June 2017 | Volume 1 Issue 6

EM:RAP C3
Continuous Core Content

www.emrap.org

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Tachyarrhythmias

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* *Drug doses are a guide only, always check second source and follow local practice guidelines*

Take Home Points:

- **Patients with tachycardias can be divided into groups based on a series of simple questions, leading to a stepwise approach:**
  - **STEP 1: IV, O2, monitor, crash cart with airway equipment and defibrillator to the bedside**
  - **STEP 2: Is the patient stable or unstable?**
  - **STEP 3: Are P waves present?**
  - **STEP 4: Is it regular or irregular?**
  - **STEP 5: Is it wide or narrow?**
- **Electricity (e.g., electrical cardioversion) is generally indicated over medications (e.g., chemical cardioversion) in unstable patients with tachyarrhythmias**
- **Cardioversion is unlikely to be of benefit hemodynamically when the ventricular rate is less than about 140 bpm**
- **Sinus tachycardia is the most common tachyarrhythmia and it is not treated with cardioversion or rate control agents – treat the underlying cause (hemorrhage, sepsis, pulmonary embolus, etc.)**

Introduction

Managing patients with tachycardia is as central to emergency medicine as airway, breathing, and circulation. In many cases, a rapid heart rate is the most prominent and obvious part of a patient's presentation – and in some cases it demands immediate action from the treatment team.

In this episode of C3, we have attempted to make the approach to patients with tachyarrhythmias simple "but not too simple", as a great man (Albert Einstein!) once said.

Background: The Primacy Of Rate

- Increasing our heart rate helps us adapt to an increased need for blood flow.
- Once the ventricular rate exceeds about 140, heart rate no longer increases cardiac output (CO); CO rather begins to decrease because there is insufficient time for the ventricle to fill in diastole
- In patients with heart rates >140 or so, we generally aim to decrease the heart rate, because it is no longer adaptive or helpful
  - In the case of sinus tachycardia, we do this through our resuscitation (e.g., fluids, blood products, antibiotics)
  - In the case of an abnormal heart rhythm (AFib or PSVT), we do this through direct action on the heart (e.g., AV nodal blocking agents)

The Approach

- **STEP 1: IV, O2, monitor, crash cart with airway equipment and defibrillator to the bedside**
  - When we intervene, whether with medications or electricity, the stakes are high and the risk of adverse events is real
  - All antiarrhythmic drugs are pro-arrhythmic and even electrical cardioversion can result in lethal rhythms
  - We must be prepared to run a full resuscitation code every time, including good vascular access and the ability to deliver electricity
    - Most defibrillators now use hands-free paddles, these should be applied as soon as possible and before any intervention
- **STEP 2: Is the patient stable or unstable?**
  - Electricity (e.g. electrical cardioversion) is generally indicated over medications (e.g. chemical cardioversion) in unstable patients
  - Patients are traditionally considered "unstable" when they have chest pain, altered mental status, pulmonary edema or systolic BP < 90 mmHg
  - This traditional definition of "unstable" does not always serve us well
    - All of these criteria are relative
    - The majority of patients will report some sort of chest pain and they are not necessarily unstable

- Many patients who have SBP <90 are stable enough to manage without electricity
- Genuinely unstable patients with tachyarrhythmias may be electrically cardioverted (typically after administration of a sedative!) even if the underlying rhythm is unknown
  - The major pitfall here is trying to cardiovert a patient with a sinus tachycardia (see Step 3) - it won't help!

- **STEP 3: Are P waves present?**
  - An upright, normal appearing P wave before each QRS complex suggests sinus tachycardia
  - Each P must look the same and be upright in leads II, inverted in avR
  - Patients with sinus tachycardia tend to have a variable rate with their movement in the bed; patients with circuit rhythms such as PSVT or Aflutter have a fixed heart rate (e.g. always almost exactly 150 on the monitor)
  - Many patients with sinus tachycardia will be unstable and in shock due to an underlying cause (hemorrhage, sepsis, PE) - they need blood or fluids or fibrinolytics, not cardioversion!
  - Are there "special" P waves? These may help make the diagnosis:
    - Extremely fast, irregular, chaotic = Atrial fibrillation (AFib)
    - Regular sawtooth pattern at 300 bpm = Atrial flutter (A-flutter)
    - Distinct P waves of variable morphology = Multifocal atrial tachycardia (MAT)

