WOLFF-PARKINSON-WHITE SYNDROME
A case for high-fidelity simulation in emergency medicine

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ABSTRACT
This educational resource provides the information and materials for a high-fidelity simulation case suitable for resident physicians in emergency medicine. This case is currently in use at our institution for upper-level emergency medicine residents completing required educational time in our Emergency Department Simulation Program. This case has been used for the past three years and has recently been edited and expanded to its existing form.

This high-fidelity patient simulation case involves a young man with palpitations and mild shortness of breath. An ECG in the emergency department demonstrates a wide complex tachycardia, and a review of old records finds a previous ECG with findings diagnostic for Wolff-Parkinson-White (WPW) syndrome. Inappropriate blockage of the AV node with pharmacologic agents will lead to patient deterioration to ventricular fibrillation, as this patient with WPW is in atrial fibrillation. Appropriate treatment with procainamide or amiodarone will allow stabilization of the tachycardia. Debriefing materials are provided to illustrate and stimulate discussion of the important concepts for diagnosing and treating patients with WPW.
I. **Title of Case:** Wolff-Parkinson-White Syndrome

II. **Target Audience:** Resident physicians.

III. **Learning Objectives:**

   A. **Primary Learning Objectives**
      1. Demonstrate appropriate initial approach for a patient with palpitations and shortness of breath.
      2. Identify irregular wide-complex tachycardia on ECG.
      3. List the diagnostic features of WPW on ECG.
      4. Understand the treatment options of atrial fibrillation in patients with WPW.

   B. **Secondary Goals**
      1. Differentiate between different causes of wide-complex tachycardia.
      2. Understand the pathophysiology of the accessory pathway in WPW.
      3. Describe the contraindications for AV nodal blocking agents.

   C. **Critical Actions**
      1. Obtain appropriate history of present illness from patient.
      2. Complete pertinent physical examination and identify irregular tachycardia.
      3. Order ECG.
      4. Identify wide-complex tachycardia on ECG.
      5. Avoid the use of AV nodal blocking agents.
      6. Use procainamide or amiodarone to control rate.
      7. Arrange appropriate cardiology consultation.

IV. **Environment:**

   A. **Lab Set up** – Emergency Department bed #5 in simulation laboratory.

   B. **Manikin Set up** – The manikin will be positioned in the bed in a head-raised position, with street clothes on. At the beginning of the simulation, there are no monitor leads on the patient, no IV has been placed, and patient is not on oxygen.

   C. **Props** – Available for use will be a heart monitor with leads, blood pressure cuff, and pulse oximeter. Supplemental oxygen by nasal cannula and non-rebreather will be available. There will be a medicine cart or tray with a full complement of vasoactive agents, ACLS medications, medicines necessary for sedation, rapid sequence intubation, and analgesia. In addition, a fully stocked code cart with defibrillator will be available for use, along with a selection of direct laryngoscopes and intubation supplies.
D. Audiovisual – (See Appendix A) Available for review when asked for by participants will be a normal chest radiograph, an electrocardiogram showing a wide complex irregular tachycardia, and an “old” electrocardiogram showing narrow complex regular rhythm with a short PR interval and delta wave. Normal values are available for any laboratory studies that are ordered.

E. Distractors – Distractors can be added at the director’s discretion. However, as written, this is the only patient that needs to be cared for and there will be no extraneous inputs to distract the participants’ attention from the case at hand.

V. ACTORS:

A. Roles can vary depending on the number of participants in the simulation session and on potential actor availability. This case can be implemented using the minimum of one participant and one facilitator/operator who can provide oral feedback via overhead audio and play additional roles via voice only. Realism can be enhanced by using physical actors, such as nursing staff members, but these are not required to successfully implement this case.

B. Roles may be played by resident or faculty physicians, nurses, or medical students.

C. Actions for the roles will be as follows:
   a. Primary physician - The main scenario participant will act as the primary physician and do the primary evaluation of the patient to include obtaining a history, conducting a physical exam, and ordering an ECG, chest X-ray, and any necessary medications or other interventions. The primary physician can perform any needed procedures, or can delegate these to other physicians.
   b. Secondary physicians - Other participants in the scenario will serve as collaborators, assistants for any necessary procedures, and consultants.
   c. Nursing staff - The role of the nurse will be to administer medications, verify orders, and perform other tasks as directed by the physicians. The nurse can also make observations as needed to stimulate case progression.

