Pediatric Arrhythmias

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Normal Impulse Conduction

Sinoatrial node

↓

AV node

↓

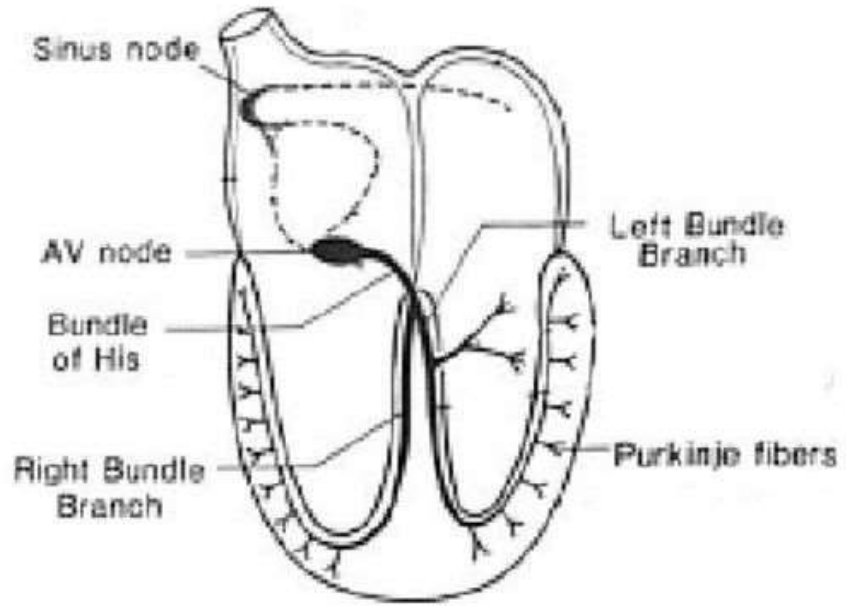
Bundle of His

↓

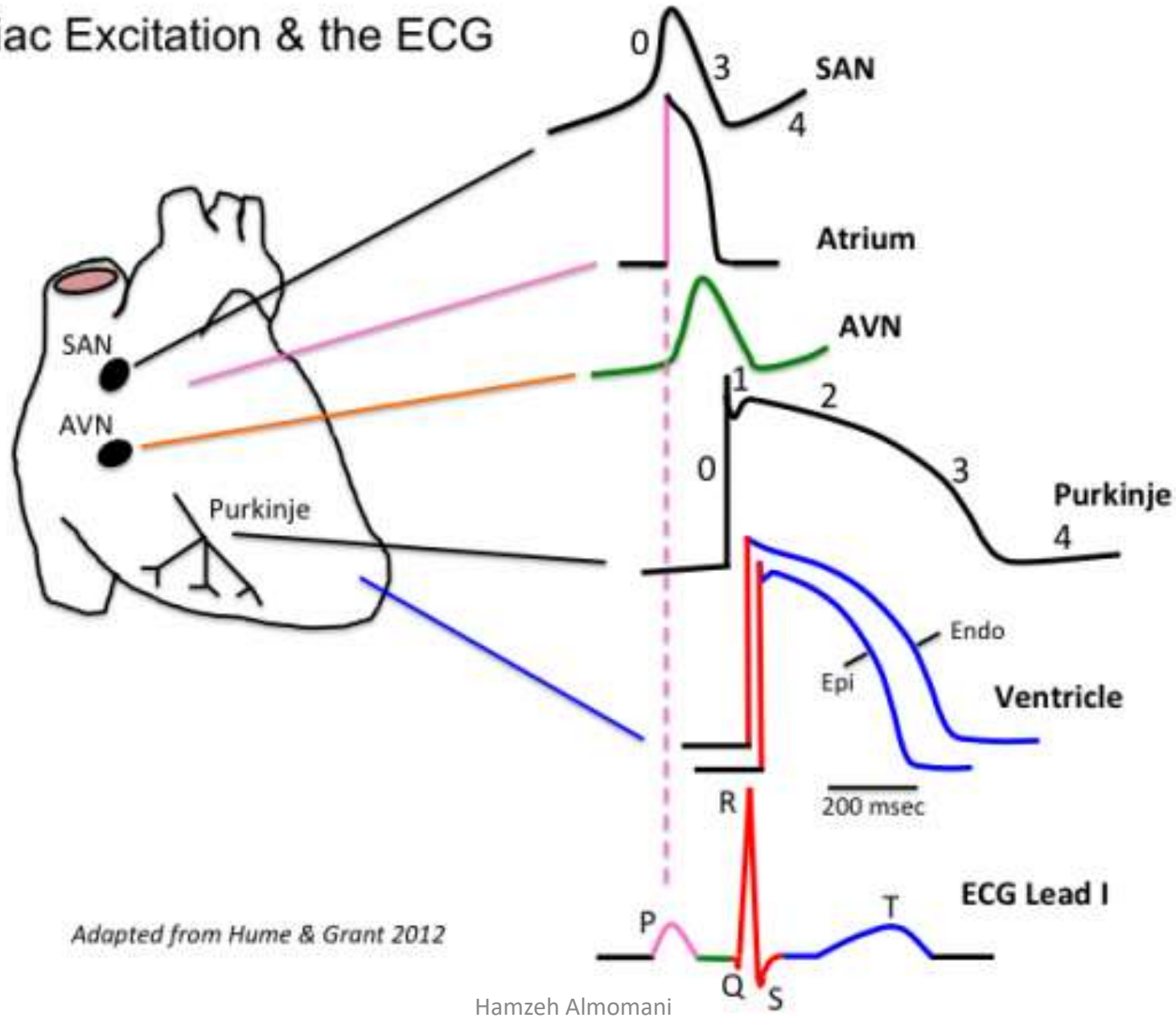
Bundle Branches

↓

Purkinje fibers



Cardiac Excitation & the ECG



Adapted from Hume & Grant 2012

NORMAL HEART RATE RANGES

Normal Heart Rate for Age

Age	Awake Rate (beats/min)	Sleeping Rate (beats/min)
Newborn – 3 months	84 to 205	80 to 160
3 months – 2 years	100 to 190	75 to 160
2 – 10 years	60 to 140	60 to 90
> 10 years	60 to 100	50 to 90

PALS 2010

NEONATAL AND INFACNT HEART WILL INCREASE HEART RATE TO INCREASE CARDIACC OUTPUT AS THEY DON'T' HAVE MECHANISM TO INCREASE STROKE VOLUME.

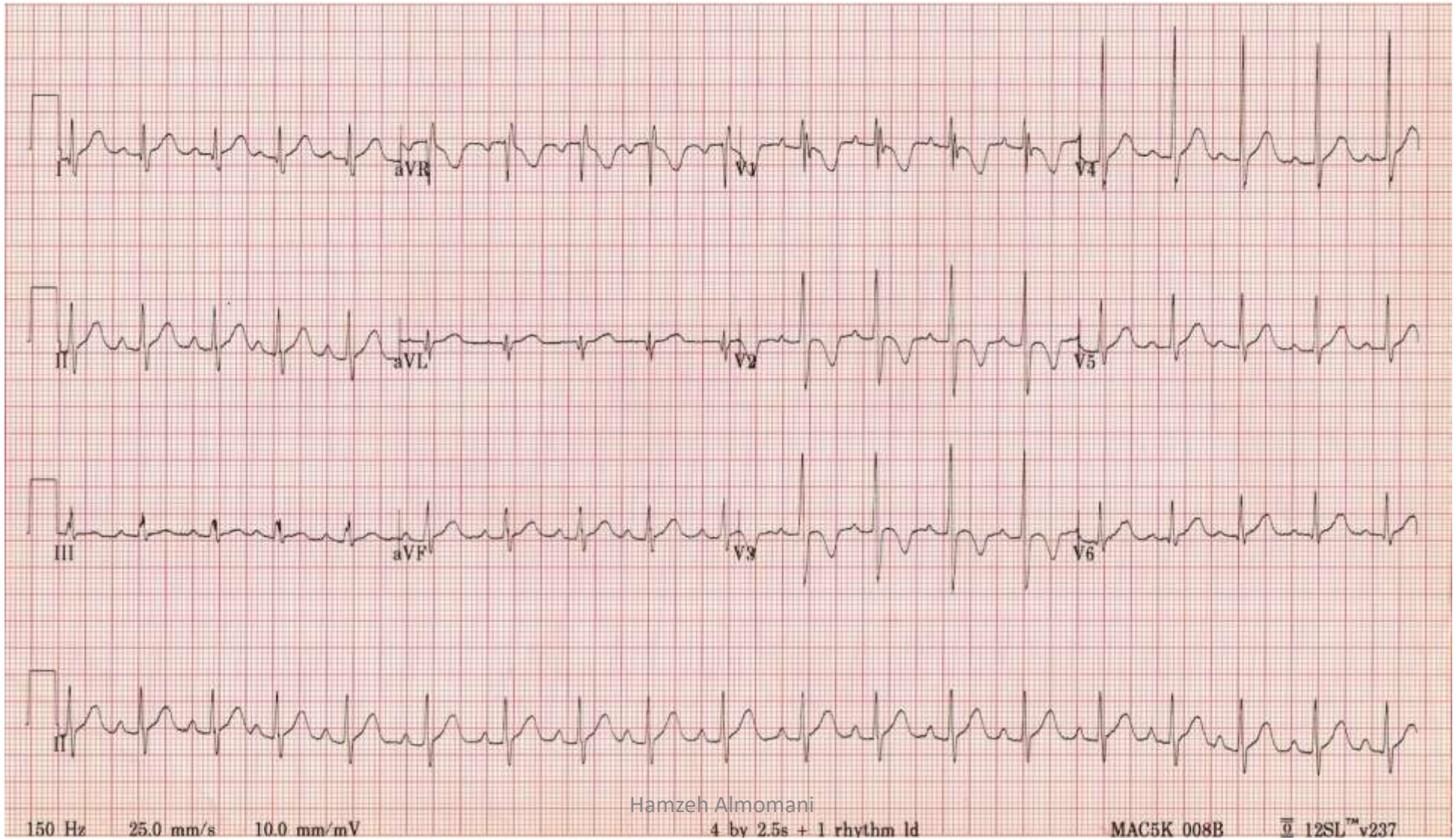
INTERVALS

- PR INTERVAL LENGTHENS <u>FROM INFANTS TO CHILDREN</u>
  – PR – FROM 0.08 -0.12 IN NEOTNATES TO 0.11-0.18 IN ADOLESCENTS

- QRS INTERVAL LENGTHENS <u>FROM INFANTS TO CHILDREN</u>
  QRS 0.05 – 0.09 IN NEONATES TO 0.07 – 0.11 IN ADOLESCENTWS

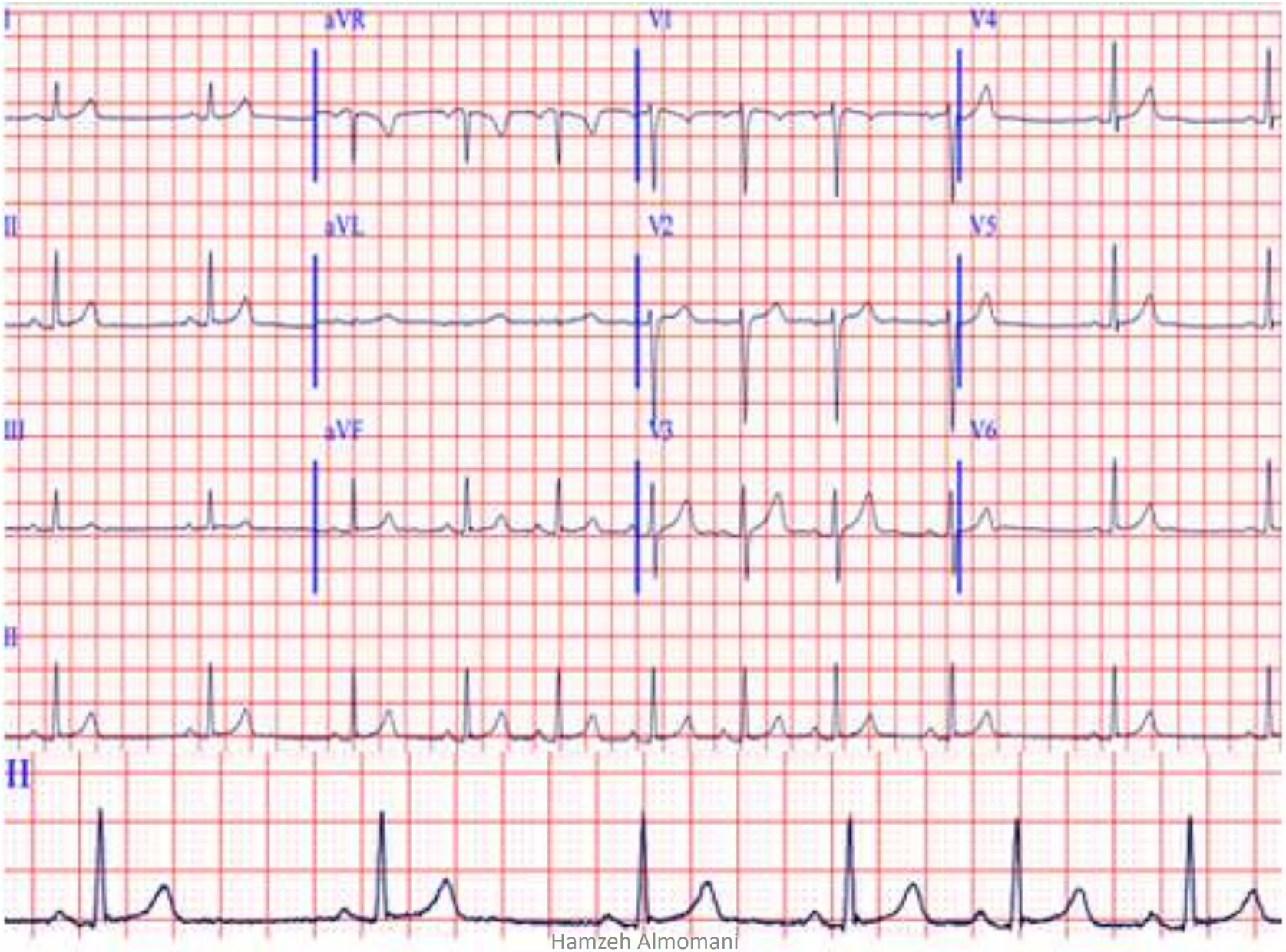
T-WAVES

- **<u>T WAVES INVERTED in Right Pericardial Leasds-V1</u>(after the first week of life)** INTO CHILDHOOD/EARLY ADOLESCENTS



Non – Pathologic Arrhythmia

- Sinus arrhythmia
  - P-P interval variation
  - Exaggerated with respirations
    - Increased HR during inspiration and decreased HR during expiration. Caused by changes in parasympathetic input to the hear which is mediated by vagus nerve.
  - Maybe more pronounced in infants


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

Other benign arrhythmias

- Isolated premature ventricular beats -PVCS – uniform morphology (up to 40%)
- Isolated supraventricular beats
- First degree A-V block
- Mobitz I second degree block
- Junctional arrhythmias

Mechanism and Pathophysiology of Tachyarrhythmia in Children

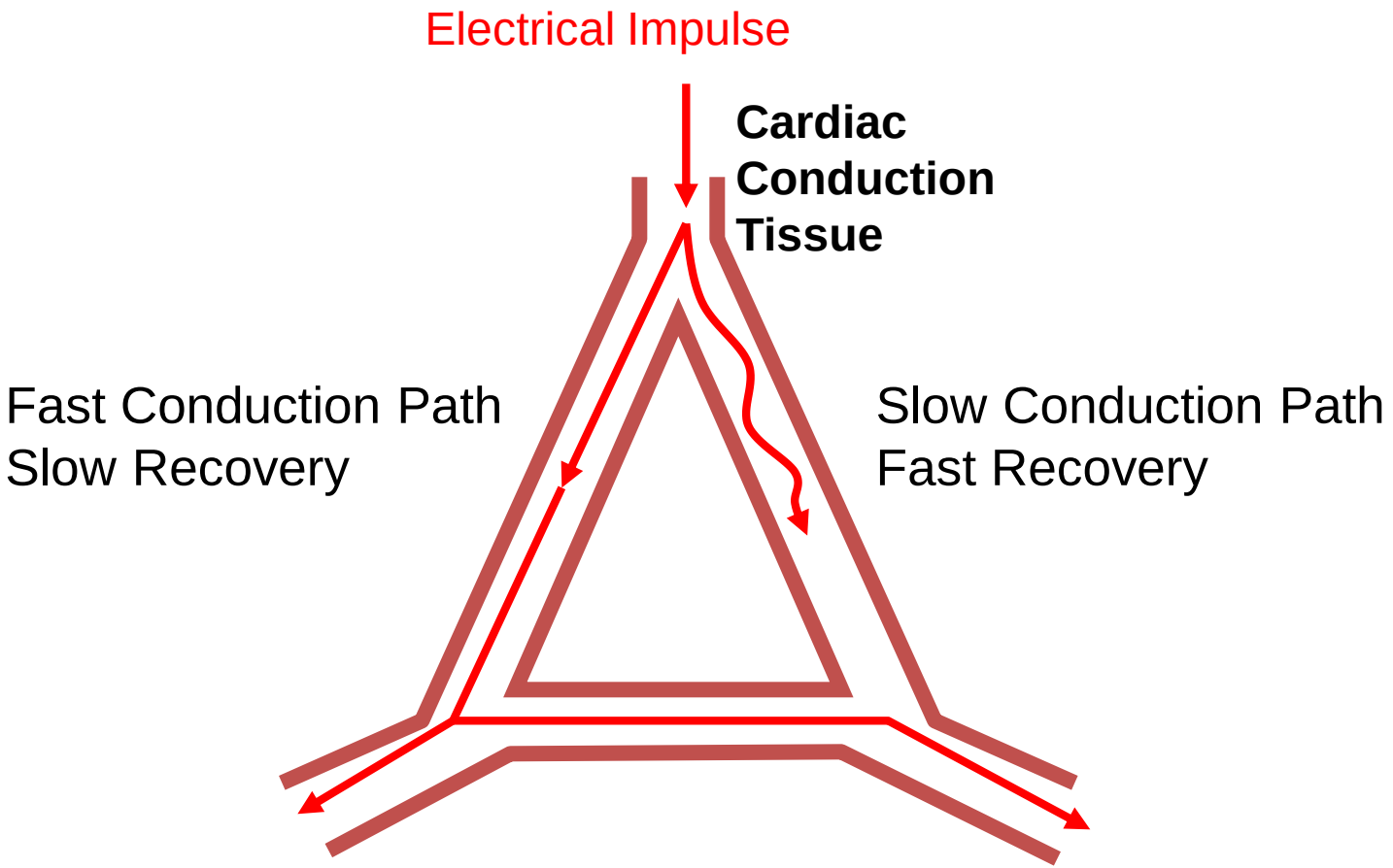
I. Reentry (most common)
II. Automaticity
III. Triggered activity

- SVT is the **most common rhythm disturbance** in children.
- Most SVTs in children are **reentrant rhythms**.

