# C R T

## (Cardiac resynchronization therapy)

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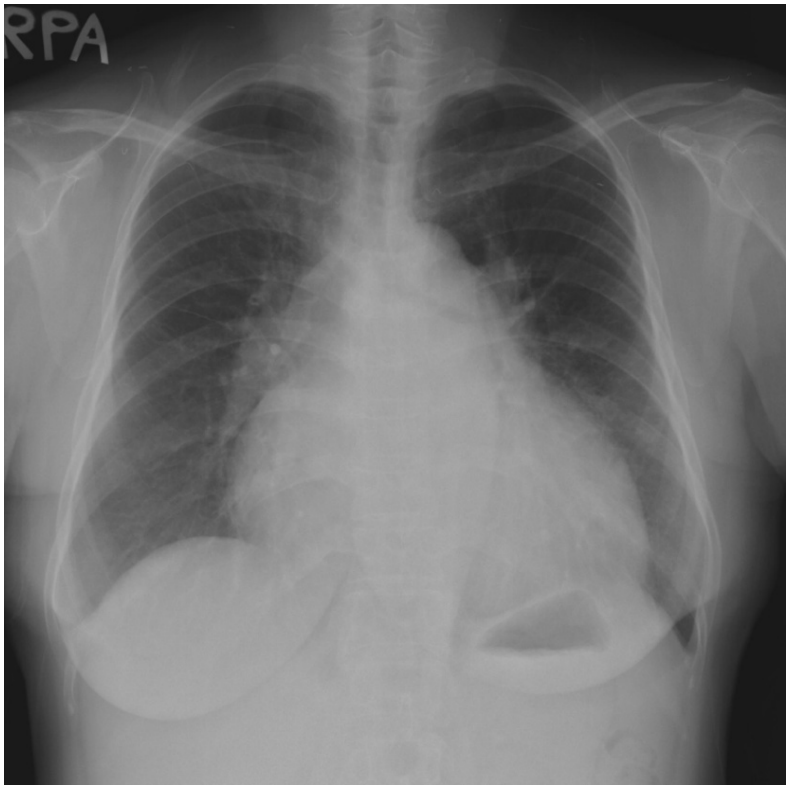
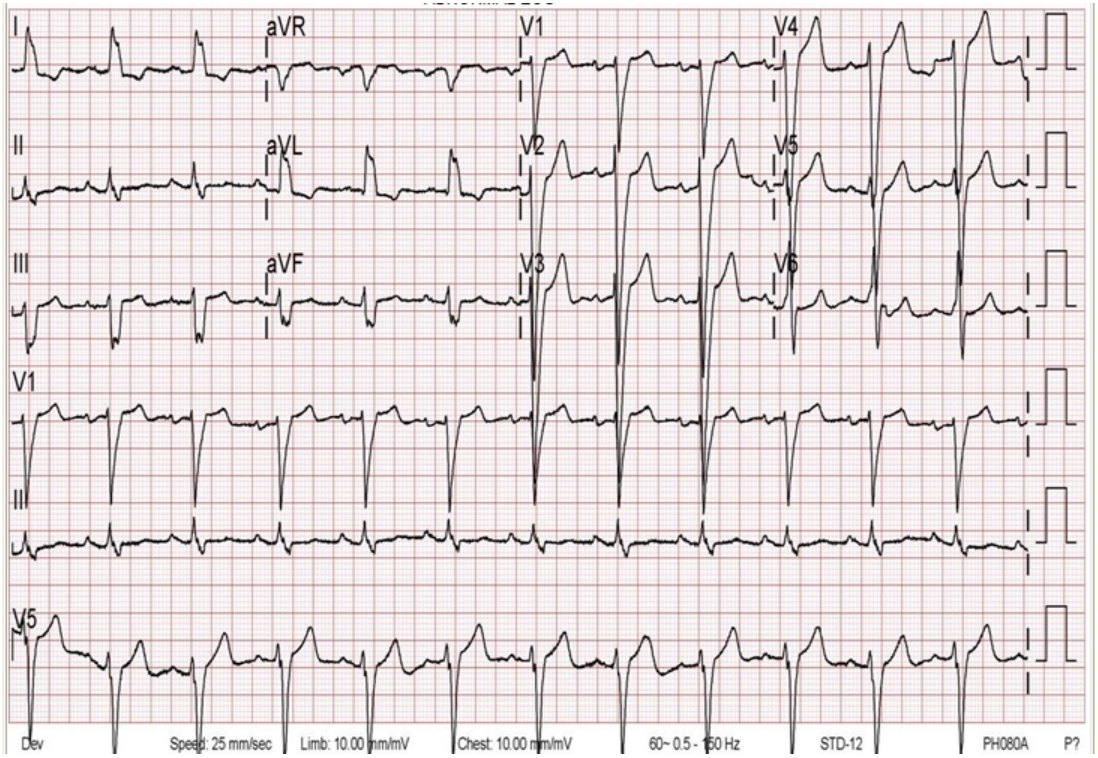
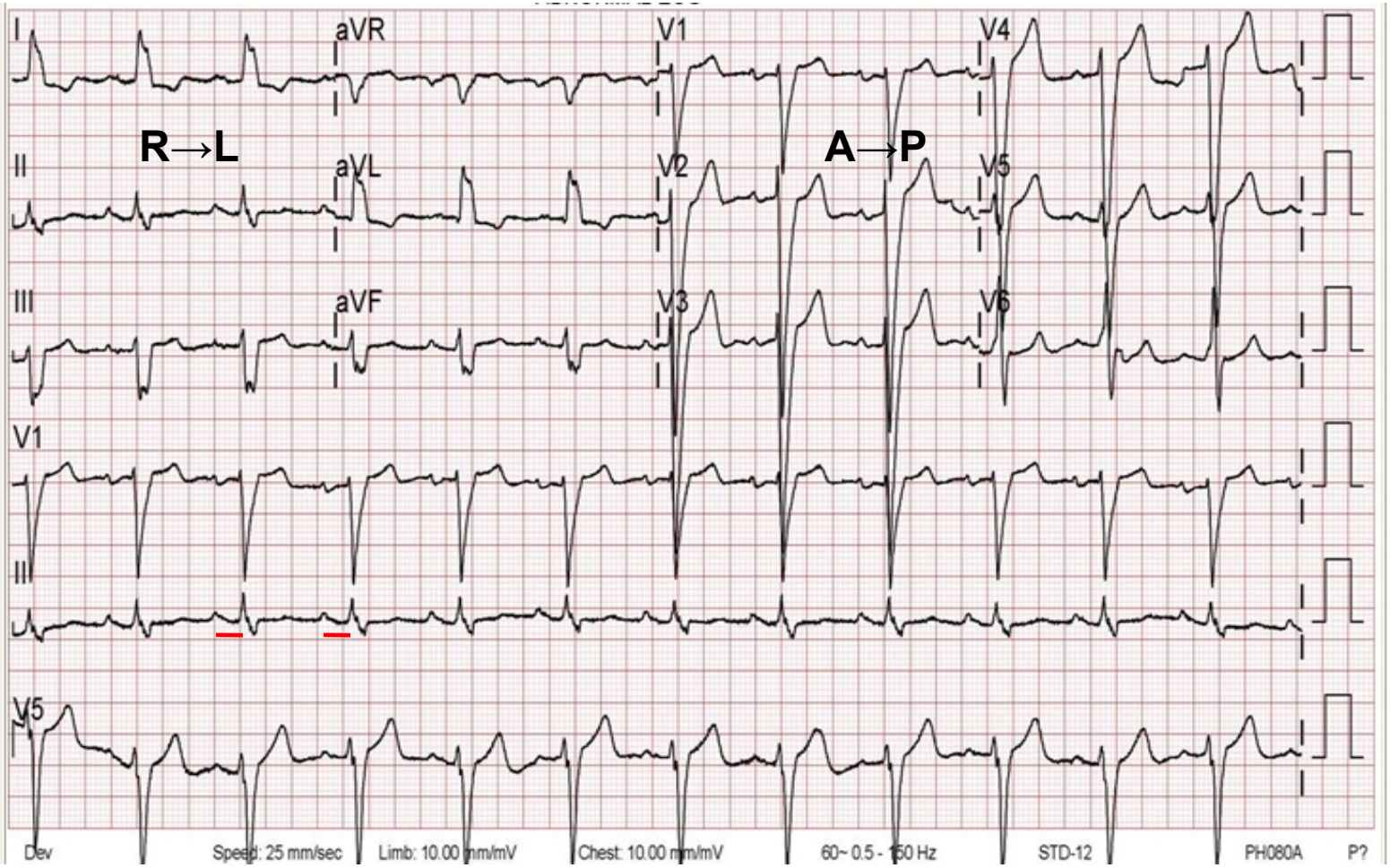
# 증 례

65 y/ F  10년 전 부터 심부전 치료 (ACE+beta blocker+aldactone),
최근 호흡곤란 악화( NYHA III, IV )



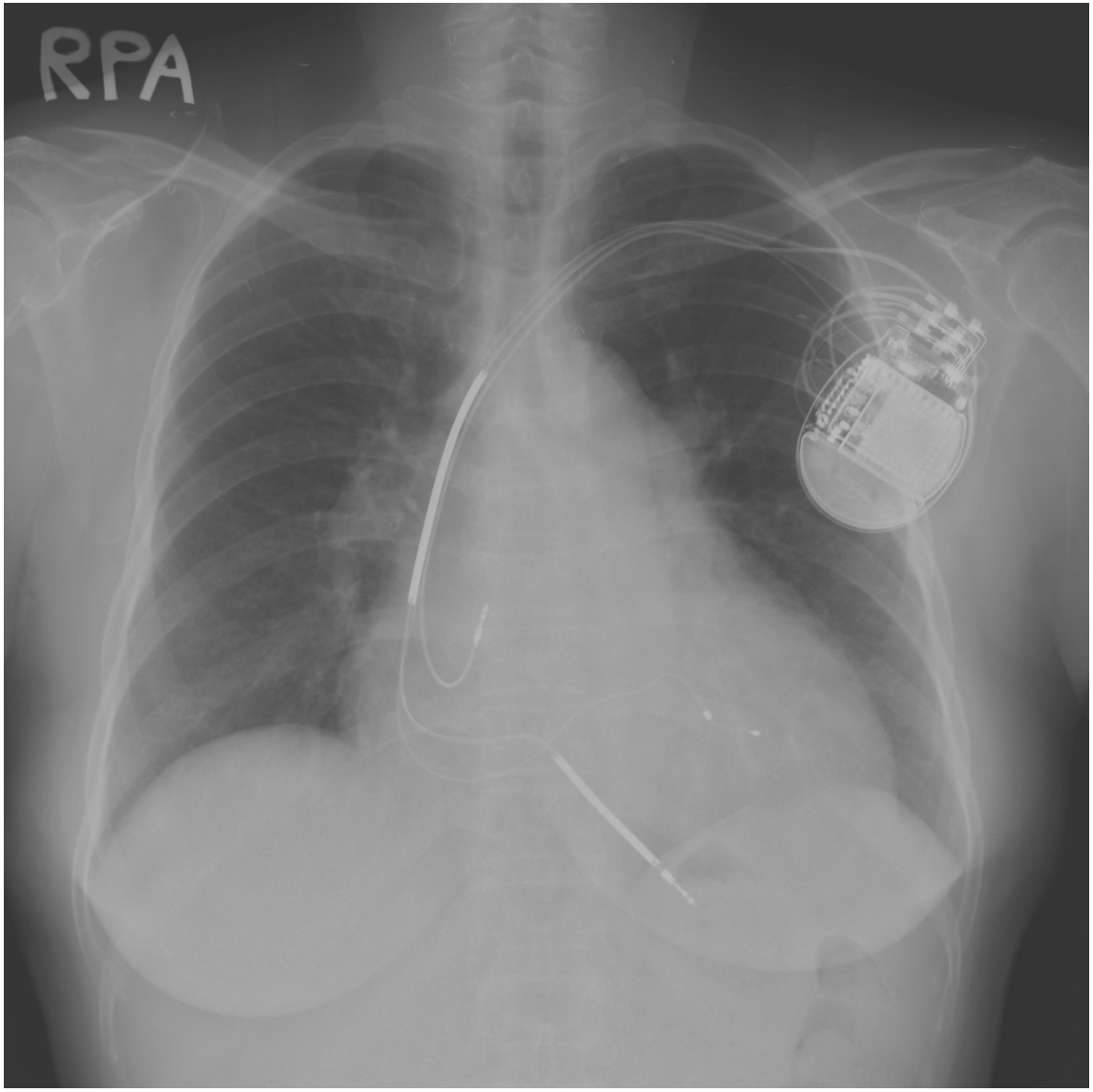



# Where is the earliest activation site ?



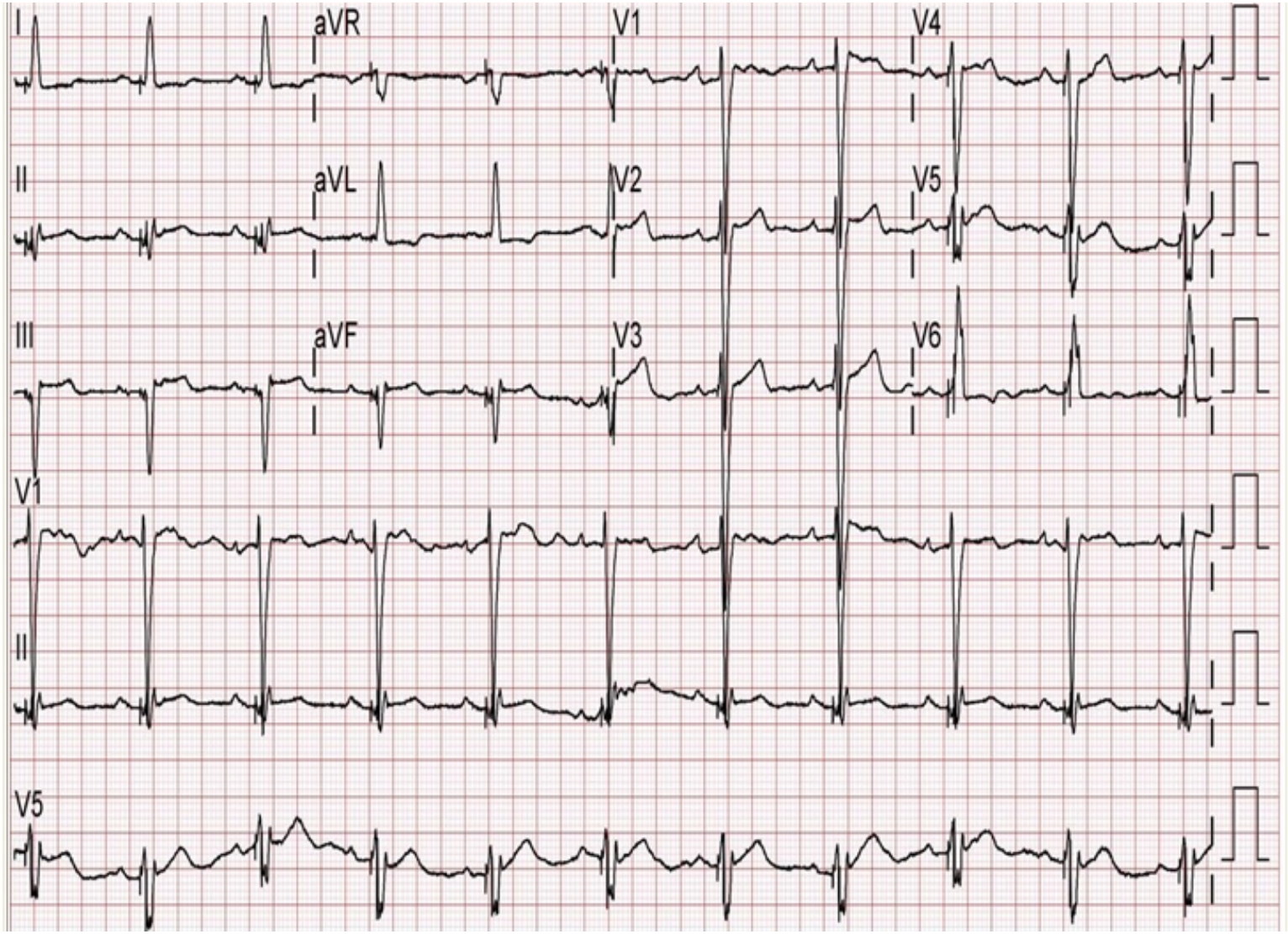

1. RV free wall　2. septum　3. LV lateral wall

# CXR (post CRT)



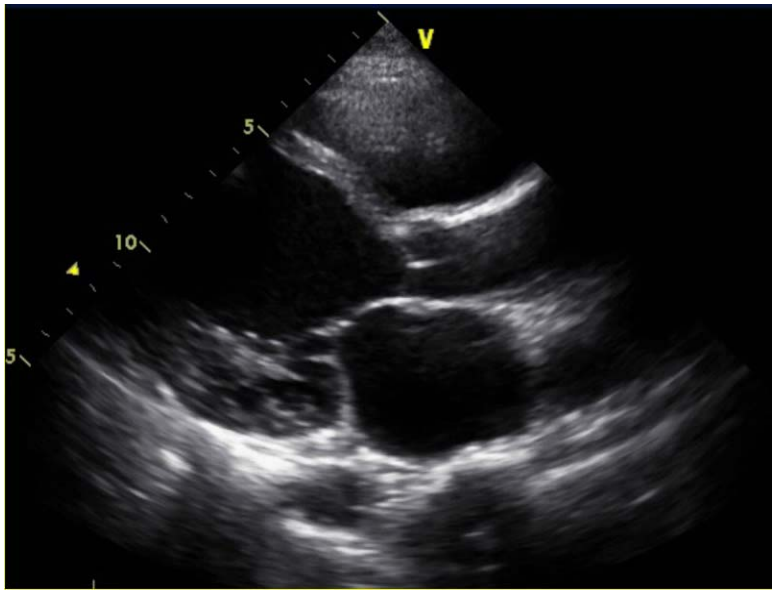
# Just after CRT

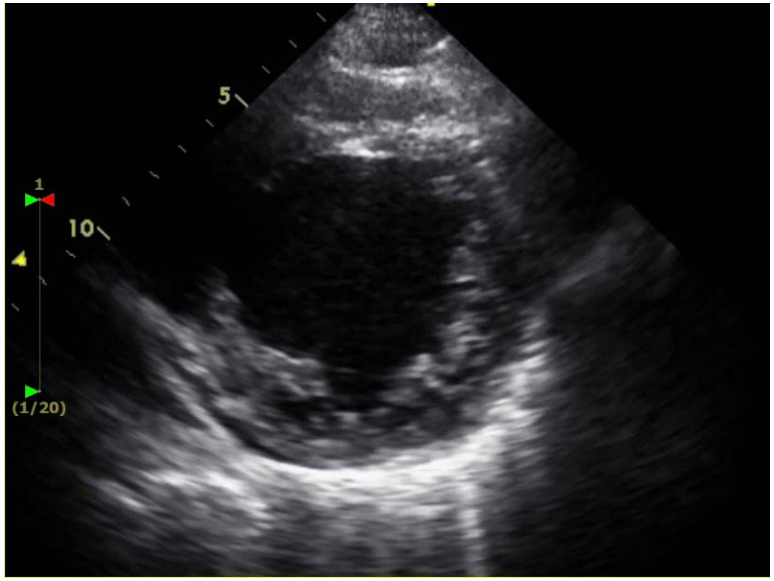
**Biv, 3.5V @ 0.5ms(160/170 ms), LV→RV 50**



