CARDIAC ARRHYHTHMIAS

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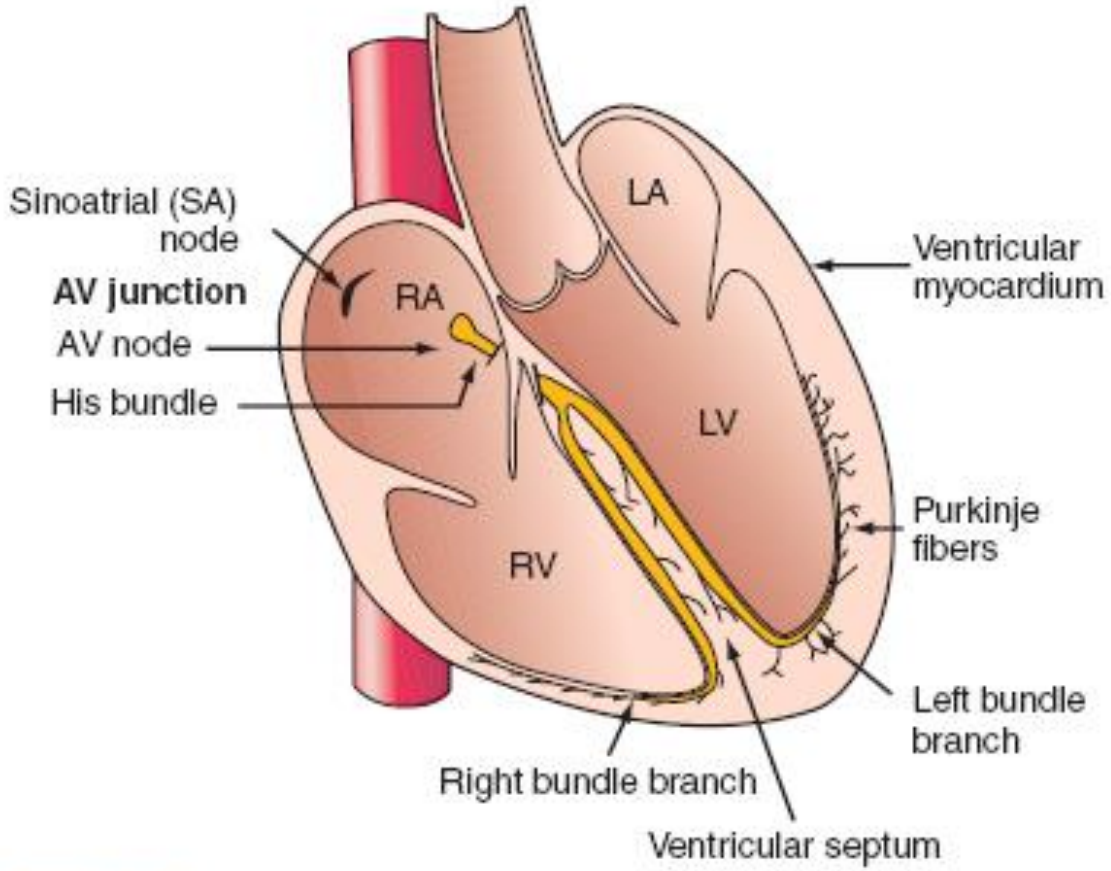
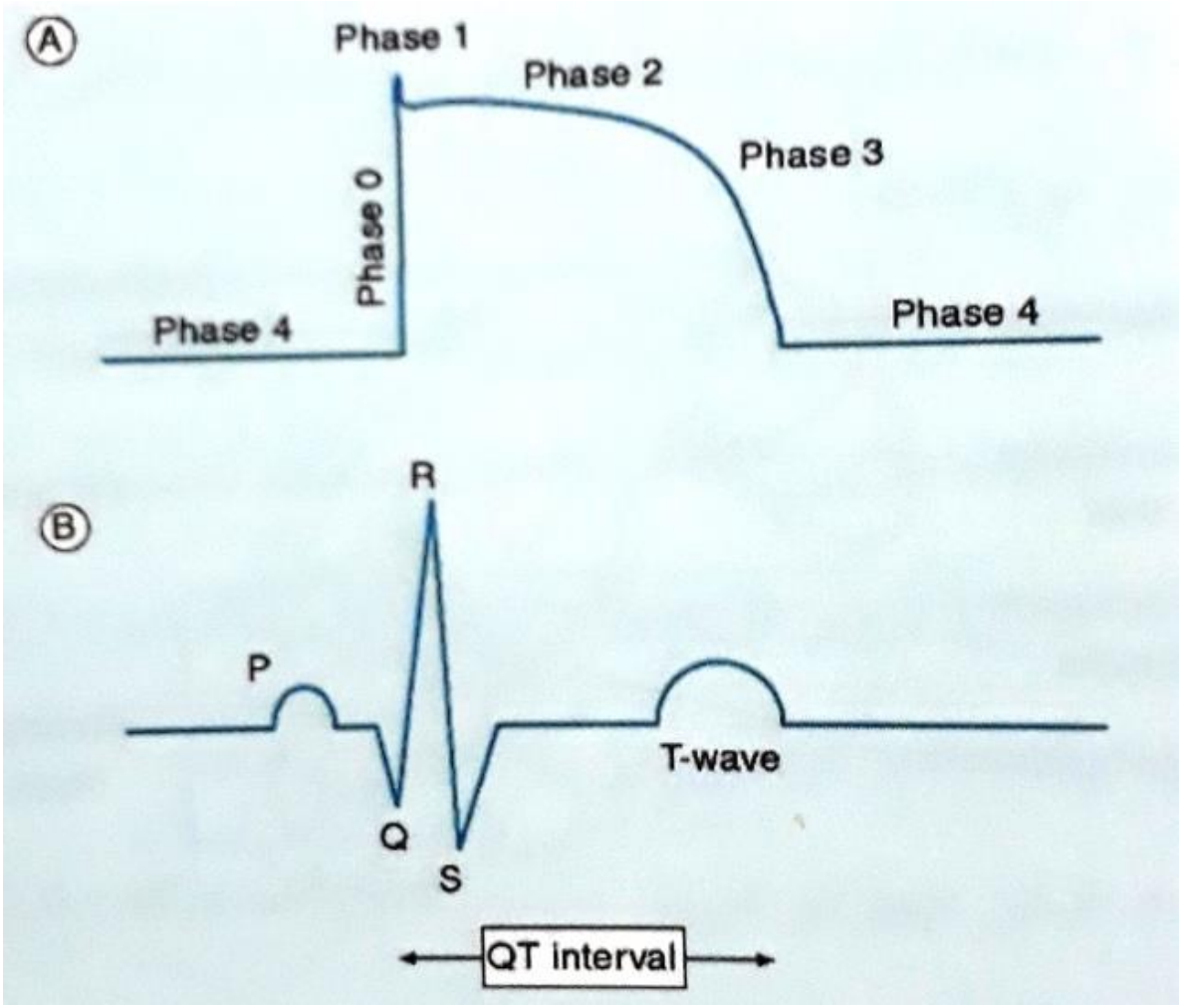


FIGURE 268-1 Schematic of the cardiac conduction system.

Normal Physiology

- Impulses originate regularly at a frequency of **60-100 beat/ min**

- **SA node – AV node – Bundle of His – Purkinje fibres**


A
Phase 1
Phase 2
Phase 3
Phase 0
Phase 4
Phase 4
B
R
P
T-wave
Q
S
QT interval

Cardiac Arrhythmias

- An abnormality of the cardiac rhythm is called a cardiac arrhythmia.
- Arrhythmias may cause sudden death, syncope, heart failure, dizziness, palpitations or no symptoms at all.
- There are two main types of arrhythmia:

bradycardia: the heart rate is slow (< 60 b.p.m).

tachycardia: the heart rate is fast (> 100 b.p.m).

Bradyarrhythmias

- Heart rate < 60 beats per minute
- Sinus or non-sinus
- Physiologic or pathologic

Physiologic Bradyarrhythmias

- Sinus bradycardia
  - Normal HR for Physiologic status
  - Sleeping, increased vagus tone, reduced sympathetic
  - In athletes at rest

- Sinus arrhythmia
  - Rspiratory variation in p-p intervals less than 120ms
  - Commonly in children
  - Decreasing by age
  - Disappears by holding the breath for 15-20 seconds

Pathologic Sinus Bradyarrhythmias

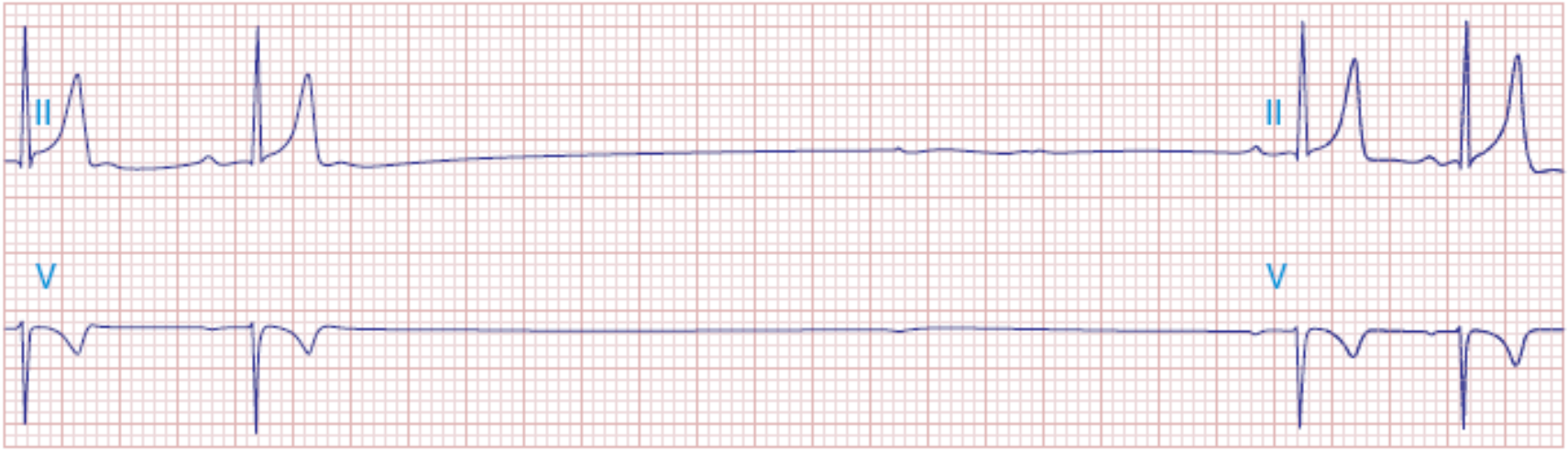
- Pathologic Sinus Bradycardia
- Sinus Pause or Sinus Arrest
- bradycardia-tachycardia syndrome
- *chronotropic incompetency*

Pathologic Sinus Bradycardia

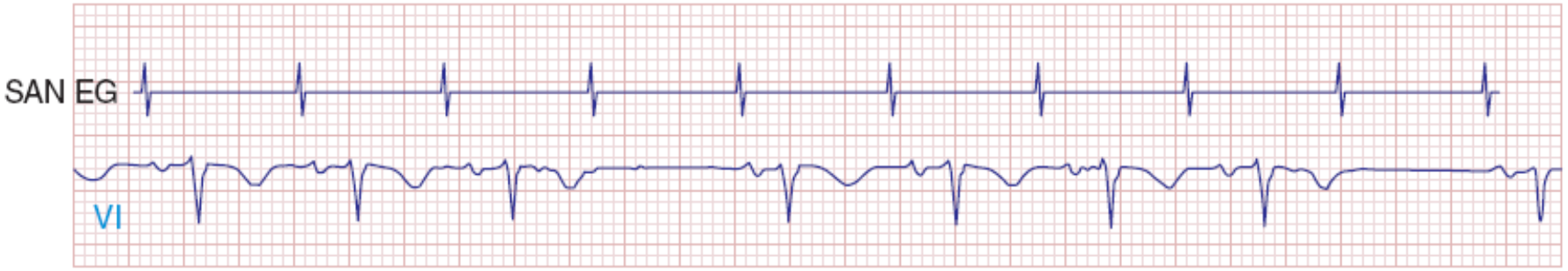
- Inappropriate HR for condition

Sinus Pause or Sinus Arrest

- Sinus Pause the P-P interval does not equal a multiple of the basic P-P interval.
- slowing or cessation of spontaneous sinus node automaticity and therefore a disorder of impulse formation
- Sinus Arrest equals a multiple of the basic P-P interval



