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Tachyarrhythmias

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This session will be recorded

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Please also note:

The information in this presentation and the video recording is up to date as of the date it was recorded on 6/18/2020.

It has not been updated to include any subsequent advances in practice, and the information presented in this video does not replace hospital, health center, or governmental guidelines.



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Disclosures

- Nothing to disclose



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Learning Objectives

By the end of this session, residents should be able to:

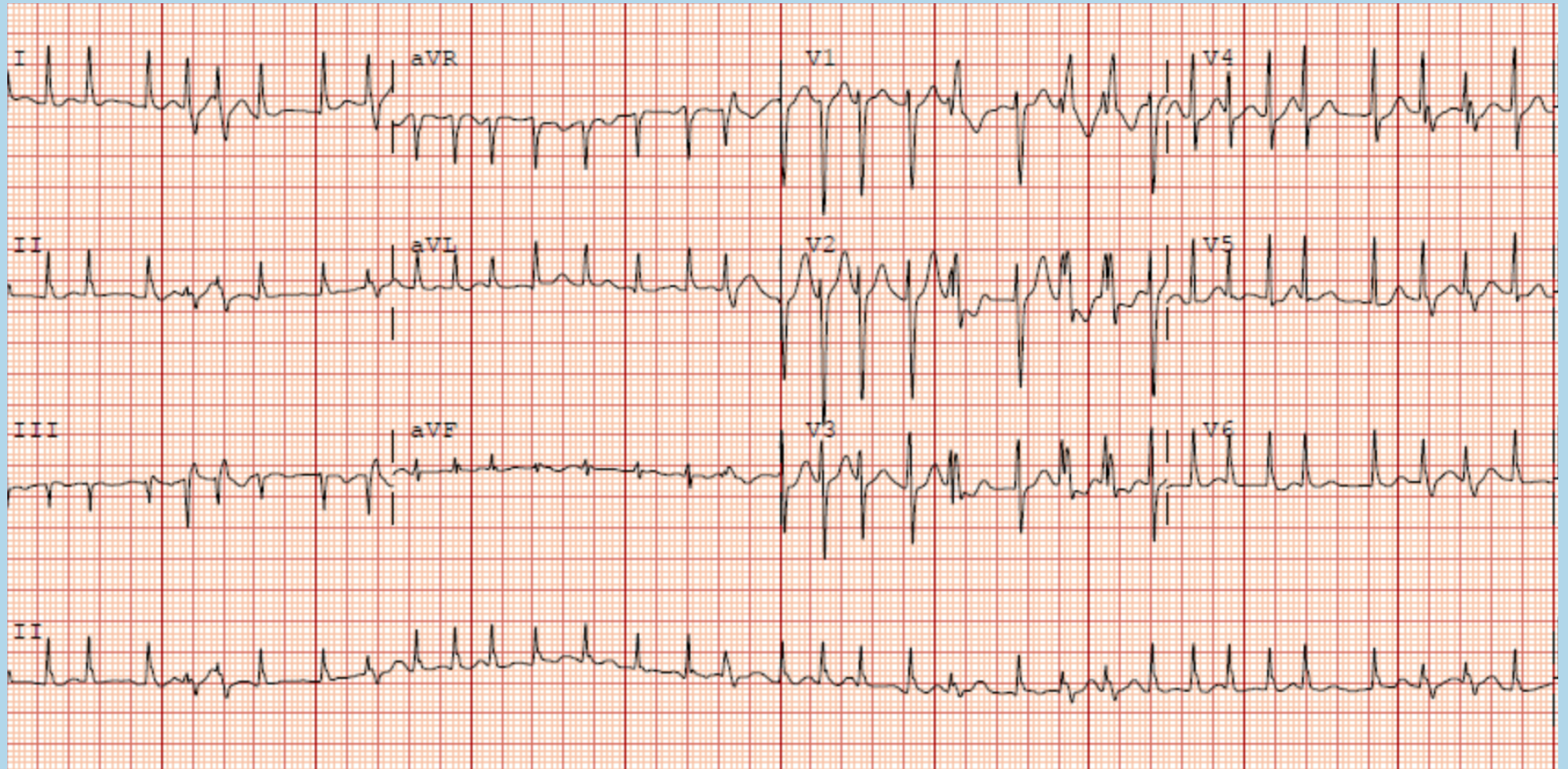
- Develop a standardized approach to tachyarrhythmias
- Differentiate between wide complex and narrow complex tachyarrhythmias
- Discuss mimics of ventricular tachycardia
- Differentiate between regular and irregular tachyarrhythmias
- Identify classes of anti-arrhythmics and general indications



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Case 1





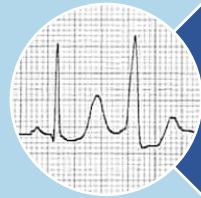
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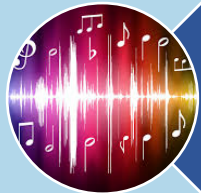
Standardized Approach



Rate



Narrow complex vs. Wide complex



Regular vs. Irregular



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Normal Rate

Regular rhythm

- Normal sinus rhythm
(P waves present)

Irregular rhythm

- Sinus arrhythmia
- SA block
- Sinus arrest
- Sick sinus syndrome
- PACs
- PVCs
- PJC's



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Bradycardia

Regular rhythm

- Sinus bradycardia
- Junctional rhythm (40-60 bpm)
- Idioventricular rhythm (30-50 bpm)
- AV block (1st and 3rd degree)

Irregular rhythm

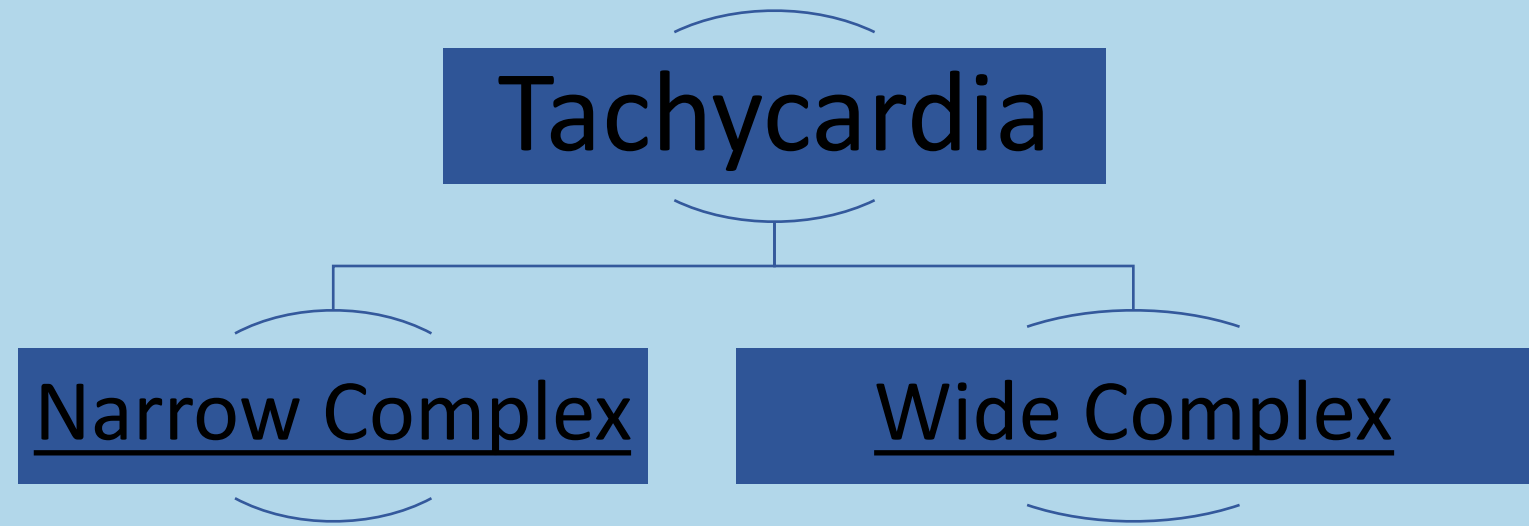
- 2nd degree AV Block



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Standardized Approach





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Narrow Complex Tachycardia

Regular rhythm

- Sinus tachycardia (P waves)
- Ectopic atrial tachycardia
- Atrial flutter
- Supraventricular tachycardia
 - Orthodromic AVRT
 - AVNRT
 - WPW

Irregular rhythm

- Atrial fibrillation
- Atrial flutter with variable conduction
- Multifocal atrial tachycardia



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Wide Complex Tachycardia

Regular rhythm

- SVT with aberrancy
- Antidromic AVRT
 - WPW
- Monomorphic ventricular tachycardia (rate >120)
- Accelerated idioventricular rhythm (rate 50-120)
- Sodium channel blockade
- Hyperkalemia

Irregular rhythm

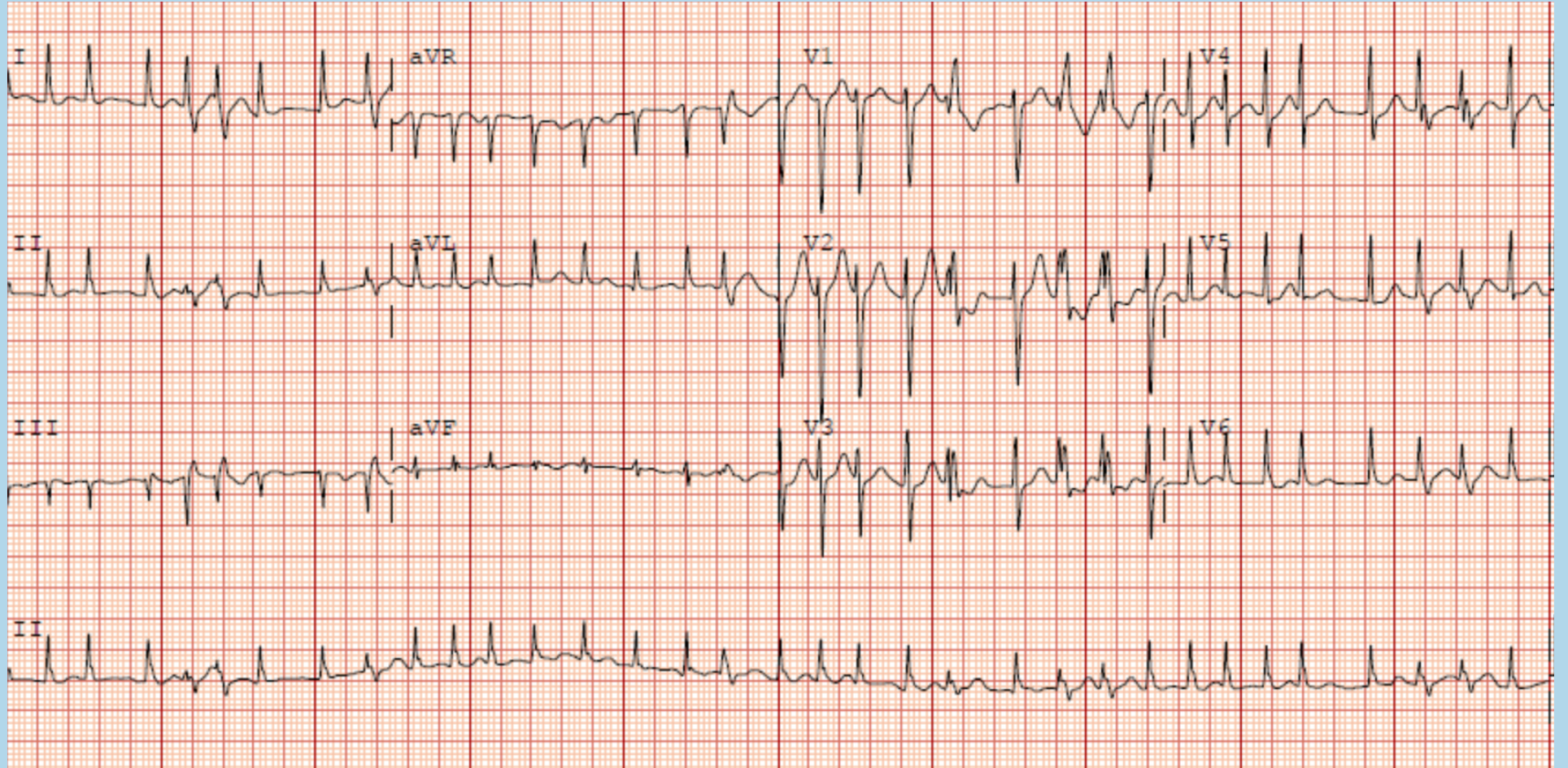
- WPW with a fib (rate >300)
- Atrial fibrillation with aberrancy (rate 200s)
- Polymorphic ventricular tachycardia
- Torsades de pointes
- Ventricular fibrillation



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Back to Case 1





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Narrow Complex Tachycardia

Regular rhythm

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Adult Tachycardia With a Pulse Algorithm

1

Assess appropriateness for clinical condition.
Heart rate typically $\geq 150/\text{min}$ if tachyarrhythmia.

2

Identify and treat underlying cause

- Maintain patent airway; assist breathing as necessary
- Oxygen (if hypoxemic)
- Cardiac monitor to identify rhythm; monitor blood pressure and oximetry

3

Persistent tachyarrhythmia causing:

- Hypotension?
- Acutely altered mental status?
- Signs of shock?
- Ischemic chest discomfort?
- Acute heart failure?

