Tachyarrhythmias

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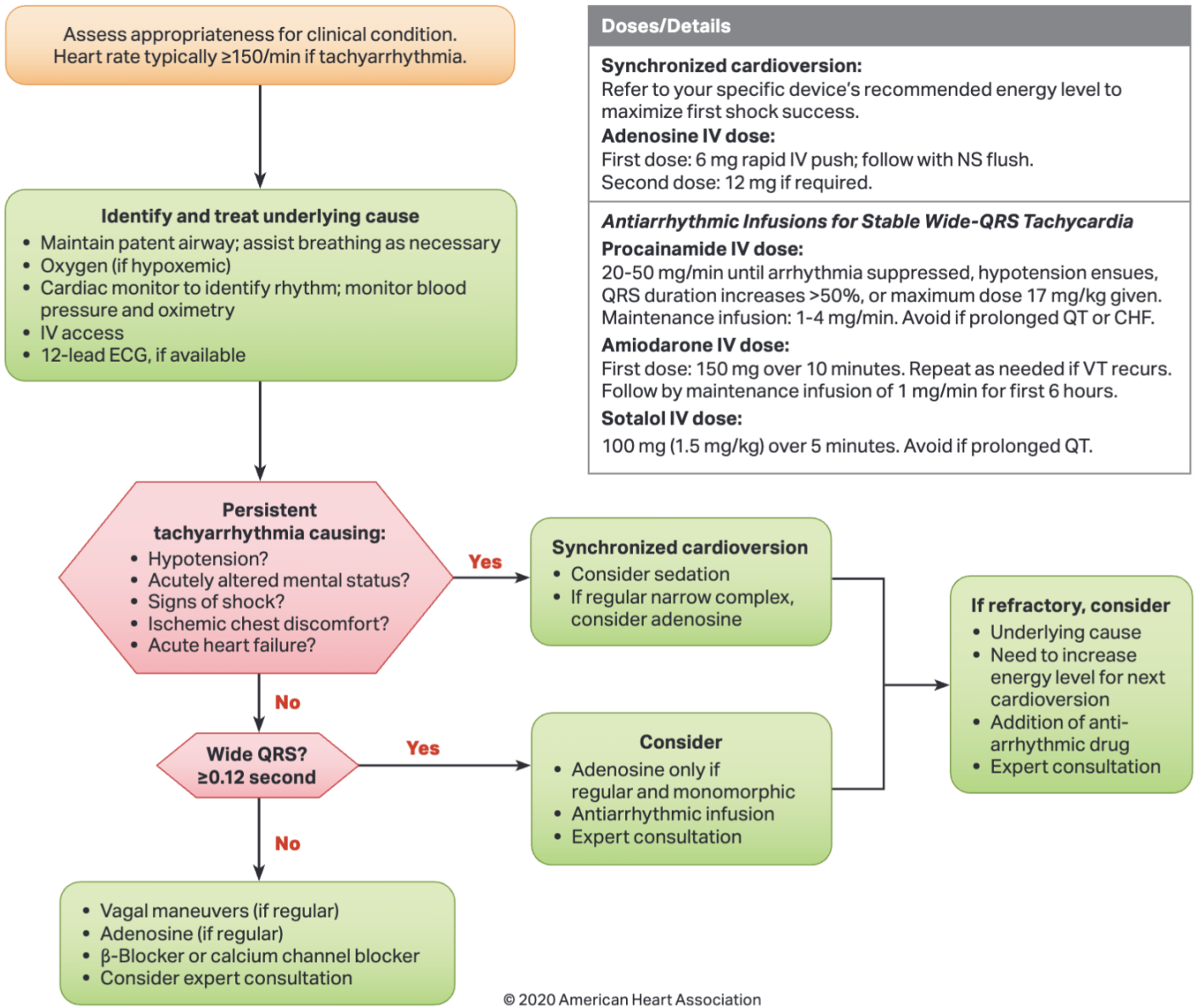
Outline

- Acute management management of any tachyarrhythmia
- Specific pearls of various tachyarrhythmias
- Differentiating VT from SVT with aberrancy

Adult Tachycardia With a Pulse Algorithm



Is the patient hemodynamically stable?
Is the QRS narrow or wide?
Is the rhythm regular or irregular?

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What is a tachyarrhythmia?

- Heart rate > 100bpm, often > 150 for tachyarrhythmia
- An abnormal electrical rhythm (not sinus tachycardia)
- Often symptomatic

Initial diagnostics

- Hook up to telemetry
- Vital signs
- 12 leads ECG if patient is stable

Signs of hemodynamic instability

- Hypotension
- Severe shortness of breath
- Severe chest pain
- Shock
- Altered level of consciousness

- If HD unstable → urgent cardioversion

Initial tachycardia characterization

- **Precise diagnosis is not necessary**
- Wide complex (>120msec) or narrow complex (<120msec)?
- Regular or irregular?

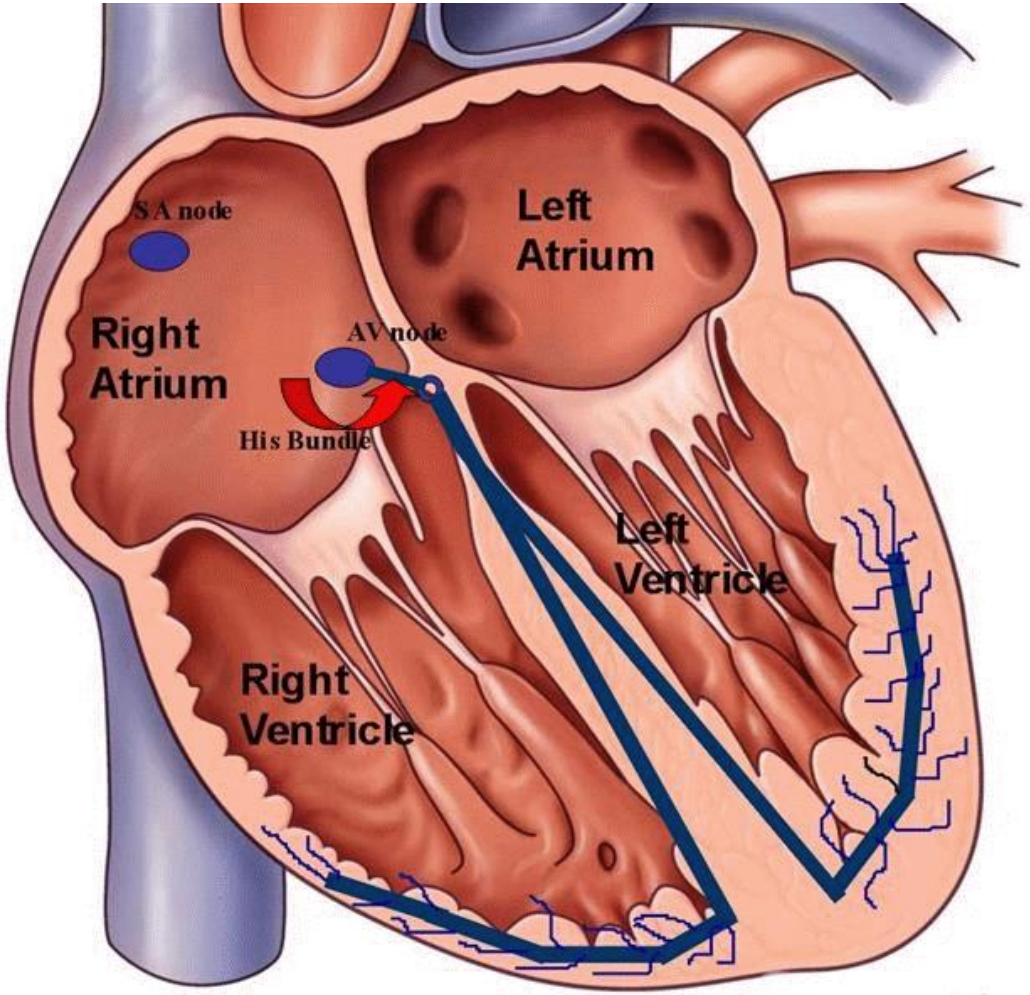
Narrow complex regular tachycardia

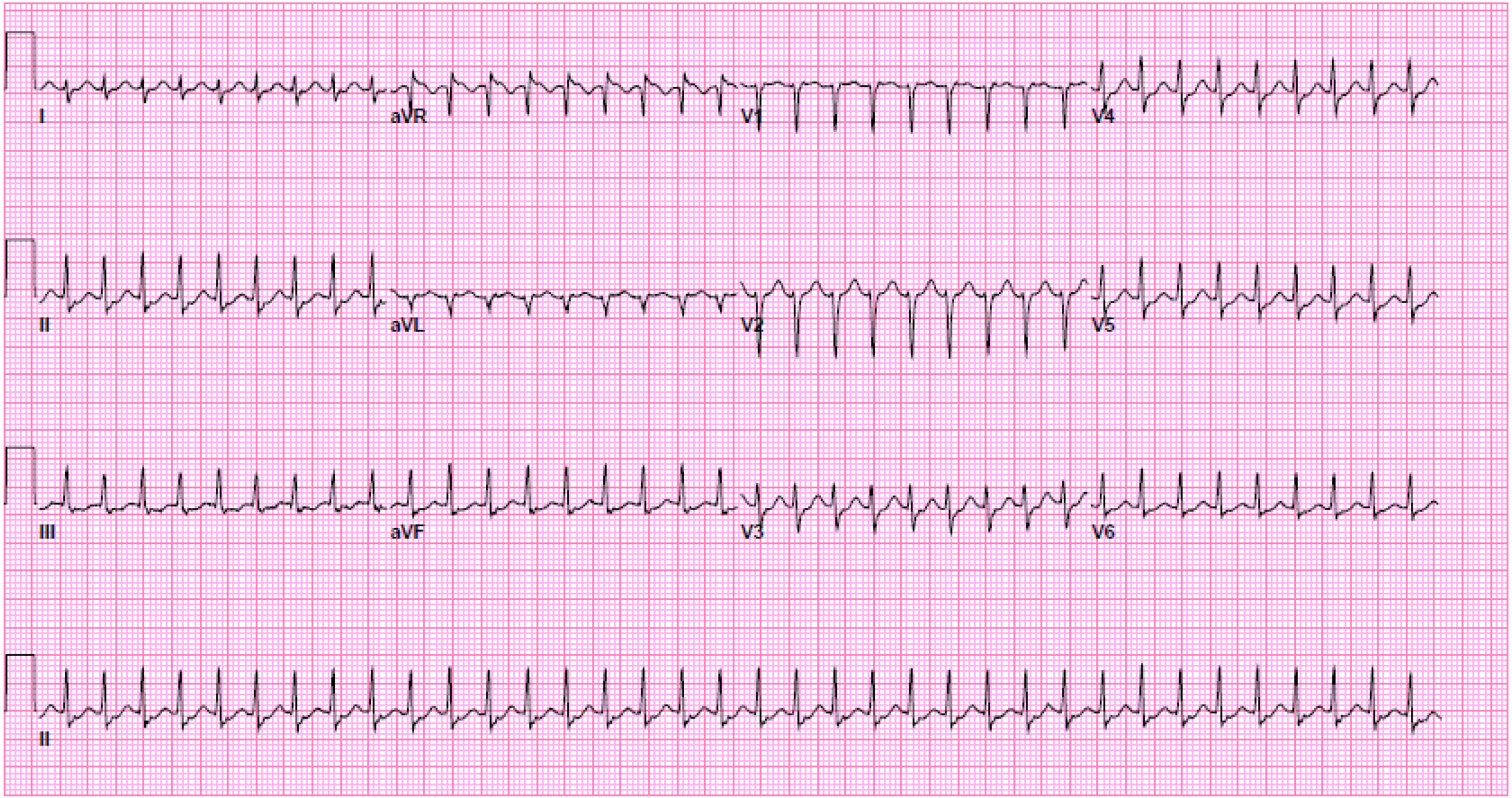
DDx of narrow complex regular tachycardia

- Sinus tachycardia
- **Atrioventricular nodal tachycardia (AVNRT)**
- **Orthodromic atrioventricular reentry tachycardia (AVRT, oAVRT, ORT)**
- **Focal atrial tachycardia**
- Atrial flutter with consistent AV conduction pattern
- Junctional tachycardia
- Sinoatrial nodal re-entry tachycardia
- Rare VT’s near the His Purkinje system

AVNRT

- Re-entry circuit around the AV node
- Usually abrupt onset/offset
- Simultaneous activation of the atrium and ventricle
- Pulsating sensation in the neck from ”cannon A waves” with atrium contracting against closed tricuspid valve





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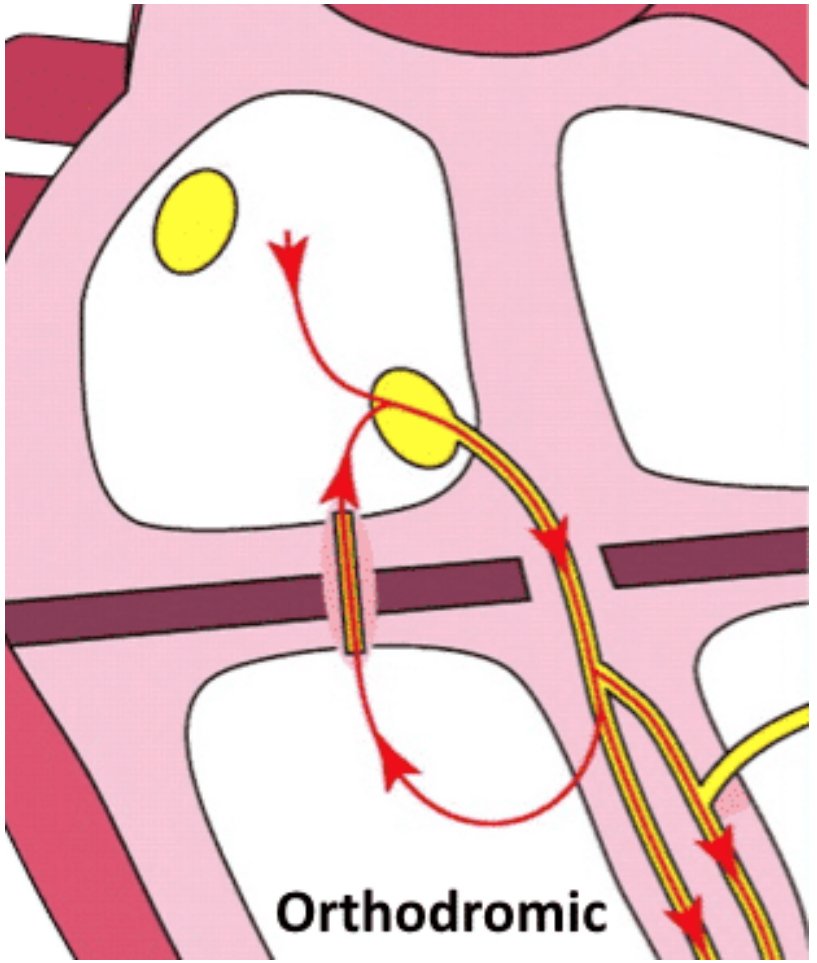
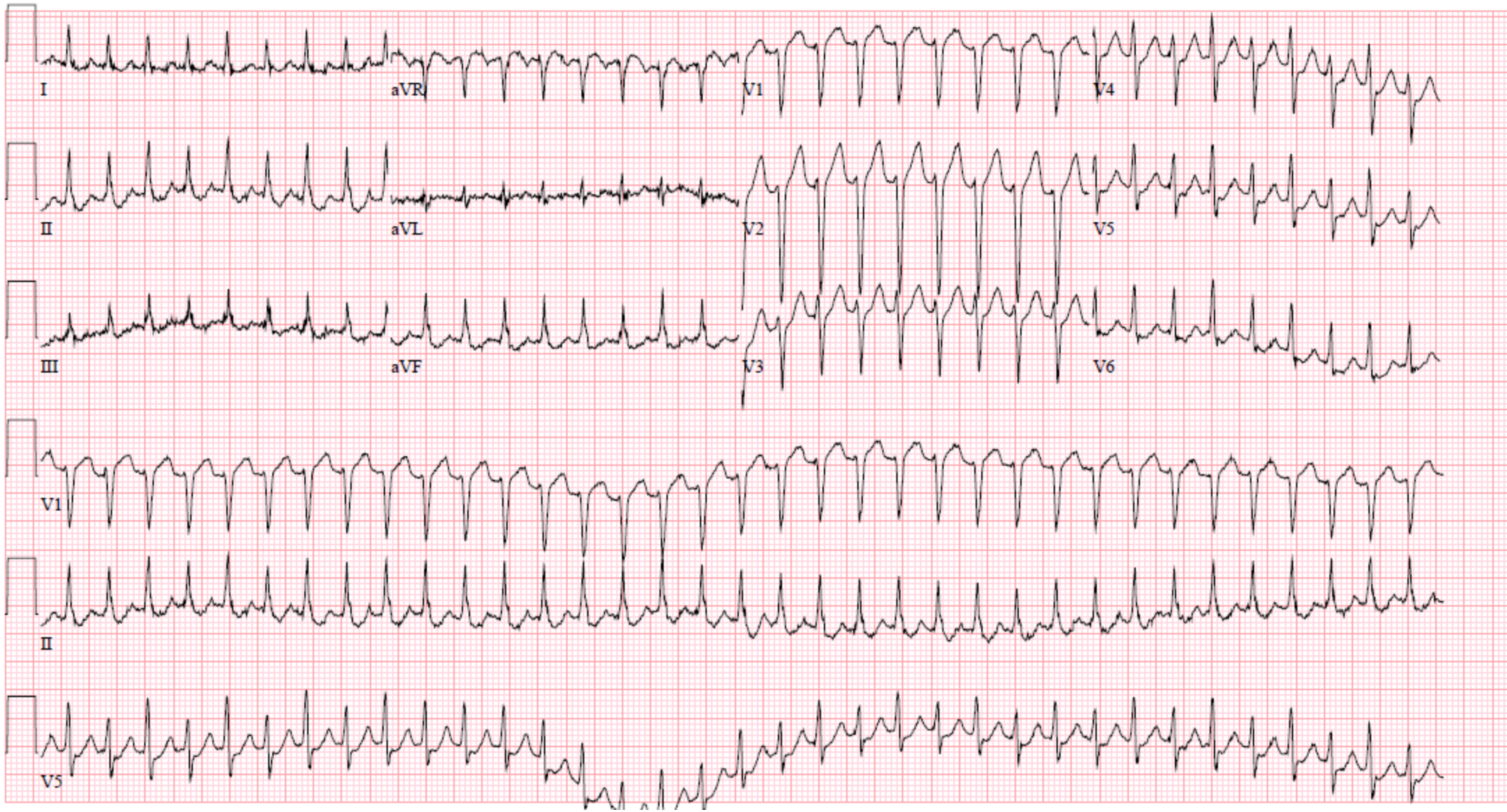
Orthodromic AVRT

- Reentry
- Anterograde limb: AV node
- Retrograde limb: accessory pathway
- Abrupt onset/offset
- Difficult to distinguish from AVNRT unless baseline WPW pattern is present





