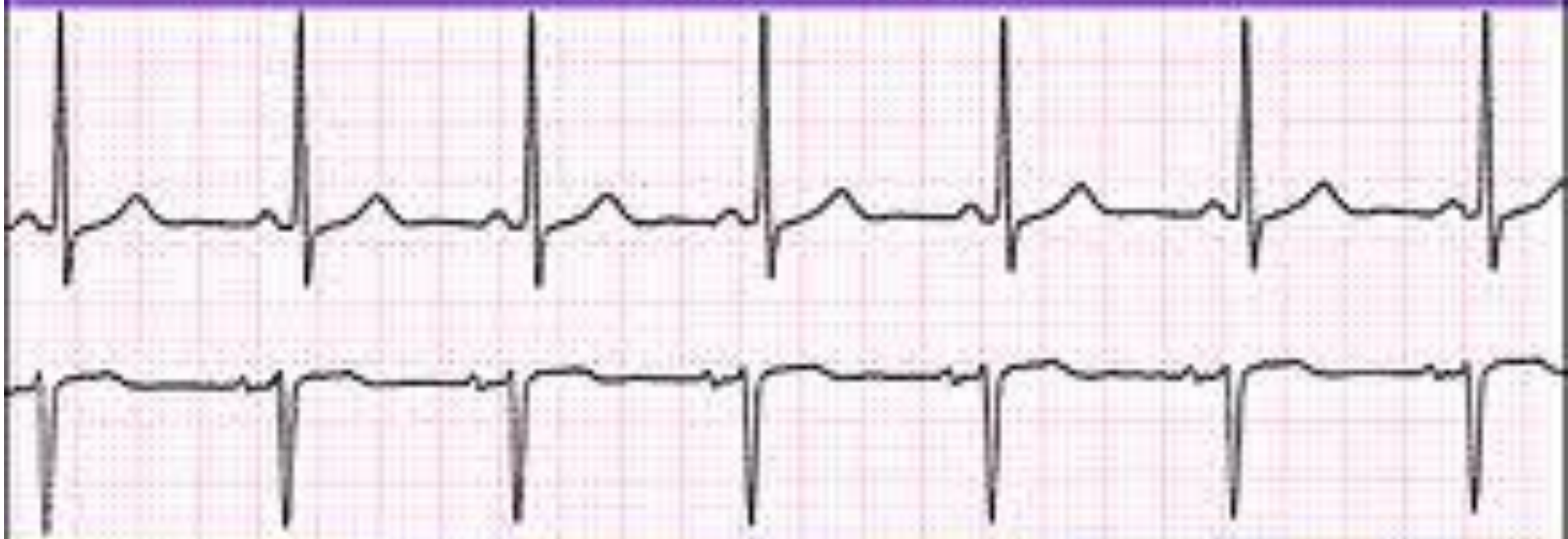

Sinoatrial
(SA) Node
Atrioventricular
(AV) Node
RA = Right Atrium
RV = Right Ventricle
LA = Left Atrium
LV = Left Ventricle
LA
RA
LV
RV

Normal Sinus Rhythm



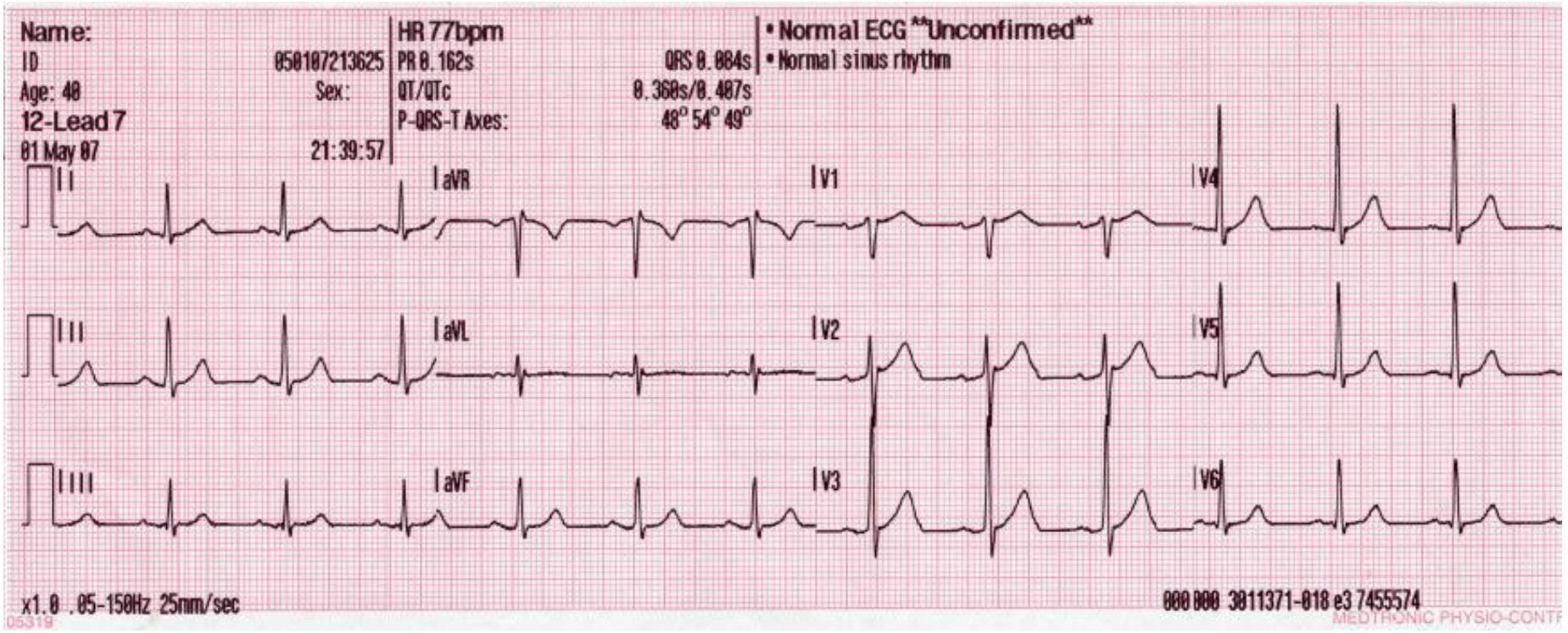
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
60-100 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

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

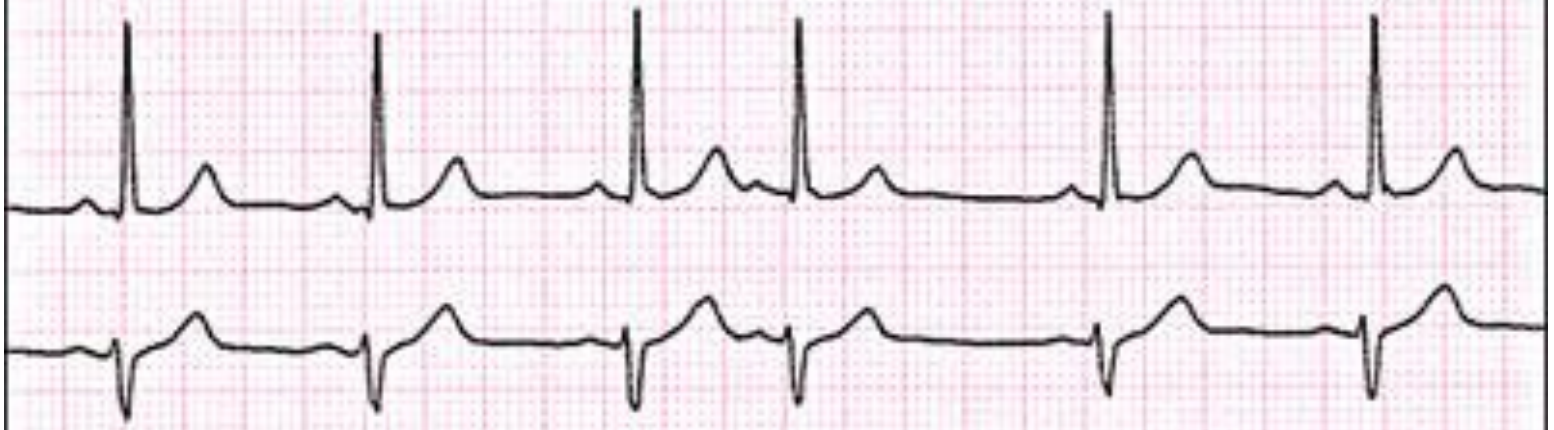


Classification

- Tachyarrhythmia:
  - Supraventricular
  - Ventricular
- Bradiarrhythmia

APB or PAC

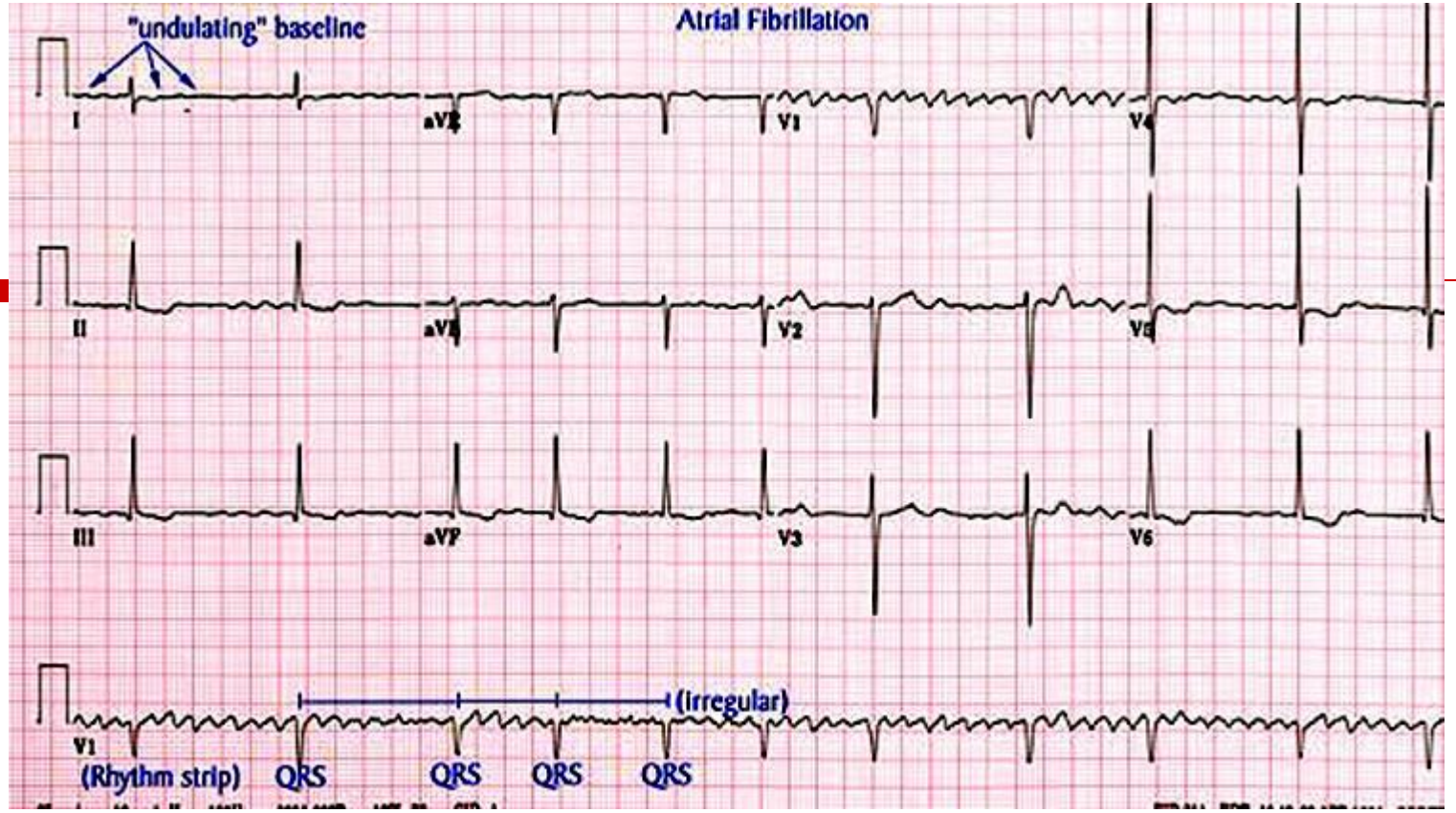
Premature Atrial Contraction • Isolated PAC's: Occur Single



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

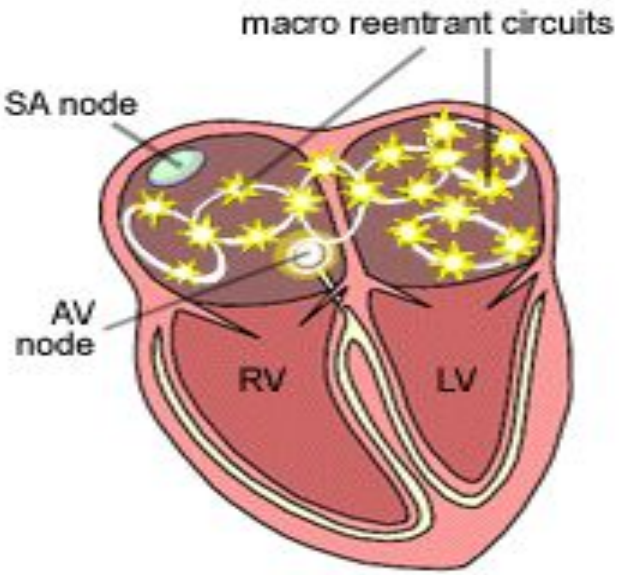
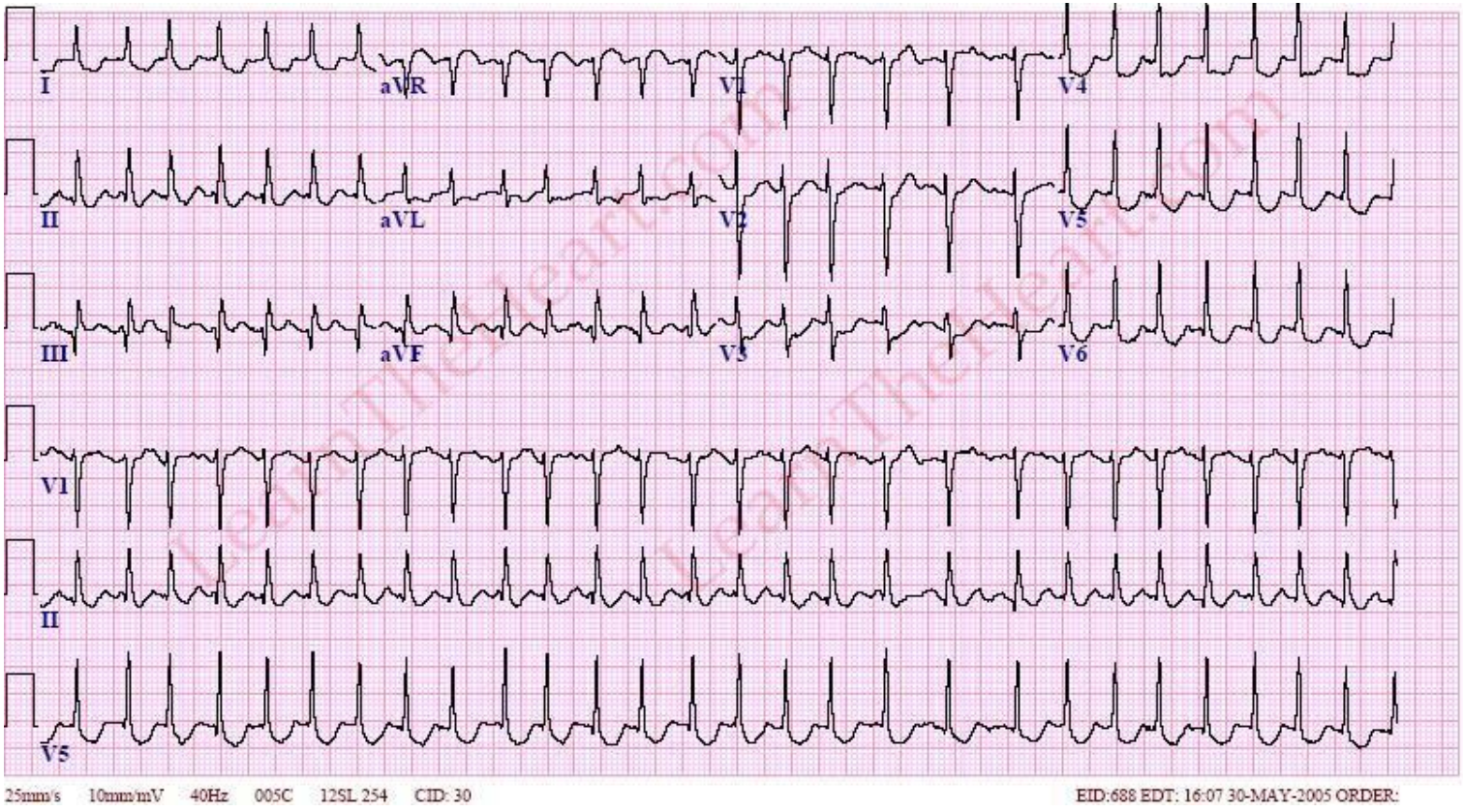


Illustration depicting macro reentrant circuit ("wavelet") activity during a run of complex atrial fibrillation (AF)

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


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005C 12SL 254 CID: 30
EID:688 EDT: 16:07 30-MAY-2005 ORDER:

Consequences of Atrial Fibrillation

Hemodynamic

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

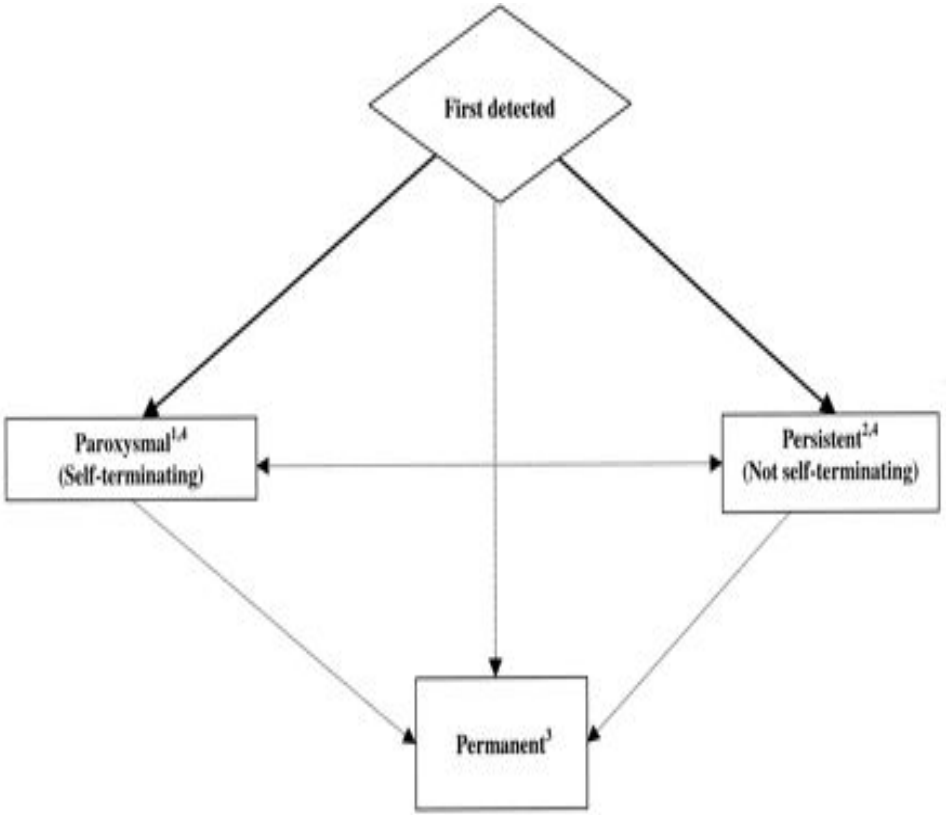
Myocardial – persistently rapid rate can lead to:

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

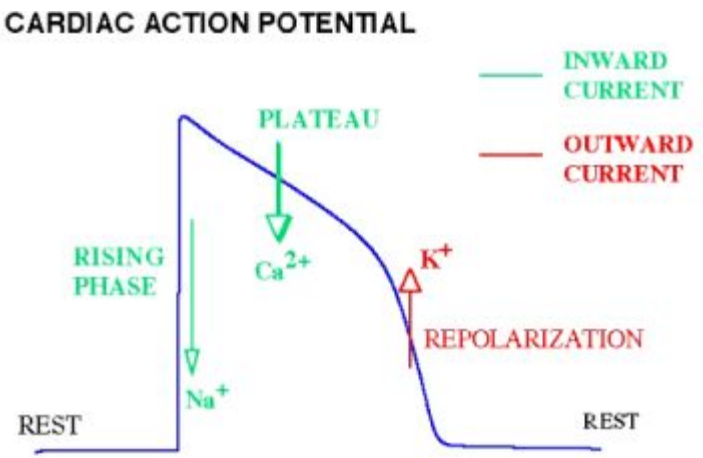
Prevention of Thromboembolysm !

Williams Classification of Antyarrhythmic Drugs

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

  IA – Reduce V max and prolong action potential

  - Quinidine
  - Procainamide
  - Disopiramide



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

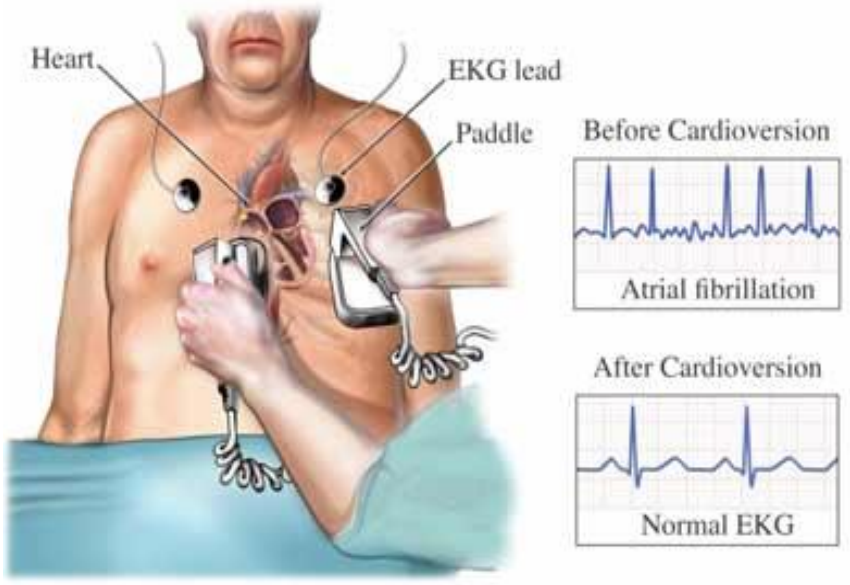
- Propafenon
- Amiodaron
- Flecainide

Cardioversion

Electric

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

  Elective cardioversion

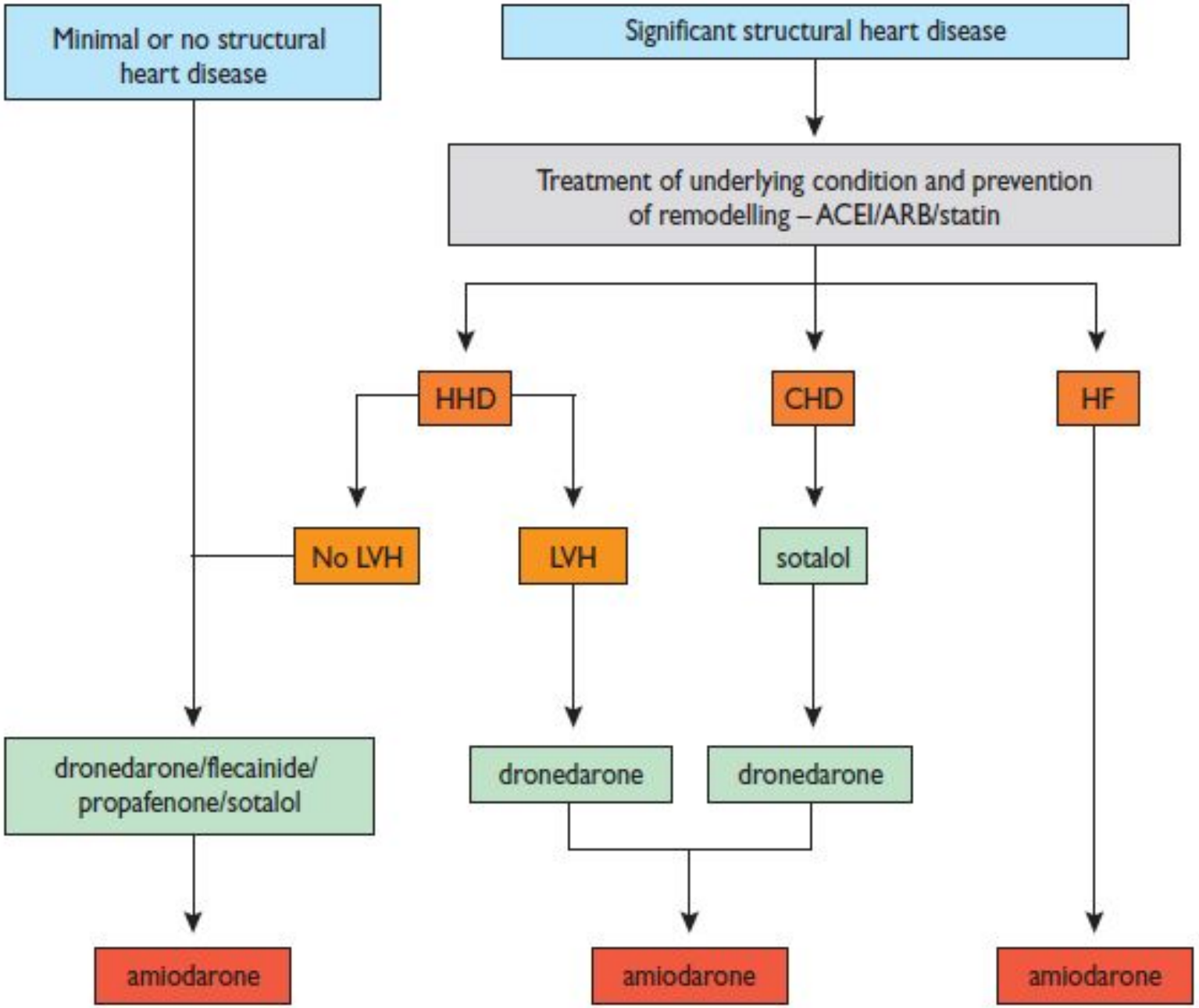


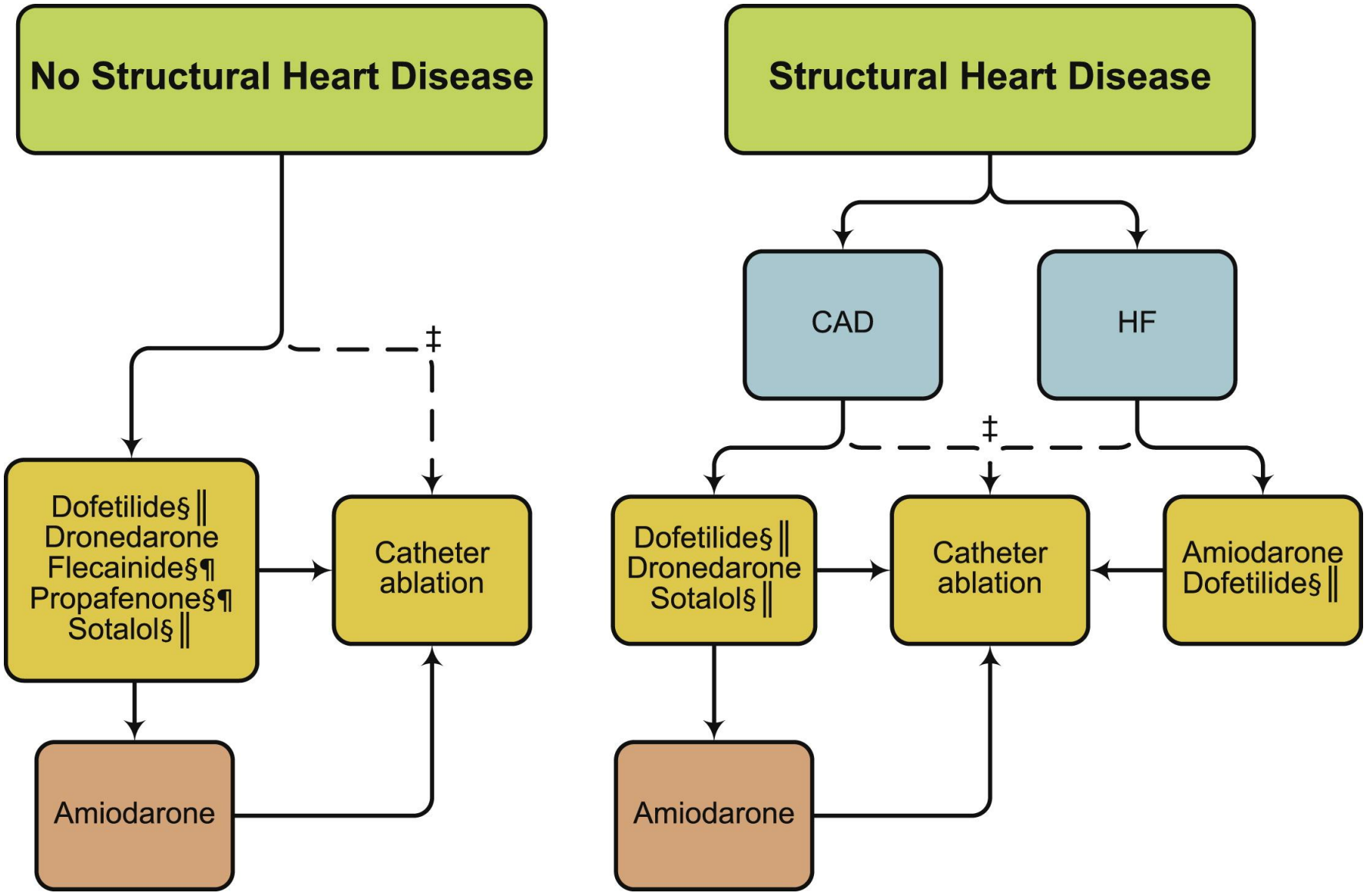
Predictors of successful cardioverson

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

Maintenance of sinus rhythm

- Propafenon
- Amiodaron
- Dronedaron
- Sotalol
- Flecainide


Minimal or no structural heart disease
Significant structural heart disease
Treatment of underlying condition and prevention of remodelling – ACEI/ARB/statin
HHD
CHD
HF
No LVH
LVH
sotalol
dronedarone/flecainide/ propafenone/sotalol
dronedarone
dronedarone
amiodarone
amiodarone
amiodarone