Re-entry

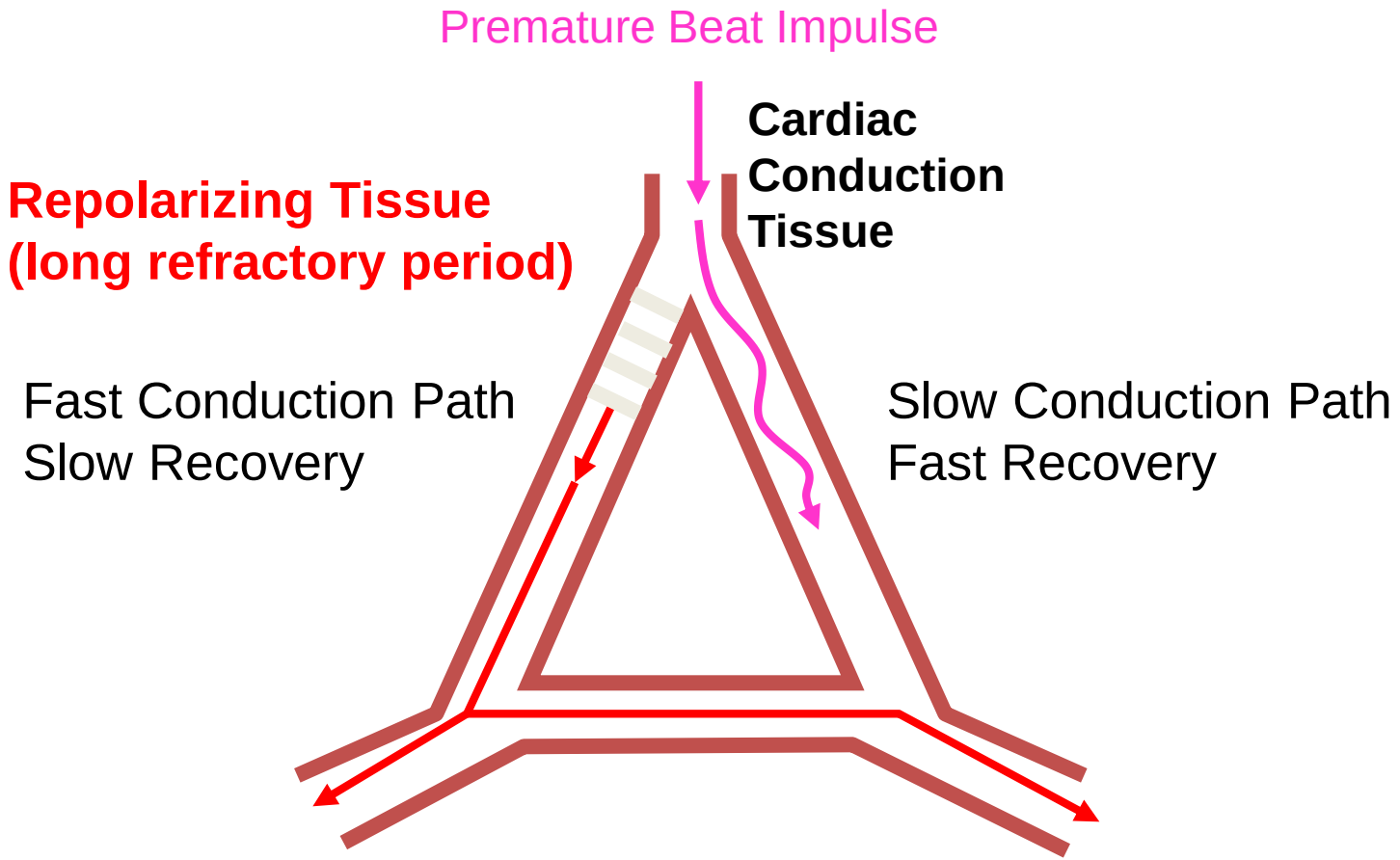
- Requires a **bypass pathway** between atria and ventricles in addition to the AV node.
- **Bypass pathway** can be either
  - **An anatomically separate accessory pathway** (the Bundle of Kent as in most cases of Wolf-Parkinson-White)
  - Or a **functionally separate pathway within the AV node** (called AV nodal re-entry tachycardia).

Reentry Requires…



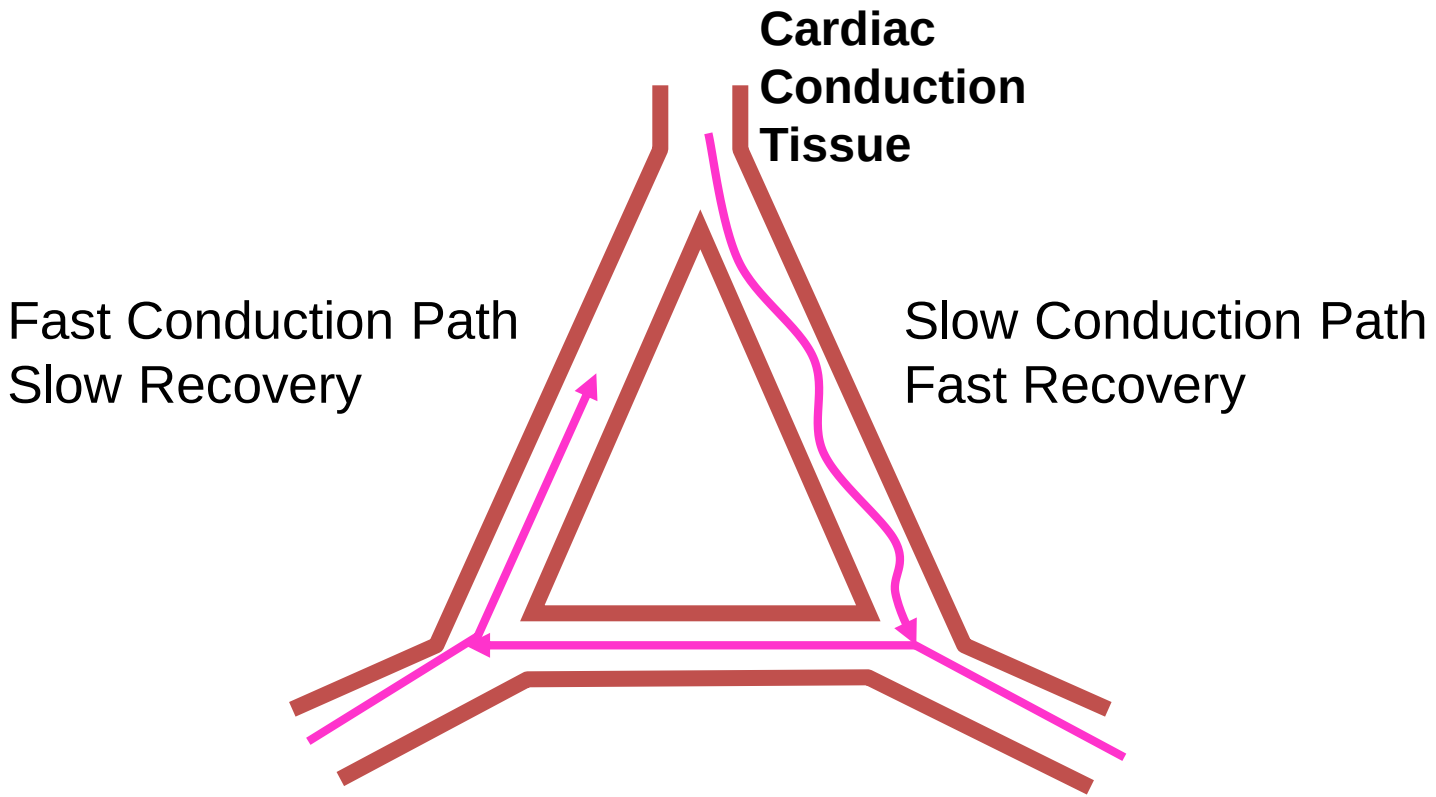
1. 2 distinct pathways that come together at beginning and end to form a loop.
2. A unidirectional block in one of those pathways.
3. Slow conduction in the unblocked pathway.

Reentry Mechanism



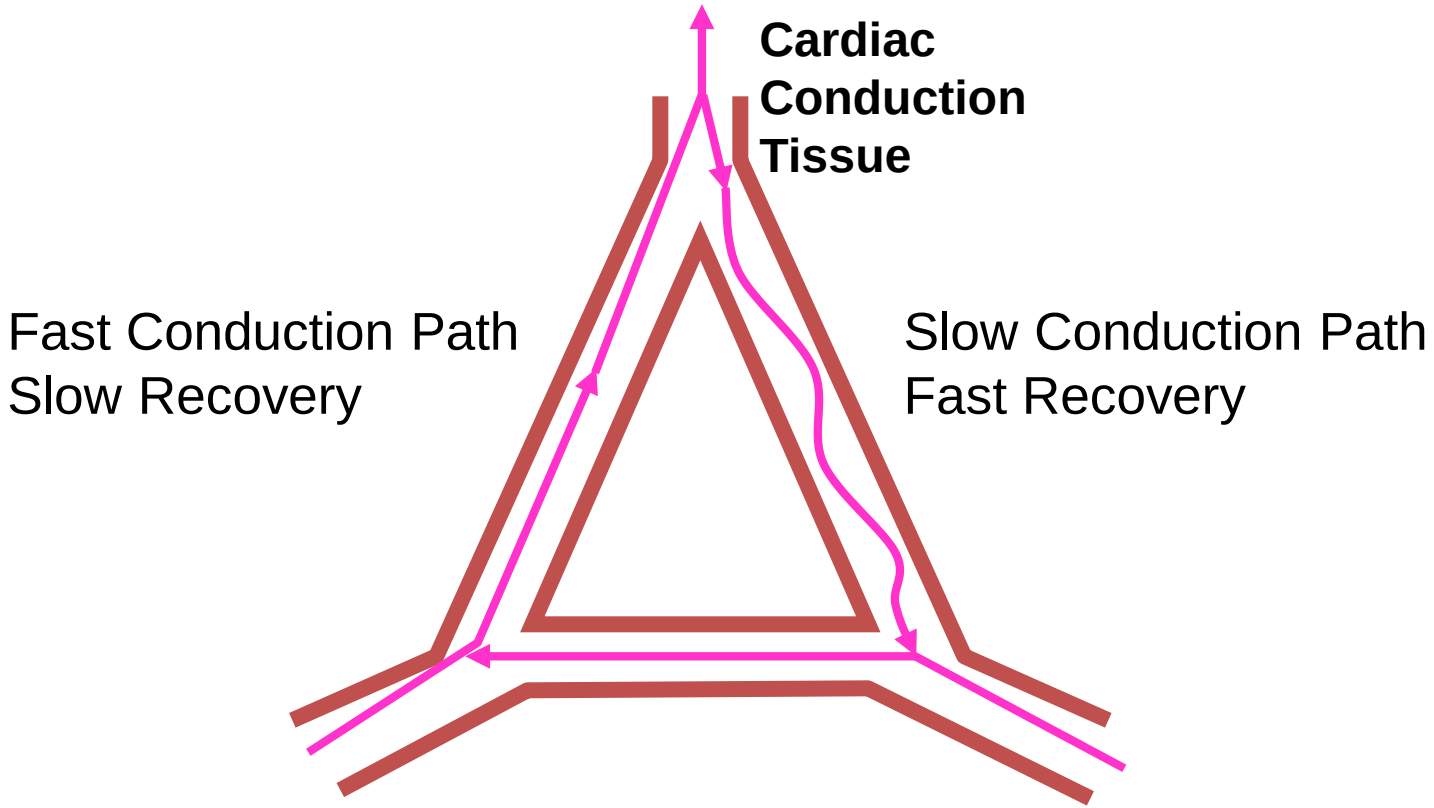
1. **An arrhythmia is triggered by a premature beat**
2. **The fast conducting pathway is blocked because of its long refractory period so the beat can only go down the slow conducting pathway**

Reentry Mechanism



3. The wave of excitation from the premature beat arrives at the distal end of the fast conducting pathway, which has now recovered and therefore travels retrogradely (backwards) up the fast pathway

Reentry Mechanism



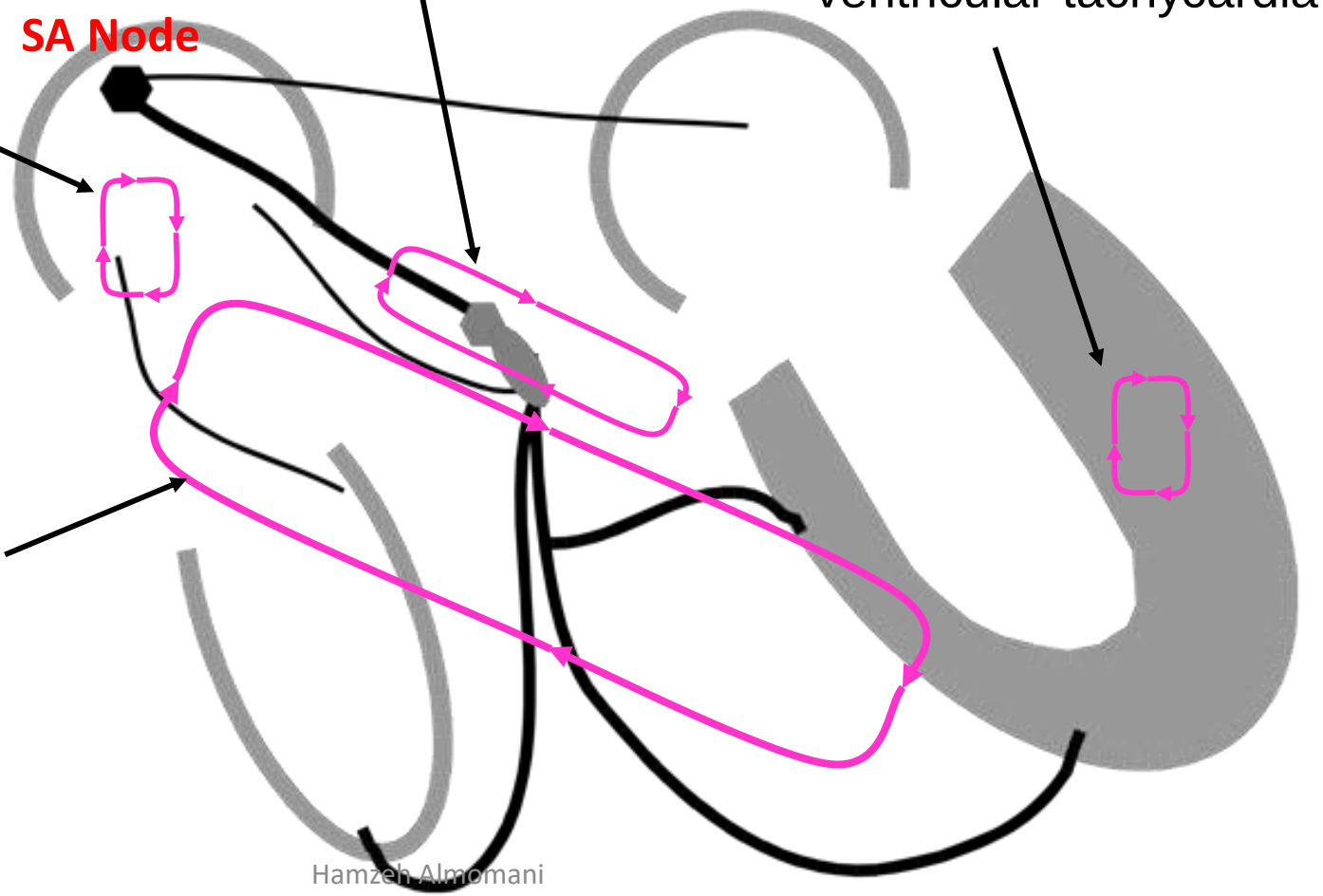
4. On arriving at the top of the fast pathway it finds the slow pathway has recovered and therefore the wave of excitation ‘re-enters’ the pathway and continues in a ‘circular’ movement. This creates the re-entry circuit

Reentry Circuits

AV Nodal Reentry
•SVT

Atrial Reentry
• atrial tachycardia
• atrial fibrillation
• atrial flutter

Ventricular Re-entry
• ventricular tachycardia



Atrio-Ventricular Reentry
• WPW
• SVT

Automaticity

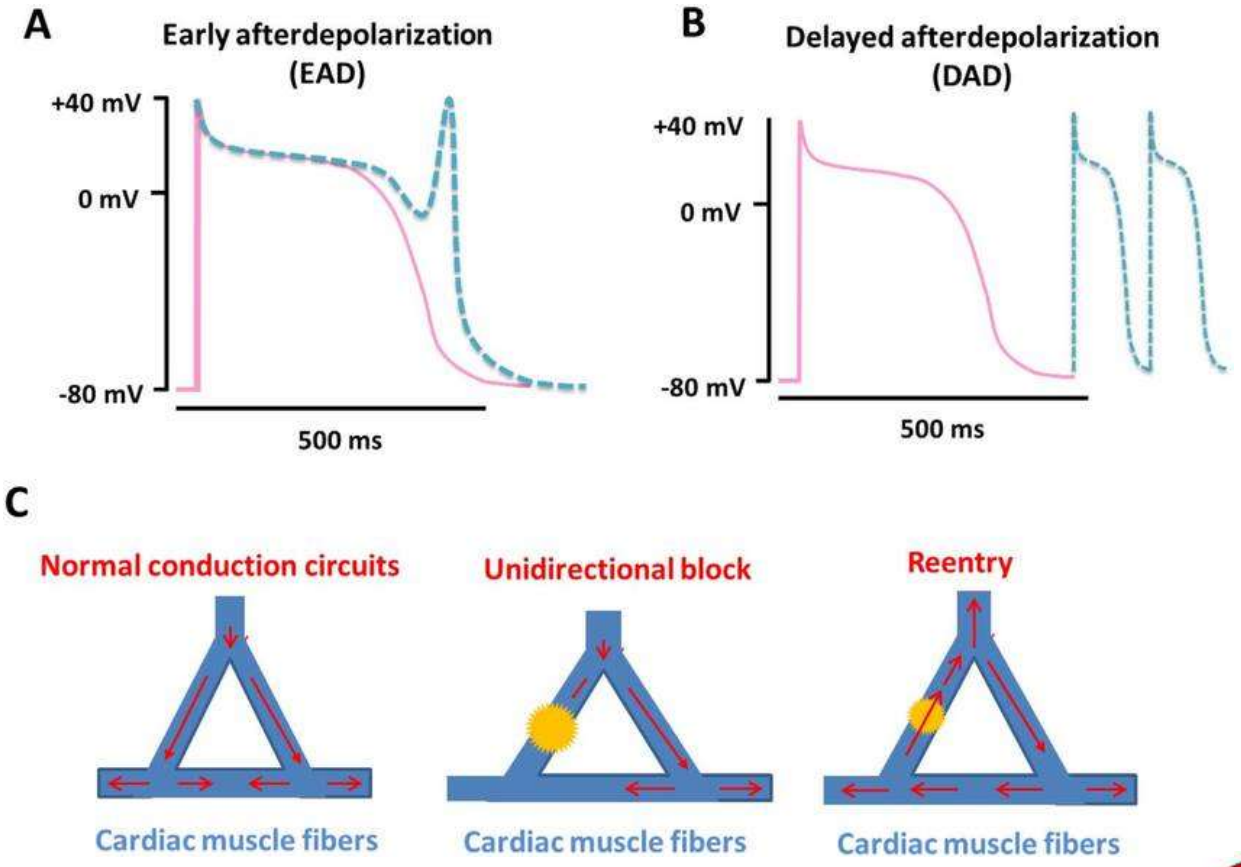
- **Automaticity is** the property of cardiac cells to generate spontaneous action potentials- When Heart cells other than those of the SA node depolarize faster than SA node cells, and take control as the cardiac pacemaker.
- Factors that enhance automaticity include:

  ↑ SANS, ↓ PANS, ↑ CO_2, ↓ O_2, ↑ H^+, ↑ stretch, hypokalemia and hypocalcaemia.