Yes

4

Synchronized cardioversion

- Consider sedation
- If regular narrow complex, consider adenosine

No

5

Wide QRS? ≥ 0.12 second

Yes

6

- IV access and 12-lead ECG if available
- Consider adenosine only if regular and monomorphic
- Consider antiarrhythmic infusion
- Consider expert consultation

No

7

- IV access and 12-lead ECG if available
- Vagal maneuvers
- Adenosine (if regular)
- β -Blocker or calcium channel blocker
- Consider expert consultation

Doses/Details

Synchronized cardioversion:

Initial recommended doses:

- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (*not* synchronized)

Adenosine IV dose:

First dose: 6 mg rapid IV push; follow with NS flush.

Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV dose:

20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases $>50\%$, or maximum dose 17 mg/kg given. Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

Amiodarone IV dose:

First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV dose:

100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.



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Atrial fibrillation

- Uncoordinated atrial activity – no P waves
- Irregularly irregular
- Variable ventricular response rate

Treatment:

- >48 hrs: rate control + anti-coagulation
- <48 hrs: consider synchronized cardioversion vs rhythm control



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A fib with RVR

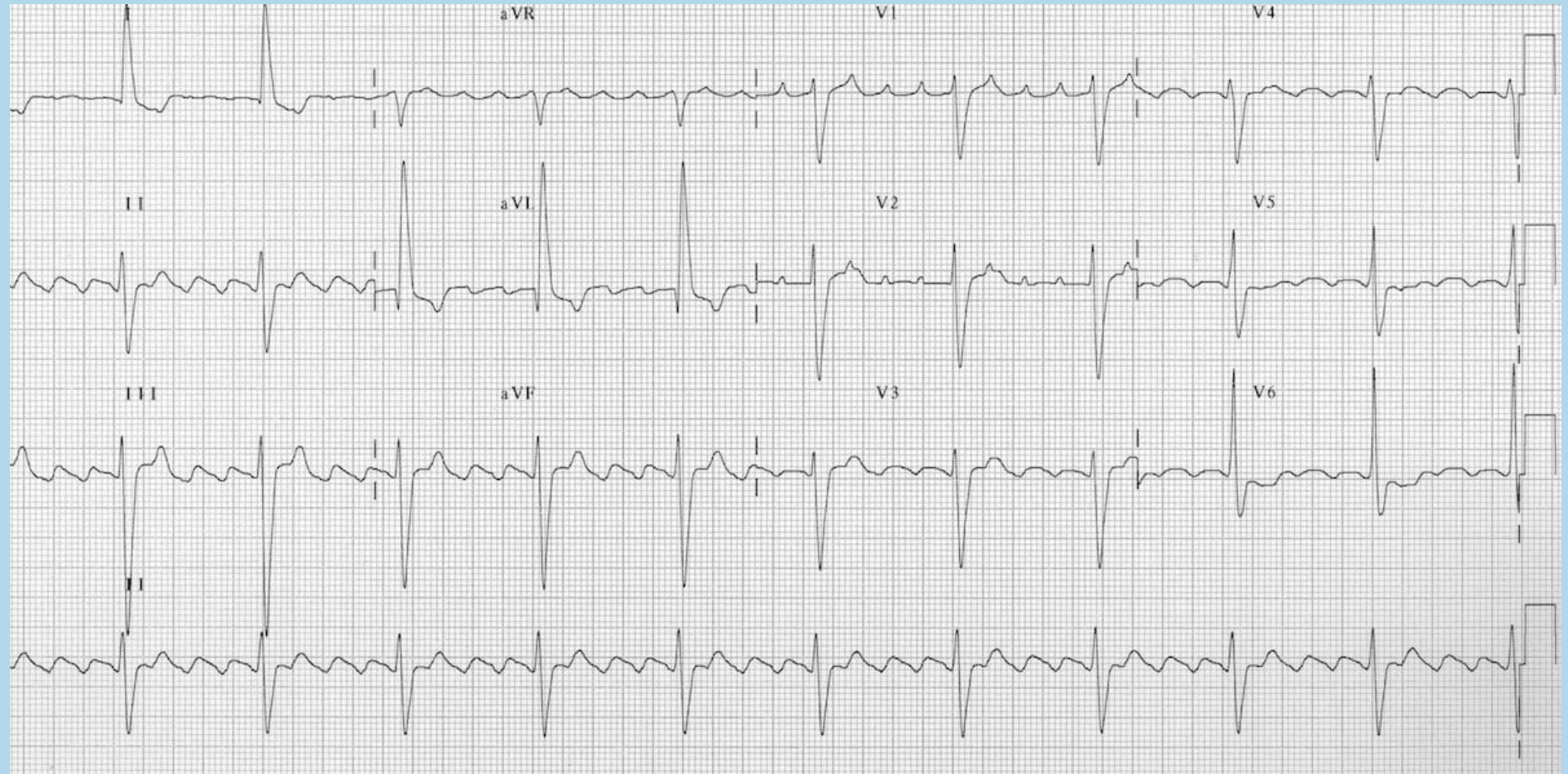
- Unstable -> synchronized cardioversion, push dose phenylephrine
- Stable:
 - Wait. Think.
 - Underlying cause
 - Normal EF -> Diltiazem 0.25 mg/kg IV bolus
 - Low EF -> Metoprolol 5 mg IV q 5 min x3
 - AKI/CKD -> Amiodarone



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Case 2



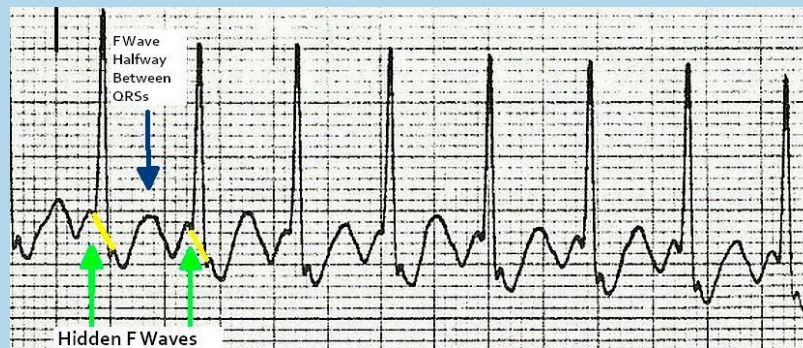


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Atrial flutter

- Organized electrical activity
- Classically 2:1 conduction (rate 150)
- Sometimes hard to see!
 - Look at leads II, III and aVF
 - Unmask flutter waves with adenosine (if SVT vs A flutter)
 - Bix rule



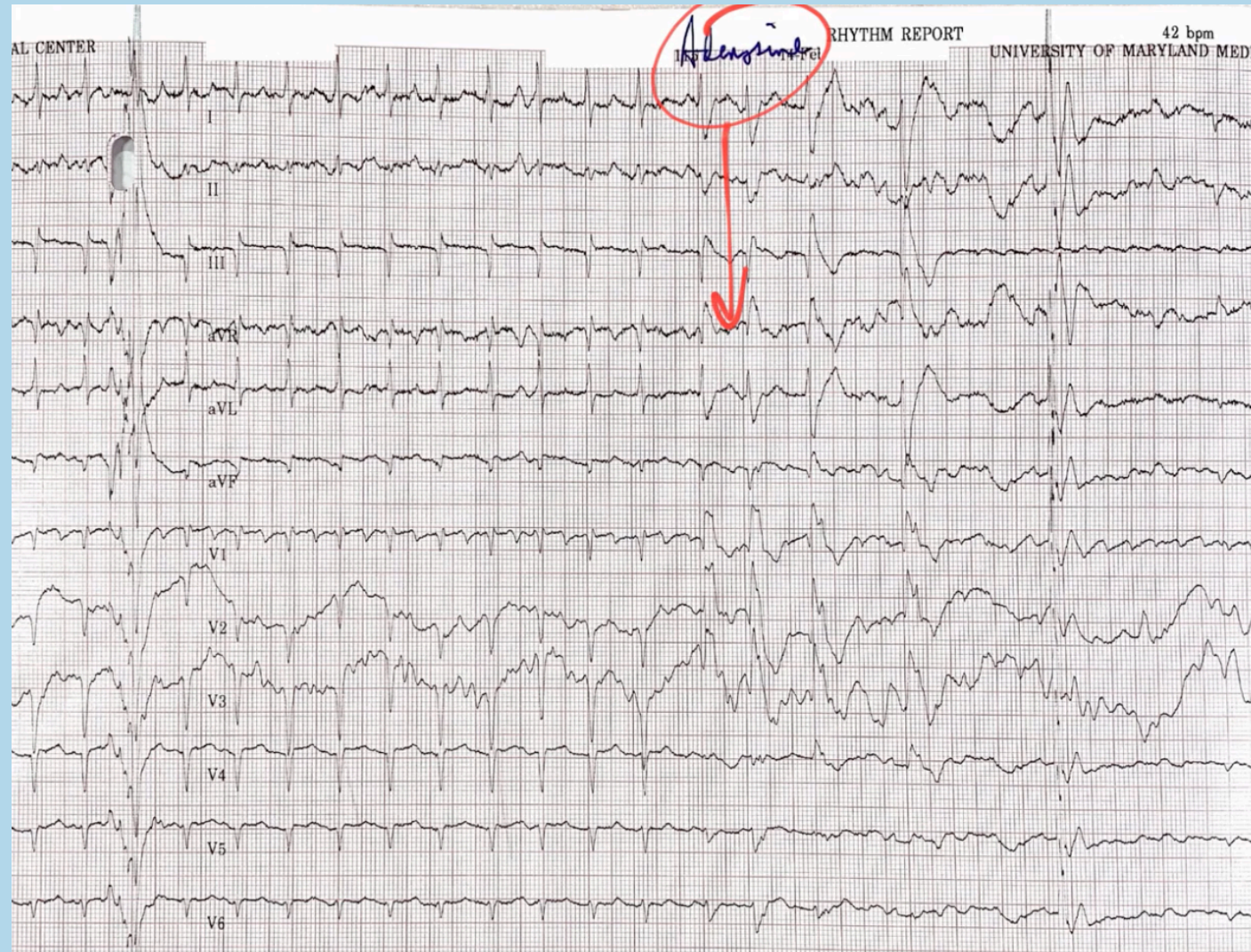
- Management: same as atrial fibrillation



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Adenosine unmasking A Flutter

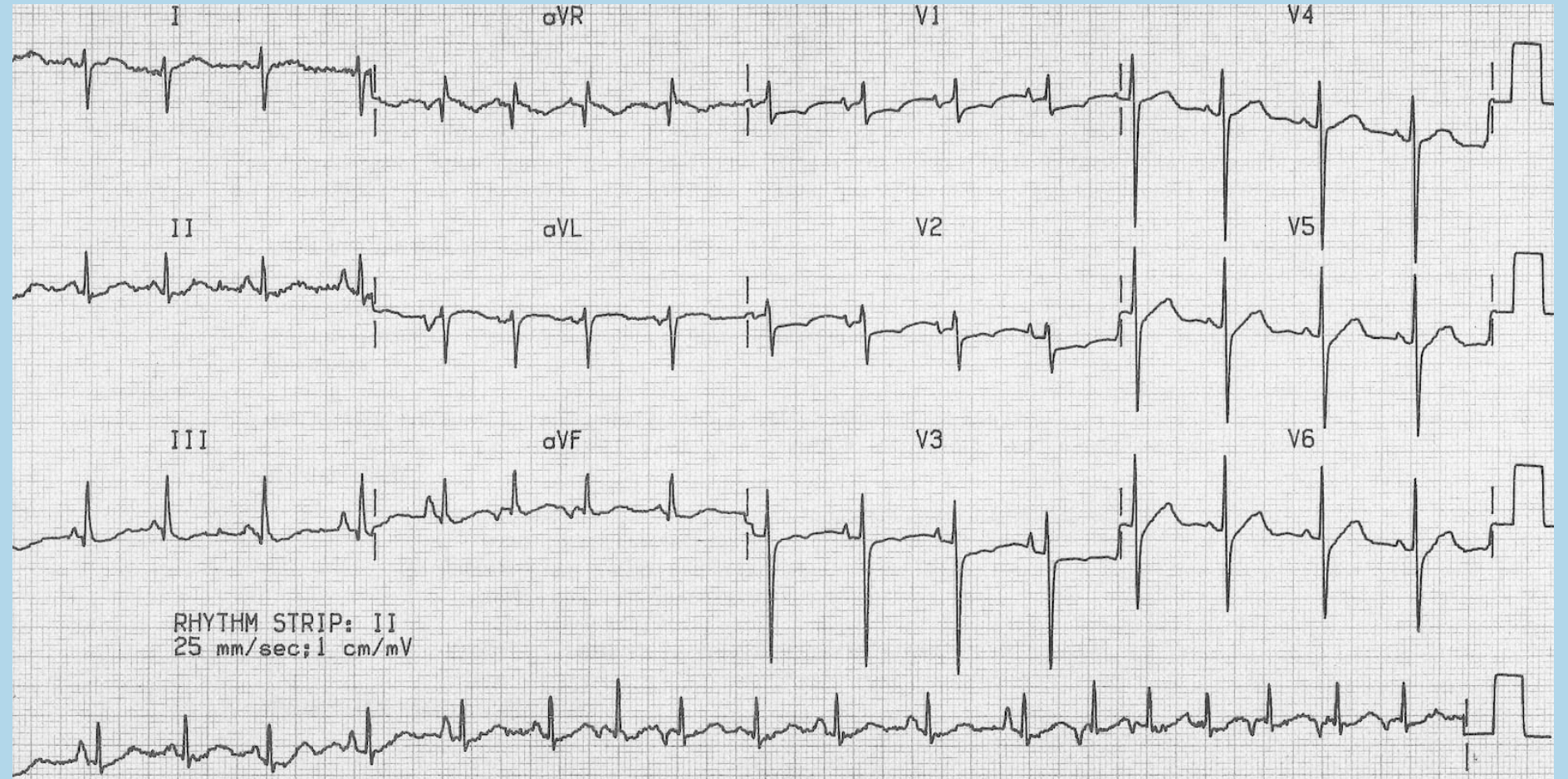




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Case 3





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Multifocal atrial tachycardia

