Class 6 – SOLAECE Course

Normal P wave, in atrial enlargements, artifacts, dextrocardia and interatrial blocks

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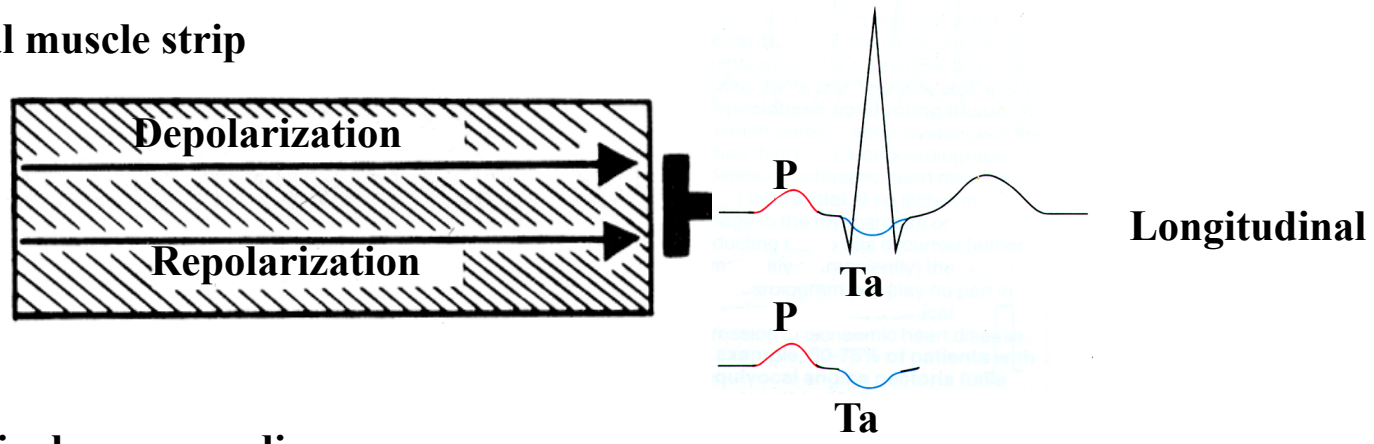
The authors do not report any conflict of interest regarding this presentation

1. Introduction

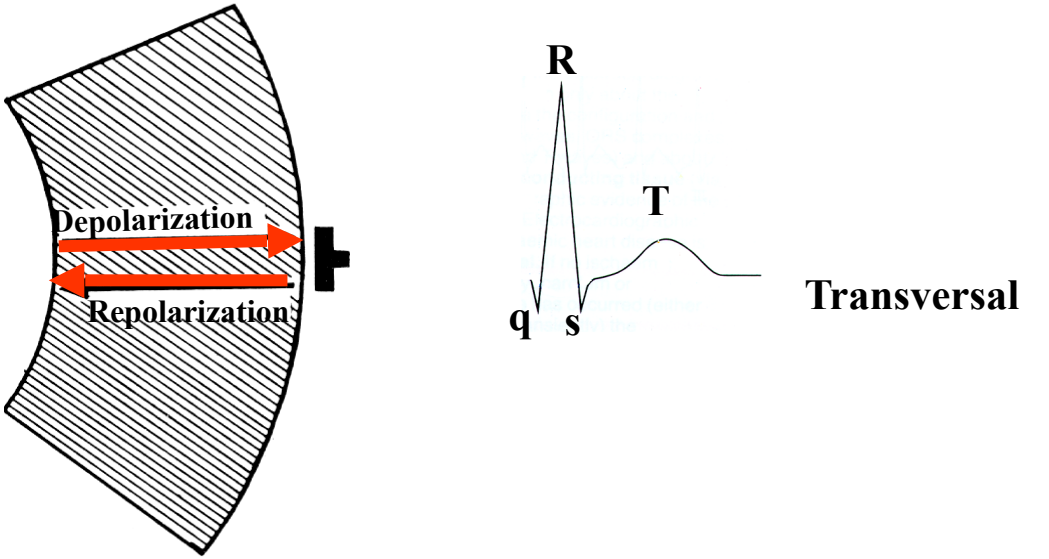
For some authors the name “atrial abnormalities” encompass the concept of atrial enlargement and atrial blocks (**Bayés de Luna 2013**)(**Lee 2007**). In this work we will expose the ECG/VCG characteristics of both concepts, emphasizing, as it happens with ventricular hypertrophy and ventricular blocks, that often the ECG/VCG pattern of atrial enlargement especially of left atrial enlargement (LAE) is explained by the coexistence of interatrial block (IAB). However as we will see later there are clear evidences that the pattern of IAB may exist without the association of atrial enlargement/hypertrophy. Therefore atrial blocks and atrial enlargement are separate entities that are often associated with each other. Therefore we do not consider it appropriate to use the “umbrella” term atrial abnormalities to include both concepts without distinguishing between them (**Lee 2007**), as some authors do (**Bayés de Luna 2013**:) (**Tsao 2008**).

Depolarization and repolarization mode of the atria and ventricles

A) Atrial muscle strip

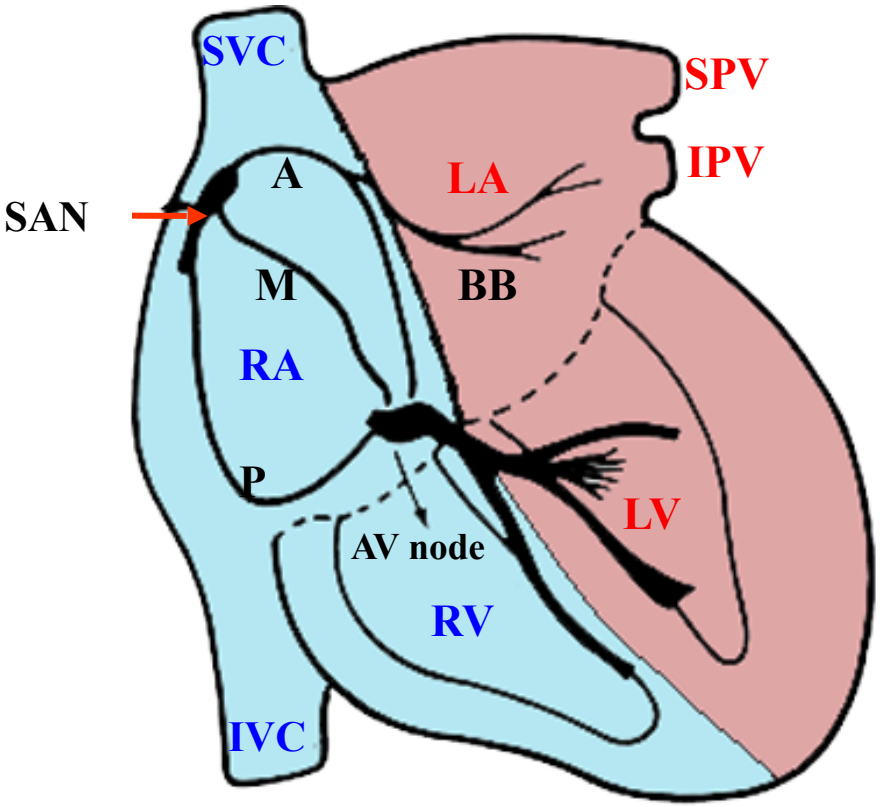


B) Ventricular myocardium

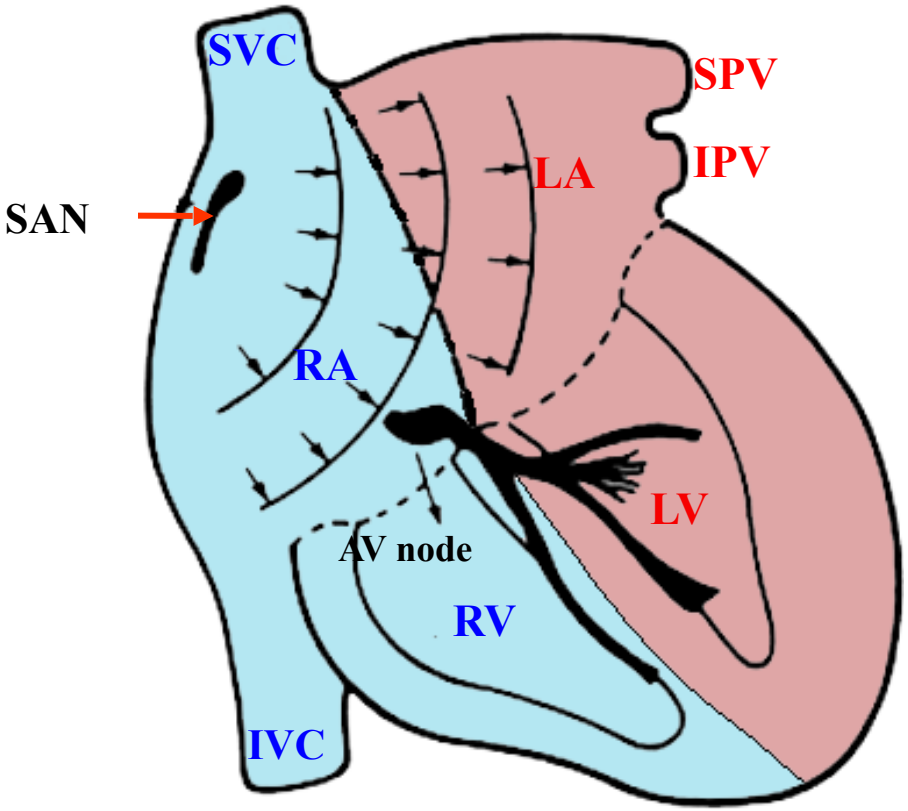


Atrial Activation Pathways

A Hypothesis currently accepted



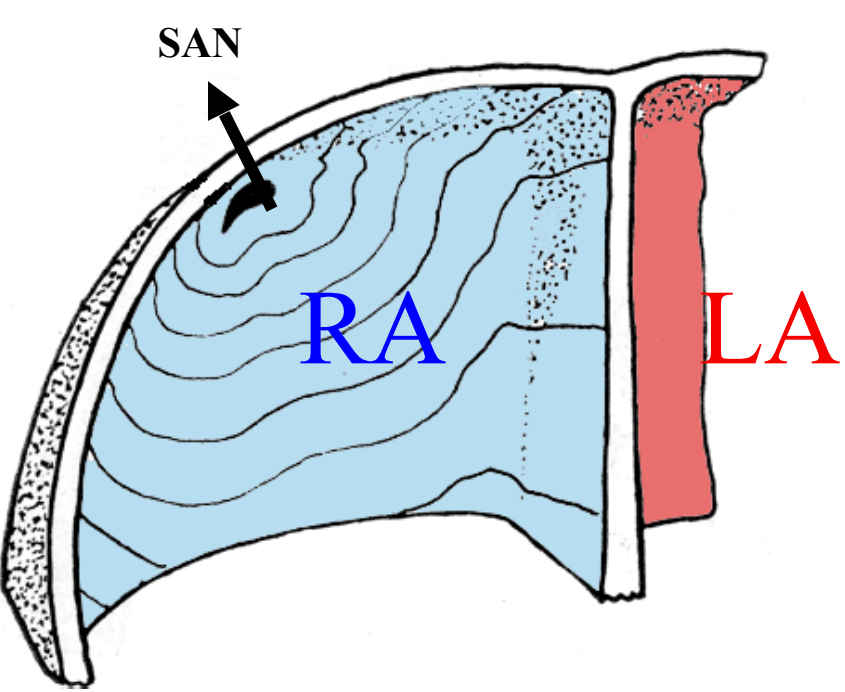
B Previous hypotesis

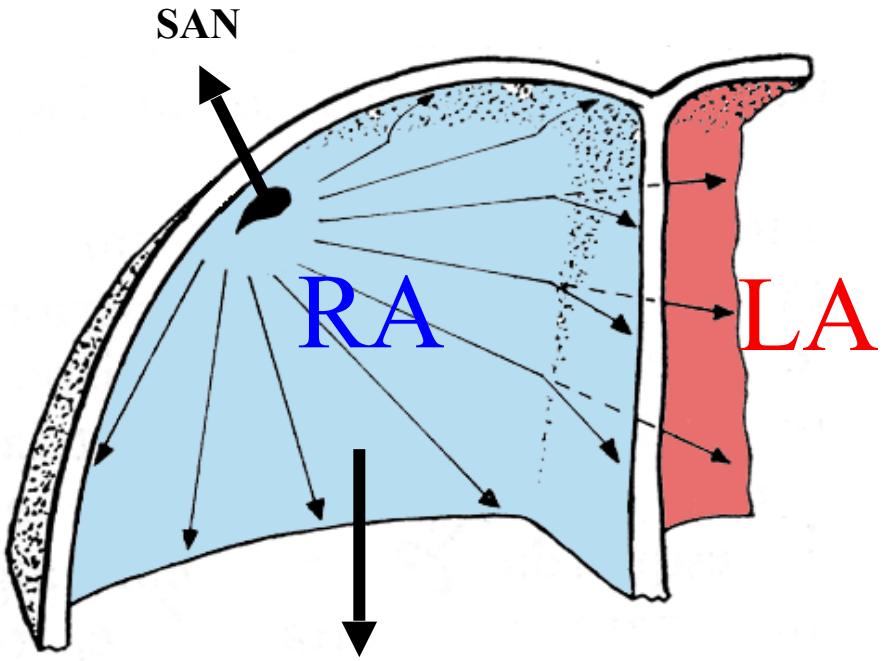


A: Anterior internodal bundle
M: Middle internodal or Wenckebach bundle
P: Posterior internodal or Thorel Bundle
BB: Bachmann's Bundle
SAN: Sinoatrial Node

SVC: Superior Vena Cava
IVC: Inferior Vena Cava
RA: Right Atrium
LA: Left Atrium

Old or previous hypothesis B – The activation wave spreads in a radiated way through the atria, just as the waves in a lake when you throw a stone in it.

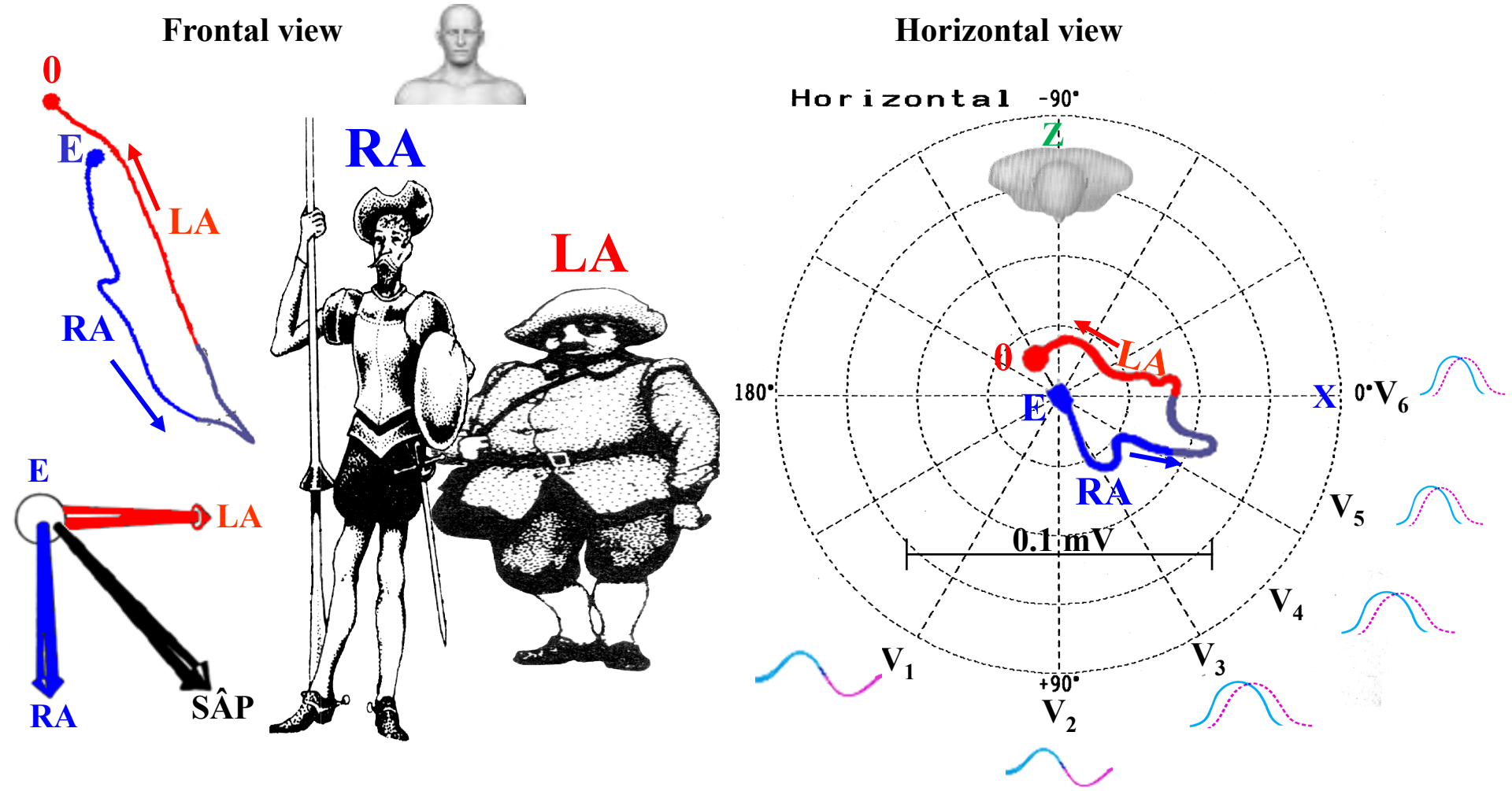




Conduction velocity 400 mm/s

RA: Right Atrium
LA: Left Atrium
SAN: Sinoatrial Node

Spatial outline of biatrial chamber activation in both the FP and HP



RA - “Don Quixote”. In the front & to the right.

LA - “Sancho Panza”. In the back and to the left.

Normal P wave

P wave: It is the first wave of ECG that represents the depolarization wave of the biatrial chamber.
Items to be analyzed:

I) Polarity
II) Voltage or amplitude
III) Duration
IV) Morphology (aspect or shape).

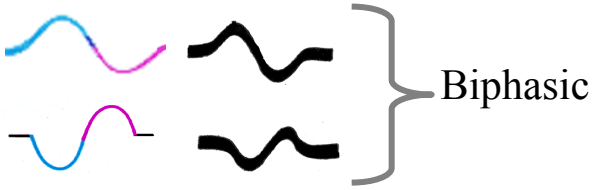
I) Polarity

a) Positive: 

b) Negative: 

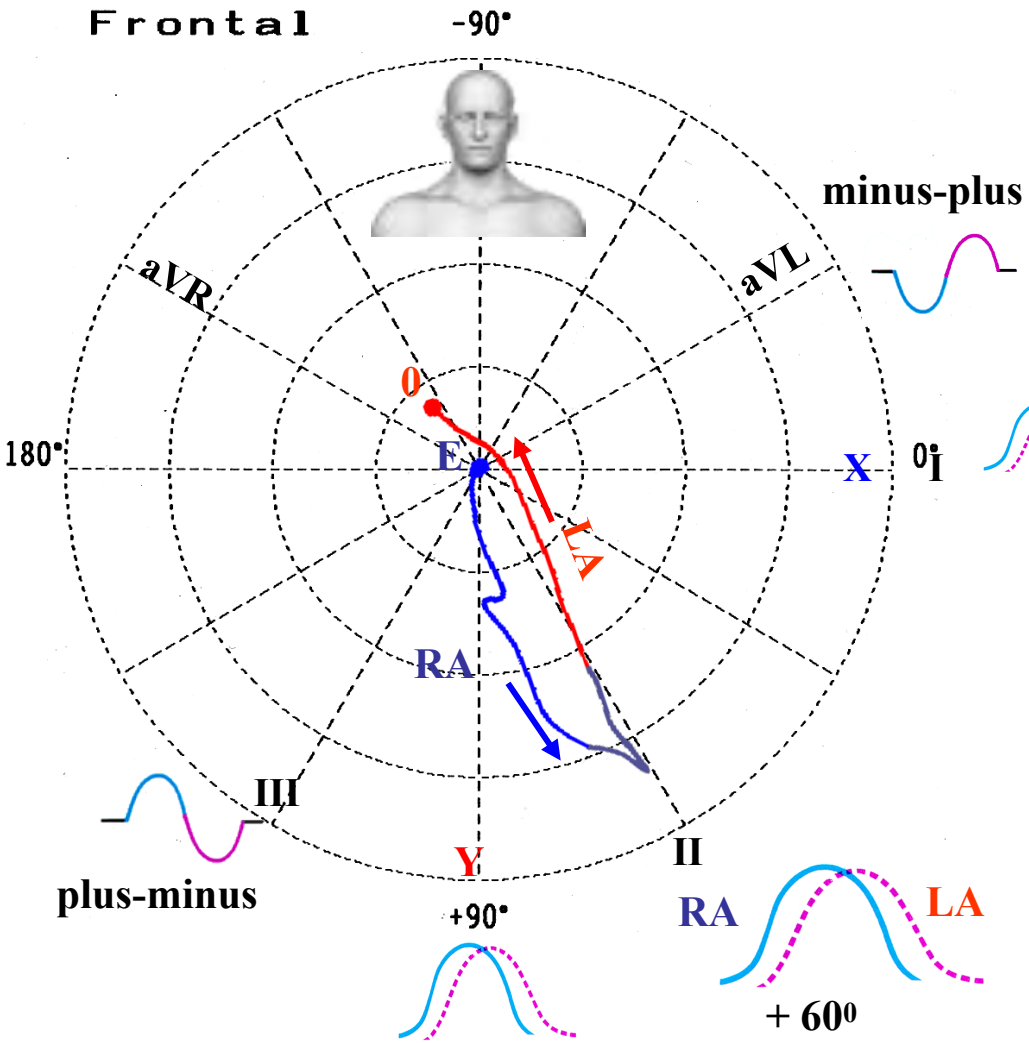
c) Positive-negative or "plus-minus": ±

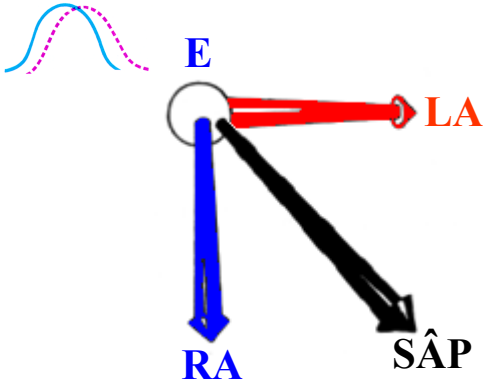
d) Negative-positive or "minus-plus":



In the frontal plane (FP) P wave is always positive in II, I and aVF, always negative in aVR, and variable in III and aVL. In III it could be plus-minus, negative or positive. In aVL it could be minus-plus, positive or negative.
In the Horizontal plane (HP) P wave is always positive from V3 to V6 and variable in V1-V2: positive or plus-minus.

P-wave polarity and P-loop in the Frontal Plane

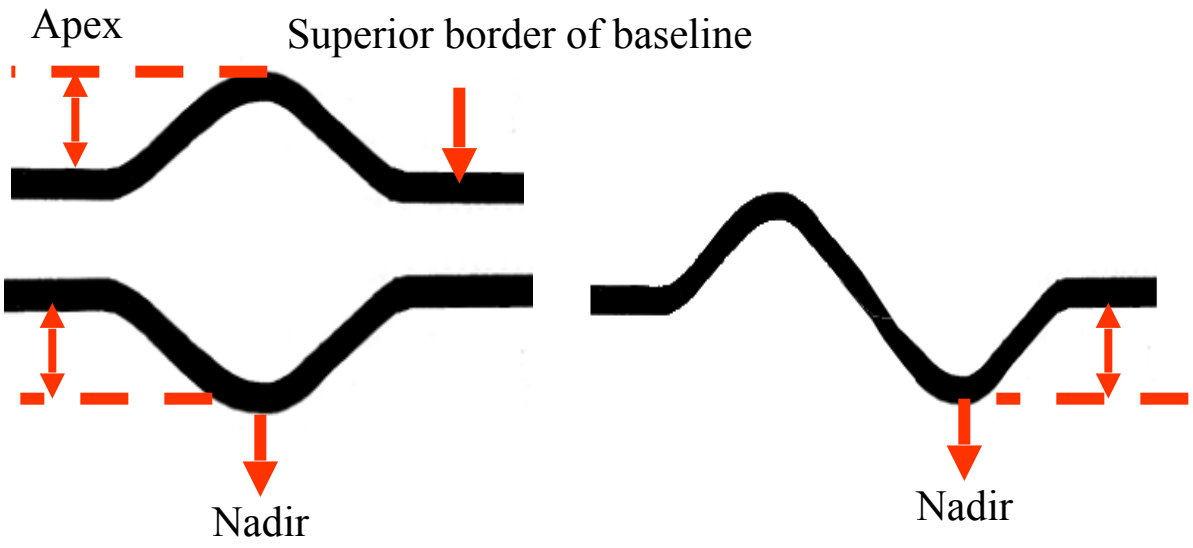




P wave polarity is always negative in aVR, always positive in II, I and aVF, and variable in III and aVL.
III could be plus-minus and aVL minus-plus
The P-loop begins in the E point located below and to the left of the 0 point.
P-loop ends in the 0 point.

II) Normal P-wave voltage or amplitude

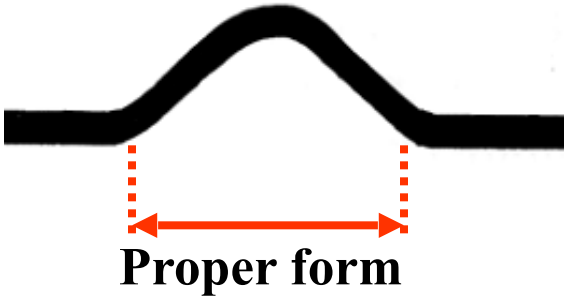
Proper measurement of voltage of P-wave

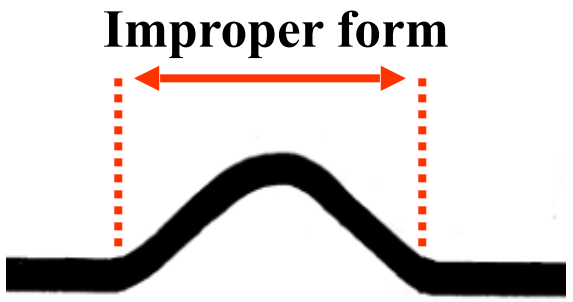


Since 1935, it is admitted that the normal maximal value of P wave voltage or amplitude is 2.5 mm, within the range of 0.5 mm to 2.5 mm (or 0.05 to 0.25 mV). However, this value has been questioned by Asad et al, because of low sensitivity in patients carriers of COPD, who suffer frequent exacerbations of pulmonary decompensation. Both the amplitude and the direction of the vector of the P wave are dynamic and may reflect the stress of the right atrial wall or "**RA** strain" (**Asad 2003**).

III) Normal P-wave duration and measurement

Appropiate measurement of P-wave duration

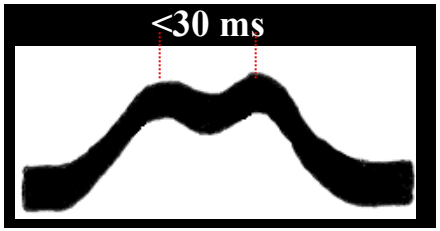




Age range	Normal maximal value of P-wave duration
0 to 12 months	80 ms (two little squares)
1 to 12 years	90 ms
> 12 years	100 ms. (2.5 little squares)
Seniors	110 ms

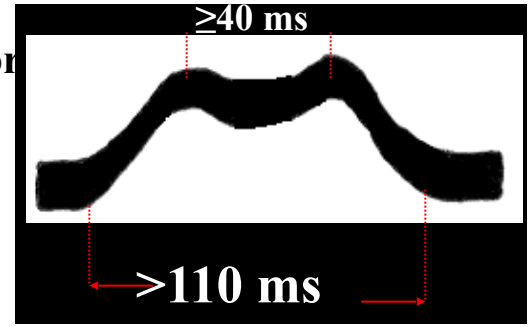
IV) Normal P-wave shape

Normal bifid or bimodal P wave



Normal, Chronic Amygdalitis, Diabetes, Vagotonia

Abnormal bifid or bimodal P wave



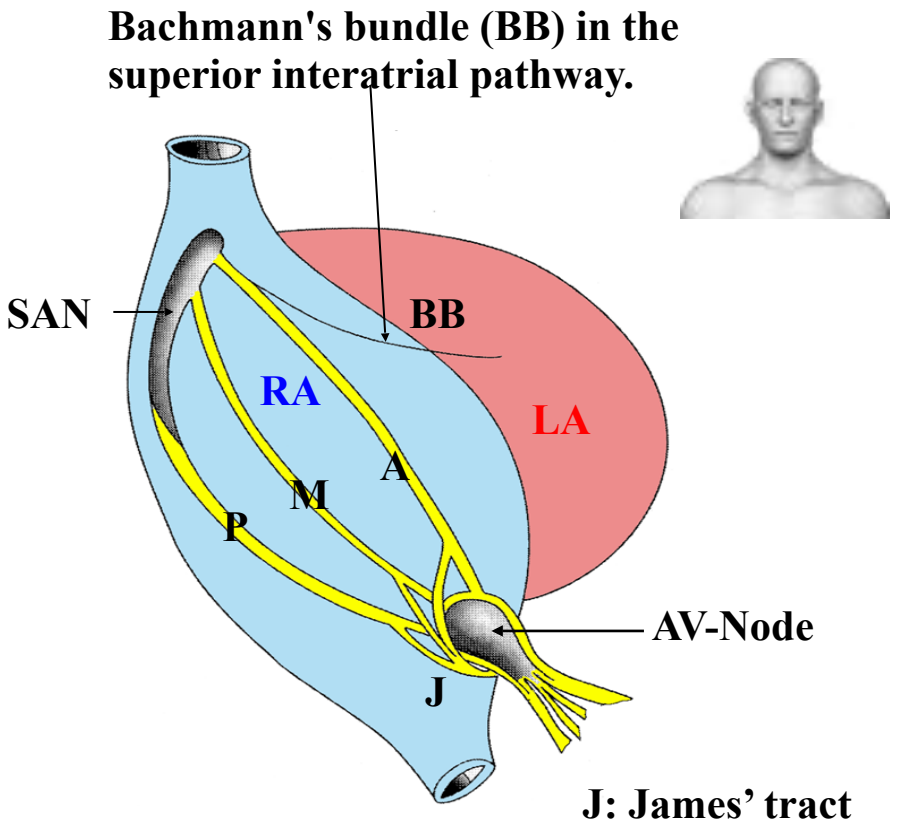
Left atrial enlargement (LAE) or interatrial block (IAB)

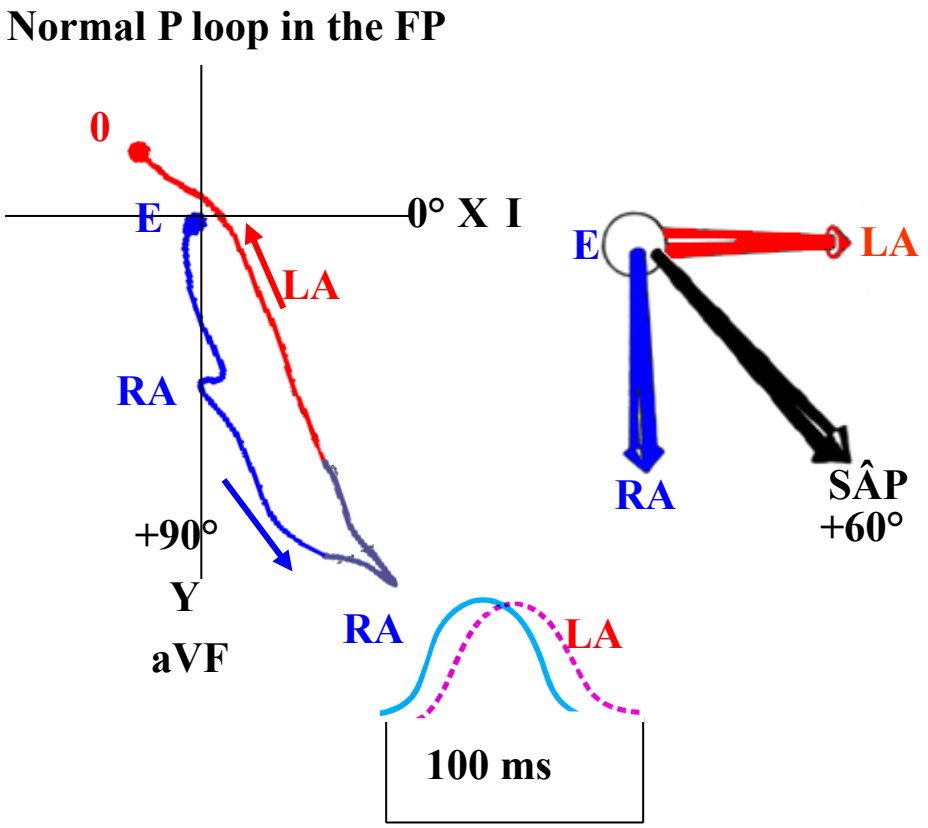
"Left atrial abnormalities"

Note: The term left atrial abnormalities was coined and widely used to encompass both atrial enlargement and interatrial block.(**Lee 2007**) Established ECG criteria for LAE do not reliably reflect LAE and lack sufficient predictive value to be useful clinically. P-wave abnormalities should be noted as nonspecific LA abnormalities, with the term "LAE" no longer used. The presence of at least one ECG criteria for LAE is sensitive but not specific for anatomic LAE. Individual criteria for LAE, including P mitrale, P wave axis <30°, or negative P terminal force in V1 (NPTF-V1) > 0.04 s.mm are highly specific, though not sensitive. ECG is highly specific but insensitive for RAE. Individual ECG P wave changes do not reliably both detect and predict anatomic atrial enlargement **(Tsao 2002)**. The normal shape of P wave is rounded and monophasic, and there may be small notches (more frequent in V_3 and V_4) and the distance between these notches should not exceed 30 ms (0.03 s). Notches in P wave with distance between the apexes of ≥ 40 ms (0.04 s) constitutes a sign of left atrial enlargement (LAE) or interatrial block by Bachmann's bundle (BB), in charge of activating the left atrium (LA).

Normal atrial conduction in the Frontal Plane

E point: it constitutes the zero point of VCG and it remains stationary before the onset of the P loop. It corresponds to the isoelectric line between the T wave and the P wave of ECG. The E letter corresponds to the cardiac dipole. It is the beginning of the P loop and ends in the so-called 0 point.



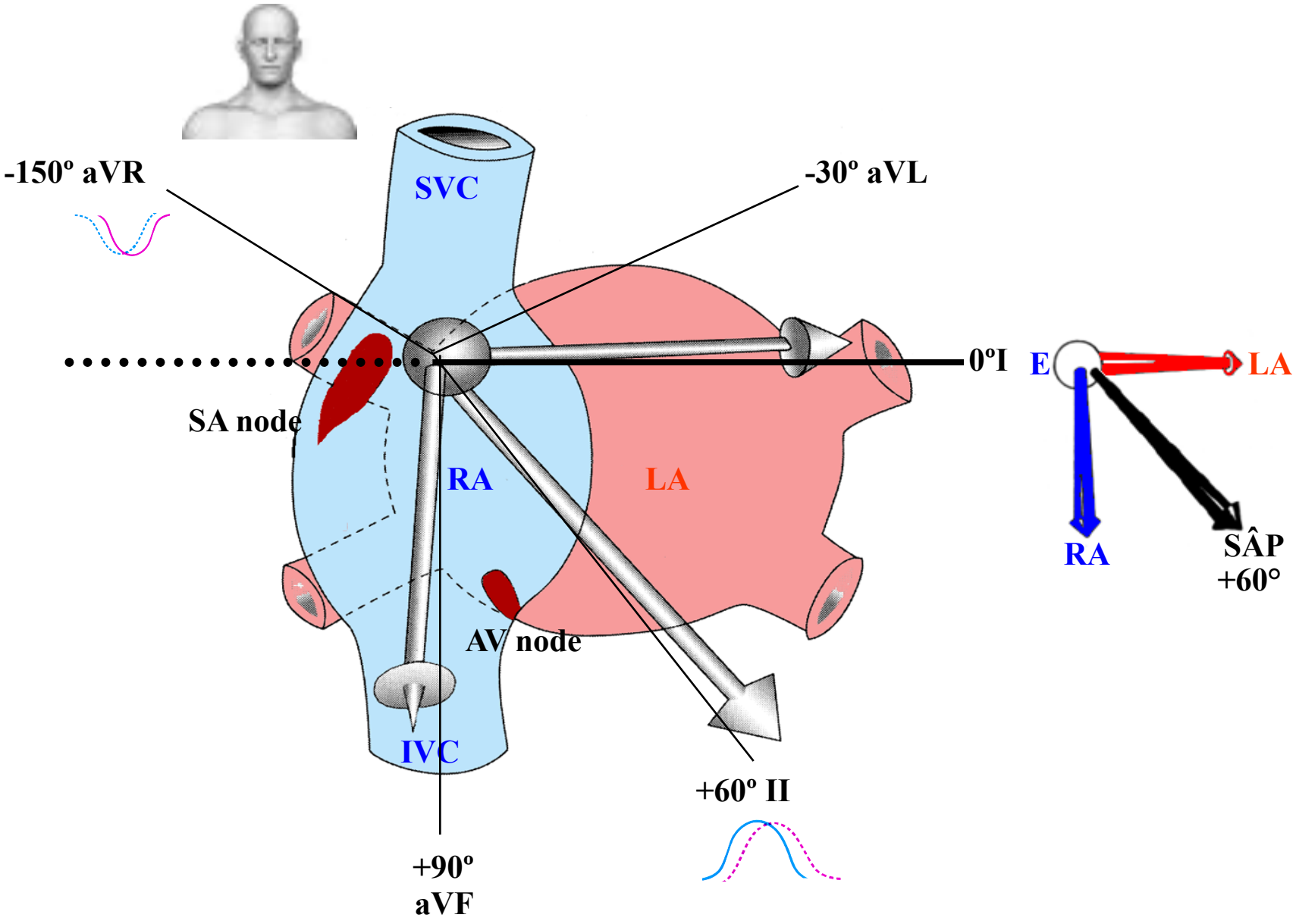


P loop: loop of small voltage corresponding to the depolarization of the biatrial chamber. The initial part corresponds to the right atrium (RA) (between 0 and 70 ms), next the interatrial septum (between 20 and 45 ms), and finally the left atrium (between 30 and 90 ms). To make an analysis possible, it is necessary to amplify: 1 mV = 30 cm. The P loop begins in the E point and ends in the so-called 0 point. The former has an anterior and inferior location in relation to the latter. The P loop is open because atrial repolarization (Tp loop) is diametrically opposite to the P loop.