VI. CASE NARRATIVE:

A. Scenario Background
   a. Chief Complaint: “Heart racing” / palpitations
   b. Triage Nursing Note: Patient is a 28 year old previously healthy male, who came to the ED today because he has been having episodes of feeling his heart “racing” several times over the past few days. Symptoms began again today approximately three hours prior to arrival without resolution.
   c. Vital Signs: Heart rate 145; Blood pressure 110/86; Respiratory rate 20; Pulse oximetry 98% on room air; Temperature 98.4 degrees Fahrenheit
d. **Past Medical History:** None (If patient is specifically asked about prior episodes, he will report a visit to the ED several years ago after an episode had resolved and he was told to “see his doctor” which he has not done.)

e. **Medications and Allergies:** None

f. **Family and Social History:** No significant family history. Patient denies use of alcohol, tobacco, or illicit drugs

**B. Initial Scenario Conditions**

a. **History given by patient:** He started noticing several days ago that for periods of about an hour or so, his heart would start racing. It has happened several times, but always stopped on its own. Today, about 3 hours prior to coming to the Emergency Department, it started again, and this time it hasn’t stopped.

b. **Circumstances at symptom onset:** If asked what he is doing when the symptoms begin, he says that he hasn’t noticed…at times he’s been exercising, at times he’s been resting quietly.

c. **Associated symptoms:** (Review of Systems must be asked for) He feels a little lightheaded with occasional mild shortness of breath, but he has had no respiratory distress. On review of systems he denies chest pain, sweatiness, abdominal pain, vomiting, or syncope. He has mild nausea without vomiting. No recent illness. No extremity pain or swelling.

d. **Initial Exam:**

   i. **General:** Patient is a healthy appearing Caucasian male. He is awake, alert, and appropriately oriented. He is comfortable without distress.

   ii. **Head, Ears, Eyes, Nose, Throat:** There is no evidence of trauma to his head. His pupils are equal, round, and reactive from 6mm to 4mm. Extraocular movements are fully intact. Ears are normal, there is no discharge, the tympanic membranes are clear, with good light reflex, no evidence of perforation. No mucus membrane rashes, dryness, or swelling.

   iii. **Skin:** Slightly pale. No diaphoresis. No rashes, petechia, or purpura.

   iv. **Cardiovascular:** Tachycardia with irregular rate and rhythm. Equal pulses in all 4 extremities. Point of maximal impulse is nondisplaced. No murmurs, rubs, or gallops

   v. **Lungs:** Clear to auscultation without wheezes, crackles, or rales. Equal breath sounds bilaterally.


   vii. **Genitourinary:** Normal external male genitalia. No hernias, no tenderness to palpation.

   viii. **Extremities:** No muscle tenderness with full range of motion in all extremities. No swelling or edema. Symmetric extremities.

   ix. **Neurological:** Alert and oriented with normal mental status. Pupils equal, round, and reactive to light and accommodation. Cranial nerves II-XII intact. 2+ deep tendon reflexes in all extremities. No sensory or motor deficits. Normal finger to nose pointing, negative Romberg, and normal gait.
e. **Physiology:** (appears when placed patient is placed on monitor)
   i. Heart rate alternates from 120s-140s, irregular, wide complex rhythm
   ii. Blood pressure is 115/92
   iii. pulse oximetry is 98% on room air, 100% if patient is on oxygen
   iv. respiratory rate is 18-20 breaths/minute

C. **Scenario Branch points**

a. **Changes in patient condition:** The patient’s condition will remain stable without changes unless any of the following interventions are performed.

b. **Request for old records:** Only if specifically requested, the patient’s old ECG from a prior ED visit will be provided. It demonstrates a short PR interval and a Delta wave, diagnostic of WPW.

c. **Intravenous fluid administration:** Blood pressure will slightly increase; there will be no change in heart rate or other vital signs.

d. **Performance of vagal maneuvers:** Carotid massage and Valsalva will have no effect on the patient’s condition or vital signs.

e. **Magnesium sulfate administration:** Will have no effect.

f. **Administration of AV nodal blocker:** Calcium channel blocker (such as diltiazem or verapamil), adenosine, beta-blocker (metoprolol, atenolol, propanolol) or digoxin administration will lead to patient deterioration. Heart rate will slightly increase, and then the rhythm will then degenerate into ventricular fibrillation with simultaneous loss of pulses and patient unresponsiveness. At this point, the participants will need to perform appropriate ACLS resuscitative measures.