## Pre CRT

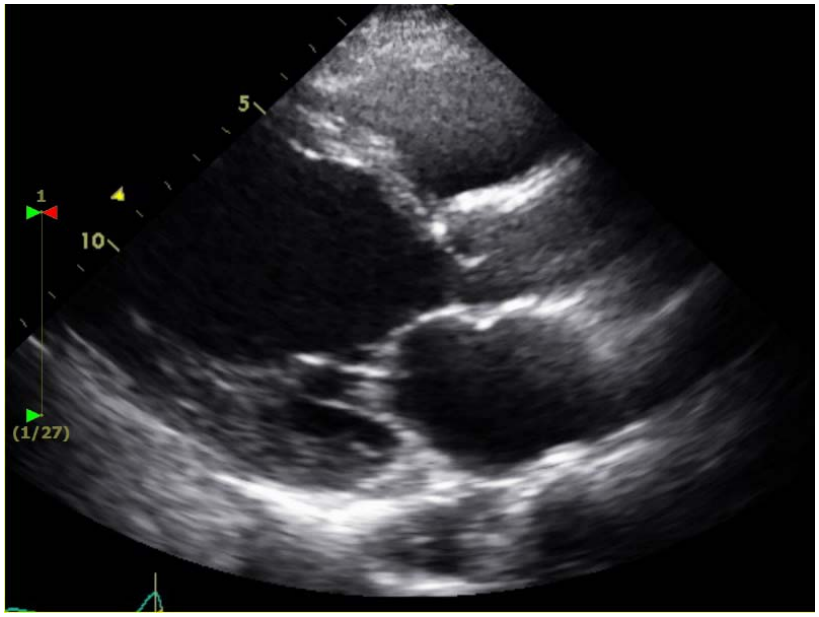
**EDV/ESV : 241 ml/189 ml, EF 23%**

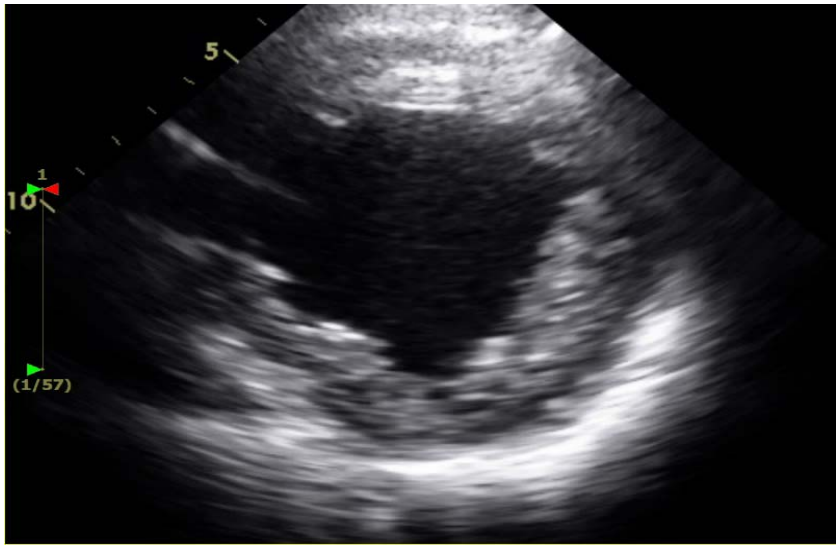



## 12 Months follow-up

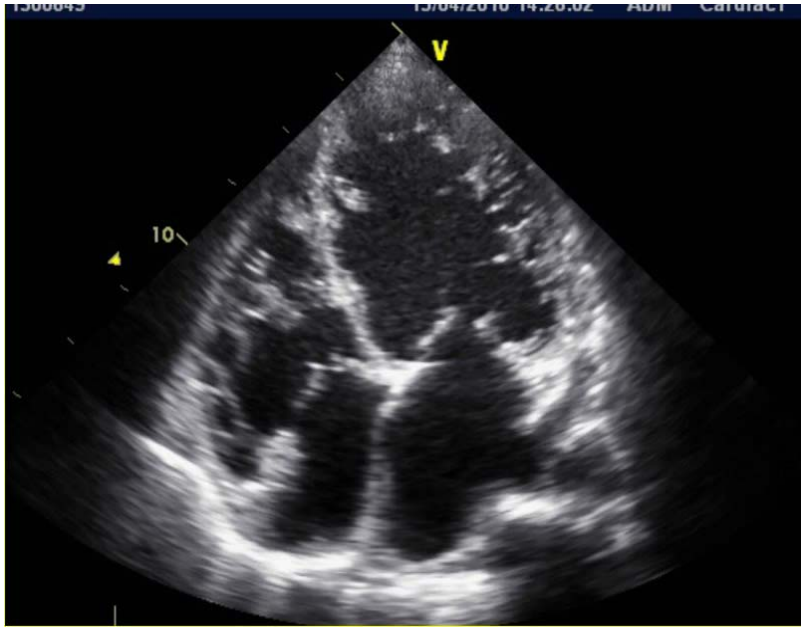
**EDV/ESV : 253 ml/165 ml, EF 34%**

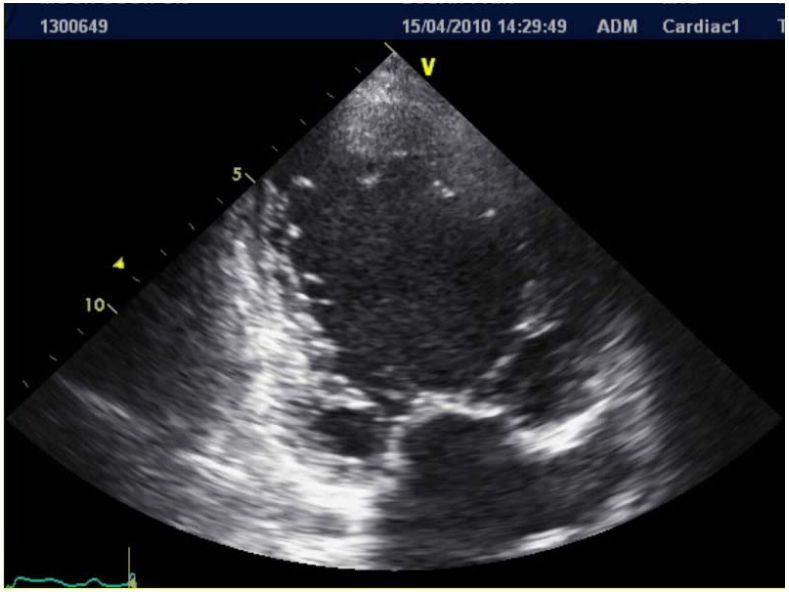


## Pre CRT

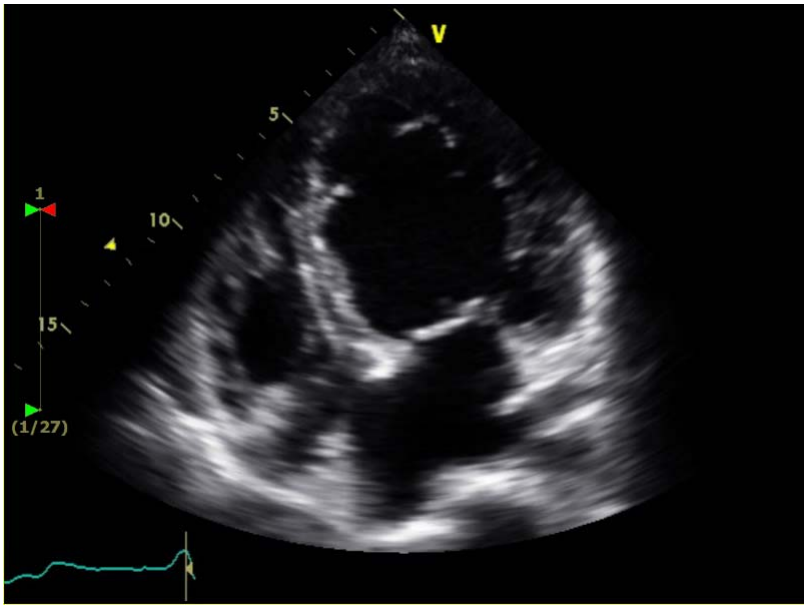
**EDV/ESV : 241 ml/189 ml, EF 23%**

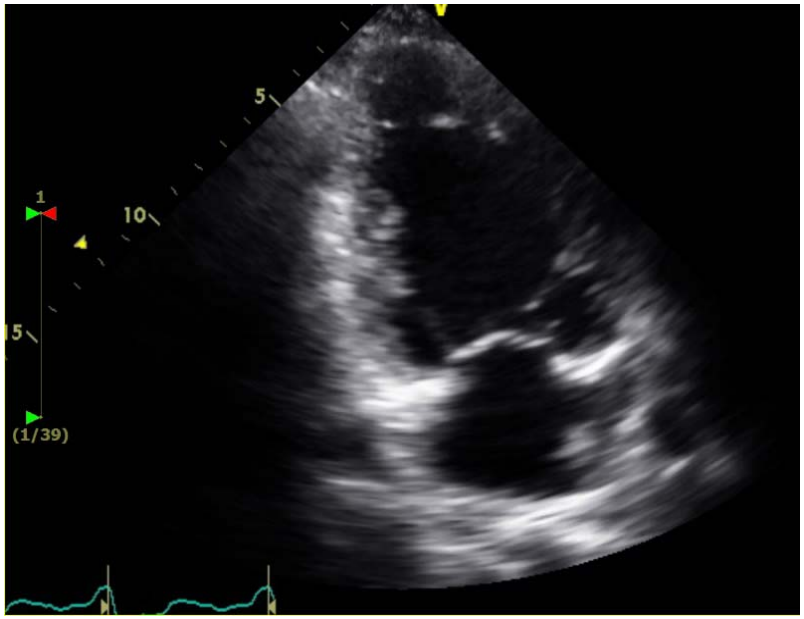


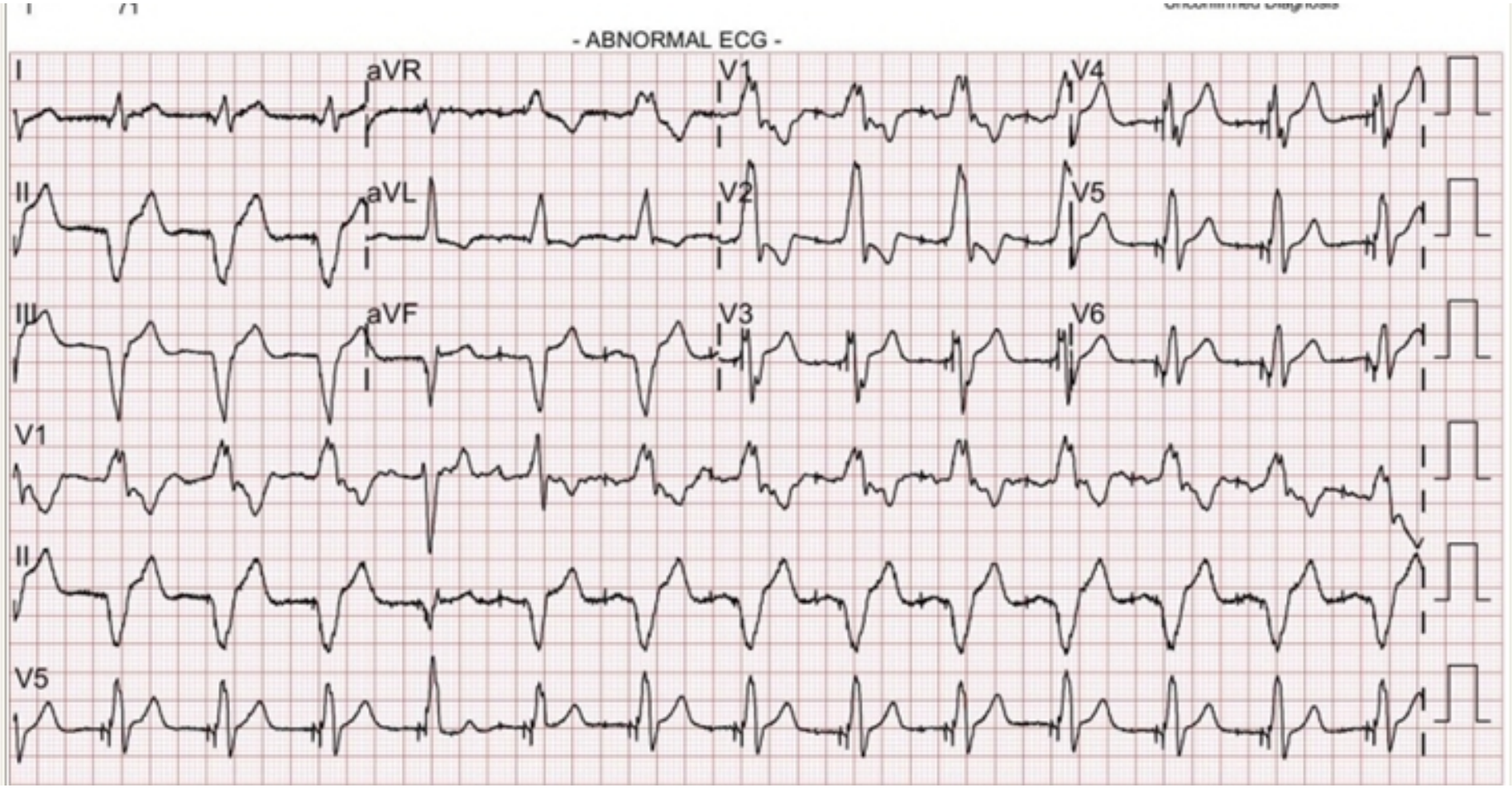

## 12 Months follow-up

**EDV/ESV : 253 ml/165 ml, EF 34%**

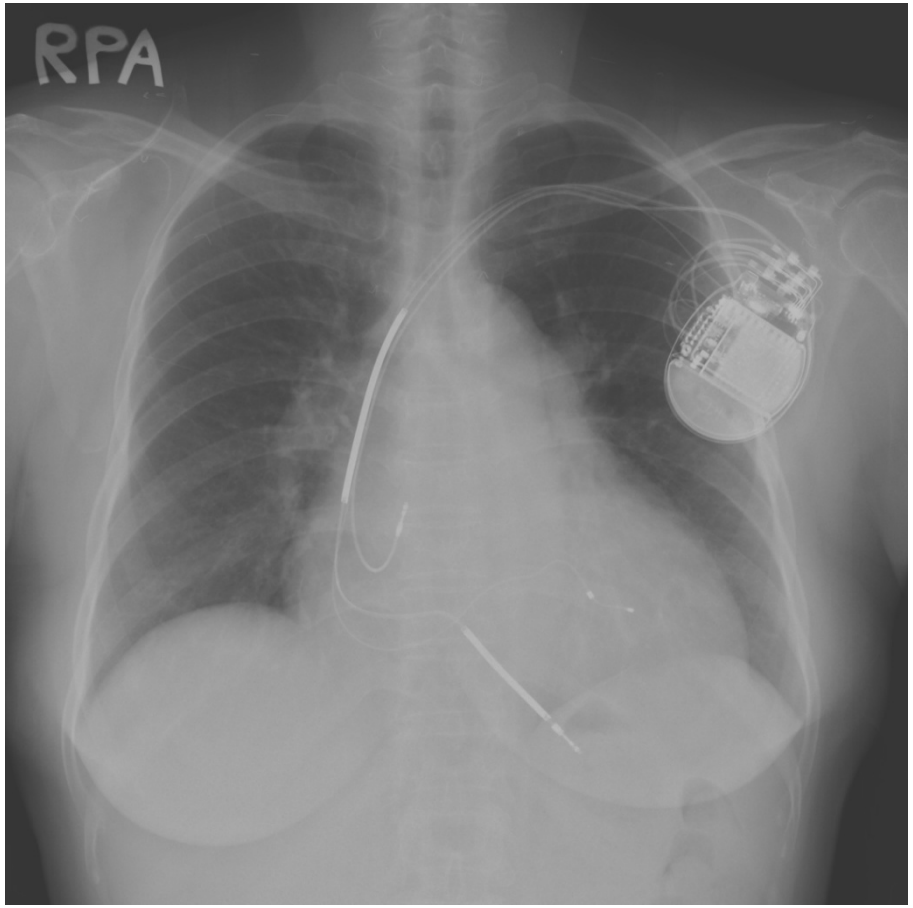


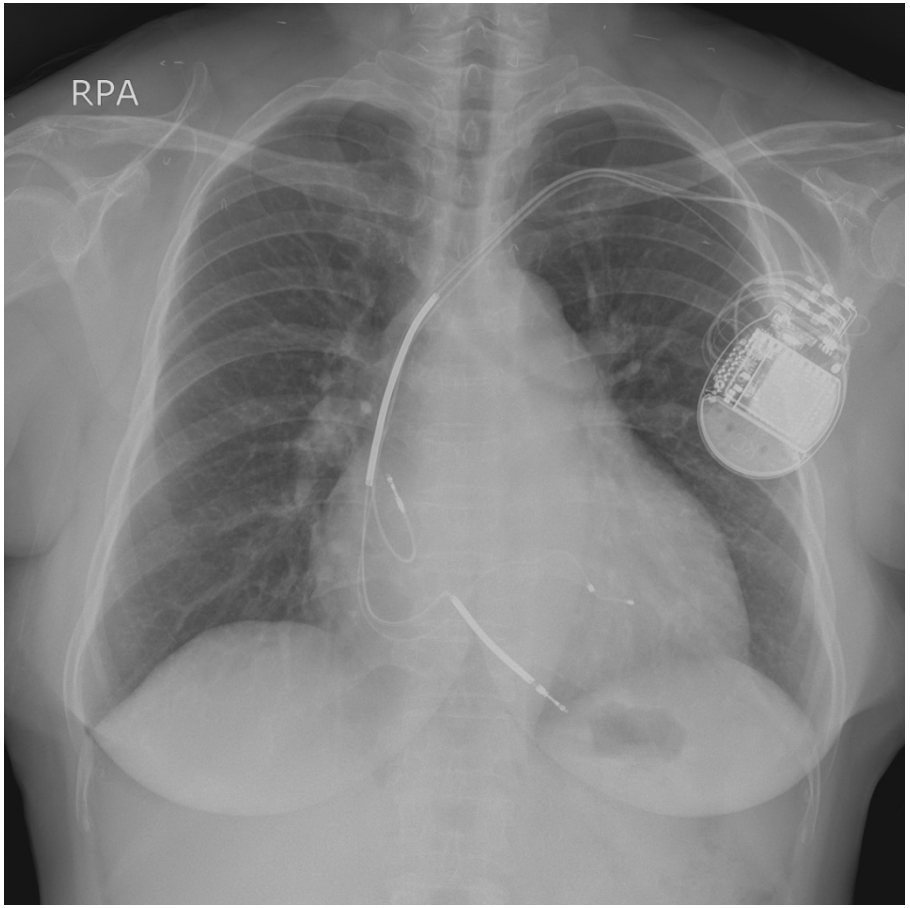



# 20 month after CRT



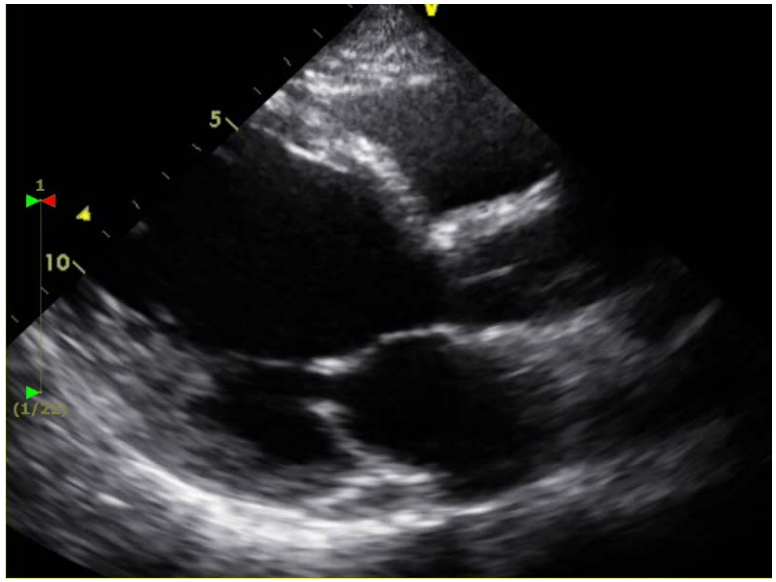
# CXR F/U

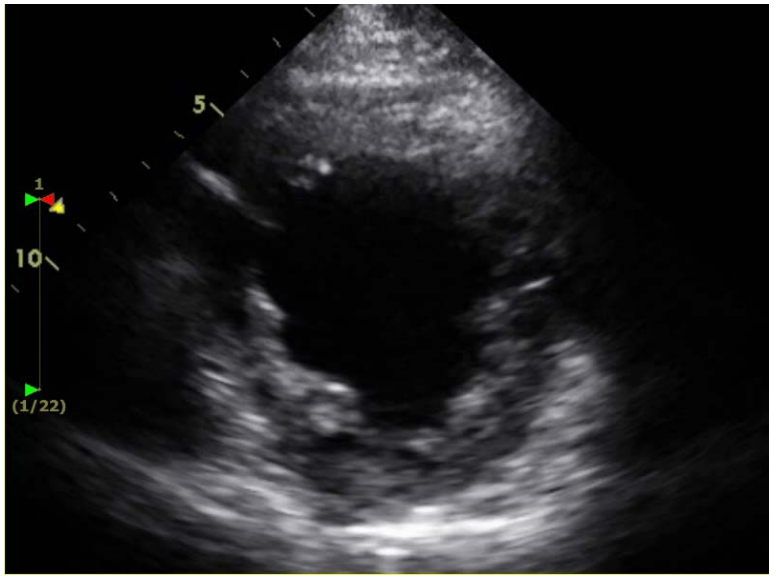


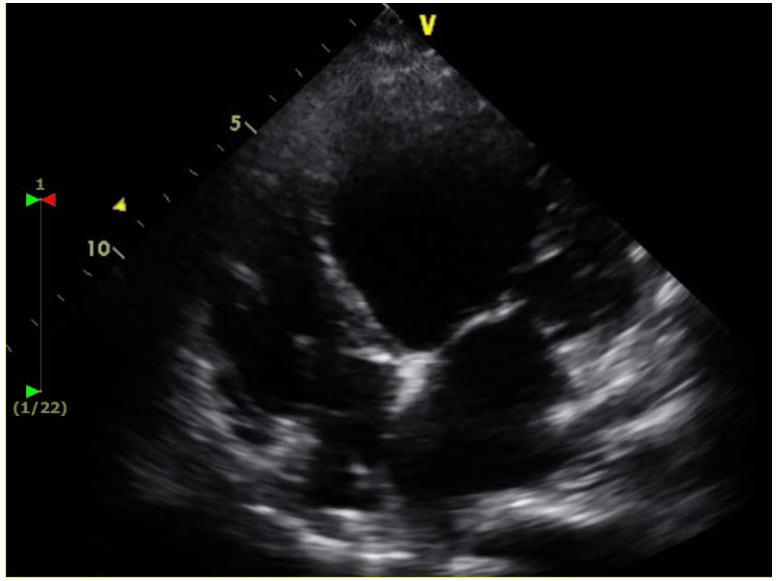



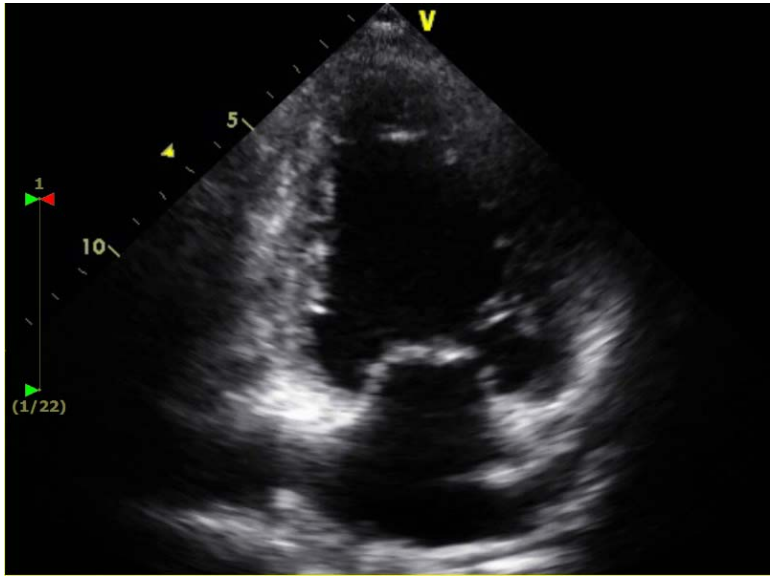
# Current Echocardiography

## EDV/ESV : 244 ml/163 ml, EF 33%



# What happened to QRS ?

- Capture failure
- RV anodal capture
- Ventricular fusion
- Latency
- Disease progression

# Background

- Electromechanical dyssynchrony; discoordinated contraction
