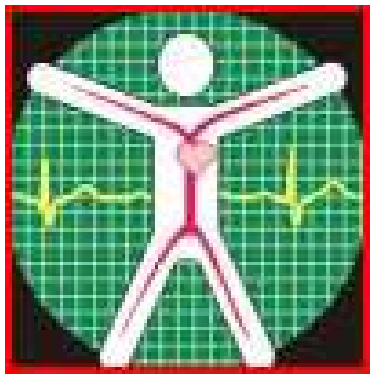
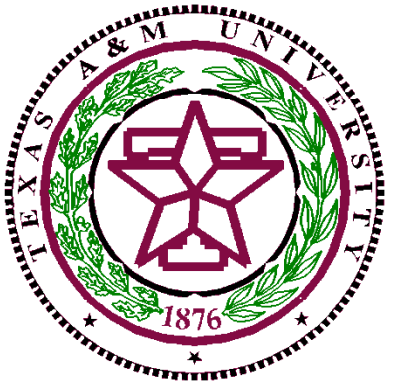


KINE 639 - Dr. Green

Section 2



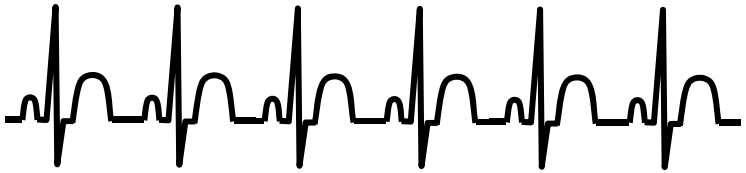
Rhythm

Rhythm Disturbances, Conduction Disturbances, Ischemia and Infarction

Rhythms from the Sinus Node

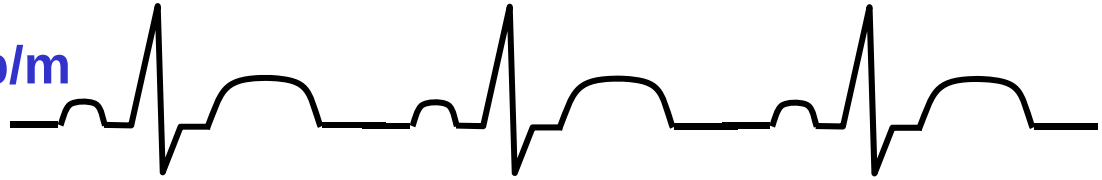
Normal Sinus Rhythm (NSR)

- **Sinus Tachycardia: HR > 100 b/m**



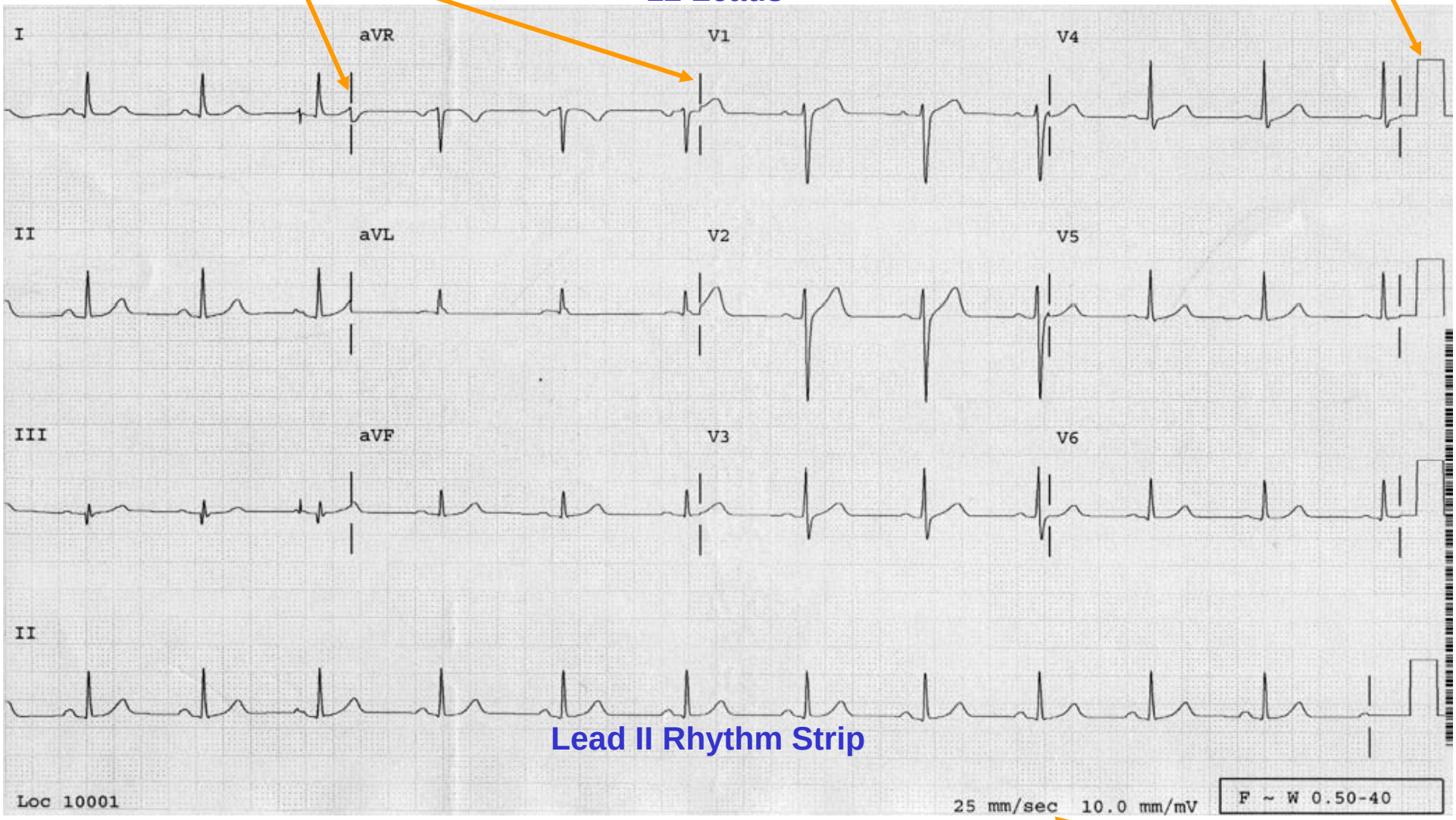
  - Causes:
    - Withdrawal of vagul tone & Sympathetic stimulation (**exercise**, fight or flight)
    - Fever & inflammation
    - Heart Failure or Cardiogenic Shock (both represent hypoperfusion states)
    - Heart Attack (myocardial infarction or extension of infarction)
    - Drugs (alcohol, nicotine, caffeine)

- **Sinus Bradycardia: HR < 60 b/m**



  - Causes:
    - Increased vagul tone, decreased sympathetic output, (**endurance training**)
    - Hypothyroidism
    - Heart Attack (common in inferior wall infarction)
    - Vasovagul syncope (people passing out when they get their blood drawn)
    - Depression

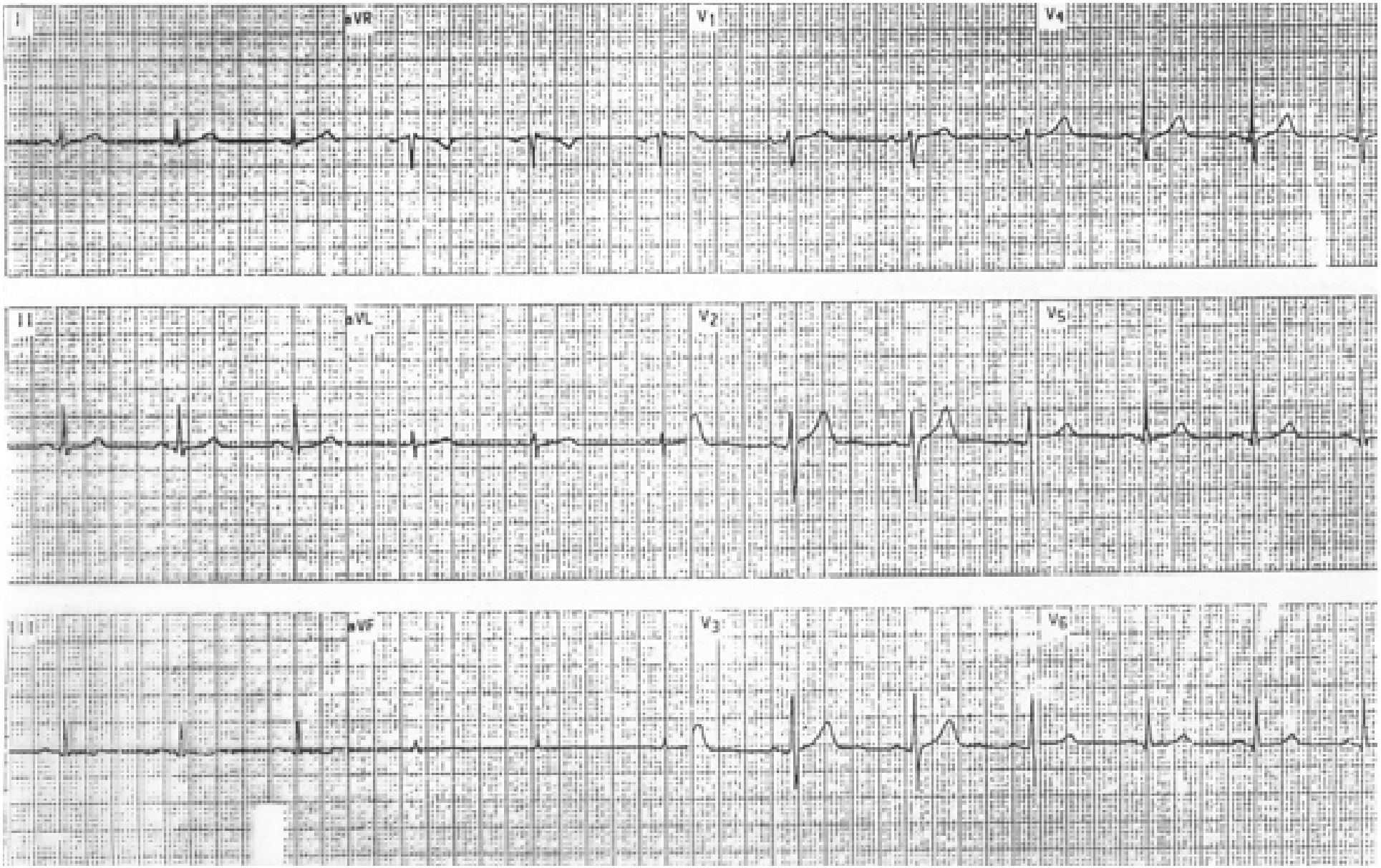
Normal 12 ECG with Lead II Rhythm Strip



Rate: **PRI:** **QRS:** **QT:**

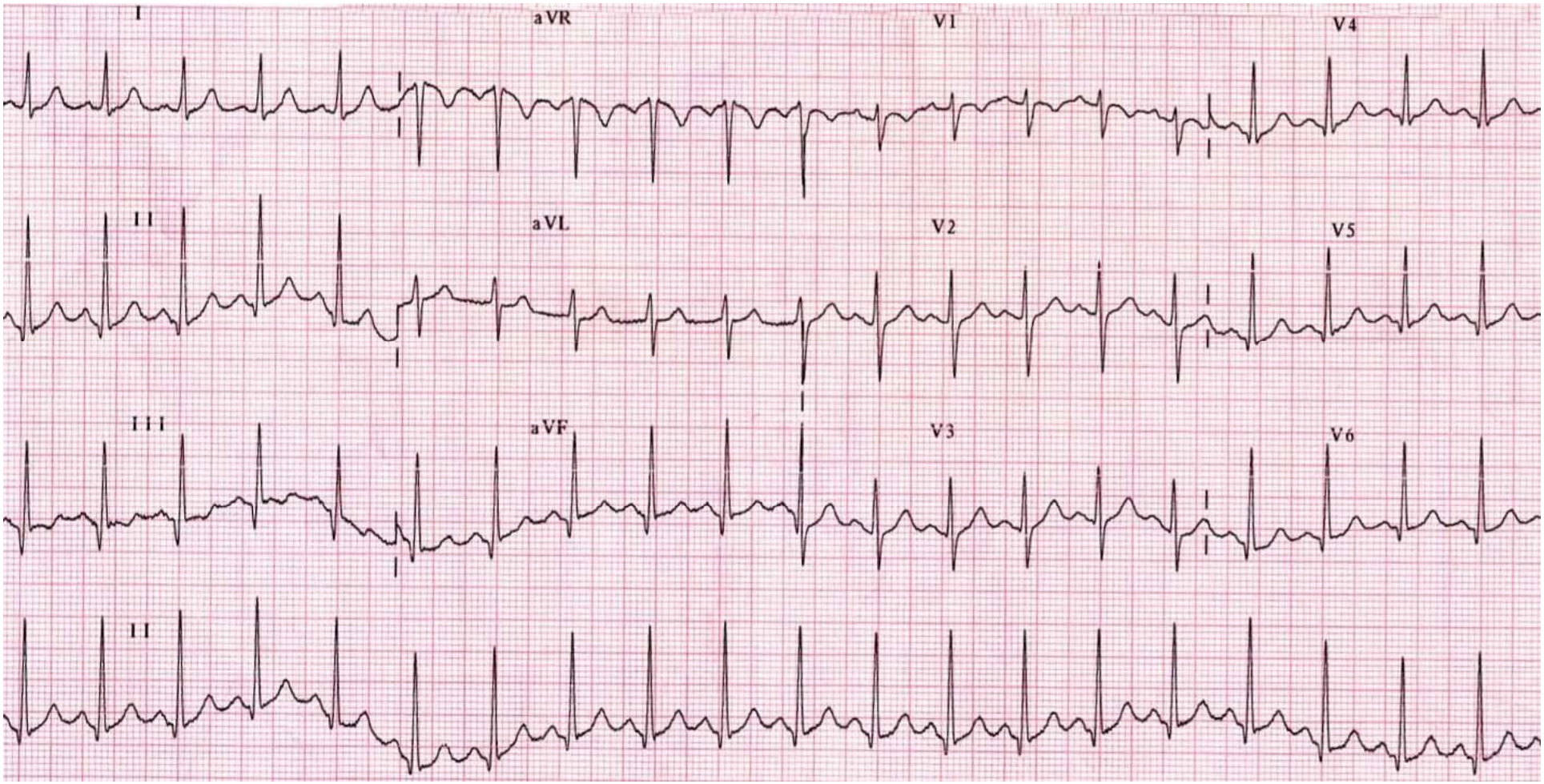
Axis:

Normal 12 Lead ECG

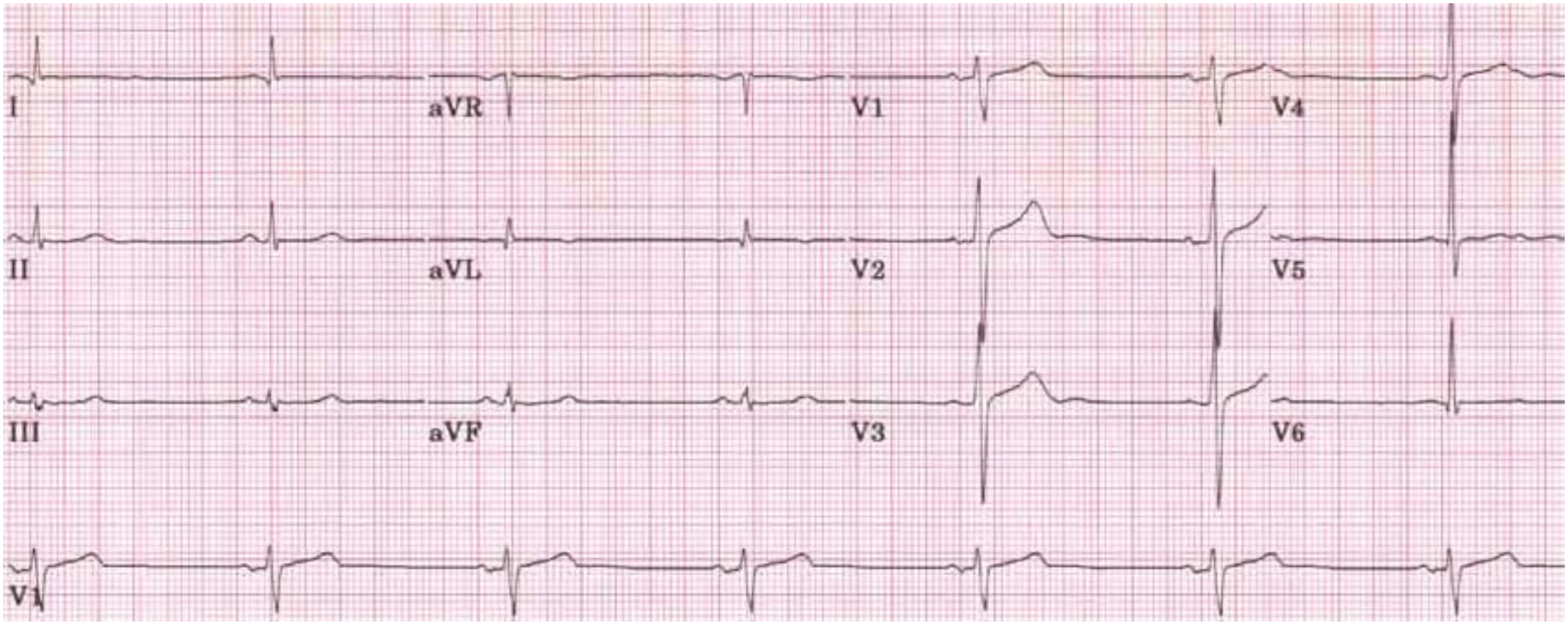


Sinus Tachycardia

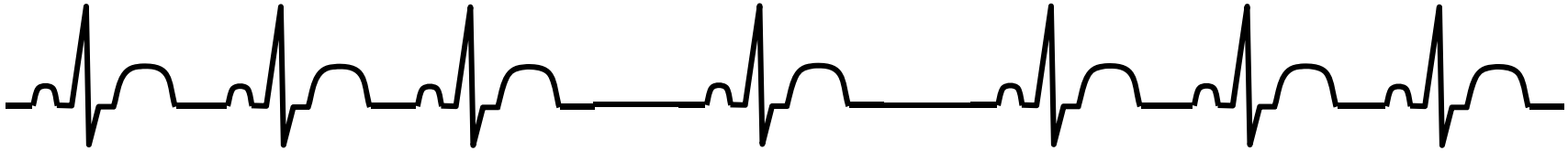
Healthy Exercising Male Runner



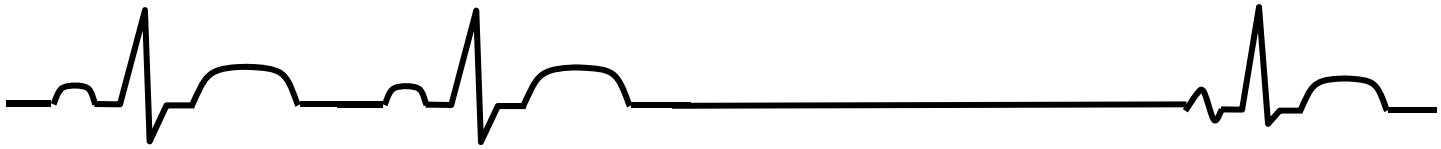
Sinus Bradycardia (V1 Rhythm Strip)



Rhythms from the Sinus Node



- **Sinus Arrhythmia: Variation in HR by more than .16 seconds**
  - Mechanism:
    - Most often: changes in vagul tone associated with respiratory reflexes
    - Benign variant
  - Causes
    - Most often: youth and **endurance training**



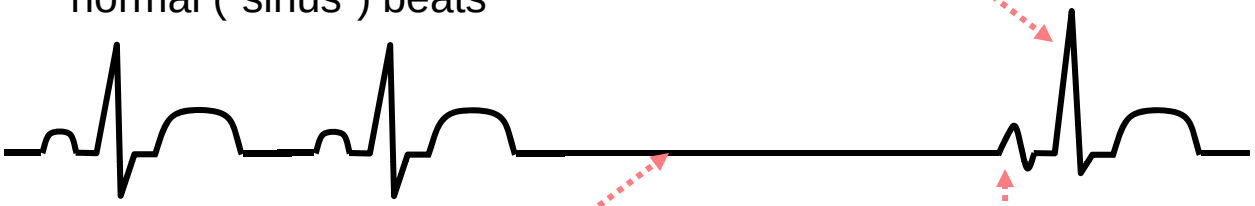
- **Sick Sinus Syndrome: Failure of the heart's pacemaking capabilities**
  - Causes:
    - Idiopathic (no cause can be found)
    - Cardiomyopathy (disease and malformation of the cardiac muscle)
  - Implications and Associations
    - Associated with Tachycardia / Bradycardia arrhythmias
    - Is often followed by an **ectopic "escape beat"** or an **ectopic "rhythm"**

Recognizing and Naming Beats & Rhythms

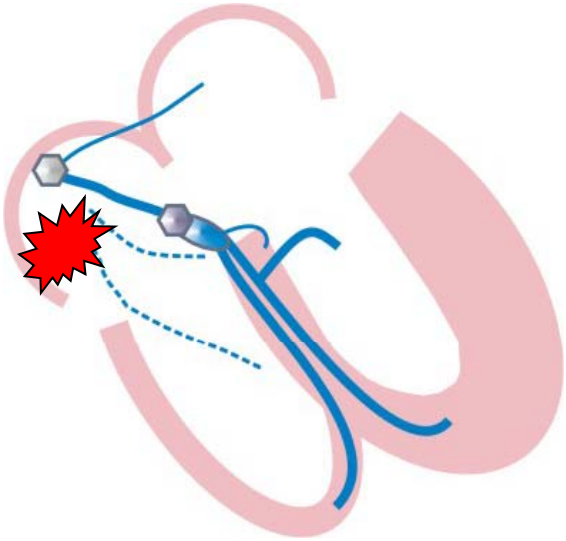
Atrial Escape Beat

QRS is slightly different but still narrow, indicating that conduction through the ventricle is relatively normal

normal ("sinus") beats



sinus node doesn't fire leading to a period of asystole (sick sinus syndrome). The atria finally fires an “escape beat”.

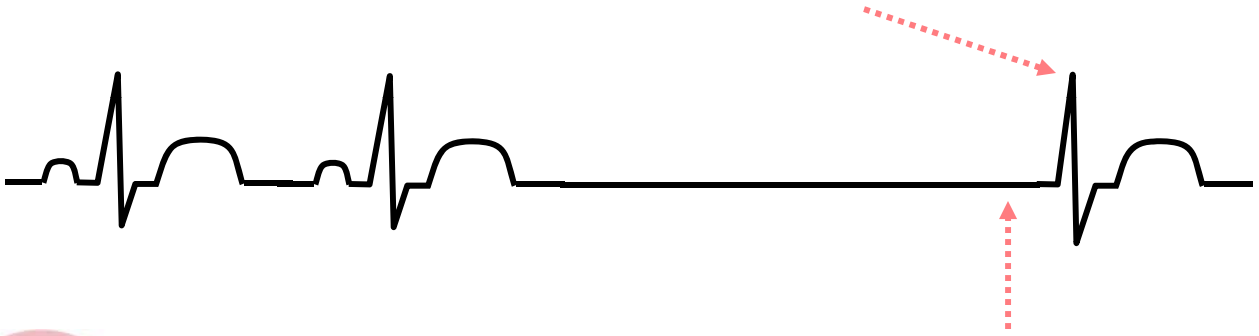


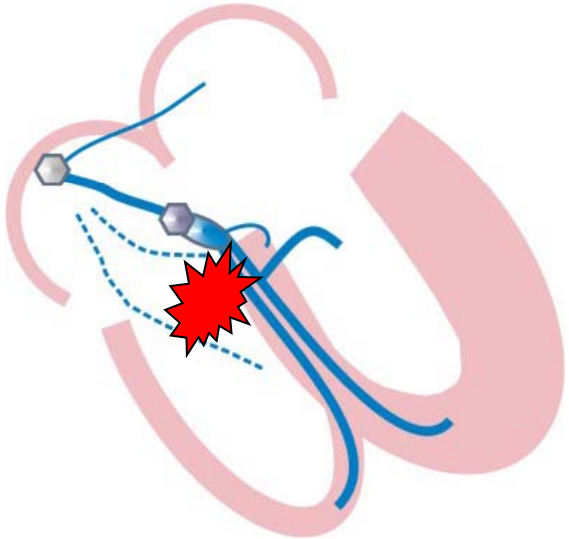
p-wave has different shape indicating it did not originate in the sinus node, but somewhere in the atria. It is therefore called an "atrial" beat

Recognizing and Naming Beats & Rhythms

Junctional Escape Beat

QRS is slightly different but still narrow, indicating that conduction through the ventricle is relatively normal



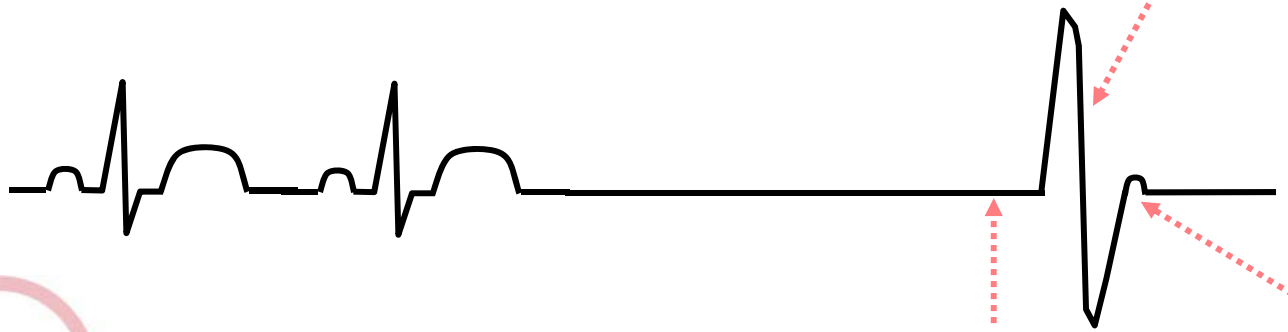


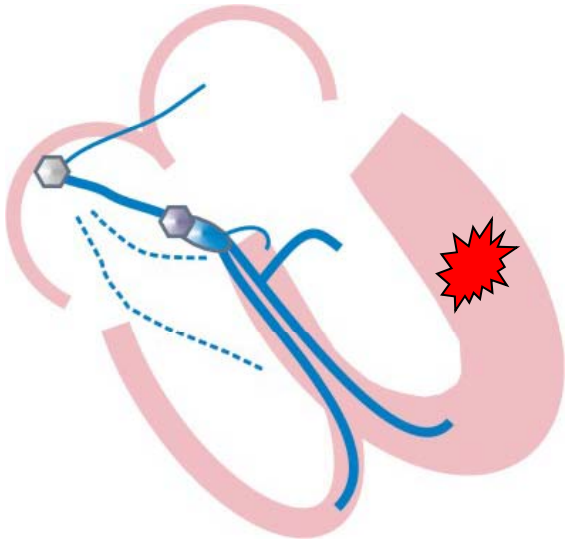
there is no p wave, indicating that the beat did not originate anywhere in the atria, but since the QRS complex is still thin and normal looking, we can conclude that conduction through the ventricles was normal and that the beat originated somewhere near the AV junction. The beat is therefore called a "junctional" beat

Recognizing and Naming Beats & Rhythms

Ventricular Escape Beat

QRS is wide and much different ("bizarre") looking than the normal beats. This indicates that the beat originated somewhere in the ventricles and consequently, conduction through the ventricles did not take place through normal pathways. It is therefore called a "ventricular" beat and the conduction through the ventricles is termed "aberrant"





There is no p wave, indicating that the beat did not originate anywhere in the atria

Actually a "retrograde p-wave may sometimes be seen on the right hand side of beats that originate in the ventricles, indicating that depolarization has spread back up through the atria from the ventricles

Recognizing and Naming Beats & Rhythms

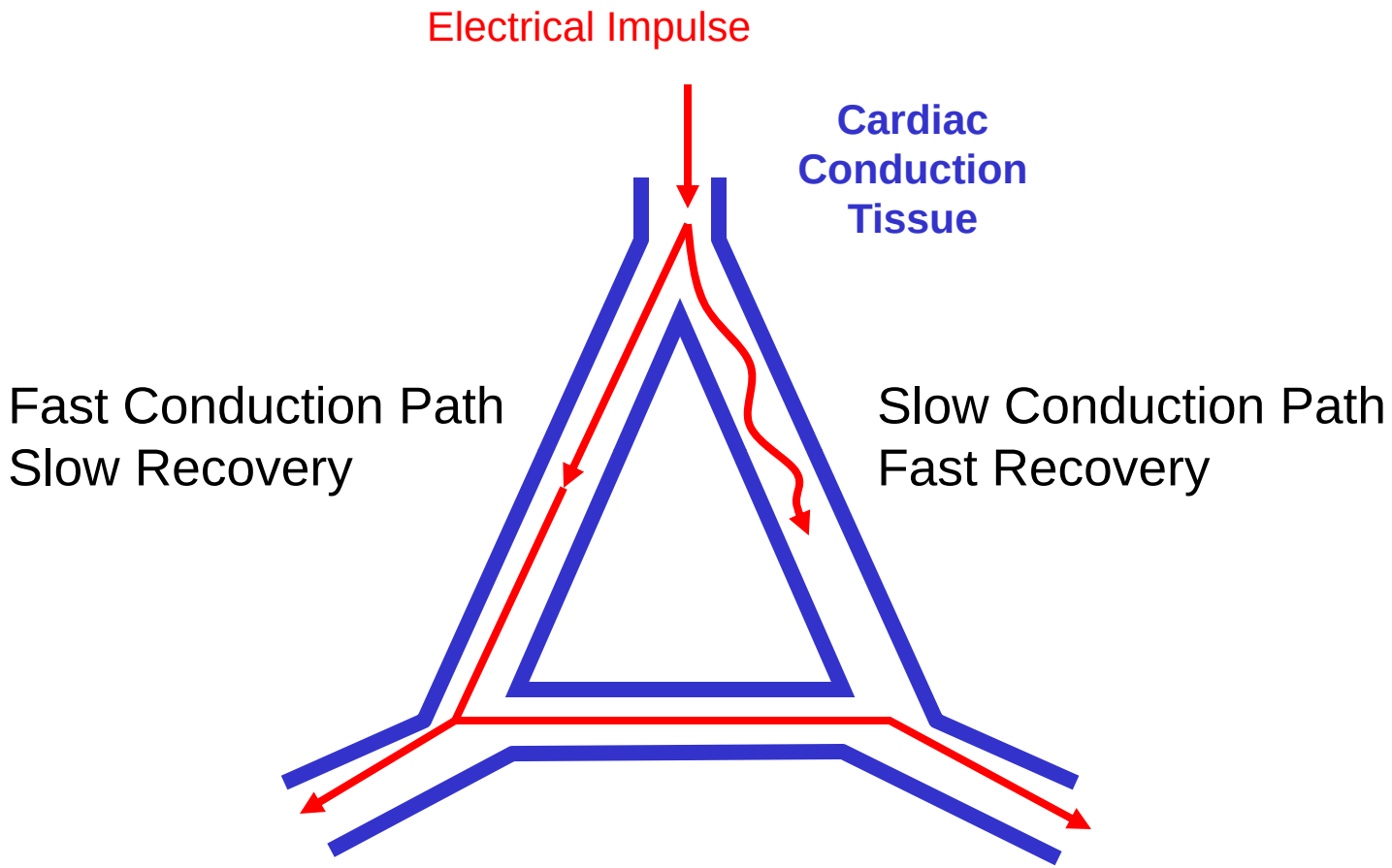
Ectopic Beats or Rhythms

- Beats or rhythms that originate in places other than the SA node
- The ectopic focus may cause **single beats** or take over and pace the heart, dictating its **entire rhythm**
- They may or may not be dangerous depending on **how they affect (or have the potential to affect) the cardiac output**

Causes of Ectopic Beats or Rhythms

- Hypoxic Myocardium - chronic pulmonary disease, pulmonary embolus
- Ischemic Myocardium - acute MI, expanding MI, angina
- Sympathetic Stimulation - nervousness, exercise, CHF, hyperthyroidism
- Drugs & electrolyte imbalances - antiarrhythmic drugs, hypokalemia, Caffeine, imbalances of calcium and magnesium
- Bradycardia - a slow HR predisposes one to arrhythmias
- Enlargement of the atria or ventricles producing stretch in pacemaker cells

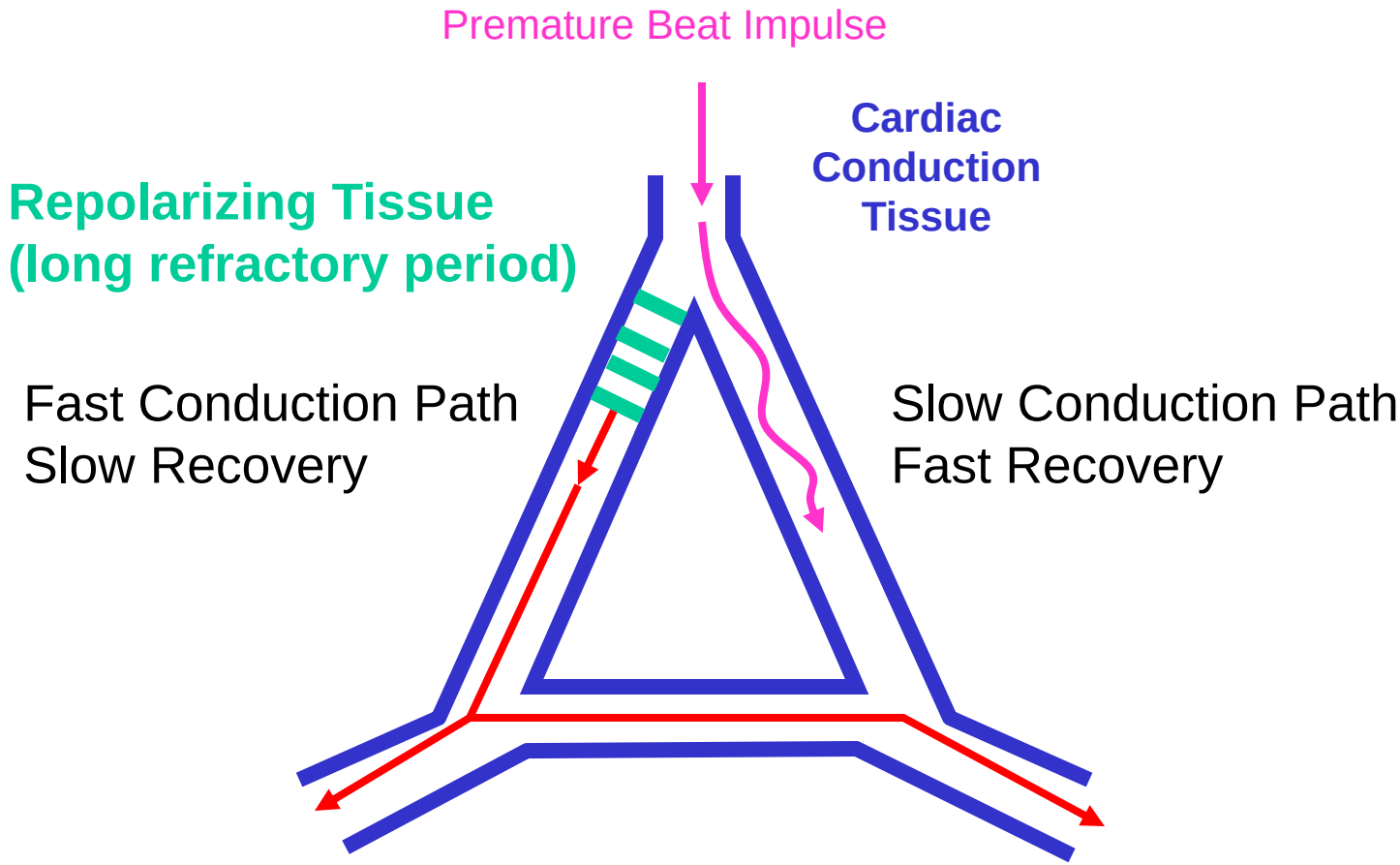
The “Re-Entry” Mechanism of Ectopic Beats & Rhythms



Tissues with these type of circuits may exist:

- **in microscopic size in the SA node, AV node, or any type of heart tissue**
- **in a “macroscopic” structure such as an accessory pathway in WPW**

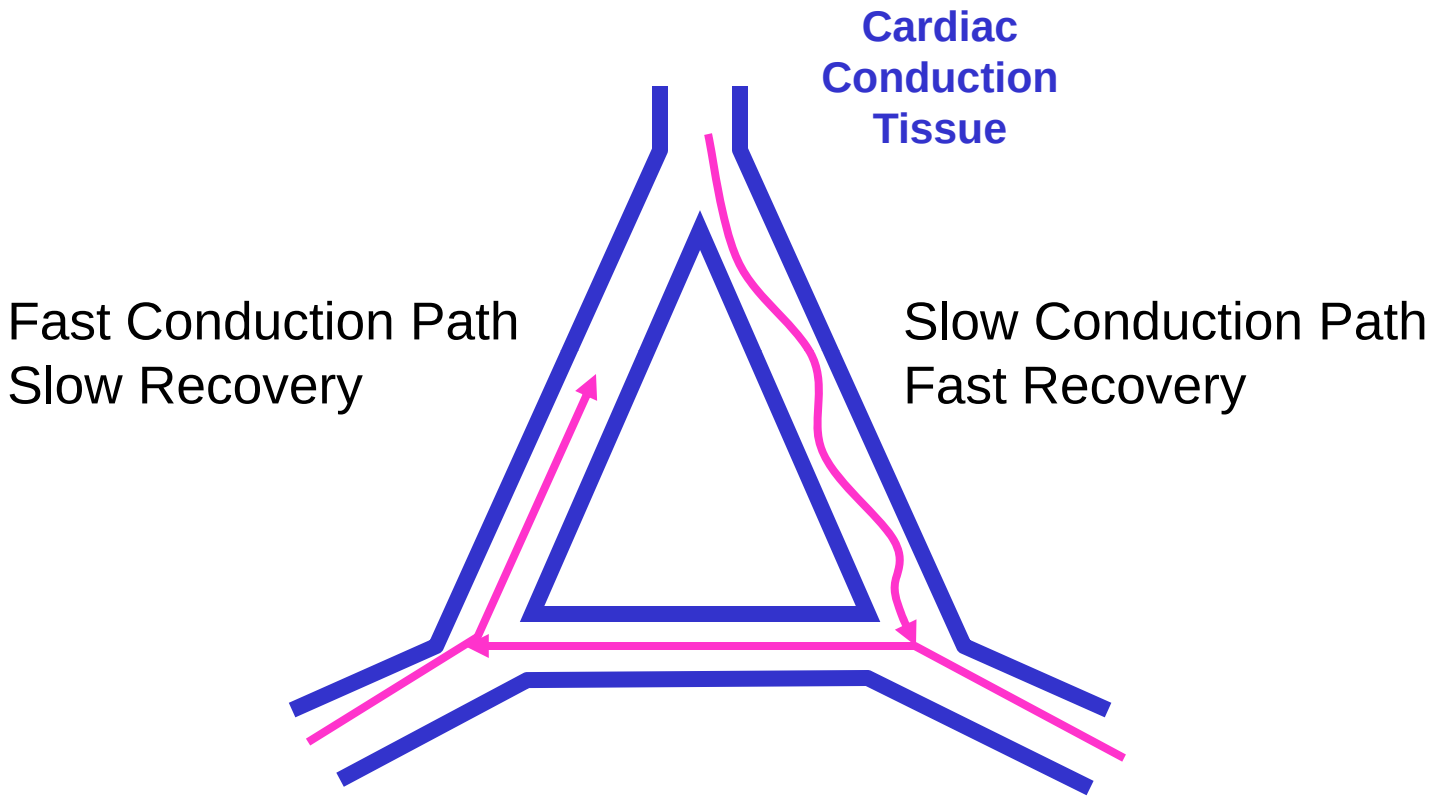
The “Re-Entry” Mechanism of Ectopic Beats & Rhythms



