SVT

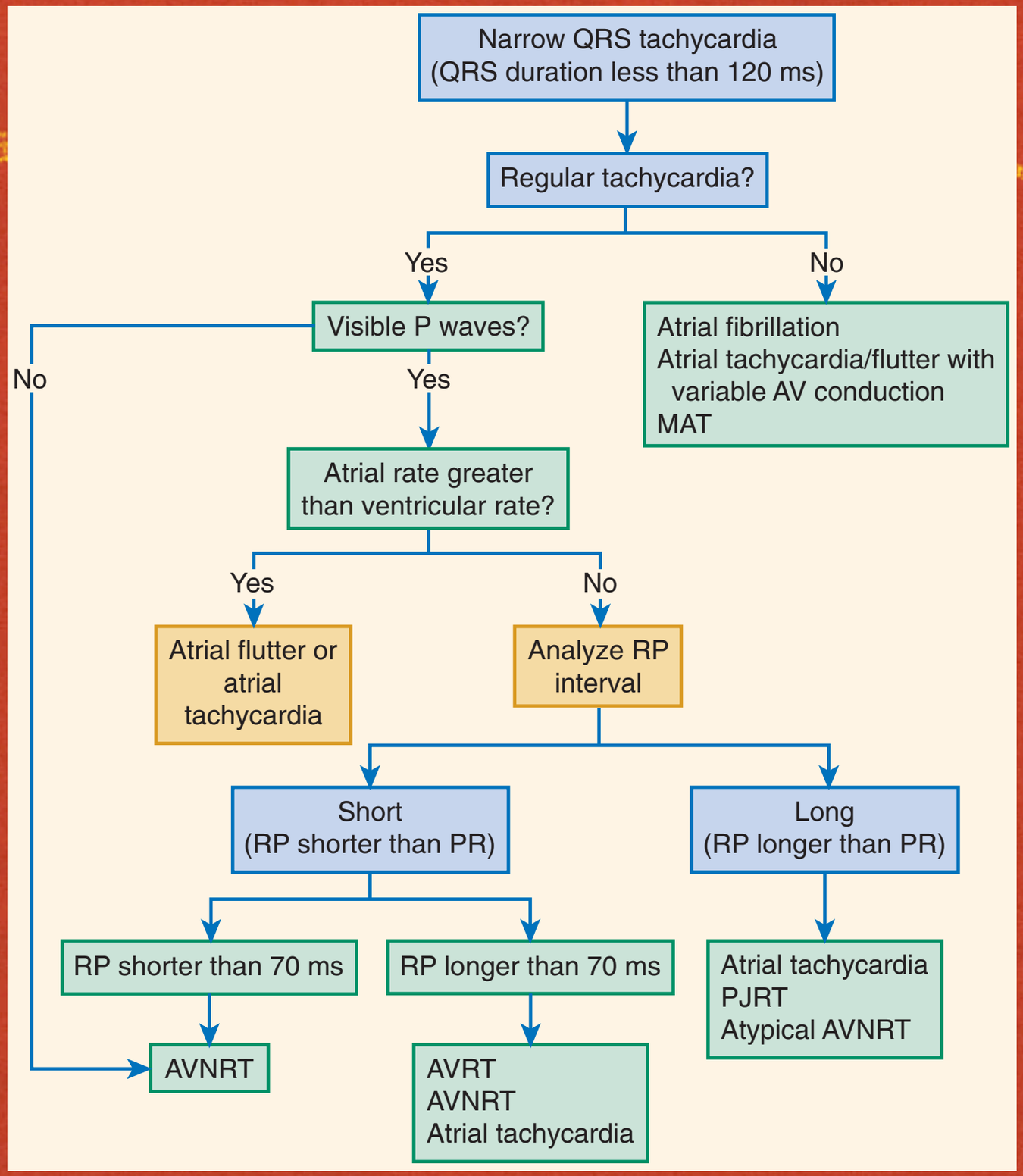
Mitul Afiniwala

10/30/13

SVT

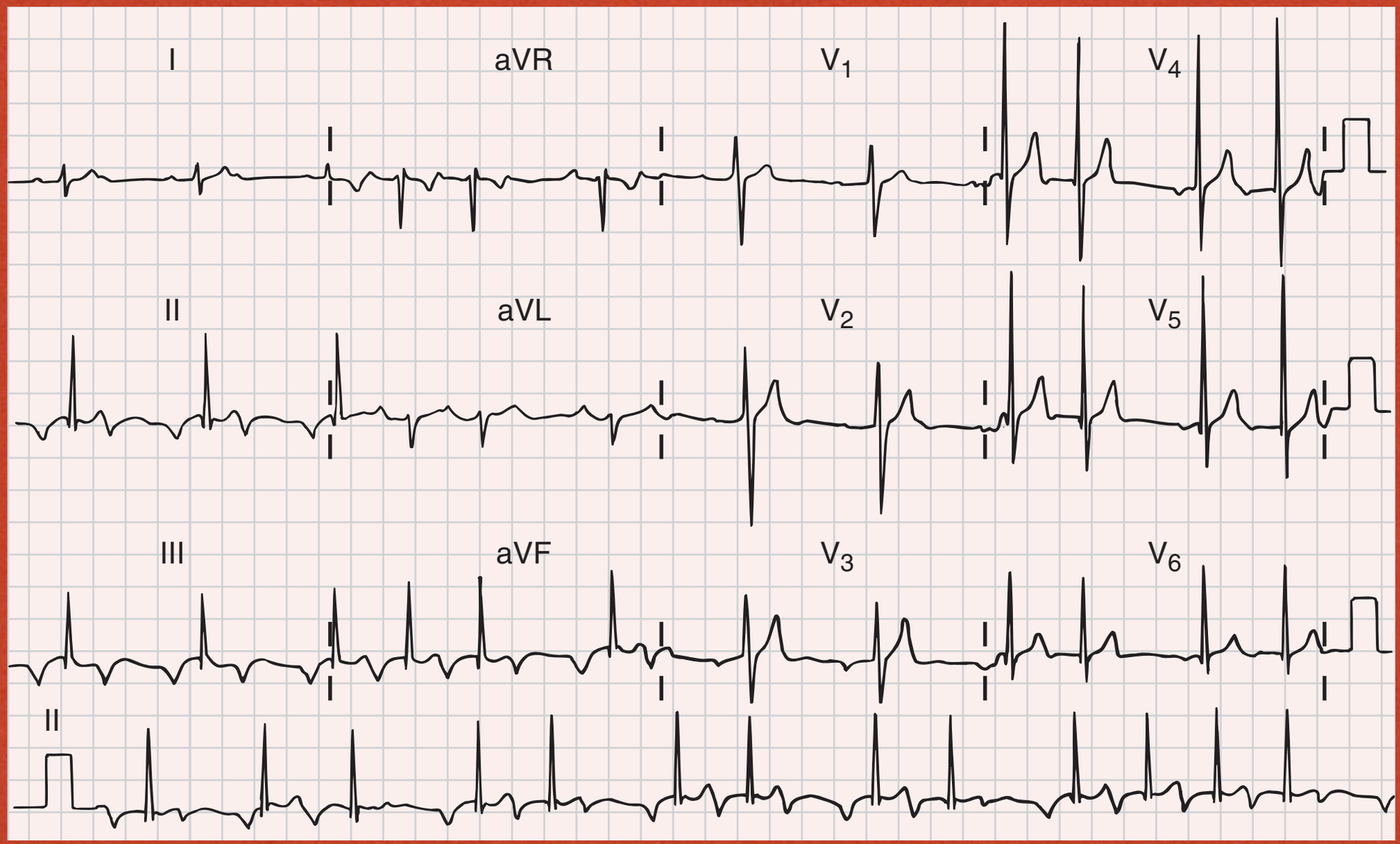
- tachycardia in which the driving circuit or focus originates, at least in part, in tissue above the level of the ventricle (i.e., sinus node, atria, AV node, or His bundle)
- usually not lethal
- often does not result in hemodynamic compromise
- more conservative measures can be applied initially to convert to sinus rhythm

DIAGNOSIS OF A NARROW QRS TACHYCARDIA



SVT versus VT

SUPPORTS SVT	SUPPORTS VT
Slowing or termination by vagal tone	Fusion beats
Onset with premature P wave	Capture beats
RP interval ≤100 msec	AV dissociation
P and QRS rate and rhythm linked to suggest that ventricular activation depends on atrial discharge, e.g., 2 : 1 AV block rSR′ V_1	P and QRS rate and rhythm linked to suggest that atrial activation depends on ventricular discharge, e.g., 2 : 1 VA block
Long-short cycle sequence	"Compensatory" pause
	Left-axis deviation; QRS duration >140 msec
	Specific QRS contours (see text)


I
aVR
V1
V4
II
aVL
V2
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III
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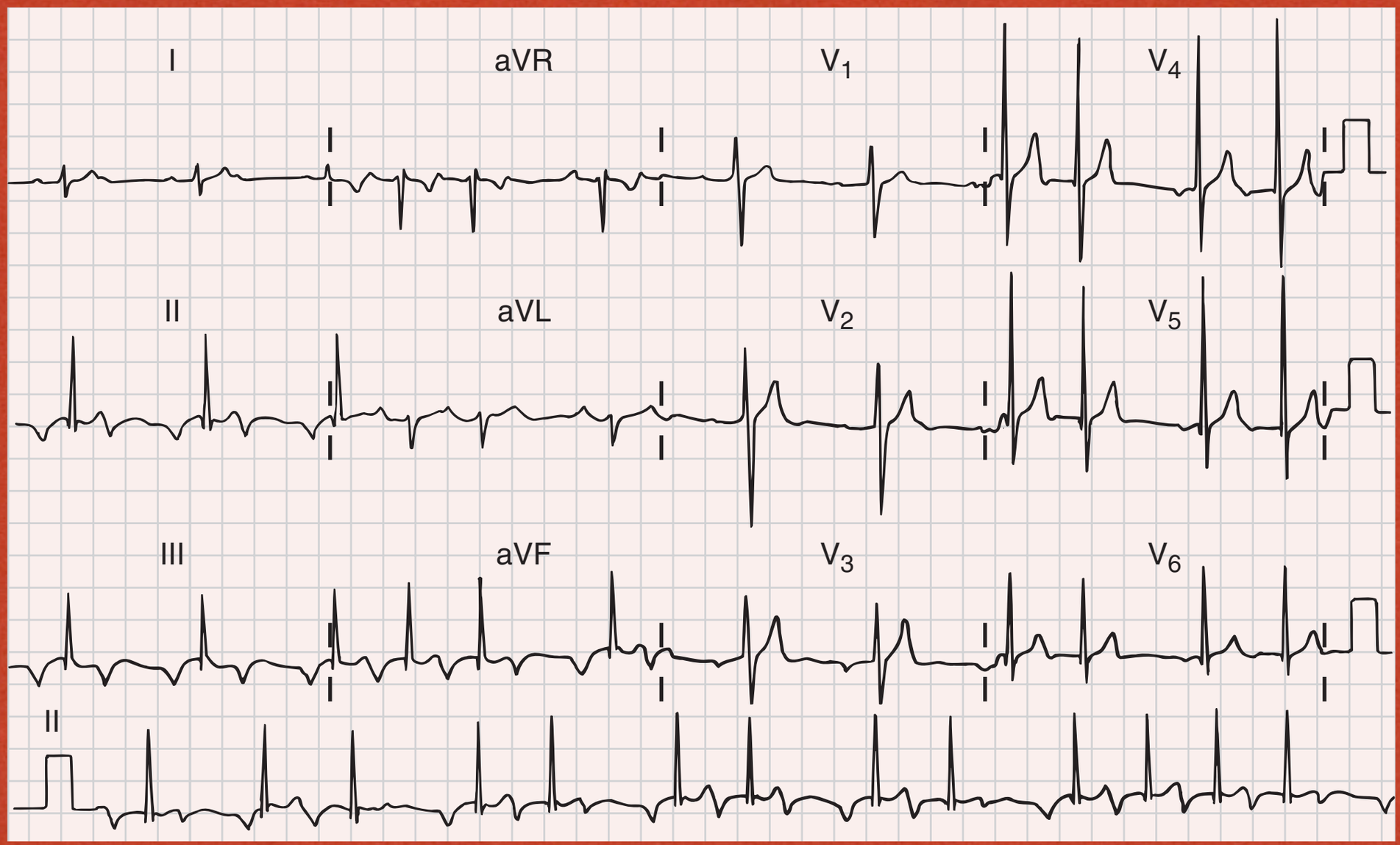
II

ATRIAL TACHYCARDIA



long RP

tachycardia persists despite block

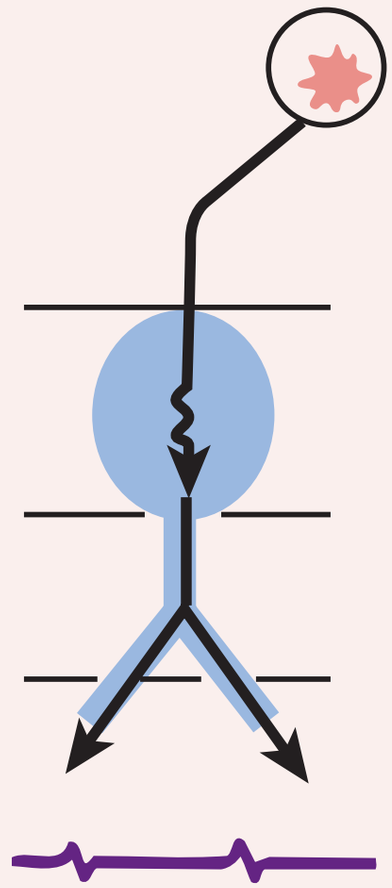
Atrium

AV Node

His

Ventricle

RA

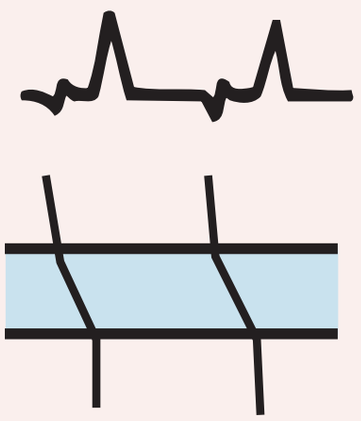


ECG II

A

AV

V



Atrial automatic

ATRIAL TACHYCARDIA

- CAD +/- MI
- Cor Pulmonale
- Digitalis intoxication
- without structural heart disease
- K depletion in patients taking digitalis

ATRIAL TACHYCARDIA

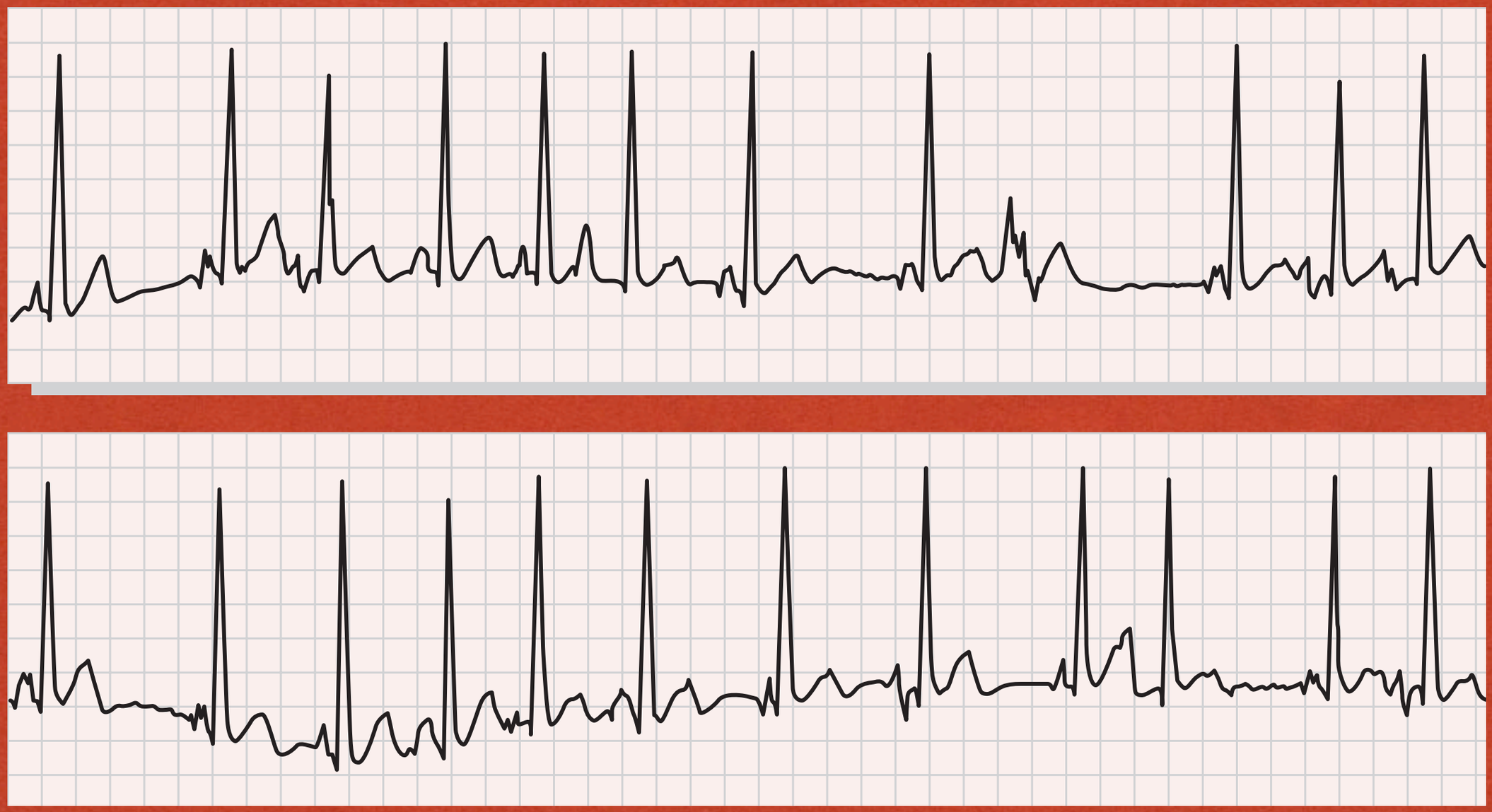
- often occur in short, recurrent bursts
- occasionally incessant
- can cause tachycardia induced cardiomypathy
- exercise, stress, change of position, caffeine, chocolate and ephedrine can provoke episodes

ATRIAL TACHYCARDIA

- S1 intensity varies, SBP varies as AV block and PR interval varies
- excess *a* waves in the JVP
- Carotid massage: increases the AV block, slows the ventricular rate in a stepwise fashion without terminating the tachycardia (most cases)

