Cardiovascular Dysrhythmia, Blocks, Delays

EMRAM Resident Review
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2020

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General Principles

- ABCD and primary survey - check for stability
  - Signs of Instability – Hypo-perfusion, chest pain from ischemia, dyspnea from pulmonary edema, extremely rapid rate, altered mental status
- Supraventricular – originate above AV node and are generally narrow complex
- Ventricular - result from below AV node and are wide complex
- Regular vs Irregular

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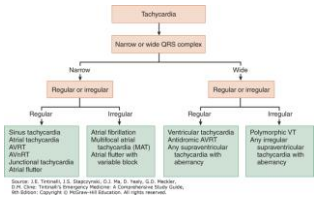

Tachycardia classification. AVNRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reentrant tachycardia; VT = ventricular tachycardia.

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Atrial Fibrillation – Narrow and Irregular

- **Irregularly irregular narrow complex without p waves**
- Commonly associated with heart disease
- Also cardiomyopathy, alcohol use, myocarditis, chest trauma
- Treatment is rate vs rhythm control, cardioversion if unstable
- Prevention of complications from thrombus – anticoagulation vs Watchman device

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Three examples of atrial fibrillation with irregular ventricular response.

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Multifocal atrial tachycardia

- **Irregularly irregular, at least 3 distinct p waves, variable P-R and R-R intervals**
- Typically found with those with **chronic lung disease**, but also in other acute disorders like sepsis
- **Treatment directed at underlying disorder**

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Multifocal atrial tachycardia. Note multiple P-wave morphologies with irregular tachycardia rhythm.

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Atrial Flutter

- **Regular narrow complex tachycardia, P waves are downward deflected and "saw tooth" in appearance**
- Atrial rate usually 250-300 and block determines ventricular rate, often 2:1 (150 rate) or 3:1 (100)
- **A regular narrow complex tachycardia with rate 150 likely Flutter with 2:1 conduction**

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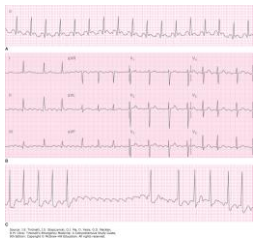

Atrial flutter. A. Regular, narrow-complex tachycardia at a ventricular rate of 155 beats/min. B. Atrial flutter with flutter waves most visible in leads 2, 3, and aVF. C. Atrial flutter response to carotid sinus massage inducing transient AV block and unmasking flutter waves.

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Narrow Complex Tachycardia Treatment

- Rate Control
  - Cardizem – 15 to 20 mg bolus followed by 5-10 mg/h
  - Metoprolol – 5 mg IV, repeat to total of 15 mg
- Rhythm Control
  - Procainamide- 15-17 mg/kg IV over 30 min, then 1-4 mg/min
  - Esmolol – 50 mcg/kg/min, increase by 50 mc/kg/min max 200
  - Amiodarone - 150 mg IV over 10 min, then 0.5 mg/min
- Synchronized Cardioversion
  - Atrial flutter 50J
  - Atrial Fibrillation 150-200J

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Supraventricular Tachycardia

- **Narrow Complex, no p waves (or inverted), usually 170 -180 bpm**
  - Can be 130-300
- Vagal maneuvers attempted, often fail in clinical practice
- Adenosine (stable) or cardioversion (unstable)
  - Adenosine is a short acting nodal blocker of AV node and interrupts reentry if AV node responsible
  - Beta blockers or calcium channel blockers if fail adenosine
  - Cardioversion

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Paroxysmal supraventricular tachycardia.

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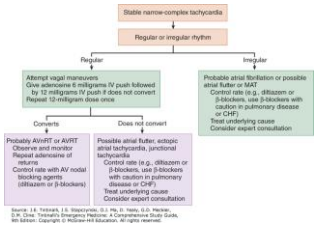

Treatment of narrow-complex tachycardia. AV = atrioventricular; AVnRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reentrant tachycardia; CHF = congestive heart failure; MAT = multifocal atrial tachycardia; VT = ventricular tachycardia.

