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OBJECTIVES

1. Identify the basic electrophysiology of the four causes of wide complex tachycardia.
 2. Develop a simple framework for acute management of wide complex tachycardia.
 3. Practice three cases with ECGs to differentiate ventricular tachycardia from other cause and determine correct management.
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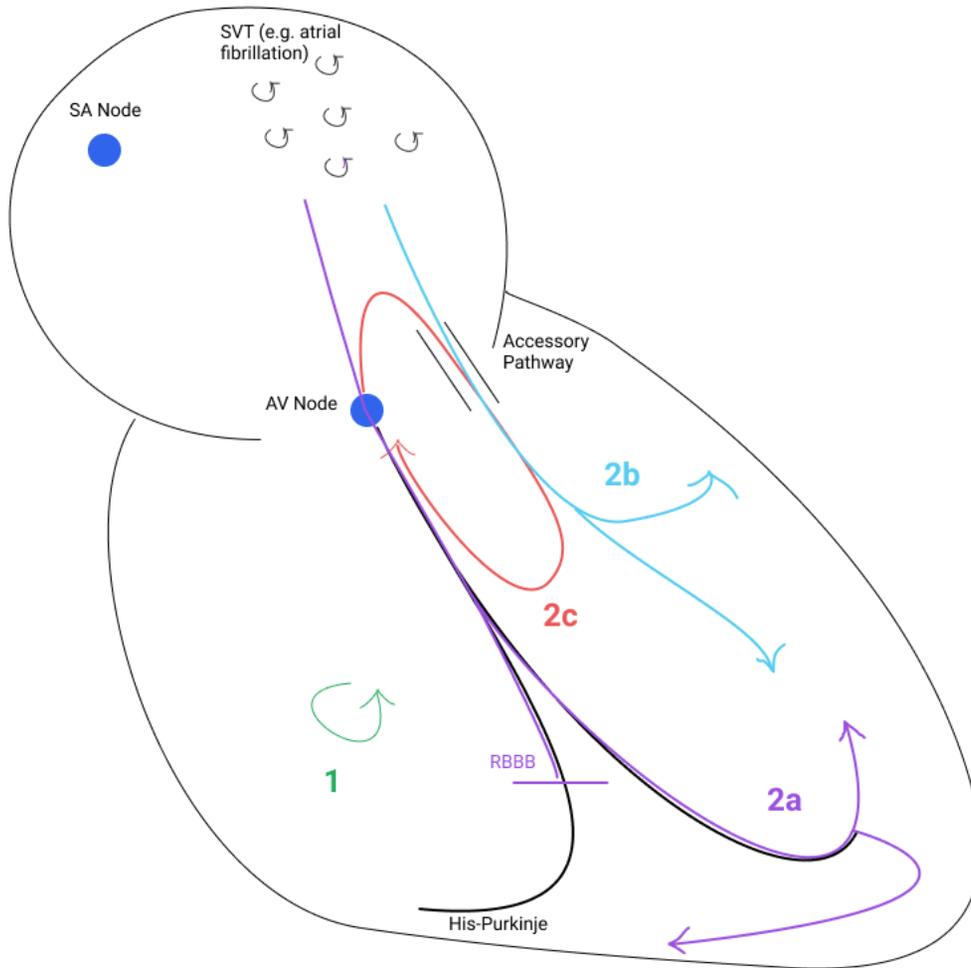
INTRODUCTION

Wide complex tachycardias are uniquely challenging due to the difficulty in interpreting the ECG and the need for immediate management. Fortunately, there are only a handful of potential rhythms and a few options for management. If you remember noting else:

1. Unstable -> SHOCK (synchronized cardioversion)
 2. Stable ->
 - a. Place defibrillation pads
 - b. If regular and monomorphic, attempt vagal maneuvers or adenosine
 - c. If it doesn't work or is irregular, consciously sedate and cardiovert.
 - d. If conscious sedation is not a safe option, try procainamide.
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Wide Complex Tachycardias