- **STEP 4: Is it regular or irregular?**
  - This is a key distinction:
    - An irregular rhythm is not ventricular tachycardia (e.g. the rhythm is coming from above the AV node)
    - Treatment of an irregular rhythm is thus to block the AV node

- **STEP 5: Is it wide or narrow?**
  - This is a key distinction:
    - A narrow rhythm is not ventricular tachycardia (e.g. the rhythm is coming from above the AV node)
    - Treatment of an narrow rhythm is thus to block the AV node
    - Narrow QRS is <0.12msec (3 small squares)

The 4 Groups of Tachyarrhythmias

- **1. Narrow, Regular (but NOT sinus tachycardia)**
  DDx: Paroxysmal supraventricular tachycardia (PSVT)
  Atrial flutter
  Orthodromic WPW

  - Paroxysmal supraventricular tachycardia (PSVT)
    - PSVT is a confusing term (there is no shortage of those here)
    - If taken literally PSVT refers to any sudden-onset tachyarrhythmia arising at or above the AV node
    - In common usage, however, it most often refers to **AV Nodal re-entrant tachycardia (AVNRT)**
      - AVNRT is a "short circuit" in the AV node
      - The circuit can be triggered by an premature atrial beat
      - Once triggered, the short circuit causes repeated regular impulses to be transmitted to the ventricle
      - AVNRT can be terminated by vagal maneuvers or AV nodal blocking agents
    - **Orthodromic WPW** (Wolf-Parkinson White) is another cause of PSVT where the "short circuit" goes down through the AV node (hence the narrow complex) and then back up to the atrium via an accessory pathway
      - It is treated the same way as AVNRT (with AV nodal blockade to break the circuit)

  - Atrial flutter
    - Atrial flutter is also a circuit, in this case a larger "macro- circuit" that runs around the atrium and includes the AV node
      - Atrial flutter typically occurs at a rate of 300
      - The AV node will only typically transmit every other (or some other fixed ratio) of these beats
        - A 2:1 transmission ratio (most common) thus results in a ventricular rate that consistently stays at 150, a very common heart rate in patients with atrial flutter

  - The **Valsalva maneuver**, which involves the patient bearing down or straining against a closed airway, can stimulate the vagus nerve and cause AV nodal blockade; this can terminate PSVT and convert the patient to sinus rhythm
    - A recently studied modification of the Valsalva (the REVERT trial), where the legs are elevated immediately after the Valsalva strain, found increased rates of success (40% vs 17% for regular Valsalva)

  - **Adenosine** is both diagnostic and therapeutic
    - Adenosine is an ultra-short acting AV nodal blocker
      - It must be given rapidly to work (e.g. through a large bore IV, rapid push, and raising the arm after administration)
      - Dose in adults is 6 mg, if ineffective 12 or 18 mg may be tried
      - Doses need to be adjusted in patients with transplanted

hearts and on certain medications such as theophylline, and doses through central lines should be halved
        - Patients should be warned that they will often feel a severe sense of discomfort or chest pain as the medication takes effect; this is uncomfortable but transient and self-limited
        - A transient period of asystole is also seen on the monitor (this few seconds can seem like hours!)
    - In PSVT, the AV nodal blockade will break the circuit and terminate the arrhythmia, restoring the patient to sinus rhythm (this makes the diagnosis clear)
    - In atrial flutter, the AV nodal blockade will transiently unmask the sawtooth-like flutter waves during the time of the blockade (also making the diagnosis clear)
- **Calcium channel blockers** such as verapamil and diltiazem can also be used
    - These are long acting AV nodal blockers
    - They will also terminate PSVT
    - In the case of atrial flutter, they will not terminate the flutter but will slow the ventricular rate of response
        - Diltiazem 10-20mg IV over 2 minutes (maximum 60mg in 30 minutes) and may need to continue as infusion
        - Verapamil 2.5-10mg IV slowly, maximum 20 mg
- **Electrical cardioversion** at a low dose (e.g., 25-100J) can also be used in patients that are unstable