Examples: *Ectopic atrial tachycardia or multifocal tachycardia in patients with chronic lung disease OR ventricular ectopy after MI*

Triggered activity…

- is like a domino effect **where the arrhythmia is due to the preceding beat.**
  - **Delayed after-depolarizations** arise during the resting phase of the last beat and may be the cause of ***digitalis****-induced arrhythmias.*
  - **Early after-depolarizations** arise during the plateau phase or the repolarization phase of the last beat and **may be the cause of torsades de pointes (ex. Quinidine induced)**



Paroxysmal Supraventricular Tachycardia

- An abnormally rapid heart rhythm originating above the ventricles, often (but not always) with a narrow QRS complex
- Is the most common rhythm disturbance in children, Up to 13% of pediatric arrhythmias
- MOST COMMON FORMS:

1) **Atrioventricular reentrant tachycardia**
2) **Atrioventricular nodal reentrant tachycardia** (AVNRT)

- Other forms of SVT include
  - **Ectopic atrial tachycardia -EAT**
  - **Junctional tachycardia-JT**
  - **Multifocal atrial tachycardia -MAT**
  - **Atrial flutter**
  - **Atrial fibrillation**

CLINICAL FEATURES

Infants

- In infants, symptoms of SVT may include **pallor, fussiness, irritability, poor feeding, and/or cyanosis**. The symptoms can be subtle, and tachycardia may go unrecognized for long periods of time. Because of this, infants often present with symptoms of heart failure (eg, tachypnea, fatigue with feeding, poor weight gain)

Children

- Common symptoms of SVT in children and adolescents include **palpitations, chest discomfort, fatigue, and lightheadedness.**
- **Syncope** is less common and may be a warning sign for increased risk of sudden death

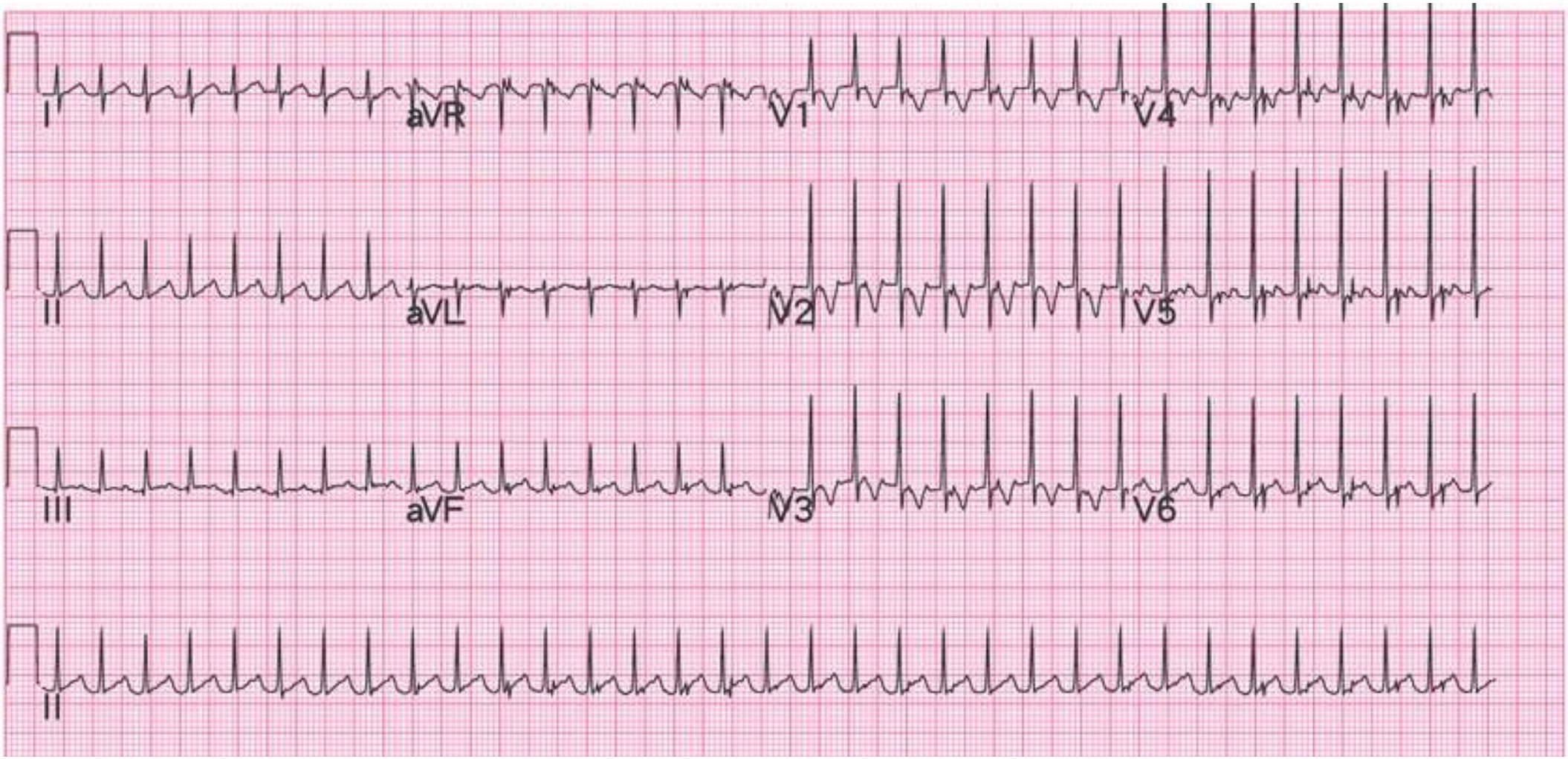
PSVT

- U**sually** paroxysmal.
- A**brupt onset and termination**.
- Mostly occurs **at rest.**
- Average duration of **10 to 15 minutes**; however, some episodes last only one to two minutes, while others persist for hours
- Most children tolerate episodes of tachycardia well. However, **prolonged episodes can precipitate heart failure.**

- Physical examination typically reveals **tachycardia without evidence of decompensation (**Some patients appear pale and diaphoretic, and the blood pressure may be reduced)

- Heart rates during SVT are age-dependent. Typical ranges are as follows:
  - **Infants: 220 to 280 beats per minute (bpm)**
  - **Children and adolescents: 180 to 240 bpm**.
- Infants with sustained SVT may have signs of heart failure .

ECG FINDINGS

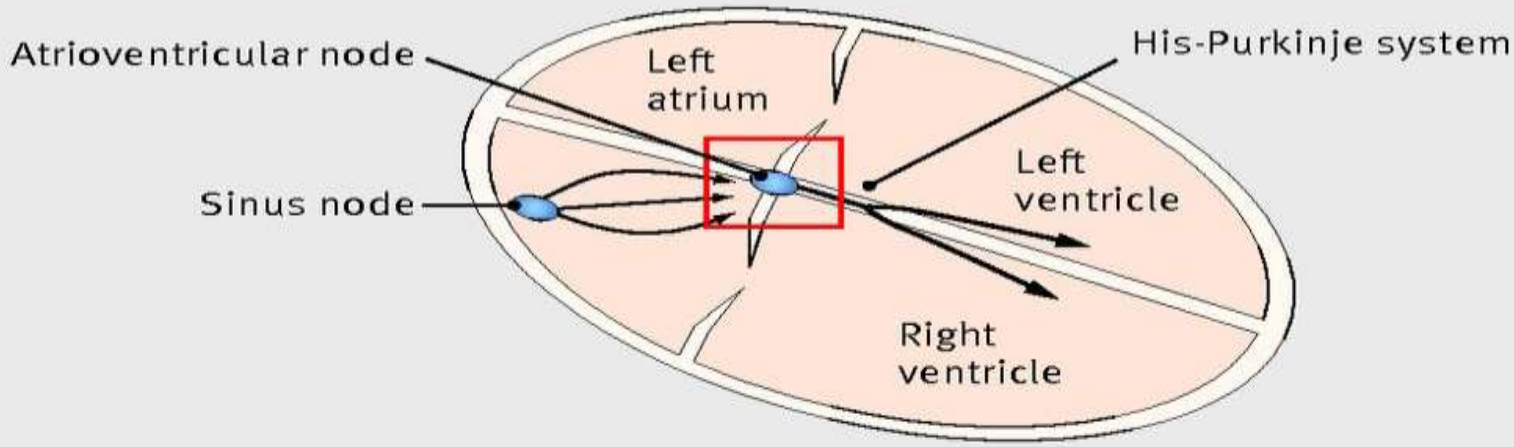


Narrow complex SVT @ 300 bpm

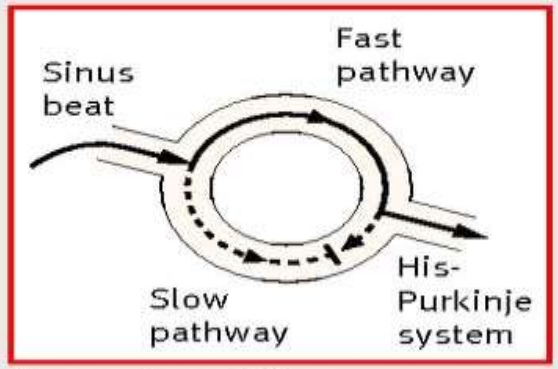
AVNRT

- Re-entrant circuit that involves AV node
- 2 conduction limbs – fast and slow
- Most commonly – anterograde slow pathway activation induced by PAC followed by **retrograde activation of the atria via the fast pathway (slow-fast) and anterograde ventricular activation** produces a narrow complex QRS tachycardia

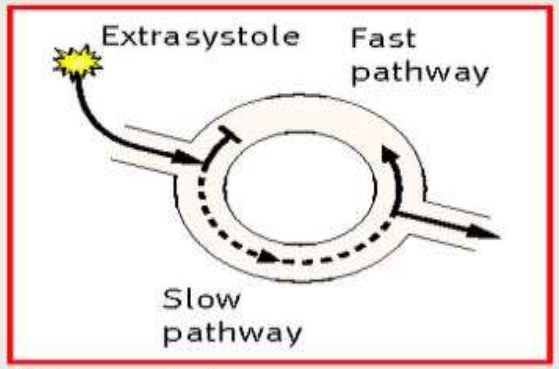
AVNRT



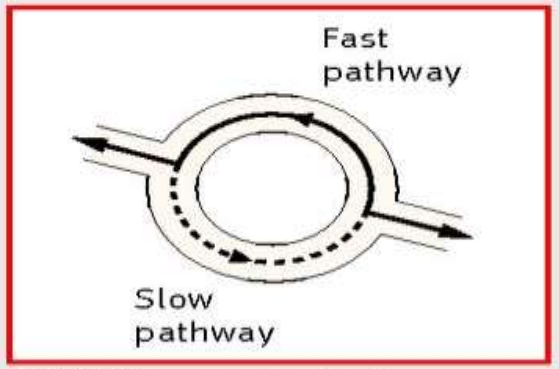
Mechanism at atrioventricular node



Normal condition:
Anterograde conduction down the fast pathway



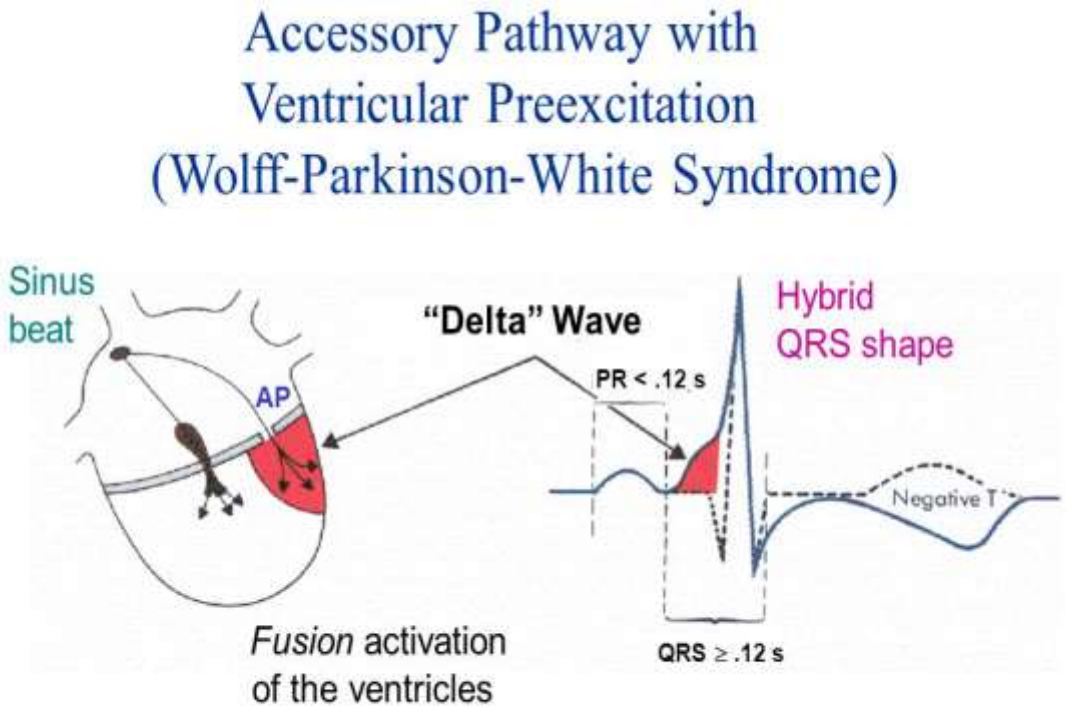
Extrasystole:
An extrasystole during the refractory period of the fast pathway propagates exclusively down the slow pathway. By the time the impulse reaches the distal end of the fast pathway it is no longer refractory and it conducts retrogradely



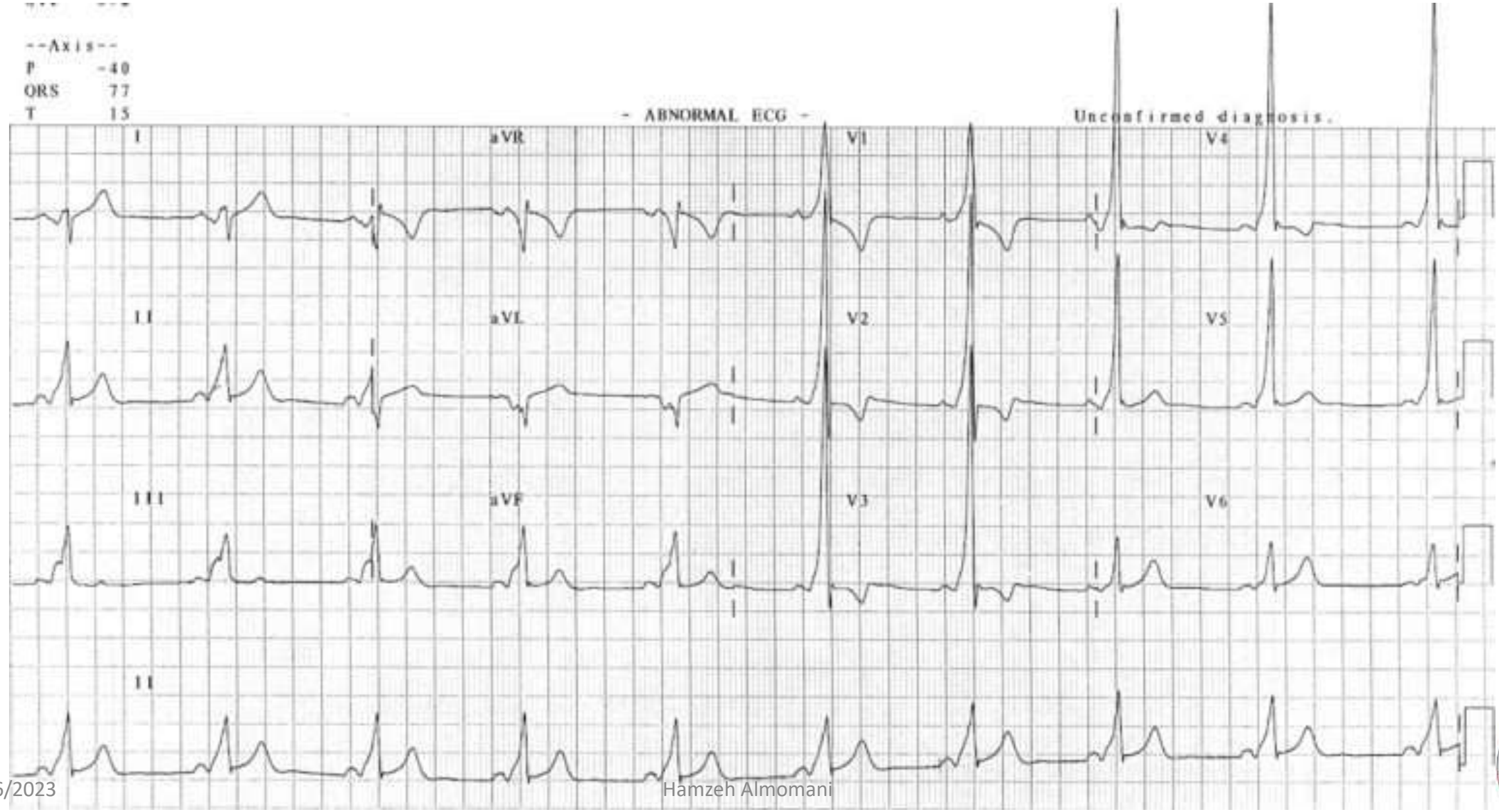
AVNRT perpetuated:
A re-entrant circuit is formed with retrograde conduction up the fast pathway, anterograde conduction down the slow pathway, and almost simultaneous activation of atria and ventricles

Ventricular Pre-excitation-WPW

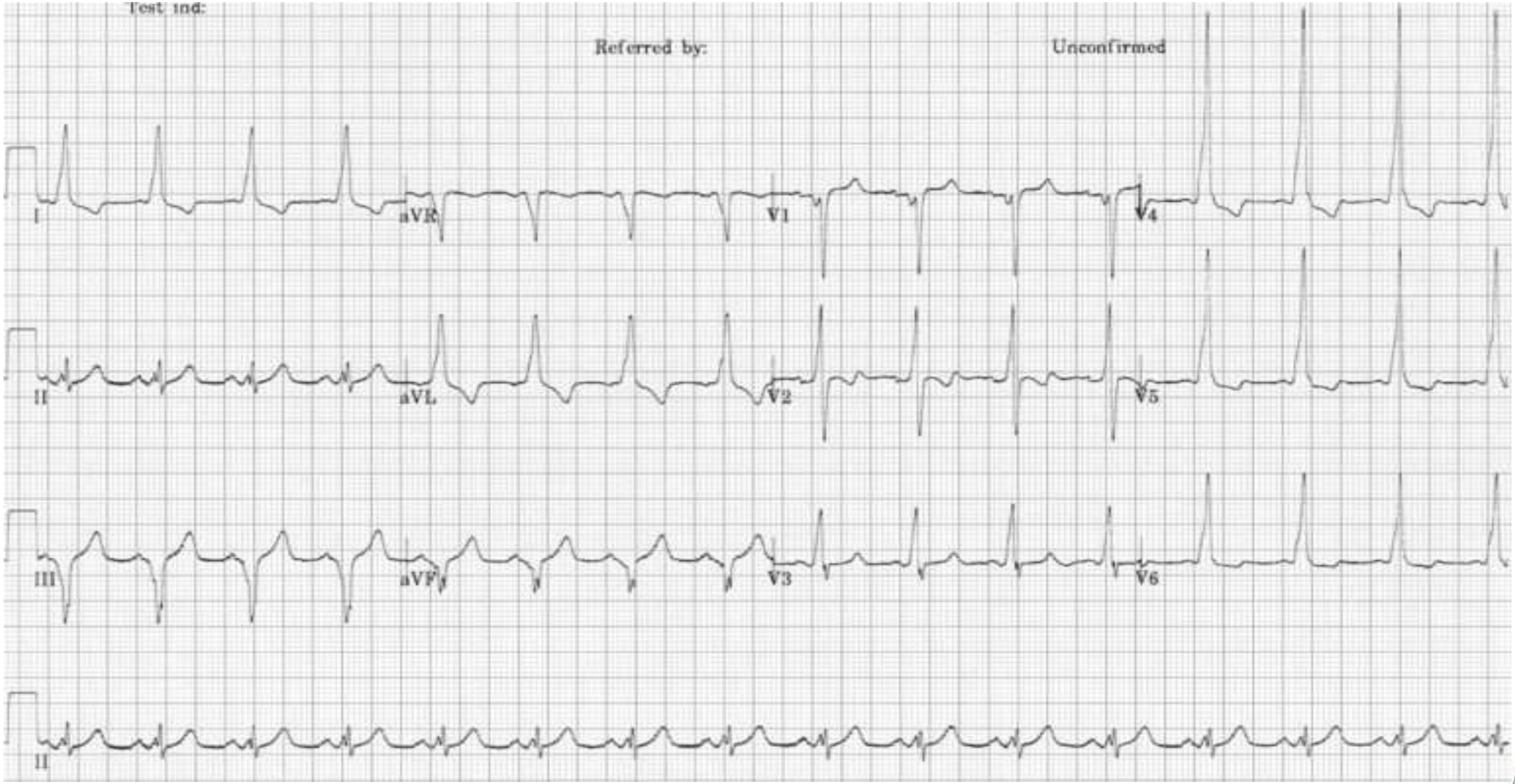
- **Pre-excitation refers to early activation of the ventricles due to impulses bypassing the AV node via an accessory pathway.**
- **ECG features of Pre-excitation in sinus rhythm are:**
  - **Short PR interval**
  - **Delta wave – slurring slow rise of initial portion of the QRS**
  - **QRS prolongation**
  - **ST Segment and T wave discordant changes – i.e. in the opposite direction to the major component of the QRS complex**



