Teachable Moments in ECG

The Mighty Duck Strategy: Remaining Calm in the Face of Wide Complex Tachycardia

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ABSTRACT

In the field of medicine and cardiology, there is perhaps no other condition or situation that stimulates an adrenalin rush for the healthcare team than a patient presenting with wide QRS complex tachycardia. These cases may be potentially fatal and are usually associated with worse outcomes. While the real-world experience in the evaluation and management of these cases can be chaotic situations, a careful, systematic and organized scrutiny of the electrocardiographic tracing is key to obtaining a correct diagnosis and proceeding with the right therapeutic management. An understanding of the physiological mechanisms of arrhythmia, the appreciation of scientific basis for electrocardiographic features and recognition of different criteria for diagnosis provides endless opportunities and *"teachable moments"* in medicine. For both learners and teachers, the academic discussion of these points and features can be an exciting journey and electrifyingly educational experience. This article provides a simplified yet beautifully complicated approach to diagnosing wide complex tachycardia.

Keywords: Wide QRS complex tachycardia, ventricular tachycardia, electrocardiograph, ECG

INTRODUCTION

Wide QRS complex tachycardias are, to say the least, "nightmare cases" for the physician manning the emergency room or intensive care unit. In majority of these cases, and especially in emergency situations, the dictum is to treat and manage as ventricular tachycardia (VT), unless proven otherwise. Since VT is a dreaded cardiac arrhythmia that is potentially fatal and more importantly associated with worse outcomes in most cases, this condition usually poses challenging and tense situations for the healthcare team. One needs to immediately assess the patient, correctly decipher the tracing and decide whether to intervene immediately or not. These cases can easily present with a stormy course, adding more anxiety and tension to an already stressful situation.

Description of the Case

A 55-year-old man was brought to the emergency department because of shortness of breath and palpitations. On assessment, vital signs showed:

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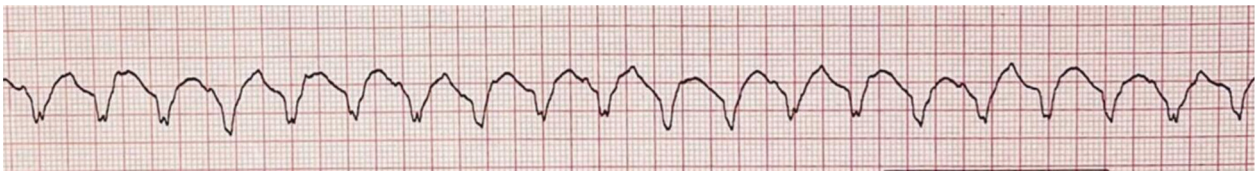

Figure 1. ECG rhythm strip of the case showing tachycardia with a rate of 125 bpm.

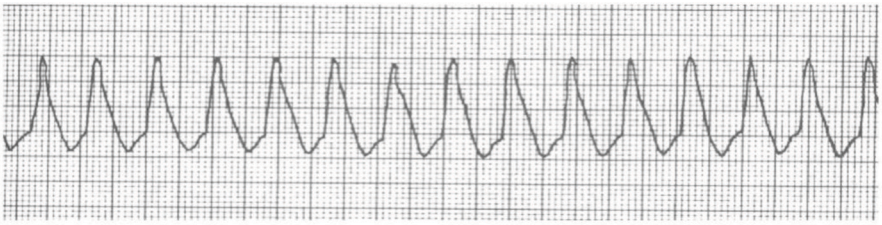

Figure 2. Wide QRS complex tachycardia is characterized by a rate of more than 100 bpm, with a QRS duration of more than 0.12 seconds or 120 ms (more than three small squares in the standard ECG strip)

blood pressure 100/70 mmHg, heart rate 130 beats per minute (bpm) regular, respiratory rate 26 per minute and temperature 36.5 degrees Celsius with an oxygen saturation of 93%. An electrocardiogram (ECG) tracing was taken (Figure 1).

Duck

It is in this crisis when the physician must adopt the duck mentality. This is a concept derived from the motivational phrase and quote from author Jason Braude: "When you are in a crisis, in a tight situation, in a struggle, in difficult moments, just be like a duck. Remain calm on the surface. But paddle like crazy underneath."[1]

Ducks epitomize calmness and composure. They appear quiet on the surface while working energetically underneath the water, paddling with a lot of vigor in the face of a storm. When faced with wide complex tachycardia, we must appear calm, but "do the necessary stuff underwater". This duck mentality is something worth adapting when we deal with challenging situations in medicine, not just tachycardias.

Decipher the Tracing

In a majority of clinical settings, complete analysis and full interpretation of the ECG tracings is usually done in retrospect, after the patient has been treated and stabilized. Within the ECG reading room of the diagnostic cardiovascular unit or Heart Station, the examination of these tracings become an engaging academic exercise between the student and expert, providing for some "teachable moments" that may be applied even up to the subspecialists' level.

Define

Wide QRS complex tachycardias or wide complex tachycardias are defined as conditions with a heart rate of more than 100 bpm and a QRS duration of more than 0.12 seconds (Figure 2).[2]

VT is a form of wide complex tachycardia which originates in the region below the Bundle of His in the cardiac conduction system. In essence, it is a tachycardia originating from the ventricular muscle. By ECG criteria, it is defined as three or more consecutive premature ventricular complexes (PVCs) with a rate of more than 100 bpm, with the QRS duration commonly more than 0.12 seconds or 120 ms in duration (Figure 3).[2]

Supraventricular tachycardia (SVT) is a tachycardia which originates above the Bundle of His, and in essence originates or involves the atrial tissue or AV node. It presents usually with a narrow QRS complex.[3] Sometimes, an SVT can conduct with an aberrancy or aberrant conduction in the form

