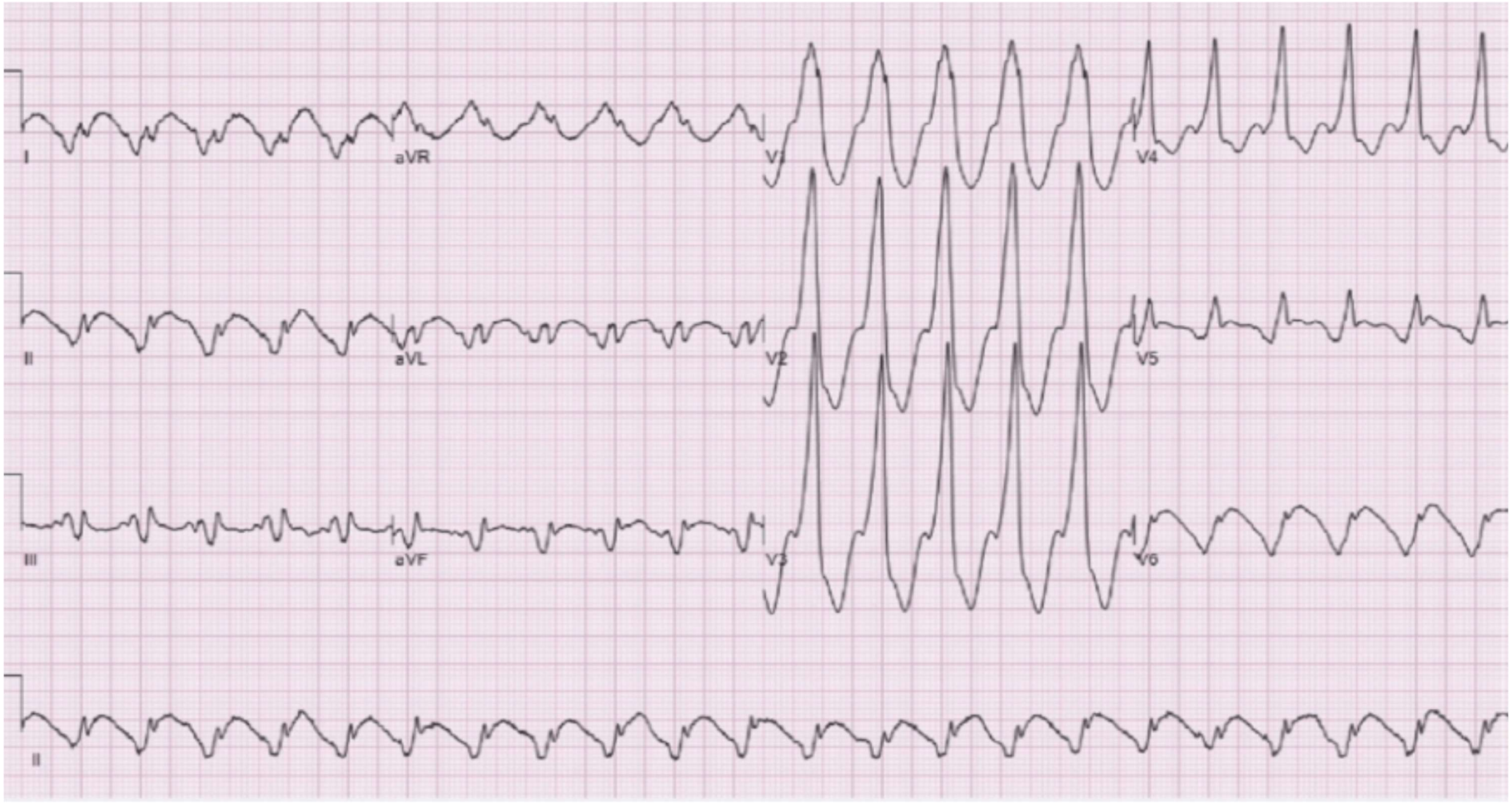
• Ventricular tachycardia (VT)



Factors favoring VT as a cause of the wide-complex tachycardia

- Very broad complexes (>160 msec), often with bazaar notching in QRS.
- Absence of typical RBBB or LBBB morphology.
- QRS is positive in aVR and negative in I + aVF. Extreme axis deviation ("northwest axis").
- AV dissociation (P waves independent of QRS complexes).
- Capture beats; sinus beat transiently 'captures' ventricles, resulting in an isolated QRS complex of normal duration.
- Fusion beats; simultaneous arrival of sinus and ventricular beat fuse into a hybrid complex of intermediate morphology.
- Positive or negative concordance across the precordial chest leads. This is where leads V1-V6 show entirely positive (R) or entirely negative (QS) complexes, and no RS complexes are present in precordial leads.
- The time from the onset of the QRS complex to the nadir of the S-wave is > 100 msec.
- RBBB-like RSR' complexes where "left rabbit ear" is taller than the right. This is in contrast to usual RBBB, in which the right rabbit ear is taller.

