

Tachyarrhythmias

Nimesh Patel, M.D.

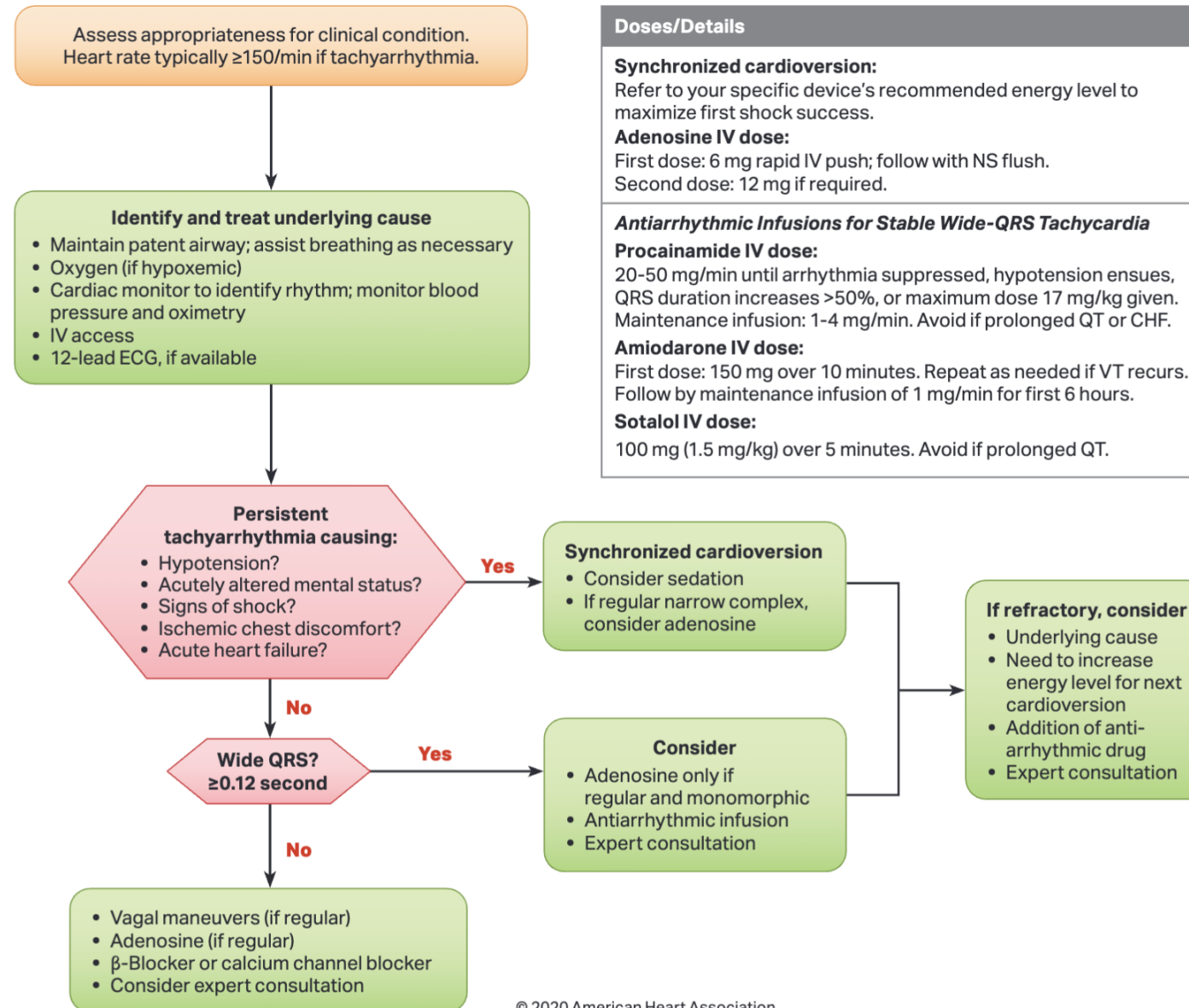
Assistant Professor of Medicine

Clinical Cardiac Electrophysiology

Outline

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

Adult Tachycardia With a Pulse Algorithm



© 2020 American Heart Association

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

What is a tachyarrhythmia?

- Heart rate > 100 bpm, 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 ⑦urgent cardioversion

Initial tachycardia characterization

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

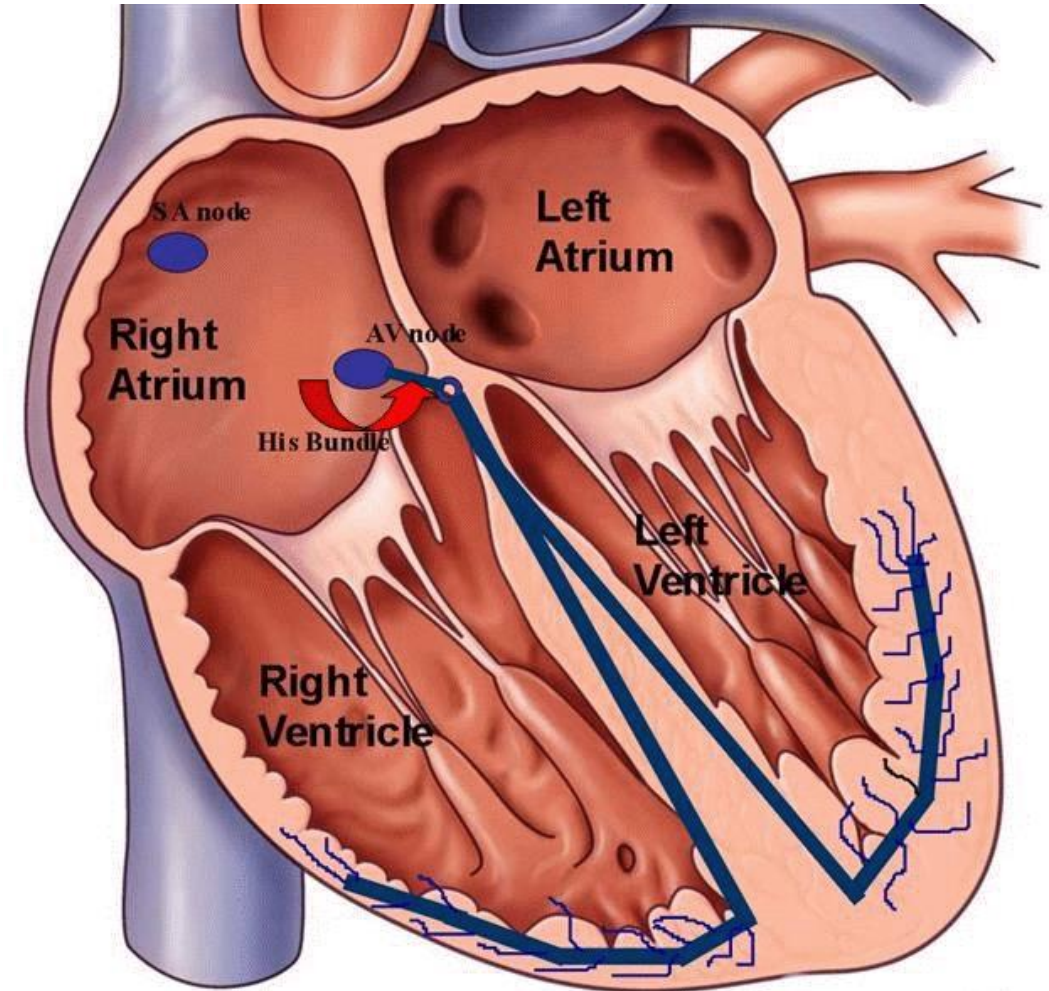
Narrow complex regular tachycardia

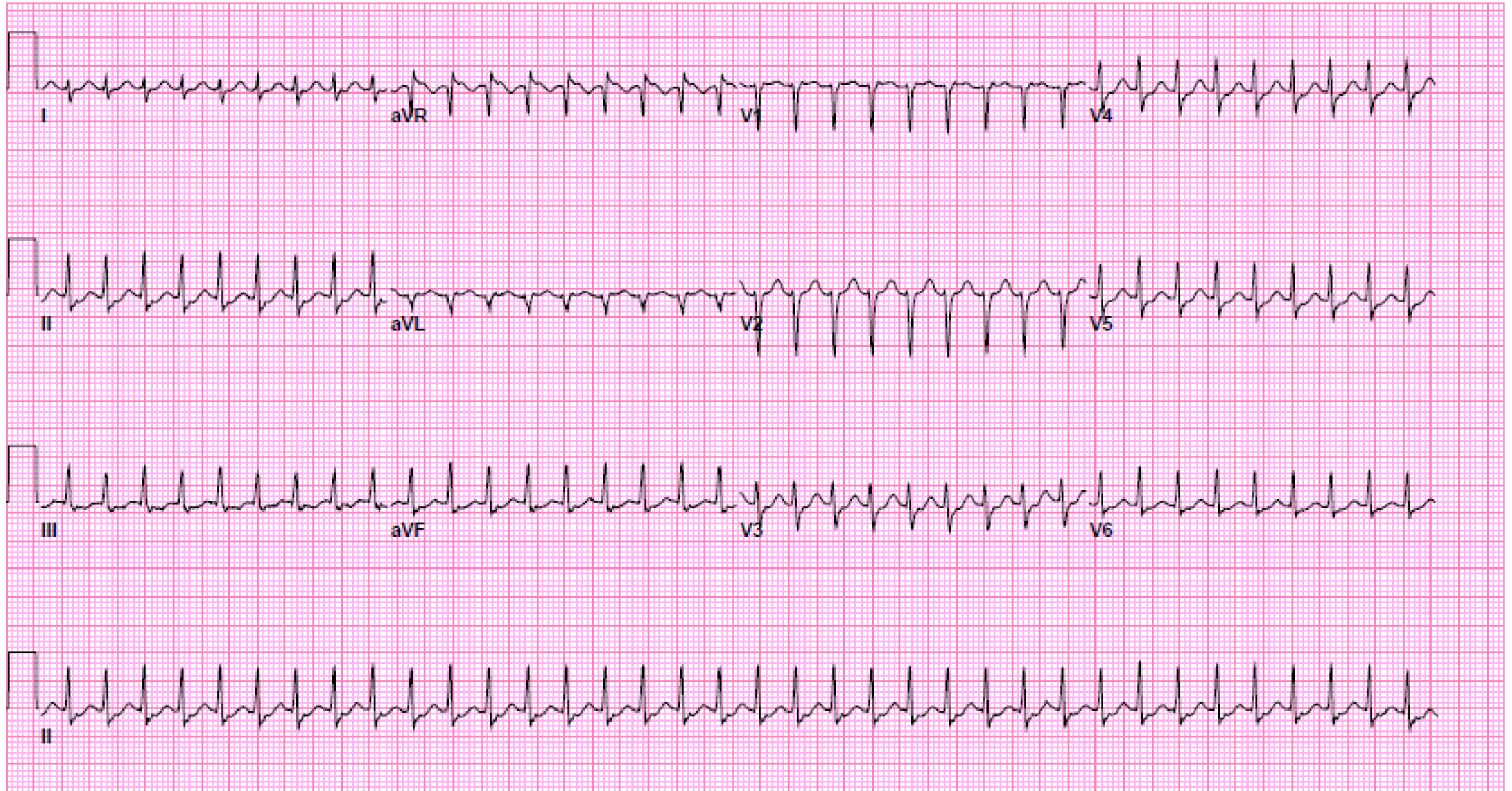
DDx of narrow complex regular tachycardia

- 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

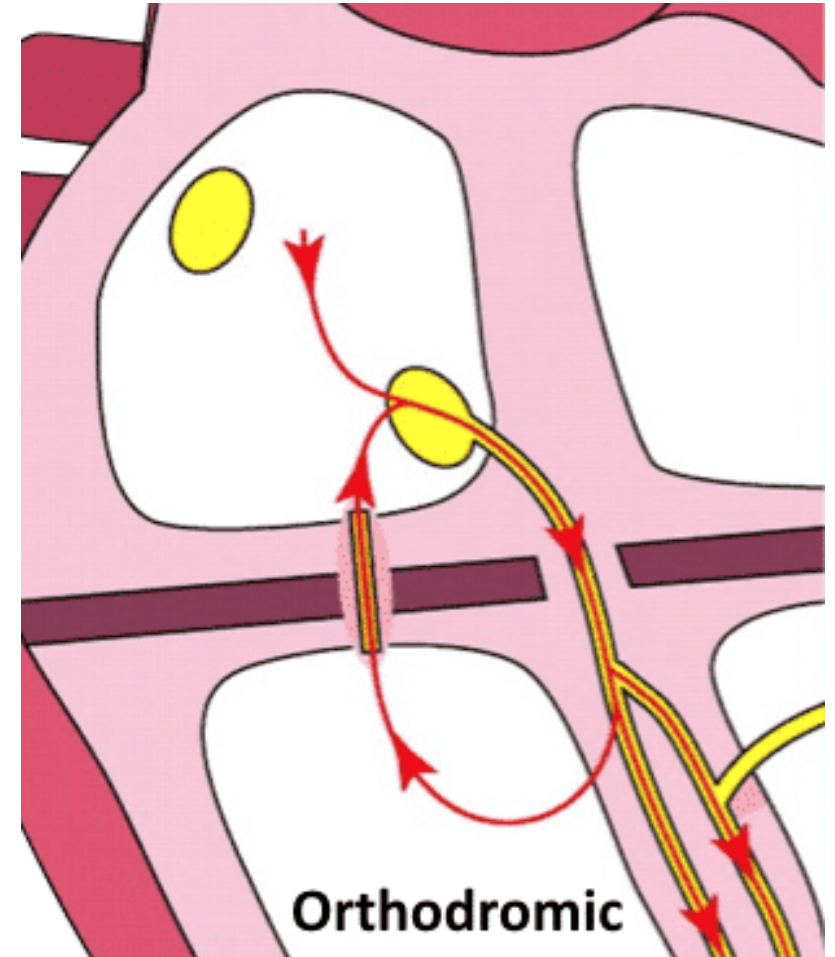
- Re-entry circuit around the AV node
- Usually abrupt onset/offset
- Simultaneous activation of the atrium and ventricle
- Pulsating sensation in the neck from "cannon A waves" with atrium contracting against closed tricuspid valve