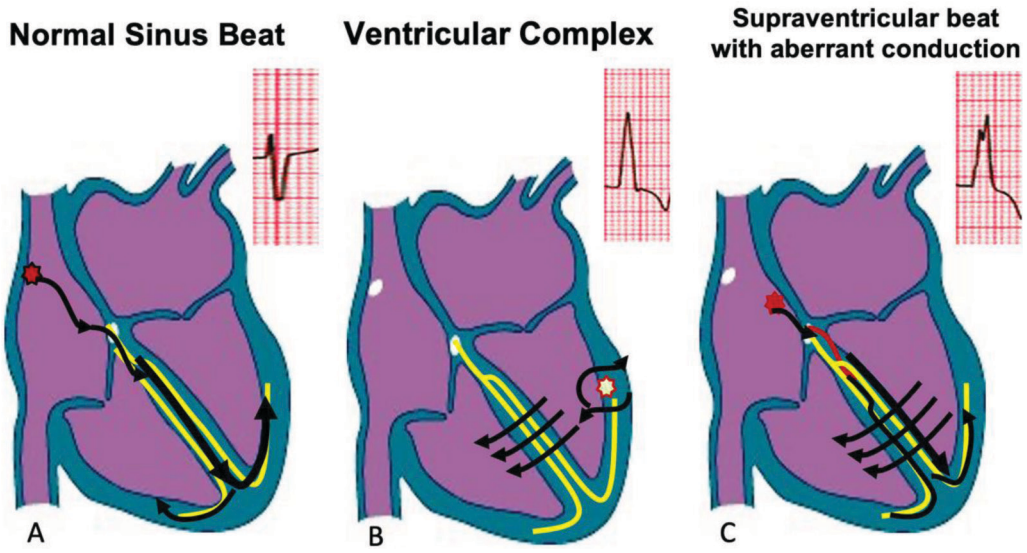

Figure 3. A. In a normal sinus beat, the impulse originates from the sinus node, leading to atrial depolarization, then conducts down the AV node and His-Purkinje system leading to ventricular depolarization and a normal narrow QRS complex. B. A ventricular complex originates from the ventricular myocardium leading to depolarization of the ventricle of origin first before the impulse conducts to the His-Purkinje system in the interventricular septum and to the opposite ventricle through cell-to-cell depolarization, leading to a wide QRS complex. C. A supraventricular beat that conducts with aberration or aberrancy originates from a focus in the atrium, which conducts to the AV node and finds one part of the conducting system (usually the right bundle branch) refractory and not able to receive the impulse. It therefore conducts down the opposite bundle branch leading to depolarization of one ventricle before the wavefront of depolarization conducts through to the other ventricle via cellto-cell depolarization, leading to a wide QRS complex.

of a bundle branch block (BBB) or intraventricular conduction delay (Figure 3), or in situations involving an antegradely conducting bypass tract or accessory pathway, leading to a wide complex pattern on the ECG.[2]

Differentials

Not all wide complex tachycardias are VT, but a majority are. By statistics, approximately 80% of wide complex tachycardias are VT, while 20% are caused by other arrhythmias.[2]

When faced with this tracing, the following differential diagnosis should come in mind:

1. Sinus tachycardia with aberrant conduction (BBB),
2. SVT with aberrant conduction (BBB),
3. Pre-excited tachycardia (sinus tachycardia with preexcitation or pre-excited atrial fibrillation seen in patients with Wolff-Parkinson-White syndrome),
4. SVT with antegrade conduction over accessory pathway (antidromic SVT),
5. Pacemaker-mediated tachycardia, and
6. VT.[4]

The first five are usually benign arrhythmias, except for pre-excited atrial fibrillation (Wolff-Parkinson-White syndrome), which can be fatal and potentially degenerate into ventricular fibrillation. Pacemaker-mediated tachycardia can happen in patients with an implanted dual chamber permanent pacemaker. As a rule, VT is a dangerous arrhythmia although there are some exceptions such as idiopathic VT, which are benign tachycardias in patients with structurally normal hearts. In most cases, VT requires immediate intervention as it can lead to hemodynamic instability and degenerate into ventricular fibrillation, resulting in cardiac arrest. Furthermore, the treatment of VT is different for other conditions in this list. Therefore, the correct interpretation of wide complex tachycardias is key to correct management.

Defibrillate (if Pulseless)

If a patient with wide complex tachycardia presents with no pulse, no blood pressure and no breathing (in cardiac arrest), a diagnosis of pulseless VT is clinched and immediate defibrillation must be done followed by chest compression and resuscitative measures, similar to the treatment of ventricular fibrillation.[5]

Document

In instances when a patient with wide complex tachycardia presents with pulse but in a hemodynamically unstable condition (hypotension,

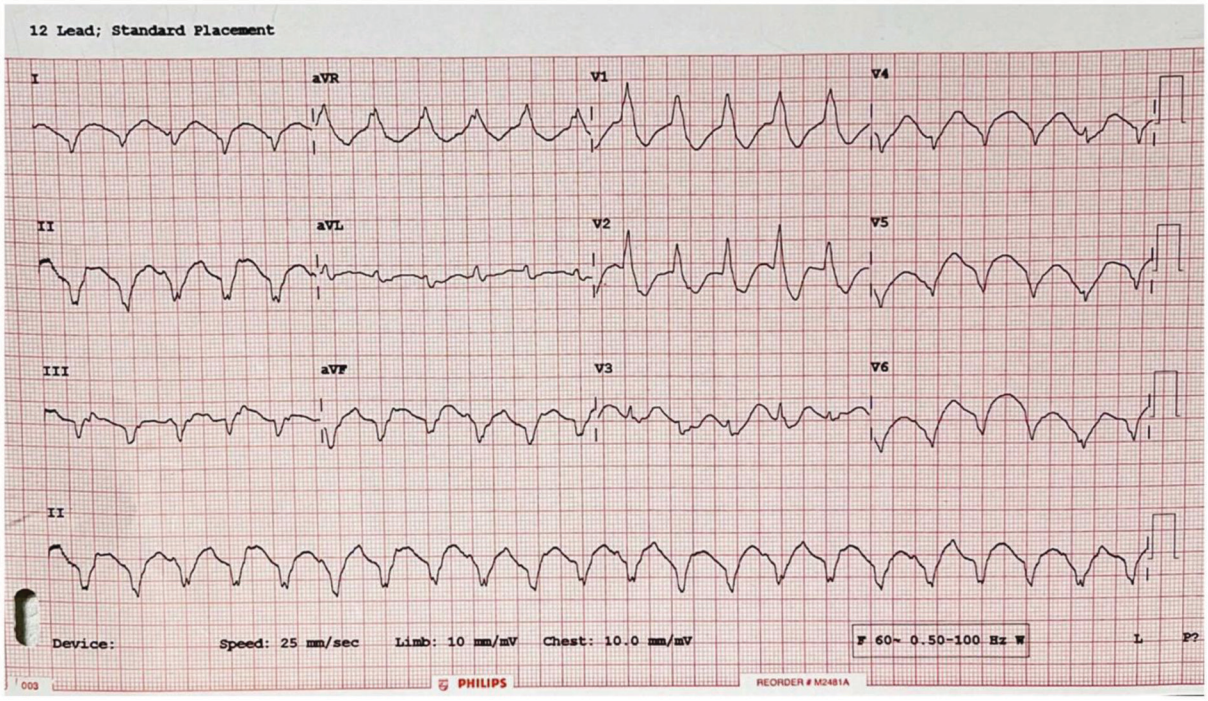

Figure 4. Full 12-lead ECG of the patient showing a wide complex tachycardia which is regular with a rate of 125 bpm.