No Structural Heart Disease
Dofetilide§‖
Dronedarone
Flecainide§¶
Propafenone§¶
Sotalol§‖
Amiodarone
‡
Catheter ablation
Structural Heart Disease
CAD
HF
Dofetilide§‖
Dronedarone
Sotalol§‖
Amiodarone
‡
Catheter ablation
Amiodarone
Dofetilide§‖

Rate Control

- Acute setting – IV
  - Esmolol
  - Metoprolol
  - Verapamil
  - Dilthiazem
  - 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

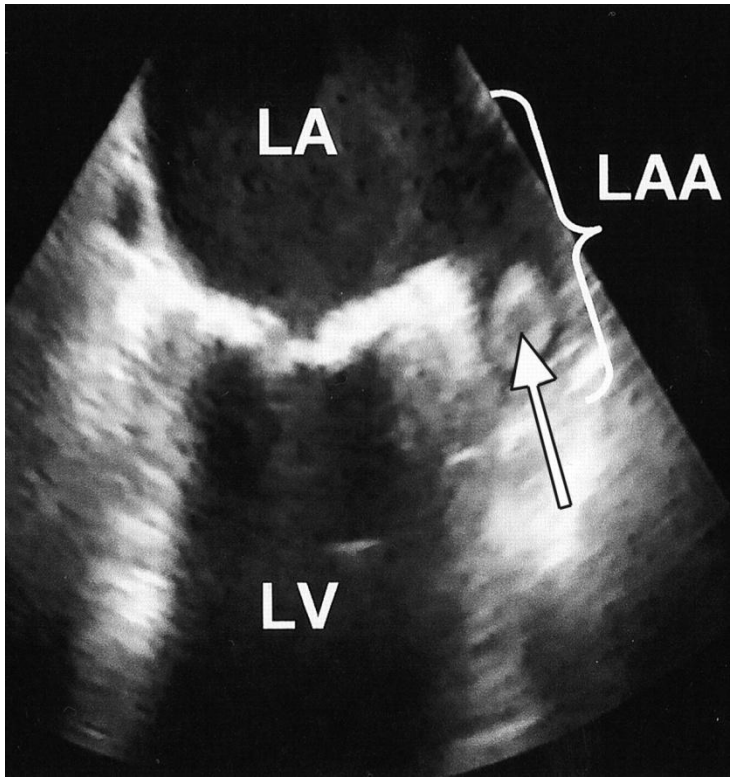
- Severe symptoms due to AF
- Patients with CHF
- Younger patients
- Patients with lone AF
- First episode of AF

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	
	Precardioversion	Postcardioversion
<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

$CHADS_2$ criteria	Points
Previous stroke or TIA	2
Age ≥ 75 years	1
Hypertension	1
Diabetes mellitus	1
Heart failure	1

Stroke risk score	Recommended therapy
High 2–6	**Warfarin** (INR 2–3)
Moderate 1	**Warfarin or aspirin**
Low 0	**Aspirin** 100–300 mg daily

Scoring Differences Between $CHADS_2$ and CHA_2DS_2-VASc

	$CHADS_2$ (Maximum score, 6)	CHA_2DS_2-VASc (Maximum score, 9)
Risk Factor	**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

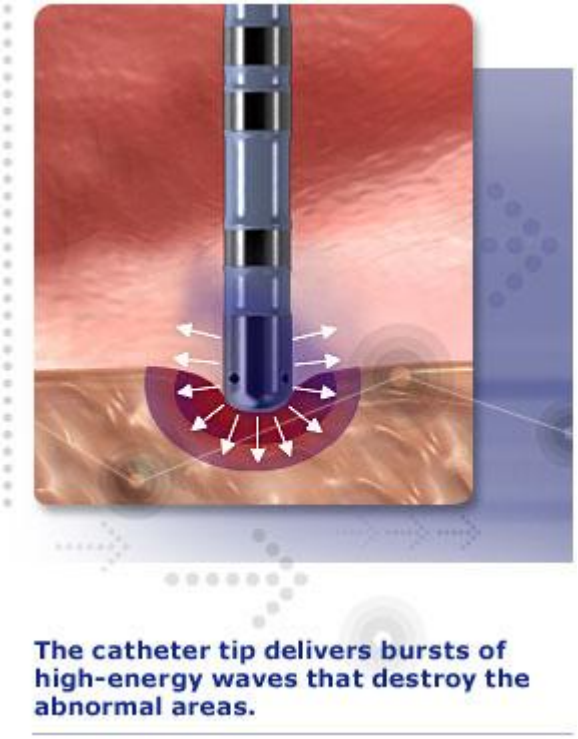
N/A – not applicable

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

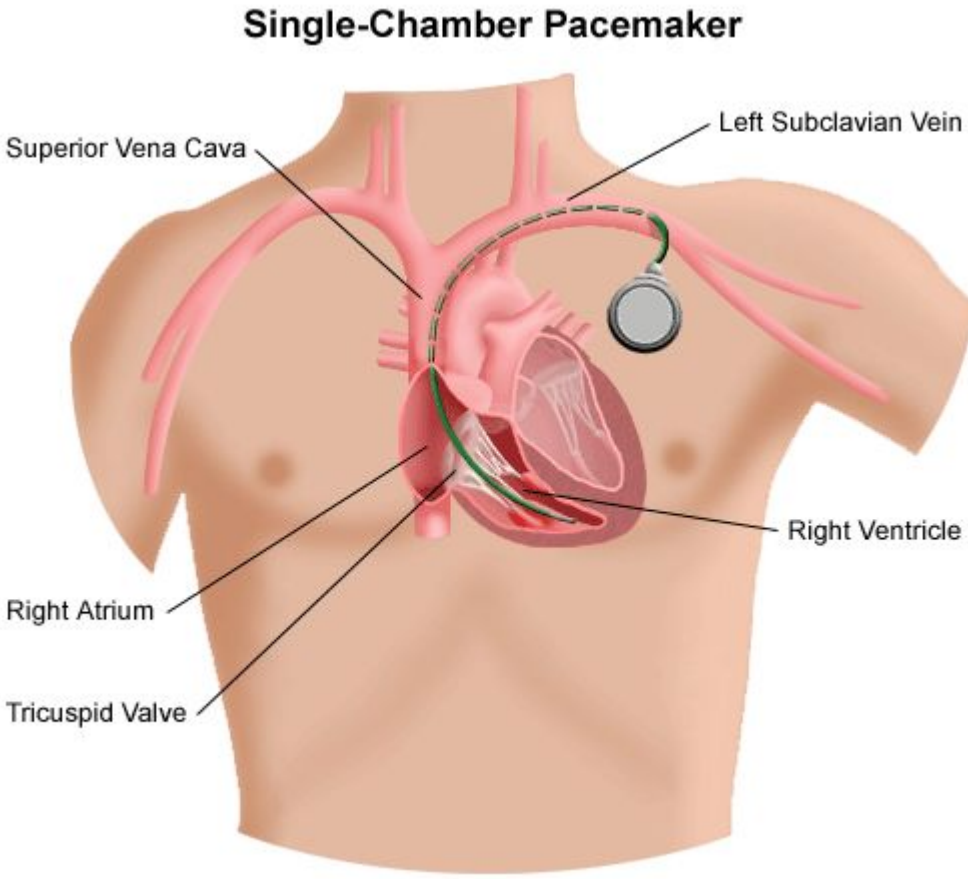


The catheter tip delivers bursts of high-energy waves that destroy the abnormal areas.

Invasive AF management

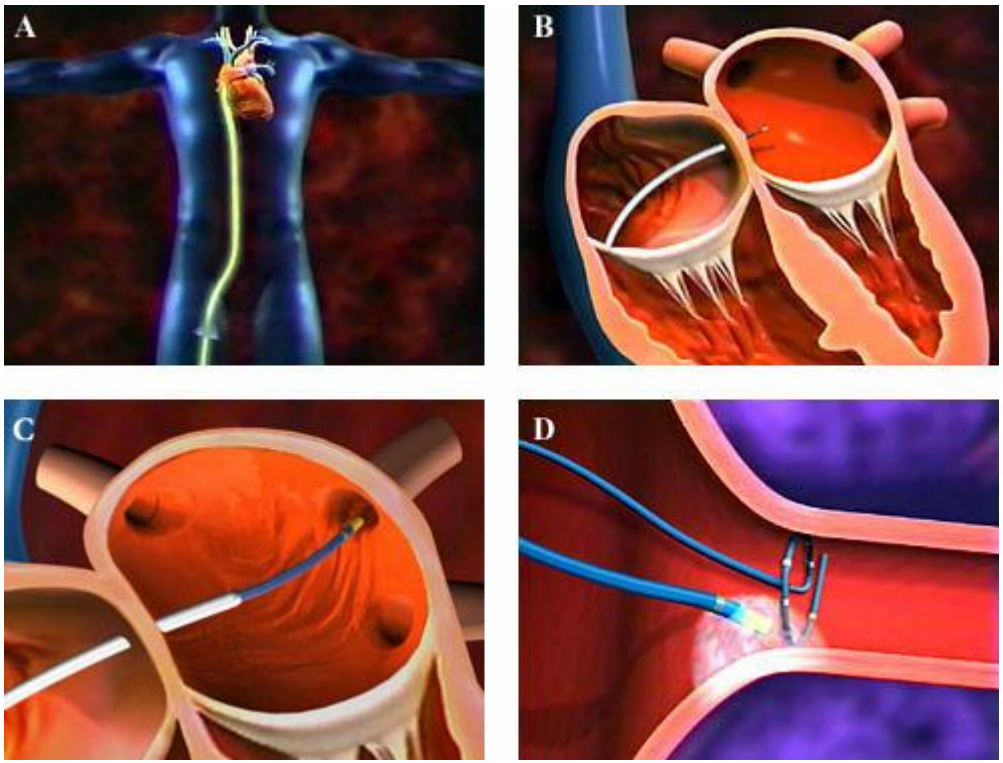
- Rate control

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

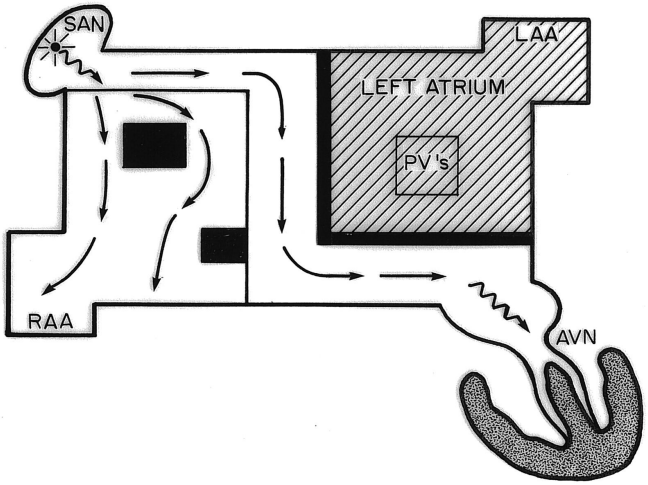


Pulmonary Venous Isolation

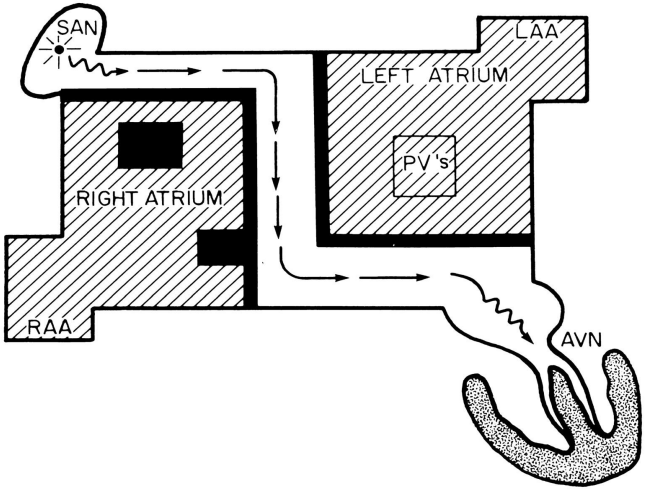
- For recurrent paroxysmal AF



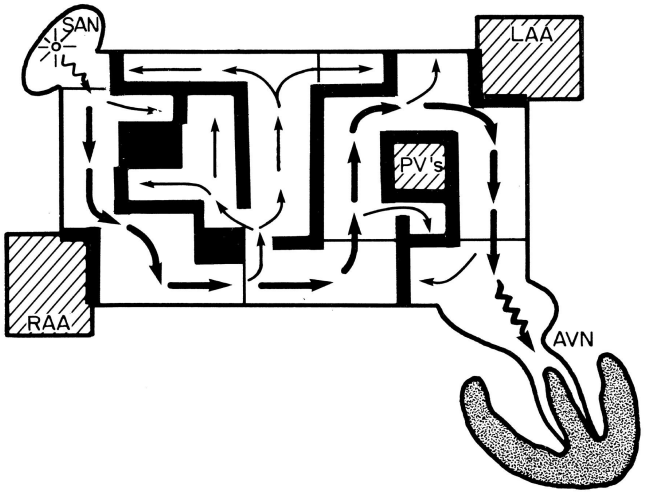
Cox-Maze Procedure



Left Atrial Isolation (1980)



Corridor Procedure (1985)



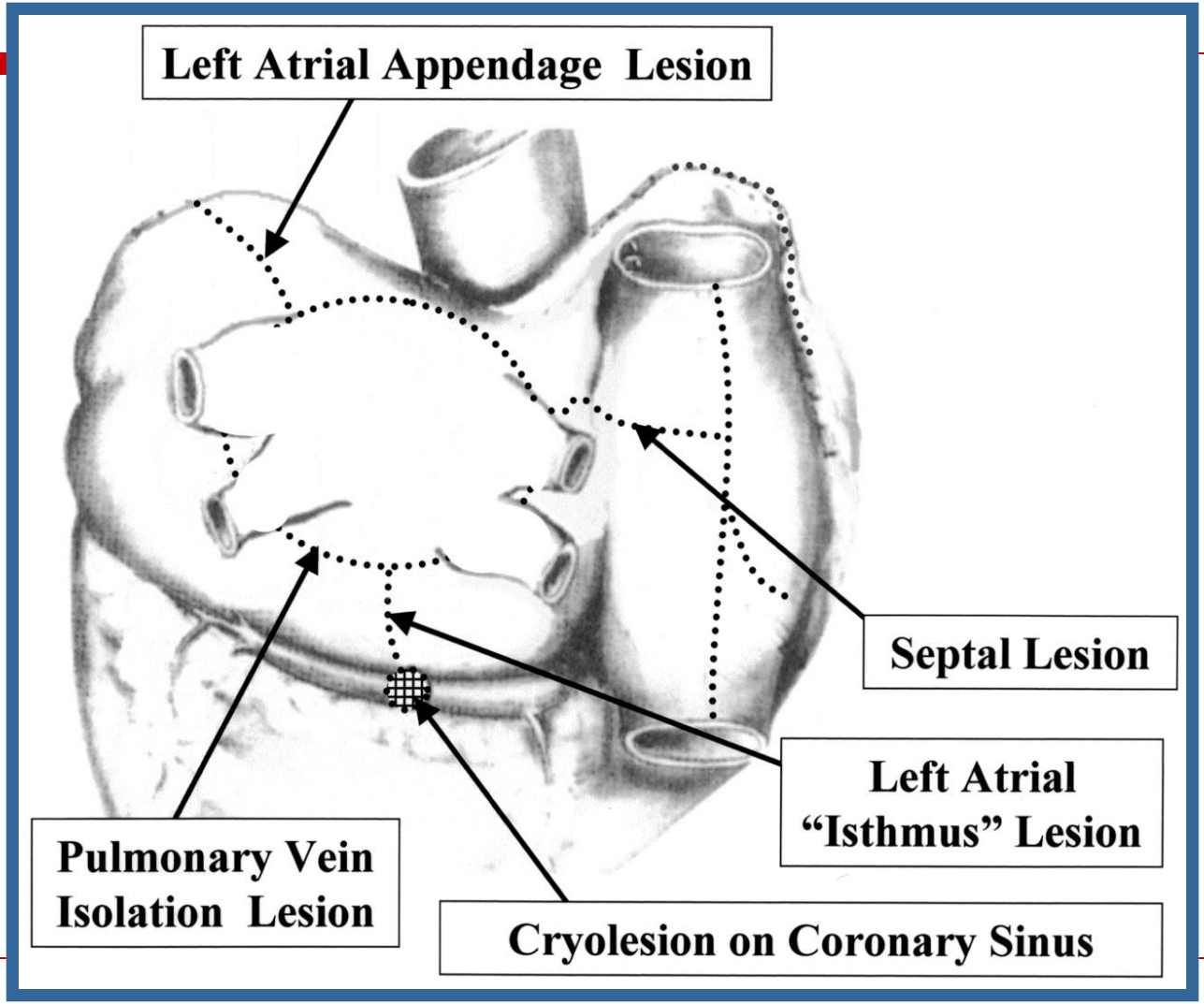
Maze Procedure (1987)

Pathway from the SA to AV Node

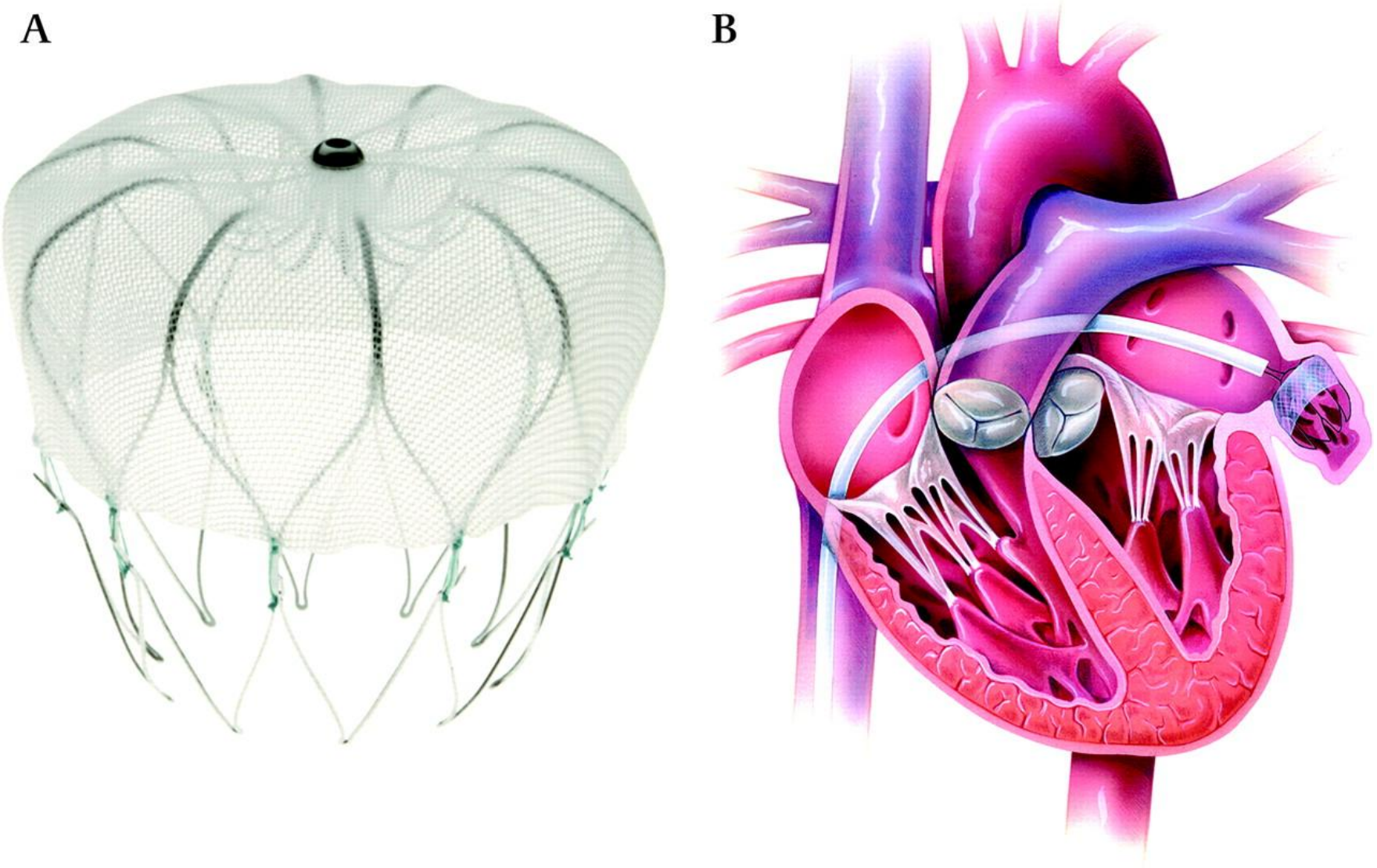
Disrupt Macro-reentrant Circuits

Allow Activation of All Atrial Tissue

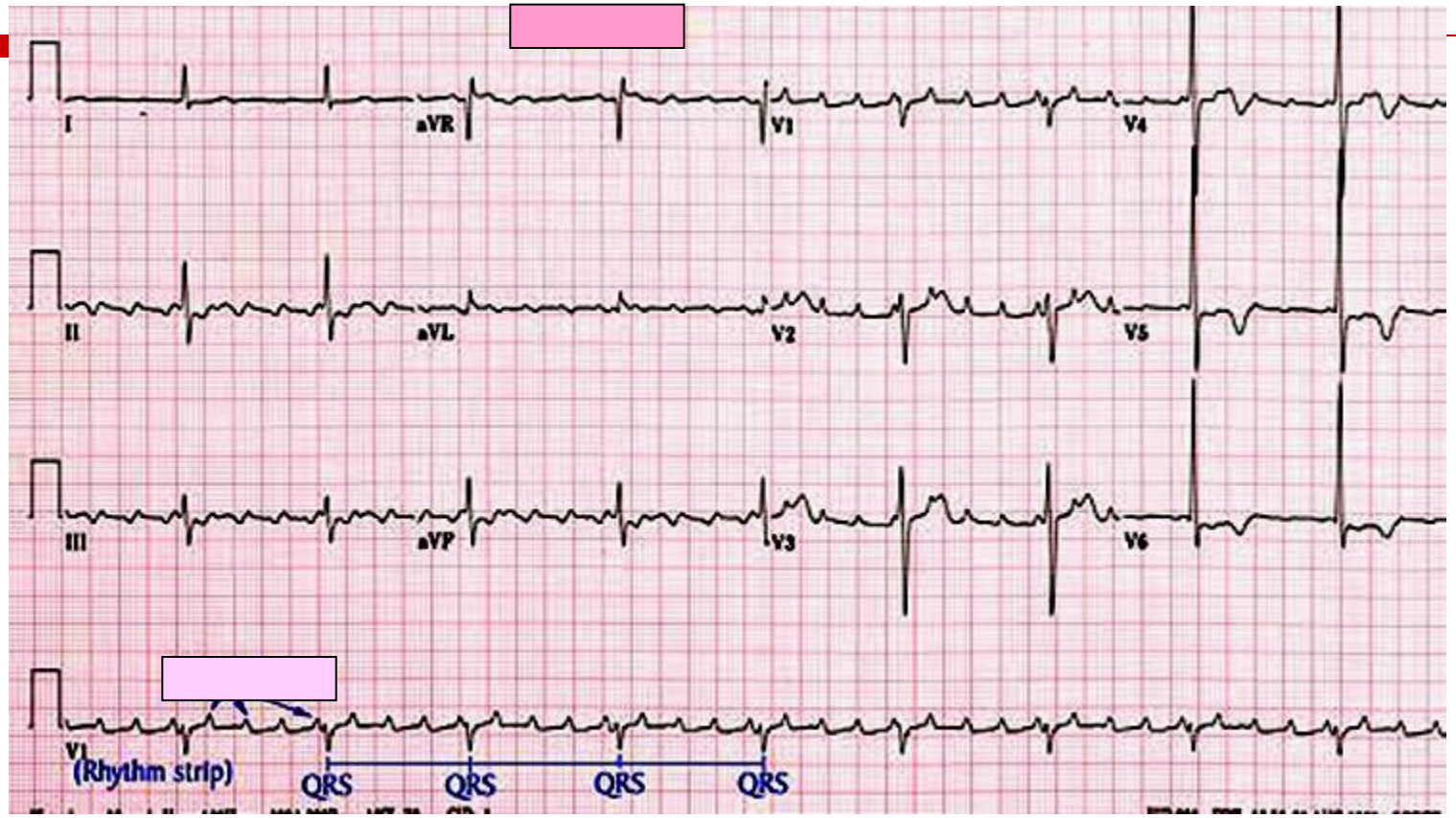
Maze

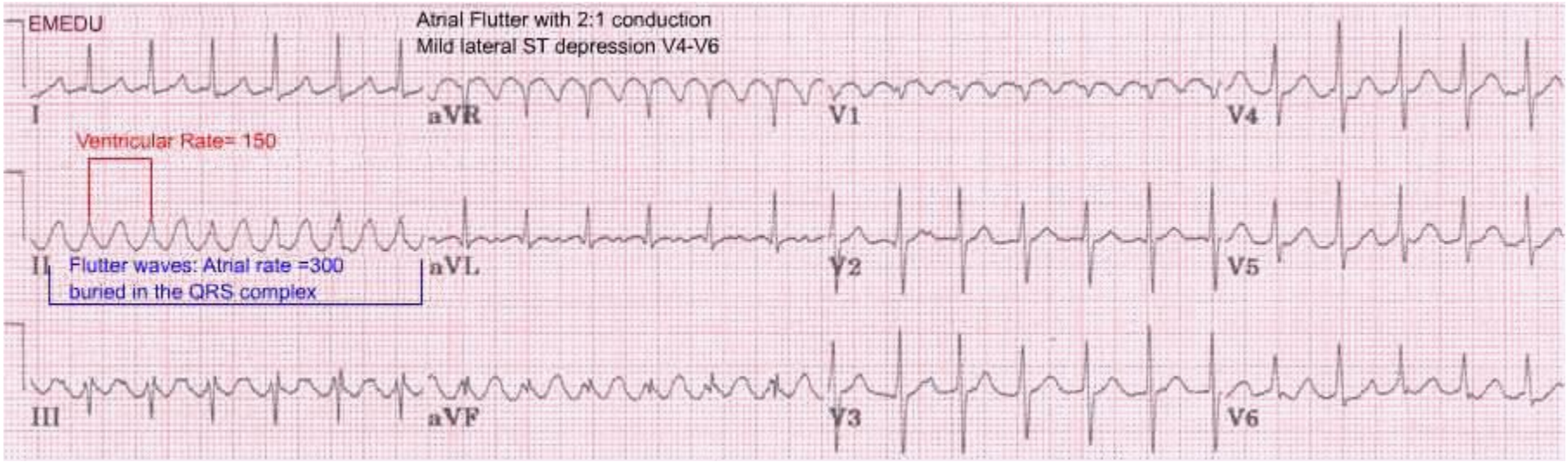

Left Atrial Appendage Lesion
Septal Lesion
Left Atrial "Isthmus" Lesion
Pulmonary Vein Isolation Lesion
Cryolesion on Coronary Sinus

