

Unusual Aspects of Bundle Branch Blocks

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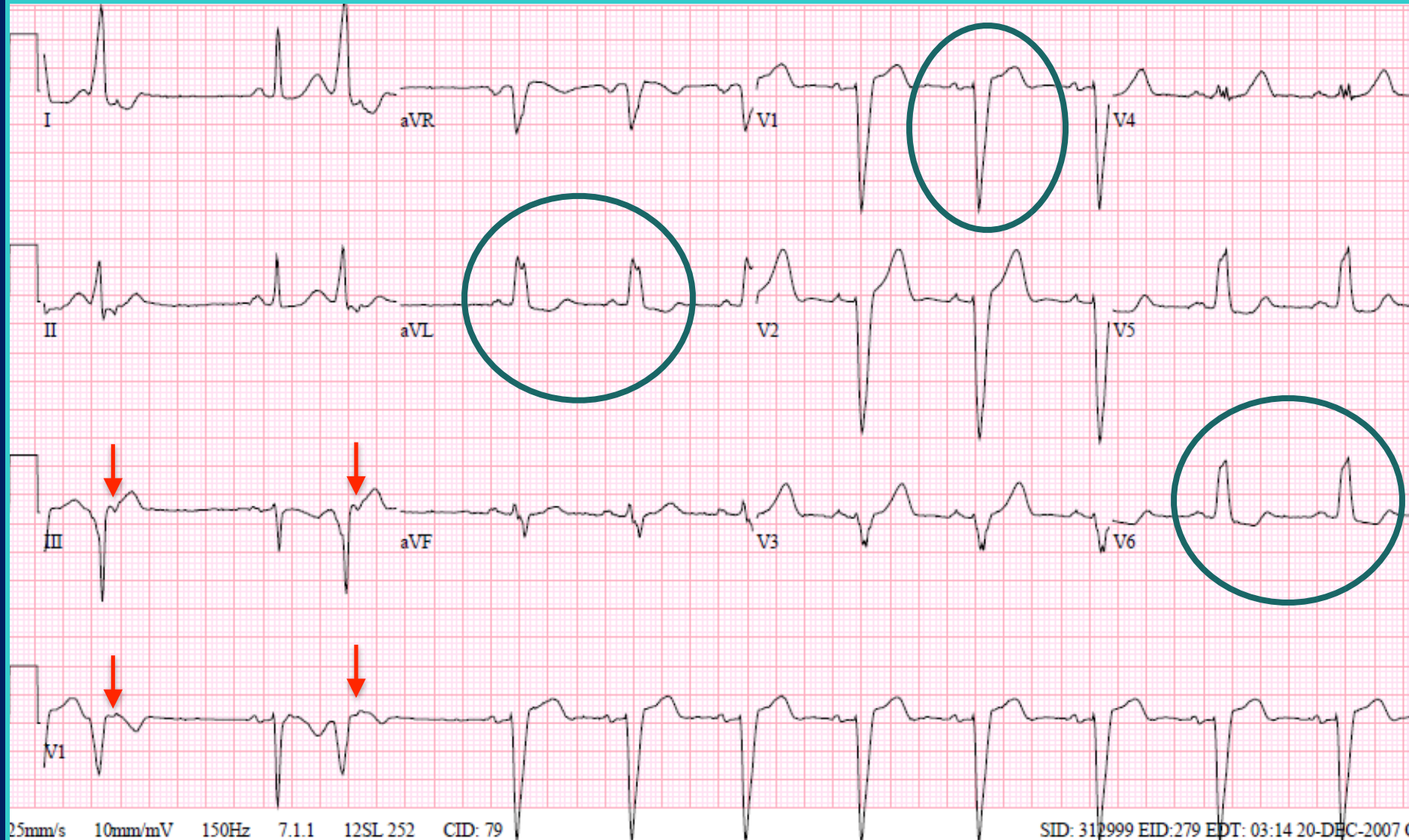
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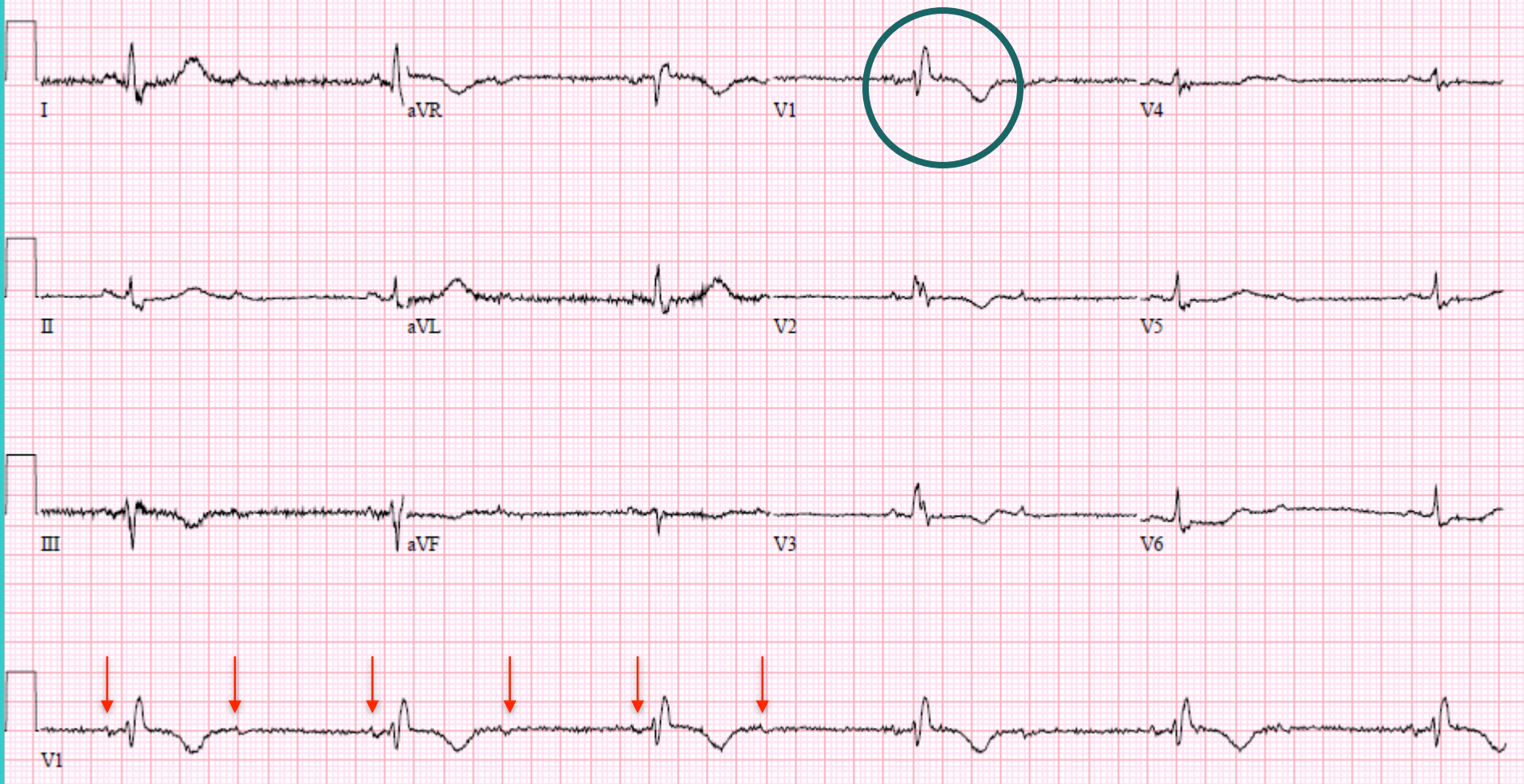
Objectives

- Brief ECG case report
- Review anatomy of ventricular conduction system
- Etiologies of BBB's
- “Functional” Bundle branch and fascicular aberrancy
 - Rate-related BBB
 - Tachycardia-dependent
 - Bradycardia-dependent



17-Dec-2007: 66 y.o. woman with long history of LBBB (routine ECG)