Mode of atrial depolarization
Current hypothesis A: preferential pathways

A = Anterior Internodal Bundle
M = Middle Internodal Bundle
P = Posterior Internodal Bundle
BB = Bachmann's Bundle
J – James's tract
SAN: The sinoatrial node, abbreviated SA node or SAN, also called the sinus node

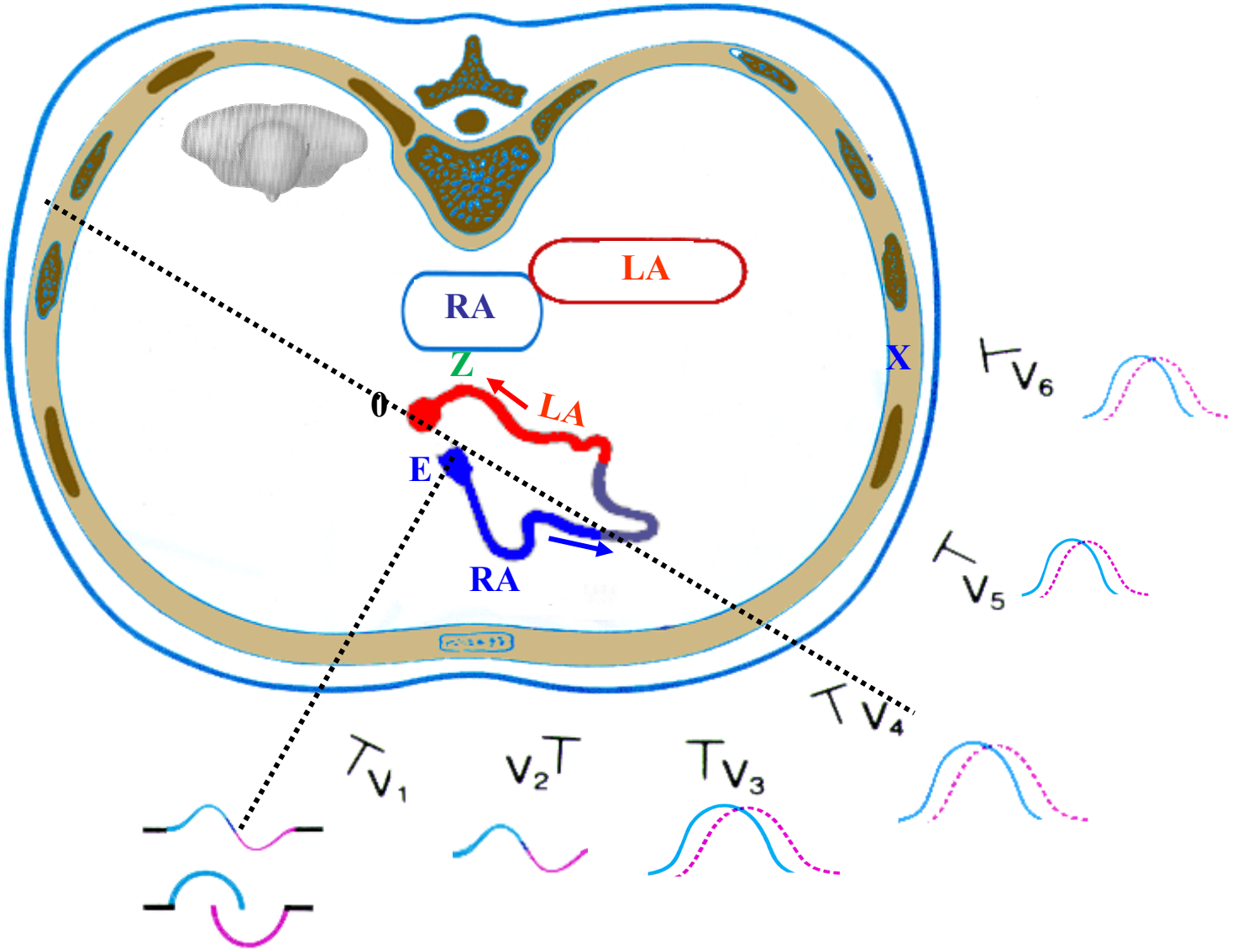
Anterior (frontal) view of biatrial chamber & its activation process



Conduction velocity in the different regions of the heart

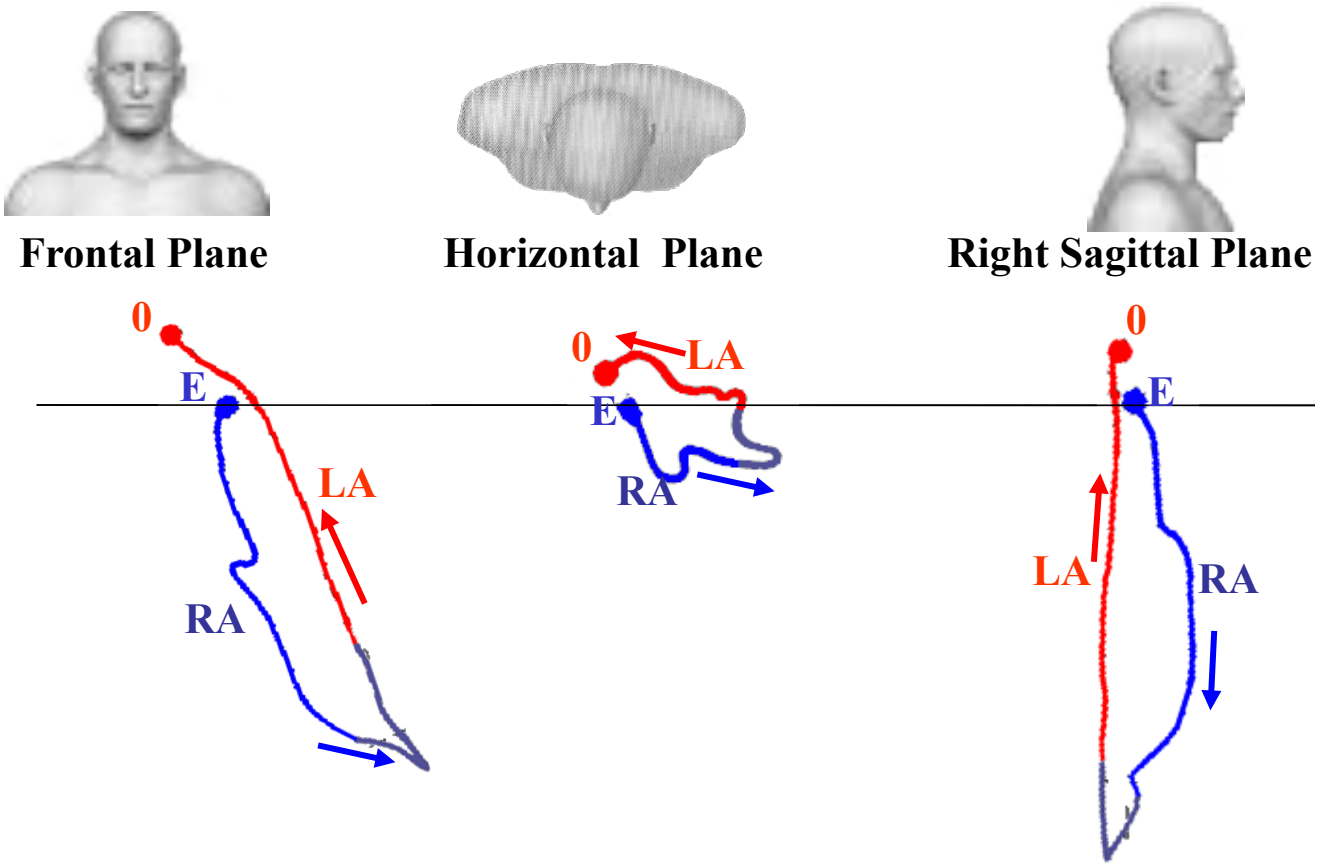
	Region	Speed
Atrium	Central region of Sinoatrial Node (SAN)	2 to 5 mm/s
	Peripheral region of SAN	7 to 11 mm/s
	Internodal bundles (anterior internodal bundle, middle internodal bundle, posterior internodal bundle, Bachmann's bundle, tract of James)	1000 mm/s
	Atrial muscle (RA and LA)	400 mm/s
	Atrio-nodal (AN) region of AV node	100 mm/s
	Nodal (N) region of AV node	20 mm/s
	Hisian node (HN) region of AV node	800 mm/s
	His, branches & arborizations of Purkinje	4000 mm/s
	Ordinary ventricular muscle cells	400 mm/s

Representation of normal P loop/P wave of biatrial chamber in the HP



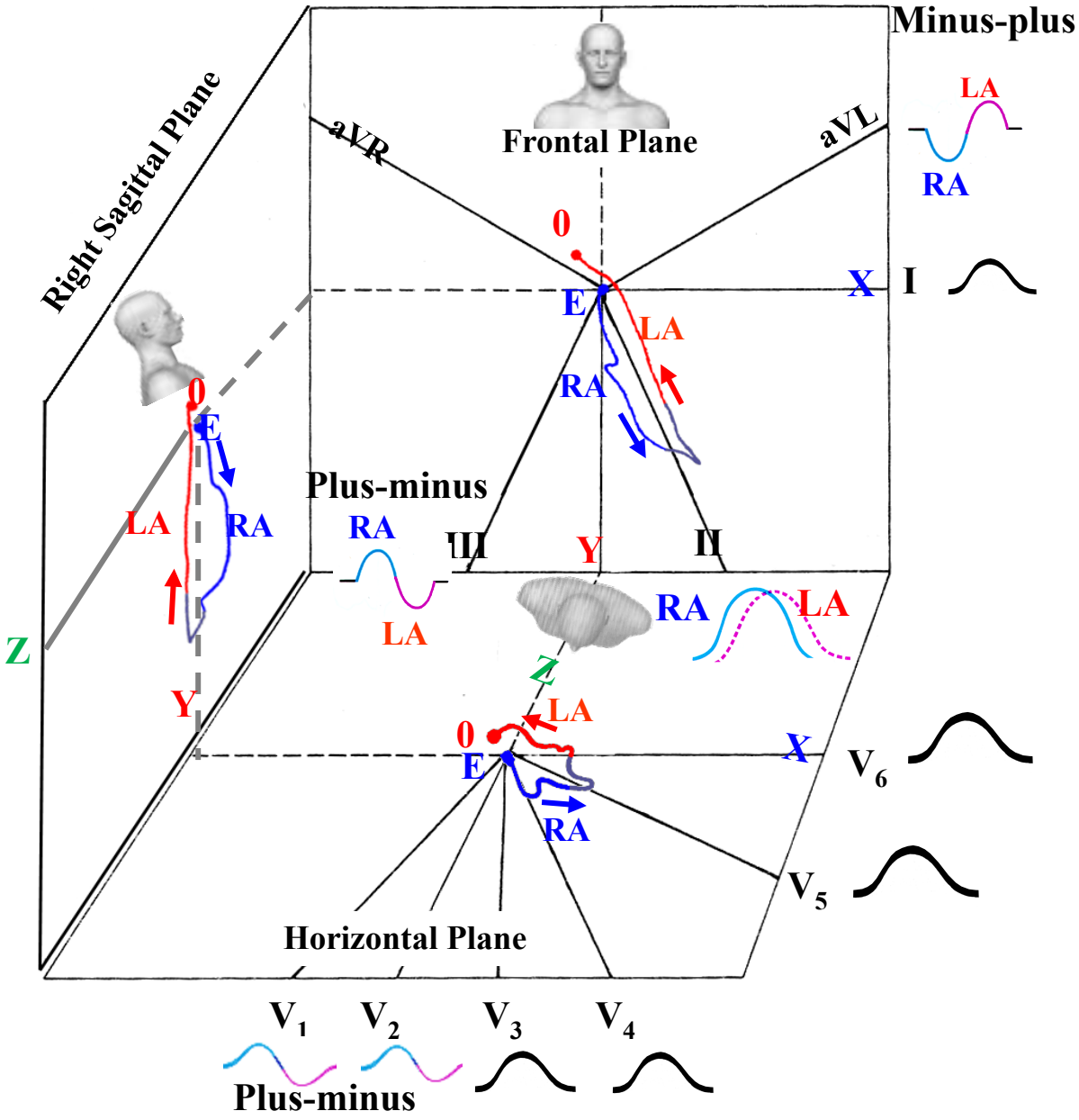
P wave is always positive from V3 to V6 and eventually plus-minus in V1-V2

P loops shape in the three planes

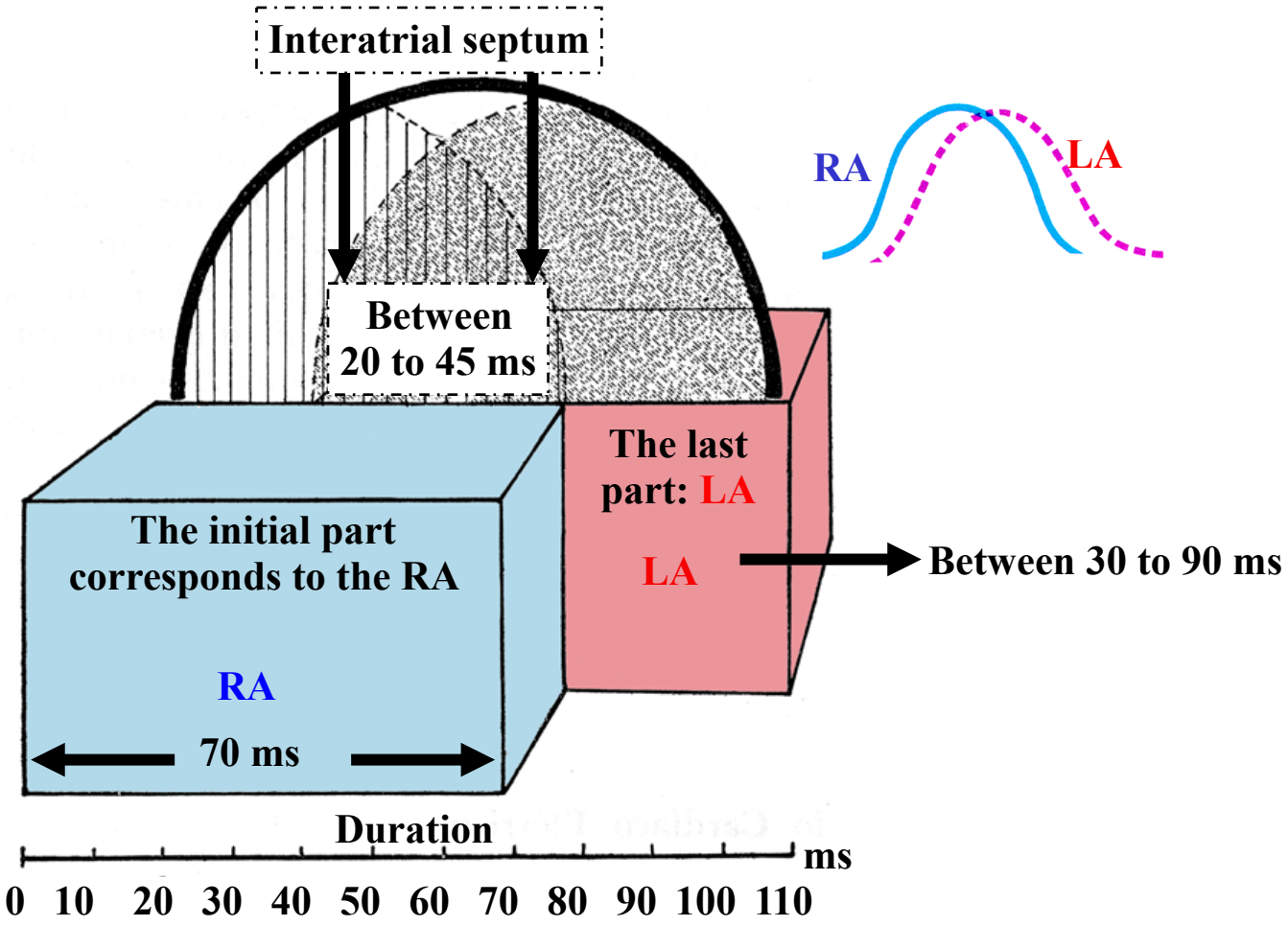


Atrial T vector (Ta vector): atrial repolarization (Ta vector) is responsible for the P loop being open and not closed (0 point does not coincide with initial E point). A straight line from the onset of the P loop (E point) to the end of it (0 point) indicates the magnitude and direction of the Ta or Tp vector.

P loops and P waves projected in the FP, HP and RSP



Normal activation of biatrial chamber



Normal duration of the P wave = 80 to 110 ms

Summary of normal characteristics of the P loop in the three planes

The P loop has a short slow conduction in the onset, in at least 2 planes.

Two small notches are usually observed: one in the efferent limb and another in the afferent limb.

	FP	HP	RSP
Rotation	CCW	CCW or in eight	CW
Direction	Inferior and left	Anterior initial and posterior final part	Antero-inferior initial and posterior final part
Morphology	Oval	Oval	Spear point or triangular
Location	Left inferior quadrant	$^1/_3$ in anterior quadrant and $^2/_3$ in posterior quadrant	$^1/_3$ anterior and inferior and $^2/_3$ posterior and inferior
Location of maximal vector	+65° (+20° to +20°)	+50° to –45°	+55° to –20°
Voltage of maximal vector	0.2 mV or less.	≤ 0.1 mV	≤0.18 mV
Maximal anterior forces		Adults up to 0.06 mV Children up to 0.08 mV.	Adults up to 0.06 mV Children up to 0.08 mV
Maximal posterior forces		Up to 0.04 mV	Up to 0.04 mV
Maximal left forces	Adults up to 0.09 mV Children up to 0.13 mV	Adults up to 0.09 mV Children up to 0.13 mV	

Age-related changes in P-wave morphology in healthy subjects (Havmoller 2007)

1) Orthogonal P-wave morphology in healthy individuals are predominantly positive in Leads X (0+ - 180° in I) and Y (+90° to -90° in aVF)

2) In Lead Z (near the V2 line), 35% had negative morphology and 65% a biphasic one with a transition from negative to positive. The latter P-wave morphology type is significantly more common after the age of 50

3) P-wave increased with age being slightly longer in subjects older than 50 years old

4) Changes of signal averaged orthogonal P-wave morphology (biphasic signal in Lead Z), are common in healthy subjects and appear predominantly after the age of 50.

5) Subtle age-related prolongation of P-wave duration is unlikely to be sufficient as a sole explanation of this finding that is thought to represent interatrial conduction disturbances.

Atrial enlargement

Concept

The ECG expression of atrial enlargement is due more to atrial dilation than atrial hypertrophy because the atrial wall is very thin and when submitted to an increase in pressure it usually dilates before increasing its myocardial mass (**Ferroglio 1979**).

The standard techniques to correlate ECG changes with the presence of atrial enlargement were previously based on anatomic, radiologic, and hemodynamic standards (**Reynolds 1953, Morris 1964**). The necropsic studies used were feasible only in cases of very advanced heart disease, and thus their utility was limited. For more than 35 years, M-mode echocardiography and especially 2D echocardiography have been considered the "gold standard" (**Reeves 1981; Kaplan 1994; Rodevan 1999**) (Tables 1 and 2).

TABLE 1 Right atrial enlargement. ECG criteria with high specificity (Bayés de Luna 2013)

ECG criteria	SE %	SP %
A. QRS criteria		
1. QR or qR in V1	≈ 15	> 95
2. QRS V1 ≤4 mm + QRS V2/V1 ≥5	46	93
3. R/S > 1 in V1	≈ 25	> 95
4. SÂQRS > 90°	34	> 95
B. P criteria		
1. P wave in inferior leads < 2.5mm	7	100
2. Positive part of P wave V1 > 1.5mm	17	100
3. Positive part of P wave V2 > 1.5mm	33	100
C. Combined		
1. Positive part of P wave in V2>1.5mm+SÂQRS>+90°+R/S>1 in V1	49	100

TABLE 2 Left atrial enlargement. ECG criteria based on P wave changes with high specificity (Bayés de Luna 2013)

ECG criteria	SE %	SP %
1. Morris index (**Morris 1964**) (P terminal force in V1 mm/s) (0.04 mm/s)	69	93
2. NYAC score P duration ≥ 120 ms in I or II + Morris index (>0.04 mm/s) + SÂP ≈ 0°	15	98
3. P duration ≥ 120 ms in lead II + Terminal mode negative of P in V1 > 40 ms	50	87
4. P duration ≥ 120 ms in lead II + Morris index (>0.04 mm/s)	69%	49%
5. Interpeaks of P wave >40 ms	15	100
6. Plus-minus P-wave ± in II, III, aVF	5	100
7. SÂP beyond + 30°	8	90
8. P wave duration in II or III leads ≥ 0.12 s	33%	88%

Cardiovascular magnetic resonance (CMR) is currently the gold standard for atrial volume assessment. It has recently been demonstrated that 2D transthoracic echocardiography consistently underestimates the left atria (LA) and right atrial (RA) volume compared with CMR imaging (≈15-20%) (10). However, as the volumes assessed by the two techniques have similar slopes, except for the underestimation by 2D echocardiography, the study performed using CMR to evaluate LA volume would likely result in similar results to those obtained using 2D echocardiography (**Rodevan 1999**). The accuracy of ECG criteria for left and right atrial enlargement as detected by 2D Echocardiography or CMR has recently been shown as is expressed in Tables 1 and 2. For full information consult Bayés de Luna (**Bayés de Luna 2013**).

The sensitivity and specificity of different criteria varies with the methodology used, and particularly with the type of population studied. The specificity is usually much higher and the sensitivity lower, but this increases if the population studied presents with a higher degree of atrial enlargement.

Left Atrial Enlargement (LAE): Etiologies

I) Congenital causes:

- Heart disease with pulmonary hyperflux: Ventricular Septal Defect (VSD), Persistence of Arterial Channel (PCA), Complete Atrioventricular Canal defect (CAVC defect).
- Congenital mitral stenosis
- Severe Aortic valve stenosis (AS)
- Coarctation of the Aorta
- Hypertrophic Cardiomyopathy

II) Acquired causes:

- Mitral valvular heart disease: pure mitral valve stenosis, Mitral insufficiency/regurgitation: “P mitrale”;
- Decrease of LV compliance. E.g.: systemic hypertension, HCM, restrictive cardiomyopathies
- LVF: left ventricular failure by increase of Pd_2 of LV. E.g.: extensive infarctions, dilated cardiomyopathy;

Left Atrial Enlargement (LAE): electrocardiographic criteria

I) Direct criteria

1) P wave of increased duration: ≥ 110 ms in adults, ≥ 120 ms in seniors, and 90 ms in children. Specificity: 90% and sensitivity: 40% in old age
2) Notched and bifid P wave in II, with interval between the apexes ≥40 ms. Voltage of 2nd module > than the 1st. A bifid P wave is a rare finding with left atrial diameter ≥60 mm. Atrial fibrillation is present in 70% of cases.
3) SÂP (P axis on frontal plane) deviated to the left: between +40° and -30°;
4) Increase in depth and duration of final negative component of the wave in V_1 (left atrial enlargement Morris' index (**Morris 1964**)); slow and deep of P in V_1 or V_1-V_2. PTFV1. P terminal force in lead V_1 equal or more negative than 0.04 mm/s Greater than 0.03 mm/s: product of the duration of the final negative component (duration expressed in seconds); while depth is expressed in mm. Values above 0.03 mm per second constitute a highly sensitive criterion for diagnosis of LAE.
5) Macruz index (**Macruz 1958**) > 1.7: $\text{Duration of P} / \text{Duration of PRs}$;
6) Intrinsic deflection of V1 of 30 ms (0.03 s) or greater. This deflection is measured from the apex of the initial positive component until the nadir of the final negative component of the P wave of V1.
7) P-terminal force (PTF-V1) exceeding 0.04 mm/s. This is the terminal, negative part of the P wave in lead V1 expressed as the multiplication of its depth in millimeters and width in seconds (mm/s). The normal PTF-V1 does not exceed 0.04 s wide and 1mm deep, i.e., 0.04 mm/s.

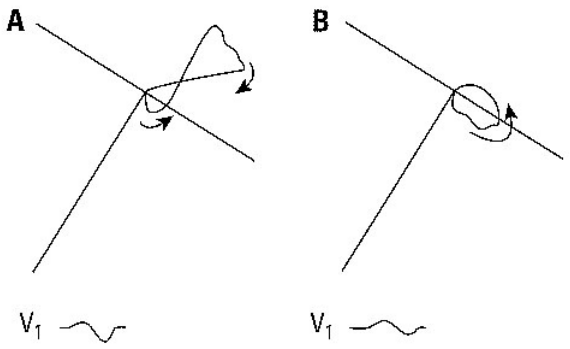
II) Indirect criterion

Presence of coarse atrial fibrillation: “f” waves with amplitude equal or higher than 1 mm in V_1 or V_1 - V_2: in 75% of the cases of coarse AF there is LAE coexisting. There is a significant relationship between the size of the f wave and the etiology of AF. Thus, 88% of the patients with coarse AF have rheumatic valvular heart disease as underlying cause and 88% of patients with fine AF present coronary artery disease.

False positive and false negative diagnoses of left atrial enlargement

The diagnosis of LAE may often be difficult to achieve because of the following factors:

- The presence of isolated interatrial block explains the increase in the duration of the P wave, without the presence of evident teminal negative mode of V1 (1). Thus, in contrast the combination of P wave duration in FP + increase of negative mode of P wave in V1 (Morris index) is a good criterion (**Morris 1964**).
- Some patients with isolated non-advanced left heart disease (e.g. mitral stenosis) without evident interatrial block show peaked P waves with no increase in duration (pseudo-P-pulmonale). In these cases, the presence of a P wave in V1 with a highly negative mode helps to reach a correct diagnosis (Figures below).



(A and B) P wave morphology in V1 and P loop in a case of isolated partial interatrial block (B) associated with left atrial enlargement (A).

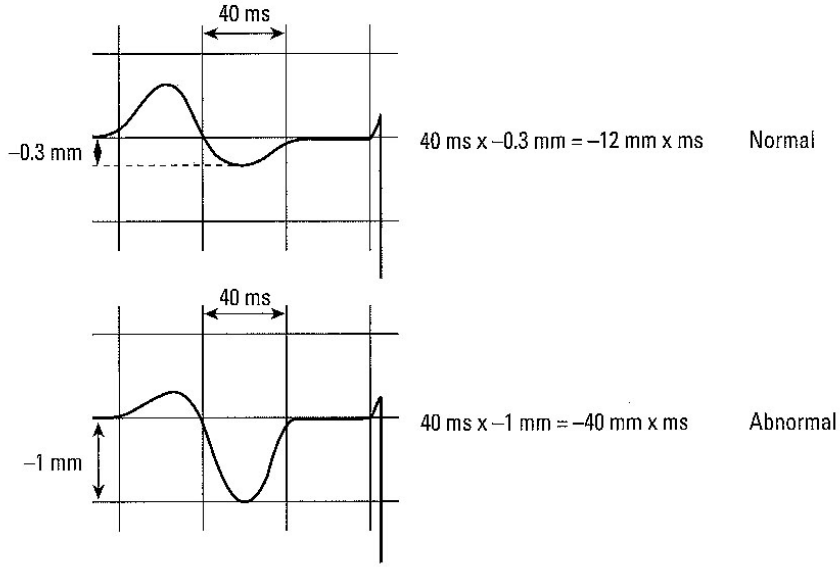
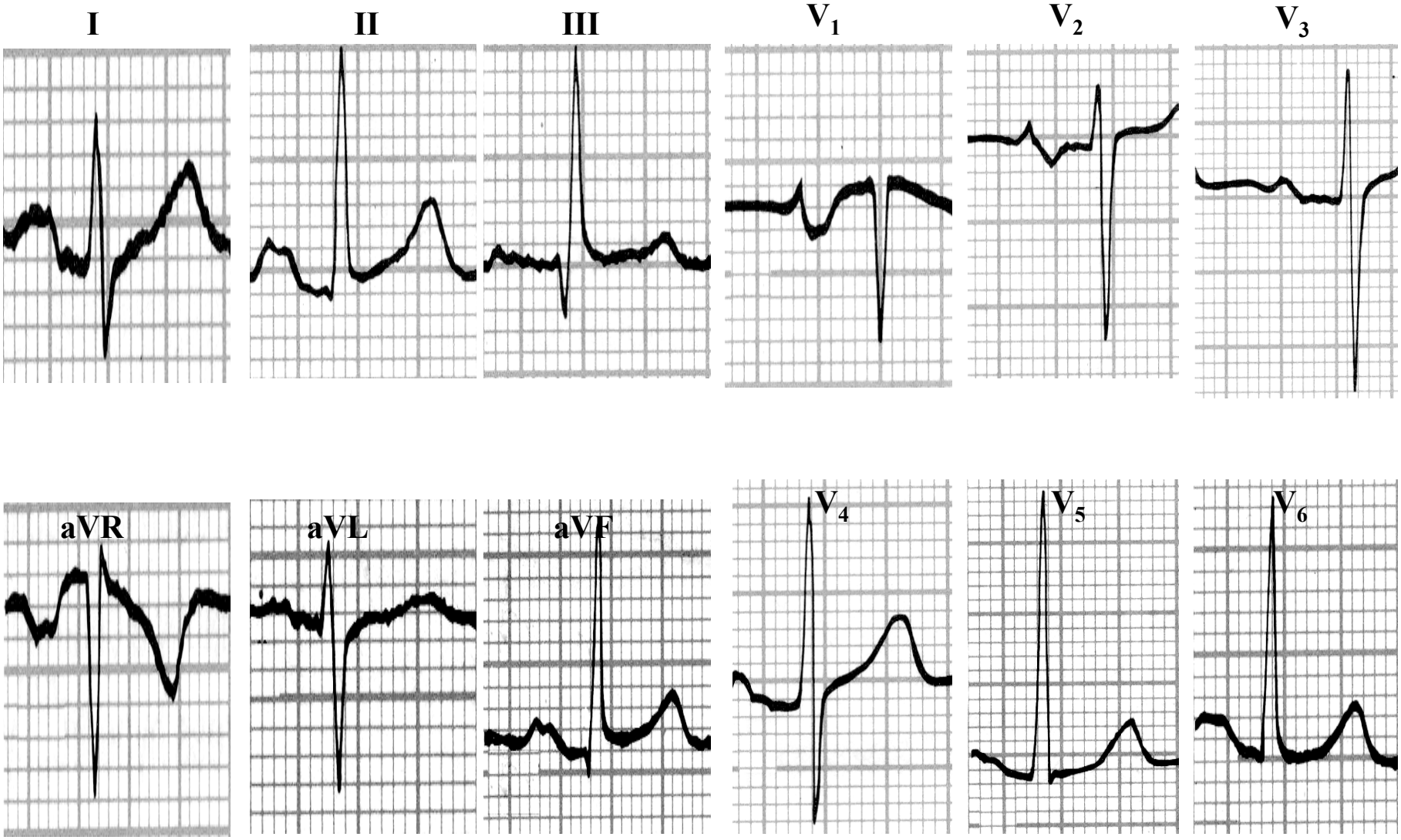


Diagram contrasting normal and abnormal negative components of the P wave in V1. When the value calculated using the width in seconds and the height in millimetres of the negative mode exceeds 40 mm + ms, it is considered abnormal.

- If important atrial fibrosis exists, small and even unapparent P waves (concealed sinus rhythm) may be seen, even in the presence of evident left atrial or biatrial enlargement (**Bayés de Luna 1978**). This problem increases the number of false negatives (low SE).

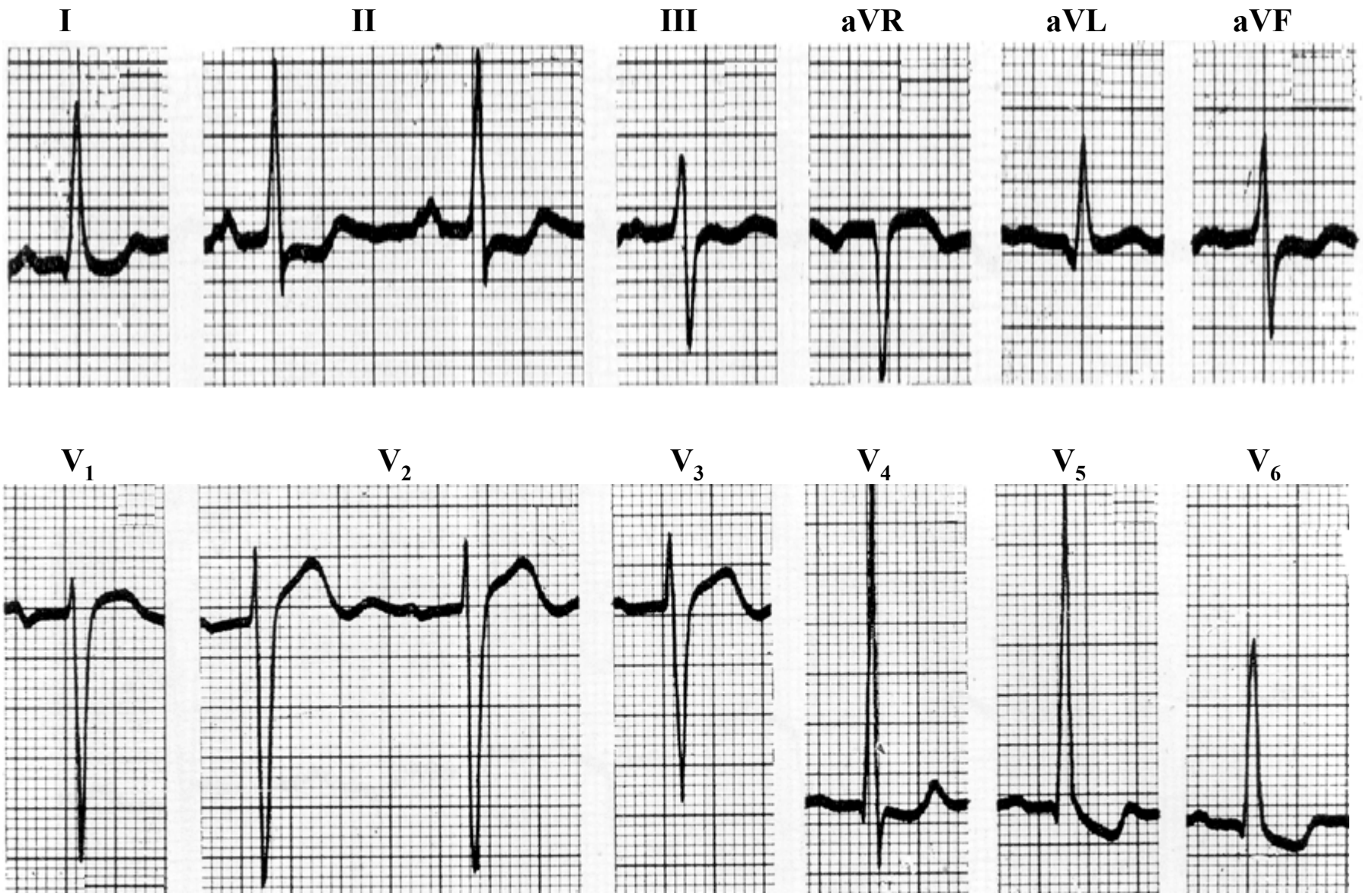
Many patients with COPD or thoracic abnormalities, pectus excavatum, and those with straight-back syndrome present with short but evident negative P waves in V1, a morphology that may be confused with LAE (**Bayés de Luna 2013**).

