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 Wake Forest University in North Carolina and Wright State University in Ohio for emergency medicine residents completing educational time in Emergency Department Simulation programs. 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 patient presenting with fatigue and weakness. Evaluation in the emergency department demonstrates bradycardia and worsening hemodynamic status from beta-adrenergic antagonist toxicity. Appropriate treatment with glucagon and vasopressors will allow stabilization of the patient. Debriefing materials are provided to illustrate and stimulate discussion of the important concepts for diagnosing and treating patients with beta-blocker toxicity.
SIMULATION SCENARIO

I. TITLE: Beta Blocker Overdose

II. TARGET AUDIENCE: Resident physicians

III. LEARNING OBJECTIVES:

   A. Primary Learning Objectives
      1. Demonstrate an appropriate initial approach to a patient with hypotension and bradycardia
      2. Consider toxicological causes for the patient’s presentation
      3. Demonstrate appropriate management of beta blocker poisoning

   B. Secondary Goals
      1. Develop a differential diagnosis for hypotension and bradycardia
      2. Identify treatment failure of standard therapies such as intravenous fluids and atropine
      3. Describe treatment options for beta blocker poisoning

   C. Critical actions checklist
      1. Obtain appropriate history of present illness
      2. Identify unstable vital signs
      3. Place the patient on a cardiac monitor and establishes IV access
      4. Administer IV glucagon
      5. Initiate treatment for bradycardia and hypotension (may include IV fluid bolus, central venous access, transthoracic or transvenous pacing, adrenergic agonists, etc…)
      6. Perform appropriate ACLS if patient decompensates
      7. Admit the patient to a critical care unit.

IV. ENVIRONMENT

   A. Lab Set Up – Emergency Department bed in a simulation facility

   B. Manikin Set Up – The manikin will be in a seated position on an ED stretcher, either in street clothes or a hospital gown. Initially, there will be no ECG leads attached, and no IV line unless requested by the physician.

   C. Props – Available for use will be a heart monitor with leads, blood pressure cuff, and pulse oximeter. Supplemental oxygen by nasal cannula and a 15-liter facemask 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 Materials** – See Appendix A for audiovisuuals provided for this case. Included are a chest radiograph, an EKG showing sinus bradycardia with a rate in the low 50s, and a set of laboratory values that may be provided if requested.

E. **Distractors** – Distractors can be added at faculty 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** – The case can be run with a minimum of one participant to play the role of physician and one case director/simulation operator; however, other roles may be those of nurse, attending physician (if two physicians, one a resident and one an attending, are desired), family members

B. **Who may play them** – Actors may include resident physicians, medical students, nurses, or attending physicians.

C. **Actions for the roles** –
   a. **Primary physician** - The main scenario participant will 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:** “weak and tired”
   b. **Triage Nursing Note:** Patient is a 65 year old previously healthy Caucasian male, who came to the ED today because he feels “weak all over”. Symptoms began 2 days ago.
   c. **Vital Signs:** Heart rate 49, Blood Pressure 90/60, Respiratory Rate 12, Pulse Oximetry 95% on room air, Temperature 96.9 degrees Fahrenheit
   d. **Past Medical History:** Provided only if requested. He has had two previous myocardial infarctions (with a stent placed in his right coronary artery 2 years ago), congestive heart failure with an ejection fraction of 40%, hypertension, hyperlipidemia, diabetes mellitus type II, osteoarthritis, depression.
e. **Medications and Allergies:** Only provide medication list if specifically requested. Medications include aspirin, glipizide, furosemide, metoprolol, clopidogrel, simvastatin, sertraline. Allergy to penicillin (rash.)

f. **Family and Social History:** 40 pack-year history of smoking, occasional alcohol use, denies illicit drug use. His mother died of a stroke at 82; his father died in a motor vehicle accident at age 40.

### B. Initial Scenario Conditions

a. **History given by patient:** The patient reports that symptoms began 2 days ago and have worsened. He feels dizzy and as if he’s going to pass out. He has had no syncope. The dizziness is worse when he goes from a sitting to standing position. The patient’s wife is also available for interview, and she reports that his medications have always been very confusing, and recent changes to his dosing have been difficult for him to remember.

b. **Circumstances at symptom onset:** Patient was going about his daily routine when he noticed the symptoms.

c. **Associated symptoms:** *(Review of Systems must be asked for)* The patient has intermittent chest pains, which are described as sharp, fleeting, substernal, and without radiation. He has no chest pain currently. He has had mild dyspnea on exertion, a persistent dry cough, and mild nausea. He denies vomiting, diaphoresis, and hemoptysis. He has had a decreased appetite for one week. He has had no recent surgery, and no history of cancer, deep venous thrombosis, or pulmonary embolism. He denies headache, neck pain, visual changes, abdominal pain, fevers, rashes, or change in his bowel movements.

