WPW syndrome and AVRT

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Supraventricular tachycardia (SVT)	An umbrella term used to describe tachycardias (atrial and/or ventricular rates in excess of 100 bpm at rest), the mechanism of which involves tissue from the His bundle or above.
Paroxysmal supraventricular tachycardia (PSVT)	A clinical syndrome characterized by the presence of a regular and rapid tachycardia of abrupt onset and termination. PSVT represents a subset of SVT.
Atrial fibrillation (AF)	
Sinus tachycardia	
• Physiologic sinus tachycardia	
• Inappropriate sinus tachycardia	
Atrial tachycardia (AT)	
• Focal AT	
• Sinus node reentry tachycardia	
• Multifocal atrial tachycardia (MAT)	
Atrial flutter	
• Cavotricuspid isthmus–dependent atrial flutter: typical	
• Cavotricuspid isthmus–dependent atrial flutter: reverse typical	
• Atypical or non–cavotricuspid isthmus–dependent atrial flutter	
Junctional tachycardia	
Atrioventricular nodal reentrant tachycardia (AVNRT)	
• Typical AVNRT	
• Atypical AVNRT	
Accessory pathway	
• Manifest accessory pathways	
• Concealed accessory pathway	
• Pre-excitation pattern	
• Asymptomatic pre-excitation (isolated pre-excitation)	
• Wolff-Parkinson-White syndrome	
Atrioventricular reentrant tachycardia (AVRT)	
• Orthodromic AVRT	
• Antidromic AVRT	
Permanent form of junctional reciprocating tachycardia	**Richard L. Page et al. Circulation. 2016;133:e506-e574**
Pre-excited AF	

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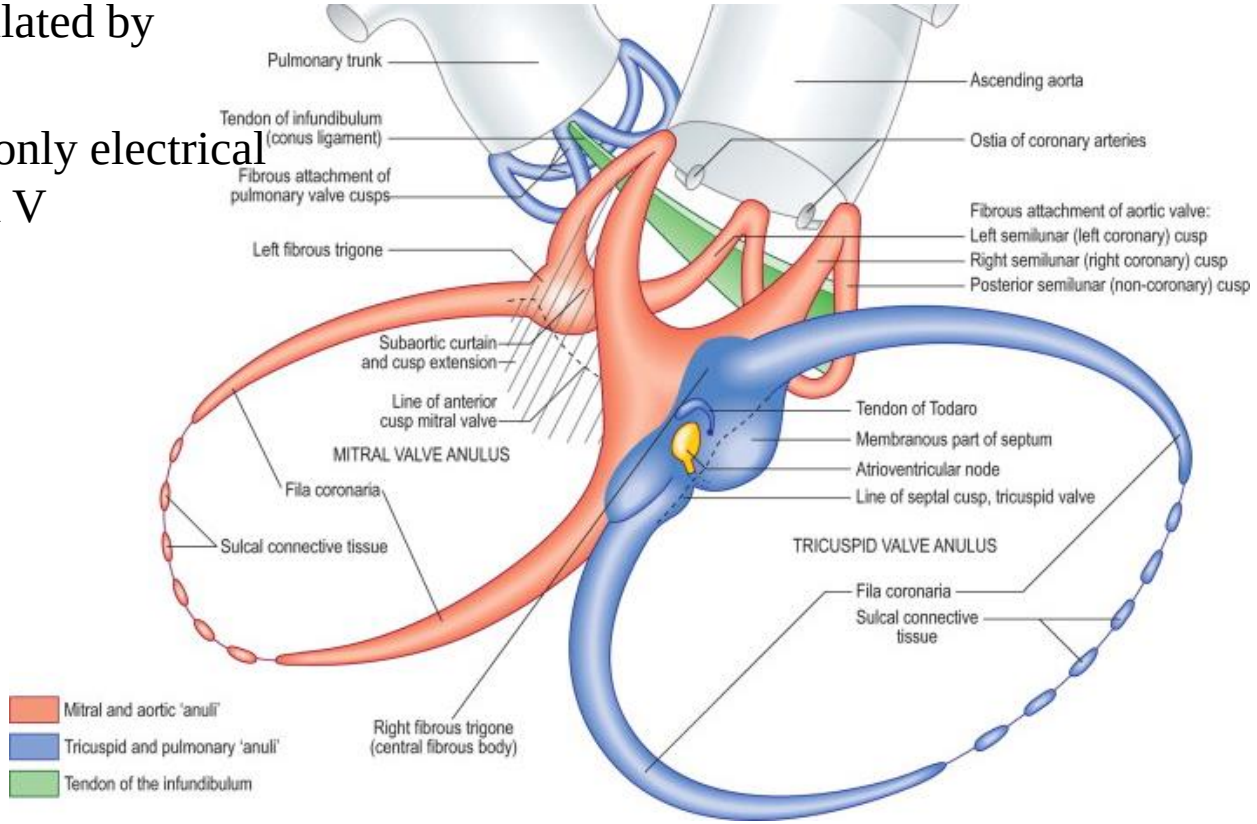
Accessory Pathways

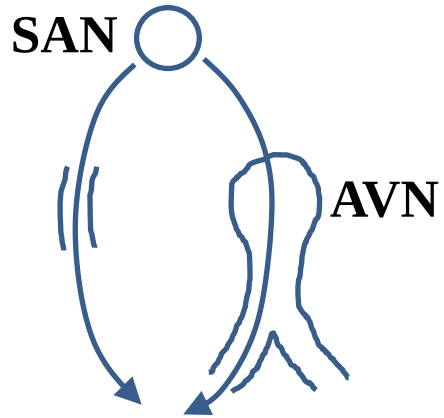
- Atrium and ventricle: insulated by cardiac skeleton
- Normally, AV node is the only electrical connection between A and V



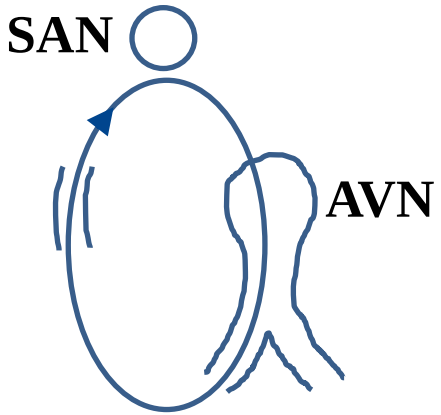
Accessory Pathways and Reentry

WPW syndrome

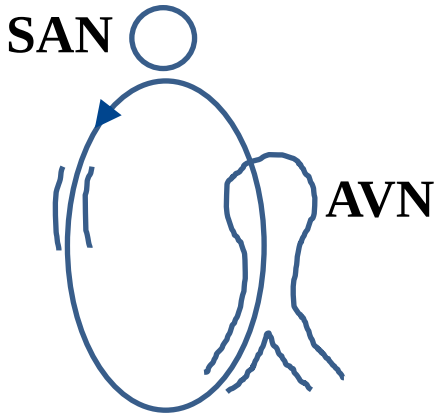
Sinus rhythm



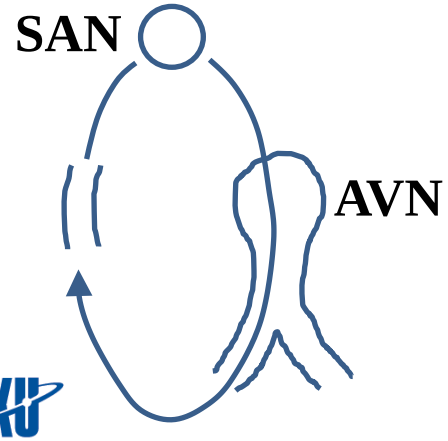
Orthodromic



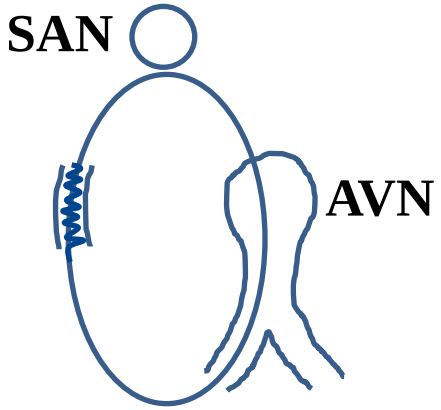
Antidromic



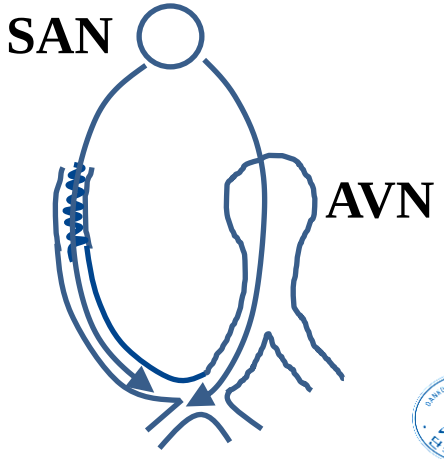
Concealed AP



PJRT



Mahaim



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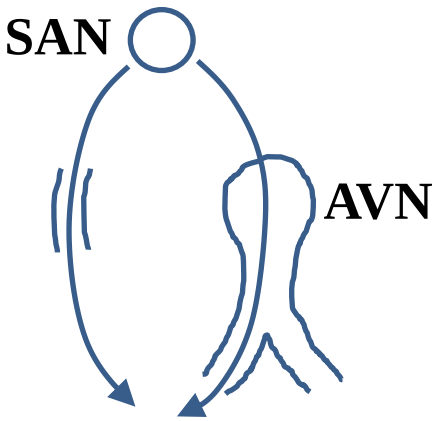
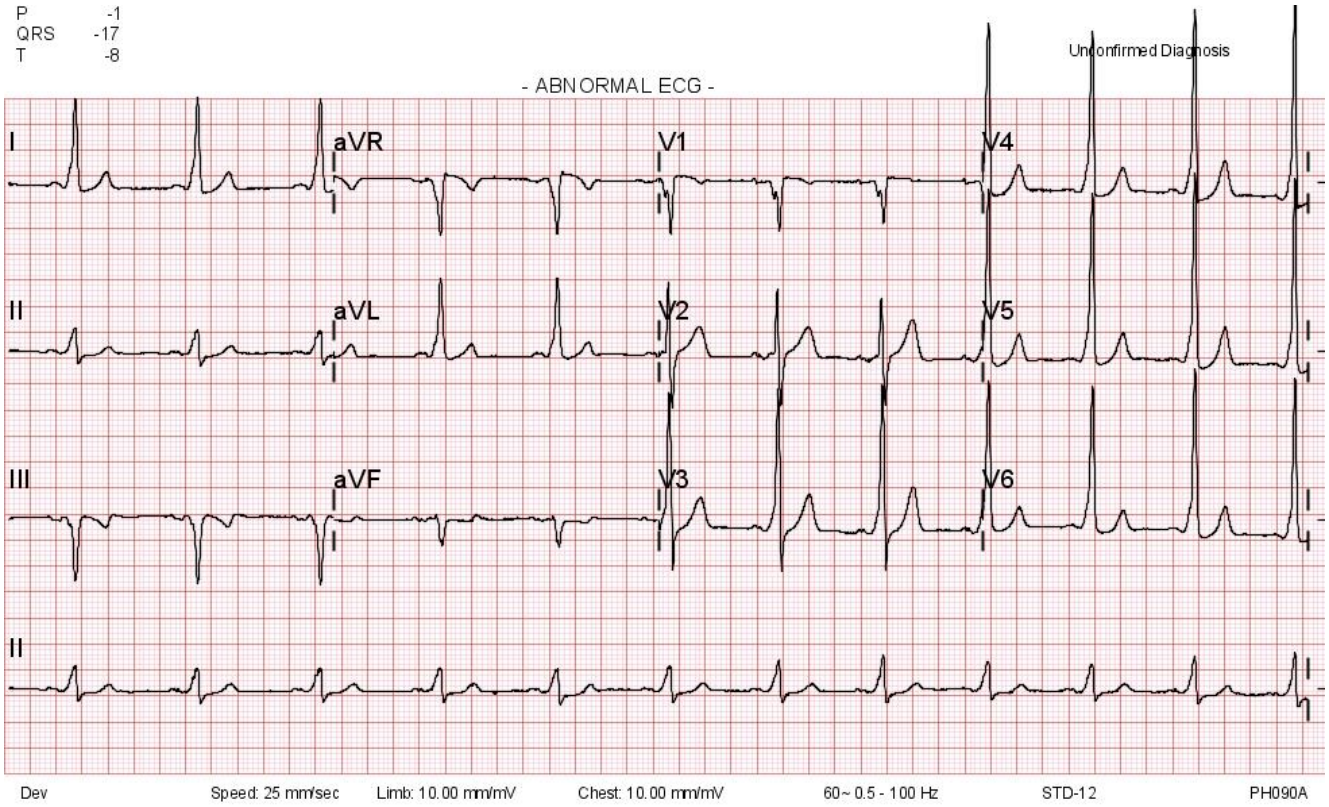
Accessory Pathways and Reentry

