

## Novel Emergency Medicine Curriculum Utilizing Self-Directed Learning and the Flipped Classroom Method: Cardiovascular Emergencies Small Group Module

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### ABSTRACT:

**Audience:** This curriculum, created and implemented at The Ohio State University Wexner Medical Center, was designed to educate our emergency medicine (EM) residents, post-graduate year (PGY)-1 to PGY-3, as well as medical students and attending physicians.

**Introduction:** In 2013, chest pain alone was the principal reason for visit for over 6 million emergency department (ED) visits in the United States. This represents 4.9% of all ED visits.<sup>1</sup> Residents must be proficient in the differential diagnosis and management of the wide variety of cardiovascular emergencies. The flipped classroom curricular model emphasizes self-directed learning activities completed by learners, followed by small group discussions pertaining to the topic reviewed. The active learning fostered by this curriculum increases faculty and learner engagement and interaction time typically absent in traditional lecture-based formats.<sup>2-4</sup> Studies have revealed that the application of knowledge through case studies, personal interaction with content experts, and integrated questions are effective learning strategies for emergency medicine residents.<sup>4-6</sup> The Ohio State University Wexner Medical Center EM Residency didactic curriculum recently transitioned to a “flipped classroom” approach.<sup>7-10</sup> We created this innovative curriculum aimed to improve our residency education program and to share educational resources with other EM residency programs. Our curriculum utilizes an 18-month curricular cycle to cover the defined emergency medicine content. The flipped classroom curriculum maximizes didactic time and resident engagement, fosters intellectual curiosity and active learning, and meets the needs of today's learners.<sup>3,11,13</sup>

# CURRICULUM

**Aims/Goals:** We aim to teach the presentation and management of cardiovascular emergencies through the creation of a flipped classroom design. This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

**Methods:** The educational strategies used in this curriculum include small group modules authored by education faculty and content experts based on the core emergency medicine content. This program also includes resident-submitted questions that were developed during review of the content. The question and answer format of the Socratic Method--with a focus on fostering an open learning environment, not negative "pimping"-type questionin--is used during small group sessions to encourage active participation and discussion; small groups also focus on the synthesis and application of knowledge through the discussion of real life experiences. The use of free open access medical education (FOAM) resources allows learners to work at their own pace and maximize autonomy.

**Topics:** Emergency medicine, flipped classroom, medical education, cardiovascular emergencies, pedagogy, teaching.



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## Learner Audience:

Medical students, interns, junior residents, senior residents, attending physicians and faculty

## Length of Curriculum:

The entire didactic curriculum was developed to utilize an 18-month curricular cycle; therefore, resident learners experience each curricular topic twice in the course of a three-year residency training. The cardiovascular emergencies module consists of thirteen small group sessions which are 45-60 minutes each.

## Topics:

Emergency medicine, flipped classroom, medical education, cardiovascular emergencies, pedagogy, teaching.

## Objectives:

Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the Cardiovascular Emergencies Module include:

1. After completing the Cardiovascular Emergencies Module, resident learners will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult cardiovascular

emergencies. Specifically:

2. Pulmonary Embolism
  - a. Review pathophysiology, diagnosis and treatment of pulmonary embolism.
  - b. Discuss the role of bedside ultrasound in the diagnosis of massive pulmonary embolus.
  - c. Review and discuss the indications for various imaging modalities, systemic thrombolytic administration, and catheter-assisted thrombolytic administration.
  - d. Critically discuss the ADJUST-PE study assessing age-adjusted d-dimer cutoff levels to rule out PE.
  - e. Critically discuss the use of trimester adjusted d-dimer in pregnancy to clinically rule out PE.
  - f. Discuss what patients, if any, diagnosed with an acute PE in the emergency department (ED) you would consider for outpatient anticoagulation versus admission.
  - g. Debate the role of heparin versus enoxaparin and warfarin versus newer oral anticoagulants in the treatment of PE.
3. Peripheral Vascular Disease
  - a. Describe the difference in the pathophysiology of atherosclerosis, arterial thrombus formation, atheroembolism, thromboembolism, aneurysms, and fistulae.
  - b. List the risk factors associated with acute limb ischemia.
  - c. Discuss the diagnostic modalities used in the acute evaluation of limb ischemia or claudication.
  - d. Describe treatment options for limb ischemia in the emergency department (ED).
  - e. Discuss the clinical significance of the Rutherford classification related to immediate management.
  - f. Describe the three phases of Raynaud's phenomenon, and describe the ED diagnosis and management.
4. Cardiac Arrhythmias
  - a. List and compare different narrow complex tachycardic arrhythmias.
  - b. List risk factors and complications of atrial fibrillation.



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- c. Describe how to differentiate Wolff-Parkinson-White (WPW) from other narrow complex tachycardias.
  - d. Discuss initial evaluation and management of patients presenting with atrial fibrillation with rapid ventricular response, including cardioversion, rate and rhythm control medications, and anti-coagulation.
  - e. Discuss indications for atrial fibrillation in patients with atrial fibrillation.
  - f. Describe management of wide complex tachycardia.
  - g. Define the following: Premature ventricular complexes (PVCs), ventricular bigeminy, ventricular trigemini, ventricular couplets, ventricular escape beats, non-sustained and sustained ventricular tachycardia (VT), monomorphic versus polymorphic VT, accelerated idioventricular rhythm and ventricular flutter.
  - h. Describe Brugada's syndrome.
  - i. Discuss the Vaughan Williams classes of medications that are used to treat dysrhythmias, the mechanism, and name a medication in these classes.
5. Congestive Heart Failure
- a. Differentiate between congestive heart failure (CHF) with volume overload and flash pulmonary edema with normal volume and high afterload.
  - b. Discuss available therapies for management of CHF with flash pulmonary edema – nitroglycerin (NTG), diuretics, intravenous angiotensin converting enzyme (ACE) inhibitors, dobutamine, morphine.
  - c. Discuss the use of bilevel positive airway pressure (BiPAP) vs. continuous positive airway pressure (CPAP) for acute pulmonary edema.
6. Congenital Heart Disease
- a. Describe unique changes in newborn anatomy and physiology that lead to the presentation of congenital heart disease (CHD).
  - b. Compare CHD findings and diagnoses in patients with pulmonary under-circulation versus pulmonary over-circulation.
  - c. Discuss the impact of anatomic (closure of

- the arterial duct) and physiologic changes (decrease in pulmonary vascular resistance) in the newborn with the presentation of congenital heart disease in infants.
  - d. Describe stabilizing initial care of infants presenting with congenital heart disease, including discernment of key aspects to be communicated to the pediatric cardiologist.
7. Valve Disorders, Endocarditis and Tumors
- a. Identify patient populations at risk for developing infective endocarditis (IE).
  - b. Describe the classic physical exam findings of IE.
  - c. Discuss appropriate workup and empiric antibiotics for presumptive IE.
  - d. Describe the classic presentation of patients with aortic stenosis (AS).
  - e. Understand the pathophysiology of AS.
  - f. Discuss the diagnosis and management of AS patients in the emergency department (ED).
8. Acute Coronary Syndrome
- a. Discuss methods to stratify patients with undifferentiated chest pain for acute coronary syndrome (ACS).
  - b. Describe commonly used decision-making rules (HEART and TIMI score) for risk-stratification of ACS.
  - c. Discuss the definition of ST-segment elevation myocardial infarction (STEMI) and possible STEMI equivalents.
  - d. List and discuss pharmacologic management of patients with confirmed or suspected ACS.
  - e. Describe the appropriate use of provocative testing for patients with suspected ACS.
  - f. List indications for urgent or emergent cardiology consultation and reperfusion therapy.
9. Brief Resolved Unexplained Event (BRUE)
- a. Define brief resolved unexplained event (BRUE) and the risk factors associated with it.
  - b. Differentiate possible mimics of BRUE in the young infant.
  - c. Discuss initial evaluation of BRUE based on history and physical examination and



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differentiate which patients require a complete evaluation and which can be observed.

- d. Discuss historical and physical exam findings consistent with non-accidental trauma (NAT) which may suggest physical abuse as a potential cause of BRUE.

## 10. Left Ventricular Assist Devices (LVADs)

- a. Identify the indications for left ventricular assist device (LVAD) placement.
- b. Discuss the physiology behind the function of an LVAD.
- c. Understand the first steps in evaluation of a patient with an LVAD.
- d. List the possible complications of a patient presenting with an LVAD.
- e. Understand steps in treating a patient with an LVAD who is unresponsive or hemodynamically unstable.

## 11. Pericardial Disorders

- a. Identify the most common etiologies of acute pericarditis.
- b. Describe the clinical features associated with acute pericarditis and pericardial tamponade.
- c. Describe the diagnostic findings associated with acute pericarditis and pericardial tamponade.
- d. Discuss the treatment and management for acute pericarditis and pericardial tamponade.
- e. Explain the pathophysiology associated with cardiac tamponade.

## 12. Aortic Dissection

- a. Review pathophysiology, diagnosis and treatment of aortic dissection.
- b. Discuss the classification systems for aortic dissection and the different associated management strategies – surgical v conservative medical management.
- c. List the various imaging modalities available to diagnosis aortic dissection and discuss in what settings they may be appropriate.
- d. Discuss medications used for treatment of aortic dissection and goals for heart rate and blood pressure management.

## 13. Syncope

- a. Describe common causes of syncope.

- b. Discuss which historical and physical examination data can help risk-stratify patients with syncope.
- c. Discuss which diagnostic testing help risk-stratify patients with syncope.
- d. List the most serious causes of syncope.
- e. Identify which patients should be admitted after a syncopal episode of unclear cause.

## 14. Resuscitation and Advanced Cardiovascular Life Support (ACLS)

- a. Discuss the key components of advanced cardiac life support (ACLS).
- b. Identify important factors in optimizing chest compressions.
- c. List the various causes of cardiac arrest and how to treat the reversible causes.
- d. Describe the appropriate use of adjunctive tools (intubation, arterial lines, etc.) in cardiac arrest.
- e. Describe the interpersonal and interdisciplinary interactions that are important in a successful resuscitation.

### Brief introduction:

The flipped classroom learning approach is becoming more commonly recognized as a preferred curricular model for mature learners, specifically those in medical education. This particular model is a natural fit for the hands-on, experiential emergency medicine learner.<sup>4</sup> The active learning fostered by this curriculum increases faculty and learner engagement and interaction time which is typically absent in traditional lecture based formats.<sup>5,12</sup> Education literature shows that resident learners prefer learning activities that involve small group discussion, are case/skill based, and emphasize the application of newly obtained knowledge.<sup>3,4</sup> This educational model also provides a clear channel for the incorporation of evidence-based medicine and increases opportunities for educator-learner conversations. A successful flipped classroom curriculum fosters learner accountability and provides robust opportunities for formal assessment in various emergency medicine milestones.<sup>4,7,10</sup> For these reasons, we developed a flipped classroom curriculum at The Ohio State University Wexner Medical Center.

### Problem identification, general and targeted needs assessment:

Traditional lecture-based didactics may not be the most effective or preferred method for emergency medicine resident education.<sup>6</sup> Previously, we used a traditional lecture format in our residency curriculum despite overwhelming evidence for a more hands-on, “flipped classroom” approach.<sup>8,9</sup> From the



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perspective of resident learners, the chance to remain fully engaged through the asking of questions developed from personal experiences, and also by learning from the experiences of others provides a manner of learning that makes a topic more difficult to forget.<sup>5</sup>

Cardiovascular emergencies are an important part of any emergency medicine residency curriculum. The ABEM model of the clinical practice of emergency medicine<sup>6</sup> includes broad cardiology content domains, and the ABEM in-training exam reports 10% of questions will be based on cardiology related content, with traumatic injuries the only content area given similar weight.<sup>14</sup> Our institution's previous lecture-based curriculum lacked engagement from faculty and residents.

As current literature reveals, both educators and learners benefit from an interactive and collaborative classroom, leading to the creation and implementation of this proposed curricular model at our emergency medicine residency program.<sup>10</sup> This weekly small group curriculum has now replaced three hours of traditional lecture-based didactics. Learners divide into small groups of about 20 participants. Each group is led by both a faculty leader and a designated senior resident who has spent extra time preparing; the senior resident is expected to guide the discussion with a question and answer format while the faculty member is there to add expertise and guidance. Since implementation, residents and educators are engaging in new, valuable flipped classroom learning communities at The Ohio State University Wexner Medical Center. Through the curriculum, we continually seek to foster self-directed learning and increased collaboration between resident learners and education faculty members. This ensures that resident time will be maximized and learning will be more efficient and effective, therefore providing a potential positive impact on patient care and physician wellness. Currently, minimal flipped classroom curricular materials dedicated to the core content of emergency medicine exist.

## Goals of the curriculum:

This curricular innovation was developed and implemented to promote self-directed/active learning and an environment of intellectual curiosity and learner accountability. This flipped classroom curriculum is specifically designed to cover the core content of emergency medicine; this module promotes the mastery of cardiovascular emergencies. Secondary goals include the increased interaction between educators and learners, and the evaluation of resident small group teaching skills.

## Objectives of the curriculum:

Each chapter within our curriculum has individual objectives; however, educational objectives for the curriculum and more specifically, the Cardiovascular Emergencies Module include:

1. After completing the Cardiovascular Emergencies Module, resident learners will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult cardiovascular emergencies. Specifically:
  - a. Review pathophysiology, diagnosis and treatment of pulmonary embolism.
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  - b. Discuss the physiology behind the function of an LVAD.
  - c. Understand the first steps in evaluation of a patient with an LVAD.
  - d. List the possible complications of a patient presenting with an LVAD.



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- e. Understand steps in treating a patient with an LVAD who is unresponsive or hemodynamically unstable.
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    - e. Describe the interpersonal and interdisciplinary interactions that are important in a successful resuscitation.

## Educational Strategies: (See curriculum chart)

Please refer to the curriculum chart of linked objectives and educational strategies.

## Evaluation and Feedback:

This innovative curriculum was literature-based and specifically designed to maximize active learning using the flipped classroom learning model. We overcame initial challenges and skepticism from both educators and learners to execute a successful, novel curricular model. Both resident learners and faculty educators provided positive feedback. The curriculum was critically evaluated and updated by education faculty members in order to ensure educational material remains current and consistent with the emergency medicine core content. Residents were surveyed on their perceived quality of instruction of the various program components. A majority of residents (60.9%) reported that the small group discussions were good or excellent, compared to only 26% of residents that felt that our grand rounds sessions during the same time were good or excellent. This cardiology curriculum has been delivered to two cohorts of learners and has delivered the content twice in three years with about 50 residents per cycle. On the most recent iteration, residents evaluated the teaching methods as effective, with an average rating of more than 4.5 out of 5 (4 being agree, 5 being strongly agree). A recommended small group session evaluation is provided in the appendices. Small group facilitators assessed residents with a rubric (see appendix), and the majority of students met or exceeded expectations.

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# DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Pulmonary Embolism	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Indications for and interpretations of d-dimer and clinical decision rules such as PERC and Wells</p> <p>-Therapeutic options including the role of catheter directed intervention, heparin and oral anticoagulants</p> <p>-Presentation, diagnosis and management of pulmonary embolism</p>	<p>By the end of this session, learners will:</p> <p>-Review pathophysiology, diagnosis and treatment of pulmonary embolism (PE)</p> <p>-Review and discuss the indications for various imaging modalities, systemic thrombolytic administration, and catheter assisted thrombolytic administration</p> <p>-Critically discuss the ADJUST-PE study assessing age-adjusted d-dimer cutoff levels to rule out PE</p> <p>-What patients, if any, diagnosed with an acute PE in the emergency department would you consider for outpatient anticoagulation vs admission?</p> <p>-Debate the role of heparin v lovenox and warfarin v newer oral anticoagulants in the treatment of PE</p> <p>-Discuss the role of bedside ultrasound in the diagnosis of massive PE</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), ultrasound (PC12), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Peripheral Vascular Disease	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and therapies for acute and chronic peripheral vascular disease</p> <p>-Imaging modalities and interpretation of results</p> <p>-Indications for emergent surgical intervention</p>	<p>By the end of this session, learners will:</p> <p>-Describe the difference in the pathophysiology of atherosclerosis, arterial thrombus formation, atheroembolism, aneurysms and fistulae</p> <p>-List the risk factors associated with acute limb ischemia</p> <p>-Discuss the diagnostic modalities used in the acute evaluation of limb ischemia or claudication</p> <p>-Explain the clinical significance of the Rutherford classification related to immediate management</p> <p>-Explain the three phases of Raynaud’s disease</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), ultrasound (PC12), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Cardiac Arrhythmias	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis, and management of common arrhythmias</p> <p>-Indications for emergent cardioversion</p> <p>-Pharmacologic management of arrhythmias</p>	<p>By the end of this session, learners will:</p> <p>-Discuss initial evaluation and management of patients presenting with atrial fibrillation with rapid ventricular response</p> <p>-Describe management of wide complex tachycardia</p> <p>-Define the following: PVCs, ventricular bigeminy, ventricular trigeminy, ventricular couplets, ventricular escape beats, VT non-sustained and sustained, monomorphic v polymorphic VT, accelerated idioventricular rhythm and ventricular flutter</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Congestive Heart Failure (CHF)	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis, and M\management of CHF</p> <p>-Pathophysiology, diagnosis, and management of pulmonary edema</p> <p>-Appropriate pharmacologic management of CHF and pulmonary edema</p> <p>-Use of noninvasive positive pressure ventilation in management of CHF and pulmonary edema</p>	<p>By the end of this session, learners will:</p> <p>-Review the potential root causes of Congestive Heart Failure.</p> <p>-Differentiate between systolic and diastolic heart failure.</p> <p>-Differentiate between CHF with volume overload and flash pulmonary edema with normal volume and high after load.</p> <p>-Describe the diagnosis of CHF.</p> <p>-Discuss available therapies for management of CHF with flash pulmonary edema – nitroglycerin, diuretics, IV ACE inhibitors, dobutamine, morphine.</p> <p>-Discuss the use of BiPAP vs. CPAP for acute CHF.</p> <p>-Discuss when a balloon pump is indicated.</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Valve Disorders and Endocarditis	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and management of infective endocarditis</p> <p>-Pathophysiology, diagnosis and management of aortic stenosis</p>	<p>By the end of this session, learners will:</p> <ul style="list-style-type: none"> <li>-Identify patient populations at risk for developing infective endocarditis (IE)</li> <li>-Describe the classic physical exam findings of IE</li> <li>-Order appropriate workup and empiric antibiotics for presumptive IE</li> <li>-Describe the classic presentation of patient with aortic stenosis (AS)</li> <li>-Understand the pathophysiology of AS</li> <li>-Demonstrate knowledge of the diagnosis and management of AS patients in the ED</li> </ul>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Acute Coronary Syndrome (ACS)	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and management of ACS</p> <p>-EKG interpretation</p> <p>-Use of clinical decision tools for risk stratification</p> <p>-Role of provocative testing in patients with chest pain</p>	<p>By the end of this session, learners will:</p> <p>-Risk stratify patients for ACS with undifferentiated chest pain</p> <p>-Describe commonly used decision-making rules for risk-stratification of ACS</p> <p>-Describe the appropriate use of provocative testing for patients with suspected ACS</p> <p>-Understand pharmacologic management of patients with confirmed or suspected ACS</p> <p>-Accurately identify patients who require urgent or emergent cardiology consultation and reperfusion therapy</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Congenital Heart Disease	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and management of cyanotic congenital heart disease</p> <p>-Pathophysiology, diagnosis and management of non-cyanotic congenital heart disease</p> <p>-Review of expected physical exam findings in an infant with congenital heart disease</p> <p>-Use of pharmacologic agents in the management of congenital heart disease</p>	<p>By the end of this session, learners will:</p> <p>-Describe unique changes in newborn anatomy and physiology that lead to the presentation of congenital heart disease</p> <p>-Associate the impact of anatomic (closure of the arterial duct) and physiologic changes (decrease in pulmonary vascular resistance) in the newborn with the presentation of congenital heart disease in infants</p> <p>-Plan stabilizing initial care of infants presenting with congenital heart disease including discernment of key aspects to be communicated to the pediatric cardiologist</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Brief Resolved Unexplained Event (BRUE)	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and management of BRUE</p> <p>- Indications for admission</p>	<p>By the end of this session, learners will:</p> <p>-Define BRUE and its risk factors</p> <p>-Differentiate possible causes of BRUE in the young infant</p> <p>-Determine initial appropriate evaluation of BRUE based on history and physical examination. Which patients require a complete evaluation and which can be observed?</p> <p>-Recognize physical abuse as a potential cause of BRUE</p> <p>-What is SIDS and what are the risk factors for this diagnosis</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Left Ventricular Assist Devices (LVADs)	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Mechanics of LVADS</p> <p>-Common complications associated with LVAD and their diagnosis/management</p> <p>-How to assess LVADs for pump failure</p> <p>-Management of unstable patients with LVAD</p>	<p>By the end of this session, learners will:</p> <p>-Identify the indications for LVAD placement</p> <p>-Discuss the physiology behind the function of an LVAD</p> <p>-Review what should be done first when a patient presents with an LVAD</p> <p>-Organize the thought process about how to evaluate a patient with an LVAD according to the most common complications: bleeding, drive line infection, device complications (thrombosis), and RV failure and hypo perfusion</p> <p>-Consider your treatment alternatives when a patient with an LVAD is coding</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Pericardial Disorders	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Pathophysiology, diagnosis and management of acute pericarditis</p> <p>-Pathophysiology, diagnosis and management of cardiac tamponade</p>	<p>By the end of this session, learners will:</p> <p>-Identify the most common etiologies of acute pericarditis</p> <p>-Be able to identify the clinical features associated with pericardial tamponade and acute pericarditis</p> <p>-Be able to identify the diagnostic findings associated with cardiac tamponade and acute pericarditis</p> <p>-Be able to appropriately treat acute pericarditis and pericardial tamponade</p> <p>-Discuss the pathophysiology associated with cardiac tamponade</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), ultrasound (PC12), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Aortic Dissection	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	-Pathophysiology, diagnosis and management of aortic dissection	<p>By the end of this session, learners will:</p> <ul style="list-style-type: none"> <li>-Review pathophysiology, diagnosis and treatment of aortic dissection</li> <li>-Discuss the different classification systems and the different associated management strategies- surgical v conservative medical management</li> <li>-Critically discuss the various imaging modalities available to diagnose aortic dissection</li> <li>-Discuss medication options and goals for heart rate and blood pressure management</li> </ul>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Syncope	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	-Pathophysiology, diagnosis and management of syncope	<p>By the end of this session, learners will:</p> <ul style="list-style-type: none"> <li>-Describe common causes of syncope and classification system</li> <li>-Understand what history and physical examination data help risk-stratify patients with syncope</li> <li>-Discuss what diagnostic testing help risk-stratify patients with syncope</li> <li>-Describe the most serious causes of syncope</li> <li>-Correctly identify which patients should be admitted after a syncopal episode of unclear cause</li> </ul>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