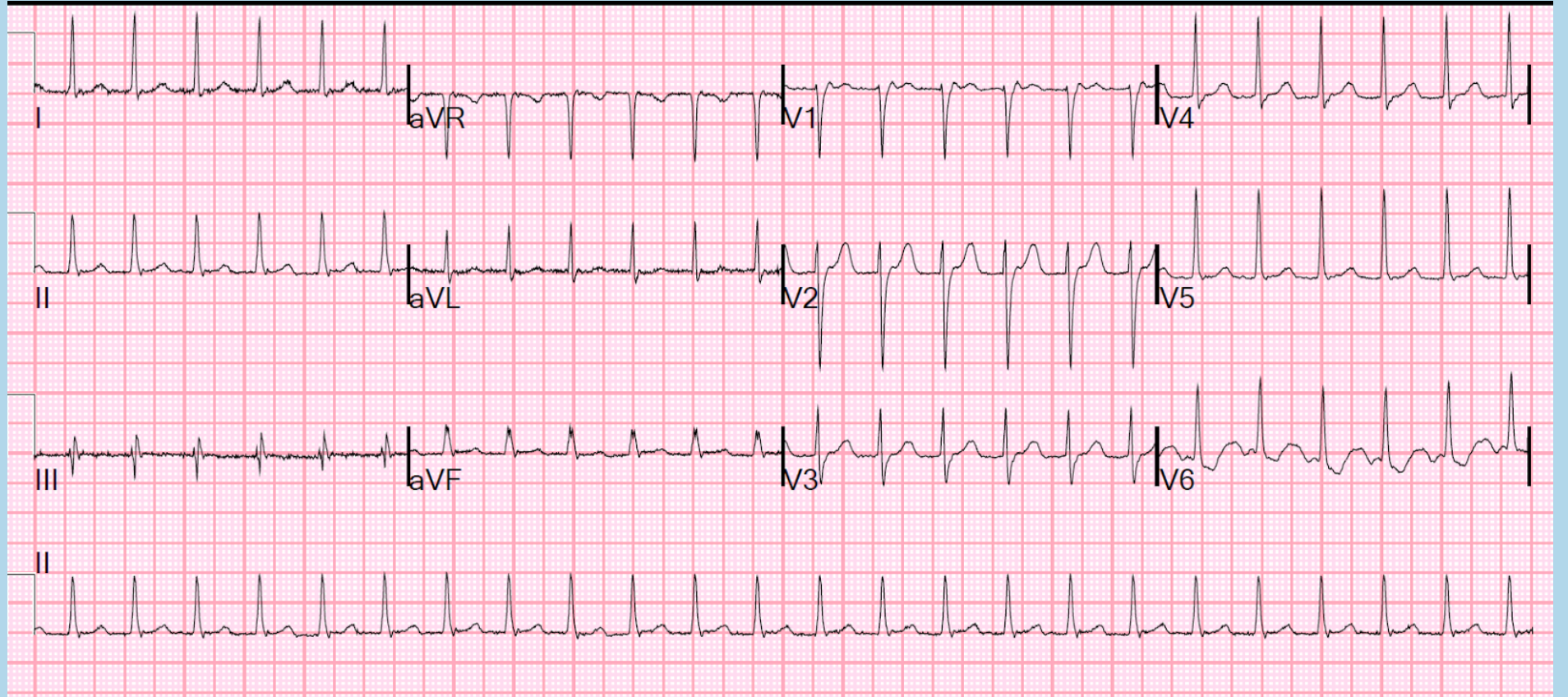
- At least 3 different P wave morphologies
- Rate 100-180, irregular
- Causes: COPD, pulmonary HTN, hypoxia
- Exacerbated by hypoK and hypoMg
- Management:
 - **Treatment of underlying condition**
 - Oxygen
 - Rate control (calcium channel blockers)



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Case 4





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Narrow Complex Tachycardia

Regular rhythm

- Sinus tachycardia (P waves)
- Ectopic atrial tachycardia
- Atrial flutter
- Supraventricular tachycardia
 - Orthodromic AVRT
 - AVNRT
 - WPW

Irregular rhythm

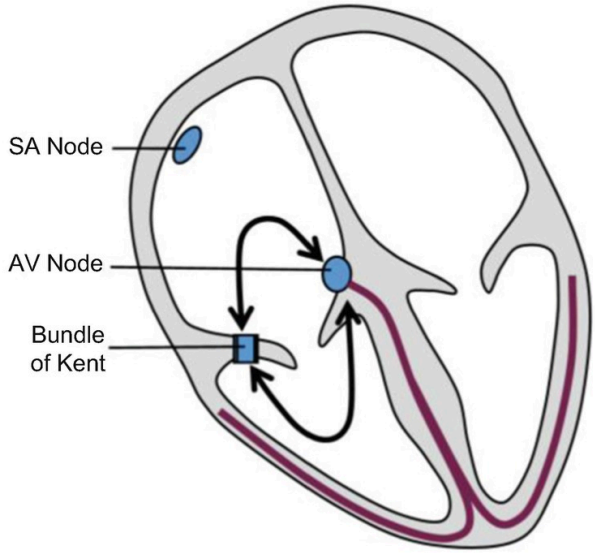
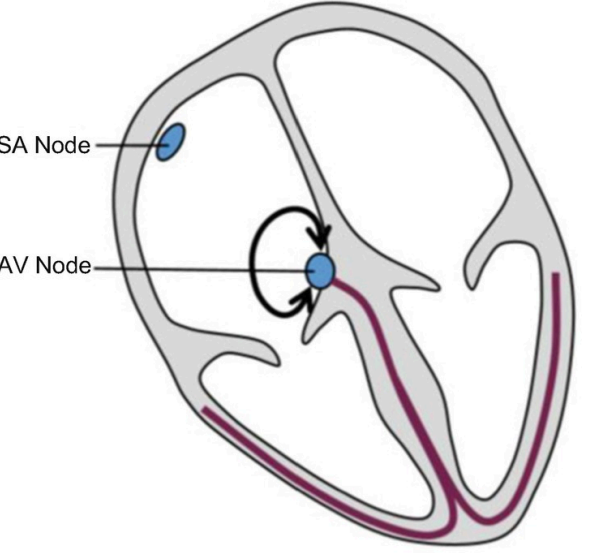
- Atrial fibrillation
- Atrial flutter with variable conduction
- Multifocal atrial tachycardia



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AVRT vs. AVNRT

 <p>The diagram shows a cross-section of the heart's conduction system. The SA Node is at the top left, the AV Node is in the center, and the Bundle of Kent is a blue box on the left. Arrows show the electrical pathway: from the SA Node to the AV Node, down the Bundle of Kent to the ventricles, and back up to the AV Node, creating a reentrant loop.</p>	 <p>The diagram shows a cross-section of the heart's conduction system. The SA Node is at the top left, and the AV Node is in the center. Arrows show the electrical pathway: from the SA Node to the AV Node, and a circular arrow indicates a reentrant loop within the AV Node itself.</p>
<p>In Atrioventricular Reciprocating Tachycardia (AVRT), there is an anomalous pathway between the atria and ventricles called the Bundle of Kent.</p>	<p>In comparison, AV Nodal Reentrant Tachycardia (AVNRT), often synonymous with PSVT, there is no <i>anatomical</i> pathway. Instead, there is a <i>functional</i> pathway within the AV node</p>
<p>ECG morphology: In both AVRT and AVNRT there is a narrow complex without visible p-waves. It is very difficult to distinguish between AVRT and AVNRT based on electrocardiographic criteria alone.</p>	



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AV Nodal Re-entry Tachycardia

- Most common re-entrant rhythm in ED
- P waves hidden in QRS, sometimes retrograde
- Causes: caffeine, drugs, exertion, idiopathic
- Approach:
 - Vagal Maneuvers
 - Modified Valsalva
 - Adenosine 6 mg, then 12 mg
 - Synchronized cardioversion
- NOTE: sometimes causes ST depressions that resolve after cardioversion



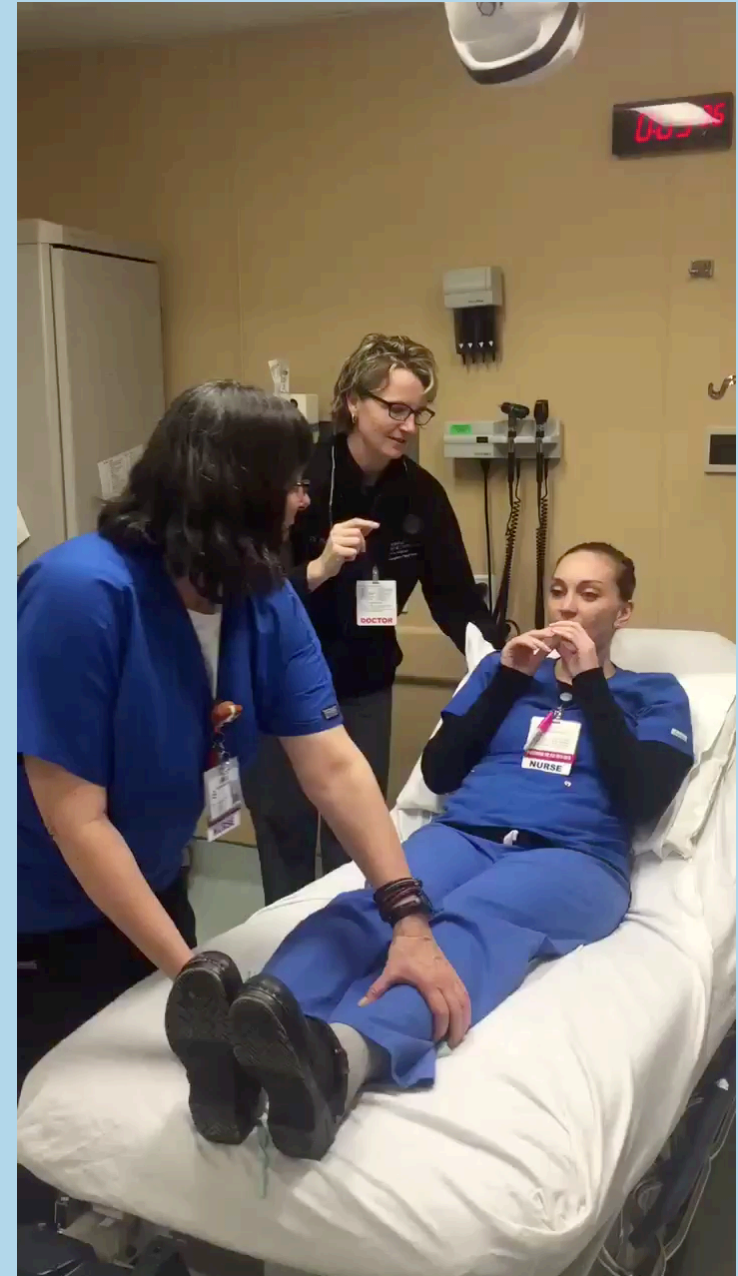
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Modified Valsalva

