NOTES
PRE-EXCITATION DISORDERS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Heart rhythm disturbances due to accessory pathway in conduction system that allows depolarization to bypass atrioventricular node and spread from atria to ventricles
- Accessory pathways can promote arrhythmias by two mechanisms
  - Acting as one limb of a reentrant circuit, with atrioventricular node acting as the other
  - Bypassing physiologic atrioventricular nodal delay → impulses reaching ventricle not regulated → very rapid ventricular responses in atrial arrhythmia setting such as atrial fibrillation, atrial flutter
- If ventricular rate becomes too high → ventricles don't have time to fill → low cardiac output → shock

SIGNS & SYMPTOMS

- Tachyarrhythmias → palpitations, chest discomfort, breath shortness, lightheadedness, syncope

DIAGNOSIS

- See individual disorders

TREATMENT

- Acute termination of preexicitation-associated arrhythmias
- Chronic prevention of preexicitation-associated arrhythmias

MEDICATIONS

- Acute termination
  - *Adenosine:* short acting; causes transient heart block (↓ rate of diastolic depolarization, ↓ HR)
  - *Diltiazem (Class IV):* calcium channel blocker (↓ AV node conduction → ↓ HR)
- Chronic prevention
  - *Amiodarone (Class III):* slows conduction rate (↑ AP duration, ↑ QT interval)
  - *Procainamide (Class 1A):* slows conduction velocity (↑ AP duration, ↑ ventricular refractory period, ↑ QT interval)

OTHER INTERVENTIONS

- Acute termination
  - Vagal maneuver (carotid sinus massage/ Valsalva maneuver) → activates vagus nerve
  - Electrical cardioversion (if pharmacological treatment ineffective/ fast heart rate is poorly tolerated)
- Chronic prevention
  - Radiofrequency catheter ablation (definitive treatment)

AV REENTRANT TACHYCARDIA (AVRT)

osms.it/av-reentrant-tachycardia

PATHOLOGY & CAUSES

- Arrhythmia due to accessory pathway between atria and ventricles that allows electrical signal to move backwards

Orthodromic atrioventricular reentrant tachycardia (AVRT)

- Signal moves downward through atrioventricular node → ventricles contract → upward through accessory pathway → atria contract → moves back down atrioventricular node → etc.

Antidromic atrioventricular reentrant tachycardia (AVRT)

- Signal moves downward through accessory pathway → ventricles contract → upwards through atrioventricular node → atria contract → moves back down the accessory pathway → etc.

SIGNS & SYMPTOMS

- Tachyarrhythmias → palpitations, chest discomfort, breath shortness, lightheadedness, syncope

DIAGNOSIS

OTHER DIAGNOSTICS

ECG

- Orthodromic AVRT
  - Regular, narrow-complex tachycardia, P waves are typically retrograde in morphology and come after QRS complex
- Delta wave is not seen
- Antidromic AVRT
  - Regular, wide-complex tachycardia, P waves often not visible

TREATMENT

MEDICATIONS

- Acute termination
  - Adenosine, Diltiazem (Class III)
- Chronic prevention
  - Amiodarone (Class III), Procainamide (Class 1A)

OTHER INTERVENTIONS

- Acute termination
  - Vagal maneuver
  - Electrical cardioversion (if pharmacological treatment ineffective/ fast heart rate is poorly tolerated)
- Chronic prevention
  - Radiofrequency catheter ablation

