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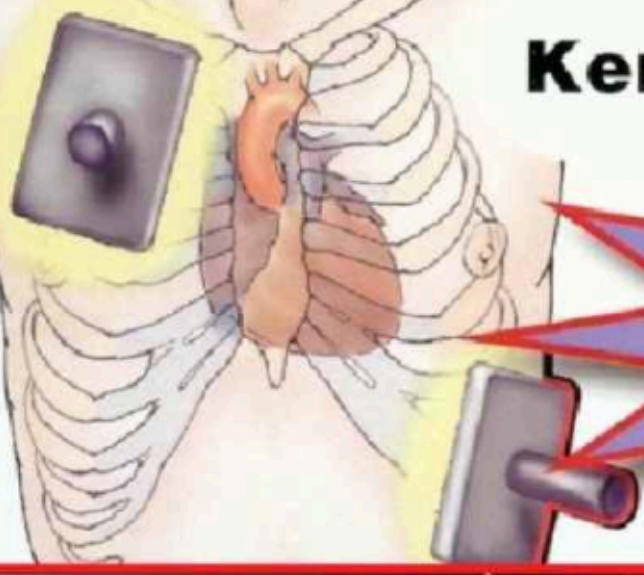


5th Edition (2013)

Completely Revised & Updated
— Expanded Content —

- CONTENTS -

WCTs of Uncertain Etiology

08.1 – *We See Tachycardia …*

You are called to a code. You see ***Tachycardia*** on the monitor (*Figure 08.1-1*). The patient is *hemodynamically* stable. *What to do next?*

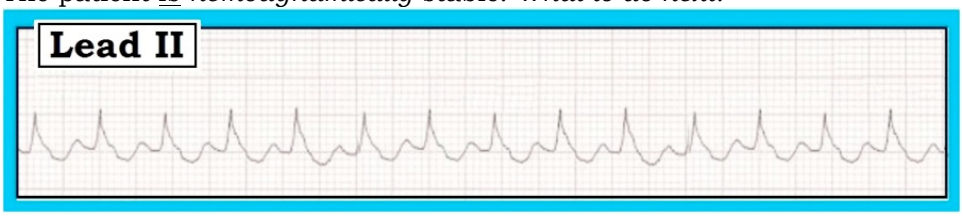


Figure 08.1-1: You are called to a code. We see ***tachycardia*** – although at the moment the patient is *hemodynamically* stable.

08.2 – Assessing Tachycardia: *Initial Thoughts*

Since the patient in Figure 08.1-1 is stable — there is *at least* a moment of time to proceed with further evaluation:

- Optimal management will depend on determining the ***specific* type** of **tachycardia**. The following steps in assessment (*beginning in Section 08.3*) should be accomplished *within* seconds!
- Some of these steps may overlap.
- *Definitive* rhythm diagnosis may *not* be possible at this time. That said — *as long as* the patient *remains* stable, we should be able to ***narrow down* diagnostic possibilities** and initiate appropriate treatment.
- NOTE: IF at *any* time the **patient *becomes* unstable** — then *immediately* cardiovert or defibrillate.

How then to proceed *diagnostically* IF your patient is *hemodynamically* stable and in the rhythm shown in Figure 08.1-1?

08.3 – Step #1: *Is the QRS Wide or Narrow?*

Step #1: *Is the QRS Wide or Narrow?*

IF the QRS complex of a tachycardia is **narrow** (ie, *not more than 0.10 sec. in any lead*) — then the rhythm is *almost* certain to be ***supraventricular***.

- **SVTs (***SupraVentricular Tachycardia***)** — are *rarely* life-threatening. Treatment is *different* than for WCTs. This is why **ACLS-PM** *highlights* assessment of **QRS width** as an early *KEY* step for determining treatment!
- ***Sometimes* it will be obvious** that the QRS is wide! This is *not* the case for Figure 08.1-1 (*We think the QRS in Figure 08.1-1 is wide — but are not 100% certain from the single lead II that is shown here*).
- Given the importance of distinguishing between SVT vs VT — obtaining a **12-lead ECG *during* tachycardia** is an *invaluable* early step in management of the stable tachycardia patient when *uncertain* about etiology.

- **Caveat:** Part of the QRS may lie on the baseline. When this happens, the QRS may *look* narrow in one lead — but be *very* wide in *other* leads (*Get a* **12-lead!**).

08.4 – HOW to Define: *Is the QRS Complex Wide?*

Although ACLS-PM defines *"wide"* as ≥0.12 second — We favor using ***more*** than ***half*** a ***large*** **box** (*≥0.11 sec.*) as our definition. The QRS of some VTs may only be 0.11 sec. in duration (*fascicular or outflow track VTs*).

- Practically speaking — it is often much *easier* to tell *at a glance* IF the QRS is *more* than half a large box or not — than to determine if the QRS is ≥0.12 second (*See Figure 02.6-1 in Section 02.6*).
- Using ≥0.11 sec. to define "wide" will *not* overlook VT (*whereas requiring 0.12 second will miss some cases of outflow track or fascicular VT — esp. if part of the QRS lies on the baseline in the lead[s] being monitored*).

08.5 – Step #1A: *IF Uncertain – Get a 12-Lead!*

Step #1A: IF uncertain – *Get a 12-Lead!*

There are *several* ways a **12-lead ECG** obtained ***during*** **tachycardia** may be helpful. These include:

- Determining *for sure* IF the QRS is wide or not (*Look in all 12 leads; Measure the widest QRS you see*). Remember – there may be distortion of QRS duration when using a portable ECG monitor/defibrillator (*due to time compression*) – so the only way to *truly* determine QRS width is by obtaining a 12-lead ECG *during* tachycardia.
- Assessing axis and QRS morphology *during* tachycardia.
- Looking in *all* 12 leads for signs of atrial activity.
- Getting a baseline *during* tachycardia may prove invaluable in management *after* conversion to sinus rhythm. The true etiology of the WCT will sometimes only be elucidated by *retrospective* comparison between the 12-lead ECG obtained *during* and *after* tachycardia.

Summary Point: IF the **QRS** on 12-lead is **narrow** (*not more than half a large box*) – then the rhythm is an **SVT** (*Section 13.0*).

- But **IF** the **QRS** is ***Wide*** — then by definition, you have a **WCT (***Wide-Complex Tachycardia***)**. Go to STEP 2 (*Section 08.6*):

08.6 – Steps #2, 2A: *Is the WCT Regular? – Monomorphic?*

Step #2: *Is the WCT a Regular Rhythm?*

Step #2A: *Is it a Monomorphic WCT?*

Practically speaking — We assess **Steps 2** and **2A** together. Our reasons for doing so are the following:

- ***Polymorphic*** **WCT** (*in which QRS morphology during tachycardia changes*) — will often require defibrillation for conversion. These rhythms are typically irregular and include *polymorphic* VT and Torsades de Pointes (*Section 11.0*).
- ***Monomorphic*** **WCT** (*in which the QRS during tachycardia stays the same*) — is often easier to treat. IF ***no*** **P waves** are seen and the rhythm is ***irregularly*** **irregular** — *Consider* AFib.

- Keep in mind that *some* VT rhythms may be *slightly* irregular (*though not nearly as irregular as AFib!*).
- But **IF** the rhythm is a **regular** (*or almost regular*) ***monomorphic*** **WCT** *without* clear evidence of normal sinus P waves – then the differential is as in **LIST #1** (*Table 08.7-1*).

08.7 – LIST #1: *Causes of a Regular WCT of Uncertain Etiology*

The common causes of a regular *monomorphic* WCT rhythm *without* clear sign of normal P wave activity are noted in LIST #1 (*Table 08.7-1*). We emphasize the following points about List #1:

- The reason we put **VT** as the first 8 entities in LIST #1 is twofold: **i)** It is *by far* **the *most* common cause** of a regular (*or almost regular*) WCT in adults when sinus P waves are lacking; and **ii)** It is **the most *serious* cause!**
- The ***likelihood* that** a **WCT** is **VT** goes up even more (*to at least 90%*) — IF the patient in question is ***older*** and has *underlying* **heart disease** (*prior MI, cardiomyopathy, angina, heart failure*). This is true *regardless* of whether the patient is alert — and *regardless* of what the BP might be *during* the tachycardia (*VT may be present even if systolic BP exceeds 180 mmHg!*).
- Availability of a ***prior* 12-lead ECG** on the patient during sinus rhythm may be invaluable for assessing the possibility of ***preexisting* BBB**.
- For *aberrant* conduction considerations — *See Section 19.0*.

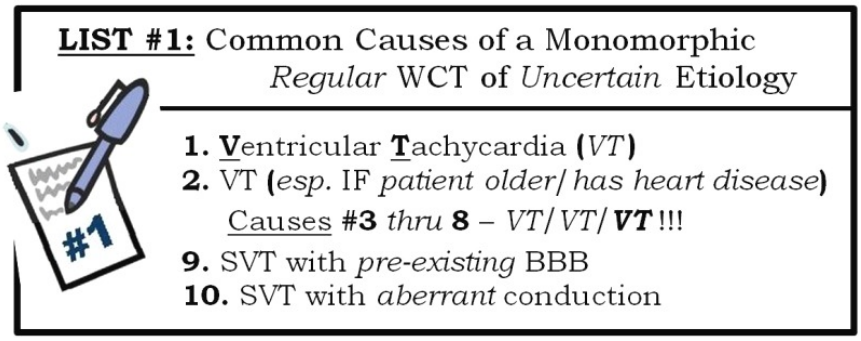
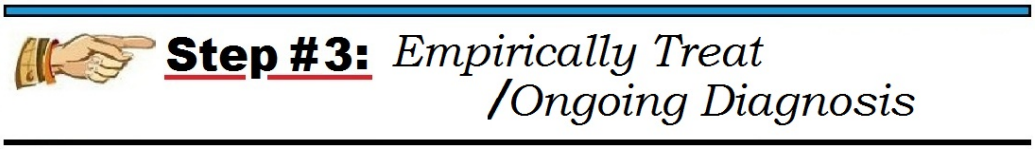


Table 08.7-1: Causes of a *Regular* WCT of *Uncertain* Etiology. Presume VT unless *proven* otherwise.

08.8 – Step #3: Empirically Treat/ Ongoing Diagnosis



Optimal management of WCT rhythms depends on the type of WCT. You will *not* always know definitive diagnosis at the time you need to begin treatment:

- **Steps #1** and **#2** should *eliminate* most SVTs and *polymorphic* VT from consideration (*Sections 08.5 and 08.6*).
- You are left with a **regular** (*or almost regular*) ***monomorphic*** **WCT** of ***Uncertain* Etiology** (*LIST #1*). *Presume* VT. Treat accordingly *until* proven otherwise.
- KEY Point: IF at *any* time the **patient *becomes* unstable** — then *immediately* cardiovert or defibrillate.

