CURRICULUM

Emergency Medicine Curriculum Utilizing the Flipped Classroom Method: Pulmonary Emergencies

Lauren D Branditz, MD[*], Andrew King, MD[*], Colin Kaide, MD[*], Jennifer Mitzman, MD[*^], Benjamin Ostro, MD[*], Daniel R Martin, MD, MBA[*], Nicholas Kman, MD[*], David Bahner, MD[*], Howard Werman, MD[*], Tatiana Thema, MD[*] and Michael Barrie, MD[*]

[*]The Ohio State University Wexner Medical Center, Department of Emergency Medicine, Columbus, OH
[^]Nationwide Children's Hospital, Department of Pediatric Emergency Medicine, Columbus, OH

Correspondence should be addressed to Lauren D Branditz, MD at lauren.branditz@osumc.edu, Twitter: @lbranditz
Submitted: October 24, 2018; Accepted: May 4, 2019; Electronically Published: October 15, 2019; https://doi.org/10.21980/J8F646

ABSTRACT:

Audience: This curriculum created and implemented at The Ohio State University Emergency Medicine Residency program was designed to educate our emergency medicine (EM) residents, PGY-1 to PGY-3, as well as medical students and attending physicians.

Introduction: Shortness of breath is a very common presenting complaint for emergency department patients. This symptom can be caused by a multitude of conditions which require various treatments and interventions. Often, shortness of breath develops due to an underlying pulmonary problem. In 2014, shortness of breath made up about 3.4 million emergency department visits in the United States. This constitutes 2.4% of all emergency department visits.[1] Therefore, resident physicians must be proficient in the differential diagnosis and management of the wide variety of pulmonary emergencies.[1] The American Board of Emergency Medicine (ABEM) EM Model curriculum lists "thoracic-respiratory disorders" as a key component of resident education.[2] 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.[3-5] 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.[5-7] The Ohio State University Emergency Medicine Residency program didactic curriculum recently transitioned to a "flipped classroom" approach.[8-11] 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. [4,7,12]

CURRICULUM

Objectives: We aim to teach the presentation and management of pulmonary emergencies through the creation of a flipped classroom design. This is accomplished via the use of case-based learning. Residents are provided access to a list of learning objectives and several example cases at least one week prior to small group sessions. Each case is associated with question prompts, which residents are encouraged to be ready to answer and discuss during small group sessions. This 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 case-based modules authored by education faculty and content experts based on the core emergency medicine content outlined in the ABEM Model EM curriculum. The Socratic method, used during small group sessions, encourages active participation; small groups also focus on the synthesis and application of knowledge through the discussion of clinical experiences. The use of free open access medical education (FOAM) resources allows learners to work at their own pace and maximize autonomy. Learners are encouraged to use such resources for preparation prior to small group sessions, and also to review and help solidify important points after the conclusion of in-person discussions.

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

USER GUIDE

List of Resources:

Learner Audience:
Medical students, interns, junior residents, senior residents, attending physicians and faculty members

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 their residency training. The pulmonary emergencies module consists of seven 45-60-minute small group sessions. Breaking up the umbrella topic of pulmonary emergencies into seven small group didactics allows for flexibility in terms of scheduling, but at our institution, all seven topics were covered over the course of four weeks.

Topics:
Emergency medicine, flipped classroom, medical education, pulmonary emergencies, pedagogy, teaching

Objectives:
Each chapter within our curriculum has individual objectives as outlined in the appendices; however, educational objectives for the overall curriculum include:

1. Appendix A: Pneumonia
    a. Define pneumonia and its diagnosis.
    b. Understand the difference between community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP).
    c. Identify common pathogens and unique features that may help with predicting a specific pathogen.
    d. Discuss the risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*.
    e. Describe why mortality is higher in elderly patients with pneumonia.
2. Appendix B: Asthma and COPD
    a. Discuss the initial approach to management in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations.
    b. Describe additional treatments for asthma/COPD exacerbations if the initial approach does not improve respiratory distress.
    c. Discuss considerations when utilizing non-invasive ventilation and intubation in patients with asthma and COPD.
    d. Describe the management of the intubated patient with acute asthma exacerbation.
    e. Discuss the role of asthma action plans and discharge regimens in preventing recurrent emergency department (ED) visits for pediatric acute asthma exacerbations.
    f. Discuss the role of antibiotics in patients with COPD exacerbation.
3. Appendix C: Pneumothorax
    a. Discuss the epidemiology, risk factors, and pathophysiology of traumatic, primary spontaneous and secondary spontaneous pneumothoraces.
    b. Discuss various imaging modalities for the diagnosis of pneumothorax.
    c. Describe differences in management of tension, traumatic, and spontaneous pneumothorax.
    d. Discuss the difference between primary and secondary spontaneous pneumothorax.
    e. List the potential complications of tube thoracostomy.
    f. Discuss the appropriate disposition of patients with pneumothorax.
4. Appendix D: Pulmonary Hypertension
    a. Define pulmonary hypertension (PH).
    b. Describe how to diagnose PH.
    c. Discuss the most common causes of PH.
    d. Describe the symptoms of a PH acute exacerbation.
    e. Discuss treatment options, acute and chronic, for PH.
5. Appendix E: Acute Respiratory Distress Syndrome (ARDS) and Inhalants

   a. Define acute respiratory distress syndrome (ARDS).
   b. Name conditions that can be associated with ARDS.
   c. Describe ventilator management in patients with ARDS.
   d. Define conditions suggesting potential benefit of prone positioning.
   e. List indications for extracorporeal membrane oxygenation (ECMO) in severe ARDS.
   f. Discuss inhalation injuries and possible treatment modalities.
6. Appendix F: Septic Pulmonary Embolism
   a. Describe the common clinical signs and symptoms in patients with septic pulmonary emboli.
   b. List the common sources of septic pulmonary emboli and the most common culprit organisms.
   c. Define what patient populations are at high risk of developing septic pulmonary emboli.
   d. Discuss the diagnostic imaging modalities of choice.
   e. Discuss what antibiotics should be initiated to cover the most common pathogens in patients with septic pulmonary emboli.
7. Appendix G: Pleural Effusions and Mediastinum Disorders
   a. Discuss a differential diagnosis for pleural effusions.
   b. List criteria used to distinguish an exudative vs transudative pleural effusion.
   c. Discuss indications and potential complications for ED thoracentesis for pleural effusions.
   d. Discuss management of massive hemothorax.
   e. Discuss causes and treatment for pneumomediastinum.
   f. Discuss workup and management of esophageal perforation.

Brief introduction:

Shortness of breath is often caused by underlying pulmonary dysfunction. In 2014, shortness of breath made up about 3.4 million emergency department visits in the United States. This constitutes 2.4% of all emergency department visits.[1] Therefore, emergency medicine physicians must be well versed in the recognition and treatment of conditions leading to dyspnea. Residency curriculum must include a thorough review of pulmonary emergencies so that residents learn to recognize and treat such conditions. The flipped classroom learning approach is 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.[5] The active learning fostered by this curriculum increases faculty and learner engagement and interaction time, which is typically absent in traditional lecture-based formats.[6,13] Education literature shows that resident learners prefer learning activities that involve small group discussion, are case- or skill-based, and emphasize the application of newly obtained knowledge.[4,5] 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.[5,10,13] For these reasons, we developed a flipped classroom curriculum at The Ohio State University Emergency Medicine Residency program. This pulmonary emergencies curriculum is one of several topics in our overall 18-month didactic curriculum.

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.[7] Previously, we used a traditional lecture format in our residency curriculum despite overwhelming evidence for a more hands-on, "flipped classroom" approach.[9,10] From the 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.[6]

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.[11] This weekly small group curriculum has now replaced two hours of traditional lecture-based didactics. Learners divide into small groups, and a faculty facilitator guides each group session. Small groups are composed of a mix of junior and senior residents. This mix of learners allows those who are more senior to teach and mentor junior residents, while simultaneously expanding their own knowledge of topics via discussion with a faculty member. Since implementation, residents and educators are engaging in new, valuable flipped classroom learning communities at The Ohio State University Emergency Medicine Residency program. 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:

We aim to teach the presentation and management of pulmonary emergencies through the creation of a flipped classroom design. This 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. After completing the Pulmonary Emergencies Module, resident learners will exhibit mastery within this content area and will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult pulmonary emergencies.