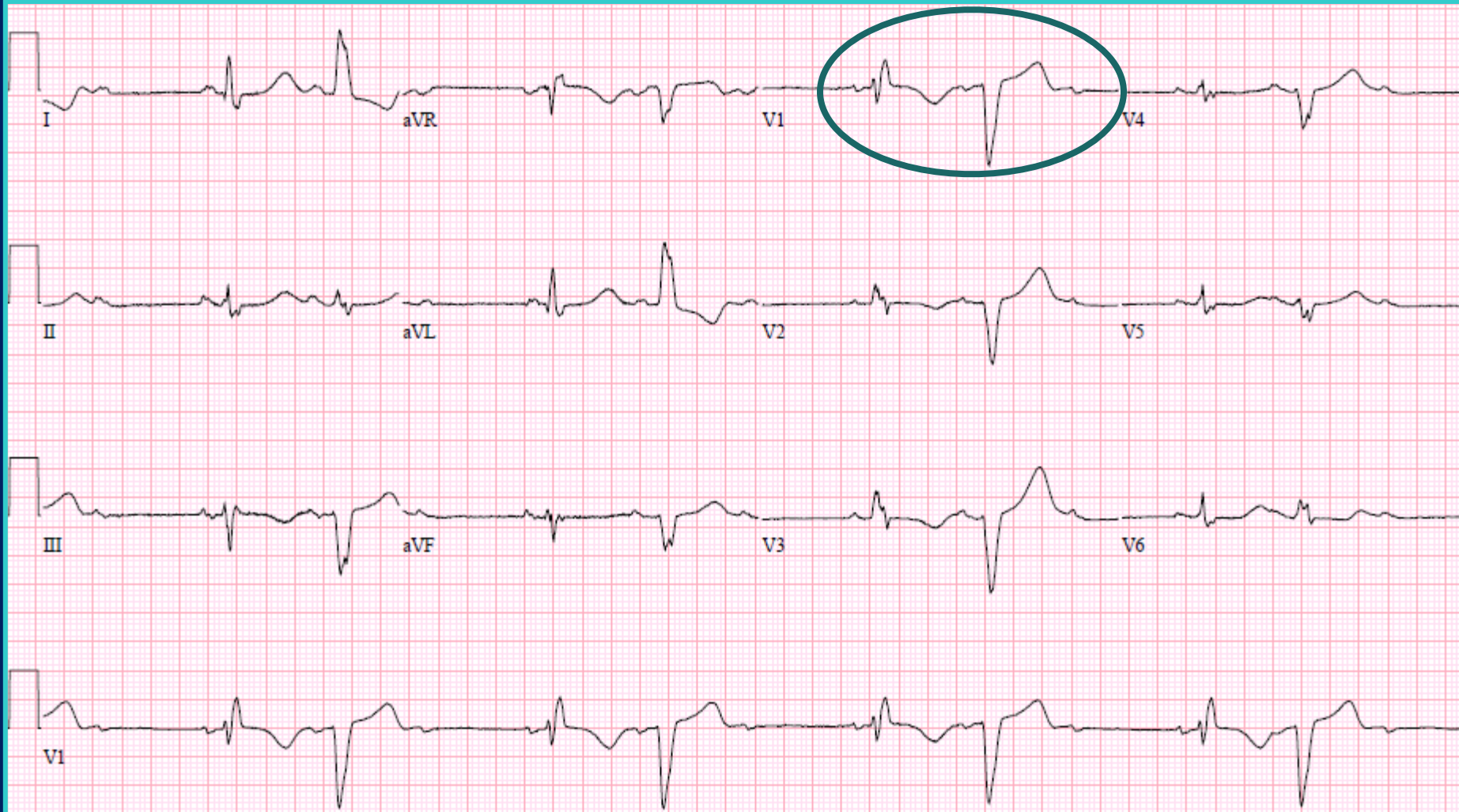
Note: 2 PVCs with retrograde VA conduction (arrows)



~ Six years later.....19-June-2013: 71 y.o. woman (CC: fatigue; HR 35 bpm)

Note: she now has **RBBB**
and
2nd degree AV block with 2:1 conduction

(left bundle must now be working!)

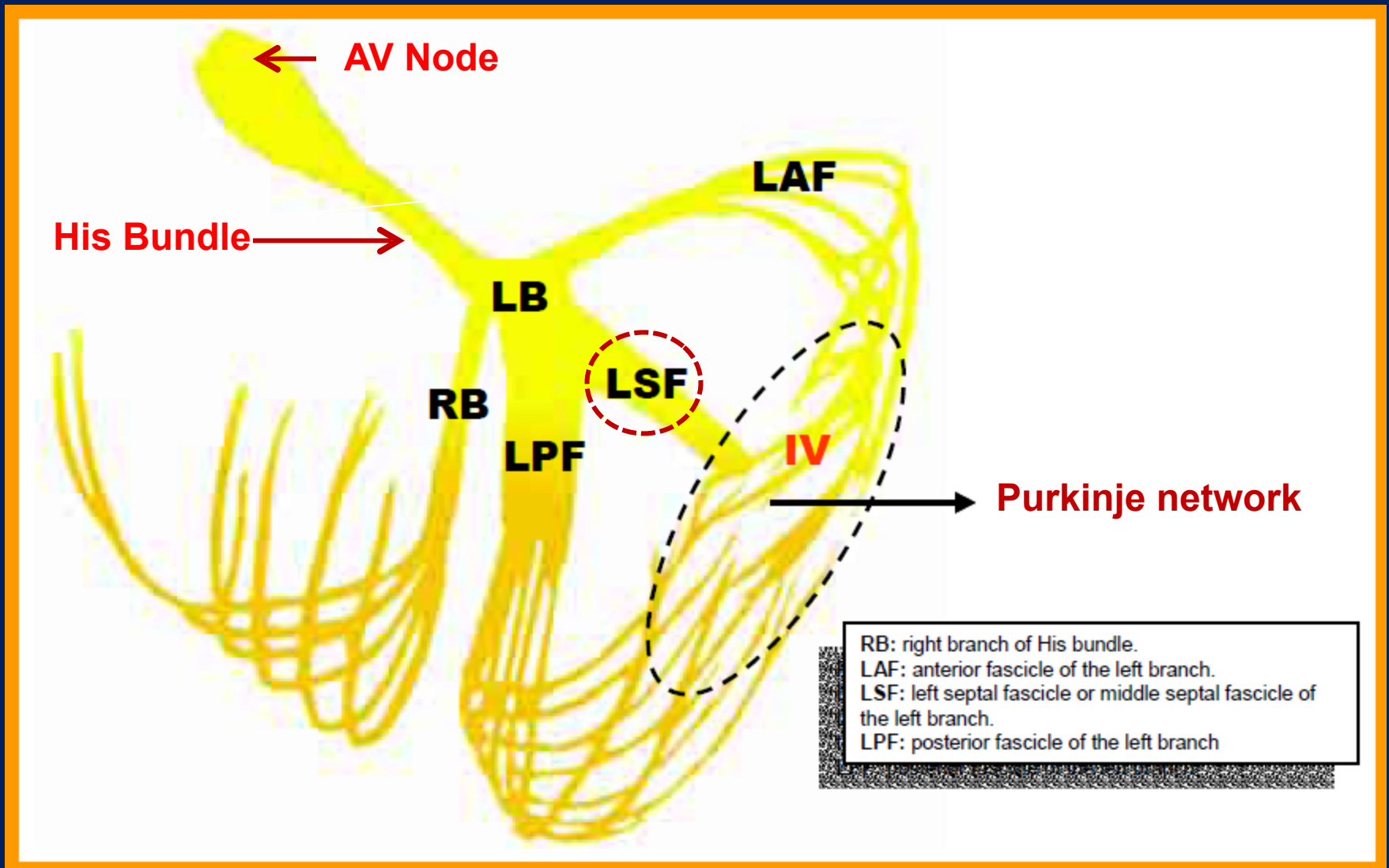


19-June-2013 (same day): 71 y.o. woman (CC: fatigue)

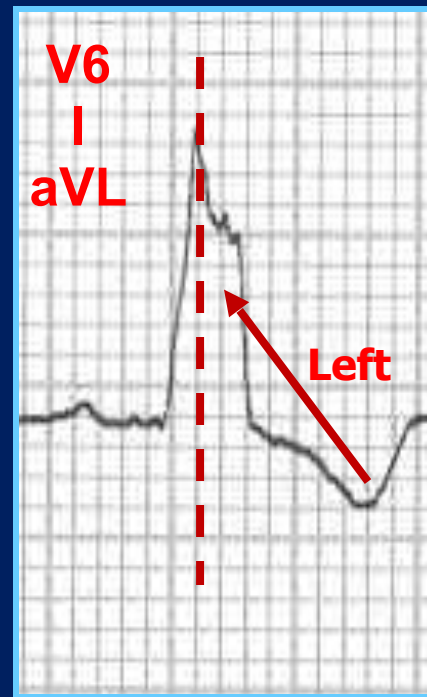
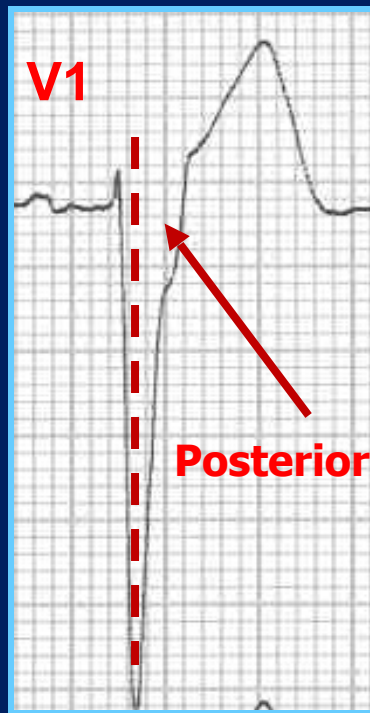
What do you see now?