Sinus slowing and pauses on the electrocardiogram (ECG)

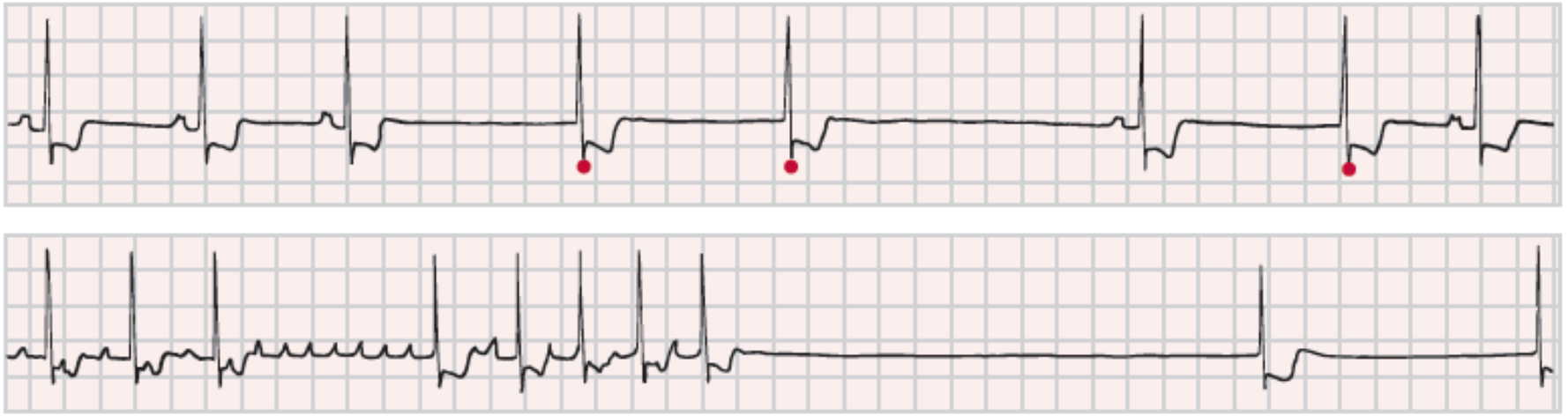


Mobitz type I SA nodal exit block

- Differentiation of sinus arrest, which is thought to be caused by slowing or cessation of spontaneous sinus node automaticity and therefore a disorder of impulse formation, from sinoatrial (SA) exit block in patients with sinus arrhythmia can be difficult without direct recordings of sinus node discharge.

Tachycardia- bradycardia syndrome (TBS)

- TBS occurs when a patient has tachyarrhythmias and bradyarrhythmias closely associated in time.
- That can occur when a tachyarrhythmia, typically atrial fibrillation or atrial flutter terminates, with a resultant excessive post- conversion pause .
- TBS can also occur during AF when periods of AF with rapid ventricular rates alternate with periods of excessive bradycardia (due to high- grade AV block) during AF.
- without medication as a result of treatment with beta blockers or calcium channel blockers.



Sick sinus syndrome with bradycardia-tachycardia

chronotropic incompetency

- Chronotropic incompetence (CI) is diagnosed when the heart rate does not increase appropriately in the setting of increased physiologic demand.

Atrioventricular Block

- First-degree atrioventricular (AV) block

- Second-degree atrioventricular (AV) block
  - Mobitz type I (Wenckebach)
  - Mobitz type II

- Third-degree atrioventricular (AV) block

First-degree atrioventricular (AV) block

- PR interval > 120ms
- 1 sinus P for 1 QRS
- conduction delay in the AV node
- Sometimes below AVN (wide QRS)

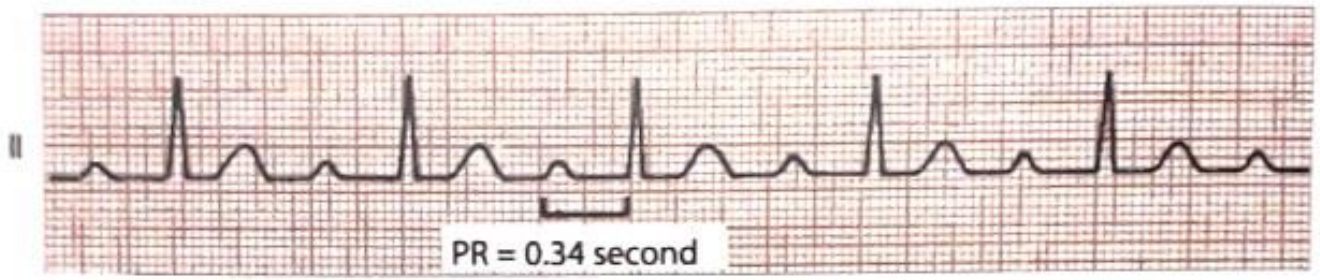
- No treatment needed, remove reversible causes

Second-degree atrioventricular (AV) block

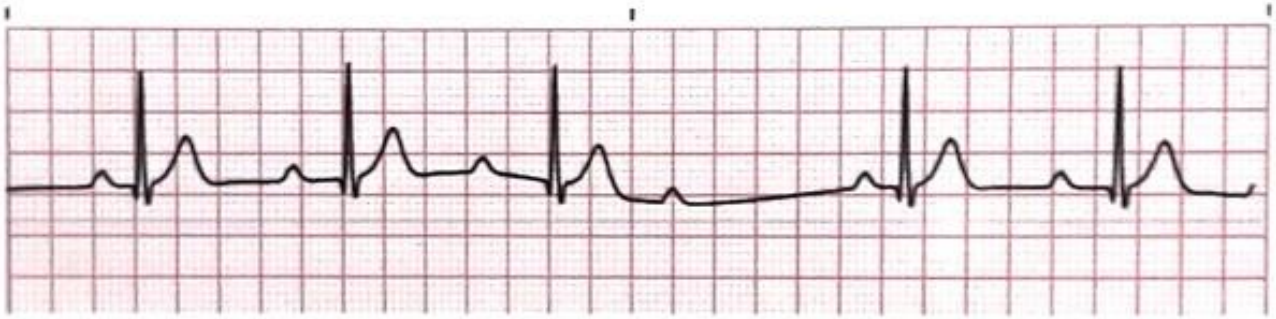
- **Type I** second degree AV block is characterized by progressive PR prolongation culminating in a nonconducted P wave
- **Type** II second- degree AV block, the PR interval remains constant before the blocked P wave

Third-degree atrioventricular (AV) block

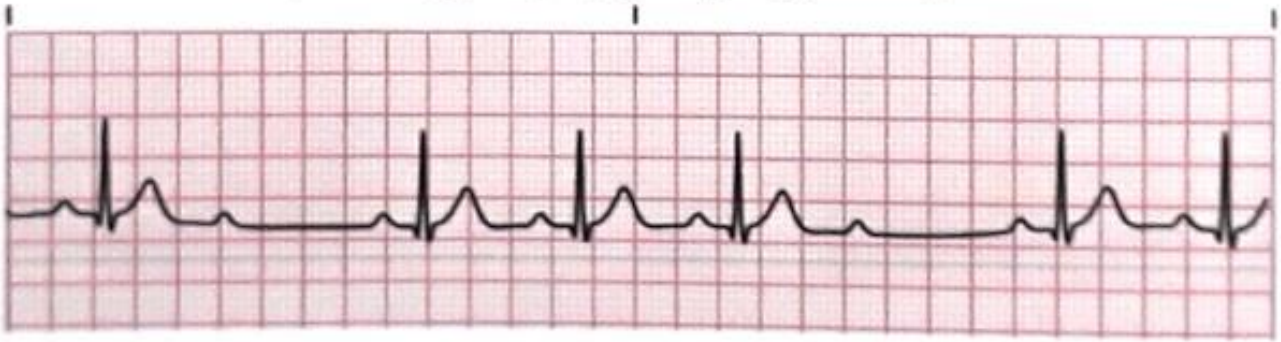
- Third- degree or complete AV block occurs when no atrial activity is conducted to the ventricles and therefore the atria and ventricles are controlled by independent pacemakers.
- Thus, complete AV block is one type of complete AV dissociation.



شکل ۱۷-۱۱: بلوک درجه ۱ گره AV

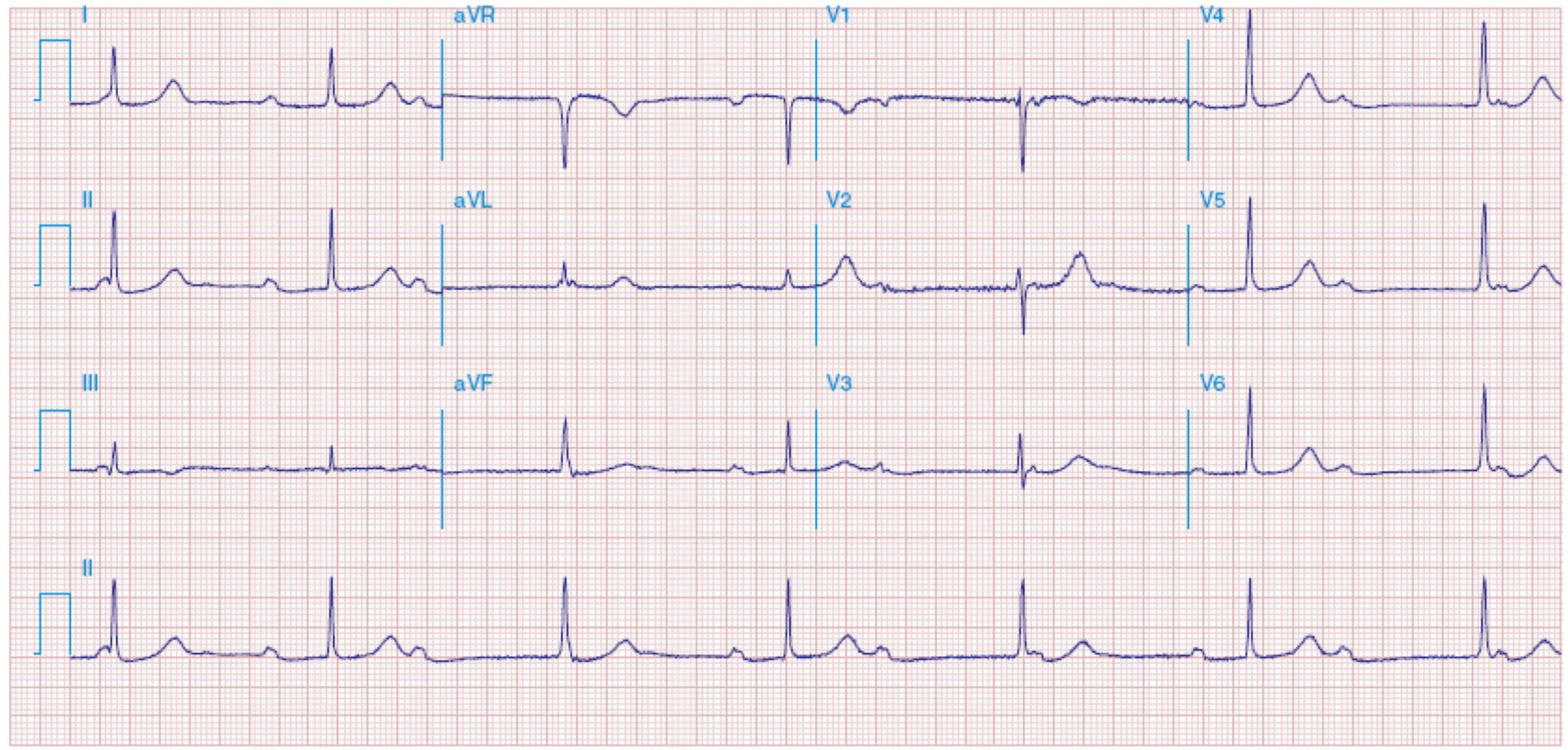


شکل ۱۸-۱۱: بلوک درجه ۲ نوع یک (ونکه باخ-Mobit2 I)



شکل ۱۹-۱۱: بلوک درجه ۲ نوع ۲ (Mobit2 II)