Objectives of the curriculum:

Each chapter within our curriculum has individual objectives which are listed below.

1. Appendix A: Pneumonia
    a. Define pneumonia and its diagnosis.
    b. Understand the difference between community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP).
    c. Identify common pathogens and unique features that may help with predicting a specific pathogen.
    d. Discuss the risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*.
    e. Describe why mortality is higher in elderly patients with pneumonia.
2. Appendix B: Asthma and COPD
    a. Discuss the initial approach to management in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations.
    b. Describe additional treatments for asthma/COPD exacerbations if the initial approach does not improve respiratory distress.
    c. Discuss considerations when utilizing non-invasive ventilation and intubation in patients with asthma and COPD.
    d. Describe the management of the intubated patient with acute asthma exacerbation.
    e. Discuss the role of asthma action plans and discharge regimens in preventing recurrent emergency department (ED) visits for pediatric acute asthma exacerbations.
    f. Discuss the role of antibiotics in patients with COPD exacerbation.
3. Appendix C: Pneumothorax
    a. Discuss the epidemiology, risk factors, and pathophysiology of traumatic, primary spontaneous and secondary spontaneous pneumothoraces.
    b. Discuss various imaging modalities for the diagnosis of pneumothorax.
    c. Describe differences in management of tension, traumatic, and spontaneous pneumothorax.
    d. Discuss the difference between primary and secondary spontaneous pneumothorax.
    e. List the potential complications of tube thoracostomy.
    f. Discuss the appropriate disposition of patients with pneumothorax.
4. Appendix D: Pulmonary Hypertension
    a. Define pulmonary hypertension (PH).
    b. Describe how to diagnose PH.
    c. Discuss the most common causes of PH.
    d. Describe the symptoms of a PH acute exacerbation.
    e. Discuss treatment options, acute and chronic, for PH.
5. Appendix E: Acute Respiratory Distress Syndrome (ARDS) and Inhalants
    a. Define acute respiratory distress syndrome (ARDS).
    b. Name conditions that can be associated with ARDS.
    c. Describe ventilator management in patients with ARDS.
    d. Define conditions suggesting potential benefit of prone positioning.
    e. List indications for extracorporeal membrane oxygenation (ECMO) in severe ARDS.
    f. Discuss inhalation injuries and possible treatment modalities.
6. Appendix F: Septic Pulmonary Embolism
    a. Describe the common clinical signs and symptoms in patients with septic pulmonary emboli.

   b. List the common sources of septic pulmonary emboli and the most common culprit organisms.
   c. Define what patient populations are at high risk of developing septic pulmonary emboli.
   d. Discuss the diagnostic imaging modalities of choice.
   e. Discuss what antibiotics should be initiated to cover the most common pathogens in patients with septic pulmonary emboli.
7. Appendix G: Pleural Effusions and Mediastinum Disorders
   a. Discuss a differential diagnosis for pleural effusions.
   b. List criteria used to distinguish an exudative vs transudative pleural effusion.
   c. Discuss indications and potential complications for ED thoracentesis for pleural effusions.
   d. Discuss management of massive hemothorax.
   e. Discuss causes and treatment for pneumomediastinum.
   f. Discuss workup and management of esophageal perforation.

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. A survey was administered to each resident prior to initiation of the curricular innovation and repeated at the conclusion of the first 18-month cycle. Learners and educators were enthusiastic about the conference structure and expressed a preference for it rather than the previous, lecture-based didactics. More recently, during the second 18-month cycle of the flipped classroom curriculum, students 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 who felt that our grand rounds sessions during the same time were good or excellent. This curriculum has been delivered to two cohorts of learners, delivering the content twice in three years to about 50 residents per cycle. On the most recent iteration, residents evaluated the teaching methods as effective, with an average rating of 4 out of 5 (4 being agree, 5 being strongly agree).

The curriculum is critically evaluated and updated by education faculty members in order to ensure educational material remains current and consistent with the emergency medicine core content.

