11th ANNUAL HEART FAILURE UPDATE 2024

Friday May 24 - Saturday May 25

Marriott Chateau Champlain, Montreal, Quebec

CHFS SCIC

Canadian Heart Failure Society
Société canadienne d'insuffisance cardiaque

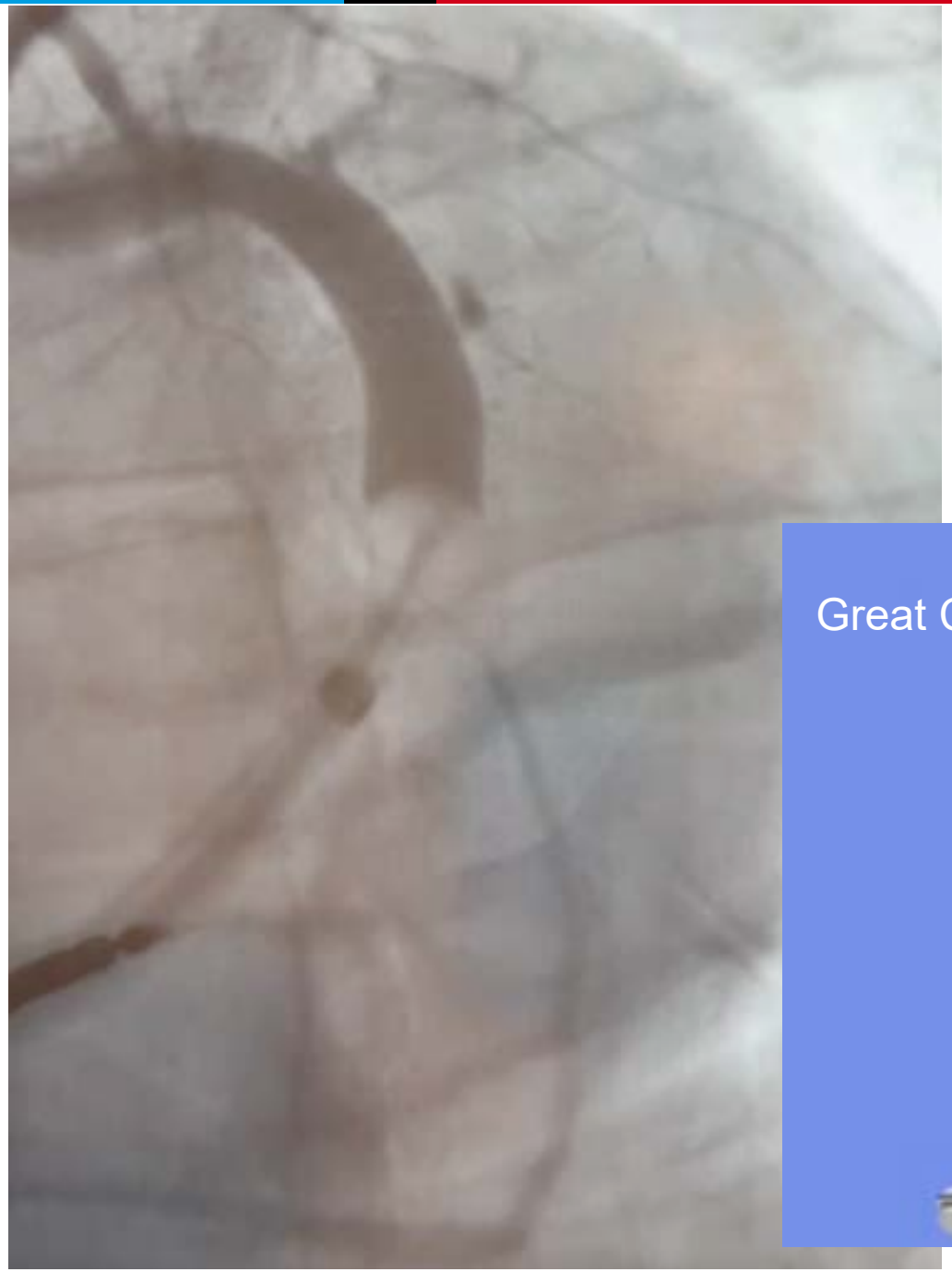
X @CanHFSociety #HFupdate

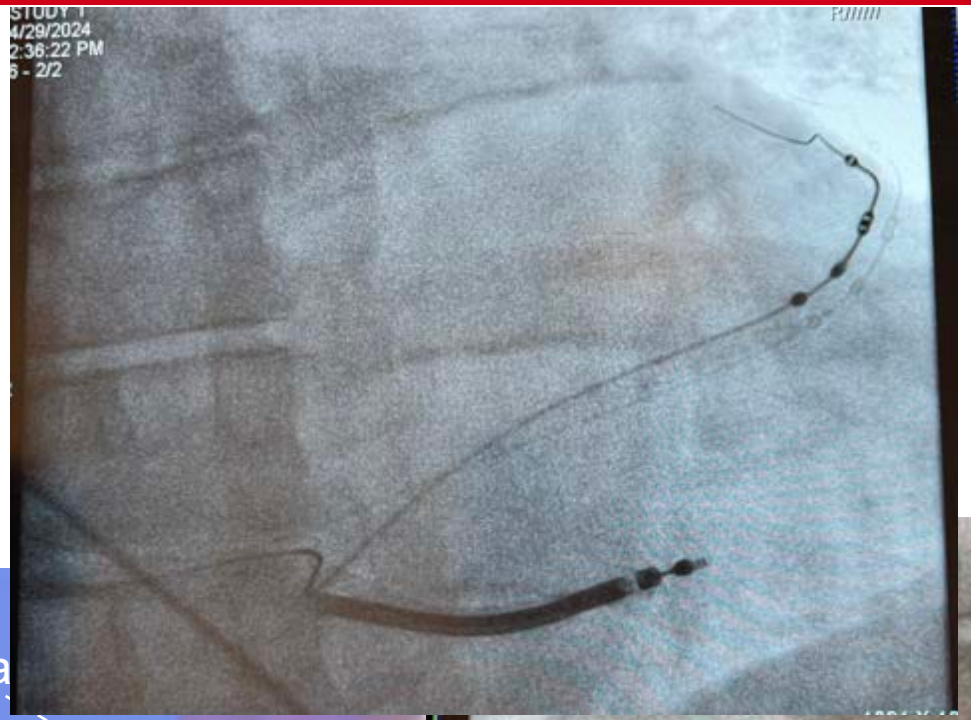
Optimisation des appareils de resynchronisation cardiaque

Dre Jacqueline Joza

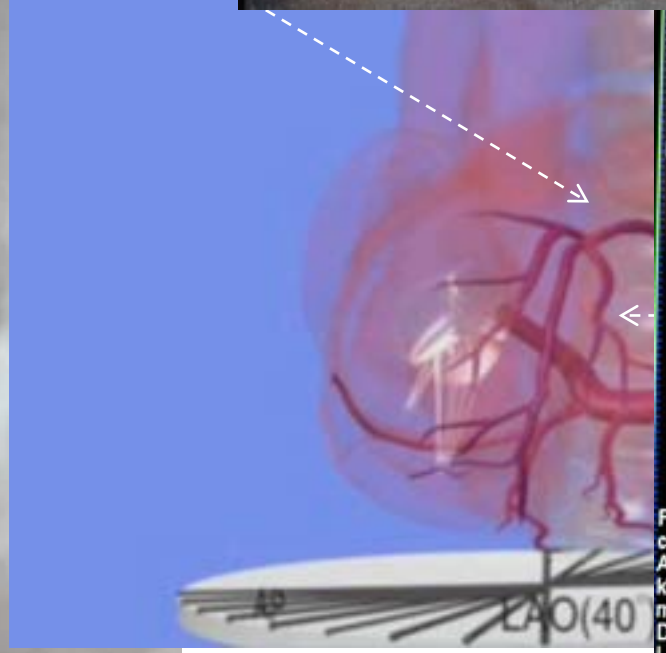
Disclosures

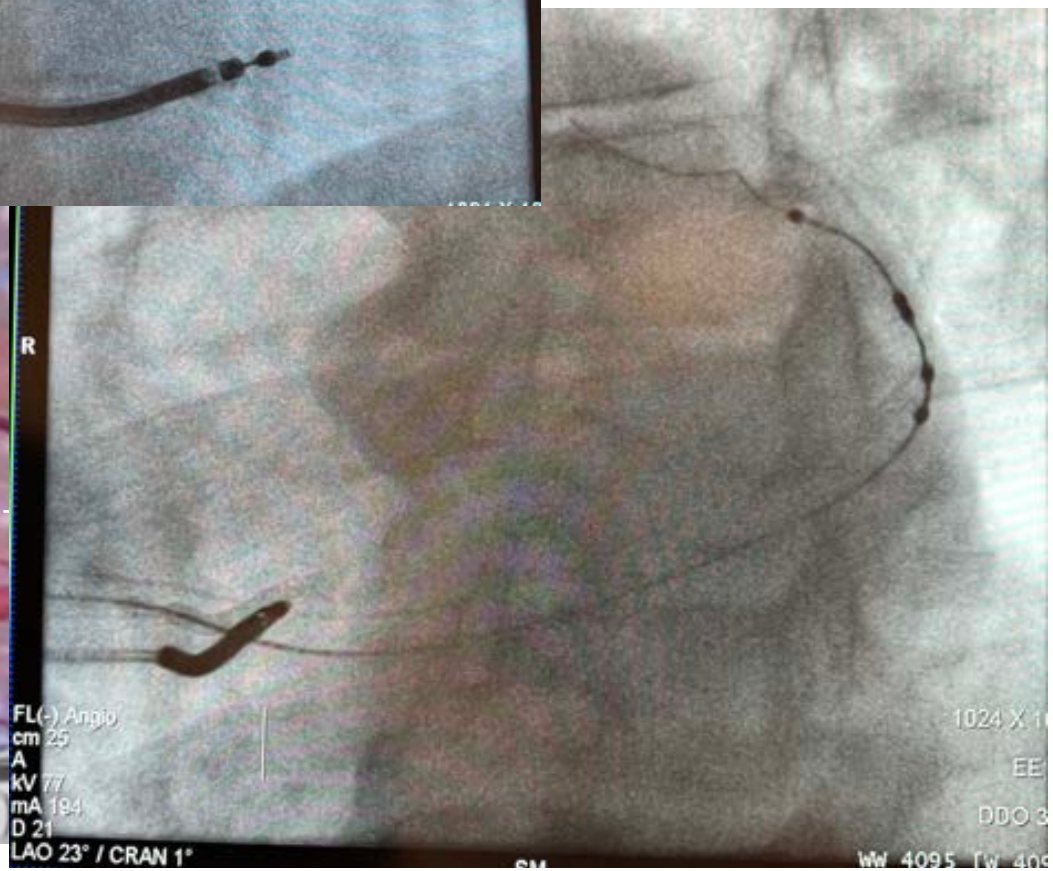
- External research grant, investigator-initiated (Medtronic)
- Advisor: Boston Scientific, Abbott

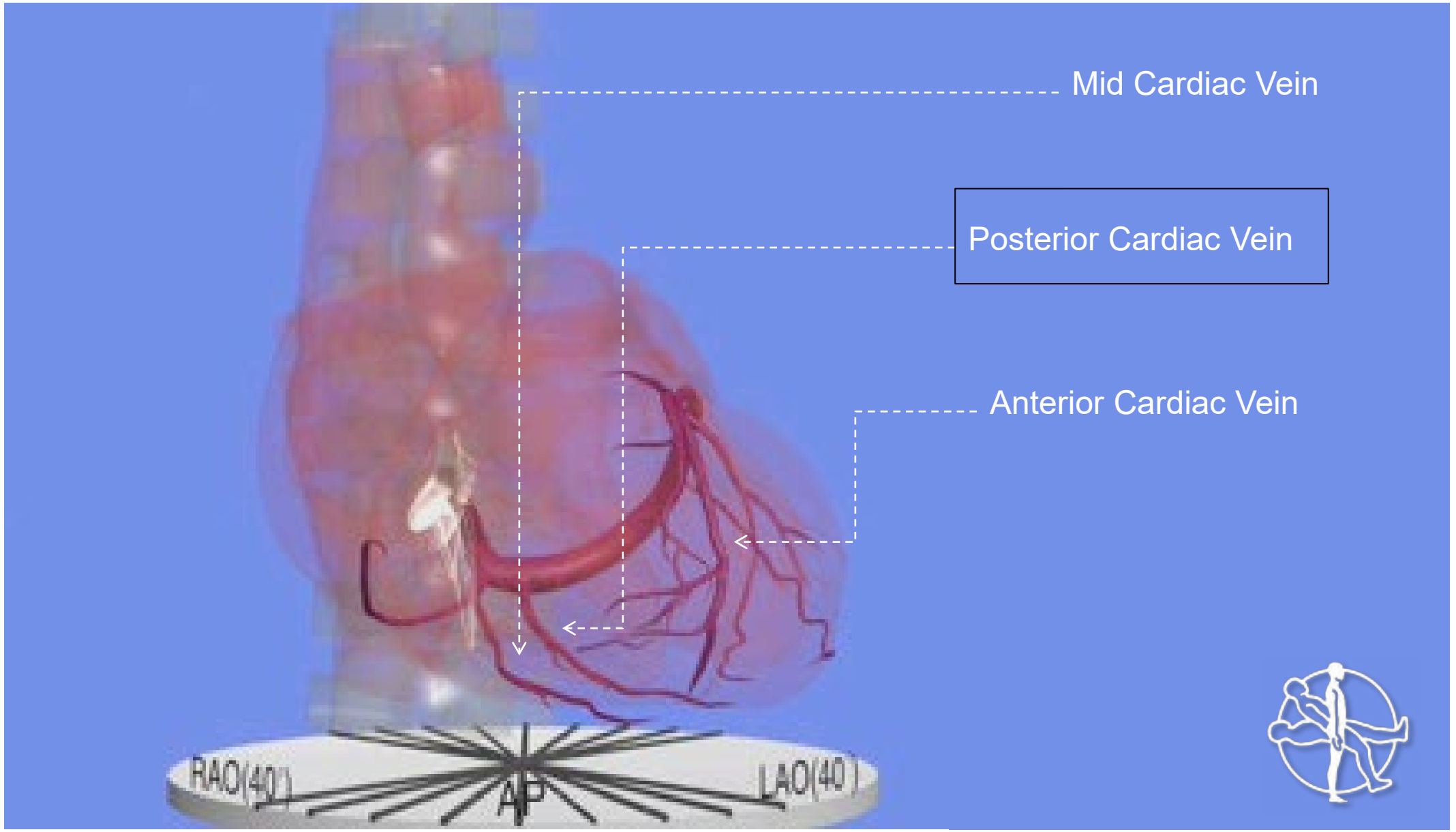



4/29/2024
2:36:22 PM
5 - 2/2

Great Ca

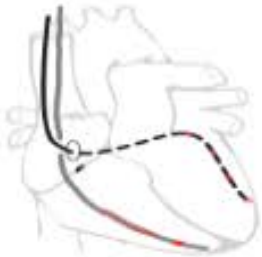

LAO(40


R
FL(-) Angio
cm 25
A
kV 77
mA 194
D 21
LAO 23° / CRAN 1°


Mid Cardiac Vein
Posterior Cardiac Vein
Anterior Cardiac Vein
RAO(40)
AP
LAO(40)

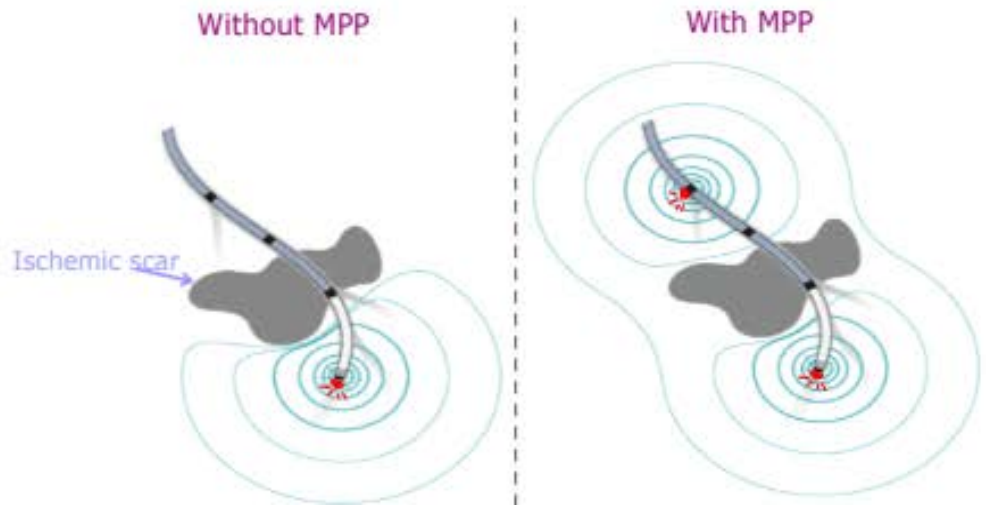
Advantages of MultiPole Pacing?

Enlarge the area of capture by pacing from two sites in the LV within te same CS vein

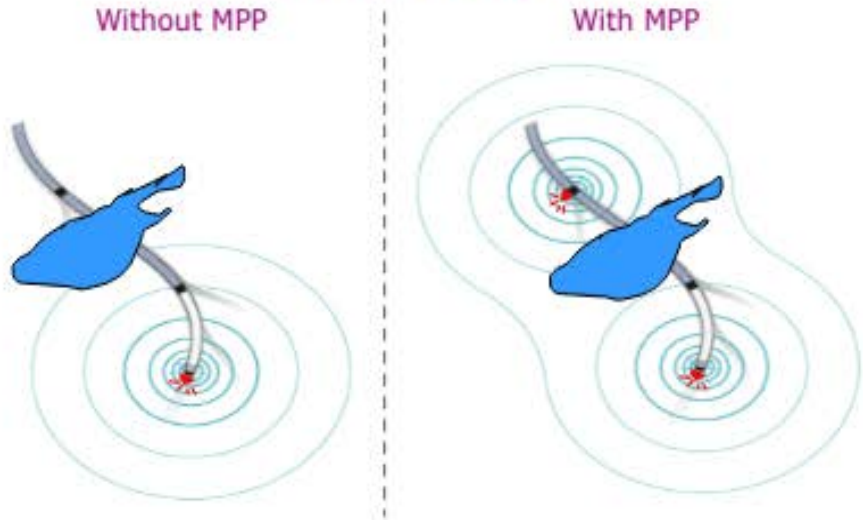


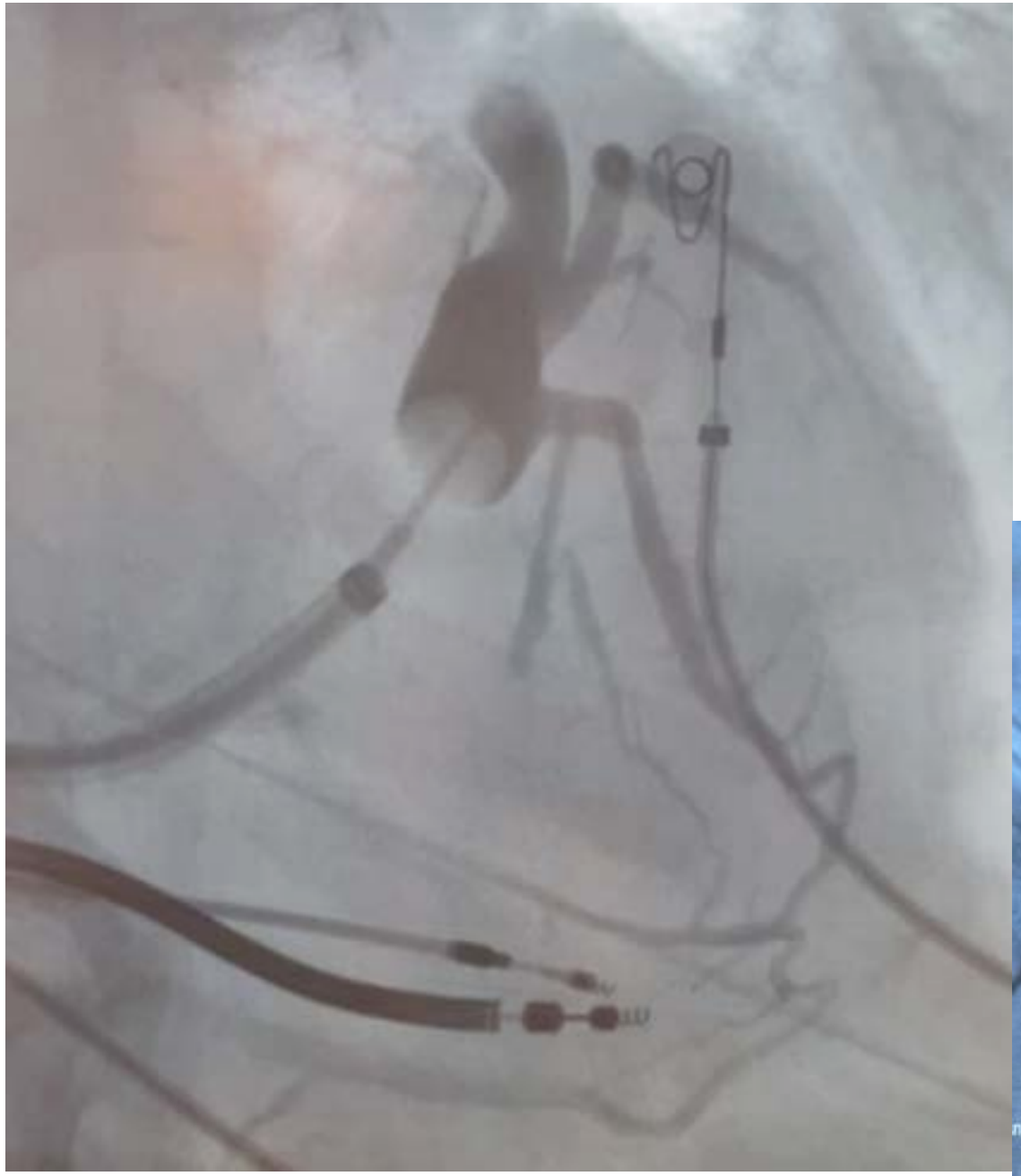
- **Depolarize the left ventricle faster**

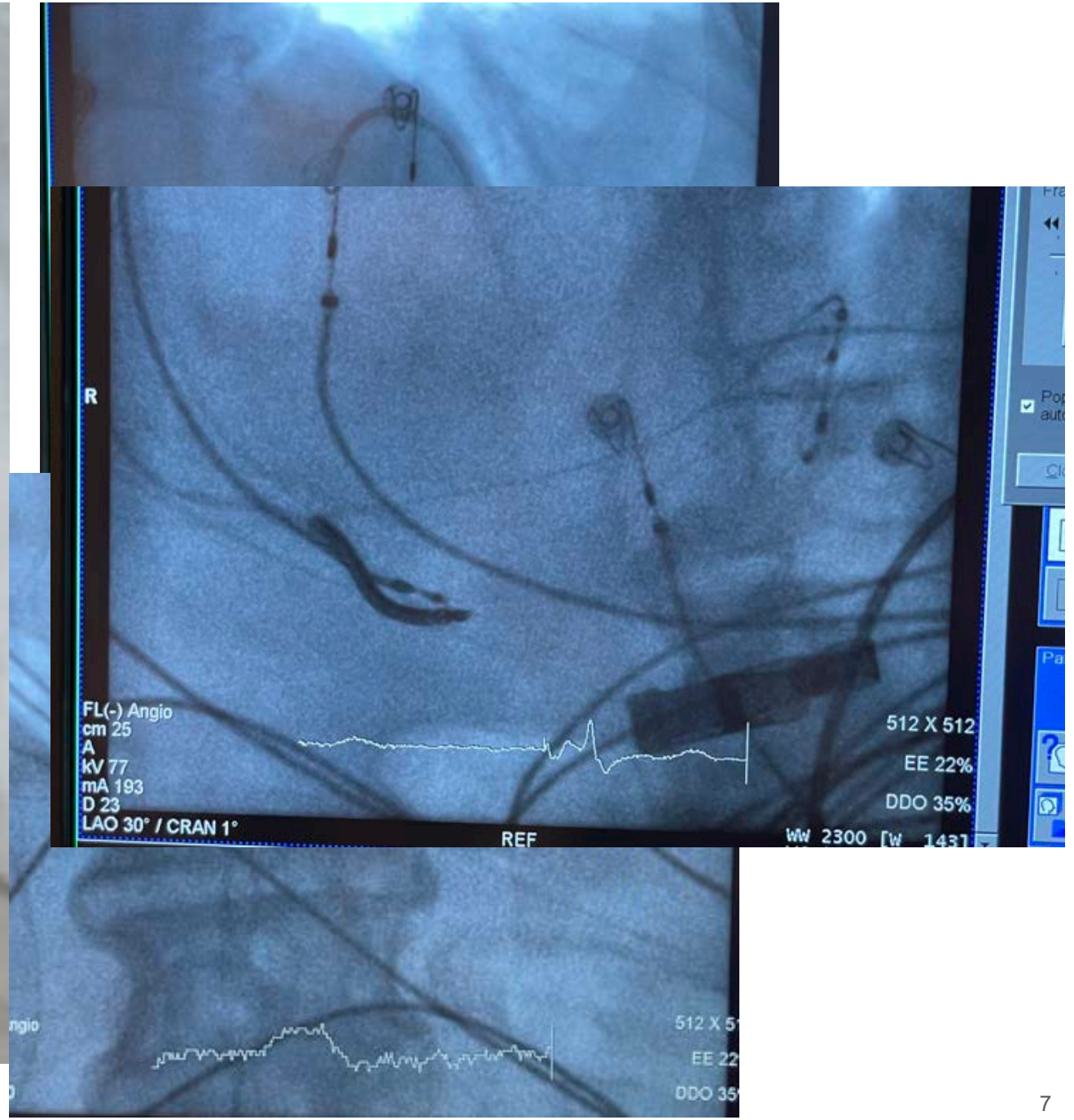
Ischemic CM



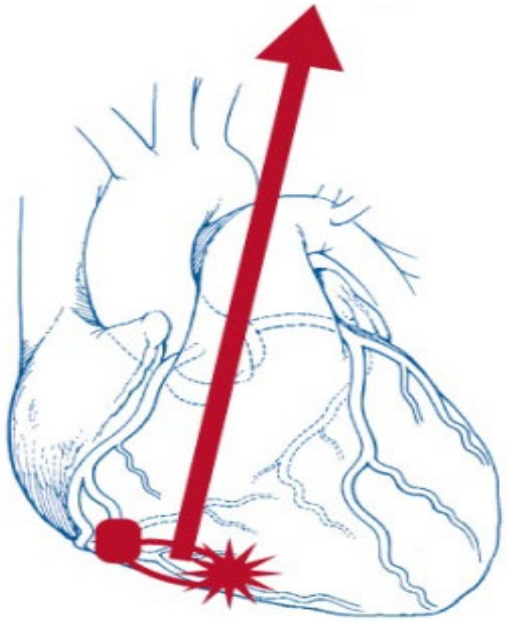
Non-ischemic CM



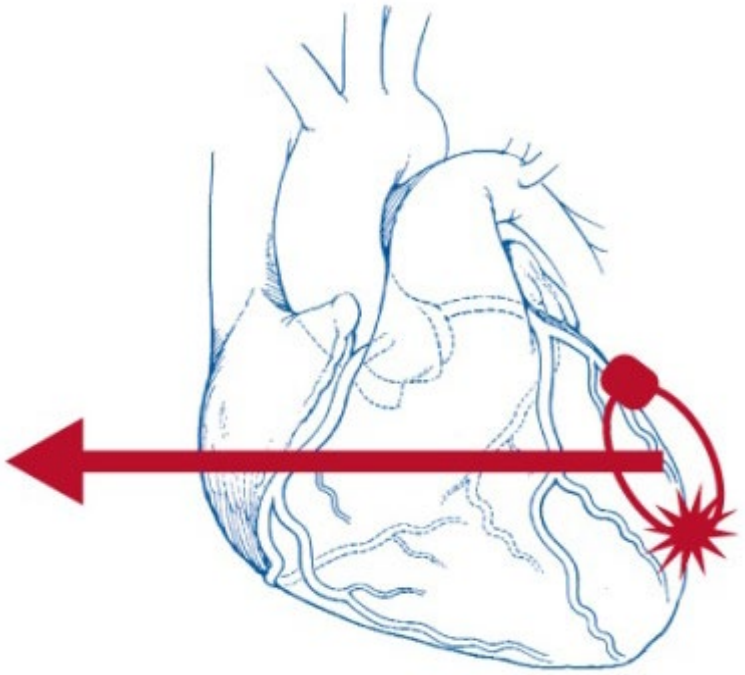



R
FL(-) Angio
cm 25
A
kV 77
mA 193
D 23
LAO 30° / CRAN 1°
REF
512 X 512
EE 22%
DDO 35%

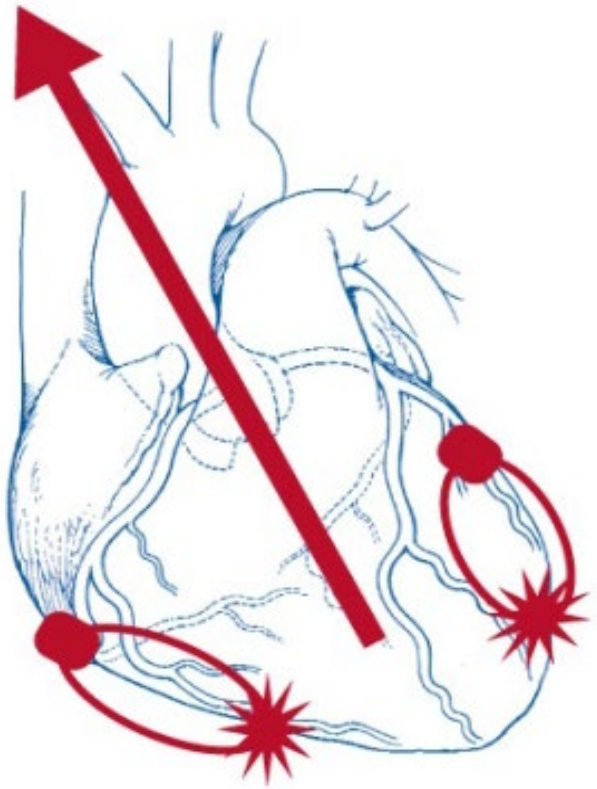
RV pacing

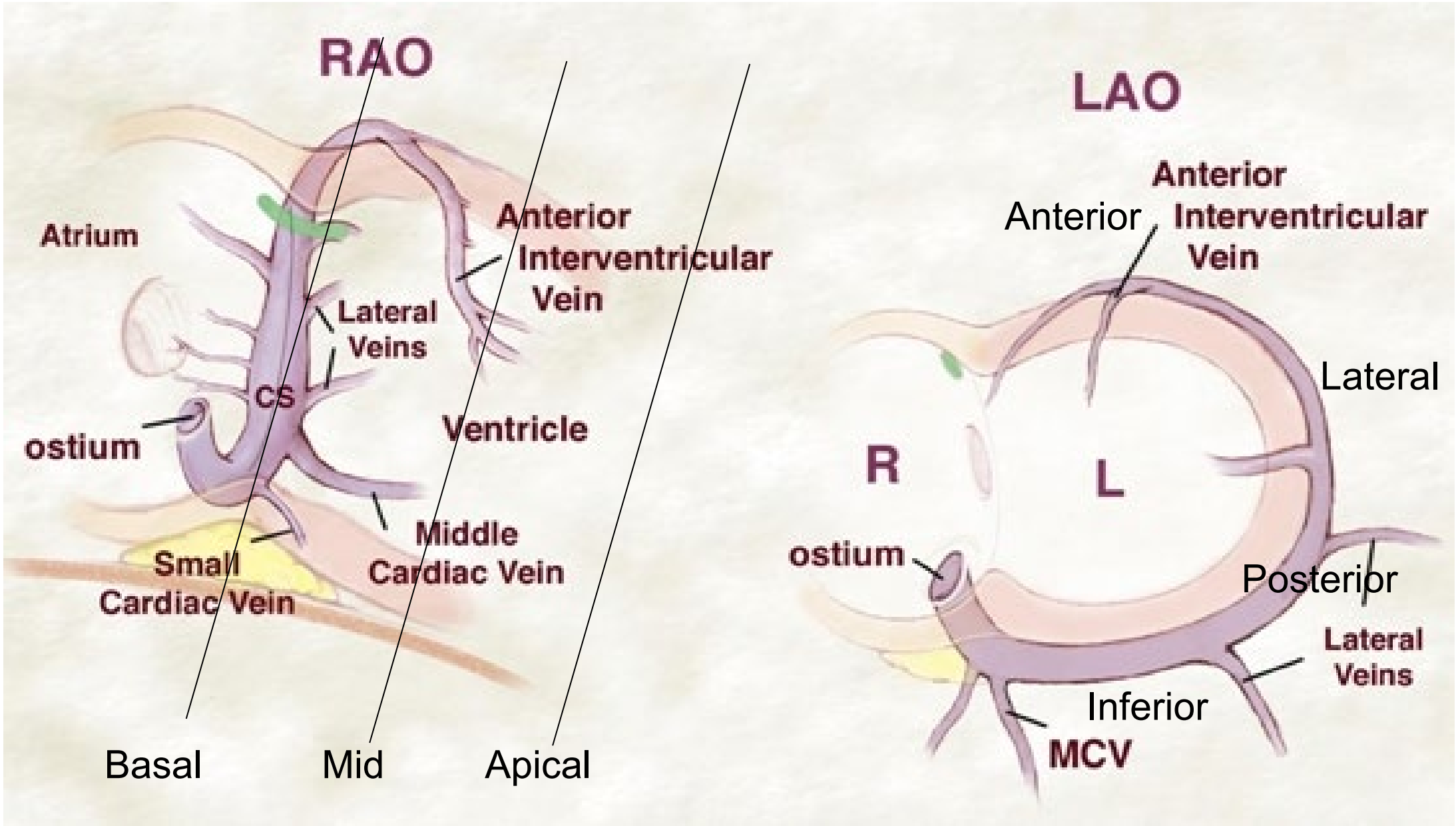


LV pacing

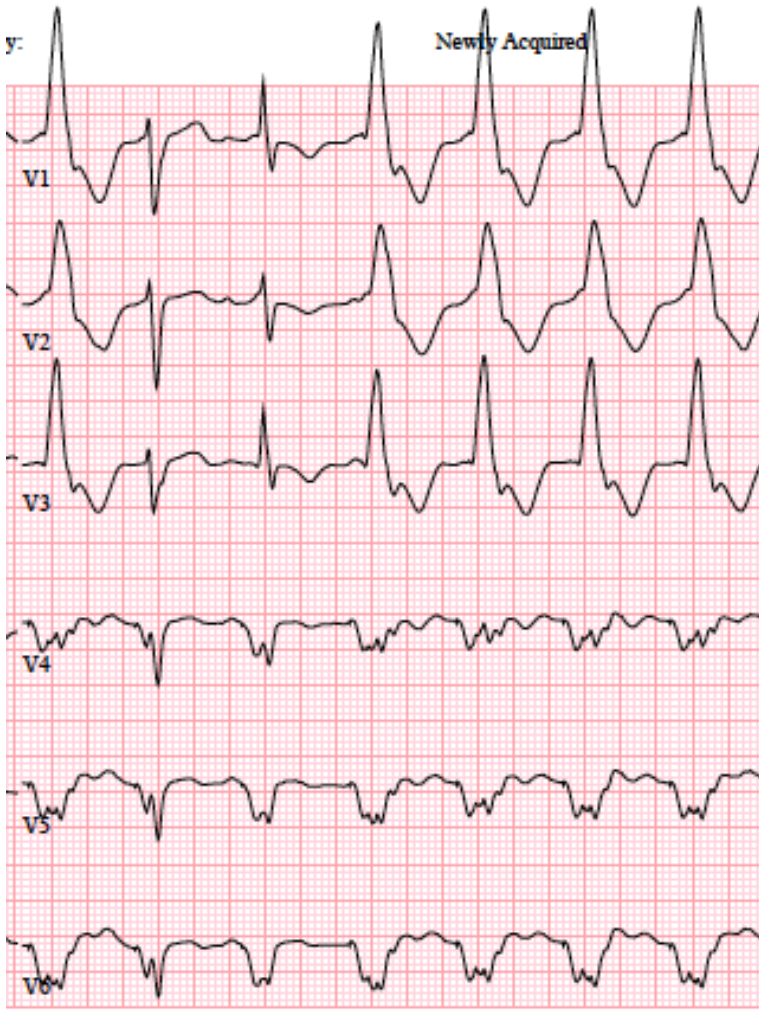


BiV pacing



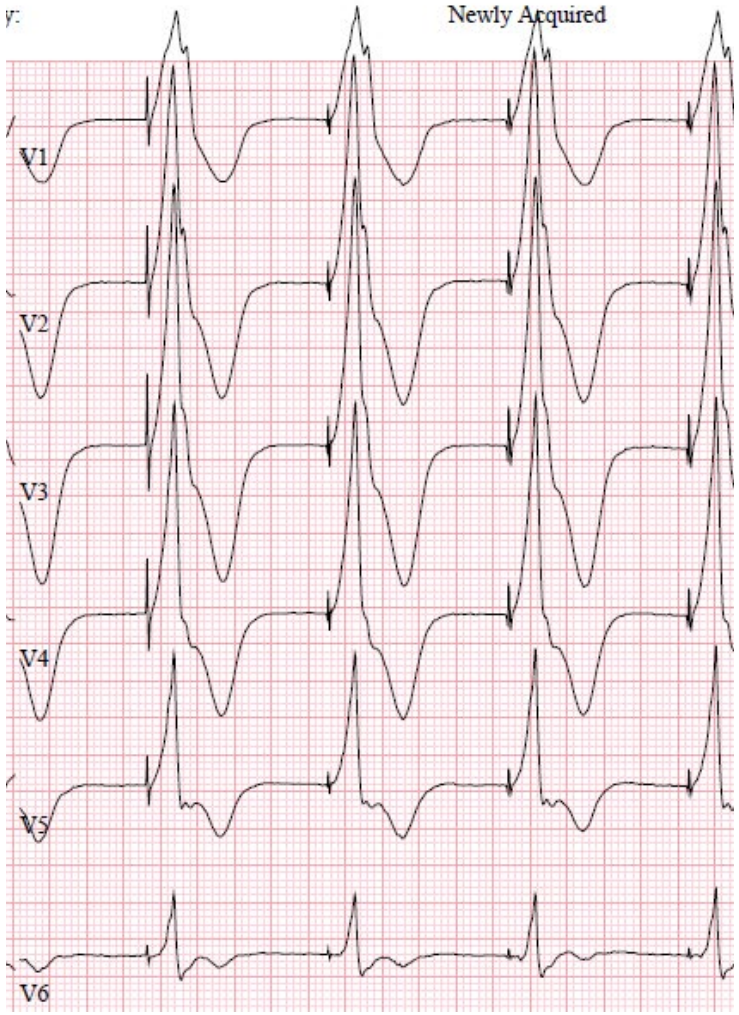

RAO
Atrium
Anterior
Interventricular
Vein
Lateral
Veins
CS
ostium
Ventricle
Middle
Cardiac Vein
Small
Cardiac Vein
Basal
Mid
Apical
LAO
Anterior
Anterior
Interventricular
Vein
Lateral
R
L
ostium
Posterior
Lateral
Veins
Inferior
MCV

Apical LV pacing



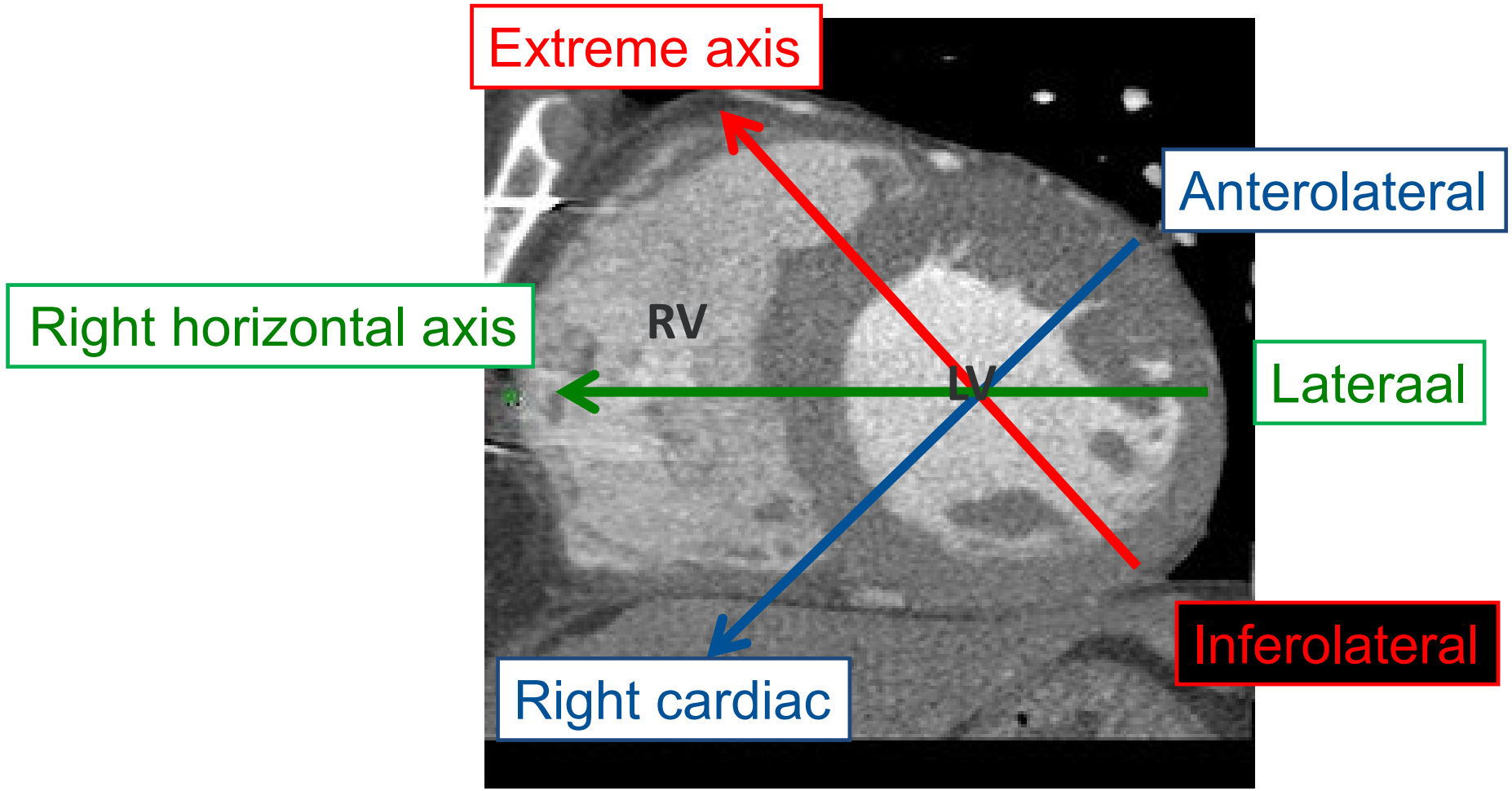
V4-6 negative: apical

Basal LV pacing



V4-6 positive: basal

Lead position relative to axis



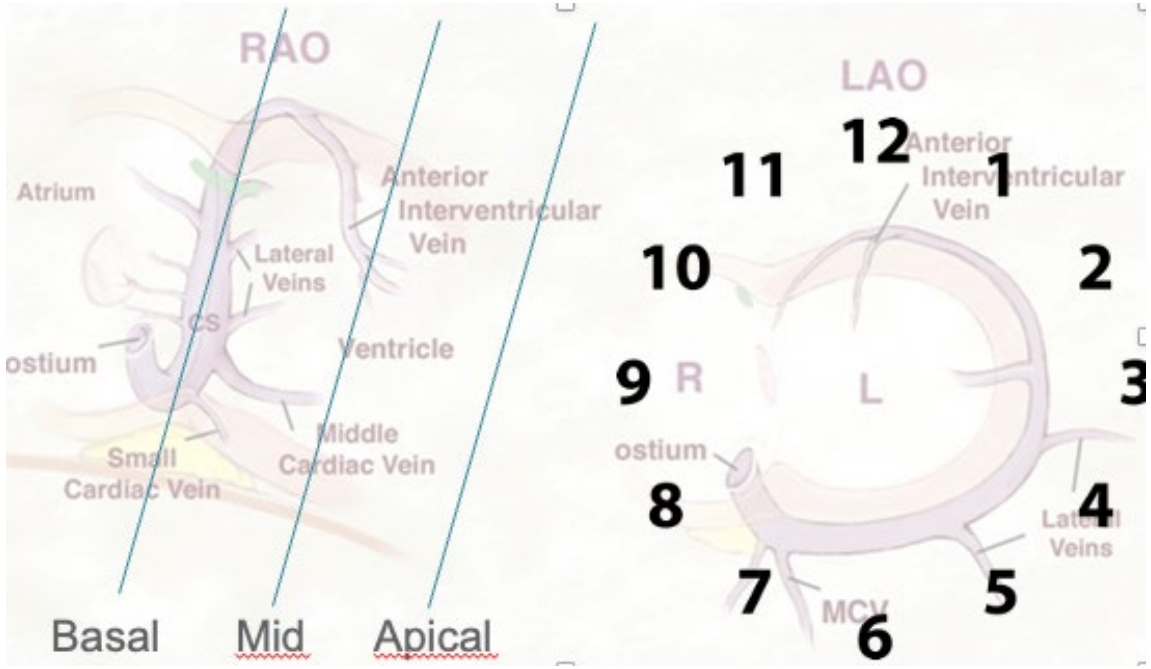
LV Lead Location on ECG

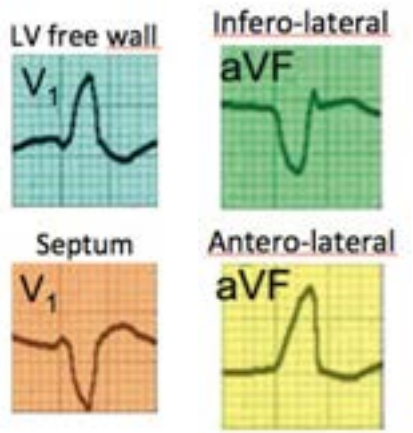
Precordial Leads

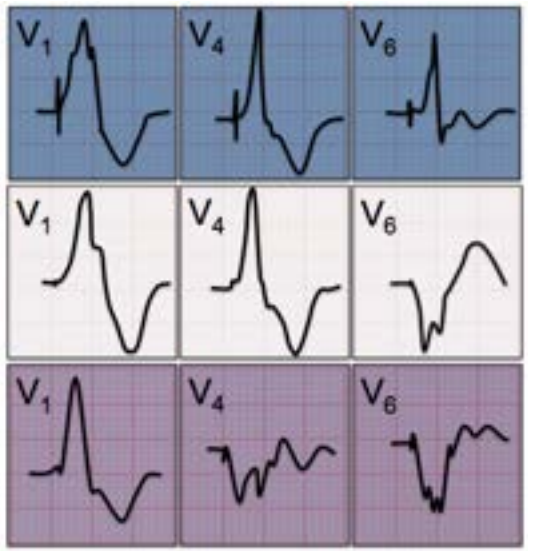
- Positive concordance: basal
- Negative concordance: apical
- Positive to negative: mid-level
- Negative to positive: septum or RV

Limb Leads

- Horizontal axis: postero-lateral (3h)
- Left or right superior axis: inferior to posterior (4-5h)
- Intermediate or right axis: antero-lateral to lateral (1-2h)
- Inferior axis: anterior (12h)
- Superior axis: inferior (6h)





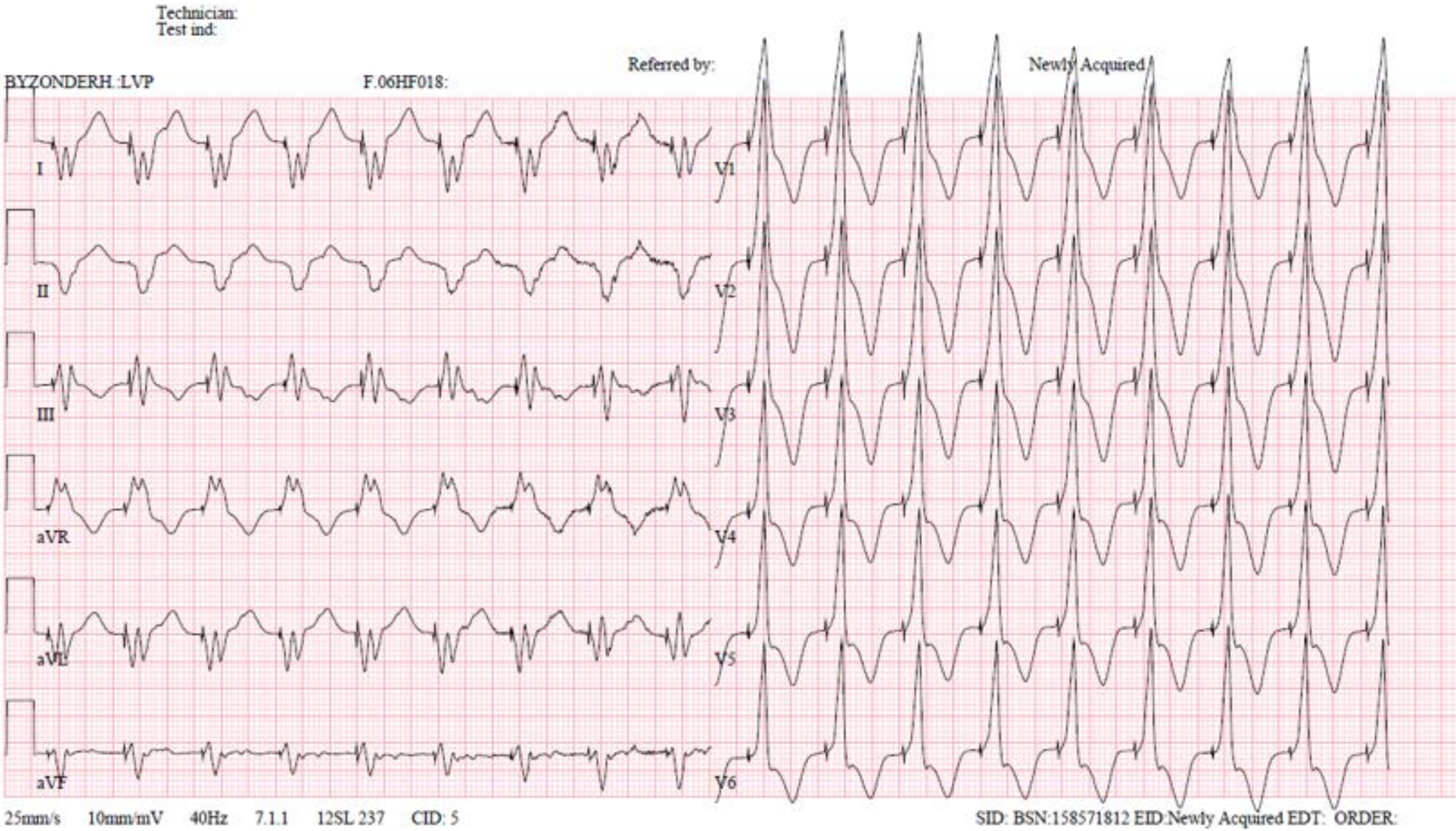


Basal

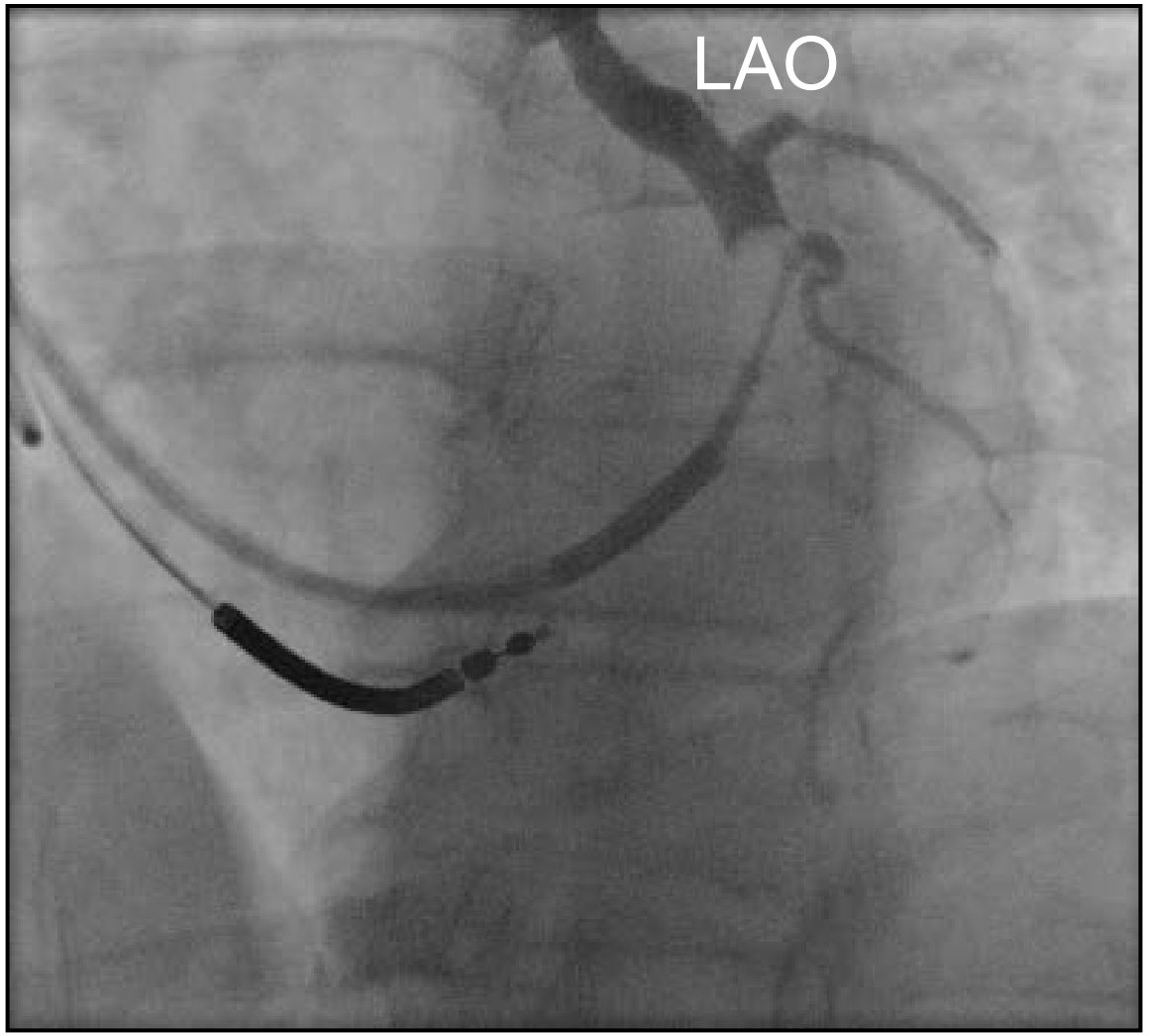
Mid

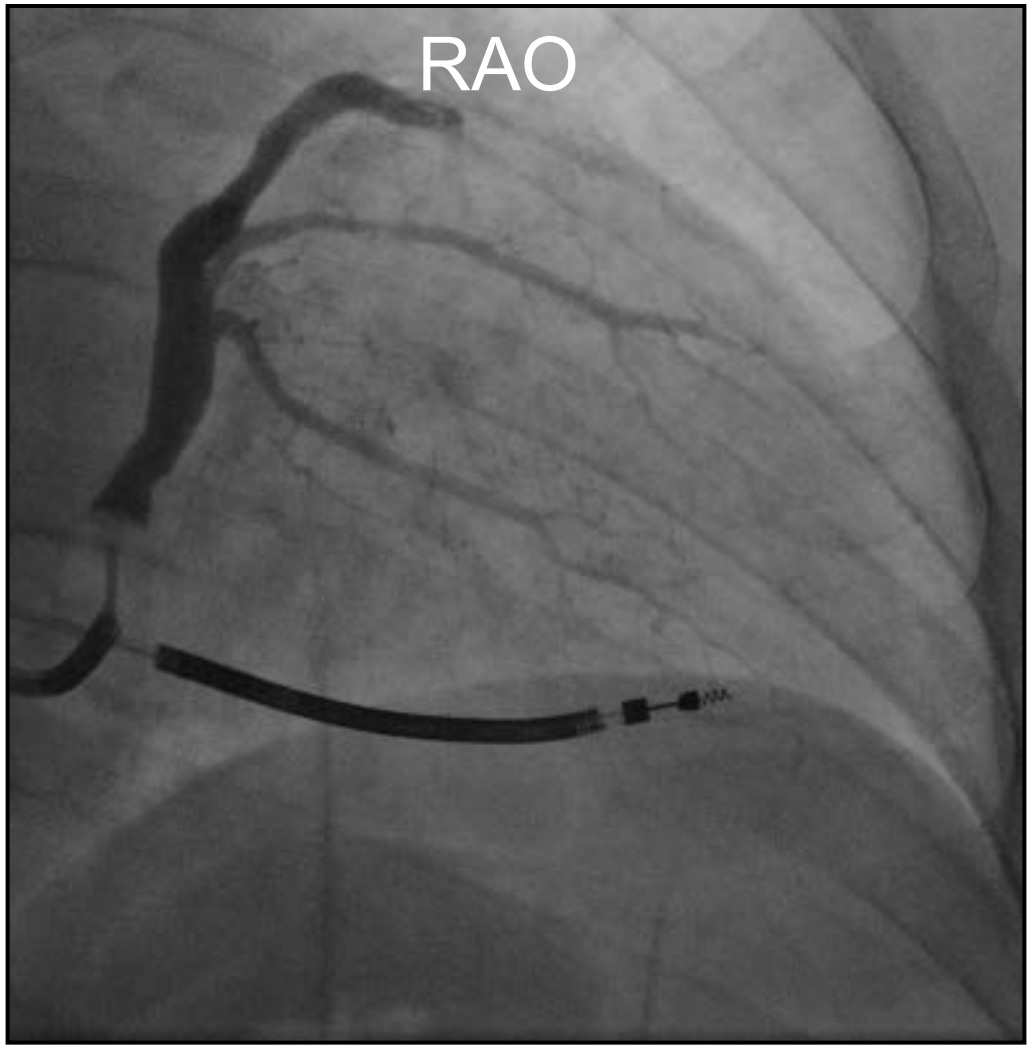
Apical

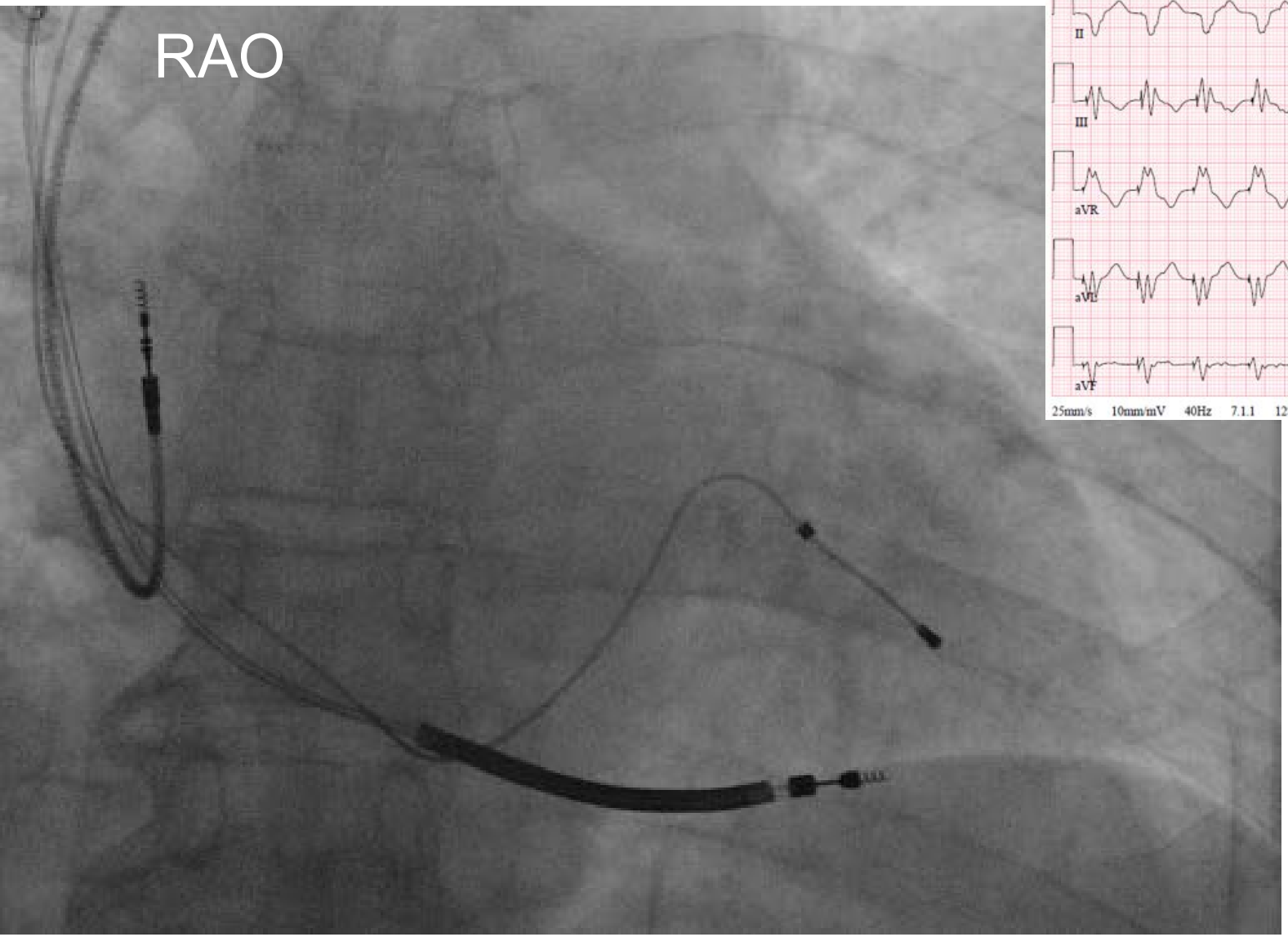
LV pacing: Where is the LV lead?

