MEDAVIE HealthEd ÉduSanté

Rhythm Interpretation

Part B: AV Junction and Ventricles

Primary Care Paramedicine

Module: 12

Section: 04b

Rhythms of the AV Node or AV Junction

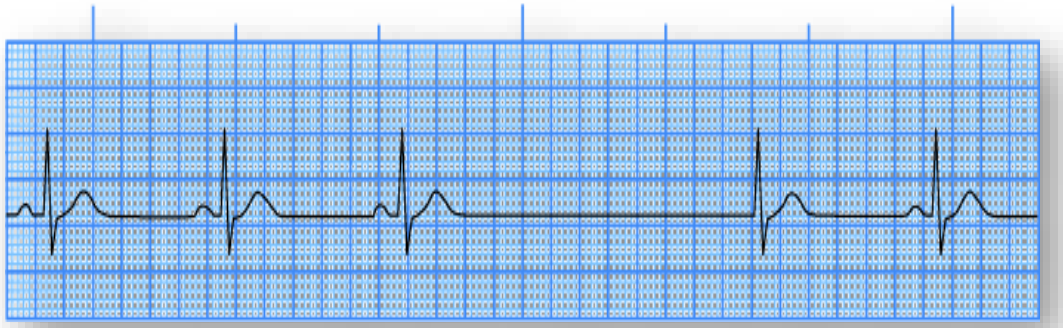
- If the SA node fails to initiate an impulse, the AV junction may take over as pacemaker.
- Because the AV junction is a secondary pacemaker, its intrinsic rate is slower than that of the SA node (40 to 60 beats/min).
- Impulse travels down through the conduction system into the ventricles.

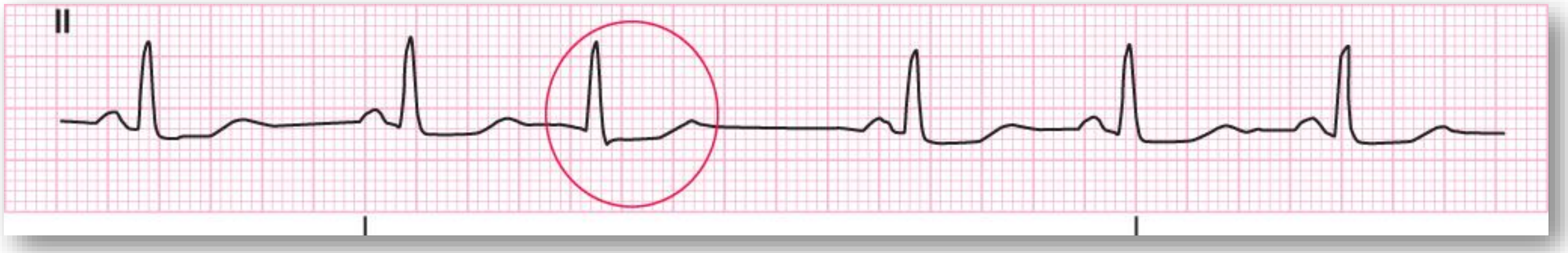
Dysrhythmias Originating in the AV Node

- Junctional Escape Complexes (Premature junctional complex)
- Junctional Escape Rhythm
- Accelerated Junctional Rhythm
- Junctional Tachycardia
- Junctional Bradycardia

Junctional Escape Complexes
Premature junctional complex (PJC)

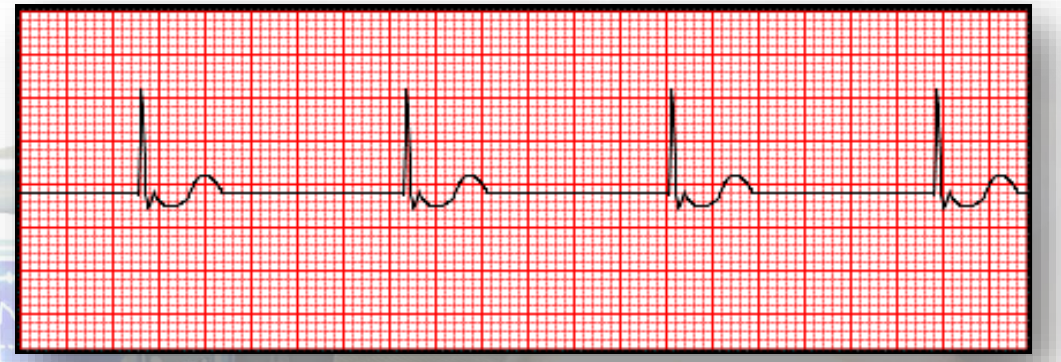
- Any early complex that appears within another rhythm
- P wave is absent or inverted
- Also known as ectopic complexes
- Occurs earlier in time than the next expected sinus complex
- Rarely treated in the prehospital setting

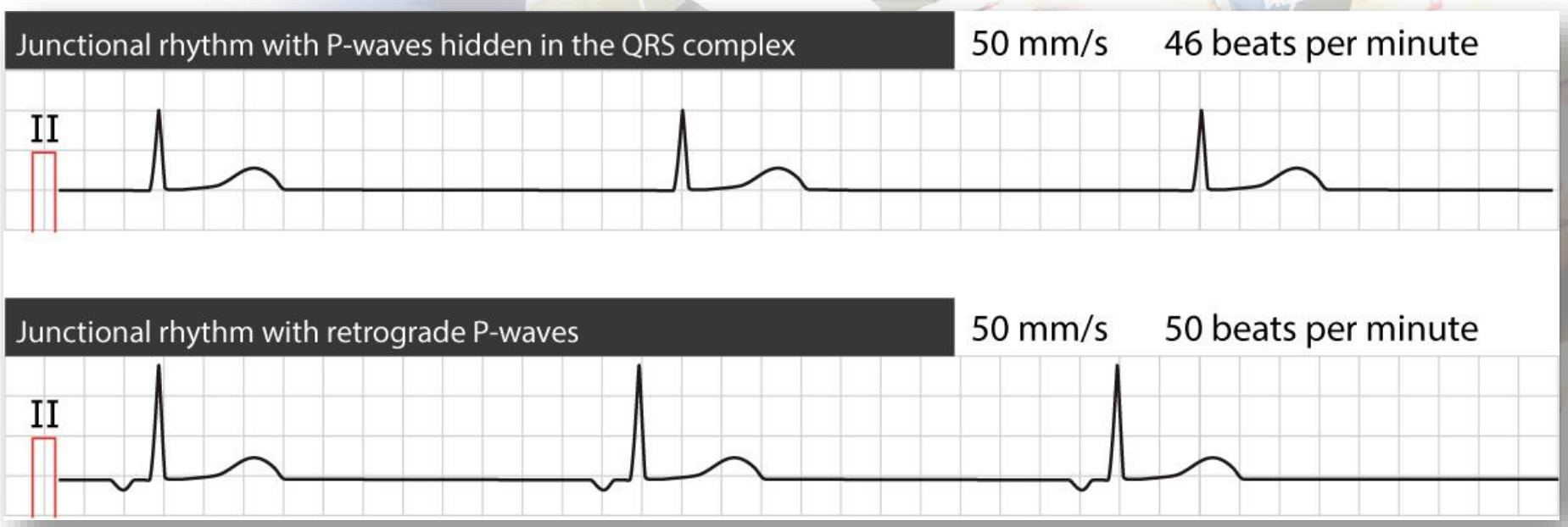




Junctional (escape) rhythm

Rate	40 - 60 bpm
Rhythm	Regular
P Waves	Inverted, or none, 1 per QRS
PRI	< 0.12 s (< 120 ms) if P waves present
QRS	<0.12 s (<120 ms), narrow





Junctional (escape) rhythm

Etiology

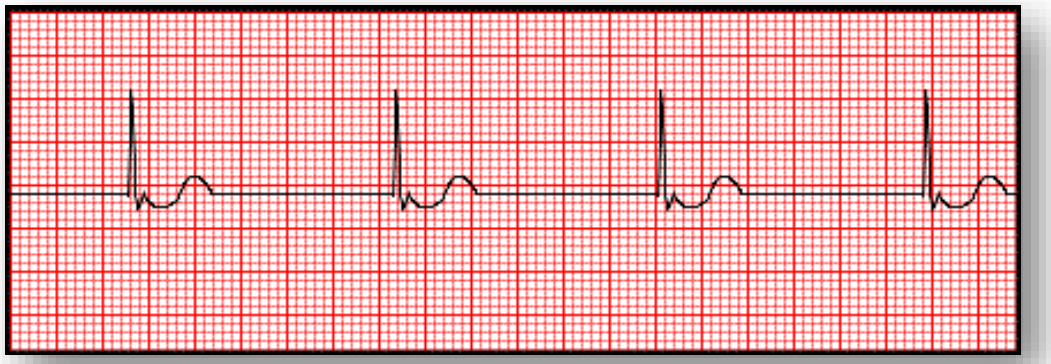
- Occurs when SA node ceases to function and AV junction takes over
- SA node overdrive suppression has stopped
- Results from increased vagal tone, pathologically slow SA discharges, or heart block