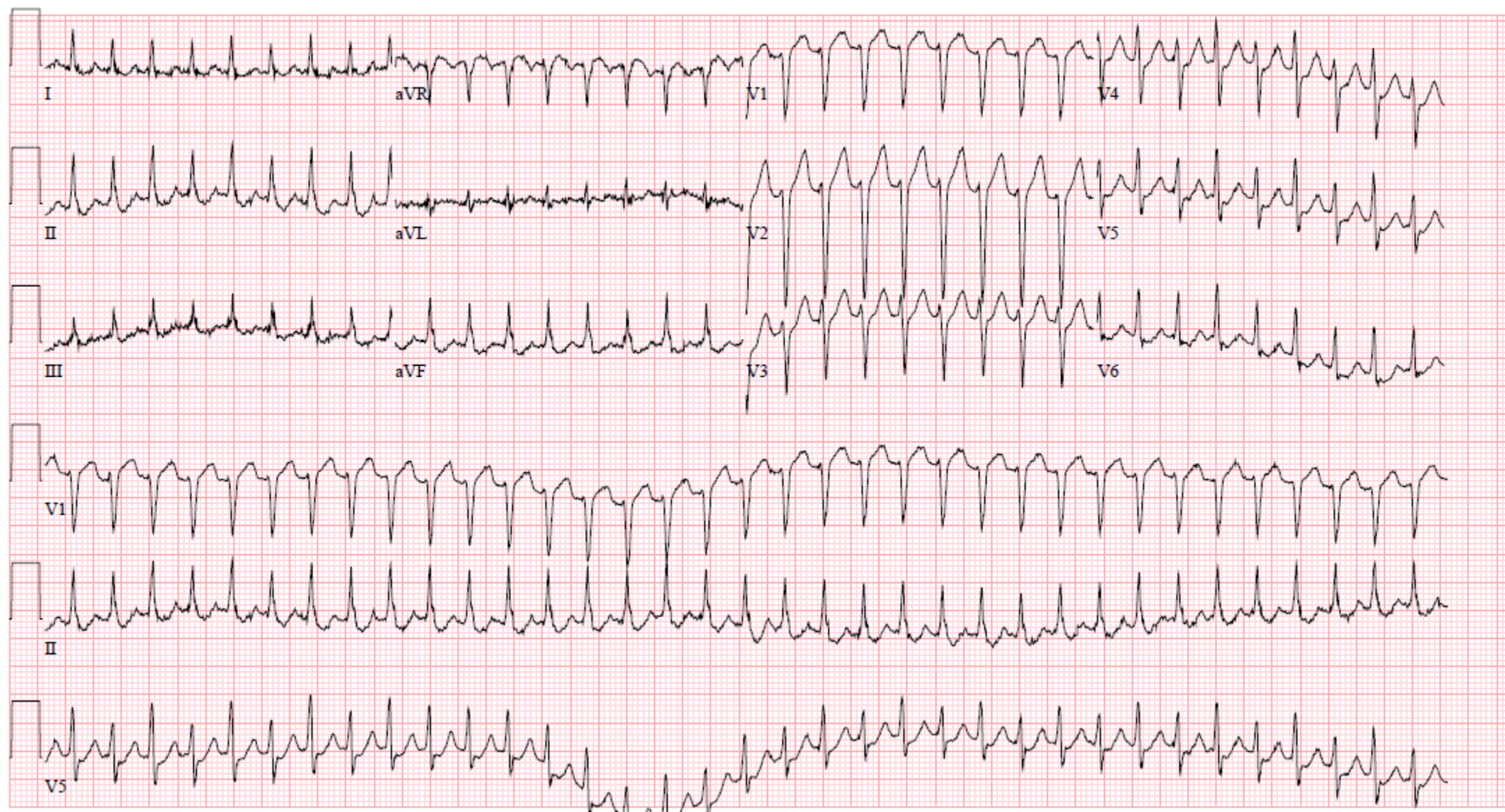




Orthodromic AVRT

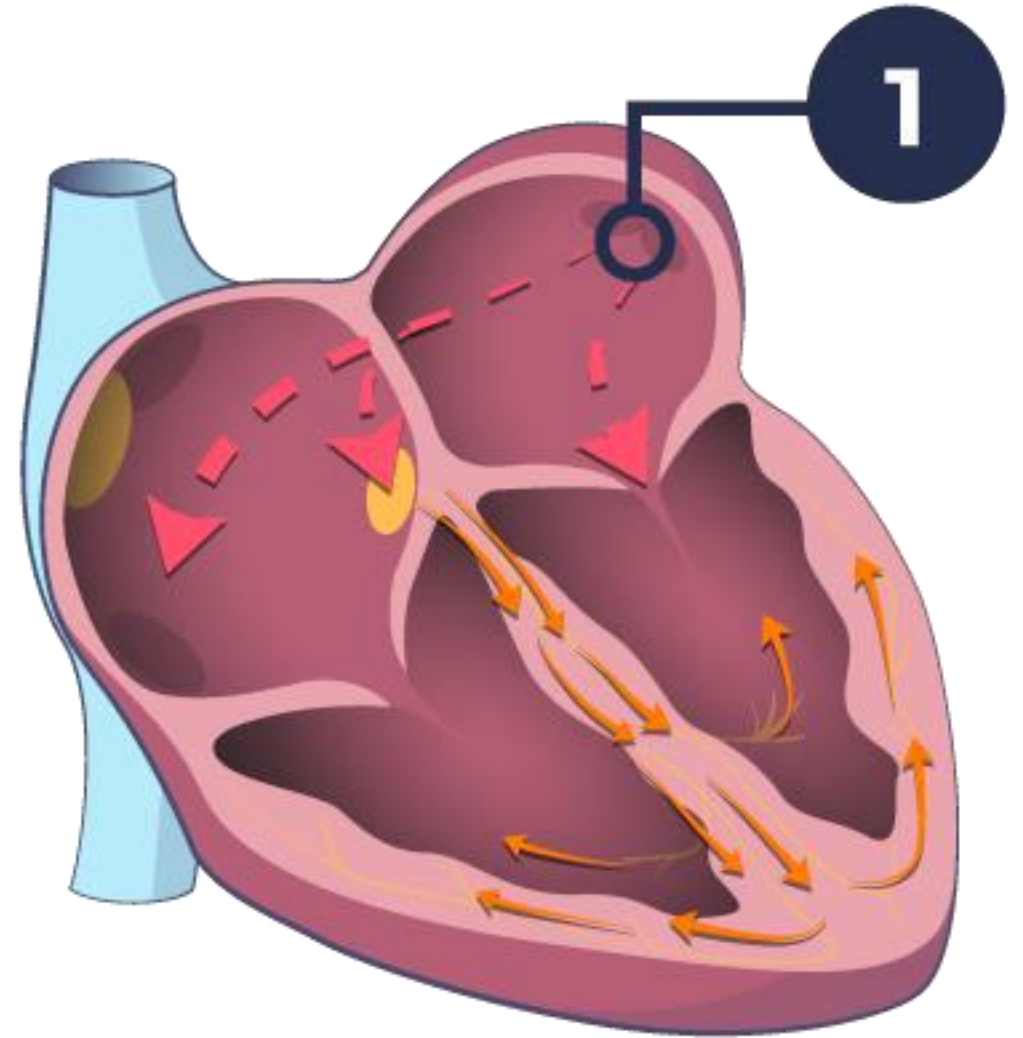
- Reentry
- Anterograde limb: AV node
- Retrograde limb: accessory pathway
- Abrupt onset/offset
- Difficult to distinguish from AVNRT unless baseline WPW pattern is present

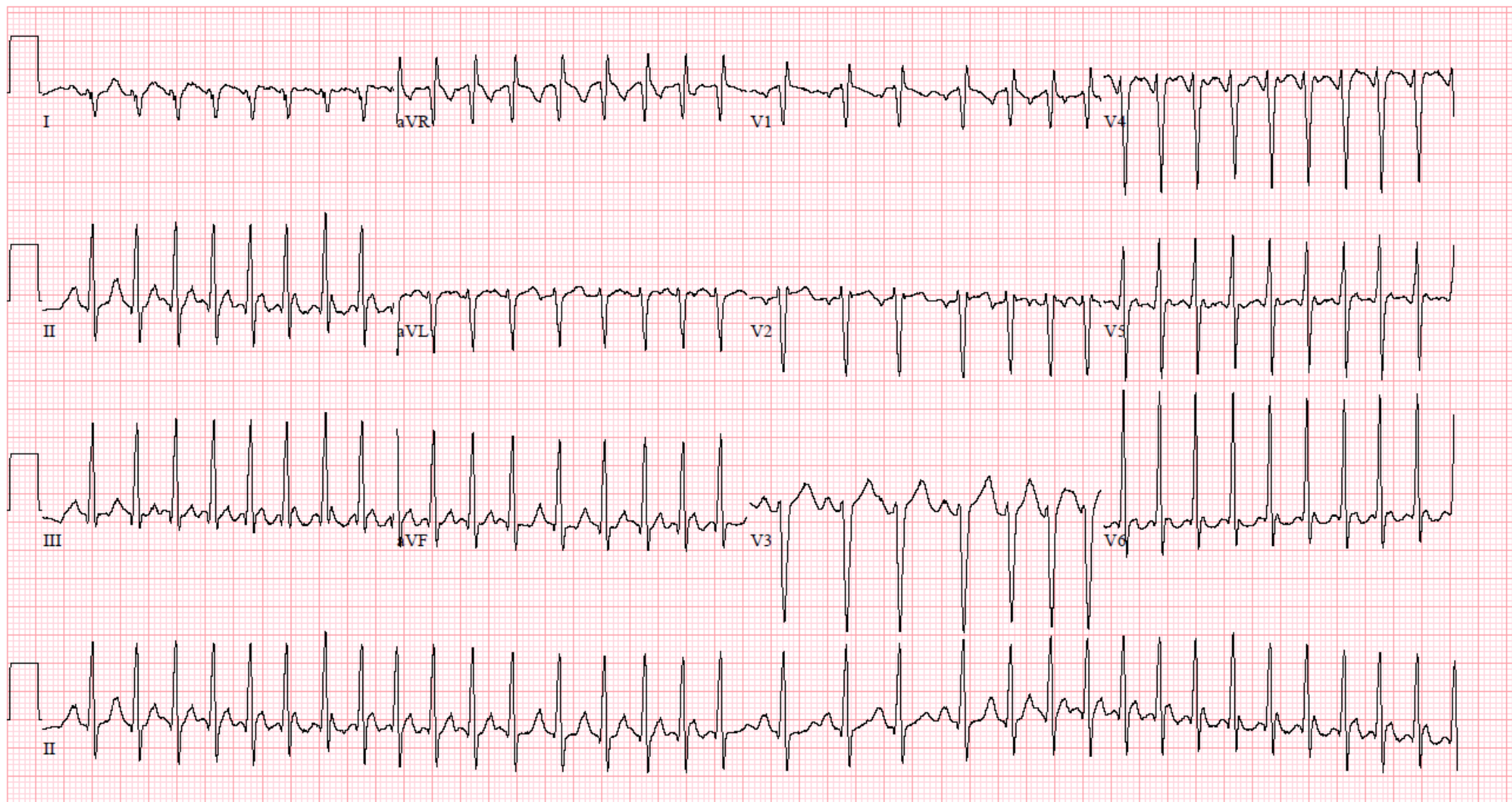




Focal atrial tachycardia

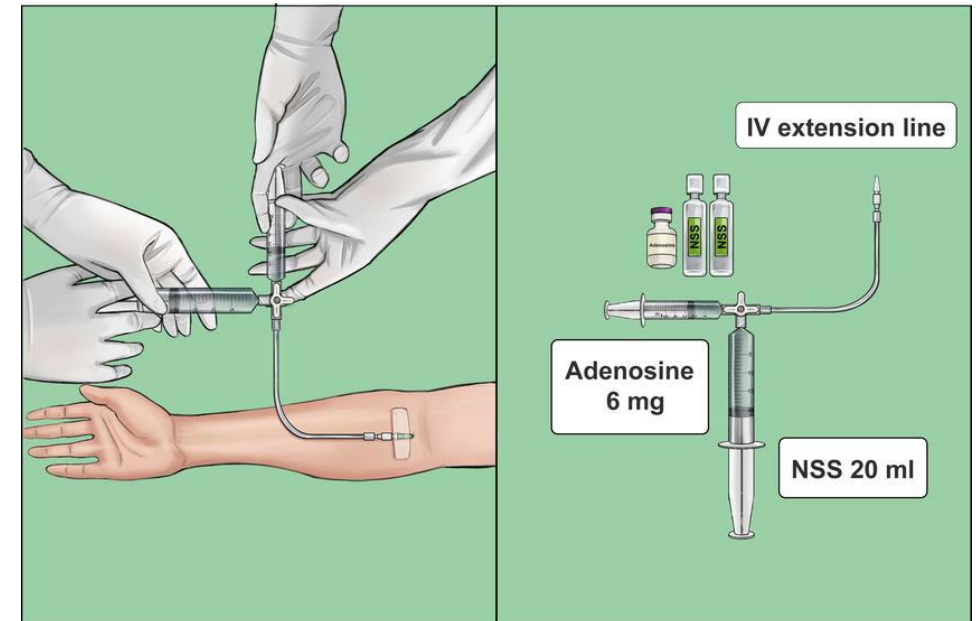
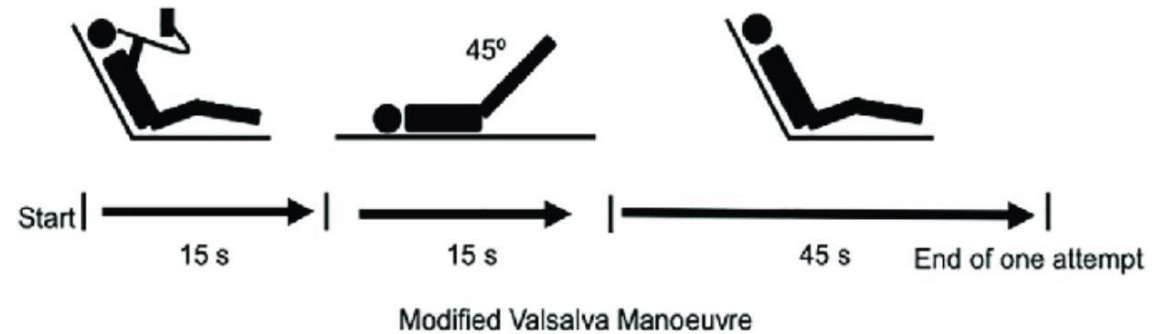
- Point source of ectopic atrial impulse formation and tachycardic
- Can have gradual onset



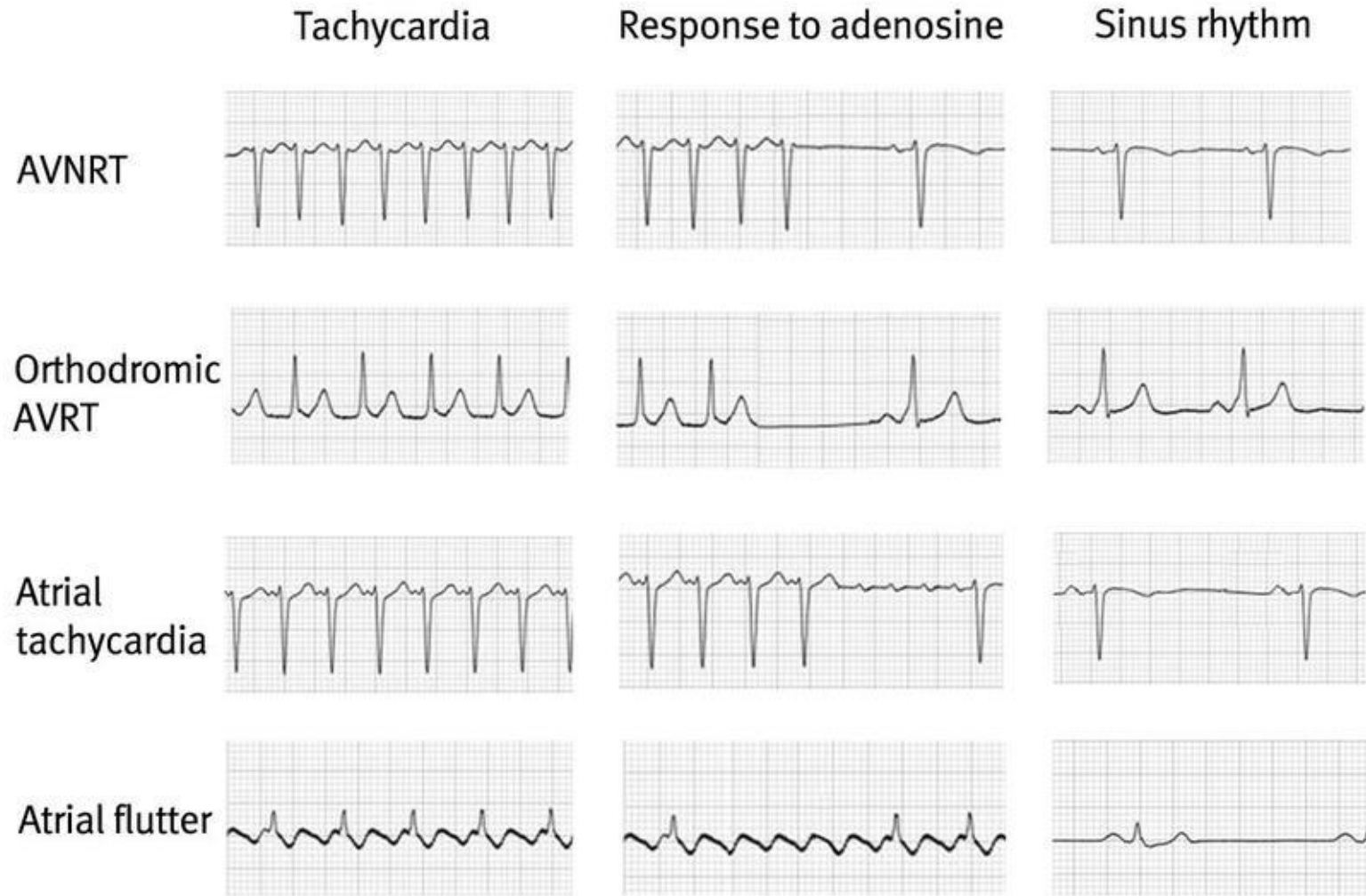


Stable patient with regular NCT

- 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



Typical adenosine responses

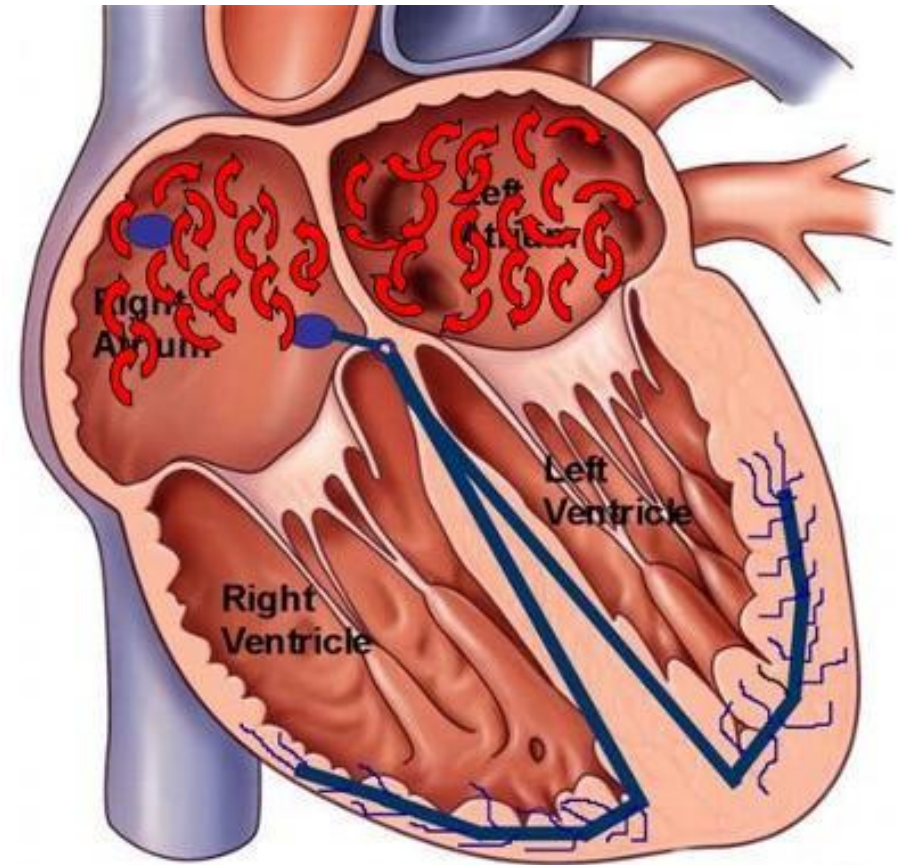


DDx of narrow complex irregular tachycardia

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

Atrial fibrillation

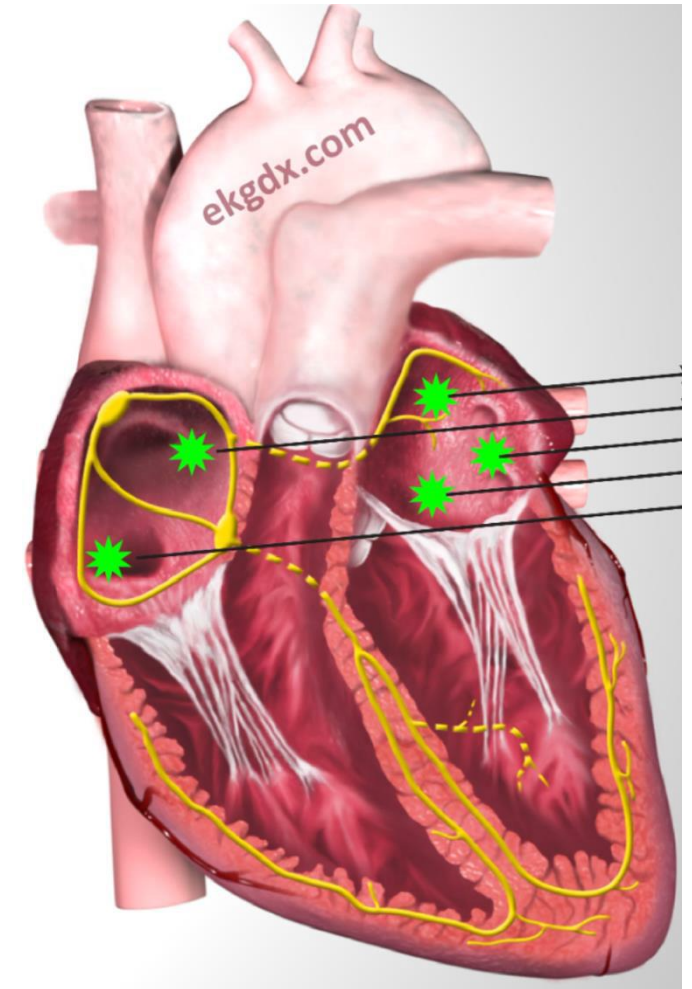
- Fibrillatory waves in the atrium at 300-600bpm
- Irregularly irregular rhythm
- Usually can be distinguished from other SVTs without need for diagnostic intervention