Electrophysiology



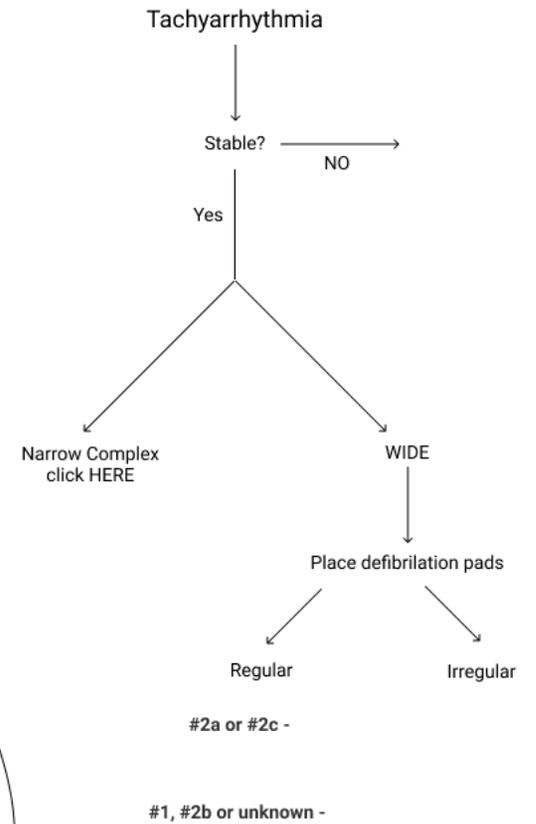
1 -

2a -

2b -

2c -

Management



Stage 1 - Electrophysiology

What are the two basic mechanisms for tachycardia?

1. Increased automaticity - every myocardial cell has a pacemaker. The pace for that given cell may increase if it is injured, hypoxic or experiences non-physiologic electrolyte shifts, and overtake the SA node.
2. Re-entry circuit - depolarization typically travels from the SA node through the AV node and down the His-Purkinje System (HPS), before starting again. If an alternative circuit manages to bypass this path (i.e. atrial flutter), it will travel at a much faster rate and overtake the SA node.

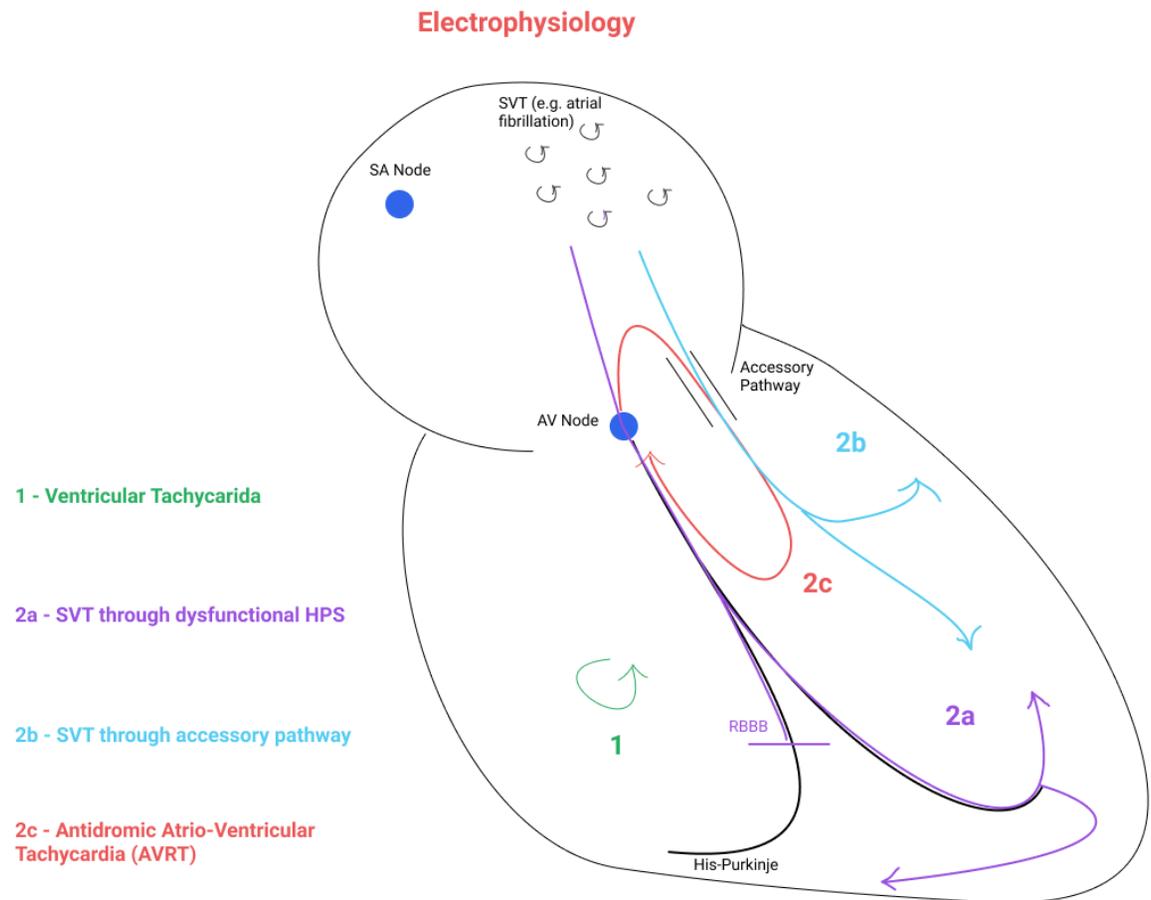
What makes a QRS wide (>120ms or 3 small boxes)?