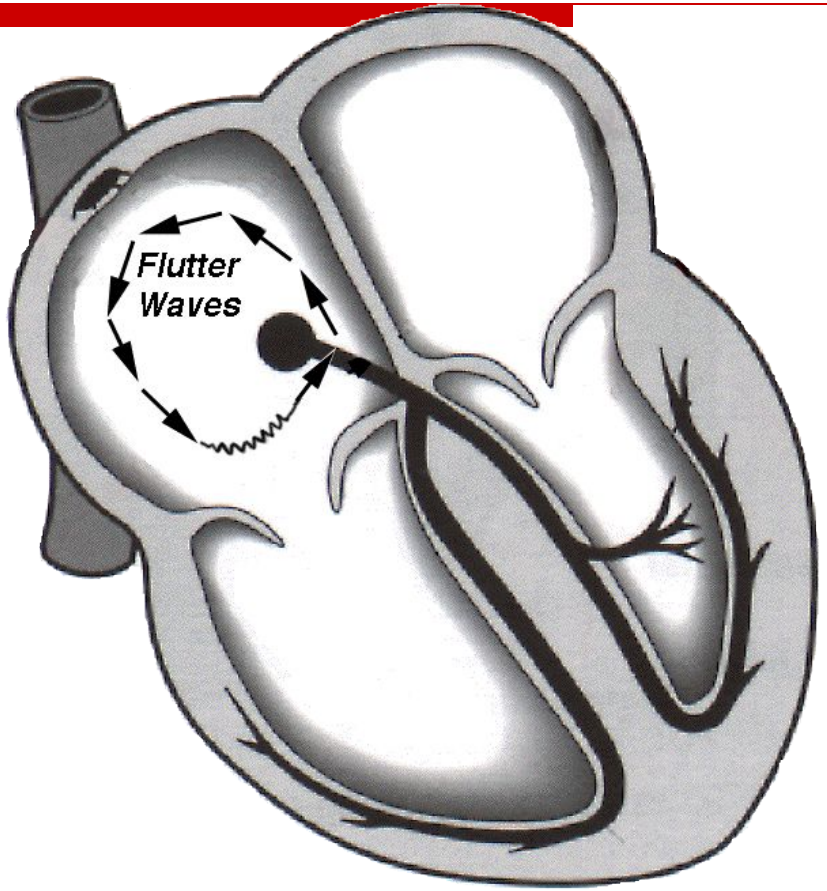
LA appendage closure



Atrial flutter




EMEDU
Atrial Flutter with 2:1 conduction
Mild lateral ST depression V4-V6
I
aVR
V1
V4
Ventricular Rate= 150
II
Flutter waves: Atrial rate =300
buried in the QRS complex
aVL
V2
V5
III
aVF
V3
V6

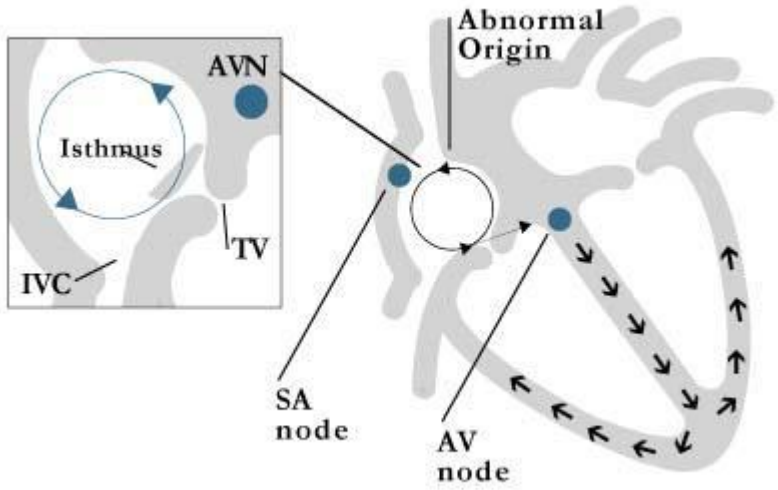

Flutter
Waves

Management

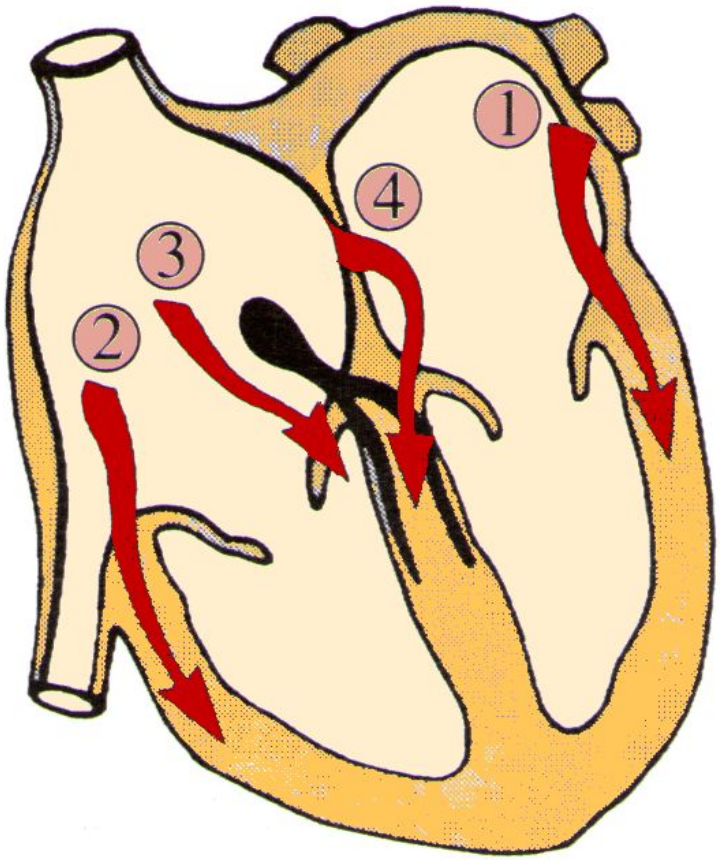
- Electric Cardioversion
- Slowing Ventricular rate
  - Beta Blockers
  - Ca Channel blocker
  - Digoxin
- Propafenon or Flecainaide

Prevention

- Isthmus ablation

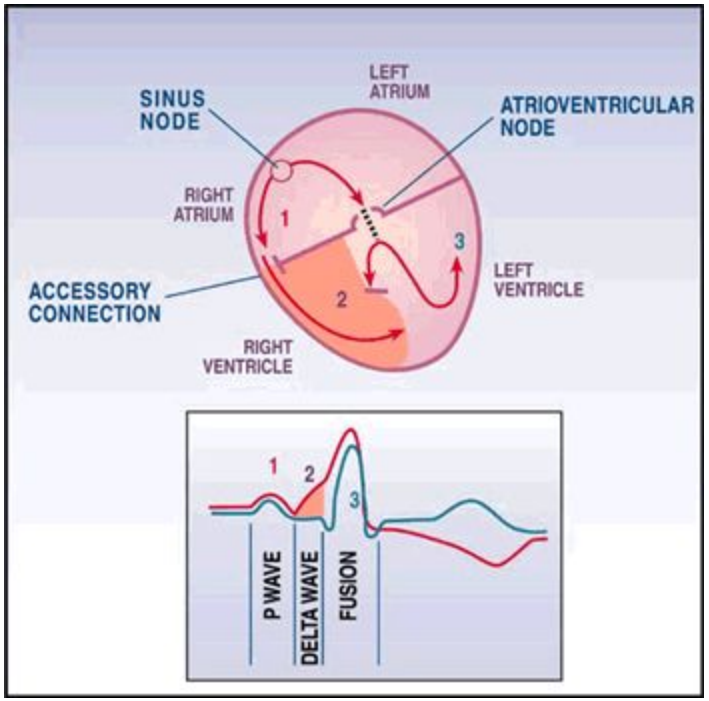


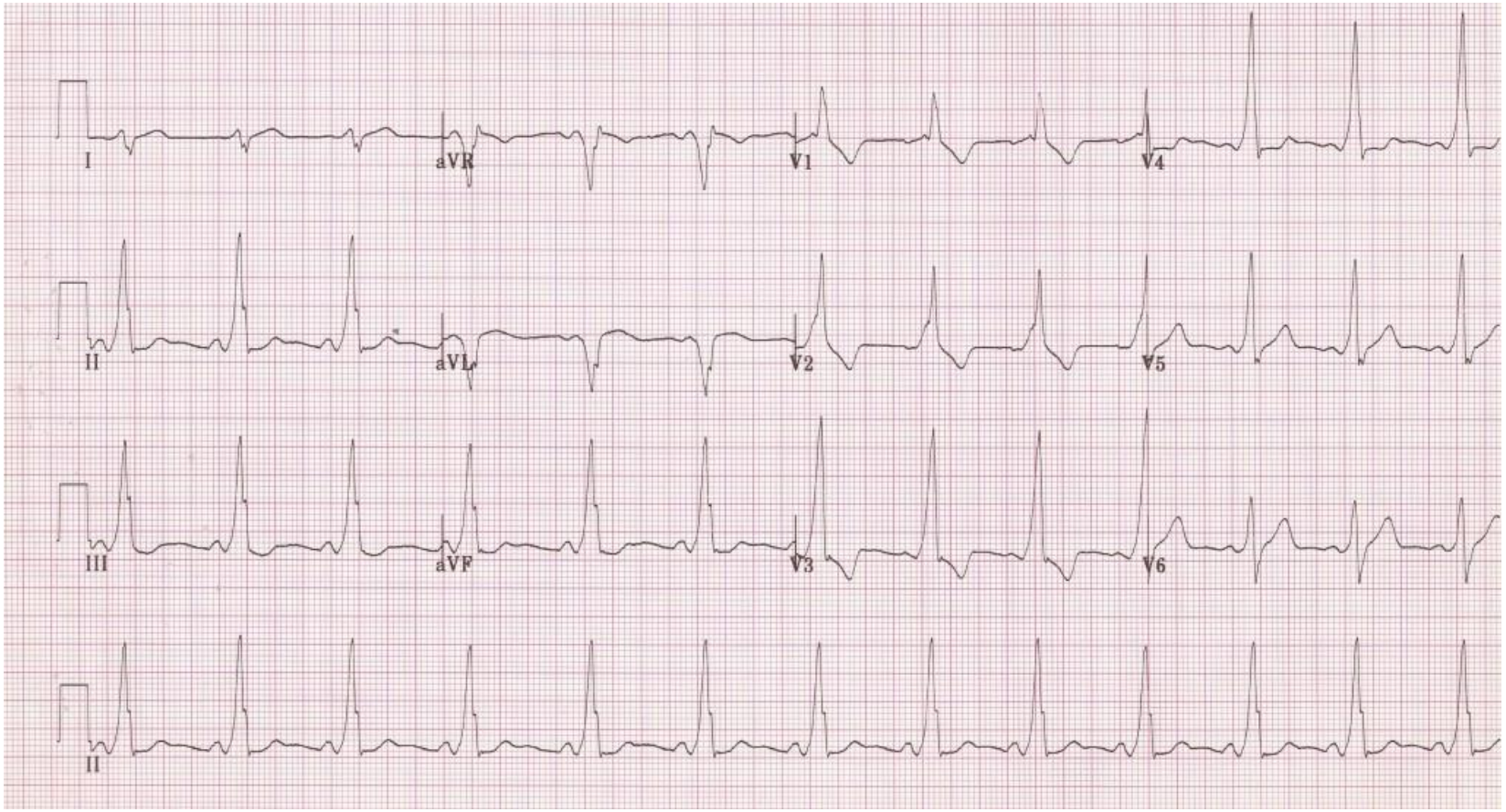
Preexitation – WPW syndrome (accessory pathway(



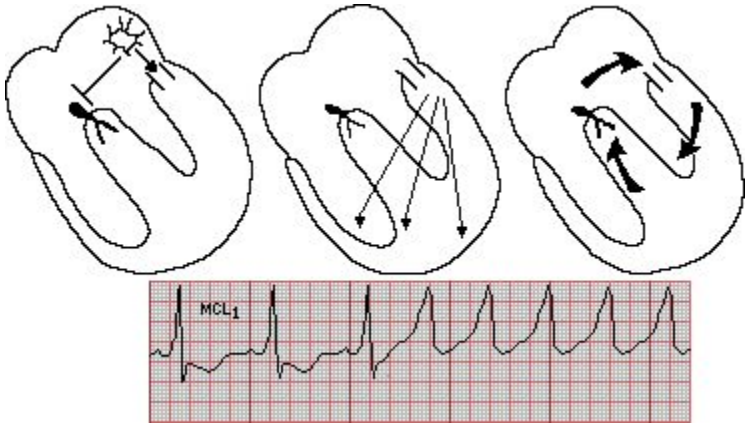
AVRT

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

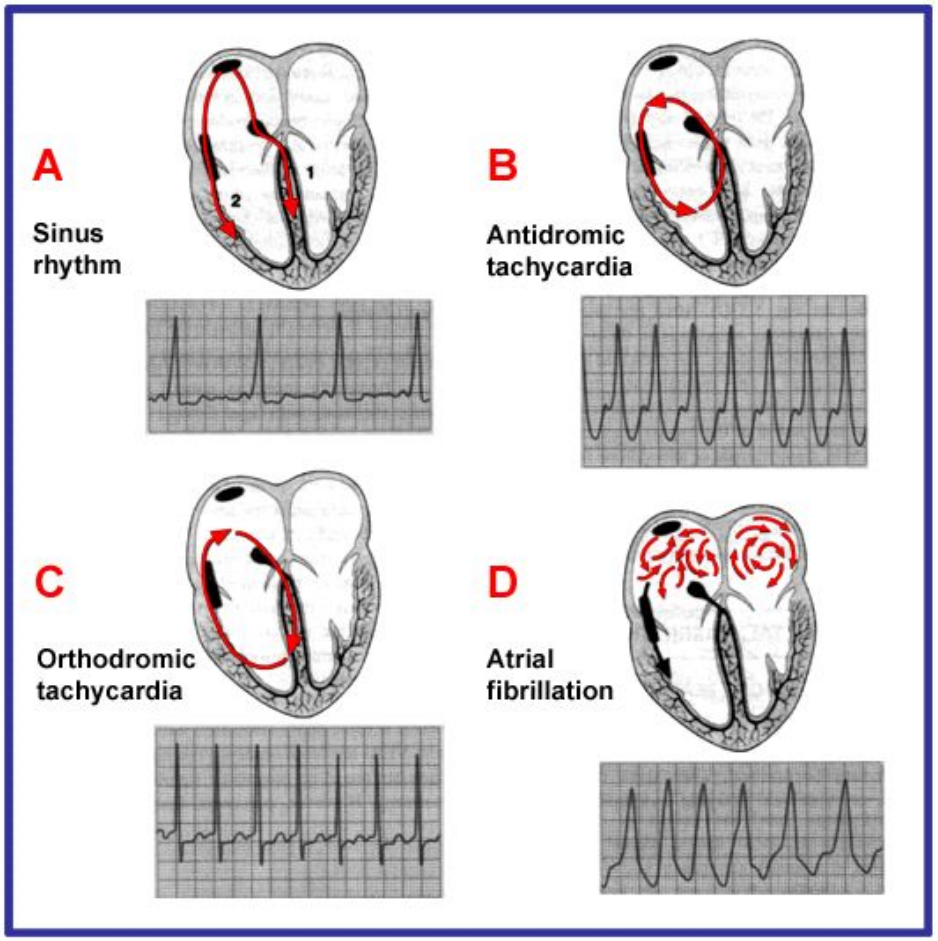



I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

AVRT



AVRT


A
Sinus rhythm
1
2
B
Antidromic tachycardia
C
Orthodromic tachycardia
D
Atrial fibrillation

Treatment

- Acute treatment:

  Wide complex – Procainamide

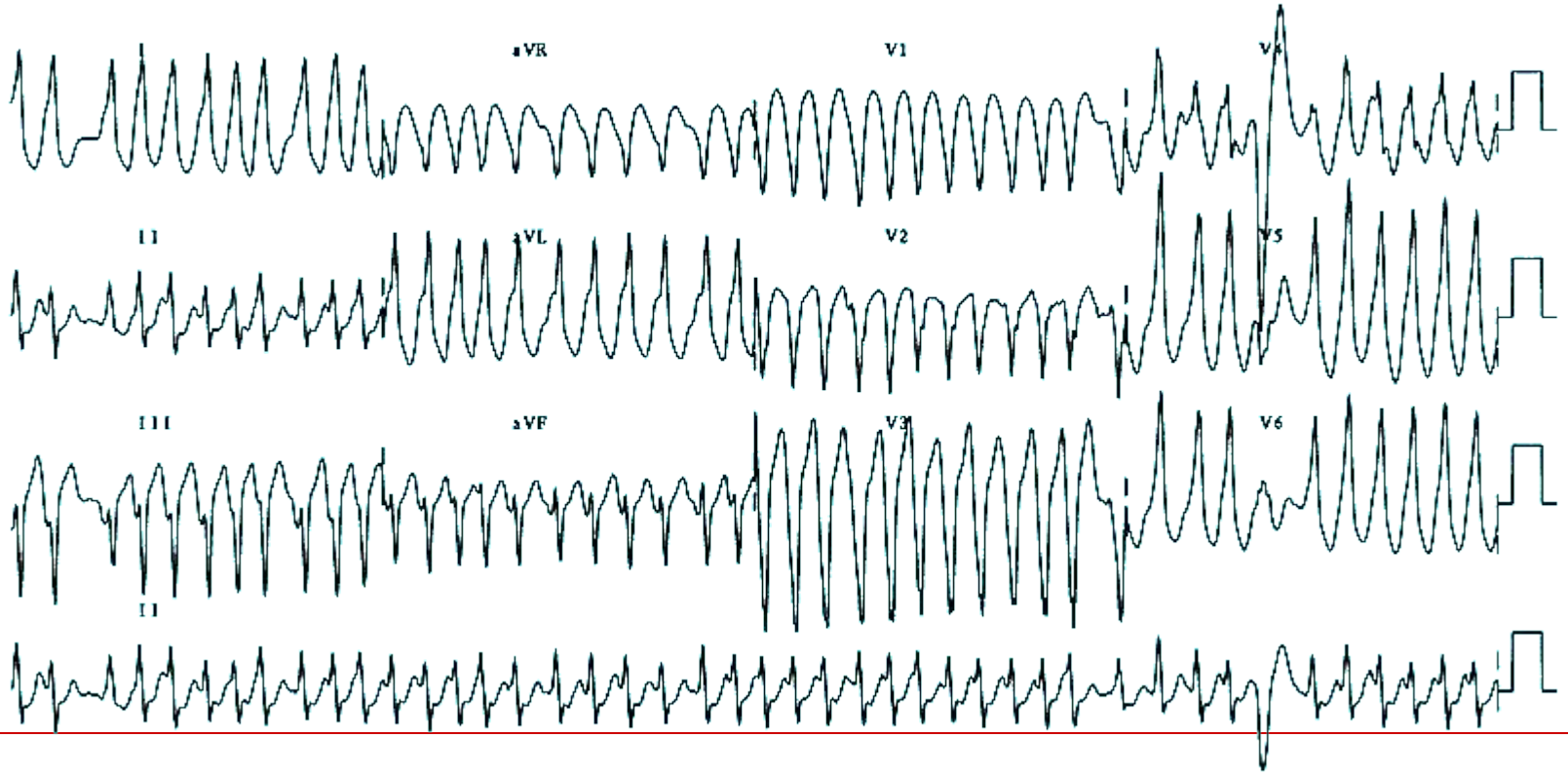
  DC Shock

  Narrow complex – Verapamil,

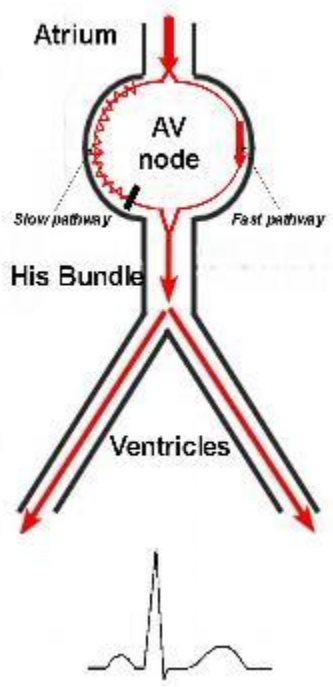
  Beta Blockers

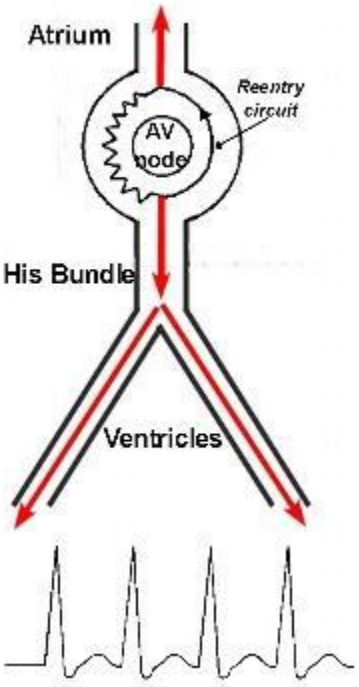
- Preventive treatment : accessory pathway ablation