Clinical Significance

- Slow rate may decrease cardiac output, precipitating angina and other problems

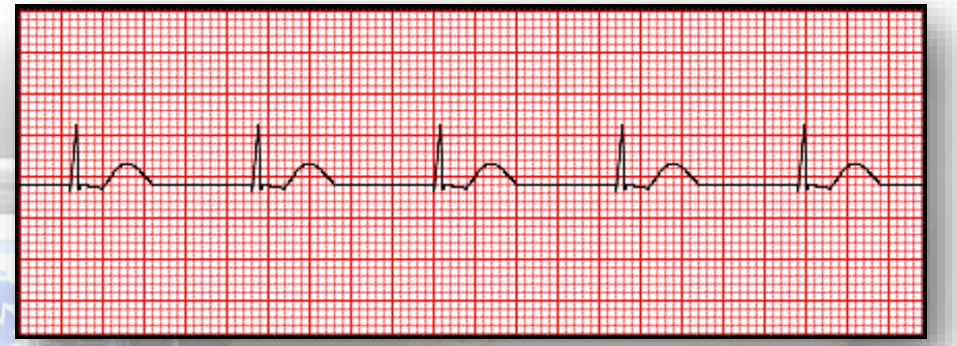
Treatment

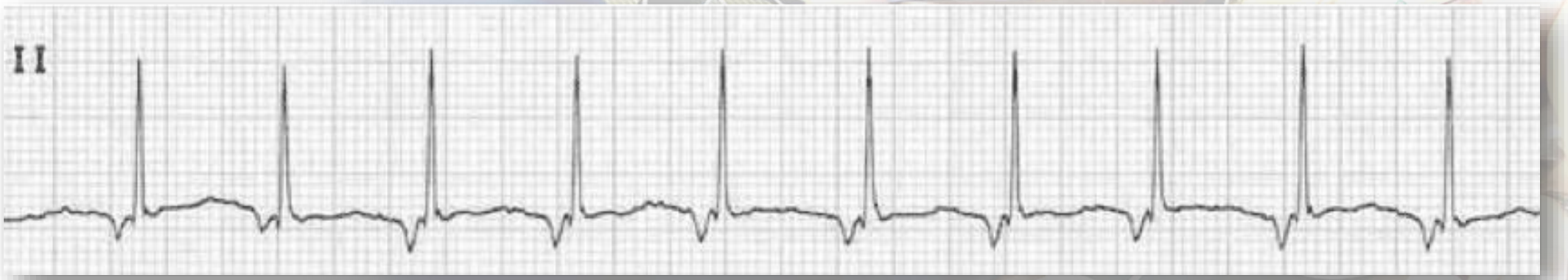
- None if the patient remains asymptomatic.
- ACP may treat symptomatic episodes with atropine or pacing as indicated



Accelerated Junctional

Rate	60 – 100 bpm
Rhythm	Regular
P Waves	Inverted, or none, 1 per QRS
PRI	< 0.12 s (< 120 ms) if P waves present
QRS	<0.12 s (<120 ms), narrow





Accelerated Junctional

Etiology

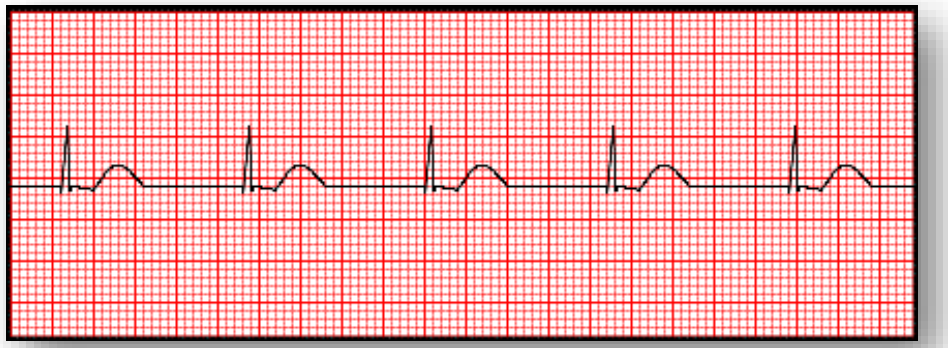
- Junctional rhythm with a rate exceeding the normal range of 60 beats/min
- Less than 100 beats/min
- Results from increased automaticity in the AV junction
- Often occurs due to ischemia of the AV junction

Clinical Significance

- Patient is usually asymptomatic
- Usually well tolerated, but monitor for other dysrhythmias

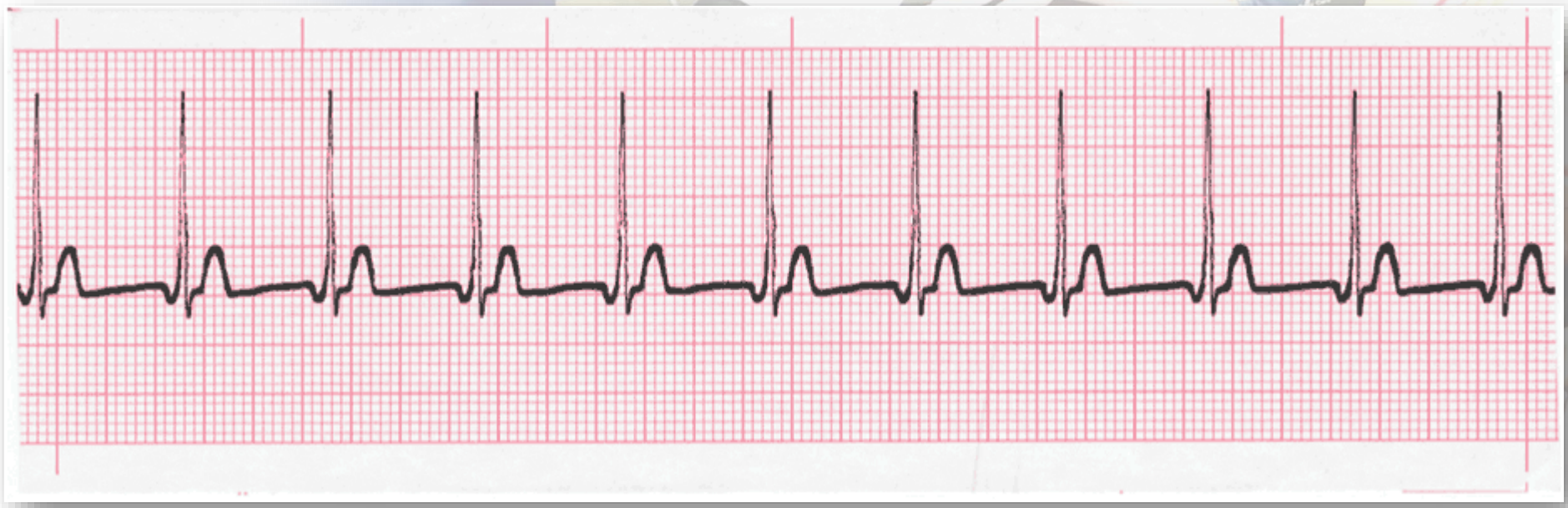
Treatment

- Seldom requires treatment in the prehospital setting (but should be monitored)



Junctional Tachycardia

Rate	100 - 150 bpm
Rhythm	Regular
P Waves	Inverted, or none, 1 per QRS
PRI	< 0.12 s (< 120 ms) if P waves present
QRS	<0.12 s (<120 ms), narrow



Junctional Tachycardia

Etiology

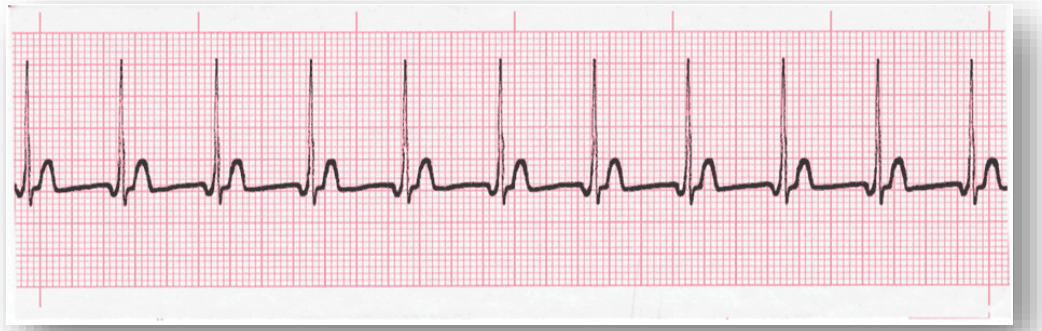
- Rapid AV junction depolarization overrides the SA node (Exceeds 100 bpm)
- Occurs with or without heart disease
- May be precipitated by stress, overexertion, smoking, or caffeine ingestion

Clinical Significance

- May be well tolerated for brief periods
- Decreased CO will result from prolonged episodes, which may precipitate angina, hypotension, or CHF
- If the rate exceeds 150 beats/min, the cardiac output could be negatively affected (SVT).