References/Further Readings:

1. Rui P, Kang K, Albert M. National Hospital Ambulatory Medical Care Survey: 2013 Emergency Department Summary Tables. http://www.cdc.gov/nchs/data/ahcd/nhamcs_emergency/2013_ed_web_tables.pdf. Accessed January 29th, 2019.
2. Counselman FL, Babu K, Edens MA, Gorgas DL, Hobgood C, Marco CA, Katz E, Rodgers K, Stallings LA, Wadman MC, for the 2016 EM Model Review Task Force; Beeson MS, Keehbauch JN, for the American Board of Emergency Medicine. The 2016 model of the clinical practice of emergency medicine. *J Emerg Med.* 2017; March 25; pii: S0736-4679(17)30108-7. doi: 10.1016/j.jemermed.2017.01.040. [Epub ahead of print.] https://www.abem.org/public/resources/em-model. Accessed July 26, 2019.
3. Galway LP, Corbett KK, Takaro TK, Tairyan K, Frank E. A novel integration of online and flipped classroom instructional models in public health higher education. *BMC Med Educ*. 2014; 29(14):181. doi: 10.1186/1472-6920-14-181.
4. Mortensen CJ, Nicholson AM. The flipped classroom stimulated greater learning and is a modern 21st century approach to teaching today's undergraduates. *J Anim Sci*. 2015; 93(7):3722-3731. doi: 10.2527/jas.2015-9087.
5. Tan E, Brainard A, Larkin G. Acceptability of the flipped classroom approach for in-house teaching in emergency medicine. *Emer Med Australas*. 2015;27(5):453-459. doi: 10.1111/1742-6723.12454.
6. Rose E, Claudius I, Tabatabai R, Kearl L, Behar S, Jhun P. The flipped classroom in emergency medicine using online videos with interpolated questions. *J Emerg Med*. 2016;51(3):284-291. doi: 10.1016/j.jemermed.2016.05.033.
7. Young TP, Bailey CJ, Guptill M, Thorp AW, Thomas TL. The flipped classroom: a modality for mixed asynchronous and synchronous learning in a residency program. *West J Emerg Med*. 2015;15(7):938-944. doi: 10.5811/westjem.2014.10.23515.
8. Heitz C, Prusakowski M, Willis G, Franck C. Does the concept of the "Flipped Classroom" extend to the emergency medicine clinical clerkship? *West J Emerg Med*. 2015;16(6):851-855. doi: 10.5811/westjem.2015.9.27256.
9. Liebert CA, Lin DT, Mazer LM, Bereknyei S, Lau JN. Effectiveness of the surgery core clerkship flipped classroom: a prospective cohort trial. *Am J Surg*. 2016; 211(2):451-457. doi: 10.1016/j.amjsurg.2015.10.004.

10. Sockalingam S, James SL, Sinyi R, et al. A flipped classroom approach to improving the quality of delirium care using an interprofessional train-the-trainer program. *J Contin Educ Health Prof*. 2016;36(1):17-23. doi: 10.1097/CEH.0000000000000025.
11. Tainter CR, Wong NL, Cudemus-Deseda GA, Bittner EA. The "flipped classroom" model for teaching in the intensive care unit: rationale, practical considerations, and an example of successful implementation. *J Intensive Care Med*. 2016;32(3):187-196. doi: 10.1177/0885066616632156.
12. Moffett J. Twelve tips for "flipping" the classroom. *Med Teach*. 2014;37(4):331-336. doi: 10.3109/0142159X.2014.943710.
13. Liebert CA, Mazer L, Bereknyei Merrell S, Lin DT, Lau JN. Student perceptions of a simulation-based flipped classroom for the surgery clerkship: a mixed-methods study. *Surgery*. 2016;160(3):591-598. doi: 10.1016/j.surg.2016.03.034.

Educational resources are available within each individual appendix of this pulmonary emergencies curricular module; however, a complete list of resources and educational materials are listed below.