1. **An arrhythmia is triggered by a premature beat**

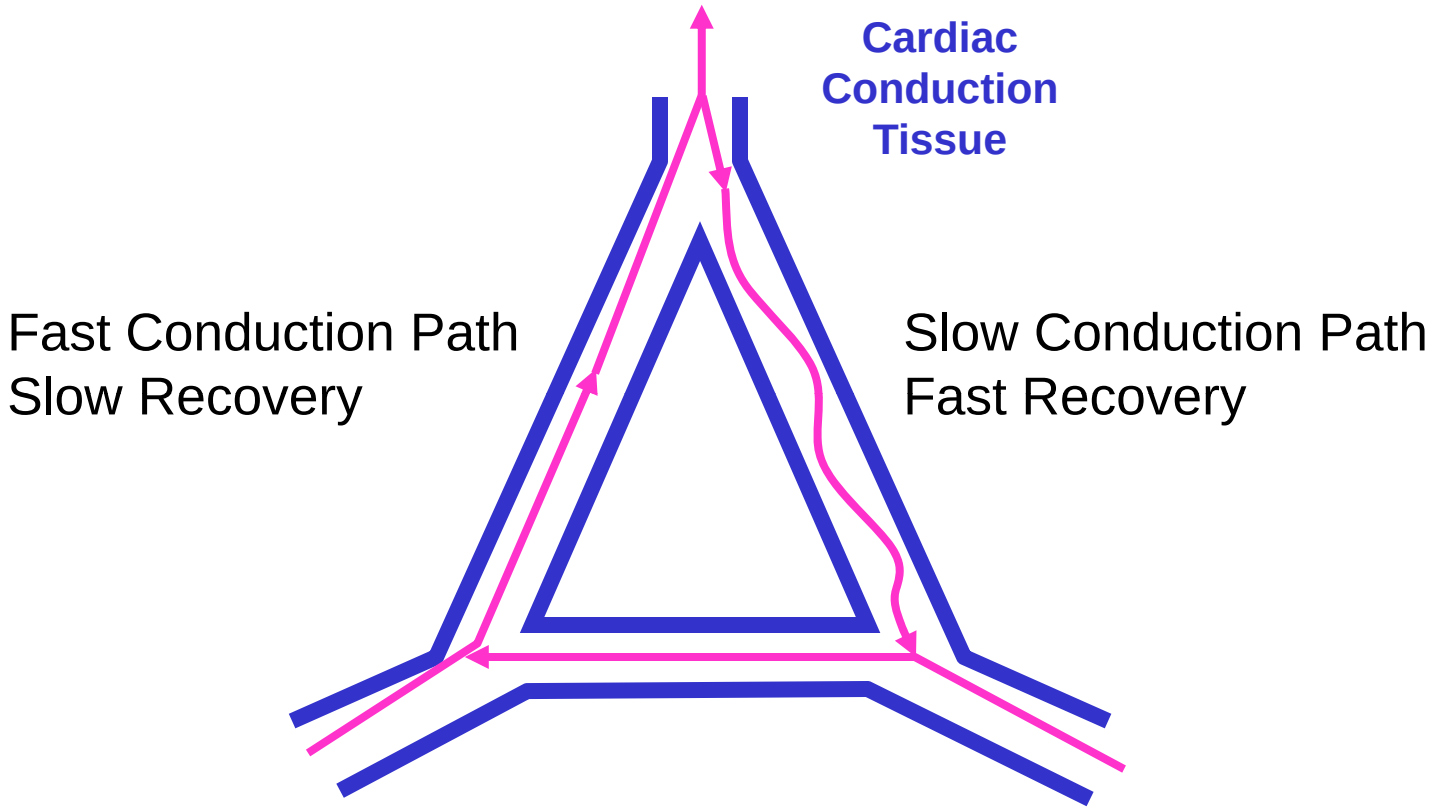
2. **The beat cannot gain entry into the fast conducting pathway because of its long refractory period and therefore travels down the slow conducting pathway only**

The “Re-Entry” Mechanism of Ectopic Beats & Rhythms



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

The “Re-Entry” Mechanism of Ectopic Beats & Rhythms



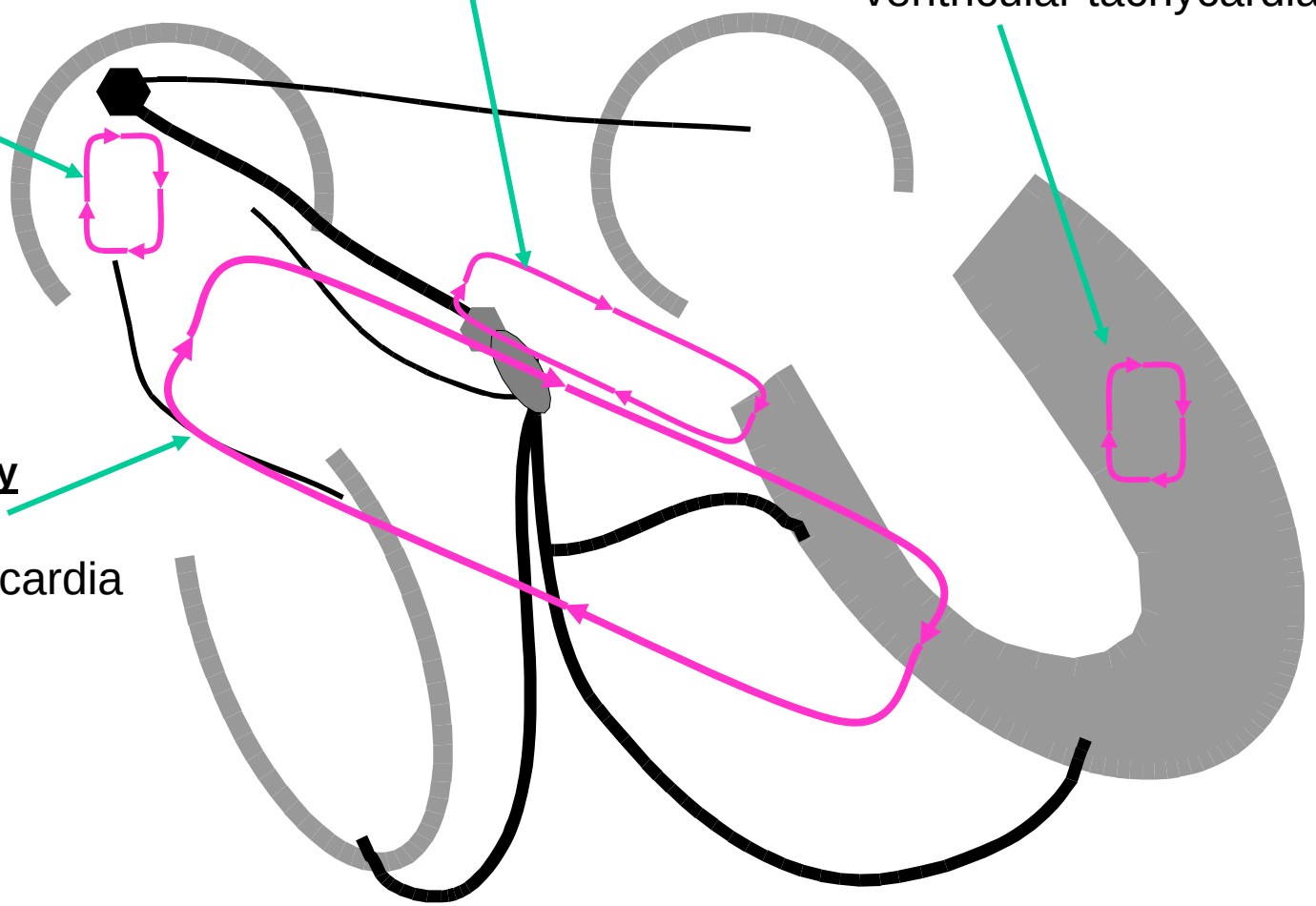
4. On arriving at the top of the fast pathway it finds the slow pathway has recovered and therefore the wave of excitation ‘re-enters’ the pathway and continues in a ‘circular’ movement. This creates the re-entry circuit

Re-entry Circuits as Ectopic Foci and Arrhythmia Generators

Atrio-Ventricular Nodal Re-entry
- supraventricular tachycardia

Atrial Re-entry
- atrial tachycardia
- atrial fibrillation
- atrial flutter

Ventricular Re-entry
- ventricular tachycardia



Atrio-Ventricular Re-entry
- Wolf Parkinson White
- supraventricular tachycardia

Recognizing and Naming Beats & Rhythms

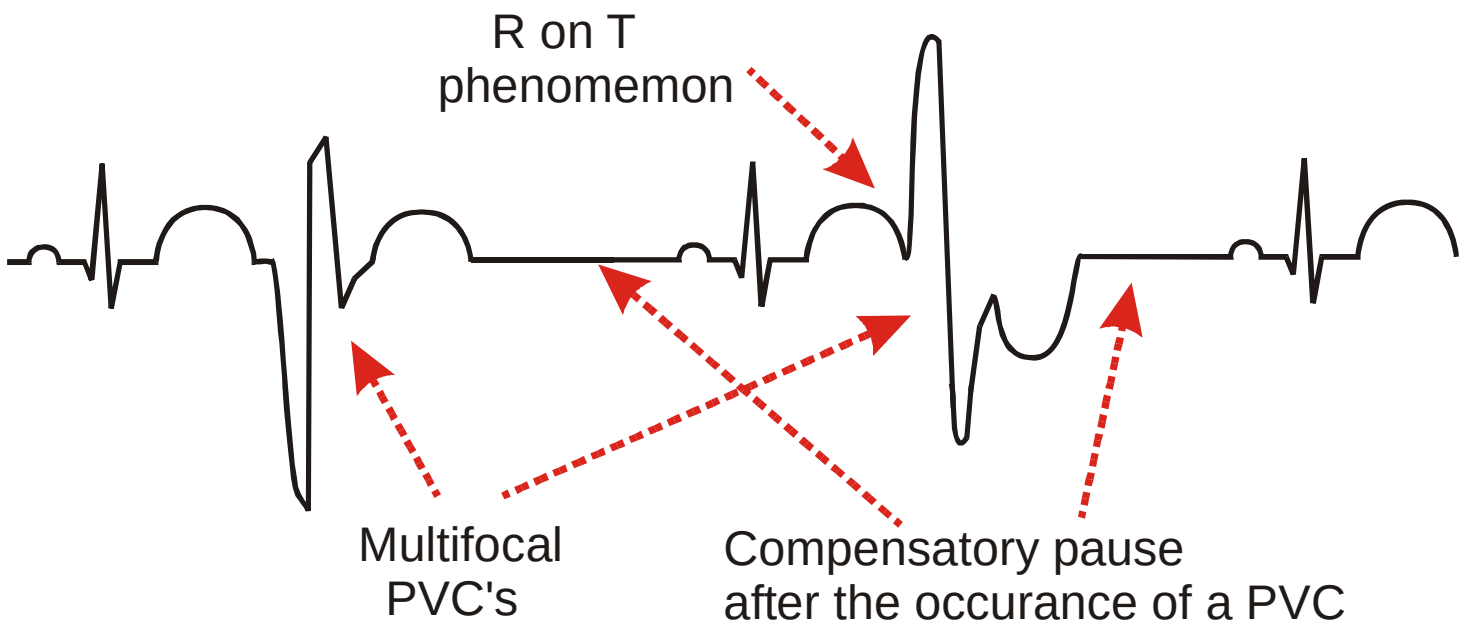
Clinical Manifestations of Arrhythmias

- Many go unnoticed and produce no symptoms
- Palpitations – ranging from “noticing” or “being aware” of ones heart beat to a sensation of the heart “beating out of the chest”
- If HR> 250 -300, $\dot{Q}$ is affected ➡ lightheadedness, syncope, fainting
- Rapid arrhythmias ⬆ myocardial O_2 demand ➡ ischemia ➡ angina
- Drug & electrolyte imbalances resulting from antiarrhythmic drugs, hypokalemia, or imbalances of calcium and magnesium
- Sudden death – especially in the case of an acute MI or aortic stenosis

Recognizing and Naming Beats & Rhythms

Premature Ventricular Contractions (PVC's, VPB's, extrasystoles):

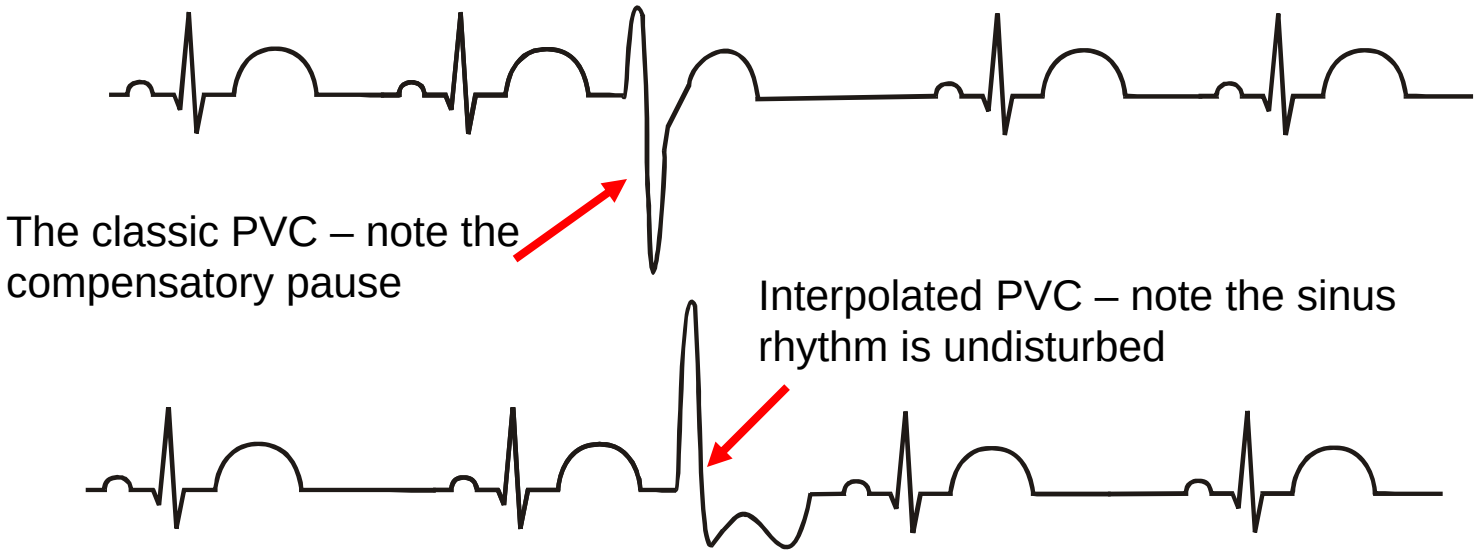
- A ventricular ectopic focus discharges causing an early beat
- Ectopic beat has no P-wave (maybe retrograde), and QRS complex is "wide and bizarre"
- QRS is wide because the spread of depolarization through the ventricles is abnormal (**aberrant**)
- In most cases, the heart circulates no blood (no pulse) because of an irregular squeezing motion
  - PVC's are sometimes described by lay people as "skipped heart beats"



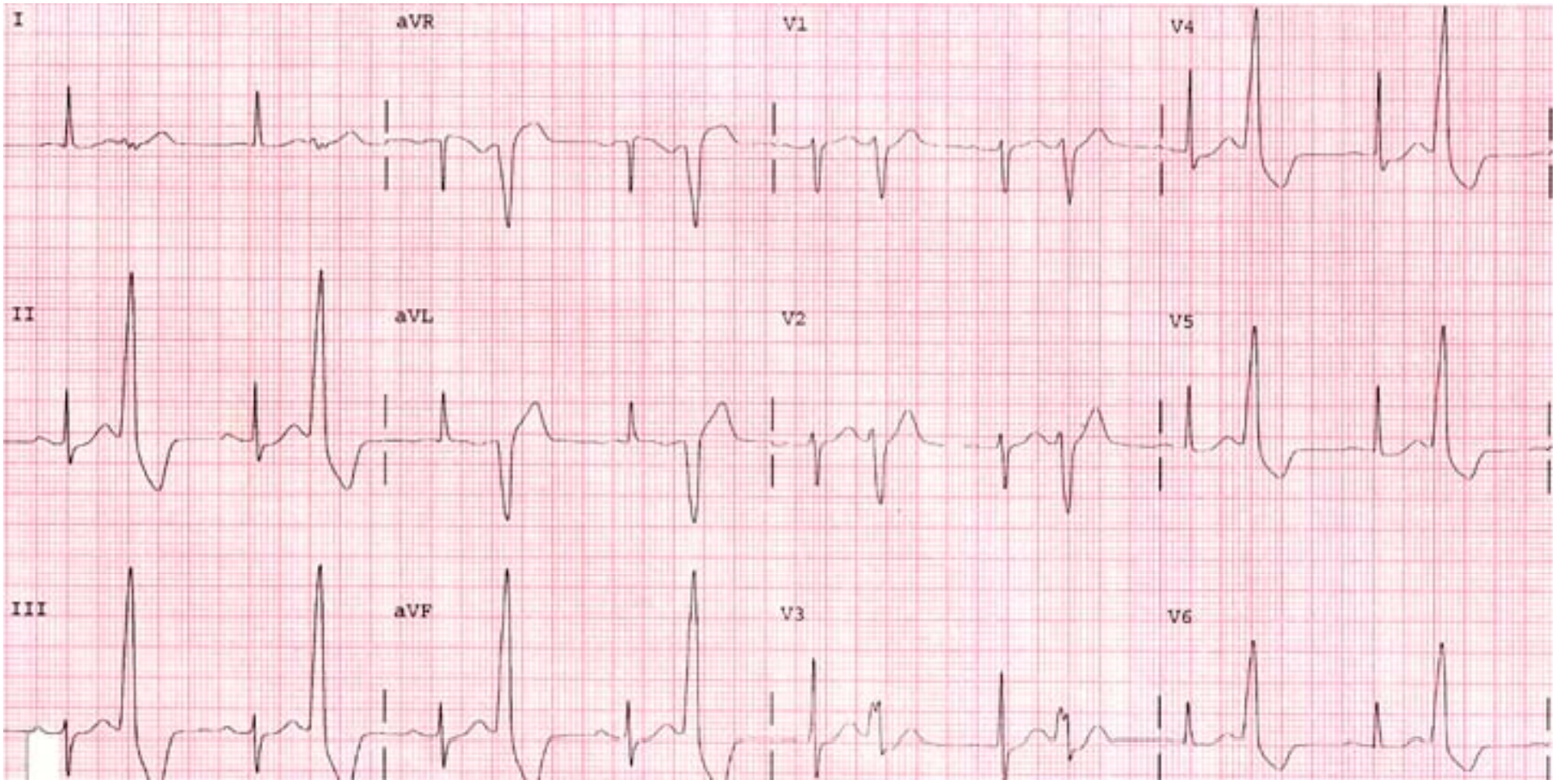
Recognizing and Naming Beats & Rhythms

Characteristics of PVC's

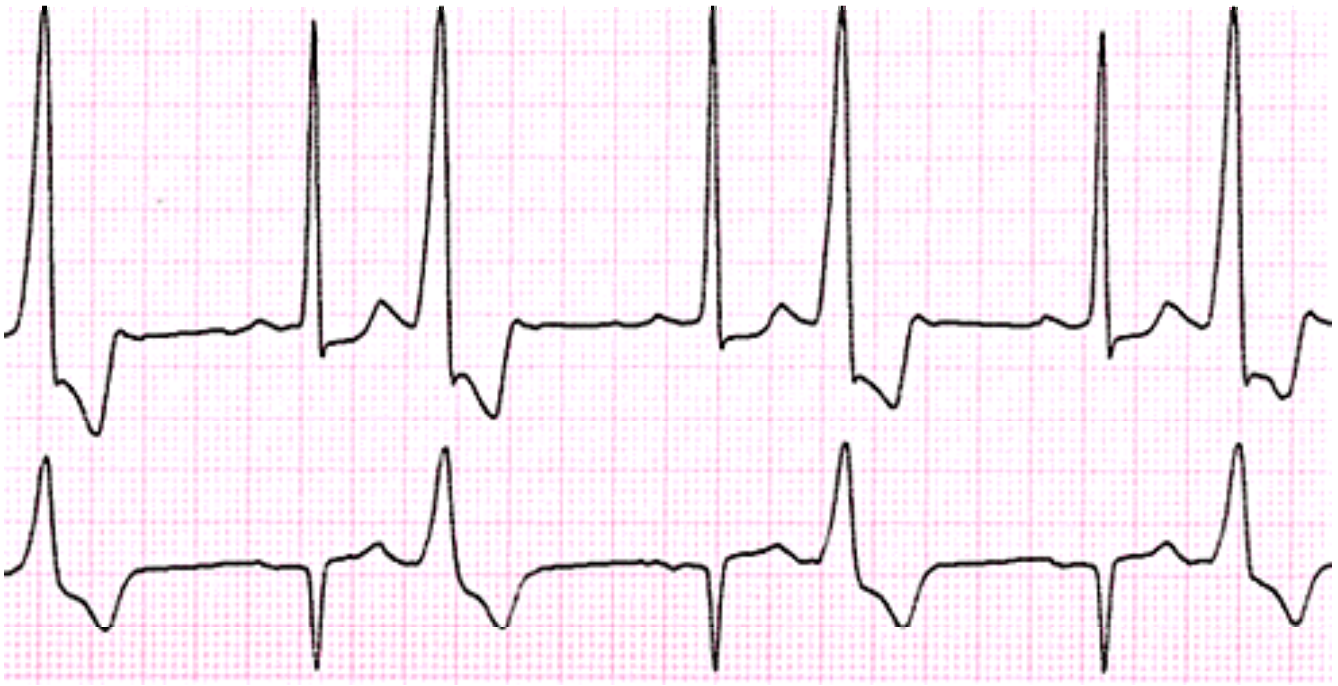
- PVC's don't have P-waves unless they are retrograde (may be buried in T-Wave)
- T-waves for PVC's are usually large and opposite in polarity to terminal QRS
- Wide (> .16 sec) notched PVC's may indicate a dilated hypokinetic left ventricle
- Every other beat being a PVC (bigeminy) may indicate coronary artery disease
- Some PVC's come between 2 normal sinus beats and are called "interpolated" PVC's



Unifocal PVC's



Example of Ventricular Bigeminy



Recognizing and Naming Beats & Rhythms

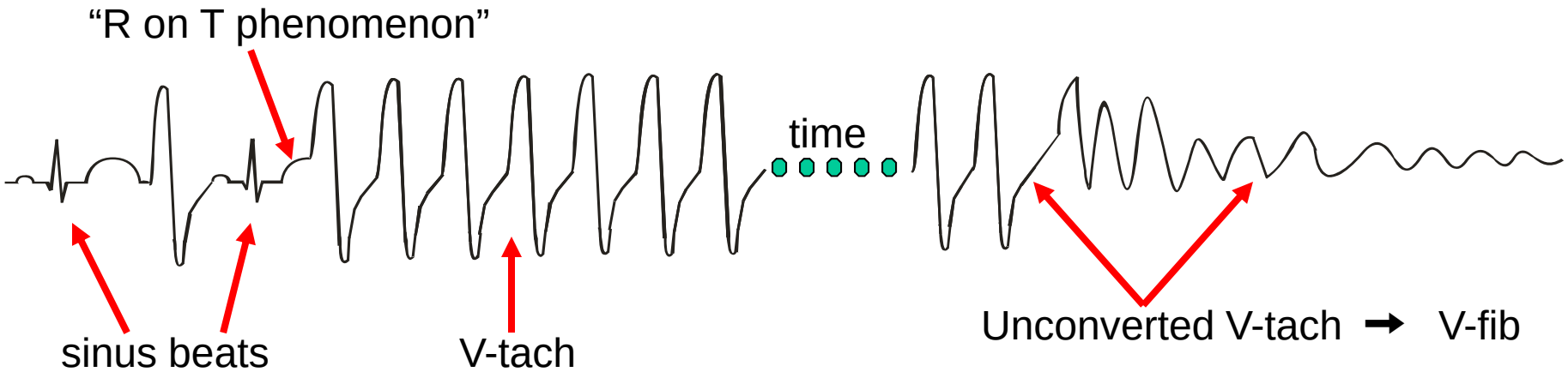
PVC's are Dangerous When:

- They are frequent (> 30% of complexes) or are increasing in frequency
- The come close to or on top of a preceding T-wave (R on T)
- Three or more PVC's in a row (run of V-tach)
- Any PVC in the setting of an acute MI
- PVC's come from different foci ("multifocal" or "multiformed")

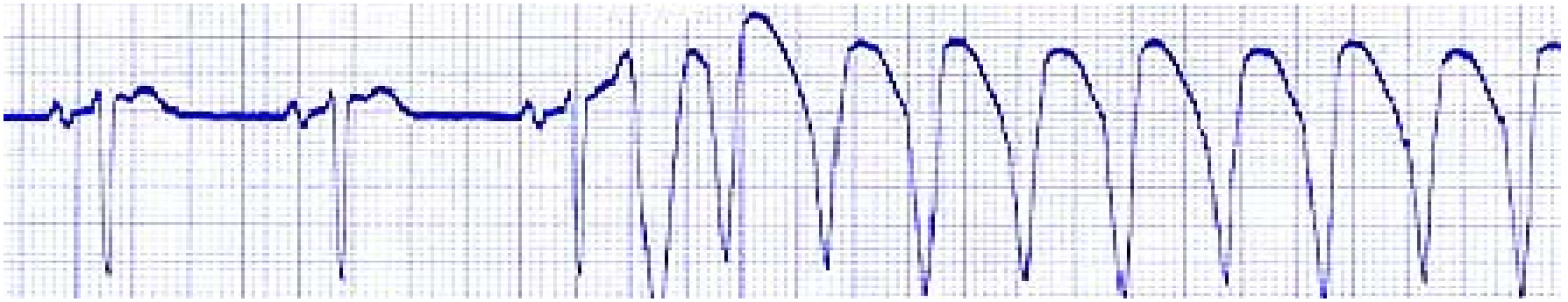
These dangerous phenomenon may preclude occurrence of deadly arrhythmias:

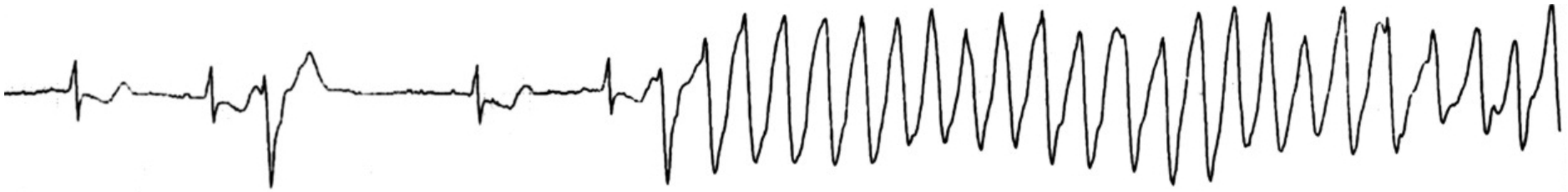
- **Ventricular Tachycardia**
- **Ventricular Fibrillation**

→ **The sooner defibrillation takes place, the increased likelihood of survival**

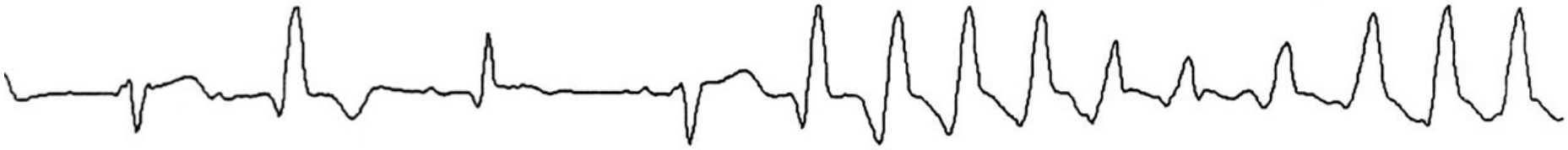


R on T event initiates Ventricular Tachycardia

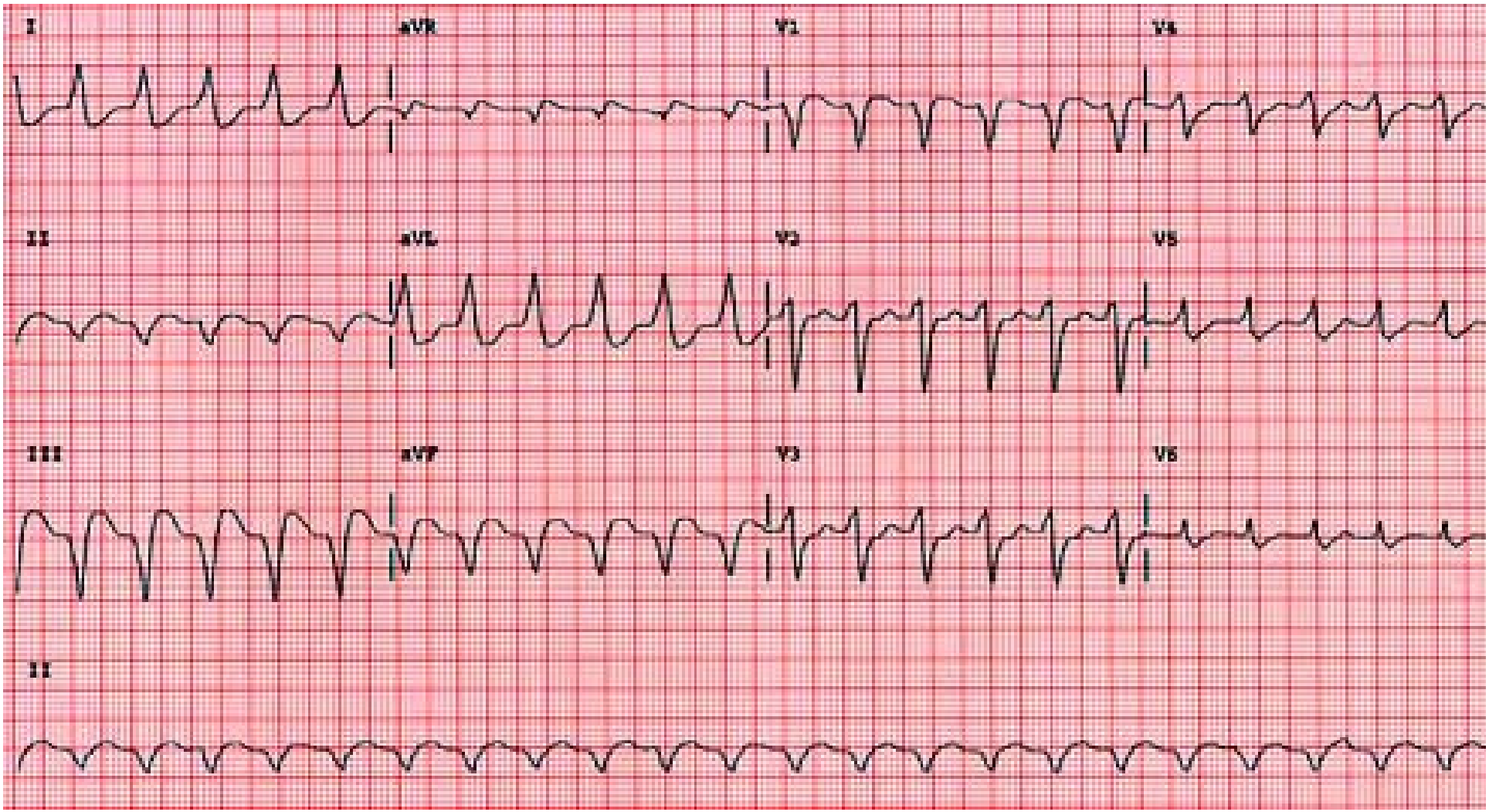




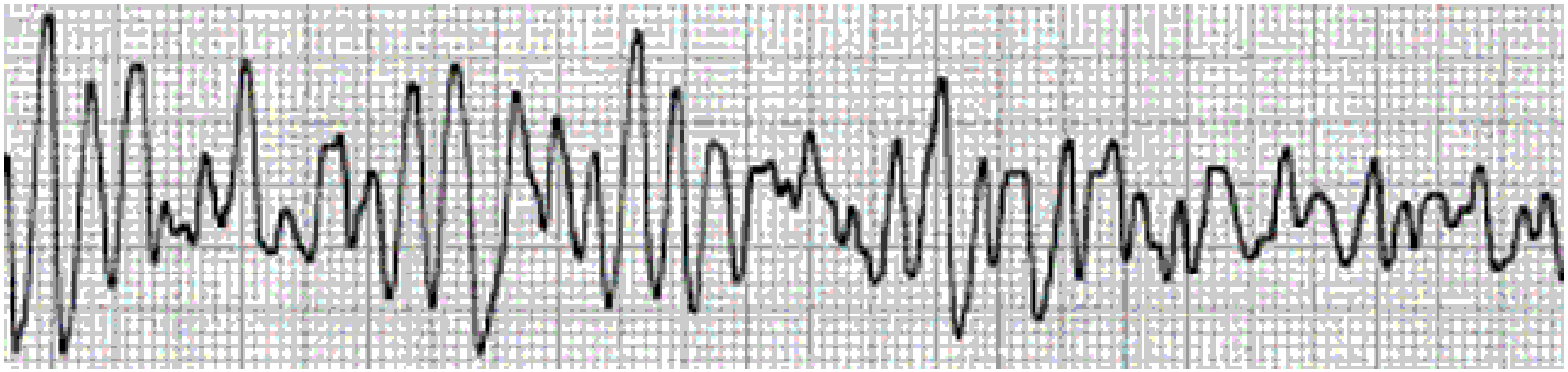
Good Example of Polymorphic Ventricular Tachycardia



Example of Ventricular Tachycardia



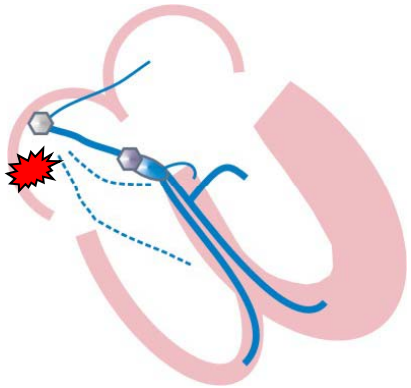
Example of Ventricular Fibrillation

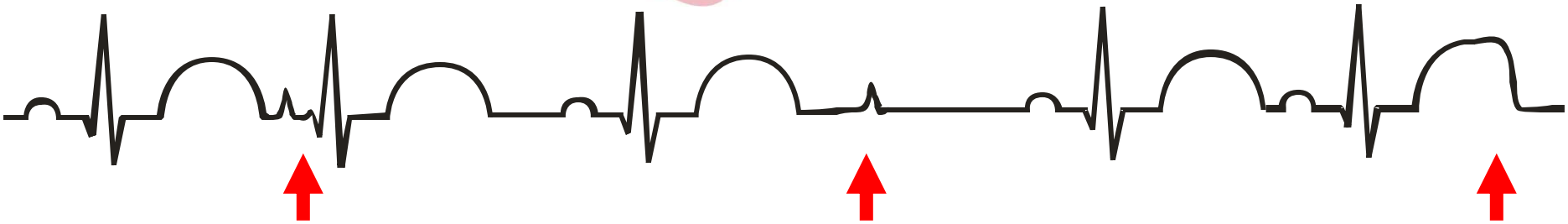


Recognizing and Naming Beats & Rhythms

Premature Atrial Contractions (PAC's):

- An ectopic focus in the atria discharges causing an early beat
- The P-wave of the PAC will not look like a normal sinus P-wave (different morphology)
- QRS is narrow and normal looking because ventricular depolarization is normal
- PAC's may not activate the myocardium if it is still refractory (non-conducted PAC's)
- PAC's are most often benign: caused by stress, alcohol, caffeine, and tobacco



