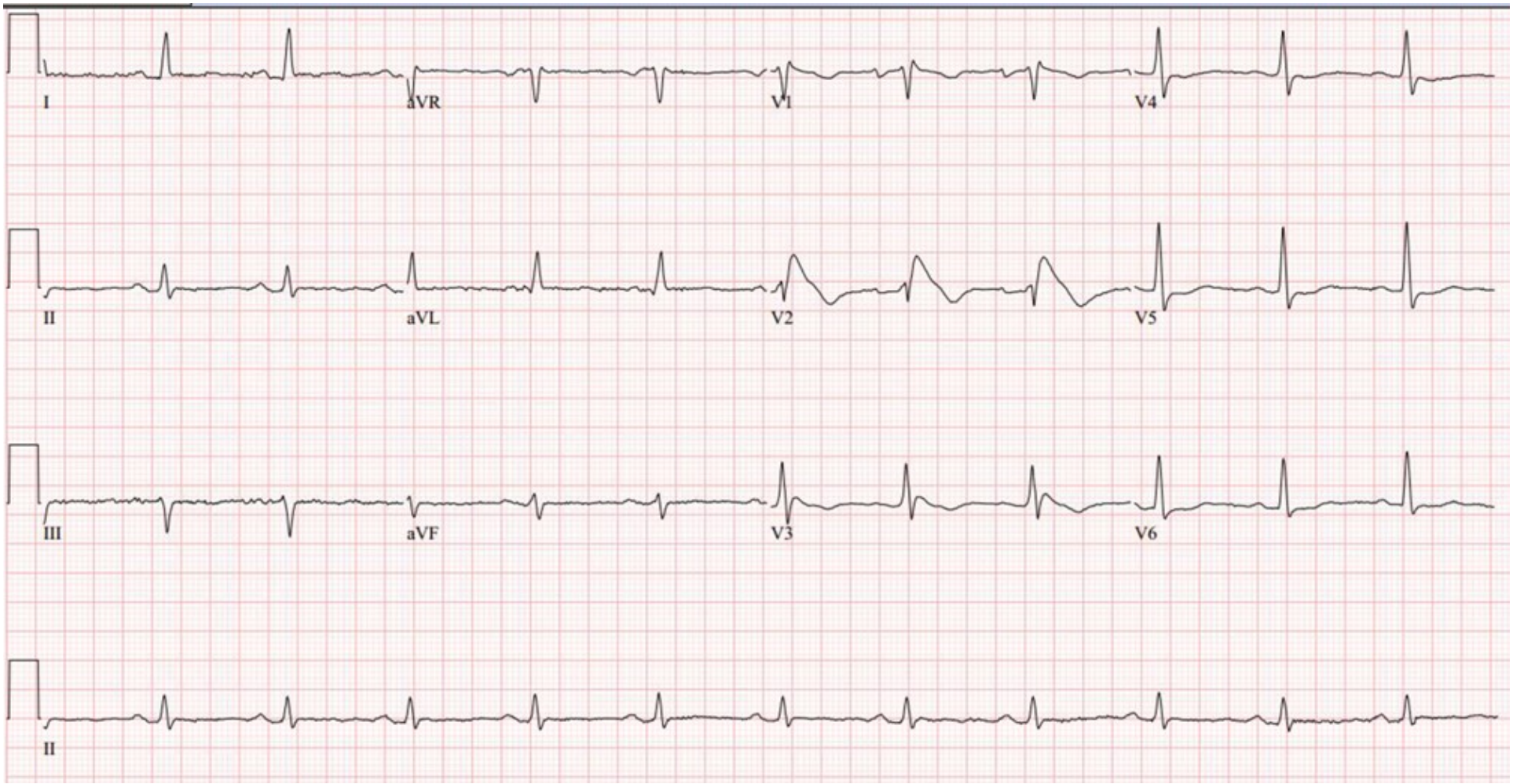
Triage EKG—Unit 3, Case 9



EKG courtesy of Shanna Jones, MD

Unit 3, Case 9—Brugada Syndrome

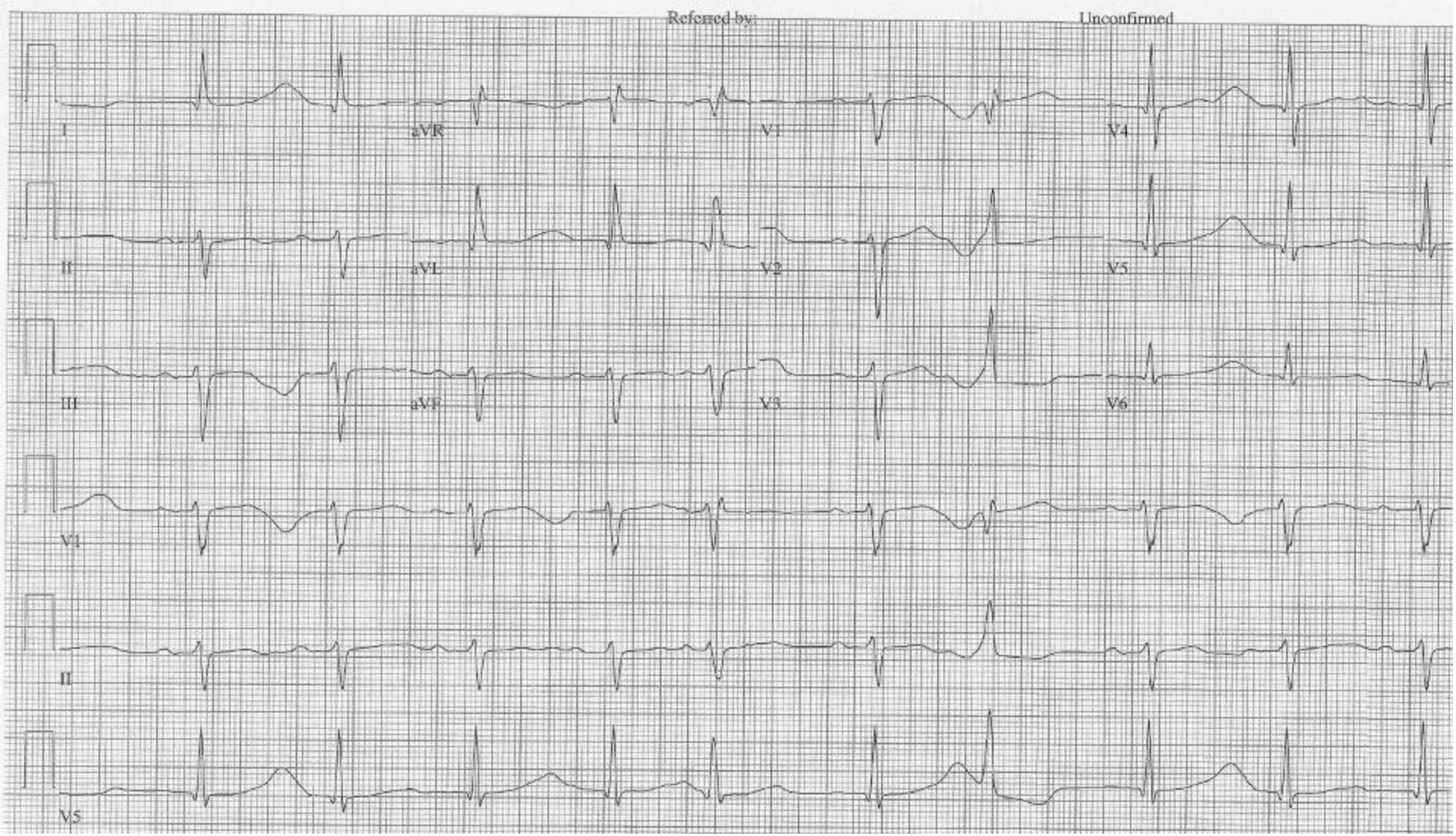
What is your interpretation of the EKG?
History/Clinical Picture— Young man presents with syncope
Rate— 66
Rhythm— sinus rhythm
Axis— normal
P Waves— present abnormal morphology. Long duration >120ms in II and deep (>1mm) terminal negative portion in V1 which are both suggestive of left atrial enlargement
Q, R, S Waves— Narrow, bizarre slurring of R wave in V2-3
T Waves— Inversions in V1-3 and diffuse flattening
U Waves— not present
PR Interval— normal
QRS Width— normal to slightly prolonged
ST Segment— coved STE in V2 & V3 sloping into an inverted t-wave consistent with Type 1 Brugada pattern. Similiar but less overt morphology in V1.
QT Interval— very likely prolonged but difficult to assess given such flat T waves. QTc based on lead V2 & V3 is approximately 500ms
Diagnosis: Brugada Sign, suspicion of Brugada Syndrome given associated syncope

