CARDIAC RHYTHM AND CONDUCT ABNORMALITIES

LECTURER:

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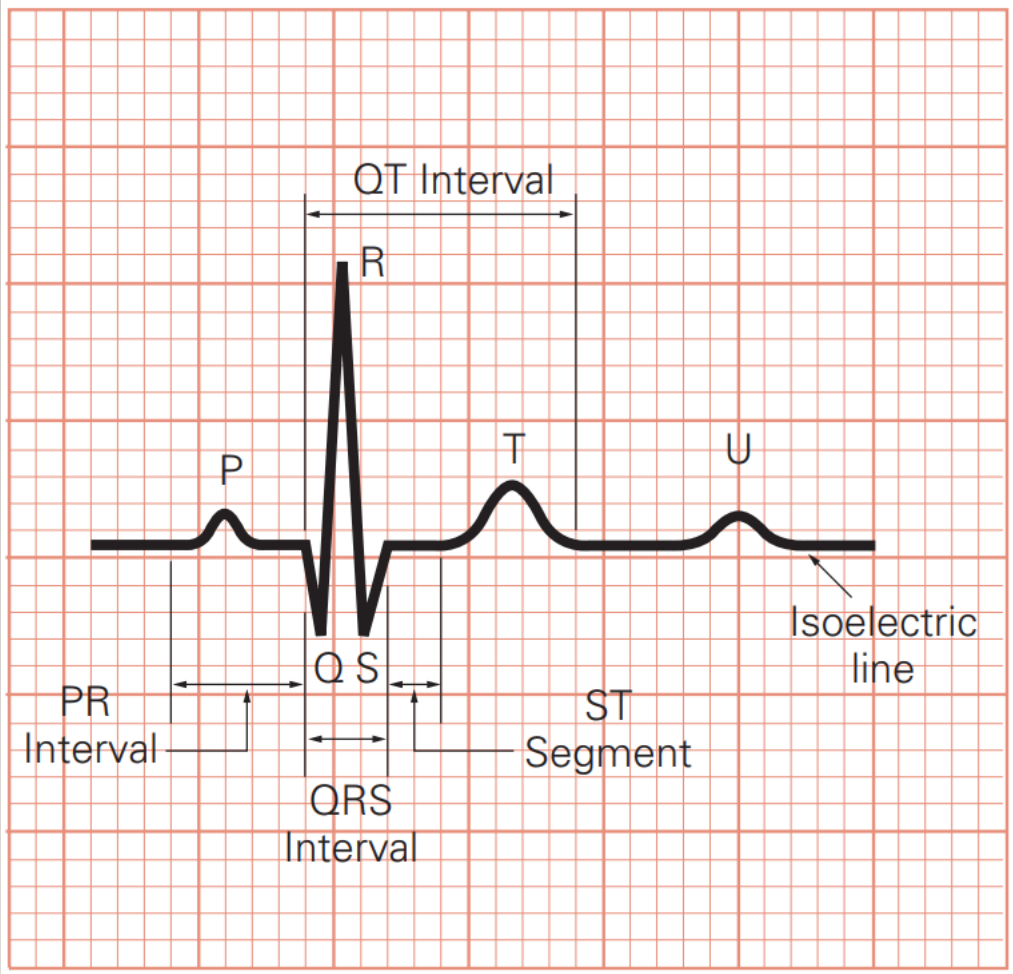
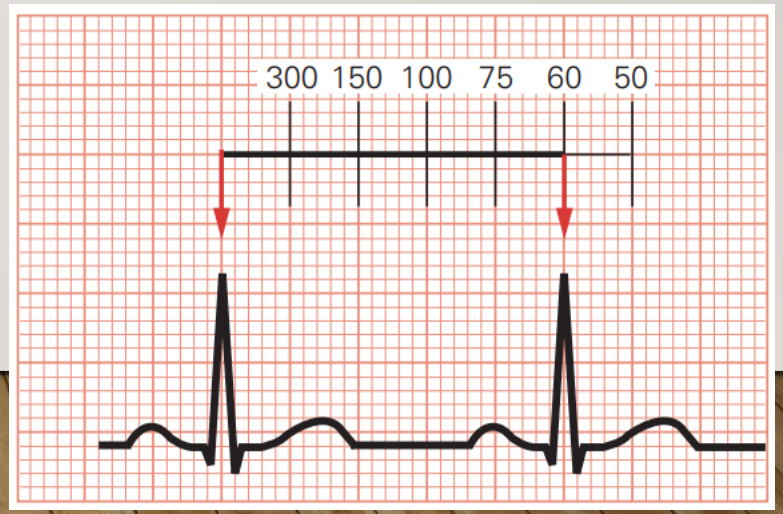
ANALYZING AN ECG RHYTHM



- **Rate**
- The bpm is commonly the ventricular rate.
- If atrial and ventricular rates differ, as in a third-degree block, measure both rates.
- Normal: 60–100 bpm
- Slow (bradycardia): <60 bpm
- Fast (tachycardia): >100 bpm



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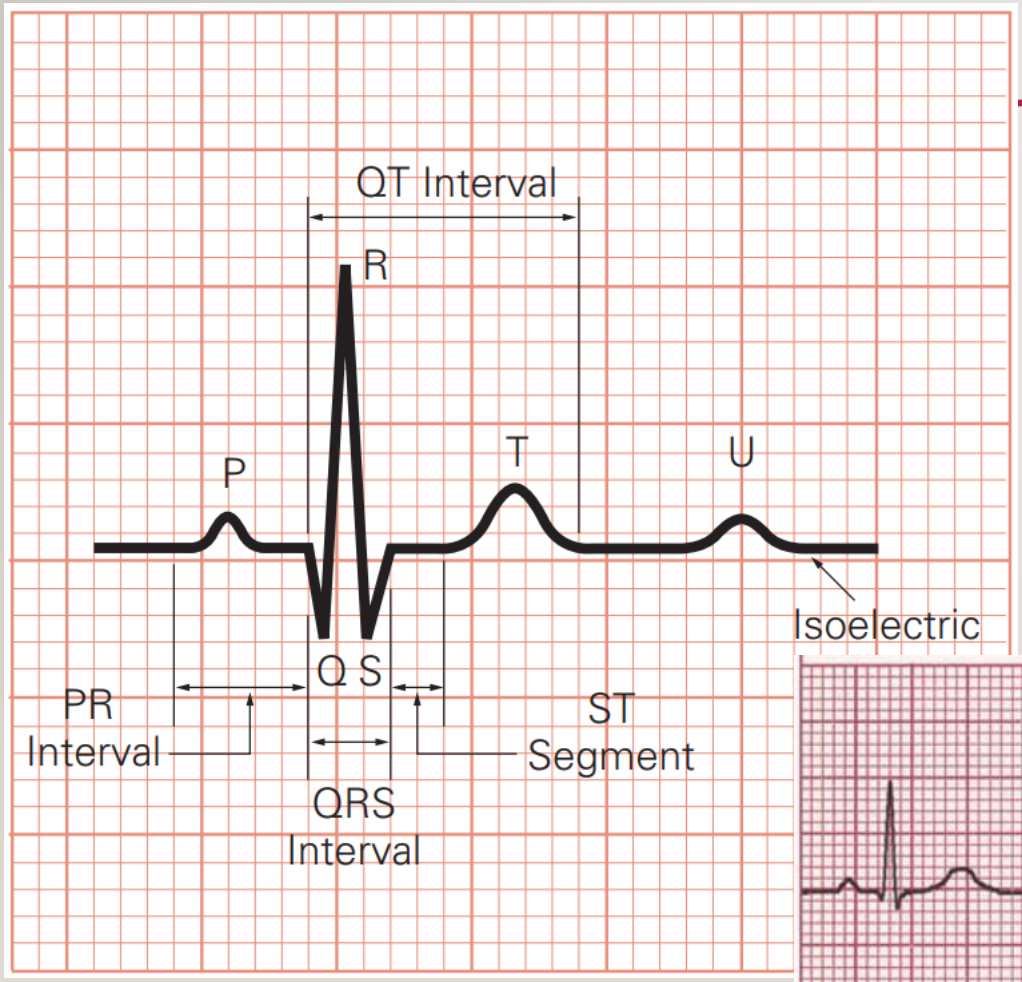
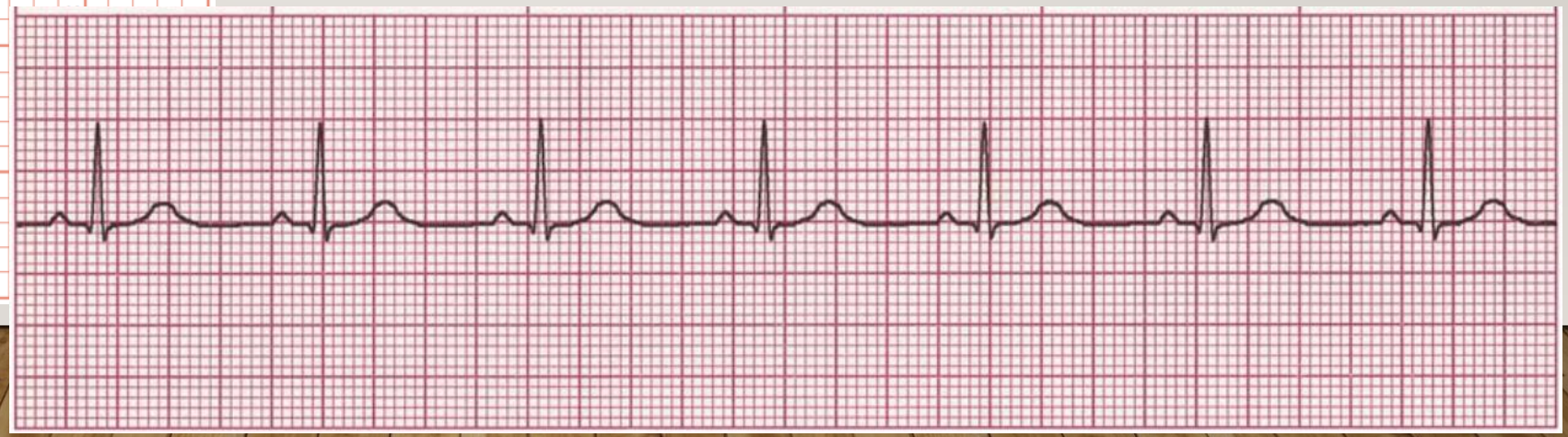


- **Regularity**
- Measure R-R intervals and P-P intervals.
- Regular: Consistent intervals
- Regularly irregular: Repeating pattern
- Irregular: No pattern



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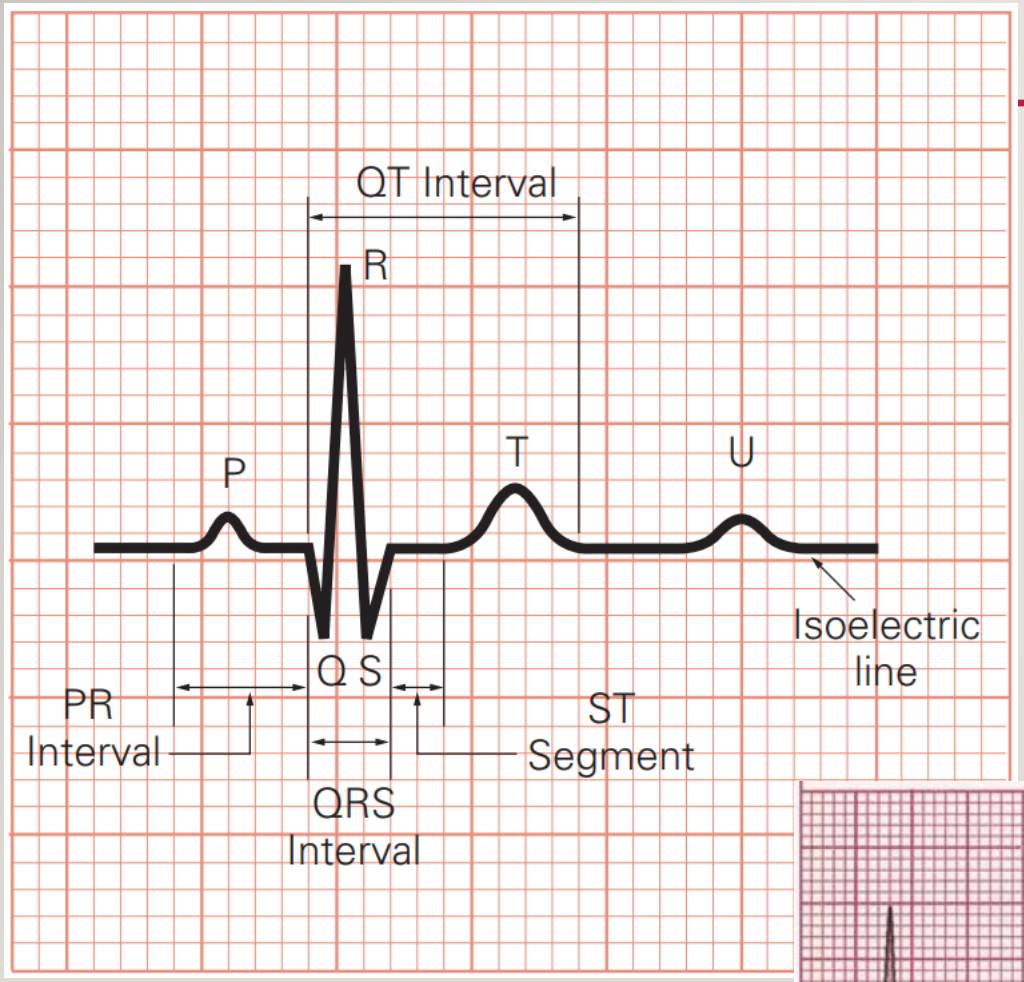
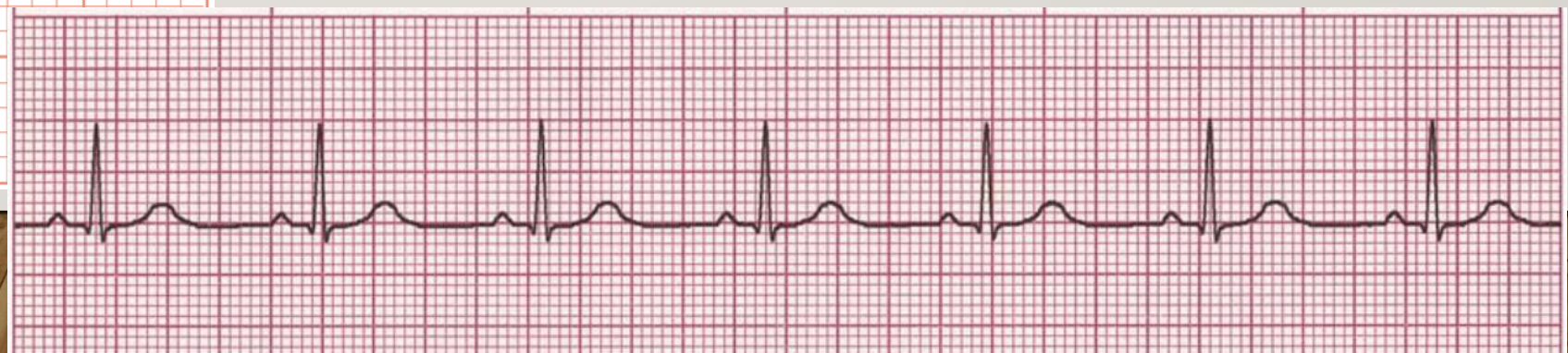


- **P waves**
- If present: Same in size, shape, position?
- Does each QRS have a P wave?
- Normal: Upright (positive) and uniform
- Inverted: Negative
- Notched: P'
- None: Rhythm is junctional or ventricular



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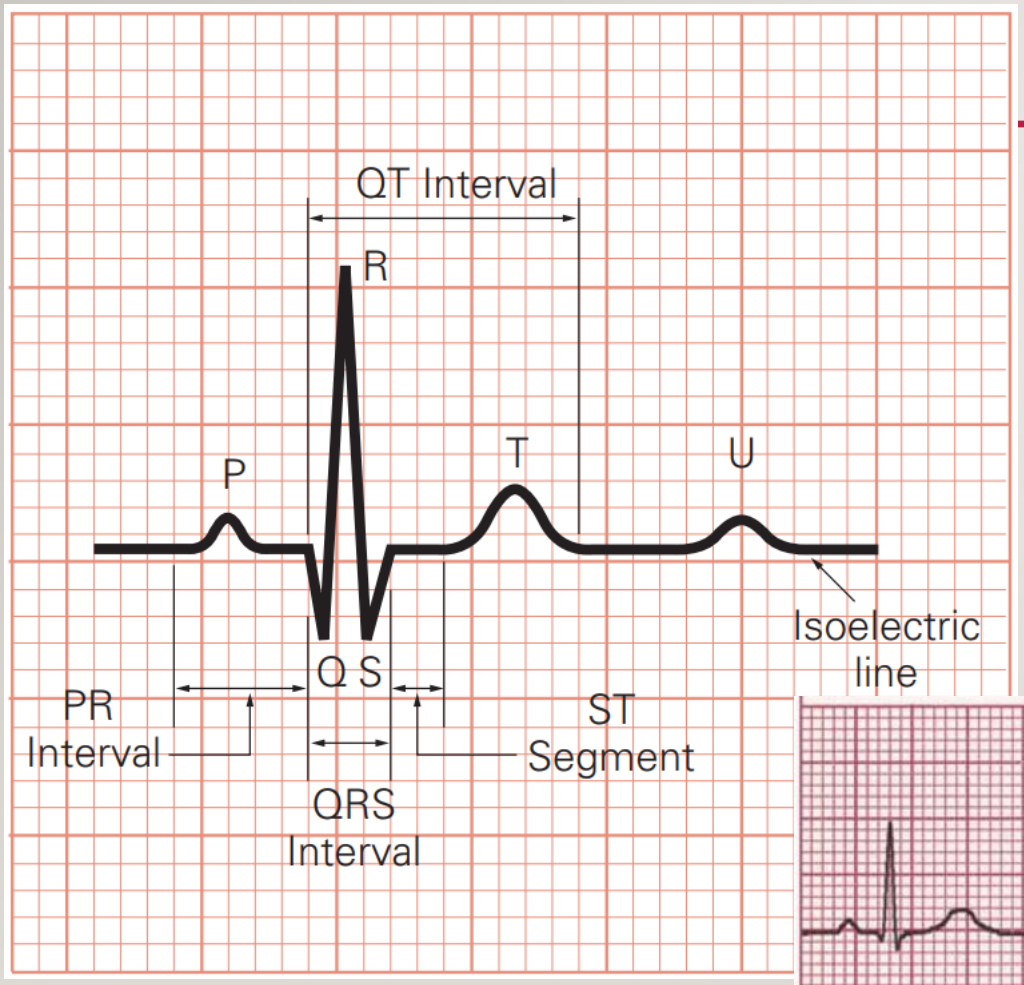
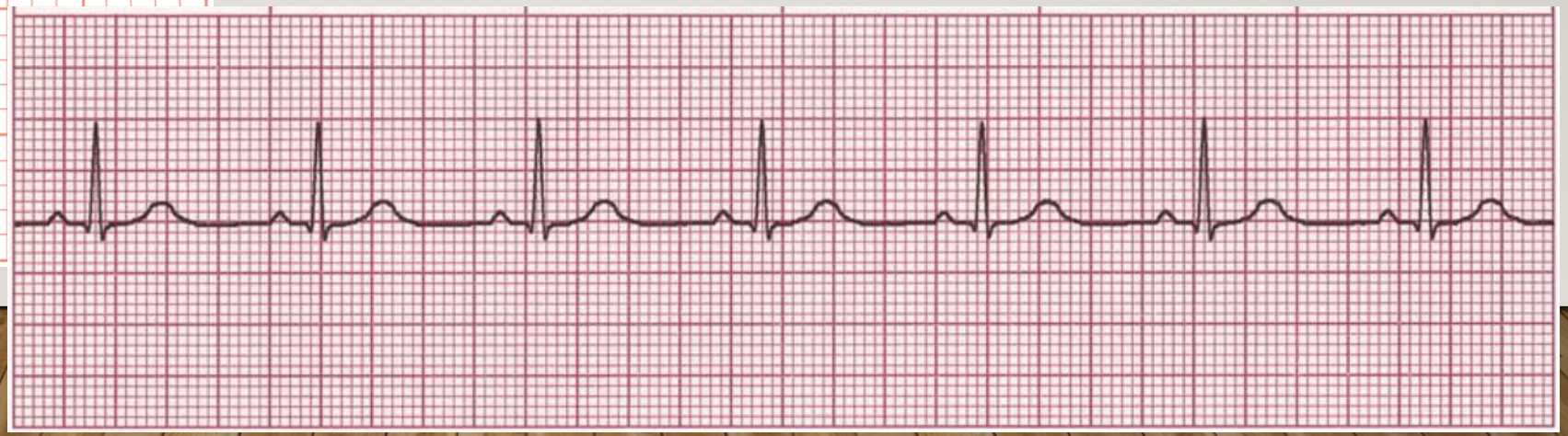


- **PR interval**
- Constant: Intervals are the same.
- Variable: Intervals differ.
- Normal: 0.12–0.20 sec and constant



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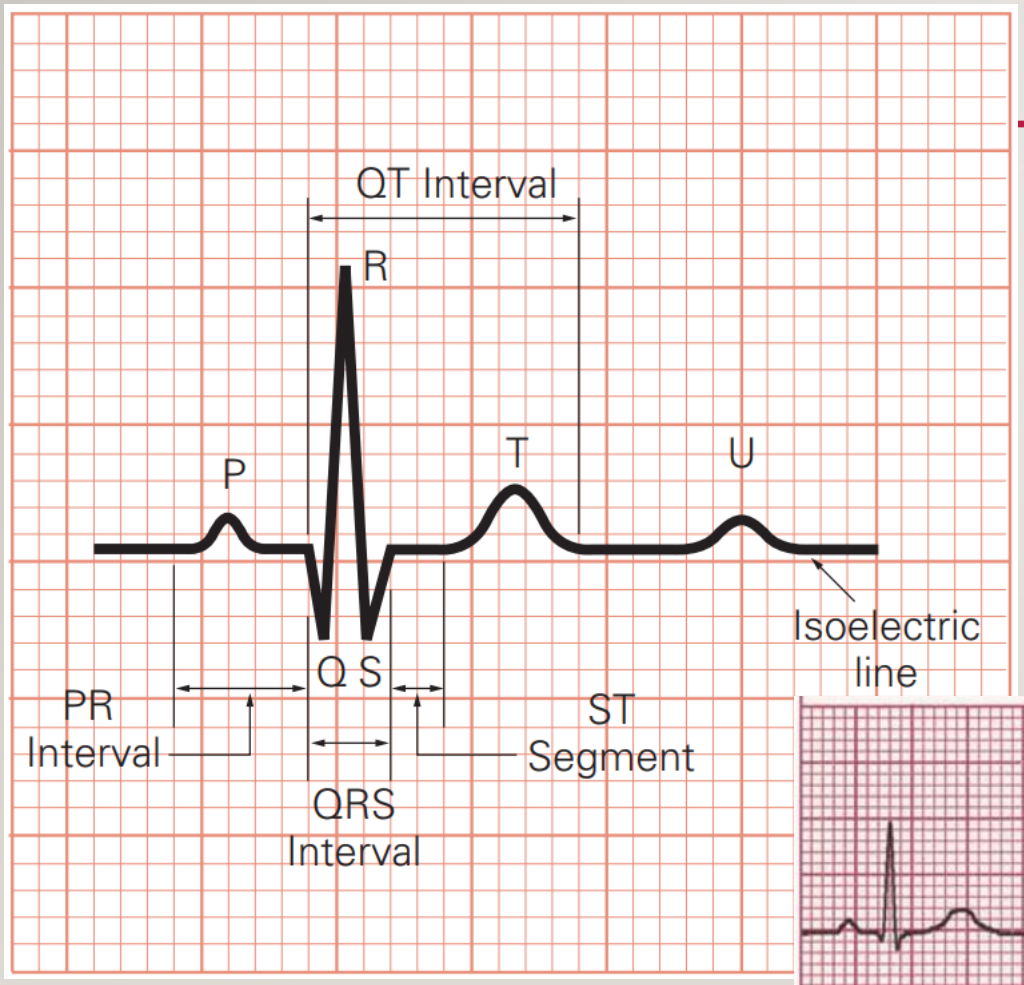
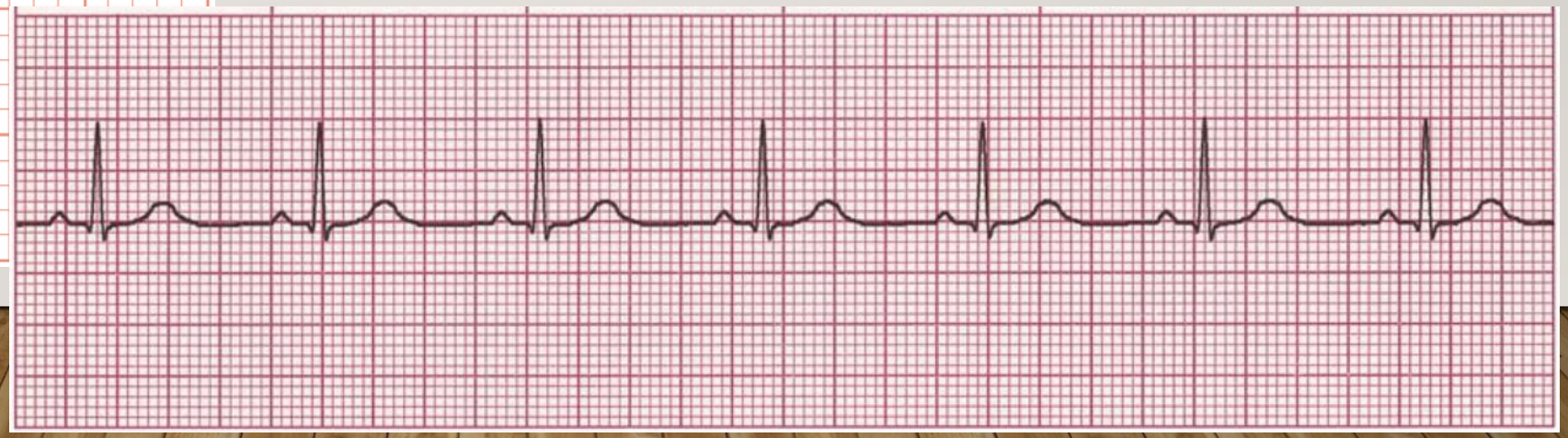