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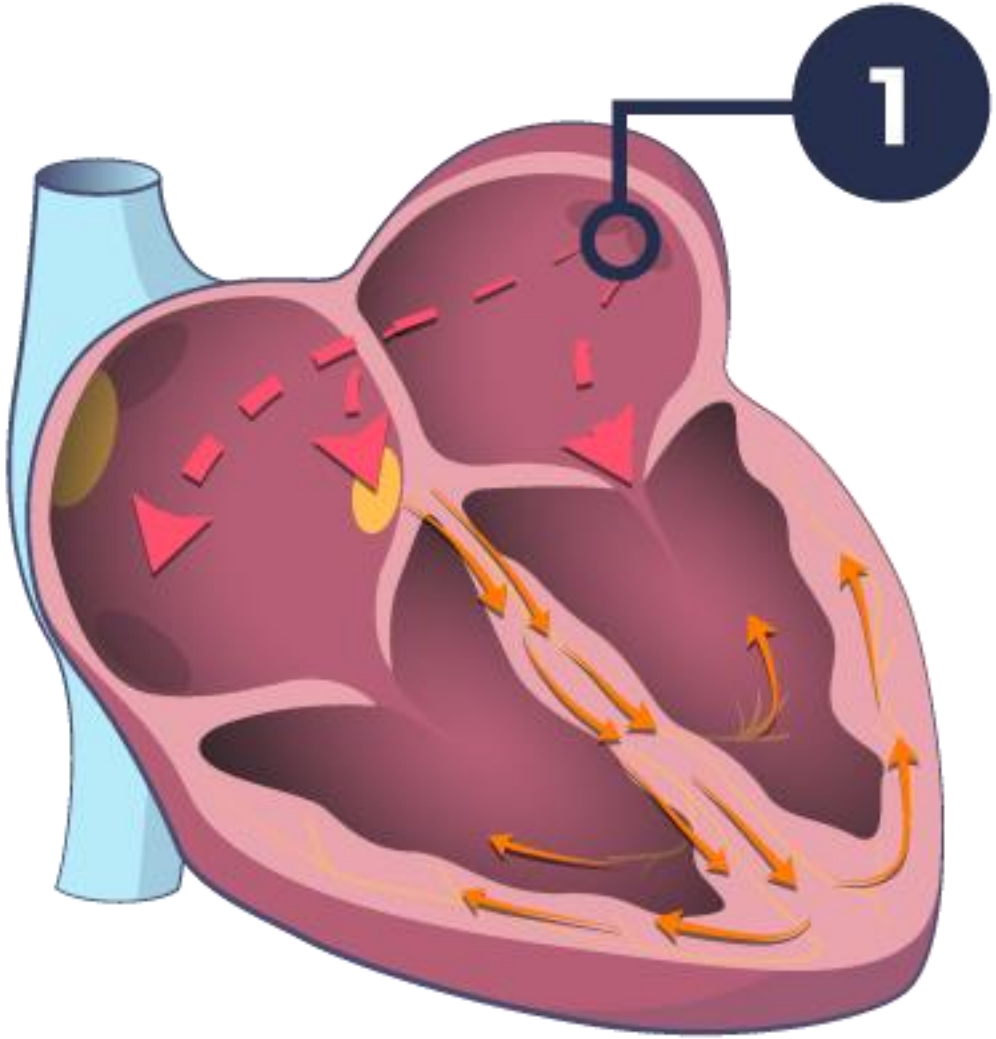
Focal atrial tachycardia

- Point source of ectopic atrial impulse formation and tachycardic
- Can have gradual onset



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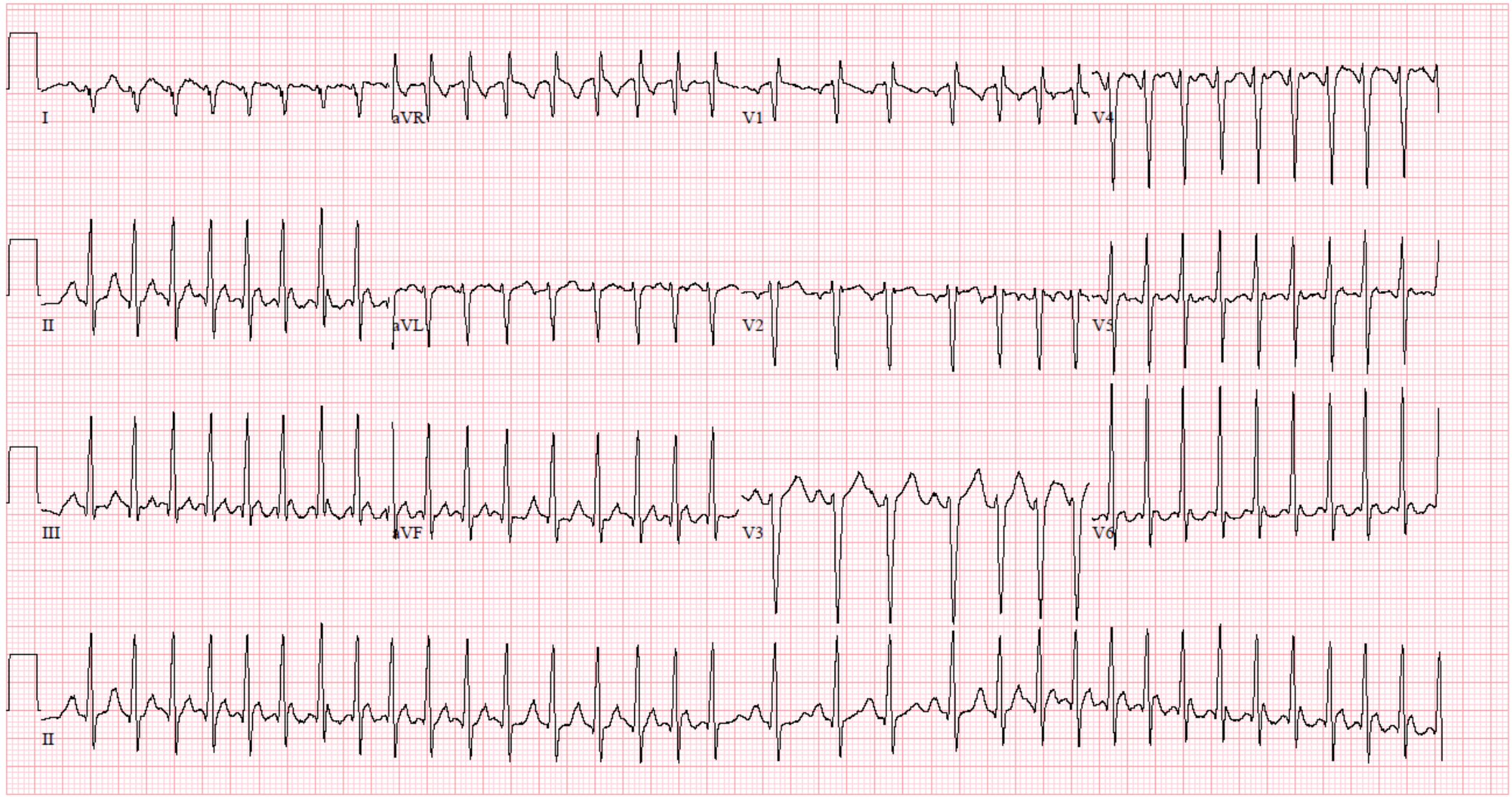

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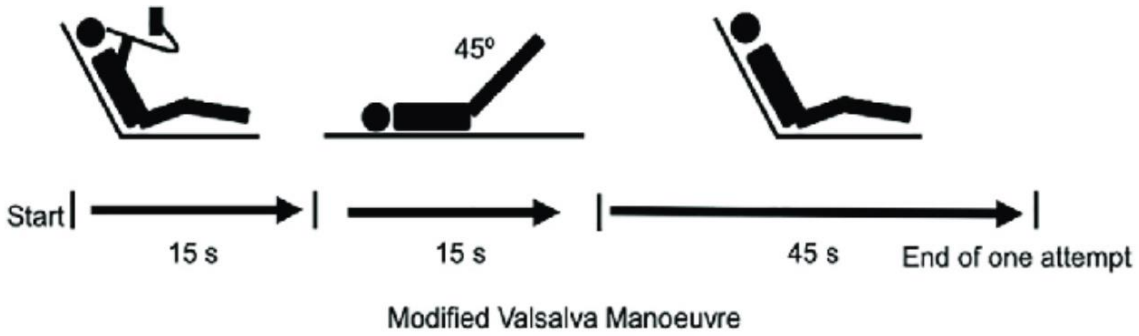
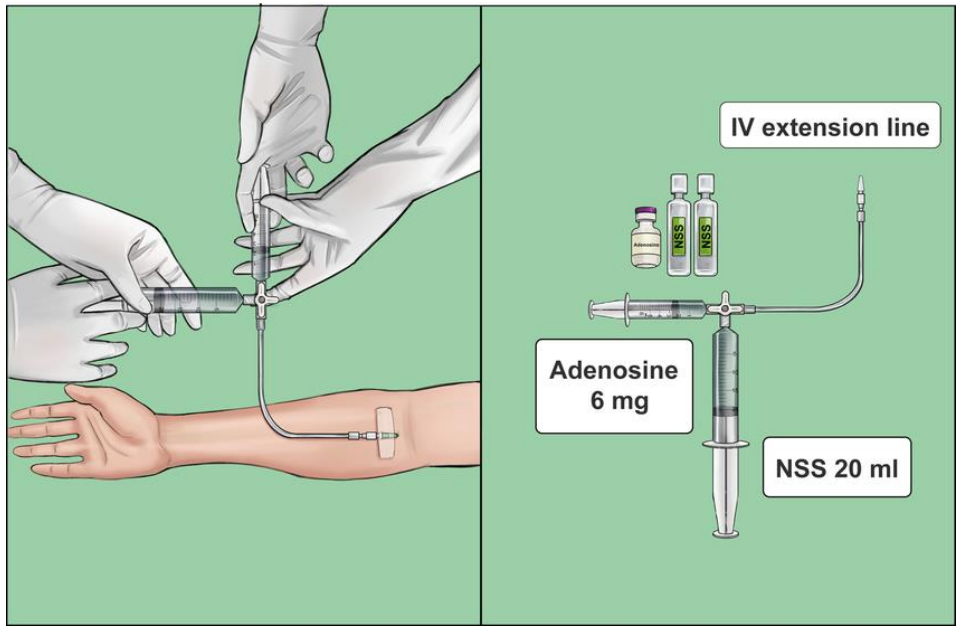
Stable patient with regular NCT

- Vagal maneuvers
  - Bearing down
  - Carotid sinus massage
  - Modified Valsalva
- Administration of IV adenosine
  - 6mg via peripheral IV, flushed with saline
  - Can follow with 12mg
  - Less or avoid in heart transplant
    - Presumed risk of prolonged AV block in setting of a parasympathetic denervated heart
  - Pads on and connected to defibrillator



Modified Valsalva Manoeuvre



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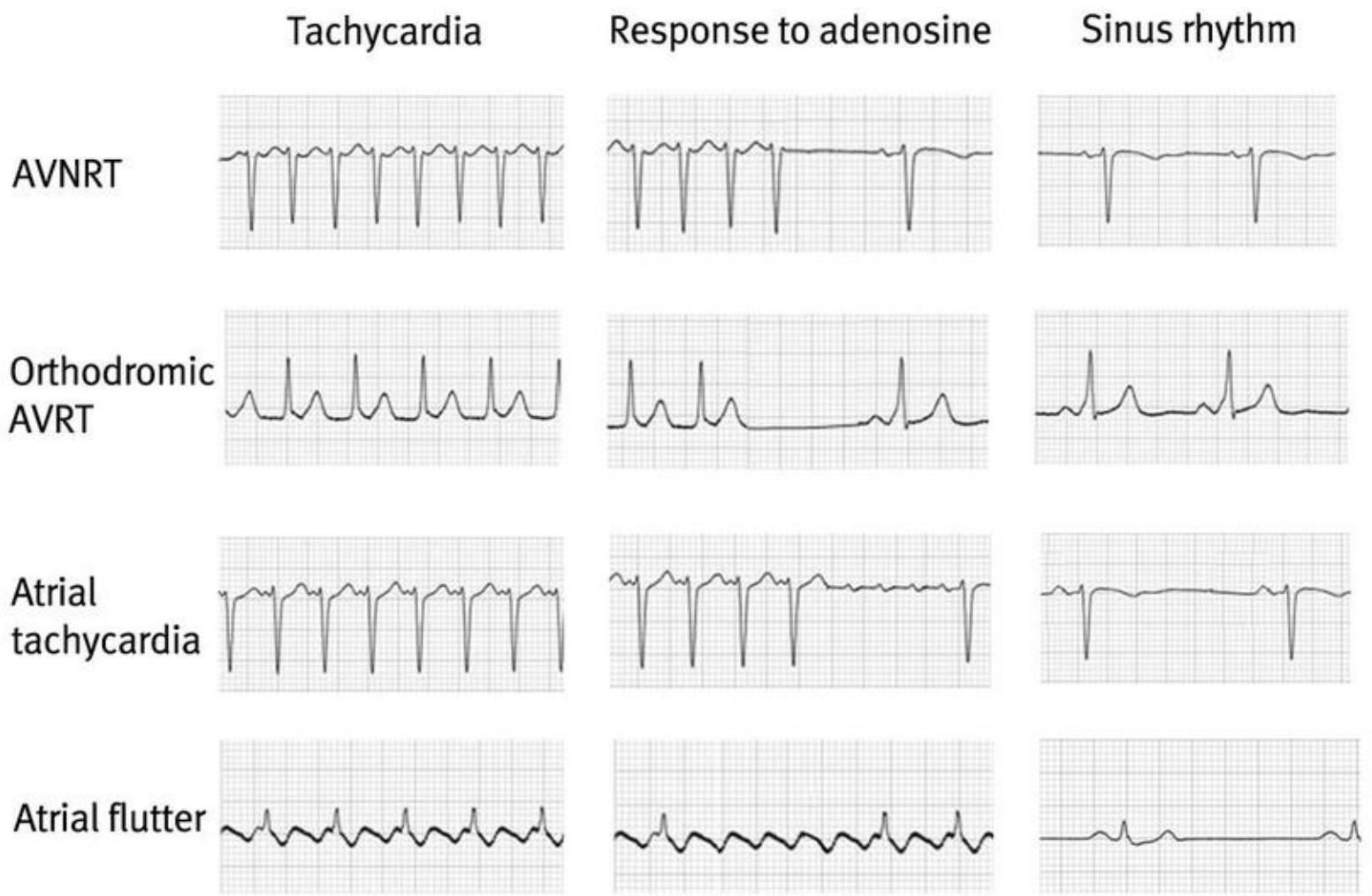
Typical adenosine responses

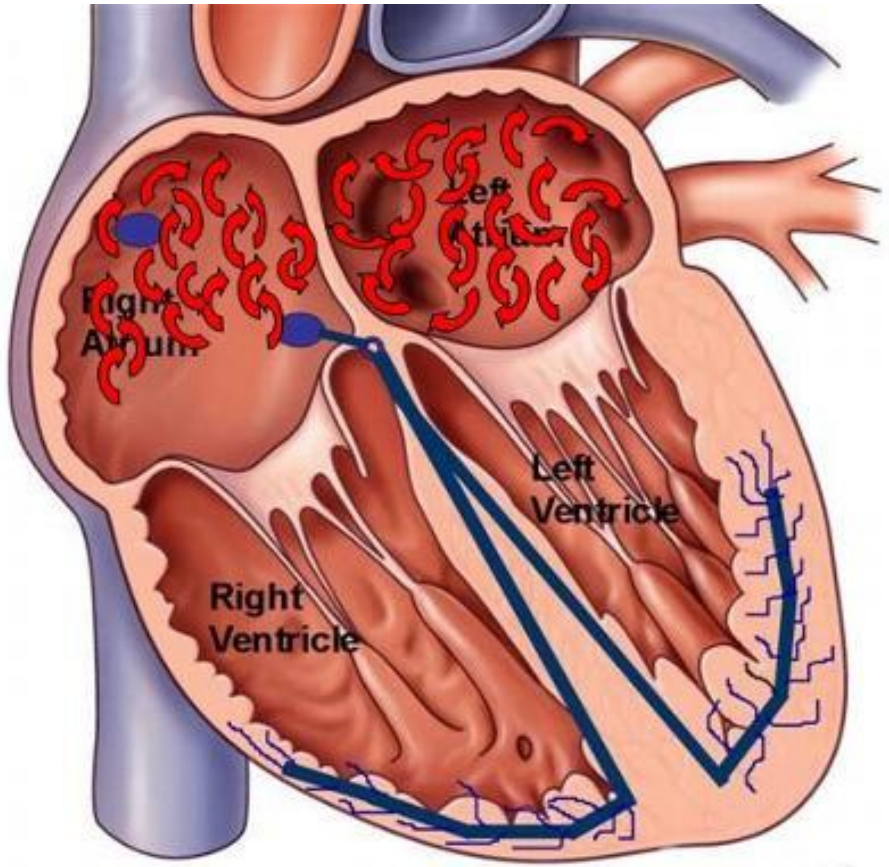


DDx of narrow complex irregular tachycardia

- Atrial fibrillation
- Multifocal atrial tachycardia
- Sinus tachycardia with frequent atrial ectopy
- Focal atrial tachycardia with variable block
- Atrial flutter with variable block

Atrial fibrillation

- Fibrillatory waves in the atrium at 300-600bpm
- Irregularly irregular rhythm
- Usually can be distinguished from other SVTs without need for diagnostic intervention



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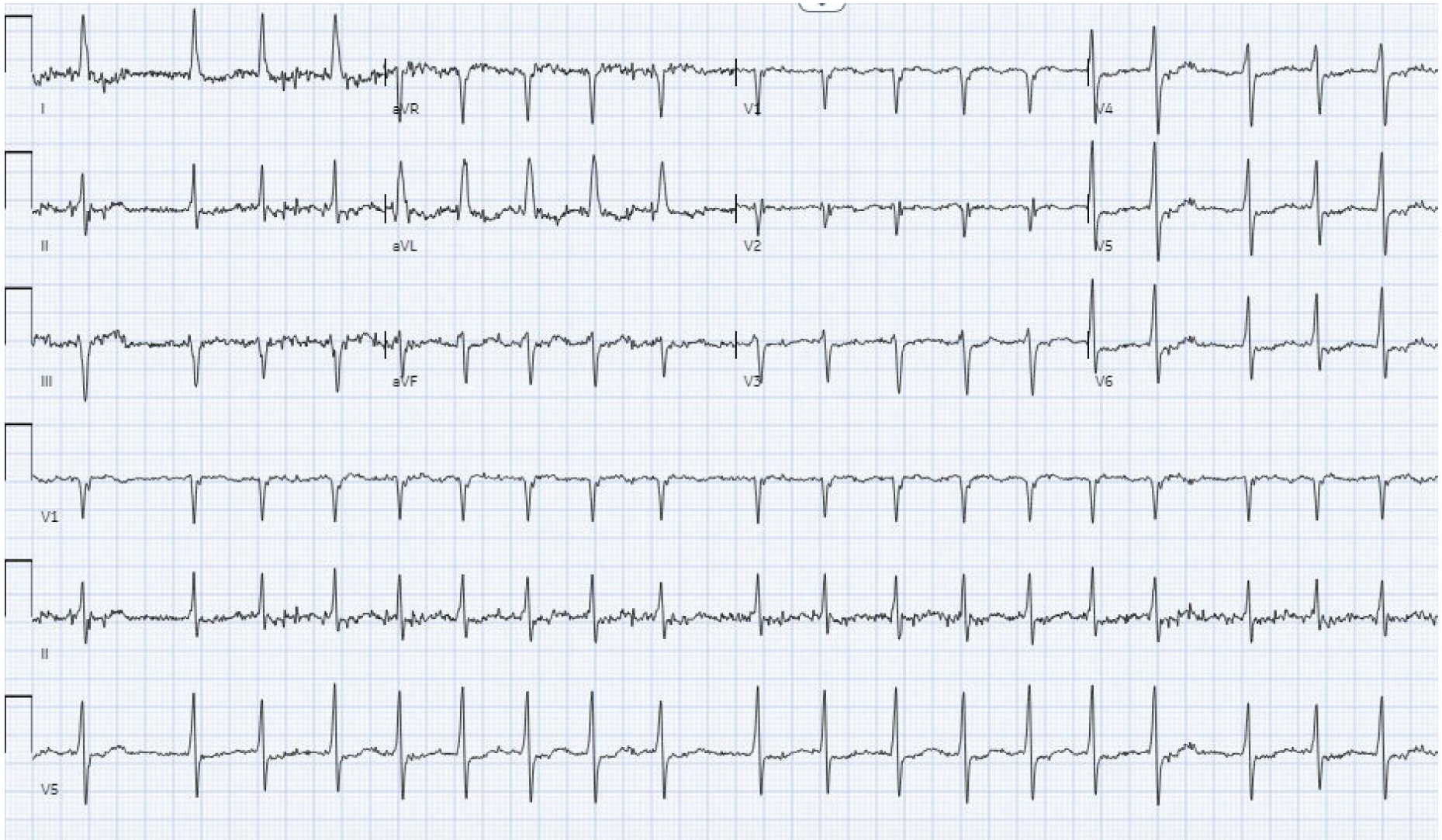