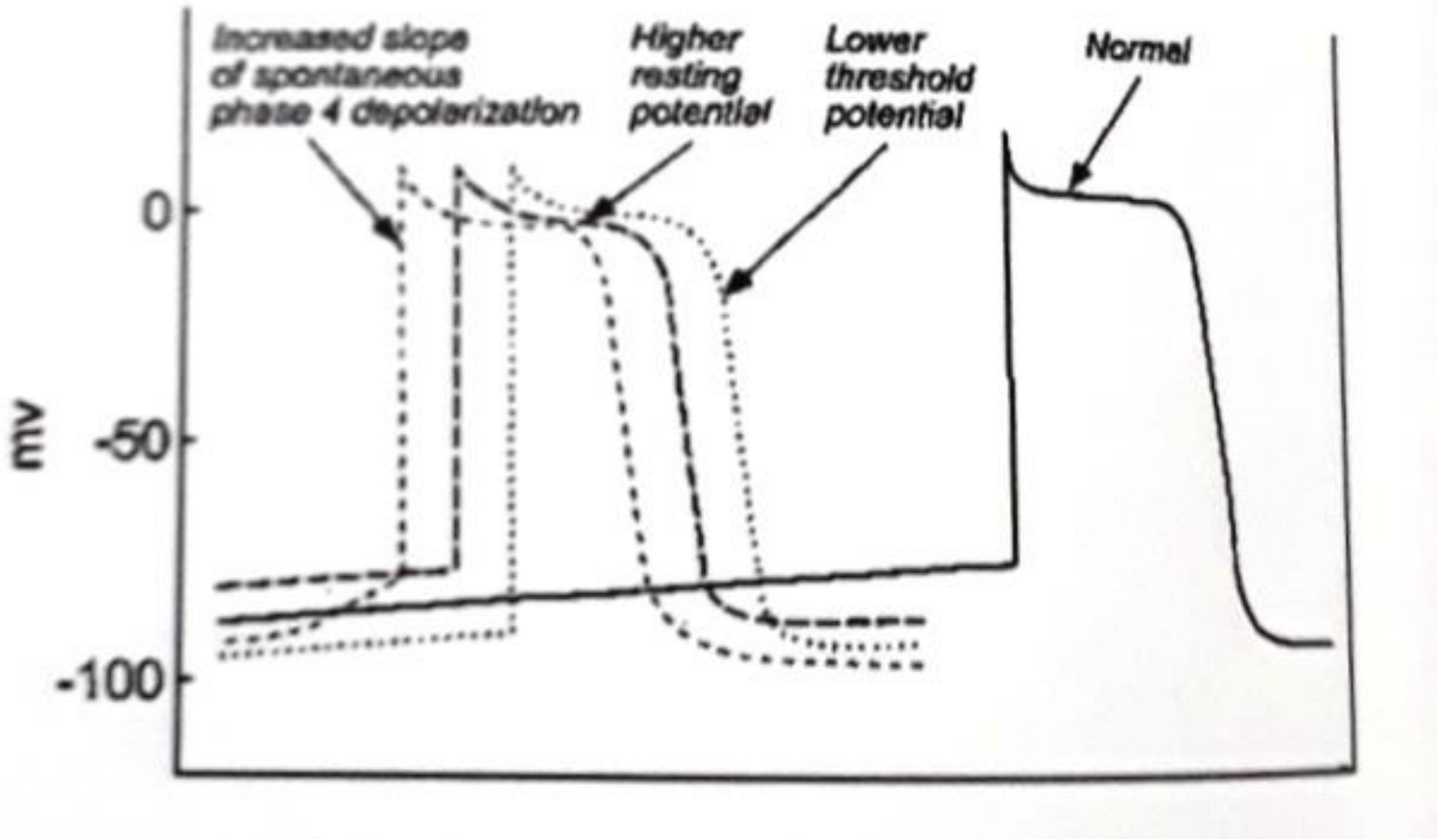
Complete Heart Block



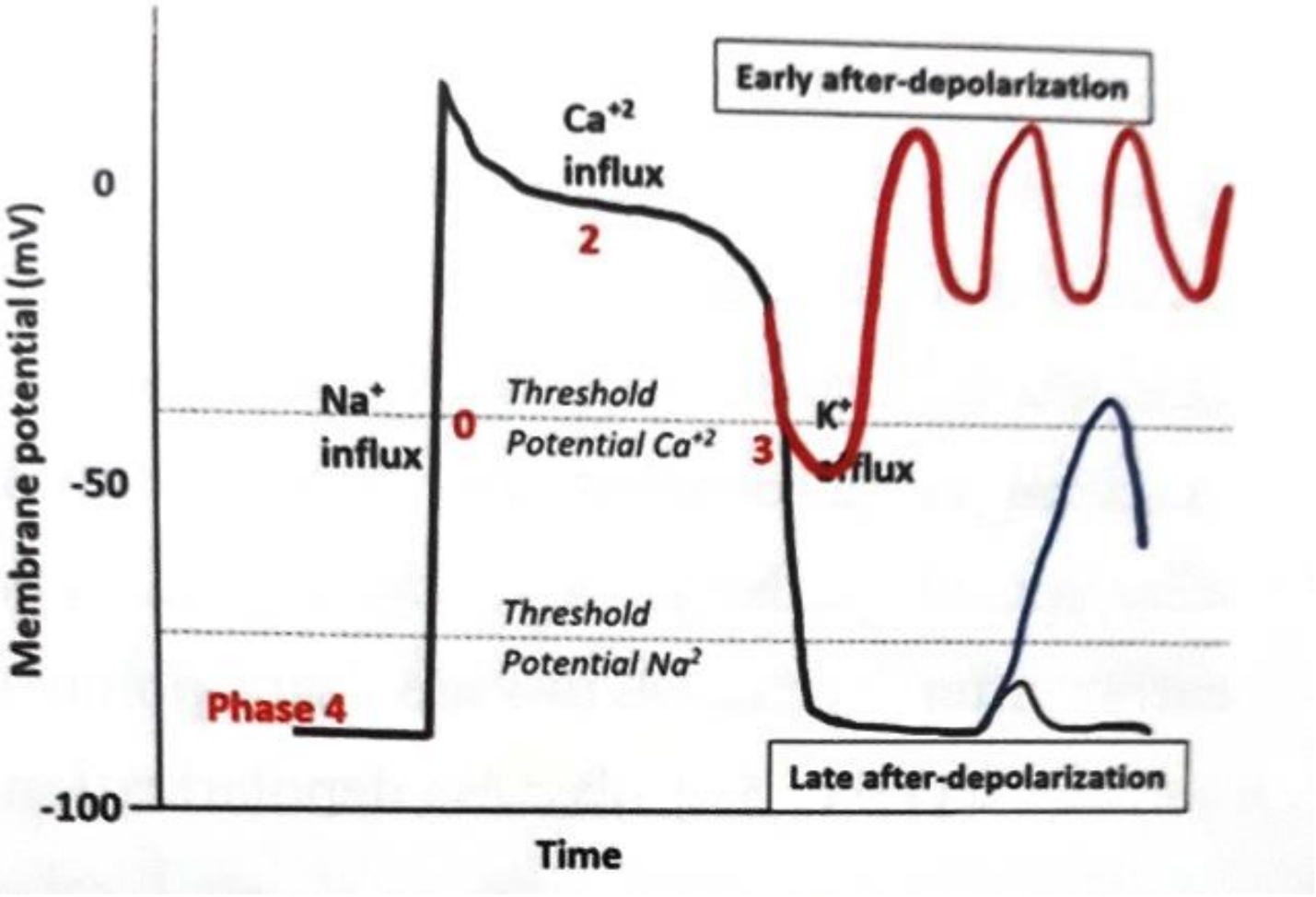
Mechanisms of tachyarrhythmias

- Enhanced Automaticity
- Triggered activity
- Reentry

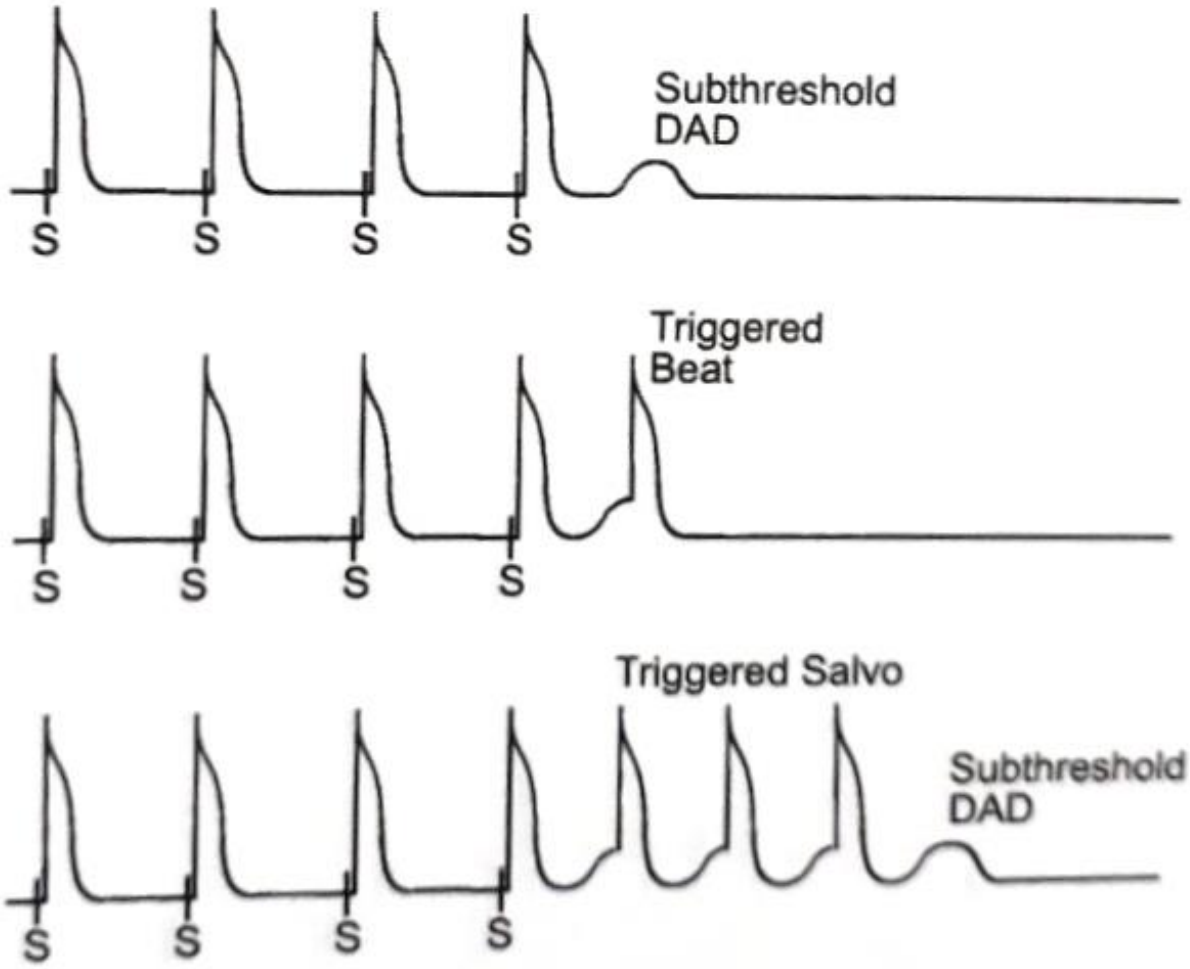
Automaticity



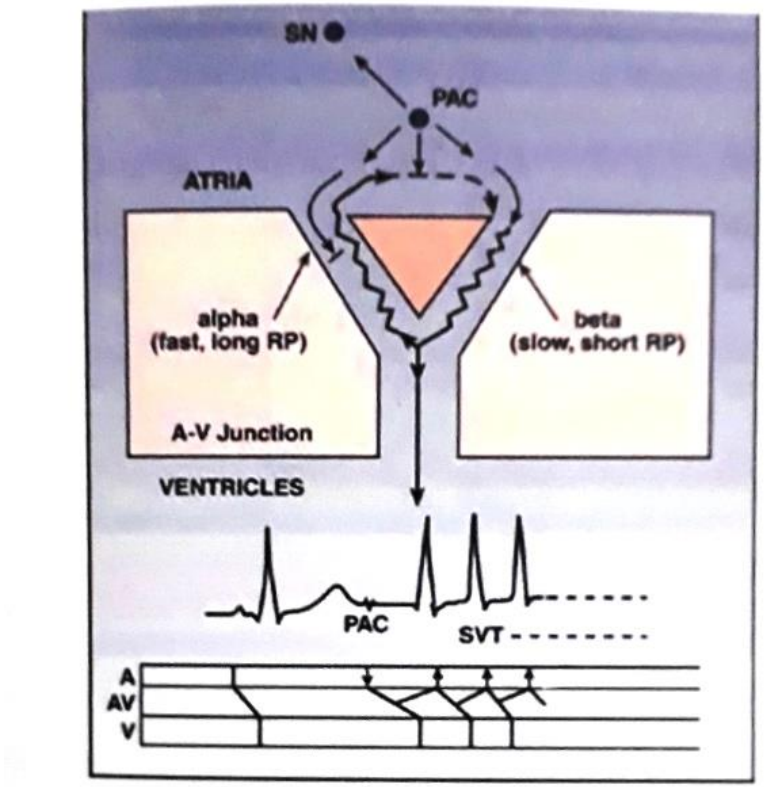
EAD, LAD

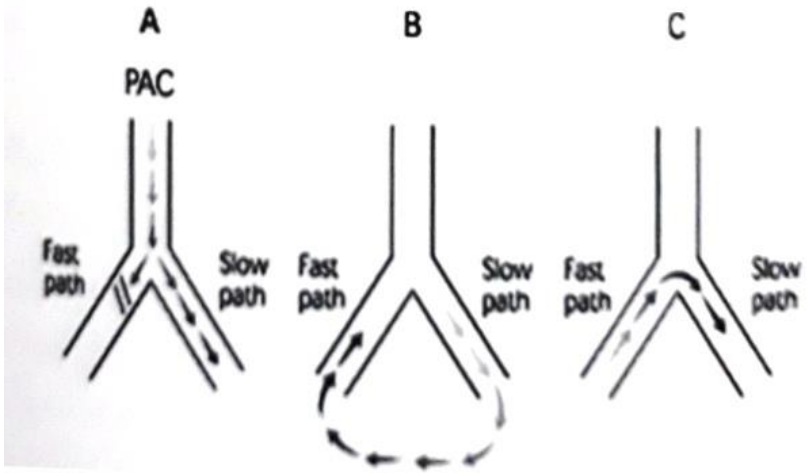

Early after-depolarization
Ca+2 influx
2
0
Membrane potential (mV)
-50
-100
Na+ influx
0
Threshold Potential Ca+2
3
K+ influx
Threshold Potential Na2
Phase 4
Late after-depolarization
Time

Triggered activity



Reentry


SN
PAC
ATRIA
alpha
(fast, long RP)
beta
(slow, short RP)
A-V Junction
VENTRICLES
PAC
SVT
A
AV
V


A
B
C
PAC
Fast path
Slow path
Fast path
Slow path
Fast path
Slow path

Reentry

1. At least 2 parallel pathways
2. Different refractory period
3. The pathway with shorter refractory period has slower conductionvelocity

Tachyarrhythmias

- Supraventricular arrhythmias
- Ventricular arrhythmias

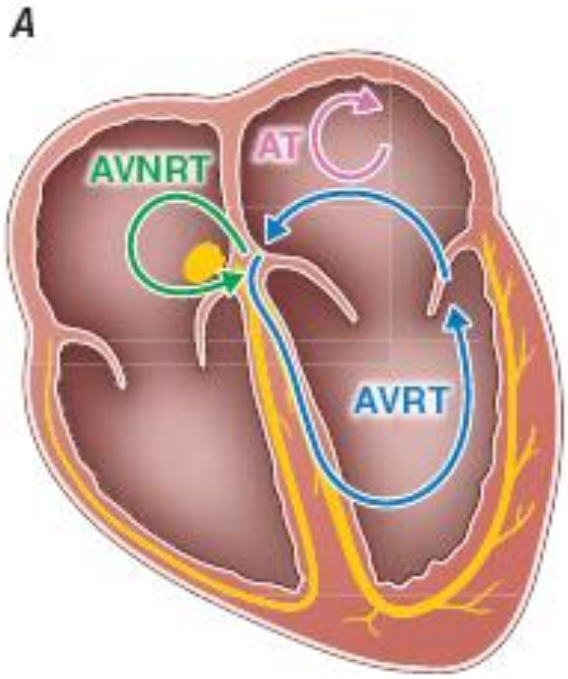
Supraventricular arrhythmias

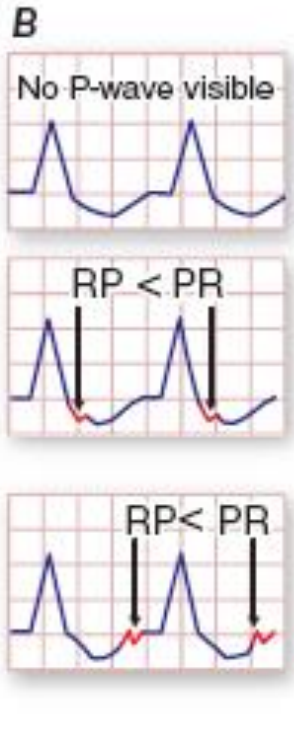
- Sinus tachycardia
- Atrioventricular nodal reentrant tachycardia(AVNRT)
- Inappropriate sinus tachycardia (IST)
- Sinoatrial reentrant tachycardia(SANRT)
- Intraatrial reentrant tachycardia(IANRT)