WPW Pattern


--Axis--
P -40
QRS 77
T 15
- ABNORMAL ECG -
Unconfirmed diagnosis.
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

WPW Pattern

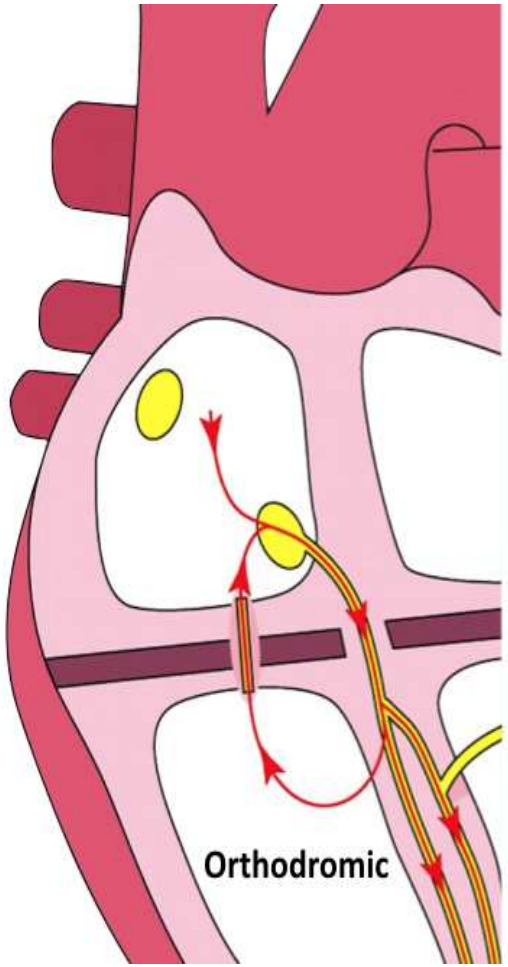

Test ind:
Referred by:
Unconfirmed
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

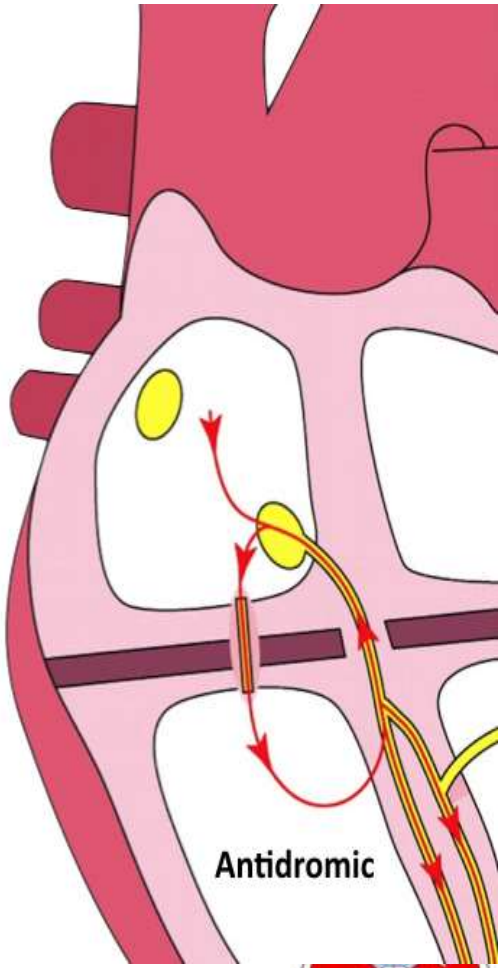
AVRT

- Re-entrant circuit that involves AV node and accessory pathway
- 2 conduction pathway – normal and accessory
- Accessory pathway as been described in the developing human heart and regresses by 20 weeks of gestation, ? if these fail to regress cause of AVRT.

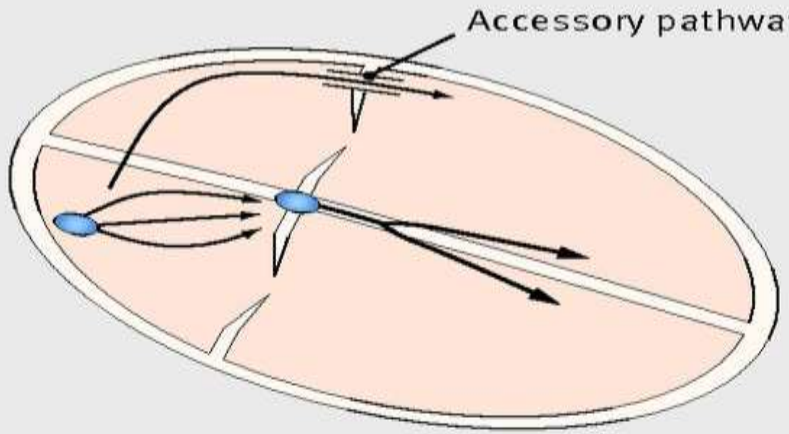
- ****Subtype – permanent junctional reciprocating tachycardia(PJRT)** <u>– only retograde conduction.</u>

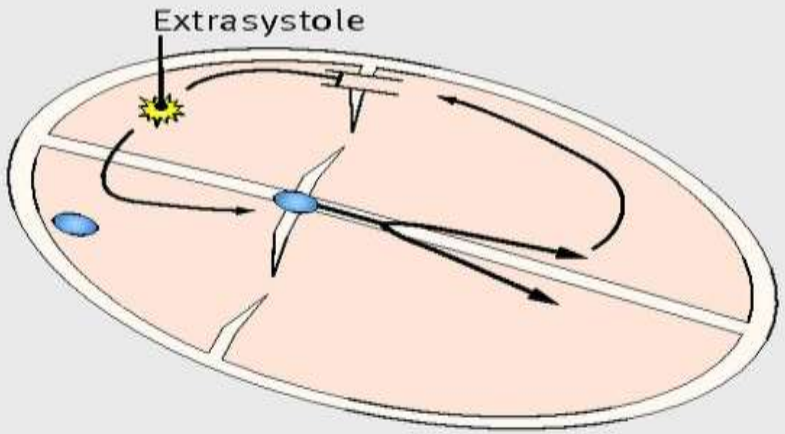
- AVRT are further divided in to **orthodromic** or **antidromic** c onduction based on direction of reentry conduction and ECG morphology.





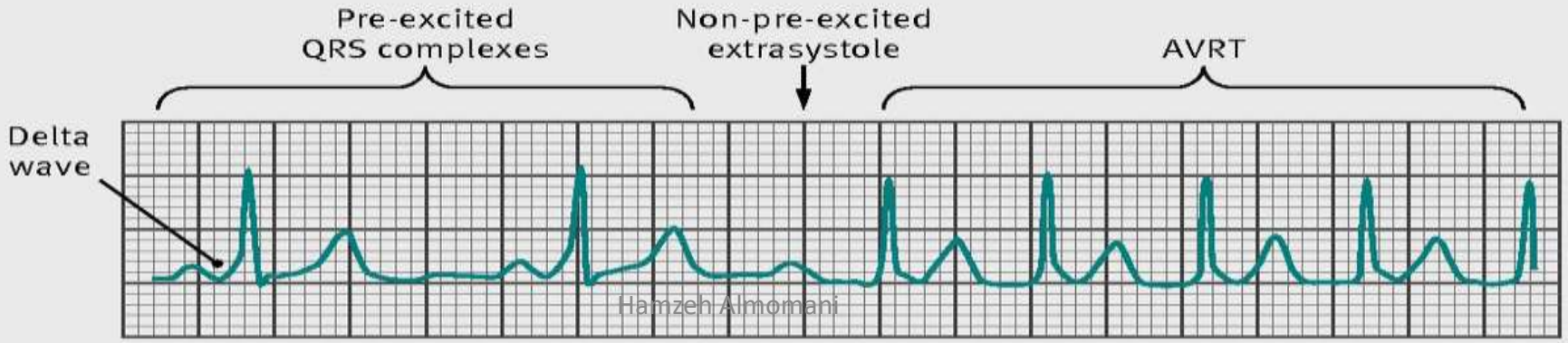
AVRT





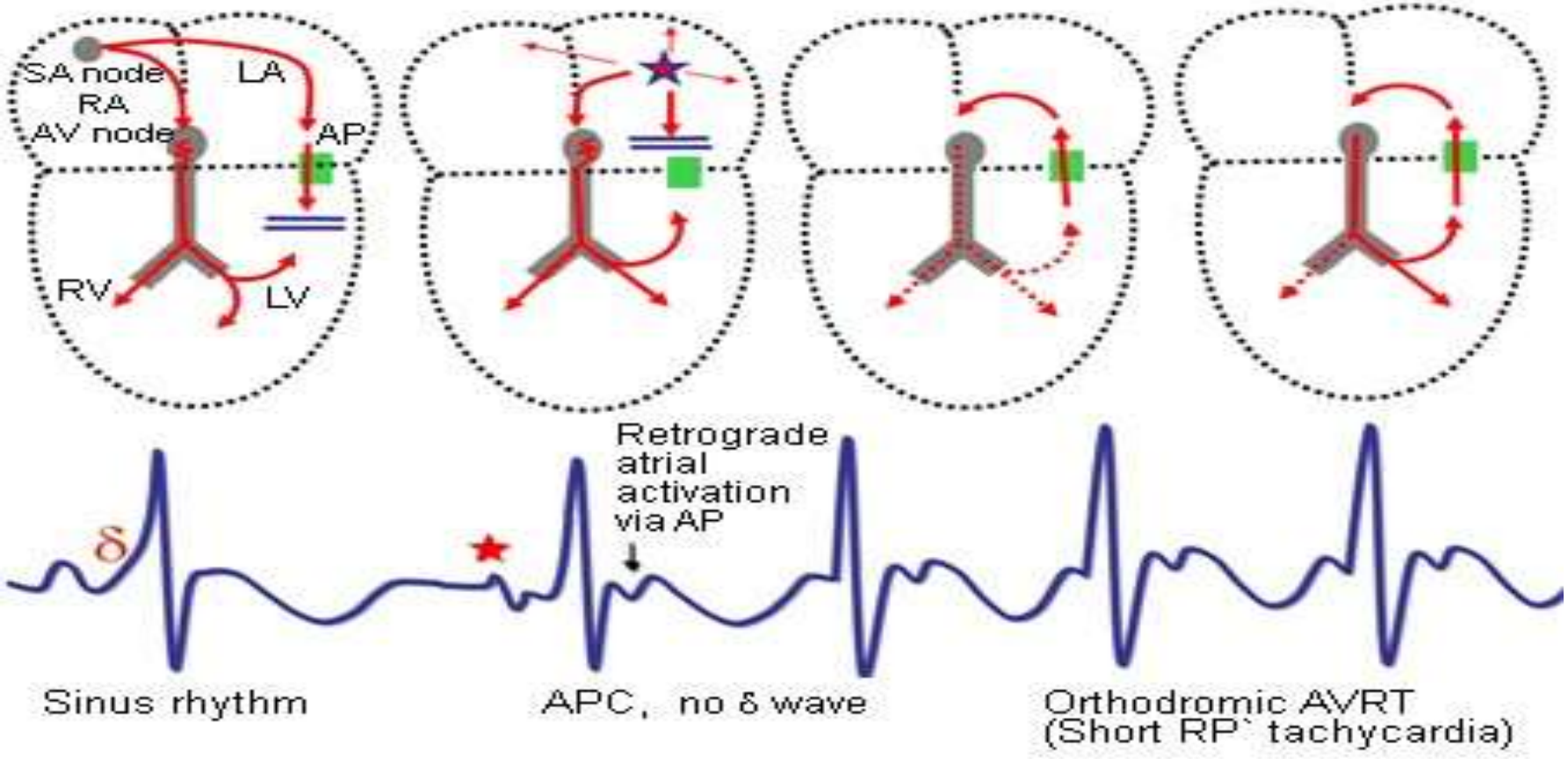
Delta wave:
Anterograde conduction via an accessory pathway usually produces pre-excitation of the ventricle, because the accessory pathway conducts more rapidly than the atrioventricular node. This early ventricular activation is manifest as a delta wave, which is a slurred upstroke at the start of the QRS complex. The terminal portion of the QRS complex is narrow, reflecting the rapid conduction via the His-Purkinje system once the atrioventricular node has been crossed

AVRT
Tachycardia is typically initiated by an extrasystole which occurs early and therefore cannot conduct via the accessory pathway but is able to conduct via the atrioventricular node (accessory pathway has a longer refractory period than the atrioventricular node). By the time the impulse reaches the accessory pathway from the ventricular side it is no longer refractory and can conduct retrogradely to the atrium

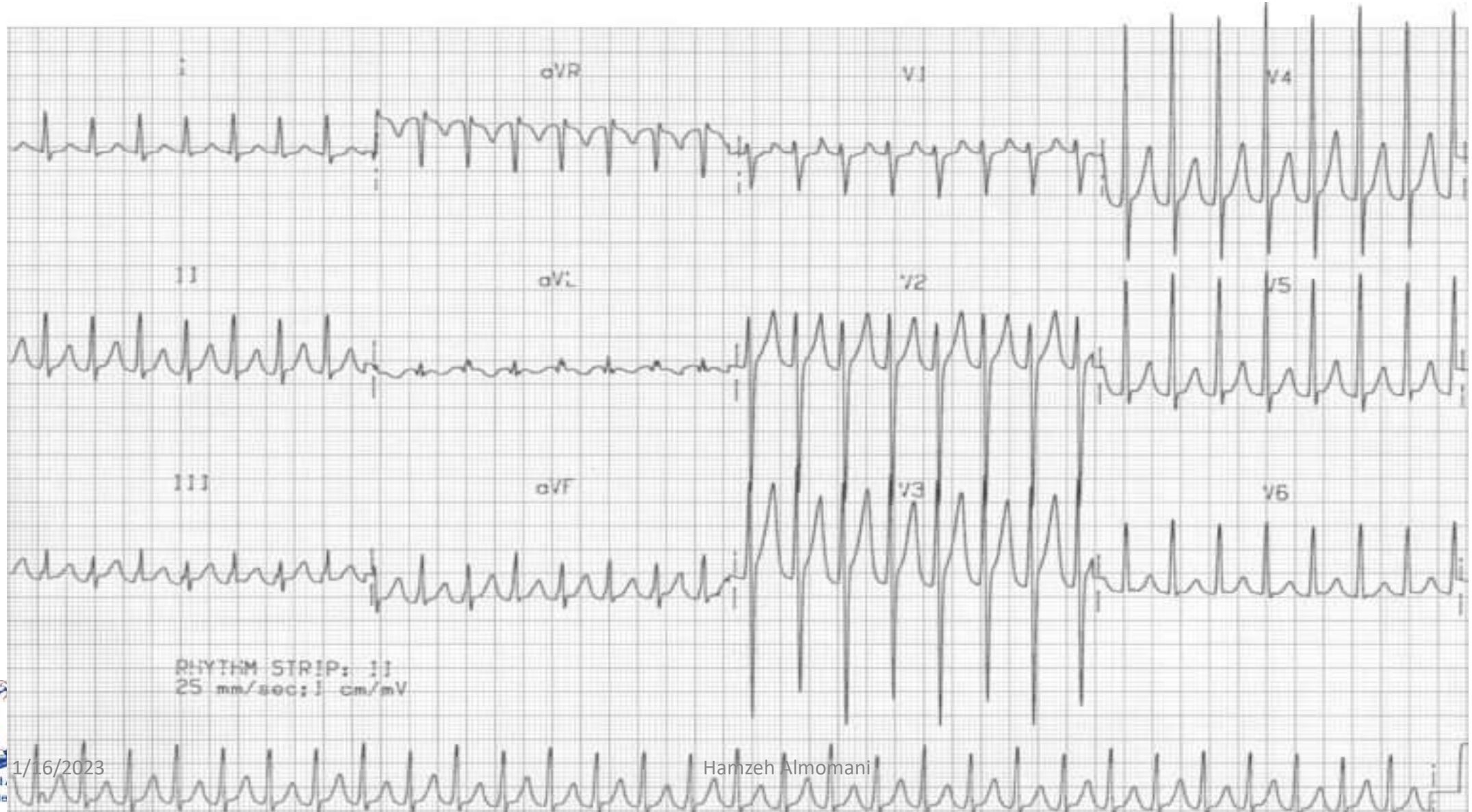


AVRT with orthodromic conduction

- **ECG features of AVRT with orthodromic conduction are:**
  - **Narrow QRS Complex (usually <120 ms) unless pre-existing bundle branch block, or rate-related aberrant conduction**

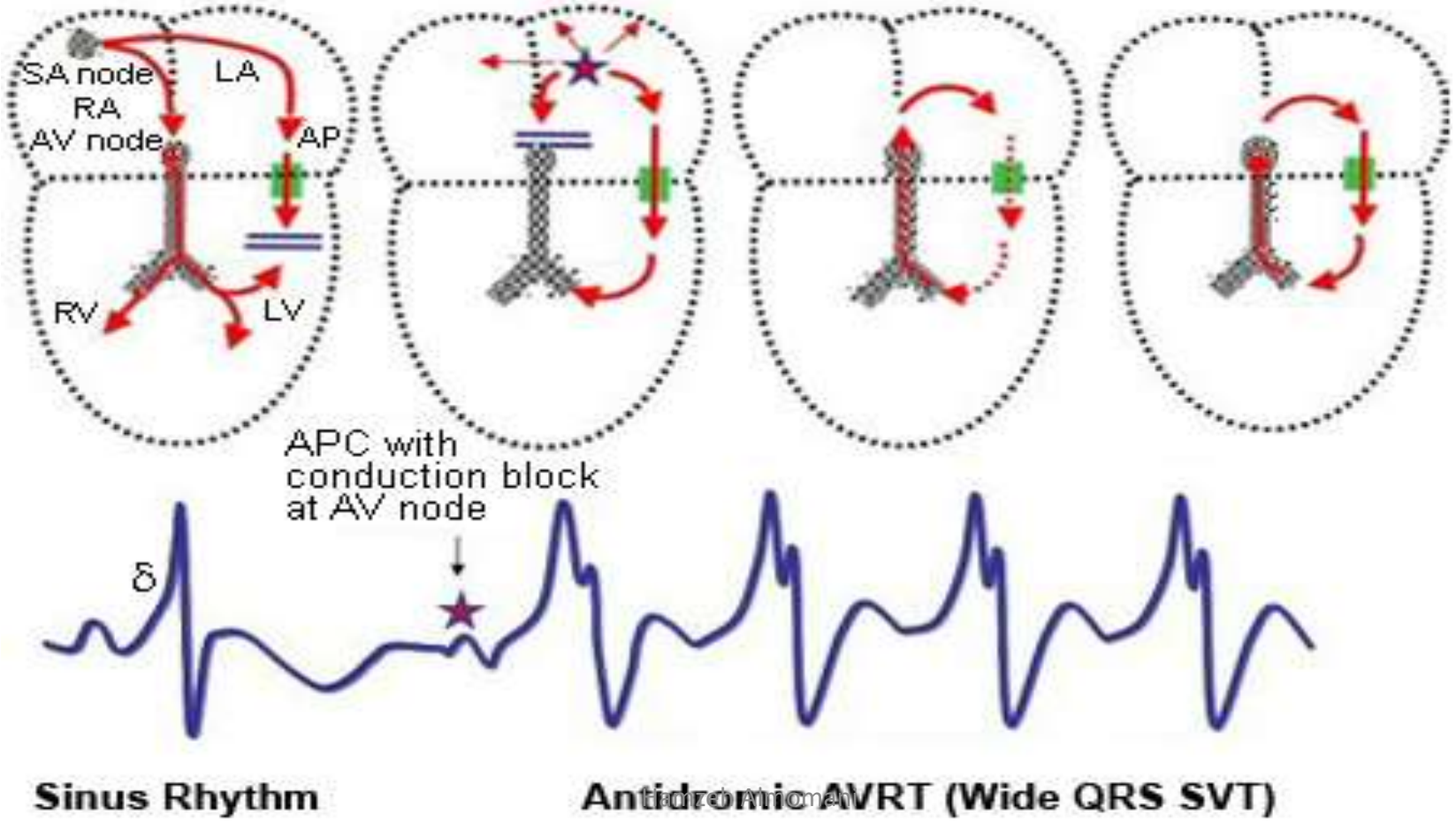


Orthodromic atrioventricular re-entry tachycardia (AVRT)
This rhythm is indistinguishable from AV-nodal re-entry tachycardia (AVNRT).