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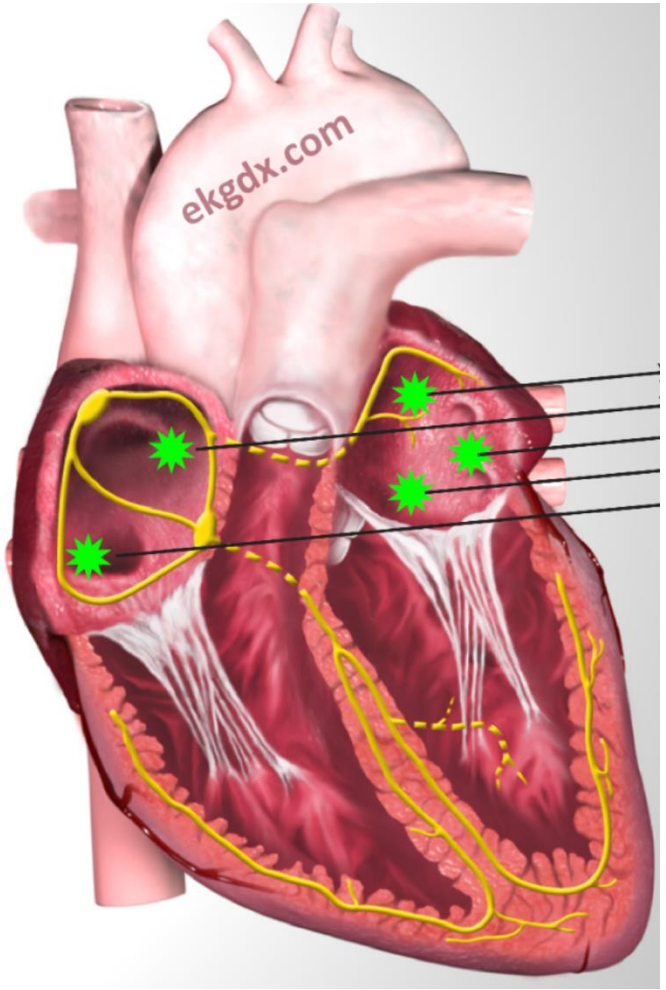
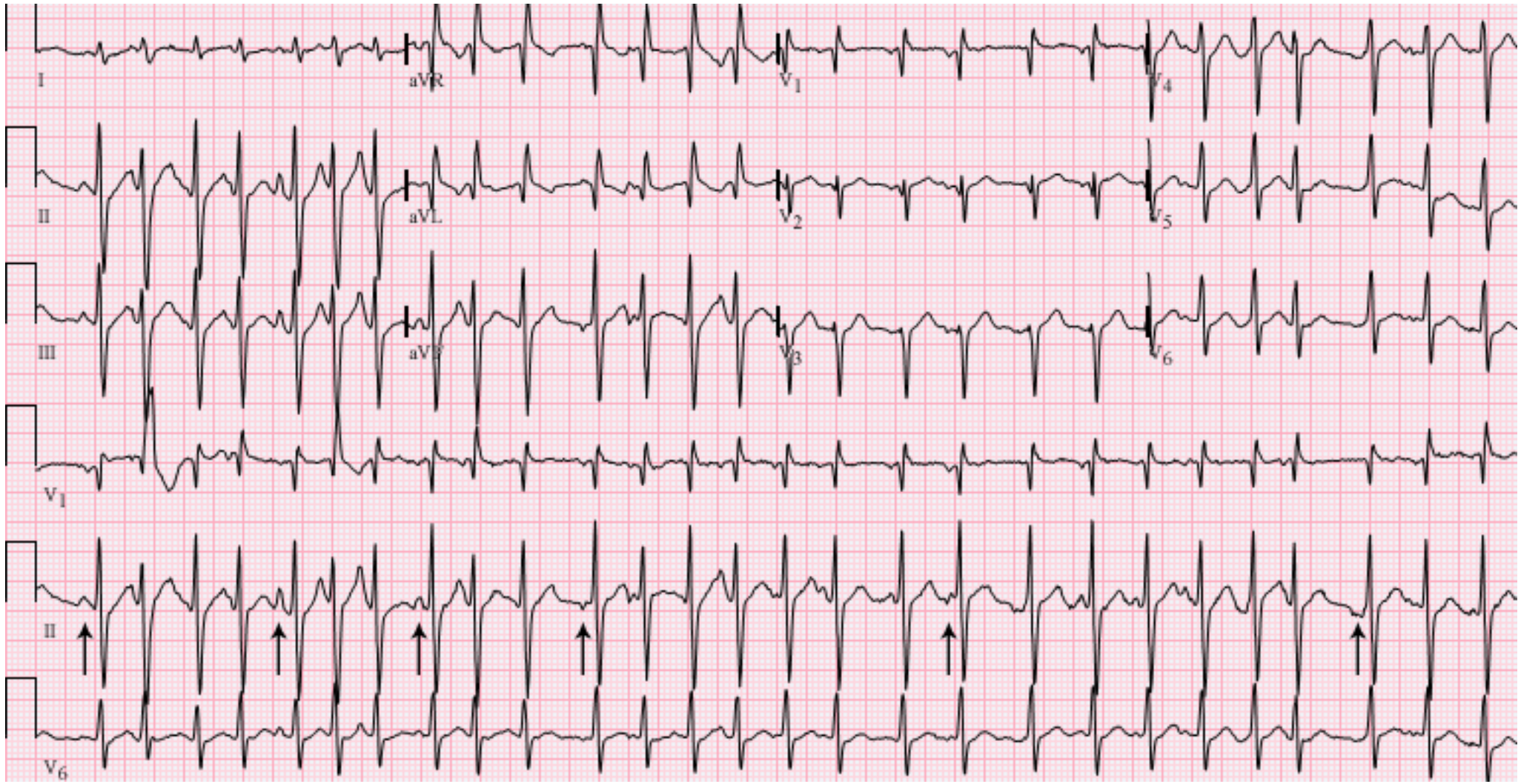
Multifocal atrial tachycardia

- Rapid, irregular rhythm
- >3 ectopic atrial foci
- Varying PR interval beat to beat
- No single dominant P wave morphology
- Associated with pulm disease/acute illness/theophylline
- Tx → treatment of underlying illness





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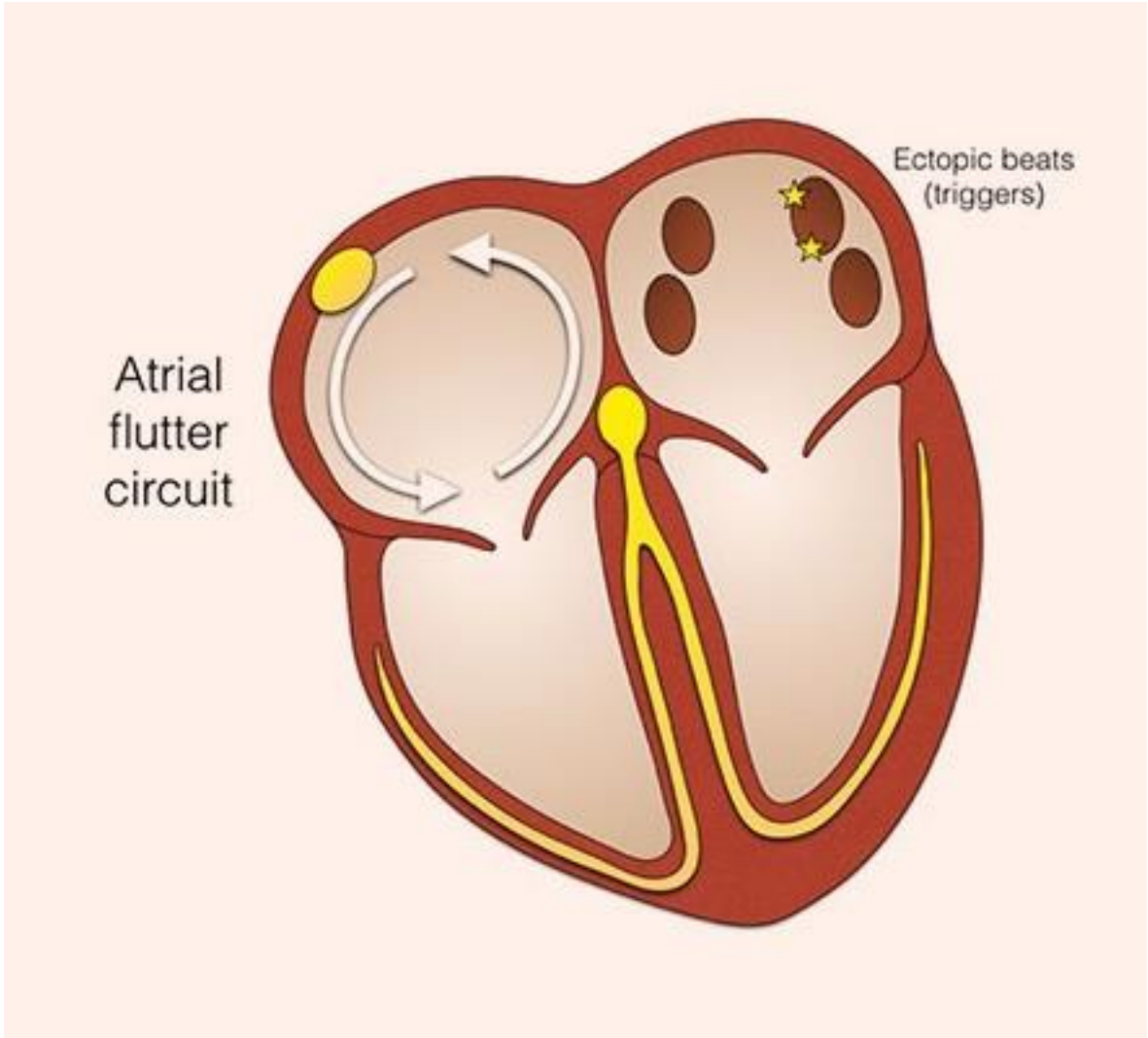
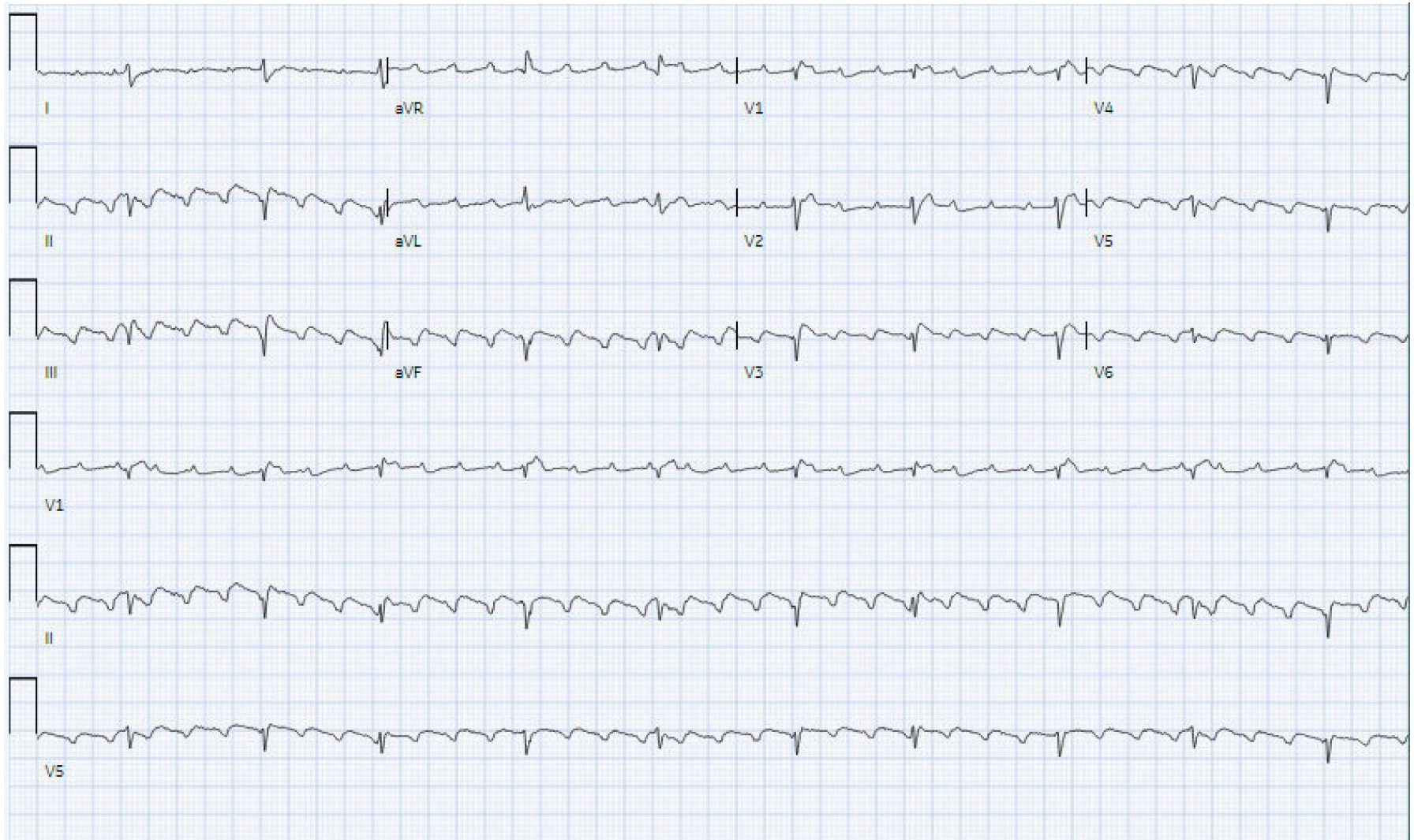
Atrial flutter

- Re-entry circuit within the atria
- Typical flutter – re-entry around the the tricuspid annulus
  - Sawtooth flutter waves





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Acute management of irregular NCT

- No role for adenosine
- Treatment of underlying illnesses
- Nodal blocking agents
- TEE/DCCV for atrial fibrillation/flutter
- Antiarrhythmic agents

Long term management

- Nodal blocking agents – beta blockers, calcium channel blockers, digoxin
- Antiarrhythmic agents
- Catheter ablation

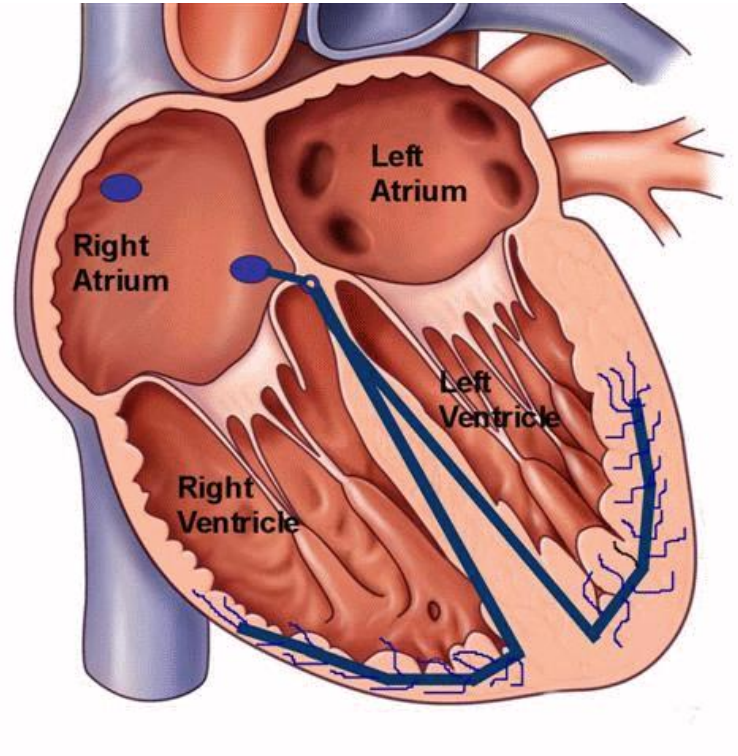
Wide complex tachycardia

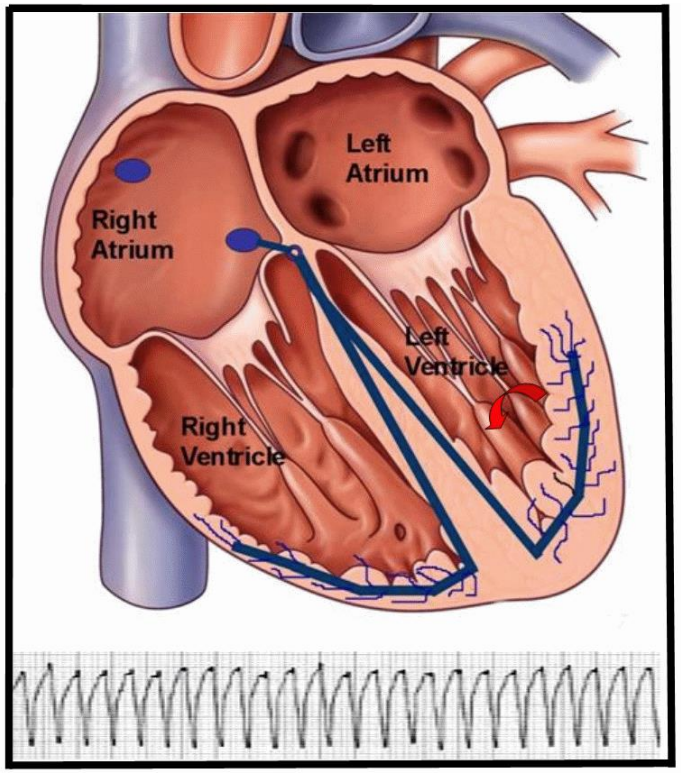
DDx of wide complex regular tachycardia

- Ventricular tachycardia
- Supraventricular tachycardia with:
  - Aberrant conduction (bundle branch block)
  - Ventricular pacing
  - Pre-excitation (WPW, anterograde accessory pathway conduction)
- Antidromic atrioventricular reentry tachycardia (AVRT, aAVRT, ART)
- Some mimickers
  - Flecainide toxicity, hyperkalemia

Ventricular tachycardia

- Originating from ventricles
- Re-entry most common mechanism
- Usually does not terminate with adenosine or nodal blockers





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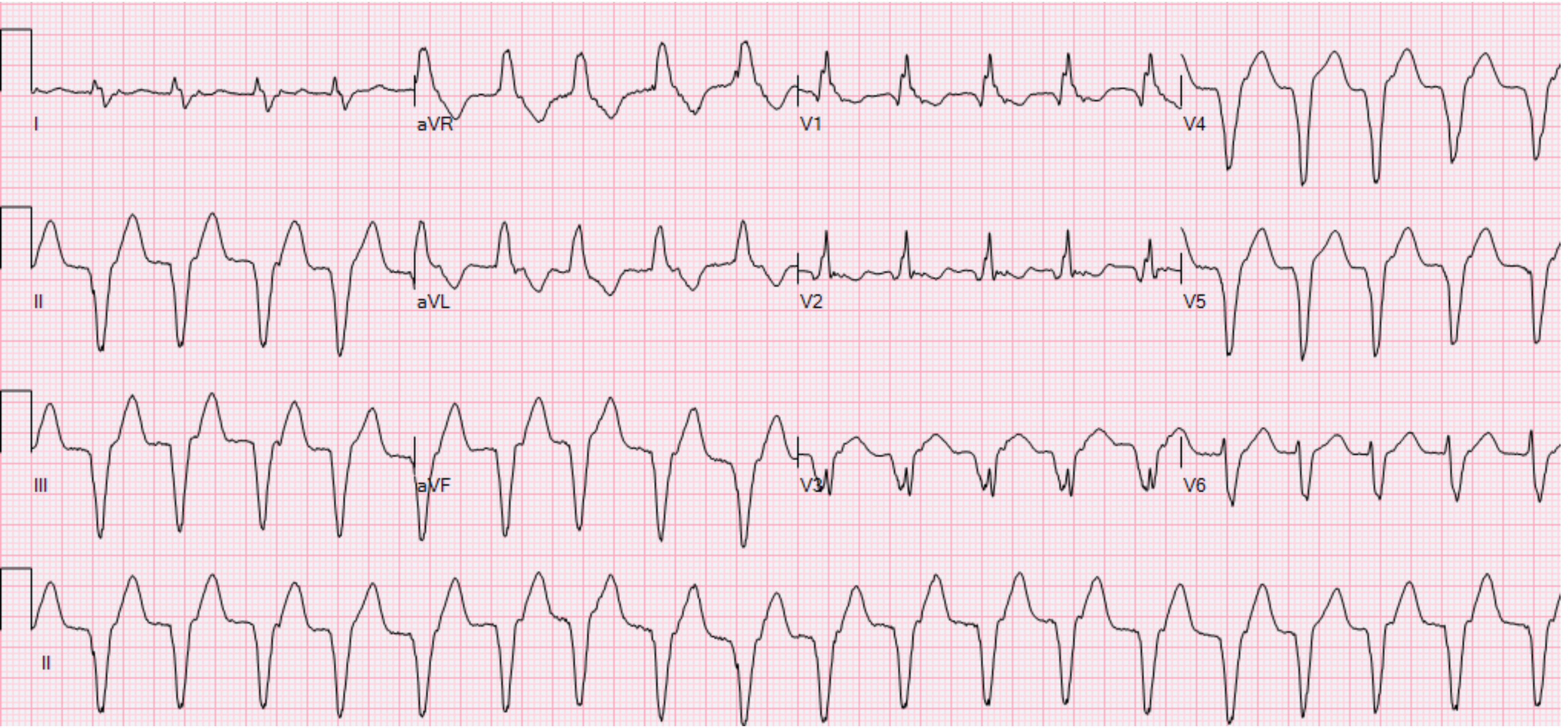