Treatment

- Serious, but seldom requires treatment in the prehospital setting

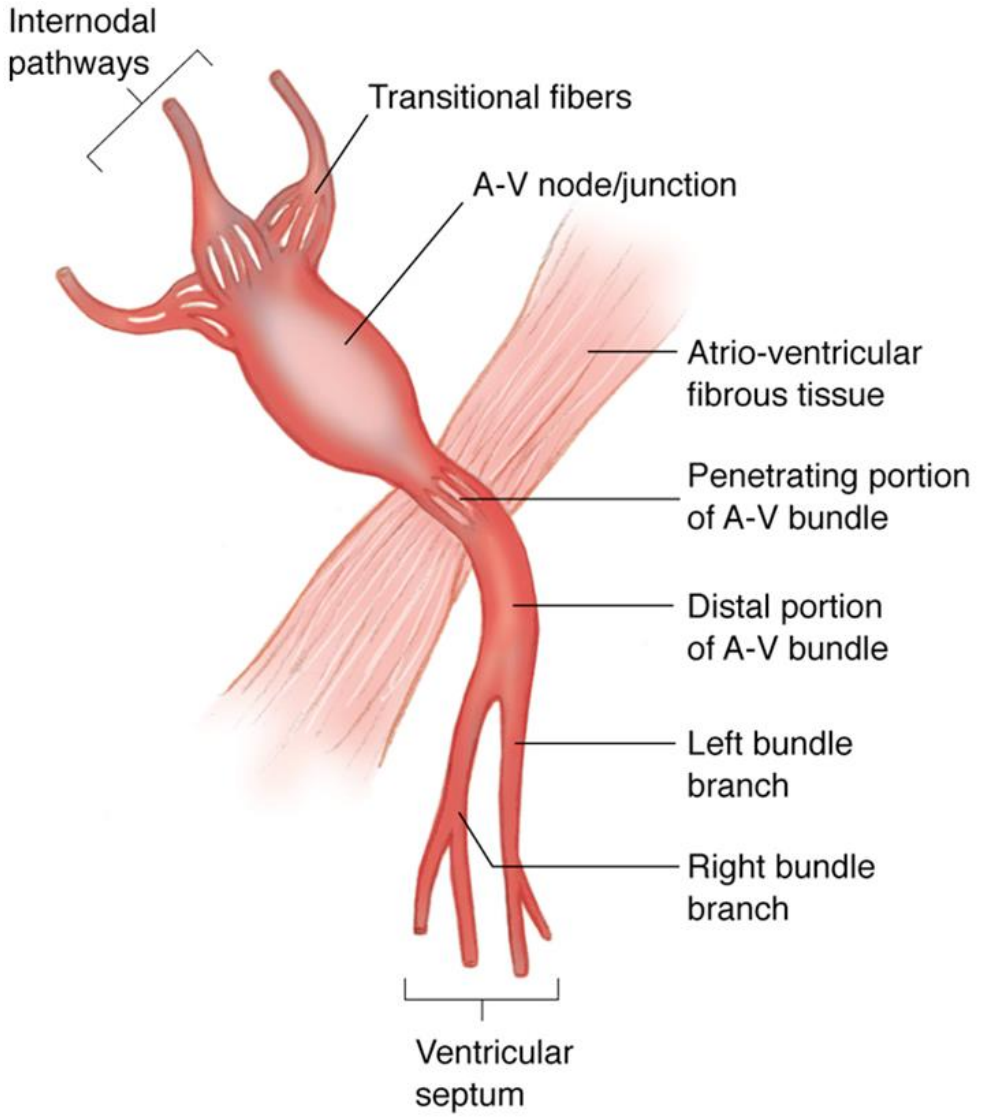


Atrioventricular Blocks

- Delays are a normal function of the AV node.
  - Occasionally the impulses are delayed more than usual, resulting in AV blocks.
  - Classified into different degrees based on the seriousness of the block and the amount of myocardial damage

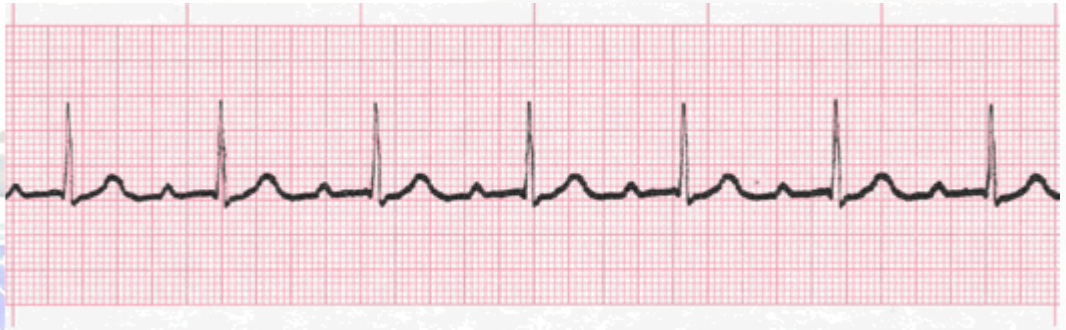
AV Blocks

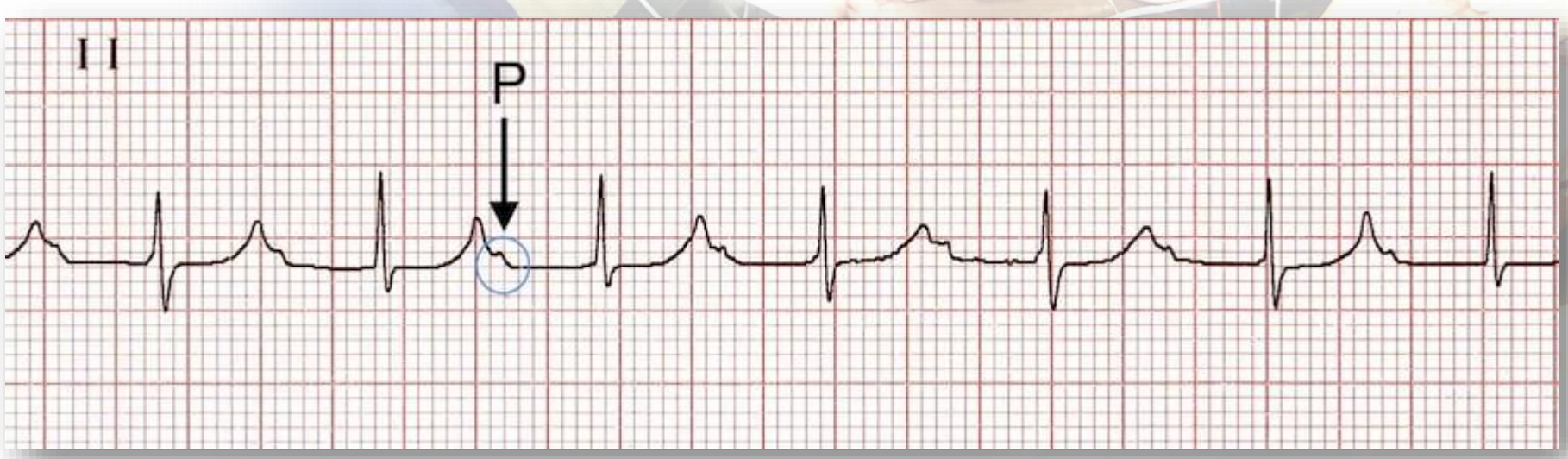
- 1st degree AV Block
- 2nd degree AV Block – Type I
- 2nd degree AV Block – Type II
- 3rd degree AV Block



1st degree AV Block

Rate	Depends on underlying rhythm
Rhythm	Regular
P Waves	Upright, 1 per QRS
PRI	> 0.20 s (> 200 ms)
QRS	<0.12 s (<120 ms), narrow





1st degree AV Block

Etiology

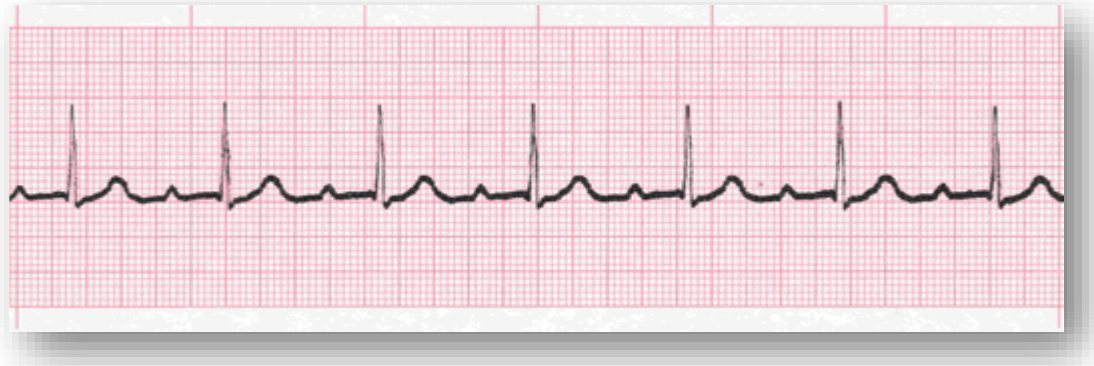
- Each impulse is delayed slightly longer than is expected.
- Impulse eventually passes through the AV node.
- Least serious of the AV blocks
- First indication of damage to the AV node

Clinical Significance

- Usually not significant, but new onset may precede a more advanced block

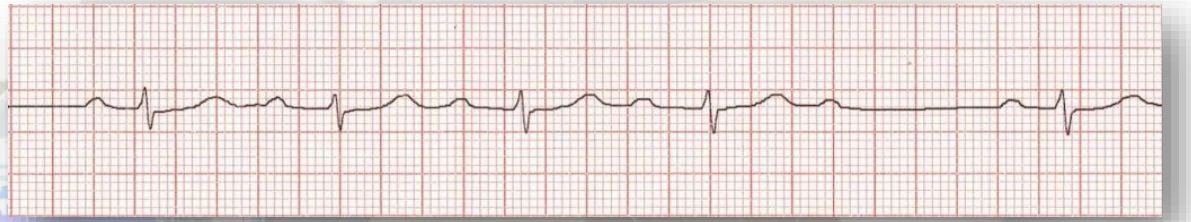
Treatment

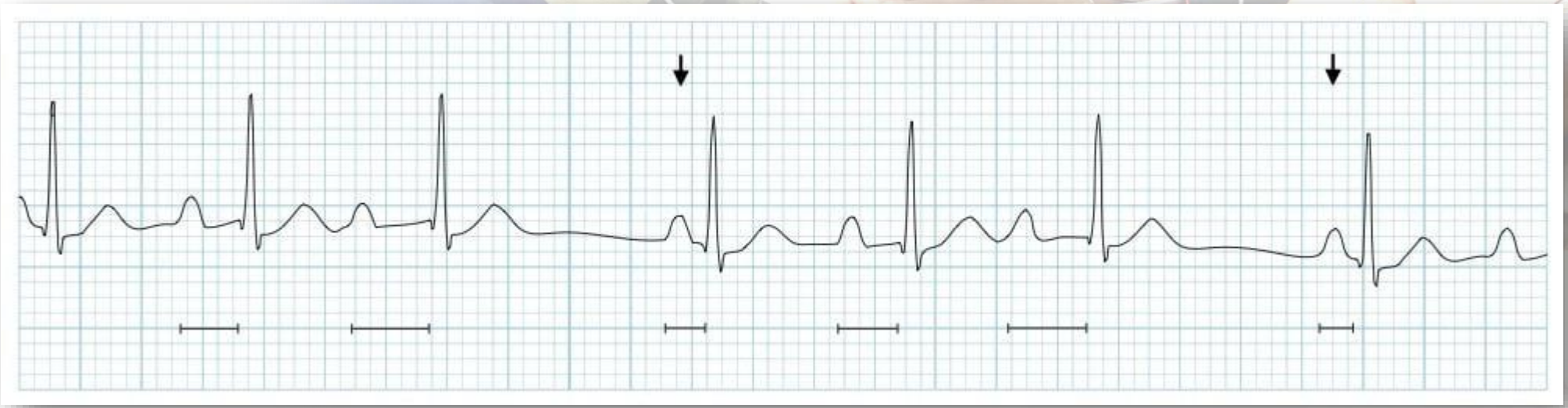
- Generally, none required other than observation
- Avoid drugs that may further slow AV conduction



