Foundations EKG I

Unit 6 Instructor—Approach to Wide Complex Tachyarrythmias

Timeline:

- Divide learners into 4 groups at different tables (*this approach is suggested for groups of 8 or more learners and should be modified to 1 or 2 groups so sites with lower numbers of learners*)
- 5 min large group review of the *Unit 6 Summary "Approach to Wide Complex Tachyarrythmias"*
- Give each group 2 copies of the *Unit 6 EKG Challenges Packet* (merged challenge EKG content for EKGs 21 -24), this allows learner groups to all review content and record their group's answers to the interpretation and questions for each EKG
- Allow 20 minutes for groups to complete the 4 challenges (give updates at 5min increments)
- 20 minutes large group discussion of answers to challenges. May consider asking each group to present their responses to a different EKG

Meeting Resources:

- Notify learners in advance of the session that they need to review the unit summary and challenge EKGs
- Before the session, have a few copies of the unit summary (pages 2-5 of this document) printed to give to learners who forgot their copies/devices and copies of the *Unit 6 EKG Challenges Packet* to give to groups
- Before the session, make sure to print this document for your own reference during the group discussion
- After the meeting, send out the answer document to learners for independent review

Foundations EKG I - Unit 6

Approach to Wide Complex Tachyarrhythmias

Unit 6, Case 21—*68 y/o M with PMHx of DM2, HTN brought to urgent care after syncopal episode at home. EKG completed and transferred to the ED. Patient mumbles/ withdraws to noxious stimuli.*

How would you manage this patient?

HR: 140	BP: 70/40
RR: 22	O2 Sat: 97%

Unit 6, Case 22—*33 y/o M with no PMH presents for palpitations and pre-syncope that started acutely 30 minutes ago.*

What underlying cardiac condition does this patient have?
What are your management options for this situation?
What medications are contraindicated in this situation?

HR: 140	BP: 100/50
RR: 20	O2 Sat: 97%

Unit 6, Case 23—*23 y/o M with a PMH of muscular dystrophy undergoes intubation for respiratory failure. The following EKG is taken immediately after successful induction and intubation.*

How would you manage this in the ED?
What medication likely precipitated this situation?

HR: 70	BP: 90/50
RR: 12	O2 Sat: 100%

Unit 6, Case 24—*27 y/o F with unknown history presents via ambulance after a worried friend called 911 for suicidality. Denies complaints on arrival. Patient became unresponsive while ED triage EKG completed.*

What aspects of the EKG changed significantly when it was repeated?
What are possible causes of this dysrhythmia?
What complications should you watch for?
What treatment options do you have for this patient?

HR: 70	BP: 80/40
RR: 18	O2 Sat: 97%

Foundations EKG I - Unit 6 Summary

Approach to Wide Complex Tachyarrhythmias

Wide QRS complex tachycardia should be thought of as **ventricular tachycardia** until proven otherwise. The first task in evaluating a wide complex tachycardia is to determine whether the patient is hemodynamically stable or unstable. **Unstable patients need to be immediately electrically cardioverted.**

In addition to ventricular tachycardia, wide QRS complexes may indicate a supraventricular rhythm with an aberrant pathway.

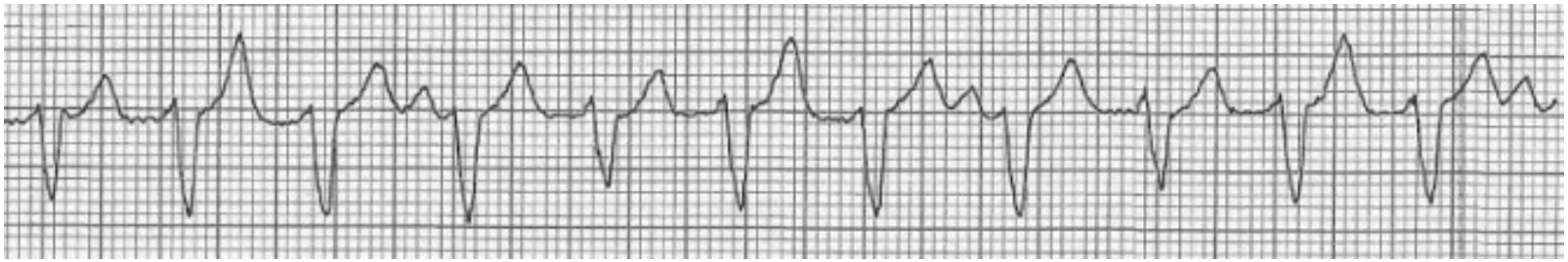
	Regular	**Irregular**
Narrow	Sinus tachycardia AVNRT Orthodromic AVRT 2:1 atrial flutter	Atrial fibrillation MAT
Wide	Ventricular tachycardia Antidromic AVRT	Atrial fibrillation with aberrancy

Ventricular tachycardia is likely if there is:

- AV dissociation
- QRS over 140s
- Positive precordial QRS complexes

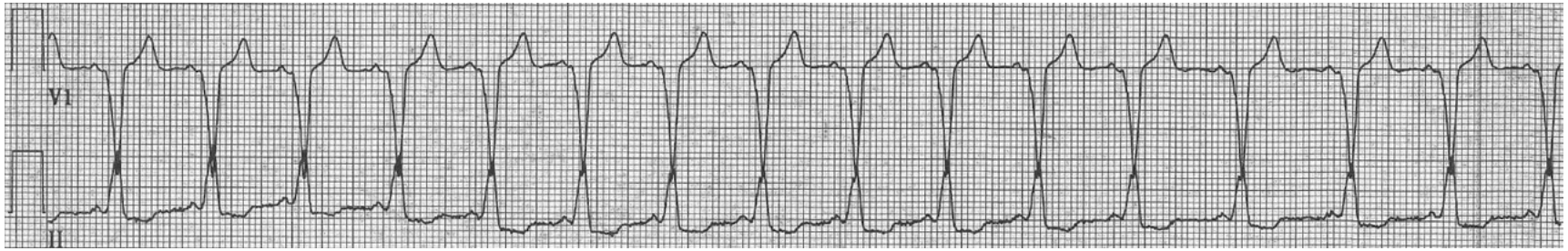
Sustained ventricular tachycardia lasts over 30 seconds.