Rapid succession of three or more ventricular premature complexes at a rate > 100 BPM. RR interval is usually regular but may be slightly irregular

Abrupt onset and termination of arrhythmia is evident. AV dissociation is common. On occasion, retrograde atrial activation, fusion complexes and ventricular capture complexes occur. Fusion and capture complexes indicate the presence of AV dissociation.

When present, fusion and capture complexes identify the origin of a wide QRS complex as VT rather than SVT with aberrancy.

Ventriculoatrial (VA) conduction may occur at 1:1 or may manifest variable, fixed, or complete block. VA Wenckebach may also occur and manifests as a gradual prolongation of the VA interval leading to VA block (absence of a retrograde P wave).

Rarely, VT can present as a narrow QRS tachycardia.

Artifact associated with rapid arm movement (e.g., tooth brushing) with a loose electrode may be mistaken for VT on telemetry and Holter monitors.

Ventricular rhythms that originate away from the normal conduction system result in abnormal ventricular activation. This usually results in a wide QRS, axis shift, and altered QRS voltage and repolarization. As such, the following diagnoses cannot be made in the setting of VT: ventricular hypertrophy, axis deviation, BBB, myocardial ischemia and myocardial infarction.

Polymorphic VT (rapid VT with changing morphology) can be TdP or secondary to ischemia. TdP starts with a late-coupled ventricular premature complex (late R-on-T) and the QT interval is prolonged. Ischemic polymorphic VT starts with an early-coupled ventricular premature complex (early R-on-T) and the QT interval is normal.

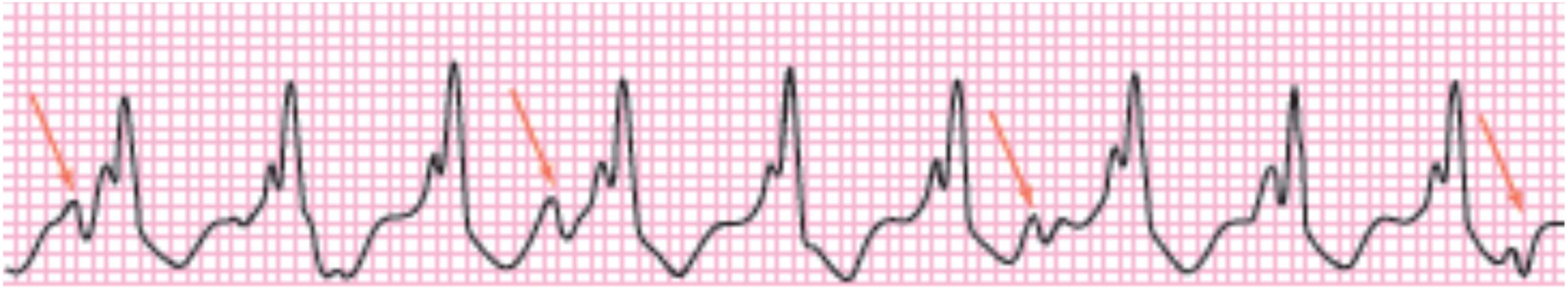
VT can be seen in:

- Heart disease (especially common in setting of heart failure)
- Hyperkalemia/hypokalemia
- Hypoxia/acidosis
- Drugs (digitalis toxicity, antiarrhythmics, phenothiazines, tricyclics, cocaine, amphetamines, alcohol, nicotine)
- Mitral valve prolapse
- Occasionally in normals

Wide QRS Complex Tachycardia: SVT with Aberrancy vs VT

A wide QRS complex tachycardia can be either SVT with aberrancy (or prior BBB) or VT.

Constant PR interval vs. AV dissociation: Searching for a P wave is the first step in identifying the cause of wide QRS complex tachycardia: The presence of a P wave before each QRS complex (often seen as a deflection in the preceding T wave) with a constant PR interval establishes the rhythm as supraventricular, whereas AV dissociation is consistent with VT.



AV dissociation (arrows mark P waves)

Fusion or capture complexes during VT help to establish that AV dissociation is present. The P wave preceding the fusion complex is often easier to identify and can then be "marched out" to identify the other P waves associated with the atrial rhythm. AV dissociation is observed in about 25% of ECGs demonstrating VT and usually requires a VT that is slow enough to allow the P wave to be distinguished from the ST-T waves.