



ECG made easy

Presented by:

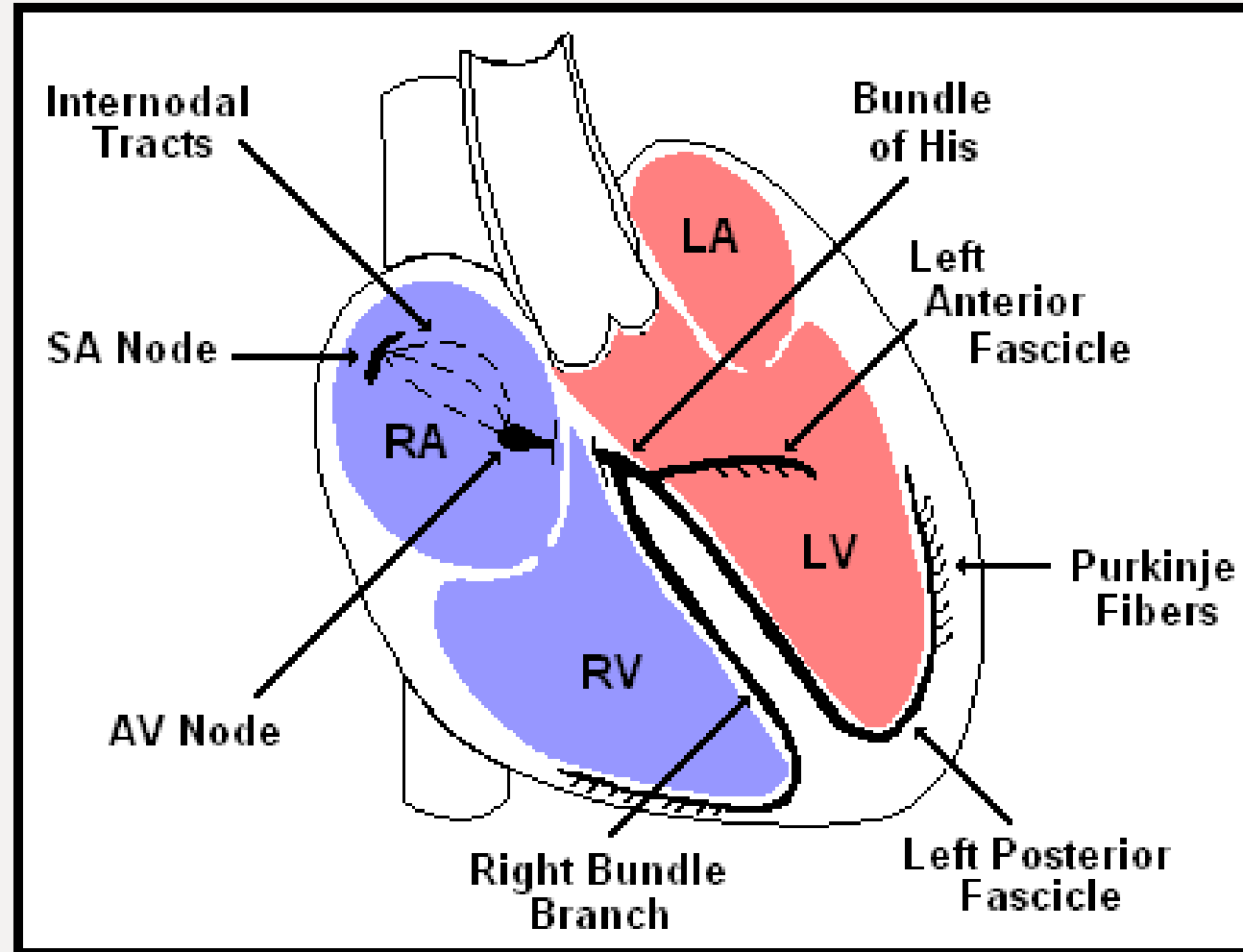
Dr Randall Hendriks, Interventional Cardiologist – Western Australia



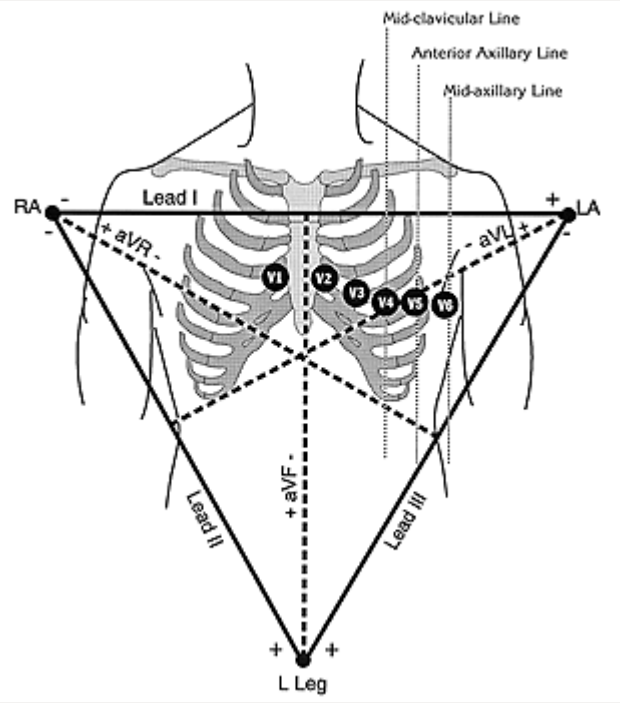
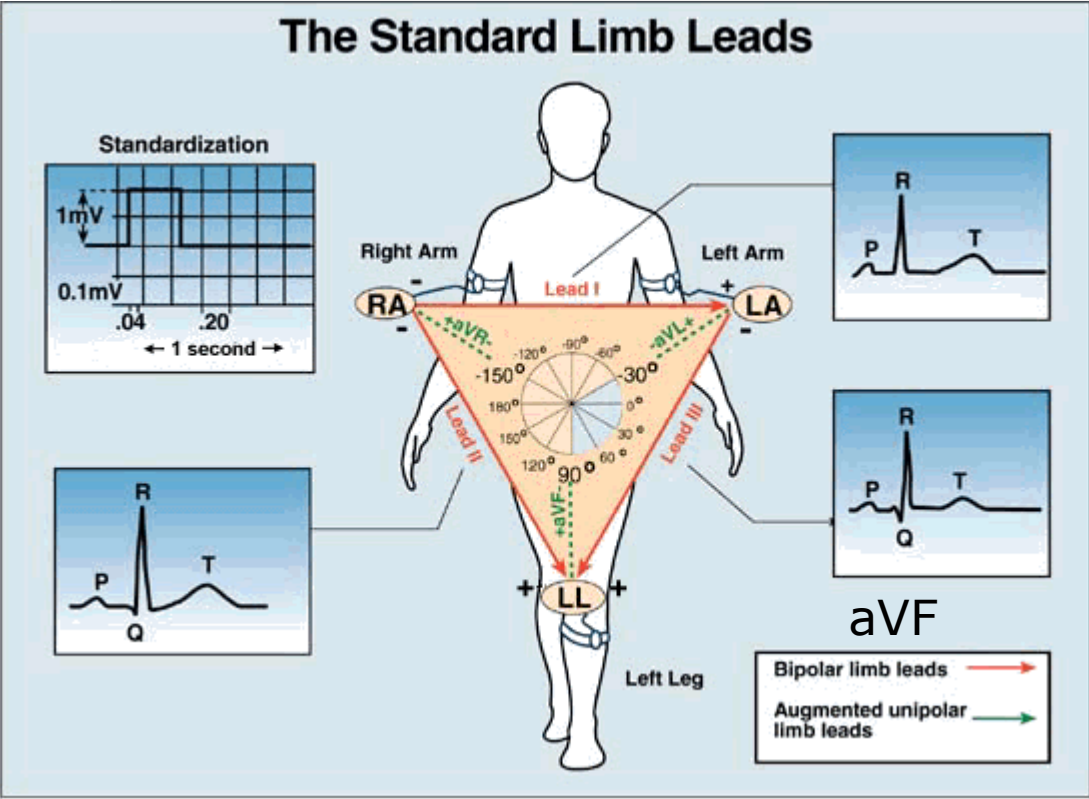
Reading an ECG

- The ECG does not have to be intimidating
- Establish a consistent approach to interpreting ECGs
- Do not rely on machine reads
- Interpret the ECG in the context of the clinical history

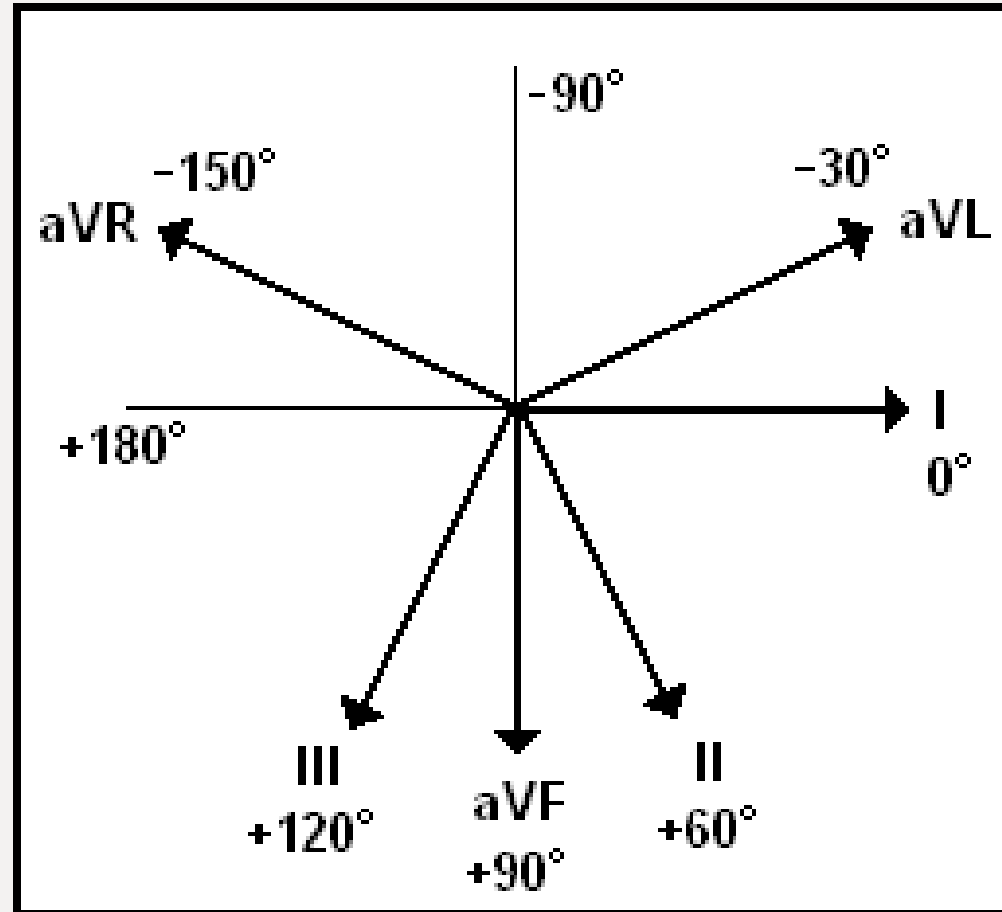
The Normal Conduction System



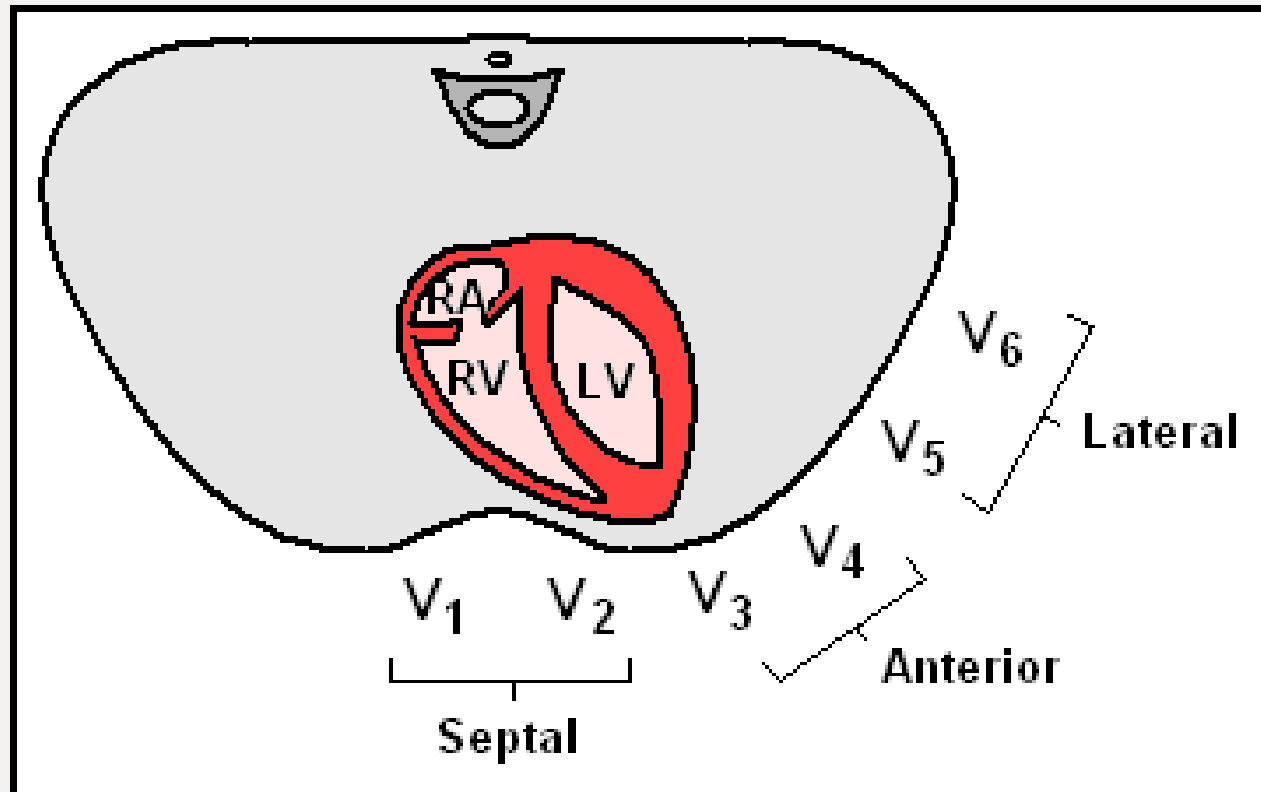
Lead Placement



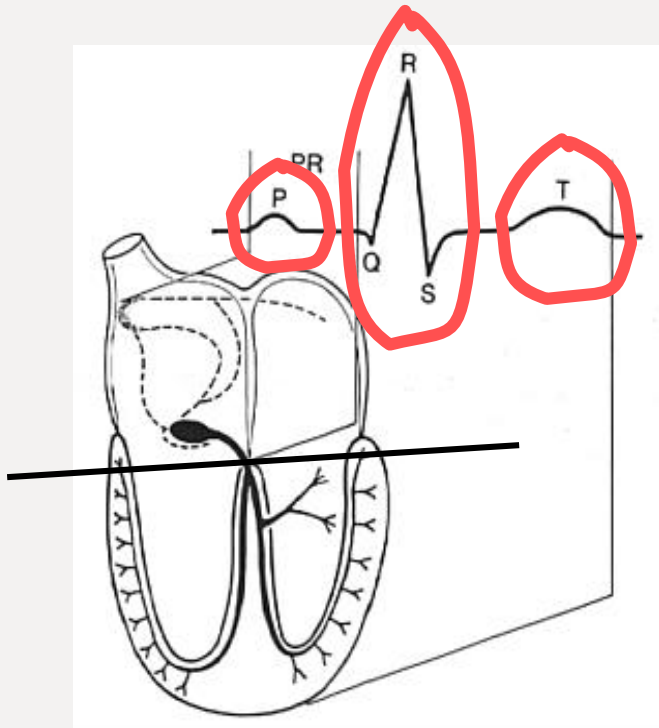
All Limb Leads



Precordial Leads



Components of a normal ECG



- **P wave** - atrial depolarisation
- **PR interval** - AV node + His-P
- **QRS** - ventricular depolarisation
- **T wave** - ventricular repolarisation



ECG interpretation

- **Rate**

- Rhythm
- Axis
- P wave
- Intervals
 - PR interval
 - QRS duration
 - QT interval
- Q waves
- R wave transition
- ST segments
- T waves (and others)

Rate

- Rule of 300 - divide 300 by the number of boxes between each QRS = rate



- Count QRS in 10 second rhythm strip x 6





Rate

- HR of 60-100 per minute is normal
- HR > 100 = tachycardia
- HR < 60 = bradycardia

Single Lead ECG: Provides

Heart rate: normal 60 – 100

Remember:

Pulse rate may not
equal heart rate



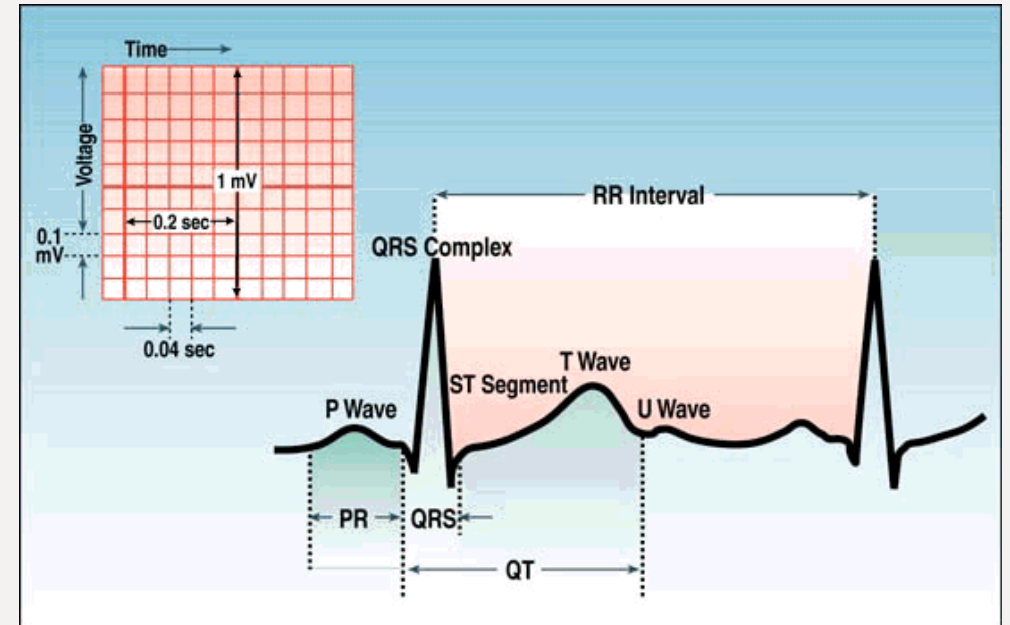


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Rhythm

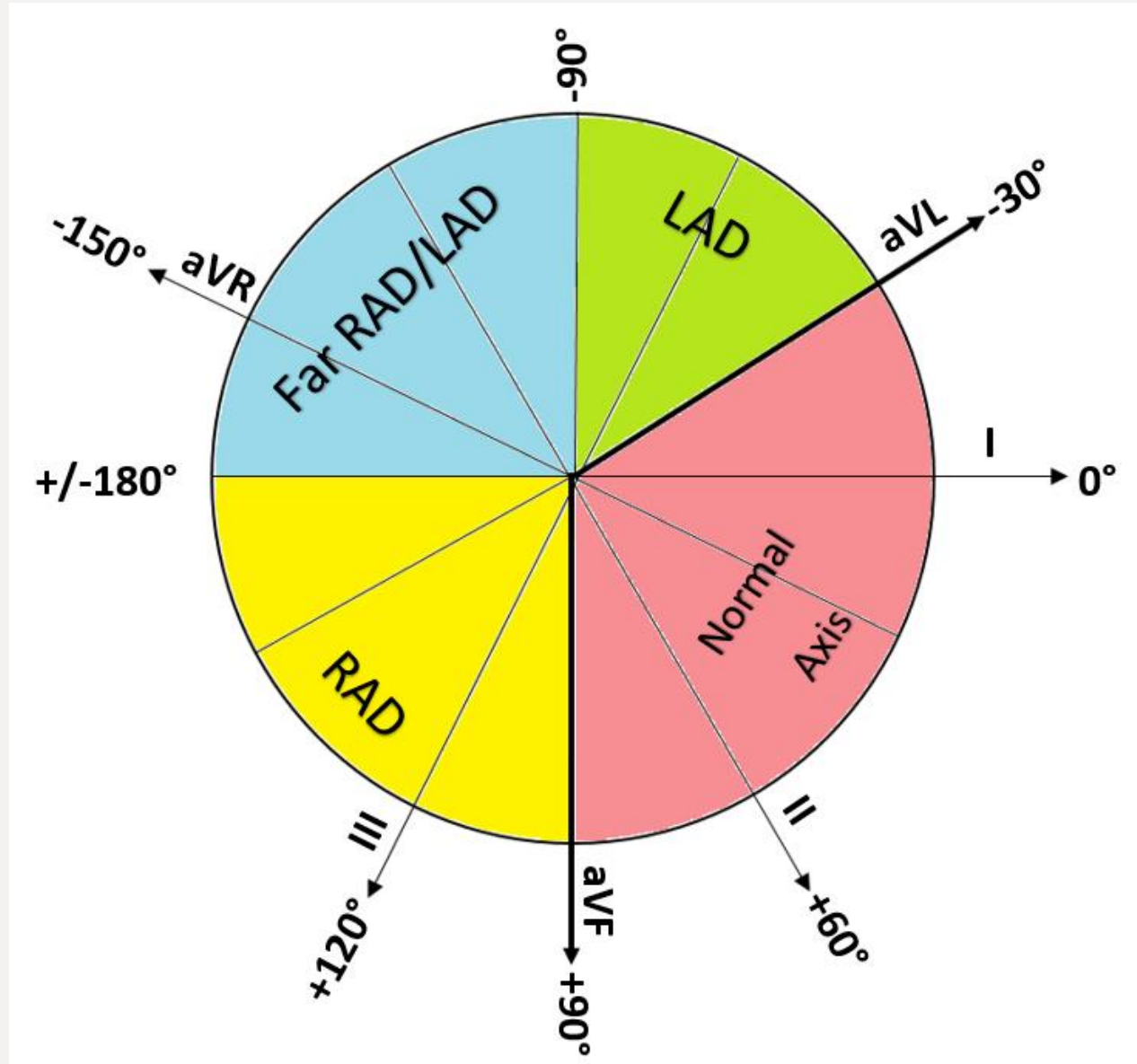
- Sinus
 - Originating from SA node
 - P wave before every QRS
 - P wave in same direction as QRS



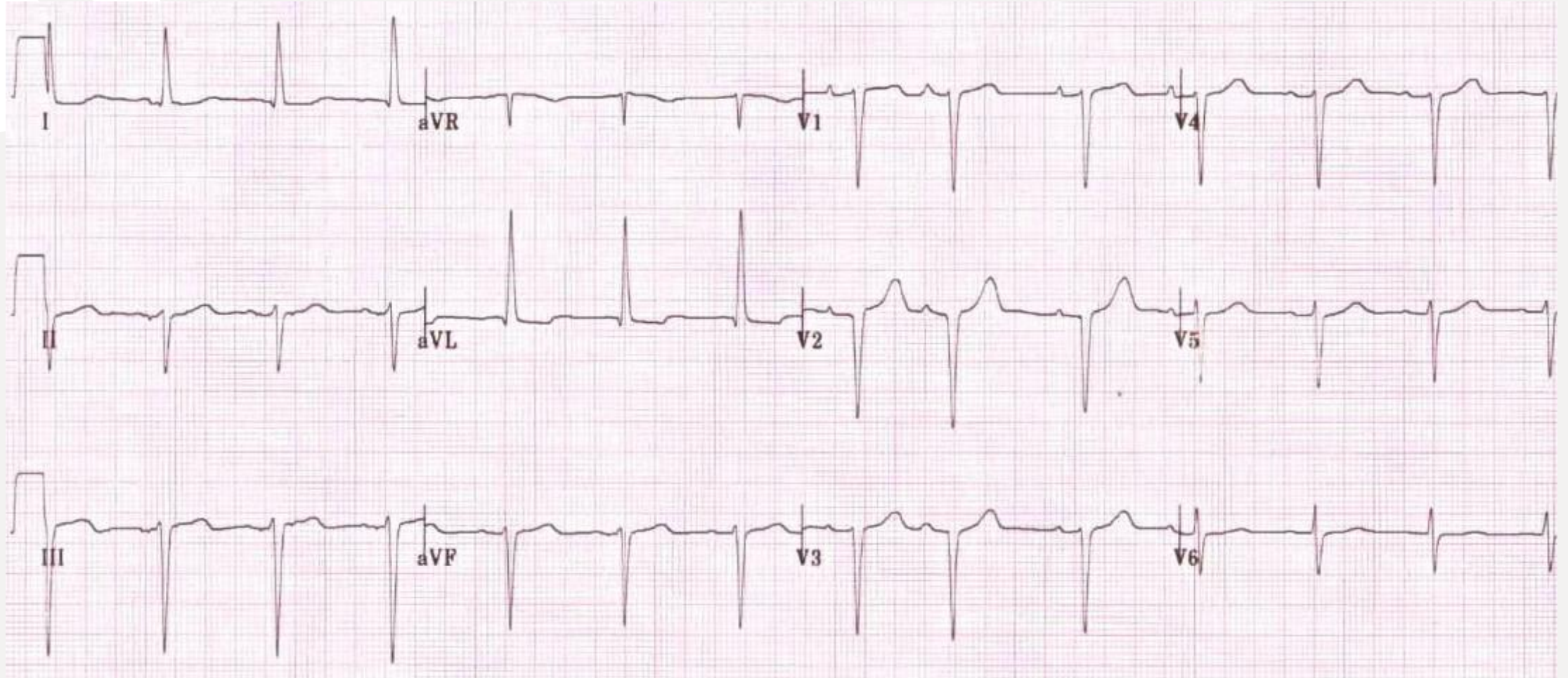


ECG interpretation

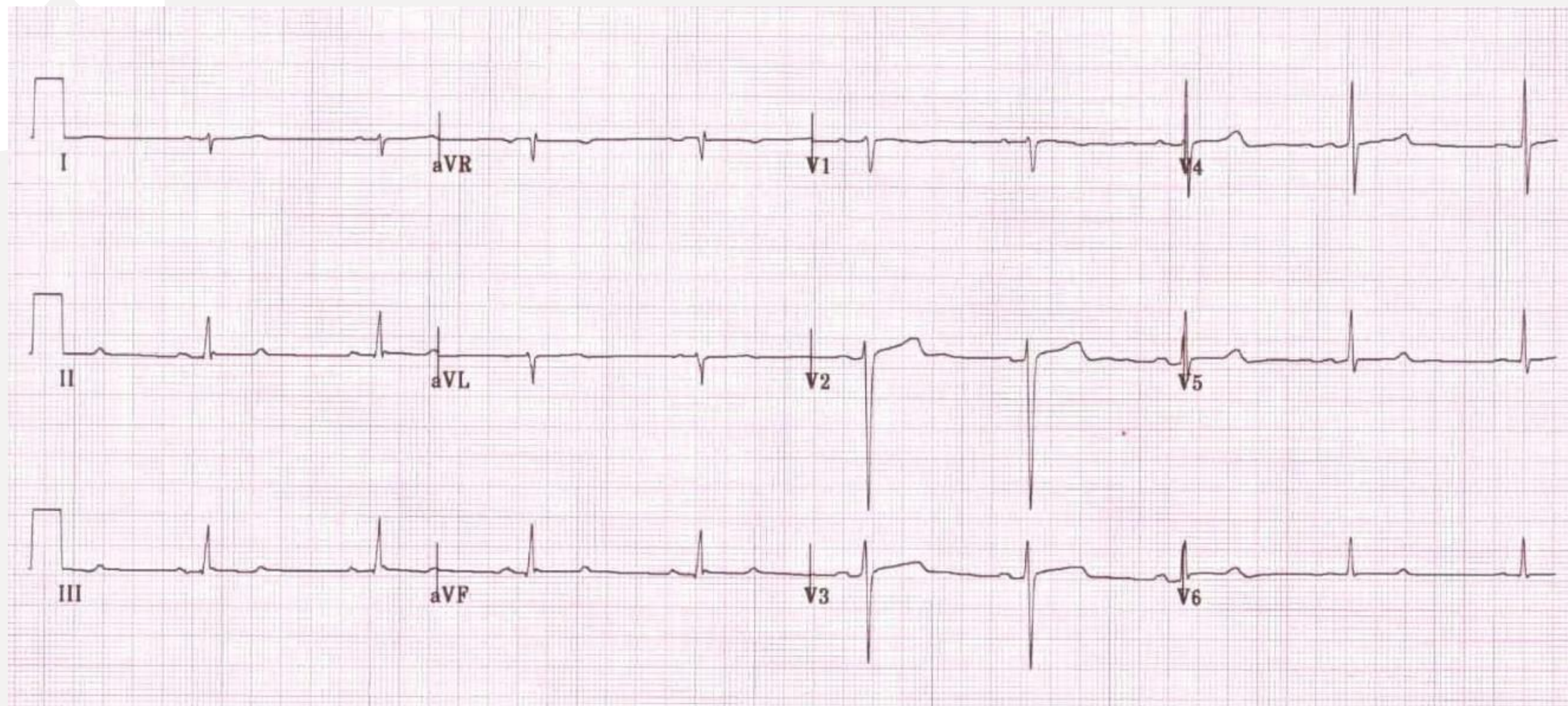
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Left axis deviation: check lead II



Right axis deviation: check lead I

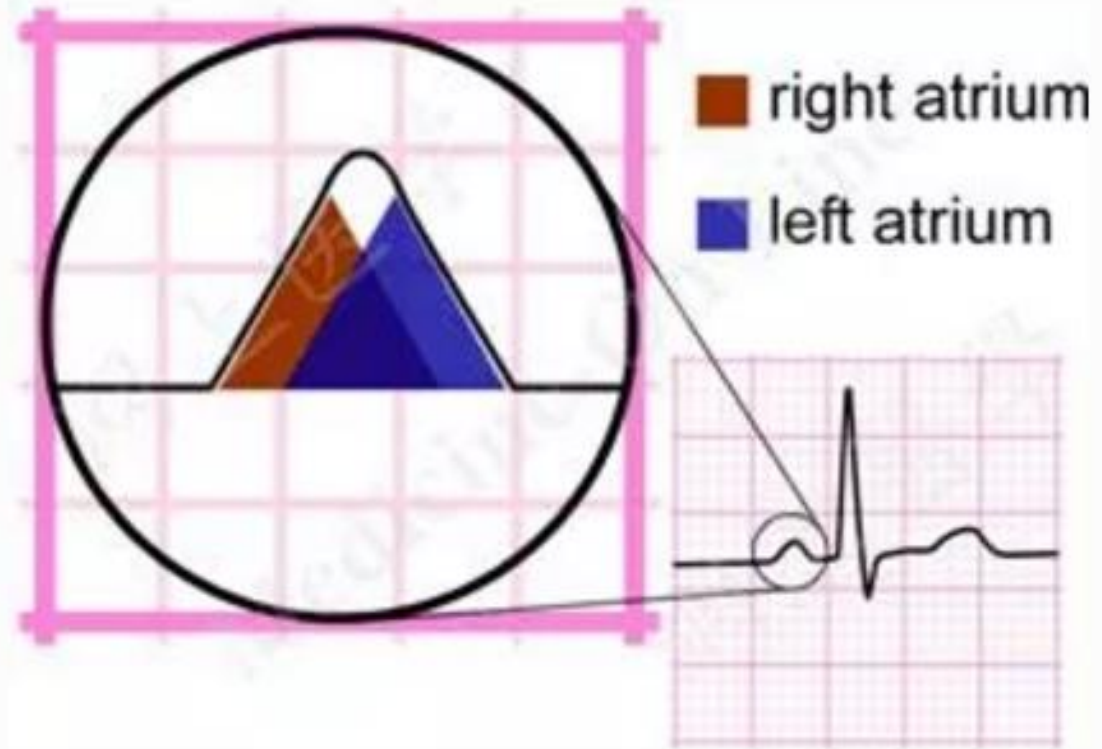




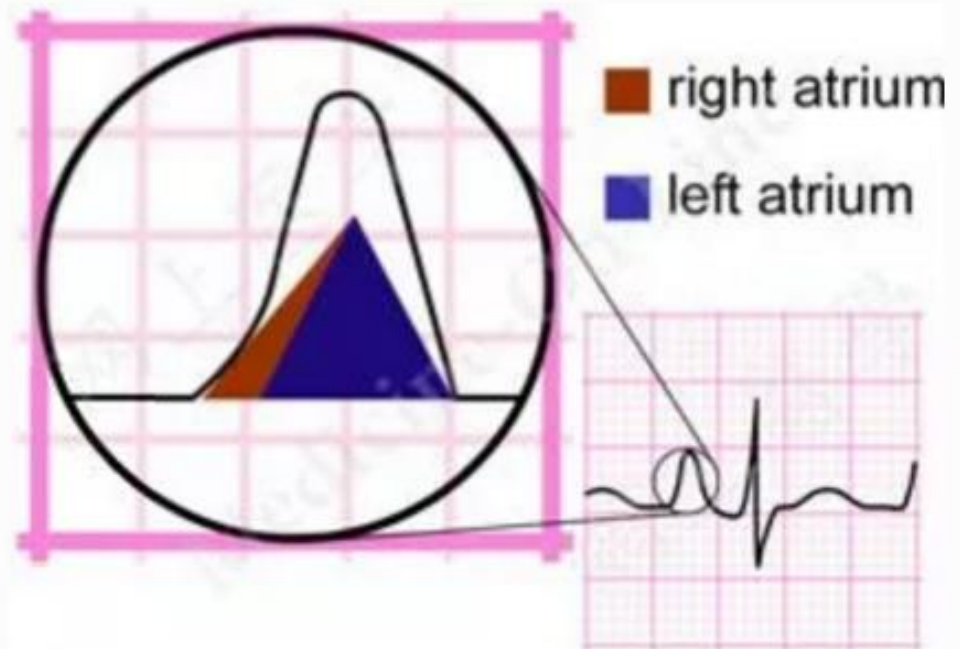
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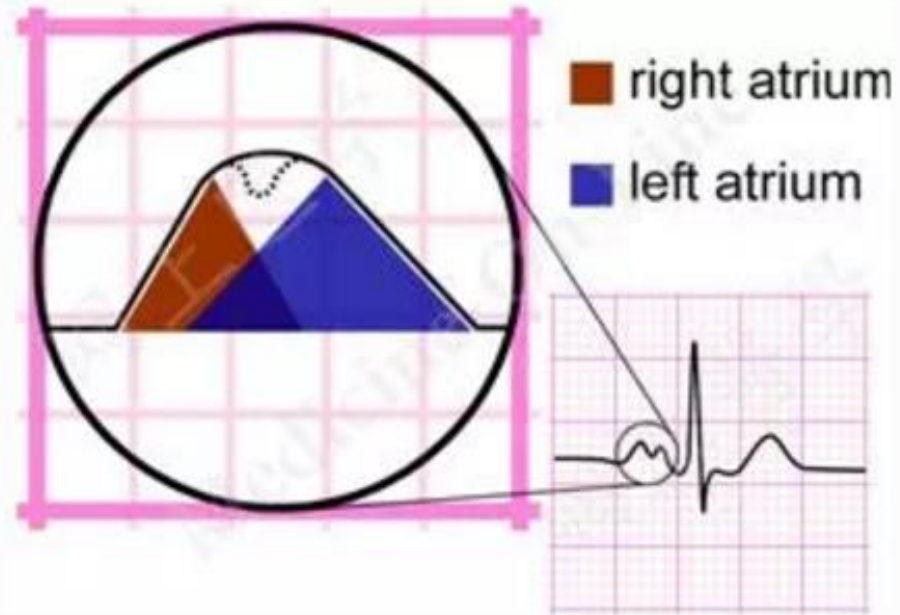
P wave