Treatment of hemodynamically **stable** ventricular tachycardia may include administration of lidocaine, procainamide, or amiodarone. It is important to consider reversible causes (commonly referred to as the "Hs and Ts"). Unstable ventricular tachycardia mandates immediate synchronized cardioversion.



SVT with aberrant conduction pathways may also cause wide complex tachycardia. Conduction of AVRT may be orthodromic (traveling down the normal direction of the nerve fibers) or antidromic (opposite to the normal conduction pathway). Antidromic conduction tends to cause a wide QRS complex whereas orthodromic often causes a narrow complex .

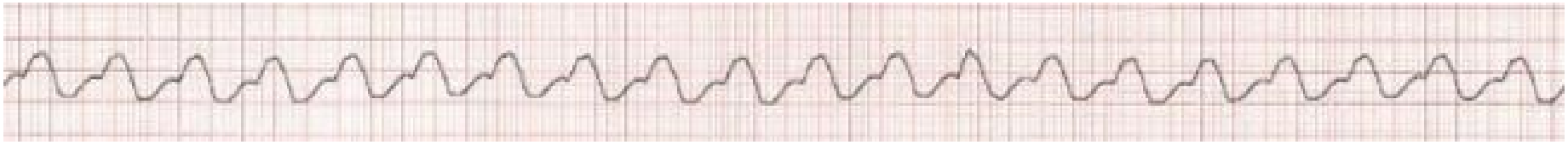
Wolff-Parkinson-White (WPW) is a pre-excitation syndrome. Evidence of the accessory pathway causing tachycardia can be seen in the delta wave causing upslurring to the QRS with a short PR interval. Other clues that an EKG shows WPW include an irregularly irregular rhythm, changing QRS morphologies, and very rapid heart rate (even 250-300bpm). Management includes synchronized electrical cardioversion or chemical cardioversion with procainamide. **Blocking of the AV node is contraindicated** in this case because it forces even more conduction down the accessory pathway. Atrial fibrillation with a wide QRS complex should prompt you to think about WPW.



Sodium channel blockage, often due to **TCA overdose,** can also cause a wide QRS complex. Sodium channel blockade on EKG may show:

- QRS greater than 100ms in Lead II
- Terminal R wave greater than 3mm in aVR
- R/S ratio greater than 0.7 in aVR

Treatment includes sodium bicarbonate 1-2mmol/kg every 3 minutes until QRS starts to narrow and blood pressure parameters improve.



Hyperkalemia can cause widening of the QRS as well as tachycardia. Peaked T-waves and possible loss of P-waves in a patient with an appropriate story should prompt consideration and rapid treatment of hyperkalemia. Treatment may include: calcium administration, insulin/dextrose, albuterol, sodium bicarbonate, kayexalate, or dialysis.

Foundations EKG I - Unit 6, Case 21

68yM with PMHx of DM2, HTN brought to urgent care after syncopal episode at home. EKG completed and transferred to the ED. Patient mumbles/withdraws to noxious stimuli.

HR: 140	BP: 70/40
RR: 22	O2 Sat: 97%

What is your interpretation of the EKG?