ATRIAL TACHYCARDIA

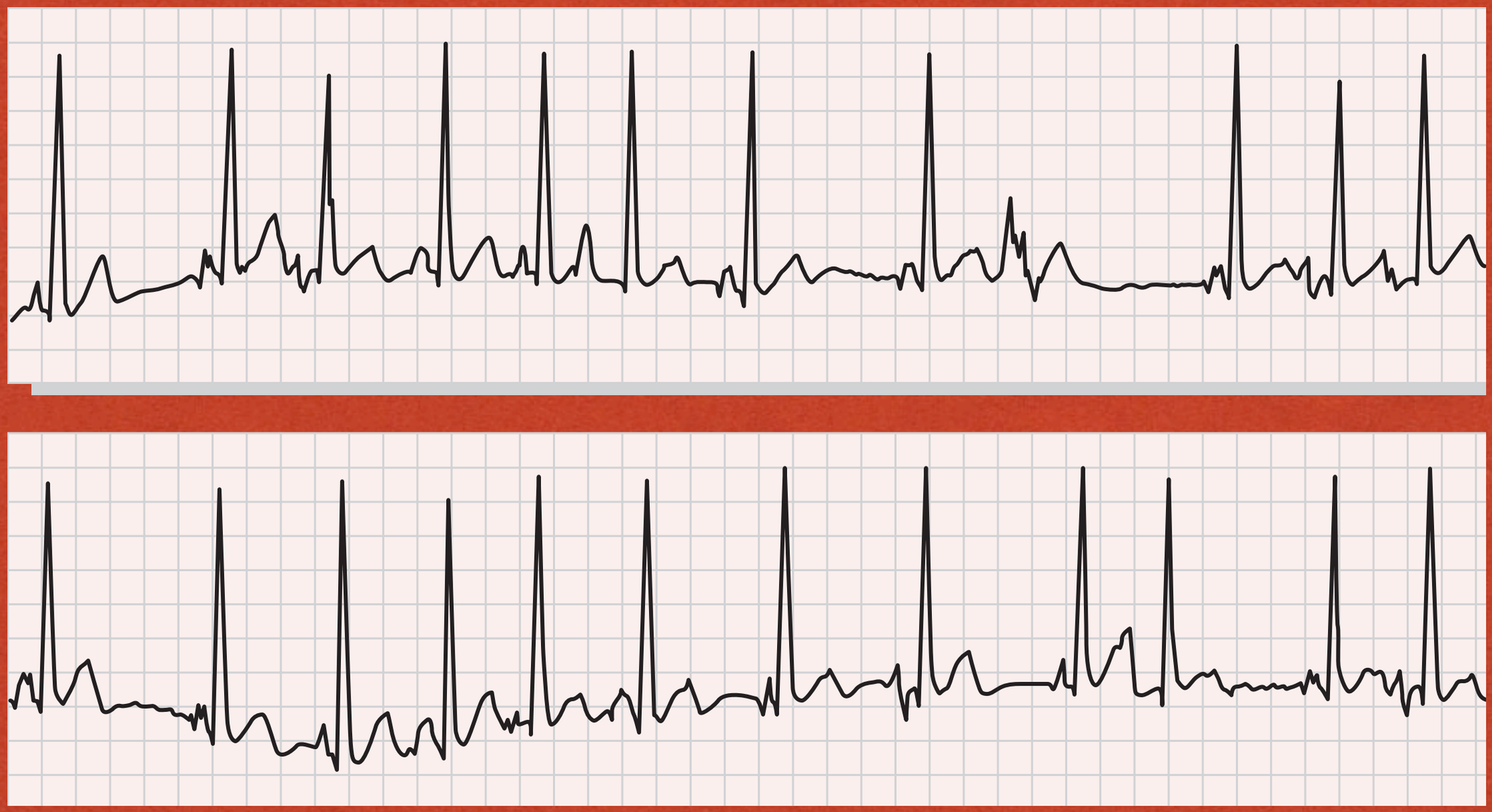
- Rx: Dig, BB or CCB
- class IA, IC or III AAD can be added
- catheter ablation is usually effective
- occasionally can recur at a different site after a successful ablation
- For Dig induced: stop digoxin, give dig AB or K



MAT

atrial rate 100-130

irregular P-P interval



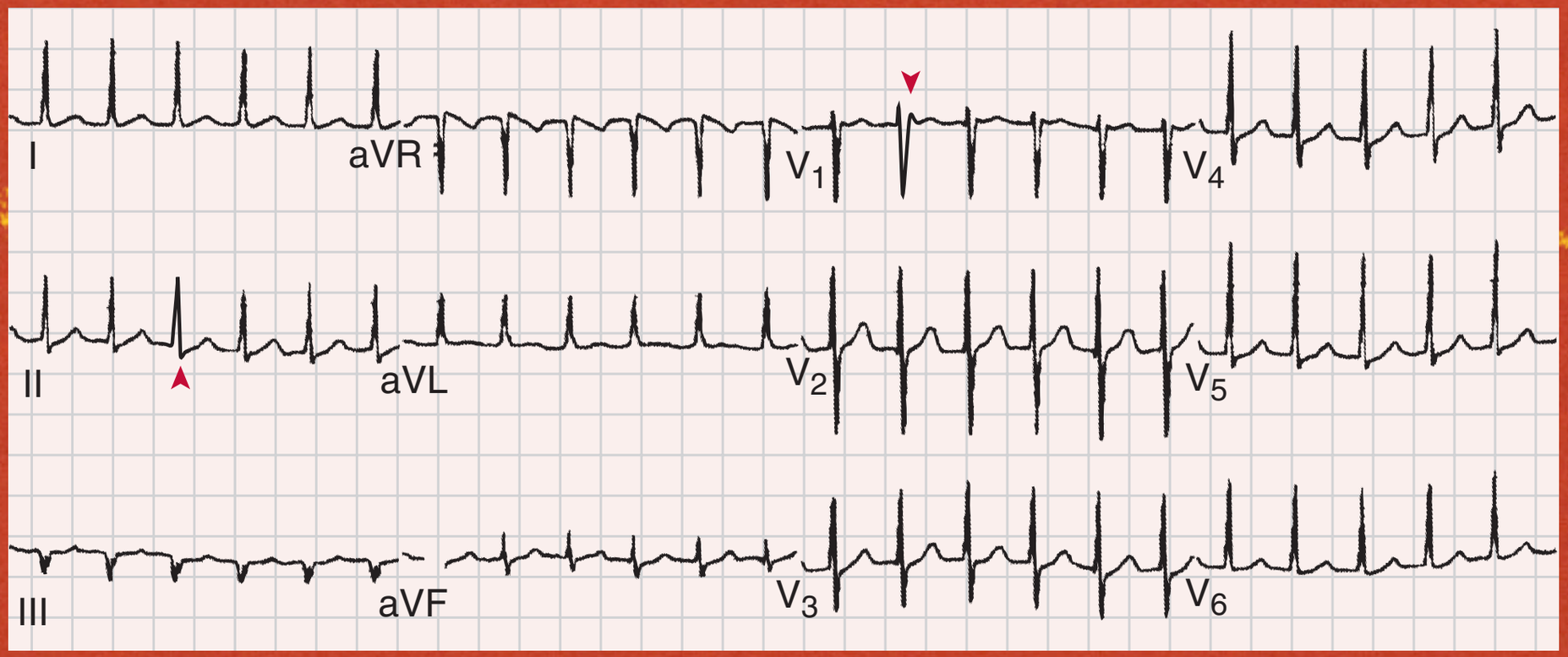
atleast 3 p wave contours

MAT

- older patients with COPD and CHF
- digitalis is an unusual cause
- may eventually develop into afib
- theophylline has been implicated

MAT

- Rx the underlying cause
- AADs are often ineffective
- BB should be avoided in bronchospastic disease
- Verapamil and amiodarone have been useful
- K and Mg replacement may suppress it
- Ablation effective in some cases


I
aVR
V1
V4
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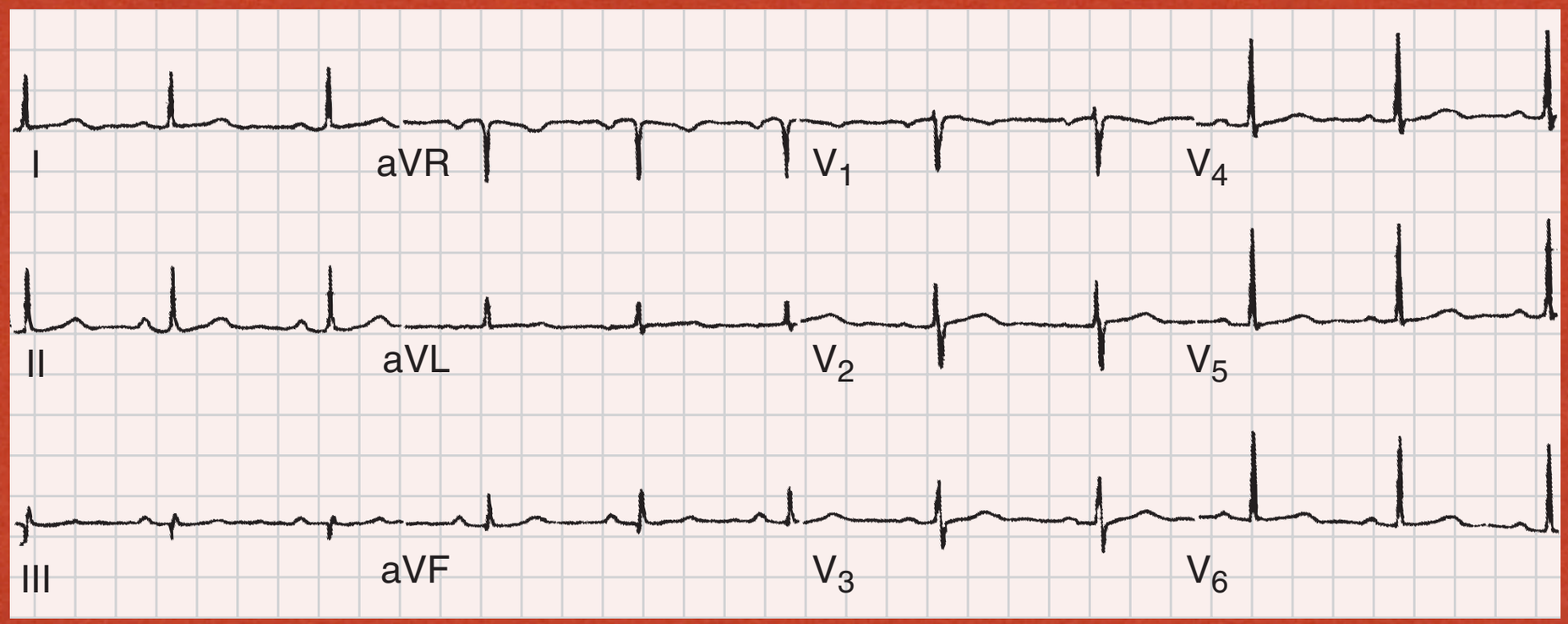
I
aVR
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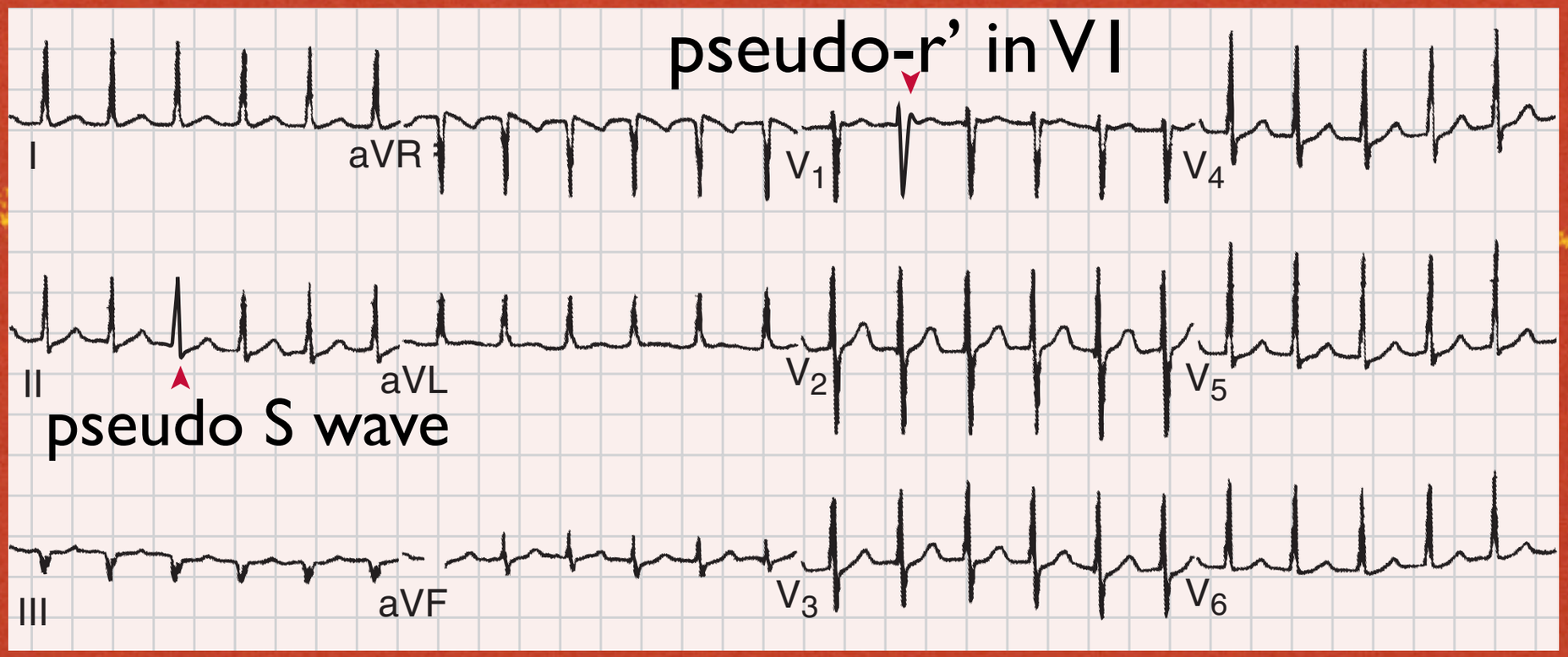
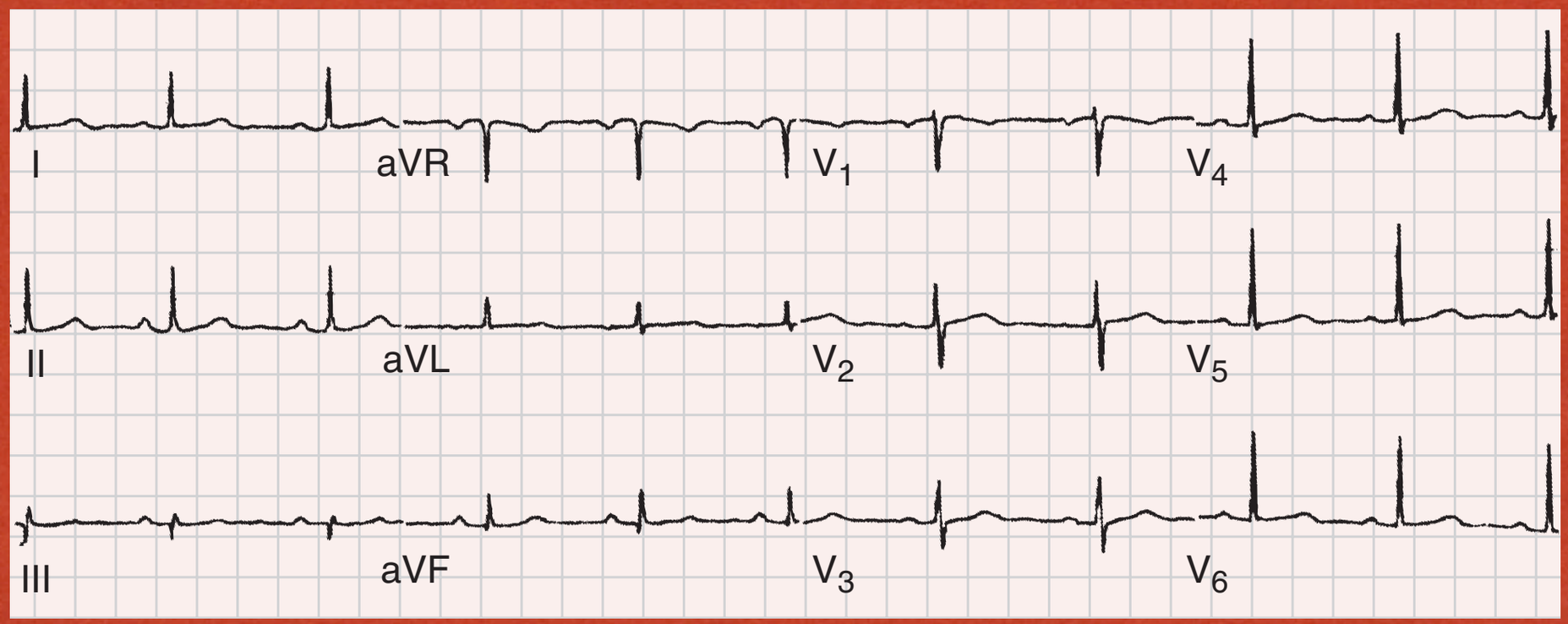
V3
V6

AVNRT


pseudo-r' in V1
I
aVR
V1
V4
II
aVL
V2
V5
pseudo S wave
III
aVF
V3
V6


I
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V1
V4
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V2
V5
III
aVF
V3
V6

TACHYCARDIAS INVOLVING THE AV JUNCTION

- supraventricular complex + regular R-R interval + no evidence of ventricular preexcitation

- Paroxysmal SVT: accounts for the various EP mechanisms; may be inappropriate as tachycardias in patients with accessory pathways require participation of both atria and the ventricles.

- Reciprocating tachycardia: offered as a substitute, but presumes the mechanism to be reentrant

AVNRT

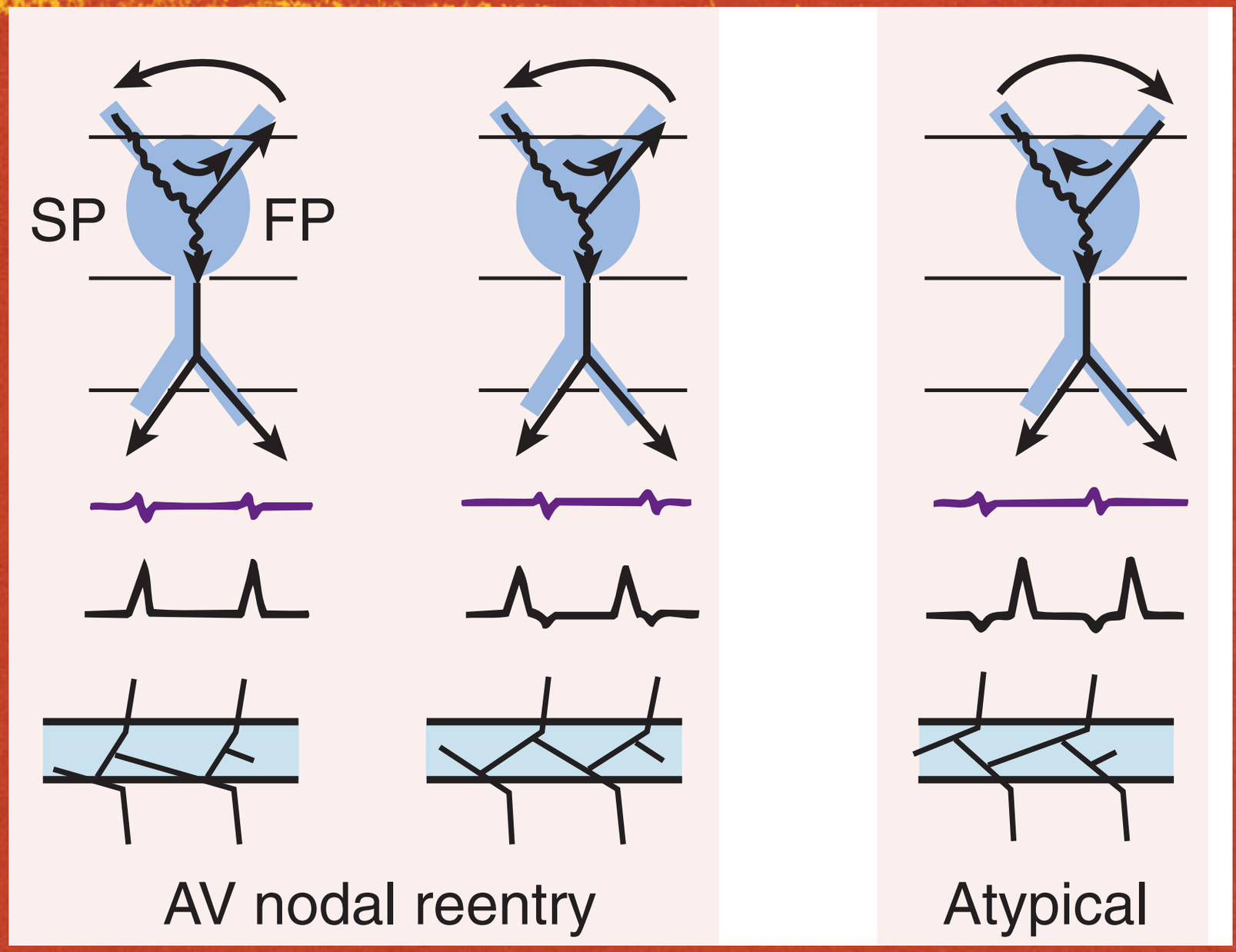
- sudden onset and termination
- rate 150 to 250 bpm (usually 180-200)
- p waves generally buried in the QRS complex, often occur just before or after (30%) the QRS (pseudo-S or pseudo-r')
- begins abruptly usually after a PAC that conducts with a prologed PR interval
- the R-R interval can shorten over the course of the 1st few beats at the onset or lengthen during the last few beats prior to the termination (variation in antergrade AV nodal conduction time)
- cycle length or QRS alternans can occur