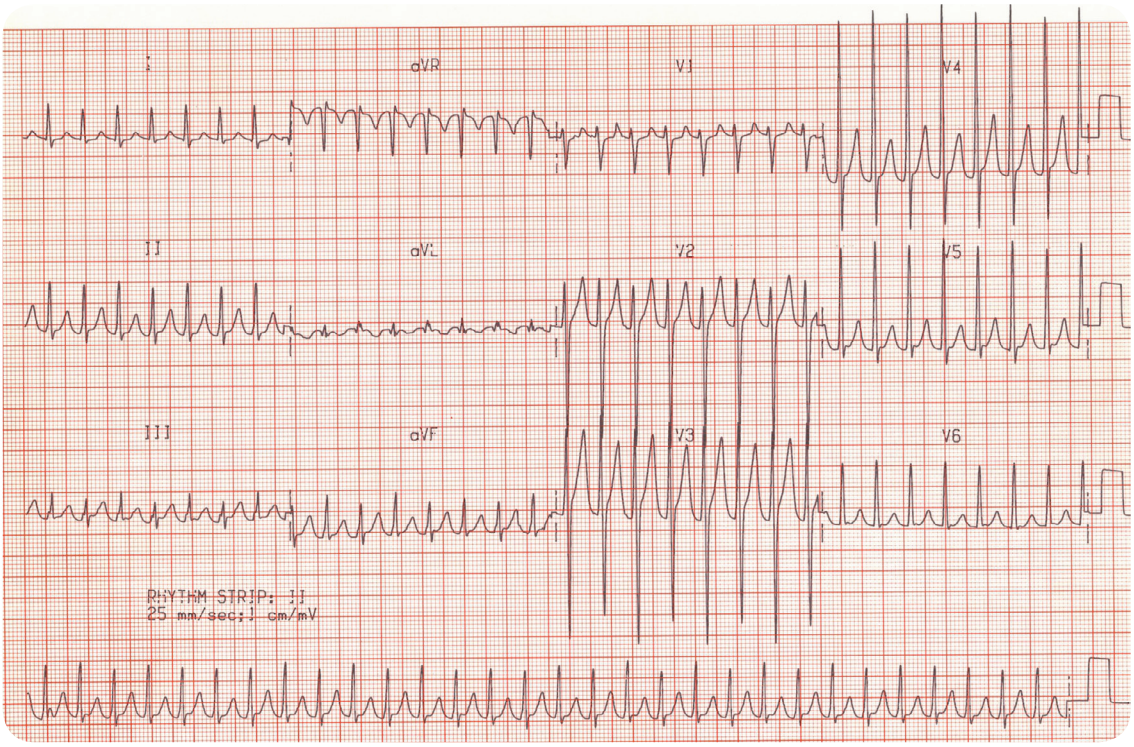

Figure 16.1 An electrocardiogram demonstrating othodromic AVRT. Note the narrow QRS complexes and absence of a discernible P wave.

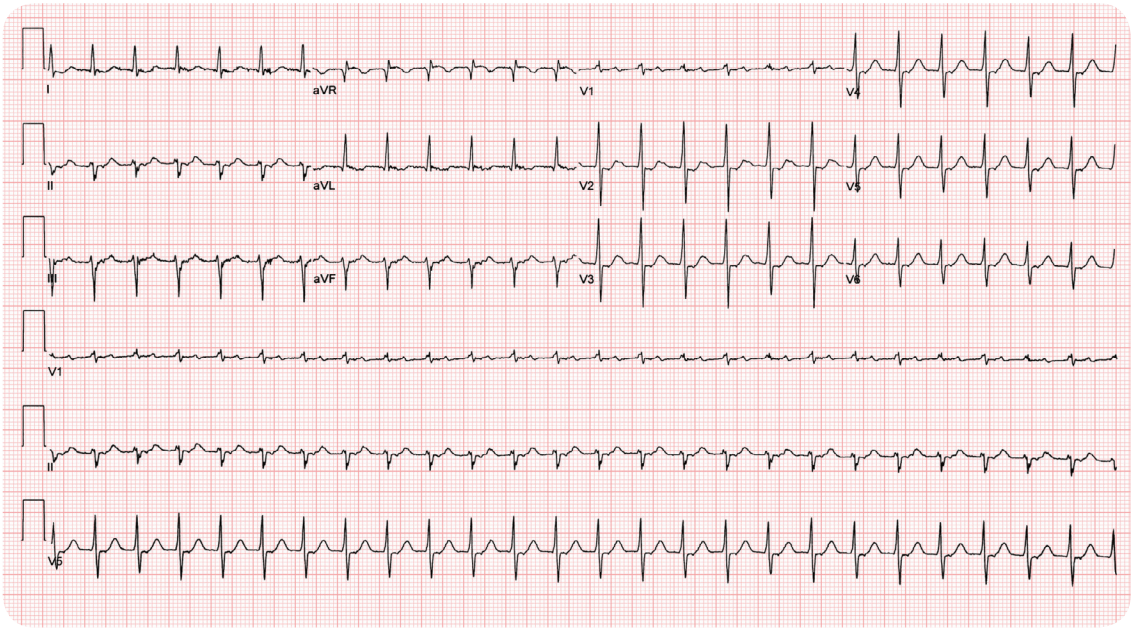

Figure 16.2 An ECG demonstrating orthodromic AVRT with regular, narrow-complex tachycardia with retrograde P waves visible just after the QRS complexes, most visible in leads II and V4-V6.