08.9 – *Unspecified* WCT: *Suggested Initial Approach*

Suggested Approach:

Until such time that the clues discussed below in *Beyond-the-Textbook* suggest a different diagnosis (*Section 08.14*) – it is most prudent to ***presume* VT** as the etiology of the WCT – and to ***treat* accordingly**. The "short answer" for our suggested *initial* treatment approach to *unspecified* WCT appears below (*Sections 08.9 thru 08.13*). More on the subject is found in Section 07.3 (*that reviews initial treatment of known monomorphic VT*).

First Fix the ***"Fixables"*** — Better than antiarrhythmic drugs is to *find* and *"fix"* any potential *precipitating* causes of VT as soon as you can. These may include:

- Electrolyte disturbance (*esp. low Mg++ or K+*).
- Acidosis/Hypoglycemia.
- Hypoxemia.
- Shock (*from hypovolemia; blood loss; sepsis, etc.*).
- Uncontrolled ischemia/acute infarction.
- Acute heart failure.
- Dig toxicity/Drug overdose.

08.10 – Use of ADENOSINE for *WCT/Presumed* VT

Adenosine is usually well tolerated – and should be considered as the 1st drug that might be given.

- Adenosine will convert (*or at least slow down*) most *regular* SVT rhythms.
- It may convert 5-10% of VT rhythms.
- Begin with **6mg** by **IV push**. IF no response in 1-2 minutes — Give **12mg** by IV push (*Section 06.2*).
- Side effects from Adenosine are usually short-lived (*due to the drug's ultra-short half-life — Section 06.3*).
- Do *not* use Adenosine for *polymorphic* VT or Torsades.
- Conversion of a WCT to sinus rhythm with use of Adenosine does *not* prove a supraventricular etiology!
- NOTE: We do *not* always start with Adenosine in all older patients with *ischemic* VT from *known* coronary disease (*as the drug is unlikely to convert ischemic-etiology VT*).

08.11 – AMIODARONE for *WCT/Presumed* VT

Amiodarone is our preferred ***initial* agent** of choice (*after Adenosine*) for *stable* sustained *unspecified* WCT:

- Give **150 mg IV** over 10 minutes. May repeat. IF the drug works — Consider maintenance **IV *infusion*** at **1 mg/ minute** (*See also Section 07.14*).
- Amiodarone may also treat *some* forms of SVT.

08.12 – PROCAINAMIDE for *WCT/Presumed* VT

Procainamide is *also* recommended by ACLS-PM (*Provider Manual*) as a 1st-line drug of choice for *unspecified* WCT:

- The efficacy of Procainamide appears comparable to Amiodarone for VT and SVT rhythms.

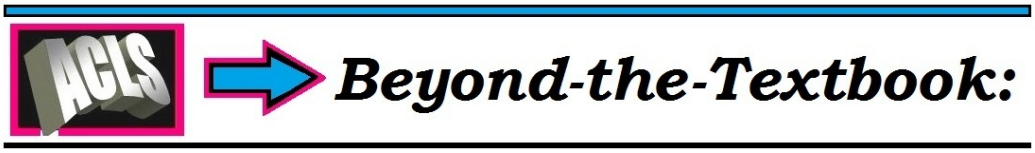
- Give **20-50 mg/minute IV** until either: **i)** the arrhythmia is suppressed; **ii)** hypotension ensues; **iii)** QRS duration increases >50%; or **iv)** a max dose = 17mg/kg has been given (*usual IV loading ~500-1,000 mg*).
- May follow with **IV *maintenance* infusion** at **2mg/minute** (*1-4 mg/min range*).
- *Potential* Drawbacks of Procainamide include: **i)** QT prolongation; **ii)** inadvisability with heart failure; **iii)** less clinician familiarity and a more complicated administration protocol; and **iv)** greater tendency to develop hypotension, especially if *faster* infusion rates are used (*Our preference is to start with increments of 100 mg IV over ~5 minutes = ~20 mg/minute*).

08.13 – *Synchronized* CARDIOVERSION for *WCT/Presumed* VT

There may well come a point during the above treatment process when ***"it becomes time"*** to get the patient out of the *unspecified* WCT rhythm:

- At that point — ***Cardiovert!*** (*Section 05.0*).

08.14 – Diagnosing the *Regular* WCT: *Beyond-the-Textbook*



Given that *optimal* management of **WCT rhythms** depends on ***specific* diagnosis** of the ***type*** of WCT — We conclude this section with insights for determining IF a **regular** (*or almost* regular) ***monomorphic* WCT** is likely to be VT (*vs SVT with aberrant conduction* or *preexisting BBB*). We emphasize the following points:

- A WCT rhythm is "guilty" (ie, *presumed VT*) until *proven* otherwise.
- As long as the patient *remains* stable — there is *little to lose* by *brief* attempt at refining your rhythm diagnosis. ***Remain ready to cardiovert*** at *any* time IF the patient begins to decompensate.
- IF unable to cardiovert – then *immediately* defibrillate.

NOTE: No set of rules is "perfect" for interpreting WCT rhythms. Even the experts are *not* always certain. Our goal is merely to increase your odds of correct diagnosis by a *time-efficient* and *easy-to-remember* approach using those criteria we have found most helpful.

- We devote Section 09.0 to a series of ***Practice* ECGs** that apply these principles.

08.15 – WCT Diagnosis: *Benefit of Statistics/Clinical Parameters*

One often forgets to recruit the wisdom inherent in the following statement: *Common things are common*. **Statistically** – VT is *by far* the most common cause of a *regular* WCT rhythm when sinus P waves are not clearly evident (*LIST #1 = Table 08.7-1*). Studies have shown that *at least* 80% of such *regular* WCT rhythms are VT.

- Is the patient *older* than 50-60 years old? Is there a history of *heart* disease? IF Yes — ***think* VT**! Statistical odds that a *regular* WCT without sinus P waves is VT attain *at least* 90% – IF the patient is older than ~50 years old *and* has underlying heart disease.
- Is there a *prior* history of VT? — *Telemetry* tracings showing PVCs or short VT runs? IF Yes — ***think* VT**!
- OR – Is the patient a *20-to-40* year old adult with *no* history of underlying heart disease who presents in a WCT *precipitated by* exercise or stress? IF

Yes — even if the WCT is VT, it is relatively *likely* to be an ***adenosine-responsive* form** of **VT** that is often *well* tolerated (*Section 06.5*).

Remember: — Even IF the patient is *asymptomatic* with BP>180 systolic for a *prolonged* period — this *in no way* rules out VT. *It simply means you have some time*.

- *Additional* steps in the diagnostic assessment of a *regular* WCT rhythm *require* a **12-lead ECG** obtained ***during*** the **WCT**.

08.16 – WCT Diagnosis: *Prior 12-Lead ECG During Sinus Rhythm?*

Some patients have *baseline* conduction defects (*baseline BBB; IVCD*). Availability of a 12-lead while the patient was in sinus rhythm allows ***lead-to-lead* comparison** *prior* and *during* the WCT to see if QRS morphology is the same.

- IF QRS morphology is *not* the same — ***think* VT**!
- Realistically — It will *not* be often that a prior ECG during sinus rhythm will be available (*or you may not have time to look at it with a WCT patient in front of you*).

08.17 – WCT Diagnosis: *Extreme* AXIS? (*Simple Rule #1*)

We favor beginning our use of the 12-lead ECG obtained *during* the WCT rhythm with attention to **3 *Simple* Rules**. The 1st of these Rules relates to assessment of ***frontal* plane axis** *during* the WCT rhythm.

- The frontal plane axis may be approximated *at a glance* – simply from inspection (*and comparison*) of the net QRS deflection in leads I and aVF. **Lead I** is the *horizontal* lead – it is situated at zero degrees. **Lead aVF** is the *vertical* lead – it is situated at +90 degrees. If the *net* QRS deflection is positive in *both* leads I and aVF – then the mean QRS axis is normal (ie, *between zero and +90 degrees*).
- While details of axis calculation extend beyond the scope of this Section on ventricular tachycardia – the ***"take-home"* message** is that the presence of ***extreme* axis deviation** *during* a WCT rhythm is ***virtually* diagnostic** of **VT**.
- *Extreme* axis deviation is *easy* to recognize. The QRS complex will be *entirely* negative in *either* lead I *or* lead aVF. This is the case for *both* **X** *and* **Y** in Figure 08.17-1. Awareness of this axis criterion *immediately* tells us that X and Y are *almost* certainly VT.
- **KEY Point:** The presence of mild or even *moderate* LAD or RAD (*Left or Right Axis Deviation*) does *not* assist in distinguishing between VT vs SVT. This is the case for **Z** in Figure 08.17-1 in which lead I is clearly positive, but lead aVF is not. Instead, we see a *slender* positive R wave in Z – and a *wider* S wave. Whether some degree of left axis deviation is present in Z *(surface area of the negative S wave appears greater than surface area within the slender positive R wave*) – is not only uncertain, but unimportant. What counts is that **extreme axis deviation** is ***not* present** in **Z** (*because the net QRS deflection in lead aVF is not all negative*). This tells us *at a glance* that use of the axis criterion is *not* helpful in distinguishing between VT vs SVT for the rhythm in Z.

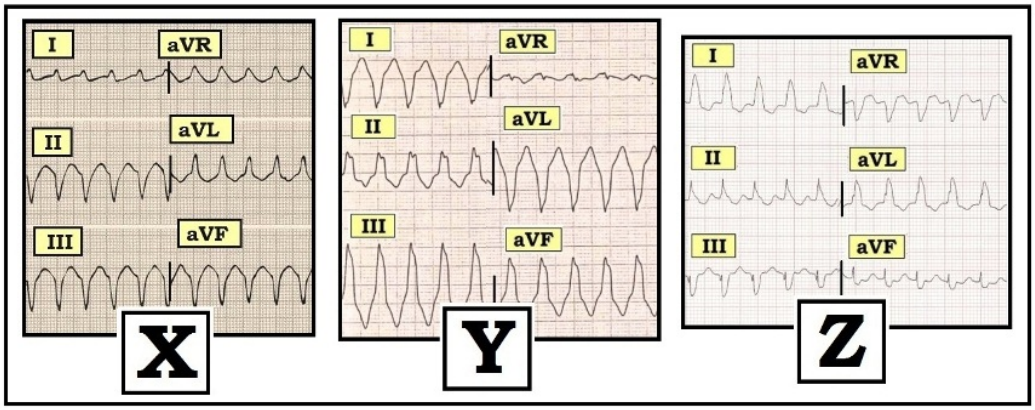


Figure 08.17-1: Use of **Axis** for WCT diagnosis. **Rhythm X** – shows *extreme* left axis (*QRS all negative in lead aVF*). This is VT. **Rhythm Y** – shows *extreme* right axis (*QRS all negative in lead I*). This is VT. **Rhythm Z** – clearly does *not* manifest extreme axis deviation, because the QRS complex in lead aVF is not all negative. Calculation of axis is of *no help* for distinguishing between VT vs SVT for Z.