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Topic	Recommended educational strategy	Educational content	Objectives	Learners	Timing, resources needed (space, instructors, equipment, citations of JETem pubs or other literature)	Recommended assessment, milestones addressed
Resuscitation and ACLS	<p>“Flipped” classroom discussion of pre-reading material, case discussions, and discussion questions</p> <p>Encourage participants to share clinical experiences to enhance discussion</p> <p>30-45 minutes for case and content discussion</p>	<p>-Review ACLS algorithms</p> <p>-Review importance of team dynamics and communication during resuscitations</p>	<p>By the end of this session, learners will:</p> <p>-Learn the various causes of cardiac arrest and how to treat the reversible causes</p> <p>-Describe the interpersonal and interdisciplinary interactions that are important in a successful resuscitation</p> <p>-State the sequence of events during ACLS</p> <p>-Identify important factors in optimizing chest compressions</p> <p>-Describe the use of adjunctive tools (intubation, arterial lines, etc.) in cardiac arrest</p>	PGY-1 PGY-2 PGY-3 Medical Students Faculty	<p>Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion</p> <p>Instructors: 1-2 faculty members or content experts. Recommended senior resident discussion leader</p> <p>Timing: small group discussions involve no more than 15 learners and last 45-60 minutes</p>	<p>Milestone: Emergency stabilization (PC1), differential diagnosis (PC4), pharmacology (PC5), ultrasound (PC12), medical knowledge (MK)</p> <p>Assessment: Faculty evaluation of resident participation during small group activities. See attached appendix for suggested rubric.</p> <p>Evaluation: Resident evaluation of small group session content and facilitators. See appendix for suggested evaluation. Yearly program evaluation of overall small group component.</p>



## Appendix A: Pulmonary Embolism (PE)

### Objectives

1. Review pathophysiology, diagnosis and treatment of pulmonary embolism.
2. Discuss the role of bedside ultrasound in the diagnosis of massive pulmonary embolus.
3. Review and discuss the indications for various imaging modalities, systemic thrombolytic administration, and catheter assisted thrombolytic administration.
4. Critically discuss the ADJUST-PE study assessing age-adjusted d-dimer cutoff levels to rule out PE.
5. Critically discuss the use of trimester adjusted d-dimer in pregnancy to clinically rule out PE.
6. Discuss what patients, if any, diagnosed with an acute PE in the emergency department (ED) you would consider for outpatient anticoagulation versus admission.
7. Debate the role of heparin versus enoxaparin and warfarin versus newer oral anticoagulants in the treatment of PE.

### Case Studies

**Case 1:** A 30-year-old female presents to the emergency department complaining of acute onset dyspnea and palpitations which began five hours prior to arrival. The patient was recently evaluated by orthopedics and placed in a knee brace for an injury she sustained while playing soccer two weeks prior. Shortness of breath is worse with exertion and she has associated pleuritic chest pain. She also states that some swelling persists in her injured leg at the knee, shin, and calf. She has no medical problems and her only medication is an oral contraceptive. On examination, the patient appears anxious and has an initial heart rate of 120 and a blood pressure of 146/54 with an oxygen saturation of 96%. Shortly after beginning the emergency department (ED) evaluation, the patient became more tachypneic and her oxygen saturation dropped to 92% on 2L nasal cannula. Her repeat blood pressure was 88/54. The BP remained low despite a fluid bolus. Bedside cardiac ultrasound demonstrates right ventricular dilation and hypokinesis (if the instructor would like an example ultrasound image for learners, one can be found here: <http://jetem.org/tapse/>).

### Question Prompts:

1. What is the most appropriate emergency management of this patient?
  - a. This patient presents with a story that is high risk for pulmonary embolism. She initially is somewhat stable in that her blood pressure is normal but she is tachycardic and tachypneic. Her bedside ultrasound shows right heart dilation indicating significant strain. Given her high probability for PE, she should be empirically started on heparin. Labs such as complete blood count (CBC), basic metabolic panel (BMP), brain natriuretic peptide (BNP) and troponin should be obtained. She should have an electrocardiogram (ECG). Chest X-ray (CXR) would make the initial evaluation complete but it should not delay definitive imaging.



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2. What other imaging is necessary in the ED, if any?
  - a. If she remains stable, then she should undergo computed tomography (CT) scanning of the chest with a PE protocol. In this case she became hypotensive and more hypoxic. It could be argued that she is such high suspicion for PE, along with corroborating evidence of right heart strain and hypotension (lasting longer than 10 minutes), she qualifies for ED-initiated thrombolytics without further imaging. It could also be argued that in a hospital with a CT scanner in the ED, that preparation for thrombolysis can be initiated and an attempt made to get a CT with the physician providing a real-time interpretation of the CT. At that time, thrombolytics can be given if a PE is confirmed. Specialists (usually interventional radiology or vascular surgery) can also be contacted to assess the possible utility of catheter-assisted thrombolytics or open embolectomy for massive PE. In this particular case, this patient should get systemic thrombolytics initiated by the ED, preferably after a CT, but without if she is unstable or the CT is far away.
3. What are the indications for systemic thrombolytics? Catheter assisted thrombolytics?
  - a. Indications for tissue plasminogen activator (tPA) in PE is evidence of massive PE with **Hemodynamically Instability**: Systolic blood pressure (SBP) < 100 mmHg for >15 minutes (not due to a cause other than PE [such as sepsis, arrhythmia, hypovolemia]), syncope, shock index >1 (heart rate/systolic blood pressure), respiratory distress.
  - b. Indications for tPA via catheter include: Massive PE not getting systemic thrombolytics and possibly some submassive PEs with right heart strain and/or positive biomarkers.
4. Would management change if the patient maintained stable vital signs?
  - a. Yes. CT would be indicated and based on the findings and evidence of right heart strain, consideration should be given to using catheter directed thrombolytics after consultation with an appropriate specialist (may be vascular or interventional radiology depending on the hospital).
5. Which patients with a PE can be sent home?
  - a. Low risk patients who are simplified PE Severity Index (sPESI) or Hestia negative and look well can be sent home on outpatient anticoagulation with a Xa inhibitor or low molecular weight heparin (LMWH) and Coumadin.
    - i. sPESI – Patients must meet all the following to be considered low risk (1.1% risk of death, 1.5% recurrent venous thromboembolism or non-fatal bleeding): Age less than 80, no history of cancer, no history of chronic cardiopulmonary disease, heart rate less than 110, systolic blood pressure greater than 100 mm Hg, and oxygen saturation above 90%.
    - ii. Hestia Criteria – Must meet all following criteria to be considered low-risk (0% mortality, 2% recurrent venous thromboembolism): Hemodynamically stable, no need for thrombolysis/embolectomy, no active bleeding, less than 24 hours requiring supplemental oxygen to keep pulse oximetry over 90%, PE not diagnosed while on anticoagulation, pain controlled, no other medical or social reason for admission, creatinine clearance greater than 30 mL/min.
    - iii. Low-risk patients that are agreeable to outpatient management could be started on lovenox while bridging to coumadin, or started on a newer oral anticoagulant such as rivaroxaban.



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1. Benefits of the newer oral medications include no need for frequent coagulation testing. These decisions are best made in consultation with their follow-up physician or a hematologist.
- iv. High-risk patients should be admitted to the hospital on a heparin infusion. If the patient has a normal creatinine function, they could alternatively be started on lovenox injections. The benefit is easier management for nursing staff and more reliable anticoagulation.

**Case 2:** A 70-year-old male presents to the emergency department for evaluation of chest pain and shortness of breath. He has a history of hypertension and hypercholesterolemia but denies cardiac history. He complains of diffuse chest pain that is worse with breathing and with coughing, and also has some mild associated shortness of breath. He has had a cough and congestion for the past week where he has intermittent paroxysms of violent coughing. Vital signs are entirely normal and his pain is reproducible on palpation. Lungs are diminished but clear. CXR and ECG performed in triage were reviewed and normal. Given the pleuritic chest pain, PE enters the differential diagnosis; therefore, PE Rule out Criteria (PERC) is unable to be utilized, so a d-dimer is ordered in association with other laboratory tests. All laboratory studies are negative except for the d-dimer which returned elevated at 670 $\mu$ g/L fibrinogen-equivalent units (FEU) (normal is <500 $\mu$ g/L).

### Question Prompts:

1. What is the likely diagnosis for this patient?
  - a. Bronchitis
2. After critically reviewing the ADJUST-PE study, is it likely for this patient to have a PE? Does he warrant imaging?
  - a. It is unlikely that the patient has a PE. Using age-adjusted d-dimer cutoffs allowed for fewer CTs with increase in the number of missed PEs.
  - b. Per the ADJUST-PE study, the age-adjusted d-dimer is the patient's age multiplied by 10 in people 50 years or older, so in this patient the age-adjusted d-dimer would be 700.

**Case 3:** A 23-year old female presents to the emergency department complaining that her "chest hurts" when she breathes. The pain has gotten progressively worse throughout the day and she has since developed some shortness of breath. She is 16 weeks pregnant. Her vital signs and physical examination are normal.

### Question Prompts:

1. How does pregnancy play into the pre-test probability that this patient has a PE?
  - a. Although pregnancy is traditionally considered a high-risk condition for PE, recent evidence shows that 2/3 of PEs occur in the 3<sup>rd</sup> trimester and in the post-partum period. Patients who are pregnant and are being considered for a PE work-up who are in the first and second trimester fall into the category of "low-risk" as the result of their pregnancy.
2. What are the main differences between the American Thoracic Society guidelines and the Kline Guideline for evaluating PE in the pregnant patient?



# DIDACTICS AND HANDS-ON CURRICULUM

- a. Kline's algorithm incorporates the use of d-dimer into the decision matrix. It is a big step to avoid imaging.
3. How does the interpretation of the d-dimer change through pregnancy?
  - a. First trimester: 750 µg/L
  - b. Second trimester: 1000 µg/L
  - c. Third trimester: 1250 µg/L

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## Appendix B: Peripheral Vascular Disease

### Objectives

1. Describe the difference in the pathophysiology of atherosclerosis, arterial thrombus formation, atheroembolism, thromboembolism, aneurysms, and fistulae.
2. List the risk factors associated with acute limb ischemia.
3. Discuss the diagnostic modalities used in the acute evaluation of limb ischemia or claudication.
4. Describe treatment options for limb ischemia in the emergency department (ED).
5. Discuss the clinical significance of the Rutherford classification related to immediate management.
6. Describe the three phases of Raynaud's phenomenon, and describe the ED diagnosis and management.

### Case Studies

**Case 1:** A 72-year-old male with a history of paroxysmal atrial fibrillation presents for evaluation of left lower extremity (LLE) numbness and weakness. The patient reports the onset of his symptoms five hours prior to arrival. He denies slurred speech, visual changes, facial and upper extremity sensory and motor deficit. He denies right lower extremity symptoms.

Triage vital signs: Temperature (T) 99.7°F Oral, heart rate (HR) 82, blood pressure (BP) 129/76, respiratory rate (RR) 24, blood oxygen saturation level (SpO<sub>2</sub>) 98% on room air.

Pertinent Exam: Left posterior tibial (PT) and dorsalis pedis (DP) pulses non-palpable, but venous phase is present on Doppler. The skin is cool and mottled with generally decreased light touch sensation and weakness of the foot. Heart is irregular but not tachycardic. ankle brachial index (ABI) on the right=0.85, ABI on the left=0.3.

### Question Prompts:

1. What is the differential for this patient?
  - a. Peripheral vascular disease (PVD), acute limb ischemia, thromboembolism, claudication, cerebrovascular accident (CVA), deep vein thrombosis (DVT), neuropathy
2. What risk factors are present in this patient for PVD? What features are consistent with PVD?
  - a. Risk factors include: age, smoking, diabetes, other vascular disease, male gender, African American ethnicity, and dyslipidemia. This patient has several risk factors for PVD including atrial fibrillation and male gender. His physical exam findings including a cool, mottled left leg with decreased ABIs is consistent with PVD.
  - b. Helpful Definitions:
    - i. **Atherosclerosis** is the process of fibrofatty plaque formation within the intimal layer of medium to large arteries (causes compromise of the arterial lumen).



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- ii. **Arterial thrombus** is in-situ formation of a blood clot—usually related to rupture of an atherosclerotic plaque (may or may not be fully occlusive). Arterial embolism includes thromboembolism and atheroembolism.
  - iii. **Thromboembolism** refers to emboli originating from a cardiac thrombus; these are the cause of most arterial emboli (~85%).
  - iv. **Atheroembolism** is the process of microemboli from proximal plaques or aneurysms translocating and occluding distal end arteries (“blue toe syndrome”).
  - v. **Aneurysm** is the abnormal dilatation of an intact arterial wall (which can lead to thrombus formation or rupture).
  - vi. **Fistulae** are abnormal communications between arteries and veins (congenital, traumatic, inflammatory, neoplastic, or iatrogenic).
  - c. **Limb ischemia** classically presents with the five P’s: pain, pallor, paresthesias, pulselessness, and paralysis (#6 could be poikilothermia: difference in temperature between proximal and distal limb or affected and unaffected limb). Absence of palpable and Doppler pulses is the true hallmark of this disease though.
  - d. **Claudication** is the pain experienced due to decreased but not absent flow, often brought on with activity and relieved with rest.
3. What is the Rutherford classification of this patient?
- a. Knowing the Rutherford classification system can be useful when talking with consultants. Because this patient has absent arterial phase, weakness and sensory loss, he would be category IIb and would require immediate revascularization to salvage the limb.
  - b. Rutherford classification system for acute limb ischemia:

Category	Description	Sensory Loss	Muscle Weakness	Arterial Doppler	Venous Doppler
I Viable	not immediately threatened	none	none	audible	audible
IIa Marginally Threatened	salvageable if promptly treated	minimal or none	none	inaudible	audible
IIb Immediately Threatened	salvageable with immediate revascularization	more than toes, associated rest pain	mild, moderate	inaudible	audible
III Irreversible	major tissue loss or permanent nerve damage inevitable	Profound, anesthetic	profound, paralysis	inaudible	inaudible

4. What further diagnostic testing is indicated? How should the ABI be interpreted?
- a. **Ankle-brachial index (ABI)** is the ratio of the ankle systolic blood pressure divided by the brachial systolic pressure detected with a Doppler probe. In patients with no or mild-



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moderate symptoms, an ABI of  $<0.90$  has a high degree of sensitivity and specificity for peripheral arterial disease (PAD). ABI results can be interpreted as follows:

- i. The normal ABI is  $\geq 0.9$  to as high as 1.3.
  - ii. An ABI  $>1.3$  suggests the presence of calcified vessels and the need for additional vascular studies, such as pulse volume recordings, measurement of the toe pressures and toe-brachial index, or arterial duplex studies.
  - iii. An ABI  $\leq 0.9$  is diagnostic of occlusive arterial disease in patients with symptoms of claudication or other signs of ischemia and has 95 percent sensitivity (and 100 percent specificity) for detecting arteriogram-positive occlusive lesions associated with  $\geq 50$  percent stenosis in one or more major vessels.
  - iv. An ABI of 0.4 to 0.9 suggests a degree of arterial obstruction often associated with claudication.
  - v. An ABI below 0.4 represents multilevel disease (any combination of iliac, femoral or tibial vessel disease) and may be associated with non-healing ulcerations, ischemic rest pain or pedal gangrene.
- b. **Duplex scanning** can very useful in identifying proximal arterial disease. Angiography is the gold standard of PVD diagnosis, though rarely needed in the emergency department (ED) to determine presence of disease. It still has much utility in planning for revascularization.
5. What treatment modalities should be considered?
- a. Treatment of acute limb ischemia requires prompt involvement of a vascular surgeon. Heparin anticoagulation is beneficial barring contraindications. Definitive treatment options include: angioplasty, bypass grafting, or thrombolytic therapy.
  - b. Patients with chronic arterial insufficiency are at risk for gangrene and ulcer formation of the distal extremities.