- **QRS interval**
- Normal: 0.06–0.10 sec
- Wide: >0.10 sec
- None: Absent



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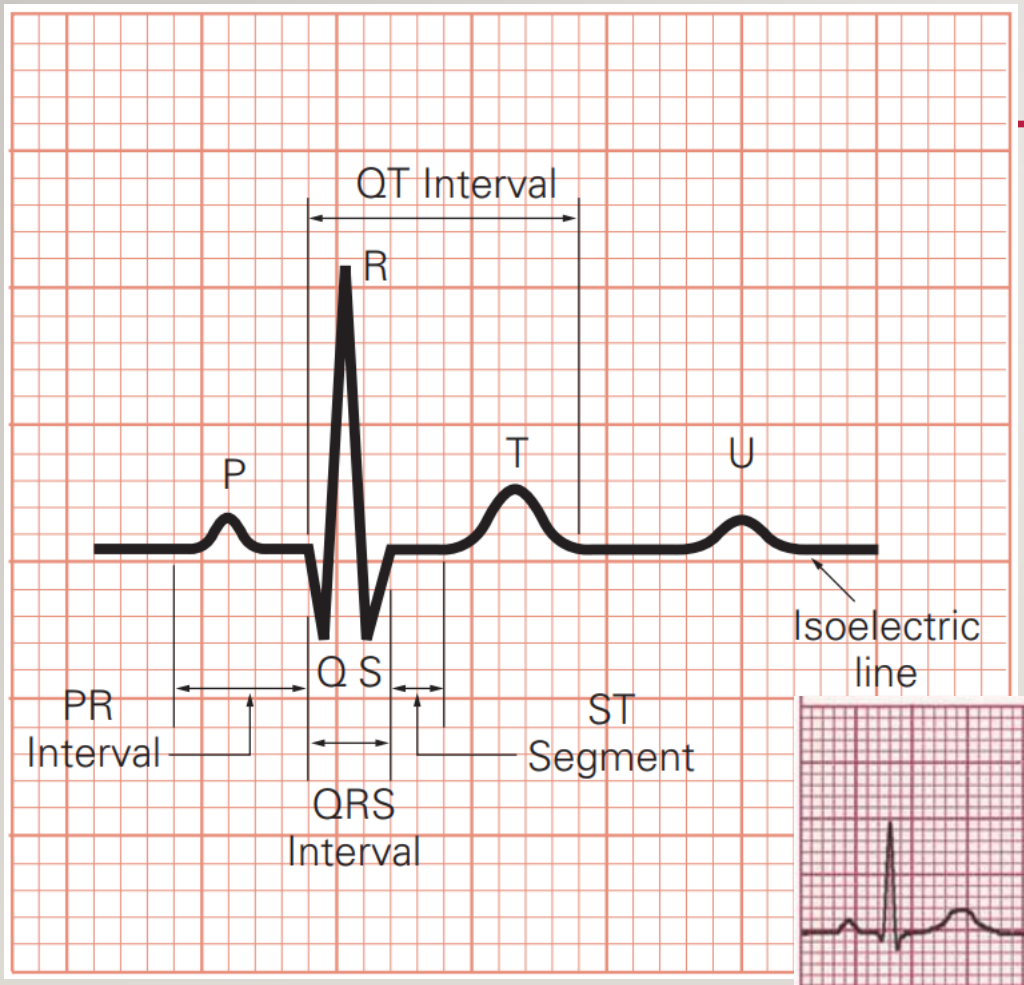
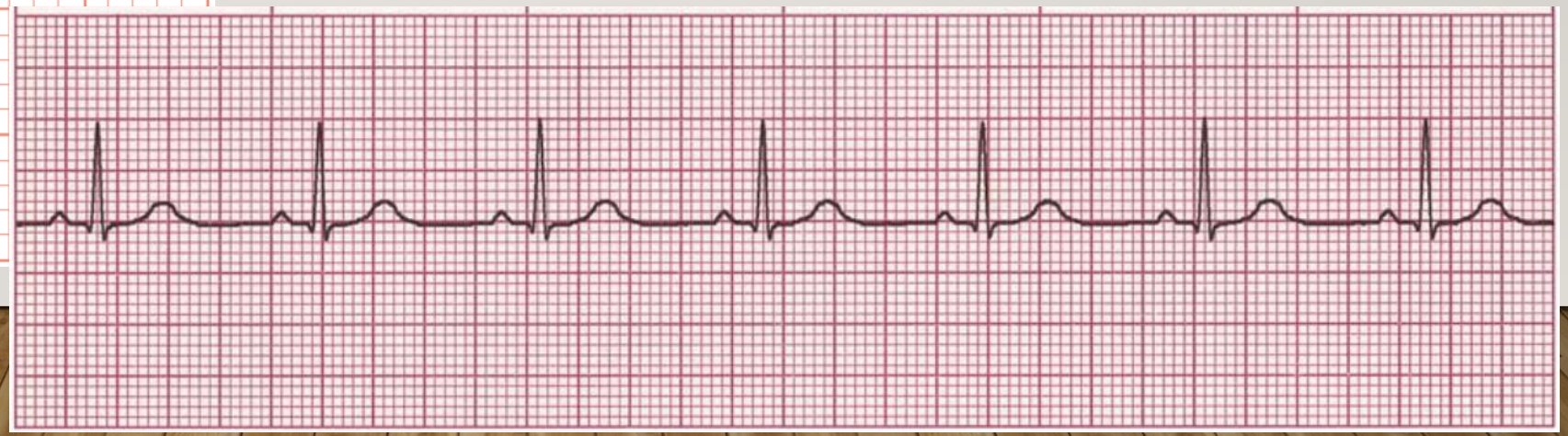


- **QT interval**
- Beginning of QRS to end of T wave
- Varies with HR.
- Normal: Less than half the R-R interval



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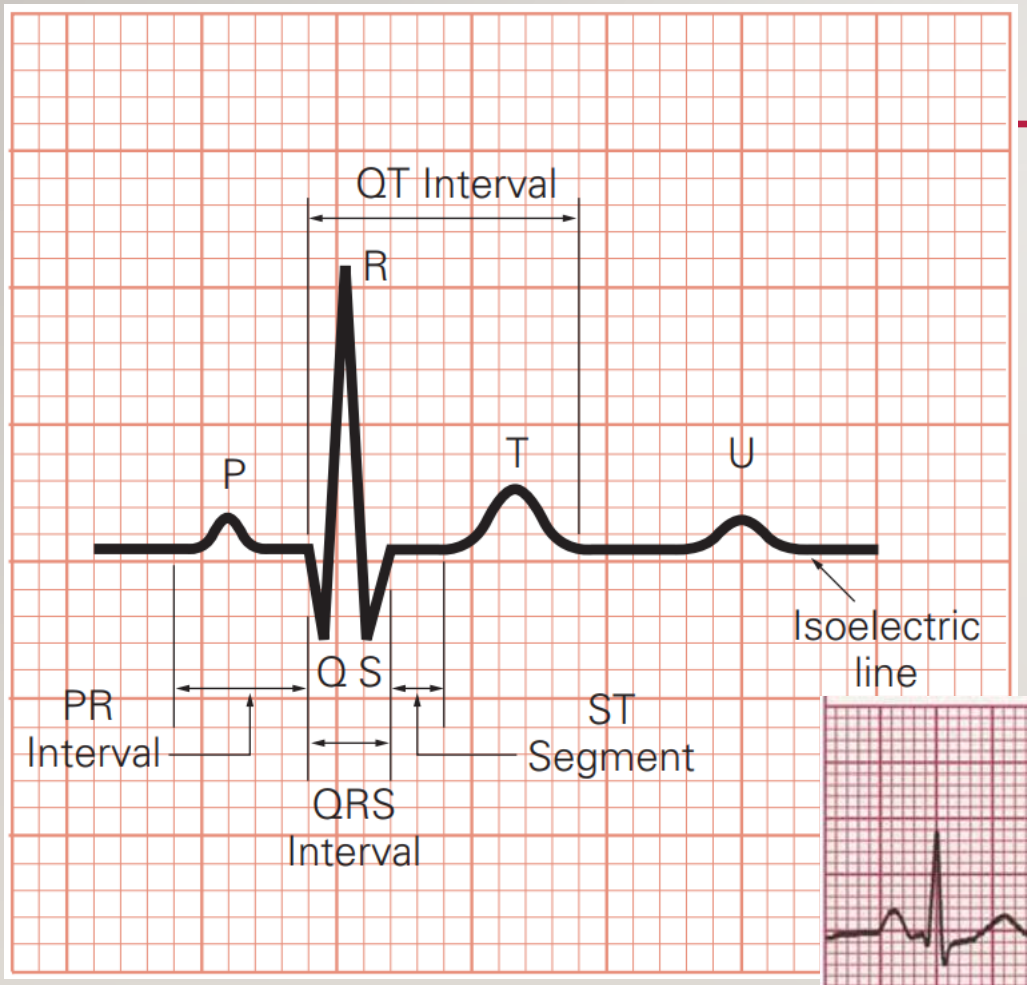
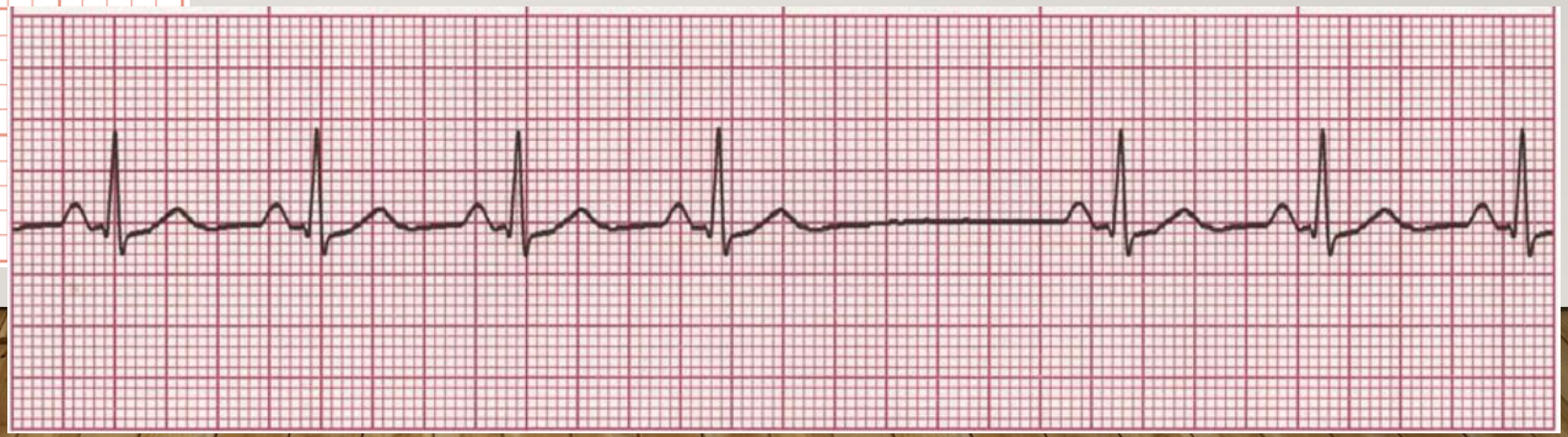


- **Dropped beats**
- Occur in AV blocks.
- Occur in sinus arrest.



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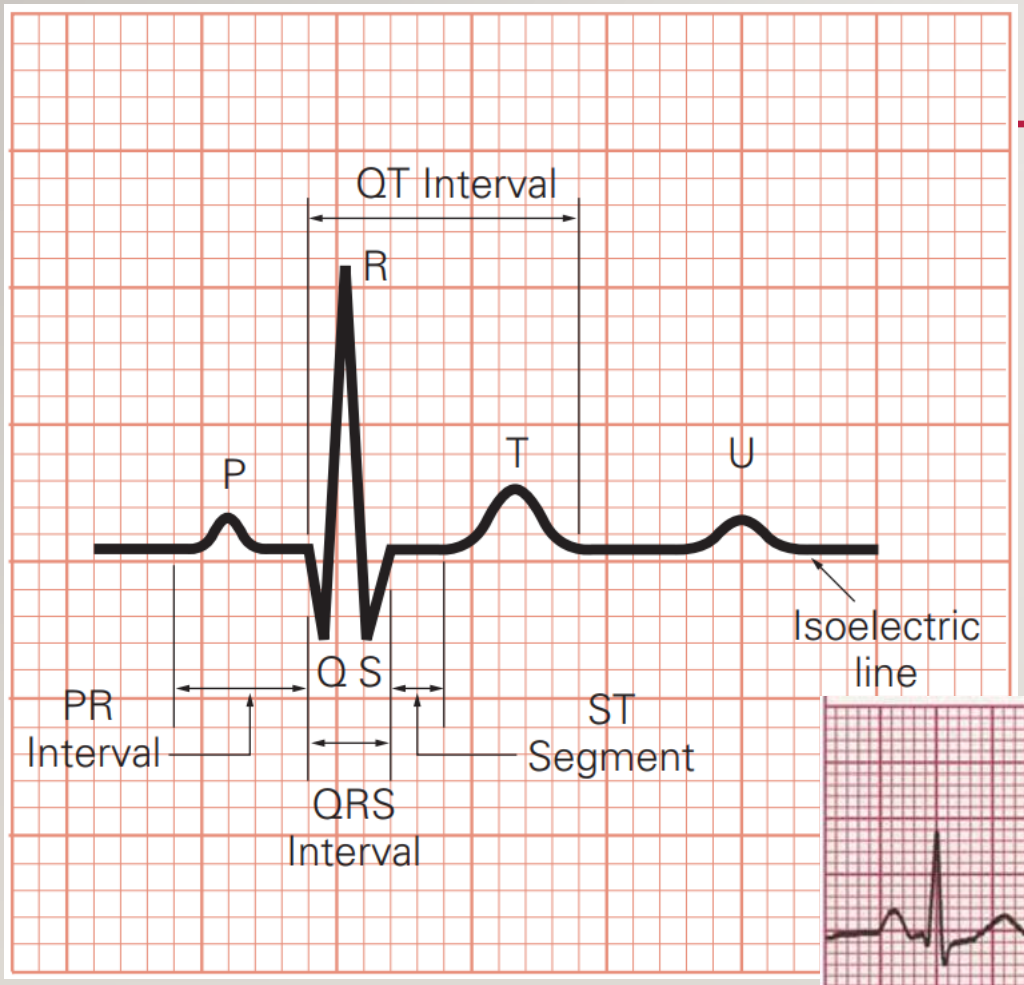
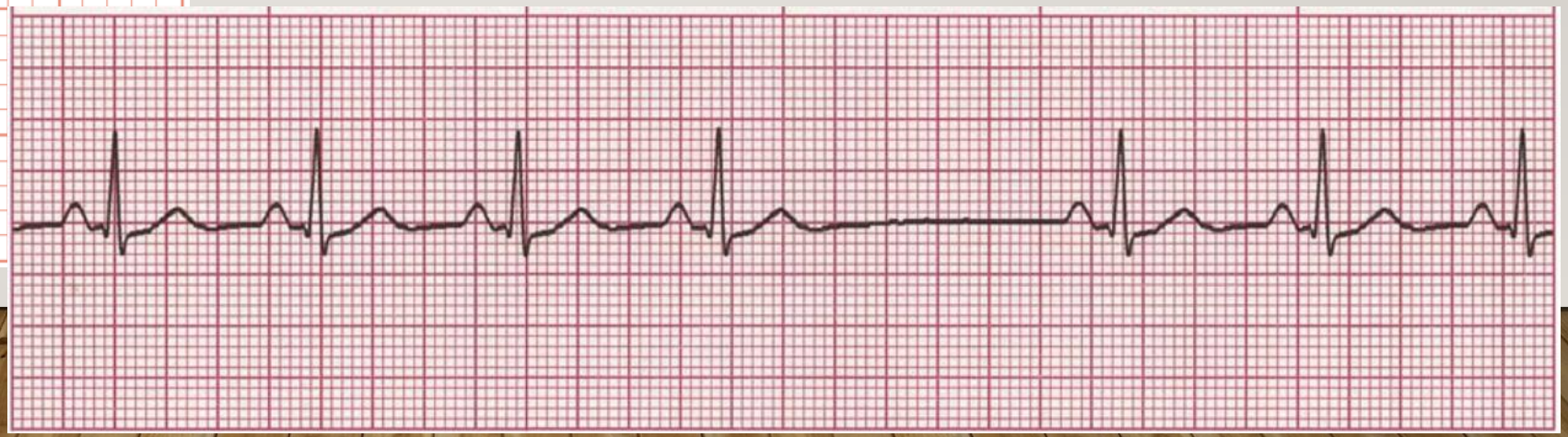


- **Pause**
- Compensatory: Complete pause following a premature atrial, junctional, or ventricular contraction (PAC, PJC, or PVC)
- Noncompensatory: Incomplete pause following a PAC, PJC, or PVC



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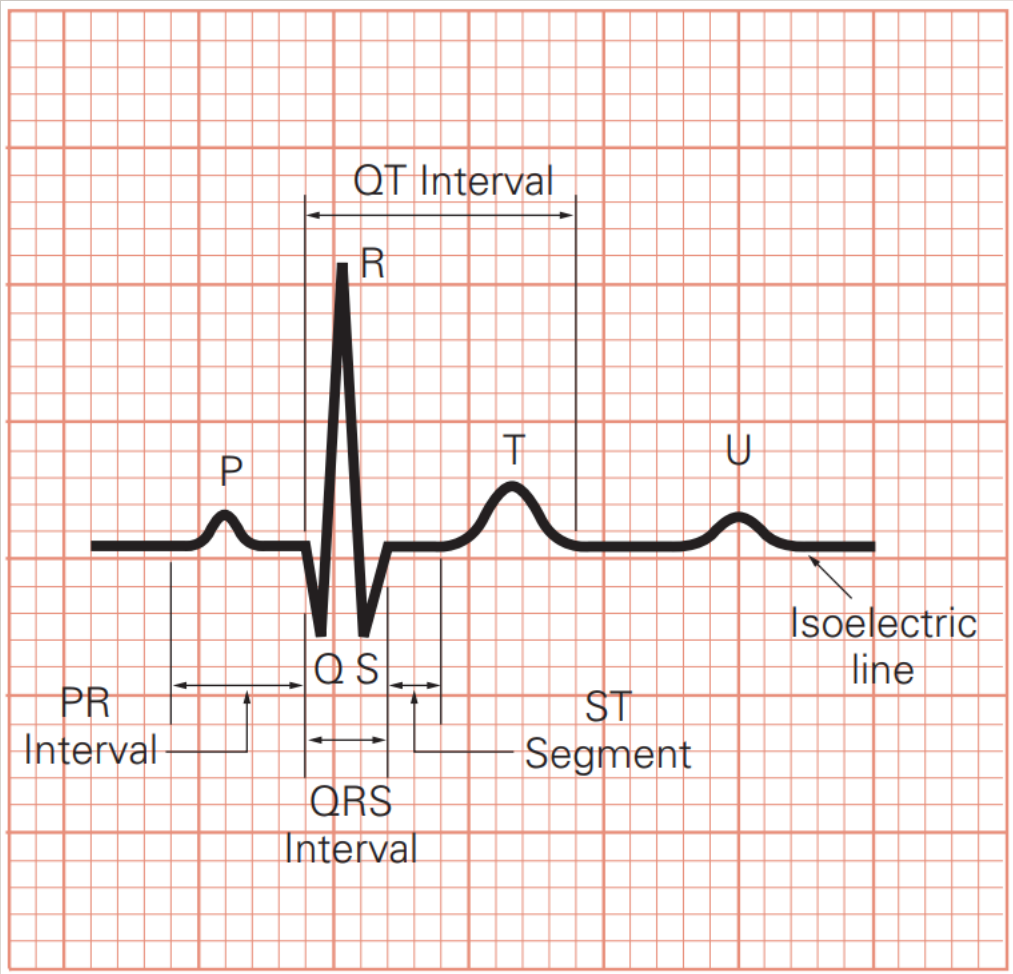
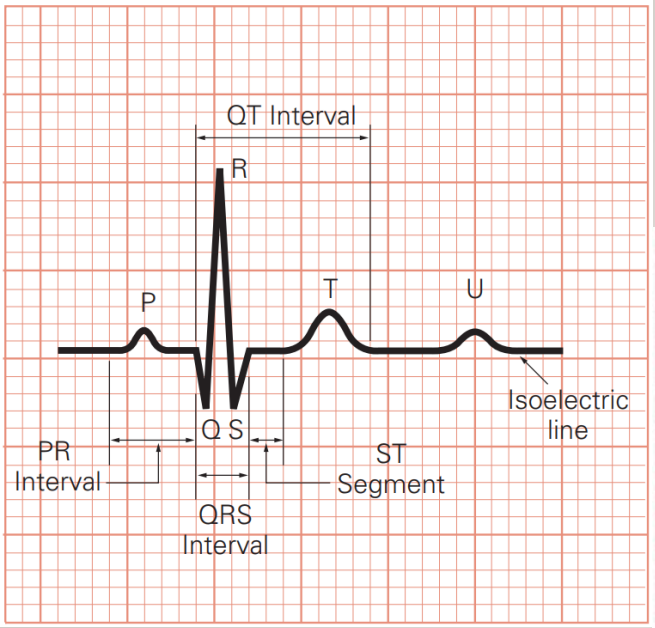
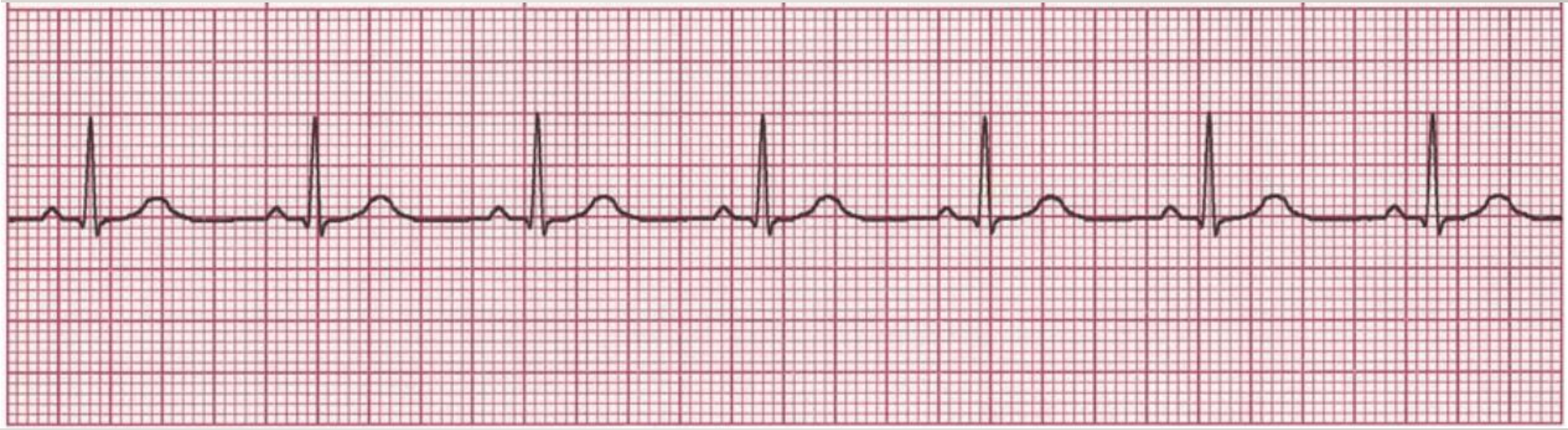


- **QRS Complex grouping**
- Bigeminy: Repeating pattern of normal complex followed by a premature complex
- Trigeminy: Repeating pattern of 2 normal complexes followed by a premature complex
- Quadrigeminy: Repeating pattern of three normal complexes followed by a premature complex
- Couplets: 2 consecutive premature complexes
- Triplets: 3 consecutive premature complexes

NORMAL SINUS RHYTHM

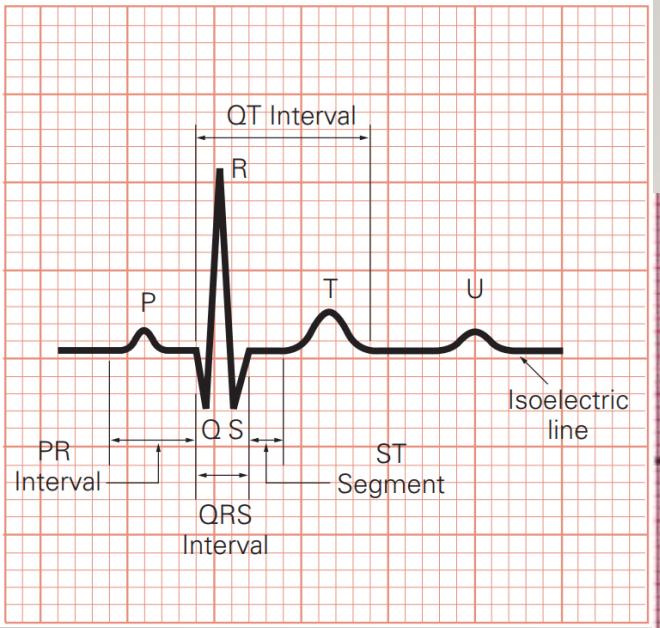


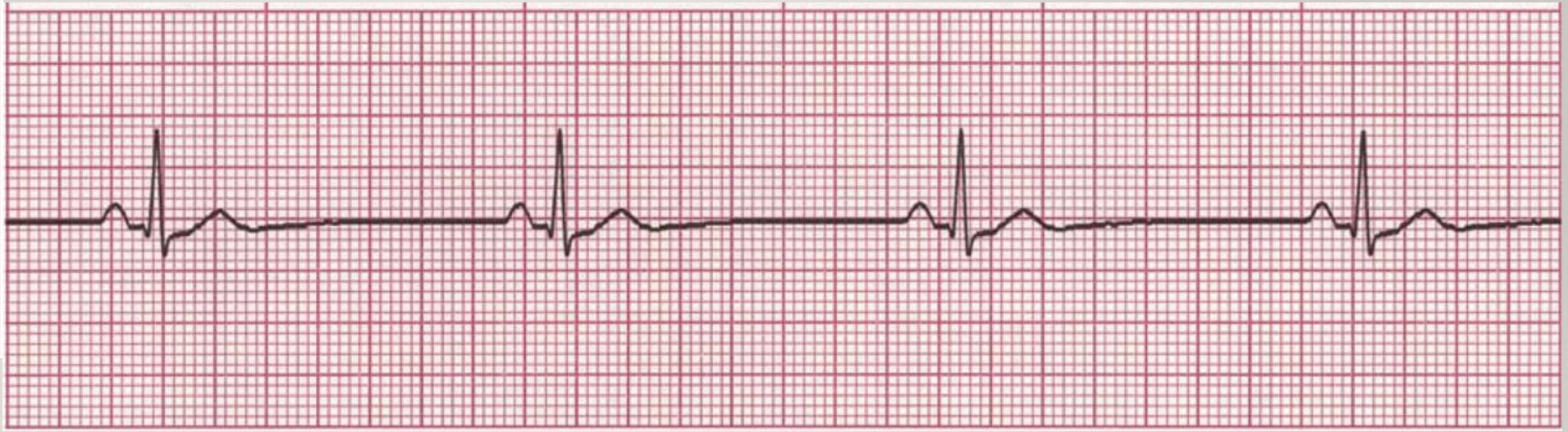


- Rate: Normal (60–100 bpm)
- Rhythm: Regular
- P Waves: Normal (upright and uniform)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- A normal ECG does not exclude heart disease.
- This rhythm is generated by the sinus node and its rate is within normal limits (60–100 bpm).