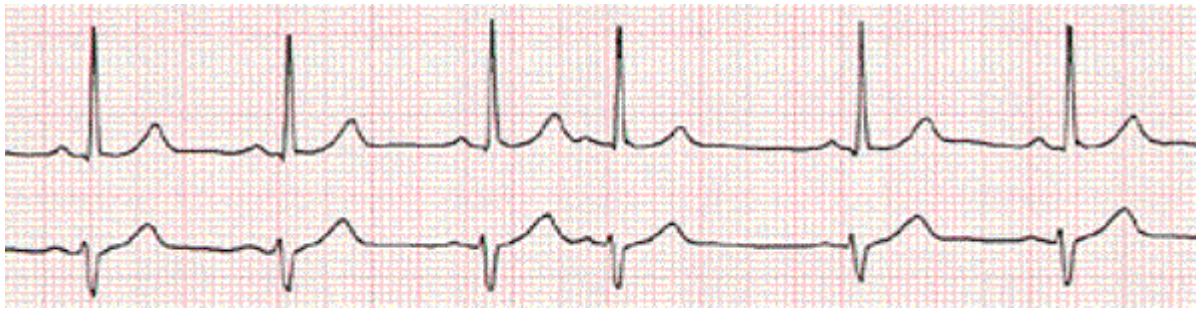


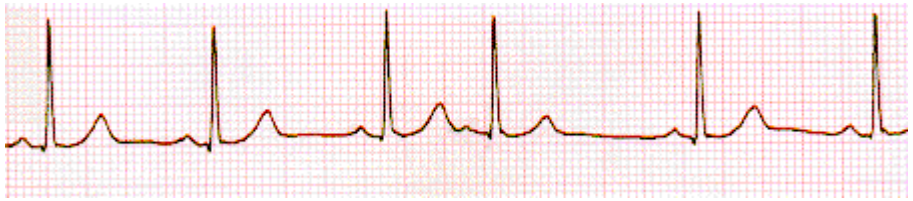
PAC

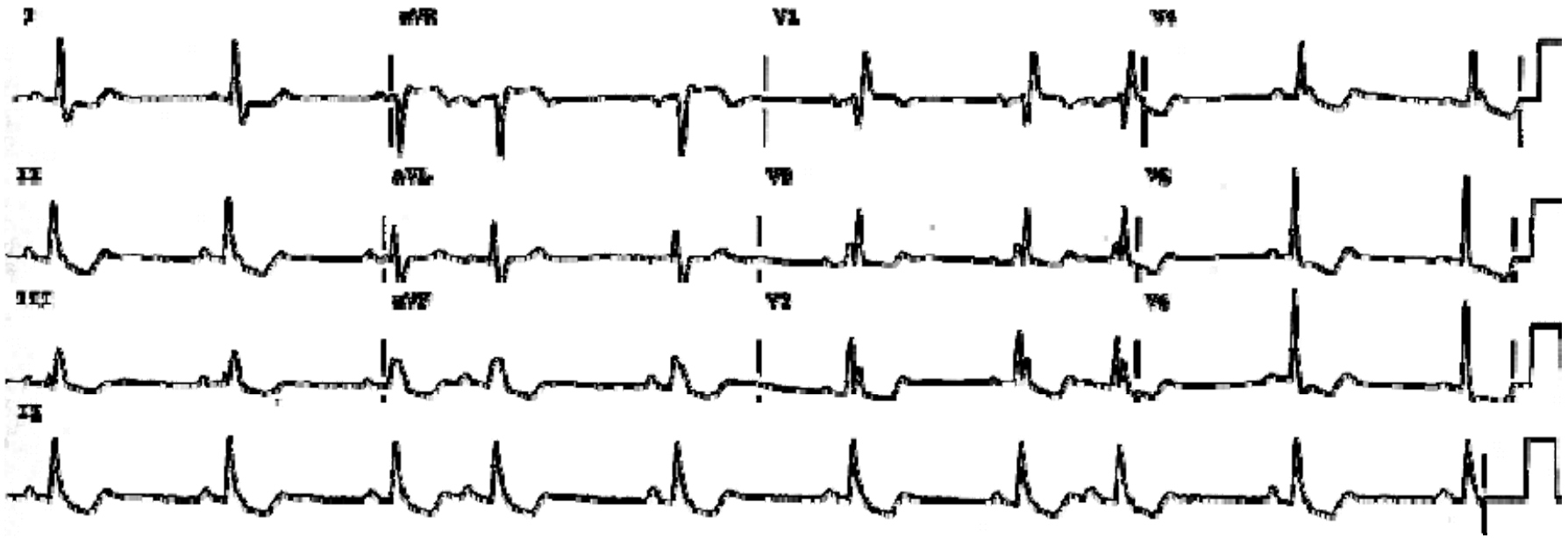
Non conducted PAC

Non conducted PAC distorting a T-wave

Examples of Premature Atrial Contractions



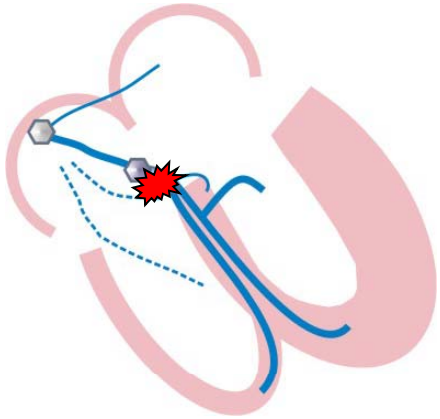


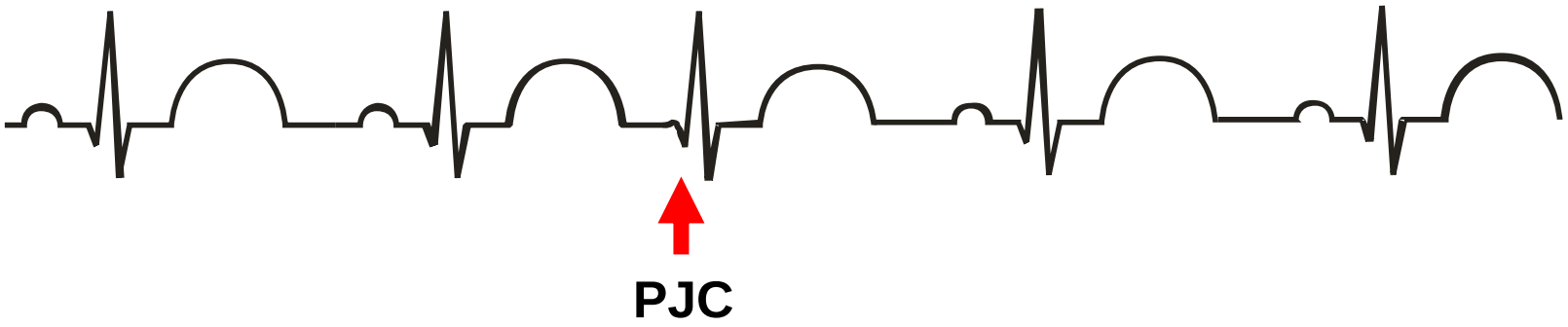


Recognizing and Naming Beats & Rhythms

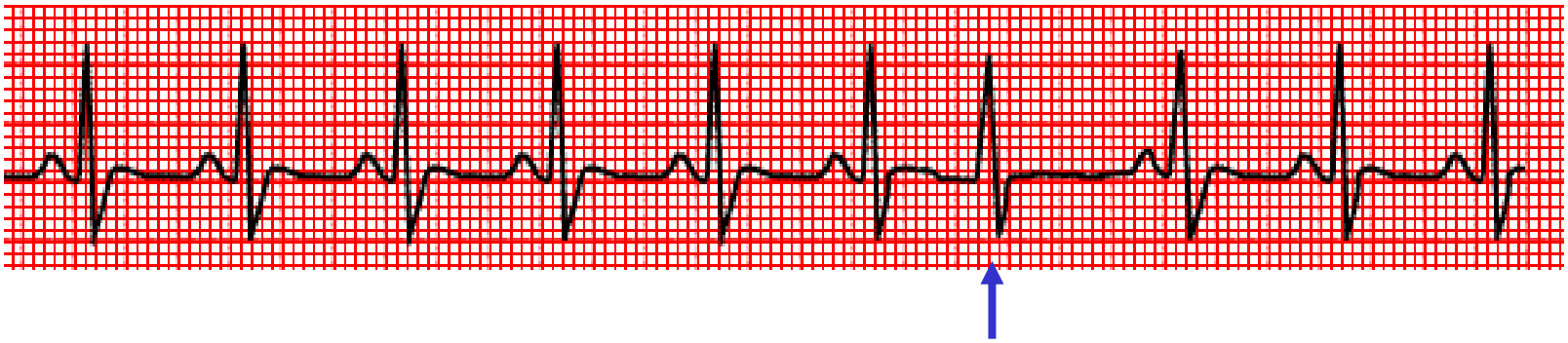
Premature Junctional Contractions (PJC's):

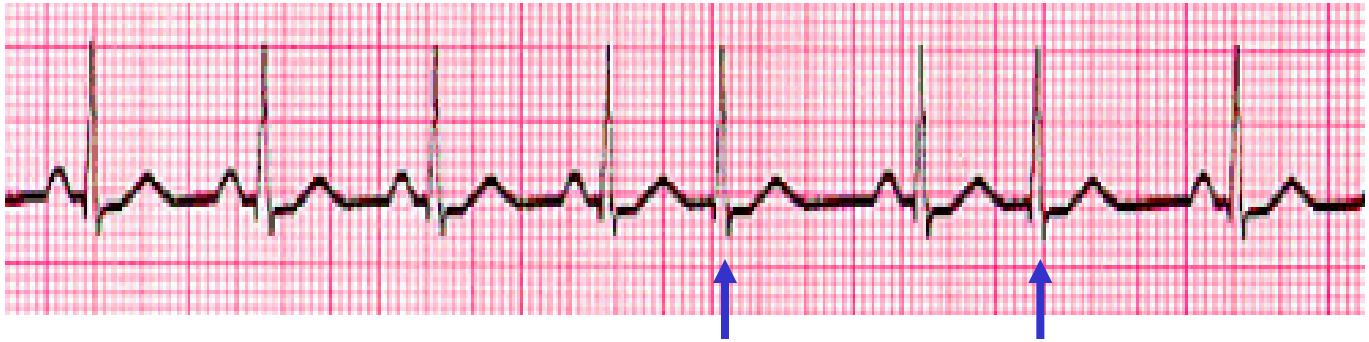
- An ectopic focus in or around the AV junction discharges causing an early beat
- The beat has no P-wave
- QRS is narrow and normal looking because ventricular depolarization is normal
- PJC's are most often benign and require no treatment

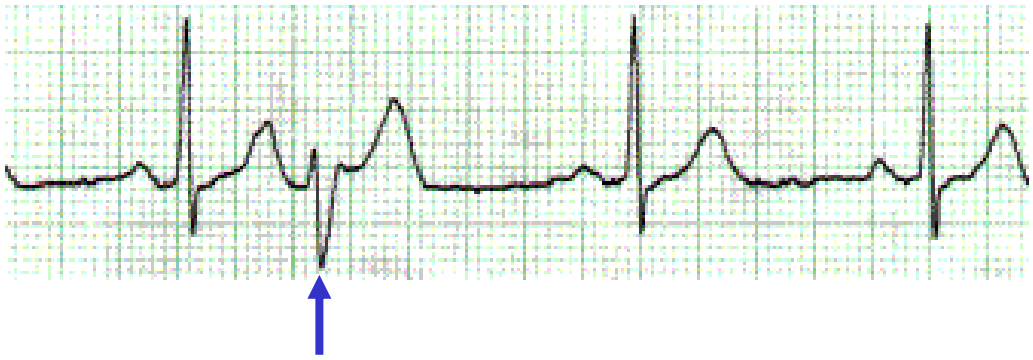




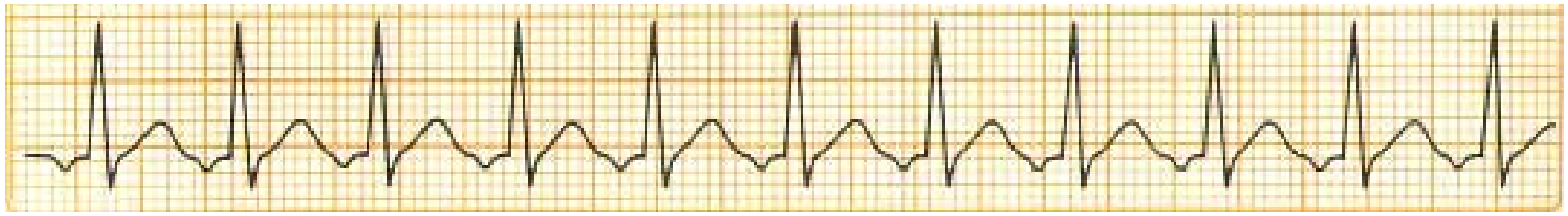
Examples of Premature Junctional Contractions

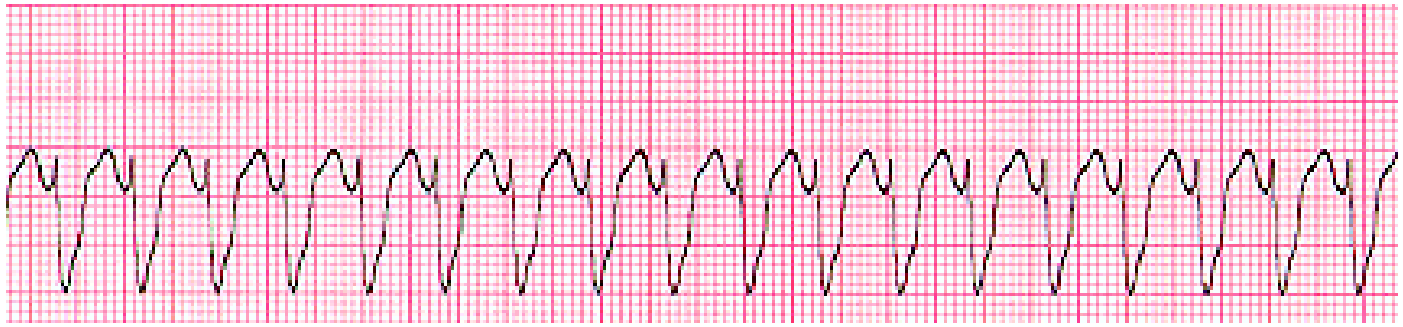


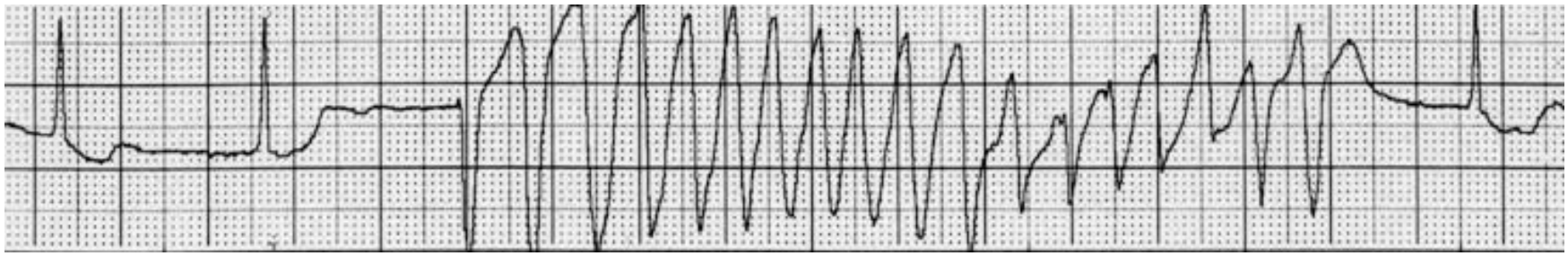




What do you see ?

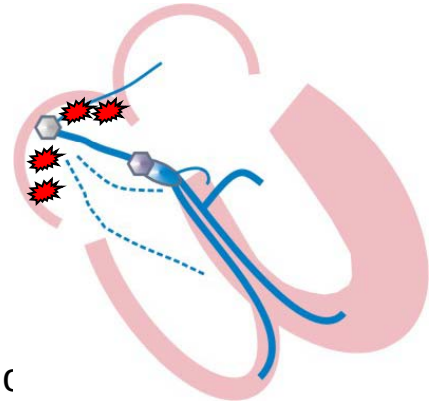




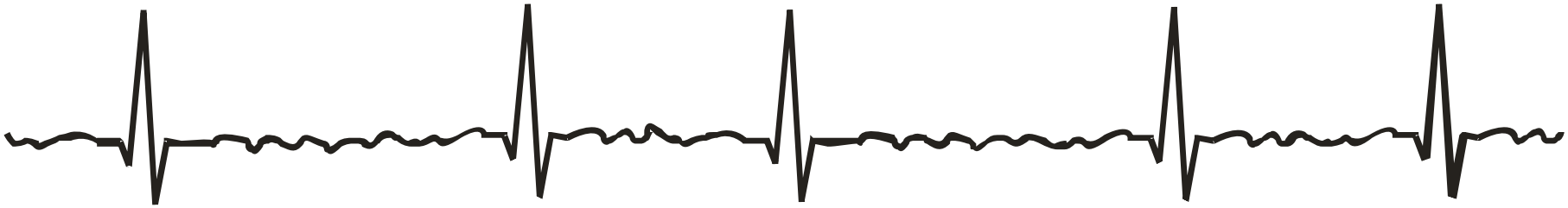


Recognizing and Naming Beats & Rhythms

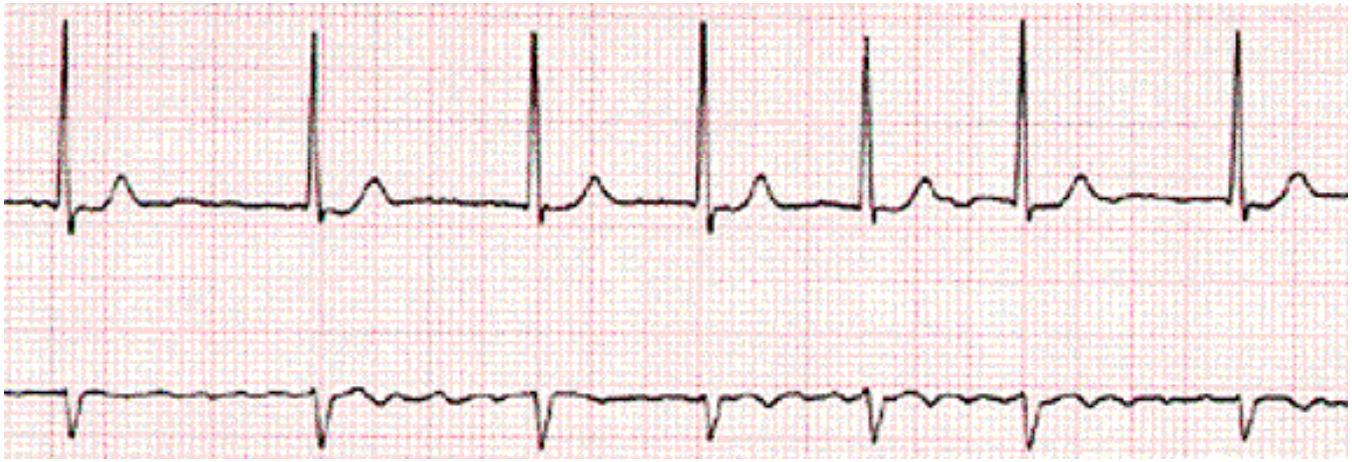
Atrial Fibrillation (A-Fib):

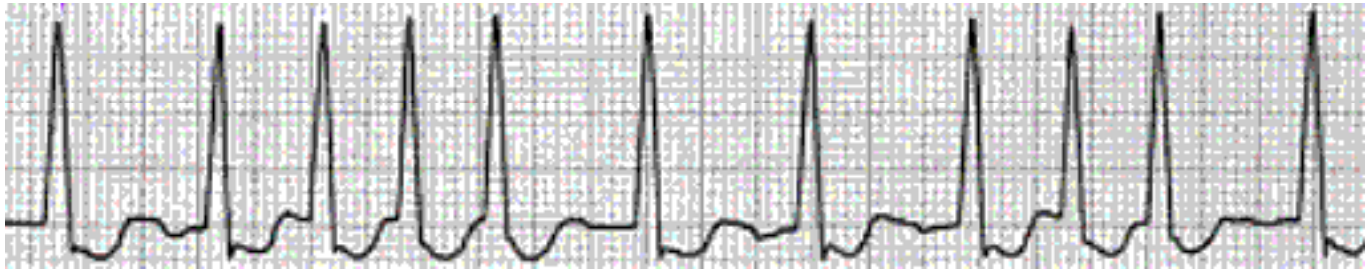


- Multiple ectopic reentrant focuses fire in the atria causing a chaotic baseline
- The rhythm is irregular and rapid (approx. 140 – 150 beats per minute)
- $\dot{Q}$ is usually ↓ by 10% to 20% (no atrial "kick" to ventricular filling)
- May be seen in CAD (especially following surgery), mitral valve stenosis, LV hypertrophy, CHF
- Treatment: DC cardioversion & O2 if patient is unstable
  - Drugs: (rate control) β & Ca^{++} channel blockers, digitalis, to ↓ AV Conductic
  - Amiodarone to ↓ AV conduction + prolong myocardial AP (↑ refractoriness of myocardium)
- The danger of thromboembolic events are enhanced due to ↓ flow in left atrial appendage
  - 5 fold ↑ in the risk of embolic stroke (5% of AF patients have a stroke)
  - 15% of stokes in US caused by AF
  - Treatment: anticoagulant drugs (Warfarin / Coumadin) Antiarrhythmics (Amiodarone)
    - International Normalized Ratio (INR – normalized PT time) should be between 2 and 3.
  - **DABIGATRAN** – new oral thrombin inhibitor….no need for follow ups to titrate dose



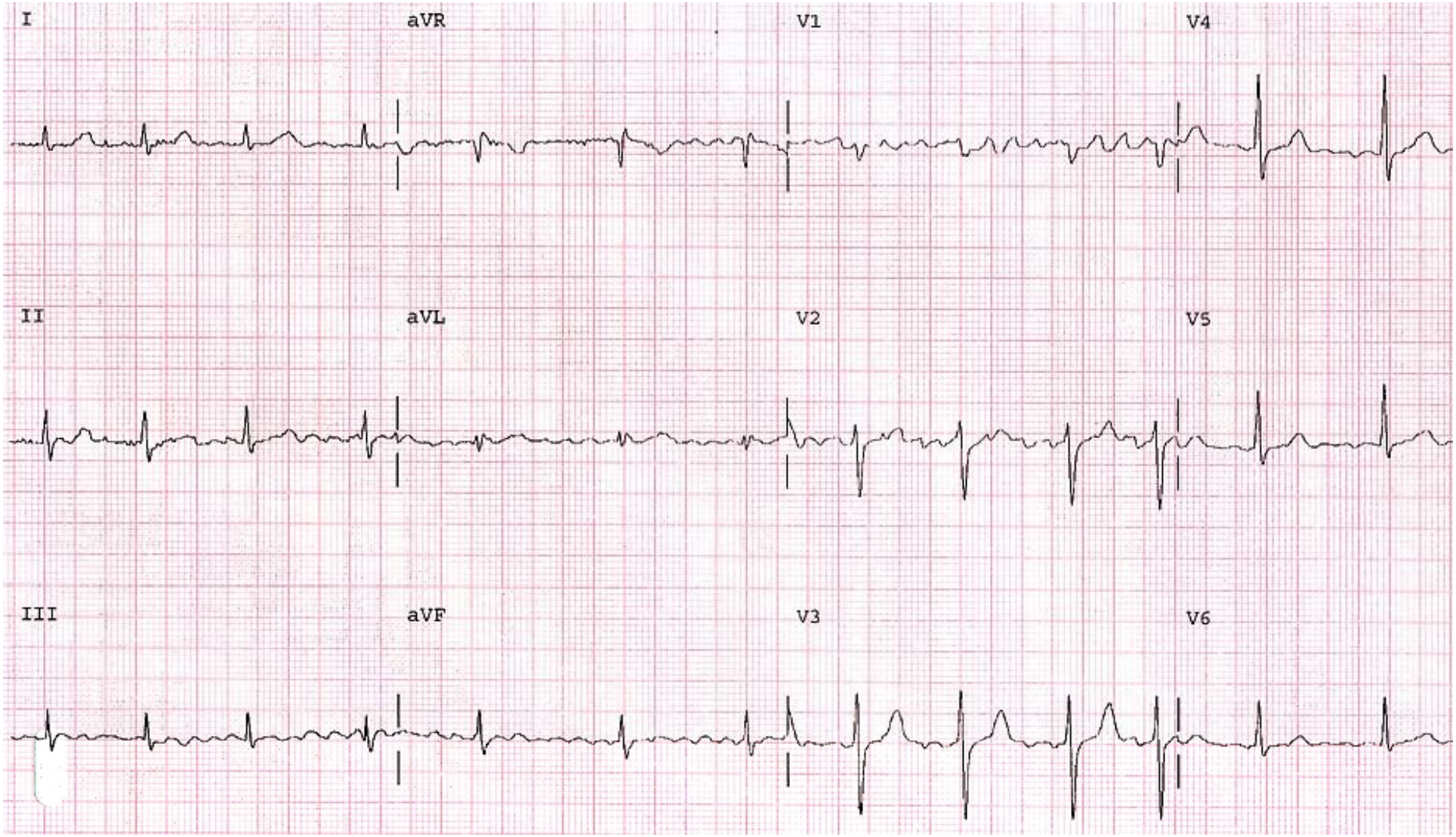
Examples of Atrial Fibrillation



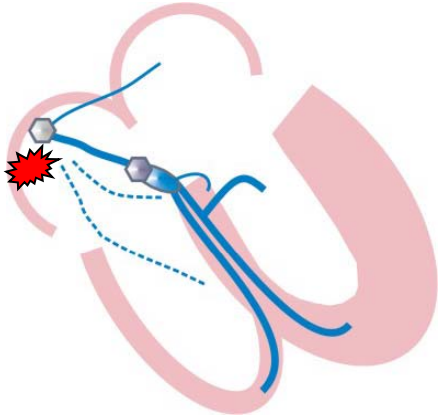


A-fib with fast ventricular response

Examples of Atrial Fibrillation

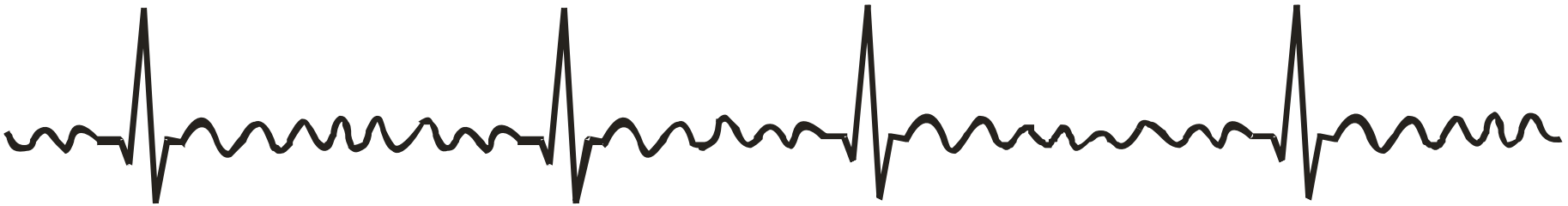


Recognizing and Naming Beats & Rhythms

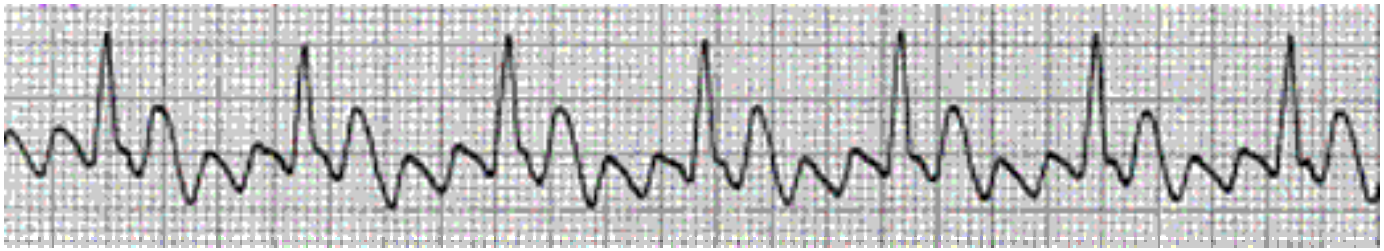


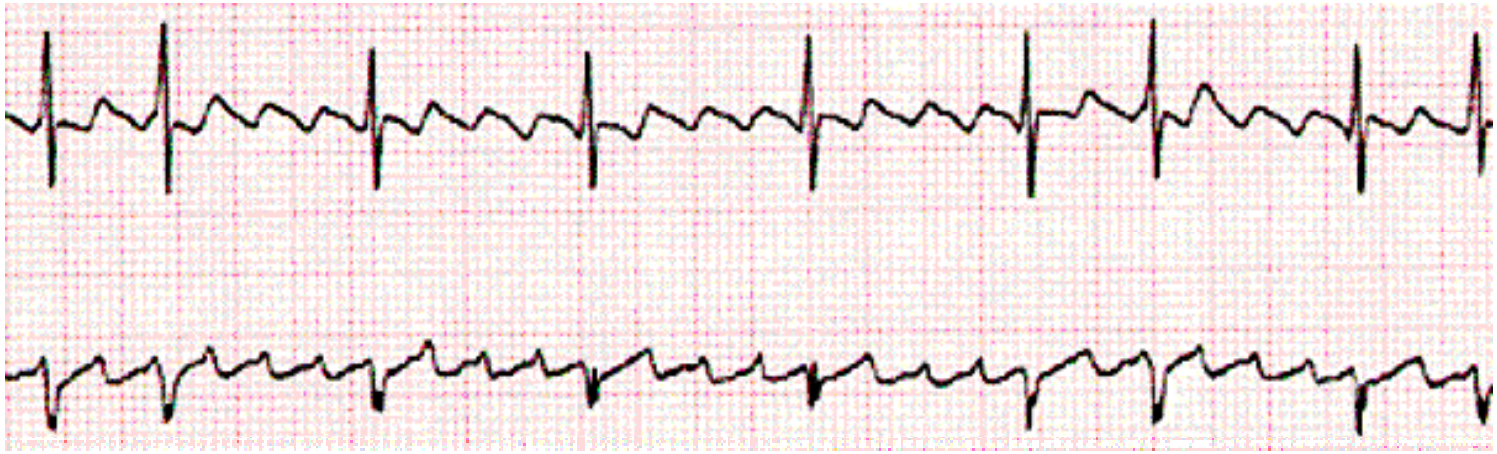
Atrial Flutter:

- A single ectopic macro-reentrant focus fire in the atria
- Single ectopic focus causes “saw-tooth” pattern
- AV node cannot transmit all impulses (atrial rate: 250 –350 per minute)
    - Ventricular rhythm may be regular or irregular and range from 150 –170 beats / minute
- $\dot{Q}$ may ⬇, especially at high ventricular rates
- A-fib and A-flutter rhythm may alternate – these rhythms may also alternate with SVT’s
- May be seen in CAD (especially following surgery), VHD, history of hypertension, LVH, CHF
- Treatment: DC cardioversion if patient is unstable
    - Drugs: (goal: rate control) Ca^{++} channel blockers to ⬇ AV conduction
    - Amiodarone to ⬇ AV conduction + prolong myocardial AP (⬆ refractoriness of myocardium)
- The danger of thromboembolic events is also high in A-flutter



Examples of Atrial Flutter

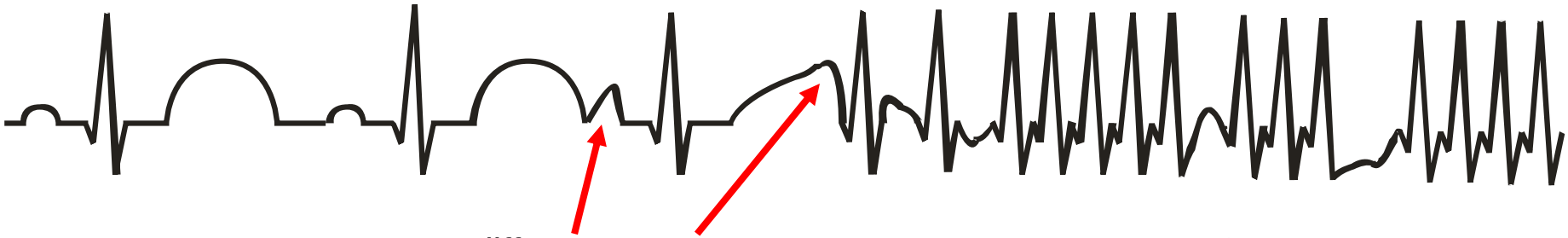




Recognizing and Naming Beats & Rhythms

Multifocal Atrial Tachycardia (MAT):

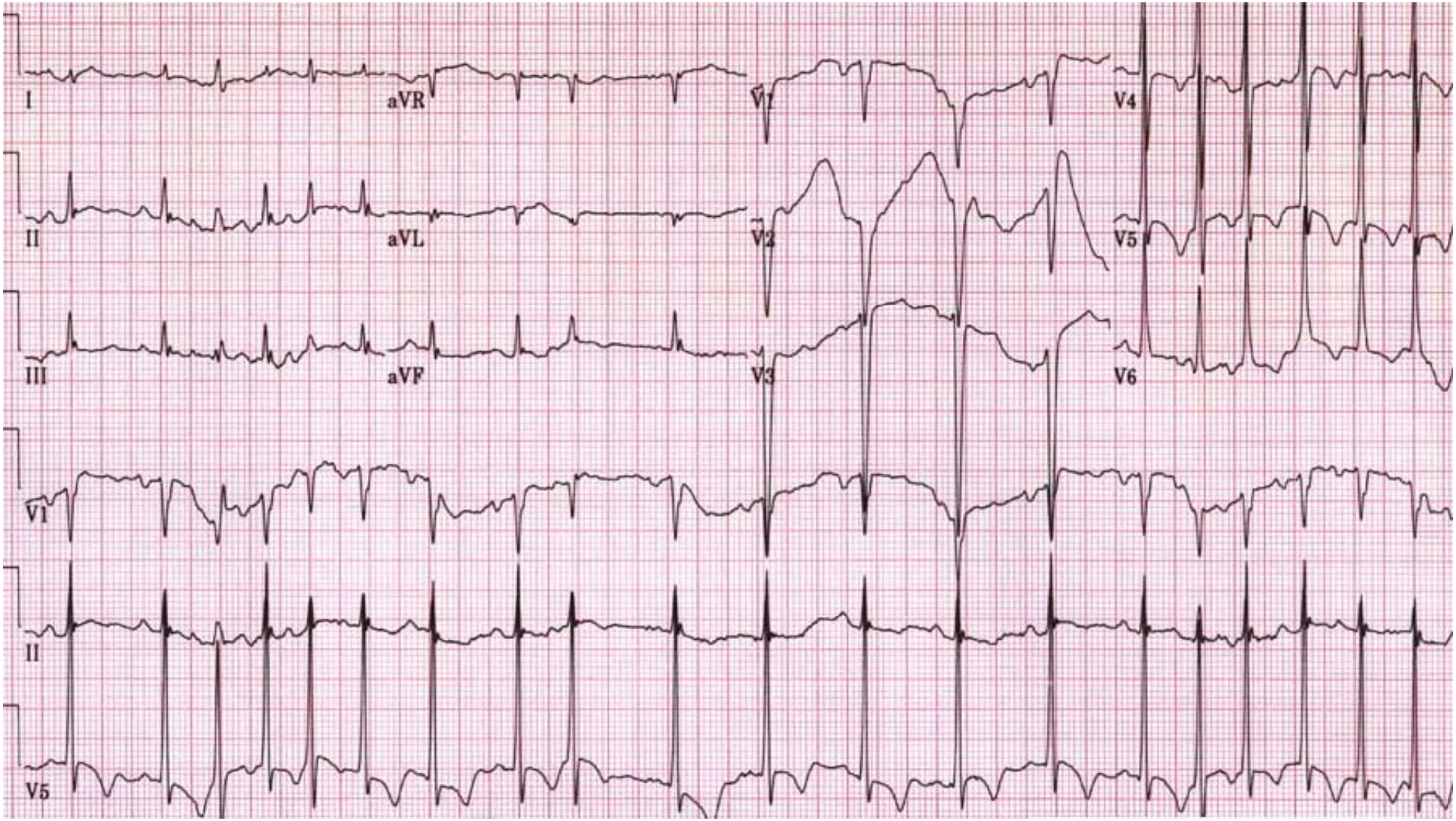
- Multiple ectopic focuses fire in the atria, all of which are conducted normally to the ventricles
    - QRS complexes are almost identical to the sinus beats
- Rate is usually between 100 and 200 beats per minute
- The rhythm is always **IRREGULAR**
- P-waves of different morphologies (shapes) may be seen if the rhythm is slow
    - If the rate < 100 bpm, the rhythm may be referred to as “wandering pacemaker”
- Commonly seen in pulmonary disease, acute cardiorespiratory problems, and CHF
- Treatments: Ca^{++} channel blockers, β blockers, potassium, magnesium, supportive therapy for underlying causes mentioned above (antiarrhythmic drugs are often ineffective)



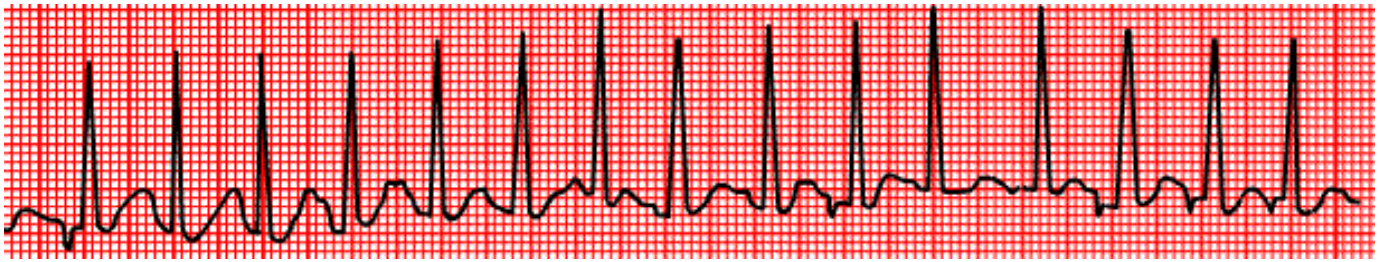
Note different P-wave morphologies when the tachycardia begins

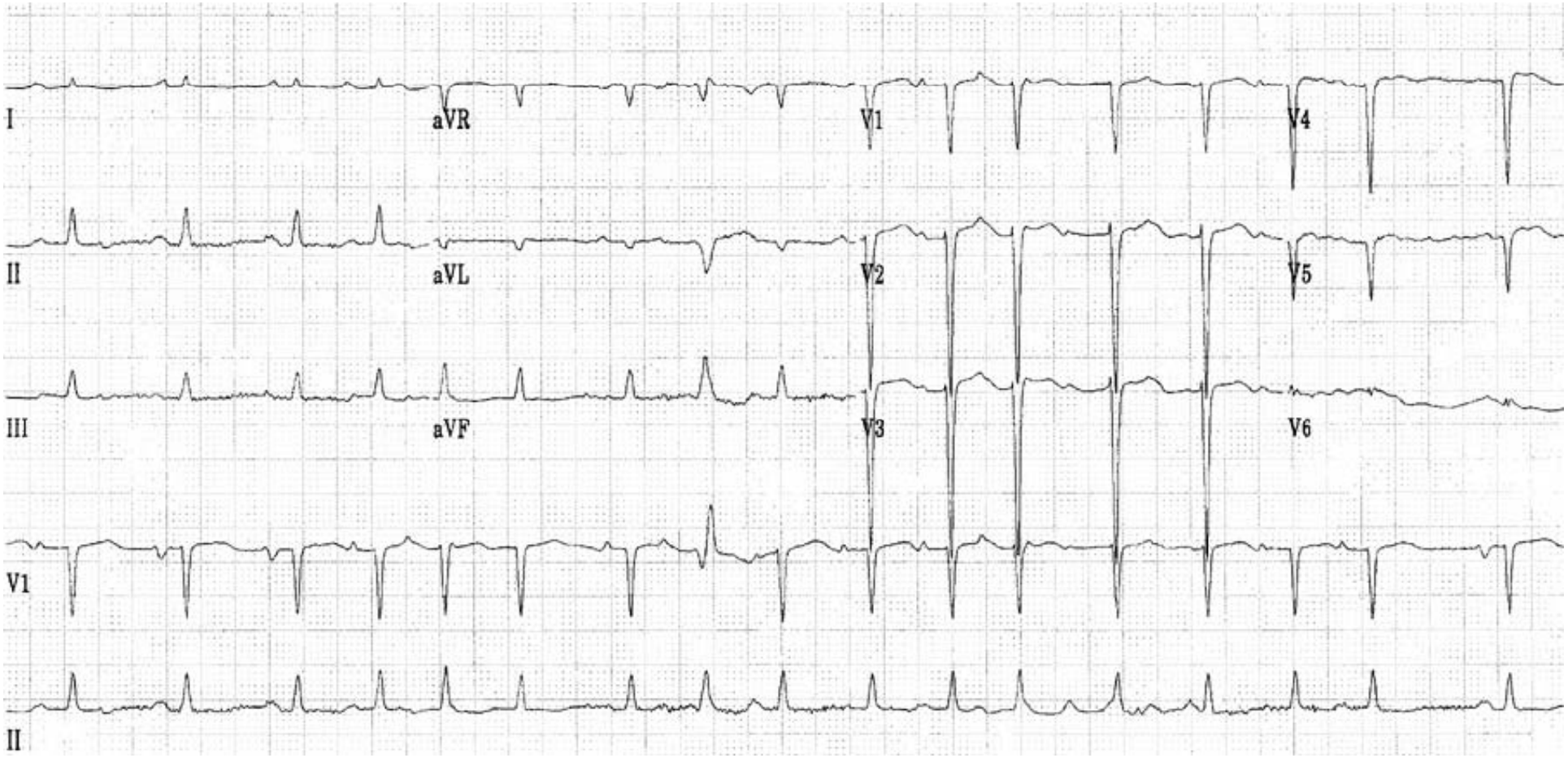
Note **IRREGULAR** rhythm in the tachycardia