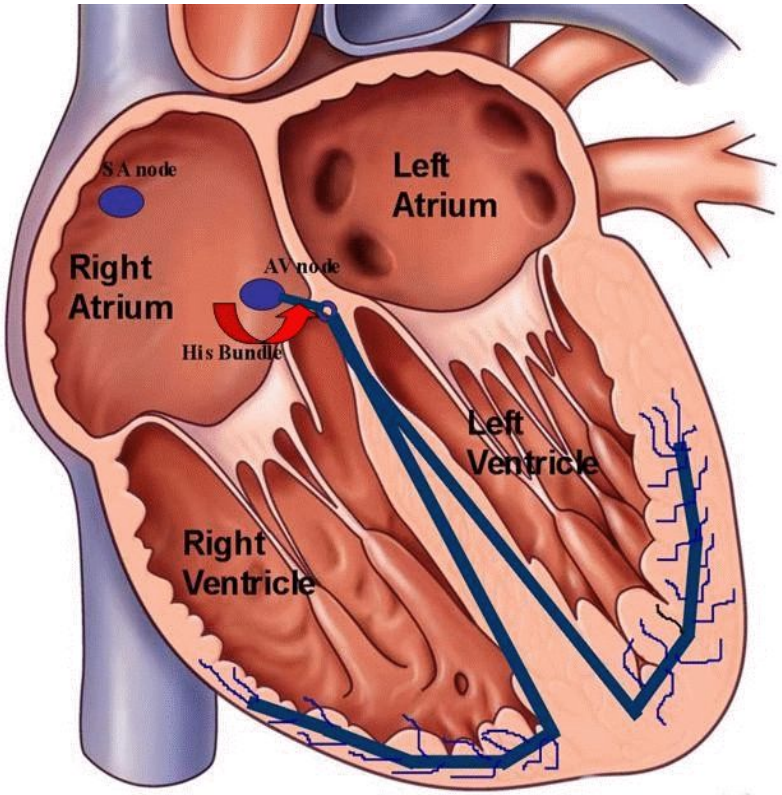
- AF with WPW – high risk of VF



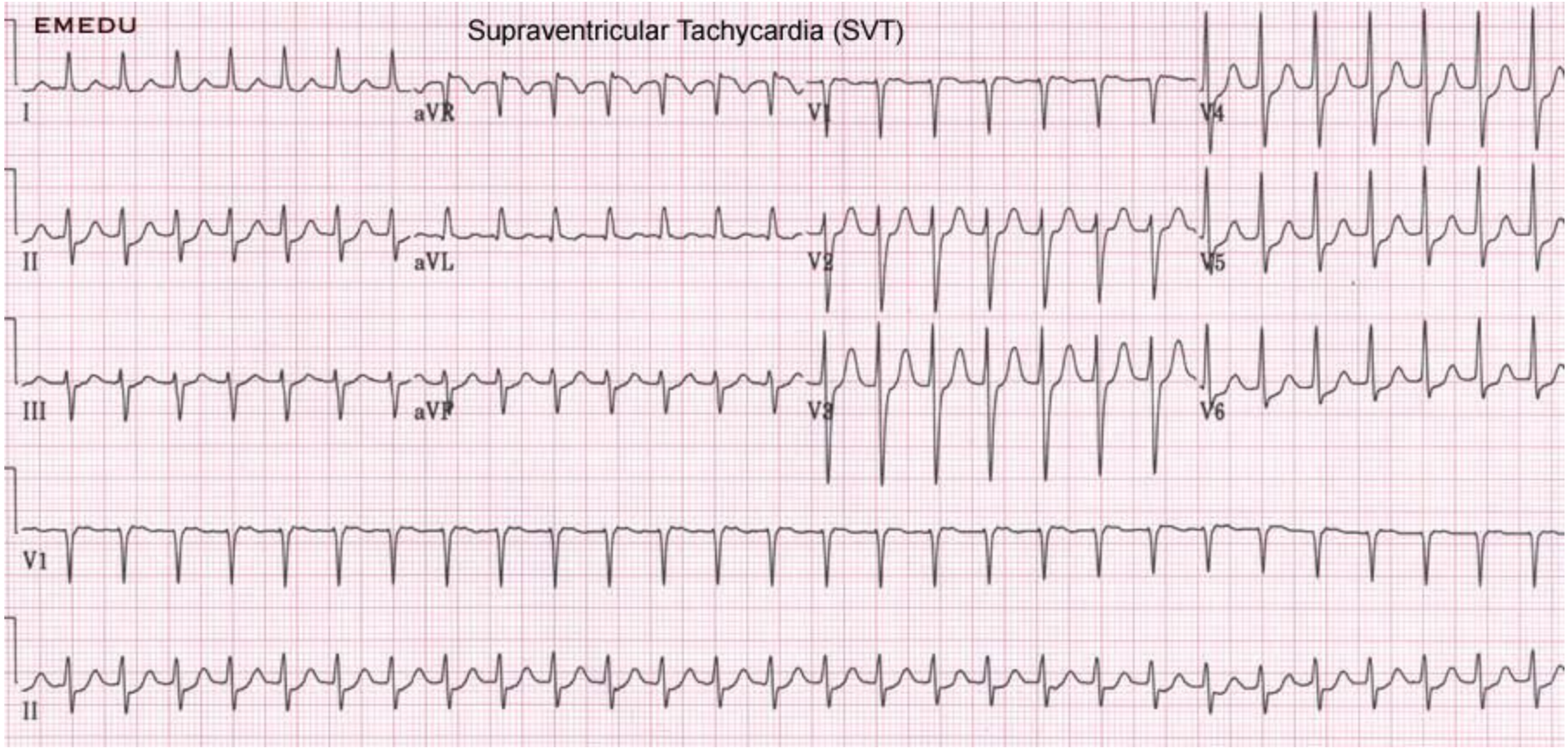
Double A-V nodal physiology






SA node
Left
Atrium
Right
Atrium
AV node
His Bundle
Left
Ventricle
Right
Ventricle

AVNRT


EMEDU
Supraventricular Tachycardia (SVT)
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II

Management of narrow complex SVT

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

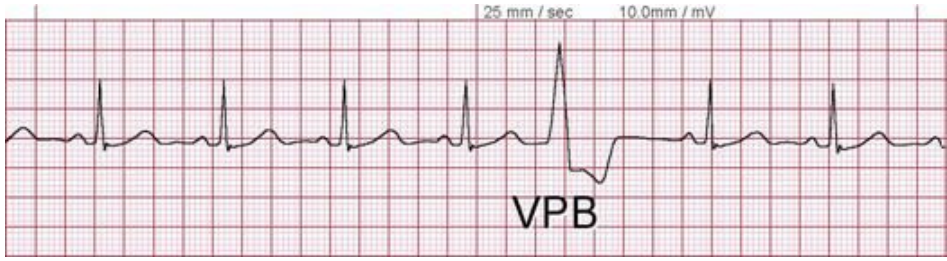
Preventive treatment

- Drugs
- EPS

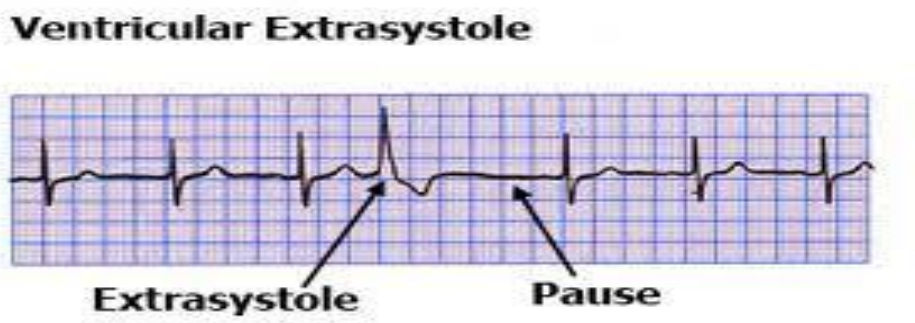
Ventricular Arrhythmias

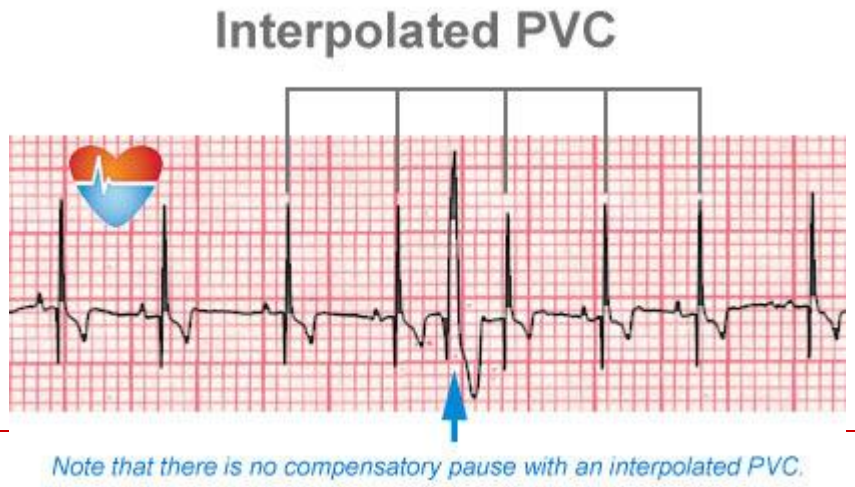
Ventricular premature beats
Ventricular premature complexes

- 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

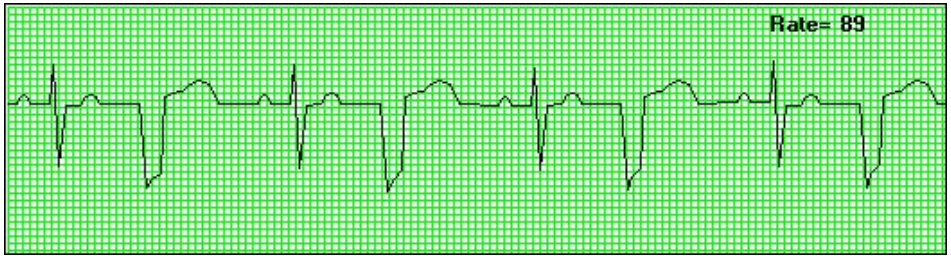


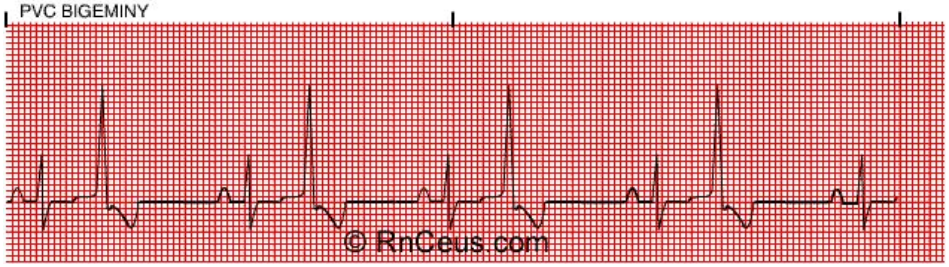
Compensatory pause



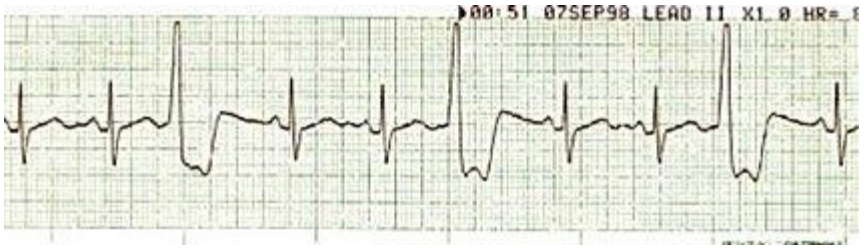


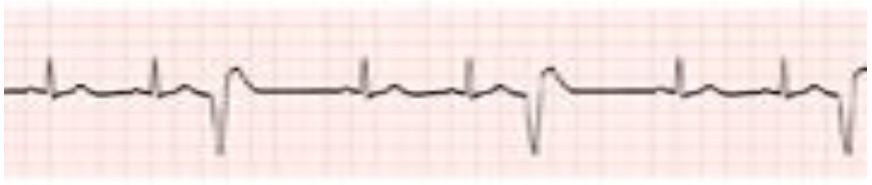
Bigeminy



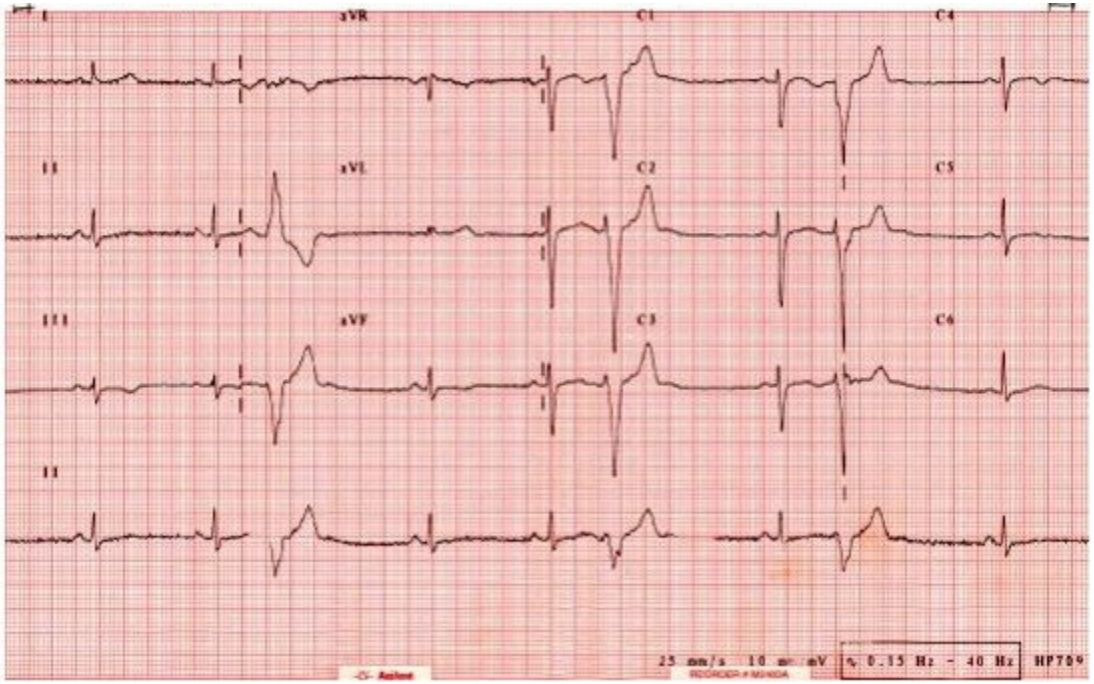


Trigeminy

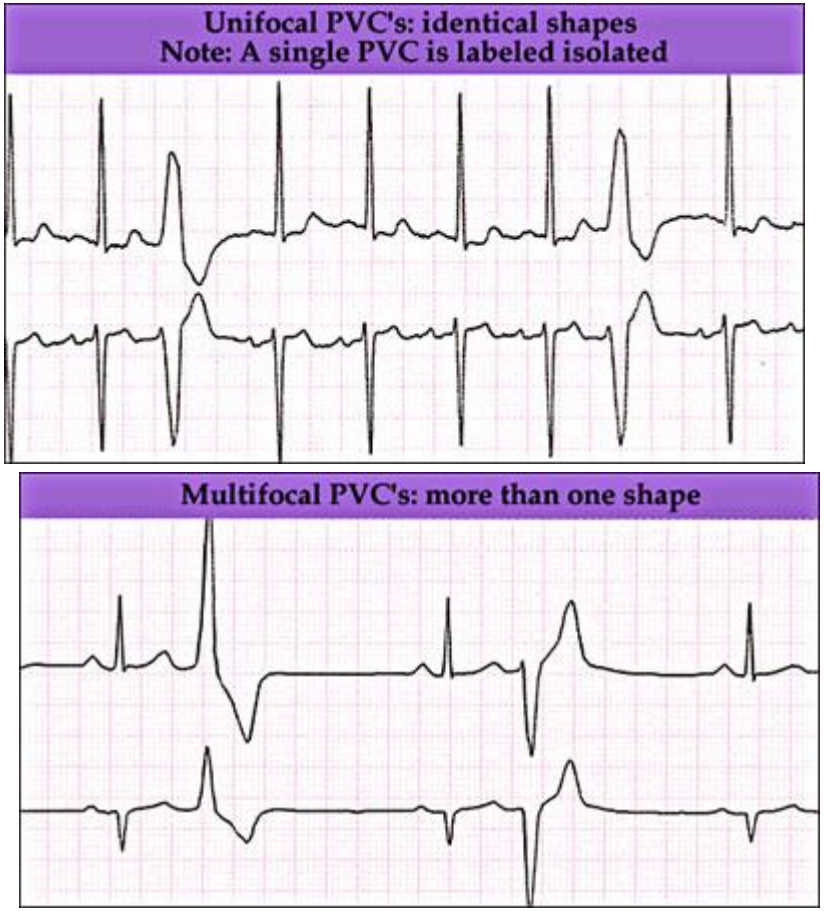




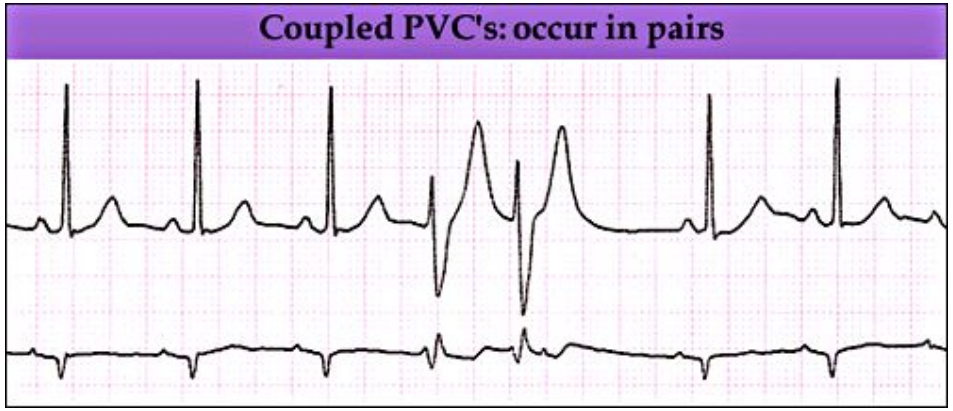
VPB’s

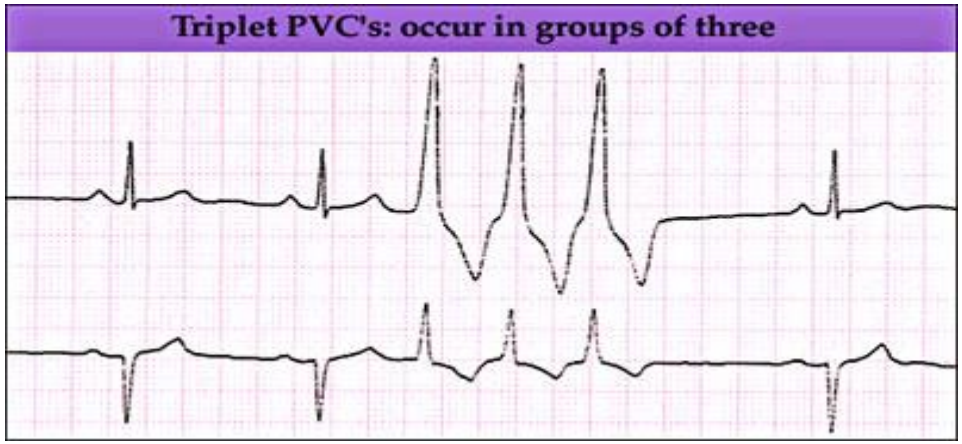

I
aVR
C1
C4
II
aVL
C2
C5
III
aVF
C3
C6
II
25 mm/s 10 mm mV
0.15 Hz - 40 Hz
HP709

Unifocal & Multifocal



Couplet & Triplet





Causes

- LV false tendons,
- infection
- in ischemic or inflamed myocardium,
- hypoxia,
- Anesthesiaor
- 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

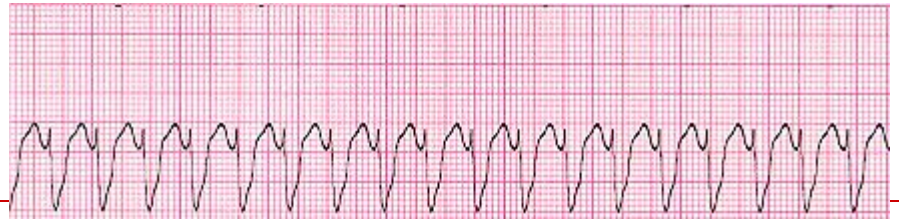
VT

Definition:

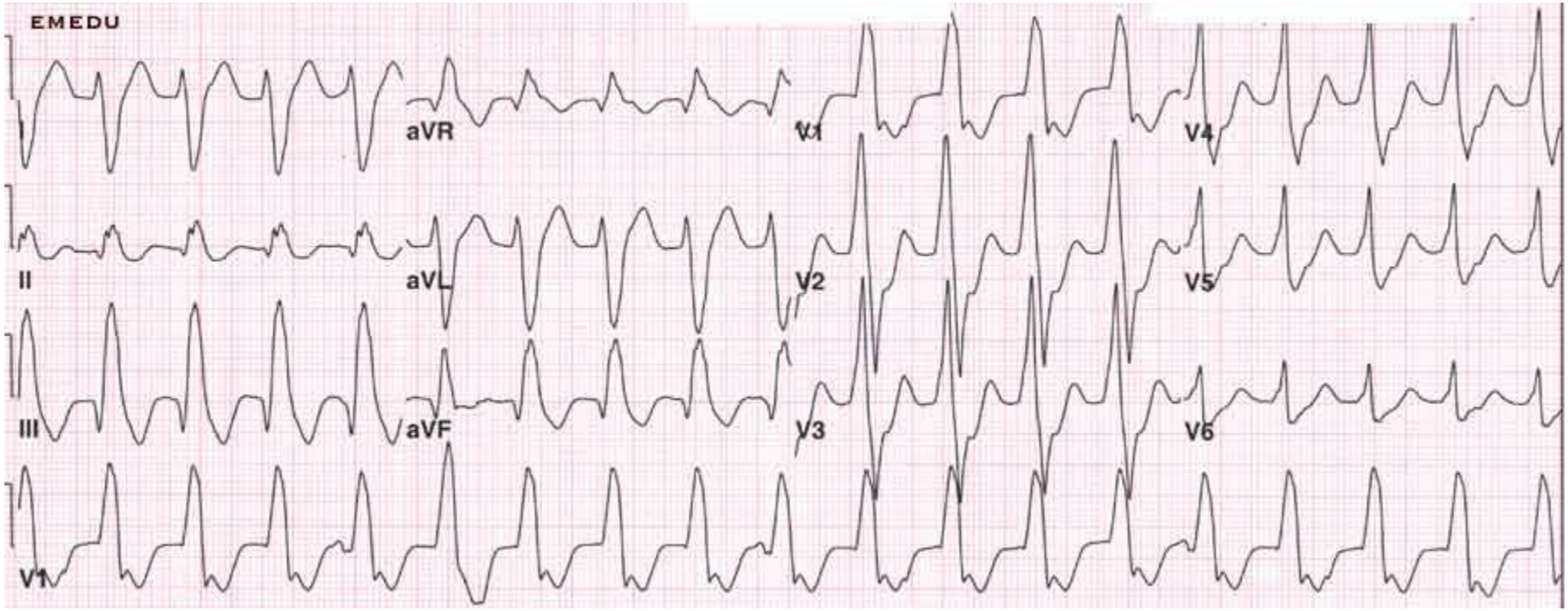
Ventricular tachycardia consist of at least three consecutive QRS complexes originating from the ventricles and recurring at a rapid rate (> 100 bpm).

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

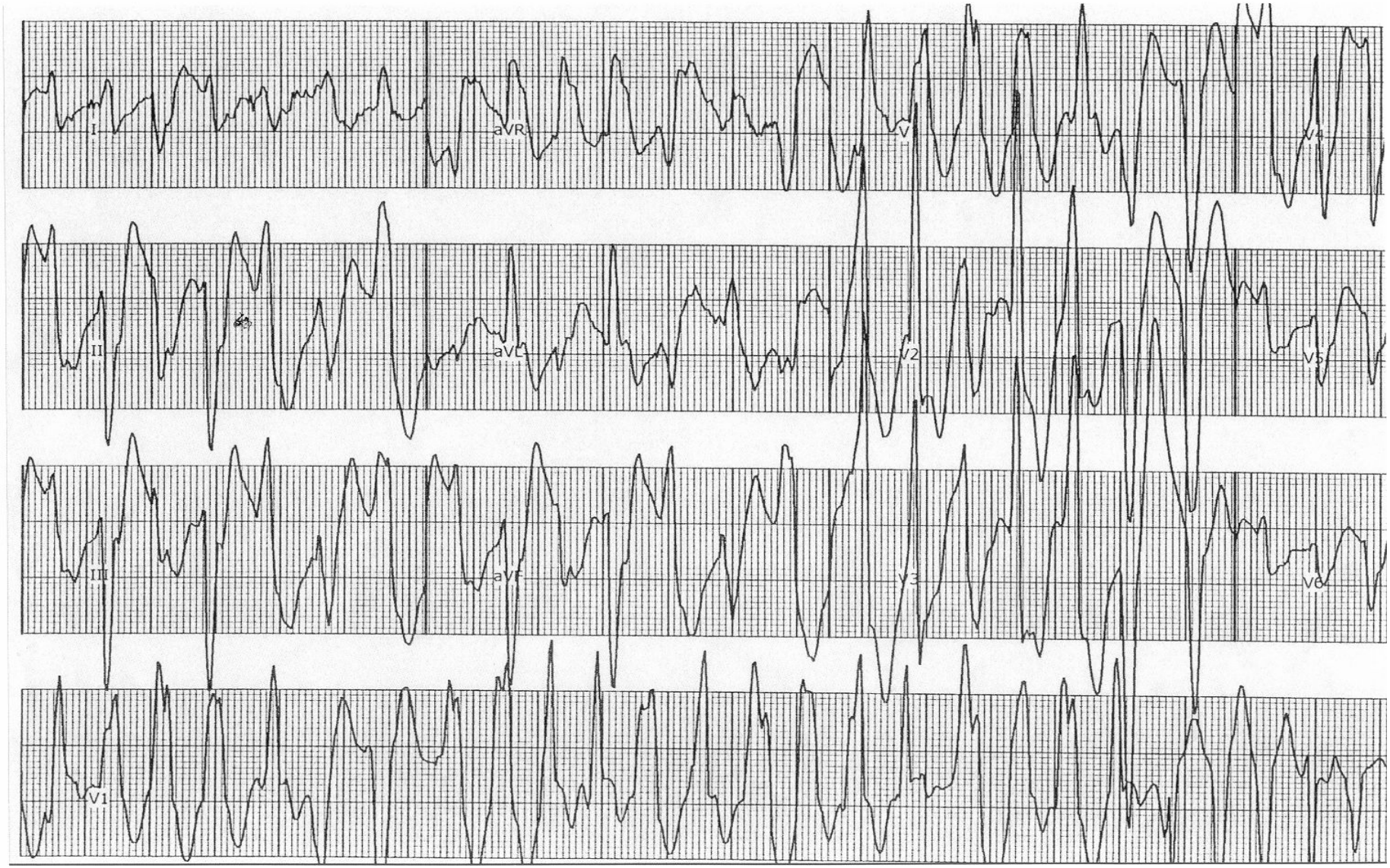
The rhythm is generally regular or slightly irregular.