- Atrial tachycardia (including focal and multifocal AT)
- macroreentrant AT (Atrial Flutter)
- Atrial Fibrillation (AF)
- Junctional ectopic tachycardia (JET)
- Non-paroxysmal junctional tachycardia
- and various forms of accessory pathway- mediated reentrant tachycardias (AVRT).

Paroxysmal Supraventricular Tachycardia (PSVT)

- A clinical syndrome characterized by the presence of a regular and rapid tachycardia of abrupt onset and termination.
- These features are characteristic of AVNRT (60%) or AVRT (30%), and, less frequently, AT.
- Reentry mechanism

Paroxysmal Supraventricular Tachycardia (PSVT)





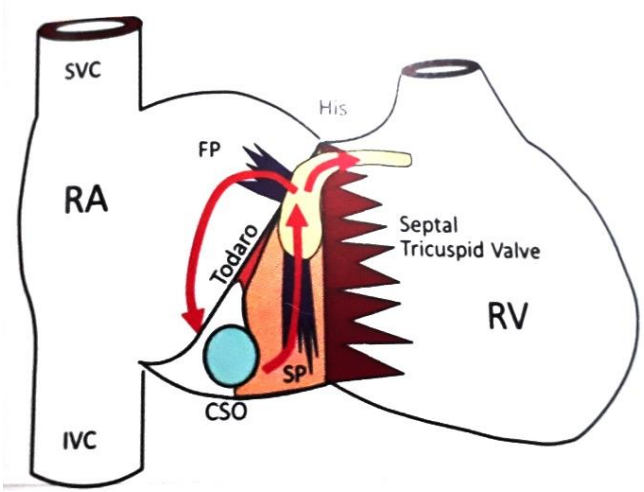
- AV node reentry
- AV node reentry
- AV reentry using an accessory pathway
- Focal atrial tachycardia
- AV reentry using an accessory pathway
- AV node reentry uncommon form

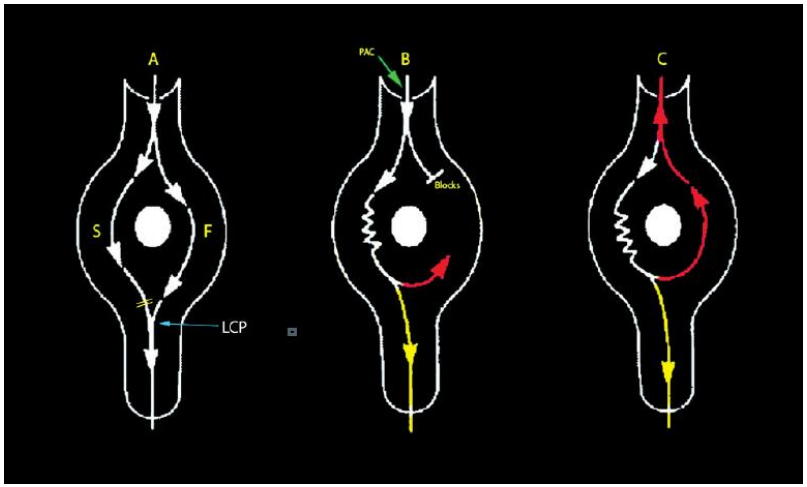
AV nodal reentrant tachycardia(AVNRT)

- Female>male
- Without structural heart disease
- Reentry

AVNRT

- Fast pathway --- long refractory period
- Slow pathway --- short refractory period

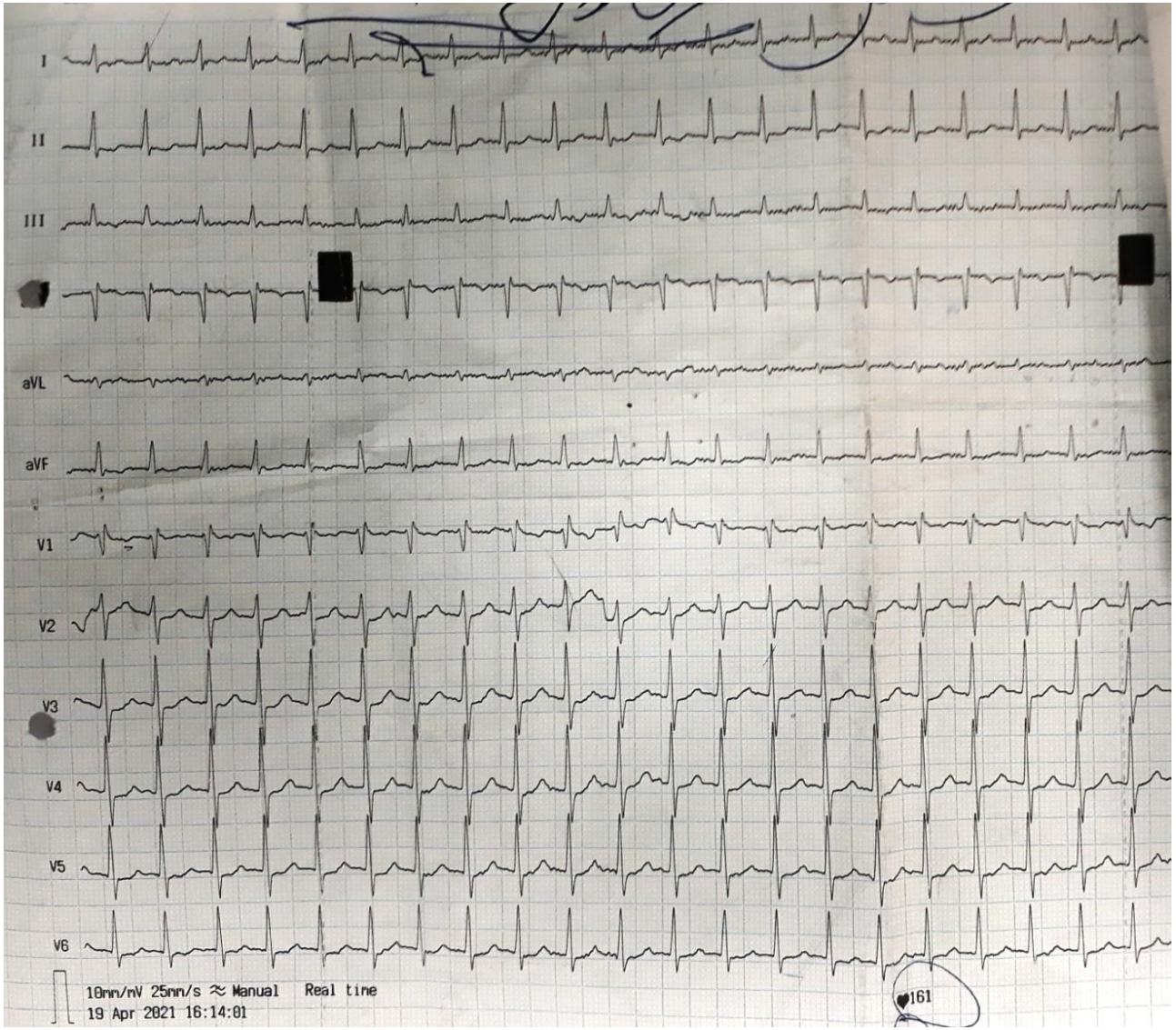




AVNRT

- Normal PR in sinus rhythm
- Narrow QRS tachycardia
- Retrograde P often not visible
- Pseudo S in inferior leads
- Pseudo R in V1