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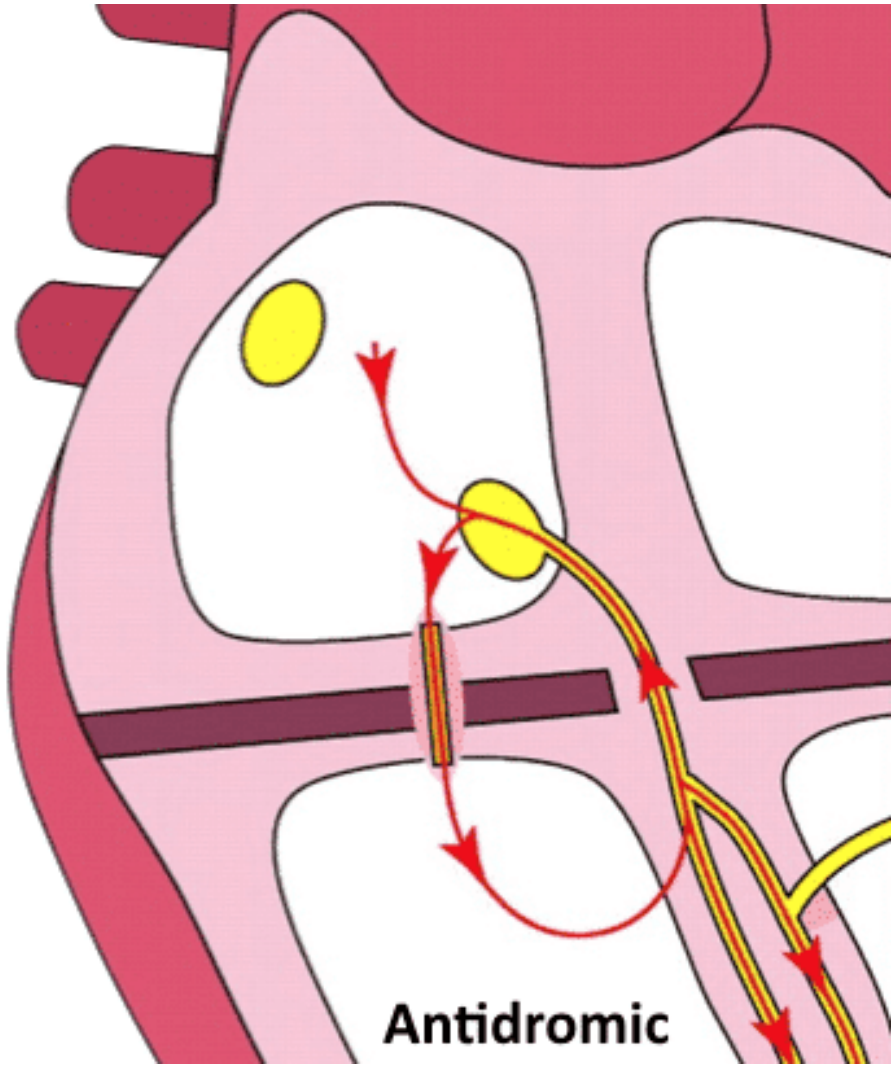
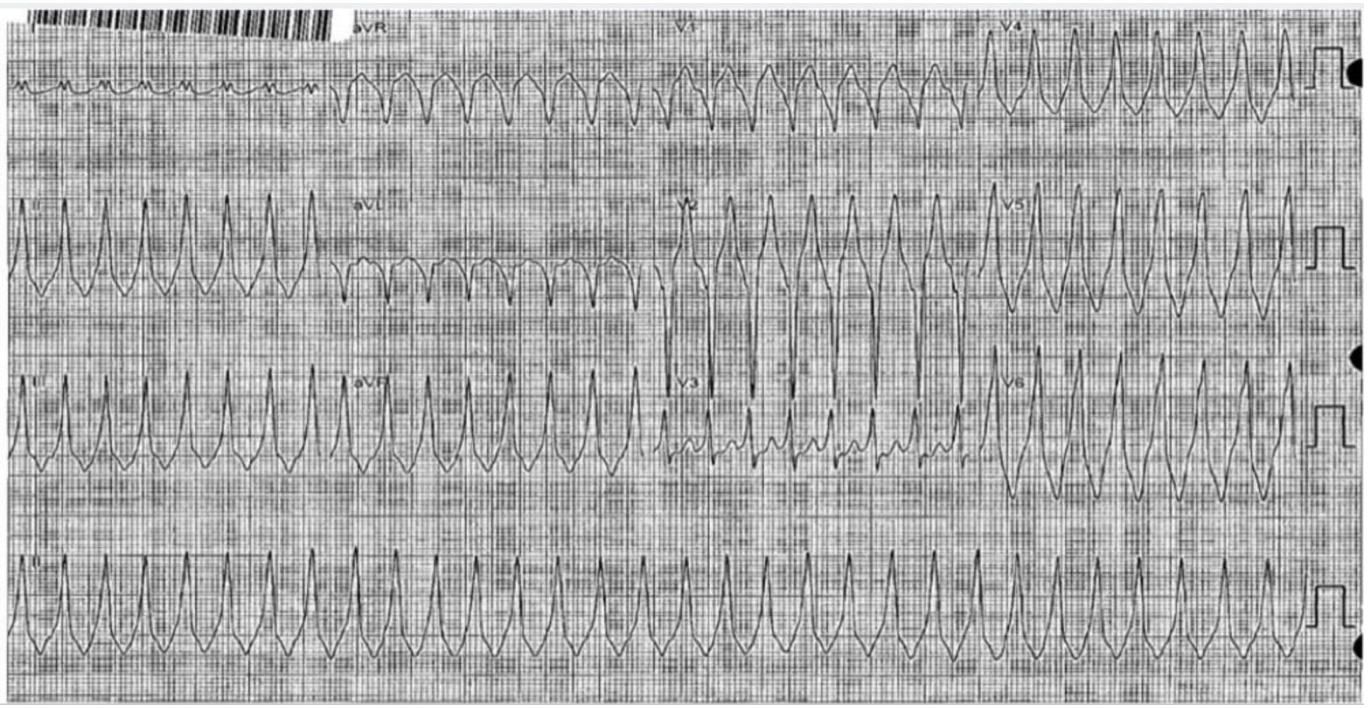
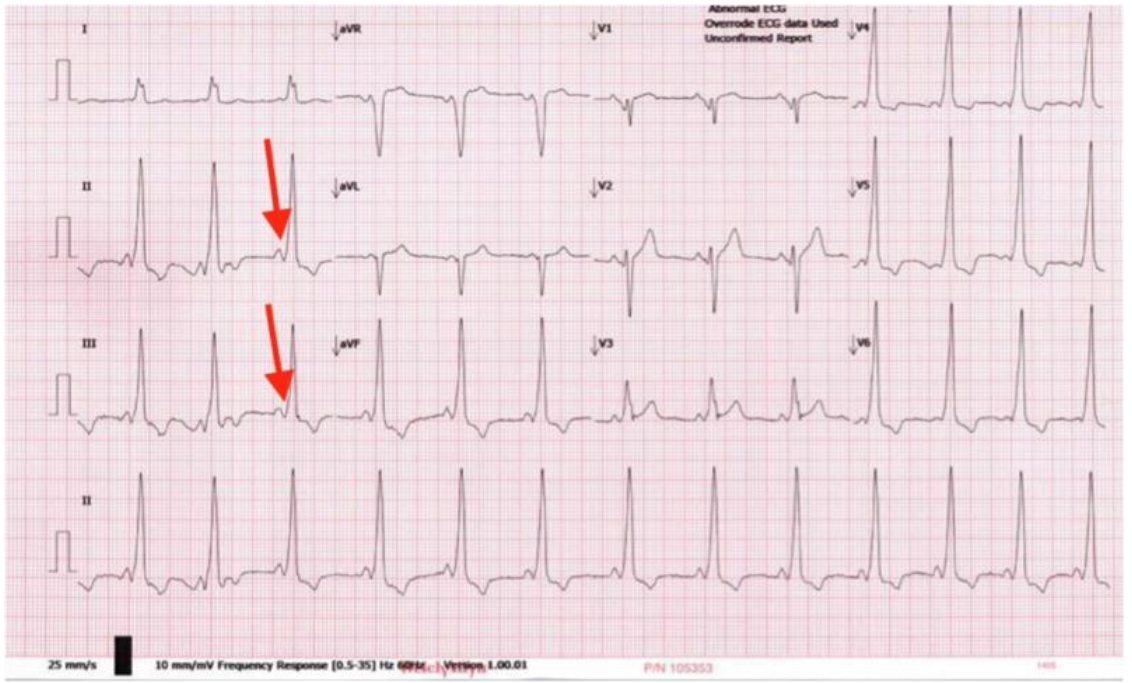
Antidromic AVRT

- Re-entry
- Anterograde limb – Accessory pathway
- Retrograde limb – AV node
- Should terminate with adenosine, nodal blocking agents
- Baseline ECG usually shows pre-excitation (WPW)



WCT treated with adenosine





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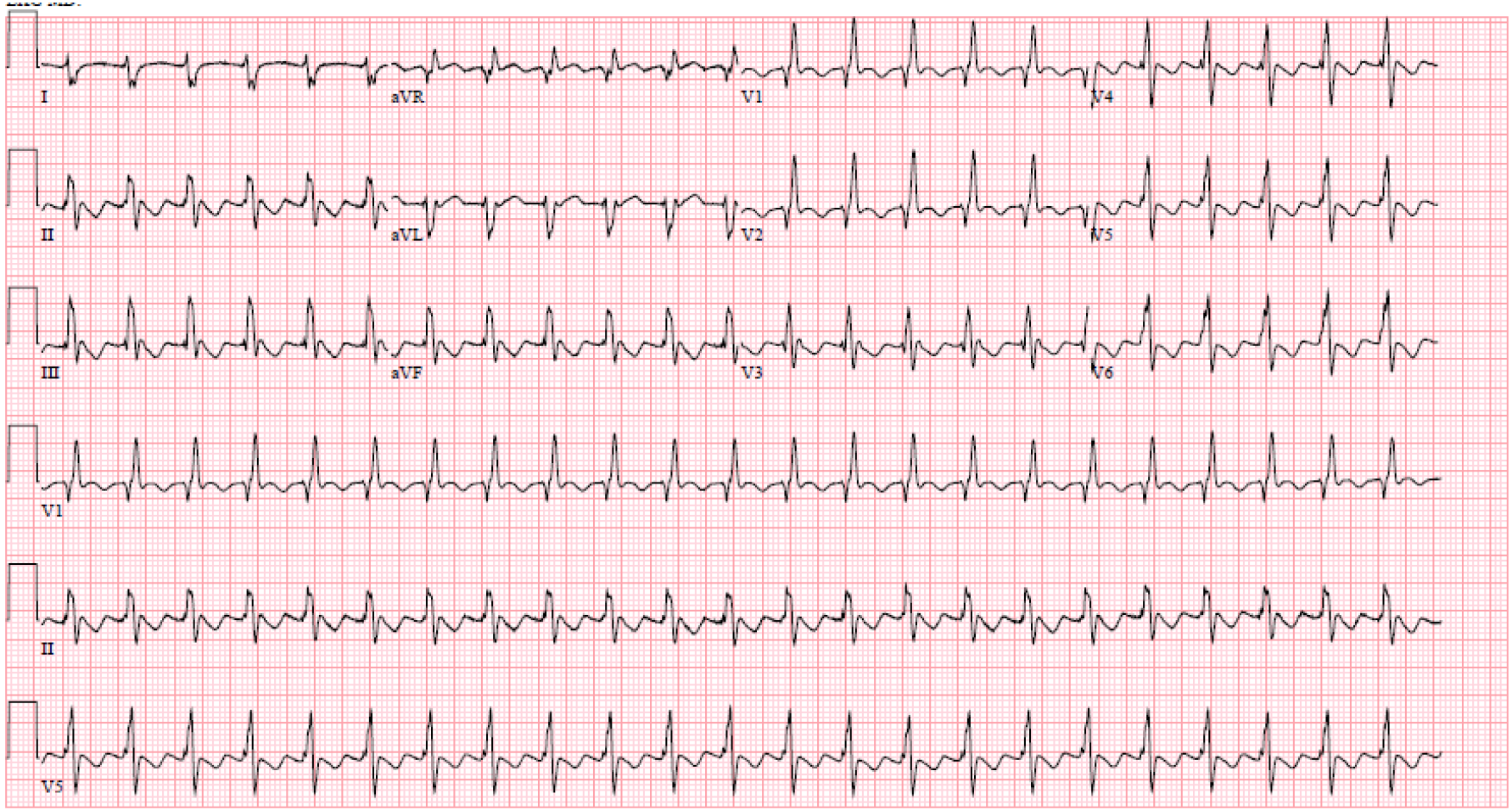
SVT with aberrancy

- Any SVT with a LBBB/RBBB
- BBB can be rate-related or can be present at baseline



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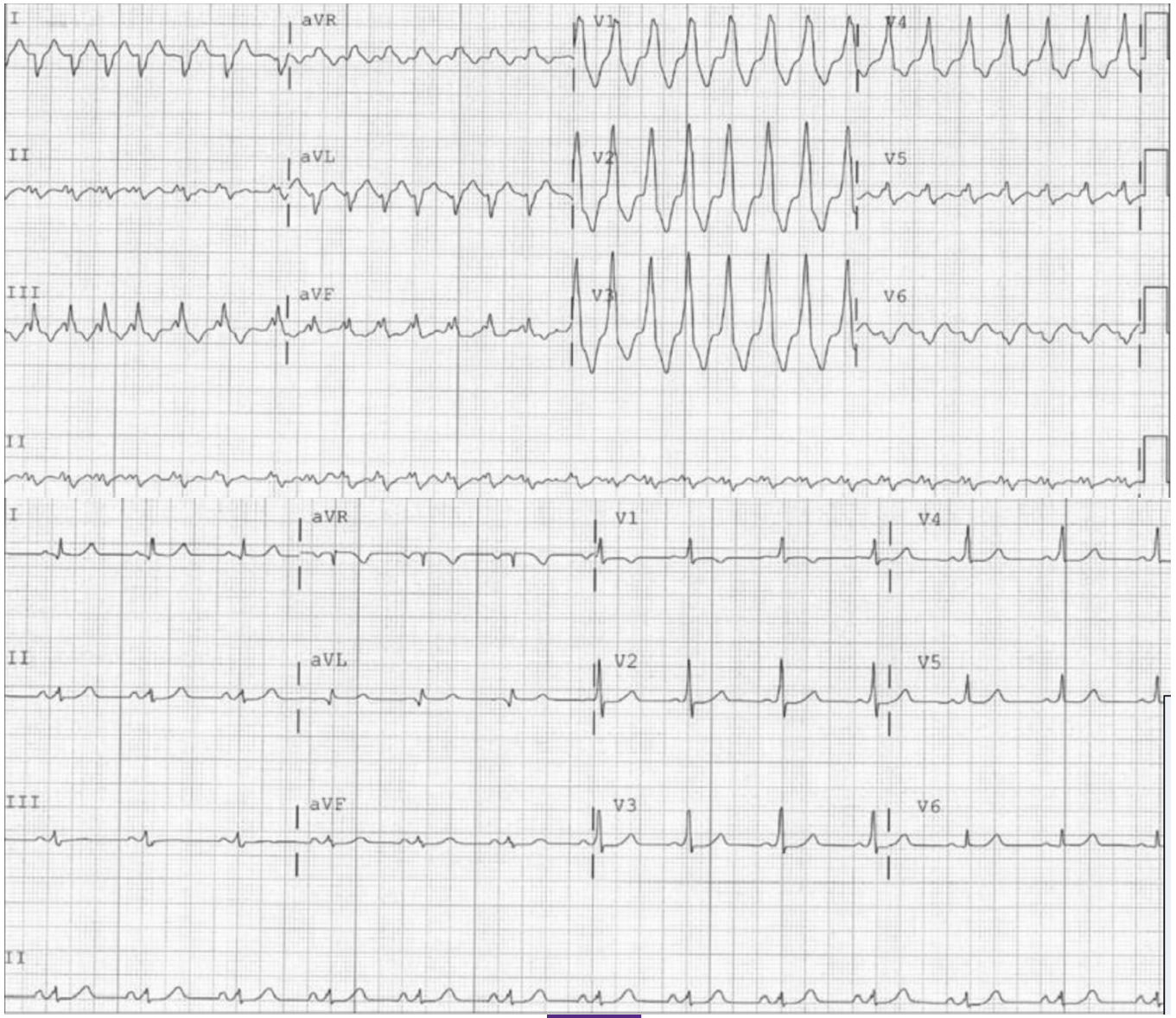
SVT with bystander pre-excitation

- SVT independent of an accessory pathway with bystander activation down an accessory pathway.
- Likely hard to distinguish from VT or on an ECG
- Baseline ECG usually will show pre-excitation (WPW)



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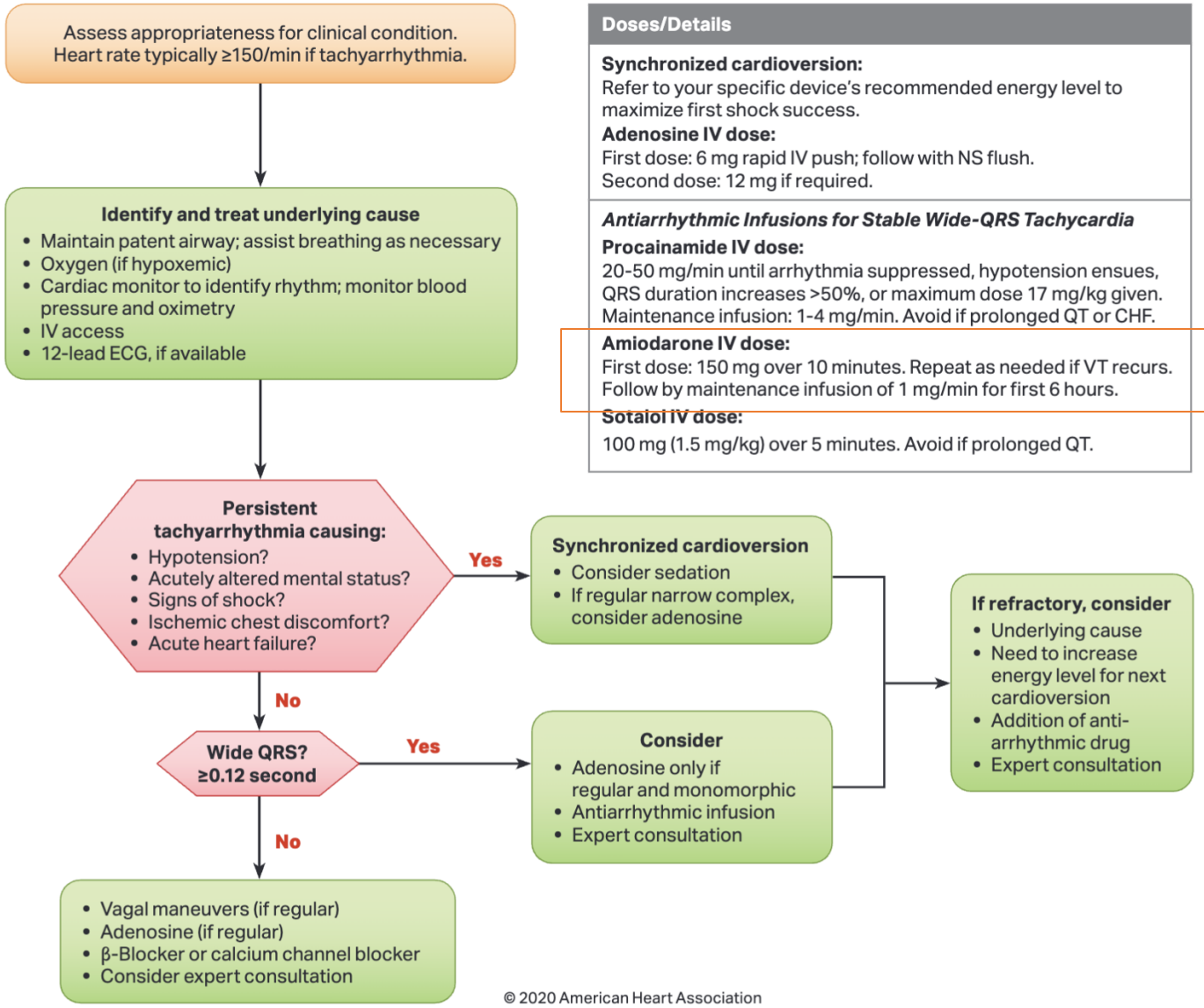
Stable patient with regular WCT