LAE: mitral failure by incompetent prolapse

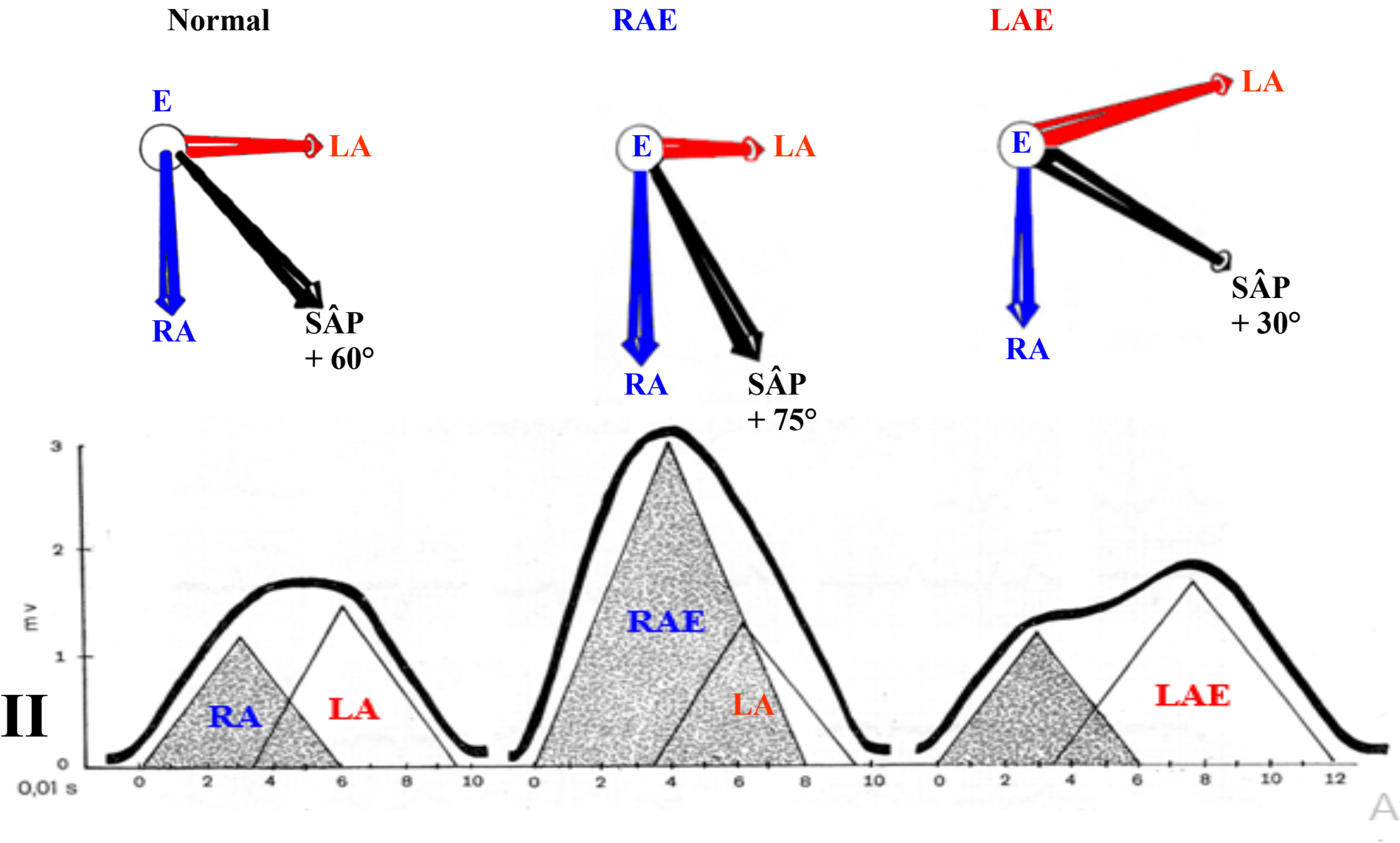


Mitral insufficiency (MI), **mitral regurgitation** or **mitral incompetence:** It is a disorder of the heart in which the mitral valve does not close properly when the heart pumps out blood. It is the abnormal leaking of blood from the left ventricle, through the mitral valve, and into the LA, when the left ventricle contracts, i.e. there is regurgitation of blood back into the left atrium.[1] MR is the most common form of valvular heart disease

Mitral failure: LAE + LVE



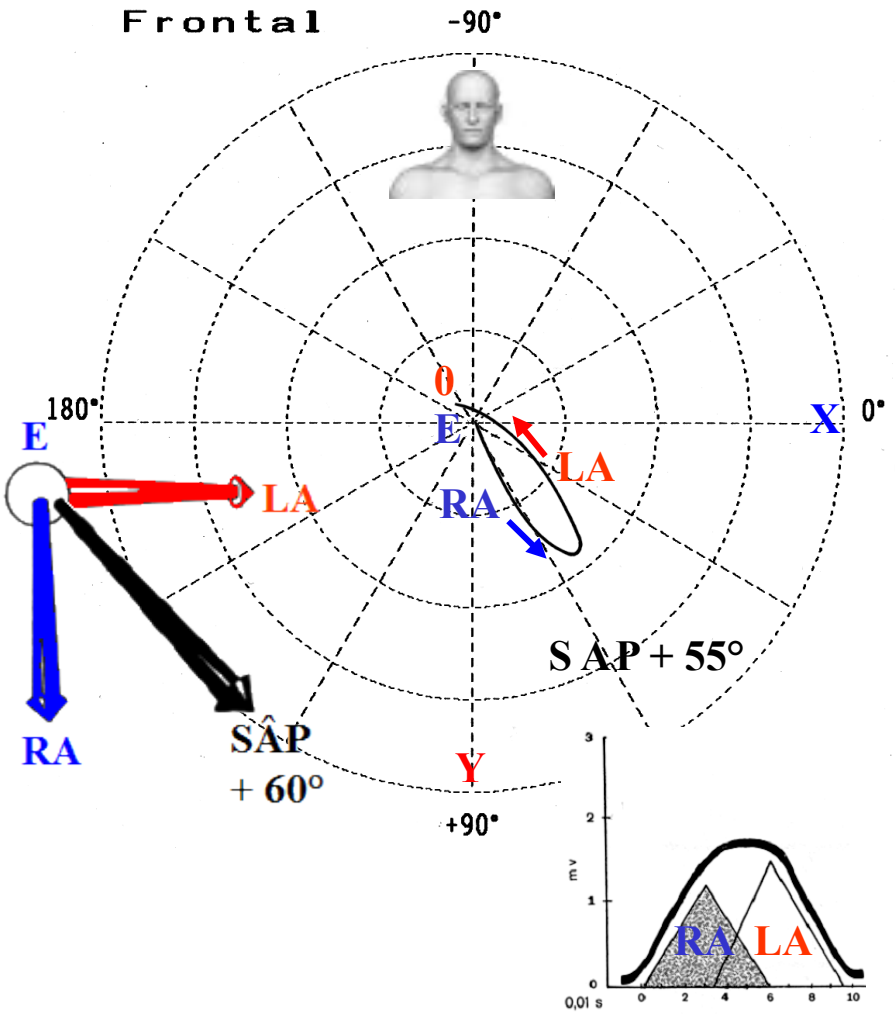
Profile of normal P wave in RAE and LAE



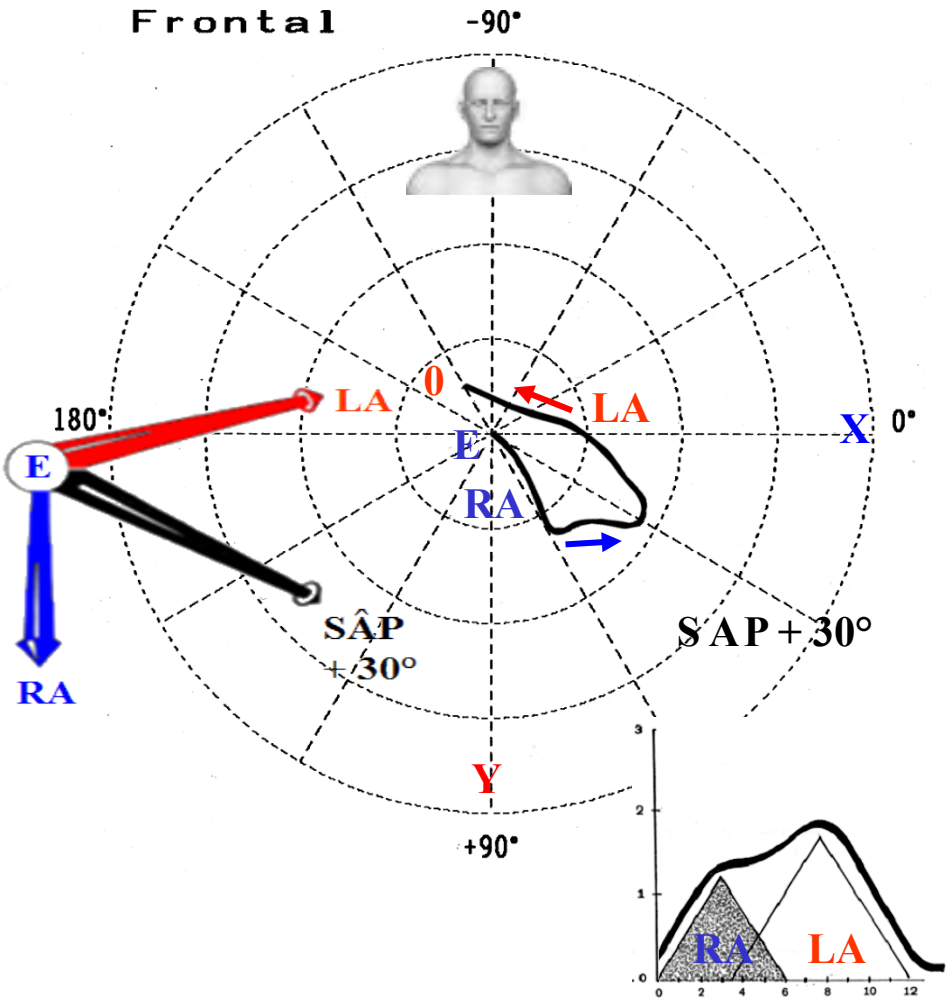
LAE in the Frontal Plane

Note: the findings in the Frontal Plane are not relevant for the diagnosis of LAE.

Normal P loop



P loop in LAE

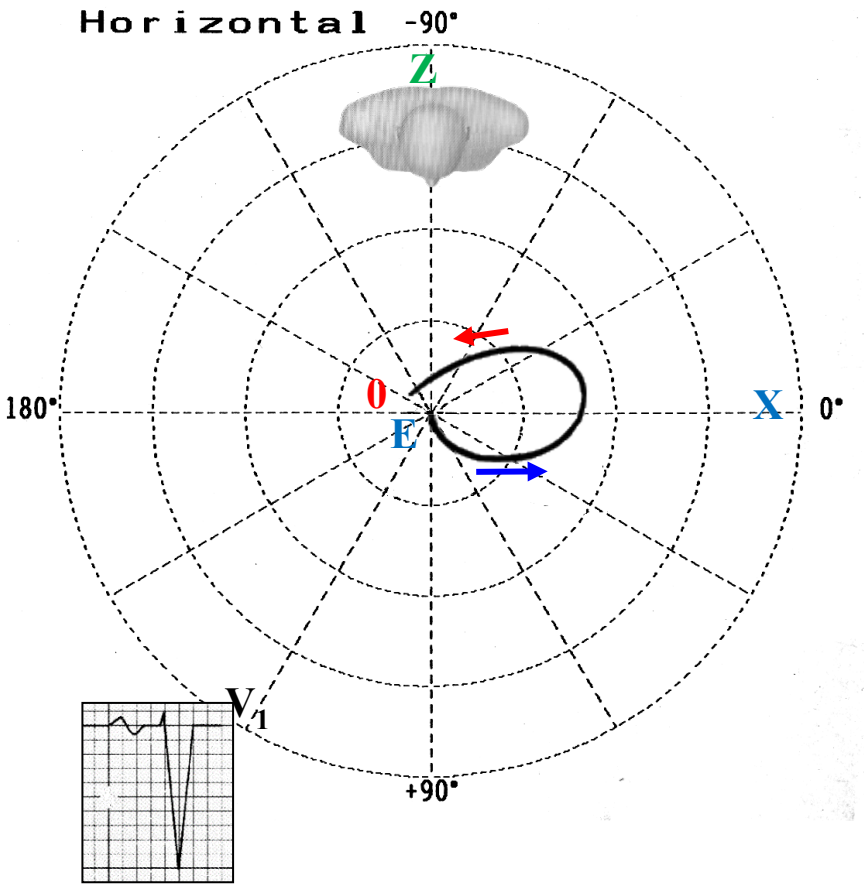


Similar to advanced interatrial block

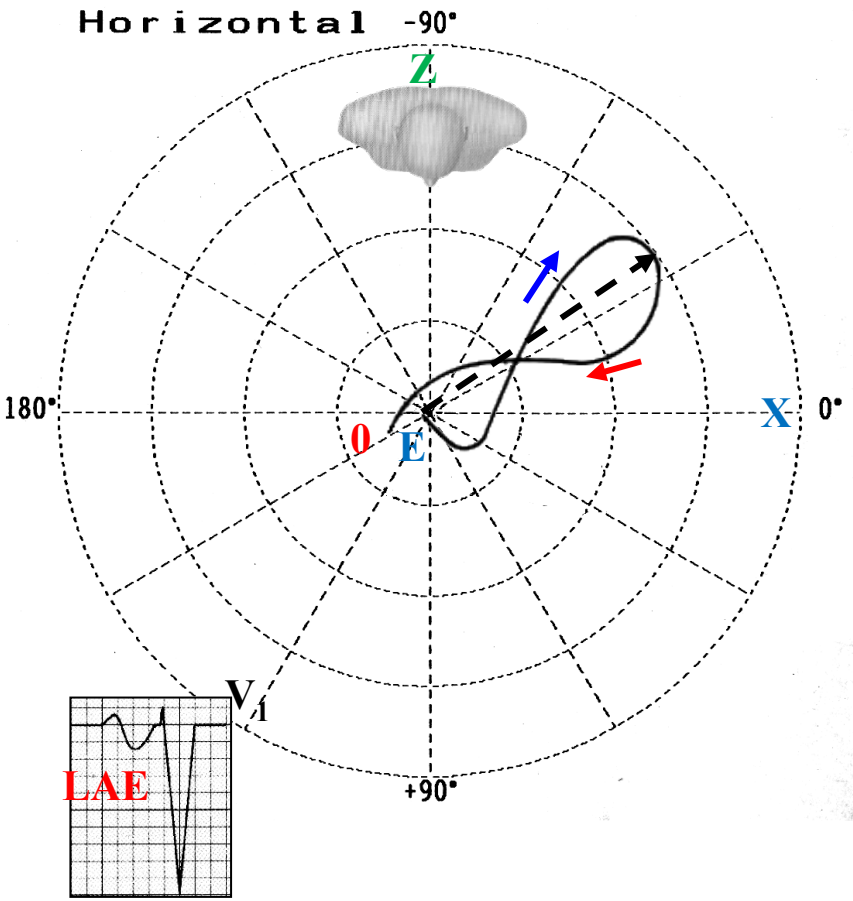
P loop in LAE on Horizontal plane

Note: the findings in the Sagittal and Frontal Planes usually are not relevant in diagnosis

Normal P loop



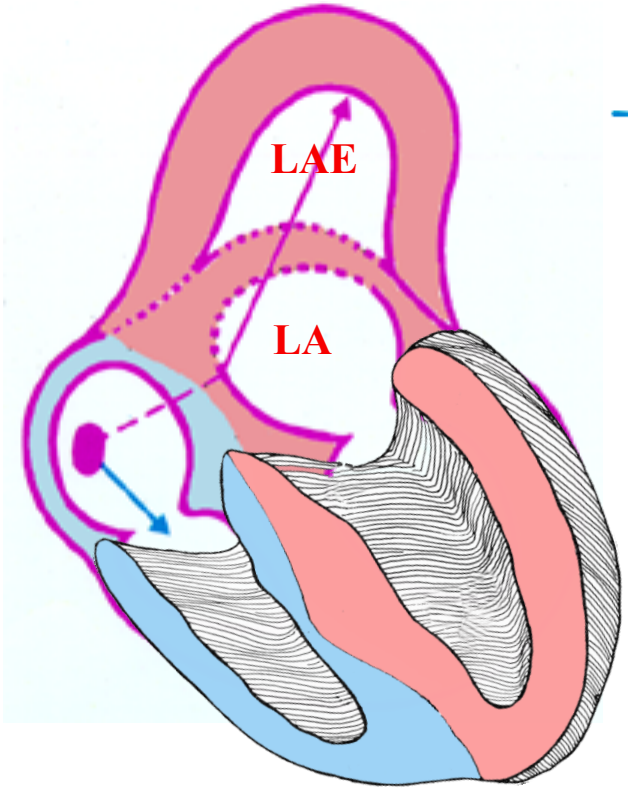
P loop in Left Atrial Enlargement



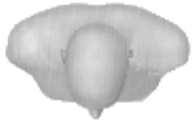
- The maximal vector of P is located to the left: ≥0.10 mV in adults and ≥0.14 mV in <15 years old
- Max. vector of P >0.05 mV
- "Bow Tie" morphology

LAE on FP

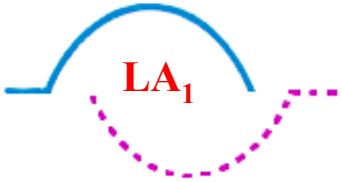
Left Atrial Enlargement in Horizontal Plane

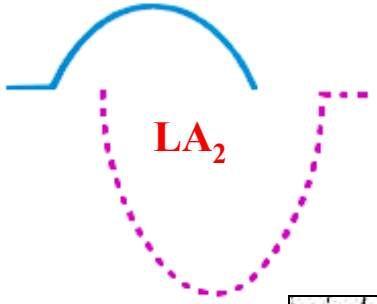


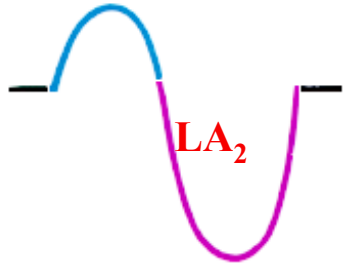
Normal P wave

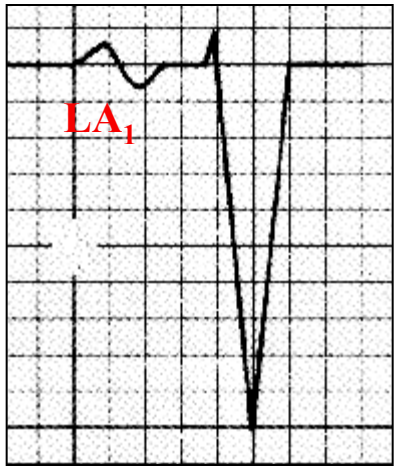


Left Atrial Enlargement P wave

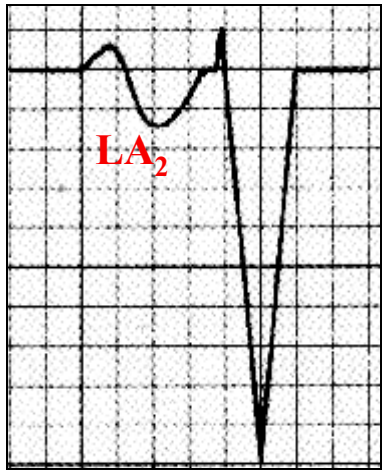








V_1



$LA_{2/}$: final deep and slow component: LAE ≥ the area of one small square; the final minus portion indicates left atrial enlargement, abnormality or advanced interatrial block.

Right Atrial Enlargement: Etiologies

I) Congenital causes: "congenital P"

- Ebstein's anomaly - "Himalayan P waves" of Taussig;
- Tricuspid atresia: "P tricuspidale" of Gamboa (10%);
- Moderate pulmonary stenosis "gothic" P (30%);
- Severe pulmonary stenosis P >2.5 mm 75%;
- Eisenmenger Syndrome;
- Atrioventricular septal defect (LV LA). RAE or BAE 60%;
- Tetralogy of Fallot (T4F): only in 5% there are criteria of RAE.

II) Acquired causes

- Cor pulmonale - emphysema - COPD: "P pulmonale"
- Tricuspid stenosis;
- Tricuspid regurgitation/insufficiency;
- Double tricuspid injury;
- Heart failure;
- Increase of RV Pd_2.
- Isolated Pulmonary Hypertension: the ECG is very sensitive in symptomatic patients with isolated pulmonary hypertension (**Bossone 2003**).

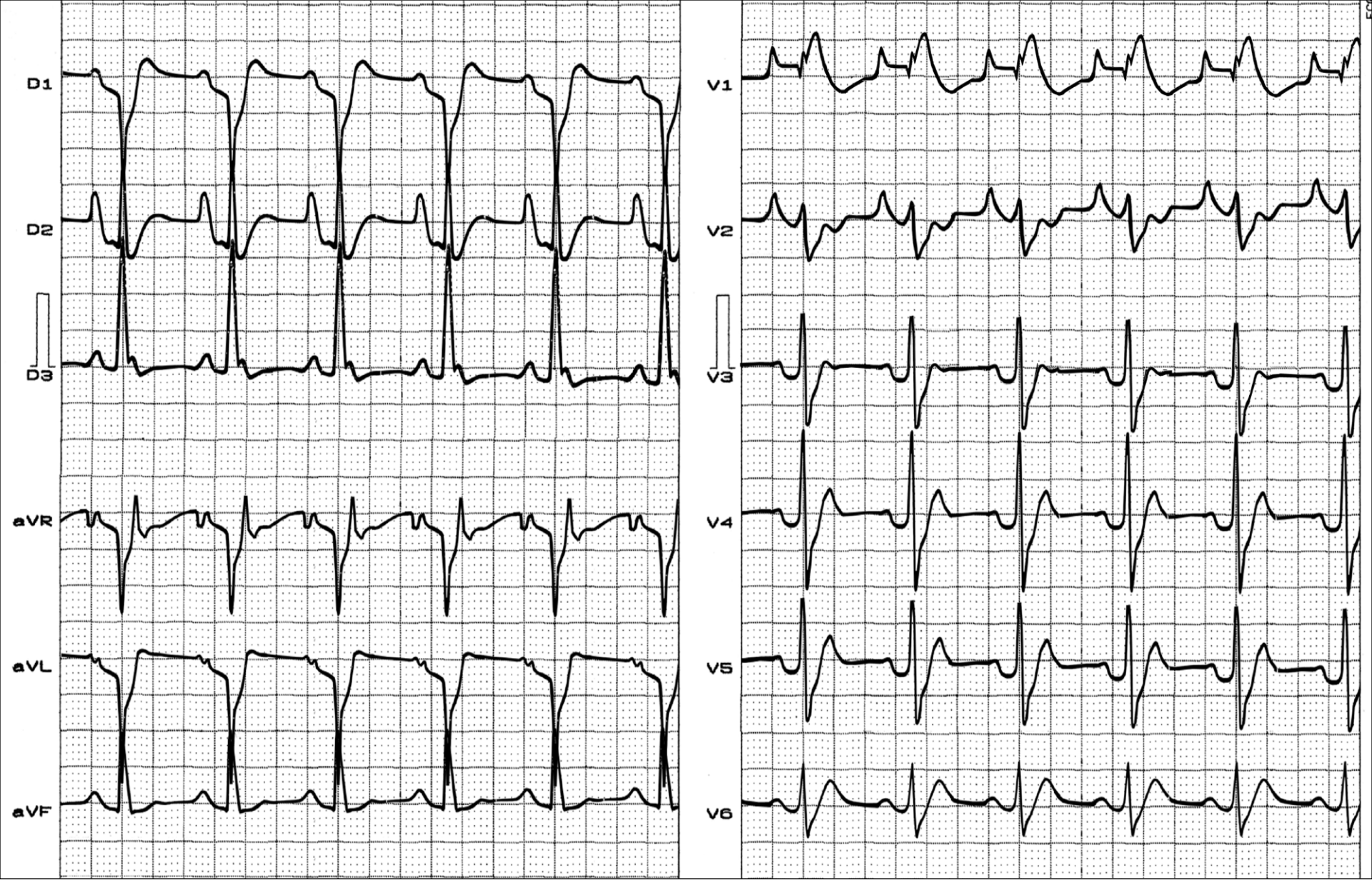
“Himalayan P wave”

Ebstein's anomaly is a rare, complex, fascinating, congenital anomaly with a broad pathologic-anatomical and clinical spectrum accounting for <1% of all congenital heart defects. Since its description in 1866, dramatic advances in diagnosis have been made. Very high “Himalaya mountain-like” P waves (**Kaushik 2007**) are observed. (The Himalayan mountain system are the planet's highest peaks around the world). P wave is >3 mm (0.3 mV) in close to 50% of cases (**Armengol 1996**). Tall P waves (≥2.5 mm) are attributable to right atriomegaly. A prolonged P-wave duration is occasionally registred (**Jaiyesimi 1982**).

In association with P wave modifications in Ebstein’s anomaly, the following are frequently observed (**Blömer 1975**): Prolonged PR interval (≥170 ms)**,** short PR interval if associated with WPW pattern (≈30% of cases)**,** bizarre low voltage right bundle branch block pattern**,** initial q wave in the QRS complexes of V1 and V2 leads.

Reduced amplitude of R-wave deflections in V3R and V1, P-dextro-atriale and bizarre low RBBB without right ventricular overload almost certainly constitutes a pathognomonic finding in Ebstein's anomaly. Higher P waves and wider QRS complexes are registered in more severe cases of Ebstein's anomaly of the tricuspid valve**.** There is a high potential for developing arrhythmia in the vast majority of tachycardia types: atrial ectopic tachycardia, atrial flutter, atrioventricular reentry tachycardia, AV-nodal reentry tachycardia, atrial fibrillation and ventricular tachyarrhythmias**.**

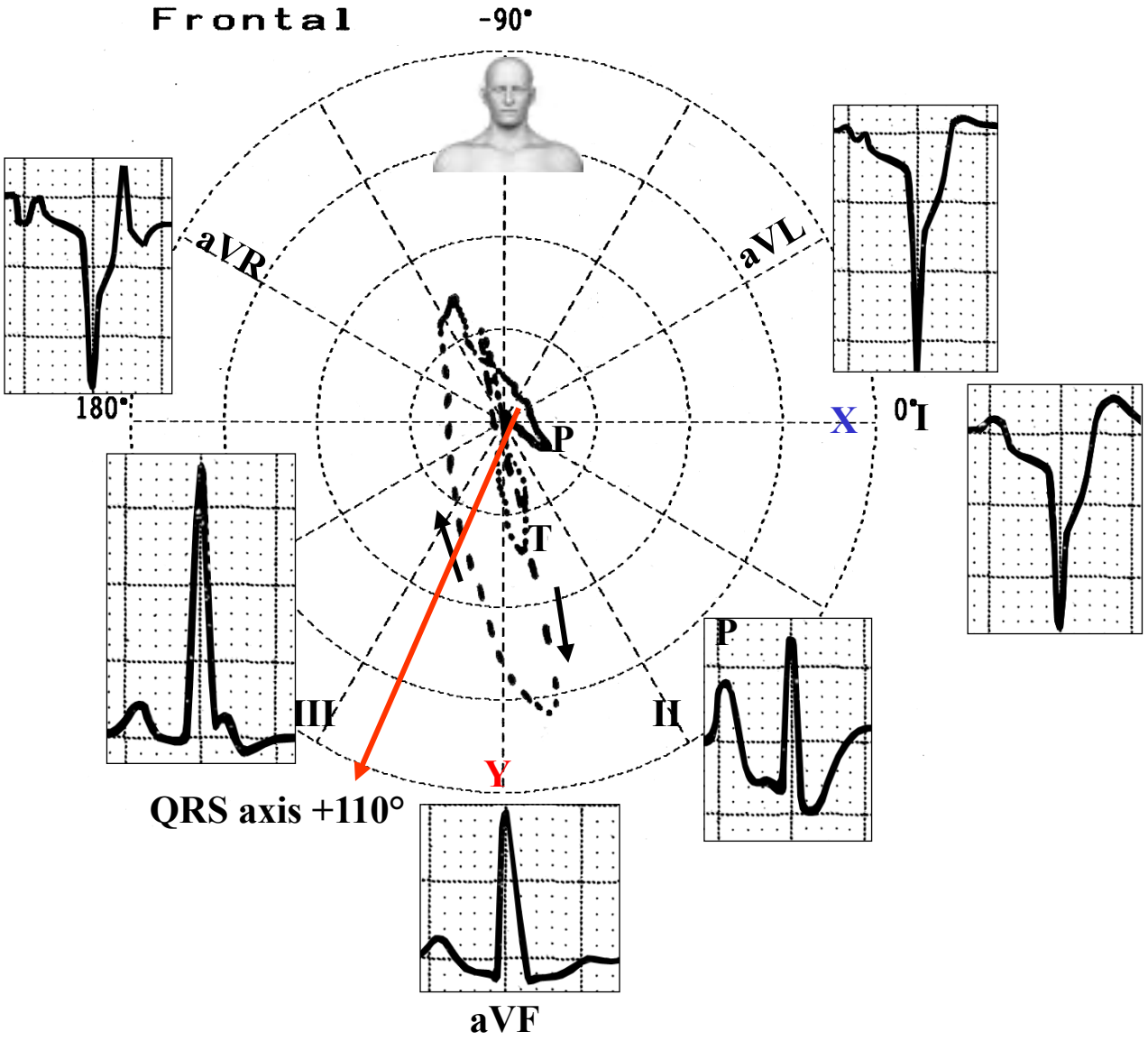
Tendency towards multiple arrhythmogenic substrates in a single patient (**Hebe 2000**).



Clinical diagnosis: Ebstein's anomaly.

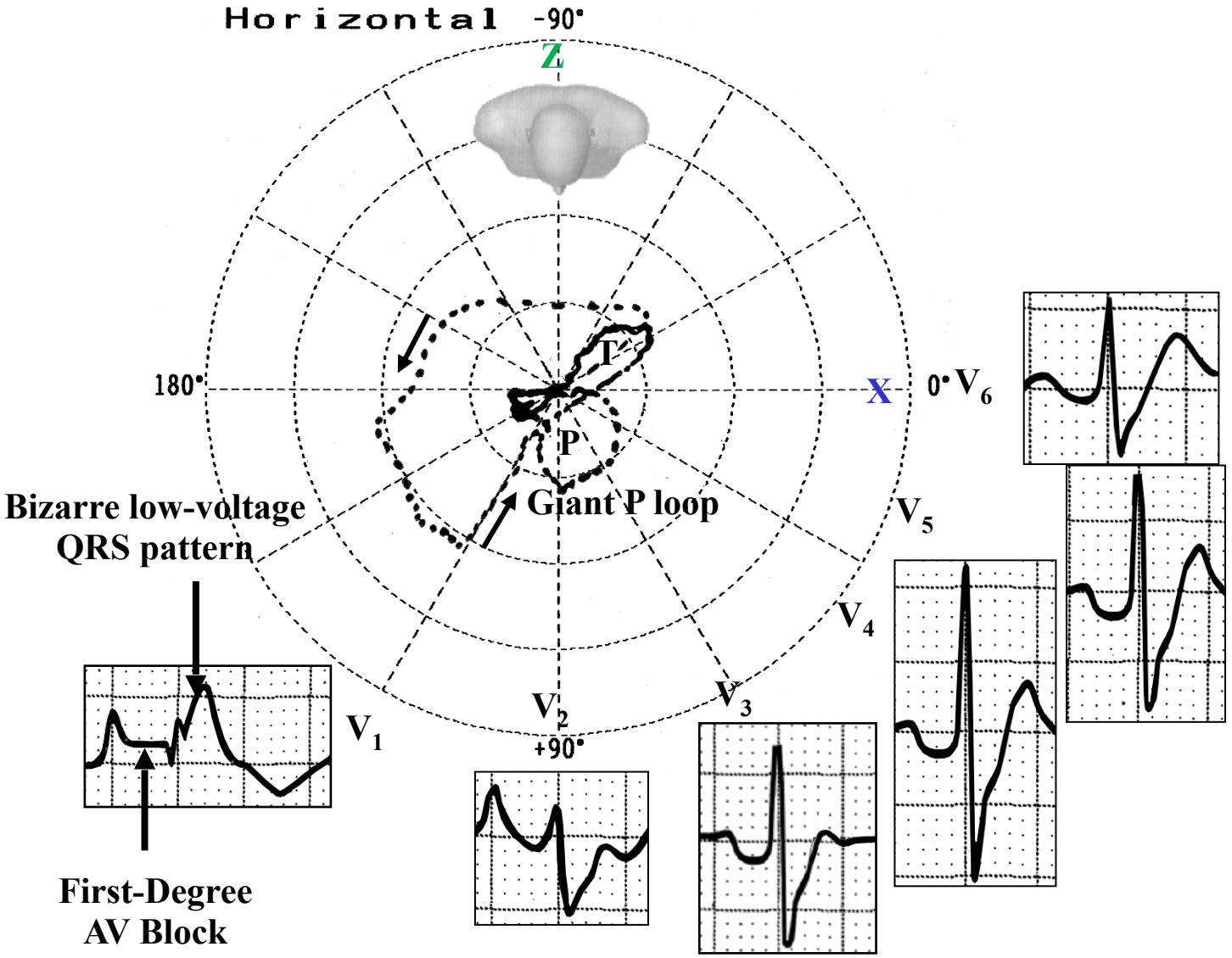
ECG diagnosis: Right atrial enlargement "himalayan P-waves", 1st degree AV block, bizarre right bundle branch block of low voltage in V1 and with initial q wave in this lead. R = S pattern from V3 to V6: RVH.

ECG/VCG correlation in the Frontal Plane



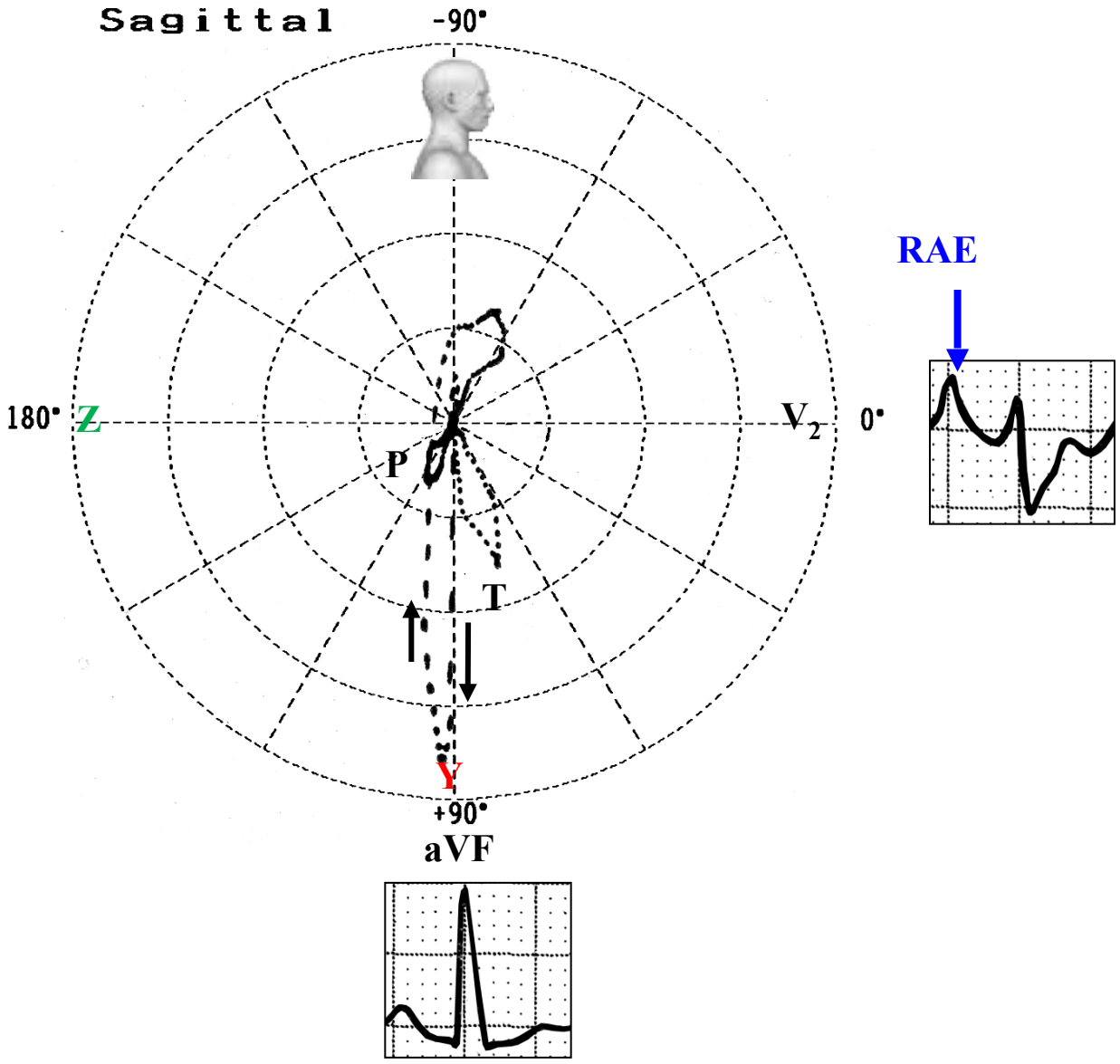
P-wave with 4 mm in II “Himalayan P wave”; very large P loop; right axis deviation.