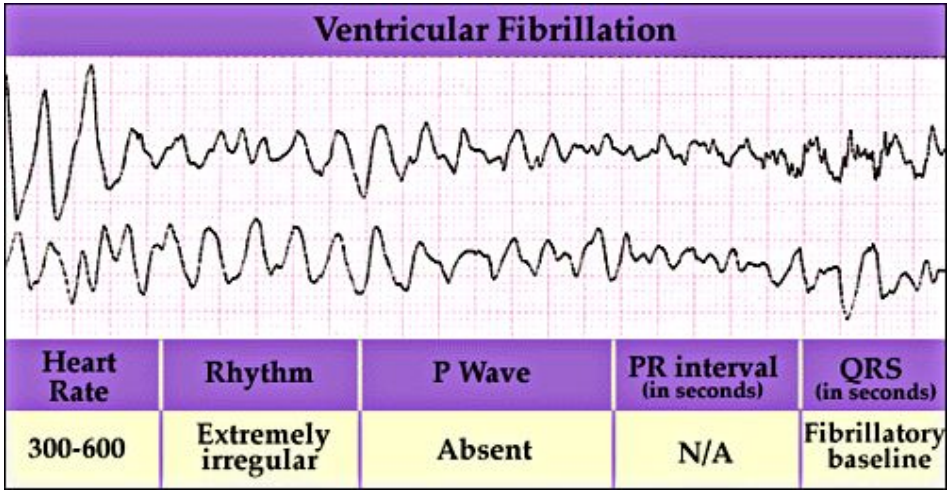
VT -monomorphic



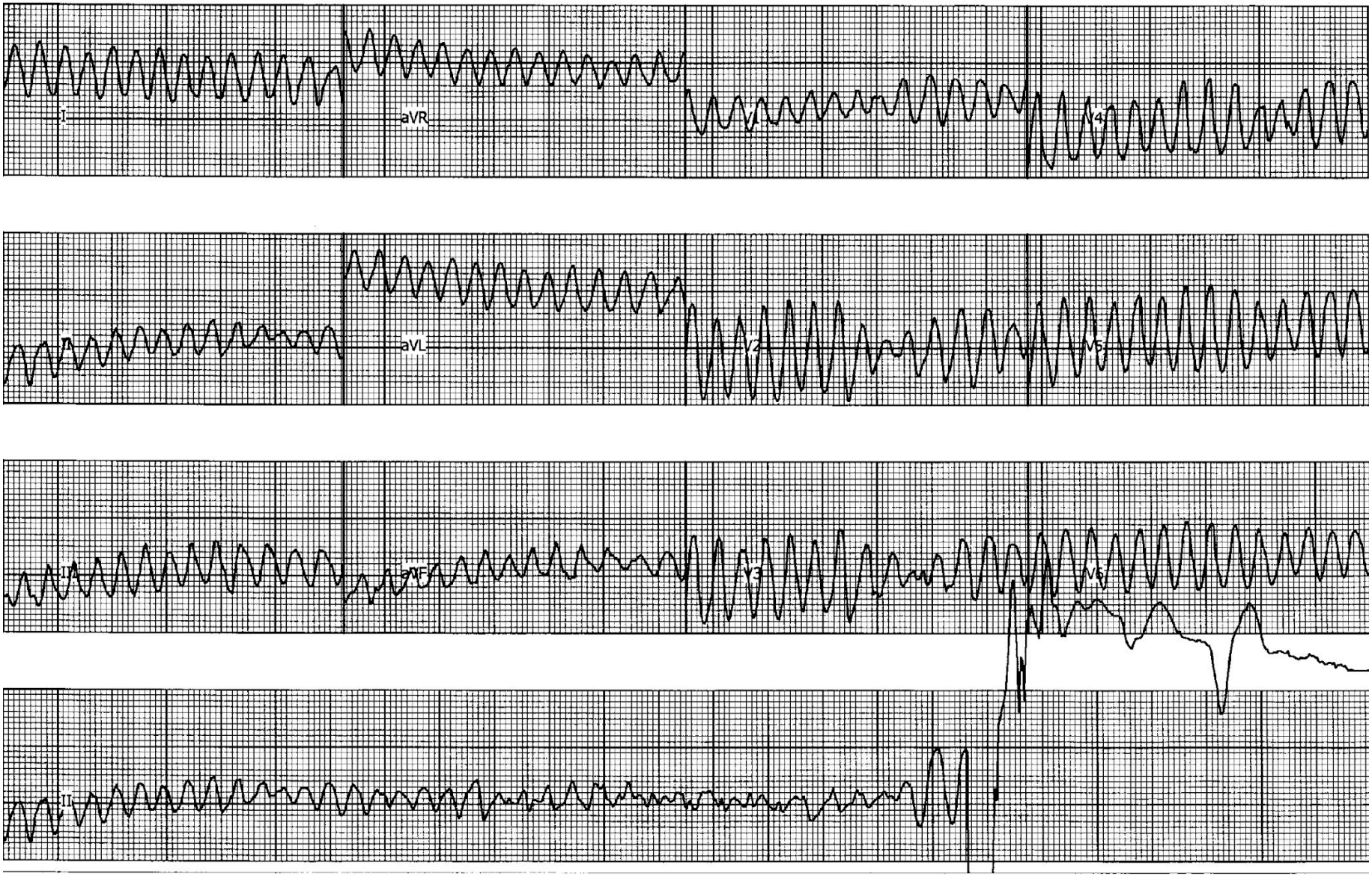
Sustained Polymorphic VT



VF


Ventricular Fibrillation
Heart Rate
Rhythm
P Wave
PR interval (in seconds)
QRS (in seconds)
300-600
Extremely irregular
Absent
N/A
Fibrillatory baseline

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 pfarmacotherapy ineffective – DC shock (synchronized)

  Ventricular pacing
- **Hemodinamically unstable – Immediate DC shock**

Polymorphic VT

- Polymorphic VT with long QT – *Torsades de pointes*

  Treatment – Mg , Pacing

- Polymorphic VT w/o long QT

  Antyarrhytmic 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 P_{ETCO_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 P_{ETCO_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

1 **Start CPR**
- Give oxygen
- Attach monitor/defibrillator

Rhythm shockable?

Yes → 2 **VF/VT** → 3 **Shock** → 4 **CPR 2 min**
- IV/IO access

Rhythm shockable?

No → 9 **Asystole/PEA** → 10 **CPR 2 min**
- IV/IO access
- **Epinephrine** every 3-5 min
- Consider advanced airway, capnography

Rhythm shockable?

Yes → 5 **Shock** → 6 **CPR 2 min**
- **Epinephrine** every 3-5 min
- Consider advanced airway, capnography

Rhythm shockable?

No → 11 **CPR 2 min**
- Treat reversible causes

Rhythm shockable?

Yes → 7 **Shock** → 8 **CPR 2 min**
- **Amiodarone**
- Treat reversible causes

Rhythm shockable?

No → Yes

Chronic Management (secondary prevention)

Evaluation

- - Rest ECG
- - Exersise test
- - Ambulatory ECG
- - Imaging (LV function, CMP, Valves etc…
- - EPS

Treatment of the underlying disease

- Revascularisation
- Valve surgery
- CHD repair

Non-antiarrhythmic Drugs

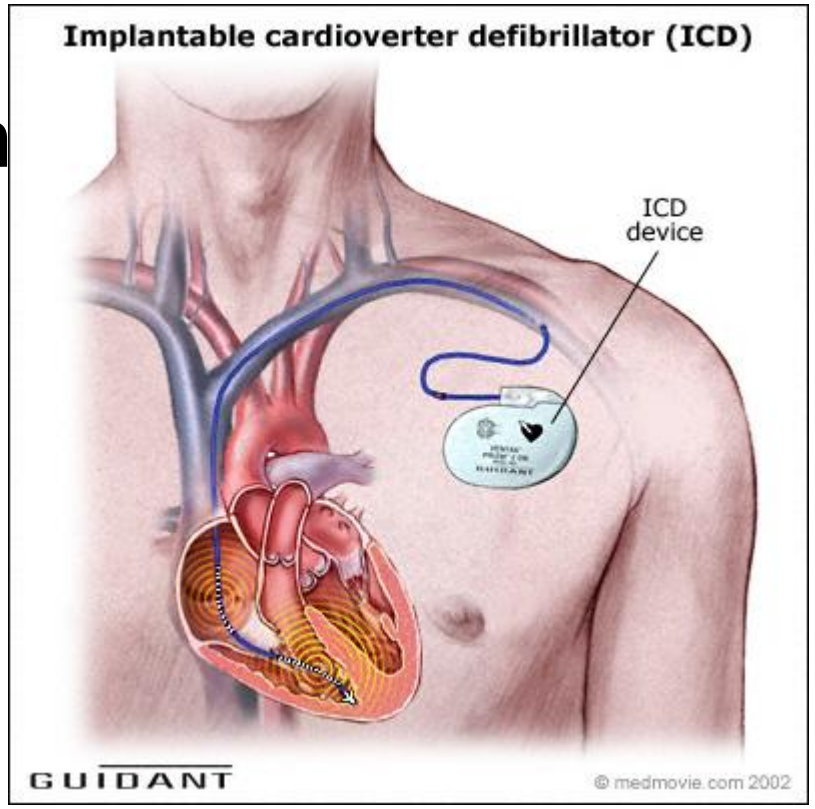
- ❤️ Electrolytes: Mg & K
- ❤️ ACE inhibitors,
- ❤️ Antithrombotic and antiplatelet agents
- ❤️ Statins

Antiarrhytmic 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



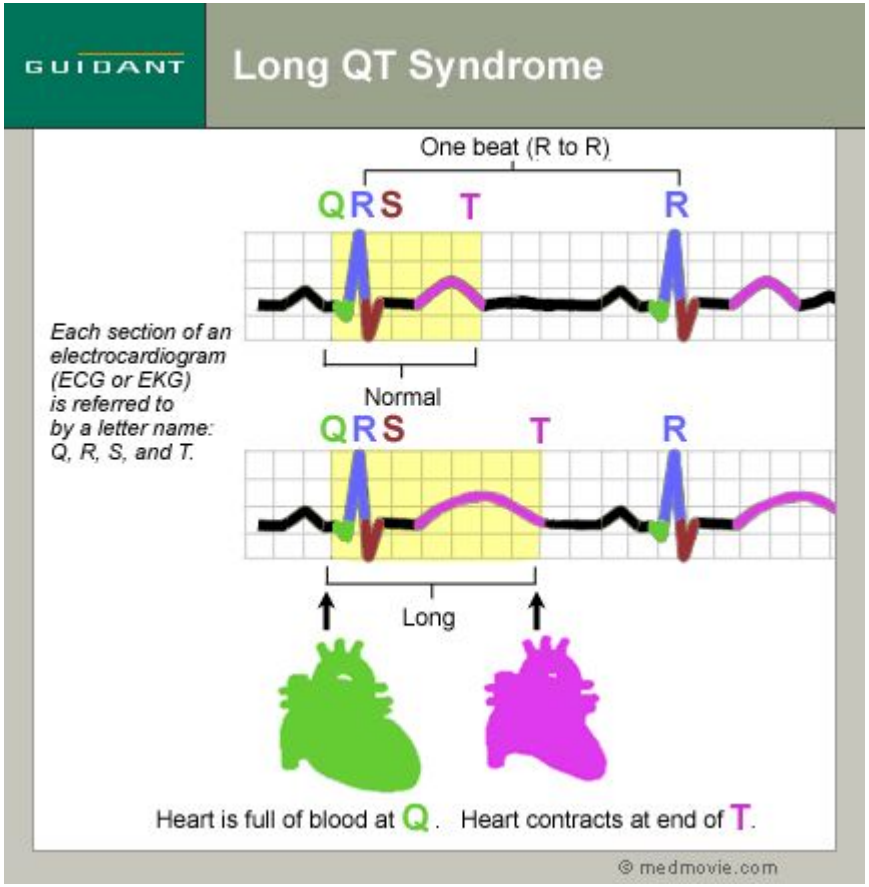
AICD for primary prevention of SCD

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

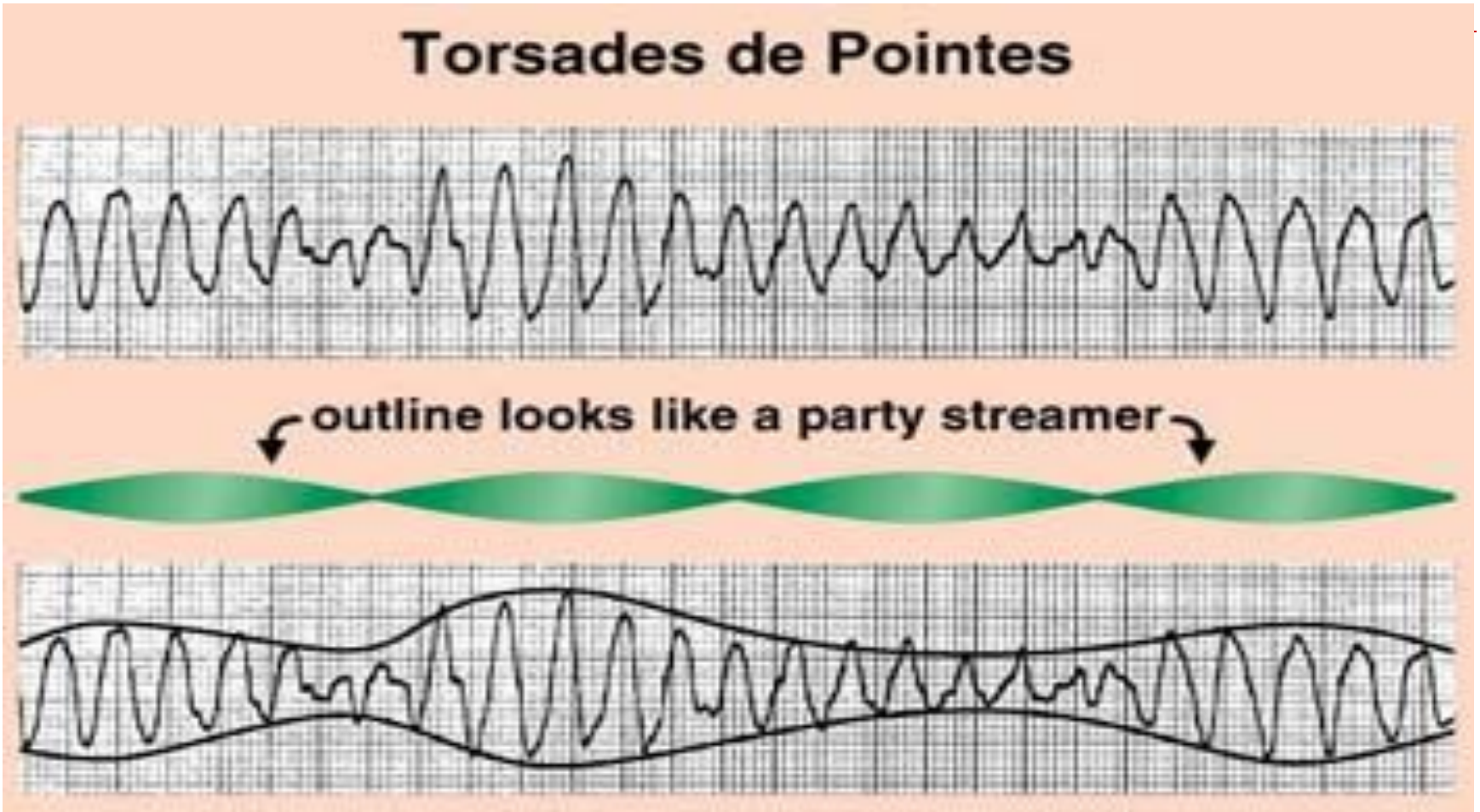
Long QT syndrome

1. Congenital (family)
2. Acquired:

- Electrolyte anomalies – K, Mg
- Drug induced
  - -Antiarrhytmics
  - - Tricyclic antydepressants
  - - Antihistamines
- CNS lesions



Torsades de Pointes



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

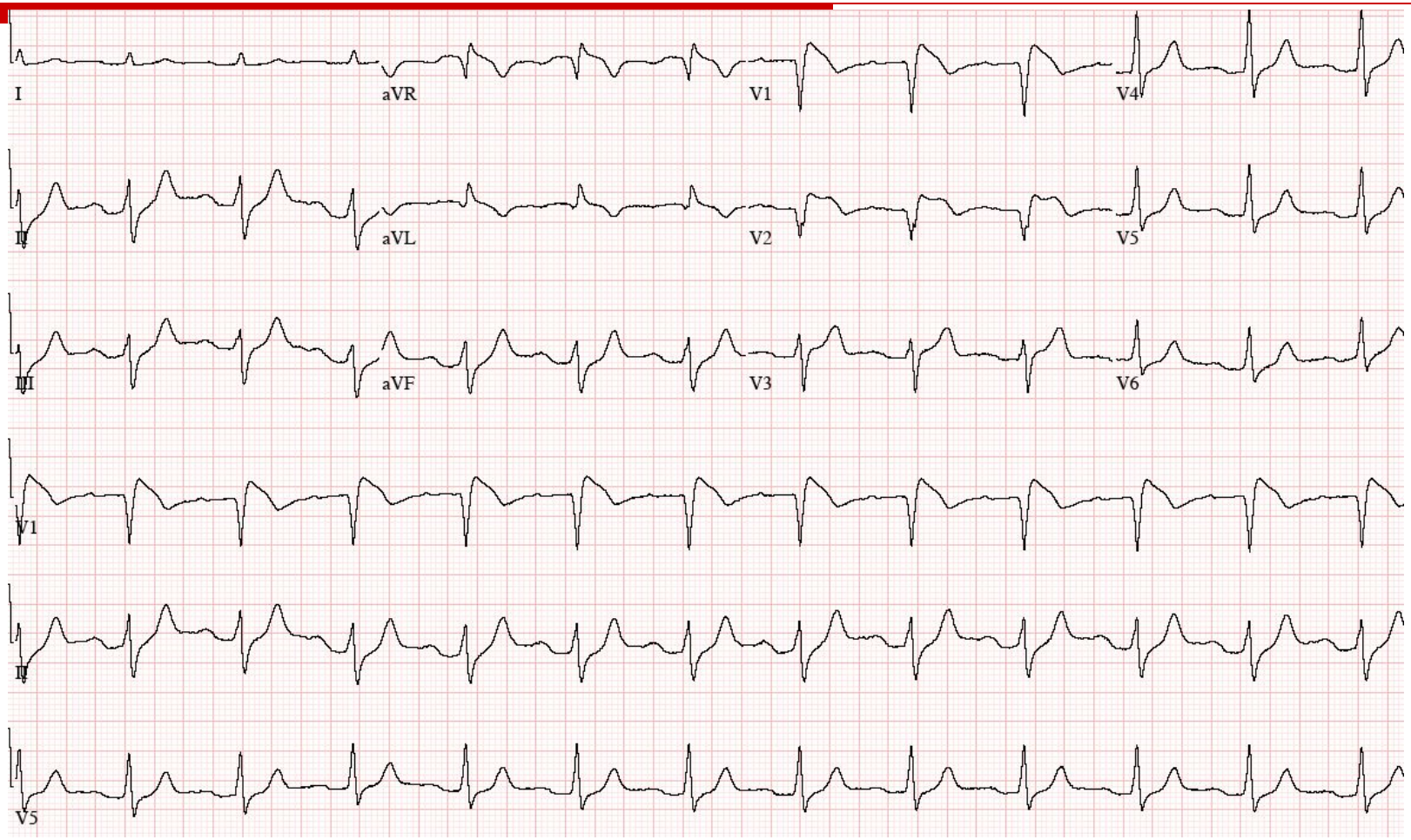
The Brugada Syndrome

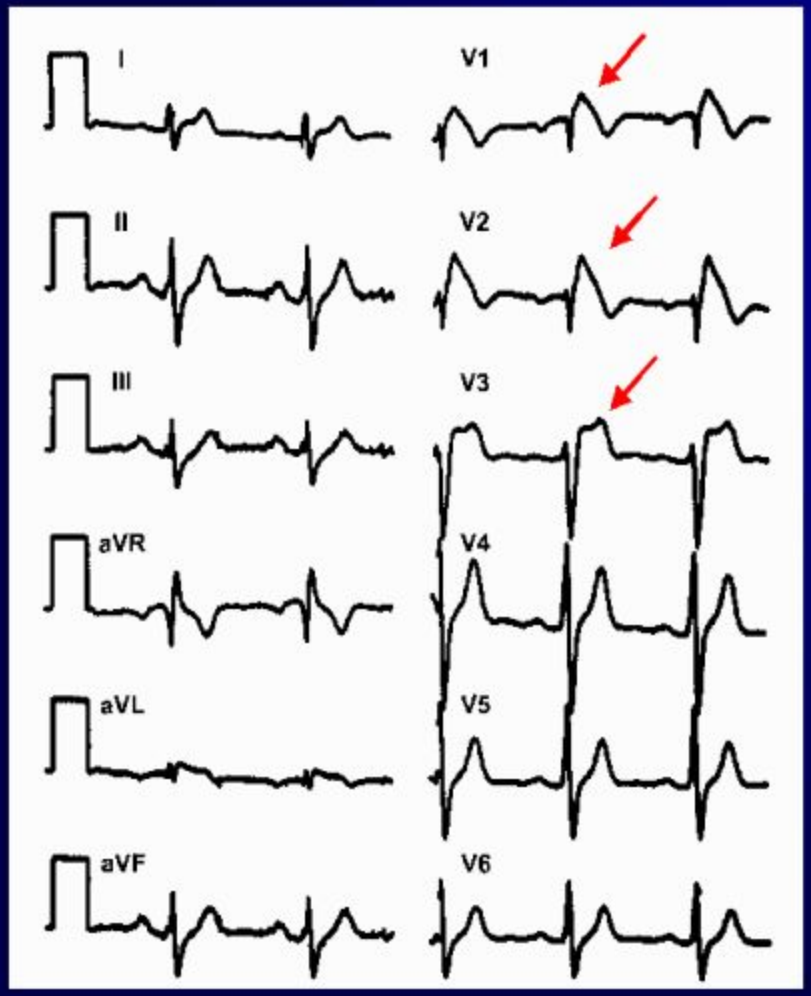
Definition

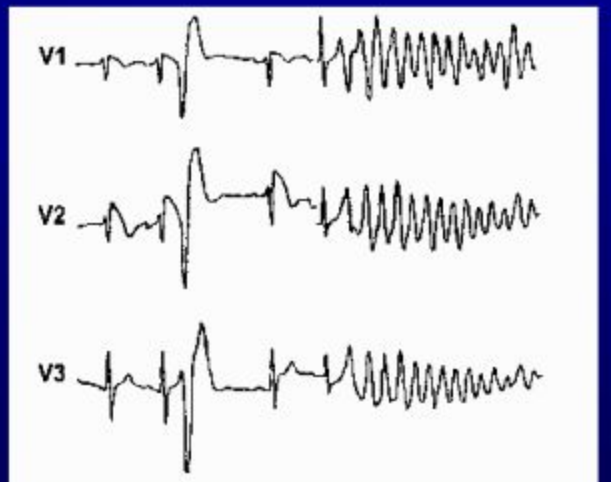
"Syncopal episodes and/or sudden death in patients with a structurally normal heart and a characteristic electrocardiogram displaying a pattern resembling right bundle branch block with an ST segment elevation in leads V1 to V3"

Brugada et al. Circ 1992

Brugada syndrome



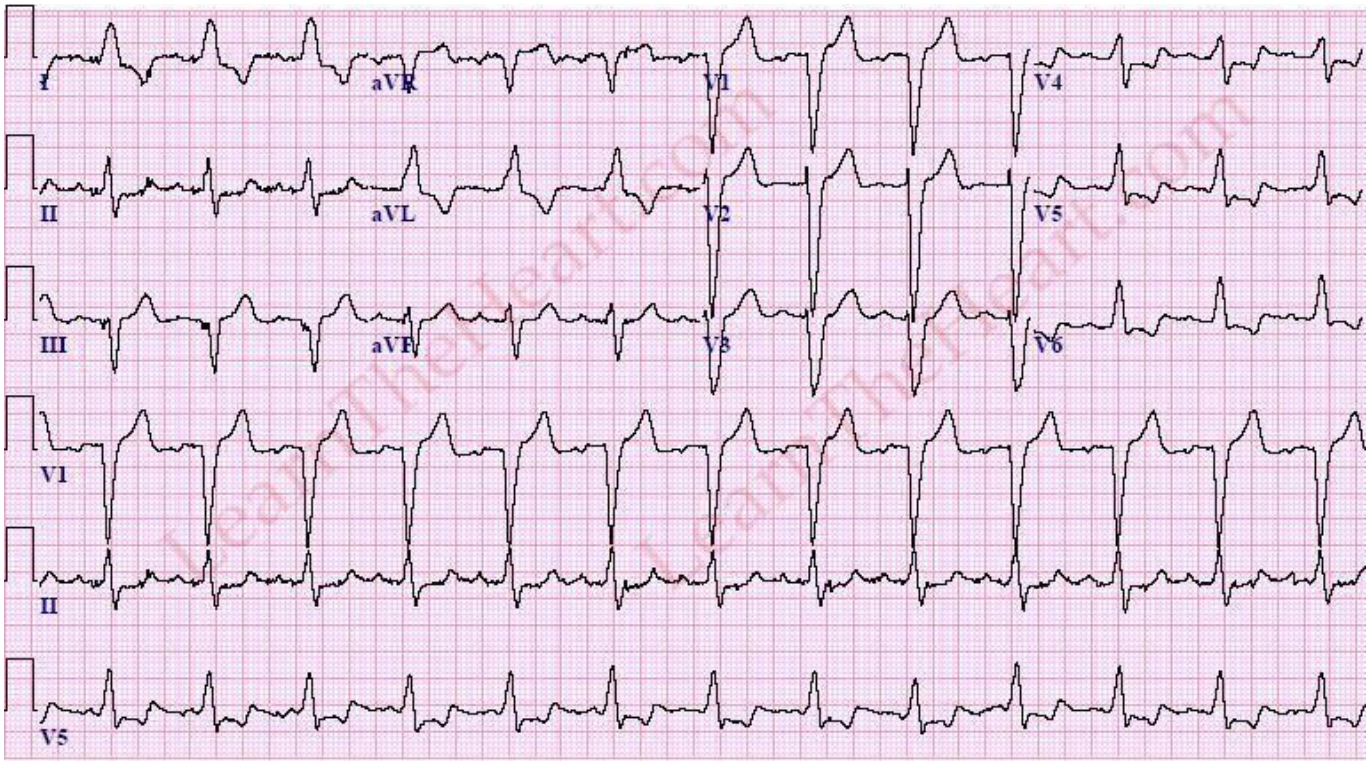

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6


V1
V2
V3

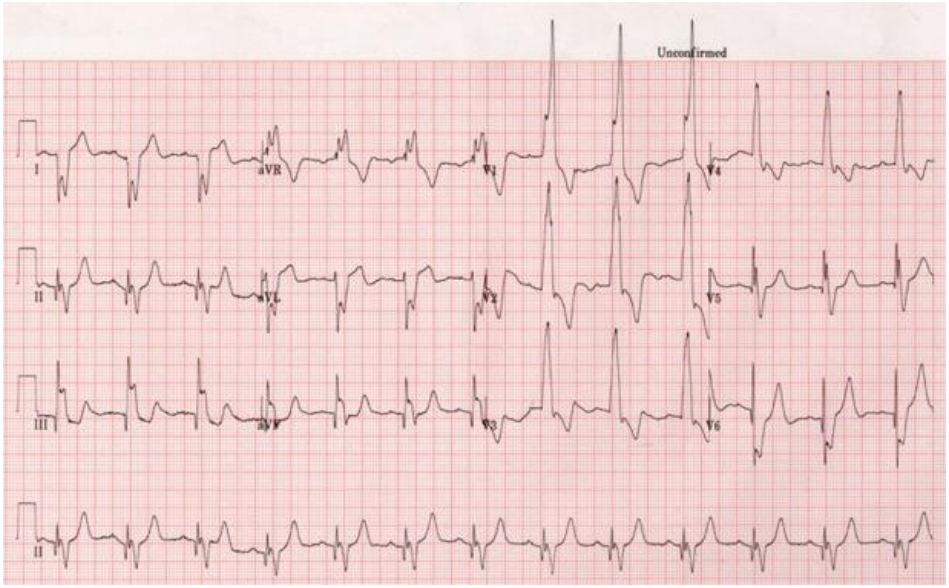
- **Typical presentation**

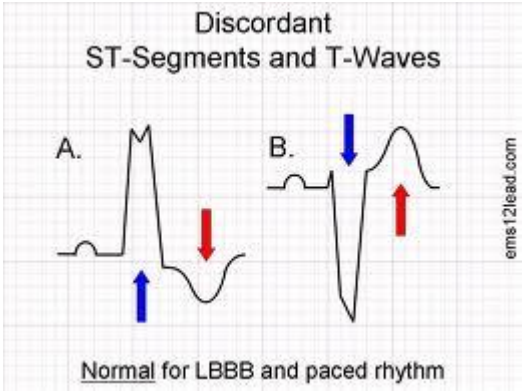
Middle-aged male with the typical ECG pattern, no structural heart disease, recovered from sudden cardiac death due to VF and with a previous history of syncopal episodes due to self-terminating rapid polymorphic VT.