Examples of Multifocal Atrial Tachycardia



Examples of Multifocal Atrial Tachycardia

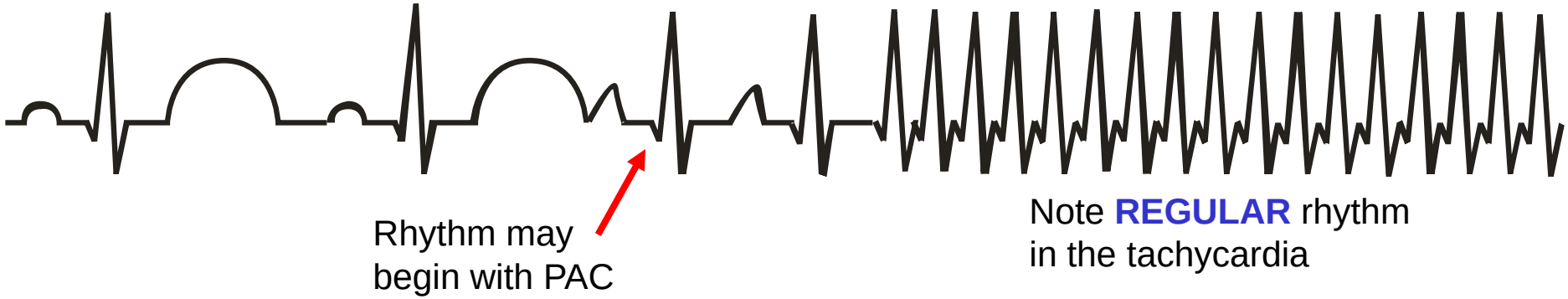




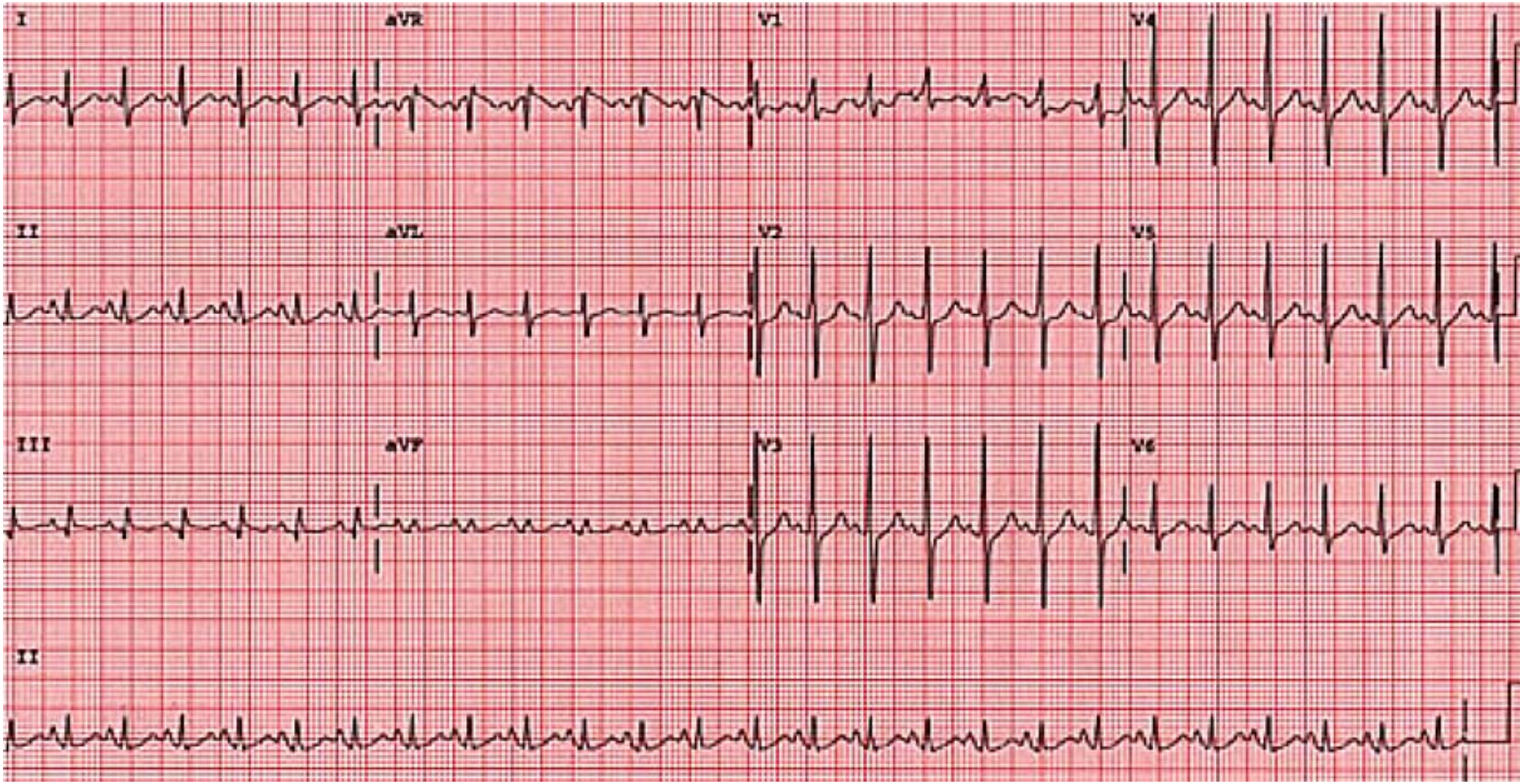
Recognizing and Naming Beats & Rhythms

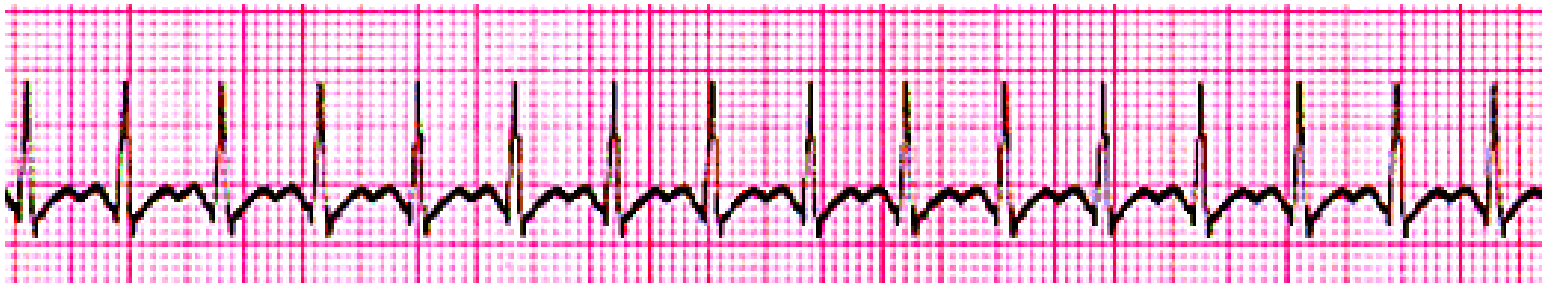
Paroxysmal (of sudden onset) Supraventricular Tachycardia (PSVT):

- A single reentrant ectopic focuses fires in and around the AV node, all of which are conducted normally to the ventricles (usually initiated by a PAC)
  - QRS complexes are almost identical to the sinus beats
- Rate is usually between 150 and 250 beats per minute
- The rhythm is always **REGULAR**
- Possible symptoms: palpitations, angina, anxiety, polyuria, syncope (↓ Q̇)
- Prolonged runs of PSVT may result in atrial fibrillation or atrial flutter
- May be terminated by carotid massage
  - ↑ carotid pressure → ↑ baroreceptor firing rate → ↑ vagal tone → ↓ AV conduction
- Treatment: ablation of focus, Adenosine (↓ AV conduction), Ca^{++} Channel blockers



Examples of Paroxysmal Supraventricular Tachycardia

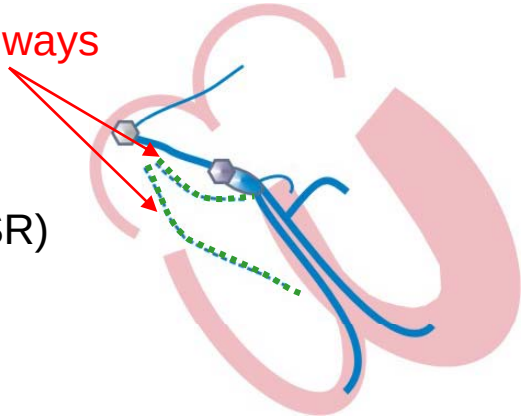


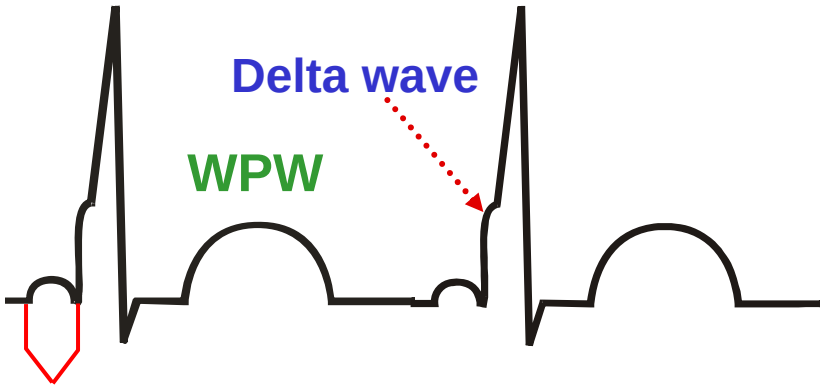


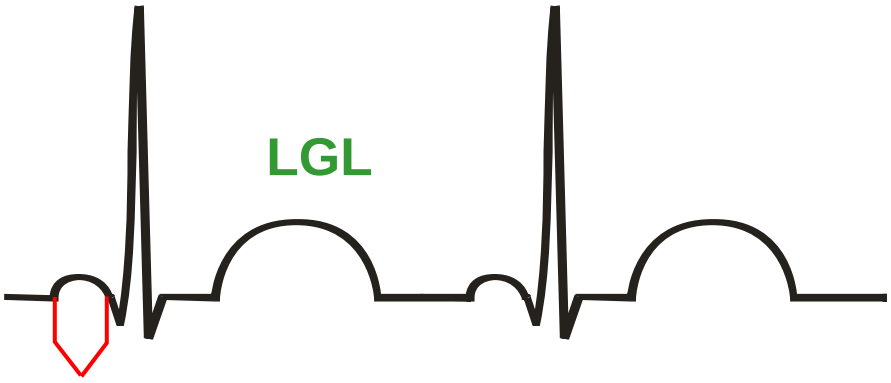
Recognizing and Naming Conduction Problems \ Rhythms

Wolf-Parkinson White Syndrome (WPW) and Lown-Ganong-Levine (LGL):

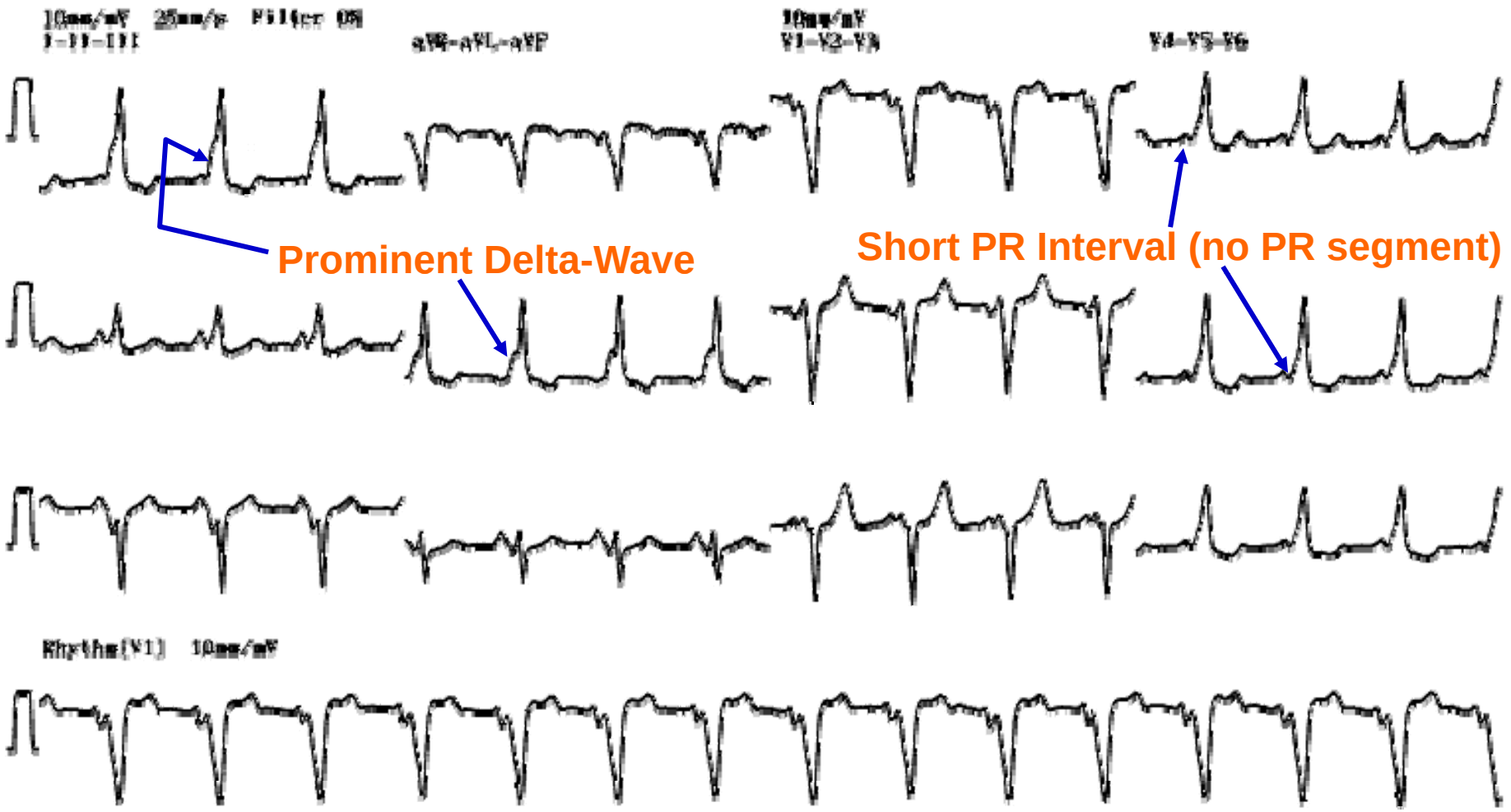
- Atrial impulses bypass the AV node through an accessory pathway or bypass tract (bundle of Kent) and reach the ventricles
  - No AV Nodal delay
  - PRI < .12 seconds, QRS complexes have a “delta” wave
- Regular Rhythm (WPW complexes may be intermittent with NSR)
- Treatment: radiofrequency ablation of bypass tract
- 50% - 70% of cases are associated with PSVT and A-fib
  - V-fib is sometimes seen in WPW induced A-fib (4% of the time) and can be deadly
- Fast ventricular response in WPW induced A-fib may look like V-tach
- LGL recognized by short PRI but no delta wave – also associated with PSVT but mostly benign



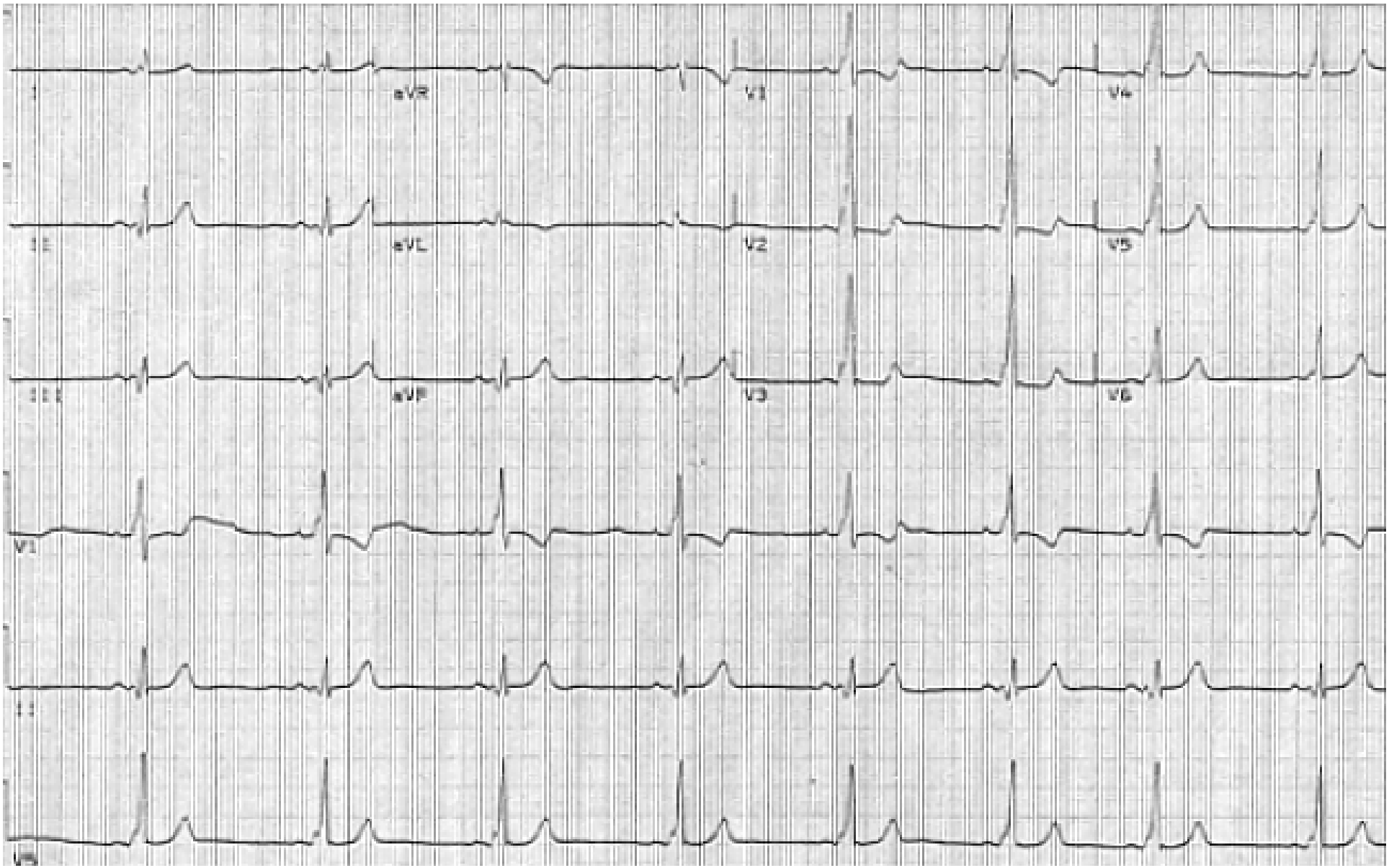




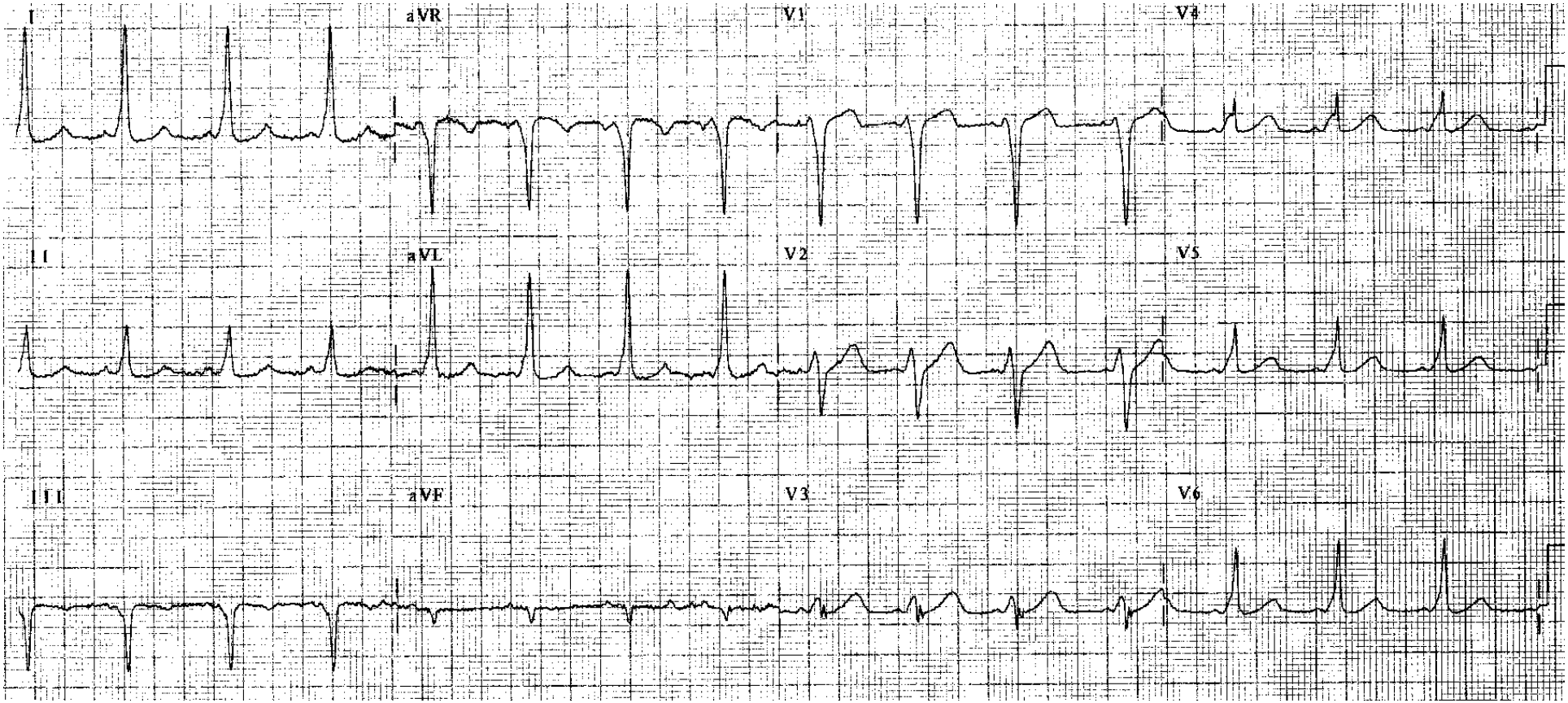
Examples of Wolf Parkinson White



Examples of Wolf Parkinson White



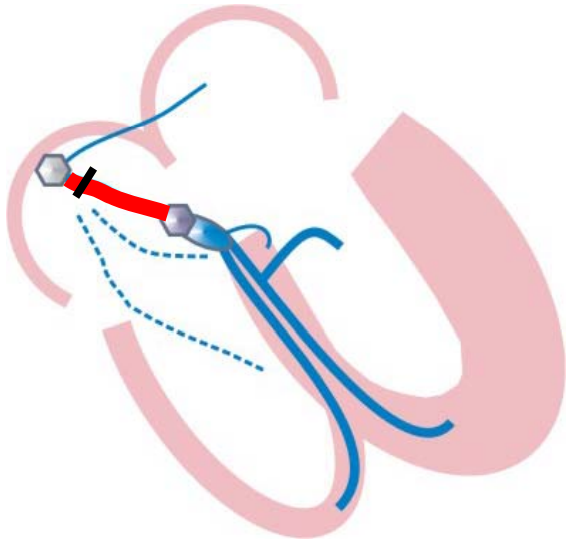
Examples of Wolf Parkinson White

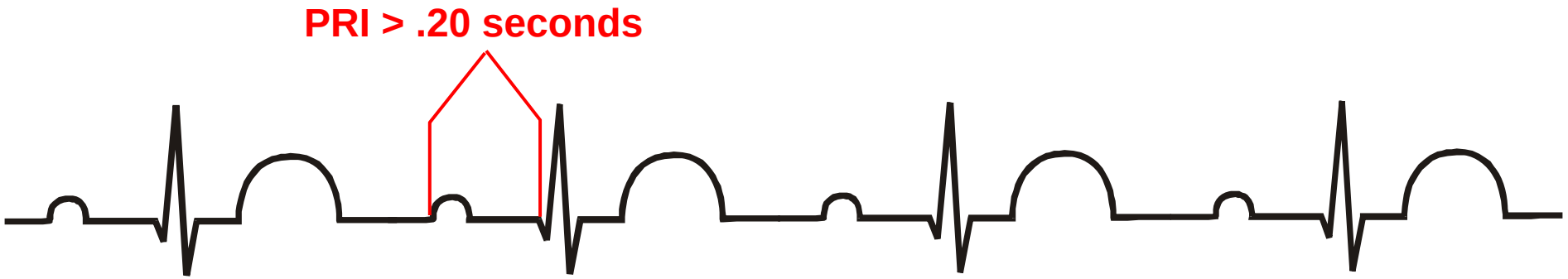


Recognizing and Naming Conduction Problems \ Rhythms

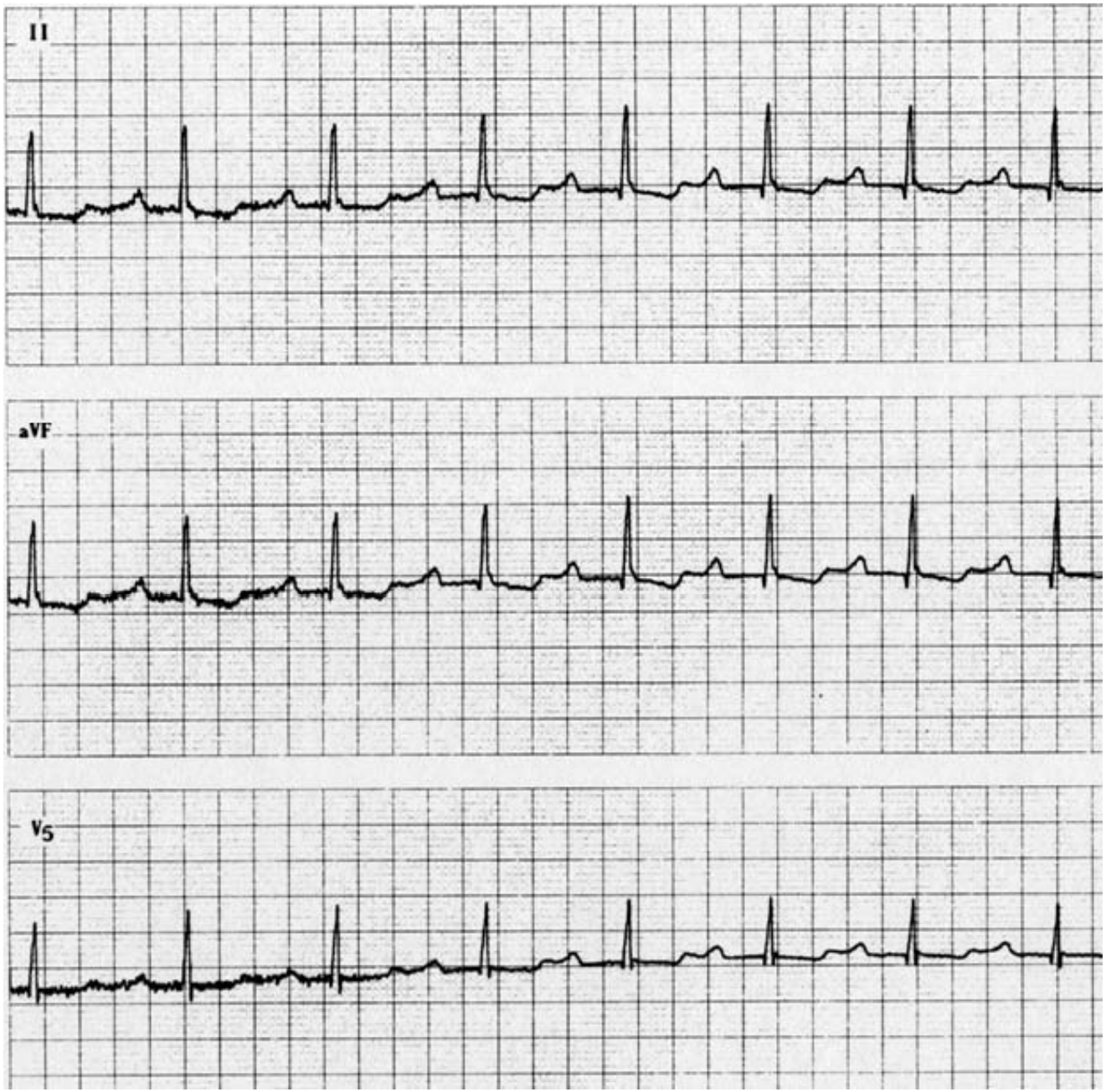
1st degree AV Block:

- Regular Rhythm
- PRI > .20 seconds and is CONSTANT
- Causes: MI, seen in healthy children, seen in healthy athletes
- Usually does not require treatment

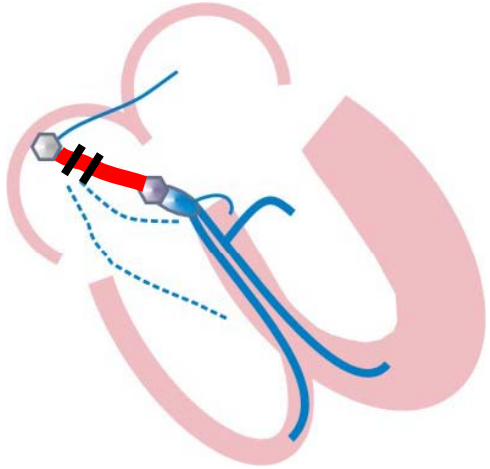




Examples of 1st degree AV Block

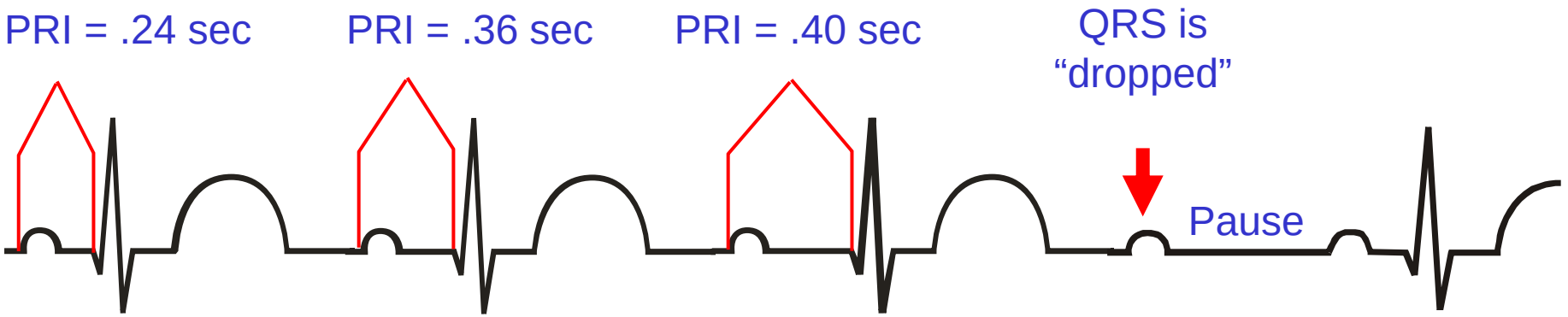


Recognizing and Naming Conduction Problems \ Rhythms



2nd degree AV Block ("Mobitz I" or "Wenckebach"):

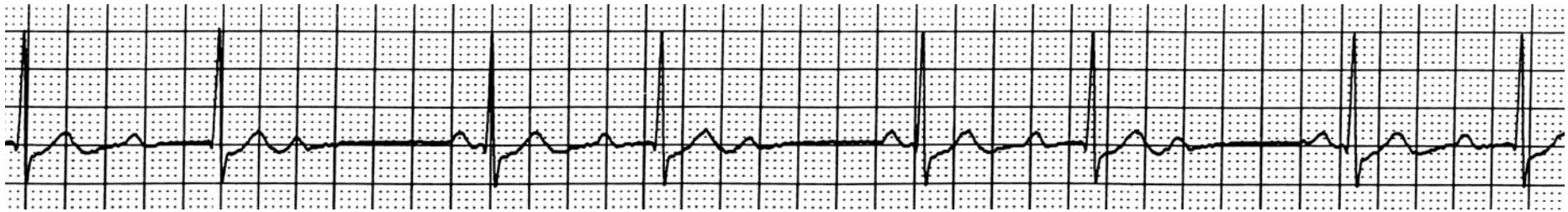
- Irregular Rhythm
- PRI continues to lengthen until a QRS is missing (non-conducted sinus impulse)
    - PRI is NOT CONSTANT
- Rhythm is usually benign unless associated with underlying pathology, i.e. MI)

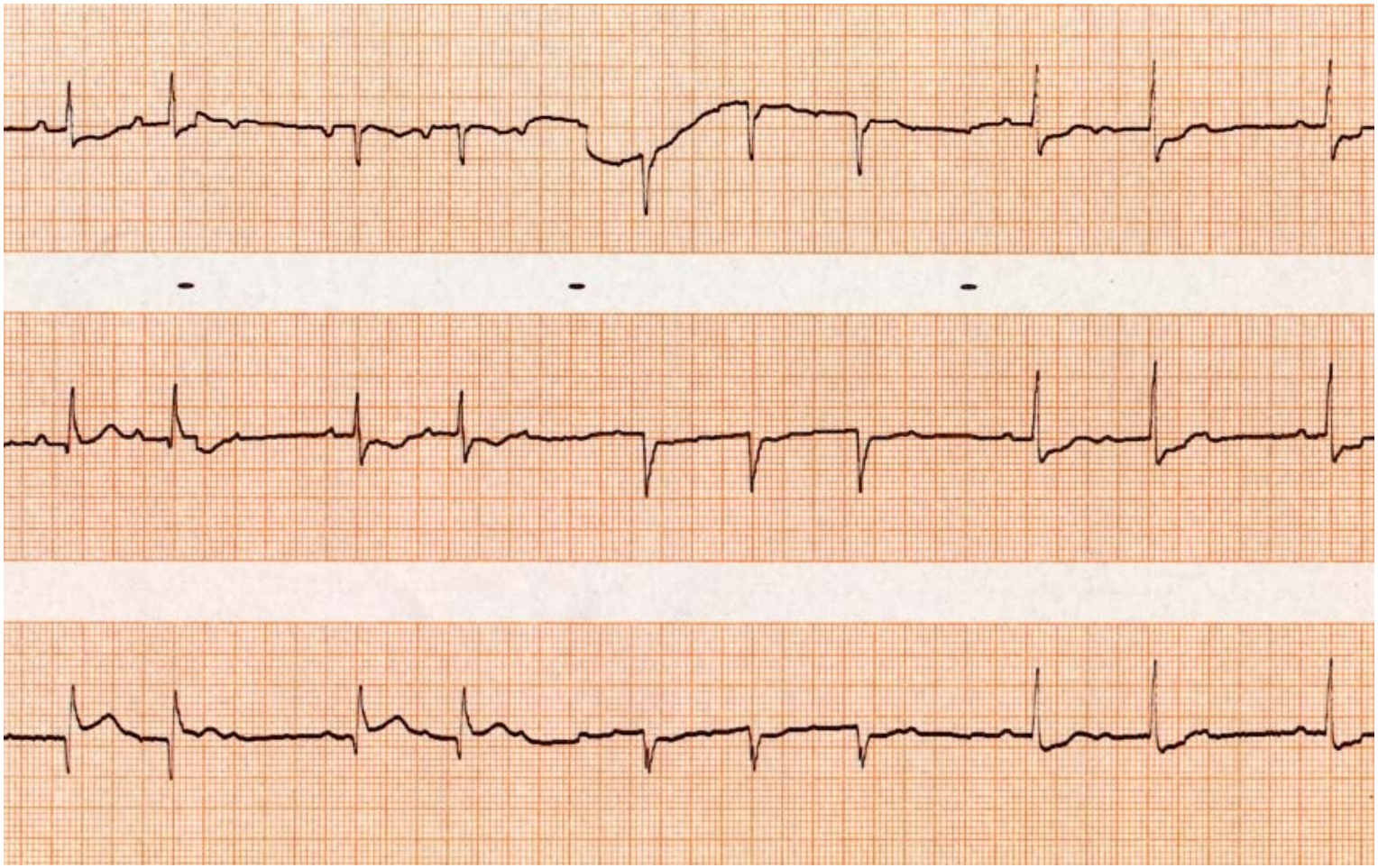


4:3 Wenckebach (conduction ratio may not be constant)

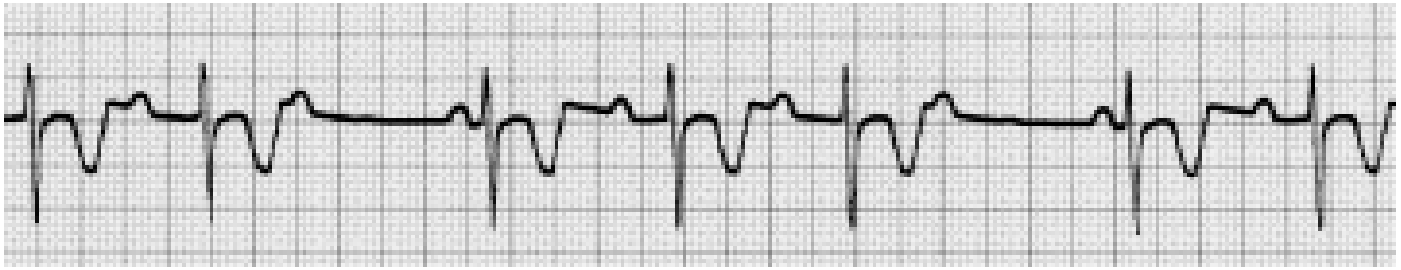
Pattern Repeats……

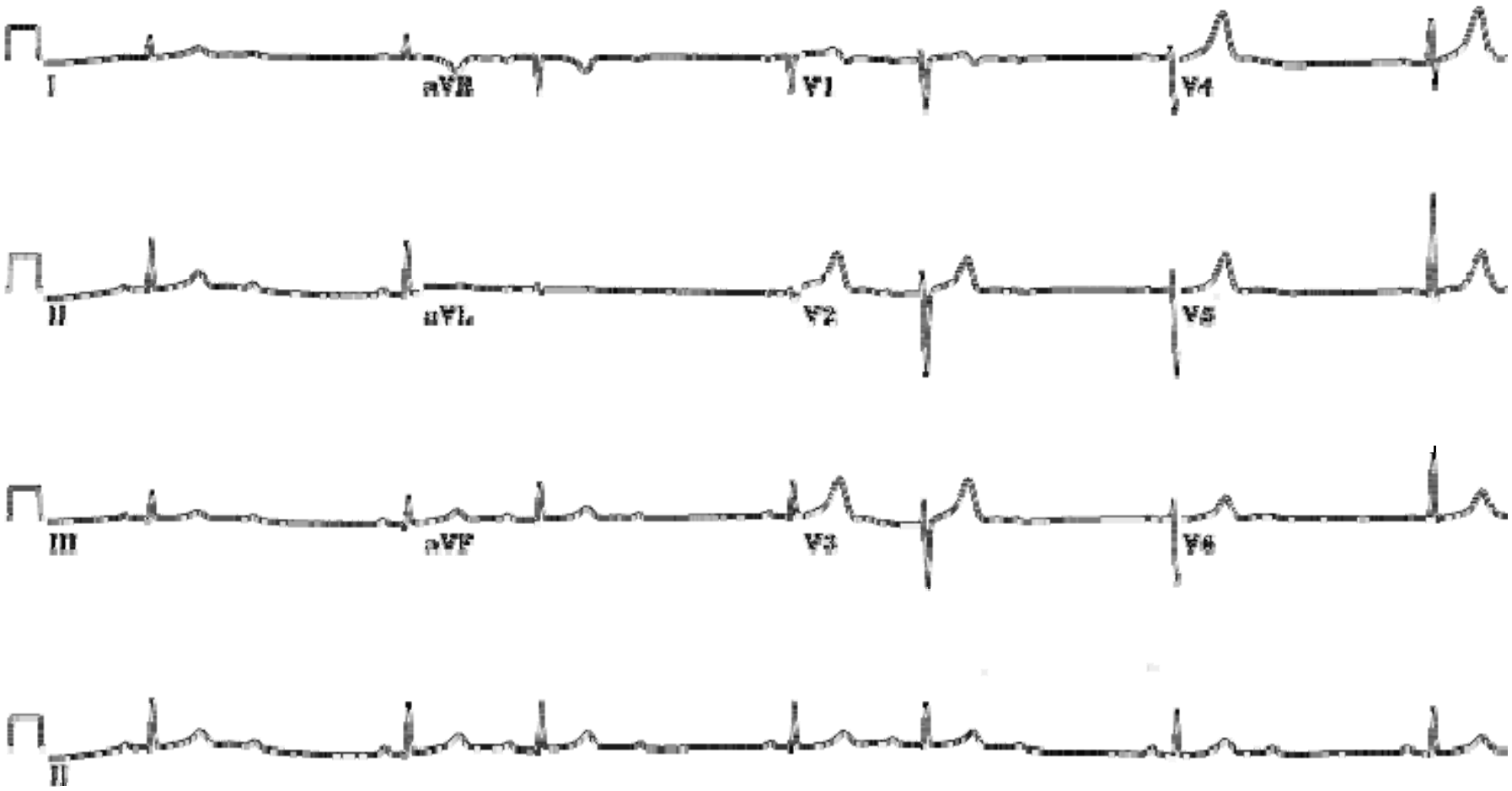
Examples of 2nd degree AV Block – Mobitz I - Wenchebach





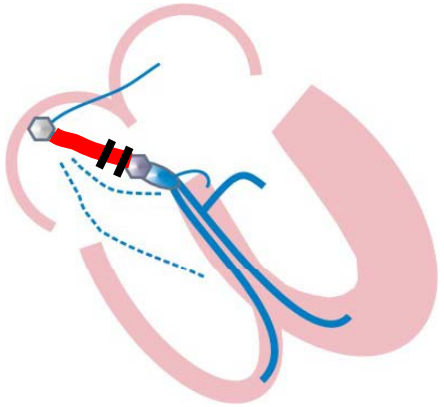
Examples of 2nd degree AV Block – Mobitz I - Wenchebach