altered sensorium, dizziness, syncope, signs of heart failure), VT is the most likely diagnosis and immediate intervention must be done in the form of synchronized cardioversion.[5] In other cases when the patient is hemodynamically stable, a full 12-lead ECG should be taken. It is advised and highly recommended that a full 12-lead ECG also be taken quickly for the first situation (unstable wide complex tachycardia) before intervening. A 12-lead tracing will give a clearer picture of arrhythmia and allows for more extensive analysis of the morphologic patterns in retrospect. It is also valuable for later planning for definitive interventions such as ablation. Hence, if the patient is stable, always document with a 12-lead ECG before intervening to convert the tachycardia. Figure 4 shows the full 12-lead ECG of the patient described.

Deal - With the Patient First, Not the ECG Tracing

In any patient presenting with tachycardia, whether wide complex or narrow complex, the physician must assess and manage the patient first. The management plan consists of the following:

1. Check the airway, breathing and circulation (pulse and blood pressure).
2. Give oxygen support if necessary.
3. Insert an intravenous line for medications if needed and for hydration.
4. Assess for the presence of symptoms and signs of hemodynamic instability.
5. Check for other possible secondary causes of tachycardia (blood loss, fever, anemia, infection, drugs, metabolic abnormalities, etc.) and manage accordingly.[5]

All of these are done by a healthcare team and involves multi-individual coordination and organization during this chaotic situation.

Decide

"If it looks like a duck, quacks like a duck, and acts like a duck, it must be a duck."

The medical maxim when it comes to wide complex tachycardia is that it must be interpreted based on information regarding the patient's clinical profile. Hence the best physician to interpret the ECG is the one who knows the patient and his/her clinical background. And in this context, based on a wealth of clinical experience, wide complex tachycardia occurring in a patient with ischemic heart disease, previous myocardial infarction or cardiomyopathy is most likely VT, unless proven otherwise. On the other hand, wide complex tachycardia occurring in a young patient with no structural heart disease is most likely supraventricular tachycardia with aberrant conduction, but there are exceptions to this rule.

Doubt

In case of any doubt as to the exact diagnosis, if the patient is unstable, and especially in an emergency situation, the default response is to treat as VT. Hence, synchronized cardioversion should be done. This intervention will be effective for both VT and supraventricular tachycardia with aberrancy.

Deliver Treatment

For unstable VT, synchronized cardioversion is recommended with a starting energy of 100 joules. This must be done under sedation. If this fails to convert the rhythm, then elevation of the energy must be done and cardioversion repeated, usually in this sequence: 150 joules, 200 joules, then maximum. [5]

For stable VT, initial treatment with intravenous amiodarone is the recommended strategy. A 150 mg bolus is given, with a second dose recommended after 10 minutes if unsuccessful. Alternative treatments include intravenous lidocaine or intravenous procainamide if available.[5]

If wide complex tachycardia is identified with certainty as supraventricular tachycardia with aberrant conduction, intravenous bolus of adenosine may be given which may convert an aberrantly conducting SVT to sinus rhythm.

In any case if stable VT does not respond to medical treatment and continues to persist, synchronized cardioversion should be done to convert arrhythmia to sinus rhythm.[5]

Dissect

After tachycardia has been effectively converted and patient stabilized, full analysis and scrutiny of the ECG can now be done. Indeed, in a real-world scenario, a thorough evaluation of the tracing is done in retrospect. Careful dissection of the ECG involves looking for clues that favor VT over aberrant conduction. The literature lists many algorithms and criteria based on morphologic patterns seen in the ECG. Knowledge of the physiologic basis and electrophysiologic mechanisms of these patterns is vital for full understanding and eventual recall of different criteria.

The criteria can be simply remembered and described by the first five letters of the alphabet: ABCDE.

A stands for Atrioventricular (AV) dissociation.
B stands for Beats – referring to capture beats and fusion beats.
C stands for Concordance – referring to ventricular concordance.
D stands for Duration of the QRS.
E stands for Etcetera – the other morphologic features.

It is imperative to take note that these criteria are not always present and specific for VT. At the end of the day, dissecting the ECG is all about looking for points that favor VT as the diagnosis. To understand the mechanisms of these criteria, it is always important to go back to the physiology behind these patterns.

AV Dissociation

AV dissociation is simply defined as having two different but simultaneous rhythms each from the atrium and ventricle occurring with no association with each other. The atria and ventricles are dissociated from each other, and their rhythms are occurring independently of each other, hence, AV dissociation. In this case, VT presents with tachycardia originating in the ventricle independent of any atrial rhythm, which is normally a slower sinus rhythm originating from the sinus node, the natural pacemaker of the heart. VT by virtue of its fast rate "usurps" control of the heart's rhythm but on the background, normal cardiac physiology still causes the sinus node to stimulate automatically and cause depolarization of the atrium at its usual rate of 60 to 100 bpm. This atrial activity, however, usually does not result in conduction to the ventricles since VT has taken over the driving rhythm of the heart and ventricles are essentially refractory to stimulation from any other source. On the surface ECG, AV dissociation is seen as occasional "bumps" or "lumps" in the intervals between the rapid wide QRS complexes. These represent the P-waves from the background atrial depolarization that is occurring (Figures 5 to 7).