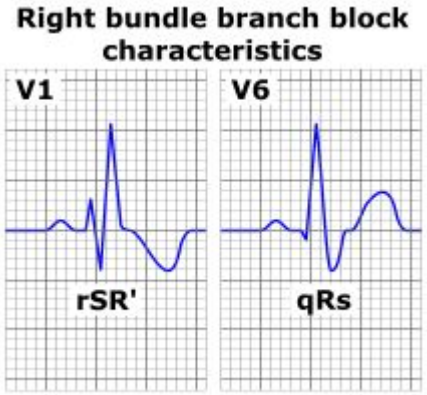
CLBBB



CRBBB

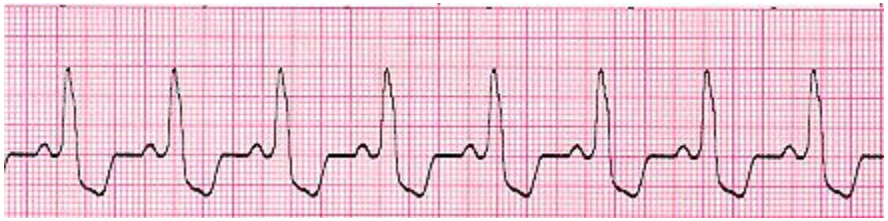



Discordant
ST-Segments and T-Waves
A.
B.
ems12lead.com
Normal for LBBB and paced rhythm


Right bundle branch block
characteristics
V1
V6
rSR'
qRs

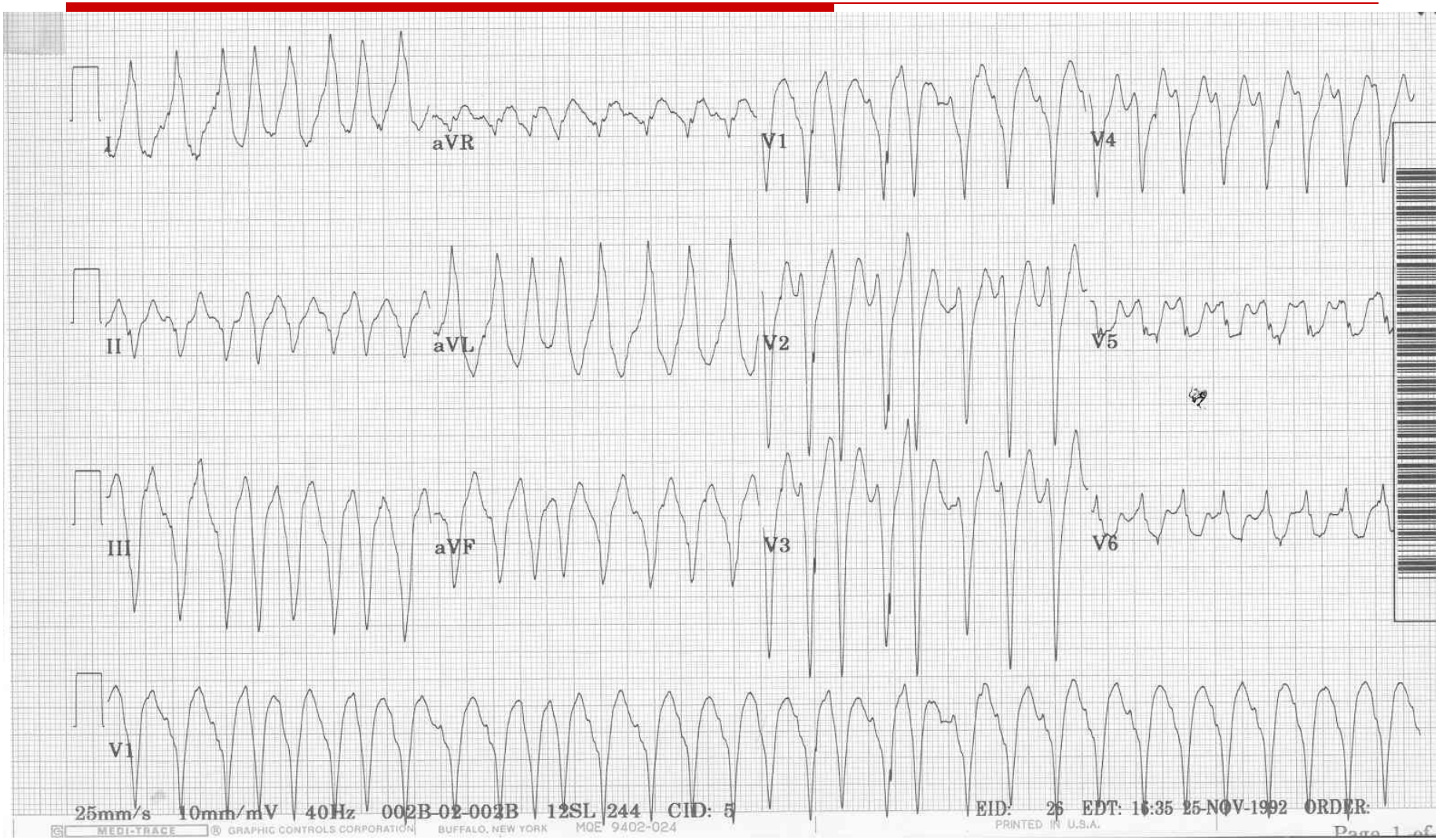
”Wide Complex Tachycardia“

- **VT**
- **SVT with**
  - Preexistent BBB
  - Rate dependent BBB
  - Preexitation



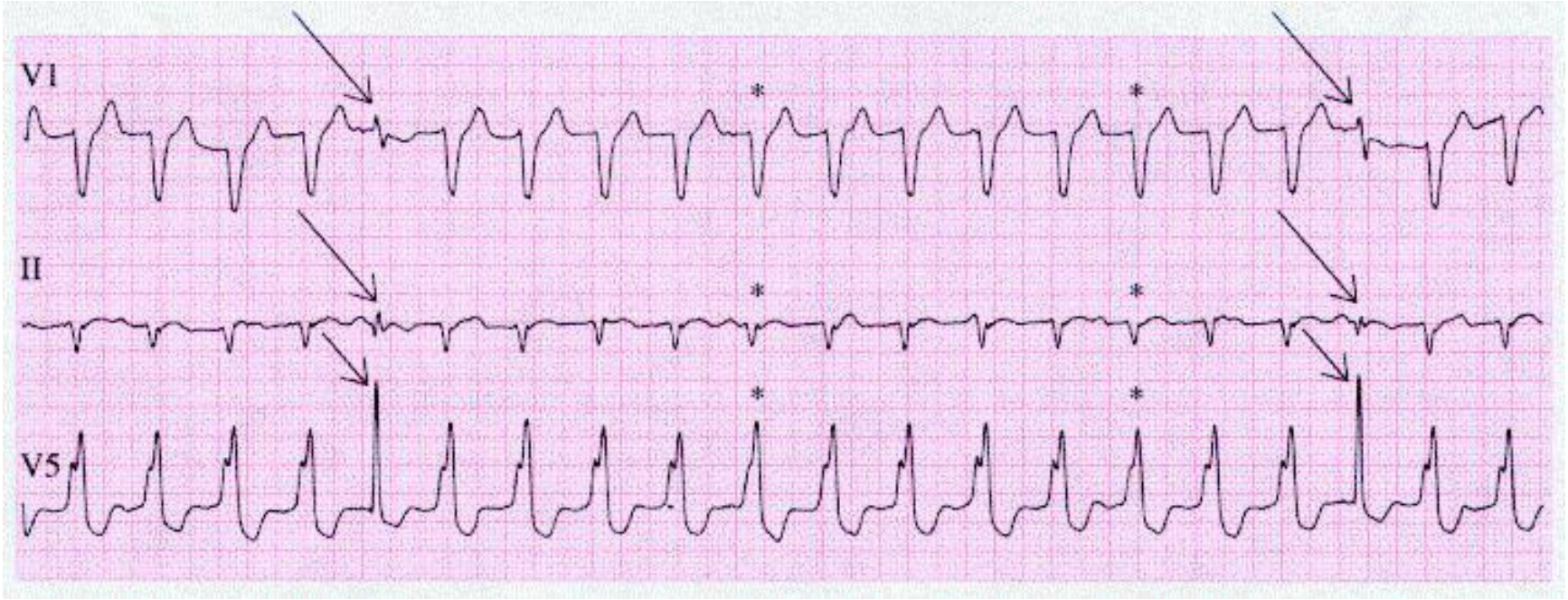
Wide QRS Irregular Tachycardia:

Atrial Fibrillation with antidromic conduction in patient with accessory pathway – Not VT



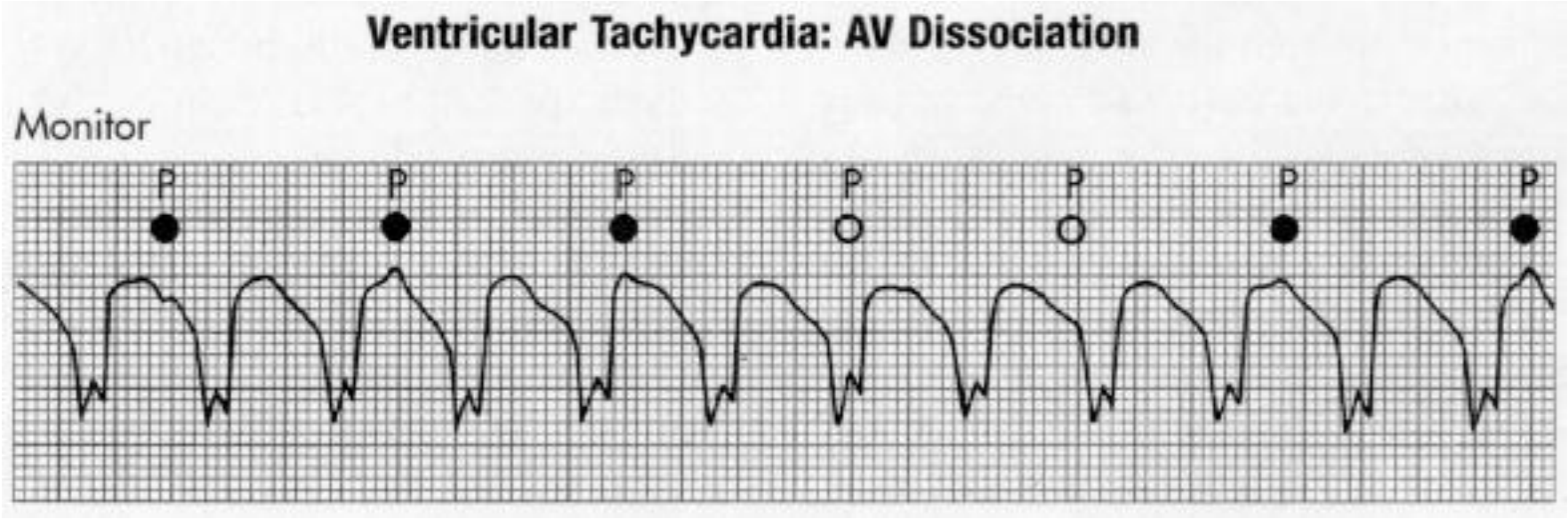
Futures favoring VT

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**



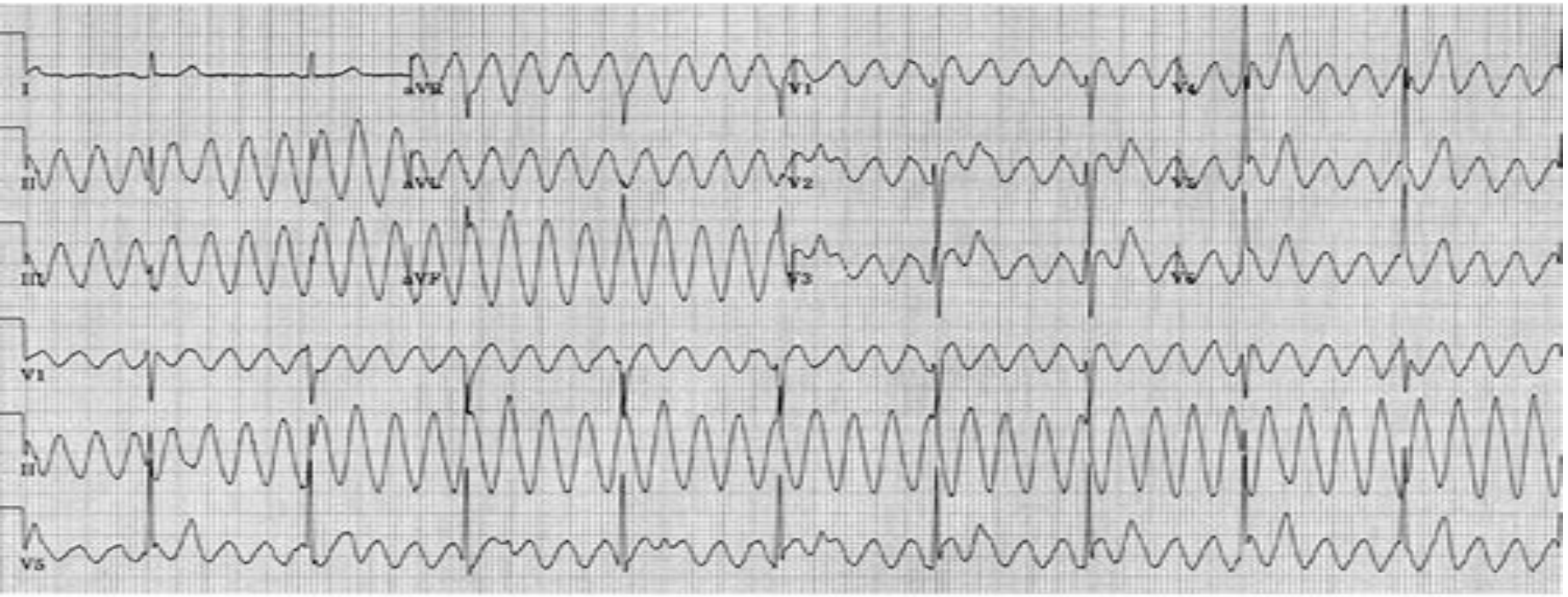
Fusion and Capture Beats

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.

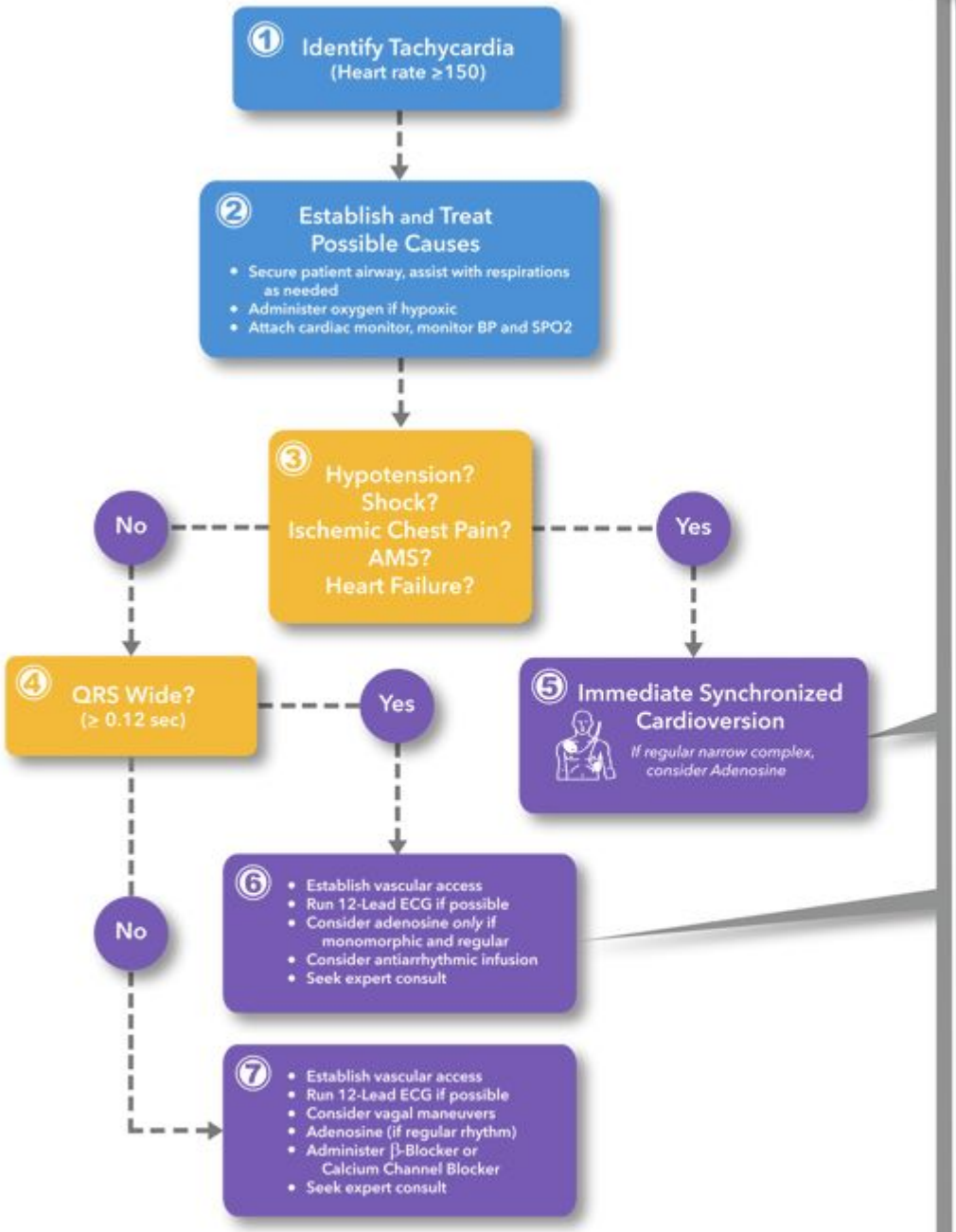


Sustained monomorphic ventricular tachycardia with atrioventricular (AV) dissociation. Note the independence of the atrial (sinus) rate (75 per minute) and ventricular (QRS) rate (140 per minute).

?


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

ACLS
certification institute
www.aclscertification.com



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

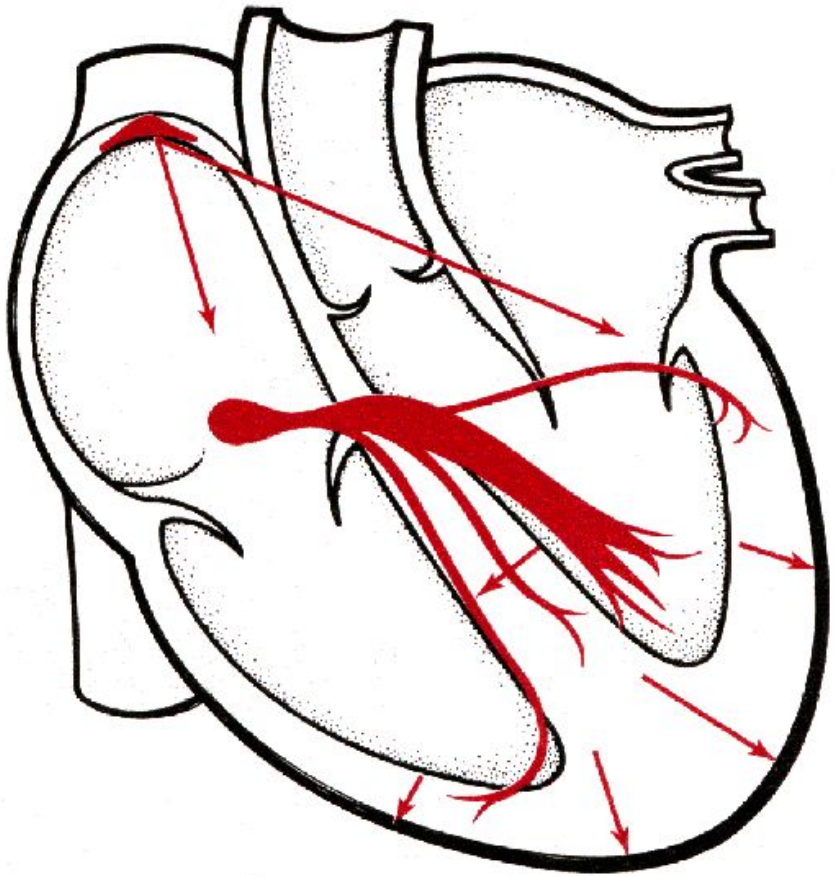
Atrioventricular Conduction Disturbances and Bradyarrhythmias

IF THE ECG ISN'T BROKEN THEN WE HAVE PROBLEM

Sites of Disturbances in Impulse Formation or Conduction Leading to Bradyarrhythmias

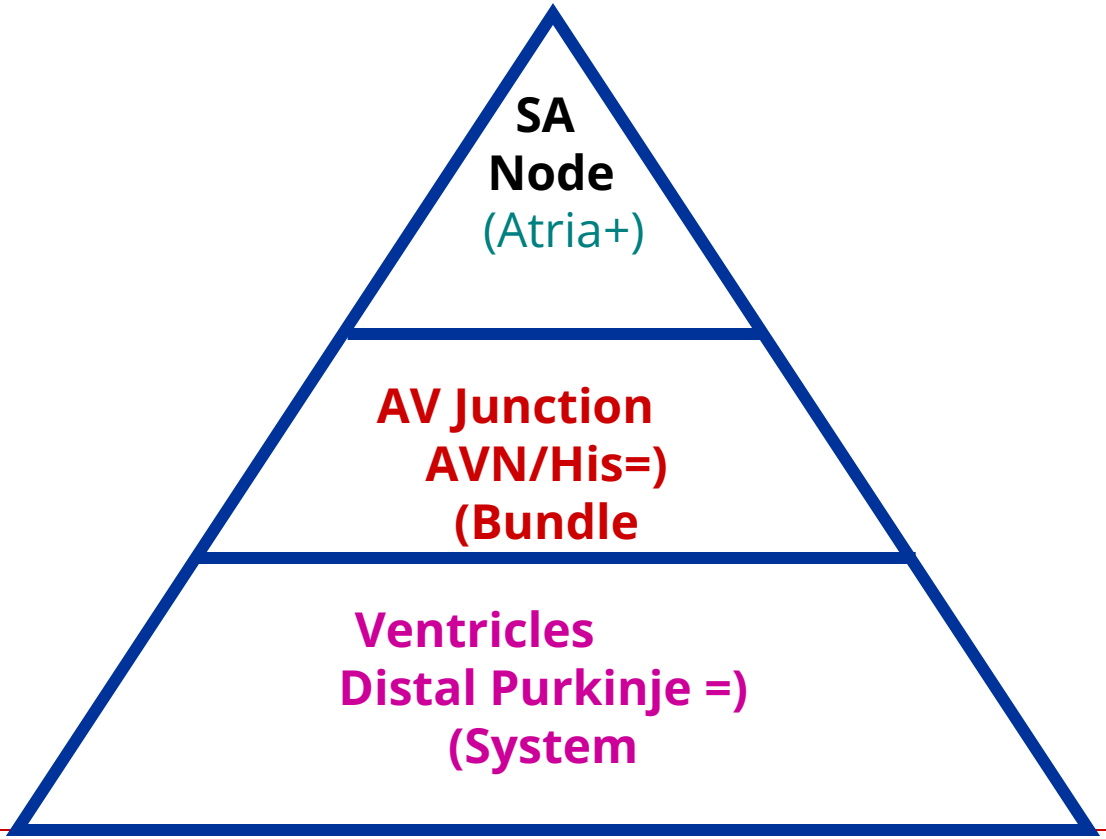
SA Node

AV Node



His-Purkinje System

Pacemaker Hierarchy
(Dominant vs Subsidiary/Escape Pacemakers)