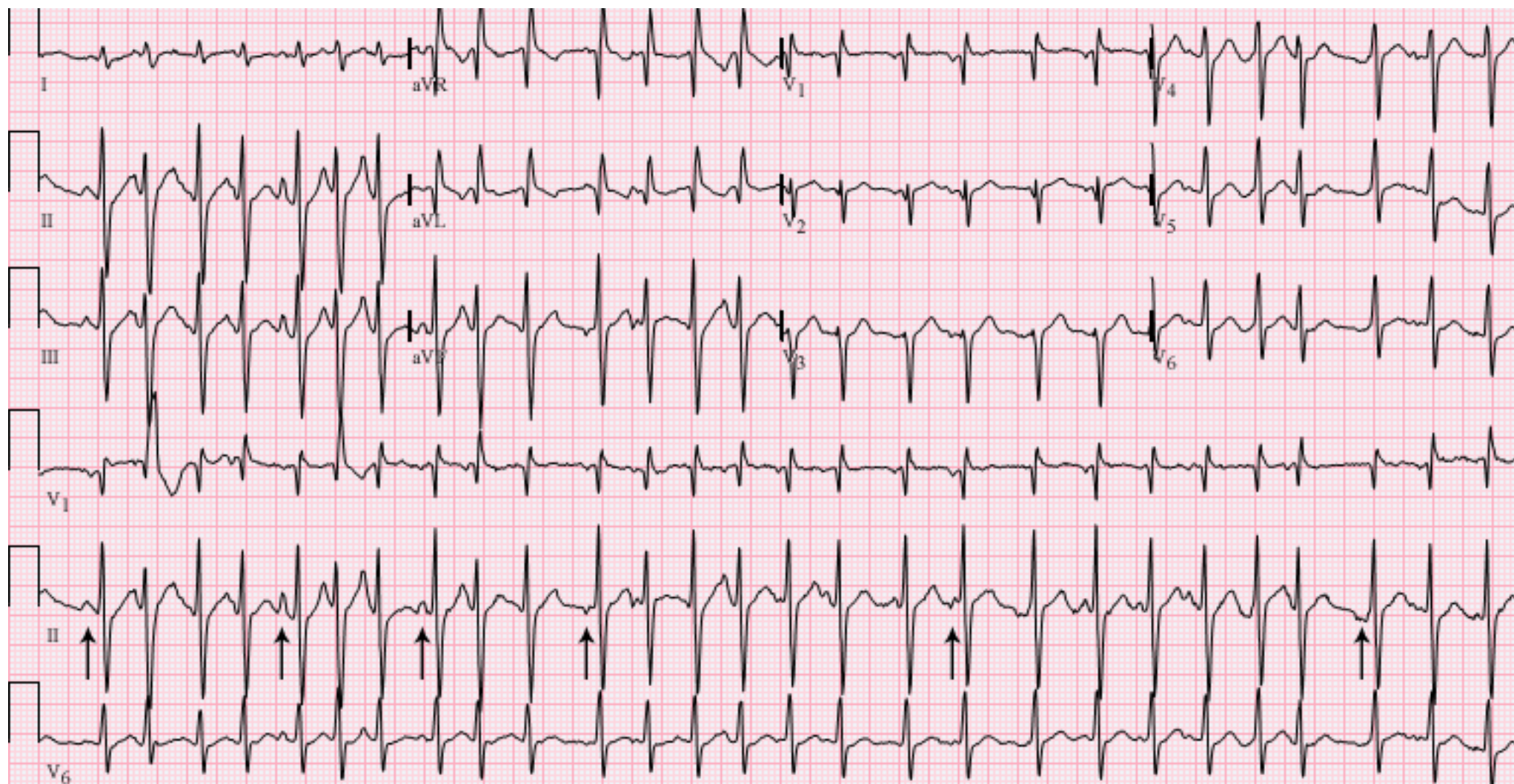




Multifocal atrial tachycardia

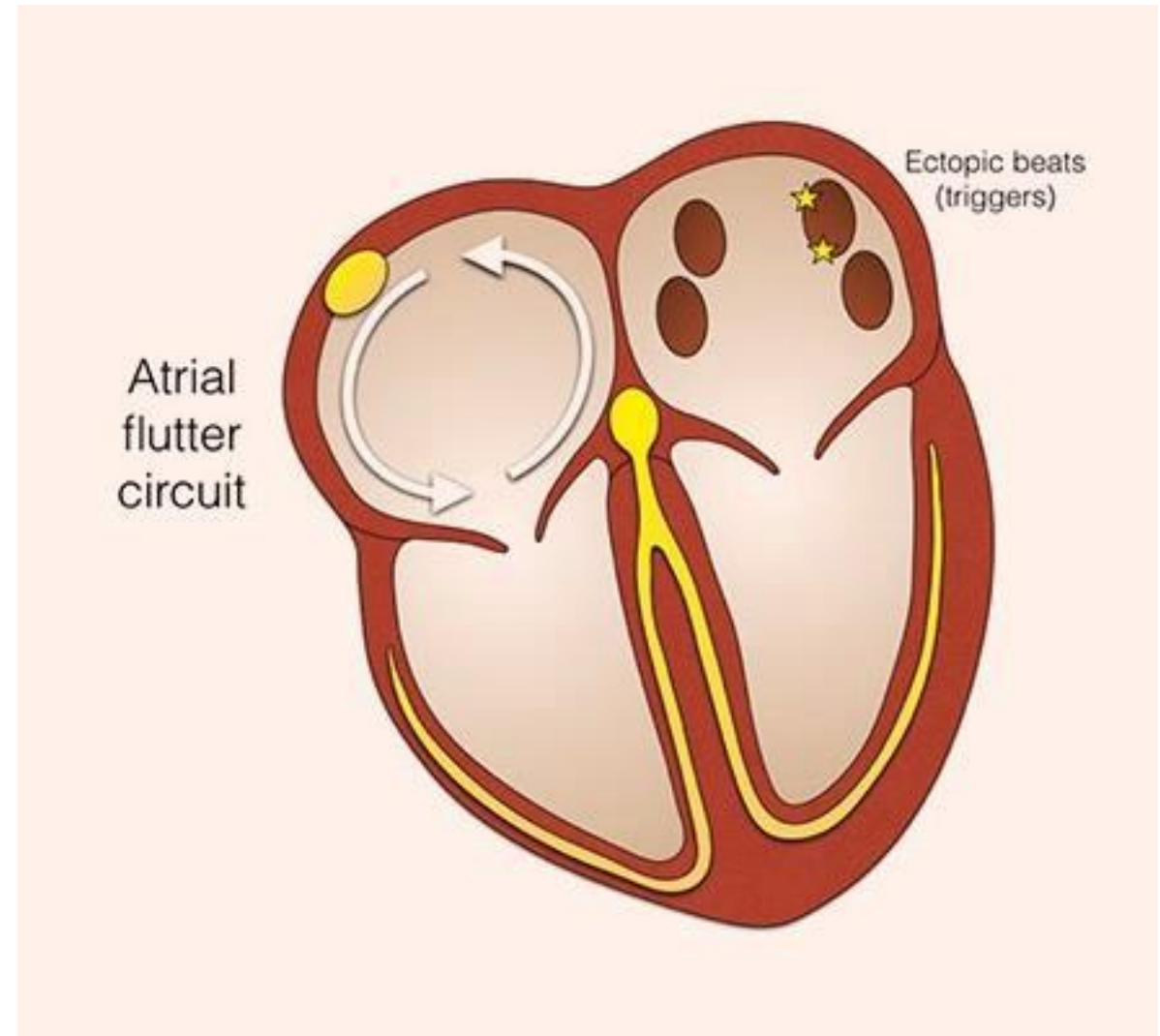
- Rapid, irregular rhythm
- >3 ectopic atrial foci
- Varying PR interval beat to beat
- No single dominant P wave morphology
- Associated with pulm disease/acute illness/theophylline
- Tx ⑦treatment of underlying illness

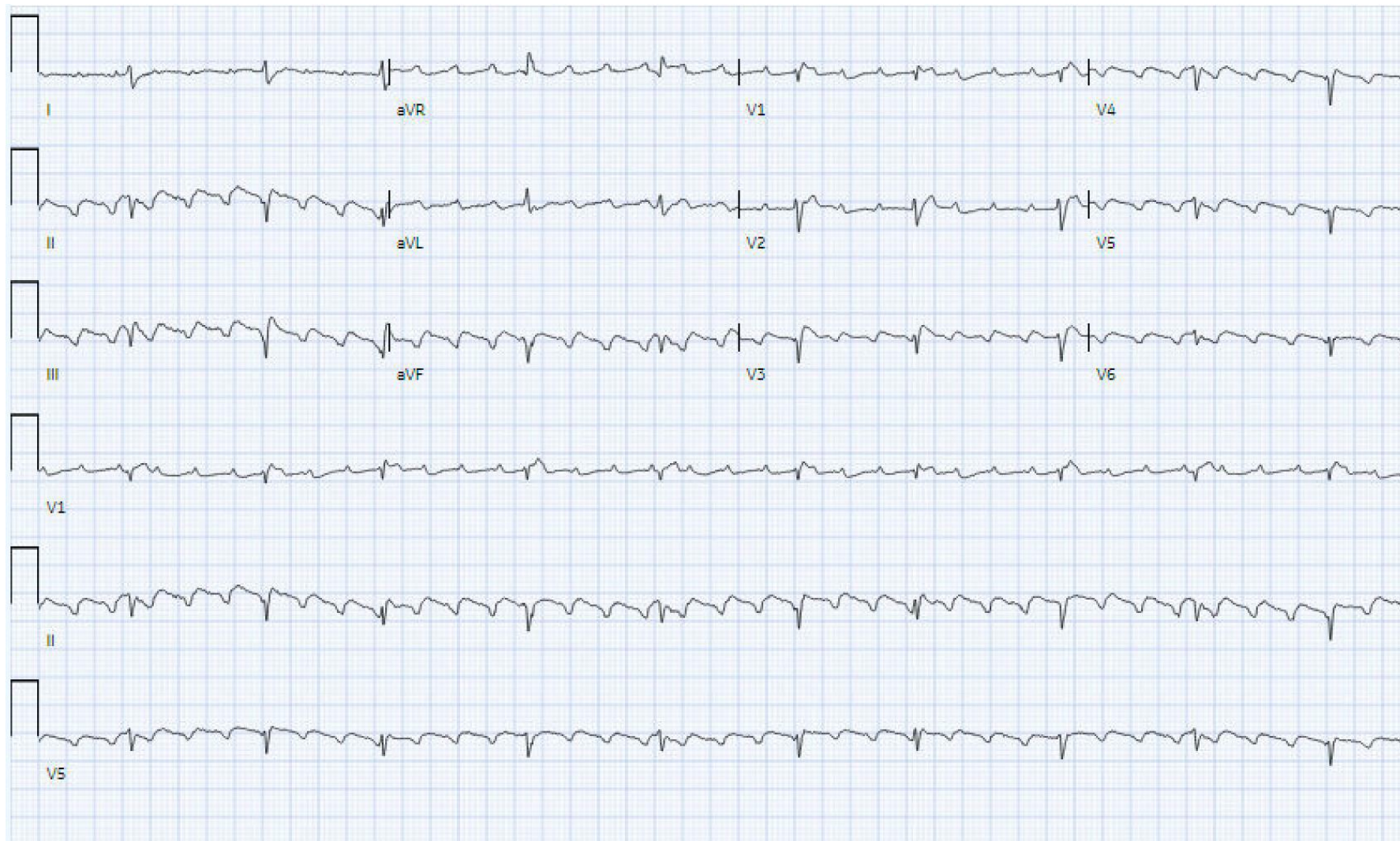




Atrial flutter

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





Parkland

UTSouthwestern
Medical Center

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

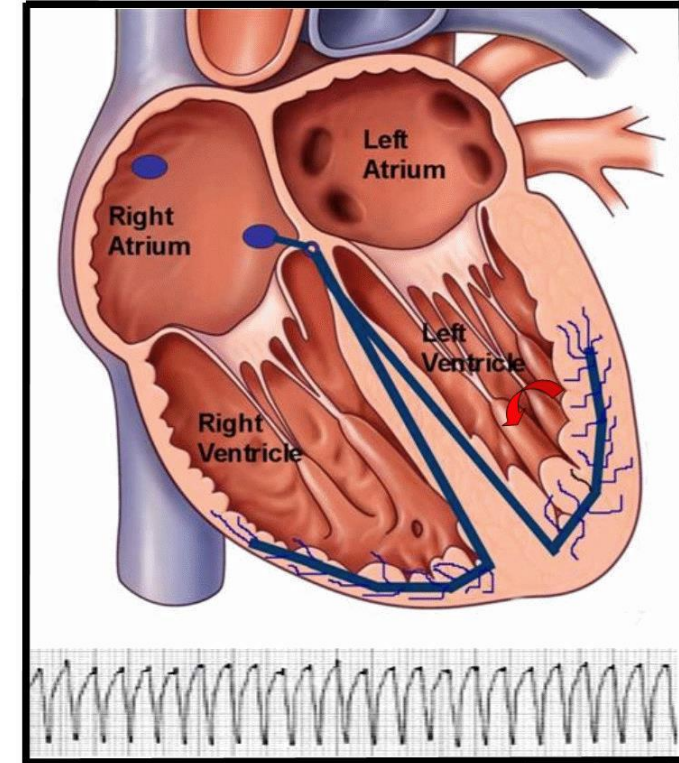
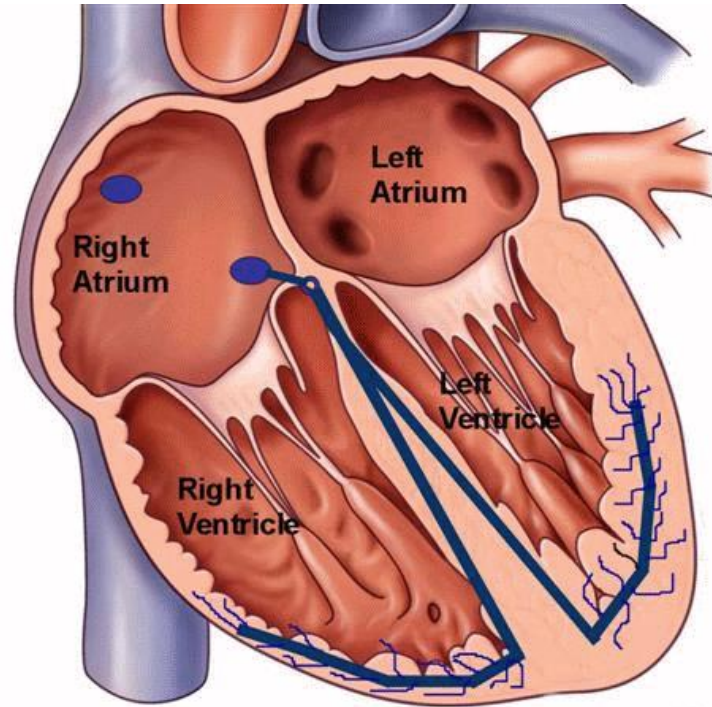
Wide complex tachycardia