BOTTOM Line: We *LOVE* this **axis criterion *during* tachycardia**. When used as intended you'll find:

- ***Calculation*** of **axis** *during* WCT rhythms using **leads I** and **aVF** is easy. IF the QRS is ***all*** **negative** in either lead – then diagnosis of **VT** is ***almost*** **certain** (*X and Y in Figure 08.17-1*).
- Remember that anything other than *extreme* axis deviation is of *no use* in distinguishing between VT vs SVT.

08.18 – WCT Diagnosis: LEAD V6 (*Simple Rule #2*)

We have found lead V6 to be the most helpful lead to look at *during* a *regular* WCT rhythm. We ask: **Is Lead V6 *all*** (*or almost all*) **Negative?**

- When the etiology of the rhythm is supraventricular — there will almost always be at least *some* positive activity traveling toward the left ventricle (*and therefore positive in lead V6*).
- IF ever the QRS in **lead V6** is either ***all*** **negative** (*or almost all negative*) as in Figure 08.18-1 — then VT is *highly* likely (*See Figures 09.1-1 and 09.2-1*).
- Lead V6 is *only* helpful if it is negative … (*A positive R or RS in lead V6 is not helpful in ruling in or out VT*).

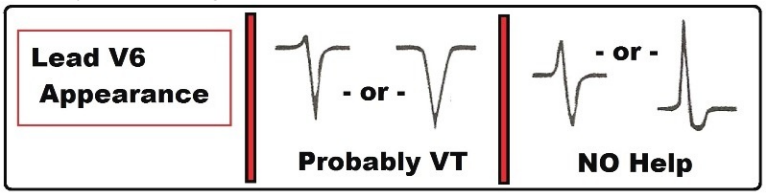


Figure 08.18-1: Using **QRS morphology** in **lead V6**. The presence of a QRS complex in lead V6 that is either all negative (*or almost all negative*) – is strongly suggestive of VT. This criterion is of *no help* if anything more than a tiny r wave is present in lead V6.

08.19 – WCT Diagnosis: Is the QRS "Ugly"? (*Simple Rule #3*)

Our 3rd *"Simple Rule"* is as follows: ***The "uglier" the QRS* — the *more* likely the rhythm is VT**. The explanation for this clinical reality is that *aberrant* conduction almost always manifests some form of conduction defect (*RBBB;*

LBBB; LAHB; LPHB — or some combination thereof) — due to *relative* delay in one or more of the hemifascicles or bundle branches.

- In contrast — VT originates from a ventricular focus *outside* of the conduction system. As a result – VT is more likely to be wider and far less organized (*therefore "uglier"*) in its conduction pattern.

PEARL: In our experience — Use of the **"3 *SIMPLE* Rules"** is easy and accurate for *recognizing* VT in the large majority of cases.

- **Rule #1** – Is there extreme axis deviation during WCT? (*Section 08.17*).
- **Rule #2** – Is lead V6 all (or almost all) negative? (*Section 08.18*).
- **Rule #3** – Is the QRS during WCT "ugly"? (*Section 08.19*).

08.20 – Beyond-the-Core: *Is there an RS in any Precordial Lead?*

What follows in Sections 08.20 thru 08.26 are a number of *Beyond-the-Core additional* ways to help distinguish *between* VT vs SVT:

- We emphasize that you do *not* have to remember all of the criteria that follow in these remaining sections. This is advanced (*beyond-the-core*) material for *experienced* providers with special interest in this fascinating area!

Is there an RS in *any* Precordial Lead?

- IF there is *no* RS complex in *any* precordial lead (*V1-thru-V6*) — then **the rhythm** is **VT!** (*with >99% specificity*).
- Caveat: IF an RS complex is seen in ≥1 precordial lead — then this criterion is of *no* help (*because both SVT and VT rhythms may have an RS complex in no more than a single precordial lead*).

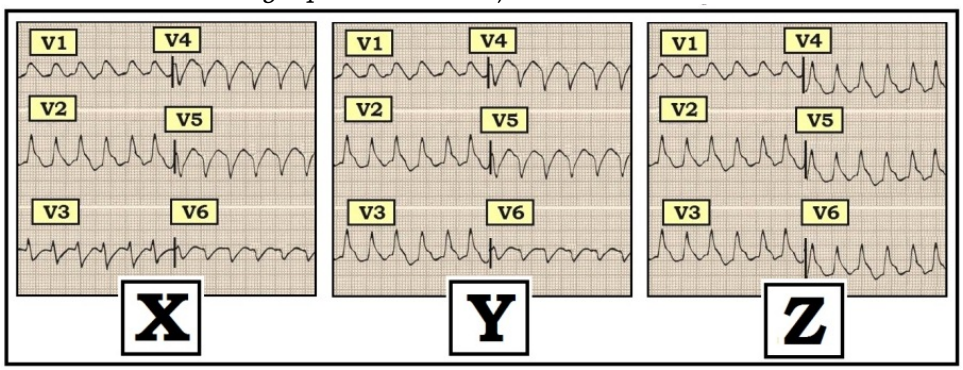


Figure 08.20-1: Is there an **RS** in *any* ***Precordial*** **Lead?** VT is almost certainly present if none of the precordial leads manifest an RS complex during the WCT (*See text*).

- *Both* **Y** and **Z** in Figure 08.20-1 are **VT** (*there is no RS complex in either*). The finding of **QRS *concordance*** in Z (*in this case global positivity*) is insensitive but 100% specific!
- In **X** — An RS is present in **lead V3** (*in the form of a small initial r wave and much deeper negative S wave*). We therefore can *not* rule out VT on the basis of this RS criterion. That said — We still think X is VT because of *other* criteria! (*'ugly' formless QRS, esp. in V1; almost entirely negative QRS in lead V6*).

08.21 – WCT Diagnosis: *Is the R-to-S Nadir Delayed?*

IF an RS complex is present in *at least* one precordial lead — then the **rhythm** is **VT** (*with >99% specificity*) — IF the **R-to-S Nadir** (ie*, interval from the beginning of the R wave until the deepest portion of the S wave*) is ***delayed*** to **>0.10 second** (*100 msec*).

- Caveat: This criterion is *only* helpful for *ruling in* VT if ≥1 precordial lead clearly manifests an R-to-S nadir >0.10 second. It is of ***no help*** if you see an RS nadir that is *not* more than 0.10 second (*and the reality is that it is often difficult to be sure of RS nadir duration*).

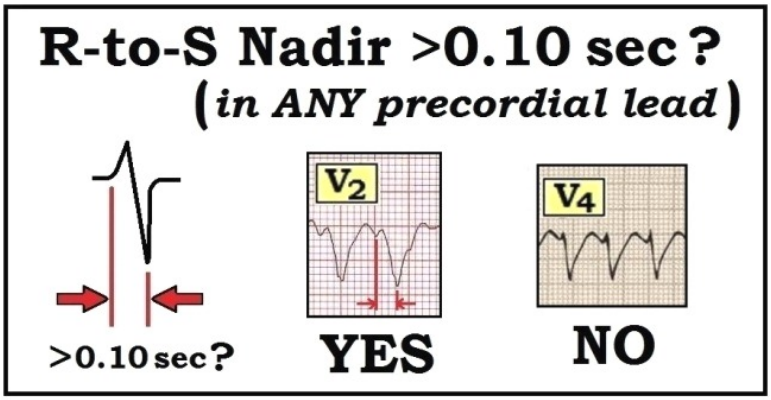


Figure 08.21-1: Is the **R-to-S Nadir >0.10 sec.** in *any* ***Precordial*** **Lead?** If an RS complex is present in one or more precordial leads – then the rhythm is *almost* certainly VT if the *R-to-S* Nadir is *delayed* to >0.10 second.

NOTE: The physiologic rationale for Figures 08.20-1 and 08.21-1 is that supraventricular activation should yield at least *some* change in the direction of ventricular activation with respect to the 6 precordial leads (*Fig. 08.21-0*) – and – that much of the time, *initial* ventricular activation will be slow (*>0.10 sec*) compared to significantly *faster* initial activation when the rhythm is supraventricular (*Fig. 08.21-1*).

08.22 – WCT Diagnosis: *Initial r or q ≥0.04 sec. in any Lead?*

Look in *all* 12 leads to see in which leads an *initial* r wave or q wave is present. IF an ***initial*** **r** or **q wave** is **≥0.04 sec** (*>1 small box*) in *any* lead – then the rhythm is *almost* certain to be **VT**.

- Caveat: This criterion is *only* helpful for *ruling in* VT if ≥1 lead clearly manifests an *initial* r or q wave ≥0.04 second. It is of ***no help*** if you do not see an initial r or q wave ≥0.04 second (*and the reality is that it is often difficult to be sure of q or r wave duration during WCT*).

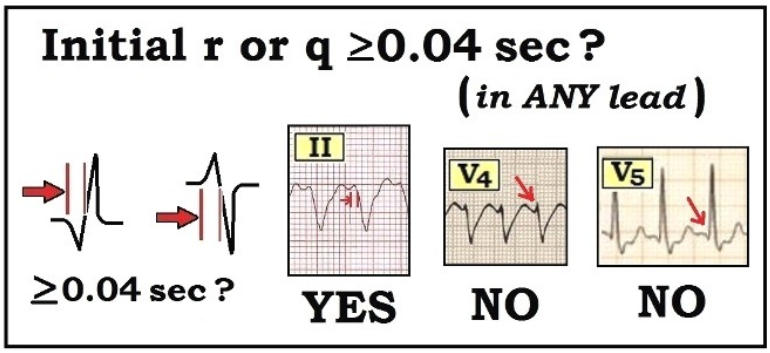


Figure 08.22-1: Is the *initial* **r or q ≥0.04 second** in *any* Lead? If an initial q or r wave is present and wide (*>0.04 sec*) in *any* lead – then the rhythm is *almost* certain to be VT.

NOTE: The physiologic rationale for Figure 08.22-1 is that initial conduction through myocardial tissue is *delayed* when the site of origin for a tachycardia is ventricular. In contrast — WCT rhythms of *supraventricular* etiology manifest more rapid *initial* conduction, because the impulse is transmitted (*at least in part*) over specialized conduction fibers.