RA enlargement



LA enlargement



Bi-atrial enlargement

	II	VI
Normal		
RAE		
LAE		
RAE + LAE		

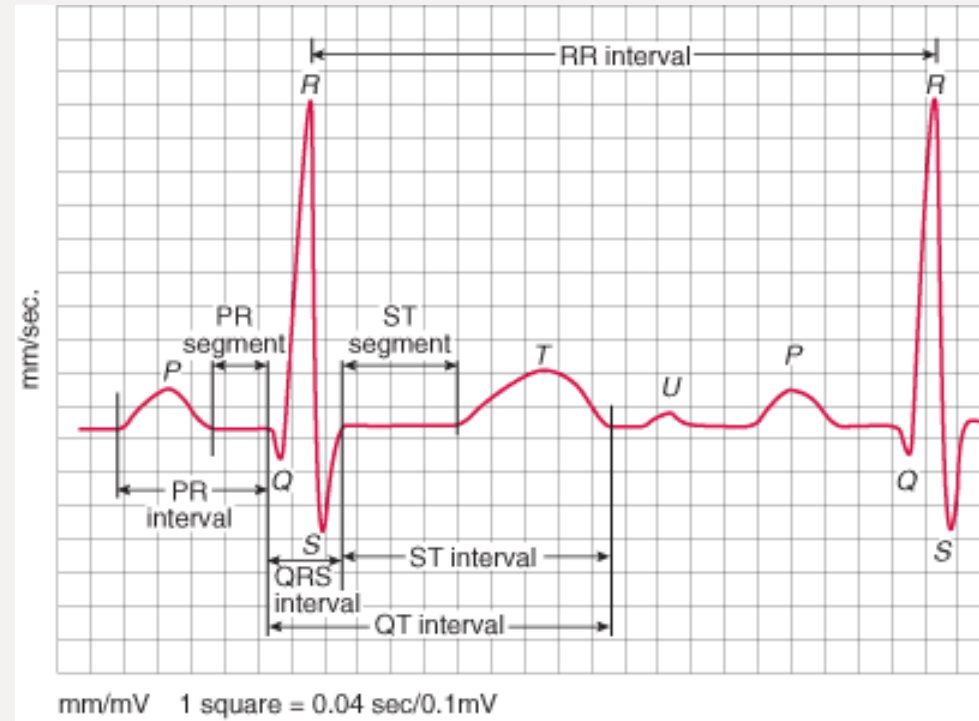


ECG interpretation

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Normal Intervals

- PR
 - 0.20 sec (less than one large box)

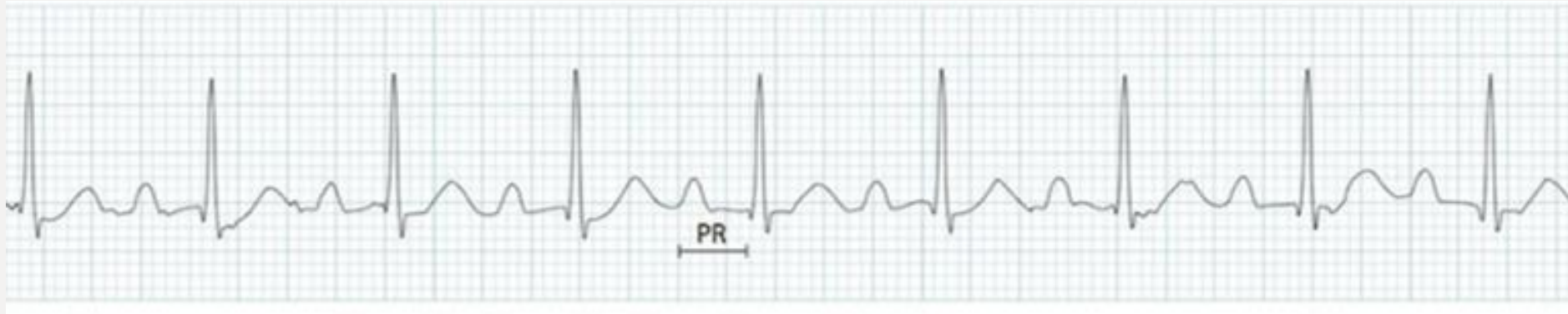




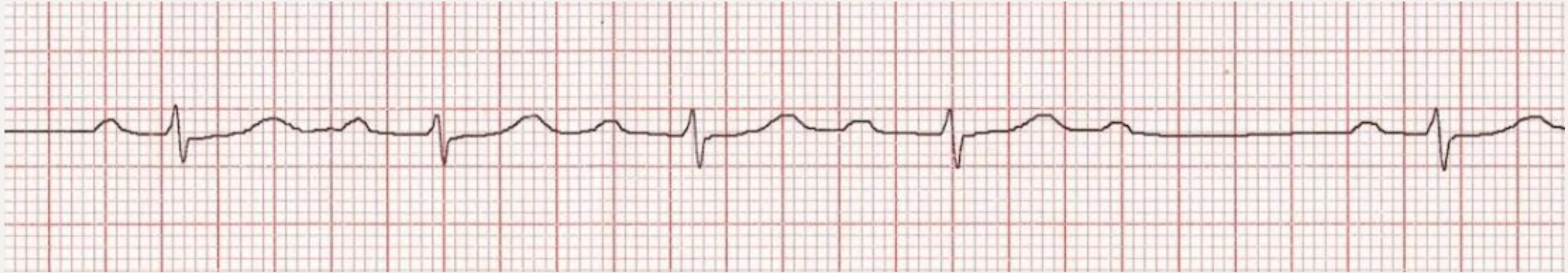
Blocks

- AV blocks
 - First degree block
 - PR interval fixed and > 0.2 sec
 - Second degree block, Mobitz type 1
 - PR gradually lengthened, then drop QRS
 - Second degree block, Mobitz type 2
 - PR fixed, but drop QRS randomly
 - Type 3 block
 - PR and QRS dissociated

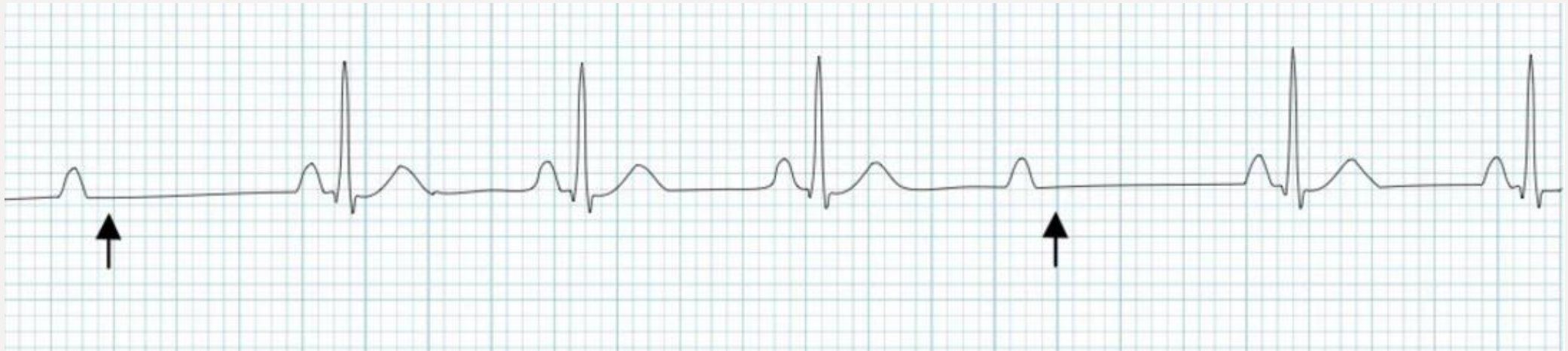
First Degree Heart Block



2nd degree, Mobitz I (Wenckebach phenomenon)



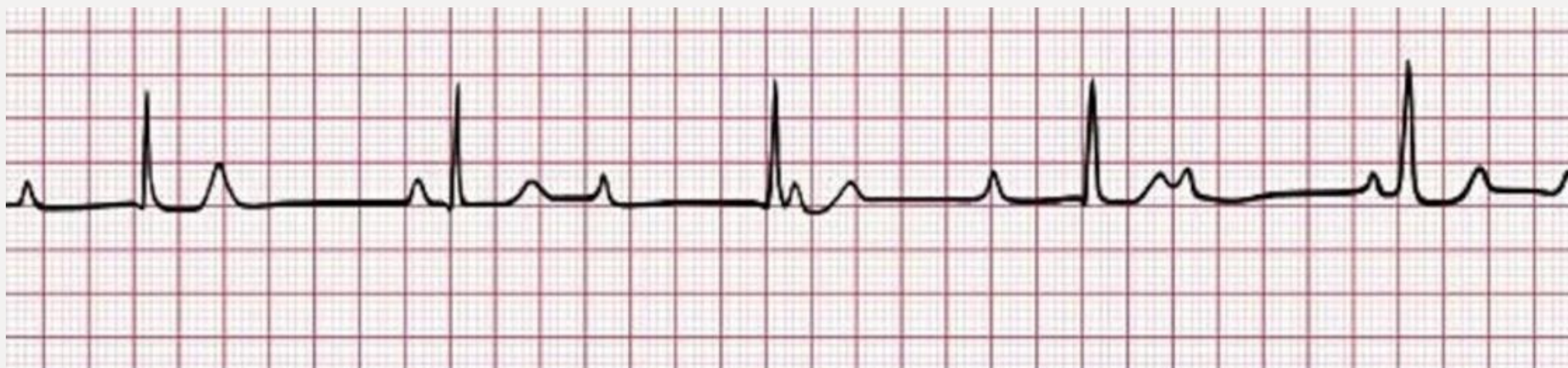
2nd degree, Mobitz II



2nd degree, "high-grade AV block"

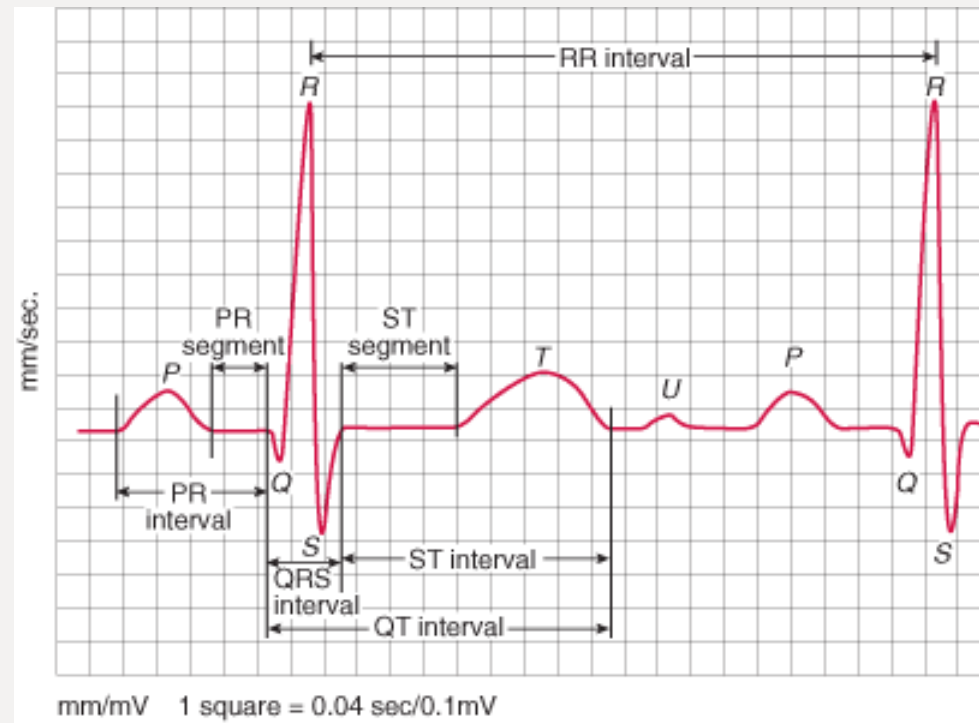


3rd degree (complete heart block)