History/Clinical Picture

Rate

Rhythm

Axis

P Waves

Q/R/S Waves

T Waves

U Waves

PR Interval

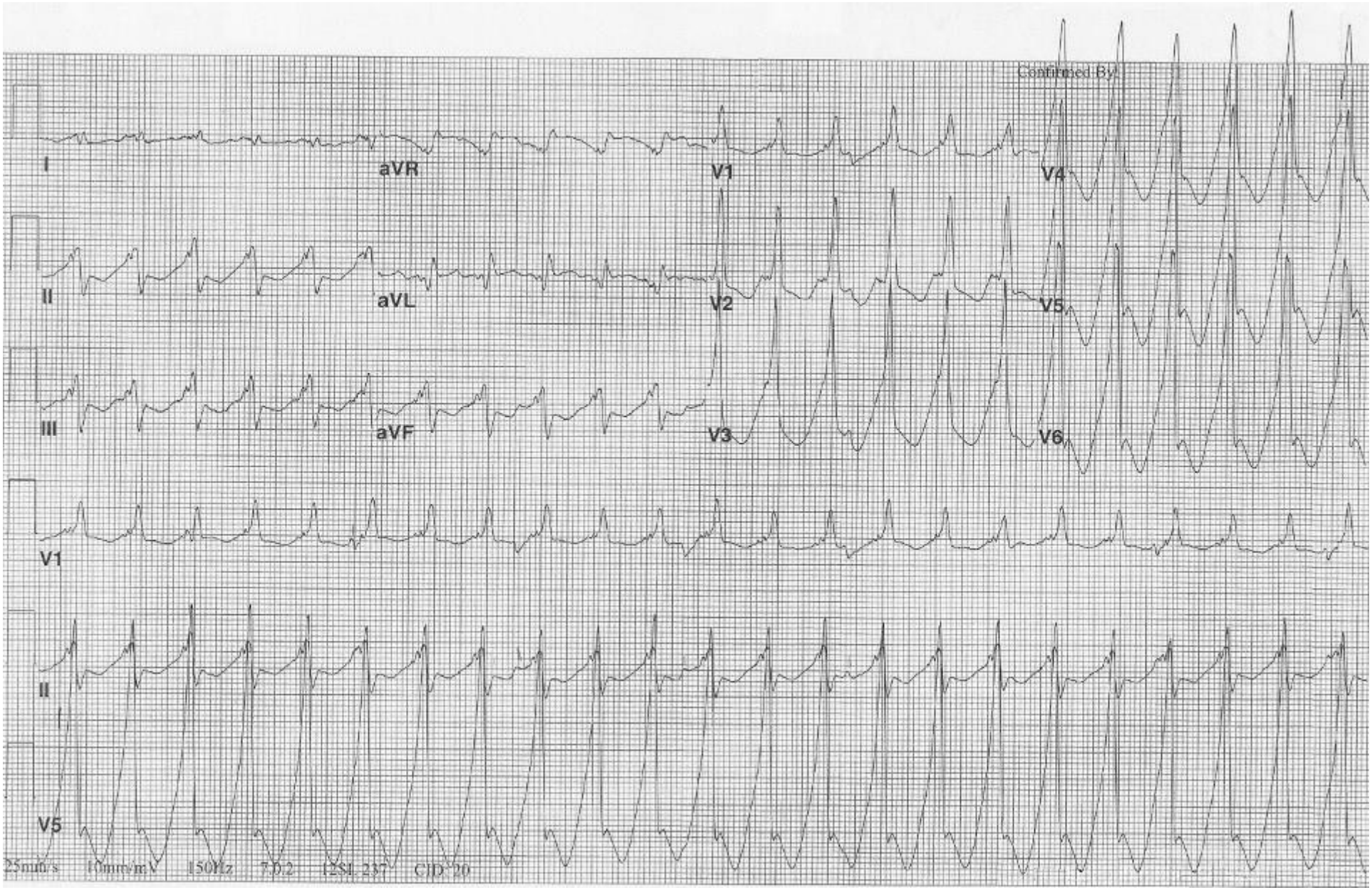
QRS Width

ST Segment

QT Interval

How would you manage this patient?

Triage EKG—Unit 6, Case 21



Unit 6, Case 21—Ventricular Tachycardia

What is your interpretation of the EKG?

History/Clinical Picture—Elderly male with cardiac risk factors presenting with syncope and hemodynamic instability

Rate—140s

Rhythm—Wide complex regular tachycardia.

Axis—Left axis

P Waves—P waves marked after the 8th, 11th, and 14th QRS complexes

Q, R, S Waves—? Q waves in aVL. RBBB morphology with QRS > 140ms

T Waves—Unable to assess given rate and QRS aberrancy

U Waves—Unable to assess given rate and QRS aberrancy

PR Interval—No consistent PR interval

QRS Width—Wide

ST Segment—Unable to assess given rate and QRS aberrancy

QT Interval—Unable to assess given rate and QRS aberrancy

Diagnosis—Likely Monomorphic Ventricular Tachycardia given the following:

AV dissociation (see P waves marked after the 8th, 11th, and 14th QRS complexes)

QRS > 140ms

Resource Links: Life in the Fast Lane Dr. Steve Smith's Blog

Positive concordance (all precordial QRS complexes positive—*although rarely this is seen in WPW*).

Unit 6, Case 21—Ventricular Tachycardia

Management of Wide Complex Tachycardia

Assess Hemodynamic Stability

- Hypotensive?
- Altered Mental Status?
- Chest Pain?
- Acute CHF Exacerbation?
- History of Long QT?
- Signs of Shock?
- Rate > 150?
- Polymorphic?

NO →

YES ↓

Interventions

1. **Defibrillator pads. Synchronized cardioversion if patient becomes unstable**
2. Consider Lidocaine (1mg/kg)
3. Procainamide (50mg/min) or Amiodarone (150mg over 10 min)
4. Consider reversible causes →
5. Replete Magnesium, Potassium, and Calcium as needed

- Hypoglycemia
- Hypo/Hyperkalemia
- Hypoxia
- Hypothermia
- Hypovolemia
- Acidosis
- Toxins
- Tamponade
- Tension Pneumo
- Trauma
- MI
- PE

1. IMMEDIATE SYNCHRONIZED CARDIOVERSION

2. Proceed down interventions pathway

Created by William Burns, MD Edited by Nick Hartman, MD & Kristen Grabow Moore, MD, MEd

Foundations EKG I - Unit 6, Case 22

33yoM with no PMH presents for palpitations and pre-syncope that started acutely 30 minutes ago.

HR: 140	BP: 100/50
RR: 20	O2 Sat: 97%

What is your interpretation of the EKG?

