Pediatric ECG Interpretation

- Basic principles of electrocardiography
- Approach to the ECG
- Criteria for chamber enlargement and hypertrophy
- Dysrhythmia diagnosis

AUGMENTED LEADS

- **INCREASE SIZE OF POTENTIALS BY 50%**
- **DEPOLARISATION FROM RIGHT TO LEFT. SO LEADS ON RIGHT –VE FOR P, QRS, T. eg aVR**

  LEFT LEADS RECEIVE DEPOLARISATION SO LEADS ON LEFT AND INFERIOR +VE
- **FOR P,QRS, T.eg aVL, I, II, III, aVF**
-

QRS AND T

- QRS is depolarisation
- T is replarisation which is counter current to depolarisation
- So expects waves to be opposite each other in the leads
- However both are in same direction in the leads

REASON

- **Depolarisation is from endocardium to epicardium**
- **Repolarisation is from epicardium to endocardium**
- **If repolarisation were from endocardium to epicardium direction would be opposite**

VENTRICULAR ACTIVATION

- **Activation of ventricles is from the septum , starting from left side to right of septum**
- **Leads to initial R wave I right leads and Q wave in right leads, ie I, AVL, and V5,V6**
- **In complete LBBB, septum is activated from right to left leading to Q waves in V1, V2**

Why everybody hates pediatric ECG's

- **Overwhelming – what do the different leads mean?**
- **Age dependence – must understand how age impacts the ECG**

Age-related Changes

- Newborns will have right ventricular dominance (right axis, right ventricular hypertrophy)
- Younger patients will have faster heart rates
- T-wave inversion steadily goes away in precordial leads
- Most intervals will get longer
- Need a chart

Principles Summary

- **ECG is a voltmeter measuring the potential difference between the 2 poles as it changes with time**
- **Excitation coming toward the positive pole written as an upward direction**
- **Biphasic signal indicative of the impulse coming toward and then away from the positive pole**
- **Greater muscle mass can hide excitation of areas of smaller mass**

SA NODE, AV NODE

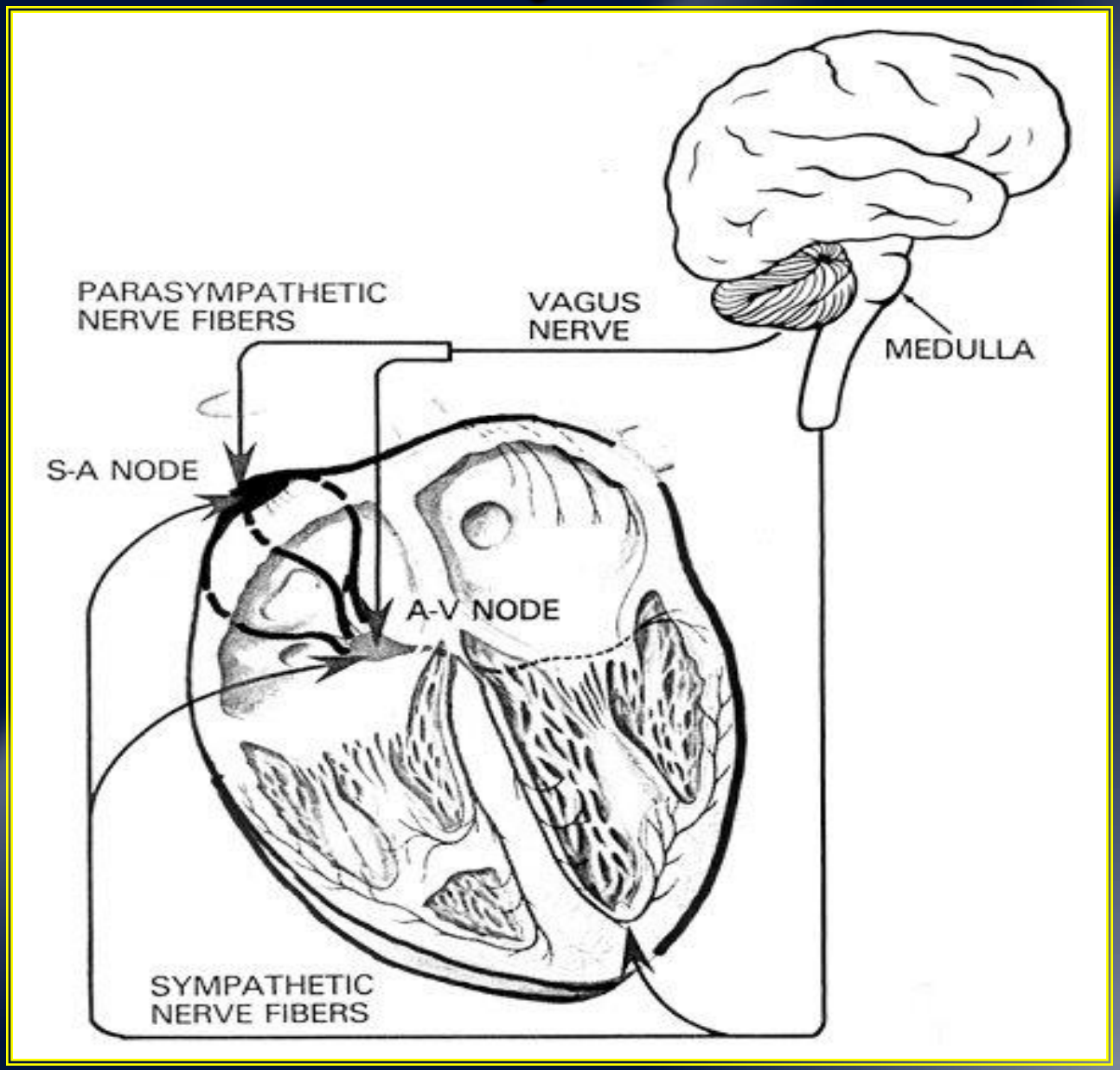
- **SA node is located high in the RA near junction with SVC**

- **AV node is located low in the RA**
- **BUNDLE OF HIS connects AV node with summit of interventricular septum**
- **Bundle then divides into right and left bundle branches into RV and LV**

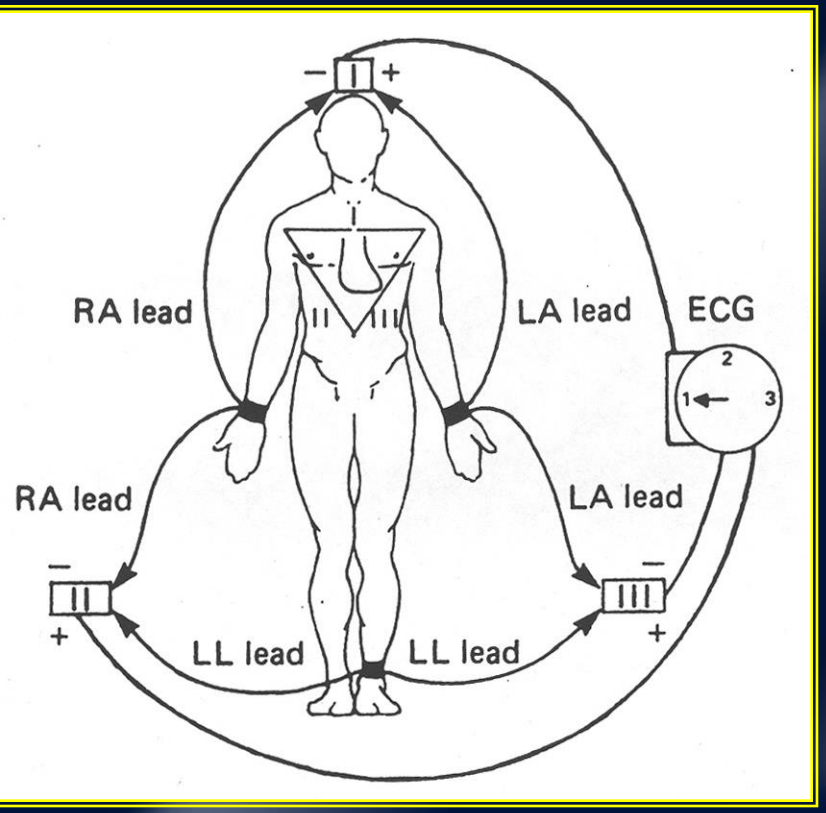
CONDUCTION

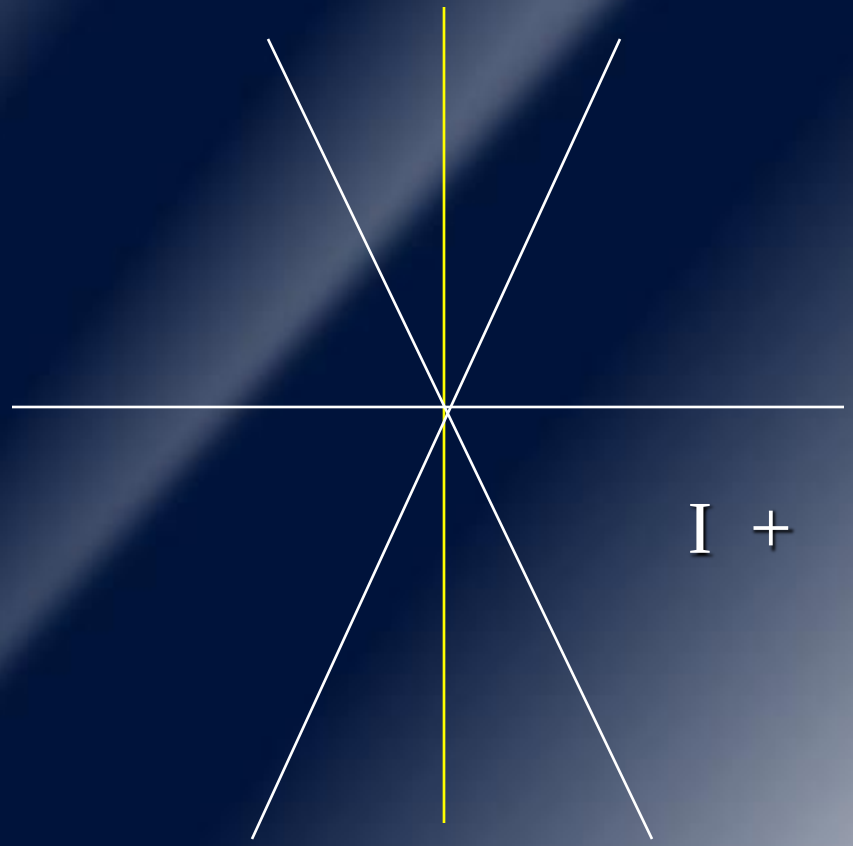
- **Activation from endocardium to epicardium in both ventricles**