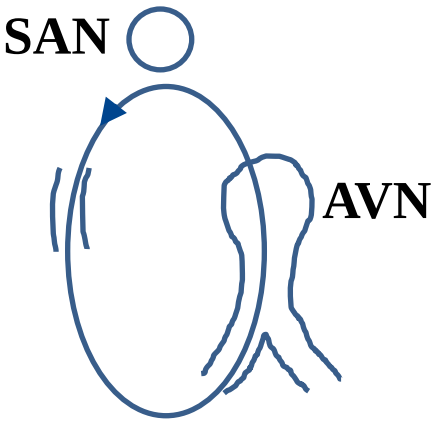
WPW syndrome
Sinus rhythm

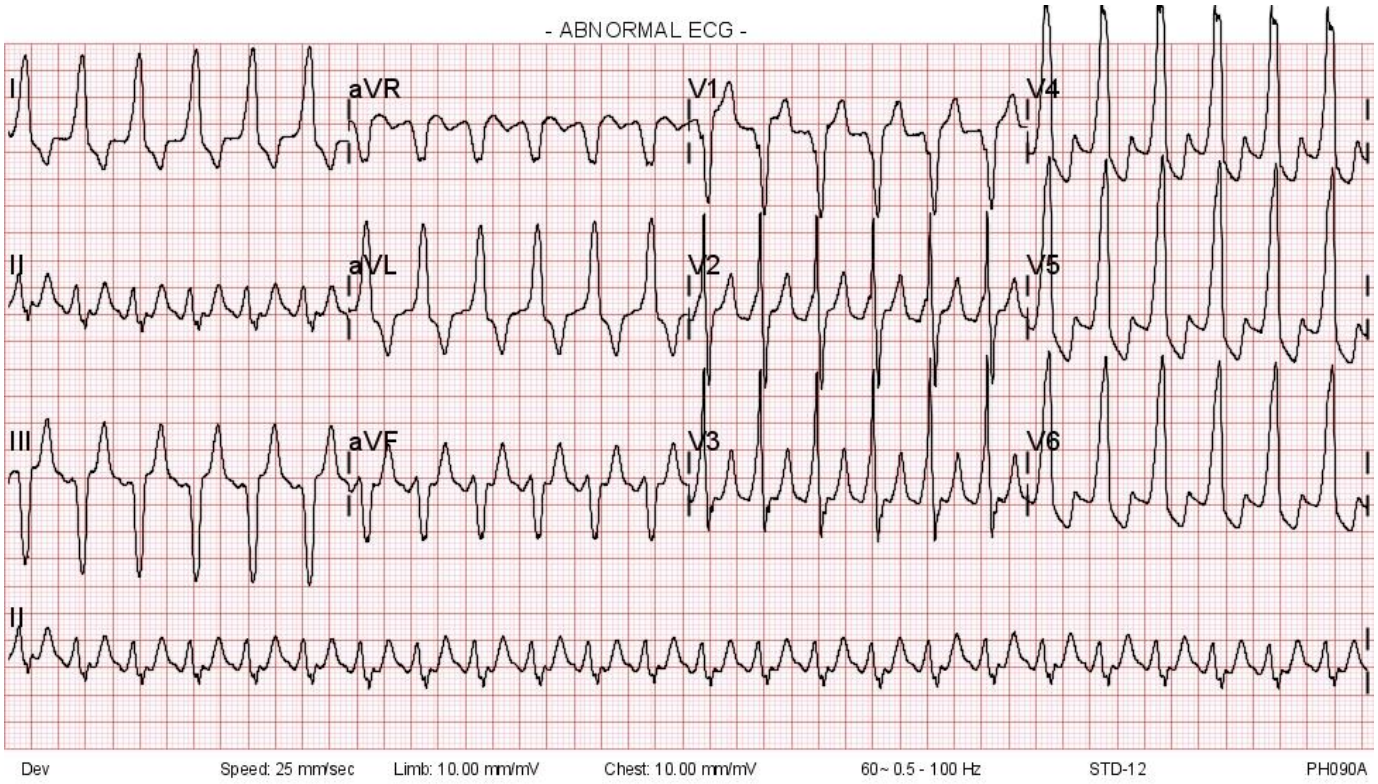




Accessory Pathways and Reentry

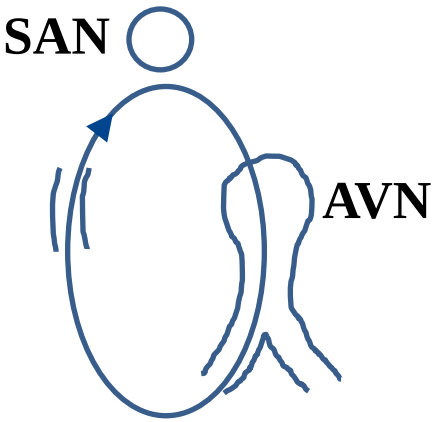
WPW syndrome
Antidromic

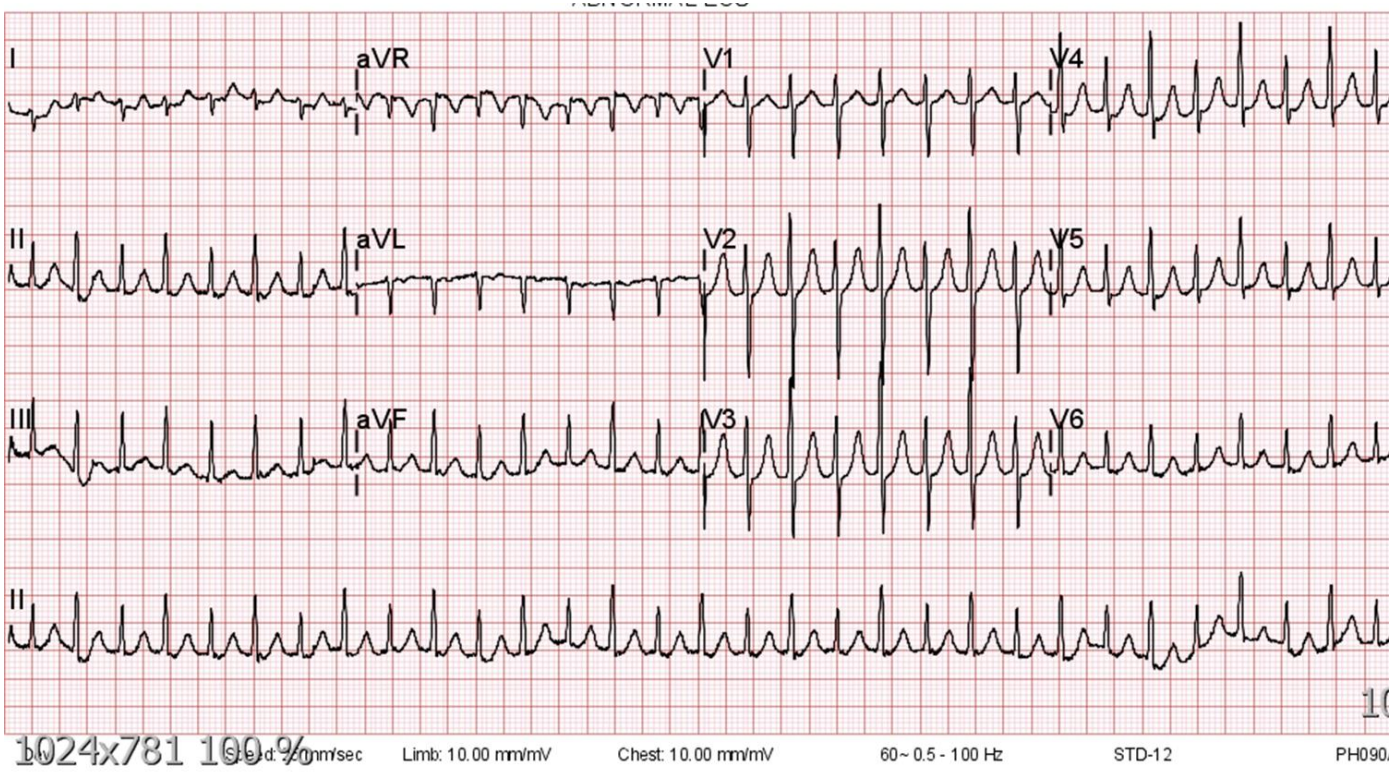




Accessory Pathways and Reentry

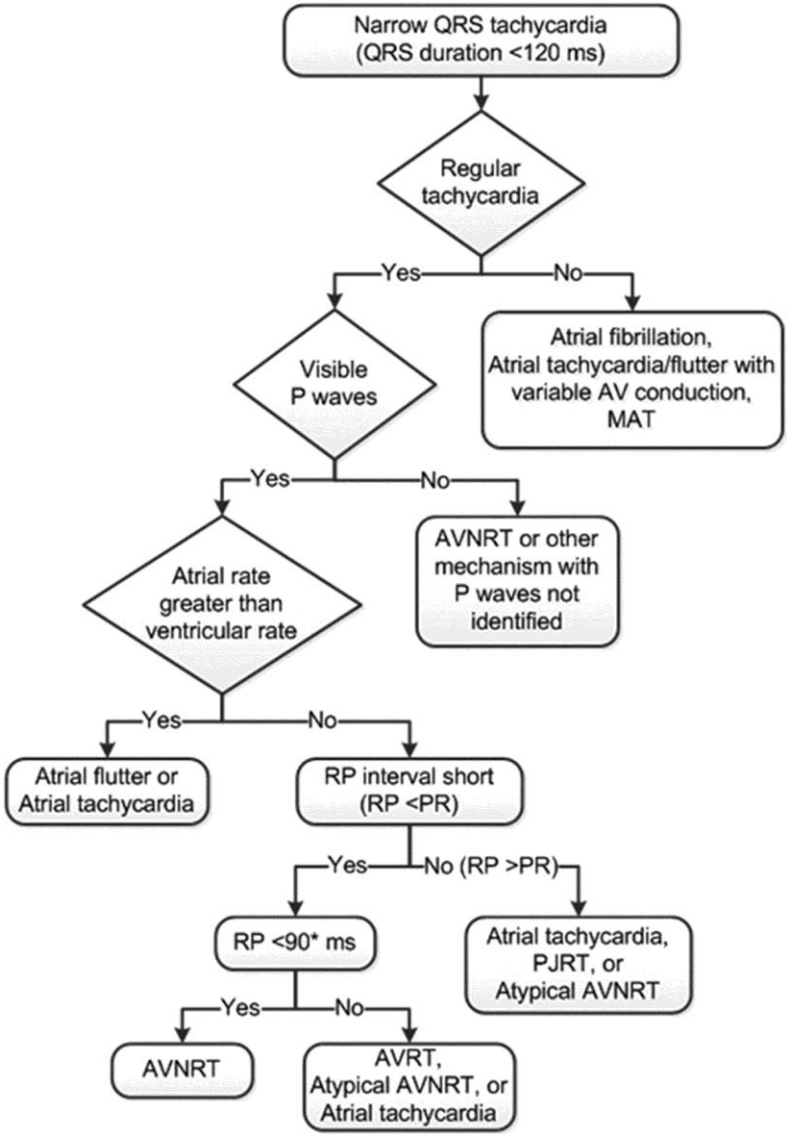
WPW syndrome
Orthodromic





Differential Diagnosis of Narrow QRS tachycardia

- QRS duration
- Regularity