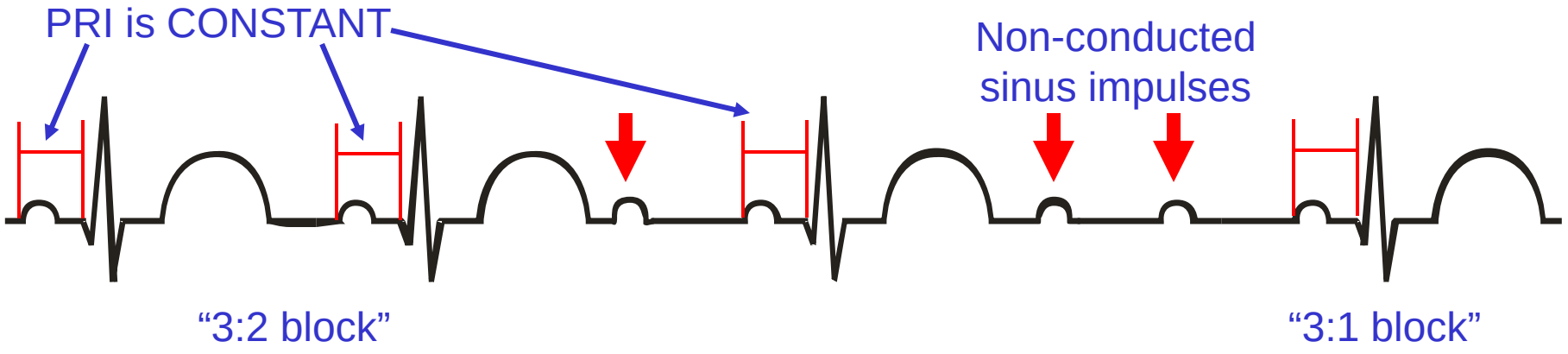


Recognizing and Naming Conduction Problems \ Rhythms

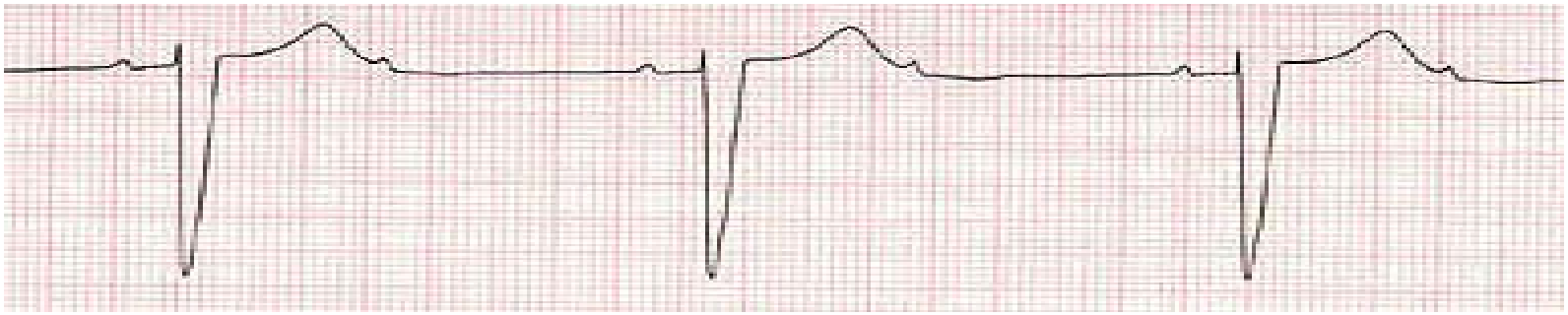
2nd degree AV Block ("Mobitz II"):

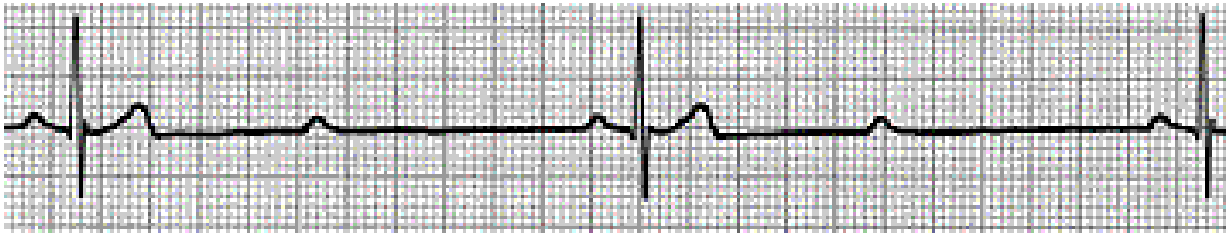


- Irregular Rhythm
- QRS complexes may be somewhat wide (greater than .12 seconds)
- Non-conducted sinus impulses appear at irregular intervals
    - PRI is CONSTANT
- Rhythm is somewhat dangerous as the block is lower in the conduction system (BB level)
- May cause syncope or may deteriorate into complete heart block (3rd degree block)
- It's appearance in the setting of an acute MI identifies a high risk patient
- Cause: anterioseptal MI, fibrotic disease of the conduction system
- Treatment: may require pacemaker in the case of fibrotic deposits in the conduction system

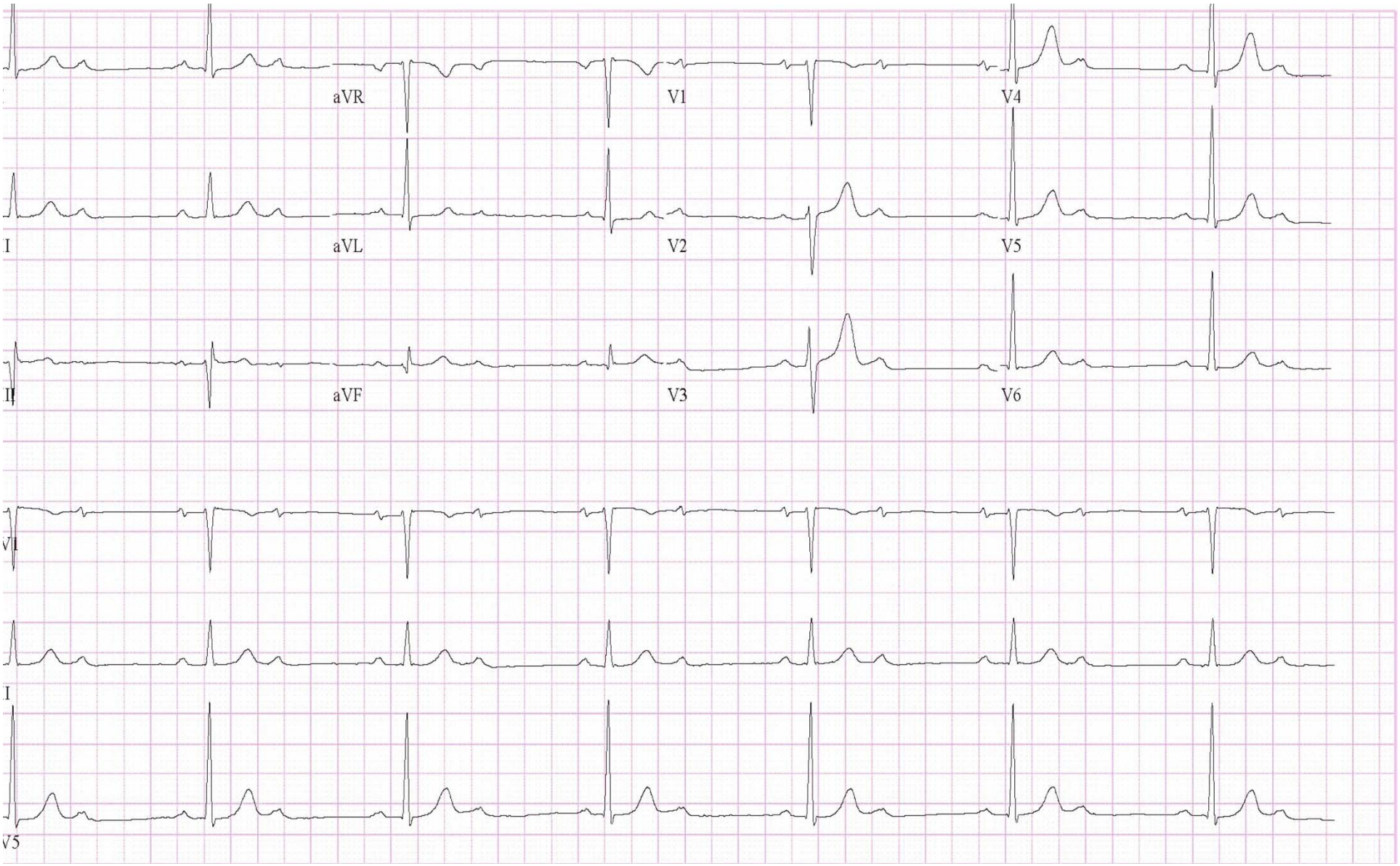


Examples of 2nd degree AV Block – Mobitz II



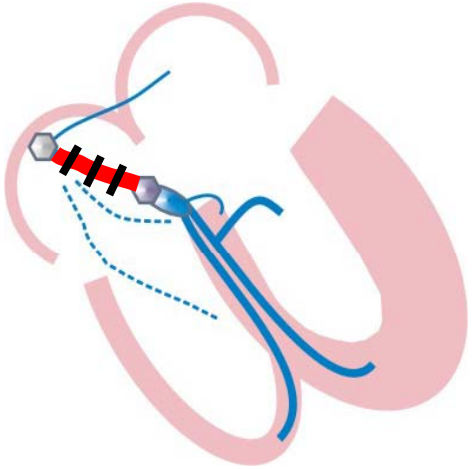


Examples of 2nd degree AV Block – Mobitz II

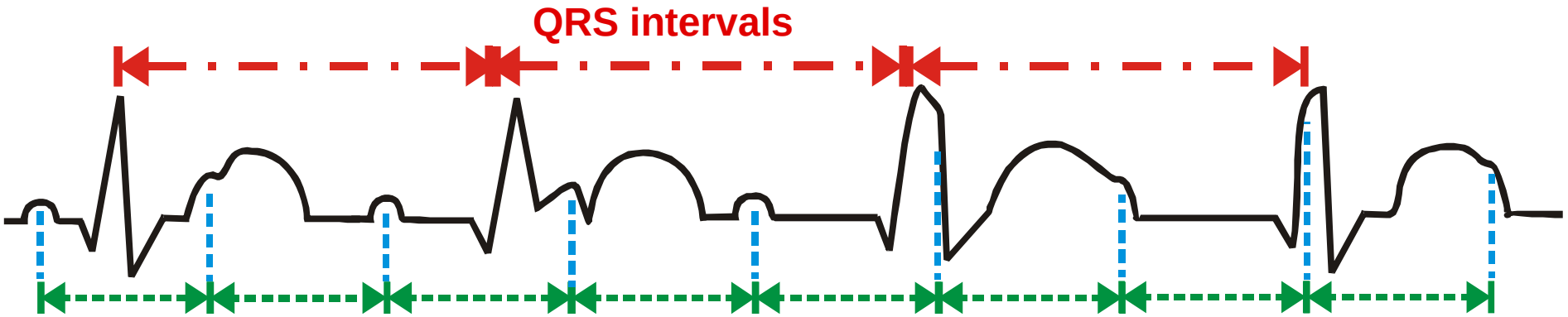


Recognizing and Naming Conduction Problems \ Rhythms

3rd degree AV Block ("Complete Heart Block"):

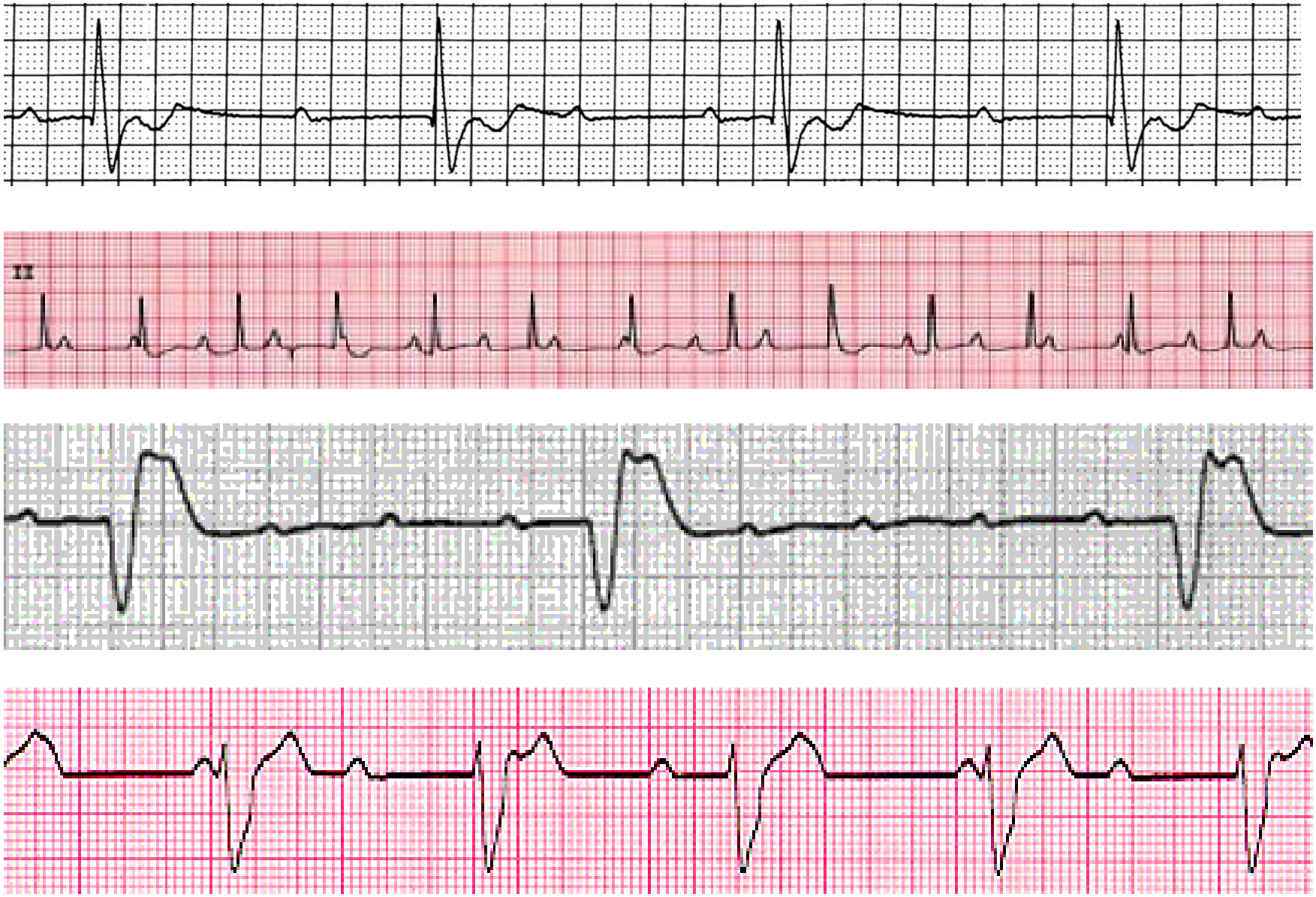


- Irregular Rhythm
- QRS complexes may be narrow or broad
- Atria and ventricles beat independent of one another (AV dissociation)
  - QRS's have their own rhythm, P-waves have their own rhythm
- May be caused by inferior MI -- it's presence worsens the prognosis
- May cause syncopal symptoms or angina, especially if ventricular rate is low
  - Also remember there is loss of atrial kick to ventricular filling → ↓ $\dot{Q}$
- Treatment: usually requires pacemaker

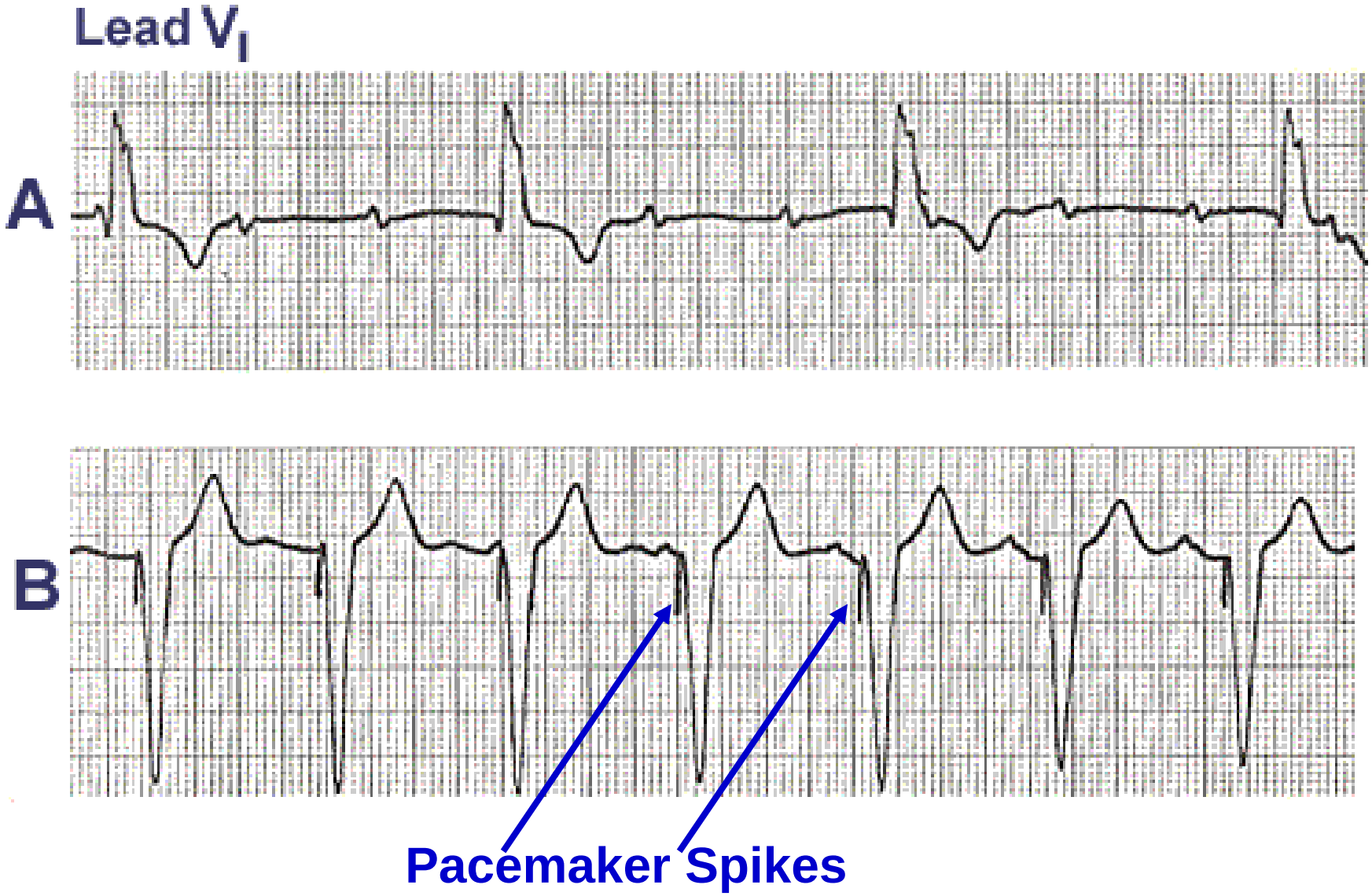


P-wave intervals – note how the P-waves sometimes distort QRS complexes or T-waves

Examples of 3rd degree AV Block – Complete Heart Block



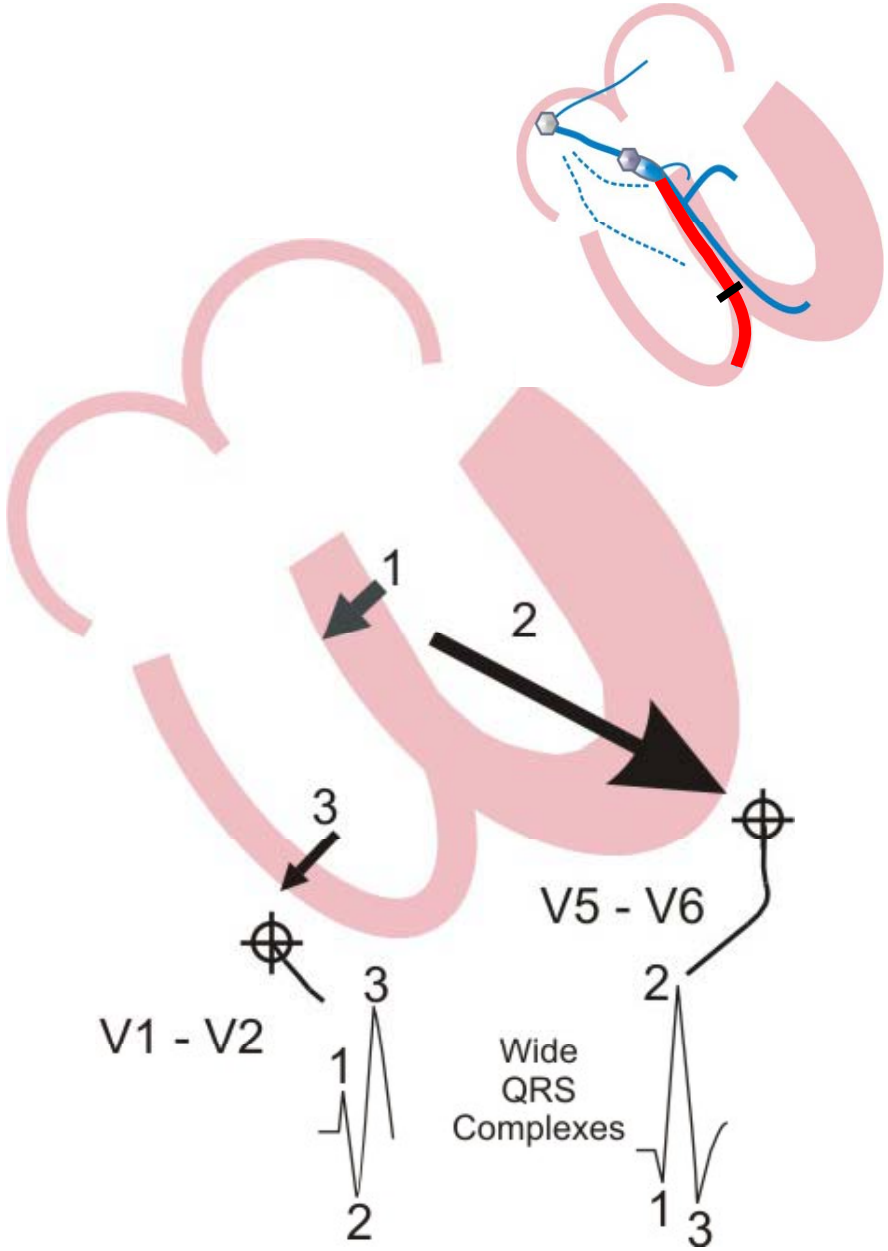
Example of 3rd degree AV Block – Complete Heart Block



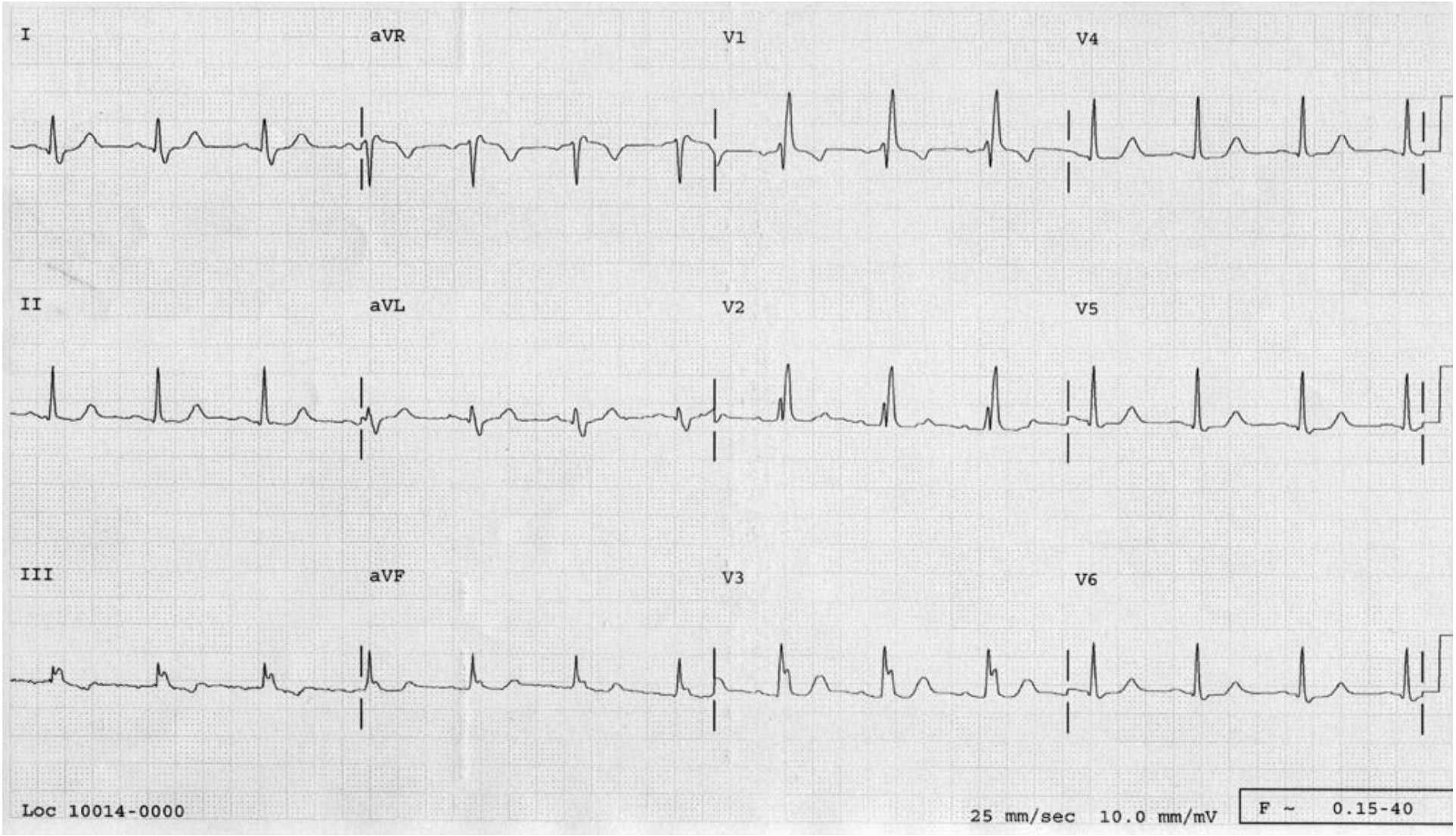
Recognizing and Naming Conduction Problems \ Rhythms

Right Bundle Branch Block (RBBB):

1. Septum depolarization occurs first inscribing an initial upward deflection in V1 - V2 and a small downward deflection in V5 - V6.
2. Left ventricular depolarization occurs next, inscribing a downward deflection in V1 - V2 and an upward deflection in V5 - V6. Since the right bundle branch is blocked, depolarization of the right ventricle is delayed.
3. Finally, depolarization spreads from the left ventricle over to the right ventricle and the right ventricle depolarizes. This inscribes a second R-wave (R') in V1 - V2, and sometimes, a slight S-wave in V5 - V6.



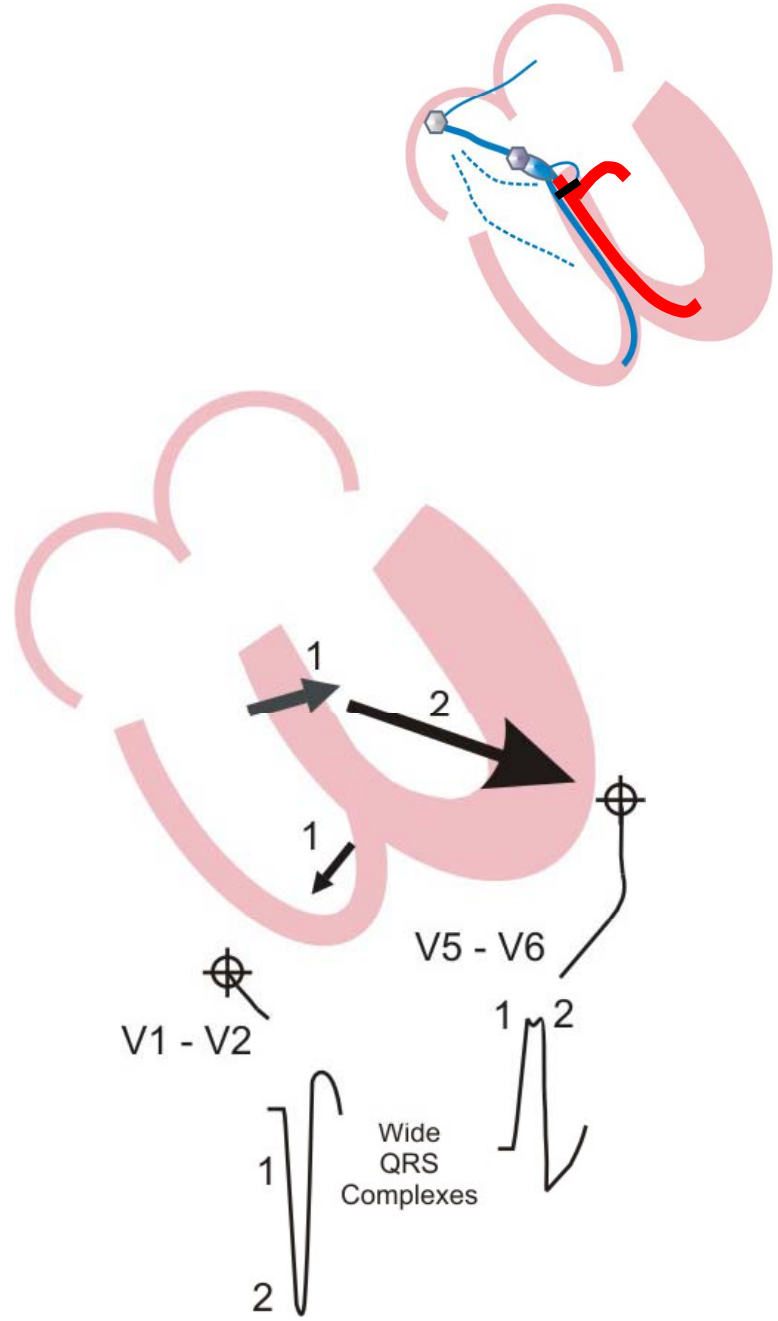
Example of Right Bundle Branch Block (RBBB)



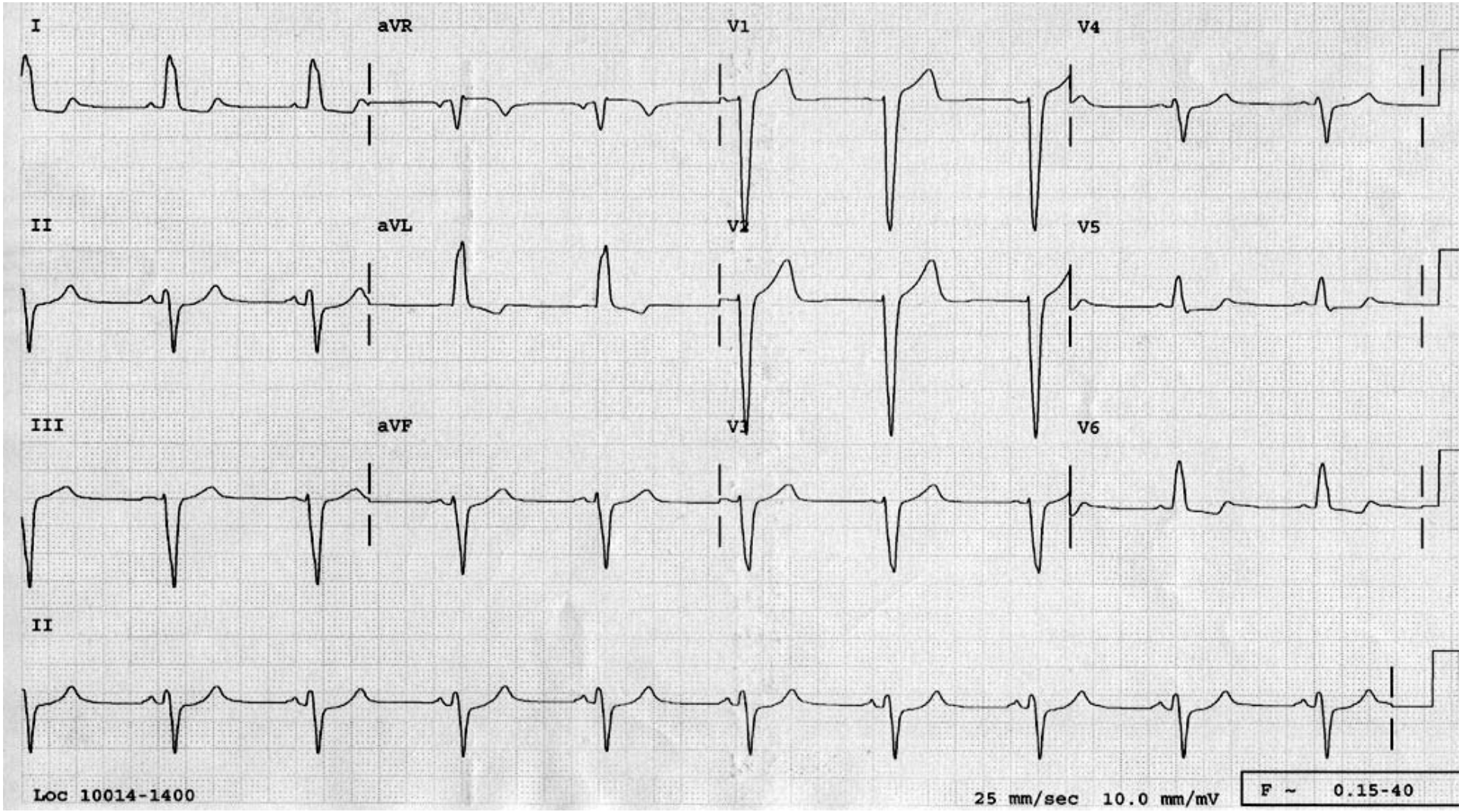
Recognizing and Naming Conduction Problems \ Rhythms

Left Bundle Branch Block (LBBB):

1. Depolarization enters the right side of the right ventricle first and simultaneously depolarizes the septum from right to left. Since the septum has more mass (and thus contributes more electricity to the depolarization vector), the dominant force moves away from V1 - V2 and inscribes a negative deflection in those leads. Leads V5 - V6 show a positive deflection.
2. Having spread over from the right ventricle, left ventricular depolarization continues and generates the main cardiac vector. This too is moving away from V1 - V2 and continues to inscribe a negative complex. Likewise, the vector proceeds toward V5 - V6 and continues to inscribe a positive complex. A slight notching of the R-wave may sometimes be seen in V5 - V6.