Intrinsic Rate of Firing

60-100 min^{-1}

40-60 min^{-1}

30-40 min^{-1}

AV Block

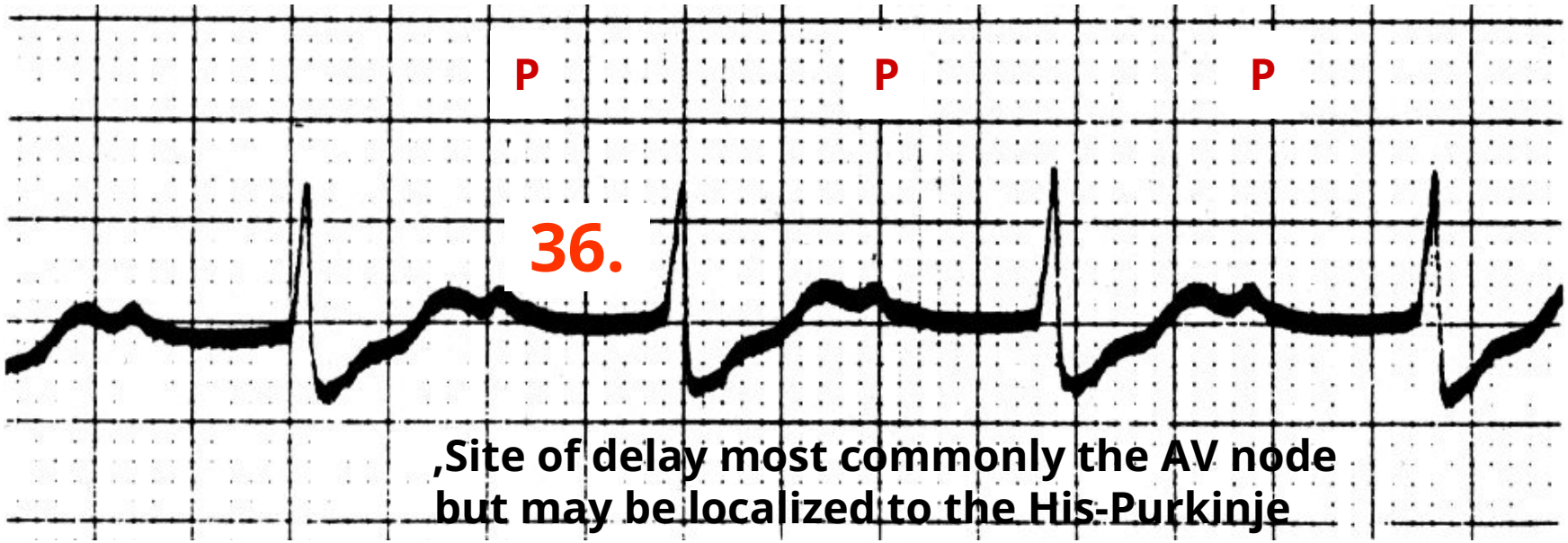
AV Block - Definitions

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

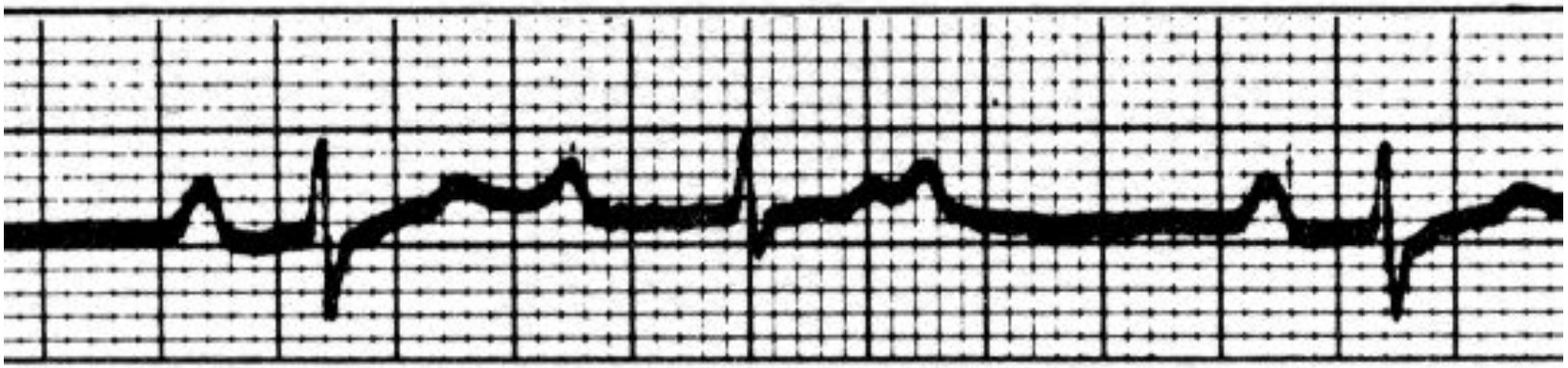
First Degree AV Block

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

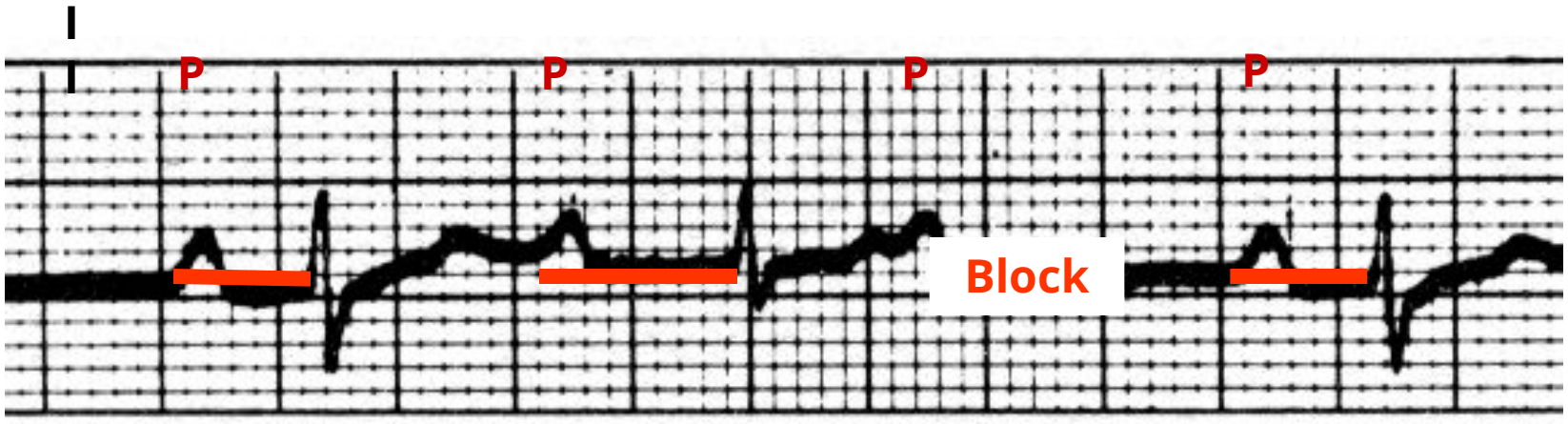
II



,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

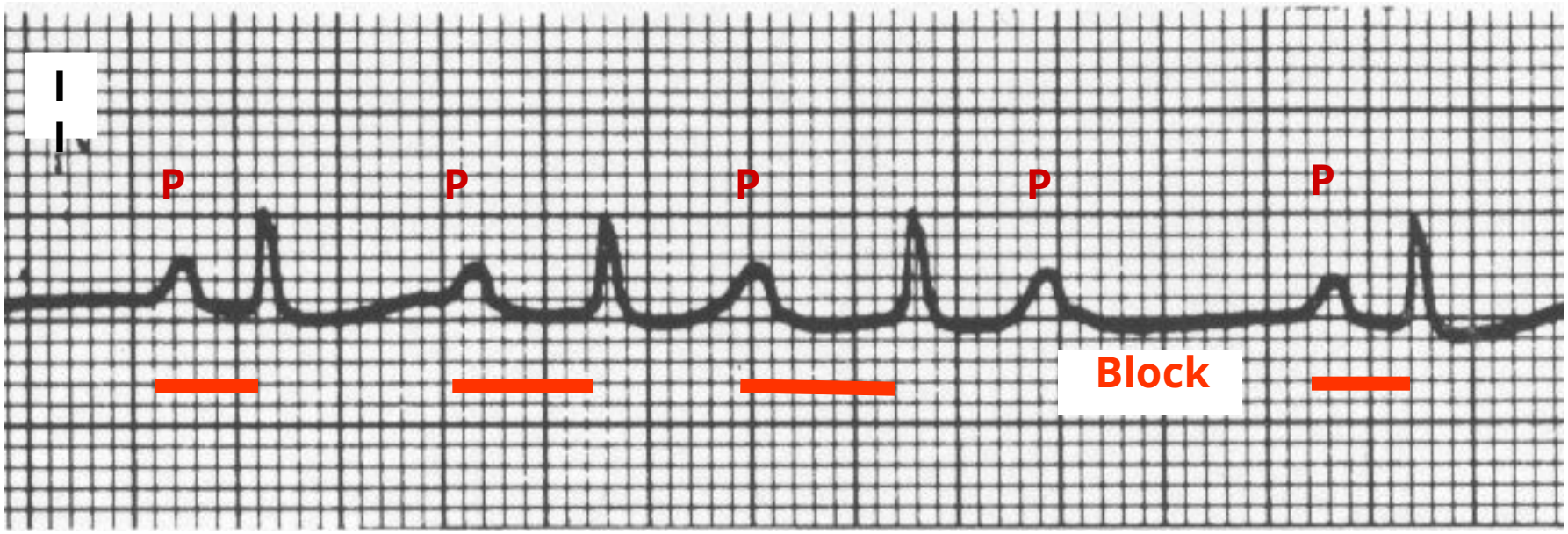


;Example of 3:2 conduction ratio •

Note PR ↑ prior to block and ↓ post-block •

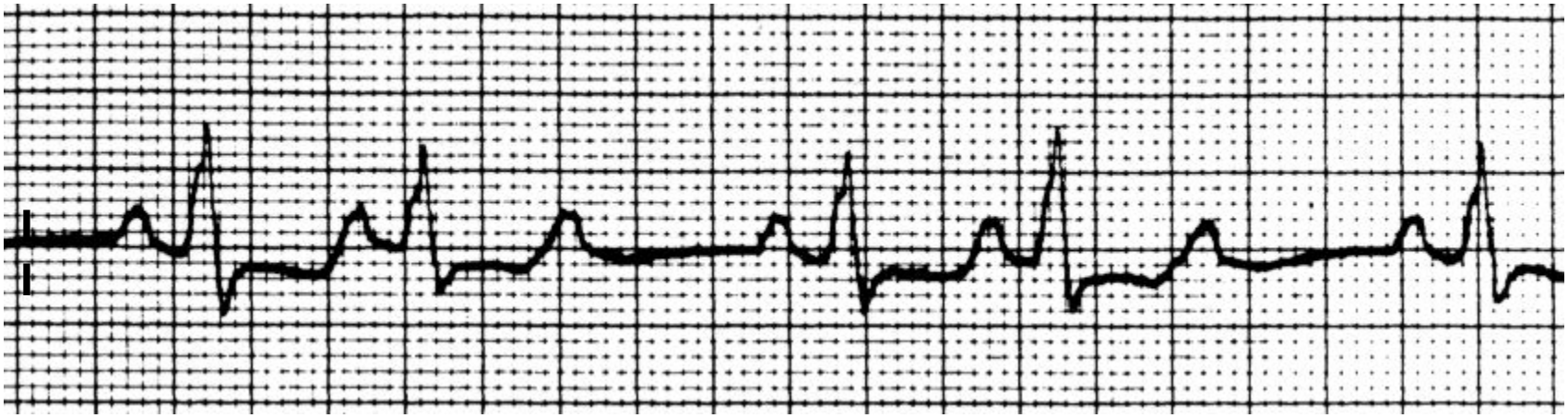
Characteristic of *AV nodal* site of block •

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

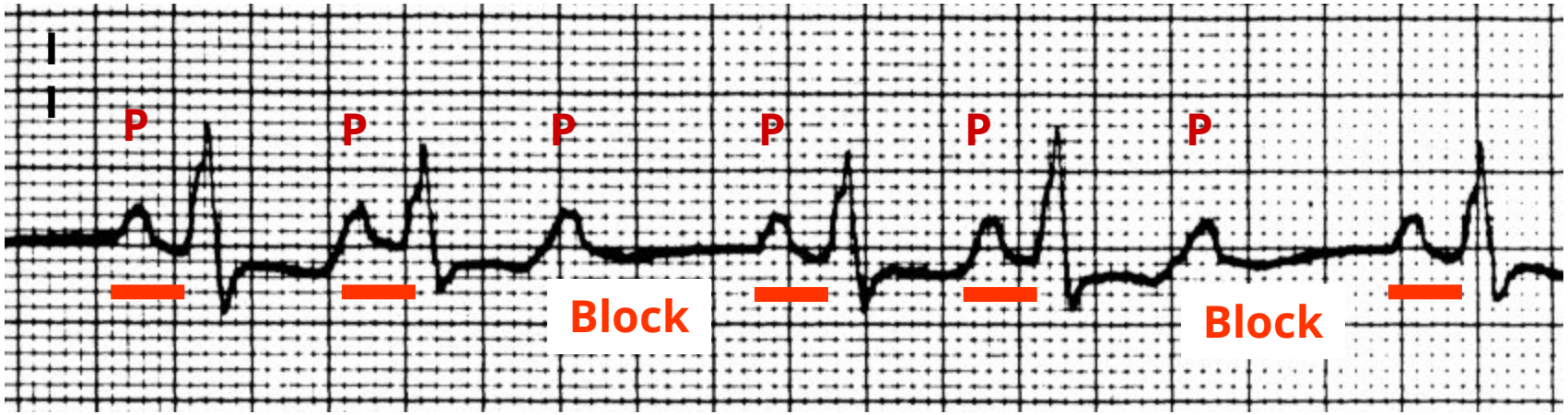


conduction ratio 4:3 •

Note first RR *longer* than second RR •


I
I

Second Degree AV Block - Type II (Mobitz II)

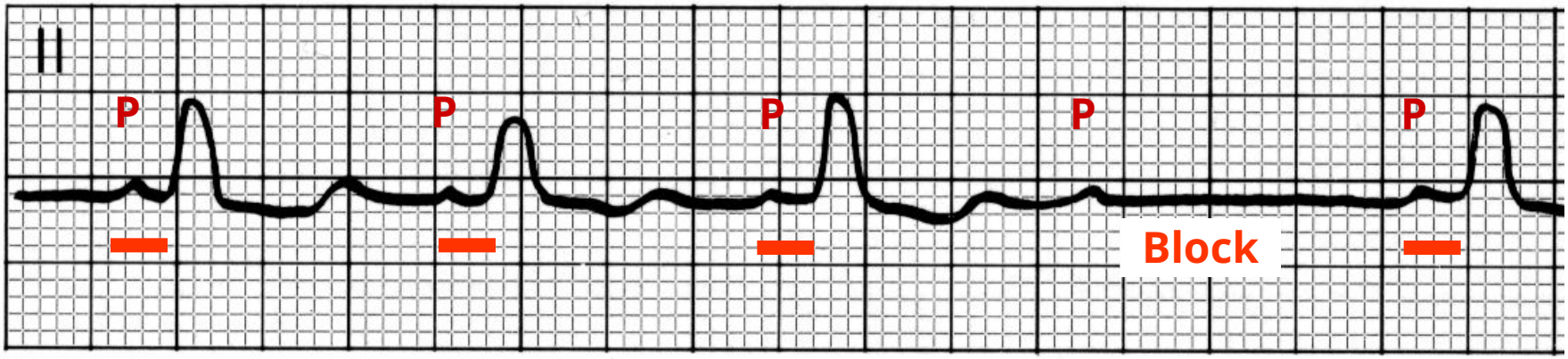


;Example of 3:2 conduction ratio •

Note *fixed* PR for all conducted beats •

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

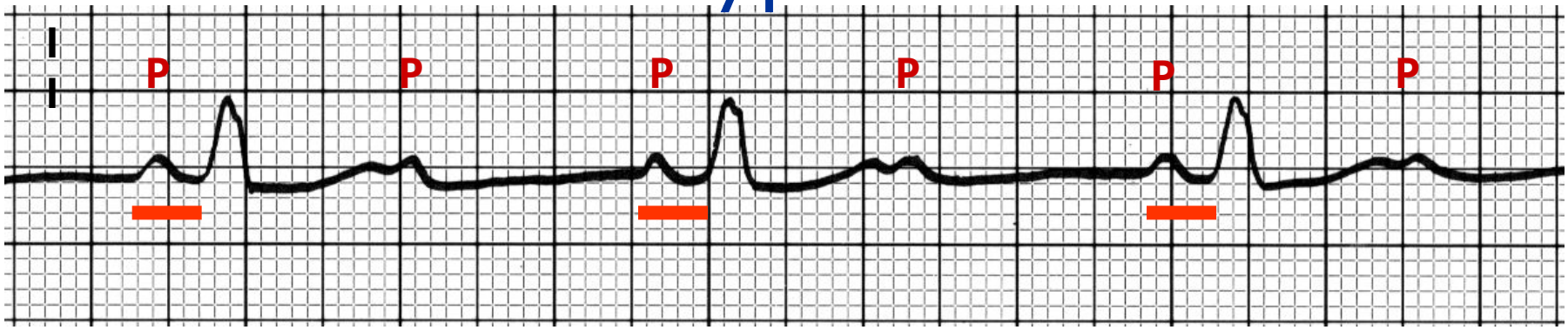
Second Degree AV Block - Type II



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

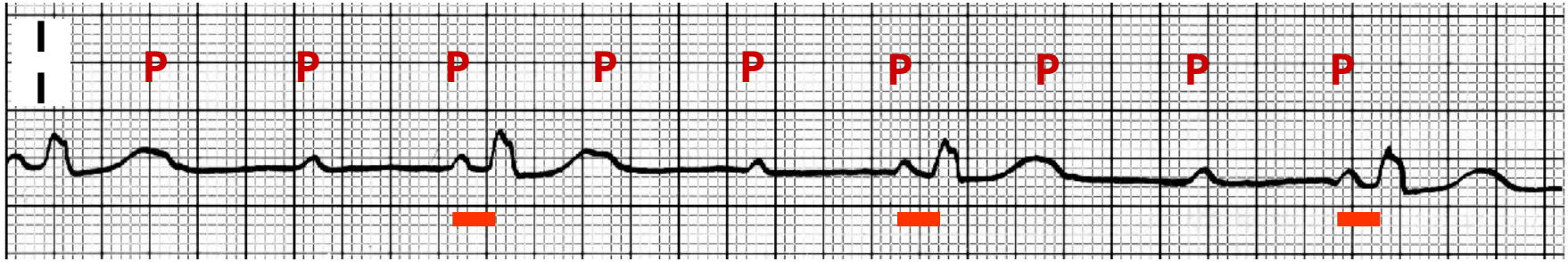
- QRS *narrow*
- Improves with exercise (catecholamine-facilitated conduction)
- Observed in setting of increased vagal tone (e.g., sleep) or AV nodal depressant drugs

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

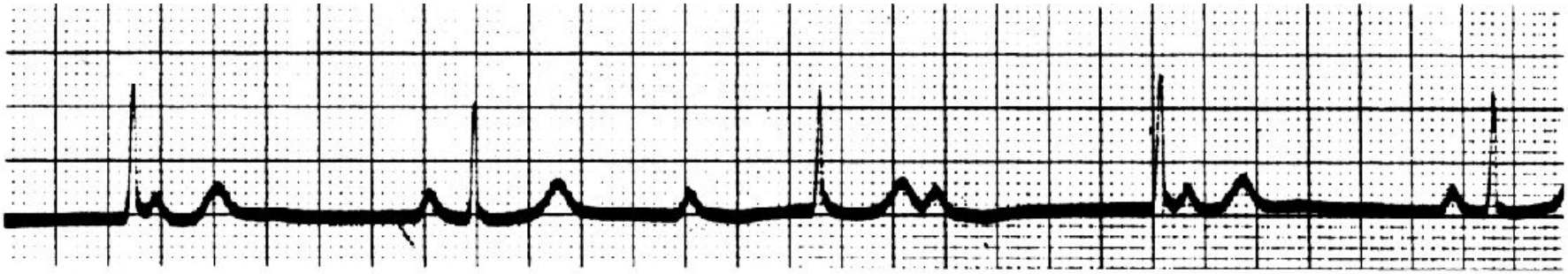
Block of ≥ 2 Consecutive) (P Waves



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

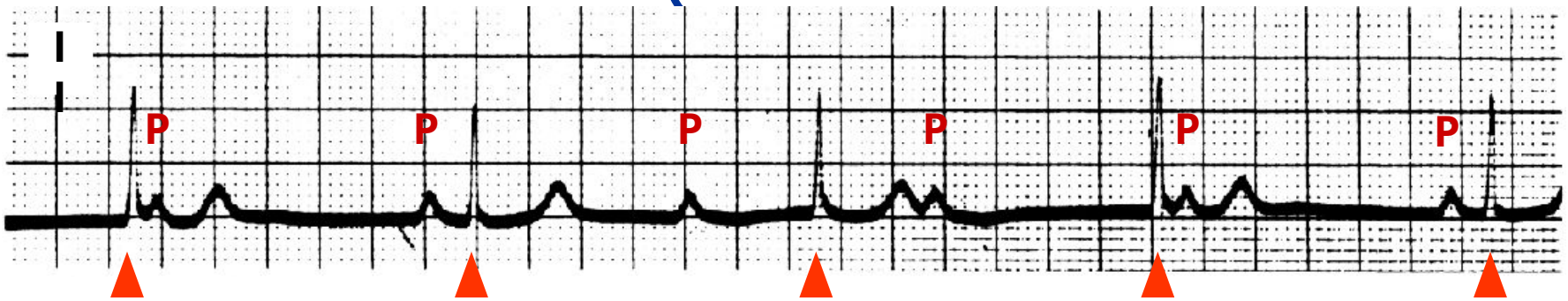
Site of AV Block vs. Escape Rhythm

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



Third Degree AV Block

Complete Heart) (Block



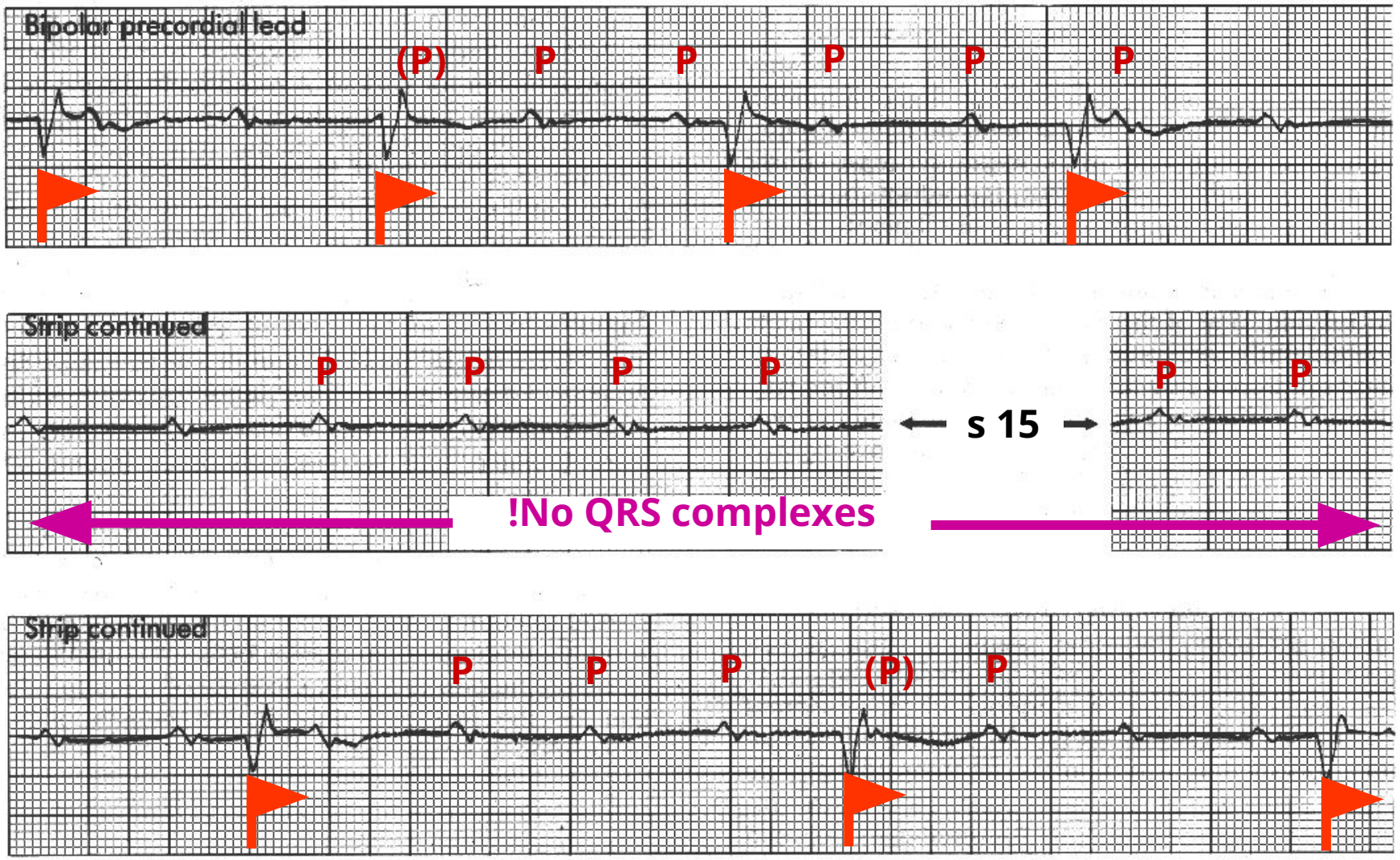
P waves at 60 beats/min •

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

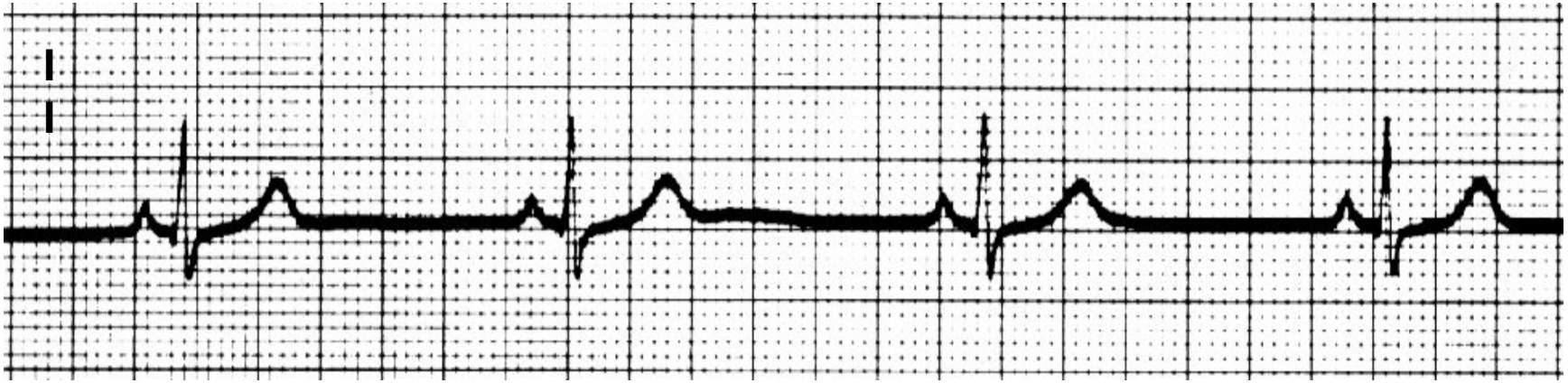


Causes of NON-Physiologic AV Block

- 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 Bradyarrhythmias

Sinus Bradycardia

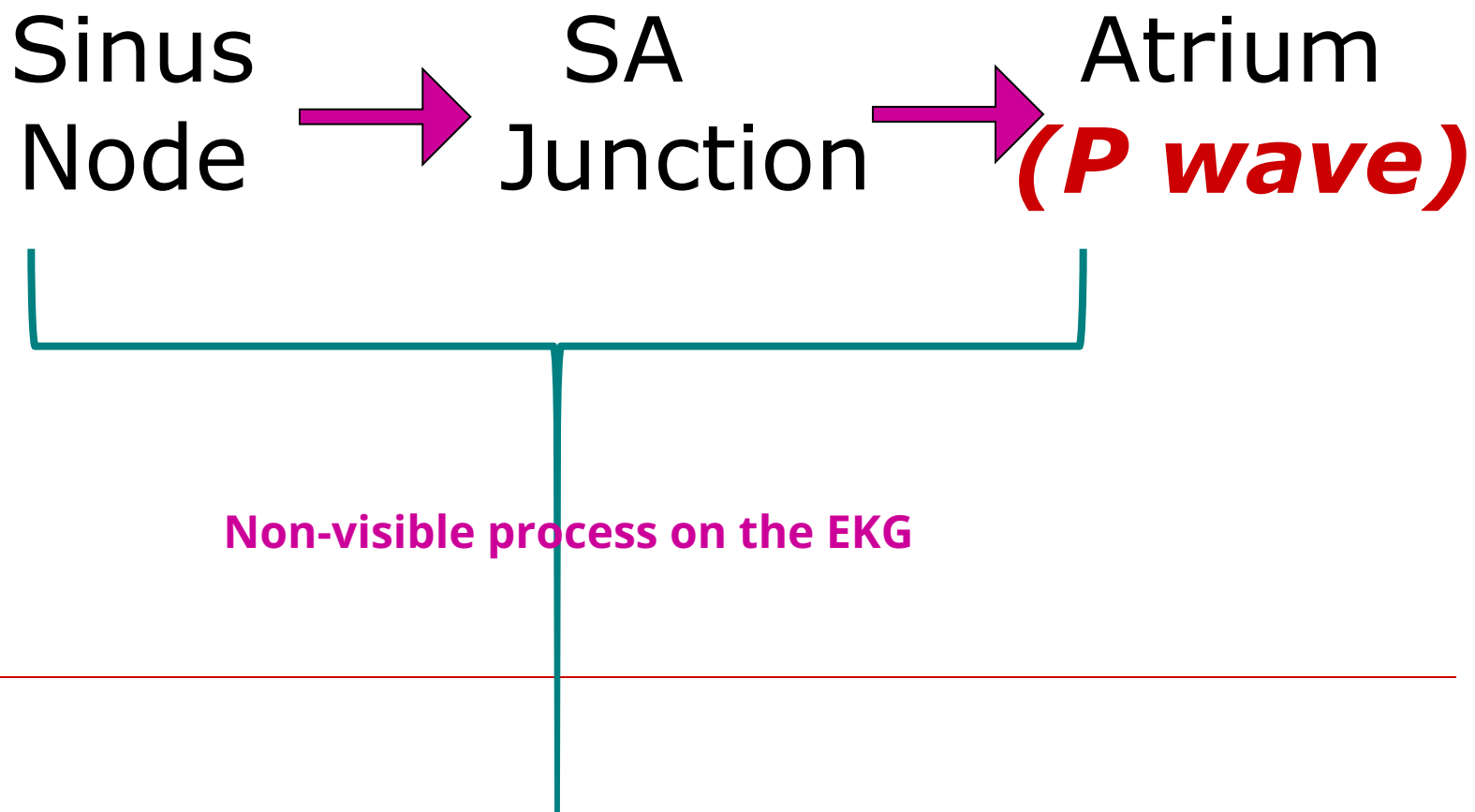


P wave upright in leads I and II, just as in normal sinus rhythm

Causes of Sinus Bradycardia

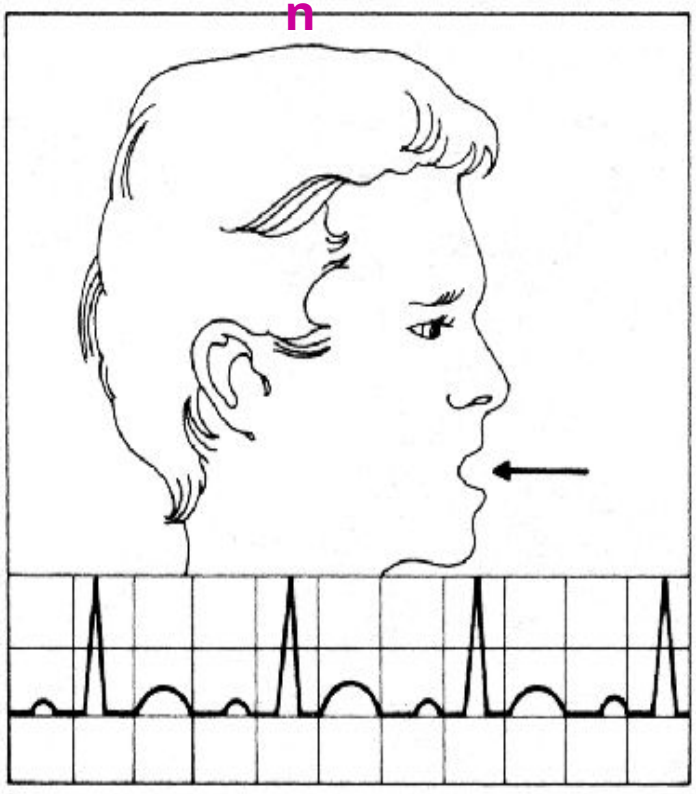
- Increased vagal tone
- Drugs: beta blockers, calcium channel blockers, amiodarone, digoxin (indirect effect)
- Myocardial ischemia/infarction
- Hypothyroidism
- “Sick sinus syndrome” - degenerative/fibrotic atrial process