REVERT Trial

43% vs 17% response
compared to the
standard Valsalva
maneuver

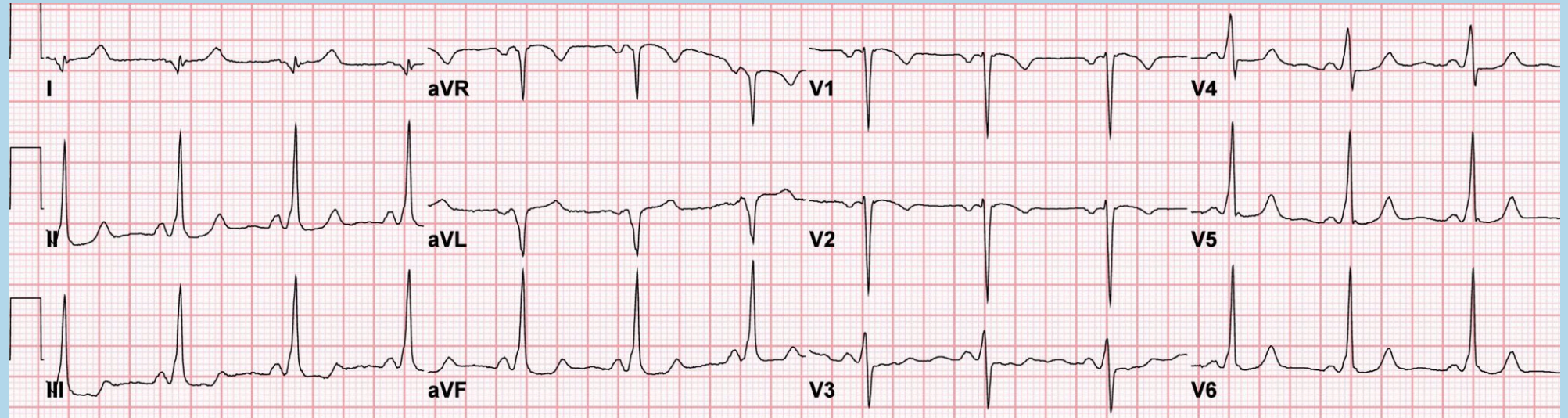




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Case 5



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AV Re-entry Tachycardia

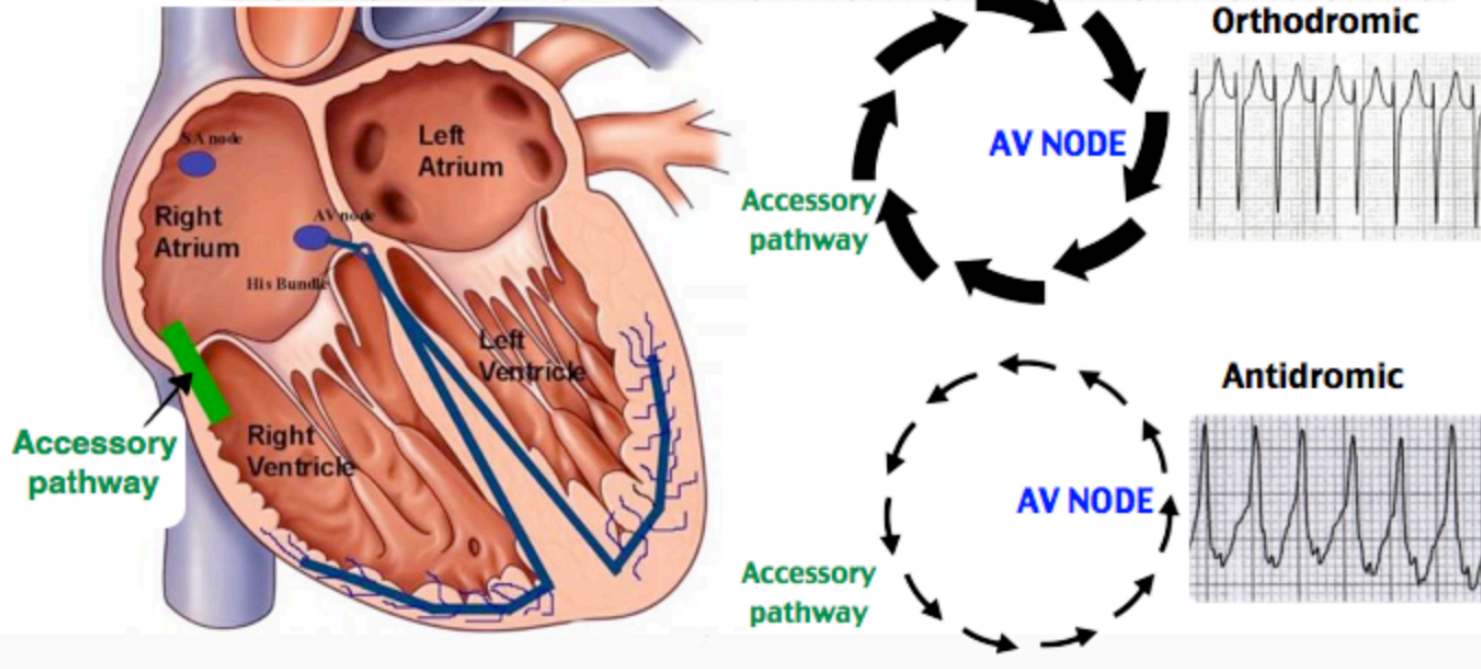
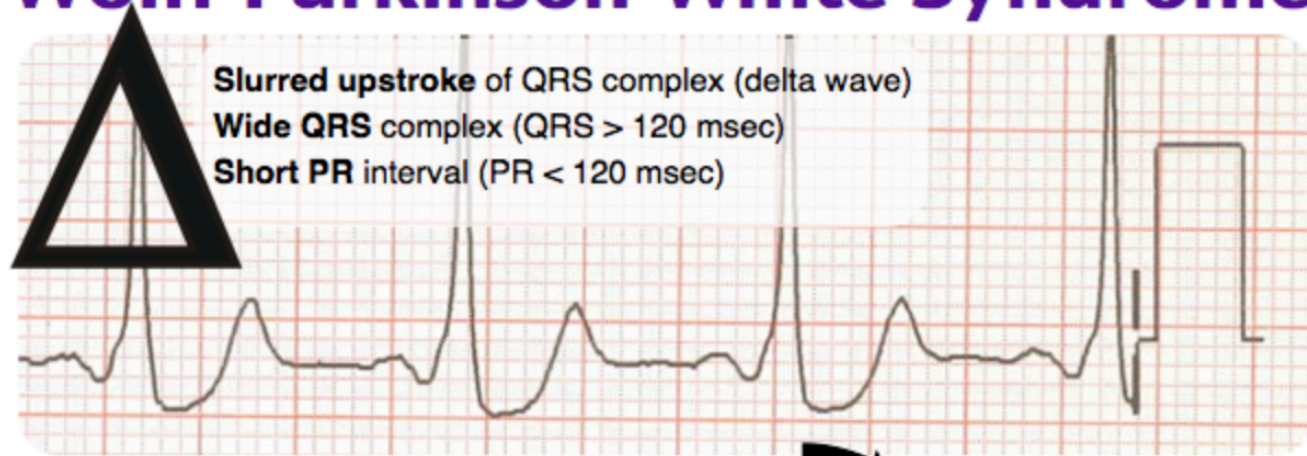
- Anatomic re-entry circuit (Bundle of Kent)
- Orthodromic - narrow complex
- Antidromic – wide complex
- Irregular – a fib + AVRT
 - Do NOT block the AV node. This can be fatal
 - Avoid adenosine, BB, CCB, digoxin and amiodarone
 - DRUG OF CHOICE: Procainamide OR synchronized cardioversion



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Wolff-Parkinson-White Syndrome

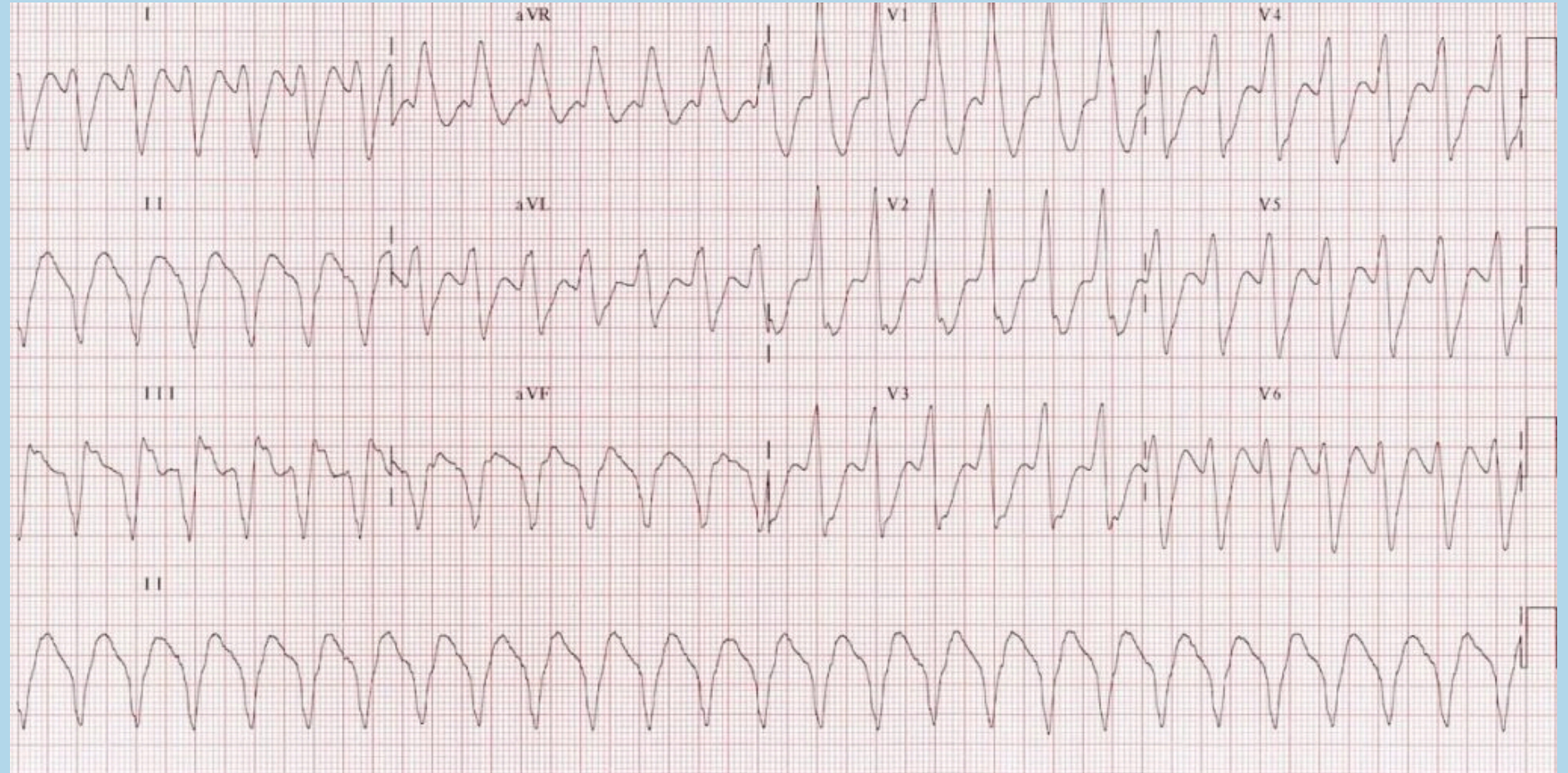




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Case 6





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Wide Complex Tachycardia

Regular rhythm

- SVT with aberrancy
- Antidromic AVRT
 - WPW
- Monomorphic ventricular tachycardia (rate >120)
- Accelerated idioventricular rhythm (rate 50-120)
- Sodium channel blockade
- Hyperkalemia

Irregular rhythm

- WPW with a fib (rate >300)
- Atrial fibrillation with aberrancy (rate 200s)
- Polymorphic ventricular tachycardia
- Torsades de pointes
- Ventricular fibrillation



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Monomorphic V Tach

- Causes: prior MI (rarely new ischemia), structural abn, electrolyte abn, hypoxia
- Sustained (>30 seconds or instability) vs non-sustained
- VT Storm = > 3 episodes of sustained V Tach in 24 hrs (responds to therapy but recurs)
- Refractory V Tach – not responding to interventions



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Monomorphic V Tach

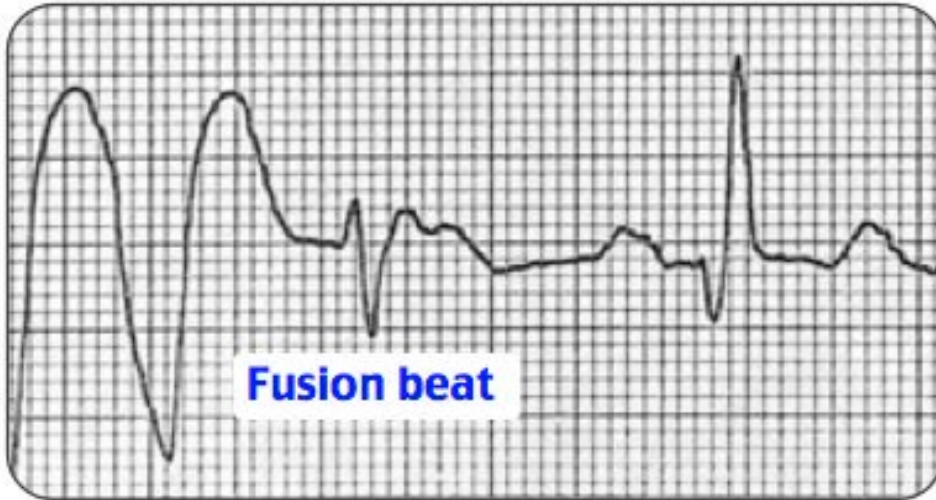
- Key EKG findings:
 - Rate >120 , QRS >140
 - Extreme (NW) axis – QRS positive in aVR, negative in I and aVF
 - AV dissociation
 - Concordant positive/negative precordial QRS complexes
 - RSR' complexes with $R > R'$ (most specific)
 - Fusion and capture beats
 - Vereckei, Brugada, Pava



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Ventricular Tachycardia



- **Supraventricular** and a **ventricular impulse** coincide to produce a **hybrid complex**
- Indicates there are **2 foci** of pacemaker cells firing simultaneously



- The return of **atrial control** over ventricular contraction, following a period of atrioventricular dissociation