AVNRT


I
II
III
aVL
aVF
V1
V2
V3
V4
V5
V6
10mm/mV 25mm/s ≈ Manual Real time
19 Apr 2021 16:14:01
161

Treatment

- Adenosine
- Beta blockers
- CCB
- Slow pathway ablation

Atrial tachycardia

- HR >100
- Originates from a focus other than SAN
- Focal AT originates from a single focus in RA or LA
- Often paroxysmal and self-limitted
- Sometimes incessant
- Enhanced automaticity, triggerd activity, micro-reentry

Atrial tachycardia

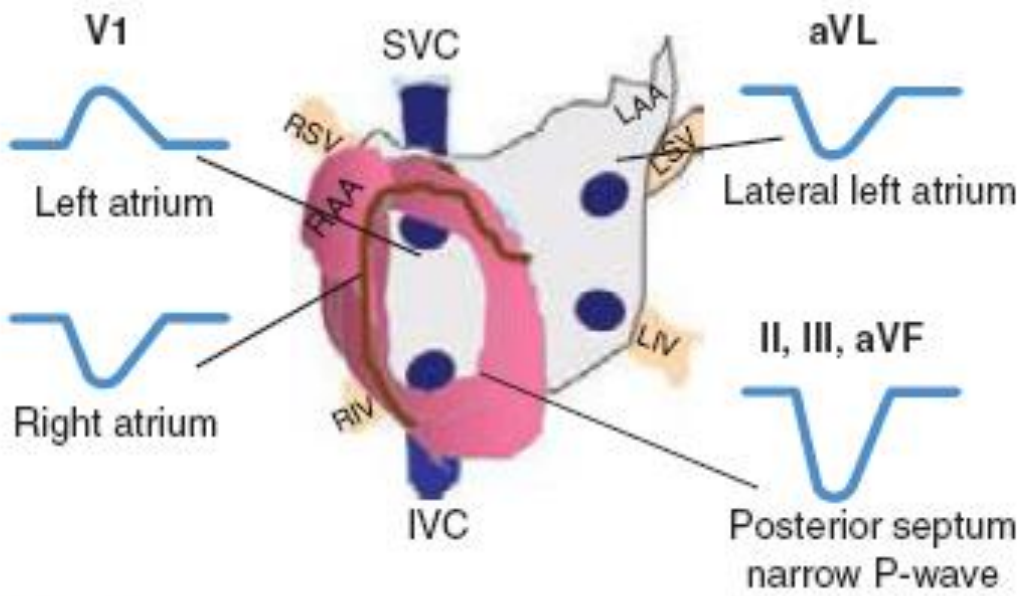
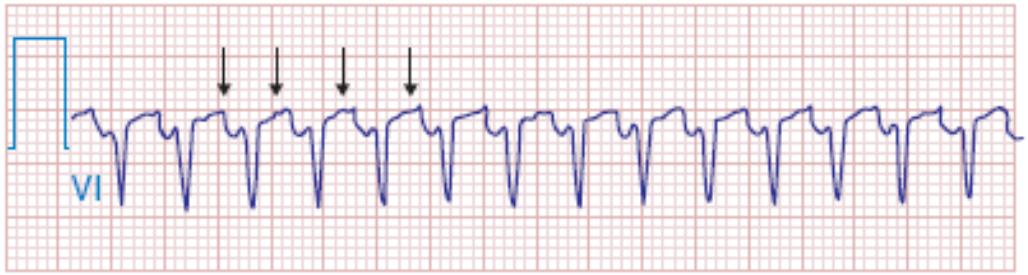
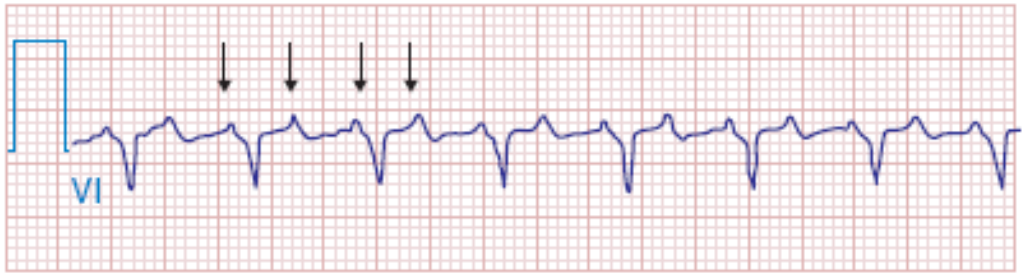


FIGURE 276-3 Location of focal atrial tachycardia focus estimated by P-wave morphology. LAA, left atrial appendage; LIV, left inferior pulmonary vein; LSV, left superior pulmonary vein; RAA, right atrial appendage; RIV, right inferior pulmonary vein; RSV, right superior pulmonary vein; SVC, superior vena cava.

Atrial tachycardia



A



B

FIGURE 276-4 Atrial tachycardia (AT) with 1:1 and 2:1 atrioventricular (AV) conduction. *Arrows* indicate p waves. ***A.*** AT with 1:1 AV relationship and R-P > P-R. ***B.*** Same AT with 2:1 AV relationship after AV nodal–blocking agent administered. *(Adapted from F Marchlinski: The tachyarrhythmias. In Longo DL et al [eds]: Harrison's Principles of Internal Medicine, 18th ed. New York, McGraw-Hill, 2012, pp 1878–1900.)*

Multifocal atrial tachycardia (MAT)

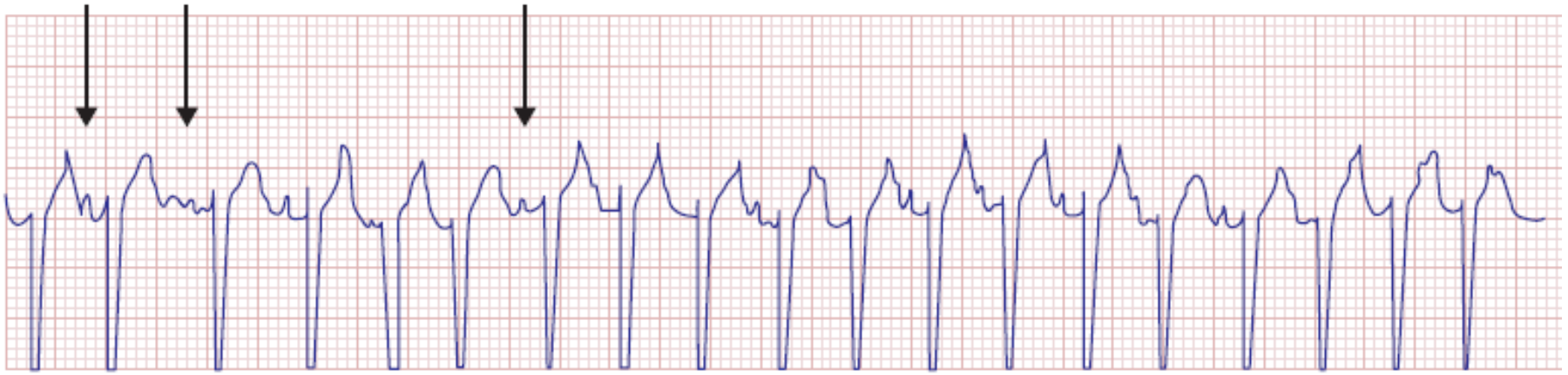


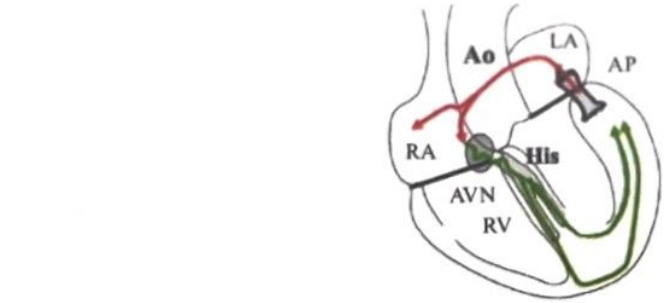
FIGURE 276-11 **Multifocal atrial tachycardia.** Rhythm strip obtained from a patient with severe pulmonary disease during an acute illness

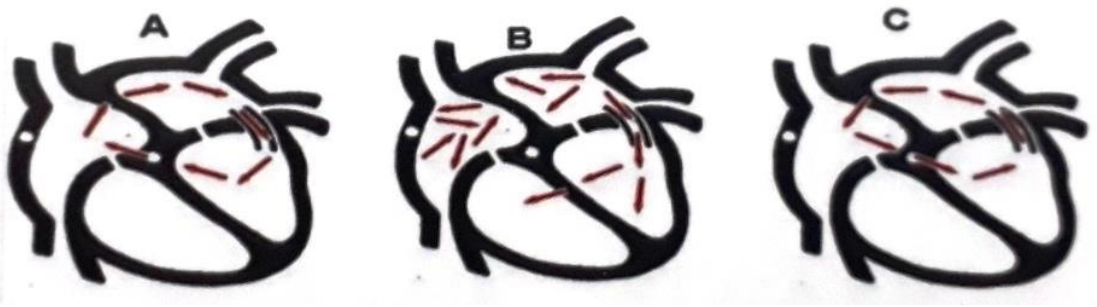
Multifocal atrial tachycardia (MAT)

- At least three distinct P-wave morphologies
- rates typically between 100 and 150 bpm.
- Unlike atrial fibrillation, there are clear isoelectric intervals between P waves
- The mechanism is likely triggered automaticity from multiple atrial foci.
- It is usually encountered in patients with chronic pulmonary disease and acute illness.
- Therapy for MAT is directed at treating the underlying disease and correcting any metabolic abnormalities.

WPW Syndrome

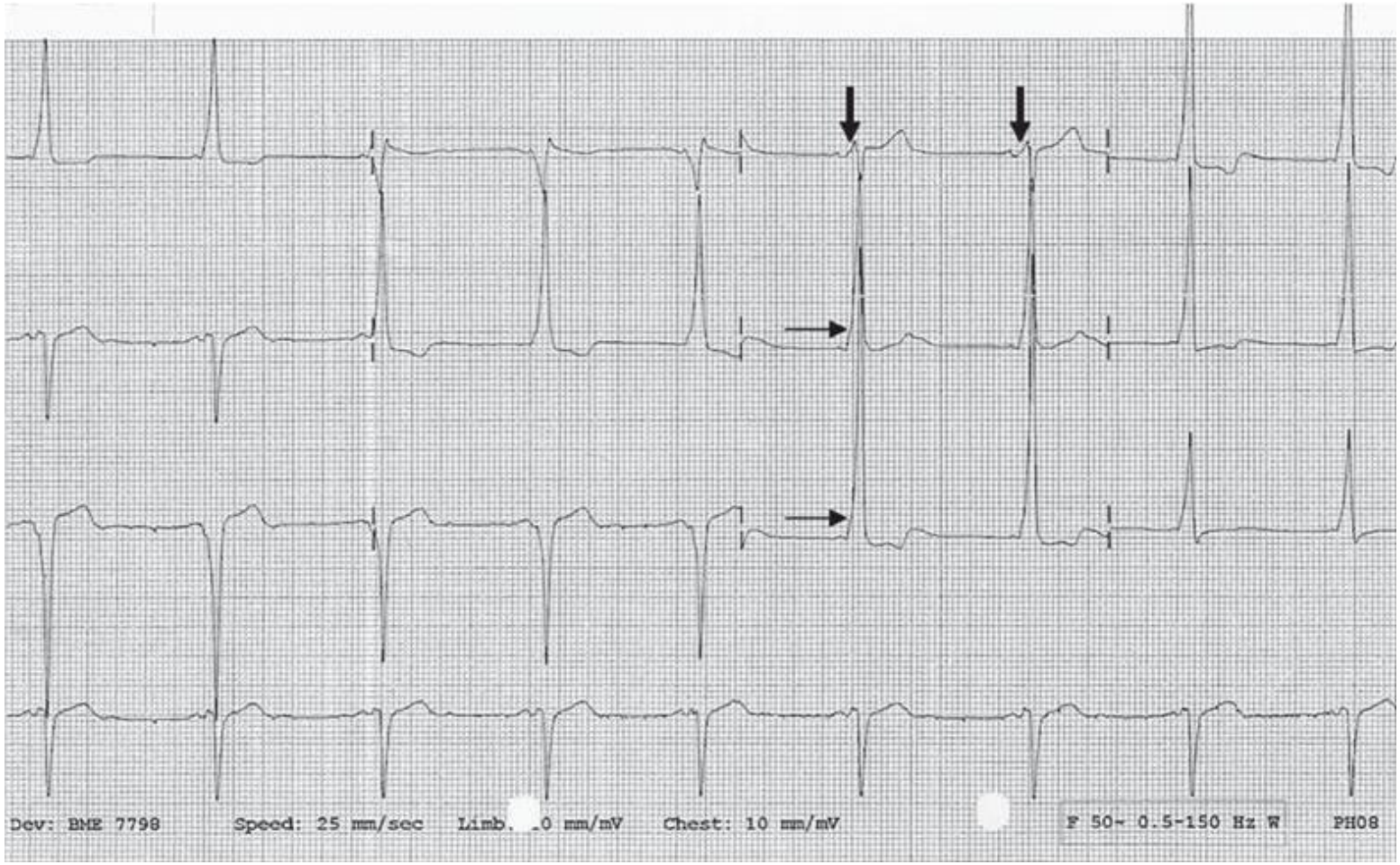
- Accessory pathway
- Pre-excitation





WPW Syndrome

- Short PR (<120ms)
- Delta wave
- Wide QRS


Dev: BME 7798
Speed: 25 mm/sec
Limb. .0 mm/mV
Chest: 10 mm/mV
F 50~ 0.5-150 Hz W
PH08