    1. Interatrial dyssynchrony
    2. Atrioventricular dyssynchrony
    3. Interventricular dyssynchrony
    4. Intraventricular dyssynchrony
- Suboptimal ventricular filling
- Reduction in LV contractility
- Prolonged duration of mitral regurgitation
- Paradoxical septal wall motion

--- further deteriorate HF

# Cardiac resynchronization therapy

- Improved functional capacity
- Decreased hospitalization
- Reverse remodeling

-Small amplitude of reduction in LV volume and increase in EF within first 24 hours

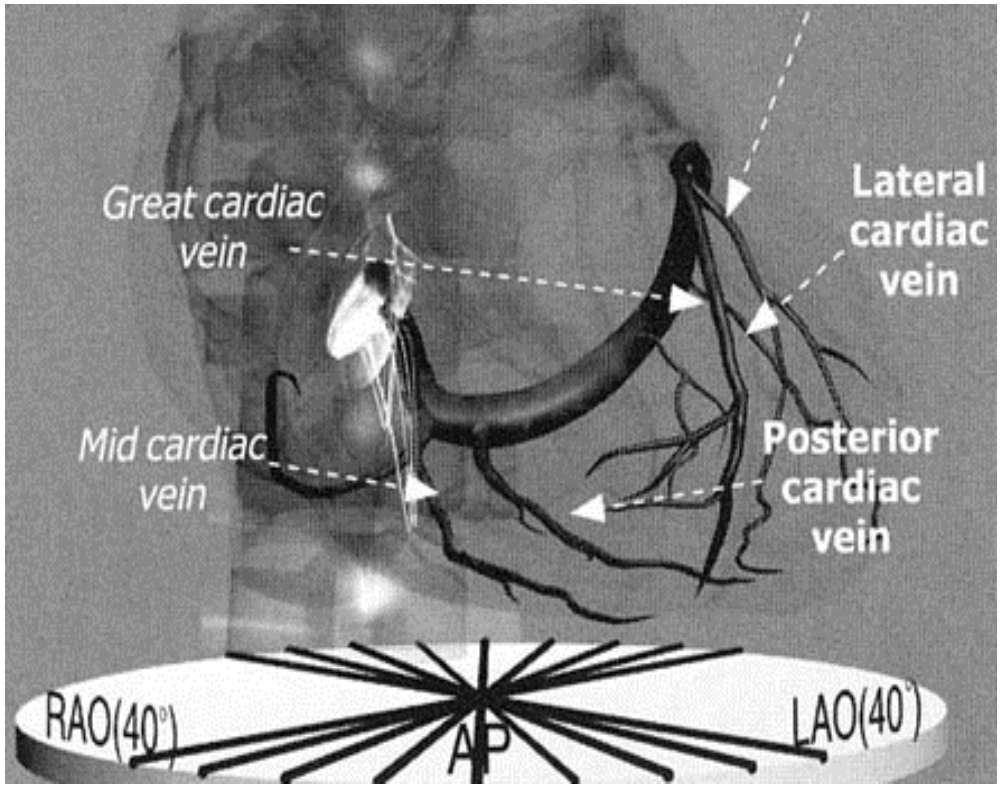
- Decrease in LV mass an LV wall thickness at the end of 3 months

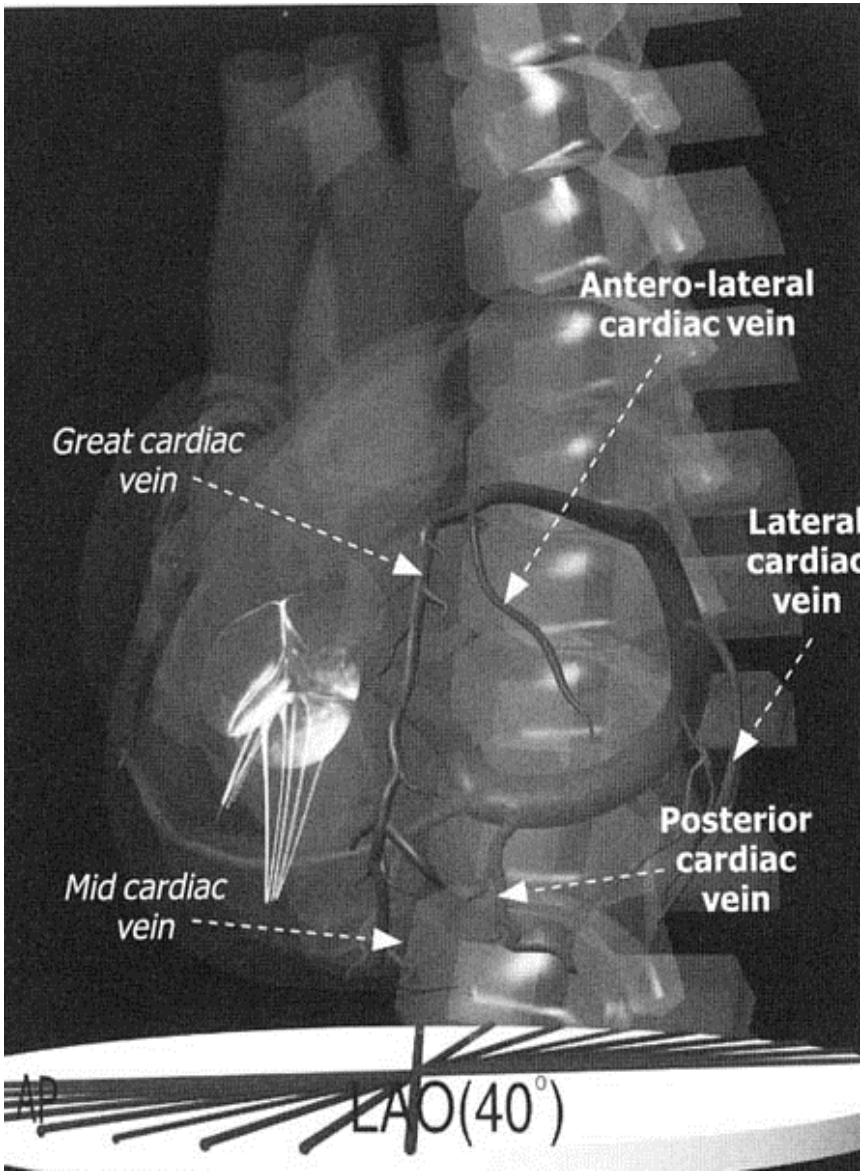
- LV volume increased gradually over 4 weeks after cessation of CRT
  ; pacing is the cause of reverse remodeling

# Implantation

- RV lead first ( apex or septum) during implantation
- Implantation of the left ventricular lead through coronary sinus to the free wall or RA
- Advance LV lead into the vein as distally as possible
- Epicardial LV pacing