Discussion: Brugada syndrome is the result of an inherited or spontaneous mutation in cardiac sodium channels that predisposes patients to ventricular tachyarrhythmias and sudden cardiac death. To be diagnosed with the syndrome, one must have ECG features and symptoms consistent with ventricular tachyarrthmia (syncope, palpitations, etc). Undiagnosed, Brugada syndrome carries a 5-10% risk of ventricular arrhythmia. Patients diagnosed in the ED should be admitted for cardiology consult and consideration of ICD placement. ECG changes consistent with Brugada syndrome come in 3 types **however only Type 1 is diagnostic for the disease.**
Type 1 - coved ST-elevation in V1-2 sloping into an inverted T-wave
Type 2 - saddle back shaped ST-elevation in V1-2 with >2mm STE
Type 3 - can have either a type 1 or 2 morphology but has <2mm STE
Patient's with Brugada syndrome may have normal ECGs at baseline, and may only manifest typical morphologies when the channelopathy is unmasked by infections, ischemia, drug use, or hypokalemia.

Resource Links: Life in the Fast Lane — great overview Dr. Steve Smith's Blog – good case

Created by Duncan Wilson, MD Edited by Nick Hartman, MD; Shanna Jones, MD; & Kristen Grabow Moore, MD, MEd

Triage EKG—Unit 3, Case 10



Unit 3, Case 10—Prolonged QT

What is your interpretation of the EKG?
History/Clinical Picture—Middle aged patient with syncope
Rate—54
Rhythm—Sinus bradycardia with a PVC
Axis—Left axis deviation. Positive in I, negative in aVF, and negative in II
P Waves—Normal morphology
Q/R/S Waves—Small non-pathologic Q waves in I, aVL. Normal R/S waves.
T Waves—Beat-to-beat decrease in T wave amplitude in all leads leading to inversions in II/III
U Waves—None apparent
PR Interval—Normal but borderline long, approximately 200ms.
QRS Width—Normal
ST Segment—No ST elevation or depression.
QT Interval—very prolonged with QTc almost 700

Diagnosis: Sinus bradycardia with markedly prolonged QT interval

If the heart rate is between 60 and 100 and the QT is greater than 50% of the R-R interval then the QTc is prolonged. For an exact number you would obviously still need to calculate a QTc however for quickly screeningif the QT is normal or prolonged this is an effective tool.

Discussion: Prolonged ventricular repolarization as manifested by a prolonged QT interval for malignant ventricular arrhythmias through the R on T phenomenon. The R on T phenomenon occurs when an ectopic beat occurs during the repolarization phase of a preceding impulse, leading to a sustained ventricular arrhythmia (classically Torsades des Pointes). A prolonged QT interval can be caused by:

1.) Congenital cardiac ion channel mutations - Na or K channelopathies

2.) Electrolyte imbalance - Hypokalemia, hypomagnesemia, hypocalcemia

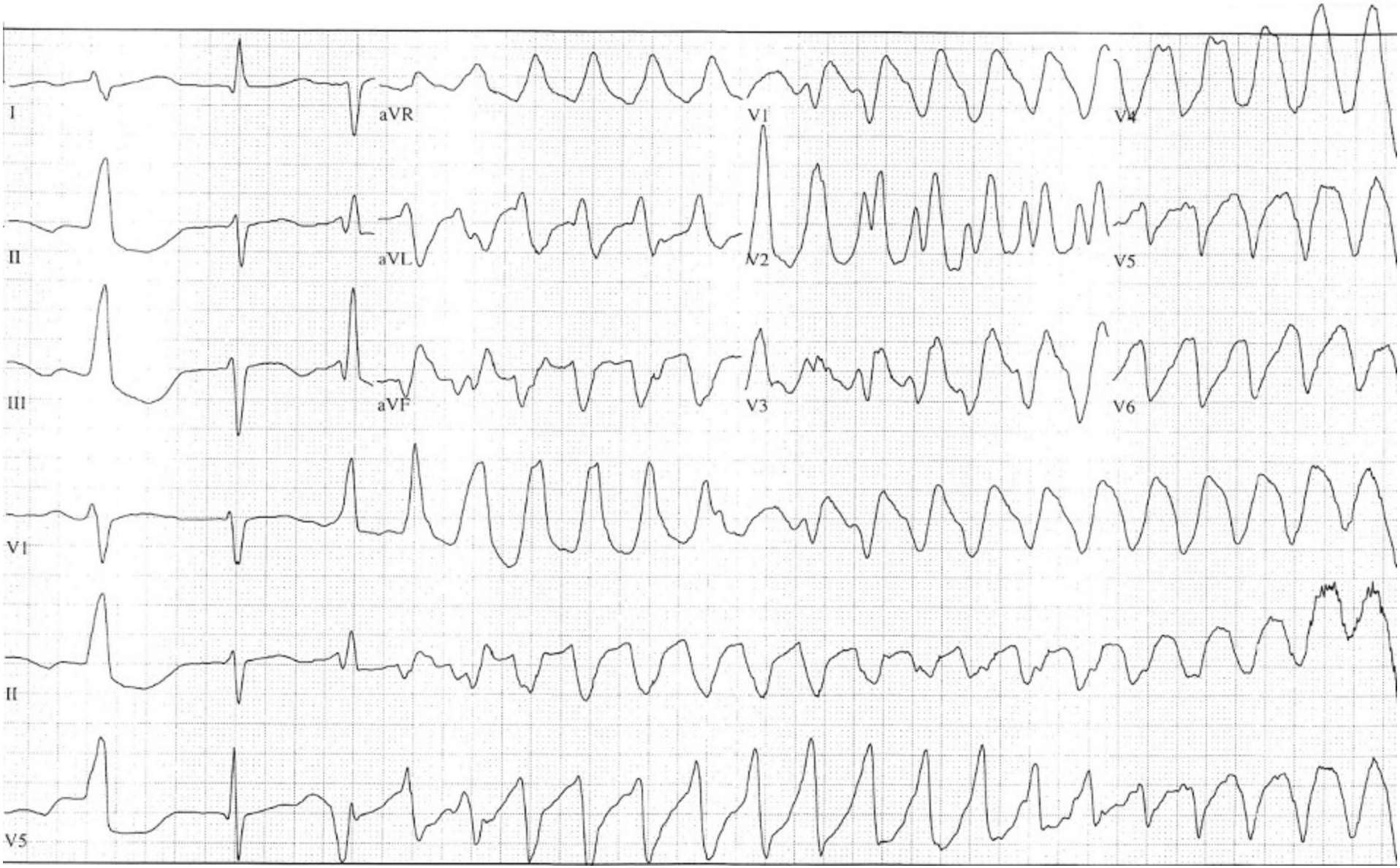
3.) Medications - amiodarone, TCAs, methadone, antibiotics, Zofran, antipsychotics, and many, many others

This patient is at risk of ventricular arrhythmia and should be admitted for tele monitoring and cardiology consult. In the event of Torsades, do the following: cardiovert or defibrillate as appropriate, give 2-4 grams $MgSO_4$ empirically, correct electrolyte abnormalities, consider isoproterenol to stimulate heart rate, and over drive pacing at a rate of 100 if the above measures fail.