- P waves
- RP interval

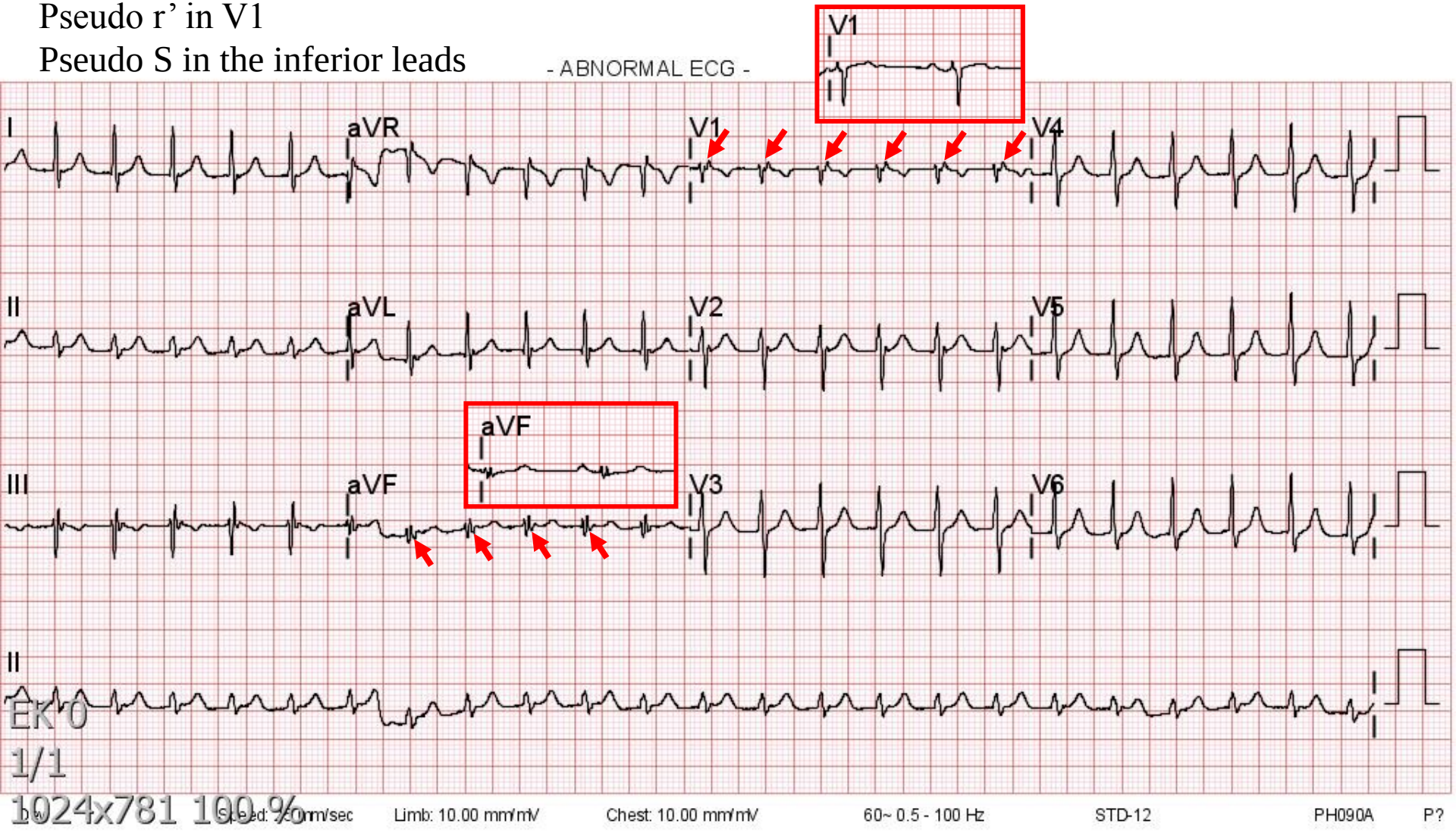


Richard L. Page et al. Circulation. 2016;133:e506-e574

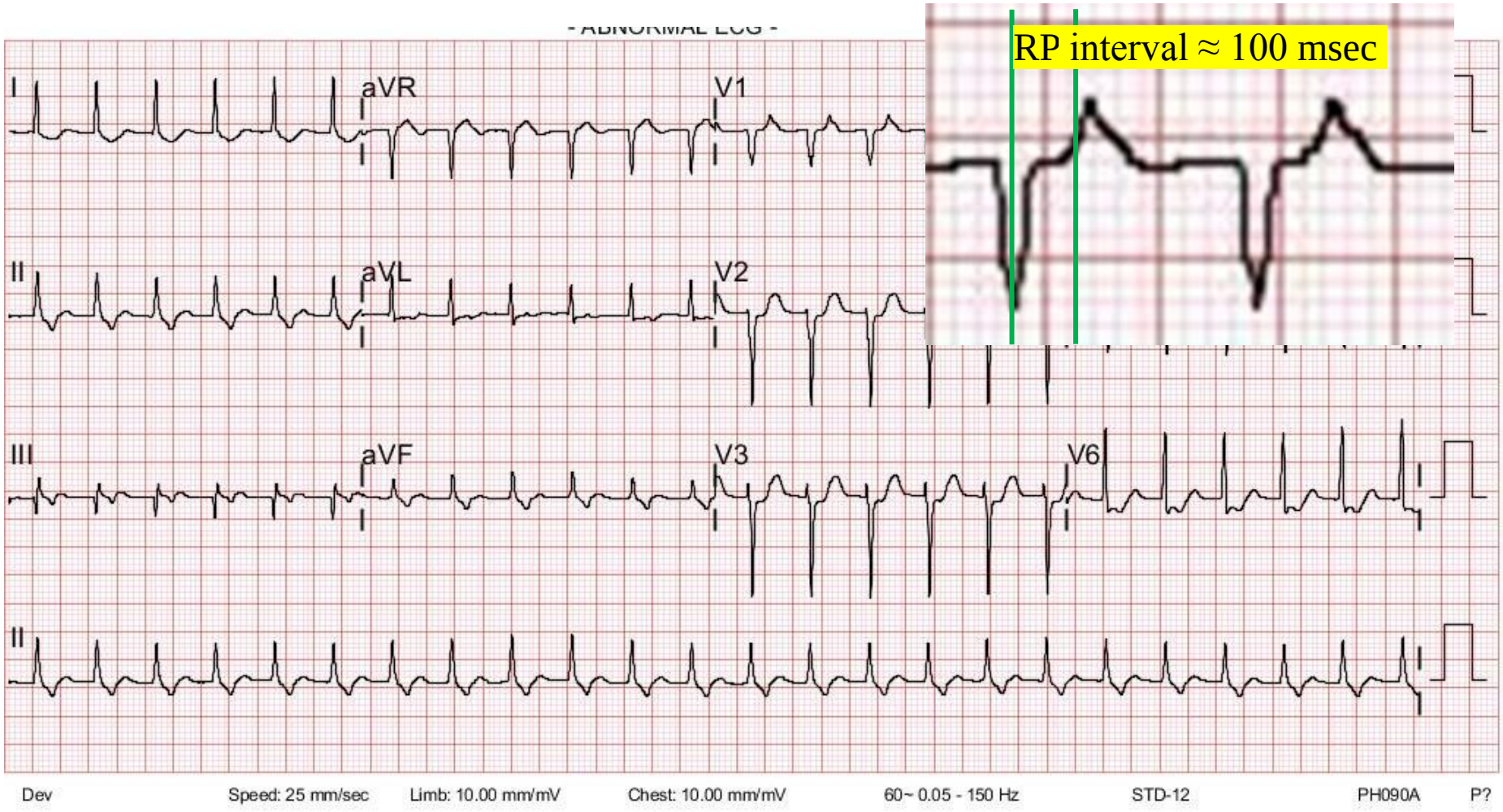
Typical AVNRT

Pseudo r' in V1

Pseudo S in the inferior leads



AVRT with Retrograde P



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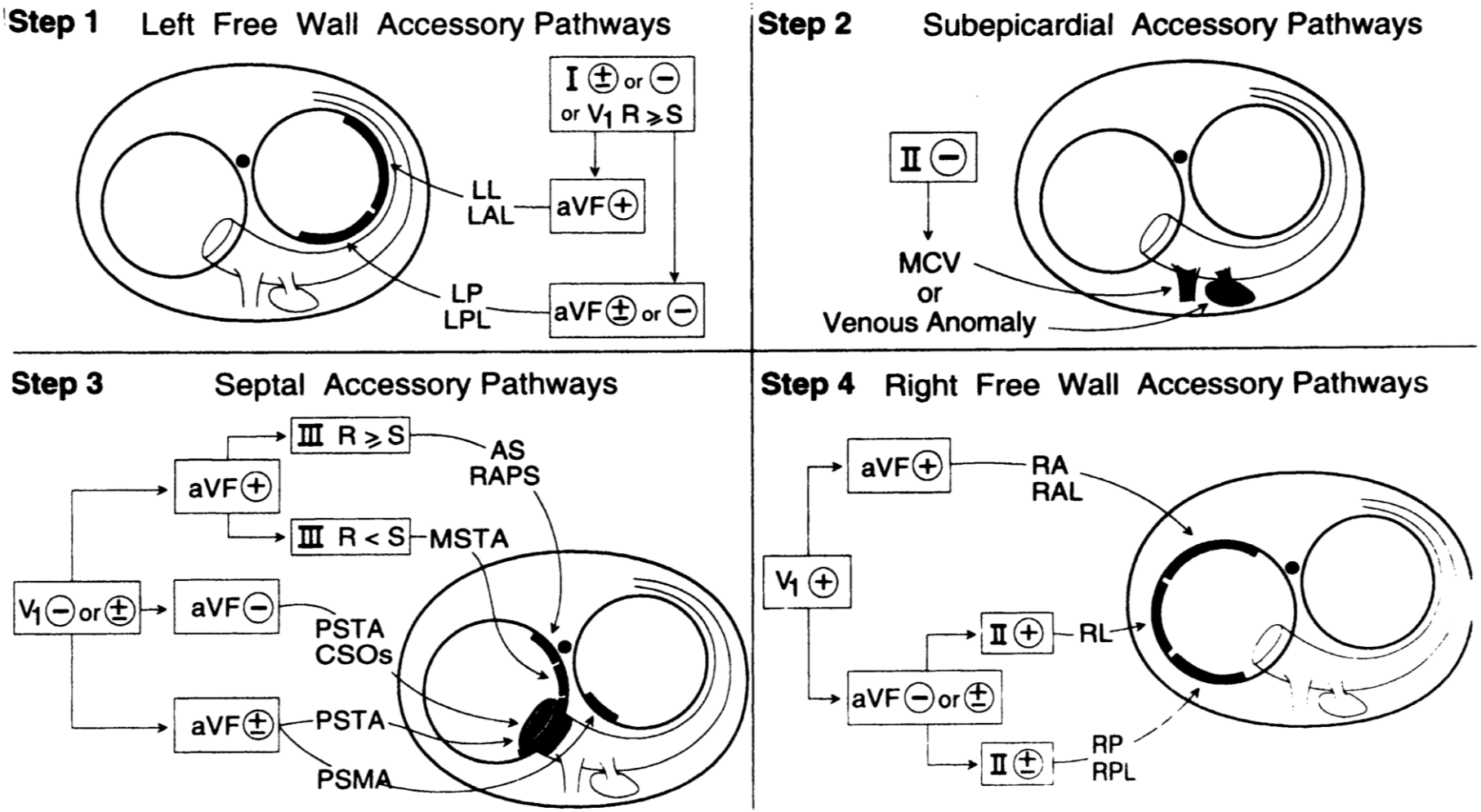
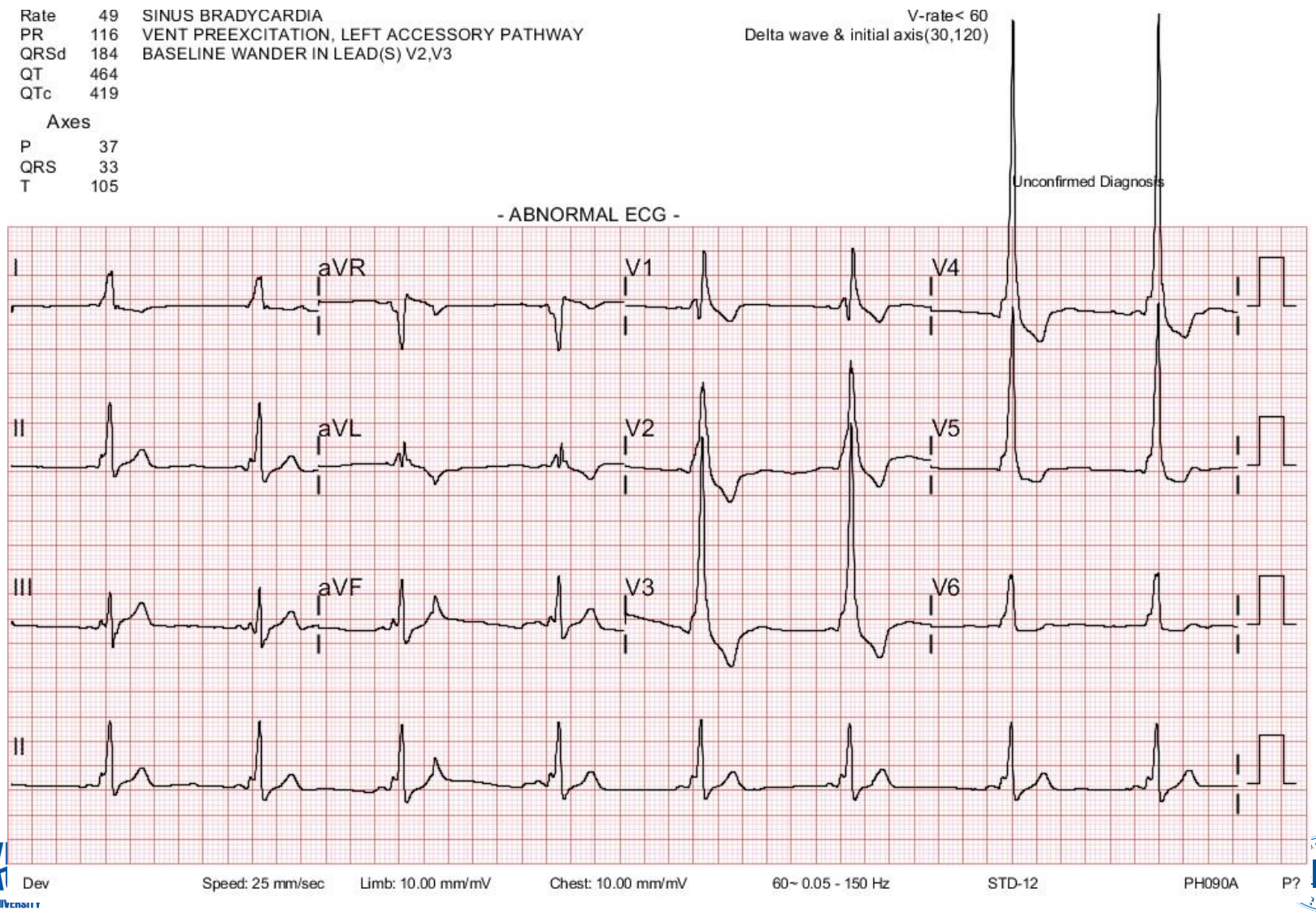
ECG algorithm for AP localization