What to do?

AV & IV Conduction System

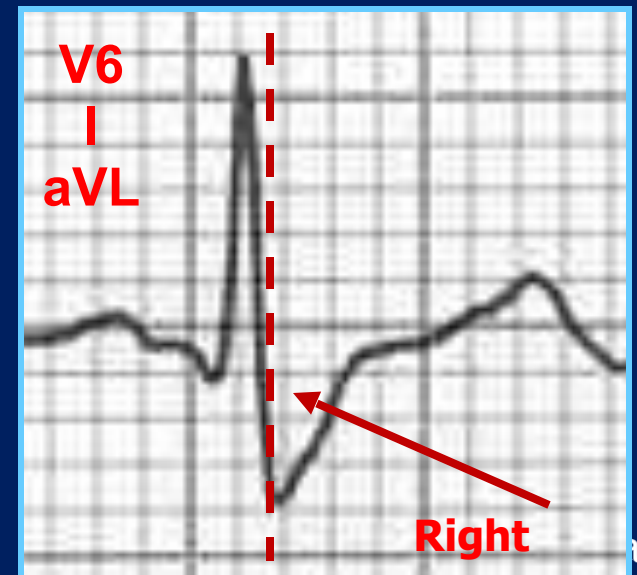
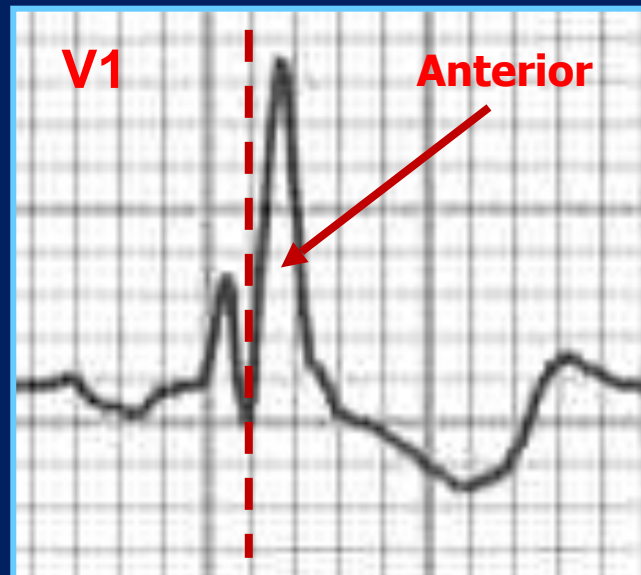


LBBB



In LBBB the LV is activated last!
(left & posterior)

RBBB



In RBBB the RV is activated last!
(right & anterior)

Etiologies of IV Conduction Disorders

- Familial (several variants described)
 - SCN5A mutations (Na⁺ channel)
 - Connexin40 mutations
- Acquired
 - Ischemic heart disease (e.g., acute MI)
 - Idiopathic and other cardiomyopathies
 - Hypertensive heart disease
 - Valvular heart disease (e.g., aortic stenosis)
 - Infiltrative cardiac diseases
 - Degenerative (aging heart – Lev's disease)

Structural
normal heart

← CONDUCTION DISEASE →

Structural
abnormal heart

Europace
2005;5:163

CONGENITAL

ACQUIRED

- drugs
- electrolyte abnormalities

ACQUIRED

- ischaemic
- traumatic
- autoimmune
- infectious
- neoplasms
- ageing (Lev disease)

CONGENITAL

INHERITED

- *SCN5A* sodium channel mutations
- gap junction gene mutations
- fatty acid oxidation disorders
- *PRKAG2* mutations (protein kinase subunit)
- *LMNA* gene mutations (Lamin A/C)

CONGENITAL BUT NOT INHERITED

- maternal Anti Ro/SSA antibodies
- exposure to toxic agents

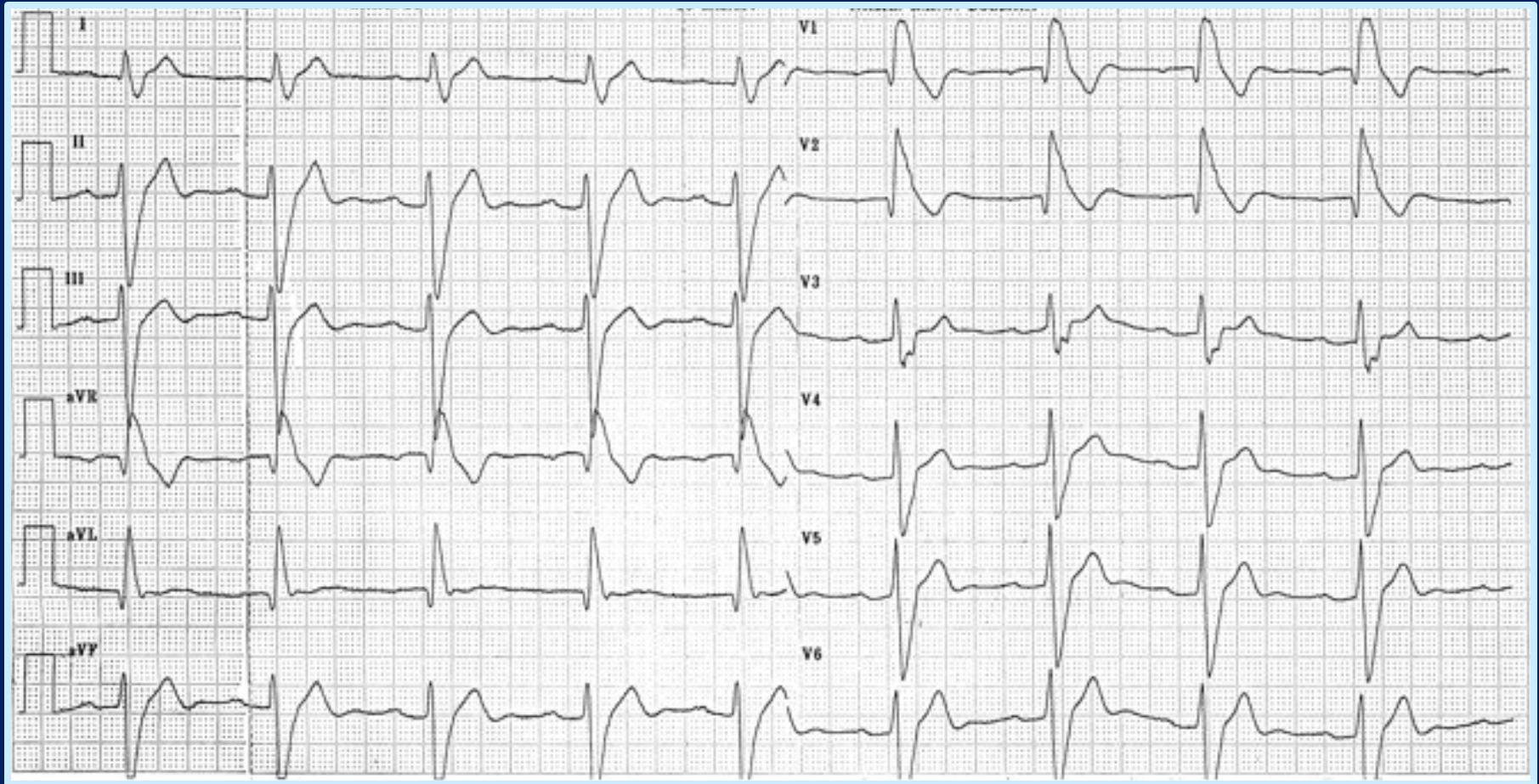
INHERITED

- *NKX2-5* mutations (transcription factors)
- *PRKAG2* mutations (protein kinase subunit)
- *LMNA* gene mutations (Lamin A/C)
- some muscular dystrophies (*SCN5A* sodium channel mutations)

CONGENITAL BUT NOT INHERITED

- maternal Anti Ro/SSA antibodies
- exposure to toxic agents
- exposure to infectious agents
- chromosomal abnormalities
- Lenegre/ Lev disease

12-lead ECG of a patient with Inherited cardiac conduction disease (ICCD) due to an SCN5A mutation.