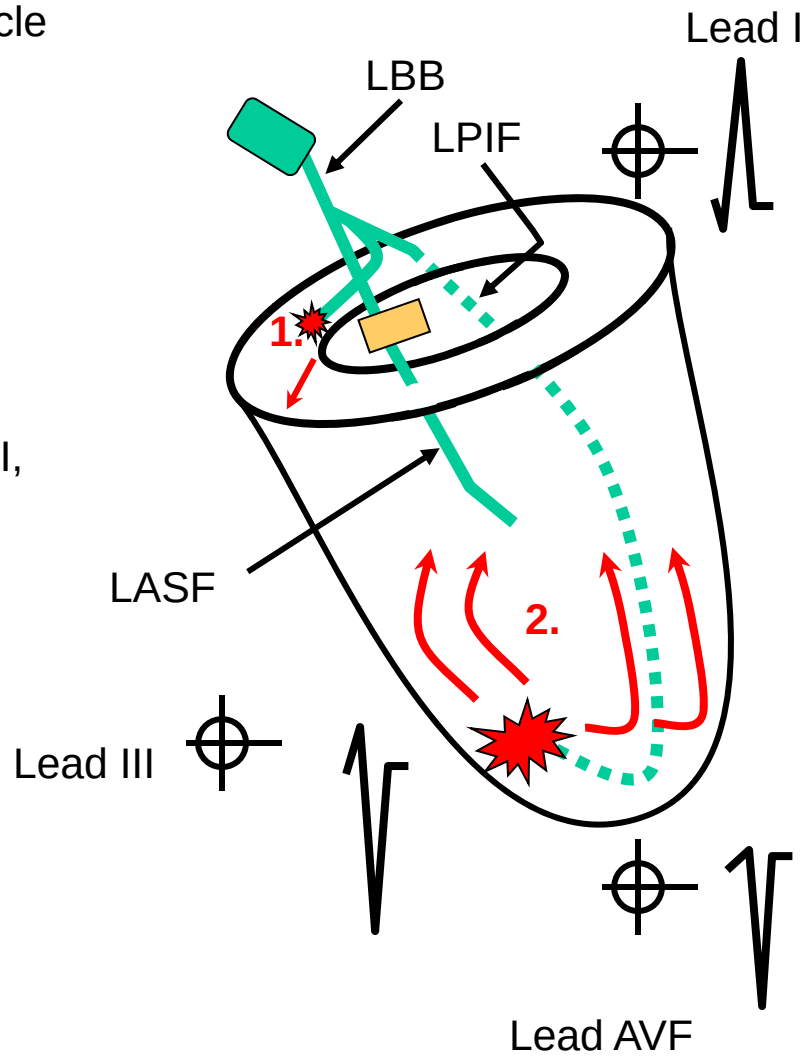
Example of Left Bundle Branch Block (LBBB)



Recognizing and Naming Conduction Problems \ Rhythms

Left Anterior Hemiblock (LAHB):

1. Conduction down the left anterior superior fascicle is blocked ➞ After initial septal depolarization provided by the septal fascicle of the LBB, depolarization spreads normally down the left posterior inferior fascicle and depolarization proceeds from the “bottom up” and from “left to right”.
2. Left axis deviation (> -30 degrees) will be noted and there will be a prominent S-wave in Leads II, and III



Notes on (LAHB):

- QRS is normal width unless BBB is present
- May be seen in the setting of an acute MI
- May interfere with the diagnosis of an old inferior wall MI by abolishing the diagnostic Q-waves in II, III, and AVF
- May interfere with the diagnosis of an old anterior wall MI because it produces small R-waves in V1 and V2

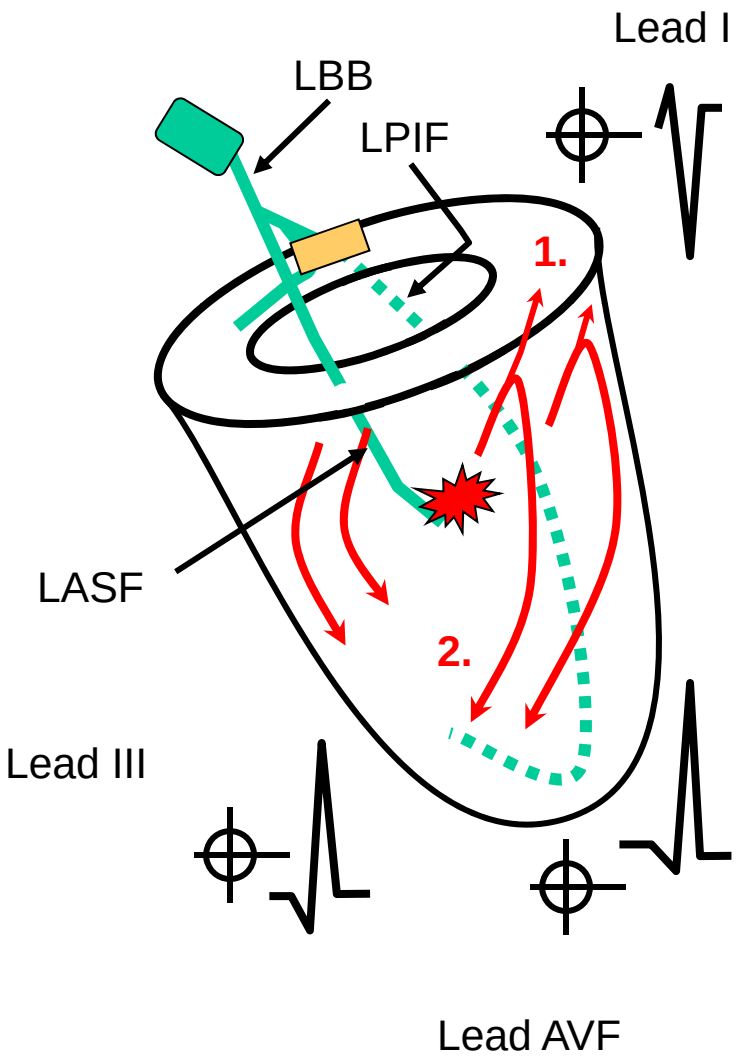
Recognizing and Naming Conduction Problems \ Rhythms

Left Posterior Hemiblock (LPHB):

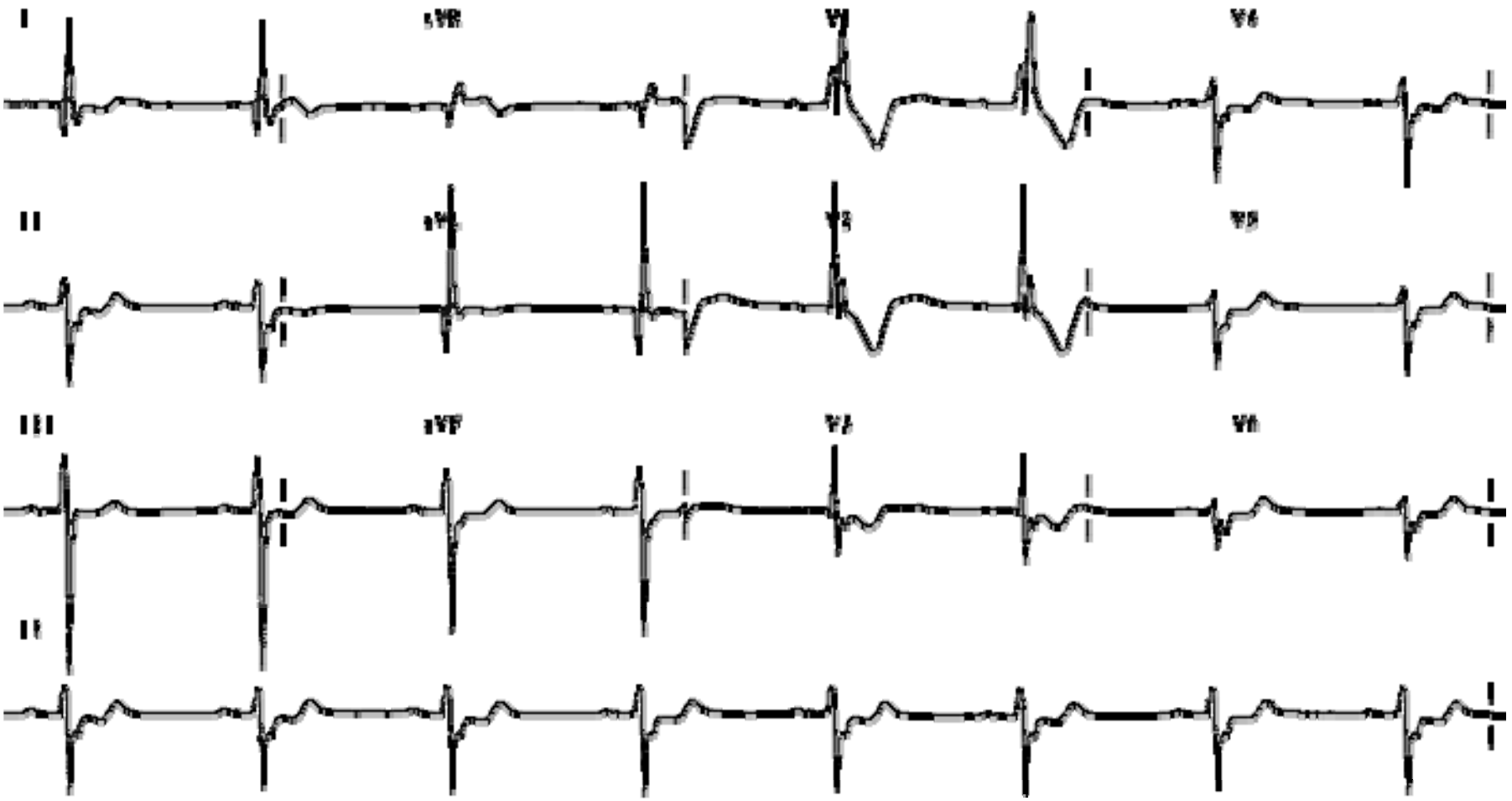
1. Conduction down the left posterior fascicle is blocked ➞ Activation of the left anterior superior fascicle produces initial forces toward the high lateral wall of the left ventricle, then depolarization spreads from the “top down” and from “left to right”.
2. Right axis deviation (≥ 120 degrees) will be noted and there will be a prominent S-wave in Leads I. Q-waves may be noted in III and AVF.

Notes on (LPHB):

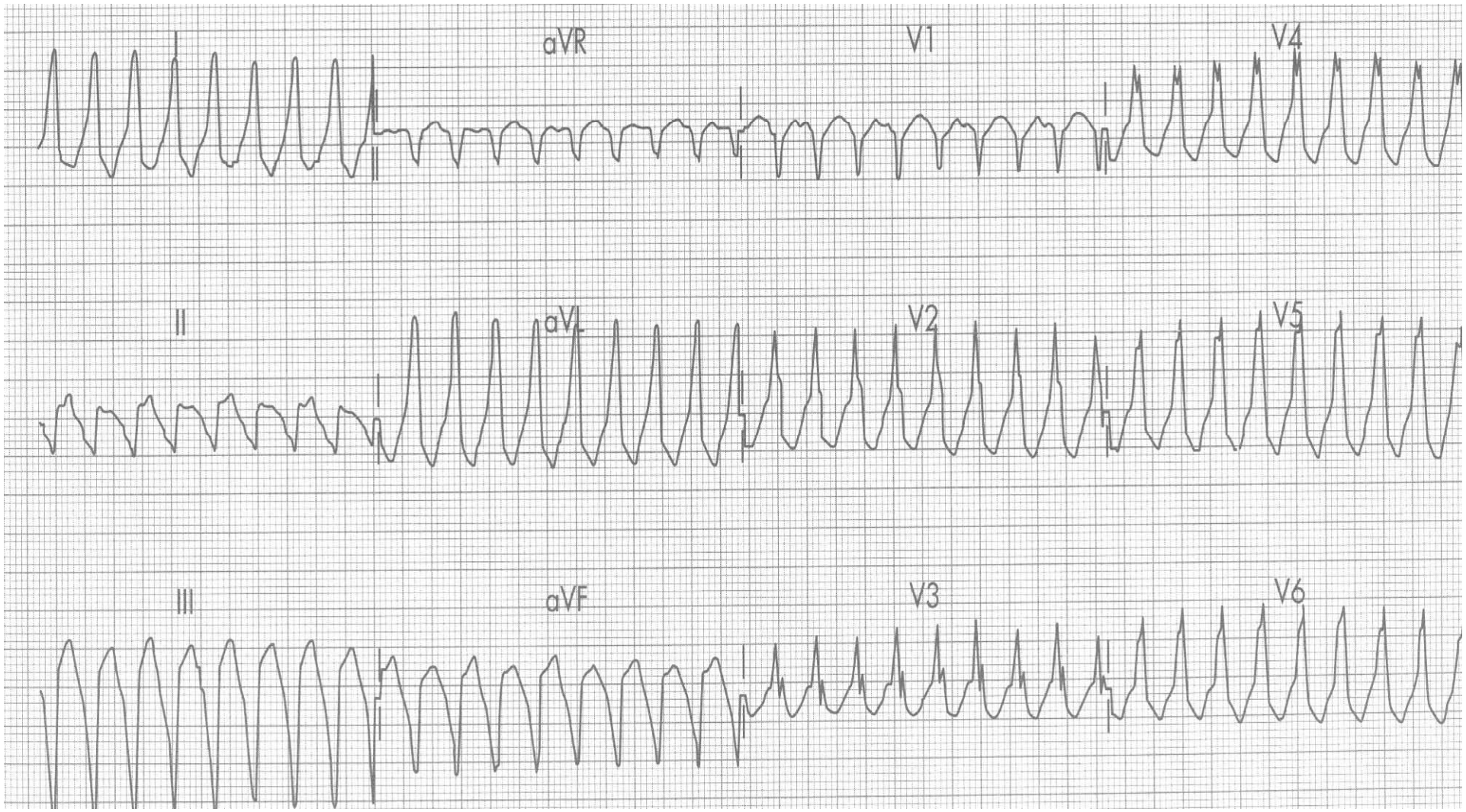
- QRS is normal width unless BBB is present
- If LPHB occurs in the setting of an acute MI, it is almost always accompanied by RBBB and carries a mortality rate of 71%



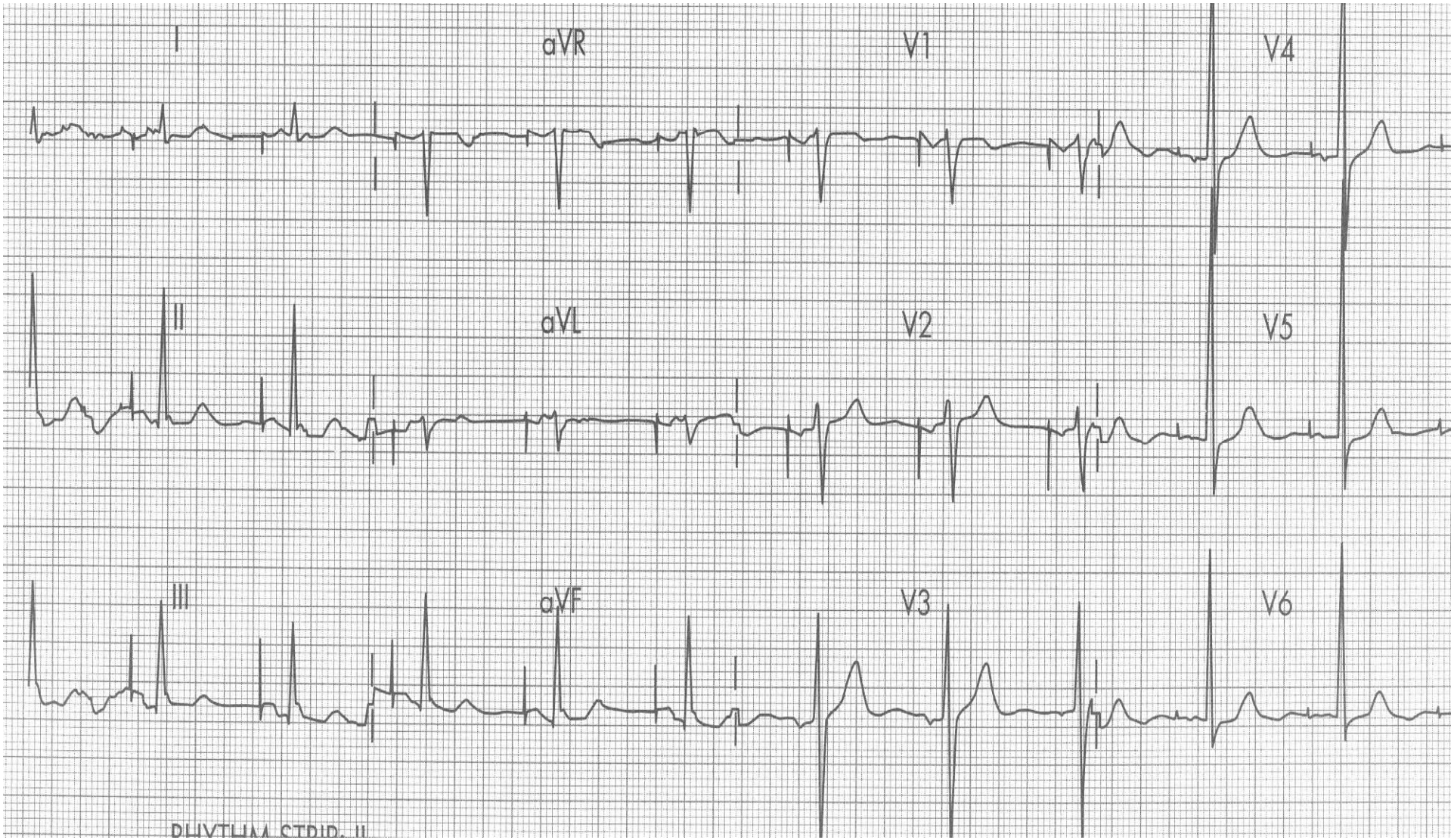
What do you see?



What do you see?

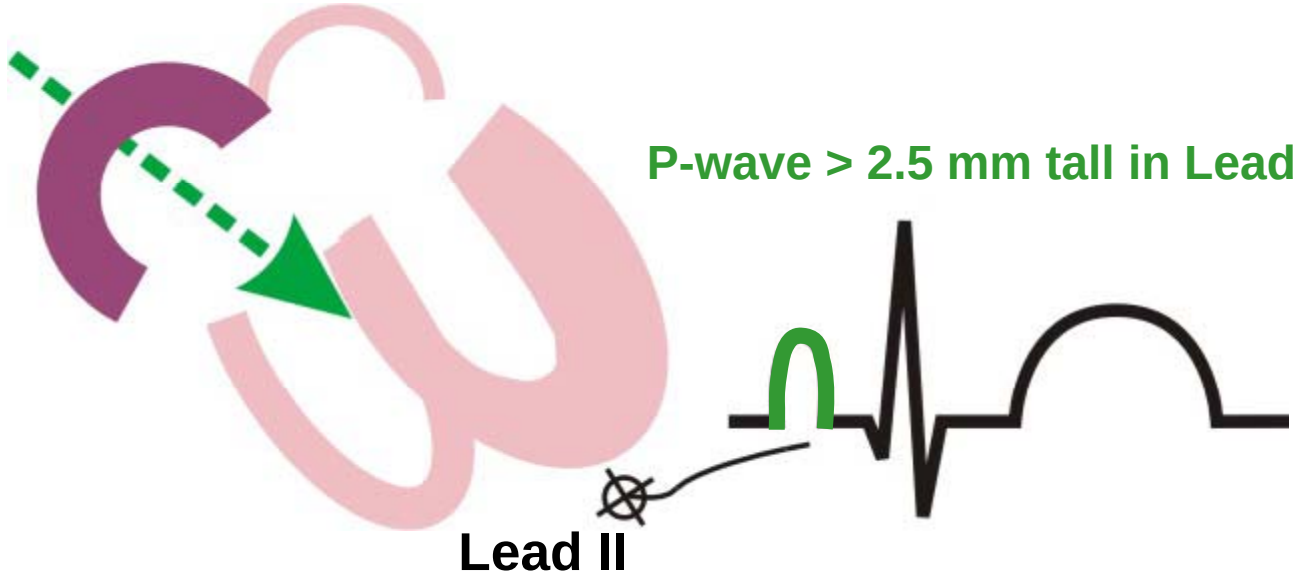


What do you see?



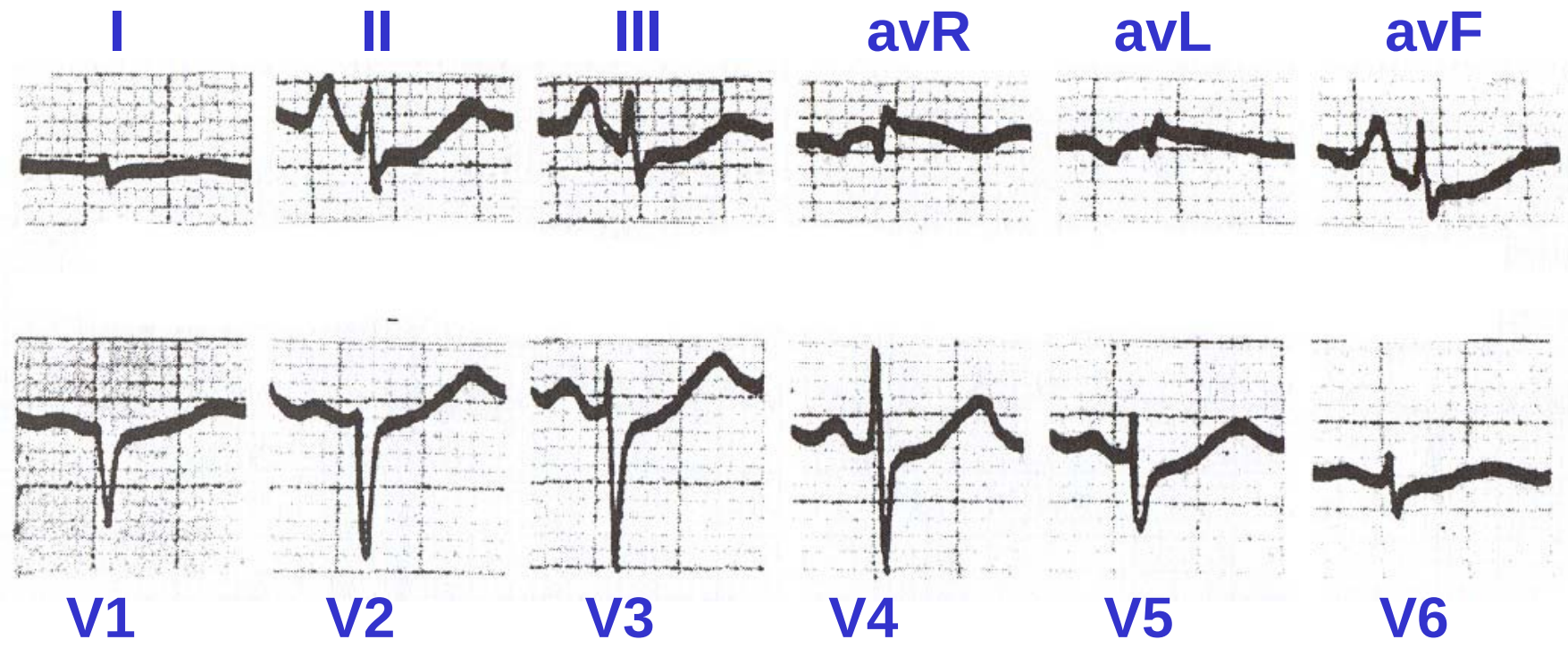
Chamber Enlargement

- **Right Atrial Enlargement (Hypertrophy)**
  - Causes:
    - Tricuspid valve disease
    - Pulmonary hypertension, emphysema
  - Characteristics
    - **Tall P-wave (> 2.5 mm) in Lead II (P-Pulmonale)**
  - Changes with emphysema
    - Low P, QRS, and T voltage in Lead I with sloping PR segments in II and III

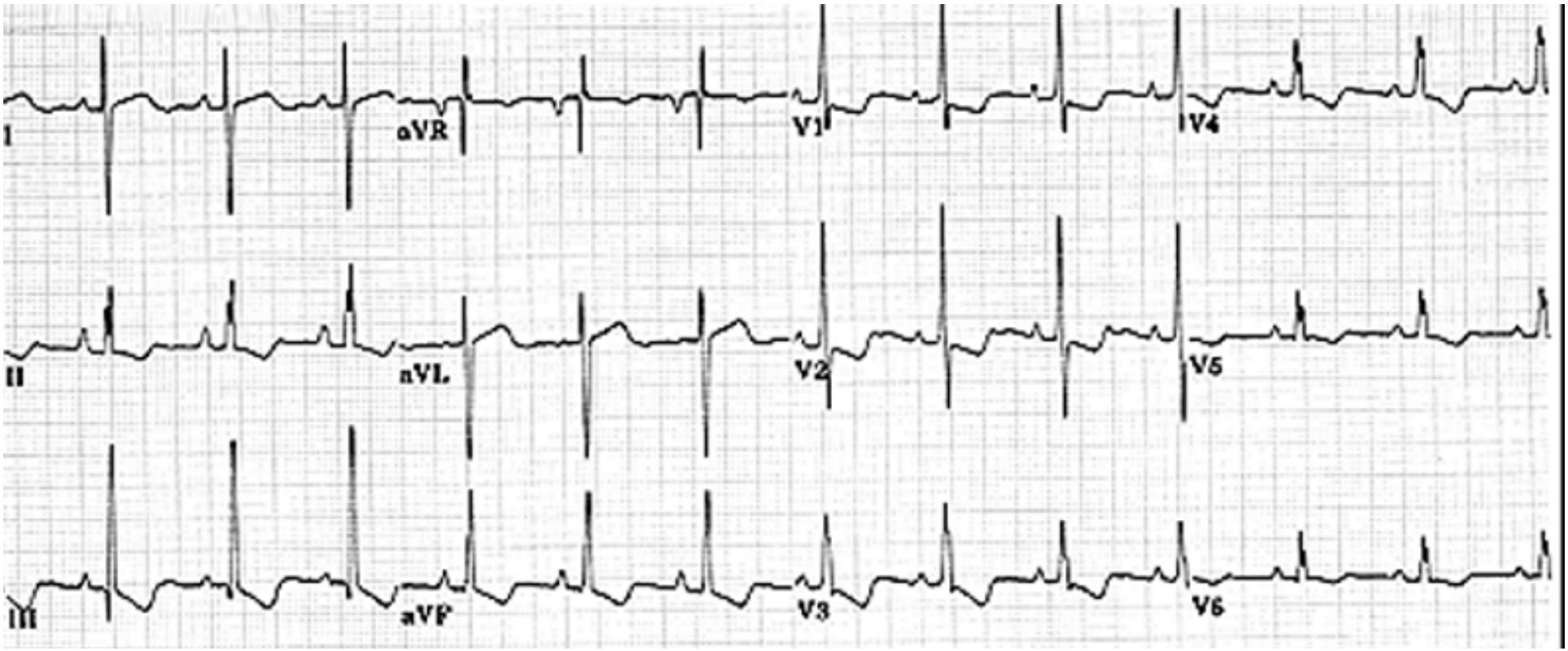


Chamber Enlargement

- **ECG of a Patient with emphysema → RA Hypertrophy**
  - **note the low voltage in Lead I**
  - **downsloping PR segment in Leads II and III**

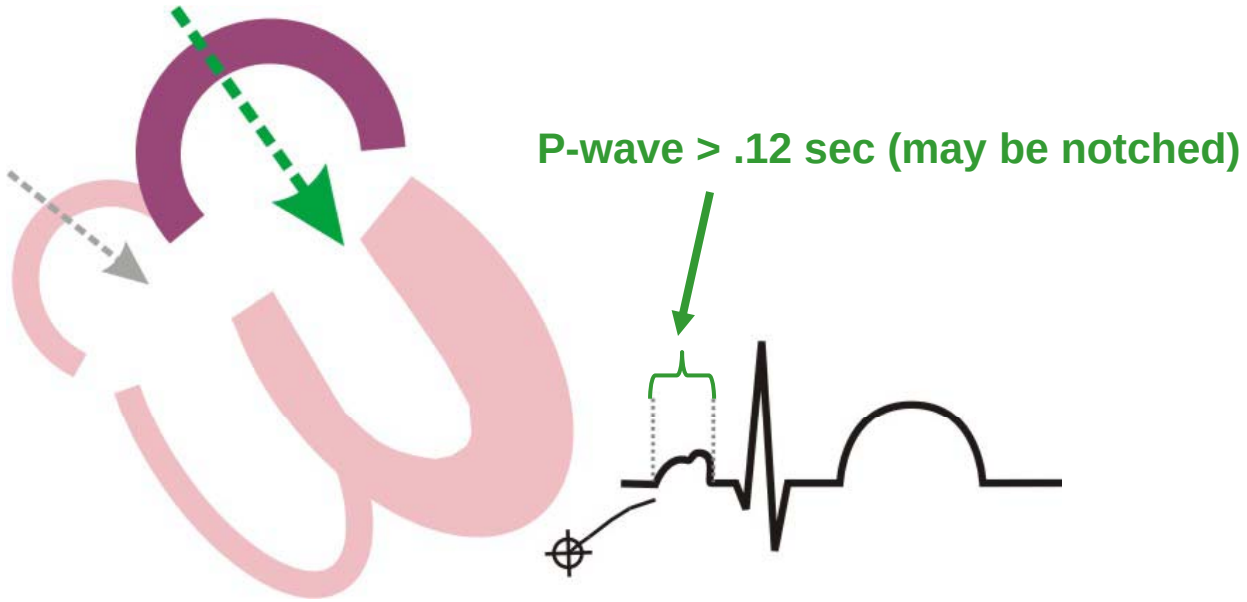


Example of Right Atrial Hypertrophy (Enlargement) (RAH) (RAE)

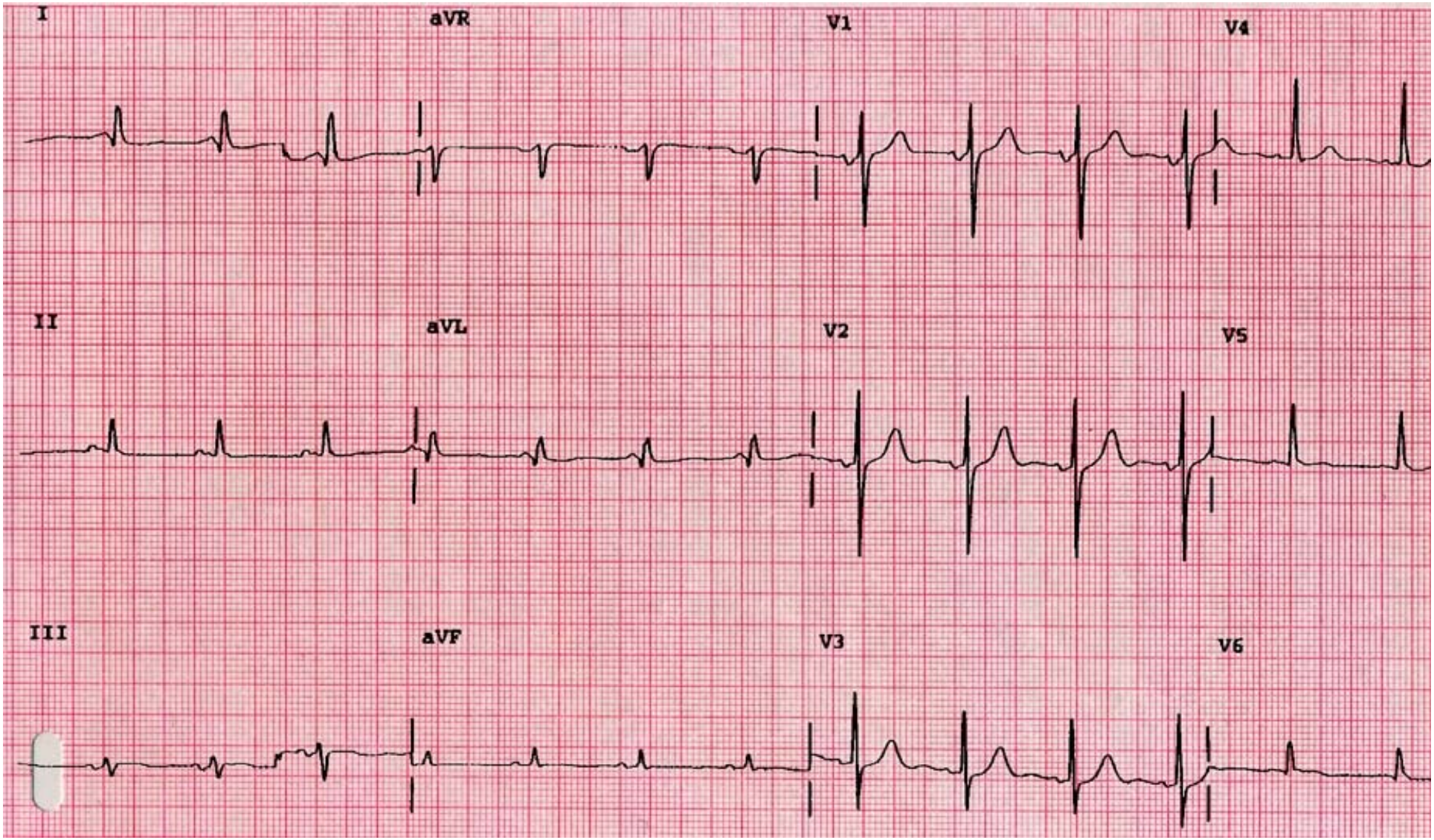


Chamber Enlargement

- **Left Atrial Enlargement (Hypertrophy)**
  - Causes:
    - Primary hypertension
    - Pulmonary edema (usually transient if edema subsides)
    - Mitral valve disease (notched P-wave in Lead II called “P-Mitrale”)
  - Characteristics
    - **Wide (> .12 sec) notched P-wave in Lead II**
    - Wide terminal trough in V1

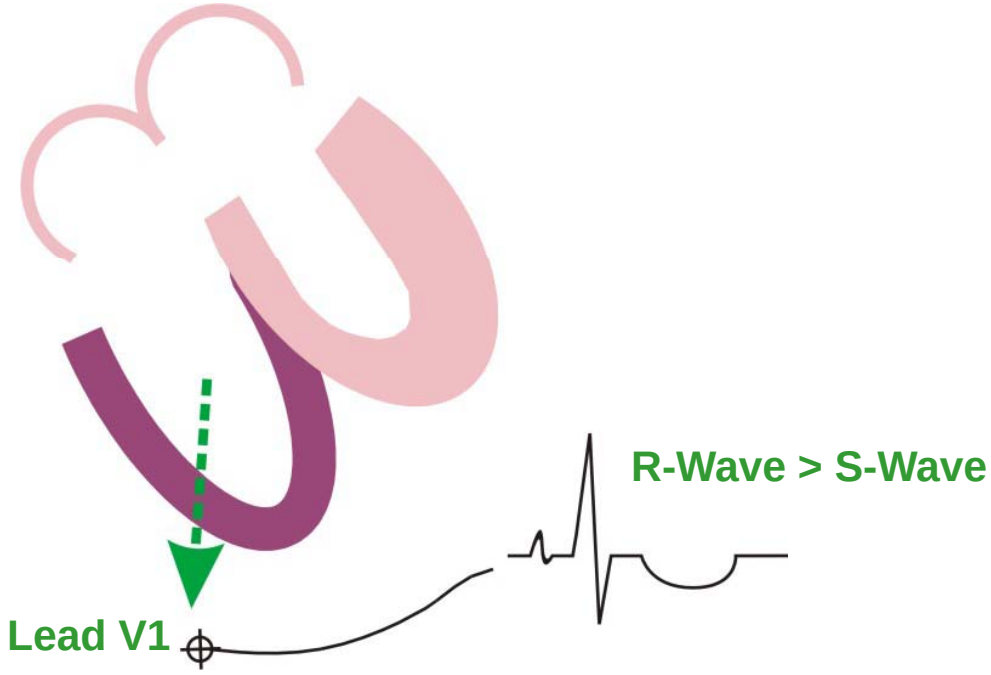


Example of Left Atrial Hypertrophy (Enlargement) (LAH) (LAE)

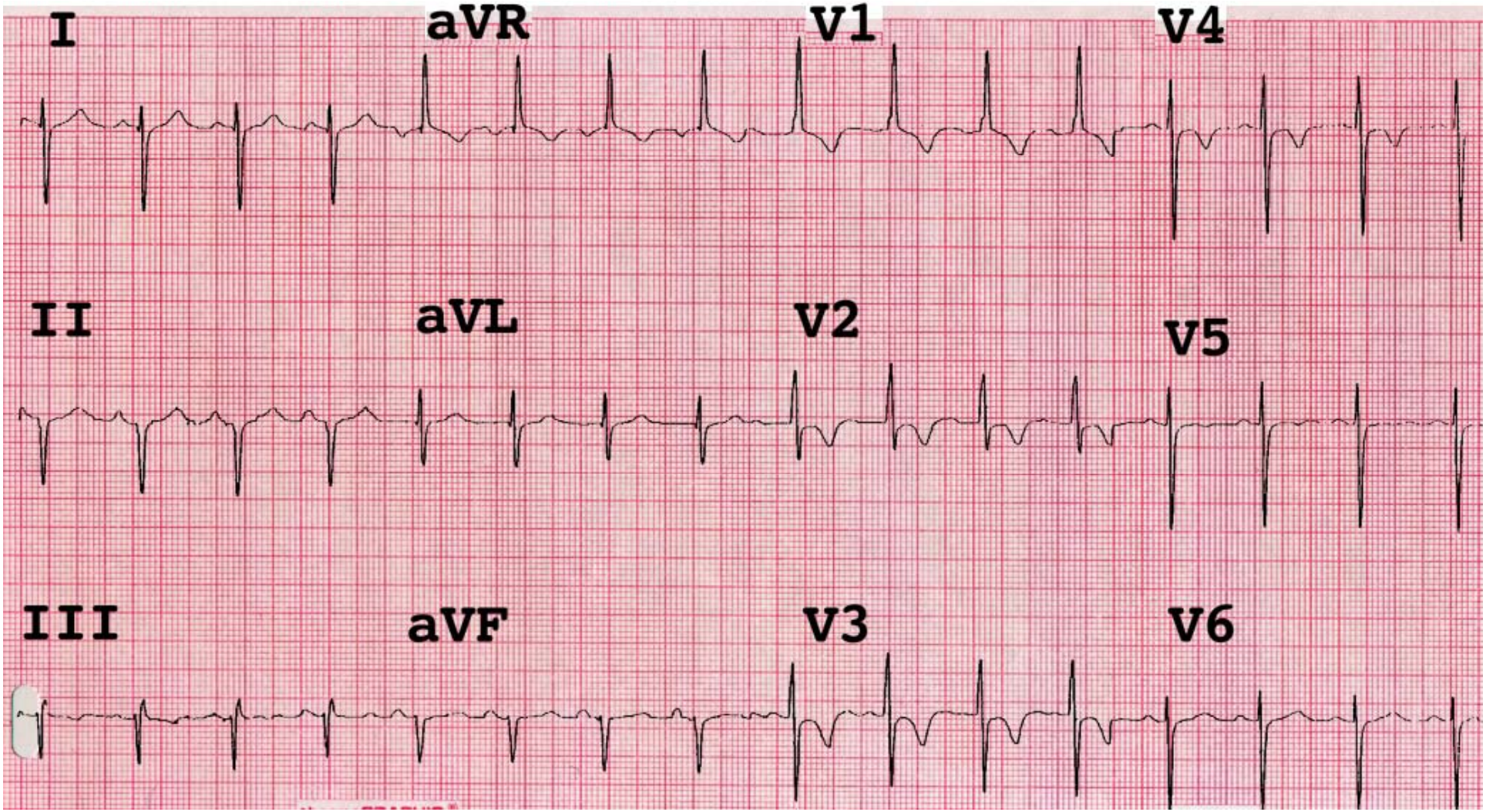


Chamber Enlargement

- **Right Ventricular Enlargement (Hypertrophy)**
  - Causes:
    - Pulmonary hypertension, Pulmonary stenosis, Tetralogy of Fallot
      - Tetralogy of Fallot:
        - Pulmonary stenosis,
        - Interventricular septal defect (ventricles communicate)
        - Aorta receives blood from both ventricles
        - RVH
  - Characteristics
    - **Any significant R-wave in V1 (usually accompanied by negative T-wave)**

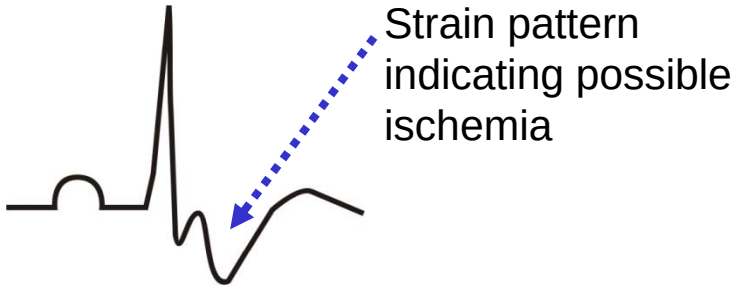


Example of Right Ventricular Hypertrophy (RVH)

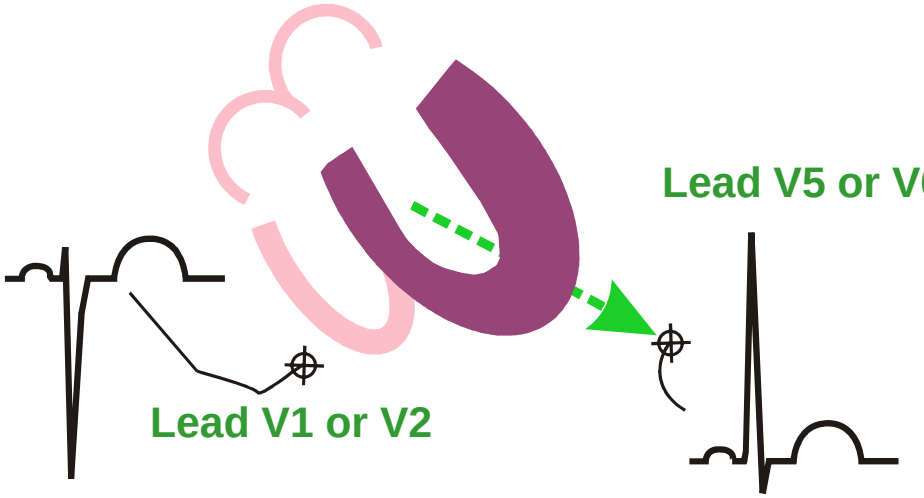


Chamber Enlargement

- **Left Ventricular Enlargement (Hypertrophy)**
  - Causes:
    - Primary hypertension (may produce strain pattern), Cardiomyopathy



  - Characteristics
    - Several diagnostic criteria to choose from…2 of the most popular
      - Largest S depth in V1 or V2 + largest R height in V5 or V6 > 35 mm
      - R in AVL > 11mm