Ventricular depolarization that is not conducted through the standard path from AV node through a healthy His-Purkinje System (HPS).

Here are the two general types (and sub-types) of wide complex tachycardias:

1. Ventricular Tachycardia – Re-entry circuit or increased automaticity originating in the ventricles outside the HPS.
2. Supraventricular Tachycardia (SVT)* with aberrancy:
 - a. SVT (e.g. sinus tachycardia, atrial tachycardia, atrial fibrillation, atrial flutter, etc.) conducted through a dysfunctional or damaged His-Purkinje System (e.g. right bundle branch block - RBBB)
 - b. SVT (e.g. atrial fibrillation) antegrade through an accessory pathway
 - c. Antidromic atrioventricular re-entry tachycardia (AVRT), e.g. Wolff-Parkinson-White

*For the purposes of this talk the term SVT include **ALL** tachycardias originating at or above the AV node, rather than just AVRT and AVnRT which are also classified as Paroxysmal SVT.



Stage 2 - Management

It is often challenging to diagnose a wide complex arrhythmia without **prior ECGs** for comparison or without first **correcting the acute arrhythmia**. While it is important to determine the correct underlying rhythm, most cases of WCT present in acute and critical situations that require immediate action. Fortunately, determining the specific etiology is most meaningful for long-term management, and the immediate treatment options are relatively limited, following a simple decision diagram:

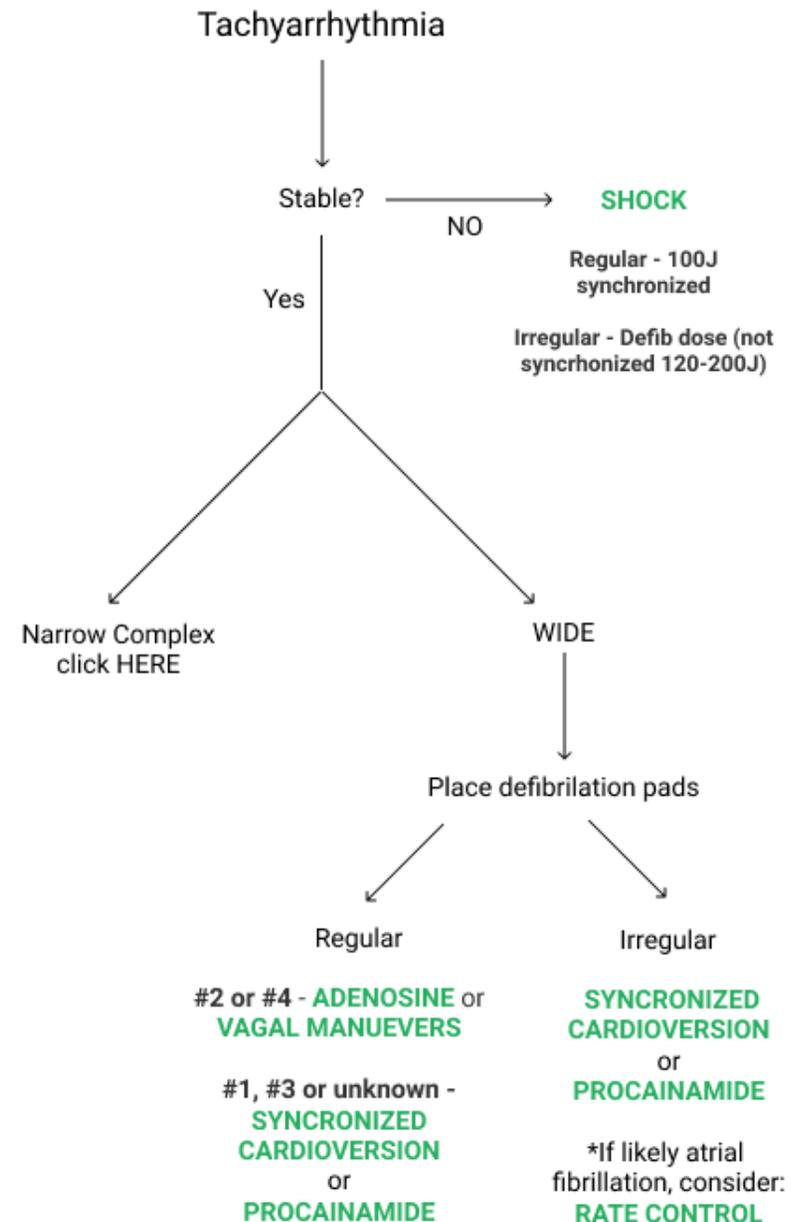
1. Once a WCT is identified -> **place defibrillation pads**
2. If **unstable** -> **SHOCK** (synchronized, if possible)