Note: 1) atypical RBBB (qR in V1-2) and 2) LAD (-60 degrees) with S3>S2 (LAFB)

Bundle Branch Disease

- Abrupt Onset
 - e.g., Acute MI (left main or proximal LAD)
- Gradual Onset (initially rate dependent)
 - e.g., The aging conduction system (Lev's disease)

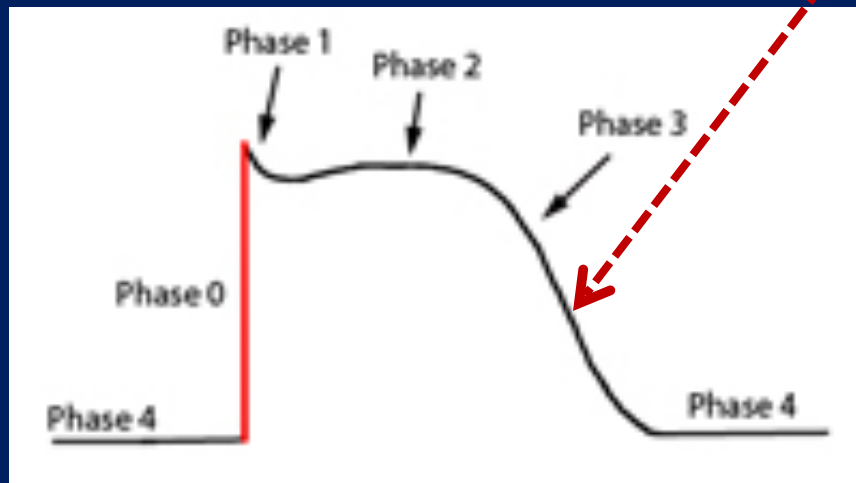
LBBB @ slightly faster HR



This is called the '*hysteresis* effect': i.e., the rate that the BBB appears during rate acceleration is higher than the rate where the BBB disappears with slowing of the heart rate.

Tachycardia-dependent BBB (Phase 3 block)

At faster rates the sinus impulse arrives during phase 3 of the bundle branch action potential and is blocked.