08.23 – WCT Diagnosis: *Is there AV Dissociation?*

It is always good to look for potential ***confirmatory* criteria** when assessing WCT rhythms — since IF found, these virtually ***ensure*** the **diagnosis** of **VT**. Confirmatory criteria include: **i)** AV dissociation; and **ii)** Fusion beats.

- Caveat: Most WCT rhythms do *not* manifest either AV dissociation or fusion beats (*especially when the rate of VT is >130/minute*). Therefore, *not* seeing these proves nothing. But sometimes you'll get lucky (*Figure 08.23-1*)!

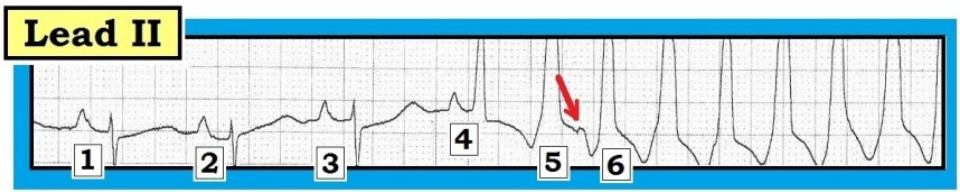


Figure 08.23-1: Use of AV Dissociation to prove VT (*arrow*). Beat #4 is a fusion beat (*See text*).

- Beats #1,2,3 in Figure 08.23-1 are sinus. The QRS then widens and dramatically changes in morphology. Although the beginning of this WCT is slightly irregular — We can ***prove*** this run is **VT** because: **i)** Beat #4 is a ***fusion* beat** (*short PR; QRS not overly wide and with QRS morphology intermediate between sinus beats and the other wide beats*); and **ii)** there is **AV Dissociation**, at least for a brief period (*arrow highlighting an on-time P wave not related to neighboring QRS complexes*).

The easiest way to explain ***"fusion beats"*** is to contemplate what the QRS would look like IF beats #4 and #6 in Figure 08.23-1 had children? (ie, *with characteristics of both beats!*).

- PEARL: You'll *need* **calipers** to look for AV Dissociation.
- Note: The reason the PR interval preceding beat #4 is *shorter-than-normal* is that it only *partially* conducts to the ventricles until its path is interrupted by a *simultaneously* occurring ventricular beat.

08.24 – WCT Diagnosis: *Large Monophasic R Wave in Lead aVR?*
With normal sinus rhythm — lead aVR manifests a predominantly *negative* QRS complex. This reflects the normal path of ventricular activation — which moves *away* from the right (*away from aVR*) — and toward the left ventricle. IF ever the **QRS** in **lead aVR *during* WCT** is ***entirely* positive** (*writing a large, monophasic R wave in aVR*) – then the rhythm is **VT** (*with virtual 100% specificity*)!

- Caveat: You will *not* often see a monophasic R in aVR during WCT. But sometimes you'll get lucky (*Figure 08.24-1*)!

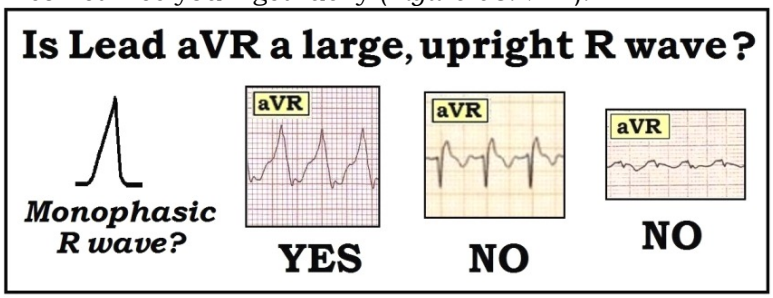


Figure 08.24-1: Is there a **large *monophasic* R** in **aVR?** If there is a monophasic R wave in lead aVR during WCT – then the rhythm is virtually certain to be VT.

NOTE: The finding of a monophasic R wave in lead aVR *during* WCT indicates that the electrical impulse *must be* **originating** from a site in the ***ventricular* apex** and traveling upward toward the base (ie, *in the direction of lead aVR*). Therefore — a *quick* look at **lead aVR** during WCT can *instantly* tell you the rhythm is **VT** if you see a large monophasic R wave.

08.25 – WCT Diagnosis: *Does Lead V1 suggest Aberrancy?*
Much has been written about *aberrant* conduction as a reason for QRS widening during WCT. For practical purposes — the *only* QRS morphology with ***high* specificity** for **SVT** is the presence of ***typical* RBBB** in lead V1. Thus, the presence of an **rsR' complex** (*with taller right 'rabbit ear'* and *S wave that descends below the baseline*) — ***strongly* suggests** a ***supraventricular* etiology** (*H-1,H-2 in Figure 08.25-1*).

- In contrast — ***any other* QRS morphology** in lead V1 (*H-3,4,5,6 in Figure 08.25-1*) ***favors* VT**.
- Caveat: This criterion is strict. Only a ***typical* RBBB** pattern in V1 (**H-1**,**H-2**) suggests aberrant conduction. *Any other* QRS pattern in lead V1 suggests VT.

We illustrate further in Figure 08.25-1 *diagnostic* use of lead V1 QRS morphology characteristics in assessment of WCT rhythms:

- Example H-7 — ***suggests* VT**. Lead V1 manifests a *monophasic* R wave with taller *left* rabbit ear (*resembles H-6, but without any notch*). Lead V6 in H-7 supports a diagnosis of VT because it is predominantly negative.
- Example H-8 — is *consistent* with a ***supraventricular* rhythm** (*either preexisting RBBB* – or – *aberrant conduction*). Lead V1 manifests an rSR' with taller *right* rabbit ear (*similar to H-2*).

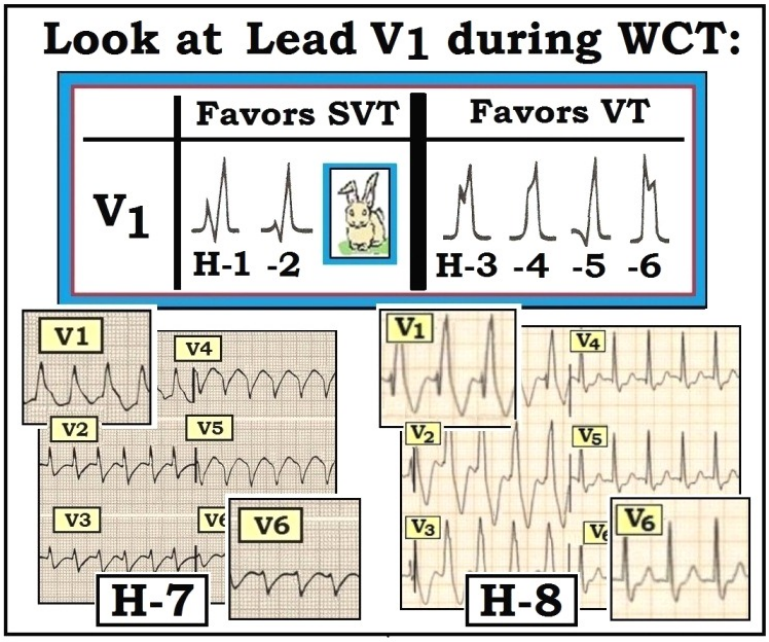


Figure 08.25-1: QRS morphology ***favoring* aberrancy** in **V1** (*See text*).

NOTE: The reason for ***aberrant* conduction** is that there is *insufficient* time for a part of the ventricular conduction system to recover. This may be precipitated by *either* an *early* beat (*like a PAC*) – or – by tachycardia. Because the right bundle branch tends to have a *longer* refractory period than both the left bundle branch and the hemifascicles — a RBBB pattern is the most common form of aberrant conduction (*but LAHB or LPHB aberration, or any combination of patterns may also be seen*) — *See Section 19.0*.

08.26 – WCT Diagnosis: *Is the run of WCT preceded by a PAC?*

The *best* way to *prove* aberrant conduction is IF you can find a ***premature* P wave** (*PAC*) preceding the run of WCT. This will often *not* be easy to do – BUT – on occasion you may see a tracing like **Figure 08.26-1:**

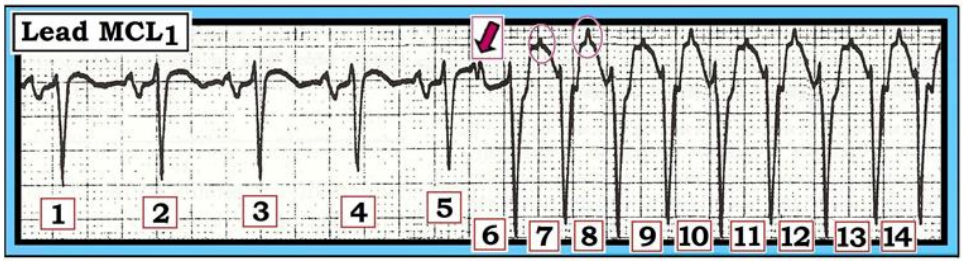


Figure 08.26-1: Beats #1-*thru*-5 in this ***right-sided*** (*comparable to V1*) **lead MCL1** are sinus conducted. There follows a 9-beat run of WCT (*beats #6-thru-14*). We *know* this is a run of **SVT** with ***aberrant* conduction** because of the PAC we see that *notches* the T wave *just prior* to the onset of the run (*arrow in Figure 08.26-1*). None of the other sinus beats (*#1-thru-4*) have this notch.

Beyond-the-Core on Figure 08.26-1: Although a *simultaneous* 12-lead ECG would be needed to know for sure – the similar initial r wave deflection with very steep S wave but *without* excessive QRS widening suggests an *incomplete* left bundle branch block form of aberrant conduction for beats #6-thru-14.

08.27 – WCT *Summary* with Review of 3 *Simple* Rules (*Figure 08.27-1*)

SUMMARY:

Despite the length and complexity of this section — the ***"message"*** is clear:

- **1st Priority:** — Is the patient stable? IF not – then immediately ***shock*** the patient!
- IF the patient **is Stable** – then *Apply* **Step #1** (*Section 08.3*) and **Step #2** (*Section 08.6*) in your attempt at determining the diagnosis (*or at least narrowing your differential*).
- Application of the **3 *Simple* Rules** (*covered in Sections 08.17, 08.18, 08.19 – and summarized below in Figure 08.27-1*) will usually allow you to greatly increase your diagnostic certainty in no more than a few seconds.
- Begin ***empiric* treatment** based on your presumptive diagnosis as you continually refine your rhythm diagnosis as indicated in **Step #3** (*Section 08.8*) and in our ***Suggested* Approach** (*Section 08.9*).