**Case 2:** A 28-year-old female who has no past medical history presents with discoloration and discomfort in her fingers of both hands. This started after she was working outside in the cold (ambient temp = 30°F). She denies any trauma to her hands and was wearing gloves while outside. She has some tingling in the fingers but denies loss of sensation or weakness.

Triage VS: T 99.7°F Oral, HR 77, BP 129/70, RR 16, SpO<sub>2</sub> 98% on room air.

Pertinent Exam: Well-appearing female in no acute distress. Hands show bilateral symmetric distribution of acral cyanosis. The skin is intact. Nailbeds and nails are normal in appearance. Distal light touch sensation is intact with normal strength testing. Radial pulse is 2+.

## Question Prompts:

1. What is the differential for this patient?
  - a. Consider Raynaud's phenomenon, Buerger's disease, cold injury as well as vascular occlusion although this would be less likely.
2. How does Raynaud's differ in character from Buerger's disease or cold injury (frostbite)?



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- a. **Raynaud's disease** is an abnormal vasomotor response in the distal small arteries with unknown cause. This is the most common type of vasospastic disease. Diagnostic criteria include: 1) precipitated by cold or emotion; 2) bilateral distribution; 3) absent or minimal gangrene; 4) no other identifiable vascular process is present; and 5) occurring >2 years.
  - b. Thromboangiitis obliterans, also called **Buerger's disease**, is a nonatherosclerotic, segmental, inflammatory disease that most commonly affects the small to medium-sized arteries and veins of the extremities. This is classically seen in relatively younger patients who smoke with manifestations in the fingers.
  - c. What are the three phases of Raynaud's phenomenon?
    - i. **WHITE** (complete arterial closure and cessation of blood flow)
    - ii. **BLUE** (marginal but inadequate return of flow leading to cyanosis)
    - iii. **RED** (resolution of arterial spasm allowing flow and resultant reactive hyperemia)
3. What clinical features differentiate primary versus secondary Raynaud's? What are some of the etiologies which can precipitate secondary Raynaud's?
- a. Primary Raynaud's (also called Raynaud's disease) is more common. It is not secondary to or the result of a concomitant medical condition and tends to be less severe. Secondary Raynaud's (Raynaud's phenomenon) is the result of an underlying medical condition and tends to be more severe. Causative conditions include connective tissue disease, vascular disease (i.e. atherosclerosis), medications (i.e. beta blockers), injury to hands/feet, smoking or carpal tunnel syndrome.
4. What is the ED management of Raynaud's?
- a. In general, supportive care is effective in managing patients suffering from Raynaud's that present to the ED. If caused by cold, warm the extremity. It is important to educate the patient to avoid potential triggers such as rapidly changing temperatures. In recurrent cases, some patients may be started on calcium channel blockers.

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## Appendix C: Cardiac Arrhythmias

### Objectives

1. List and compare different narrow complex tachycardic arrhythmias.
2. List risk factors and complications of atrial fibrillation.
3. Describe how to differentiate Wolff-Parkinson-White (WPW) from other narrow complex tachycardias.
4. Discuss initial evaluation and management of patients presenting with atrial fibrillation with rapid ventricular response, including cardioversion, rate and rhythm control medications, and anti-coagulation.
5. Discuss indications for atrial fibrillation in patients with atrial fibrillation.
6. Describe management of wide complex tachycardia.
7. Define the following: Premature ventricular complexes (PVCs), ventricular bigeminy, ventricular trigemini, ventricular couplets, ventricular escape beats, ventricular tachycardia (VT); non-sustained and sustained, monomorphic versus polymorphic VT, accelerated idioventricular rhythm and ventricular flutter.
8. Describe Brugada's syndrome.
9. Discuss the Vaughan Williams classes of medications that are used to treat dysrhythmias, the mechanism, and name a medication in these classes.

### Case Studies

**Case 1:** A 65-year-old female presents with a “rapid heart rate” that was noticed today during her routine visit with her physician. She had no symptoms. The ECG is irregular, narrow (QRS < .12), and a rate greater than 150.

### Question Prompts:

1. What are the potential arrhythmias in this patient?
  - a. Consider atrial fibrillation (AF), atrial flutter, multifocal atrial tachycardia (MAT), paroxysmal atrial tachycardia (PAT).
  - b. Atrial fibrillation is the most common arrhythmia and the risk increases with age. Most common underlying disorders are hypertensive heart disease and coronary artery disease (CAD). It is associated with CAD, congestive heart failure (CHF), obstructive sleep apnea (OSA), hypertension (HTN), pulmonary embolism (PE), hyperthyroidism, alcohol use, Wolff-Parkinson-White (WPW), valvular heart disease and shock states. The rapid firing comes from pulmonary veins and other tissues near these areas. These are the targets of ablation.



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- c. The usual rate of atrial fibrillation is 140 to 180, so if slower consider medications on board, ischemia, hyperkalemia, hypothermia or digoxin toxicity. If faster rates consider sympathomimetics, PE, shock, or WPW (especially if > 200).
2. What are the complications of AF?
  - a. Cardiac output can be reduced due to the lack of atrial kick. Risk of embolic stroke is as high as 5% per year. This is especially problematic in patients with chronic atrial fibrillation.

**Case 2:** The above patient is admitted and eventually successfully discharged in a sinus rhythm but scheduled for ablation. On the day of her ablation, she presents to the emergency department with palpitations. She is already anticoagulated and knows she went into this rhythm an hour before presenting. Her heart rate is from 100 to 120.

### Question Prompts:

1. What should you do?
  - a. Rule out acute coronary syndrome (ACS), CHF and PE but send her to electrophysiology (EP) for ablation without cardioverting her since being in atrial fibrillation actually helps with her ablation. Now the electrophysiology physicians can better localize where the ectopic foci are originating which is usually in the pulmonary veins.
2. What would make you worry about Wolff-Parkinson-White (WPW) or the presence of an accessory pathway? Explain the difference between orthodromic and antidromic.
  - a. Short PR interval (< 0.12), QRS duration > 0.10 and a slurred QRS upstroke (delta wave) are the classic triad but usually T waves are opposite the QRS deflection. There are a number of possible accessory pathways.
  - b. Orthodromic tachycardia refers to conduction anterograde through the atrioventricular (AV) node and retrograde through the accessory pathway, and the QRS is usually narrow. Treatment of orthodromic tachycardia is the same as for paroxysmal supraventricular tachycardia (PSVT). Giving adenosine in these patients will stop the anterograde conduction through the AV node and stop the tachyarrhythmia.
  - c. Antidromic tachycardia refers to anterograde conduction through the **accessory pathway** and retrograde conduction through the AV node. Here the QRS is usually wider, often with a delta wave. Procainamide or amiodarone may be the drugs of choice for symptomatic stable patients. Giving adenosine in these patients is potentially very dangerous, because blocking the AV node will allow unchecked rate through the accessory pathway. This can be seen when there is atrial fibrillation with antidromic tachycardia through the accessory pathway. With the AV node blocked, all electrical signals will transmit via the accessory pathway and send the patient into ventricular fibrillation!
    - i. The key take away: Patients with an irregular, wide complex tachycardia should get procainamide or synchronized cardioversion.
3. What are the criteria for emergency cardioversion of AF?
  - a. Active ischemia (symptomatic, ie, angina, or ECG evidence)
  - b. Evidence of organ hypoperfusion (cold clammy, confusion, acute kidney injury)



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- c. Severe manifestations of heart failure (pulmonary edema)
- d. The recommend dose for AF is 200 J
- 4. What pharmacologic treatment would you recommend for AF? Who could be cardioverted?
  - a. First treat contributing or triggering conditions. In a stable patient with AF for longer than 48 to 72 hours use rate control medications (calcium channel blockers, beta blockers, amiodarone and digoxin if low ejection fraction). Agents slowing AV conduction (calcium channel blockers, beta blockers, digoxin) are not recommended if there is a known accessory because of the risk of ventricular fibrillation (i.e. WPW). Intravenous (IV) magnesium sulfate is said to be a third line treatment which may decrease ventricular rate but is also listed as rhythm converting. Evidence shows no long-term benefits to converting the rhythm. Goal of rate slowing is 80 per minute if unstable and 110 if stable. If blood pressure (BP) is low consider giving calcium before Cardizem.
  - b. Conversion medications include procainamide, amiodarone, flecainide, propafenone, and ibutilide with conversion rates of 50% to 70%. It is recommended to use these agents after the rate has been slowed to 100 to 120. If no structural heart disease, then flecainide or propafenone are recommended. The conversion rate of these meds is 50% to 70% versus 89% for electrical cardioversion. The provider should weigh the risks of sedation for cardioversion versus the adverse effects and decreased efficacy of medication. For unstable patients, electrical cardioversion is the first choice. In the stable patient who would prefer to avoid sedation, chemical cardioversion can be offered. Most emergency department (ED) patients can tolerate etomidate sedation well, and because of this, electrical cardioversion is most commonly used.
  - c. Preventive or suppressive medications, such as flecainide or propafenone, are not recommended for patients who are cardioverted and will be discharged from the ED.
  - d. Only cardiovert stable patients if there is a clear onset less than 48 hours or if the patient has been on anticoagulation for at least 3 weeks.
- 5. Who should be anticoagulated and how should the CHAD2VASC and HASBLED scoring systems be used?

C	Congestive heart failure	1
H	Hypertension	1
A <sub>2</sub>	Age>74	2
D	Diabetes mellitus	1
S <sub>2</sub>	Prior stroke, TIA, embolism	2
V	Vascular disease	1
A	Age 65-74	1
Sc	Female	1

- a. HAS-BLED – get one point for each: uncontrolled hypertension, renal disease, liver disease, stroke history, prior major bleeding, labile INR, age>65, medications (ASA, clopidogrel, NSAIDs), Alcohol use.



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- b. Anticoagulation reduces risk of embolization by 70%. Aspirin alone is believed to confer no benefit in AF patients. These scores are used to determine the lifelong risk of stroke in AF patients and a score of 2 or more requires anticoagulation unless the risk of bleeding is too high. If the HASBLED score is greater than 3 then the risk of bleeding may be too high.
6. Should you anticoagulate those patients successfully cardioverted with AF of less than 48 hours duration?
  - a. There is no clear consensus. There are differing philosophies and CHADS2VASC score can be used for risk stratification. If anticoagulation is employed, many recommend initiating this with heparin or low molecular weight heparin (LMWH) prior to cardioversion. Theoretically, atrial stunning can occur after cardioversion, which is a reason given by anticoagulation proponents to initiate anticoagulation even if AF is less than 48 hours duration.
7. Do patients with atrial fibrillation need an ablation and what are the indications?
  - a. Indications:
    - i. Symptomatic AF refractory or intolerant to at least one Class 1 or 3 antiarrhythmic medications
      1. Paroxysmal: Catheter ablation is recommended (this is the only class I, level A recommendation).
      2. Persistent: Catheter ablation is reasonable (IIa recommendation).
      3. Longstanding Persistent: Catheter ablation may be considered (IIb recommendation).
    - ii. Symptomatic AF prior to initiation of antiarrhythmic drug therapy with a Class 1 or 3 antiarrhythmic agent (all level II recommendations)
      1. Paroxysmal: Catheter ablation is reasonable
      2. Persistent: Catheter ablation may be considered
      3. Longstanding Persistent: Catheter ablation may be considered

**Case 3:** A 50-year-old female with a rapid narrow regular complex tachycardia and no chest discomfort, but she can feel a rapid pounding in her heart. Her BP and pulse oximetry are normal and heart rate is 160, narrow and regular.

### Question Prompts:

1. What is the diagnosis and potential treatment for stable and unstable patients?
  - a. The differential includes sinus tachycardia, sinoatrial nodal reentrant tachycardia (SANRT), atrioventricular reentrant tachycardia (AVNRT), atrial tachycardia, atrial flutter.
    - i. Sinus tachycardia – treat the underlying cause
    - ii. AVNRT – Direct current (DC) cardioversion for instability (hypotension) or adenosine then cardioversion
    - iii. Atrioventricular Reentrant Tachycardia (AVRT) – DC cardioversion if unstable but if stable try vagal maneuvers, adenosine and consider calcium channel blockers or beta blockers



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2. Now the patient in Case 3 has the same presentation but is in a regular wide complex tachycardia. How can one differentiate between ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant conduction? Discuss the differential diagnosis and management.
  - a. There are specific criteria such as Wellen's, Griffith's and Brugada's criteria.
  - b. VT suggested by:
    - i. History of heart disease, implanted cardioverter-defibrillator (ICD), age greater than 35, history of long QT or medications that cause long QT
    - ii. Labs with low potassium (K), low magnesium (Mg), ST depression on ECG
    - iii. ECG with atrioventricular (AV) dissociation on ECG, wider QRS > 140 to 160 ms, concordance, fusion beats or captures
    - iv. Chest X-ray (CXR) with CHF or cardiomegaly
  - c. If unclear about VT versus SVT then treat as VT because it is the most common cause of wide complex tachycardia. The differential includes monomorphic VT, SVT with aberrant conduction, antidromic AVRT, SVT with paced ventricular response.
  - d. If the patient is unstable, then DC cardioversion. Start at 100J synchronized cardioversion if the QRS and T wave can be established. If the QRS cannot be distinguished from the T wave then defibrillate at 120 to 200 J if biphasic or 360 J if monophasic. If stable check the ECG to determine if VT or SVT. For VT consider amiodarone, lidocaine or procainamide.
  - e. If this is AVNRT, AVRT, or an SVT, then vagal maneuvers, adenosine, intravenous (IV) calcium (Ca) channel blockers, or beta blockers.
  - f. If unclear diagnosis and wide complex tachycardia present that is regular and monomorphic, then can try adenosine but other drugs are not recommended.
  - g. If pacemaker related, then try a magnet so the pacer can overdrive the rapid arrhythmia.

### Additional Discussion points as time allows or for resident self-study

#### Question Prompts:

1. Define the following: Premature ventricular contraction (PVC), ventricular bigeminy, ventricular trigeminy, ventricular couplets, ventricular escape beats, non-sustained and sustained VT, monomorphic versus polymorphic VT, accelerated idioventricular rhythm and ventricular flutter.
  - a. PVC – need wide complex greater than 0.16, may look like right bundle branch block (RBBB) or left bundle branch block (LBBB), full compensatory pause
  - b. Bigeminy – PVC after every sinus beat
  - c. Trigeminy – PVC after 2 sinus beats
  - d. VT – three or more PVCs in a row at a rate greater than 100
    - i. Non-sustained VT is less than 30 seconds
    - ii. Sustained VT is greater than 30 seconds
    - iii. Monomorphic VT – all the QRS complexes have the same morphology
    - iv. Polymorphic VT – all the QRS complexes have different morphologies
  - e. Accelerated idioventricular rhythm (AIVR) = ventricular rhythm at 60 to 100
  - f. Ventricular flutter = VT at rapid rate of 300



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2. Describe Brugada's syndrome.
  - a. Inherited disorder of sodium channels
  - b. ST elevation in V1 and V2 with a notched or coved appearance of the ST segment with or without RBBB (we recommend instructors pull up images from google to show learners examples).
3. The sinoatrial (SA) node is located in the posterior right atrium and is usually the dominant pacemaker. It is innervated by both parasympathetic and sympathetic nervous systems. Describe ways the SA node can be influenced to speed or slow the heart rate.
  - a. Increased vagal tone and hypothermia can slow the heart rate. Vagal tone can be increased by ischemia (right coronary artery [RCA] or left coronary artery [LCA]). It can also be increased by medications such as beta blockers, calcium channel blockers, and endocrine problems such as low thyroid states and low adrenal states.
  - b. Decreased vagal tone, hyperthermia, and increased sympathetic influence can speed the heart rate. Sympathomimetic meds and vagolytic medications can increase heart rate as can hyperthyroid states.
4. Describe the 3 main mechanisms for dysrhythmia formation.
  - a. Altered automaticity phase 4 depolarization in non-pacemaker cells: VT after myocardial infarction, other pacemaker takes over as in idioventricular rhythm, atrial or junctional tachycardia with digitalis toxicity, catecholamine excess.
  - b. Reentry, a path of rapid conduction exists: Narrow complex tachydysrhythmias, SVT, VT and bigeminy.
  - c. Triggered dysrhythmias due to after depolarizations that are propagated and can continue, such as Torsade's.
5. Discuss the Vaughan Williams classes of medications that are used to treat dysrhythmias, the mechanism and name a medication in these classes.
  - a. Class I effect sodium (Na) fast channels and slow depolarization
  - b. Class IA moderate slowing of depolarization and prolong repolarization - Quinidine (atrial fibrillation and Brugada) and Procainamide (VT and SVT)
  - c. Class IB minimal slow depolarization and shorten repolarization - Lidocaine (ventricular dysrhythmias)
  - d. Class IC marked slow depolarization and slow repolarization - Flecainide (ventricular and supraventricular dysrhythmias) and propafenone (atrial fibrillation)
  - e. Class II beta blockers - Propranolol, esmolol, metoprolol
  - f. Class III anti-fibrillatory agents - Amiodarone
  - g. Class IV calcium channel agents - Diltiazem and verapamil
  - h. Miscellaneous - Digitalis, Adenosine, Magnesium
6. What are the symptoms or signs that make a dysrhythmia unstable?
  - a. Hypotension, chest pain consistent with ischemia, dyspnea or pulmonary edema, altered mental status

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## Appendix D: Congestive Heart Failure

### Objectives

1. Differentiate between congestive heart failure (CHF) with volume overload and flash pulmonary edema with normal volume and high afterload.
2. Discuss available therapies for management of CHF with flash pulmonary edema – nitroglycerin (NTG), diuretics, intravenous angiotensin converting enzyme (ACE) inhibitors, dobutamine, morphine.
3. Discuss the use of bilevel positive airway pressure (BiPAP) vs. continuous positive airway pressure (CPAP) for acute pulmonary edema.

### Case Studies

**Case 1:** A 58-year-old male with ischemic cardiomyopathy presents with worsening shortness of breath over the last few days. He has had a 15-pound weight gain over the last week. He is very dyspneic with an increased respiratory rate (RR) of 24 and a blood pressure (BP) of 140/85 and a heart rate (HR) of 100.