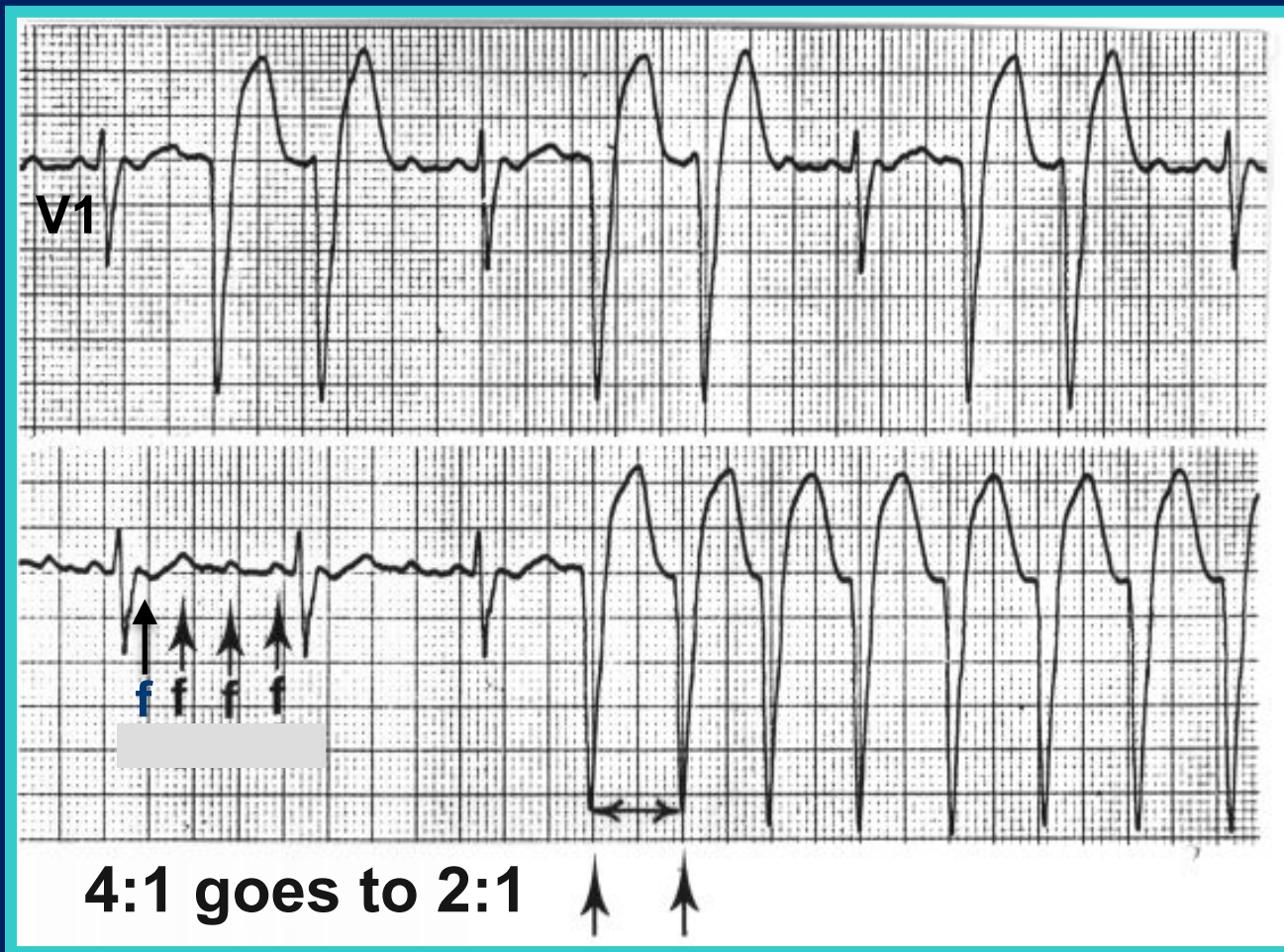


Bundle branch action potential

RBBB @ slightly faster HR

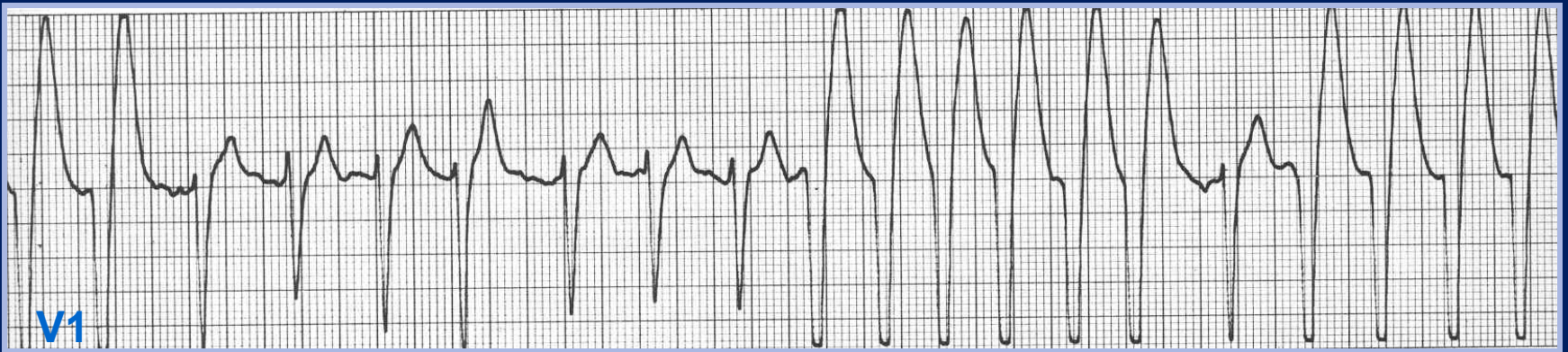


Tachycardia-dependent LBBB in Atrial Flutter with variable AV block

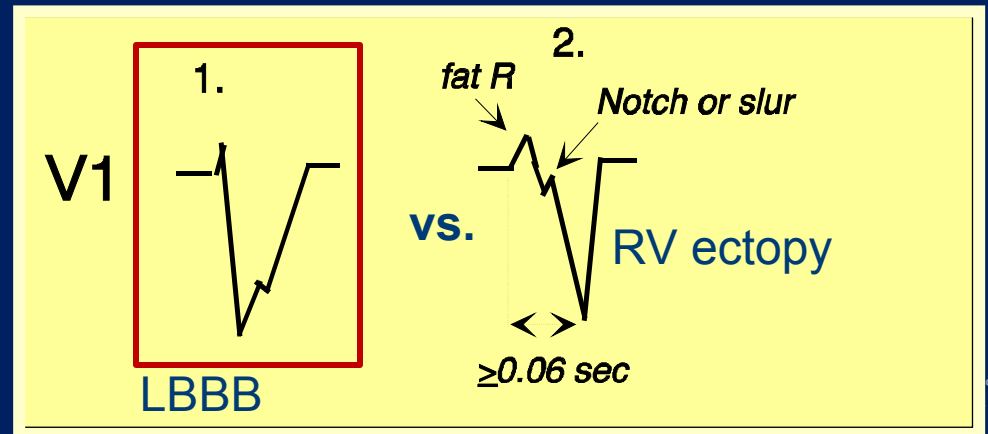


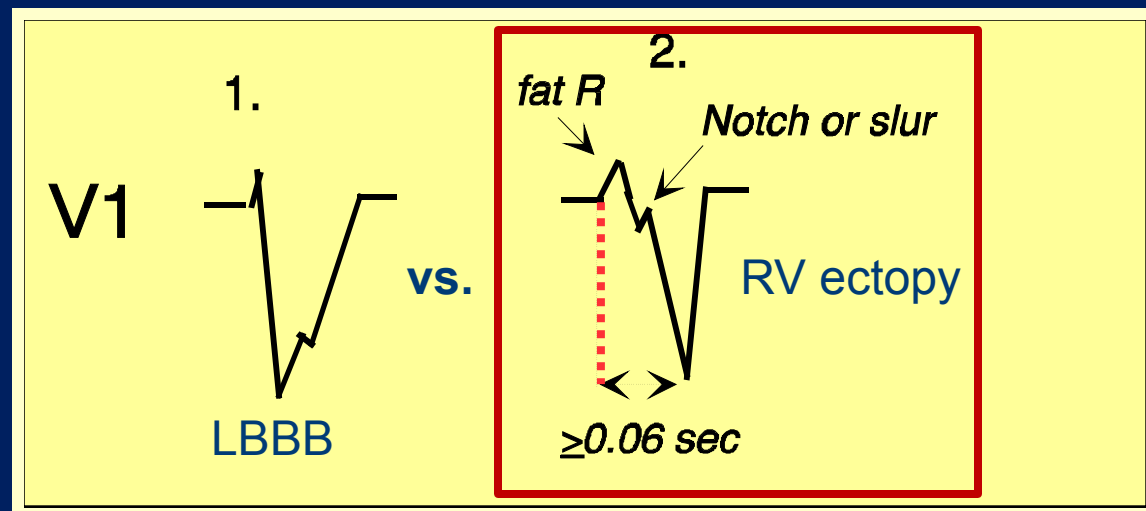
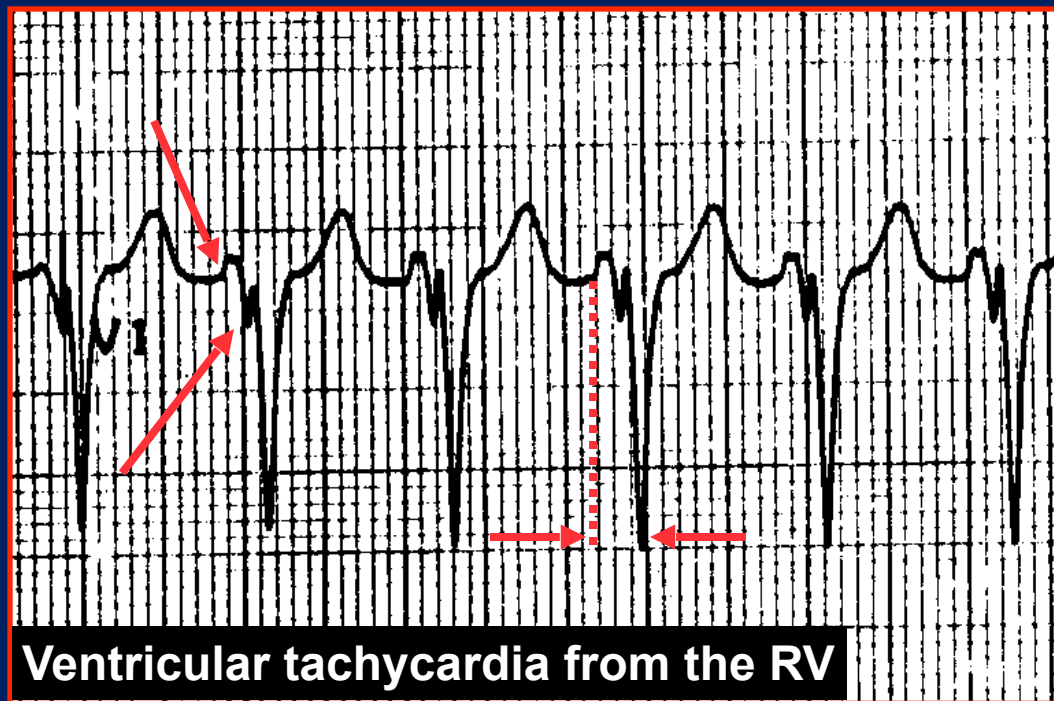
Wide QRS Tachycardia (WQRST) (Nonsustained)

Atrial fibrillation (RVR) with intermittent tachycardia-depend LBBB



**Don't confuse this WQRST for
ventricular tachycardia!**





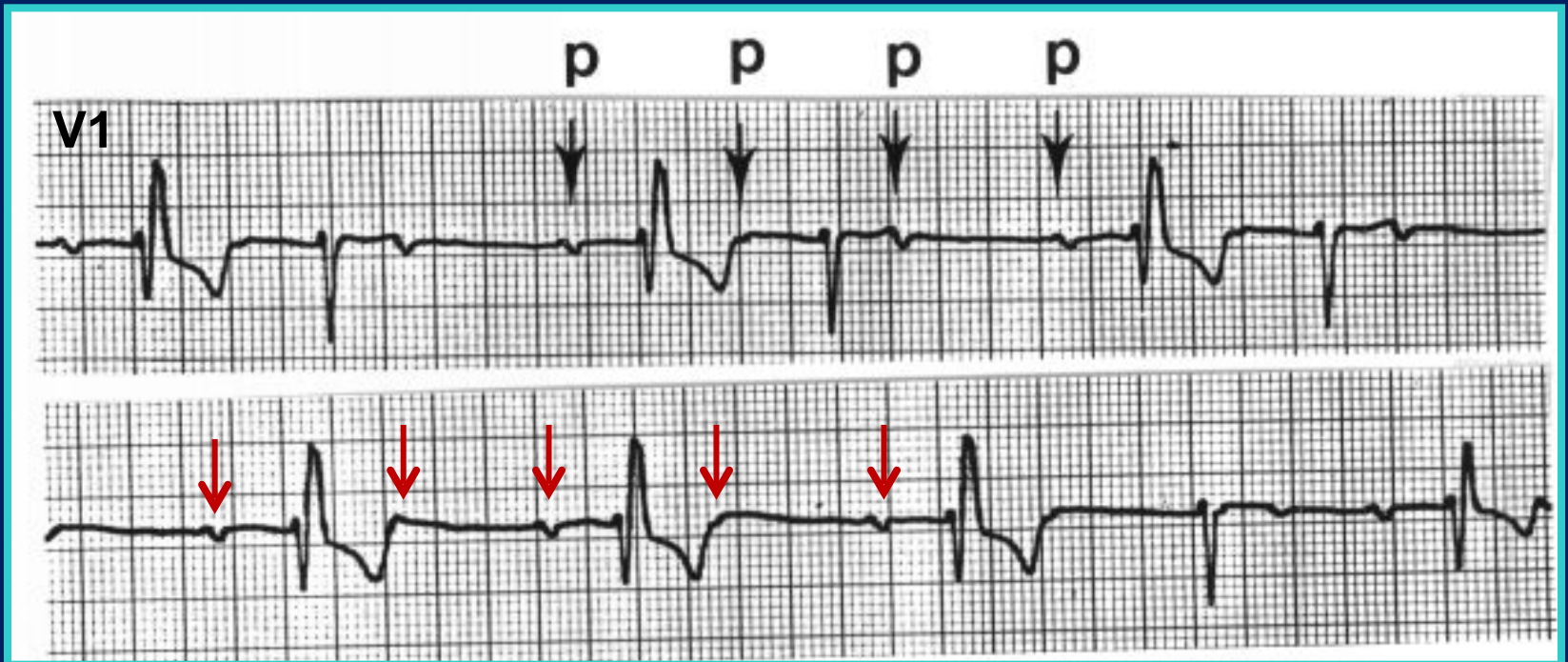
Why does the RBB fail @ longer RRs?