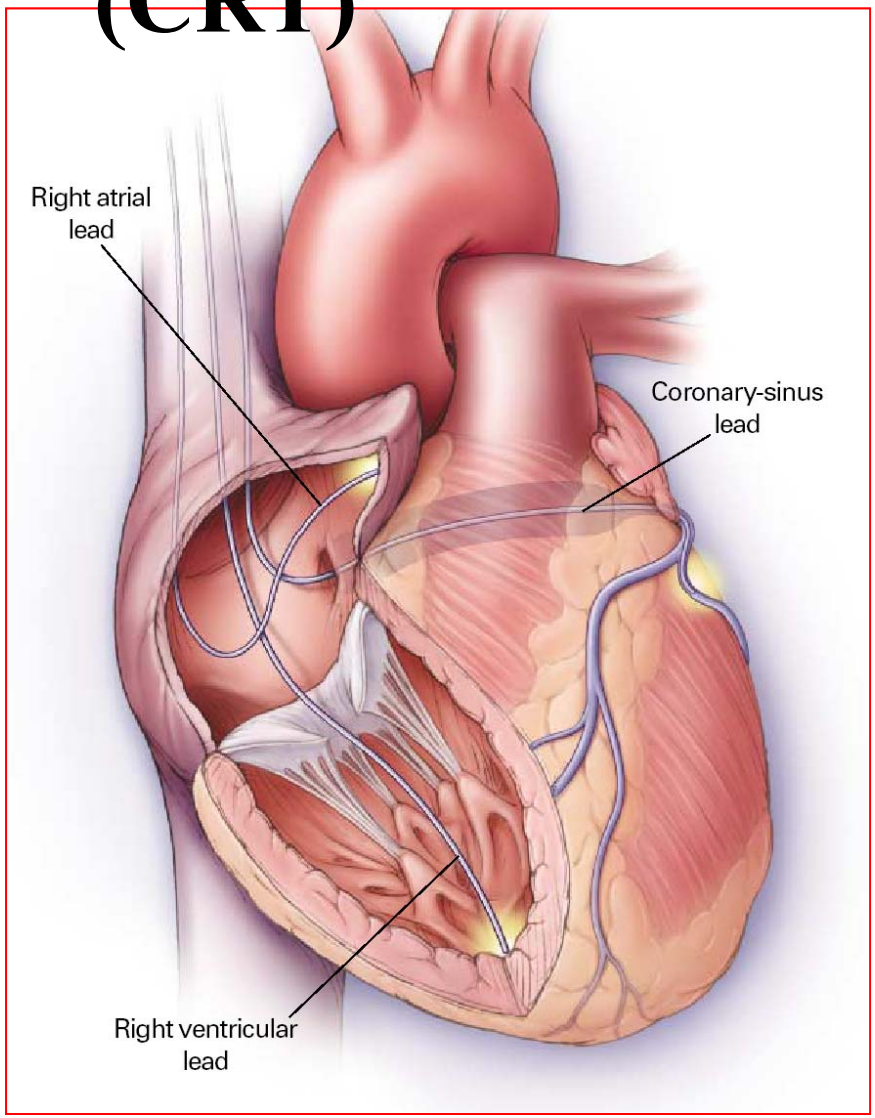
# Optimal LV lead positioning ?




lateral vein > posterior > anteolateral cardiac vein
Great cardiac vein–middle cardiac **vein - avoid** d/t cross septum
Stimulation of LV apex, anterior wall– avoid

# Cardiac Resynchronization Therapy (CRT)




**Randomized controlled trials**

MUSTIC studies

MIRACLE trials

CONTAK CD trial

CARE HF trial

COMPANION trial

REVERSE trial

MADIT-CRT

# How to select candidate for CRT

- Dilated CM; EF ≤ 35%
- NYHA functional class III/IV despite optimal medication
- NSR,  prolonged QRS complex

- Ventricular dyssynchrony
  - QRS ≥ 130 ms
  - QRS< 130 ms : echo criteria
    1. aortic preejection delay ≥ 140 ms
    2. interventricular mechanical delay ≥ 40 ms
    3. LV segmental post  systolic contraction
    4. intraventricular dyssynchrony > 65msec

# Non-responder

- Inappropriate patient selection

  - the absence of significant mechanical dyssynchrony

  ( **Wide QRS ≠ mechanical dyssynchrony** )

  - ischemic, uncontrolled
- Suboptimal location of LV lead
- Loss of LV pacing d/t lead dislodgement
- Incorrect programming of the device

  - AV delay - VV delay

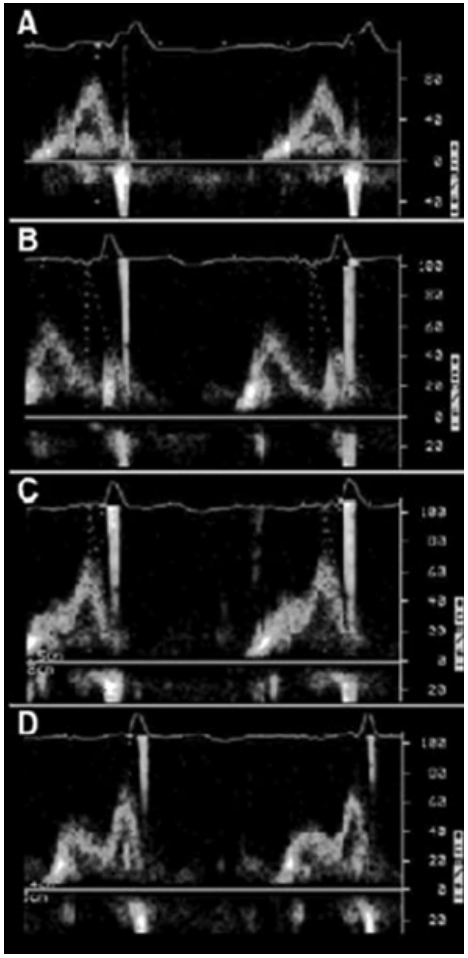
  - RV anodal stimulation ( capture)
- Uncontrolled Arrhythmia
- Suboptimal medical treatment
- Ventricular scar, heart disease progression

# Optimization of CRT

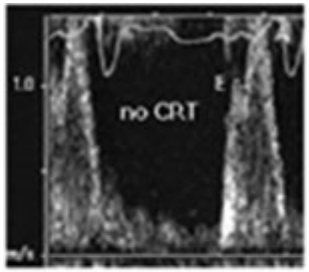
- ❖ AV synchrony and biventricular pacing > 90%
- ❖ Optimal AV and VV delays
- ◆ Methods for AV optimization
  - Echocardiographic methods
  - Device-based methods
  - Surface EKG
- ◆ Methods for VV optimization
  - Echocardiographic methods
  - Device-based methods
  - Surface EKG

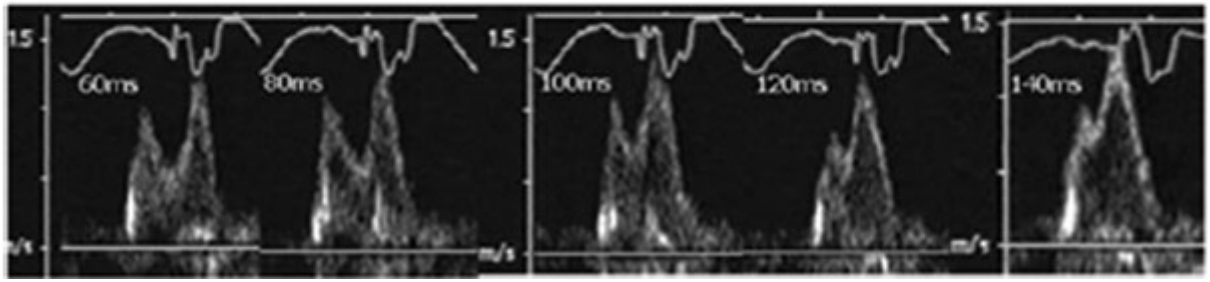
*J Cadiovasc Electrophysiol, Vol.23.110-118, Jan 2012*

# Echocardiographic methods




Ritter method




Iterative method(M/C)
- Maximal E/A separation

# Methods for VV optimization

- Echocardiographic technique
  - maximal aortic VTI

- IEGM technique ( QuickOpt)
  - RV and LV paced wavefronts meet at VV septum

- Surface EKG method
  - shortest QRS duration or the fastest QRS deflection

# 12 leads EKG

1. Balance btn RV and LV activation; dorminant R(±) in V1

2. The presence or absence of fusion with the conducted intrinsic QRS complex

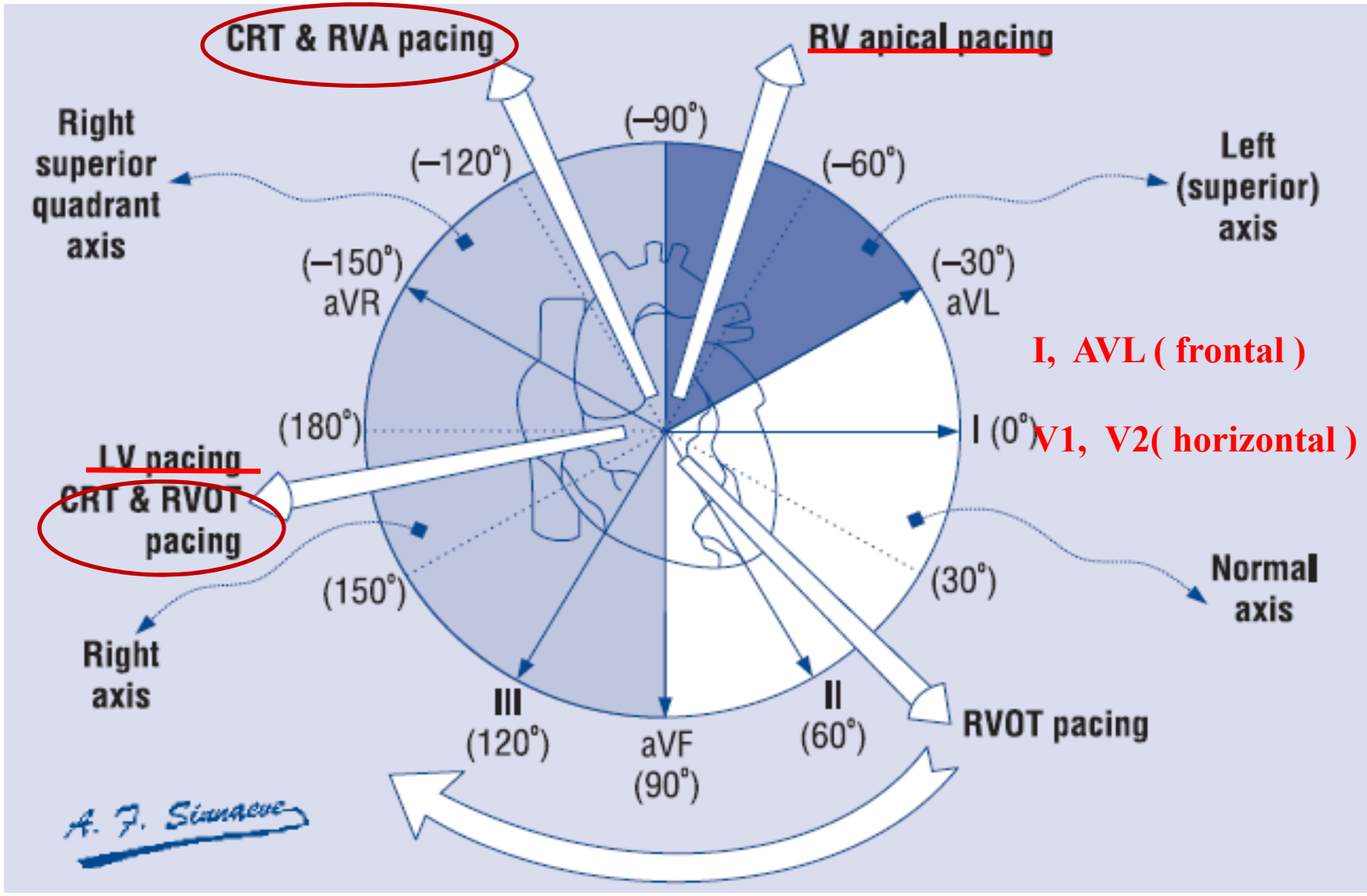
3. The presence of RV anodal capture

   * QRS comparison

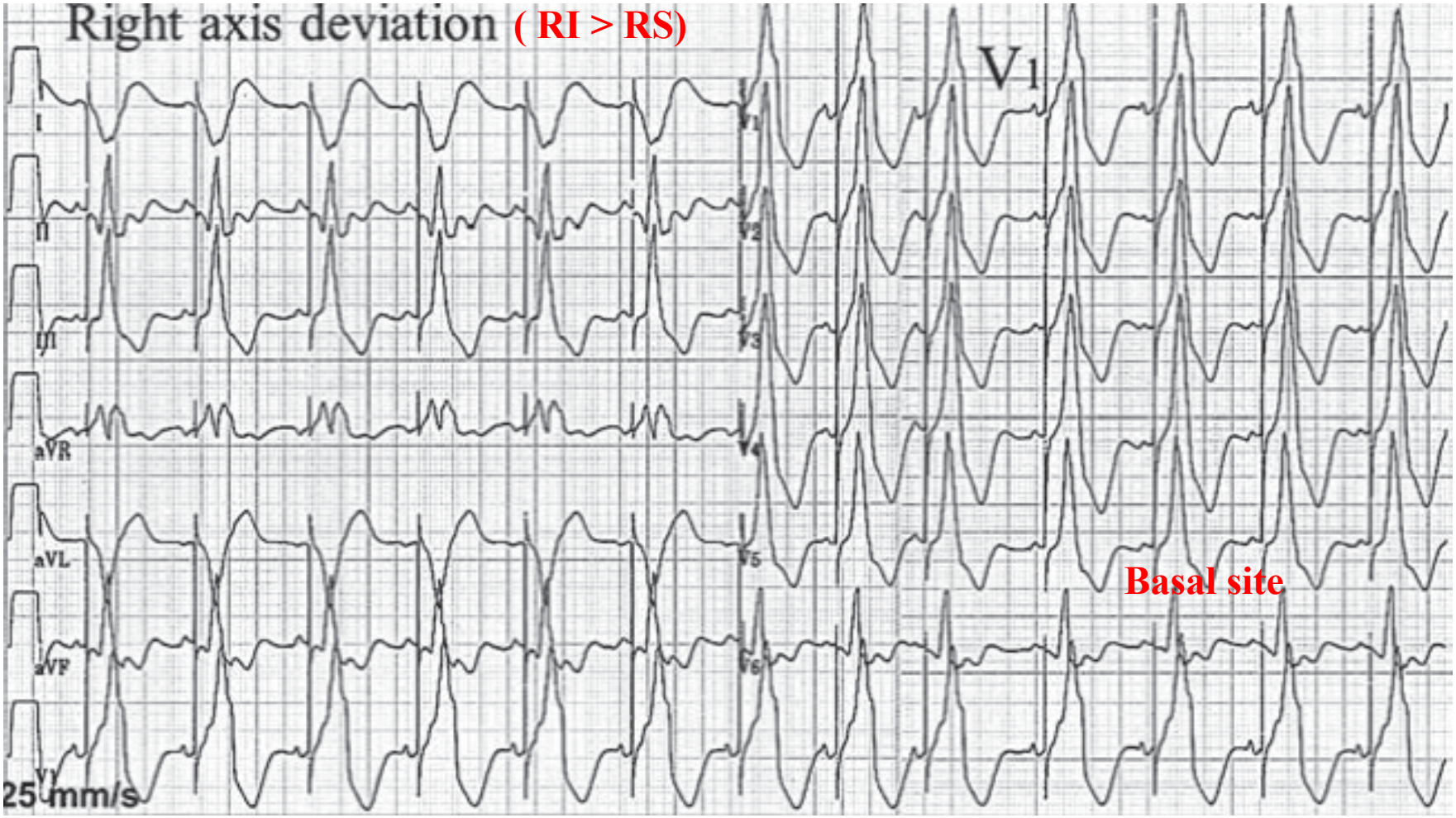
   - native conduction EKG
   - RV pacing EKG
   - LV pacing EKG
   - biventricular pacing EKG
   - RV anodal stimulation EKG