AVRT with Antidromic Conduction

- **ECG features of AVRT with antidromic conduction are:**
  - **Wide QRS complexes due to abnormal ventricular depolarisation via accessory pathway.**

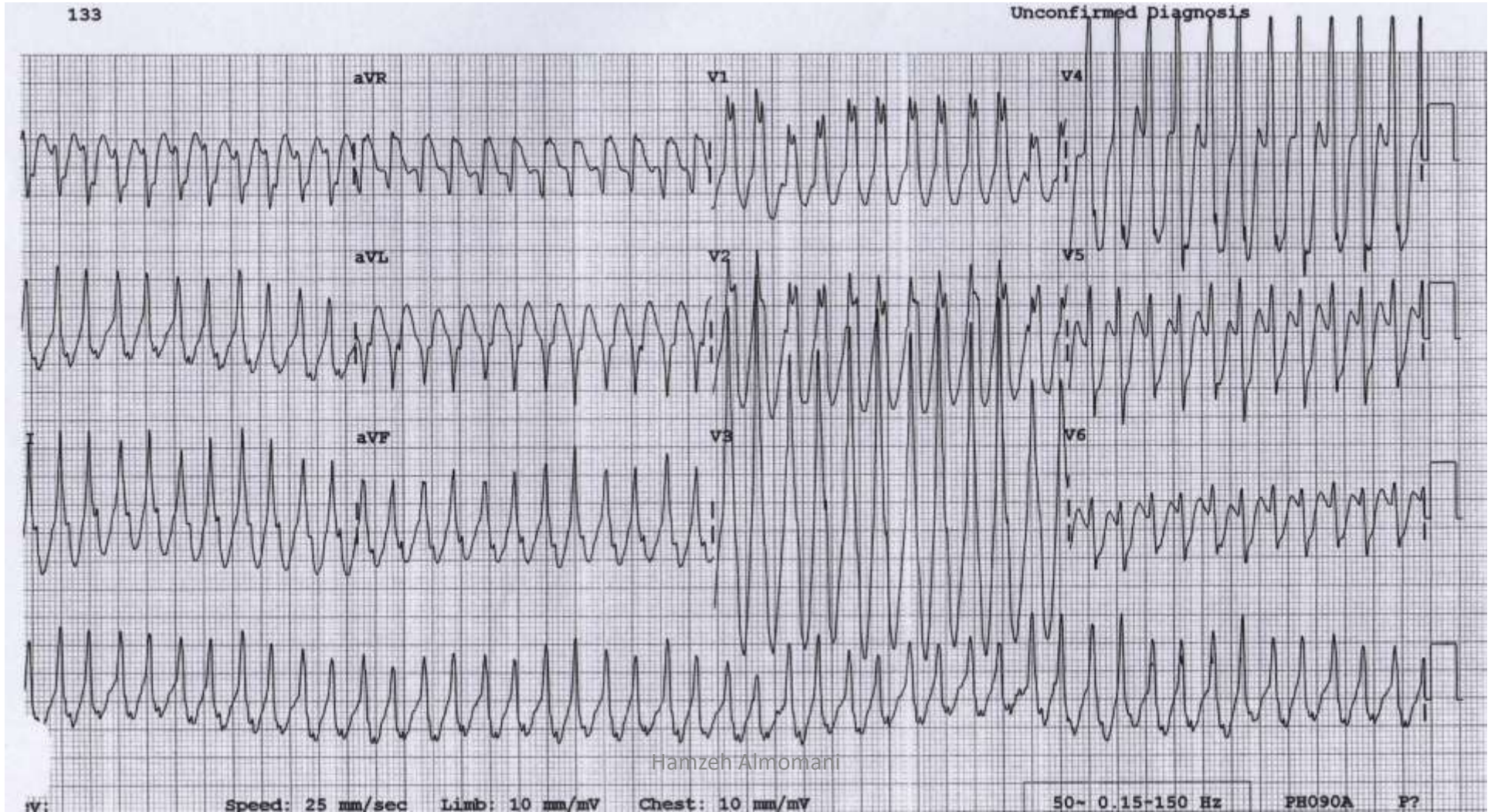


5 YEARS old boy, AVRT resolved with vagal manoeuvres

Broad complex tachycardia at ~280 bpm.

This could easily be mistaken for VT; **however, remember that >95% of broad complex tachycardias in paediatrics are actually SVT with aberrancy (usually a re-entrant tachycardia).**

This is an **antidromic atrioventricular re-entry** tachycardia due to WPW.



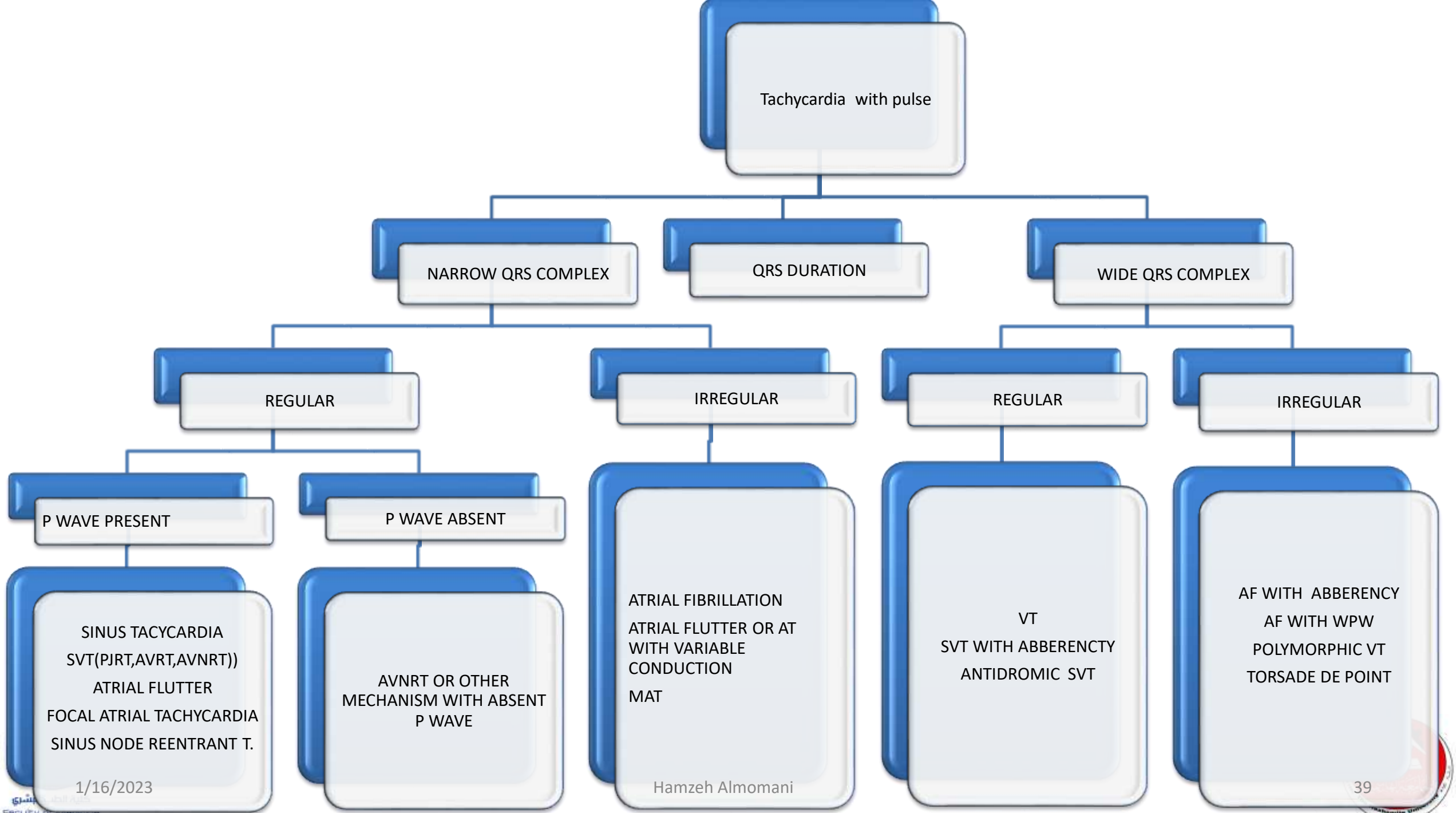
Diagnostic Assessment

❖ **Rapid hemodynamic assessment and initial management: WHY?**

- The most important initial clinical determination is whether there are signs of hemodynamic instability, including **hypotension, heart failure, shock, or decreased level of consciousness**.
- Unstable patients require immediate intervention to terminate the rhythm.

❖ ECG during arrhythmias :

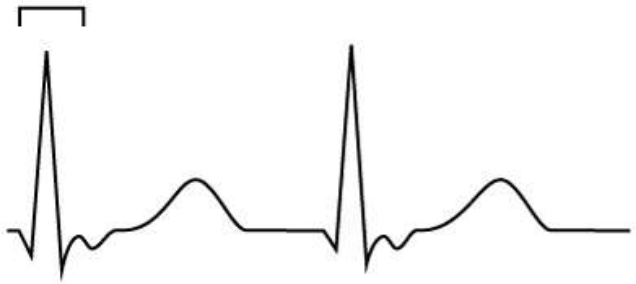
- **QRS width assessment**: In most cases, **the QRS complex is narrow (<80 msec**) An exception is SVT **with aberrant conduction, RBBB or pre-excitation.**
- **R-R wave assessment**: **constant regular except MAT, AF with variable block(P: flutter waves), and atrial fibrillation (absent p wave)**
- **P wave assessment**
  - **AVNRT, AVRT, PJRT: retrograde p wave, R-P interval assessment**
  - **MAT: three or more discrete p wave morphologies**
  - **ATRIAL FLUTTER: flutter waves**
  - **ATRIAL FIBRILLATION: absence of p waves**


Tachycardia with pulse
NARROW QRS COMPLEX
QRS DURATION
WIDE QRS COMPLEX
REGULAR
IRREGULAR
REGULAR
IRREGULAR
P WAVE PRESENT
P WAVE ABSENT
SINUS TACYCARDIA
SVT(PJRT,AVRT,AVNRT))
ATRIAL FLUTTER
FOCAL ATRIAL TACHYCARDIA
SINUS NODE REENTRANT T.
AVNRT OR OTHER MECHANISM WITH ABSENT P WAVE
ATRIAL FIBRILLATION
ATRIAL FLUTTER OR AT WITH VARIABLE CONDUCTION
MAT
VT
SVT WITH ABBERENCTY
ANTIDROMIC SVT
AF WITH ABBERENCY
AF WITH WPW
POLYMORPHIC VT
TORSADE DE POINT

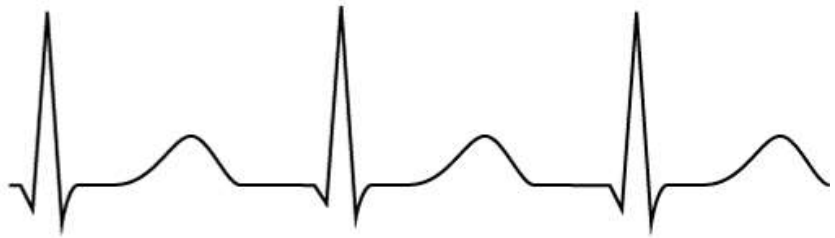
AVNRT v/s Orthodromic AVRT v/s PJRT

- The following criteria are useful in making this distinction
  - Buried p wave **in or just at the end** of the QRS complex(create a small **"pseudo r' wave" in lead V1 or a "pseudo S wave" in the inferior leads**. **If such terminal r' or S waves are not present during sinus rhythm, their appearance during SVT can be assumed to be diagnostic of AVNRT)**, which is typical of **AVNRT**
  - **An RP interval ≥70 msec is more characteristic of AVRT than AVNRT**
  - An extremely **long RP interval and normal PR (RP>>PR), and a P wave axis that is directed superior (negative in leads II, III, and aVF**) is seen in **permanent junctional reciprocating tachycardia**.

RP interval	Short and <70 ms
	Typical AVNRT. AVRT is unusual.

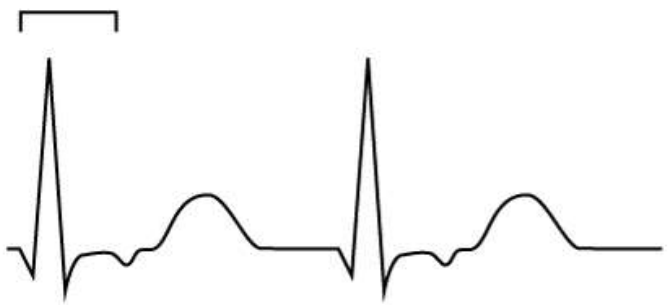


RP interval	No visible P-wave
	Typical AVNRT

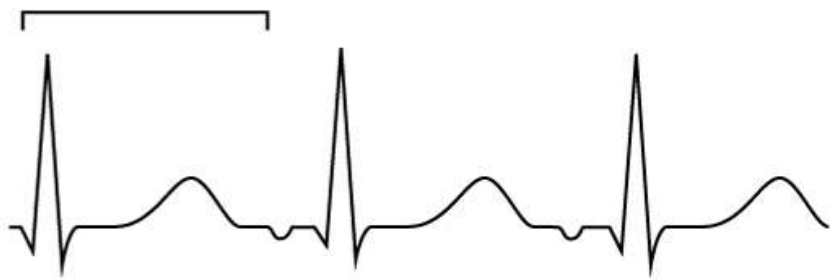


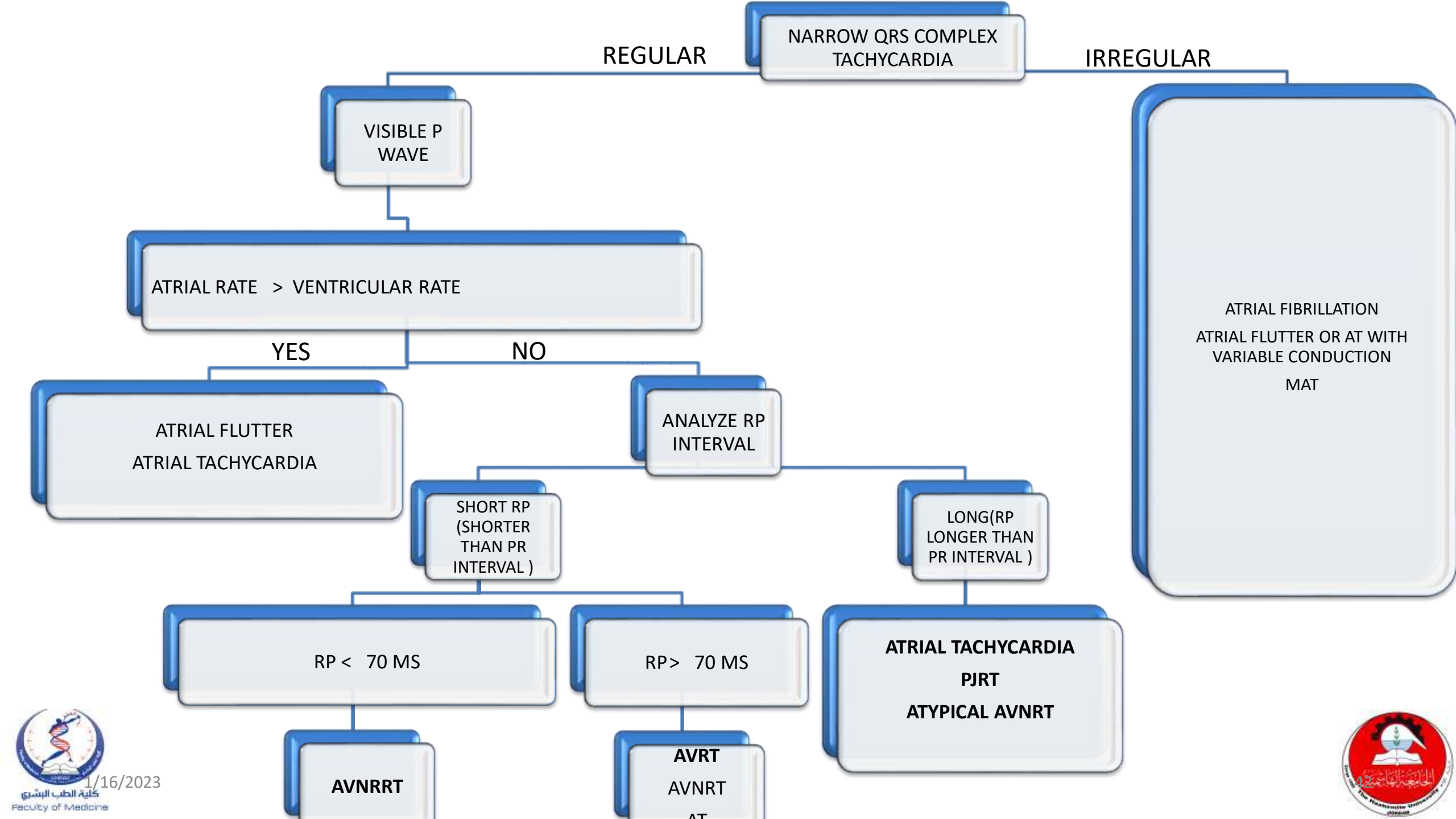
If the P-wave is invisible, it is classified as short RP interval.

RP interval	Short but >70 ms
	In most cases AVRT. Occasionally atypical AVNRT or AT.