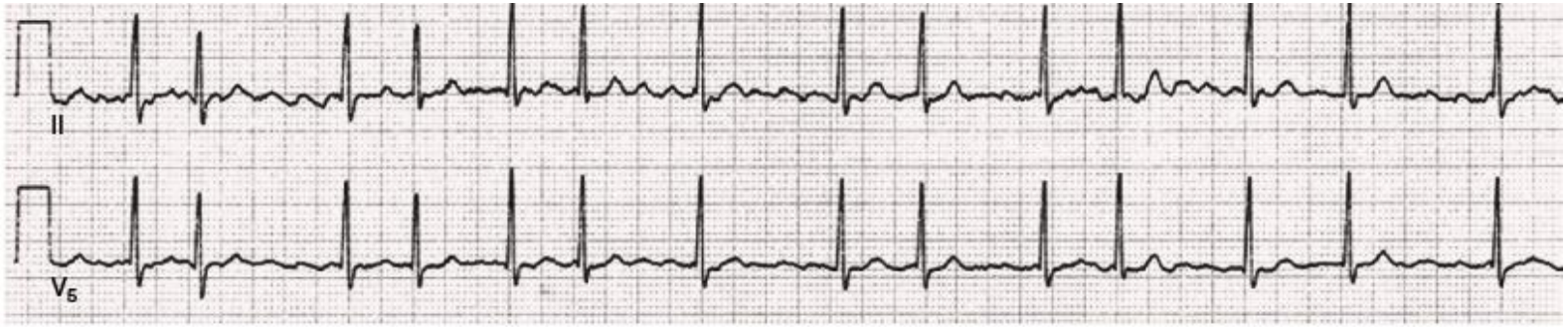
Atrioventricular reentrant tachycardias (AVRT)

- Orthodromic
  - Narrow complex
- Antidromic
  - Wide complex

Atrial Fibrillation

- Most common sustained arrhythmia
- irregularly irregular ventricular rhythm
- The f waves, 300 to 600 beats/min, are variable in amplitude, shape, and timing.
- The ventricular rate during untreated AF typically is 100 to 160 bpm
- No prominent P

Atrial Fibrillation



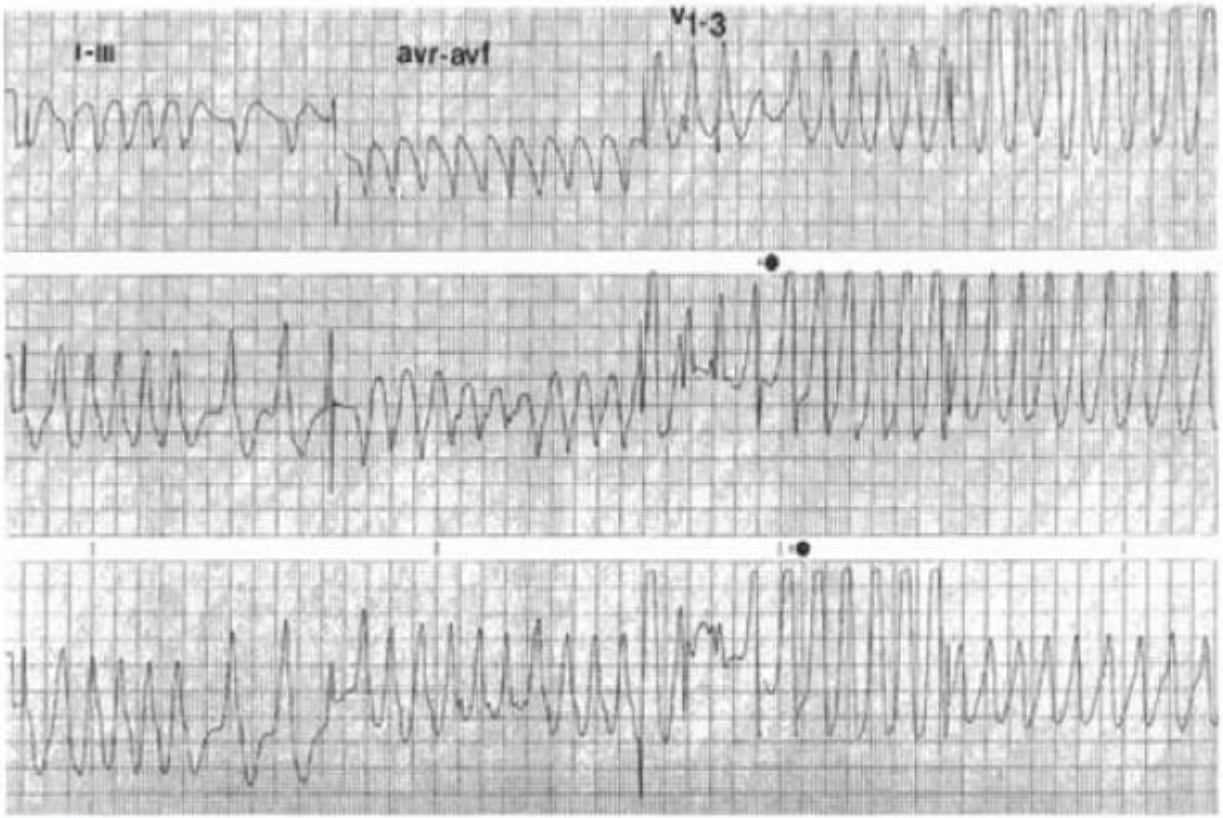
Atrial Fibrillation clinical importance

- Loss of atrial effective contraction
- Palpitation, chest pain, heart failure when RVR
- Loss of LAA contraction leads to increased clot formation and embolic CVA risk

AF treatment considerations

- Patient clinical status
- Acute or chronic
- Anticoagulation level
- CVA risk
- Symptoms
- Ventricular response (rapid or slow)
- Bleeding risk

AF+WPW



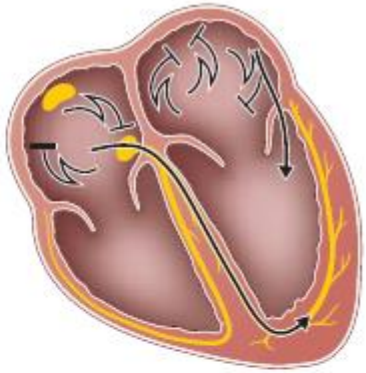


FIGURE 276-8 Preexcited atrial fibrillation (AF) due to conduction over a left free wall accessory pathway (AP)

AF+WPW

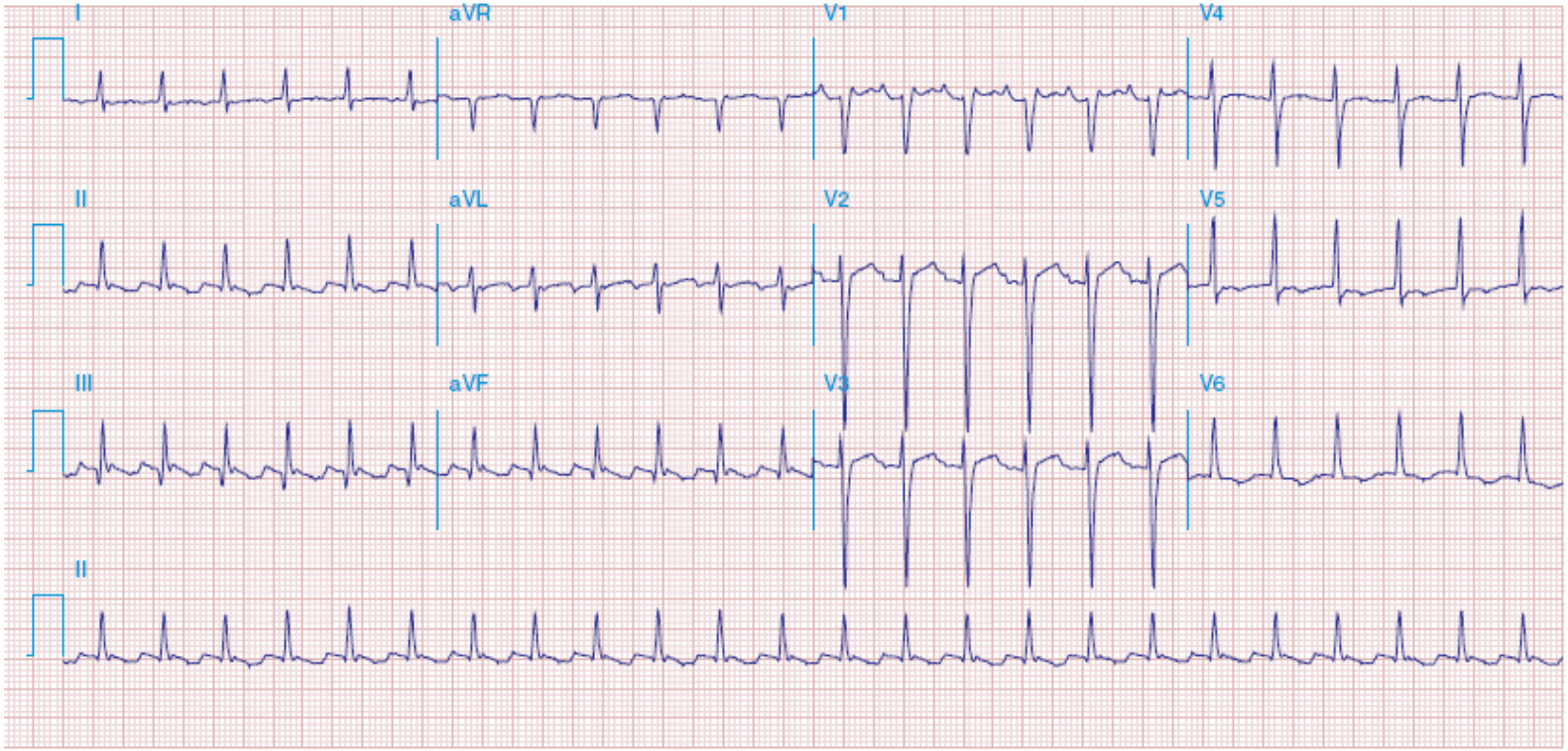
- SCD risk
- BB or CCB or digoxin may lead to VF

AF+WPW treatmnet

- Procainamide in acute setting
- AP radiofrequency ablation

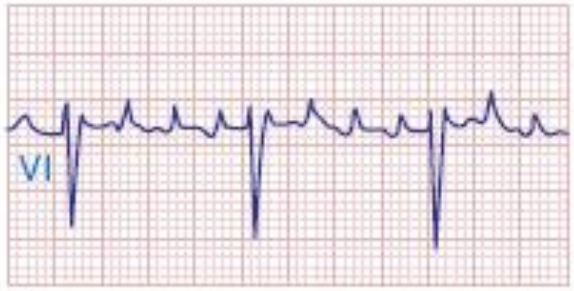
Atrial Flutter

- Macro-reentry
- Atrial rate:260-300
- 2:1 ventricular response
- Most common around tricuspid valve
- Rate control as AF (more difficult)
- DC shock (50-100 j) maybe needed for maintaining sinus rhythm
- CTI ablation