*Cardiol J 2011;18,5:476-486*

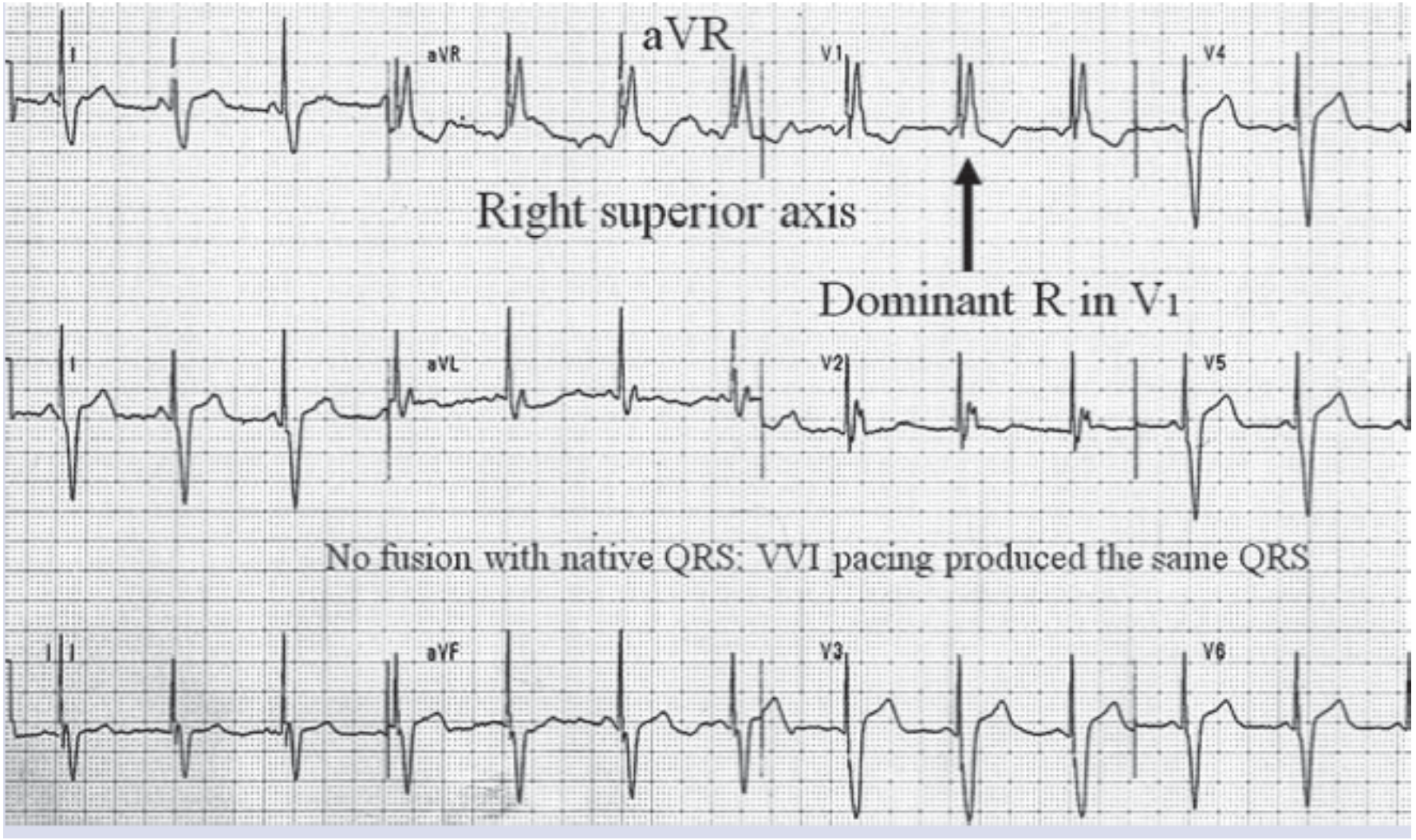
# 12 leads EKG


CRT & RVA pacing
RV apical pacing
Right superior quadrant axis
(–90°)
(–120°)
(–60°)
Left (superior) axis
(–150°) aVR
(–30°) aVL
I, AVL ( frontal )
(180°)
I (0°)
V1, V2( horizontal )
LV pacing
CRT & RVOT pacing
(150°)
(30°)
Normal axis
Right axis
III (120°)
aVF (90°)
II (60°)
RVOT pacing
A. F. Sinnaeve

# LV pacing



# Biventricular pacing(RVA)



# Negative V1(QS) in biventricular pacing (RVA)

- Reflecting RV preponderance in the depolarization process

- Incorrect V1 (too high on the chest)
- Lack of LV capture
- LV lead displacement
- Pronounced latency (true exit block)
- Conduction delay around the LV stimulation site
- Ventricular fusion with intrinsic QRS complex
- Pacing in the middle or anterior cardiac vein
- Severe myocardial disease

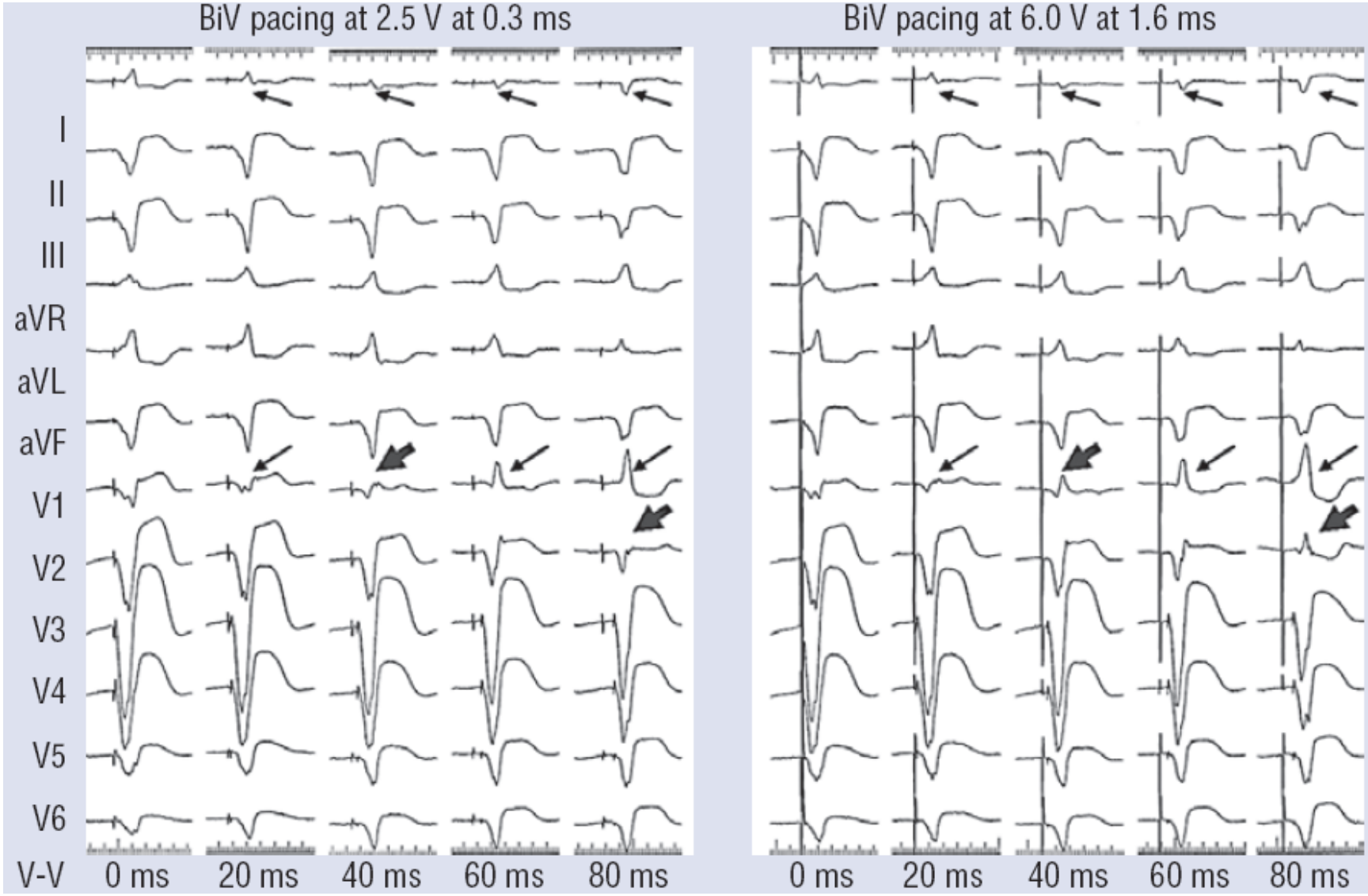
# Nondominant R wave in V1

- Dominant R in V1 ( posterior to anterior activation)
  - contribution of LV stimulation to the overall depolarization

- Programming the VV interval with LV preceding RV → the emergence of dominant R wave
  - r/o LV lead malfunction or misplacement
  - prolonged LV latency(exit delay), LV intramyocardial conduction abnormality near the pacing site

# latency
- interval from pacemaker stimuli to the onset of the earliest paced QRS

# Emergency of R wave in latency via pre-excitation



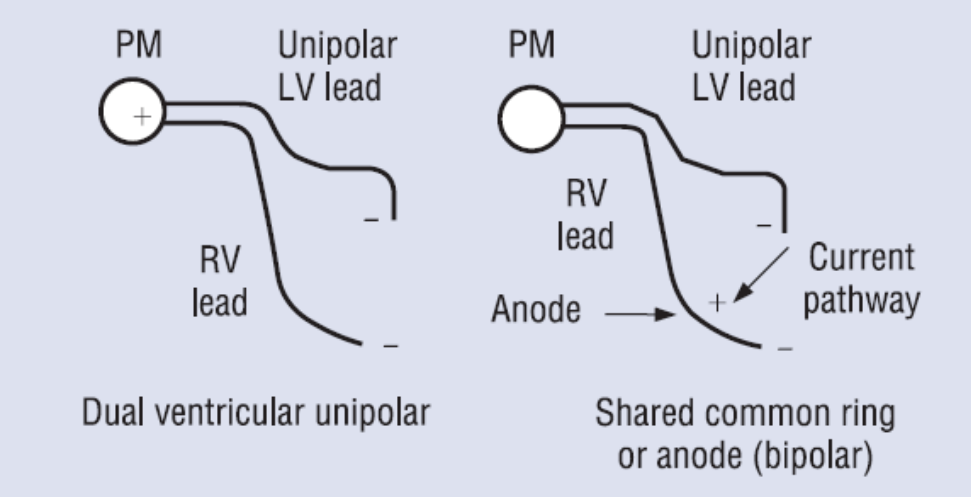
# RV anodal capture

- Unipolar LV lead
- Common anode for both RV and LV pacing
- High LV output during Biventricular pacing

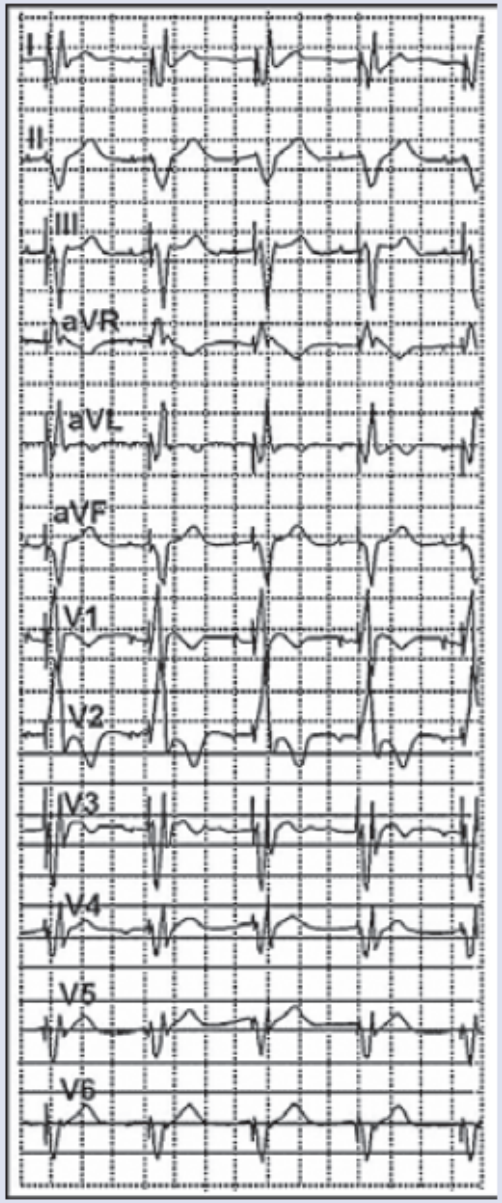
  - slight EKG change
- During LV only pacing

  - EKG looks like that during BiV pacing
- Nullify VV interval
- Must be avoided

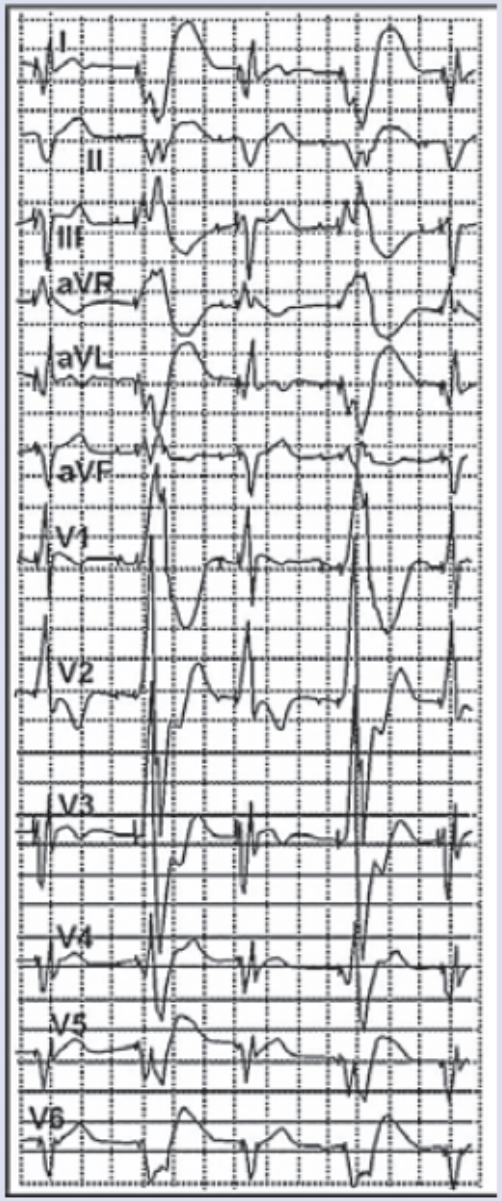
  - prevent early LV stimulation



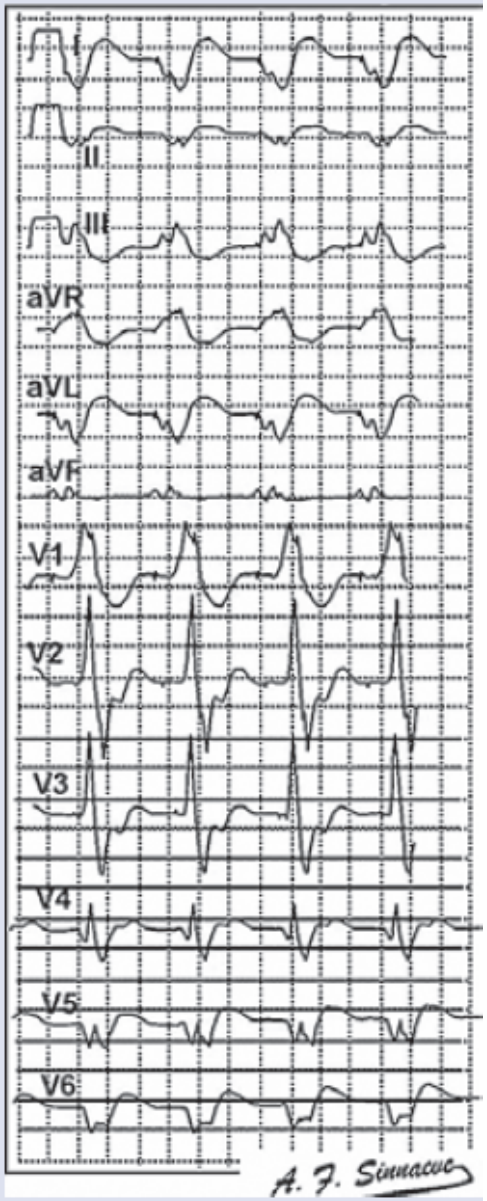
**A** High output monochamber
LV pacing (RV output off)
RV anodal capture
AV = 90 ms