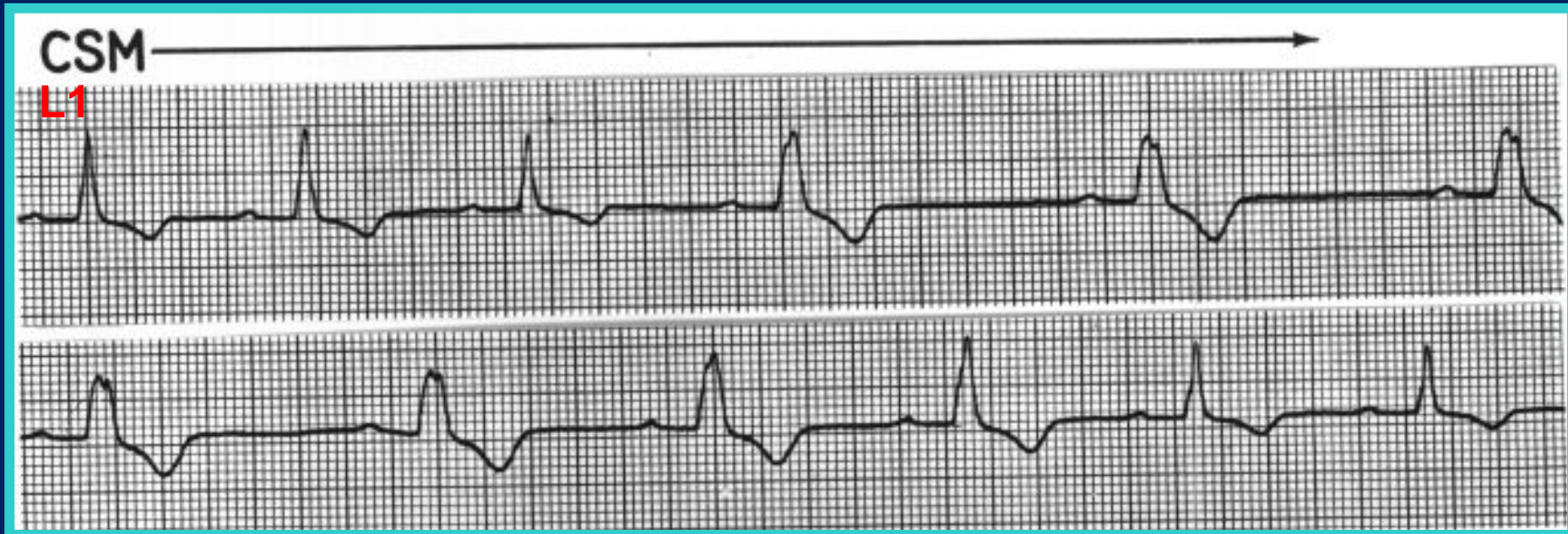
Normal sinus rhythm (arrows); 2nd degree (type I) 3:2 AV block

Note: Intermittent bradycardia-dependent RBBB

Bradycardia-dependent RBBB (3:2 and 2:1 2° AV Block, type I)

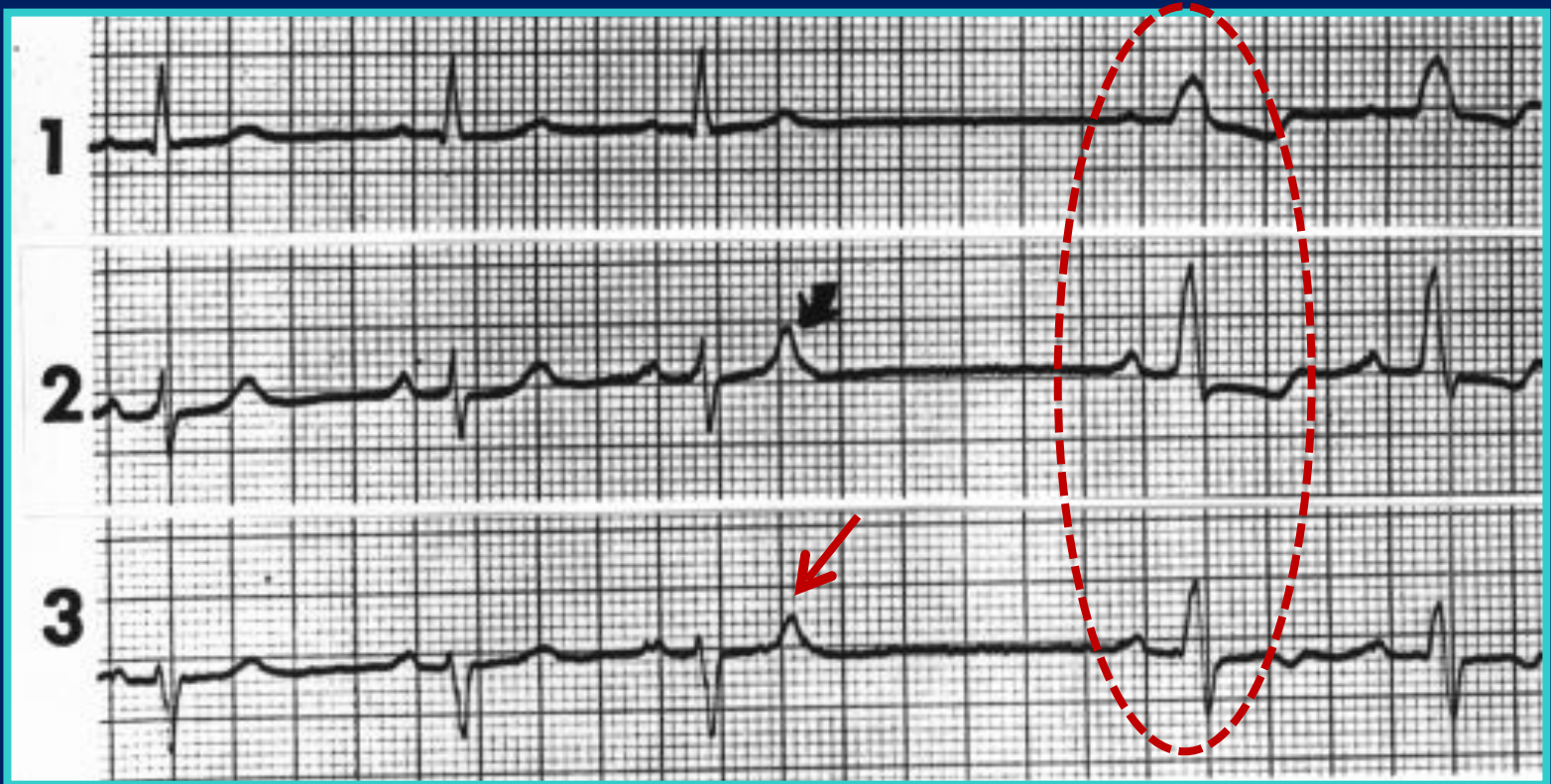


Why does the LBB fail @ slower HRs?



CSM: carotid sinus massage

An unexpected pause.....



