EM CASES SUMMARY

Episode 112 Tachydysrhythmias

With Amal Mattu & Paul Dorion
Prepared by Shaun Mehta, July 2018

General Approach to Tachydysrhythmias

Asking these three questions will help classify any tachydysrhythmia in most cases.

1. Regular or irregular?
2. Narrow or wide QRS?
3. Are there P waves? P-QRS relationship? How many P waves for each QRS?

	REGULAR	IRREGULAR
NARROW	ST vs SVT (AVNRT, OAVRT, Aflutter 2:1)	Afib vs Aflutter + variable block
WIDE	VT >>SVT+aberrancy HyperK, Na-blocker	Afib+WPW or BBB vs PMVT

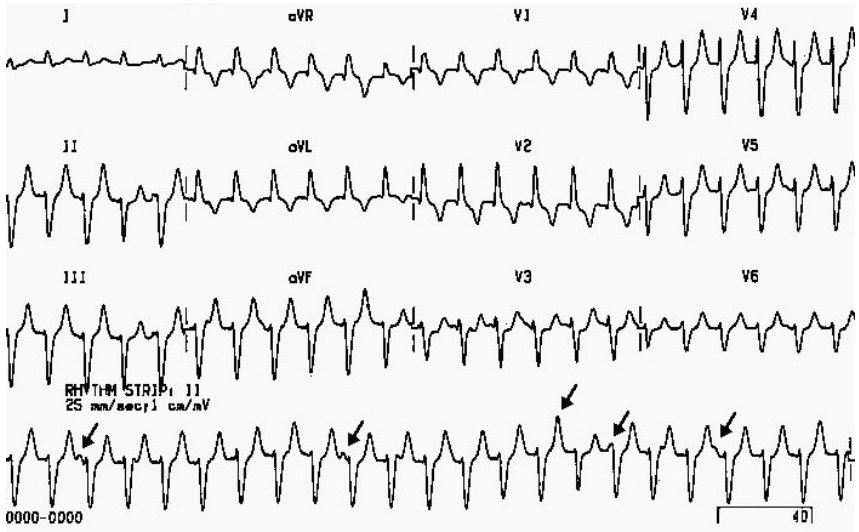
Wide & regular tachydysrhythmias

Ventricular Tachycardia (VT) vs SVT with aberrancy: Assume VT

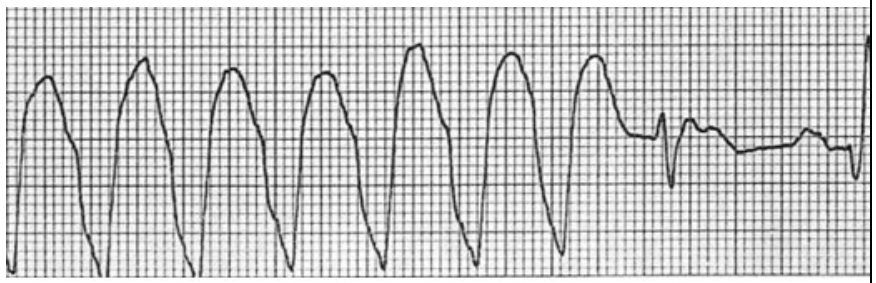
Wide & regular = ventricular tachycardia until proven otherwise. Clinical stability does not differentiate between VT and SVT with aberrancy. Despite multiple ECG algorithms and rules to distinguish VT from SVT with aberrancy (Brugada, Wellens, Vereckei, R wave peak time) none are better than 90% specific to identify SVT with aberrancy. No feature or combination of ECG features is 100% specific for SVT with aberrancy. Hence, using an algorithm/rule, there is a 10% chance that you will label VT as SVT with aberrancy erroneously and if you treat the patient with AV nodal blockers, cardiovascular collapse may result.

There are several factors that make VT very likely:

1. Prior MI, heart failure, recent angina and advanced age.
2. AV dissociation (P and QRS complexes at different rates) and fusion complexes (sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology) on ECG (see images below)
3. Pave Criteria – R-wave peak time >50ms in Lead II
4. Presence of 1st degree heart block on previous ECG



Arrows show AV dissociation (from Life in the Fast Lane blog)