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Monomorphic V Tach

- Management
 - Underlying cause
 - Non-sustained – amiodarone, magnesium
 - Sustained:
 - +Pulse - Cardioversion
 - Unstable – synchronized cardioversion
 - Stable – procainamide, amiodarone, lidocaine
 - Pulseless – ACLS + DEFIB

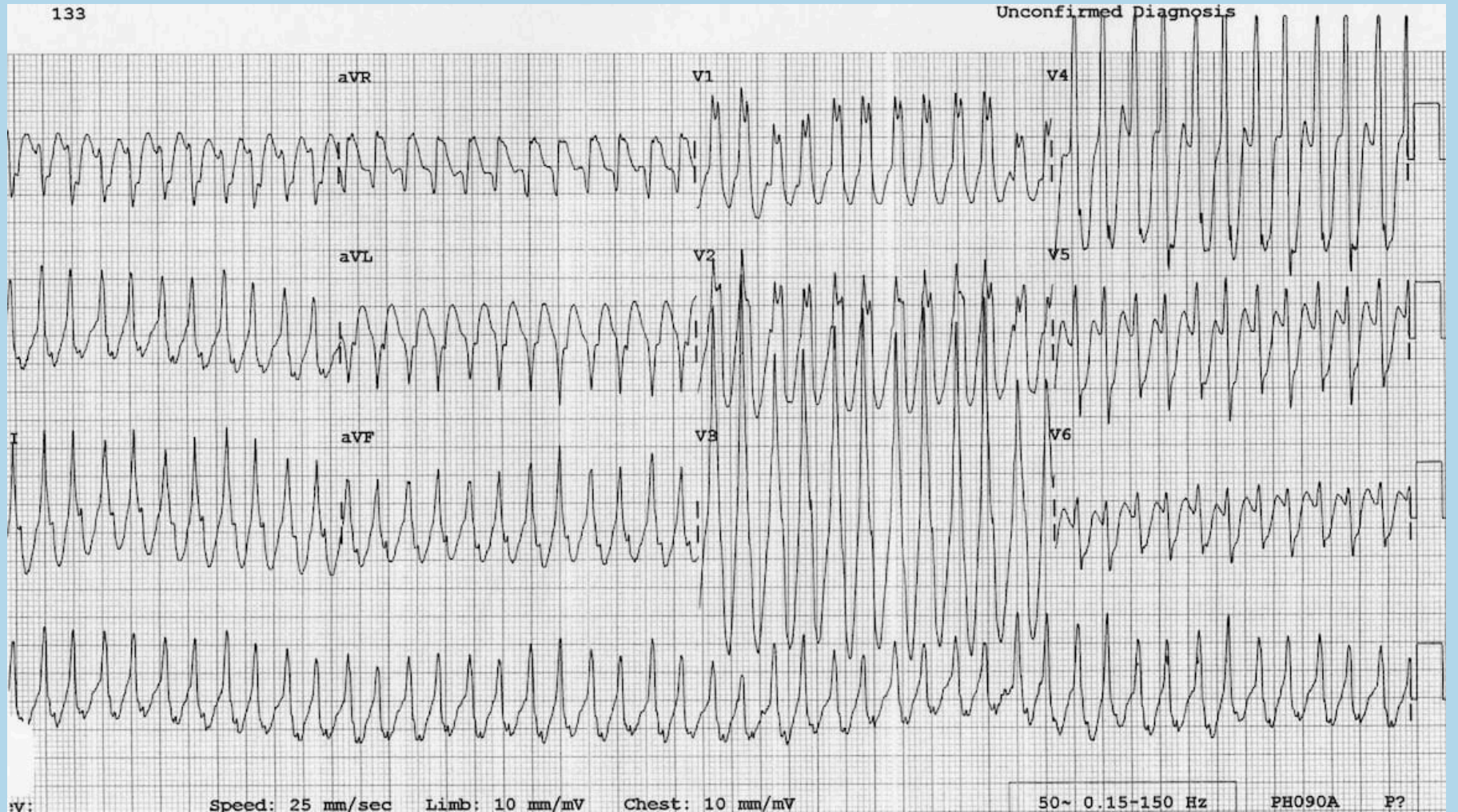


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Case 7

- A. Monomorphic V Tach
- B. SVT with aberrancy (LBBB)
- C. SVT with aberrancy (WPW)



<https://litfl.com/vt-versus-svt-ecg-library/>

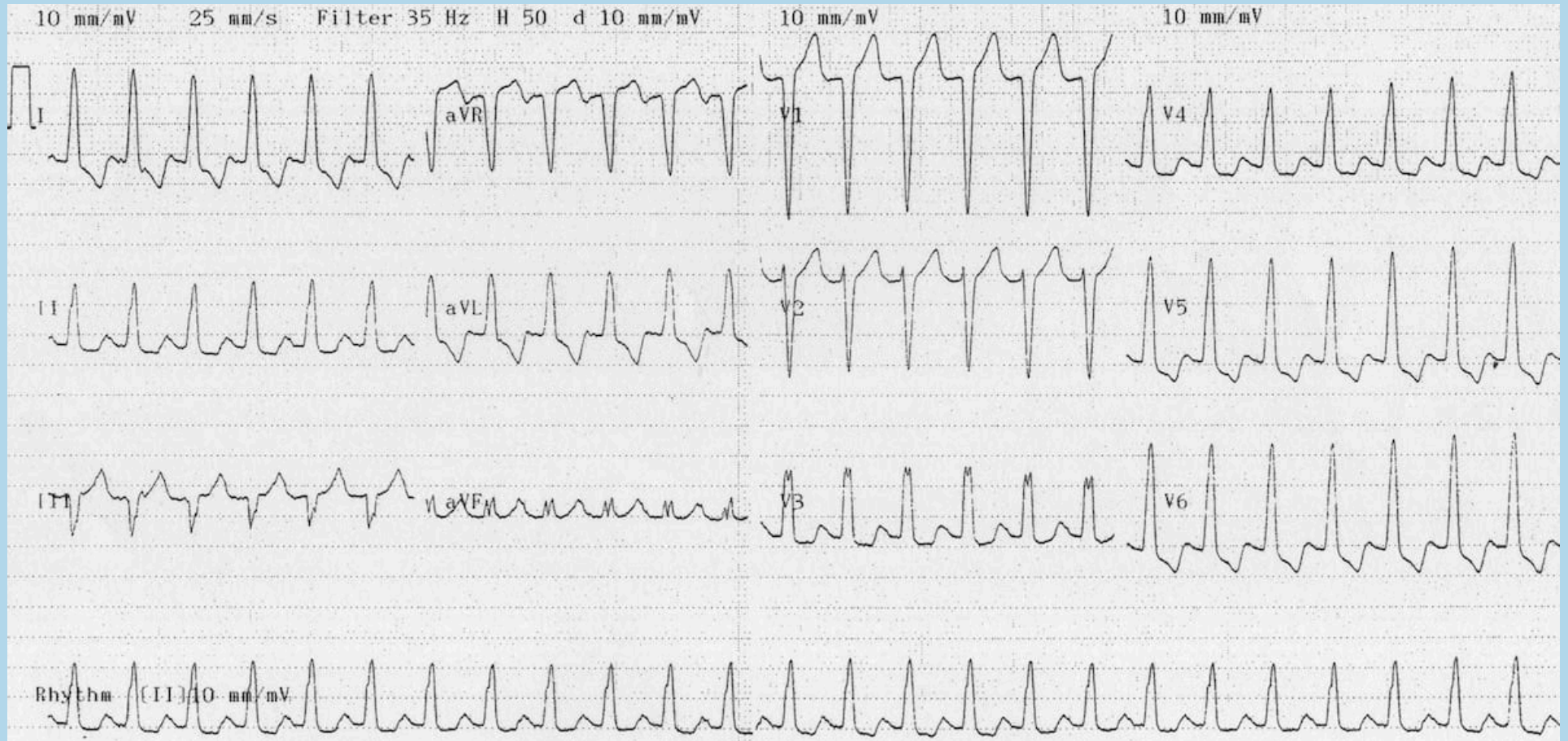


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Case 8

- A. Monomorphic V Tach
- B. SVT with aberrancy (LBBB)**
- C. SVT with aberrancy (WPW)





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Monomorphic V Tach mimics

- SVT with aberrancy – Lewis lead
- Antidromic WPW – very difficult to differentiate
- Hyperkalemia
- Sodium channel blockade

- Approach
 - Past medical history, HPI
 - Look at prior EKG (h/o LBBB vs pre-excitation)
 - POC lytes



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Lewis Lead

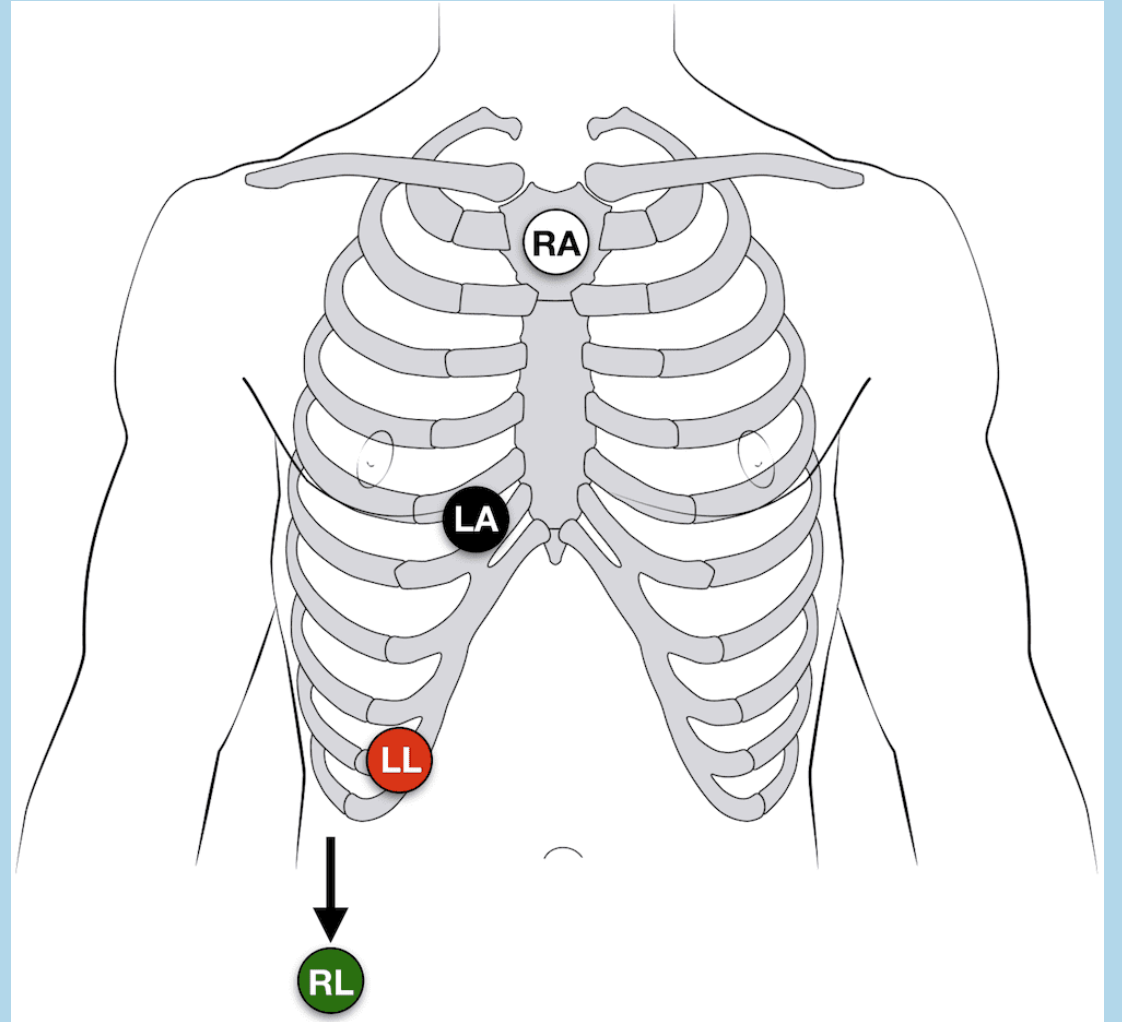
Move RA to
manubrium (below
suprasternal notch)