Resource Links: Life in the Fast Lane — great overview

Unit 3, Case 10—Prolonged QT

This EKG shows a PVC followed by a normally conducted beat with a very long QT. Then an R on T event occurs and the rhythm transitions to polymorphic ventricular tachycardia (or more specifically Torsade de Pointes). Take a close look at the rhythm strips which clearly demonstrate the shifting axis.



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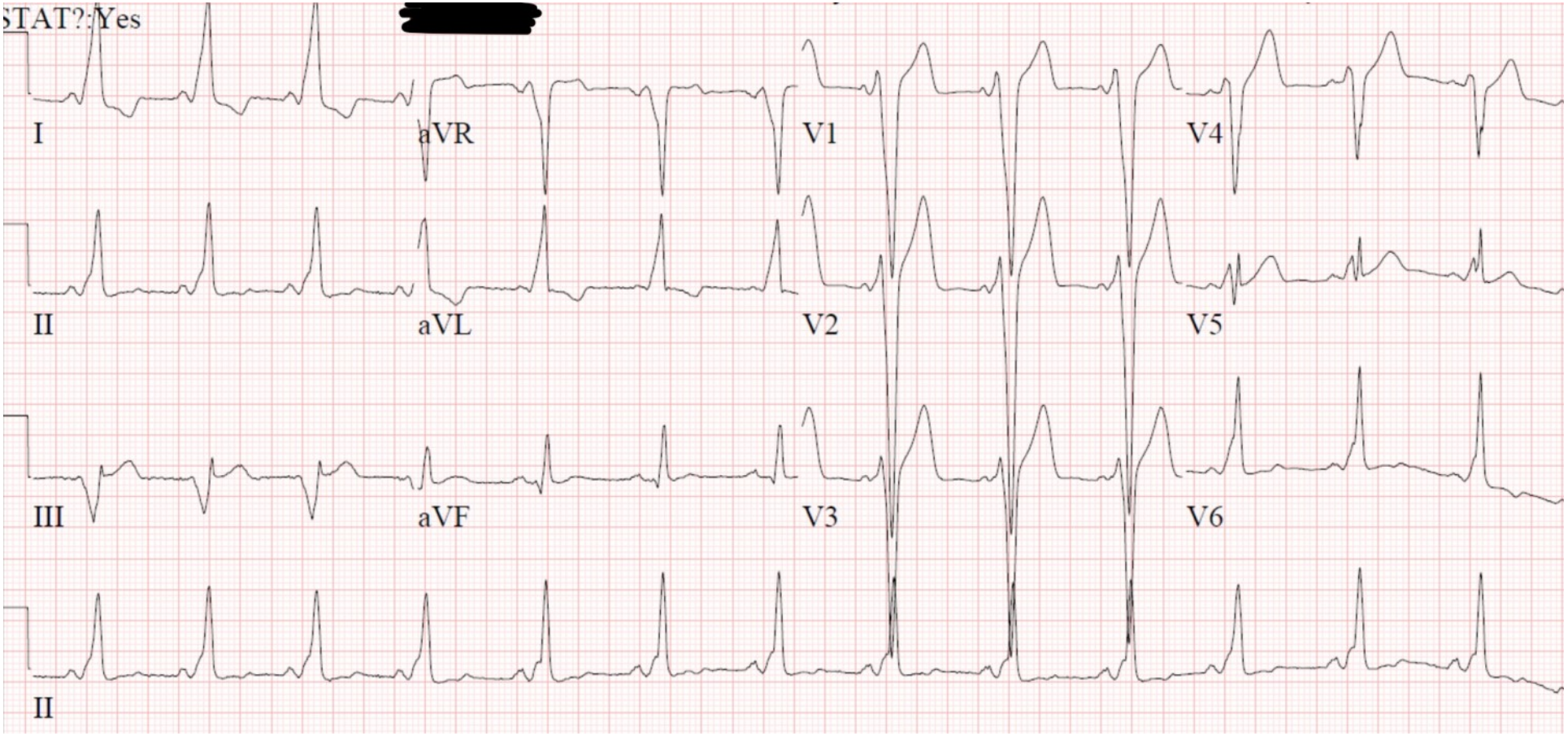
Triage EKG—Unit 3, Case 11



