Foundations EKG II - Unit 11 Summary

Miscellaneous EKGs

A wide complex tachycardia should always be assumed to be ventricular tachycardia and all unstable wide complex tachycardias should be immediately electrically cardioverted.

With that said, wide complex tachycardia may also represent antidromic **AV Re-entry tachycardia (AVRT)** or **AVNRT with aberrancy** (for example a bundle branch block). Features that **favor VT** over AVRT include:

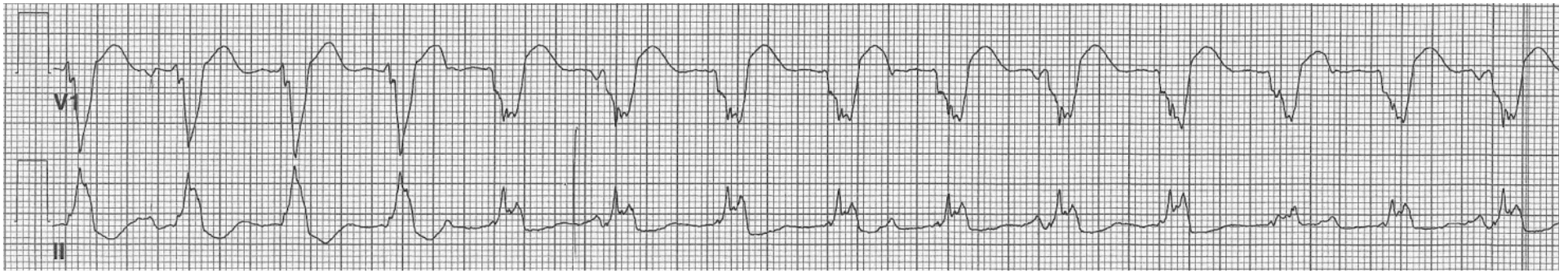
- QRS > 140 ms
- AV dissociation
- fusion or capture beats
- unidirectional V1-V6, and extreme axis dissociation.

These can be hard to distinguish. When in doubt treat as VT with synchronized cardioversion, amiodarone, or procainamide. One important thing to keep in mind is that if you suspect antidromic AVRT (in other words, a tachycardia with a re-entry rhythm traveling retrograde through the AV node by an accessory pathyway (Bundle of Kent)) avoid amiodarone as it has AV nodal blocking properties.

When an ectopic ventricular pacemaker exists and conducts at a rate higher than the sinus node's rate, the resulting rhythm is an **Accelerated Idioventricular Rhythm (AIVR)**. Most classically this is seen in the reperfusion phase of an acute STEMI. This is a benign rhythm and does not require administration of antiarrhythmics.

The identifying features of AIVR include:

- Regular rhythm at a rate of 50-110 bpm
- Wide QRS complexes > 120ms
- Fusion and capture beats



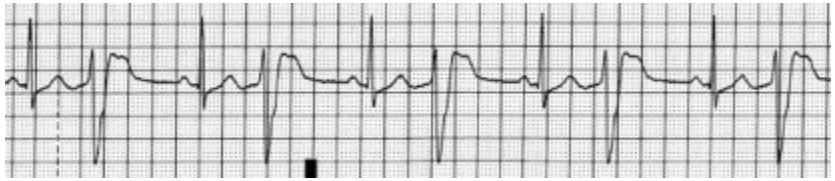
Note that the **RATE** of AIVR separates it from other ventricular rhythms. A ventricular rate of less than 50 bpm is called a **ventricular escape rhythm**, while a rate over 110 bpm is called **ventricular tachycardia.**

In addition to reperfusion, possible causes of AIVR include:

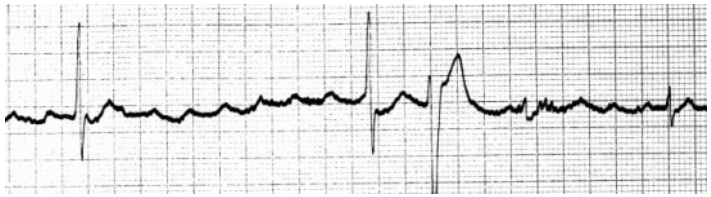
- Epinephrine
- Cardiomyopathy
- ROSC after a cardiac arrest
- Drug toxicity including cocaine and digoxin

In addition to causing GI upset as well as CNS and visual disturbances, **digoxin toxicity** can cause dysrhythmias. Because of increased automaticity and decreased AV conduction, digoxin toxicity may cause supraventricular tachycardia with a slowed ventricular rate. Frequently seen dysrhythmias include:

- Frequent PVCs. Consider digoxin toxicity in the appropriate patient who has ventricular bigeminy



- Slow atrial fibrillation (remember, this is the increased supraventricular rate with slow ventricular response)



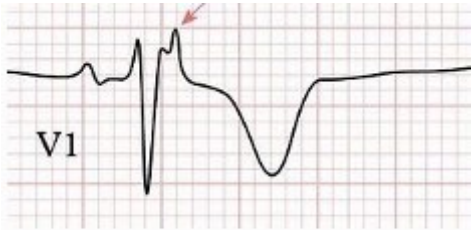
- Ventricular tachycardia
- AV block

For patients with acute toxicity (unstable dysrhythmia, K > 5, or over 10mg single ingestion by an adult) or chronic toxicity with lift threatening arrhythmias, treatment with digibind is indicated. For slow ventricular rhythms, atropine may also be used.

Patients with a family history of sudden cardiac death should be evaluated both for HOCM (discussed elsewhere) and for **arrhythmogenic right ventricular cardiomyopathy (ARVC)**. In this inherited condition right ventricular myocardium is replaced by fat or by fibrous tissue. This deadly condition causes palpitations, syncope, or arrest due to ectopic ventricular beats or ventricular tachycardia.

EKG findings suggestive of ARVD includes:

- Epsilon wave (a small positive deflection at the end of the QRS complex)
- T wave inversion
- Prolonged S wave upstroke in V1-V3
- QRS widening in V1-V3
- Paroxysmal episodes of ventricular tachycardia with left bundle branch



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Patients with suspected ARVC should have immediate cardiology consultation for possible AICD placement. As with HOCM, beta-blockers may be used as initial treatment.

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