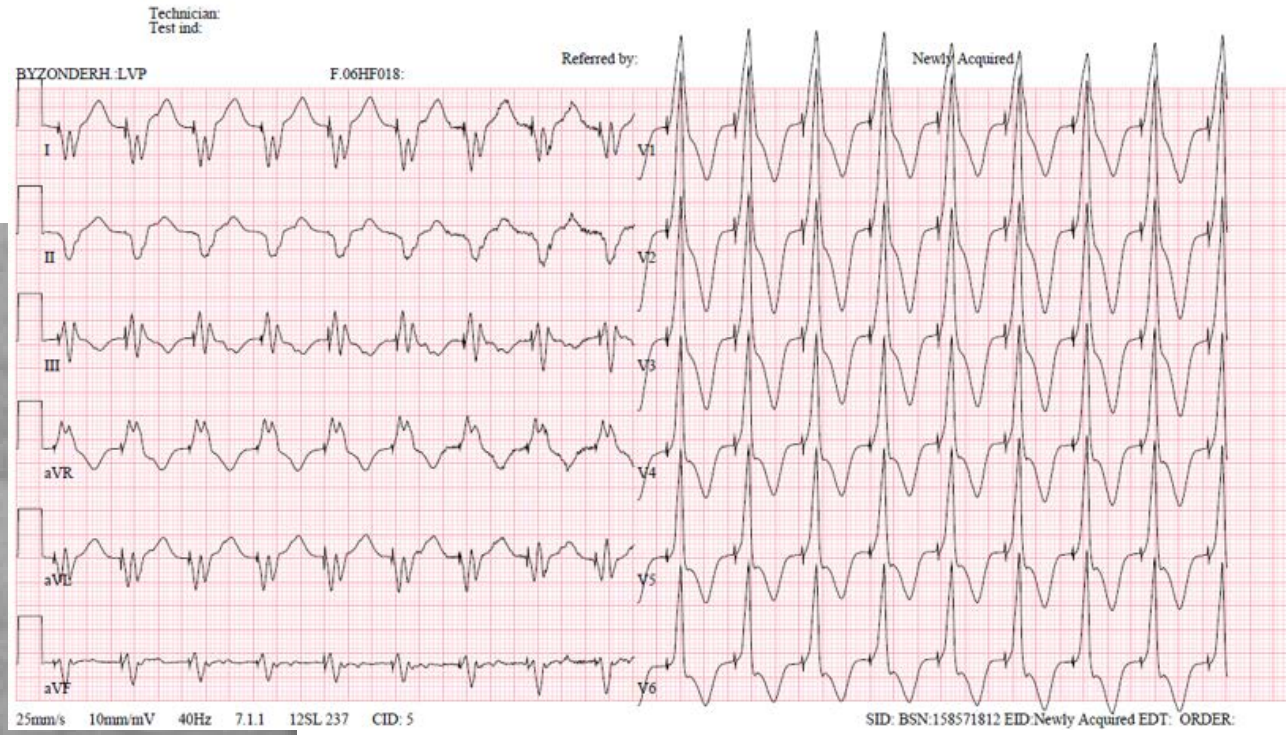


Fluoroscopy





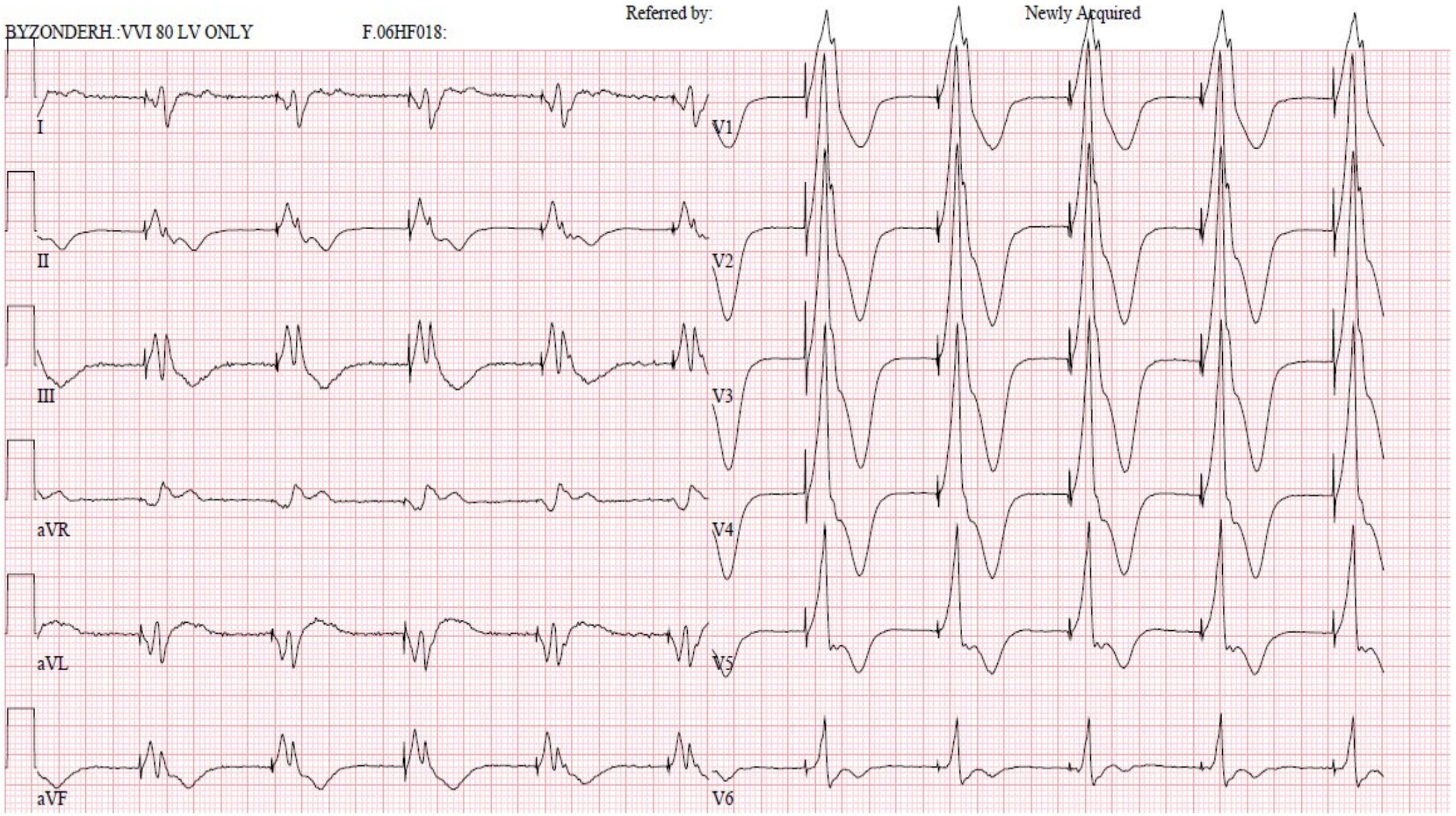




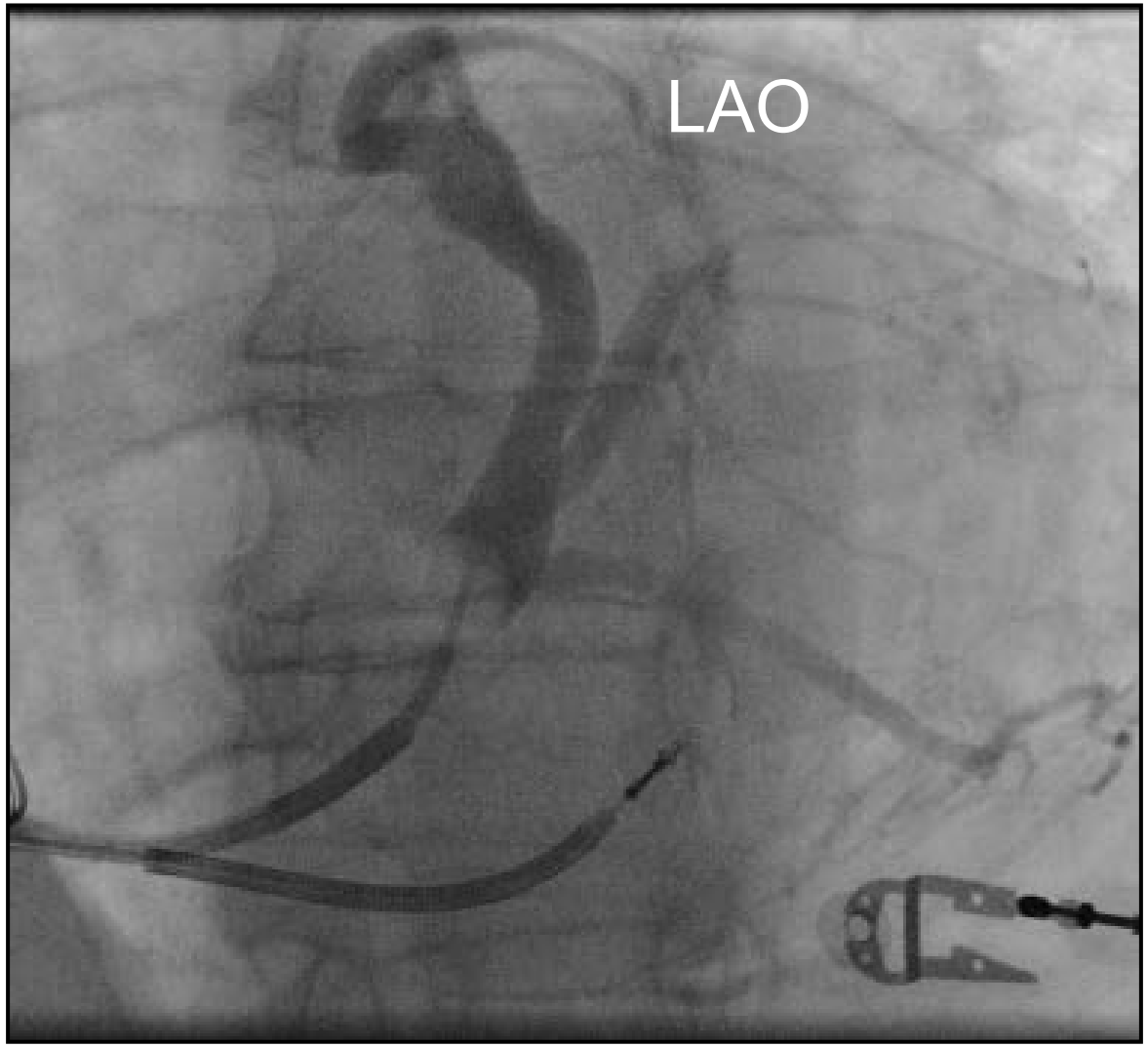
<u>Precordial Leads</u>
Positive concordance: basal

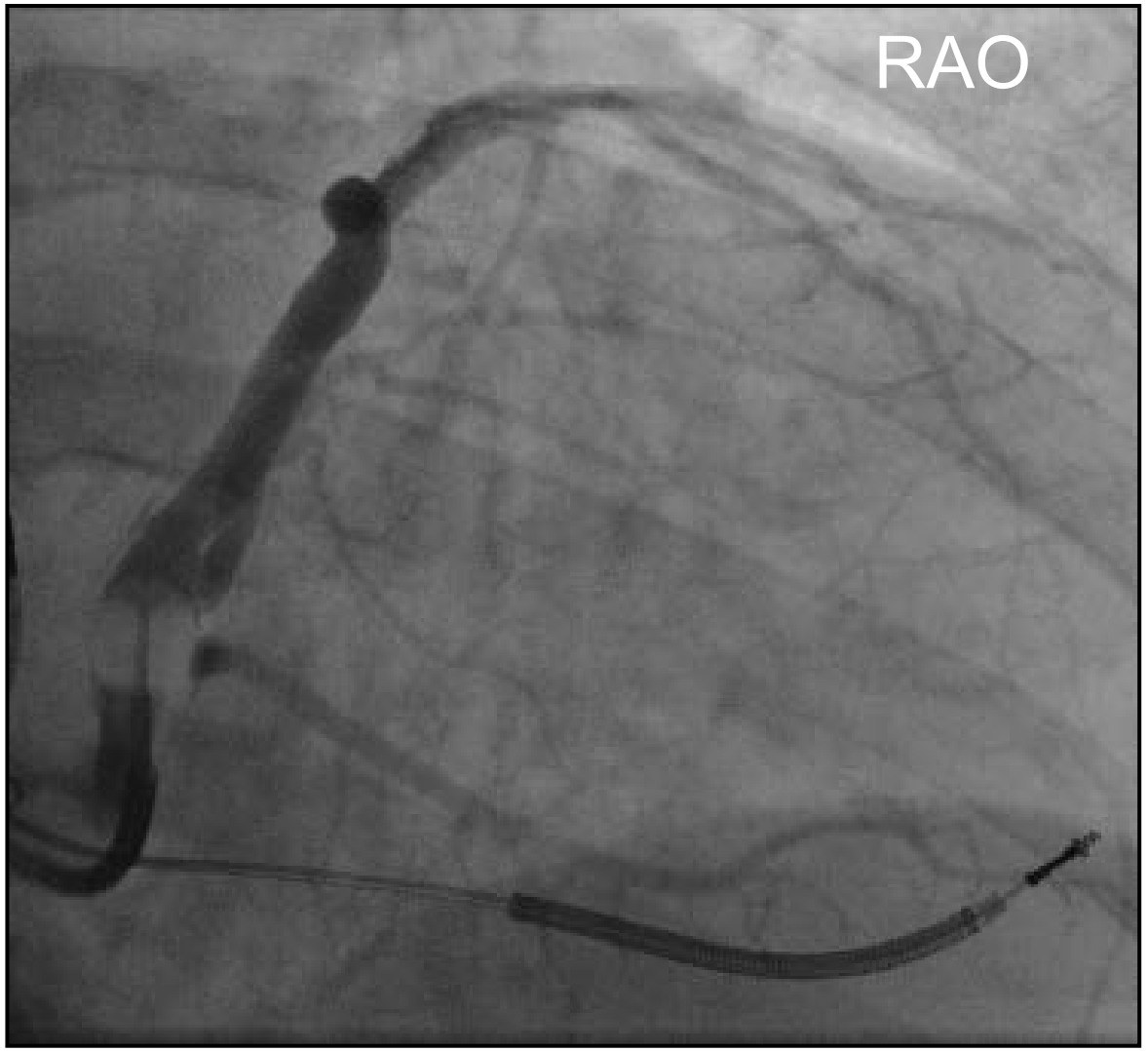
<u>Limb Leads</u>
Horizontal axis (lead I): postero-lateral (3h)
Left or right superior axis: inferior to posterior (4-5h)
Intermediate or right axis: antero-lateral to lateral (1-2h)
Inferior axis: anterior (12h)
Superior axis: inferior (6h)

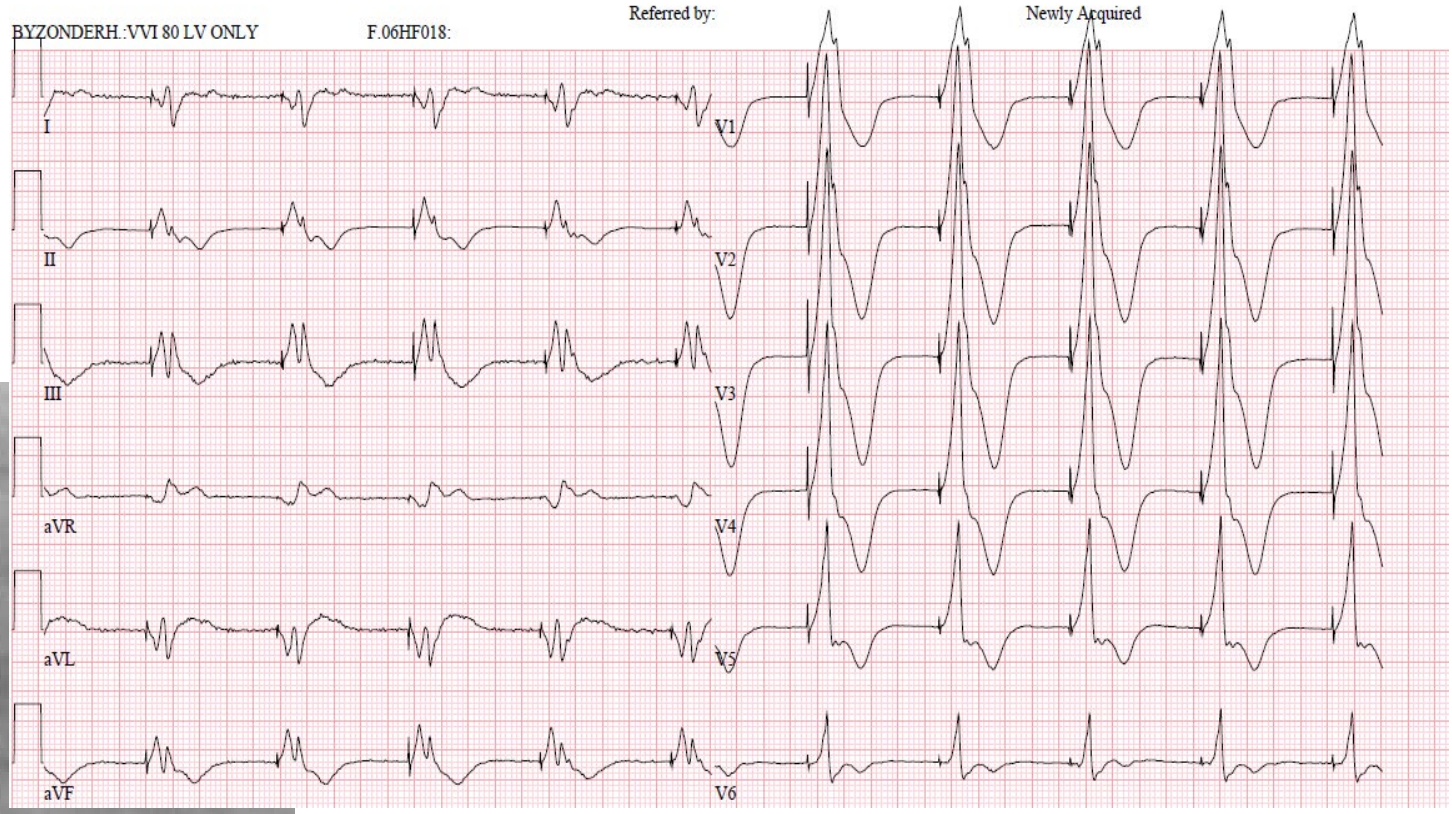
Where is the LV lead?

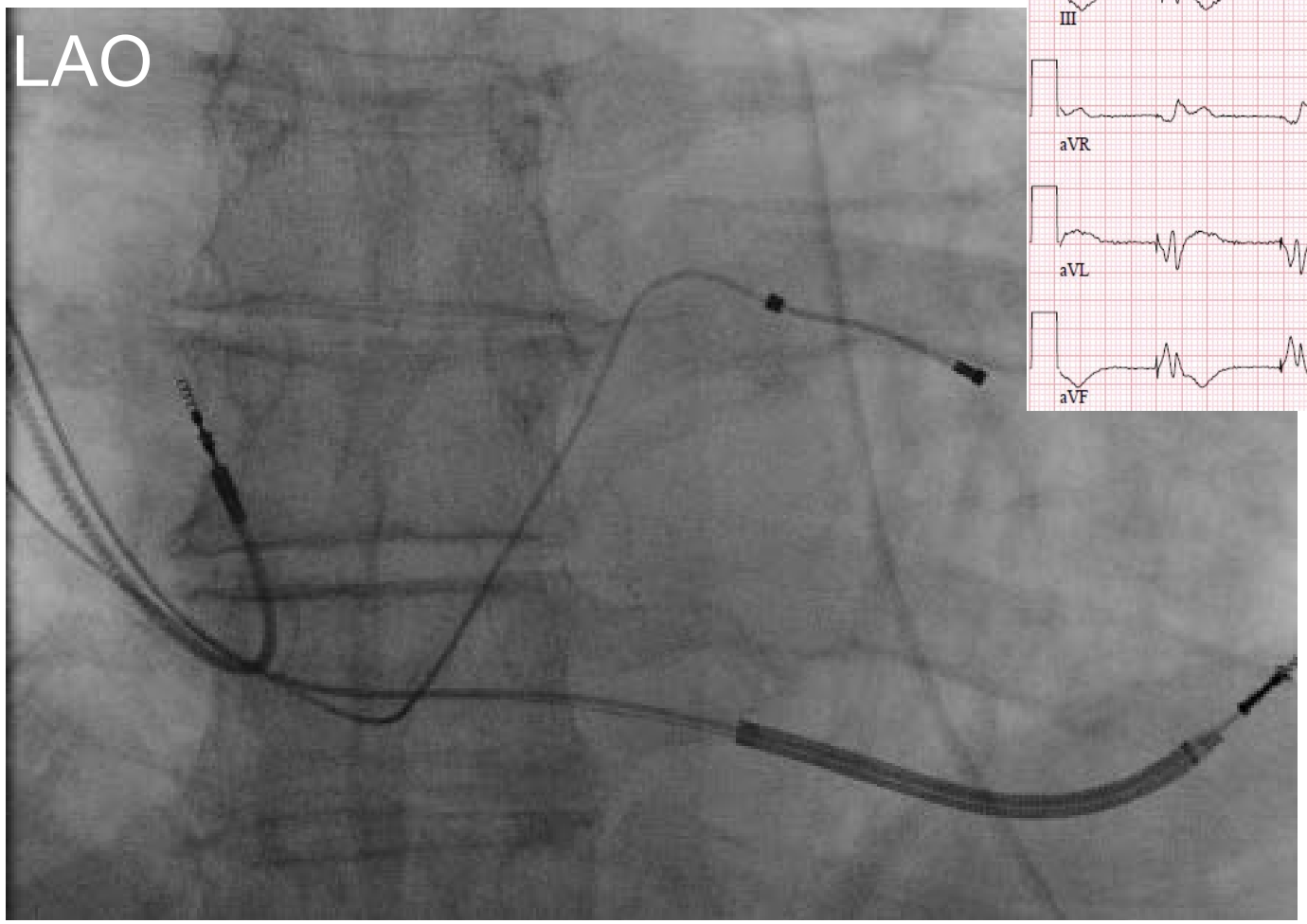


Fluoroscopy

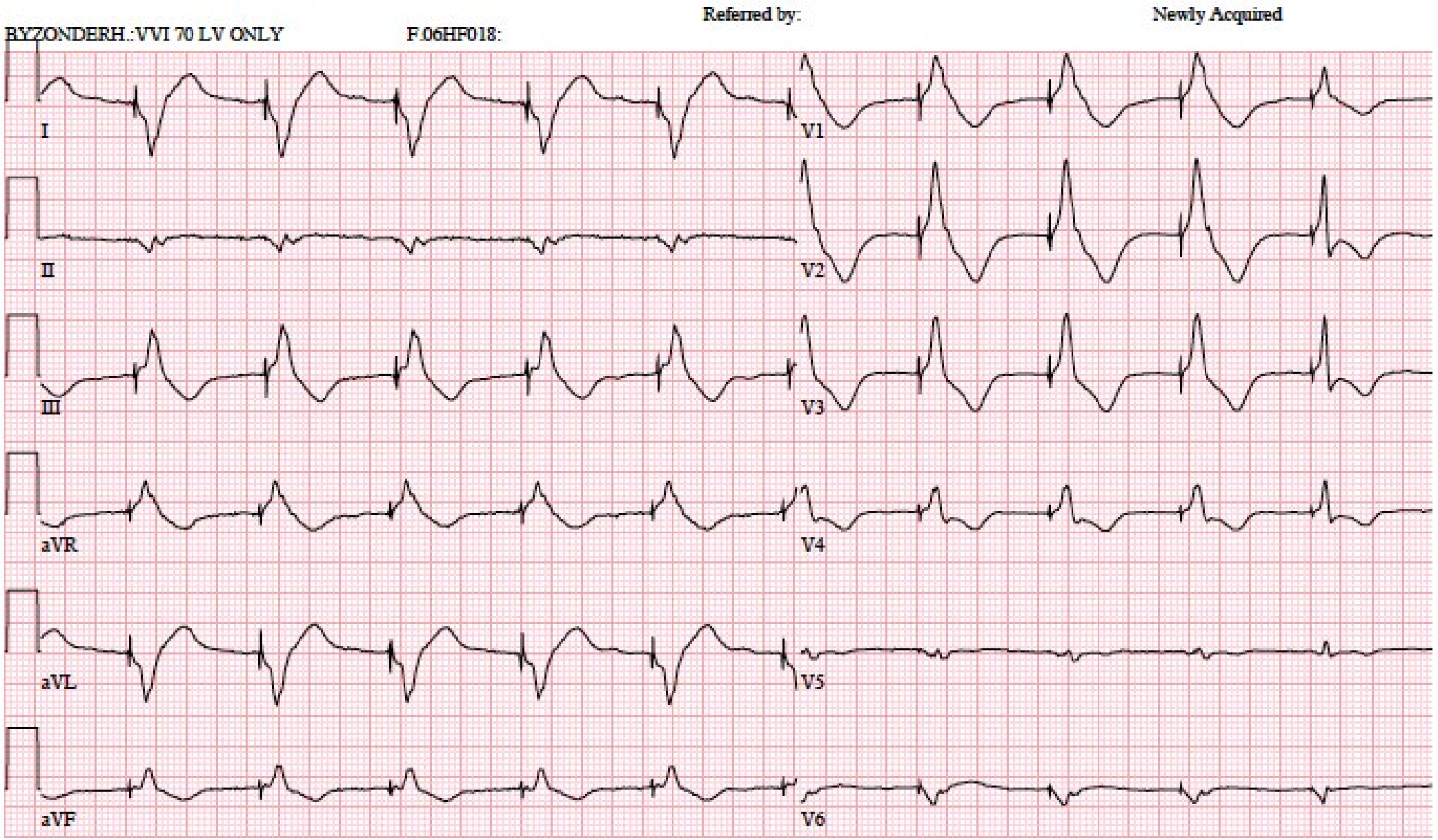


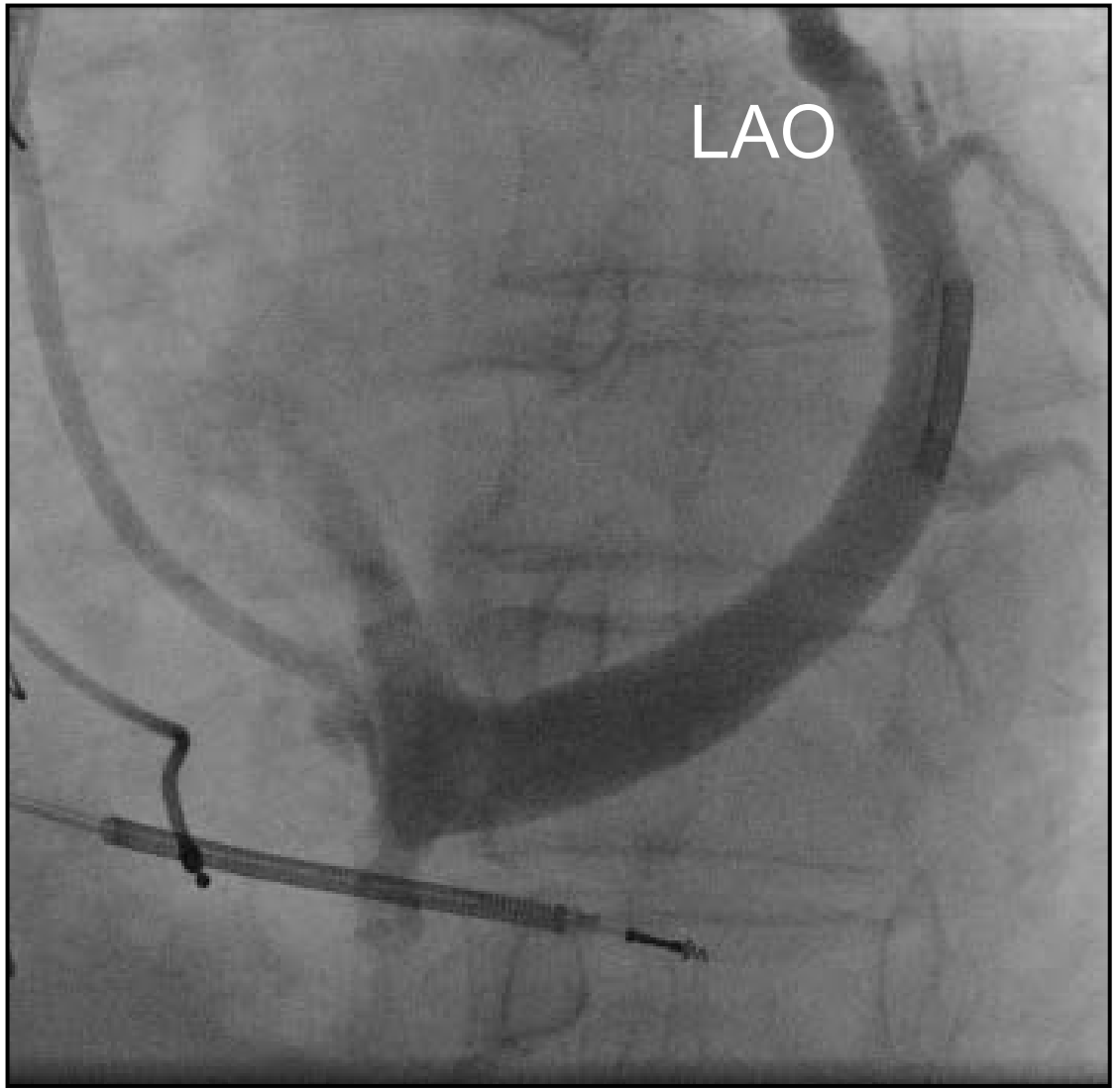


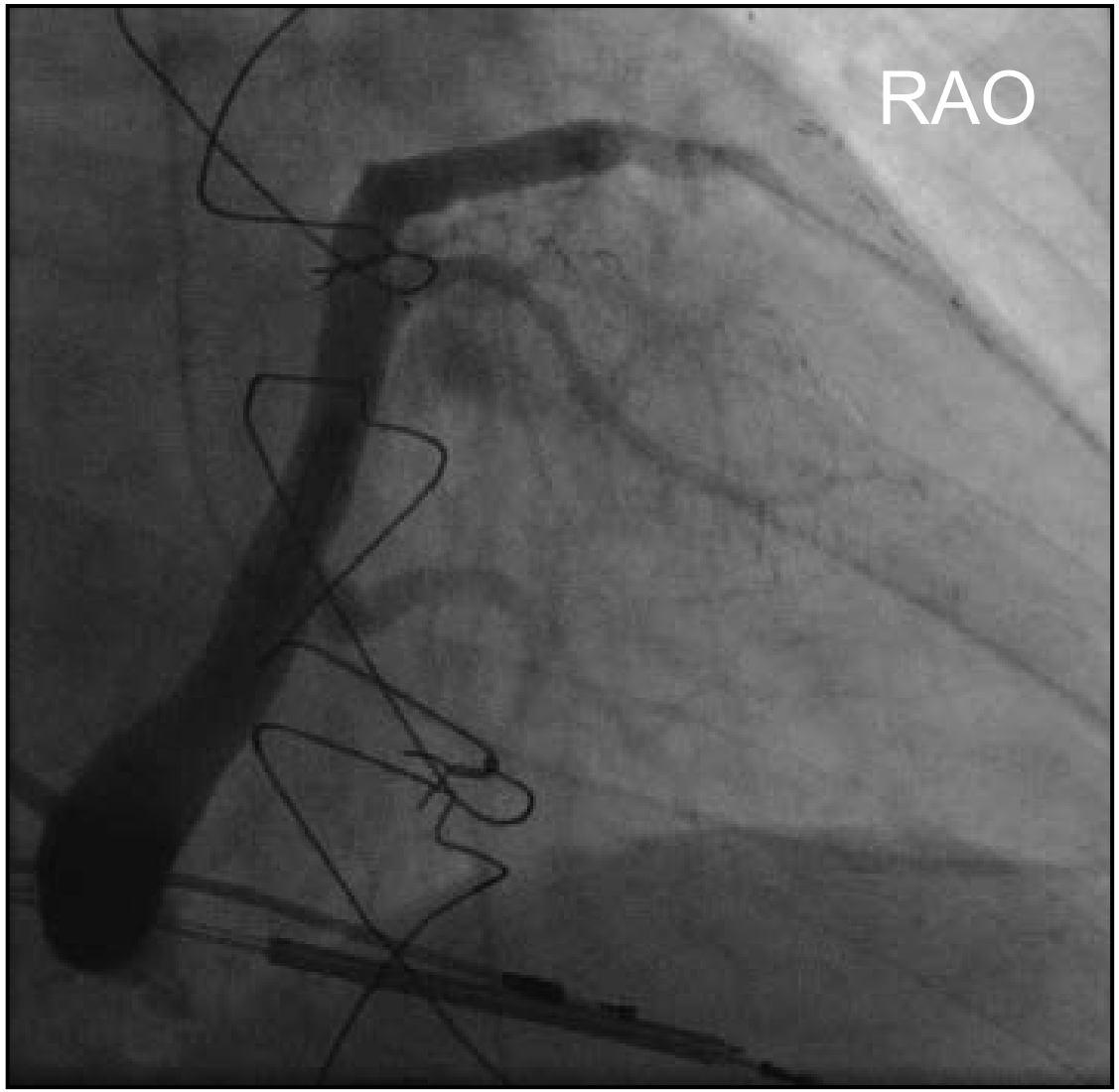

BYZONDERH.:VVI 80 LV ONLY
F.06HF018:
Referred by:
Newly Acquired
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

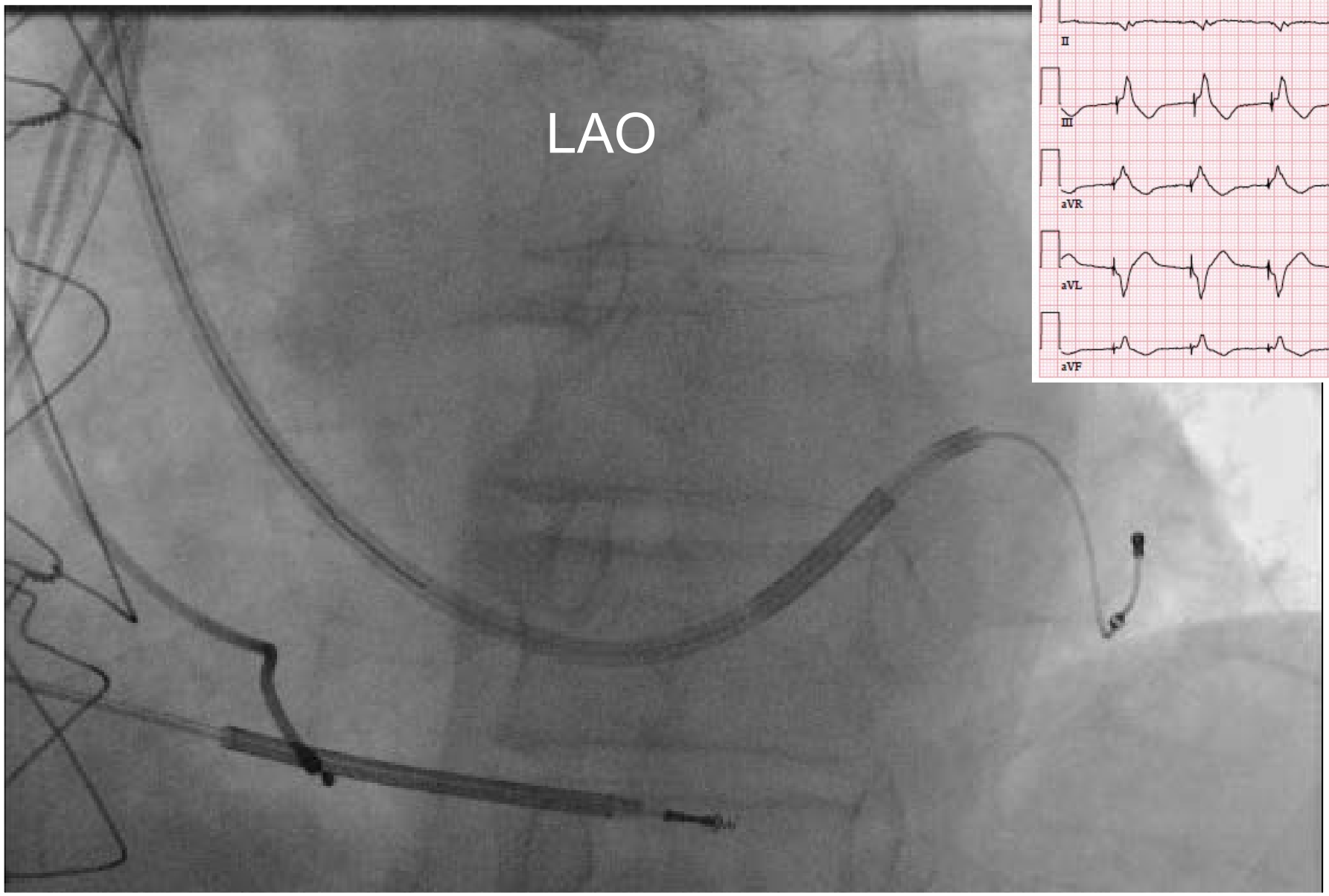

LAO

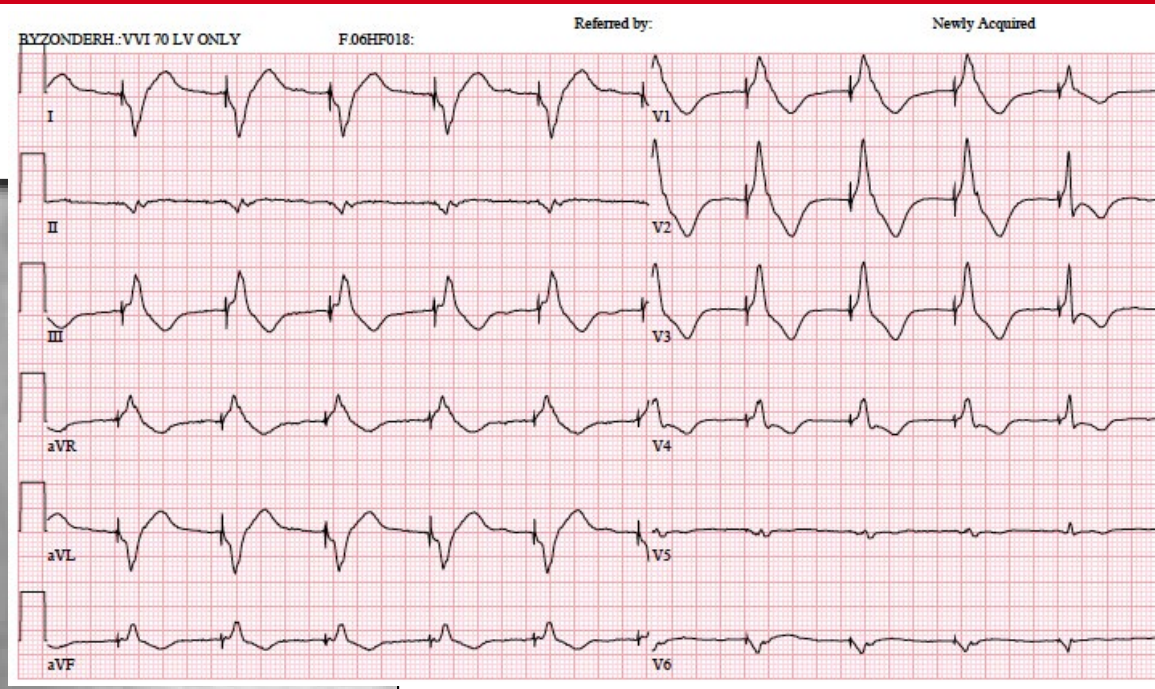
Where is the LV lead (LV pacing)



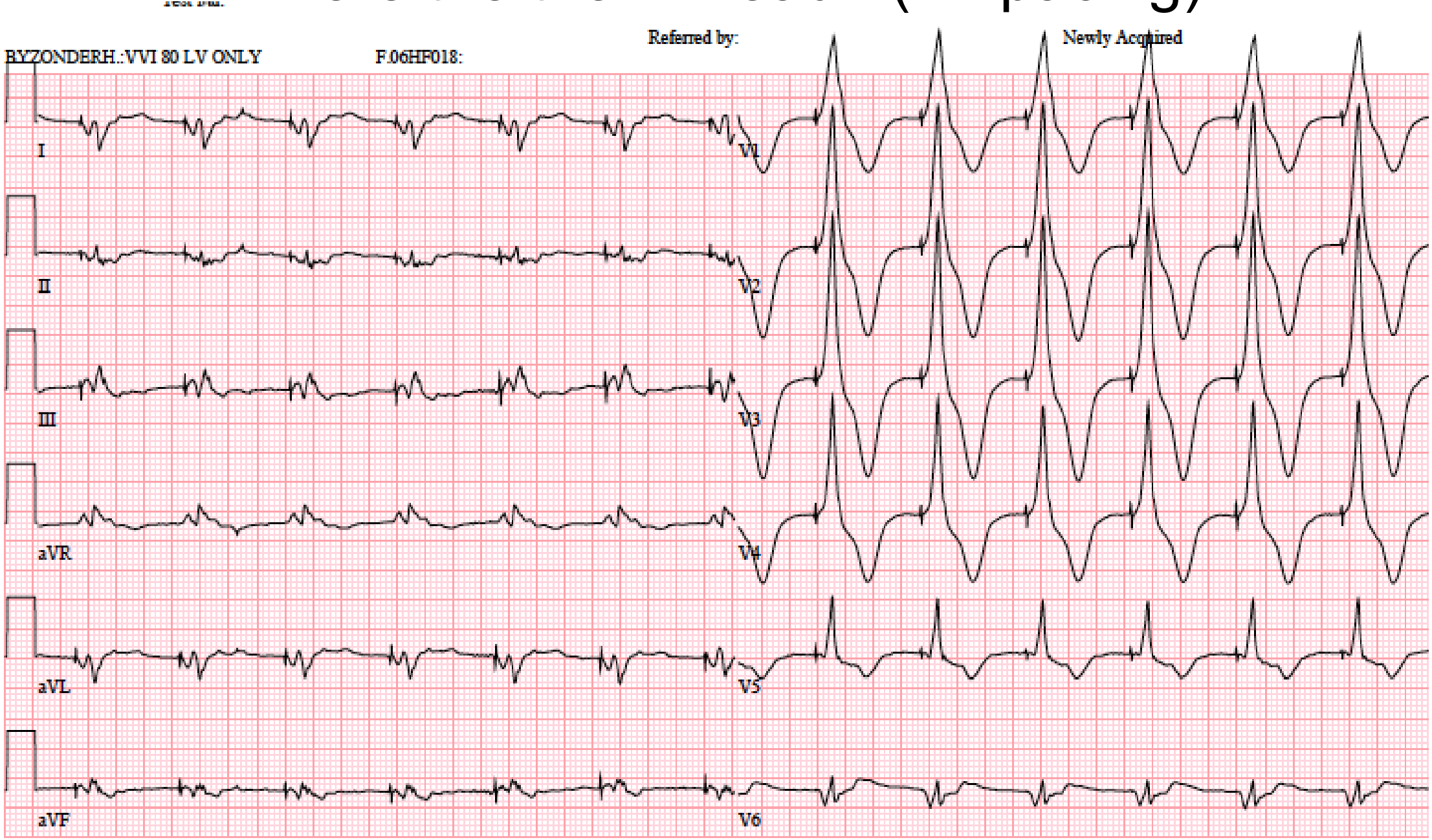

LAO

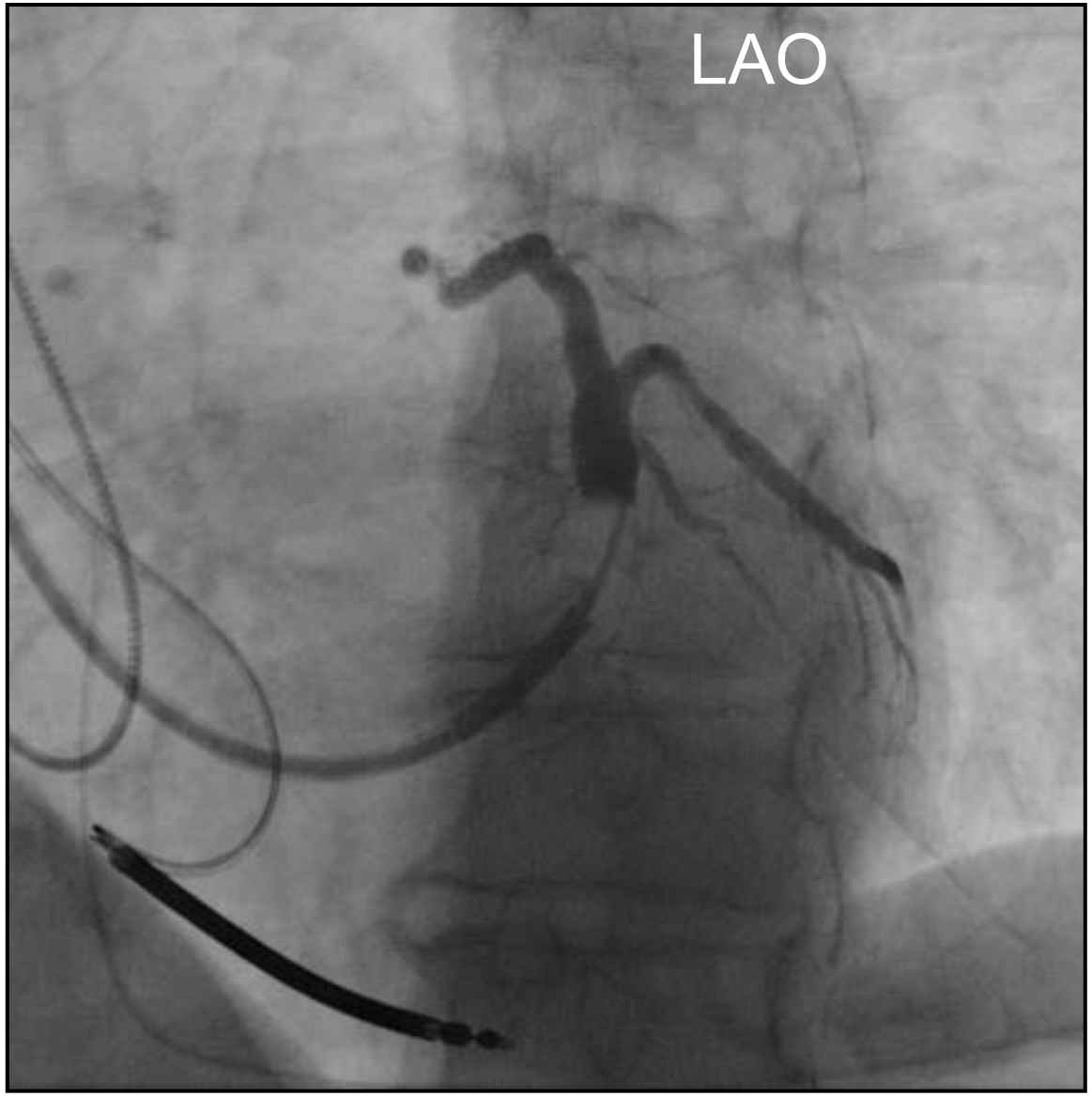

RAO

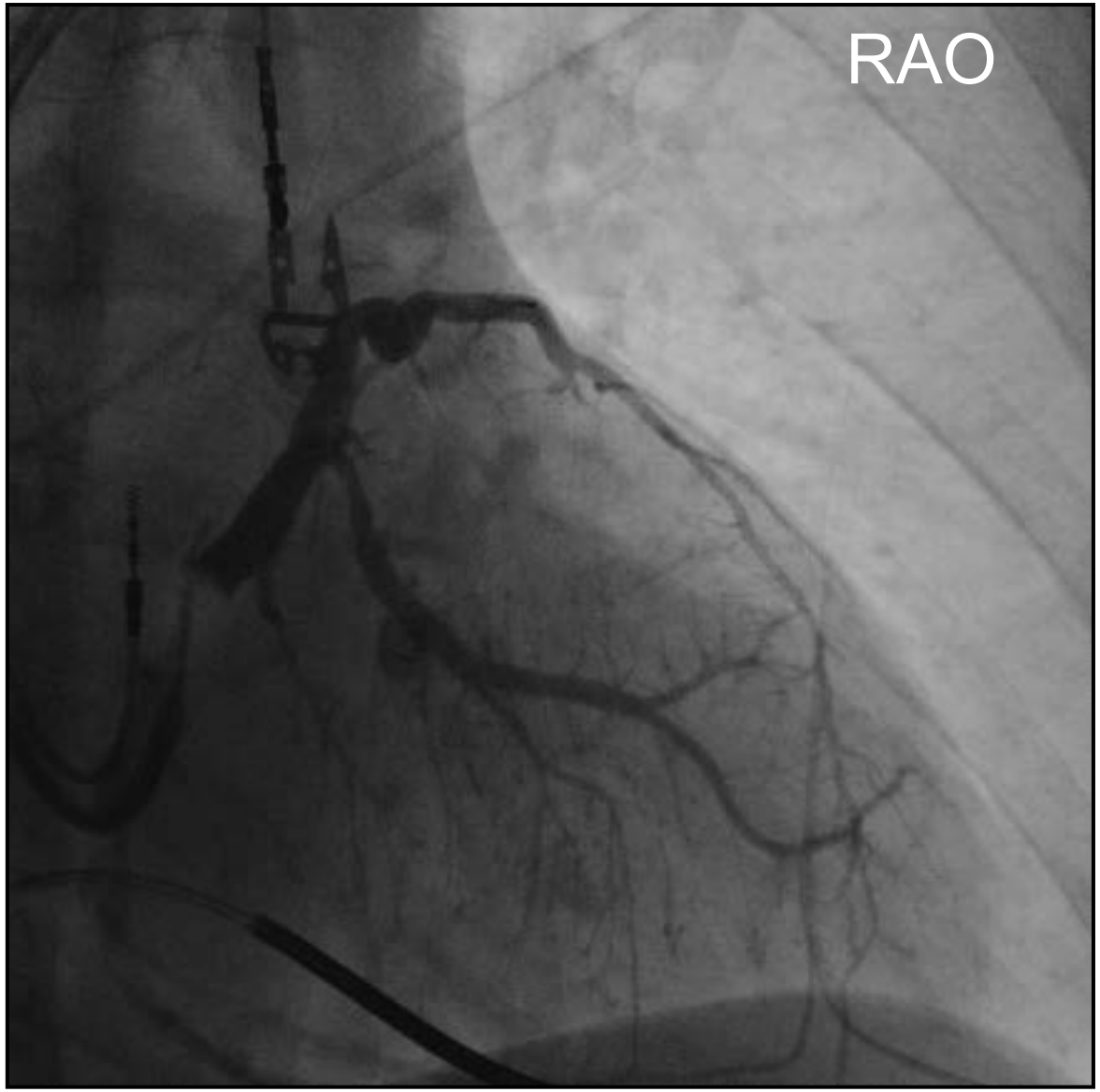

LAO


RYZONDERH.:VVI 70 LV ONLY
F.06HF018:
Referred by:
Newly Acquired
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Where the the LV lead? (LV pacing)