ECG/VCG correlation in the Horizontal Plane

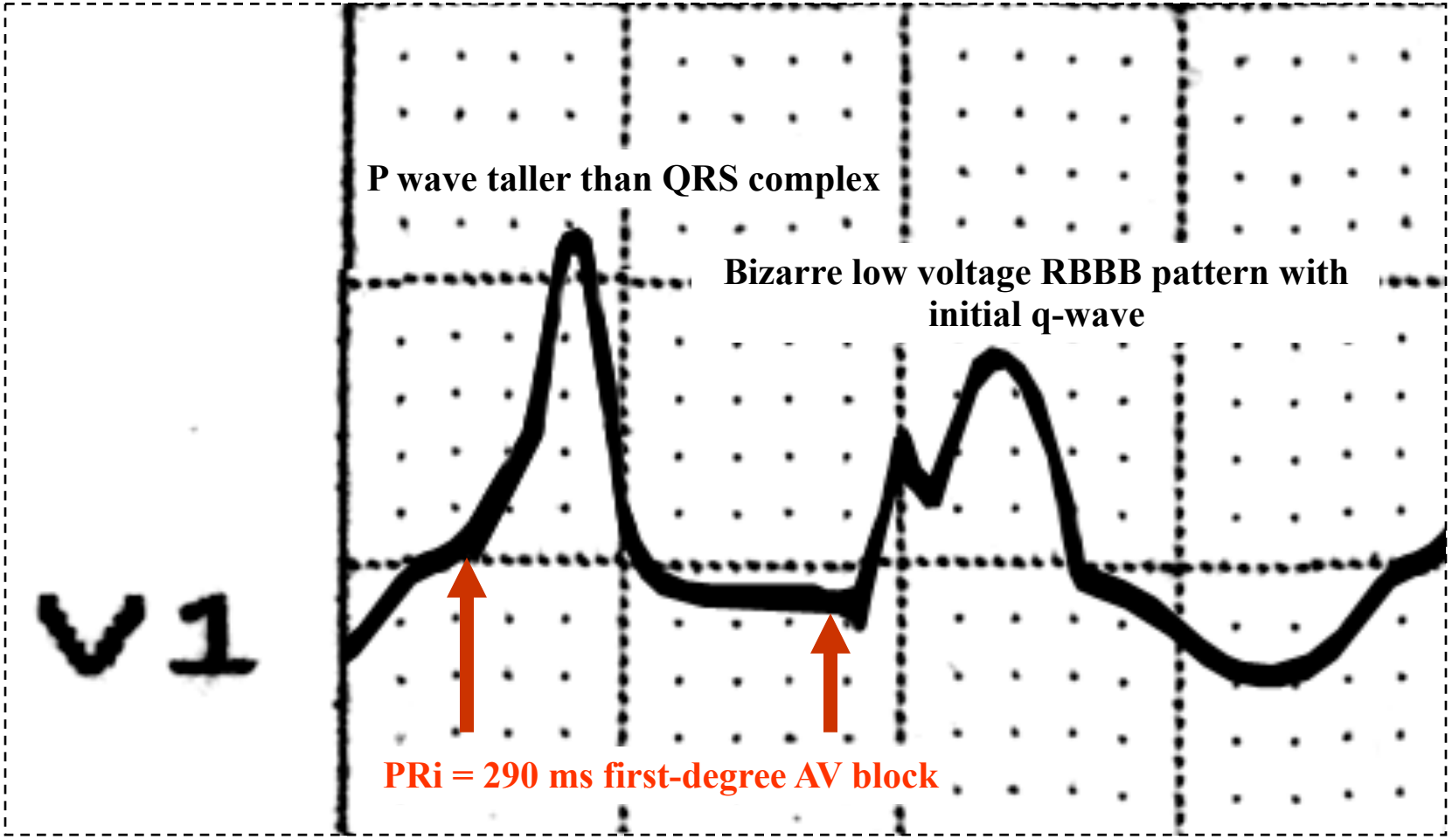


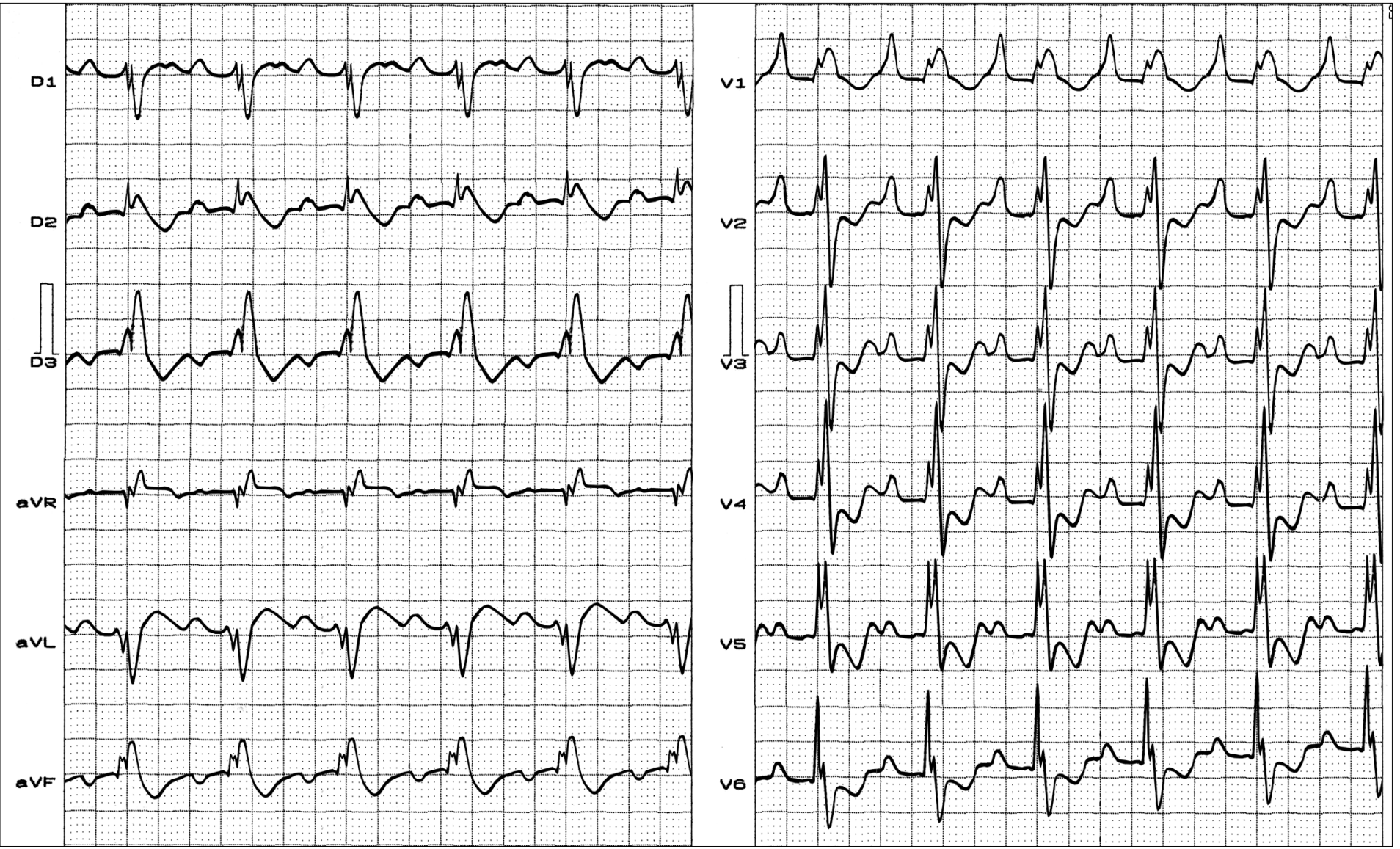
Giant round P loop, very tall P wave in V1-V2, first-degree AV block, bizarre low voltage CRBBB with initial q wave, RS pattern from V3 to V6 and QRS loop predominatly located in the right quadrants: RVH.

ECG/VCG correlation of Right Sagittal Plane



Just with the V_1 lead, which is the diagnosis?





Clinical diagnosis: Ebstein's anomaly of the tricuspid valve.

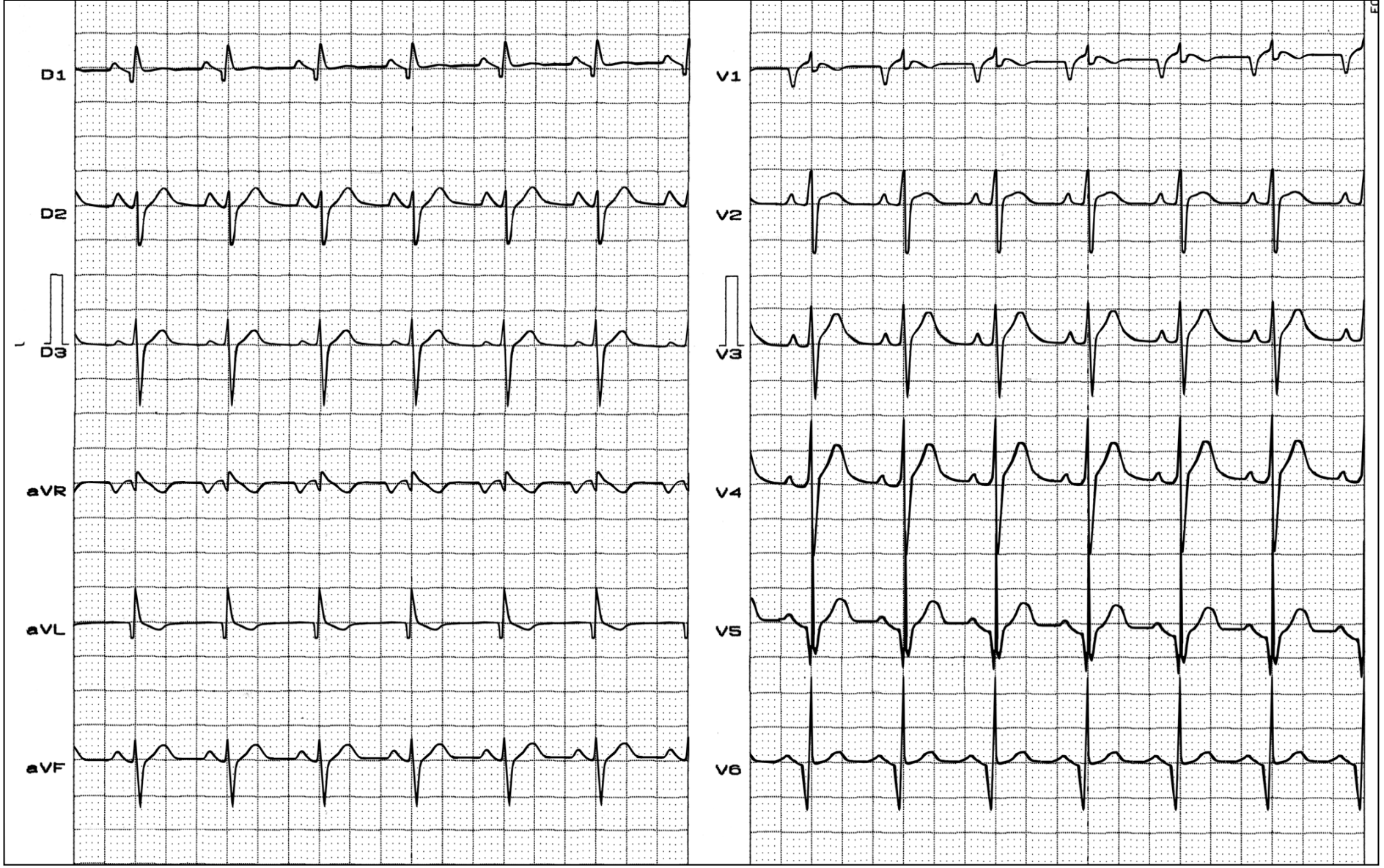
ECG diagnosis: P wave: of great voltage from V_1 to V_4 and in inferior leads: P "Himalayan" waves. SAP-15º, 1st degree AV block. PR: length of 290 ms (1st degree AV block). QRS: bizarre with RBBB of low voltage and with initial small q wave in V1.

The "Gamboa (Gamboa 1966) P wave"

The association of:

1. Right atrial enlargement
2. Diastolic, volumetric or eccentric left ventricular hypertrophy
3. Extreme left axis QRS deviation in the frontal plane: LAFB pattern
4. Counterclockwise rotation of QRS loop in the frontal plane: LAFB pattern
5. Cyanotic baby (neonate or infant). It is very suggestive of tricuspid atresia diagnosis.

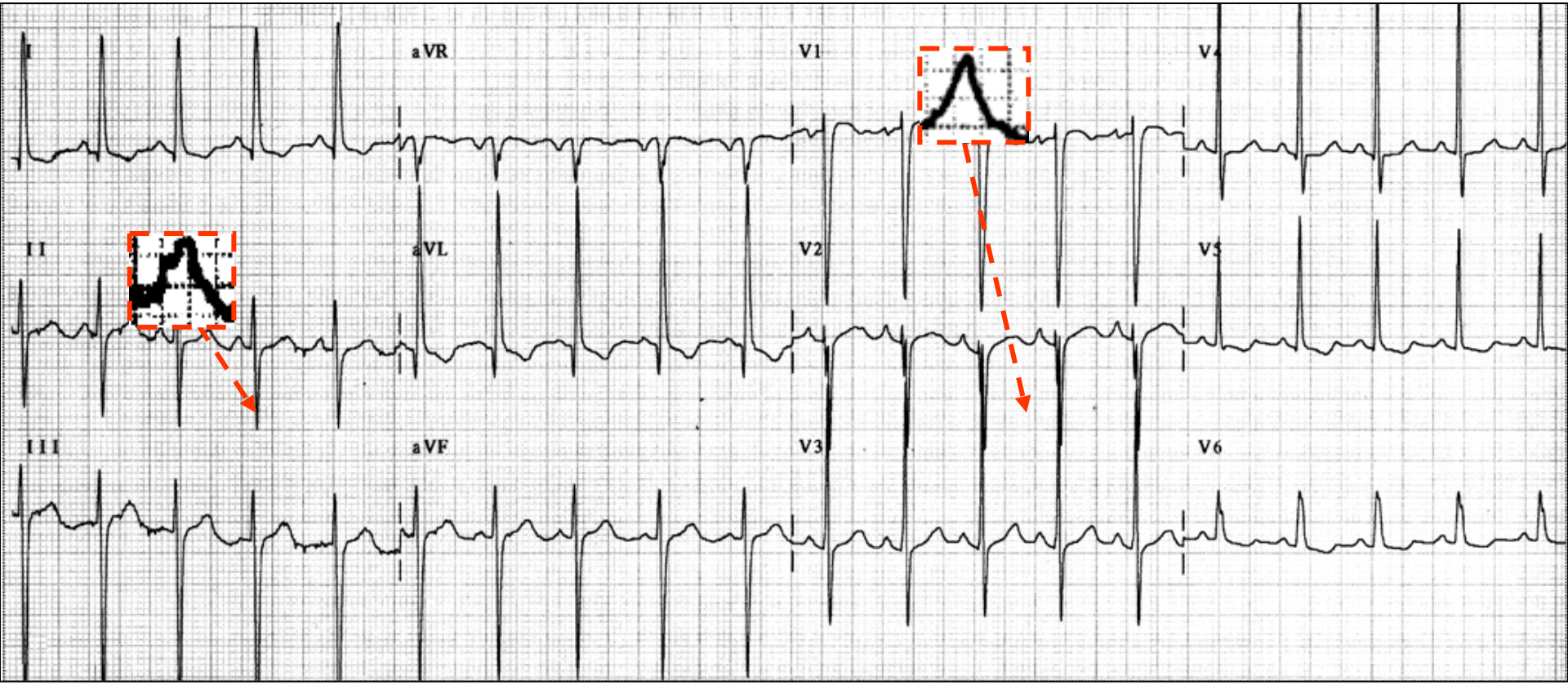
From 120 children with tricuspid atresia, ECG with left axis axis deviation is observed in 94%, ERA in 58%, LVH in 96% and LAE: 47.5% (**Rosado 1987**).



Clinical diagnosis: Tricuspid atresia.

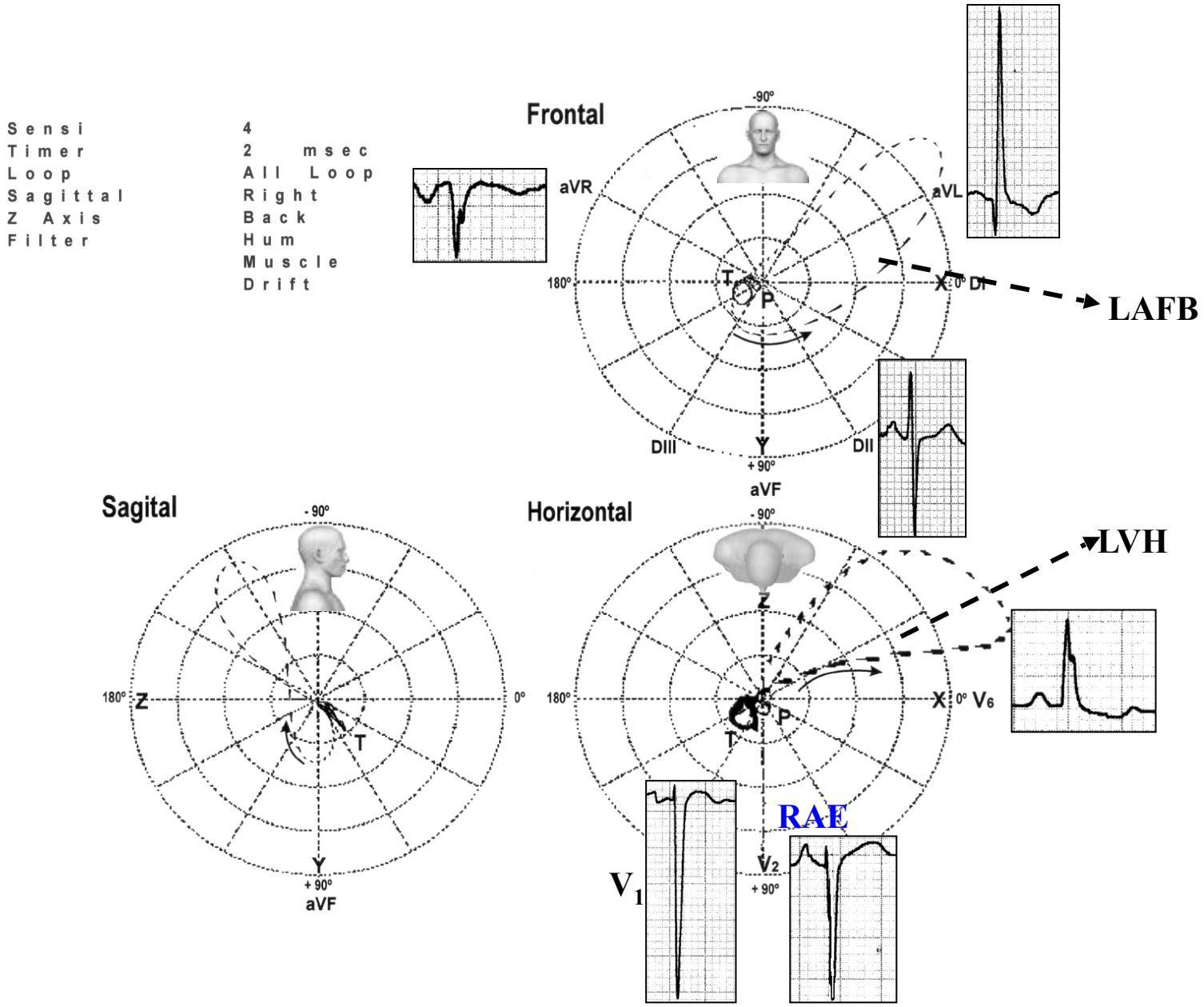
ECG diagnosis: P wave: "tricuspidity of Gamboa" SAP +60º, great voltage in II and in V_2 - V_4. Deep negative component in V1. QRS: axis of QRS with extreme deviation in the left superior quadrant - 65º: LAFB + LVH of the diastolic type: deep Q waves in V5-V6, I and aVL.

Name: LBR; **Age:** 4 y.o.; **Gender:** F; **Ethnic group:** W. **Date:** 12/13/2001; **Weight:** 16 Kg;
Height: 1.10 m **Biotype:** N **Medication in use:** no use of medication.



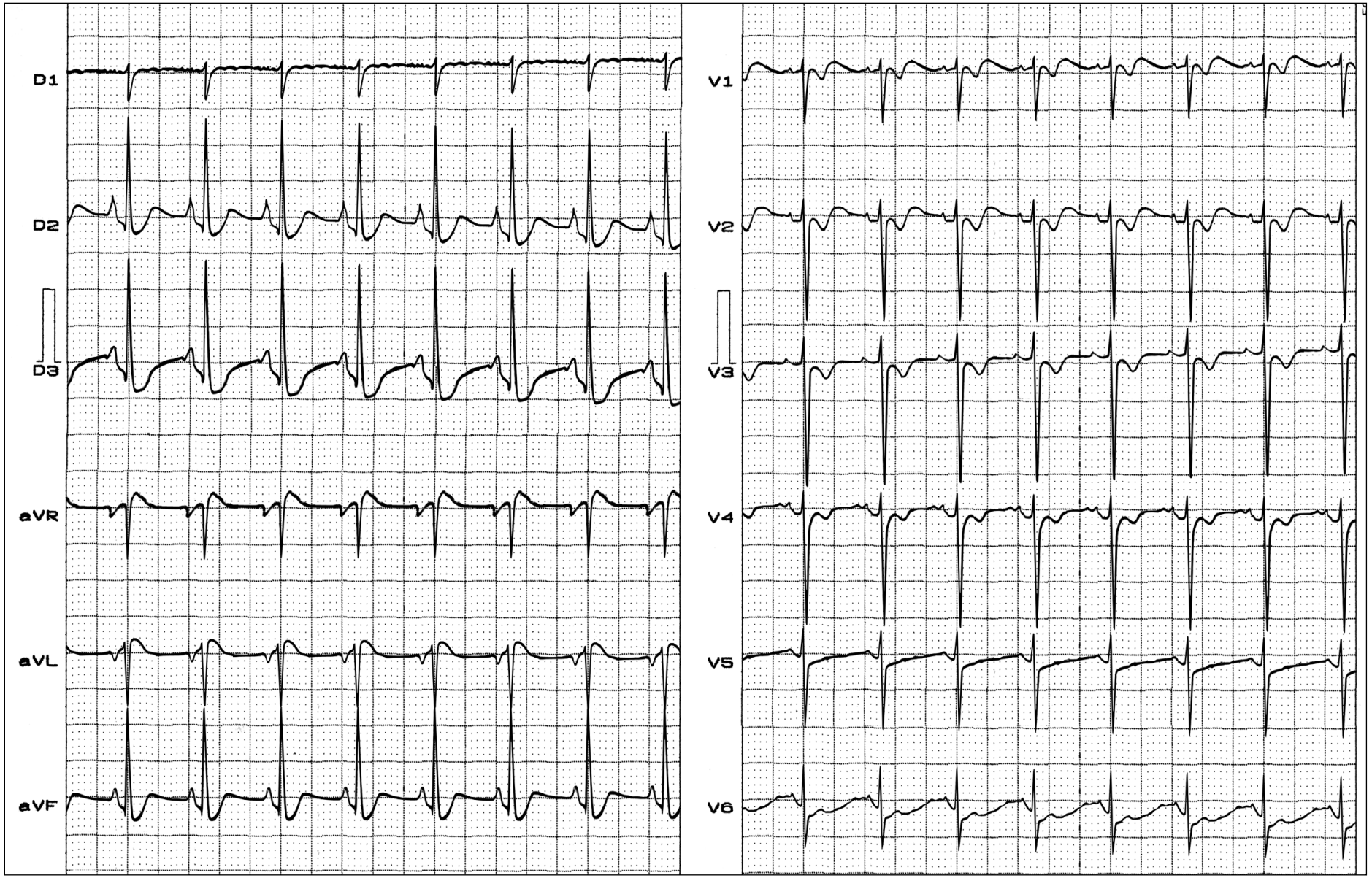
Clinical diagnosis: Tricuspid atresia in a four-year-old child.
ECG diagnosis: RAE: visible in V_2 and with notch in the ascending limb of P wave. LVH: deep S in V_1 and R of increased voltage in V_5. In V_6 is similar to ILBBB. LAFB: SÂQRS with extreme shift in the left superior quadrant and counterclockwise rotation in the FP. qR in I and aVL. rS in inferior leads.
Conclusion: RAE + LVH + LAFB.

ECG/VCG correlation



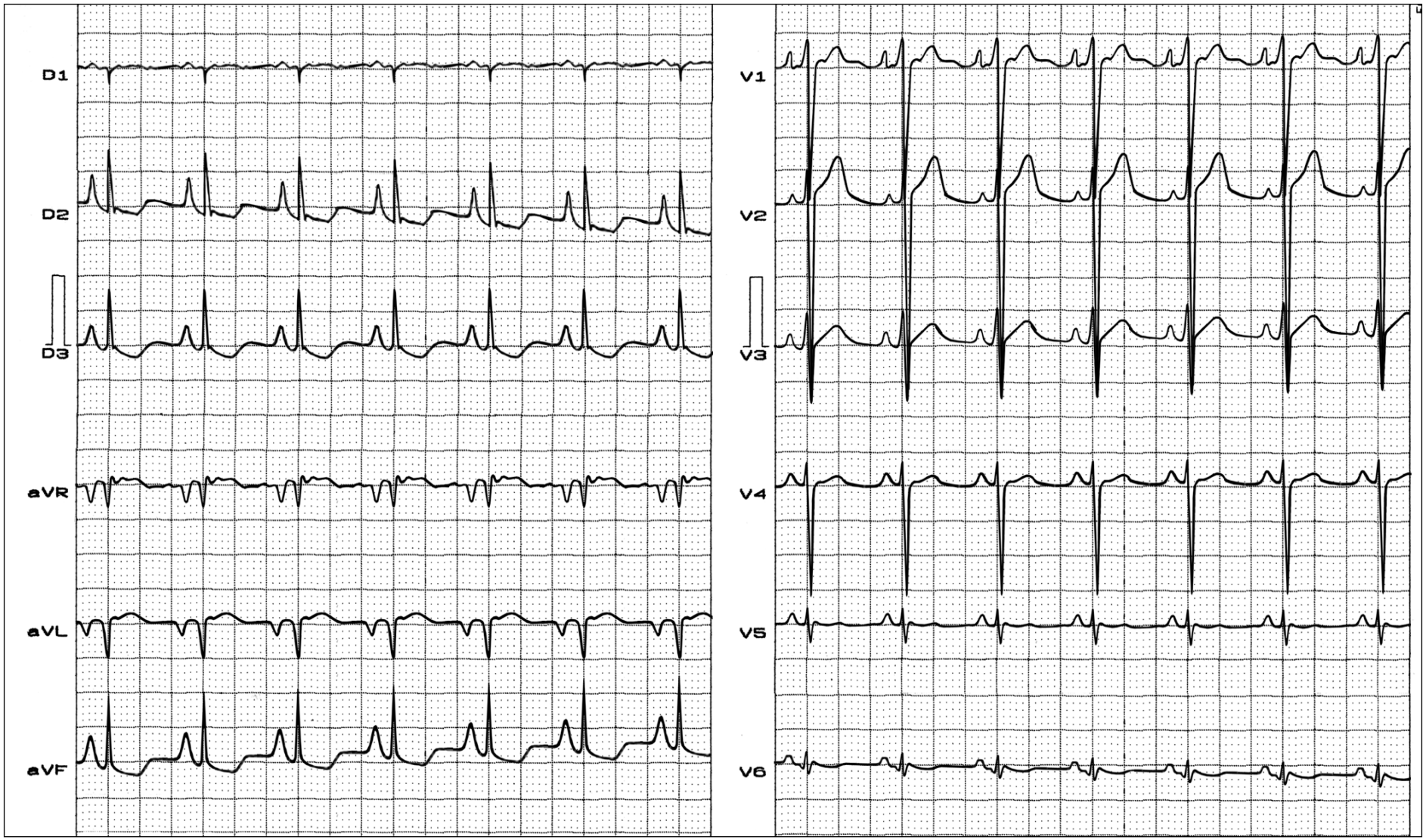
Clinical diagnosis: Tricuspid atresia in a four-year-old child.

ECG/VCG diagnosis of TA: RAE + LVH with deep initial Q-wave in aVL+ LAFB.



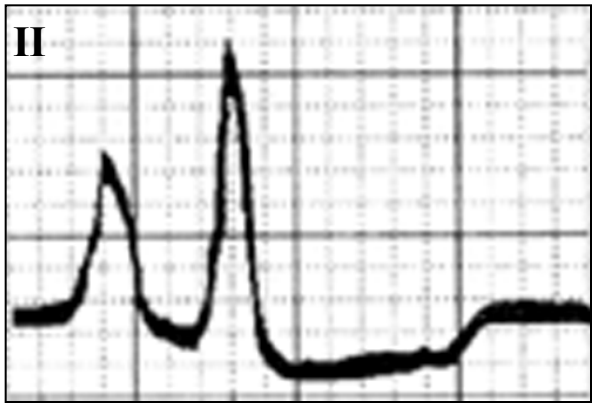
Clinical diagnosis: Chronic severe COPD cor pulmonale.
ECG diagnosis: P wave: of great voltage in inferior leads. SÂP + 90º. QRS: RVH type C, rS from V_1 to V_5. SÂQRS + 105º and dislocated to the back.

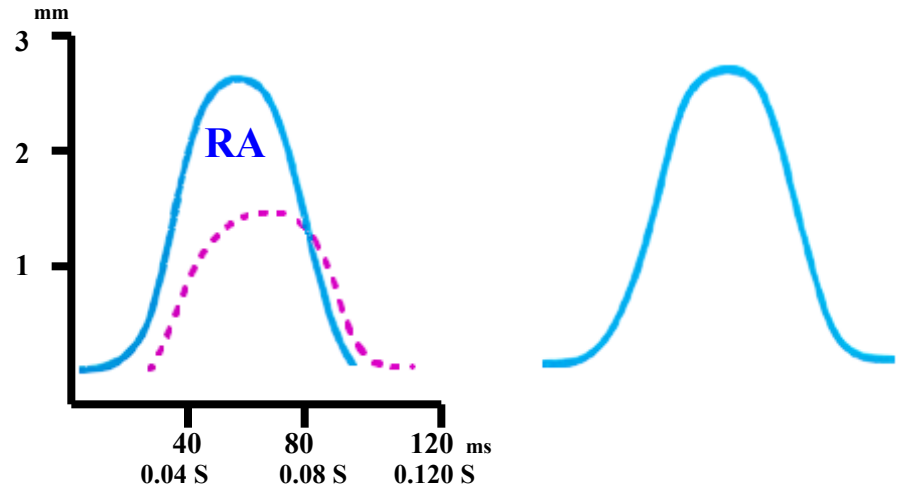
Name: **ASS; Date:** 02/05/91; **Number:** 102-03; **Age:** 70 y.o.; **Gender:** F; **Race:** W; **Weight:** 35 Kg.
Height: 1.42 m;

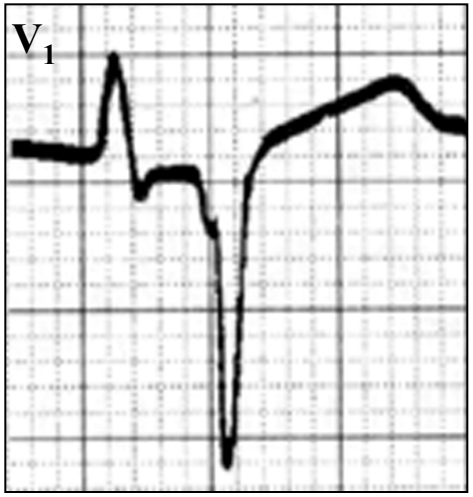


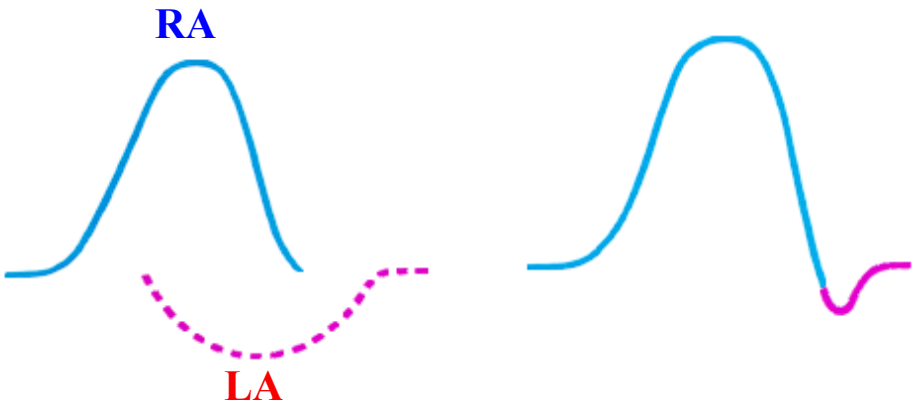
Clinical diagnosis: COPD, emphysema.
ECG diagnosis: P wave: SAP: $+90^0$. Voltage: 4 mm in II: "P pulmonale": RAE + RVH type C

P wave of RAE in II and V1









Right Atrial Enlargement criteria (RAE)

I) Direct ECG criteria: Direct P wave criteria are very specific but their sensitivity is very low.

- Voltage of P ≥2.5 mm in at least one of inferior leads: P pulmonale or P pulmonale parenchymal: tall and occasionally pointed P wave in inferior leads
- Aspect in apex of P wave: "Goth P"
- P wave height >1.5 mm in lead V_2. The criteria has 100% specificity preserved
- P waves with "plus-minus" pattern in right precordial leads with initial plus component ≥1.5 mm
- P wave deeply negative or positive in V_1
- P wave of voltage ≥ at 1.5 mm in V_2 in association to R/S ratio >1
- SÂP to the right of +80º (negative P wave in VL). In congenital heart diseases, SAP is not deviated to the right.
- P wave with increase in voltage and in duration in cases of extreme RAE
- Macruz index lower than 1 = $^{\text{P duration}}/_{\text{PRs duration}}$ (Macruz 1958)
- The QRS criteria with a QRS amplitude in V1 <4 mm + ratio V2/V1 > 5 are highly specific (>90%) with moderate SE (≈ 45 %)
- The combined P + QRS criteria with a P wave amplitude in V2 > 1.5 mm + SÂQRS > 90° + R/S ratio >1 in V1 in the absence of RBBB have 100% specificity and ≈ 50% sensitivity.

II) Indirect ECG criteria

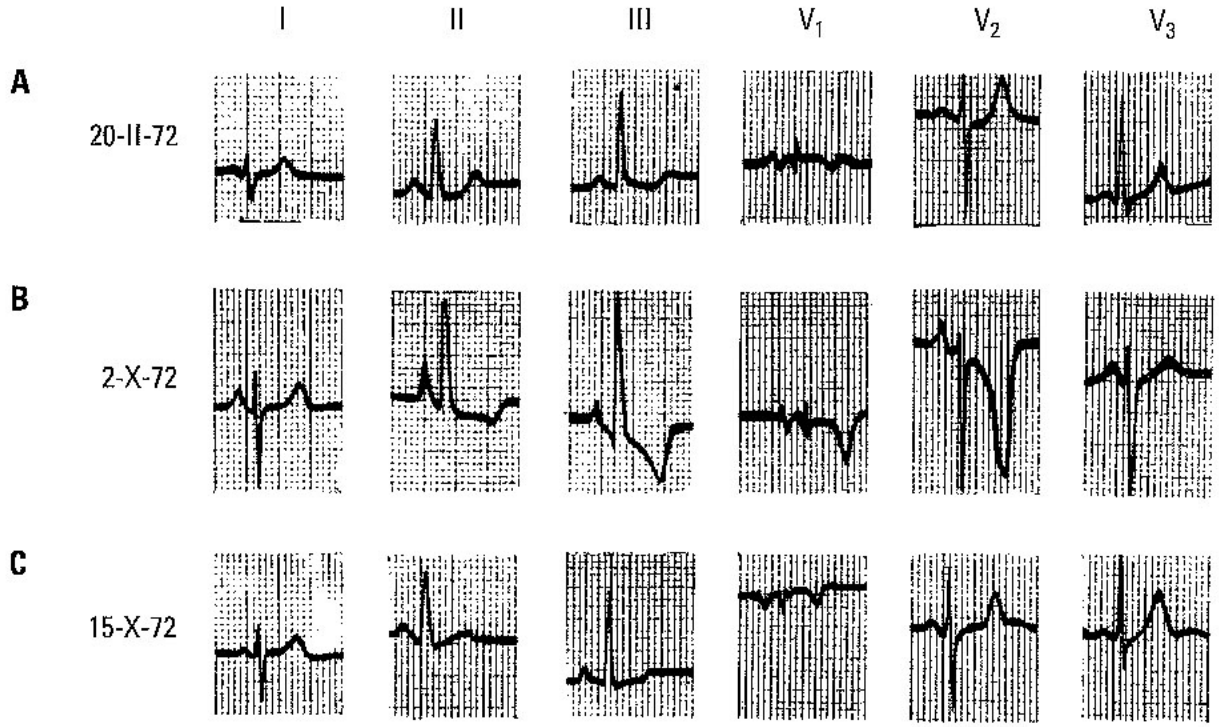
- $SÂQRS_F$: > 90º.The criteria has 100% specificity preserved
- Sodi Pallares sign[1]: qR, QR or qRs in V_1 and V_2
- Peñaloza and Tranchesi sign: QRS complexes of low voltage in V_1 contrasting with QRS complexes of normal voltage or increased in V_2.
- R/S ratio >1 in lead V_1 without RBBB. The criteria has 100% specificity preserved.