1. Schuetz P, Branche A, Mueller B. Low procalcitonin, community acquired pneumonia, and antibiotic therapy – Authors reply. *The Lancet Infectious Diseases*. 2018;18(5):497-498. doi: 10.1016/s1473-3099(18)30226-3.
2. Adams D, Welch JL, Kline JA. (2014). Clinical utility of an age-adjusted D-dimer in the diagnosis of venous thromboembolism. *Annals of Emergency Medicine,* 64(3):232-234. doi: 10.1016/j.annemergmed.2013.12.018.
3. Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis*. 2007;44 Suppl 2: S27-72. doi: 10.1086/511159.
4. Dressler DD. NEJM Journal Watch: Summaries of and commentary on original medical and scientific articles from key medical journals. https://www.jwatch.org/na46012/2018/03/06/steroids-community-acquired-pneumonia-body-evidence-grows. Published February 1, 2018. Accessed July 16, 2019.
5. Prats M. Acute Dyspnea. Ultrasound GEL. https://www.ultrasoundgel.org/?search=pneumonia. Published May 22, 2017. Accessed June 27, 2019.
6. Aujesky D, Auble TE, Yearly DM, et al. Prospective comparison of three validated prediction rules for prognosis in community-acquired pneumonia. *Am J Med*. 2005;118(4):384-92. doi: 10.1016/j.amjmed.2005.01.006.
7. Bartlett JG. Diagnostic approach to patients with CAP. In: Ramirez JA, Bond S, eds. *UpToDate*. Waltham, MA: UpToDate Inc. https://www.uptodate.com/contents/diagnostic-approach-to-community-acquired-pneumonia-in-adults? Updated Jan 23, 2019. Accessed July 16, 2019.
8. Gottlieb M, Bailitz J. Do corticosteroids provide benefit to patients with community-acquired pneumonia? *Ann Emerg Med*. 2016;67(5):640-642. doi: 10.1016/j.annemergmed.2015.12.008.
9. Sharp AL, Jones JP, Wu I, et al. CURB-65 performance among admitted and discharged emergency department patients with community acquired pneumonia. *Acad Emerg Med.* 2016;23(4):400-5. doi: 10.1111/acem.12929.
10. Viasus D, Núñez-Ramos JA, Viloria SA, Carratalà J. Pharmacotherapy for community-acquired pneumonia in the elderly. *Expert Opin Pharmacother.* 2017;18(10):957-964. doi: 10.1080/14656566.2017.1340940.
11. Yandiola PP, Capelastegui A, Quintana J, et al. Prospective comparison of severity scores for predicting clinically relevant outcomes for patients hospitalized with community-acquired pneumonia. *Chest*. 2009;135(6):1572-9. doi: 10.1378/chest.08-2179.
12. Balk DS, Lee C, Schafer J, et al. Lung ultrasound compared to chest X-ray for diagnosis of pediatric pneumonia: a meta-analysis. *Pediatr Pulmonol.* 2018;53(8):1130-1139. doi: 10.1002/ppul.24020.
13. Jat KR, Khairwa A. Levalbuterol versus albuterol for acute asthma: A systematic review and meta-analysis. Pulmonary Pharmacology & Therapeutics. https://www.sciencedirect.com/science/article/pii/S10945391200171X?via=ihub. Published November 30, 2012. Accessed July 17, 2019.
14. Rowe BH, Bretzlaff J, Bourdon C, Bota G, Blitz S, Camargo Jr CA. Magnesium sulfate for treating exacerbations of acute asthma in the emergency department. https://www.cochrane.org/CD001490/AIRWAYS_magnesium-sulfate-treating-exacerbations-acute-asthma-emergency-department. Accessed July 17, 2019.
15. Soroksky A, Klinowski E, Ilgyev E, et al. Noninvasive positive pressure ventilation in acute asthmatic attack. European Respiratory Society. https://err.ersjournals.com/content/19/115/39. Published March 1, 2010. Accessed June 27, 2019.
16. Reuben AD, Harris AR. Heliox for asthma in the emergency department: a review of the literature. *Emergency Medicine Journal*. https://emj.bmj.com/content/21/2/131. Published March 1, 2004. Accessed July 17, 2019.
17. Walker G. Nebulized Ipratropium for Asthma Attack. TheNNT. http://www.thennt.com/nnt/nebulized-ipratropium-for-asthma-attack/. Accessed July 17, 2019.
18. Bhogal SK, Mcgillivray D, Bourbeau J, Benedetti A, Bartlett S, Ducharme FM. Early Administration of Systemic Corticosteroids Reduces Hospital Admission Rates for