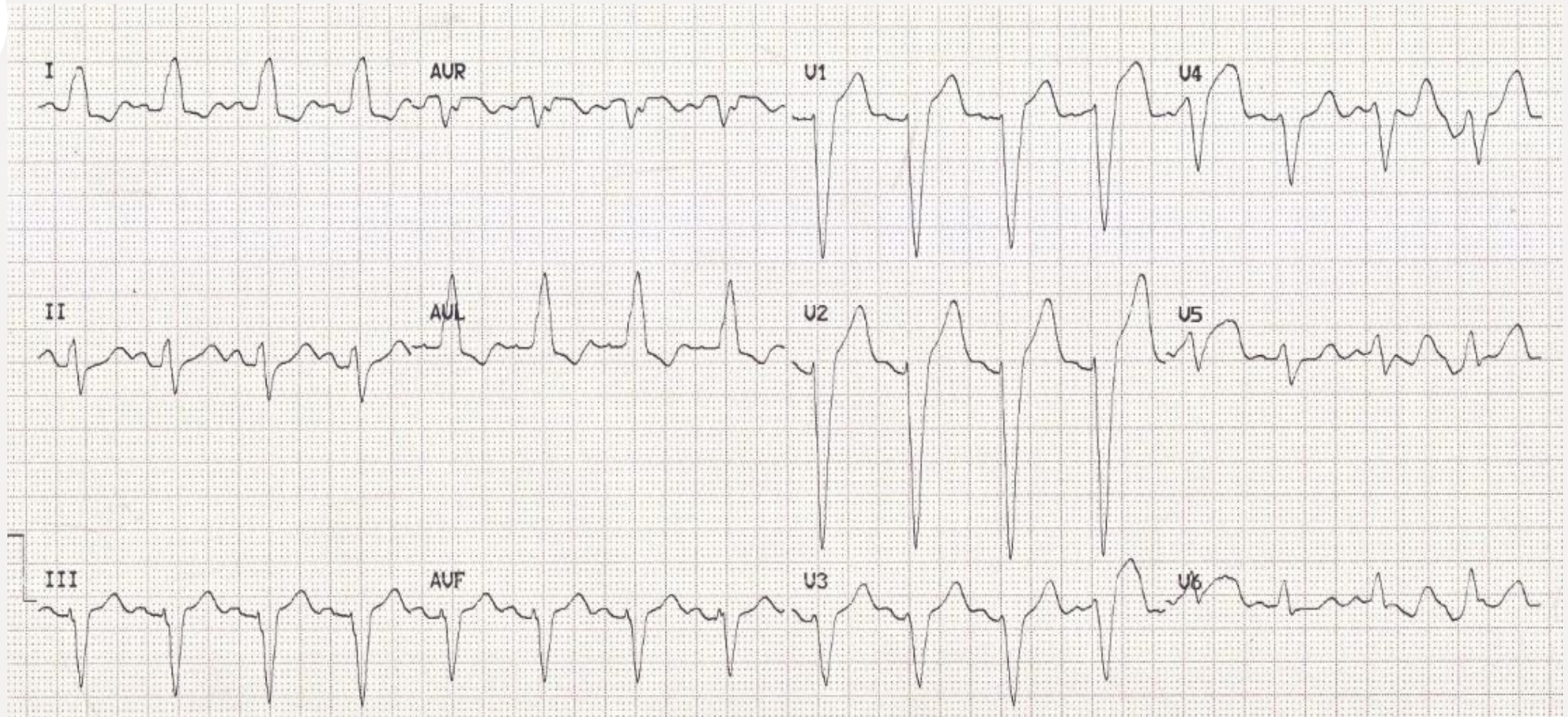


Normal Intervals

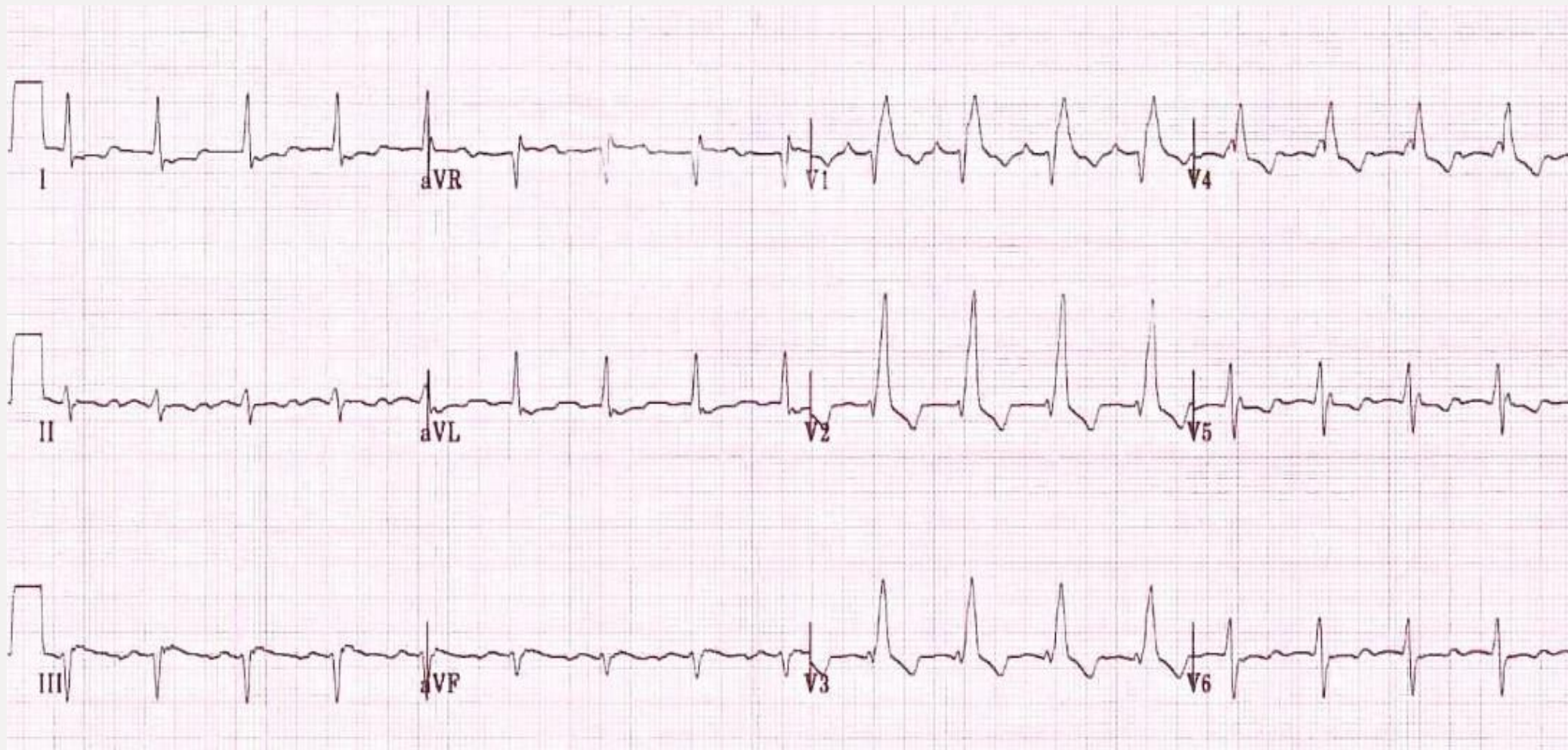
- PR
 - 0.20 sec (less than one large box)
- QRS
 - 0.08 – 0.10 sec (1-2 small boxes)



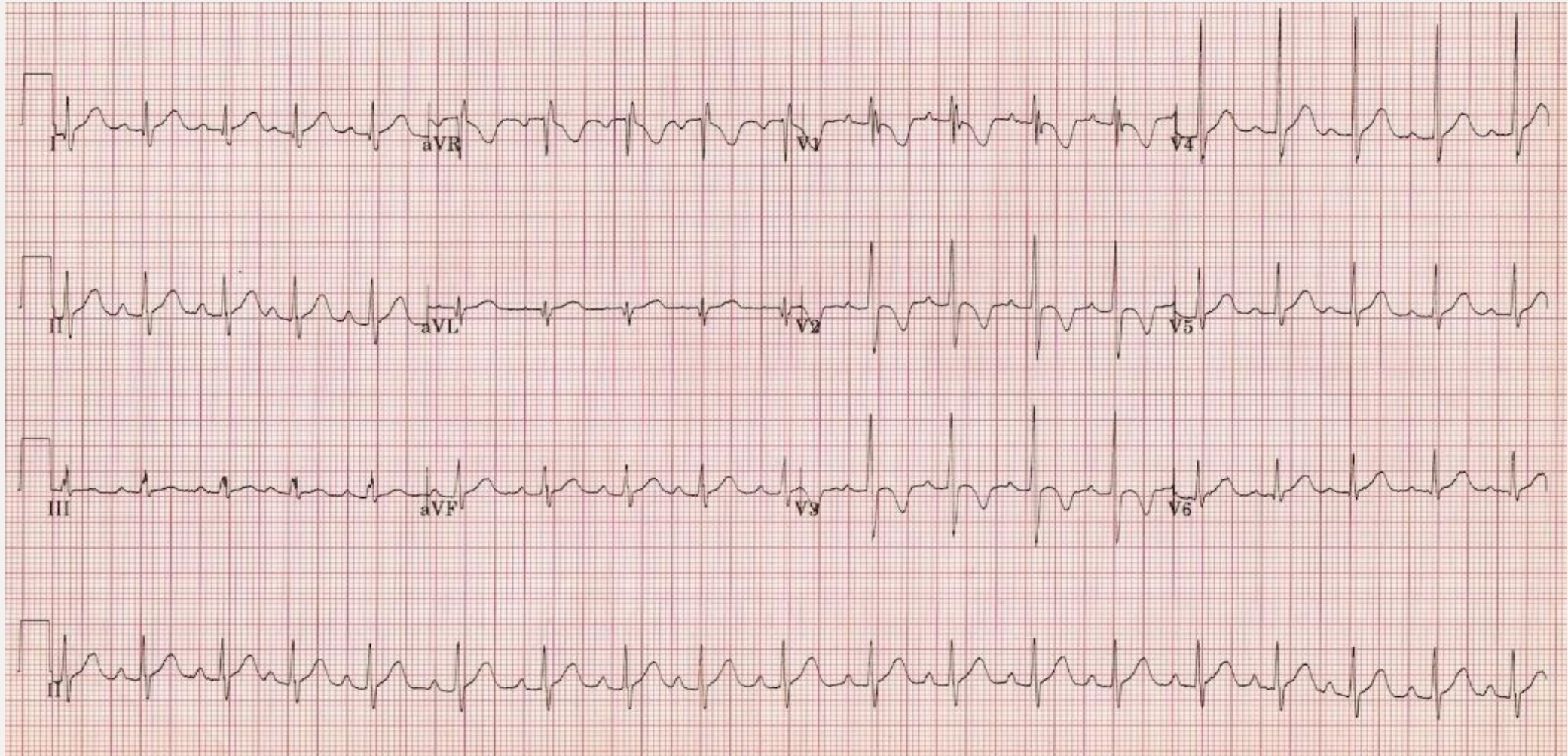
LBBB: QRS >120ms



RBBB: QRS >120ms

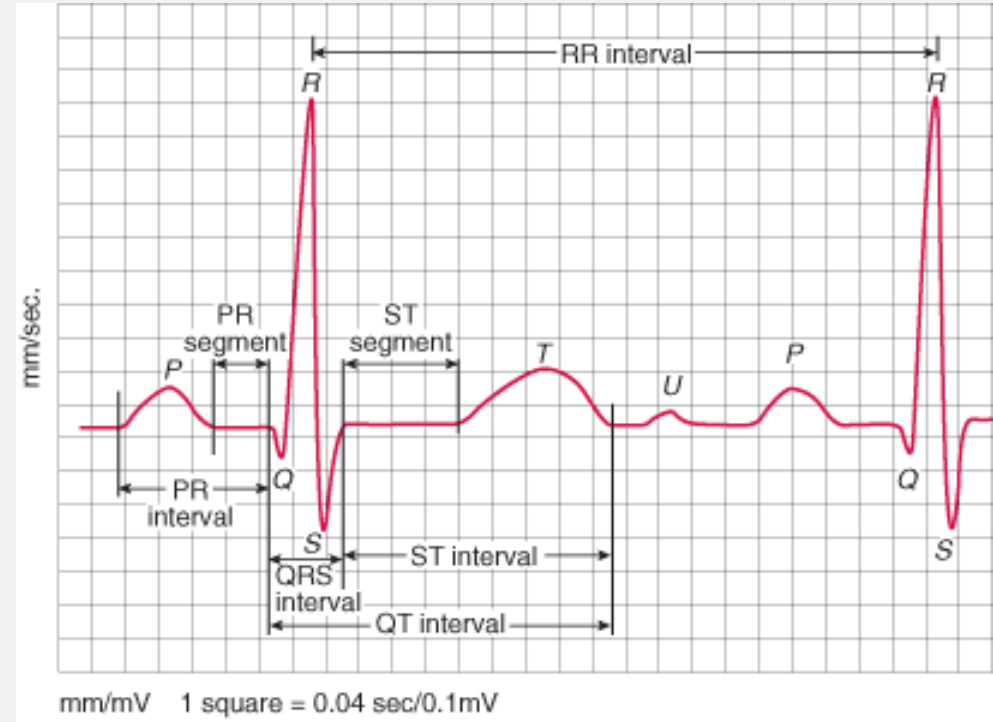


Incomplete RBBB: QRS < 120ms

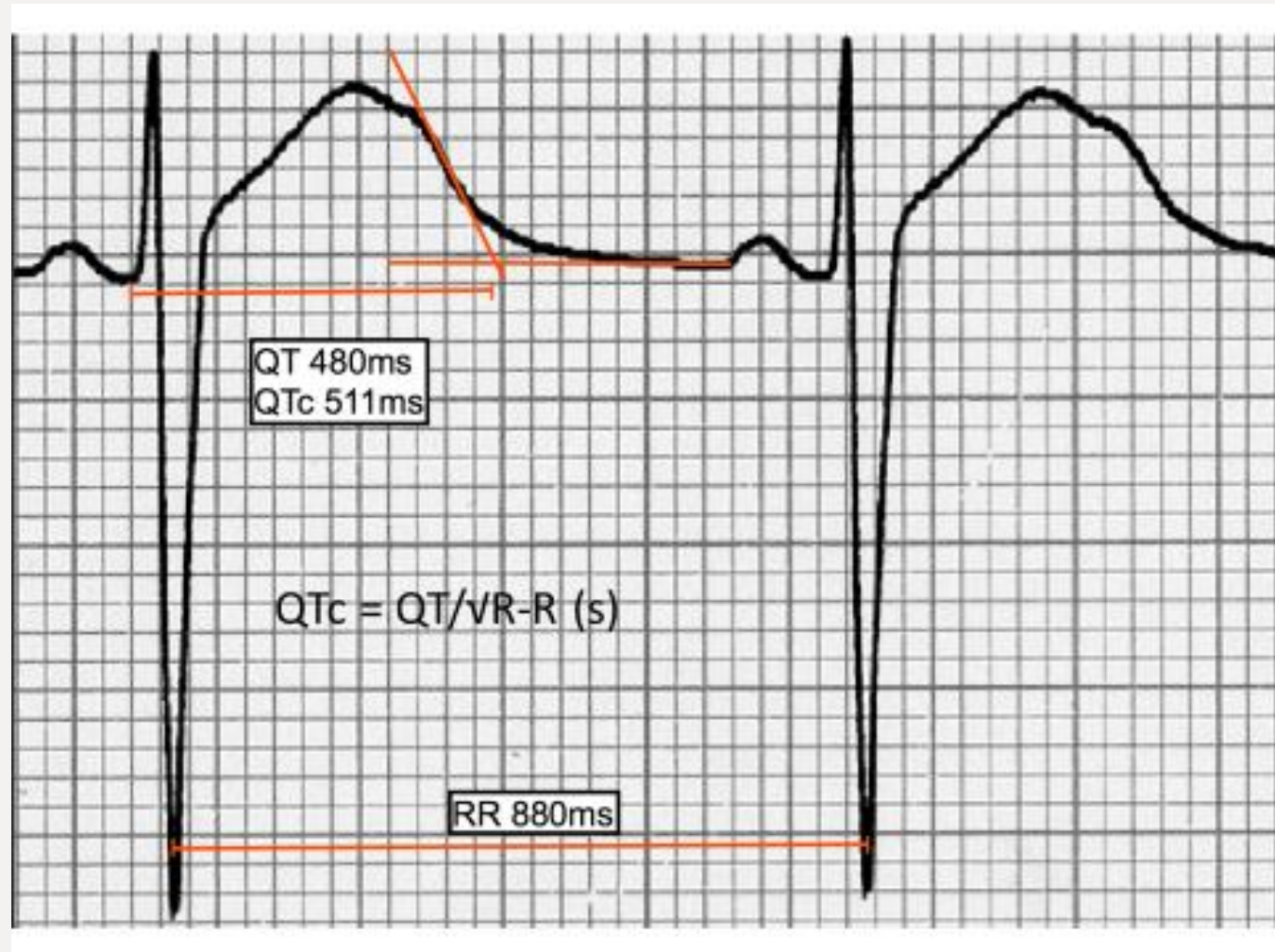


Normal Intervals

- PR
 - 0.20 sec (less than one large box)
- QRS
 - 0.08 – 0.10 sec (1-2 small boxes)
- QT
 - 450 ms in men, 460 ms in women
 - Based on sex / heart rate
 - Half the R-R interval with normal HR



QT interval (lead II or V5-6)





Prolonged QT

- Normal
 - Men 450ms
 - Women 460ms
- Corrected QT (QTc)
 - $QTm/\sqrt{(R-R)}$
- Causes
 - Drugs (Na channel blockers)
 - Hypocalcemia, hypomagnesemia, hypokalemia
 - Hypothermia
 - AMI
 - Congenital
 - Increased ICP



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- R wave transition
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Pathological Q waves

- > 40 ms (1mm) wide
- > 2 mm deep
- > 25% of depth of QRS complex
- Seen in leads V1-3

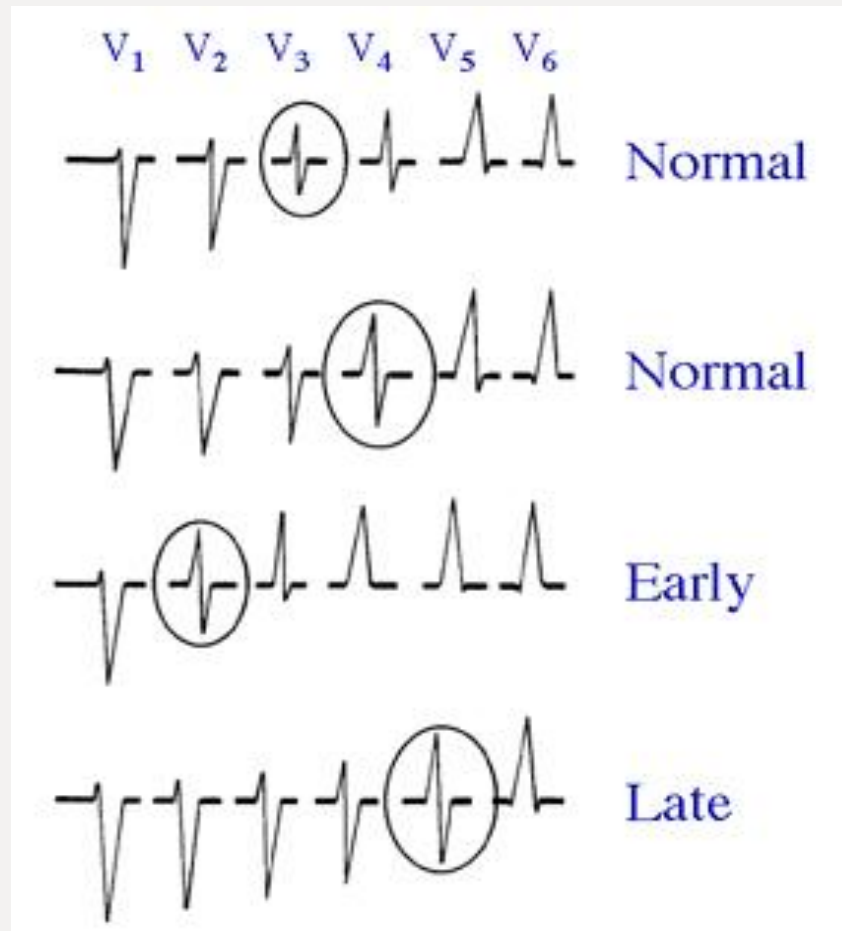




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R wave transition

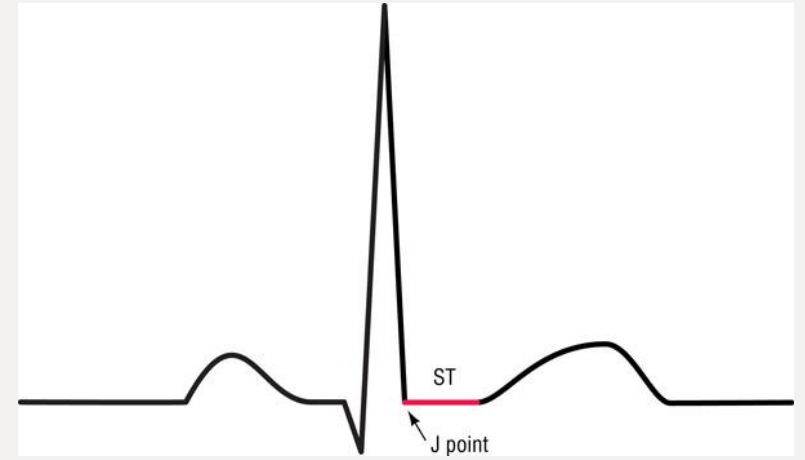




ECG interpretation

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- T waves (and others)