False positive and false negative diagnoses of right atrial enlargement

The ECG diagnosis of RAE may be very difficult to reach for the following reasons:

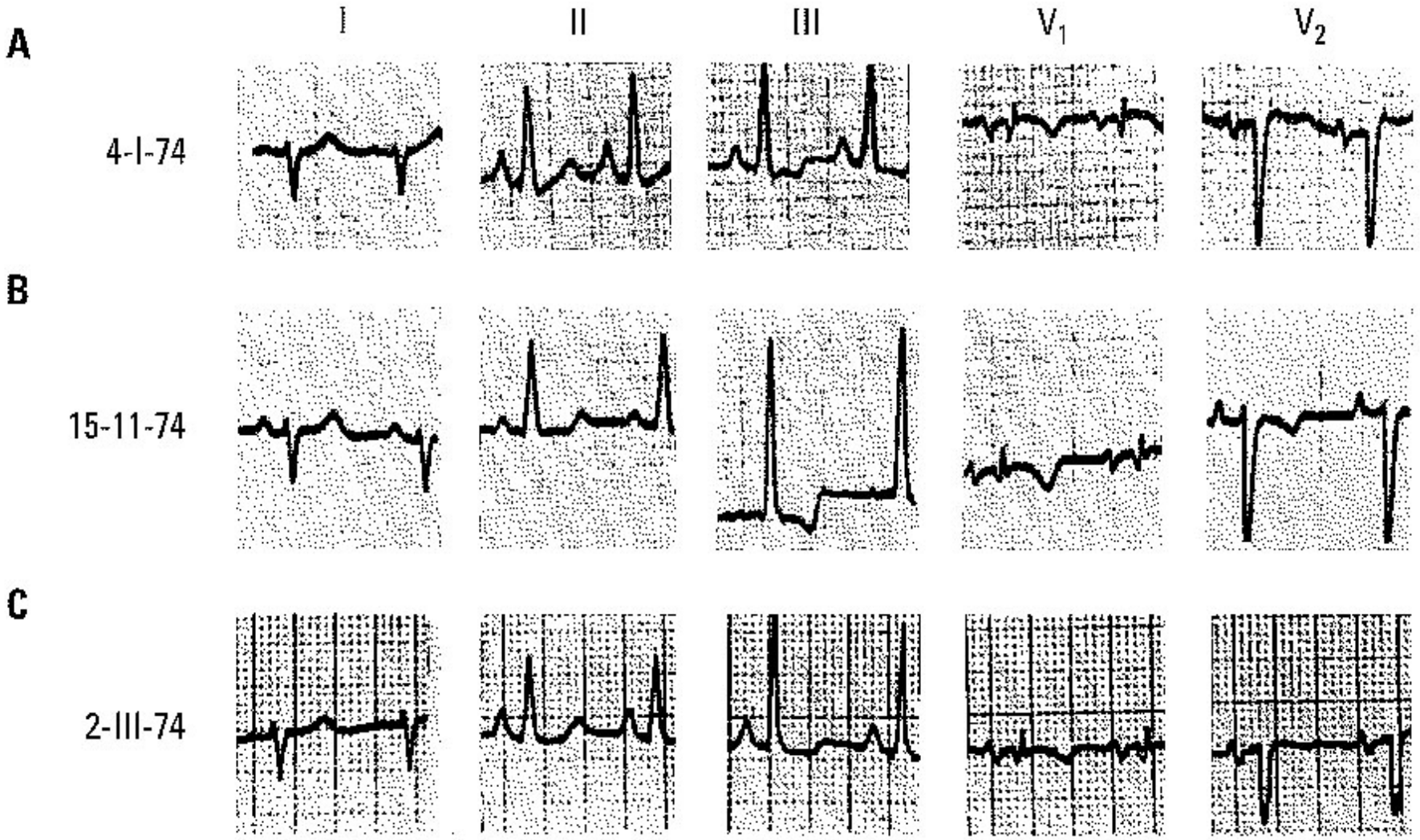
- The voltage of the P wave is strongly influenced by extra cardiac factors (Figure below), which may result in increases (hypoxia, sympathetic overdrive, etc.) (false positive) or decreases in voltage (emphysema, other barrier factors, atrial fibrosis, etc.) (false negative)
- The presence of associated fixed or intermittent atrial block may result in the transient or permanent disappearance of the ECG criteria for right atrial enlargement (false negative) (Figure in next slide)
- On the other hand, a high-voltage P wave may be seen in patients with exclusively left heart pathology and possible left atrial enlargement (false positive) (pseudo-P-pulmonale) (**Bayés de Luna 2011**)

These are some of the reasons why changes in the atriogram are generally not very sensitive (many false negatives) for the diagnosis of RAE. Although there are some factors that increase the incidence of false positives, they are fewer and therefore the specificity of ECG criteria for RAE is much higher.



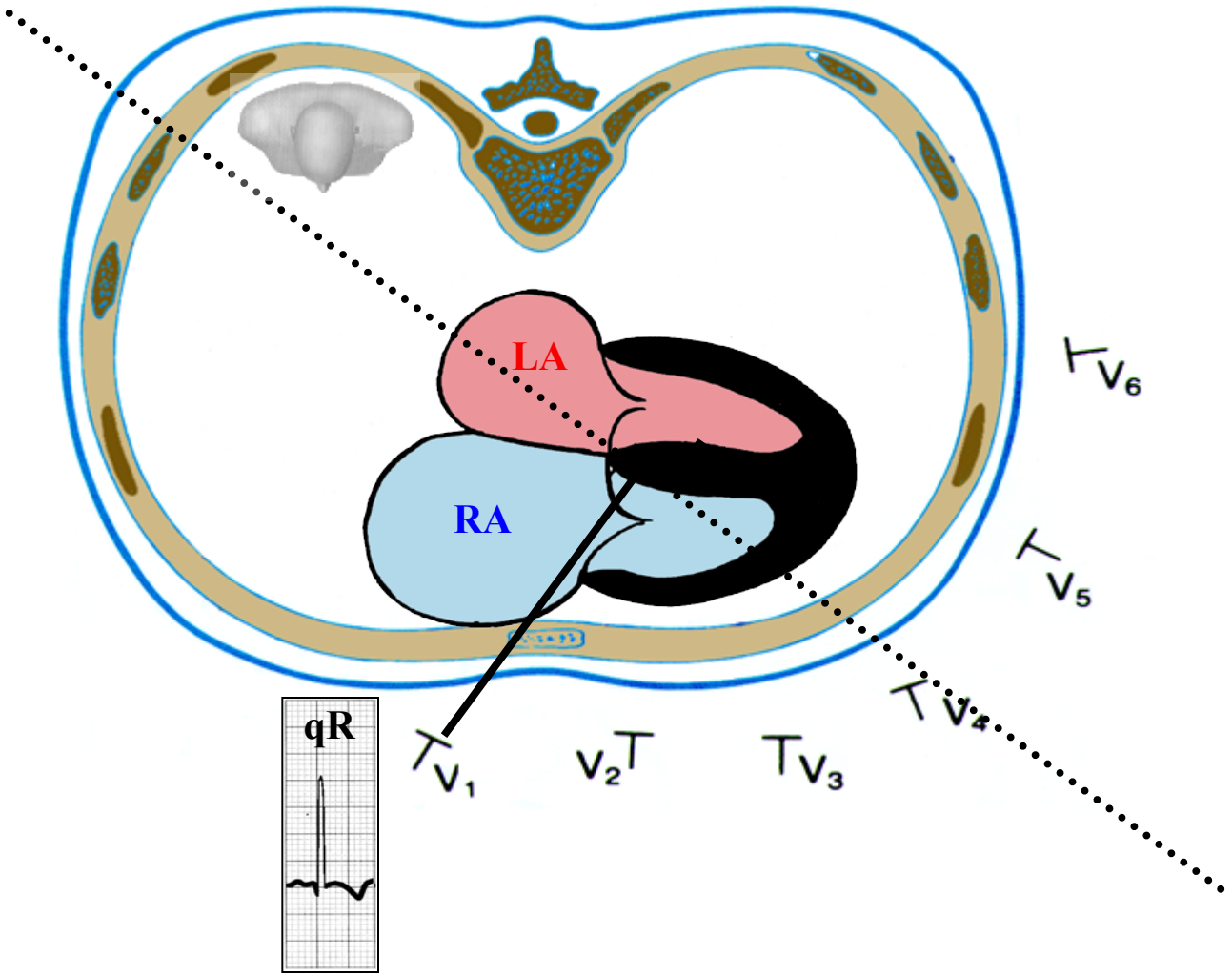
Clinical diagnosis: patient with chronic cor pulmonale and an acute respiratory affection.

ECG diagnosis: A tall, peaked P wave (B) that did not previously exist (A) appeared, disappearing a few days later (C). Observe how the negativity of the T wave increases in V1 and V2 in the B tracing.



Clinical diagnosis: A 45-year-old patient with subacute cor pulmonale.
ECG diagnosis: Note the right SÂP. Some days later (B), the SÂP was left, returning to the right in a third ECG (C) recorded at 15 days. This example shows how P waves that fail to suggest right atrial enlargement can be seen in cases where the right cavities are affected by right atrial enlargement due to atrial aberration.

Significant dilatation of Right Atrium: Indirect sign of RAE conditioning qR pattern in V_1 and V_3R (Sodi-Pallares' sign) (Sodi-Pallares 1952)



Outline that explains the indirect sign of RAE: qR in V1 (sign of Sodi-Pallares). The volumetric increase of the RA, gets closer to the exploring electrode V_1, recording initial QRS negativity in this lead, because this electrode records the epicardial morphology of the right atrium.

Significance of RAE in Congestive Heart Failure (CHF)

Patients with electrocardiographic pattern of RAE, atrial flutter, AF, 1st or 2nd degree AV block Mobitz Type I, Complete LBBB and interstitial pulmonary edema with bilateral pleural effusion in chest X-rays, frequently associated to CHF(**Fonseca 2004**).

Atrial high resolution ECG

The duration of P wave in high resolution ECG (signal-averaged P wave (SAPWD)) significantly correlates with the size of the right atrium.(**Dixen 2004**)

Pseudo-P-pulmonale

These cases are frequently misdiagnosed. In pure left atrial overload rarely a very high P wave (≥ 2.5 mm in inferior leads) is observed with right axis deviation (P axis located to the right of +60°: negative P in VL) and major duration (≥120 ms).

The vector of left atrial activation is directed not only posteriorly, but also more inferiorly than to the left pointing abnormally downwards.

Thus a P wave with two humps is observed: The first one, registered within the first 40 ms and responsible for a slight notch corresponds to the first part of the normal right atrial activation, while the second greater hump corresponds to the activation of the enlarged left atrium (**Chow 1965, Chung 1972, Gertsch 2003**).

P-pulmonale-like patterns causes without RAE

- Severe hyperkalemia (see next slide for explanation)
- Aesthenic habitus
- Enhanced sympathetic tone
- Cyanosis.

Severe hyperkalemia imitating P-pulmonale

Hyperkalemia is one of the more common acute life-threatening metabolic emergencies seen in the emergency department.

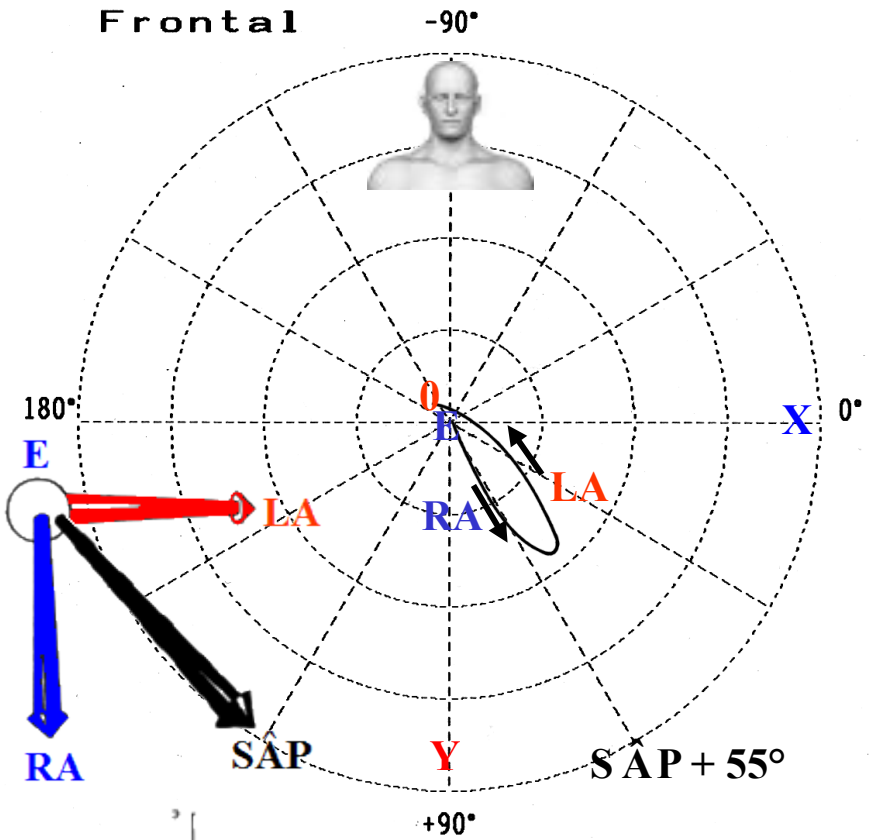
Early diagnosis and empirical treatment of hyperkalemia is dependent in many cases on the emergency physician's ability to recognize the ECG manifestations.

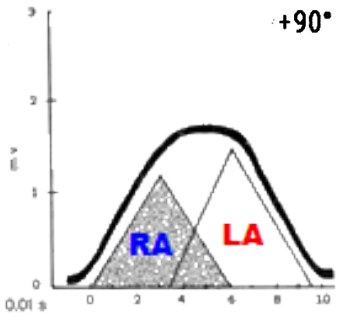
The ECG manifestations of hyperkalemia include (**Mattu 2000**):

- Diminished, flattening or absence of the P wave (sinoventricular conduction)
- Exceptionally enhanced P wave amplitude in the inferior leads imitating P pulmonale (**Gertsch 2003**).
- PR interval prolongation
- Widening of the QRS complexes
- A "sine-wave" appearance at severely elevated levels. The possible mechanism for the genesis of the sine wave, including loss of electrical gradient with resulting phase difference of QRS and T, associated with maintenance of His bundle activity with progressive, distal, Purkinje blockade (**Sridharan 1979**).
- Peaked and narrow based T waves in the precordial leads

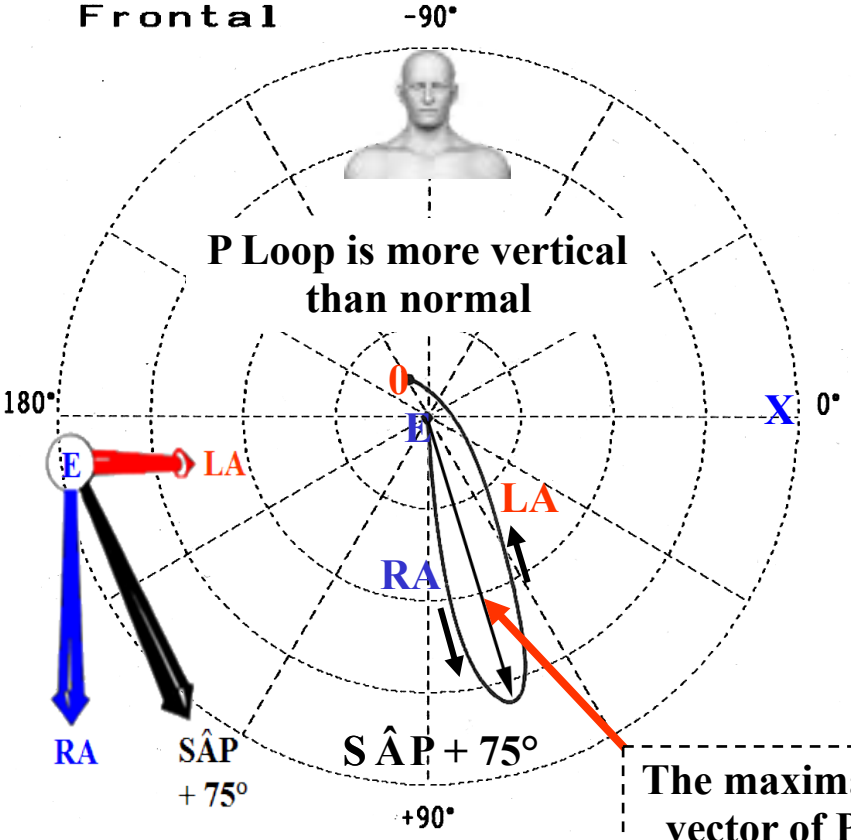
P loop in Right Atrial Enlargement in the Frontal Plane

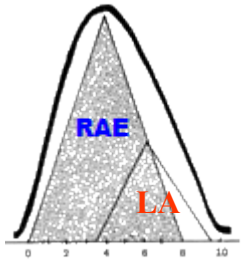
Normal P loop

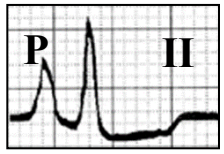




P loop in Left Atrial Enlargement



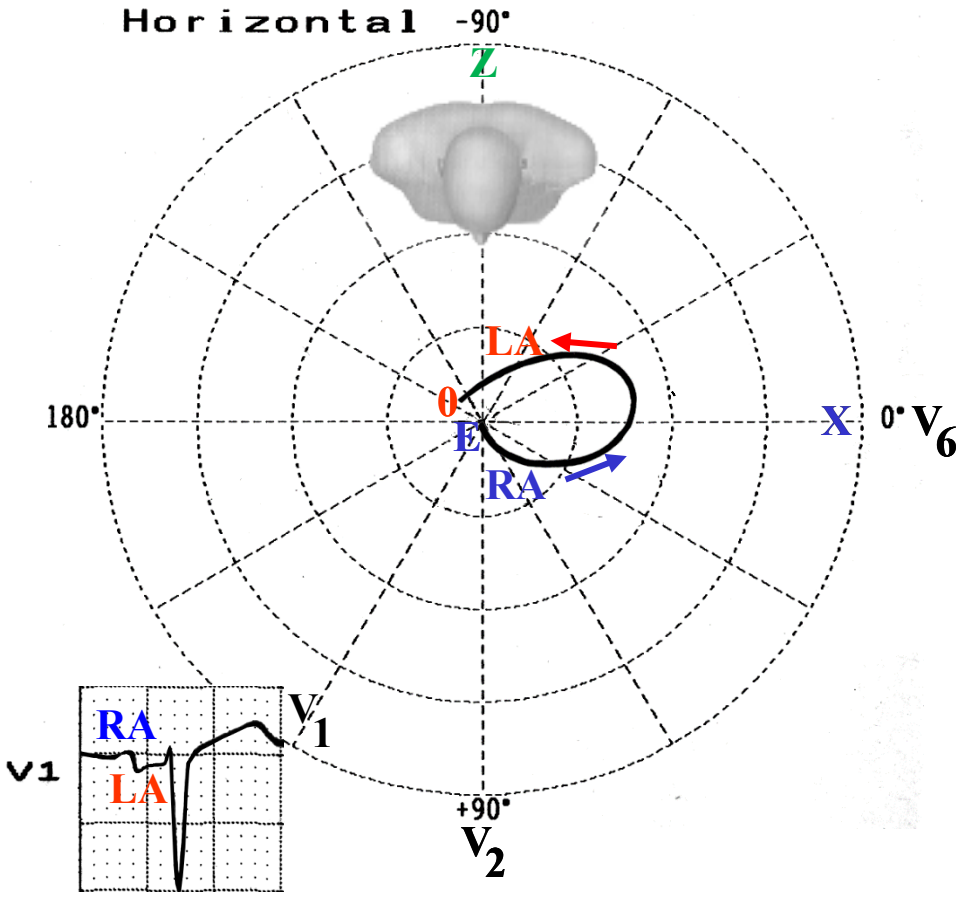




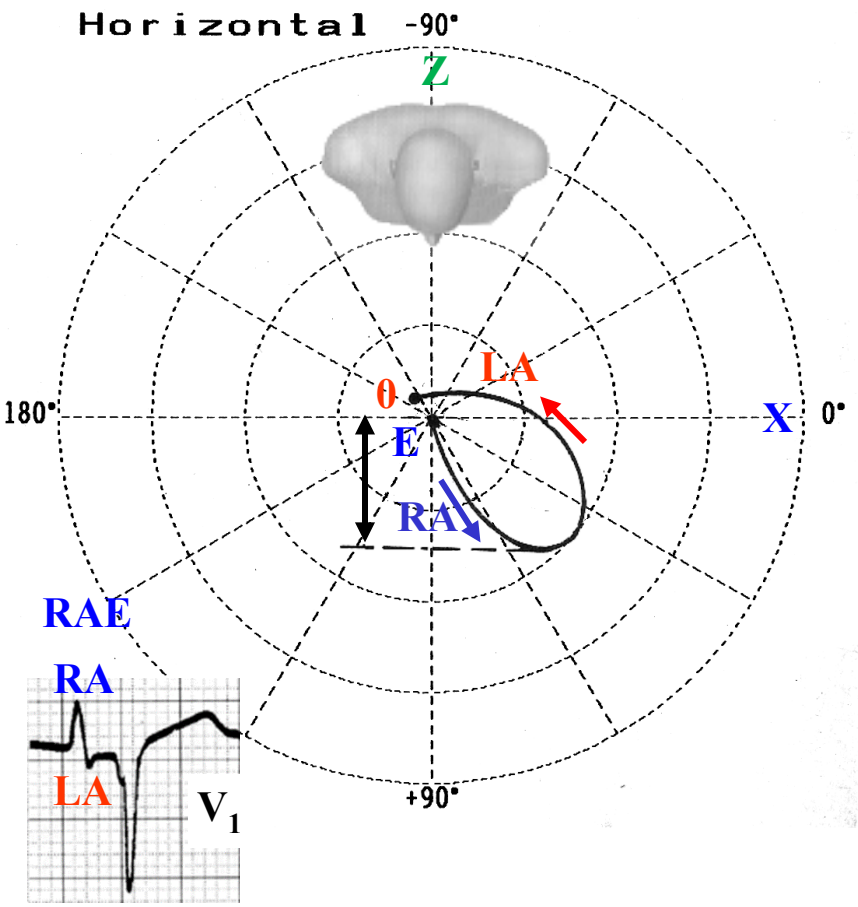
The maximal vector of P exceeds >0.2 mV

P loop in Right Atrial Enlargement in the Horizontal Plane

Normal P loop

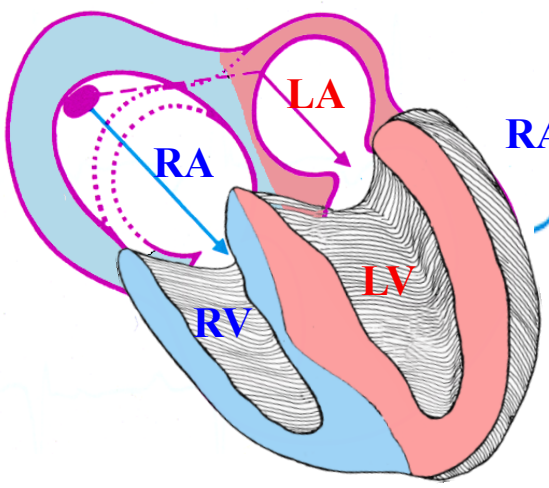


P loop in Left Atrial Enlargement



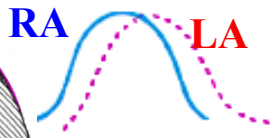
Maximal vector of P may exceed >0.1 mV
Most of the P loop heads to the front.
Magnitude of anterior forces: ≥0.07 mV
Max. ant. forces ≥0.07 mV

P-wave components in RAE

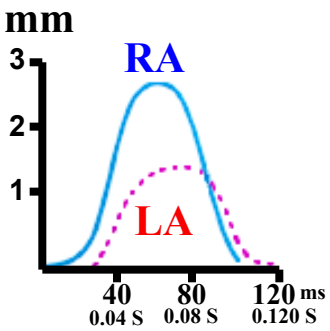


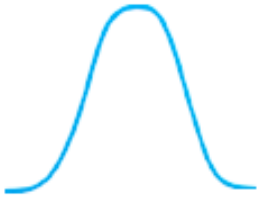
Normal

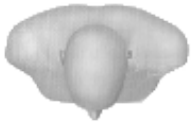
II

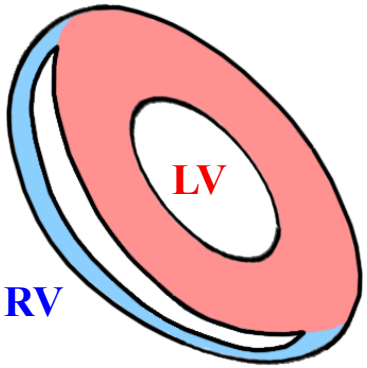


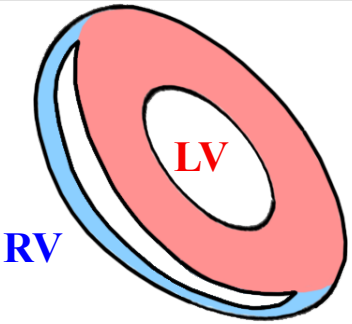
RAE



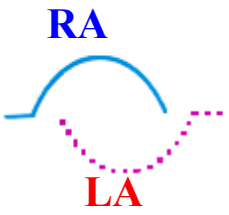






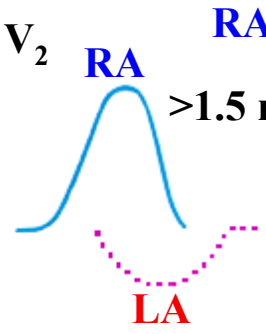


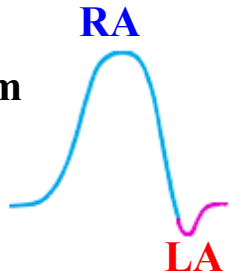
V_1



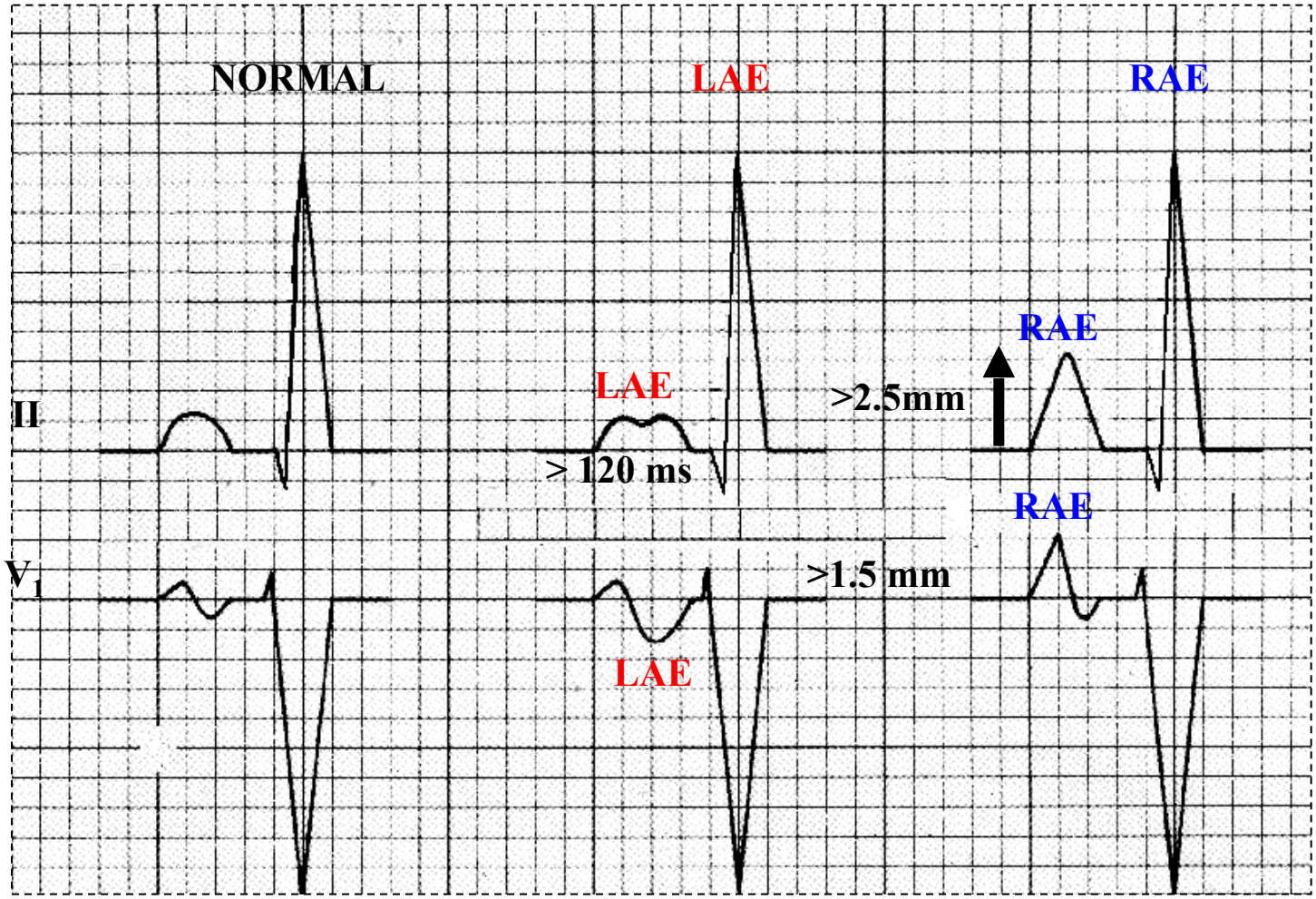
V_2

RAE





Normal P wave in II and V_1 and in LAE and RAE

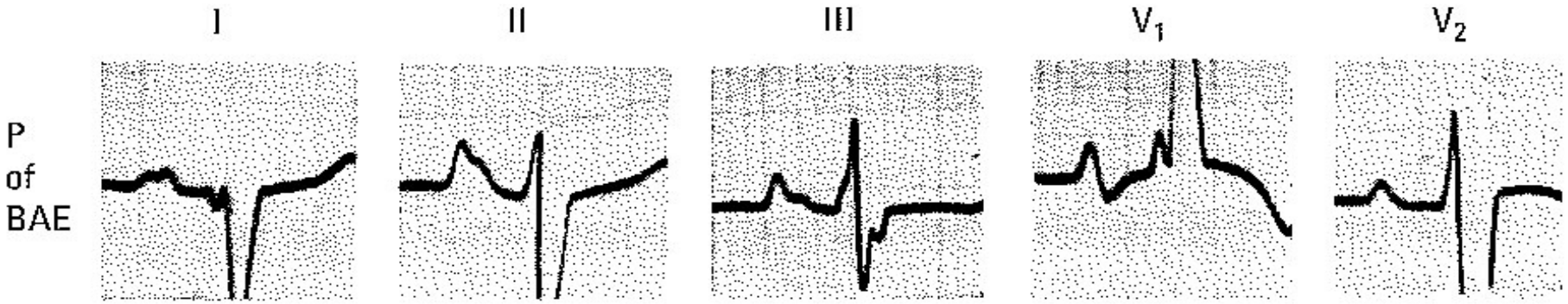


Biatrial Enlargement (BAE) electrocardiographic criteria

The most important diagnostic criteria are as follows

1. Initial component of P wave in V1 >1.5 mm and final slow and deep negative component > 1 mm in depth and 40 ms in duration
2. P waves of voltage >2.5 mm and duration ≥120 ms in II
3. Initial part of P waves is peaked in V_1 and V_2 with voltage >1.5 mm and terminal negative mode slow (width ≥1mm)
4. Signs of LAE (P waves of duration >120 ms and bimodal) with right SÂP. The opposite case is not valid because the SÂP can be on the left side in isolated RAE of patients with congenital heart diseases.
5. Atrial Fibrillation associated with qR and QR type complexes in right precordial leads in the absence of myocardial infarction in V_1 or V_1-V_2 (Sodi Pallares sign) (**Sodi Pallares 1952**)
6. QRS complexes of low voltage in V_1 contrasting with QRS complexes of normal voltage or increased in V_2.

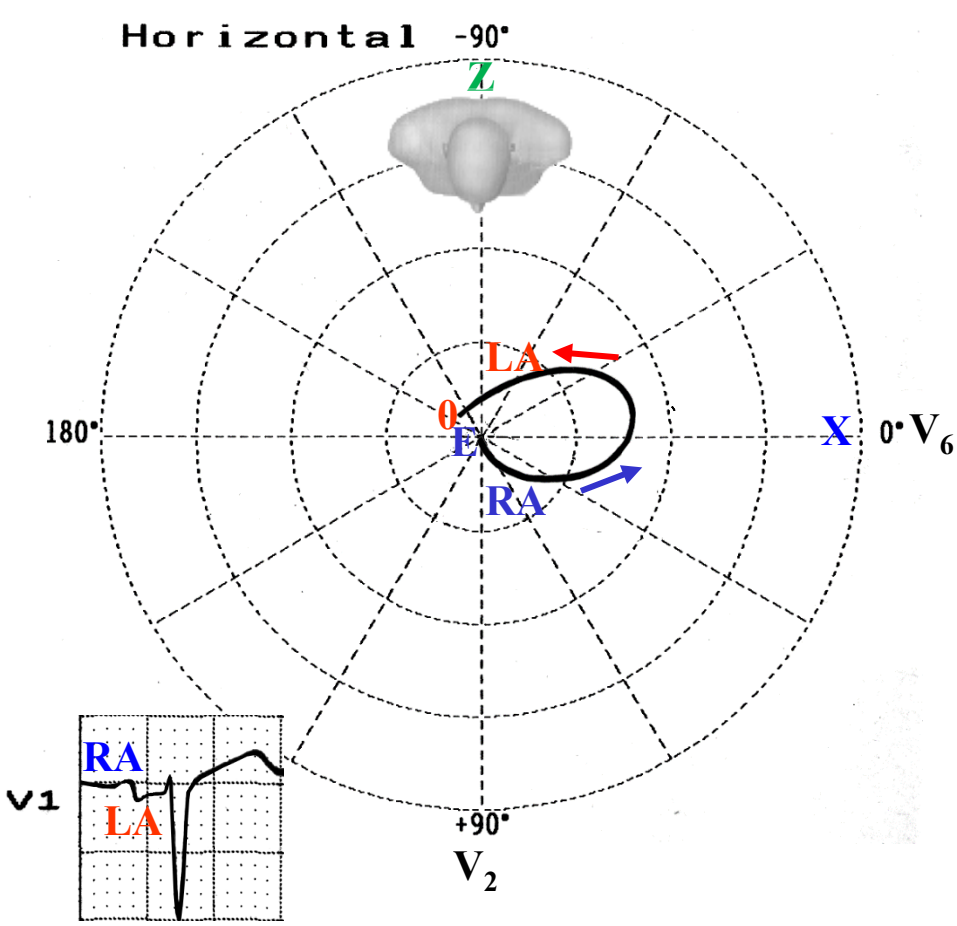
Frequently, more than one criterion is found (Figure 6) (P duration ≥ 120 ms in FP + P ± in V1 with first part peaked and the second part broad and deep).



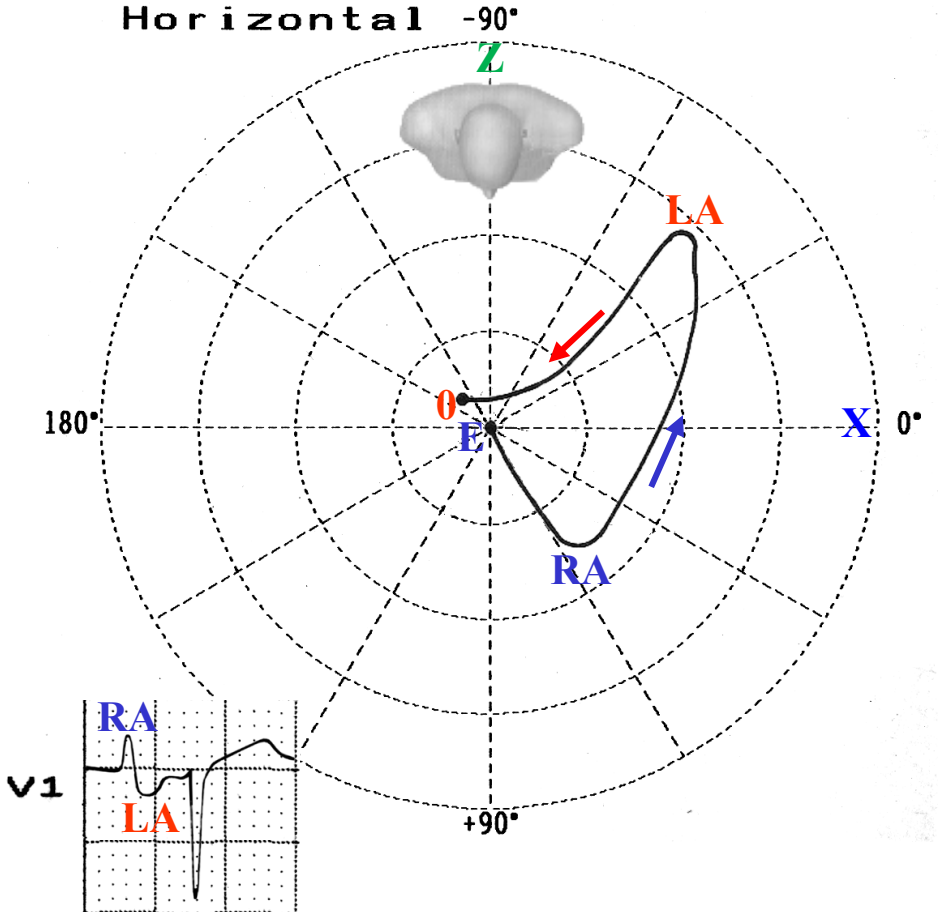
Example of P-wave morphology with biatrial enlargement (BAE).

