When evaluating a tachyarrhythmia one should first evaluate whether the QRS is a narrow complex or a wide complex. Next one should determine if the rhythm is irregular or irregular, as this will help narrow down your differential diagnosis of possible causes. **Supraventricular tachycardia (SVT)** refers to any tachyarrhythmia that arises from above the Bundle of His. To further classify SVT one can distinguish between regular and irregular rhythms.

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AV Nodal Re-entry Tachycardia (AVNRT) is the most common re-entrant rhythm seen in the ED. Common causes include caffeine ingestion, exertion, or other drug ingestions in patients with structurally normal hearts. Note the regular narrow complex tachycardia (typically 140-280 bpm).

Treatment includes:

- Vagal Maneuvers to increase vagal tone
- Adenosine to block transmission through the AV node
- Synchronized cardioversion (especially for hemodynamically unstable or refractory SVT)
- Beta/calcium channel blockers can be considered but caution should be used as their effects can last long after conversion of rhythm

AVRT is an anatomic re-entry circuit (Bundle of Kent) within the heart.

Conduction of AVRT may be orthodromic (traveling down the normal direction of the nerve fibers) or antidromic (opposite to the normal conduction pathway). Antidromic conduction tends to cause a wide QRS complex whereas orthodromic often causes a narrow complex.
Atrial fibrillation is an irregularly irregular rhythm with variable conduction through the AV node. In evaluation of a patient with atrial fibrillation with RVR or atrial flutter, first clinicians must determine whether a patient is hemodynamically stable or not. In unstable patients treatment is immediate synchronized cardioversion.

For stable patients with atrial fibrillation over 48 hours, rate control is the primary focus as rhythm control risks thromboembolism.

Rate control can be achieved with beta blockers (for example metoprolol 2.5-5 mg IV bolus over 2 minutes up to 3 doses), calcium channel blockers (diltiazem 0.25 mg/kg IV bolus over 2 minutes with a second bolus if needed), or digoxin (0.25mg IV every 4-6 hours up to 1mg)

All patients with atrial fibrillation should have their thromboembolic risk assessed by using the CHA$_2$DS$_2$-VASc score. This accounts for the patient’s age, sex, and history of CHF, hypertension, stroke/TIA, vascular disease, or diabetes. A score greater than or equal to 2 should prompt consideration of anticoagulation.

For hemodynamically stable patients with onset of atrial fibrillation less than 48 hours ago the following treatments may be considered:

- Elective synchronized cardioversion
- Medication for rhythm control (amiodarone, flecainide, ibutilide, etc)
**Atrial flutter** is a re-entry tachycardia in the right atrium which causes an atrial rate of 300 bpm. The actual ventricular rate is determined by the degree of the AV block (how often the atrial impulse results in ventricular conduction). For example, in atrial flutter with 2:1 block the EKG would show a regular rate of about 150 bpm as every other atrial impulse results in ventricular conduction. Atrial flutter with 3:1 block would have a rate of about 100 bpm. Flutter waves are usually best seen in leads II, III, and aVF.

When approaching a narrow complex rhythm, some sources suggest unmasking flutter waves with adenosine (AVNRT/AVRT will often convert to sinus rhythm whereas atrial flutter should show flutter waves). Suspect atrial flutter with a 2:1 block if you see a very regular narrow complex rhythm at 150 bpm.

Rate controlling medications can be used to attempt to control atrial flutter (as you would use in atrial fibrillation). Unlike atrial fibrillation, atrial flutter is often responsive to synchronized cardioversion at low voltages.
The other “irregularly irregular” rhythm is **multifocal atrial tachycardia (MAT)**. MAT is most often seen in patients with severe lung disease. The criteria for diagnosing MAT are at least 3 different P wave morphologies typically with slightly different PR intervals. Varying P wave morphologies and PR intervals are the result of different ectopic foci. Treatment of MAT is not usually necessary—treating the underlying disease will treat the MAT, however keep in mind that hypokalemia and hypomagnesemia can exacerbate MAT.