### Question Prompts:

1. Describe initial management of a patient with volume overload and pulmonary edema.
  - a. Airway/breathing/circulation (ABC's), intravenous (IV) line, oxygen, monitor. Initiate BiPAP early if the patient appears to have an increased work of breathing. The use of NTG is geared toward decreasing preload. Diuretics have an important role in volume overload and some argue that they should be given after NTG has been started to facilitate renal blood flow.
2. How is this patient different from one with high afterload?
  - a. This patient is a classic volume overload case. Although the initial management is geared toward improving oxygenation and decreasing the work of breathing, if the patient's BP is not severely elevated, afterload reduction is less important initially.

**Case 2:** A 45-year-old male presents with sudden onset of severe shortness of breath after using cocaine. His blood pressure is 250/120. He has no previous medical history other than mild hypertension. He has not used any other drugs. His pulse oximetry is 83% on a non-rebreather.

Physical exam shows a patient in acute distress with a respiratory rate of 30 and a heart rate of 120. He is diaphoretic. He has crackles throughout both lungs in all fields. He has no peripheral edema and the remainder of his exam is normal.



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## Question Prompts:

1. How is this different than volume overloaded patients?
  - a. Acute flash pulmonary edema is not the same thing as decompensated congestive heart failure. This patient has the right volume of fluid in his body; it is just in the wrong place. The pathophysiology of this is acute elevation of afterload that is high enough that his heart is not acutely able to overcome the resistance. The blood backed up into his lungs. His volume is normal.
2. How does one effectively manage this patient?
  - a. If you manage this patient correctly and aggressively there is a very low chance that you will need to intubate him. **You should stand at the bedside until the initial sequence of orders is carried out and the patient is beginning to improve.** Be ready to intubate if the patient won't tolerate the treatments or they tire out. By being very aggressive with medical therapy, only rarely do flash pulmonary edema patient require intubation.
  - b. The first drug is oxygen. It should be delivered by BiPAP. Start at 10/5 (IPAP/EPAP) and work quickly up to 15-20/10. If the normal intrathoracic pressure during breathing is -40 and his mean arterial pressure (MAP) is around 160, then a transthoracic pressure gradient is around 200. This represents the difference between the pressure in the chest and the pressure in the rest of the body. It also represents the workload the heart is working against to push blood forward. If a patient is given a positive pressure during BiPAP of around +10, the transthoracic pressure gradient drops to 150. A lower transthoracic pressure gradient means the heart can pump more efficiently in the "forward direction."
  - c. Judicious use of small doses of midazolam or ketamine may facilitate the BiPAP and tone down the catecholamine release associated with the cocaine use. It may help in general with decreasing the catecholamine surge that can happen from the stress of the event.
  - d. Prior to and during the BiPAP, nitroglycerin needs to be given. Start with giving a sublingual nitroglycerin or even two while waiting for the nitroglycerin drip to arrive from pharmacy. Remember that this catecholamine charged patient will probably have a dry mouth so consider placing a piece of ice or some water under his tongue with the nitroglycerin so it actually dissolves. Nitroglycerin spray may be better in this situation.

A single sublingual dose of nitroglycerin delivers 400 microgram (mcg). One sublingual nitroglycerin every 5 minutes delivers 80 mcg/minute. The bioavailability of nitroglycerin when given sublingual is about 40%, meaning approximately 30 mcg of nitroglycerin is delivered per minute. Consider putting 2 sublingual nitroglycerin at a time into the patient's mouth. (~60 mcg/min).

- e. An alternative "bolus approach" involves using IV NTG boluses as described in EMRAP:
  - i. *"Nitroglycerin comes in bottles of 200 mcg/mL or 400 mcg/mL. Before the nurse spikes the bottle, draw up a 1-2 mg into syringe and push a bolus. You are trying to break the hypersympathetic, hypertensive cycle. If you give 400mcg sublingually, it will take several minutes to reach peak levels and the bioavailability is around 30%.*



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*Rubin Strayer pushes 1mg of nitroglycerin over a few seconds. He starts the drip at 200mcg/min. If they still look terrible, he gives a second 1mg bolus”<sup>1</sup>*

- f. The minimum starting dose of IV nitroglycerin for flash pulmonary edema is 50 mcg per minute. Be aggressive and consider doubling this number very quickly in the course of the resuscitation. Continue to double as needed. At lower doses, nitroglycerin is a pre-load reducing agent. At higher doses it acts as an afterload reducing agent also. The highest approved dose of nitroglycerin according to the Food and Drug Administration is 640 mcg per minute!
- g. The next afterload reducing agent to consider is IV enalapril. The dose is 0.625 mg IV up to 2.5 mg IV.
- h. The final step in this process is to use dobutamine if contractility is an issue and the patient is not responding to the other aggressively administered treatments. Increasing the contractility of the heart while simultaneously causing some vasodilation is a good thing in these patients.
- i. Morphine has no role in the management of heart failure. Studies that have looked at it showed a higher mortality when morphine is given.
- j. Lasix is not indicated during the initial resuscitation, as these patients’ total body volume is normal. It is indicated only in patients who have an abnormal volume of fluid in the body. It is not an effective pulmonary artery dilator.

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## Appendix E: Congenital Heart Disease: Detection in the Undiagnosed Newborn Infant

### Objectives

1. Describe unique changes in newborn anatomy and physiology that lead to the presentation of congenital heart disease (CHD).
2. Compare CHD findings and diagnoses in patients with pulmonary under-circulation versus pulmonary over-circulation.
3. Discuss the impact of anatomic (closure of the arterial duct) and physiologic changes (decrease in pulmonary vascular resistance) in the newborn with the presentation of congenital heart disease in infants.
4. Describe stabilizing initial care of infants presenting with congenital heart disease, including discernment of key aspects to be communicated to the pediatric cardiologist.

### Case Studies

**Case 1:** A 2-week-old term male who was born at home to a Gravida 4, Para 4 (G4P4) mother with a midwife. Birth weight was 7 lbs. 4 oz. Feeding well, breast milk on demand. Patient was referred to the emergency department (ED) from primary care physician (PCP) after 2-week well check.

Mom states color has been a little dusky at times but since he is feeding well and it seemed to be after baths, she did not worry too much.

Vital signs: Heart Rate (HR) 130, Blood Pressure (BP) 74/42, Respiratory Rate (RR) 28, O<sub>2</sub> saturation 85% on Room Air, temperature (T) 99.2, Weight 3.9 kg.

General: Alert and well appearing. No dysmorphic features.

Head/ears/eyes/nose/throat (HEENT): Mucous membranes (MM) dusky, pupils equal, round, reactive to light (PERLL), red reflex x 2.

Chest: Bilateral breath sounds clear.

Cardiovascular (CV): Active precordium, Normal S<sub>1</sub>. S<sub>2</sub> with possible click but not well heard due to a systolic ejection murmur. The murmur is long, high-pitched, grade III/IV systolic crescendo decrescendo murmur, best at left upper sternal border (LUSB) and radiating to the back. No diastolic murmur.

Abdomen: No hepatosplenomegaly (HSM).

Extremities: Cyanosis of feet and hands. Femoral and brachial pulses are equal with no delay.

Chest X-ray (CXR): Decreased PV markings and a “boot shaped” heart.



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Electrocardiogram (ECG):

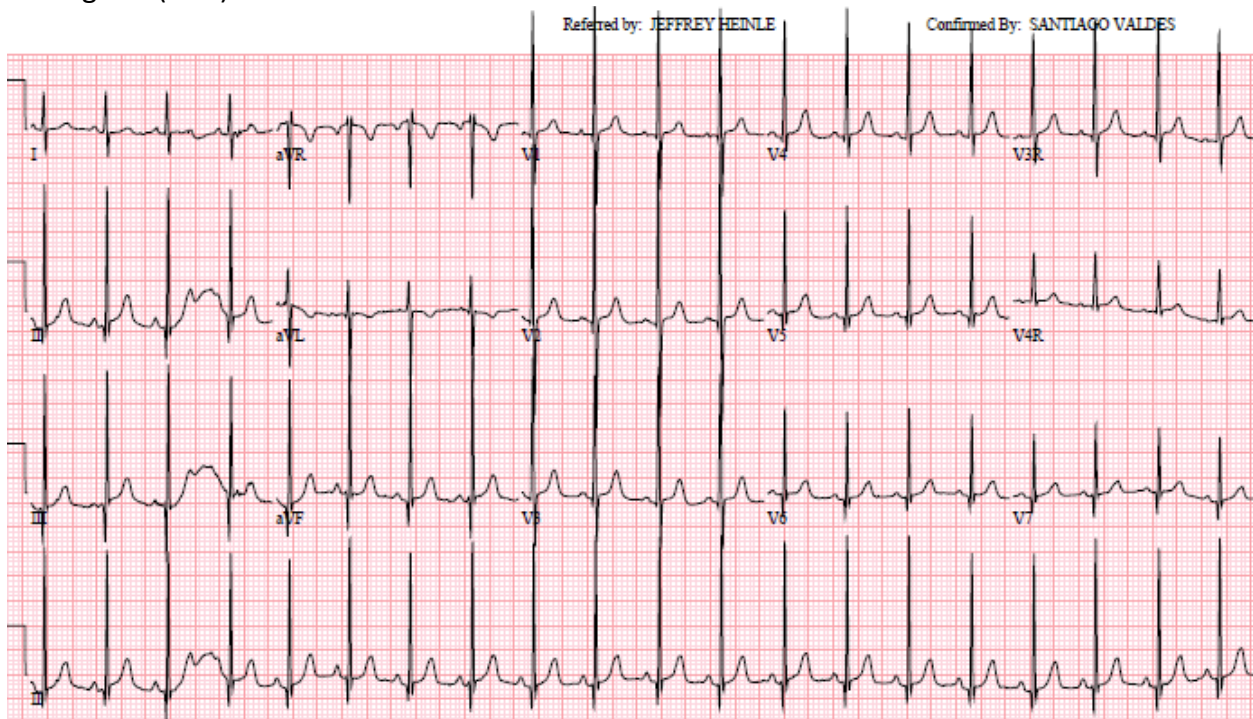


Image courtesy of Beth Bubolz

## Question Prompts:

1. What type of CHD do you expect to discover?
  - a. This baby is presenting with cyanotic heart disease most likely due to tetralogy of Fallot (TOF) or a similar defect, which limits blood flow to the lungs. Tetralogy of Fallot physiology is determined by two items of the tetrad: 1) Pulmonary stenosis and 2) a non-restrictive ventricular septal defect (VSD). The other 2 elements in the tetrad include an overriding aorta, which means the aorta is positioned over the VSD and right ventricular hypertrophy.
2. What anatomic change has occurred to cause it to present at this time?
  - a. As the patent ductus arteriosus (PDA or patent arterial duct) closes, blood flow to the lungs decreases due to pulmonary stenosis and the baby becomes cyanotic. The arterial duct closes between one day and two weeks of life.
  - b. The pulmonary blood flow for the patient in this case is diminished but not severely. In cases of severe pulmonary stenosis, ductal dependent lesions may present with cyanotic cardiovascular collapse.
3. What are the key elements of the vital signs in the evaluation of this infant?
  - a. Age: The arterial duct usually closes in the first 2 weeks of life. Typically, CHD presenting in the first 2 weeks of life is due to closure of the arterial duct in a ductal dependent lesion. The arterial duct is a vessel that connects the aorta and the pulmonary artery (PA). Its purpose in utero is to shunt blood from the PA to the aorta thus bypassing the waterlogged lungs, which have infinitely high vascular resistance.



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- b. RR: If the lungs have normal or decreased circulation the RR will be normal. If the lungs are not over-circulated the infant usually exhibits normal feeding and growth. Conversely, over-circulated lungs present with poor weight gain and increased RR.
  - c. Weight: A normal newborn will initially lose weight, but should regain birth weight by 10 days of life, followed by an increase of approximately 30g per day. This patient is on target with weight gain.
    - i. Key Concept: Under-circulation of the lungs = normal RR = normal growth.
  - d. Oxygen saturation: There is a spectrum of severity of pulmonary stenosis (PS) in TOF ranging from critical (i.e. PS so severe that the child experiences cyanotic cardiovascular collapse) to mild cyanosis as in the case presented here. Rarely, patients with TOF may even present in heart failure, suggesting that the pulmonary stenosis is negligible or the arterial duct has not closed and VSD physiology dominates the picture.
    - i. In this case, blood flow to the lungs is diminished and cyanosis has become more notable as the arterial duct has closed. Therefore, you may deduce that blood flow to the lungs is limited, but not critically. If the duct is not completely closed, cyanosis may worsen.
4. What are the salient features of the physical exam and diagnostic studies in this patient?
- a. General: TOF is not usually associated with genetic syndrome but noting dysmorphic features in other cases may help to define the type of CHD that is most likely.
  - b. HEENT: Central cyanosis may be detected by examining the mucous membranes. Acrocyanosis and perioral cyanosis are normal findings in infants.
  - c. Chest: Lungs are usually clear in cases of cyanotic and under-circulated CHD.
  - d. Click: indicates that the pulmonary valve leaflets are thickened and stenotic. The right ventricle (RV) is working overtime to pump blood out the stenotic PA, thus the RV heave or increased activity.
  - e. Murmur: In TOF the murmur is caused by turbulent flow through the stenotic pulmonary outflow tract. Stenosis may be subvalvar (also called infundibular), valvar, or supra-valvar, or stenosis may be present at all the levels. The branch pulmonary arteries may also be underdeveloped.
    - i. The spectrum of pulmonary stenosis in TOF ranges from complete pulmonary atresia to very mild stenosis.
  - f. A palpable thrill along the sternal border (Grade IV/VI murmur) or high-pitched murmur indicates severe PS.
  - g. By definition, the VSD in TOF is non-restrictive. This means the RV pressure is equal to left ventricular (LV) pressure. (i.e. the VSD is so large that the pressure in the ventricles is the same). If there is no pressure gradient, then there is no murmur related to the VSD.
  - h. Abdominal: Liver is not enlarged (i.e. no evidence of over-circulation, or what is loosely called heart failure).
  - i. Extremities: Check the femoral pulses vs. brachial pulses. Normal pulses rule out coarctation of the aorta (although coarctation of the aorta is not a cyanotic lesion, knowing the status of the pulses may become important when deciphering more complex cardiac lesions).



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- j. ECG: Features of right ventricular hypertrophy (RVH) are usually seen: Pure R wave in  $V_1$  or  $rsR'$  with  $R'$  greater than 15 mm, and possibly upright T wave in  $V_1$  beyond 3 days of life. At 2-weeks the QRS frontal plane axis normal value is 65-159 degrees (mean 110 degrees), therefore greater than 159 degrees is abnormal.
  - k. Chest X-ray: Under-circulated lungs are due to decreased pulmonary blood flow. The boot shaped heart is due to the small pulmonary artery segment.
5. How would you manage the care of this infant?
- a. In all CHD patients, if there is a concern for evolving ductal closure and progressive cyanosis, prostaglandin ( $PGE_1$ ) should be started at 0.1 mcg/kg/hour. In this case, the worsening cyanosis is likely due to PDA closure so  $PGE_1$  would be indicated. When  $PGE_1$  is given, watch for apnea, elevated temperature, and rash. Discuss anticipatory intubation prior to transfer with the pediatric cardiologist.
  - b. If ductal closure is complete, and with the guidance of a pediatric cardiologist, this child may be stable for transfer to a tertiary care center for evaluation without starting  $PGE_1$ . Some patients require no intervention until the time for corrective surgery (6-9 months at most centers).
  - c. The immediate disposition involves referral to a pediatric cardiologist. Most specialists will admit for monitoring, full evaluation with echo, electrocardiogram and X-ray, and education of the parents.
  - d. Considering the art of medicine, almost all parents feel guilt when CHD is diagnosed in their baby. Just stating: "This is not your fault," goes a long way to alleviating a lifetime of guilt for the parents.

**Case 2:** A 2-month-old female presenting to the emergency department with difficulty feeding. The mom states that although she had been doing well initially, the past week or so the baby gets fussy and does not finish her bottle. She sometimes sweats with feeding.

Vitals: HR 160, RR 55, BP 76/43 Right arm, 83/45 R leg, O<sub>2</sub> sat 93%, Temp 98.8

Wt. 4.1 kg (15%) (Birth weight 3.4 kg / 50%)

General: Fussy infant but consolable. No dysmorphic features.

HEENT: Mucous membranes are moist and pink.

Chest: No increased work of breathing but tachypneic. Bilateral breath sounds are slightly coarse at bases. No retractions.

CV: Active precordium. Normal  $S_1$ ,  $S_2$  No click. There is a grade II/VI, low pitched, non-radiating, holosystolic murmur best heard at the lower left sternal border (LLSB). There is an associated diastolic rumble over LLSB.

Abdomen: Liver edge palpable at 3 cm below the right costal margin.

Ext: No cyanosis. Femoral and brachial pulses are equal with no delay.

### Question Prompts:

1. What type of CHD do you expect to discover? Discuss the differential diagnosis.



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- a. This child is presenting with CHD characterized by pulmonary over-circulation. The most common defect in this category is a ventricular septal defect, but could be a persistent PDA, or more complex heart disease.
  - b. Coarctation of the aorta may present with increased RR and + HSM, and normal oxygen saturations, but typically presents at the time of ductal closure (within the first 2 weeks of life).
  - c. Complete atrioventricular (AV) septal defect (CAVSD) could present with heart failure, but typically also has cyanosis due to total mixing. Murmur might be similar to this case. CAVSD is often associated with Down's syndrome/Trisomy 21.
    - i. Pearl: CAVSD in non-Down's patients is more likely to be unbalanced and/or "single ventricle".
2. What physiologic change has occurred to cause it to present at this time?
- a. Infants with a large left to right shunt (i.e. VSD) typically present between 6-8 weeks of life. Presentation is dictated by the natural decrease in pulmonary vascular resistance (PVR) after birth. PVR is infinitely high in utero. When a newborn baby takes his first breath, inflating the alveoli, PVR falls significantly. Over the next 2 months smooth muscle around the pulmonary vasculature regresses and the PVR falls even further. It is this decrease in PVR that allows for increased pulmonary blood flow and increased left to right shunting across the ventricular septal defect.
3. What are the key elements of the vital signs in the evaluation of this infant?
- a. Age: Pulmonary vascular resistance falls significantly by about two months of life. Typically, CHD presenting around two months of life is due to resultant increased left to right shunting when the PVR falls, thus flooding the lungs.
  - b. RR: As PVR falls, the lungs have increased circulation, and the RR increases. If the lungs are over-circulated the infant will fail to feed and grow normally. They cannot continue to feed and breathe at the same time. So, typically until PVR falls, they will grow well, then feeding slows down and sweating with feeds develops.
  - c. HR: Usually a bit elevated due to sympathetic output.
  - d. Weight: A normal newborn will initially lose weight, but should regain birth weight by 10 days of life, followed by gaining approximately 30g per day. In CHD lesions with over-circulation of the lungs the baby will develop difficulty feeding as the PVR falls or around two months. Weight gain is normal until that point and then abnormally levels off.
  - e. Oxygen saturation: Since this child has left to right shunting the oxygen saturation is typically normal.
    - i. Pearl: Babies with VSDs are more prone to developing pneumonia, which may cause low oxygen saturations but is accompanied with fever etc.
4. What are the salient features of the physical exam and diagnostic studies in this patient?
- a. General: VSDs are not usually associated with genetic syndrome, but may be part of a more complex CHD, so noting dysmorphic features may be helpful.
  - b. HEENT: No specific findings, they are not cyanotic. If cyanosis is present, consider a more complex lesion.