ST Segment



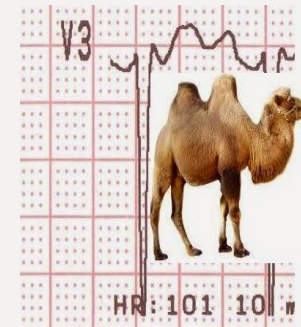
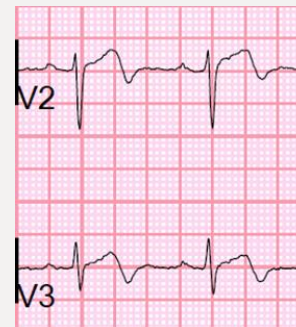
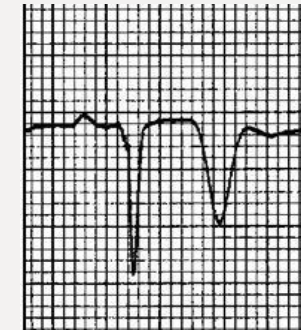
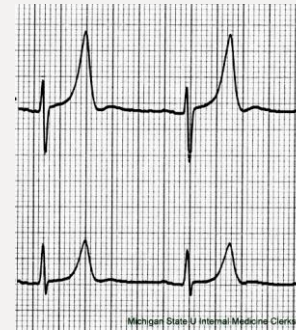


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- **T waves (and others)**

T waves

- Hyperacute / peaked
- Inverted (symmetrical and deep: $> 3\text{mm}$)
 - Children (normal), MI, ischaemia, BBB, ventricular hypertrophy, PTE, HCM, raised ICP
- Biphasic
 - Myocardial ischaemia, hypokalaemia
- "Camel hump"
 - Prominent U or hidden P wave
- Flattened
 - Nonspecific, ischaemia, hypokalaemia





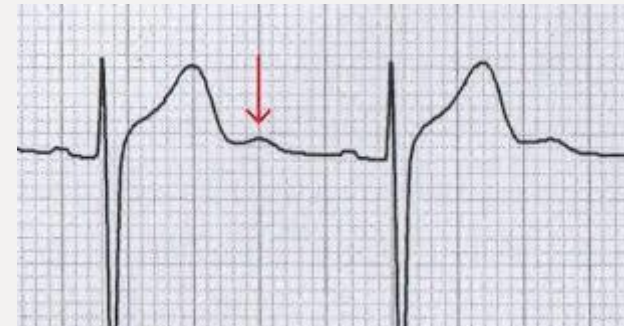
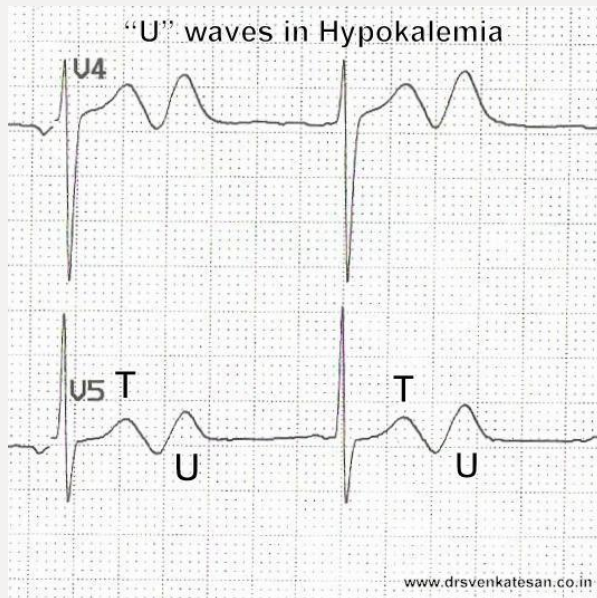
U waves

- ? Delayed Purkinje fibre repolarisation
- Prolonged repolarisation of mid-myocardial "M-cells"
- After potentials from mechanical forces in ventricular wall

- Same direction as T wave
- < 25% of T wave voltage
- Max amplitude is 1-2 mm

U waves

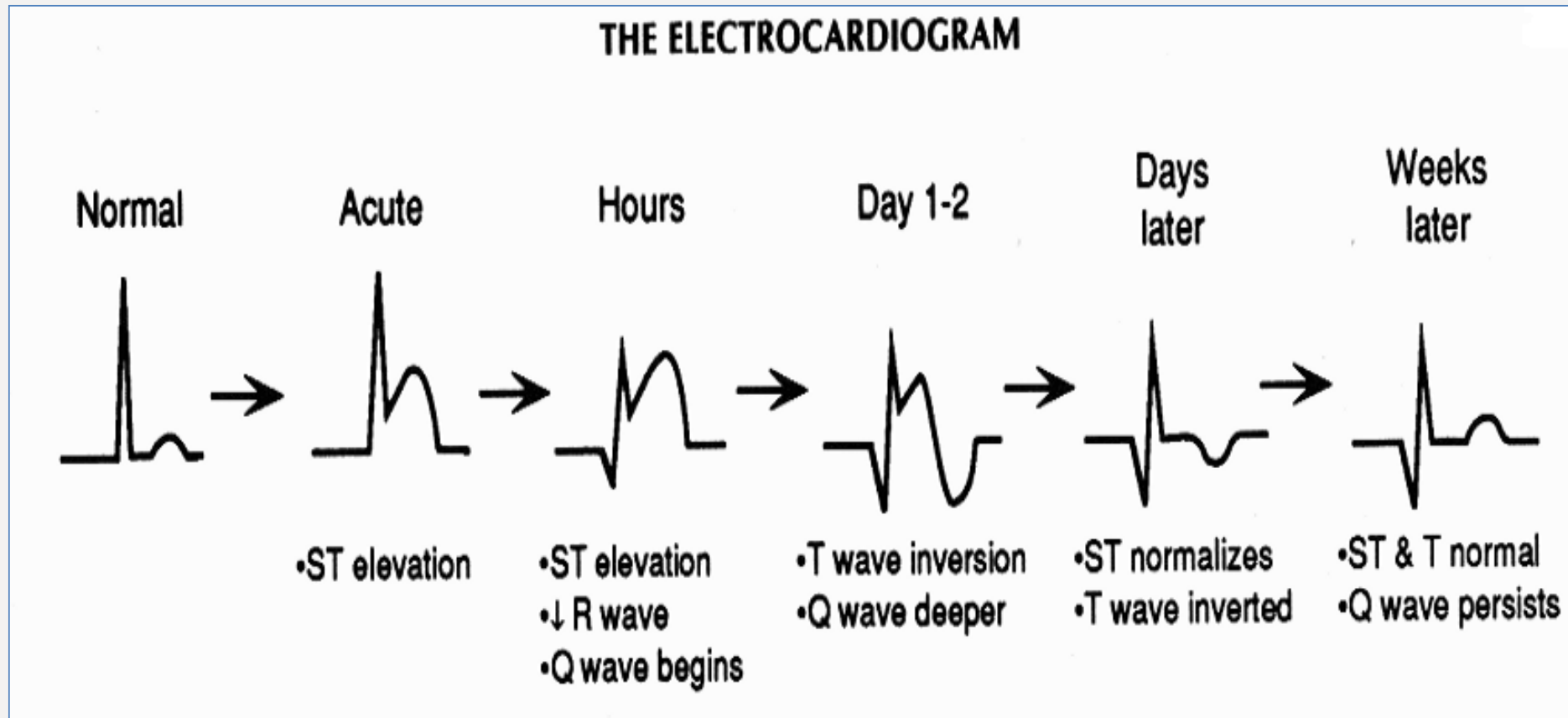
- Prominent
 - Bradycardia, hypokalaemia, hypocalcaemia, hypomagnesaemia, hypothermia, raised ICP, LVH, HCM, digoxin
- Inverted
 - IHD, HBP, valvular HD, congenital HD, cardiomyopathy, hyperthyroidism





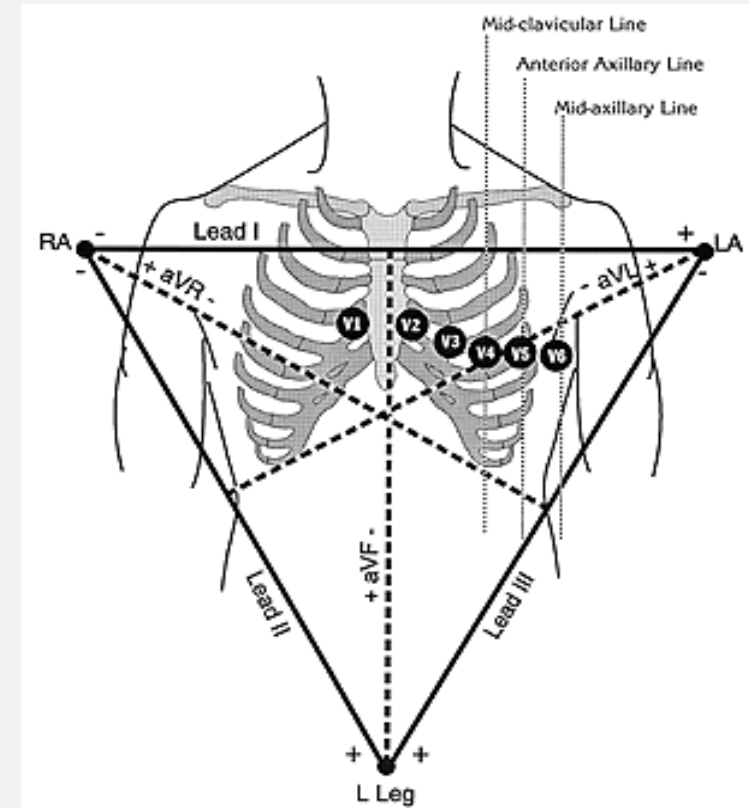
AMI evolution

AMI ECG evolution

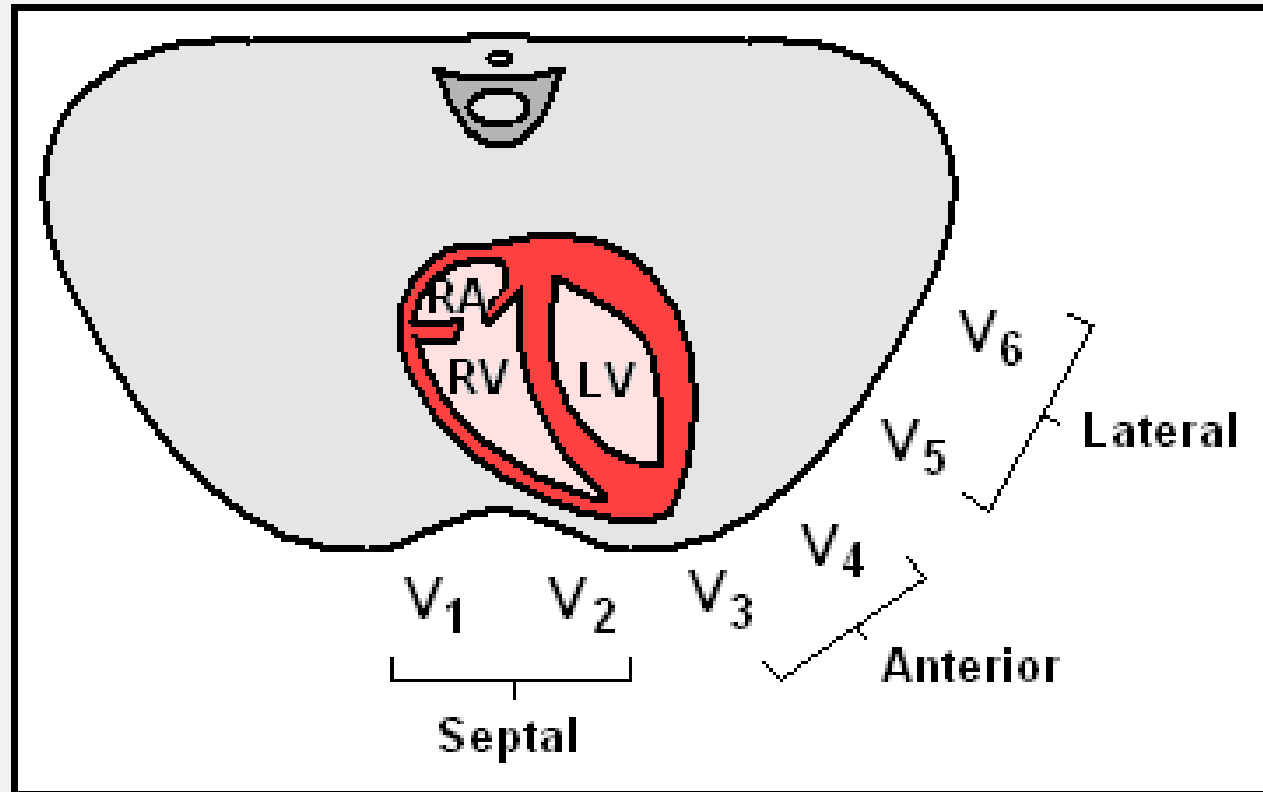


ECG Distributions

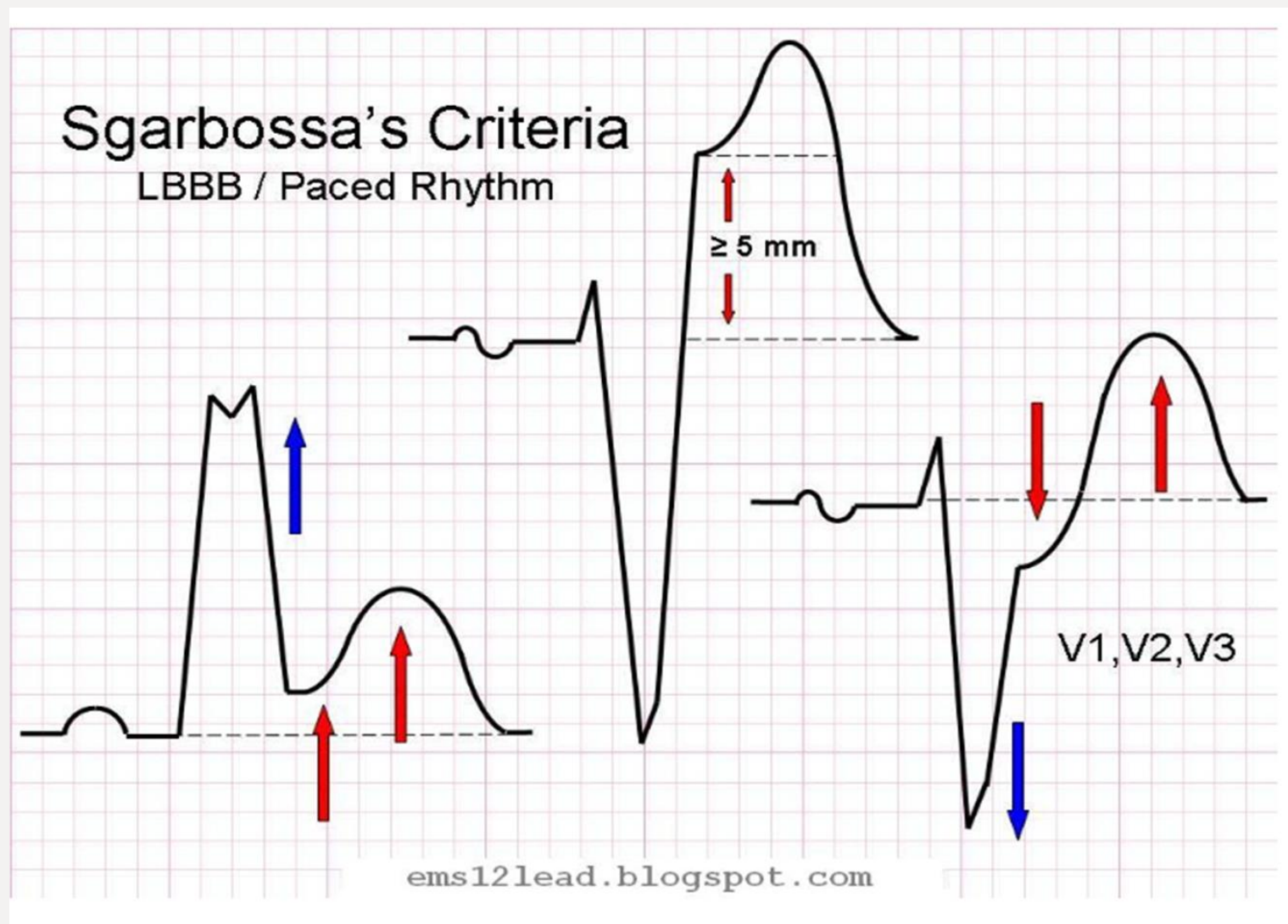
- Septal: V1, V2
- Anterior: V3, V4
- Anteroseptal: V1, V2, V3, V4
- Anterolateral: V4-V6, I, aVL
- Lateral: I and aVL
- Inferior: II, III, and aVF
- Inferolateral: II, III, aVF, and V5 and V6