History/Clinical Picture

Rate

Rhythm

Axis

P Waves

Q/R/S Waves

T Waves

U Waves

PR Interval

QRS Width

ST Segment

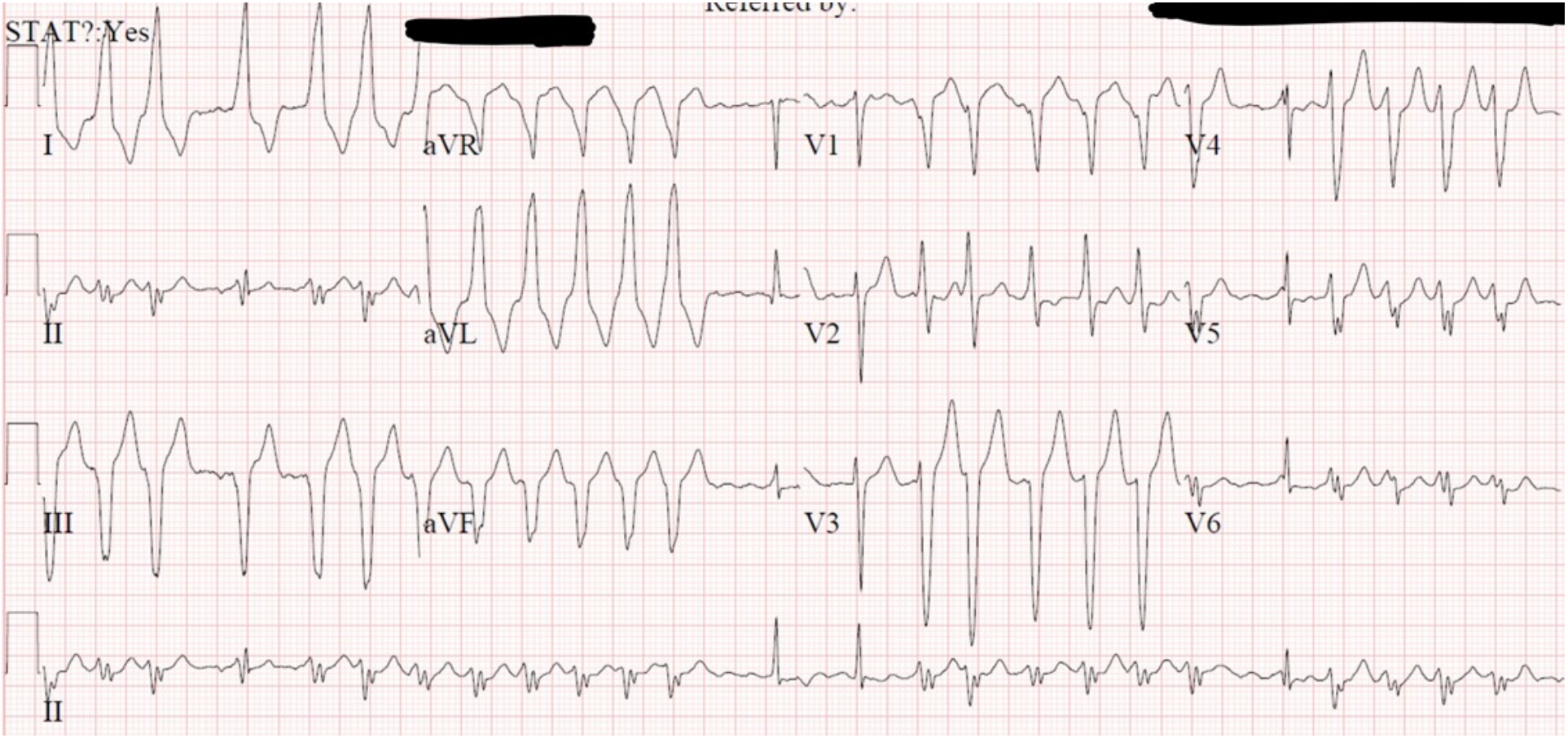
QT Interval

What underlying cardiac condition does this patient have?

What are your management options for this situation?

What medications are contraindicated in this situation?

Triage EKG—Unit 6, Case 22



EKG courtesy of Shanna Jones, MD

Unit 6, Case 22—Afib with RVR & WPW

What is your interpretation of the EKG?

History/Clinical Picture—young man with symptomatic irregular, wide complex tachycardia & borderline unstable vitals
Rate— ~150
Rhythm— atrial fibrillation
Axis— left axis deviation
P Waves— absent
Q, R, S Waves— inferior q waves, poor R-wave progression
T Waves— lateral TWI (I and aVL)
U Waves— not present
PR Interval— not applicable
QRS Width— wide, around 140 ms
ST Segment— depression laterally (V5, V6, I, aVL)
QT Interval— prolonged ~530
Diagnosis: Atrial Fibrillation with RVR in the setting of an accessory pathway (i.e. Wolff Parkinson White)

Discussion: It is critical that you recognize this rhythm even if the patient does not tell you that they have WPW. An irregularly irregular rhythm, changing QRS morphologies, and a very rapid rate are the hallmarks. Delta waves may also be seen. The ventricular rate at times is in the 250-300 range. This is faster than the AV node can conduct, and it implies the presence of an accessory pathway.

What are your management options for this situation?

Appropriate management options include synchronized cardioversion or chemical cardioversion with procainamide.

What medications are contraindicated in this situation?

Beta-blockers, calcium channel blockers, adenosine, and amiodarone are all *incorrect* choices as AV-nodal blockade can lead to preferential conduction down the accessory pathway with subsequent hemodynamic collapse, often from ventricular fibrillation.

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 & Kristen Grabow Moore, MD, MEd

Foundations EKG I - Unit 6, Case 23

55 y/o F with a PMH of myasthenia gravis undergoes intubation for respiratory failure. The following EKG is taken immediately after successful induction and intubation.

HR: 70	BP: 90/50
RR: 12	O2 Sat: 100%

What is your interpretation of the EKG?