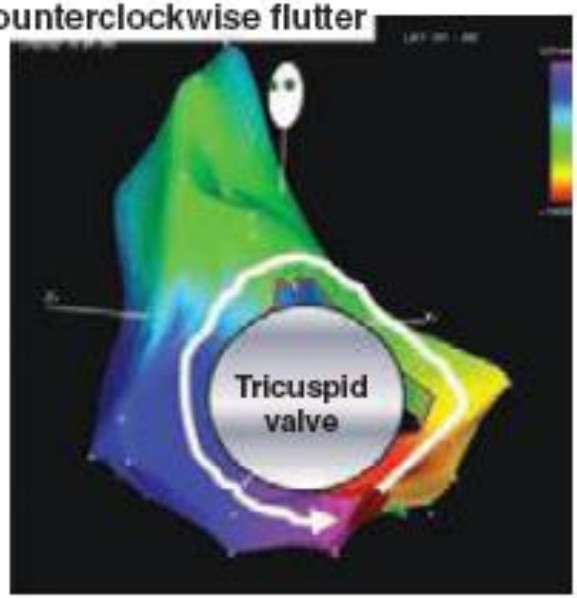


Atrial flutter with 2:1 AV conduction. Note typical atrial flutter waves, partly hidden in the early ST segment, seen,

Saw toothed waves in typical AFL



A



B

AF vs. AFL

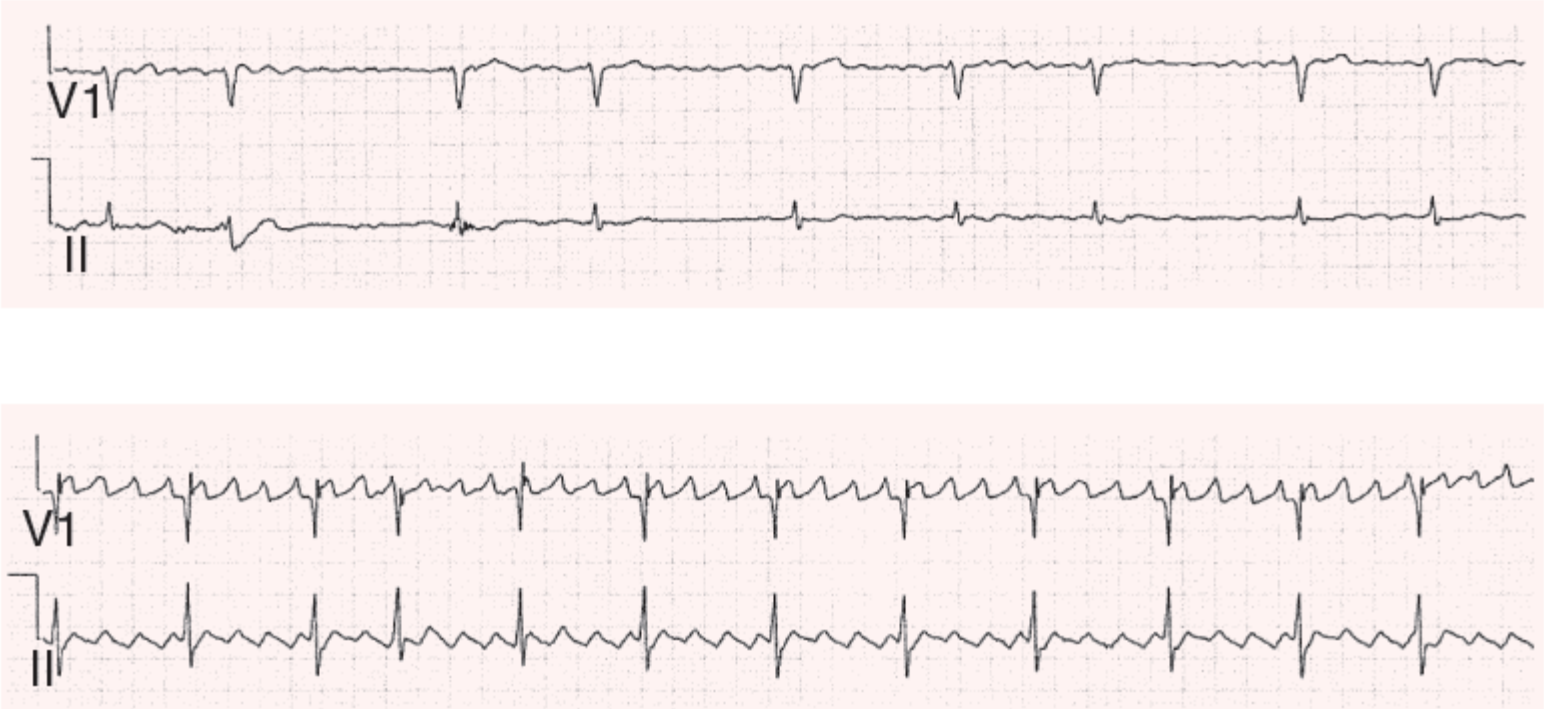


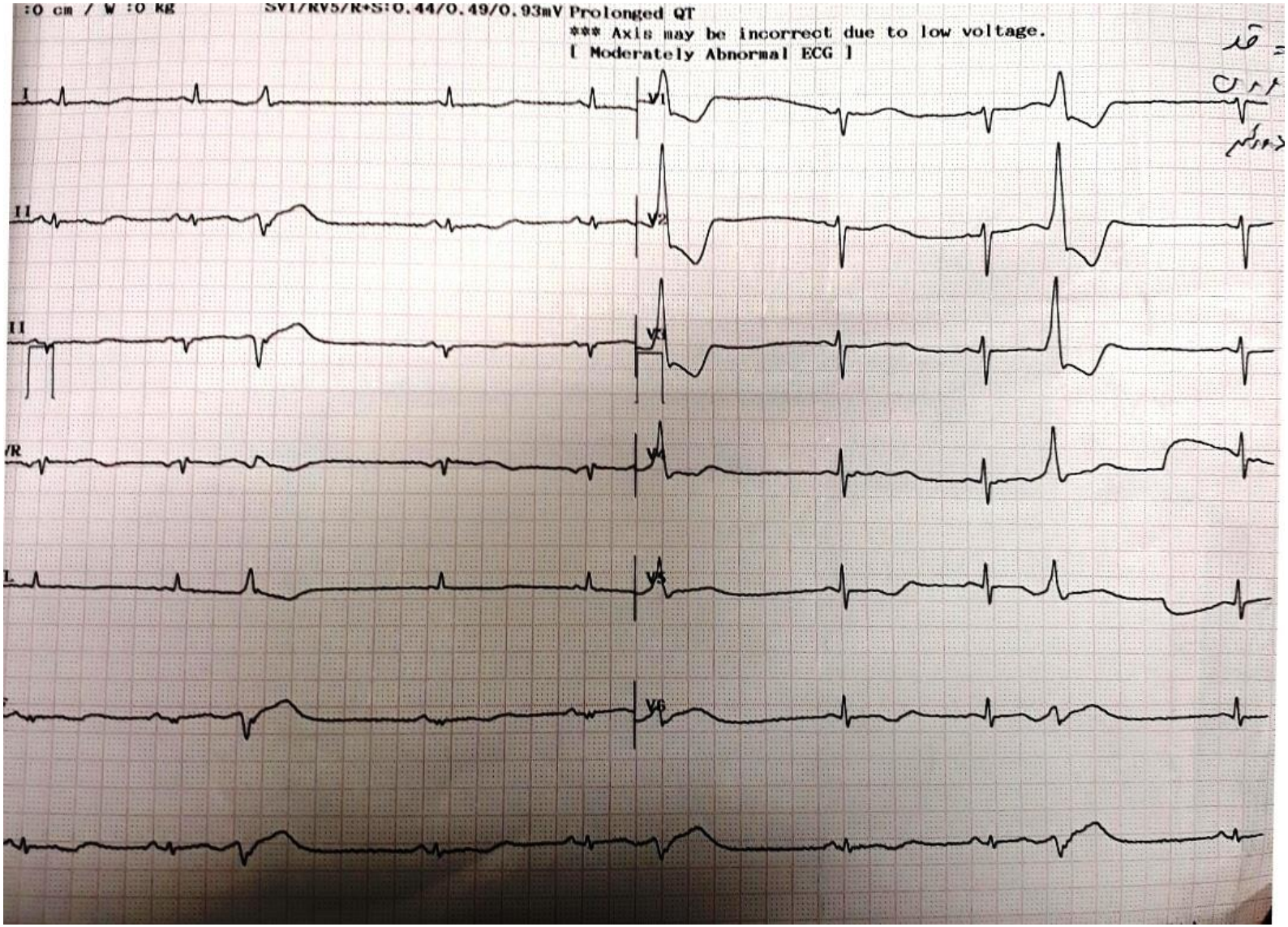
FIGURE 38.1 Comparison between the f waves of AF **(top panel)** and the flutter waves of atrial flutter **(bottom panel).** Note that f waves are variable in rate, shape, and amplitude, whereas flutter waves are constant in rate and all aspects of morphology. Shown are leads V_1 and II.

Ventricular arrhythmias

- premature ventricular complex (PVC)
- Ventricular Tachycardia
- Ventricular Fibrillation

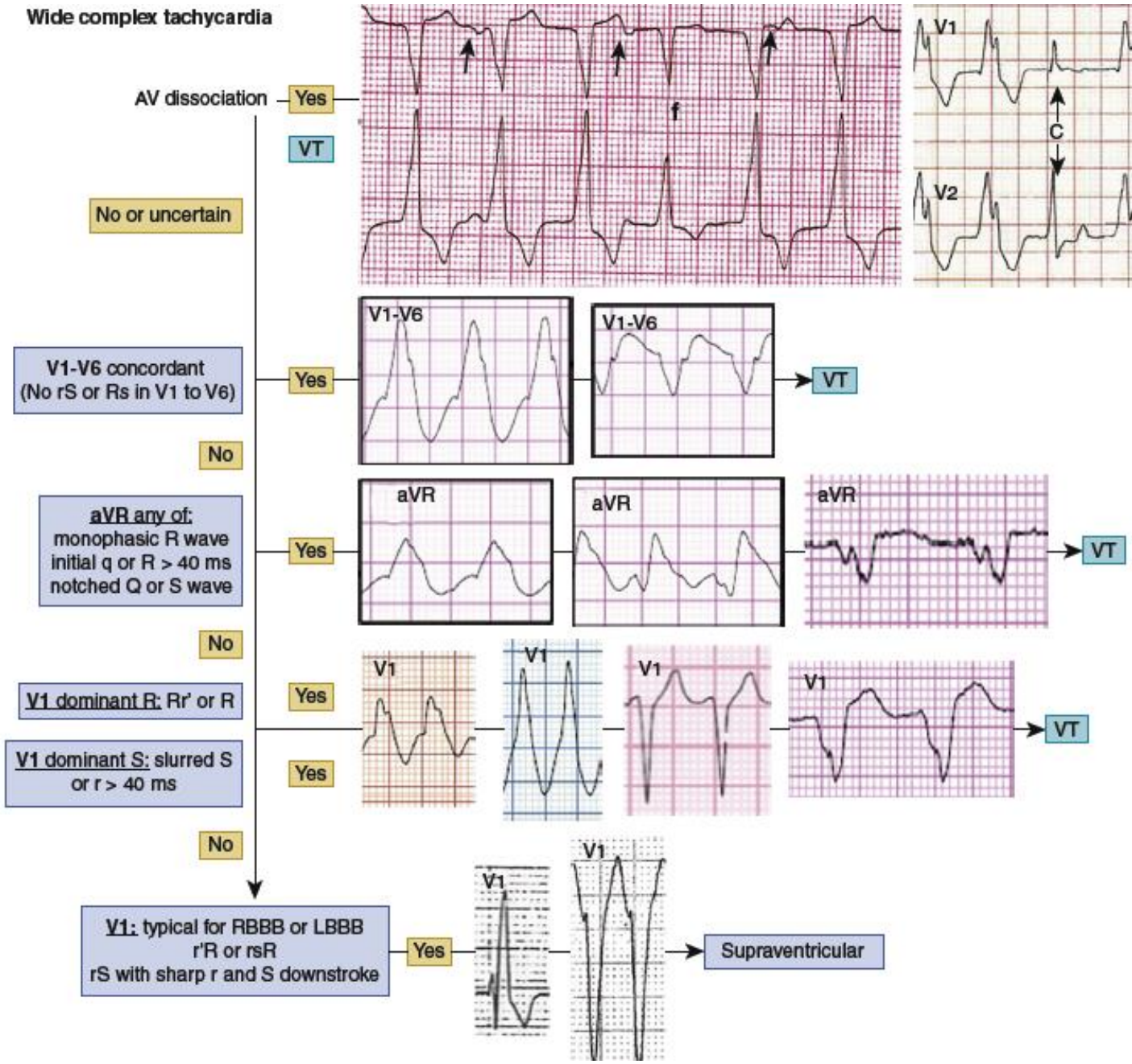
Premature ventricular complex (PVC)

- PVCs are due to abnormal impulse formation (automaticity, triggered activity) or reentry in the ventricular myocardium or Purkinje system,
- producing a depolarization wavefront that propagates through the ventricles independent of activation from the atrium and AV node.
- The mechanism cannot usually be determined with certainty.
- A PVC is characterized by the premature occurrence of an abnormal QRS complex that usually has a duration exceeding 120 msec.
- The corresponding T wave is typically broad and in the opposite direction of the major QRS deflection.
- It is typically not preceded by a P- wave.
- Premature atrial or junctional beats that conduct with bundle branch block can mimic PVCs.