Move LA to R 5th
ICS next to sternum

Move LL to R lower
costal margin

Keep all others the
same

Lewis lead = Lead I

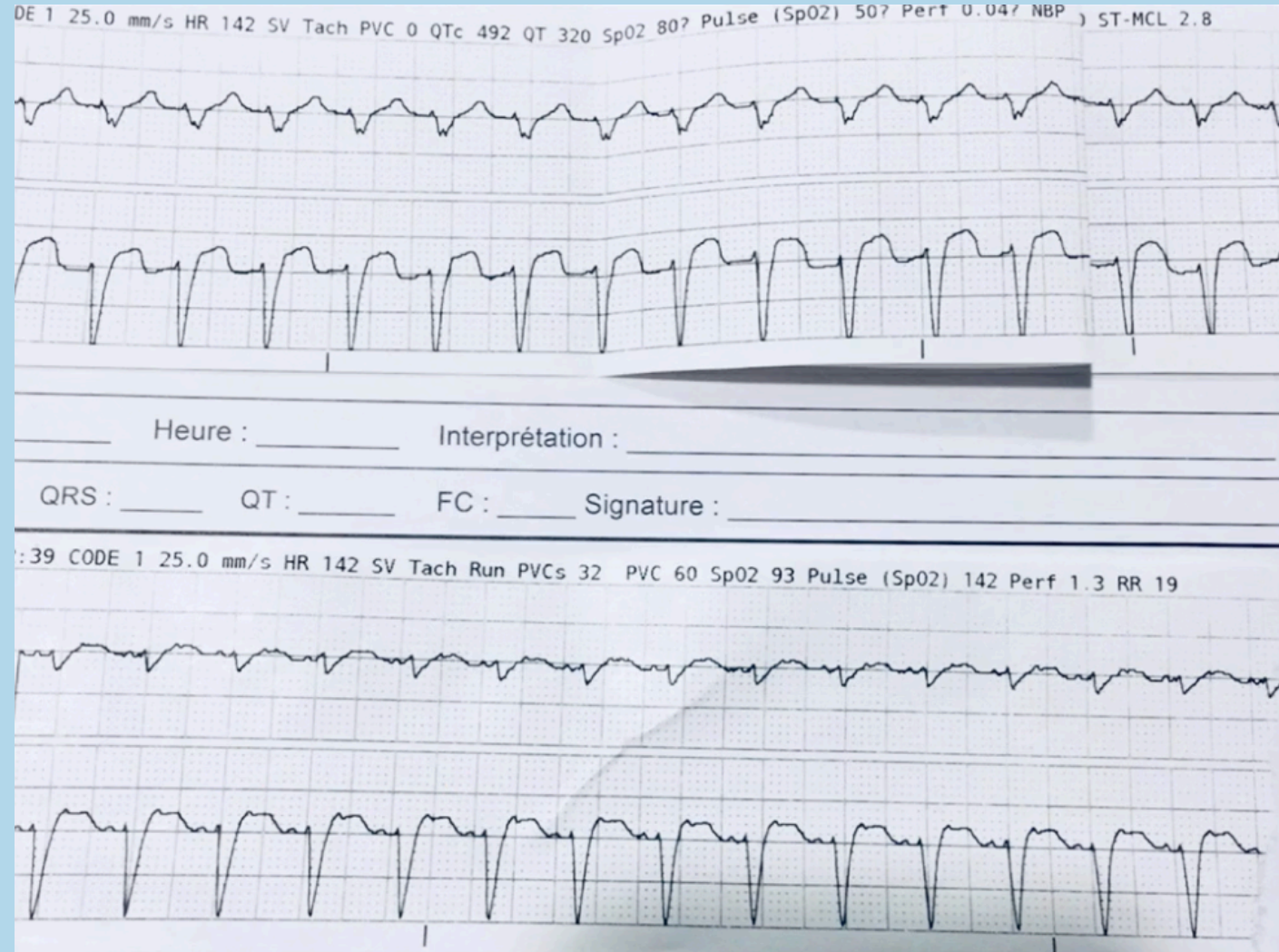




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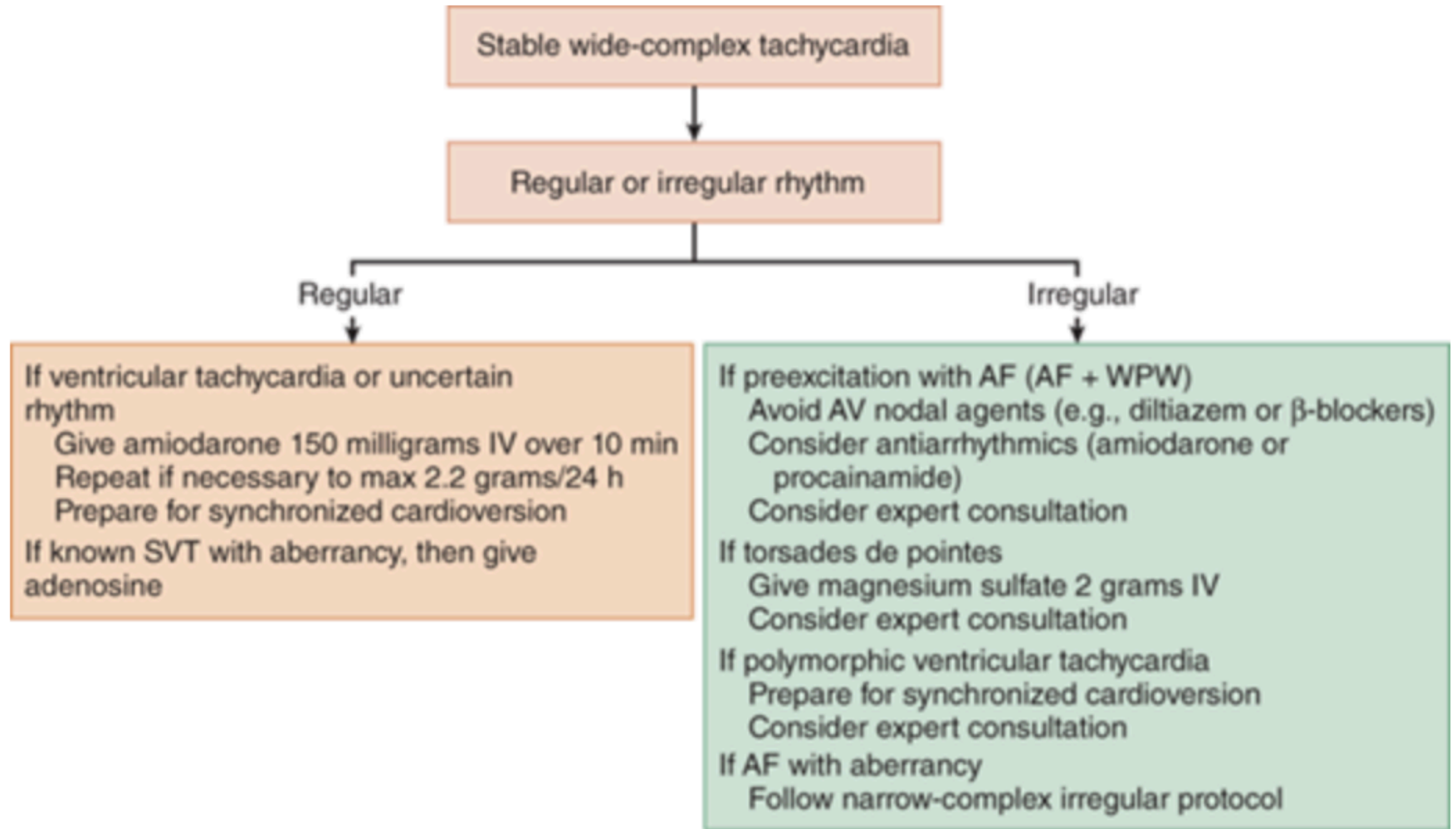
Lewis Lead unmasking A Flutter





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Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline:
Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
www.accessmedicine.com

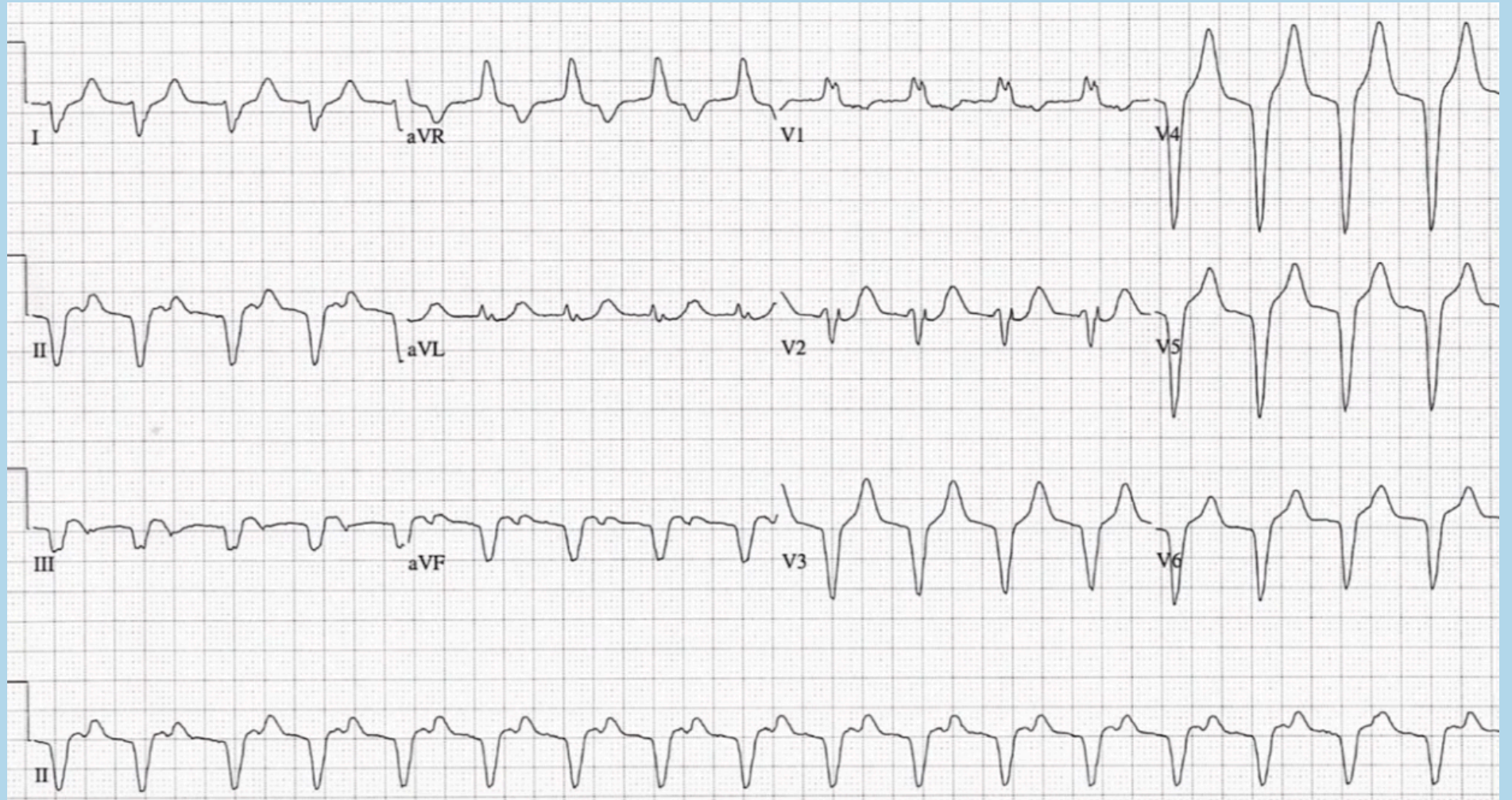
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Case 9



<https://ecgweekly.com/2020/04/amal-mattus-ecg-case-of-the-week-april-27-2020/>



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Accelerated Idioventricular Rhythm (AIVR)

- “Slow V Tach”
- Regular, wide-complex
- Rate 40-120
- Causes: reperfusion after lytics, digoxin toxicity, severe electrolyte abnormalities, post-arrest rhythms
- **Usually a reperfusion rhythm lasting a few minutes**
 - Management:

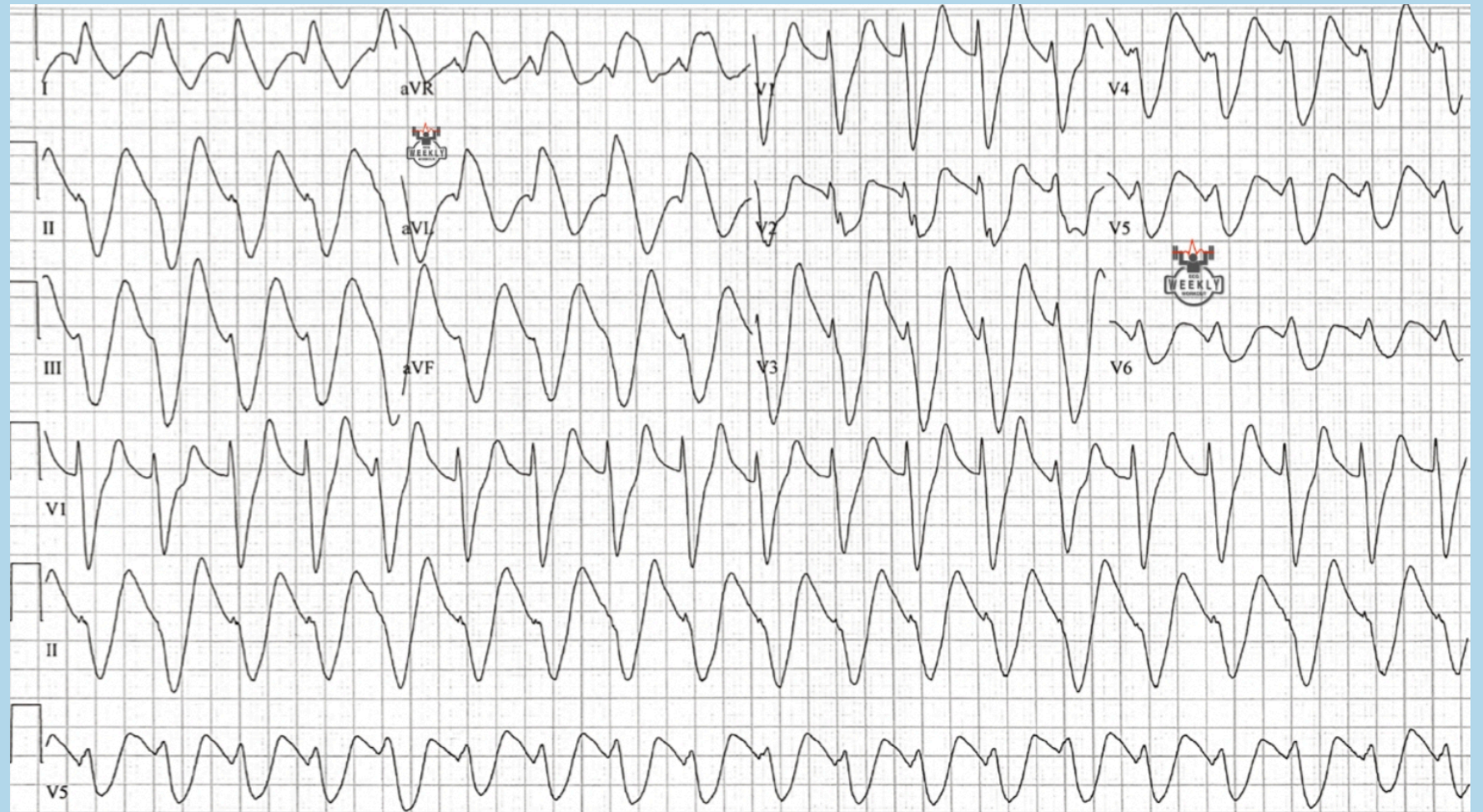




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Case 10



<https://ecgweekly.com/2020/01/amal-mattus-ecg-case-of-the-week-january-13-2020/>