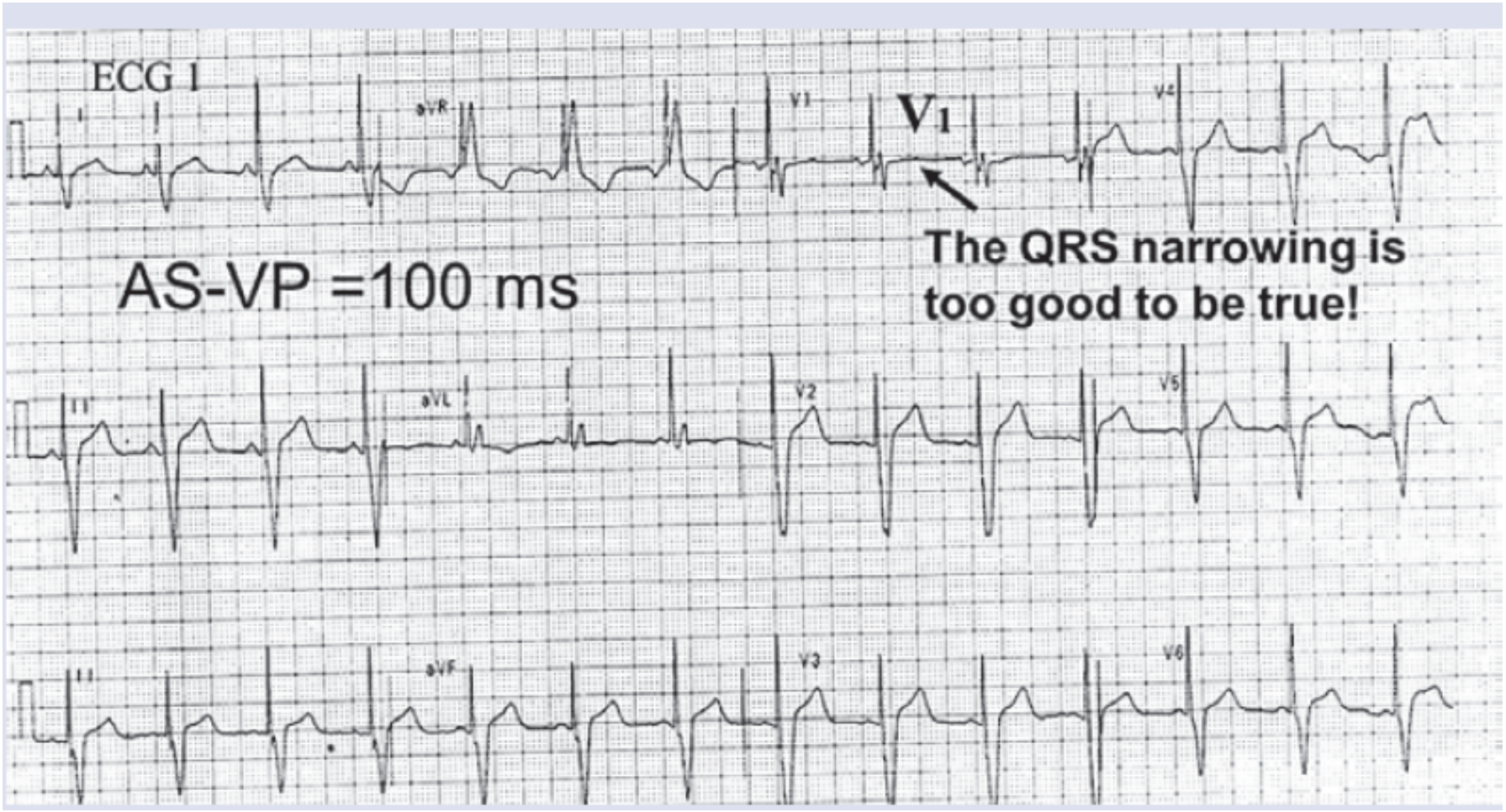

**B** LV output at 3.5 V
(RV output off)
2:1 anodal RV stimulation
intermittent RV capture




**C** LV output at 2.8 V
(RV output off)
Pure LV pacing
LV stimulation only



# Ventricular fusion with native conduction




**QRS morphology during progressive shortening of AV interval**

# Ventricular fusion
# Clinically beneficial or harmful ?

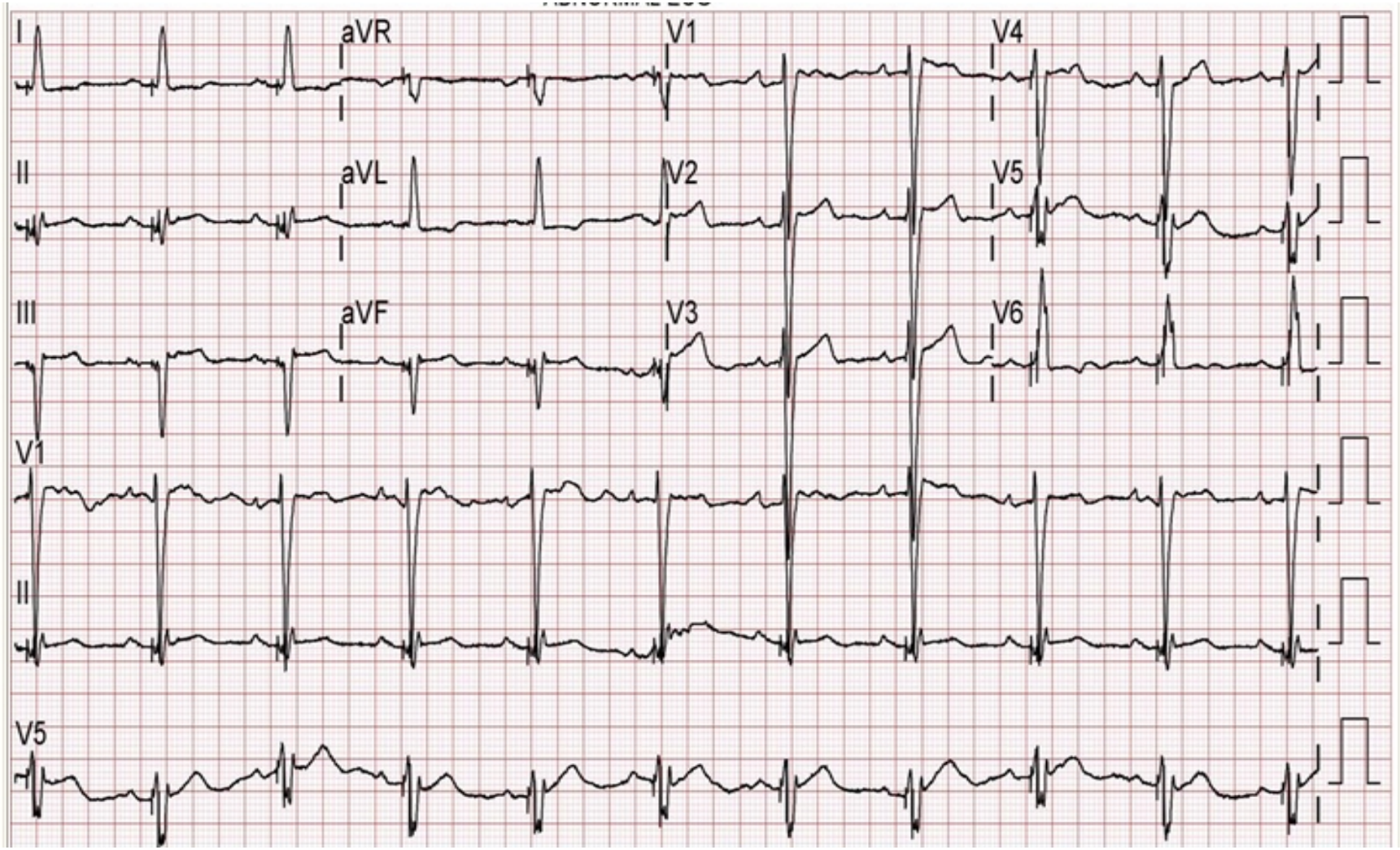
- Presumed variability of AV conduction
  Lack of data about the chronic effect of fusion