SINUS BRADYCARDIA

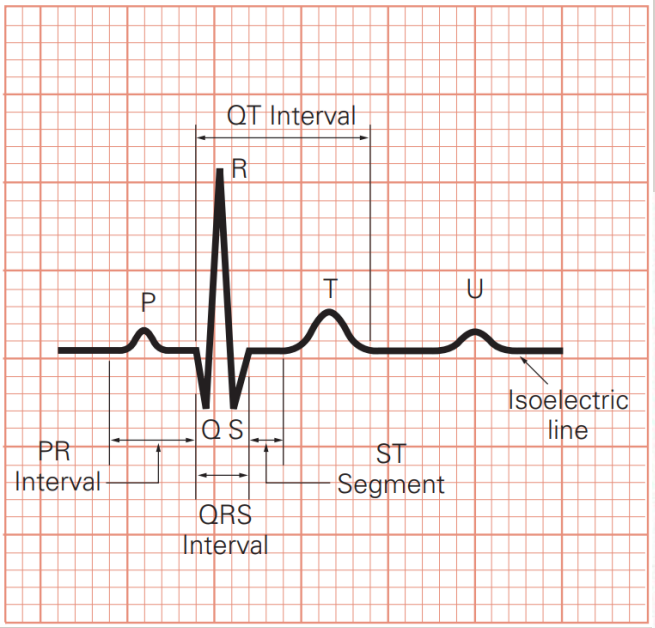


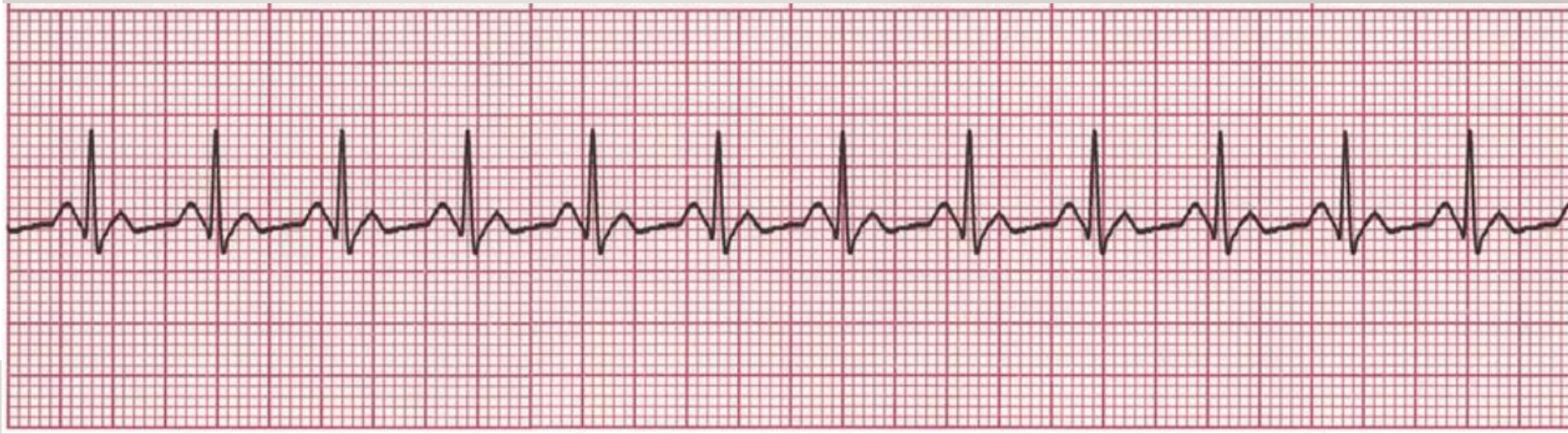


- Rate: Slow (<60 bpm)
- Rhythm: Regular
- P Waves: Normal (upright and uniform)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- The SA node discharges more slowly than in NSR.
- Sinus bradycardia is normal in athletes and during sleep. In acute MI, it may be protective and beneficial or the slow rate may compromise cardiac output.
- Certain medications, such as beta blockers, may also cause sinus bradycardia.

SINUS TACHYCARDIA

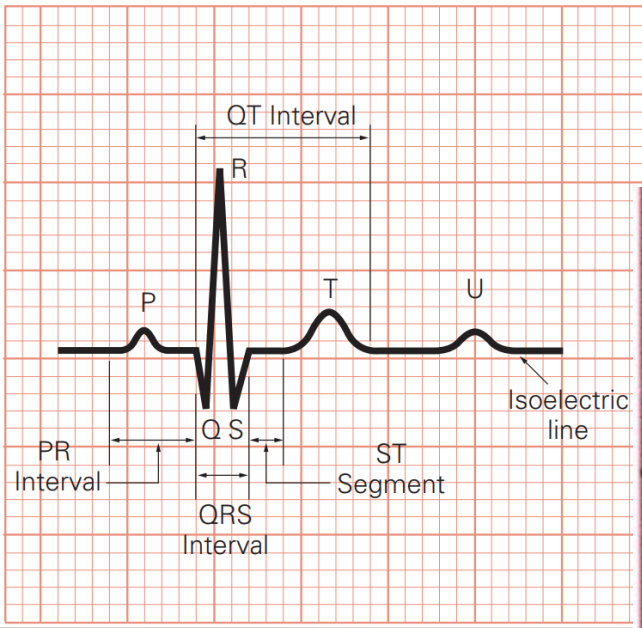


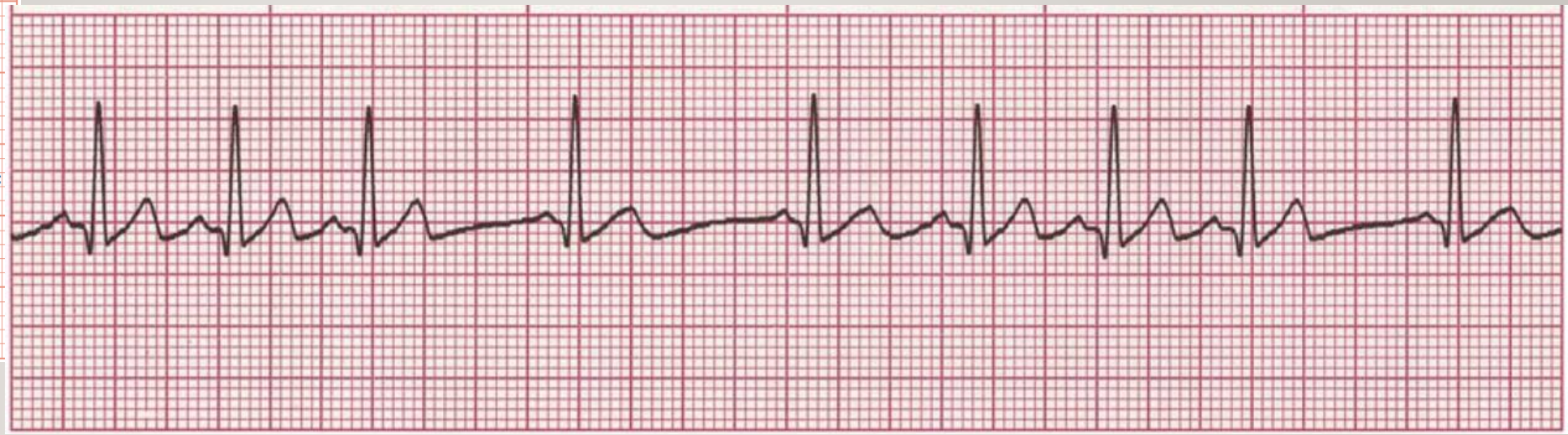


- Rate: Fast (>100 bpm)
- Rhythm: Regular
- P Waves: Normal (upright and uniform)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- The SA node discharges more frequently than in NSR.
- Sinus tachycardia may be caused by exercise, anxiety, fever, hypoxemia, hypovolemia, or cardiac failure.

SINUS ARRHYTHMIA

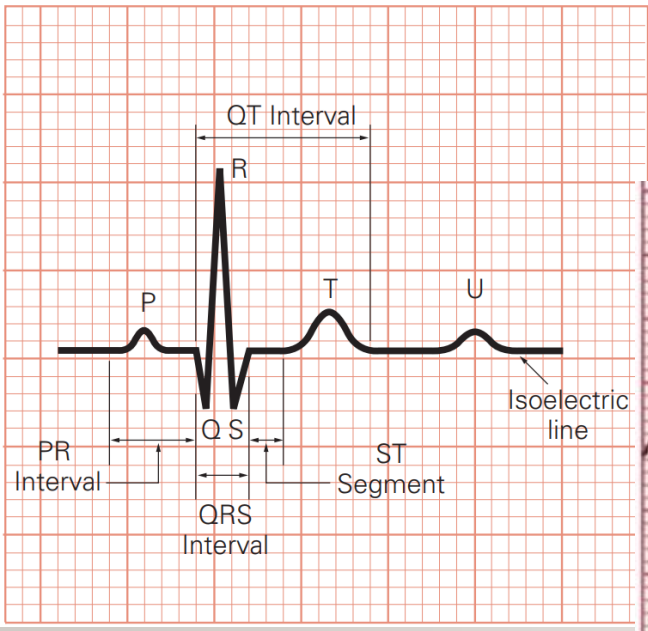


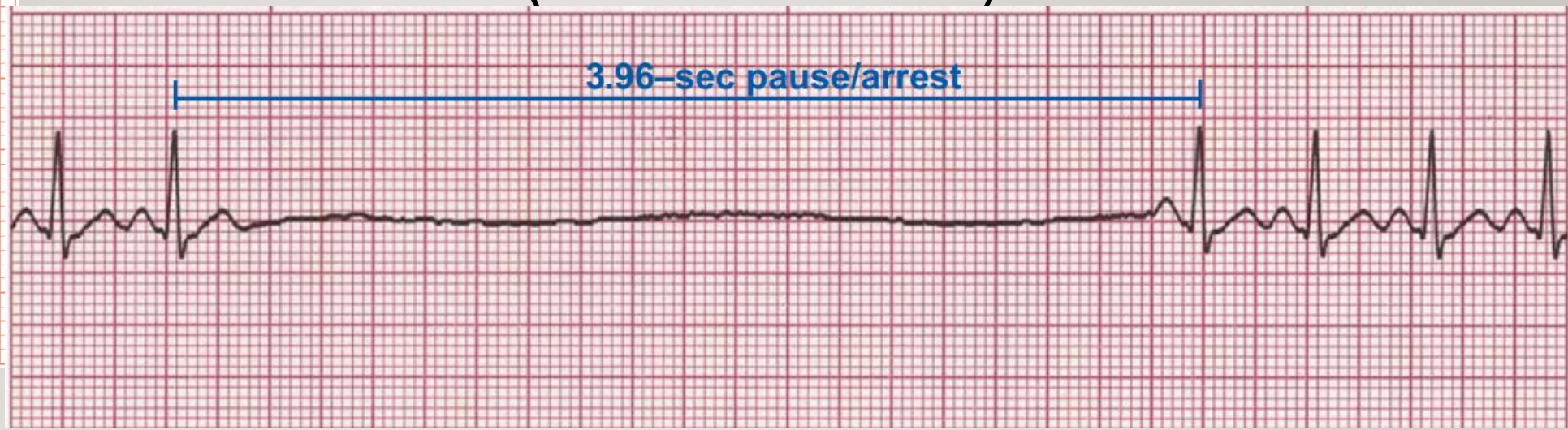


- Rate: Usually normal (60–100 bpm); frequently increases with inspiration and decreases with expiration; may be <60 bpm
- Rhythm: Irregular; varies with respiration; increases with inspiration; difference between shortest R-R and longest R-R intervals is >0.12 sec
- P Waves: Normal (upright and uniform)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- The SA node discharges irregularly.
- The R-R interval is irregular
- The pacing rate of the SA node varies with respiration, especially in children and elderly people.

SINUS PAUSE (SINUS ARREST)

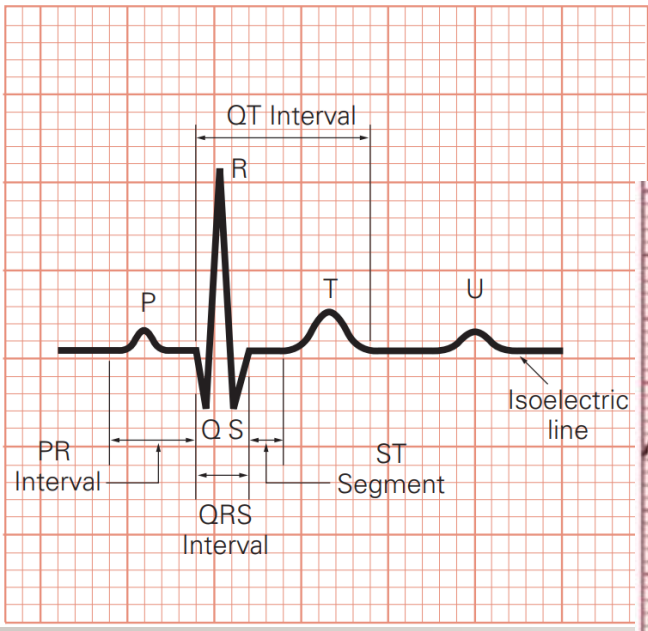


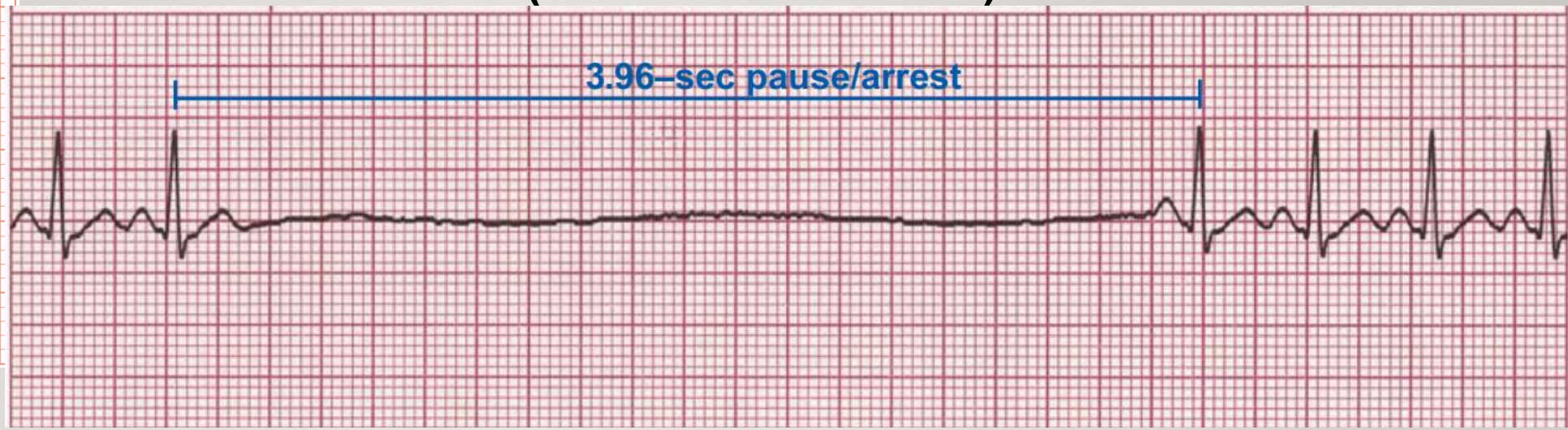


- Rate: Normal to slow; determined by duration and frequency of sinus pause (arrest)
- Rhythm: Irregular whenever a pause (arrest) occurs
- P Waves: Normal (upright and uniform) except in areas of pause (arrest)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- The SA node fails to discharge and then resumes.
- Electrical activity resumes either when the SA node resets itself or when a lower latent pacemaker begins to discharge.
- The pause (arrest) time interval is not a multiple of the normal P-P interval
- Cardiac output may decrease, causing syncope or dizziness.

SINUS PAUSE (SINUS ARREST)





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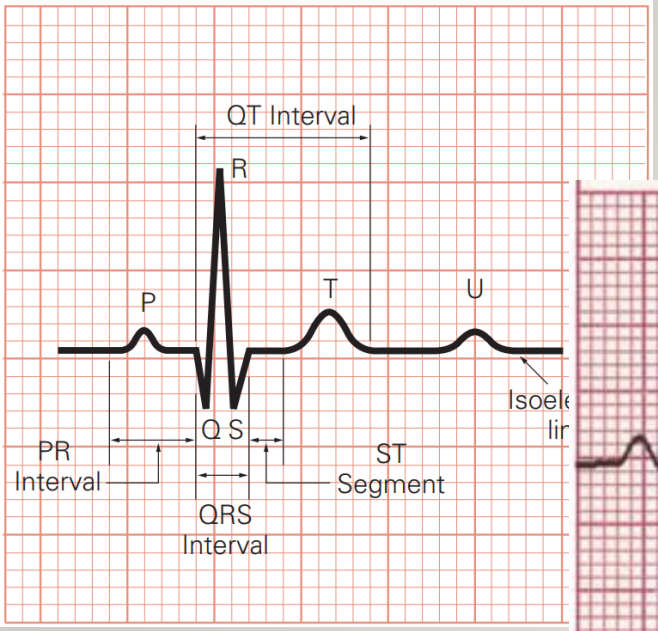
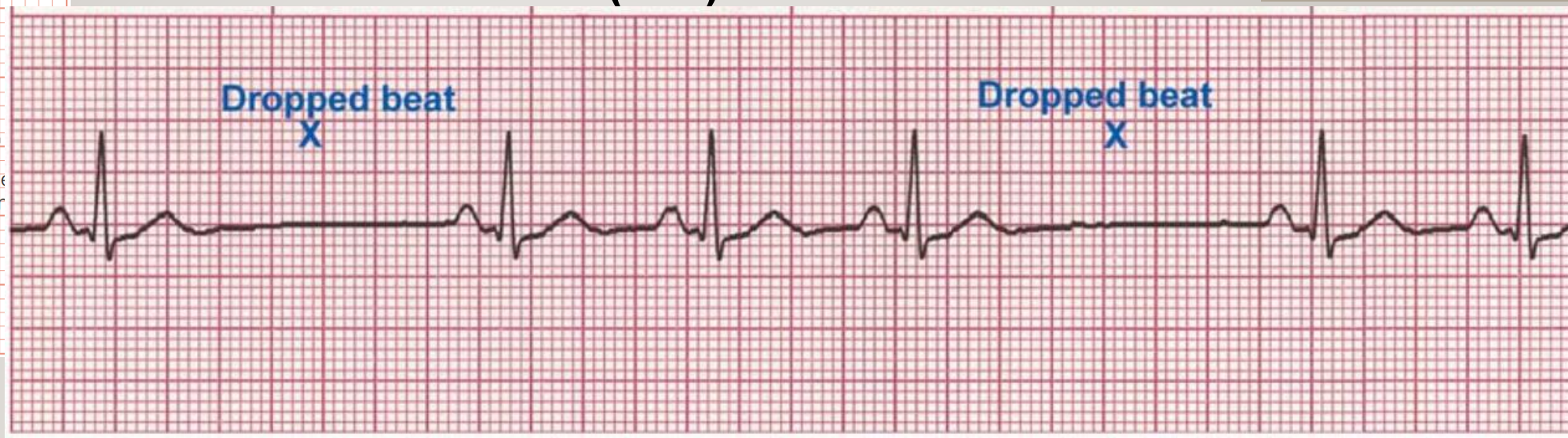
SINOATRIAL (SA) BLOCK

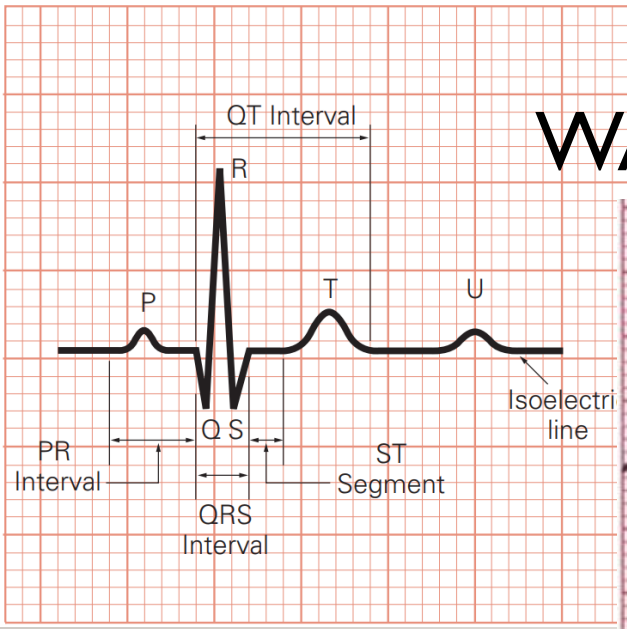


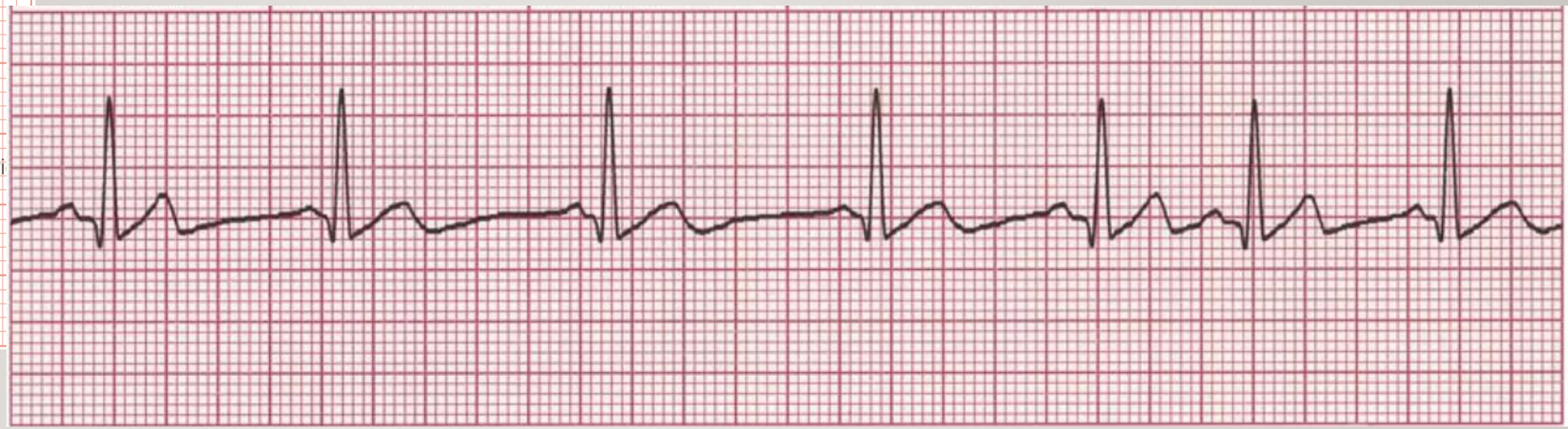


- Rate: Normal to slow; determined by duration and frequency of SA block
- Rhythm: Irregular whenever an SA block occurs
- P Waves: Normal (upright and uniform) except in areas of dropped beats
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- The block occurs in some multiple of the P-P interval.
- After the dropped beat, cycles continue on time.
- Cardiac output may decrease, causing syncope or dizziness.

WANDERING ATRIAL PACEMAKER (WAP)

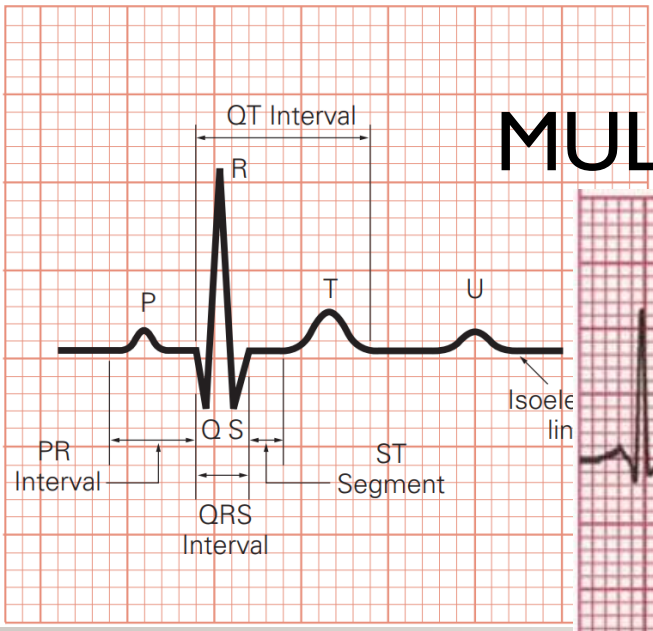


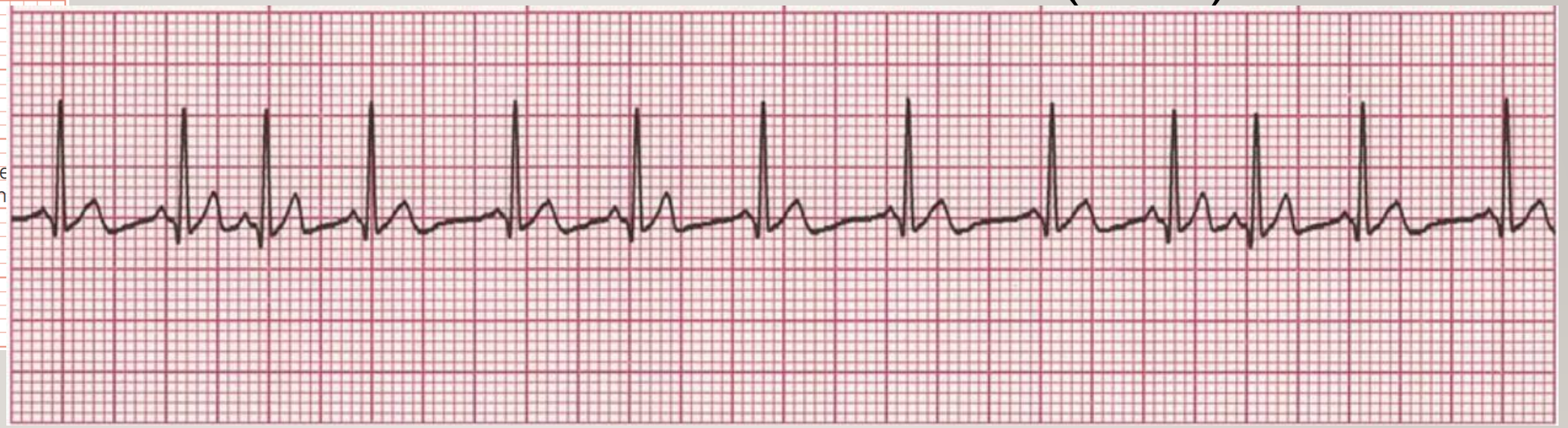


- Rate: Normal (60–100 bpm)
- Rhythm: Irregular
- P Waves: At least three different forms, determined by focus in atria
- PR Interval: Variable; determined by focus
- QRS: Normal (0.06–0.10 sec)

- The pacemaker site transfers from the SA node to other latent pacemaker sites in the atria and the AV junction and then moves back to the SA node.
- Wandering atrial pacemaker may occur in normal hearts as a result of fluctuations in vagal tone. It may also be seen in patients with heart disease or COPD.