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Hyperkalemia

- Regular REALLY Wide Complex Tachycardia (>200 ms)
 - Think Tox or Metabolic FIRST!
- Can have some irregularity
- Poisoned sodium channels
 - Sodium channel blockers can kill!
- Management: sodium bicarb
- Treat hyperkalemia

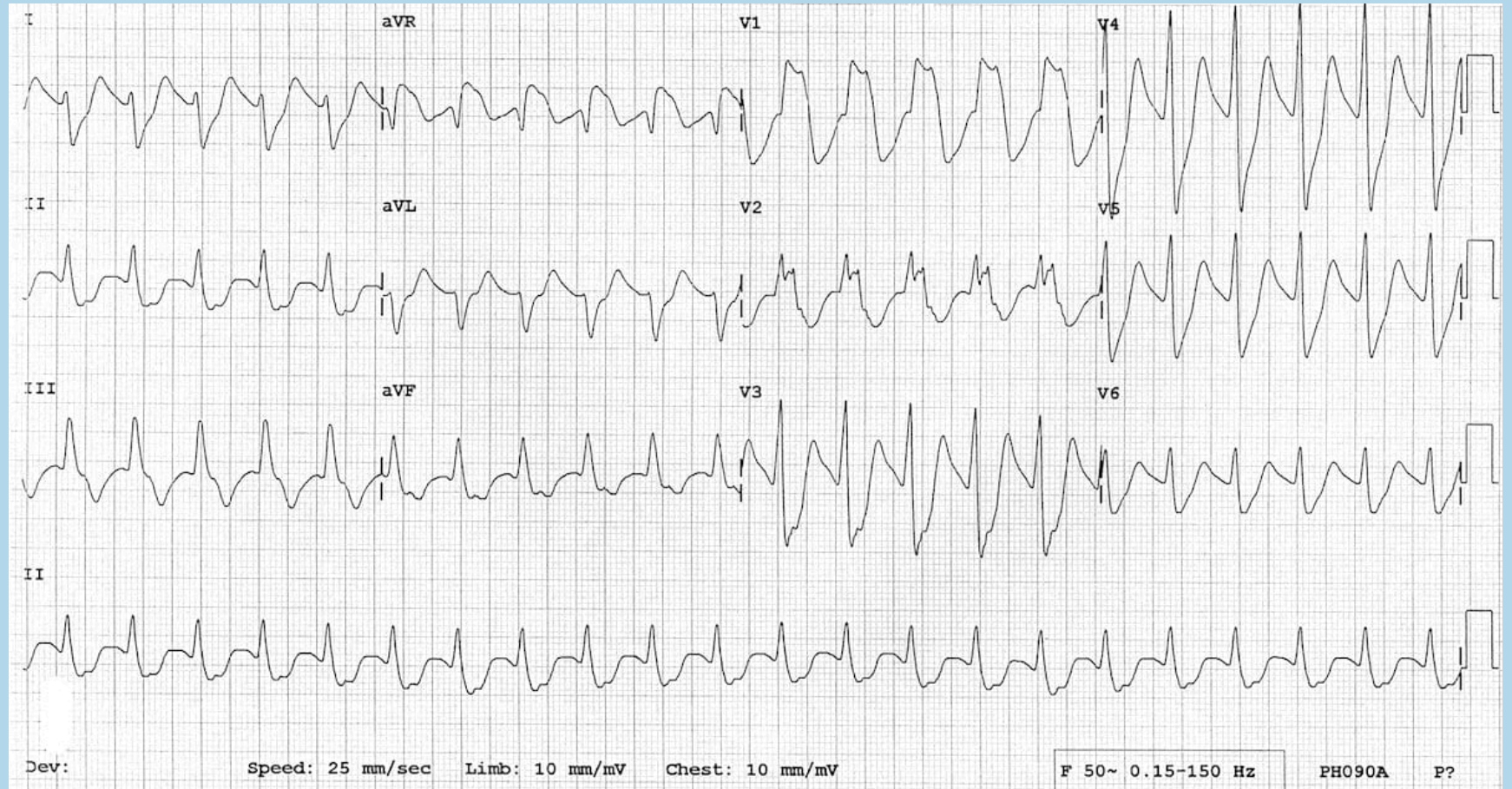




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Case 11





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Sodium Channel Blockade

- History and clinical picture is key
- EKG findings:
 - QRS complexes are broad
 - RAD
 - Positive R' in aVR
 - Deep S in lead I
 - Prolonged PR (P wave hidden in previous T)
- Management: sodium bicarb



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Case 12



https://www.utmb.edu/ped_ed/CoreV2/Cardiology/cardiologyV2/cardiologyV217.html



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Wide Complex Tachycardia

Regular rhythm

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- Monomorphic ventricular tachycardia (rate >120)
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- Sodium channel blockade
- Hyperkalemia

Irregular rhythm

- WPW with a fib (rate >300)
- Atrial fibrillation with aberrancy (rate 200s)
- Polymorphic ventricular tachycardia
- Torsades de pointes
- Ventricular fibrillation



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Torsades de Pointes

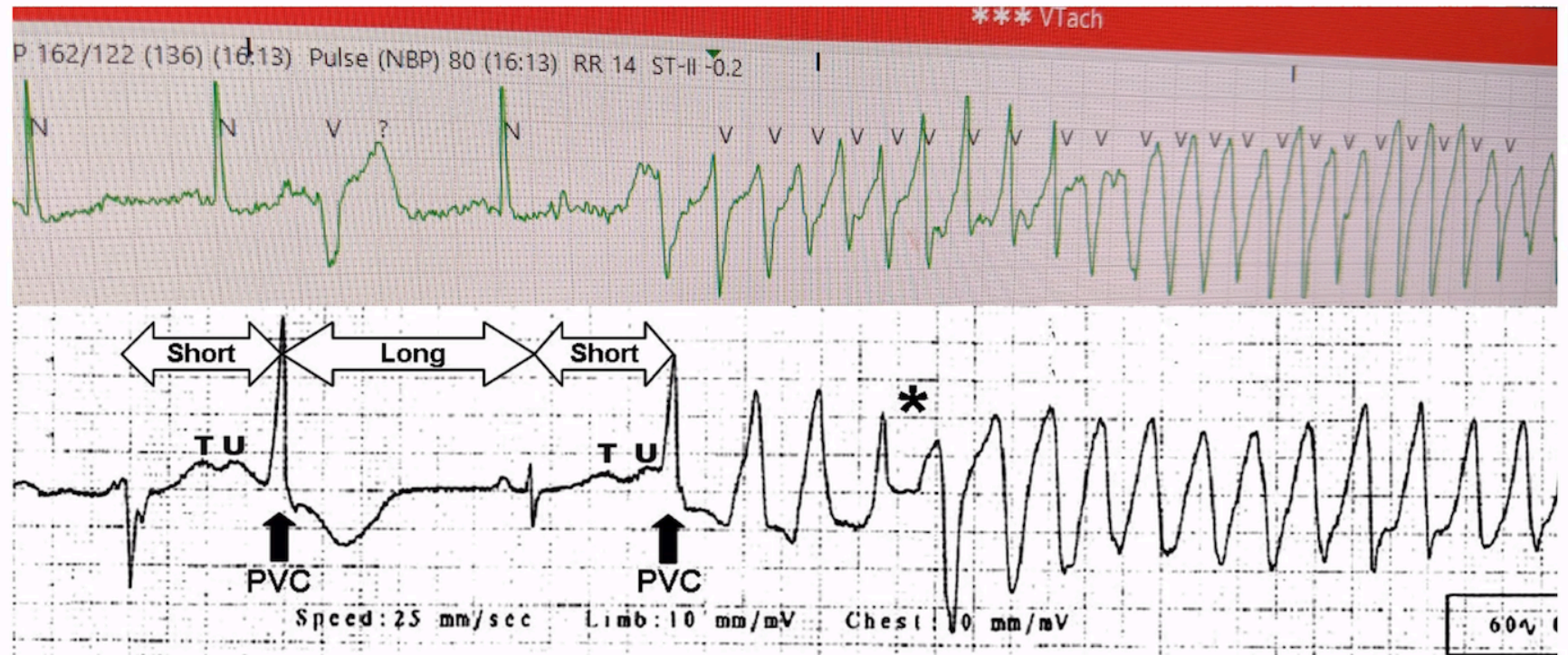
- Polymorphic V Tach **due to QT prolongation**
- Causes:
 - Congenital
 - Electrolytes – hypoMg, hypoK, hypoCa
 - Hypothermia
 - Anti-arrhythmics – Class IA and IC
 - Anti-emetics
 - Antibiotics
 - Antidepressants
 - Other - methadone
- Management:
 - Unstable: cardioversion
 - Stable: IV Mag, overdrive pacing (electrically or chemically with isoproterenol or epinephrine)
 - **CONTRAINDICATION:** procainamide and amiodarone



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45M. Syncope. No PMHx. MAP 110.



Case Credit: Dr. Max Hockstein

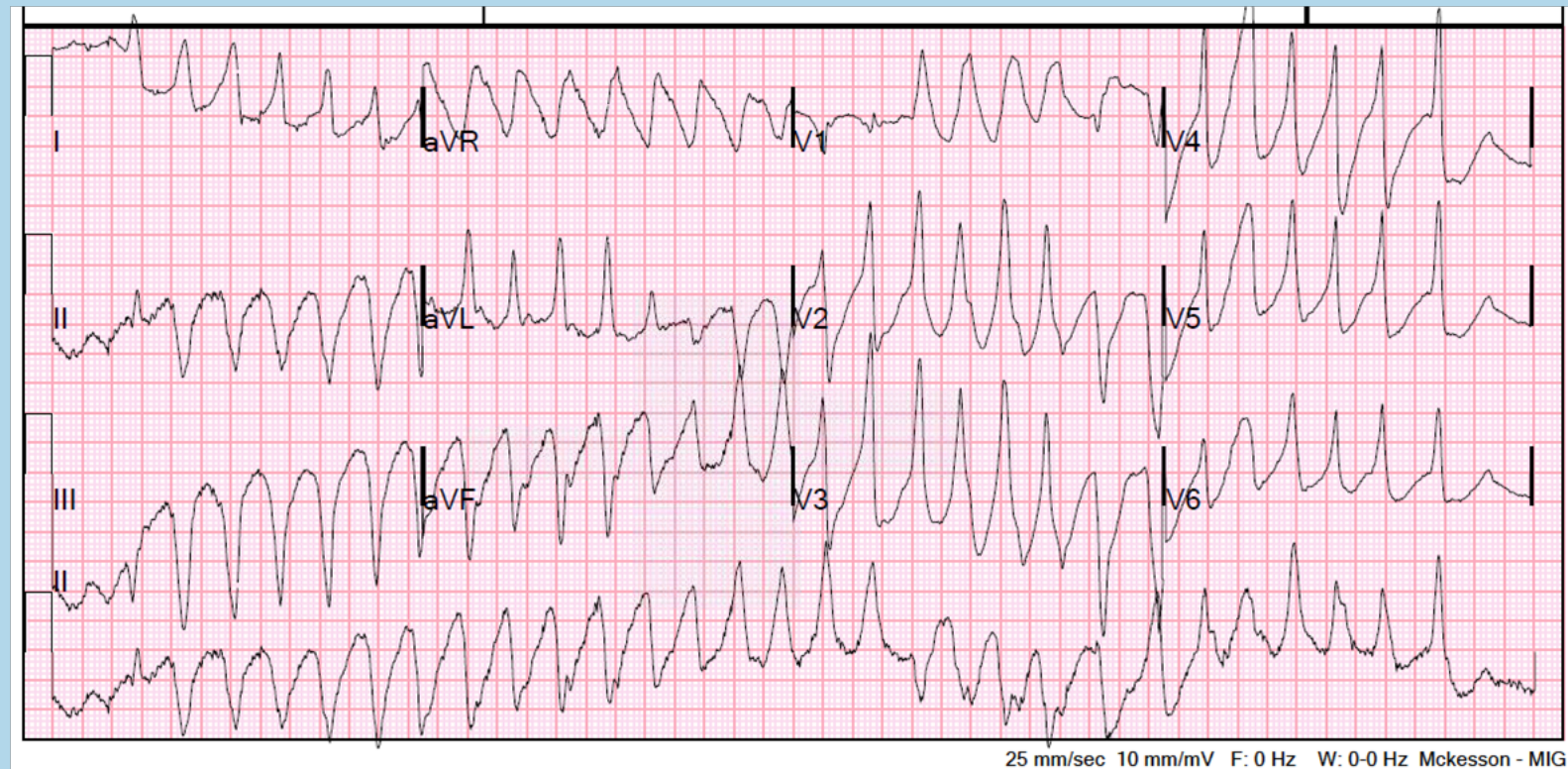


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Generic Polymorphic V Tach

- Unlike monomorphic, usually caused by ischemia
- Management: Shock, Cath Lab, amiodarone