d. **Initial Exam:**
   i. **General:** Patient is a healthy appearing male. Patient is awake, alert, and oriented. Comfortable without distress. Appears slightly fatigued.
   
   ii. **Head, Ears, Eyes, Nose, Throat:** There is no evidence of head trauma. 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 normal.
   
   iii. **Skin:** Normal color and turgor. No diaphoresis. No skin rash.
   
   iv. **Cardiovascular:** Bradycardia with regular rhythm. Equal pulses in all 4 extremities. Point of maximal impulse is nondisplaced. No murmurs, rubs, or gallops.
   
   v. **Lungs:** Scattered rhonchi and end-expiratory wheezes bilaterally, with no rales. Equal breath sounds bilaterally.
   
   
   vii. **Genitourinary:** Normal external genitalia. No hernias, no tenderness.
   
   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 and normal gait.
e. **Physiology:** (appears when placed patient is placed on monitor)
   i. Heart rate 49
   ii. Blood pressure is 90/60
   iii. Pulse oximetry is 95% on room air, 100% if patient is on oxygen
   iv. Respiratory rate is 12-15 breaths/minute

C. **Scenario Branch points**

a. **Changes in patient condition:** The patient’s condition will deteriorate over several minutes, during the course of the history and physical exam. His blood pressure will begin to trend lower, as will his heart rate. If the correct interventions are not initiated in a timely fashion, he will go into a PEA arrest and appropriate ACLS protocols will need to be followed. His arrest will not resolve until appropriately treated (see below.)

b. **Request for old records:** Unavailable at this time.

c. **Intravenous fluid administration:** IV fluids can be administered in response to his worsening hypotension, but IV fluid alone will not correct his low blood pressure.

d. **Laboratory studies:** Laboratory results can be found in Appendix A. None of them will be helpful in determining the etiology of the patient’s symptoms.

e. **Administration of medications:**

   i. **Atropine:** If given according to ACLS protocol, atropine will have no effect on the patient’s bradycardia.

   ii. **Glucagon:** If the physician correctly suspects beta-blocker toxicity, glucagon administration will result in a transient return of the patient’s vital signs to baseline (normotensive 110/80 with a normal heart rate of 82). However, a single dose will not be sufficient, and the patient will slowly become hypotensive and bradycardic again.

   iii. **Vasopressors:** After multiple doses of glucagon fail to maintain the patient’s vital signs, vasopressors should be initiated. Appropriate choices include dopamine, norepinephrine, or phenylephrine. The patient’s blood pressure will return to a normal range of 110/70 to 120/80 with the appropriate use of vasopressor agents.

   iv. **Insulin and glucose:** This is another acceptable treatment pathway for this patient, who will improve transiently with bolus therapy of glucose and insulin. If learners contact the Poison Control Center by telephone during the case, this therapy can be recommended.
v. **Glucagon infusion**: Bradycardia will not resolve permanently until a continuous glucagon infusion is initiated.

f. **Consultation of specialist**: Once stabilized, the patient should be admitted to an intensive care unit, optionally under the care of a cardiologist.

**VII. INSTRUCTORS 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. **Obtain ECG** - One key aspect of the case flow is requesting the ECG. A delay in obtaining an ECG will not allow the learners to recognize the significant bradycardia as a clue to the etiology of the patient’s symptoms.
   
b. **Review list of medications** – The patient’s long list of medications includes a beta-blocker, which should trigger the managing physician to suspect this medication as a possible source of pharmacologic bradycardia. Additional history from the patient’s wife will direct learner’s to this list if needed.

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. Our programs typically present this scenario with the initial vital signs as presented above, with the two major branch points to be worsening bradycardia and hypotension, or appropriate treatment to increase heart rate and blood pressure.

**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. We typically take 5-10 minutes after the debriefing session for this didactic review.

c. Formal participant evaluation – We have included an evaluation form for use with this case in Appendix C.

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

C. Rules for the debriefing – An informal discussion format for the initial portion of the debriefing often leads to an open discussion of aspects of the case management that were managed well, and other areas where improvement can be made. Encourage participants to discuss the management decisions in a non-judgmental way. When this case is used for experiential learning (without formal evaluation) such discussions can be very productive. 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 possible toxicological etiologies of hypotension and bradycardia?
   b. Name another disease process that may result in hypotension and bradycardia
   c. What are appropriate initial steps to take in treating hypotension?
   d. What cardiac drugs should be avoided in this setting?
   e. What other etiology for bradycardia may be resistant to medical therapy?
   f. Name other, rare, effects of beta blocker toxicity.
   g. What are indications for transvenous pacing in the bradycardic patient?