AVNRT ~ EP FEATURES

- precipitated by an atrial complex that conducts with a critical prolongation of the AV nodal conduction time
- presence of differential atrial inputs into the AV node, the fast and the slow pathways
- the atria are a necesary link between the fast and slow pathways: discrete (anisotropy) vs. functional

AVNRT


Atrium
AV Node
His
Ventricle
RA
ECG II
A
AV
V

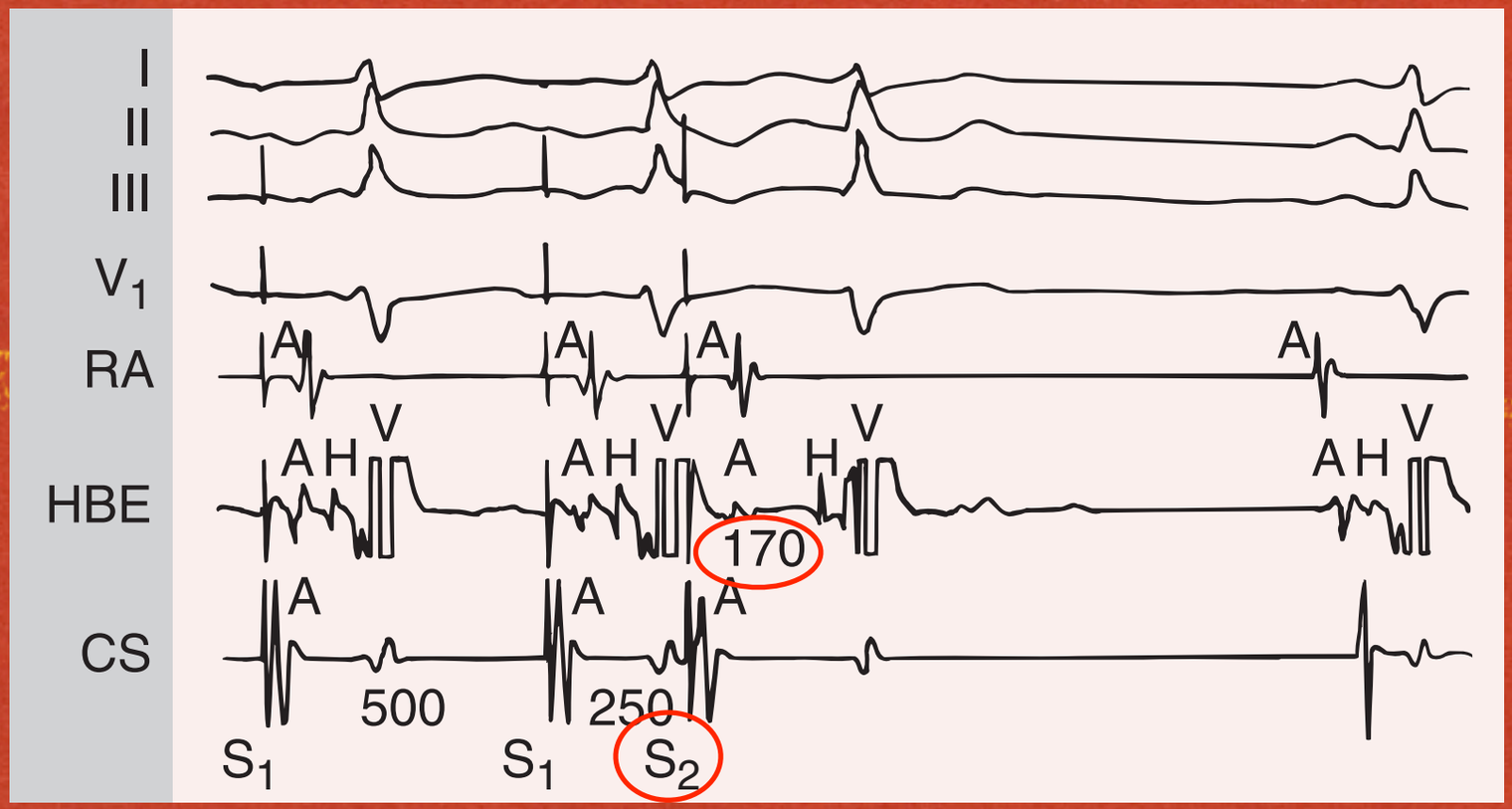

SP
FP
AV nodal reentry
Atypical

AVNRT

- **typical** AVNRT: down the slow and up the fast, early atrial complex blocks the antegrade fast pathway and conducts down the slow, circus movement, slow-fast reentry
- **atypical** AVNRT: down the fast and up the slow, < 5-10%
- slow-slow reentry: rentry over 2 slow or a slow and an intermediate pathway
- conduction time in the slow pathway is a major determinant of the cycle length of the tachycardia

TYPICAL AVNRT

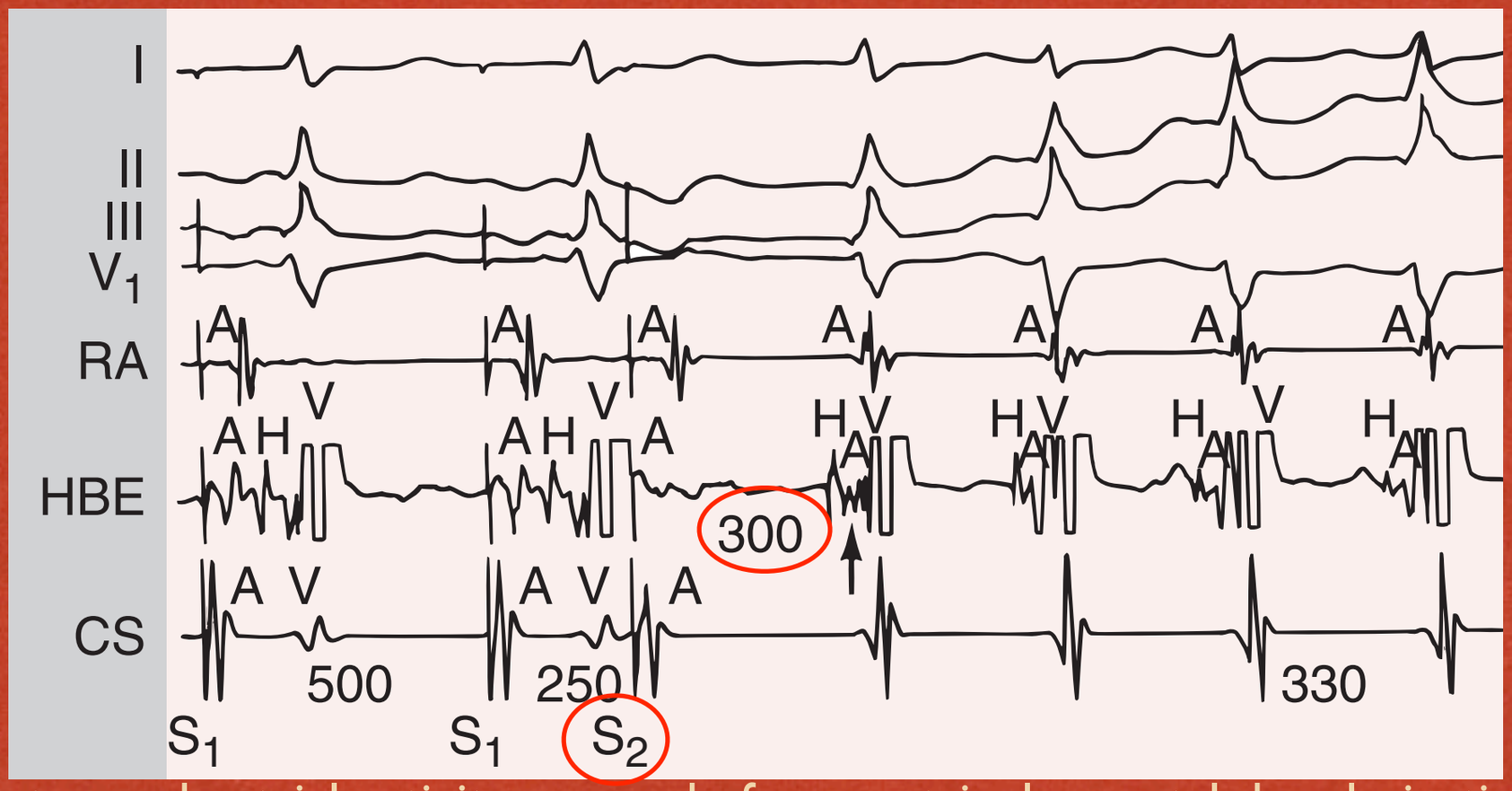
- usually VA interval < 50% the R-R interval (short RP)
- usually AV to VA ratio >1.0
- VA interval is longer in patients with tachycardia related to accessory pathways and in atypical AVNRT



sinus

premature atrial stimuli, S1-S2 interval 250 ms

AVNRT initiated



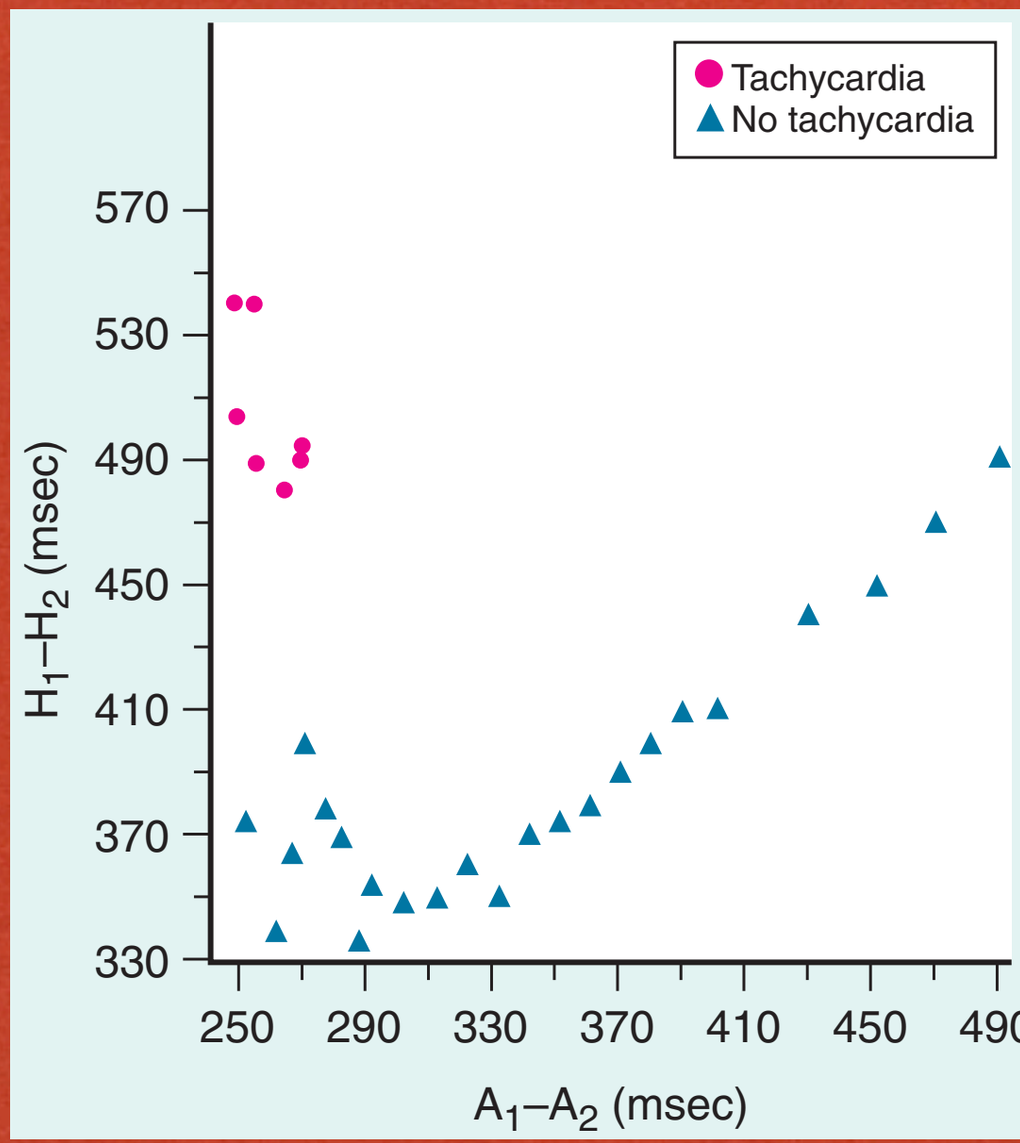
AVNRT

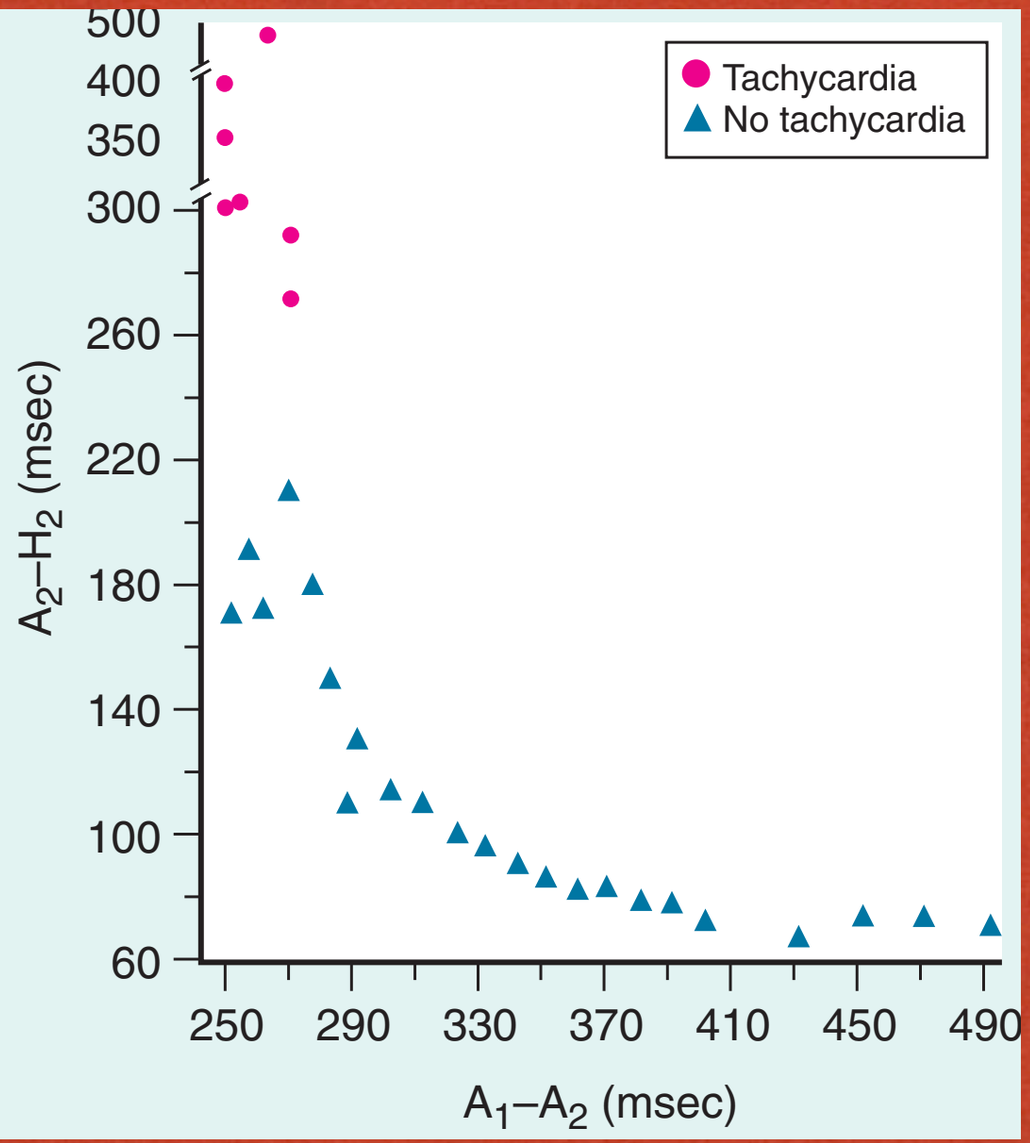
retrograde atrial activity occurs before ventricular septal depolarization

DUAL AV NODE PHYSIOLOGY

- RF catheter ablation of the slow pathway eliminates AVNRT
- Discontinous curve on a plot of A1-A2 intervals versus A2-H2 and H1-H2 intervals respectively: At a crucial A1-A2 interval the impulse is blocked in the fast pathway and conducts down the slow pathway with sudden prolongation of the A2-H2 (or H1-H2) interval
- usual increase in A-H interval is atleast 50 ms for a 10 ms decrease in the coupling interval of the PAC
- 2 QRS complexes in response to one P wave