LAO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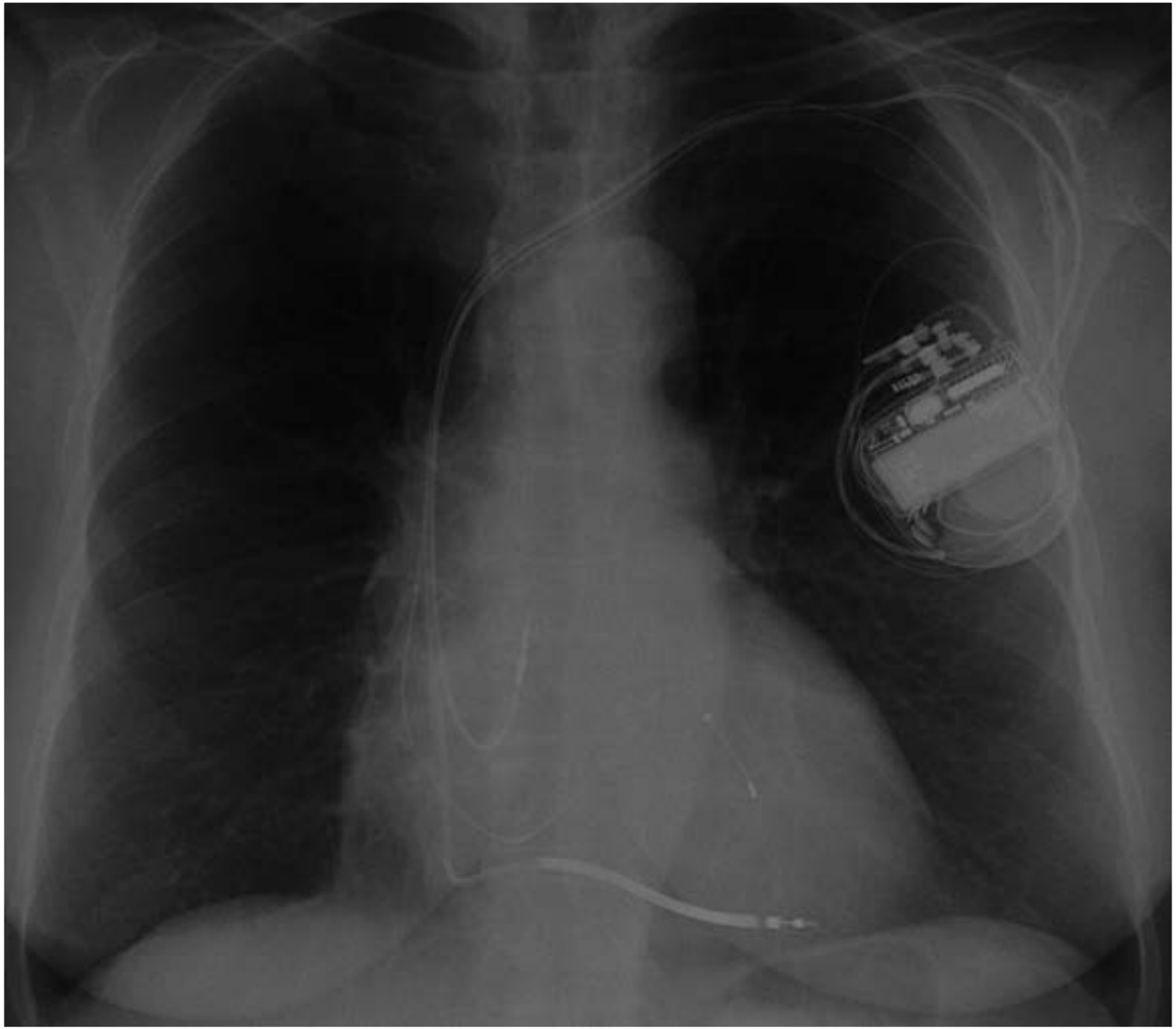
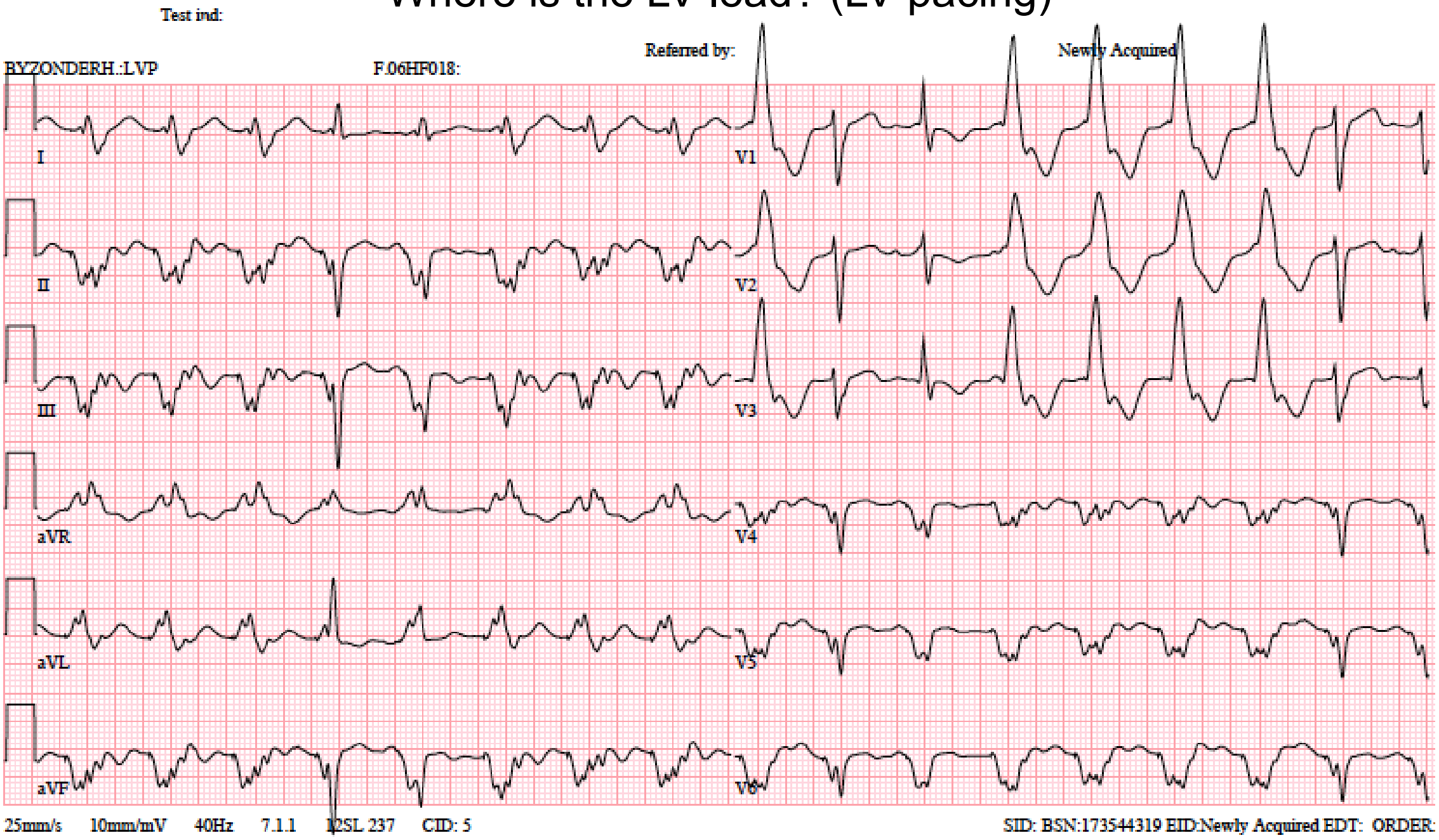
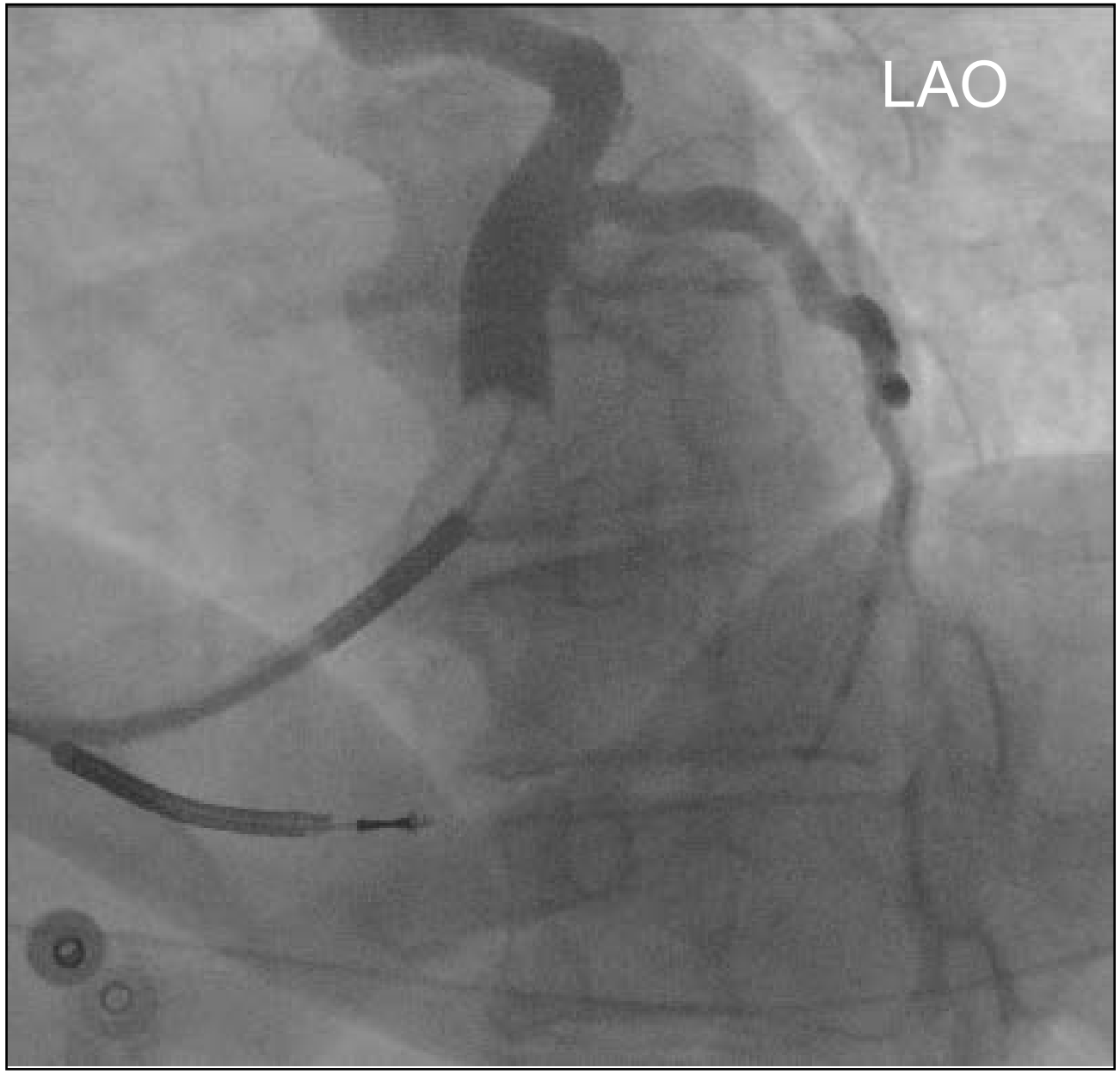
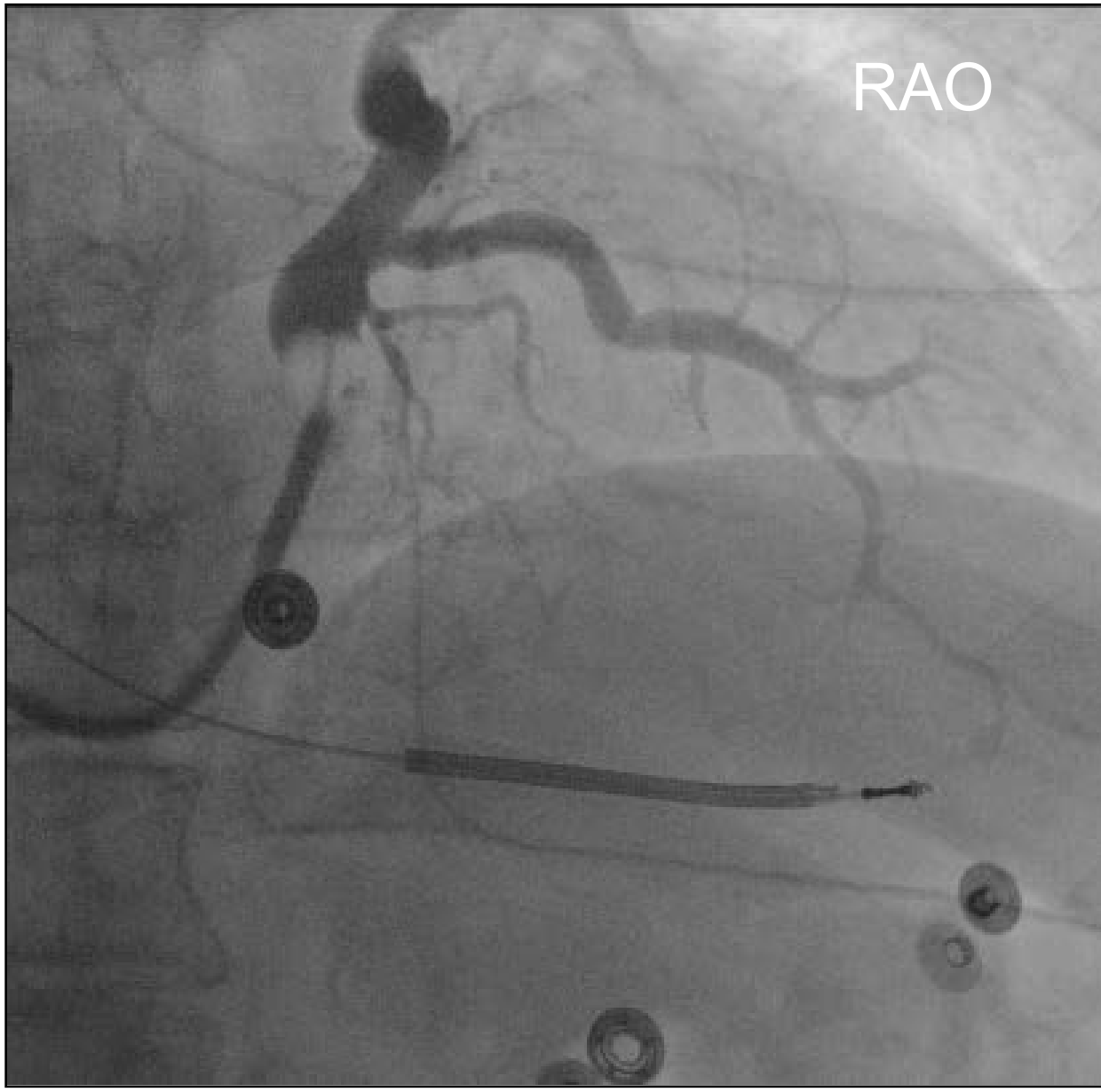


Where is the LV lead? (LV pacing)




LAO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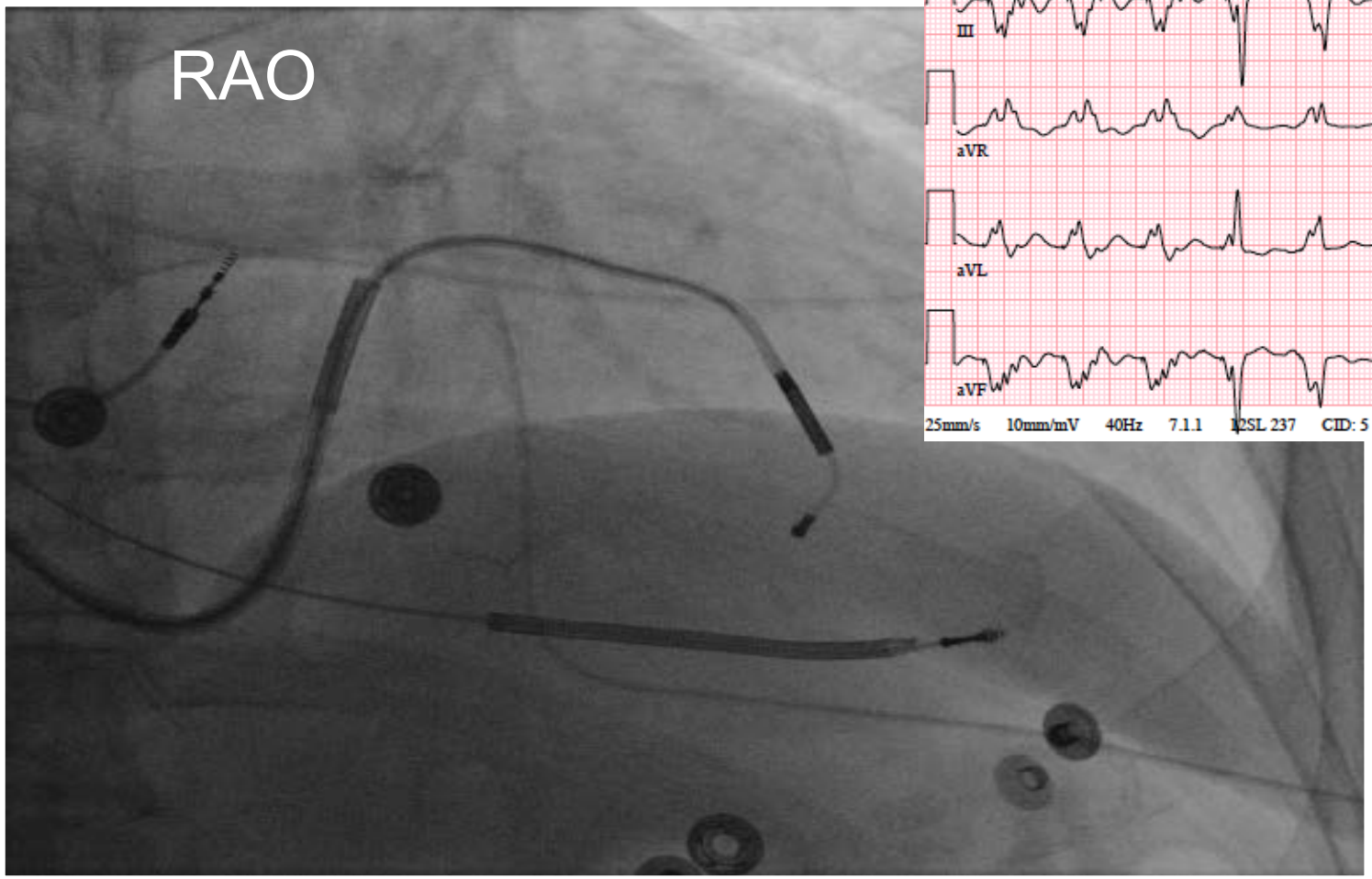
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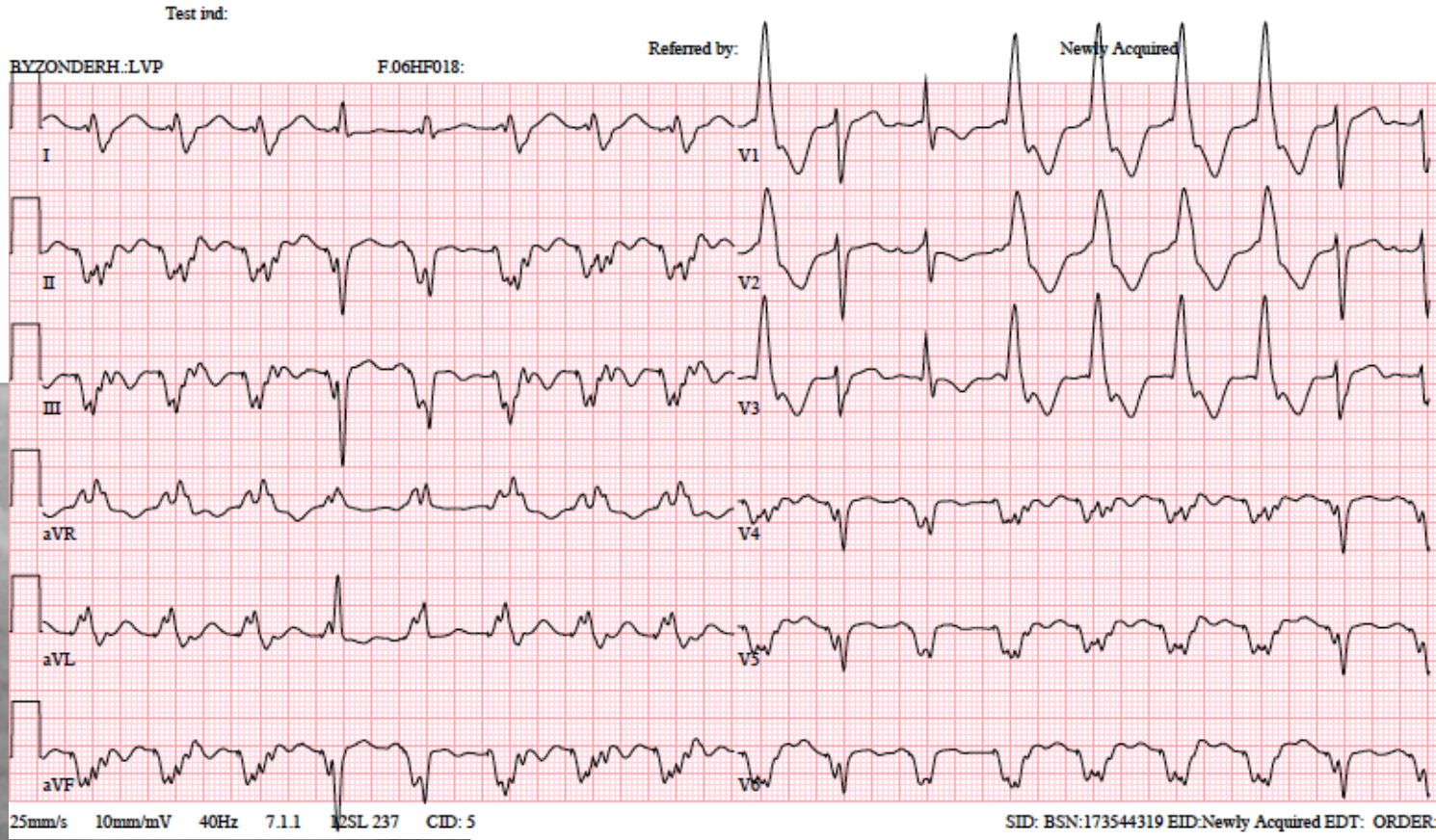
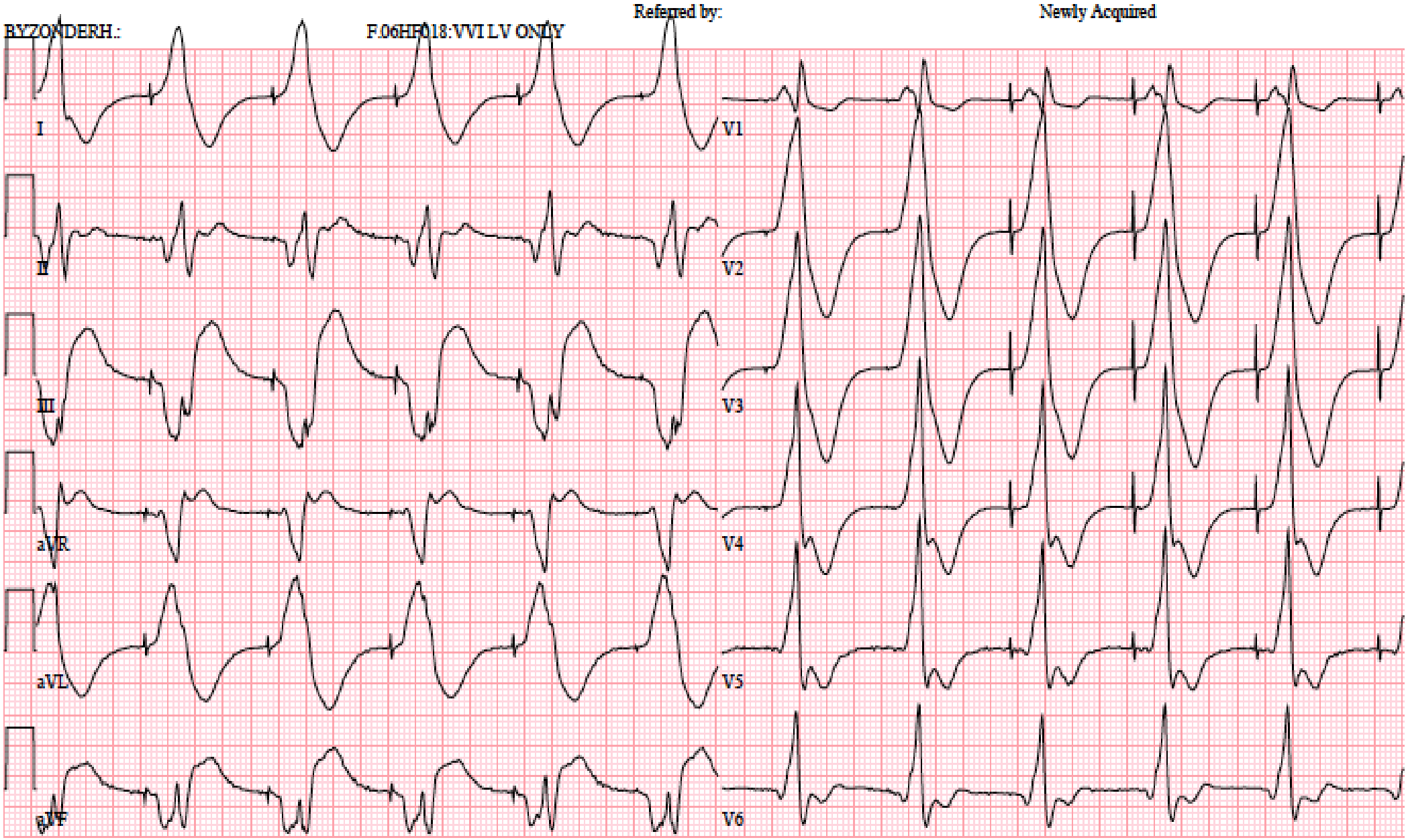
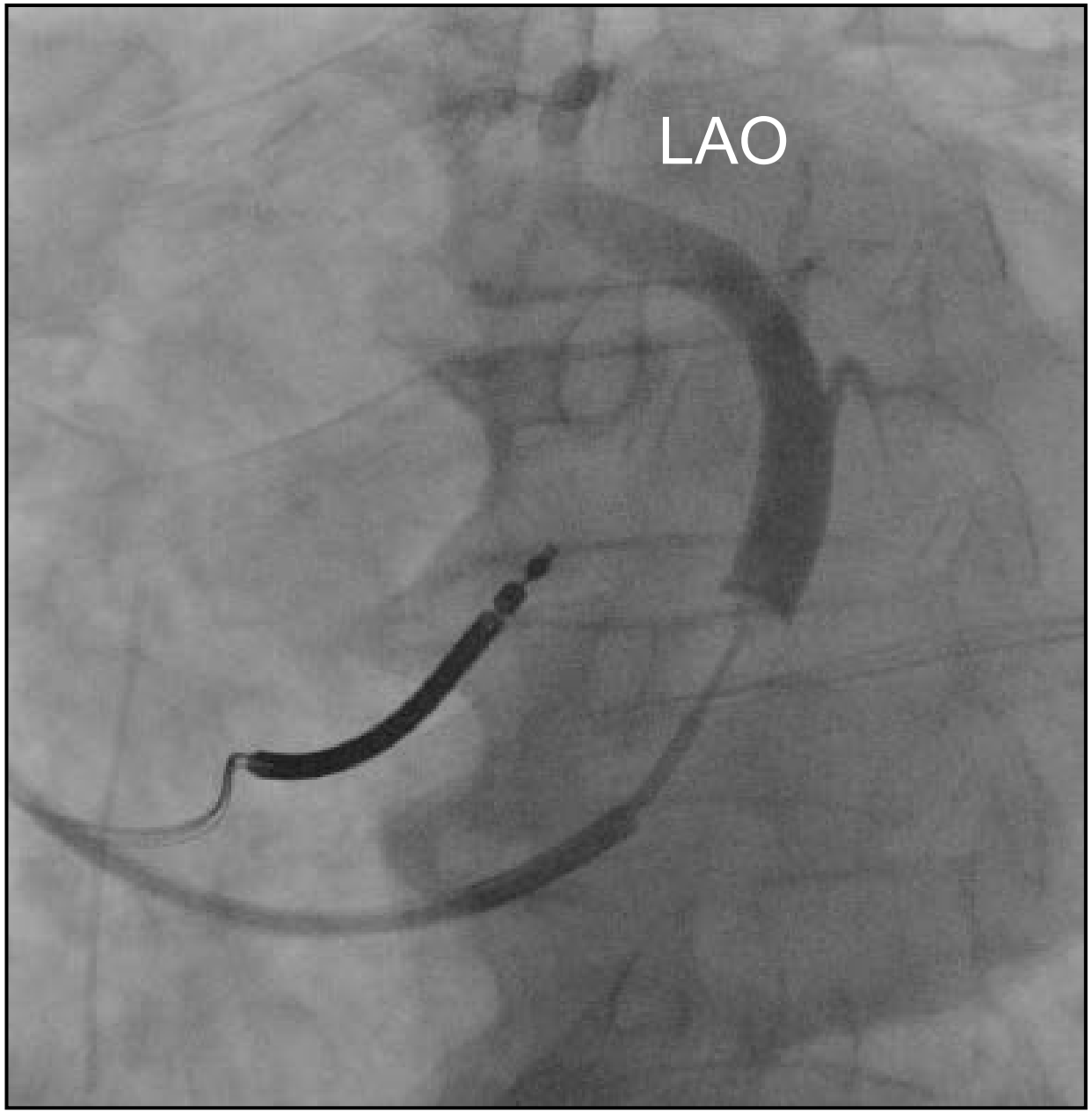

Test ind:
RYZONDERH.:LVP
F.06HF018:
Referred by:
Newly Acquired
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
25mm/s 10mm/mV 40Hz 7.1.1 12SL 237 CID: 5
SID: BSN:173544319 EID:Newly Acquired EDT: ORDER:

LV Pacing: Where is the LV Lead?

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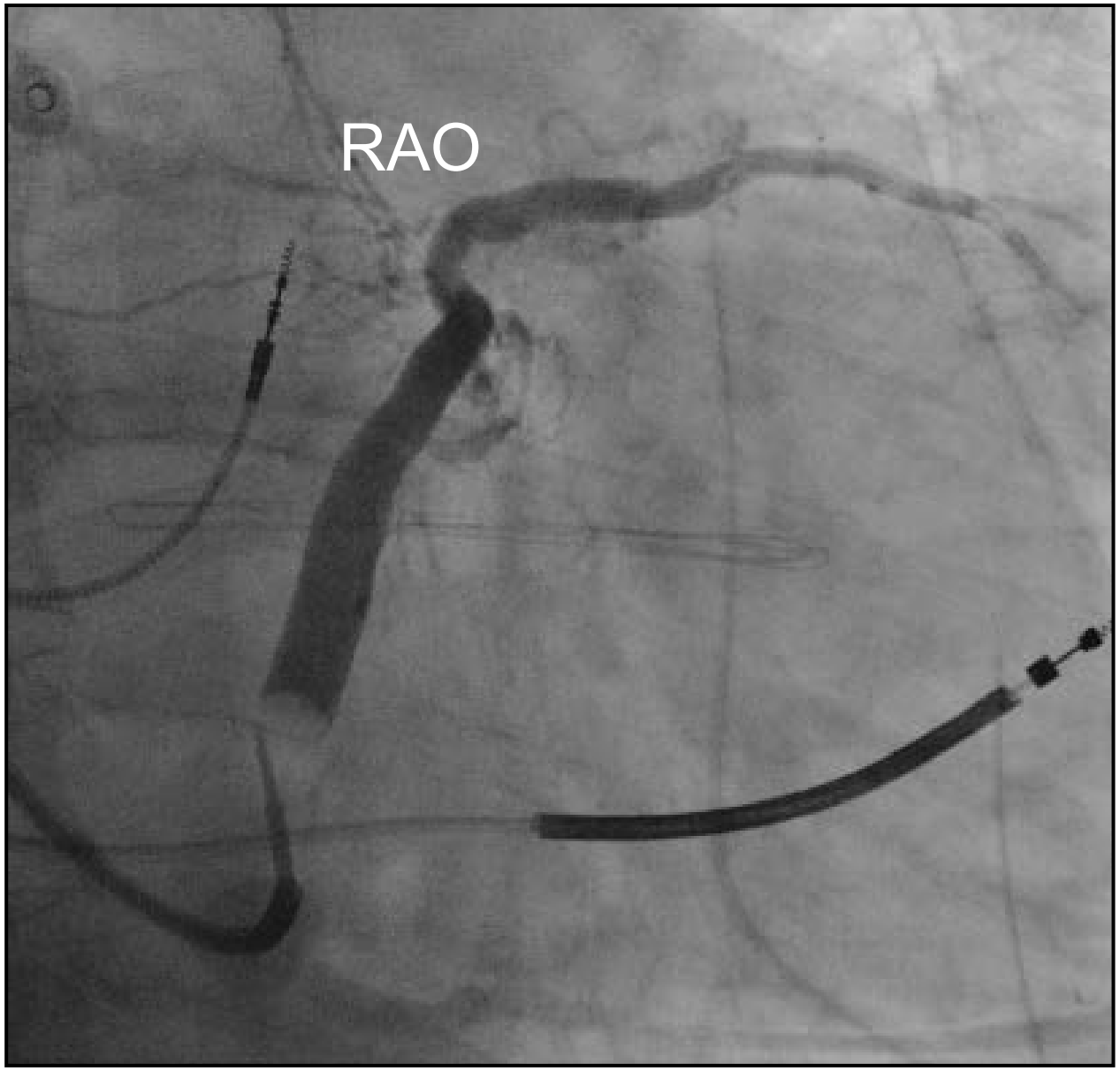
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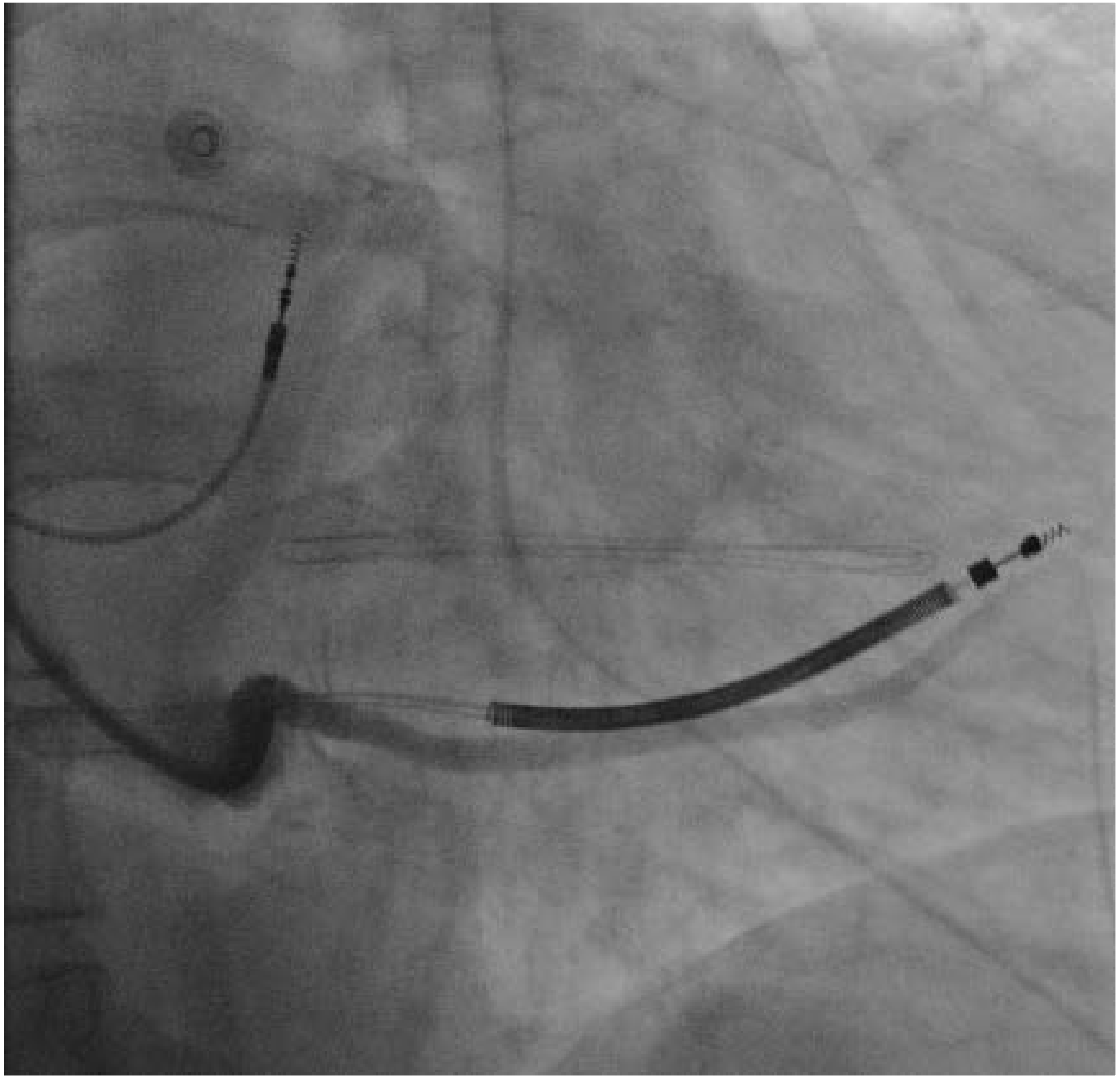
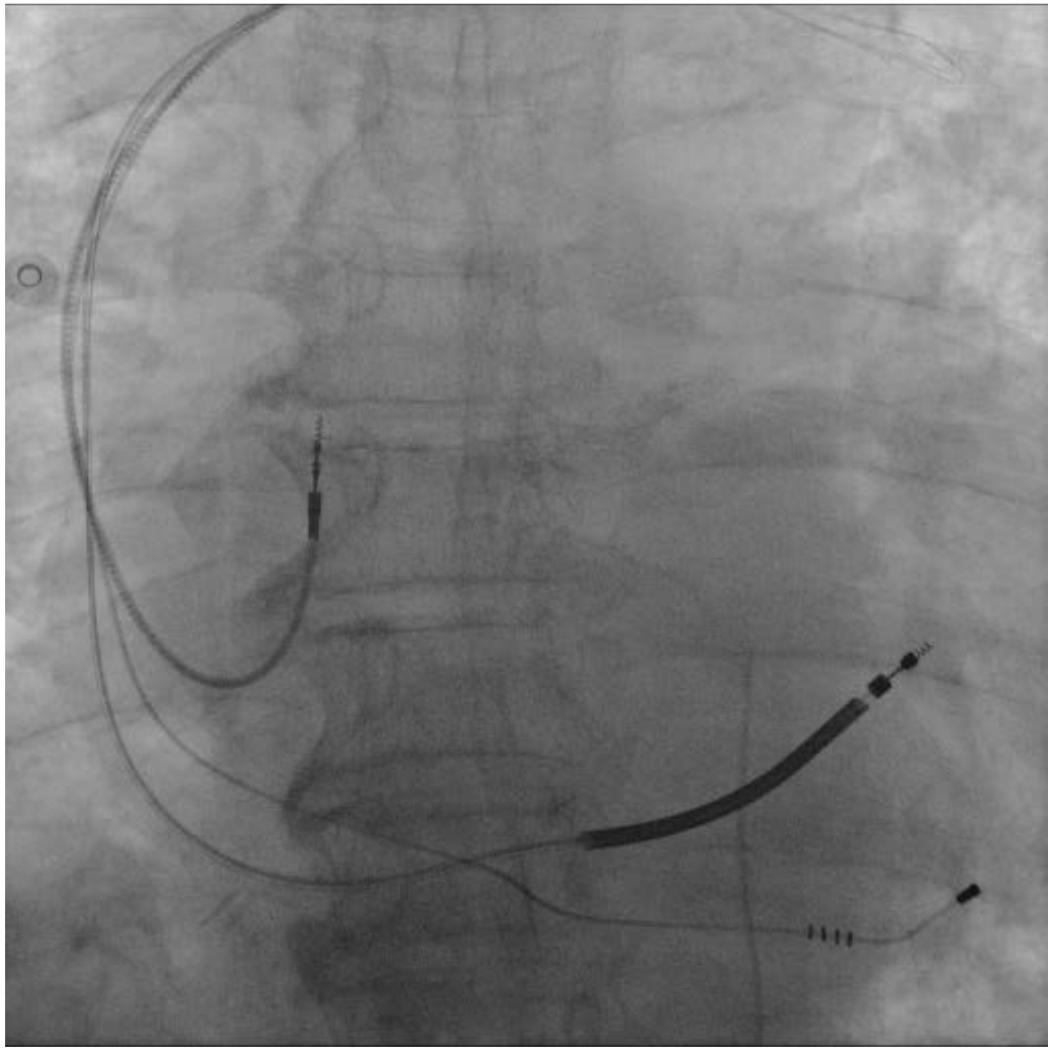
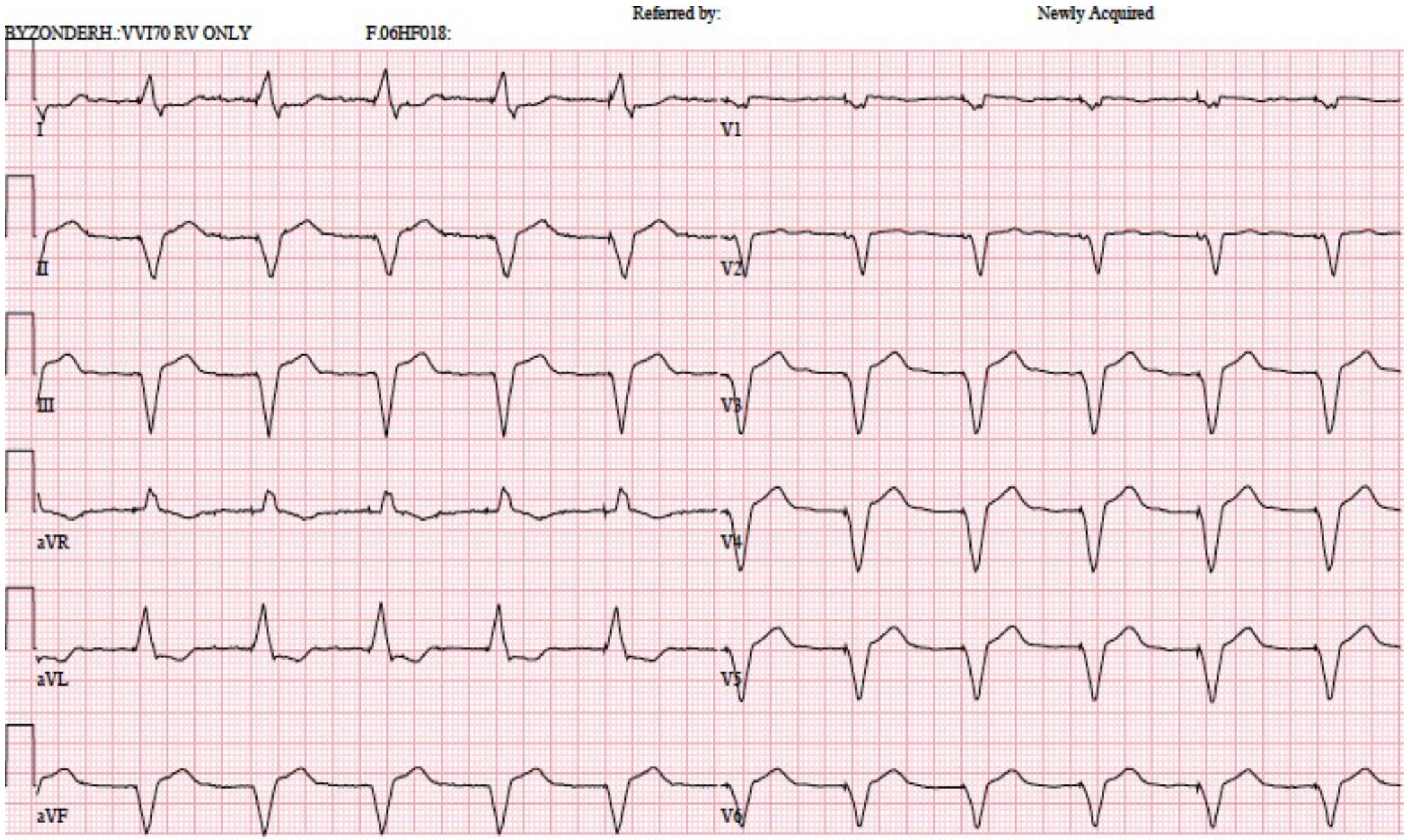
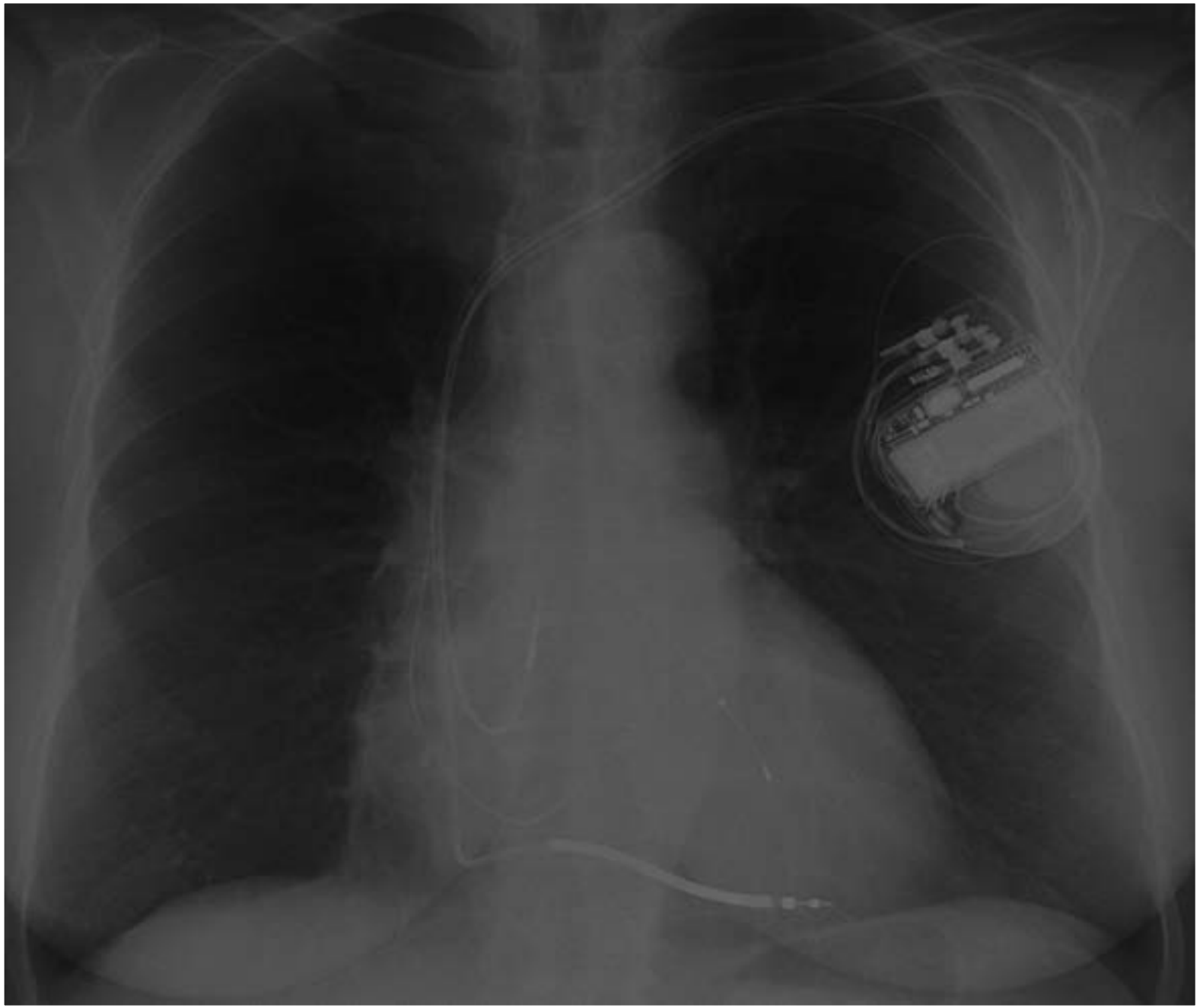




Infero-posterior vene: Suitable?