- At present, optimize AV delay regardless of fusion

- If suboptimal CRT response, reprogramming AV delay to avoid ventricular fusion

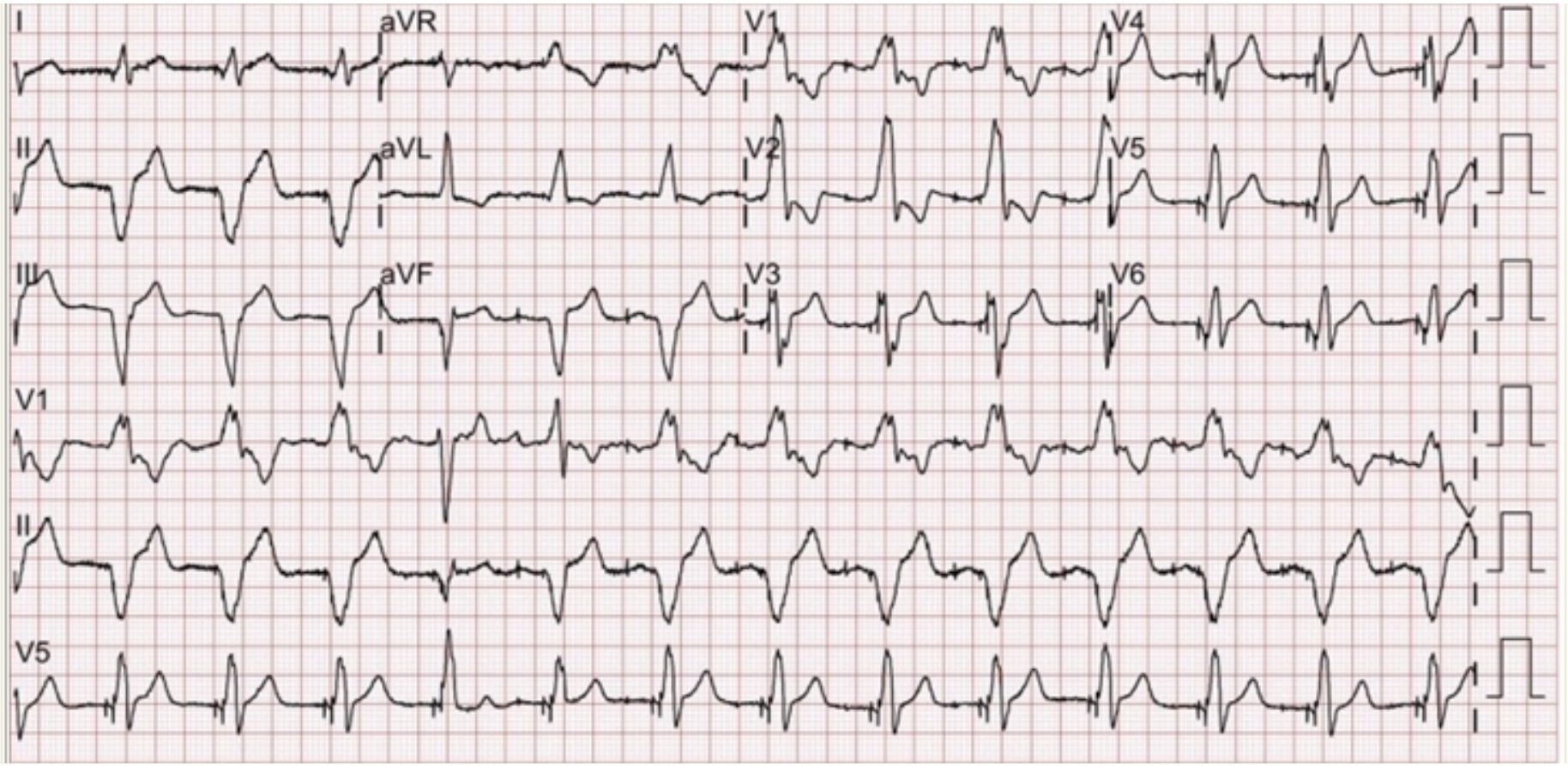
# Just after CRT

**Biv, 3.5V @ 0.5ms(160/170 ms), LV→RV 50**

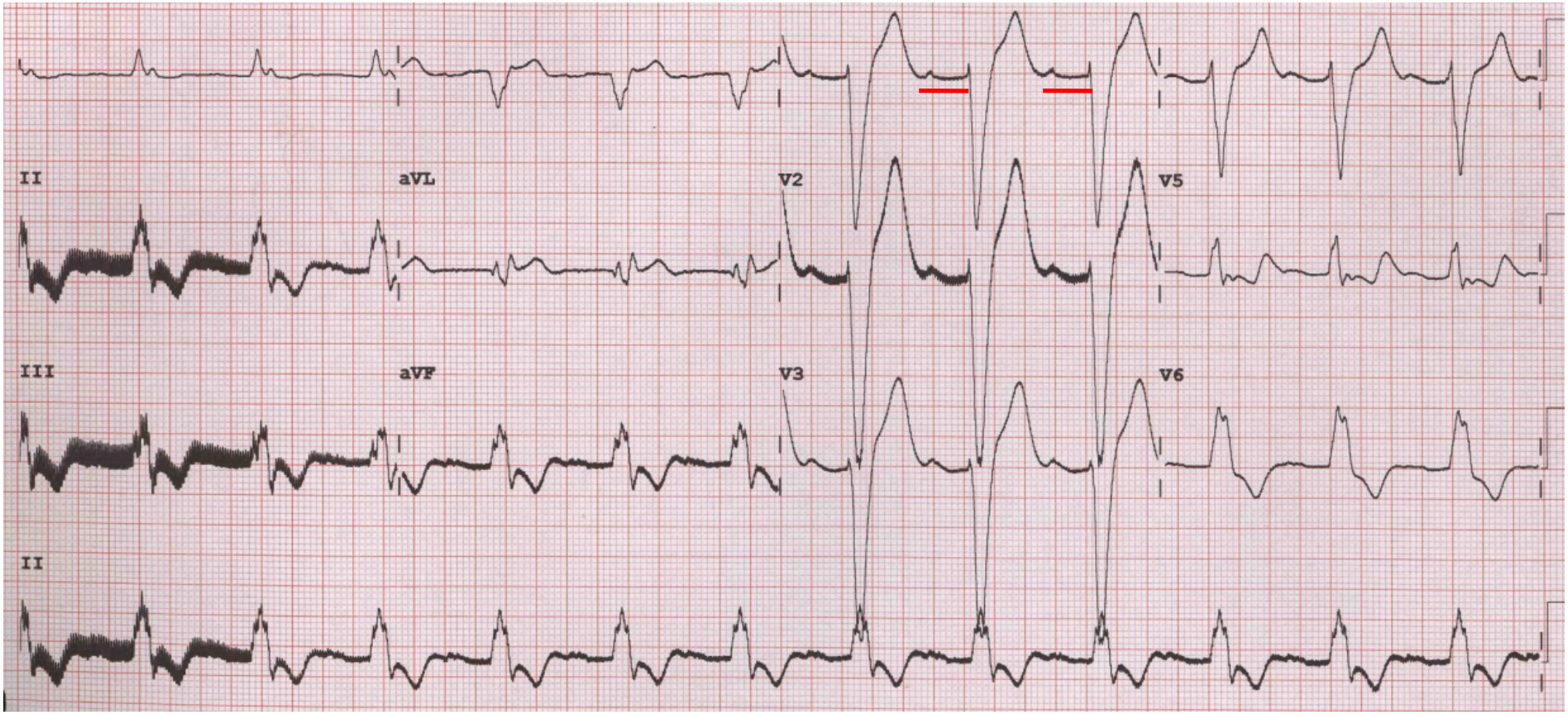


# 20 month after CRT

**Biv, 3.5V @ 0.5ms(160/170 ms), LV→RV 50**

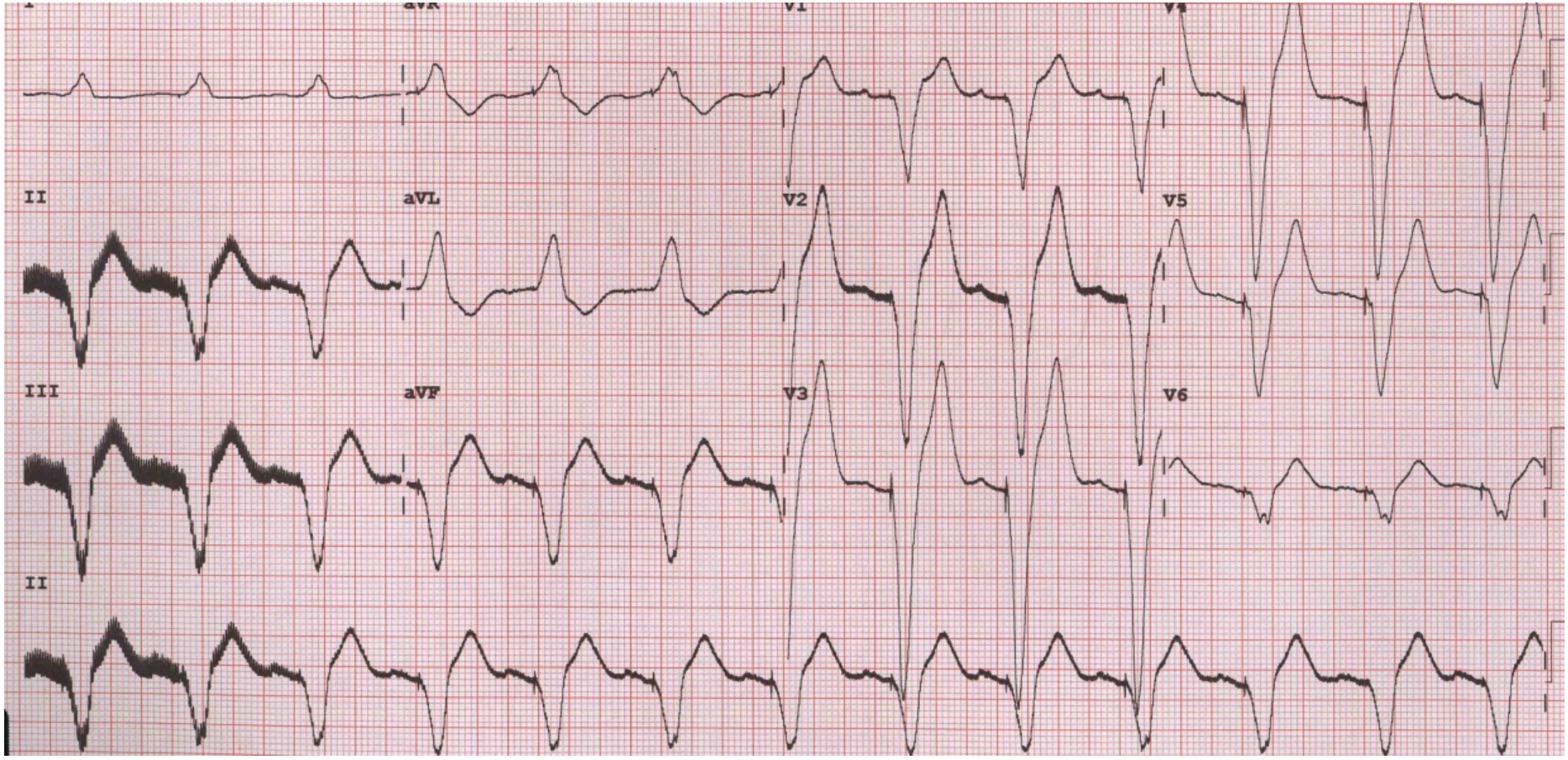


# Pacing off



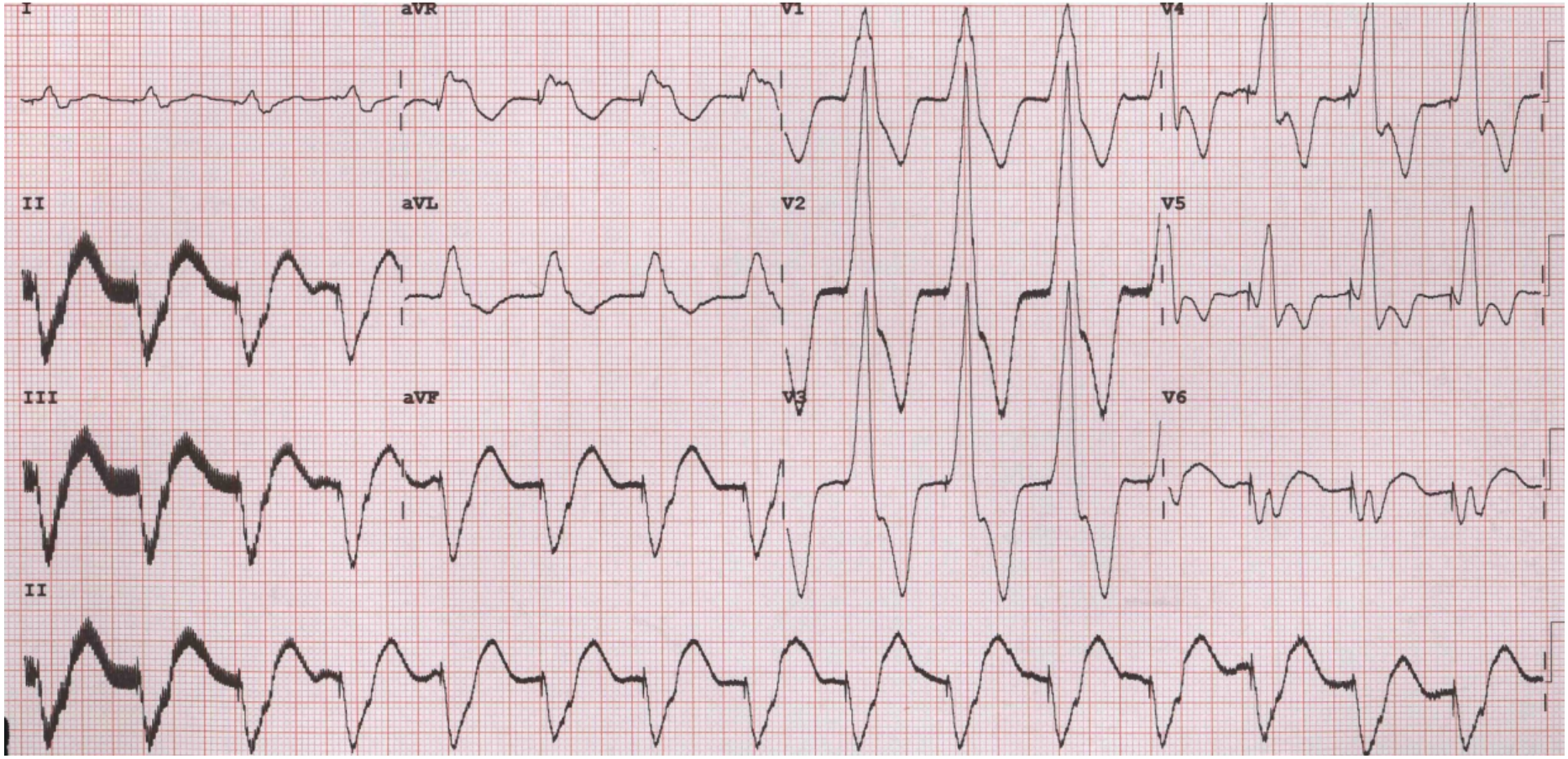
# RV pacing

**3.5 v @0.5ms ( 130/180 msec)**



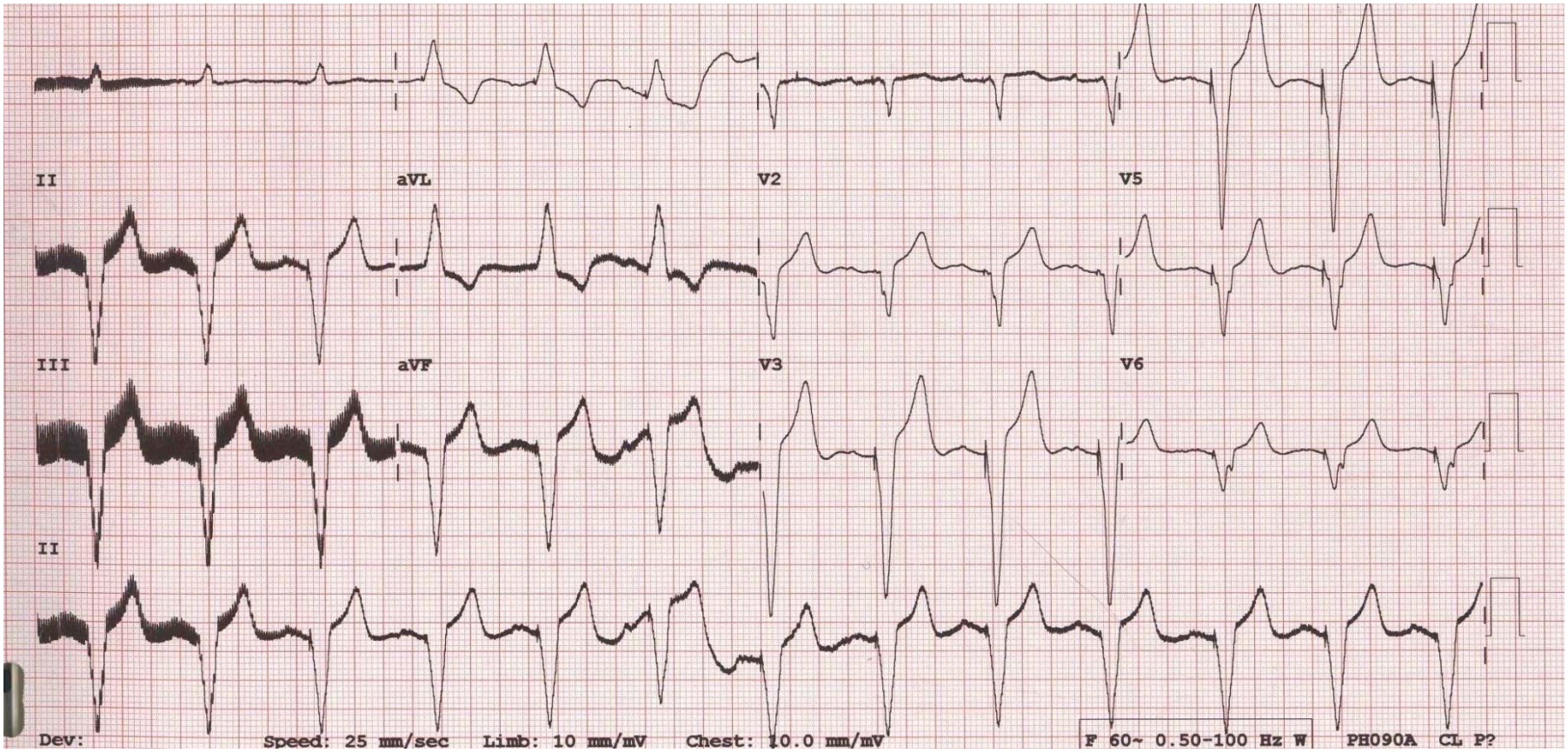
# LV pacing

**3.5 v @0.5ms**



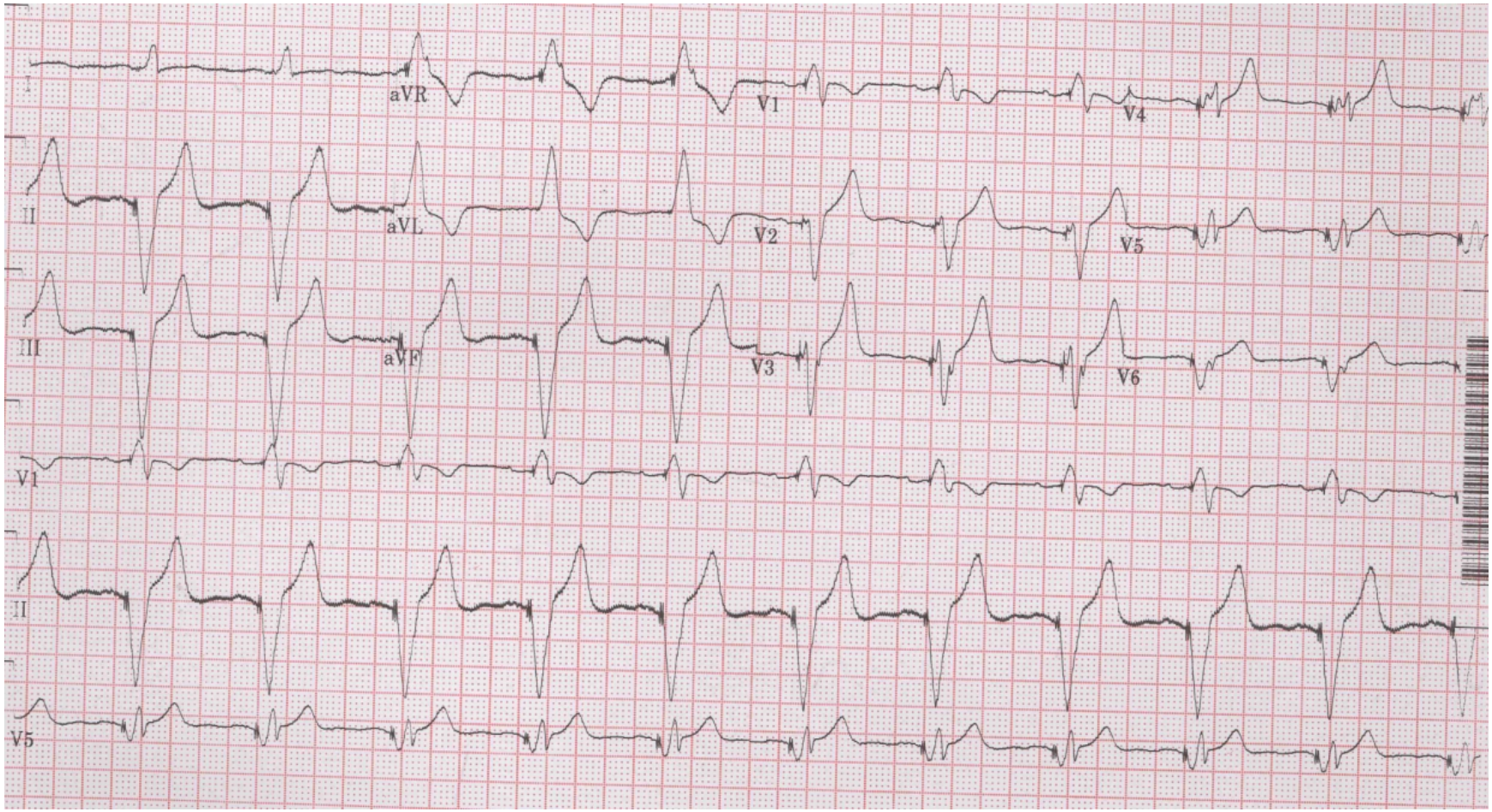
# Biventricular pacing

**3 v @0.5ms(130/180 ms), LV→RV,  0ms**



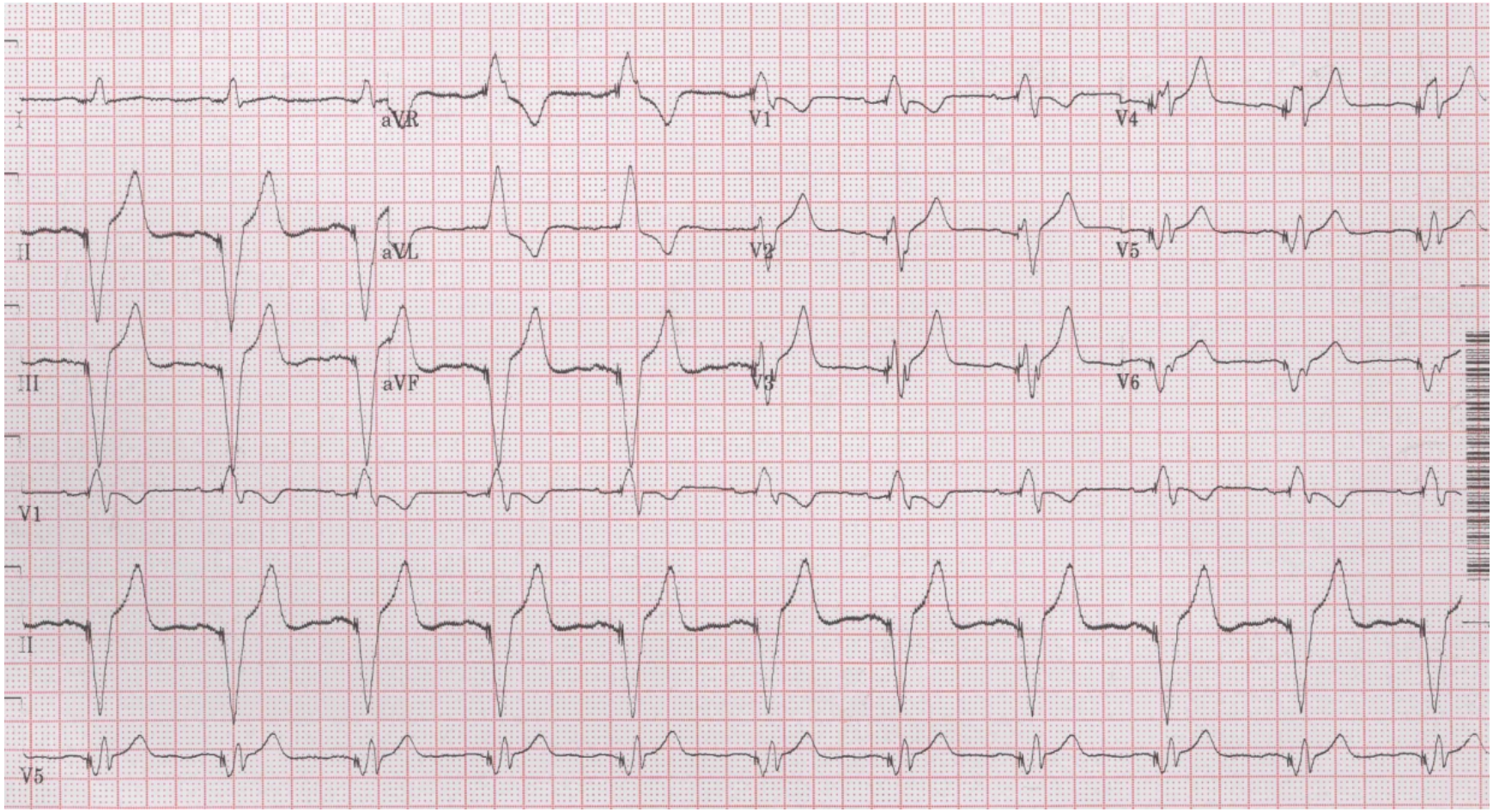
# Biventricular pacing

**3.5 v @0.5ms(130/180 ms), LV→RV,  15 ms**



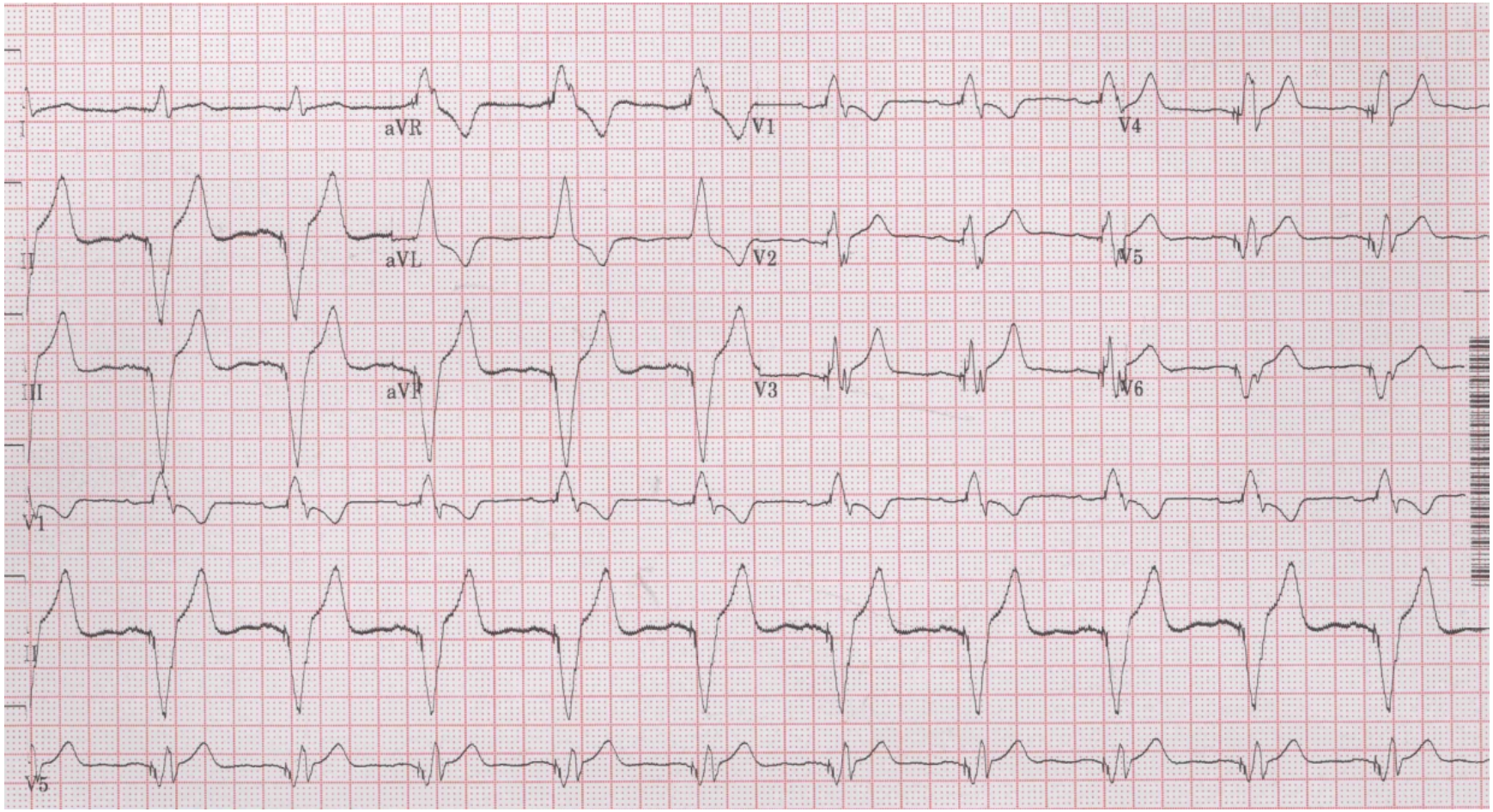
# Biventricular pacing

**3.5 v @0.5ms(130/180 ms), LV→RV,  20 ms**



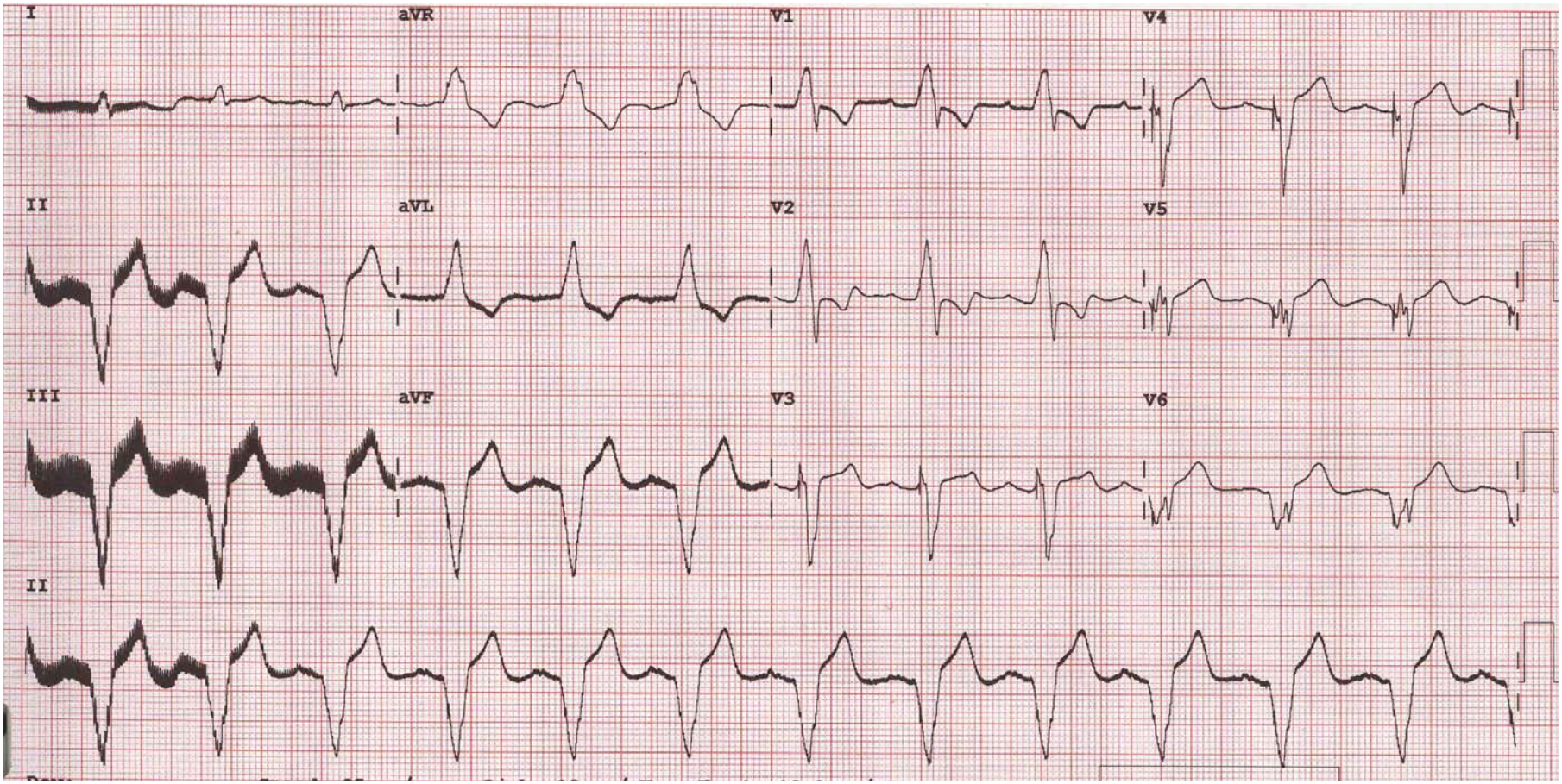
# Biventricular pacing

**3.5 v @0.5ms(130/180 ms), LV→RV,  30 ms**



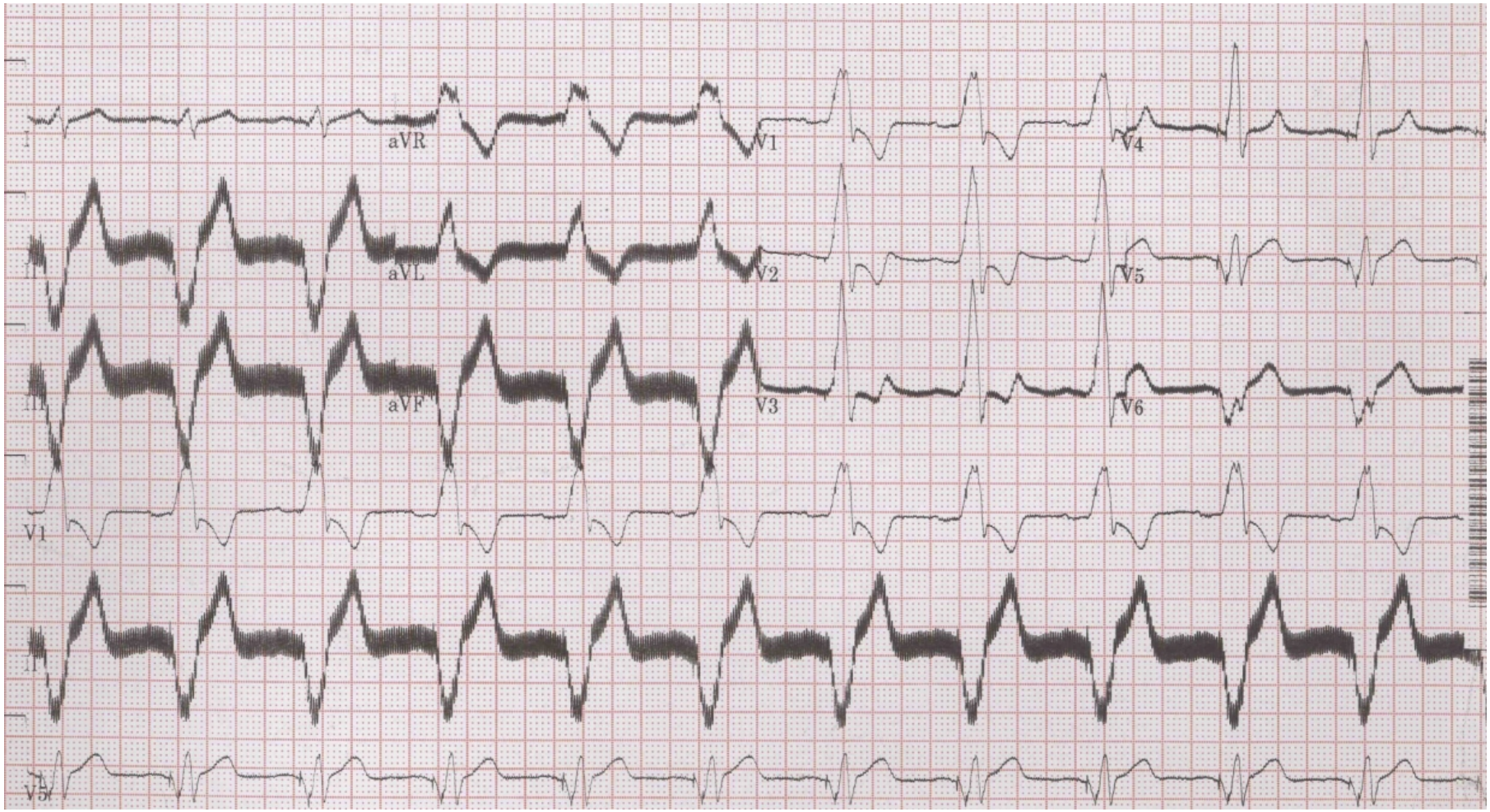