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Ventricular tachycardia

- **Regular wide complex, rate usually 140-180 bpm**
- Rare in those without heart disease, common in ischemia or valvular disorder, toxic ingestion, cardiomyopathy
- Monomorphic vs polymorphic
- Incorrectly assumed to be unstable

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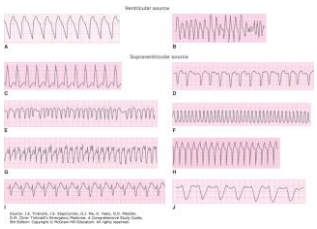

Wide-complex tachycardias. A. Ventricular tachycardia (monomorphic). B. Ventricular tachycardia (polymorphic). C. Sinus tachycardia with bundle branch block. D. Atrial fibrillation with preexisting bundle branch block. E. Preexcited atrial fibrillation in the Wolff-Parkinson-White syndrome. F. Atrioventricular reentrant tachycardia in the Wolff-Parkinson-White syndrome. G. Paroxysmal supraventricular tachycardia with rate-related bundle branch block in an infant. H. Paroxysmal supraventricular tachycardia with fixed (preexisting) bundle branch block. I. Sodium channel–blocker toxicity with wide-complex tachycardia. J. Wide-complex tachycardia in patient with severe hyperkalemia.

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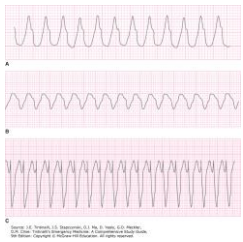

Three examples of monomorphic ventricular tachycardia. A. Ventricular tachycardia with a rate of 270 beats/min. B. Ventricular tachycardia with a rate of 220 beats/min. C. Ventricular tachycardia with a rate of 180 beats/min.

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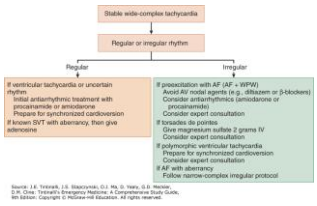

Treatment of wide-complex tachycardia. AF = atrial fibrillation; AV = atrioventricular; SVT = supraventricular tachycardia; WPW = Wolff-Parkinson-White syndrome.

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Wide Complex Tachycardia Treatment

- Procainamide - 15-17 mg/kg IV over 30 min, then 1-4 mg/min
- Lidocaine – 1-1.5 mg/kg IV then repeat after 5 min, max 300
- Amiodarone - 150 mg IV over 10 min, then 0.5 mg/min
- Magnesium – if long QT or Torsades

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Ventricular Fibrillation

- **Fine, irregular, without p waves or organized QRS complex**
- Seen most often in those with significant ischemic heart disease
- Can happen after period of shock, direct instrumentation to the heart, or trauma *commotio cordis*
- ACLS and defibrillation

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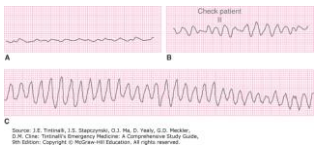

Three examples of ventricular fibrillation. A. Fine amplitude. B. Coarse amplitude. C. Coarse amplitude mimicking ventricular tachycardia.

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AV Blocks

- First degree - delay of conduction, prolonged PR
- Second degree – intermittent AV conduction vs block
  - Second degree Mobitz I and first degree not usually associated with problems
- Third degree – AV block
  - Third degree and Mobitz II are abnormal and often associated with compromised hemodynamics
- If unstable most are 3rd degree, followed by 2nd degree. If stable most likely 2nd degree

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First Degree

- **Prolonged PR interval due to abnormal conduction**
- Usually asymptomatic, often incidentally found
- Can be seen with increased vagal tone, ischemia, cardiomyopathy
  - In setting of ischemia can lead to increased likelihood of abnormal conduction

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Sinus rhythm with first-degree atrioventricular block (PR interval, 300 milliseconds).

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Second Degree - Mobitz I

- **Mobitz I (Wenckebach) – Prolonged PR widening, leading to drop**
  - Each depolarization leads to prolonged refractory period, leading up to a non-conducted beat
  - Often transient, associated with ischemia, recent cardiac surgery, cardiomyopathy
  - Most often treatment not necessary, but will respond to atropine

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Second-degree, type I atrioventricular block. Note the nonconducted P waves (arrows).

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Second Degree – Mobitz II

- **Mobitz II – PR interval constant, but then dropped**
  - Usually due to infra-nodal conduction system delay, often associated with fascicular or bundle branch blocks
  - Usually wider complex
  - "High grade" if more than one non-conducted p wave
  - If 2:1 block, then can be difficult to differentiate between I and II, if wide assume II
  - Usually irreversible problem with pacemaker, can progress to Third degree and will need pacer if unstable

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Three examples of second-degree, type II atrioventricular (AV) block: (A) with narrow QRS complex rhythm; (B) with wide QRS complex rhythm; and (C) with wide QRS complex rhythm and high-grade AV block indicated by two or more consecutive nonconducted P waves (arrows).