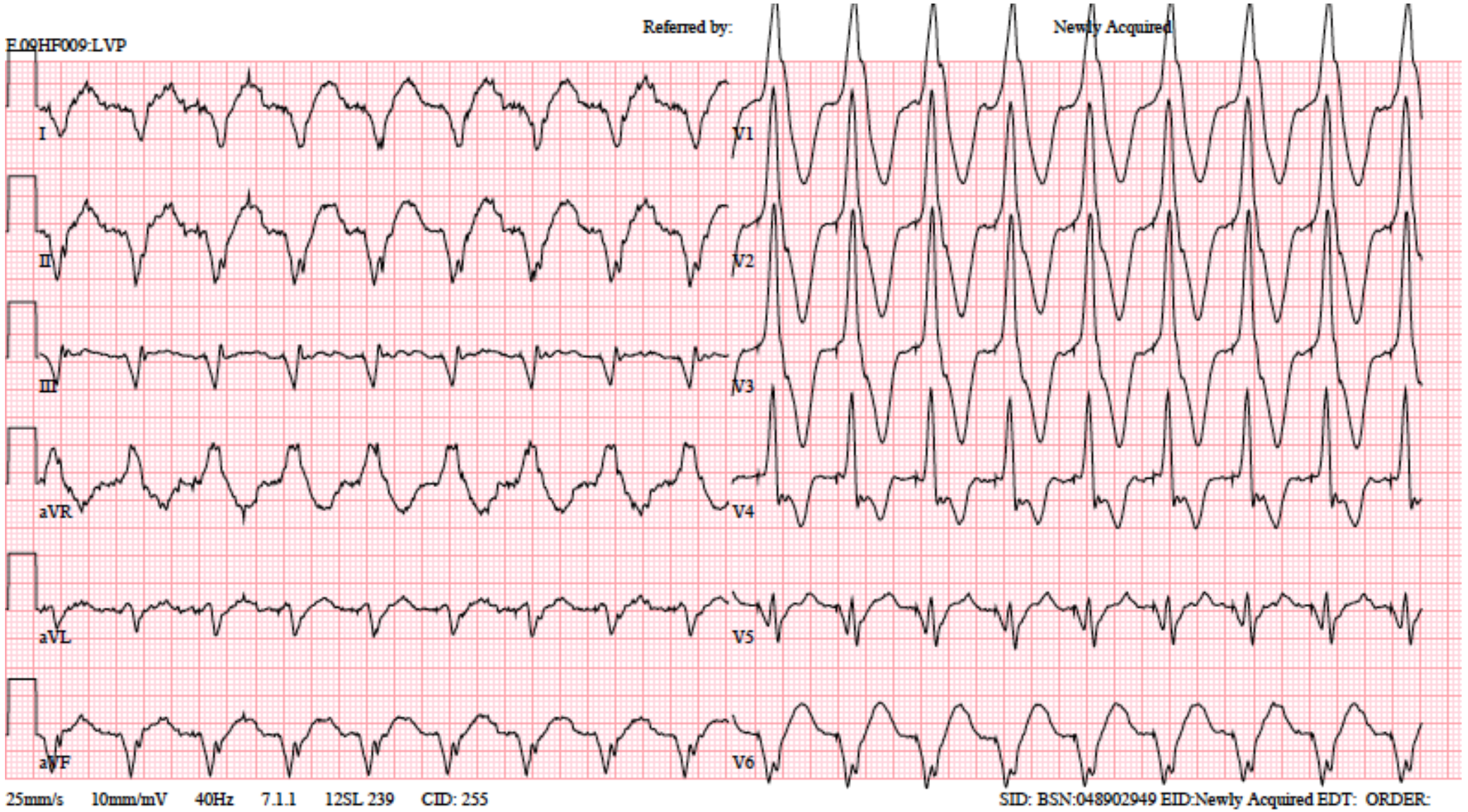
VVI LV Pacing: Where is the Lead?

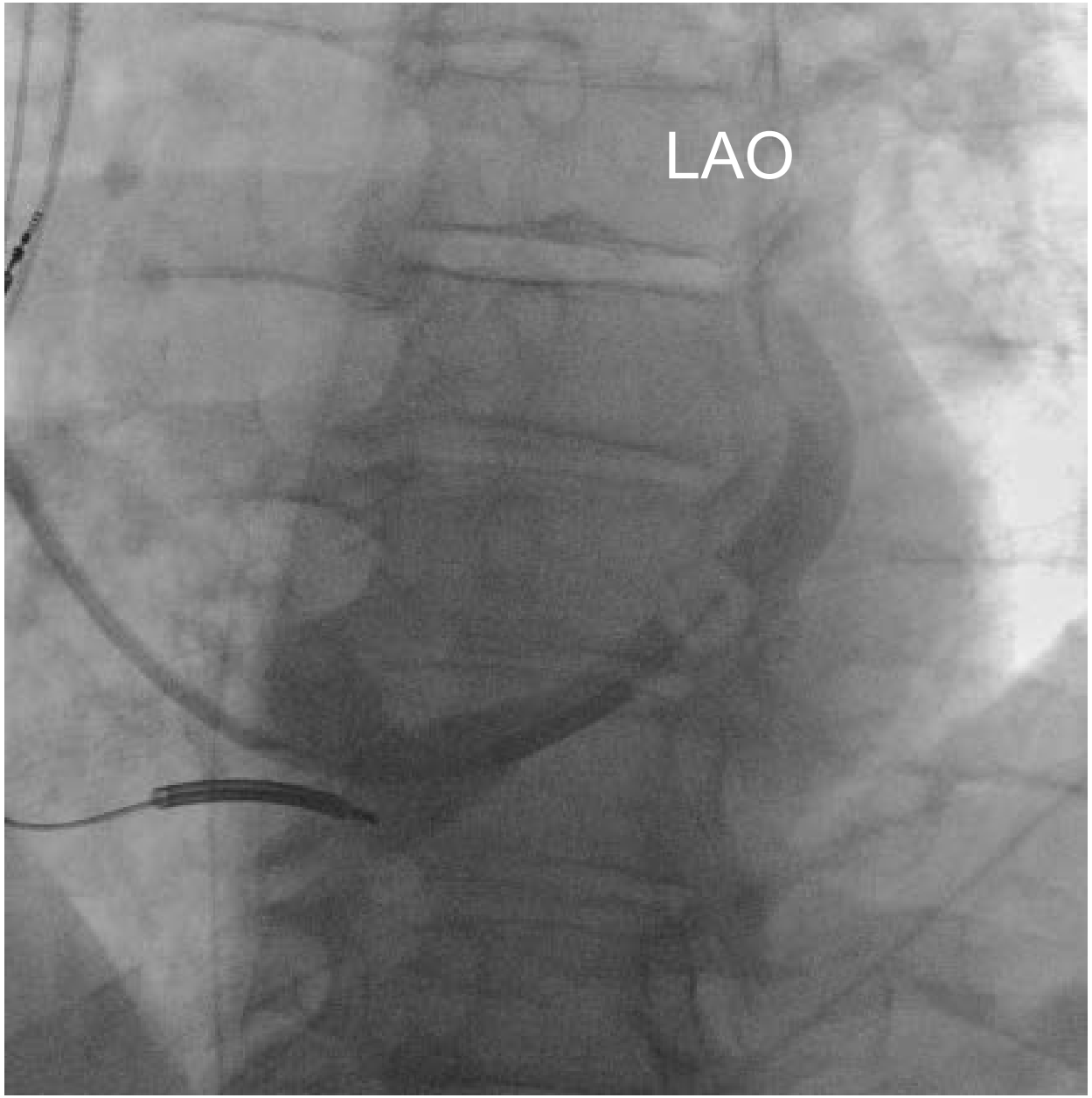


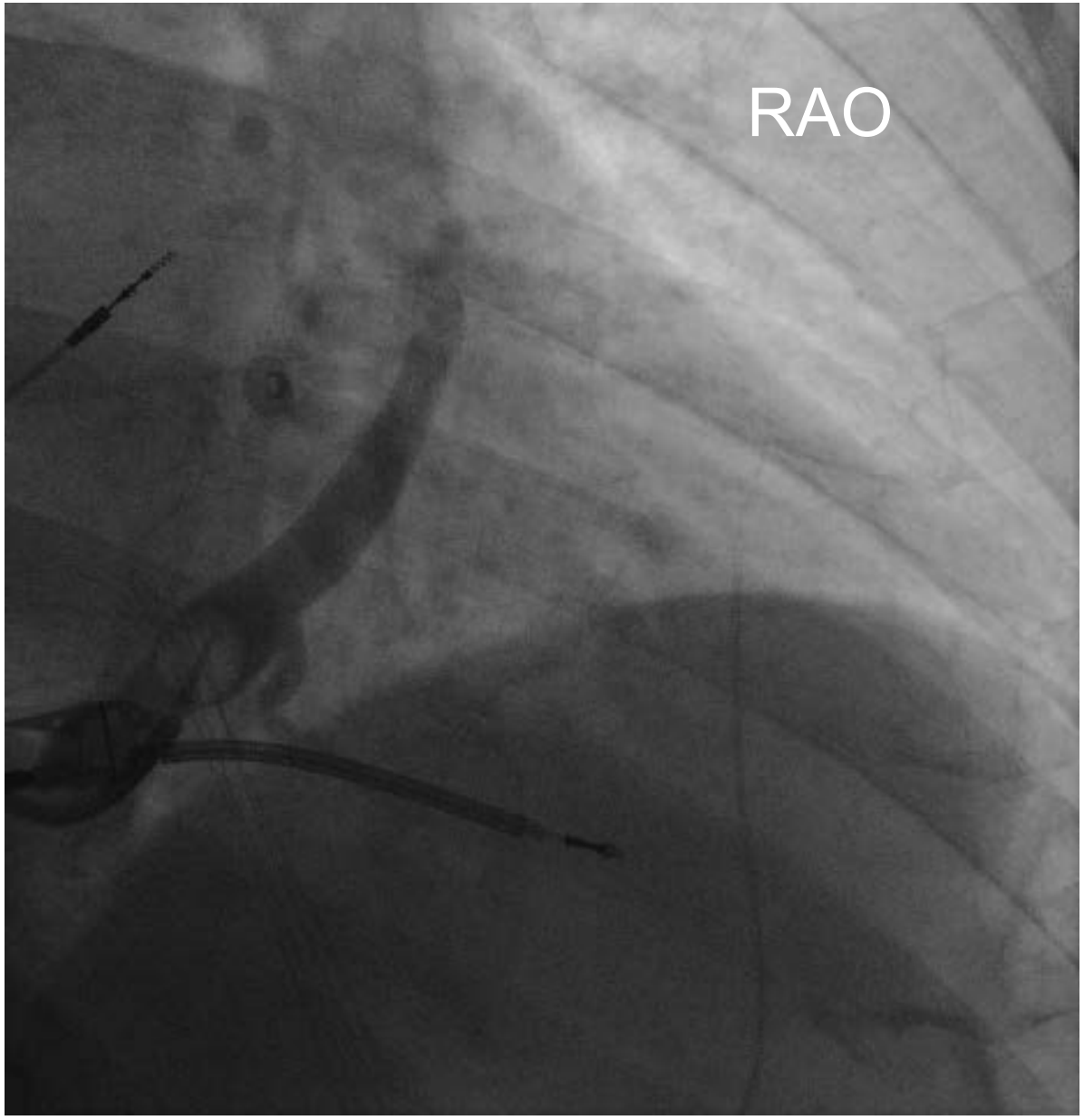


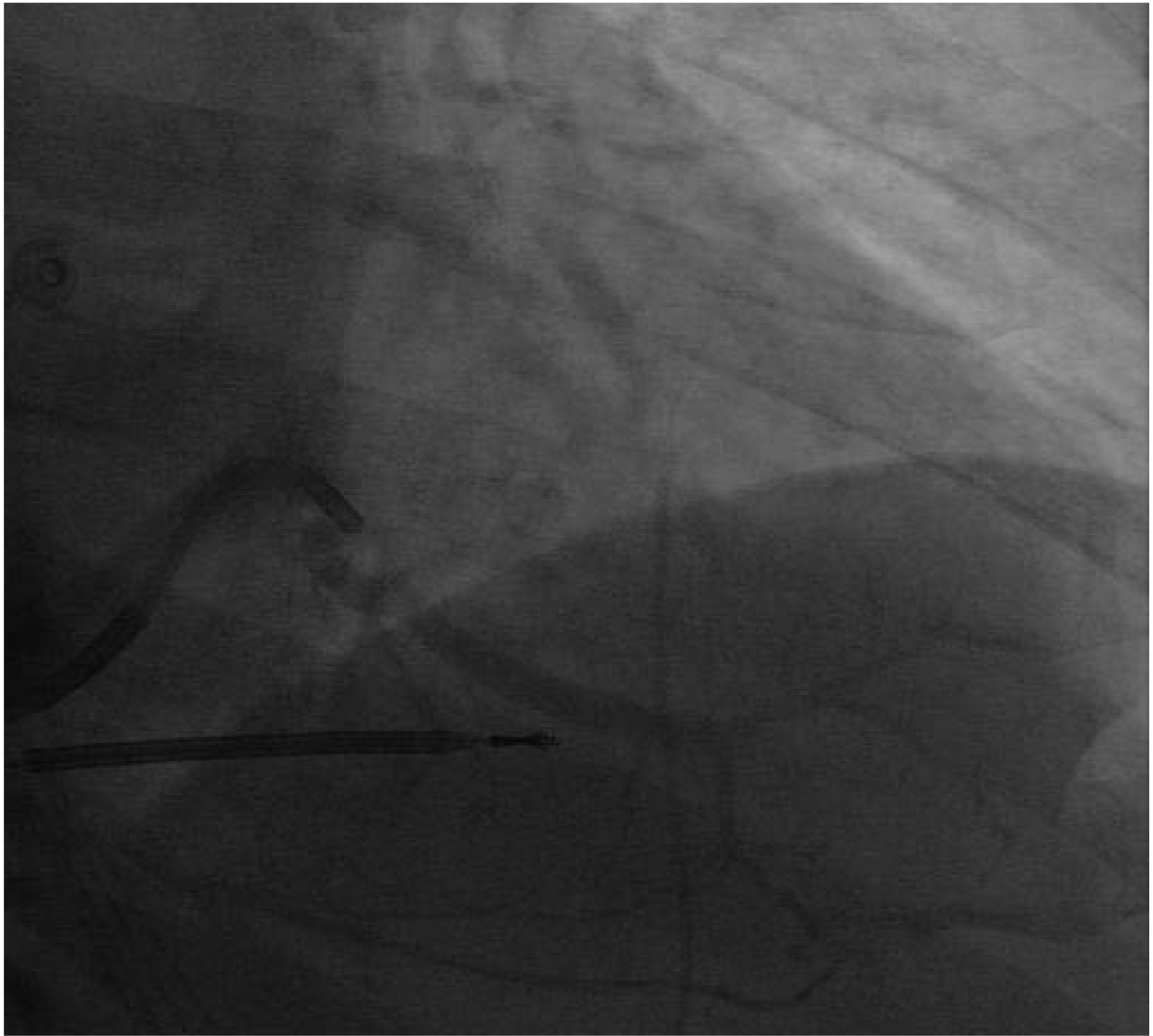
RV apex Pacing

Where is the LV lead? (LV Pacing)

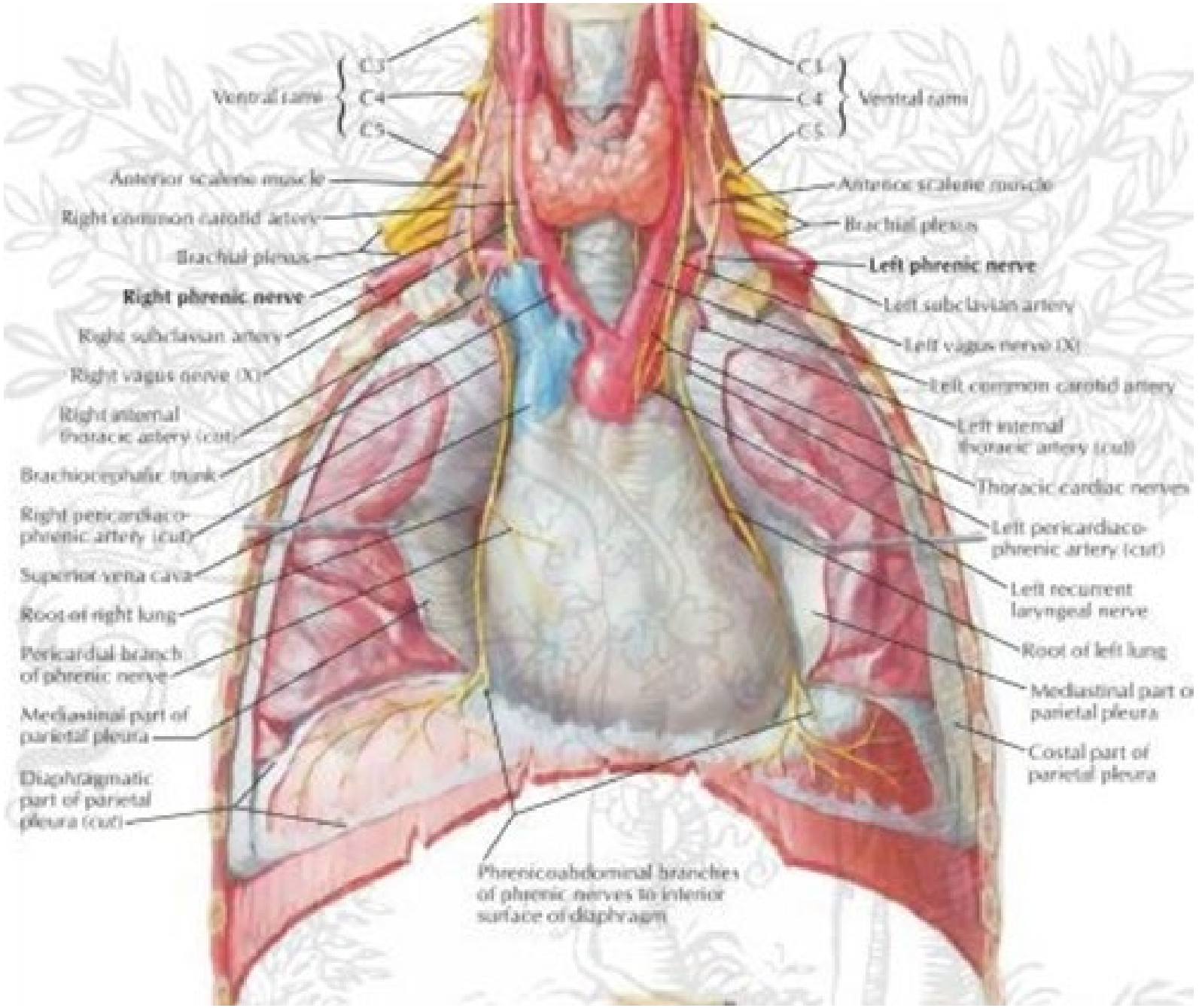



LAO


RAO



Phrenic Nerve



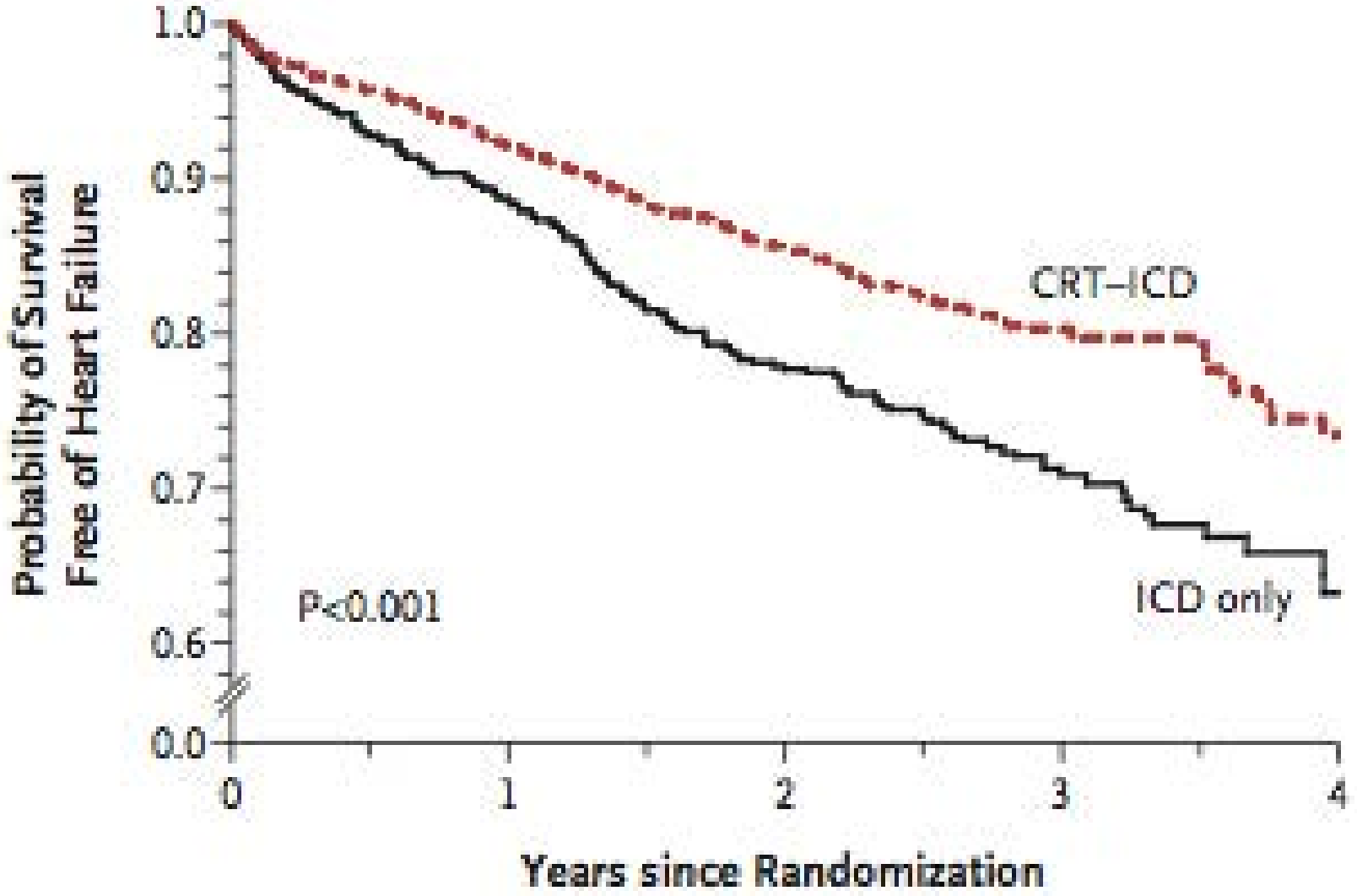
LANDMARK Trial: MADIT-CRT

ESTABLISHED IN 1812 OCTOBER 1, 2009 VOL. 361 NO. 14

Cardiac-Resynchronization Therapy for the Prevention of Heart-Failure Events

Arthur J. Moss, M.D., W. Jackson Hall, Ph.D., David S. Cannom, M.D., Helmut Klein, M.D., Mary W. Brown, M.S., James P. Daubert, M.D., N.A. Mark Estes III, M.D., Elyse Foster, M.D., Henry Greenberg, M.D., Steven L. Higgins, M.D., Marc A. Pfeffer, M.D., Ph.D., Scott D. Solomon, M.D., David Wilber, M.D., and Wojciech Zareba, M.D., Ph.D., for the MADIT-CRT Trial Investigators*

Variable	ICD-Only Group (N=731)	CRT-ICD Group (N=1089)
Age — yr	64±11	65±11
Male sex — no. (%)	553 (75.6)	814 (74.7)
Race — no./total no. (%)†		
White	657/724 (90.7)	979/1083 (90.4)
Black	56/724 (7.7)	87/1083 (8.0)
Other	11/724 (1.5)	17/1083 (1.6)
Cardiac history — no. (%)		
Ischemic heart disease		
NYHA class I	113 (15.5)	152 (14.0)
NYHA class II	288 (39.4)	446 (41.0)
Nonischemic heart disease		
NYHA class II	330 (45.1)	491 (45.1)
NYHA class III or IV >3 mo before enrollment — no. (%)	73 (10.0)	109 (10.0)
Left bundle-branch block — no./total no. (%)	520/729 (71.3)	761/1088 (69.9)
Right bundle-branch block — no./total no. (%)	92/729 (12.6)	136/1088 (12.5)
QRS duration ≥150 msec — no. (%)	476 (65.1)	699 (64.2)
Left ventricular ejection fraction	0.24±0.05	0.24±0.05


Probability of Survival
Free of Heart Failure
1.0
0.9
0.8
0.7
0.6
0.0
CRT–ICD
ICD only
P<0.001
0
1
2
3
4
Years since Randomization

Echocardiographic Dysynchrony Parameters

Table 7. Logistic Regression Analysis for Predefined Predictors of Volume Responders

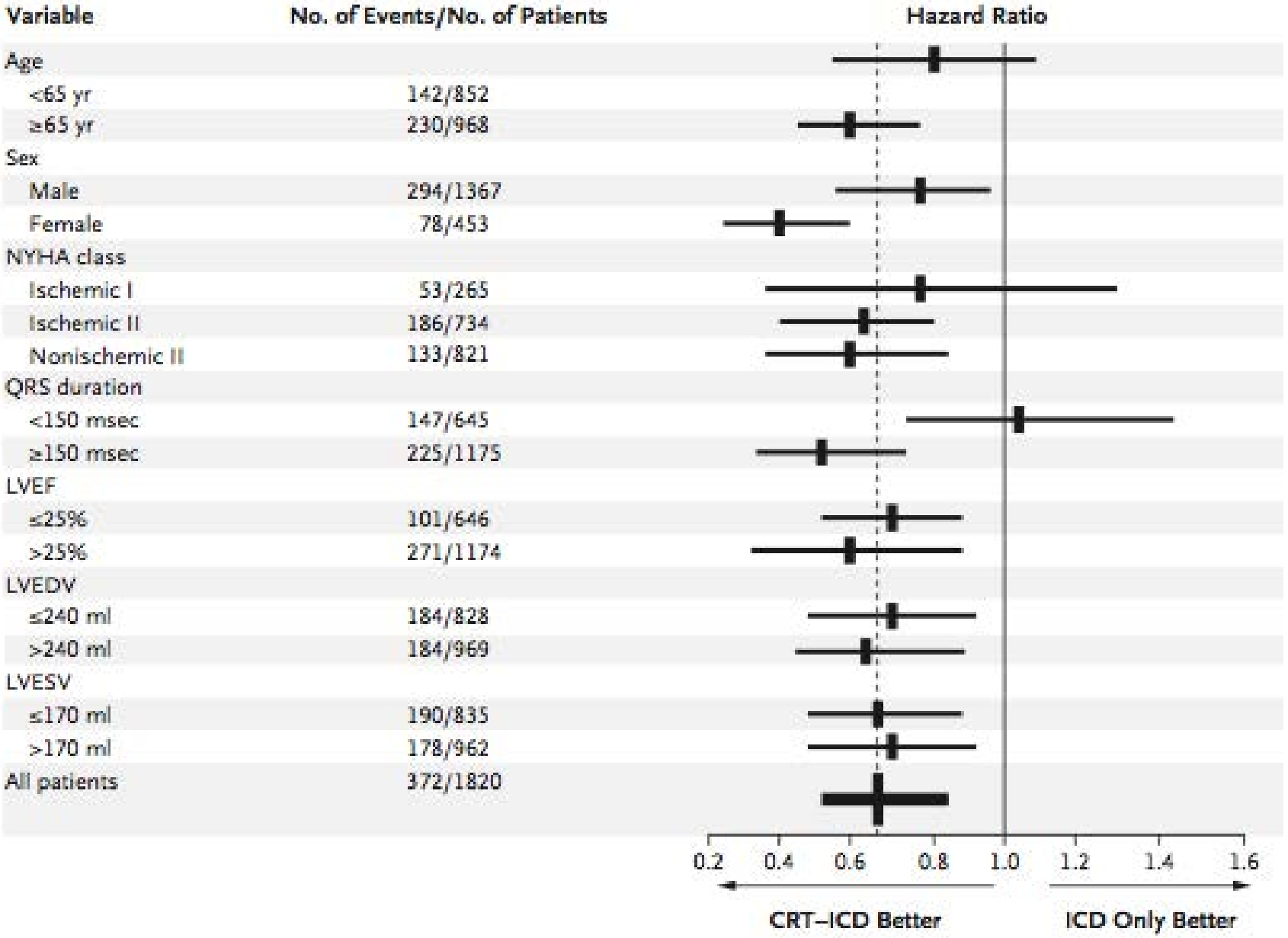
Characteristic	Univariate		Multivariate model 1		Multivariate model 2	
	HR (95%CI)	P value	HR (95%CI)	P value	HR (95%CI)	P value
Age, years	1.02 (0.99–1.04)	0.05	1.01 (0.89–1.05)	0.26	1.02 (0.99–1.05)	0.16
LBBB	2.72 (1.51–4.89)	<0.001	2.91 (1.47–5.79)	0.02	2.58 (1.34–4.98)	0.004
Baseline BNP >350 pg/ml	1.34 (0.76–2.34)	0.31				
Intravenous inotropes	0.11 (0.03–0.38)	<0.001	0.07 (0.01–0.30)	<0.001	0.11 (0.03–0.43)	<0.001
β-blocker	0.24 (0.10–0.53)	<0.001	0.21 (0.08–0.57)	0.002	0.20 (0.09–0.53)	0.001
SP WMD >130 ms	2.12 (1.16–3.83)	0.01	1.71 (0.86–3.38)	0.12		
Ts (lateral-septal) >65 ms	1.65 (0.93–2.90)	0.08				
Sum of asynchrony >102 ms	0.91 (0.44–1.85)	0.73				
Combined SPWMD and Ts (lateral-septal)	2.53 (1.35–4.79)	0.004			2.19 (1.05–4.56)	0.03

Table 5. Ability of Dyssynchrony Parameters to Predict Volume Responders

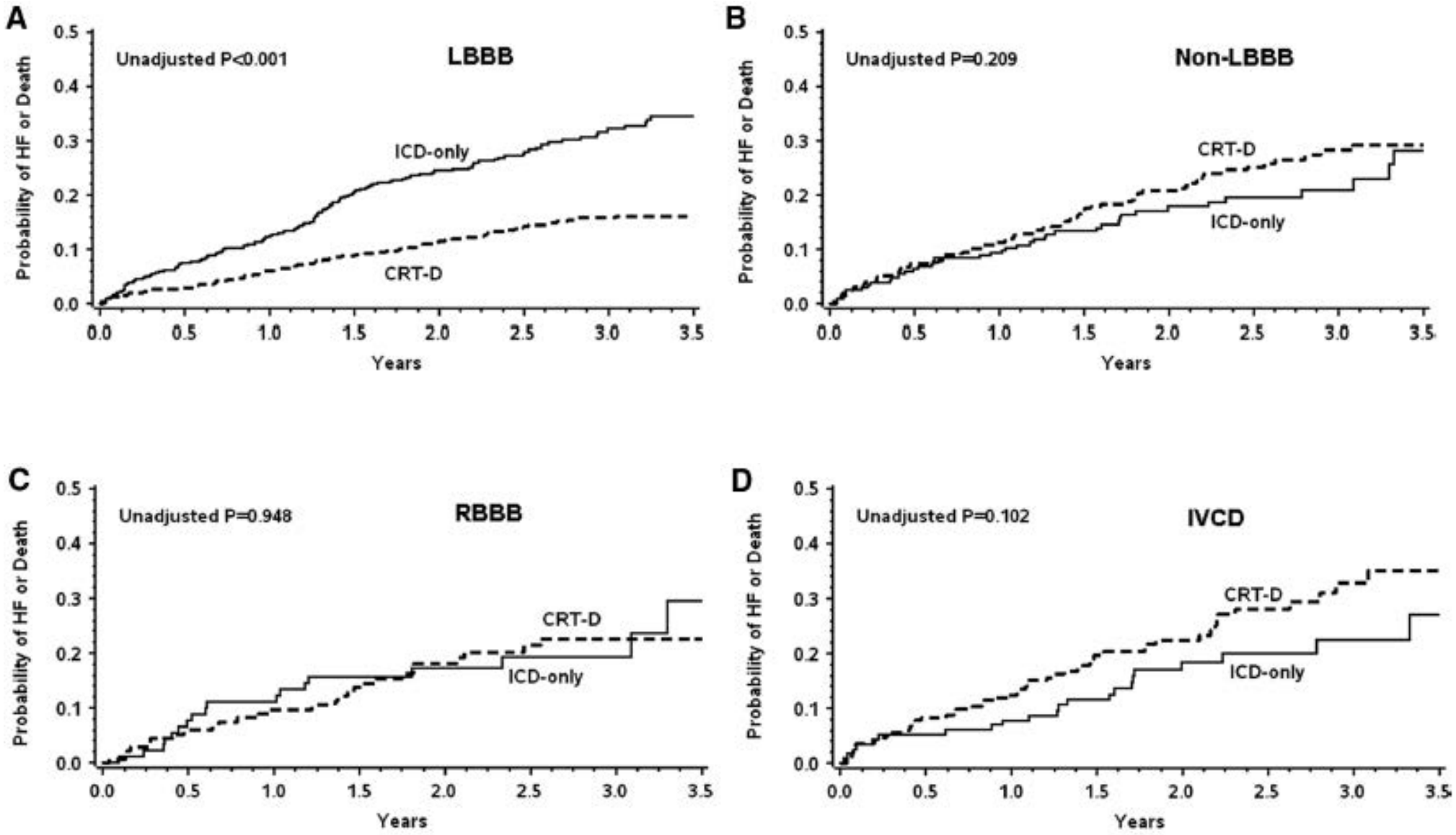
Dyssynchrony parameters	Sensitivity	Specificity	Positive predictive value	AUC
SPWMD >130 ms	0.61	0.66	0.64	0.68
LV-PEP >140 ms	0.58	0.64	0.62	0.59
IMD >40 ms	0.50	0.62	0.61	0.55
DFT/RR <40%	0.70	0.46	0.56	0.60
12Ts-SD >34.4 ms	0.68	0.41	0.54	0.62
Ts (lateral-septal) >65 ms	0.49	0.66	0.59	0.60
Sum of asynchrony >102 ms	0.54	0.62	0.59	0.60

Abbreviations as in Tables 1,2,5.

Seo, Circulation J 2011



Subanalyze MADIT-CRT Trial



Zereba, Circulation, 2011

European Heart Journal (2013) **34**, 2281–2329
doi:10.1093/eurheartj/eht150

ESC GUIDELINES

2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy

The Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA).

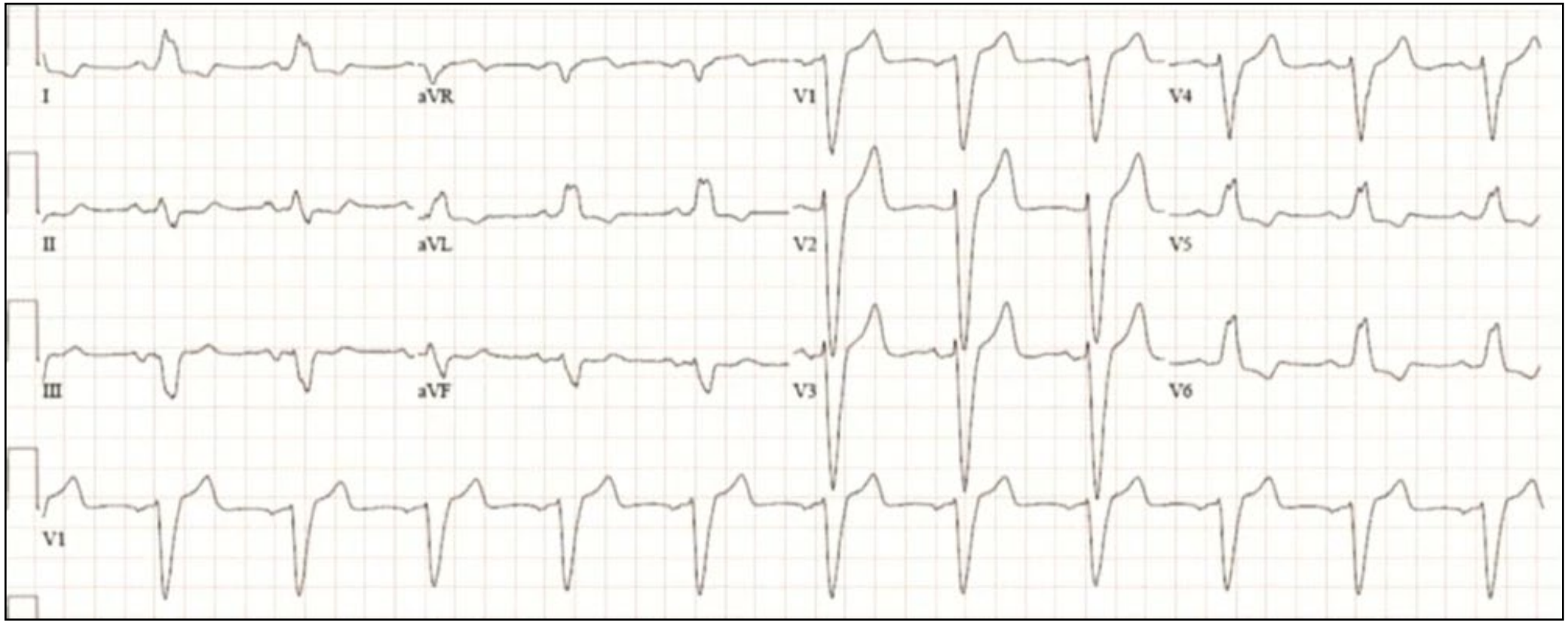
1) LBBB with QRS duration >150 ms. CRT is recommended in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment.[d]	I	A
2) LBBB with QRS duration 120–150 ms. CRT is recommended in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment.[d]	I	B
3) Non-LBBB with QRS duration >150 ms. CRT should be considered in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment.[d]	IIa	B
4) Non-LBBB with QRS duration 120–150 ms. CRT may be considered in chronic HF patients and LVEF ≤35% who remain in NYHA functional class II, III and ambulatory IV despite adequate medical treatment.[d]	IIb	B
5) CRT in patients with chronic HF with QRS duration <120 ms is not recommended.	III	B

1) The goal of CRT should be to achieve BiV pacing as close to 100% as possible since the survival benefit and reduction in hospitalization are strongly associated with an increasing percentage of BiV pacing.	IIa	B
2) Apical position of the LV lead should be avoided when possible.	IIa	B
3) LV lead placement may be targeted at the latest activated LV segment.	IIb	B

1) Patients with HF, wide QRS and reduced LVEF:		
1A) CRT should be considered in chronic HF patients, intrinsic QRS ≥120 ms and LVEF ≤35% who remain in NYHA functional class III and ambulatory IV despite adequate medical treatment[d], provided that a BiV pacing as close to 100% as possible can be achieved.	IIa	B
1B) AV junction ablation should be added in case of incomplete BiV pacing.	IIa	B
2) Patients with uncontrolled heart rate who are candidates for AV junction ablation. CRT should be considered in patients with reduced LVEF who are candidates for AV junction ablation for rate control.	IIa	B

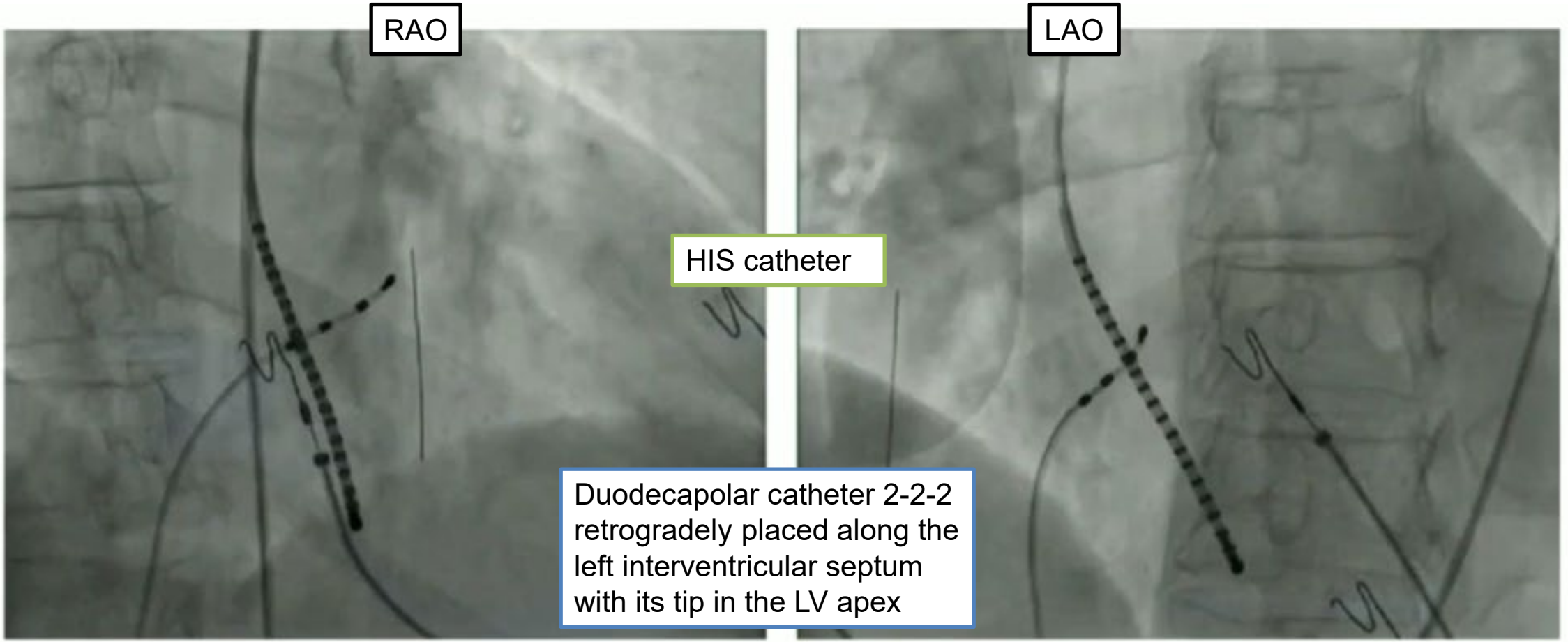
1. Sélection du patient pour la thérapie de resynchronisation cardiaque

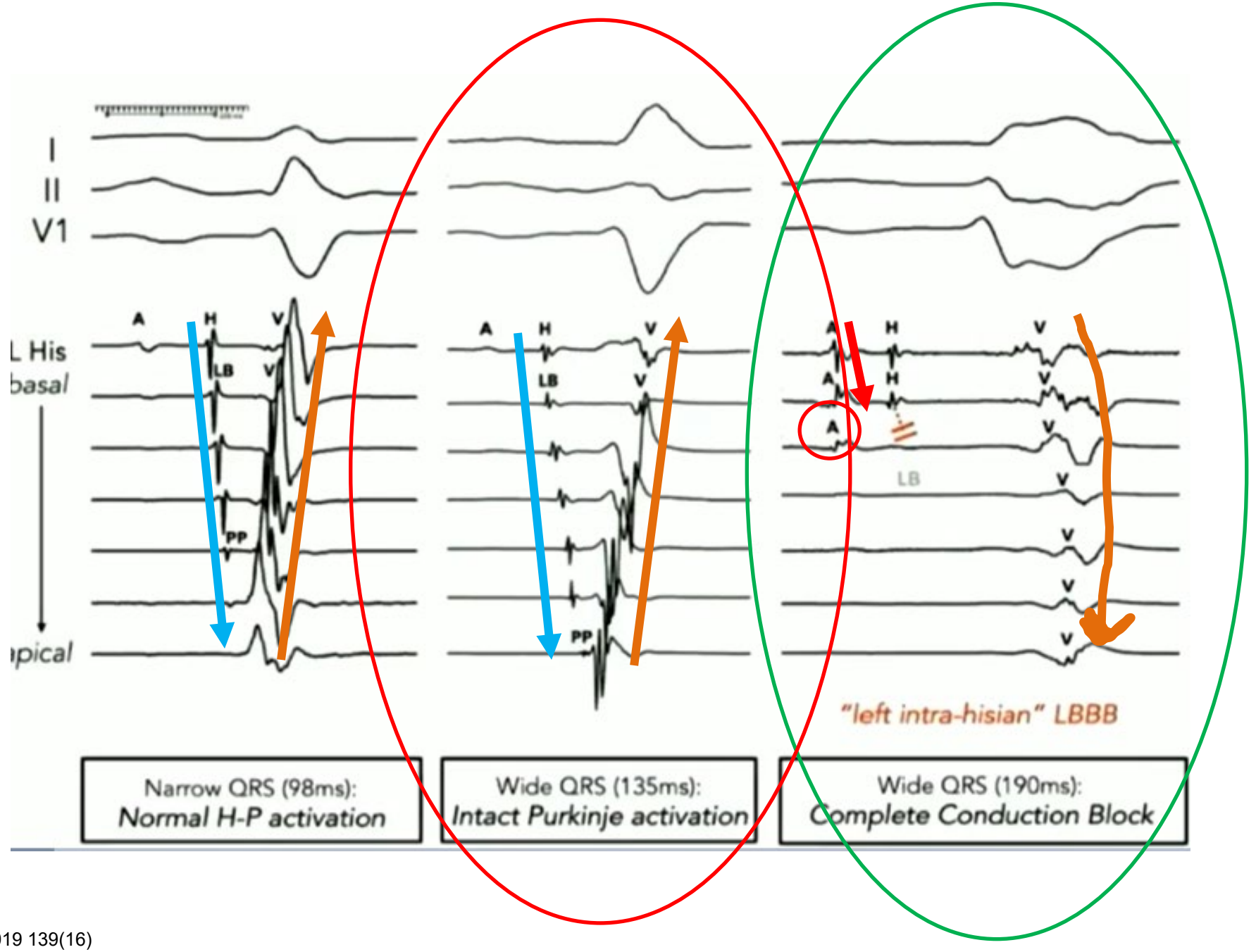
Quelle est la définition d'un bloc de branche gauche?



QRS >= 120ms, notched/slurred R wave in I aVL V5 V6
Absent q wave in I V5 V6
R peak time >60ms in V5 and V6
ST and T wave usually opposite to the QRS

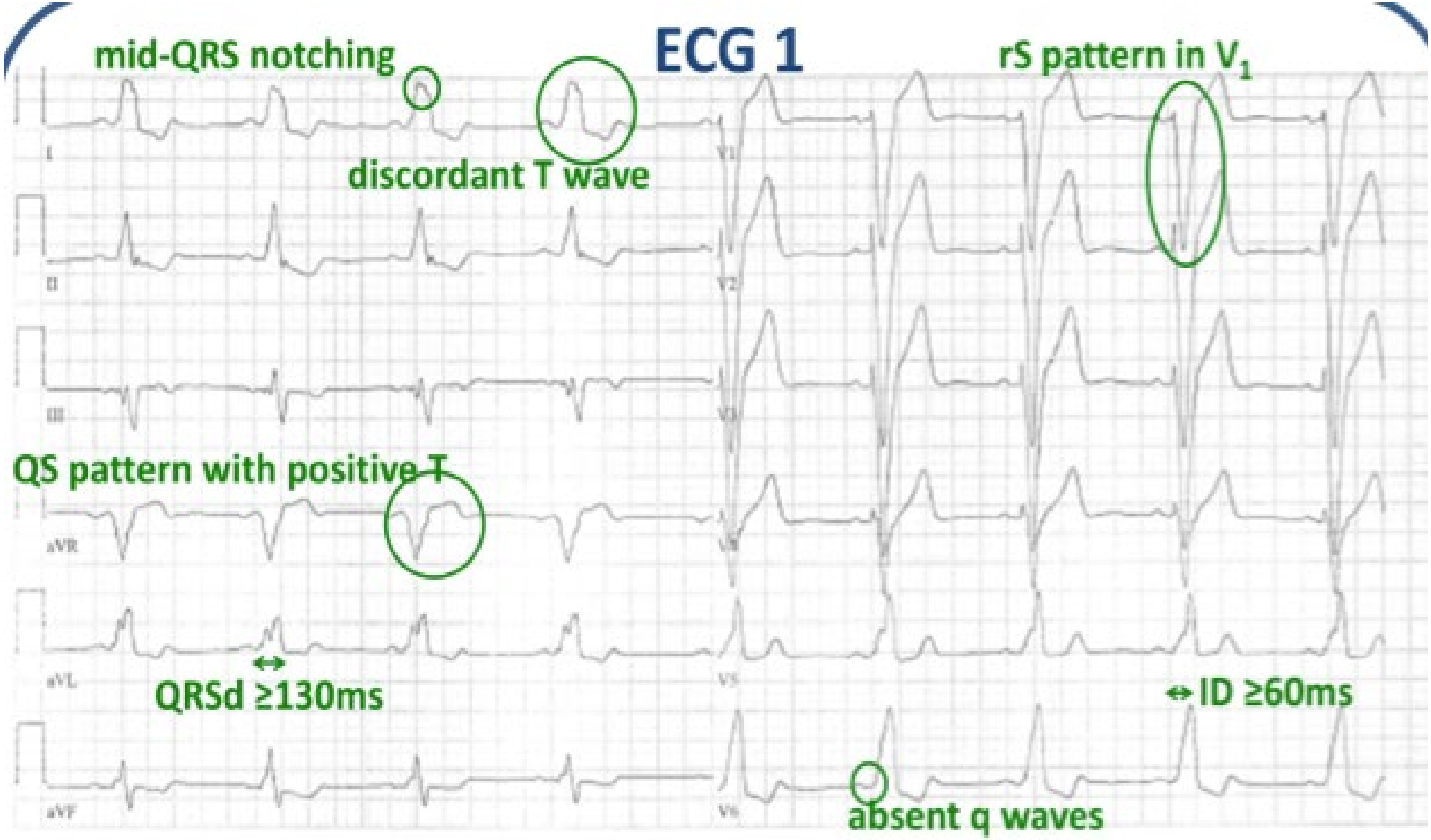
LBBB pattern does not necessarily mean LBBB

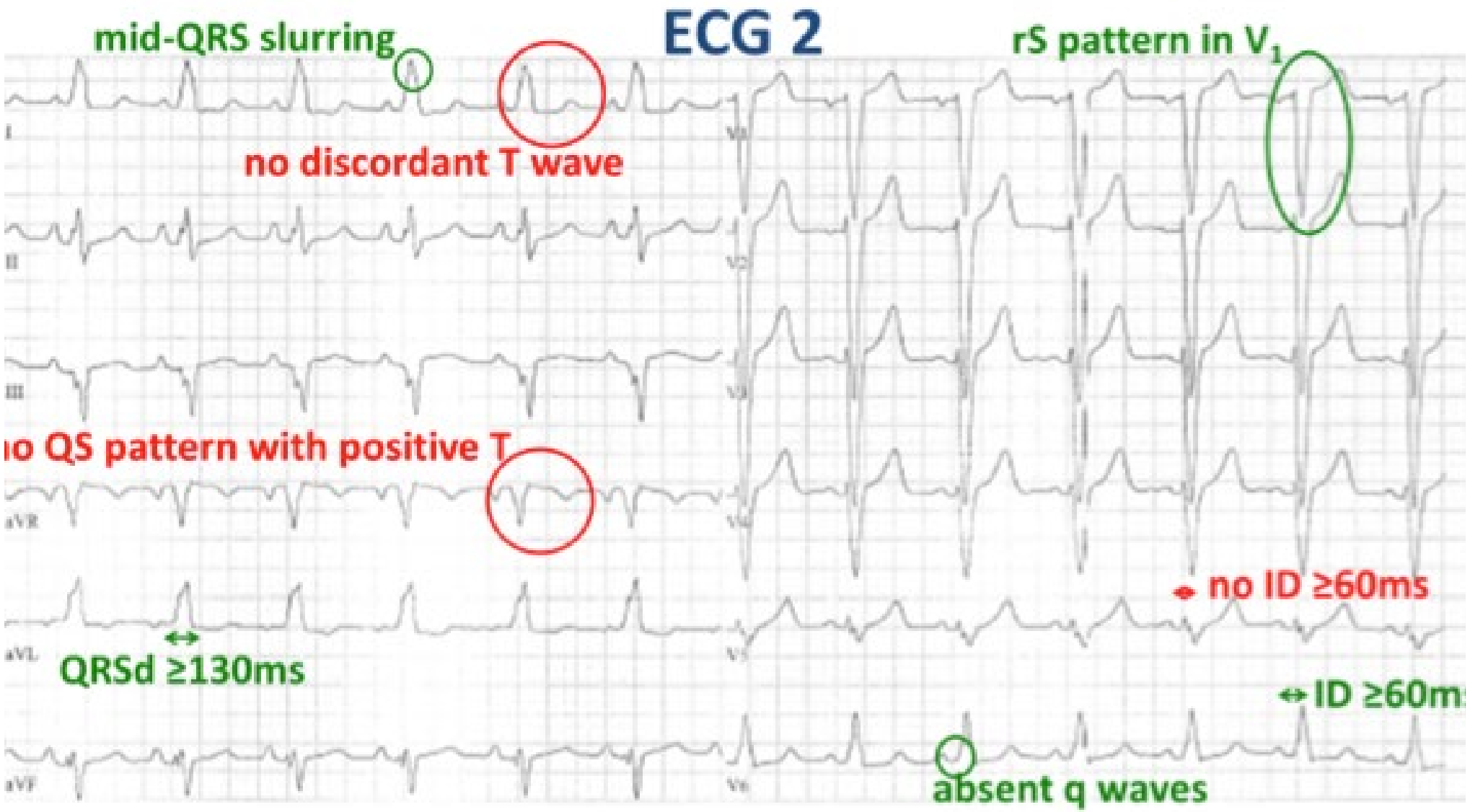

RAO
LAO
HIS catheter
Duodecapolar catheter 2-2-2 retrogradely placed along the left interventricular septum with its tip in the LV apex



Upadhyaya et al. Circulation 2019 139(16)

ECG parameter for complete LBBB	ESC	AHA	Strauss	MADIT	REVERSE
QRS duration ≥	120	120	□130 □140	130	120
QS or rS in V_1	yes	yes	yes	yes	yes
Positive T in V_1	yes	no	no	no	no
Normal ID R in V_1-V_3	no	yes	no	no	no
ID R in V_5 ≥60ms	no	yes	no	no	no
ID R in V_6 ≥60ms	yes	yes	no	no	no
ID R in I ≥60ms	yes	no	no	no	no
Notch-/slurred R in I, aVL and V_5-V_6	no	yes	no	no	no
Mid-QRS notch-/slurring in ≥2 leads of V_1-V_2, V_5-V_6, I, aVL	no	no	yes	no	no
RS pattern allowed in V_5-V_6	no	yes	yes	yes	yes
Absent q in V_5-V_6	no	yes	no	yes	yes
Absent q in I	no	yes	no	no	no
QS with positive T in aVR	yes	no	no	no	no
Usually discordant T	yes	yes	no	no	no