# Biventricular pacing

**3 v @0.5ms(130/180 ms), LV→RV,  50 ms**



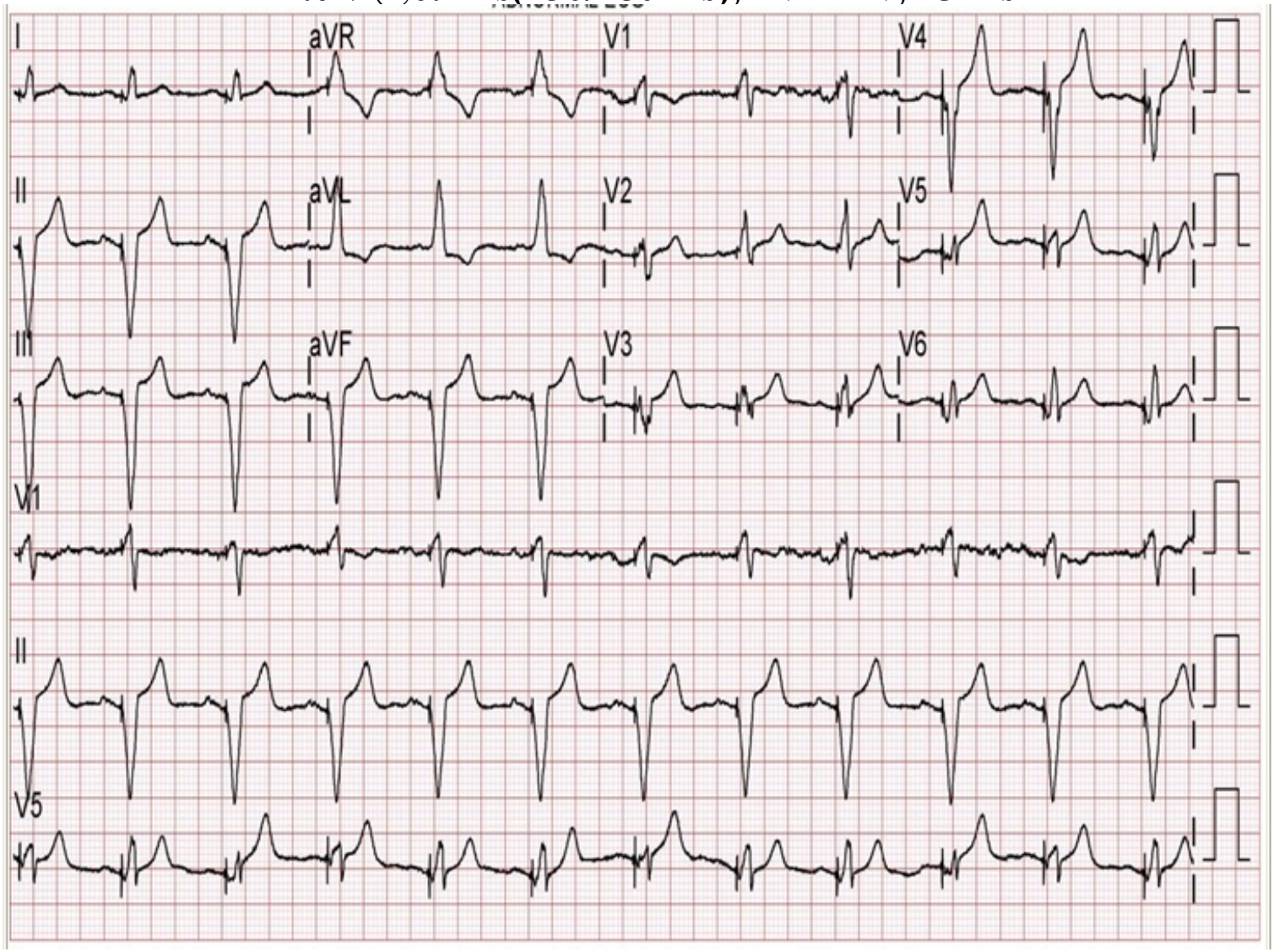
# Biventricular pacing

**3.5 v @0.5ms(130/180 ms), LV→RV,  60 ms**



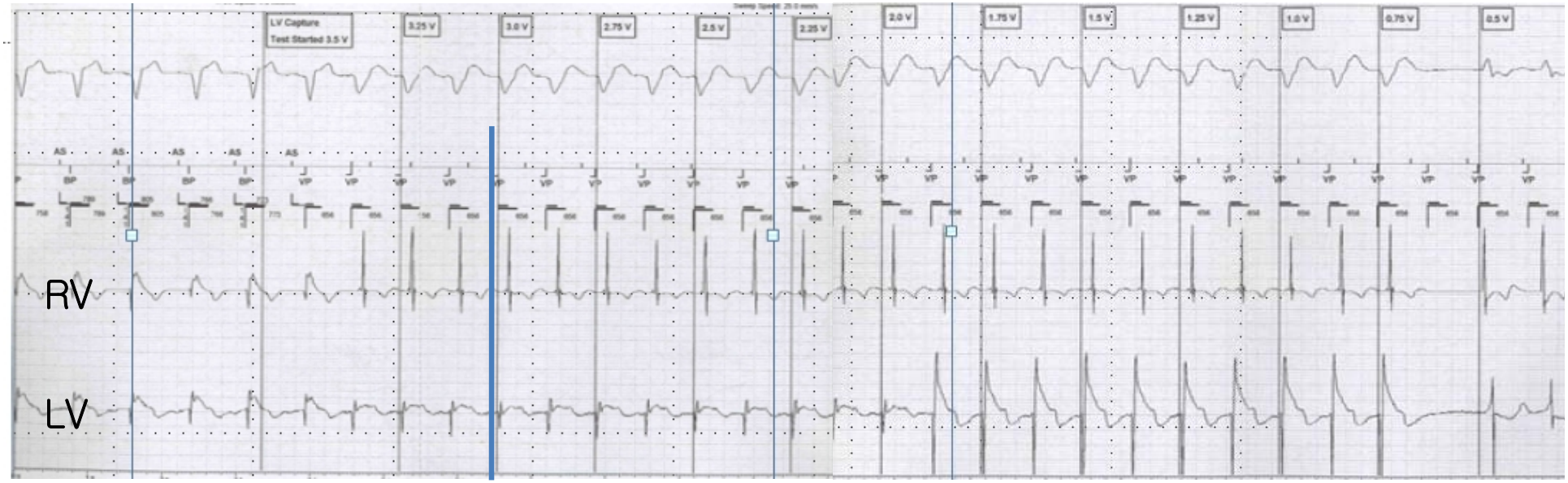
# Biventricular pacing

**2.0 v @0.2ms(130/180 ms), LV→RV, 15 ms**



# Why, not anodal pacing

- EKG change with V-V timing
- There is a distance between  RV and LV IEGM
- LV bipolar configuration



# Ventricular fusion

- Most common cause of different EKG
- Just change AV delay( 50 ms)
- Look at 12 leads EKG

# LV latency

- Can be treated with V-V programming
- Advancing LV stimulation

# Summary

- Effective ,important Tx strategy in selected patients
  - Dilated CM; EF ≤ 35%
  - NYHA functional class III/IV despite optimal medication
  - NSR, prolonged QRS complex

- No difference in second endpoints despite any AV optimization methods at FREEDOM trial, SMART-AV trial

- Routinely tweaking the parameter is ???
  - Check Echo after 3 month
  - Check EKG in intrinsic QRS morphology, RV pacing, LV pacing, Biv pacing and PV/AV delay and V-V timing