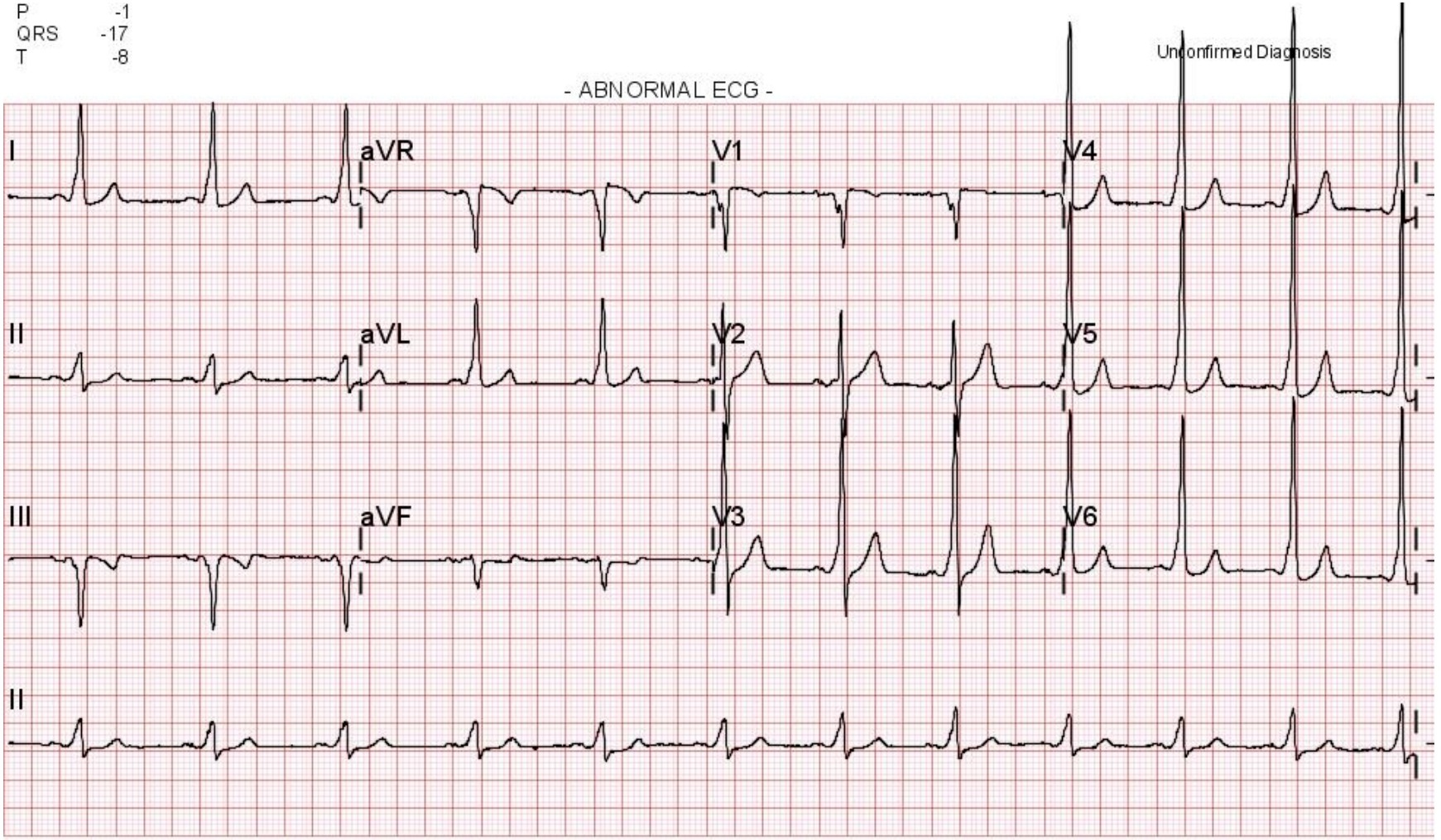


J Cardiovasc Electrophysiol 1998;9:2

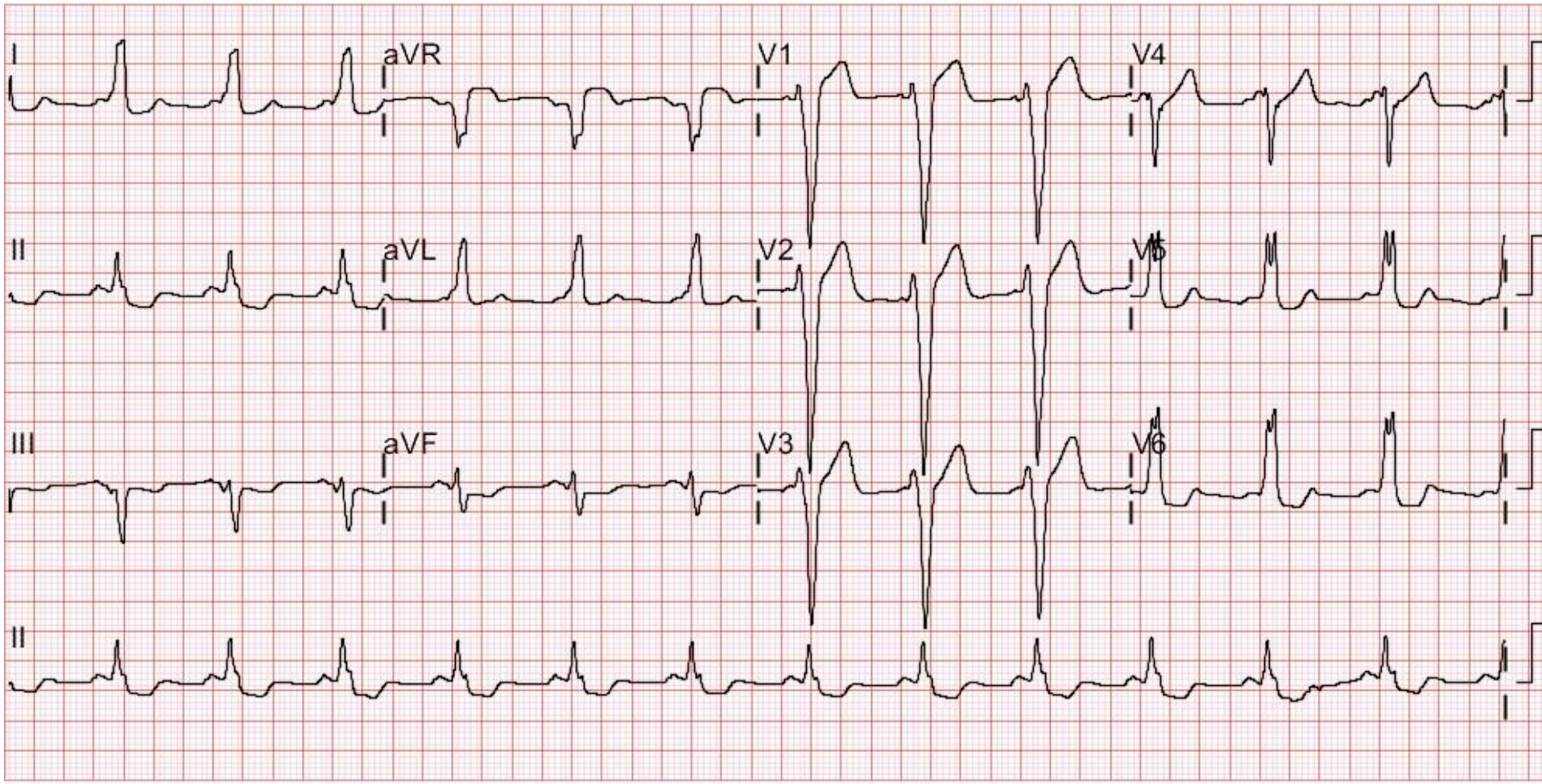
Accessory Pathway: Left Free Wall



Accessory Pathway: Septal



Accessory Pathway: Right Free Wall



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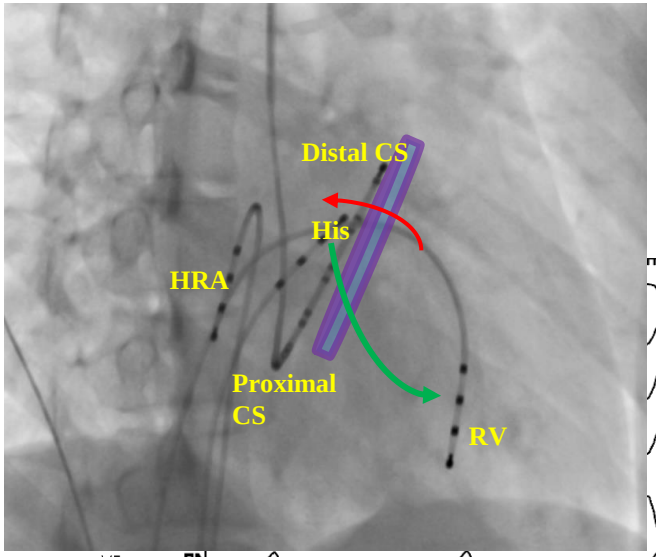
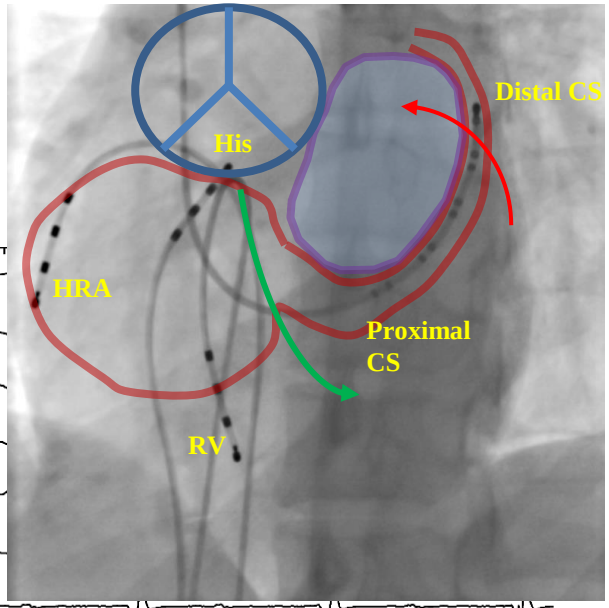
Differentiation of PSVT in EPS

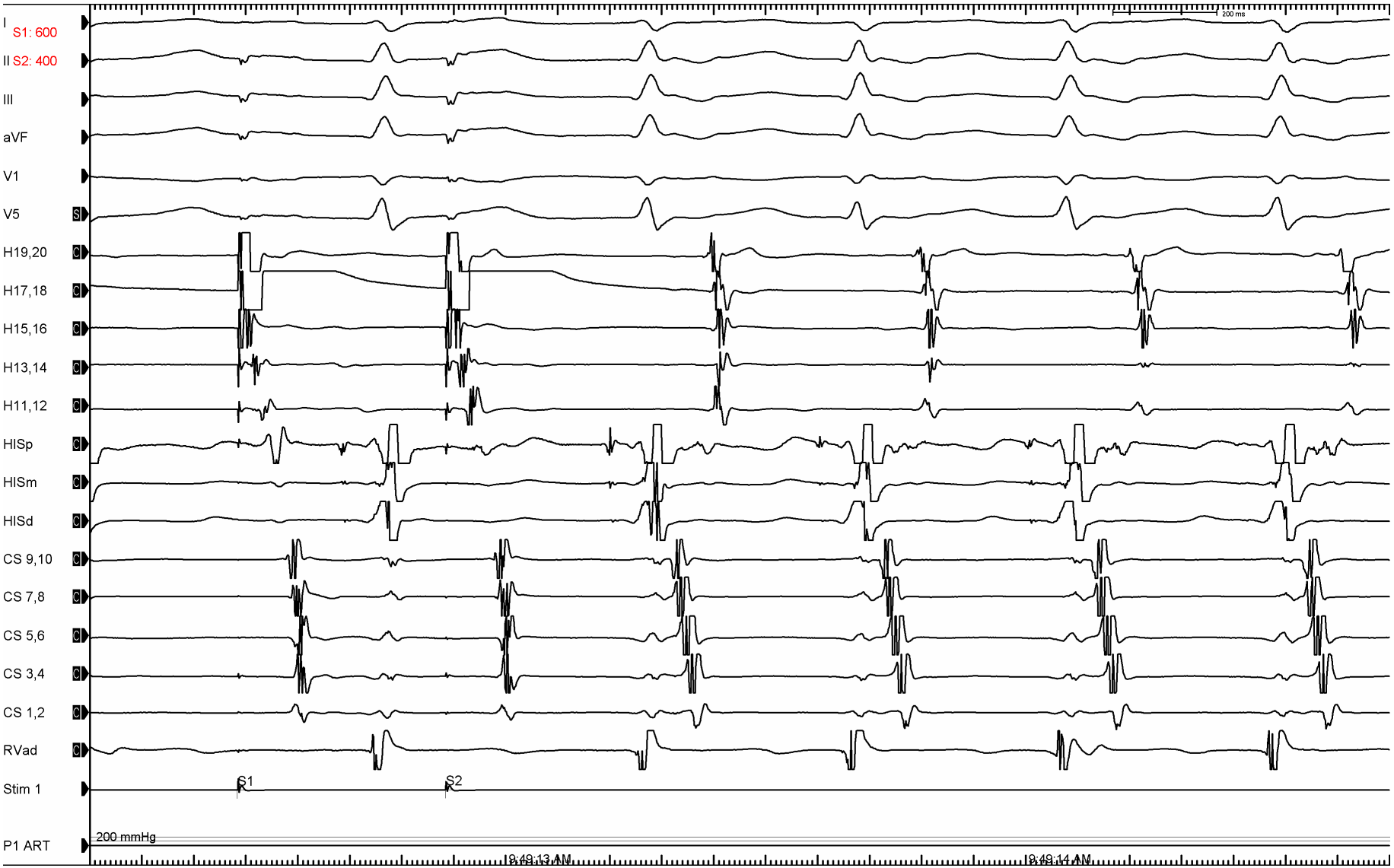
Baseline Observations and Tachycardia Features	Prevalence (%)	Positive Predictive Value (%)		
		AVNRT	ORT	AT
Pre-excitation present during sinus rhythm	15	10	86	3
Extranodal response to para-Hisian pacing	18	17	83	0
VA block cycle length > 600 msec at baseline	11	41	5	55
Septal VA interval > 70 msec	53	17	59	24
Eccentric atrial activation	31	0	76	24
Spontaneous AV block during tachycardia	10	60	0	40
Spontaneous termination with AV block	28	66	34	0
Development of LBBB	12	4	92	4
Increase in VA interval > 20 msec with BBB	7	0	100	0