Conduction System Anatomy

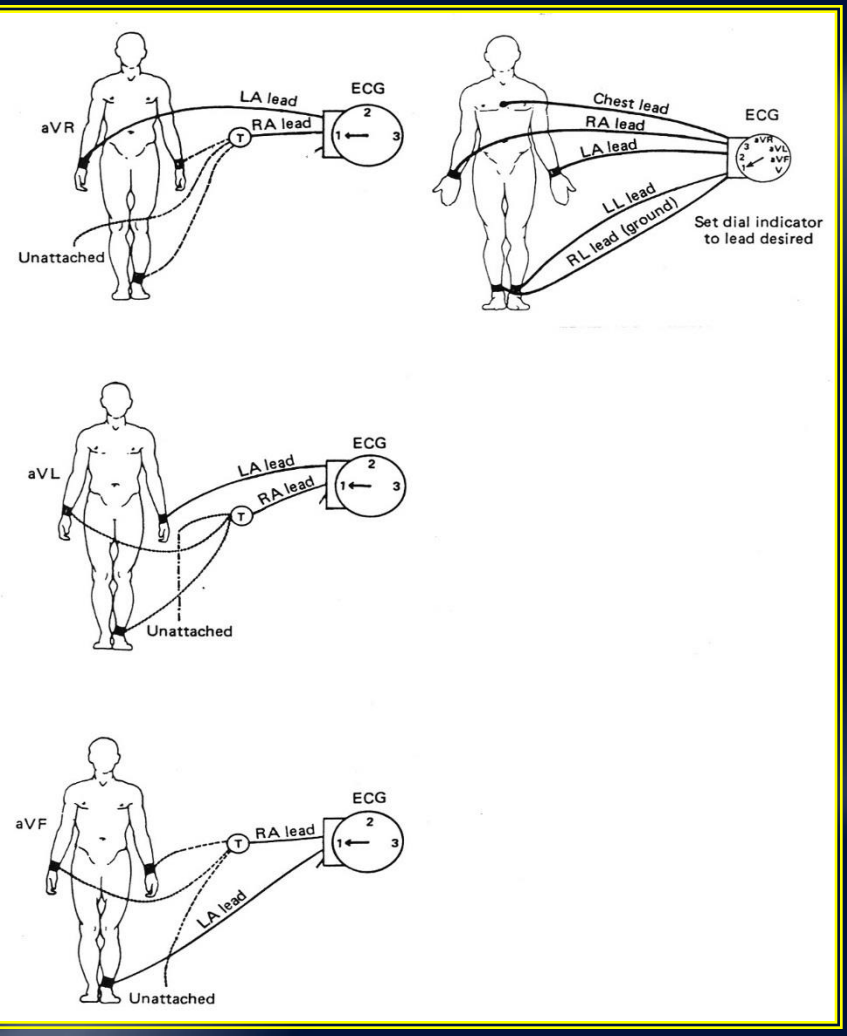


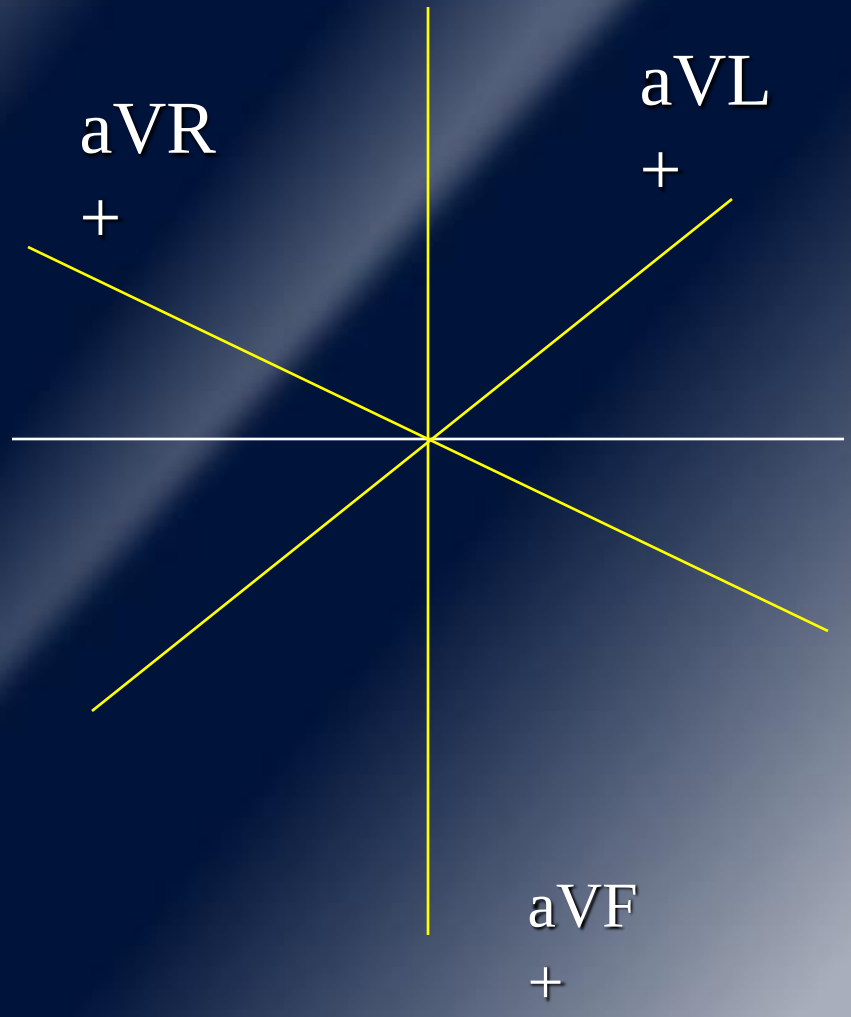
Limb Leads
Einthovan Triangle



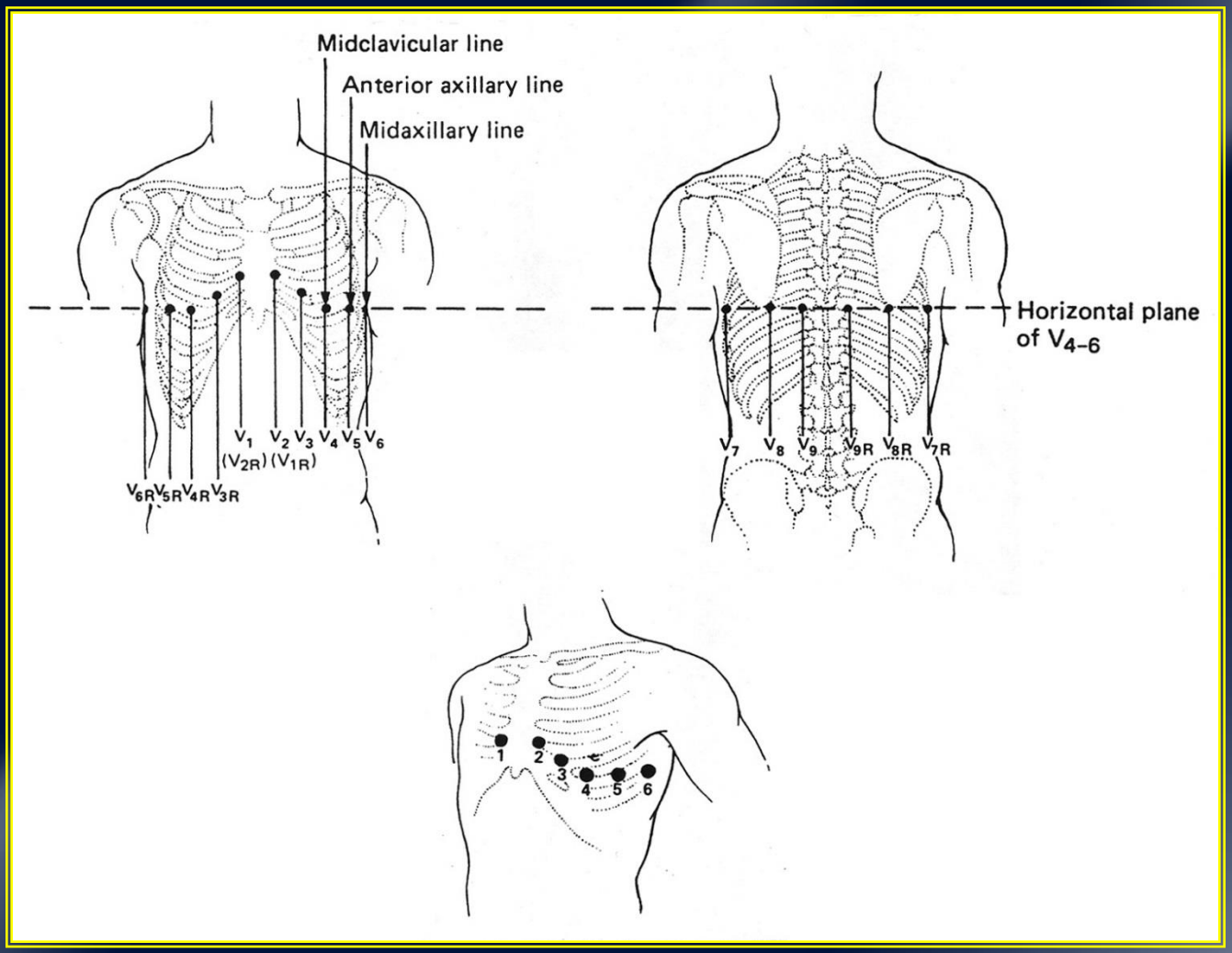


Augmented Leads
Wilson's Central Terminal



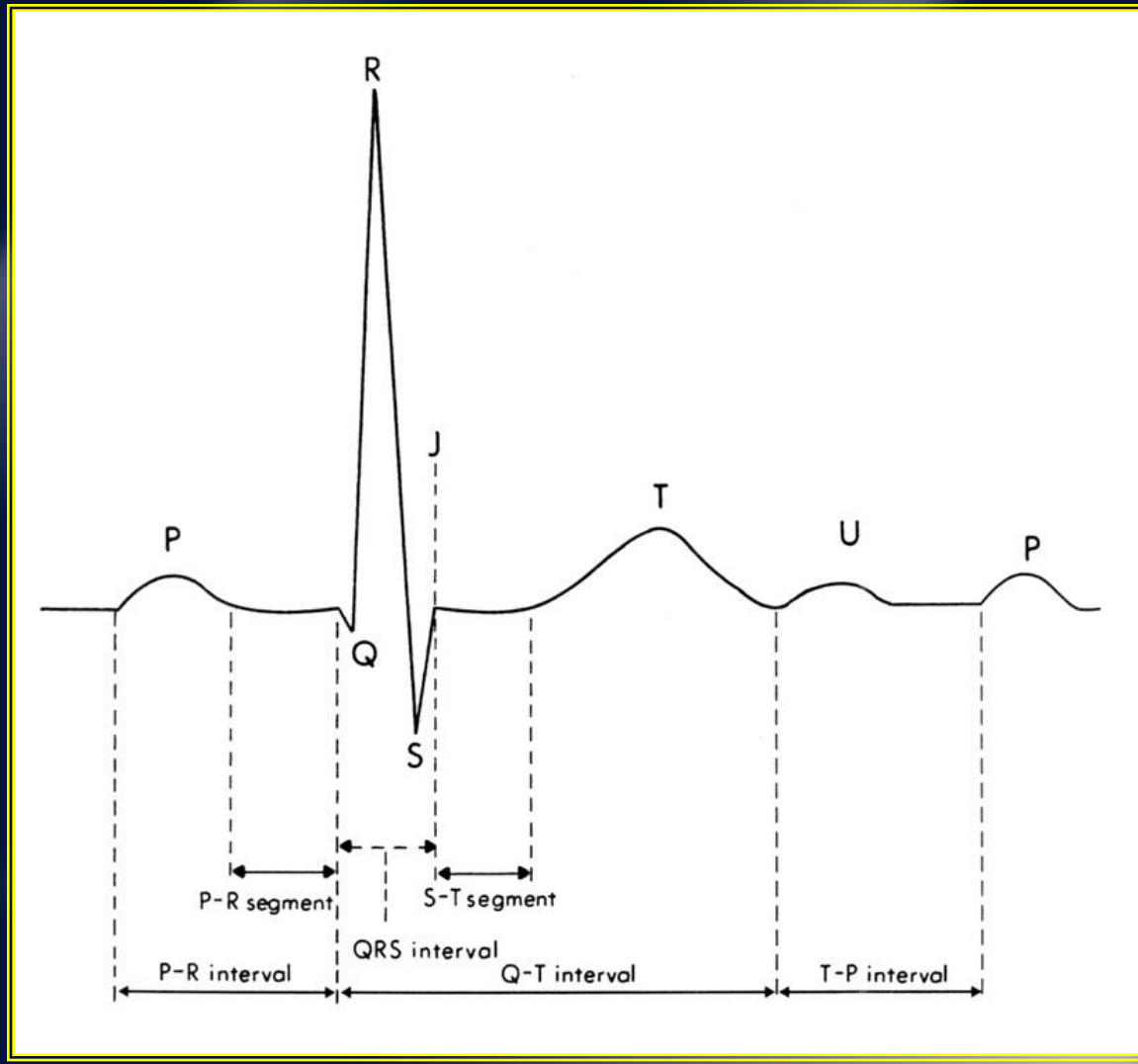


Precordial Leads



Nomenclature & Measured Intervals

- **Rates**
  - **Atrial**
  - **Ventricular**
- **PR interval**
- **QRS duration**
- **QT interval**
- **QTc: QT/$\sqrt{\text{R-R}}$**



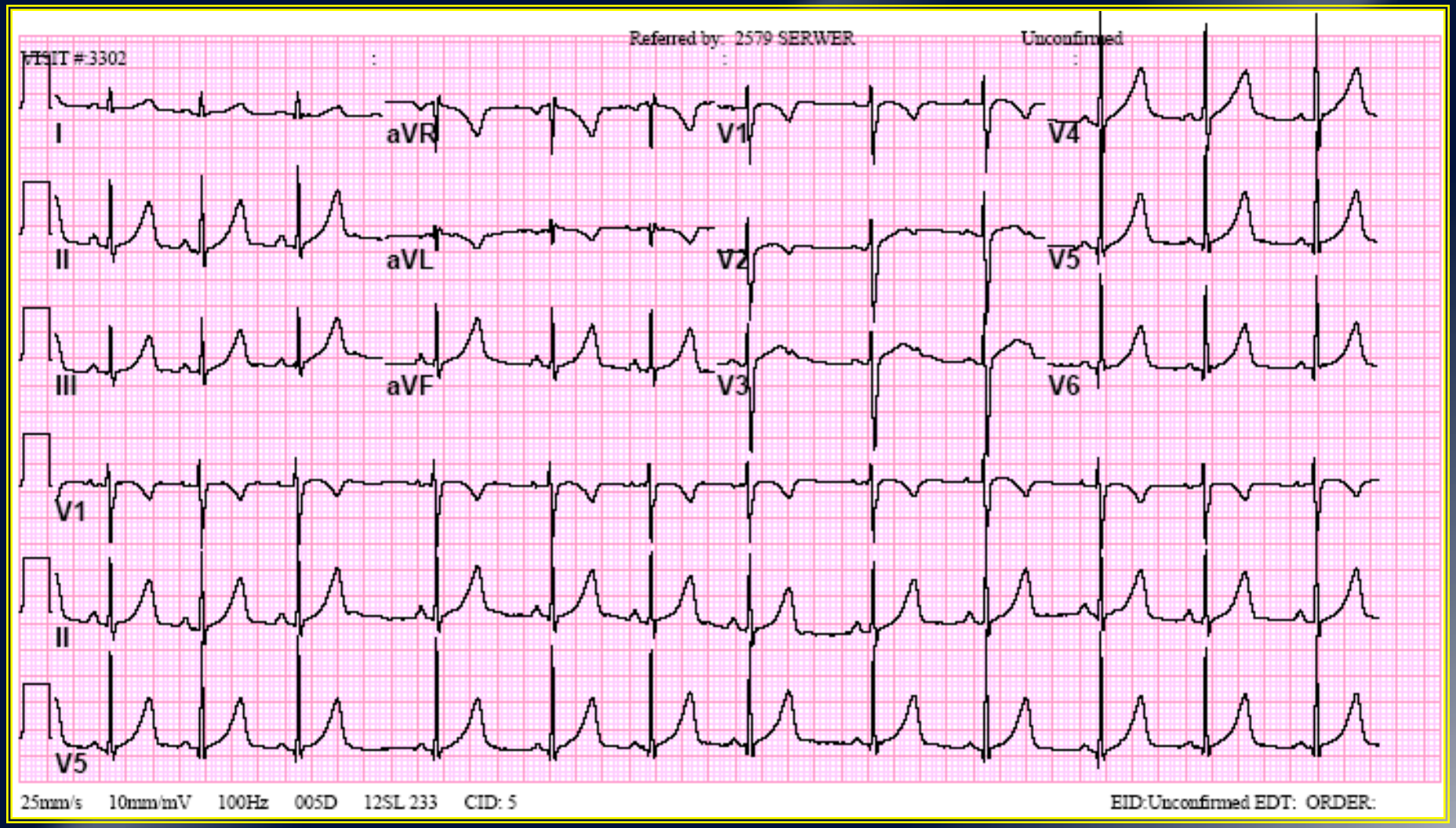
Approach to the ECG

- **Rate**
- **Rhythm**
- **Axis**
- **Hypertrophy/Enlargement**
- **QRS Morphology**
- **ST-T waves**

DURATION

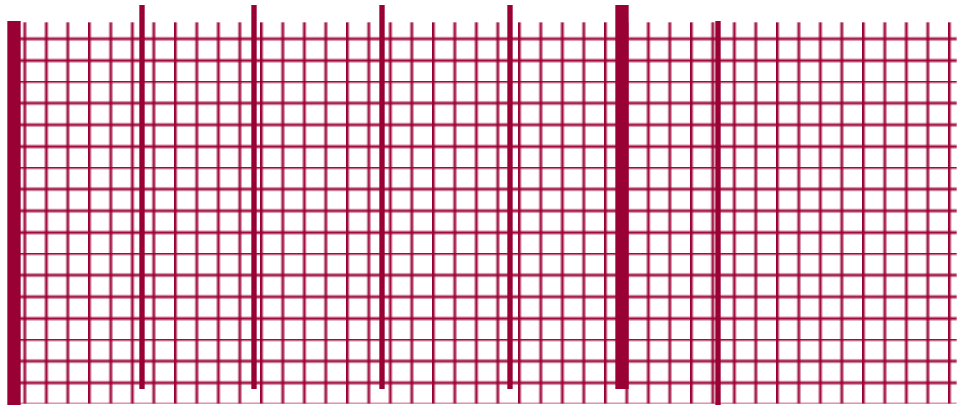
- **P:HEIGHT:3 SMALL SQUARES**
  WIDTH:21/2 SMALL SQUARES
  INITIAL PART BY RA
- **QRS:WIDTH:21/2 SMALL SQUARES**
-

Normal ECG

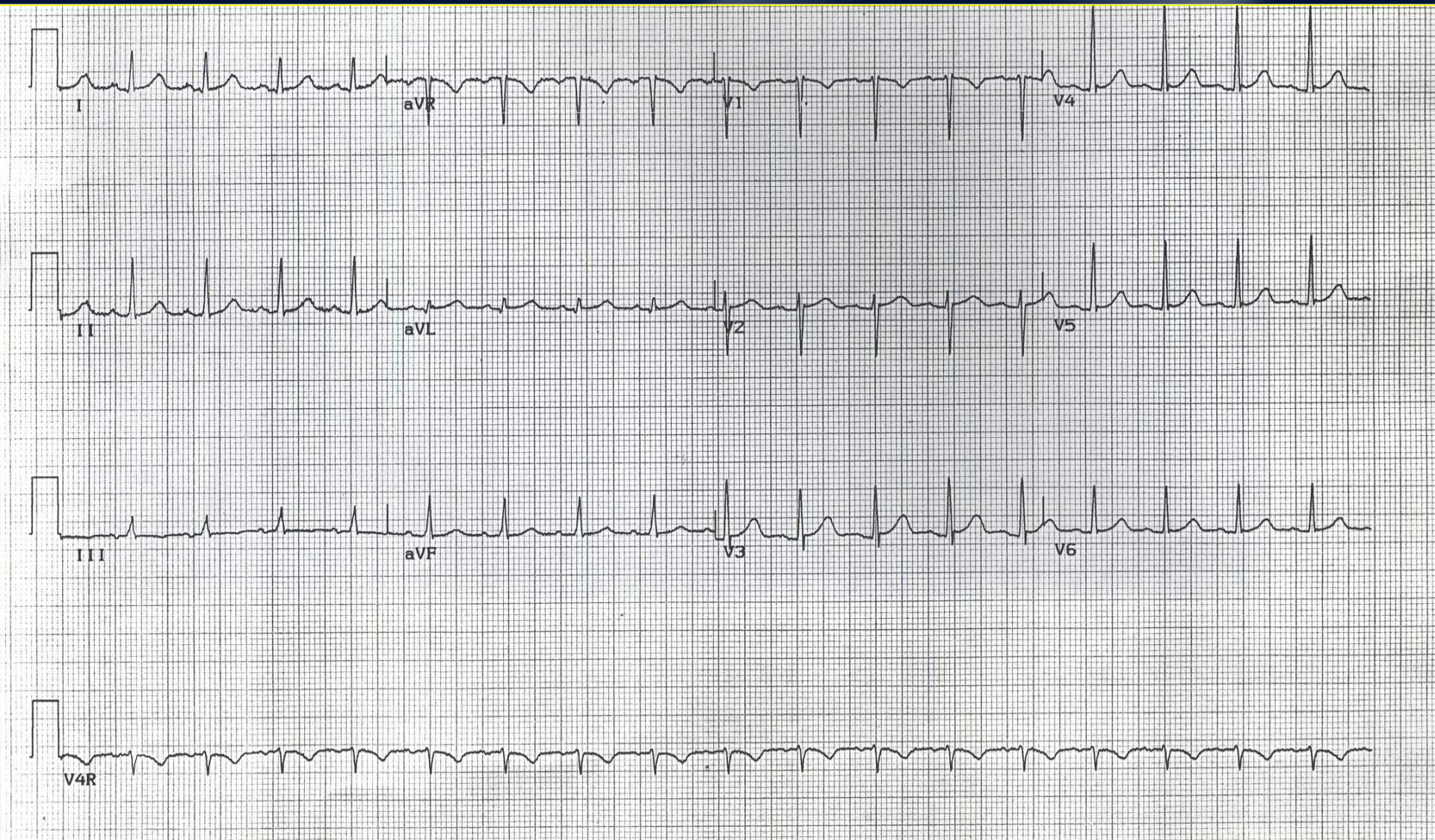

Referred by: 2579 SERWER
Unconfirmed
VISIT #:3302
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 100Hz 005D 12SL 233 CID: 5
EID:Unconfirmed EDT: ORDER:

Determining Heart Rate

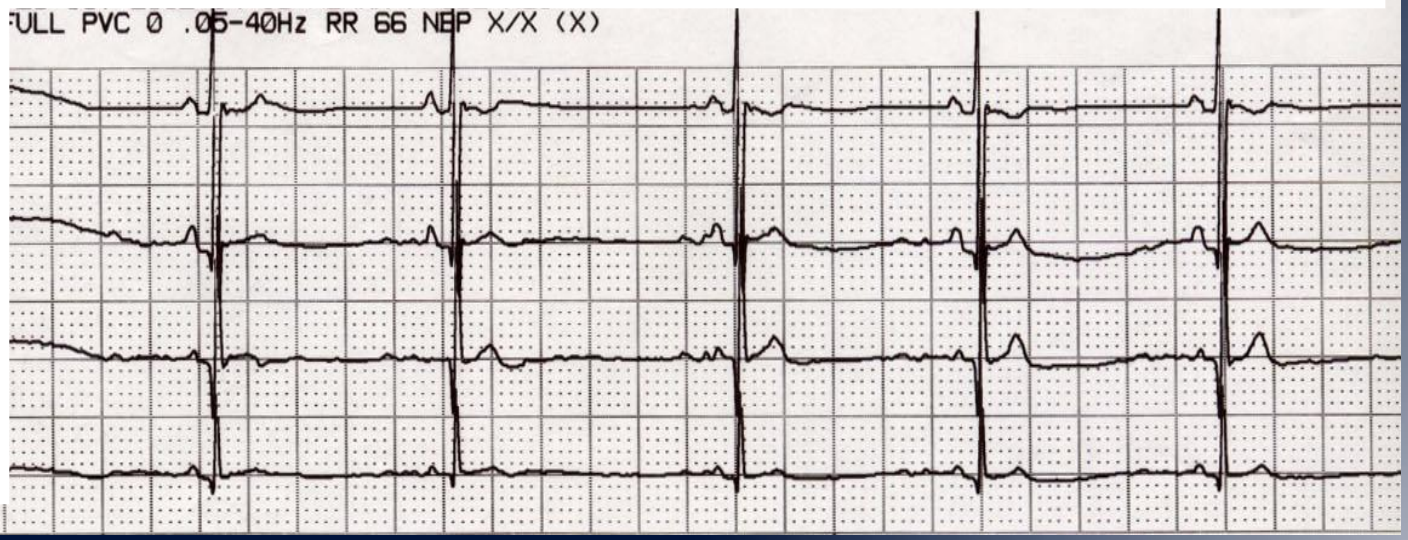
- **One small box = 40ms**
- **One large box = 200ms**
- **One very large box = 1000ms = 1 second**



What's the Heart Rate?



What's the heart rate?



Approach to the ECG

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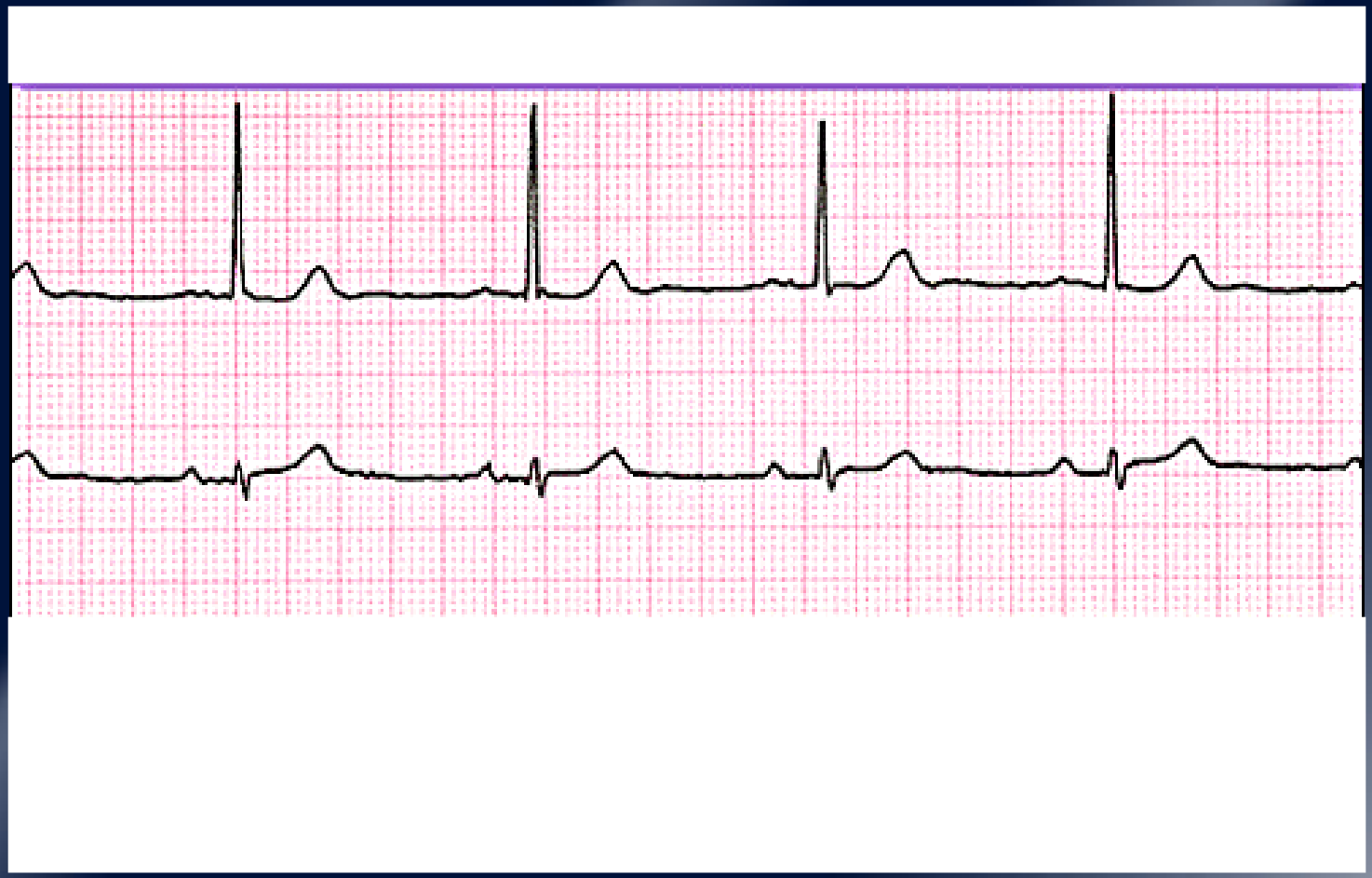
Sinus rhythm Rhythm

- Is there a p-wave before every qrs?
- Is there a qrs after every p-wave?
- Is the p-wave axis normal (sinus rhythm)
- Is the rate fast, slow, or within the normal range ? (age dependent)
- Is the PR interval normal for age? (1st degree AV block)

Coronary sinus rhythm

- In verted p waves in inferior leads
- ie Leads II, III, aVf.
- PR interval is normal unlike in SVT

What's the rhythm?



Approach to the ECG

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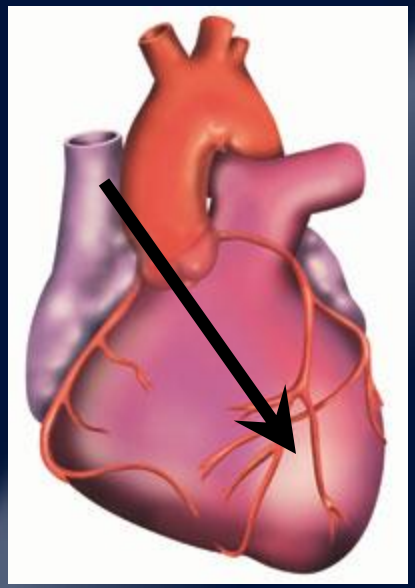
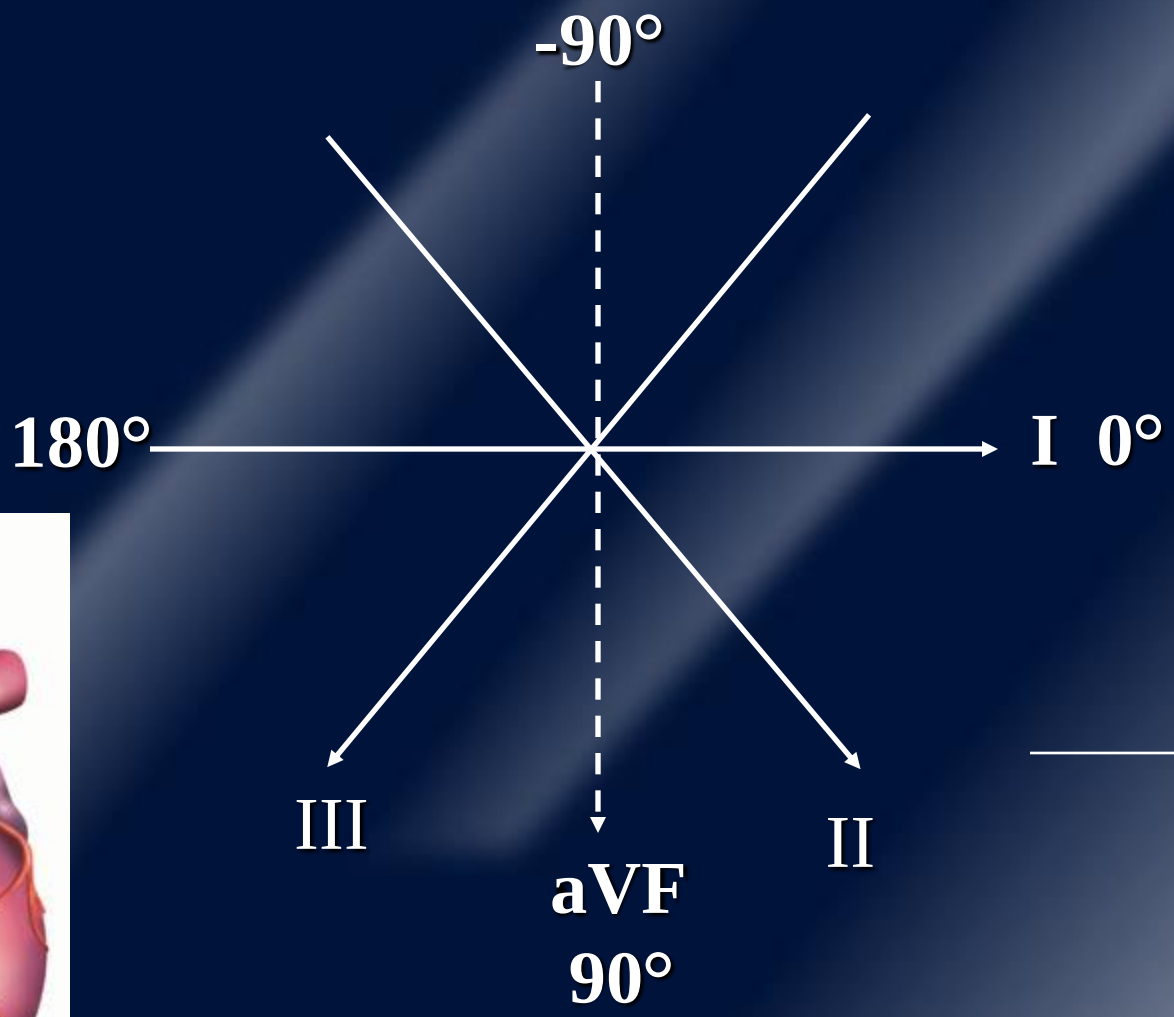
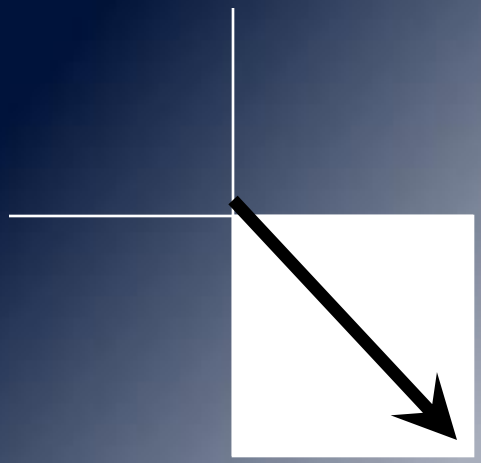
Axis Determination







Axis Determination

-90°

"Northwest Axis"

Left Axis

180°

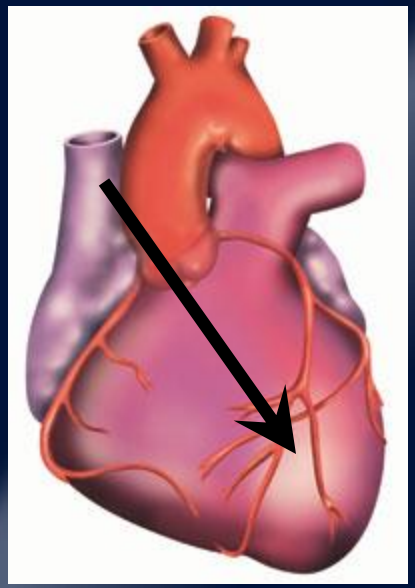
I 0°

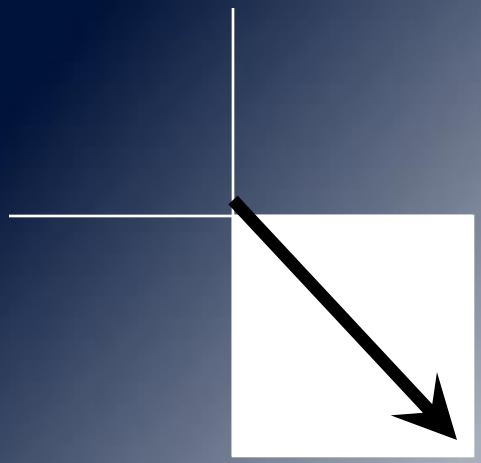
III

aVF

II

Right Axis 90°





AXIS

- **PERPENDCULAR TOLIMB LEAD :R+S=0**
- **PARALLEL TO LIMB LEAD WITH DOMINANT R**
- **OPPOSITE TO LIMB LEAD WITH DOMINANT S.**