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- c. Chest: May hear wheezes or rales, patient will have increased work of breathing. Sometimes “happy tachypnea,” when the patient has increased rate of breathing from hypoxic drive, but otherwise does not appear distressed.
  - d. Murmur: In a VSD the murmur is caused by turbulent flow from the high pressure left ventricle to the lower pressure RV. The smaller the ventricle, the louder the murmur, the less likely the patient is to present with pulmonary over-circulation symptoms. With significant over-circulation, a diastolic rumble may be noted.
    - i. Pearl: Listen intentionally for “the absence of silence in diastole”, to detect a diastolic murmur.
    - ii. These infants may not have had an audible murmur at birth due to high PVR. Murmur may not be audible if the VSD is so large that there is no pressure gradient between the left and right ventricle.
  - e. A thrill palpable along the sternal border (Grade IV/VI murmur) or higher pitched murmur might indicate a very small VSD, but if this is the case, pulmonary over circulation is not expected.
  - f. Abdominal: Liver edge is usually down.
  - g. Extremities: Check the femoral pulses vs. brachial pulses. Normal pulses rule out coarctation of the aorta, which could also cause similar symptoms, although the murmur is different.
  - h. ECG: Features of left ventricular hypertrophy (LVH) may be seen including: R wave >25-30 mm in both V<sub>5</sub> and V<sub>6</sub>.
  - i. Left atrial enlargement may be noted on ECG.
  - j. Chest X-ray: Over-circulated lungs due to increased pulmonary blood flow. The heart is generally enlarged and pulmonary edema may be seen.
5. How would you manage the care of this infant?
- a. There is no indication for PGE<sub>1</sub> since there is no cyanosis or shock. Additionally, the timing of symptoms is a little later than expected for a ductal dependent lesion.
  - b. These patients should be referred to a pediatric cardiologist for admission. Inpatient evaluation will include echocardiogram, electrocardiogram and X-ray, and education of the parents. Medication will be started, typically furosemide with either spironolactone (potassium sparing) or enalapril. Increased caloric density or even nasogastric (NG) tube feedings may be necessary to get enough calories for growth.
  - c. As with any CHD, just stating: “This is not your fault,” goes a long way to alleviating a lifetime of guilt for the parents.

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## Appendix F: Valve Disorders

### Objectives

1. Identify patient populations at risk for developing infective endocarditis (IE).
2. Describe the classic physical exam findings of IE.
3. Discuss appropriate workup and empiric antibiotics for presumptive IE.
4. Describe the classic presentation of patients with aortic stenosis (AS).
5. Understand the pathophysiology of AS.
6. Discuss the diagnosis and management of AS patients in the emergency department (ED).

### Case Studies

**Case 1:** A 28-year-old female with no previous past medical history presents with multiple chief complaints. She states that over the last 2 weeks she has had fevers, fatigue, malaise, anorexia, shortness of breath, productive cough, and pleuritic chest pain. The patient states she has been to two urgent cares since her symptoms started and treated twice with antibiotics for community acquired pneumonia but she has never achieved resolution of her symptoms. She denies any recent travel, incarcerations, or known sick contacts. She does reluctantly endorse intravenous (IV) drug use (IVDU) for the last 6 months. The patient's vitals are as follows: Temperature (Temp) 101.2°F, Heart rate (HR) 110, blood pressure (BP) 120/80, respiratory rate (RR) 20, and oxygen saturation (O<sub>2</sub> sat) 94% on room air (RA). On exam you hear coarse breath sounds throughout. You faintly appreciate a rumbling systolic murmur at the sternal boarder and note some neck pulsations with each heartbeat. You order a chest X-ray (CXR) that shows multifocal pneumonia.

### Question Prompts:

1. Does this patient have simple community acquired pneumonia? What historical and physical exam findings suggest a more complex underlying pathology?
  - a. This patient has several risk factors both historically and on physical exam that suggest infective endocarditis (IE). Patient endorses a history of IVDU which is an increasingly common risk factor for infective endocarditis. Other risk factors include recent dental procedures, indwelling intravenous or arterial catheters, recent pacemaker placement, prosthetic valves, unrepaired congenital heart defects, chronic rheumatic heart disease, age-related valvular lesions, hemodialysis therapy, and any immunocompromising diseases such as human immunodeficiency virus (HIV).
  - b. Physical exam revealed a cardiac murmur which should be treated as new. Infectious vegetations most commonly develop along the valve margins and impair proper valve closing. As such, patients with IE will often have regurgitation murmurs. The location of the



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- murmur depends on which valve is involved. This patient has had multiple presentations for similar complaints suggesting that she may be persistently bacteremic.
2. As the astute resident that you are, you suspect this patient may have infective endocarditis and return to the patient's room to perform a thorough skin examination. What type of cutaneous findings might you find in this patient?
    - a. Petechiae: These are the most common skin manifestation of IE and can occur anywhere on the body including mucous membranes. They are caused by microemboli from valvular vegetations.
    - b. Splinter hemorrhages: Thin red lines underneath the nails that run in the direction of nail growth.
    - c. Osler nodes: Small tender nodules found on the pulp surfaces of the distal phalanges, soles of feet, and thenar/hypothenar eminences caused by circulating immunocomplexes. These are most commonly seen with left sided IE.
    - d. Janeway lesions: Painless macules noted on the thenar and hypothenar eminences of the hands and feet caused by microabscesses of the dermis from septic emboli.
    - e. Note that cutaneous manifestations in IE are relatively late findings. A patient who presents early on in the disease process may lack these classic features.
  3. Describe the pathophysiology of infective endocarditis in a patient with IVDU such as this one.
    - a. The initial step in development of any kind of IE is endocardial injury. The injured endocardial/valve surface becomes coated by platelets and fibrin which then become a nidus for infection. The most common cause of injury is turbulent blood flow from a congenital heart defect. In the case of IV drug users, however, the tricuspid valve is injured by direct injection of contaminating debris into the blood stream.
    - b. Once the valve is infected it leads to persistent bacteremia and produces septic emboli. In IV drug users with tricuspid valve involvement, septic emboli manifest as multifocal pneumonia. Progressive damage of the valve can lead to fulminant heart failure. With left sided involvement (which is overall more common) septic embolic can damage any organ in the body. The mitral valve is most commonly affected, followed in decreasing frequency by the aortic, tricuspid, and pulmonic valves.
    - c. The three most common species of bacteria causing IE are *Staphylococcus aureus*, *Streptococcus viridans*, and coagulase negative streptococcus. HACEK (*Haemophilus*, *Aggregatibacter* (previously *Actinobacillus*), *Cardiobacterium*, *Eikenella*, *Kingella*), fungal species, and *Pseudomonas* are less common.
  4. What is the appropriate workup for IE in the emergency department? Is there a diagnostic decision rule that can guide you to making the diagnosis of IE?
    - a. The most commonly used criteria for diagnosing IE is the Duke criteria with a specificity of 99% and a negative predictive value of 92%. See MDCalc (<https://www.mdcalc.com/duke-criteria-infective-endocarditis>) for a full description of the Duke criteria, but for quick reference:
      - i. Pathological criteria: If either is positive, diagnosis is definite.
        1. Microorganisms in a vegetation.
        2. Pathologic lesions.



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- ii. Major clinical criteria: If both positive, diagnosis definite.
    1. Blood cultures positive for typical microorganisms consistent with infective endocarditis.
    2. Evidence of endocardial involvement on echocardiogram.
  - iii. Minor clinical criteria: If all positive, diagnosis definite.
    1. Predisposing heart condition, IV drug use.
    2. Fever.
    3. Vascular phenomena.
    4. Immunologic phenomena.
    5. Microbiological evidence.
  - b. No laboratory testing is diagnostic of IE but routine labs may have prognostic value and should be obtained. The most important tests to order are 3-5 sets of blood cultures which are positive in 95% of cases.
  - c. Transthoracic echocardiogram (TTE) is the first line image modality for diagnosis of IE. Transesophageal echocardiography (TEE) can be used when the TTE quality is poor or if the TTE study is negative but there remains a high clinical index of suspicion. While echocardiography is the gold standard, more advanced imaging studies such as electrocardiogram (ECG) gated cardiac computed tomography (CT) have shown promise but still require further research validation.
5. The admitting hospitalist asks you to start the patient on antibiotics and hangs up before you can ask what kind. What antibiotics are recommended for this patient? Would the choice be different if the patient had a prosthetic valve?
- a. In the ED, culture results are rarely available to help guide antibiotic selection. Empiric antibiotic therapy should be aimed at the most common bacteria keeping local resistance patterns in mind. *Streptococcus* and *Staphylococcus* species are the most common pathogens. Given the increasing incidence of methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin should be a staple of empiric treatment. Vancomycin alone may be sufficient in cases of IVDU-associated IE. For cases of native valve IE without identifiable risk factors, gentamycin should be added to vancomycin.
  - b. Vegetations on prosthetic valves consist of a thick biofilm that evades several common antimicrobials. Empiric treatment of prosthetic valve should include rifampicin which in theory has better biofilm penetration. Up to 6% of prosthetic valves will be complicated by endocarditis within five years.
6. The nurse calls you to bedside. The patient is now hypotensive. You note worsening jugular venous distention (JVD) and a more pronounced cardiac murmur. Is there any indication for emergent surgical consultation?
- a. Cardiogenic shock from valve failure or papillary muscle rupture are indications for emergent cardiac surgery consultations.

**Case 2:** A 78-year-old male presents with his wife with a chief complaint of lightheadedness, dyspnea, and fatigue. The patient is markedly lethargic so all history is obtained from the patient's wife. They are visiting from out of town and no medical records are available. Per the wife, three days ago he complained of



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lightheadedness, dyspnea, and chest pain with exertion that has progressed to decreased responsiveness. She is not sure of his medical history but she did note that he was admitted to a hospital for syncope 6 months ago and was told he had “some sort of problem with his heart valve.” His vitals are as follows: Temp 98°F, HR 95, RR 22, BP 85/45, O<sub>2</sub> sat 92% on RA. On exam, the patient is lying supine with his eyes closed but responds to questions appropriately. You auscultate a harsh systolic murmur at the right upper sternal border and crackles at the lung bases.

## Question Prompts:

1. Given the type of the murmur heard on exam, what valve disorder does this patient have? What is the mortality rate for someone with this disease?
  - a. This patient presents with a story and physical exam concerning for severe aortic stenosis (AS). The classic murmur of AS is systolic, crescendo-decrescendo, heard best at the right upper sternal boarder radiating to the carotids.
  - b. AS develops slowly over years to decades and patients often only develop symptoms late in the disease course. Once symptomatic, the mortality rate rises dramatically to 25% per year. Left untreated 75% of patients will die within three years of symptom onset.
2. Describe the pathophysiology of this patient’s disease.
  - a. AS progresses to a fixed outflow obstruction. As the valve opening narrows, left ventricular hypertrophy (LVH) develops as a compensatory mechanism to preserve ejection fraction. Once the valve narrows beyond a critical point, LVH becomes severe leading to a drop in left ventricular end diastolic volumes and ultimately a decline in cardiac output. When this happens, the patient will develop clinical signs and symptoms of congestive heart failure. The high resistance across the aortic valve also prevents increased cardiac output during exercise culminating in syncope. In addition, LVH itself reduces diastolic function and impedes coronary perfusion which can cause angina, a common symptom of AS.
3. What ECG and CXR findings support the diagnosis of severe aortic stenosis as the cause of the patient’s symptoms? What is the gold standard diagnostic test?
  - a. Diagnosis of new-onset aortic stenosis is difficult in the ED. However, ECG and CXR in combination with your history and physical exam can help aid in the diagnosis. In patients with severe aortic stenosis an ECG may show left ventricular hypertrophy and non-specific repolarization abnormalities. A CXR can show a boot-shaped enlarged cardiac silhouette indicating cardiomegaly. If the patient is in cardiogenic shock (such as this patient) you can also see pulmonary edema.
  - b. Echocardiography is the diagnostic test of choice but is often not readily available in the emergency department. As such, the emergency physician must rely on a thorough history, physical exam, and ancillary tests to arrive at the diagnosis. When available, a STAT echocardiogram can greatly expedite patient disposition and definitive management.
4. You suspect that this patient is in cardiogenic shock from severe aortic stenosis. How should you treat this patient?
  - a. There are two types of symptomatic AS patients that present to the emergency department: those who are hemodynamically stable and those who are not.



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- b. associated with Down's syndrome/Trisomy 21.
  - i. For patients who are hemodynamically stable (by which we mean those who are maintaining cardiac output), the role of the emergency physician is to make the correct diagnosis and arrange for appropriate consultation and disposition. These patients should all be admitted to a monitored bed for urgent echocardiogram and cardiac surgery consultation for aortic valve replacement.
  - ii. Patients who are hemodynamically unstable (in cardiogenic shock) are pre-load dependent and resuscitation should begin with IV fluids. Inotropic agents such as dobutamine should be considered as a temporizing measure. Ultimately these patients need a cardiac surgeon and aortic valve replacement. If the patient is too unstable to go to the operating room they can be temporized with percutaneous aortic balloon dilation which can act as a bridge to valve replacement. There is some evidence in the critical care literature that these patients benefit from nitroprusside infusion. This is somewhat counterintuitive in a patient who is already hypertensive. The theory is that a further reduction in afterload can improve systolic and diastolic function thereby improving flow across the fixed outflow obstruction. This medication is best administered in consultation with a cardiac intensivist.
5. The patient undergoes successful mechanical aortic valve replacement and is eventually discharged home. He returns to your ED three months later complaining of dyspnea. On exam you note a diastolic murmur at the sternal border and bounding peripheral pulses. What surgical complication is this patient likely experiencing?
  - a. This patient is likely suffering from aortic regurgitation (AR) as a consequence of valve thrombosis. Both mechanical and bioprosthetic valves are at highest risk for thrombosis within the first three months following surgery. Patient's with mechanical aortic valves should be on life-long coumadin with a goal international normalized ratio (INR) of 2.5-3.5. Thrombosis of the prosthetic valve can cause AR which presents with dyspnea, weakness, and cardiogenic shock. These patients require emergency cardiac surgery consultation and operative intervention.

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## Appendix G: Acute Coronary Syndrome

### Objectives

1. Discuss methods to stratify patients with undifferentiated chest pain for acute coronary syndrome (ACS).
2. Describe commonly used decision-making rules (HEART and TIMI score) for risk-stratification of ACS.
3. Discuss the definition of ST-segment elevation myocardial infarction (STEMI) and possible STEMI equivalents.
4. List and discuss pharmacologic management of patients with confirmed or suspected ACS.
5. Describe the appropriate use of provocative testing for patients with suspected ACS.
6. List indications for urgent or emergent cardiology consultation and reperfusion therapy.

### Case Studies

**Case 1:** A 52-year-old female with a history of end-stage renal disease on hemodialysis, hypertension, diabetes and tobacco use presents with a chief complaint of chest pain. She states she had just completed dialysis when she developed a gradual onset of dull chest pressure radiating to both arms, associated with shortness of breath. She has not exerted herself since the onset of her symptoms and denies a pleuritic component to the pain. She has never had similar symptoms and denies a known history of cardiac disease. She is hypertensive at 160/90 but the remainder of her vital signs are within normal limits. On physical examination, she is anxious but in no acute distress. The remainder of her exam is unremarkable.

### Question Prompts:

1. Based purely on the information provided in the history of present illness, is this patient sick or not sick?
  - a. The patient's classic risk factors include hypertension, diabetes and tobacco use. Although not factored into common ACS clinical risk scores such as HEART and TIMI, end-stage renal disease (ESRD) is a major risk factor for cardiovascular disease. ESRD causes progressive coronary artery calcification and inflammation which, in turn, lead to plaque formation. Additional coronary artery disease (CAD) risk factors include age, hypercholesterolemia, obesity, family history of CAD (parents or siblings with CAD diagnosed prior to age 65), and known peripheral arterial disease or cerebrovascular disease.
  - b. While historical features of a patient's chest pain cannot definitively rule in or out ACS, the description of the patient's discomfort can help risk stratify the patient. Pain that radiates to the shoulders or arms, is worse with exertion, and is associated with diaphoresis is most predictive of ACS. Conversely, pain that is described as sharp/stabbing, positional, pleuritic, or reproducible is least predictive. This patient has pain that radiates to both arms which on



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- its own is highly predictive of ACS and more specific for cardiac pain than pain radiating to the left or right arm alone.
- c. Remember that women, elderly, and diabetic patients commonly present with atypical symptoms of ACS. These include pain located outside the chest, lack of pain, shortness of breath, and nausea.
2. The patient's electrocardiogram (ECG) shows non-specific T-wave changes and evidence of left ventricular hypertrophy. How should you interpret the ECG in this clinical setting?
    - a. ECGs with obvious ischemic changes such as ST-elevations and significant ST-depressions are easy to interpret and confer high positive likelihood ratios (LR) for 30-day major adverse cardiac events (MACE) defined as all-cause mortality, myocardial infarction, or coronary revascularization.
    - b. What about non-specific ECG changes and repolarization abnormalities such as we see in this case? A recent retrospective study revealed that even common non-specific changes such as T-wave inversions in III and V1 confer increased likelihood of MACE at 30 days with a LR of 1.2.
    - c. The HEART score for major cardiac events includes non-specific repolarization abnormalities as a risk factor for MACE. As such, while this patient's ECG does not show overt signs of myocardial ischemia, it should be taken seriously and factored into her overall risk for ACS.
  3. Her troponin is elevated at 0.09 ng/mL but stable from her baseline. How should you interpret the elevated troponin in this clinical setting?
    - a. Patients with chronic kidney disease (CKD) commonly have baseline elevation of troponin. Although the exact reason is unclear, it appears to be relate to increased enzyme release from structural heart disease rather than decreased renal clearance. As these patients have chronic troponin leak, the specificity of the troponin assay is lower and poses a challenge to the diagnosis of ACS in patients with CKD. Patients with heart failure likewise can have chronic troponin elevations.
    - b. As with any diagnostic study, it must be interpreted within the clinical setting. An elevated troponin in an asymptomatic ESRD patient is much less worrisome than an elevated troponin in patient such as ours who is complaining of chest pain.
    - c. In this case, delta troponin measurements are valuable in making the diagnosis of non-ST Elevation Myocardial Infarction (NSTEMI). The National Academy of Clinical Biochemistry recommends using a change in troponin level greater than 20% from baseline to diagnose ACS.<sup>6</sup> Of note, this change can be an increase or a decrease. An increase in troponin signifies an evolving infarct while a decrease suggests a resolving infarct—both of which are worrisome and warrant inpatient admission.
    - d. Not all troponin elevations are caused by ACS. Troponins can rise in several disease processes that place stress on the heart and cause "demand" ischemia. These include sepsis, myocarditis, pulmonary embolism, and cardiac trauma.
  4. Using the HEART score for major cardiac events, what is this patient's risk of MACE in the next 6 weeks? What is the appropriate disposition?
    - a. The HEART score was developed to risk stratify patients who present with undifferentiated chest pain to the emergency department. It has been externally validated internationally



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and performs similarly to TIMI. Although the score requires only a single troponin, serial biomarkers increase sensitivity and negative predictive value (NVP) to nearly 100%.