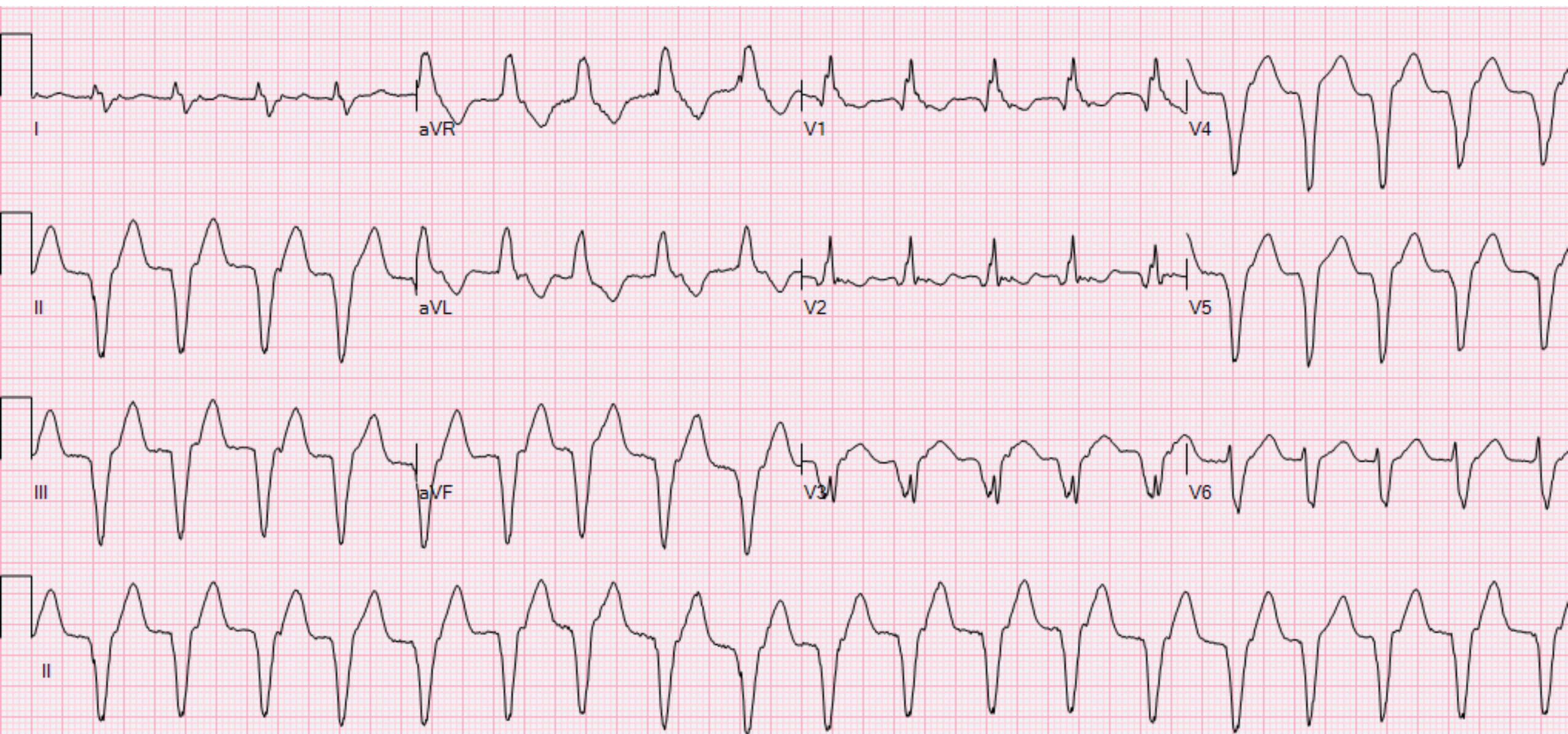
DDx of wide complex regular tachycardia

- 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



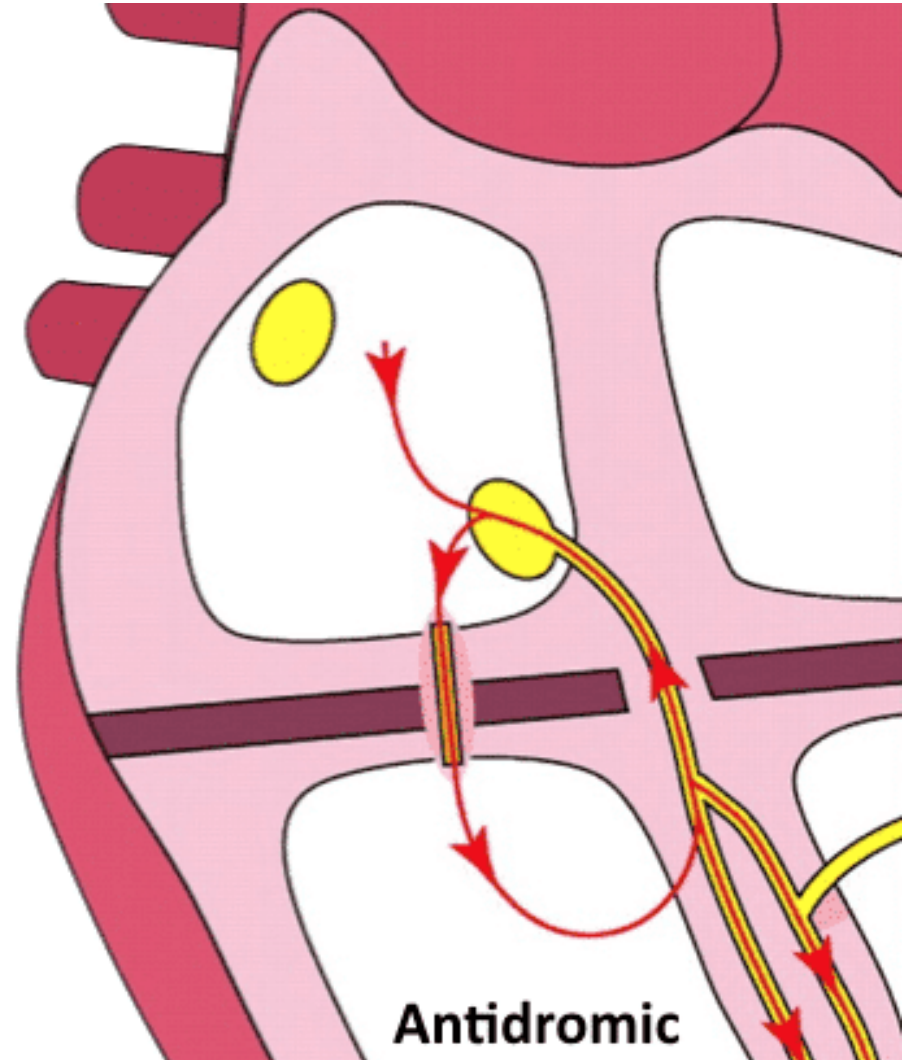


Parkland

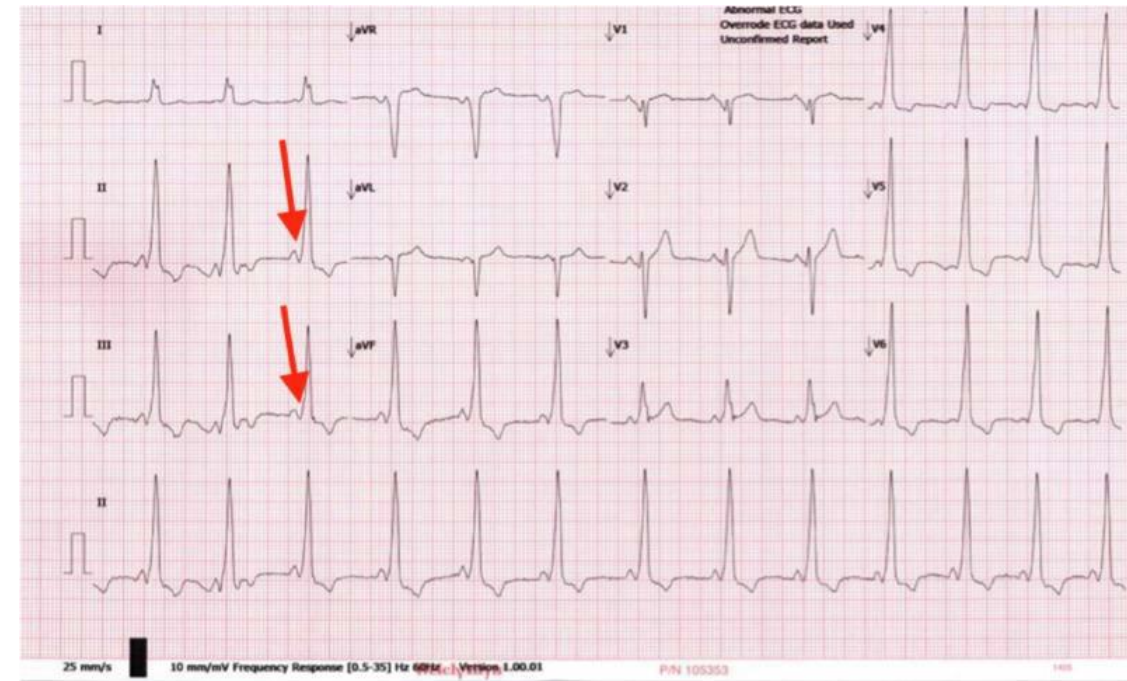
UTSouthwestern
Medical Center

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

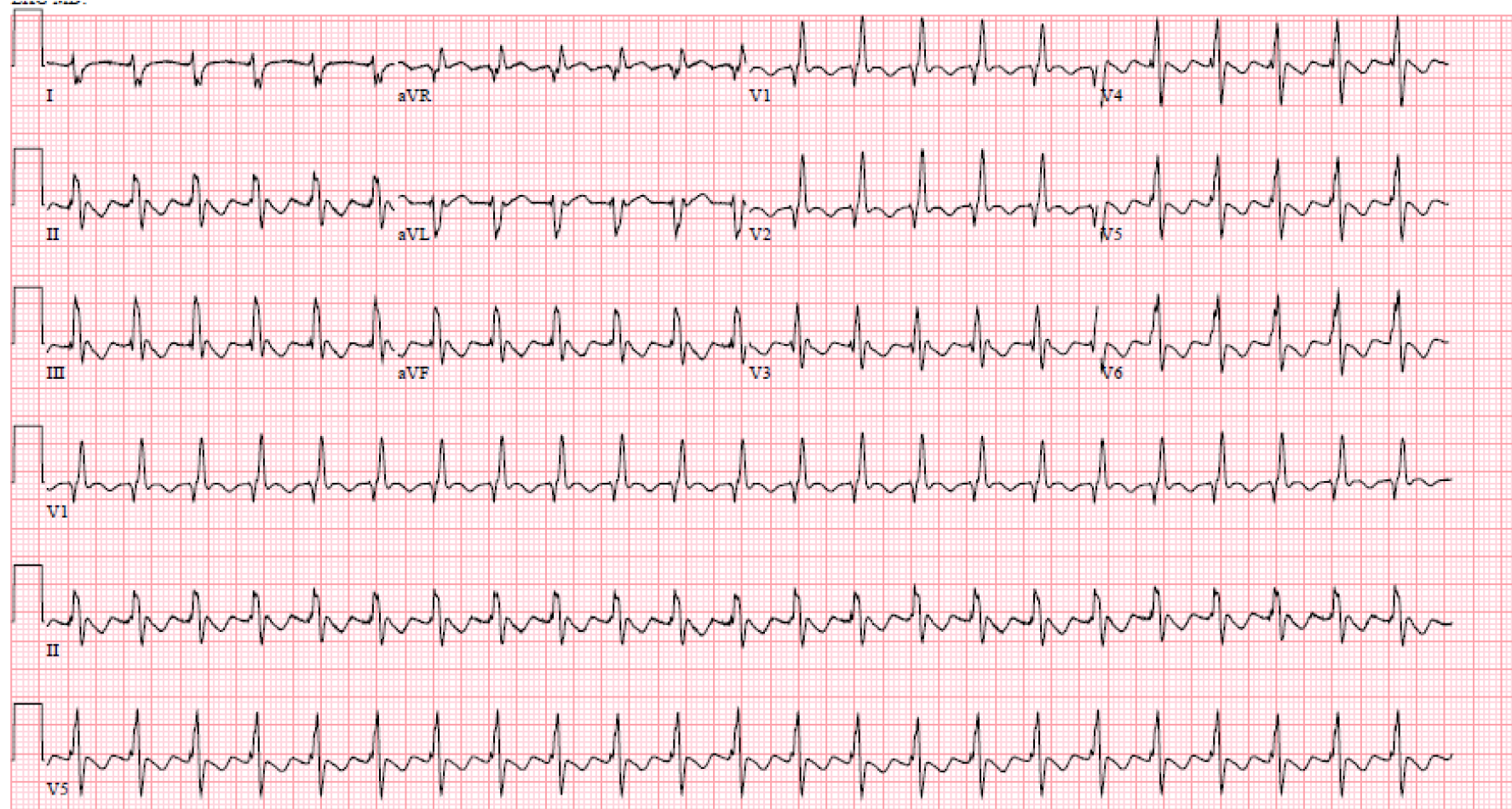


Parkland

UTSouthwestern
Medical Center

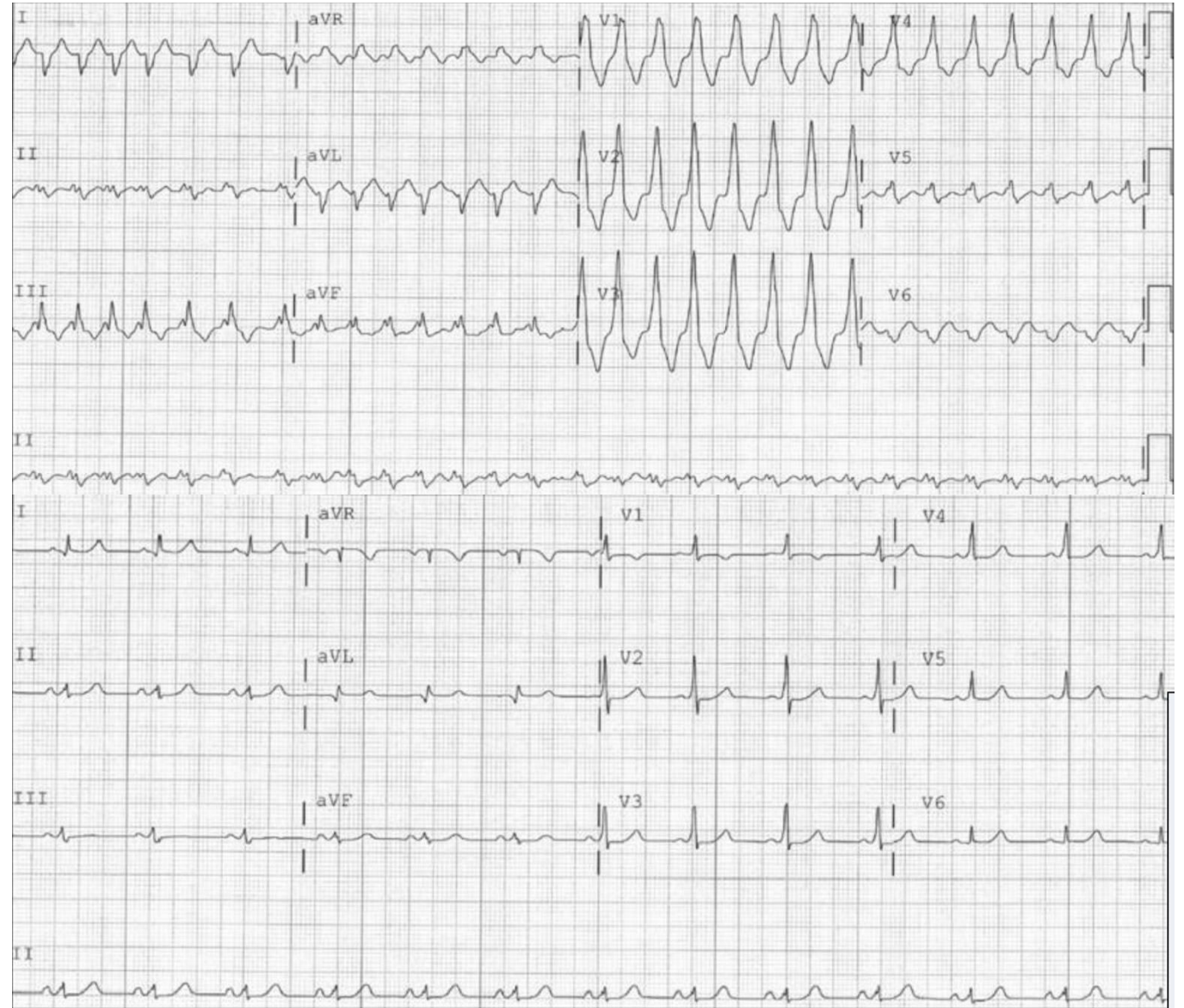
SVT with aberrancy

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



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)