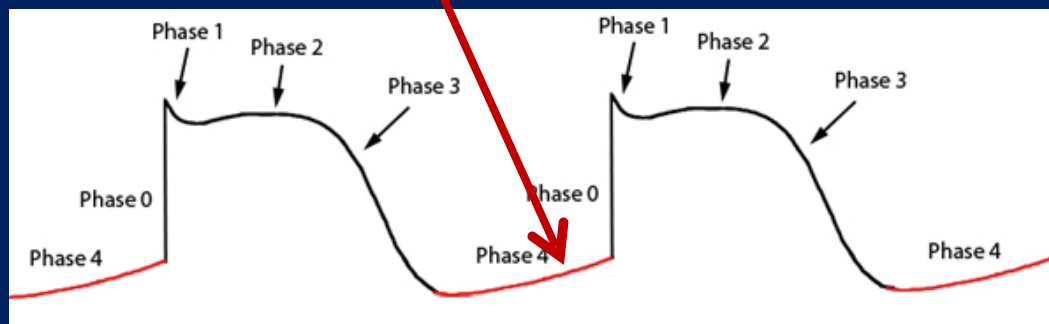
The most common cause of an unexpected pause is a nonconducted PAC

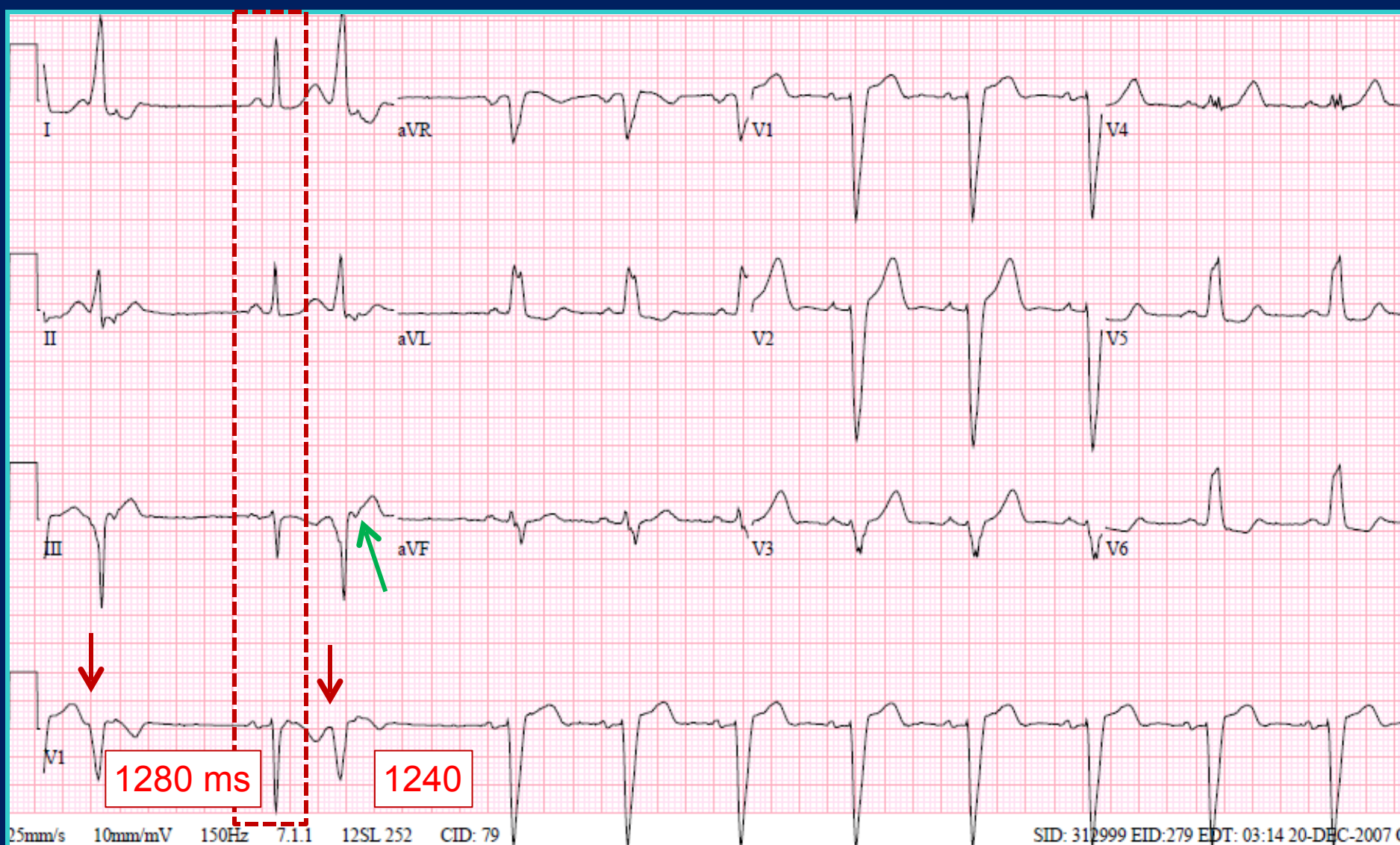
Note: the pause ends with sinus P wave conducted with **LBBB**

Proposed Mechanism: Bradycardia-dependent BBB (phase 4 block)

Bradycardia-dependent IVCD is attributed to the inherent capacity of one or the other bundle branch to “spontaneous diastolic depolarization” or “diastolic hypopolarization”) during phase 4 of the action potential in an attempt to become a ‘backup’ pacemaker when the sinus slows too much.

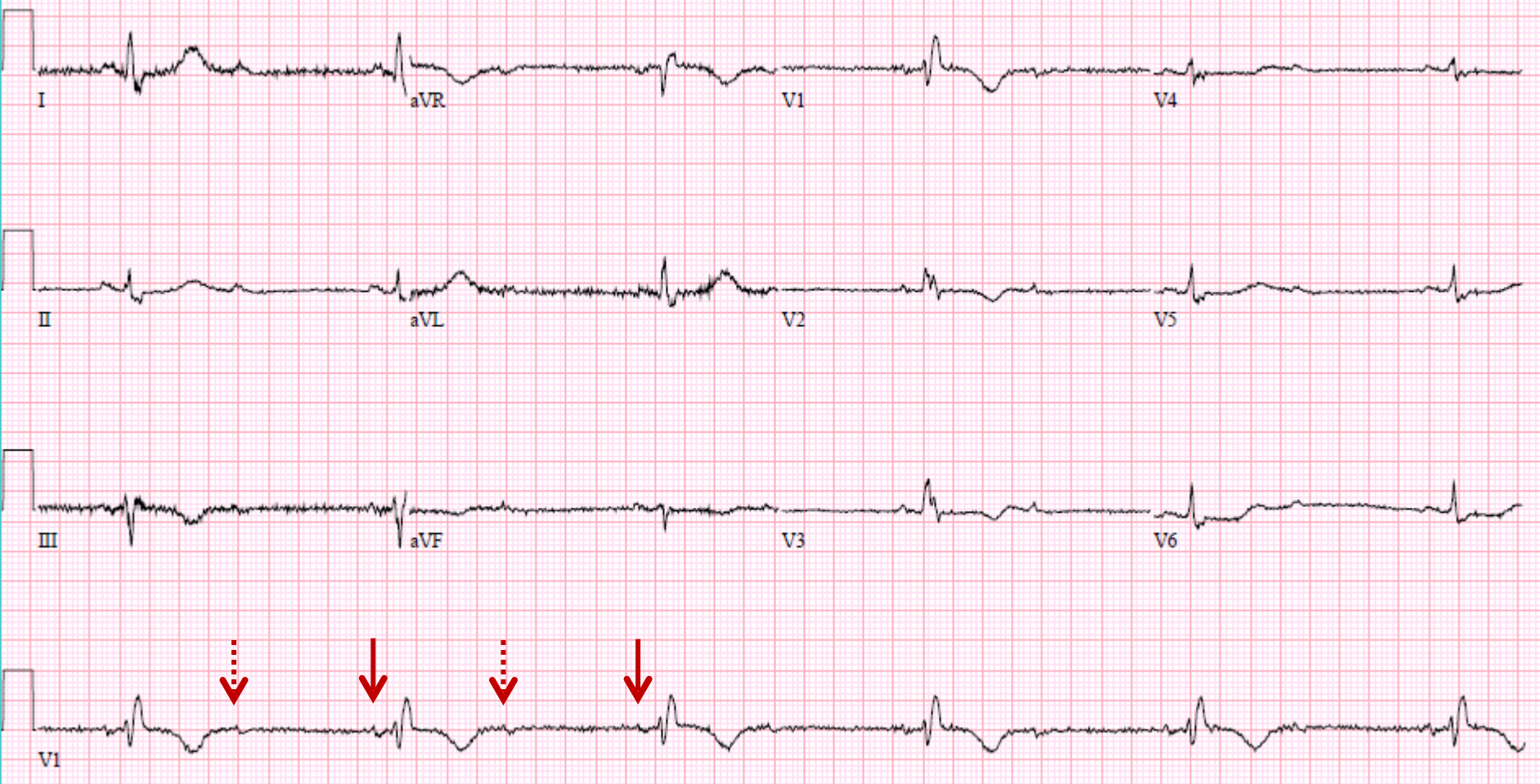
If the next impulse from the atria or the atrioventricular node arrives at the bundle branch during a time period when the cells are partially depolarized, conduction may not take place in that bundle (i.e., a BBB)





17-Dec-2007: 66 y.o. woman (routine ECG).... back to our patient!