- b. History
    - i. Highly suspicious: +2
    - ii. Moderately suspicious: +1
    - iii. Slightly suspicious: 0
  - c. EKG
    - i. Significant ST depression: +2
    - ii. Nonspecific repolarization disturbance: +1
    - iii. Normal: 0
  - d. Age
    - i.  $\geq 65$ : +2
    - ii. 45-65: +1
    - iii.  $\leq 45$ : 0
  - e. Risk Factors
    - i. *Risk factors include:*
      - a. Hypercholesterolemia
      - b. Hypertension
      - c. Diabetes Mellitus
      - d. Cigarette smoking
      - e. Positive family history
      - f. Obesity
    - ii.  $\geq 3$  risk factors or history of atherosclerotic disease: +2
    - iii. 1-2 risk factors: +1
    - iv. No risk factors known: 0
  - f. Troponin
    - i.  $\geq 3\times$  normal limit: +2
    - ii. 1-3 $\times$  normal limit: +1
    - iii.  $\leq$  normal limit: 0
  - g. This patient's HEART score is 7 (History: 2, ECG: 1, Age: 1, Risk factors: 2, and Troponin 1). According to this scoring system the patient is high risk for MACE at 6 weeks at 50-65%. This patient should be given 4 baby aspirin to chew and swallow. These patients are often candidates for early invasive measures and should be admitted to the hospital.
5. While this patient is boarding in the ED, what treatments should be initiated?
- a. The following are recommended treatment for Non-ST elevation ACS and unstable angina (NSTEMI/Ua) from the 2014 American Heart Association (AHA) guidelines.
    - i. Bed rest: Theoretically reduces myocardial oxygen demand.
    - ii. Oxygen: Poor overall evidence. Should not be given liberally as hyperoxia can be toxic due to free radical production. Oxygen should be delivered to maintain O<sub>2</sub> saturation  $>90\%$ . (AHA Class IC recommendation).
    - iii. Nitrates: Increases myocardial oxygen delivery through coronary artery dilation and decreased myocardial oxygen demand via preload and afterload reduction.



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Should be given sublingual and then, if pain not improved, transitioned to continuous infusion titrated to resolution of pain. (AHA Class IC recommendation). Should be avoided in hypotension, suspected right ventricular infarct, aortic stenosis, pulmonary hypertension or if patient has recently used phosphodiesterase inhibitors.

- iv. Morphine: Can be given to reduce chest pain in patients who have not responded to nitrates. Controversial because it is thought to decrease gut absorption of aspirin and other oral medications. (AHA Class IIB recommendation).
- v. Aspirin: 325mg non-enteric coated chewable aspirin should be given to all patients without contraindications (AHA Class IA recommendation). This intervention shows high efficacy with a number needed to treat (NNT) of 40.
- vi. Anticoagulation: Enoxaparin (AHA Class 1A recommendation) with adequate creatinine clearance or heparin infusion (AHA Class 1B recommendation). There is conflicting data regarding morbidity and mortality benefit of anticoagulation. However, there appears to be an overall trend towards benefit, albeit not statistically significant.

**Case 2:** You've just begun your first moonlighting shift at Our Lady of Faint Hope community hospital, minding your own business, when a nurse wheels back a patient from triage and asks for your help immediately. You walk in the room to see a male in his 60s clutching his chest complaining of chest pain. He is diaphoretic, anxious, and dyspneic. He is in obvious distress and unable to provide a history. He is hypertensive and oxygen saturations are 90% on room air (RA). His respiratory rate is 18. The remainder of his exam is unremarkable. Just as you turn to the computer to place orders he looks up to you and says "Doctor, don't let me die."

### Question Prompts:

1. What actions should be performed immediately?
  - a. Intravenous line (IV) placement and monitor. Oxygen should only be administered to maintain oxygen saturations greater than 90%. ECG should be performed within 10 minutes of patient arrival in the emergency department (ED).
2. Your ECG shows 1.5mm ST-segment elevations in leads II, III, and aVF. You are concerned for ST-segment elevation myocardial infarction (STEMI), but as a good emergency medicine physician you develop a differential for ST segment elevations. What else could be causing this ECG abnormality?
  - a. STEMI is defined as ST segment elevation greater than or equal to 1mm in 2 anatomically contiguous ECG leads. In V1-V3, ST elevations greater 1.5mm in women and 2mm in men increase diagnostic specificity. The presence of reciprocal changes (ST depressions in anatomically opposing leads) also increases diagnostic specificity. The differential for ST segment elevations include:
    - i. Aortic dissection involving the origin of the coronary artery: Looks identical to a true STEMI. Must be differentiated from STEMI clinically.
    - ii. Benign early repolarization: No reciprocal ST-depressions, no Q waves.



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- iii. Left ventricular aneurysm: No reciprocal ST-depressions, large Q waves.
    - iv. Pericarditis: No reciprocal ST-depressions, no Q waves, + PR depressions.
  - b. ST segment depressions in V1-V3 can be seen with posterior MIs. Turning the ECG upside down or obtaining an ECG with posterior leads (V8-V9) will show your classic ST elevations.
  - c. ST elevations in aVR with diffuse ST depressions can be seen with left main coronary artery occlusions and should be considered a STEMI equivalent.
  - d. DeWinter ST/T wave complex in V1-V6, with ST depression and peaked T-waves; caused by proximal left anterior descending (LAD) occlusion.
  - e. A note on STEMI equivalents: The presence of a new left bundle branch block (LBBB) is classically considered a STEMI equivalent. However, recent studies have shown that most patients with chest pain and new LBBB do not have a STEMI. It is reasonable to evaluate these patients in the context of their clinical history, exam and/or cardiac biomarkers and discuss concerning cases with cardiology for possible emergent catheterization. Look for LBBB meeting Sgarbossa criteria.
    - i. Modified Sgarbossa criteria:
      1.  $\geq 1$  lead with  $\geq 1$  mm of concordant ST elevation
      2.  $\geq 1$  lead of V1-V3 with  $\geq 1$  mm of concordant ST depression
      3.  $\geq 1$  lead anywhere with  $\geq 1$  mm STE and proportionally excessive discordant STE, as defined by  $\geq 25\%$  of the depth of the preceding S-wave.
3. You are confident that the patient is having a STEMI. What medications should you order?
  - a. Aspirin 325mg
  - b. Second antiplatelet drug: This is institution dependent, usually clopidogrel 600mg, prasugrel 60mg, or ticagrelor 180mg.
  - c. Heparin 60 units/kg (maximum: 4,000 units).
  - d. Nitroglycerin 0.3 to 0.6 mg every 5 minutes, maximum of 3 tablets in 15 minutes.
4. Given the location of his MI, what interventions might be contraindicated?
  - a. Patients with inferior STEMI may involve the right ventricle (RV). ST elevations in III>II and also in V1 are indicative of RV involvement. You can also obtain a right-sided ECG. These patients are pre-load dependent. Nitroglycerin will decrease pre-load and can lead to hypotension.
  - b. In other patients (not in those with inferior STEMI), is nitroglycerin paste preferable?
    - i. A nitroglycerin drip is preferred due to erratic absorption of paste and unpredictable absorption. It is difficult to know how much nitroglycerine your patient is receiving.
5. You are two hours from the nearest catheterization lab. You're worried you may not meet the mandated 90-minutes door-to-balloon time. You've arranged for transport but what else can you do while you're waiting?
  - a. Fibrinolysis. If percutaneous coronary intervention (PCI) cannot be achieved within 90 minutes of patient arrival, fibrinolytics should be administered provided there are no contraindications. The recommended door to needle time for STEMI is 30 minutes. Fibrinolysis does not preclude PCI. Administer the fibrinolytic while arranging transport to the nearest PCI-capable facility.



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**Case 3:** A 62-year-old male with a history of hypertension and diabetes presents with a chief complaint of chest pain. He states that over the last few weeks he has experienced episodes of chest pressure that radiate to his left jaw and shoulder while on his morning walk. He denies any associated symptoms. Prior to these past few weeks he has never had similar symptoms. His father had a heart attack at age 60. He does not use tobacco products. He is currently chest pain free and vital signs are all within normal limits. His ECG, chest X-ray, and troponin are all normal. The patient asks if he can be discharged home to follow up with his primary care physician.

## Question Prompts:

1. What is the patient's risk for MACE?
  - a. The patient's HEART score is 5 placing him at 12-16.6% chance of MACE at 6 weeks. This is considered moderate risk.
2. What is the most appropriate disposition for this patient?
  - a. Given the patient's moderate risk for MACE, the percentages listed above should be shared with the patient so that he can make an informed decision on his disposition.
3. What options are there for provocative testing? What is the sensitivity of a stress test?
  - a. Stress tests are not diagnostic of coronary artery disease. Rather, they are a tool that helps us further risk stratify those patients who have presented with signs and symptoms suggestive of unstable angina.
  - b. Current AHA guidelines recommend that these patients undergo provocative testing within 72 hours of their ED visit.
  - c. Stress tests are far from perfect: at best, the sensitivity is 90%, and they have poor specificity.
  - d. False positives are common. Roughly one-third of patients with an abnormal stress test will go on to have coronary angiography that shows no significant obstructive coronary artery disease.
  - e. Stress testing will miss approximately 10% of patients with significant CAD and ACS. Do not be lulled into a false sense of security if a patient with a concerning story has had a recent negative stress test. Have a low threshold to consult cardiology and admit them to the hospital.

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## Appendix H: Brief Resolved Unexplained Event (BRUE)

### Objectives

1. Define brief resolved unexplained event (BRUE) and the risk factors associated with it.
2. Differentiate possible mimics of BRUE in the young infant.
3. Discuss initial evaluation of BRUE based on history and physical examination and differentiate which patients require a complete evaluation and which can be observed.
4. Discuss historical and physical exam findings consistent with non-accidental trauma (NAT) which may suggest physical abuse as a potential cause of BRUE.

### Case Studies

**Case 1:** A 10-week-old formerly full-term male is brought to the emergency department (ED) by emergency medical services (EMS). Mom states that the child appeared to stop breathing for about 20 seconds during which time his lips looked blue. She did not provide any stimulation, rescue breaths or chest compressions. She informs you that she was present for the entire event but that it was so quick, that by the time she stood to pick him up it was over. EMS reports no issues while en route to the hospital. Your exam of the patient is reassuring and entirely normal.

### Question Prompts:

1. What is the definition of BRUE?
  - a. Brief resolved unexplained event (BRUE) is defined as an event occurring in a patient younger than 1 year of age in which an observer sees at least one of the following:
    - i. Cyanosis or pallor.
    - ii. Absent, decreased or irregular respirations.
    - iii. Significant change in tone (can be hypotonia or hypertonia).
    - iv. Altered level of responsiveness.
  - b. The event must be brief (lasting < 1minute) and spontaneously resolve.
  - c. A BRUE is a diagnosis of exclusion reached only after history and physical exam rule out other etiologies for the event.
  - d. The definition of BRUE is not met:
    - i. If additional symptoms or vital sign anomalies are presents during ED evaluation.
    - ii. If event defining criteria outlines above are absent.
    - iii. If another explanation for the spell or event is identified on history and physical.In such cases, the BRUE guidelines should not be applied to these patients.
2. What criteria define a low risk BRUE?
  - a. Age  $\geq$  60 days.



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- b. Born at  $\geq 32$  weeks with a corrected gestation age  $\geq 45$  weeks (in other words be careful adjusting for prematurity).
  - c. No cardiopulmonary resuscitation (CPR) by a trained medical provider.
  - d. Event lasted  $< 1$  minute.
  - e. First BRUE (overall lifetime, not just on this particular physician visit).
3. Given that this patient is low risk, what management considerations would be, may be, and would not be appropriate?
  - a. Education about BRUE and outpatient resources for CPR training for caregivers are appropriate for low risk BRUE.
  - b. Screening electrocardiogram (ECG) and surveillance pertussis swabs may be obtained. Additionally a brief period of ED observation with continuous pulse oximetry and several physical assessments may be helpful. This can provide additional reassurance to the family and prevent errors of premature closure by the ED team.
  - c. There is no indication for blood work including chemistries, ammonia, cultures, complete blood counts or metabolic workups. There is no indication for cerebral spinal fluid (CSF) studies of any kind. Echocardiogram, electroencephalogram and radiographic imaging are also not indicated in these patients. Home cardio-respiratory monitoring is not recommended. Initiation of medications for seizure or reflux fall outside of the scope of BRUE as these indicate the physician is concerned for a more likely alternate diagnosis.
  - d. The patient does not require admission purely for the purpose of cardiopulmonary monitoring.

**Case 2:** A 5-month-old male is brought to the ED by EMS. Parents report that they are visiting family in your area. Parents report that the patient was in his usual state of health and began to cry. Parents noticed that as he was crying he looked blue. EMS was called to the home. Family reports they have been following with cardiology at their local hospital and were told this kind of spell could happen. They report that they could not remember what to do so they called 911. By the time EMS arrived the child was calm and his appearance had normalized. On your exam, vital signs are normal but physical exam is remarkable for a harsh systolic ejection murmur at the left sternal border.

### Question Prompts:

1. What is the most appropriate initial diagnostic evaluation for this patient?
  - a. This history is concerning for a hypercyanotic episode associated with Tetralogy of Fallot, often termed as “tet spell”. Patient should be placed on a cardiac monitor. ECG should be obtained. Intravenous (IV) access should be obtained as a precaution. While the patient is back to baseline, he should be closely monitored for recurrent spells. Patient should be discussed with cardiology either at your local institution or where the patient is currently followed.
2. What are the historical clues which suggest the need for further evaluation and hospitalization?
  - a. Brief: The episode was of unclear duration but if calmed quickly could last less than 1 minute.



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- b. Resolved: The child does meet this criteria with a return to baseline.
- c. Unexplained: This episode is concerning for tet spell which would not be unexplained but rather signal a high risk non-BRUE diagnosis.
- d. Event: This child had an event which included cyanosis.

**Case 3:** A 2-month-old female is brought to the ED by mother after an event. Mom describes that the patient was sleeping, noted to stop breathing with no respiratory effort and blueness to the face. Mom picked her up, noted her to be limp and gave several rescue breaths. The patient is currently at baseline with normal vital signs, normal physical exam. Child has no prior medical history. She has one prior visit to the ED for a bruise to the leg. Evaluation in the ED includes a chest X-ray, which is concerning for posterior rib fractures.

### Question Prompts:

1. What further evaluation and consultation is indicated for this patient?
  - a. The posterior rib fractures are extremely specific for non-accidental trauma (NAT). Patient should undergo CT head and full skeletal survey to evaluate for NAT. Additionally, the patient should have screening labs including complete blood count (CBC), coagulation panel and liver/pancreas enzymes to evaluate for occult intra-abdominal injury. A report should be made to children's services.
  - b. Transaminase levels meet the threshold for imaging for abdominal injury in NAT at much lower levels than for accidental trauma. AST or ALT >80 should prompt CT abdomen and pelvis with intravenous (IV) contrast in patients undergoing NAT evaluation. This lower threshold exists because the goal is to identify any trauma, as this could be important evidence in protecting the child. This is different than the higher threshold for accidental trauma where the goal is to find *clinically significant* trauma.
2. What are the historical and physical exam clues that suggest non-accidental trauma rather than BRUE?
  - a. Are BRUE criteria met?
    - i. Brief: The episode should be less than 1 minute. This episode may meet that criterion.
    - ii. Resolved: The child does meet this criteria with a return to baseline.
    - iii. Unexplained: There is bruising noted on this non-mobile patient which is always suspicious for non-accidental trauma. This would provide an alternate explanation and therefore utilization of BRUE guidelines would be inappropriate in this case.
    - iv. Event: This child had an event which included cyanosis, absent breathing, hypotonia and altered consciousness.
  - b. The findings of any fracture in a non-mobile child should increase suspicion for NAT. Rib fractures are one of a number of high specificity injuries along with metaphyseal corner fractures. Prior visit for a bruise is also concerning in a child this age.
3. Identify sentinel injuries as indication for non-accidental trauma.
  - a. Sentinel injuries are those seemingly minor injuries which in retrospect have a correlation to NAT or abusive head trauma. Bruising and abrasions in non-mobile infants should raise



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suspicion for abuse. Additionally, sub-conjunctival hemorrhage and intraoral injuries such as torn frenula are concerning.

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## Appendix I: Left Ventricular Assist Devices

### Objectives

1. Identify the indications for left ventricular assist device (LVAD) placement.
2. Discuss the physiology behind the function of an LVAD.
3. Understand the first steps in evaluation of a patient with an LVAD.
4. List the possible complications of a patient presenting with an LVAD.
5. Understand steps in treating a patient with an LVAD who is unresponsive or hemodynamically unstable.

### Case Studies

**Case 1:** A 57-year-old male with ischemic cardiomyopathy status post LVAD presents with shortness of breath. He has been having increased swelling in his lower extremities, increased orthopnea, and now has shortness of breath at rest. These symptoms have been progressive over the last 2-3 days. The nurse calls you because the patient does not have a blood pressure, she cannot get a pulse oximetry reading. On your evaluation the patient is awake, alert and pleasant but somewhat dyspneic. He also appears pale.

### Question Prompts:

1. What are the indications for LVAD placement?
  - a. Left ventricular assist devices (LVAD) augment left ventricular (LV) output in patients with severe cardiomyopathy. The implanted pump transfers blood from the apex of the LV to the proximal aorta. The pump is powered by an external power source, regulated by a controller. Both the battery and controller reside outside of the body. The controller drives the pump through a driveline, connected through a surgical incision in the abdominal wall.
2. What are the types of LVADs available?
  - a. 2 types of LVADs—Continuous flow VADs and Pulsatile flow VADS.
    - i. Pulsatile flow LVADs: These are rarely used now as they were a first-generation device.
    - ii. Continuous flow (2<sup>nd</sup> generation) LVADs: These are the most common to encounter as of 2017. These use a corkscrew mechanism to continuously move blood forward. These patients will typically have a very weak or absent pulse. However, many patients retain some cardiac contractility; in these individuals, a pulse may be present. LVAD patients still rely upon reasonable right ventricular (RV) function to pump blood to the lungs.
    - iii. Continuous flow (3<sup>rd</sup> Generation) LVAD: These devices use electromagnets and centrifugal force to move blood forward. The spinning impeller device is completely suspended by magnetic force (MagLev) and does not make contact with other parts



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of the device. This decreases hemolysis and improves flow. Some are currently in use in the United States (US) in the “bridge to transplant setting.” Approval was just granted for destination therapy (long-term support in patients who are not candidates for heart transplant).