P loop in Biatrial Enlargement in the Horizontal Plane

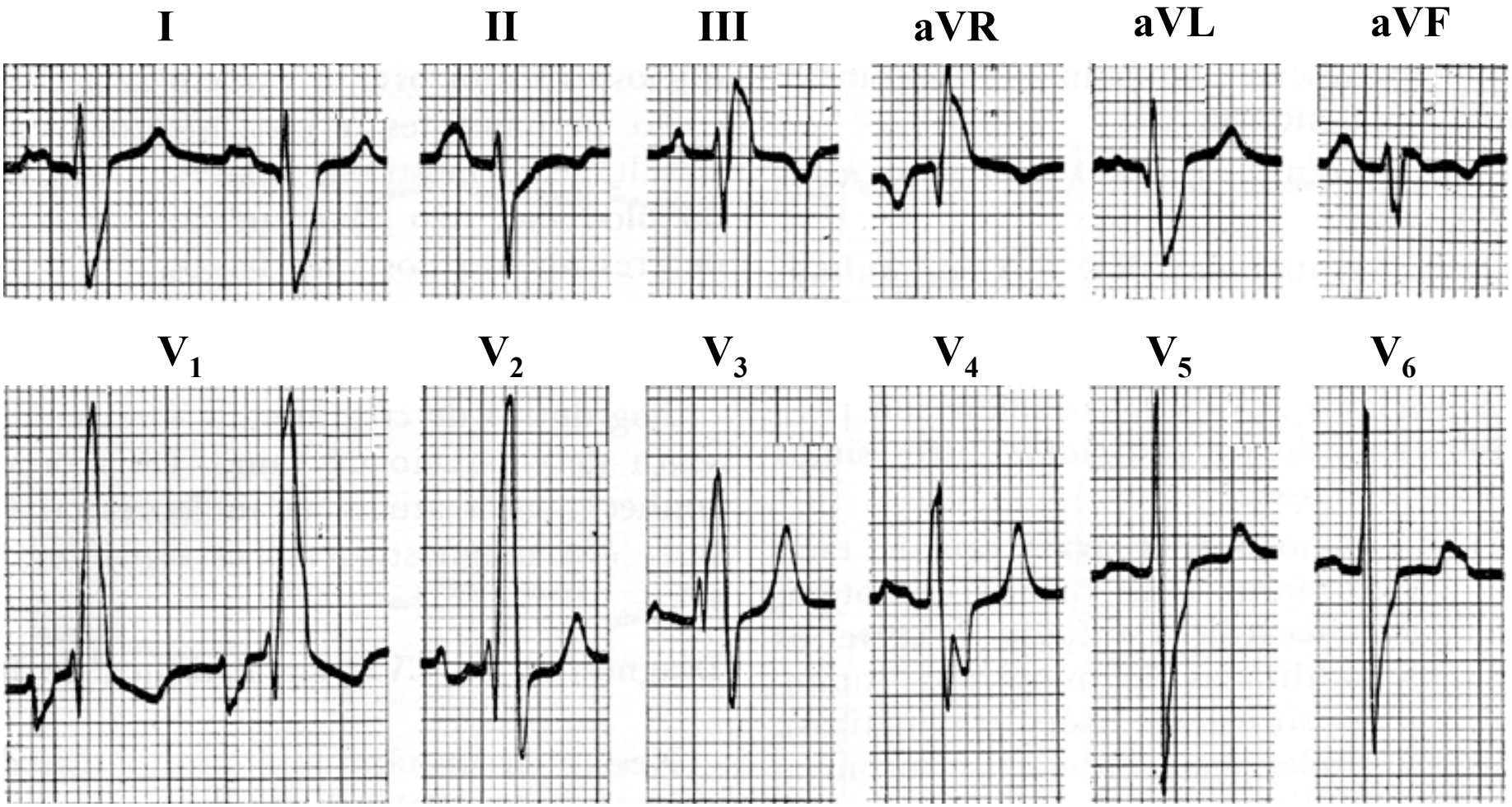
Normal P loop



P loop in Left Atrial Enlargement



Increase of anterior and posterior voltages of P loop



Clinical diagnosis: Rheumatic valvular mitrotricuspid disease.
ECG diagnosis: Sinus rhythm, SÂP +60°, P duration 120 ms, P voltage 3 mm, negative deep and slow final component in V1: biatrial enlargement, QRS axis +170°, QRS duration 160 ms, rsR' in V1, broad final S wave in V1, aVL, V5-V6, and final broad R wave in aVR: RVH and CRBBB.

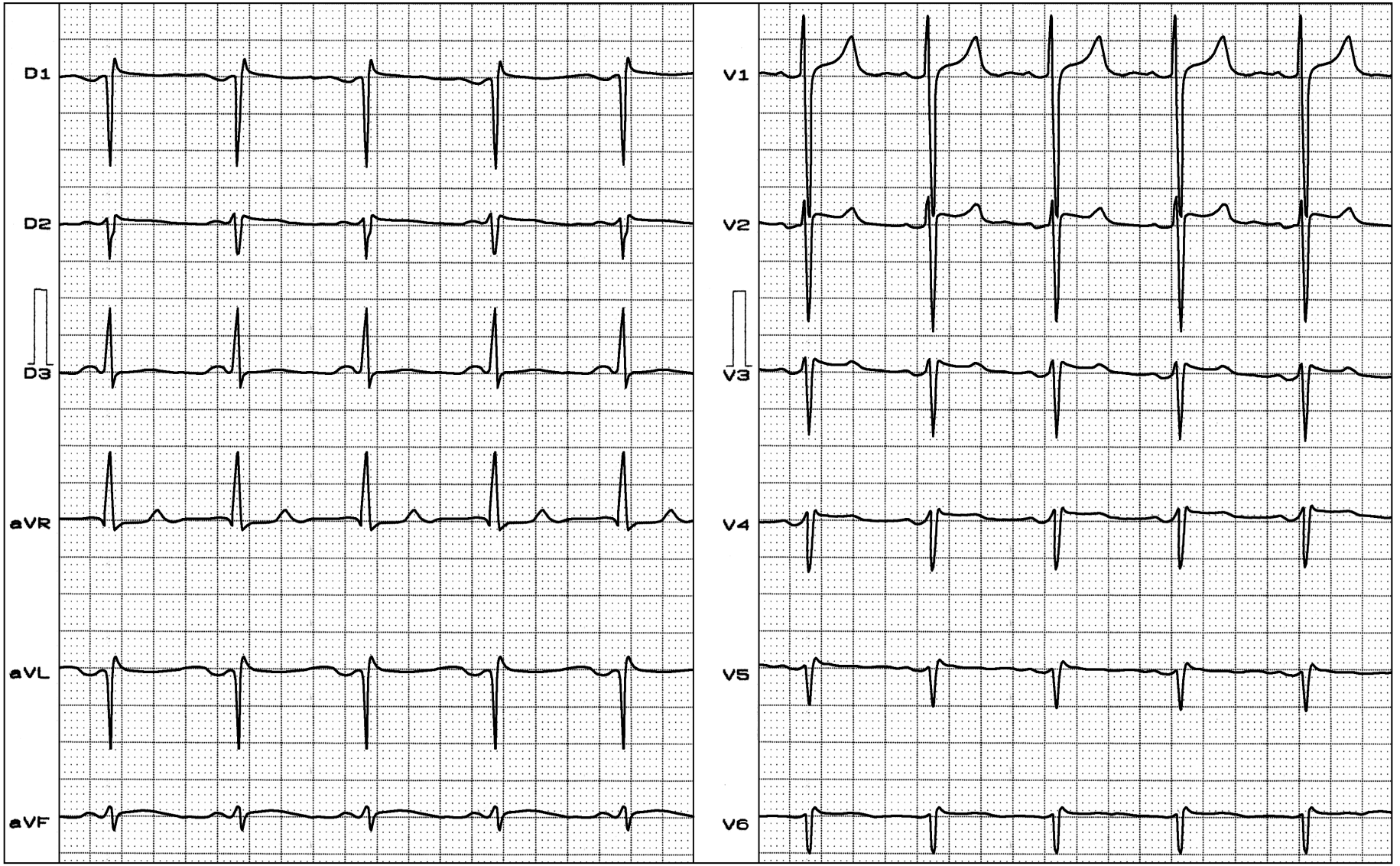
Possible causes of negative P wave in lead I

1) **Incorrect limbs electrode cable connection during electrocardiographic recording (right to left)**

The frequency of ECG artifacts due to switched electrodes is 0.4% at the outpatient clinic and 4.0% at the intensive care unit (**Rudiger 2007**). Limb electrode misconnection: Pseudo dextrocardia by exchange of limb electrodes. All P, QRS, and T waves are negative in I, but normal progression of QRS in precordial leads rules out this hypothesis, pointing out the exchange of arm electrodes.

2) **Simple true dextrocardia:** mirror image. Total atrio-visceral situs inversus with no heart disease. SÂP directed to the right and below, pointing at around +120 degrees (III). Negative P wave in VL and I, positive in III. Reverse progression of r wave in precordial leads V2 to V5 (decreasing).

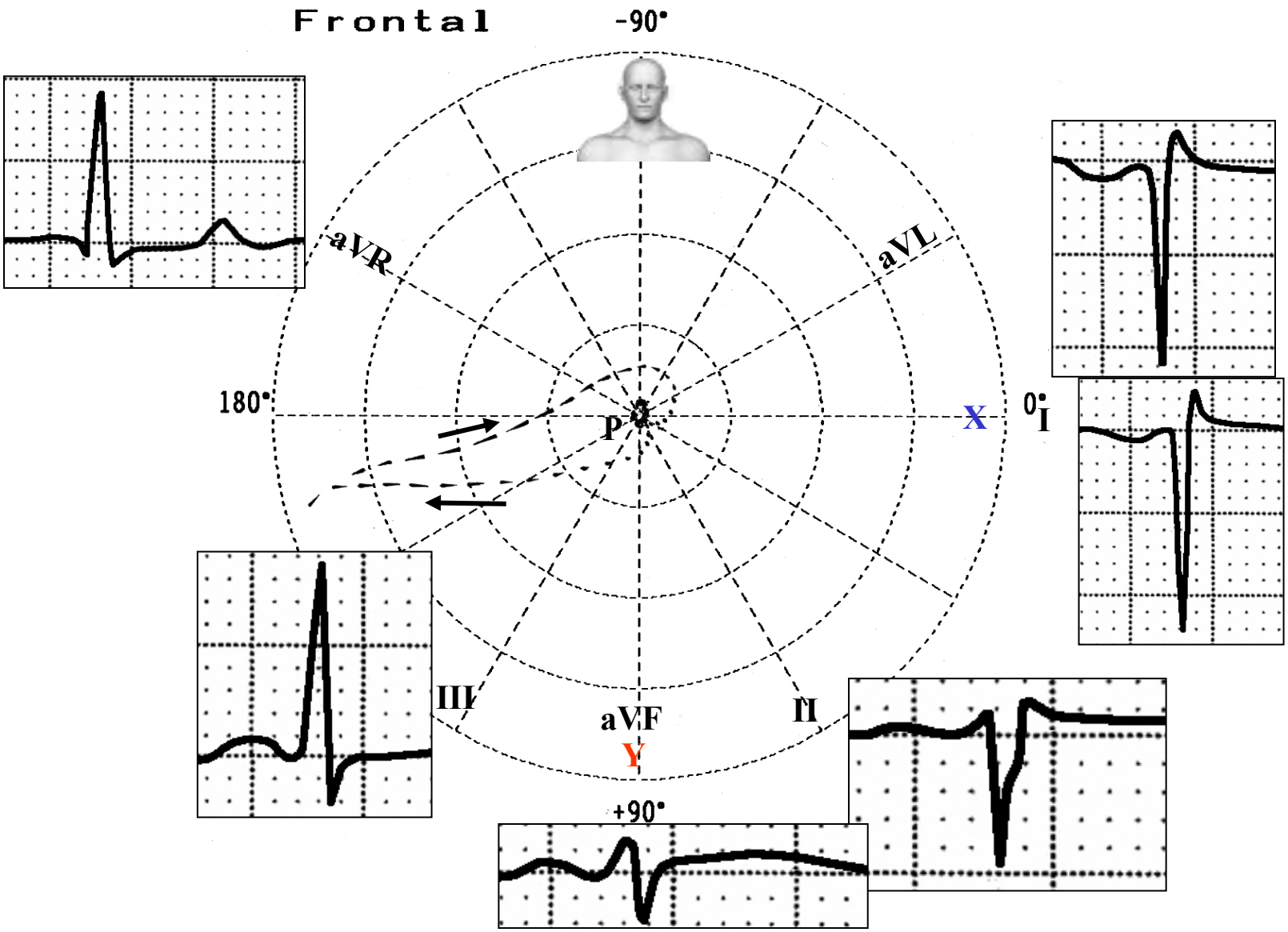
Name: JHY; **Age**: 39 y.o.; **Gender**: Male; **Ethnic Group**: Caucasian; **Weight**: 68 kg; **Height**: 1.70 m; **Biotype**: Asthenic; **Date**: 11/08/2010; **Medications in use**: None.



Clinical diagnosis: True dextrocardia.

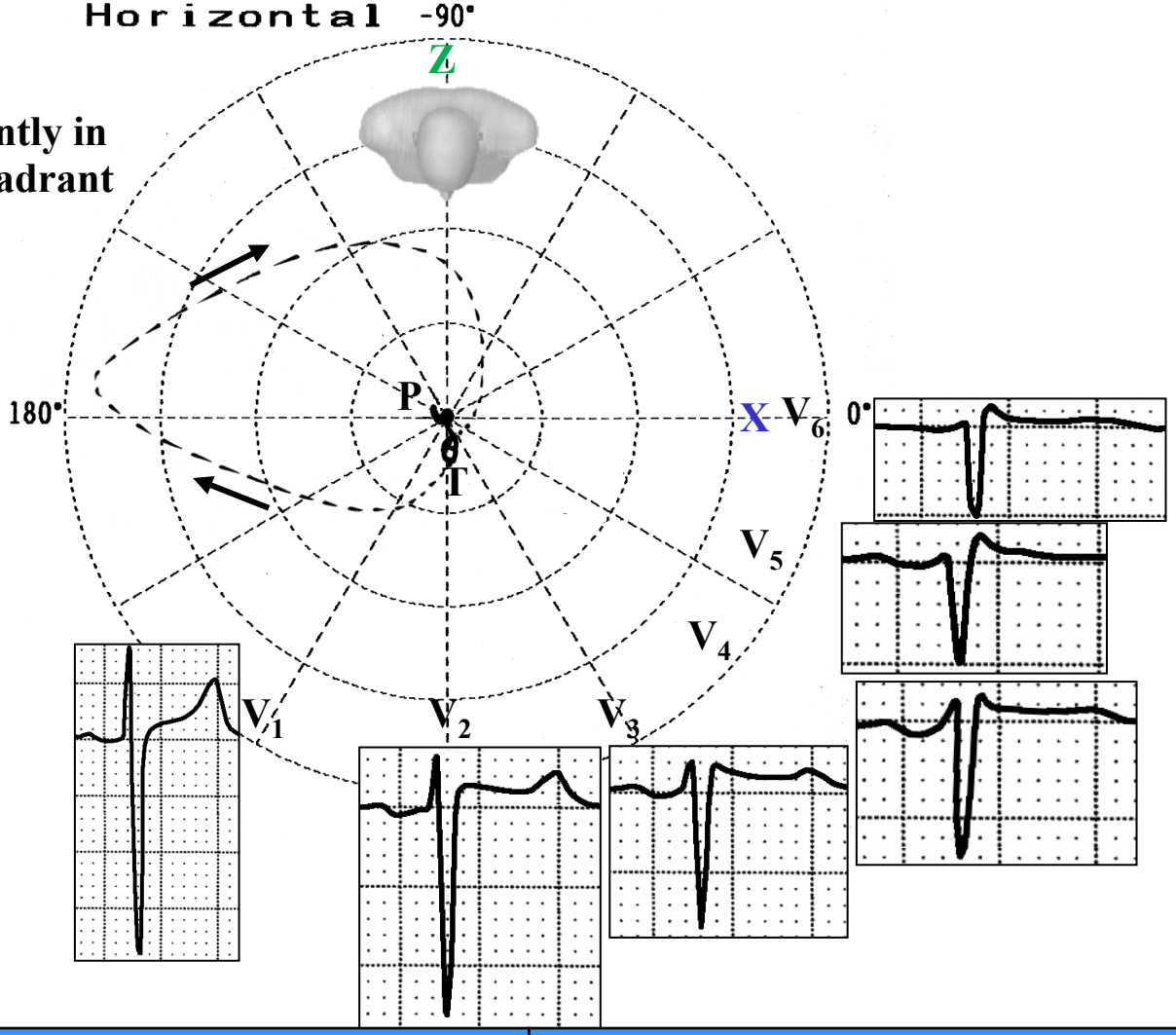
ECG diagnosis: Negative P waves in lead I and aVL, V5-V6, and positive P waves in right leads, aVR and III, negative P, QRS and T waves in I, reverse progression: R wave of decreasing voltage from V_2 to V_5 .

ECG/VCG correlation in the Frontal Plane



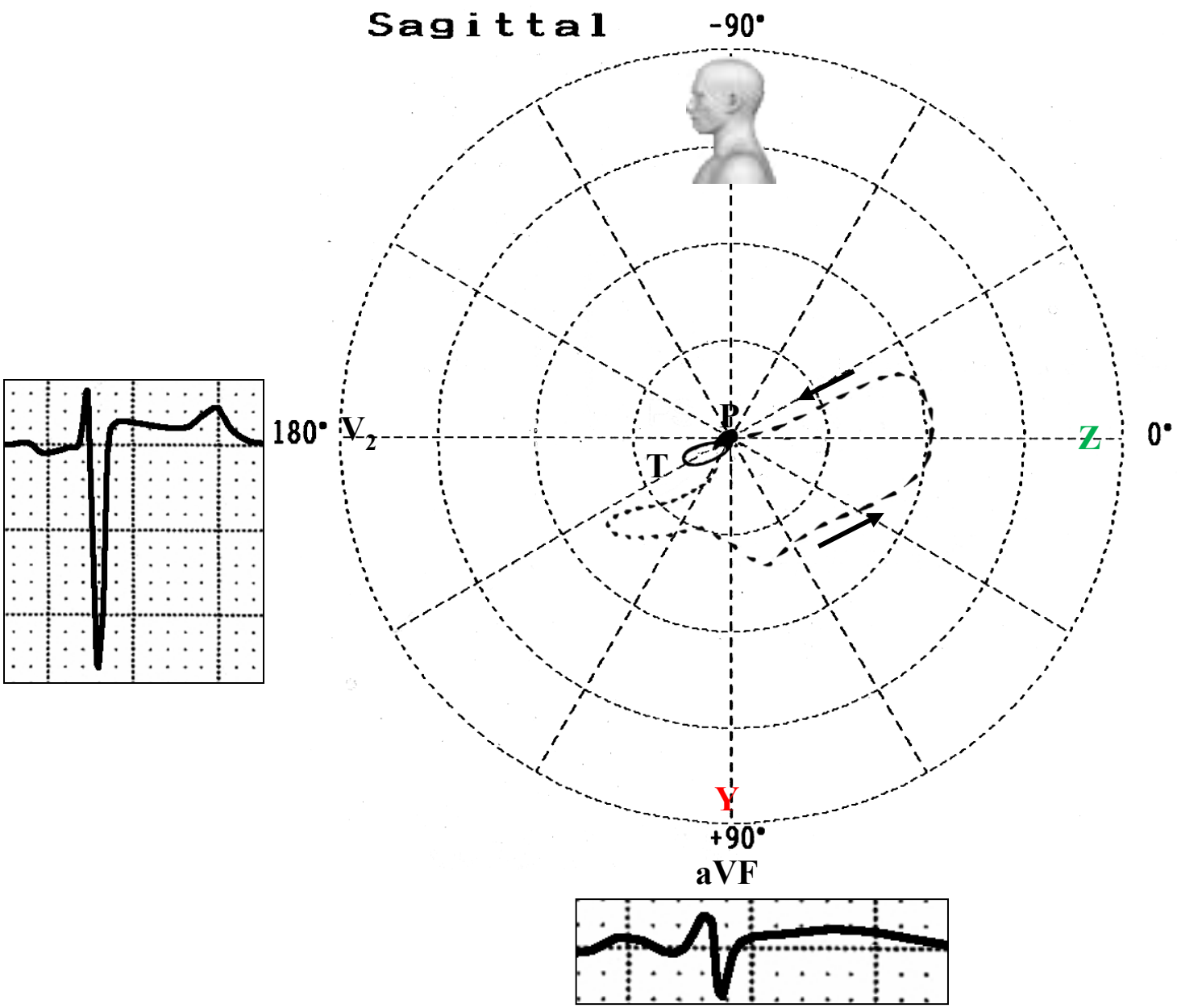
ECG/VCG correlation in the Horizontal Plane

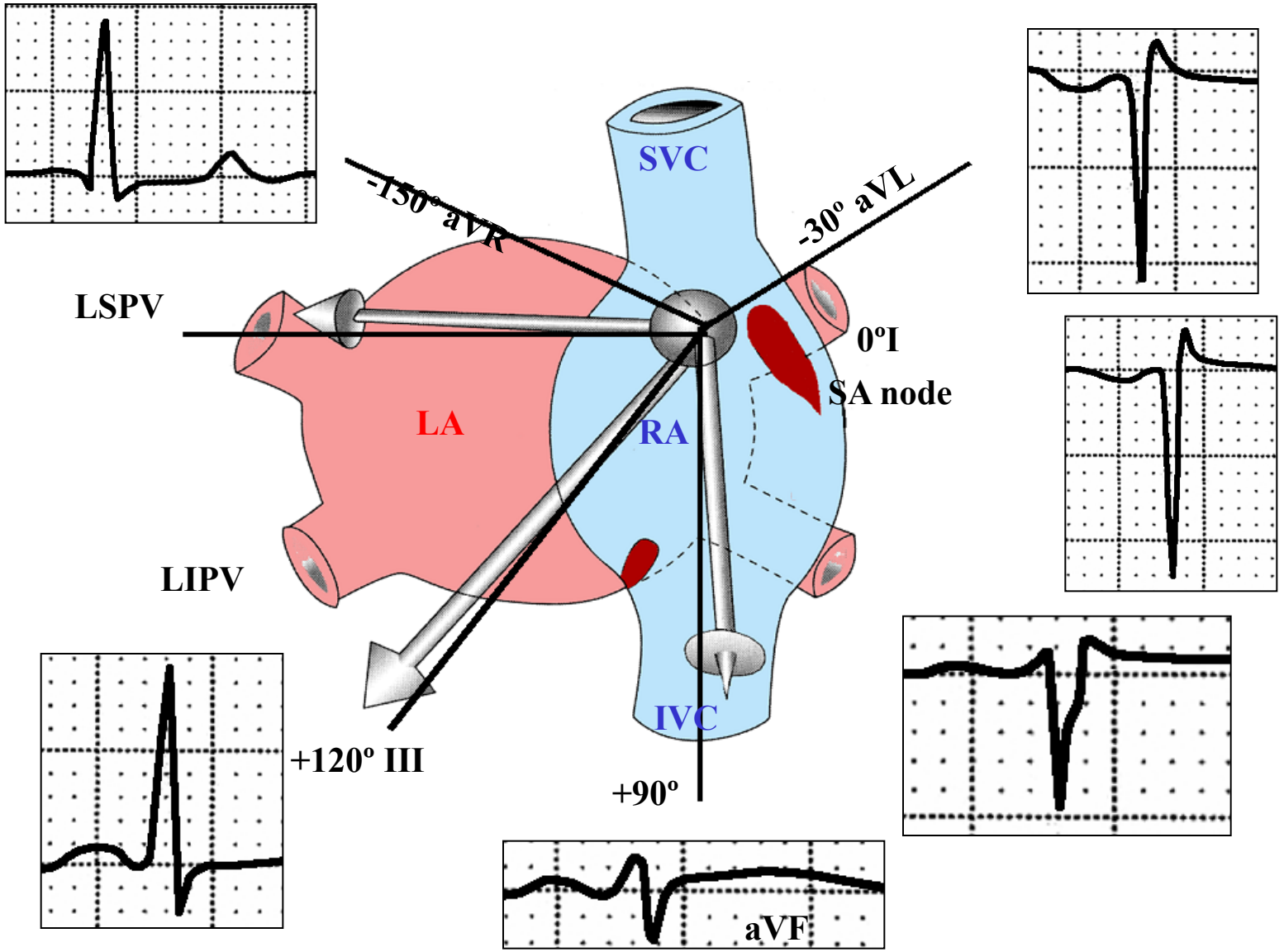
QRS loop predominantly in the right posterior quadrant



Dextrocardia	Normal heart
Predominant location of QRS loop: Right posterior quadrant	Predominant location of QRS loop: Left posterior quadrant
QRS Rotation: Clockwise Rotation (CW-R)	QRS Rotation: Counterclockwise Rotation (CCW-R)
Reverse Progression of the R wave in precordial leads	Normal Progression of increased R wave V1-V4

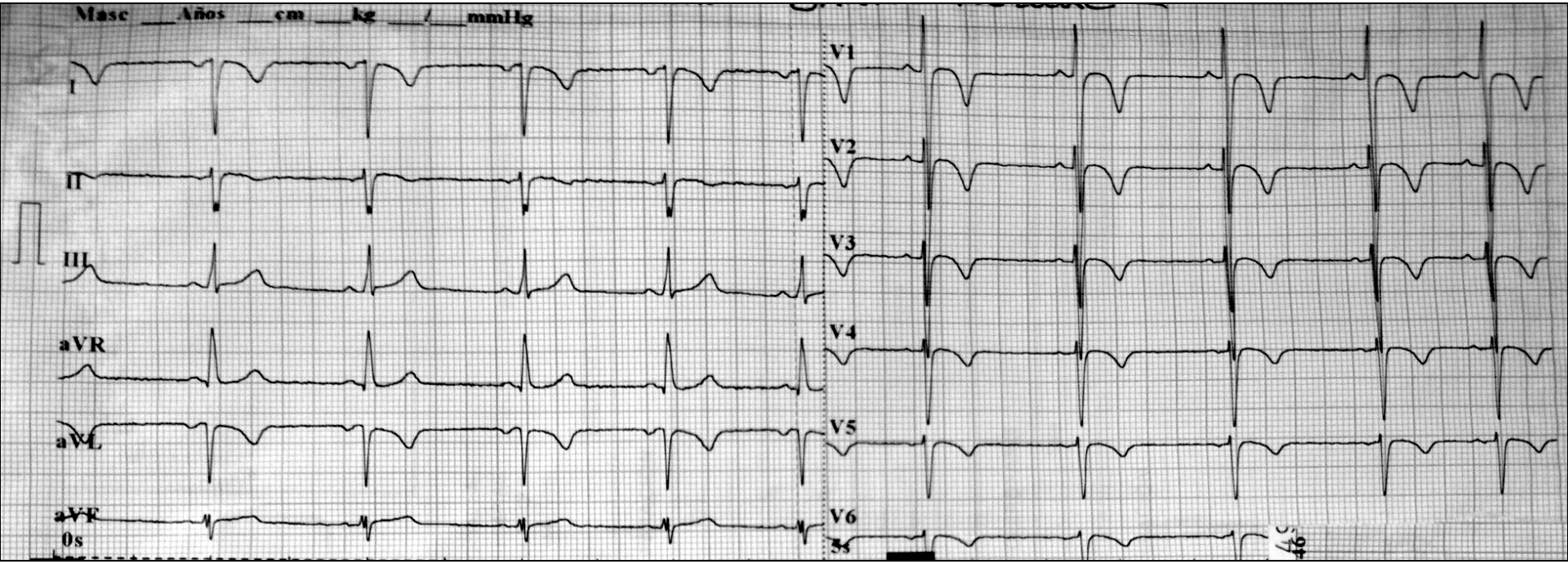
ECG/VCG correlation in the Left Sagittal Plane





ECG leads must be placed in reversed positions on a person with dextrocardia. In addition, when defibrillating someone with dextrocardia, the pads should be placed in reverse positions. That is, instead of upper right and lower left, pads should be placed upper left and lower right.

Dextrocardia with situs inversus



The ECG findings are:

Lead I: P, QRS and T waves inverted or upside down.

Lead II: represents the usual lead III and vice versa.

aVR and aVL are reversed and prominent negative deflections are seen in aVL rather than in aVR.

aVF is unaffected.

P axis (SÂP) to the right and below, pointing at around +140º. Negative P wave in aVL and I, positive in III and aVR.

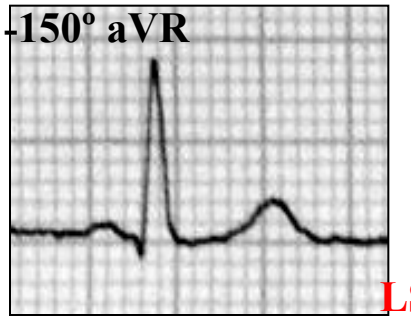
Precordial leads: From V1 to V6: QRS complexes show progressive decreasing R/r wave amplitude:

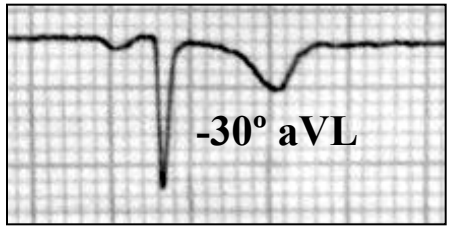
Reverse progression of r wave in precordial leads from V1 to V6 (decreasing).

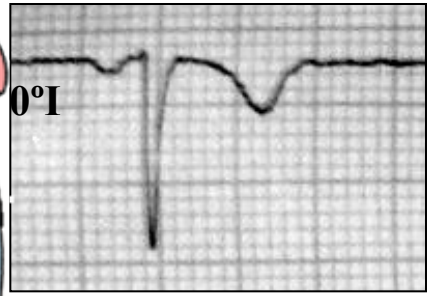
V1 is the equivalent of the usual V2 and vice versa.

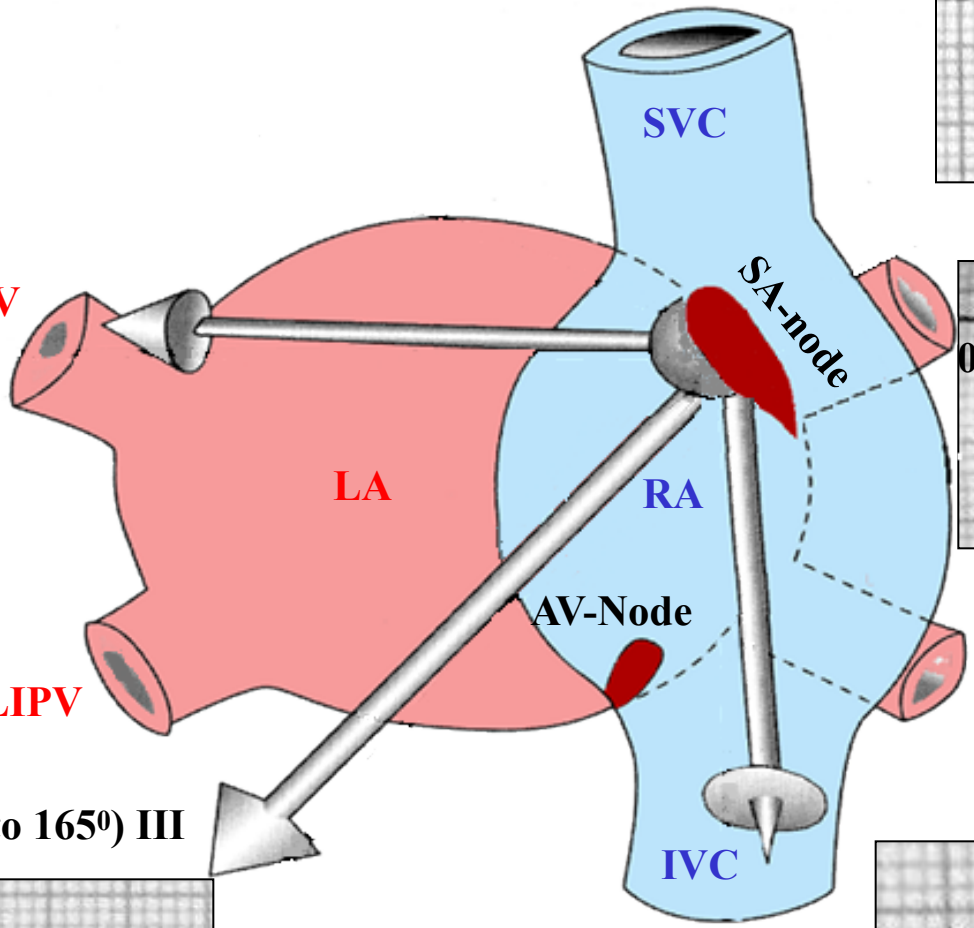
Conclusion: True dextrocardia.

Atrial inversion (situs inversus)

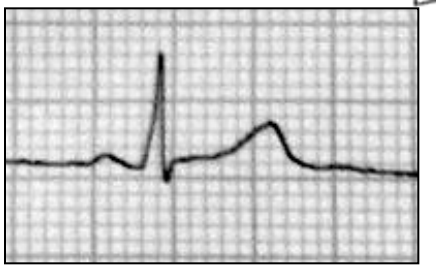


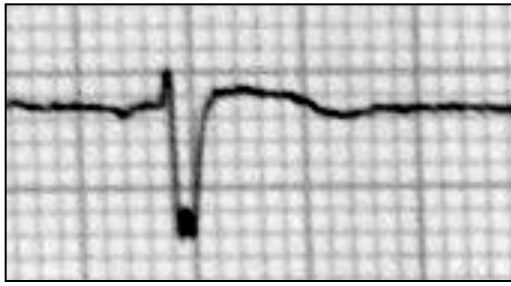




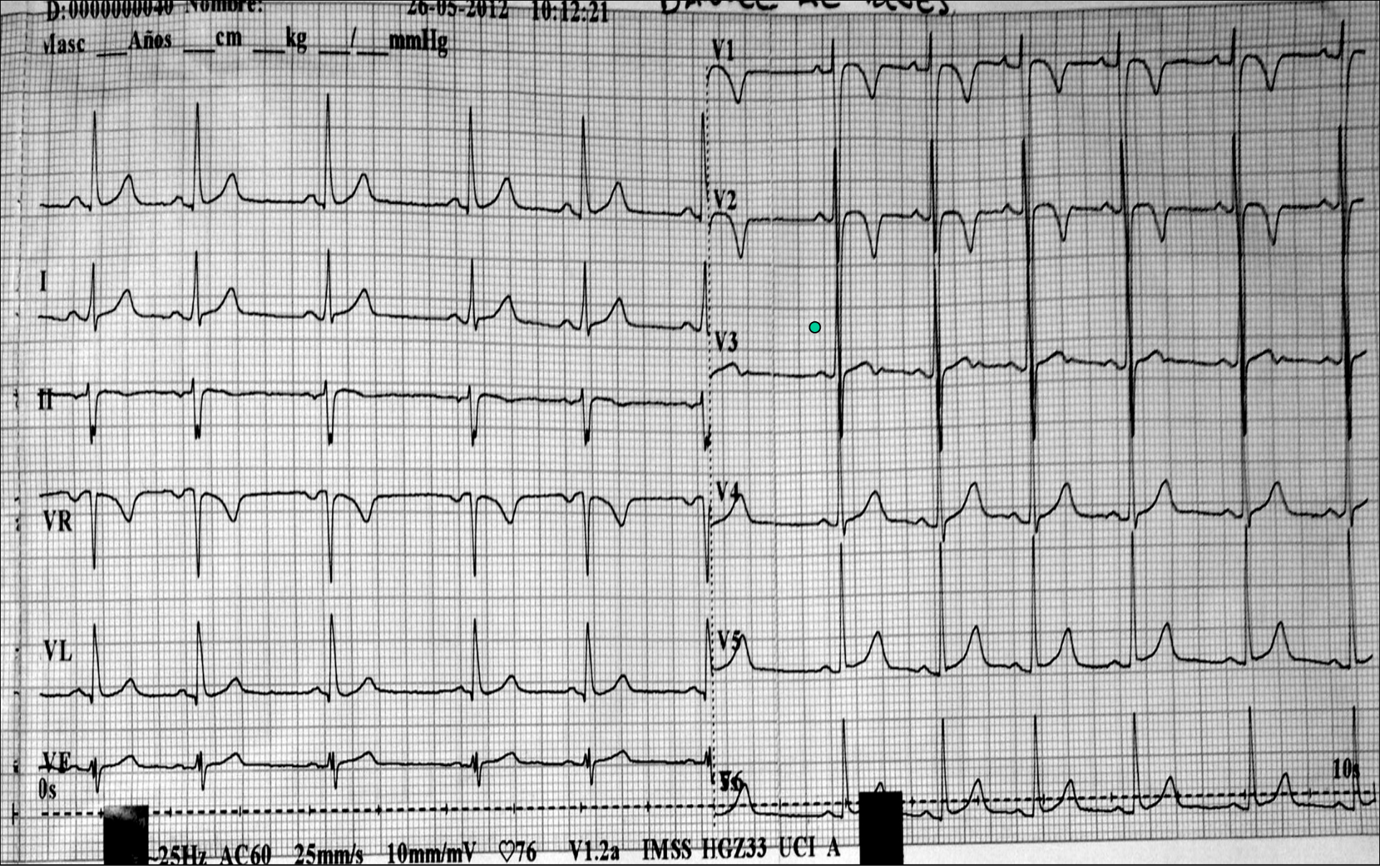


SÂP +120º (between 105 to 165⁰) III





SÂP vector directed from left to right, top to bottom, pointing at around +120⁰ (III). This fact will yield negative P wave in aVL and I, positive in III and aVF and variable II and aVR



This ECG was performed latter willfully inverting the cables of both limbs as the position of the precordial electrodes, which were placed right at points corresponding to the left leads.