- **2. Narrow, Irregular**
  DDx: Atrial fibrillation
  Atrial flutter with variable block
  Multifocal atrial tachycardia
    - Atrial fibrillation (AFib)
        - If a rhythm is very irregular (e.g. irregularly irregular) it is likely AFib or a variant of AFib
        - Treatment consists of either conversion or rate control
            - We will discuss cardioversion for Afib (both chemical and electrical) in a separate C3
            - Rate control is with long acting AV nodal blockade; here we are not breaking a circuit but rather slowing the conduction of impulses from the erratic atrial activity to the ventricles
                - Calcium channel blockers PO or IV (see above)
                - Beta blockers
                    - Metoprolol PO (25-50mg) or IV (2.5-5mg)
                    - Esmolol IV (can be easily titrated or stopped due to its short half-life)
                - Digoxin
                    - Used to be more commonly employed for rate control in AFib
            - **Electrical cardioversion** at a higher doses (e.g., 200J) can also be used in patients that are unstable
    - Atrial flutter with variable block
        - It can sometimes be difficult to distinguish between AFib and Aflutter with a highly variable block (e.g. a block of 2:1 then 3:1 then 3:2 in rapid succession)
        - Some have even coined the term "AFib-flutter" e.g., an irregularly irregular rhythm with sawtooth like flutter waves
        - Treatment is generally the same either way (e.g. AV nodal blockade)
    - Multifocal atrial tachycardia (MAT)
        - This resembles AFib but instead of fibrillation waves (representing hundreds of ectopic foci in the atrium), there are only a few variable appearing P waves (a few different ectopic foci)
        - Classically associated with COPD exacerbations, the treatment is directed at the underlying cause (e.g. albuterol for airway obstruction)

- **3. Wide, Regular**
  DDx: Ventricular tachycardia (VTach)
  Supraventricular tachycardia (SVT) with aberrancy
  Antidromic WPW
    - Ventricular tachycardia (VTach)
        - VTach should always be the initial assumption with a fast, wide, regular rhythm, especially in older patients
- If untreated, VTach can deteriorate into a cardiac arrest rhythm
- Stable patients can receive an antiarrhythmic agent such as adenosine, magnesium, or amiodarone
- Unstable patients can be cardioverted (200J)
    - Supraventricular tachycardia (SVT) with aberrancy
        - This is a common mimic of VTach
        - It is essentially a tachycardia from the atrium (e.g. PSVT or Aflutter) in a patient with a bundle branch block (BBB), which results in a fast, wide, regular rhythm mimicking VTach
        - These can be distinguished by administering adenosine (see above)
            - In SVT with aberrancy, the patient will convert to sinus rhythm (they may or may not have a wide complex QRS (secondary to their bundle branch block)
            - In VTach, there will be no change, and you may then proceed to cardioversion
    - Antidromic WPW
        - Another VTach mimic
        - Here, the "short circuit" goes down the accessory pathway (hence the wide complex) and then back-up to the atrium via the AV node
        - It is treated the same way as orthodromic (narrow complex) WPW - with AV nodal blockade to break the circuit

- **4. Wide, Irregular**
  DDx: Atrial fibrillation (AFib) with aberrancy
  Polymorphic VTach
  WPW with AFib

  - Atrial fibrillation (AFib) with aberrancy (e.g., BBB)
    - This is the most common cause of a wide irregular rhythm
    - Treatment is long-acting AV nodal blockade (see above), and cardioversion if unstable
  - Polymorphic VTach
    - This is a the so-called "Torsades de pointes"
    - It is a wide complex tachycardia characterized by **undulating amplitude** (bigger and then smaller and then bigger sine wave pattern)
    - It is treated with magnesium IV (in 2g increments), overdrive pacing and/or cardioversion
  - WPW with AFib
    - This is rare and deadly
    - It is characterized by a rapid, **chaotic appearance**, where no 2 QRS complexes look the same
    - These patients are usually ill-appearing and unstable
    - Treatment is with **cardioversion** (200J)
      - AV nodal blockade (with any drug) is dangerous
      - AV nodal blocking agents accelerate the conduction through the accessory pathway and can result in ventricular fibrillation and cardiac arrest)
      - Procainamide is the sole medical agent thought to be safe in this context

Disposition

- Disposition varies widely depending on the scenario above
- Patients with PSVT that are converted to sinus in the ED and many patients with AFib can be safely discharged home after appropriate treatment and follow-up instructions
- Patients with potentially lethal arrhythmias (typically VTach) and/or hemodynamic instability are usually admitted to a cardiac care unit or intensive care unit
- In some cases, it is better not to intervene on a tachyarrhythmia – especially if it is well tolerated and the patient appears stable
  - In major centers such patients can sometimes be taken directly to the electrophysiology suite with the cardiologists for definitive diagnosis and management

References

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