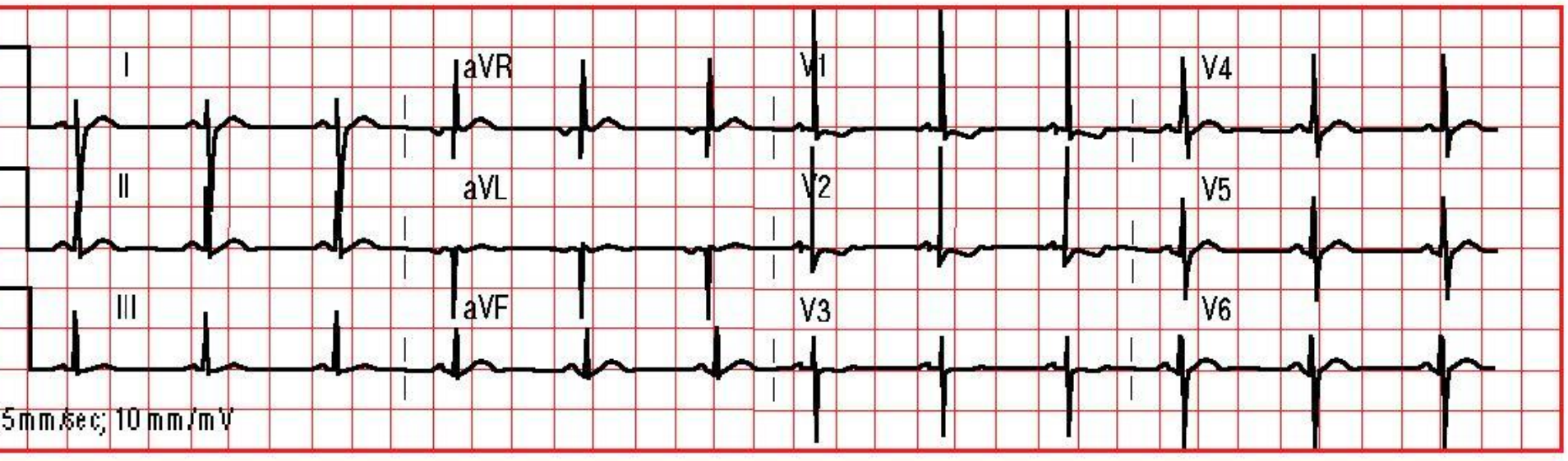
Axis Abnormalities

- **Left Axis Deviation**
  - **Left ventricular hypertrophy**
    - **Aortic stenosis**
    - **Hypertrophic cardiomyopathy**
  - **Defects with minimal right sided forces (tricuspid atresia)**
  - **Atrioventricular septal defect**

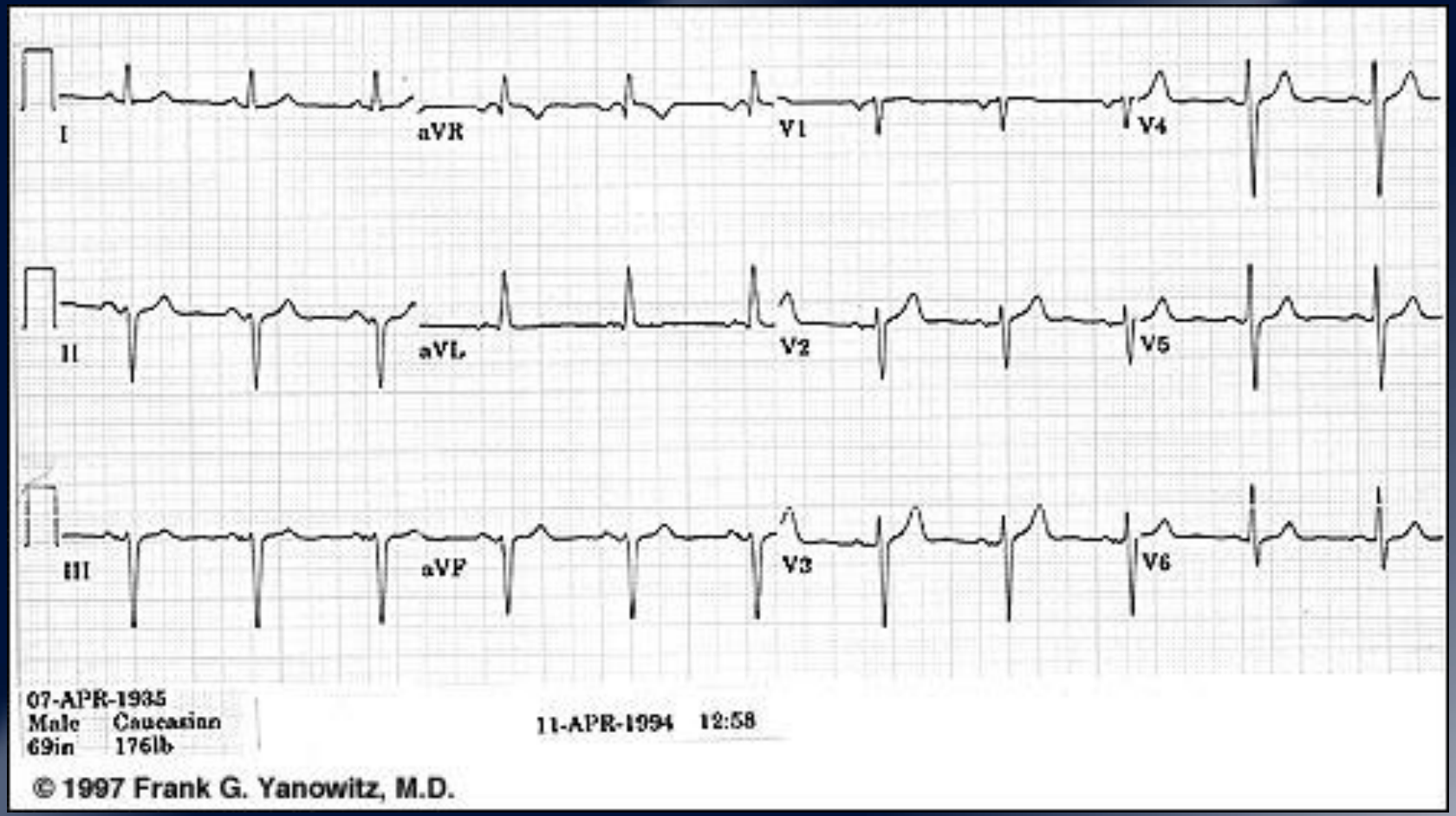
Axis Abnormalities

- **Right Axis Deviation**
  - **Normal newborn**
  - **Right Ventricular Hypertrophy**
    - **Tetralogy**
  - **Minimal Left Sided Forces**
    - **Hypoplastic left heart syndrome**
  - **Systemic Right Ventricle**

What's the axis?



What's the axis?



Approach to the ECG

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Principles of Hypertrophy and Enlargement

- **Chamber enlargement results in a longer depolarization time (wider QRS or P)**
- **Chamber hypertrophy results in greater QRS amplitude**
- **When 1 chamber is affected look for others**
- **When normal conduction pathways are disrupted diagnostic criteria become questionable**

Principles of Hypertrophy and Enlargement

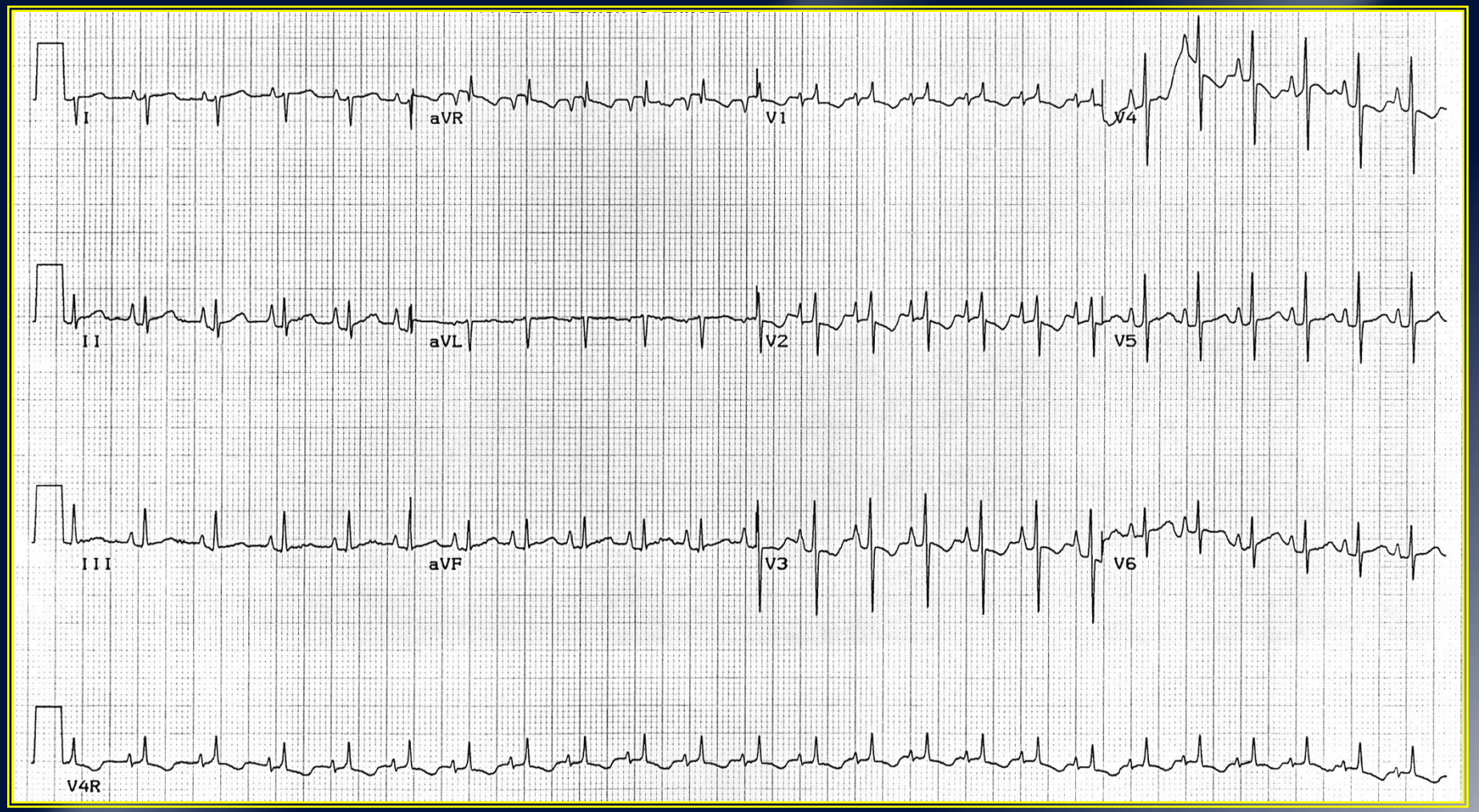
- When cardiac position is altered diagnostic criteria become questionable
- Enlargement is accompanied by hypertrophy
- Hypertrophy is not necessarily accompanied by enlargement
- Always interpret the ECG in light of the clinical circumstances

Atrial Enlargement

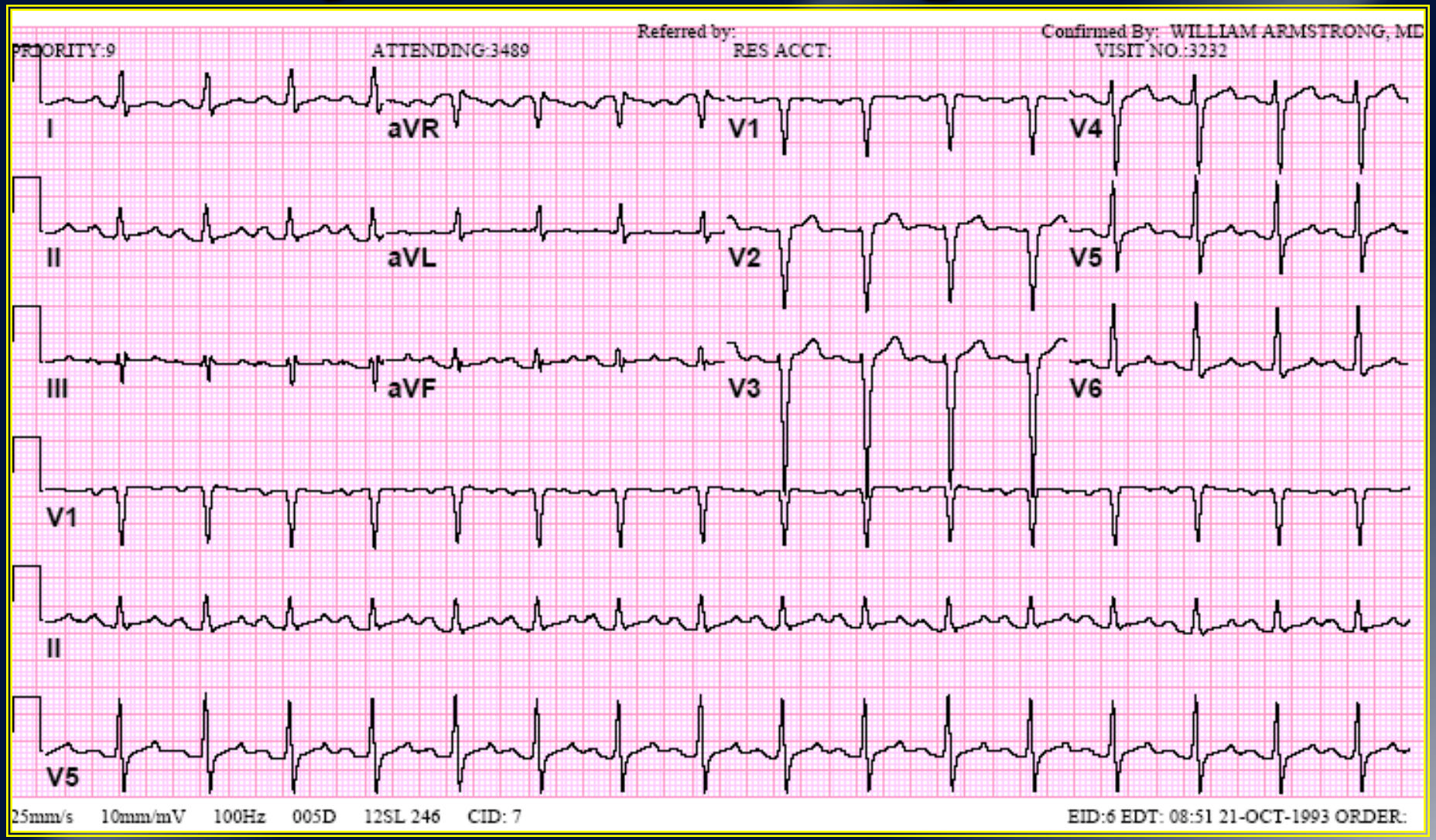
- **Right Atrial Enlargement**
  - **P wave in lead II > 2.5-3.0**

  “p-pulmonale”
- **Left Atrial Enlargement**
  - **Broad P wave in 11, V1**
  - **Broad negative component (more than one small box)**