Children with Moderate and Severe Asthma Exacerbation. *Annals of Emergency Medicine*. 2012;60(1). doi: 10.1016/j.annemergmed.2011.12.027.
19. Kenyon, CC, Rubin DM, Zorc JJ, Mohamad Z, Faerber JA, Feudther C. Childhood asthma hospital discharge medication fills and risk of subsequent readmission. *J Pediatr*. 2015;166(5):1121-1127. doi: 10.1016/j.jpeds.2014.12.019.
20. Andrews AL, Russell WS, Titus MO, et al. Quality improvement methods improve inhaled corticosteroid prescribing in the emergency department. *J Asthma*. 2014;51(7)737-742. doi: 10.3109/02770903.2014.911885.
21. Ducharme, FM, Zemek RL, Chalut D, et al. Written action plan in pediatric emergency room improves asthma prescribing, adherence, and control. *Am J Resp Crit Care Med*. 2011;183(2):195-203. doi: 10.1164/rccm.201001-0115OC.
22. Koninckx M, Buysse C, de Hooq M. Management of status asthmaticus in children. *Paediatr Respir Rev*. 2013;14(2):78-85. doi: 10.1016/j.prrv.2013.03.003.
23. Yoos HL, Kitzman H, McMullen A, Sidora K. Symptom perception in childhood asthma: how accurate are children and their parents. *J Asthma*. 2003;40(1):27-39. doi: 10.1081/JAS-120017204.
24. Alrajhi K, Woo MY, Vaillancourt C. Test characteristics of ultrasonography for the detection of pneumothorax: a systematic review and meta-analysis. *Chest*. 2012;141(3):703-708. doi: 10.1378/chest.11-0131.
25. Inaba K, Lustenberger T, Recinos G, et al. Does size matter? A prospective analysis of 28 - 32 versus 36 - 40 French chest tube size in trauma. *J Trauma Acute Care Surg*. 2012;72(2):422- 427. doi: 10.1097/TA.0b013e3182452444.
26. Andrivet P, Djedaini K, Teboul JL, Brochard L, Dreyfuss D. Spontaneous pneumothorax. comparison of thoracic drainage vs immediate or delayed needle aspiration. *Chest*. 1995:108(2):335-339. doi: 10.1378/chest.108.2.335.
27. Bense L, Eklund G, Wiman LG. Smoking and the increased risk of contracting spontaneous pneumothorax. *Chest*. 1987;92(6):1009-1012. doi: 10.1378/chest.92.6.1009.
28. Carfarotti S, Dall'Armi V, Cusumano G, et al. Small-bore wire-guided chest drains: safety, tolerability, and effectiveness in pneumothorax, malignant effusions, and pleural empyema. *J Thorac Cardiovasc Surg*. 2011;141(3):683-687. doi: 10.1016/j.jtcvs.2010.08.044.
29. Carson-Chahhoud KV, Wakai A, van Agteren JE, et al. Simple aspiration versus intercostal tube drainage for primary spontaneous pneumothorax in adults. *Cochrane Database Syst Rev*. 2017;9:CD004479. doi: 10.1002/14651858.CD004479.pub3.
30. Clemency BM, Tanski CT, Rosenberg M, May PR, Consiglio JD, Lindstrom HA. Sufficient catheter length for pneumothorax needle decompression: a meta-analysis. *Prehosp Disaster Med*. 2015;30(3):249-253. doi: 10.1017/S1049023X15004653.
31. Devanand A, Koh MS, Ong TH, et al. Simple aspiration versus chest-tube insertion in the management of primary spontaneous pneumothorax: a systematic review. *Respir Med*. 2004;98(7):579-80. doi: 10.1016/j.rmed.2004.04.006.
32. Inaba K, Ives C, McClure K, et al. Radiologic evaluation of alternative sites for needle decompression of tension pneumothorax. *Arch Surg*. 2012;147(9):813-818. doi: 10.1001/archsurg.2012.751.
33. Light RW. Pneumothorax. In: *Pleural Diseases*. 5th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott, Williams and Wilkins; 2013: 306-339.
34. MacDuff A, Arnold A, Harvery J, BTS Pleural Disease Guideline Group. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. *Thorax*. 2012;65 Suppl 2: ii18-31. doi: 10.1136/thx.2010.136986.
35. Millo NZ, Plewes C, Rowe BH, Low G. Appropriateness of CT of the chest, abdomen, and pelvis in motorized blunt force trauma patients without signs of significant injury. *AJR Am J Roentgenol*. 2011;197(6):1393-1398. doi: 10.2214/AJR.11.6536.
36. Radhi S, Alexander T, Ukwu M, Saleh S, Morria A. Outcome of HIV-associated *Pneumocystis* pneumonia in hospitalized patients from 2000 through 2003. *BMC Infect Dis*. 2008; 8:118. doi: 10.1186/1471-2334-8-118.
37. Tsai WK, Chen W, Lee JC, et al. Pigtail catheters vs large-bore chest tubes for management of secondary spontaneous pneumothoraces in adults. *Am J Emerg Med*. 2006;24(7):795-800. doi: 10.1016/j.ajem.2006.04.006.
38. Wilkerson RG, Stone MB. Sensitivity of bedside ultrasound and supine anteroposterior chest radiographs for the identification of pneumothorax after blunt trauma. *Acad Emerg Med*. 2010;17(1):11-17. doi: 10.1111/j.1553-2712.2009.00628.x.
39. McLaughlin VV, Archer SL, Badesch DB, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension, a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. *Circulation*. 2009;119(16):2250-2294. doi: 10.1161/CIRCULATIONAHA.109.192230.
40. New 2014 pulmonary hypertension guidelines released. PulmCCM. https://pulmccm.org/review-articles/new-2014-pulmonary-hypertension-guidelines-released/. Published September 5, 2014. Accessed March 19, 2018.
41. Sildenafil worked in pulmonary hypertension due to CHF with preserved EF (RCT). PulmCCM.