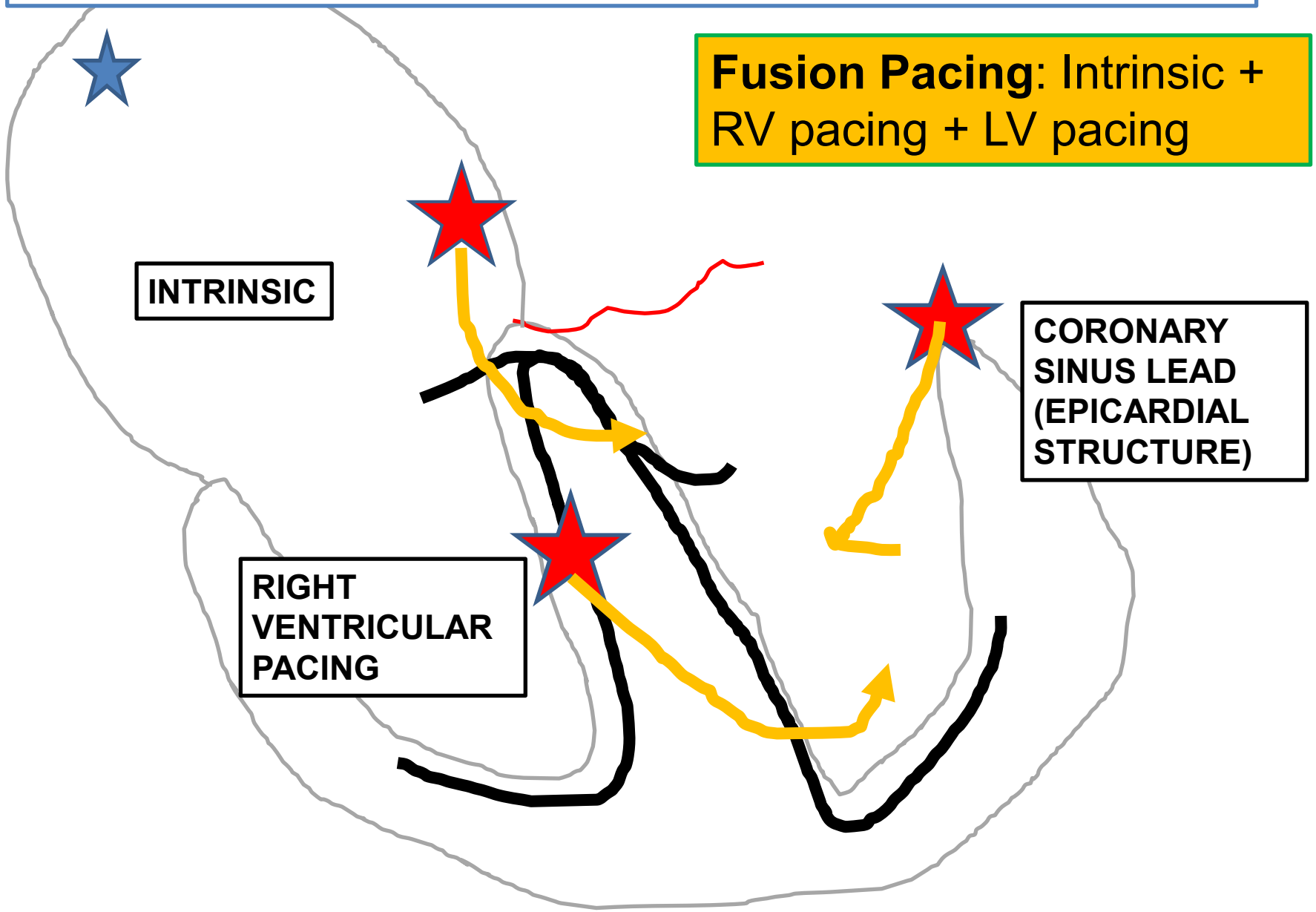

ECG 1
mid-QRS notching
discordant T wave
rS pattern in V1
QS pattern with positive T
QRSd ≥130ms
ID ≥60ms
absent q waves

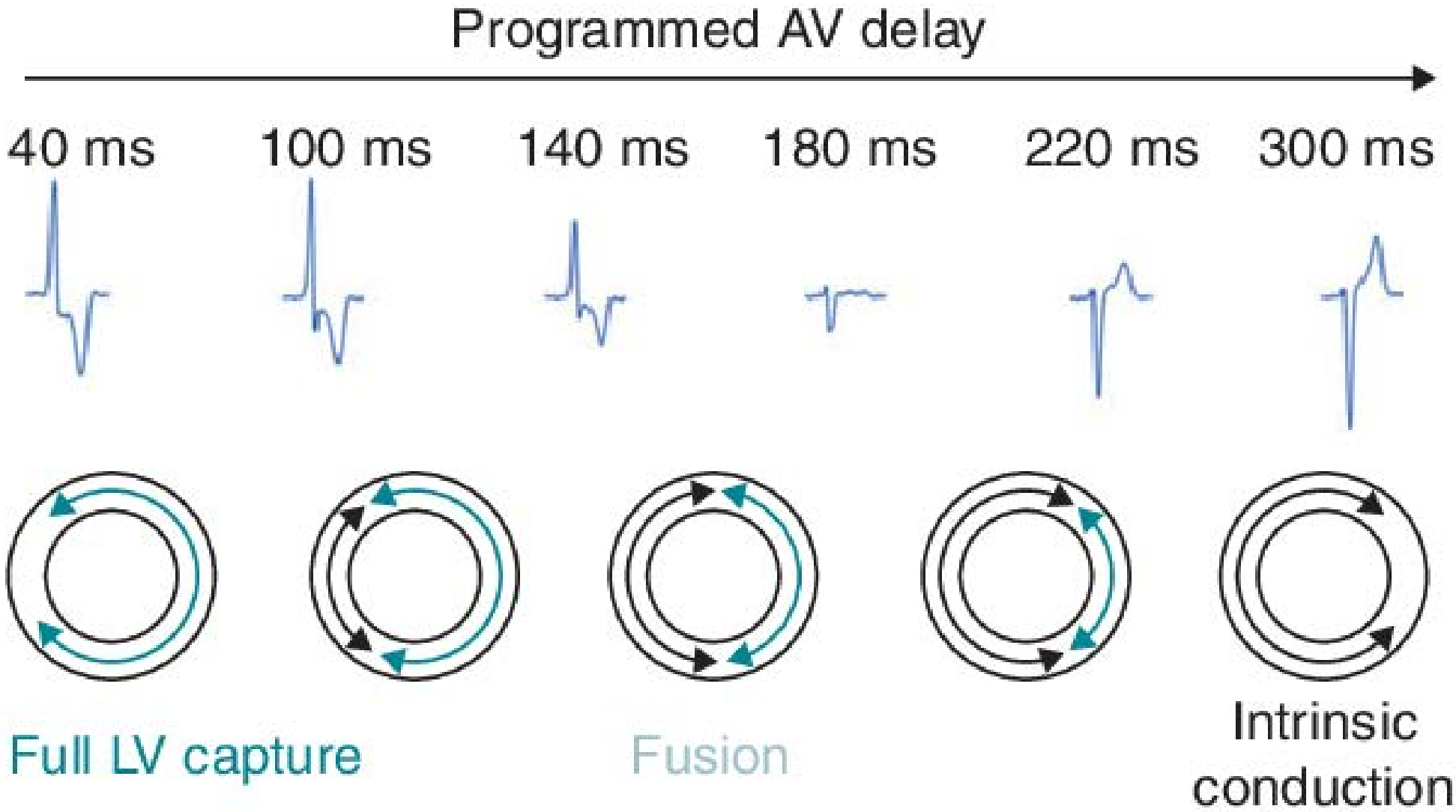

ECG 2
mid-QRS slurring
no discordant T wave
rS pattern in V1
no QS pattern with positive T
no ID ≥60ms
QRSd ≥130ms
ID ≥60m
absent q waves

2. Sélection du bon appareil

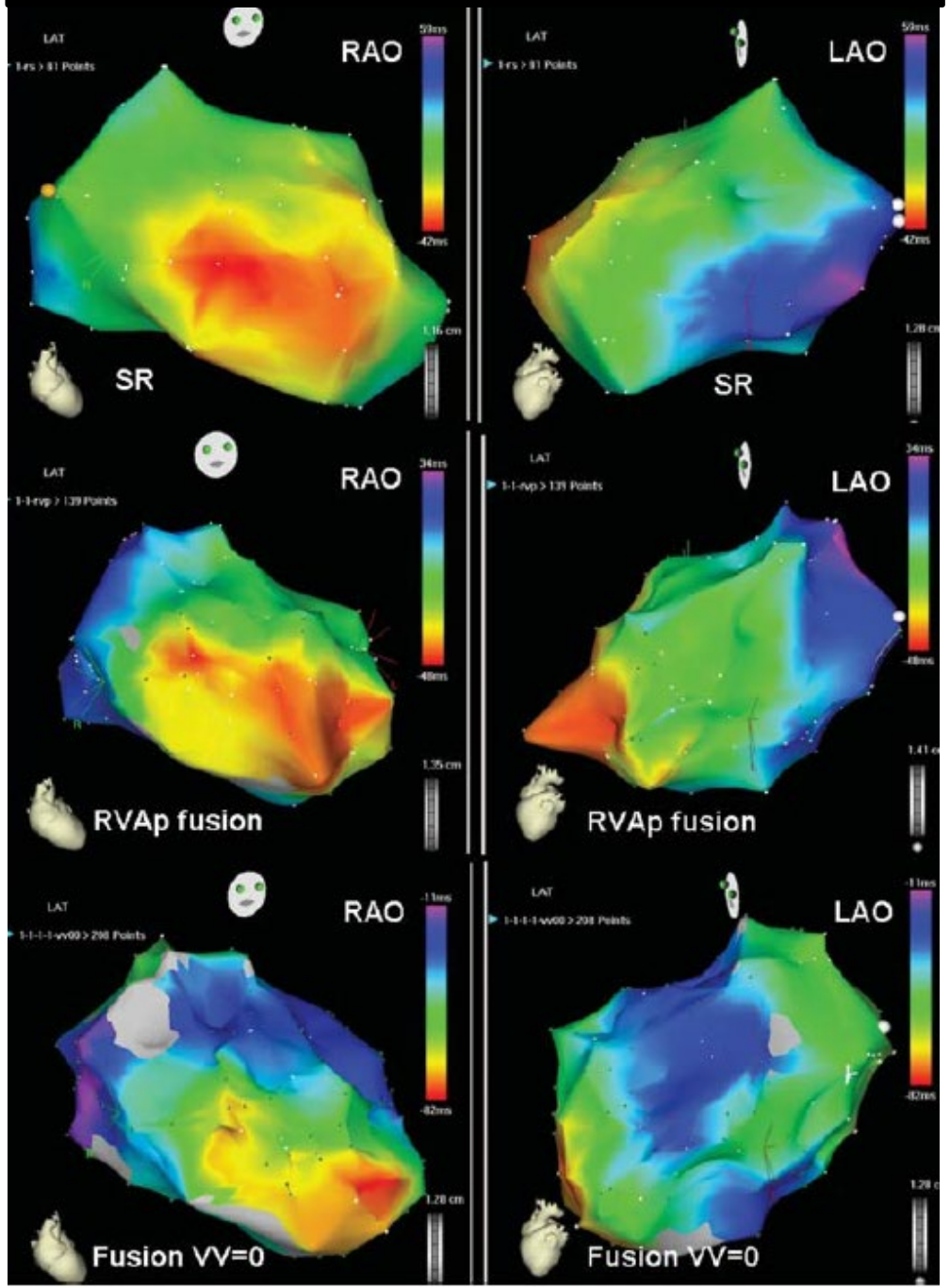
Situation 1. When there is an intrinsic ventricular rhythm

Fusion Pacing: Intrinsic + RV pacing + LV pacing




Programmed AV delay
40 ms
100 ms
140 ms
180 ms
220 ms
300 ms
Full LV capture
Fusion
Intrinsic conduction

AV optimization allowing CRT with fusion.

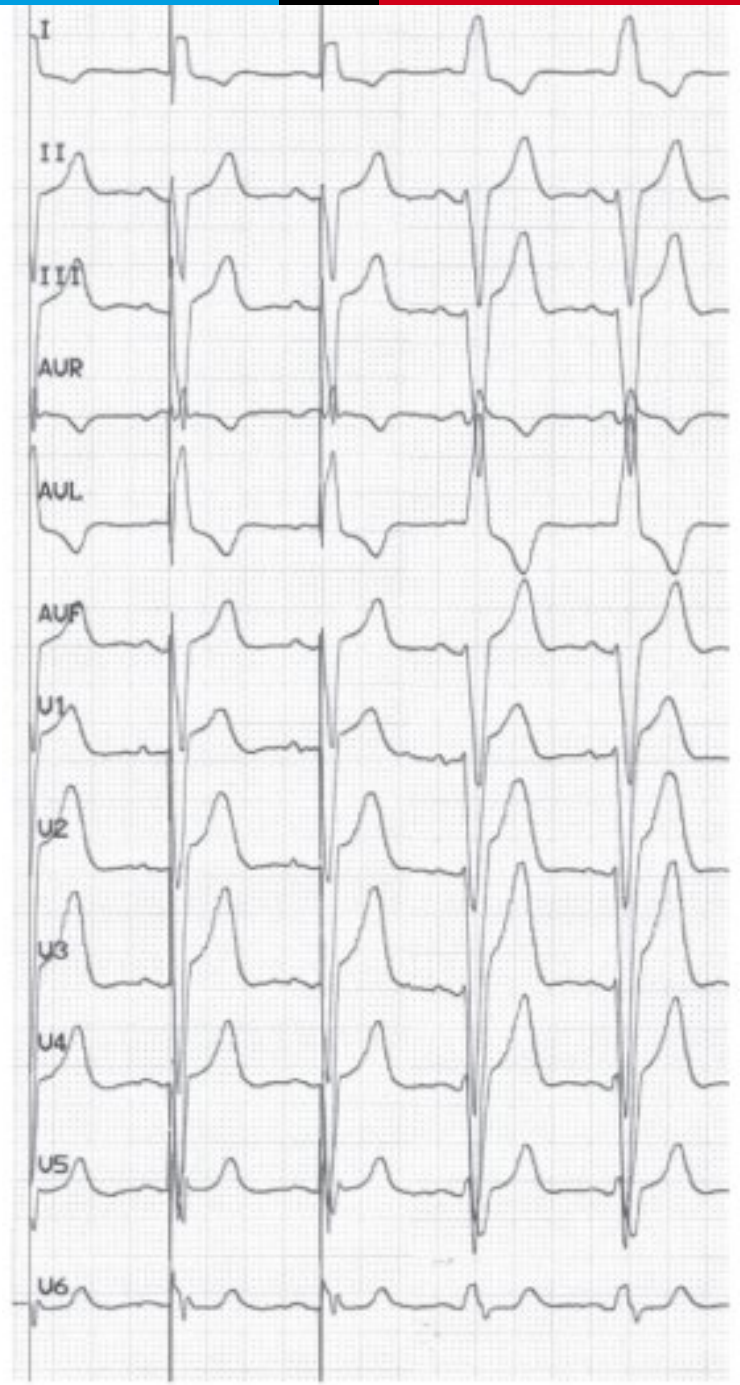


LBBB
Sinus rhythm with mid-septal breakthrough creating a very delayed postero-lateral activation with a long left ventricular activation time >100ms.

Right ventricular apex pacing with fusion with intrinsic depolarization = 2 septal breakthroughs and the anterolateral wall the most delayed, and shortening of the LVAT to 82ms

3 wavefronts: Intrinsic + RV pacing + LV pacing (coronary sinus lead): 2 septal breakthroughs with most delayed activation at intermediate positions and LVAT 71ms

Vatasescu, Brugada..
Europace 2009



- Ventricular fusion pacing using the **VVT (trigger) pacing** mode
- The first two beats show triggered biventricular pacing, synchronized to right ventricular sensing
- Last two beats show intrinsic rhythm with LBBB during temporary inactivation of the algorithm

How and why may an individual's AV delay change?

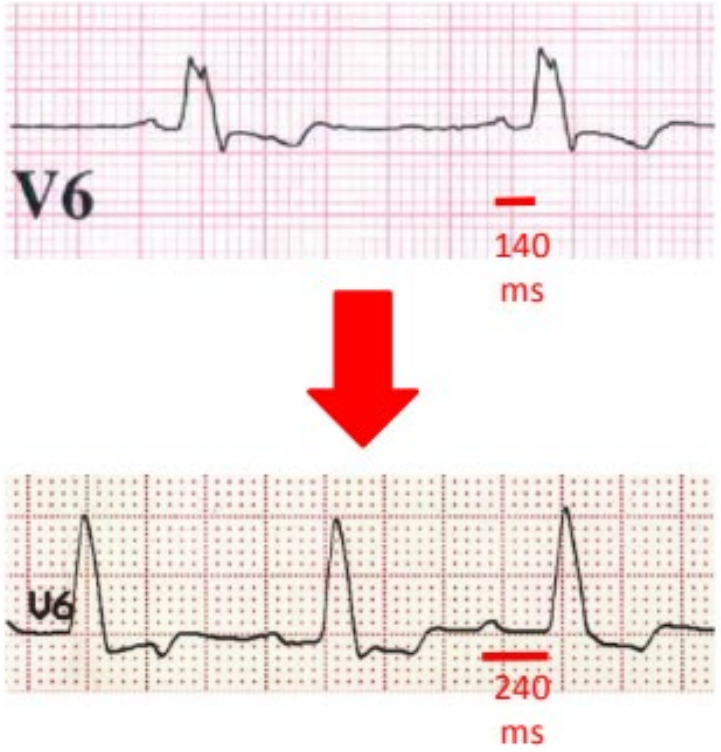
Minutes or hours

Variation in Activity

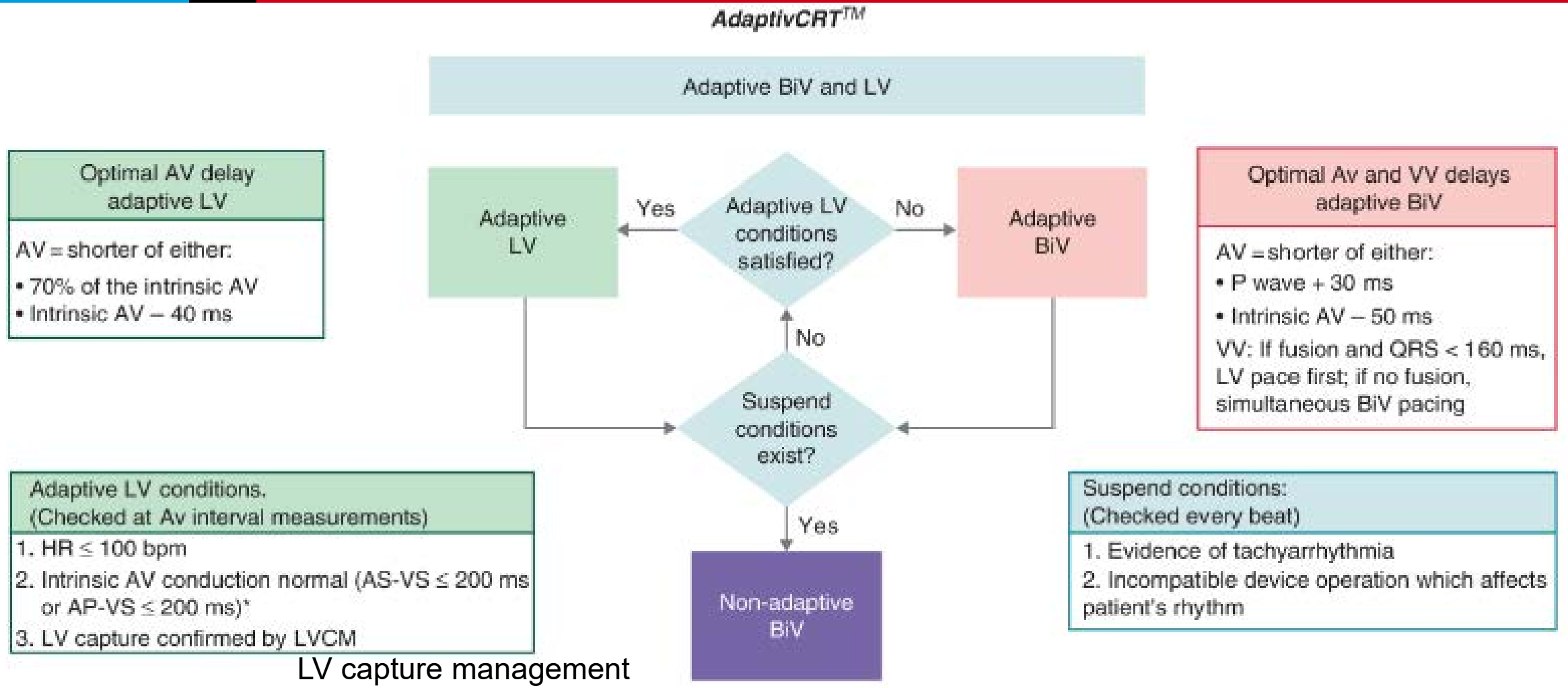
Days or weeks

Change of Meds

Months or years



Disease Progression

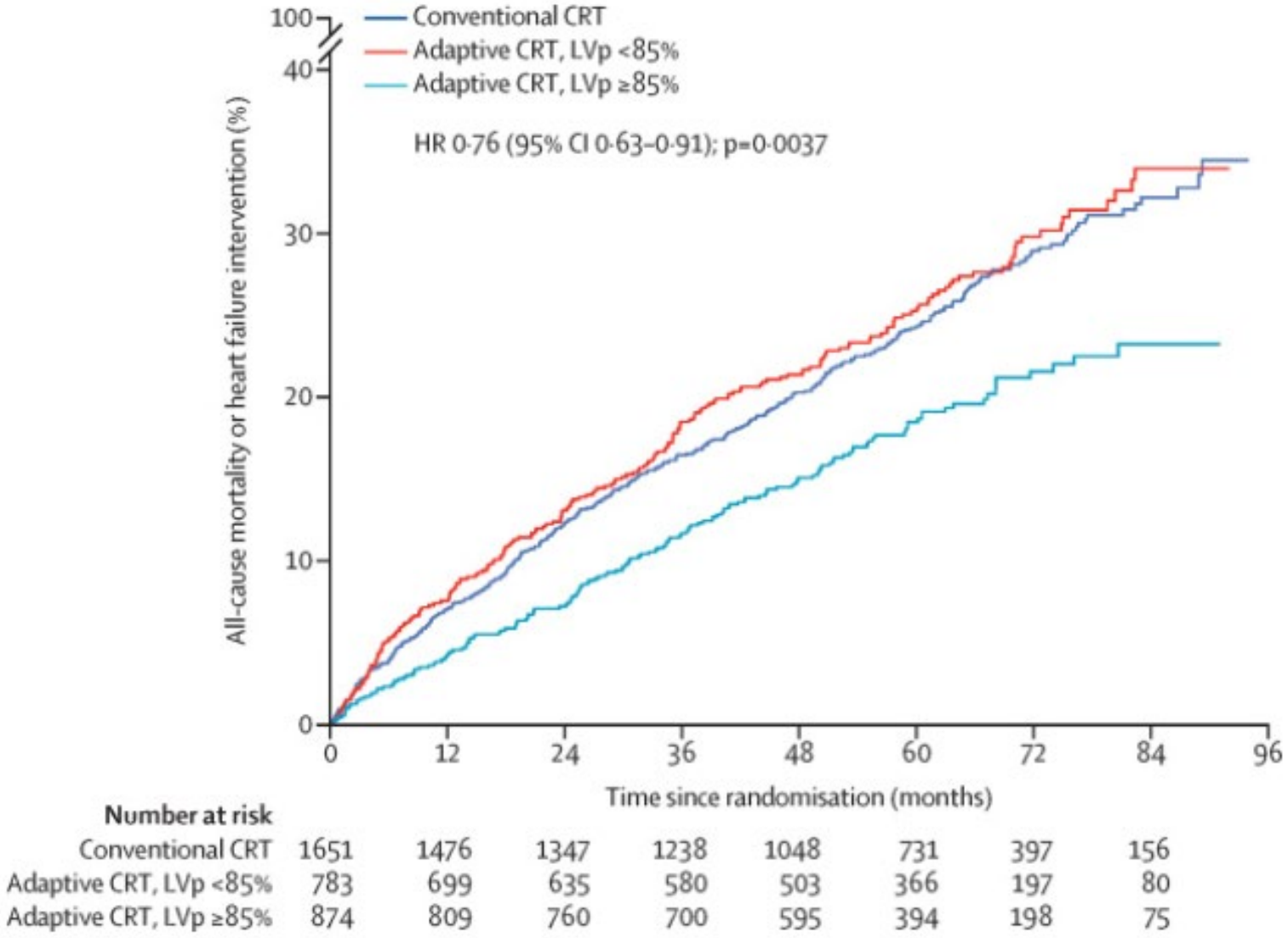


LV capture management

- Adaptive LV algorithm measures the intrinsic AV conduction delay + p wave + QRS duration (far-field EGM) every minute for 1 beat (the QRS is measured 1x per 16 hours where no trigger pacing is performed)
- If intrinsic AV interval is normal and HR <100bpm, then optimized LV pacing with an AV delay of 70% of intrinsic is applied
- This results in fusion and is hemodynamically preferable. Otherwise BiV pacing is performed (pacing occurs 30ms after the end of the p wave, but at least 50ms before intrinsic QRS

Medtronic: Adaptive BiV

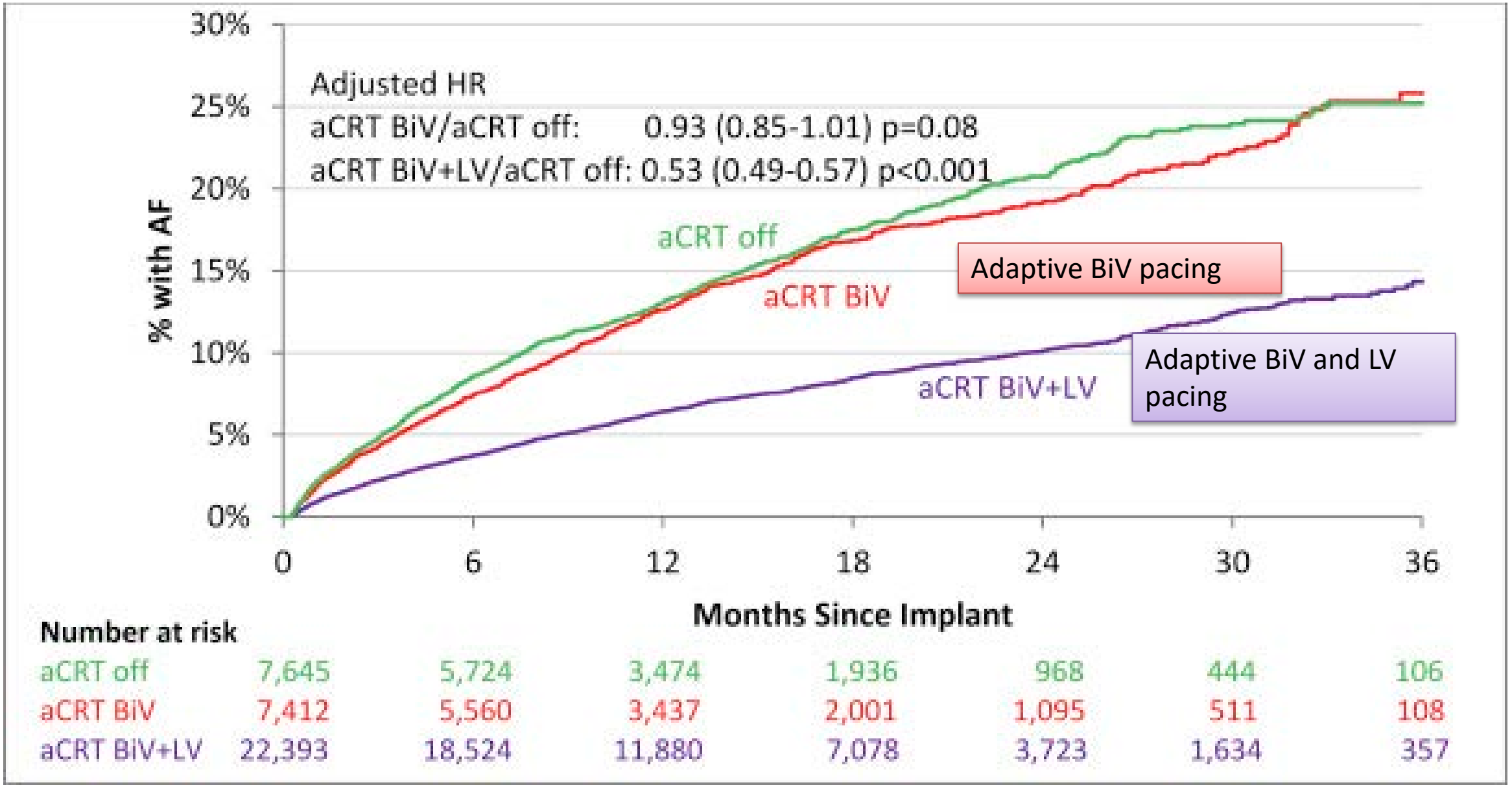
- Used primarily when the PR interval at baseline is <220ms



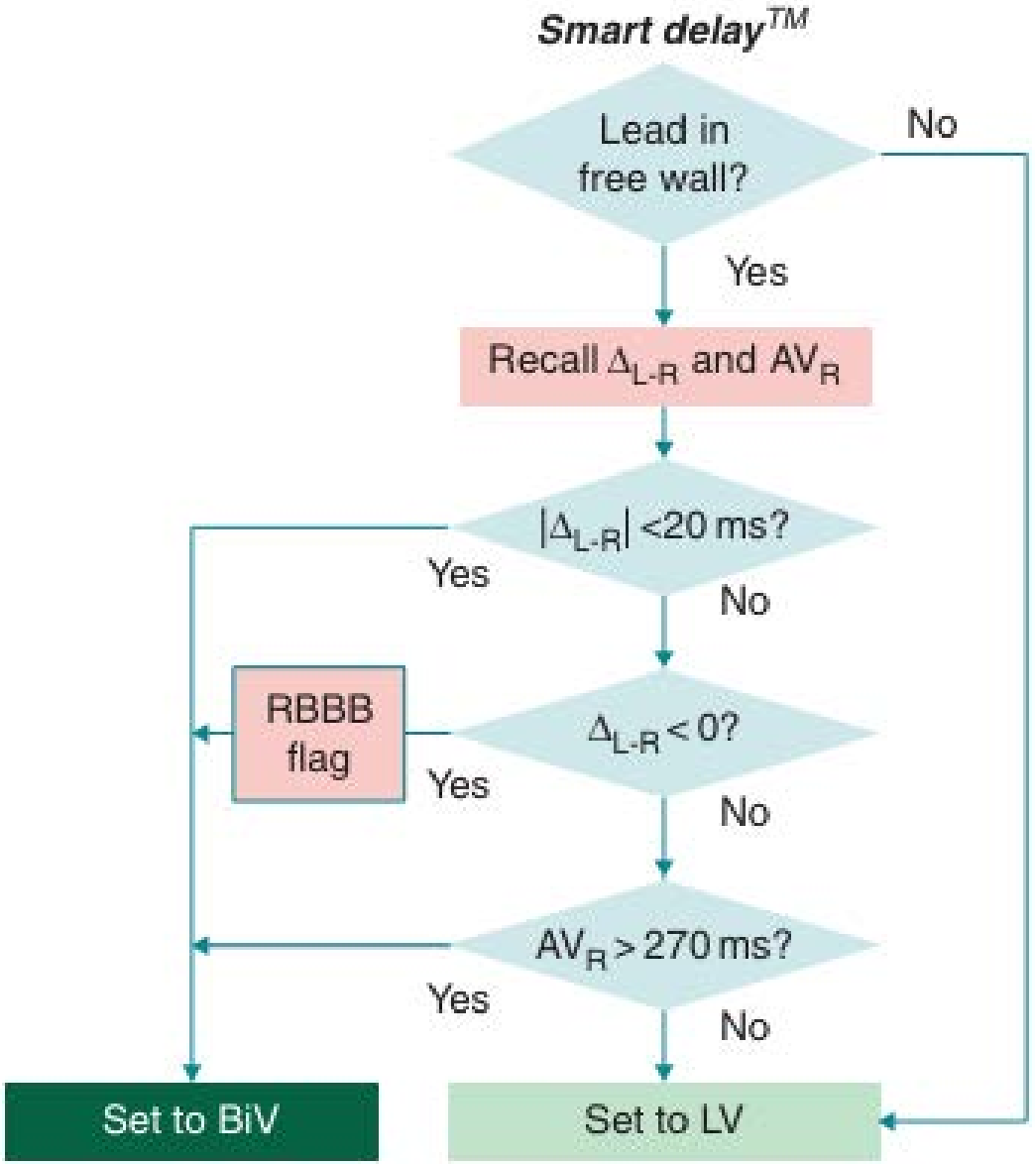
- AdaptResponse Trial 2023
- Adaptive CRT vs conventional CRT
- Patients with Strauss-LBBB (140ms male, 130ms female), EF<=35%, PR <=200ms, and sinus rhythm.
- 3617 pts
- Follow-up 59 months
- Excellent response to CRT
- Improvement with LV pacing >85%

Wilkoff, Lancet 2023

Development of Atrial fibrillation less with Adaptive BiV and LV pacing



Hsu heart rhythm 2019

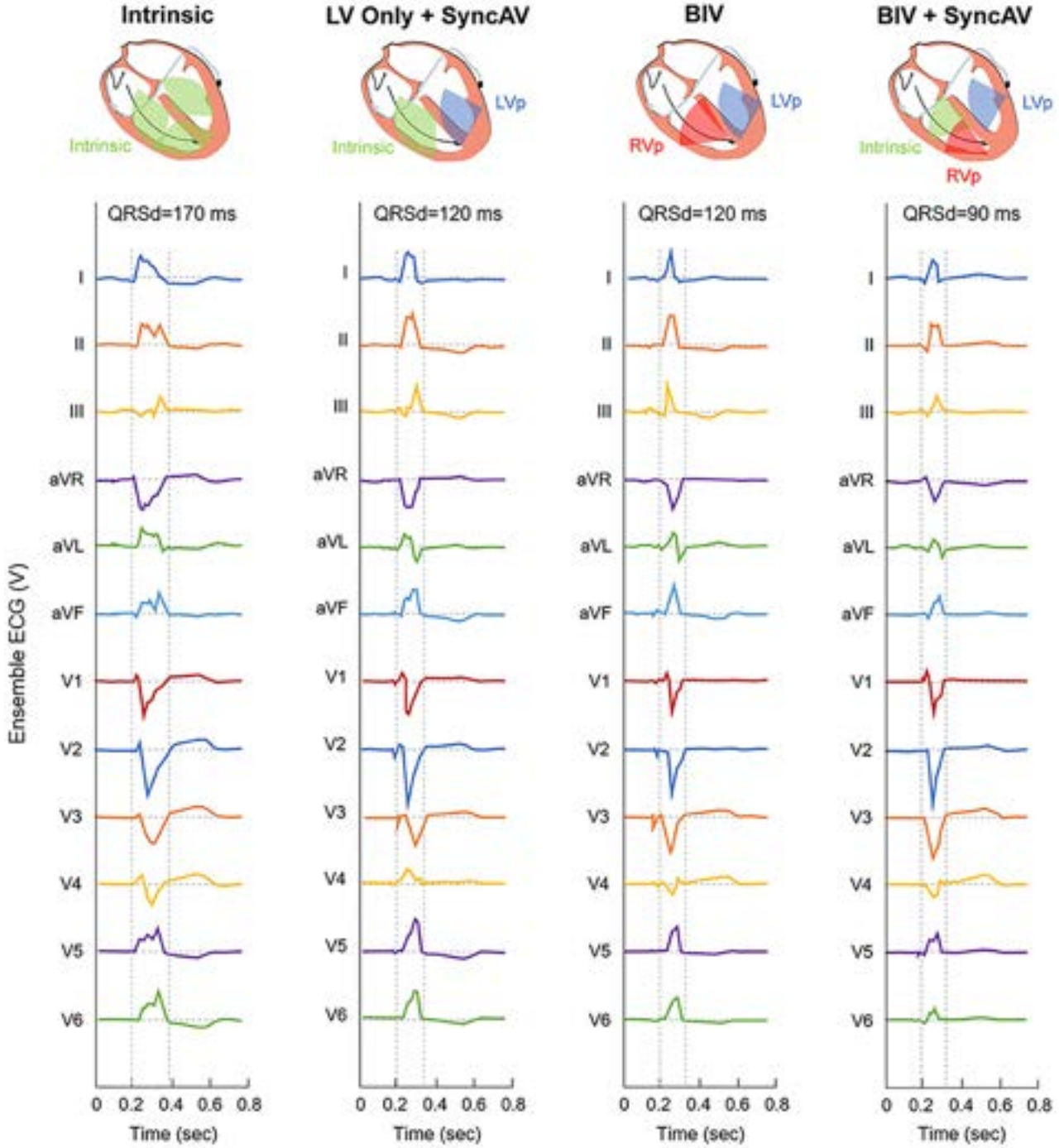


Smart Delay

Boston Scientific

Based on hemodynamic data from PaTH CHF II studies

- In-office only, measures intrinsic AV conduction time of RV and LV during atrial sensing and atrial pacing
- Algorithm recommends simultaneous BiV or LV pacing



Sync AV

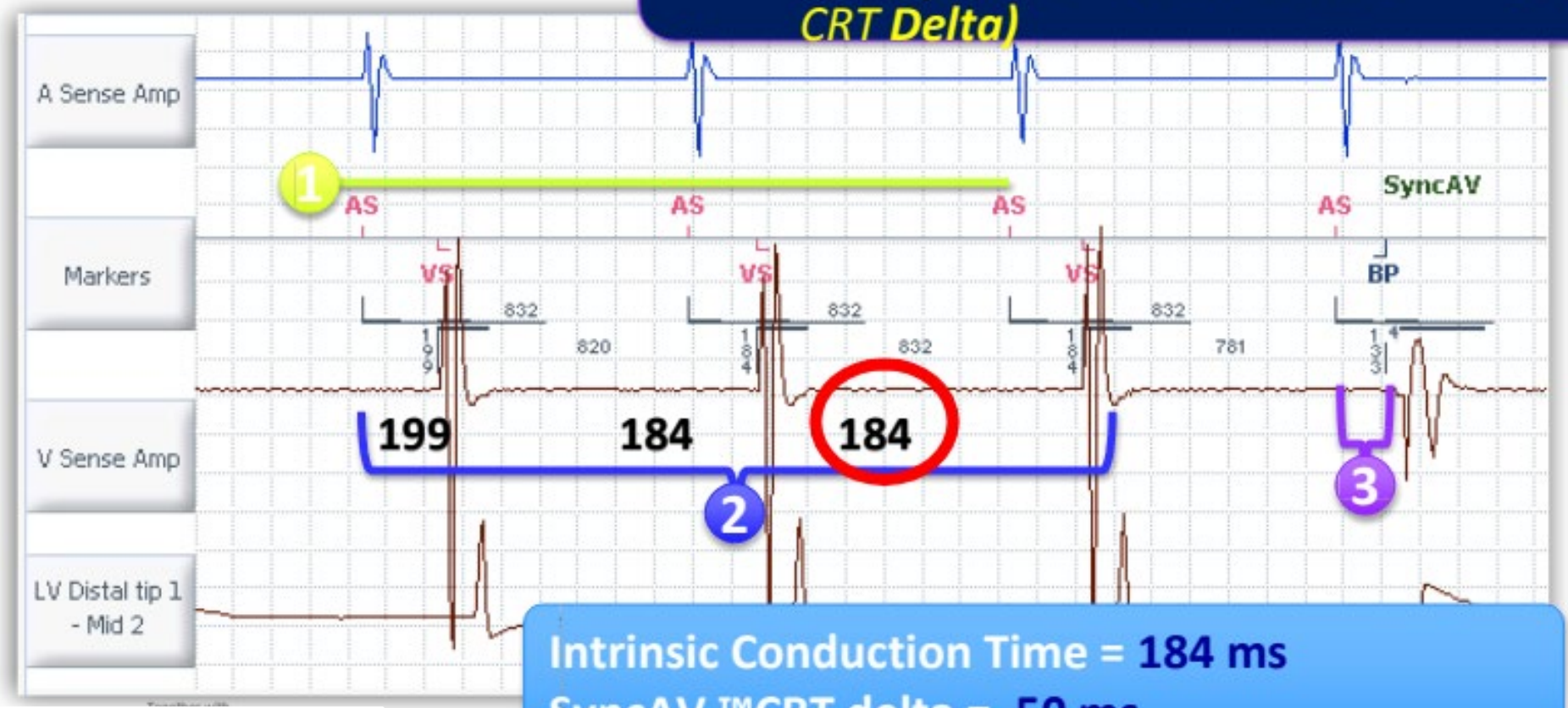
- Abbott
- Based on negative hysteresis algorithm that is already present in Abbott pacemakers

SyncAV™ CRT in action

SyncAV CRT adjusts the AV delay for the next 256 cycles using the following equation:

3

AV_{Delay} = ***(Intrinsic Conduction Time) – (SyncAV CRT Delta)***

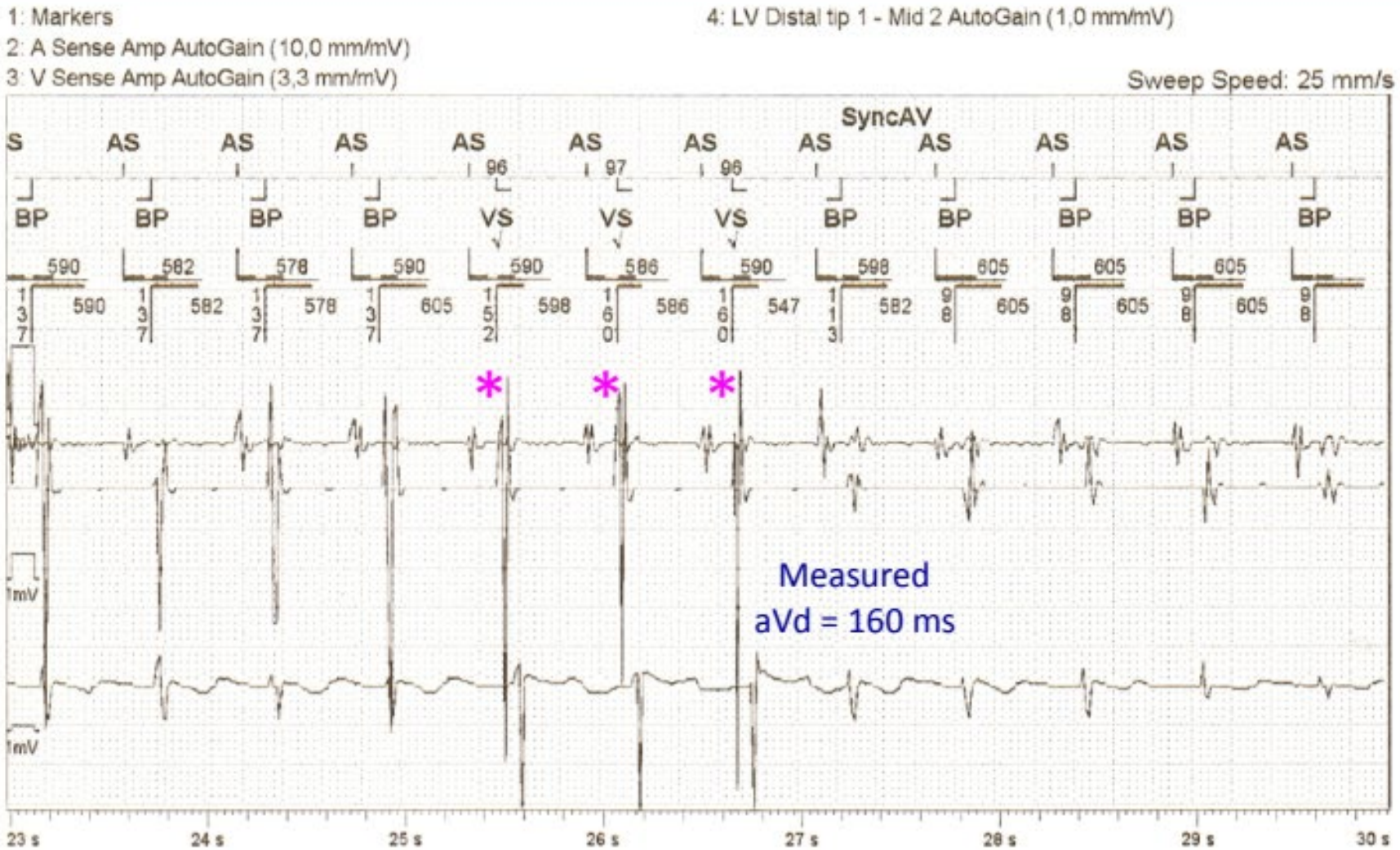


Intrinsic Conduction Time = 184 ms

SyncAV ™CRT delta = -50 ms

AV_{Delay} w/SyncAV™ CRT = (184 – 50) = 134 ms

Sync AV CRT adjusts AV delay

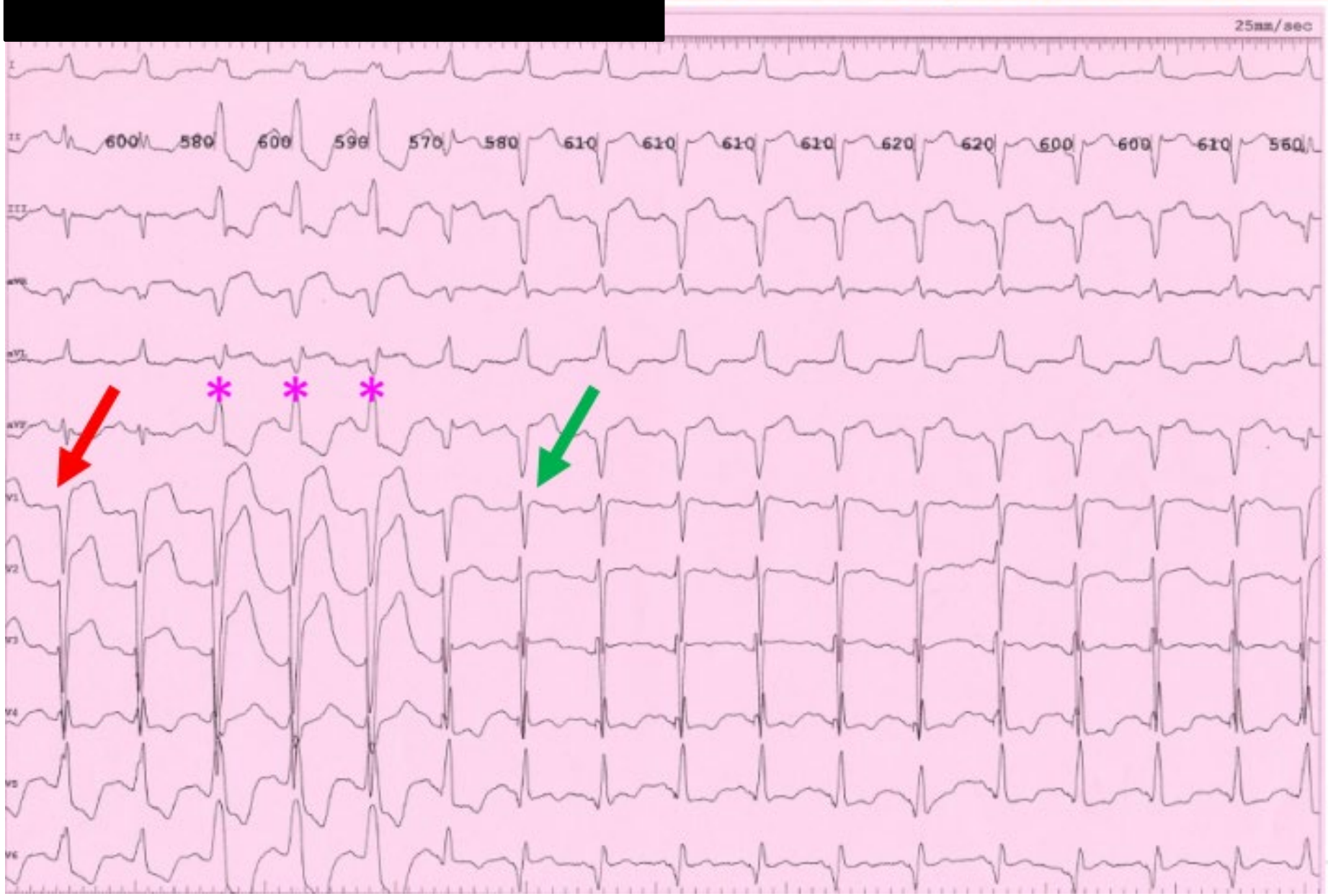


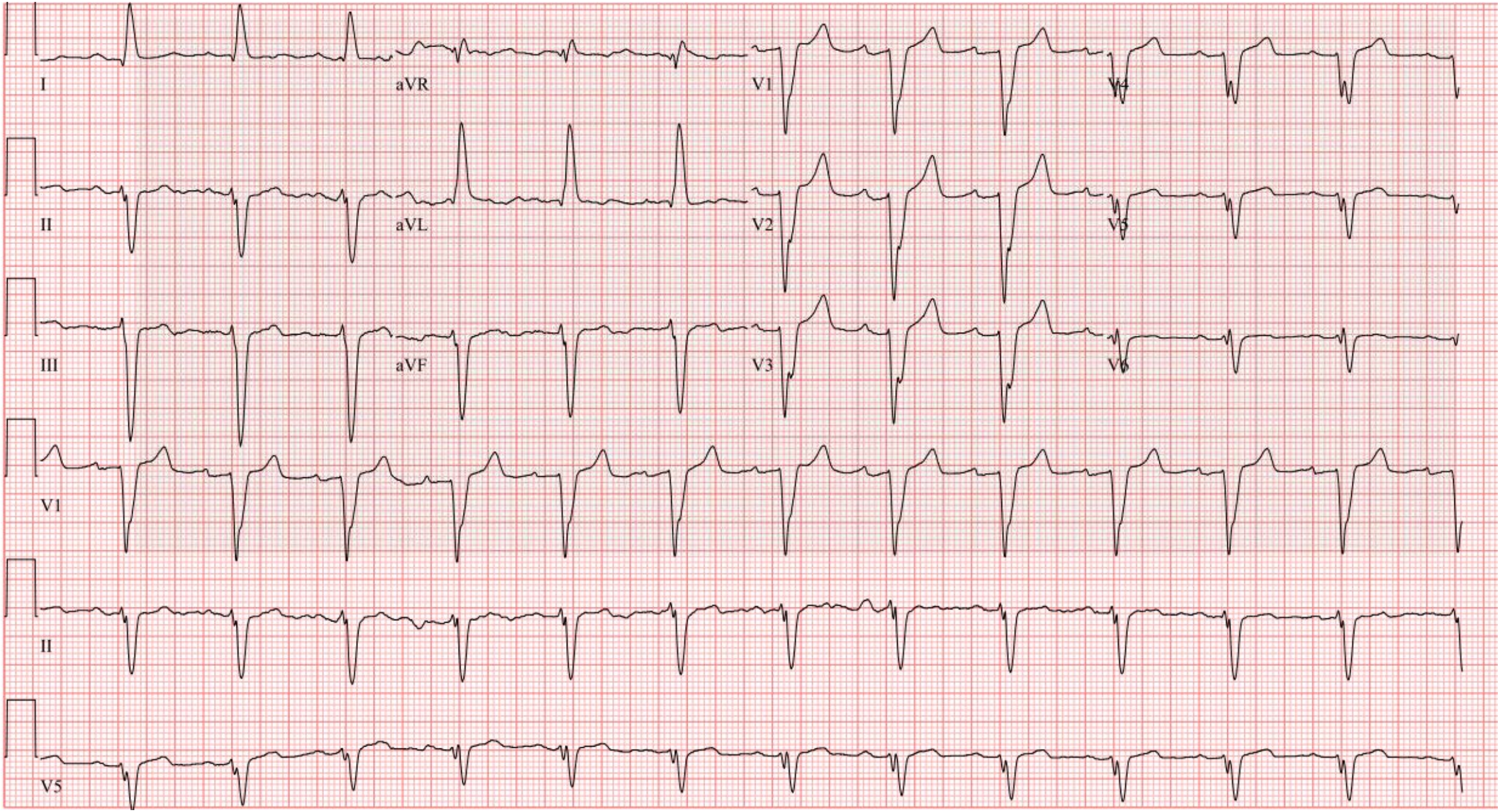
Pre AVd = 137ms

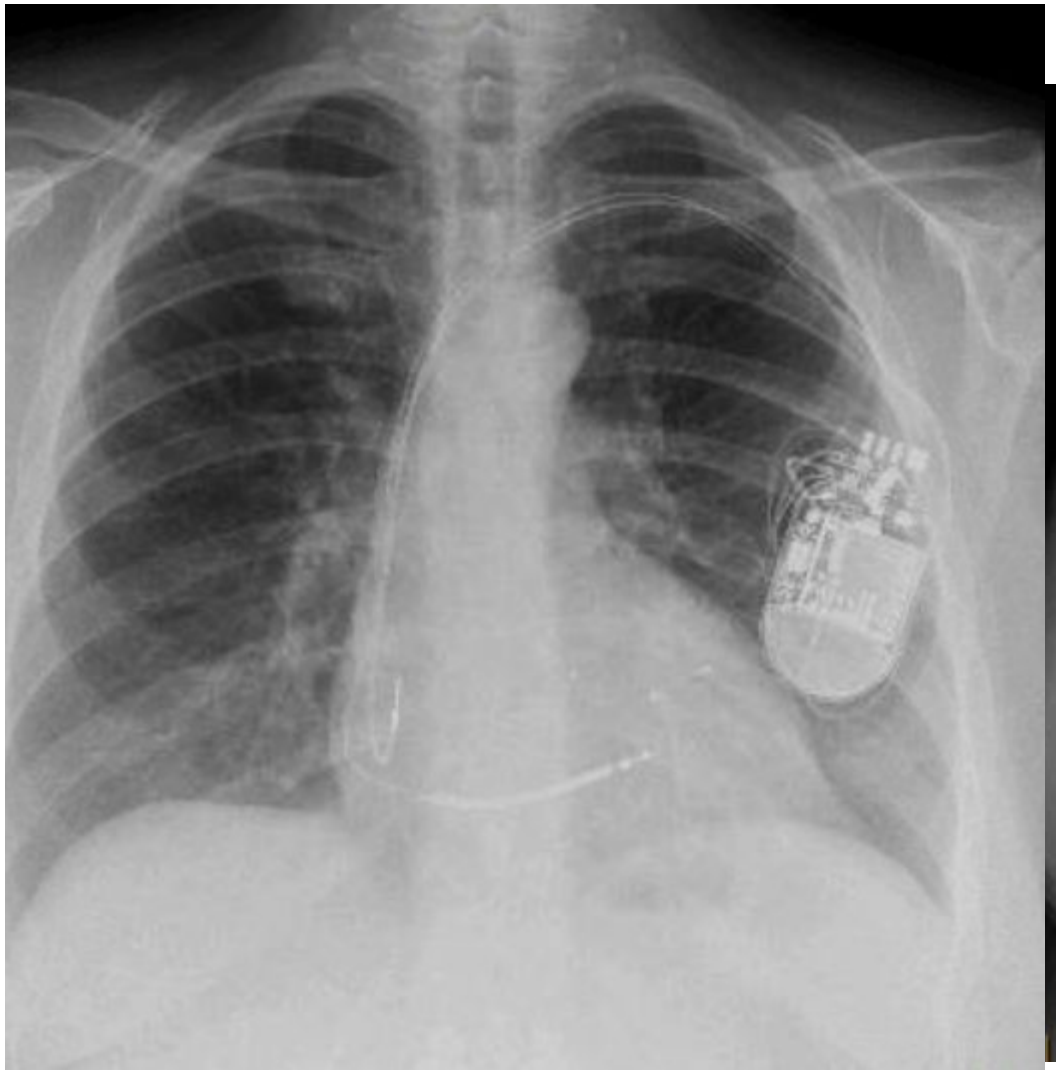
Post AVd = 98ms

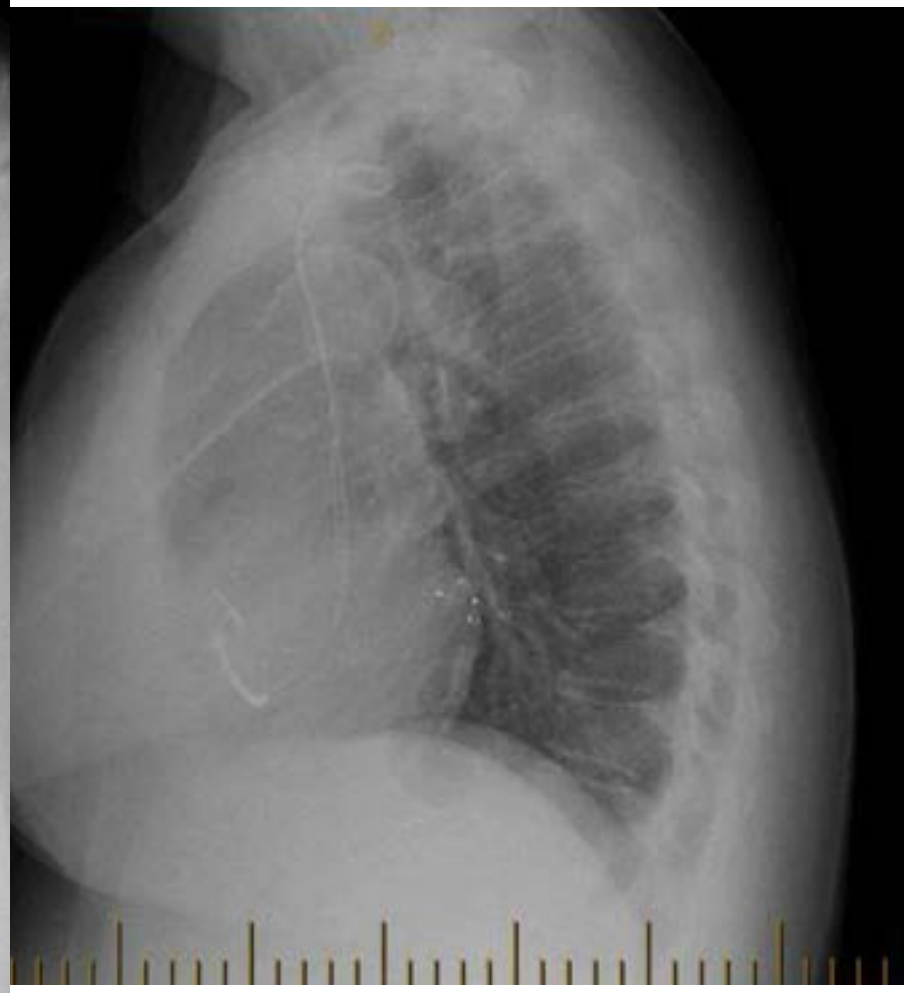
ESC Congress

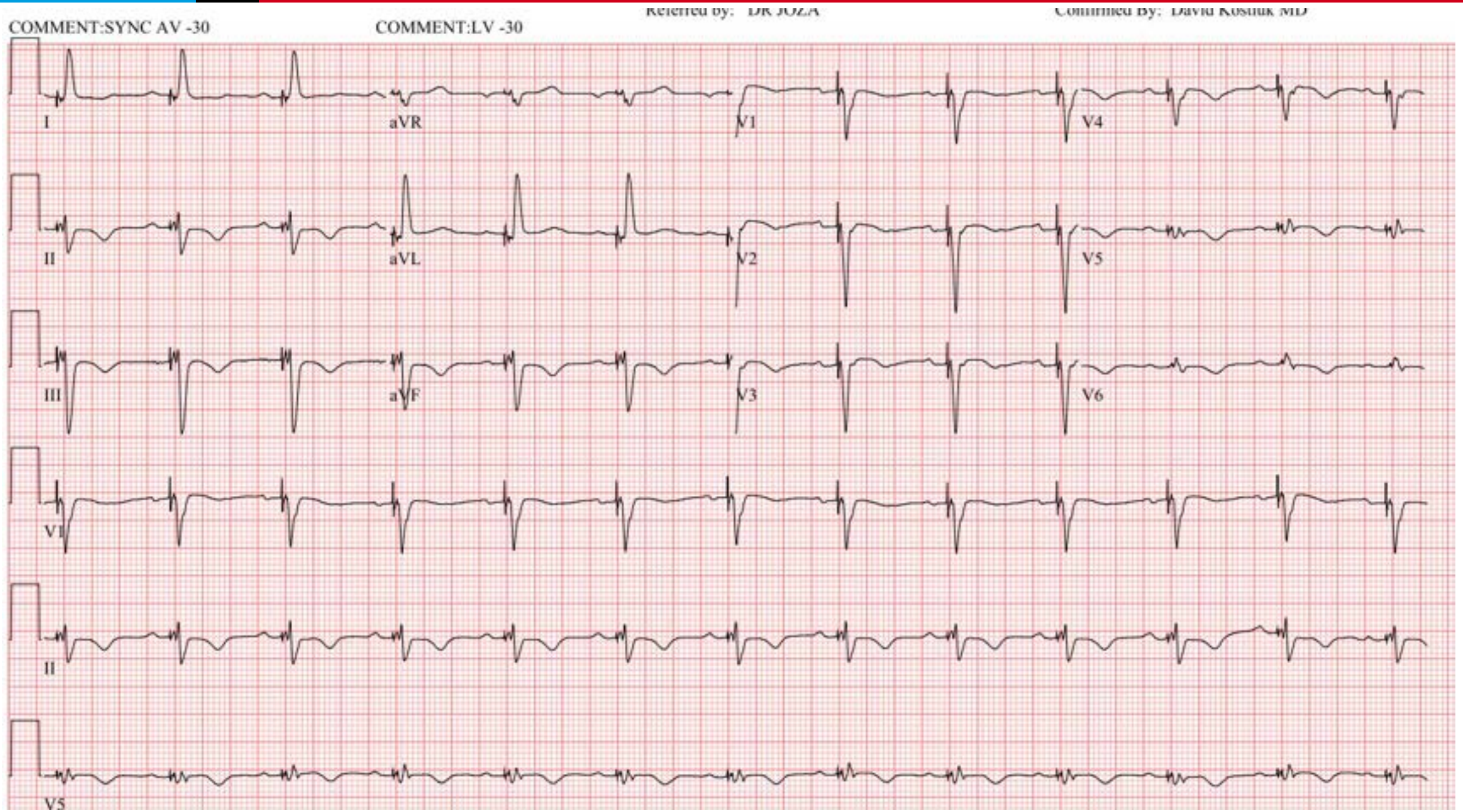
QRS narrows and R wave reappears in V1




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5



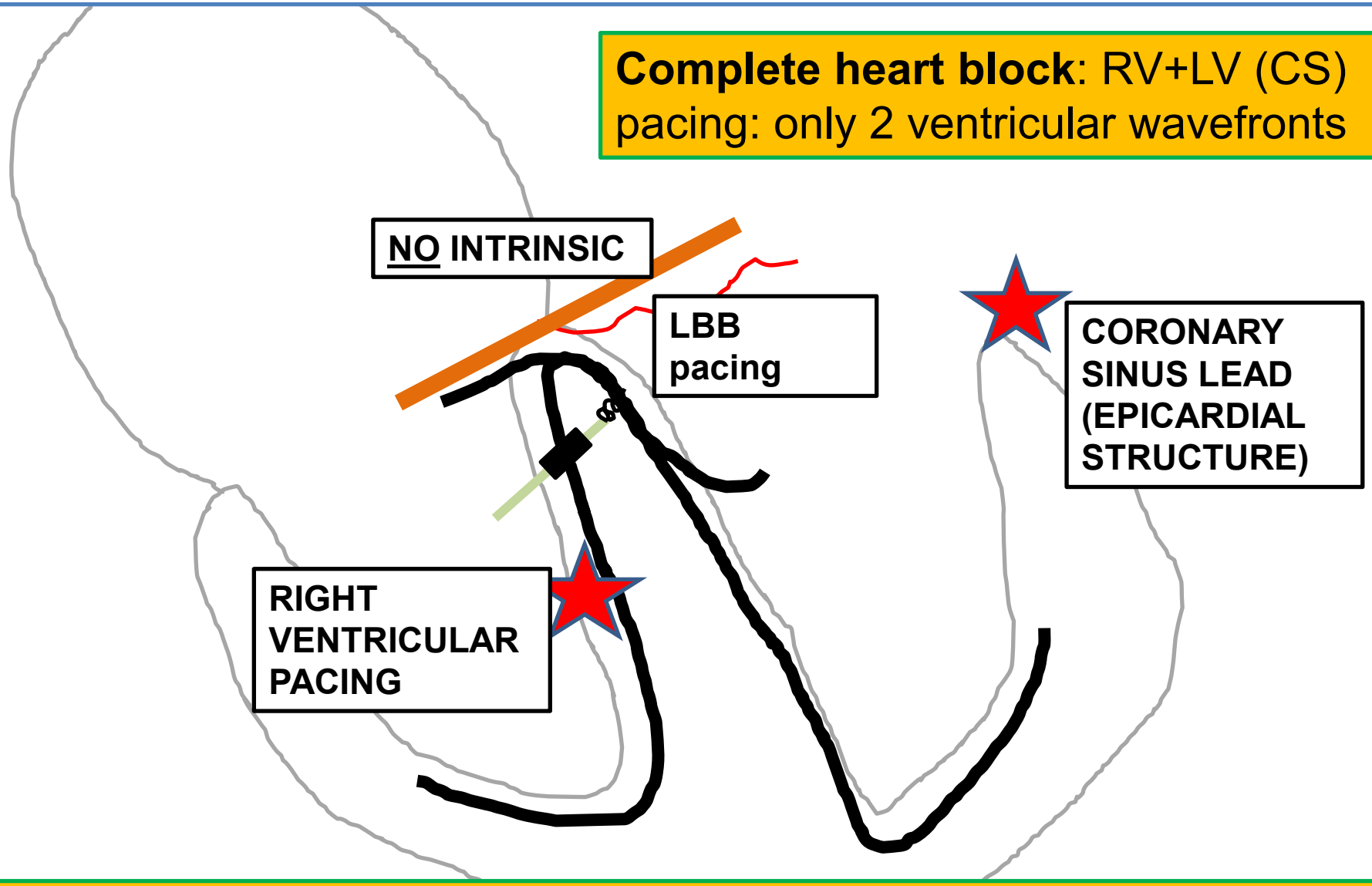




AV optimization, with LV occurring 30ms prior to RV: Abbott Device

Situation 2. When there is NO intrinsic ventricular rhythm. Ie. Heart Block

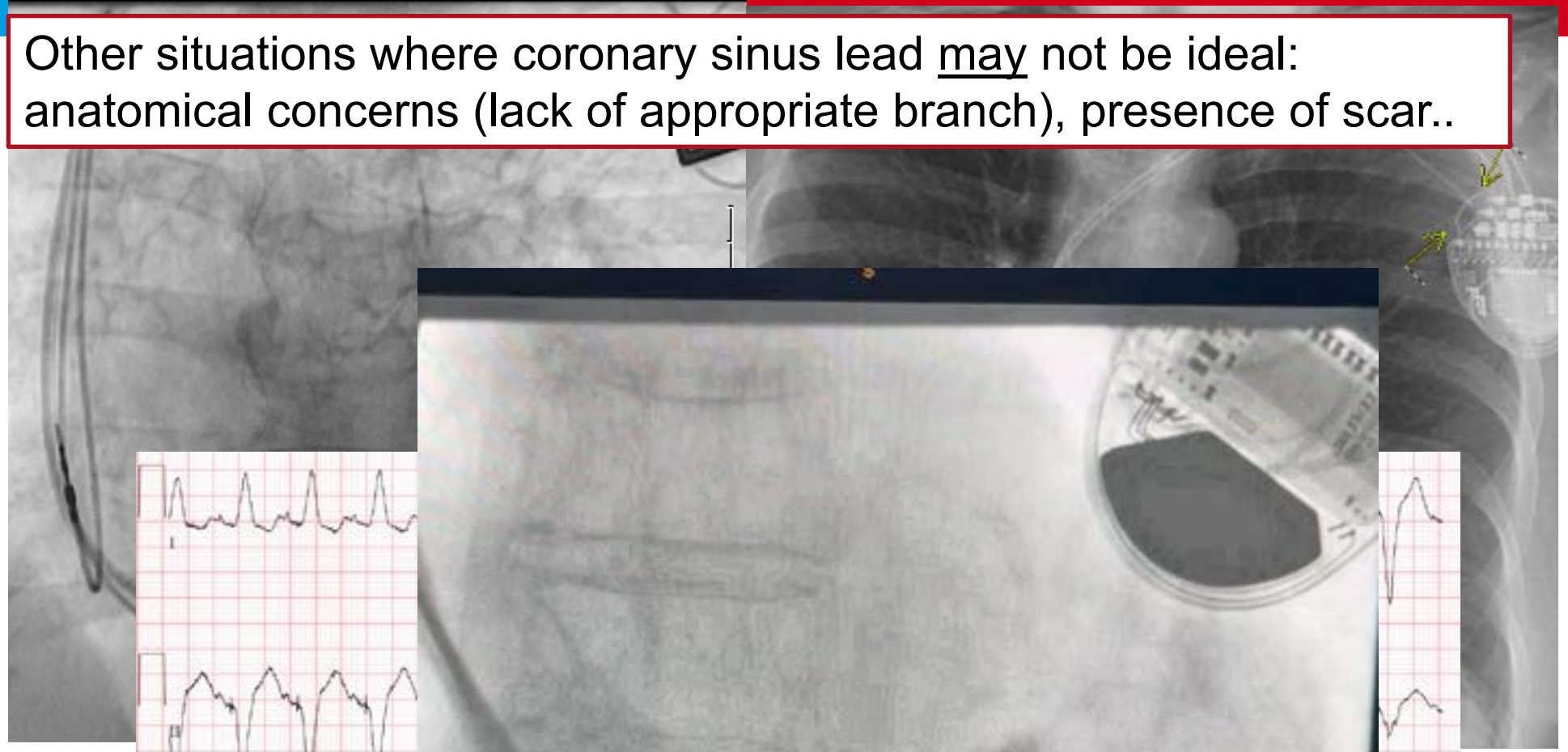
Complete heart block: RV+LV (CS) pacing: only 2 ventricular wavefronts

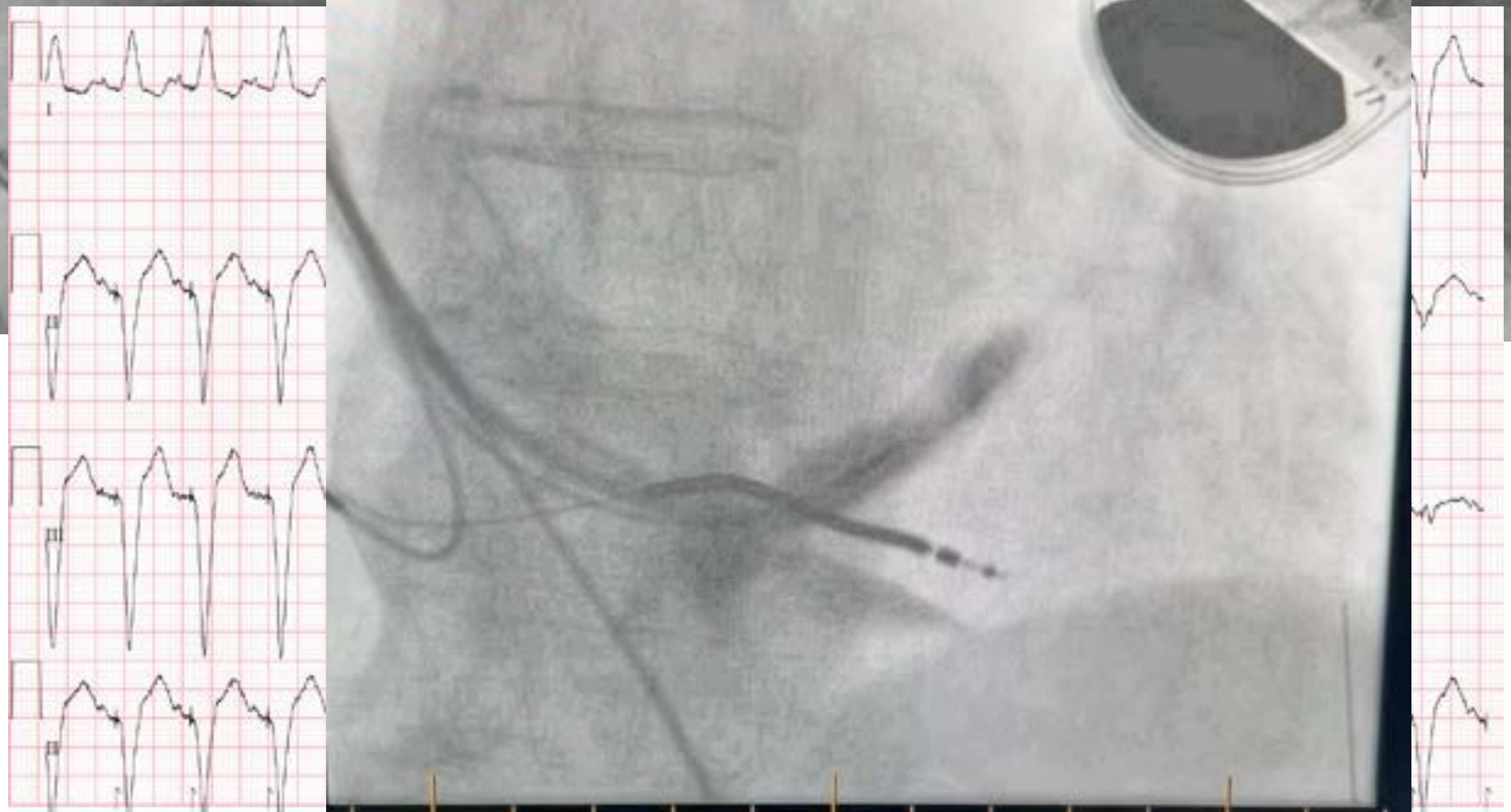


"Up to 1/3rd of CRTs do not improve after biventricular pacing".

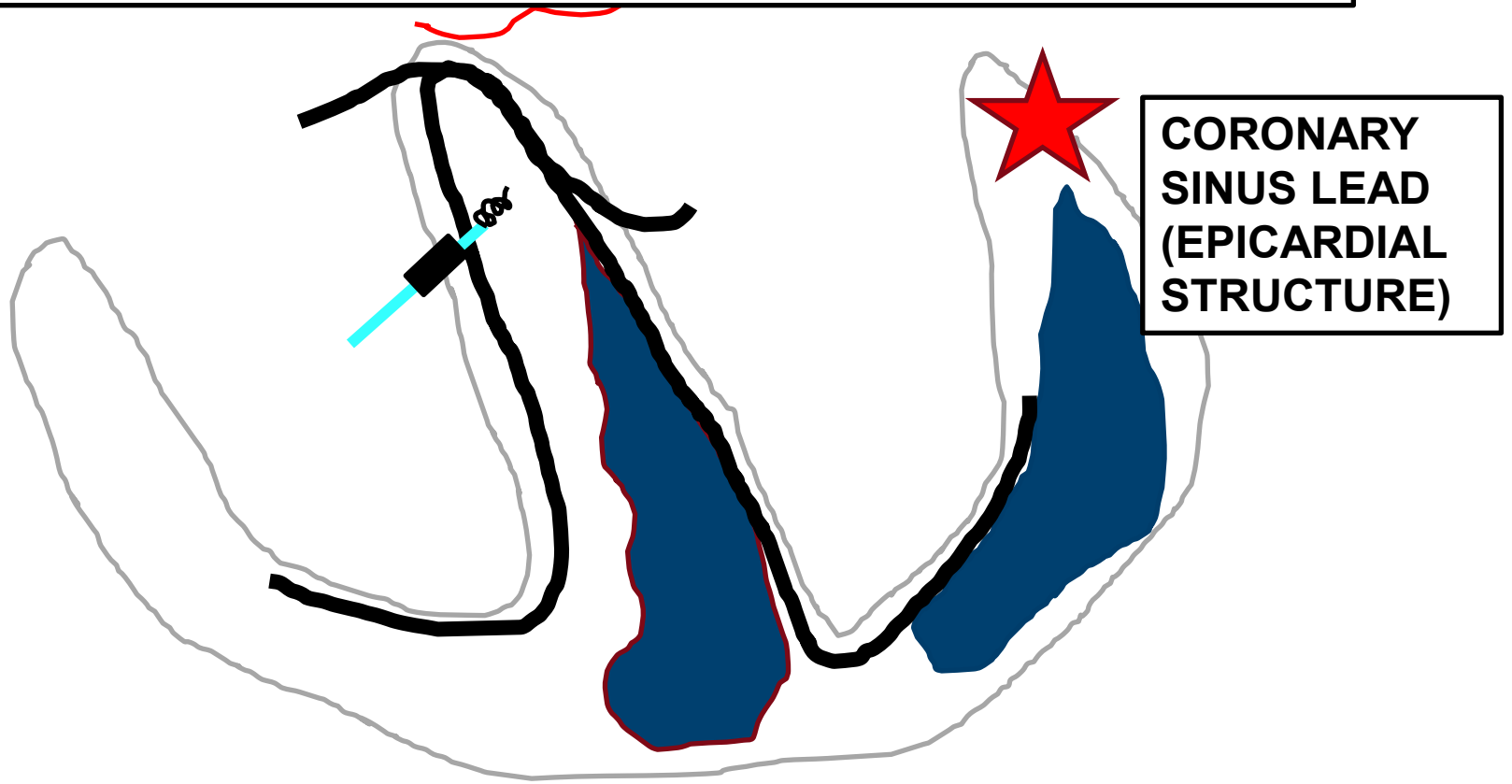
- Non-response is not necessarily a failure of CRT, but of appropriate patient selection

Other situations where coronary sinus lead may not be ideal: anatomical concerns (lack of appropriate branch), presence of scar..





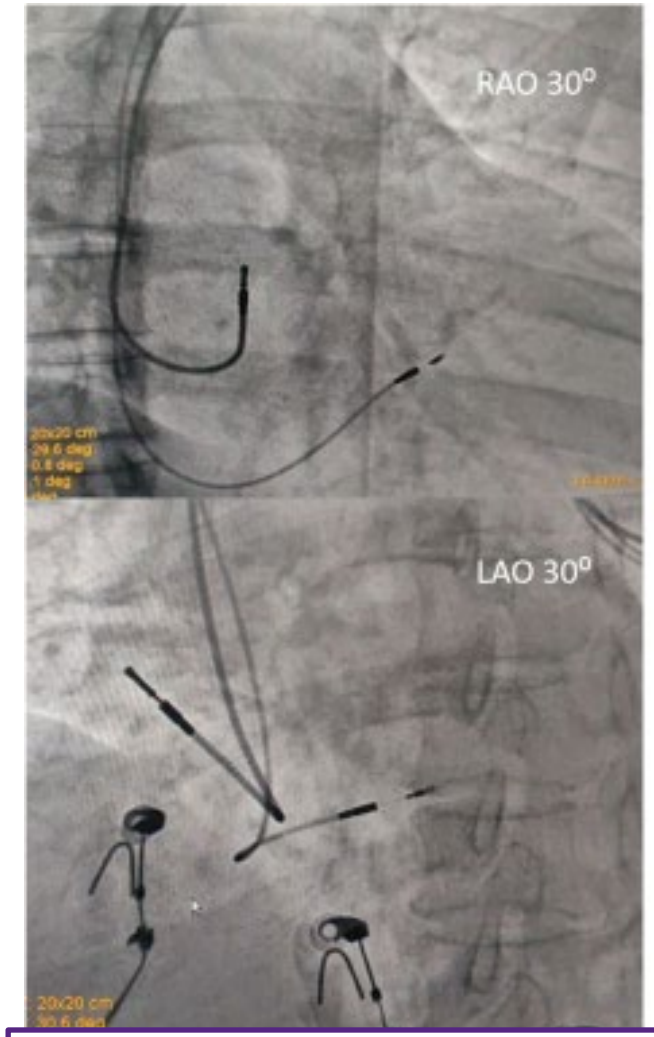
Inappropriate patient selection: IVCD – not an electrical disease, but distal pathology or myopathy leading to dysregulated ventricular activation

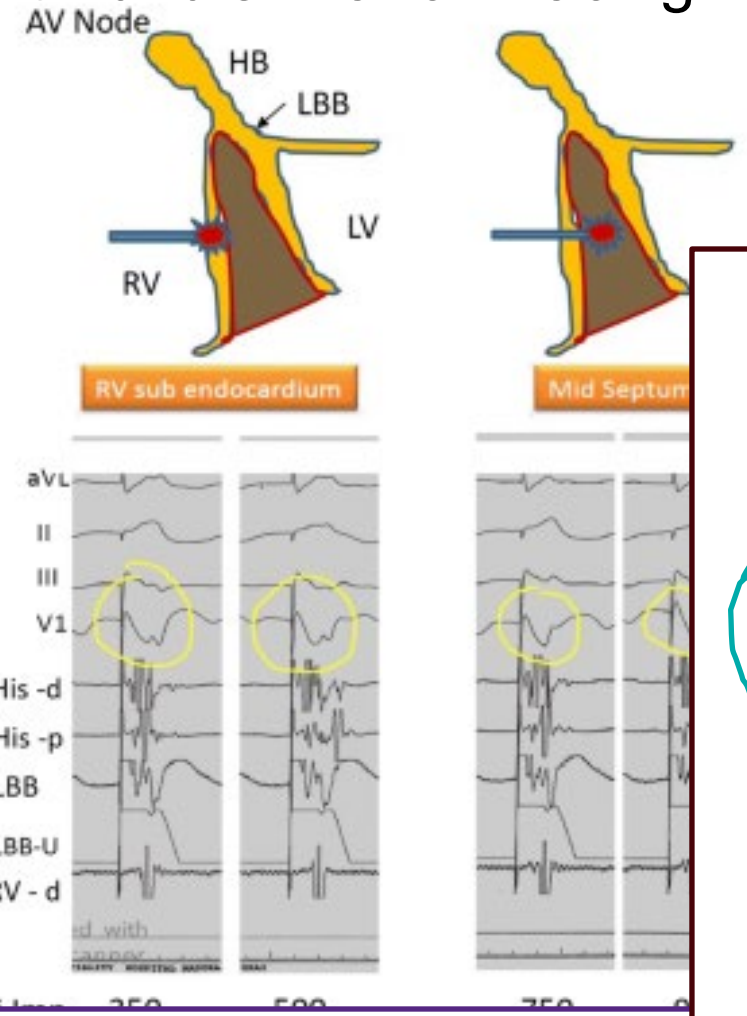


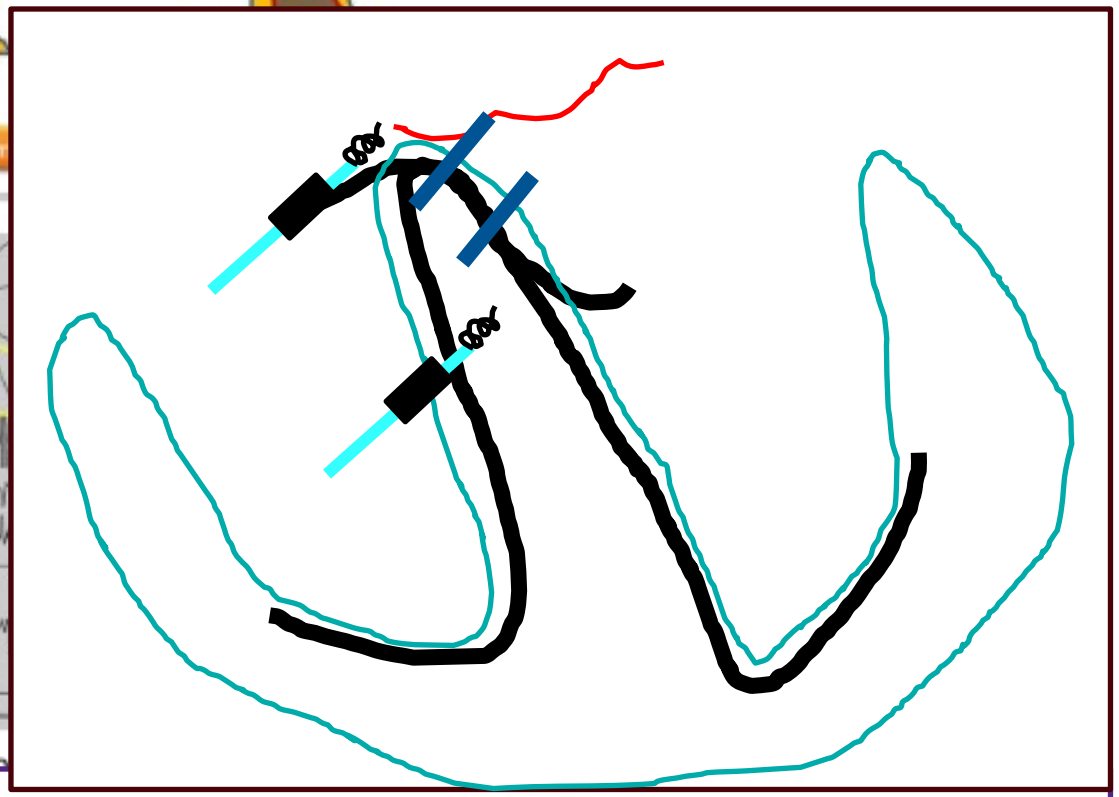
Patients may benefit from CRT

No potential benefit from conduction system pacing

Left Bundle Branch Pacing: 1st case report in 2017

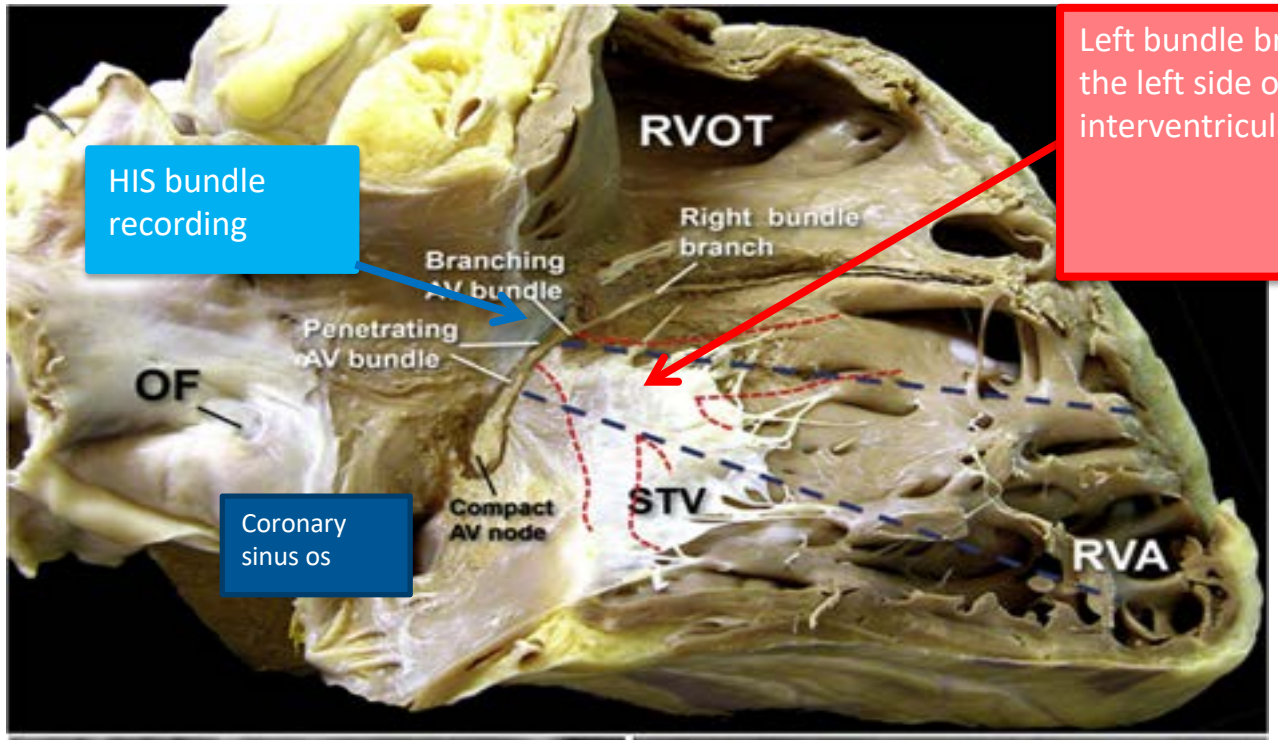




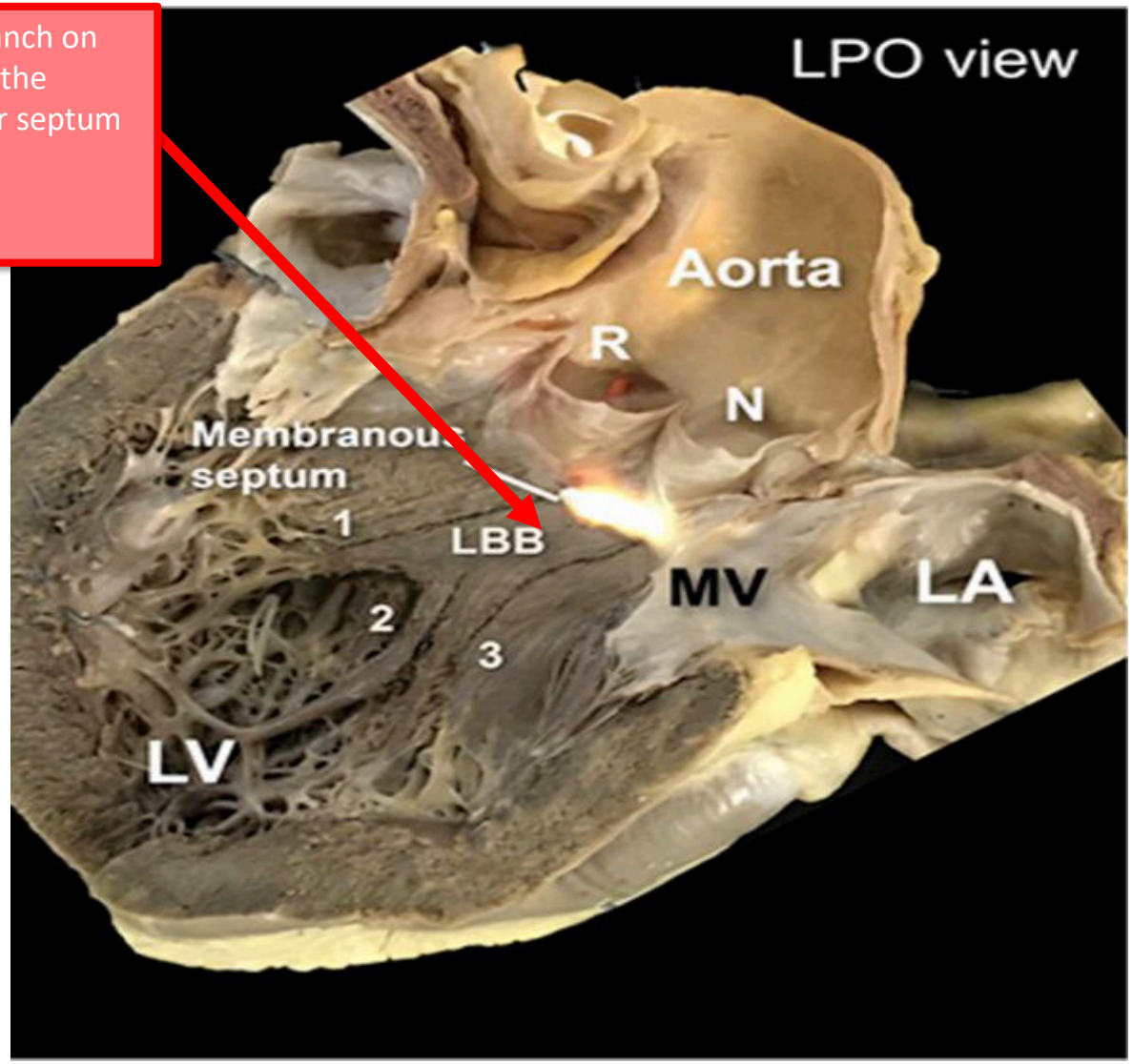


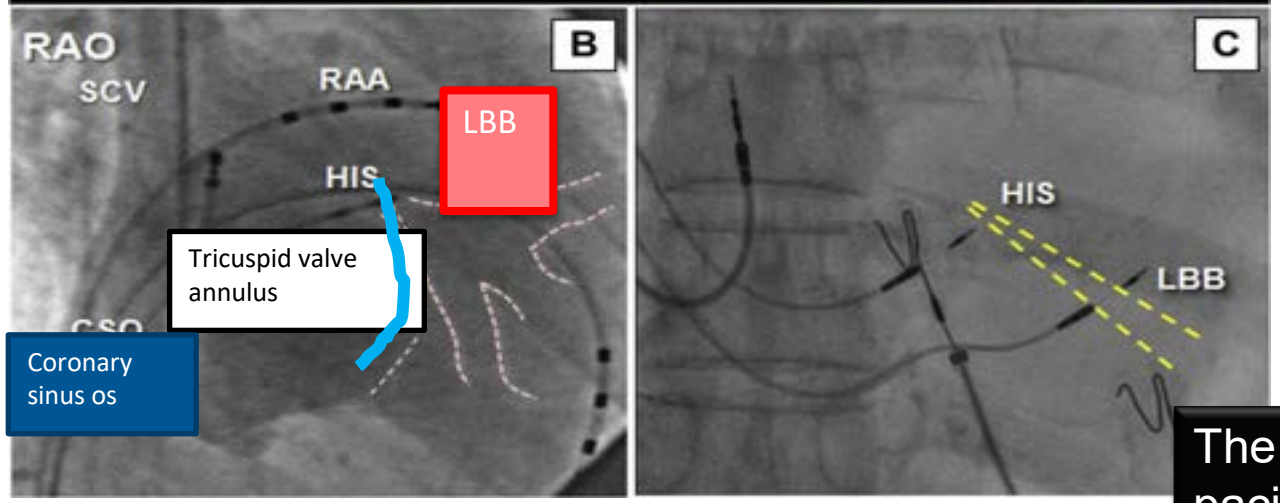
What about HIS Bundle Pacing?

- Still being performed in highly experienced centers
- Concern for development of block distal to the HIS pacing lead, increasing thresholds over time, frequent need for back-up RV apical pacing

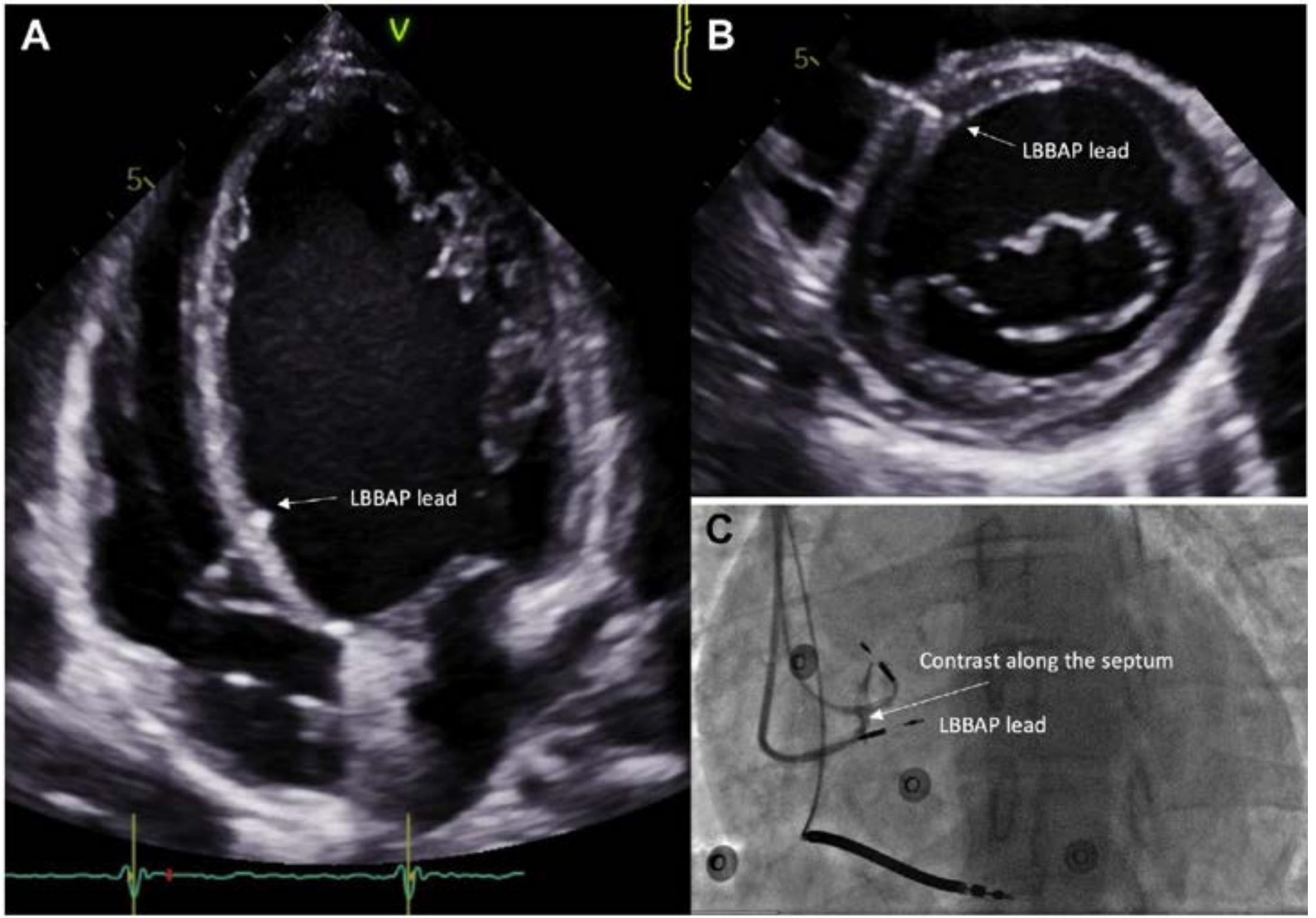


Left bundle branch on the left side of the interventricular septum

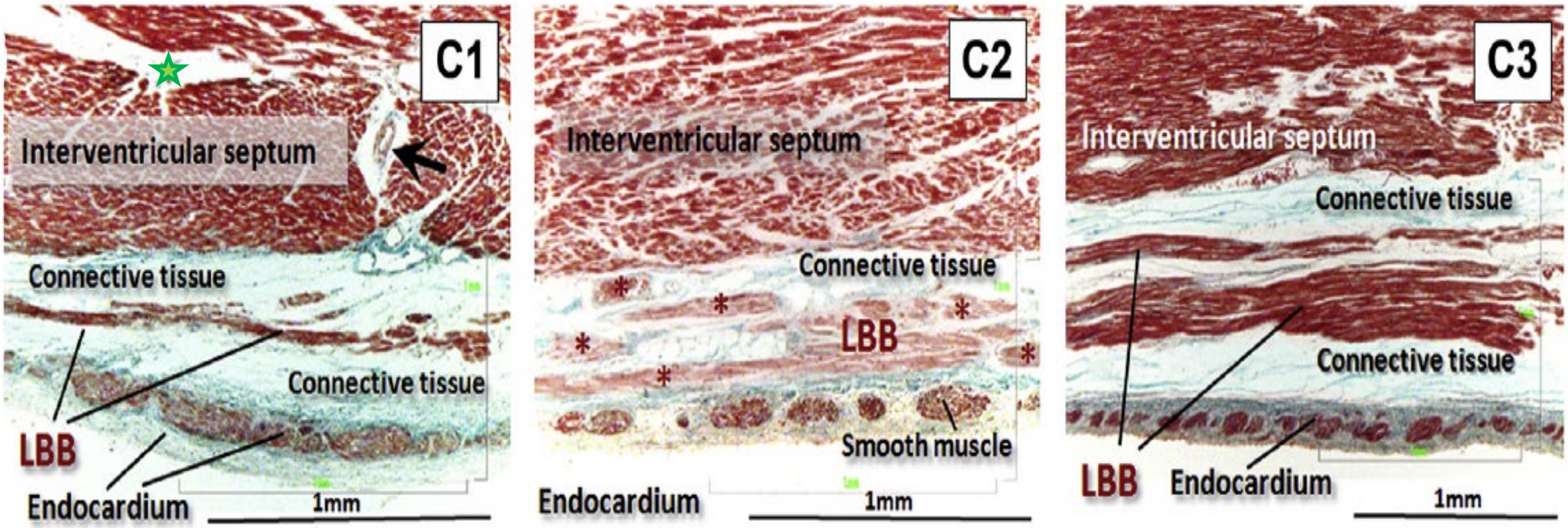




The relationship with a His pacing lead: Left bundle pacing lead is typically 1-1.5mm further distally along the septum


A
V
LBBAP lead
B
LBBAP lead
C
Contrast along the septum
LBBAP lead

The thickness of the LBB is highly variable = variability in ‘implant success’

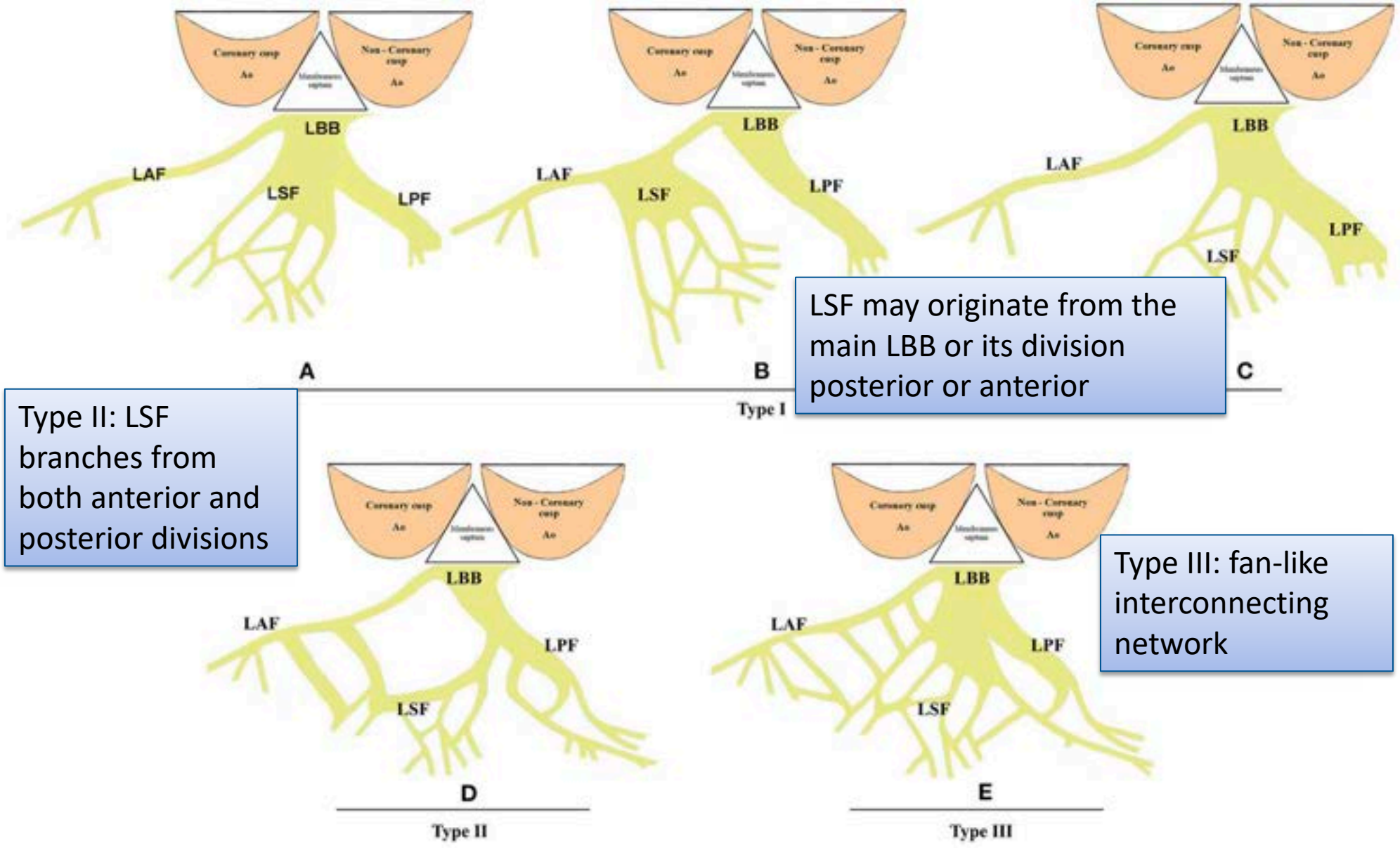


C1: very small LBB

C3: very large LBB

Endocardium has smooth muscle cells in its thickness and surrounding the LBB there is connective tissue of variable thickness.
Green star in C1= fibrous tissue; black arrow = septal artery

Cabrera et al. JACC Case reports Vol 2 No5 2020



LSF may originate from the main LBB or its division posterior or anterior

Type II: LSF branches from both anterior and posterior divisions

Type III: fan-like interconnecting network

Description of the Left septal fascicle's anatomical variants in 20 normal human hearts

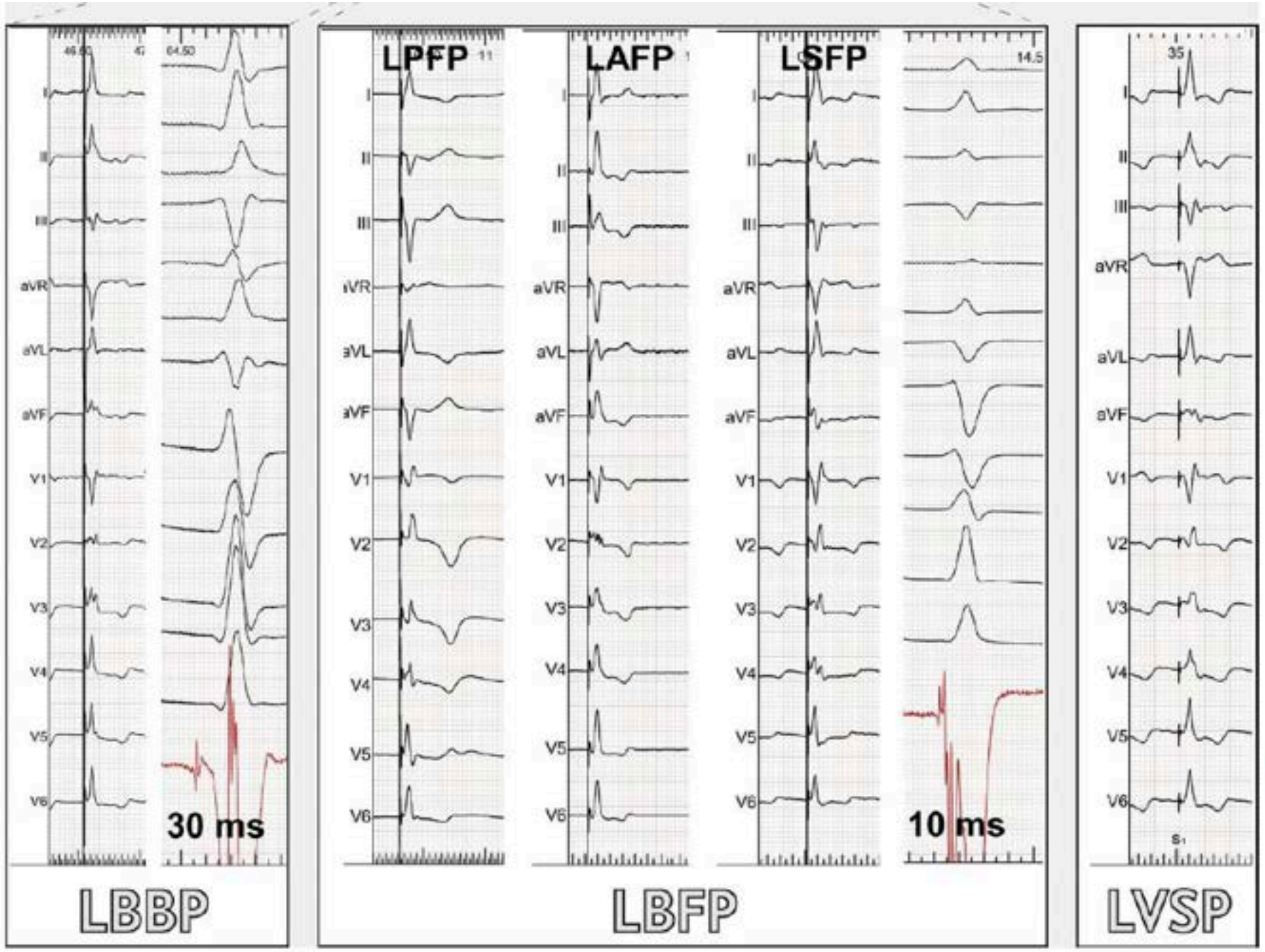
Type 1 = most common pattern showing definite septal division.

"The existence and importance of the Left septal fascicle cannot be ignored"

- Produce a network of interwoven strands covering the inferior third of the septum

FIGURE 2 | The anatomical variants of LSF. **(A–C)** The most common LSF pattern, known as type I. In this type, LSF may originate from the main LBB or any of its division (PD or AD). **(D)** Type II anatomical variants of LSF. The LSF branches concomitantly from the AD and PD. **(E)** In type III, LSF is a "fan-like interconnecting network." LSF, left septal fascicle; LBB, left bundle branch; AD, anterior division; PD, posterior division.