Right Atrial Enlargement



Left Atrial Enlargement



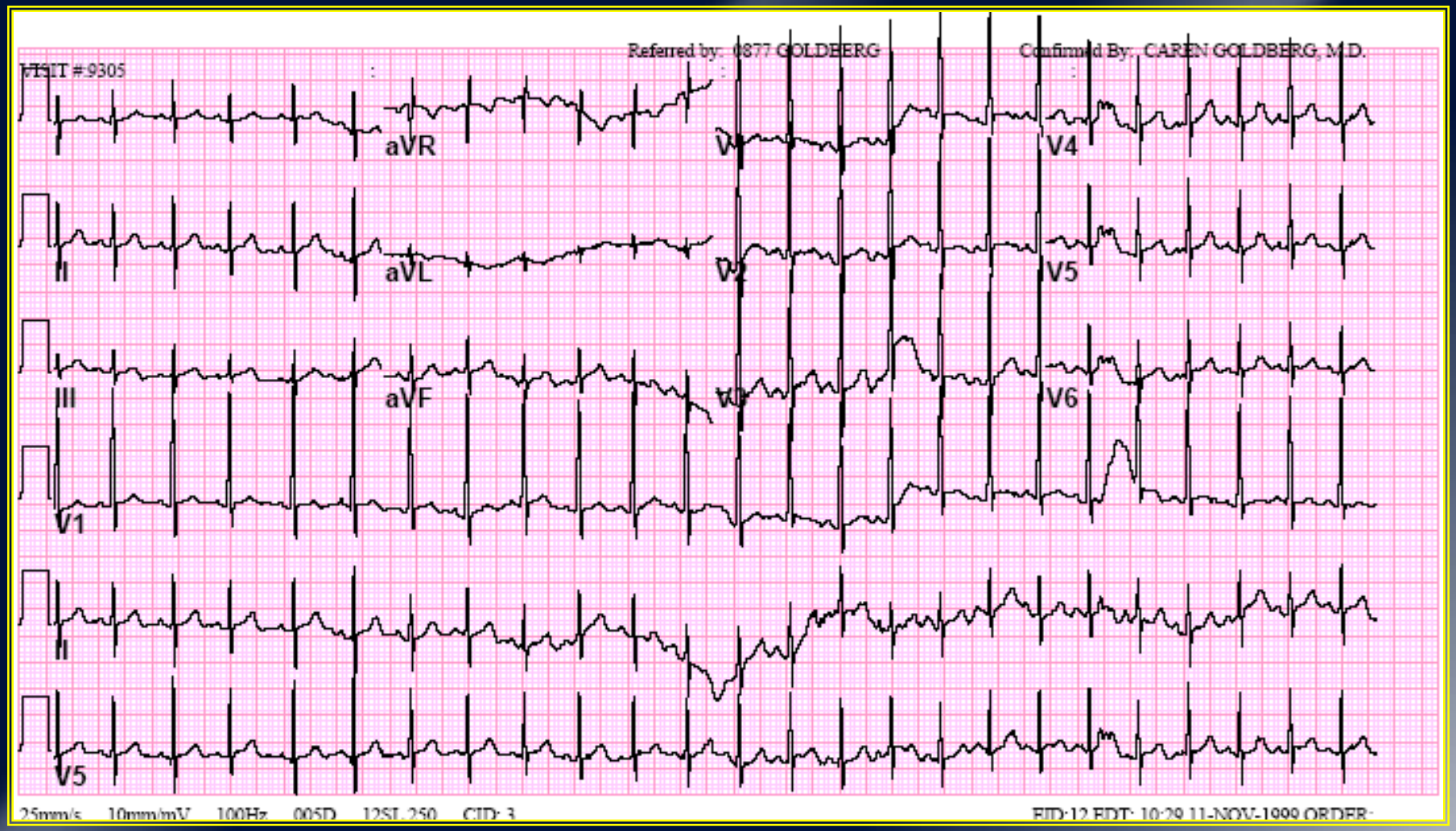
Hypertrophy Summary
Right Sided Chambers

- **Right ventricular hypertrophy**
  - **R > 1 mv in V_1 when age > 18 months**
  - **Upright T in V_1 – 8 days to 8 years**
  - **rsR` pattern in V_1**
  - **qR pattern in V_1**

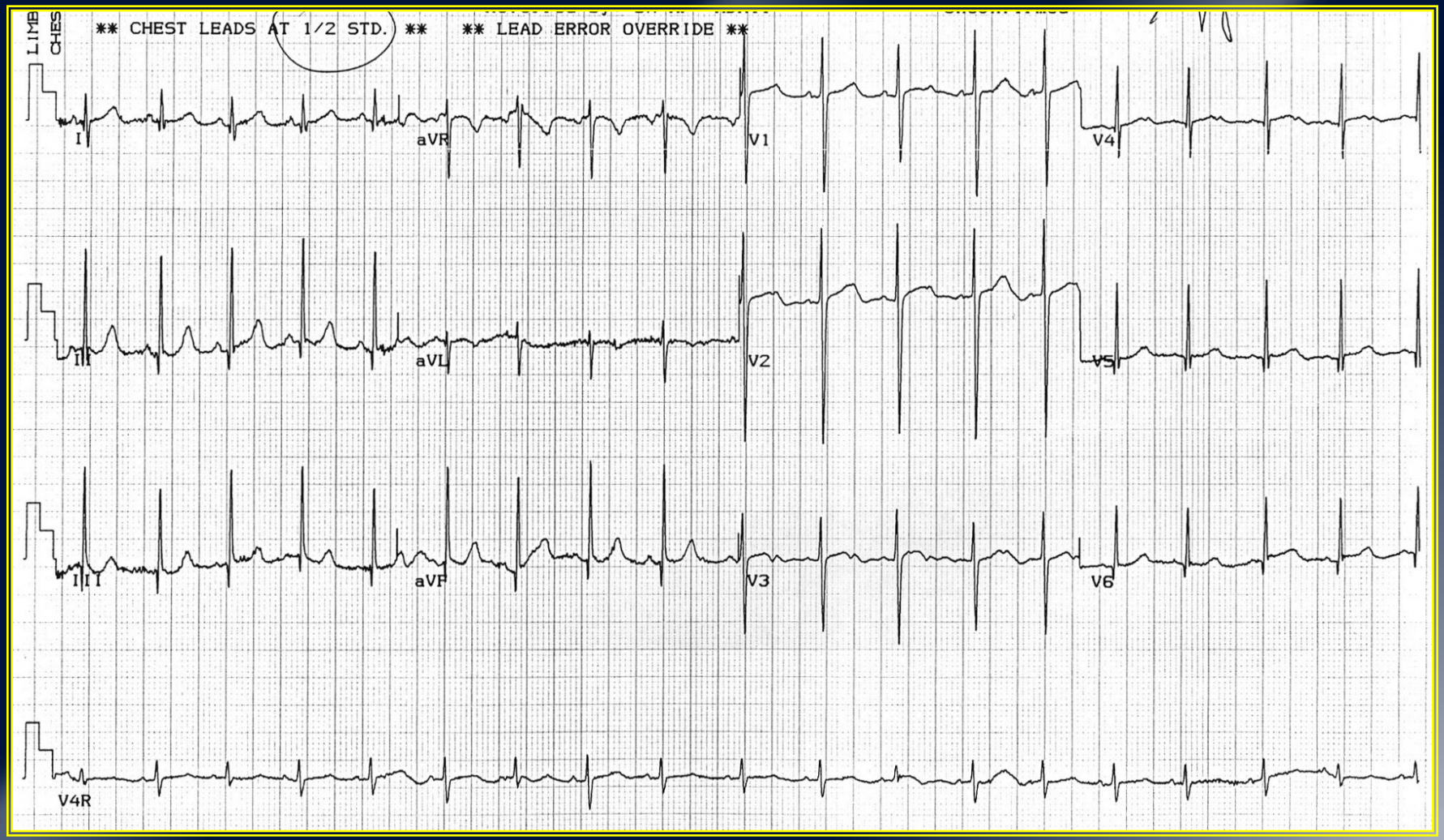
TALL R IN aVR

BOTH V1 AND V2 OVERLIE RV
SO CAN BE USED FOR RVH

Right Ventricular Hypertrophy
Prominent R Wave



Right Ventricular Hypertrophy
Upright T Wave



Hypertrophy Summary
Left Sided Chambers

- **Left atrial enlargement – P wave > 120 msec in duration in any lead**
- **Left ventricular hypertrophy**
- **Age dependent criteria**

  -TALL R IN aVL: =/> 11 mm

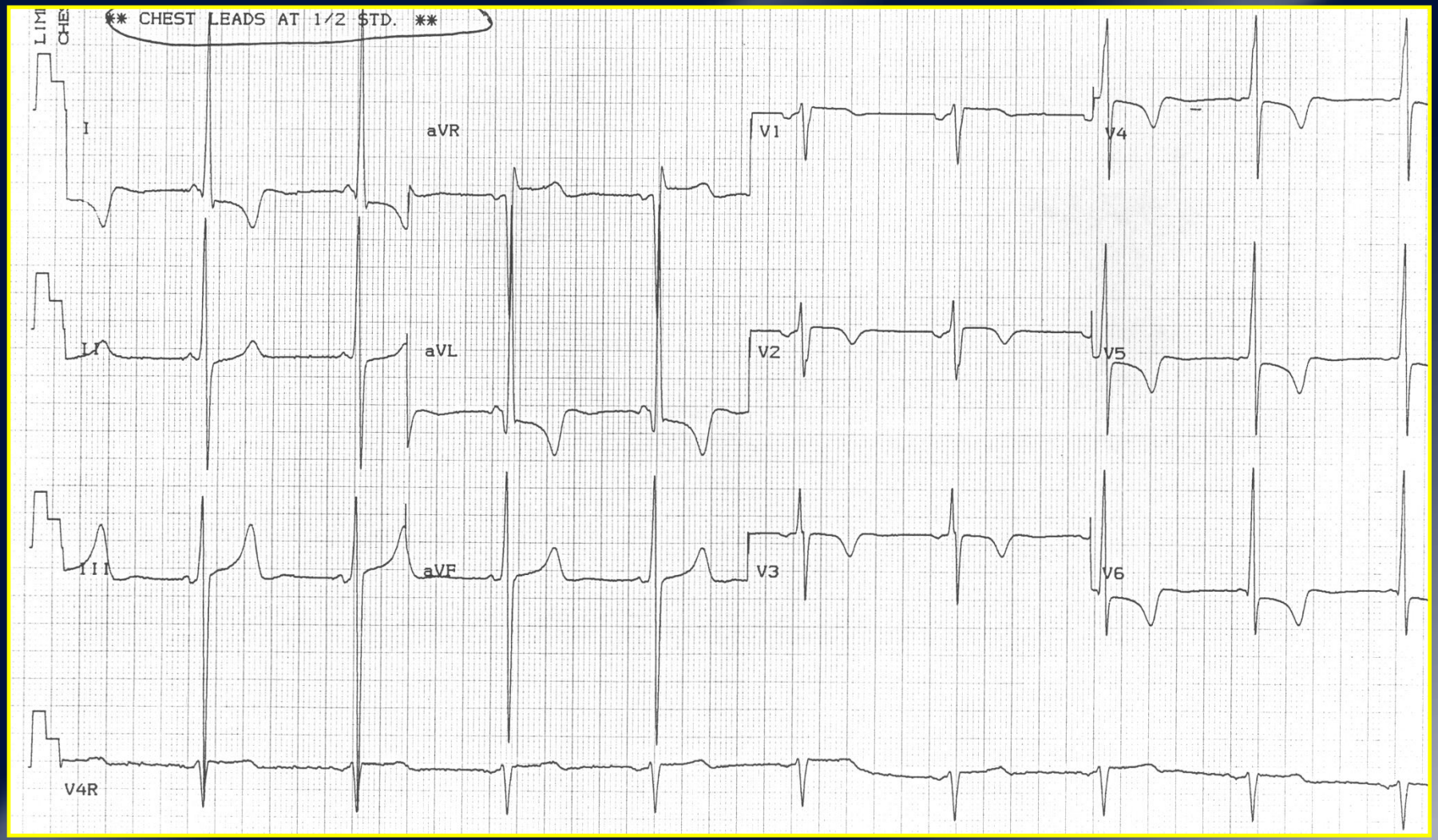
  -Large R in V5-6, Large S in V1-2
- **Left ventricular strain – negative T in V_5 or V_6**

LVH

- S in V1 + R in V5 or V6 (whichever is larger) =/> 35 mm
- R in aVL

-Prominent Q in V6

Left Ventricular Hypertrophy
Hypertrophic Myopathy



KATZ-WACHTEL PHENOMENON

1.COMBINED RVH AND LVH

2.EQUIPHASIC COMPLEXES IN ≥2 LIMB LEADS AND MID PRECORDIAL LEADS

3.SEEN IN VSD, PDA. Etc.