If the patient is **stable**, you want to determine if the rhythm is VT or something else. VT will require cardioversion or if conscious sedation is not a safe option, an anti-arrhythmic (procainamide or amiodarone). All other causes will be managed with AV nodal blockade or management of the underlying cause for the arrhythmia.

3. **Rhythm is regular and monomorphic** -> If it is not clearly VT, attempt brief AV nodal blockade with vagal maneuvers or adenosine.
 - AV nodal blockade will slow the rate of SVTs traveling through dysfunctional HPS^{2a} and likely reveal the underlying rhythm.
 - If it is AVnRT with a BBB^{2a} or antidromic AVRT^{2c}, AV nodal blockade will interrupt the re-entry circuit and resolve the arrhythmia.
 - If there is no change in the rate and rhythm, it is independent of the AV node and likely ventricular tachycardia -> cardioversion or procainamide*
4. **Rhythm is irregular or polymorphic** – an irregularly irregular rhythm is likely atrial fibrillation with aberrancy (BBB or through an accessory pathway) or polymorphic VT. If you have access to a prior ECG that confirms the same aberrant morphology at baseline then you could manage as atrial fibrillation with BBB. Otherwise, manage with cardioversion or procainamide.

*Procainamide and amiodarone are both reasonable anti-arrhythmic options for WCTs. However, procainamide has fewer complications with similar rates of successful cardioversion