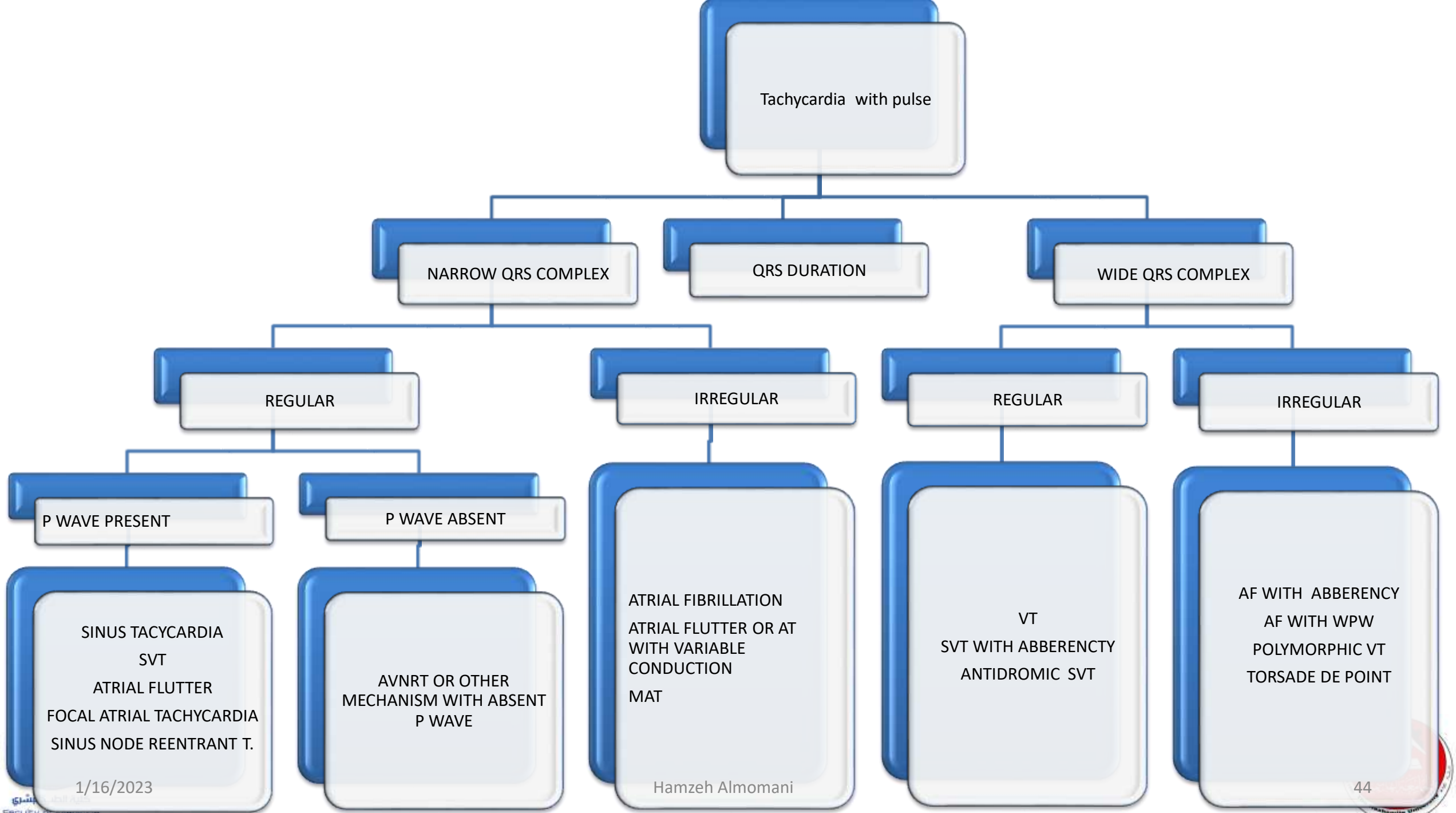
RP interval	Long
	In most cases AT. Occassionally atypical AVNRT. Rarely PJRT.

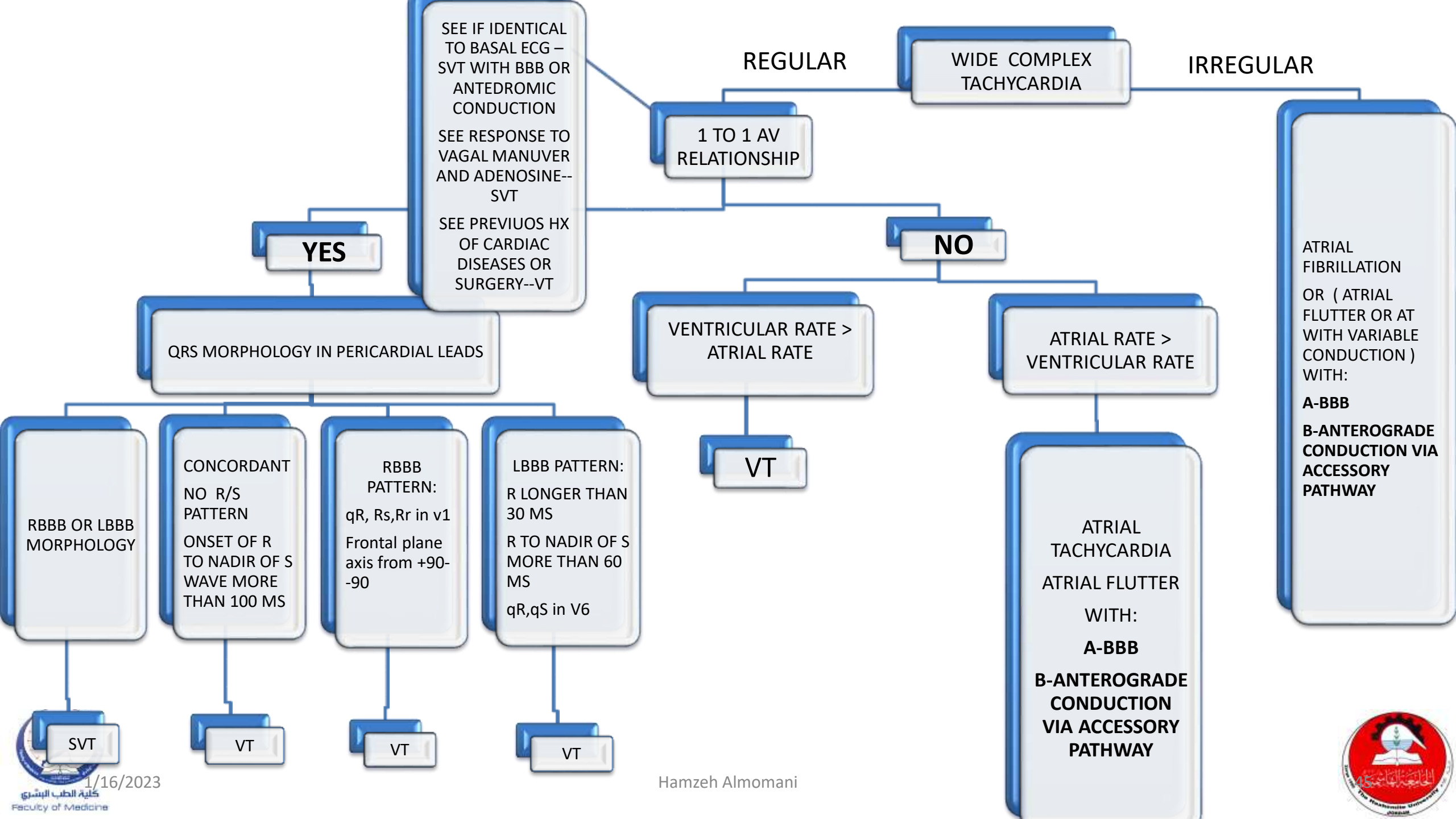



NARROW QRS COMPLEX TACHYCARDIA
REGULAR
IRREGULAR
VISIBLE P WAVE
ATRIAL FIBRILLATION
ATRIAL FLUTTER OR AT WITH VARIABLE CONDUCTION
MAT
ATRIAL RATE > VENTRICULAR RATE
YES
NO
ATRIAL FLUTTER
ATRIAL TACHYCARDIA
ANALYZE RP INTERVAL
SHORT RP (SHORTER THAN PR INTERVAL)
LONG(RP LONGER THAN PR INTERVAL)
RP < 70 MS
RP> 70 MS
ATRIAL TACHYCARDIA
PJRT
ATYPICAL AVNRT
AVNRRT
AVRT
AVNRT
AT

Wide QRS complex tachycardia

- Wide QRS complex tachycardia should generally **prompt consideration of ventricular tachycardia (VT).**
- However, in children, **most wide QRS complex tachycardia that occurs with a regular rate represents SVT, not VT.**
- SVT can have a wide QRS complex :
  - if the mechanism is **antidromic AVRT**
  - **or if there is aberrant conduction** (eg, functional bundle branch block or "bundle branch aberration")
- The ECG can be helpful in differentiating between VT and SVT. However, making the distinction can be challenging, especially for clinicians unfamiliar with interpreting pediatric ECGs.


Tachycardia with pulse
NARROW QRS COMPLEX
QRS DURATION
WIDE QRS COMPLEX
REGULAR
IRREGULAR
REGULAR
IRREGULAR
P WAVE PRESENT
P WAVE ABSENT
SINUS TACYCARDIA
SVT
ATRIAL FLUTTER
FOCAL ATRIAL TACHYCARDIA
SINUS NODE REENTRANT T.
AVNRT OR OTHER MECHANISM WITH ABSENT P WAVE
ATRIAL FIBRILLATION
ATRIAL FLUTTER OR AT WITH VARIABLE CONDUCTION
MAT
VT
SVT WITH ABBERENCTY
ANTIDROMIC SVT
AF WITH ABBERENCY
AF WITH WPW
POLYMORPHIC VT
TORSADE DE POINT


WIDE COMPLEX TACHYCARDIA
REGULAR
IRREGULAR
SEE IF IDENTICAL TO BASAL ECG – SVT WITH BBB OR ANTEDROMIC CONDUCTION
SEE RESPONSE TO VAGAL MANUVER AND ADENOSINE--SVT
SEE PREVIUOS HX OF CARDIAC DISEASES OR SURGERY--VT
1 TO 1 AV RELATIONSHIP
YES
NO
QRS MORPHOLOGY IN PERICARDIAL LEADS
VENTRICULAR RATE > ATRIAL RATE
VT
ATRIAL RATE > VENTRICULAR RATE
ATRIAL FIBRILLATION
OR (ATRIAL FLUTTER OR AT WITH VARIABLE CONDUCTION) WITH:
A-BBB
B-ANTEROGRADE CONDUCTION VIA ACCESSORY PATHWAY
RBBB OR LBBB MORPHOLOGY
CONCORDANT
NO R/S PATTERN
ONSET OF R TO NADIR OF S WAVE MORE THAN 100 MS
RBBB PATTERN:
qR, Rs,Rr in v1
Frontal plane axis from +90--90
LBBB PATTERN:
R LONGER THAN 30 MS
R TO NADIR OF S MORE THAN 60 MS
qR,qS in V6
ATRIAL TACHYCARDIA
ATRIAL FLUTTER
WITH:
A-BBB
B-ANTEROGRADE CONDUCTION VIA ACCESSORY PATHWAY
SVT
VT
VT
VT

- **ECG in sinus rhythm**
  - The ECG in sinus rhythm is normal in patients with concealed accessory pathways or AVNRT and In patients with pre-excitation ECG shows Wolff-Parkinson-White (WPW) pattern

- **Ambulatory monitoring-Holter 24 hr**
  - Ambulatory monitoring helps to establish the frequency and duration of SVT; however, it is less useful in making a diagnosis, since only a few ECG leads are used
- **Exercise testing-stress test**
- **Electrophysiologic evaluation**

Acute treatment

- **12-lead electrocardiogram (ECG)**
- **Hemodynamic assessment**

A- ***If the child is hemodynamically unstable***

(hypotension, heart failure, shock, or decreased level of consciousness)

– synchronized direct current cardioversion with 0.5-2 J/kg should be performed .

B- *If the child is hemodynamically stable:*

1- Vagal maneuvers should be attempted to terminate the tachycardia .

- infants: **ice plus water in bag placed on face for up to 10 seconds – often effective.**
- Older children: **valsalva manoeuvre (10 - seconds), deep inspiration/cough/gag reflex, headstand.**

2- Adenosine: If does not convert to normal sinus rhythm with vagal maneuvers, **we recommend intravenous adenosine** rather than other antiarrhythmic drugs

- ❖Adenosine should be administered in an initial bolus dose of 0.1 mg/kg, followed by a rapid saline flush. If the rhythm does not convert with the initial dose, adenosine is repeated with increases to a maximum dose of 0.25 to 0.35 mg/kg or a total dose of 12 mg.
- ❖Transient AV nodal block as well as sinus node block, negative Chronotrope, inotrope.
- ❖Side effects: flushing, nausea, dyspnea, bronchospasm are short lived.

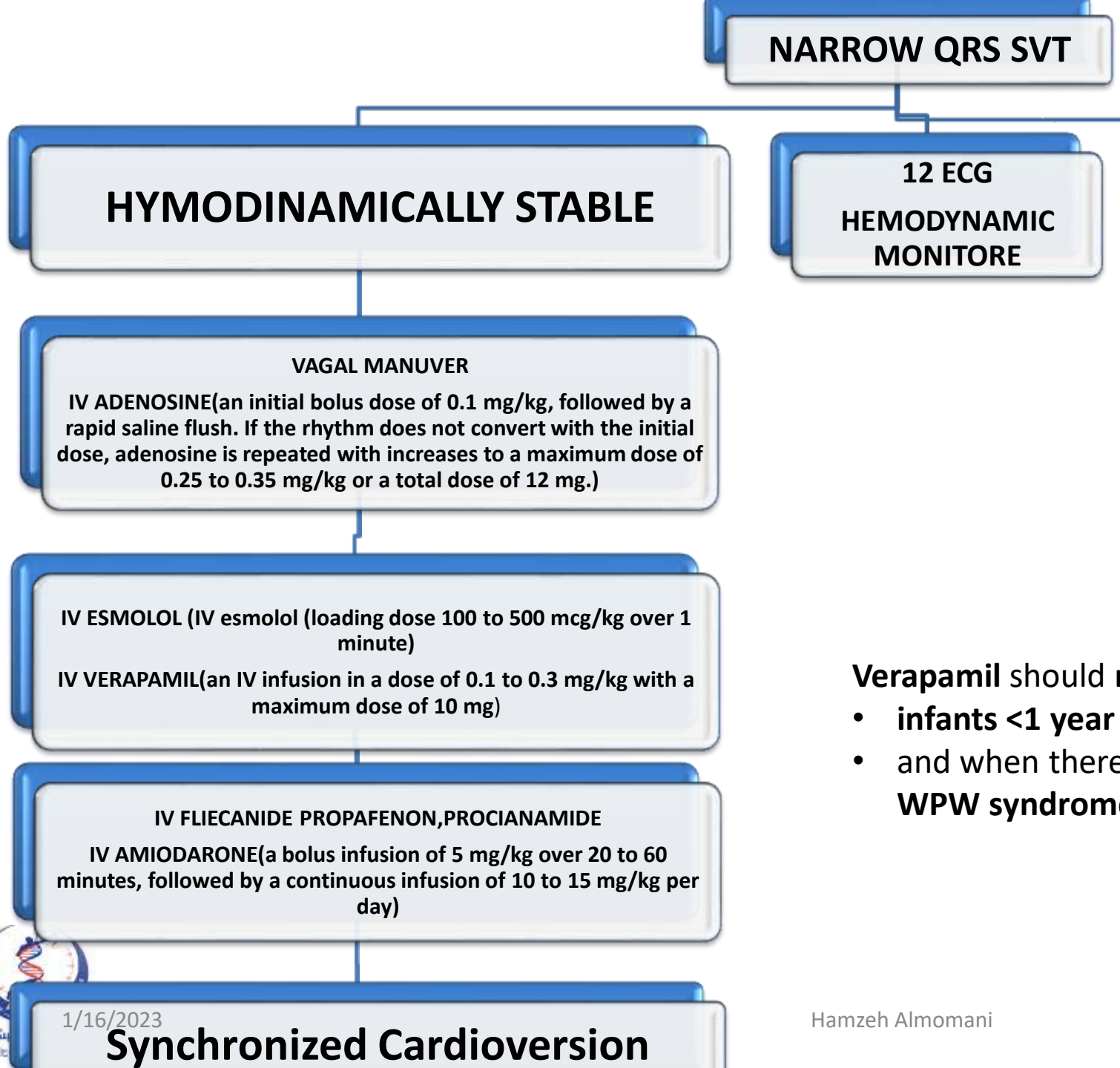
IF No Conversion to Sinus Rhythm

- IV **ESMOLOL** (IV esmolol (loading dose 100 to 500 mcg/kg over 1 minute) or
- IV **VERAPAMIL**(an IV infusion in a dose of 0.1 to 0.3 mg/kg with a maximum dose of 10 mg)

****

❑ IV FLIECANIDE PROPAFENON,PROCIANAMIDE

❑ IV **AMIODARONE**(a bolus infusion of 5 mg/kg over 20 to 60 minutes, followed by a continuous infusion of 10 to 15 mg/kg per day)

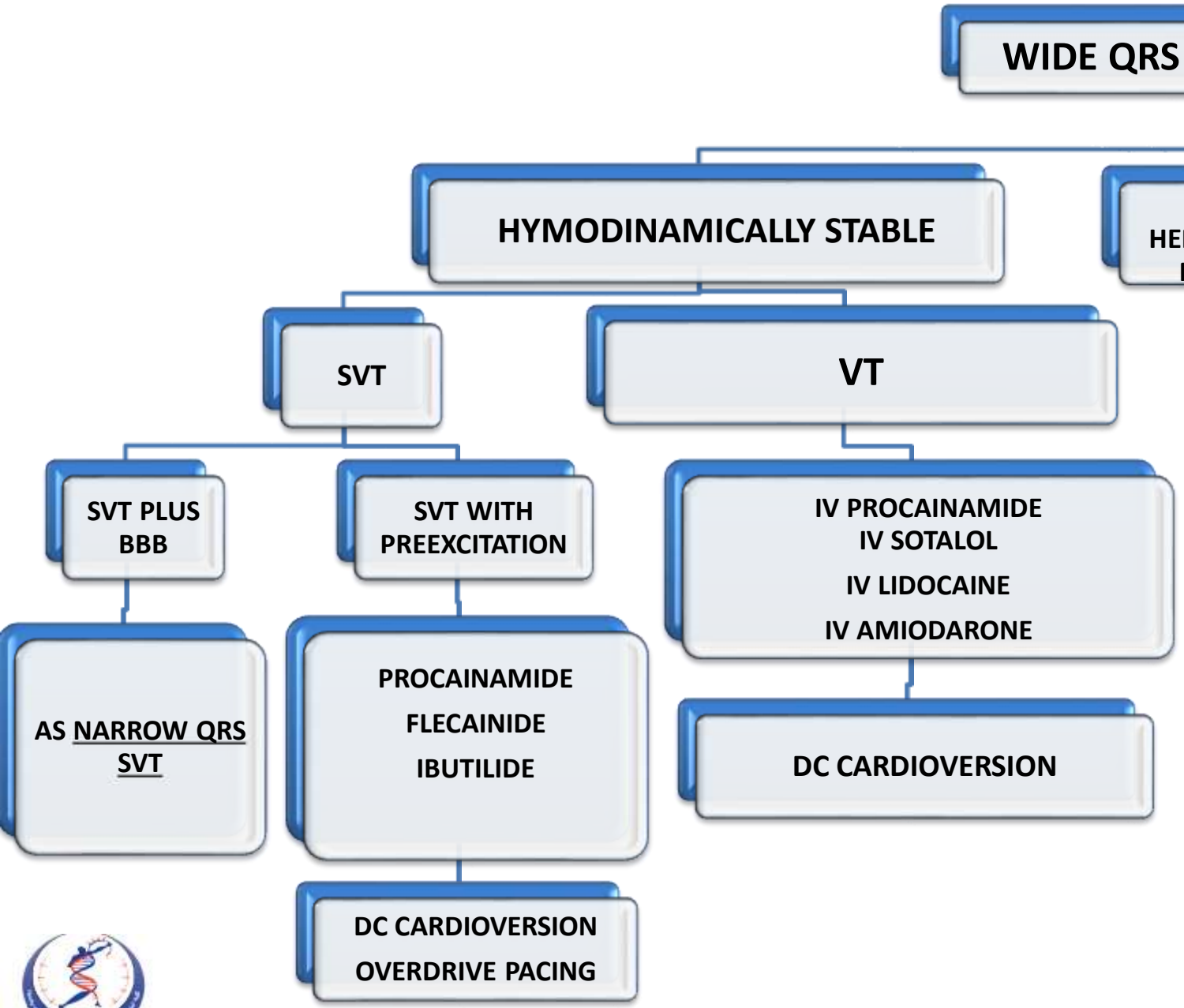


Verapamil should **not** be used In

- **infants <1 year** old,
- and when there IS **heart failure,** suspected to have **WPW syndrome, wide QRS complex tachycardia**

Further Workup

- Repeat ECG once in NSR
- LABWORK – ELECTROLYTES, TSH, CBC
- ADMISSION IF **less than one year , and hemodynamically unstable**
- FIRST TIME EPISODE – observation overnight
- Cardiology consult – management of meds