MULTIFOCAL ATRIAL TACHYCARDIA (MAT)

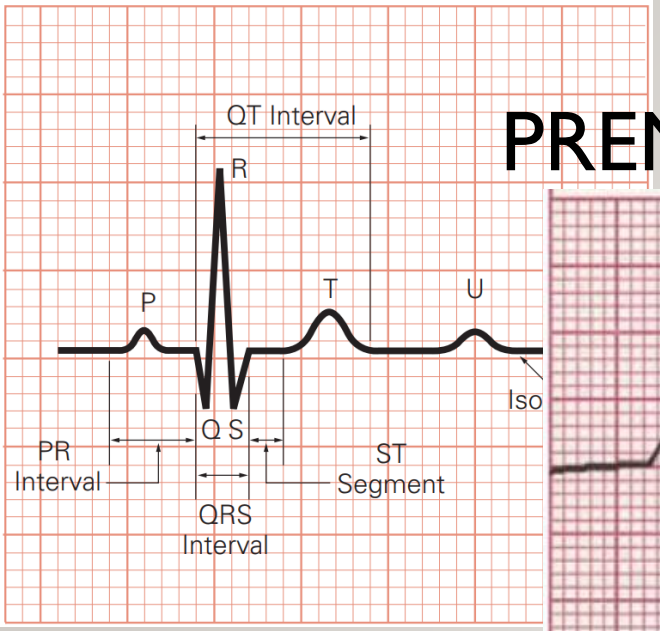


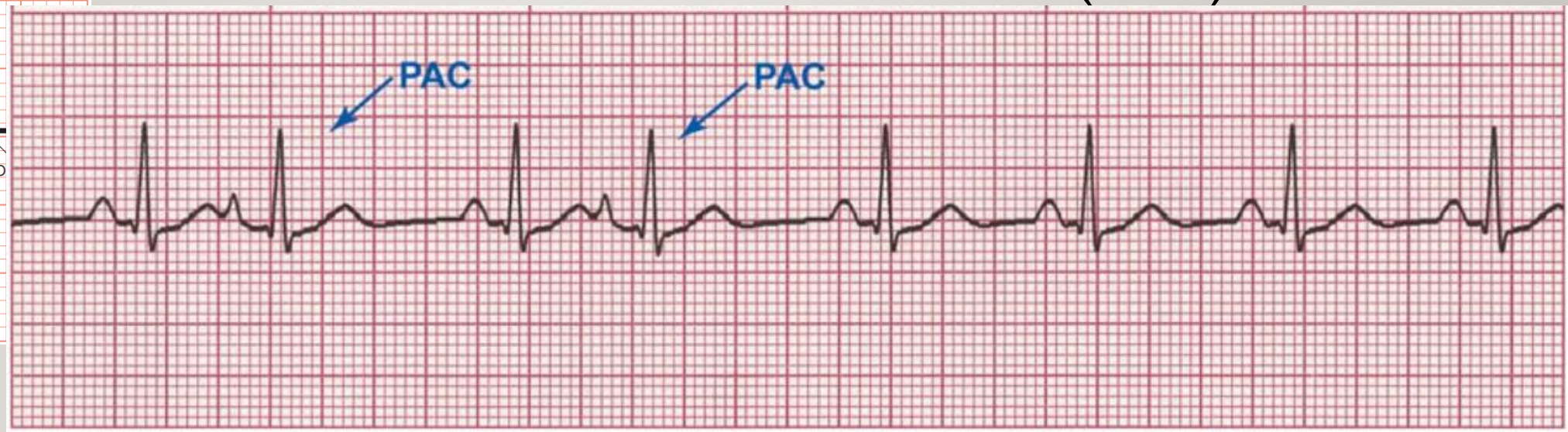


- Rate: Fast (>100 bpm)
- Rhythm: Irregular
- P Waves: At least three different forms, determined by focus in atria
- PR Interval: Variable; determined by focus
- QRS: Normal (0.06–0.10 sec)

- This form of WAP is associated with a ventricular response of >100 bpm.
- MAT may be confused with atrial fibrillation (A-fib); however, MAT has a visible P wave
- MAT is commonly seen in patients with chronic obstructive pulmonary disease but may also occur in acute MI

PREMATURE ATRIAL CONTRACTION (PAC)

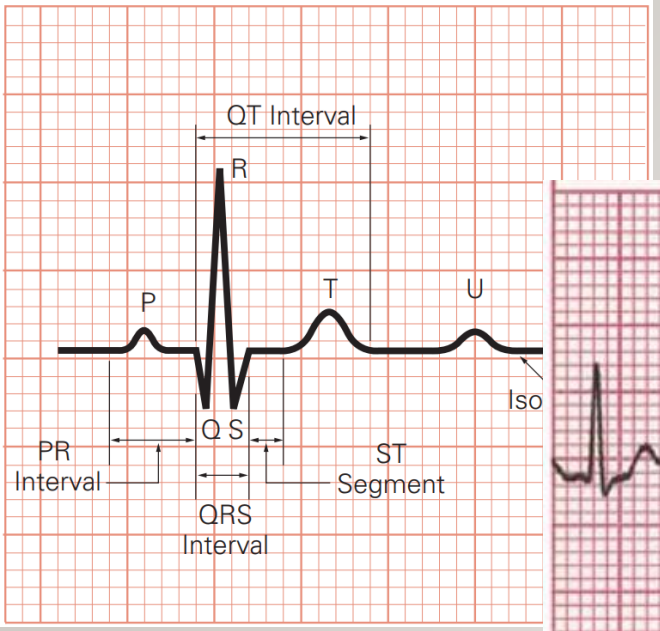


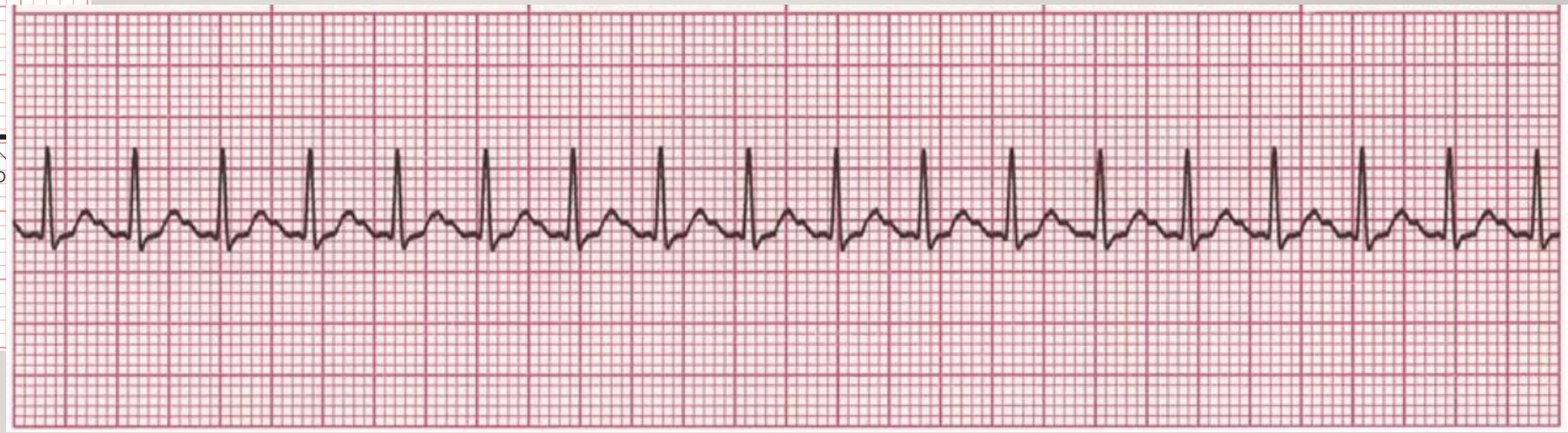


- Rate: Depends on rate of underlying rhythm
- Rhythm: Irregular whenever a PAC occurs
- P Waves: Present; in the PAC, may have a different shape
- PR Interval: Varies in the PAC, otherwise normal (0.12–0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- A single contraction occurs earlier than the next expected sinus contraction.
- After the PAC, sinus rhythm usually resumes
- In patients with heart disease, frequent PACs may precede PSVT, A-fib, or A-flutter

ATRIAL TACHYCARDIA

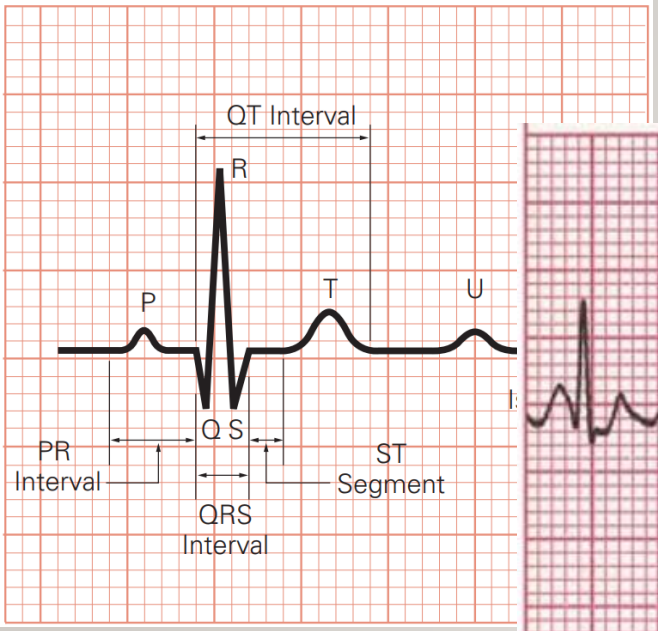


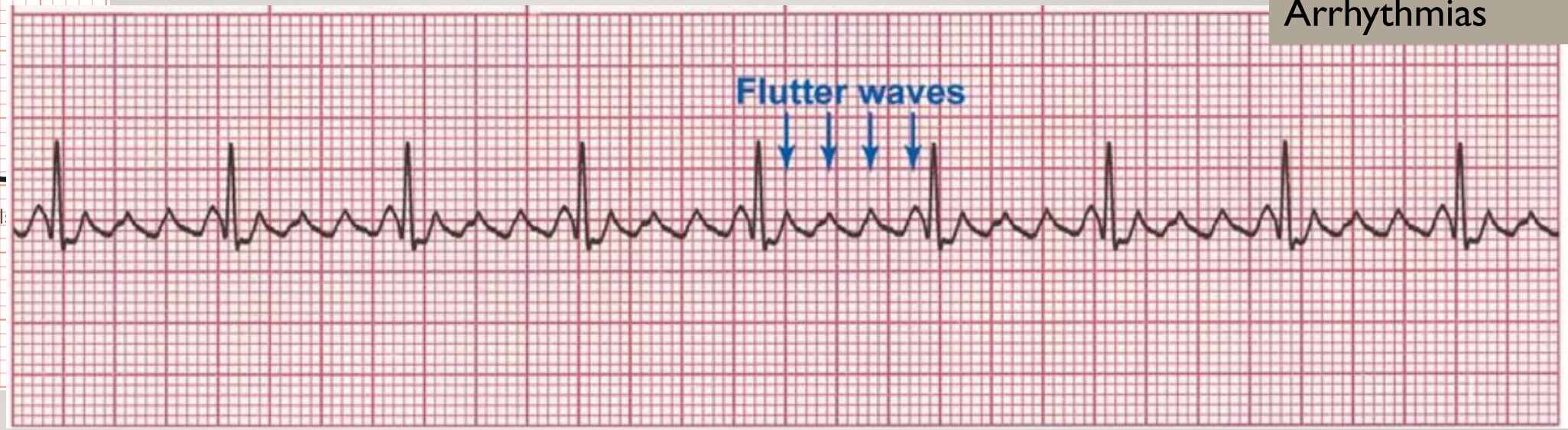


- Rate: 150–250 bpm
- Rhythm: Regular
- P Waves: Normal (upright and uniform) but differ in shape from sinus P waves
- PR Interval: May be short (<0.12 sec) in rapid rates
- QRS: Normal (0.06–0.10 sec), but can be aberrant at times

- A rapid atrial rate overrides the SA node and becomes the dominant pacemaker.
- Some ST wave and T wave abnormalities may be present.

ATRIAL FLUTTER

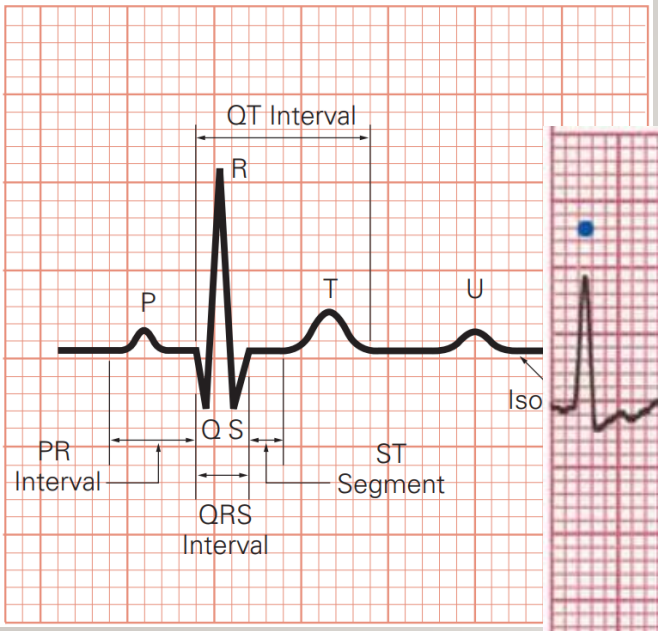


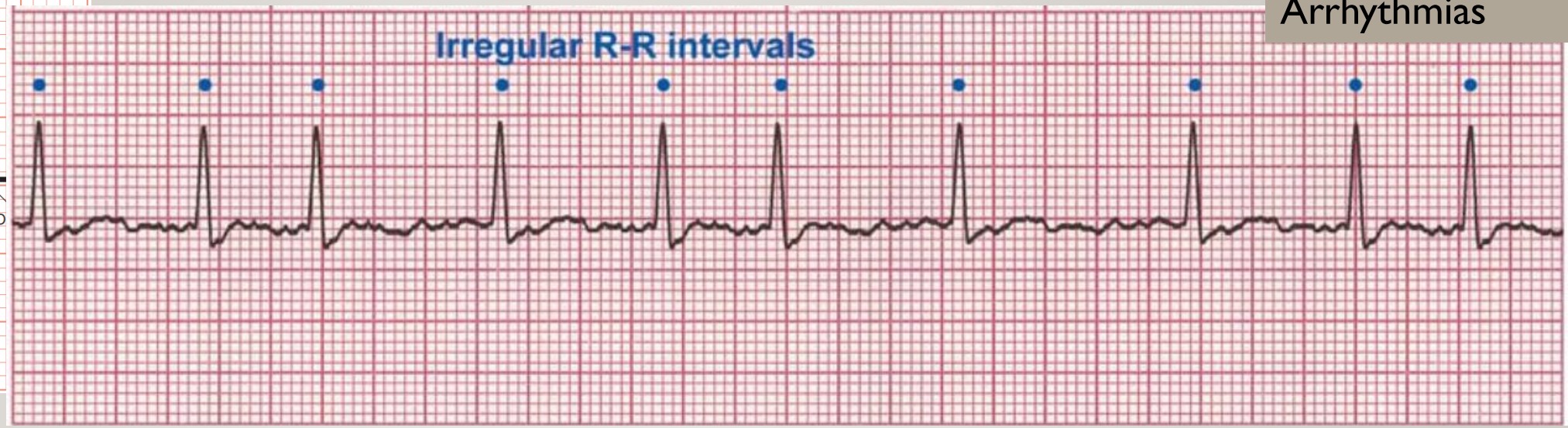


- Rate: Atrial: 250–350 bpm; ventricular: variable.
- Rhythm: Atrial: regular; ventricular: variable
- P Waves: Flutter waves have a saw-toothed appearance; some may not be visible, being buried in the QRS
- PR Interval: Variable
- QRS: Usually normal (0.06–0.10 sec), but may appear widened if flutter waves are buried in the QRS

- The AV node conducts impulses to the ventricles at a 2:1, 3:1, 4:1, or greater ratio (rarely 1:1).
- The degree of AV block may be consistent or variable
- The presence of A-flutter may be the first indication of cardiac disease.
- Signs and symptoms depend on the ventricular response rate.

ATRIAL FIBRILLATION

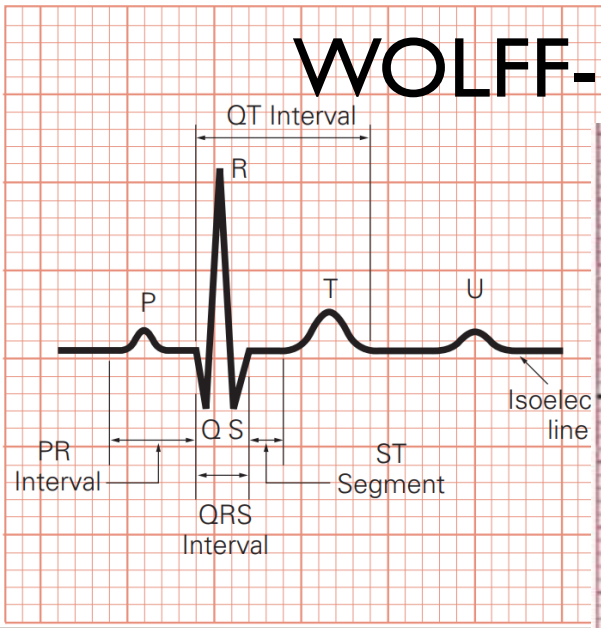


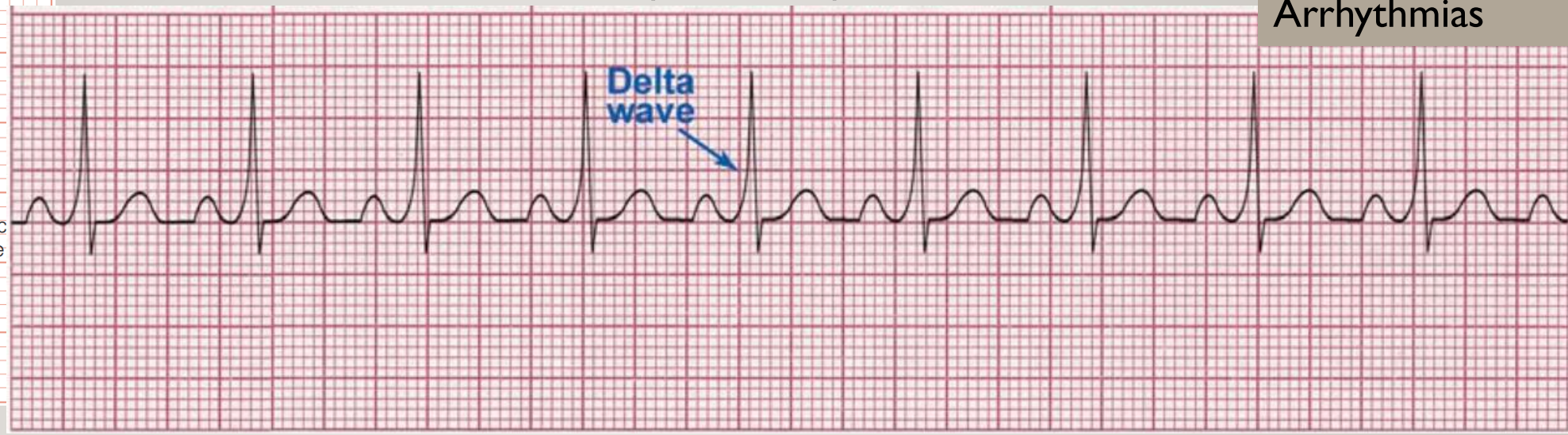


- Rate: Atrial: ≥350 bpm; ventricular: variable
- Rhythm: Irregular
- P Waves: No true P waves; chaotic atrial activity
- PR Interval: None
- QRS: Normal (0.06–0.10 sec)

- Rapid, erratic electrical discharge comes from multiple atrial ectopic foci.
- No organized atrial depolarization are detectable
- Atrial fibrillation is often a chronic arrhythmia associated with underlying heart disease
- Signs and symptoms depend on the ventricular response rate.

WOLFF-PARKINSON-WHITE (WPW) SYNDROME

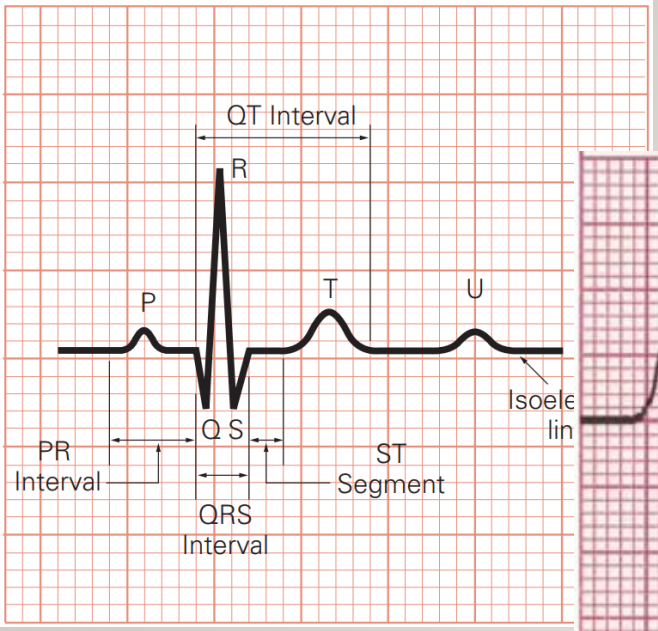


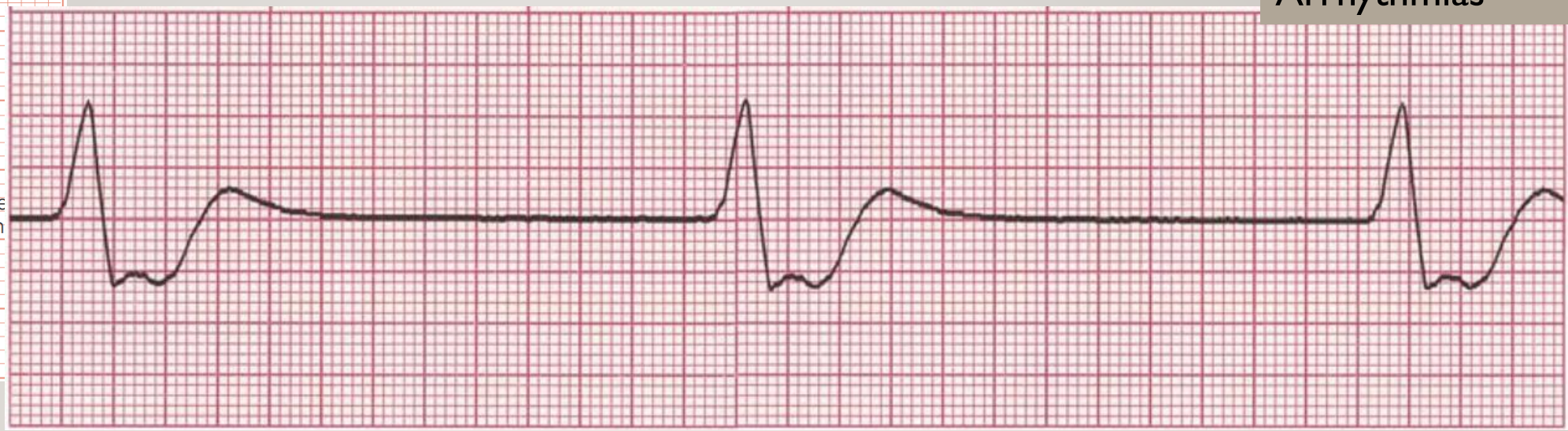


- Rate: Depends on rate of underlying rhythm
- Rhythm: Regular unless associated with A-fib
- P Waves: Normal (upright and uniform) unless A fib is present
- PR Interval: Short (<0.12 sec)
- QRS: Wide (>0.10 sec); delta wave present

- In WPW an accessory conduction pathway is present between the atria and the ventricles. Electrical impulses may be rapidly conducted to the ventricles.
- These rapid impulses create a slurring of the initial portion of the QRS; the slurred effect is called a delta wave
- WPW is associated with narrow-complex tachycardias, including A-flutter and A-fib.

IDIOVENTRICULAR RHYTHM

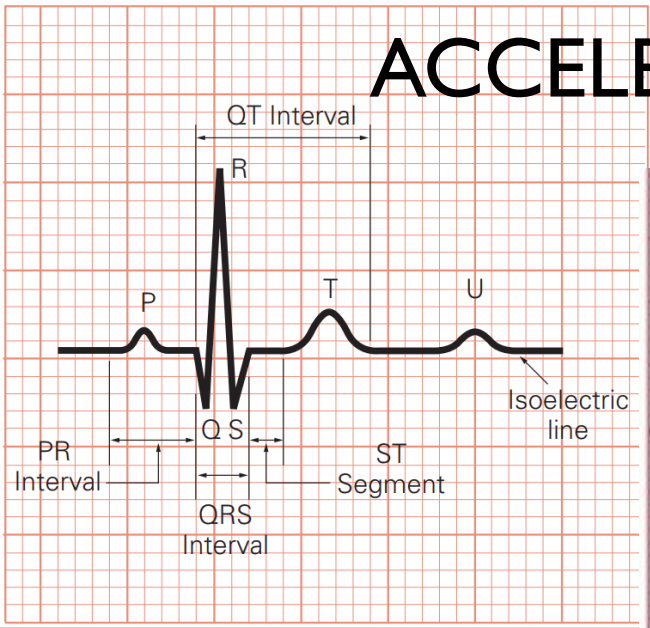


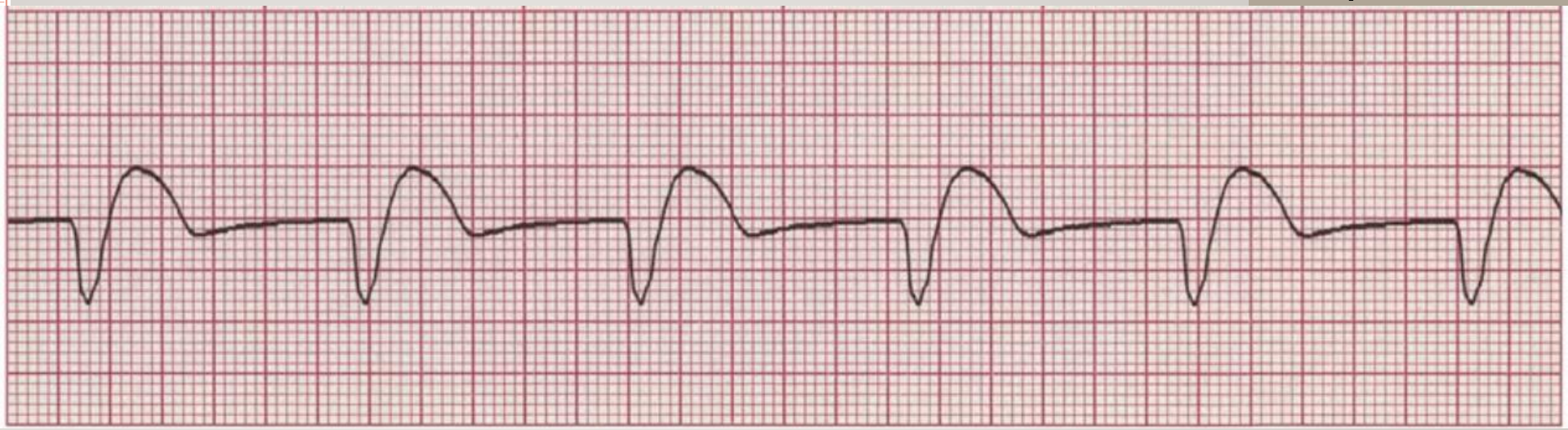


- Rate: 20–40 bpm
- Rhythm: Regular
- P Waves: None
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance

- Diminished cardiac output is expected because of the slow heart rate.
- An idioventricular rhythm may be called an agonal rhythm when the heart rate drops below 20 bpm.
- An agonal rhythm is generally a terminal event and is usually the last rhythm before asystole.