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 8 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.

C. Anticipated management mistakes –
   a. Failure to identify beta-blockers as culprit – Some residents have been slow to identify a possible toxicological cause of this patient’s symptoms. Redirecting learners to the medication list for possible etiologies of the symptoms can be effective in this situation.
b. **Fixation on atropine** – Some learners have used repeated doses of atropine in the hopes that this will change patient’s bradycardia, even though it has no effect in this patient. This has been a valuable learning experience for residents in training to develop a “back-up plan” when more traditional management decisions are unsuccessful.

c. **Delayed use of glucagon** – If the learners fail to identify beta-blockers as a possible issue in this patient, they will not appropriately initiate glucagon treatment. This will lead to PEA arrest, and the patient will not respond to external cardiac pacing despite maximal efforts. This can be a good opportunity to review ACLS guidelines for the management of PEA.

D. **Evaluation form for participants** – We have included an evaluation form for use with this case in Appendix C.

X. **AUTHORS AND THEIR AFFILIATIONS**

**Michael T. Fitch, M.D., Ph.D.**
Associate Professor  
Director, Emergency Medicine Simulation Program  
Wake Forest University, Department of Emergency Medicine  
Medical Center Boulevard, Winston-Salem, North Carolina 27157

**Corey Heitz, M.D.**
Faculty and Academic Fellow  
Wright State University, Department of Emergency Medicine, 3525 Southern Blvd, Kettering, Ohio 45429

**Grant Williams, M.D.**
Staff Physician  
Central Texas Medical Center, Department of Emergency Medicine  
1301 Wonder World Dr, San Marcos, Texas 78666

**Jennifer Hannum, M.D.**
Assistant Professor  
Toxicology Program Director  
Wake Forest University, Department of Emergency Medicine  
Medical Center Boulevard, Winston-Salem, North Carolina 27157

**Ethan Freeborn, M.D.**
Staff Physician  
Carilion New River Valley Medical Center, Department of Emergency Medicine  
2800 Lamb Circle  
Radford, VA 24141
XI. REFERENCES


Appendix A

Case Supplemental Materials

Corey R. Heitz, M.D.
Grant Williams, M.D.
Ethan Freeborn, M.D.
Jennifer Hannum, M.D.
Michael T. Fitch, M.D., Ph.D.
Electrocardiogram

- Vent. rate: 48 BPM
- PR interval: 144 ms
- QRS duration: 92 ms
- QT/QTc: 464/414 ms
- P-R-T axes: 34 57 40
Chest Radiograph
<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>8.4</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>11.1</td>
</tr>
<tr>
<td>Platelets</td>
<td>383</td>
</tr>
<tr>
<td>WBC</td>
<td>34.0</td>
</tr>
<tr>
<td>Sodium</td>
<td>144</td>
</tr>
<tr>
<td>Chloride</td>
<td>98</td>
</tr>
<tr>
<td>BUN</td>
<td>36</td>
</tr>
<tr>
<td>Glucose</td>
<td>138</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.5</td>
</tr>
<tr>
<td>Bicarb</td>
<td>18</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.7</td>
</tr>
<tr>
<td>PT</td>
<td>11.1</td>
</tr>
<tr>
<td>INR</td>
<td>1.0</td>
</tr>
<tr>
<td>PTT</td>
<td>33.7</td>
</tr>
<tr>
<td>CK</td>
<td>106</td>
</tr>
<tr>
<td>CK-MB</td>
<td>6.82</td>
</tr>
<tr>
<td>Troponin I</td>
<td>&lt;0.07</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>0.94</td>
</tr>
<tr>
<td>Cardiac BNP</td>
<td>89</td>
</tr>
<tr>
<td>Lactate</td>
<td>3.6</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>Normal</td>
</tr>
</tbody>
</table>
Appendix B

Case Debriefing Materials

Corey R. Heitz, M.D.
Grant Williams, M.D.
Ethan Freeborn, M.D.
Jennifer Hannum, M.D.
Michael T. Fitch, M.D., Ph.D.
Beta-Blocker Overdose

Corey R. Heitz, M.D.¹
Grant Williams, M.D.²
Ethan Freeborn, M.D.³
Jennifer Hannum, M.D.⁴
Michael T. Fitch, M.D., Ph.D.⁴

Departments of Emergency Medicine
¹ Wright State University, Boonshoft School of Medicine
² Central Texas Medical Center
³ Carilion New River Valley Medical Center
⁴ Wake Forest University School of Medicine
Beta-Blocker Overdose

- 2004 Toxic Exposure Surveillance System report
  - 162 deaths due to cardiovascular medications
    - 5th leading cause of death due to poisonings