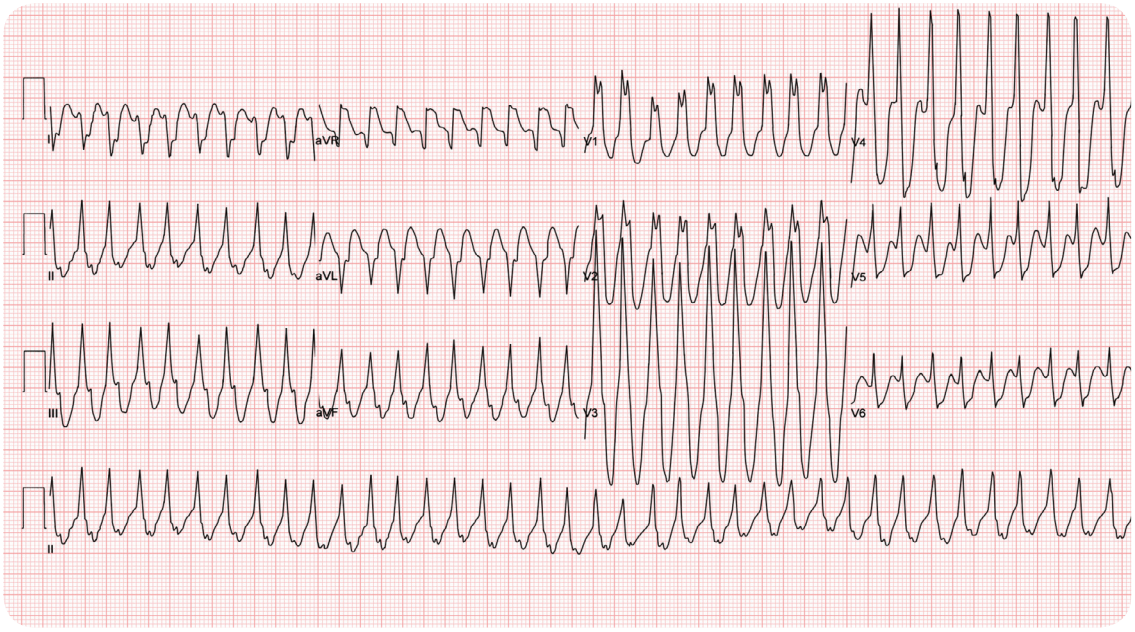

Figure 16.3 An ECG demonstrating antidromic AVRT. There is a regular, wide complex tachycardia that is usually indistinguishable from VT.

WOLFF–PARKINSON–WHITE SYNDROME

osms.it/wolff-parkinson-white-syndrome

PATHOLOGY & CAUSES

- Congenital accessory pathway conducts electrical signals between atria and ventricles → preexcitation, predisposes individuals to clinically significant arrhythmias up to sudden cardiac death
  - Though "bundle of Kent" is a common eponym for congenital accessory pathway, several different pathways can occur, most commonly direct atrioventricular connections, but also atriofascicular, nodofascicular, atrio-Hisian, etc.
  - *Wolff–Parkinson–White pattern:* benign asymptomatic form, solely described by compatible electrocardiographic changes
- Most common type of ventricular preexcitation syndrome
  - 0.1% of individuals have Wolff–Parkinson–White pattern, a small proportion of them develops syndrome

SIGNS & SYMPTOMS

- Tachyarrhythmias → palpitations, chest discomfort, breath shortness, lightheadedness, syncope

DIAGNOSIS

OTHER DIAGNOSTICS

ECG

- Short PR interval (< 120ms)
- Delta wave
  - Slurred upstroke of QRS
- Widening of the QRS complex (> 110ms)
- Secondary ST segment, T wave changes

TREATMENT

MEDICATIONS

- Acute termination
  - Adenosine, Diltiazem (Class III)
- Chronic prevention
  - Amiodarone (Class III), Procainamide (Class 1A)

OTHER INTERVENTIONS

- Acute termination
  - Vagal maneuver
  - Electrical cardioversion (if pharmacological treatment ineffective/ fast heart rate is poorly tolerated)
- Chronic prevention
  - Radiofrequency catheter ablation