History/Clinical Picture

Rate

Rhythm

Axis

P Waves

Q/R/S Waves

T Waves

U Waves

PR Interval

QRS Width

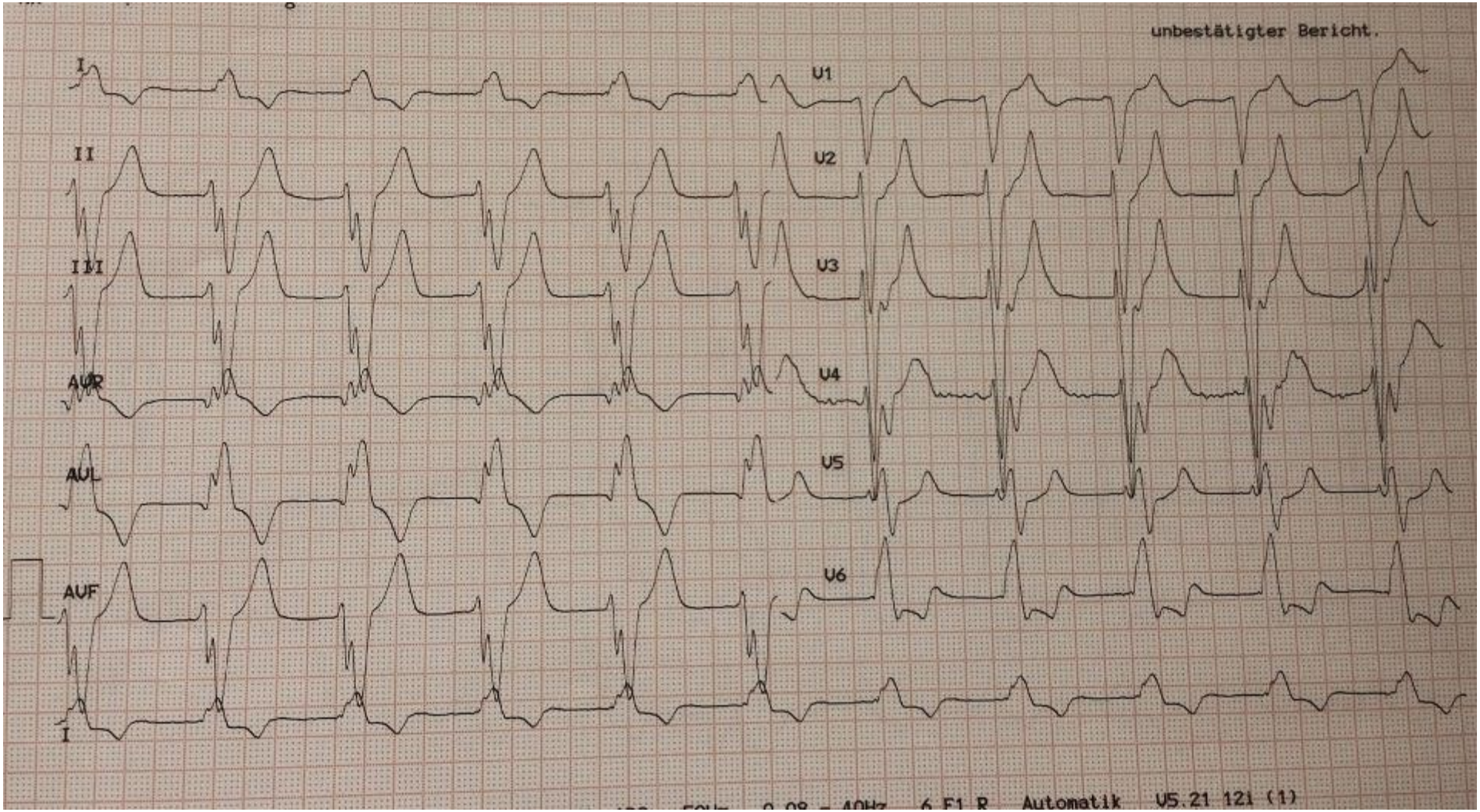
ST Segment

QT Interval

How would you manage this in the ED?

What medication likely precipitated this situation?

Triage EKG—Unit 6, Case 23



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

Unit 6, Case 23—Hyperkalemia

What is your interpretation of the EKG?

History/Clinical Picture— A patient with myasthenia gravis with a bizarre wide complex rhythm

Rate— 60-70

Rhythm— lack of p-waves suggest the origin of this rhythm is either junctional or ventricular, or a sinus rhythm deranged by electrolyte imbalance (which is the case here)

Axis— left axis deviation

P Waves— not present

Q, R, S Waves— bizarre wide R and S waves, some with notching, throughout

T Waves— very peaked, most notable in V2 and V3

U Waves— not seen

PR Interval— not applicable

QRS Width— very wide and bizarre, just under 200ms

ST Segment— discordant STD in V5/6

QT Interval— appears grossly normal

Diagnosis: Hyperkalemia

Discussion: This patient has acute hyperkalemia after induction with succinylcholine in the setting of neuromuscular junction disease, in this case myasthenia gravis. EKG findings suggestive of hyperkalemia that are seen in this EKG include: peaked T-waves, prolonged QRS duration with bizarre morphology, and disappearance of p-waves. The potassium level returned at 6.9 and the EKG changes resolved with treatment for hyper K. Treatment should include: stabilization of cardiac membrane potential with calcium gluconate or calcium chloride, shifting potassium intracellularly with some combination of insulin/dextrose, albuterol, sodium bicarb, and enhanced clearance with kayexelate, loop diuretics, or renal replacement therapy.

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 & Kristen Grabow Moore, MD, MEd

Foundations EKG I - Unit 6, Case 24

27 year-old woman with unknown history presents via ambulance after a worried friend called 911 for suicidality. Denies complaints on arrival. Patient became unresponsive while ED triage EKG completed.