Frontiers 2021

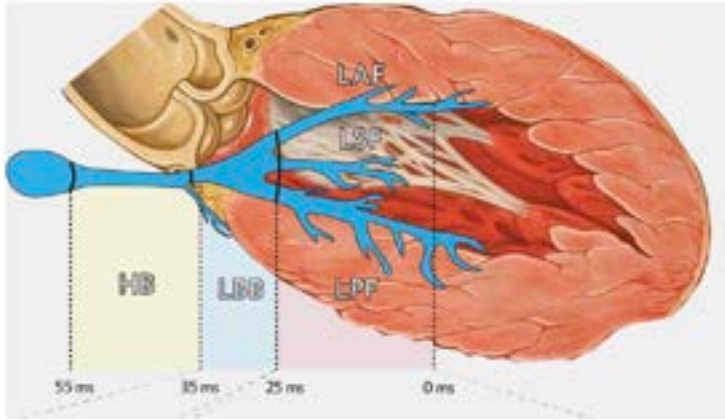


LB Fascicular pacing divides into:

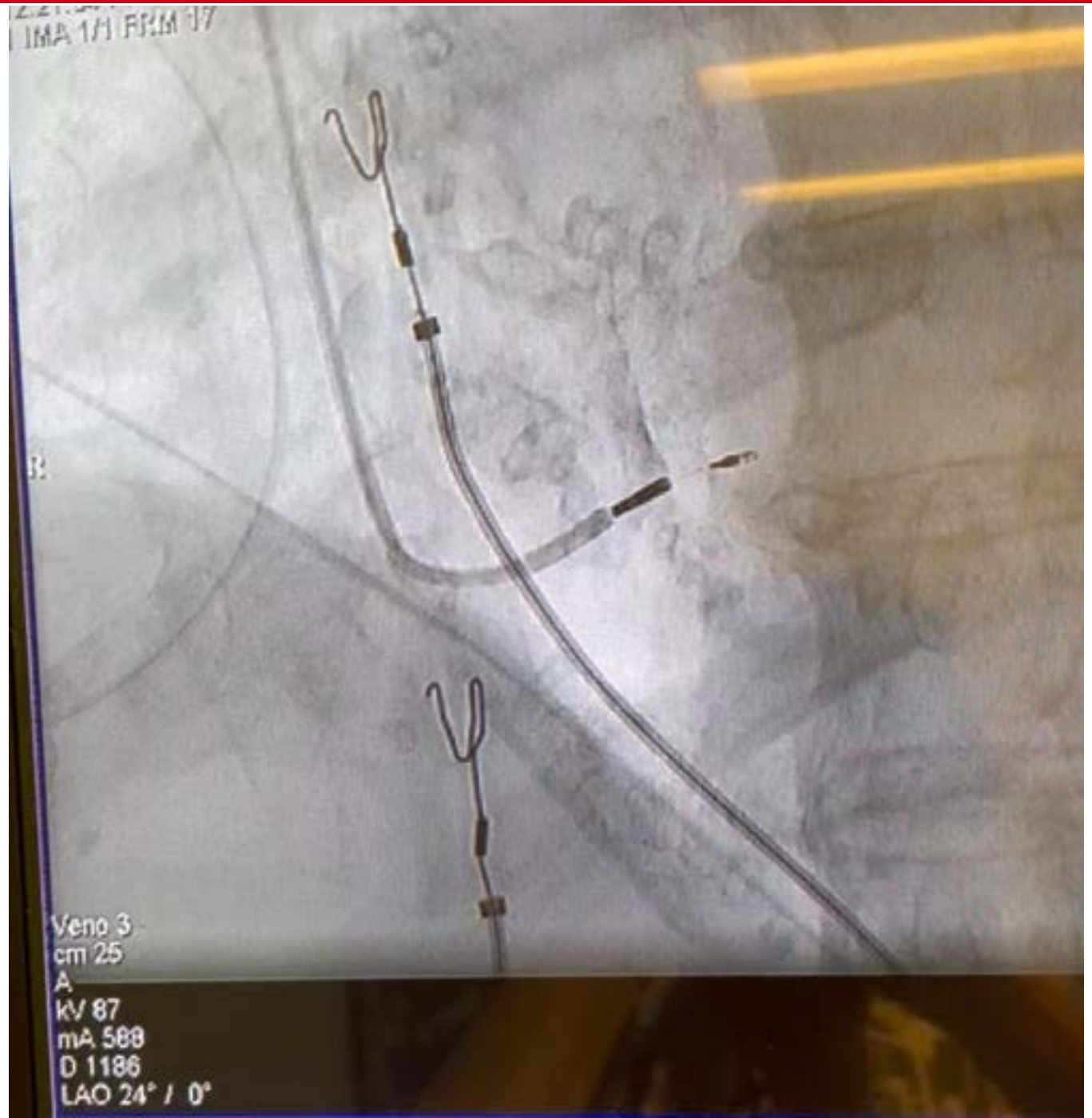
I. **LPFP**: left posterior fascicle pacing: superior QRS axis (leads II and III mostly negative)

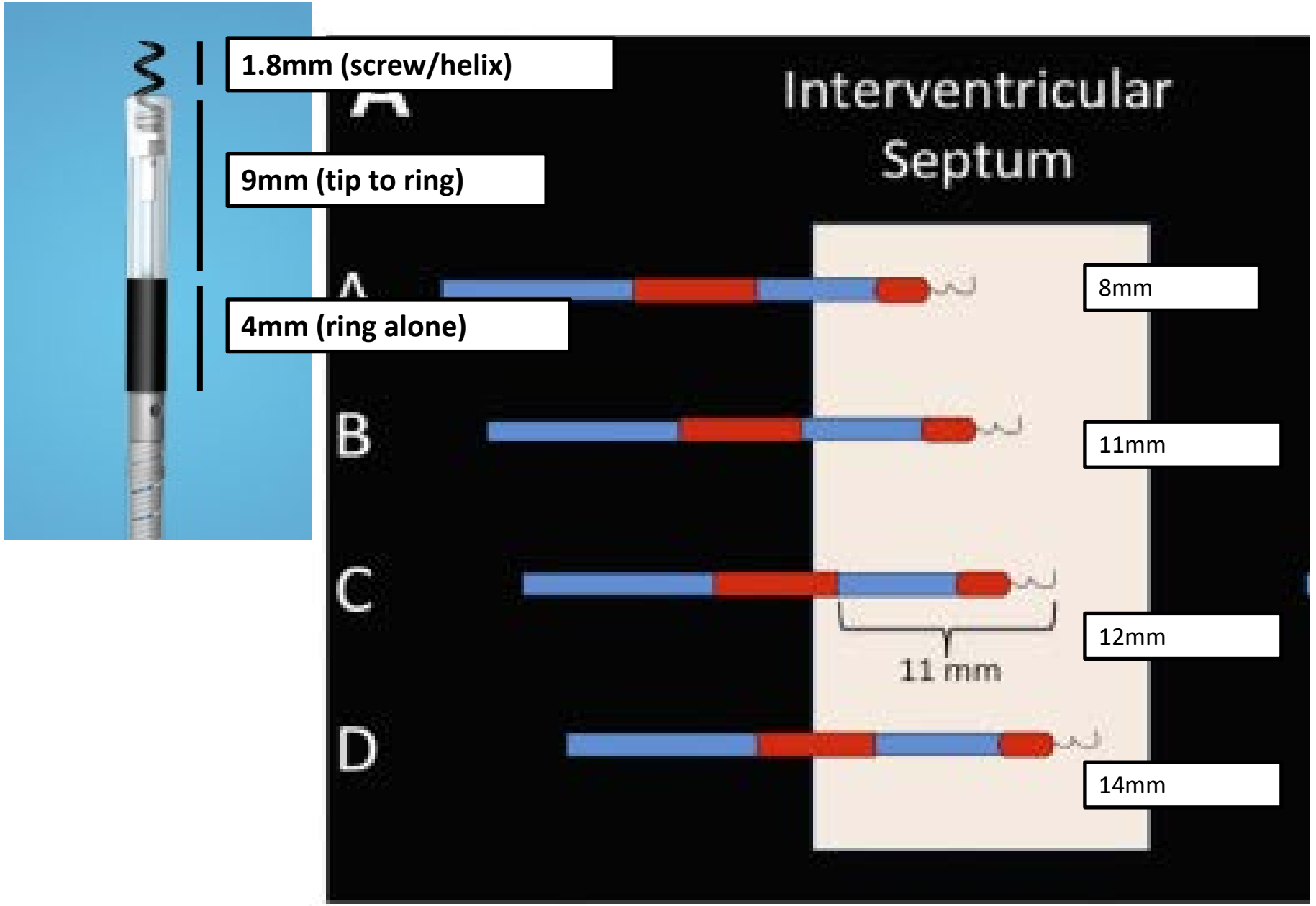
II. **LAFP**: left anterior fascicle pacing: inferior QRS axis (leads II and III positive)

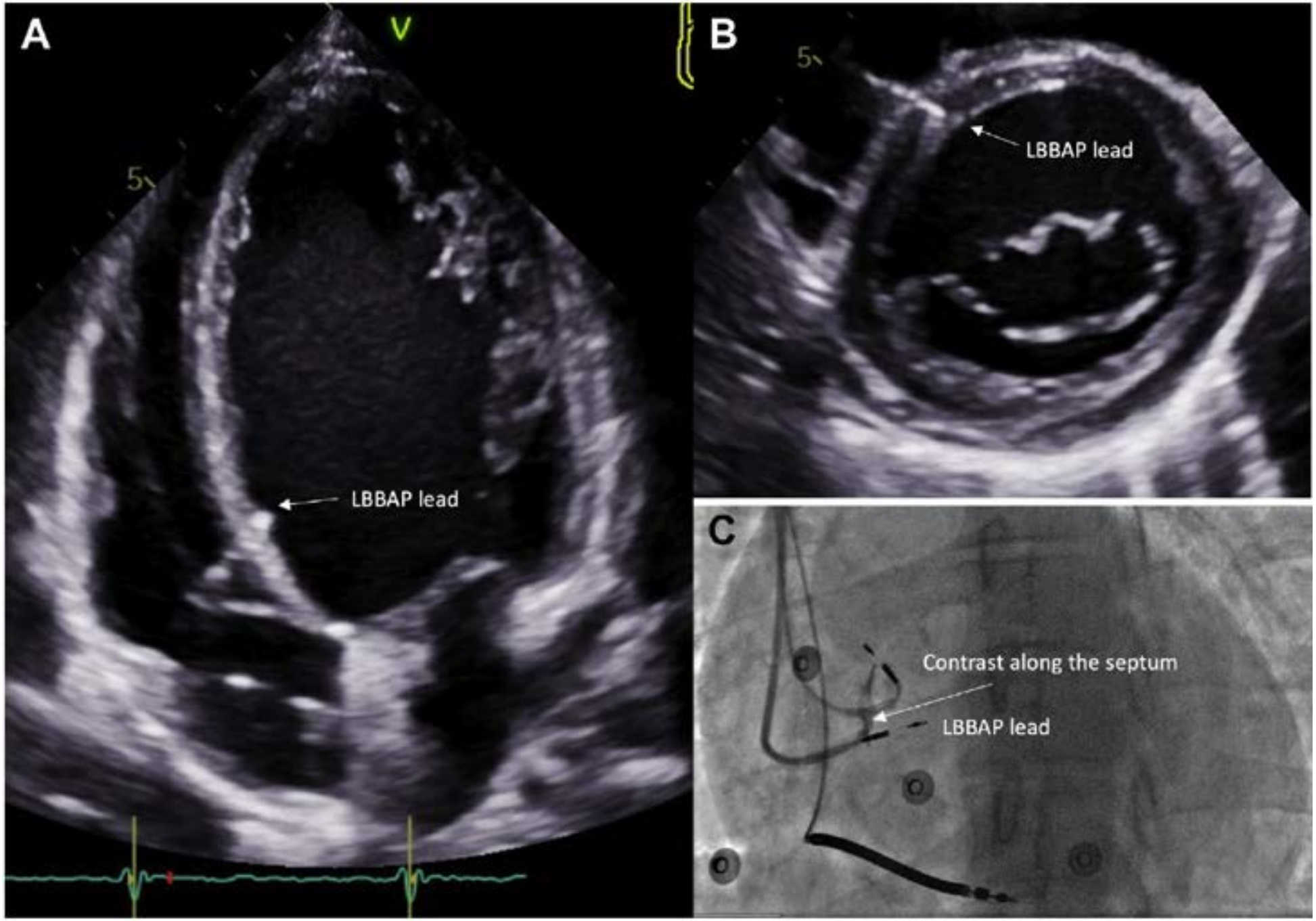
III. **LSFP**: intermediate QRS axis (II mostly positive, III with negative component)

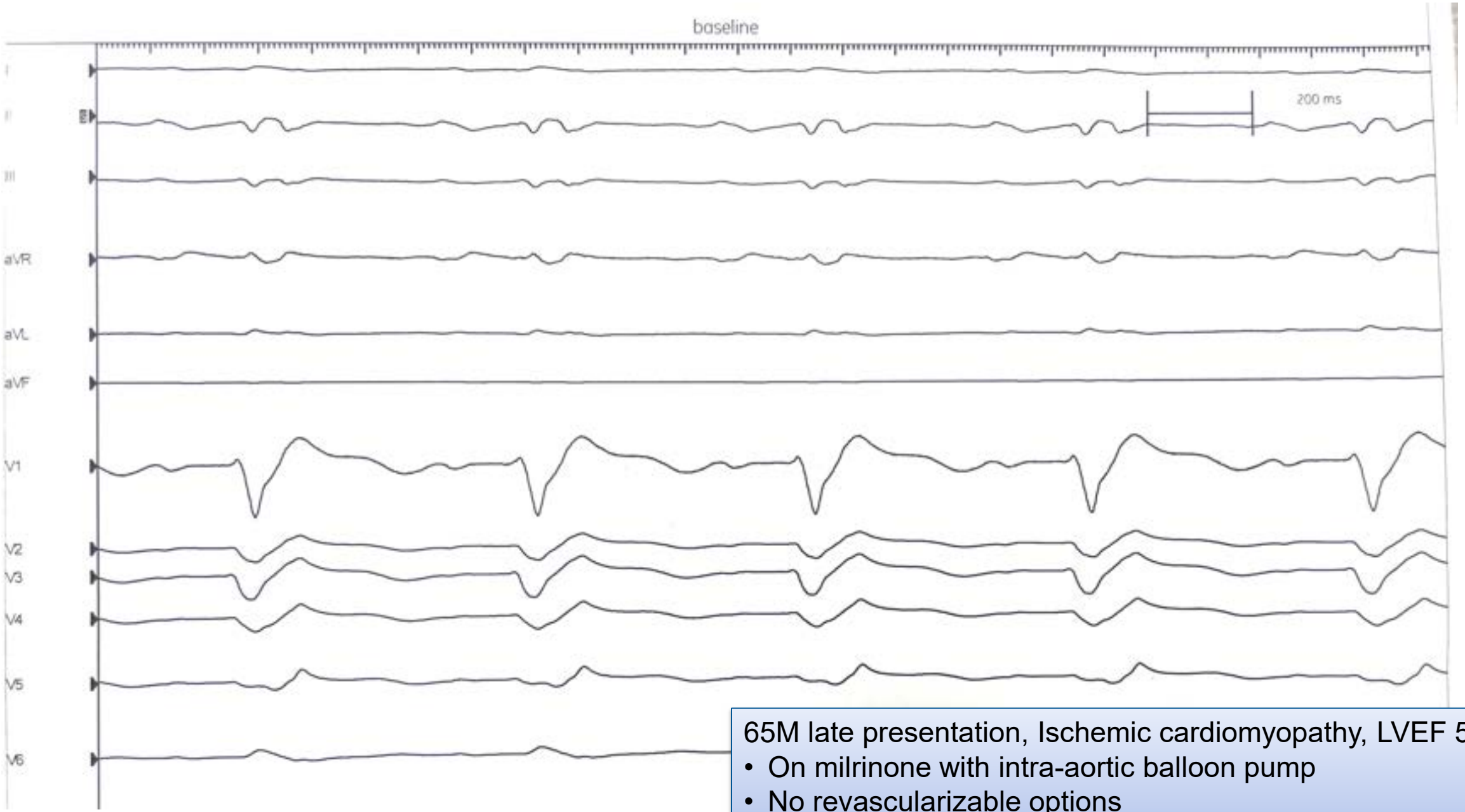


Melos Study Jastrzebski. 2022


Veno 3
cm 25
A
kV 87
mA 588
D 1186
LAO 24° / 0°

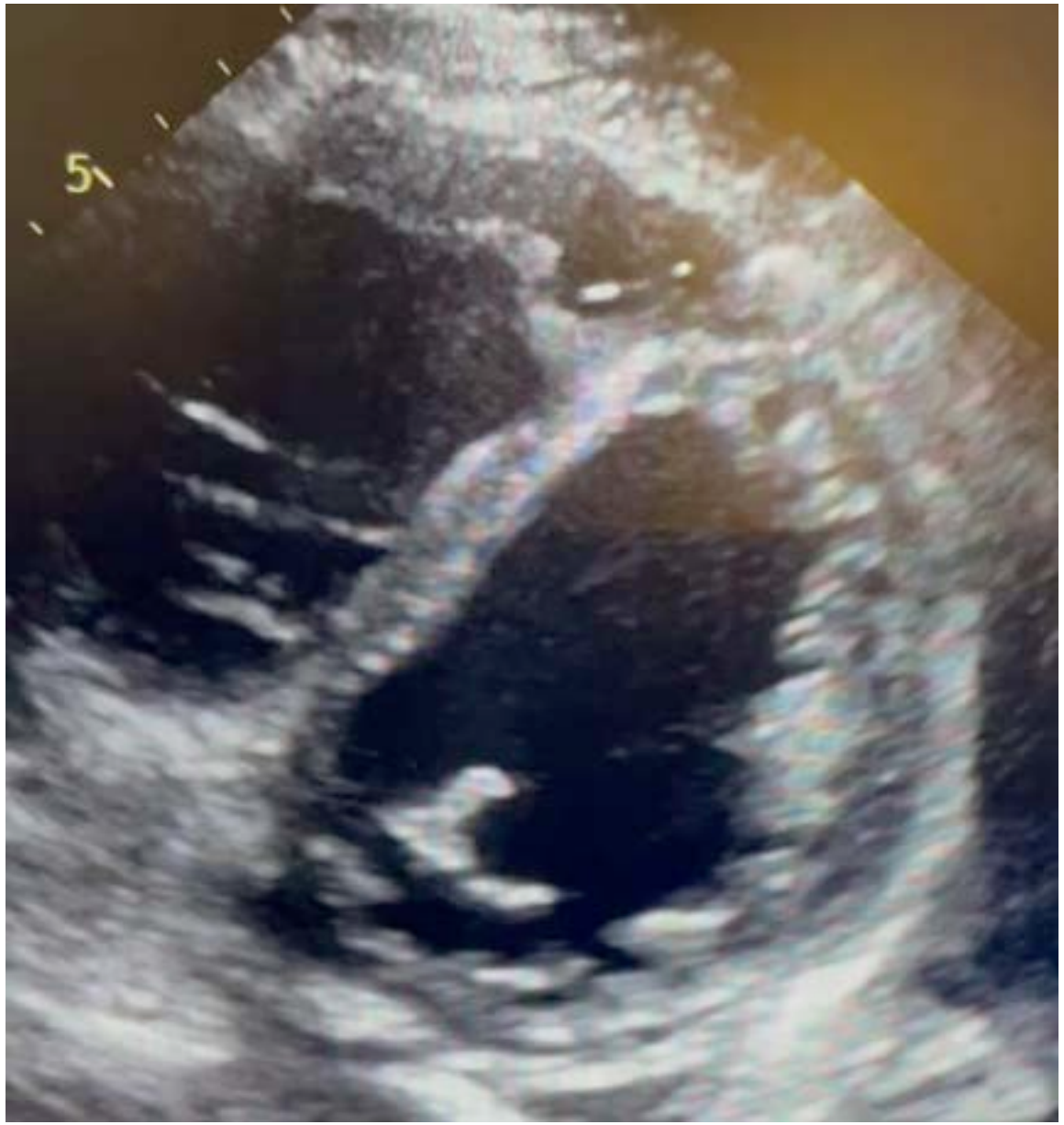

1.8mm (screw/helix)
9mm (tip to ring)
4mm (ring alone)
Interventricular Septum
A
B
C
D
11 mm
8mm
11mm
12mm
14mm

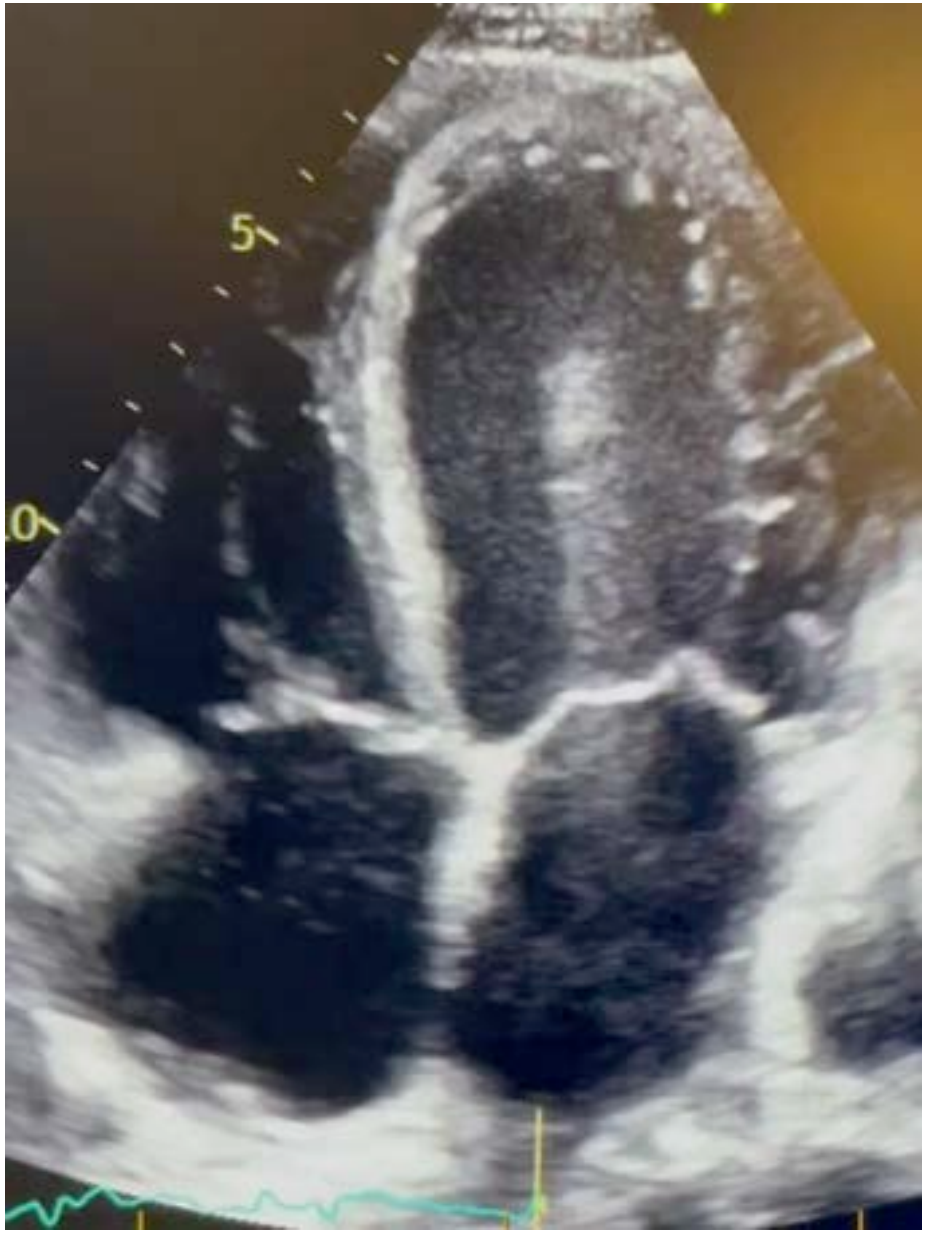

A
V
5
LBBAP lead
B
5
LBBAP lead
C
Contrast along the septum
LBBAP lead

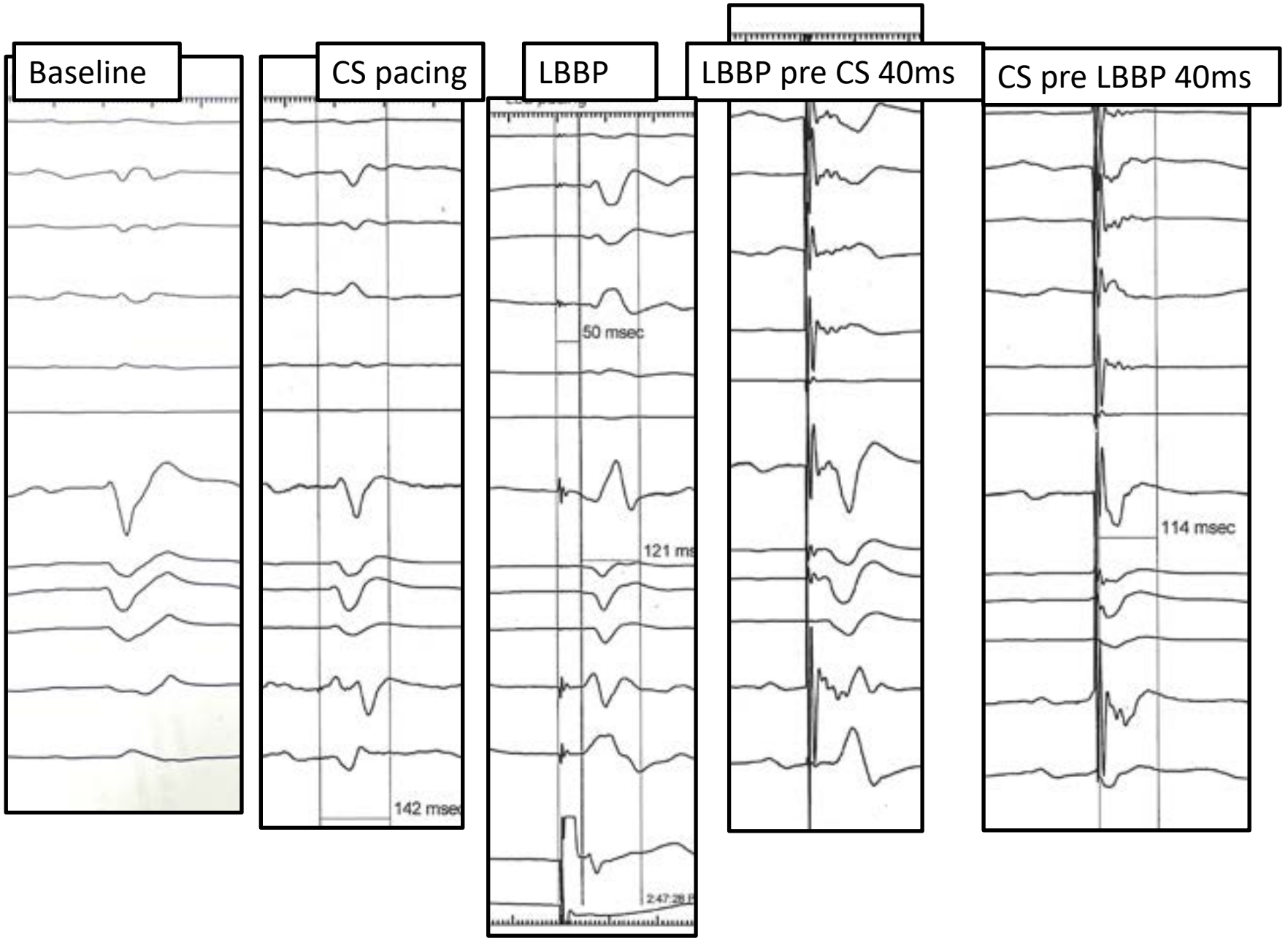


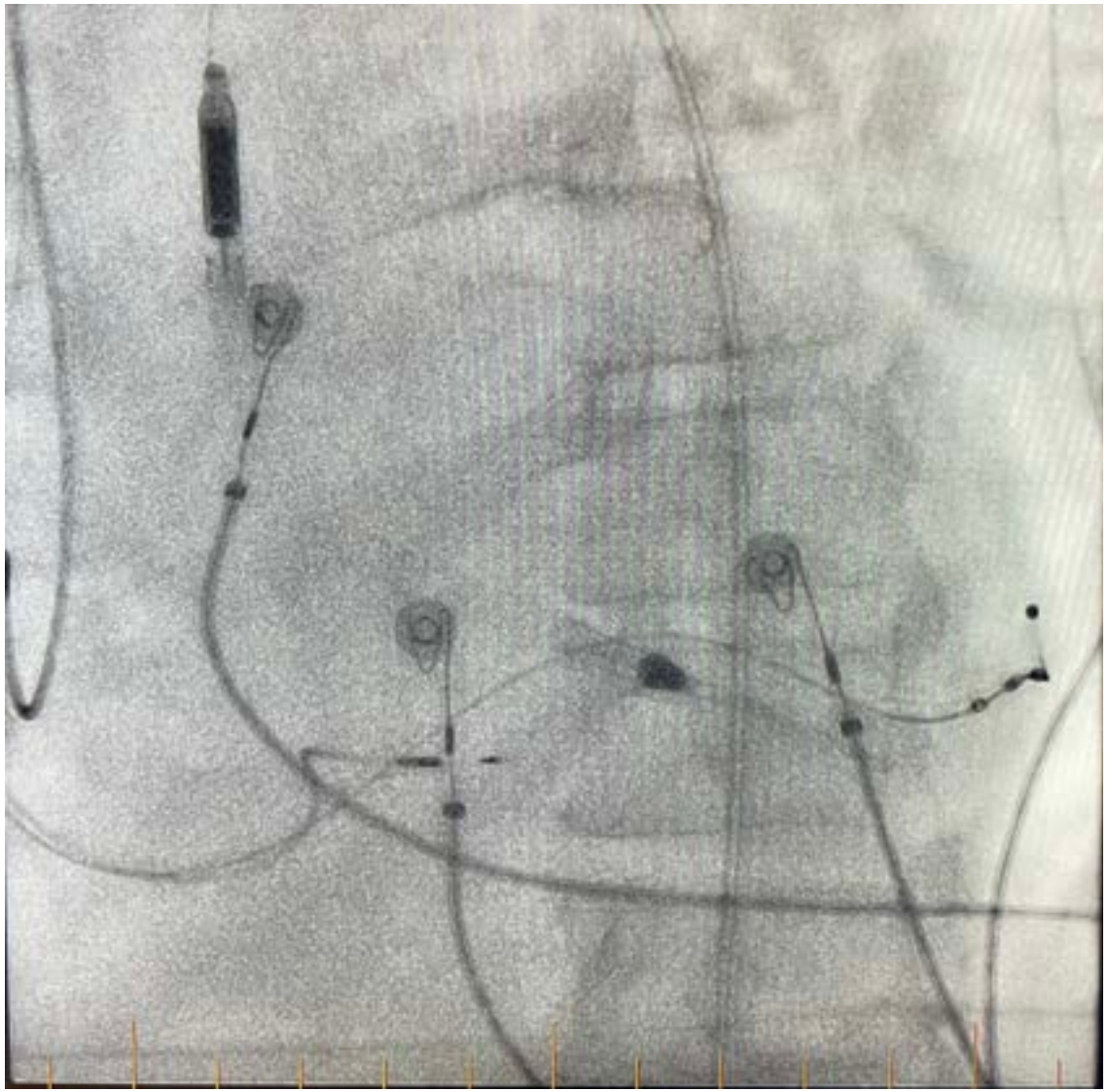
65M late presentation, Ischemic cardiomyopathy, LVEF 5%

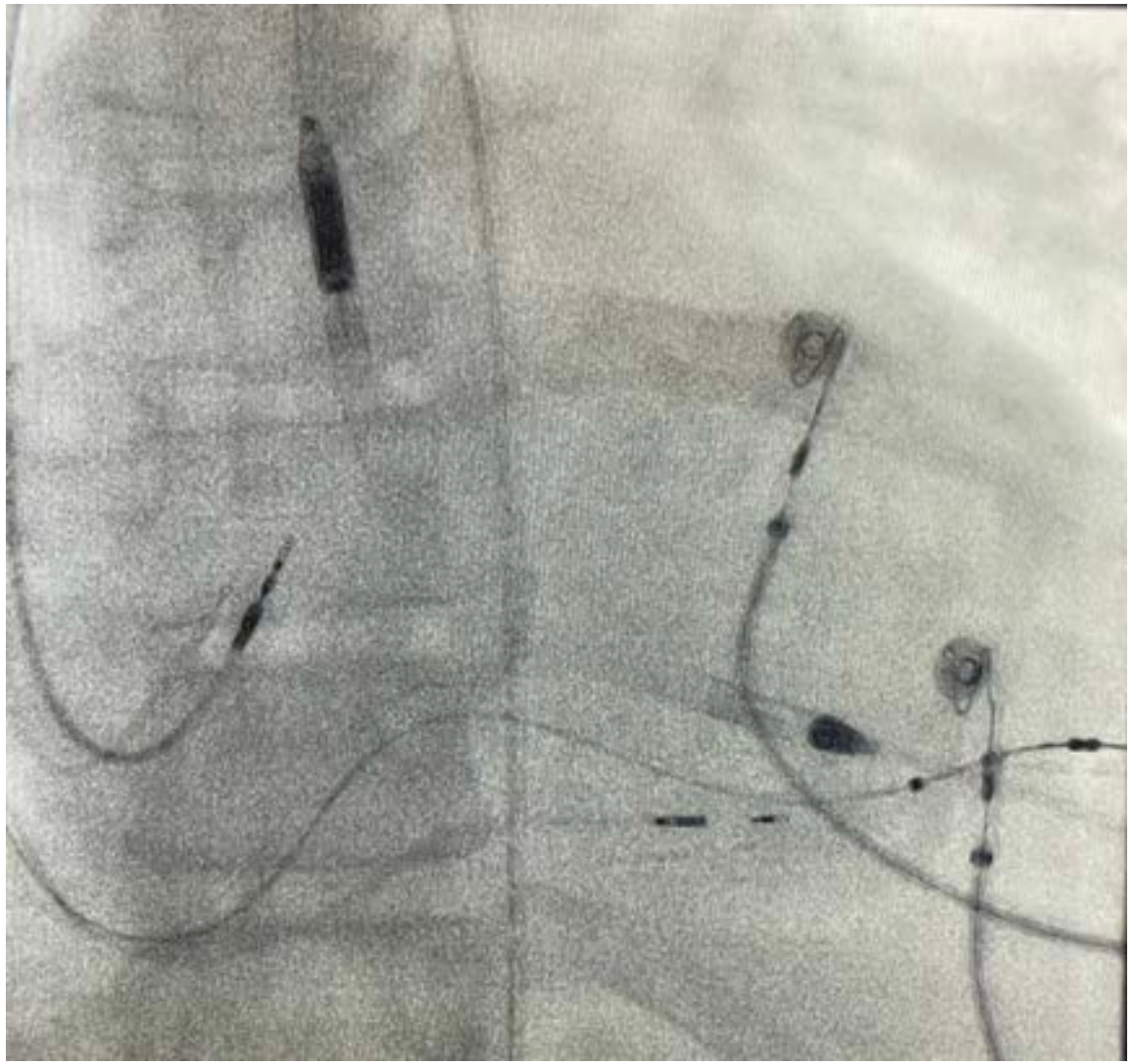
- On milrinone with intra-aortic balloon pump
- No revascularizable options
- Refusing ventricular assist device/transplant

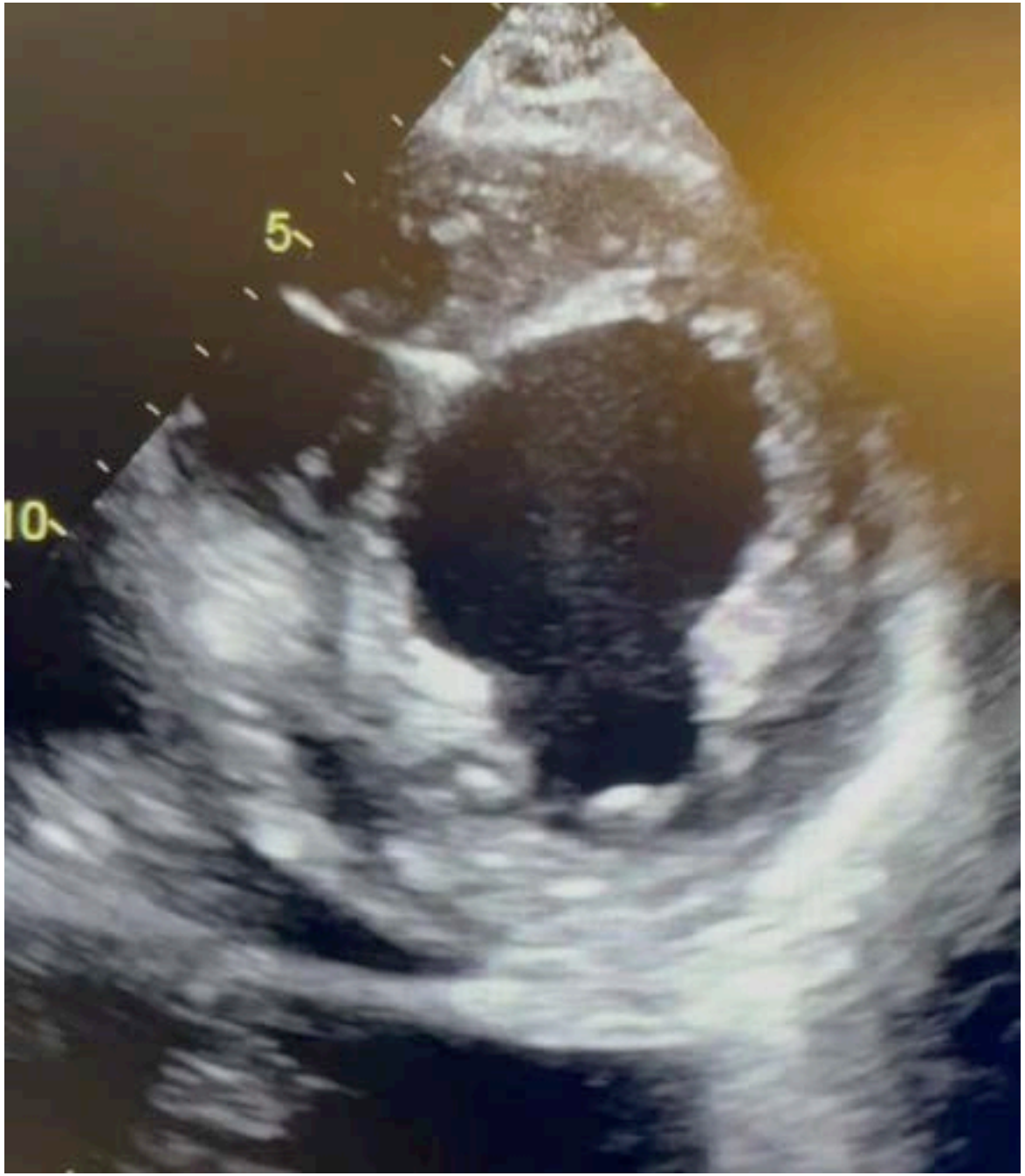


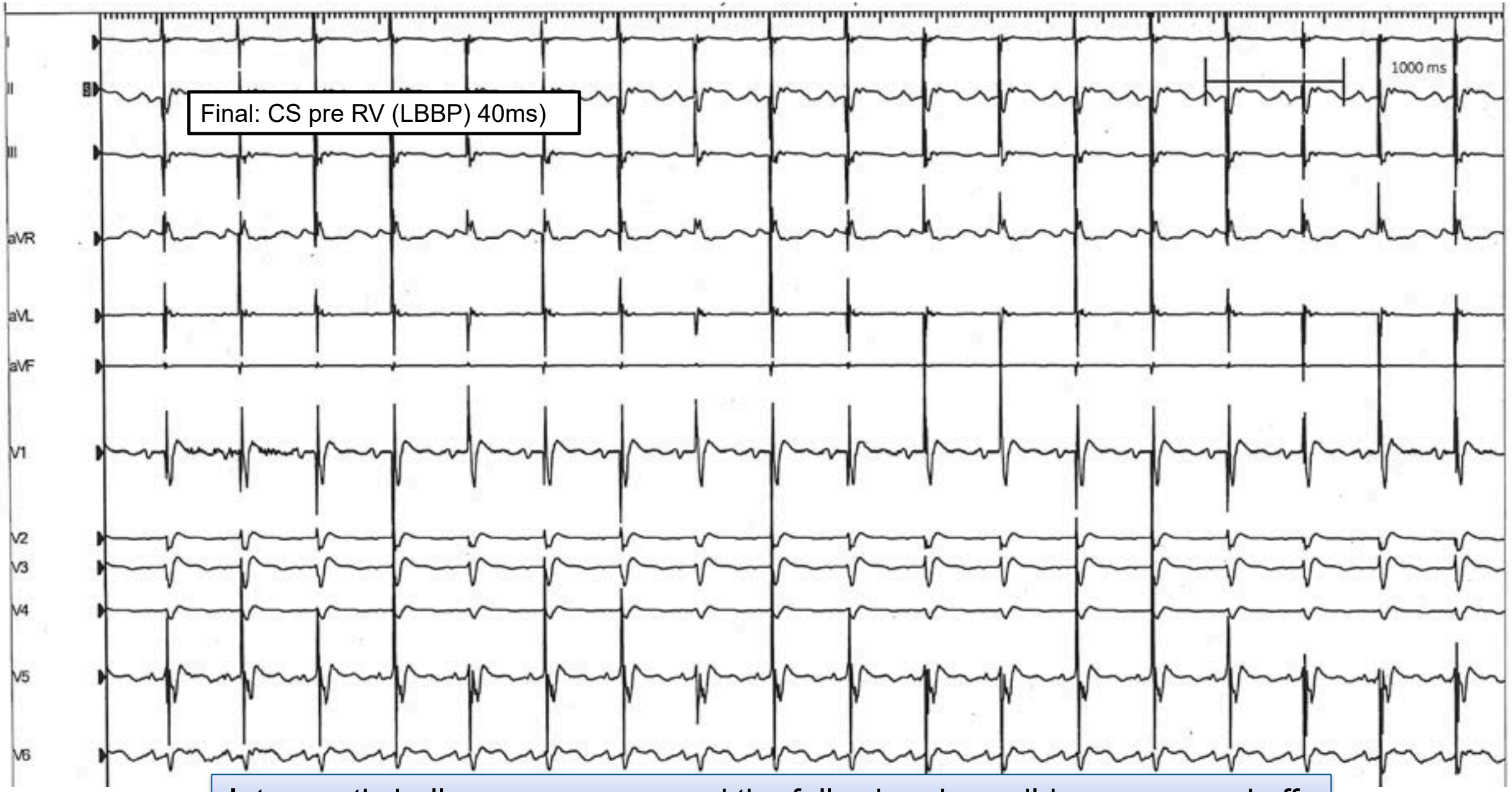



Baseline
CS pacing
LBBP
LBBP pre CS 40ms
CS pre LBBP 40ms
142 msec
50 msec
121 ms
114 msec



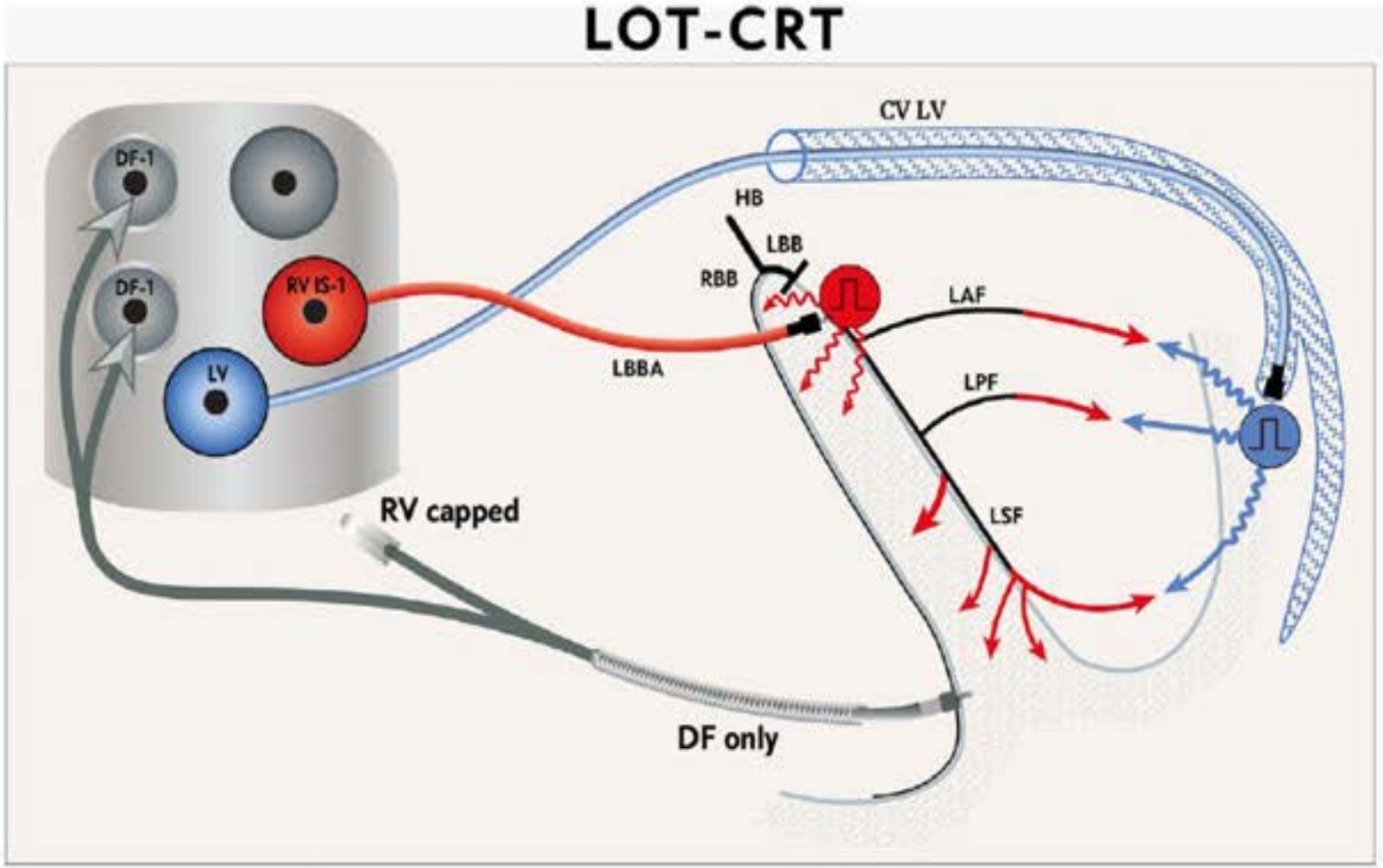


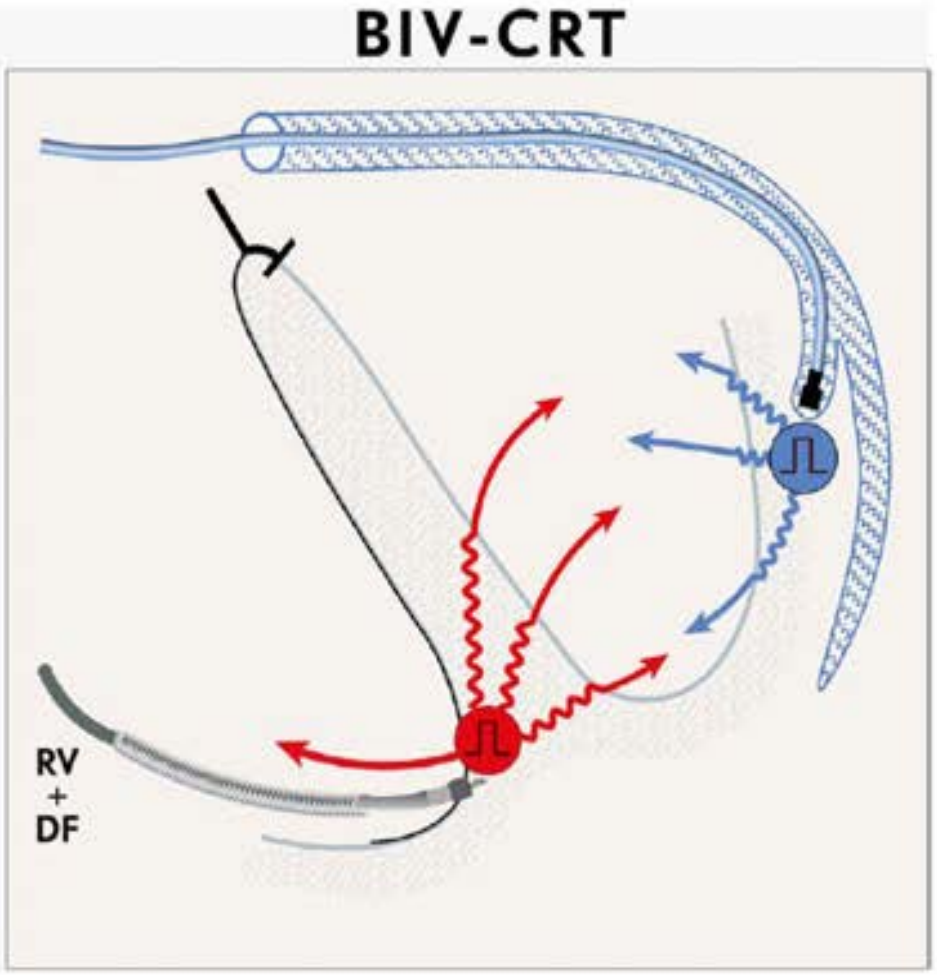

5
10



Intra-aortic balloon pump removed the following day, milrinone weaned off

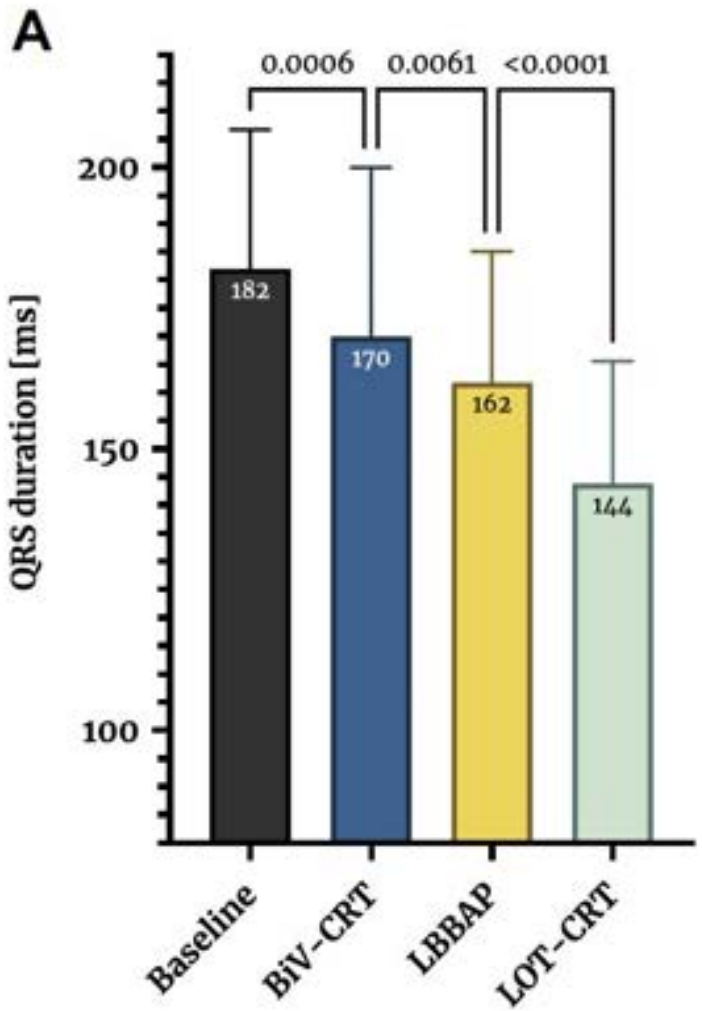
Left bundle branch optimized CRT

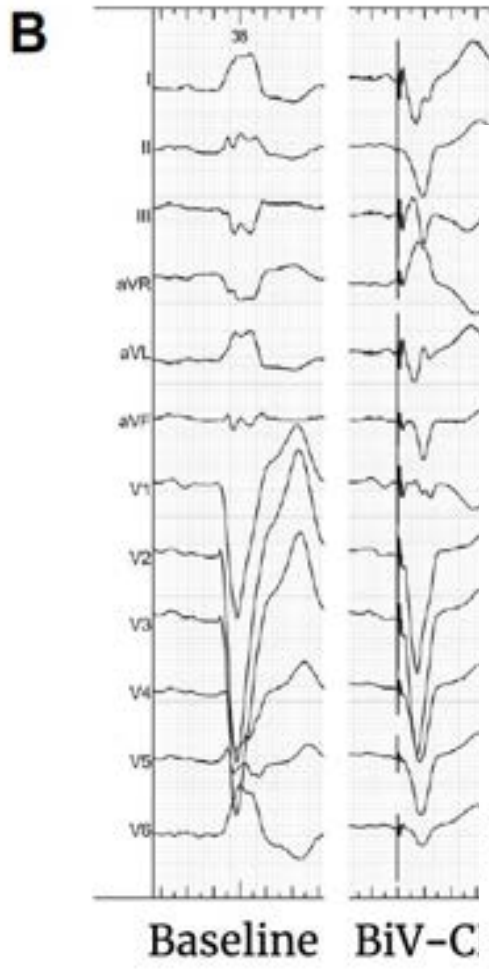


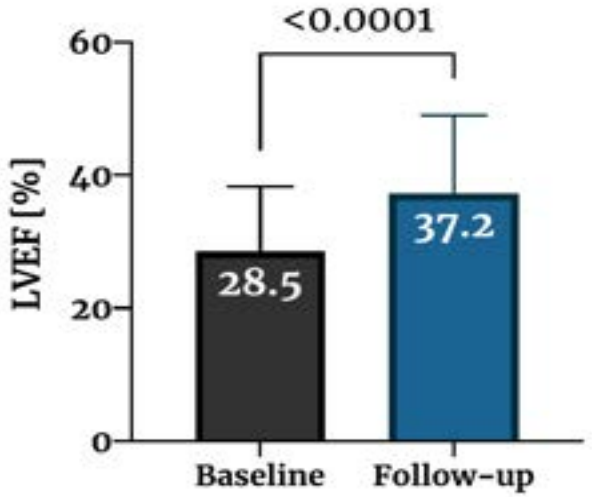


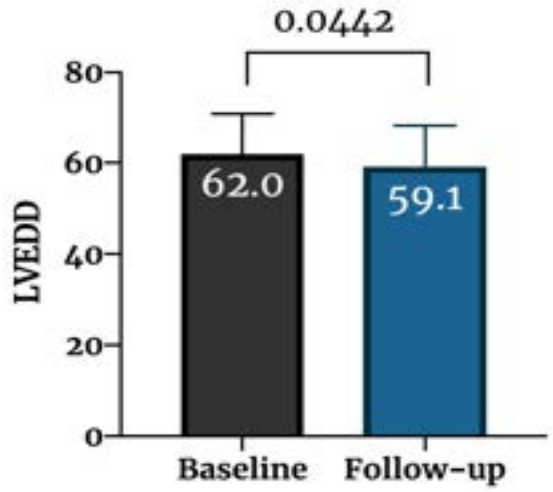
Jastrzebski, Heart Rhythm 2021

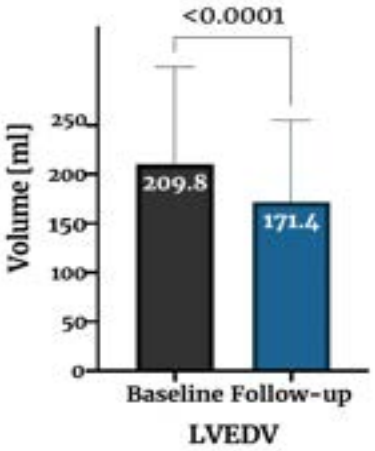
Left bundle branch pacing optimized CRT (LOT-CRT)