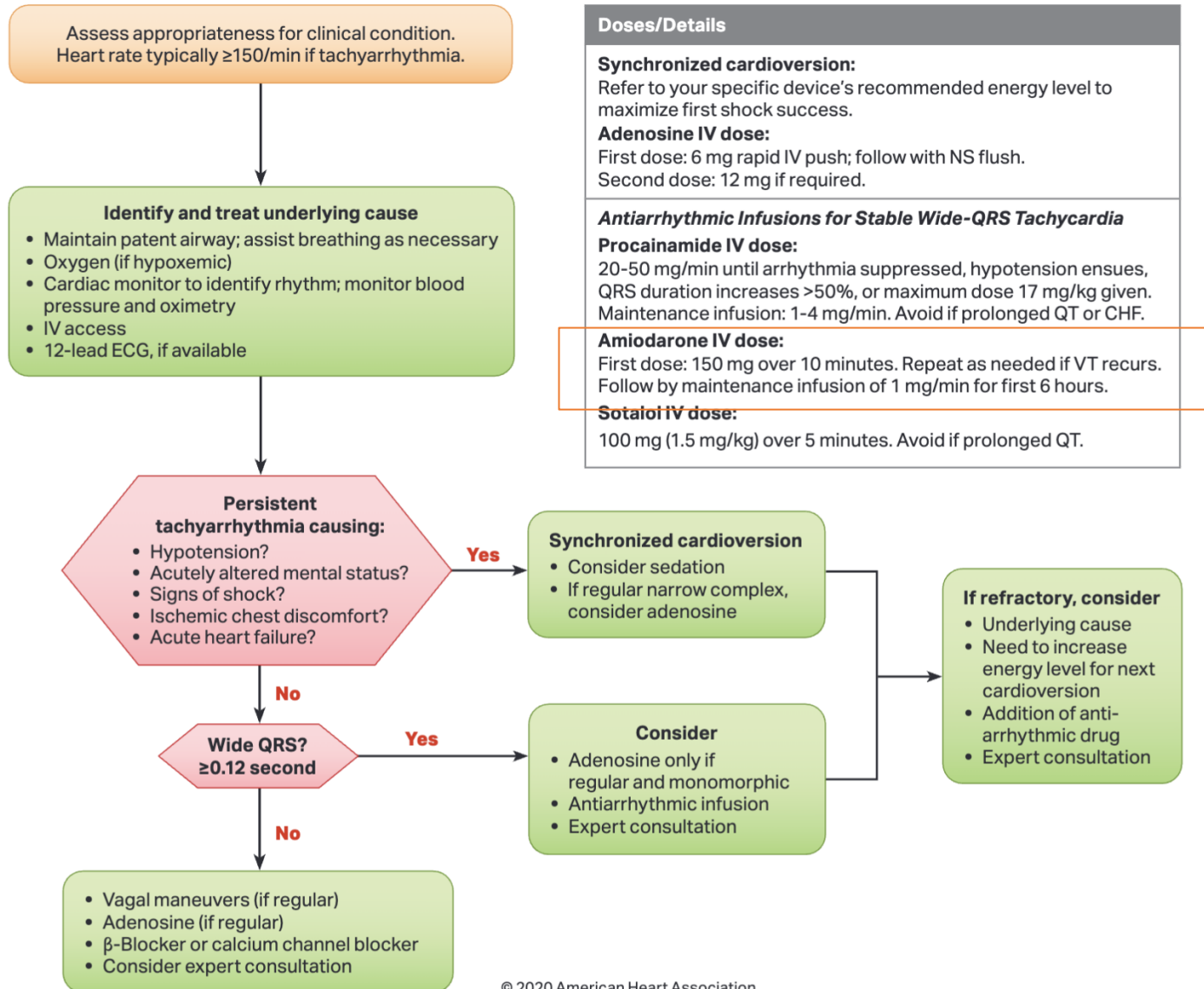


Stable patient with regular WCT

- 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



© 2020 American Heart Association

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



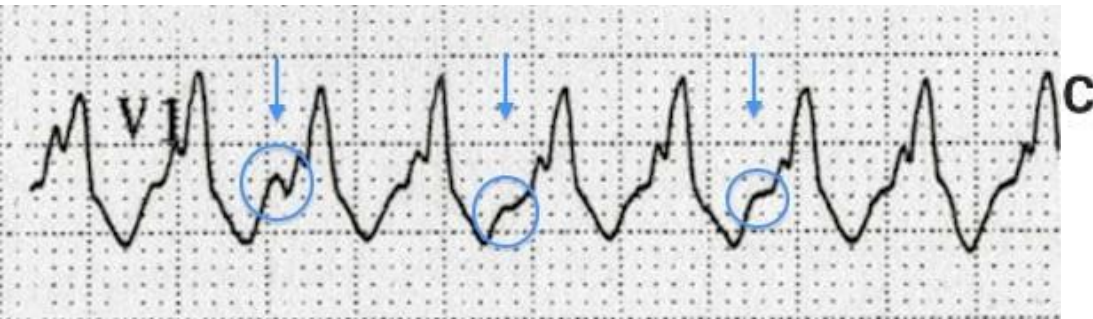
Parkland

UTSouthwestern
Medical Center

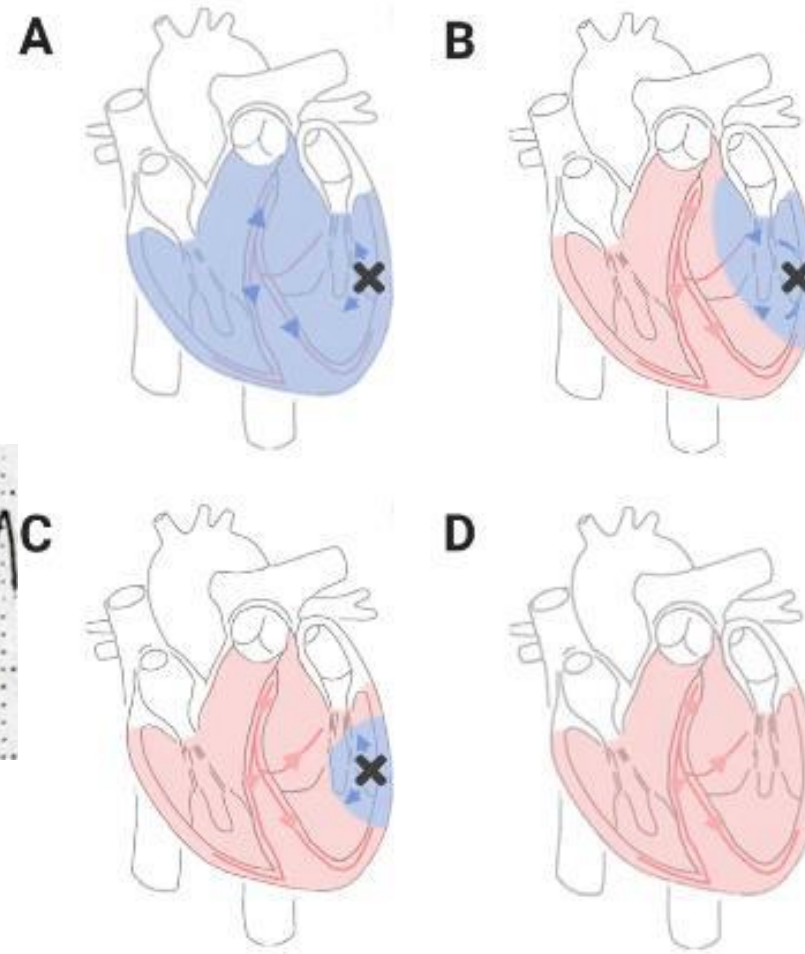
Factors pointing toward VT

- 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



Fusion (B/C) and capture (D) beats

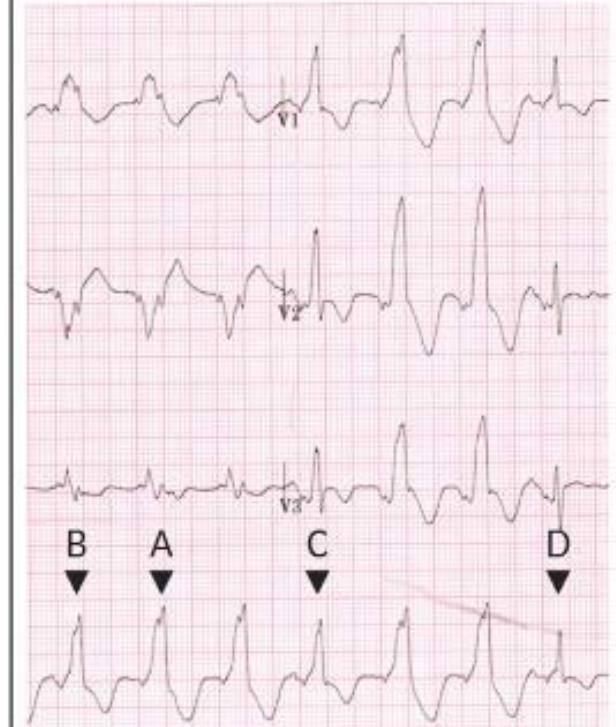


Figure 1 Generation of capture and fusion beats with corresponding changes on electrocardiogram due to competing activation from supraventricular and ventricular tachycardia source. Activation from a ventricular tachycardia source is indicated in *blue* and sinus rhythm is indicated in *red*.

Atypical QRS morphology 7VT

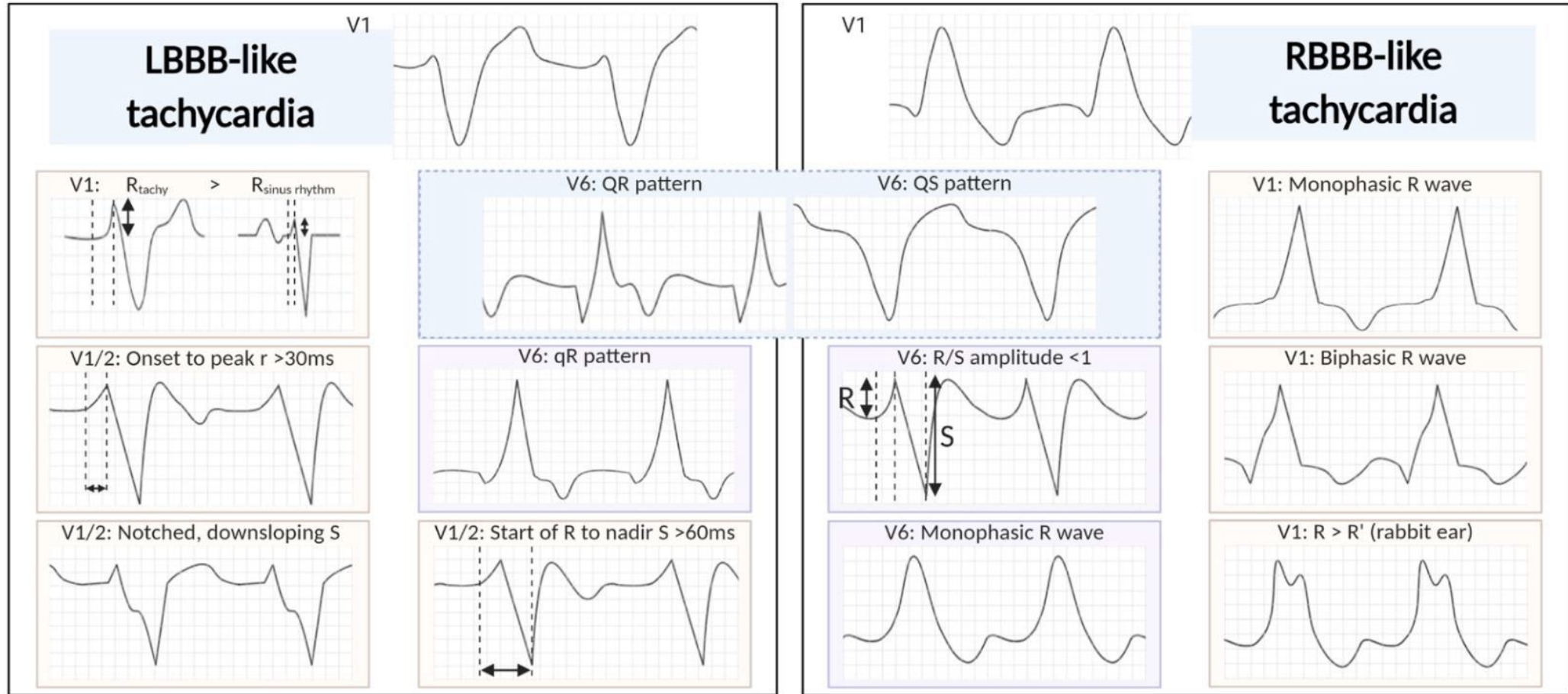
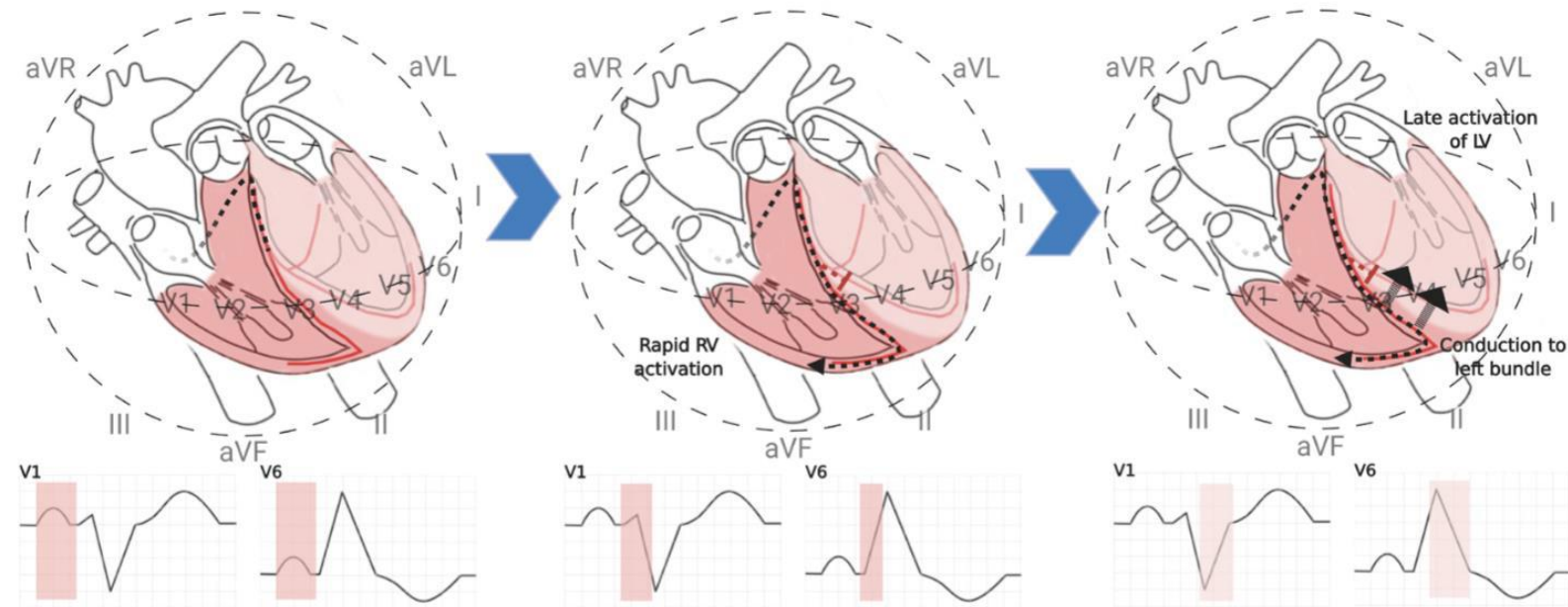


Figure 3 Atypical electrocardiographic features of right bundle branch block (RBBB) and left bundle branch block (LBBB) that are suggestive of ventricular tachycardia.

Typical LBBB

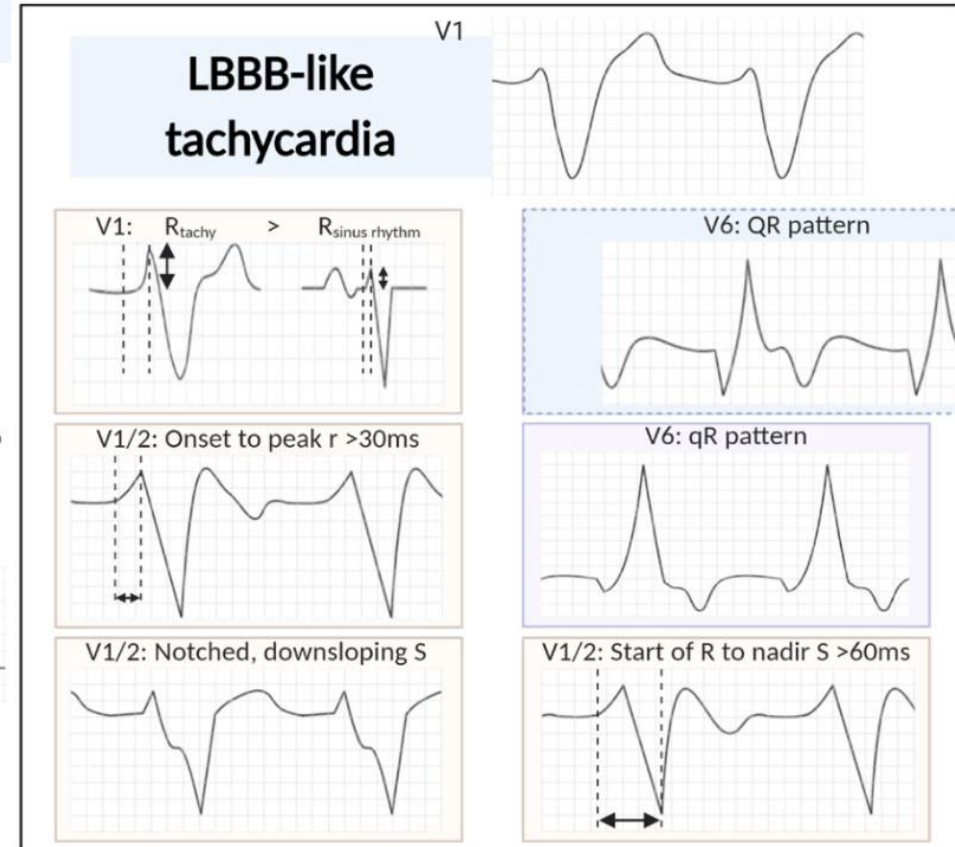


Conduction through AVN and His bundle

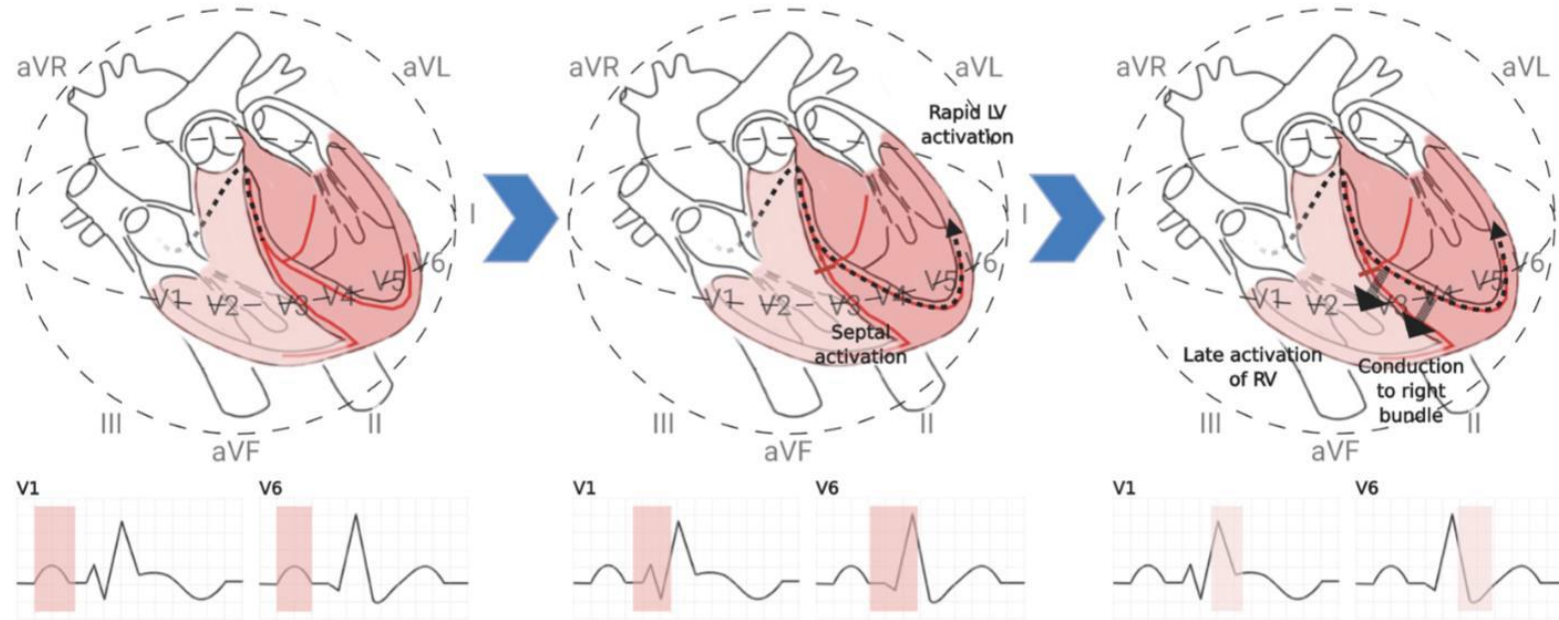
Rapid initial activation of the RV
 ⚡ small r in V1, small s in V6