- If clearly VT, treat as VT
  - Criteria for distinguishing VT from SVT
  - Clinical substrate
- If uncertain and patient stable, consider adenosine
  - Most VTs will be unaffected by adenosine
  - Many SVTs with aberrancy will be terminated by adenosine
  - AT with BBB or pre-excitation → AT with AV block

Adult Tachycardia With a Pulse Algorithm



- Amiodarone usually drug of choice in the acute setting for WCT of uncertain etiology or VT.
- If unstable, don't wait on initiation

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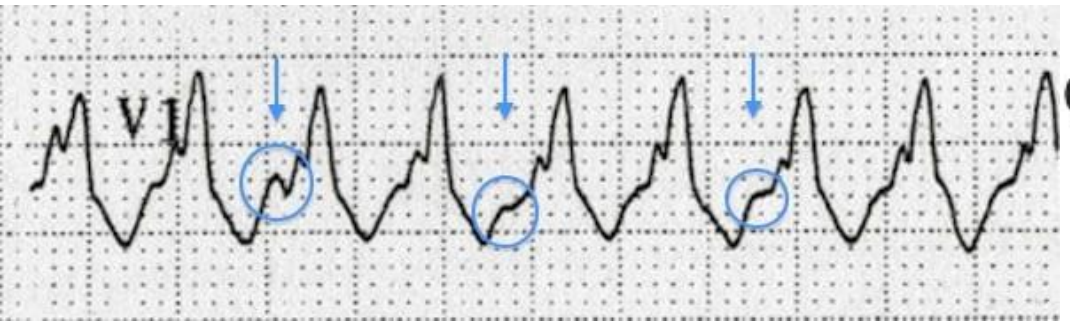
Factors pointing toward VT

- Atypical appearance of BBB
- Precordial concordance
  - All positive in precordial leads
  - All negative in precordial leads
- Extreme “northwest” axis deviation - +AVR, - in I and aVF
- AV dissociation
  - P waves independent of ventricular rhythm
  - Capture beats – Sinus beats conduct through HPS alone
  - Fusion beats – Sinus beats conducting through HPS fused with VT beat

Signs of AV dissociation



Detectable atrial rhythm independent of ventricular rhythm

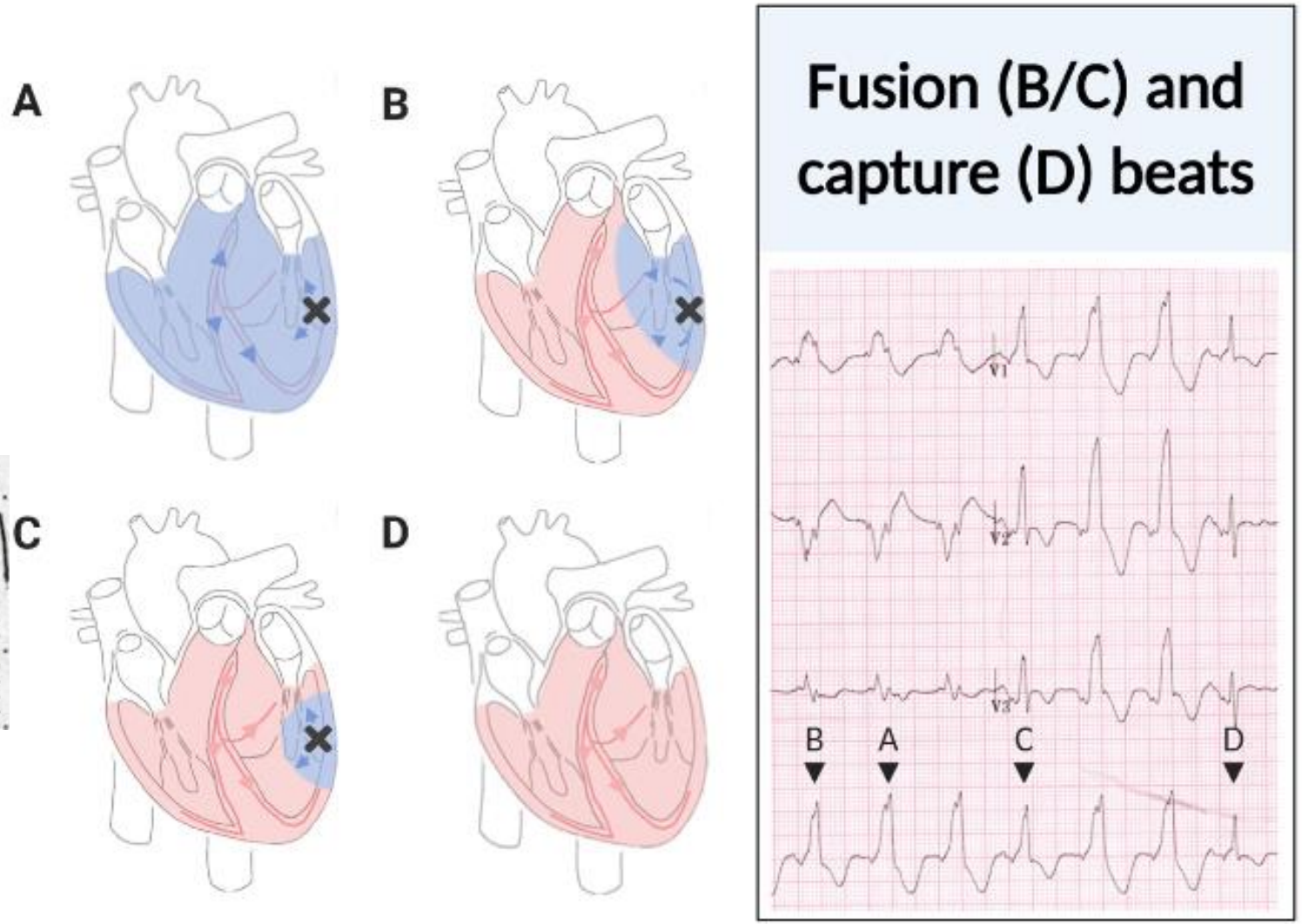


Figure 1 Generation of capture and fusion beats with corresponding changes on electrocardiogram due to competing activation from supraventricular and ventricular tachycardia source. Activation from a ventricular tachycardia source is indicated in *blue* and sinus rhythm is indicated in *red*.

Ding, Wern Yew, and Saagar Mahida. 2021. "Wide Complex Tachycardia: Differentiating Ventricular Tachycardia from Supraventricular Tachycardia." *Heart* 107 (24): 1995–2003.

Atypical QRS morphology → VT

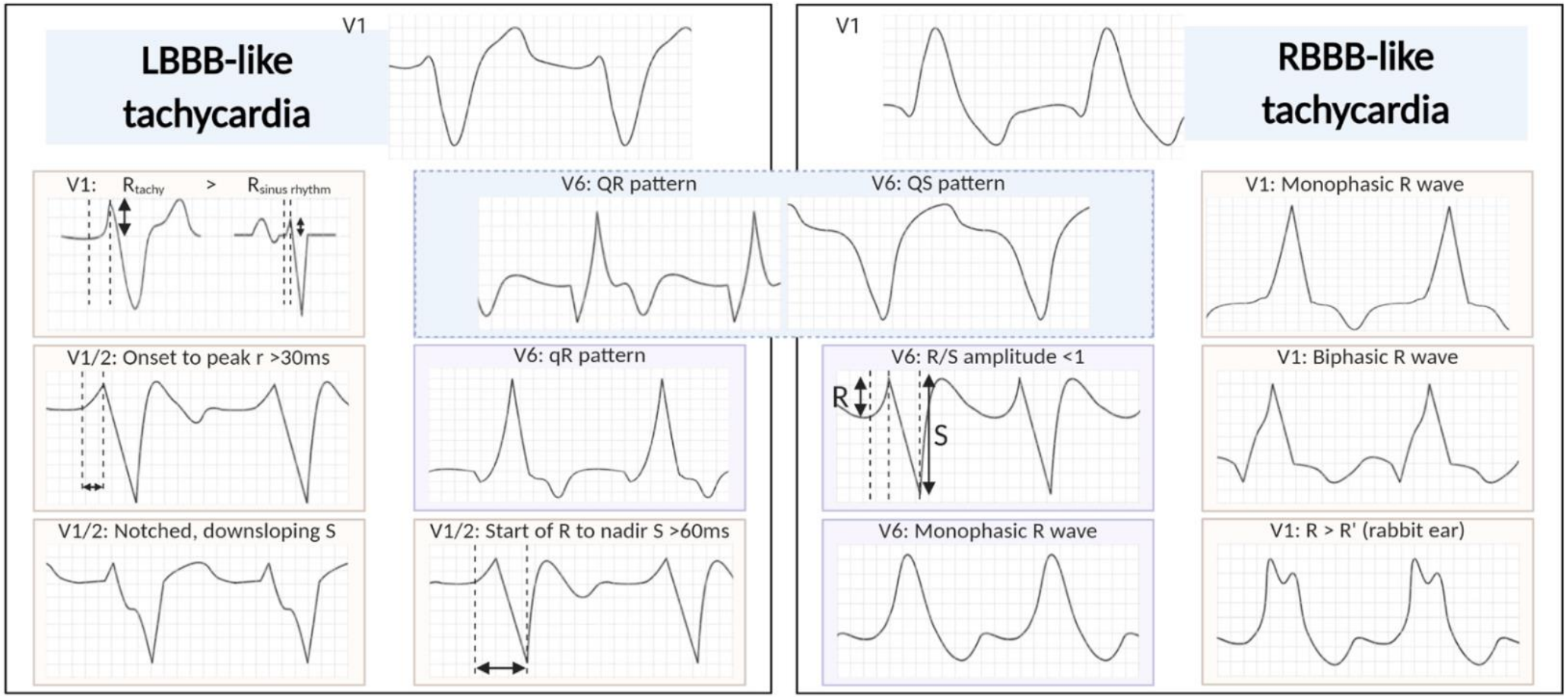


Figure 3 Atypical electrocardiographic features of right bundle branch block (RBBB) and left bundle branch block (LBBB) that are suggestive of ventricular tachycardia.

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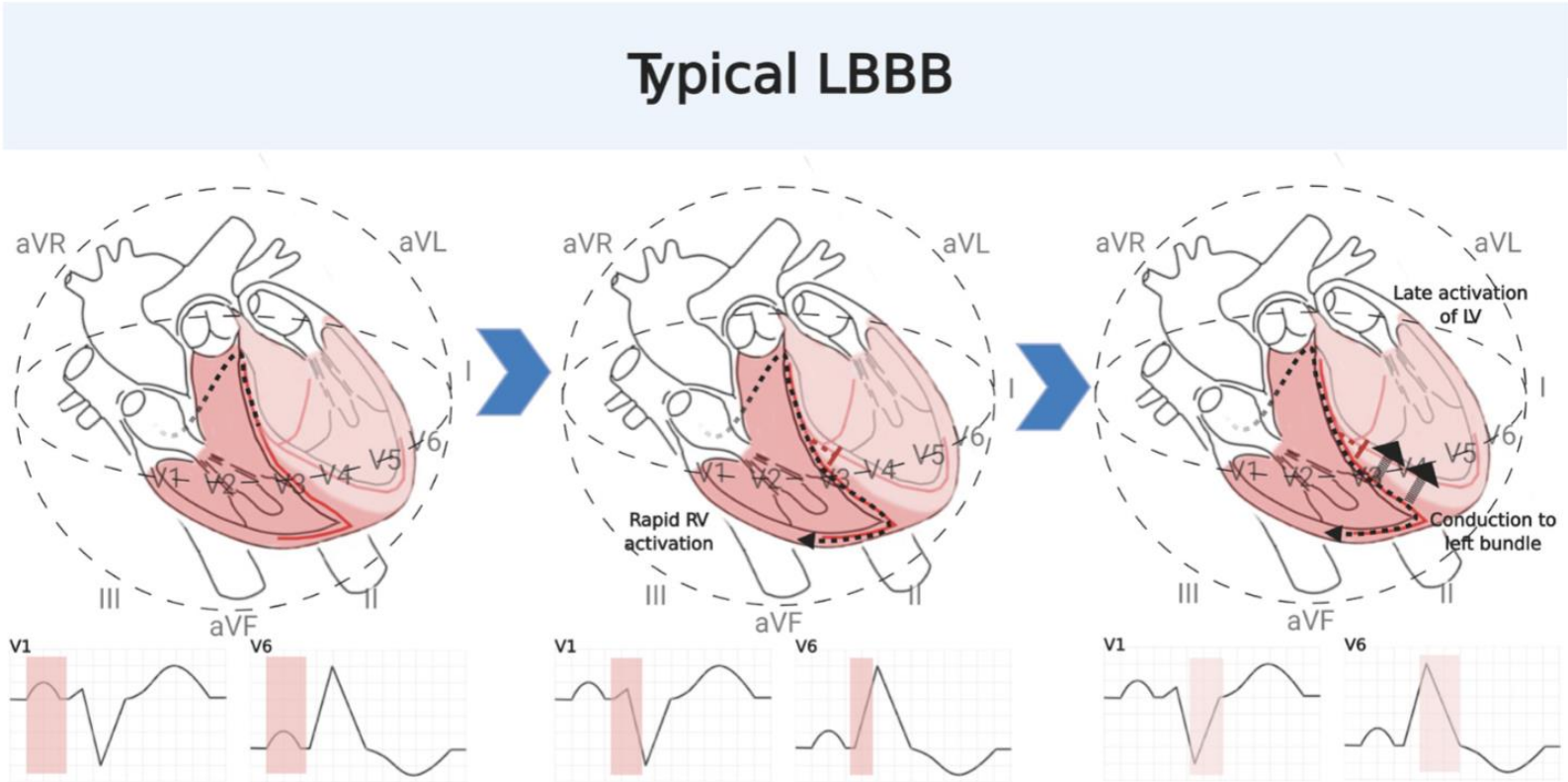
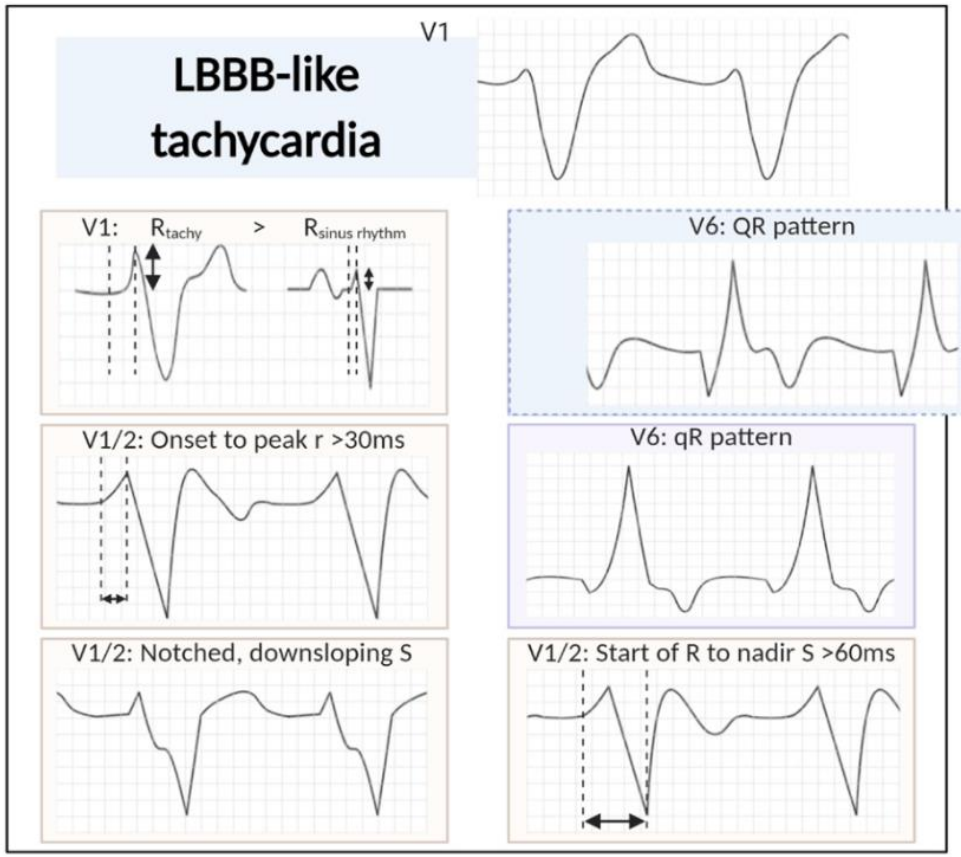


Conduction through AVN and His bundle

Rapid initial activation of the RV → small r in V1, small s in V6

Late activation of LV and left bundle → S in V1, R in V6

Patterns that suggest VT



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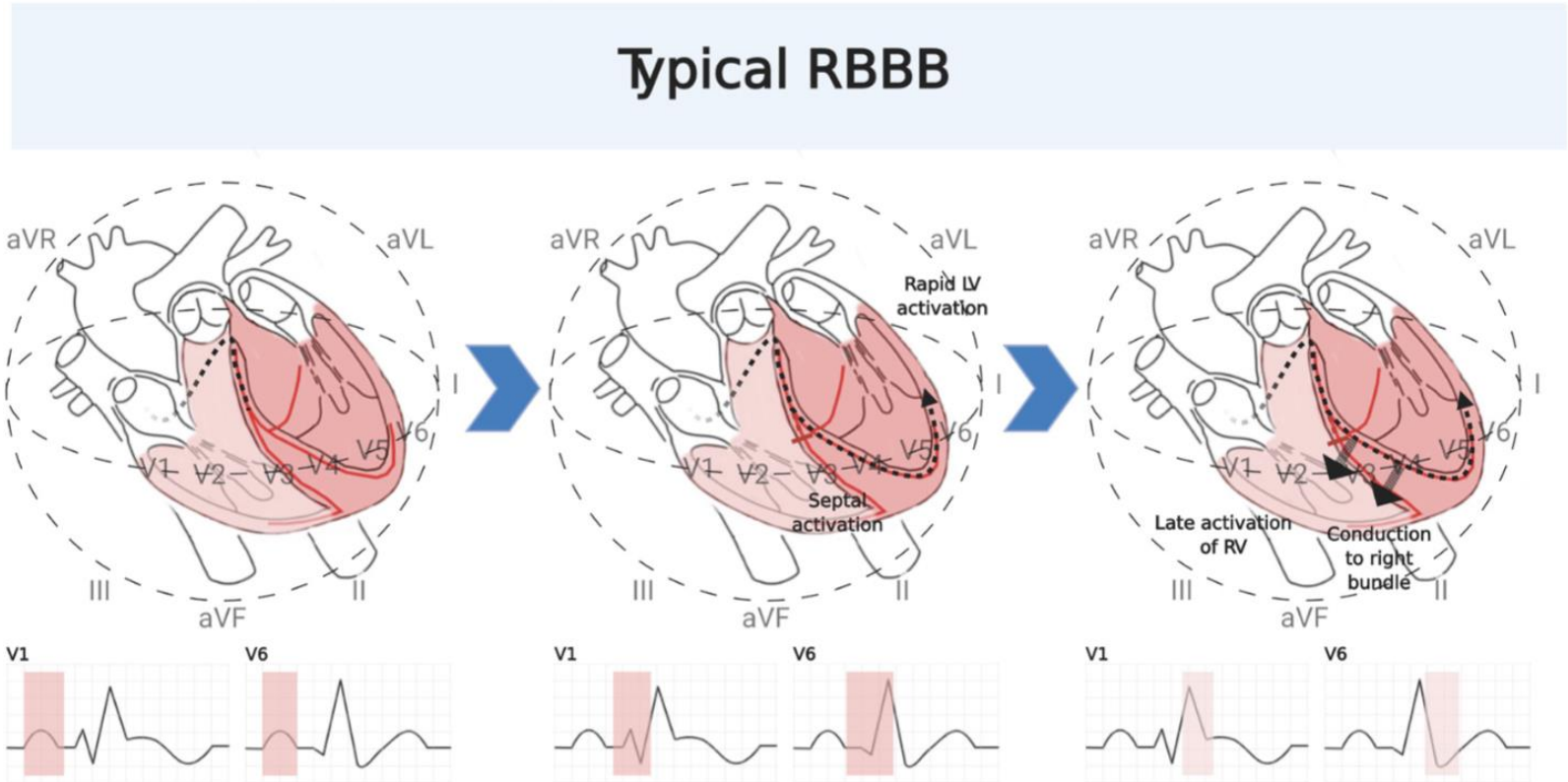
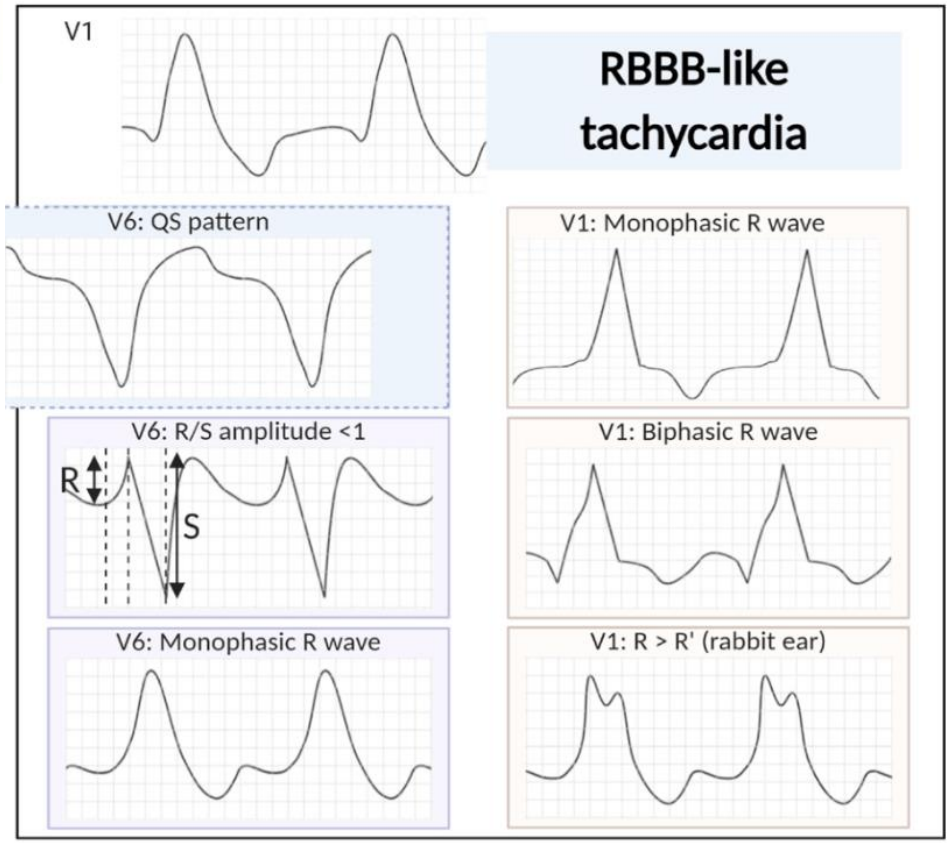