Conclusion: Normal ECG in both planes.

Causes and symptoms

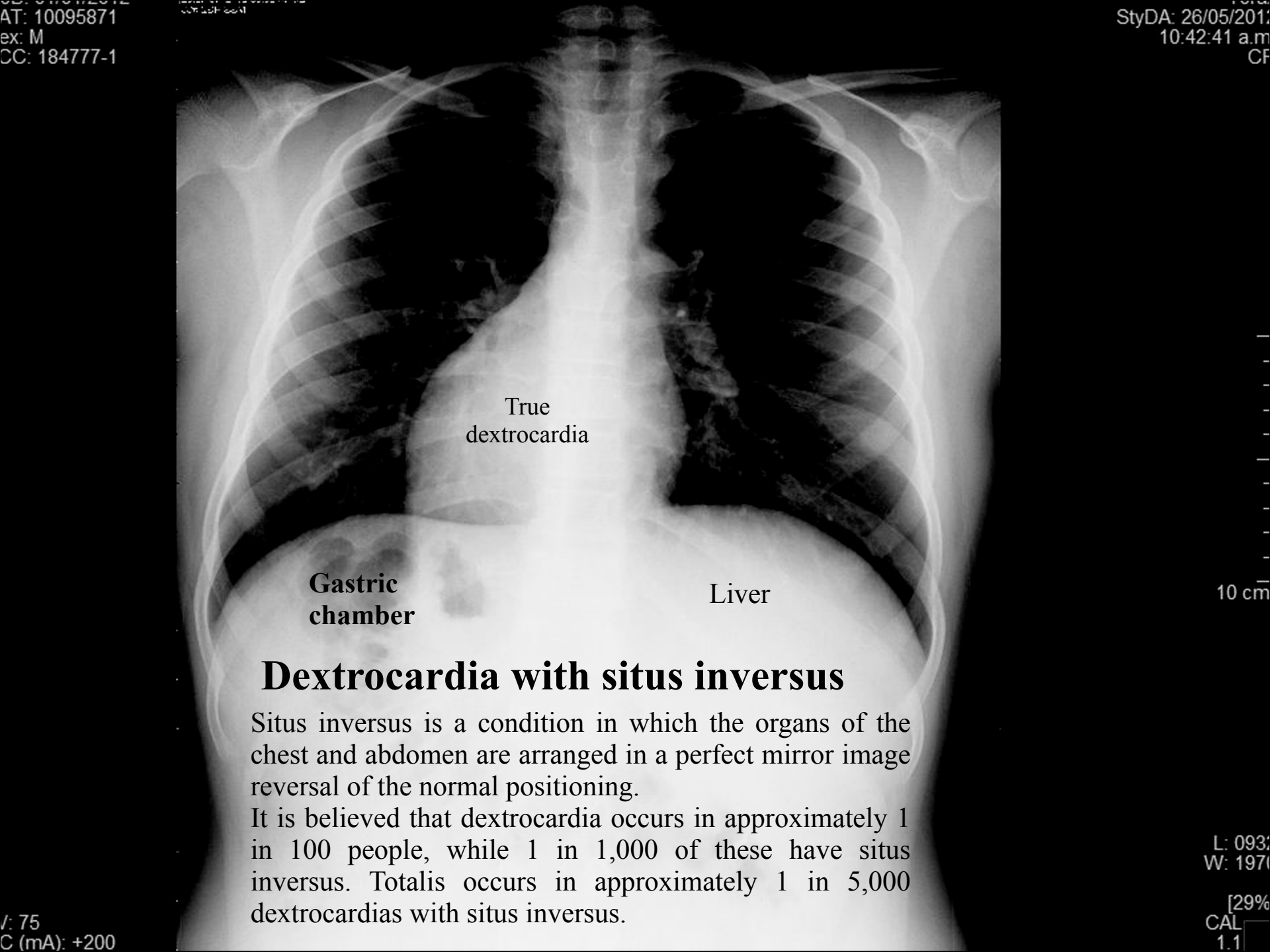
Early in the normal development of an embryo, the tube-like structure that becomes the heart forms a loop toward the left, identifying the left/right axis along which the other organs should be positioned. Although the mechanism that causes the heart loop to go left is not fully understood, at least one gene has been identified to have a role in this process. However, it is thought that many factors may be involved in causing situs inversus. Rarely, situs inversus can run in families, but most often it is an isolated and accidental event occurring in an individual for the first time in the family.

Most people with situs inversus have no medical symptoms or complications resulting from the condition. Although only 3-5% of people with situs inversus have any type of functional heart defect, this is higher than the rate of heart defects in the general population, which is less than 1%. Dextrocardia occurs in approximately 0.01% of live births and can be discovered in various clinical settings and at various patient ages.

The condition affects all major structures within the chest and abdomen. Generally, the organs are simply transposed through the sagittal plane. The heart is located on the right side of the thorax, the stomach and spleen on the right side of the abdomen and the liver and gall bladder on the left side. The left lung is trilobed and the right lung bilobed, and blood vessels, nerves, lymphatics and the intestines are also transposed.

If the heart is swapped to the right side of the thorax, it is known as situs inversus with dextrocardia or situs inversus totalis. If the heart remains on the normal left side of the thorax, a much rarer condition (1 in 22,000 of the general population), it is known as situs inversus with levocardia or situs inversus incompletus. Situs inversus with levocardia, or dextrocardia without situs inversus, present much higher rates of congenital defects than situs inversus with dextrocardia.

It is estimated that about 25% of people with situs inversus have an underlying condition called primary ciliary dyskinesia (PCD). PCD, also known as Kartagener's syndrome, is characterized as situs inversus, chronic maxillary sinusitis, bronchiectasis and "situs inversus totalis" increased mucous secretions from the lungs, and increased susceptibility to respiratory infections. PCD is caused by a defect in the cilia that impairs their normal movements.



Dextrocardia with situs inversus

Situs inversus is a condition in which the organs of the chest and abdomen are arranged in a perfect mirror image reversal of the normal positioning.

It is believed that dextrocardia occurs in approximately 1 in 100 people, while 1 in 1,000 of these have situs inversus. Totalis occurs in approximately 1 in 5,000 dextrocardias with situs inversus.

	Normal	Simple true dextrocardia
Initial 10 to 20 ms vector	Forward and to the right.	Forward and to the left.
QRS loop rotation	Counterclockwise.	Clockwise.
Location and voltage of maximal vector	Left posterior quadrant. Voltage does not exceed 2 mV.	Right posterior quadrant. The maximal vector represents the systemic or arterial ventricle. Voltage does not exceed 2 mV.
I	Positive P; qRs type QRS and positive T.	Negative P, QRS and T.
aVL-aVR	The first positive and the second negative: P, QRS and T.	The first negative and the second positive: P, QRS and T.
II-III	Variable.	Usual image of II in III and vice-versa.
V_1 - V_2	rS.	There may be initial q: qRs.
V_5 -V_6	qRs or Rs with positive T wave.	rS or rSr' with negative T.
Progression of R wave in precordial leads	Progressive increase of R wave voltage from V_1 to V_5 .	Reverse progression: R wave of decreasing voltage from V_2 to V_5 .
T loop	To the left and in adults, slightly forward: always positive in V_2 -V_6.	To the right and in adults, slightly forward: negative T in V_5 -V_6.

Dextrocardia is a congenital defect in which the heart is situated on the right side of the body. There are two main types of dextrocardia, **dextrocardia of embryonic arrest** (also known as **isolated dextrocardia**) and **dextrocardia situs inversus**. Dextrocardia situs inversus is further divided.

Dextrocardia of embryonic arrest

In this form of dextrocardia, the heart is simply placed farther right in the thorax than is normal. It is commonly associated with severe defects of the heart and related abnormalities including pulmonary hypoplasia.

Dextrocardia situs inversus

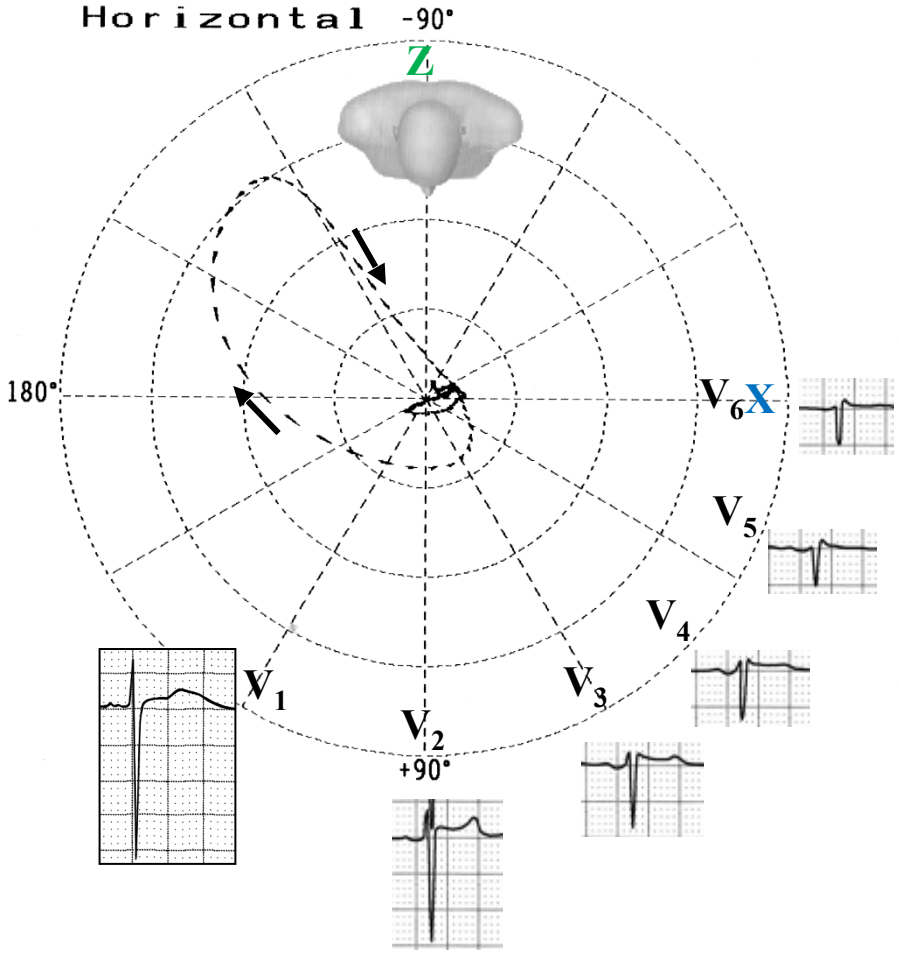
Dextrocardia situs inversus refers to the heart being a mirror image situated on the right side. For all visceral organs to be mirrored, the correct term is dextrocardia situs inversus totalis.

The incidence of dextrocardia was estimated to be 1 in 12,019 pregnancies (**Bohun 2007**); while one of 3 of these will have situs inversus. Totalis occurs in approximately 1 in 5,000 of dextrocardia situs inversus.

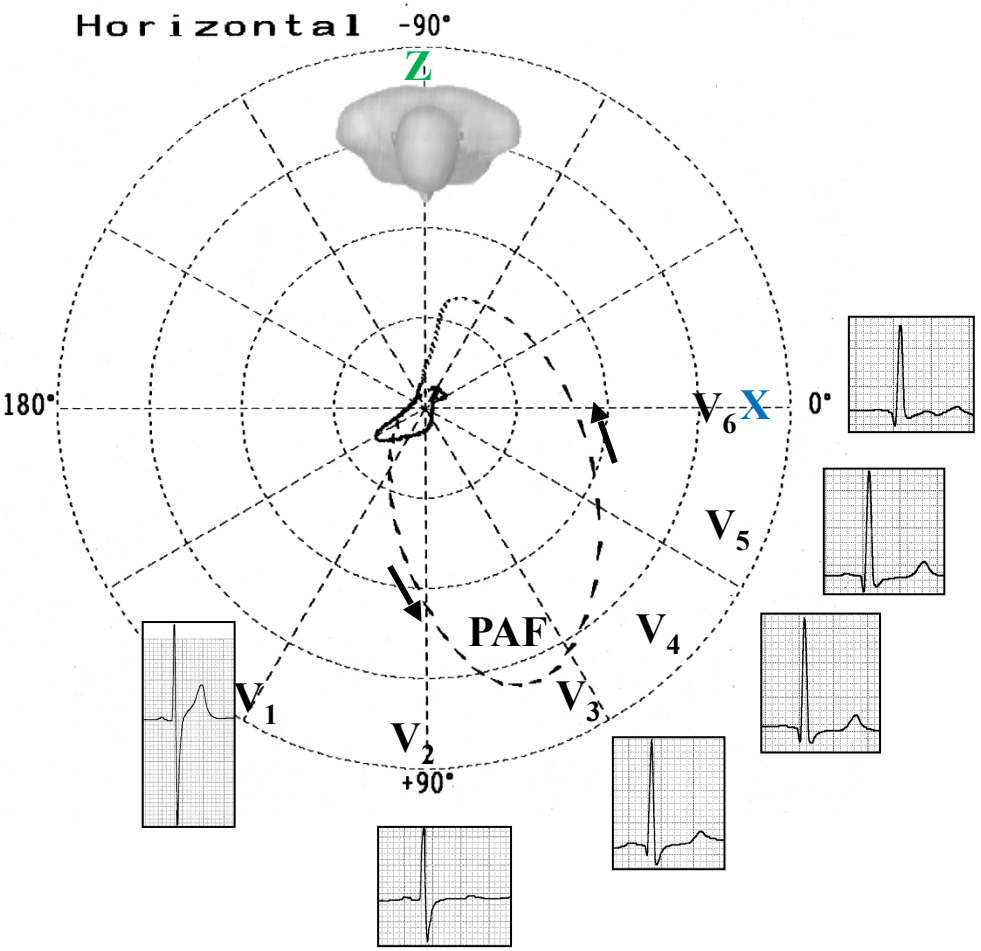
Kartagener's syndrome occurs in approximately 1 in 25 of totalis. This disorder affects the sinus and bronchial cilia causing constant sinus and bronchial symptoms that medication cannot rectify. With Kartagener's both are usually present all year rather than being seasonal.

Although statistically people with dextrocardia situs inversus do not have any medical problems from the disorder, some are prone to a number of bowel, esophagus, bronchial and cardiac problems. Some of these conditions can be life threatening if left unchecked.

True dextrocardia



Dextroposition or pseudo-dextrocardia

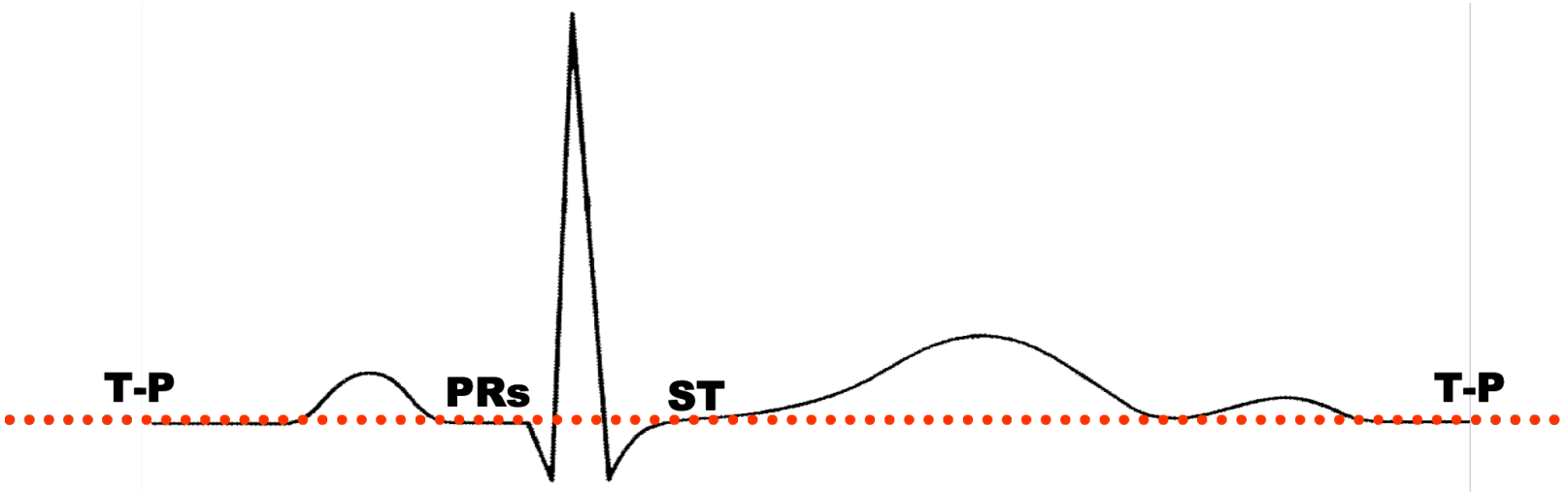


Predominant location of QRS loop: Right posterior quadrant	Predominant location of QRS loop: Left anterior quadrant
QRS Rotation: Clockwise	QRS Rotation: Counterclockwise
Reverse progression of the R wave in precordial leads	Prominent Anterior QRS Forces (PAF)

Atrial T vector

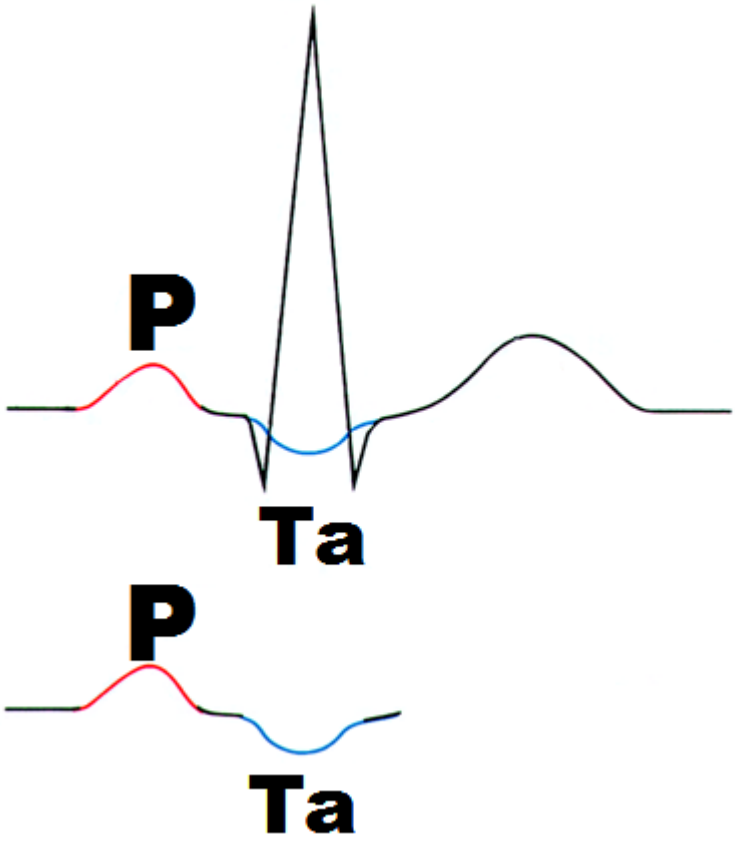
Atrial repolarization (Ta vector) is responsible for the P loop being open and not closed (0 point does not coincide with initial E point). A straight line from the onset of the P loop to the end of it, shows the magnitude and direction of the Ta vector.

Exteriorization of segments in vectorcardiography



In vectorcardiography, isoelectric lines corresponding to PR (PRs), ST and T-P segments are not recorded if they do not show depression or elevation. Thus, the segments manifest as stationary points. This non-manifestation is the reason why the ECG is superior to VCG in the analysis of segments and intervals.

P loop is open because atrial repolarization (TP or TA loop) is completely opposite to the P loop



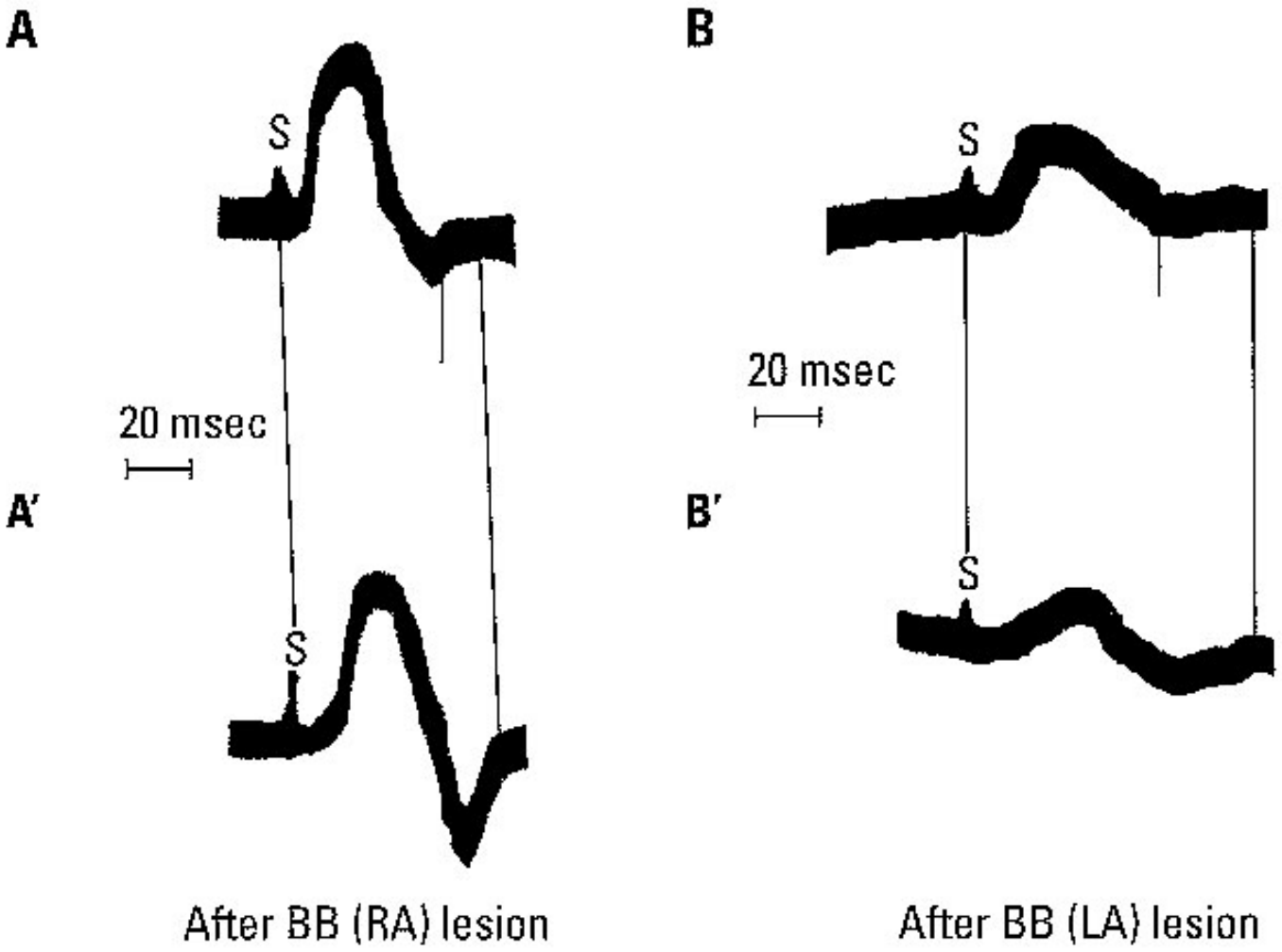
In ECG, Ta or Tp wave: wave generally not visible because it is hidden by QRS. It represents atrial repolarization. Ta polarity is opposite to the P wave and its magnitude is 100 to 200 muV.

It may possibly reach the ST segment and the T wave, causing ST segment depression and resembling myocardial ischemia (**Sapin 1991**).

The interatrial blocks: an anatomical-electrical pandemic "Bayés' syndrome" (Conde 2014: Spodick 2009)

Probably interatrial blocks are not considered as an individual pattern in the majority of books on ECG, because the ECG patterns found in atrial enlargement, especially in LAE, are in many cases influenced by the presence of interatrial blocks. The term "atrial abnormalities", that we do no support, has been coined to encompass both atrial enlargement and atrial block (**Tsao 2008; Lee 2007**). While it is true that atrial blocks are often associated with atrial enlargements (sometimes this also happens at ventricular level), the ECG patterns of atrial block are independent entities because there are 3 criteria that define an ECG pattern as being caused by a block or deterioration of conduction (**Bayés de Luna 2013; 2012; Waldo 1971**).

a) The ECG pattern may appear transiently, and the pattern may change abruptly and progressively to more advanced forms;
b) The ECG pattern may appear without association to other processes such as cardiac chamber enlargement or ischemia, although in many cases, one or more of these conditions may coexist; and
c) Similar ECG pattern may be reproduced experimentally.(**Bayés de Luna 1985**) (Figure next slide)



Adapted from experimental Bachmann's bundle block (**Waldo 1971**). (A) Control P wave recorded in ECG lead II when the atrias were paced from the right atrium. See the change of morphology after Bachmann's bundle lesion in the right side. (B) P wave recorded in lead II after the creation of a lesion in the left atrial (LA) portion of Bachmann's bundle (BB). In both cases the changes in conduction time and morphology after block are shown. (Adapted with permission from (**Waldo 1971**)).

Various studies performed by Bayés de Luna (**Bayés de Luna 1985; 1988; 1999; 1989**) and others, such as Spodick (**Spodick 2008; 2009**), Holmqvist et al. (**Holmqvist 2007**), and Platonov (**Platonov 2008**), have re-evaluated the concept that IABs and atrial enlargement are separate entities, although in many cases, they are associated. The IAB that occurs between the right and left atrium is well detected by surface ECG and will be discussed in depth. The figure below, shows the schematic diagnosis of different types of IAB.

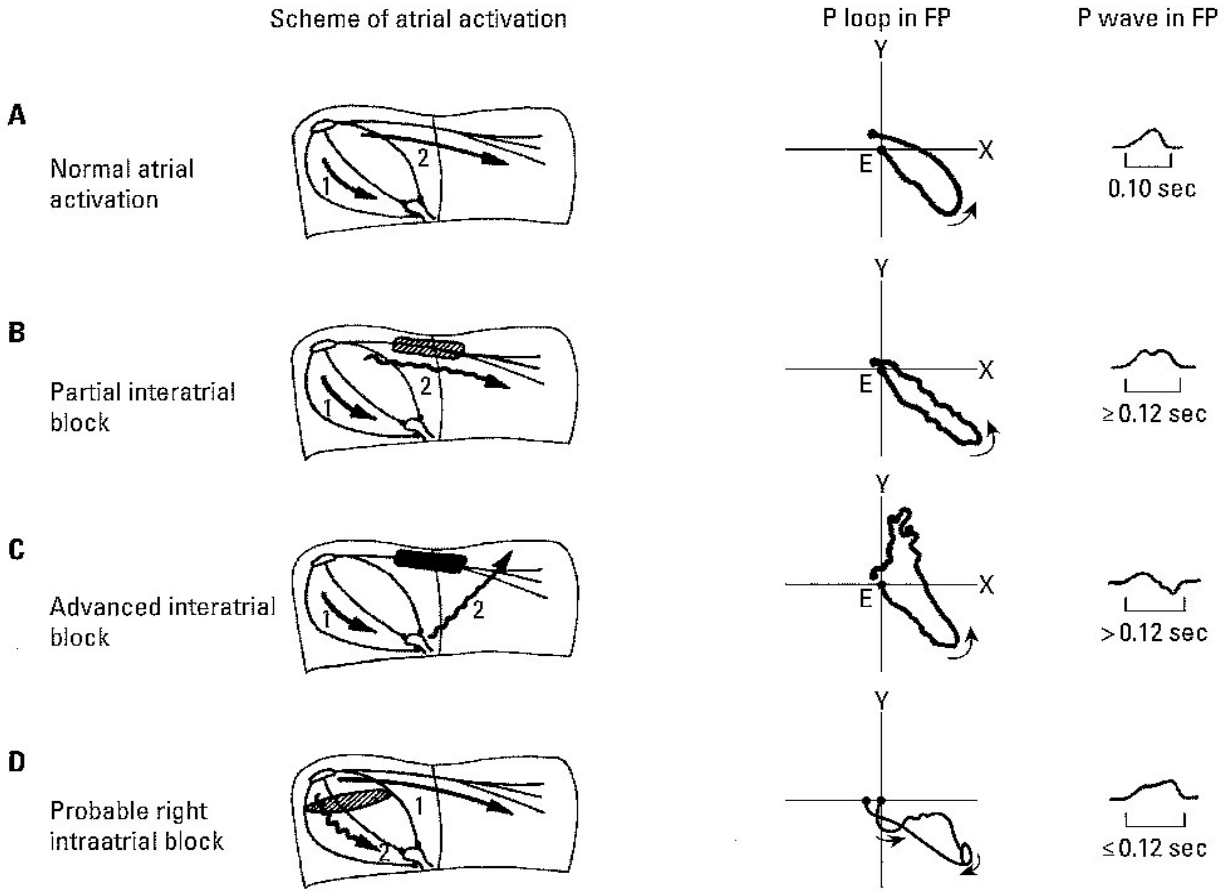
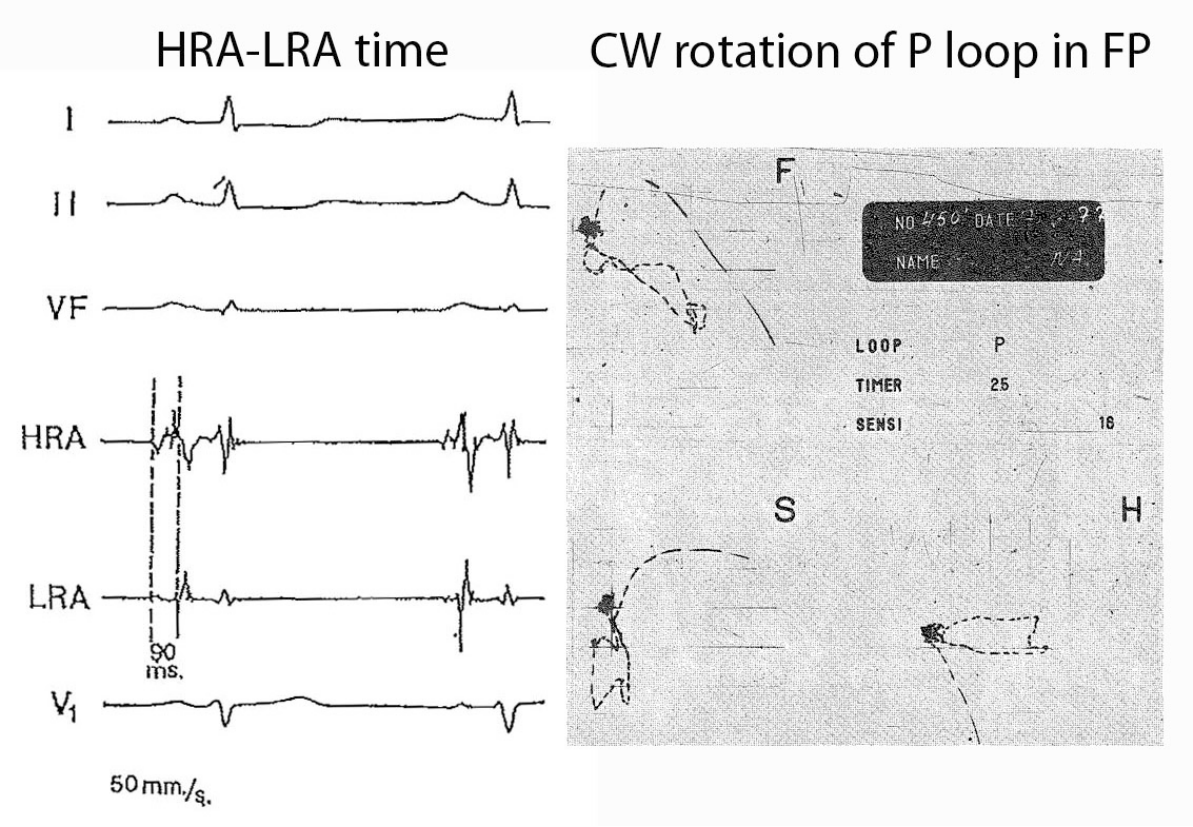


Diagram of atrial conduction under normal circumstances (A), partial interatrial block (B), advanced interatrial block with left atrial retrograde activation (AIB with LARA) (C) and probable right intra-atrial block (D).

IABs, like other types of block (sinoatrial, atrioventricular, and ventricular) may be first, second (transient block, atrial aberrancy), or third degree. The evolution from first-degree IAB to third-degree (advanced) IAB may be seen, as it also happens in other types of block. It is very difficult to identify the presence of a block within a single atrium (intra-atrial block) using surface ECG. We have seen that occasionally apparently normal P waves (≤ 0.12 s with some small notches) present with total or near total clockwise rotation of the loop in the FP, and we hypothesize that this is due to the presence of a localized block in some part of the right atrium (Previous slide figure D and figure below).



One case of nearly total clockwise rotation of the P loop in frontal plane (FP) that shows a normal P wave (morphology and duration) in the ECG. In the previous slide figure A, the loop also rotates clockwise in the horizontal plane (HP), suggesting that the right atrial block is located in the anterior part or the right atrium. See the increase of time between high right atrium and low right atrium (HRA-LRA) that assures the presence of block in the right atrium.

Comments: the exact diagnosis of right intra-atrial block can only be performed by intracavitary ECG (long high right atrium to low right atrium (HRA-LRA) interval) (figure from previous slide). It is very important to perform a new study on that additionally; the atrial delay may also be located in the left atrium in some cases of very long and notched P waves, especially if large slurring is present in the second part of the P wave.
Currently, we must consider interatrial blocks as a pandemic among senior individuals with anatomical and electrical substrate for atrial arrhythmias. It is considered a true syndrome.

Prevalence

High. Several studies have reported that the prevalence of IAB is more than 40% in hospital inpatients. Despite this, IAB remains largely underdiagnosed and commonly ignored. The advanced form is much less common than the partial form. The condition remains largely underdiagnosed and commonly ignored. **(Kitkungvan 2009)** Spodick considers this ECG abnormality is present in pandemic proportions especially at ages 60 and over and in unselected hospital patients. Because of its pathologic implications it requires widespread attention as a "pandemic".

Associated condition

1. Coronary Artery Disease
2. Hypertension
3. Diabetes Mellitus
4. Atrial fibrillation (strong associations)
5. Potential risk for embolism
6. Left Atrial Enlargement (LAE).
7. Left atrial electromechanical dysfunction.

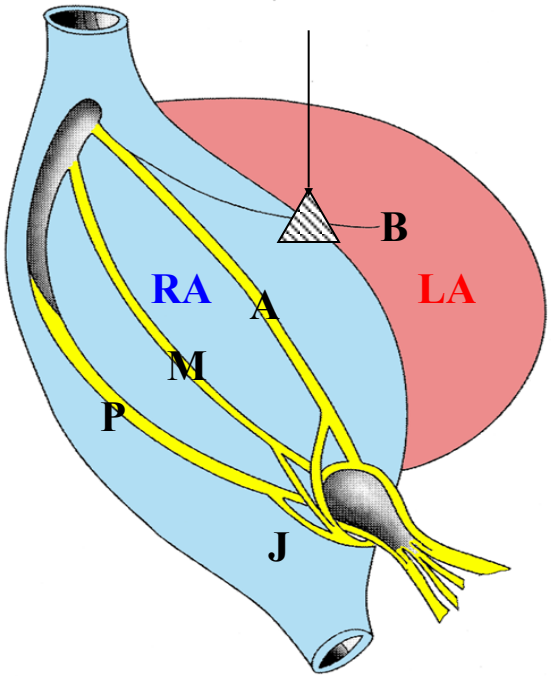
Interatrial block: classification and identification

1. ***First degree or partial interatrial block***
2. ***Second-degree or transient interatrial block (atrial aberrancy)***
3. ***Third degree, complete or advanced interatrial block.***

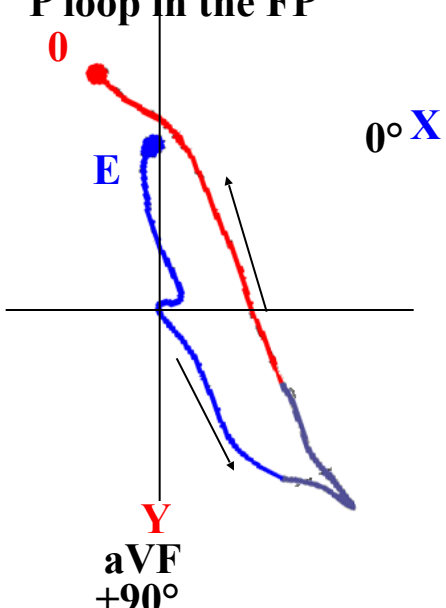
First degree or partial interatrial block

Definition: It is the block between the right and the left atrial in the zone of the Bachmann bundle. The electrical impulse is conducted from the right atrium (RA) to the left atrium (LA) trough Bachmann's bundle, but conduction is delayed. The ECG shows only P wave duration of ≥110 -120 ms in several leads with a variable negative wave in V_1. The P-wave morphology is similar to that in left atrial enlargement (LAE), but usually a negative P wave in V_1 is less evident. IAB is associated in most of cases with LAE and dysfuction, decreased left ventricular (LV) filling, a propensity for LA appendage thrombosis, reduced atrial natriuretic peptide levels, and is a predictor of paroxysmal supraventricular tachyarrhythmias such as atrial fibrillation, atrial flutter as well as an exacerbation of the LV failure. The prevalence of first degree IAB is much higher than advanced or complete IAB. Really, the ECG pattern of advanced IAB is an extremely strong marker of supraventricular tachyarrhythmias in a short period of time, much more so than the presence of first degree or partial Interatrial AV block.

