Introduction

Wide QRS Complex tachycardia (WCT) a common arrhythmia with important therapeutic and prognostic implication and often present a diagnostic challenge. When confronted with a tachycardia having a broad QRS complex, it is important to be able to differentiate between a supraventricular (SVT) and a ventricular tachycardia (VT). Medication given for the treatment of SVT may be harmful to a patient with a ventricular tachycardia (VT). Familiarity with the electrocardiogram (ECG) sign allowing the diagnosis of a VT is therefore essential. ECG not only help to diagnose type of arrhythmia but also its etiology and its site of origin. Both these aspects are important in decision making about the prognostic significance of the WCT and correct treatment.

Definition

- Wide QRS complex tachycardia may be defined as tachycardia irrespective of site of origin having QRS duration of < 120 msec.
- Ventricular tachycardia is defined as three or more consecutive ventricular beats with a rate of 100 beat/min or more. It is defined nonsustained if it lasts less than 30 seconds and sustained if lasts more than 30 sec or requires therapeutic intervention for termination. It can originate anywhere below the AV node, including the His bundle, bundle branch, fascicles, Purkinje fibers, and ventricular tissue.
- Supraventricular tachycardia can be defined as any tachycardia using the normal AV conduction system for ventricular excitation, with tachycardia originating in the atria or AV node and requiring the AV node for its maintenance.

Etiology

Under normal circumstances, activation through the His bundle depolarizes both ventricles simultaneously through the bundle branches and the specialized Purkinje network. This process of depolarization normally takes 80–120 msec. Prolongation of QRS duration occurs in the two under mentioned condition

1. Sequentially rather than simultaneous ventricular activation seen in
   - Bundle branch block
   - Ventricular tachycardia
   - Accessory pathway (WPW synd).
Conduction abnormality over H. P. M pathway (His- Purkinje – myocardium) seen in

• Ischemia
• Drug (Procainamide)
• Electrolyte imbalance (Hyperkalemia)

### Classification

The wide complex tachycardia (WCT) can be divided in two broad group depending on regularity of the arrhythmia as depicted in Table-1

<table>
<thead>
<tr>
<th>Classification</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wide QRS tachycardia with regular rhythm</td>
<td>Ventricular tachycardia, Supraventricular Tachycardia with BBB, Antidromic AV re-entry Tachycardia</td>
</tr>
<tr>
<td>Wide QRS tachycardia with irregular rhythm</td>
<td>Torsades de pointes, LBBB with AF or AFL with Variable block, WPW with AF or AFL with variable block</td>
</tr>
</tbody>
</table>

BBB = Bundle branch block; AF = Atrial Fibrillation; AFL = Atrial Flutter

2. Conduction abnormality over H. P. M pathway (His- Purkinje – myocardium)

### Approach to Patient

The ECG and hemodynamic status of the patient is the prime guide for the management of a patient with WCT. When time available and hemodynamic status allows, history should be elicited as it gives clue regarding diagnosis as well as etiology hence should not be omitted when confronting wide QRS complex tachycardia.

#### A. History

Risk Factor: Presence of structural heart disease especially coronary artery disease and previous myocardial infarction or congestive heart failure strongly suggests for VT. In > 98% of patients with previous myocardial infarction, the cause of WCT is VT, whereas only 7% of patients with SVT have this history.

Duration: It is some help and when it has been present for more than 3 years, any SVT is more likely.

#### B. Physical Examination

AV Dissociation: Dissociation between atrial and ventricular activity during tachycardia is a hallmark of VT and the clinical sign of AV dissociation should be looked during physical examination, which are as follows.

• Cannon a waves
• Variable first heart sound
• Changes in systolic blood pressure

Vagal Maneuvers: Carotid sinus massage leading to termination of tachycardia suggests that AV node is a critical link in the tachycardia circuit and favor diagnosis of SVT.

#### C. Chest Radiograph

Presence of cardiomegaly or evidence of prior cardiovascular surgery strongly favors the diagnosis of VT because it implies underlying structural heart disease.

#### D. Electrocardiogram

Evaluation of 12-lead & rhythm strip is most important step in determining etiology of WCT. The following points should be given utmost importance while going through the ECG

1. AV Dissociation

   - Demonstration of atrio-ventricular dissociation during tachycardia is suggestive of VT.
   - But AV dissociation is present in only 60 – 75% of patient, while 25% VT patients demonstrate ventriculo-atrial conduction (VA conduction) especially at slow VT rate.
• Capture beats and fusion beats may be seen in the presence of AV dissociation which occur when a dissociated p wave totally (capture) or partially (fusion) activates the ventricle in advance of the next VT cycle.