2nd degree AV Block – Type I
Mobitz I (Wenckeback)

Rate	Depends on underlying rhythm
Rhythm	Regular irregular
P Waves	Upright, some not followed by QRS
PRI	Increasing until QRS dropped
QRS	<0.12 s (<120 ms), narrow





2nd degree AV Block – Type I
Mobitz I (Wenckeback)

Etiology

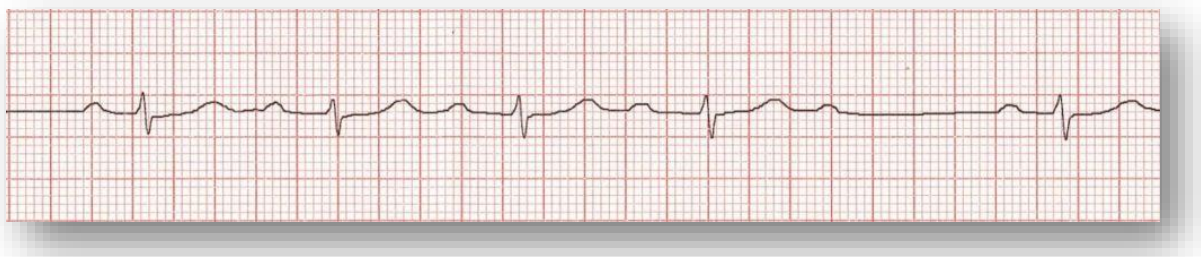
- Impulse is occasionally prevented from proceeding to the ventricles.
- Each is delayed a little longer.
- Intrinsic rate of 60 to 100 beats/min
- Increasing PRI

Clinical Significance

- May be indicative of ischemia at the AV junction
- Frequently dropped beats can result in cardiac compromise

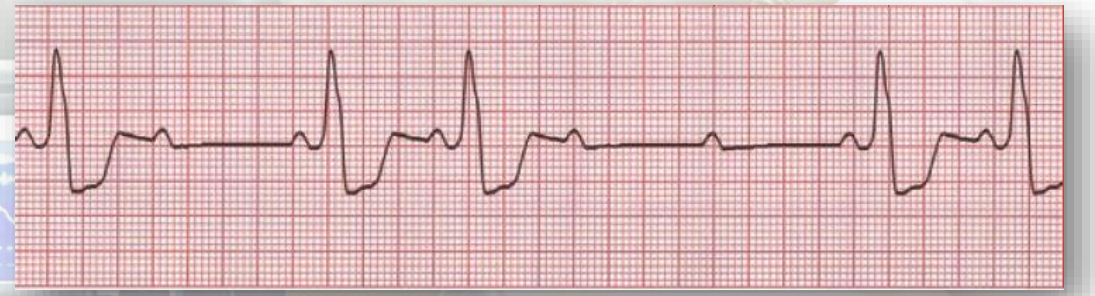
Treatment

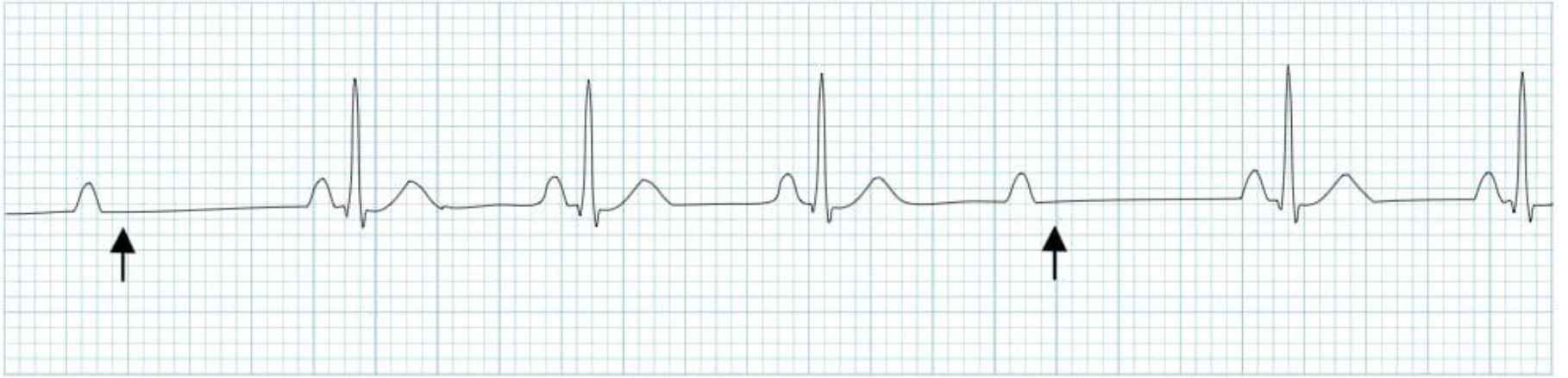
- Generally, none required other than observation
- Avoid drugs that may further slow AV conduction
- Treat symptomatic bradycardia



2nd degree AV Block – Type II
Mobitz II

Rate	Depends on underlying rhythm
Rhythm	Regularly irregular
P Waves	Upright, some p-waves not followed by QRS
PRI	Constant for conducted beats, may be > 0.21 s (> 210 ms)
QRS	Normal or > 0.12 s (> 120 ms)





2nd degree AV Block – Type II
Mobitz II

Etiology

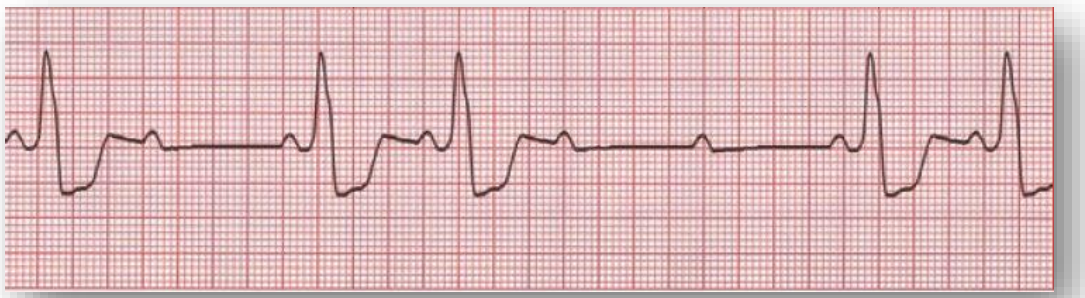
- Intermittent block of impulses
- Several impulses are not allowed to continue.
- Rhythm can be regular or irregular.

Clinical Significance

- Usually associated with MI or septal necrosis
- May compromise cardiac output and is indicative of MI
- Often develops into 3rd degree AV block

Treatment

- Avoid drugs that may further slow AV conduction
- Treated in the prehospital setting only if associated with bradycardia

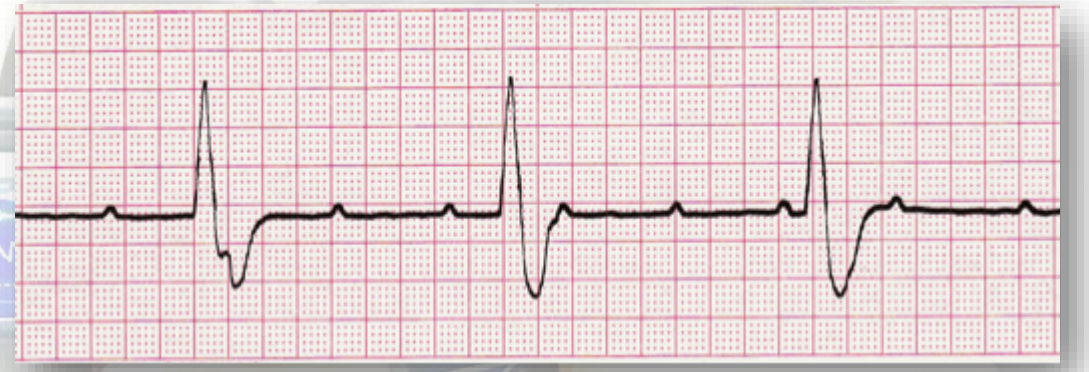


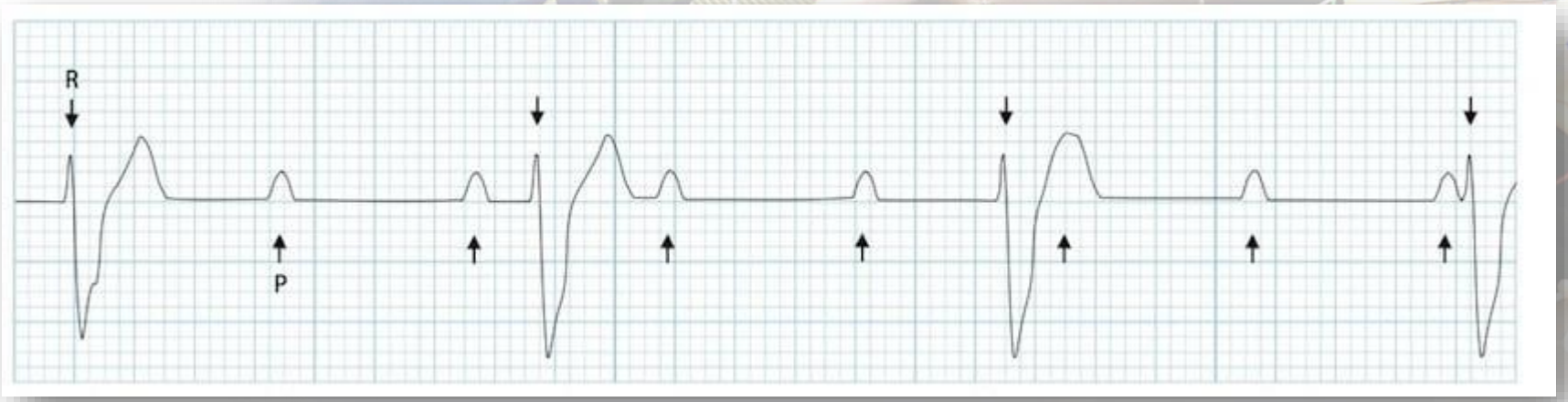
Second-Degree AV Blocks

- Both Type I and Type II typically occur in fixed ratio
  - Reported as ratio of # of P waves: # of QRS complexes
    - 4:3 Second-degree represents a dropped QRS complex every fourth atrial beat
  - It not possible to determine if a 2:1 second-degree is Type I or Type II since you cannot determine if PRI is lengthening