Note: the first **PVC** and pause allows the LBB to recover, but **LBBB** reoccurs after the 2nd **PVC** because the pause is shorter and the heart rate picks up again. This indicates the LBB can still work at slower heart rates.

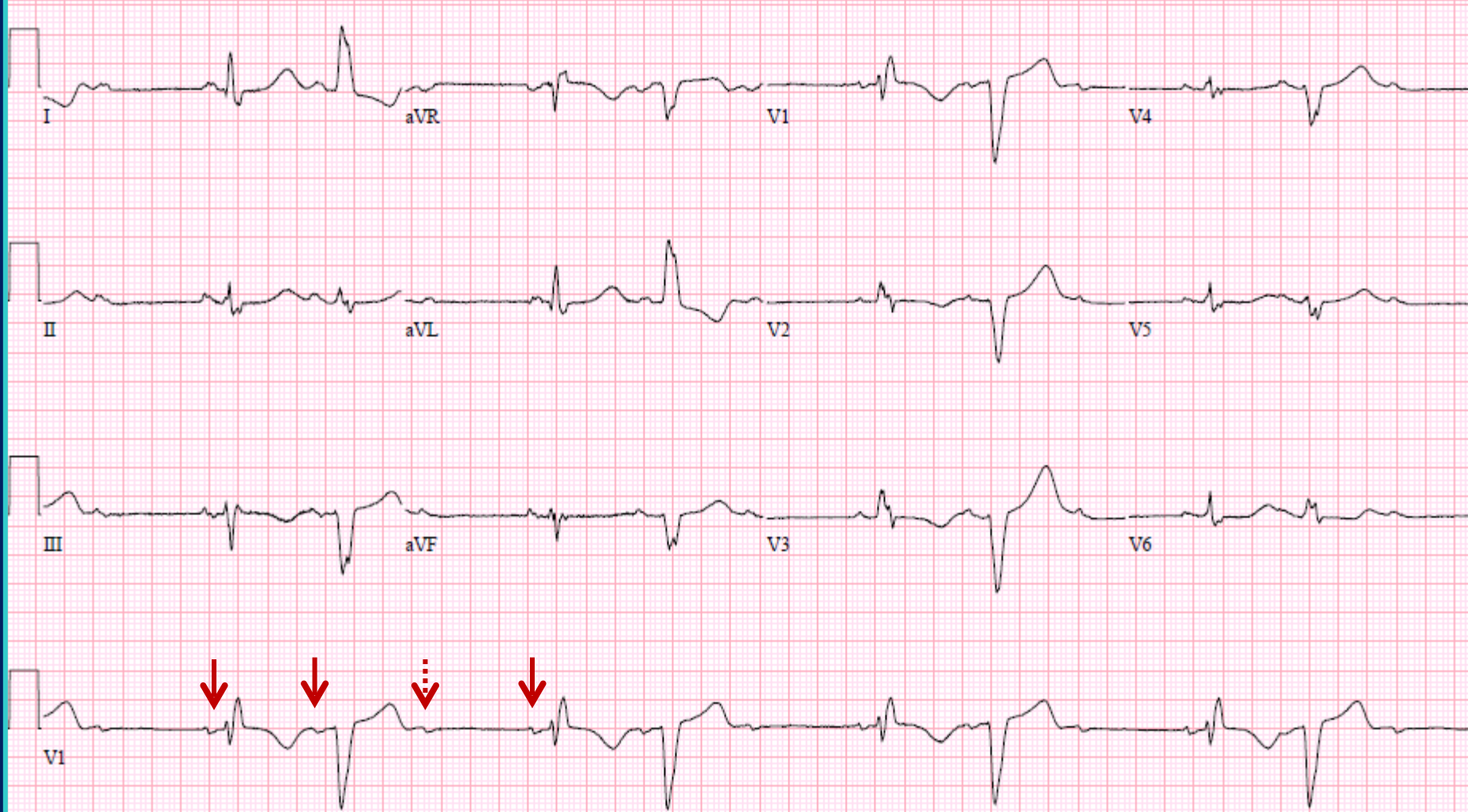


Six years later.....

19-June-2013: now 71 y.o. woman (fatigue)

2:1 AV block and RBBB

Note: the conducted P waves with RBBB indicate the LBB must still be able to conduct, but the RBB is now not working!

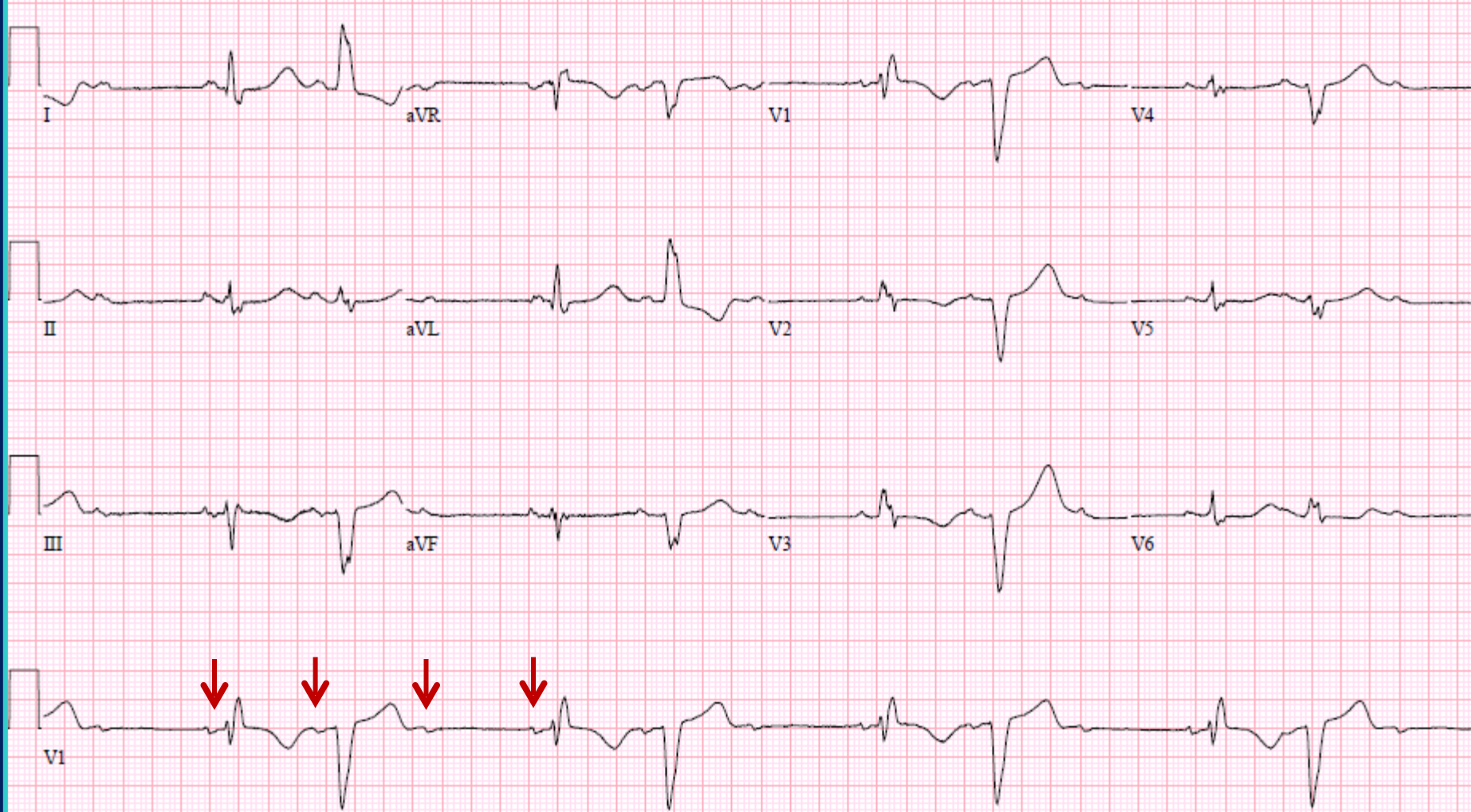


19-June-2013: 71 y.o. woman, later that same day.....

Note: the conducted beats with LBBB indicates the RBB must be working!

Summary: 2° AV block (type II) with 3:2 conduction.

- RBBB is bradycardia-dependent
- LBBB is tachycardia-dependent



19-June-2013: PL: 71 y.o. woman (fatigue)... not only is this an amazing ECG but:

YES! She needs a PACEMAKER!

Summary: 2° AV block (type II) with 3:2 conduction.

- RBBB is bradycardia-dependent (Phase 4 block)
- LBBB is tachycardia-dependent (Phase 3 block)

Thank You !

Staying up to date

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