Eccentric Atrial Activation

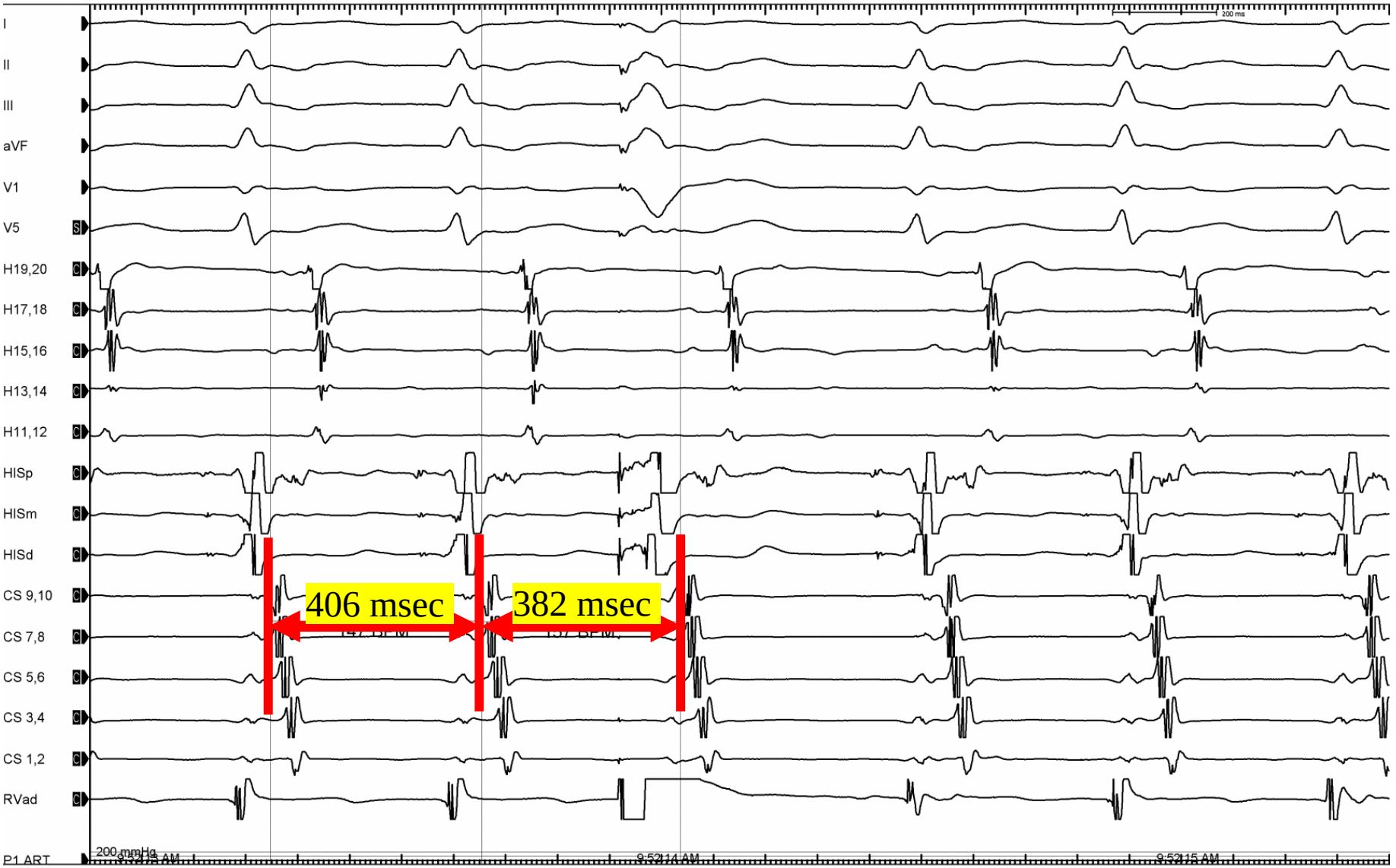




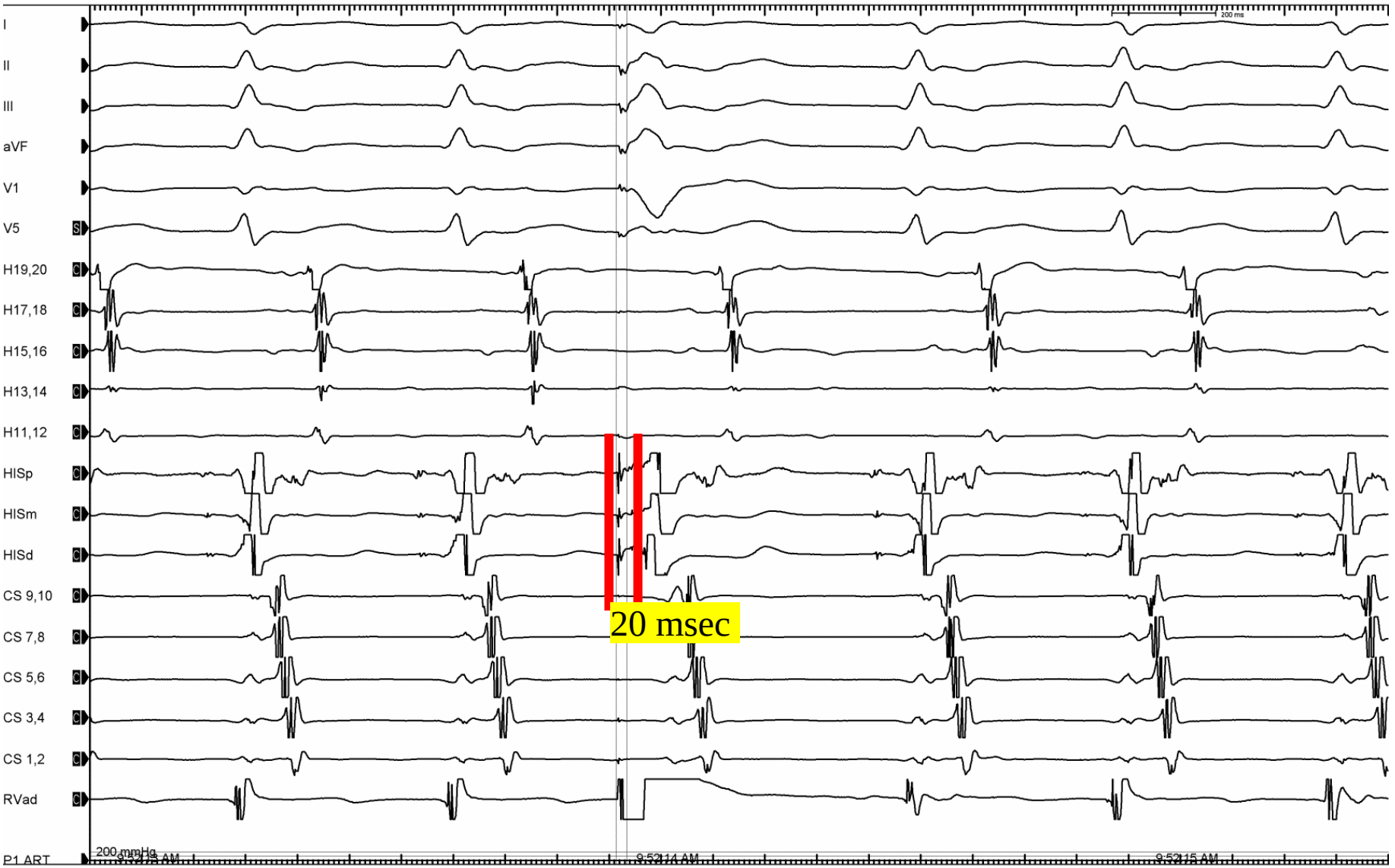
Induction with AH Jump and Concentric Atrial Activation



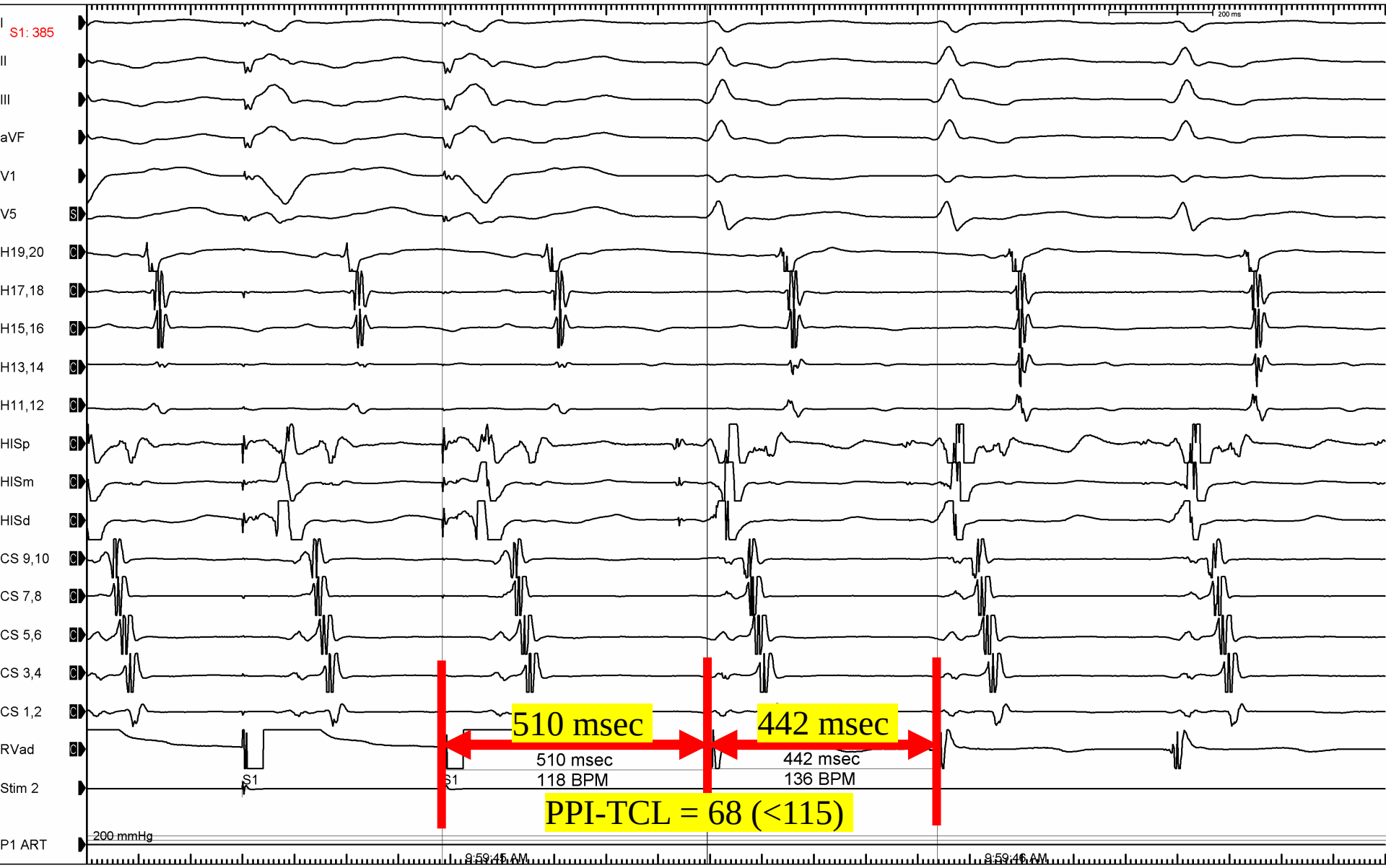
His Synchronious Ventricular Extrastimulation (V reset)



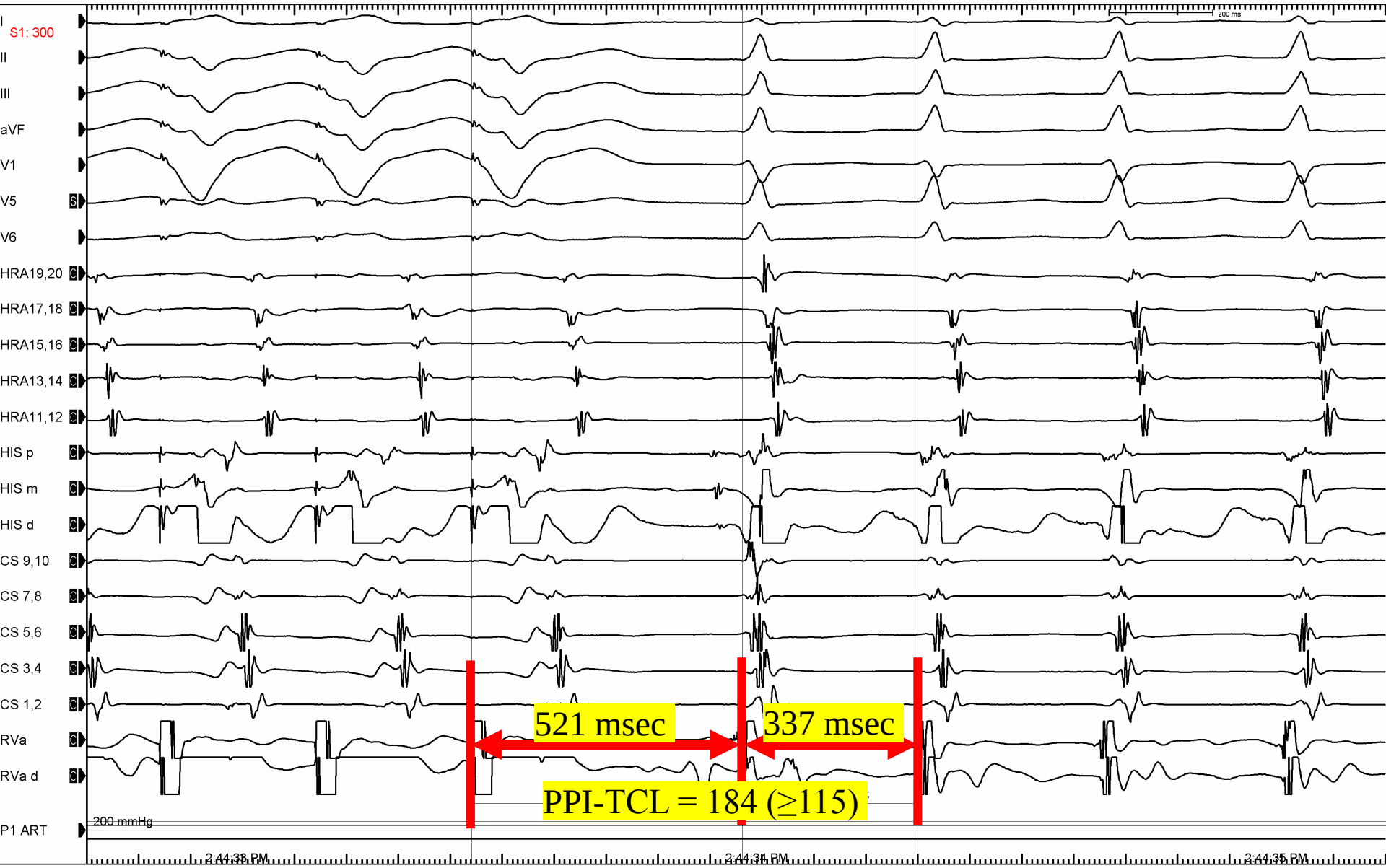
His Synchronious Ventricular Extrastimulation (V reset)