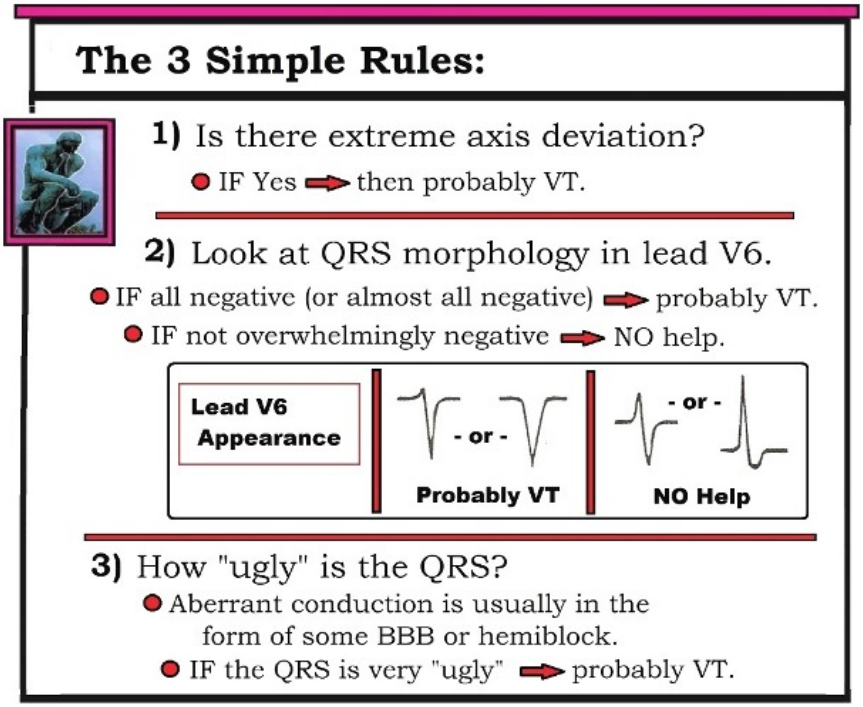


Figure 08.27-1: The 3 Simple Rules for assessing the 12-lead of a WCT rhythm (*Details in text of Sections 08.17, 08.18 and 08.19*).

Although you may not be certain of the rhythm diagnosis at the beginning of this process (*you are after all, dealing with an "unspecified" WCT*) — the chances are great that with ongoing monitoring, treatment, and follow-up — that you'll arrive at the correct diagnosis.

- In any event — the ***Suggested* Approach** (*Section 08.9*) will be an appropriate course to follow.
- Now – **Test *yourself*** in our **WCT PRACTICE!** (*Section 09.0*).

Section 09.0 – WCT Practice Examples

WCT Practice

We reinforce the principles discussed in Section 8.0 with a series of WCT (*Wide-Complex Tachycardia*) Practice Examples …

Section 09.1.0 – WCT Practice Example-1

Practice Example:

09.1.1 – WCT: *VT or SVT?*

Your patient is a 55-year-old man with CAD. His 12-lead ECG is shown below in **Figure 09.1-1**. The patient is *hemodynamically* stable with a BP =150/80.

- What should you do first?
- What is your diagnosis of the WCT rhythm in Figure 09.1-1?
- *How certain* are you of your rhythm diagnosis?

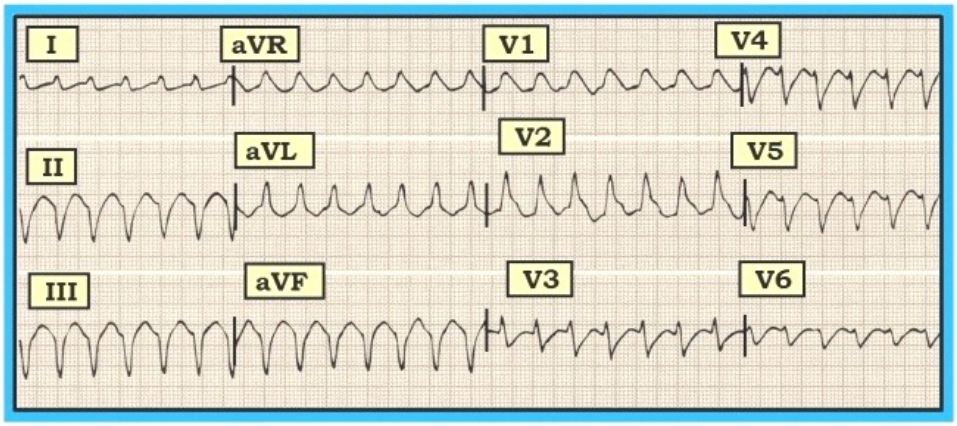


Figure 09.1-1: WCT Example-1. The patient is *hemodynamically* stable.

09.1.2 – KEY Points: *What To Do First?*

As discussed in Section 03.0 on *Overview of Unspecified Tachycardia* – ***the very 1st thing to do*** is assess the patient for *hemodynamic* stability. This has been done – and we are told that **the patient** whose rhythm is seen in Figure 09.1-1 **is *hemodynamically* stable**

- Since the patient is stable – there is *no need* to immediately cardiovert. Instead — there is *at least* a moment of time to ***assess the* rhythm**.
- Key concepts in *Rhythm Diagnosis* were discussed in Section 02.0 (*in which we reviewed clinical application of the Ps,Qs,3R Approach*). The diagnostic approach to *WCTs of Unknown Etiology* was then reviewed in detail in Section 08.0. *Feel FREE to refer back to these sections as needed.*
- The rhythm in **Figure 09.1-1** is a ***regular* WCT** *without* clear sign of atrial activity. Given that the patient is a 55-year-old man with *known* CAD – the ***likelihood*** of **VT** is already ***at least* 90%** *even without* looking further (*See List #1 and Sections 08.7 and 08.15*).
- As suggested by **Step #3** in Section 08.8 – one could at this point *either* empirically treat the rhythm in Figure 09.1-1 as a **WCT** of ***Unknown***

Etiology (*Sections 08.9 through 08.13*) – or – one could further assess the rhythm to see if we can ***increase*** **certainty** of our rhythm diagnosis.

- We emphasize that it would *not* be wrong to begin empiric treatment (*with either Adenosine, Amiodarone and/or Procainamide – as described in Sections 08.10 through 08.12*). That said – We feel the treatment approach will be *far better* IF we can hone in on the rhythm diagnosis. It should take *no more* than *2-to-5 seconds* to assess the rhythm in Figure 09.1-1 by applying the **3 *Simple* Rules** (*Sections 08.17 – 08.18 – 08.19*) – and this is the approach we favor.
- NOTE: IF at *any* time the **patient *becomes* unstable** — then *immediately* cardiovert or defibrillate.

09.1.3 – Figure 09.1-1: *Applying the 3 Simple Rules*

It should take no more than a few seconds to apply the 3 Simple rules (*Figure 09.1-2*) to the rhythm in Figure 09.1-1:

- **Rule #1: *Extreme Axis Deviation?*** — There is *extreme* LAD (*Left Axis Deviation*) in Figure 09.1-1 (*the QRS is entirely negative in the inferior leads*). This is virtually *never* seen with SVT …
- **Rule #2: *Is Lead V6 Negative?*** — The QRS in lead V6 is *almost* entirely negative. This is rarely seen with SVT …
- **Rule #3: *Is the QRS "Ugly"?*** — The QRS in Figure 09.1-1 is *extremely* wide (*almost 0.20 sec*) and formless. We say it is ***"ugly"*** because QRS morphology does *not* resemble any form of BBB or hemiblock. This *strongly* favors VT.

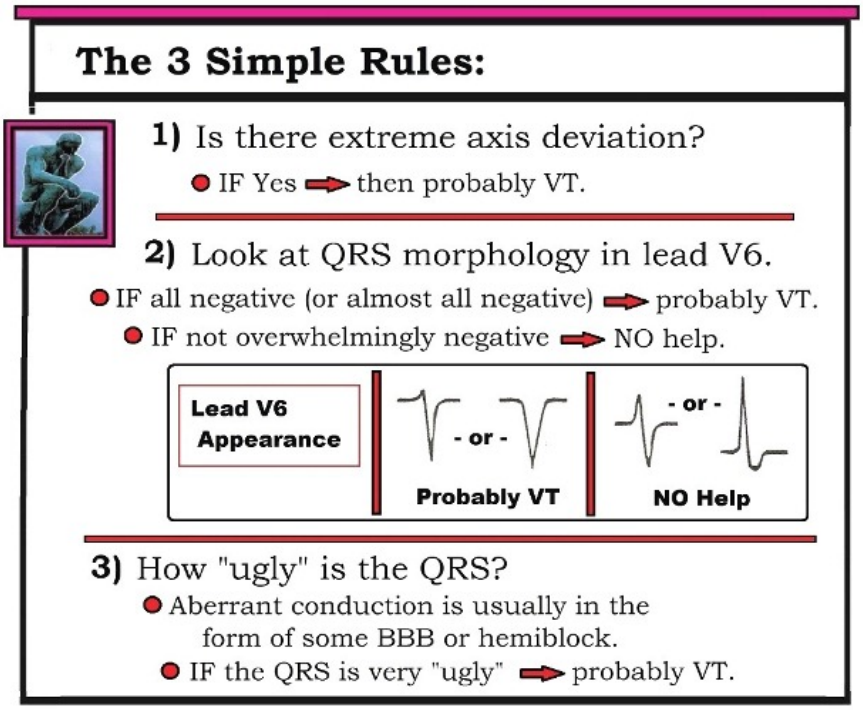


Figure 09.1-2: Summary of the 3 Simple Rules (*Details in Sections 08.17, 08.18 and 08.19*).

Conclusion: In *less* than 5 seconds – By use of the **3 Simple Rules** we have become 99% certain that the WCT in Example-1 is VT.

- It would *not* have been wrong to start with **Adenosine**. That said — We would *promptly* switch to other treatments if Adenosine didn't work since the

patient's age, history of CAD, and ECG appearance do <u>not</u> suggest an *adenosine-responsive* form of VT is likely (*Section 06.5*).
- Our preference after Adenosine would be Amiodarone — but *other* options are available (*Section 07.3*).

09.1.4 – Figure 09.1-1: *Beyond-the-Core*

We can actually be **100% *certain*** this WCT is VT:
- There is a monophasic *upright* R wave in lead aVR. Although insensitive — this finding is highly specific for VT when it is found (*Section 08.24*).

<u>QUESTION:</u> Does the upright R wave in lead V1 of Figure 09.1-1 suggest RBBB or *aberrant* conduction?
- <u>HINT:</u> Feel free to review Section 08.25 and Figure 08.25-1 before answering.