ACCELERATED IDIOVENTRICULAR RHYTHM

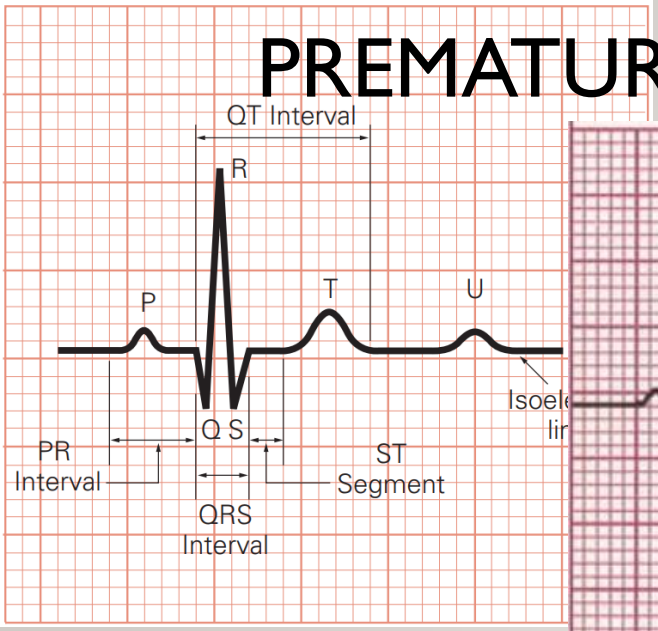


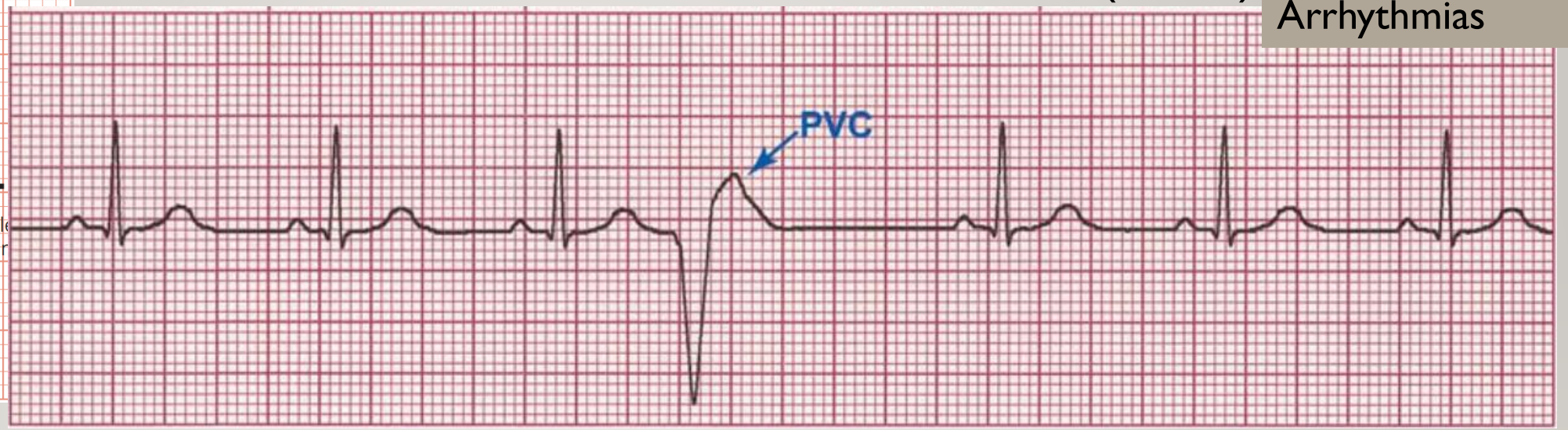


- Rate: 41–100 bpm
- Rhythm: Regular
- P Waves: None
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance

- Idioventricular rhythms appear when supraventricular pacing sites are suppressed or absent.

PREMATURE VENTRICULAR CONTRACTION (PVC)

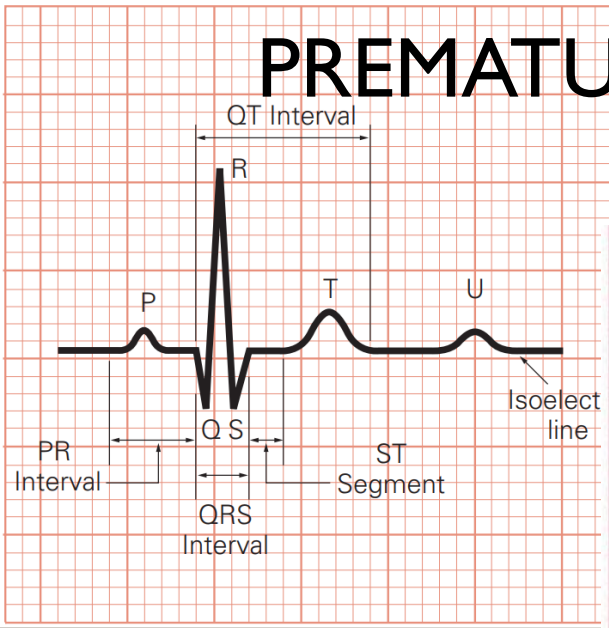




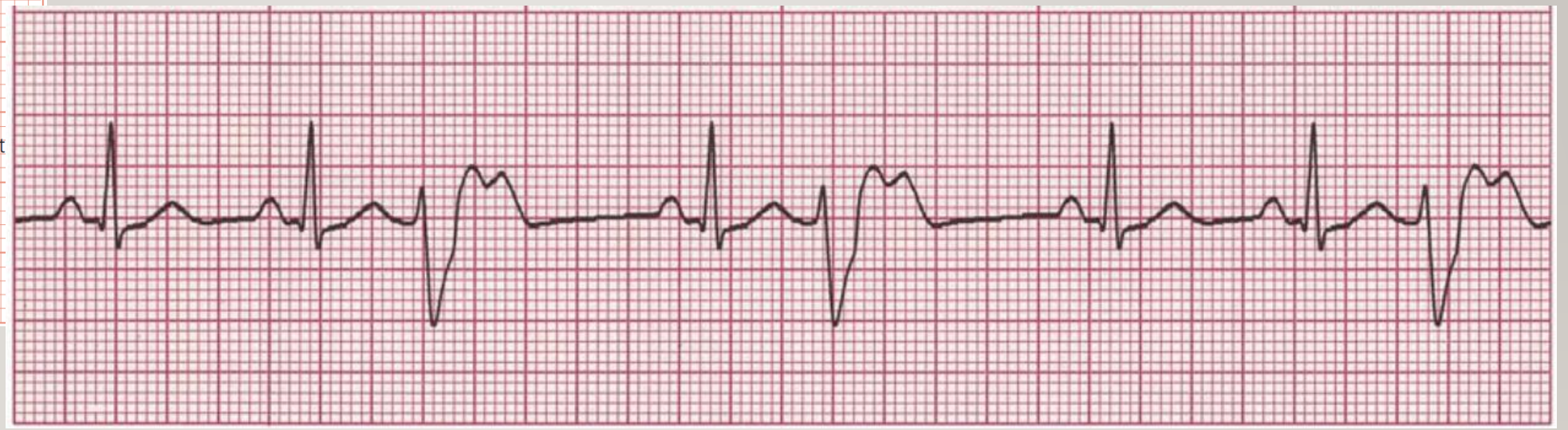
- Rate: Depends on rate of underlying rhythm
- Rhythm: Irregular whenever a PVC occurs
- P Waves: None associated with the PVC
- PR Interval: None associated with the PVC
- QRS: Wide (>0.10 sec), bizarre appearance

- PVCs result from an irritable ventricular focus.
- PVCs may be uniform (same form) or multiform (different forms).
- Usually a PVC is followed by a full compensatory pause because the sinus node timing is not interrupted.
- Normally the sinus rate produces the next sinus impulse on time.
- In contrast, a PVC may be followed by a noncompensatory pause if the PVC enters the sinus node and resets its timing; this enables the following sinus P wave to appear earlier than expected
- Patients may sense the occurrence of PVCs as skipped beats.
- Because the ventricles are only partially filled, the PVC frequently does not generate a pulse.

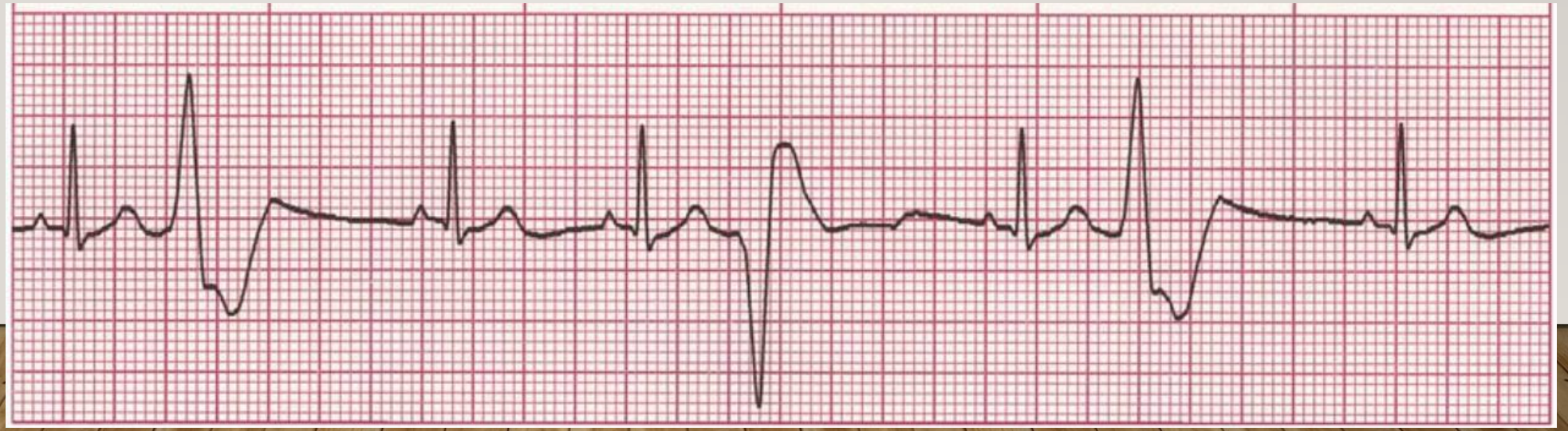
PREMATURE VENTRICULAR CONTRACTION (PVC)



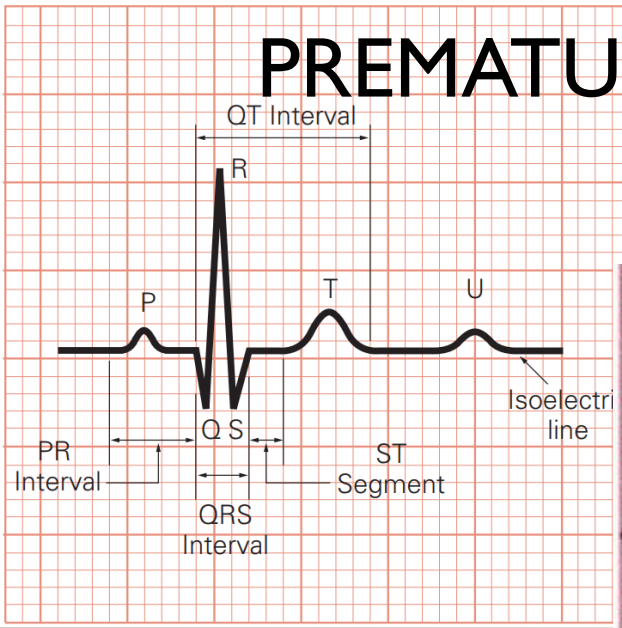
- Uniform



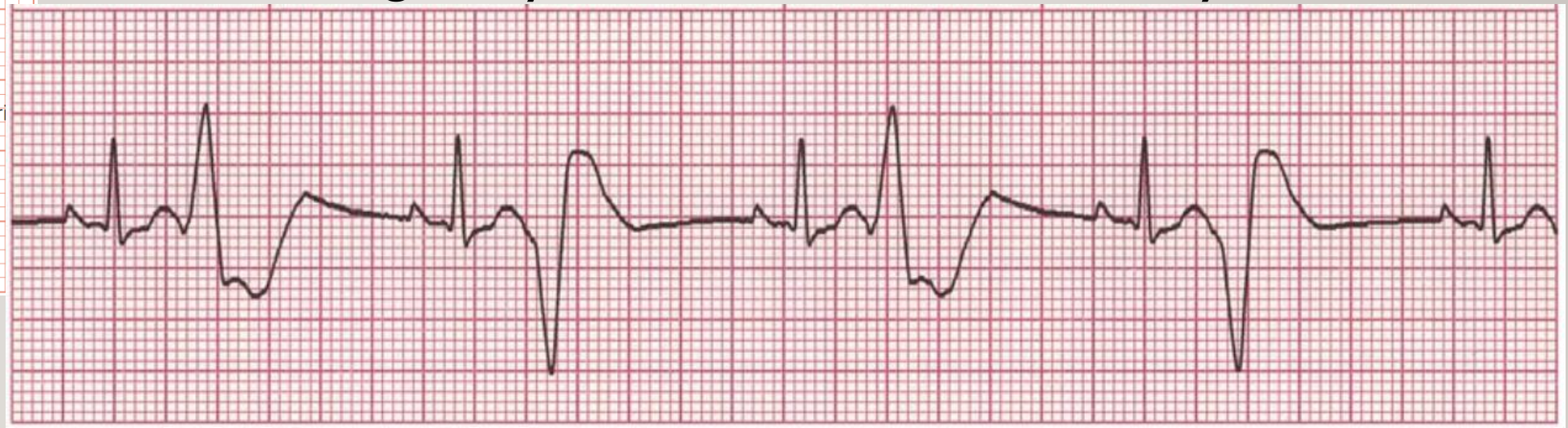
- Multiform



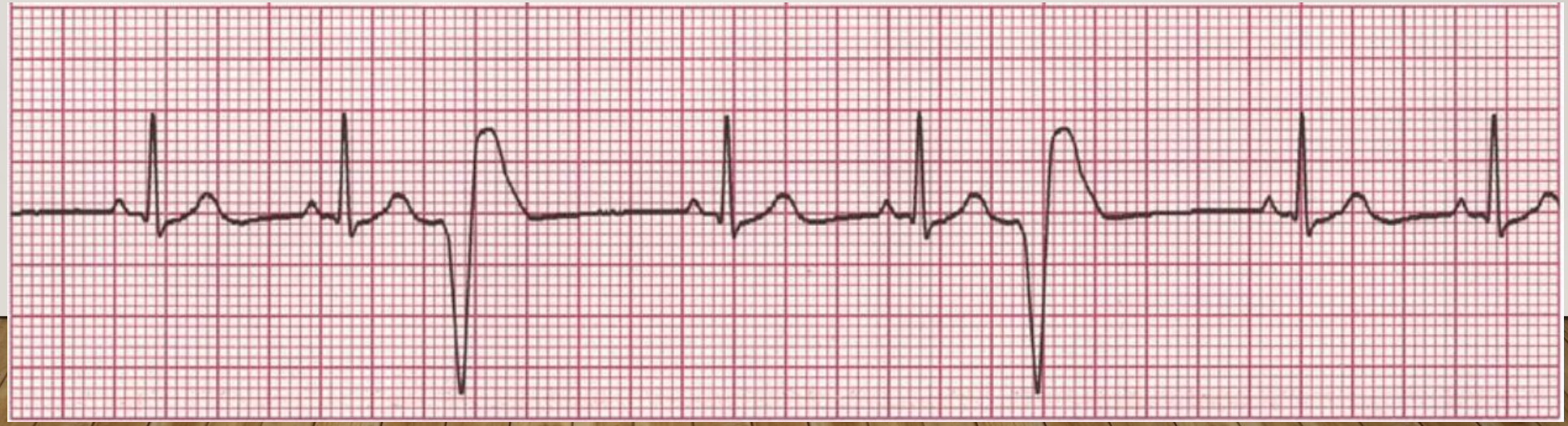
PREMATURE VENTRICULAR CONTRACTION (PVC)



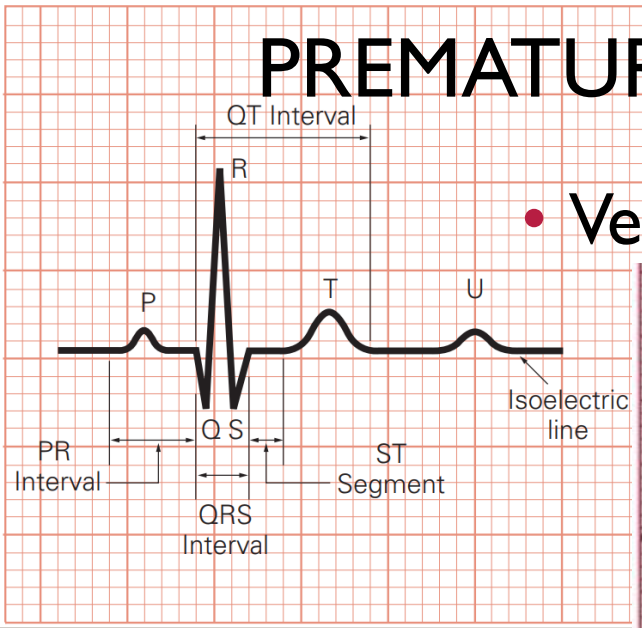
- Ventricular bigeminy, the PVC occurs with every other beat



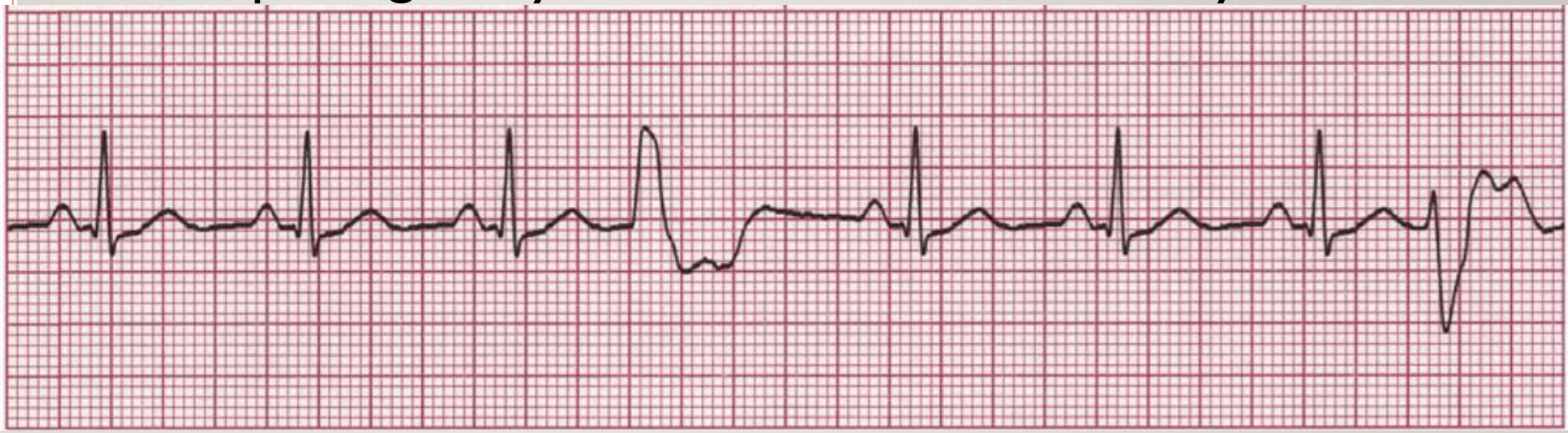
- Ventricular trigeminy - the PVC occurs with every third beat



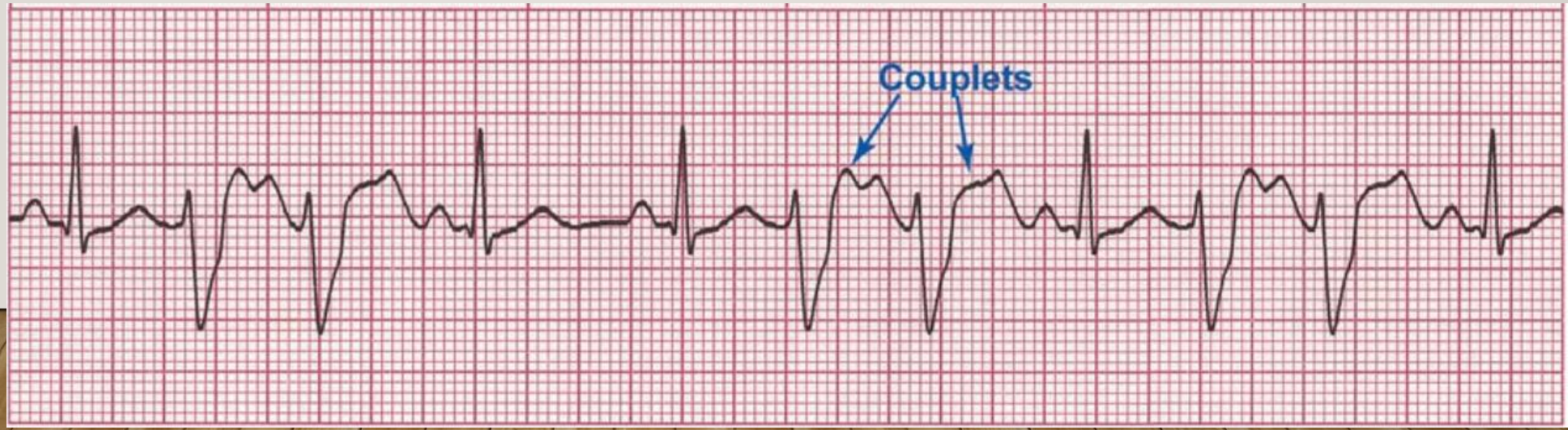
PREMATURE VENTRICULAR CONTRACTION (PVC)



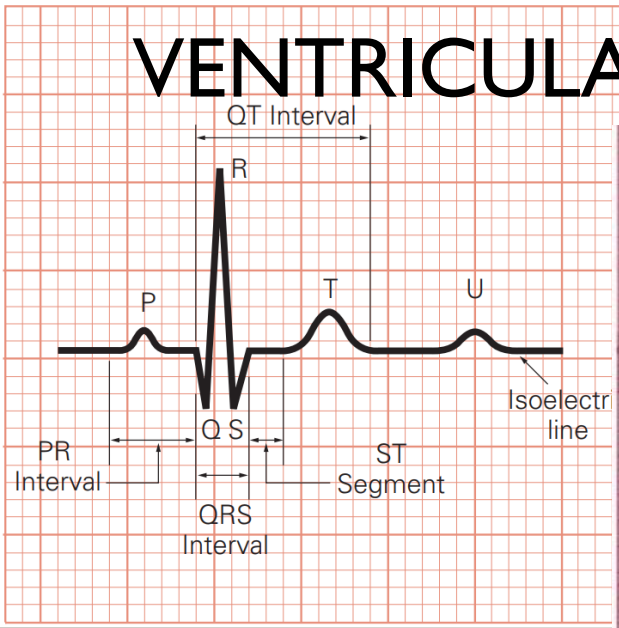
- Ventricular quadrigeminy, the PVC occurs with every fourth beat

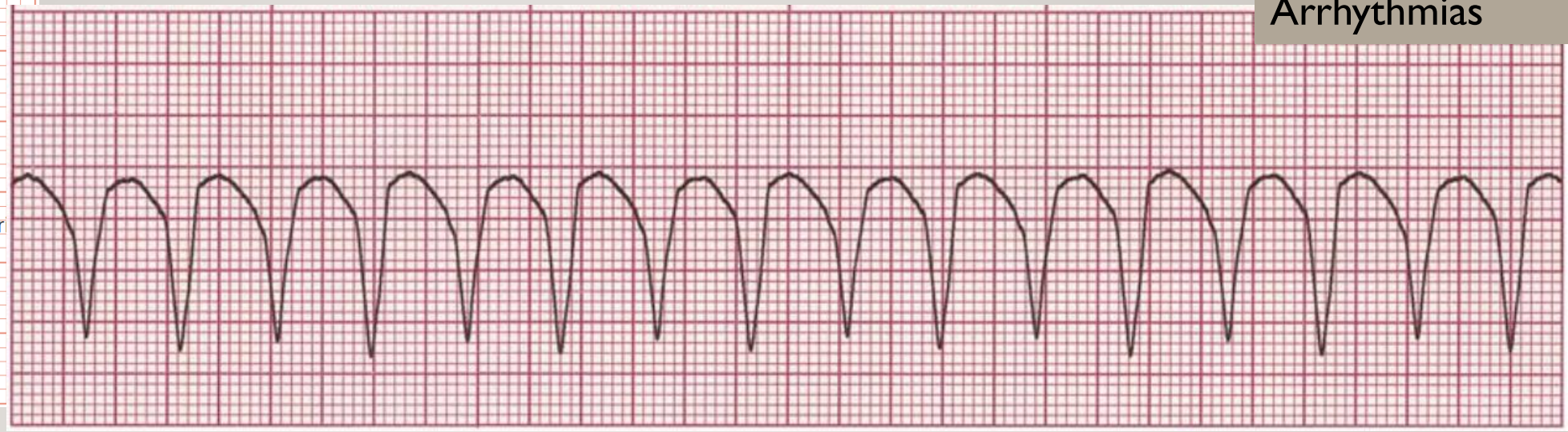


- In a rhythm with PVC couplets, the PVCs occur in pairs



VENTRICULAR TACHYCARDIA (VT): MONOMORPHIC

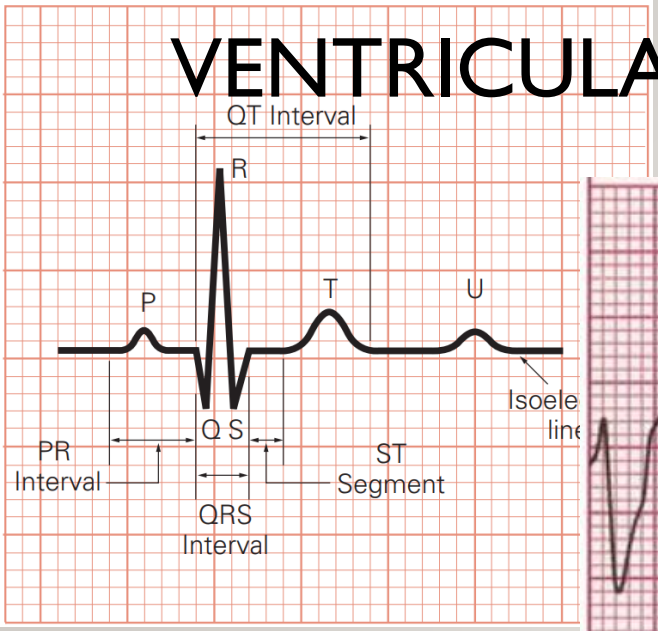


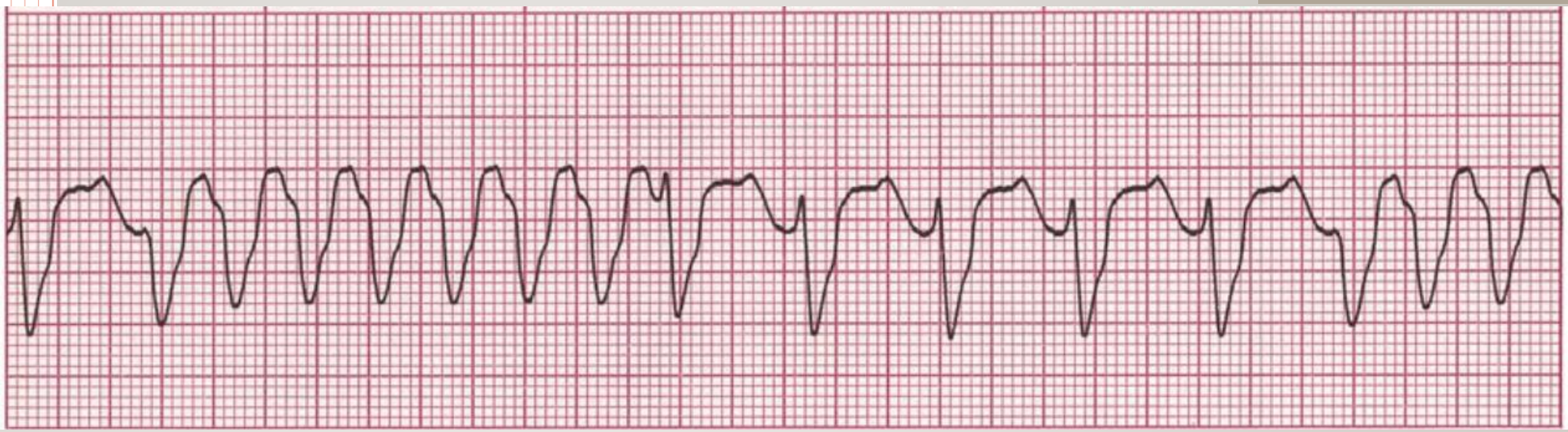


- Rate: 100–250 bpm
- Rhythm: Regular
- P Waves: : None or not associated with the QRS
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance

- In monomorphic VT, QRS complexes have the same shape and amplitude
- It is important to confirm the presence or absence of pulses because monomorphic VT may be perfusing or nonperfusing.
- Monomorphic VT will probably deteriorate into VF or unstable VT if sustained and not treated.

