

Out of breath: Guide to Tachyarrhythmias

Check out our other study guides as well as free podcasts and free board review quizzes at www.emboardbombs.com

Author: Blake Briggs, MD

Twitter/Instagram: @emboardbombs



Board Bombs

Objectives: approach to tachyarrhythmias, their EKG features, treatments, and complications

Stable vs Unstable

-the most important step. Determines treatment, disposition.

Steps:

- 1- Stable vs unstable?
- 2- P waves present?
- 3- Regular or irregular?
- 4- QRS wide or narrow (<3 small boxes)?

Sinus tachycardia is NOT included in this discussion today as it is considered an “appropriate” response to some pathology. One should address underlying causes (infection, trauma, PE, etc).

Today in a general nutshell: Stable = meds first Unstable = shock first
Heart rates >140 are generally considered to be pathologic as this is when cardiac output begins to decrease.

- 1) Stable vs unstable: Assess ABC’s (protecting airway and verbalizing, breathing, BP >90)
- 2) P waves? Initial step to determine if sinus rhythm or not. Rate?
 - a. Must be upright and in every lead.
- 3) Wide or narrow?
 - a. Narrow = above the AV node (atrial origin)
 - b. Wide = below the AV node (BBB, Purkinje system, ventricular myocytes)
- 4) Regular or irregular?
 - a. Regular = predictable rhythm.
 - b. Irregular = rhythm is not predictable due to electricity coming in variable responses from the SA node.

Narrow		Wide	
Regular	Irregular	Regular	Irregular
<p>DDx:: PSVT (AVNRT), Atrial flutter, Orthodromic WPW</p> <p>AVNRT: premature atrial beats sent to ventricle WPW: premature atrial beat sent through ectopic channel. A flutter: large circuit of premature beats in the atrium at rate of ~300. Only half of those make it to the ventricle (hence 150 being a constant heart rate).</p> <p><u>Stable tx:</u> Vagal maneuvers (CN X blockade of AV node). Syringe, lift legs. Adenosine → cures most PSVT, unveils WPW and A flutter rhythm as it slows. Verapamil, Diltiazem</p> <p><u>Unstable tx:</u> Synchronized cardioversion at 100 J with increasing by 50 as needed.</p>	<p>DDx: A fib, A flutter with block, Multifocal atrial tachycardia</p> <p><u>Stable tx:</u> A fib: irregularly irregular. Rate control with long acting Diltiazem > Metoprolol > Esmolol > Digoxin. Not trying to “short the circuit”, but slow it done to improve cardiac output.</p> <p>A flutter with block: (A fib-A flutter mix) same tx as above.</p> <p>MAT: looks like A fib but has weird looking p waves piled on each other. Associated with COPD/chronic lung disease exacerbations. Treat lung disease.</p> <p><u>Unstable tx:</u> Synchronized cardioversion at 200J.</p>	<p>DDx: Ventricular tachycardia, SVT with aberrancy, Antidromic WPW, hyperkalemia, TCA OD</p> <p>“VTach”: monomorphic (most common variant). Sustained = >30 seconds, nonsustained <30s. Features that suggest VT: broad QRS complexes >160 ms, “capture and fusion beats”, AV dissociation with p waves marching through, Josephson’s Sign, RSR’ complex with L>R taller, each QRS is nearly identical.</p> <p><u>Stable tx:</u> IV Amiodarone, Mg. <u>Unstable tx:</u> 100J</p> <p>SVT with aberrancy: mimics VT. SVT with BBB which results in wide fast rhythm. <u>Stable tx:</u> give adenosine → converts back to SR.</p> <p>Antidromic WPW: treat as orthodromic. Due to current moving first down the accessory pathway then back up. Procainamide.</p>	<p>DDx: Atrial fibrillation with aberrancy, Polymorphic VT, WPW with A fib</p> <p>A fib with aberrancy: most common cause. Same tx as before for A fib. <u>Stable tx:</u> Drugs, <u>Unstable tx:</u> shock</p> <p>Polymorphic VT: “Torsades”. Give Mg, pace into tachycardia (fast HR = shorter QT intervals)</p> <p>WPW with A fib: rare, deadly, hard to interpret. No 2 QRS complexes look the same! <u>Stable and Unstable tx:</u> cardioversion 200J. Procainamide is the only safe agent here as it won’t block AV nodal conduction (which would kill the patient as it sends the WPW accessory pathway into overdrive).</p>