Sequence of P Wave Generation



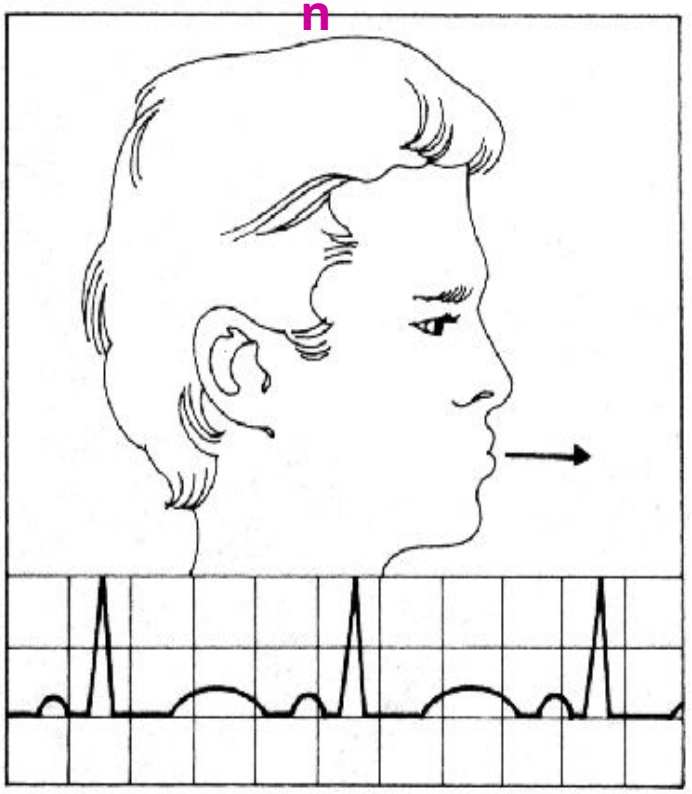
Sinus Arrhythmia

Inspiration



SA nodal acceleration

Expiration

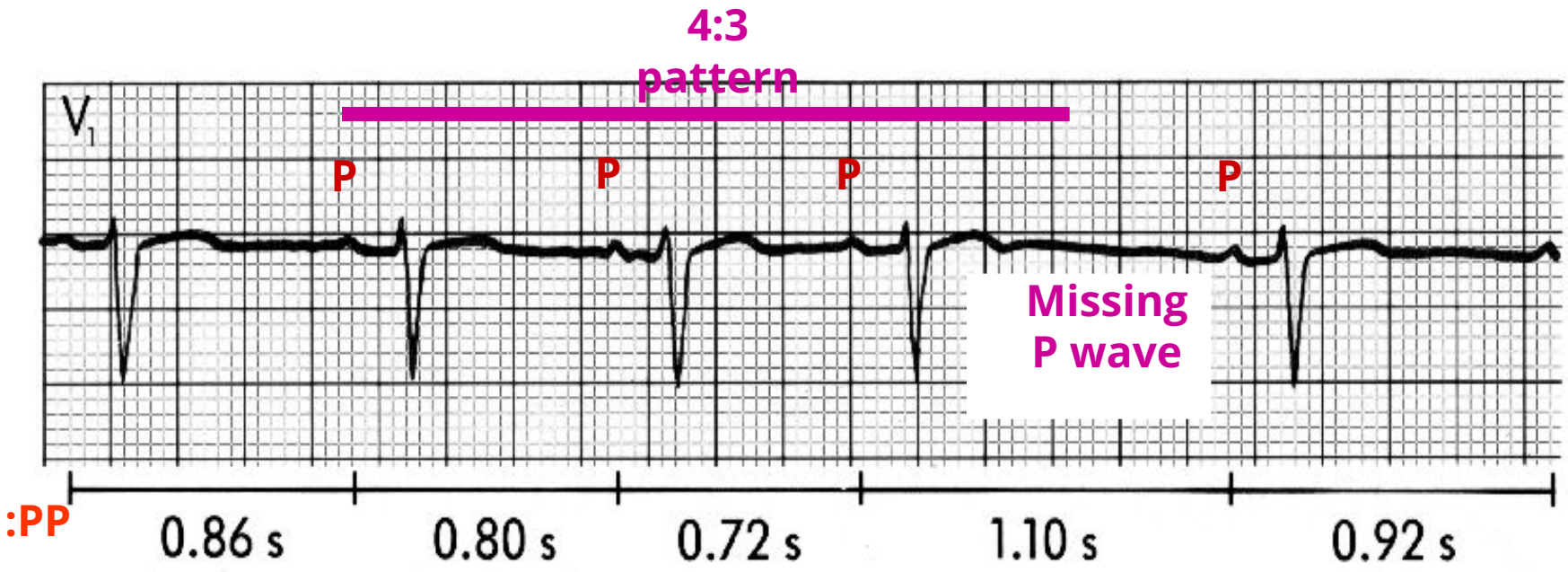


SA nodal deceleration

Sinoatrial (SA) Exit Block - Definitions

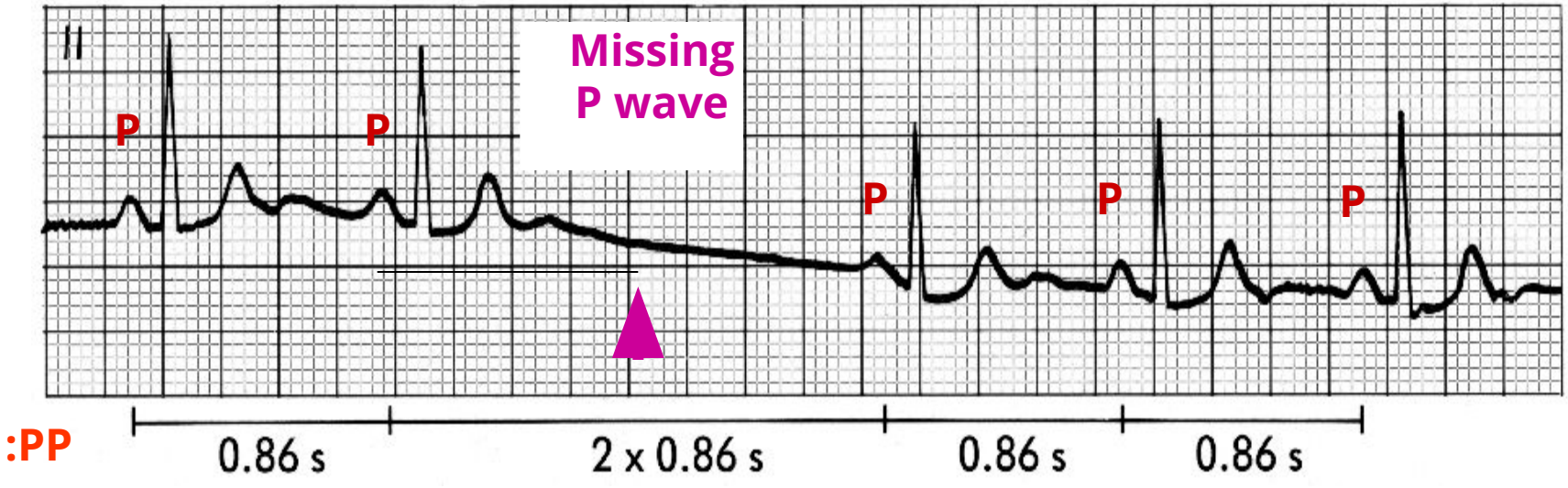
- **First Degree:** Prolonged SA conduction time (non-detectable on EKG; no missing P waves)
- **Second Degree:** Intermittent non-conduction (intermittent absence of P waves)
- **Third Degree:** Persistent non-conduction (complete absence of P waves; escape rhythms only)

Second Degree SA Exit Block - Type I (Wenkebach)



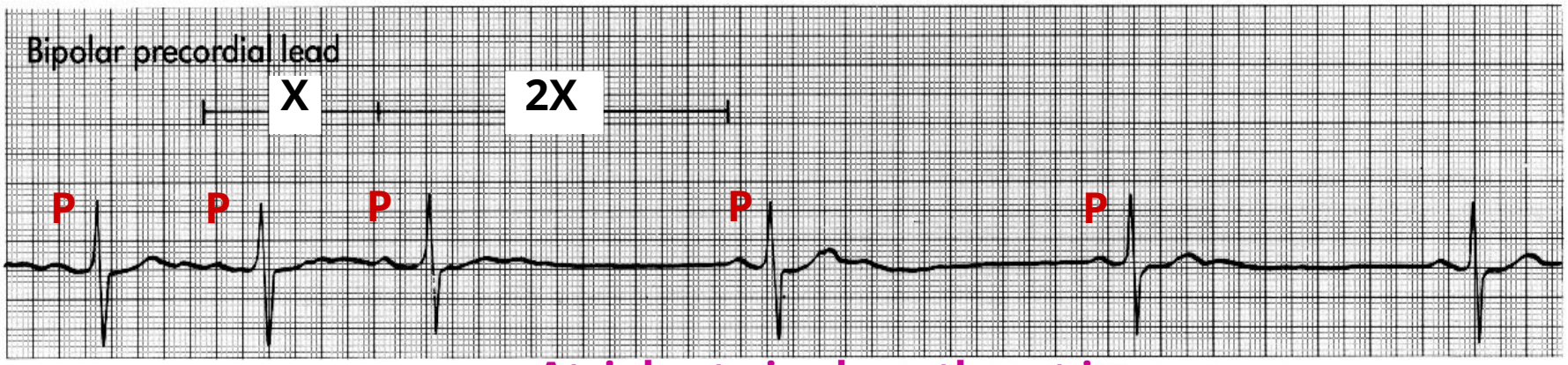
- PP intervals shorten prior to block
- Note unaffected, *fixed* PR intervals

Second Degree SA Exit Block - Type II

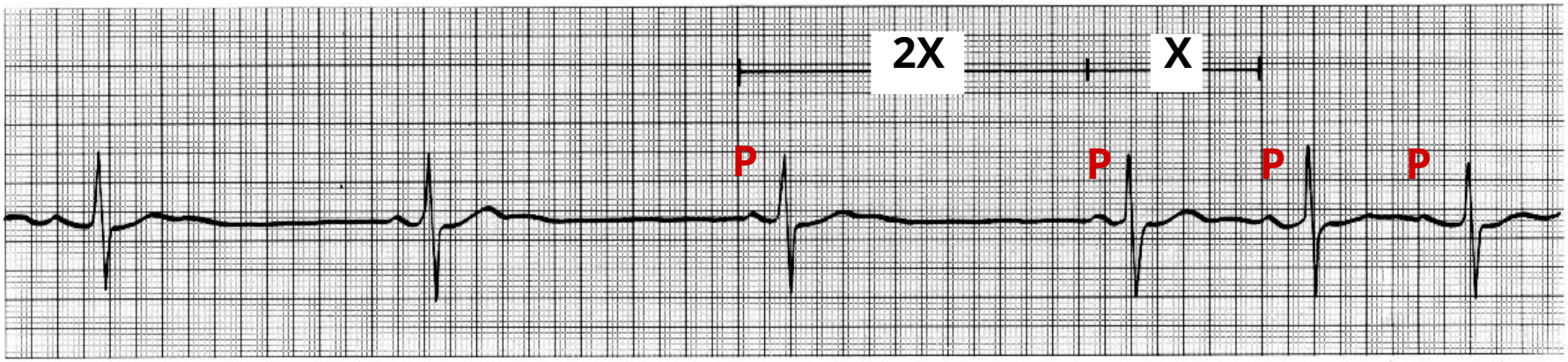


One P wave abruptly "drops out" on time

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

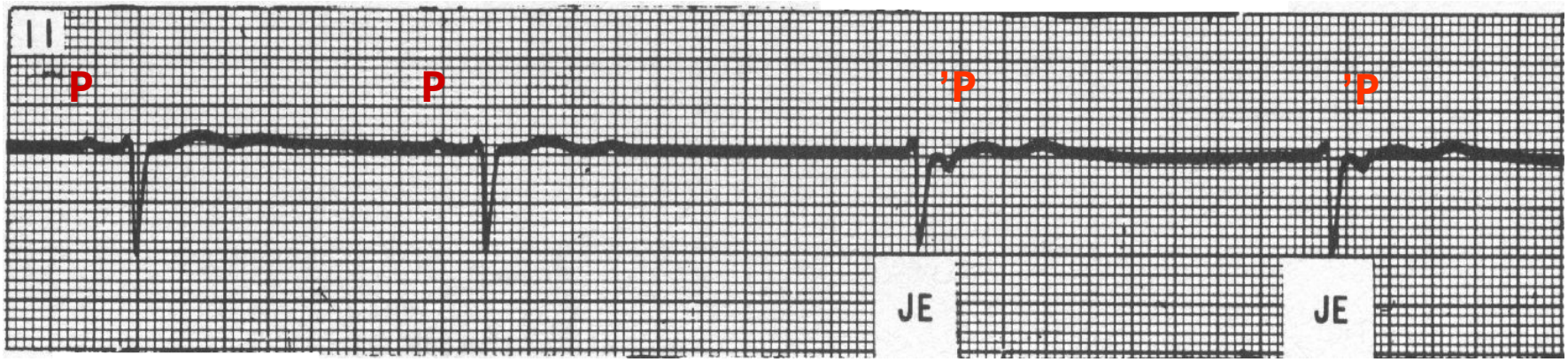


Atrial rate is abruptly cut in half



Resolution of block

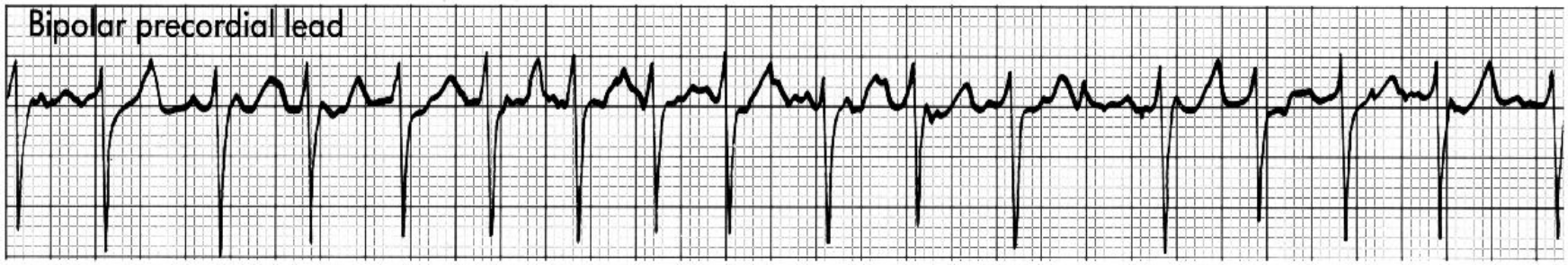
Sinus Arrest



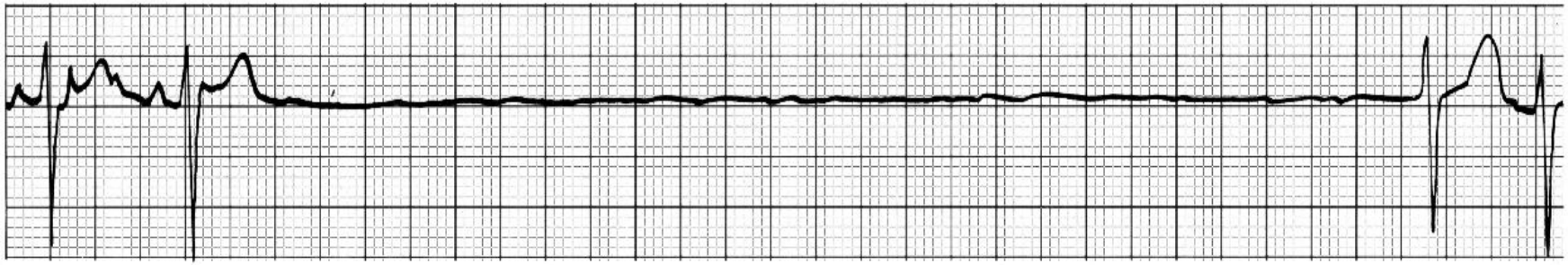
Sinus bradycardia → Sinus arrest → Slow junctional escape rhythm

(waves with retrograde p)

Tachycardia-Bradycardia Syndrome (”Form of “Sick Sinus)



Atrial Flutter →



Atrial Flutter terminates

Sinus arrest

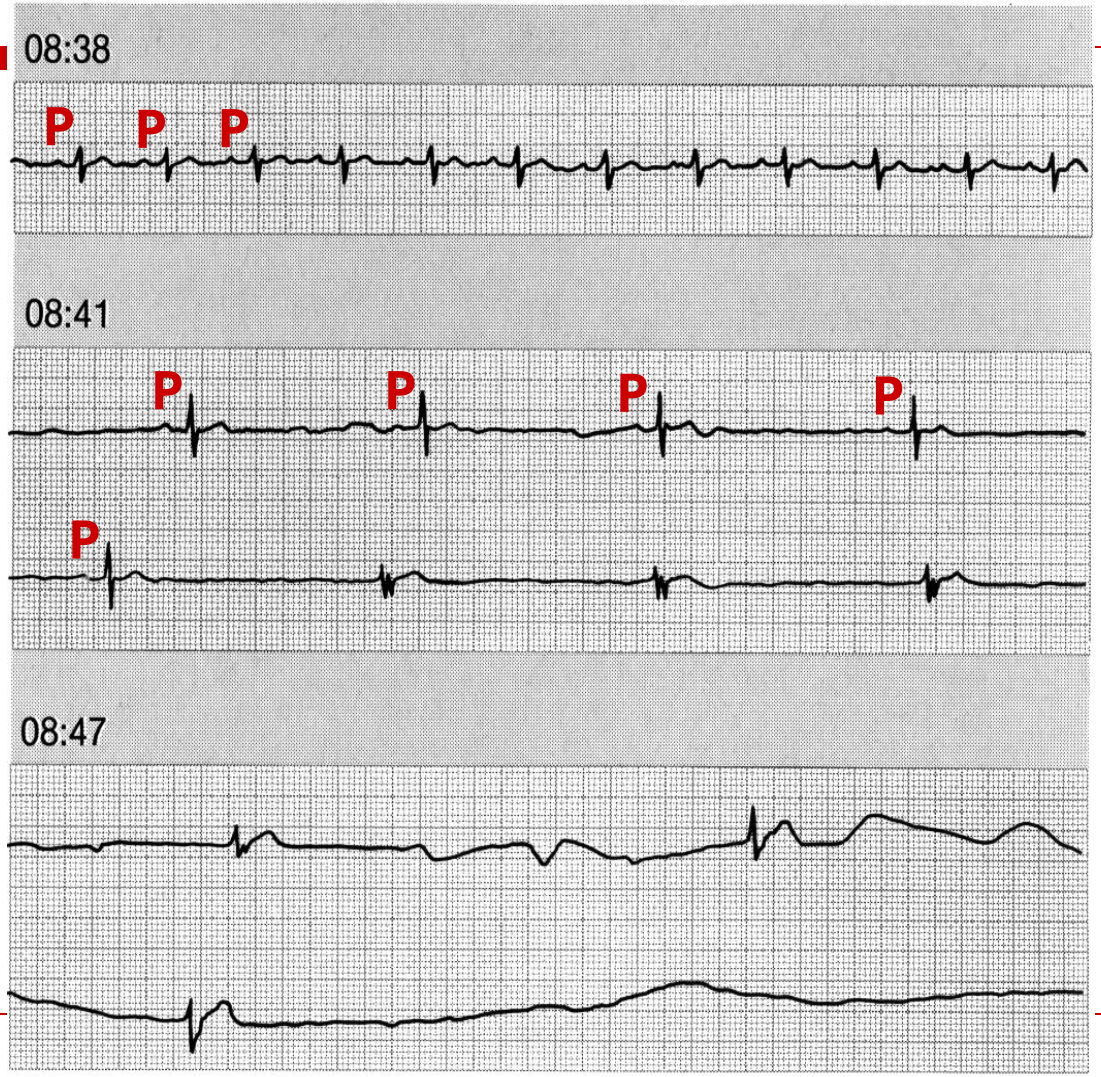
Junctional escape (tardy)

Sinus Arrest → Asystole

Sinus rhythm

.Sinus brady
Sinus →
arrest
V. escape →
rhythm

.Failure of V escape rhythm
Asystole →



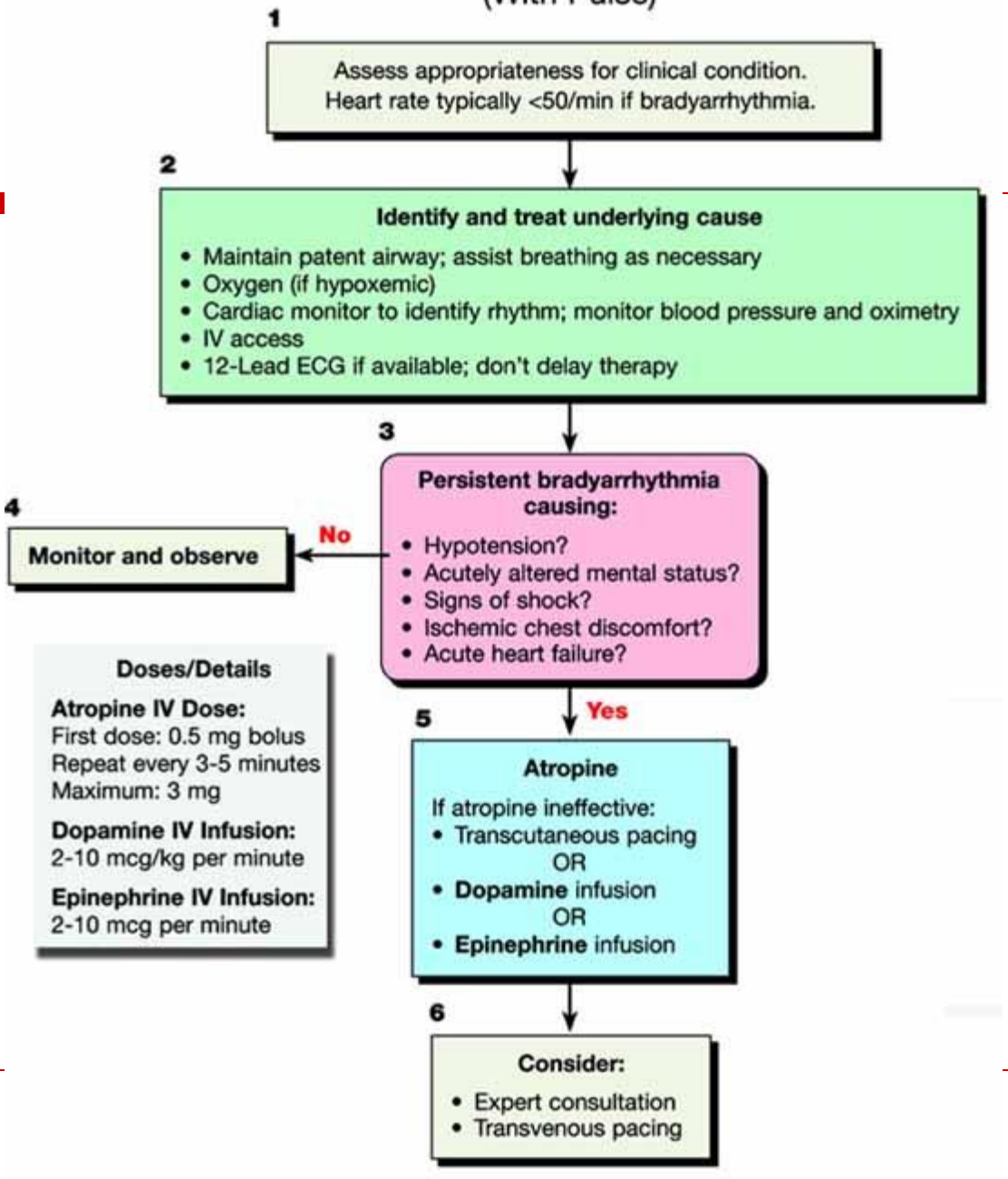
Causes of SA Exit Block and Sinus Pauses/Arrest

- 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)




dreamstime.com