https://pulmccm.org/randomized-controlled-trials/sildenafil-worked-in-pulmonary-hypertension-due-to-chf-with-preserved-ef/. Published December 26, 2011. Accessed March 19, 2018.
42. Taichman DB, Ornelas J, Chung L, et al. Pharmacologic Therapy for pulmonary arterial hypertension in adults: CHEST guidelines and expert panel report. *Chest*. 2014;146(2):449-475. doi: 10.1378/chest.14-0793.
43. ARDSnet NIH NHLBI ARDS Clinical Network Mechanical Ventilation Protocol Summary. NHLBI ARDS Network. http://ardsnet.org/files/ventilator_protocol_2008-07.pdf. Published July 2008. Accessed July 30, 2019.
44. Extracorporeal Membrane Oxygenation for ARDS in Adults: *NEJM*. https://www.nejm.org/doi/full/10.1056/NEJMct1103720. Accessed July 19, 2019.
45. Baydala L. Inhalant abuse. *Paediatrics and Child Heath*. 2010;15(7):443-448. doi: 10.1007/springerreference_44310.
46. Ranieri VM, Rubenfeld GD, Thompson BT, et al, ARDS Definition Task Force. Acute respiratory distress syndrome: The Berlin Definition. *JAMA*. 2012;307(23):2526-2533. doi: 10.1001/jama.2012.5669.
47. Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. *N Engl J Med*. 2011;365(20):1905-1914. doi: 10.1056/NEJMct1103720.
48. Centers for Disease Control and Prevention. Facts about Chlorine. Centers for Disease Control and Prevention. https://emergency.cdc.gov/agent/chlorine/basics/facts.asp . Updated April 10, 2013. Accessed January 24, 2018.
49. Gorguner M, Aslan S, Inandi T, Cakir Z. Reactive airways dysfunction syndrome in housewives due to a bleach-hydrochloric acid mixture. *Inhal Toxicol*. 2004;16(2):87-91. doi: 10.1080/08958370490265004.
50. Guérin C, Reignier J, Richard JC, et al. Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med*. 2013;368(23):2159-2168. doi: 10.1056/NEJMoa1214103.
51. Henderson WR, Griesdale DE, Dominelli P. Does prone positioning improve oxygenation and reduce mortality in patients with acute respiratory distress syndrome? *Can Respir J*. 2014;21(4):213-215. doi:10.1155/2014/472136.
52. Lemière C, Boulet LP, Cartier A. Reactive airways dysfunction syndrome and irritant-induced asthma. In: Barnes PJ, Hollingsworth H, eds. UpToDate, Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/reactive-airways-dysfunction-syndrome-and-irritant-induced-asthma. Updated May 31, 2018. Accessed July 19, 2019.
53. Przybysz TM, Heffner AC. Early treatment of severe acute respiratory distress syndrome. *Emerg Med Clin North Am*. 2016;34(1):1-14. doi: 10.1016/j.emc.2015.08.001.
54. Rogers R, Herbert M, Swadron S. Episode 125 C3 Project: Asthma, Pneumothorax and ARDS. EM: RAP. https://www.emrap.org/episode/february2012/c3projectasthma. Published February 2012. Accessed January 24, 2018.
55. Walker PF, Buehner MF, Wood LA, et al. Diagnosis and management of inhalation injury: an updated review. *Crit Care*. 2015;19:351. doi: 10.1186/s13054-015-1077-4.
56. Anderson CE, Loomis GA. Recognition and prevention of inhalant abuse. *Am Fam Physician*. 2003;68(5):869-874.