g. **Synchronized cardioversion:** Will convert the patient to sinus rhythm at a rate of approximately 80 beats per minute.

h. **Defibrillation:** Will result in deterioration of the cardiac rhythm into ventricular fibrillation, with concomitant loss of pulses, unresponsiveness. Participants will need to perform appropriate ACLS resuscitative measures.

i. **Administration of procainamide or amiodarone:** Procainamide at 20-30 mg/min with a maximum dose of 17mg/kg; or amiodarone, 150mg over 10 min, will terminate the wide-complex rhythm and result in sinus rhythm at a rate of approximately 80 beats per minute.
VII. INSTRUCTOR NOTES:

A. Scenario flow – Instructors can directly influence the flow of the scenario by providing the initial patient history via both nursing report and patient verbal responses, since the patient is awake and talking and a source of information.
   a. Old electrocardiogram - One key aspect of the case flow is requesting the old ECG. While not mandatory, it does provide important information to guide decisions. Hopefully, participants will request an old ECG based on their usual practice patterns, or the patient’s report of a prior evaluation. Depending on learner level, instructors may have the patient provide a more obvious “clue” by reporting a prior problem the “last time they did a heart tracing.”

B. Information for actors – This case can easily be presented without formal “actors” and by using other participants to serve as the nursing staff or other collaborating physicians. Any specific actors used outside of participants should be briefed about the critical actions and anticipated flow of the case ahead of time.

C. Scenario programming – The settings for a high-fidelity patient simulator are fairly straightforward for this scenario and do not require specific programming. Our program typically presents this scenario with the initial vital signs as presented above, with the two major branch points to be conversion to normal sinus rhythm or deterioration to cardiopulmonary arrest with ventricular fibrillation.
   a. Wide complex tachycardia – One technical challenge we have encountered is our ability to provide a wide complex irregular tachycardia on the monitor at bedside. With the equipment we use for this case, the closest we can provide is ventricular tachycardia with pulses – which gives an appropriate wide complex but is not irregular. The alternative, using atrial fibrillation settings, gives the irregular rate but without the wide complex.

VII. DEBRIEFING PLAN:

A. Method of debriefing – A post-case debriefing conference can be completed immediately following the end of the scenario. Consider including some of the following elements:
   a. Open-ended questions by facilitator – Consider beginning the session with a question to the primary participant about how they felt the scenario went. This often leads to extensive participant-led discussion that will touch on many of the major issues in the case. Invite any secondary participants and/or observers to comment about how the case unfolded.
   b. Brief didactic review – Potential materials for review after the scenario have been provided in Appendix B.
   c. Formal participant evaluation – Our group does not use this case for formal evaluations and we have no standardized form or format for such feedback. However, this case can easily be adapted to the standardized evaluation method used at your institution using the following critical actions:
1. Obtain appropriate history of present illness from patient.
2. Complete pertinent physical exam and identify irregular tachycardia.
3. Order ECG.
4. Identify wide-complex tachycardia on ECG.
5. Avoid the use of AV nodal blocking agents.
6. Use procainamide or amiodarone to control rate.
7. Arrange appropriate cardiology consultation.

B. Actual debriefing materials – See Appendix B for debriefing materials.

C. Rules for the debriefing – You may find that an informal discussion format for the initial portion of the debriefing leads to an open discussion of aspects of the case management that were good, and those areas where improvement can be made. Encourage your participants to discuss the case management decisions in a non-judgemental way. When this case is used for experiential learning (without formal participant evaluation on the specific case) such discussions may be more productive as compared to situations where formal feedback is anticipated. We have found that placing an emphasis on the learning that occurs from experiencing a case like this is very effective, rather than focusing on the “correct answer” or whether the specific management decisions turned out to be the most appropriate.

D. Questions to facilitate the debriefing –
   a. What are the possible etiologies of an irregular wide-complex tachycardia?
   b. What are the diagnostic features of WPW on an ECG?
   c. What cardiac drugs should be avoided in these patients?
   d. Why are AV nodal blocking agents contraindicated in patients with WPW who are in atrial fibrillation?
   e. What are the recommended therapeutic options for wide-complex atrial fibrillation in WPW patients?