Approach to the ECG

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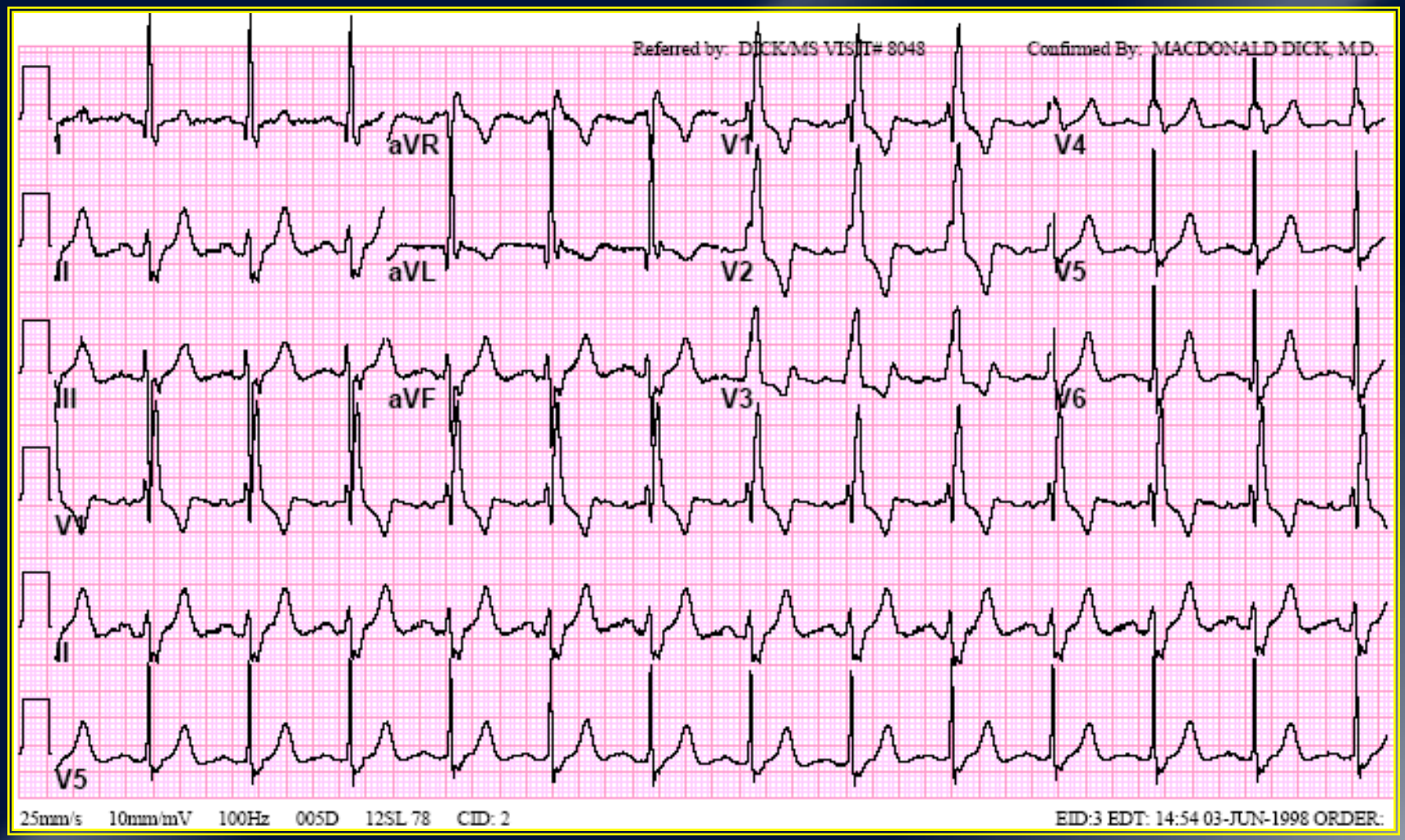
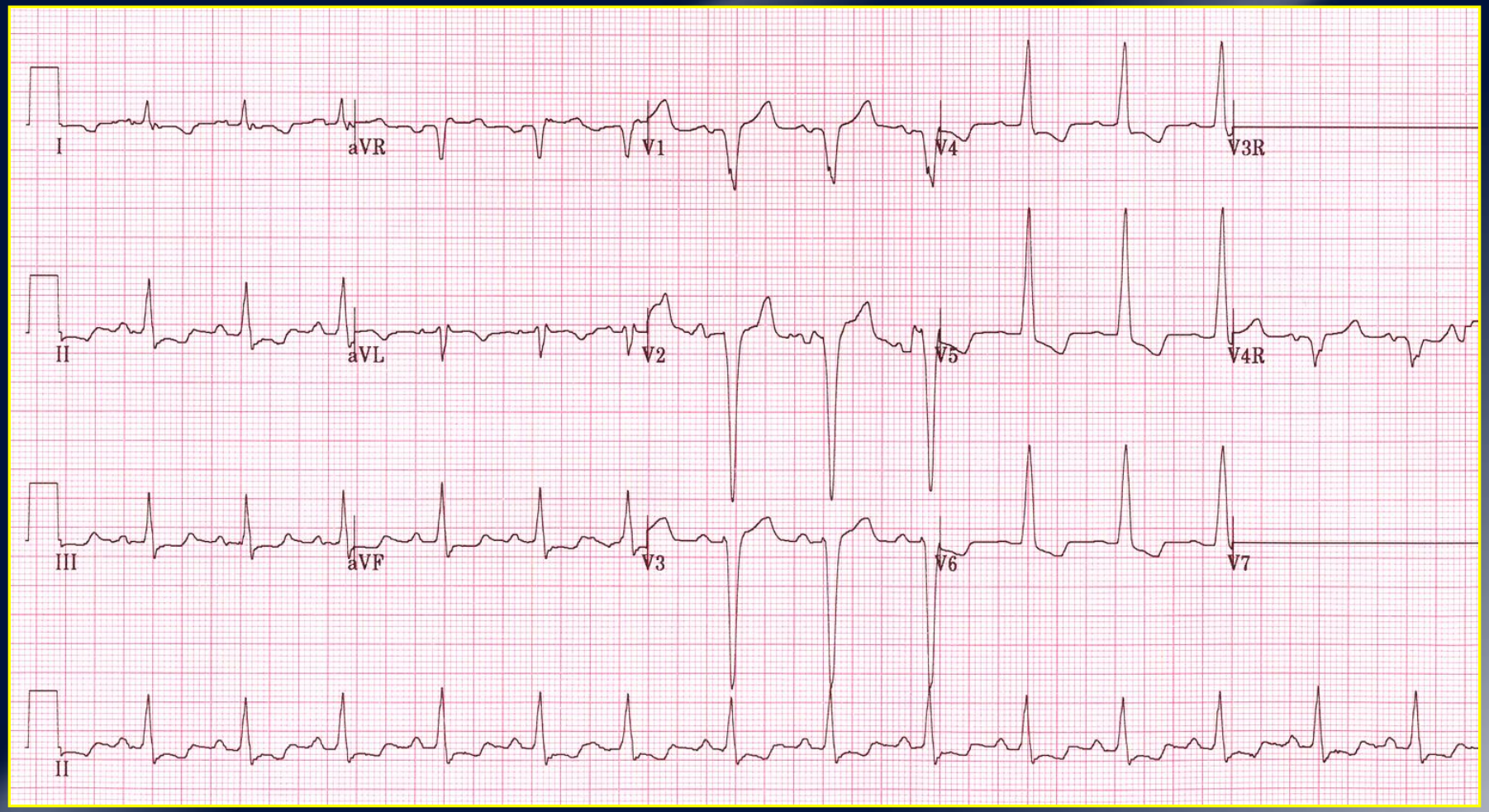
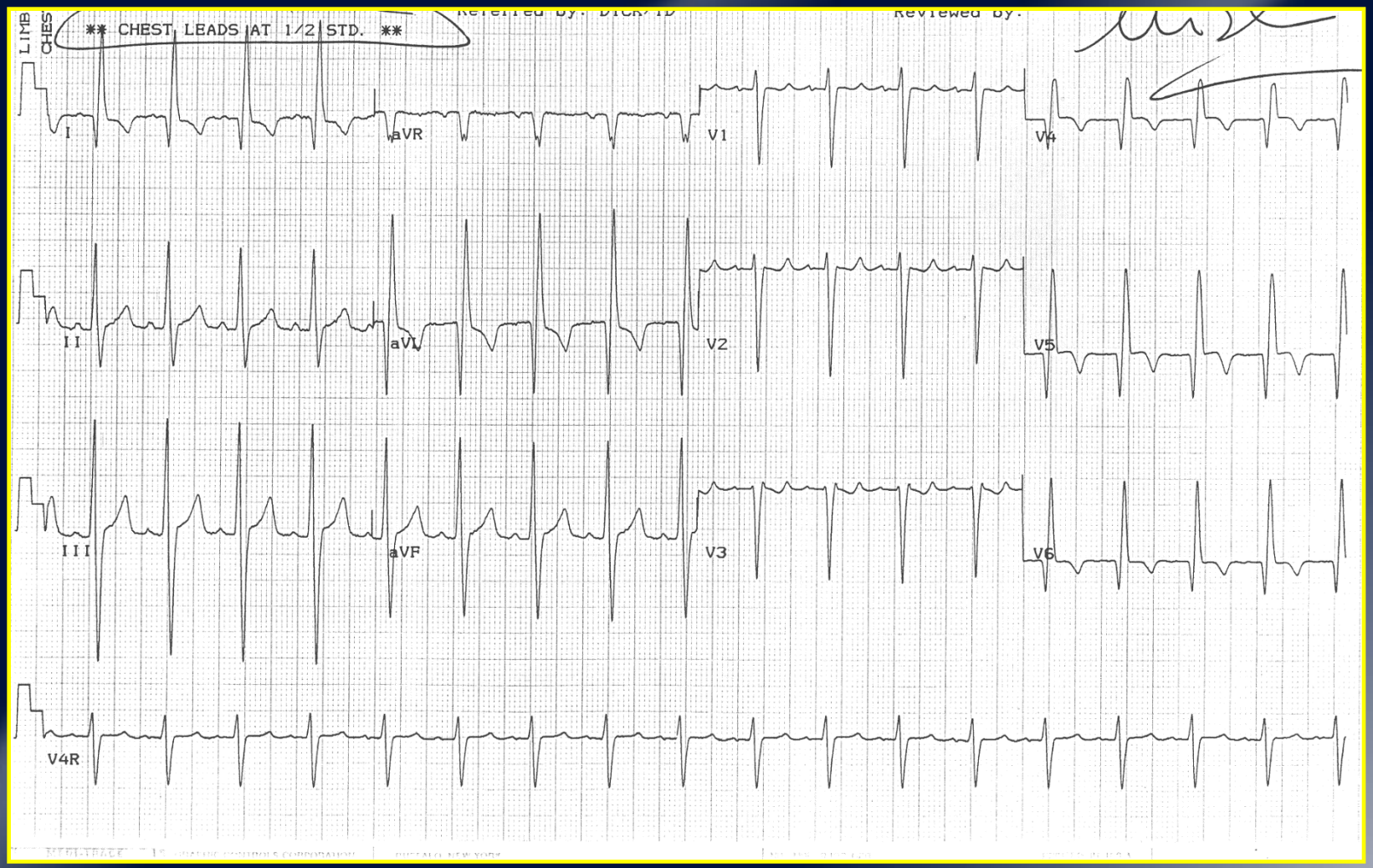
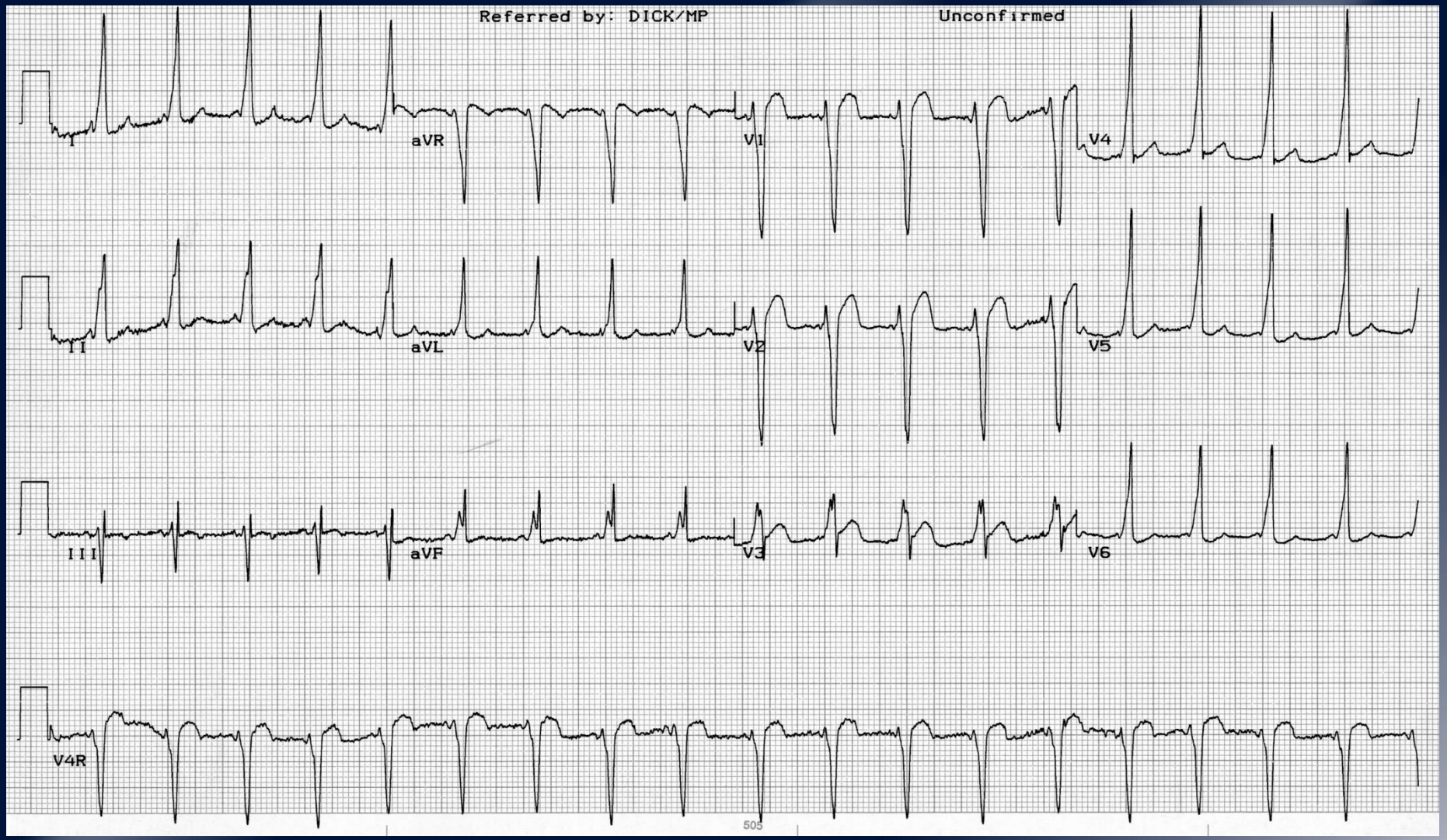
Right Bundle Branch Block



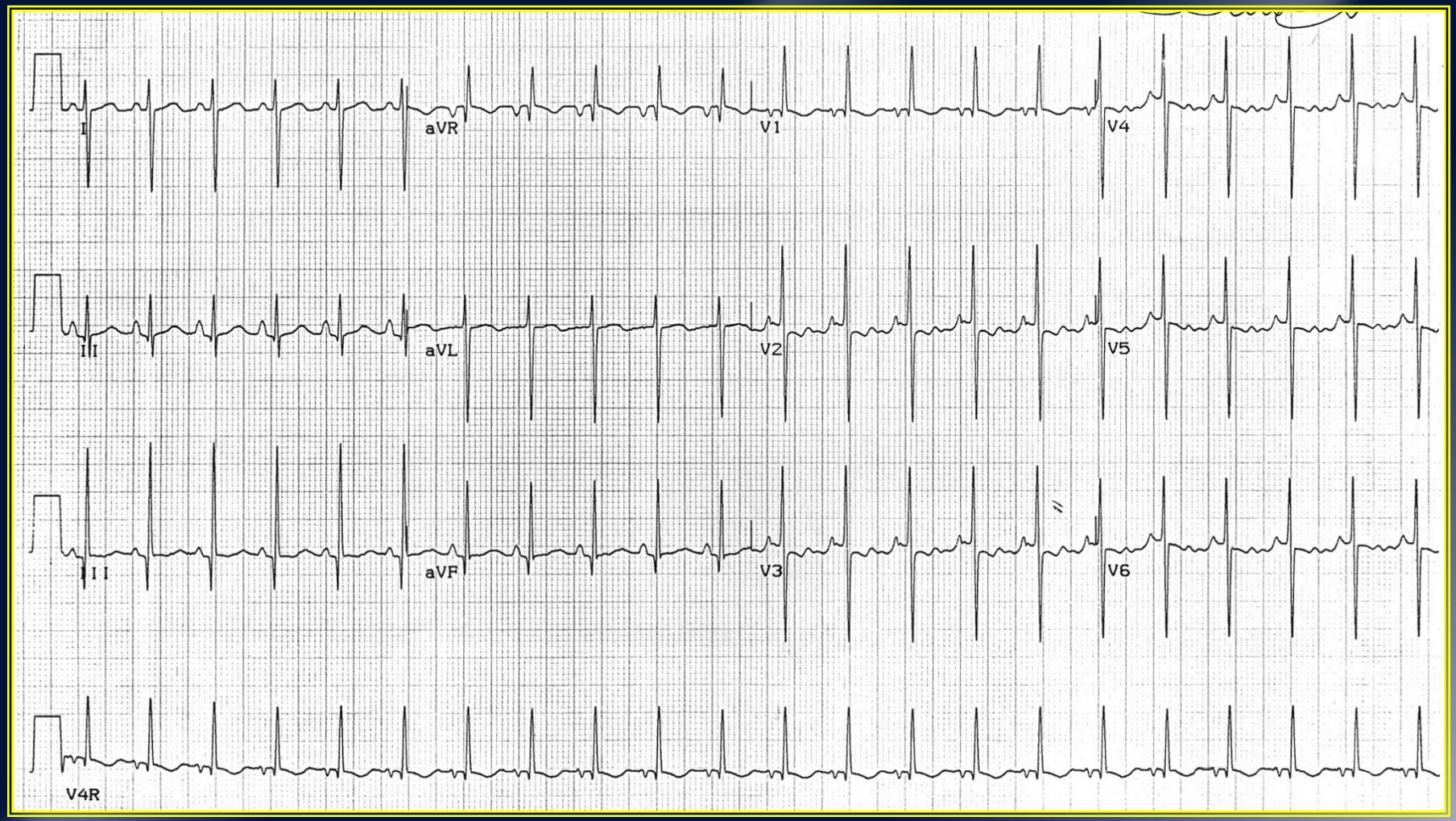
Left Bundle Branch Block




LIMB
CHES
** CHEST LEADS AT 1/2 STD. **
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V4R


Referred by: DICK/MP
Unconfirmed
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V4R
505

Right Ventricular Hypertrophy
qR Pattern



DEXTROCARDIA

P, QRST AND T WAVES INVERTED IN LEAD 1

- **THANK YOU**

ABNORMAL RHYTHM

- Rhythms other than regular sinus rhythm
- Arrhythmias are primarily classified according to their rate
- Usually the atria and ventricles have the same rates

ORIGIN OF RHYTHM

- **If atrial and ventricular rhythms are associated and have the same rates then**
- **Rhythm originates in the atria or ventricular**