EKG courtesy of Shanna Jones, MD

Unit 3, Case 11—WPW

What is your interpretation of the EKG?
History/Clinical Picture— 20yoF presents with palpitations
Rate— ~78
Rhythm— sinus rhythm
Axis— normal
P Waves— present, no clear atrial enlargement
Q, R, S Waves— large S waves in I, II, aVL, and V6. Borderline Q wave in III. QRS complexes with sloping upstroke
T Waves— T-wave inversion I, aVL. T wave flattening in V6
U Waves— not present
PR Interval— narrow at just under 90 ms
QRS Width— wide at 160ms
ST Segment— slight depressions in I and aVL. STE in III, V1 & V2
QT Interval— prolonged with QTc ~500ms

Diagnosis: Sinus rhythm with pre-excitation (Wolff-Parkinson-White)

Discussion: The presence of a narrow PR-interval and sloping upstroke to the QRS complex suggests the presence of an accessory pathway. Symptomatic tachy-arrhythmias in the presence of an accessory pathway constitute the Wolff-Parkinson-White syndrome. An accessory pathway is an abnormal congenital electrical pathway that connects the atria to the ventricles, allowing supraventricular impulses to conduct to the ventricles without travelling through the AV-node. The presence of the bypass tract makes possible the development of re-entrant circuits (and re-entrant tachycardias) between the atria and ventricles. AV nodal re-entrant tachycardia (AVRT) is the name for the rhythm that occurs when a re-entrant impulse begins to self-propagate by travelling back to the atria via either the AV-node or the accessory pathway and triggering another round of depolarization.

Orthodromic AVRT occurs when the anterograde (first direction) conduction is through the AV-node, down the normal intraventricular conduction system, and the retrograde (second direction) conduction up the accessory (aka WPW) pathway. Because it utilizes the normal intraventricular conduction system, the QRS is narrow.

Antidromic AVRT is just the opposite (anterograde conduction down the accessory pathway, retrograde pathway up the AV node, wide QRS).

Resource Links: Life in the Fast Lane — great overview Dr. Steve Smith's Blog – brief (14 min) lecture

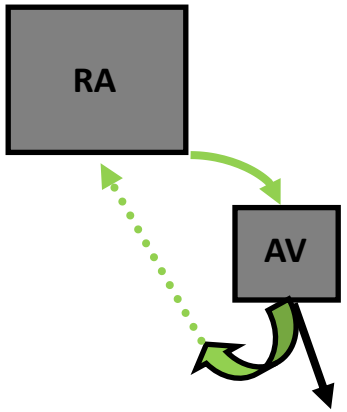
Unit 3, Case 11—WPW

Discussion: Atrioventricular Re-Entrant Tachycardia (AVRT) is a general descriptor for any rhythm that involves a self-propagating signal reciprocating between the atria and ventricles via the AV node and an accessory pathway. It is very important to realize that AVRT does not define the underlying rhythm. The normal AV nodal delay protects the ventricle from excessively fast stimulation however some accessory pathways are extremely fast and have essentially no delay. Considering the lack of delay it is easy to understand how **atrial fibrillation or atrial flutter with their 260-300bpm electrical signals can trigger ventricular fibrillation** when they are transmitted directly to the ventricle via the accessory pathway. Atrial fibrillation still causes an irregularly irregular rhythm in AVRT but you may have to look very closely because the rate is so fast. Atrial flutter is more difficult and may not be possible to identify prior to conversion. Procainamide should be used for irregular AVRT because it directly acts to slow the speed of the accessory pathway.