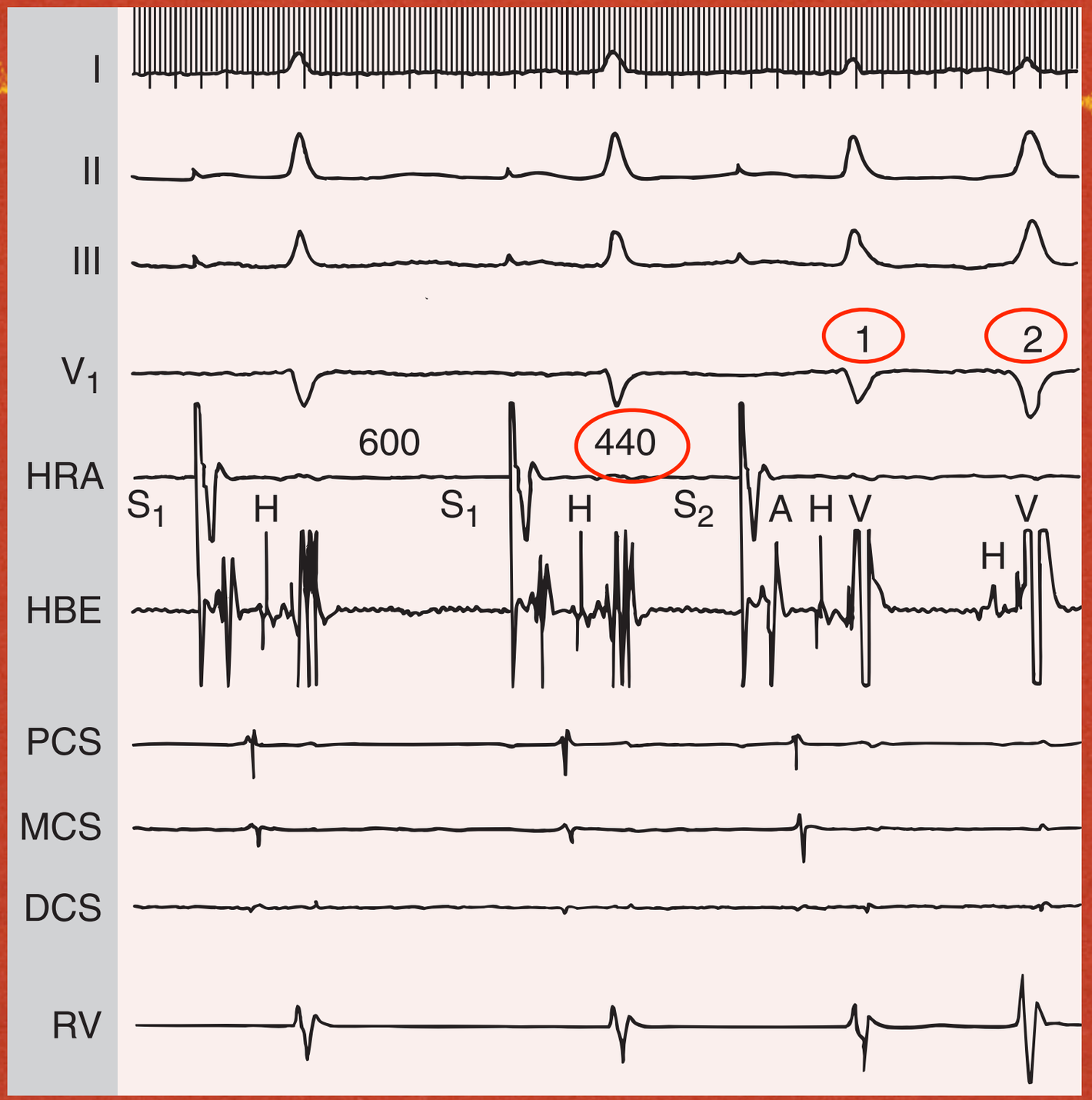
DISCONTINOUS REFRACTORY PERIOD CURVES





DUAL PATHWAY EVIDENCE

2 QRS complexes in response to a single PAC



AH1 95 ms

AH2 430ms

AVNRT

- no structural heart disease is common
- presents in 3rd and 4th decade in adults
- palpitations, nervousness, anxiety, angina, HF, syncope, shock
- syncope: cerebral hyoperfusion **or** asystole when tachycardia terminates due to depression of sinus node automaticity by prolonged tachycardia
- prognosis is good if no heart disease

AVNRT ACUTE RX

- rest, reassurance, sedation

- vagal maneuvers: carotid sinus massage, valsalva maneuver, muller's maneuver, gagging or exposure of face to ice water

- adenosine is the initial drug of choice (depress anterograde conduction in the slow pathway), 6-12 mg iv will successfully terminate in 90% of cases

- Dig, CCB and BB: Verapamil 5-10 mg iv or Diltiazem 0.25-0.39 mg/kg iv terminates AVNRT successfully in 90% cases when vagal/adenosine fails

- DC shock (10 to 50J) or Pacing

- Class IA, IC and III not usually used (cardioversion should be attempted before), may be administered to prevent recurrence; IA and IC drugs decrease conduction in the retrograde fast pathway

AVNRT RECURRENCE PREVENTION

- long acting CCB, BB and digoxin are reasonable initial choices
- RF ablation: >95% effective in curing patients long term, considered early in management of symptomatic patients

ACCESSORY PATHWAYS

ACCESSORY AV PATHWAYS

- Fibers that connect the atrium or AV node to the ventricle, outside the normal AV nodal-his-purkinje conduction system
- potential substrates for reentrant tachycardias (AV reciprocating tachycardia or AVRT)
- **Preexcitation**: conduction antegrade over the accessory pathway resulting in a delta wave in the QRS complex
- **WPW syndrome**: prexcitation + symptoms compatible with the tachycardia
- **Concealed**: pathways conduct in the retrograde direction only, no prexcitation seen

AVRT

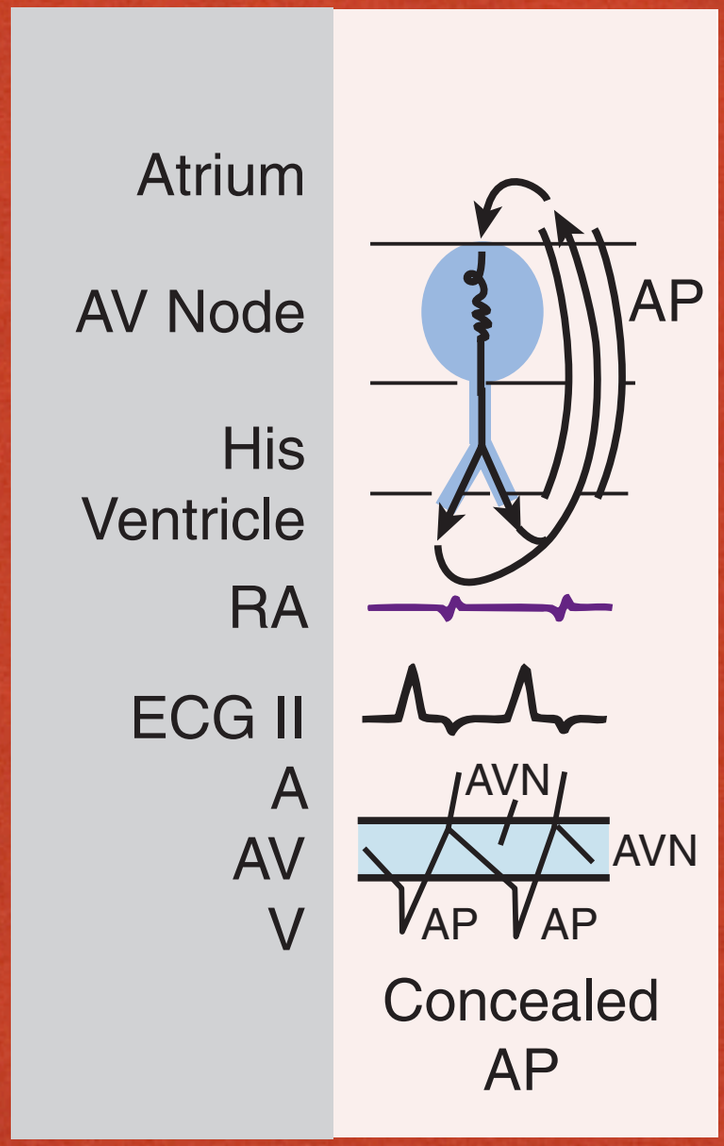
- macroreentry
- anterograde conduction over the AV node-his bundle pathway and retrograde conduction over the AP is most common
- QRS complex is normal, the retrograde p wave occurs after completion of the QRS complex, in the ST segment or early in the t wave
- sometimes the p wave is not clearly visible and can result in depression of the ST segment (resolves with tachycardia termination)

AVRT

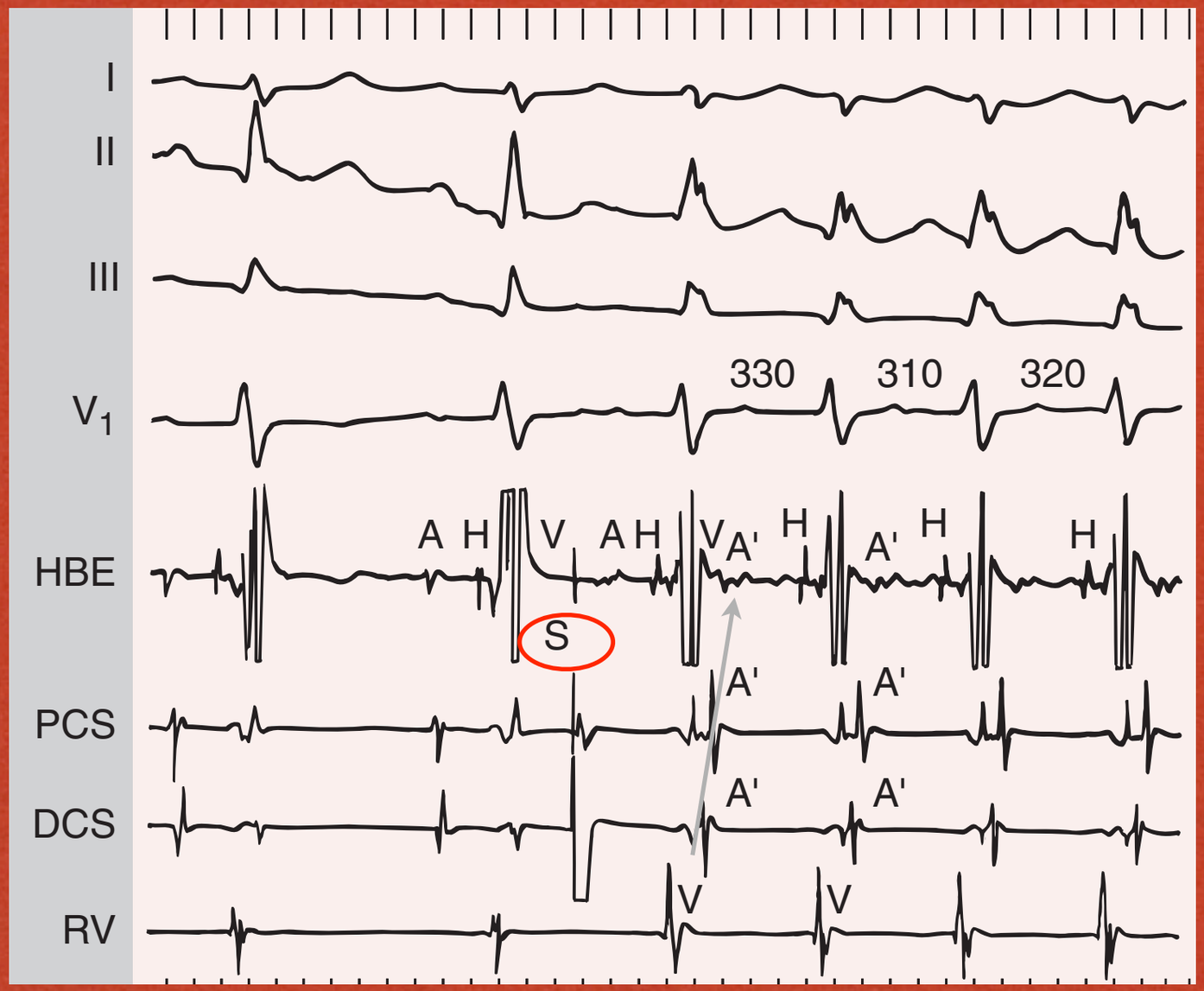
- P wave follows the QRS complex during tachycardia
- the retrograde P wave must occur after ventricular excitation, in contrast to AV nodal reentry, in which the atria are usually excited during ventricular activation
- the contour of the retrograde P wave can differ from that of the usual retrograde P wave because the atria may be activated eccentrically, this occurs because the concealed AP in most cases is left-sided

CONCEALED ACCESSORY PATHWAYS

CONCEALED ACCESSORY PATHWAY



CONCEALED ACCESSORY PATHWAY



A premature stimulus in the coronary sinus (S) precipitates SVT

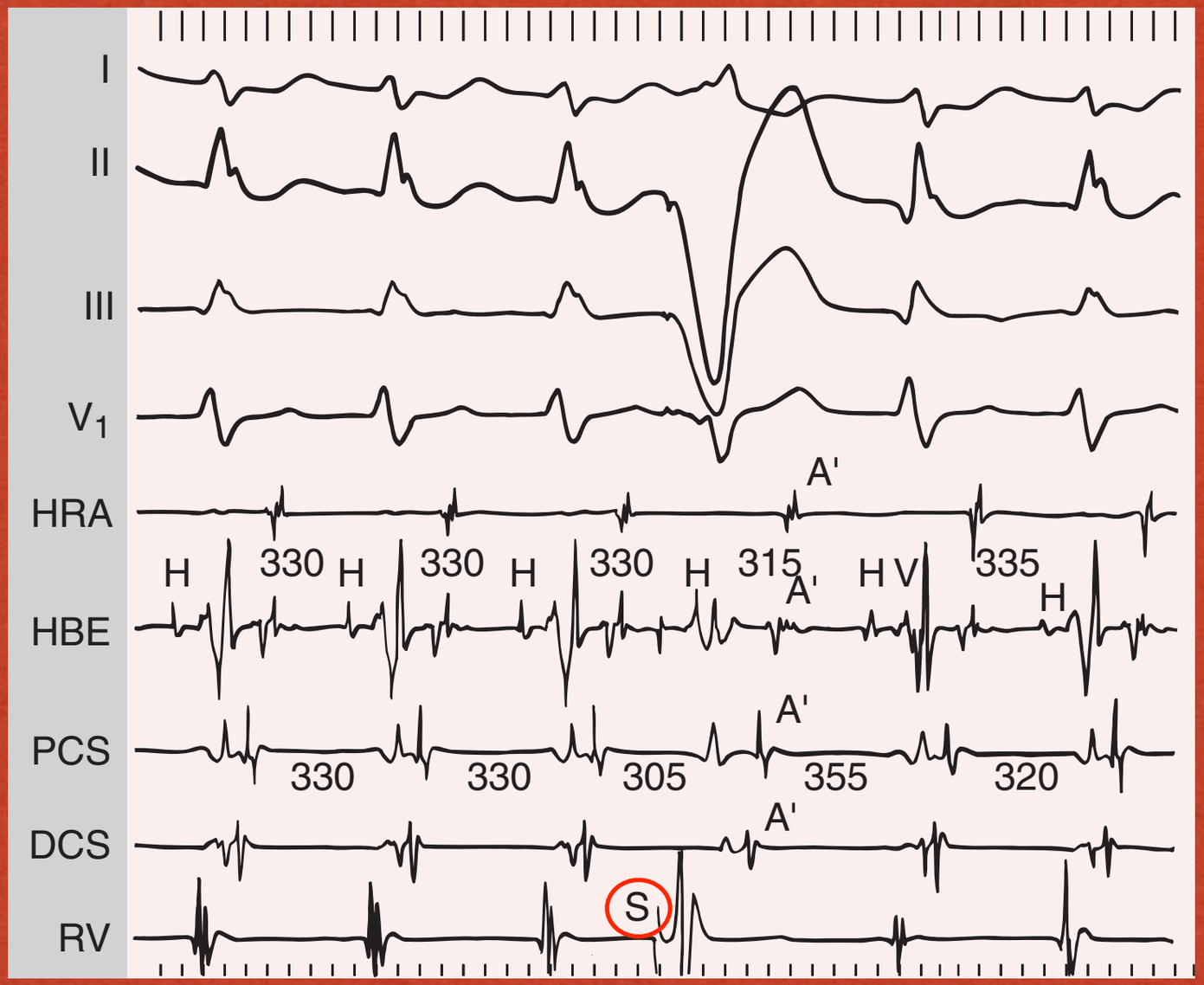
retrograde atrial activation sequence begins first in the DCS (A′), followed by activation in the PCS and low RA (HBE)

QRS complex is normal and identical to the sinus-initiated QRS complex.

the RP interval is short and the PR interval is long.

The shortest VA interval exceeds 65 ms, consistent with conduction over a retrogradely conducting AV pathway.

CONCEALED ACCESSORY PATHWAY



PVC when the His bundle is still refractory from anterograde activation

shortens the A-A interval from 330 to 305 milliseconds without a change in the retrograde atrial activation sequence.

SEPTAL AP

- concealed septal AP is an exception to the rule
- retrograde atrial activation is normal
- VA interval and cycle length of the tachycardia increases 25 ms or less with development of an ipsilateral functional BBB
- vagal maneuvers have a response similar to AVNRT

EP FEATURES OF A CONCEALED PATHWAY

- initiation of tachycardia depends on a critical degree of AV delay, either the AV node or his-purkinje system (a critical degree of A-H delay as in AVNRT is not necessary)

- the AV nodal refractory period curve is smooth (not discontinous as in AVNRT)

DIAGNOSIS OF AP

- premature ventricular stimulation activates the atria before retrograde depolarization of the His bundle
- if the ventricle is stimulated prematurely during the tachycardia at a time when the His bundle is refractory, the impulse still conducts to the atrium
- the VA interval is generally constant over a wide range of ventricular paced rates and coupling intervals of PVCs as well as during the tachycardia
- the VA interval is usually less than 50% of the R-R interval
- the tachycardia can be easily initiated by a PVC that conducts retrograde over the AP but blocks conduction in the AV node or His bundle

CLINICAL FEATURES OF AP

- accounts for about 30% of the patients wit apparent SVT referred for EP evaluation
- tachycardia rates tend to be bit faster (200 bpm)
- palpitations, syncope
- regular ventricular rhythm, S1 inensity is constant

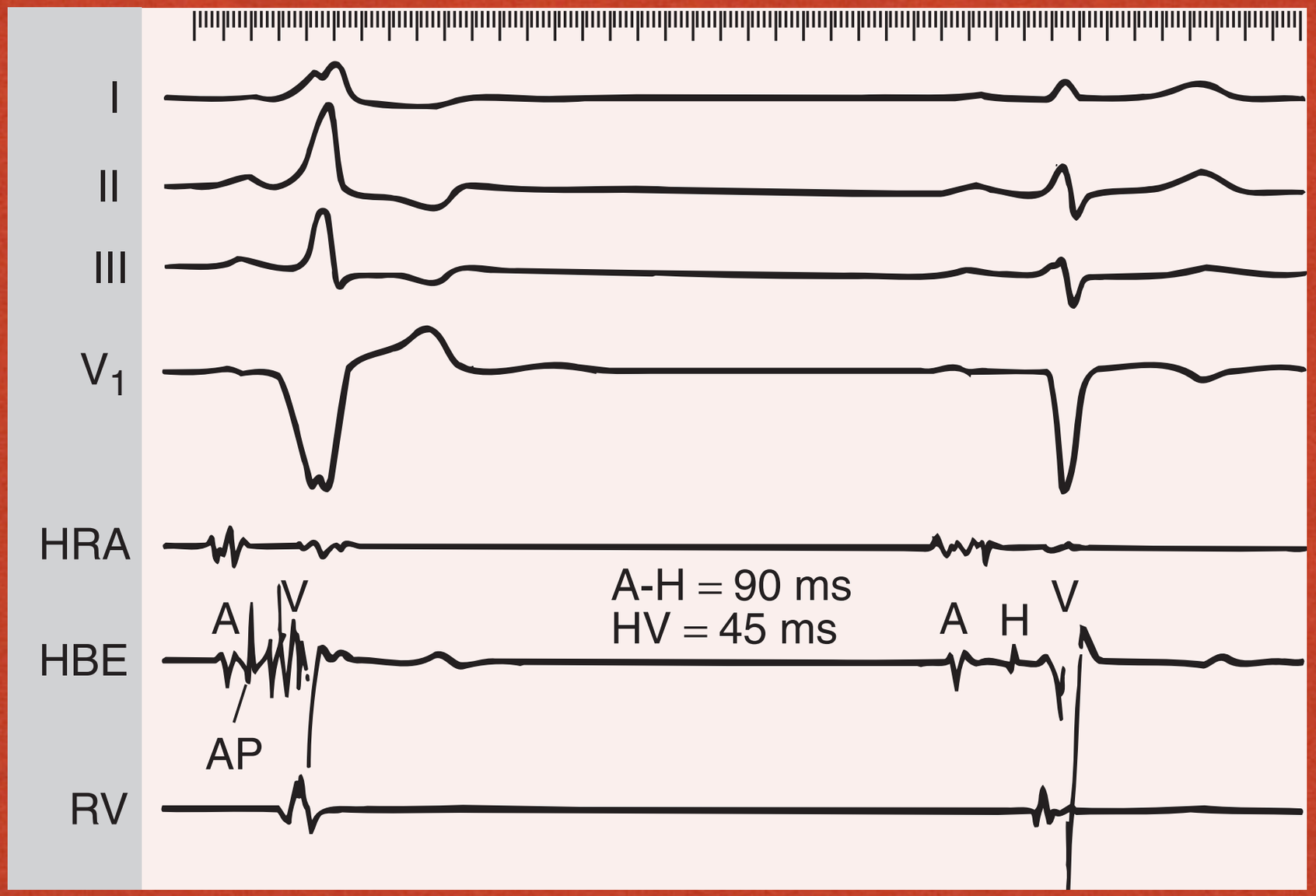
MANAGEMENT

- vagal, iv adenosine, verapamil, diltizaem, dig and BB
- RF catheter ablation: curative, low risk, should be considered early
- AAD can be considered