PPI-TCL: AVRT

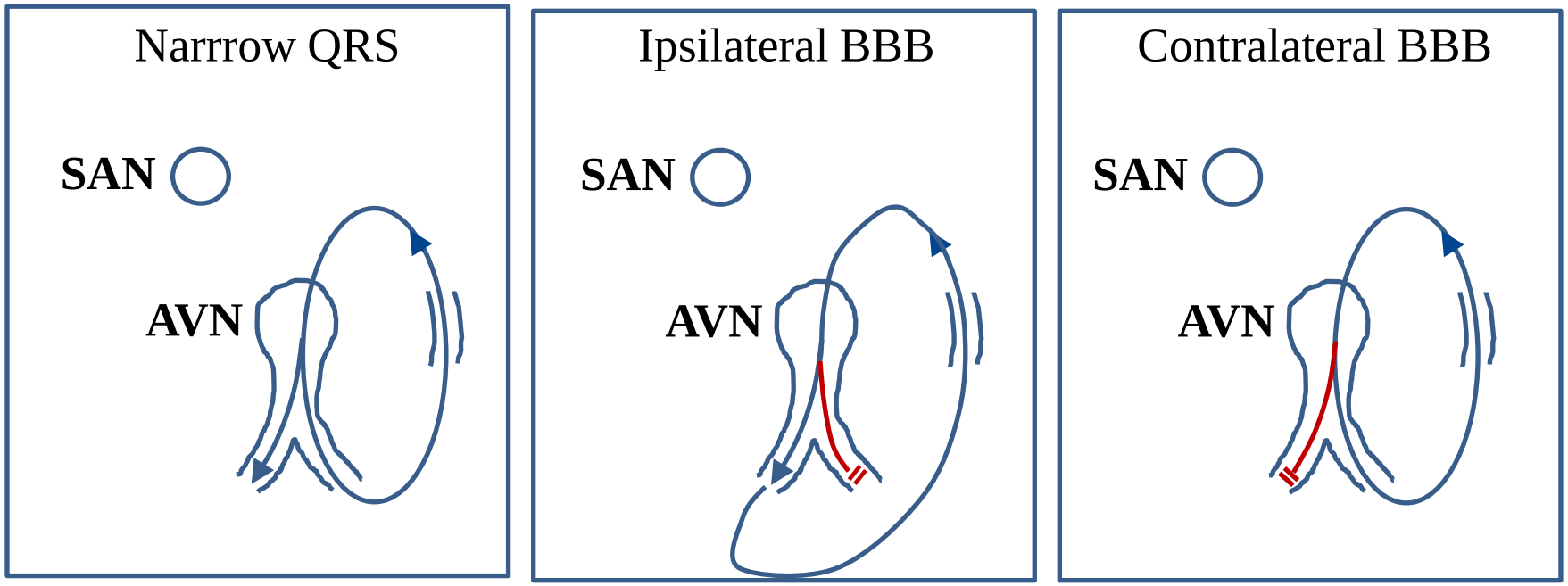

I
S1: 385
II
III
aVF
V1
V5
H19,20
H17,18
H15,16
H13,14
H11,12
HISp
HISm
HISd
CS 9,10
CS 7,8
CS 5,6
CS 3,4
CS 1,2
RVad
Stim 2
P1 ART
200 ms
200 mmHg
S1
S1
510 msec
510 msec
118 BPM
442 msec
442 msec
136 BPM
PPI-TCL = 68 (<115)
9:59:45 AM
9:59:46 AM

PPI-TCL: AVNRT

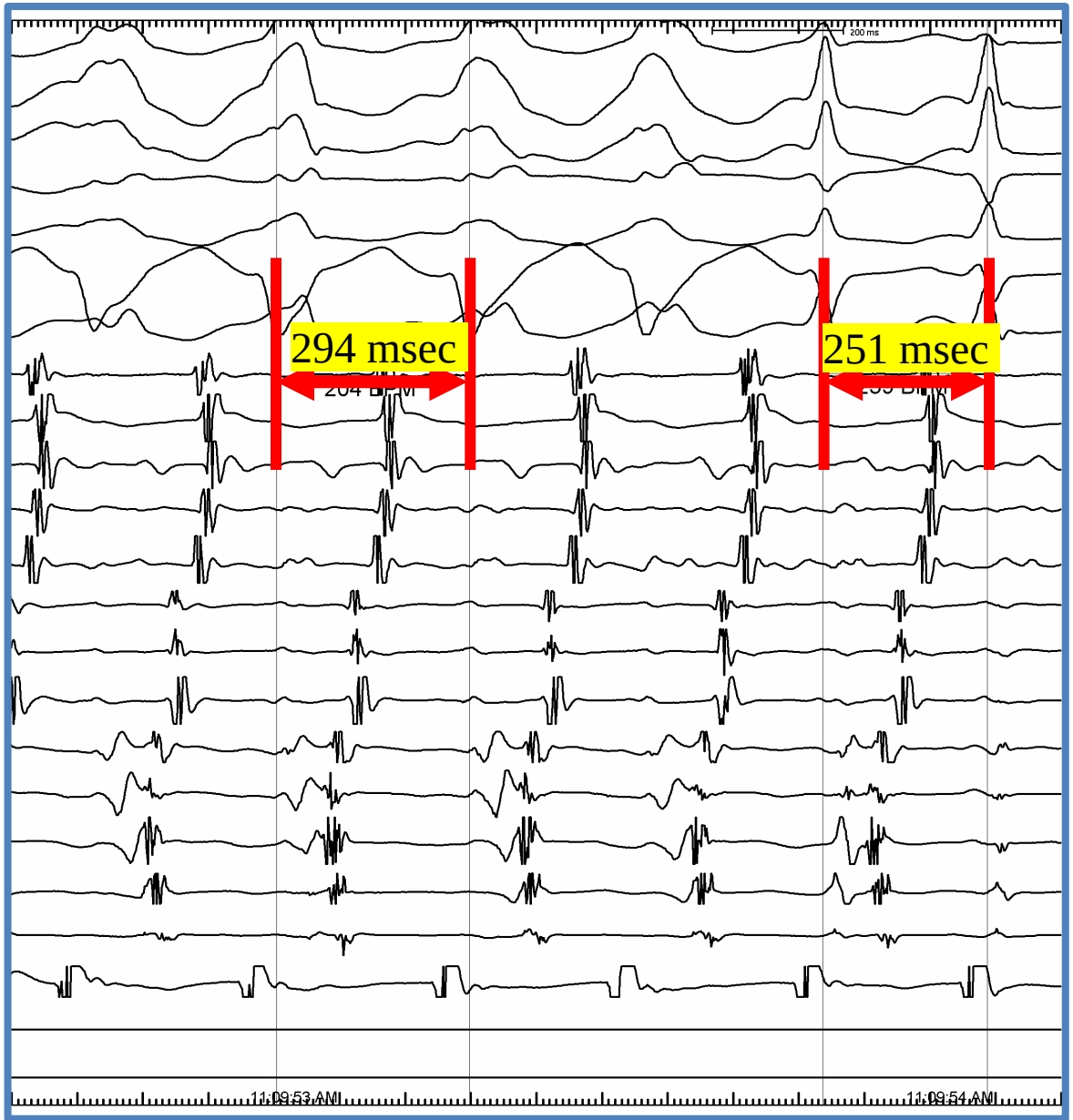


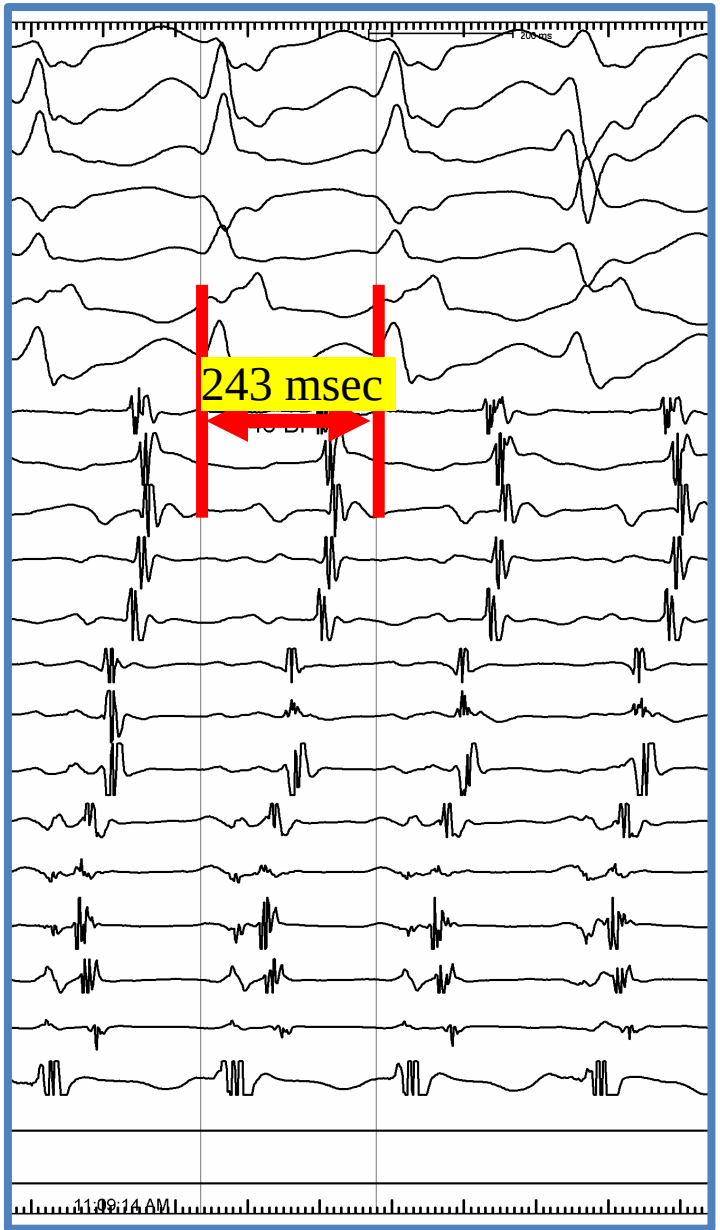
Coumel's law

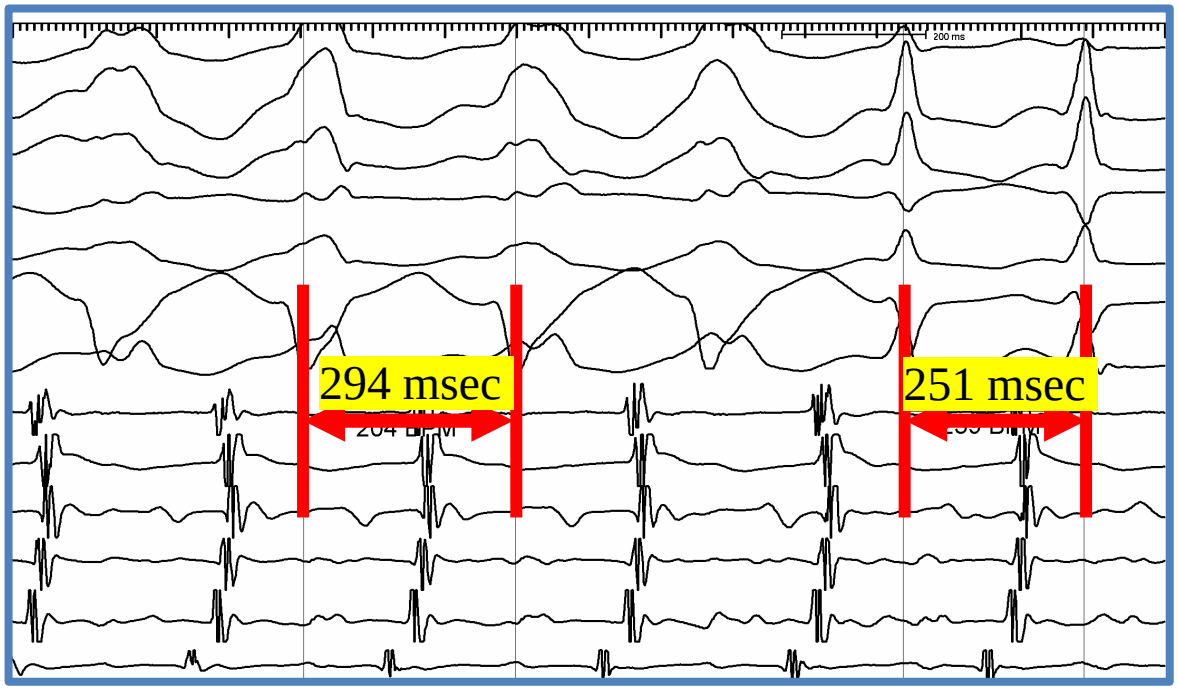
- Positive Prediction Value: 100% in ORT
- Ipsilateral BBB produces prolonged TCL