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Third Degree

- **No AV conduction, escape pacemaker at slower rate than atrial**
  - At AV node - Junctional escape rhythm, narrow complex 40-60 rate
  - Infra-nodal – Ventricular escape, wide, less than 40 rate
- **Can be associated with RCA infarct**
- Nodal blocks will respond to atropine, infra-nodal usually will not

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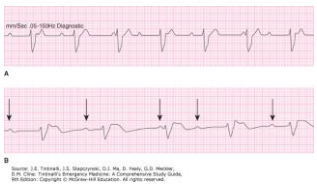

Two examples of third-degree atrioventricular block. Both have an atrial rate of 83 with ventricular escape rate of 50. Not all P waves are visible; some are hidden by the QRS complex or T wave.

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Bradycardia External

- 80% of brady-dysrhythmia are from factors external to the electrical system such as ACS, medications or overdose, hypoxia and hypoperfusion
  - Tox/Medications: ABCD
    - Alpha adrenergic agents
    - Beta-Blockers
    - CCB
    - Digoxin
    - Exposure (tick bites, hypothermia) or Everything Else (Thyroid)

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Bradycardia Treatment

Emergent treatment is not required unless:

- Heart rate is slower than 50-60 with signs of hypotension or hypoperfusion – resuscitation and treatment of underlying cause
  - Vasopressors – Epinephrine or Norepinephrine
  - Atropine – Increases automaticity of SA node and increases conduction. Useful for sinus or junctional rhythms but not useful for blocks
  - Glucagon – stimulates ionotropic and chronotropic cardiac activity independent of Beta-adrenergic receptors, useful for CCB or BB overdose, Temporary
  - High dose Insulin for CCB or BB
  - Transcutaneous/transvenous pacing is definitive care in structural disease

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Conduction Abnormalities

- WPW
- Brugada
- Long QT
- ARVD

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Wolf Parkinson White

- Accessory pathway leads to pre-excitation conduction abnormality
- **Triad of EKG findings**
  - **PR shortened <120 msec**
  - **Slurring of initial QRS, called delta wave**
  - **Slightly widened QRS (due to delta)**
- Usually found in asymptomatic individuals
- Most common dysrhythmia is an orthodromic AV reciprocating tachycardia (narrow) where 5-10% will get antidromic AV reciprocating tachycardia (wide)

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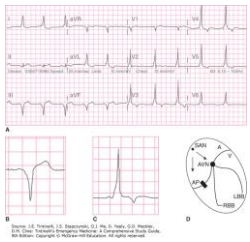

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A. A 12-lead ECG of a patient with the Wolff-Parkinson-White (WPW) syndrome in sinus rhythm. B and C. Single P-QRS-T complex; note the shortened PR interval, delta wave, and widened QRS complex. D. The direction of cardiac impulse conduction in the WPW syndrome patient in sinus rhythm. The two arrows indicate the direction of the impulse, moving from atrial (A) tissues to ventricular (V) tissues via the accessory pathway (AP) and atrioventricular node (AVN). The impulse arrives at the ventricular tissues via the AVN and moves through ventricular tissues via the right bundle branch (RBB) and left bundle branch (LBB); the impulse also arrives at ventricular tissues via the AP and moves through ventricular tissues using cell-to-cell conduction. SAN = sinoatrial node.

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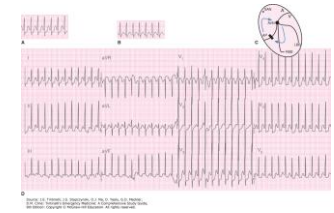

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Narrow-complex tachycardia (orthodromic atrioventricular [AV] reciprocating tachycardia [AVRT]), the most common dysrhythmia of the Wolff-Parkinson-White syndrome. A and B. Rapid, regular, narrow QRS complexes without delta waves. C. The direction of cardiac impulse conduction in orthodromic AVRT; anterograde from atrial (A) tissues to ventricular (V) tissues through the AV node (AVN) and returning to the atrial tissues from ventricular tissues via the accessory pathway (AP). D. A 12-lead ECG demonstrating orthodromic AVRT. AP = accessory pathway; LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.