3rd degree AV Block
Complete Heart Block

Rate	Atrial rate is normal; ventricular, 40–60
Rhythm	Both atrial and ventricular are regular but not the same
P Waves	Normal in appearance with no correlation to QRS
PRI	No relationship to QRS
QRS	0.12 s (120 ms) or greater





3rd degree AV Block
Complete Heart Block

Etiology

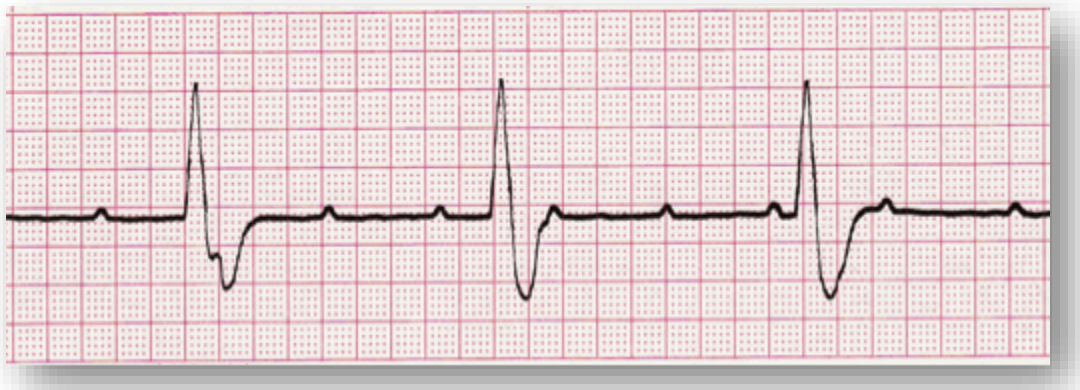
- All impulses are prevented.
- Ventricles develop their own pacemaker.
- Rate 40 to 60 beats/min if the pacemaker originates in the AV junction and 20 to 40 beats/min if it originates in the ventricles
- Nonconducted P waves

Clinical Significance

- Severely compromised cardiac output

Treatment

- Treat symptomatic bradycardia
- ACP:
  - Transcutaneous pacing for acutely symptomatic patients
- Avoid drugs that may further slow AV conduction



Rhythms of the Ventricles

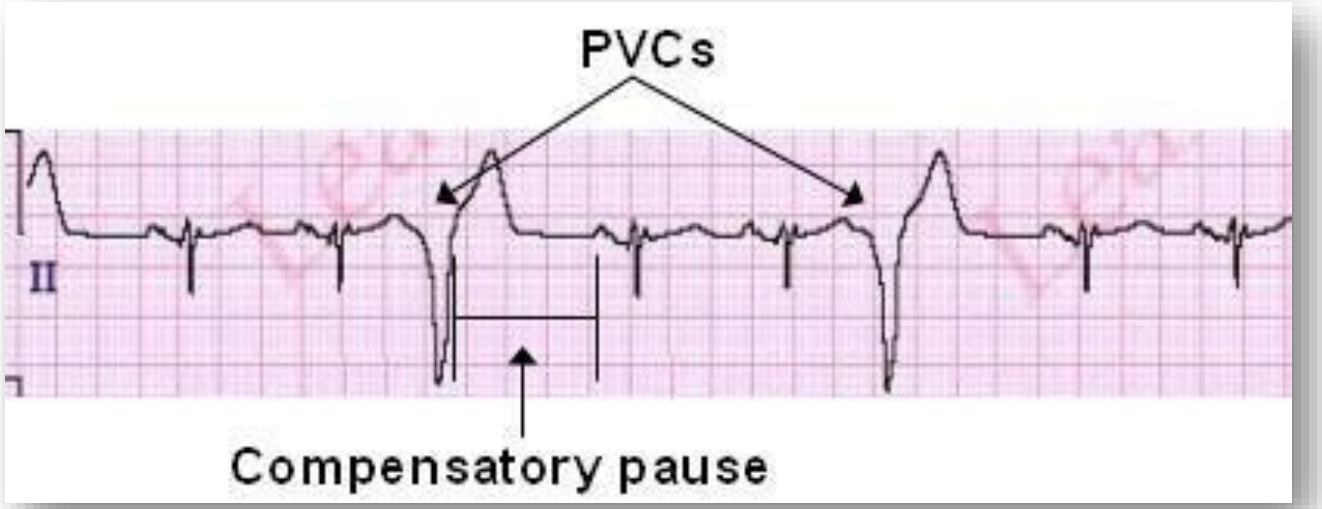
- Ventricles may begin to originate their own impulses.
  - Missing P waves and wide QRS complexes
  - Travels cell-to-cell
  - 20 to 40 beats/min

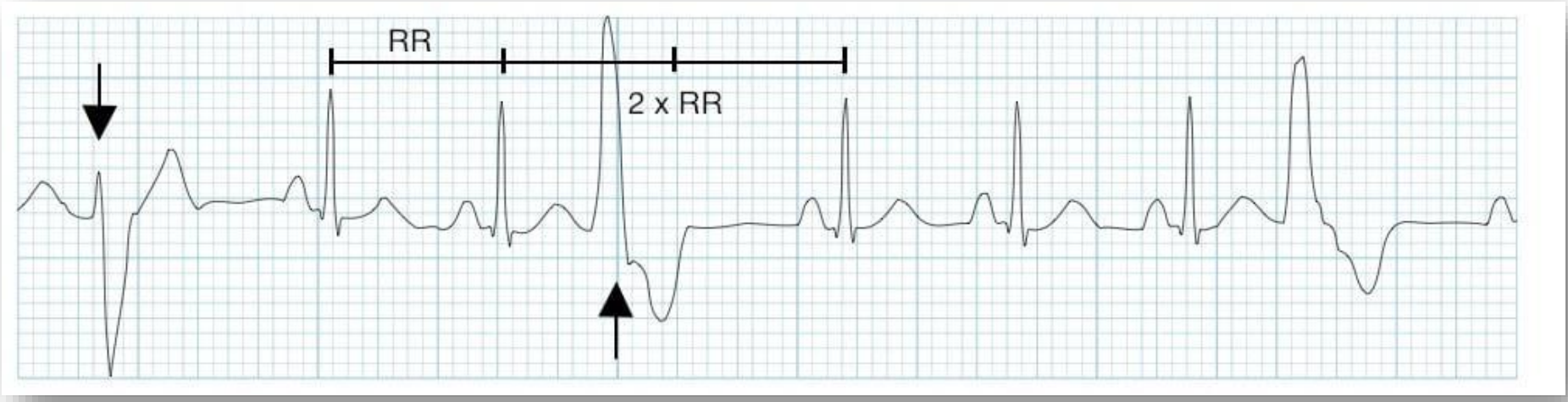
Dysrhythmias Originating in the Ventricle

- Premature ventricular complex
- Idioventricular rhythm
- Accelerated Idioventricular rhythm
- Ventricular tachycardia

Premature Ventricular Complex (PVC)

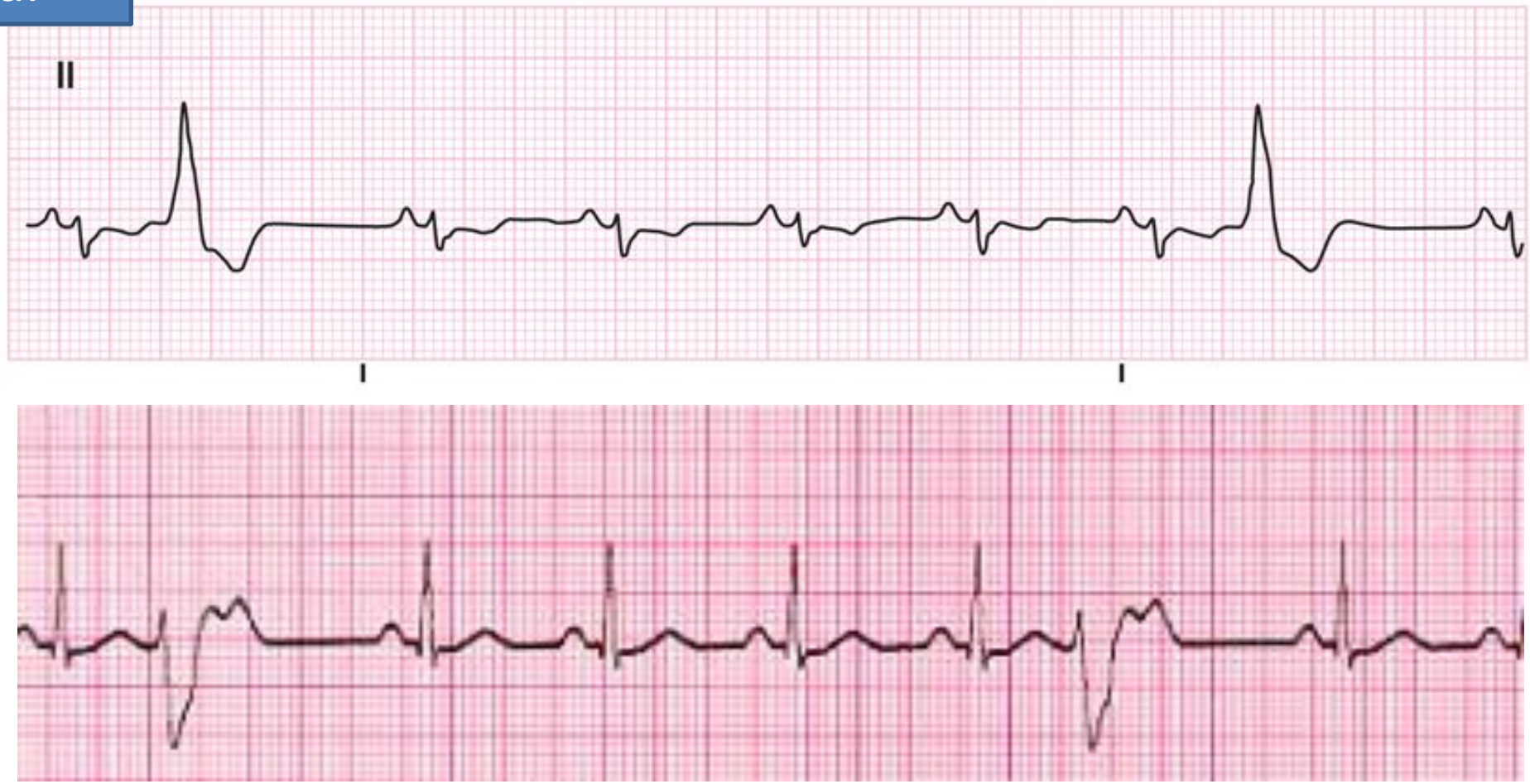
- Early complex that appears within another rhythm; also called ectopic complexes
- Occurs earlier than the next expected complex
- May be:
  - Unifocal
  - Multifocal