AV dissociation is best seen in lead V1 and inferior leads II, III and aVF, where the P-waves are more evident.[6] It must be pointed out that all leads should be examined for AV dissociation as sometimes they are not evident in certain leads. While it is specific for VT, it is seen in only 50% of cases, hence its absence does not exclude VT.[2] Its presence, however, makes the diagnosis of VT

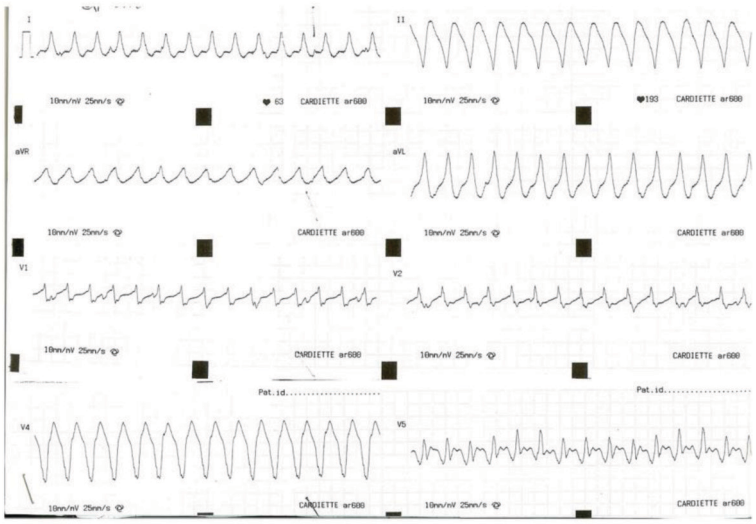

Figure 5. ECG tracing of wide complex tachycardia with a rate of 166 bpm and QRS duration of 0.16 seconds. There is evidence of AV dissociation as seen in the "bumps" and "lumps" in between the QRS complexes, evident in lead V1 and V2.

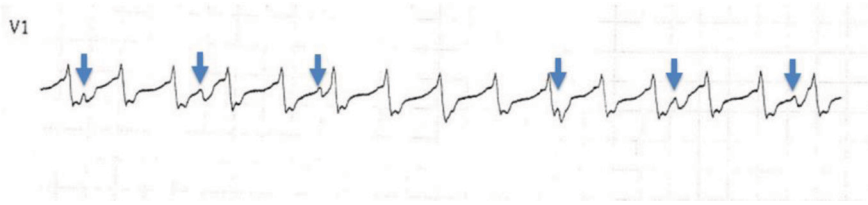

Figure 6. Enlarged segment from lead V1 in figure 5 showing AV dissociation as evident from the "bumps" pointed out by the arrows. These represent the dissociated P-waves.

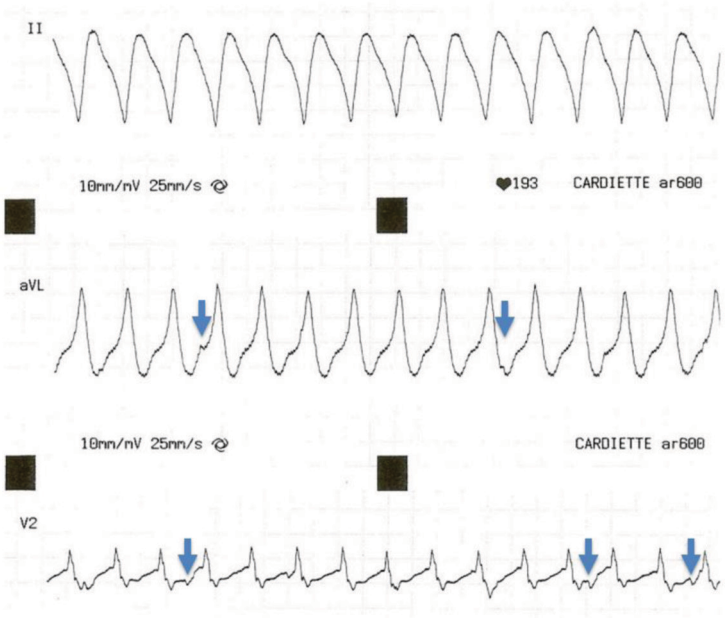

Figure 7. Enlarged tracing from leads II, aVL and V2 in figure 5 showing AV dissociation as evident from the "bumps" pointed out by the arrows more evident in leads aVL and V2 but not easily seen in lead II, hence the need to examine all leads.

likely. In very fast VT, AV dissociation may not be very evident since the intervals between the wide QRS complexes may be too short to appreciate the P-waves. In addition, in as much as 30% of VT, the ventricular impulses may conduct retrogradely through the AV node to the atrium leading to atrial depolarization, causing regular "ventriculo-atrial" association.[2]

Beats: Capture Beats and Fusion Beats

Following the same electrophysiologic mechanism as AV dissociation wherein the VT presents with wide complex tachycardia independent of the background slower sinus rhythm, the presence of sinus capture beats and fusion beats favor the diagnosis of VT.[6] Capture beats are beats caused when a background dissociated sinus beat at a right timing, is able to conduct to the ventricle fully, leading to a narrow QRS complex (similar to the QRS morphology of the baseline sinus rhythm of the patient) appearing in between the rapid wide QRS complexes of the VT (Figure 8 and 9).

Fusion beats occur when a background dissociated sinus beat at a right timing is able to conduct to the ventricle simultaneously with the ventricular depolarization caused by the VT, hence partially activating the ventricle, leading to a QRS complex that is "slightly wider" than the usual narrow

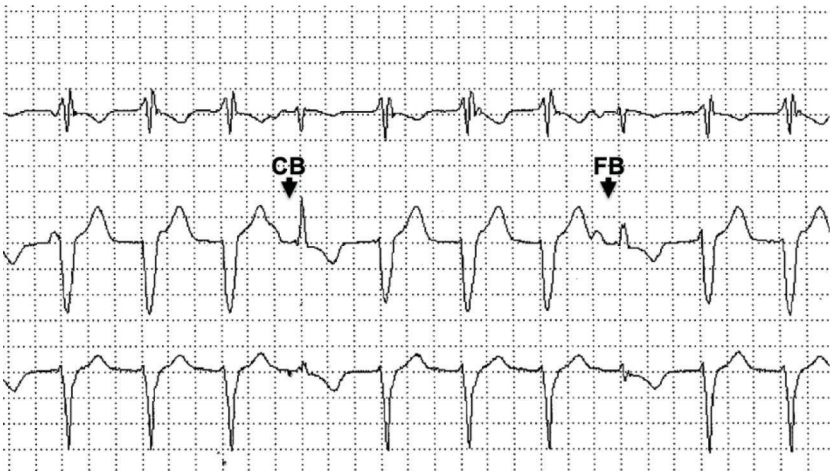

Figure 8. ECG tracing of a wide complex tachycardia showing a capture beat (*CB) and a fusion beat (*FB). These features are diagnostic of VT. Take note that the P-wave associated with sinus capture beat is slightly embedded in the previous T wave from VT, while the P-wave from the fusion beat is evident on the surface ECG. This points out that atrial depolarization associated with the capture beat came early enough to invade and depolarize the AV node and His-Purkinje conduction system and proceed with normal ventricular depolarization, while the atrial depolarization associated with fusion beat came slightly later, leading to simultaneous conduction to the ventricle with the wavefront of depolarization from the VT.

