Wide Complex Tachycardias

Definitions: Tachycardia is defined as a sustained rhythm in an adult with a rate equal to or exceeding 100 beats per minute. Wide complex is defined as a QRS complex equal to or greater than 120 msec. A non-sustained tachycardia is a rhythm disturbance lasting for 3 or more QRS complexes at a rate exceeding 100 and lasting less than 30 seconds.

Tachycardias are characterized by their origin proximal to (supraventricular) or distal to the AV Node (ventricular), and/or whether they result from ectopic or “automatic” foci or a reentry mechanism:

Supraventricular Tachycardias
1. Sinus Tachycardia
2. Atrial Tachycardia
3. Atrial Flutter
4. Atrial Fibrillation
5. Multifocal Atrial Tachycardia
6. Reentry Tachycardias
   a. AV Nodal Reentry Tachycardia (AVNRT)
   b. AV Reentry Tachycardia (AVRT)

Ventricular Tachycardias
1. Ventricular Tachycardia, Unifocal (monomorphic)
2. Ventricular Tachycardia, Multifocal (or polymorphic, including Torsades de Pointes)
3. Ventricular Flutter
4. Ventricular Fibrillation

Wide Complexes can result from the following mechanisms:

Conduction Blocks
1. Intraventricular conduction delay (IVCD)
2. Bundle Branch Block Fixed or Rate Related
   a. Right Bundle Branch Block (RBBB)
   b. Left Bundle Branch Block (LBBB)

Fusion Complexes

Infranodal Origin
1. Fascicular
2. Ventricular
3. Accessory Pathway
3. Paced

Toxicity or Electrolyte Abnormality
1. Hyperkalemia
2. Antiarrhythmic Drugs
ECGs with Wide Complex Tachycardia: Supraventricular Origin

058 Sinus Tachycardia with Rate-Related Left Bundle Branch Block. The rate is 125 and the QRS duration is 160 msec, with broad R waves in Leads I, aVL, and V5-6 indicating LBBB. A break in the rhythm is caused by a right ventricular premature complex, followed by a compensatory pause that allows the ventricular conduction system to recover partially, and reveal P waves exhibiting left atrial enlargement that are partially obscured by the T waves during tachycardia.

Calipers demonstrate the Compensatory Pause. The R-R interval bracketing the VPC is equal to 2 RR intervals during the tachycardia because the sinus node is impervious to the premature beat.
352. Atrial Flutter with 2:1 AV Block and Right Bundle Branch Block. The atrial rate is 240 and the ventricular rate is 120, indicating 2:1 block which is a result of the protective Wenckebach mechanism in the AV node. A saw tooth pattern is seen in Lead III, (partially obscured by T waves), helping to establish the mechanism of the underlying rhythm as atrial flutter.

The obscured atrial waves are identified by dividing the interval between obvious atrial waves (12 small boxes = 480 msec) by two, and placing plumb lines midway between (6 small boxes = 240 msec) as shown in this close up. The saw tooth pattern in Lead III indicates the continuous circular wave of atrial depolarization parallel to the frontal plane, while V1 records their appearance as discrete P waves in the horizontal plane.
169 Atrial Fibrillation with Accessory Bypass Tract Conduction (WPW). The rhythm is irregularly-irregular, and the QRS complexes are wide (>160 msec) with slurred upstrokes and followed by T waves with opposite polarity. The 6 second ruler is used to determine the rate in this irregular rhythm, and 20.5 cycles occur in 6 seconds, yielding an average rate of 205 per minute, with some RR intervals equivalent to a rate of 300. The QRS complexes are rapidly conducted from the fibrillating atria through an accessory atrioventricular connection and resemble ventricular tachycardia, except for the grossly irregular rhythm. The next ECG was recorded from the same patient several minutes later.
170. **Atrial Fibrillation with WPW (same patient as 169).** The extremely rapid, irregular, wide-complex rhythm has a mean ventricular rate of 240 (range 167 to 375). The majority QRS complexes are wide and bizarre in appearance, typical of ventricular tachycardia, but the gross irregularity is incompatible with VT. Several narrower QRS complexes are seen in the first and last third of the ECG. These narrow complexes indicate variable contribution from conduction through the AV Node and His-Purkinje system. Beats a and b are fusion beats, as are the last two complexes in V1-3, while c represents “pure” AV conduction. This potentially fatal rhythm was promptly treated with countershock. The V1 rhythm strip below was obtained after defibrillation.

171. **Sinus Tachycardia (108) with WPW.** The PR interval is 100 msec and the QRS resembles b complexes recorded during atrial fibrillation (above) and represent fusion between concurrent AV Nodal conduction and conduction through the accessory pathway. The QRS duration is 120 msec and the QRS upslopes are slurred as are the majority beats in 170. The c beat duration in 170 is 80 msec, lacking contribution from the accessory pathway, and therefore a result of normal His-Purkinje conduction.
705. WPW with AV Reentry Tachycardias: Orthodromic and Antidromic. Two rhythm strips obtained 38 seconds apart demonstrate both narrow and wide complex tachycardias. The narrow complex tachycardia (above) has a rate of 190 that accelerates to 214 during the last 5 complexes. The wide complex rhythm has a rate of 235. The arrhythmias utilize both atrioventricular conduction pathways in sequence rather than concurrently as seen in 171. **Orthodromic AVRT** results from normal anterograde conduction through the AV Node followed by reentry from ventricle to atrium via the accessory pathway, so the QRS configuration is normal. **Antidromic AVRT** is less common form of AVRT, and is easily mistaken for ventricular tachycardia since the QRS configuration reflects depolarization initiated in, and propagated through, the ventricles.
1738  Atrial Flutter with Intermittent Conduction through Accessory Pathway (WPW).
Atrial flutter, rate 290 is present throughout, and AV conduction is 2:1 conduction through the AV node. Bypass tracts lack the “breaking action” inherent in the AV Node, and as shown in the last three electrocardiograms, are capable of conducting 1:1 in atrial flutter and atrial fibrillation, often with lethal outcomes. As noted previously, depolarization of the ventricles from the bypass tract’s site of insertion emulates the wave of depolarization in ventricular tachycardia so the QRS complexes are broad and slurred, with T waves opposite in polarity to the QRS.
713. Non-sustained Ventricular Tachycardia (NSVT) with AV Dissociation and Fusion Complexes. This ECG was chosen to introduce Ventricular Tachycardia because of the clear demonstrations of features that may be difficult to recognize in sustained Ventricular Tachycardia. Note the presence of 3 different populations of QRS complexes in the V1 rhythm strip: 4 upright, followed by 3 isoelectric, 5 more upright, and ending with 4 negative complexes. The two "parents" in this ECG are the first 4 QRS complexes of ventricular tachycardia (VT) and the last 4 in sinus rhythm (SR). Fusion complexes are hybrids, with contributions from both parents. Slight fluctuations in the rates of the 2 parent rhythms permit the 2 to coincide to form the fusion complexes. AV Dissociation is seen when the first three P waves of sinus origin follow the VT complexes, and retrograde (V-A) conduction prevents the P waves from capturing the ventricle. The 4th P wave breaks through to contribute, along with the VT to cause 3 fusion complexes. These two features, AV Dissociation and Fusion in about 1/3 of VT episodes, and when seen in a wide complex tachycardia, confirm the diagnosis of VT. The 4th from last QRS also exhibits fusion, best seen in lead V4.
145. **Ventricular Tachycardia with AV Dissociation and Fusion Complexes.** This wide complex tachycardia has 2 fusion complexes (near the beginning and the end of the ECG) which strongly support the diagnosis of VT because it implies that AV Dissociation is present.