<u>ANSWER:</u> The very wide and formless (*very 'ugly'*) QRS in lead V1 does not in the least resemble either H-1 or H-2 in **Figure 08.25-1**. If anything — QRS morphology in lead V1 of Figure 09.1-1 is strongly in favor of VT.

Practice Example:

09.2.1 – Heart "Awareness" and Tachycardia: *What is the Rhythm?*
Your patient is a 50-year-old man with CAD and "heart awareness". His ECG is shown below. BP=140/90.

- What should you do next?

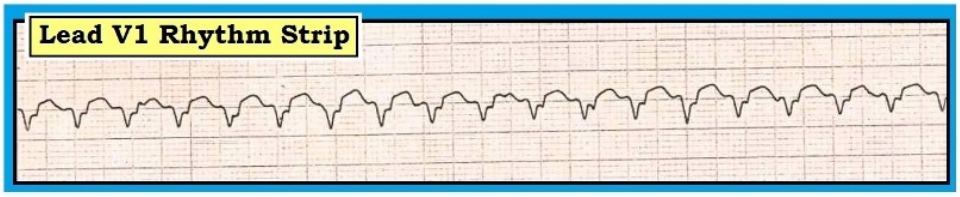


Figure 09.2-1: The patient is stable. Is this SVT?

09.2.2 – KEY Points: *What To Do First?*
This patient seems to be stable (*BP=140/90*). The Lead V1 rhythm strip in Figure 09.2-1 *appears* to show a regular *narrow* tachycardia at a rate just over 150/minute. The "good news" is that since the patient is stable – there is time to look further into what the rhythm might be!

- All we see is a *single* monitoring lead. Given that the patient is stable — We'd like to see ***more* leads** *before* proceeding. Therefore — *Get a* **12-lead ECG** *during* tachycardia!
- **NOTE:** IF at *any* time during the process the **patient *becomes* unstable** — then *immediately* cardiovert or defibrillate.

09.2.3 – Does Figure 09.2-1 *belong* in this WCT Section?
The answer as to whether the rhythm in Figure 09.2-1 "belongs" in this WCT Practice Tracing Section is forthcoming on seeing the **12-lead ECG** recorded at the *same* time as the lead V1 rhythm strip (*Figure 09.2-2*):

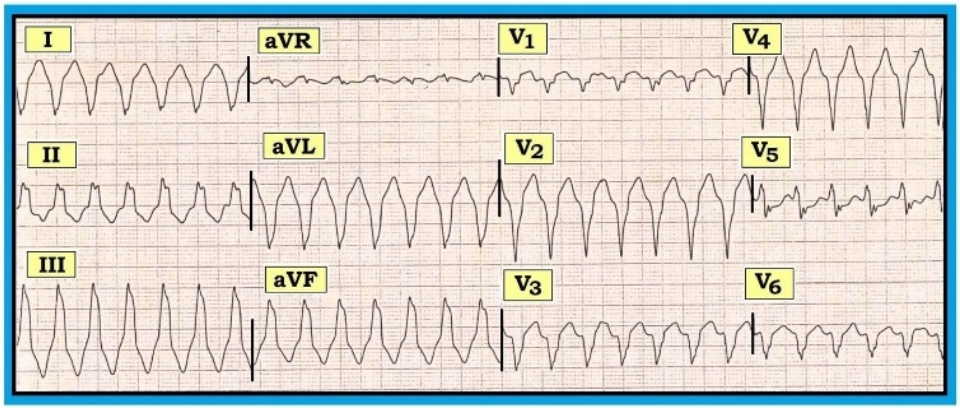


Figure 09.2-2: 12-lead ECG recorded at the *same* time as the lead V1 rhythm strip shown in Figure 09.2-1. The patient is stable (*BP=140/90*).

QUESTIONS:

- What is the rhythm in Figure 09.2-2: *VT or SVT?*
- What *degree of certainty* do you have about your rhythm diagnosis?

09.2.4 – What is the Rhythm in Figure 09.2-2?
Comparison of the *single-lead* V1 rhythm strip (*Figure 09.2-1*) with the *simultaneously* recorded **12-lead ECG** from this patient (*Figure 09.2-2*) illustrates the following KEY concept: ***"12 leads are better than 1"***. Part of the QRS may sometimes lie on the baseline in the *single* lead being monitored. For this reason — it is best *whenever possible* to always get a **12-lead ECG *during* tachycardia** to verify QRS width.

- It should now be *obvious* that the **QRS complex** in this case is **wide**! In fact — the *only* lead in which the QRS looks to be narrow on the 12-lead tracing obtained *during* tachycardia is lead V1.
- We strongly suspect VT. Applying the **3 Simple Rules** to the 12-lead ECG shown in Figure 09.2-2 allows us to *greatly* increase certainty of our rhythm diagnosis (*Section 09.2.5*):

09.2.5 – Figure 09.2-2: *Applying the 3 Simple Rules*
Although it would *not* be wrong to give Adenosine at this point — it should take *no more* than *2-to-5 seconds* to apply the 3 Simple Rules (*Figure 09.2-3*).

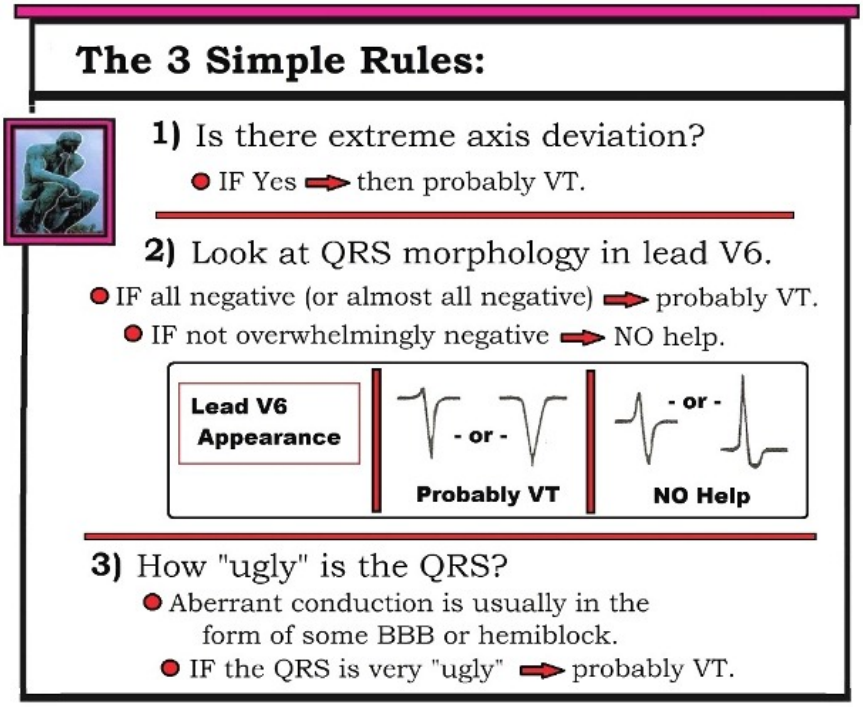


Figure 09.2-3: Summary of the 3 Simple Rules (*Details in Sections 08.17, 08.18 and 08.19*).

Applying the 3 Simple Rules to Figure 09.2-3:

- **Rule #1: *Extreme Axis Deviation?*** — There is *extreme* RAD (*Right Axis Deviation*) in Figure 09.2-3 (*the QRS is entirely negative in lead I*). This is *not* seen with SVT.
- **Rule #2: *Is Lead V6 Negative?*** — The QRS in lead V6 is entirely negative. This is virtually *never* seen with SVT.
- **Rule #3: *Is the QRS "Ugly"?*** — The QRS in Figure 09.2-3 is *extremely* wide (*almost 0.20 sec*) and formless. We say it is ***"ugly"*** because QRS morphology does *not* resemble any form of BBB or hemiblock. This *strongly* favors VT.

Conclusion: In *less* than 5 seconds — We have become virtually **100%** ***certain*** the WCT in Figure 09.2-2 is **VT**.

- Although acceptable to start with Adenosine — our preference would be to select Amiodarone first in view of the virtual *certainty* of *ischemic* etiology VT (*given patient's age; history of CAD; ECG characteristics*) — and that this VT is unlikely to be *adenosine-responsive*.
- *Other* options for VT are available (*Section 07.3*).
- *Synchronized* cardioversion may be needed IF the patient fails to respond to antiarrhythmic drugs.

09.2.6 – Figure 09.2-2: *Beyond-the-Core*

So certain are we at this point that diagnosis of the rhythm in Figure 09.2-2 is VT — that clinically, we would *not* need to spend time looking further to confirm this. That said — for teaching purposes:

- The *blow-up* of **Lead V5** from Figure 09.2-2 provides an excellent example of an RS complex in which the ***initial*** **R** is ***clearly*** **≥0.04 sec.**, which virtually *ensures* VT (*Section 08.22*).

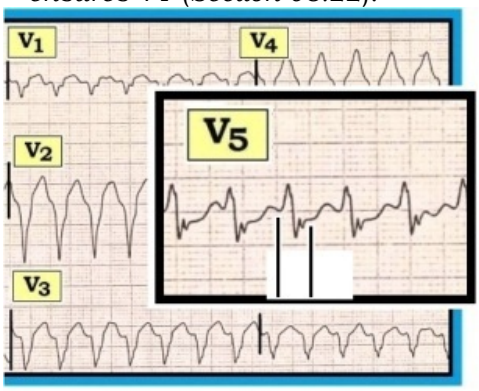


Figure 09.2-4: Blow-up of lead V5 from the 12-lead ECG previously shown in Figure 09.2-2. The very wide (>0.04 second) initial R wave in this lead virtually confirms VT as the diagnosis (*See Section 08.22 for details*).

Final Point: The rationale for *routine* incorporation of Adenosine at an *early* point in VT management is that one can *not* reliably identify all *adenosine-responsive* cases on the basis of ECG characteristics.

- ***Adenosine-responsive*** forms of **VT** (*Section 06.5*) — are most likely to occur in *younger* adults *without* underlying heart disease. The ECG is more likely to manifest *minimal* QRS widening *without* bizarre morphology — and VT episodes are more likely to be precipitated by exercise (*or other causes of catecholamine release*). This is *not* the case here.

Section 09.3.0 – WCT Practice Example-3

Practice Example:

09.3.1 – Heart Failure and Tachycardia: *What is the Rhythm?*
Your patient is a 65-year-old woman with heart failure exacerbation. Her ECG is shown below. BP=140/90.

- What should you do next?