VENTRICULAR TACHYCARDIA (VT): POLYMORPHIC

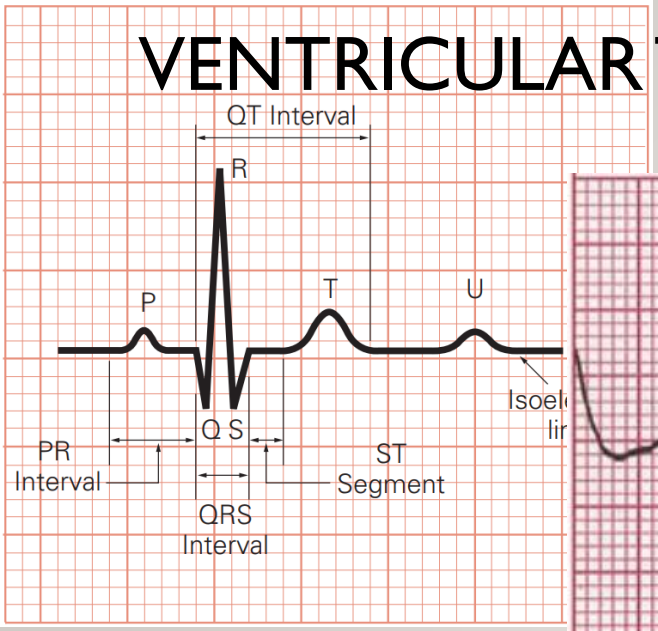


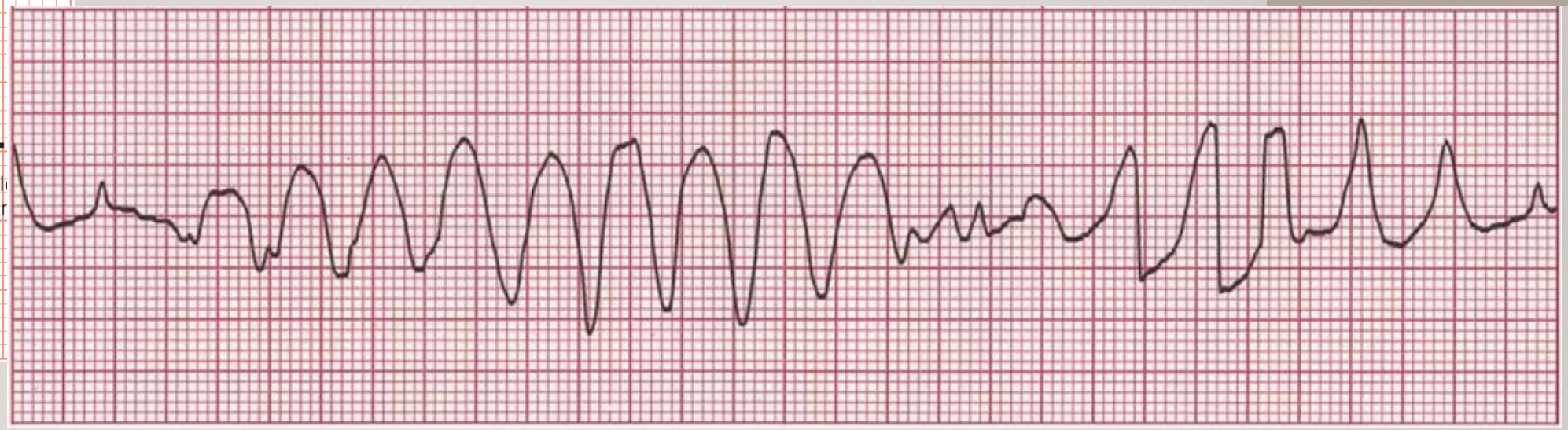


- Rate: 100–250 bpm
- Rhythm: Regular or irregular
- P Waves: : None or not associated with the QRS
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance

- In polymorphic VT, QRS complexes vary in shape and amplitude.
- The QT interval is normal or long
- It is important to confirm the presence or absence of pulses because polymorphic VT may be perfusing or nonperfusing.
- Consider electrolyte abnormalities as a possible cause

VENTRICULAR TACHYCARDIA (VT): TORSADE DE POINTES

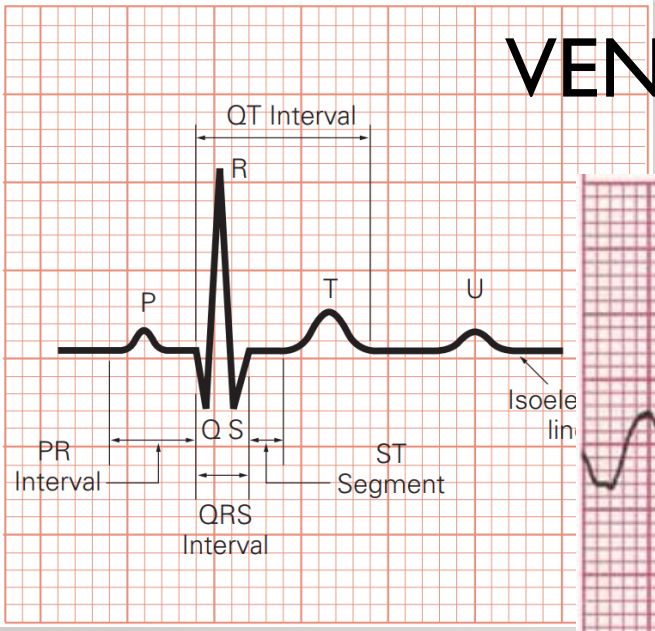


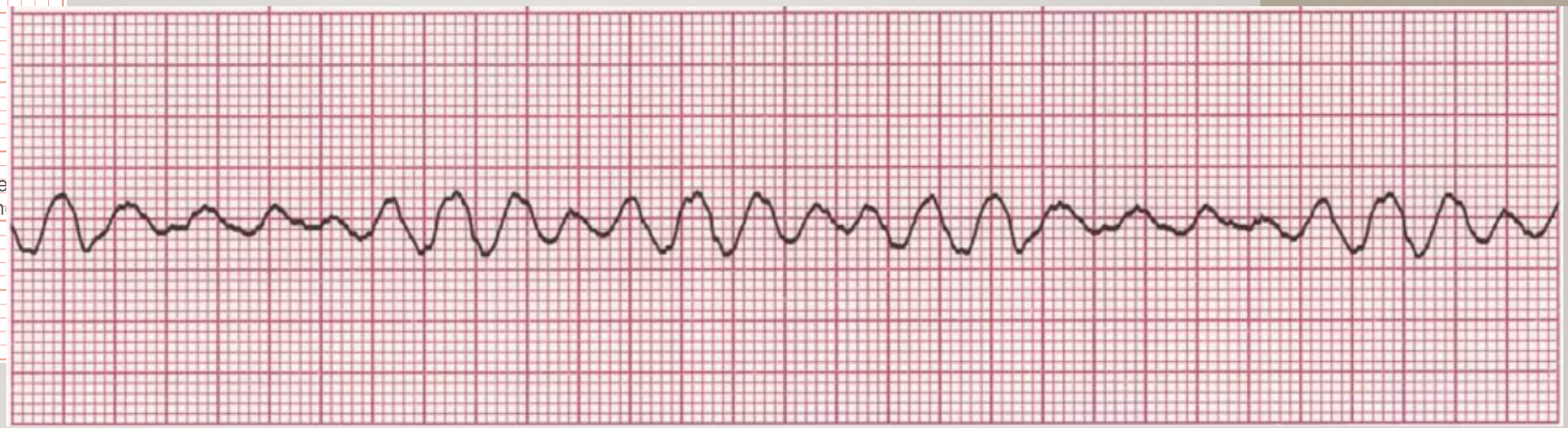


- Rate: 200–250 bpm
- Rhythm: Irregular
- P Waves: : None
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance

- The QRS reverses polarity and the strip shows a spindle effect.
- This rhythm is an unusual variant of polymorphic VT with long QT intervals.
- In French the term means "twisting of points."
- Torsade de pointes may deteriorate to VF or asystole.
- Frequent causes are drugs that prolong QT interval, and electrolyte abnormalities such as hypomagnesemia

VENTRICULAR FIBRILLATION (VF)

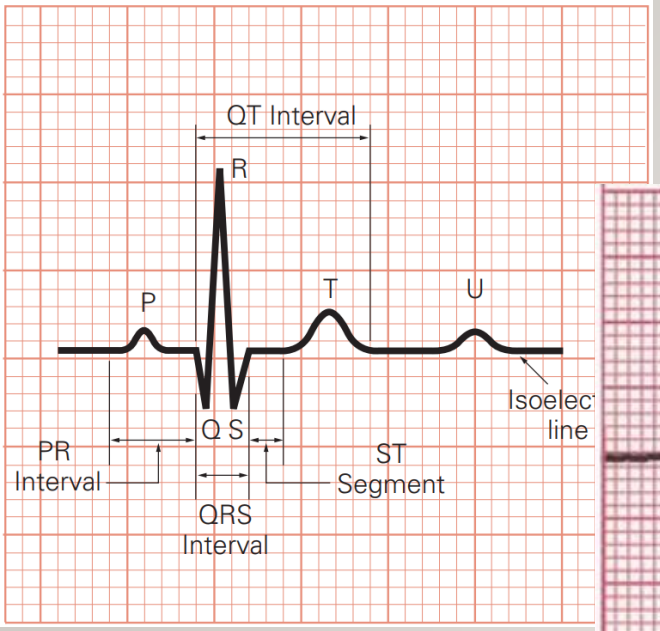


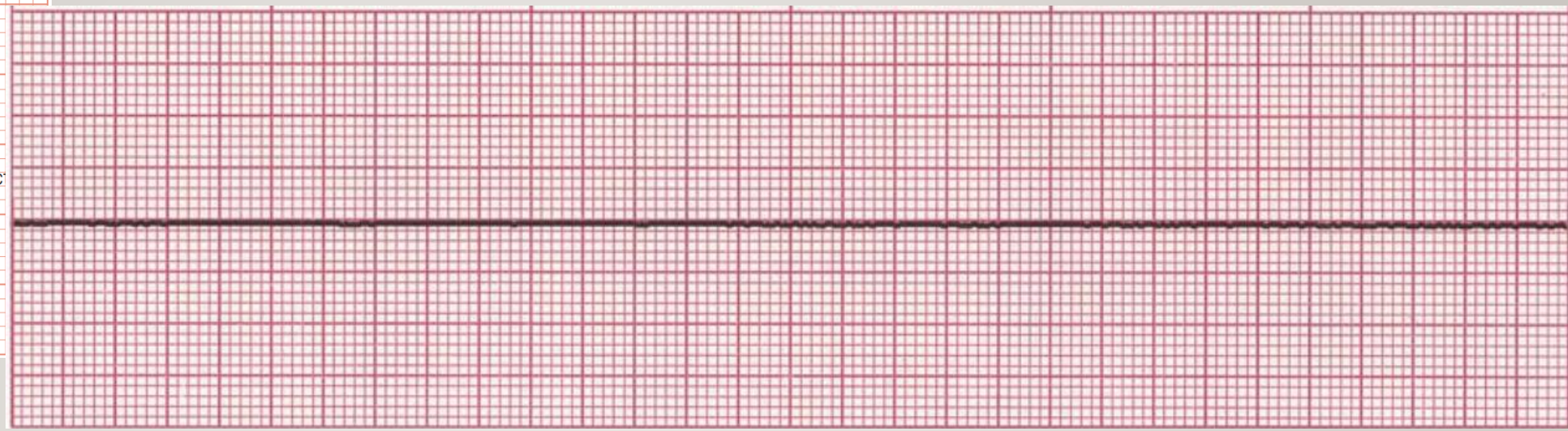


- Rate: Indeterminate
- Rhythm: Chaotic
- P Waves: : None
- PR Interval: None
- QRS: None

- Chaotic electrical activity occurs with no ventricular depolarization or contraction.
- The amplitude and frequency of the fibrillatory activity can be used to define the type of fibrillation as coarse, medium, or fine.
- There is no pulse or cardiac output.
- Rapid intervention is critical.
- The longer the delay, the less the chance of conversion.

ASYSTOLE

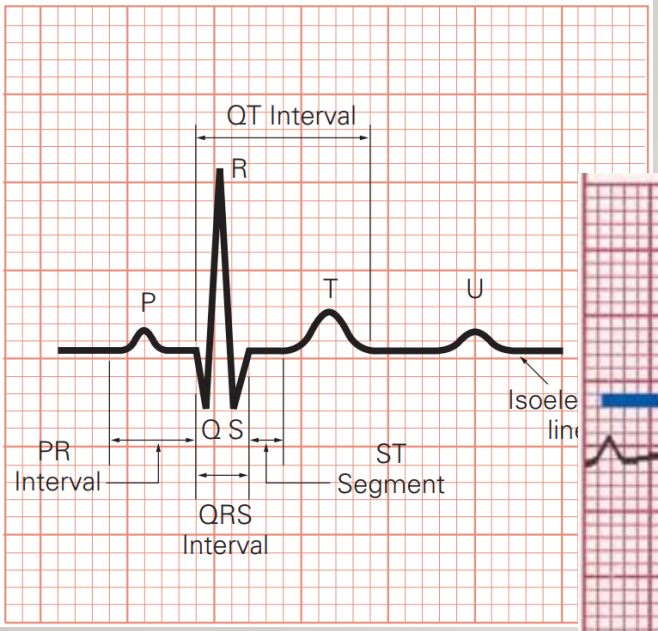


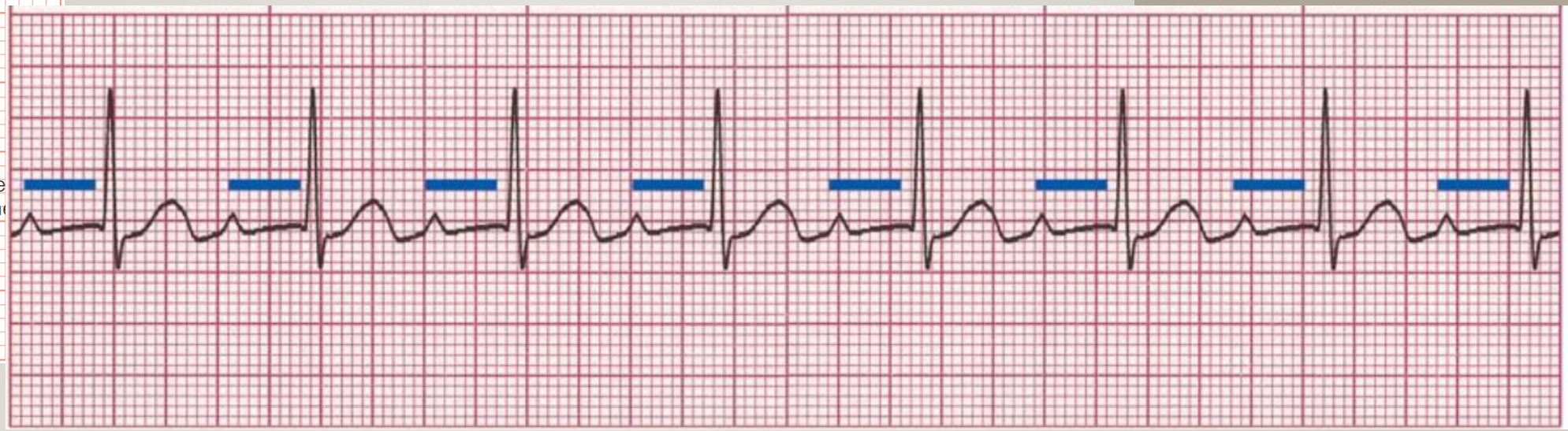


- Rate: None
- Rhythm: None
- P Waves: : None
- PR Interval: None
- QRS: None

- Electrical activity in the ventricles is completely absent.
- Rule out other causes such as loose leads, no power, or signal gain too low.
- Seek to identify the underlying cause: trauma, tension pneumothorax, thrombosis (pulmonary or coronary), cardiac tamponade, toxins, hypo- or hyperkalemia, hypovolemia, hypoxia, hypoglycemia, hypothermia, and acidosis.

FIRST-DEGREE AV BLOCK

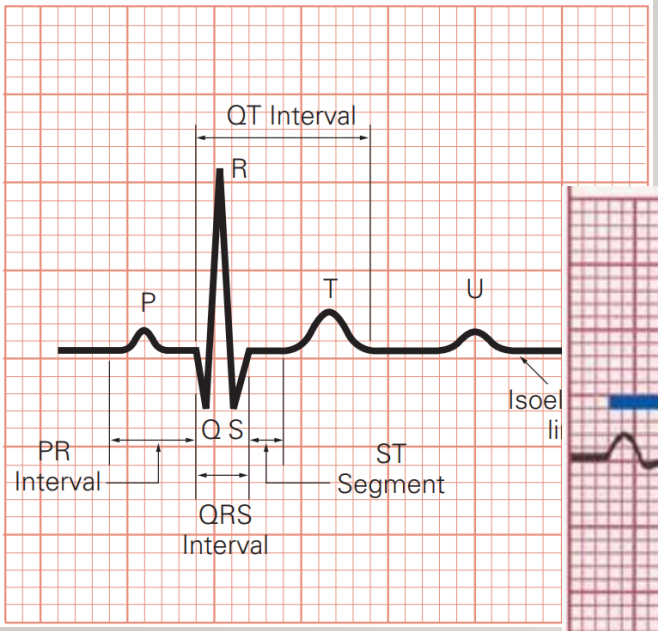


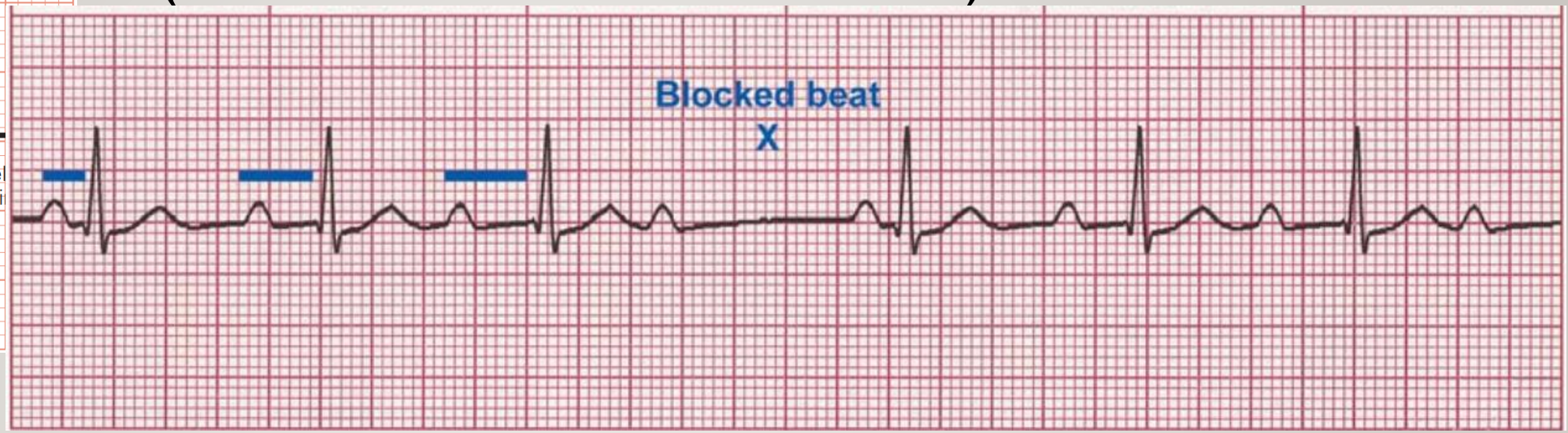


- Rate: Depends on rate of underlying rhythm
- Rhythm: Regular
- P Waves: Normal (upright and uniform)
- PR Interval: Prolonged (>0.20 sec)
- QRS: Normal (0.06–0.10 sec)

- Usually a first-degree AV block is benign, but if associated with an acute MI it may lead to further AV defects.
- Often AV block is caused by medications that prolong AV conduction; these include digoxin, calcium channel blockers, and beta blockers

SECOND-DEGREE AV BLOCK: TYPE I (MOBITZ I OR WENCKEBACH)

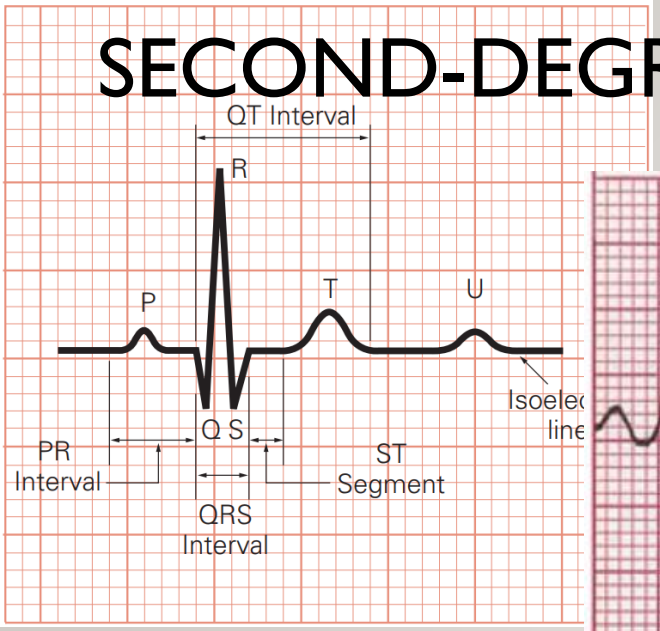


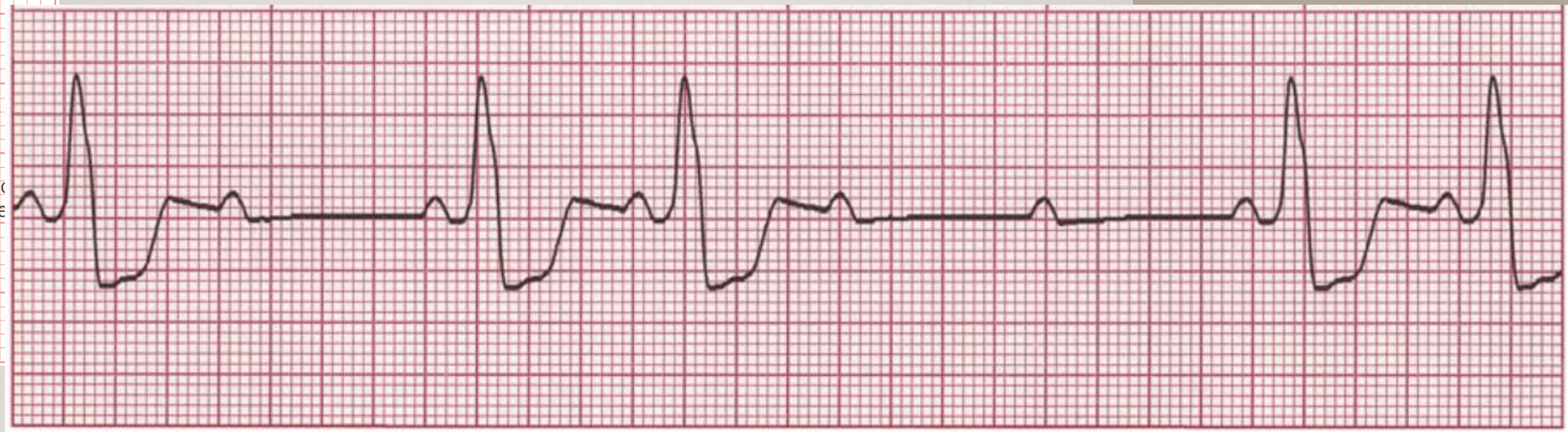


- Rate: Depends on rate of underlying rhythm
- Rhythm: Atrial: regular; ventricular: irregular
- P Waves: Normal (upright and uniform), more P waves than QRS
- PR Interval: Progressively longer until one P wave is blocked and a QRS is dropped
- QRS: Normal (0.06–0.10 sec)

- PR intervals become progressively longer until one P wave is totally blocked and produces no QRS complex.
- After a pause, during which the AV node recovers, this cycle is repeated.
- This rhythm may be caused by medication such as beta blockers, digoxin, and calcium channel blockers. Ischemia involving the right coronary artery is another cause.