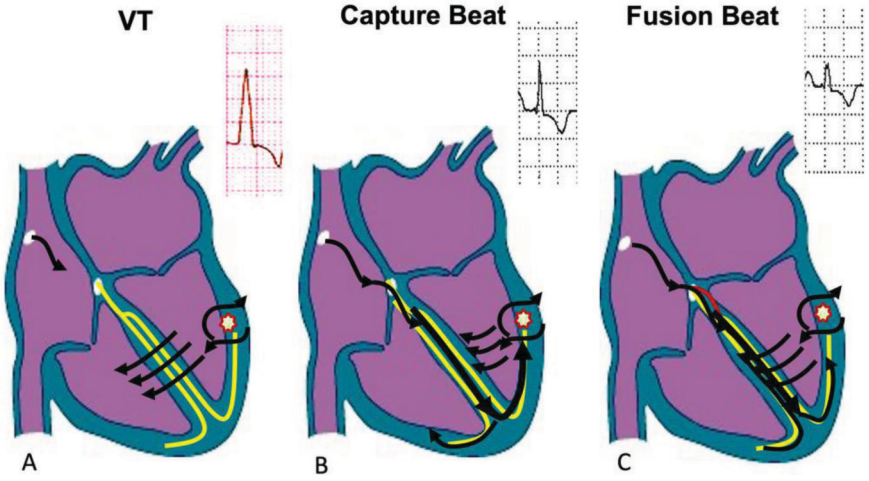

Figure 9. The physiology behind the pattern: A. During VT, the driving impulse emanates from the ventricular myocardium leading to depolarization of the ventricle of origin before depolarizing the His-Purkinje system and opposite ventricle by cell-to-cell depolarization, leading to a wide QRS complex. B. During a capture beat, the dissociated sinus impulse leads to atrial depolarization, and at a perfectly right timing, conducts down to the AV node and His-Purkinje system leading to normal ventricular depolarization and normal QRS complex similar to a sinus beat. C. In a fusion beat, the dissociated sinus impulse leads to atrial depolarization, then conducts down the AV node and His-Purkinje conduction system simultaneously with the wavefront of depolarization from the VT, leading to a QRS complex that is slightly wider compared to the sinus beat and slightly narrower than the VT beat.

sinus conducted beat and "slightly narrower" than the wide QRS complex from the VT, hence termed "fusion" beat, since it is a fusion between the sinus beat and wide QRS beat (Figure 8 and 9). Therefore, when a dissociated P-wave totally activates the ventricle serendipitously, it leads to a capture beat. If it partially activates the ventricle, it leads to a fusion beat. These findings, though highly specific for VT, are not frequently seen. Like AV dissociation, these features may not be evident in VT with very fast rate, but may only be seen with slightly "slower" VT rates. This is because a "slower" rate will at some point in time allow a dissociated sinus beat to conduct normally to the ventricle.[6]

Concordance

Ventricular QRS concordance refers to the presence of QRS complexes with all positive polarity or all negative polarity in the chest leads (V1-V6), described more completely as positive or negative concordance. To simplify, positive concordance means all the complexes from V1-V6 feature tall RS complexes while negative concordance means all the complexes exhibit a QS pattern (Figure 10). Its presence favors VT as the diagnosis of wide complex tachycardia. Negative concordance is more strongly suggestive of VT, while positive concordance may suggest VT, but is not totally specific.[6,7] Electrophysiologically, a QS complex in any lead means that the wavefront of depolarization is moving away from that site. Therefore, when a QS pattern is seen in the precordial lead, the wavefront of activation is originating from the anterior wall of the left ventricular site corresponding to that lead. Hence, when a wide complex tachycardia presents with QS pattern (negative concordance) in all the precordial leads, it means the impulses are all emanating away from the precordium, and specifically originating from the left ventricular apex.[6] On the other hand, positive concordance in the precordial leads may suggest that the VT originates from the posterior wall of the left ventricle and wavefront of activation moves toward the anterior wall, hence manifesting as positive R waves in the chest leads. However, it should also be noted that while this pattern is suggestive of VT, in some instances it may also be caused by supraventricular tachycardia conducting through an accessory pathway.[6]

Duration of the QRS

The duration of QRS complex of a wide complex tachycardia may provide more clues suggesting a VT. As a general rule, the wider the duration, the more likely it is VT. For VT presenting with a right bundle branch block (RBBB) pattern, a QRS duration >0.14 seconds favors VT; while for a VT with a left bundle branch block (LBBB) pattern, a duration >0.16 seconds favors VT.[6,7]

Physiologically, the farther the origin of arrhythmia from the interventricular septum where the Bundle of His and the conducting system is located, the wider the QRS duration, hence favoring VT. The width of QRS during VT is also related to other factors: a wider QRS suggests a more diseased ventricle such as in cardiomyopathies and post-infarct patients, therefore favoring VT as the cause. However, exceptions to this feature include VT that may originate close to the septum, which may present with a slightly