Precordial Leads



Sgarbossa's criteria

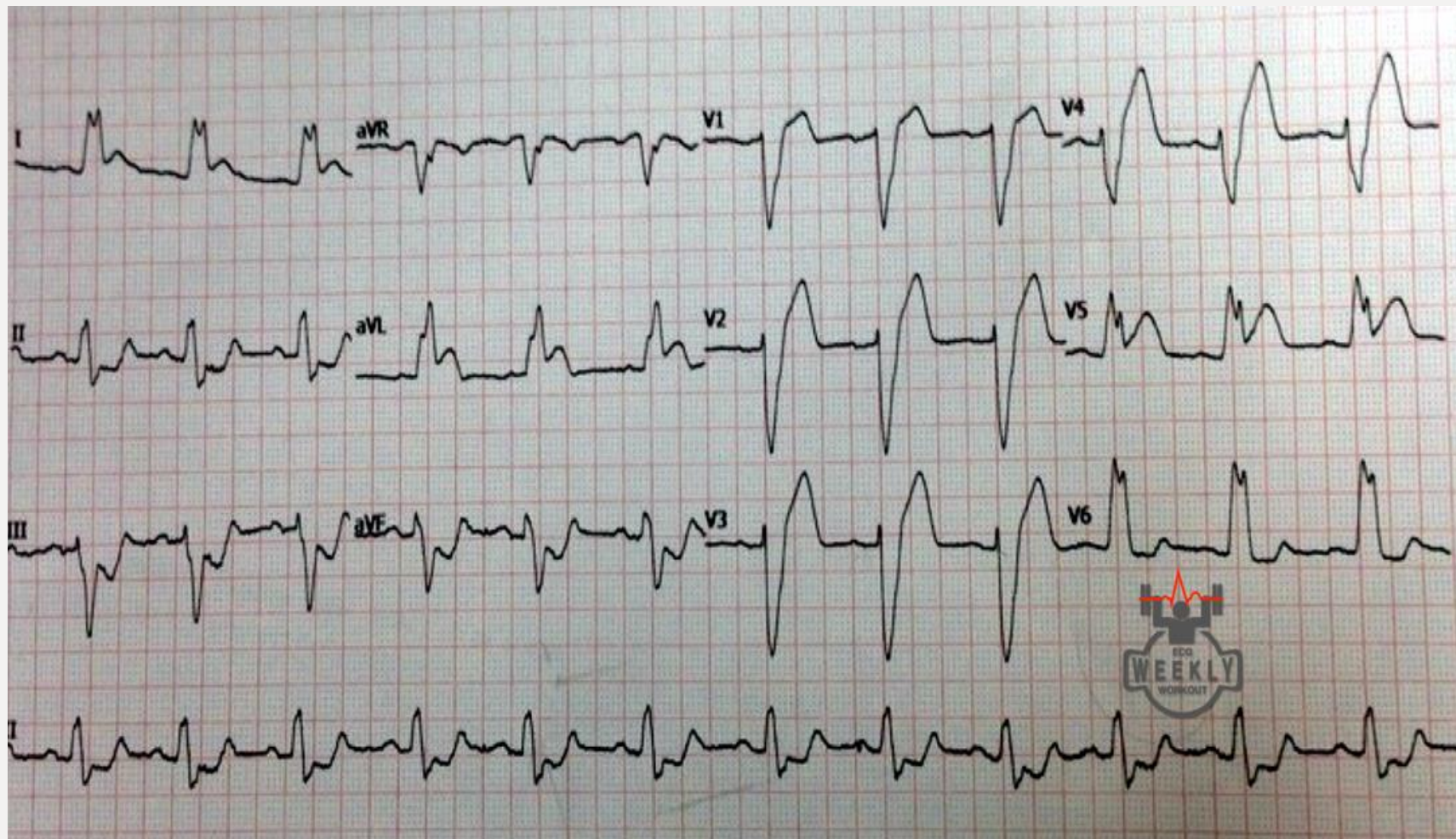




Sgarbossa's criteria

- Concordant ST depression $> 1\text{mm}$ in V1-3 (score 3)
 - Concordant ST elevation $> 1\text{mm}$ in leads with positive QRS complex (score 5)
 - Excessively discordant ST elevation $> 5\text{mm}$ with a negative QRS complex (score 2)
-
- A score ≥ 3 has a specificity of 90% for diagnosing myocardial infarction

Sgarbossa's criteria





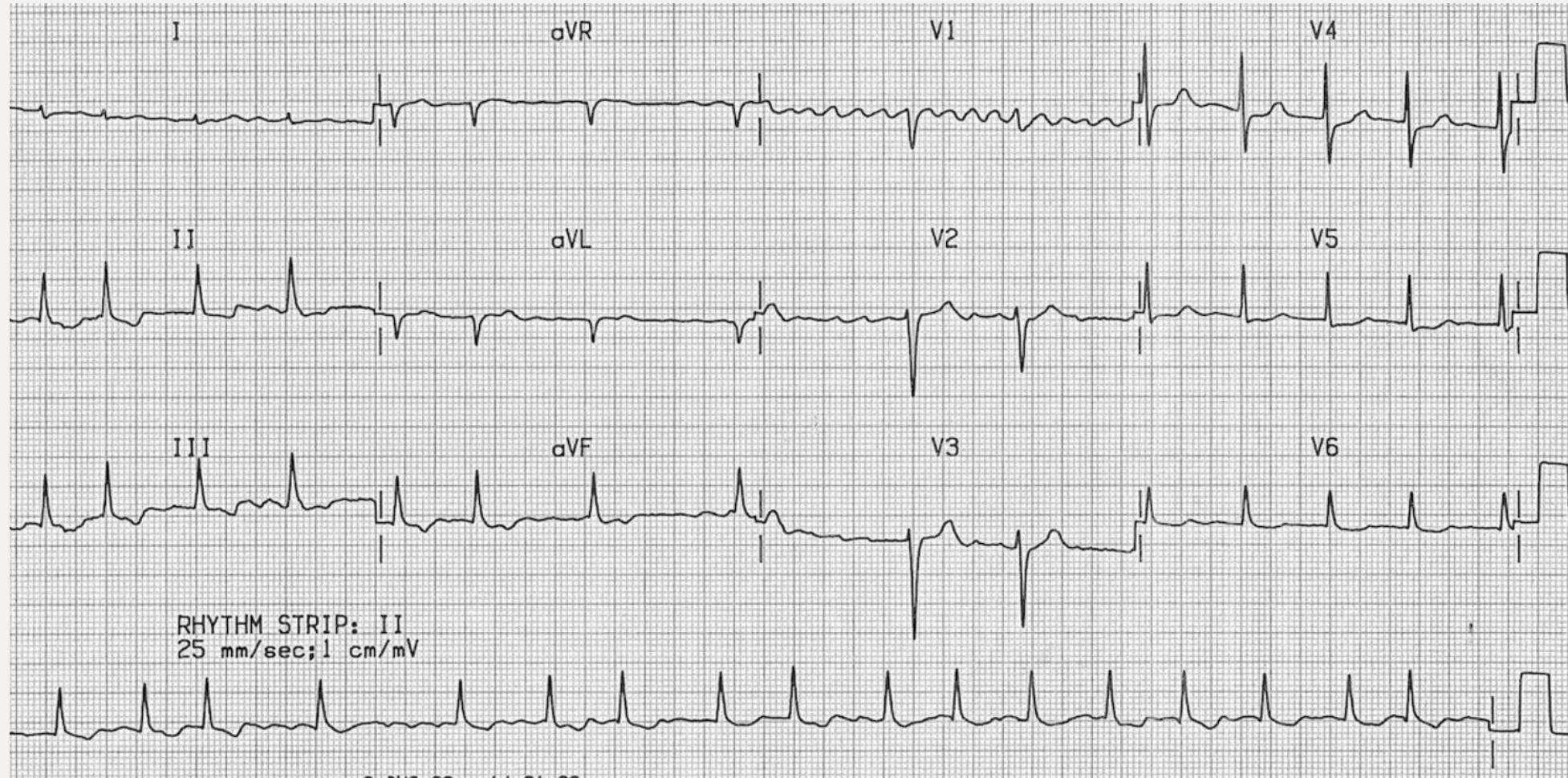
Supraventricular arrhythmias



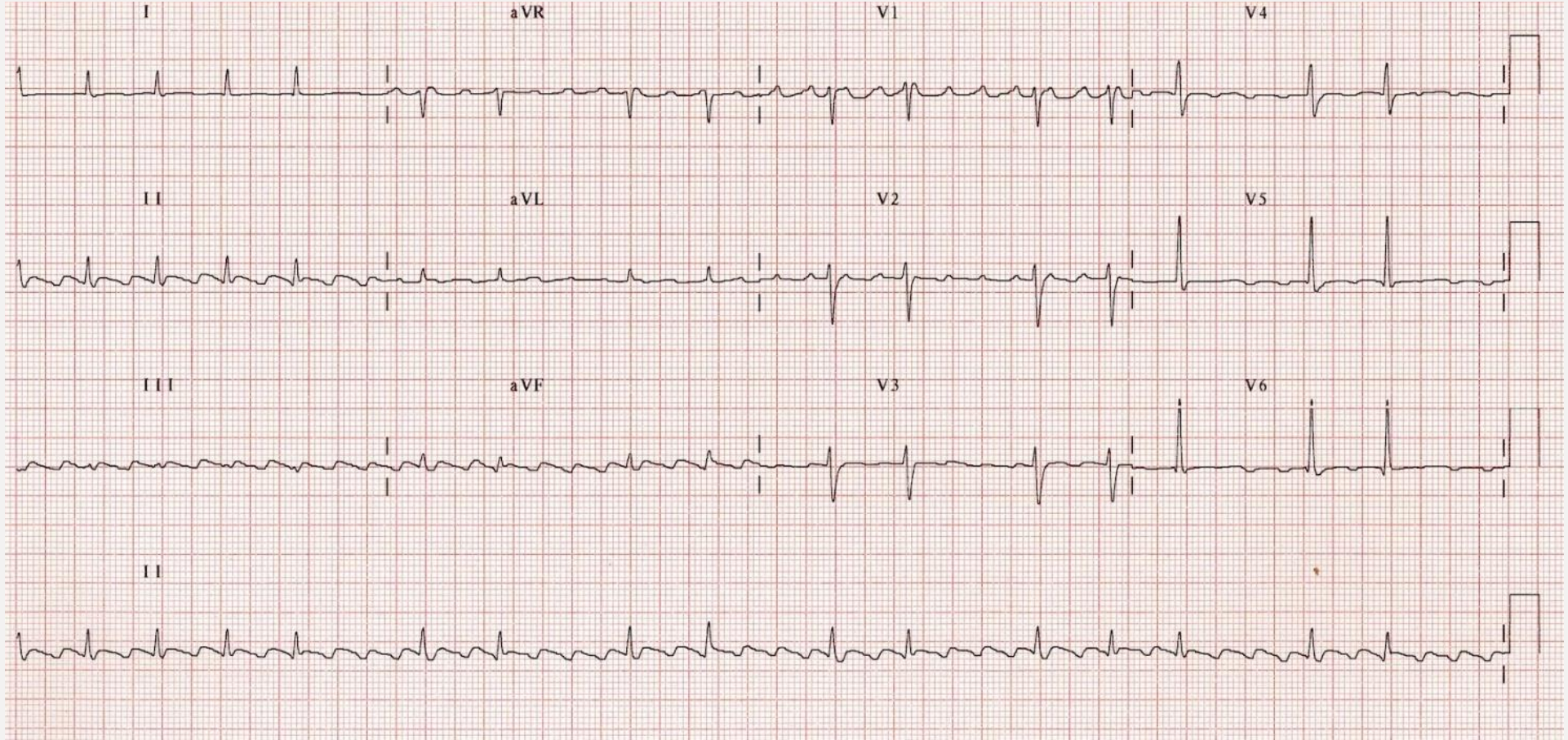
Supraventricular arrhythmias

- Atrial fibrillation
- Atrial flutter
- Supraventricular tachycardias
 - Atrioventricular nodal re-entrant
 - Atrioventricular re-entrant
 - Atrial
 - Sinus
 - Physiological
 - Inappropriate
 - Postural orthostatic tachycardia syndrome
- Others
 - Permanent junctional reciprocating
 - Junctional ectopic
 - Mahaim