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

Patterns that suggest VT



Typical RBBB

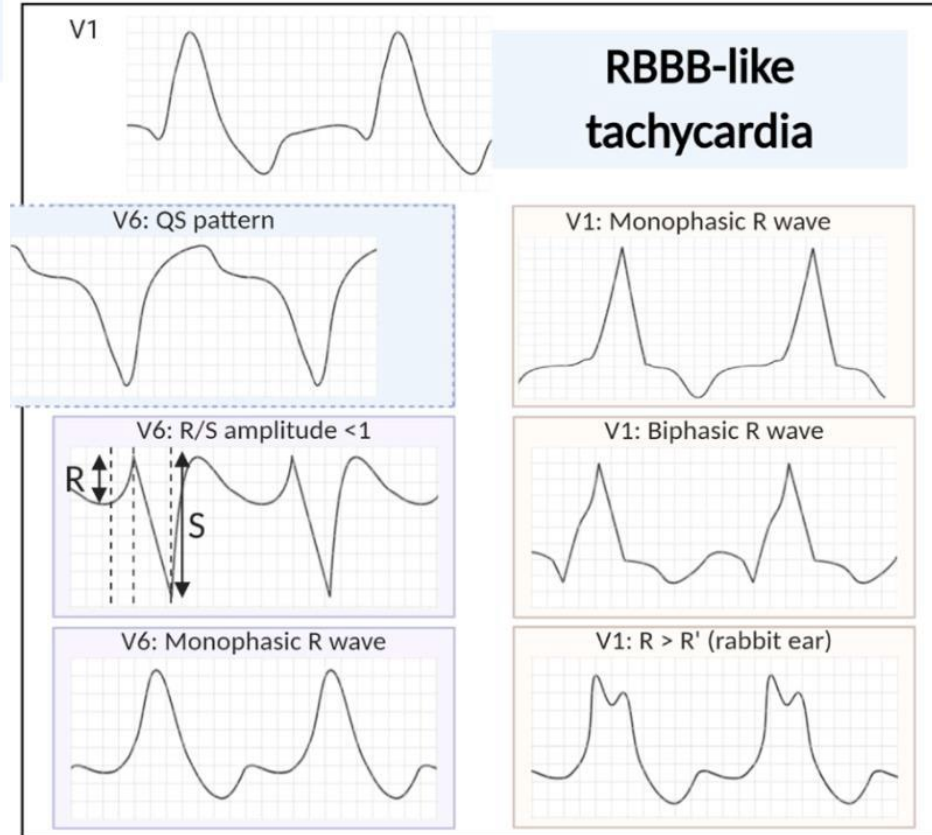


Conduction
through AVN
and His bundle

Rapid initial
activation of the
septum and LV ⑦
small rs complex in
V1, tall R in V6

Late activation of
RV and right
bundle ⑦ late R in
V1, s in V6

Patterns that suggest VT



**RBBB-like
tachycardia**

V6: QS pattern

V1: Monophasic R wave

V6: R/S amplitude < 1

V1: Biphasic R wave

V6: Monophasic R wave

V1: R > R' (rabbit ear)

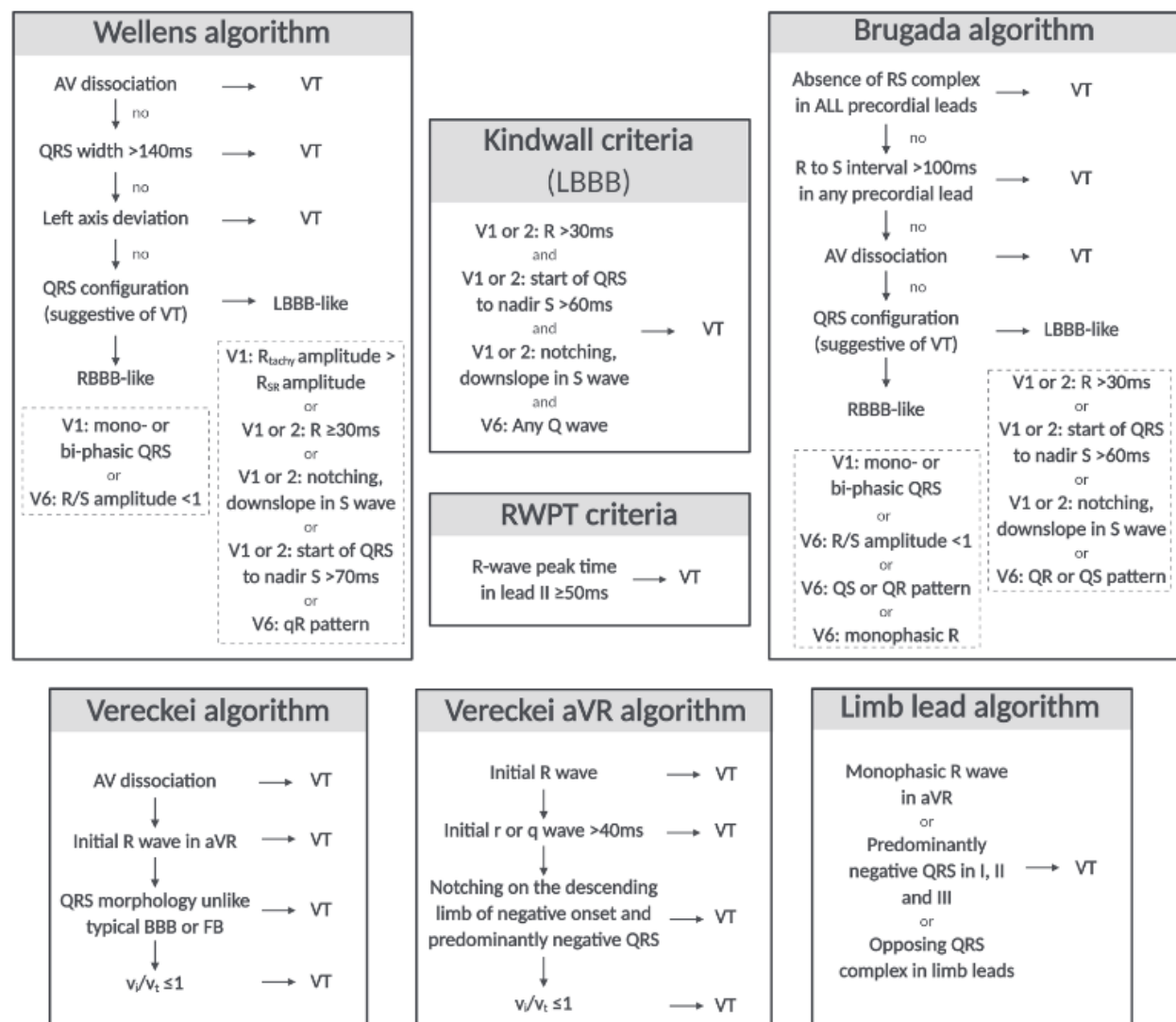


Figure 8 Wide complex tachycardia algorithms to distinguish between ventricular and supraventricular tachycardias. All the algorithms rely on specific features of ventricular tachycardia. If none are present, a diagnosis of supraventricular tachycardia is most likely. AV, atrioventricular; BBB, bundle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

Long term management

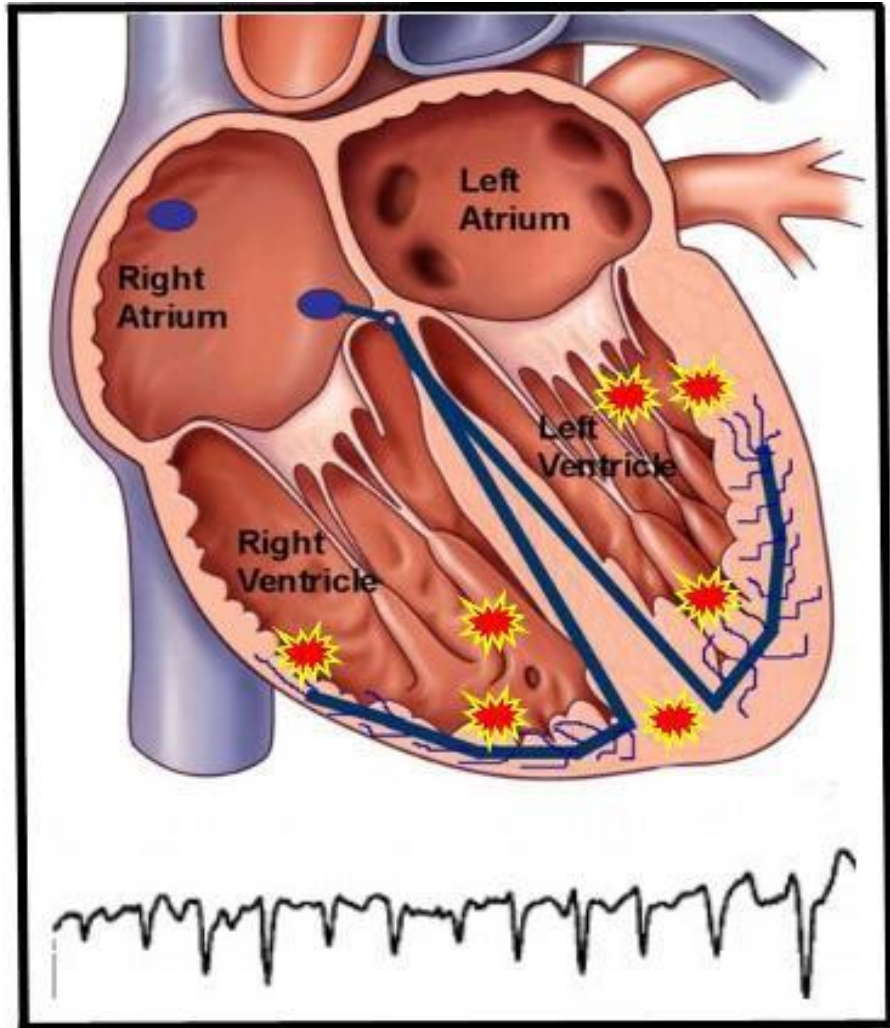
- Antiarrhythmic agents
- Nodal blocking agents
- Catheter ablation

DDx of wide complex irregular tachycardia

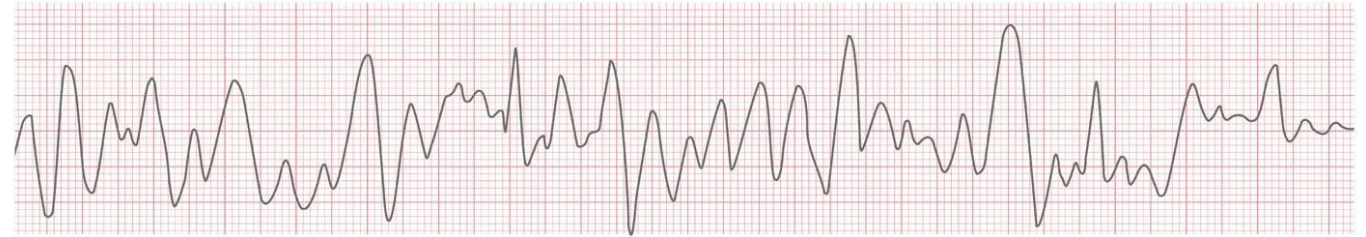
- Atrial fibrillation, flutter, or focal AT with aberrancy
- Atrial fibrillation, flutter, or focal AT with anterograde accessory pathway conduction (WPW)
- Polymorphic VT
- Ventricular fibrillation



Polymorphic VT



Polymorphic ventricular tachycardia

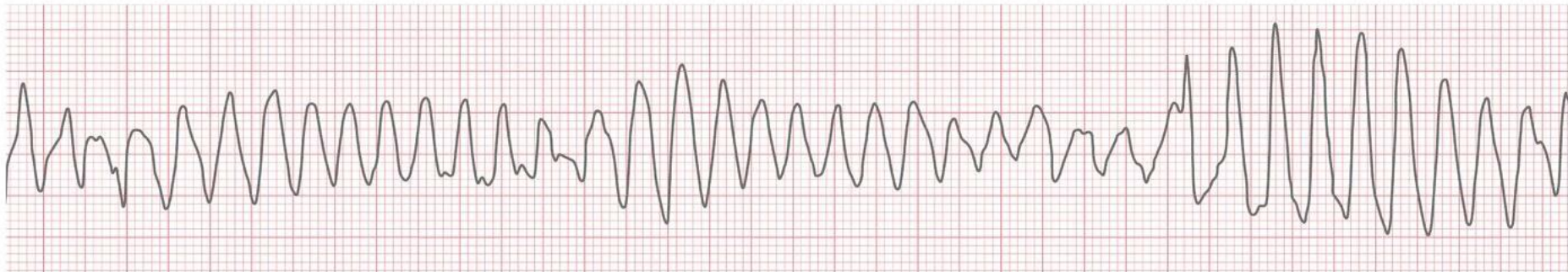


- Alternative QRS patterns reflecting various sites of ventricular activation
- Usually unstable or pulseless requiring immediate DCCV/defibrillation

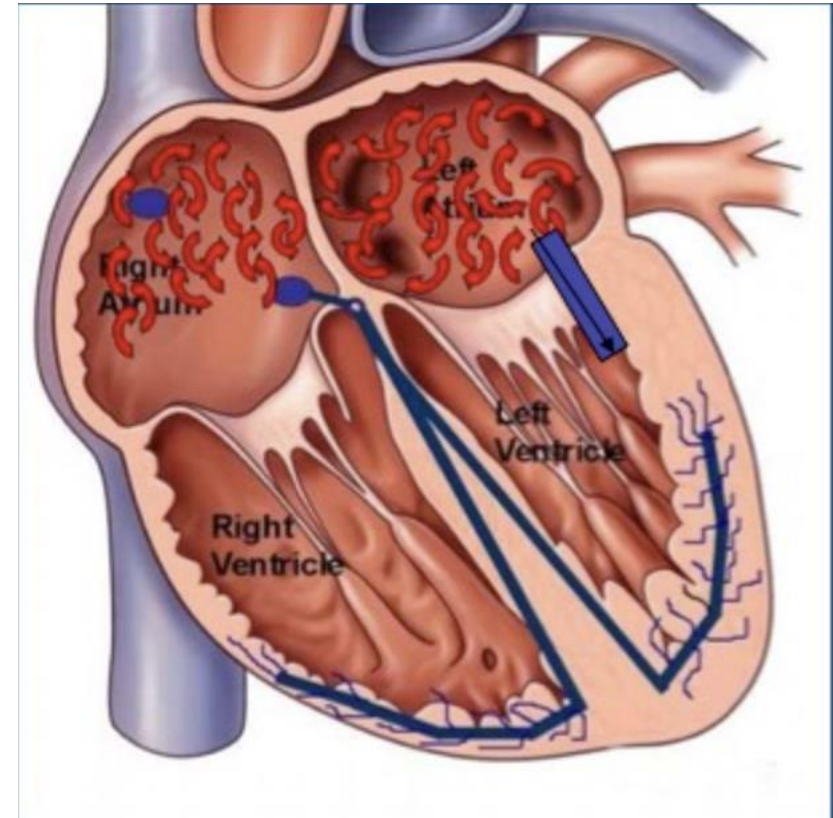
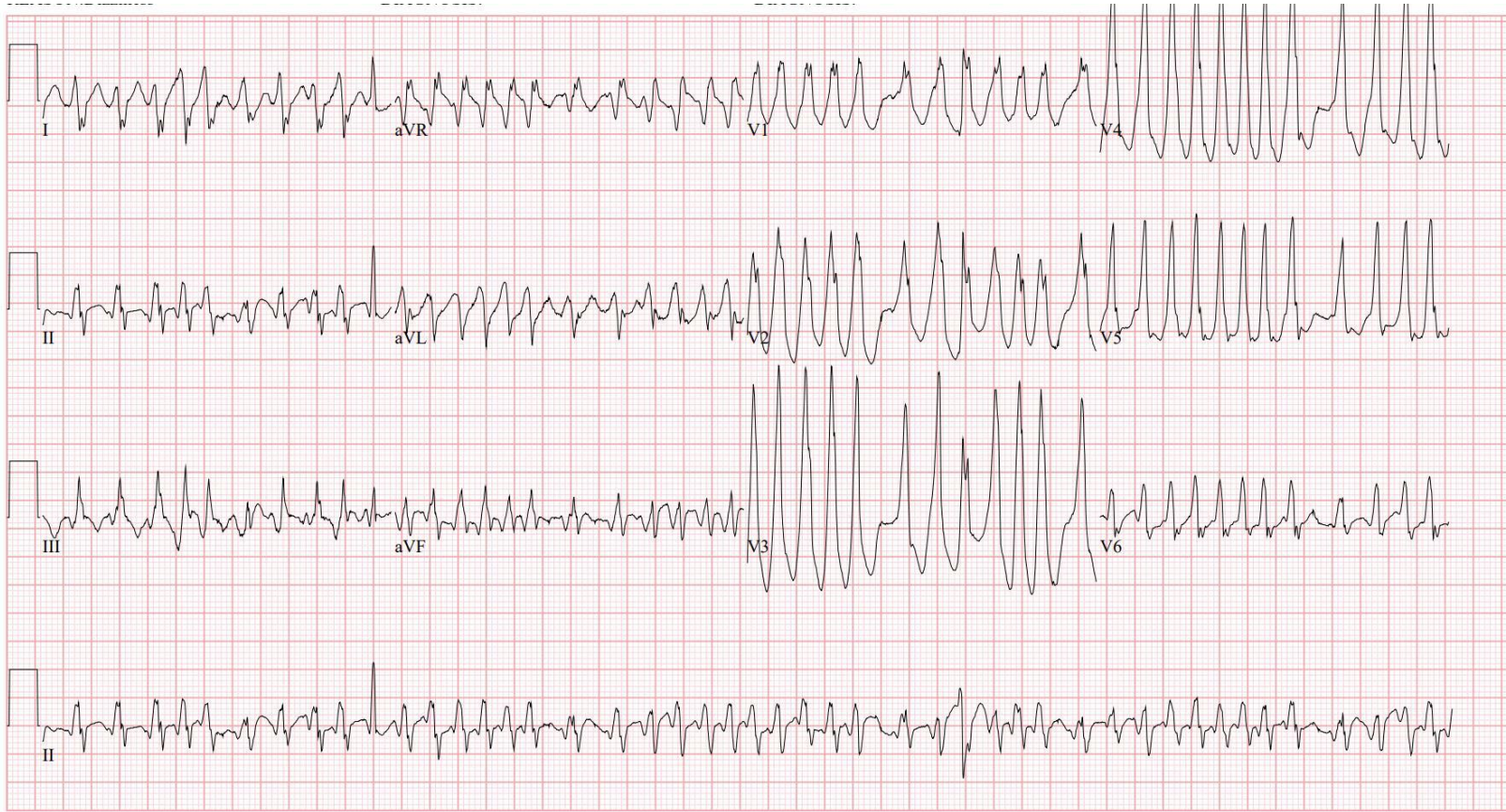
Torsades de pointes