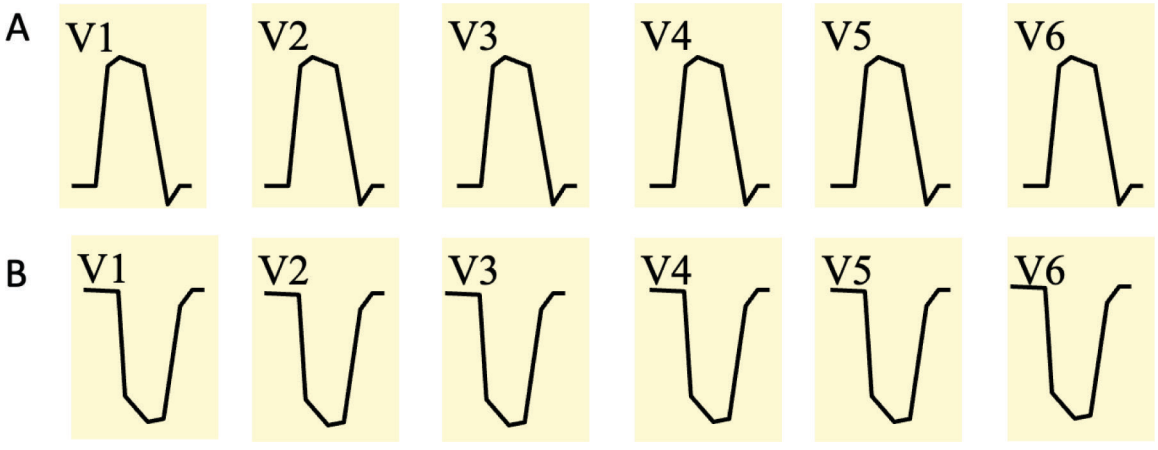

Figure 10. A. Positive ventricular concordance means positive polarity of all QRS complexes in the chest leads. B. Negative ventricular concordance means negative polarity or QS patterns in all complexes in the chest leads. This is more specific for VT.

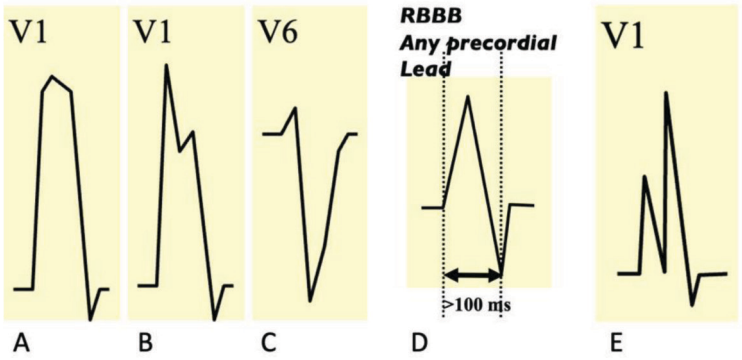

Figure 11. Morphologic patterns of wide QRS tachycardia with a RBBB configuration. A to D favor VT while E favors SVT with aberrancy. A. Monophasic positive R wave in V1. B. rSr' pattern in V1 with a taller left "rabbit ear". C. S deeper than R wave in V6. D. R to S interval of >100 ms in any precordial lead. E. rSr' or typical RBBB pattern more suggestive of SVT with aberrancy.

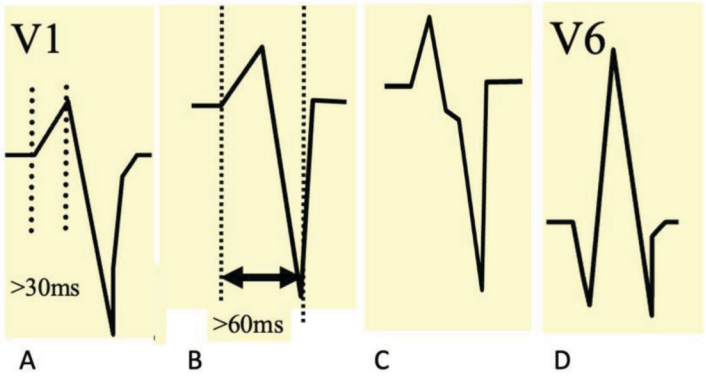

Figure 12. Morphologic patterns of wide QRS tachycardia with a LBBB configuration. All these patterns favor VT. A. R wave duration in V1 >30 ms. B. R to S interval of more than 60 ms. C. Notch in the descending part of the S wave. D. Q wave in lead V6.

narrower QRS duration than usual VTs. Furthermore, intake of antiarrhythmic medications such as Class I antiarrhythmic agents can cause SVT to present with a very wide QRS duration mimicking VT.[6]

Etcetera

Certain ECG features favor VT as the cause of wide complex tachycardia. These miscellaneous features are grouped into the following: (1) R to S interval, (2) morphologic patterns, (3) axis, (4) lead aVR morphology, (5) clues from the baseline sinus rhythm ECG.

1. R to S interval:

   Based on the criteria of Brugada, et al., an R to S interval of >100 ms in any precordial lead favors VT. Specifically, if the VT has an RBBB pattern, RS interval >100 ms suggests VT; while for VT with a LBBB pattern, RS interval >60 ms in lead V1 or V2 suggests VT (Figure 11).[6,7]
2. Morphologic patterns:

   If a wide complex tachycardia presents with a RBBB pattern (predominantly positive polarity of the QRS complexes in leads V1 and V2), the following morphologic features are more in favor of VT:
   a. Monophasic R in V1 or an rSr' in V1 (rabbit ear configuration with a taller left ear than the right) [Figure 11 and 13][6,8]
   b. rS complex (S amplitude larger than the R wave) in V6[6,8]
   c. R to S interval in precordial lead >100 ms[6,8]

   If wide complex tachycardia presents with a LBBB pattern (predominantly negative polarity of QRS in leads V1 and V2), the following morphologic features are more in favor of VT:
   a. R wave in V1 or V2 >30 msec[6,8]
   b. R to S interval >60 msec in V1 or V2[6,8]
   c. Notching on the downstroke of the S wave in V1 or V2[6,8]
   d. Any Q-wave in V6 [Figure 12 and 13][6,8]

   Therefore, a typical RBBB or LBBB morphology or pattern of the wide complex tachycardia suggests

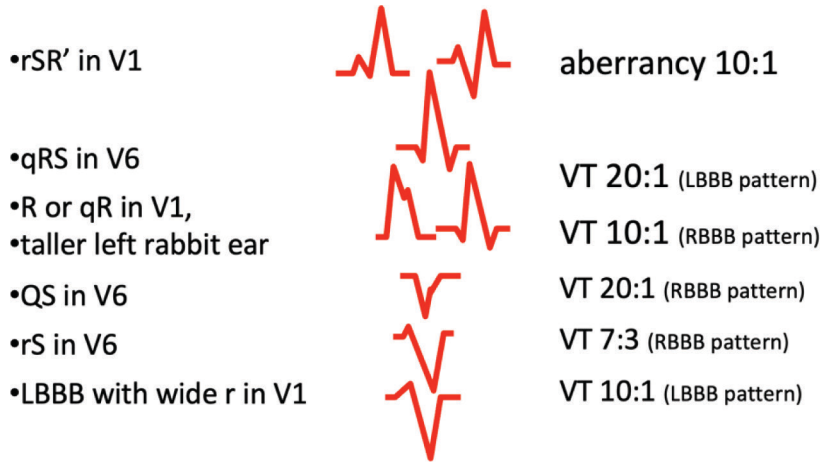

Figure 13. Morphologic patterns of wide QRS tachycardia which favor VT or aberrancy with corresponding probability. Figures derived from Friedman HH. Diagnostic electrocardiography and vectorcardiography. 3rd ed. Singapore: McGraw-Hill; 1986.