Atrial fibrillation



Atrial flutter

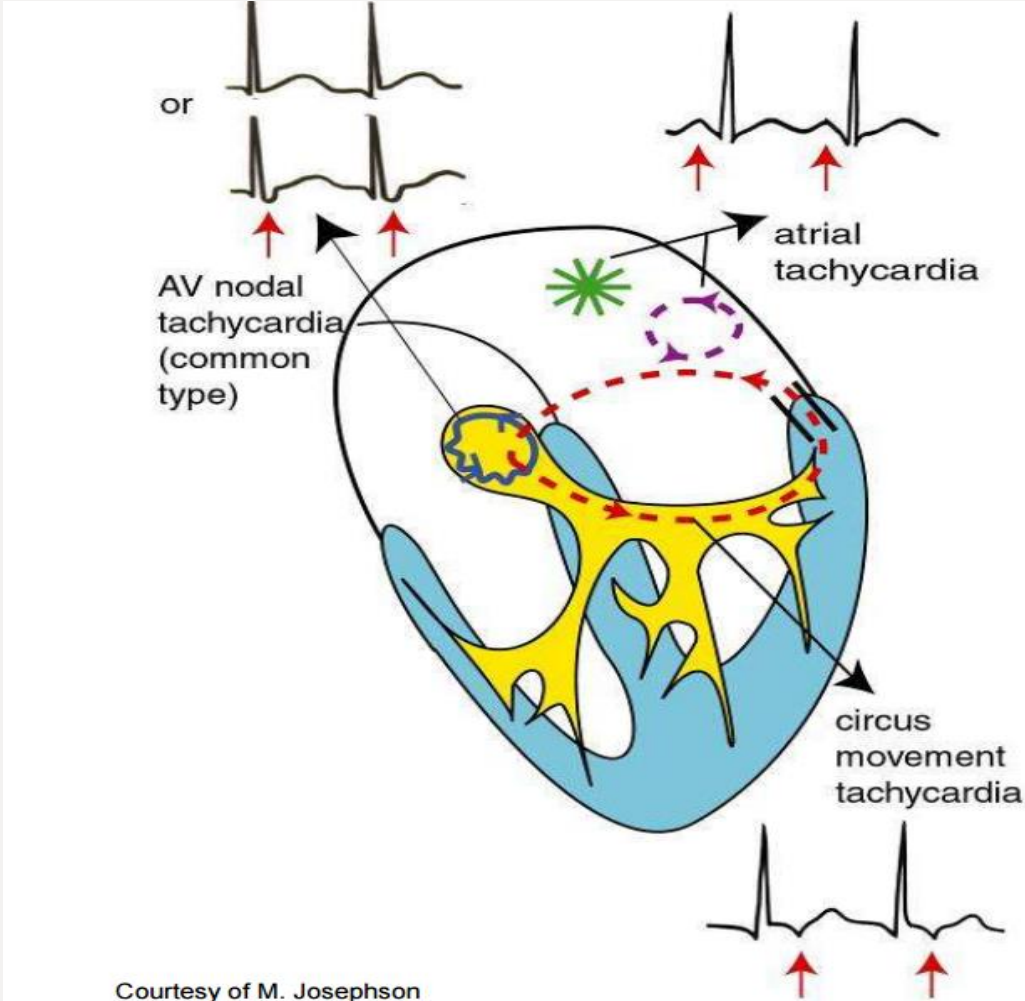




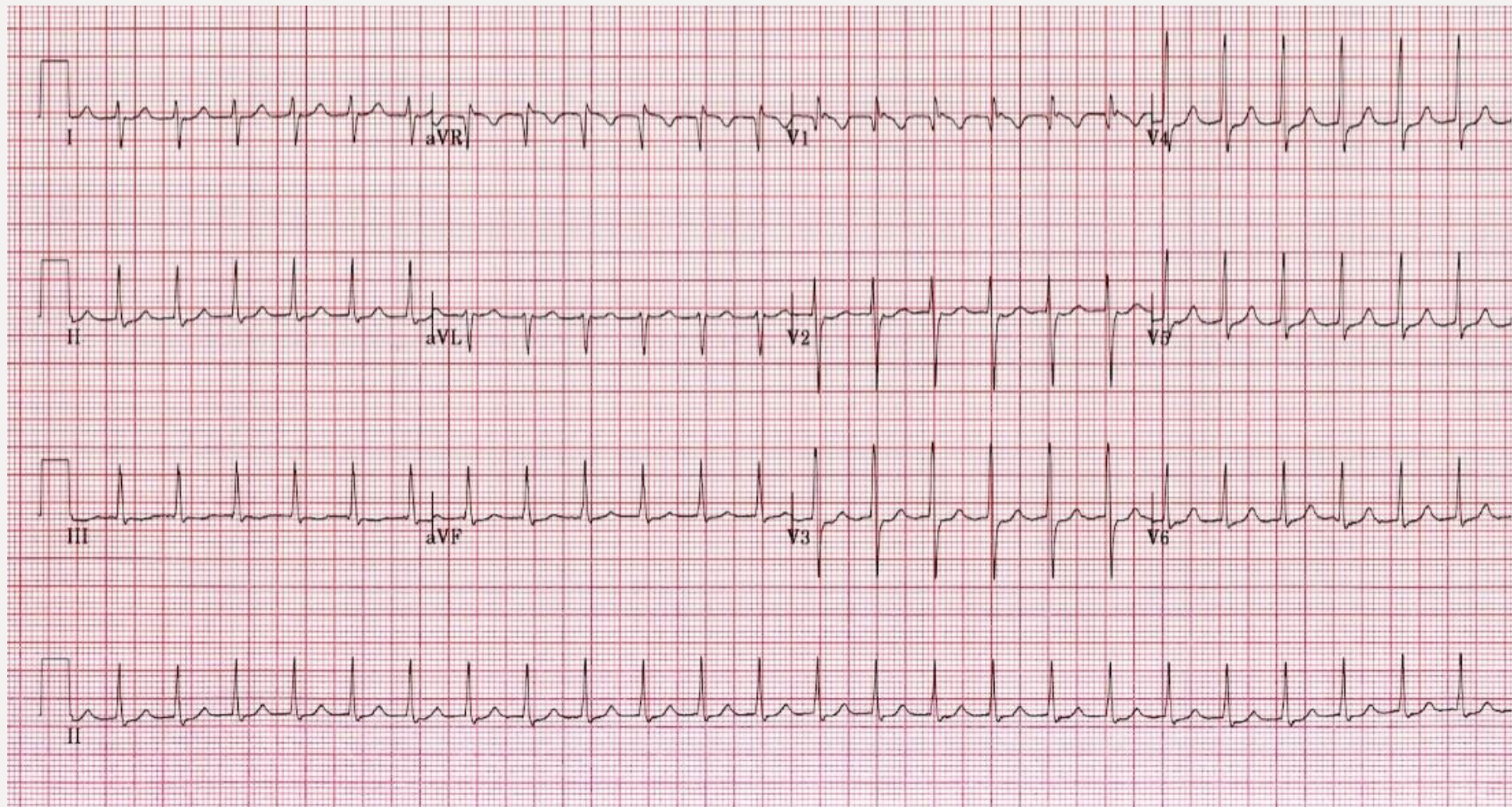
Supraventricular tachycardias

- Most common SVT is AVNRT (60%), followed by AVRT (30%) and AT (10%)
- AVNRT is more common in women (70%)
 - Mean age of onset 32 years
- AVRT is more common in men
 - Mean age of onset 23 years
- AT is more common in older age and structural disease

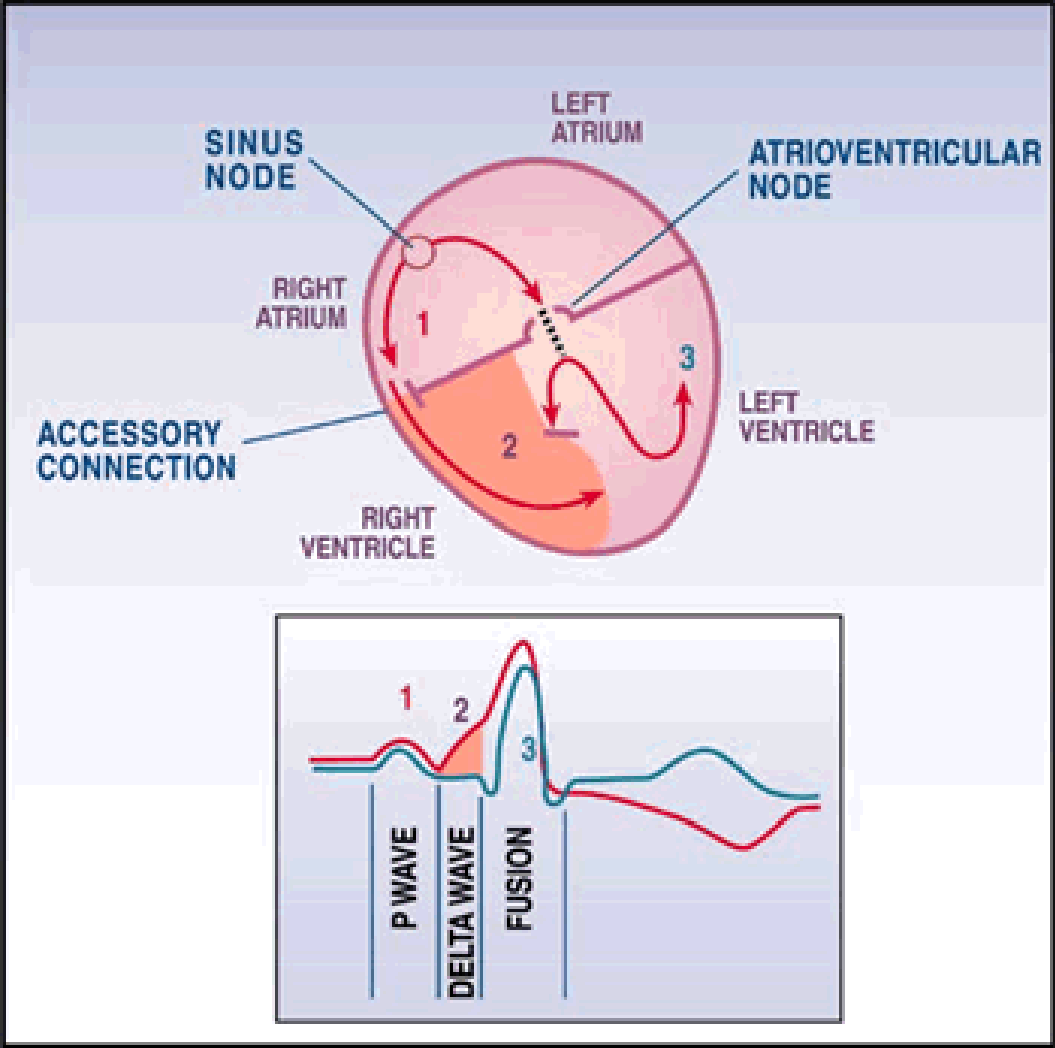
Supraventricular tachycardias (P wave)



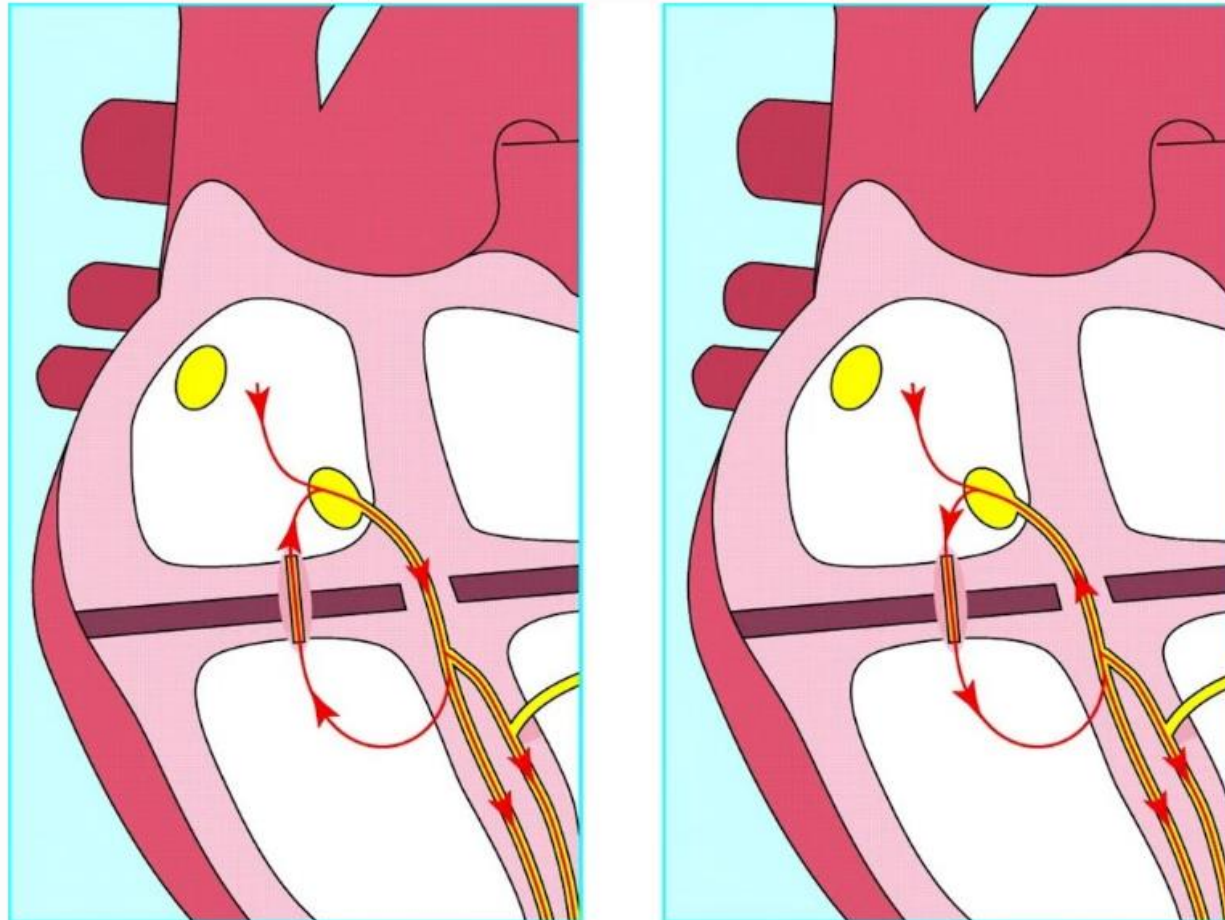
AVNRT



Wolff-Parkinson-White syndrome



Wolff-Parkinson-White syndrome



AVRT with orthodromic (left) and antidromic (right) AV nodal conduction



Broad complex tachycardias

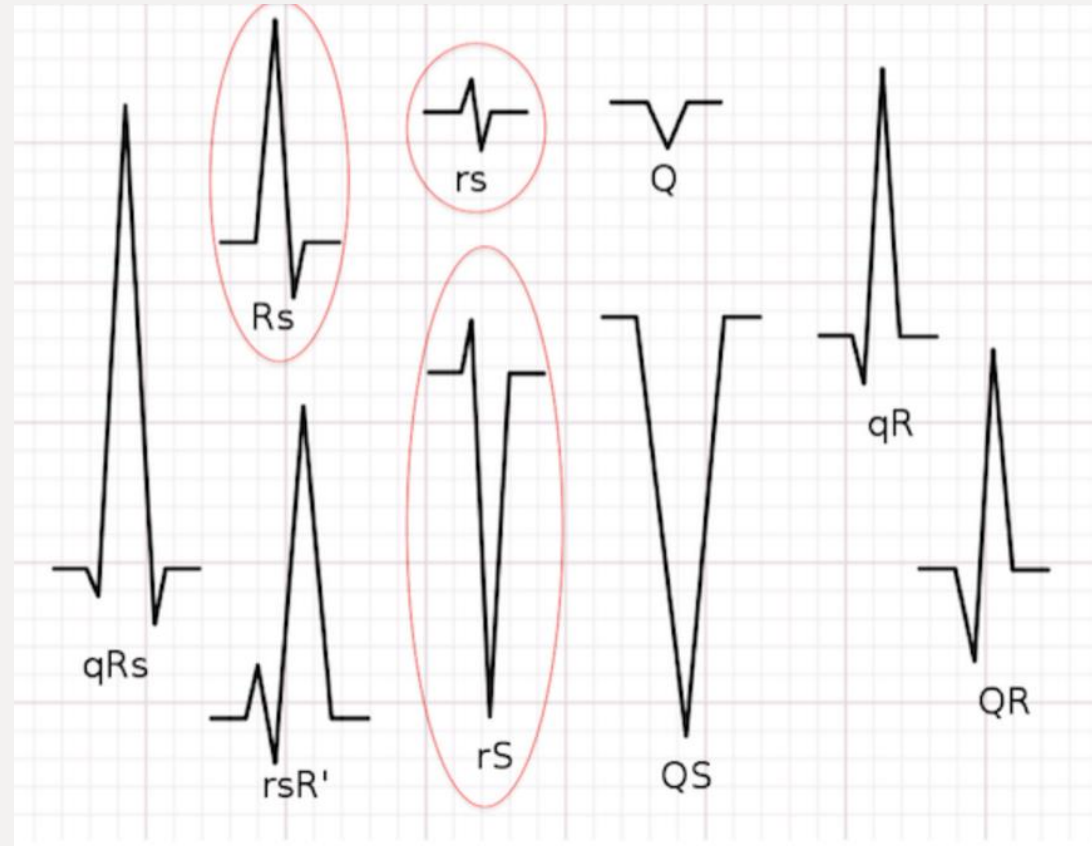


Broad complex tachycardia

- VT
- SVT with aberrant conduction due to bundle branch block
 - Pre-existing BBB
 - Rate related BBB
- SVT with aberrant conduction due to Wolff-Parkinson-White Syndrome

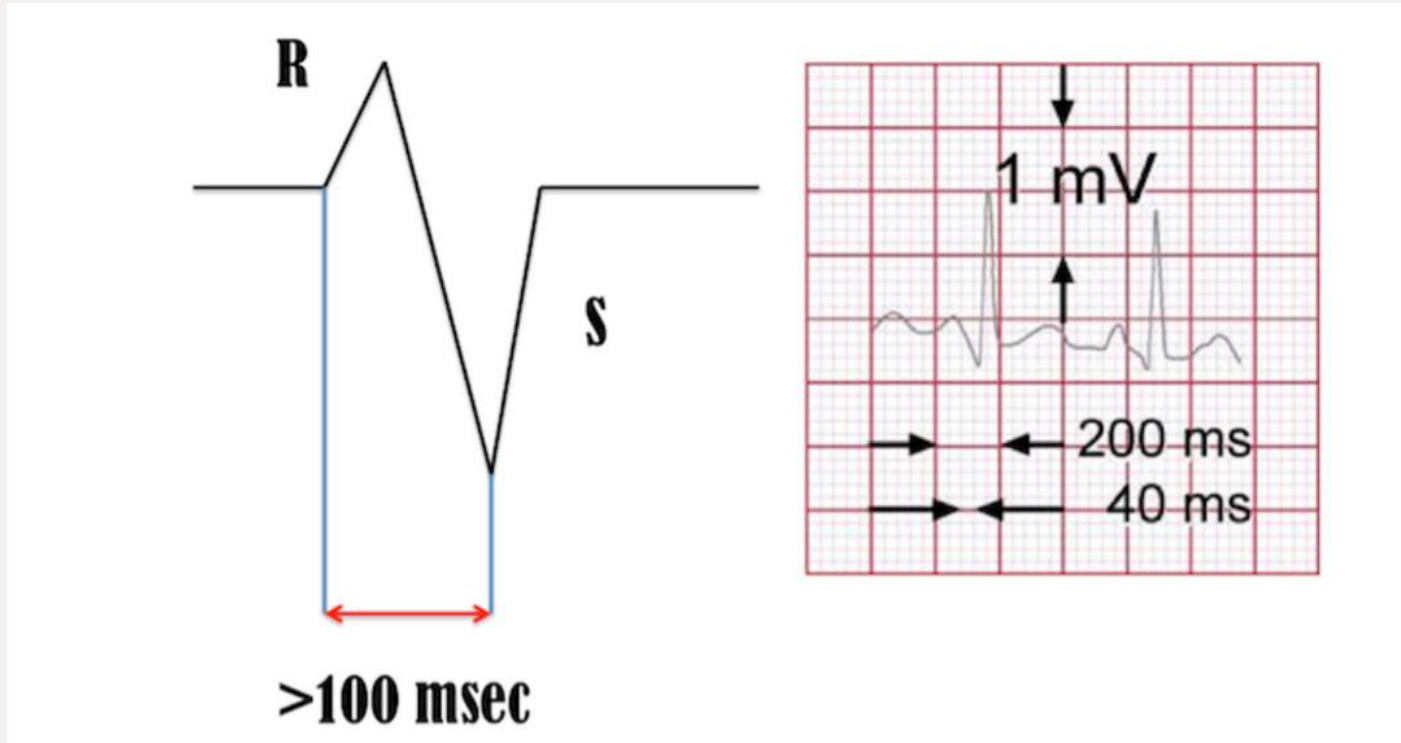
VT Versus SVT with aberrancy - Brugada

- 1. Is there an absence of an RS complex in all precordial leads?
 - Yes = VT, No = next question