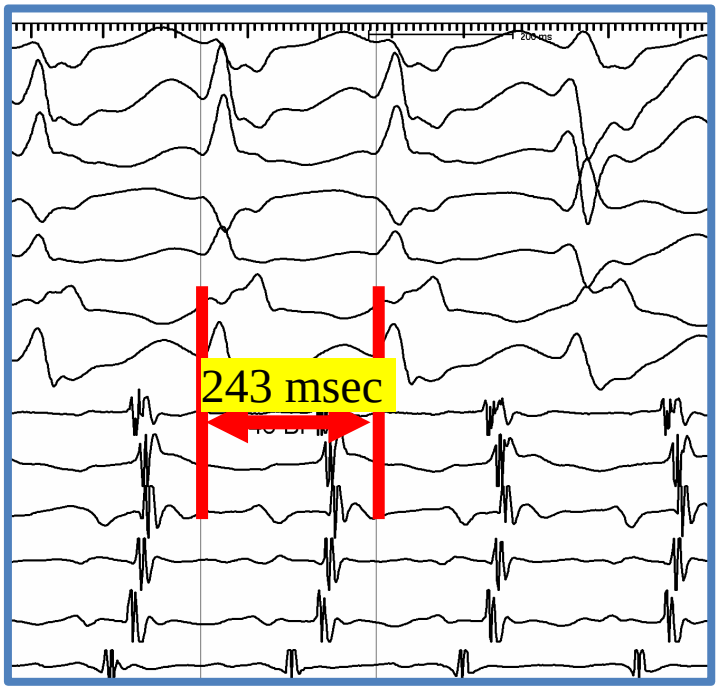


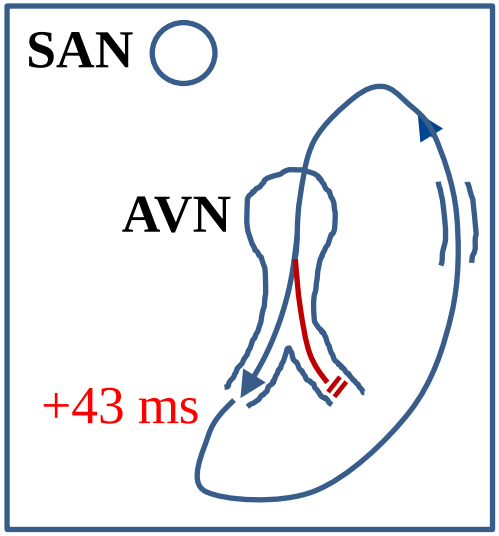
Coumel's law





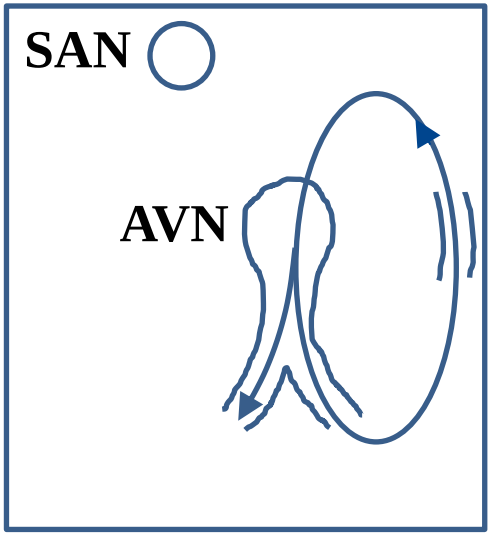






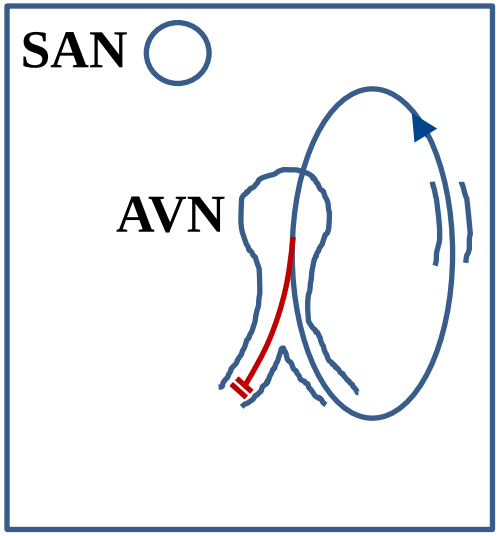
Ipsilateral BBB

TCL: 294 ms



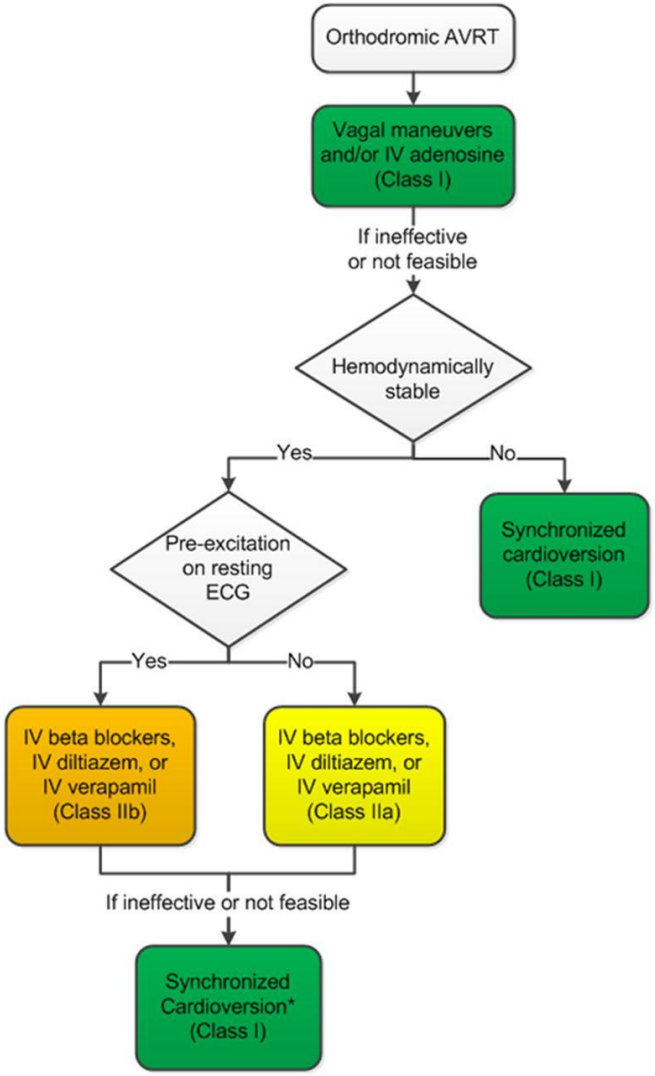
Narrrow QRS

TCL: 251 ms



Contralateral BBB

Acute treatment of orthodromic AVRT



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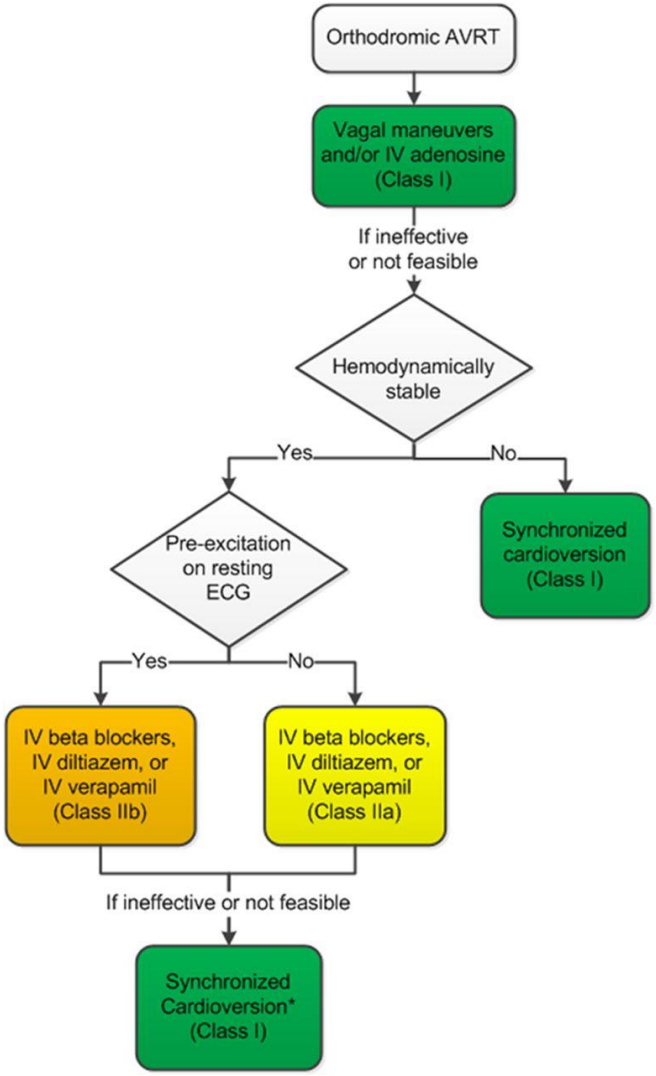
Recommendations for Acute Treatment of Orthodromic AVRT

COR	LOE	Recommendations
I	B-R	**1. Vagal maneuvers are recommended for acute treatment in patients with orthodromic AVRT.[42,75,235,259]**
See Online Data Supplements 11 and 12.		For acute conversion of orthodromic AVRT, vagal maneuvers, including Valsalva and carotid sinus massage, can be performed quickly and should be the first-line intervention to terminate SVT. These maneuvers should be performed with the patient in the supine position. There is no "gold standard" for proper Valsalva maneuver technique, but in general, the patient raises intrathoracic pressure by bearing down against a closed glottis for 10 to 30 seconds, equivalent to at least 30 to 40 mm Hg.[82,84] Carotid massage is performed after absence of bruit has been confirmed by auscultation, by applying steady pressure over the right or left carotid sinus for 5 to 10 seconds.[83,84] Another vagal maneuver based on the classic diving reflex consists of applying an ice-cold, wet towel to the face[85]; in a laboratory setting, facial immersion in water at 10°C (50°F) has proved effective in terminating tachycardia, as well.[86] One study involving 148 patients with SVT demonstrated that Valsalva was more successful than carotid sinus massage, and switching from 1 technique to the other resulted in an overall success rate of 27.7%.[82] The practice of applying pressure to the eyeball is potentially dangerous and has been abandoned.
I	B-R	**2. Adenosine is beneficial for acute treatment in patients with orthodromic AVRT.[42,260,261]**
See Online Data Supplements 11 and 12.		Adenosine is effective for conversion of orthodromic AVRT in 90% to 95% of patients, with minor and brief (<1 min) side effects occurring in approximately 30% of patients.[42,260,261] Patients often have atrial or ventricular premature complexes immediately after conversion that, on occasion, may induce further episodes of AVRT. In this situation, an antiarrhythmic drug may be required to prevent acute reinitiation of tachycardia. Because adenosine may precipitate AF that may then conduct rapidly to the ventricle and even cause ventricular fibrillation, electrical cardioversion should be available.
I	B-NR	**3. Synchronized cardioversion should be performed for acute treatment in hemodynamically unstable patients with AVRT if vagal maneuvers or adenosine are ineffective or not feasible.[75,262,263]**
See Online Data Supplement 10.		Synchronized cardioversion is highly effective in terminating AVRT.[75] Cardioversion avoids complications associated with antiarrhythmic drug therapy and should be considered early in the management of hemodynamically unstable patients. Patients often have atrial or ventricular premature complexes immediately after cardioversion that, on occasion, may induce further episodes of AVRT. In this situation, an antiarrhythmic drug may be required to prevent acute reinitiation of tachycardia.
I	B-NR	**4. Synchronized cardioversion is recommended for acute treatment in hemodynamically stable patients with AVRT when pharmacological therapy is ineffective or contraindicated.[87,95]**
See Online Data Supplements 3 and 10.		Synchronized cardioversion is highly effective in terminating SVT (including AVRT and AVNRT), and when the patient is stable, this is performed after adequate sedation or anesthesia.[94] Most stable patients with SVT respond to pharmacological therapy, with success rates of 80% to 98% for agents such as verapamil, diltiazem, or adenosine. In some resistant cases, a second drug bolus or higher dose of initial drug agent might prove effective.[87,96] Nevertheless, in rare instances, drugs may fail to successfully restore sinus rhythm.
I	B-NR	**5. Synchronized cardioversion should be performed for acute treatment in hemodynamically unstable patients with pre-excited AF.[75,94]**
See Online Data Supplement 10.		Synchronized cardioversion is highly effective in terminating pre-excited AF.[75] When AF occurs in patients with ventricular pre-excitation, if the accessory pathway has a short refractory period, this may allow for rapid pre-excited AV conduction; the resulting fast, often irregular, broad-complex tachycardia is often unstable and may lead to ventricular fibrillation. It is therefore important to achieve early restoration of sinus rhythm in these patients. Patients often have atrial or ventricular premature complexes immediately after cardioversion that, on occasion, may induce AVRT or recurrent pre-excited AF.
I	C-LD	**6. Ibutilide[264] or intravenous procainamide[265] is beneficial for acute treatment in patients with pre-excited AF who are hemodynamically stable.**
See Online Data Supplements 11 and 12.		Small observational studies support the use of ibutilide or intravenous procainamide for the treatment of pre-excited AF in patients who are not hemodynamically compromised.[264,265] Both medications can decrease ventricular rate by slowing conduction over the accessory pathway and have the additional benefit of possibly terminating AF.[264,265]
IIa	B-R C-LD	**1. Intravenous diltiazem, verapamil[42,260,266,267] (*Level of Evidence: B-R*), or beta blockers[268] (*Level of Evidence: C-LD*) can be effective for acute treatment in patients with orthodromic AVRT who do not have pre-excitation on their resting ECG during sinus rhythm.**
See Online Data Supplements 11 and 12.		Intravenous diltiazem or verapamil effectively terminate approximately 90% to 95% of AVRT episodes in patients without pre-excitation on their resting sinus-rhythm ECG, with drug-induced hypotension occurring in approximately 3% of patients.[42,260,266,267] Intravenous beta blockers have not been studied in clinical trials; however, clinical experience suggests they are useful for terminating AVRT, with a low risk of associated complications.[268]

Acute treatment of orthodromic AVRT



Recommendations for Acute Treatment of Orthodromic AVRT (Continued)		
COR	LOE	Recommendations
IIb	B-R	1. Intravenous beta blockers, diltiazem, or verapamil might be considered for acute treatment in patients with orthodromic AVRT who have pre-excitation on their resting ECG and have not responded to other therapies.[42,266,267,269]
See Online Data Supplements 11 and 12.		Intravenous beta blockers, diltiazem, and verapamil have a risk of enhancing conduction over the accessory pathway if the AVRT converts to AF during administration of the medication. Should the patient have a rapidly conducting manifest accessory pathway, further enhancing accessory-pathway conduction during AF by shortening the refractory period (digoxin) or decreasing BP and increasing catecholamines (diltiazem, beta blockers, verapamil) may place the patient at risk of AF degenerating into a malignant ventricular arrhythmia. The ability to promptly perform electrical cardioversion must be available should AF with rapid ventricular conduction occur. Before intravenous beta blockers, diltiazem, and verapamil were available, intravenous digoxin was commonly used for acute treatment of patients with orthodromic AVRT who had pre-excitation on their resting ECG[270]; this agent is rarely used now because other agents are available and digoxin may put patients at risk of ventricular fibrillation.[271]
III: Harm	C-LD	1. Intravenous digoxin, intravenous amiodarone, intravenous or oral beta blockers, diltiazem, and verapamil are potentially harmful for acute treatment in patients with pre-excited AF.[269,271–275]
See Online Data Supplements 11 and 12.		Patients with pre-excited AF should not receive intravenous digoxin, intravenous amiodarone, or intravenous/oral beta blockers, diltiazem, or verapamil because these medications may enhance conduction over the accessory pathway, increase the ventricular rate, and increase the risk of provoking a life-threatening ventricular arrhythmia.[269,271–275] Digoxin increases the ventricular rate by shortening refractoriness of the accessory pathway, whereas amiodarone, beta blockers, diltiazem, and verapamil may increase the ventricular rate as a result of drug-induced hypotension with increased catecholamines. In addition, these medications may enhance conduction over the accessory pathway by slowing or blocking conduction through the AV node, preventing competitive concealed retrograde conduction into the accessory pathway.