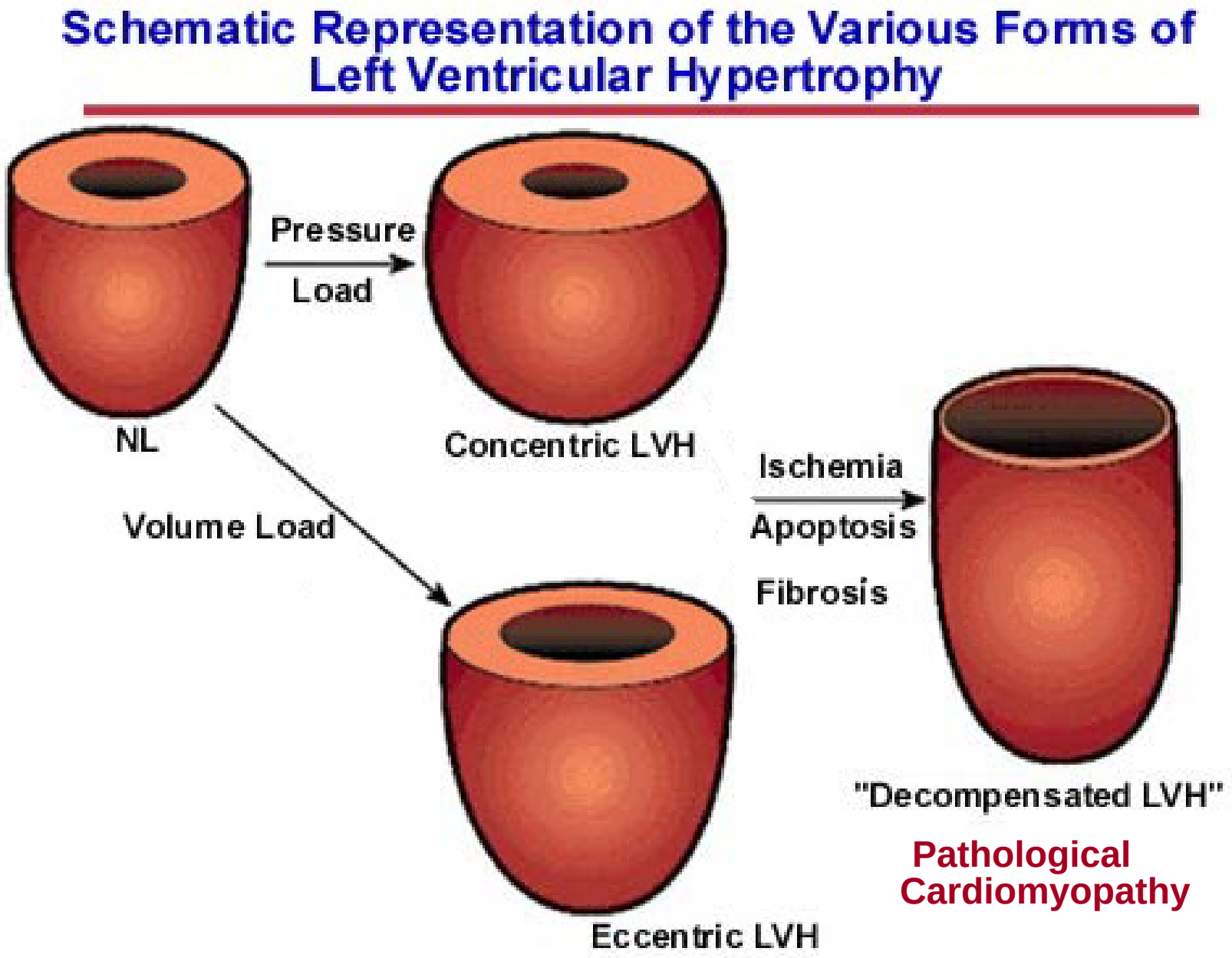
Depth of deepest S-wave in V1 or V2

+

Height of tallest R-wave in V5 or V6

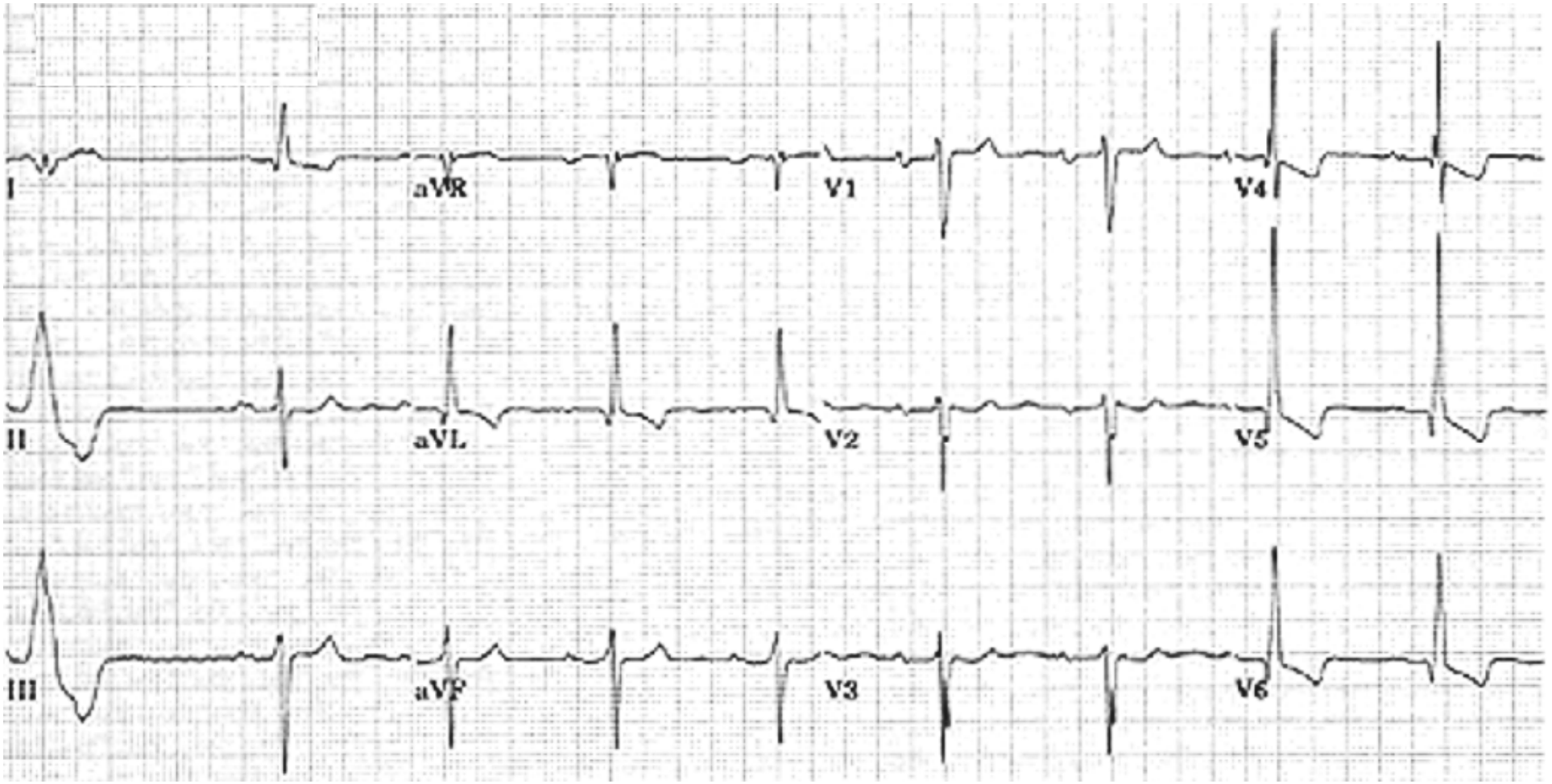
> 35 mm….. voltage criteria for LVH

Chamber Enlargement

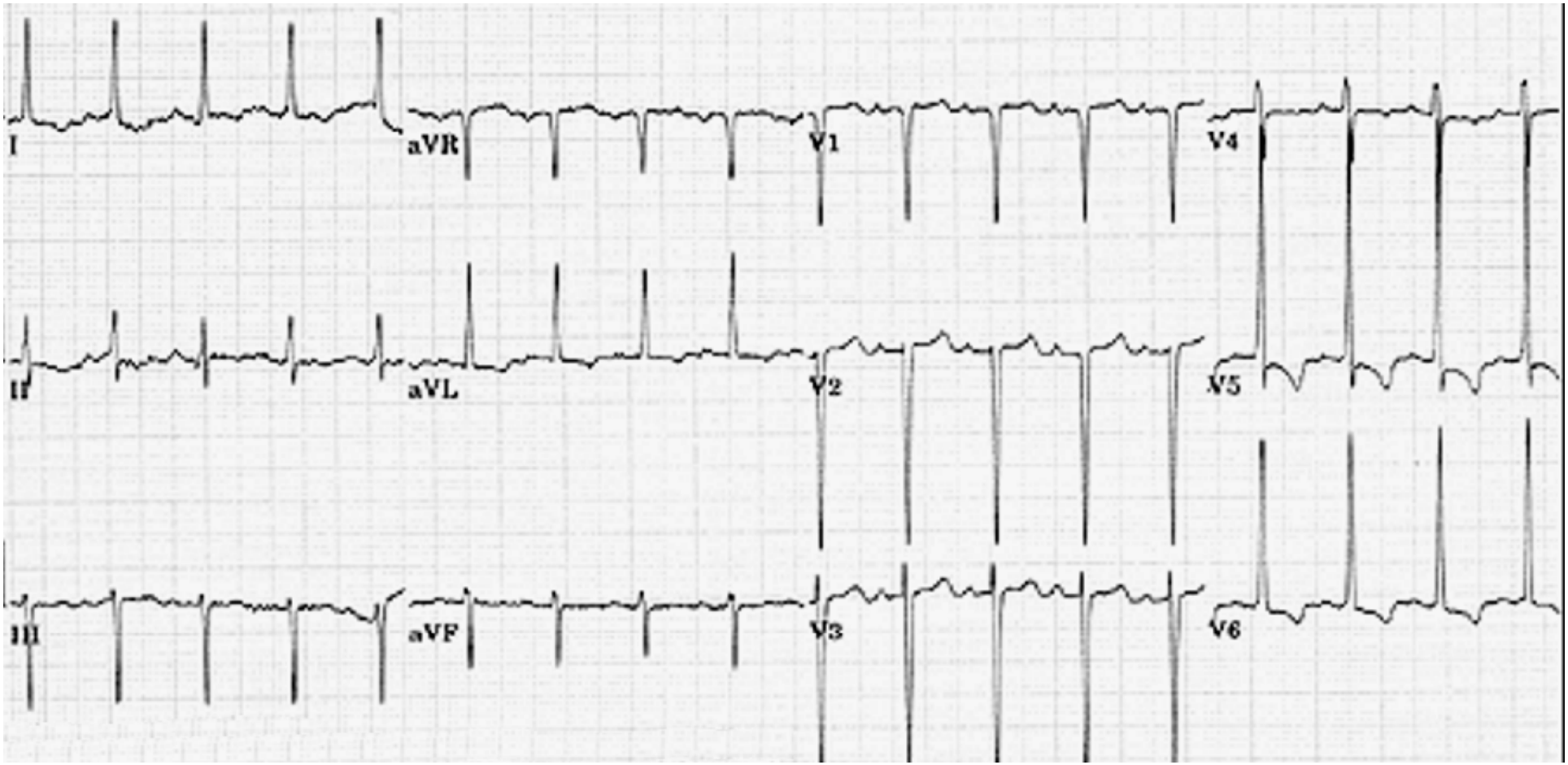


Pathological Cardiomyopathy

Example of Left Ventricular Hypertrophy (LVH)



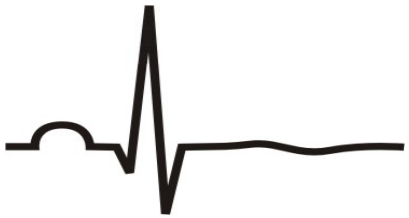
Example of Left Ventricular Hypertrophy (LVH)



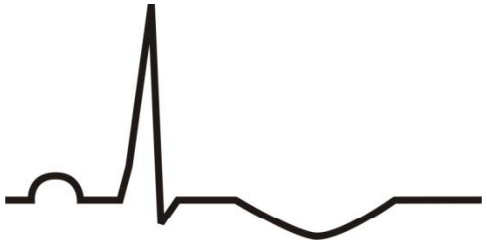
Ischemia & Infarction

- **Ischemia at rest: "non-specific T and ST changes"**
  - **changes in the T-wave or the ST segment that are "out of place"**
    - **normally, the T-wave and the QRS complex have similar polarity**

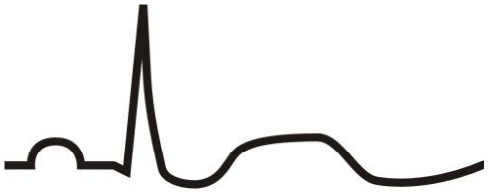
- **T-wave flattening:**



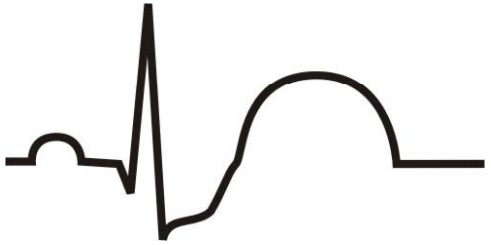
- **T-wave inversion:**



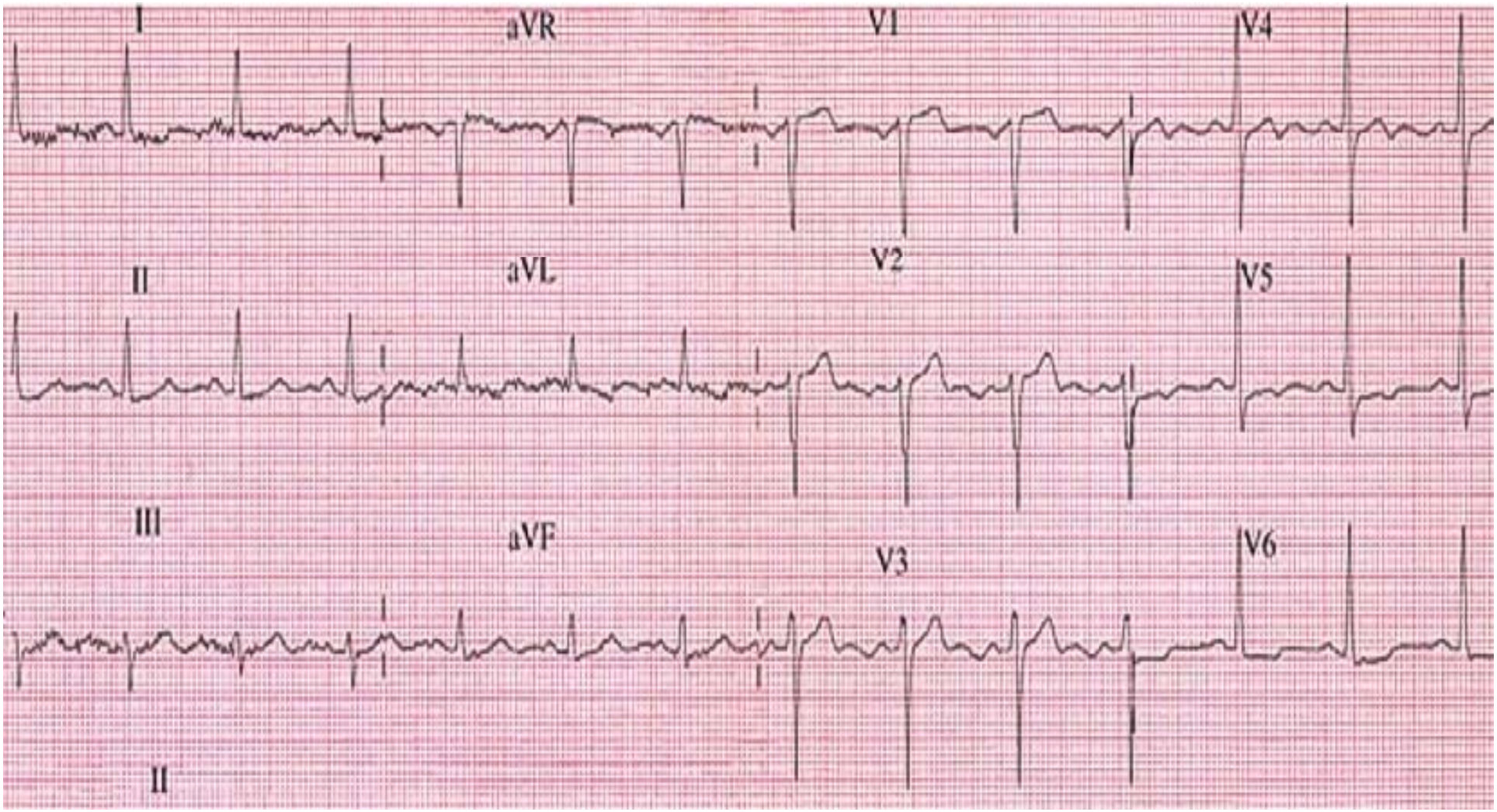
- **ST-segment scooping:**



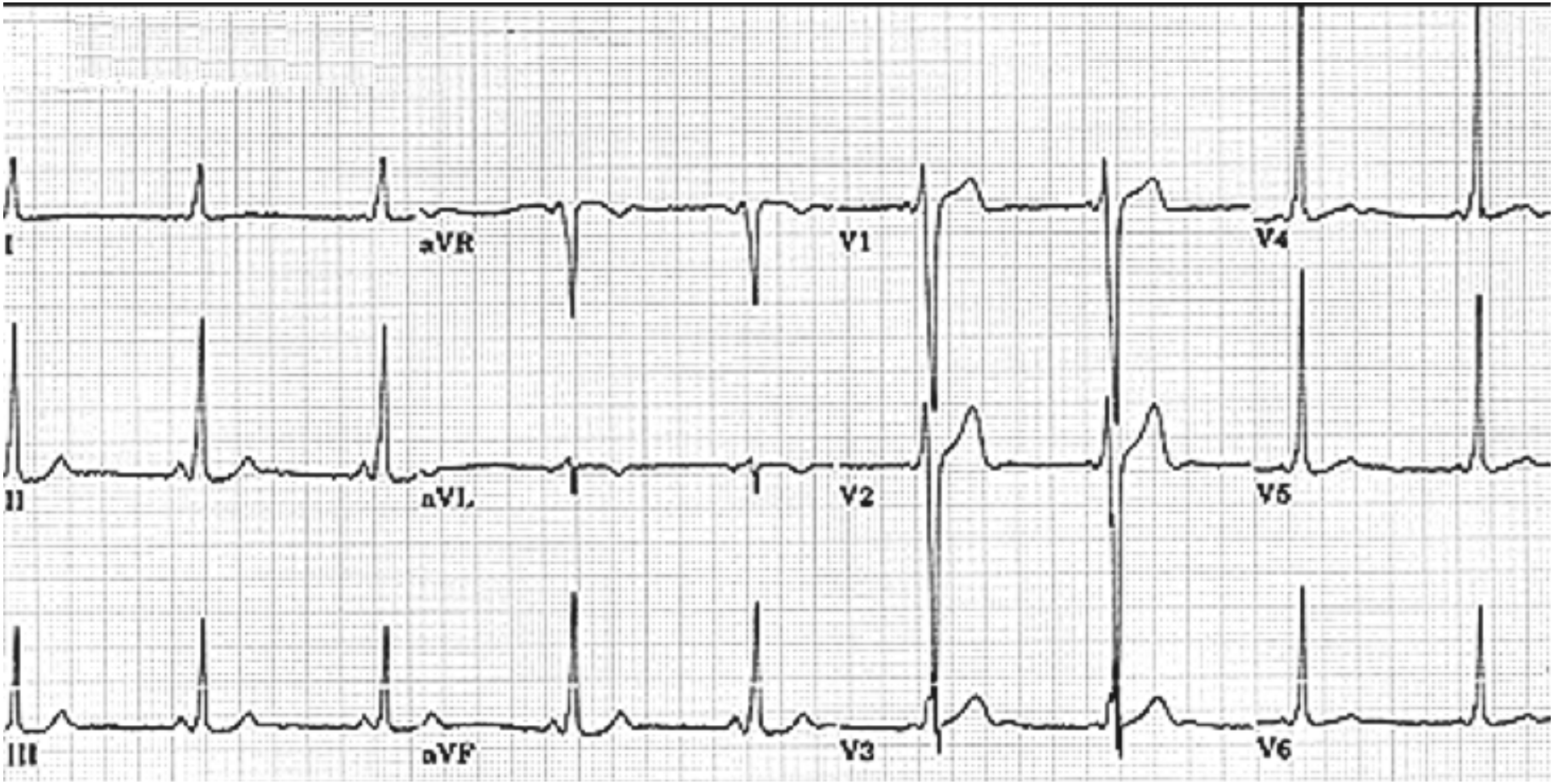
- **ST-segment depression**



T-wave Flattening / Inversion: Ischemia

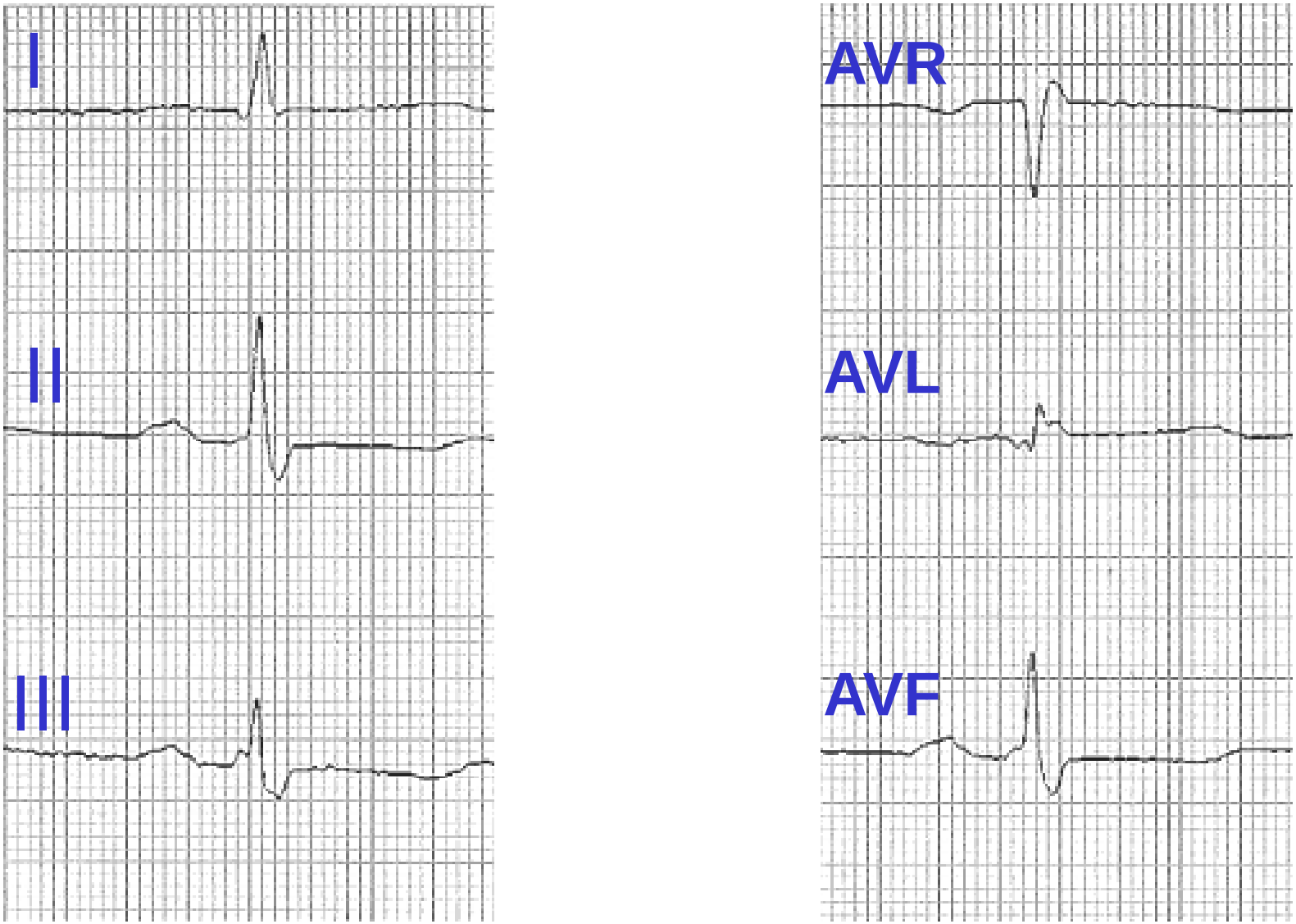


T-wave Flattening & ST Scooping: Ischemia



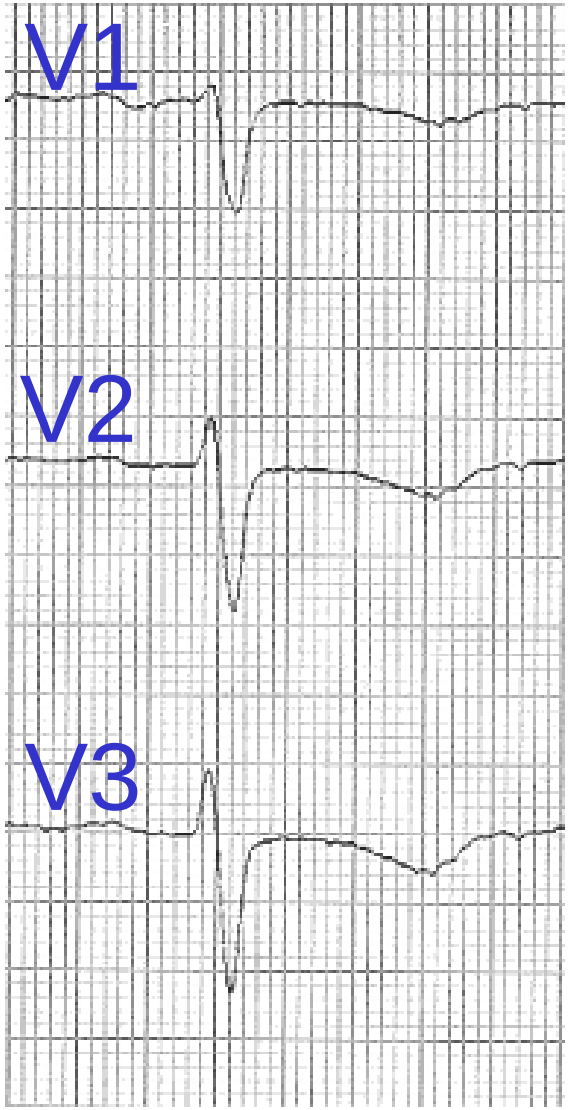
Ischemia

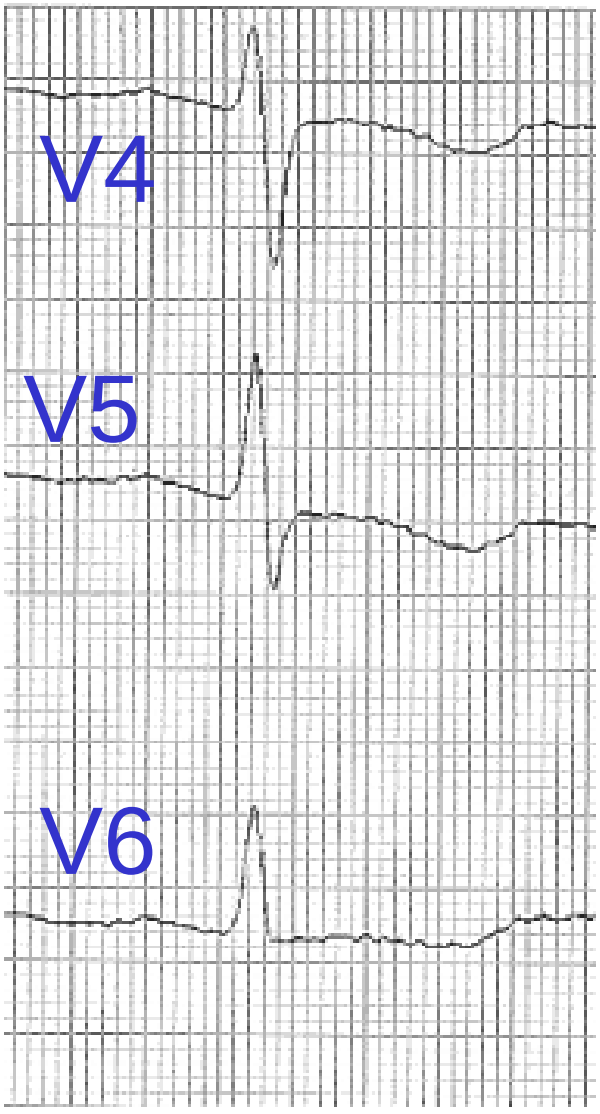
Inferior Ischemia in a 42 year old male while at rest



Ischemia

Anteriolateral Ischemia in a 67 year old female while at rest

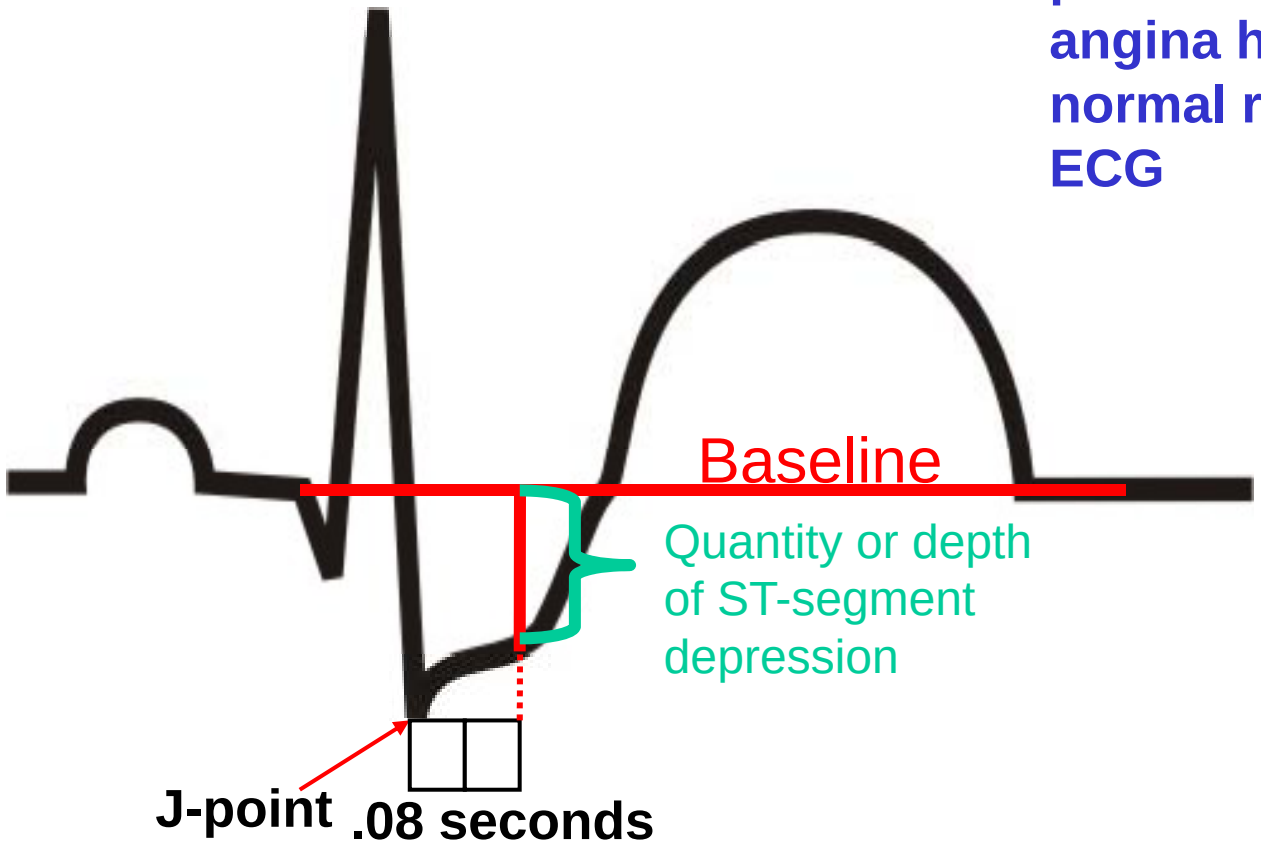




Ischemia

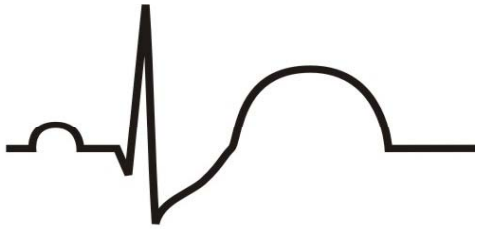
- **Ischemia during exercise: "ST-segment depression"**
  - **Usually indicative of subendocardial ischemia**
  - **Location of ischemia does not always correspond to the leads in which it is seen**

NOTE: 50% of patients with angina have a normal resting ECG

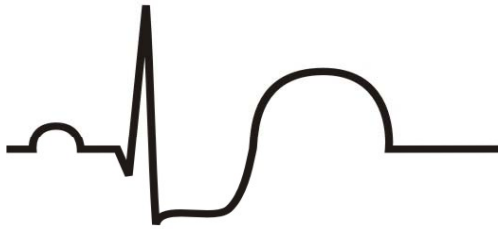


Ischemia

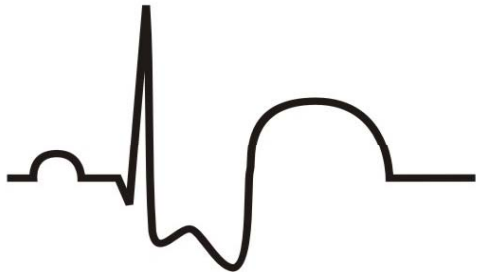
- **Types of ST-segment depression**



UPSLOPING – very nonspecific for the diagnosis of ischemia. Associated with a lot of false positive exercise tests.



HORIZONTAL – likely associated with ischemia.

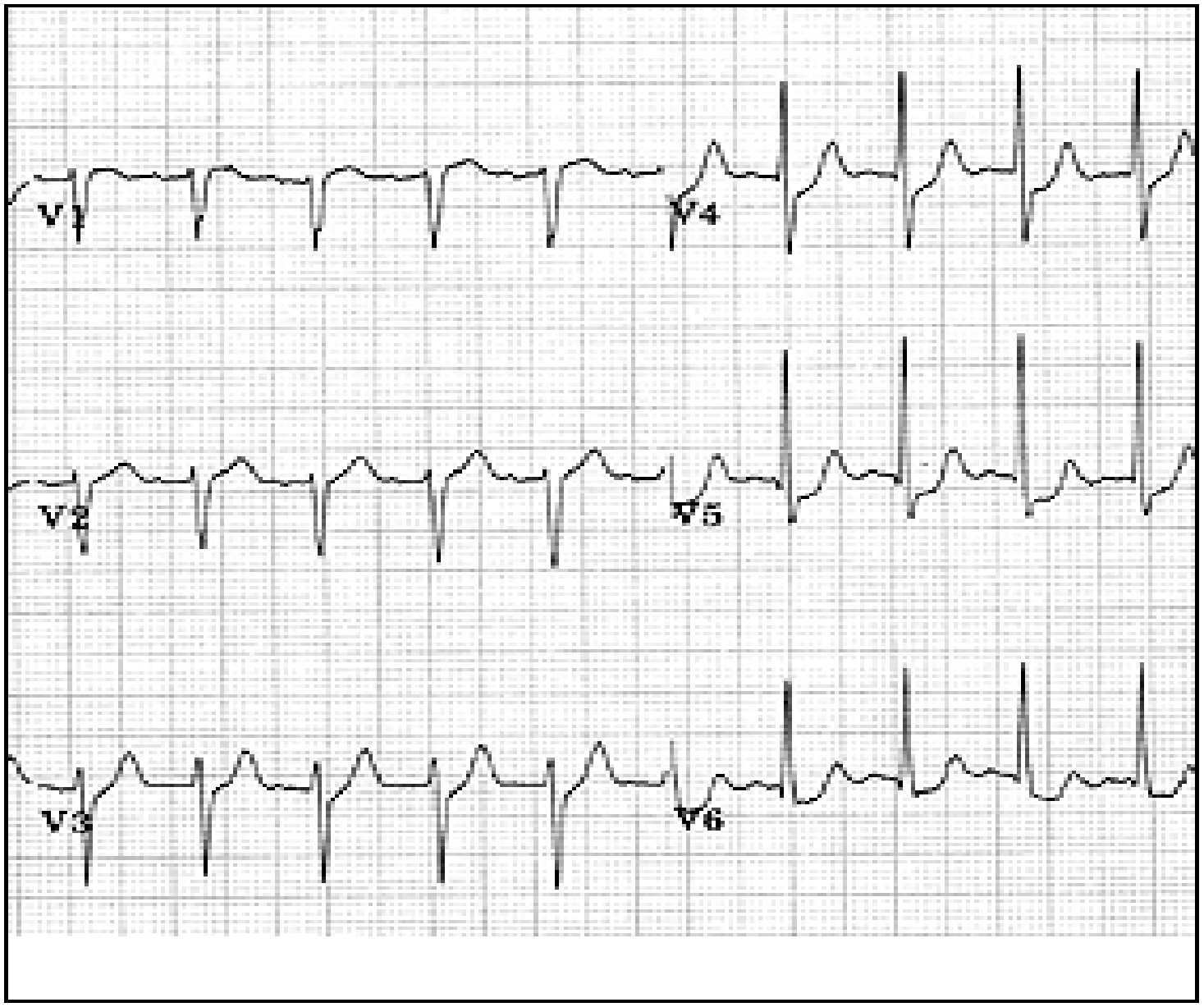


DOWNSLOPING – almost certainly associated with an ischemic myocardium

Ischemia

Ischemia during graded exercise test (GXT)

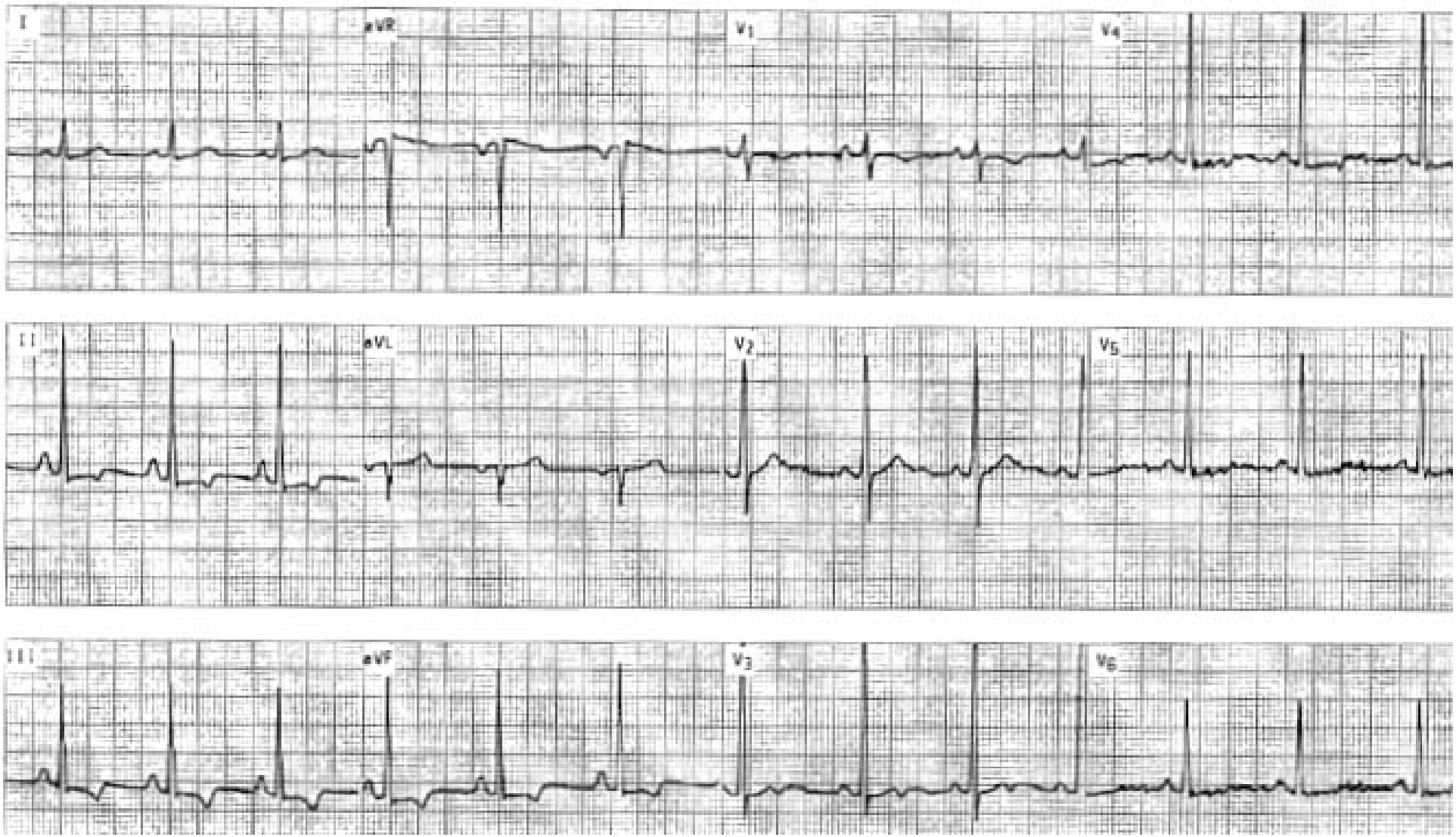
Horizontal-Upsloping ST depression in lateral leads



Stress Test #1: Ischemia at rest, pre-exercise

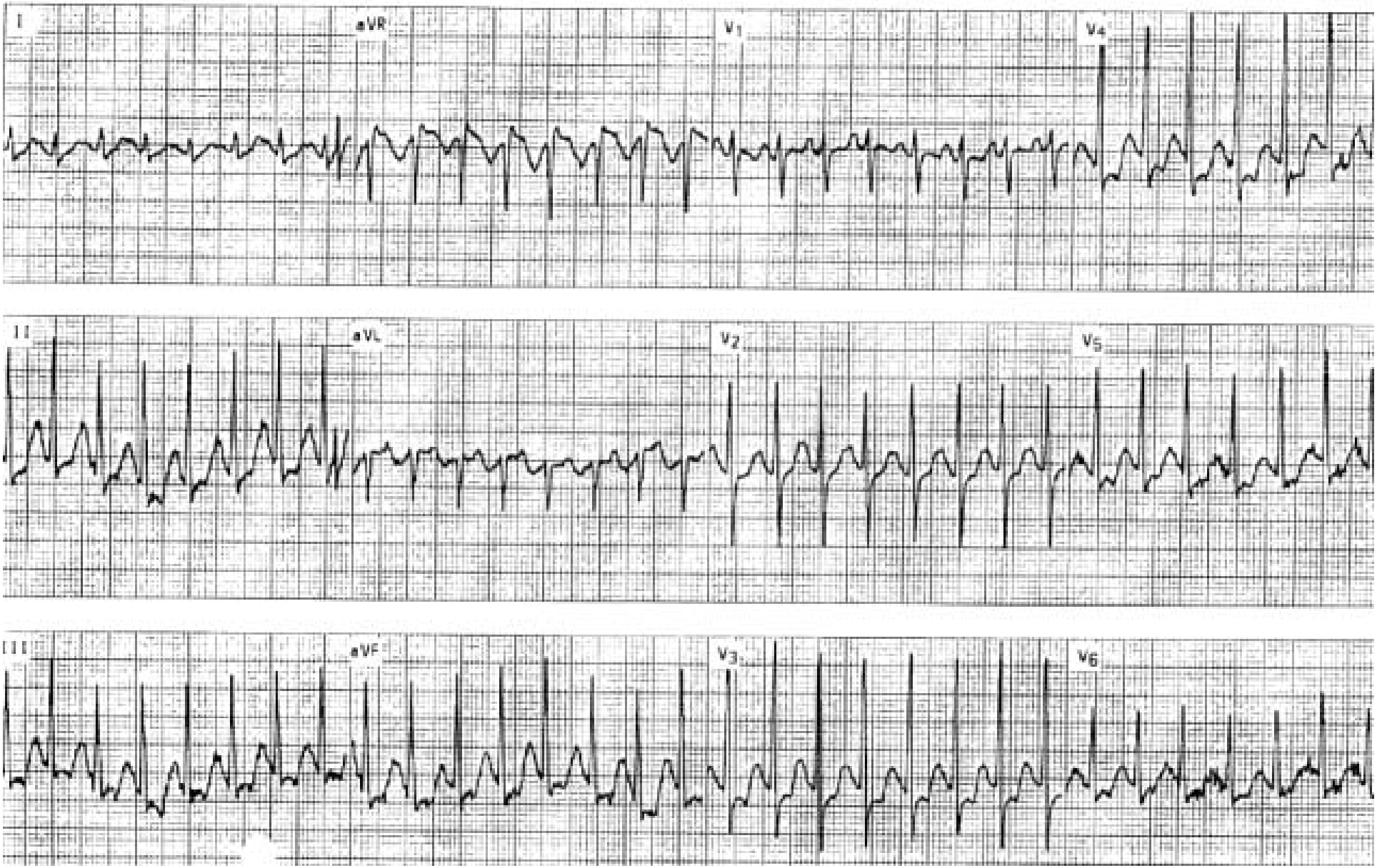
Note “non specific T-wave abnormalities” in II, III, AVF, V3 – V6

See anything else ? ? ?