- **β-blockers** and calcium-channel antagonists accounted for **37% of exposures**

- Majority of cases resulted in life-threatening cardiac effects or death
• **Beta-1 receptors**
  – Regulate myocardial tissue
  – Affect rate of contraction via impulse conduction

• **Beta-2 receptors**
  – Regulate smooth muscle
  – Influence vascular and bronchial relaxation

• **Beta-3 receptors**
  – Primarily affect lipolysis
  – May have some effect on cardiac inotropy
Toxic Effects

- Beta Blockers therefore
  - Decrease rate of contraction (negative chronotropy)
  - Depress myocardium (negative inotropy)
  - May result in bronchoconstriction and vasodilation
  - Decrease lipolysis (may result in hypoglycemia)
  - Decrease calcium entry into cells
Clinical Features

• **Presenting Signs/Symptoms**
  – Weakness, dizziness, decreased level of consciousness
  – Symptoms are due to hypotension and bradycardia
  – Other possible symptoms:
    • Normal to low blood glucose
    • Rarely can have bronchospasm

• **Typically there are no specific physical findings**
Bradycardia

Differential Diagnosis

• **Other medications**
  – Calcium channel blocker
  – Central-acting alpha agonist (*i.e.*, clonidine)
  – Digoxin
  – Cholinergics
  – Sedatives / hypnotics

• **Metabolic abnormalities**
  – Hyperkalemia, hypothermia, myocardial ischemia, intracranial hypertension

• **Arrhythmias**
  – Sick sinus syndrome
Undifferentiated Hypotension

Differential Diagnosis

- Other conditions to think about in this case
  - Myocardial infarction
  - Myocarditis
  - Sepsis
  - Dehydration
  - Hypovolemia
  - Sick sinus syndrome
  - Neurological disease
  - Oncologic disease
Undifferentiated Hypotension

Bedside Ultrasound / Echocardiography

• **Real time imaging of cardiac function**
  – Preload
  – Contractility
  – Presence of pericardial effusion
  – Right ventricular strain
Beta-Blocker Overdose

Treatment Recommendations

- Glucagon
- Atropine may be ineffective for severe toxicity
- Large doses of epinephrine may be needed
- Consider calcium to overcome channel blockade
- Catecholamines
- Insulin
Beta-Blocker Overdose

Treatment Recommendations

• **Glucagon**
  - **Dose**
    • 50-150 µg/kg (3-10 mg in adult)
    • Repeat every 3-5 minutes
    • Initiate infusion
      - Effective bolus dose per hour
  - **Adverse effects**
    • Nausea and vomiting
    • Hyperglycemia
Treatment Recommendations

- **Catecholamines**
  - No one catecholamine is superior – may require large doses
  - Base choice on cardiodynamic and hemodynamic monitoring
    - Norepinephrine or epinephrine are recommended for patient with depressed contractility and decreased peripheral resistance
  - Be cautious with isoproterenol and dobutamine
    - Have predominant β-receptor activity
    - May decrease peripheral resistance and worsen hypotension
Beta-Blocker Overdose

**Treatment Recommendations**

- **High Dose Insulin – Euglycemia**
  - During drug-induced shock, metabolic demands shift
    - Preferred myocardial energy substrate shifts from free fatty acids to carbohydrates
    - Supplemental insulin supports carbohydrate metabolism
    - Improved function following insulin treatment occurs without an increase in myocardial work
Beta-Blocker Overdose

Treatment Recommendations

• High Dose Insulin – Euglycemia
  – Evidence
    • Animal model of propranolol toxicity
      – Reversed myocardial failure and increased coronary blood flow
      – Improved survival when compared to standard treatment
    • 68 patients ingested calcium channel blockers, beta-blockers, or both:
      – Blood pressure and contractility increased in 63 patients
      – Little chronotropic effect
      – Overall survival 85%
Beta-Blocker Overdose

Non-pharmacological Therapy

• **Hemodialysis**
  – May help with atenolol, nadolol, or sotalol

• **Pacing**
  – May have difficulty with capture
  – Intracellular calcium is needed
  – Keep rate ~60, which allows for contraction time

• **Extraordinary Measures**
  – ECMO, balloon pump, prolonged CPR
Selected References


Beta-Blocker Overdose

Corey R. Heitz, M.D.¹
Grant Williams, M.D.²
Ethan Freeborn, M.D.³
Jennifer Hannum, M.D.⁴
Michael T. Fitch, M.D., Ph.D.⁴

Departments of Emergency Medicine
¹ Wright State University, Boonshoft School of Medicine
² Central Texas Medical Center
³ Carilion New River Valley Medical Center
⁴ Wake Forest University School of Medicine