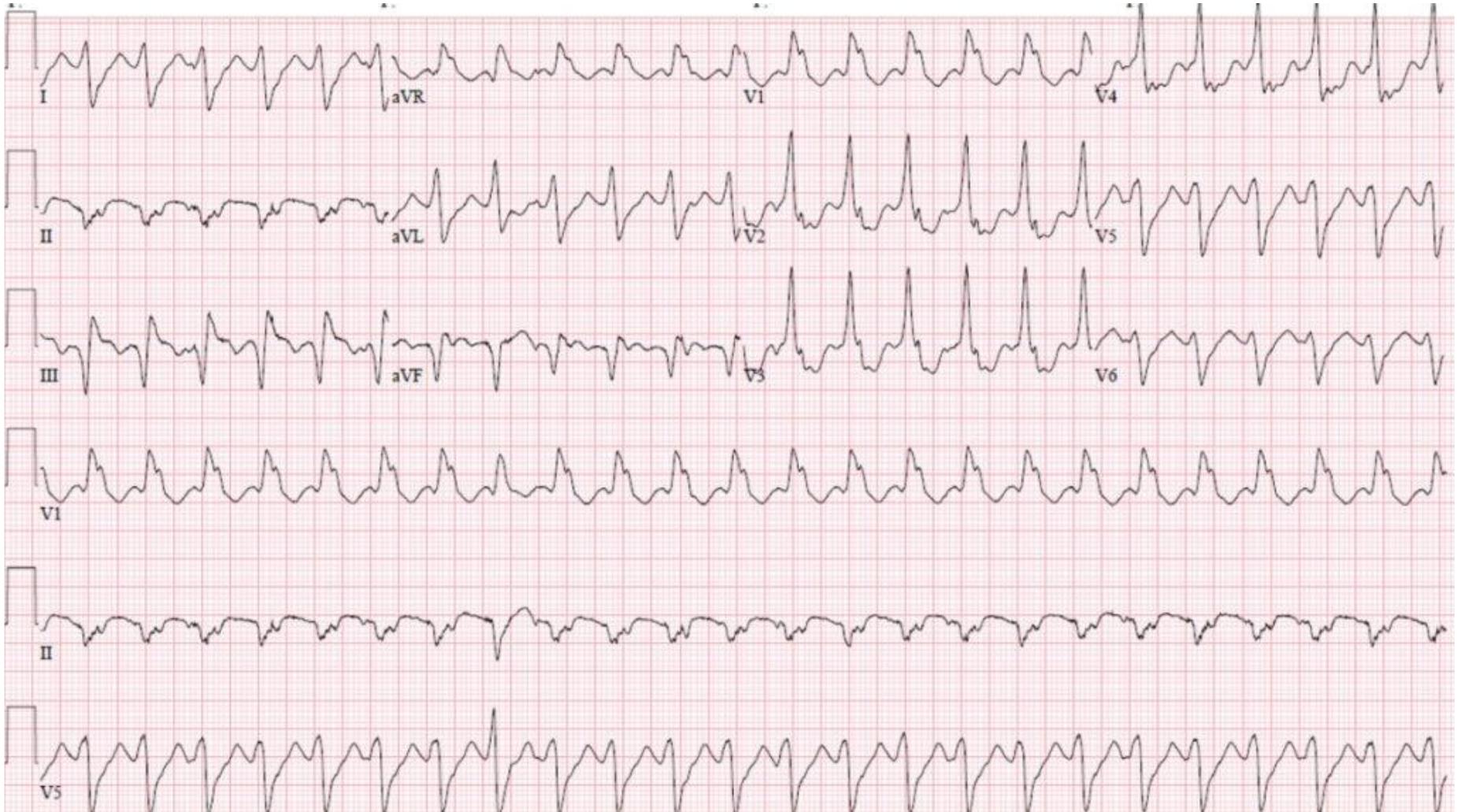
Management



PRACTICE CASES

Case 1

A 65-year-old man with a recent MI presents to the ED complaining of palpitations. He is cognitively intact without lightheadedness and has palpable pulses.



Courtesy of Andrew Prouse, MD

What is the rhythm? **Monomorphic VT** (review the attached diagram for identifying VT)

How do you want to manage it? **Cardioversion**

1. Cardioversion with conscious sedation. If sedation is not a safe option, then procainamide.
2. Once converted, prevent recurrence with lidocaine or amiodarone
3. Correct electrolyte abnormalities
4. Manage ongoing ischemia

Brief Guide to Identifying Ventricular Tachycardia

Clinical Context:

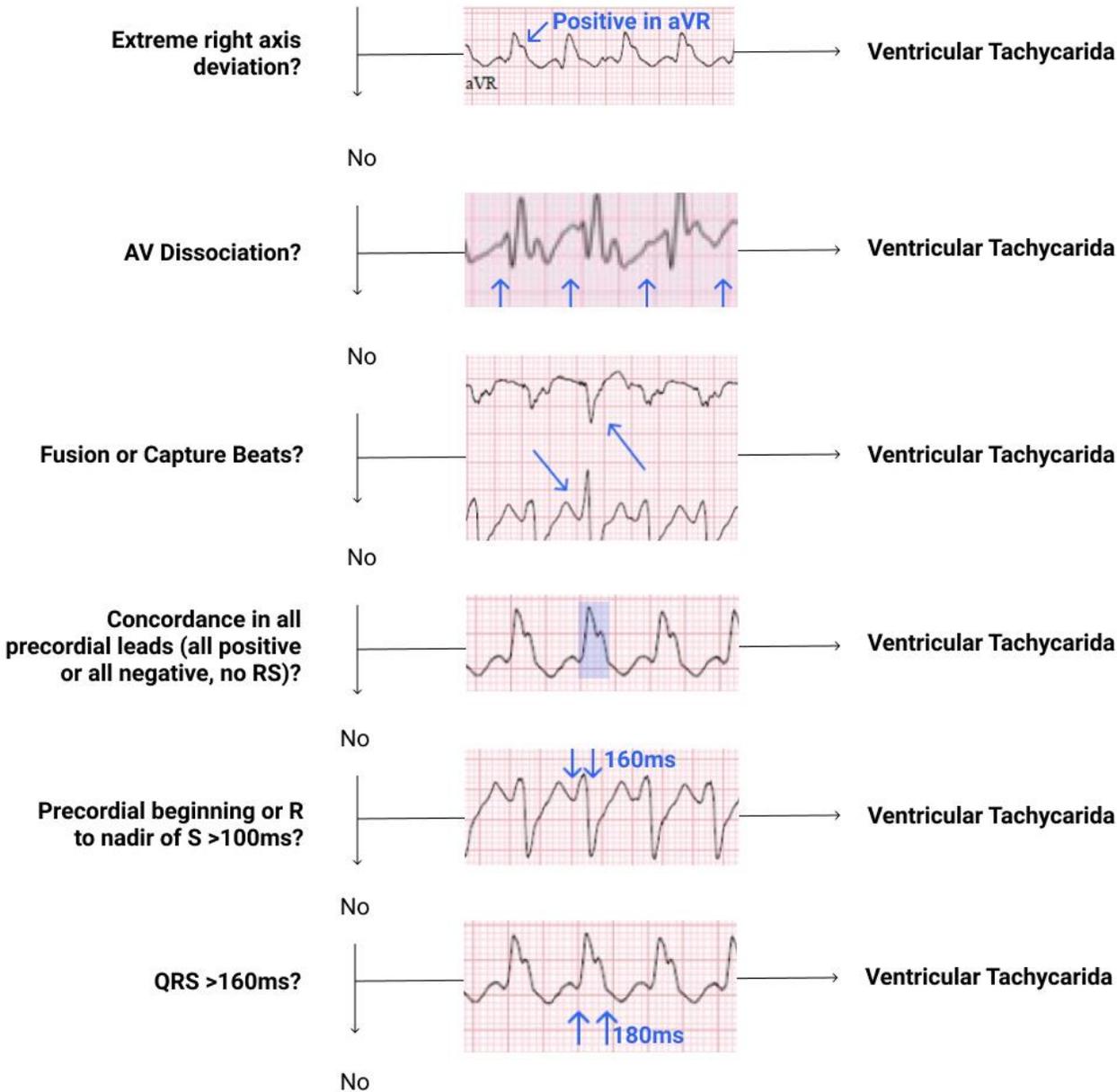
VT is more common than SVT with aberrancy, especially in older patients and those with ischemic heart disease or cardiomyopathies