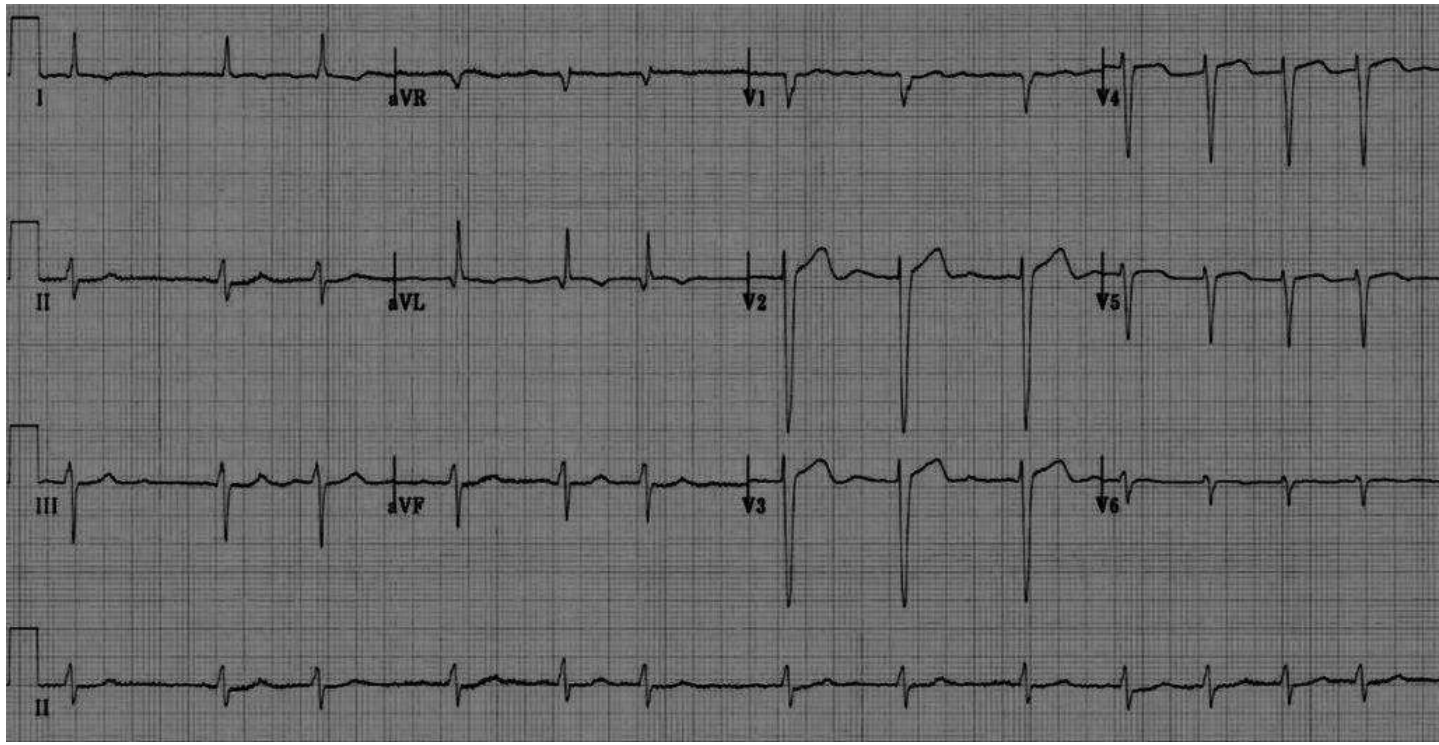
- Amiodarone in poor myocardial contraction
- Amiodarone is considered **contraindicated in preexcited atrial fibrillation**, which has an irregular and wide QRS complex.

- **Digoxin and calcium channel blockers** should not be prescribed in patients with WPW prior to full cardiology evaluation.

ATRIAL FIBRILLATION

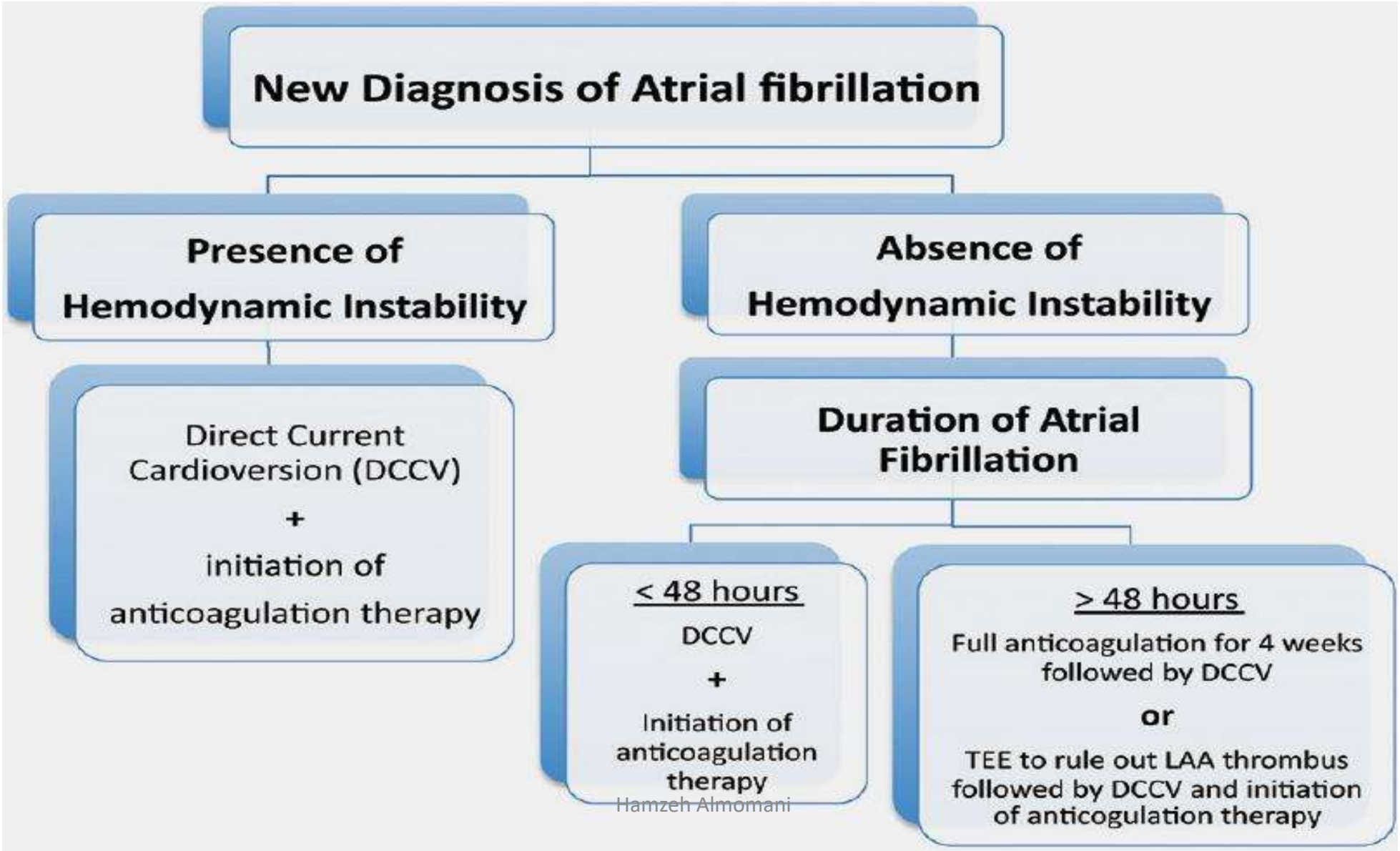
- Rapid and irregular beating of the atria (Characterized by disorganized atrial electrical activity and contraction.)
- Most common in structurally abnormal hearts, prior cardiac surgery,…….etc.
- Structurally normal hearts + association with accessory pathway conduction -----sudden death.
- Seen in myocarditis, pericarditis, hyperthyroid, genetic causes, others
- Presented with palpitation ,SOB, fainting, or chest pain

- **Irregularly irregular rhythm.**
- **No P waves.**
- **Variable ventricular rate.**
- **Narrow QRS complexes unless pre-existing bundle branch block, accessory pathway, or rate related aberrant conduction.**
- **Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude >0.5mm).**
- **Fibrillatory waves may mimic P waves leading to misdiagnosis.**



Acute management

Depends mainly on hemodynamic status and duration of symptoms



FURTHER MANAGEMENT

- Labs – CBC, chemistry, thyroid, toxicology
- Further testing if cardiomyopathy is considered – viral panel, enzymes.
- Echocardiogram
- Admission for observation /treatment
- Anticoagulation in most cases

- The ultimate goal of antiarrhythmic drug therapy:
  - Restore normal sinus rhythm and conduction
  - Prevent more serious and possibly lethal arrhythmias from occurring.
- Antiarrhythmic drugs are used to:

✓ decrease conduction velocity

✓ change the duration of the effective refractory period (ERP)

✓ suppress abnormal automaticity

Antyarrhythmic drugs

•Most antiarrhythmic drugs are pro-arrhythmic (promote arrhythmia)
•They are classified according to ***Vaughan William*** into four classes according to their effects on the cardiac action potential

class		mechanism	action	ECG QT	Conduction velocity	Refractory period	notes
IA	Quinidine *Procainamide*	Na^+ channel blocker	Change the slope of phase 0	++	↓	↑	Can abolish tachyarrhythmia caused by reentry circuit
IB	lidocaine mexiletine tocainide			0	no	↓	
IC	flecainide propafenone			+	↓	no	

Antyarrhythmic drugs

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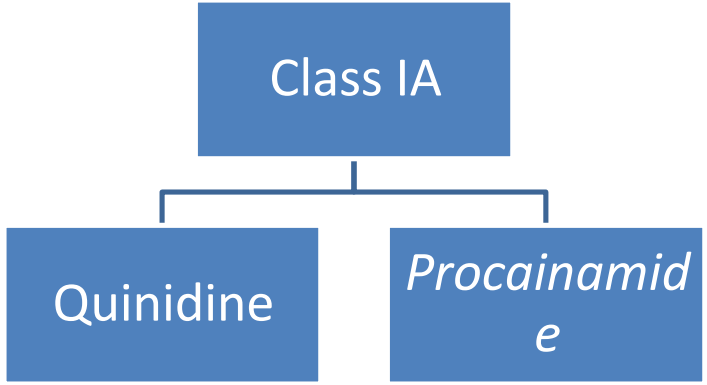
class		mechanism	action	ECG QT	Conduction velocity	Refractory period	notes
II		β blocker	↓heart rate and conduction velocity	0	↓In SAN and AVN	↑ in SAN and AVN	Can indirectly alter K and Ca conductance
III		K^+ channel blocker	1. ↑action potential duration (APD) or effective refractory period (ERP). 2. Delay repolarization.	++	No	↑	Inhibit reentry tachycardia
IV	verapamil & diltiazem	Ca^{++} channel blocker	Slowing the rate of rise in phase 4 of SA node	0	↓ in SAN and AVN	↑ in SAN and AVN	↓conduction velocity in SA and AV node

Class IA Drugs

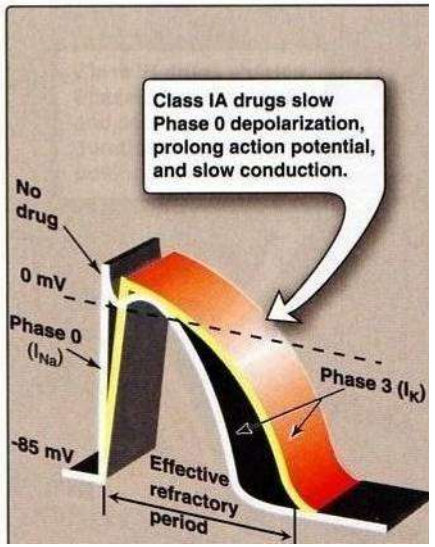


Slowing of the rate of rise in phase 0 → ↓conduction velocity

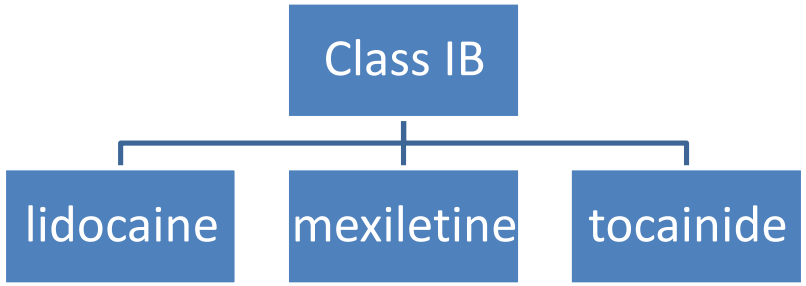
↓of V_{max} of the cardiac action potential

They prolong muscle action potential & ventricular (ERP)

They ↓ the slope of Phase 4 spontaneous depolarization (SA node) → decrease enhanced normal automaticity



Class IB Drugs



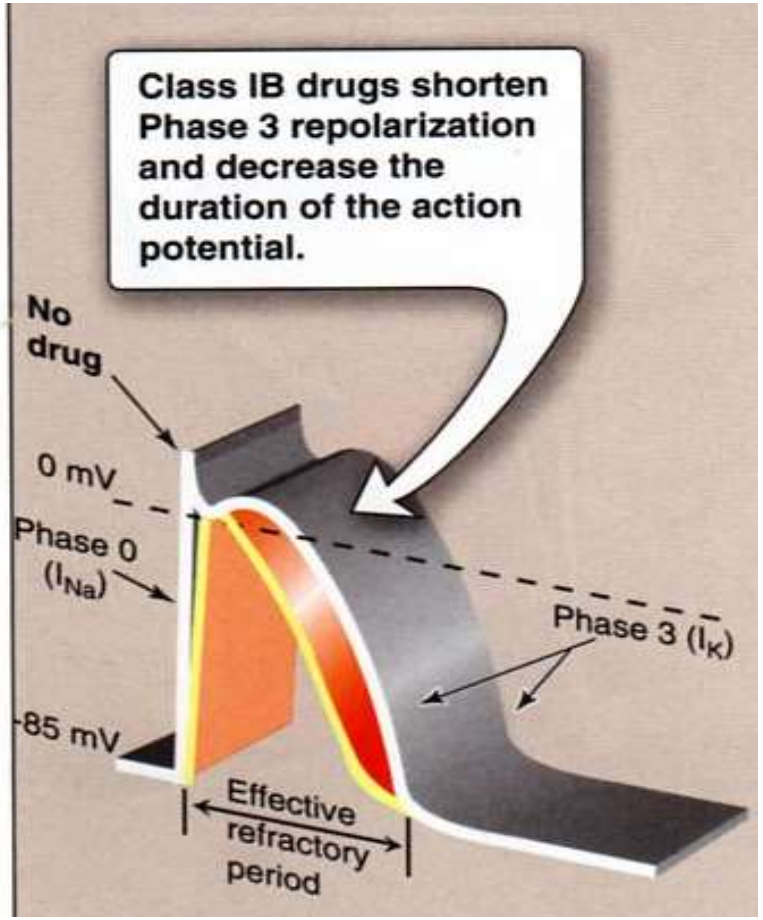
They shorten Phase 3 repolarization

↓ the duration of the cardiac action potential

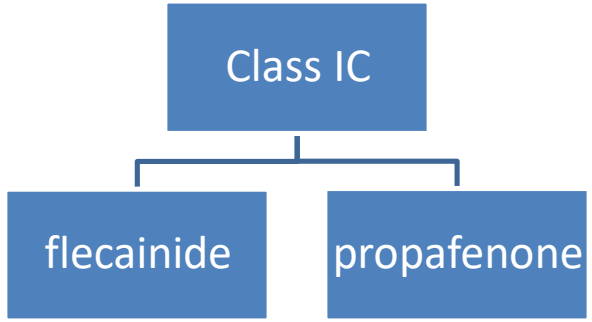
They suppress arrhythmias caused by abnormal automaticity

They show ***rapid association & dissociation*** (weak effect) with Na^+ channels with appreciable degree of use-dependence

No effect on conduction velocity



Class IC Drugs



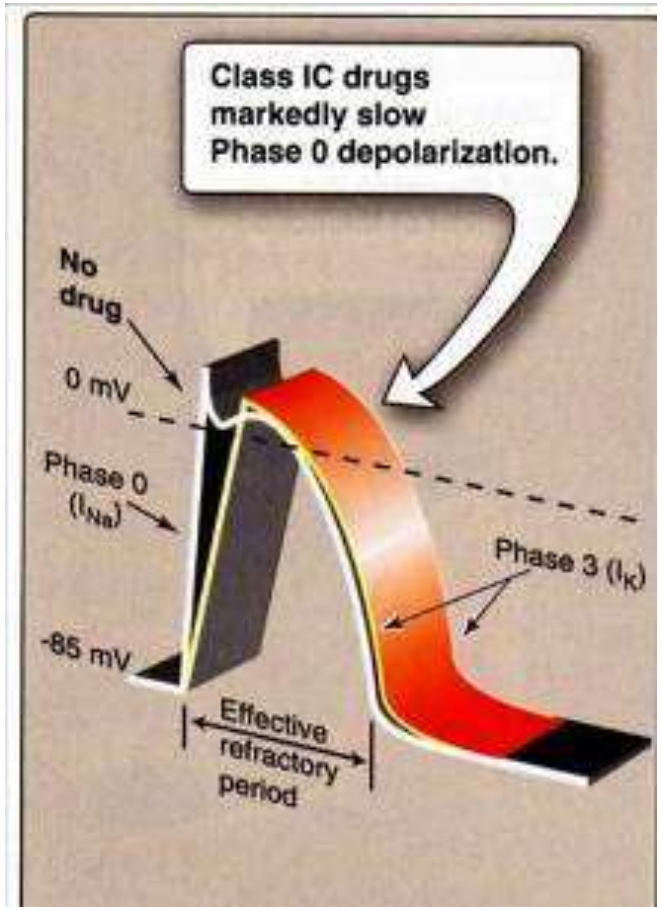
They ***markedly slow Phase 0*** fast depolarization

They markedly slow conduction in the myocardial tissue

They possess ***slow rate of association and dissociation (strong effect)*** with sodium channels

They only have **minor effects on the duration of action potential and refractoriness**

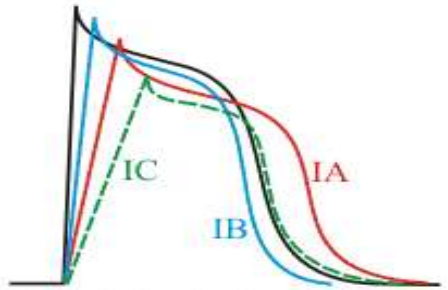
They reduce automaticity by increasing the threshold potential rather than decreasing the slope of Phase 4 spontaneous depolarization.