Stress Test #1: Ischemia during max exercise

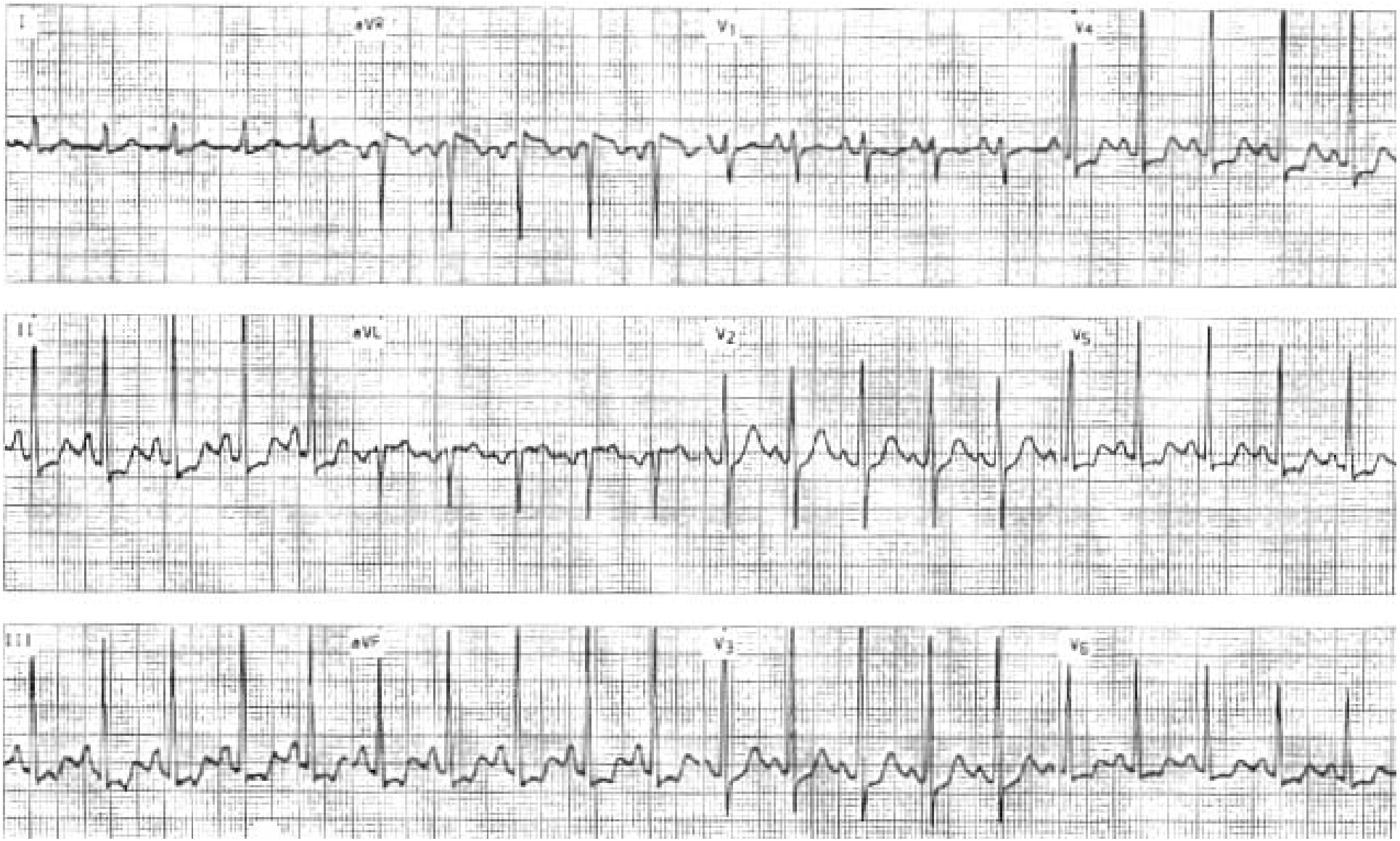
Note ST-segment depression in II, III, AVF, V3 – V6



Stress Test #1: Ischemia 9 minutes post-exercise

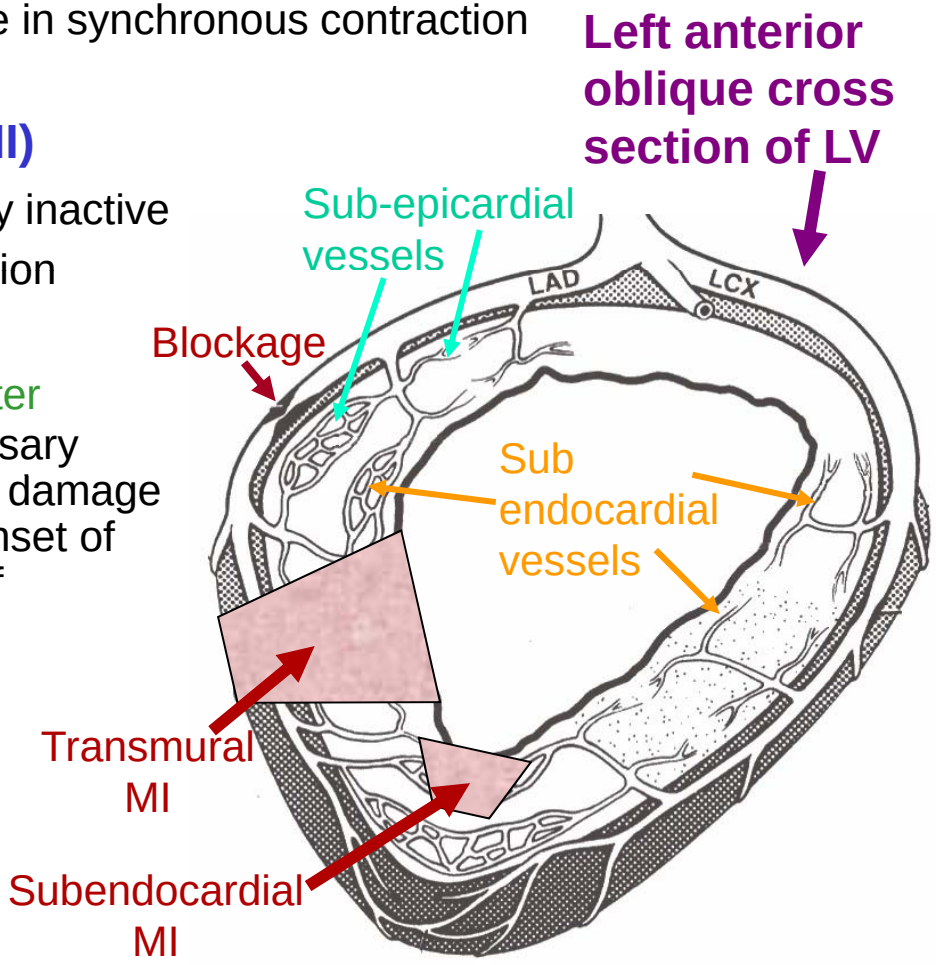
Note persistence of ST-segment depression in II, III, AVF, V3 – V6

Anything else noteworthy ? ? ?



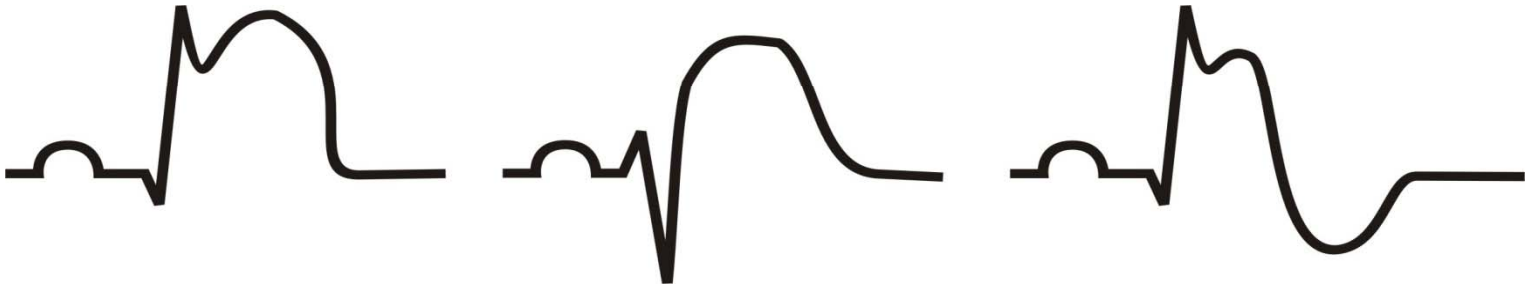
Myocardial Infarction

- **Myocardial Infarction Pathology:**
  - CA plaque is injured, ulcerates, or is ruptured
    - Platelet aggregation ➞ clot formation ➞ occlusion
  - Cells robbed of O2 begin relying on anaerobic glycolysis
  - After 10 seconds, fuel is depleted and cells become stunned
    - Stunned cells are unable to participate in synchronous contraction
    - Reversible if O2 supply is restored
  - If O2 is not restored ➞ **INFARCTION (MI)**
  - Cells die and become necrotic & electrically inactive
    - Electrically inactive cells ➞ ↓ wall motion
- **Emergency Treatment for infarction:**
  - Critical point: The earlier ER arrival the better
    - ER arrival within 1 to 2 hours is necessary for preventing / reducing myocardial damage
    - ER arrival within 6 to 12 hours after onset of symptoms ➞ re-profusion may be of benefit and limit infarct size
  - PCI (percutaneous coronary intervention)
    - (best results)
  - Fibrinolytic therapy (Streptokinase, TPA)
  - Fibrinolytic therapy + PCI
  - CABG
  - Drugs: aspirin, β blockers, nitro, morphine, benzodiazapines, ACE inhibitors, antiarrhythmics, platelet inhibitors

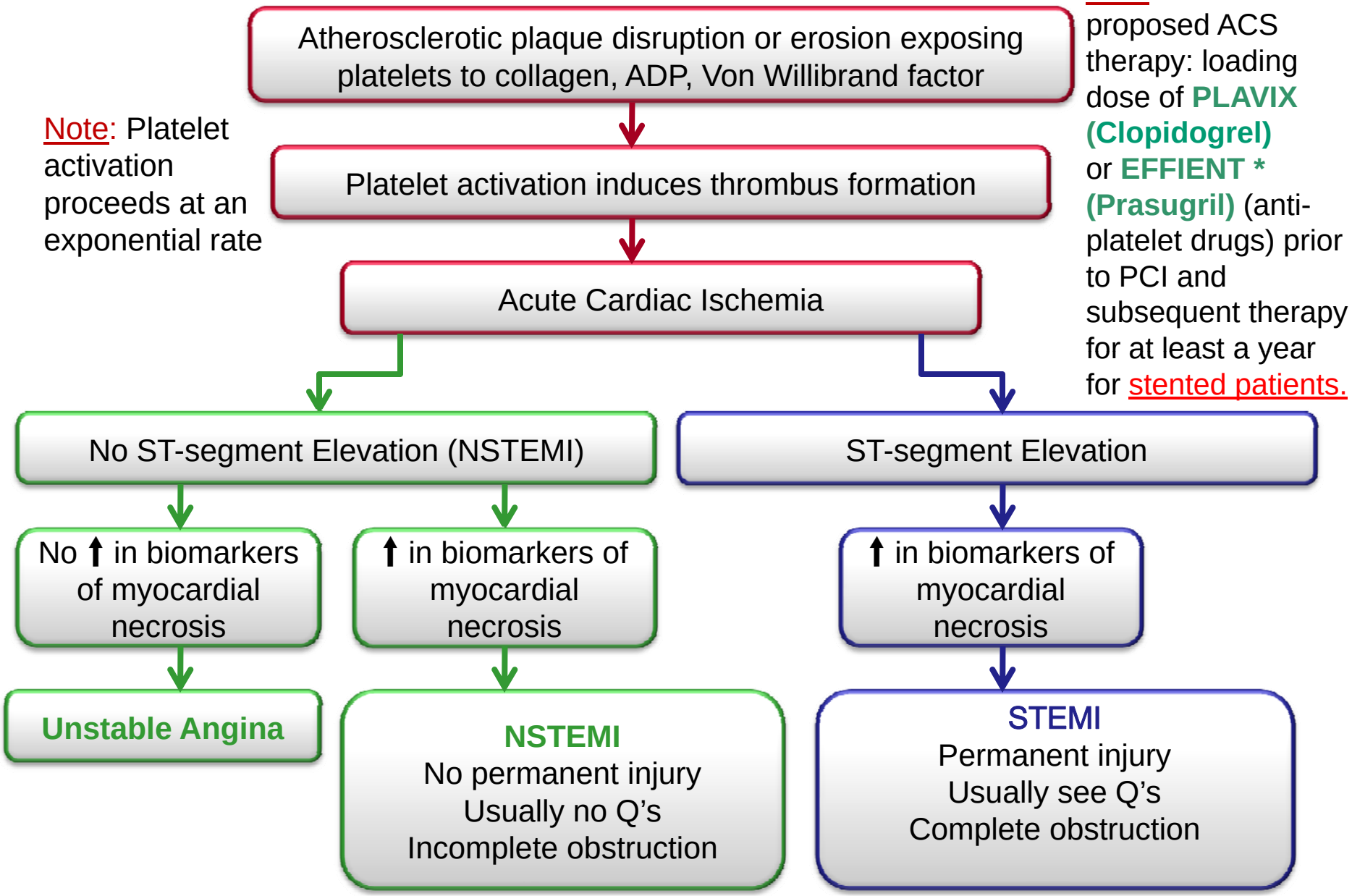


Infarction

- **The development of an acute STEMI infarction**
  1. **Usually no ECG changes are seen in the first few minutes after occlusion**
  2. **Appearance of tall narrow T-waves or ST-segment elevation**
     - 5 to 30 minutes post occlusion
  3. **A few hours later, the T-waves invert (ischemia)**
     - In an MI, the T-wave inversion is symmetrical an may persist for years
     - Inverted T-waves without other indications are not diagnostic of an MI
  4. **ST-segment elevation (STEMI) – indication of transmural ischemia and or injury**
     - Usually the first and most common sign of an infarction
     - May or may not be accompanied by T-wave inversion
     - The larger the ischemic area, the greater the ST displacement
     - ST elevation persisting for more than a few hours may indicate ventricular aneurysm
     - **ST depression may be seen in reciprocal leads during an MI which may indicate:**
       - **Nothing**
       - **Stenosis and ischemia in other areas of the heart, especially the LAD**
       - **Multi-vessel disease**



" Acute Coronary Syndrome " or "ACS"

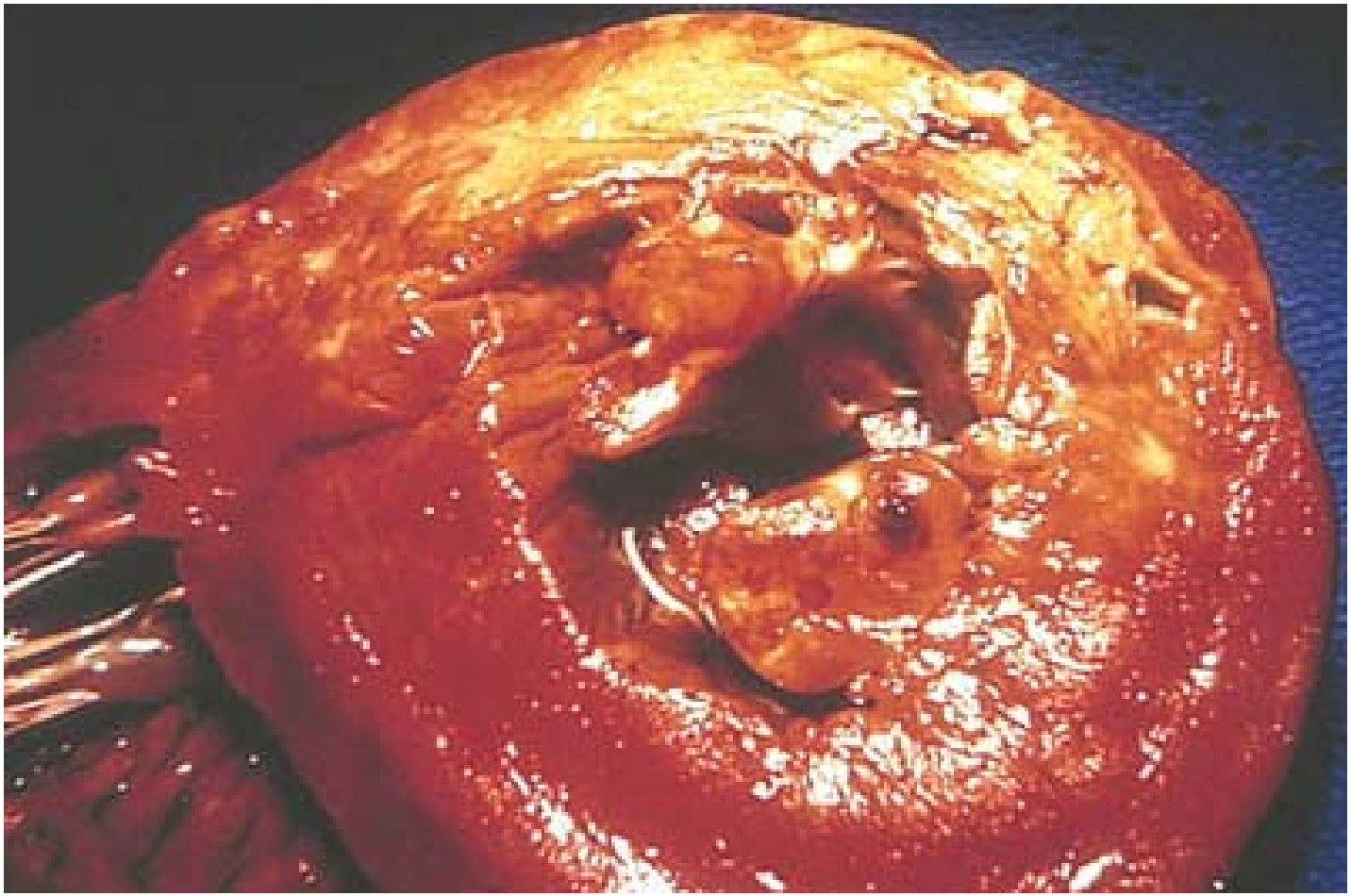


Note: Platelet activation proceeds at an exponential rate

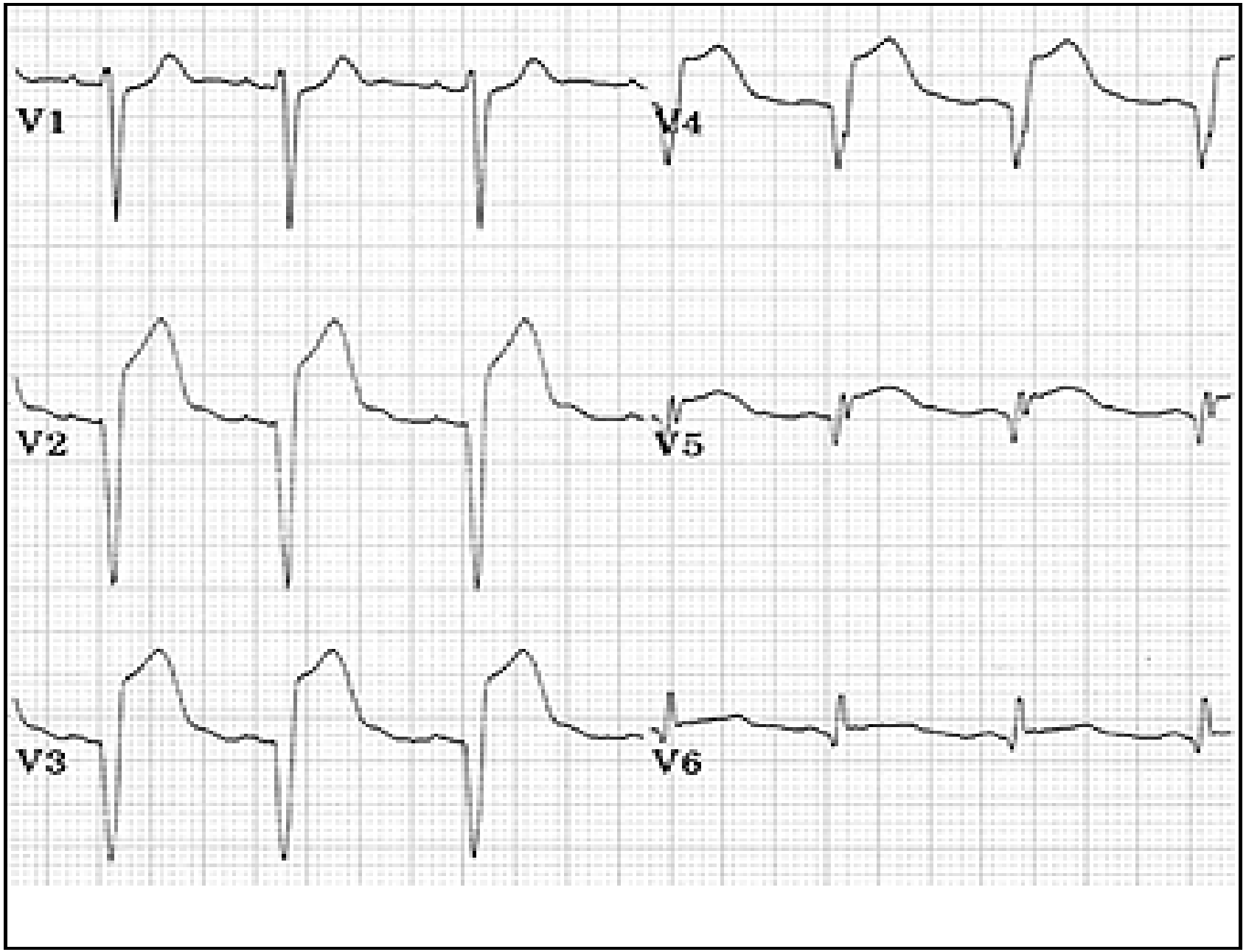
Note: current proposed ACS therapy: loading dose of **PLAVIX (Clopidogrel)** or **EFFIENT * (Prasugril)** (anti-platelet drugs) prior to PCI and subsequent therapy for at least a year for stented patients.

Old Anterior Transmural MI: Left Ventricular Cross Section

Note white necrotic / fibrotic myocardium

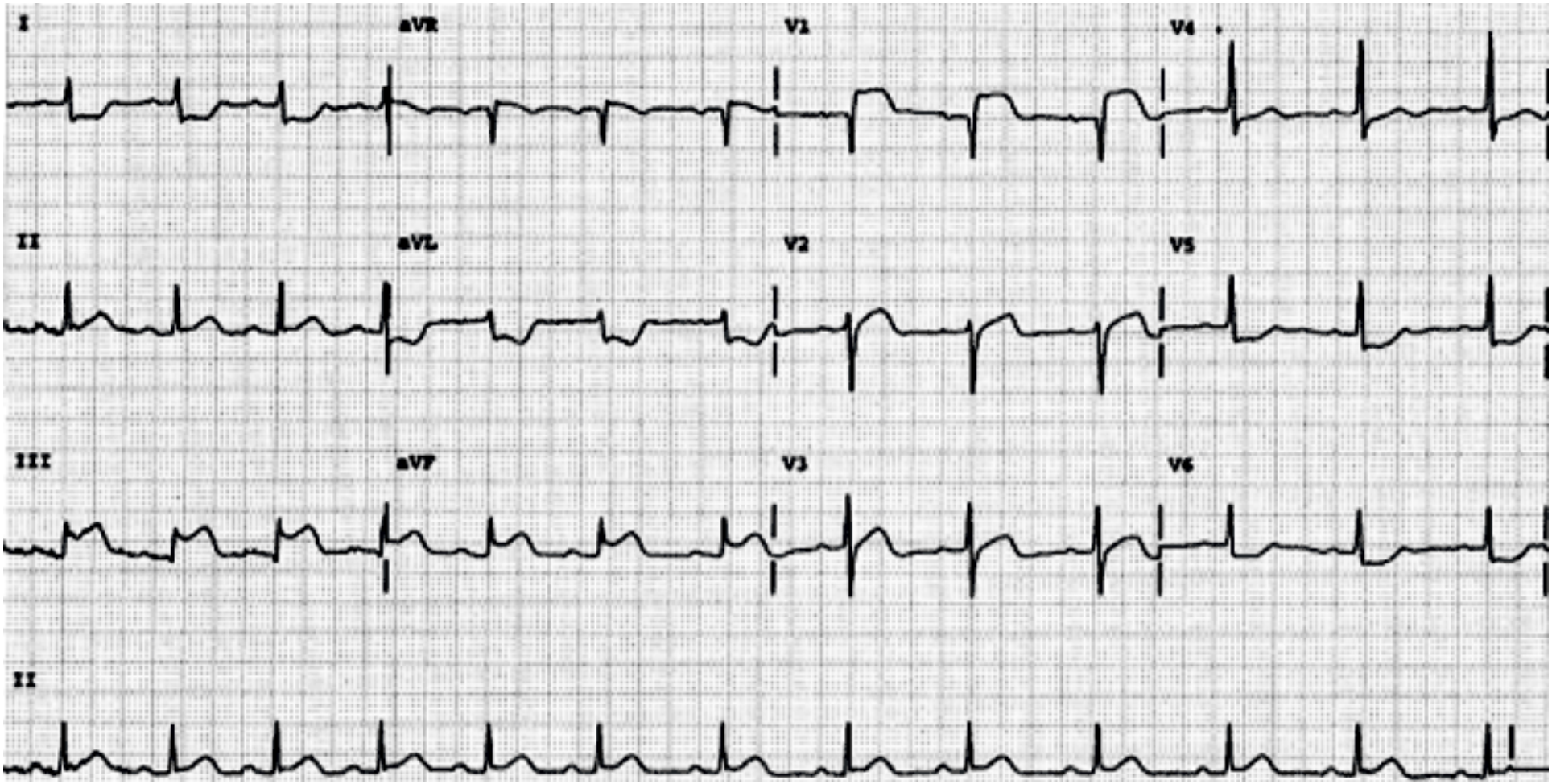


Acute Anterio-Lateral Myocardial Infarction



Acute Inferior Myocardial Infarction

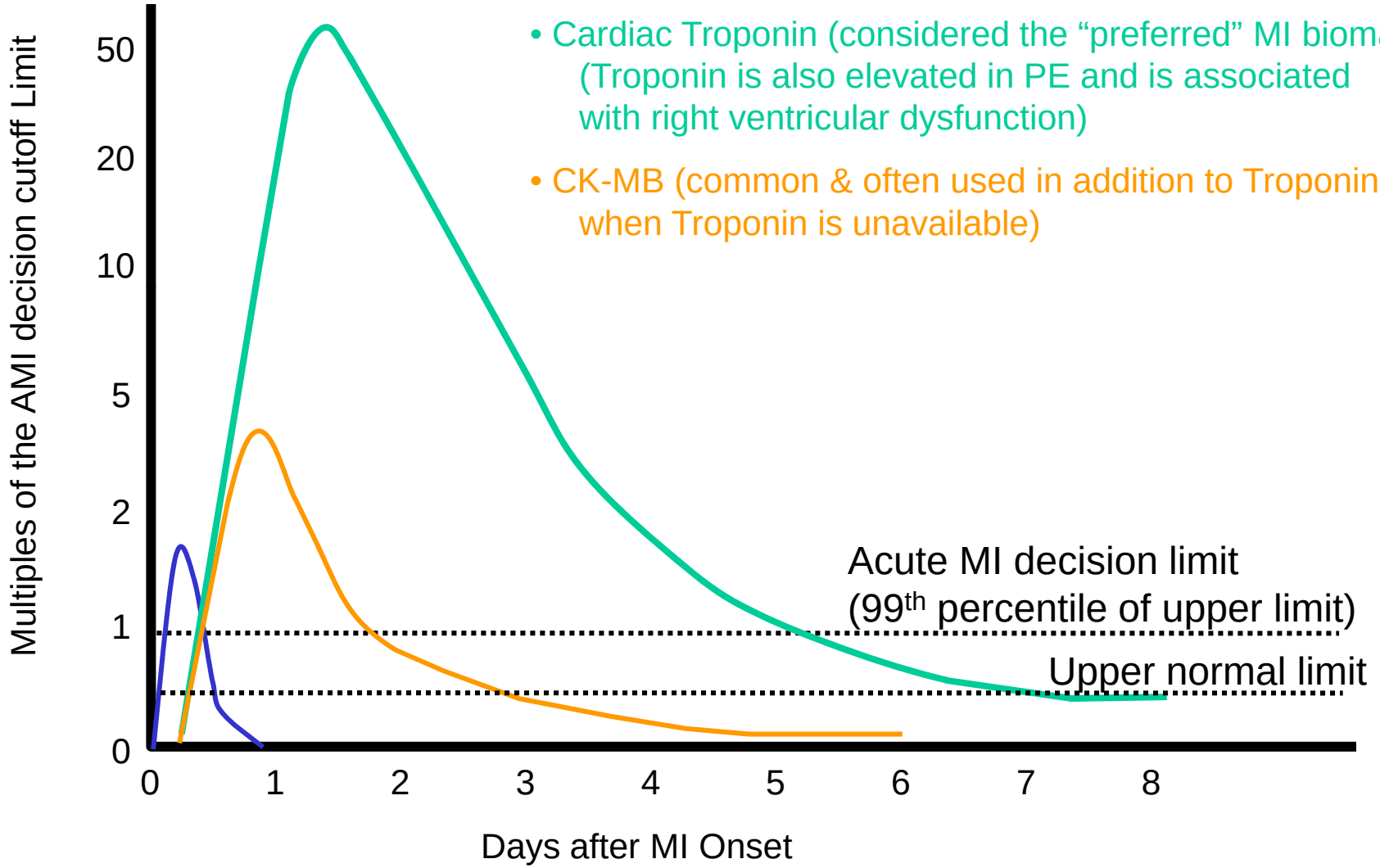
Note ST segment elevation in III and AVF (Also V1)
Reciprocal ST segment depression in I, AVL, V5, V6



Ischemia & Infarction

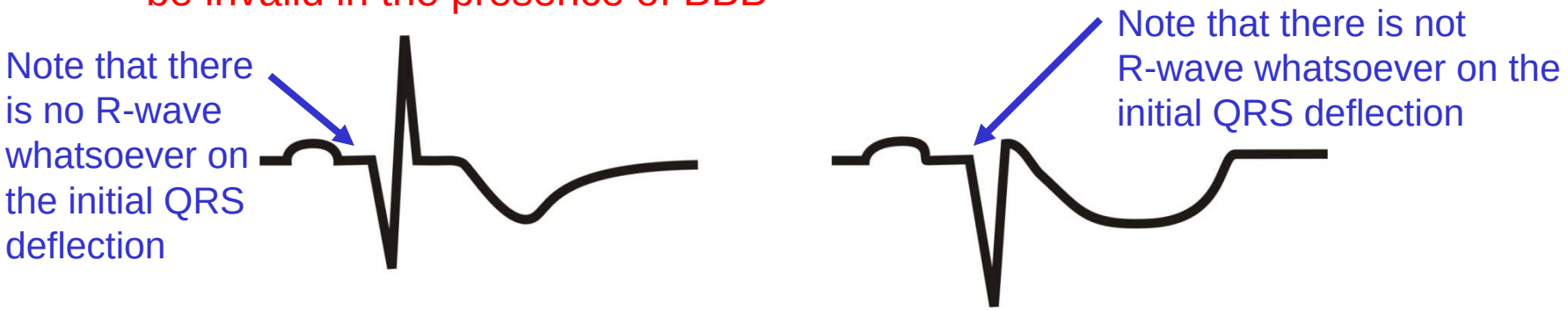
Biomarkers in an MI:

- Myoglobin
- Cardiac Troponin (considered the “preferred” MI biomarker) (Troponin is also elevated in PE and is associated with right ventricular dysfunction)
- CK-MB (common & often used in addition to Troponin or when Troponin is unavailable)



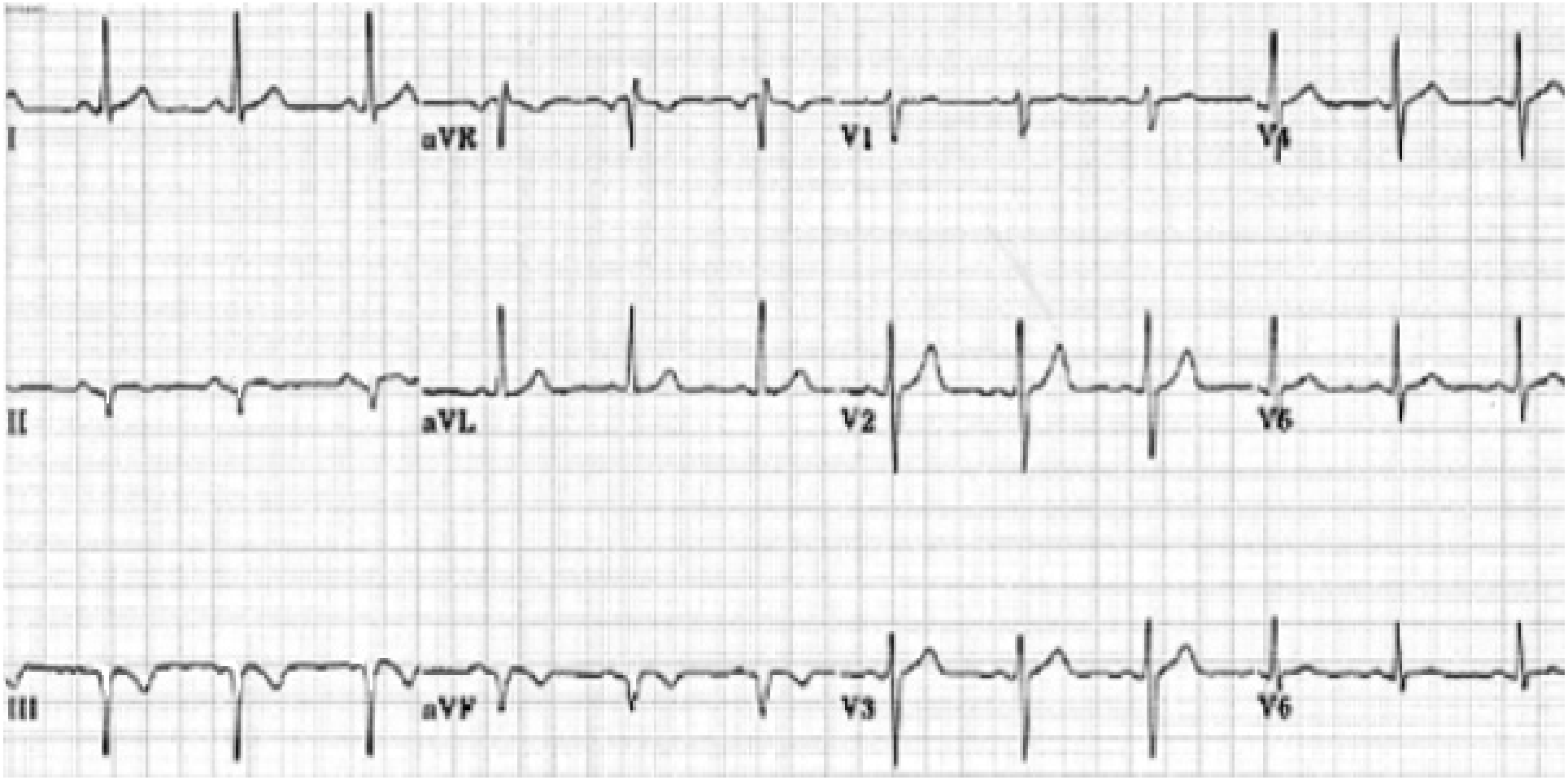
Ischemia & Infarction

- **Indications of an old or "resolved" infarction**
  - **Significant Q-waves where they don't belong:**
    - Nonsignificant Q's may be found in V1 alone, III alone, and AVR
  - These Q's represent necrotic tissue ➞ main vector directed away from that lead
    - In general (not true all the time), for Q's to be significant:
      - They must be at least .03 - .04 seconds wide
      - They must be at least 1/3 of the height of the R-wave in the QRS
      - They must be "new" (not seen in a previous tracing)
      - In most cases, they must be in more than one lead
  - **Speculating as to the location of the infarct:**
    - Significant Q's in II, III, AVF – old inferior infarction
    - Significant Q's in I, V1, V2, V3 – old anterior infarction
    - Significant Q's in V4, V5, V6 – old lateral infarction
  - Infarct criteria (as well as all other voltage related diagnoses) may be invalid in the presence of BBB



Ischemia & Infarction

Old Inferior MI – Significant Q-waves in III & AVF



Old Anterio-Lateral MI