- **If atrial and ventricular rhythms are associated but atrial rate is faster than ventricular rate then**
- **Rhythm originates in the atria**

SINUS RHYTHM

- P wave before each QRS complex
- Normal P-R interval
- P wave axis 0-90
- P wave upright in Leads 1 and aVf

- If atrial and ventricular rhythms are associated but ventricular rate is faster than atrial rate then rhythm originates in the ventricles

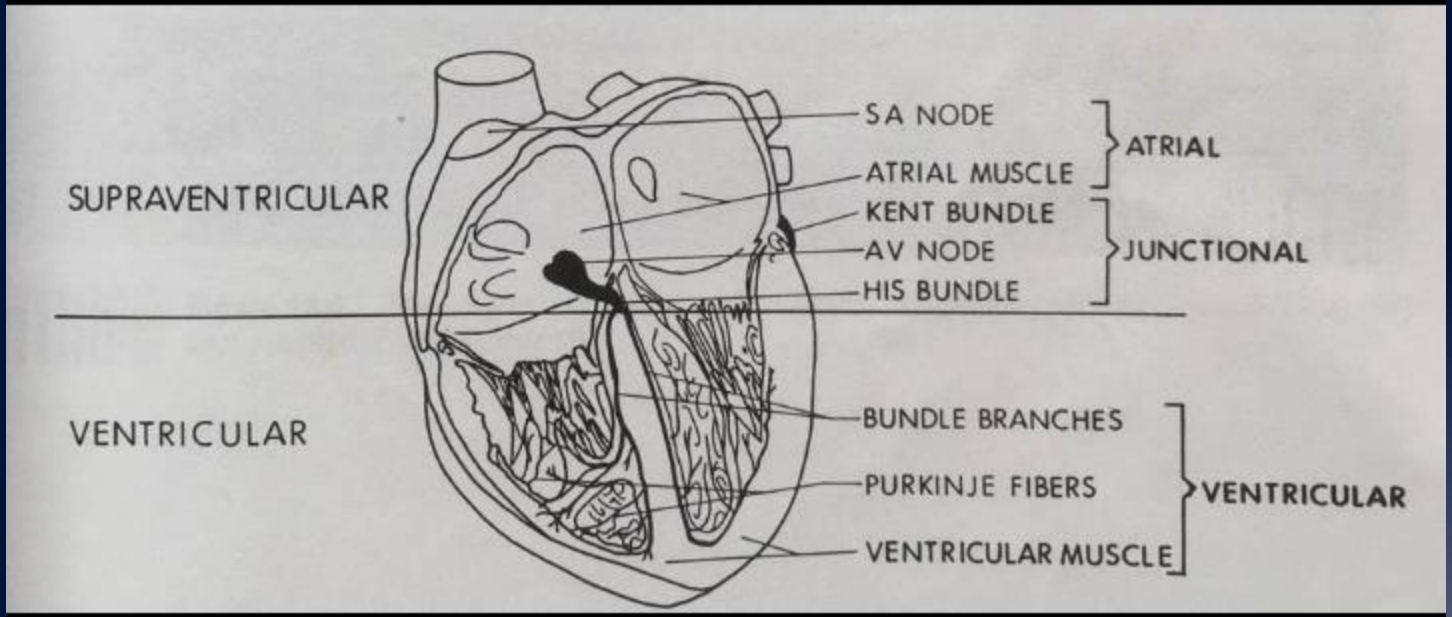
- If atrial and ventricular rhythms are not associated then there is AV dissociation.

MECHANISMS PRODUCING ARRHYTHMIAS

- **Automaticity, ie problems of impulse formation**
- **Block or re-entry, ie problems of impulse conduction**

AUTOMATICITY

- These originate from pacemaker cells which include
- SA node
- Purkinje cells
- Common His bundle
- Right and left bundle branches and


SUPRAVENTRICULAR
VENTRICULAR
SA NODE
ATRIAL MUSCLE
ATRIAL
KENT BUNDLE
AV NODE
HIS BUNDLE
JUNCTIONAL
BUNDLE BRANCHES
PURKINJE FIBERS
VENTRICULAR MUSCLE
VENTRICULAR

- Supraventricular arrhythms include those from
- SA node
- Atrial muscle
- AV node
- His bundle

- Ventricular arrhythmias include
- Bundle branches
- Purkinje fibres
- Ventricular muscle

IMPORTANCE OF QRS COMPLEX

- **An extension of the Willie Sutton law**
- **Sutton robbed banks because that is where the money was**
- **The behaviour of the QRS is what matters at the end despite what the atria are doing**

- **In tachyarryhthmias, if QRS of normal duration in at least two leads the rhythm is supraventricular(SVT)**

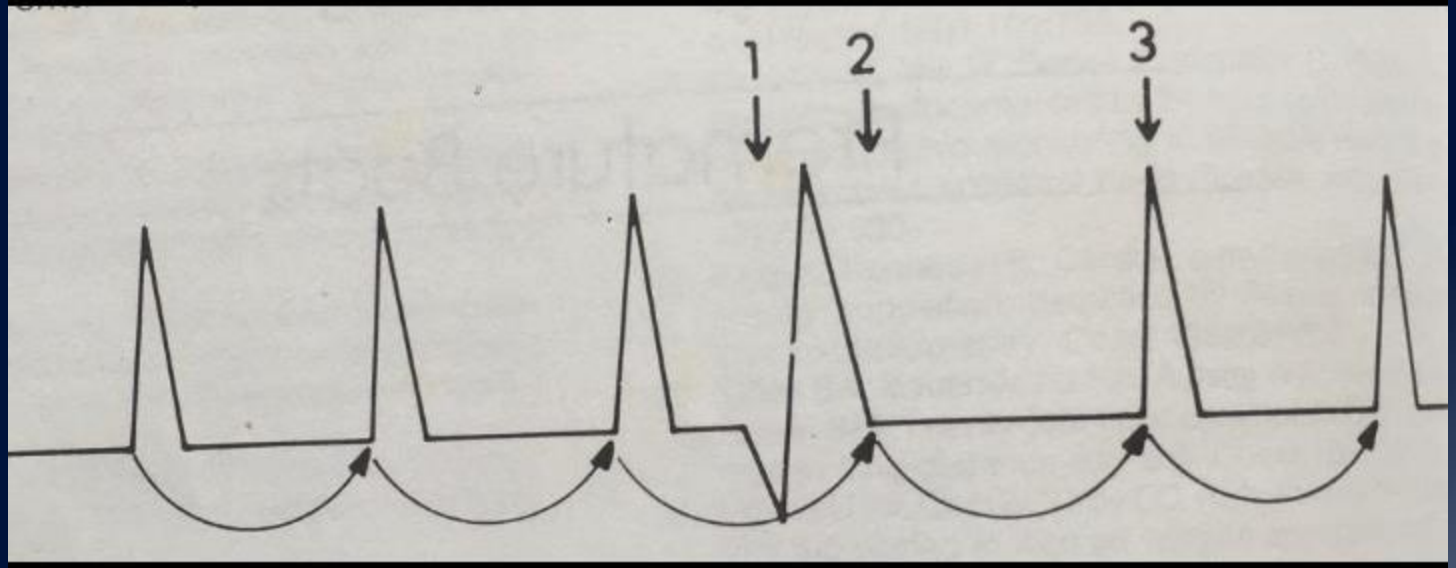
- **Wide and bizarre QRS means it is either SVT with ventricular aberration or ventricular tacchyarrhthmia**

PREMATURE BEATS(PB)

- **Normal sinus rhythm commonly interrupted by premature beat(PB)**
- **The PB itself does not cause symptoms but a palpitation may be felt following the next normal heart beat**
- **There is a pause following the PB until the next normal beat**

PREMATURE BEATS

- **May originate from supraventricular, which includes SA NODE, ATRIAL MUSCLE, AV NODE OR HIS BUNDLE**
- **May also be from Ventricular origin, ie BUNDLE BRANCHES, PURKINJE FIBRES, VENTRICULAR MUSCLE**


1
2
3

- The timing of normal rhythm is indicated by the curved lines with arrows
- A ventricular premature beat interrupts the rhythm indicated by (1)
- This prevents occurrence of the next normal beat(2).
- The next normal beat(3) occurs at the normal time

SINUS TACCHYCARDIA

- SA node is regulated by both parasympathetic and sympathetic systems
- Any flight or fright condition leads to sympathetic activation in the body
- There is no pathologic cardiac condition
- Therefore treatment is correcting the condition leading to sympathetic activation rather drugs to suppress the SA node

- Common conditions leading to sympathetic activation includes stress and anxiety, anaemia, shock
- BP:CO × Peripheral vascular resistance
- CO:Stroke volume × heart rate

ECG FEATURES SINUS TACHYCARDIA

- **Maximal stimulation of SA NODE by sympathetics is 220/min and rarely 160/min in non exercising adults**
- **Normally P wave before QRS.**
- **Shorter PR interval than normal, since the increased sympathetic tone also affects the AV nodal conduction**
- **QRS complex is normal in morphology**

SUPRAVENTRICULAR TACHYCARDIA(SVT)

- **Atrial tachycardia**
- **Atrioventricular nodal tachycardia**
- **Atrioventricular re-entry tachycardia**

AVNRT

- Additional conduction from atria to ventricles
- This additional path involves the AV node
- An antegrade path since from atria to ventricles

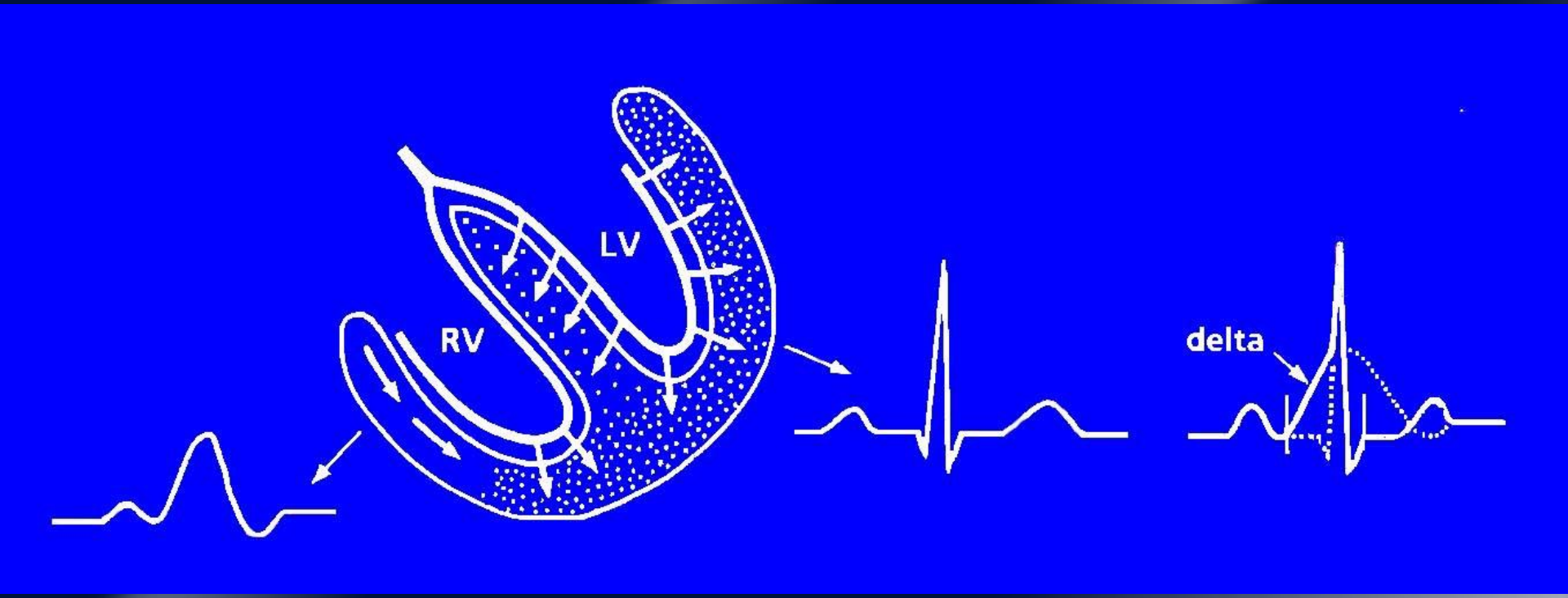
AVRT

- Constitute about 30% of SVT
- Re-entry does not involve the AV node
- Usually a retrograde conduction from ventricle-atria-ventricle

AVRT

- **The first activation of ventricle is premature .**
- **This is followed by normal activation, thus prolonging contraction of ventricles.**
- **This produces a wide QRS complex called delta wave eg WPW syndrome**

Wolff-Parkinson-White Syndrome



HISTORY

- **In 1893, Kent had described muscular connection s between atria and ventricles but wrongly assumed they were normal connections**
- **In 1930 Wolf and White in Boston and Parkinson in London published ECG's on 11 patients with bizarre QRS complexes and short PR interval**
-

- In 1914 Mines suggested that this bundle of Kent may mediate re-entry tachycardias
- Finally in the same year Segers connected the short PR interval, widened QRS complex into WPW syndrome
- Mediated by the bundle of Kent. He termed the QRS complex delta wave

SUPRAVENTRICULAR TACHYCARDIA(SVT)

- **Rates between 250-300/min**
- **Rates More than 230/min unlikely to be sinus rhythm**
- **P waves visible in about 60%**
- **P wave axis is abnormal**

ATRIAL AUTOMATICITY

- **This about 10% of SVT**
- **There is re-entry within the atria itself**
- **This leads to atrial re-entry circuit**

ATRIAL FLUTTER

- **Instead of P waves there are sawtooth flutter waves at a rate of 300-600/min**

MANAGEMENT OF SVT WITH COLLAPSE

- **DC CARDIOVERSION**

SVT WITH STABLE CVS STATUS

- **Vagal manouvres, icepacks on face carotid masssage**
- **Management involves blocking the AV node with adenosine or digoxin**
- **Blocking the accessory path with flecainide**
- **Maintain with drugs**

VENTRICULAR ARRHYTHMIAS

- **Includes Ventricular premature beats or ventricular extra systoles**
- **Premature ≥QRS, or prolonged QRS(0.08 sec)**
- **Abnormal QRS MORPHOLPGY**
- **Absent preceding P waves**
- **If frequent may lead to VT**

VENTRICULAR TACHYCARDIA(VT)

- **Defined as 3 or more successive beats of ventricular origin at rate more than 120/min**
- **Stable CVS status: GIVE LIGNOCAINE**
- **If CVS compromise DC cardioversion**
- **Eg is Torsades de pointes:sinusoidal polymorphic QRS complexes**

VENTRICULAR FIBRILLATION

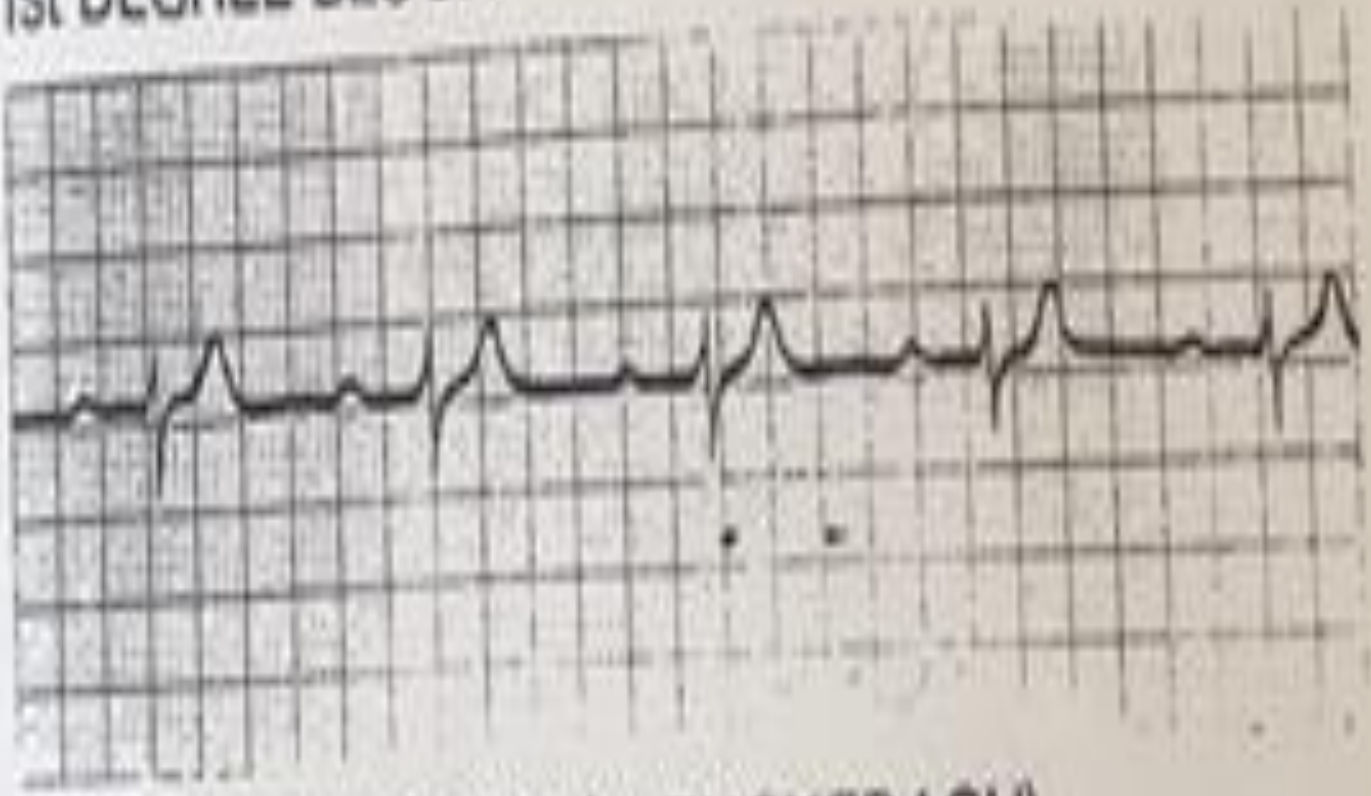
- **Bizzare QRS complexes of varying sizes and shapes**
- **Rapid rates and irregular**
- **Treatment is defribillation**

Coronary sinus rhythm

- Inverted p waves in inferior leads
- ie Leads II, III, aVf.