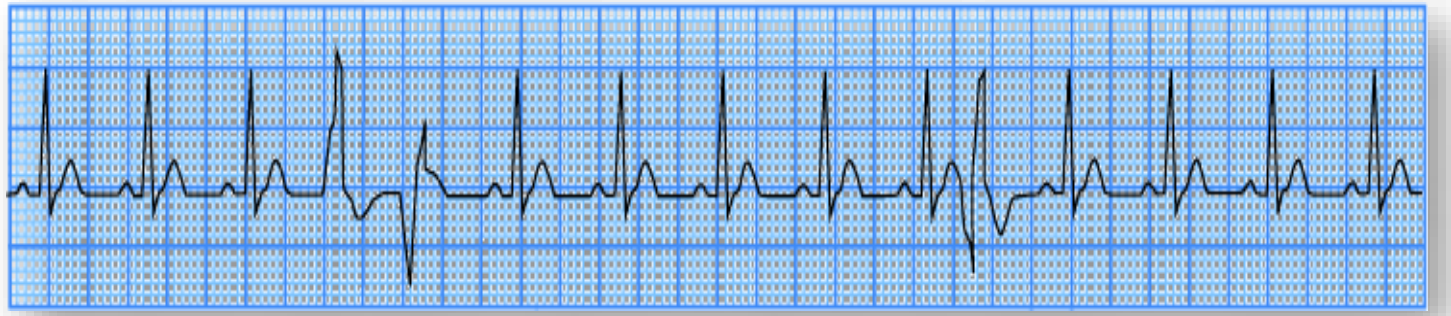
Premature Ventricular Complex (PVC)

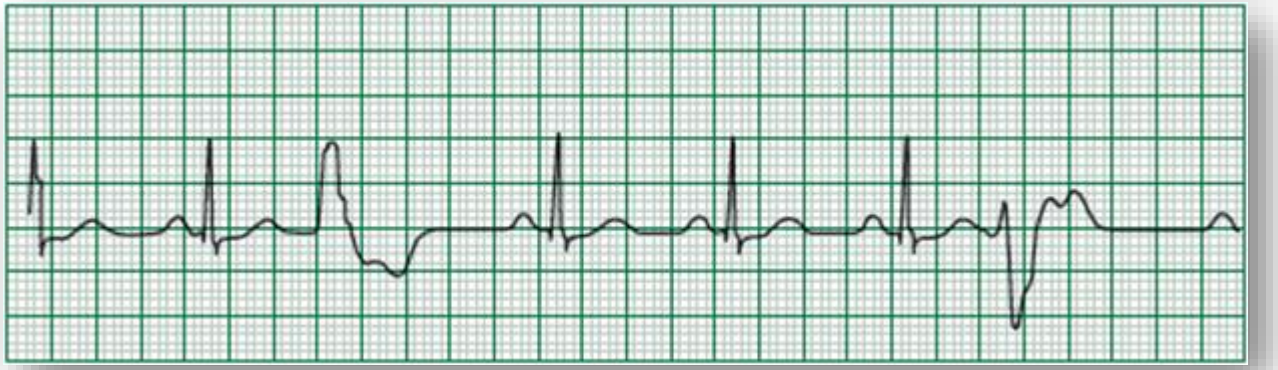
Unifocal



Premature Ventricular Complex (PVC)

Multifocal

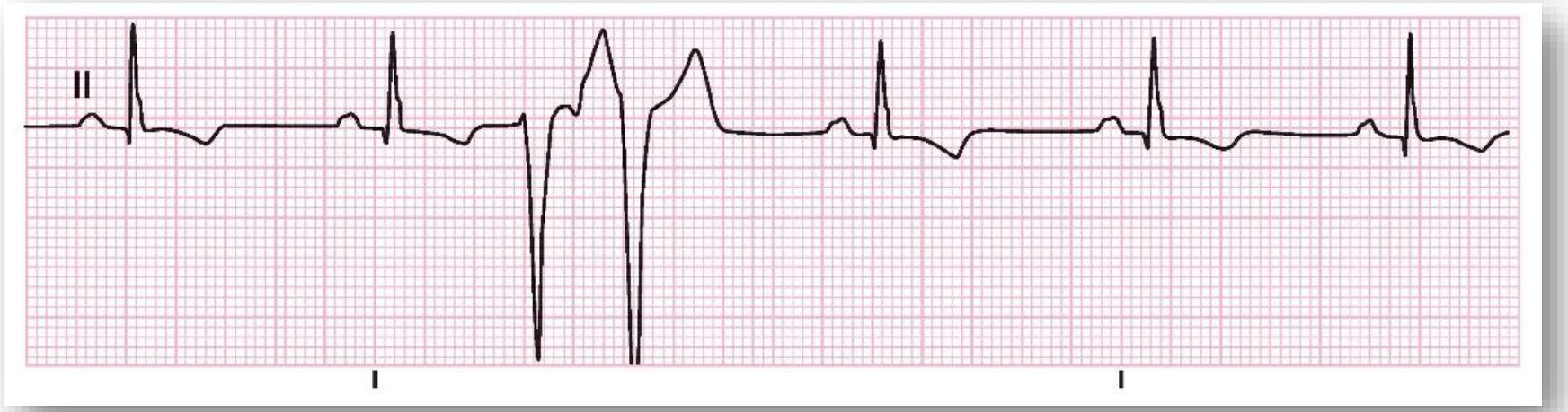




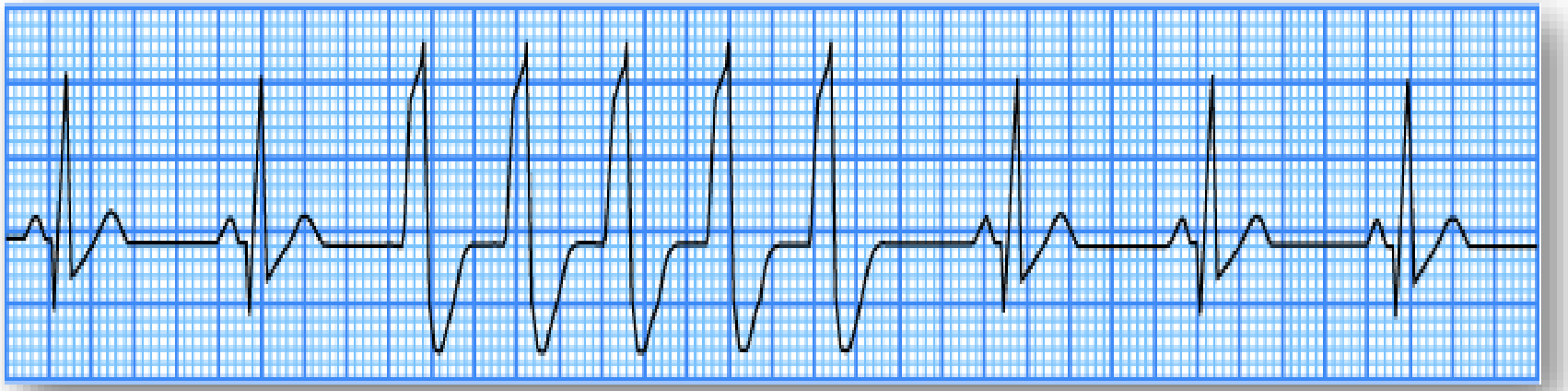
Premature Ventricular Complex (PVC)