HR: 70	BP: 80/40
RR: 18	O2 Sat: 97%

What is your interpretation of the Triage EKG?

History/Clinical Picture

Rate

Rhythm

Axis

P Waves

Q/R/S Waves

T Waves

U Waves

PR Interval

QRS Width

ST Segment

QT Interval

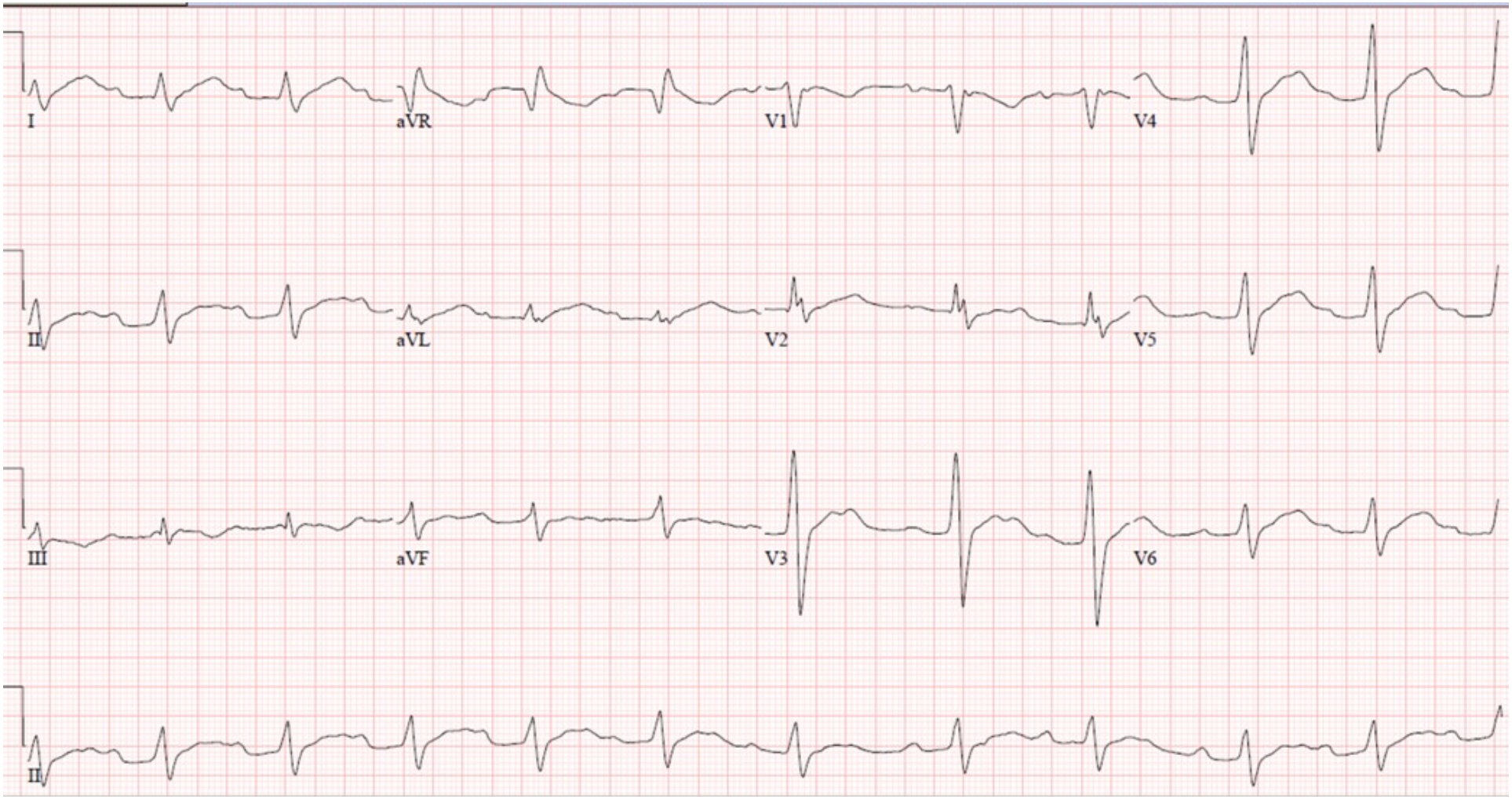
What aspects of the EKG changed significantly when it was repeated?

What are possible causes of this dysrhythmia?

What complications should you watch for?