Conduction through AVN and His bundle

Rapid initial activation of the septum and LV → small rs complex in V1, tall R in V6

Late activation of RV and right bundle → late R in V1, s in V6

Patterns that suggest VT



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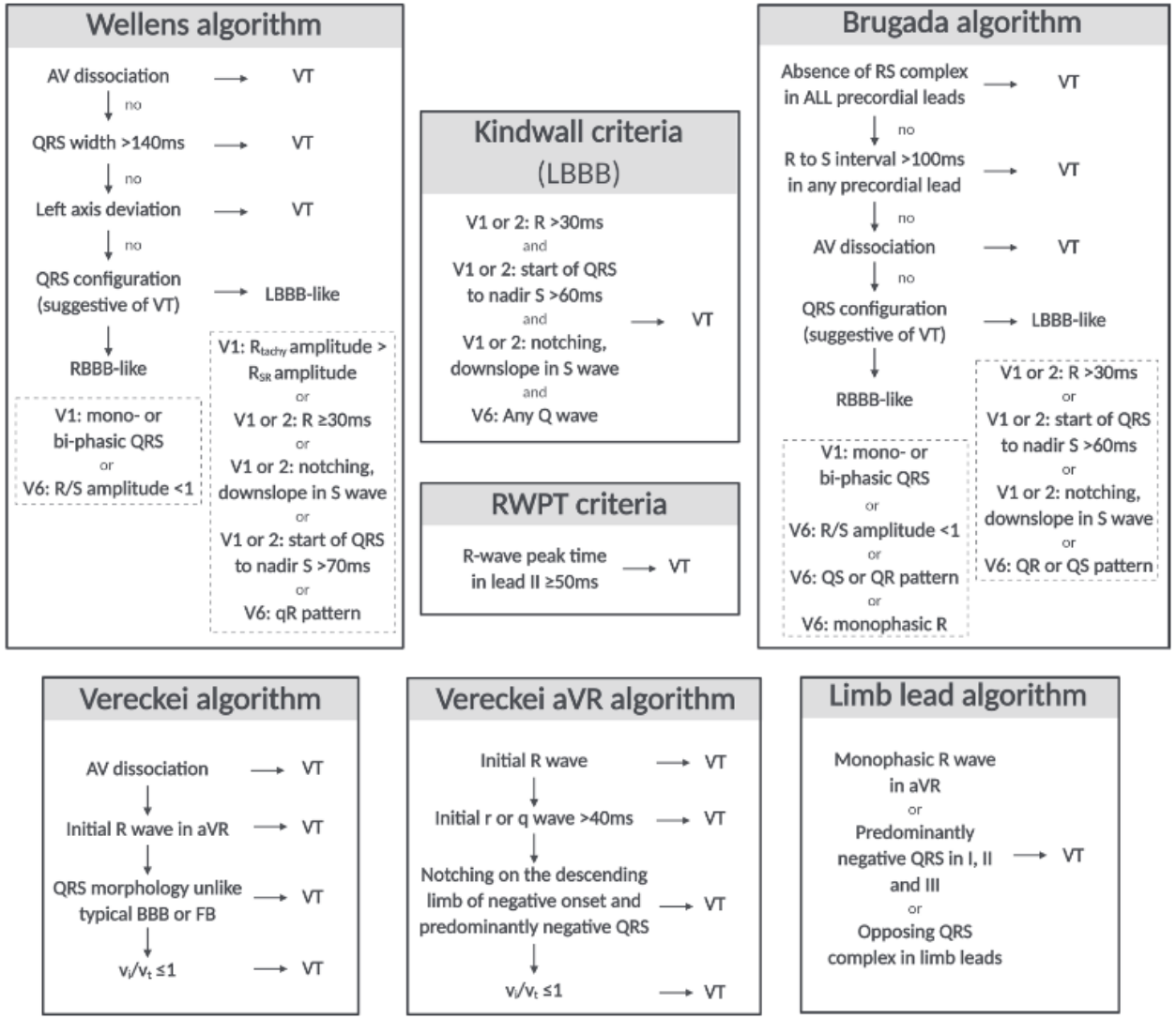


Figure 8 Wide complex tachycardia algorithms to distinguish between ventricular and supraventricular tachycardias. All the algorithms rely on specific features of ventricular tachycardia. If none are present, a diagnosis of supraventricular tachycardia is most likely. AV, atrioventricular; BBB, bundle branch block; FB, fascicular block; LBBB, left bundle branch block; RBBB, right bundle branch block; RWPT, R-wave peak time; VT, ventricular tachycardia.

Ding, Wern Yew, and Saagar Mahida. 2021. "Wide Complex Tachycardia: Differentiating Ventricular Tachycardia from Supraventricular Tachycardia." *Heart* 107 (24): 1995–2003.

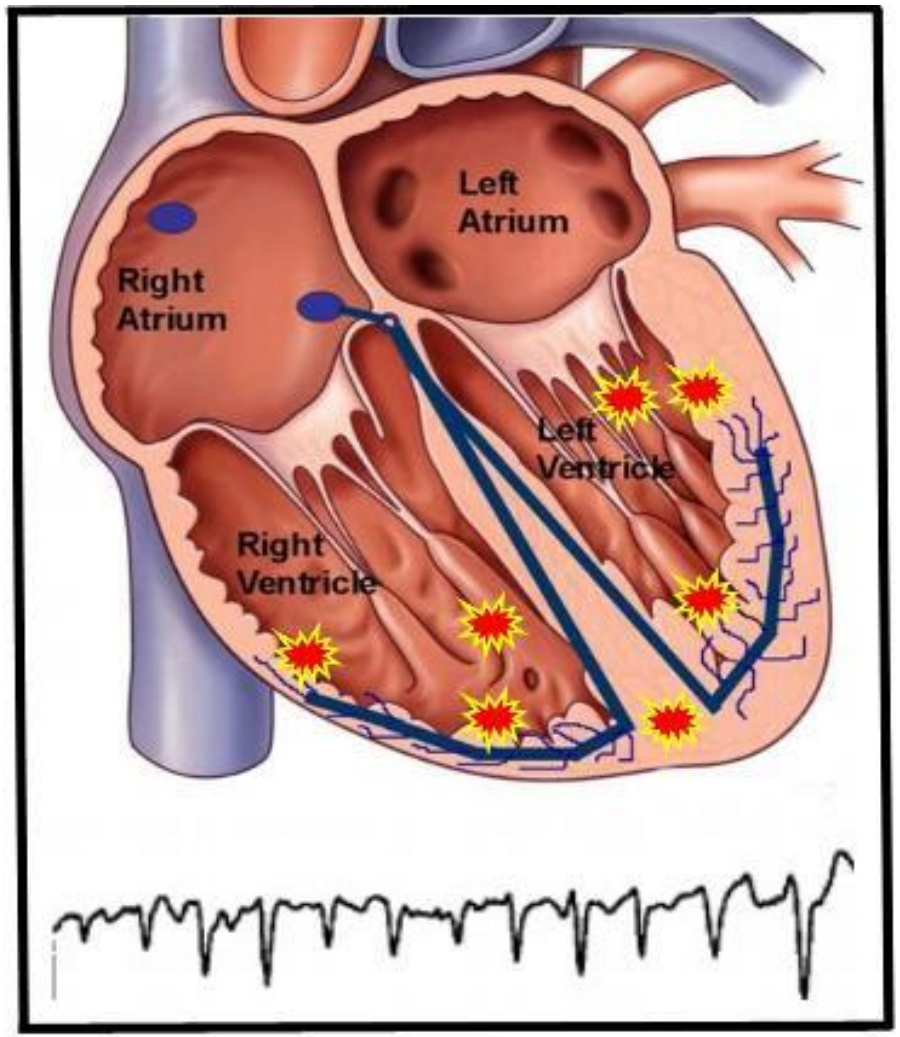
Long term management

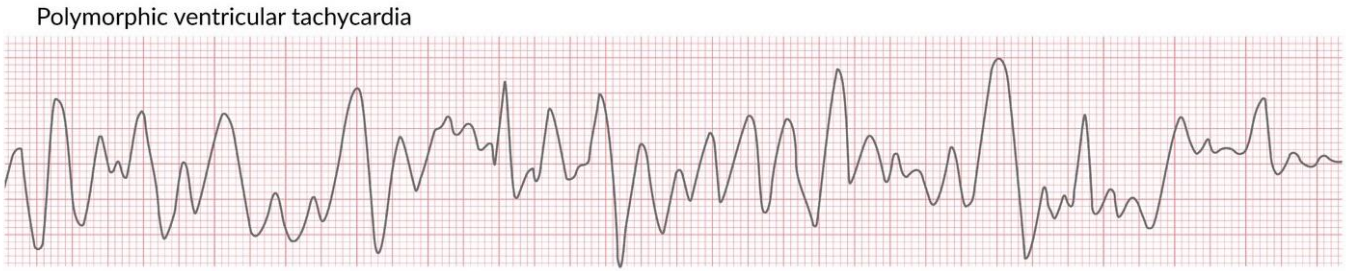
- Antiarrhythmic agents
- Nodal blocking agents
- Catheter ablation

DDx of wide complex irregular tachycardia

- Atrial fibrillation, flutter, or focal AT with aberrancy
- Atrial fibrillation, flutter, or focal AT with anterograde accessory pathway conduction (WPW)
- Polymorphic VT
- Ventricular fibrillation

Polymorphic VT





- Alternative QRS patterns reflecting various sites of ventricular activation
- Usually unstable or pulseless requiring immediate DCCV/defibrillation

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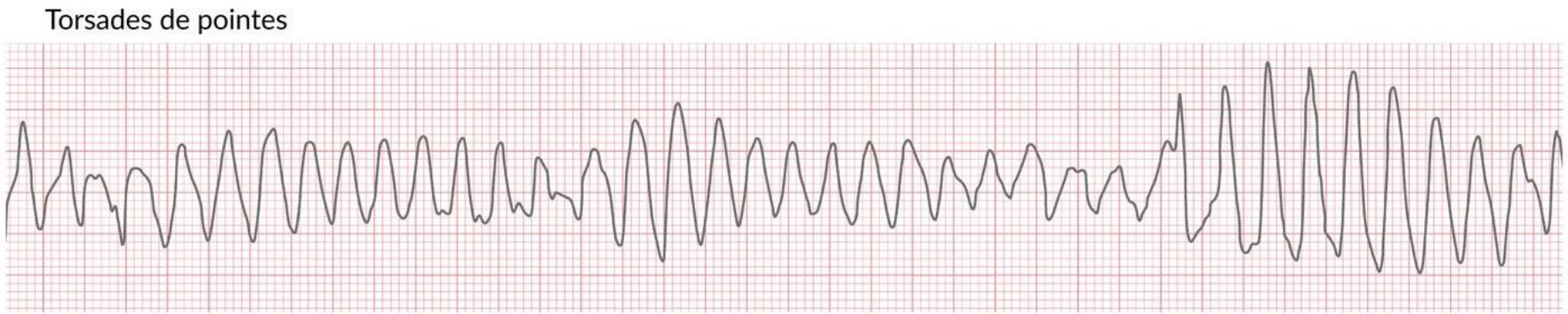
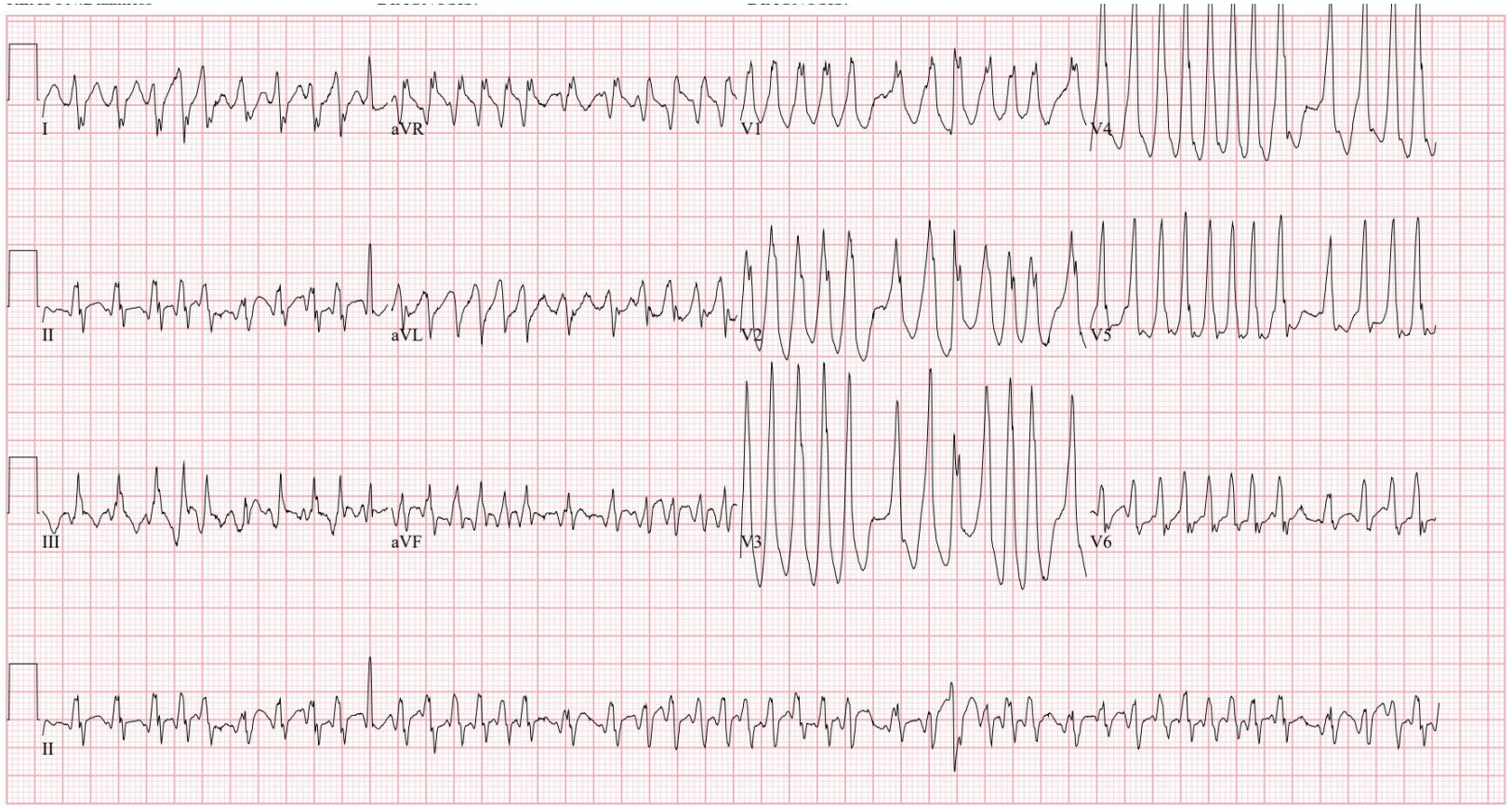
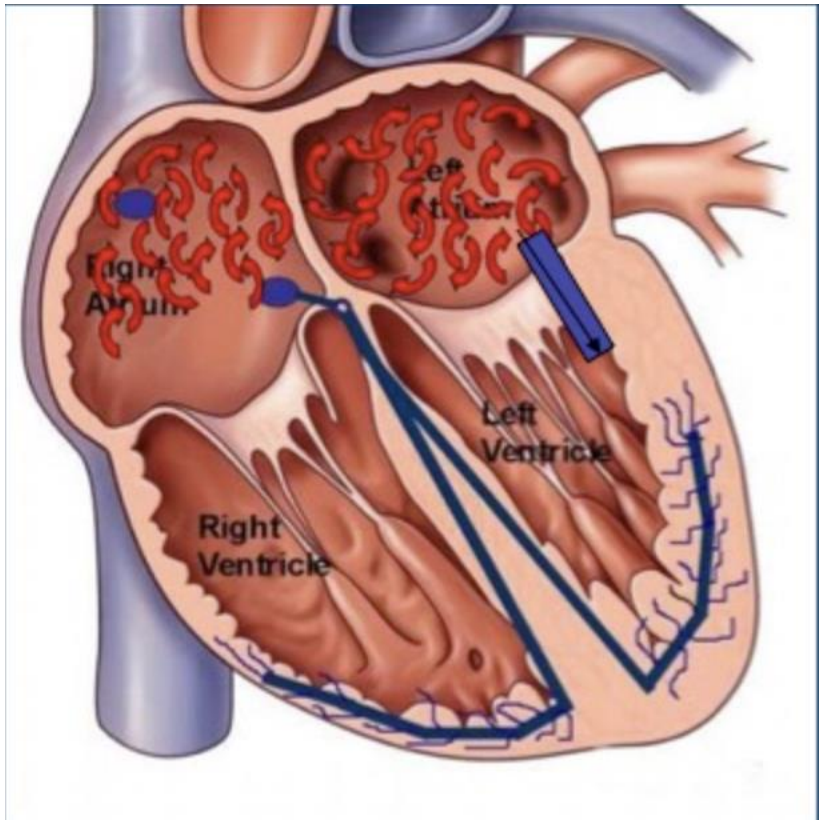
Torsades de pointes

- Usually transient salvos in setting of QT prolongation
- ”Twisting on a point”
- Unstable and sustained → defibrillation
- Treatment – IV magnesium, potassium, chronotropic agents or temporary pacing



Atrial fibrillation with pre-excitation





AF with simultaneous conduction down AV node and a rapidly conducting accessory pathway
If stable → procainamide or ibutilide