PREXCITATION SYNDROME

DEPOLARIZATION OF AN ACCESSORY PATHWAY

delta wave



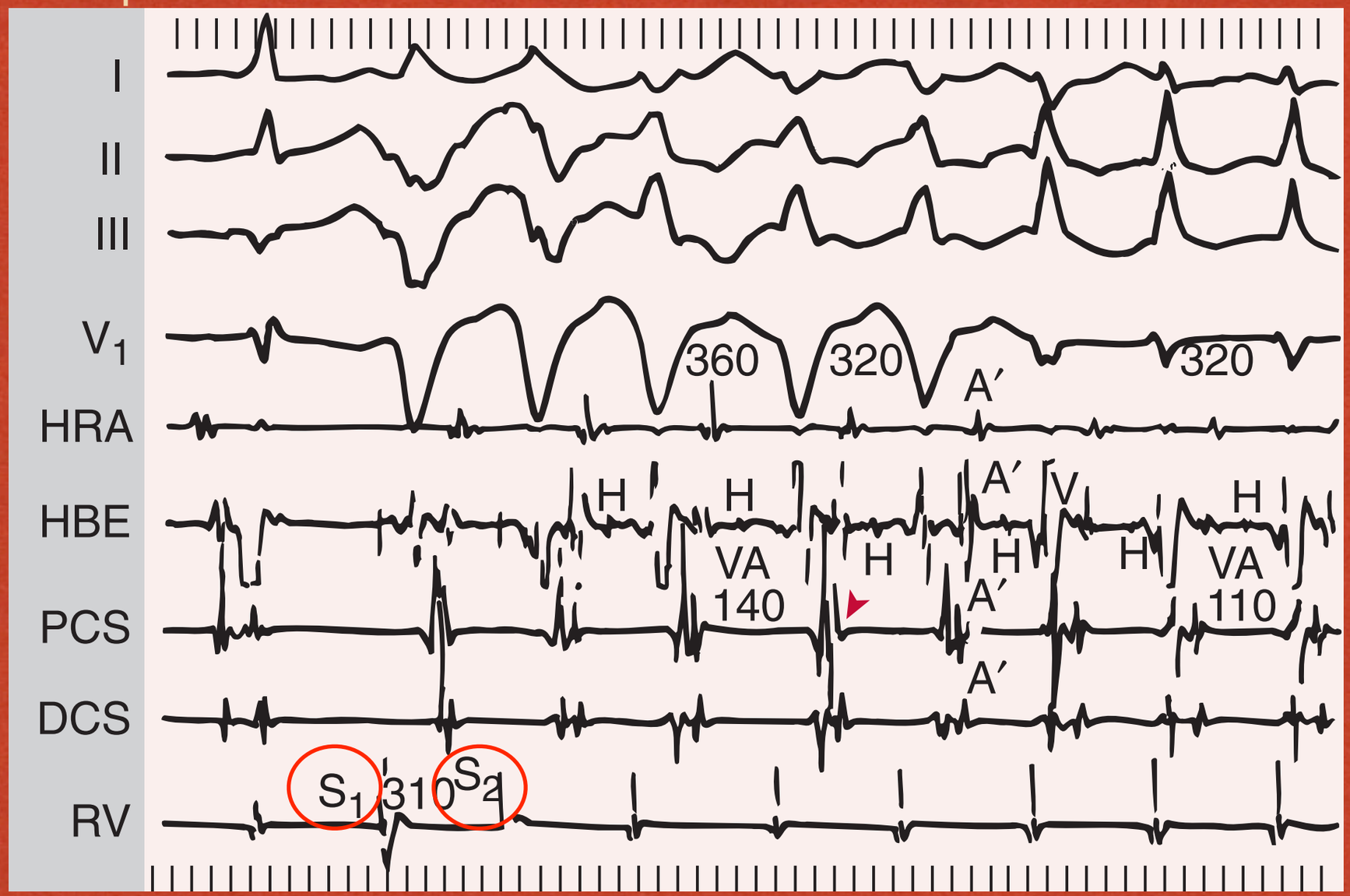
conduction over the AP

conduction over the AP is blocked

Influence of functional ipsilateral bundle branch block on the VA interval during an AVRT

SVT with a LBBB that then reverts to normal

partial preexcitation



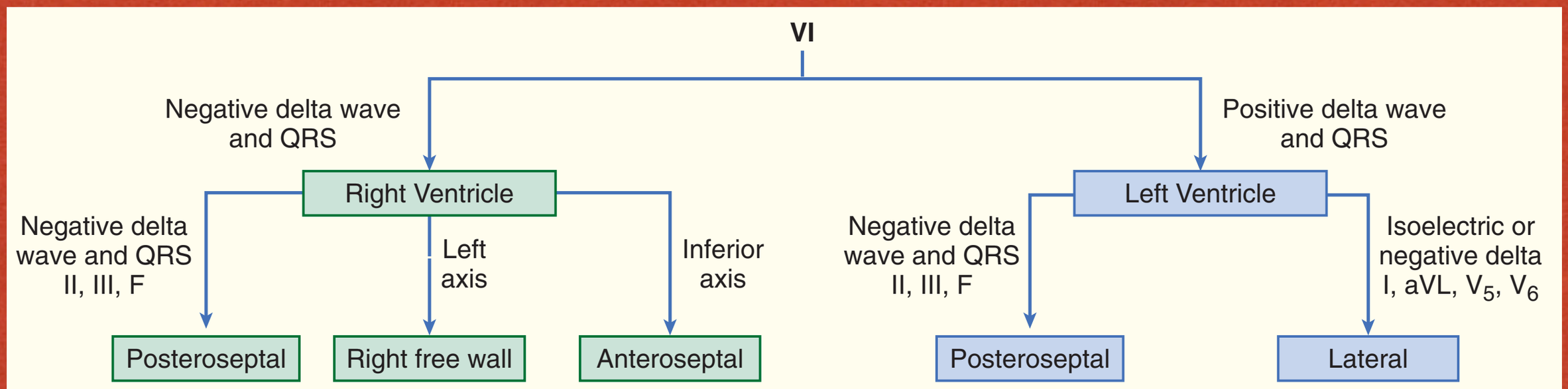
2 premature ventricular stimuli initiates a SVT

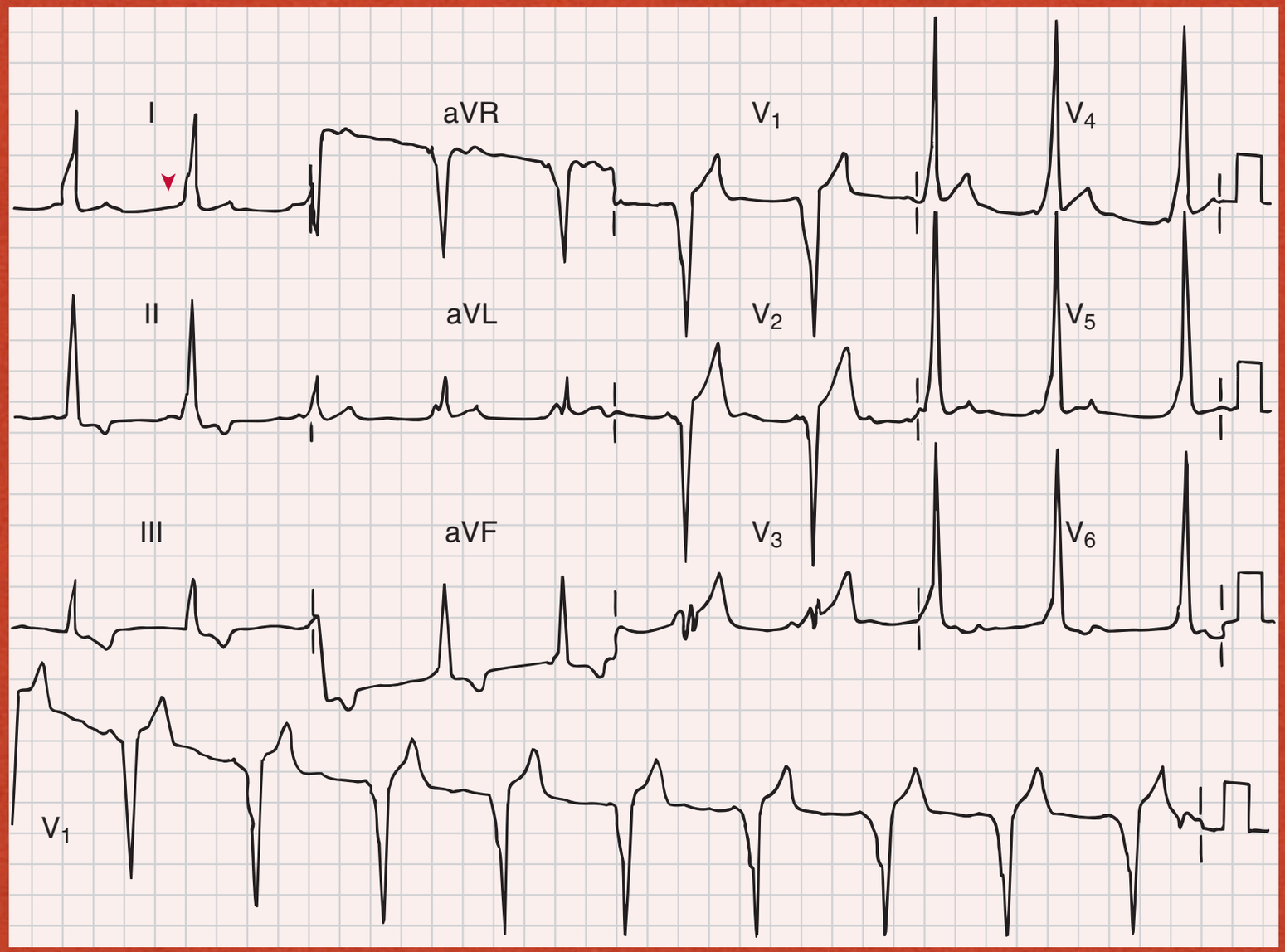
VA interval shortens from 140 ms to 100 ms

PREEXCITATION

- the atrial impulse activates the entire ventricle or some part of it or the ventricular impulse activates the entire atrium or some part of it earlier than would be expected if the impulse traveled by way of the normal specialized conduction system
- via accessory AV pathways
- when tachyarrhythmias occur as a result of the AP it is called pre-excitation syndrome

LOCALIZING AP




I
aVR
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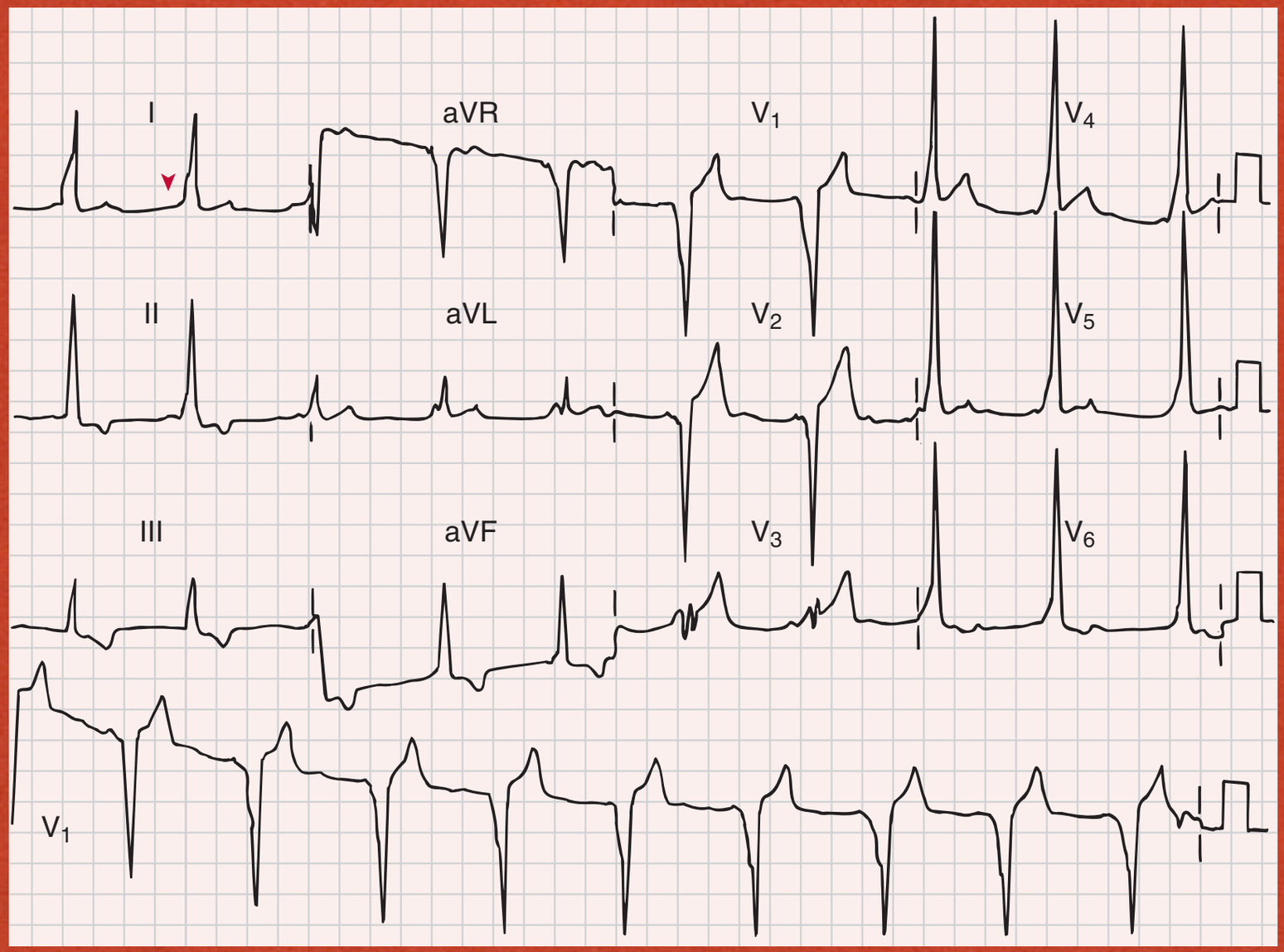
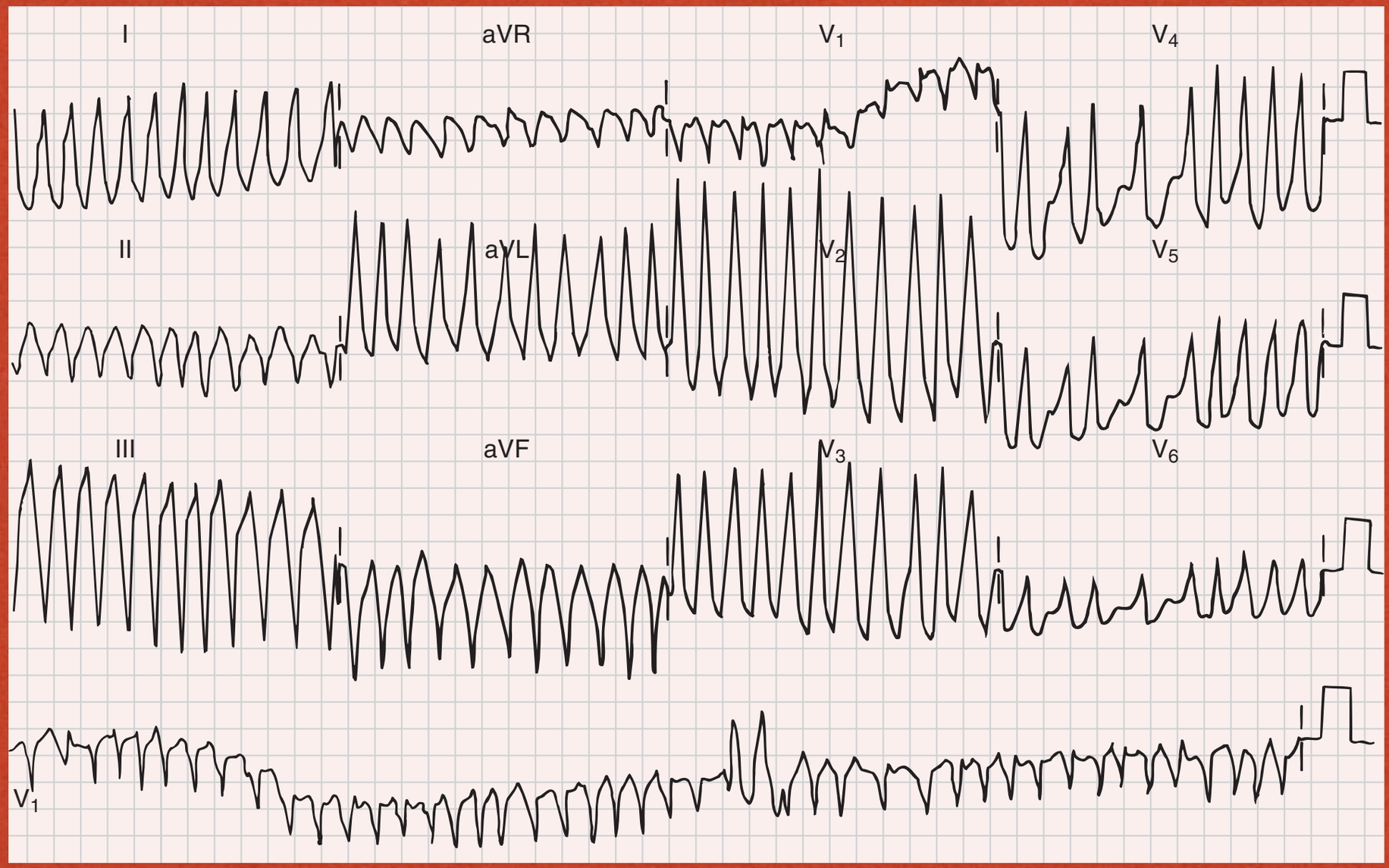
II
aVL
V2
V5
III
aVF
V3
V6
V1