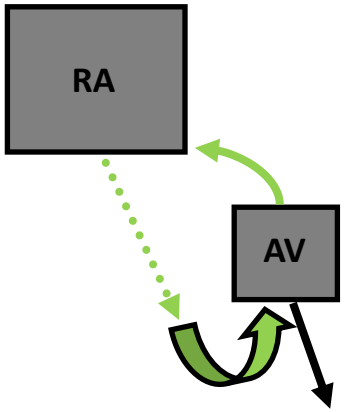
Orthodromic (narrow complex) AVRT can frequently be cardioverted with vagal maneuvers or adenosine because blocking the AV node also breaks the reciprocating circuit. Although AV-nodal agents are frequently effective cardioversion may be necessary and certainly required for any unstable patient.

Classically, AV-nodal blocking agents have been contraindicated in antidromic (wide complex) AVRT based on concern for possible conversion to VT. Some experts (and likely all textbooks) recommend treating it presumptively as VT by avoiding AV nodal blockers and instead using procainamide which preferentially slows the accessory pathway. The most recent AHA-ACC guidelines support the use of adenosine in patients who have monomorphic, regular, and hemodynamically stable wide complex rhythms. However, they note that if there is <u>any doubt</u> that the rhythm is VT then it is **safest to assume it is VT** and avoid the use of AV nodal blocking agents. Definitive therapy is catheter ablation of the accessory pathway.

AHA/ACC Guidelines (page E38)



Orthodromic AVRT: *Anterograde conduction through the AV node, retrograde conduction up accessory pathway (dashed line). <u>Narrow QRS</u>.*



Antidromic AVRT: *Retrograde conduction through the AV node, anterograde conduction up accessory pathway (dashed line). <u>Wide QRS</u>.*

<u>General Summary of AVRT</u>

Narrow Complex = Definitely okay to use AVNBs

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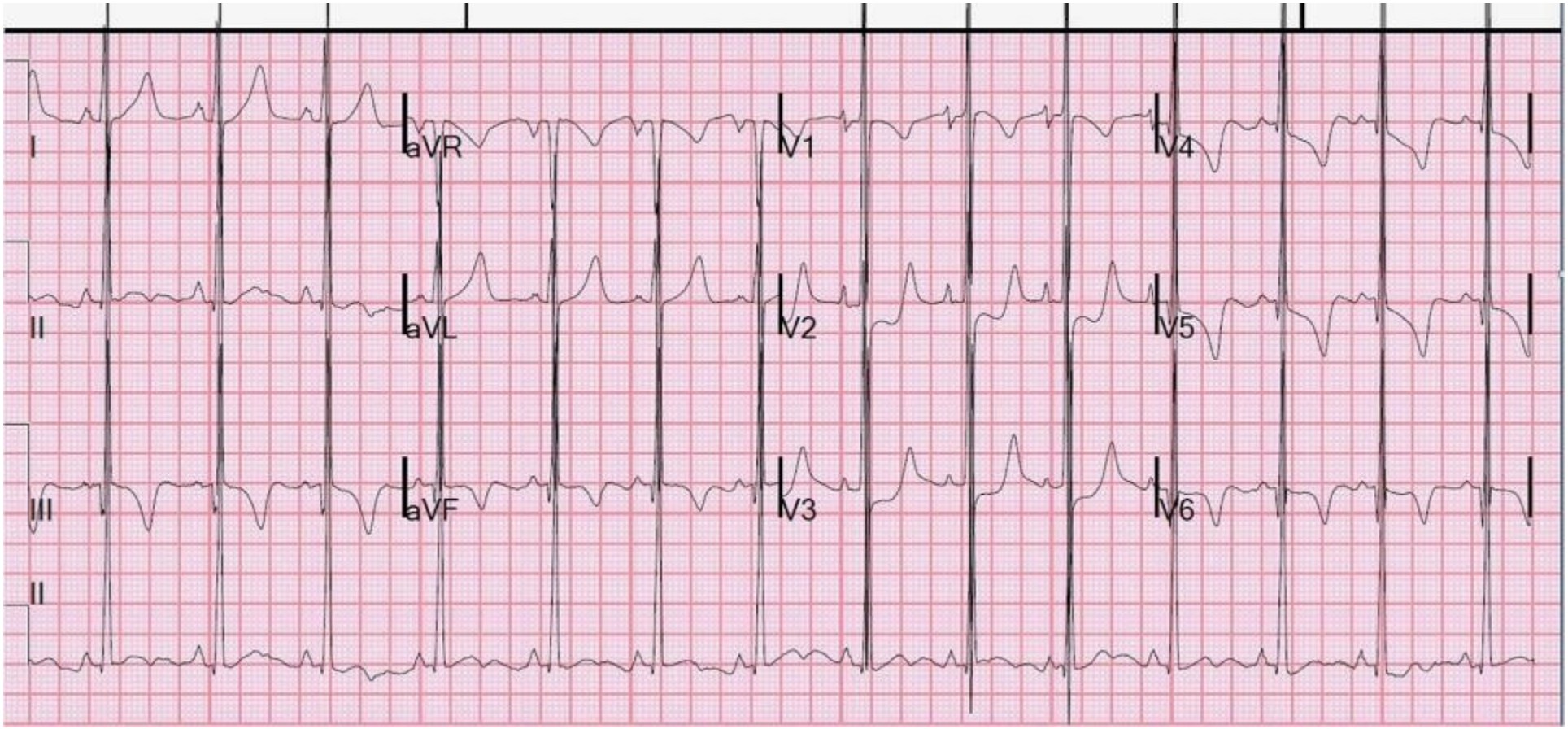
Triage EKG—Unit 3, Case 12