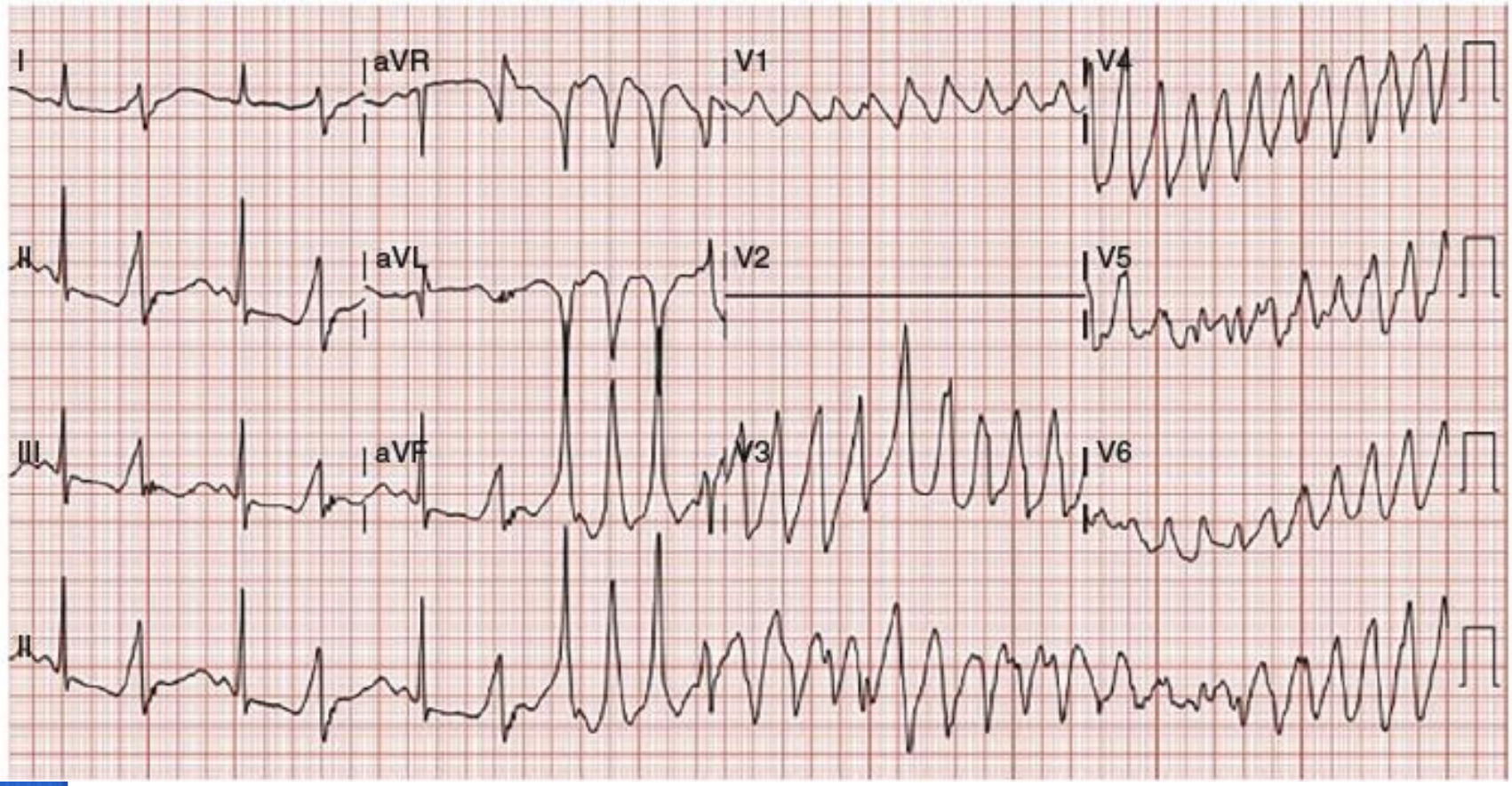

:0 cm / W :0 kg
SV1/RV5/R+S:0.44/0.49/0.93mV Prolonged QT
*** Axis may be incorrect due to low voltage.
[Moderately Abnormal ECG]
I
II
II
/R
L
V1
V2
V3
V4
V5
V6

Ventricular Tachycardia

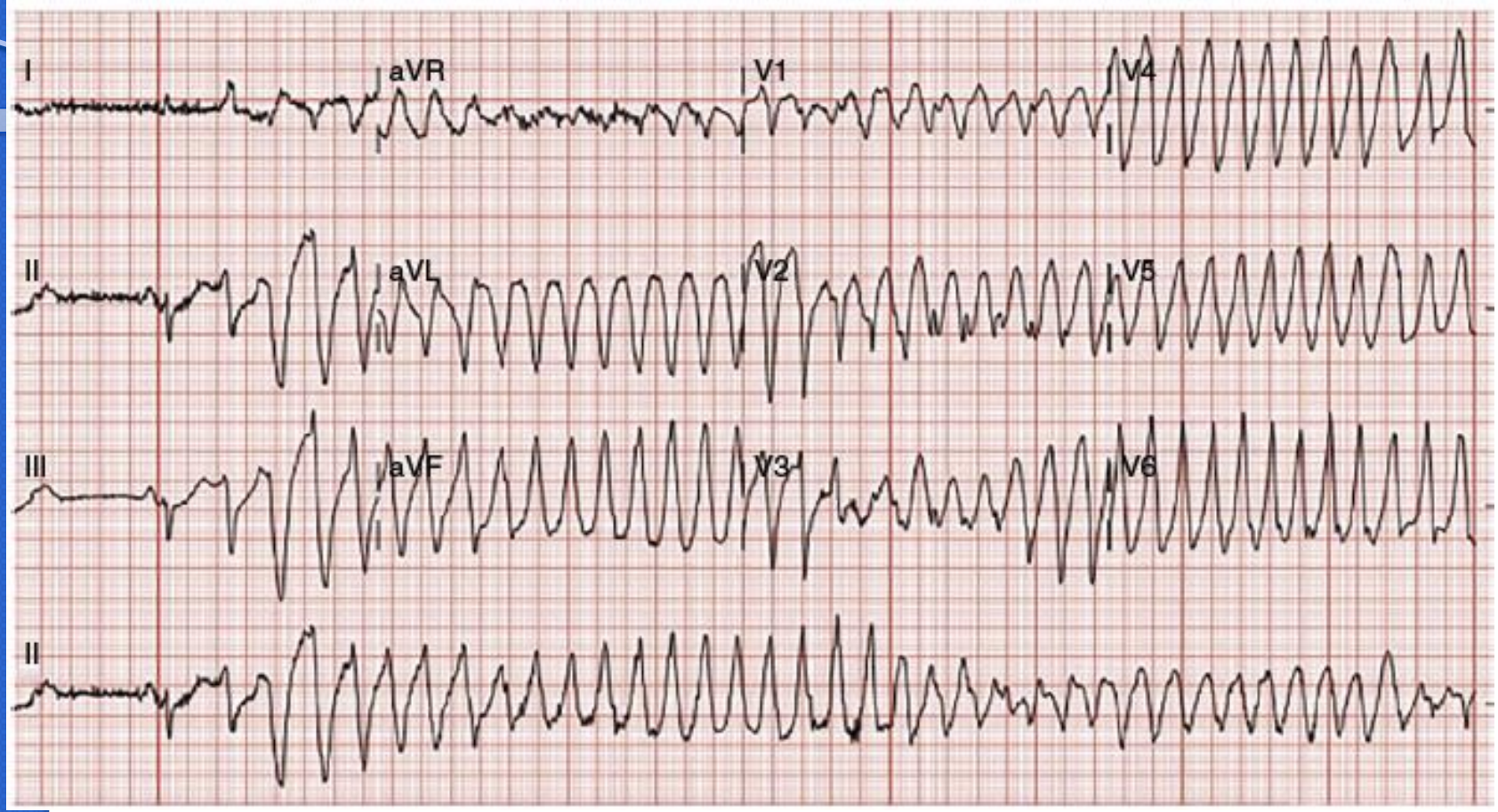
- Wide complex
- Regular
- In structural heart disease or normal heart
- Monomorphic or polymorphic


Wide complex tachycardia
AV dissociation
Yes
VT
No or uncertain
V1
C
V2
V1-V6 concordant
(No rS or Rs in V1 to V6)
Yes
V1-V6
V1-V6
VT
No
aVR any of:
monophasic R wave
initial q or R > 40 ms
notched Q or S wave
Yes
aVR
aVR
aVR
VT
No
V1 dominant R: Rr' or R
Yes
V1
V1
V1
V1
VT
V1 dominant S: slurred S
or r > 40 ms
Yes
No
V1: typical for RBBB or LBBB
r'R or rsR
rS with sharp r and S downstroke
Yes
V1
V1
Supraventricular

Polymorphic VT



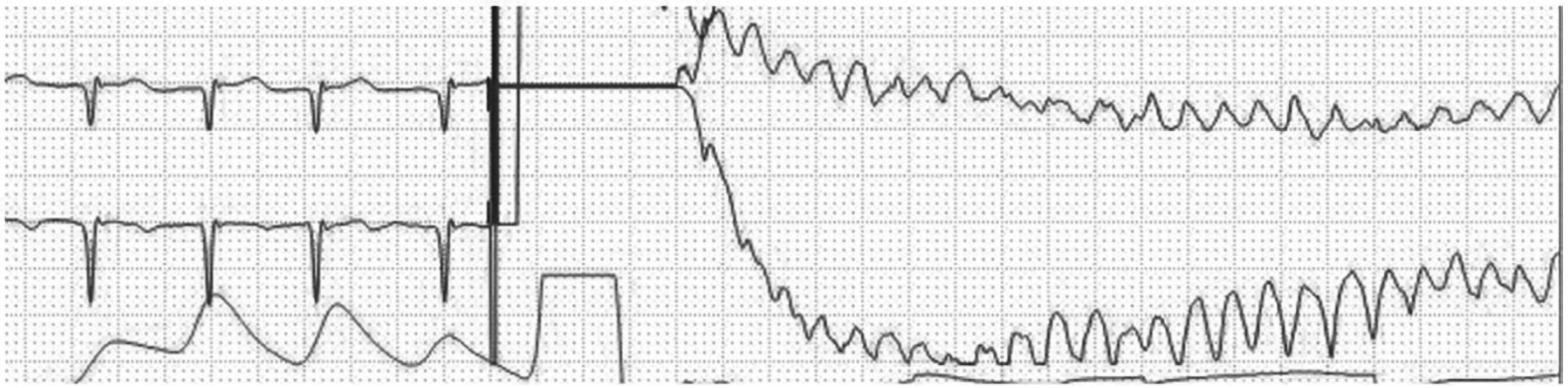
Torsade de pointes



Ventricular Fibrillation

- VF is a terminal arrhythmia followed by death or severe brain injury from lack of perfusion if not corrected within 3 to 5 minutes.
- It is characterized by irregular undulations of varying contour and amplitude without distinct QRS complexes

- Asynchronous DC cardioversion using 200 J
- Cardiopulmonary resuscitation is performed until cardioversion can be performed and resumed immediately after each shock



Thanks for your attention