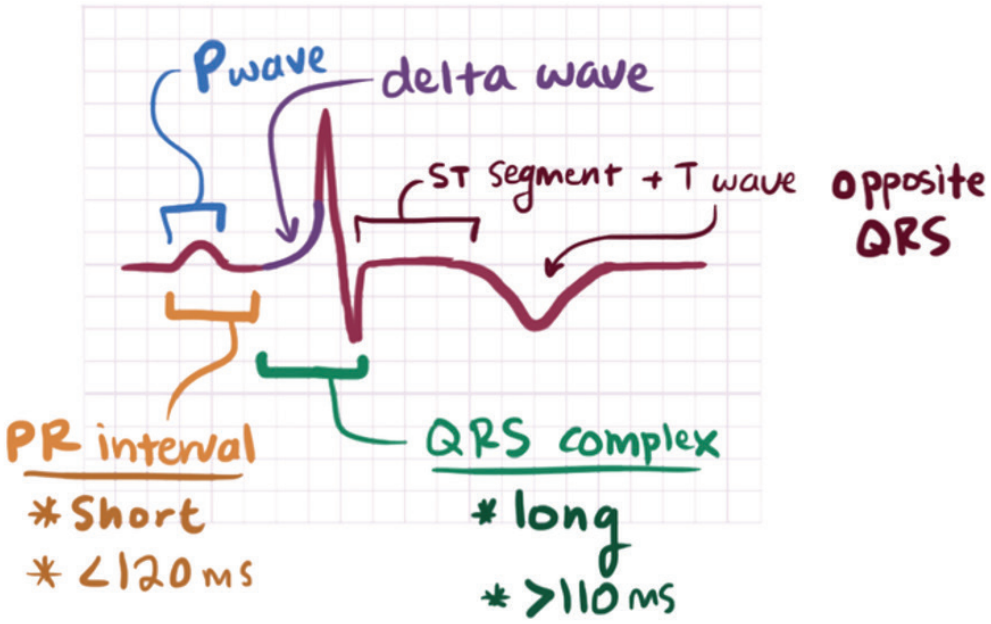

Figure 16.4 ECG pattern in Wolff–Parkinson–White syndrome.

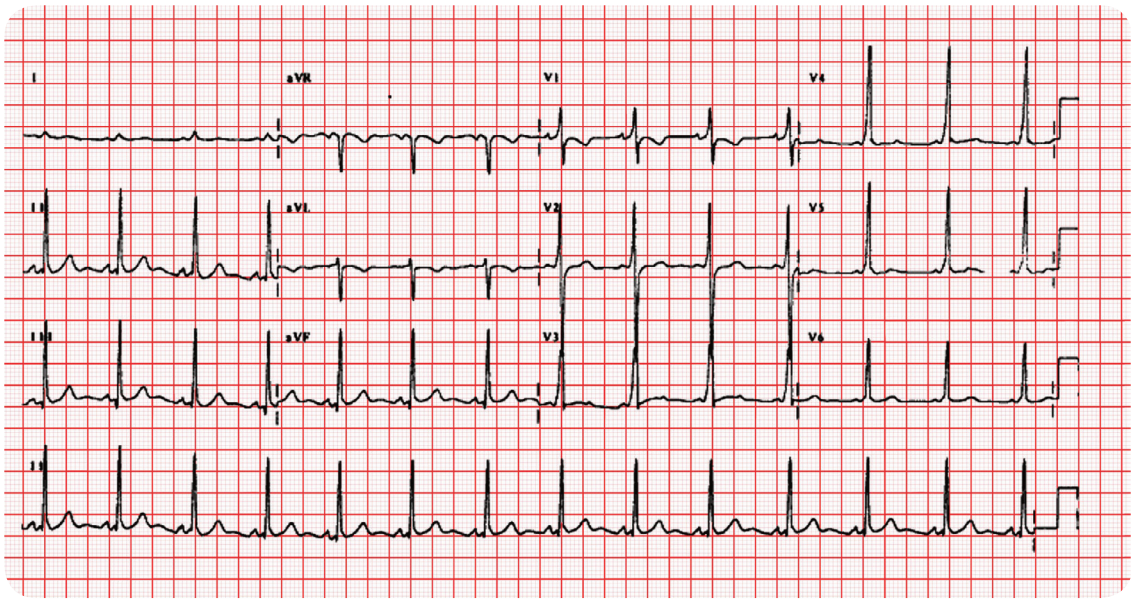

Figure 16.5 An ECG of an individual with Wolff–Parkinson–White syndrome (sinus rhythm). Delta waves are most visible in the V leads.

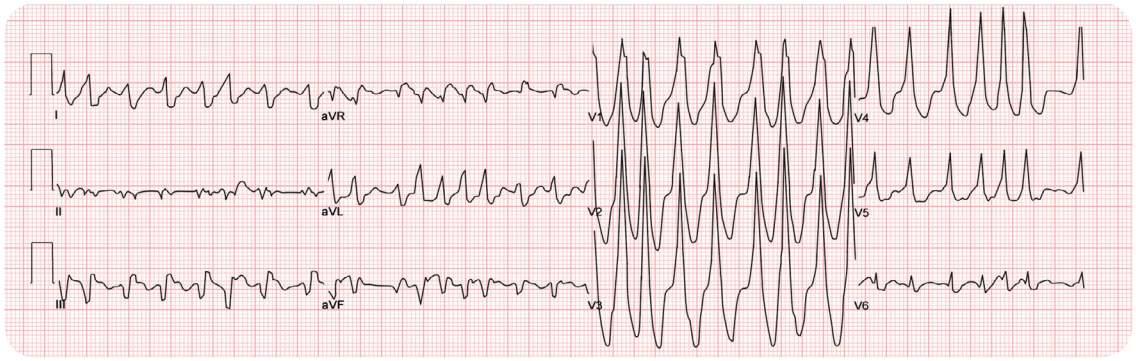

Figure 16.6 An ECG demonstrating "pre-excited a-fib" or atrial fibrillation in a person with Wolff–Parkinson–White. It's an irregularly irregular wide-complex rhythm with no discernible P waves.