SECOND-DEGREE AV BLOCK: TYPE II (MOBITZ II)

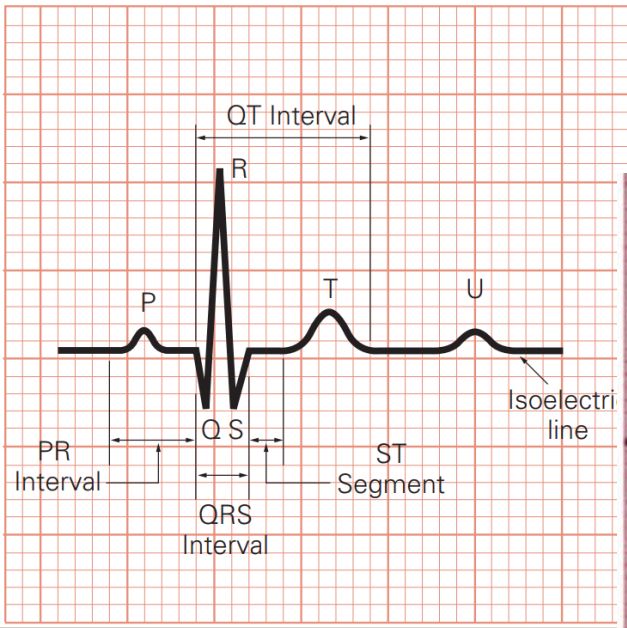


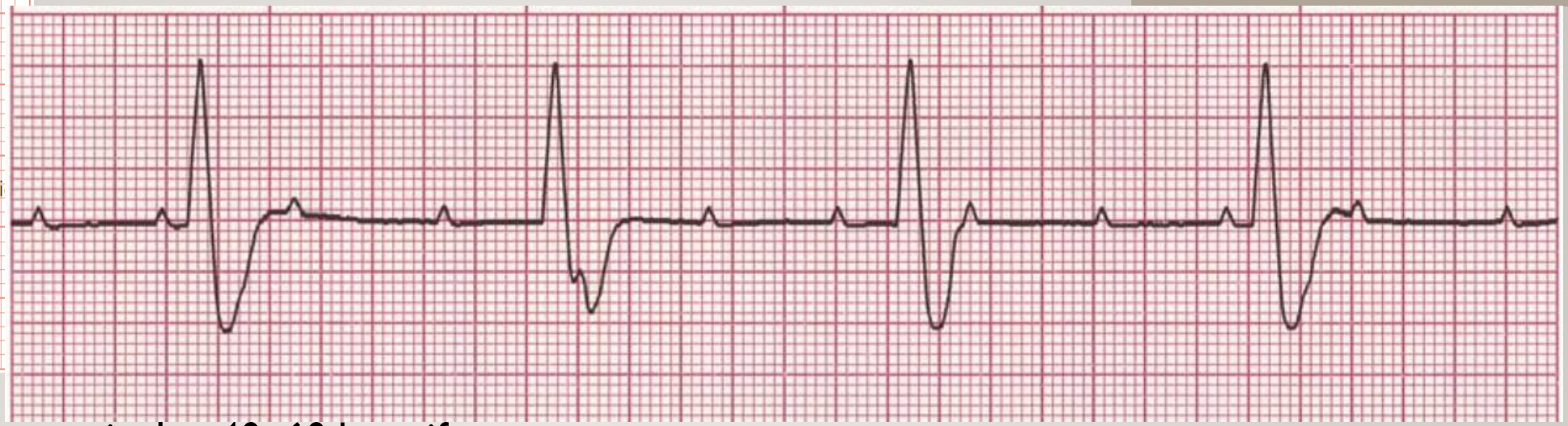


- Rate: Atrial: usually 60–100 bpm; ventricular: slower than atrial rate
- Rhythm: Atrial regular and ventricular may be regular or irregular
- P Waves: Normal (upright and uniform), more P waves than QRS
- PR Interval: Normal or prolonged but constant
- QRS: May be normal, but usually wide (>0.10 sec) if the bundle branches are involved

- Conduction ratio (P waves to QRS complexes) is commonly 2:1, 3:1, 4:1, or variable.
- QRS complexes are usually wide because this block usually involves both bundle branches
- Resulting bradycardia can compromise cardiac output and lead to complete AV block. This rhythm often occurs with cardiac ischemia or an MI.

THIRD-DEGREE AV BLOCK



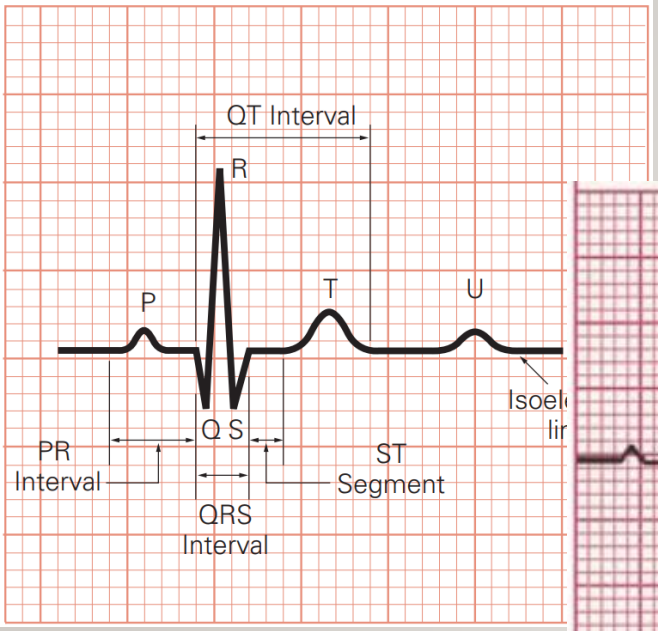


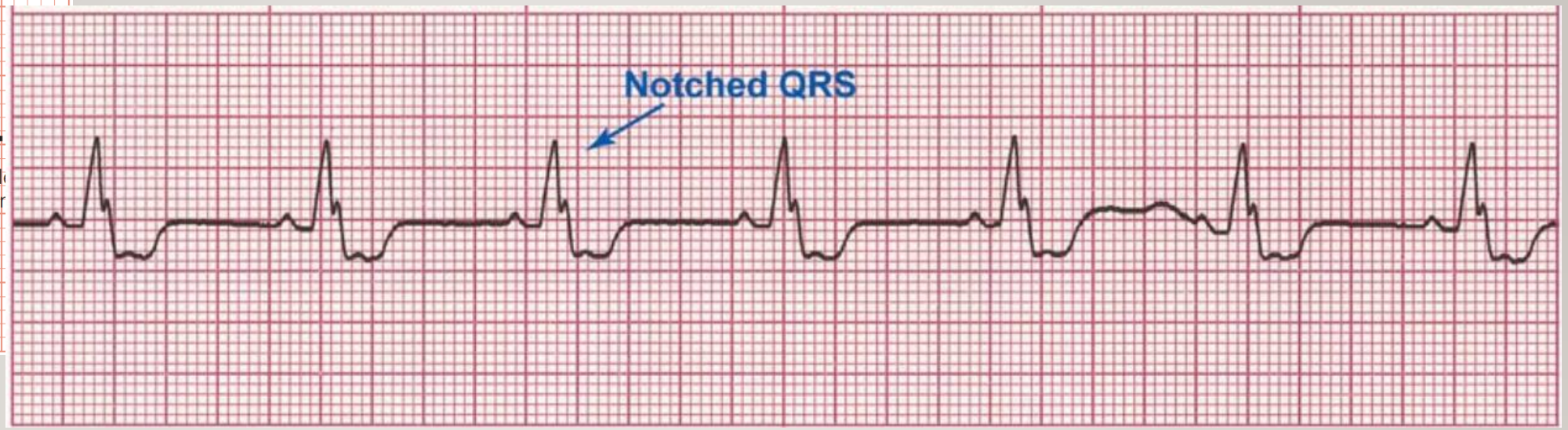
- Rate: Atrial: 60–100 bpm; ventricular: 40–60 bpm if escape focus is junctional, <40 bpm if escape focus is ventricular
- Rhythm: Usually regular, but atria and ventricles act independently
- P Waves: Normal (upright and uniform), may be superimposed on QRS complexes or T waves
- PR Interval: Varies greatly
- QRS: Normal if ventricles are activated by junctional escape focus; wide if escape focus is ventricular

- Conduction between the atria and the ventricles is totally absent because of complete electrical block at or below the AV node. This is known as AV dissociation.
- "Complete heart block" is another name for this rhythm
- Third-degree AV block may be associated with ischemia involving the left coronary arteries.

BUNDLE BRANCH BLOCK (BBB)

Atrioventricular and Bundle Branch Blocks





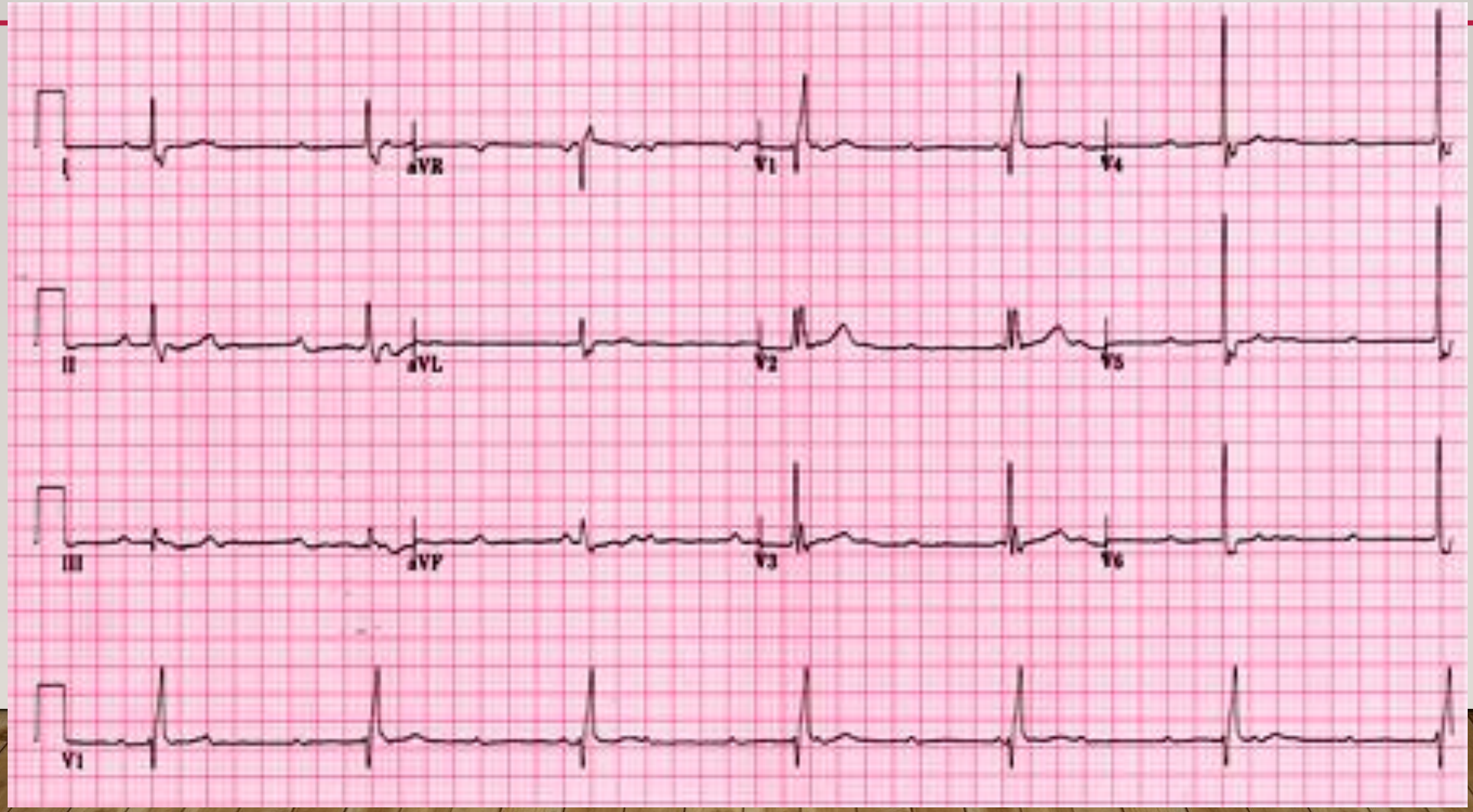
- Rate: Depends on rate of underlying rhythm
- Rhythm: Regula
- P Waves: Normal (upright and uniform)
- PR Interval: Normal (0.12–0.20 sec)
- QRS: Wide (>0.10 sec) with or without a notched appearance

- Either the left or the right ventricle may depolarize late, creating a "wide" or "notched" QRS complex.
- Bundle branch block commonly occurs in coronary artery disease

CASE HISTORY
AV-BLOCK

- A 78-year-old man with a history of hypertension presents to his primary care physician with 1 episode of dizziness while watching television.
- On physical examination, his heart rate is measured at about 40 bpm.
- A 12-lead ECG is obtained showing sinus rhythm at about 75 bpm and complete heart block with a wide escape rhythm at about 40 bpm.

CASE HISTORY
AV-BLOCK



CASE HISTORY
AV-BLOCK

- On further questioning, the patient admits to increasing fatigue and dyspnoea on exertion for the past few weeks.
- Notably, the patient has bifascicular block at baseline (right bundle-branch block and left anterior fascicular block).

CASE HISTORY AV-BLOCK

- The degree of AV block and the severity of symptoms are not necessarily directly related.
- For example, patients with complete (third-degree) AV block may be minimally symptomatic or completely asymptomatic.
- Ultimately, these patients may be diagnosed incidentally on undergoing an evaluation for other reasons. On the other hand, patients with type I second-degree AV block may be very symptomatic, presenting with syncope or pre-syncope due to very slow ventricular rates.
- AV block may also occur in the setting of other acute illnesses such as an acute coronary syndrome, severe electrolyte or pH disturbances, or severe hypoxaemia.
- AV block of any degree may also occur in post-cardiac surgery patients and in patients with Lyme disease.

TREATMENT ALGORITHM

AV-block

first-degree AV block or type I second-degree AV block		
■ asymptomatic	1st	monitoring
■ symptomatic	1st	discontinuation of AV-nodal blocking medications

- Patients are at low risk for progression to higher-degree AV block.
- ECGs may be rechecked if symptoms develop, but do not need to be re-checked on a routine basis.

type I third-degree AV block		
■ asymptomatic or mildly to moderately symptomatic	1st	condition-specific management and discontinuation of AV node-blocking drugs
	2nd	PPM or cardiac resynchronisation therapy ± ICD placement
■ severely symptomatic	1st	condition-specific management, discontinuation of AV-nodal blocking drugs, and temporary (transcutaneous or transvenous) pacing
	2nd	PPM or cardiac resynchronisation therapy ± ICD placement

TREATMENT ALGORITHM

AV-block

first-degree AV block or type I second-degree AV block

asymptomatic	1st	monitoring
symptomatic	1st	discontinuation of AV-nodal blocking medications
	2nd	infrequently: PPM or cardiac resynchronisation therapy ± ICD

- Pts with first-degree AV block and a PR>300 msec may experience symptoms related to the haemodynamic consequence of such prolonged AV delay.
- An increased pulmonary capillary wedge pressure and attendant symptoms of dyspnoea result, as well as decreased ventricular filling leading to decreased stroke volume and cardiac output.
- Some patients with type I second-degree AV block can experience symptoms, ranging from those related to first-degree AV block to more generalised symptoms of fatigue, pre-syncope, or syncope.
- The most common AV-nodal blocking medications include β-blockers, nondihydropyridine CCB, and digitalis.

TREATMENT ALGORITHM

AV-block

first-degree AV block or type I second-degree AV block

asymptomatic	1st	monitoring
symptomatic	1st	discontinuation of AV-nodal blocking medications
	2nd	infrequently: PPM or cardiac resynchronisation therapy ± ICD placement

type II second-degree AV block or third-degree AV block

- If symptoms are severe enough, permanent pacemaker (PPM) implantation should be considered.
- Usually a dualchamber (1 right atrial and 1 right ventricular lead) pacemaker is placed.
- Biventricular pacemaker (placement of a third wire, in a branch of the coronary sinus, to enable left ventricular pacing), with or without an implantable cardioverter-defibrillator (ICD) placement, may be considered when the left ventricular ejection fraction is ≤35%.
- An ICD is not indicated for patients with NYHA Class IV heart failure symptoms

	2nd	PPM or cardiac resynchronisation therapy ± ICD placement

TREATMENT ALGORITHM

type II second-degree AV block or third-degree AV block

asymptomatic or mildly to moderately symptomatic

1st condition-specific management and discontinuation of AV node-blocking drugs

- Mild to moderate symptoms include fatigue or dyspnoea, especially exertional, mild symptoms of CHF (pedal oedema, orthopnoea), or mild, episodic lightheadedness.
- While discontinuing these medicines may improve AV conduction, they are not likely to completely reverse clinically significant AV block.
- Condition-specific management includes
  - treating acute coronary syndrome (i.e., antiplatelet medications, urgent revascularisation) and
  - medication toxicity (e.g., glucagon for beta blocker toxicity, calcium for calcium-channel toxicity, or digoxin antibody for digitalis toxicity).
  - When present, electrolyte or pH disturbances and hypoxaemia should be treated appropriately.
- Risks and balances of discontinuing possible causative medications (e.g., beta-blockers) should be weighed in each instance.

TREATMENT ALGORITHM

AV-block

type II second-degree AV block or third-degree AV block		
▪ **asymptomatic or mildly to moderately symptomatic**	**1st**	**condition-specific management and discontinuation of AV node-blocking drugs**
	2nd	**PPM or cardiac resynchronisation therapy ± ICD placement**
▪ **severely symptomatic**	**1st**	**condition-specific management, discontinuation of AV-nodal blocking drugs, and temporary (transcutaneous or**

- In the absence of a reversible cause, these patients should undergo permanent pacemaker (PPM) implantation.
- Usually a dual-chamber pacemaker is placed.
- Biventricular pacemaker, with or without an implantable cardioverter-defibrillator (ICD), when LVEF is ≤35%.
- In patients with highdegree or complete heart block and LVEF of 36% to 50%, cardiac resynchronisation therapy (CRT) may reduce total mortality and hospitalisations, and improve symptoms and QOL.

TREATMENT ALGORITHM

type II second-degree AV block or third-degree AV block		
■ asymptomatic or mildly to moderately symptomatic	1st	condition-specific management and discontinuation of AV node-blocking drugs
	2nd	PPM or cardiac resynchronisation therapy ± ICD placement
■ severely symptomatic	1st	condition-specific management, discontinuation of AV-nodal blocking drugs, and temporary (transcutaneous or

- Severe symptoms include syncope or persistent, severe lightheadedness indicating profound decreases in the ventricular rate.
- When the ventricular rate is significantly low (<40-45 bpm) or the BP is low (mean arterial pressure <65 mmHg), temporary (transcutaneous or transvenous) pacing should be considered.
- Transvenous pacing is much more reliable than transcutaneous pacing and should be performed by a cardiologist when the heart block leads to haemodynamic instability.
- Condition-specific management includes treating ACS, medication toxicity, electrolyte or pH disturbances, hypoxaemia.

TREATMENT ALGORITHM

AV-block

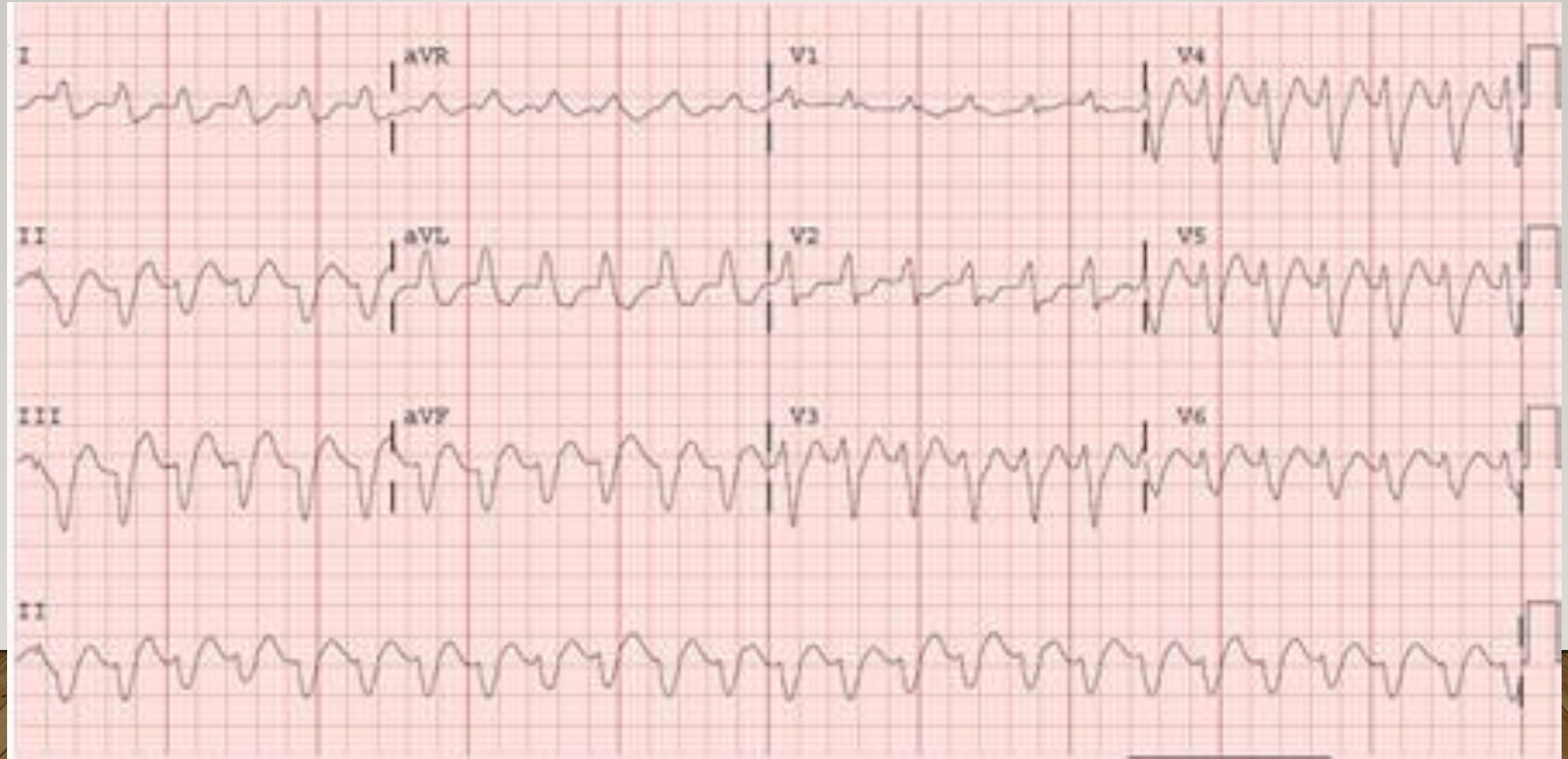
type II second-degree AV block or third-degree AV block		
▪ asymptomatic or mildly to moderately symptomatic	1st	condition-specific management and discontinuation of AV node-blocking drugs
	2nd	PPM or cardiac resynchronisation therapy ± ICD placement
▪ severely symptomatic	1st	condition-specific management, discontinuation of AV-nodal blocking drugs, and temporary (transcutaneous or transvenous) pacing
	2nd	PPM or cardiac resynchronisation therapy ± ICD placement

- Severe symptoms include syncope or persistent, severe lightheadedness indicating profound decreases in the ventricular rate.
- PPM
- ICD
- CRT

CASE HISTORY
SUSTAINED VENTRICULAR TACHYCARDIAS

- A 65-year-old man has a history of prior anterior wall myocardial infarction that occurred 2 years ago, complicated by severe left ventricular systolic dysfunction.
- While walking to the shops, he suddenly became aware of palpitations, diaphoresis, dizziness, and a sense of overwhelming malaise.
- One minute later, he turned grey, lost consciousness, and collapsed onto the floor.
- An ECG revealed sustained monomorphic ventricular tachycardia at 150 bpm.

CASE HISTORY
SUSTAINED VENTRICULAR TACHYCARDIAS



CASE HISTORY
SUSTAINED VENTRICULAR TACHYCARDIAS

- Cardiopulmonary resuscitation was initiated and the patient was cardioverted to sinus rhythm with a 200-J biphasic shock delivered from an external defibrillator.
- The patient regained consciousness.
- There was no antecedent chest discomfort and cardiac enzymes were negative after the event.
- Serum electrolytes were also normal.
- He received an implantable cardioverter/defibrillator the next day.

CASE HISTORY
SUSTAINED VENTRICULAR TACHYCARDIAS

- Ventricular arrhythmias may present with a diverse spectrum of symptoms, including palpitations, chest pain, and/or syncope, or may be asymptomatic.
- The type of symptom associated with the arrhythmia depends on its duration, its rate, and whether or not it is associated with abnormal perfusion to the heart or brain.
- Short-lived arrhythmias frequently are asymptomatic or manifest as brief palpitations, whereas longer and more rapid arrhythmias are frequently associated with presyncope or syncope.
- Sudden cardiac death is the most severe manifestation of ventricular arrhythmias; victims of sudden cardiac arrest rarely survive without cardiopulmonary resuscitation and prompt electrical defibrillation.

TREATMENT ALGORITHM

Initial (summ

haemodynamically unstable ventricular tachycardia with a pulse

1st	synchronised cardioversion according to advanced cardiac life support protocol + treatment of reversible cause (if present)
adjunct	anti-arrhythmic medication

- Cardioversion is essential for the acute treatment of haemodynamically unstable ventricular tachycardia (VT) (symptomatic or severely hypotensive VT).
- Synchronised cardioversion should be considered before attempting anti-arrhythmic drug therapy in patients who have syncope, presyncope, frequent palpitations, or hypotension (particularly those with symptoms of diminished cerebral perfusion), even if they have apparently stable haemodynamics.
- Cardioversion may be repeated as needed until rhythm is controlled.
- In patients with an identifiable reversible cause of VT (e.g., ischaemia, myocardial infarction, toxicity, drug overdose) management will also involve treatment of the reversible cause.