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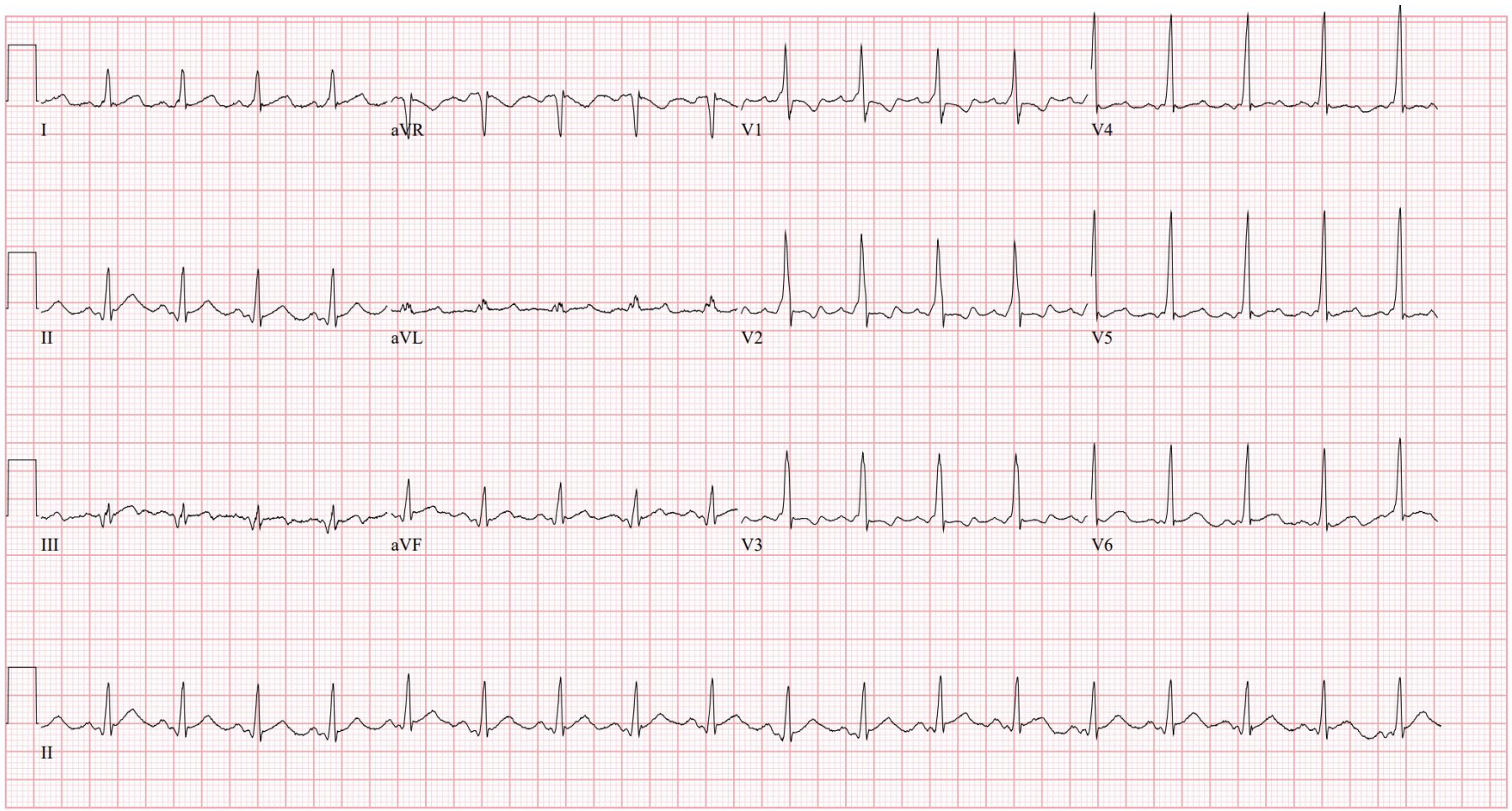
Follow-up ECG



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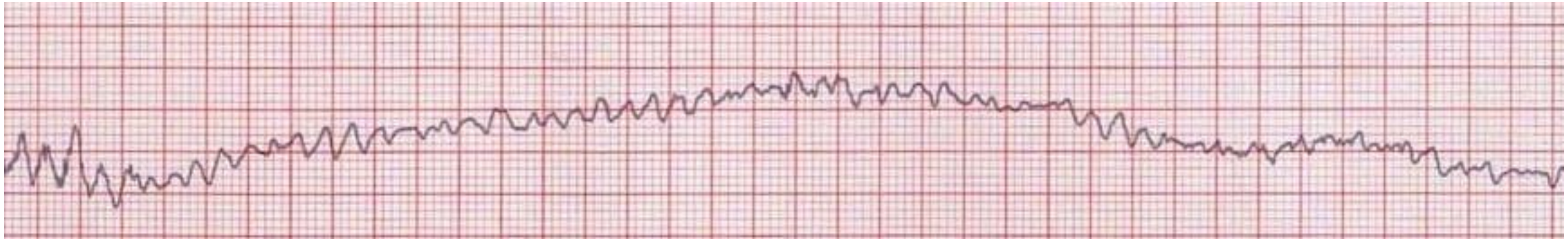
Ventricular fibrillation

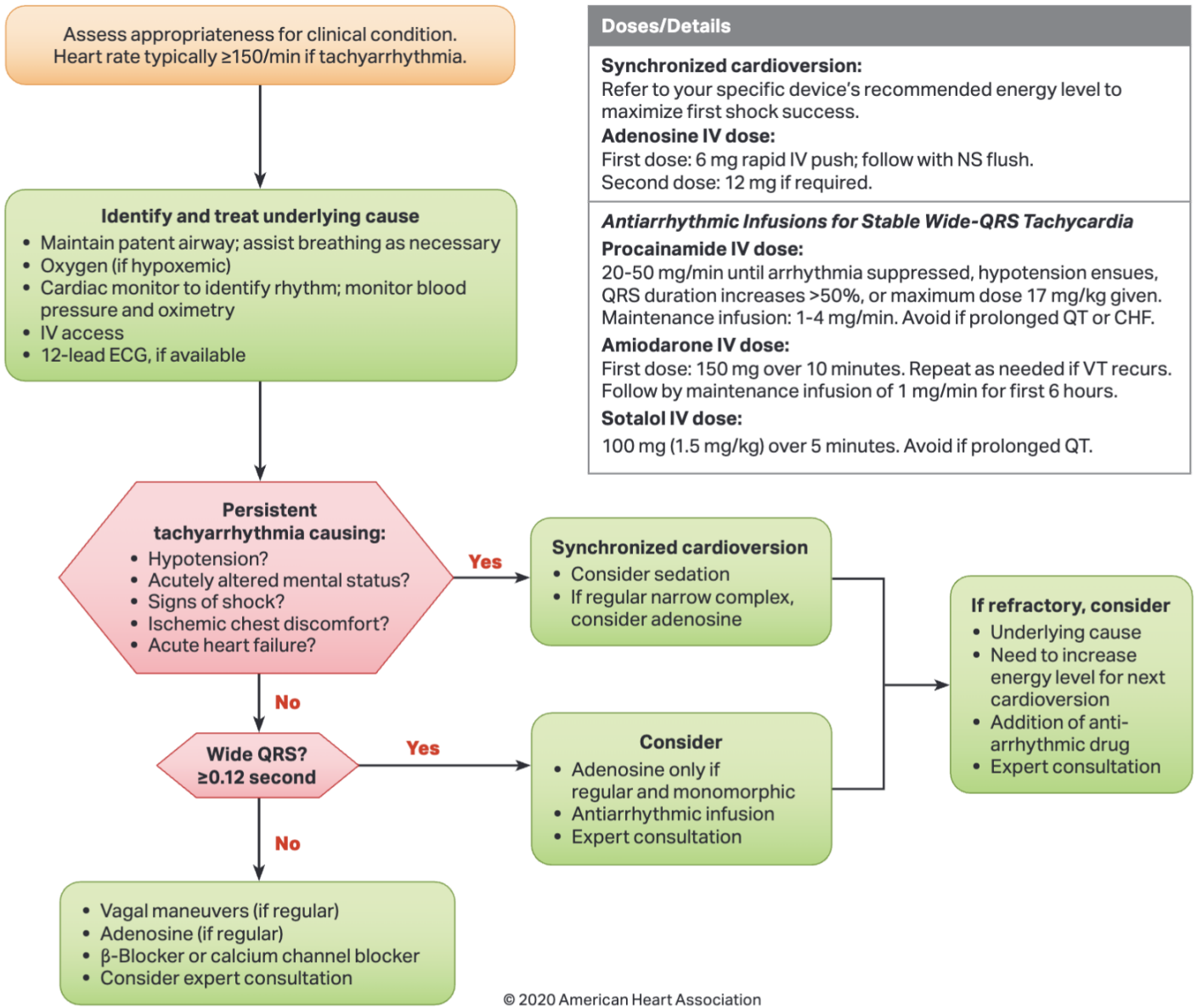
- Absence of identifiable P waves or QRS complexes
- Pulseless rhythm that requires immediate CPR and defibrillation



Wide irregular tachycardia

- Avoid adenosine or nodal blocking agents
  - Will not be effective for any of these rhythms
  - May have negative hemodynamic effects
- Urgent cardioversion if unstable
- Defibrillation if pulseless
- PMVT or VF → amiodarone usually initial choice
- Torsades → IV magnesium, potassium repletion, chronotropic agents, possibly pacing
- Pre-excited atrial fibrillation → procainamide or ibutilide

Adult Tachycardia With a Pulse Algorithm



Is the patient hemodynamically stable?
Is the QRS narrow or wide?
Is the rhythm regular or irregular?

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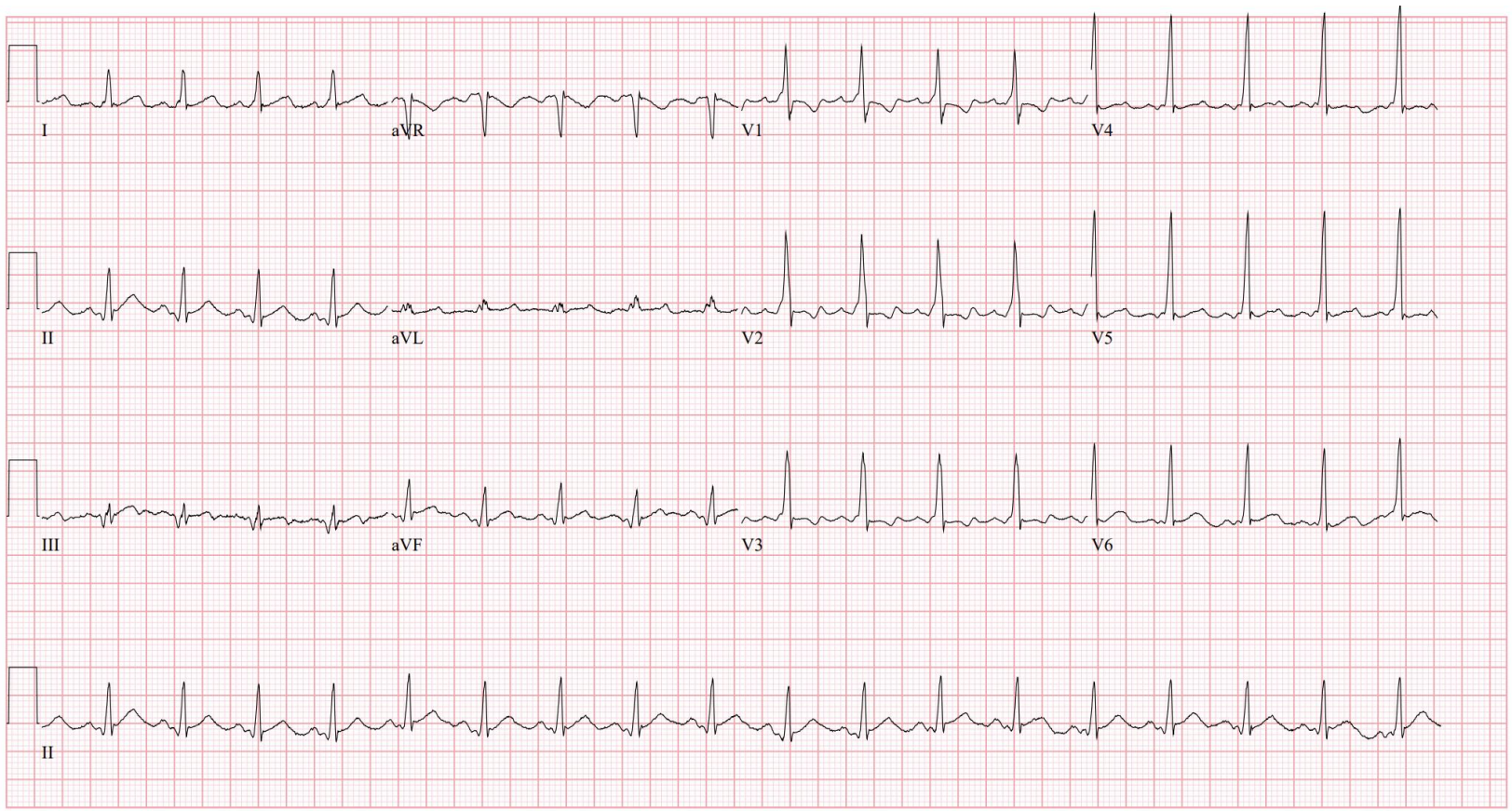
Questions?



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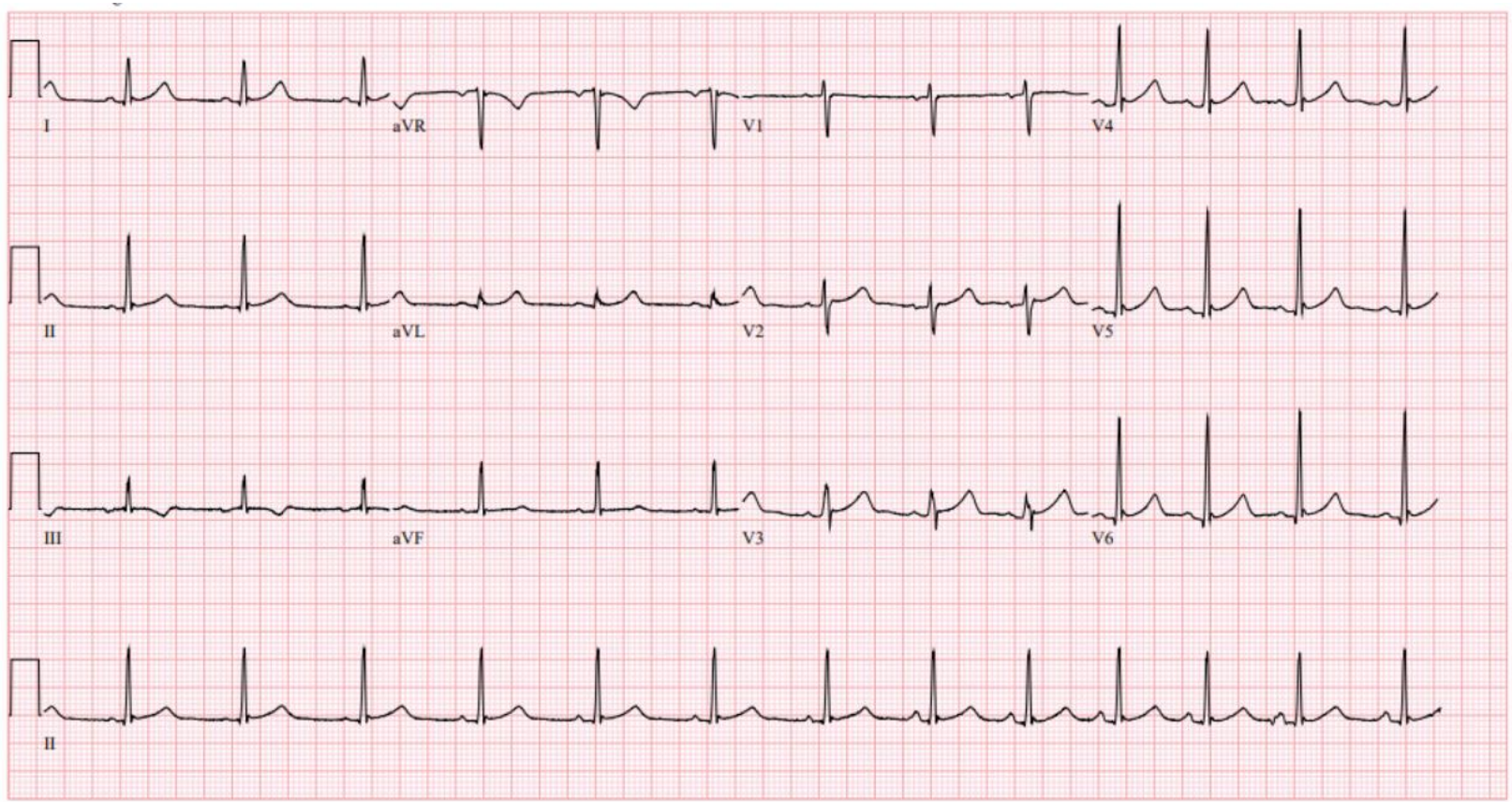
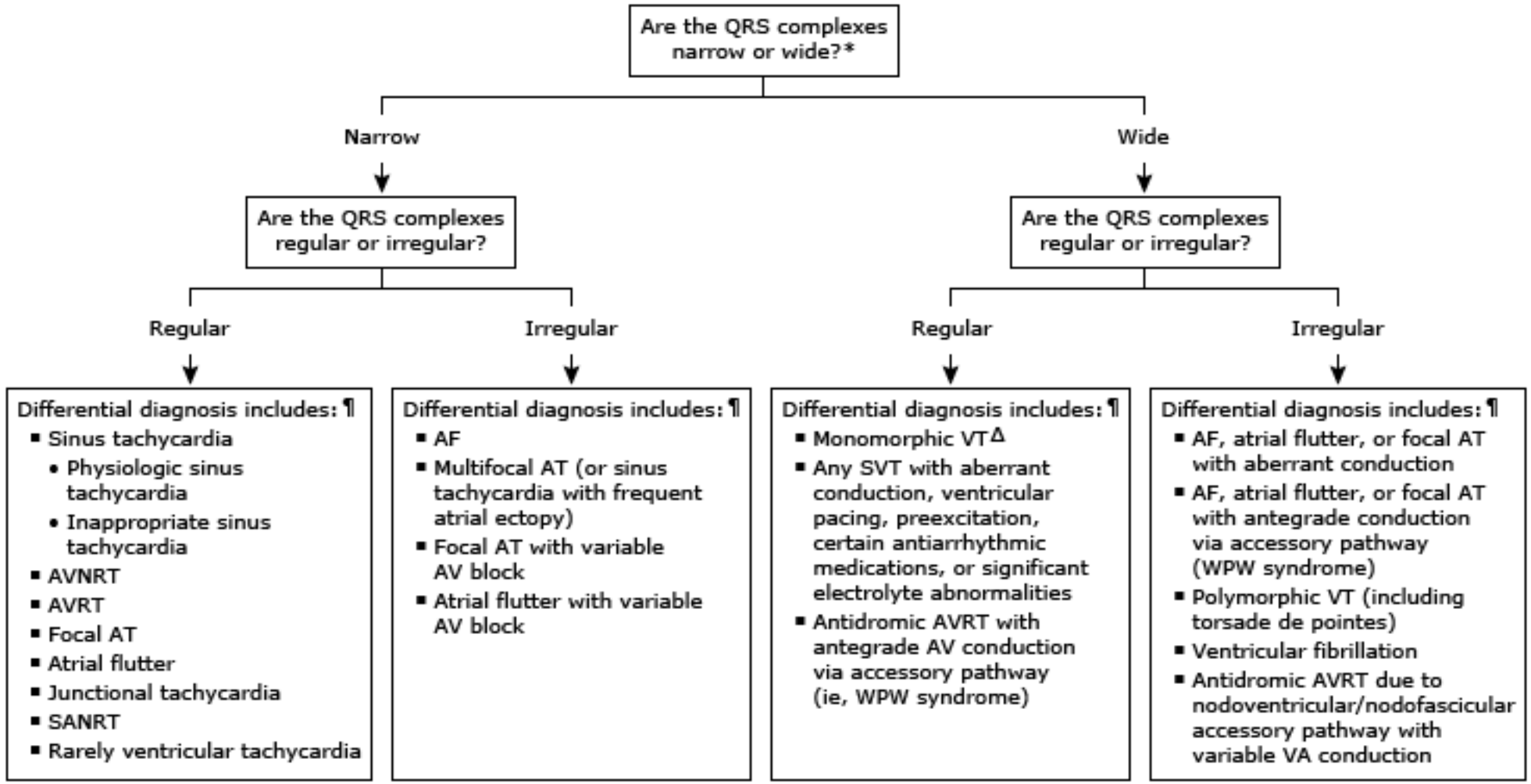