*This is not an exhaustive review of VT diagnosis and management. Here are two resources that offer a more thorough guide:

[Life in The Fast Lane - VT vs. SVT with aberrancy](#)

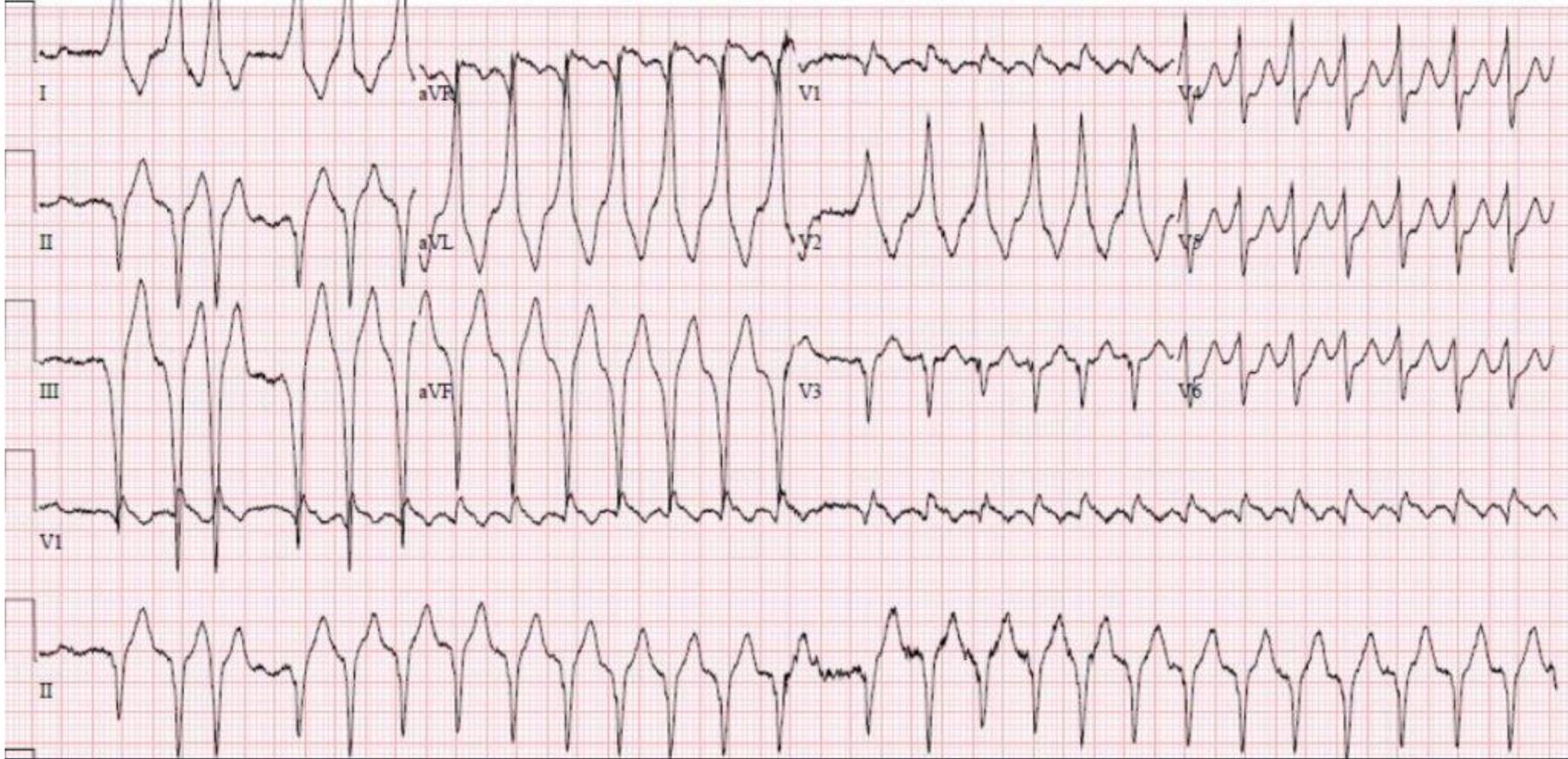
[Dr. Smith's ECG Blog - VT case series](#)