The first narrow complex is a fusion complex

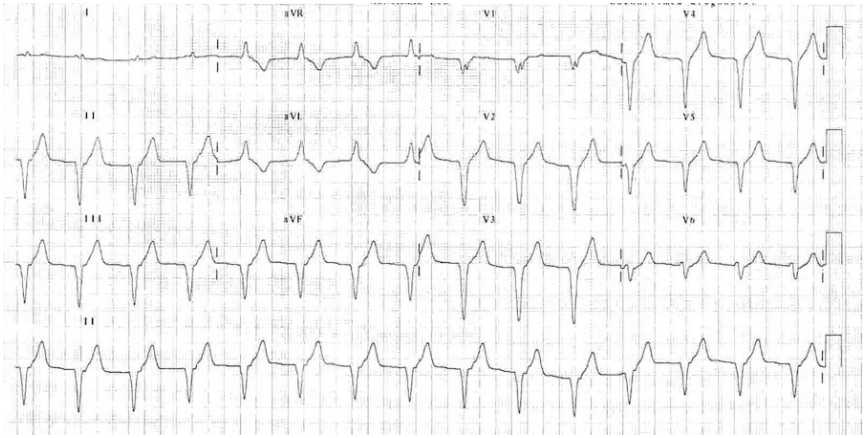
Remember that although advanced age makes a wide complex tachycardia VT much more likely than SVT with aberrancy, up to 50% of patients under 40 years of age who present with a wide complex regular tachycardia with have VT. In addition, response to adenosine does not rule out VT.
As per ACLS guidelines, If any tachydysrhythmia presents as unstable, the treatment of choice is synchronized electrical cardioversion.

For many stable patients, electrical cardioversion may be the preferred treatment of choice for VT. For example, electrical cardioversion should be considered in all patients with known heart disease and VT regardless of clinical stability, as the risks of antidysrhythmic medication are probably higher than those of electrical cardioversion in this patient population. Many patients with known heart disease and VT may not be considered "unstable" according to ACLS guidelines (those with hypotension, decreased LOA, acute heart failure or ischemic chest pain), but nonetheless may have poor cardiac output and be unable to tolerate antidysrhthmic medications. In patients with known LV dysfunction and VT, even with normal BP, consider incipient shock and immediate cardioversion. Remember that cardiac output can be dangerously low while the patient maintains a "normal" BP. Blood pressure ≠ cardiac output.

Avoid the "verapamil death test"! Do not give a calcium channel blockers to a patient with a wide complex tachycardia.

For wide and irregular tachycardia consider other diagnoses (especially when standard treatments are not effective at restoring normal sinus rhythm) such as:

- Hyperkalemia (HR usually < 120 bpm)
- Sodium channel blocker toxicity (often very wide QRS > 200 ms)
- Accelerated Idioventricular Rhythm (AIVR). This is a reperfusion rhythm often seen post-lytics for STEMI; Think of "slow VT". The treatment is observation, not medication.



Accelrated Idioventricular Rhythm (AIVR) From Life in the Fast Lane blog

Pitfall: *Mistaking AIVR (post-lytics for STEMI) for VT and treating with lidocaine may cause cardiovascular collapse.*

VT is not a single entity

There are 4 types of VT that EM providers need to be aware of:

1. Scar mediated monomorphic VT – the classic VT we see in older patients with a cardiac history

->Rx: procainamide, as per the PROCAMIO study.

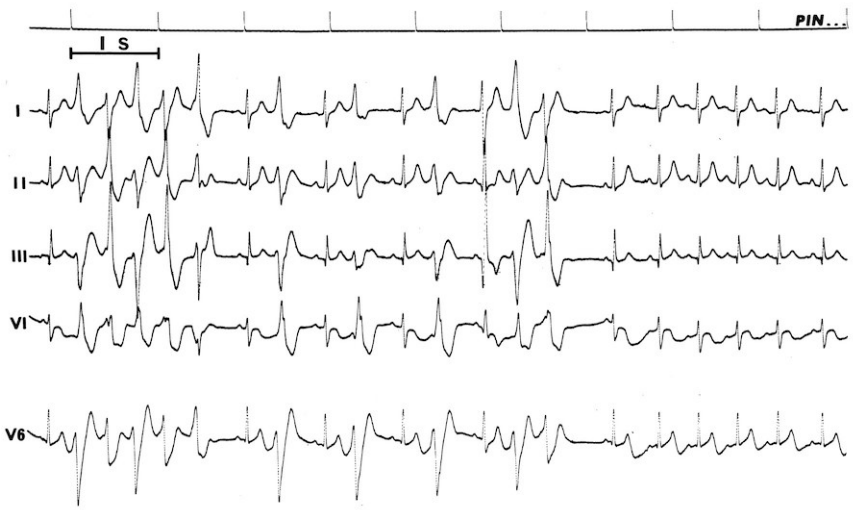
2. Polymorphic VT – usually related to a cardiac ischemic event
->Rx: amiodarone

3. Exercise induced non-sustained monomorphic VT – in young patients (e.g. 20's)

->Rx: no ED treatment required; outpatient beta blockers

4. Catecholaminergic Polymorphic VT (CPVT): Heritable VT in young patients (teens/20's) presenting as polymorphic or bidirectional with a LBBB pattern and inferior axis.