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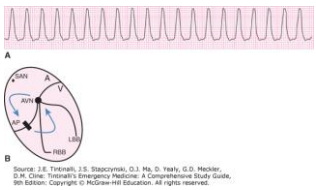

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Antidromic atrioventricular (AV) reciprocating tachycardia (AVRT; wide-complex tachycardia). A. Wide-complex tachycardia that mimics ventricular tachycardia. B. The direction of cardiac impulse conduction in a Wolff-Parkinson-White syndrome patient during antidromic AVRT. The two arrows indicate the direction of the impulse movement, anterograde from atrial (A) tissues to ventricular (V) tissues through the accessory pathway (AP) and returning to the atrial tissues from ventricular tissues via the AV node (AVN). LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.

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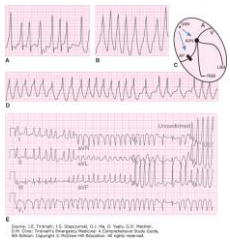

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Atrial fibrillation in Wolff-Parkinson-White (WPW) syndrome. A, B, and D. Rapid, wide, irregular QRS complex tachycardia with varying QRS complex morphologies. C. The direction of cardiac impulse conduction in a WPW syndrome patient during atrial fibrillation. The two arrows indicate the direction of the impulse movement, both anterograde from atrial (A) tissues to ventricular (V) tissues through both the accessory pathway (AP) and the atrioventricular nodle (AVN). The larger of the two arrows indicates that the majority of impulses travel to the ventricular via the AP. E. A 12-lead ECG demonstrating a rapid, wide, irregular QRS complex tachycardia with varying QRS complex morphologies. LBB = left bundle branch; RBB = right bundle branch; SAN = sinoatrial node.

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Treatment

- Stable vs unstable
- Orthodromic AVRT and regular – SVT pathway (vagal, adenosine, CCB and BB, then procainamide)
- **If Wide complex and irregular – procainamide for Atrial Fibrillation with WPW, then cardioversion**
  - **Don't use BB, CCB, Adenosine or Amiodarone in Wide complex and irregular**

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Brugada Syndrome

- Congenital abnormality of Na, K, or Ca channels in the heart
- Leads to sudden cardiac death and malignant dysrhythmia
- Usually asymptomatically found
- Symptoms include palpitations, presyncope, "seizures"

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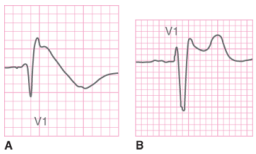

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Brugada ECG pattern. A. Type 1. B. Type 2.

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Long QT Syndrome

- Congenital cation channelopathy that results in prolonged Qtc
  - Predisposed to ventricular tachydysrhythmia or sudden cardiac death
- Acquired
  - Hypokalemia, hypomagnesia
  - Medication side effects
  - Ischemia
- Over 440 msec is considered abnormal, greater than 500 msec is where problems typically occur
- Beta blockade is usually treatment of choice

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Arrhythmogenic Right Ventricular Dysfunction

- ARVD is inherited form of right ventricular dysplasia and at risk for sudden cardiac death
- Usually found with palpitations and syncope, from V tach, but can be seen in shock, cardiac arrest
- EKG
  - Epsilon wave - J point irregularity found in only 30% of patients
  - T wave inversions in V1-V3
  - QRS widening in V1-V3

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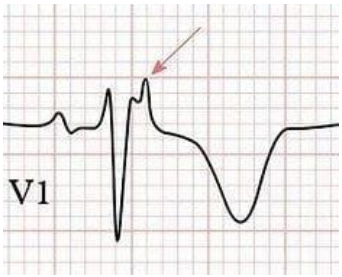
Epsilon Wave

- https://litfl.com/epsilon-wave-ecg-library/



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Epsilon Wave

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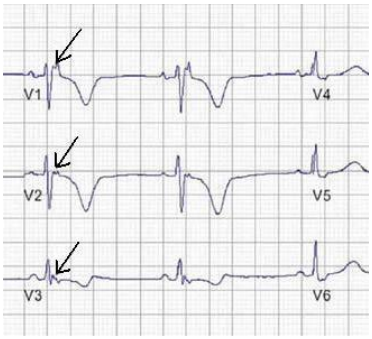

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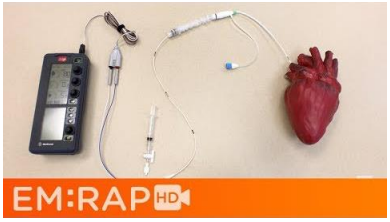
Transvenous Pacing

(https://emcrit.org/ibcc/bradycardia/#transvenous_pacing)



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Resources

- https://litfl.com
- Tintinalli Emergency Medicine Study Guide 9th edition online
- https://emcrit.org/ibcc/bradycardia/#transvenous_pacing
  - EMRAP HD video from emcrit

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