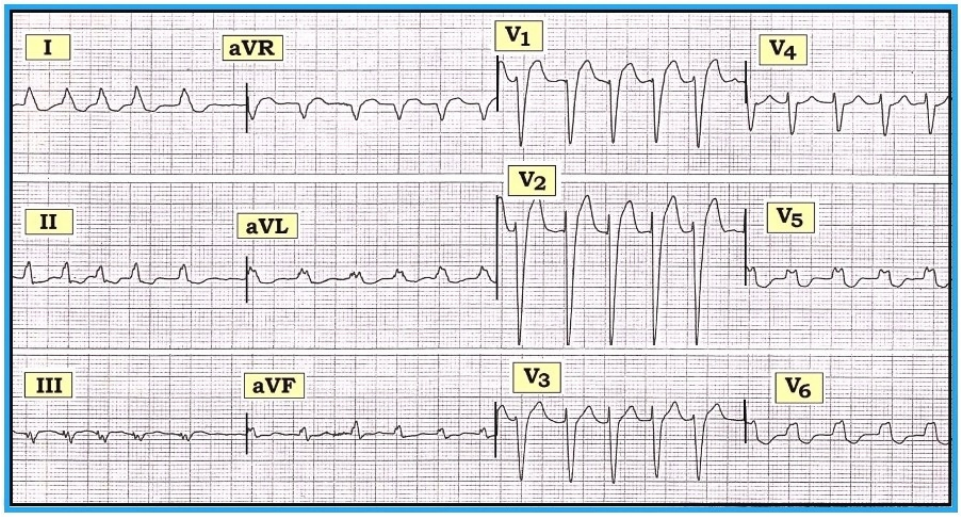


Figure 09.3-1: The patient is stable. Is this VT or SVT?

09.3.2 – KEY Points: *What is the Rhythm in Figure 09.3-1?*
Despite first glance impression that the rhythm in Figure 09.3-1 appears to be regular — it is *not*. Fortunately, this patient is stable — so there is time to look further.

- **HINT:** Use of ***calipers*** greatly facilitates assessment of rhythm regularity …
- The *underlying* rhythm in Figure 09.3-1 is *irregularly* irregular. No P waves are seen. We suspect this is **AFib (***Atrial Fibrillation***)** with a **fairly *rapid* ventricular response**.
- The **QRS complex** is ***wide***. Although VT is *usually* a fairly regular rhythm — it may at times be irregular. Thus, we can *not* with 100% certainty exclude the possibility of VT. That said — VT is rarely as *irregularly* irregular as is seen in Figure 09.3-1. We therefore ***suspect*** this patient has ***preexisting* LBBB**.

09.3.3 – When You Don't Know For Sure What the Rhythm Is …
This case provides an excellent example of how one will *not* always know with 100% certainty what the rhythm is at the time treatment decisions need to be made.

- Assessment of **ECG features** in Figure 09.3-1 is consistent with our presumption of a *supraventricular* etiology because: **i)** there is typical LBBB morphology (*upright monophasic QRS in leads I,V6; predominantly negative QRS in V1*); **ii)** the QRS is *not* overly wide; **iii)** there is no extreme axis deviation; and **iv)** the downslope of the S waves in anterior leads is very steep (*unlike the delay that is often seen with VT*).

- It would be wonderful IF we had access to a ***prior* ECG** on this patient. Evidence of LBBB in the past would confirm the rhythm in Figure 09.3-1 is AFib and *not* VT.
- Review of ***additional* rhythm strips** on this patient should also help to confirm the *irregularly* irregular nature of AFib (*vs VT that tends to regularize after an initial period of irregularity when the rhythm persists*).
- The "good news" — is that this patient is stable. Essential to management will be treatment of her heart failure exacerbation. One would expect the rate of her presumed AFib to *slow* as her clinical condition improves.
- **Bottom Line:** We strongly suspect that the rhythm in Figure 09.3-1 is AFib with *preexisting* LBBB. While remaining ready to cardiovert this patient IF at *any* time she were to decompensate — We would begin by treating her heart failure and cautiously use drugs to slow the rate of her presumed AFib (*See Section 14.1*).

09.3.4 – Figure 09.3-1: *Beyond-the-Core*

The case scenario presented here is a common one. Progressive diastolic dysfunction from longstanding hypertension may predispose to *both* AFib and to development of LBBB. Sudden loss of the 'atrial kick' with onset of AFib may precipitate acute heart failure. Given minimal R-R interval variation when the rate of AFib is fast — the resultant ECG picture may mimic VT.

- It really helps to know if the patient has baseline LBBB.
- The best clues that the rhythm in Figure 09.3-1 is **AFib** are: **i)** Awareness of the above common scenario; and **ii)** realization that the R-R interval *continually* changes.

09.3.5 – PEARL: *Using Calipers*

Use of ***calipers*** is *invaluable* as a tool to assist in assessment of arrhythmia tracings such as Figure 09.3-1 — as well as for assessment of AV blocks.

- **Calipers *instantly* enhance your skills** in arrhythmia interpretation! They make obvious relationships between atrial activity and QRS complexes that would not otherwise be apparent. Detecting subtle variation in atrial or ventricular rate becomes easy. And – Using calipers conveys to others that YOU *know* what you are doing. All it takes is a *little* bit of practice to become facile in using calipers.
- Clearly – You will *not* have time to pull out calipers if your patient is "crashing" in front of you. That said, in such situations – a patient with hemodynamically *unstable* tachycardia (*where instability is due to the rapid rate*) should be immediately cardioverted or defibrillated *regardless* of whether the rhythm is regular or not.
- **BOTTOM Line:** The diagnosis of certain cardiac arrhythmias will be missed if you never use calipers. While you may not necessarily need them for interpretation of many (*most*) arrhythmias – it is good to be aware of situations in which calipers will be of invaluable assistance!

Practice Example:

09.4.1 – Palpitations and Tachycardia: *What is the Rhythm?*
Your patient is a previously healthy 30-year-old woman who presents with palpitations. Her ECG is shown below. BP=145/80.
• What should you do next?

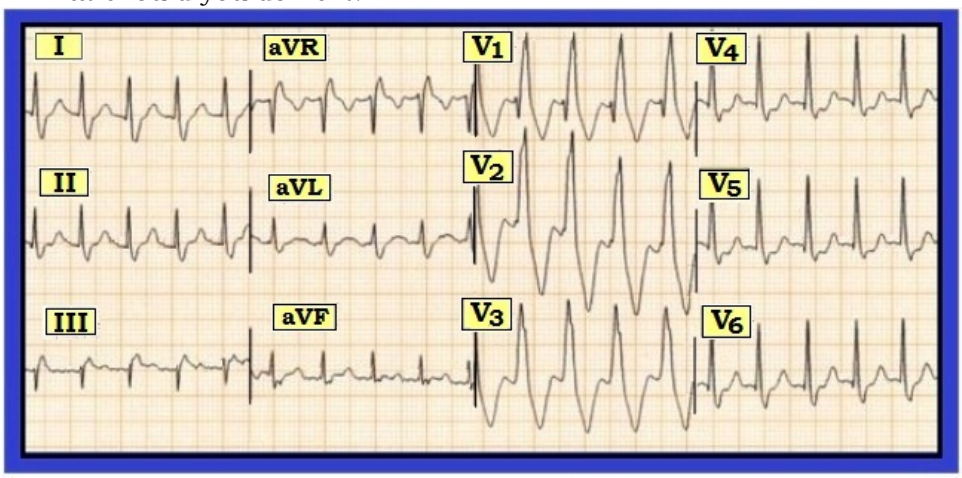


Figure 09.4-1: The patient is stable. Is this VT or SVT?

09.4.2 – KEY Points: *What is the Rhythm in Figure 09.4-1?*
A ***regular* WCT** at ~150/minute is seen in Figure 09.4-1. There is *no* clear sign of atrial activity. Although VT *always* needs to be presumed until proven otherwise (*Table 08.7-1 - LIST #1*) — there are a number of reasons why we ***strongly* suspect** a ***supraventricular* etiology** in this case. Consider the following:
• The patient is young (*30 years old*) — she has been *previously* healthy – and – she is *hemodynamically* stable. While *none* of these clinical features rules out the possibility of VT — they do make VT much *less* likely.
• Even if VT is present — the patient's age, *lack* of cardiac history, and hemodynamic status *increase* the likelihood of some type of fascicular VT or *adenosine-responsive* form of VT (*Section 06.5*). In *either* case — ***trial*** of **Adenosine** is the appropriate initial step.
• **QRS morphology** in Figure 09.4-1 — is ***typical* RBBB** (*rsR' with taller-right-rabbit-ear in V1; wide terminal S waves in I,V6*). This strongly suggests **PSVT** *with* QRS widening from ***RBBB* aberration** as the etiology.

09.4.3 – Figure 09.4-1: *Approach to Management*
There would seem to be *no* downside from *initial* management of the rhythm in Figure 09.4-1 with **Adenosine** (*Section 06.0*) — since this drug stands *high* probability of converting the arrhythmia (*be the rhythm PSVT with aberrant conduction or some form of adenosine-responsive VT in this relatively young adult*).
• Application of a ***vagal* maneuver** (*Section 13.8*) might also be tried (*even before Adenosine*). Vagal maneuvers often work for PSVT and on occasion, even for *adenosine-responsive* forms of VT and fascicular VT.

- Be ready to cardiovert IF at *any* time during the treatment process the patient decompensates.
- Obtaining a ***post-conversion* 12-lead ECG** would be very important in this case in the hope of determining IF there is *baseline* RBBB.
- An **Echo** should be done to assess for *underlying* structural heart disease – and – ***referral*** may be in order (*especially IF fascicular VT is suspected* or *if there is recurrence of WCT*).

09.4.4 – Figure 09.4-1: *Beyond-the-Core*

In general — *neither* Verapamil *nor* Diltiazem should ever be given for a WCT rhythm unless one is 100% certain that the WCT is *not* VT. This is because the *vasodilating* and *negative* inotropic effects of these drugs is likely to precipitate deterioration of VT to VFib …

- The above said — it is well to be aware that the special form of VT known as ***fascicular* VT** may respond (*and convert to sinus rhythm*) with use of Verapamil/Diltiazem. ECG recognition of fascicular VT may be subtle (*usually presents with a RBBB/LAHB pattern without P waves in a previously healthy younger adult*).

Bottom Line: For the non-expert — it is probably best to *avoid* Verapamil/Diltiazem in the acute setting *unless* you are 100% certain that the WCT rhythm is *not* VT.

- Access to a ***prior* ECG** on this patient showing baseline RBBB of *identical* QRS morphology as during the WCT would confirm a supraventricular etiology. (Unfortunately — *Most of the time, no prior tracing will be available*…).
- In 2013 — Certain forms of VT as well as many (*most*) reentry SVTs are potentially *curable* by ablation. **EP referral** may at some point be in order.