SVT with aberrancy, while an atypical RBBB or LBBB morphology or pattern favors VT.

3. Axis

   As a general rule, a normal QRS axis or an axis that is the same as baseline sinus rhythm axis if it is available, favors SVT with aberrancy. The presence of a left or right axis during wide complex tachycardia favors VT. The presence of an extreme right superior axis (northwest axis) strongly favors VT as the diagnosis.[6]

4. Lead aVR morphology

   The presence of a positive initial R wave in lead aVR during wide complex tachycardia has 98%-99% accuracy for VT, according to one study.[9] Specifically, such a pattern suggests VT arising from the inferior or apical region of the ventricle.[6] The physiologic basis for this is that in SVT with aberrancy, the main direction of ventricular depolarization proceeds away from lead aVR, hence leading to a negative QRS complex, except in the presence of an inferior wall infarct.

5. Clues from the baseline sinus rhythm ECG

   If the patient has a previous baseline ECG during sinus rhythm available, certain features may suggest whether the wide complex tachycardia is VT or not (Figure 14). The presence of preexcitation pattern consistent with Wolff-Parkinson-White syndrome (short PR interval, delta wave and wide QRS) suggests either an antidromic SVT if regular or a preexcited atrial fibrillation if irregular. The presence of an old myocardial infarct (Q wave) suggests the presence of an ischemic myocardial scar and highly points to VT as the cause. Abnormal chamber enlargement, specifically left ventricular hypertrophy with features suggestive of hypertrophic cardiomyopathy (large QRS complexes consistent in chest leads with deep T wave inversion) suggests VT. ECG features that may be consistent with dilated cardiomyopathy or severe myocardial disease (left ventricular hypertrophy with low voltage complexes in the limb leads) suggests VT. The presence of bundle branch block (RBBB or LBBB) or intraventricular conduction delay at baseline may suggest SVT as the cause, but wide complex tachycardia morphology must be compared with baseline to see if they are the same QRS patterns. On the other hand, a baseline LBBB or intraventricular conduction delay during sinus rhythm may be seen in myocardial diseases such as ischemic and dilated cardiomyopathy with low ejection fraction putting them at high risk for VT. A wide complex tachycardia occurring in this setting highly suggests VT, especially if the QRS morphology during tachycardia is different from baseline sinus patterns. In rare instances, an epsilon wave (notch in the ascending limb of the S wave in lead V1) seen in patients with arrhythmogenic right ventricular cardiomyopathy during sinus rhythm, highly suggests VT as the cause of wide complex tachycardia. The presence of fragmented QRS complexes in the limb leads and chest leads are suggestive of the presence of myocardial scars seen in post-infarct and ischemic heart disease patients, hence suggesting VT as the cause of wide complex tachycardia. When a baseline sinus rhythm tracing has evidence of occasional

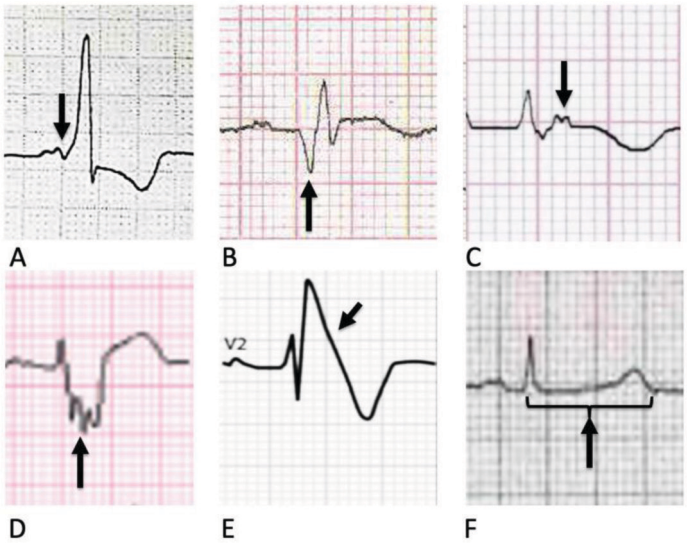

Figure 14. ECG clues from the baseline sinus rhythm tracing which favor VT or SVT with aberrancy as the cause of wide complex tachycardia. A. Preexcitation pattern or Wolff-Parkinson-White syndrome characterized by short PR interval, delta wave (arrow) and wide QRS complex. This condition may present with preexcited tachycardia or antidromic SVT leading to wide complex tachycardia. B. Presence of significant Q waves (arrow) signifies old infarct and ischemic heart disease, favoring VT as the cause. C. Epsilon wave, a notch seen in the terminal S wave (arrow) in lead V1 or V2, may sometimes be present in arrhythmogenic right ventricular cardiomyopathy and may favor VT. D. Fragmented QRS complexes (arrow) in the limb leads and chest leads may suggest the presence of myocardial scar and favor VT. E. Type 1 Brugada pattern showing the coved type ST elevation in leads V1 and V2 (arrow) is an ion channelopathy, which may place the patient at risk for polymorphic VT or VF. F. Prolonged QT interval may suggest long QT syndrome which may lead to polymorphic VT.