RIGHT ANTEROSEPTAL AP

normal or inferior axis



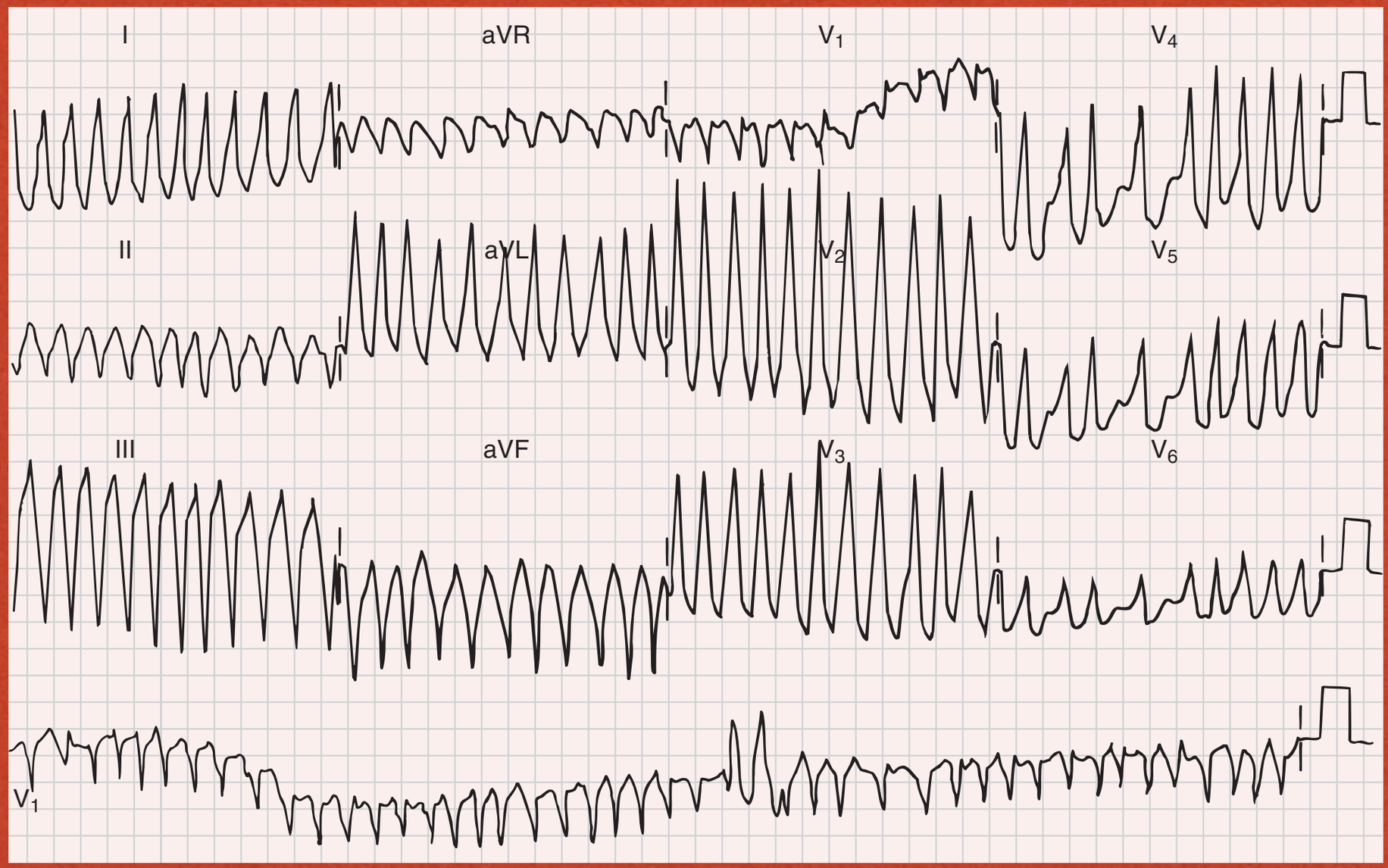
negative delta wave and QRS in VI


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

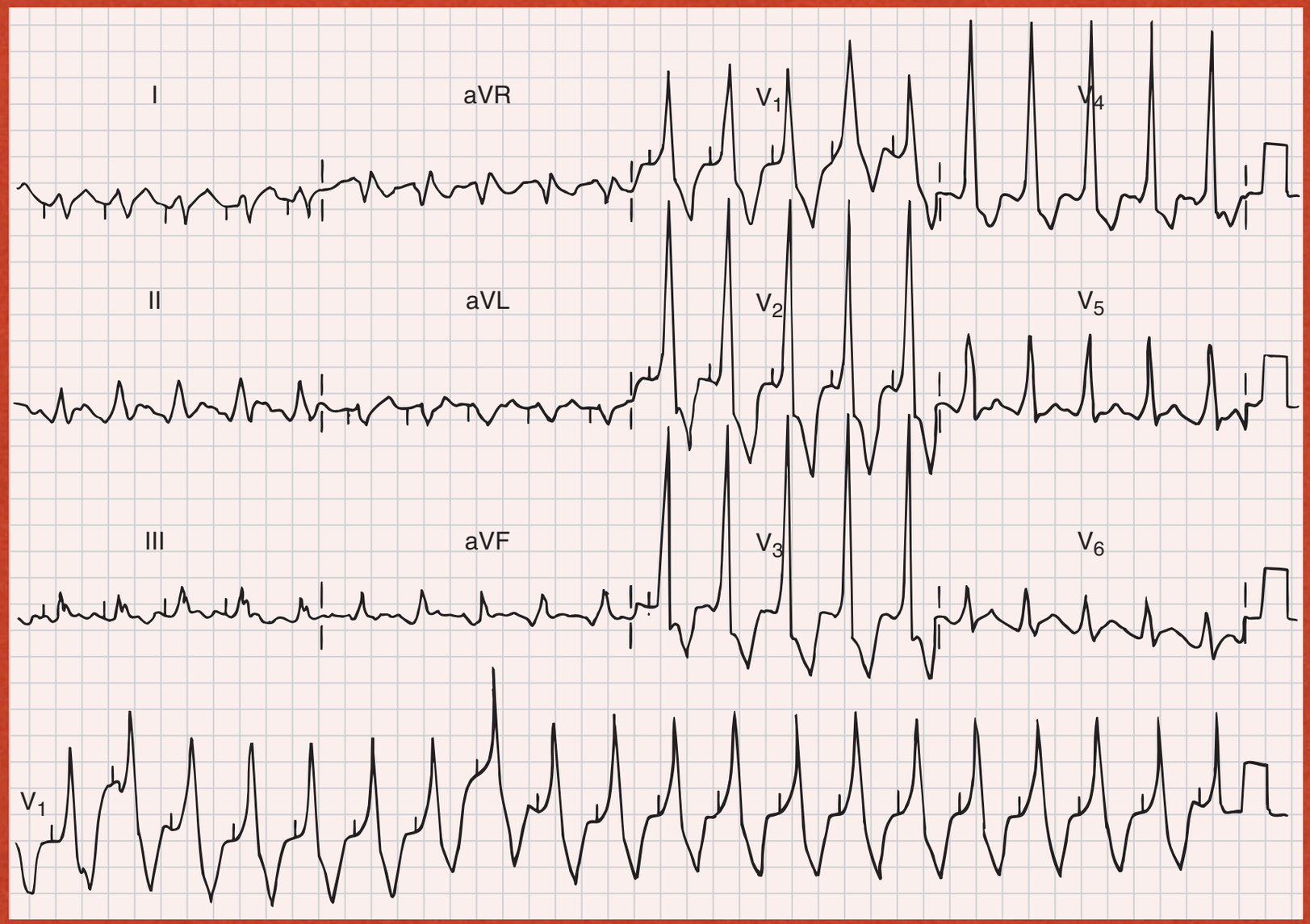
RIGHT POSTEROSEPTAL

positive delta waves in I, aVL

negative delta waves in the inferior leads

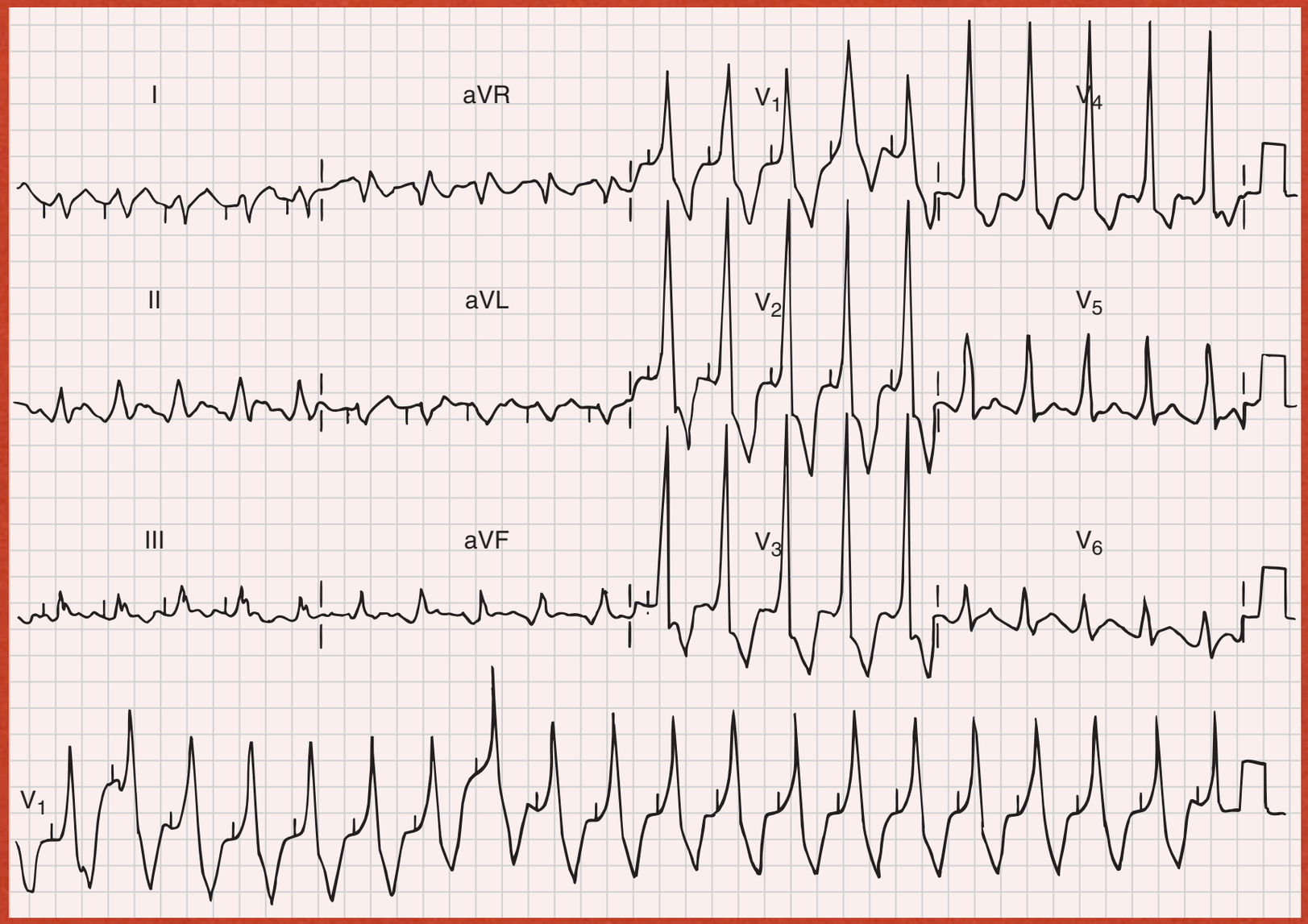


negative delta wave and QRS in VI


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

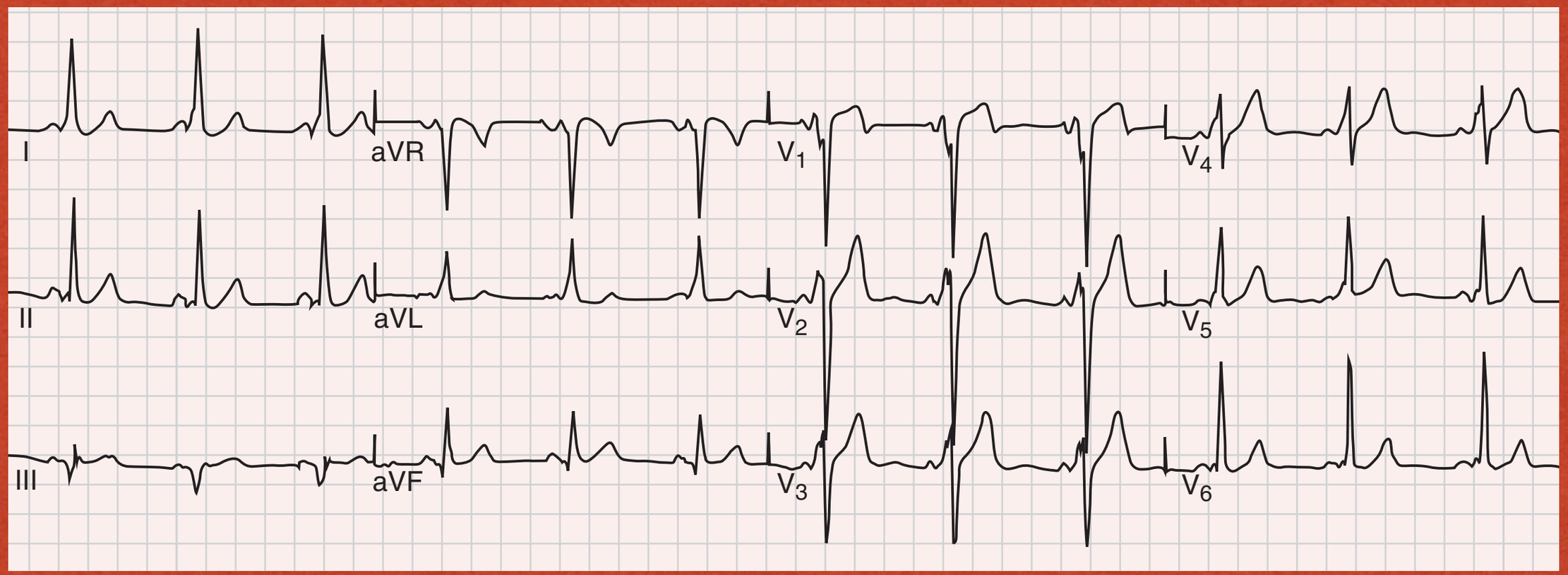
LEFT LATERAL AP

positive delta wave in the inferior leads



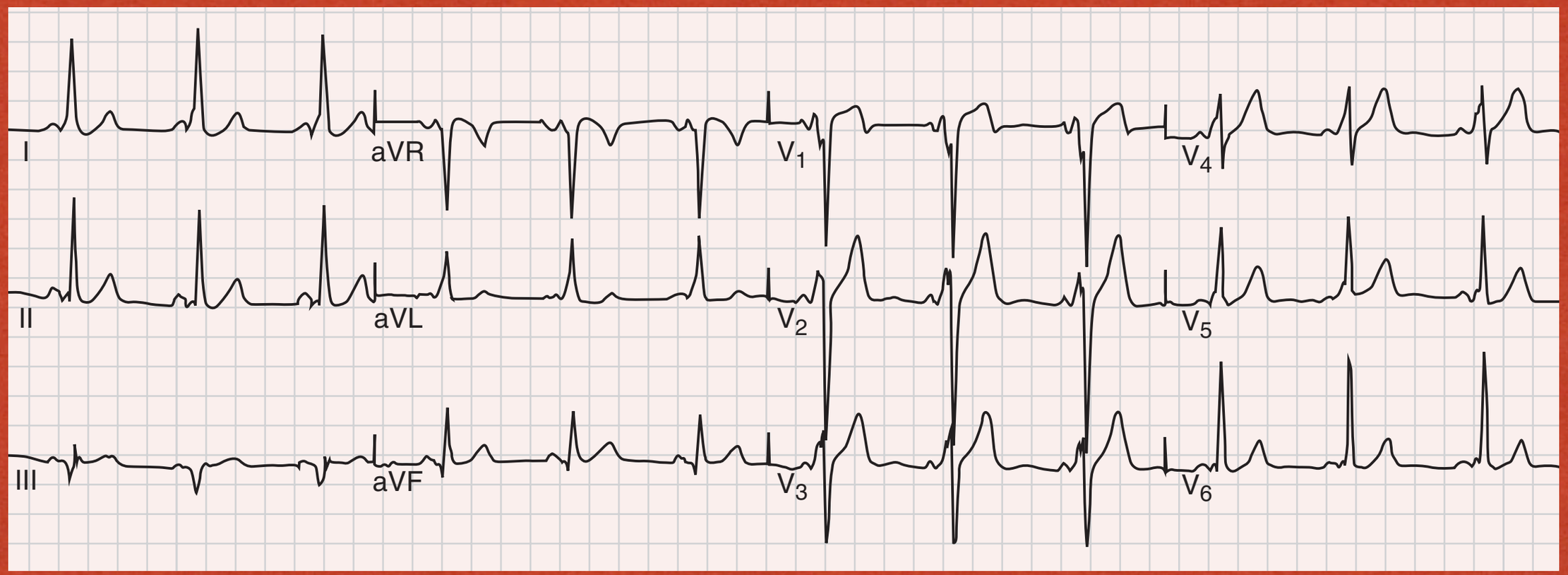
positive delta wave in the anterior precordial leads

rapid coronary sinus pacing to induce preexcitation


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

RIGHT FREE WALL AP

negative delta wave and QRS in V1



more leftward axis compared
to a right anterospetal AP

WPW SYNDROME

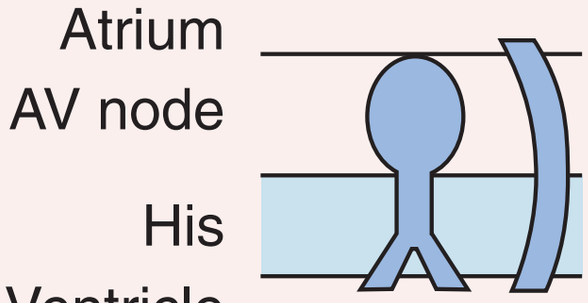
- tachycardia characterized by a normal QRS, a regular rhythm, VR of 150 to 250 bpm, sudden onset and termination

- capacity for anterograde conduction over the accessory pathway during afib or flutter

VARIANTS

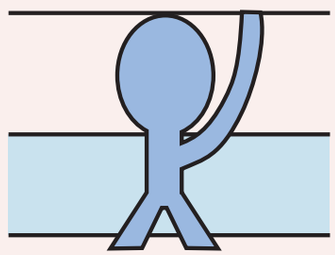
WPW syndrome

A



Atrioventricular

B

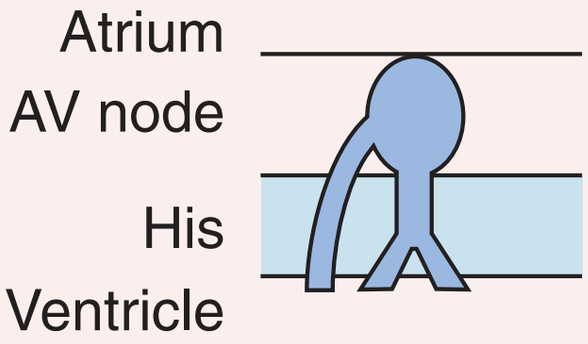


Atriohisian

Lown-Ganong-Levine syndrome

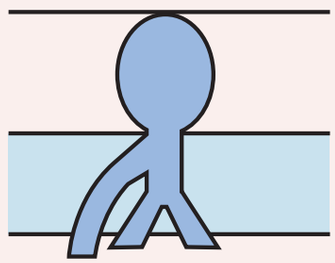
short PR
normal QRS

C



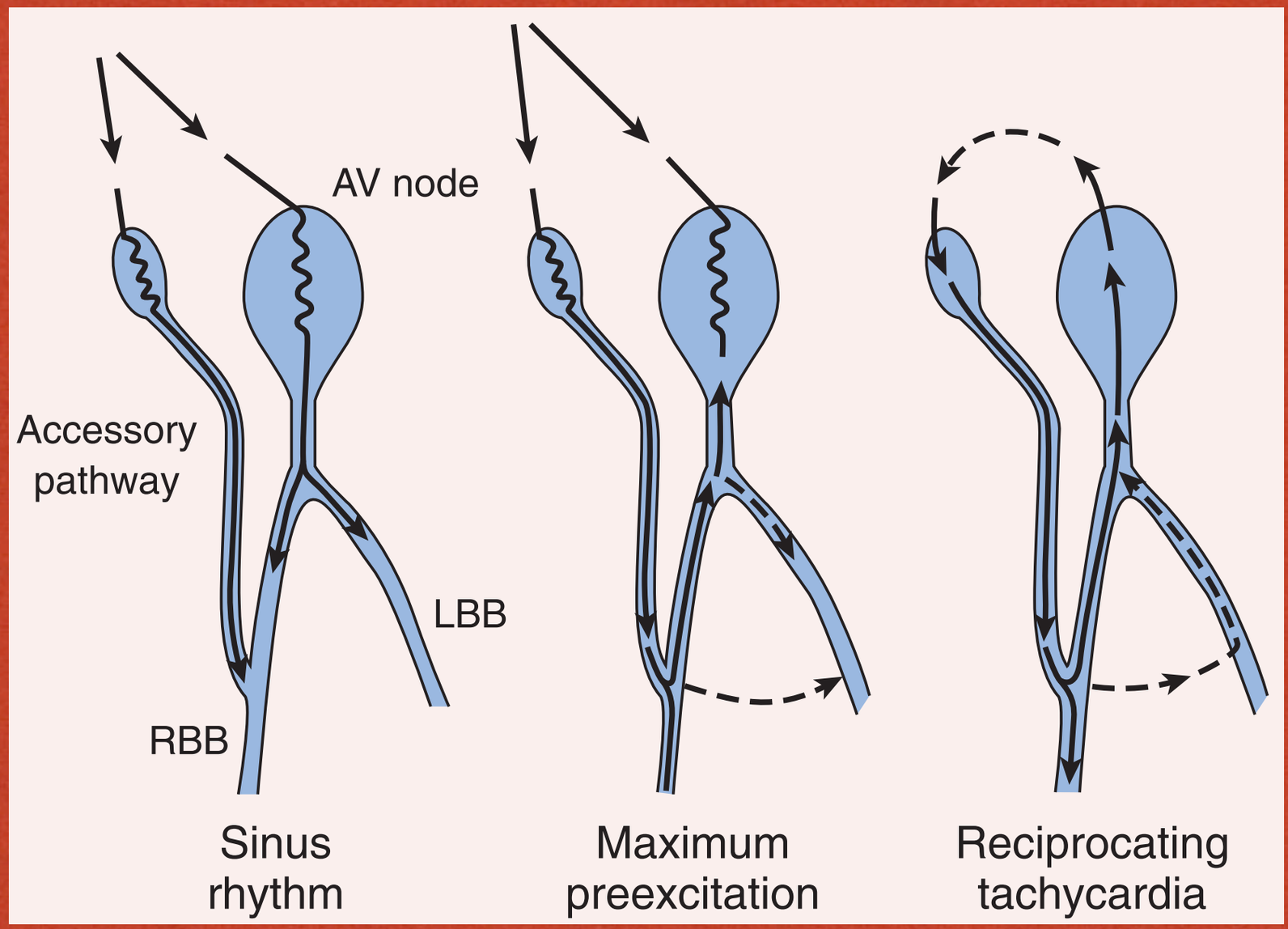
Nodoventricular

D



Fasciculoventricular

NODOFASCICULAR AP



NODOFASCICULAR OR NODOVENTRICULAR AP

- **Mahaim conduction**: Ventricular preexcitation (widened QRS and short H-V interval) with a progressive increase in the AV interval in response to atrial overdrive pacing
- because the AP responsible for this conduction pattern usually inserts in the RBB, preexcitation generally results in a LBBB pattern
- phenomenon caused by fibers passing from the AVN to the ventricle (nodoventricular) or AVN to RBB (nodofascicular)
- Nodoventricular fibers cause a normal or short PR interval and QRS complex is a fusion beat
- These fibers almost always represent dupliction of the AVN; the distal conducting system is located in the RV free wall