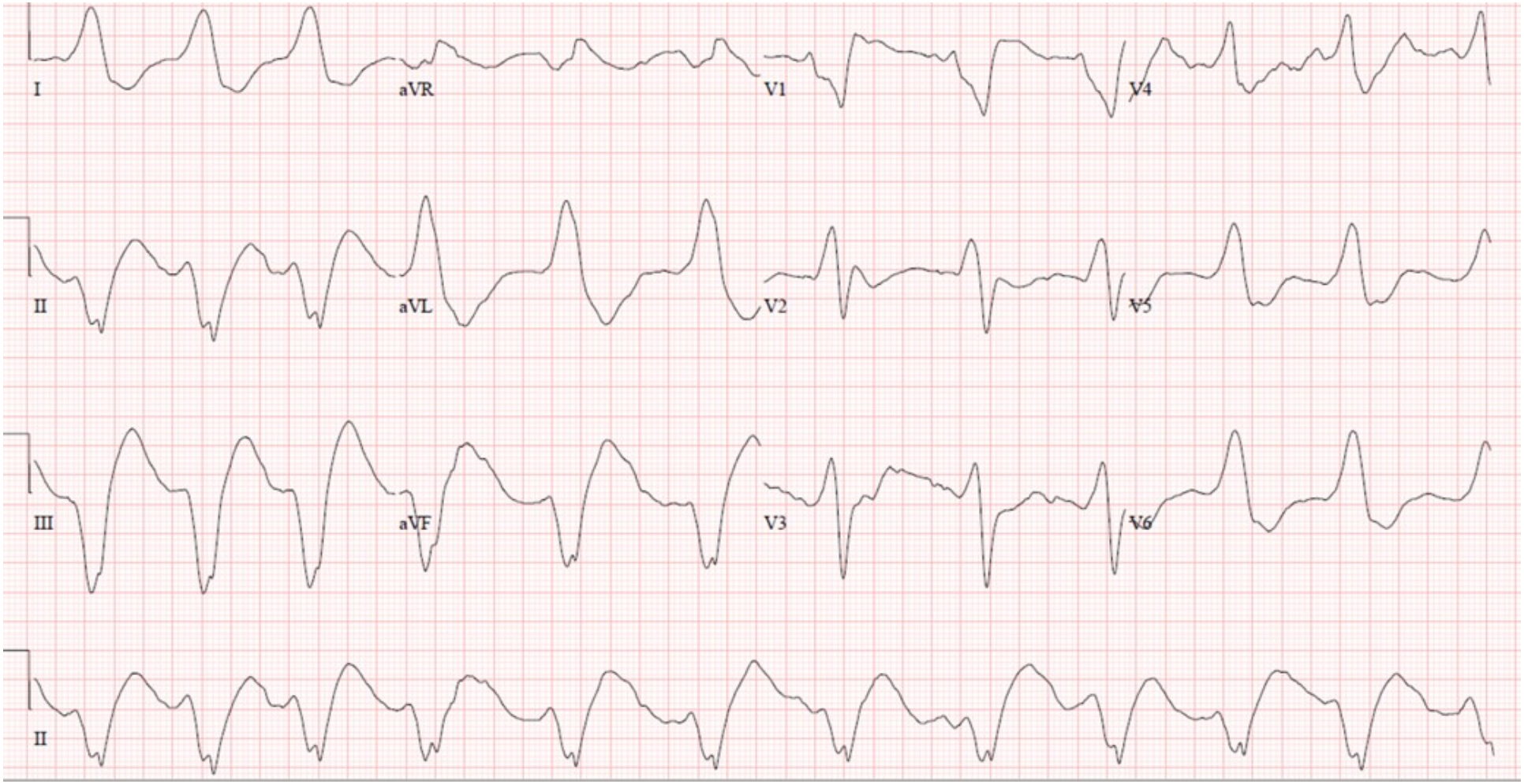
What treatment options do you have for this patient?

Pre-Hospital EMS EKG—Unit 6, Case 24



EKG courtesy of Shanna Jones, MD

Triage EKG—Unit 6, Case 24

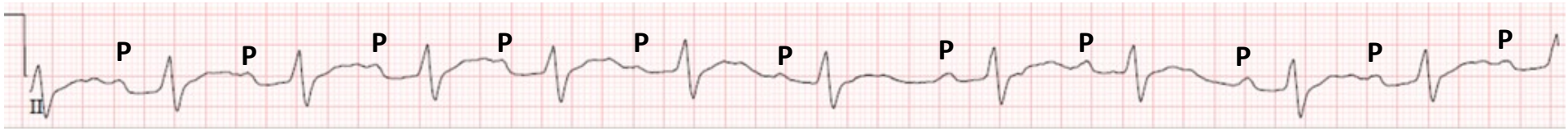


EKG courtesy of Shanna Jones, MD

Unit 6, Case 24—Sodium Channel Blockade

What is your interpretation of the Pre-Hospital EMS EKG?

History/Clinical Picture—young, potentially suicidal woman with hypotension, altered mental status
Rate— 66
Rhythm— sinus rhythm
Axis—normal axis
P Waves—present



Q, R, S Waves—Q wave in aVR, tall R wave in aVR, S waves in the inferior (II, II, aVF), anterior (V2, V3, V4), and lateral (V5, V6, I,) leads
T Waves—abnormal morphology - enlarged in the anterolateral leads with a biphasic component likely incorporating a U wave, flattening/inversion in III, flattening in aVF
U Waves—present, biphasic component of the T wave most obvious in V3 and V4, inverted T in III likely a terminal U wave
PR Interval—prolonged ~268
QRS Width—wide ~168ms
ST Segment— somewhat difficult to assess but no obvious elevation or depression
QT Interval— prolonged ~519

Diagnosis—Sinus Rhythm with conduction delay, prolonged QRS and QT concerning for TCA overdose

Physiology: Sodium channel blockade leading to interventricular conduction delay

ECG Characteristics of Na Channel or TCA Overdose

QRS > 100 in Lead II, Terminal R > 3mm in aVR, R/S Ratio > 0.7 in aVR

Unit 6, Case 24—Sodium Channel Blockade

What is your interpretation of the Triage EKG?