- 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

Torsades de pointes



Atrial fibrillation with pre-excitation



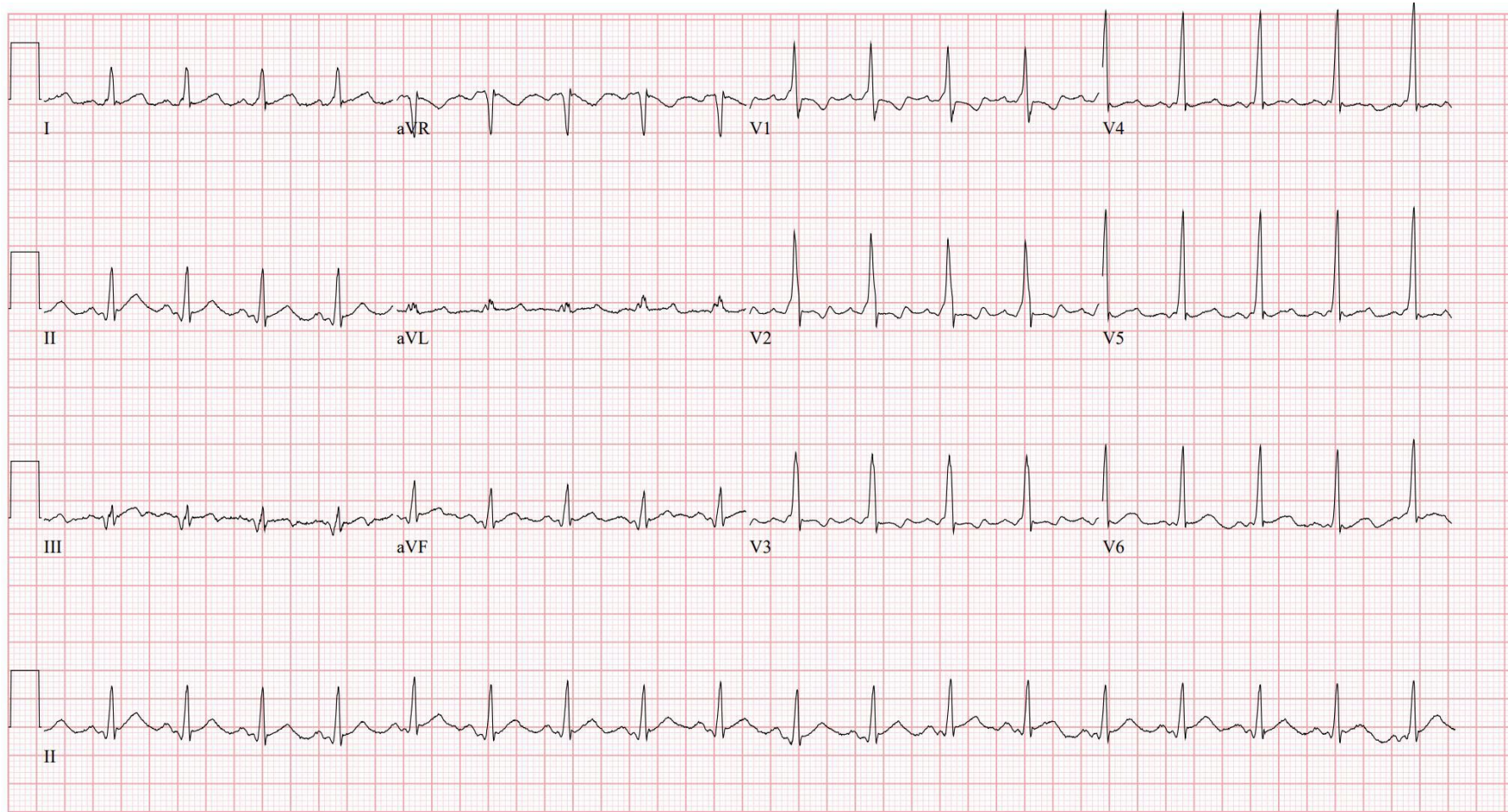
AF with simultaneous conduction down AV node and a rapidly conducting accessory pathway
If stable ⑦procainamide or ibutilide



Parkland

UTSouthwestern
Medical Center

Follow-up ECG



Parkland

UTSouthwestern
Medical Center

Ventricular fibrillation

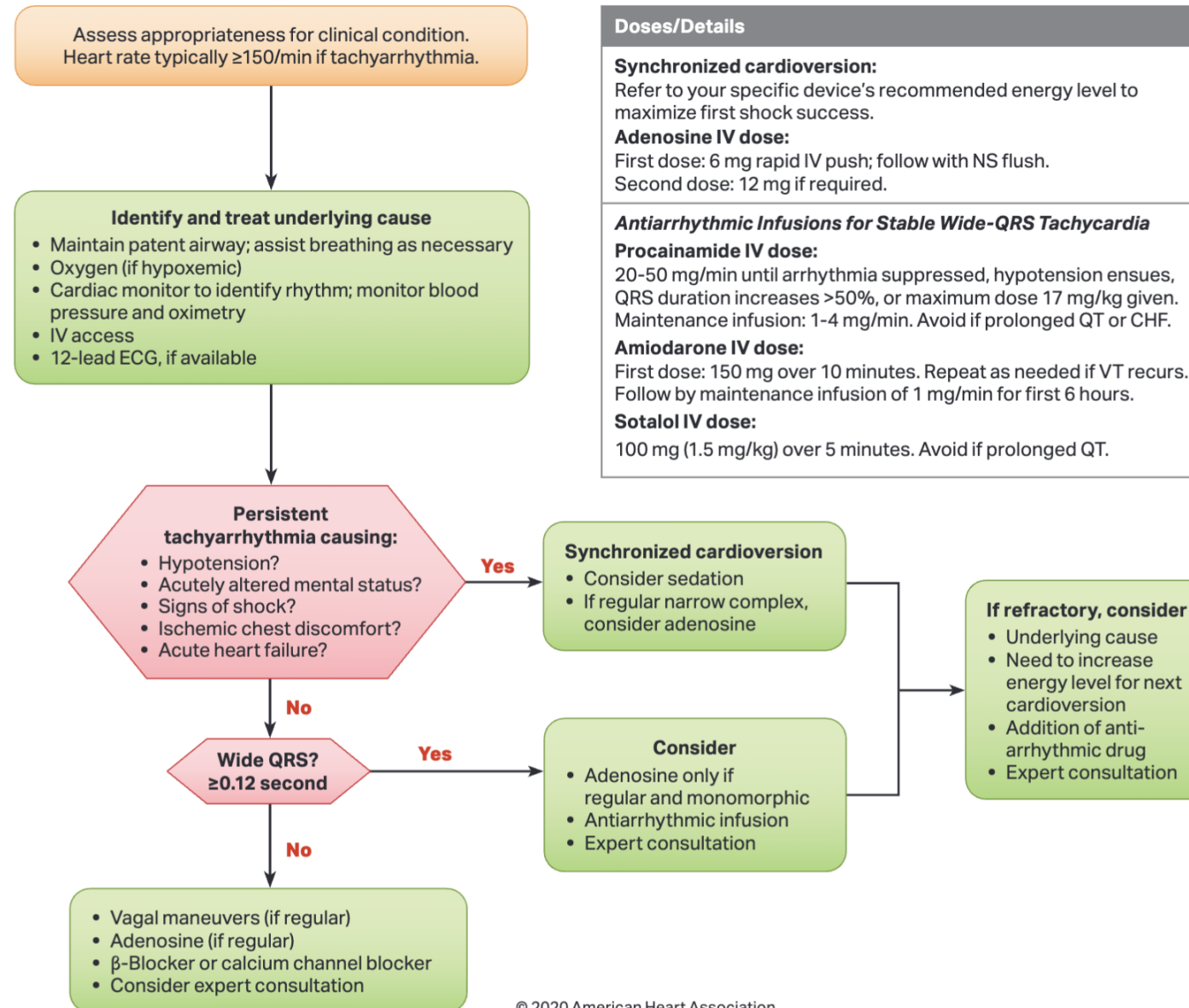
- Absence of identifiable P waves or QRS complexes
- Pulseless rhythm that requires immediate CPR and defibrillation



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 ⑦ amiodarone usually initial choice
- Torsades ⑦ IV magnesium, potassium repletion, chronotropic agents, possibly pacing
- Pre-excited atrial fibrillation ⑦ procainamide or ibutilide

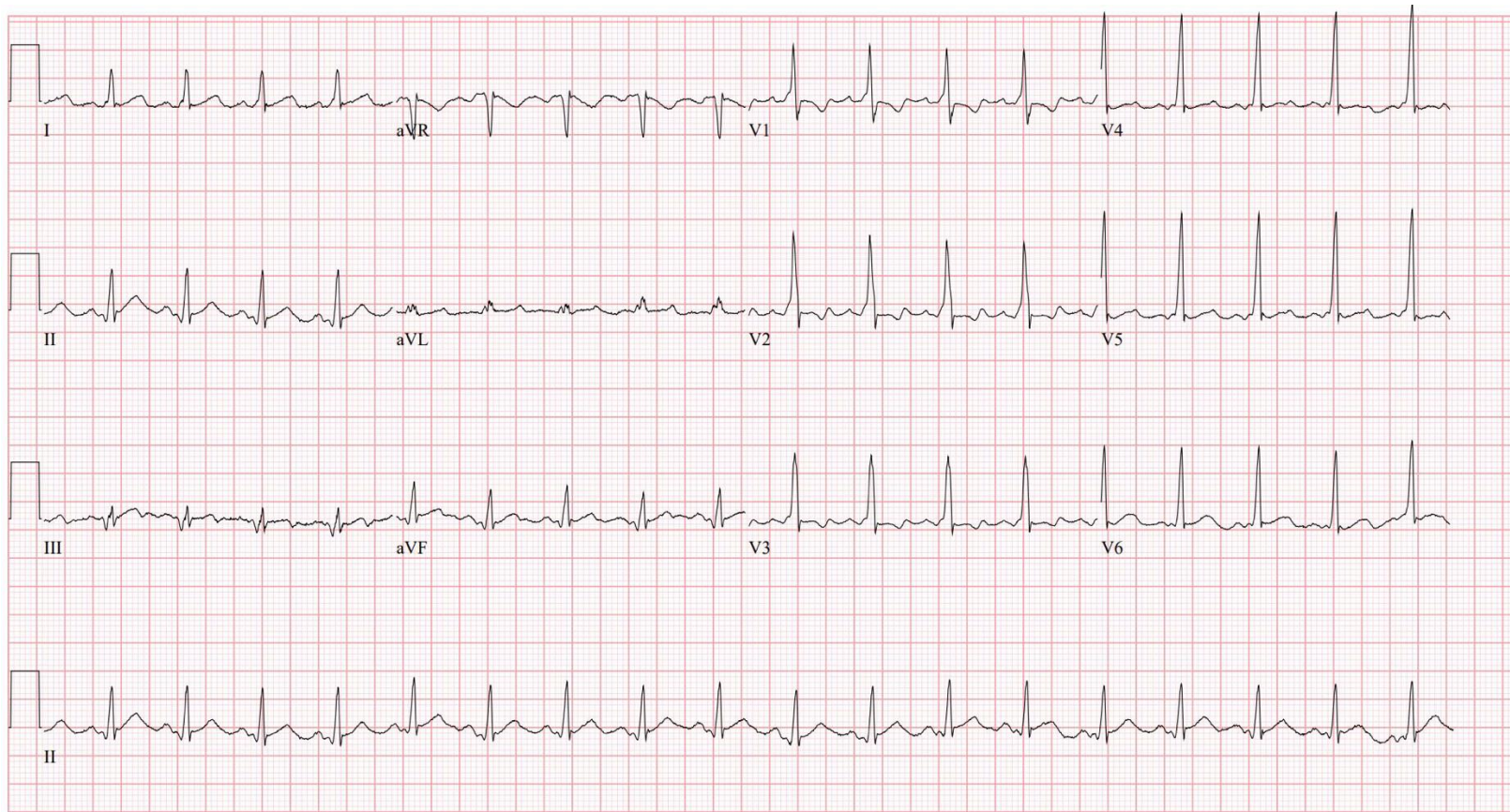
Adult Tachycardia With a Pulse Algorithm

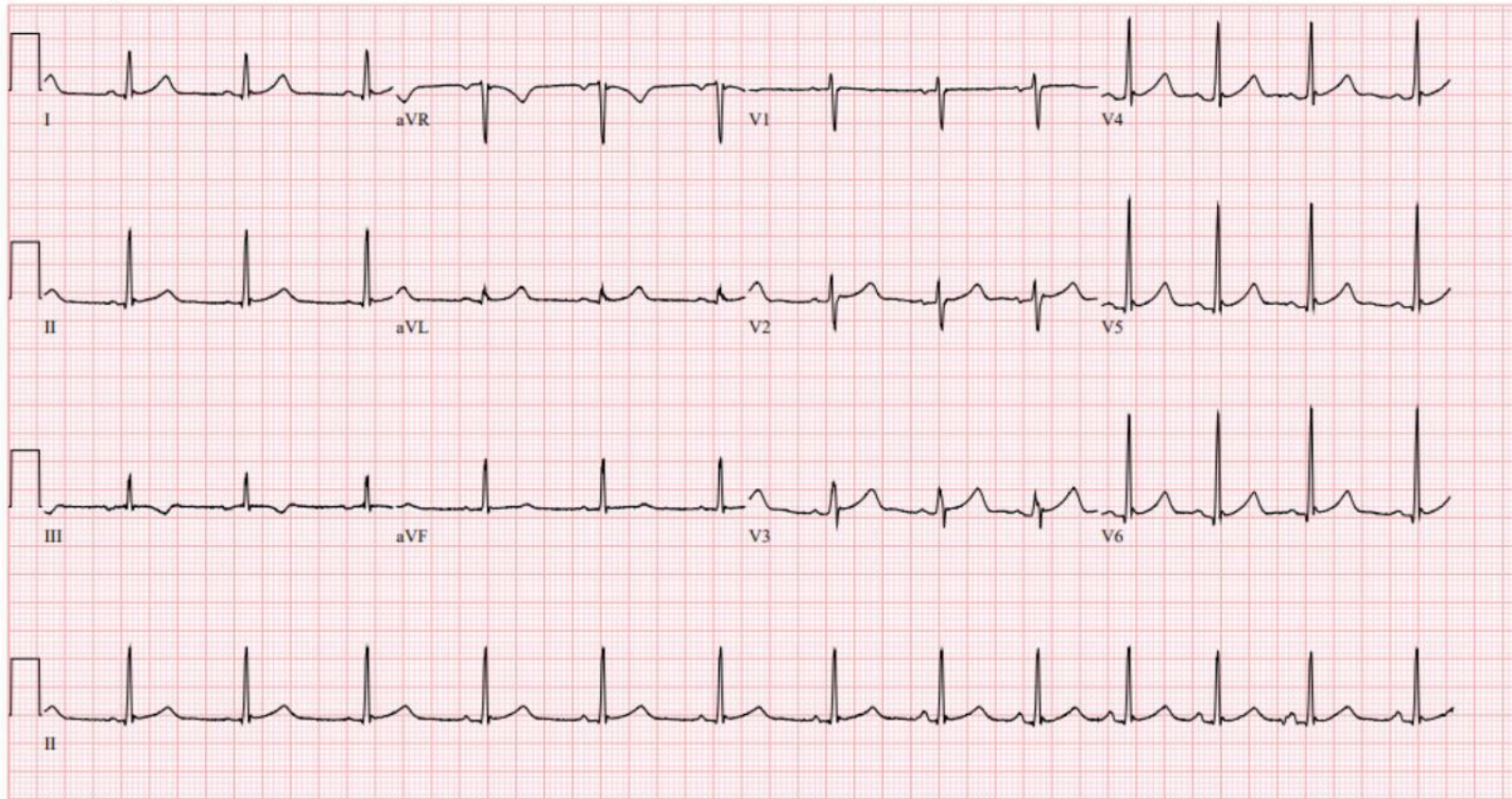


© 2020 American Heart Association

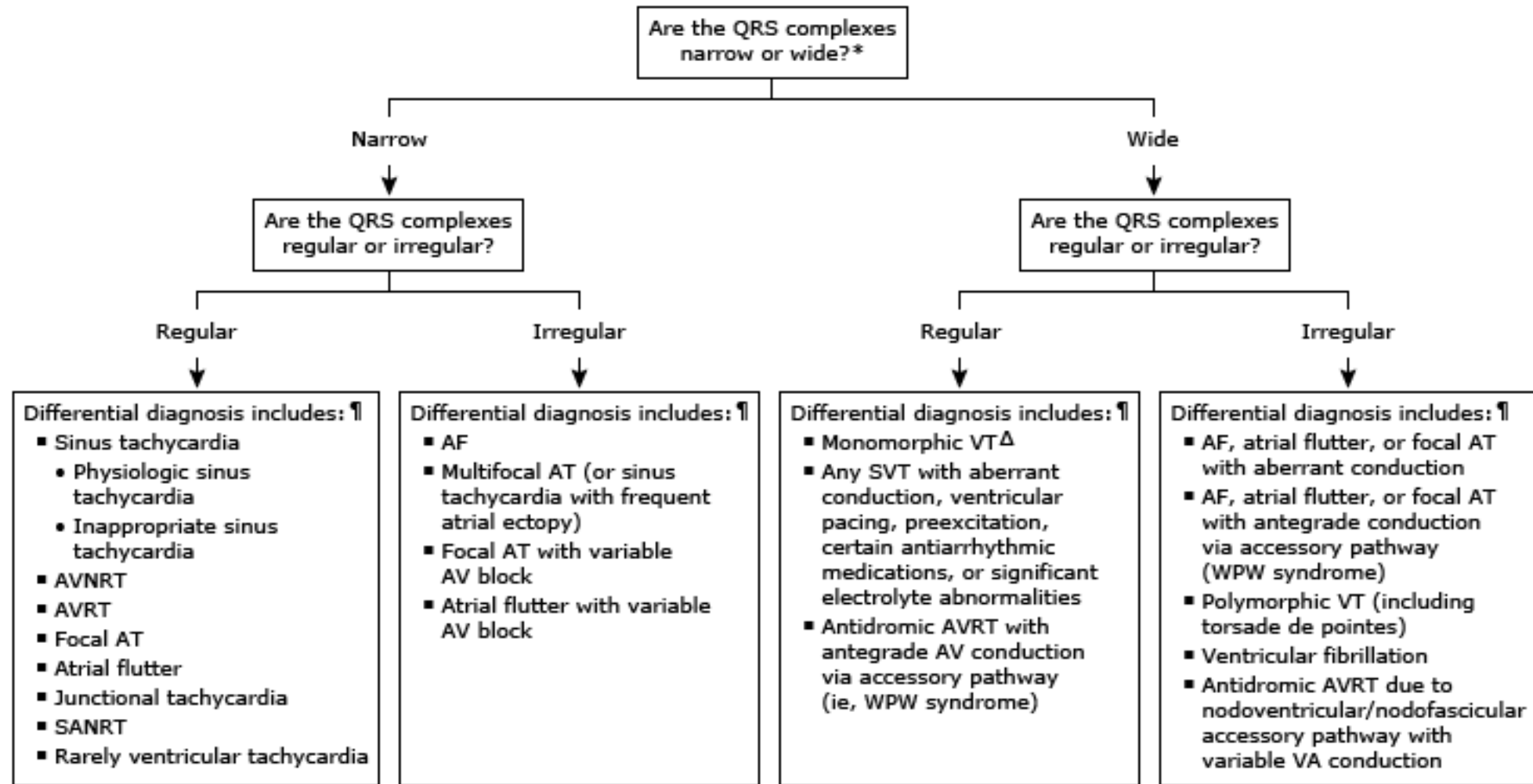
Is the patient hemodynamically stable?
Is the QRS narrow or wide?
Is the rhythm regular or irregular?