->Rx: IV beta blocker, AVOID amio and procainamide



CPVT with bidirectional VT

Management of Stable Ventricular Tachycardia

The 2016 PROCAMIO RCT trial compared IV procainamide and amiodarone for the treatment of acute but stable sustained monomorphic VT. Procainamide was associated with less major cardiac adverse events and a higher proportion of tachycardia termination within 40 minutes. Procainamide is currently considered to be the first line medication for sustained monomorphic VT in stable patients.

Indications for amiodarone in VT

While procainamide is currently considered to be the first line medication for stable sustained VT, there remain three important indications for amiodarone in the setting of VT:

1. Polymorphic VT related to cardiac ischemia
2. ICD patient with VT above detect rate (usually >175 bpm)
3. VT in the cardiac arrest patient

VT in the ICD patient

VT below detect (usually <175 bpm)

VT is too slow for ICD to recognize. Treat as you would any VT.

VT above detect (usually >175 bpm)

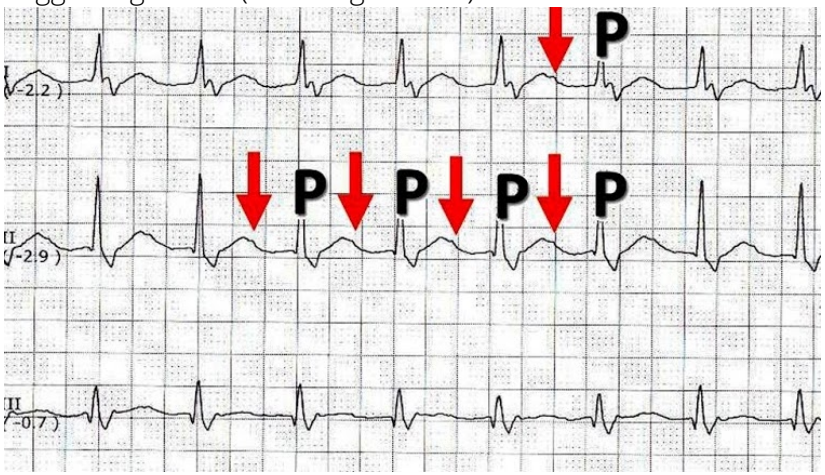
Recurrent episodes of VT. Treatment involves prevention, which is usually a combination of IV amiodarone, beta blockade and sedation. Consider causes such as ICD malfunction, electrolyte imbalance and severe CHF.

Magnet? If an ICD patient is not in VT but their ICD is delivering shocks, place a magnet on the ICD to put it into VVI mode (pacing preserved, shocking disabled).

Narrow Complex Tachydysrhythmias

How to distinguish Atrial Flutter from SVT

1. **Bix rule**: a P wave seen halfway between two QRS complexes implies there is likely another P buried in the QRS, suggesting flutter (see image below)



2. Examine all 12 leads: look for signs like a sawtooth pattern or 2:1 conduction to suggest flutter.

3. Continuous atrial activity: in flutter, there is usually no isoelectric baseline compared to SVT.

4. SVT is regular, like clockwork.

How to distinguish Sinus Tachycardia from SVT

1. SVT is regular while sinus tachycardia shows some variability with respiration
2. Sinus tachycardia maximum = (220 bpm – age)

SVT Treatment

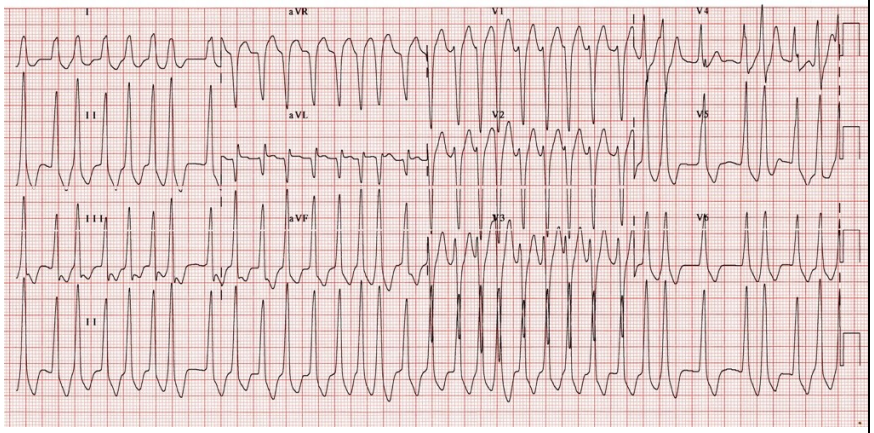
Vagal maneuvers

The REVERT trial that compared the effectiveness of a modified vs. "standard" Valsalva to convert SVT to sinus rhythm showed a NNT = 3, however real world experience does not seem as promising. This difference may be due to the study control group being seated in the "standard" Valsalva group as apposed to supine.