E. ACLS Guidelines for wide complex tachycardia – The faculty member conducting the debriefing may want include a general discussion of how to manage patients with a wide complex tachycardia. The most recent ACLS guidelines from the American Heart Association for the management of wide complex tachycardias are contained in the 2005 supplement to Circulation (see reference list), and the algorithm found as Figure 2 on page IV-70 of these guidelines provides a good overview of the treatment recommendations. A wide complex tachycardia with a regular rhythm that is ventricular tachycardia or uncertain rhythm carries recommendations for amiodarone (150 mg IV over 10 minutes, repeated as needed to a maximum dose of 2.2g/24 hours) and preparation for possible synchronized cardioversion. If the rhythm is SVT with aberrancy, the recommendations are to administer adenosine and treat as a narrow complex rhythm. Irregular wide complex rhythms, such as that seen in this case, carry recommendations for expert consultation if pre-excited atrial fibrillation is present (atrial fibrillation plus WPW), the avoidance of AV nodal blocking agents, and the consideration of antiarrhythmics. Amiodarone is listed in the flowchart presented in Figure 2 as an example of an antiarrhythmic option, and procainamide is
discussed in the text as another option for the treatment of patients with a wide complex rhythm consistent with atrial fibrillation and WPW (see 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, Part 7.3).

IX. PILOT TESTING AND LESSONS LEARNED:

A. Number of participants – This scenario has been presented for small groups of 3-4 participants, and has been field tested approximately 15 times over the past 3 years. All participants have been PGY1, PGY2, and PGY3 emergency medicine residents.

B. Performance expectations – Our experience to date has been that experienced emergency medicine residents, typically PGY3 and late-PGY2 levels of training, are able to successfully negotiate this case. Several residents have identified WPW as a diagnostic consideration when initially presented with the first ECG, and most others correctly identified WPW on the old ECG when requested. While many residents are unfamiliar with the dosages of procainamide, the majority of participants have correctly identified this drug (or amiodarone) as recommended treatment options.

C. Anticipated management mistakes –
   a. Failure to recognize WPW – The minority of residents who have participated in this scenario and failed to recognize WPW have been residents in earlier training years. Many of the groups experiencing this case have included PGY1, PGY2, and PGY3 residents working together – which appears to have influenced the fact that the majority of groups correctly identified WPW, as this often occurs with the influence of the senior residents who have more experience interpreting ECG tracings.
   b. Confusion from the cardiac monitor tracing – As was identified in Section VII (above), one technical issue that we have encountered is our inability to reproduce a wide complex irregular tachycardia on the real-time cardiac monitor with our high-fidelity simulation equipment. This has occasionally led to confusion, as the rhythm we have chosen to most closely represent our patient’s rhythm is a wide complex ventricular tachycardia that appears regular on the monitor. This has infrequently misled some learners who note the regular rate, which argues against atrial fibrillation. We have compensated for this by having the “nursing staff” report feeling that the patient’s pulses are irregular. While this has been a potential distractor, we have not found it to significantly derail the progress of the case.
   c. Use of AV nodal blocking agents – The major learning issue and the biggest potential management mistake in this case is the inappropriate use of AV nodal blocking agents (typically a beta-blocker or calcium channel blocker). This leads directly to patient deterioration. This mistake is typically made when the old ECG has not been requested to help make the diagnosis of WPW, or infrequently when resident learners forget or are not aware of the contraindication for these drugs in this clinical situation. In our experience,
this unfamiliarity with the management of WPW is more common in residents who are in their PGY1 or PGY2 years of training.

D. Evaluation of participants – This case is currently used in our simulation program for emergency medicine residents, but is not used to provide formal performance feedback on this specific case. Therefore, our group does not utilize a standardized form for formal feedback. This is an aspect we are considering for the future, and if this case is selected for formal feedback the objectives in Section III (above) could be adapted to produce a standardized evaluation form for use at your institution.