Is this VT?



Case 2

26-year-old male with chest pain and SOB and no history of structural heart disease. Hemodynamically stable.



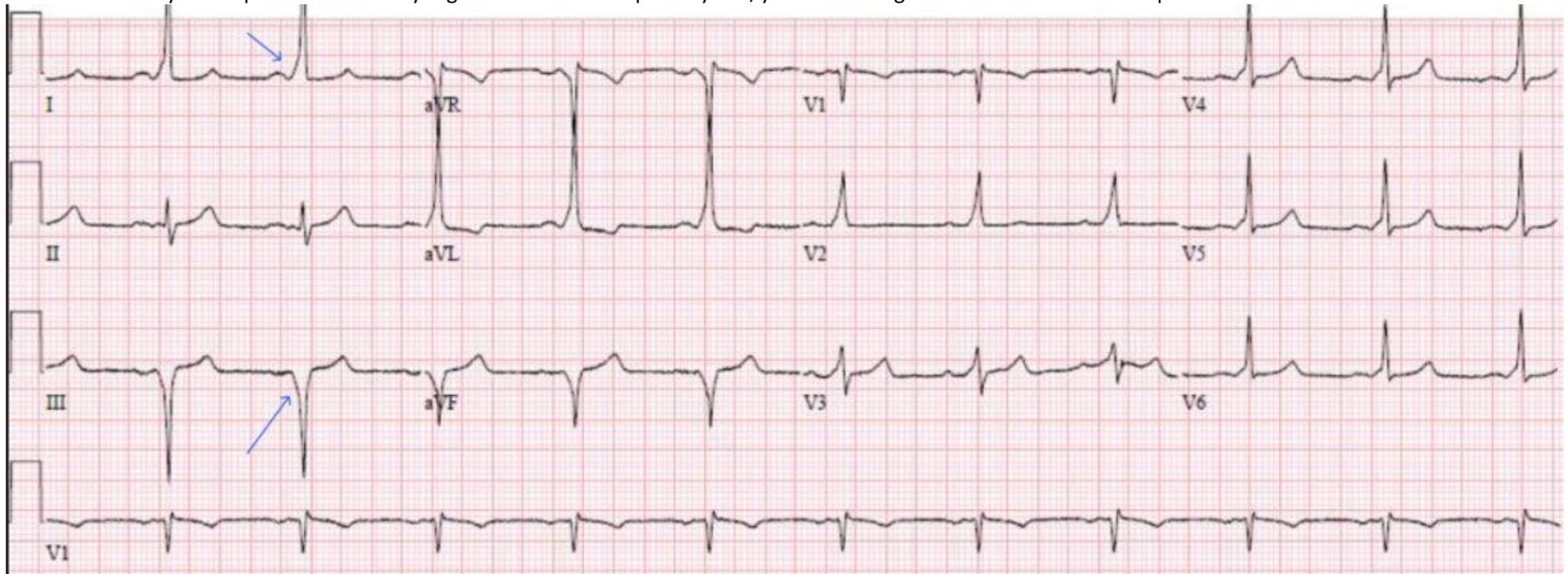
Adapted with permission from Dr. Smith's ECG blog, <http://hqmeded-ecg.blogspot.com/>

What is the rhythm? **Initial rhythm: Antidromic AVRT (Wolff-Parkinson-White)**

The post-conversion ECG reveals 1. Prominent delta waves, 2. short PR and 3. prolonged QRS.