Section 09.5.0 – WCT Practice Example-5

Practice Example:

09.5.1 – "Heart Disease" and Tachycardia: *What is the Rhythm?*
Your patient is a 60-year-old man with "heart disease". His ECG is shown below. BP=160/90.

- What should you do next?

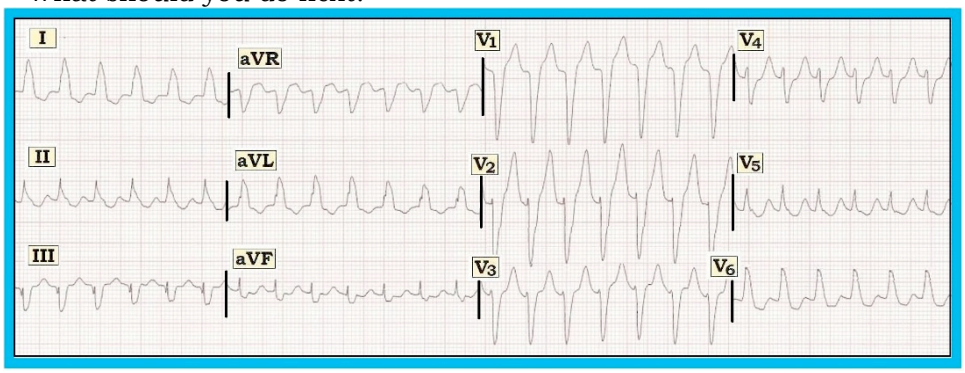


Figure 09.5-1: The patient is stable. Is this VT or SVT?

09.5.2 – KEY Points: *What is the Rhythm in Figure 09.5-1?*
A ***regular* WCT** is seen at a rate of ~160/minute. There is *no* clear evidence of atrial activity. The ***differential* diagnosis** is that as shown in **LIST #1** (*Section 08.7*): = VT, VT, VT until *proven* otherwise.

- Given the patient's age and history of "heart disease" — *statistical* likelihood of VT is ~90% *without* going further.
- The above said — there IS a chance that the rhythm in Figure 09.5-1 *could* be SVT (*with either aberrant conduction* or *preexisting BBB*).

09.5.3 – Figure 09.5-1: *Approach When Uncertain of the Diagnosis*
The 12-lead tracing in Figure 09.5-1 provides an excellent example of how to approach a **WCT Rhythm** when you do *not* know for sure what the diagnosis is (*Section 08.0*):

- The patient is **stable** (ie, *there is time to look further*).
- The WCT is ***regular***. Therefore - this is *not* AFib (*Step #2 in Section 08.6*).
- The QRS is **monomorphic** (*all QRS complexes in a given lead look the same*). Thus, this is *not* polymorphic VT or Torsades (*Step #2A in Section 08.6*).
- Assessment of **QRS morphology** is ***inconclusive***. That is — the *'3 Simple Rules'* do *not* suggest VT (*Figure 08.27-1*). Specifically — the **axis** *during* WCT is normal — **lead V6** is upright – and – the **QRS** is ***not*** **"ugly"**, but instead is perfectly consistent with LBBB.

Bottom Line: We *don't* know for sure what the rhythm in Figure 09.5-1 is. Although our initial assessment does *not* point to a ventricular etiology — We still need to *assume* VT until *proven* otherwise. That said — the patient is stable and **Adenosine** is the most appropriate *initial* treatment (*Sections 08.9,08.10*).

- ***Failure of Adenosine*** — to either temporarily *slow* the rate or convert the rhythm would support the premise that the rhythm in Figure 09.5-1 is VT. At this point — We would then move on to **Amiodarone** (*or other VT drug*).
- *Successful* conversion of the rhythm by Adenosine would support (*but not definitively prove*) a supraventricular etiology (*Section 06.6*).
- *Remain* ready to cardiovert — IF at *any* time the patient *becomes* hemodynamically unstable.
- Ask someone to search this patient's chart in the hope of finding a ***prior* 12-lead ECG** that might tell if this patient had *baseline* LBBB.

09.5.4 – Figure 09.5-1: *Beyond-the-Core*

This case highlights a number of important points:

- *Definitive* diagnosis of the rhythm in Figure 09.5-1 is *not* needed to effectively treat the patient. Instead we follow the course laid out for WCT of *Uncertain* Etiology (*Sections 08.9 thru 08.13*).
- Use of the '3 Simple Rules' does not point toward VT in this case. Nevertheless, these Rules still help because they make SVT a more *plausible* possibility.
- Assessment of more *advanced* QRS morphologic features likewise fails to yield a definitive answer (*Sections 08.20 thru 08.26*). That is — *at least* one rS complex is present in precordial leads (*seen here in V2,V3,V4*) and there is *no delay* in S wave downslope in V1,V2,V3. The initial r wave in lead V4 is *not* wide.
- The ECG shown in Figure 09.5-1 is the 12-lead *during* tachycardia for the **lead II *rhythm* strip** previously shown in **Figure 03.1-1** and in **Figure 08.1-1**. It is now obvious that the QRS complex is wide (*whereas QRS width was not certain in Figures 03.1-1 and 08.1-1*). "12 leads are *better* than one!"
- Use of the **12-lead *during* tachycardia** is also helpful in clarifying questions about atrial activity. For example — one might wonder IF the *upright* deflection midway between QRS complexes in lead II (*and in other leads*) could be a sinus P wave? (*arrows in Figure 09.5-2*).

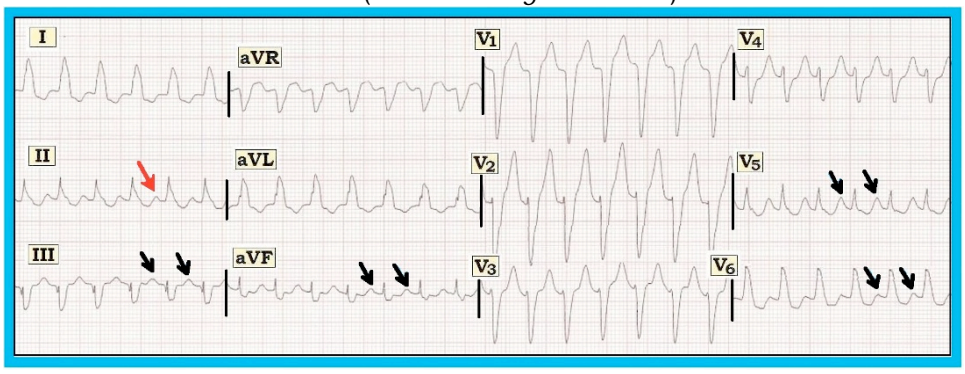


Figure 09.5-2: We have added *arrows* to Figure 09.5-1. There is a *regular* WCT rhythm. *No* definite P waves are seen (*See text*).

While we *cannot* rule out the possibility that sinus P waves *might* be hiding within preceding T waves in Figure 09.5-2 (*arrows*) — lack of "telltale" atrial notching defines this rhythm as a **monomorphic *regular* WCT** of ***uncertain***

etiology (*List #1 – Section 08.7*). Ventricular tachycardia *must* be assumed until *proven* otherwise.

- Access to a ***prior* 12-lead ECG** on this patient would favor SVT if LBBB with *identical* morphology was seen.
- IF the patient remained stable — Use of a ***Lewis* Lead** might be attempted looking for atrial activity (*Section 10.0*).

Final Point: The measures listed on **Sections 08.20 *thru* 08.26** under *'Beyond-the-Core'* are just that = *advanced* and aimed for *experienced* providers desiring to know more.

- Appropriate management of this case is possible *without* necessarily pursuing these advanced measures …

Use of a Lewis Lead

10.1 – Use of *Special* Lead Systems

Use of *special* lead systems may sometimes provide diagnostic insight in the search for atrial activity. By varying the anatomic landmarks used for electrode lead placement — a different electrical viewpoint is obtained, which may reveal atrial activity *not* previously visualized when using the 12 standard leads.

10.2 – Application of a Lewis Lead (*Figure 10.1-1*)

Do the following to record a ***Lewis* Lead:**

- Place the **RA** (*Right Arm*) **electrode** on the *right* side of the sternum at the 2nd ICS (*InterCostal Space*).
- Place the **LA** (*Left Arm*) **electrode** on the *right* side of the sternum at the 4th ICS.
- Record the ECG. The ***Lewis* Lead** will now be seen in **Lead I**. Adjust calibration to 1mV=20mm (*which is twice normal size*) to facilitate visualization of atrial activity.

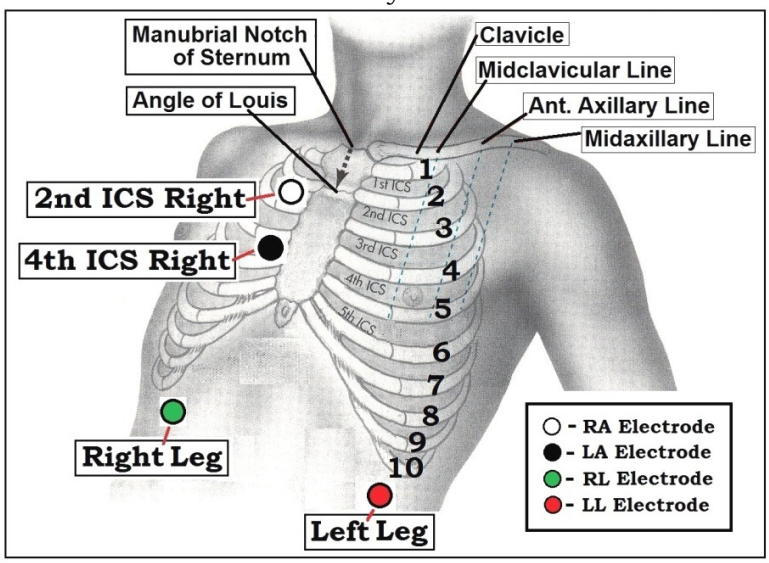


Figure 10.1-1: Application of a ***Lewis* Lead** (*See text*).

NOTE: Use of a ***Lewis* Lead** recording is an *advanced* intervention. It is *not* needed for appropriate evaluation and management in the overwhelming majority of cases. Nevertheless — this *extra* monitoring lead (*first described by Sir Thomas Lewis in 1931*) may at times help with diagnosis of **problematic *sustained* WCT rhythms** of ***uncertain* etiology**.

- By facilitating detection of atrial activity — occult but *"telltale"* AV dissociation (*that is diagnostic of VT*) is much more easily recognized.
- By definition — the patient must be *stable* and in a *sustained* tachycardia for a Lewis Lead to be used.
- IF at *any* time the patient shows sign of decompensating — *STOP* monitoring and *immediately* cardiovert.