What is the preferred medication for conversion of SVT to sinus rhythm, Adenosine or Calcium Channel Blockers (CCBs)? Diltiazem is at least as effective as adenosine for conversion of SVT and has the advantage of lasting longer and not inducing an uncomfortable experience (often described as a feeling of near death) for the patient as observed with adenosine.

Atrial fibrillation with Wolff Parkinson White (WPW)

- Irregularly irregular tachycardia
- Changing QRS morphologies (as opposed to AF with a bundle branch block, which will be monomorphic)
- Rate 250-300 bpm



WPW with atrial fibrillation – Avoid all AV nodal blockers including amiodarone! From Life in the Fast Lane blog

Avoid all AV nodal blockers including amiodarone. Blocking the AV node may precipitate a fatal ventricular tachydysrhythmia as conduction will preferentially travel through the accessory pathway. Treat with electrical cardioversion or procainamide.

Clinical features of Atrial fibrillation vs. Atrial Flutter

Atrial Flutter is easier to electrically cardiovert, but more difficult to chemically cardiovert or rate control compared to Atrial Fibrillation.

	Atrial fibrillation	*Atrial flutter*
Electrical cardioversion	Sometimes resistant	Almost always effective
Chemical cardioversion	Almost always effective	Sometimes resistant
Rate control	Almost always effective	Sometimes resistant
Ablation	Sometimes resistant	Almost always effective

Refer patients with new atrial flutter to an electrophyiologist as it is almost always responsive to ablation while difficult to chemically cardiovert or rate control.

Disposition & Patient Education for Atrial Fibrillation

A recent study by Stiell et al. in the Annals of Emergency Medicine looked at 30-day outcomes for patients presenting to the ED with AF or flutter.

- Oral anticoagulants were under-prescribed by ED physicians – approximately 50% didn't receive them
- 10% had "adverse outcomes" which included recurrent presentations, hospitalization and 1 stroke but no deaths

ED patients are often told on discharge from the ED to return if they have recurrent symptoms of AF resulting in a high bounce back rate. Many of these patients are young and otherwise healthy. In these patients, isolated AF is almost always a benign entity. Educate otherwise healthy patients with paroxysmal AF that their disease is not life threatening and that it usually resolves on it's own or with outpatient treatment. Teaching patients to fear AF results in needless visits to the ED and increased patient anxiety.

References

1. Jastrzebski M, Kukla P, Czarnecka D, Kawecka-jaszcz K. Comparison of five electrocardiographic methods for differentiation of wide QRS-complex tachycardias. Europace. 2012;14(8):1165-71.
2. Szelényi Z, Duray G, Katona G, et al. Comparison of the "real-life" diagnostic value of two recently published electrocardiogram methods for the differential diagnosis of wide QRS complex tachycardias. Acad Emerg Med. 2013;20(11):1121-30.
3. Lim SH, Anantharaman V, Teo WS, Chan YH. Slow infusion of calcium channel blockers compared with intravenous adenosine in the emergency treatment of supraventricular tachycardia. Resuscitation. 80(5):523-8. 2009.
4. Appelboam A, Reuben A, Mann C, et al. Postural modification to the standard Valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): a randomised controlled trial. Lancet. 2015;386(10005):1747-53.
5. Ortiz M et al. Randomized Comparison of Intravenous Procainamide vs. Intravenous Amiodarone for the Acute Treatment of Tolerated Wide QRS Tachycardia: the PROCAMIO Study. Eur Heart J 2016.
6. Marill KA, Wolfram S, Desouza IS, et al. Adenosine for wide-complex tachycardia: efficacy and safety. Crit Care Med. 2009;37(9):2512-8.
7. Sheinman BD, Evans T. Acceleration of ventricular rate by fibrillation associated with the Wolff-Parkinson-White syndrome. Br Med J (Clin Res Ed). 1982;285(6347):999-1000.
8. Schützenberger W, Leisch F, Gmeiner R. Enhanced accessory pathway conduction following intravenous amiodarone in atrial fibrillation. A case report. Int J Cardiol. 1987;16(1):93-5.
9. Gaita F, Giustetto C, Riccardi R, Brusca A. Wolff-Parkinson-White syndrome. Identification and management. Drugs. 1992;43(2):185-200.
10. Boriani G, Biffi M, Frabetti L, et al. Ventricular fibrillation after intravenous amiodarone in Wolff-Parkinson-White syndrome with atrial fibrillation. Am Heart J. 1996;131(6):1214-6.
11. Tijunelis MA, Herbert ME. Myth: Intravenous amiodarone is safe in patients with atrial fibrillation and Wolff-Parkinson-White syndrome in the emergency department. CJEM. 2005;7(4):262-5.
12. Stiell IG, Clement CM, Rowe BH, et al. Outcomes for Emergency Department Patients With Recent-Onset Atrial Fibrillation and Flutter Treated in Canadian Hospitals. Ann Emerg Med. 2017;69(5):562-571.e2.