- b. Indications for LVAD placement:
  - i. Food and Drug Administration (FDA) approved as a bridge to cardiac transplant in those with clinical evidence of cardiogenic shock despite revascularization. Some case studies have described using LVADs to wean patients suffering from cardiogenic shock after acute infarction.
3. How do you obtain vital signs in a patient with an LVAD?
  - a. A patient with an LVAD may not have a pulse because LVAD is continuous flow. This makes automatic blood pressure cuffs unreliable and will make it hard to get a pulse oximetry. Instead, physicians should rely on clinical indicators of perfusion, such as mental status, color, and capillary refill.
  - b. A manual blood pressure (BP) with a Doppler or an arterial line can provide a mean arterial pressure. Most patient should have a MAP of about 65.
  - c. Arterial blood gas (ABG) can be used for venous pulse oxygen level (sPO<sub>2</sub>).
4. How do you evaluate the LVAD?
  - a. Assess machine function – listen to whir, see if control box is hot, check the battery.
  - b. Call the LVAD coordinator. (ABC – Airway, Breathing, Call LVAD coordinator!)
5. List the possible complications of a patient presenting with an LVAD.
  - a. Bleeding: The most common reasons for an emergency department (ED) visit in an LVAD patient is bleeding. It can occur because of a leaky pump connection or from leaks in the polyester grafts owing to problems with the process of graft healing. Bleeding also occurs from mucosal surfaces often from gastrointestinal tract arteriovenous malformations (AVMs) which develop from lack of pulsatile flow. Similar problems can occur in intracranial vessels, and intracranial hemorrhage (ICH) is the leading cause of death in patients with an LVAD. Patients are also at higher risk for bleeding due to anticoagulation. Acquired von Willebrand’s disease can occur by what is believed to be impeller-induced shearing forces breaking down von Willebrand multimers. Overall, bleeding occurs at a rate of around 1.5 events per patient-year.
    - i. In cases of severe bleeding, consider reversing anticoagulation with tranexemic acid (TXA), prothrombin complex concentrate (PCC), desmopressin or fresh frozen plasma (FFP). The risk of thrombosing an LVAD by reversing anticoagulation is relatively low. Risk of thromboembolic events is typically discussed in terms of over a year. Stopping the anticoagulation due to a bleed is unlikely to result in a thromboembolic event in the next few days. You should weigh the risks and benefits and consult the cardiothoracic surgery service that placed the device.
  - b. Infection: Infection is the second most common complication and the driveline and pocket are most common sites of infection. Infections occur in up to 50% of LVAD patients. Continuous flow pumps have a lower risk of infection than pulsatile pumps (due to smaller size and surface area).



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- c. Pump thrombosis with low output state: Pump thrombosis has an 8% incidence in LVAD patients. On exam there should be a whirring noise or “hum” over the heart in a functioning LVAD. The “hum” may not heard in patients with pump thrombosis. Hemolysis and changes in pump performance also suggest pump thrombosis.
  - i. Hemolysis can be identified by elevated lactate dehydrogenase (LDH), decreased haptoglobin, reddish-brown urine, and increases in serum free hemoglobin (Hgb).
  - ii. Transient increases in power consumption manifesting as power spikes or gradual increase in power requirement for the pump to operate are suggesting of poor pump performance.
  - iii. Thrombosis causes a decrease in pump output and the need for increased pump speed. These patients may require supplemental anticoagulation.
- d. Right heart failure: Patients often have biventricular dysfunction and while the LVAD supports normal cardiac output (CO) from the left side, the right ventricle may not be able to keep up leading to progressive failure. Bowing of the septum can cause obstruction to right ventricular (RV) outflow. Milrinone and dobutamine intravenously can help augment RV function. Inhaled nitric oxide and epoprostenol may also be used in the acute setting. Oral medications such as the PDE5 inhibitors (sildenafil and tadalafil) can be useful by decreasing pulmonary vascular resistance. Target mean arterial pressure (MAP) is 70 to 90. If blood pressure is low or there is evidence of hypoperfusion, fluids should be administered.
- e. Aortic Regurgitation: Aortic insufficiency is a common complication after continuous-flow LVAD placement, with approximately 25% developing at least mild regurgitation. This may be secondary to the continuous flow causing aortic valve commissural fusion and leaflet deterioration and increased shear stress. Mild–moderate aortic regurgitation can be helped by adjusting the pump speed of the continuous-flow device. Severe cases require aortic valve replacement (AVR), patch closure of the aortic root, or aortic valve closure.

**Case 2:** A 45-year-old male with non-ischemic cardiomyopathy status post LVAD presents via emergency medical services (EMS) with chest pain and shortness of breath. The patient is diaphoretic, unable to speak in full sentences, and confused. While you are checking for pulses, the patient stops breathing and turns blue.

### Question Prompts:

1. How do you manage the hemodynamically unstable LVAD patient?
  - a. Assess for pump failure:
    - i. Auscultate the chest. This is the fastest way to assess whether the pump is working. If the “hum” of the motor is heard, then you know electricity is getting to the pump. If the pump is not working, then the priority of the resuscitation will be to troubleshoot the LVAD.
    - ii. Are the batteries working? Are there any error messages on the controller? If the battery is malfunctioning or low, have the patient use the backup battery or replace with a fully charged battery. It is important that the device is always attached to at



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least one battery at a time otherwise the LVAD will stop. Some models may have the opportunity to plug directly into an outlet. Take note of any error messages on the controller and discuss with the LVAD coordinator.

- iii. Most patients have a tag located around the waist controller that will describe the type of device. EMS providers may have access to tip sheets to provide basic LVAD troubleshooting. Check the patients LVAD bag for information about troubleshooting. One example troubleshooting resource can be found here (<https://www.mylvad.com/sites/mylvadrp/files/Field%20Guides%20Master%20Document.pdf>)
  - b. Treat the patient: Airway, breathing, circulation (ABCs), pressors if hypotensive, assess for bleeding, electrocardiogram (EKG), labs, chest X-ray, echocardiogram.
  - c. If the patient is in ventricular fibrillation, defibrillation should be done. Restoring their sinus rhythm can significantly improve their cardiac output.
2. How do you manage the coding LVAD patient?
- a. It is generally recommended to *never* perform chest compressions on an LVAD patient. Chest compressions can dislodge the LVAD from the heart and aorta, causing left ventricular (LV) rupture and intractable hemorrhage.
  - b. However, providers can consider chest compressions to maintain circulation while attempting to address other reversible causes, because the patient will die anyway. However, it would be important to differentiate the patient that is truly in arrest from the unconscious, yet perfusing, patient without a pulse. Remember that many properly functioning LVADs will not have a measurable pulse. Consider doppler or placement of a femoral arterial line to verify absence of a pulse.

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## Appendix J: Pericardial Disorders

### Objectives

1. Identify the most common etiologies of acute pericarditis.
2. Describe the clinical features associated with acute pericarditis and pericardial tamponade.
3. Describe the diagnostic findings associated with acute pericarditis and pericardial tamponade.
4. Discuss the treatment and management for acute pericarditis and pericardial tamponade.
5. Explain the pathophysiology associated with cardiac tamponade.

### Case Studies

**Case 1:** A 45-year-old male with no past medical history presents to the emergency department (ED) with chest pain. He states the pain started four hours ago and has progressively worsened. The pain is worse with laying down and improves with sitting up. He endorses mild shortness of breath and reports a recent upper respiratory infection.

### Question Prompts:

1. What are key clinical factors when evaluating a patient with suspected pericarditis?
  - a. One should assess for other possible causes for chest pain including acute coronary syndrome (ACS), myocarditis, pulmonary embolism (PE), and other high-risk diagnosis. Some key history and physical exam findings include assessing for jugular venous distention (JVD), pulmonary edema, general appearance, S3 or S4 heart sounds, murmurs, rubs.
  - b. When completing the evaluation with labs and imaging one should evaluate for pleural effusions, pneumothoraces, and pulmonary edema with ultrasonography and chest X-rays. Electrocardiogram (ECG), troponin, creatine kinase-MB (CKMB), brain natriuretic peptide (BNP) and D-dimer may be necessary to assess for other pathologies.
2. What are the most common etiologies of pericarditis?
  - a. Infection, uremia, systemic diseases (systemic lupus erythematosus, scleroderma, amyloidosis, etc.), radiation, myocardial infarction, surgery, tumors, aortic dissection.
3. What are the diagnostic criteria for acute pericarditis?
  - a. At least 2 out of the 4 criteria: Classic positional chest pain with radiation to the trapezius area, pericardial friction rub, pericardial effusion, characteristic ECG findings.
4. What ECG findings are classically associated with acute pericarditis?
  - a. Diffuse ST segment elevation and PR depression. EKG changes may normalize over time and there may be a transition to T-wave inversion which can persist.
5. What is the management for acute pericarditis?



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- a. Non-steroidal anti-inflammatory drugs (NSAIDs) and aspirin should be given as a tapered regimen over 3-4 weeks. Steroids should be avoided in suspected cases of viral myocarditis as they increase the recurrence. However, steroids may be indicated in cases of autoimmune pericarditis such as systemic lupus erythematosus.
- b. Disposition should include outpatient management and cardiology referral in uncomplicated cases. Patients with elevated troponin and pericardial effusion should be admitted for further evaluation.

**Case 2:** A 62-year-old dialysis dependent female presents to the ED with altered mental status. Over the past few days the patient had worsening shortness of breath. Family notes her shortness of breath and generalized ill appearance had been worsening over the past week. They also state that she refused to go to her dialysis appointments the past two weeks. The physical exam is concerning for an ill-appearing patient with mottled skin and shortness of breath. Breath sounds are normal without rales or crackles, and heart sounds are barely audible.

### Question Prompts:

1. What are some clinically important findings in cardiac tamponade?
  - a. The typical history and physical exam findings of cardiac tamponade include hypotension, jugular venous distention, pulsus paradoxus and muffled heart sounds. Additionally, you may find peripheral edema and hepatic congestion in cases of chronic effusion.
  - b. Chest X-rays may show an enlarged cardiac silhouette, although it is neither sensitive nor specific. Ultrasonography will show pericardial effusion and decreased right atrial filling during systole. Other findings may include a plethoric inferior vena cava (IVC) indicating an obstructive process. ECG findings may show low voltage and electrical alternans.
2. How should pericardial tamponade be managed?
  - a. The management of the cardiac tamponade depends on the etiology and the patient's clinical status. The ultimate goal is to increase preload and contractility of the heart. Patients who do not present in extremis likely do not need immediate drainage in the ED, especially given the complications associated with pericardiocentesis.
    - i. Patients who are stable with uremia, infection or a systemic illness as the underlying cause of the pericardial effusion likely need admission for management of their underlying disease process.
    - ii. All unstable patients need immediate pericardiocentesis EXCEPT for those with aortic dissection as the underlying cause.
3. Should a patient be intubated if they have a cardiac tamponade?
  - a. If the patient is suffering from imminent respiratory failure, then the patient absolutely should be intubated. However, it is important to understand the physiology when intubating these patients. The typical respiratory cycle involves negative pressure gradients. When the intrathoracic volume increases, it creates a negative pressure



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gradient which allows air to flow into our lungs. It also allows blood to return to the heart. This is compromised in tamponade situations when the pressure generated by the fluid in the pericardial space decreases the blood flow back to the right atrium. When a patient is intubated the patient is ventilated using positive pressure which could further decrease blood flow back to the right atrium and may cause hemodynamic collapse.

4. Why should a cardiac tamponade secondary to an aortic dissection not be drained in the emergency department?
  - a. In contrast to aortic dissection, the hemodynamic goals for a cardiac tamponade is to increase preload and increase inotropy and chronotropy. Pericardiocentesis will increase inotropy and chronotropy immediately after releasing the tamponade and may acutely worsen the aortic dissection, as such, it should be performed with extreme caution and likely in the operative setting. A few studies have shown worsening outcomes for patients shortly after successful pericardiocentesis. One study has shown controlled preoperative pericardiocentesis to be lifesaving in critical cardiac tamponade in the setting of an aortic dissection.

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## Appendix K: Aortic Dissection

### Objectives

1. Review pathophysiology, diagnosis and treatment of aortic dissection.
2. Discuss the classification systems for aortic dissection and the different associated management strategies – surgical v conservative medical management.
3. List the various imaging modalities available to diagnosis aortic dissection and discuss in what settings they may be appropriate.
4. Discuss medications used for treatment of aortic dissection and goals for heart rate and blood pressure management.

### Case Studies

**Case 1:** A 40-year-old male with no past medical history presents to the emergency department (ED) via emergency medical services (EMS) for evaluation of severe, tearing low back pain associated with left leg pain, numbness and weakness for the past two hours. Pain began suddenly while lifting a ladder in his garage and has gotten progressively worse. The patient appears anxious, diaphoretic, and uncomfortable. He is unable to move his left leg and his sensation is severely diminished objectively. His left leg is also pale and cool to touch. Initial blood pressure is 235/130, heart rate is 99, and the remainder of his vital signs are unremarkable.



Image Courtesy of: Gaillard, F. Aortic dissection and aberrant right subclavian. rID 8935. In: Radiopaedia. <https://radiopaedia.org/cases/aortic-dissection-and-aberrant-right-subclavian>. Published March 10, 2010. Updated November 24, 2015. Accessed June 27, 2018. CC BY-NC-SA 3.0.

Barrie M, et al. Novel Emergency Medicine Curriculum Utilizing Self-Directed Learning and the Flipped Classroom Method: Cardiovascular Emergencies Small Group Module. JETem 2018. 3(3):C1-96. <https://doi.org/10.21980/J8X334>



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## Question Prompts:

1. What is the most appropriate diagnostic evaluation for this patient?
  - a. Computer tomography angiography (CTA) is imaging modality of choice, but in patients with renal failure consider emergent magnetic resonance imaging (MRI) and transesophageal echocardiogram (TEE).
2. Describe the management algorithm for this patient. What medication(s) would you administer? What are your goal heart rate (HR) and blood pressure (BP)?
  - a. Contact vascular surgery! Medications to be administered include nitrate or nicardipine titrated to a goal BP of 100-120, esmolol to a goal HR less than 60 and aggressive opiate pain control.
3. The patient's dissection only involves the descending aorta. What type of aortic dissection is this? Can this patient be managed conservatively, or does he require operative intervention?
  - a. Stanford B (DeBakey III) aortic dissections only involve the descending aorta. Many Type B dissections can be managed medically as described above. With vascular compromise of the lower extremity, this patient will need to be evaluated by vascular surgery for intervention which may require open operative repair of the dissection or placement of a stent to open flow to the lower extremity.

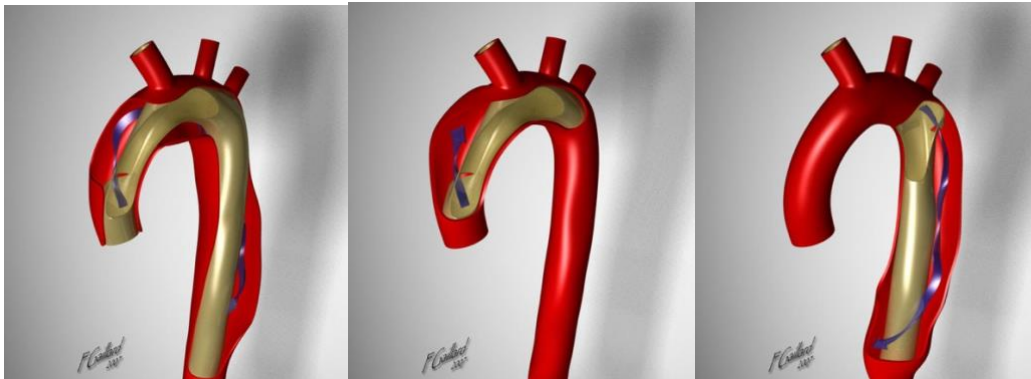


Image Courtesy of: Gaillard, F. Aortic dissection DeBakey classification (illustration). rID 7690. In: Radiopaedia. <https://radiopaedia.org/cases/aortic-dissection-debakey-classification-illustration>. Published November 16, 2009. Updated April 16, 2018. Accessed June 27, 2018. CC-NC-BY-SA.

### b. DeBakey/Sanford classification

- i. **Type I:** Both the ascending and descending aorta (Stanford A).
- ii. **Type II:** Only the ascending aorta (Stanford A).
- iii. **Type III:** Involves descending aorta only, commencing after the origin of the left subclavian artery (Stanford B).

**Case 2:** A 72-year-old male presents to the ED via EMS for severe chest pain. He has a history of coronary artery disease (CAD) and states that this pain is far different from past cardiac pain. The pain began suddenly and has gotten worse; it is currently radiating to his back. He took a nitroglycerin tablet without relief. The patient is diaphoretic, anxious, distressed, and appears critically ill. On review of his chart, you also find that

Barrie M, et al. Novel Emergency Medicine Curriculum Utilizing Self-Directed Learning and the Flipped Classroom Method: Cardiovascular Emergencies Small Group Module. JETem 2018. 3(3):C1-96. <https://doi.org/10.21980/J8X334>



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he has a history of diabetes for which he takes metformin. He also has chronic renal insufficiency (stage III). He has a blood pressure of 180/110 and a heart rate of 76. Remainder of his vital signs and physical examination are unremarkable.

## Question Prompts:

1. What imaging study or studies must be considered in this patient with chronic renal insufficiency?
  - a. While in patients with renal failure one should strongly consider emergent MRI and TEE, they may not be readily available in all hospitals. It may also take significant time to get these tests done. The high pre-test probability for dissection in this patient may warrant the need for emergent CTA despite the renal insufficiency, especially given that recent studies have called into question the injury purported to come from intravenous (IV) contrast use.
2. What is the utility of D-dimer in screening for aortic dissection?
  - a. At this time D-dimer is not ready for use in patients with suspected aortic dissection. A meta-analysis showed that when applied to a low-risk patient population, that the incidence of aortic dissection was very low in those with a normal D-dimer (0.3% rate of dissection) with 98% sensitivity. However, this low-risk patient population already had less than 10% likelihood of aortic dissection. Thus, these results do not support using D-dimer in those with moderate risk of aortic dissection. Also, like other applications of D-dimer, the specificity was low.
3. What is the IRAD study and what does it tell us about aortic dissection?
  - a. The IRAD study is the International Registry of Acute Aortic Dissection and provides a large database of data regarding aortic dissections.
  - b. The study shows that dissection does not always present like you were classically taught.
  - c. Classic presentation is tearing chest and/or back pain; however, IRAD study shows that this presentation is not universal. Only 72% present with chest pain and 53% with back pain.
  - d. Documentation of blood pressures in all four extremities is important, yet insensitive for dissection. Only 15% had a documented pulse deficit.

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## Appendix L: Acute Coronary Syndrome

### Objectives

1. Describe common causes of syncope.
2. Discuss which historical and physical examination data can help risk-stratify patients with syncope.
3. Discuss which diagnostic testing help risk-stratify patients with syncope.
4. List the most serious causes of syncope.
5. Identify which patients should be admitted after a syncopal episode of unclear cause.