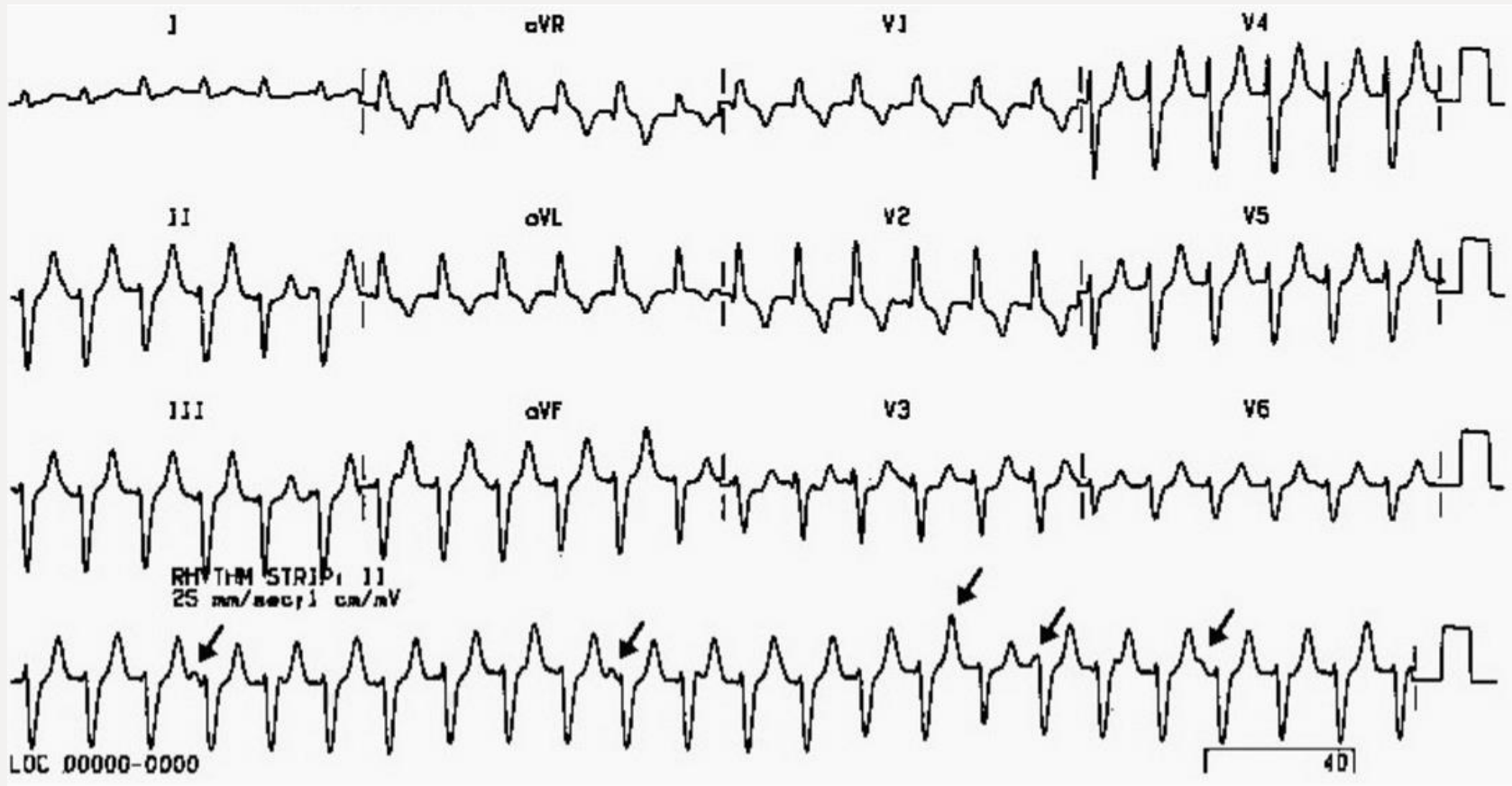
VT Versus SVT with aberrancy - Brugada

- 2. Is the R to S interval >100 msec?
 - Yes = VT, No = next question



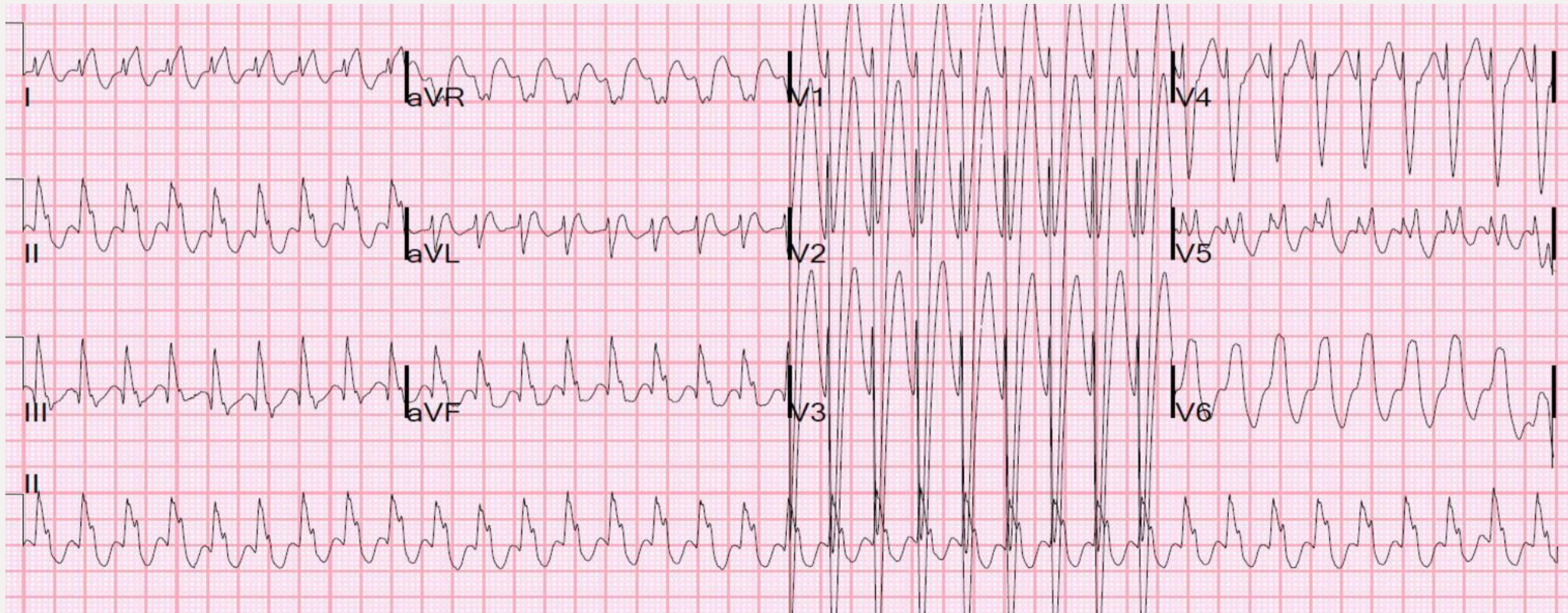
VT Versus SVT with aberrancy - Brugada

- 3. Is there atrioventricular (AV) dissociation?
 - Yes = VT, No = next question



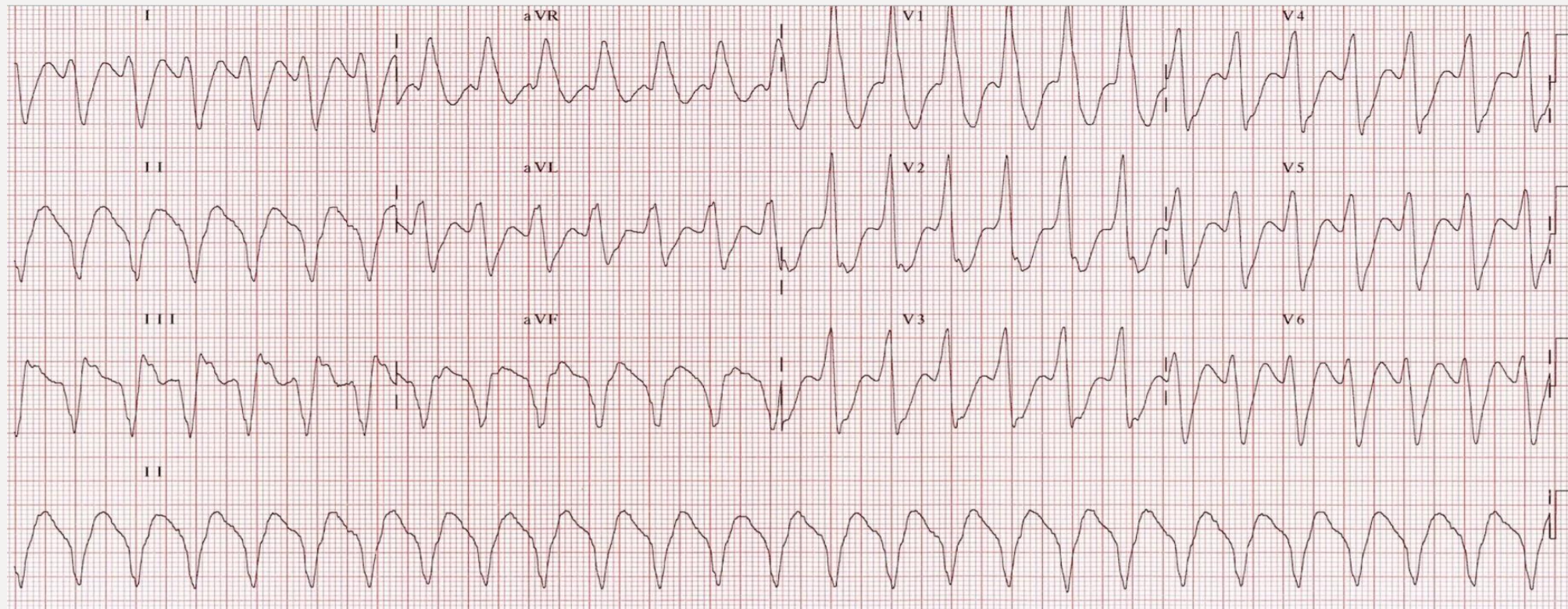
4. Is there morphology criteria for VT present in precordial leads V1/V2 and V6?

LBBB morphology VT



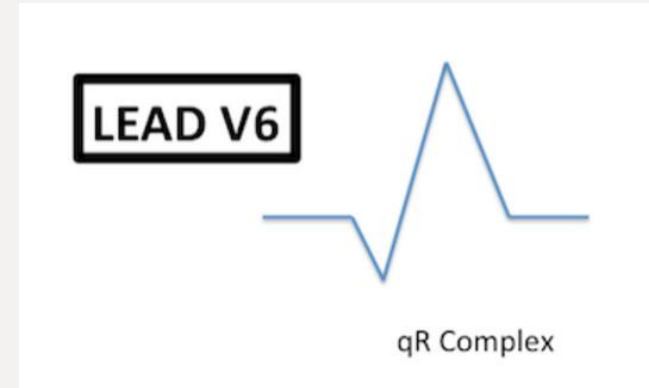
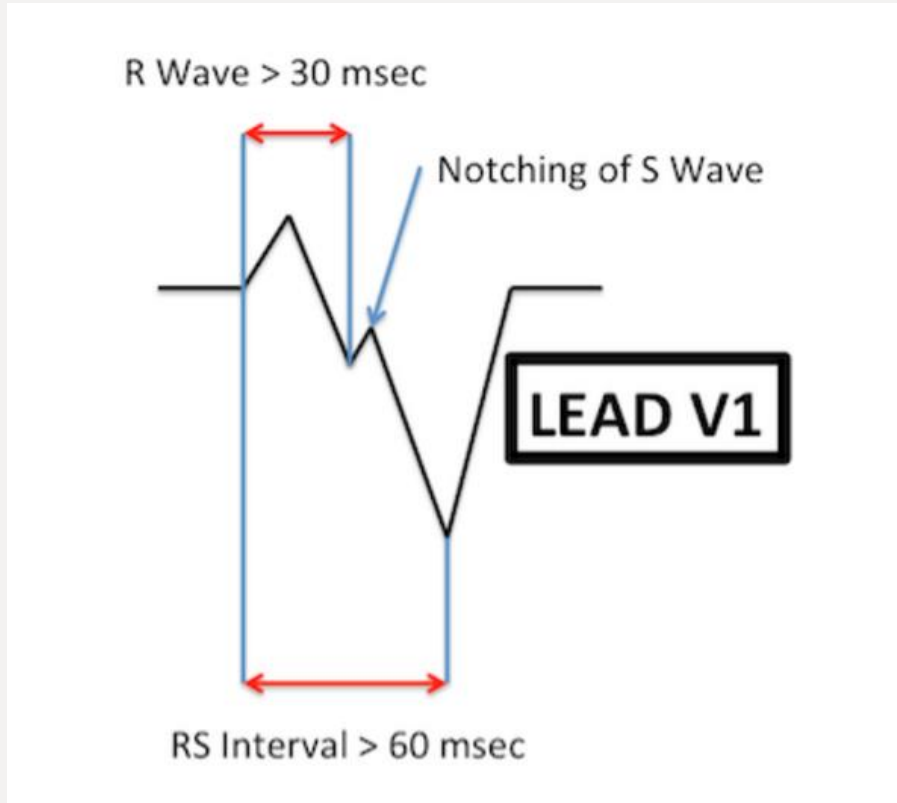
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RBBB morphology VT



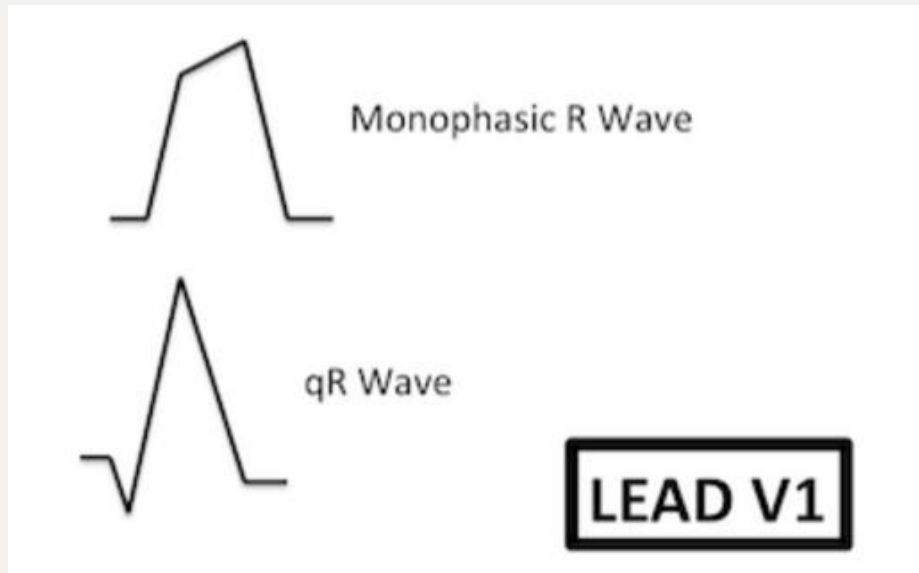
4. Is there morphology criteria for VT present in precordial leads V1/V2 and V6?

- LBBB morphology: dominant S wave in V1 or V2
- LBBB morphology: V6

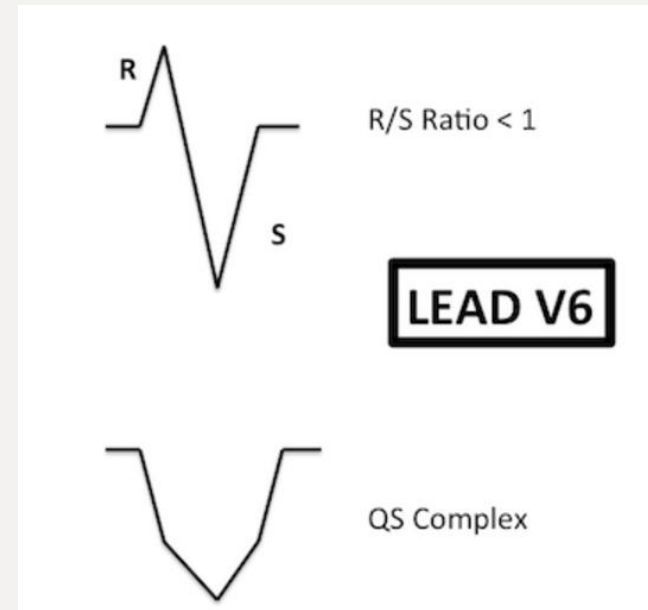


4. Is there morphology criteria for VT present in precordial leads V1/V2 and V6?

- RBBB morphology: dominant R wave in V1 or V2



- RBBB morphology: V6





VT Versus SVT with aberrancy

- IF IN DOUBT, TREAT AS VT



ECG Quiz
available as separate download