Differential diagnosis



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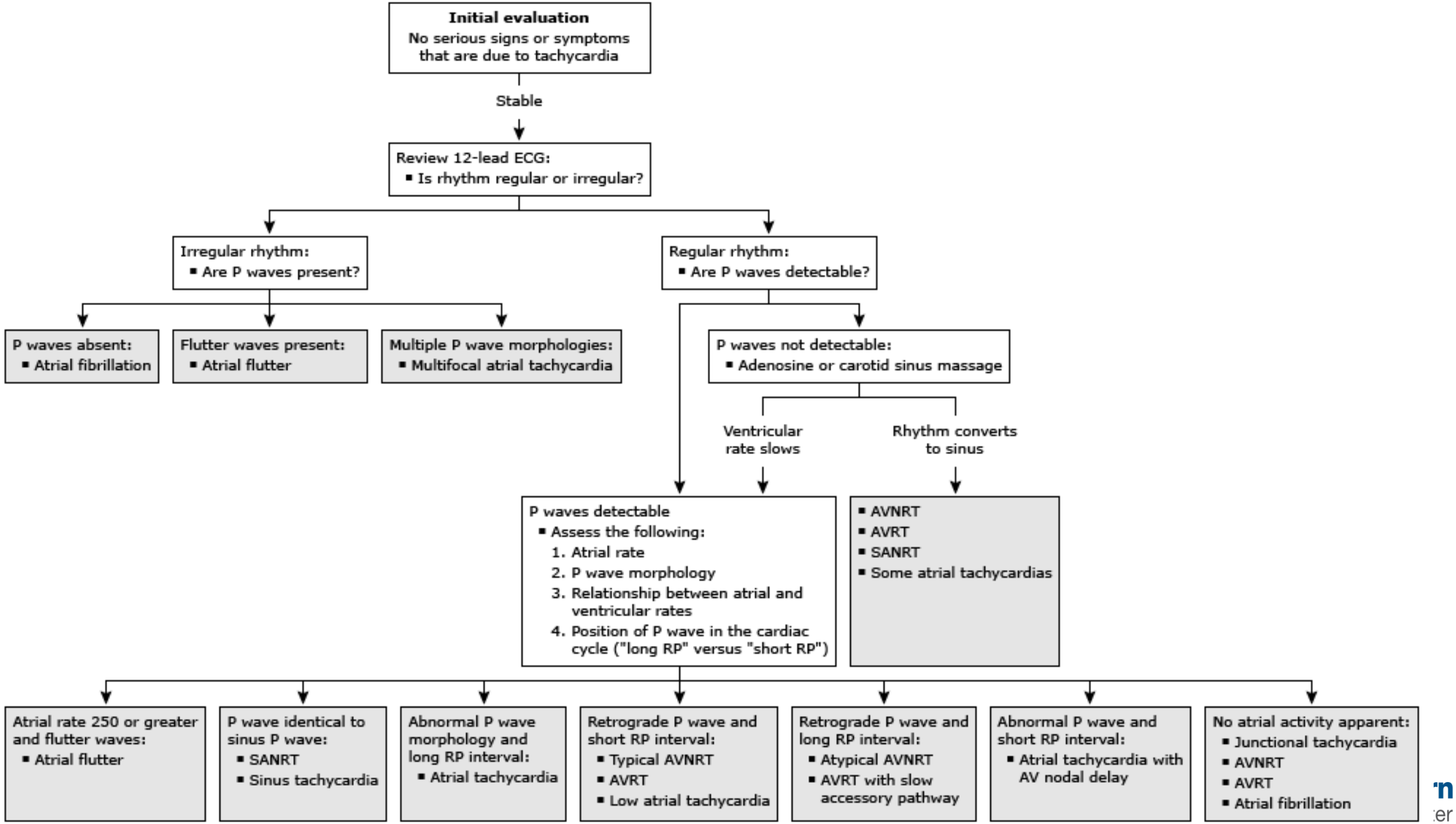
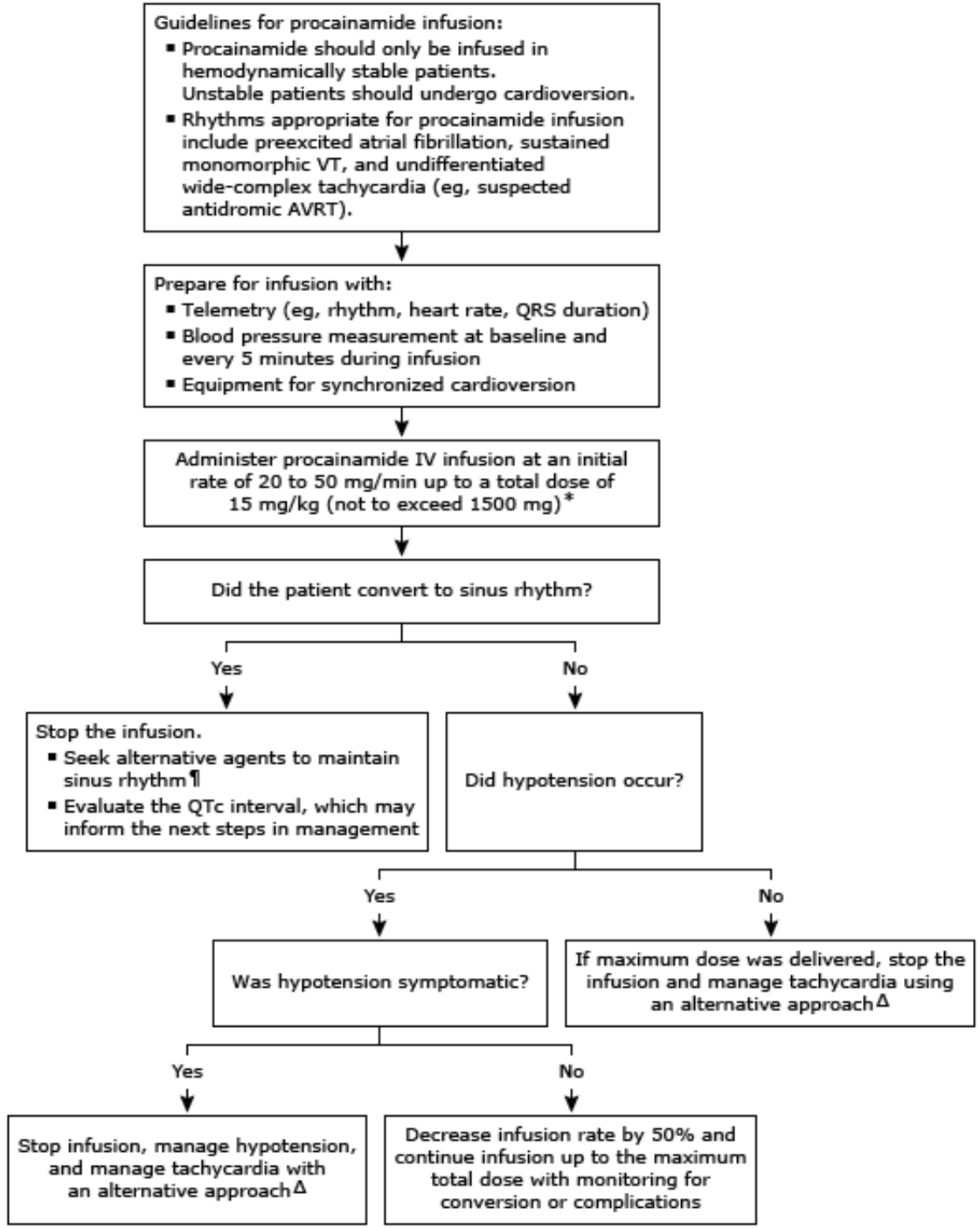

Initial evaluation
No serious signs or symptoms that are due to tachycardia
Stable
Review 12-lead ECG:
Is rhythm regular or irregular?
Irregular rhythm:
Are P waves present?
Regular rhythm:
Are P waves detectable?
P waves absent:
Atrial fibrillation
Flutter waves present:
Atrial flutter
Multiple P wave morphologies:
Multifocal atrial tachycardia
P waves not detectable:
Adenosine or carotid sinus massage
Ventricular rate slows
Rhythm converts to sinus
P waves detectable
Assess the following:
1. Atrial rate
2. P wave morphology
3. Relationship between atrial and ventricular rates
4. Position of P wave in the cardiac cycle ("long RP" versus "short RP")
AVNRT
AVRT
SANRT
Some atrial tachycardias
Atrial rate 250 or greater and flutter waves:
Atrial flutter
P wave identical to sinus P wave:
SANRT
Sinus tachycardia
Abnormal P wave morphology and long RP interval:
Atrial tachycardia
Retrograde P wave and short RP interval:
Typical AVNRT
AVRT
Low atrial tachycardia
Retrograde P wave and long RP interval:
Atypical AVNRT
AVRT with slow accessory pathway
Abnormal P wave and short RP interval:
Atrial tachycardia with AV nodal delay
No atrial activity apparent:
Junctional tachycardia
AVNRT
AVRT
Atrial fibrillation



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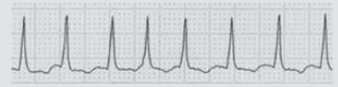
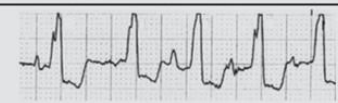
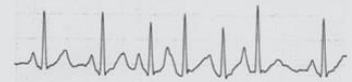
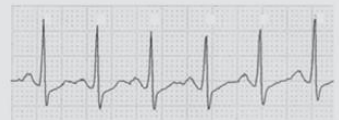
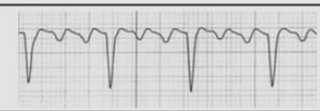
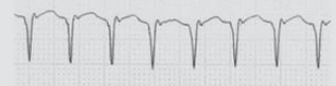
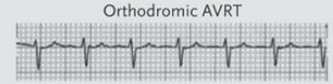
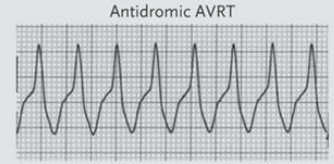
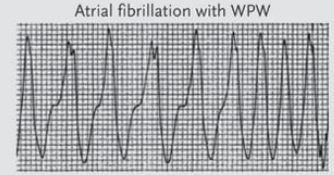
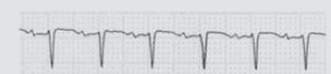
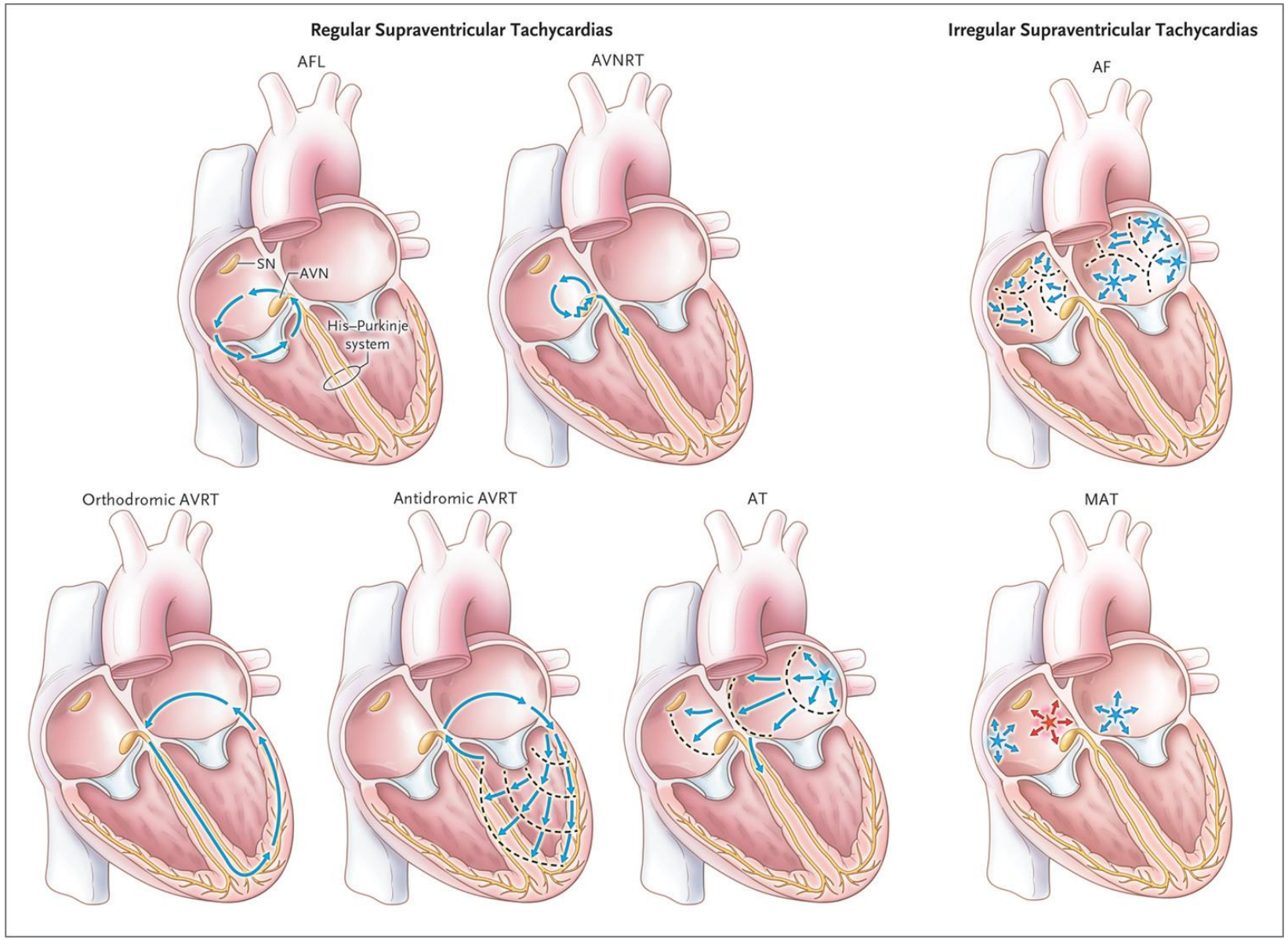
SVT	Underlying Causes	Regularity	Rate (beats/min)	Onset	Atrial Activity and P:QRS Relationship	Response to Adenosine	Electrocardiogram
Atrial fibrillation	Cardiac disease, pulmonary disease, pulmonary embolism, hyperthyroidism, postoperative	Irregular	100-220	Sudden or gradual (if in chronic atrial fibrillation)	Fibrillatory waves, no relationship to QRS	Transient slowing of ventricular rate	
Multifocal atrial tachycardia	Pulmonary disease, theophylline therapy	Irregular	100-150	Gradual	Changing P morphologic features before QRS	None	
Frequent atrial premature contractions	Caffeine, stimulants	Irregular	100-150	Gradual	P before QRS	None	
Sinus tachycardia	Sepsis, hypovolemia, anemia, pulmonary embolism, pain, fear, fright, exertion, myocardial ischemia, hyperthyroidism, heart failure	Regular	220 minus the patient's age	Gradual	P before QRS	Transient slowing	
Atrial flutter	Cardiac disease	Regular (occasionally irregular if variable AV conduction)	150	Sudden	Flutter waves, usually 2:1	Transient slowing of ventricular rate	
AV nodal reentrant tachycardia	None	Regular	150-250	Sudden	No apparent atrial activity or R' at termination of QRS	Termination of tachycardia	
AV reciprocating tachycardia	Rarely, Ebstein's anomaly	Regular	150-250	Sudden	In narrow complex, P after QRS; in wide complex, P rarely observed; in irregular rhythm (atrial fibrillation), no apparent P wave	Termination of tachycardia	Orthodromic AVRT  Antidromic AVRT  Atrial fibrillation with WPW 
Atrial tachycardia	Cardiac disease, pulmonary disease	Regular	150-250	Sudden	P before QRS	Termination of 60–80%	



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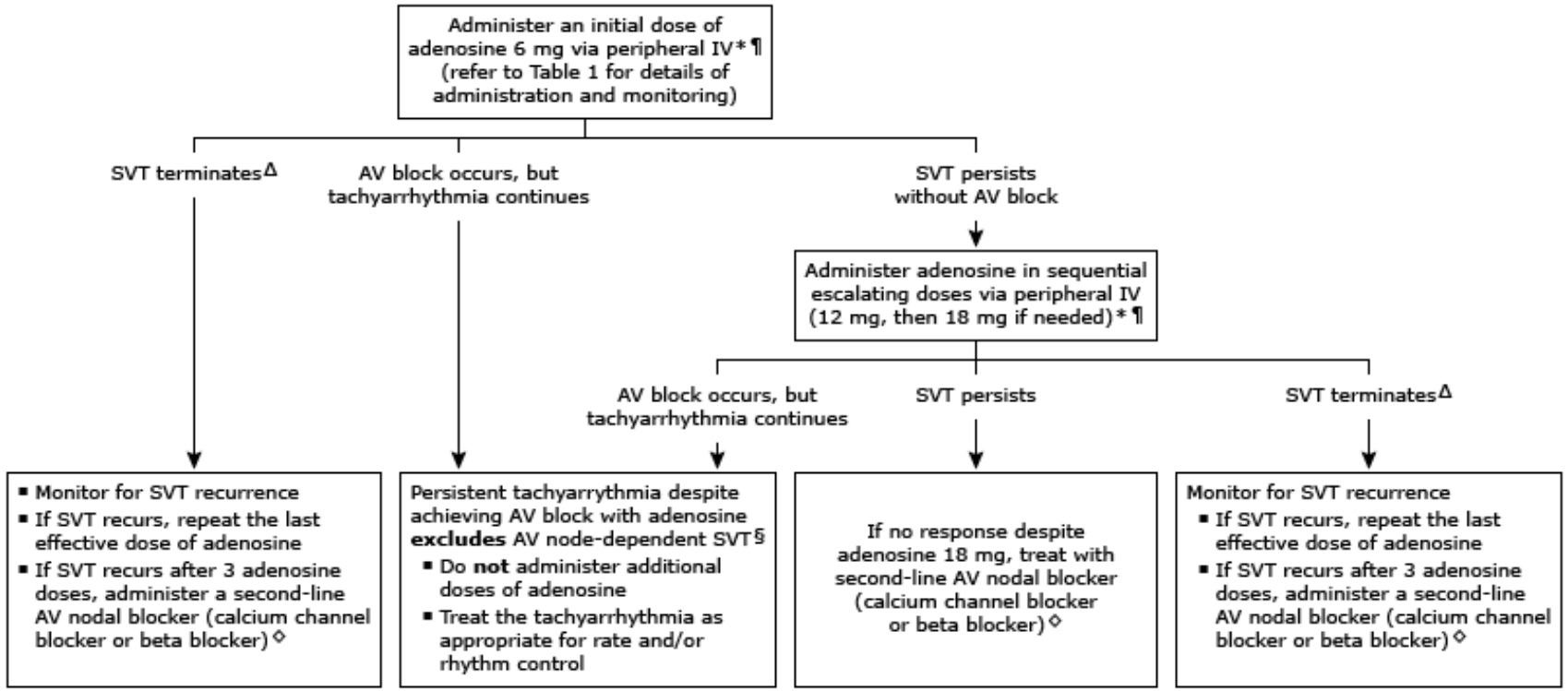

Table 1

Best practices for adenosine administration:

- The patient should be supine during adenosine administration and subsequent monitoring.
- Monitor blood pressure and record a 12-lead ECG during and for 1 to 2 minutes after administration to assess the response to adenosine. If a 12-lead ECG is impractical, record telemetry.
- Administer adenosine in the most proximal available peripheral IV site (eg, antecubutal vein); administration via a hand IV site is generally avoided. If central venous administration of adenosine is performed, lower doses are used than for peripheral administration.¶
- Administer adenosine via a 3-way stopcock, with 1 port leading to the patient, 1 port for the adenosine dose, and 1 port for normal saline flush (10 to 20 mL). Both the adenosine and normal saline syringes should be attached before adenosine is given.
- The IV adenosine dose is pushed over 1 to 2 seconds, immediately followed by turning the stopcock for normal saline flush over 1 to 2 seconds.