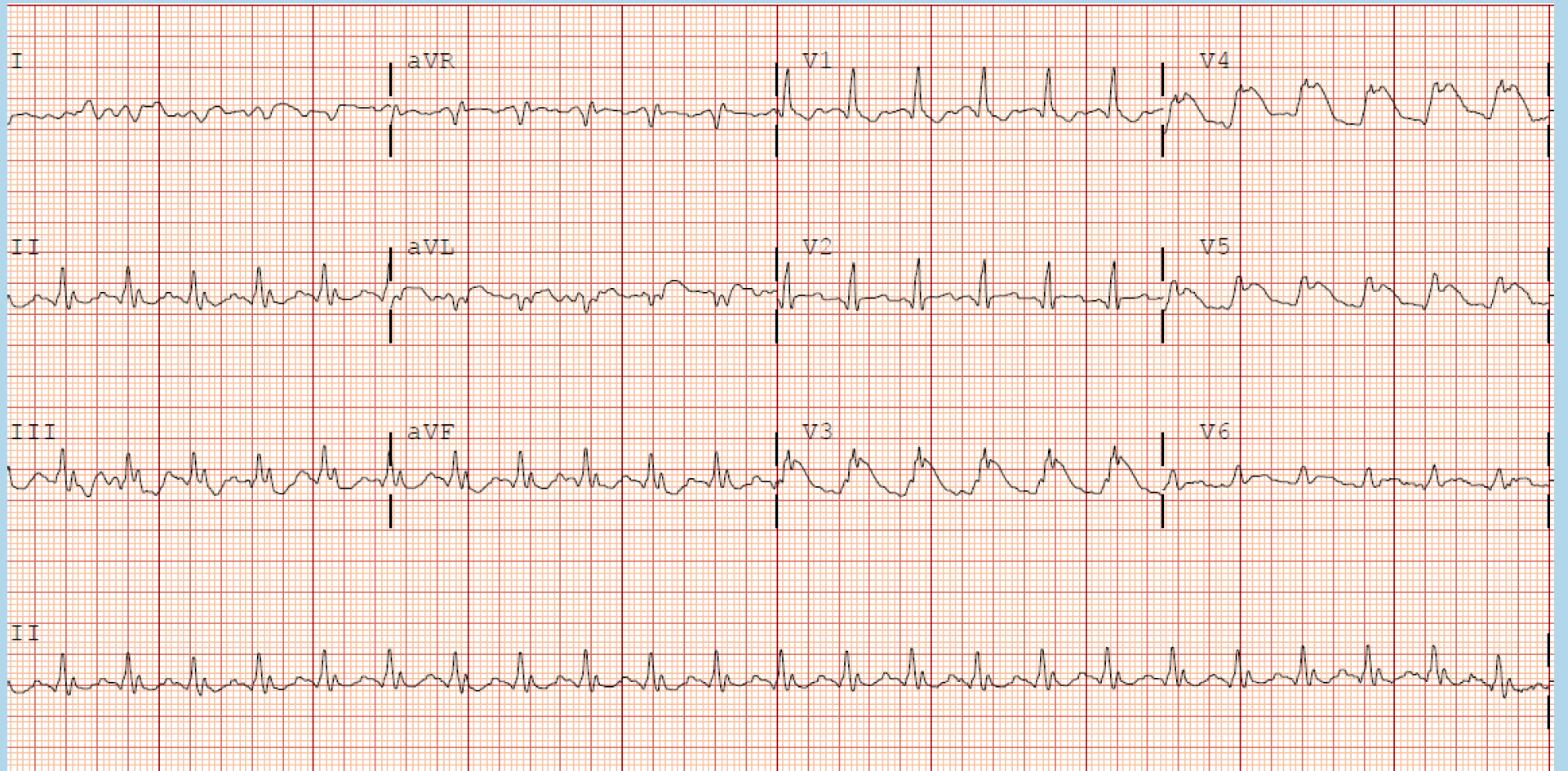




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Case 13





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Ventricular fibrillation

- Defibrillation
- Refractory
 - Dual external or Dual sequential defibrillation (DSD)
 - 12 published papers with 38 cases (2018)
 - 77% of 38 cases had VF successfully terminated
 - 54% had ROSC
 - 28% had positive neuro outcome
 - DOSE VF Trial 2020 (first RCT)
 - 152 patients
 - ROSC: 25% standard care, 39% vector change, 40% DSD
 - Esmolol
 - Consider after 3 shocks, 3 mg of epi and amiodarone
 - Increased sympathetic tone from ACLS -> lower threshold for v fib
 - Small studies, no RCT
 - US 2014 – 25 patients, 50% vs 11% survival to discharge + good neuro outcome
 - Korea 2016 – 41 patients, 56% vs 16% sustained ROSC

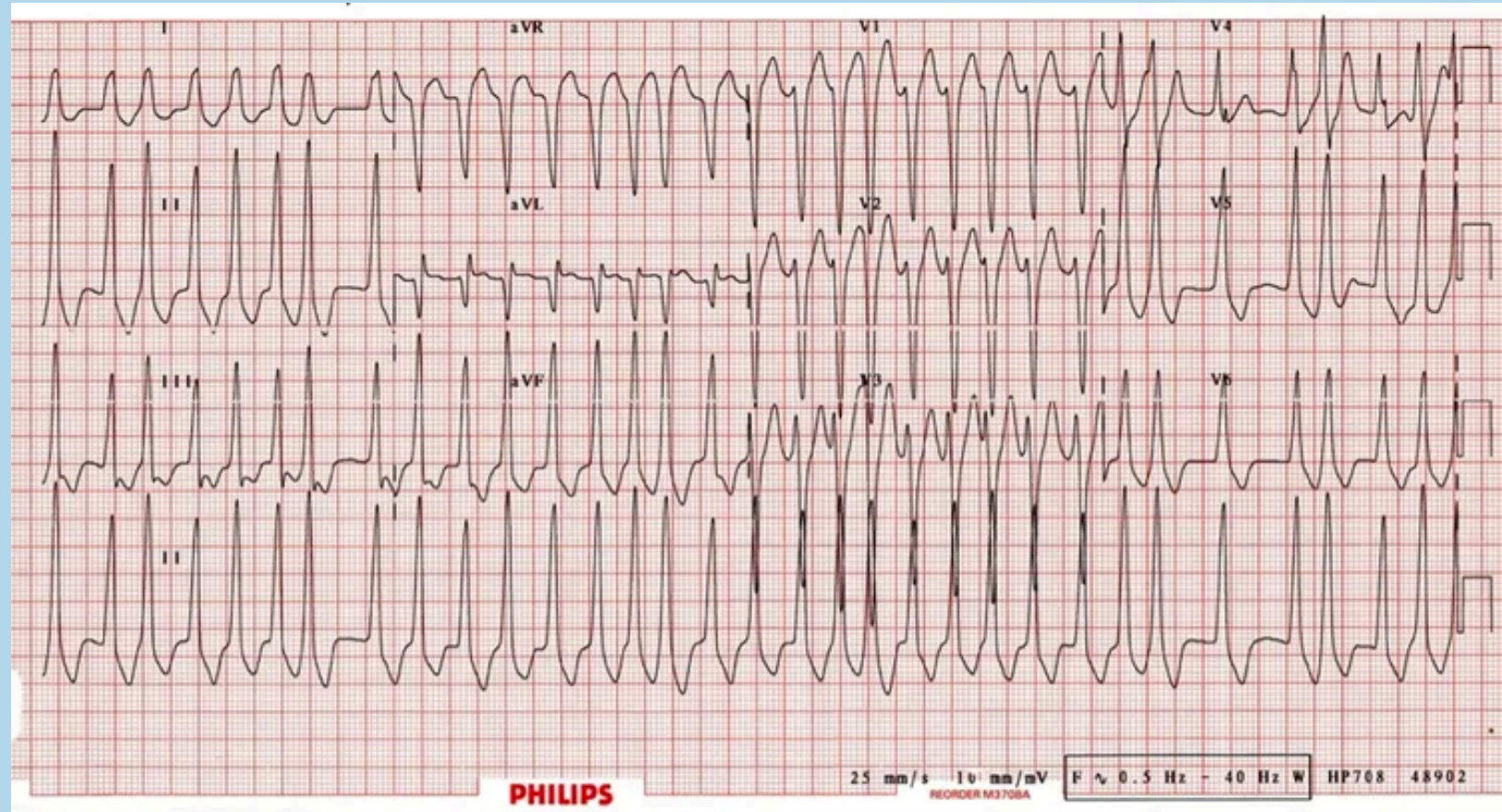


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Case 14

- A. Polymorphic V Tach
- B. A fib with RVR + LBBB
- C. A fib with AVRT (WPW)



<http://blog.clinicalmonster.com/2018/02/22/>



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Anti-arrhythmics – CLASS I

Name	MOA	Use	Caution/Toxicity
CLASS I Sodium Channel blockers	Slows/blocks conduction of depolarization		
IA Procainamide Quinidine Disopyramide	Increases AP duration	Atrial/ventricular arrhythmias; AVRT	<u>Increases QTc -> torsades</u>
IB Lidocaine Mexiletine Tocainide Phenytoin	Decreases AP duration, preferentially affects ischemic tissue	Acute ventricular arrhythmias, especially post-MI	CNS stimulation/depressions, CV depression
IC Flecainide Propafenone Encainide	Prolongs ERP in AV node	SVTs, atrial fibrillation	Pro-arrhythmic, <u>contraindicated in MI and CHF</u>



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Anti-arrhythmics – CLASS II

Name	MOA	Use	Caution/Toxicity
CLASS II			
Beta Blockers	Decrease SA and AV nodal activity	SVT, a fib, a flutter	COPD/asthma, bradycardia, AV block, unopposed alpha
Metoprolol			
Propranolol	(decreases slope of repolarization)		
Labetalol			
Esmolol			



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Anti-arrhythmics – CLASS III

Name	MOA	Use	Caution/Toxicity
CLASS III K channel blockers Amiodarone Ibutilide Dofetilide Sotalol	Increases AP duration, increases ERP		<u>Increases QTc -> torsades</u> <u>bradycardia, heart block, hypotension,</u> pulmonary fibrosis, hepatotoxicity, hypo/hyperthyroidism, corneal/skin deposits, neuro effects, constipation



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Anti-arrhythmics – CLASS IV

Name	MOA	Use	Caution/Toxicity
CLASS IV Ca channel blockers Diltiazem Verapamil	Decreases conduction velocity, increases ERP, increases PR	Prevents SVT, a fib rate control	HF, AV block, sinus node



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Anti-arrhythmics – OTHER

Name	MOA	Use	Caution/Toxicity
Adenosine	Increases K out of cells -> hyper polarizes cells -> decreases ionized Ca; short acting	SVT	Does not work for caffeine Flushing, hypotension, chest pain, doom, bronchospasm
Magnesium	Sodium channel blocker	Torsades, digoxin toxicity	



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PROCAMIO Trial

- Wide complex tachycardia – procainamide 10 mg/kg vs amiodarone 5 mg/kg
- Procainamide had lower incidence of major cardiac adverse events (9% vs. 41%)
- Procainamide had higher efficacy of tachycardia termination (67% vs. 38%)
- Indications for amiodarone first:
 - ICD patient with V Tach
 - Pulseless V Tach

Mercedes Ortiz et al "Randomized Comparison of intravenous procainamide vs. intravenous amiodarone for the acute treatment of tolerated wide QRS tachycardia: the PROCAMIO study". *European Heart Journal*. 2017. 38(17):1329-1335.



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Take home points

- Standardized approach
- Rate
- Rhythm – Regular or irregular
- Narrow or Wide Complex
- Consider your anti-arrhythmic agents
 - Shock if unstable or unsure!
 - Consider procainamide 1st line for wide complex EXCEPT
 - Sodium channel blockade
 - Hyperkalemia
 - ICD patient
 - Pulseless V tach



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Thank You

Any Questions?

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