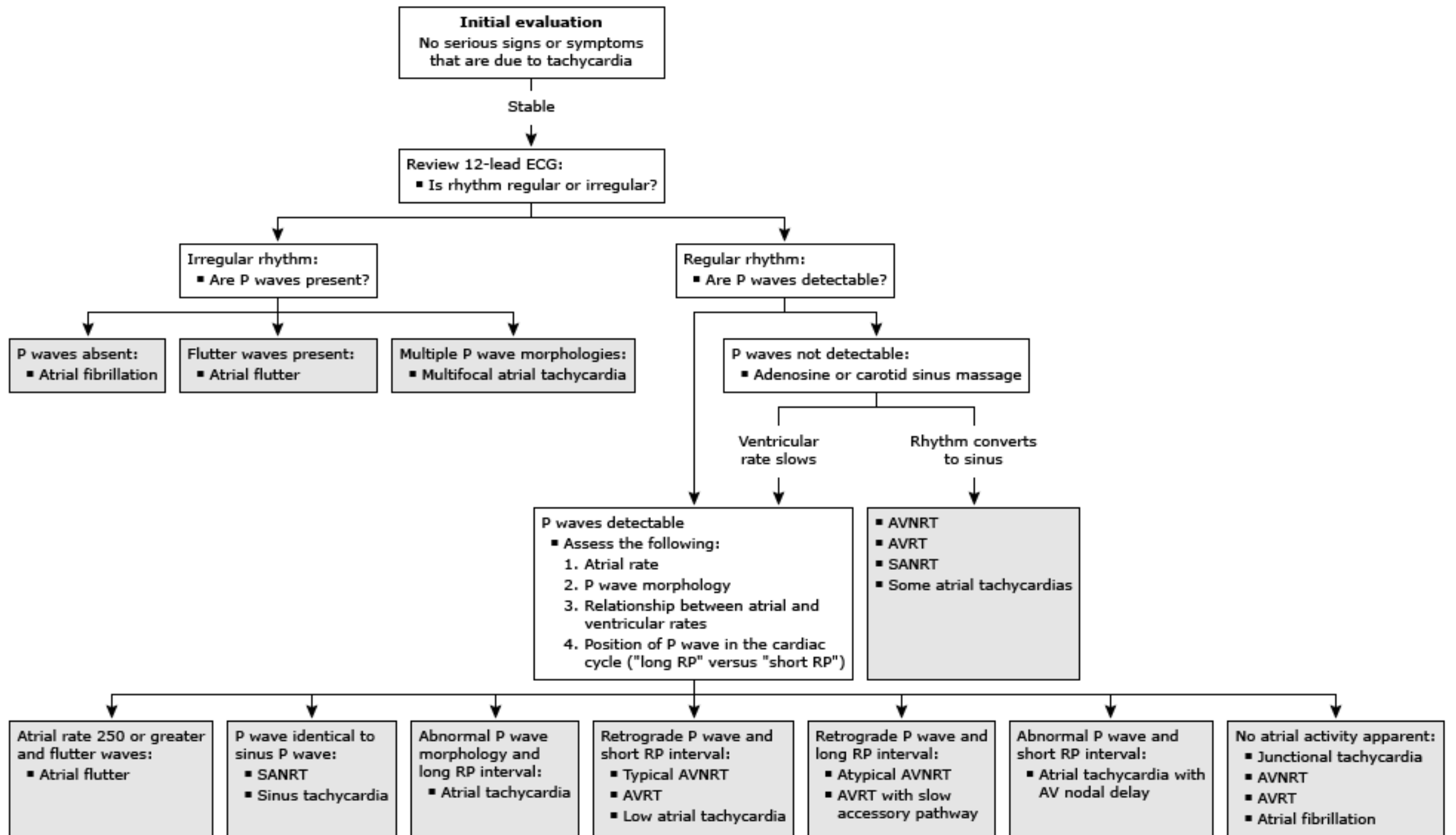
Questions?

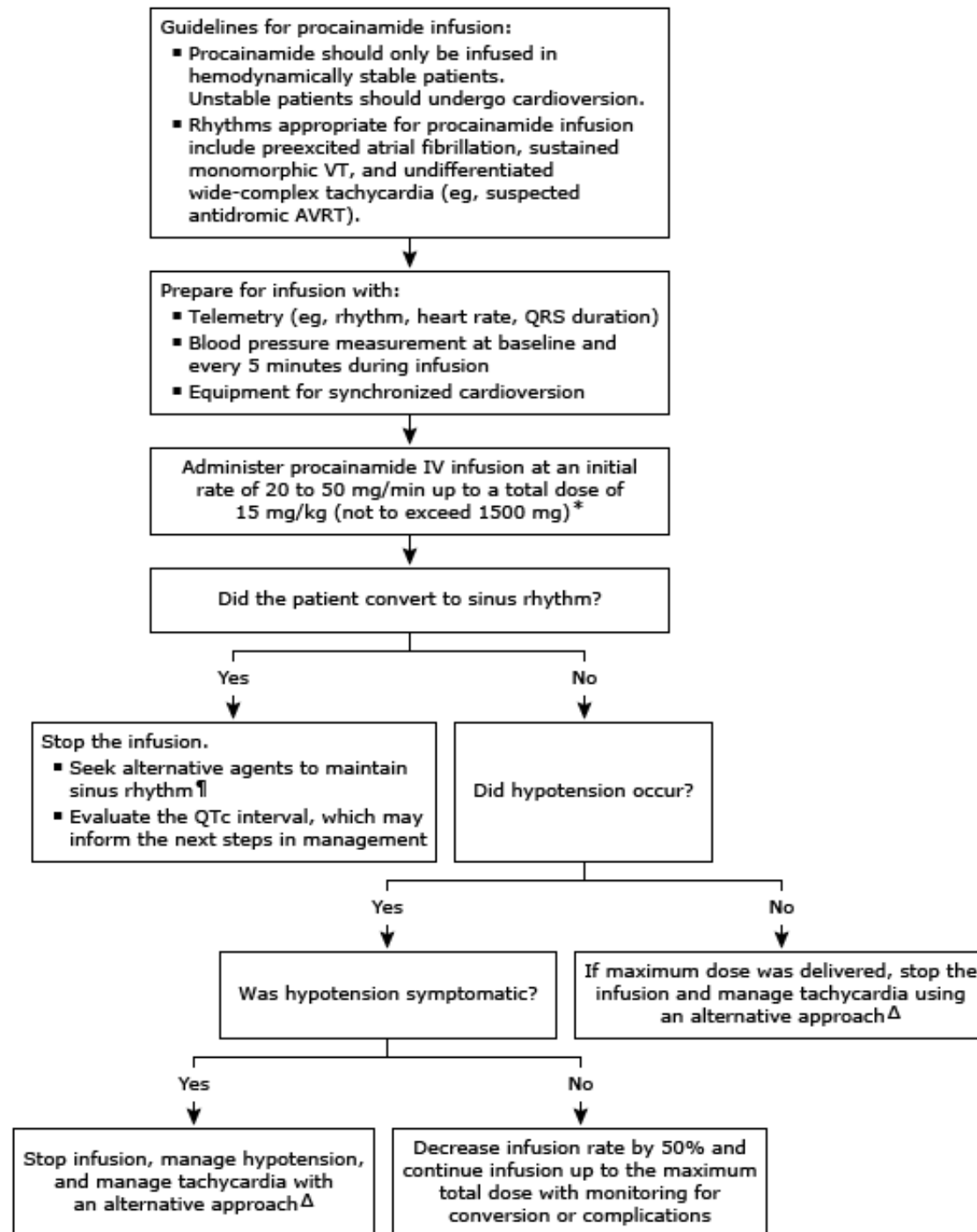


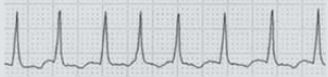
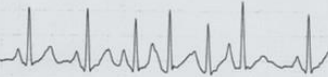
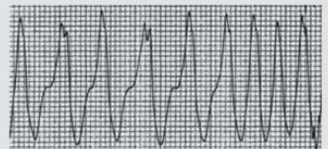


Differential diagnosis



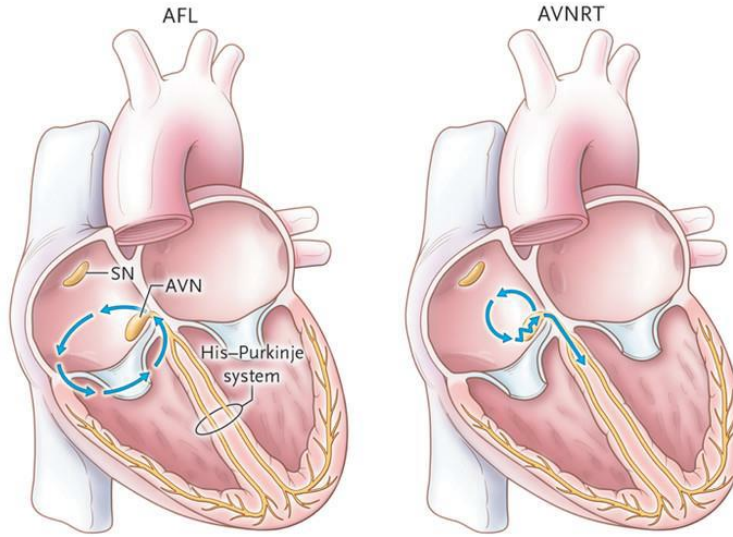




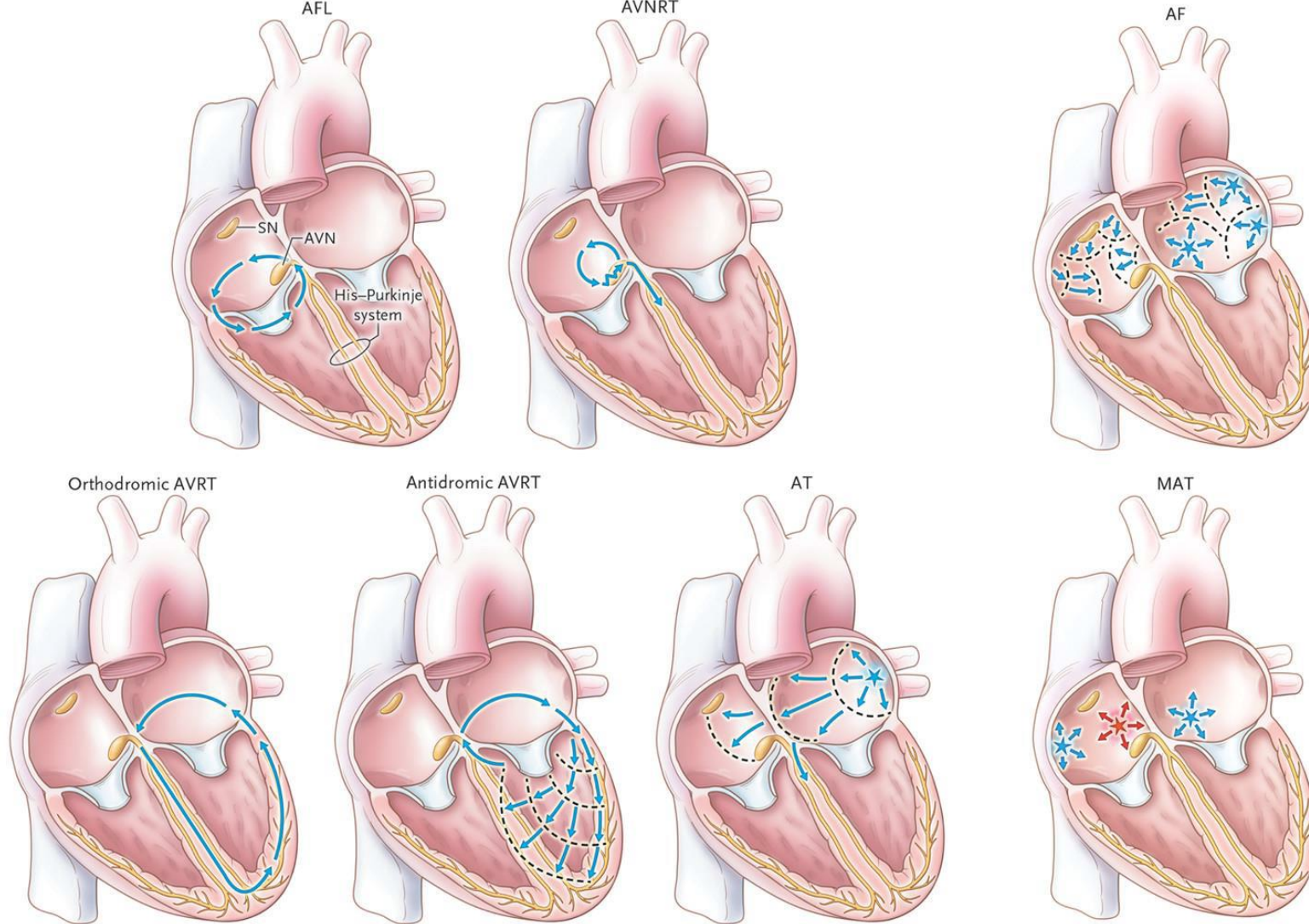
SVT	Underlying Causes	Regularity	Rate (beats/min)	Onset	Atrial Activity and P:QRS Relationship	Response to Adenosine	Electrocardiogram
Atrial fibrillation	Cardiac disease, pulmonary disease, pulmonary embolism, hyperthyroidism, postoperative	Irregular	100-220	Sudden or gradual (if in chronic atrial fibrillation)	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%	



Regular Supraventricular Tachycardias



Irregular Supraventricular Tachycardias



Parkland

UTSouthwestern
Medical Center

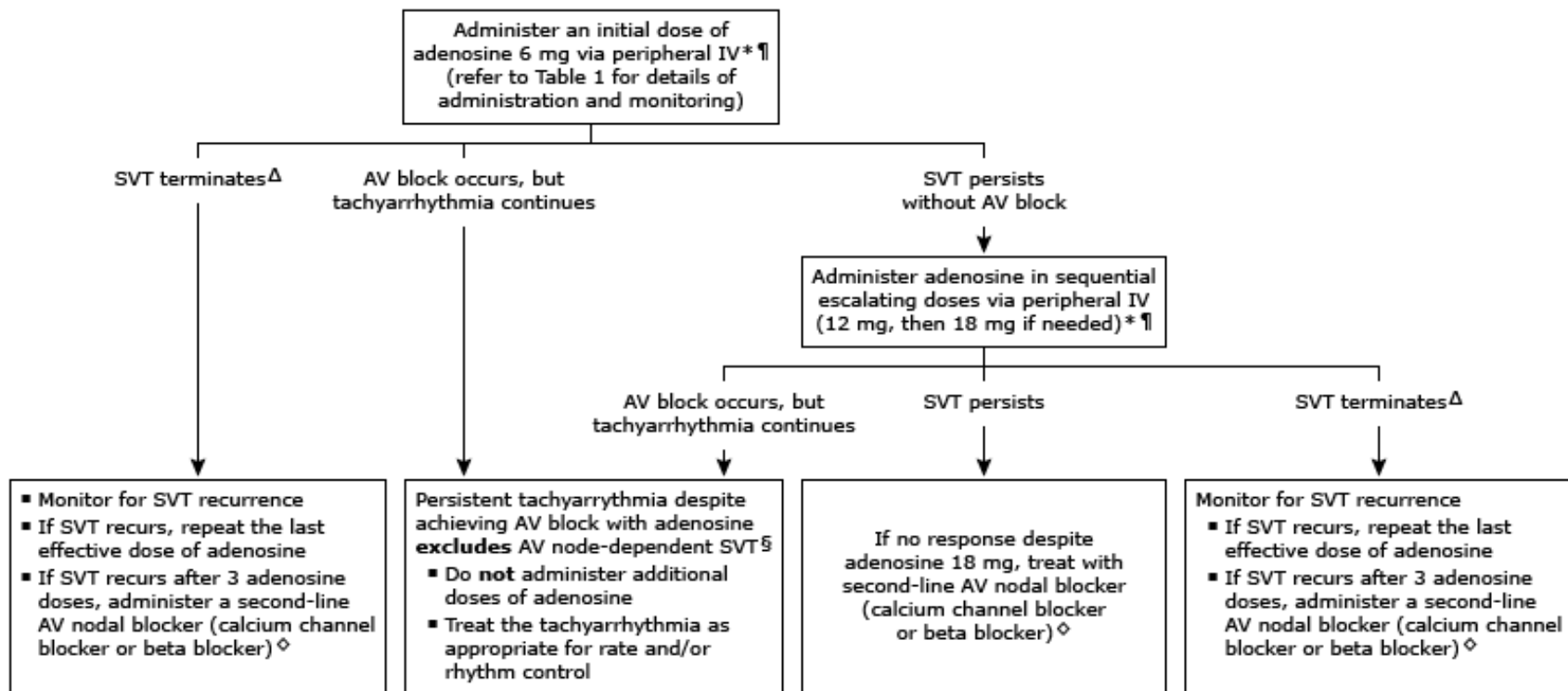


Table 1

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, antecubital 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.