2. QRS Axis

• A significant shift in axis during tachycardia is suggestive of VT.
• Mean QRS axis within normal range favor SVT.
• Extreme LAD (Lt. Axis deviation) or Extreme RAD or northwest axis seldom seen in conditions other than VT.

3. QRS Duration

• QRS duration more that 0.14 sec in RBBB morphology WCT and more than 0.16 sec in LBBB morphology WCT argues for a VT.5
• QRS duration is very wide when arrhythmia originate from lateral free wall leading to sequential activation of ventricle, while duration is small when it has its origin in or close to intraventricular septum.

VT origin infero-apical
→ Frontal QRS axis ↑

VT origin antero-apical
→ Frontal QRS axis ↓

• VT originating from apex has superior axis while that of originating from base has inferior axis.
• RBBB shaped WCT with superior axis and LBBB shaped WCT with inferior axis strongly suggest VT.

4. QRS Concordance.

• Not helpful when WCT is due to accessory pathway
• When all precordial leads show either negative or positive QRS complexes are called negative or positive concordance respectively.
• Negative concordance is diagnostic of VT arising from antero–apical region.
- Positive concordance is seen in VT originating from postero-basal left ventricle, or SVT due to left posterior accessory pathway.

5. QRS Narrowing During Tachycardia
- When during tachycardia the QRS is more narrower than during sinus rhythm a VT should be diagnosed. Because wide QRS during sinus rhythm is due to sequential activation become narrower during tachycardia can only be explained by a ventricular origin close to the intraventricular septum and more simultaneous activation of the two ventricles.

- When in V6 R:S ratio < 1 or QS pattern is suggestive of VT.

Left bundle branch Block pattern: 
预见 the negative in V1
- Initial positive QRS with positivity measuring more than 0.03 seconds favor VT.
- Slurring or notching of down slope of S wave
- When the V6 shows QR or QS is suggestive of VT.

- When the V6 shows QR or QS is suggestive of VT.

6. QRS Morphology

Right bundle branch Block Pattern: -预见 predominantly positive in V1
- Triphasic complex or biphasic with r < R' in V1 lead favor SVT.
- A monophasic or QR pattern in V1 lead favours VT

7. Ventricular Activation Velocity Ratio (Vi/Vt):

An index of slow conduction at the beginning and at the end of the QRS complex obtained by measuring voltage in millivolt on the ECG tracing the impulse traveled vertically during initial 40 m sec (Vi) and the terminal 40 sec(Vt) of bi or multiphasic QRS complex, in any lead having initial ventricular activation most rapid.
A ratio of $>1$ suggestive of SVT

A ratio of $<1$ is highly suggestive of VT

6. Diagnostic Criteria

Most commonly used diagnostic criteria for diagnosing SVT over VT in clinical parlance is Brugada criteria.

It has sensitivity of 99% and specificity of 97%.

A recently published new algorithm for diagnosing ventricular tachycardia by Verecrie et al using Vi / Vt ratio is that during WCT due to SVT, the activation of septum should be invariably rapid and the intraventricular conduction delay causing the wide QRS complex occurs in the mid to terminal part of the QRS. While during WCT due to VT, however, an initial slower muscle to muscle spread of activation occurs until the impulse reaches the His-purkinje system, after which the rest of myocardium is more rapidly activated and hence Vi /Vt will be greater than $<1$.

Value and limitation of E.C.G. finding in diagnosing Broad QRS tachycardia

- AV dissociation suggests VT, but VA conduction may be present during VT.

- A QRS width of $>160$ ms suggests VT, but need to rule out:
  - Pre–existent BBB (especially LBBB)
  - SVT with AV conduction over an AP
  - Use of drugs slowing intraventricular conduction (flecainide).

Keep in mind – VT arising close to or in the intraventricular conduction system may have a width of $<140$ ms

- Left axis deviation (to the left of -30 suggests VT, but is not helpful in:
  - LBBB shaped QRS
  - SVT with conduction over a right sided or posteroseptal AP
  - SVT during use of class 1 C drugs

- Right axis deviation (to the right of +90) suggests VT in LBBB shaped QRS

- Concordant pattern in precordial leads suggests VT, but positive concordance may occur during SVT with AV conduction over a left posterior AP

- R nadir S $>100$ ms in one or more precordial leads suggests VT, but may be found in:

  - SVT on drugs slowing intraventricular conduction
  - SVT with AV conduction over an AP
  - Pre–existence BBB (especially LBBB)
  - QR complexes during VT suggests previous myocardial infarction as etiology
References