### Case Studies

**Case 1:** A 55-year-old female with a past medical history of hypertension presents to the emergency department (ED) with a chief complaint of lightheadedness. The patient, who is an avid biker, developed a sudden onset of lightheadedness and a sensation that she was going to lose consciousness while on her daily morning bike ride. When emergency medical service (EMS) arrived to the scene her blood pressure (BP) was 90 systolic and did not improve after 1L normal saline (NS). Her blood glucose was 130. On arrival in the ED she is hypotensive at 90/60 and electrocardiogram (ECG) shows sinus bradycardia at 58 bpm. The remainder of her vitals are within normal vitals. She continues to complain of severe lightheadedness and a sharp pain in the left neck and shoulder region.

### Question Prompts:

1. List a prioritized differential for this patient.
  - a. This patient presents with exertional pre-syncope with thoracic and neck discomfort and hypotension. This constellation of historical and physical exam findings should prompt diligent evaluation for diseases that compromise cardiac outflow.
  - b. Myocardial infarction (MI): This patient's neck and shoulder discomfort could be an angina equivalent and her bradycardia could be a manifestation of sinoatrial (SA) node dysfunction caused by a myocardial infarction (MI).
  - c. Pulmonary embolism (PE): The sudden onset of sharp pain associated with lightheadedness and hypotension is concerning for massive PE.
  - d. Aortic dissection: A patient with hypertension who presents with sudden onset sharp pain and syncope should prompt the clinician to consider aortic dissection. Roughly one in eight patients with aortic dissection will experience syncope.
2. Explain the physical exam findings that would help you evaluate for these potentially life-threatening disease processes.



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- a. A thorough vascular exam can be helpful when evaluating a patient with possible aortic dissection. Depending on where the intimal tear originates, patients may have asymmetric radial pulses and upper extremity blood pressures.
  - b. A thorough neurologic examination is also important. Aortic dissections can involve the carotid and/or vertebral arteries producing stroke-like symptoms.
  - c. Pulse asymmetry and neurologic deficits are not commonly associated with either myocardial infarction nor pulmonary embolism.
  - d. Type A dissections involving the aortic root can result in aortic insufficiency with a diastolic murmur.
  - e. Evaluating for signs of deep venous thrombosis (DVT) can steer a clinician towards a diagnosis of pulmonary embolism.
3. This patient never lost consciousness and as such does not meet criteria for true syncope. Does your approach to near-syncope differ from true syncope?
- a. Near-syncope is defined as a sensation of almost losing consciousness. A 2012 cohort study by Grossman et al. compared 244 patients with near-syncope to 293 with true syncope. They found that patients with near-syncope are as likely as those with true syncope to experience critical interventions or adverse events. However, patients with near-syncope were less likely to be admitted, 49% versus 69%. Near-syncope and true syncope should be evaluated and treated similarly.
4. What diagnostic tests would you order?
- a. ECG: The most important test for any patient who presents with syncope. The overall diagnostic yield of an ECG in syncope is 2%-9%. As you might expect, the diagnostic yield is much higher in older patients with known cardiac disease than with young healthy patients. Be aware that 5% of ECGs in patients with Type A dissections will show ST-segment elevation myocardial infarction (STEMI).
  - b. Labs: Complete blood count (CBC), basic metabolic panel (BMP), coagulation studies, type and screen, fecal occult blood test (FOBT), cardiac biomarkers.
  - c. Imaging: Computed tomography (CT) angiography has a 96-100% sensitivity for aortic dissection and is the most readily available imaging modality in the ED.

**Case 2:** A 19-year-old male with no past medical history presents via EMS after suddenly losing consciousness at the gym. The emergency medical technician (EMTs) report that when they arrived the patient was alert and oriented x4 and complaining of pain over his nose from having fallen while he was on the treadmill. His pre-hospital vital signs and glucose are all normal. The patient states that he was running on the treadmill feeling great and then next thing he remembers is waking up on the floor with a bloody nose. He states he was unconscious for maybe a few seconds. He states this happened once before while jogging but he never sought medical attention. His vitals in the ED are all within normal limits. His physical examination is normal. Just before leaving the ED, the EMTs inform you that the gym staff noted some jerking movements in his arms while he was unconscious.



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## Question Prompts:

1. What additional questions should you ask this patient? Is family history pertinent in this case?
  - a. Eliciting a family history of sudden cardiac death is crucial in risk stratifying patients with syncope. Many channelopathies, structural cardiac diseases, and electrophysiological abnormalities are genetic.
2. What is the most important diagnostic test to order on this patient?
  - a. ECG. Chest radiographs, non-contrast head CTs, and blood work have extremely low yield in otherwise young healthy patients with syncope with normal vitals and no current complaints and should not be ordered routinely. Your workup should be dictated by abnormal history and physical exam findings.
3. What ECG findings are cause for alarm?
  - a. Ischemia
  - b. Signs of right heart strain: Right bundle branch block (RBBB), new right axis deviation (RAD), S1Q3T3, deep inverted T waves in V1-V3
  - c. Conduction abnormalities: Left bundle branch block (LBBB), bifascicular block, delta waves, heart block
  - d. Hypertrophic cardiomyopathy: Dagger Q waves in lateral leads, left ventricular hypertrophy (LVH)
  - e. Arrhythmogenic right ventricular cardiomyopathy: Epsilon waves
  - f. Brugada syndrome
  - g. QT prolongation
4. What do you make of his “jerking movements?” What historical and physical exam details can help you distinguish syncope from seizure?
  - a. Myoclonic movements occur in 28% to 90% of patients with neurally mediated syncope.
  - b. Features associated with seizure: Preceding aura, tonic-clonic movements greater than 15-30 seconds, bite on the lateral aspect of the tongue, incontinence, post-ictal state
5. What is the most appropriate disposition for this patient? Why?
  - a. Disposition for any patient with syncope of unclear etiology depends on their risk stratification. This patient has at least 1 high risk factor for adverse outcome—syncope with exertion. This in and of itself is an indication for admission. Other high-risk features include syncope without prodrome, known or signs of, structural heart disease, abnormal ECG, history of heart failure, dyspnea, hypotension, anemia or evidence of gastrointestinal (GI) bleeding, and family history of early sudden cardiac death (less than 50 years of age).
  - b. **Note to facilitators:** This case was inspired from a real clinical case. The patient’s ECG showed a widened QRS with Epsilon waves in V1 and V2. The patient had no family history of sudden cardiac death. He was admitted to a telemetry bed with cardiology consultation. An echocardiogram and cardiac magnetic resonance imaging (MRI) confirmed the diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC). An automatic implantable cardioverter defibrillator (AICD) was placed and he was



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discharged home. Arrhythmogenic right ventricular cardiomyopathy is the second most common cause of sudden cardiac death in young people after hypertrophic cardiomyopathy (HCM). It is an autosomal dominant disease with variable penetrance that causes fibro-fatty displacement of the right ventricular myocardium. The abnormal architecture of the right ventricle is associated with paroxysmal ventricular arrhythmias causing sudden death.

**Case 3:** A 75-year-old male with a history of hypertension, diabetes and coronary artery disease status post coronary artery bypass graft (CABG) presents after an episode of brief loss of consciousness. Patient states he was sitting watching television with his daughter when he became lightheaded and then “blacked out.” Daughter states he slumped over for a few seconds and regained consciousness spontaneously. The patient denies any preceding chest pain or shortness of breath. He is currently asymptomatic. His vitals are all within normal limits and blood glucose is 110. His ECG shows a LBBB which is unchanged from previous studies. Patient is requesting to be discharged but the daughter is worried and does not want to take him home.

## Question Prompts:

1. This patient presents with syncope of unknown cause. Based on the information provided, is this patient low, intermediate, or high-risk?
  - a. This patient has many high-risk features including his age, male sex, abnormal ECG, lack of prodrome, and history of coronary artery disease and structural heart disease. Geriatric syncope should be approached in the same way as all other adults. However, not surprisingly, the incidence of cardiovascular causes of syncope is higher in the elderly. Moreover, many studies have demonstrated higher morbidity and mortality of syncope in the geriatric population. One prospective cohort study showed a mortality of 30% compared to 8% in younger patients. Syncope in elderly patients is also commonly due to polypharmacy and adverse drug reactions.
2. What additional workup might help you risk stratify this patient further?
  - a. In general, routine laboratory evaluation of patients with syncope is low yield. However, in patients with known heart disease, cardiac biomarkers may help risk stratify patients. Although troponin I should not be used to rule out myocardial infarction, an elevated troponin predicts adverse cardiac outcome and may be used for risk stratification. Several small prospective cohort studies have looked at the utility of brain natriuretic peptide (BNP) in distinguishing cardiac from non-cardiac causes of syncope. The pooled sensitivity was around 90%. More research needs to be conducted to determine the true utility of BNP, but it can be useful for risk stratification in certain patients. Hemoglobin and hematocrit can be helpful when hemorrhage is suspected, particularly in patients with risk factors for GI bleed. A metabolic panel can be useful in patients on diuretics or other medications that can predispose to electrolyte derangements and secondary dysrhythmias.
3. Describe the “Risk stratification of syncope in the emergency department (ROSE)” rule.



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- a. According to the BRACES mnemonic, patients should be considered high risk and admitted to the hospital if they meet any of the following seven criteria:
  - i. B: BNP > 300, Bradycardia of <50 bpm
  - ii. R: Rectal exam showing fecal occult blood (if suspicion for GI bleed)
  - iii. A: Anemia with hemoglobin <9.0mg/dL
  - iv. C: Chest pain associated with syncope
  - v. E: ECG showed Q waves in leads other than III
  - vi. S: Saturation of oxygen <94% on room air
- b. A clinical decision rule using the mnemonic BRACES to predict short-term serious outcomes and all-cause death in syncope cases at 1 month resulted in 87% sensitivity, 66% specificity, and 99% negative predictive value in a single-center, prospective trial. While internally validated, this study was a single center, prospective, observational study. The rule has not been adequately externally validated and does not perform well at predicting 1-year adverse outcome of ED syncope patients. It also ignores several social factors that may dictate need for admission.
- c. The San Francisco syncope rule is commonly used to guide physicians to determine patients at low risk for serious outcomes after syncope. Patients are determined low risk if they have no history of congestive heart failure, are not anemic, have a normal EKG, no shortness of breath and are not hypotensive. While initial studies had promising sensitivity, subsequent external validation had lower sensitivity.

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## Appendix M: Resuscitation and Advanced Cardiac Life Support (ACLS)

### Objectives

1. Discuss the key components of advanced cardiac life support (ACLS).
2. Identify important factors in optimizing chest compressions.
3. List the various causes of cardiac arrest and how to treat the reversible causes.
4. Describe the appropriate use of adjunctive tools (intubation, arterial lines, etc.) in cardiac arrest.
5. Describe the interpersonal and interdisciplinary interactions that are important in a successful resuscitation.

### Case Studies

**Case 1:** A 45-year-old male presents to the emergency department (ED) in cardiac arrest. The patient was just discharged from the ED a few minutes prior. He dropped to the ground in the parking garage. Bystanders found him pulseless, initiated cardiopulmonary resuscitation (CPR) and called emergency medical services (EMS).

### Question Prompts:

1. Upon the patient's arrival how would you begin your resuscitation?
  - a. Know the key concepts of ACLS protocol. Continue with high quality chest compressions and determine your algorithm.
  - b. High-quality chest compressions are the only intervention that have consistently shown to have improved outcomes in cardiac arrest. As a result, chest compressions should take priority over intubation, performing ultrasound, giving epinephrine or other medication adjuncts.
  - c. When able, evaluate for a shockable rhythm (ventricular fibrillation or ventricular tachycardia). If present, shock. Every two minutes assess the patient's rhythm and defibrillate if shockable. Protocol states to give epinephrine 1mg every 3-5 minutes. Can also give amiodarone 300mg.
  - d. If rhythm is asystole or pulseless electrical activity (PEA), then give epinephrine 1mg every 3-5 minutes and evaluate for reversible causes.
2. When performing chest compressions, what are appropriate metrics that you should monitor?
  - a. Ensure adequate rate (100-120bpm), full chest recoil and adequate chest compression depth, reduce time during pulse check, ensure chest compressions are being done at the appropriate location.



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3. What important information will help identify possible reversible causes of the patient's cardiac arrest?
  - a. Does the patient have a history of renal failure? Do they have any concern for pulmonary embolism or acute myocardial infarction? Did the patient have any trauma or concern for pneumothorax? What was the patient seen for in the ED prior to arrival? Identify the H's and T's and treat them if present.
    - i. Hypothermia: Rewarm the patient.
    - ii. Hypoxia: Intubate as appropriate.
    - iii. H<sup>+</sup>: Acidosis, treat reversible causes (such as diabetic ketoacidosis). Bicarbonate in cardiac arrest has not been shown to affect outcomes.
    - iv. Hypo/hyperkalemia: If there is a history of renal failure, given an ampule of calcium chloride.
    - v. Hypovolemia: Give fluids or blood products if bleeding.
    - vi. Toxins: Consider toxic causes of cardiac arrest. Medications include tricyclic antidepressants, digoxin, beta-blockers, and calcium channel blockers. Recreational drugs such as opiates or cocaine could contribute to cardiac arrest.
    - vii. Tamponade: Perform a cardiac ultrasound and if present attempt pericardiocentesis.
    - viii. Tension pneumothorax: Perform thoracic ultrasound or needle decompress empirically if considering tension pneumothorax.
    - ix. Thrombosis: Massive pulmonary embolism (PE) or myocardial infarction (MI). Consider thrombolytics in those with high risk for thrombosis.
4. What adjunctive tools are helpful in optimizing your resuscitation?
  - a. Arterial lines may be helpful in determining presence of a pulse, especially in larger patients. It can also help guide CPR because it has been suggested that keeping a diastolic pressure greater than 30mmHg is associated with better outcomes.
  - b. Intubation may not always be associated with better outcomes. This may be due to intubation delaying definitive defibrillation when a shockable rhythm is present. Additionally, over-ventilating can increase intra-thoracic pressure with subsequent decreased perfusion.
  - c. Central lines may be helpful if other forms of access are not available or if there is concern for hypovolemic shock. In these cases, you should use a central line with high flow rates instead of defaulting to a triple lumen catheter.
  - d. Intraosseous (IO) lines allow for quick access; however, flow rates can be somewhat slow especially in tibial IO lines.

**Case 2:** A 65-year-old female presents to the ED in cardiac arrest. The patient is on dialysis. She had just begun dialysis when the nurse noticed she had lost her pulse. CPR was initiated immediately and EMS was called. The patient is 15 minutes away.

### Question Prompts:



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1. How do you prepare yourself prior to the patient's arrival?
  - a. Typically, it's a good idea to simulate the resuscitation in your head prior to arrival. There is no science behind this and people prepare differently.
2. Assuming you are notified of the patient's arrival how would you prepare your room and staff?
  - a. Make sure that appropriate medications are available and ensure that appropriate airway management tools are present. Assign roles to nurses and technicians based on their skill levels.
3. How do you ensure effective communication between you and your team? Why is effective communication important?
  - a. Effective communication optimizes resuscitative efforts. Closed-loop communication is extremely important. It helps ensure that there is no misunderstanding in the message being sent.
4. Why is debriefing after a resuscitation important?
  - a. The debrief is an extremely important element of the resuscitation. This is an open and non-judgmental atmosphere where all participants improve. The debrief is where participants can air their grievances, question certain aspects of care, encourage one another, and reflect on the case. In short, this is a time to prime the process and improve for the next, inevitable resuscitation.

## Suggested Readings:

Hanif MA, Kaji AH, Niemann, JT. Advanced airway management does not improve outcome of out-of-hospital cardiac arrest. *Acad Emerg Med*.2010; 17(6): 926-931. doi: 10.1111/j.1553-2712.2010.00829.x

Sutton RM, Friess SH, Maltese MR, Naim MY, Bratinov G, Weiland TR, et al. Hemodynamic-directed cardiopulmonary resuscitation during in-hospital cardiac arrest. *Resuscitation*. 2014;85(8):983-986. doi: 10.1016/j.resuscitation.2014.04.015

Body R. Teamwork in resus: just like football? St. Emlyn's. <http://stemlynblog.org/teamwork-in-resus-just-like-football/>. Published January 31, 2013. Accessed September 26, 2017.

## Additional References:

Kurz MC, Neumar RW. Adult resuscitation. In: Walls RM, Hockberger RS, Gausche-Hill M, Bakes KM, eds. *Rosen's emergency medicine: concepts and clinical practice*. 9<sup>th</sup> ed. Philadelphia, PA: Elsevier; 2018:85-95e.2.



# DIDACTICS AND HANDS-ON CURRICULUM

## Small Group Evaluation

**The moderator demonstrated adequate knowledge of subject.**

5) Strongly Agree    4) Agree    3) Slightly Agree    2) Disagree    1) Strongly Disagree

**The moderator’s facilitation of the conference facilitated my learning.**

5) Strongly Agree    4) Agree    3) Slightly Agree    2) Disagree    1) Strongly Disagree

**The overall discussion was relevant to the stated topic(s).**

5) Strongly Agree    4) Agree    3) Slightly Agree    2) Disagree    1) Strongly Disagree

**The faculty/resident’s teaching methods (slides, handouts, videos, etc.) were effective.**

5) Strongly Agree    4) Agree    3) Slightly Agree    2) Disagree    1) Strongly Disagree

### Faculty Facilitator Evaluation

#### **1. Preparation – was faculty well prepared?**

Needs Improvement                      Effective                      Exemplary

#### **2. Engaged residents - Encouraged discussion and actively participated, demonstrated enthusiasm?**

Needs Improvement                      Effective                      Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

### Resident Facilitator Evaluation

#### **1. Preparation – was the resident facilitator well prepared?**

Needs Improvement                      Effective                      Exemplary



# DIDACTICS AND HANDS-ON CURRICULUM

## 2. Engaged residents – Controlled and led the session and encouraged discussion, active involvement, and demonstrated enthusiasm?

Needs Improvement

Effective

Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

### Evaluation of the Teaching materials

#### 1. Were the objectives appropriate for the topic?

Needs Improvement

Effective

Exemplary

#### 2. Was the amount of material appropriate?

Too Short

Appropriate

Too Long

Strengths:

Areas for Improvement:

Reviewer Recommendations:



# DIDACTICS AND HANDS-ON CURRICULUM

## Small Group Resident Assessment

Session:		
Facilitator (s):		
DATE:		
Small Group 3	Contributes to group discussion	
	BE/ME/EE	Comments
Resident 1		
Resident 2		
Resident 3		
Resident 4		
Resident 5		
Resident 6		
Resident 7		
Resident 8		
Resident 9		
Resident 10		
Resident 11		
Resident 12		

### BE—Below Expectations

- Minimal discussion during the session
- No discussion on the site discussion board
- Comments not contributory to discussion or distracting to discussion
- Has minimal knowledge of topic as expected of PGY year

### ME—Meets Expectations

- Contributes to group discussion in a meaningful way
- Incorporate textbook/website/podcast reading into discussion
- Has knowledge of topic appropriate to level of training

### EE—Exceeds Expectations

- Contributes to group discussion in a meaningful way
- Incorporate literature into discussion
- Has advanced knowledge of topic