History/Clinical Picture—young, potentially suicidal woman with hypotension, altered mental status
Rate— 70
Rhythm— wide complex, irregular
Axis—left axis
P Waves— unclear if present, possibly buried in the T waves
Q, R, S Waves—q waves in II, II, and aVF, improved R wave progression in the precordial leads, tall R wave in aVR
T Waves—Abnormal morphology given increased size diffusely and particularly slurred appearance in aVR and V1
U Waves—None apparent but very limited assessment given abnormal T waves
PR Interval—not applicable
QRS Width—wide ~182ms
ST Segment—very difficult to assess given abnormal T wave morphology
QT Interval—prolonged ~632

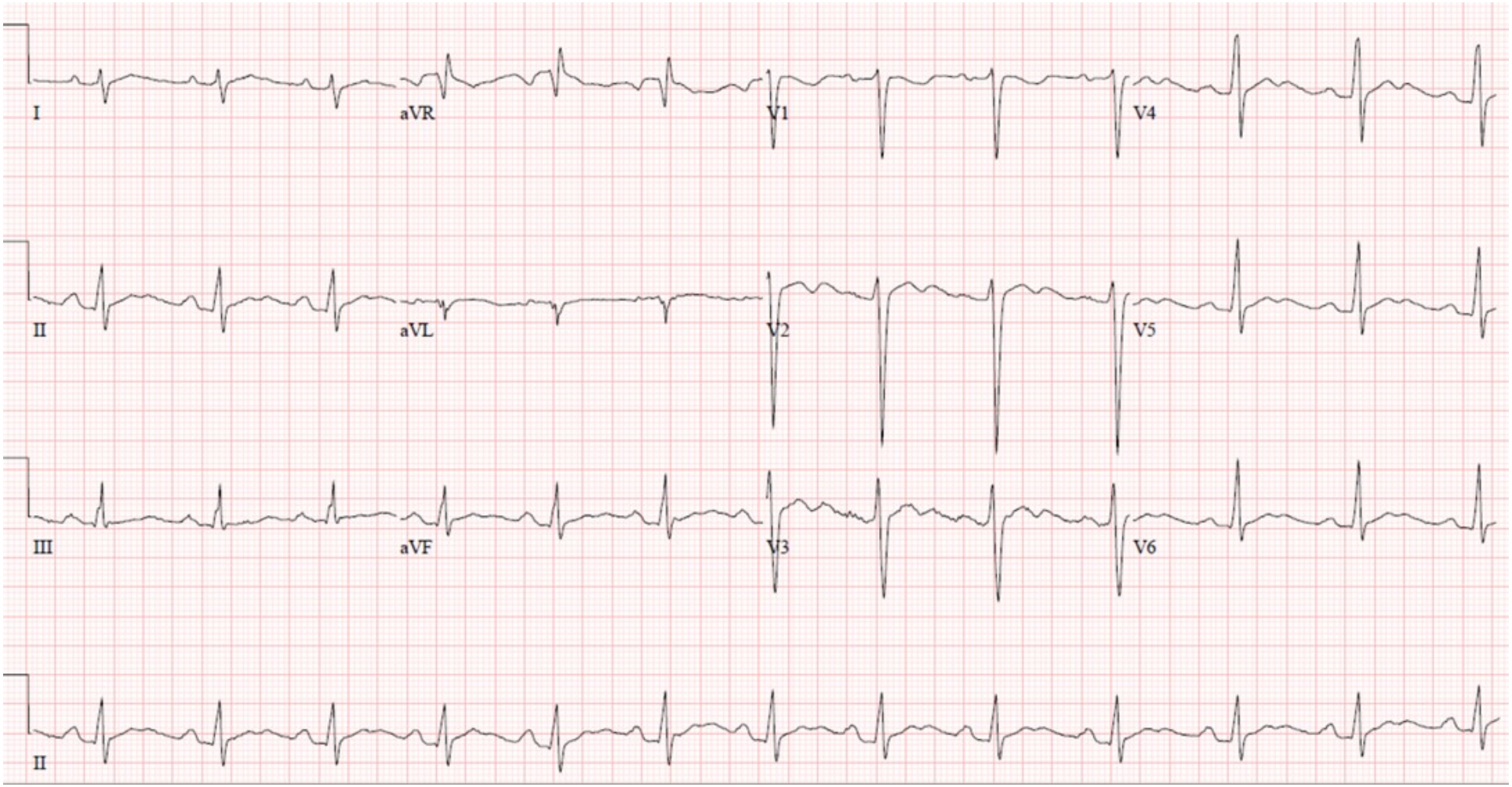
Diagnosis—Severe Interventricular Conduction Delay consistent with Sodium Channel Blockade consistent with TCA Overdose

ECG Characteristics of Na Channel or TCA Overdose
QRS > 100 in Lead II, Terminal R > 3mm in aVR, R/S Ratio > 0.7 in aVR

Resource Links: Life in the Fast Lane Dr. Steve Smith's Blog

Unit 6, Case 24—Sodium Channel Blockade

Repeat EKG the next day after treatment with sodium bicarbonate



EKG courtesy of Shanna Jones, MD

Unit 6, Case 24—Sodium Channel Blockade

What aspects of the EKG changed significantly when it was repeated?

1. The QRS widened dramatically

What are possible causes of this dysrhythmia?

Tricyclic Antidepressants (Elavil, Doxepin, __triptylines, __ipramine)

Antiarrythmics (Procainamide, Flecainide, Encainamide, Amiodarone)

Local Anesthetics (Bupivacaine, Ropivacaine)

Antimalarials (Chloroquine, Hydroxychloroquine)

Propanolol

Carbamazepine

What complications should you watch for?

QRS > 100 predictive of seizures

QRS > 160 predictive of VT/VF

What treatment options do you have for this patient?

Sodium bicarb (1-2 mg/kg) q3-5 minutes until BP improves and QRS begins to narrow

Support BP with boluses and pressors as needed

Created by William Burns, MD Edited by Nick Hartman, MD & Kristen Grabow Moore, MD, MEd