Couplet

When 2 PVCs occur back to back = **couplet**
3 = triplet
4 = quadruplet

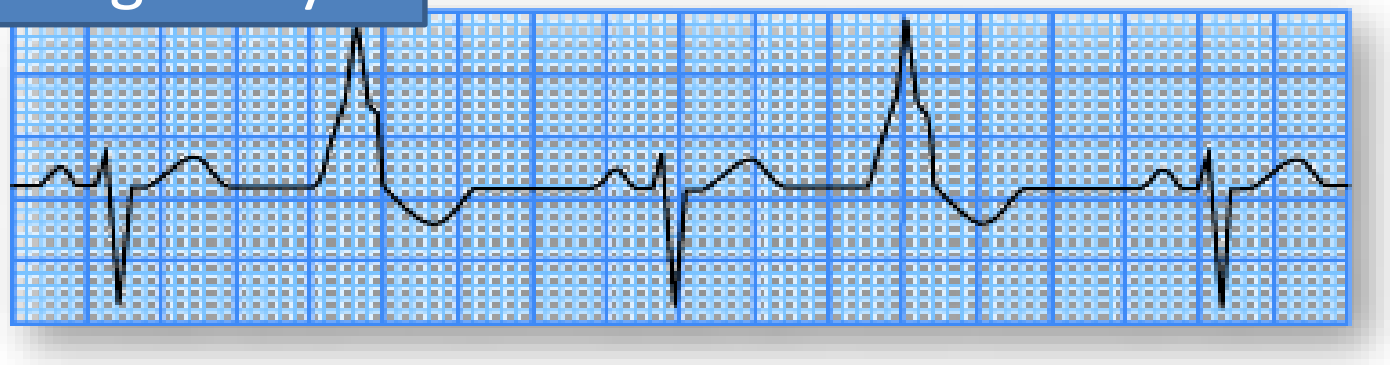


When 3 or move PVCs occur back to back to back = **run of VTach**



Premature Ventricular Complex

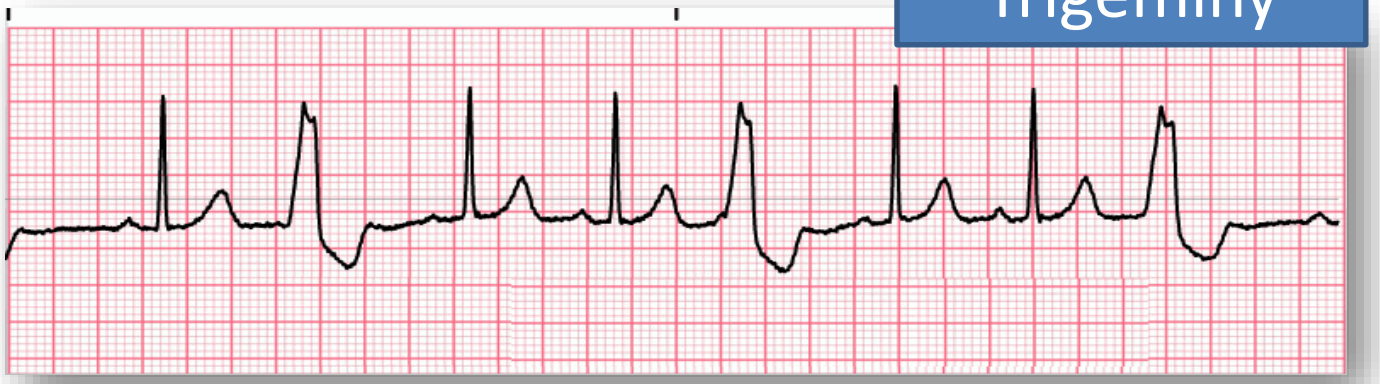
Bigeminy



When a PVC occurs every second complex = **bigeminy**

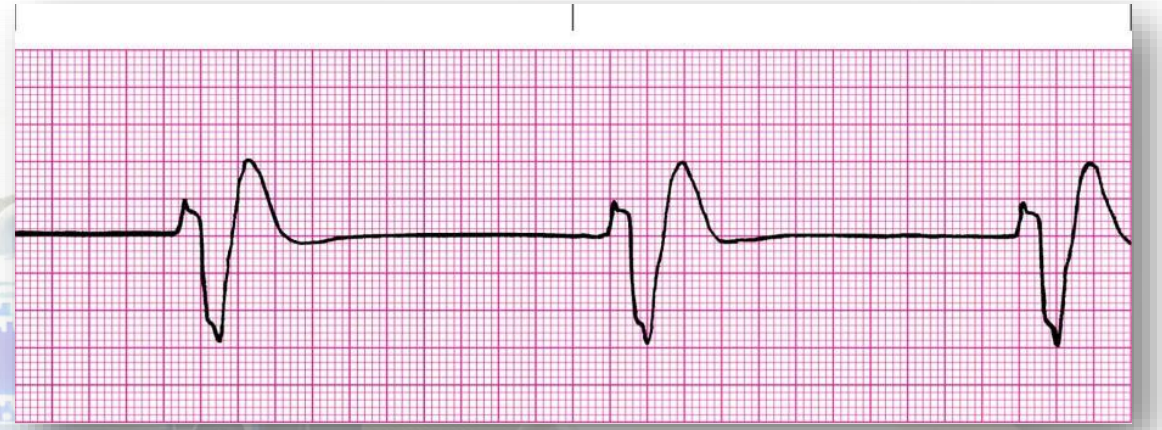
When a PVC occurs every third complex = **trigeminy**

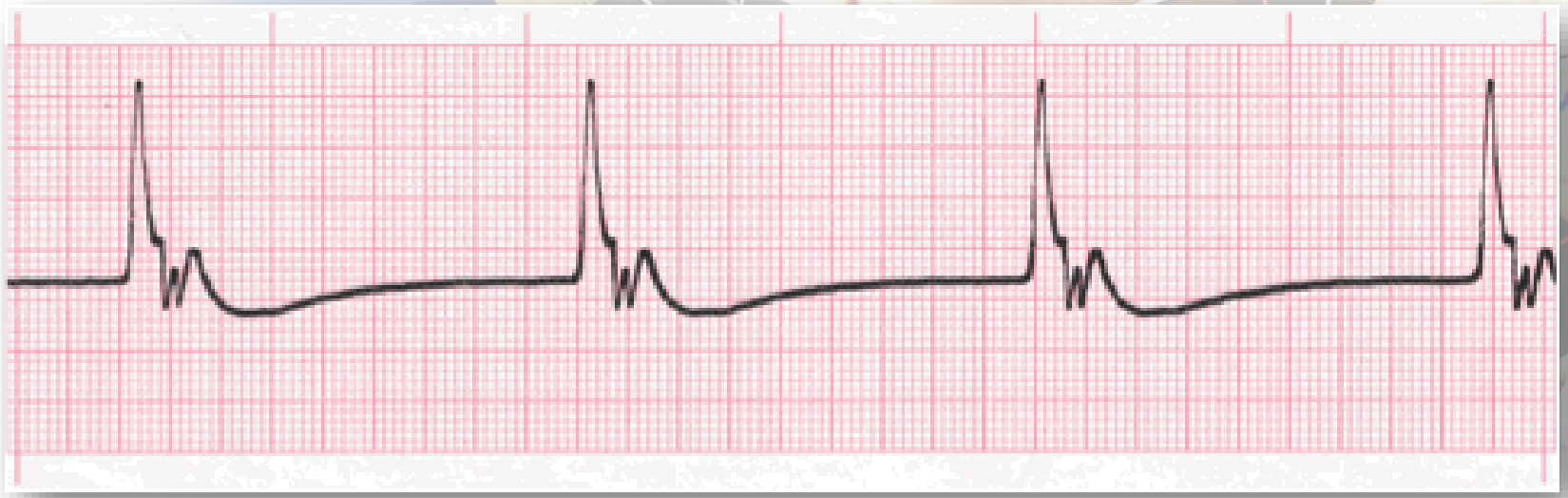
Trigeminy



Idioventricular rhythm

Rate	20 – 40 bpm
Rhythm	Usually regular
P Waves	None
PRI	None
QRS	> 0.12 s (> 120 ms), wide





Idioventricular rhythm

Etiology

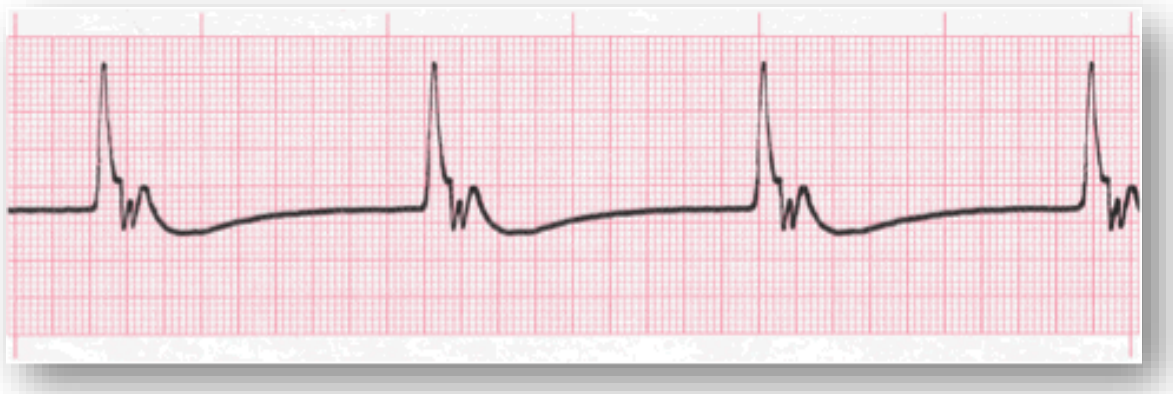
- Safety mechanism to prevent cardiac standstill
  - Produced by the ventricles
  - May or may not result in a palpable pulse
- Results from failure of atrial or junctional pacemaker sites or high-degree AV block