Compare between class IA, IB, and IC drugs as regards effect on Na^+ channel & ERP

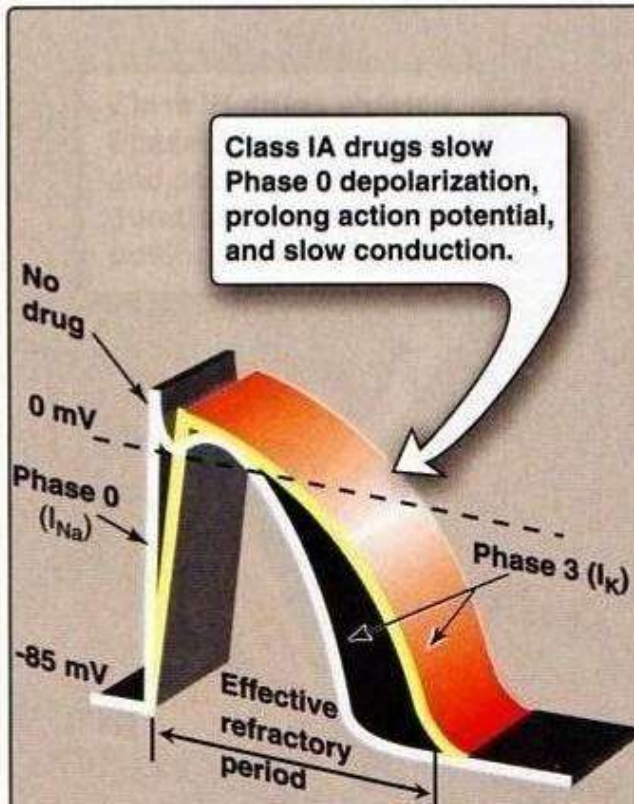
- **Sodium channel blockade:**
  IC > IA > IB
- **Increasing the ERP:**
  IA>IC>IB (lowered)

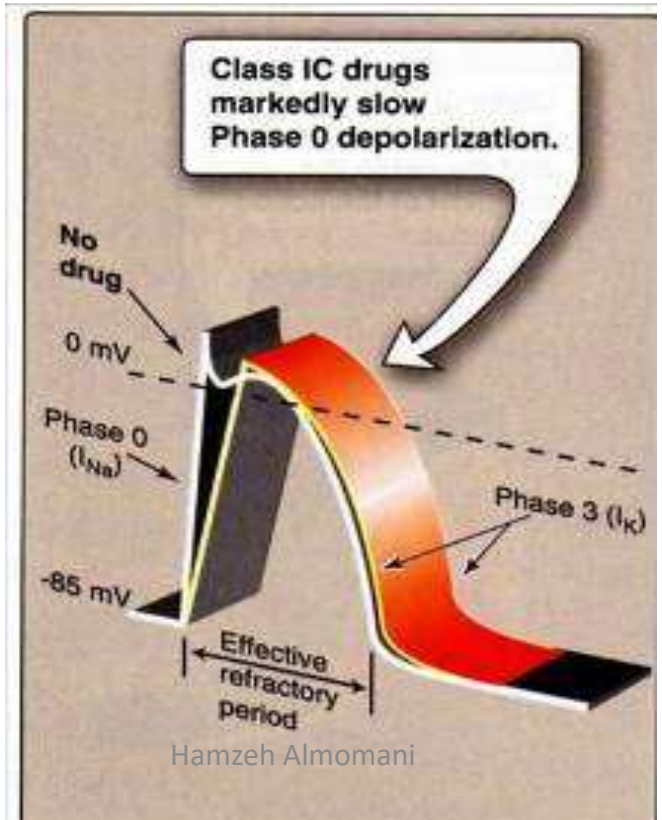
Because of K^+ blockade

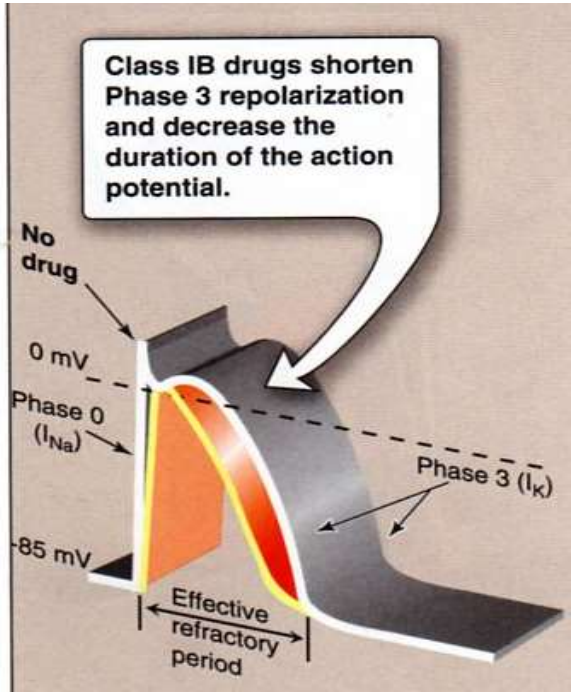


Ventricular Action Potential

- Class IA: e.g., quinidine
  - Moderate Na^+-channel blockade
  - ↑ ERP
- Class IB: e.g., lidocaine
  - Weak Na^+-channel blockade
  - ↓ ERP
- Class IC: e.g., flecainide
  - Strong Na^+-channel blockade
  - → ERP





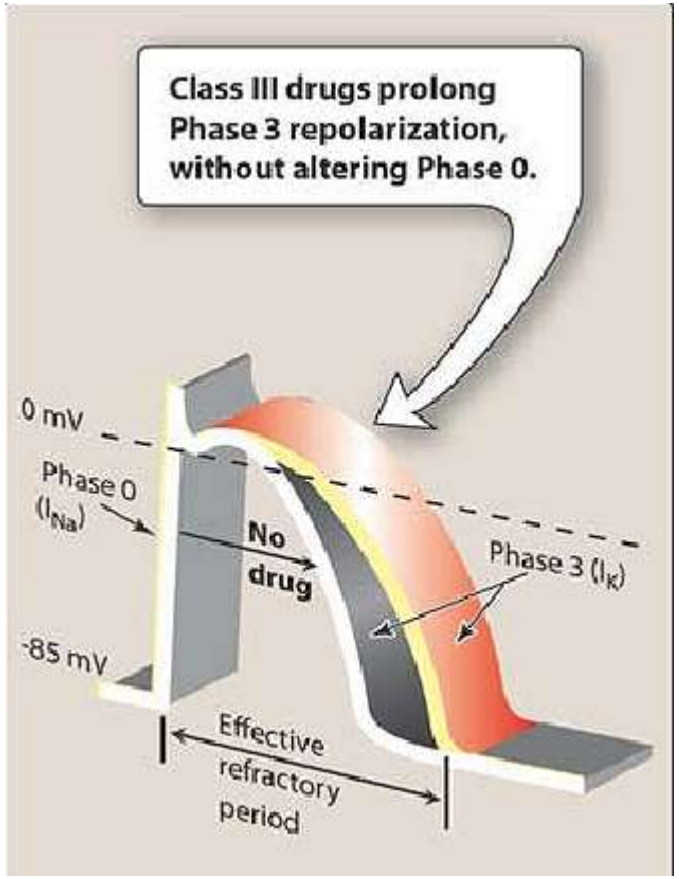


Class III ANTIARRHYTHMIC DRUGS
K^+ blockers

Prolongation of phase 3 repolarization without altering phase 0 upstroke or the resting membrane potential

They prolong both the duration of the action potential and ERP

Their mechanism of action is still not clear but it is thought that they block potassium channels



Class IV ANTIARRHYTHMIC DRUGS (Calcium Channel Blockers)

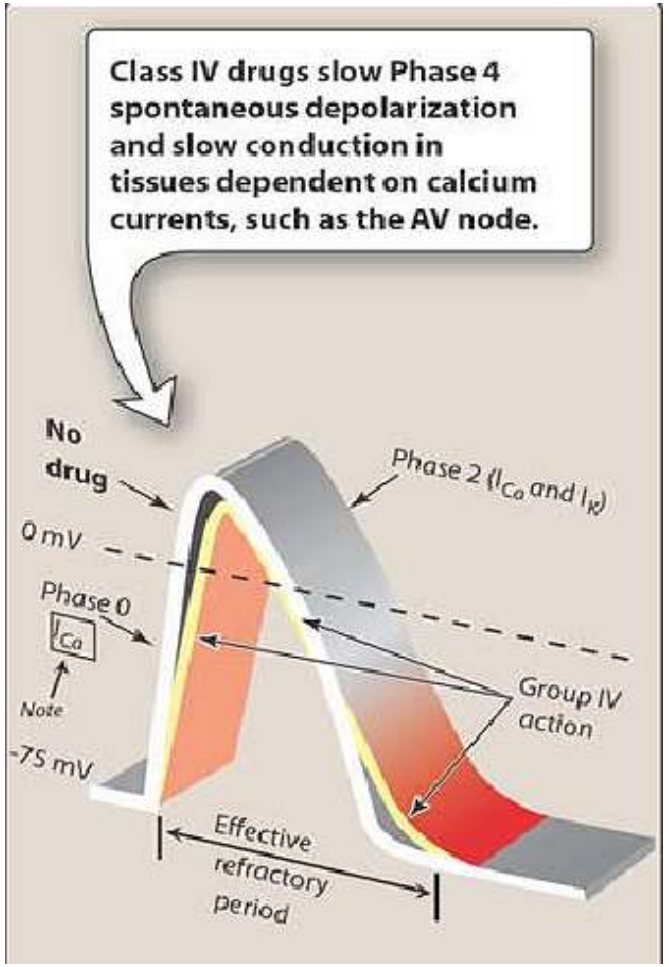
Calcium channel blockers decrease inward Ca^{2+} currents resulting in a decrease of phase 4 spontaneous depolarization (SA node)

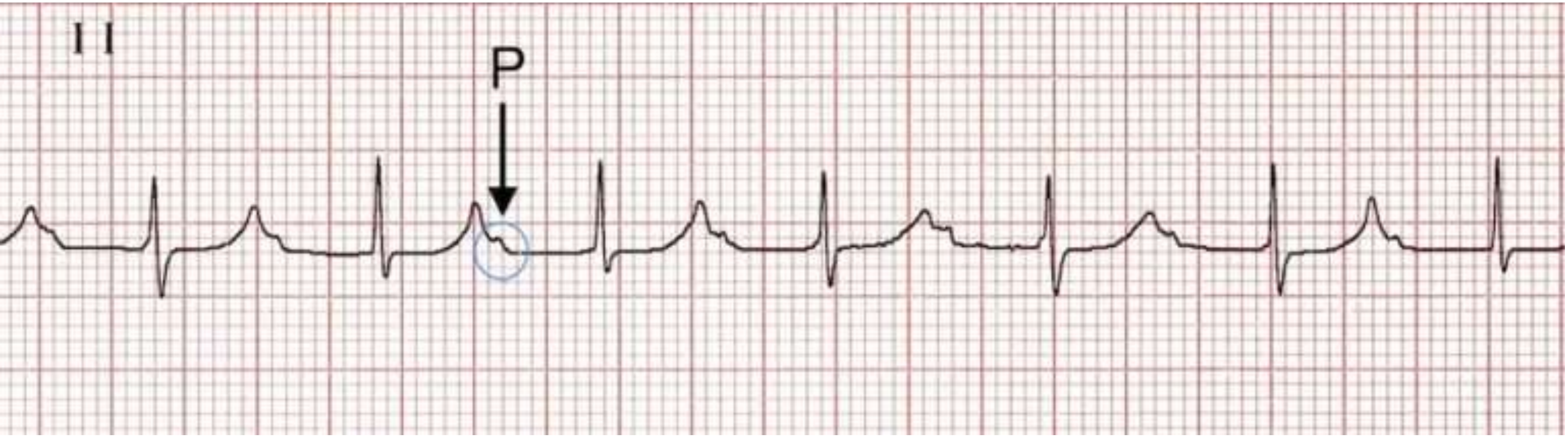
They slow conductance in Ca^{2+} current-dependent tissues like AV node.

Examples: verapamil & diltiazem

Because they act on the heart only and not on blood vessels.

Dihydropyridine family are not used because they only act on blood vessels




II
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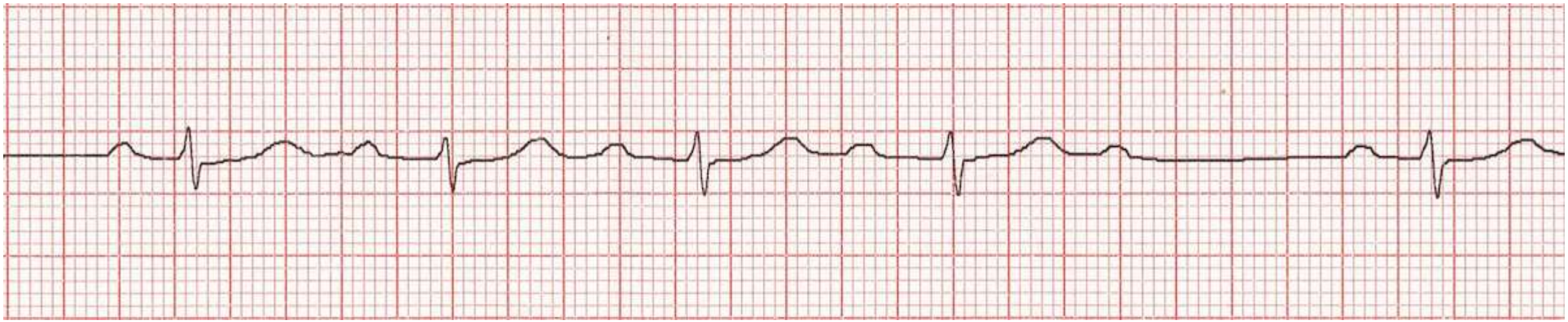
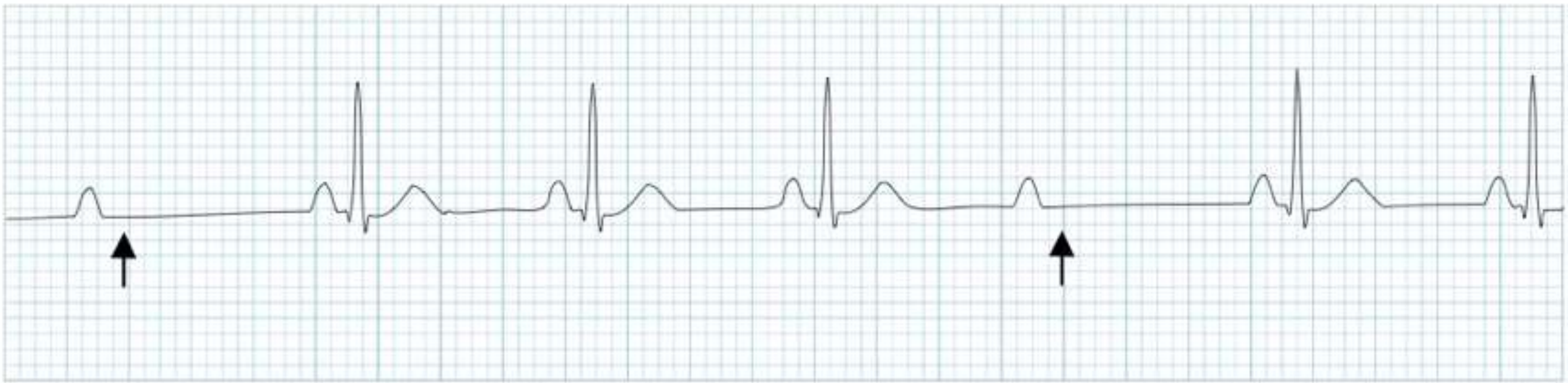
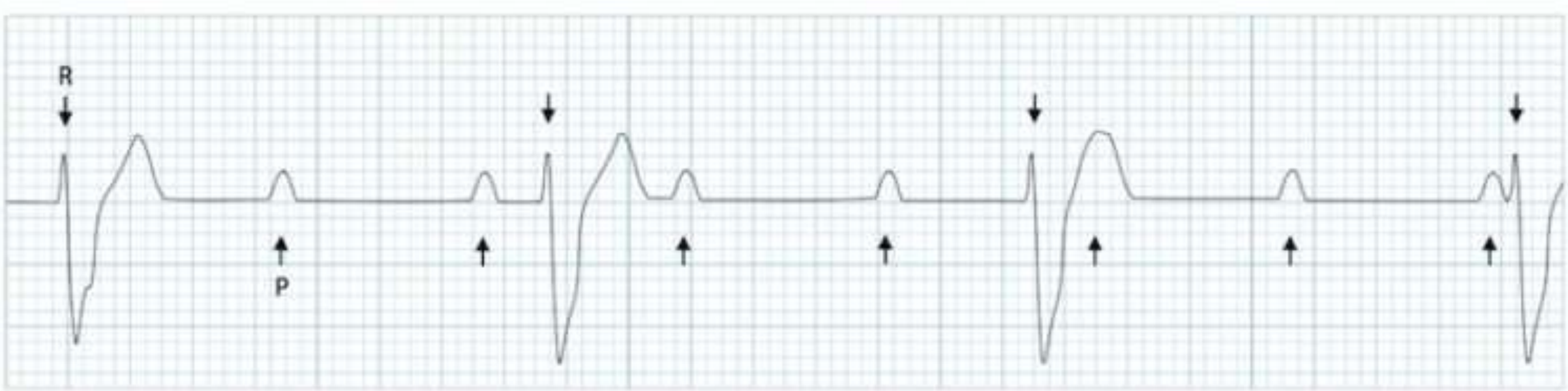
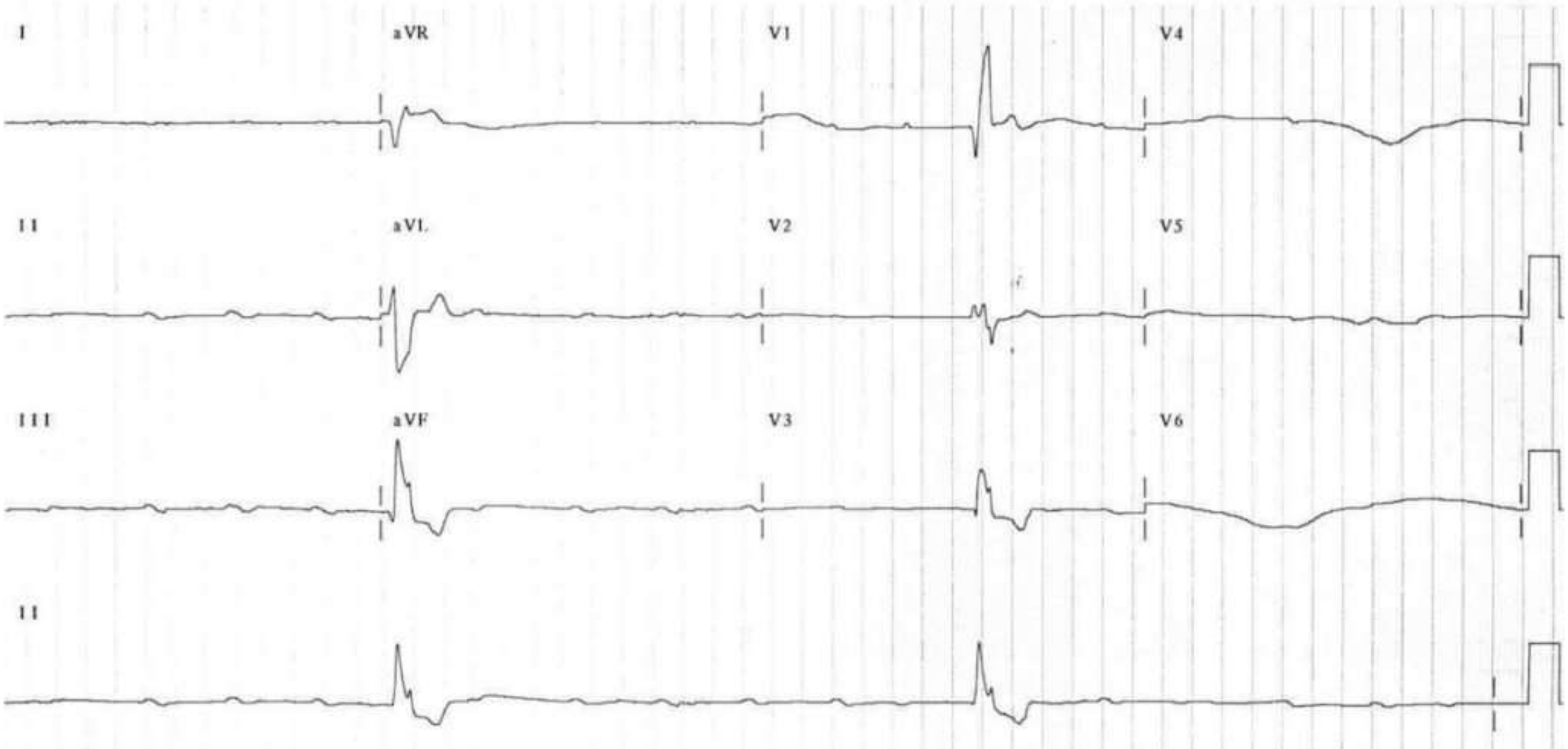





R
P


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