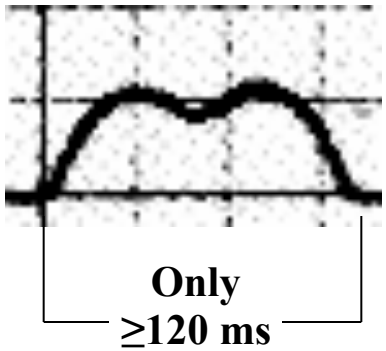
Partial block in Bachmann's bundle



P loop in the FP



P wave in lead "y"

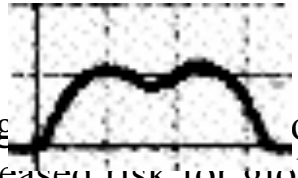


Electrocardiographic characterization

1) P-wave duration ≥110 ms or 120 ms.(Platonov 2008) P-wave duration is generally accepted as the most reliable non-invasive marker of atrial conduction and its prolongation is associated with AF. However, patients with paroxysmal AF without structural heart disease may not have P-wave prolongation, thus suggesting that the global conduction slowing is not an obligatory requirement for development of AF. (**Ariyarajah 2006**)

The widest P wave defining the degree of block could be found in any leads

2) Bifid P waves



Finally, the prevalence of first-degree block in the general population is very high, and their relation with atrial fibrillation and an increased risk for global and cardiovascular mortality has been shown.(**Bayés de Luna 2012**)

The intracavitary recording shows that the HRA-coronary sinus distance is increased.

Second-degree or transient interatrial block (atrial aberrancy)

Definition: It is a transient pattern of partial or advanced IAB, or atrial aberrancy; in other words deviating from the proper or expected course of stimulus inside the atrium. Atrial aberrancy is also possible when premature atrial contractions or premature parasystolic atrial beats are present.(**Bayés de Luna 1978**) **(Julia J Rev. Esp. Cardiol. 1978; 31: 207) (Chung 1972)**

Atrial aberrancy may also be present as a transient bizarre P wave without the morphology of first or third degree IAB. In the figures of the next 2 slides, an example of second degree IAB may be seen.

These changes in P wave morphology are caused by variations in the atrial path of the sinus impulse through the atria. They should be differentiated from changes induced by breathing, atrial fusion beats, and artifacts, including diaphragmatic contraction. Second degree of IAB can be induced by atrial or ventricular premature complexes, which appear and disappear suddenly and transiently in one ECG and show a P wave that changes morphology transiently in successive ECGs, leading to misdiagnosis.

Differential diagnosis

Aberrant atrial conduction must be differentiated from:

1.Immediatly after atrial premature contractions (60% of cases of atrial aberrancy)

2.A wandering atrial pacemaker;

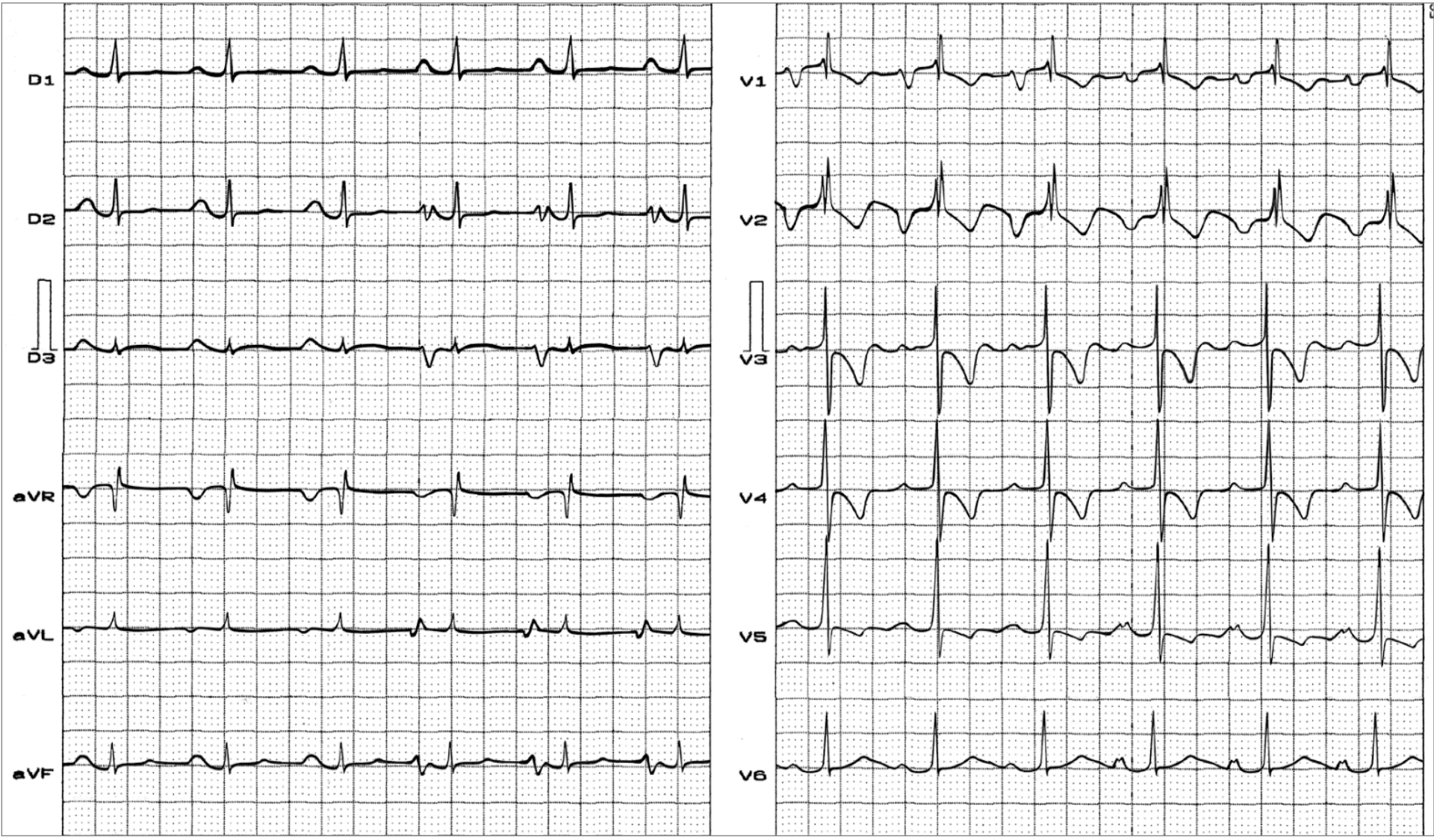
3.Aberrancy after AV junctional escape beats or AV junctional premature contraction

4.Coexisting multifocal premature beats;

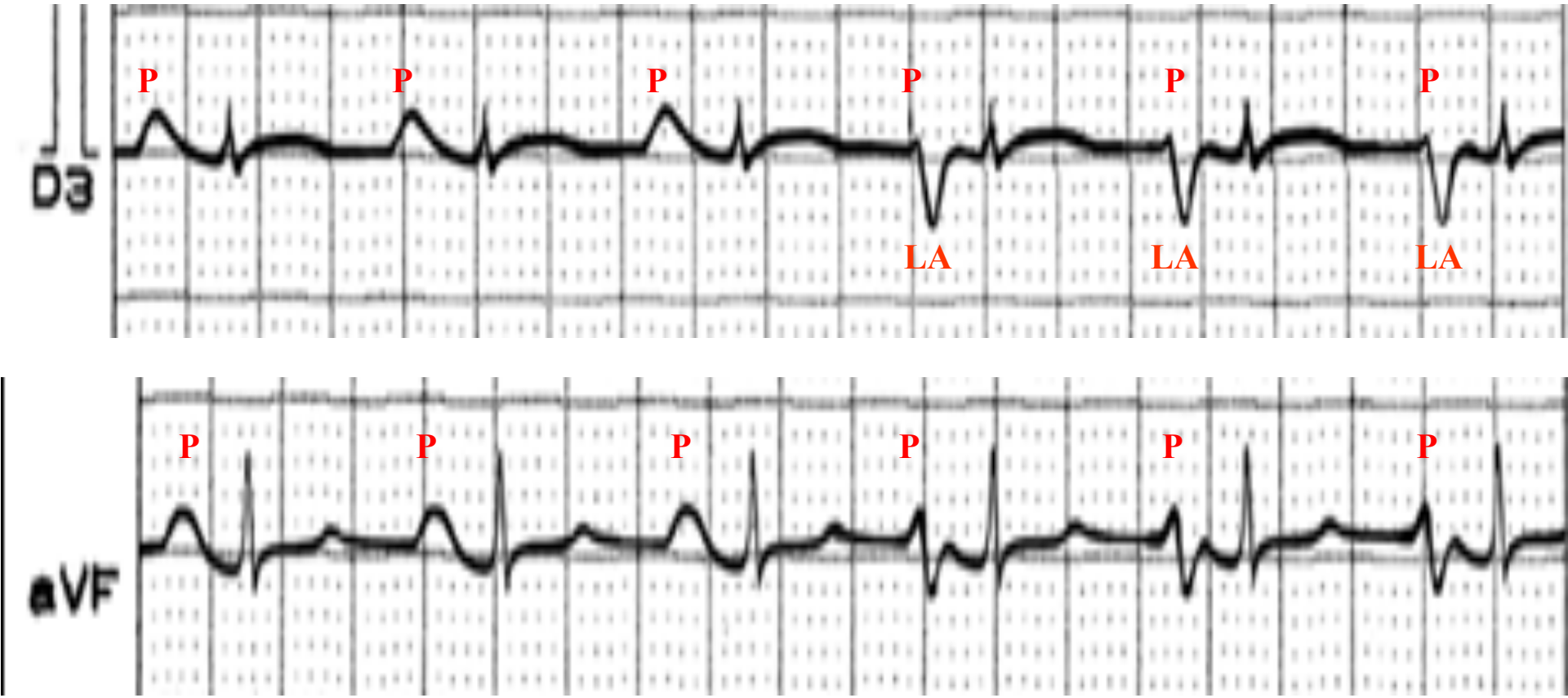
5.Aberrrant atrial conduction after parasystolic beats, mainly interpolated atrial parasystolic beat.

6.Several artifacts**.**

Name: AS; **Age:** 32 y.o.; **Sex:** Fem; **Race:** White; **Weight:** 52 Kg; **Height:** 1.55 m; **Date:** 12/3/2003;
Drugs in use: Digoxin 0.25 mg, Potassium chloride and Benzetacil 1200.000 U every 21 days.



Clinical diagnosis: Pure mitral stenosis
ECG Diagnosis: Left atrial enlargement (P duration 120 ms, notched and final component of P wave in V_1 and V_2 deep and slow). Intermitent or transient IAB (aberrancy). PR interval: 220 ms: first-degree AV block..



The first 3 beats show an enlarged P wave of left atrial enlargement and the last 3 show a positive-negative biphasic P wave indicating retrograde activation of the left atrium down to the top. The first part of the P wave is the right atrium activation and the second negative part of the P wave corresponds to left atrium activation from the bottom to top.

Third degree, complete or advanced interatrial block

Definition: In these cases the stimulus is blocked in the zone of the Bachmann bundle and LA is activated retrogradelly with a P wave duration ≥120 ms and plus-minus ± P wave in inferior leads II, III and aVF. There is an open angle ≥90° between the vector of the first part and of the second part of the P wave in the inferior leads. Orthogonal Y lead plus-minus with a negative mode >40 ms appear with notches and slurrings in the last part of the P loop. The concept of complete IAB is not frequently used in cardiology.

IAB is associated in most of cases with LAE (90° of cases) and dysfunction, decreased left ventricular (LV) filling, a propensity for LA appendage thrombosis, reduced atrial natriuretic peptide levels, and is a predictor of paroxysmal supraventricular tachyarrhythmias such as atrial fibrillation, atrial flutter as well as an exacerbation of the LV failure. The prevalence of first degree IAB is much higher than advanced or complete IAB. Really the ECG pattern of advanced IAB is an extremely strong marker of supraventricular tachyarrhythmia in a short period of time, much more so than the presence of first degree or partial Interatrial block. Bayés de Luna A et al.(**Bayés de Luna 1988**) studied 16 patients with ECG evidence of advanced IAB with retrograde activation of the left atrium (LA): P duration ≥120 ms, and plus-minus (+/-) biphasic P waves in inferior leads II, III, and aVF. Eight patients had valvular heart disease, four had dilated cardiomyopathy and four had other forms of heart disease. Patients with valvular heart disease and cardiomyopathy were compared with a control group of 22 patients with similar clinical and echocardiographic characteristics, but without this type of interatrial block. Patients with advanced IAB and retrograde activation of the LA had a much higher incidence of paroxysmal supraventricular tachyarrhythmias (93.7%) during follow-up than did the control group. Eleven of 16 patients (68.7%) with advanced IAB and retrograde activation of LA had atrial flutter (atypical in seven cases, typical in two cases, and with two or more morphologies in two cases). Six patients from the control group (27.7%) had sustained atrial tachyarrhythmias (five AF and one typical atrial flutter). The atrial tachyarrhythmias were due more to advanced IAB and retrograde activation of LA and frequent PACs than to LAE, because the control group with a LA of the same size, but without advanced interatrial block and retrograde activation of LA and with less incidence of PACs, had a much lower incidence of paroxysmal tachycardia.

Bayés de Luna et al.(Bayés de Luna 1989) demonstrated the value of preventive antiarrhythmic treatment in patients with advanced interatrial block. In this population LAE is present in 90% of cases. Using drugs (amiodarone, quinidine or verapamil) this percentage was greatly lowered (25%).

From 81,000 ECGs, Bayes de Luna et al (Bayes de Luna 1985) collected 83 cases that fulfilled the criteria of Interatrial Conduction Disturbances with Left Atrial Retrograde Activation (IACD-LARA) (P +/- in II, III and VF with P width ≥120 ms).

The authors present the detailed study of 35 cases with surface ECG and VCG and 29 cases with orthogonal ECG leads.

The results are then compared against two control groups: with heart disease (30 cases) and without heart disease (25 cases).

The prevalence of IACD-LARA was nearly 1% globally, and 2% among patients with valvular heart disease.

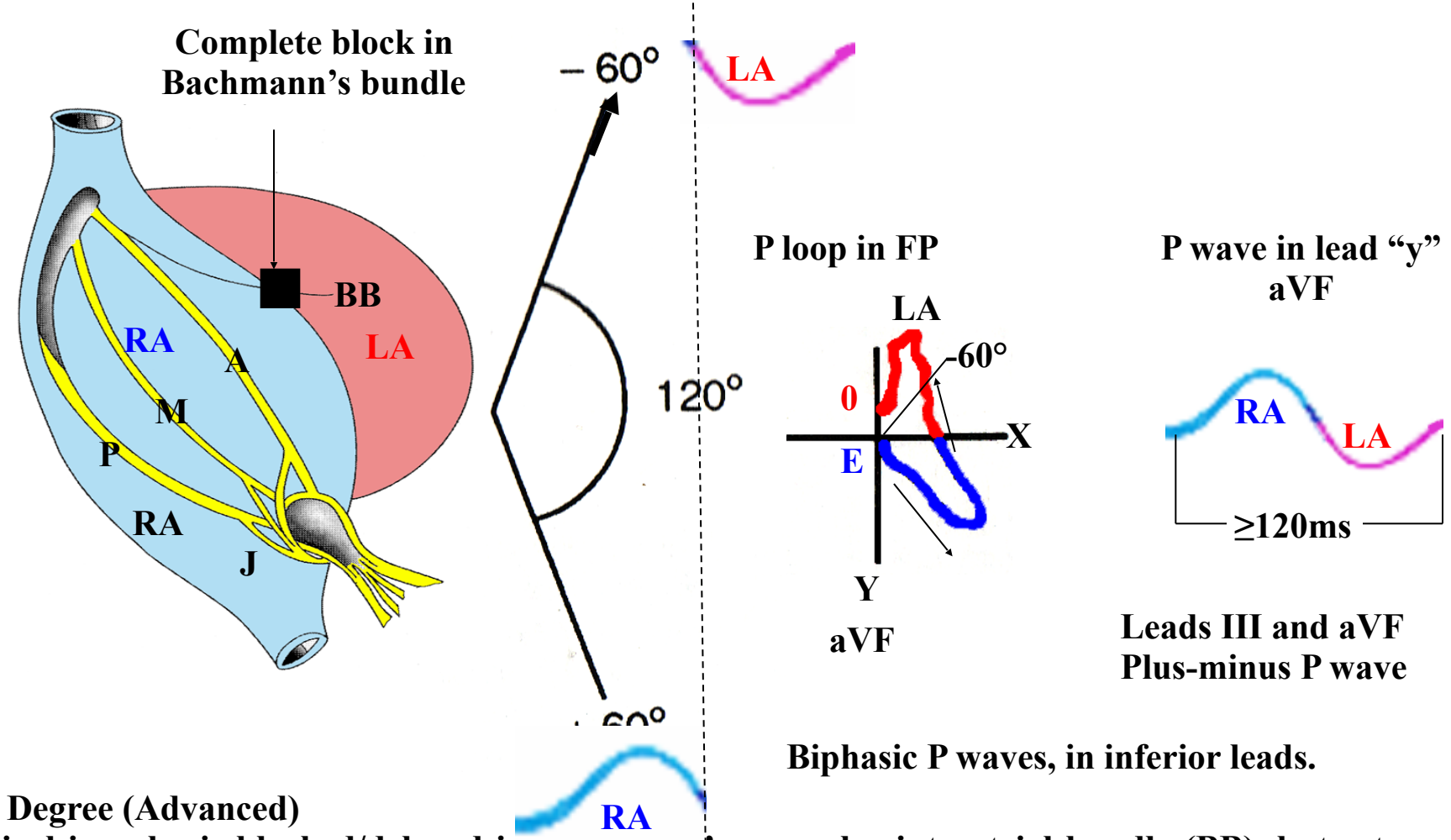
Arrhythmias such as atrial fibrillation and atrial flutter in advanced IAB is observed in > 90% of cases.

Diagnosis criteria of advanced interatrial block and retrograde activation of the left atrium (LA) (Bayés de Luna 1977;1988)(Bayés de Luna 2012) (Bayés de Luna 2014):

1. Biphasic, bifid, or notched "plus-minus" P waves, in inferior leads II, III and aVF of ECG and Y orthogonal lead of VCG
2. P duration ≥120 ms
3. Angle between the first portion (RA) and end portion (LA) >90º
4. Orthogonal Y lead plus-minus with the final negative portion ≥40 ms
5. ≥40 ms final portion of P loop of upstart orthogonal X and Z leads.
6. Final portion of P loop delayed, notches and slurring in the last part of the P loop
7. High Esophageal lead with positive P wave polarity and delayed
8. Low Esophageal lead with plus-minus P wave polarity and delayed
9. Intracavitary ECG with P wave craniocaudal activation inside the RA.
10. Intracavitary ECG with P wave caudal-cranial activation inside LA.

This clinical-electro-vectorcardiographic manifestation of advanced IAB should be considered a syndrome.

Third degree block, complete or advanced interatrial block

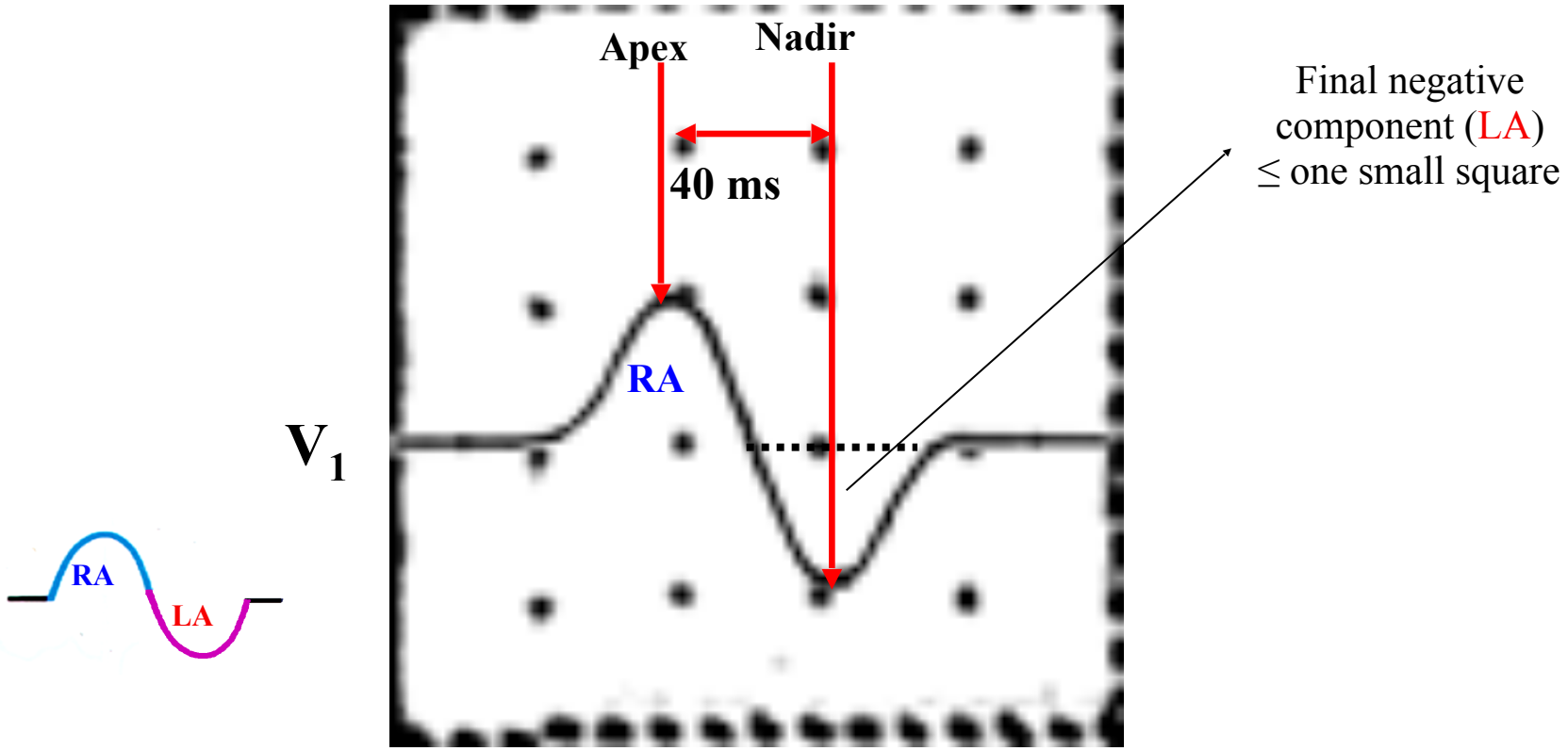


Third Degree (Advanced)

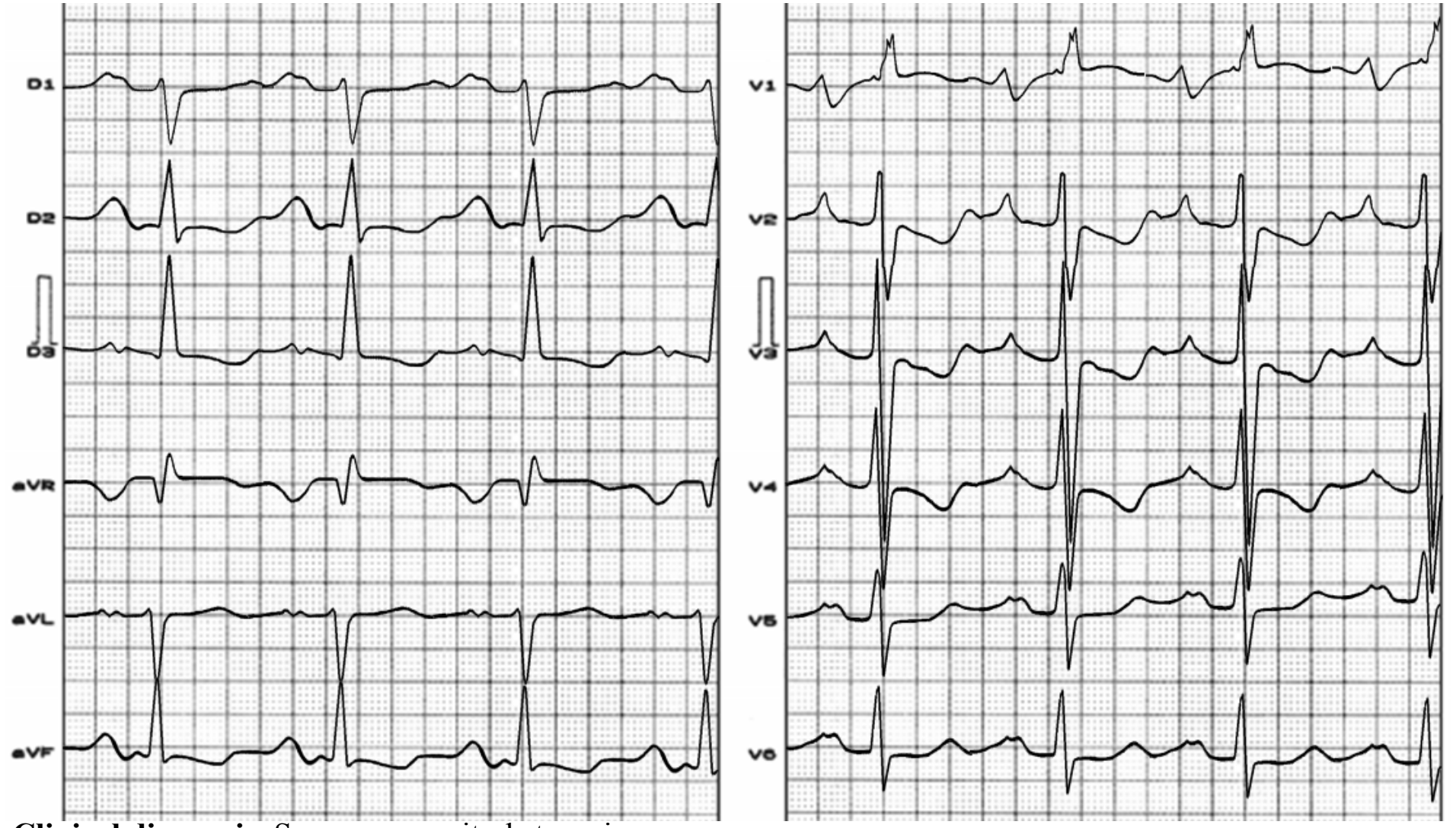
Electrical impulse is blocked/delayed in Bachmann's muscular interatrial bundle (BB), but retrograde left atrial activation usually occurs.(Ariyarajah 2005) Note the existence of an open angle between the vector of the first portion of P wave (RA) and the last portion (LA). Electrophysiological study demonstrates retrograde activation of the LA. Consequently P loop/wave in orthogonal lead "Y", aVF and III is biphasic plus-minus ±. LA activation occurs by an alternate route rather than proceeding from right to left via the BB.(Spodick 2007)

1. P terminal force (Ptf) plus-minus P wave (biphasic configuration) in lead V1≥ the area of one small square; the final minus portion indicates left atrial abnormality, particularly LAE, which is a strong correlate of IAB. (**Ariyarajah 2006**)
2. Prolonged intrinsicoid P-wave deflection (from the apex to nadir) of the biphasic P wave in lead V1 >40 ms.

Representation of normal P wave in V_1



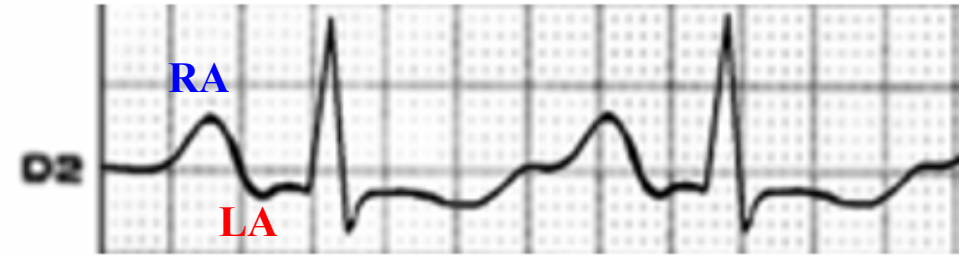
3. Often bifid ("notched") P waves
4. "Dome-and-spike" P waves. 4 and 5 predominatly on leads II and from V_3 to V_6[1].

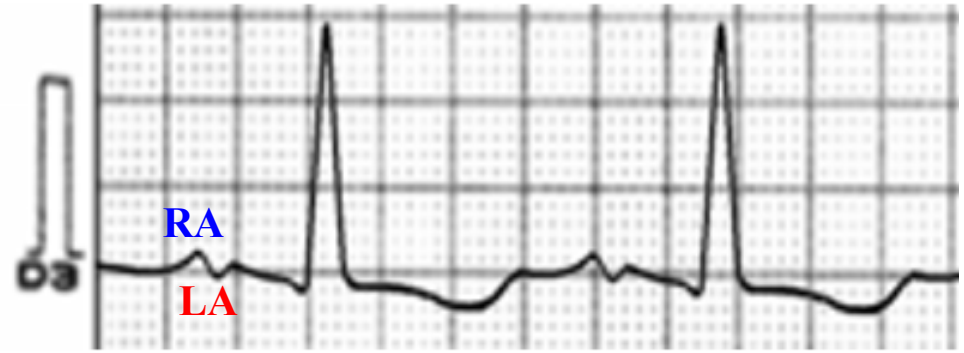


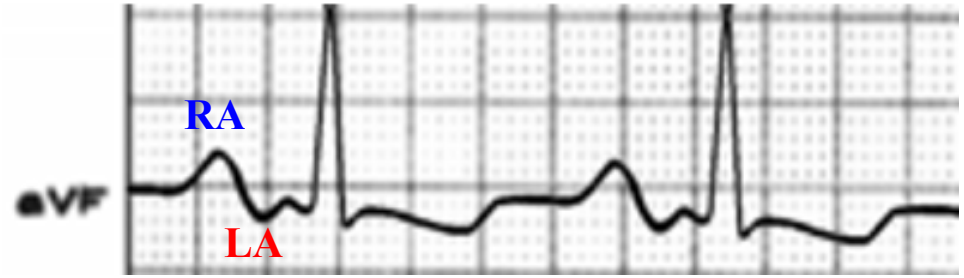
Clinical diagnosis: Severe pure mitral stenosis.

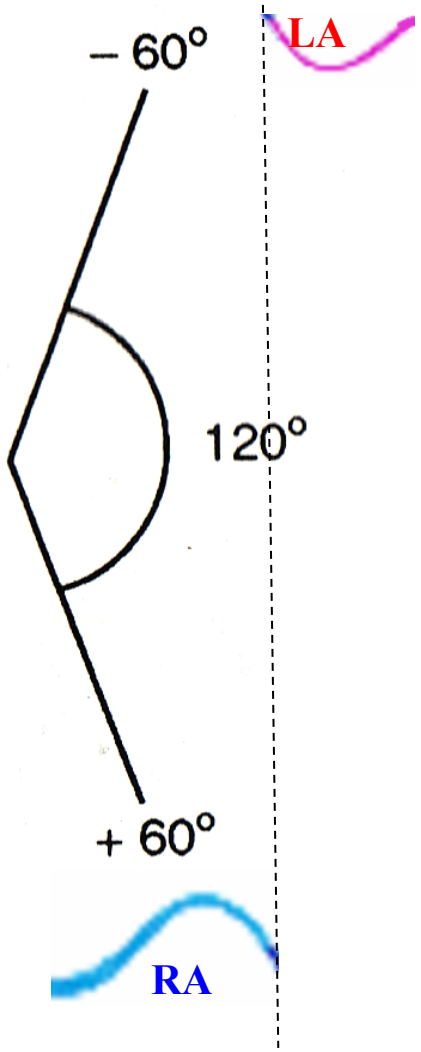
ECG diagnosis: Biatrial enlargement. SÂP: +58°, P waves of voltage >2.5 mm and duration ≥120 ms in II, P waves of duration >120 ms and bimodal plus minus in inferior leads, P wave plus minus with slow and deep terminal component in V1, PR interval 40 ms: first degree AV block, QRS axis +130°, prominent final R wave in aVR, pure R wave in V1 and R = S in V5-V6: RVH.

Conclusion: Biatrial enlargement, third-degree interatrial block, first-degree AV block, right ventricular hypertrophy.


RA
D2
LA


RA
D3
LA


RA
aVF
LA


LA
– 60°
120°
+ 60°
RA

Reasons why interatrial atrial blocks are little known

1. Most of the literature studies employ only lead II or a combination of 2 or 3 leads (**Stefanadis 2001**)
2. Any of the 12 leads of the standard ECG may have the widest P waves, which establish the magnitude of block. Given the high prevalence of IAB in hospital patients and its ominous portents (LA enlargement, thrombosis and embolism, arrhythmias), physicians should be aware of its frequency and computer software should be programmed to recognize it. (Jairath 2001).
3. Much of the literature concerning IAB has loosely named it for one of its correlates in nearly every case; in IAB an abnormally wide P is seen on ECG and IAB should be named by its precise name
4. ECGs encoding systems do not have a code for IAB

Bayés de Luna A et al.(**Bayés de Luna 1988**) studied 16 patients with ECG evidence of advanced interatrial block with retrograde activation of the left atrium (LA): P duration ≥120 ms, and plus-minus (+/-) biphasic P waves in inferior leads II, III, and VF.

Eight patients had valvular heart disease, four had dilated cardiomyopathy and four had other forms of heart disease. Patients with valvular heart disease and cardiomyopathy were compared with a control group of 22 patients with similar clinical and echocardiographic characteristics, but without this type of interatrial block.

Patients with advanced interatrial block and retrograde activation of the LA had a much higher incidence of paroxysmal supraventricular tachyarrhythmias (93.7%) during follow-up than did the control group. Eleven of 16 patients (68.7%) with advanced interatrial block and retrograde activation of LA had atrial flutter (atypical in seven cases, typical in two cases, and with two or more morphologies in two cases). Six patients from the control group (27.7%) had sustained atrial tachyarrhythmias (five atrial fibrillation and one typical atrial flutter). The atrial tachyarrhythmias were due more to advanced interatrial block and retrograde activation of LA and frequent PACs than to LAE, because the control group with a LA of the same size, but without advanced interatrial block and retrograde activation of LA and with less incidence of PACs, had a much lower incidence of paroxysmal tachycardia.

Bayés de Luna et al.(**Bayés de Luna 1989**) demonstrated the value of preventive antiarrhythmic treatment in patients with advanced interatrial block. In this population LAE is present in 90% of cases. Using drugs (amiodarone, quinidine or verapamil) this percentage was greatly lowered (25%).

Atrial tachyarrhythmias such as atrial fibrillation and atrial flutter in advanced IAB is observed in >90% of cases.

•From 81,000 ECGs, Bayes de Luna et al (Bayes de Luna 1985) collected 83 cases that fulfilled the criteria of Interatrial Conduction Disturbances with Left Atrial Retrograde Activation (IACD-LARA) (P +/- in II, III and VF with P width ≥120 ms).

•The authors present the detailed study of 35 cases with surface ECG and VCG and 29 cases with orthogonal ECG leads.

•The results are then compared against two control groups: with cardiopathy (30 cases) and without cardiopathy (25 cases).

•The prevalence of IACD-LARA was nearly 1% globally, and 2% among patients with valvular heart disease.

•The diagnostic criteria for Interatrial Conduction Disturbances with Left Atrial Retrograde Activation (IACD-LARA) are:

1. ECG: P +/- in II, III and VF with P ≥120 ms.
2. Open angle >90º between the first and the second part of the P.
3. Orthogonal ECG: P +/- in Y lead with a negative mode greater than 40 m.
4. VCG: More than 50 ms above the X or Z axis
5. Duration of the P loop ≥110 ms
6. Open angle between the two parts of the P loop in both the frontal and right sagittal planes
7. Presence of notches and slurring in the last part of the P loop.

Treatment Options (Spodick 2004)

Reduction of interatrial block

Prevention of atrial fibrillation an other arrhythmias

Pacemaker

- Biatrial: DD pacemakers with dual atrial leads with synchronous biatrial pacing correct inter-atrial assynchrony and also prevent arrhythmia recurrence.(Dubert 1994)
- Pacing
- Right atrial
- Atrial multisite
- Atrial septal
- Bachmann bundle (**Bailing 2005**) in patients undergoing coronary artery bypass surgery (CABGS). Bachmann bundle pacing is superior to right atrial / no pacing in the post operative period for preventing occurrence of AF and reducing intensive care unit stay, commensurate with a reduction in mean P wave duration on surface ECG.(**Chavan 1011**)
- Ventricular.

Nonelectric approach

- Reduction of contributory commorbidities
- Myocardial failure: LV disorders; correcting these disorders may reverse the LA abnormalities.
- Inflammatory markers (e.g. high-sensitivity C-reactive protein)
- Ectopic beats
- Valve abnormalities
- Metabolic abnormalities
- Anticoagulation
- Angiotensin converting enzyme inhibition
- Angiotensin receptor blockers (**Mehrzad 2014**)
- Antiarrhythmic: Bayés de Luna et al,(**Bayés de Luna 1989**) demonstrated the value of preventive antiarrhythmic treatment in patients with advanced IAB. In this population LAE is present in 90% of cases. Using drugs (amiodarone, quinidine or verapamil) this percentage was greatly lowered (25%)

Observation: All suggested modalities should undergo appropiately designed, prospective ramdomized clinical trials with sufficient statistical power.

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