Clinical Significance

- Decreased cardiac output, possibly to life-threatening levels

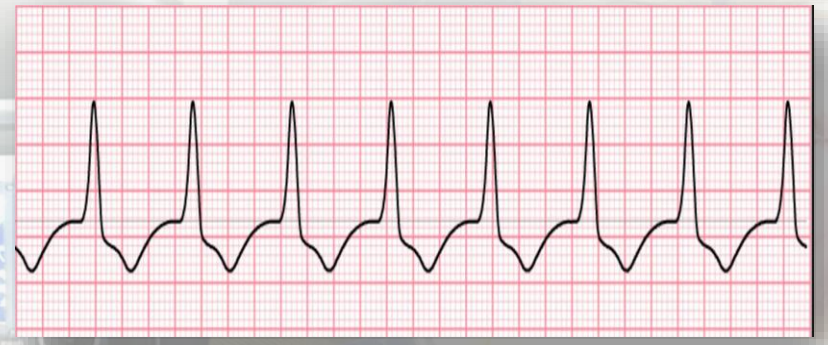
Treatment

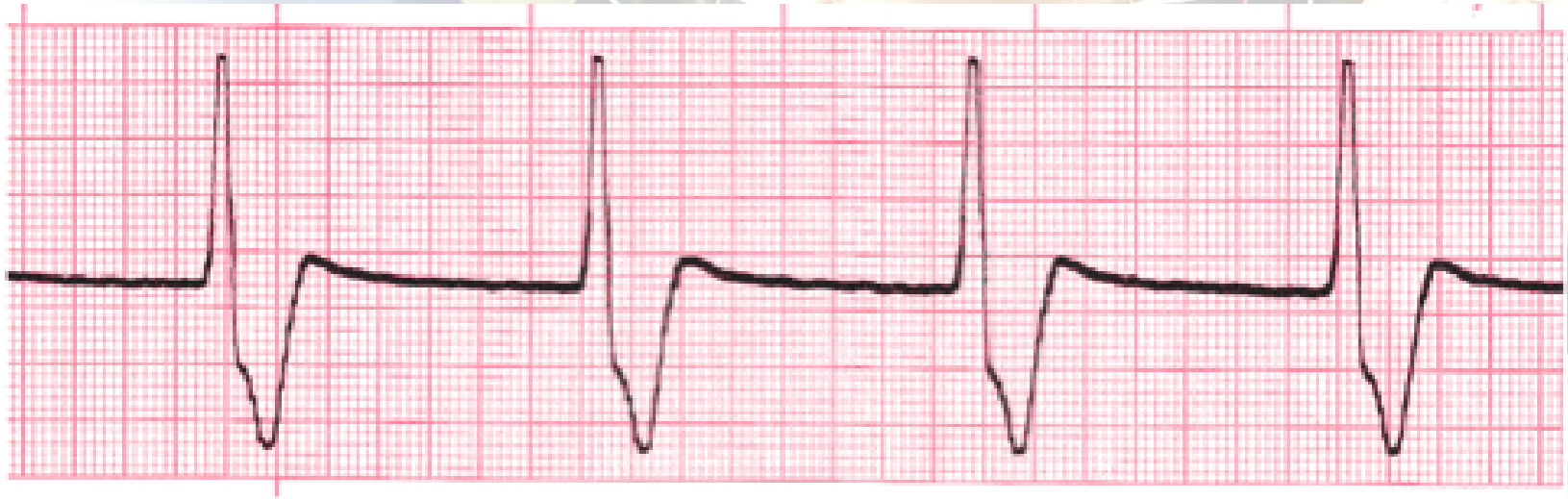
- Treatment is geared toward improving the cardiac output.



Accelerated Idioventricular

Rate	40 – 100 bpm
Rhythm	Regular
P Waves	None
PRI	None
QRS	> 0.12 s (> 120 ms), wide





Accelerated Idioventricular

Etiology

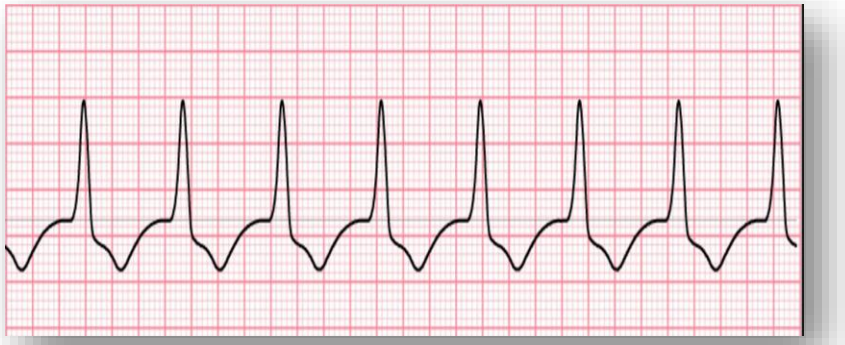
- Frequently occurs with reperfusion after acute STEMI
- Ventricular escape rhythm with a rate of 40–100 bpm

Clinical Significance

- May cause decreased cardiac output if the rate slows

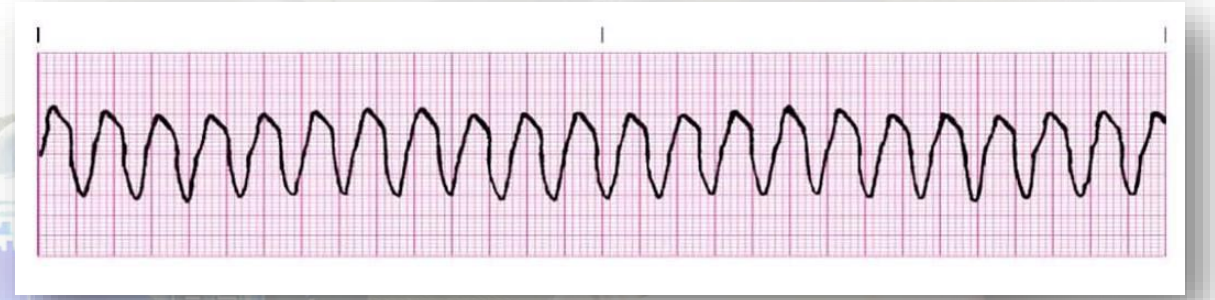
Treatment

- Does not usually require treatment unless the patient becomes hemodynamically unstable
- Primary goal is to treat the underlying issue
- Antidysrhythmic medications usually not indicated

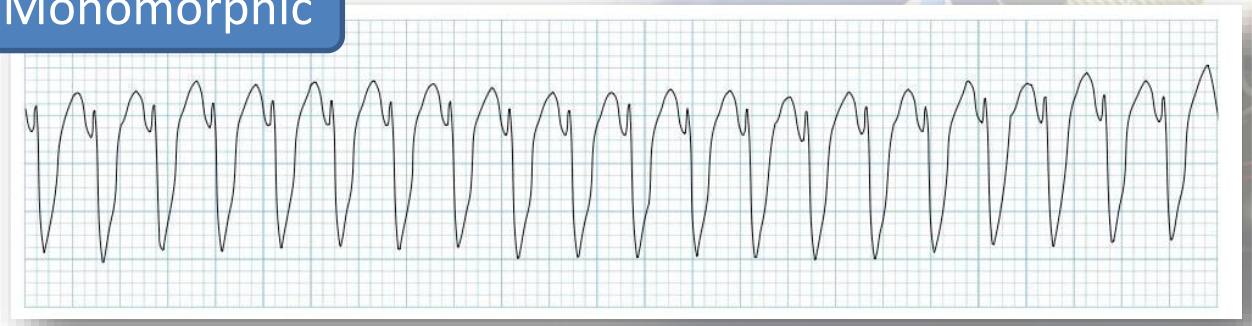


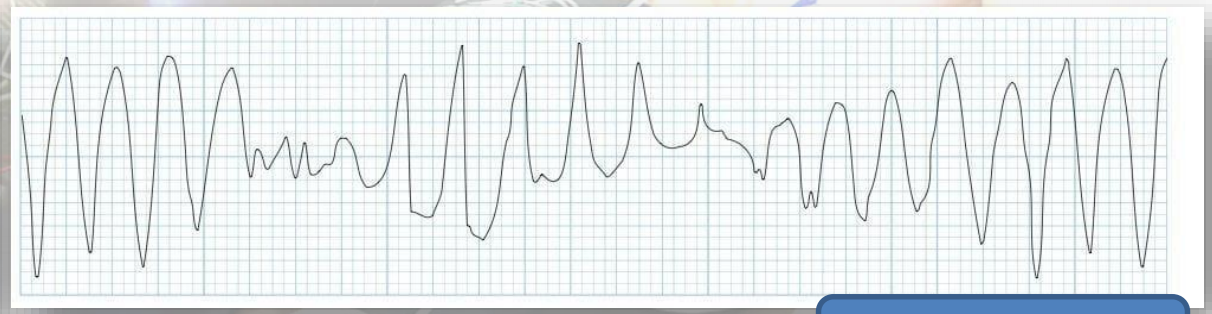
Ventricular Tachycardia

Rate	> 100 bpm
Rhythm	Regular
P Waves	None
PRI	None
QRS	> 0.12 s (> 120 ms), wide



Monomorphic





Polymorphic

Ventricular Tachycardia

Etiology

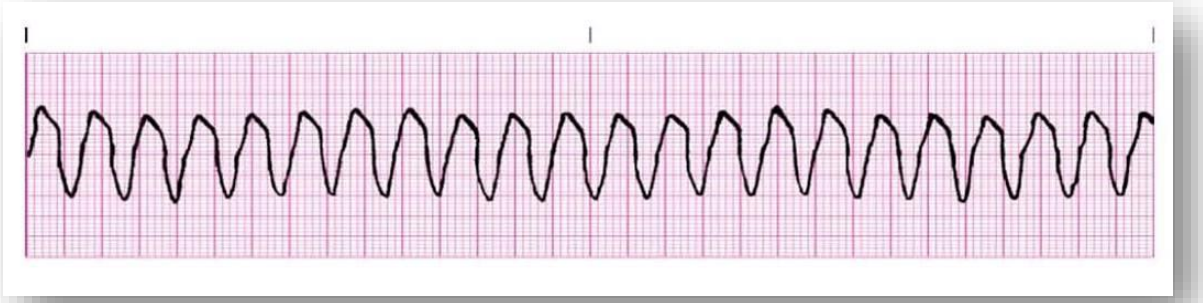
- Causes include myocardial ischemia, increased sympathetic tone, hypoxia, idiopathic causes, acid–base disturbances, or electrolyte imbalances
- VT may appear monomorphic or polymorphic

Clinical Significance

- Decreased cardiac output, possibly to life-threatening levels
- May deteriorate into ventricular fibrillation

Treatment

- Perfusing
- Nonperfusing



Note

- If rhythm ceases before 30 seconds it is referred to as a run of Vtach
- If rhythm persists for > 30 seconds it is referred to as sustained VTach