Table 1. The duck strategy for wide complex tachycardia

Definition	Wide QRS Complex Tachycardia: Rate >100 bpm QRS duration >0.12 secs (120 ms)
Differential Diagnosis	Sinus tachycardia with aberrant conduction (BBB) SVT with aberrant conduction (BBB) Pre-excited tachycardia in Wolff-Parkinson-White syndrome (sinus tachycardia with preexcitation, or atrial fibrillation with preexcitation) Antidromic SVT Pacemaker-mediated tachycardia VT
Dictum	Unless proven otherwise, treat as VT, especially in an emergency situation
Decisions on Management	Pulseless VT: Treat as ventricular fibrillation (cardiac arrest) Defibrillation followed by chest compression and resuscitation measures VT with pulse: Check BP Hypotension, altered sensorium → Unstable VT: Synchronized cardioversion under sedation; start with 100 joules Normal BP, normal sensorium → Stable VT: Give amiodarone 150 mg IV. May repeat after 10 minutes if persistent. Persistent stable VT → Synchronized cardioversion
Dissection of the ECG Tracing	ECG Signs in Wide QRS Tachycardia that favor VT: ABCDE A – AV Dissociation B – Beats: Capture Beats and Fusion Beats C – Concordance D – Duration of QRS E – Etcetera (morphologic QRS patterns)

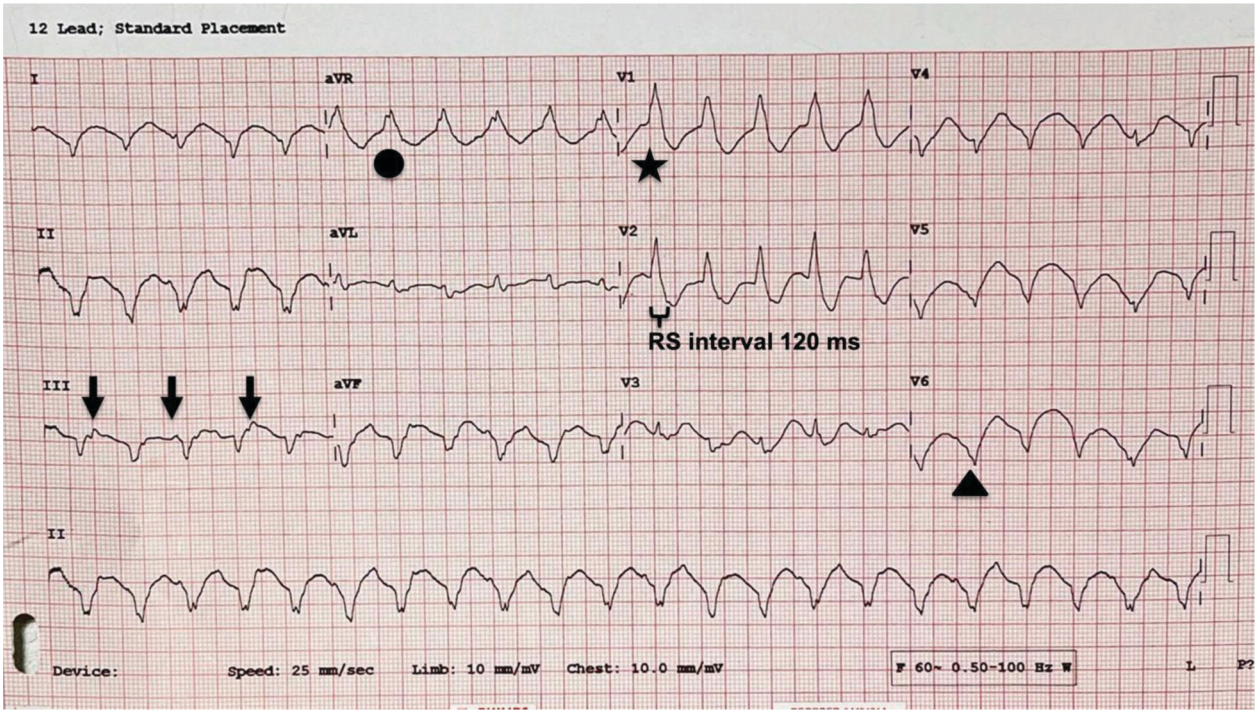

Figure 15. Applying the duck strategy and ABCDE criteria to the ECG tracing, this wide complex tachycardia has evidence of AV dissociation as seen by the dissociated P-waves (bumps and lumps pointed by the arrows in lead III). The QRS duration is approximately 140 ms. The QRS has an RBBB pattern with a monophasic positive R in lead V1 (star), consistent with an atypical RBBB pattern. The RS interval is 120 ms (measured in lead V2). The complexes in V6 have a QS pattern (triangle). The axis is extreme right or "northwest" axis with negative polarity in leads I and aVF. Finally, lead aVR shows positive R wave (black dot). All these features favor VT as the cause of wide complex tachycardia.

premature ventricular complexes (PVCs), the wide complex tachycardia would be most likely VT if the morphology of the QRS complexes of tachycardia and the PVC are similar. Finally, a Brugada ECG pattern (coved ST elevation in V1 and V2), a prolonged corrected QT interval and a markedly short QT interval during sinus rhythm, all point to the presence of ion channelopathies which put the patient at risk for ventricular arrhythmias, although these usually present as polymorphic VT or ventricular fibrillation (sudden death) (Figure 14).[6,7]

It must be pointed out that all these features are just suggestive clues and are not rules. Finally, one has to bear in mind that it may sometimes be impossible to differentiate VT from SVT with aberrancy by ECG. But knowledge and recognition of these features can provide a scientific basis for favoring one diagnosis over the other.

Diagnose

Applying all these concepts now to the presenting patient with the 12-lead ECG tracing shown (Figure 15), we can come up with more scientific basis pointing to VT as the cause of tachycardia based on clues in the tracing. There is evidence of AV dissociation. There is, however, no evidence of capture or fusion beats. Ventricular concordance is not seen. The QRS duration is wide (140 ms), the RS interval is more than 100 ms with an atypical RBBB pattern, northwest axis and morphologic features in favor of VT. All of these are done in retrospect after the patient has been managed and stabilized.

Dictum

At the end of the day, just remember, whenever in doubt, in the face of wide complex tachycardia, treat as VT, unless proven otherwise.

Doctor: Teachable Insights in Medicine

Ultimately, be a doctor. Treat the patient, not the ECG tracing. This holds true for any case in medicine. When faced with a dilemma between what is seen on a certain ancillary test with what is observed in the patient, always go back to the patient. In wide complex tachycardia, the clinical and hemodynamic status of the patient takes precedence over doing

a meticulous discussion of the diagnosis. But while doing so, the clinician acts like a duck, appearing calm in applying the therapeutic algorithms while trying to figure out the exact etiology at the back of his mind.

Ducks are more than just their quacks and feathers. Being calm, cool, resilient and confident are just some of their positive attributes that clinicians can adopt in the face of difficulty. Being able to handle multiple tasks and difficult situations and adapt to adversity is the key to successfully managing challenging cases.

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