How do you want to manage it? AV nodal blockade with vagal maneuvers or adenosine (rapid IV push of 6mg IV x1, if ineffective give 12mg IVx1)

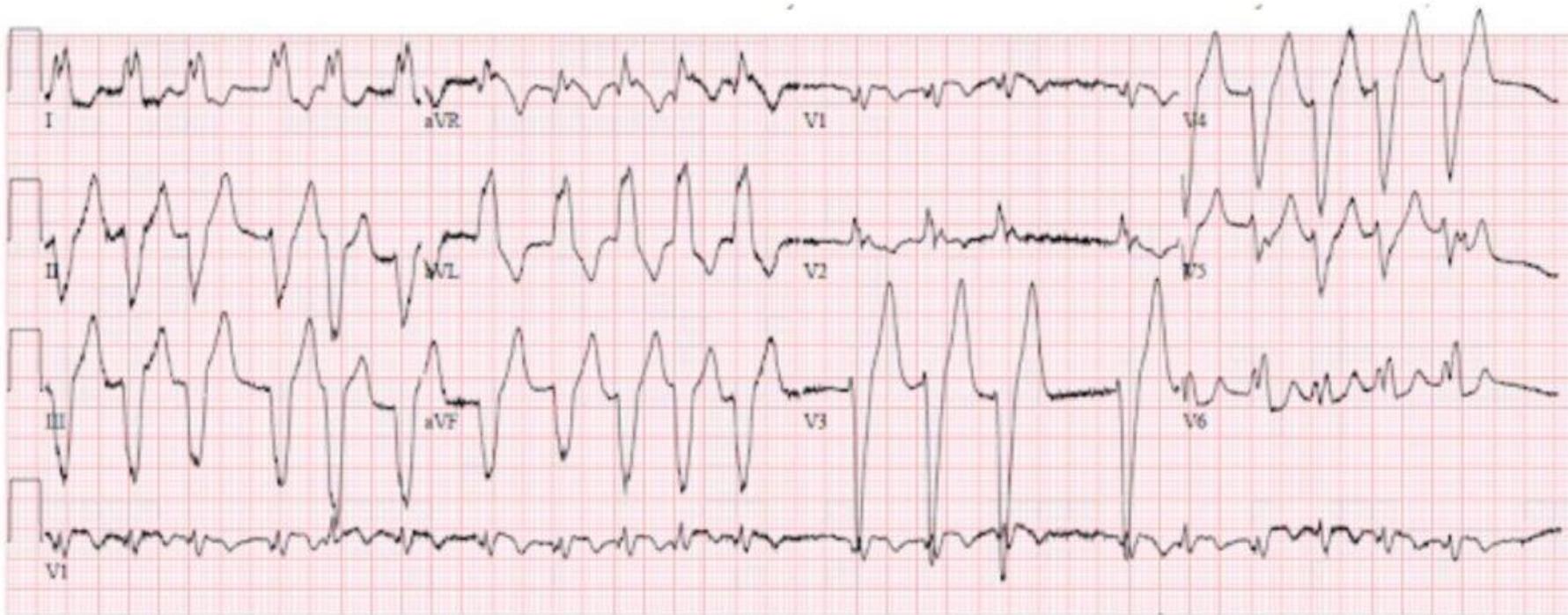
Given the stability of the patient and mostly regular and monomorphic rhythm, you decide to give **adenosine**. Here is the post-conversion ECG.



Adapted with permission of Dr. Smith's ECG blog, <http://hqmeded-ecg.blogspot.com>

Case 3

A 60-year-old woman presents to the ED with fever, cough, and shortness of breath found to have the following ECG. She is tachycardic but has a normal blood pressure and cognitively intact. There is no prior ECG available for comparison.



Adapted with permission from Dr. Smith's ECG Blog (<http://hqmeded-ecg.blogspot.com>)

What is the rhythm? This is highly suspicious for **atrial fibrillation with a LBBB** based on the irregularly irregular rhythm, clinical context and lack of features suggestive of VT. Ventricular tachycardia is only irregular if it is polymorphic, which this does not appear to be.

How do you want to manage it? The arrhythmia and/or rapid ventricular response (RVR) is likely in response to her acute illness, sepsis. If the heart rate does not improve after resuscitation, and the patient is otherwise stable, then consider rate control with AV nodal blockade. Alternatively, if the patient becomes increasingly unstable despite appropriate management of her acute illness, provide conscious sedation and synchronized cardioversion vs. pharmacologic cardioversion with amiodarone.

TAKE HOME POINTS

1. Place defibrillation pads once you identify a wide complex tachycardia.
 2. Adenosine will reveal the underlying rhythm in SVT with aberrancy, resolve AVRT or AVnRT and not impact VT.
 3. Extreme right axis deviation (positive in aVR) is a quick and reliable way to diagnosis VT.
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