- PR interval is normal unlike in SVT

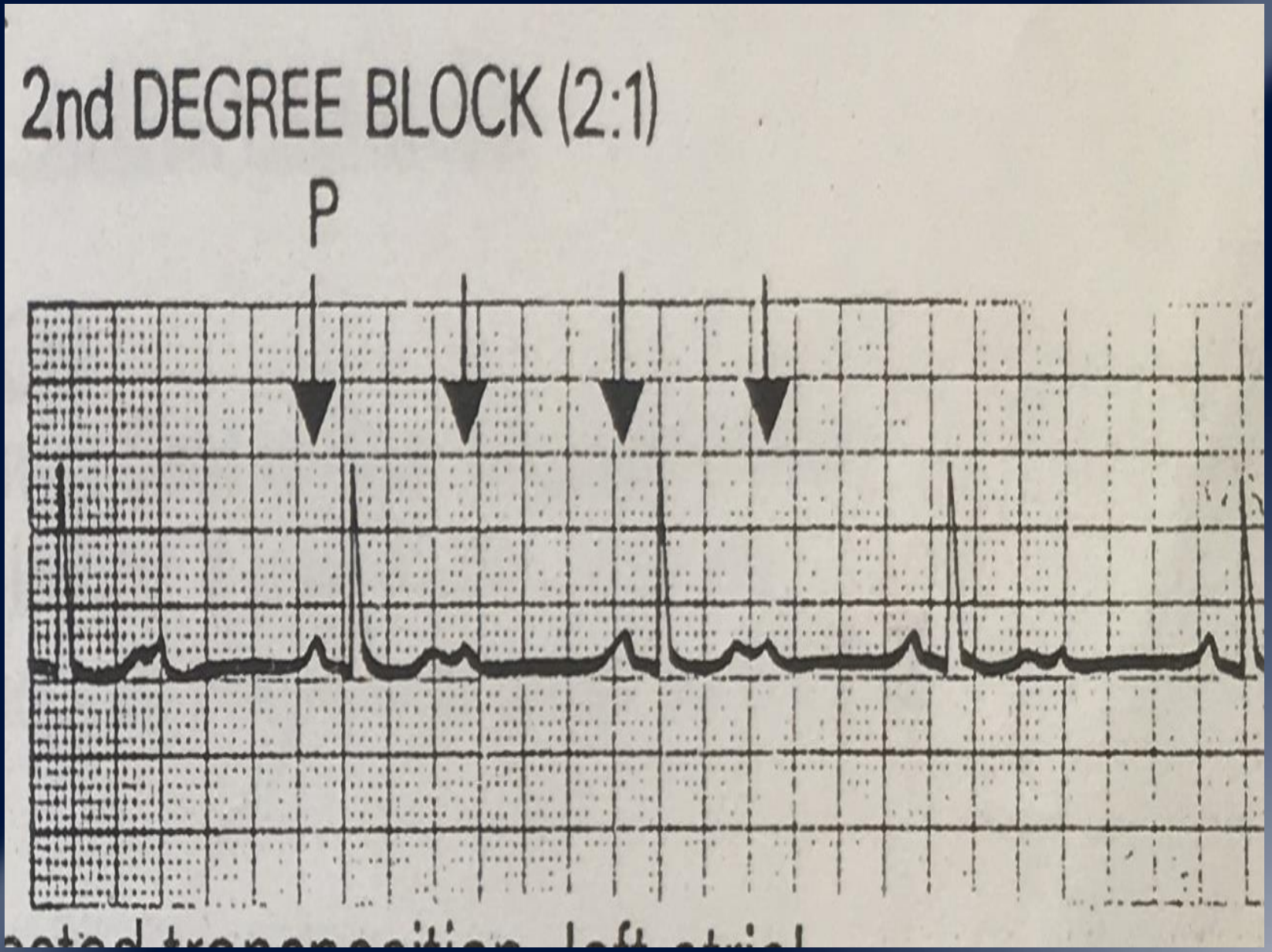
1st DEGREE BLOCK



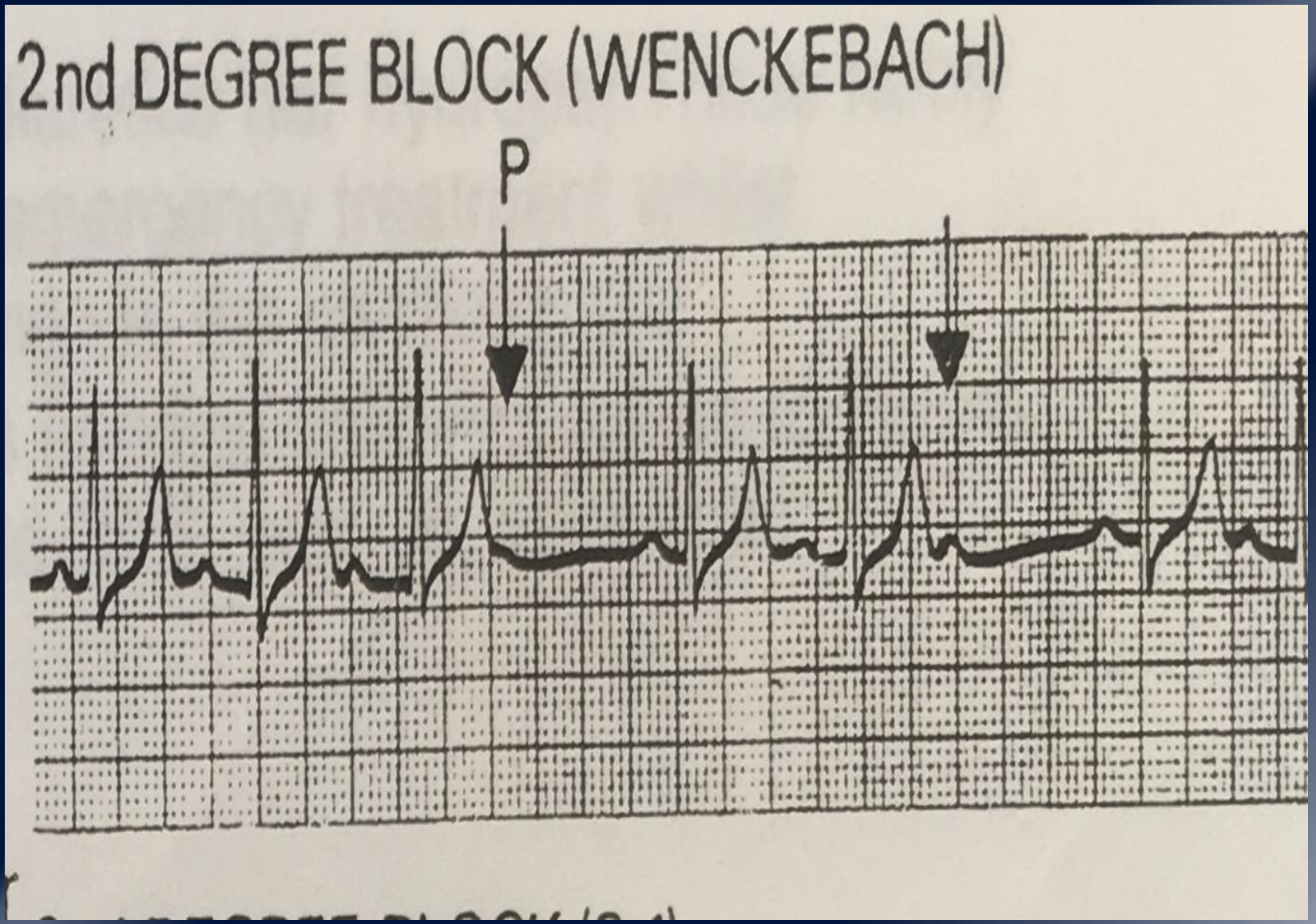
2nd DEGREE BLOCK (WENCKEBACH)

P

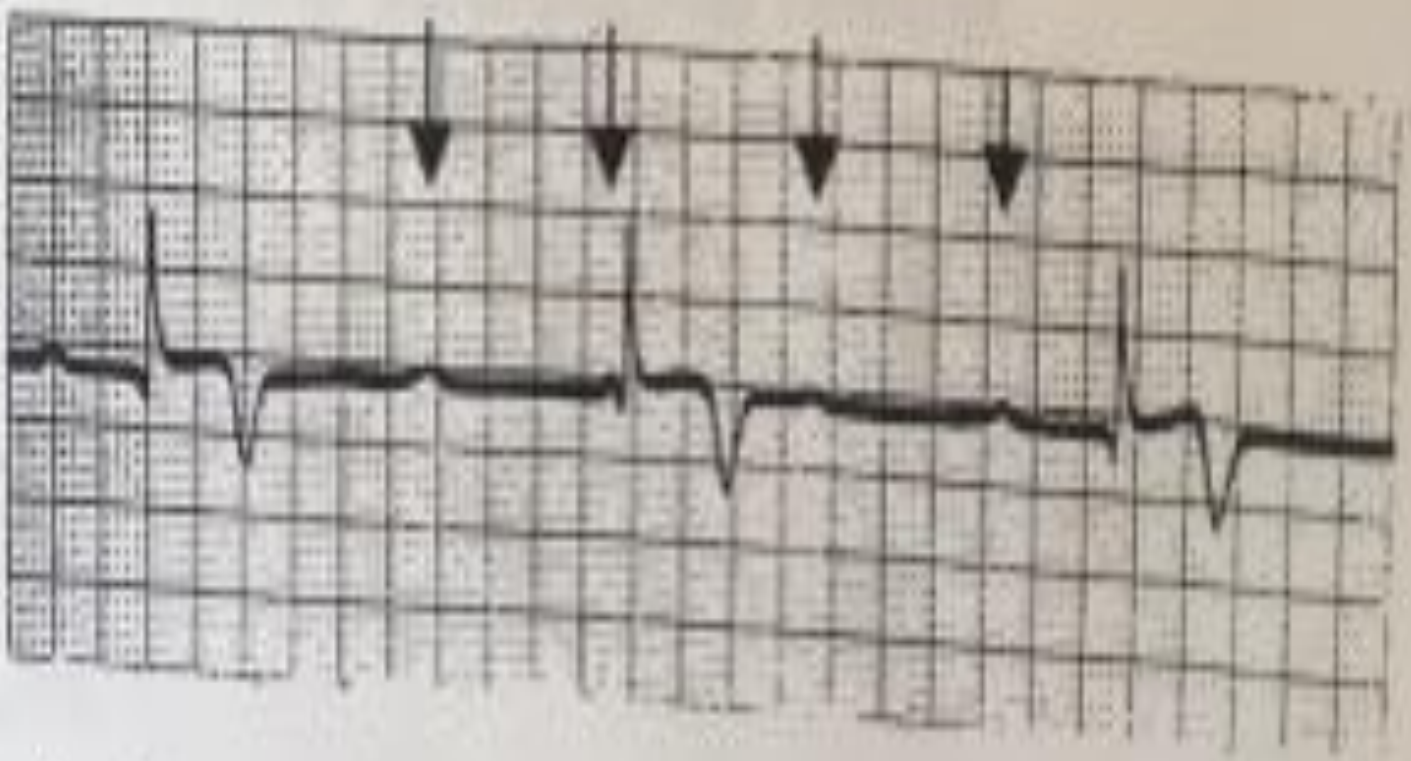
2nd DEGREE BLOCK (2:1)



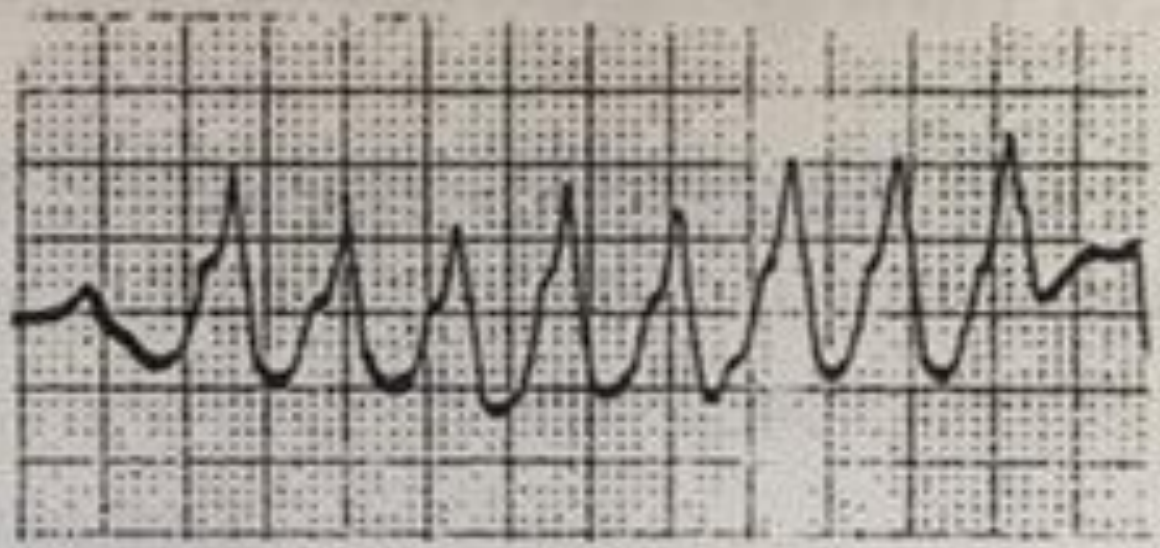
2nd DEGREE BLOCK (WENCKEBACH)



COMPLETE (3rd DEGREE) BLOCK



VENTRICULAR TACHYCARDIA



SUPRAVENTRICULAR TACHYCARDIA