TREATMENT ALGORITHM

VT

Initial		(summ
haemodynamically unstable ventricular tachycardia with a pulse		
	1st	synchronised cardioversion according to advanced cardiac life support protocol + treatment of reversible cause (if present)
	adjunct	anti-arrhythmic medication
torsades de pointes		
	1st	intravenous magnesium sulfate +
	adjunct	implantable cardioverter defibrillator

- Treatment recommended for SOME patients in selected patient group
- Primary options » amiodarone: 300 mg intravenous push
- Secondary options » lidocaine: 1 to 1.5 mg/kg intravenously as a single dose
- Medical therapy provides an important adjunctive therapy to emergency cardiovascular care, based on the advanced cardiac life support protocol.
- Amiodarone and/or lidocaine are considered useful anti-arrhythmic drugs in these circumstances.

TREATMENT ALGORITHM

treatment of reversible cause (if present)

adjunct	anti-arrhythmic medication

torsades de pointes

1st	intravenous magnesium sulfate + withdraw offending drugs + correct

- magnesium sulfate: 1-2 g intravenously as a single dose
- Torsades de pointes, a specific type of polymorphic ventricular tachycardia (VT) characterised by a twisting appearance around the baseline, occurs in the setting of QT prolongation due to either the congenital or the acquired forms of the long QT syndrome.
- Torsades de pointes should be treated as any other form of VT according to the advanced cardiac life support protocol, with special recognition of the fact that hypokalaemia and hypomagnesaemia are frequently associated with torsades.
- Electrolyte deficiencies should be replenished aggressively.
- Offending drugs should be withdrawn.
- Intravenous magnesium sulfate should be administered.
- Additionally, overdrive pacing and isoprenaline infusion may be useful in this arrhythmia as they reduce the QT interval.

TREATMENT ALGORITHM

		treatment of reversible cause (if present)
	adjunct	anti-arrhythmic medication
torsades de pointes		
	1st	intravenous magnesium sulfate + withdraw offending drugs + correct electrolyte abnormalities
	adjunct	isoprenaline infusion
	adjunct	temporary or permanent pacing
catecholaminergic polymorphic ventricular tachycardia		

- Treatment recommended for SOME patients in selected patient group
- isoprenaline: 2 micrograms/minute intravenous infusion initially, dose titrated according to response, maximum 10 micrograms/minute
- Indicated in patients who present with recurrent torsades de pointes after initial acute therapy.
- It may be useful in this arrhythmia as it reduces the QT interval.
- It is important to be certain that the patient does not have acute ischaemia before administering isoprenaline

TREATMENT ALGORITHM

VT

	adjunct	anti-arrhythmic medication
torsades de pointes		
	1st	intravenous magnesium sulfate + withdraw offending drugs + correct electrolyte abnormalities
	adjunct	isoprenaline infusion
	adjunct	temporary or permanent pacing
catecholaminergic polymorphic ventricular tachycardia		

- Treatment recommended for SOME patients in selected patient group
- Indicated in recurrent torsades de pointes after acute therapy.

TREATMENT ALGORITHM

Initial **(summ**

haemodynamically unstable

- Nadolol
- Medical therapy for catecholaminergic polymorphic ventricular tachycardia includes the use of beta-blockers for both acute and chronic treatment.
- High dose of beta-blockers is usually required.
- Other treatment strategies have been proposed, including a stepwise addition of alternative treatment options, such as calcium-channel blockers and flecainide, to beta-blockers in patients who do not respond sufficiently or who cannot tolerate beta-blockers.
- Left cardiac sympathetic denervation appears to be effective, but has only been tested on small cohorts, and is not universally available.

adjunct **temporary or permanent pacing**

catecholaminergic polymorphic ventricular tachycardia

1st **beta-blockers**

adjunct **implantable cardioverter defibrillator**

TREATMENT ALGORITHM

VT

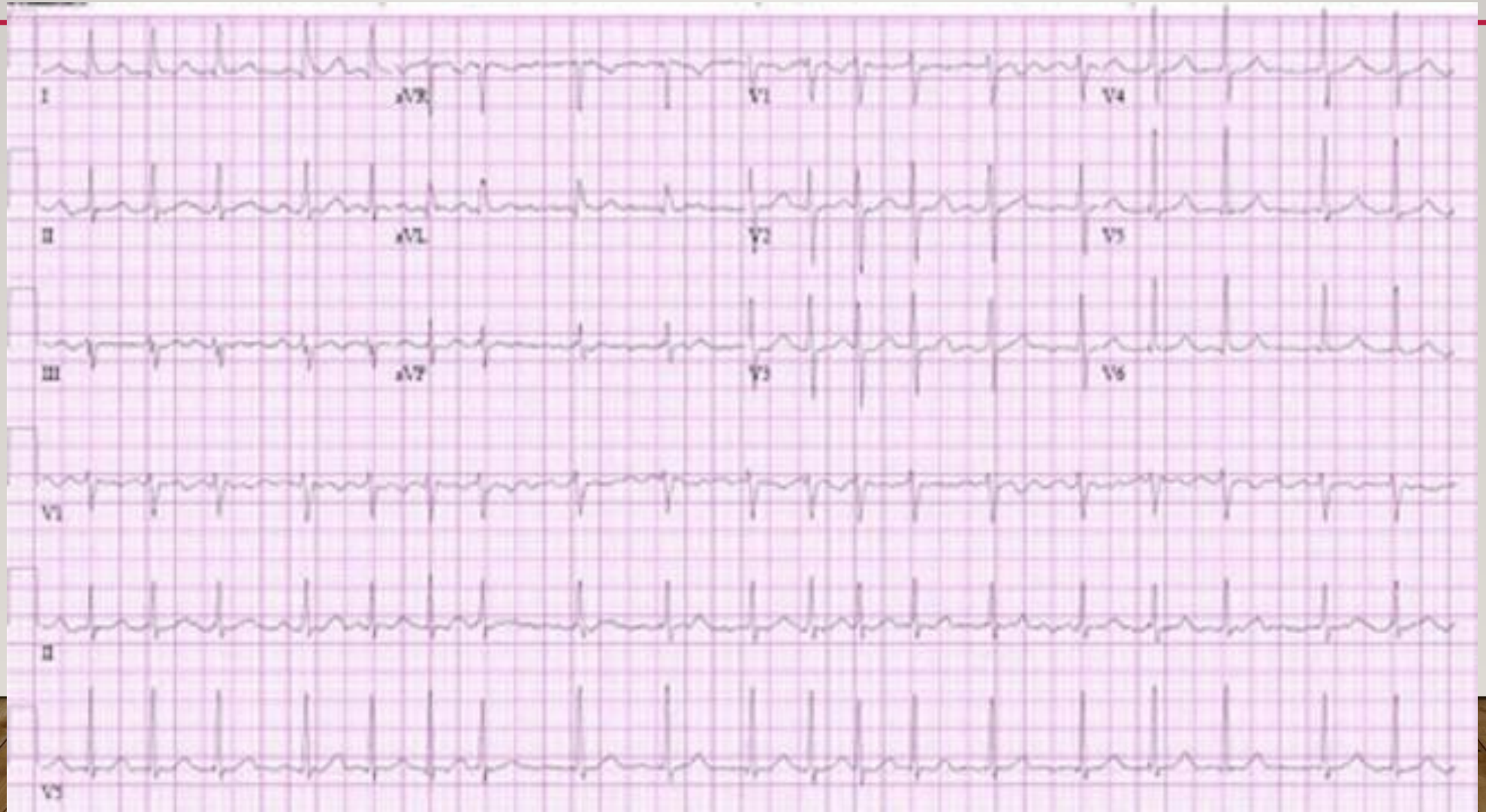
- Implantable cardioverter defibrillator (ICD) insertion is needed in patients with recurrent syncope despite beta-blockers, or those who are survivors of cardiac arrest, especially in the setting of coronary artery disease.
- ICDs should not be implanted without concomitant betablocker therapy, as ICD shocks will increase catecholamine surge, potentially leading to a vicious cycle of ventricular arrhythmias and ICD shocks.
- ICD therapy provides a continuous monitor for the cardiac rhythm and the capability of terminating ventricular tachycardia by overdrive pacing and/or cardioversion defibrillation.
- ICD implant requires surgery and is associated with a small risk of procedural mortality.
- ICD shocks can be painful and if frequent may impair the patient's quality of life.

	adjunct	temporary or permanent pacing
catecholaminergic polymorphic ventricular tachycardia		
	1st	beta-blockers
	adjunct	implantable cardioverter defibrillator

CASE HISTORY
ATRIAL FIBRILLATION

- A 65-year-old man with a history of hypertension, diabetes mellitus, and hyperlipidaemia presents to the accident and emergency department with the first episode of rapid palpitations, shortness of breath, and discomfort in his chest.
- His symptoms came on suddenly 4 hours ago.
- Physical examination shows an irregularly irregular radial pulse rate at 90 to 110 bpm, a BP of 110/70 mmHg, and respiratory rate of 20 breaths per minute.
- Heart sounds are irregular, but no S3 or S4 gallop or murmurs are audible.
- There are no other abnormalities on examination.

CASE HISTORY
ATRIAL FIBRILLATION



TREATMENT ALGORITHM

haemodynamically unstable		
	1st	emergency electrical cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	consider	amiodarone
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset <48 hours		
	1st	rate control
	plus	consider early rhythm control
	plus	longer-term anticoagulation strategy

- Electrical cardioversion quickly and effectively converts AF to sinus rhythm.
- Electrical cardioversion restores sinus rhythm quicker and more effectively than pharmacological cardioversion and is associated with shorter hospital stays.
- Do not use pharmacological cardioversion in haemodynamically compromised patients.
- Urgently admit to an acute medical unit.
- Call for anaesthetic support to sedate the patient before DC cardioversion.
- This will usually be with a short-acting general anaesthetic.
- Record and store an ECG rhythm strip during and immediately after shock delivery.
- Continuously monitor the patient's BP and oximetry during the procedure.

TREATMENT ALGORITHM

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
consider	amiodarone
plus	longer-term anticoagulation strategy

haemodynamically stable: onset <48 hours

- Check the patient's oral anticoagulation status as soon as possible.
- In patients not already on therapeutic anticoagulation, immediately start anticoagulation precardioversion.
- Use a low molecular weight heparin (LMWH), such as enoxaparin, or unfractionated heparin.
- This is important to prevent potential thromboembolic complications and should be given in a timely manner even in haemodynamically unstable patients.
- After cardioversion, transition patients who are started on a LMWH or unfractionated heparin to a DOAC, such as rivaroxaban, apixaban, edoxaban, or dabigatran, or warfarin when appropriate.

plus longer-term anticoagulation strategy

TREATMENT ALGORITHM

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
consider	amiodarone

- Identify and manage risk factors and concomitant conditions.
- Correct, where possible, treatable causes of AF or refer, as appropriate.
- In practice, refer to a cardiologist, any patient:
  - Who is young and has suspected underlying structural heart disease
  - With a pre-excitation syndrome such as Wolff-Parkinson-White syndrome.
  - With valvular heart disease associated with AF
  - With suspected heart failure.
- Signs of stroke or heart failure may be subtle in some instances.
- Bear in mind that rhythm control is often unsuccessful in critically ill patients and those with severely impaired ventricular systolic function, because AF is often precipitated/ exacerbated by increased sympathetic tone, inotropes, and vasopressors.
- In these patients, work to identify and correct precipitating factors and secondary causes and optimise background treatment.

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
consider	amiodarone

- amiodarone: 5 mg/kg intravenously over 20-120 minutes initially, repeat infusion according to response, maximum 1200 mg/ day
- The European Society of Cardiology recommends 300 mg intravenously over 30-60 minutes initially, followed by 900-1200 mg intravenously over 24 hours.
- Consider intravenous amiodarone for acute control of heart rate in these patients.
- Do not use rate control drugs in people with AF with a pre-excitation syndrome such as Wolff-Parkinson-White syndrome.
- These drugs accelerate conduction down the accessory pathway to the ventricle putting the patient at risk of life-threatening arrhythmias, such as ventricular fibrillation and sudden death.
- AF is the second most common arrhythmia in WPWs, occurring in approximately 1/3 pts.
- Atrial activity predominantly conducts down the accessory pathway, causing ventricular pre-excitation.

haemodynamically unstable

1st emergency electrical cardioversion

- Apixaban: 2.5 to 5 mg orally twice daily OR Edoxaban: 30-60 mg orally once daily OR Rivaroxaban: 15-20 mg orally once daily OR Dabigatran: 110-150 mg orally twice daily OR
- Use a direct oral anticoagulant (DOAC) in preference to a vitamin K antagonist.
- Warfarin: 5-10 mg orally once daily initially, adjust dose according to target INR
- Use the CHA_2DS_2-VASc score to calculate stroke risk in all patients presenting with AF
- When indicated by the CHA_2DS_2-VASc score, start oral anticoagulation as soon as possible.
- Use the HAS-BLED score to assess the risk of a major bleed; Identify modifiable risk factors for bleeding, such as uncontrolled hypertension, harmful alcohol consumption, labile INR if the patient is on warfarin, concurrent use of medication and reversible causes of anaemia
- Do not use bleeding risk scores to exclude anticoagulant treatment; a high score should not rule out anticoagulation.
- Bleeding risk is dynamic and requires regular re-assessment; it should not be based on a single one-off assessment.
- For most people the benefit of anticoagulation outweighs the bleeding risk
- For people with an increased risk of bleeding the benefit of anticoagulation may not always outweigh the bleeding risk, and careful monitoring of bleeding risk is important.

TREATMENT ALGORITHM

haemodynamically stable: onset <48 hours

AF

1st rate control

- Bisoprolol: 1.25 mg orally OD initially, increase gradually, maximum 20 mg/day OR
- Metoprolol: 2.5 to 5 mg i.v. or 50 mg orally BID or TID, maximum 300 mg/day OR
- Esmolol: 500 μg/kg/min i.v. as a loading dose, maintenance dose 50-200 μg/kg/min OR
- Carvedilol: 3.125 mg orally TID initially, maximum 50 mg/day OR
- Verapamil: 5-10 mg i.v.; 40-120 mg orally TID initially, maximum 480 mg OR
- Diltiazem: 60 mg orally TID initially, maximum 360 mg OR
- Digoxin: 0.75-1.5 mg/day orally for rapid digitalisation, then 0.125-0.25 mg/day
- Use rate control to slow the patient's heart rate in the presence of tachycardia.
- Use atrioventricular nodal blocking drugs – either a standard beta-blocker, or a rate-limiting non-dihydropyridine calciumchannel blocker – as initial monotherapy.
- Base the choice of drug on the person's symptoms, heart rate, comorbidities, and preferences when considering drug treatment.
- If monotherapy does not control symptoms, consider combination therapy
- Do not use rate control drugs in people with AF with a pre-excitation syndrome such as Wolff-Parkinson-White syndrome.

TREATMENT ALGORITHM

haemodynamically stable: onset <48 hours

AF

1st rate control

plus consider early rhythm control

consider pre-cardioversion anticoagulation

- Flecainide: 200-300 mg orally as a single dose OR
- Amiodarone: 5-7 mg/kg i.v. over 1-2 hours, followed by 50 mg/hour i.v., max 1200 mg/day
- Start anticoagulation precardioversion as soon as possible
- In patients with AF duration 24 to 48 hours who are undergoing cardioversion, initiate oral anticoagulation for at least 4 weeks after cardioversion.
- This is an optional step for those with AF onset definitely less than 24 hours.
- Consider either pharmacological or electrical cardioversion depending on clinical circumstances, patient preferences, and resources, to reduce symptoms.
- Electrical cardioversion restores sinus rhythm quicker and more effectively than pharmacological cardioversion and is associated with shorter hospital stays.
- Pharmacological cardioversion, however, does not require fasting or sedation.
- A choice of flecainide or amiodarone to people with no evidence of structural or ischaemic heart disease OR
- Amiodarone to people with evidence of structural heart disease

TREATMENT ALGORITHM

AF

haemodynamically stable: onset <48 hours		
	1st	rate control
	plus	consider early rhythm control
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset ≥48 hours or uncertain		

- Apixaban: 2.5 to 5 mg orally twice daily OR
- Edoxaban: 30-60 mg orally once daily OR
- Rivaroxaban: 15-20 mg orally once daily OR Dabigatran: 110-150 mg orally twice daily OR
- Use a direct oral anticoagulant (DOAC) in preference to a vitamin K antagonist.
- Warfarin: 5-10 mg orally once daily initially, adjust dose according to target INR

TREATMENT ALGORITHM

haemodynamically stable: onset <48 hours		
	1st	**rate control**
	plus	**consider early rhythm control**
	consider	**pre-cardioversion anticoagulation**
	plus	**treat underlying cause**
	plus	**longer-term anticoagulation strategy**
haemodynamically stable: onset ≥48 hours or uncertain		
	1st	**rate control**
	plus	**elective electrical or pharmacological cardioversion**
	consider	**pre-cardioversion anticoagulation**
	plus	**treat underlying cause**
	plus	**longer-term anticoagulation strategy**

TREATMENT ALGORITHM

AF

haemodynamically stable: onset <48 hours

1st	rate control
plus	consider early rhythm control
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
plus	longer-term anticoagulation strategy

haemodynamically stable: onset ≥48 hours or uncertain

- Apixaban: 2.5 to 5 mg orally twice daily OR
- Edoxaban: 30-60 mg orally once daily OR
- Rivaroxaban: 15-20 mg orally once daily OR Dabigatran: 110-150 mg orally twice daily OR
- Use a direct oral anticoagulant (DOAC) in preference to a vitamin K antagonist.
- Warfarin: 5-10 mg orally once daily initially, adjust dose according to target INR

TREATMENT ALGORITHM

haemodynamically unstable

- Bisoprolol: 1.25 mg orally OD initially, increase gradually, maximum 20 mg/day OR
- Metoprolol: 2.5 to 5 mg i.v. or 50 mg orally BID or TID, maximum 300 mg/day OR
- Esmolol: 500 µg/kg/min i.v. as a loading dose, maintenance dose 50-200 µg/kg/min OR
- Carvedilol: 3.125 mg orally TID initially, maximum 50 mg/day OR
- Verapamil: 5-10 mg i.v.; 40-120 mg orally TID initially, maximum 480 mg OR
- Diltiazem: 60 mg orally TID initially, maximum 360 mg OR
- Digoxin: 0.75-1.5 mg/day orally for rapid digitalisation, then 0.125-0.25 mg/day

plus	consider early rhythm control
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
plus	longer-term anticoagulation strategy

haemodynamically stable: onset ≥48 hours or uncertain

1st	rate control
plus	elective electrical or pharmacological cardioversion
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
plus	longer-term anticoagulation strategy

TREATMENT ALGORITHM

- If the onset of AF is 48 hours or more, or is uncertain:
- Make a plan for therapeutic anticoagulation for a minimum of 3 weeks followed by elective electrical or pharmacological cardioversion
- Continue therapeutic anticoagulation for 4 weeks after cardioversion (in patients without a need for long-term anticoagulation).
- If the patient is already on therapeutic anticoagulation, proceed with cardioversion.

haemodynamically unstable	
1st	rate control
plus	consider early rhythm control
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
plus	longer-term anticoagulation strategy
haemodynamically stable: onset ≥48 hours or uncertain	
1st	rate control
plus	elective electrical or pharmacological cardioversion
consider	pre-cardioversion anticoagulation
plus	treat underlying cause
plus	longer-term anticoagulation strategy

TREATMENT ALGORITHM

haemodynamically unstable		
	1st	emergency electrical cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	consider	amiodarone
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset <48 hours		
	1st	rate control
	plus	consider early rhythm control
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset ≥48 hours or uncertain		
	1st	rate control
	plus	elective electrical or pharmacological cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy

TREATMENT ALGORITHM

haemodynamically unstable		
	1st	emergency electrical cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	consider	amiodarone
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset <48 hours		
	1st	rate control
	plus	consider early rhythm control
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset ≥48 hours or uncertain		
	1st	rate control
	plus	elective electrical or pharmacological cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy

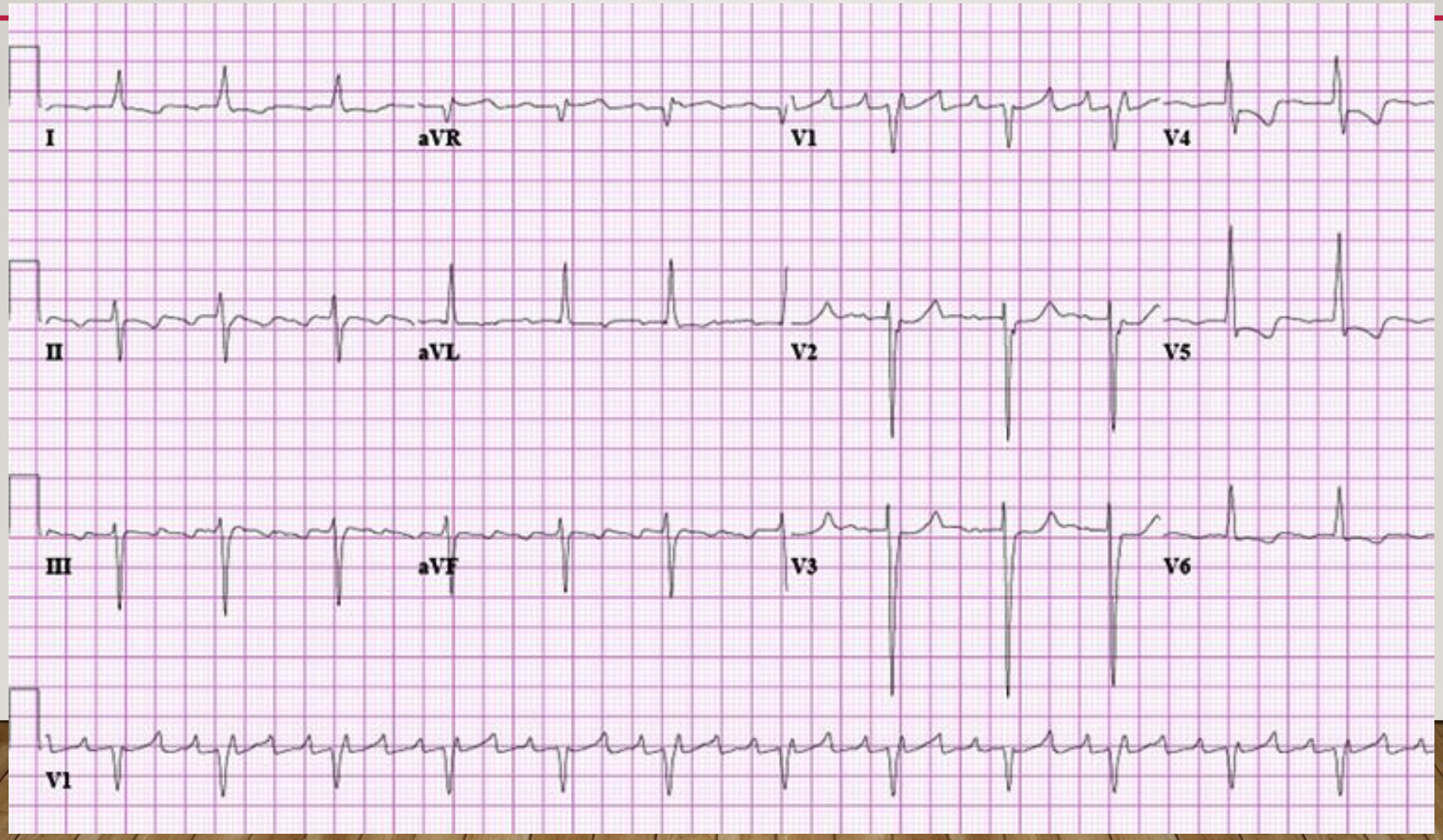
TREATMENT ALGORITHM

haemodynamically unstable		
	1st	emergency electrical cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	consider	amiodarone
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset <48 hours		
	1st	rate control
	plus	consider early rhythm control
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy
haemodynamically stable: onset ≥48 hours or uncertain		
	1st	rate control
	plus	elective electrical or pharmacological cardioversion
	consider	pre-cardioversion anticoagulation
	plus	treat underlying cause
	plus	longer-term anticoagulation strategy

CASE HISTORY
ATRIAL FLUTTER

- A 77-year-old man presents with complaints of palpitations and new shortness of breath, especially with exertion.
- He has a history of rheumatic fever in childhood.
- He has been told he has a murmur but does not recall having had an echocardiogram.
- He is otherwise healthy.

CASE HISTORY
ATRIAL FLUTTER



TREATMENT ALGORITHM

Initial (summary)

haemodynamically unstable

1st emergency electrical cardioversion

- Urgently identify any patient with features of haemodynamic instability, which include:
  - Shock
  - Syncope
  - Myocardial ischaemia
  - Acute, severe heart failure (characterised by acute pulmonary oedema or raised jugular venous pressure)
- Organise immediate synchronised direct current (DC) cardioversion.
- Call for anaesthetic support to ensure any conscious patient has sedation or general anaesthesia.
- Give up to three shocks.
- For the initial shock, use 70 to 120 J of energy.
- Give subsequent shocks using stepwise increases in energy.

TREATMENT ALGORITHM

Initial (summary)

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	supportive care
plus	long-term anticoagulation strategy

- If the patient has new-onset atrial flutter and is receiving no, or subtherapeutic, anticoagulation, start initial parenteral anticoagulation and continue this until you have made a full assessment.
- Unfractionated heparin or a low molecular weight heparin (LMWH) such as enoxaparin are options.
- Transition patients who are started on unfractionated heparin or an LMWH to a direct oral anticoagulant (e.g., rivaroxaban, apixaban, edoxaban, or dabigatran), or warfarin, when appropriate, and after assessment of stroke and bleeding risk.

TREATMENT ALGORITHM

Initial (summary)

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	supportive care
plus	long-term anticoagulation strategy

- While assessing the patient, start resuscitation measures.
- Monitor controlled oxygen therapy.
- An upper SpO2 limit of 96% is reasonable when administering supplemental oxygen to most patients with acute illness who are not at risk of hypercapnia.
- A lower target SpO2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.
- Monitor the ECG and blood pressure.
- Obtain intravenous access.
- Identify and treat any reversible causes (e.g., electrolyte abnormalities).

TREATMENT ALGORITHM

Initial (summary)

haemodynamically unstable

1st	emergency electrical cardioversion
consider	pre-cardioversion anticoagulation
plus	supportive care
plus	long-term anticoagulation strategy

- Treatment recommended for ALL patients in selected patient group
- Apixaban: 2.5 to 5 mg orally twice daily OR
- Edoxaban: 30 – 60 mg orally once daily OR
- Rivaroxaban: 15-20 mg orally once daily OR
- Dabigatran: 110-150 mg orally twice daily OR
- Warfarin: 5-10 mg orally once daily initially, adjust dose according to target INR