Richard L. Page et al. Circulation. 2016;133:e506-e574

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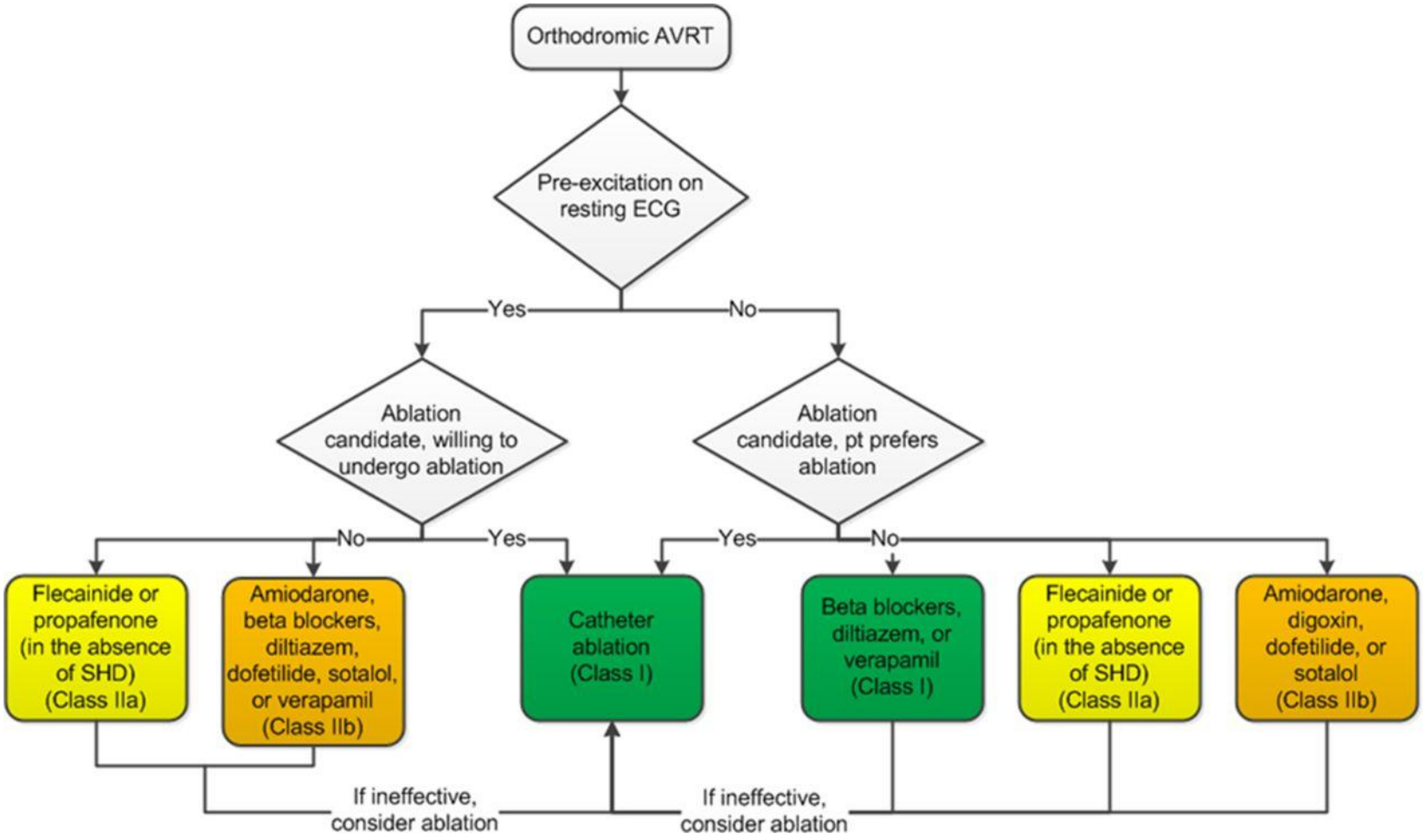
Ongoing management of orthodromic AVRT



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Success and Complication Rate for Ablation of AVRT

- Acute success rate: 93%
- Recurrent Rate: 8%
- Major Complications

  Overall 2.8%

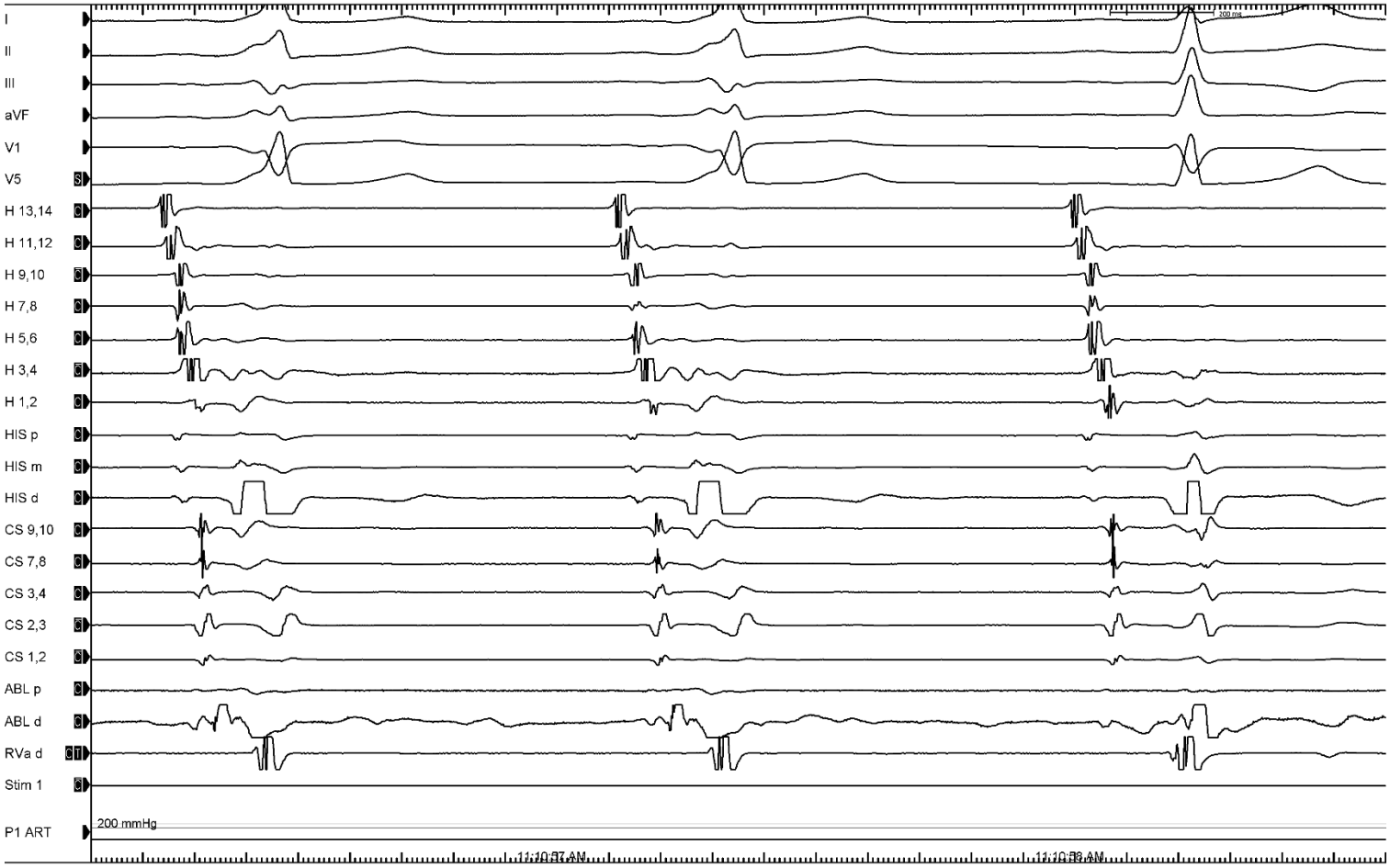
  PPM 0.3%

  Death 0.1%

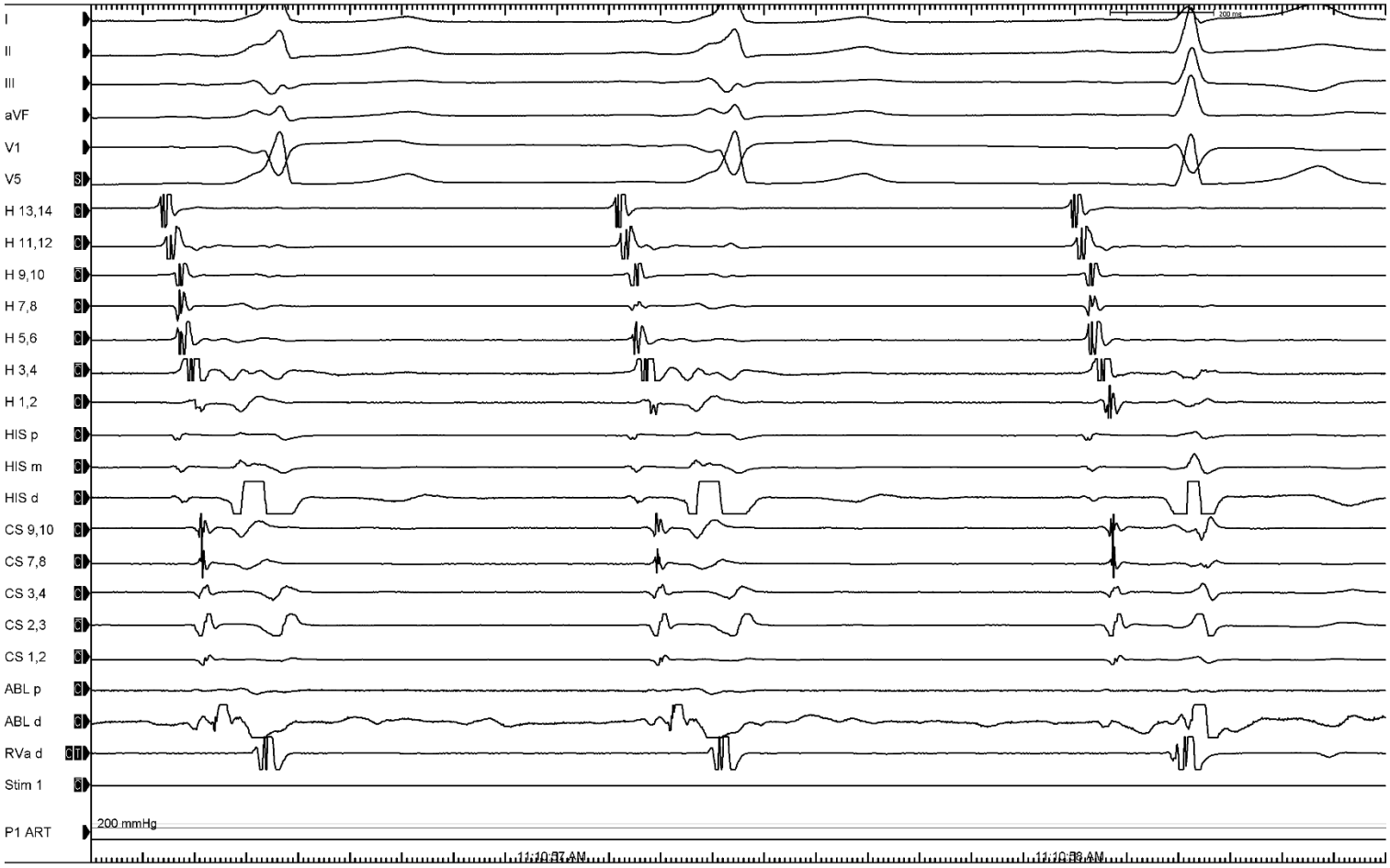
  Tamponade 0.4%

Spector P, et al. Am J Cardiol. 2009;104:671–7.
Calkins H, et al. Circulation. 1999;99:262–70.

RFCA


I
II
III
aVF
V1
V5
H 13,14
H 11,12
H 9,10
H 7,8
H 5,6
H 3,4
H 1,2
HIS p
HIS m
HIS d
CS 9,10
CS 7,8
CS 3,4
CS 2,3
CS 1,2
ABL p
ABL d
RVa d
Stim 1
P1 ART
200 mmHg
200 ms

Hooray!!


I
II
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H 1,2
HIS p
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CS 1,2
ABL p
ABL d
RVa d
Stim 1
P1 ART
200 mmHg
200 ms

Thank you for your attention