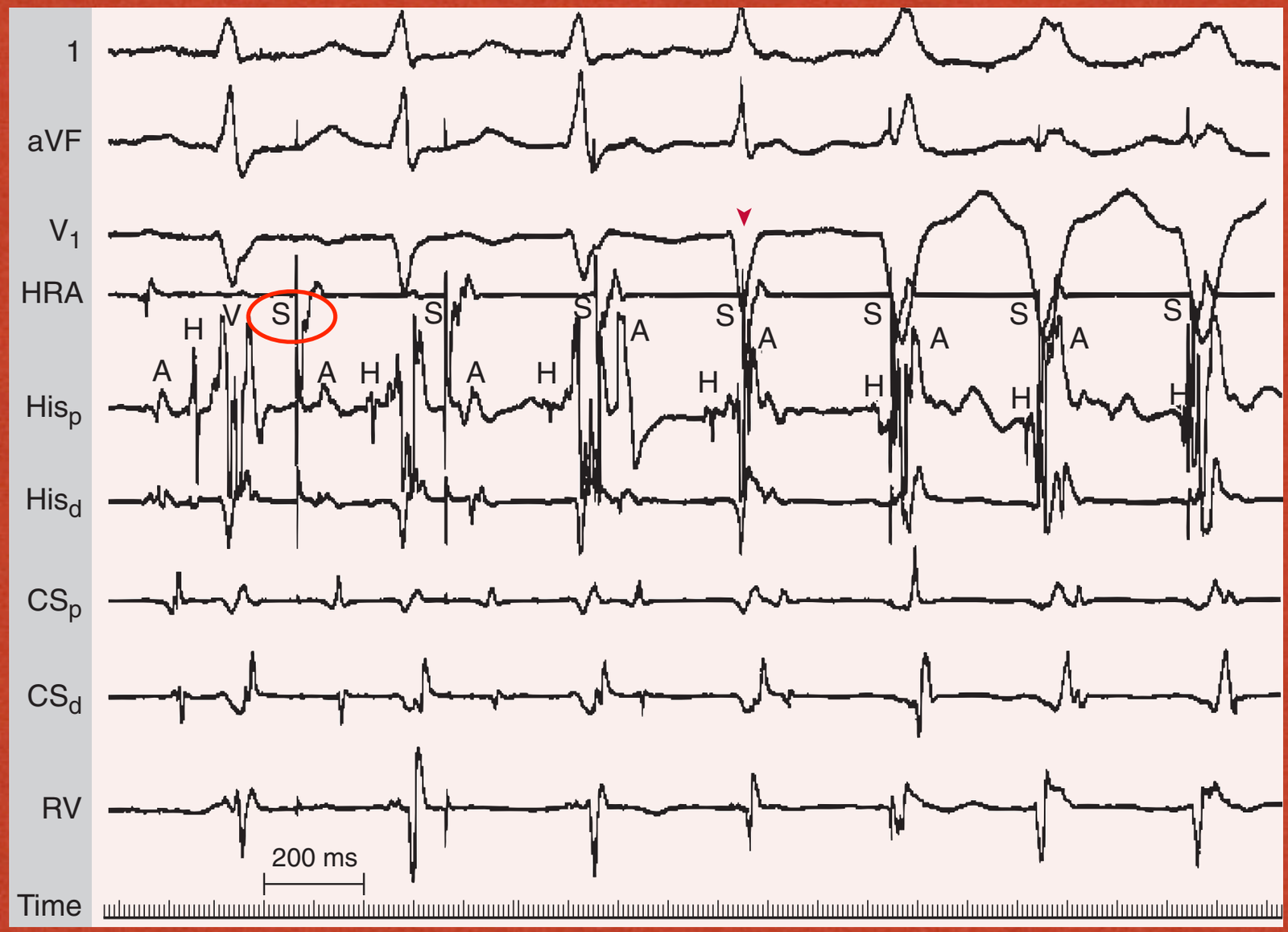
NODOFASCICULAR OR NODOVENTRICULAR AP

- the apical end lies close to the tricuspid annulus and conducts slowly with AVN like properties
- After a long course the distal portion of these fibers, which conducts rapidly inserts into the distal RBB or the apical RV
- no preexcitation is generally apparent during sinus rhythm, but it can be exposed by premature atrial stimulation
- retrograde conduction is usually absent, produces only an antidromic AVRT (preexcited tachycardia): anterograde over AP and retrograde over the RBB-his bundle-AVN
- LBBB pattern seen for the preexcited tachycardia, long AV interval and short VA interval
- RBBB is proarrhythmic by increasing the length of the tachycardia circuit and can lead to incessant tachycardia

PREEXCITATION OVER AN ATRIOFASCICULAR PATHWAY

atrial pacing (S) results in conduction down the AVN

normal QRS and normal H-V interval



arrowhead conducts down the atriofascicular fiber

preexcited QRS, widened QRS + short H-V interval

VARIANTS

PATHWAY TYPE	PR	QRS	TACHYCARDIA
Atriohisian	Short	Normal	Unlikely
Atriofascicular	Normal	Preexcitation (LBBB, superior axis)	Antidromic AVRT
Nodofascicular	Normal	Preexcitation (LBBB, superior axis)	Antidromic AVRT; AVNRT with bystander activation of AP
Fasciculoventricular	Normal	Anomalous (short H-V)	?

EP FEATURES OF PREEXCITATION

- **delta wave**: ventricular activation from input over the acessorry pathway
- the typical QRS is a fusion beat of the delta wave and conduction down the AVN
- rapid atrial pacing or a PAC causes AV nodal conduction delay, more of the ventricle is activated by the AP, and the QRS becomes more anomalous in contour
- if the AP is relatively far from the sinus node (e.g, left lateral AP) or AVN conduction time is relatively short, normal ventricular activation predominates
- the normal fusion beat during sinus rhythm has a short H-V interval or his bundle activation begins after onset on ventricular depolarization (as part of the ventricle gets to depolarize early)
- finding of a **short or negative H-V interval** occurs only during conduction over an AP or retrograde his activation as in VT

EP FEATURES OF PREEXCITATION

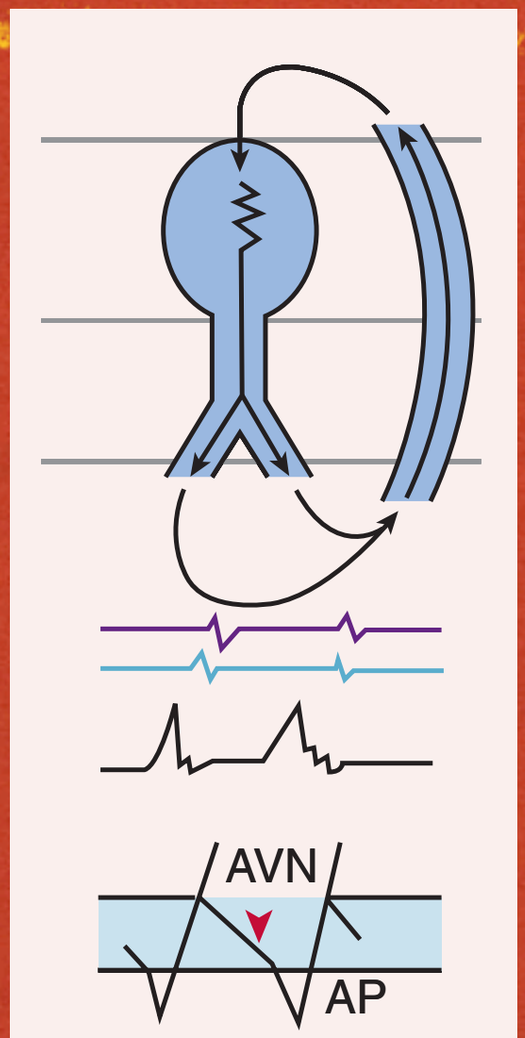
- rapid atrial pacing, at premtaure intervals or close to the atrial insertion of the AP accentuates the anomalous activation of the ventricle and shortens the H-V interval even nore
- 12-lead ecg can help localize the position of the AP further
- after dissapearance of preexcittation, t wave abnormalities can occur (t wave memory)

ORTHODROMIC AVRT

- AP conducts more rapidly than the AVN but has a longer refractory period during sinus rhythm (long cycle length)
- thus an early PAC may block at the AP and conduct normally down the AVN
- orthodromic AVRT: conduction down the AVN and retrograde over the AP