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XI. References:


Appendix A

Supplemental Case Materials

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Current ECG
Cardiac Enzymes
Troponin negative
CK and CK-MB normal
Appendix B

Case Debriefing Materials

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Wolff-Parkinson-White Syndrome

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Wolff-Parkinson-White Syndrome

- Most common form of ventricular preexcitation
- Twice as prevalent in men
- Often diagnosed in young adults
- Found in approximately 0.2% of people
- 7-20% of patients have congenital heart defects

- Incidence of sudden cardiac death: 0 - 4 %
Pathophysiology of WPW

• **Accessory conduction pathway**
  – Connects the atria and ventricles
  – Bypasses the AV node
  – This accessory pathway is the “Bundle of Kent”

• **Why does this lead to tachycardia?**
  – Accessory pathway does not exhibit decremental conduction
  – Atrial impulses reach ventricles sooner than impulses conducted through the AV node
Development of an Accessory Pathway

• **Normal atria and ventricles**
  – Electrically insulated from one another
  – Annulus fibrosis is nonconductive fibrous tissue

• **Accessory Pathways**
  – Form during embryologic growth
  – Faulty development of annulus fibrosis or valves
  – Ectopic strands of myocardial cells in the annulus
Clinical Features

• Which patients with WPW have symptoms?
  – About 50% of patients experience symptoms
  – Symptoms are due to arrhythmias

• What are the symptoms?
  – Palpitations are the most frequent symptom
  – Other common symptoms:
    • Dizziness, syncope, shortness of breath, chest discomfort

• No abnormal physical findings on examination
• **Short PR interval** (< 0.12 seconds)
  – Caused by non-decremental conduction through the accessory pathway

• **Delta waves** (slurring of initial QRS complex)
  – Preexcitation of ventricles produces delta waves

• **Prolonged QRS complex** (> 0.12 seconds)
  – Caused by ventricles being activated by 2 separate pathways
Characteristic ECG findings
Dysrhythmias in WPW patients

• Most common: AV reciprocating tachycardia
  – Reentry circuit between atria, AV node, ventricles, and accessory pathway
  – 90% orthodromic (narrow-complex tachycardia)
  – 10% antidromic (wide-complex tachycardia)

• Atrial fibrillation
  – Presents with a wide-complex irregular tachycardia
AV Reciprocating Tachycardia

- **Orthodromic:** narrow complex QRS
- **Antidromic:** wide-complex QRS

- Accessory pathway
- AV node
Wide Complex Tachycardia

Atrial fibrillation with underlying WPW
Wide Complex Tachycardia

Differential Diagnosis

• Atrial fibrillation with aberrant conduction

• Atrial fibrillation with underlying WPW

• Polymorphic ventricular tachycardia
  – Including torsades de pointes
Wide Complex Tachycardia

Distinguishing WPW from other causes

- Polymorphic ventricular tachycardia
  - Widened QRS complex
  - Changing R-R intervals
  - Changing QRS complex
  - Subtypes present with an undulating baseline

- Atrial fibrillation in WPW
  - Stable baseline
  - No alterations in the polarity of the QRS complexes
Wide Complex Tachycardia

Distinguishing WPW from other causes

• **Atrial fibrillation with aberrant conduction**
  – Occurs in presence of preexisting bundle branch block
  – Wide-complex irregular rate
  – Stable beat-to-beat QRS configuration

• **Atrial fibrillation in WPW**
  – Has variable beat-to-beat QRS morphology
Atrial Fibrillation in WPW

• How common is it?
  – Occurs in 12 - 39% of patients with WPW
  – Consider the diagnosis in young patients with palpitations, tachycardia, or syncope

• Why is it dangerous?
  – Accessory pathway has non-decremental conduction
  – All atrial impulses transmit to the ventricles
  – Can lead to life-threatening ventricular fibrillation!
Atrial Fibrillation in WPW

Treatment Recommendations

• Do not block the AV node!
  – AV node blockade promotes accessory conduction
  – Can lead to ventricular fibrillation and sudden death

• Drugs to avoid:
  – Beta-blockers
  – Calcium channel blockers
  – Adenosine
  – Digoxin
Atrial Fibrillation in WPW

**Treatment Recommendations**

- **Procainamide** is treatment of choice
  - 20 - 30 mg / min IV, maximum 17 mg / kg
  - Alternative dosing: 100 mg IV every 5 minutes slow IV push until arrhythmia disappears, or up to 1000 mg.
  - Slow rate of infusion due to potential for severe hypotension

- **Amiodarone** has also been recommended
  - 150 mg IV over 10 min
  - **Caution**: can cause accelerated ventricular rate or fibrillation
Atrial Fibrillation in WPW

Treatment Recommendations

• **Unstable patients:**
  – Hypotension
  – Pulmonary edema
  – Ischemic chest pain
  – Altered mental status

• **Use immediate electrical cardioversion!**
References