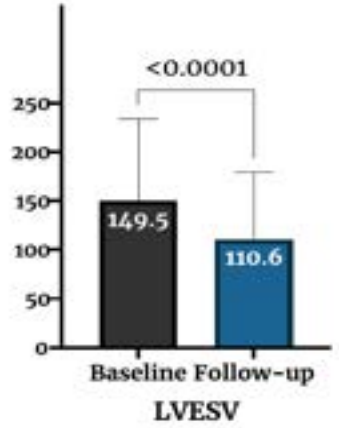


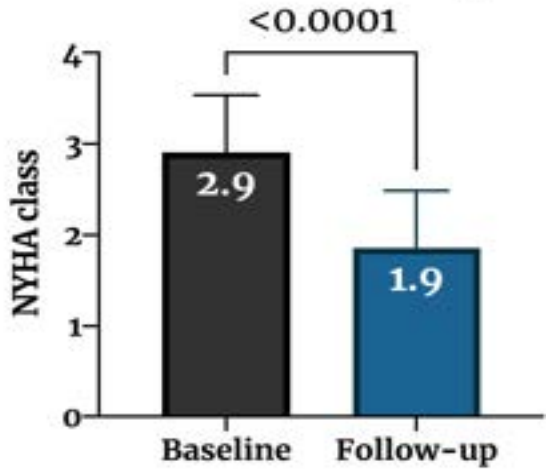








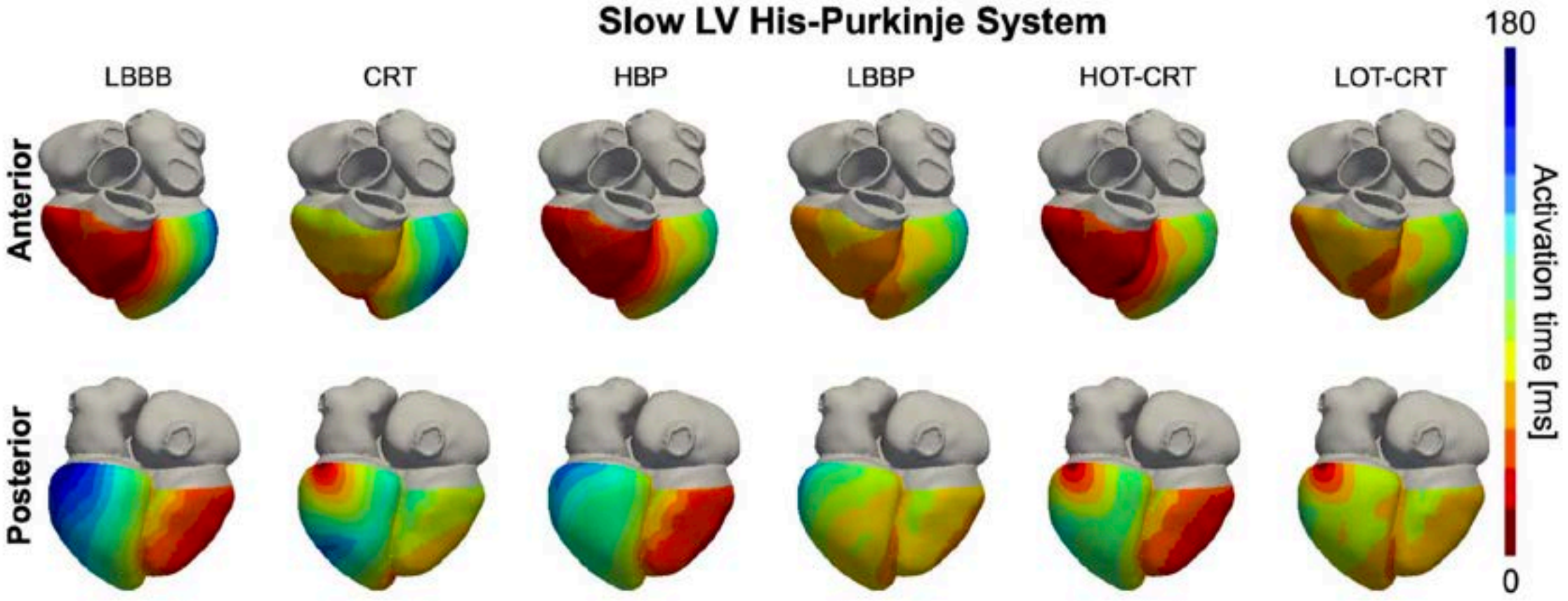




Jastrzebski, Heart Rhythm 2021

Ventricular Electrical Activation Simulations generated from HF patients

- Diffuse conduction disease, septal scar, and lateral scar were simulated
- Electrical synchrony was measured during pacing with varying modalities



Strocchi et al. JCE 2023;1-10

LVAT95

CRT or combination CRT + CSP able to achieve shortest activation time

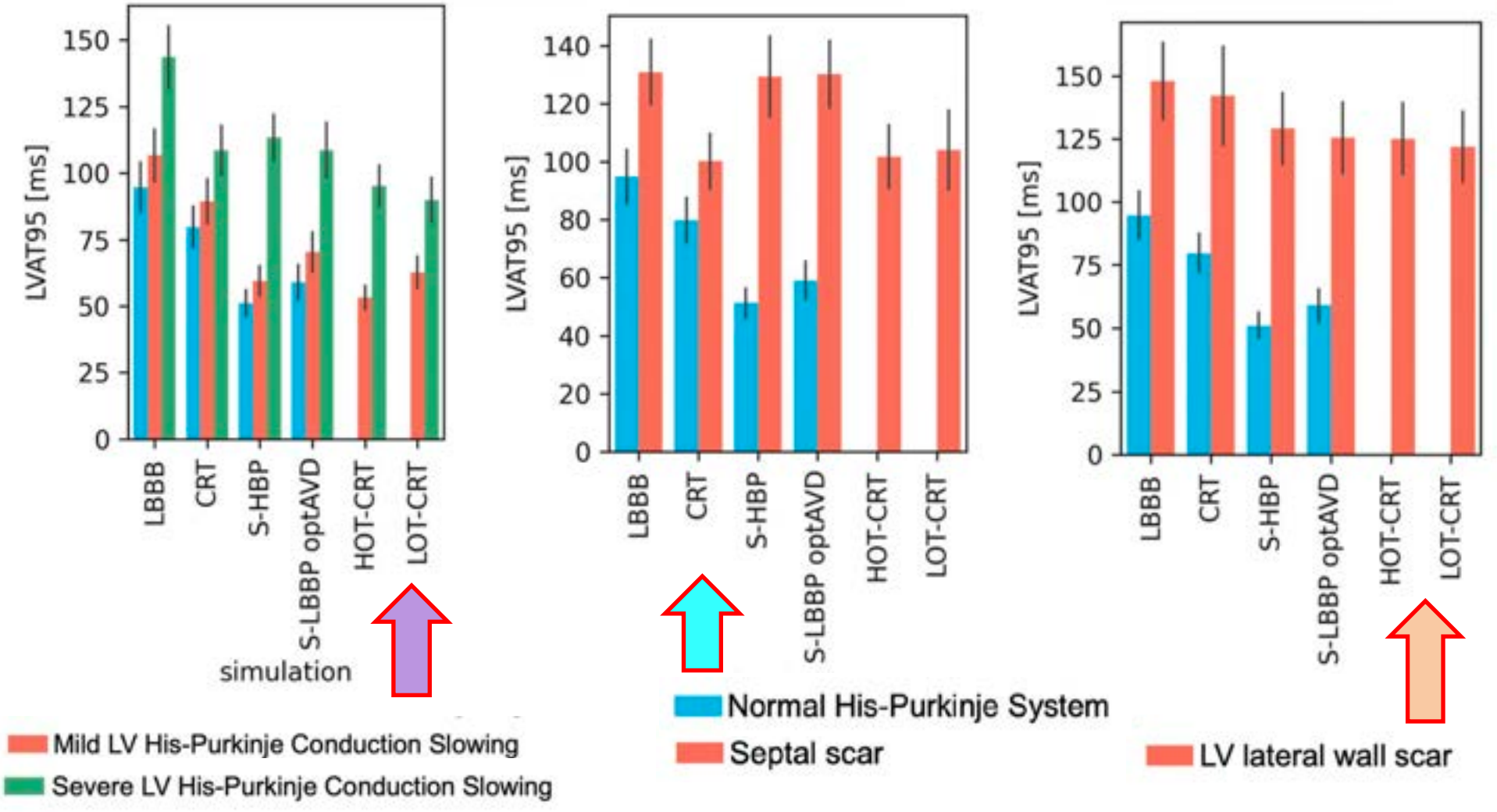
His Purkinje Disease

CRT achieves shortest LV activation

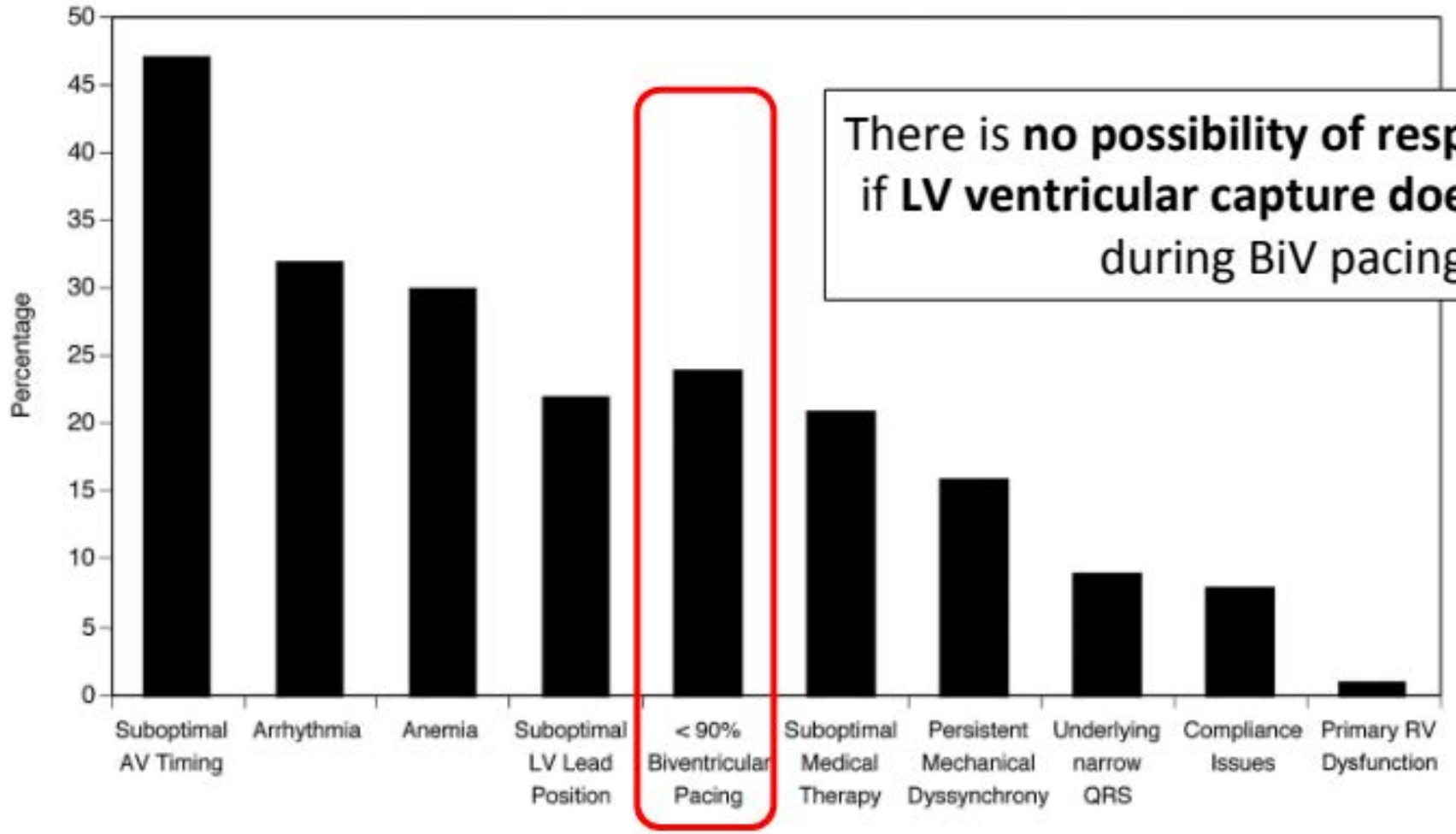
Septal Scar

CSP achieves shortest LV activation

Lateral Scar



MAGNITUD OF RESPONSE TO CRT

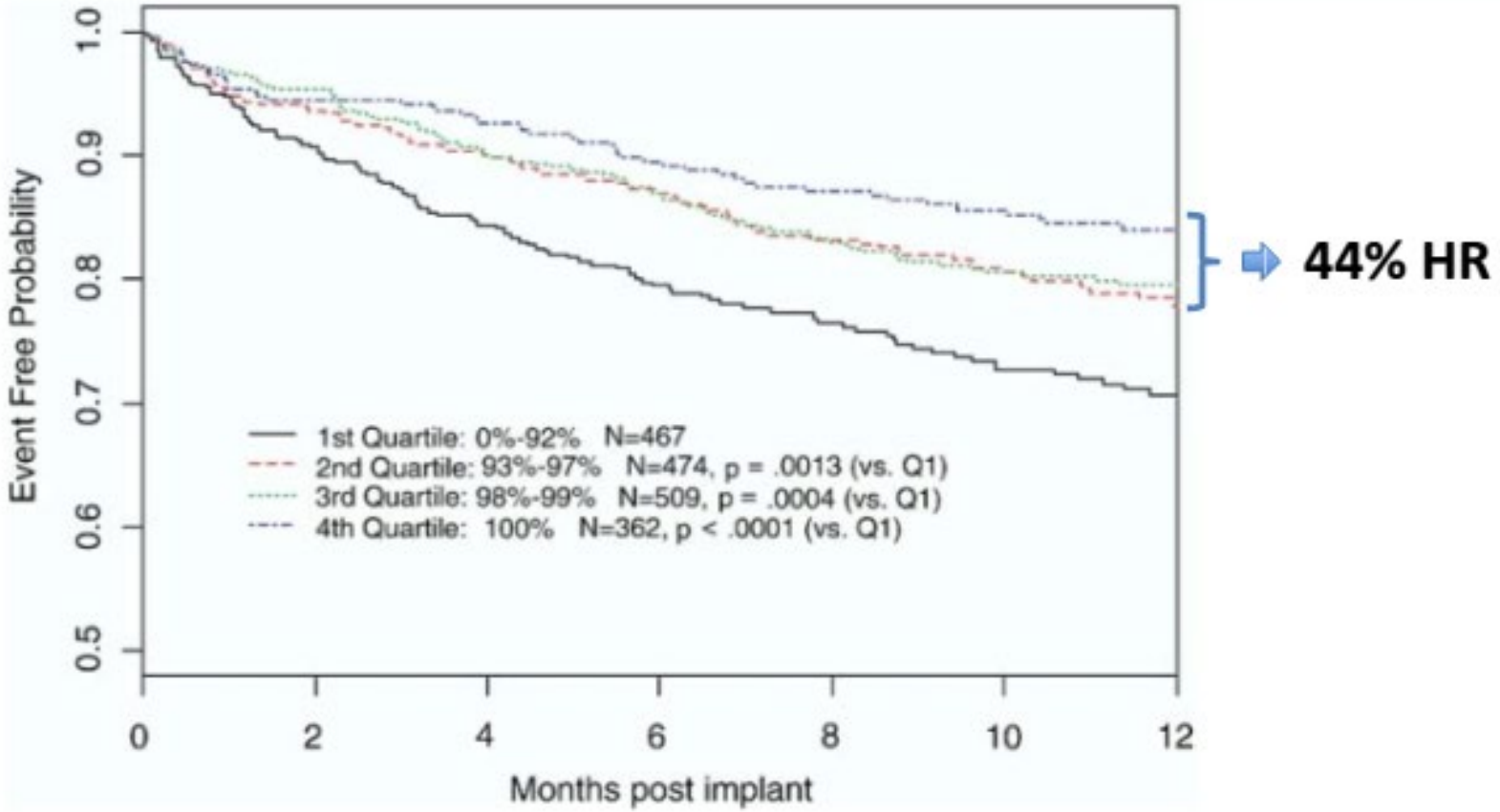


There is **no possibility of response** to CRT if **LV ventricular capture does not occur** during BiV pacing

Ypenburg et al. J Am Coll Cardiol 2009; 53: 483-490

IMPACT OF VENTRICULAR PACING

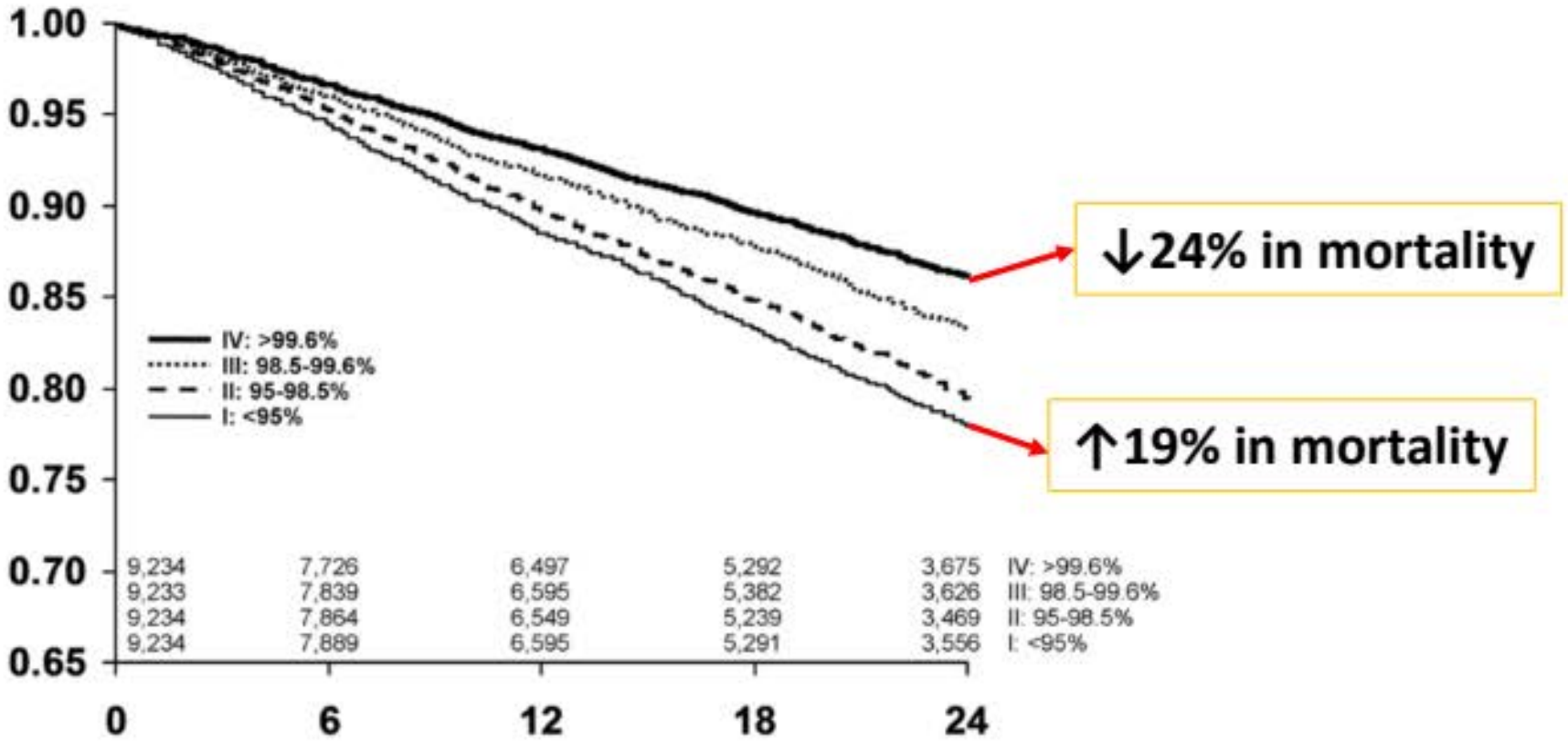
- Small **decreases in CRT pacing** have a significant impact on **long term outcomes**:



Koplan BA. et al. J Am Coll Cardiol 2009; 53: 355-360

IMPACT OF VENTRICULAR PACING

- Small **decreases in CRT pacing** have a significant impact on **long term outcomes**:



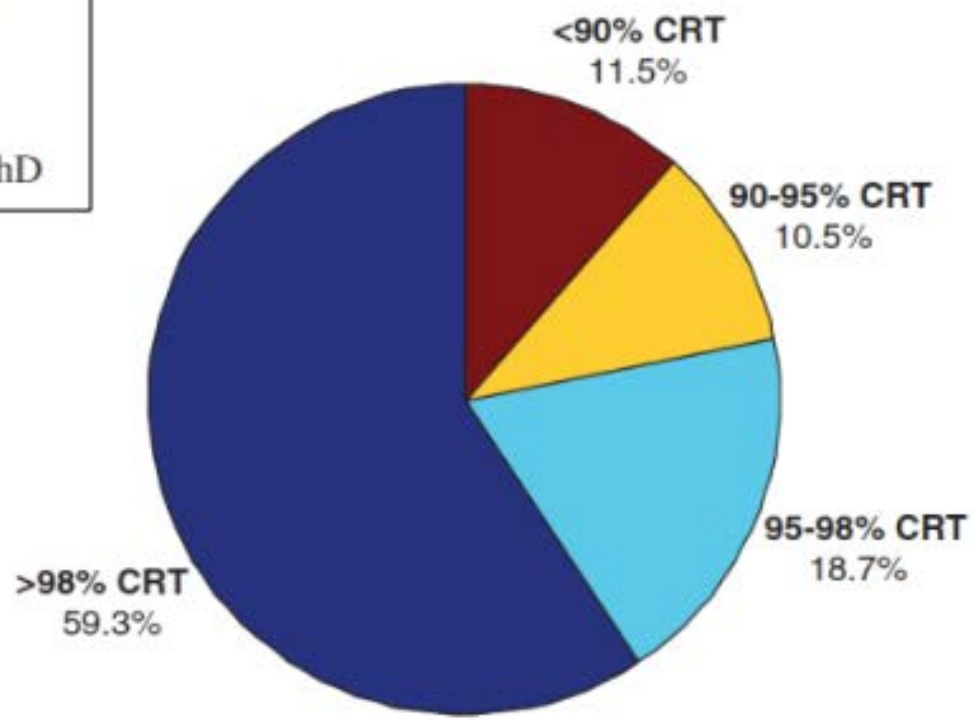
Buch TJ. et al. Heart Rhythm 2011; 8: 1460-1466

Reasons for Loss of Cardiac Resynchronization Therapy Pacing

Insights From 32844 Patients

Alan Cheng, MD; Sean R. Landman, MS; Robert W. Stadler, PhD

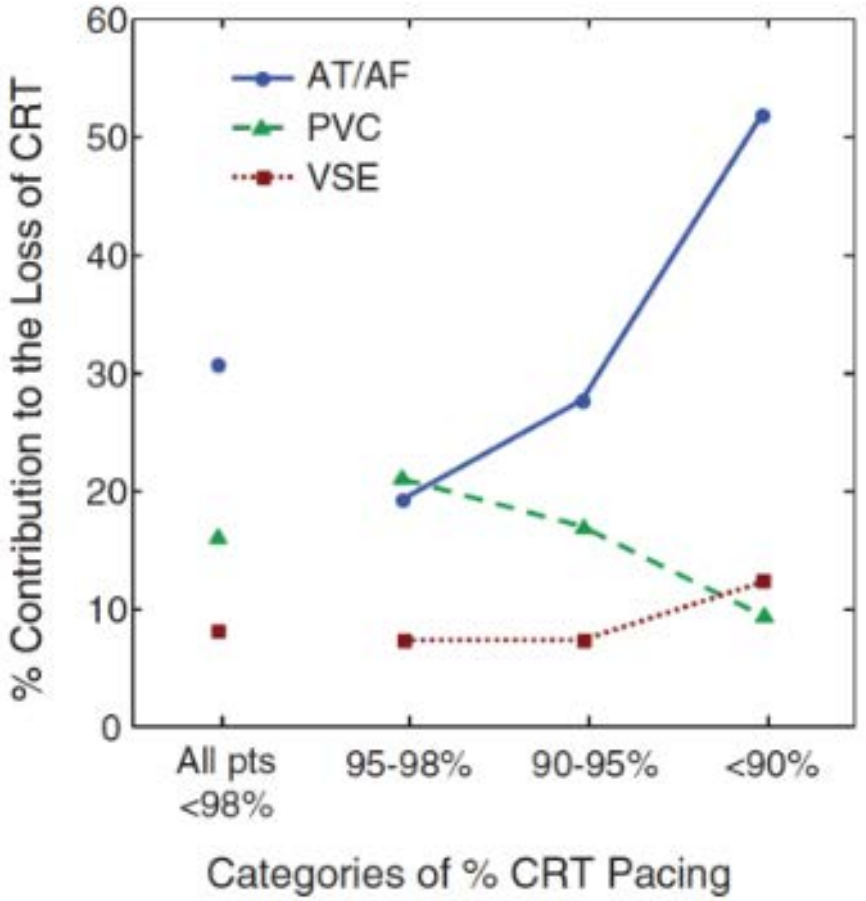
- In 80.768 patients,
- 40.7% <98% CRT pacing.
- Evaluated **sensed events**.



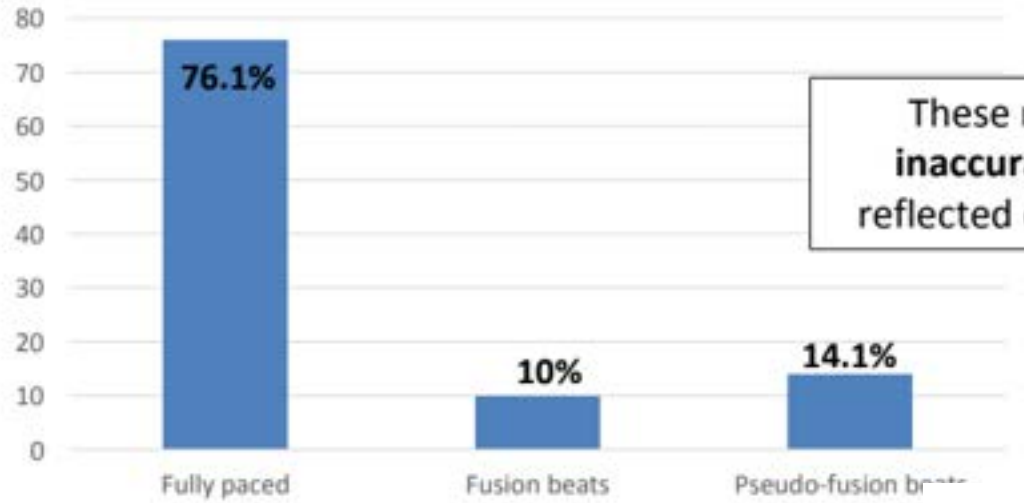
Cheng A et al. Circ Arrhytm Electrophysiol 2012;5: 884-888

- Reasons to loss CRT pacing:

- AT/AF accounted for the largest % of lost CRT.
- An important reason was an inappropriate programming of SAV/PAV interval.
- Another important reason was PVC.

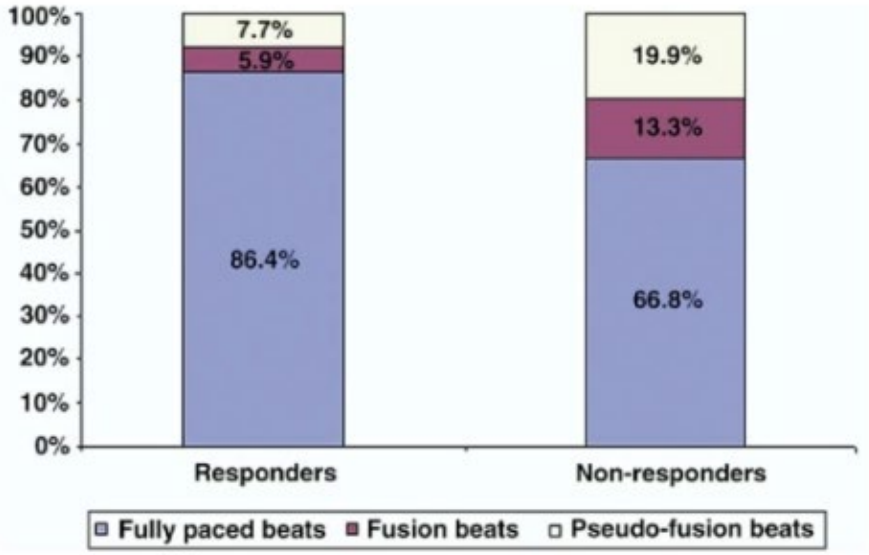


- Patients with a **high degree of BiV pacing (device counters): 95.4±3.2%.**



These results show the **inaccuracy of %V pacing** reflected on **device counters**

- Impact of ineffective capture on clinical response:



Kamath et al. J Am Coll Cardiol 2009; 53: 1050-1055

-2013 ESC Guidelines:

1

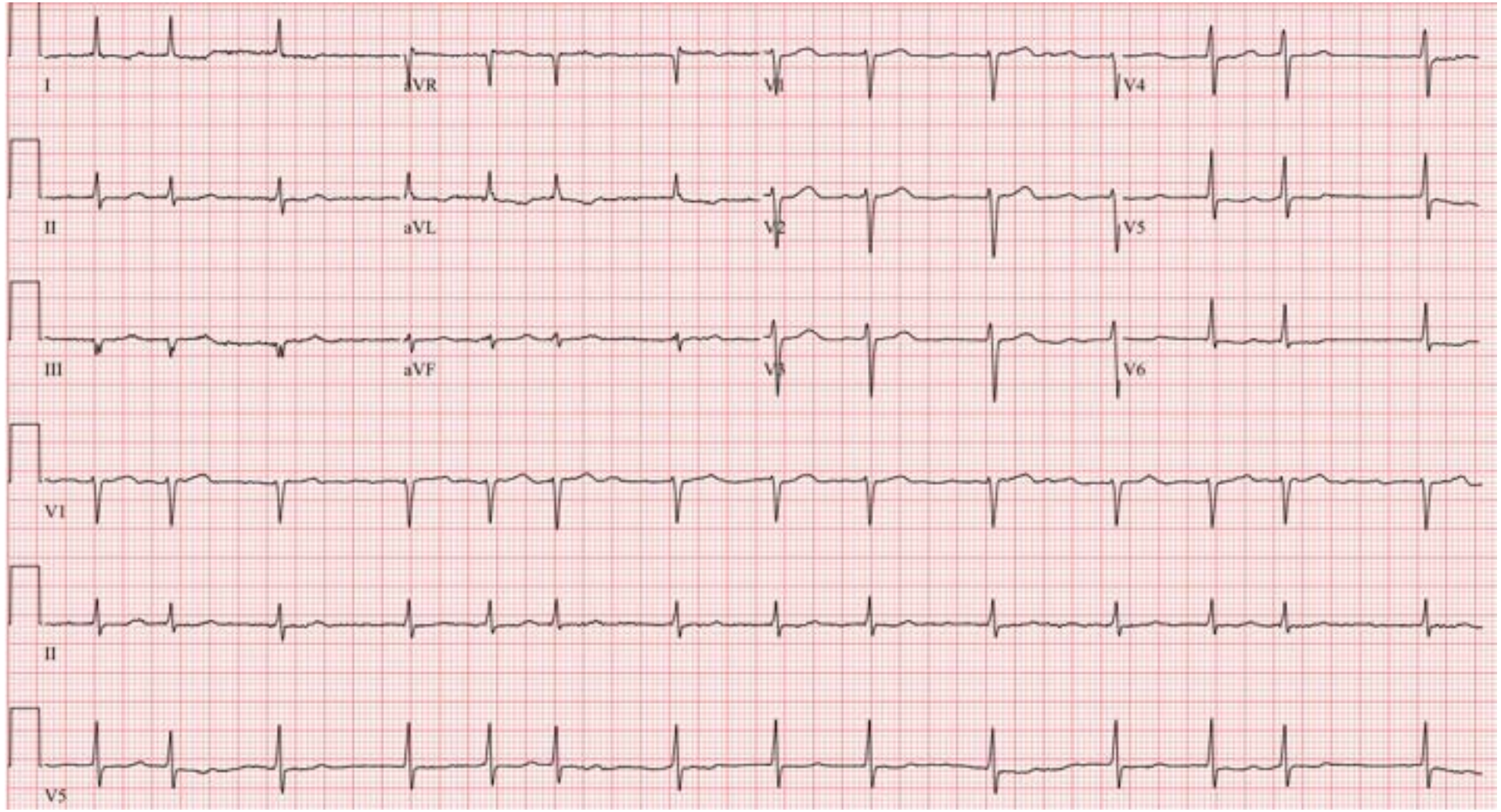
a major determinant of the success of CRT is the effective
d f biventricular pacing. Data from large registries showed that a high percentage of biventricular pacing (≥99%) was a prerequisite for successful CRT pacing and that AF was a major determinant of loss of biventricular pacing (see also section 3.2.2).[67–69] Failure of CRT was associated with new-onset AF during
fo [w151,w152] A particular aspect of AF patients is that AF
r th fast ventricular rate and irregularity may interfere with
ade e biventricular pacing delivery. Competing AF rhythm—by creating spontaneous, fusion or pseudo-fusion beats—may reduce the rate of real biventricular capture. A careful analysis of the surface ECG is mandatory and in some cases a Holter recording could be useful, to assess the completeness of biventricular capture and to exclude pseudo-fusion, which the device algorithms might register as paced beats.[96] In most AF patients with intact AV

2

How to assess Effective BiV Pacing?

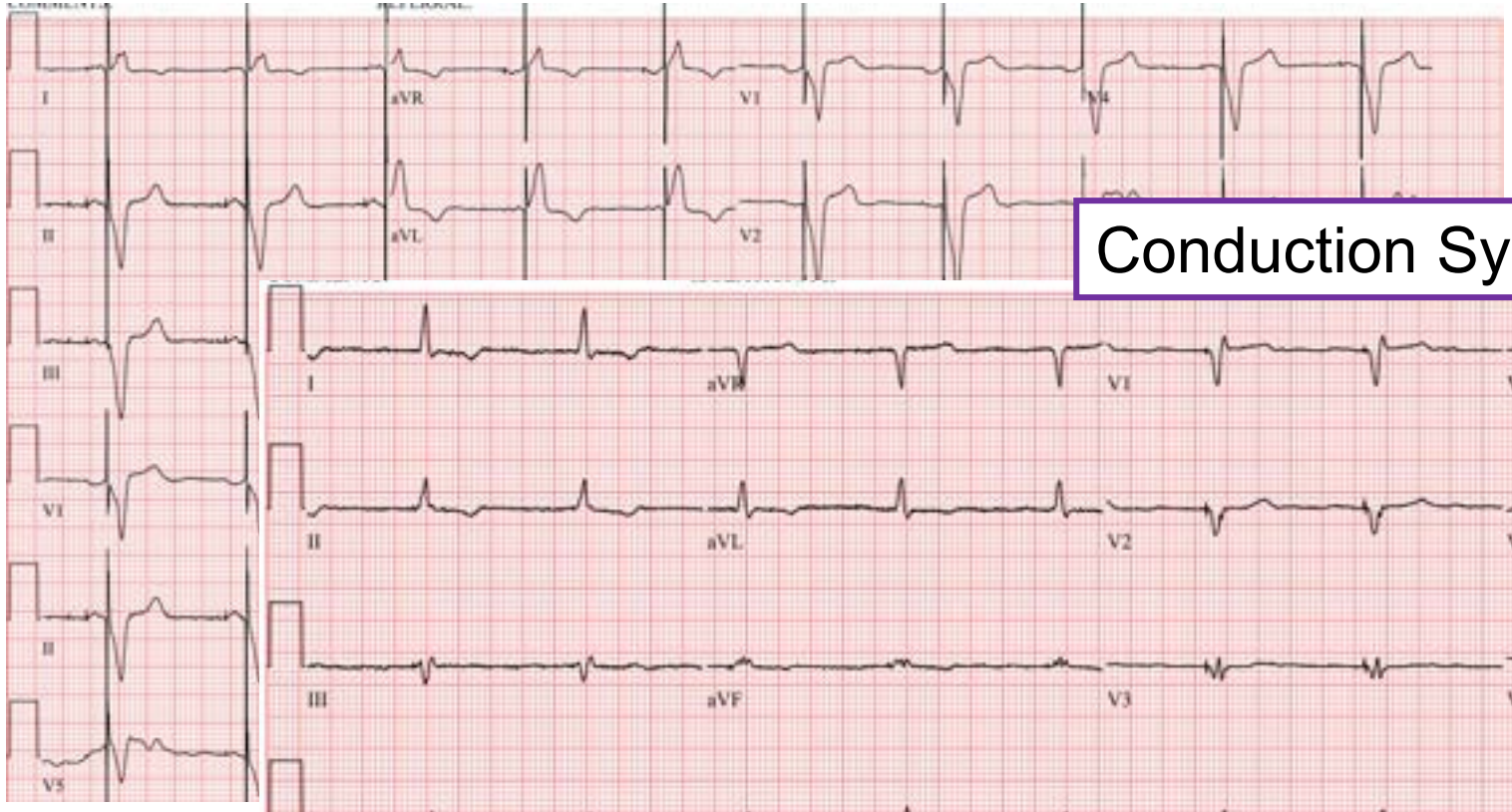
1. A high % of BiV pacing **does not confirm** effective LV pacing.
2. Reasons for **ineffective LV pacing** are:
   i. **Loss of LV capture**.
   ii. **Pseudo-Fusion**: AVI too long or effective pacing is prevented by intrinsic AV conduction (atrial fibrillation).
3. To confirm effective pacing: **surface ECG**.

80F Pre-op TAVI. Severely enlarged LA with chronic AF. Severe TR, EF 45%

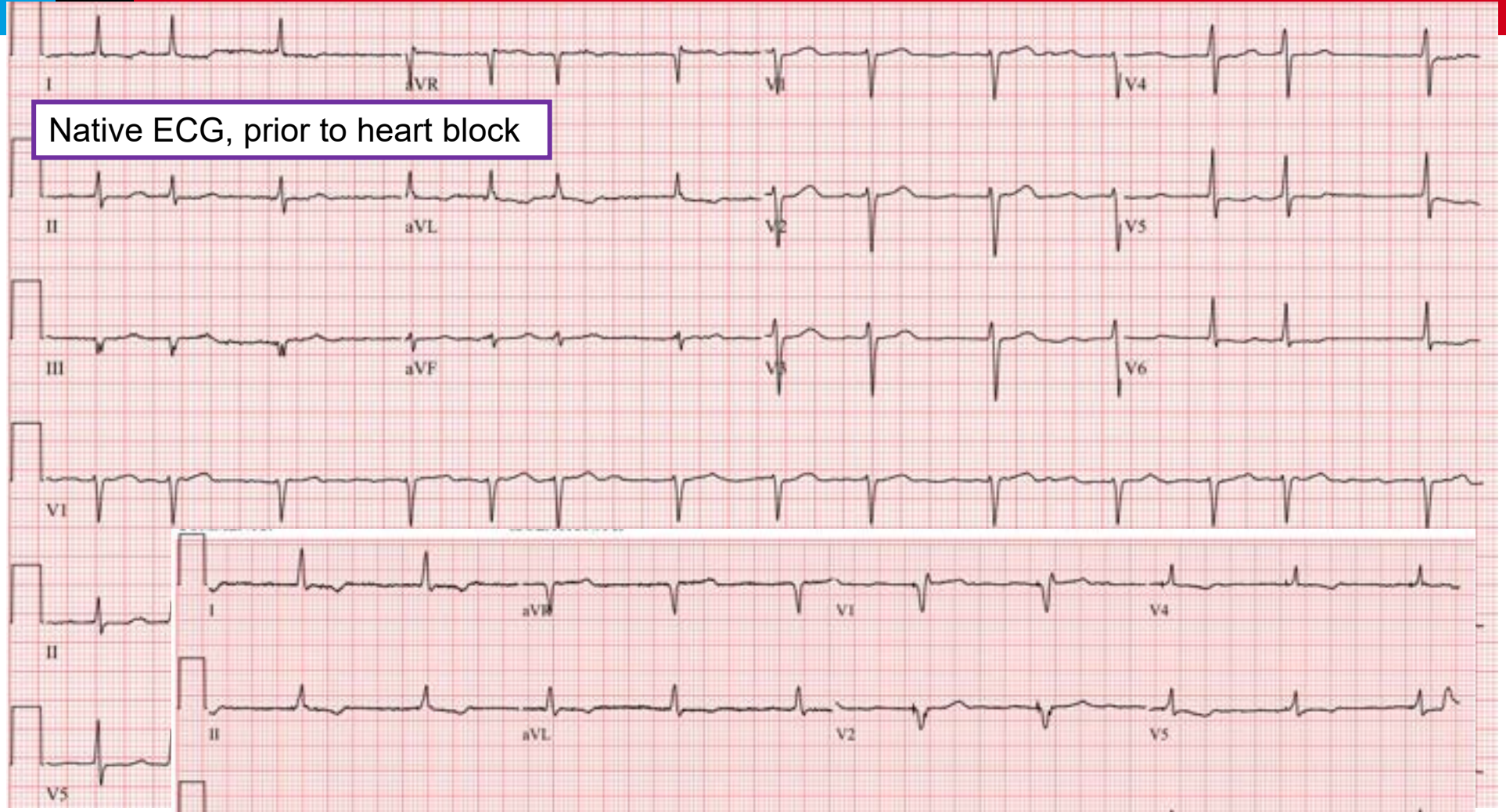


80F Post-op TAVI develops complete heart block, dependent on temporary pacing wire. What should we do now?

Standard RV pacing



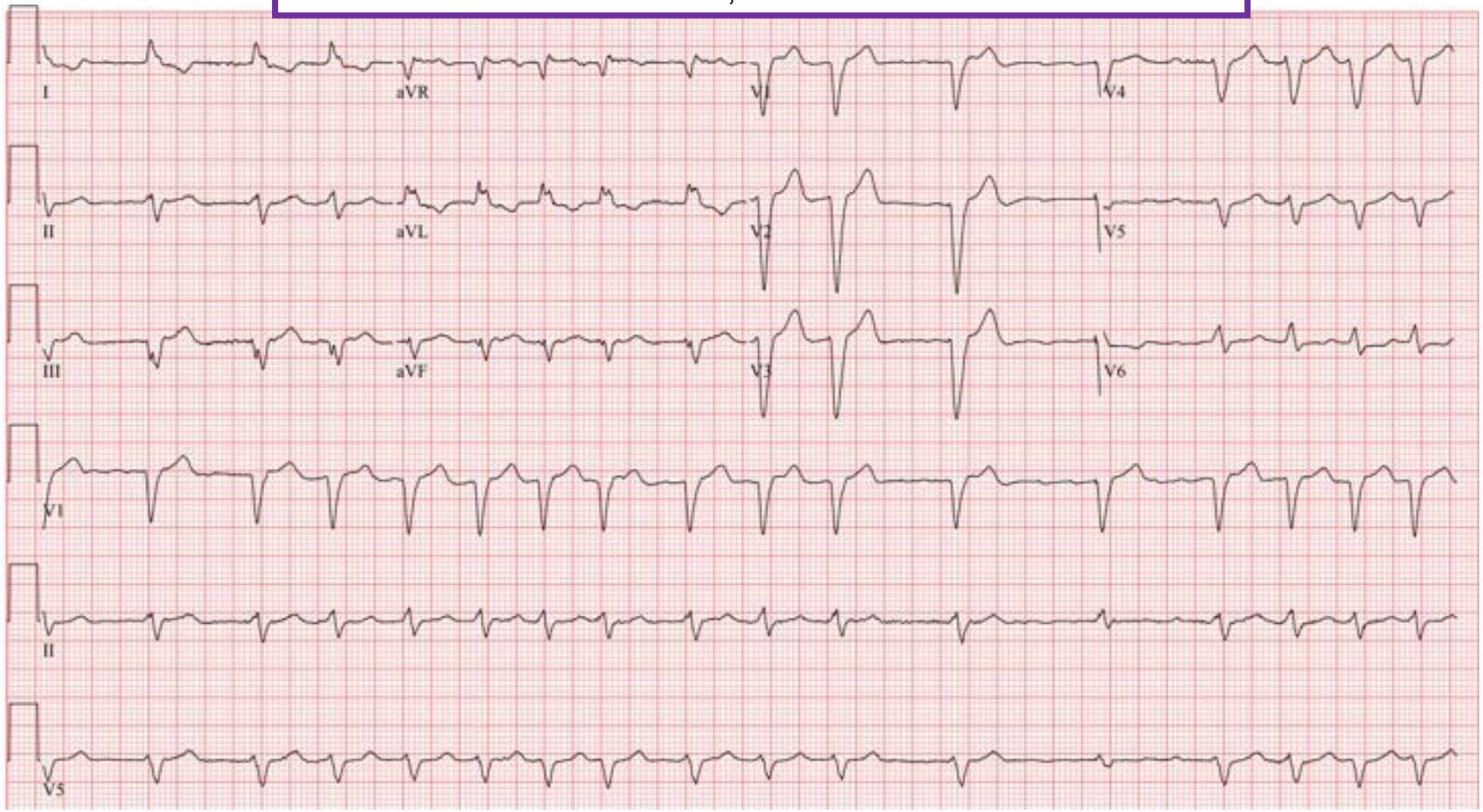
Conduction System Pacing

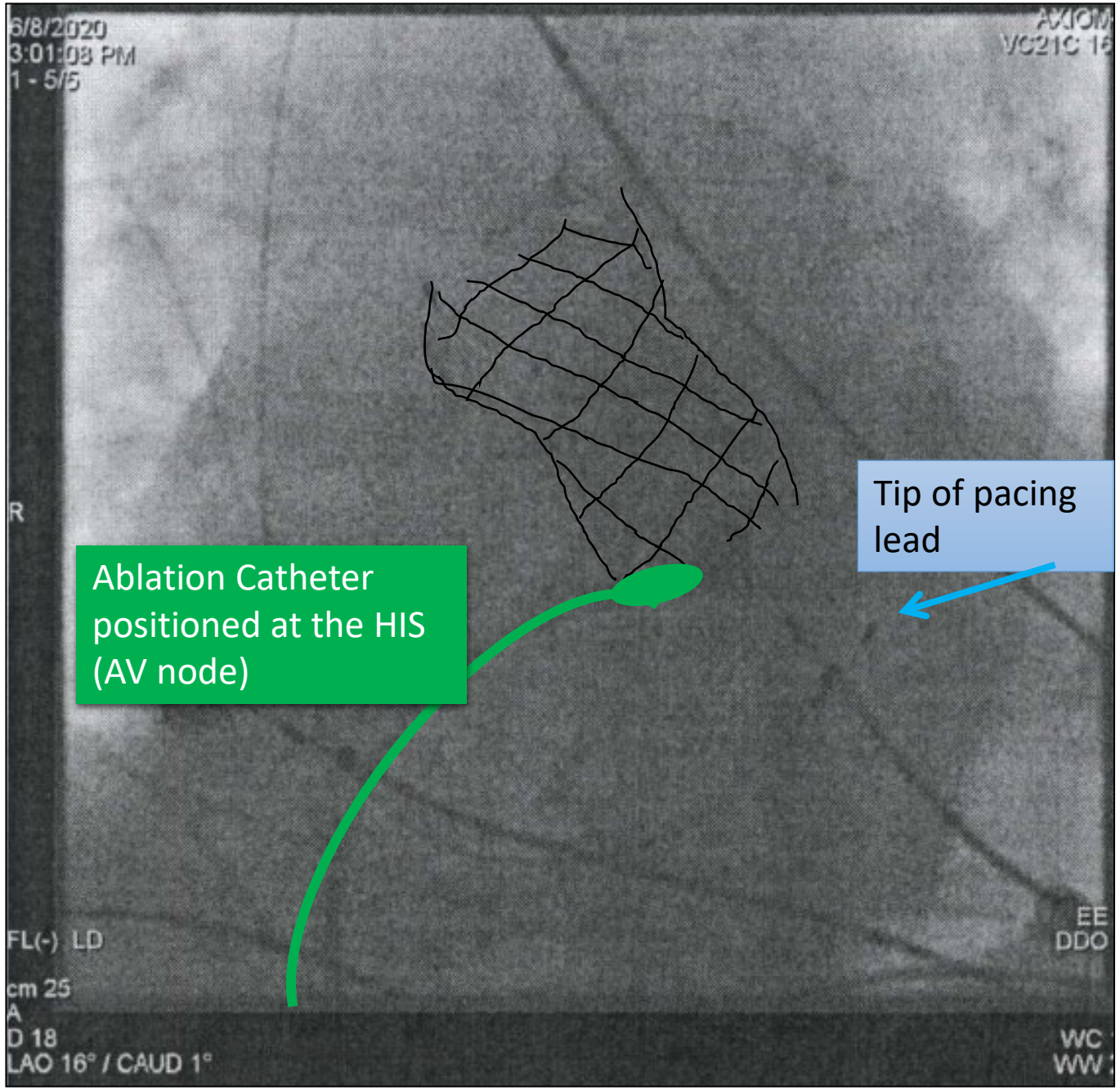


Left bundle branch pacing

1 week later, presents to ED with pulmonary edema.

AV node conduction recovers, but with new LBBB due to TAVR.




6/8/2020
3:01:08 PM
1 - 5/5
R
Ablation Catheter positioned at the HIS (AV node)
Tip of pacing lead
FL(-) LD
cm 25
A
D 18
LAO 16° / CAUD 1°
EE
DDO
WC
WW

80F Post AV node ablation

28-OCT-1939 (80 yr)
Female

Room:
Loc:1

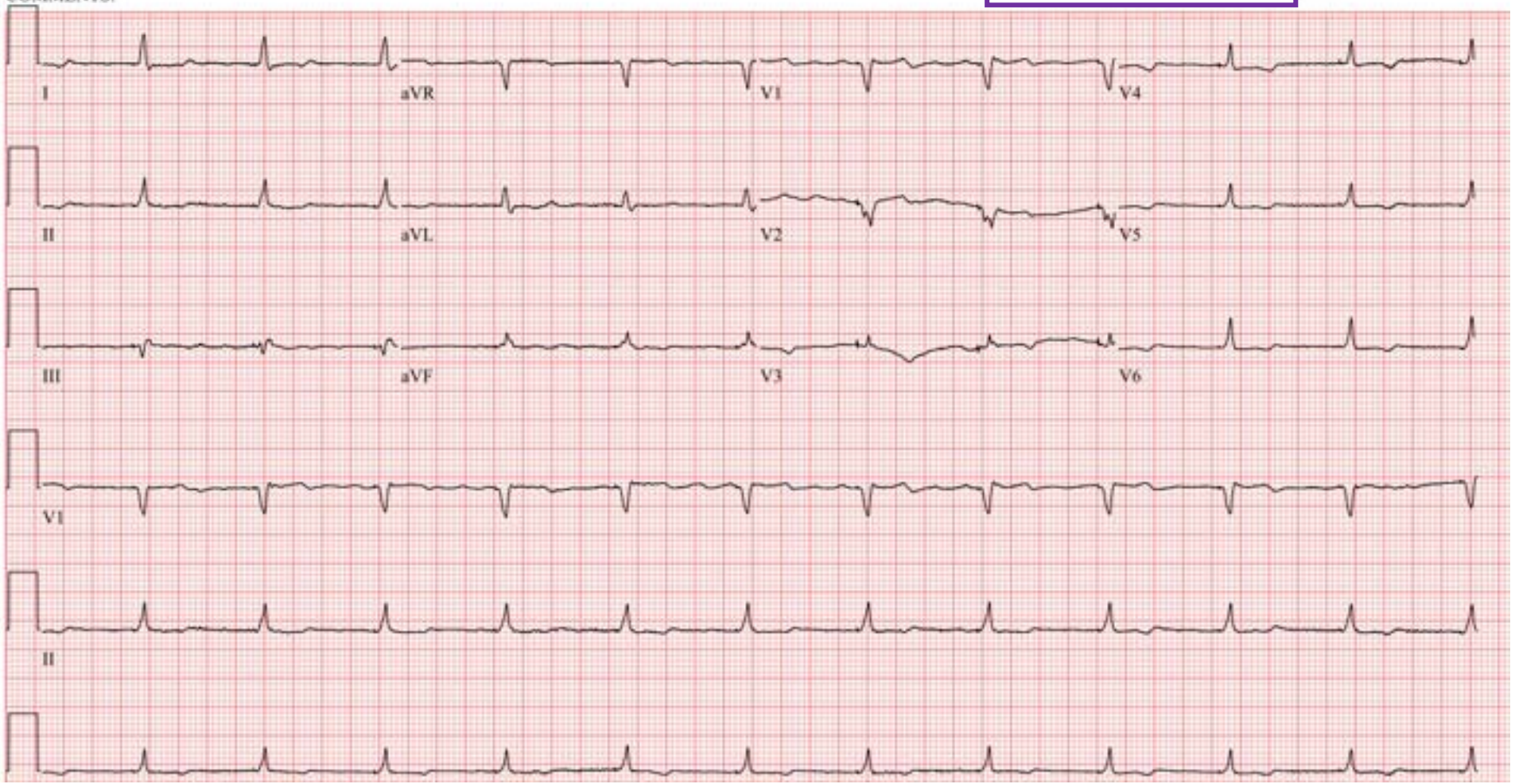
Vent. rate	71	BPM
PR interval	*	ms
QRS duration	98	ms
QT/QTc	408/443	ms
P-R-T axes	* 31	245

Electronic ventricular pacemaker

Technician:* Nurse
Test ind:

Post AV node ablation Threshold 0.5V@0.4ms

COMMENTS:



Séance Q&R

Tous les panélistes

UN GRAND MERCI!

N’oubliez pas de compléter l’évaluation de la séance.

Ce n’est pas terminé! Veuillez-vous rendre dans le hall d’exposition (Samuel ABC) pour une pause santé, puis à la salle de bal Champlain pour la séance plénière 2 intitulée *Clinical Pearls and Conundrums in HF Clinical Care* qui débutera à 15 h 00.