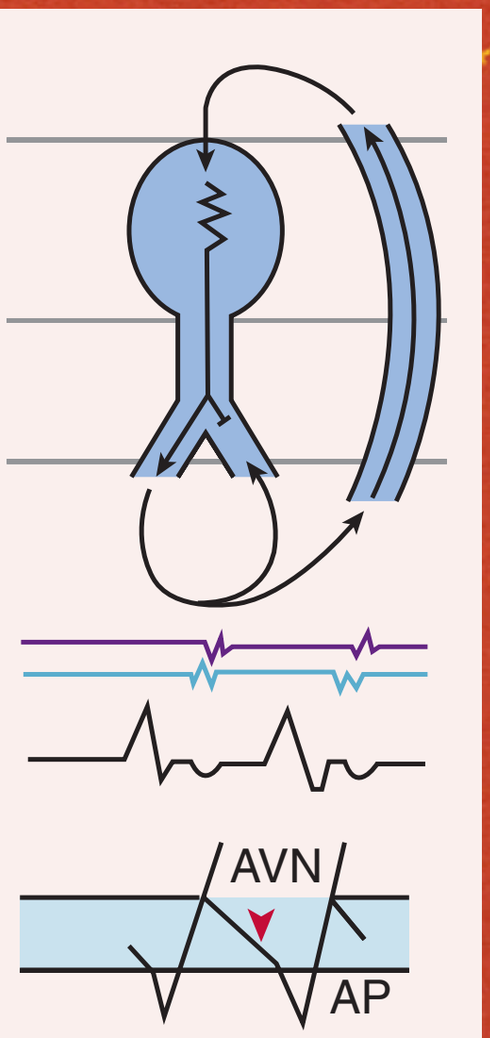
ORTHODROMIC AVRT

Atrium
AV node
His
Ventricle
RA
LA
ECG
II
A
AV
V



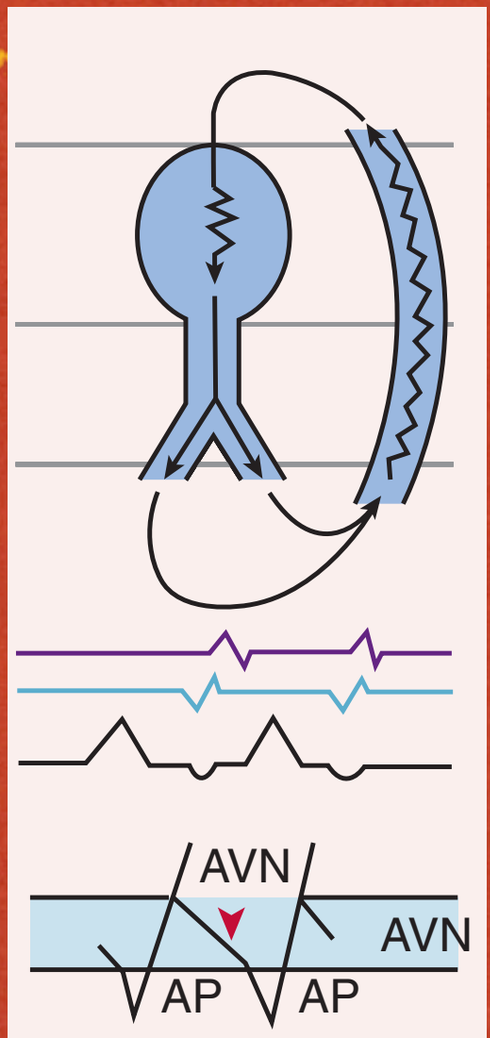
A

Usual orthodromic



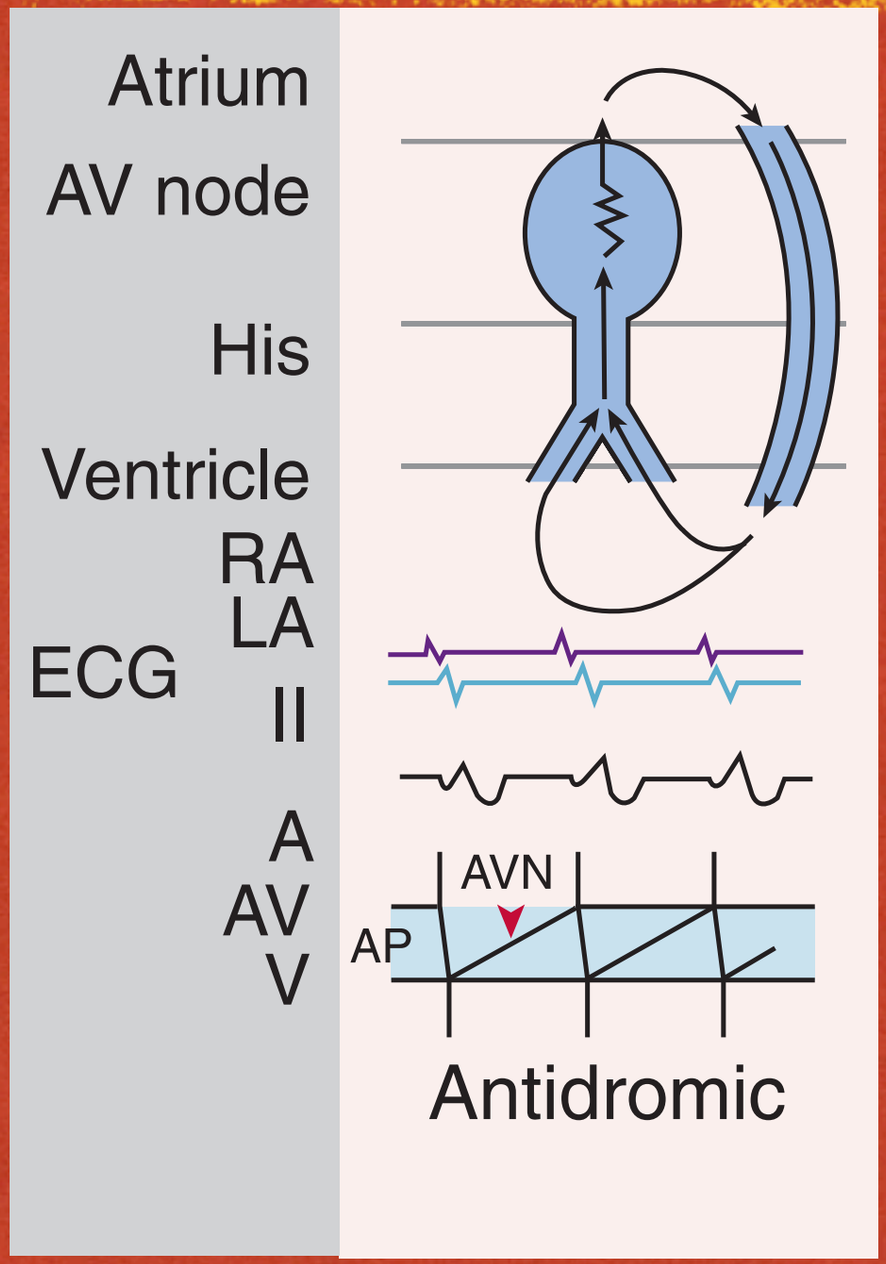
B

Bundle branch block



Slow retrograde

ANTIDROMIC AVRT

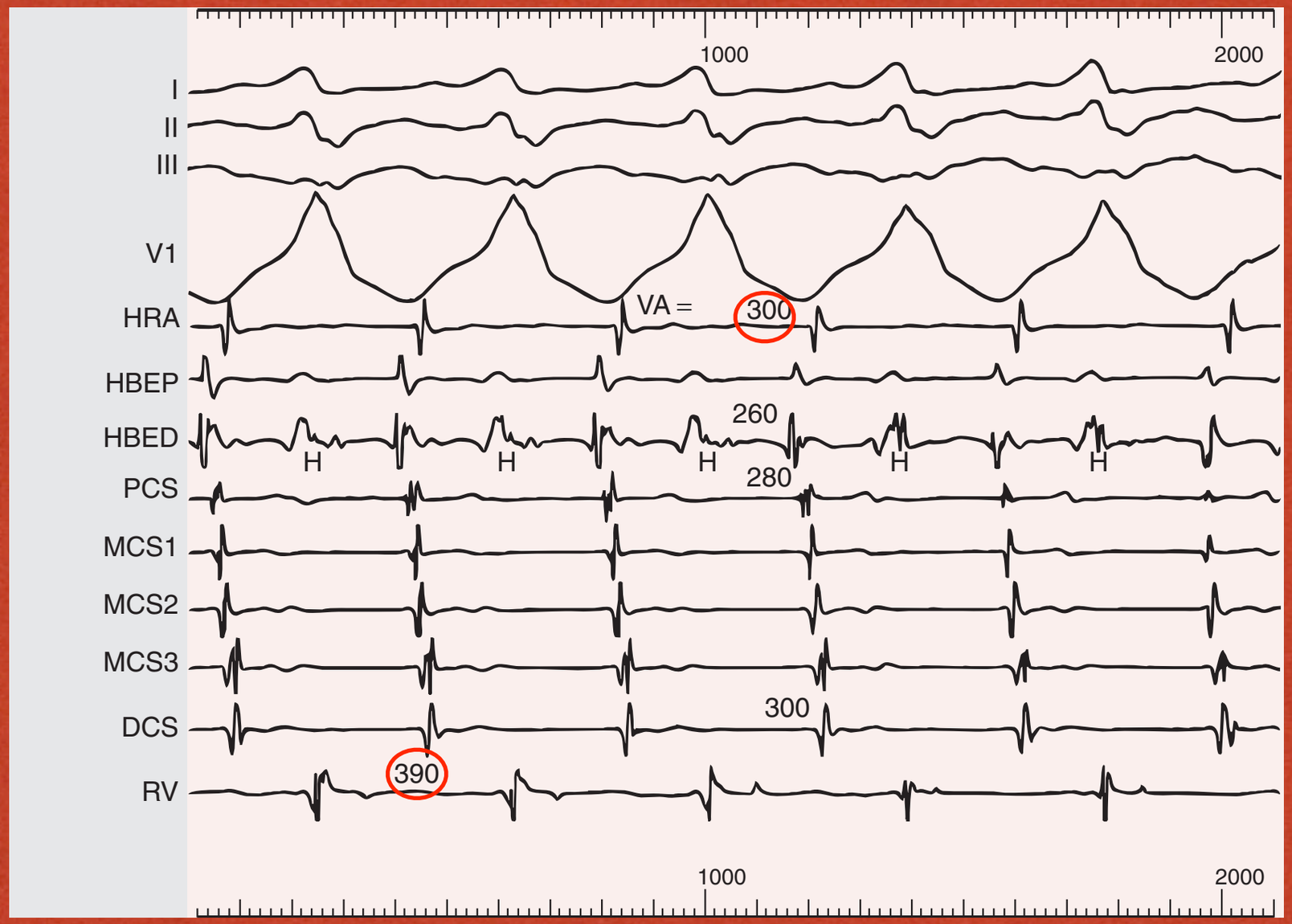


QRS pattern
is abnormal

ANTIDROMIC AVRT

anterogarde conduction over the AP

abnormal QRS



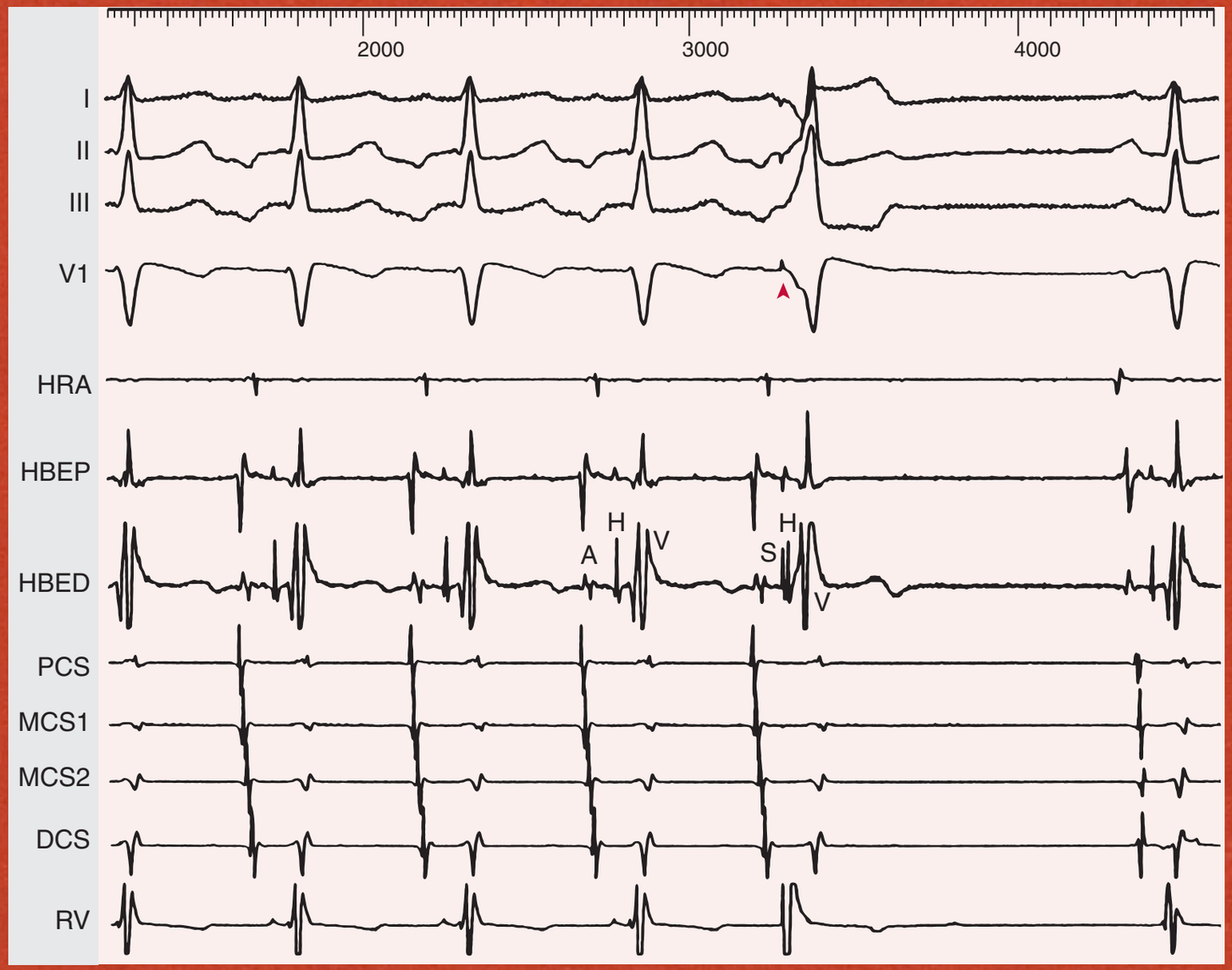
normal retrograde activation sequence

PJRT

- permanent form of AV junctional reciprocating tachycardia
- incessant form of SVT
- long RP tachycardia
- usually a posteroseptal AP (most often RV) that conducts very slowly (long and tortuous route)
- anterograde over AVN and retrograde over the AP

PJRT

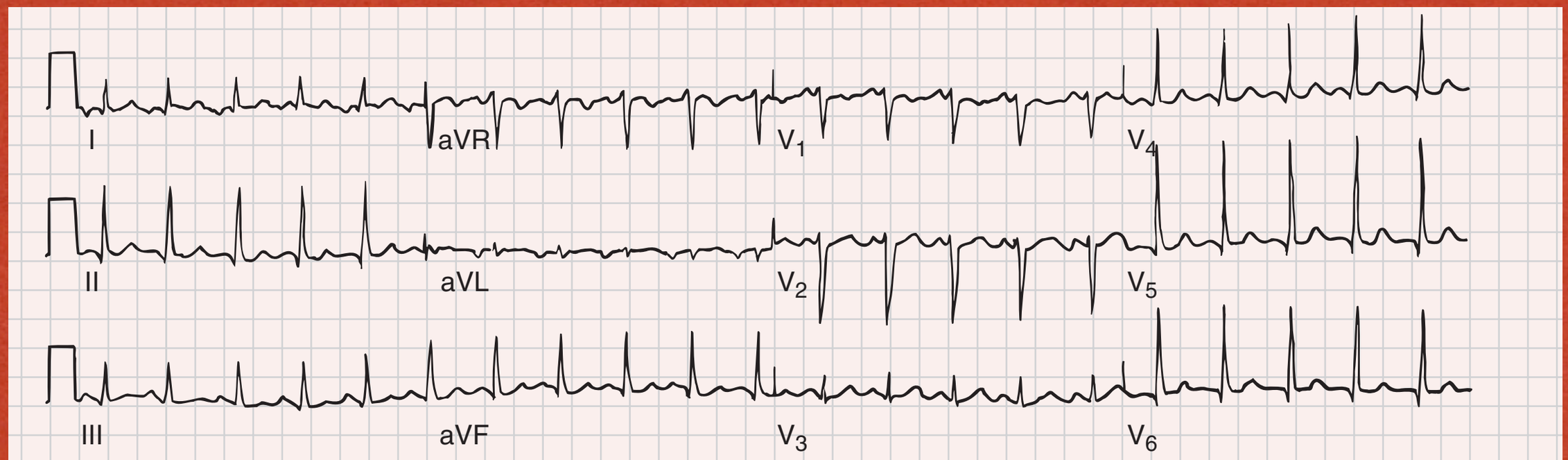
atrial activation sequence is indistinguishable from atypical AVNRT or AT from the low RA

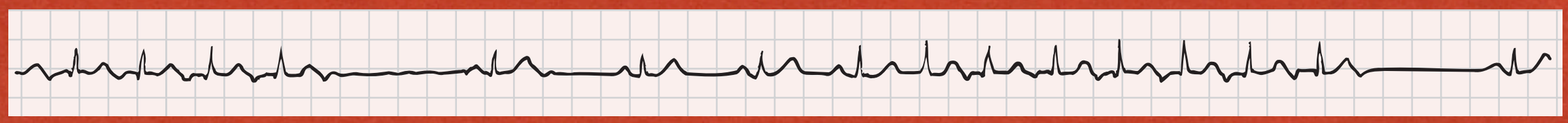


PJRT

PVC does not reach the AVN or RA but terminates the tachycardia by blocking the retrograde AP

PJRT





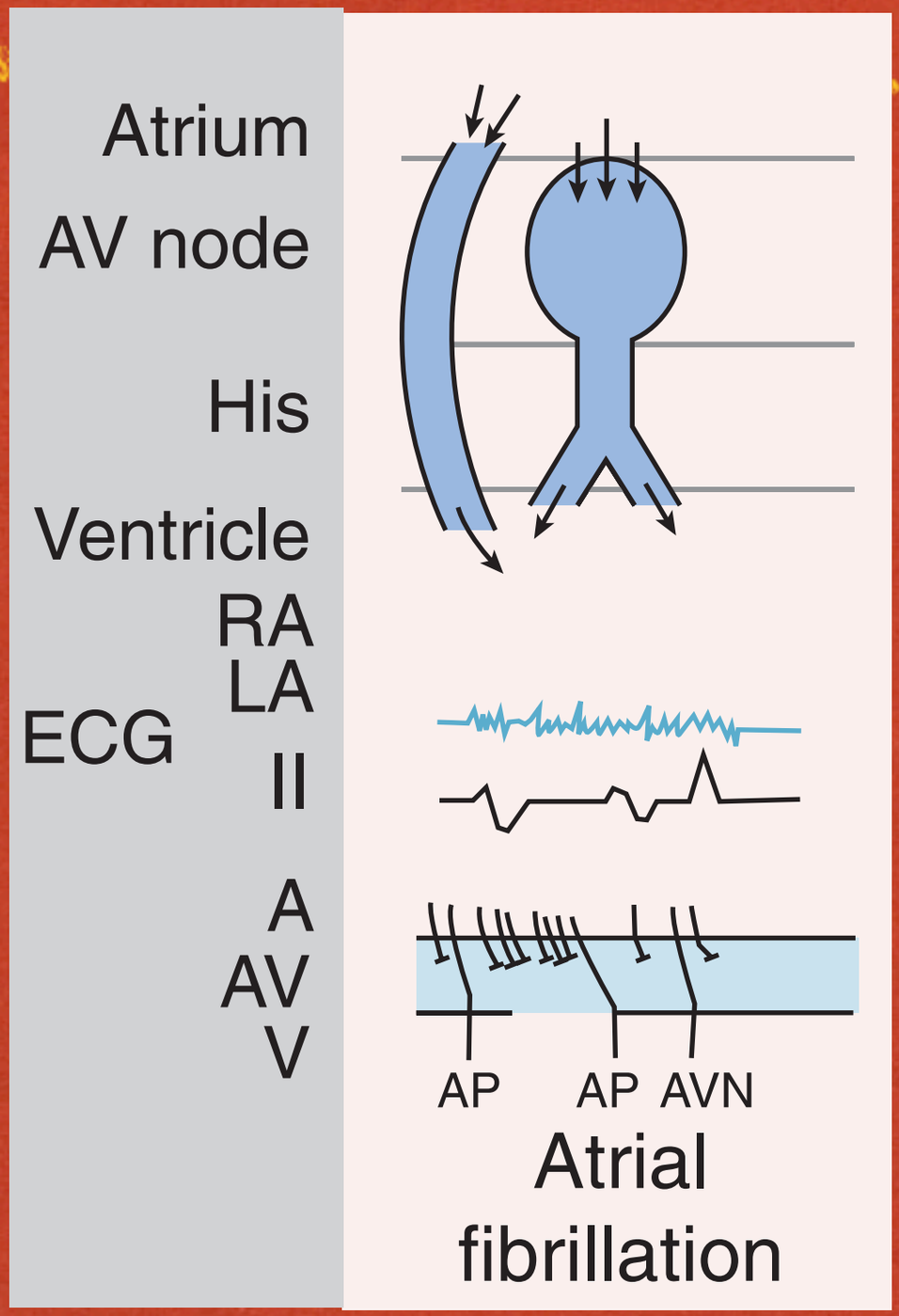
nonconducted p wave terminates the tachycardia unlike atrial tachycardia

RECOGNITION OF AP

- AP mapped on EP study using site of earliest retrograde activation: left AP ~ coronary sinus, right AP ~ lateral RA, septal AP ~ low RA
- inducing PVC's during the tachycardia to see if retrorade atrial activation occurs
- VA interval are > 70 ms for atrial activity recorded on an esophageal lead and > 95 ms when measured in HRA (unlike AVNRT where VA is < 70 ms)

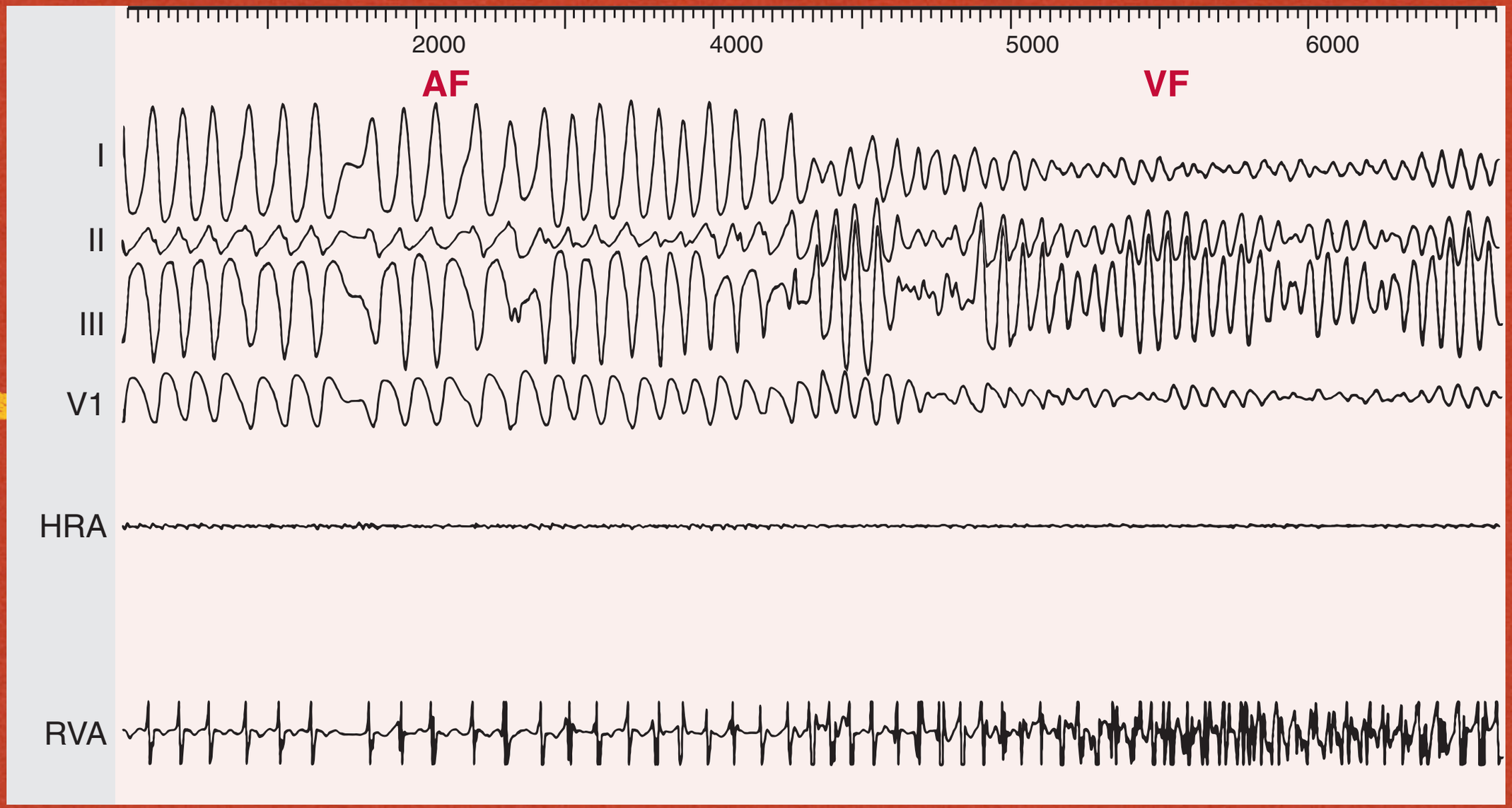
AFIB

AP is a bystander



Atrial fibrillation

AFIB OVER AN AP



"WIDE QRS" TACHYCARDIAS

- sinus or AT, AVNRT or afib/flutter with anterograde conduction over the AP
- orthodromic AVRT with functional or preexisting BBB
- antidromic AVRT
- reciprocating tachycardia with anterograde conduction over one AP and retrograde over a 2nd one
- tachycardias using nodofascicular or atriofascicular fibers
- VT

PREEXCITATION SYNDROME

- 0.1-3 /1000 incidence
- ECG WPW pattern incidence 0.25%
- prevalence of documented tachyarrhythmias 1.8%
- AP: male predominance, frequency decreases with age
- ebstein anomaly have multiple rt sided APs
- paroxysmal tachycardias in wpw syndrome increases with age: 10/100 to 36/100 (from age 20 to 60)
- can mask or mimic BBB, MI and ventricular hypertrophy
- sudden death frequeny is 0.1%, generally good prognosis

RX OF PREECITATION

- drugs that prolong the refractroy period of the AVN and AP, class IC and III prolong AP refractoriness
- lidocaine and iv verapamil can ppt vfib in pts with afib and wpw
- acute: vagal, adenosine, iv verapamil or diltiazem
- flecainide + propranolol: decreases conduction in both limbs of the circuit
- amiodarone and sotalol prolong refractoriness in both pathways
- RF catheter ablation to prevent recurrence

P WAVES IDENTICAL TO SINUS P WAVES

- long RP and short PR
- sinus nodal reentry
- sinus tachycardia
- atrial tachycardia arising near the SA node

RETROGRADE P WAVES

- p waves inverted in II, III and aVF
- AVNRT
- AVRT using a paraseptal AP

NO MANIFEST P WAVES

- AVNRT (retrograde p buried in the QRS)

AVRT

- Depression of ST segment
- RP interval > 90 ms
- AV dissociation or AV block during the tachycardia excludes the participation of an AP and makes AVNRT less likely
- QRS alternans (rapid rate related phenomena)

SHORT RP–LONG PR INTERVAL	LONG RP–SHORT PR INTERVAL
AV node reentry	Atrial tachycardia
AV reentry	Sinus node reentry Atypical AV node reentry AVRT with a slowly conducting accessory pathway (e.g., PJRT)

THANK YOU