Courtesy of Steve Smith of [Dr. Smith's ECG Blog]

Unit 3, Case 12—LVH

What is your interpretation of the EKG?

History/Clinical Picture— adolescent female presents with exertional cardiac symptoms and a heart murmur

Rate— ~75

Rhythm— sinus rhythm

Axis— normal

P Waves— present

Q, R, S Waves— Characteristic Needle/Dagger-like Q waves. Massive R-wave voltage throughout, most prominent in the antero-septal leads, highly suggestive of asymmetric septal hypertrophy

T Waves— T-wave inversion infero-laterally

U Waves— not present

PR Interval— normal

QRS Width— normal

ST Segment— STD V2-6

QT Interval— normal

Diagnosis: Massive LVH highly suggestive of Hypertrophic Cardiomyopathy

Discussion: Hypertrophic Cardiomyopathy (HCM) is a common inherited disorder (affects 1 in 500 people) that classically causes sudden death in young athletes. Mutations in genes coding for sarcomeric proteins lead to asymmetric and disorganized LVH. Asymmetric thickening of the interventricular septum can lead to dynamic left ventricular outflow tract obstruction (LVOTO). LVOTO occurs when rapid ventricular rates and/or reduced preload lead to reduced left ventricular end diastolic volumes, thereby bringing the anterior mitral valve leaflet closer to the septum. If the anterior mitral valve leaflet moves too close to the intraventricular septum it can become trapped against the septum, blocking blood flow through the LVOT. Patients are at high risk for ventricular dysrhythmia due to disorganized myocyte architecture. Patients may present with syncope (due to LVOTO or ventricular dysrhythmia), pulmonary congestion, or angina (due to increased demand from the hypertrophied ventricle). Classic ECG findings include massive LVH, deep anterior T-wave inversions, and deep, narrow, "dagger-like" q-waves in the interior and anterior leads. Treatment includes minimizing LVOTO with beta-blockers and adequate hydration. ICD placement, surgical myomectomy, septal ablation, or heart transplant may be necessary in severe cases. A new diagnosis such as this requires admission to telemetry with formal echocardiography and cardiology consultation.

Resource Links: Life in the Fast Lane — great overview Dr. Steve Smith's Blog – good case

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