57. Nickson C. Infective endocarditis. Life in the Fast Lane Medical Blog. https://litfl.com/infective-endocarditis/. Published April 24, 2019. Accessed July 30, 2019.
58. Duke criteria for infective endocarditis. MDCalc. https://www.mdcalc.com/duke-criteria-infective-endocarditis. Accessed July 30, 2019.
59. Smith C. Infective endocarditis – spoon feed. Journal Feed. https://journalfeed.org/article-a-day/2018/infective-endocarditis-spoon-feed-version. Published July 28, 2018. Accessed July 30, 2019.
60. Kuhlman JE, Fishman EK, Teigen C. Pulmonary septic emboli: diagnosis with CT. *Radiology*. 1990;174(1):211-231. doi: 10.1148/radiology.174.1.2294550.
61. Chow AW. Deep neck space infections in adults. In: Durand ML, Sullivan M, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/deep-neck-space-infections-in-adults. Updated June 6, 2019. Accessed August 26, 2019.
62. Holm K, Bank S, Nielsen H, Kristensen LH, Prag J, Jensen A. The role of Fusobacterium necrophorum in pharyngotonsillitis – a review. *Anaerobe.* 2016:42:89-97. doi: 10.1016/j.anaerobe.2016.09.006.
63. Johannesen KM, Bodtger U. Lemierre's syndrome: current perspective on diagnosis and management. *Infect Drug Resist.* 2016; 9:221-227. doi: 10.2147/IDR.S95050.
64. Karpel JP. Overview of pulmonary disease in people who inject drugs. In: King TE Jr, Hollingsworth H, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/overview-of-pulmonary-disease-in-people-who-inject-drugs. Updated August 5, 2019. Accessed August 26, 2019.
65. Spelman, D. Suppurative (septic) thrombophlebitis. In: Sexton DJ, Sullivan M, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/suppurative-septic-thrombophlebitis. Updated Apr 4, 2018. Accessed July 23, 2019.
66. Stubington TJ, James P. Lemeirre's syndrome: a pain in the neck with far-reaching consequences. *BMJ Case Rep.* 2018;1-4 pii: bcr-2017-222723. doi: 10.1136/bcr-2017-222723.
67. Wong KS, Lin TY, Huang YC, Hsia SH, Yang PH, Chu SM. Clinical and radiographic spectrum of septic pulmonary

embolism. *Arch Dis Child.* 2002;87(4):312-315. doi: 10.1136/adc.87.4.312.
68. Ye R, Zhao L, Wang C, Wu X, Yan H. Clinical characteristics of septic pulmonary embolism in adults: a systematic review. *Respir Med.* 2014; 08(1):1-8. doi: 10.1016/j.rmed.2013.10.012.
69. Pickens A, Koyfman A, Long B, Singh M. EM in 5: Lemierre's Disease. *EmDocs.* https://emin5.com/2013/10/26/lemierres-disease/. Published October 26, 2013. Accessed October 16, 2018.
70. PACCM MICU Education. Pleural disease diagnostics. https://paccm.pitt.edu/MICU/PACCM_Fellows_Ed_2017/handouts/Pleural Disease Diagnostics.pdf. Published 2015. Accessed July 23, 2019.
71. Petiot A, Tawk S, Ghaye B. Re-expansion pulmonary oedema. *Lancet.* 2018:392(10146):507. doi: 10.1016/S0140-6736(18)31722-7.
72. Mowery NT, Gunter OL, Collier BR, et al. Practice management guidelines for management of hemothorax and occult pneumothorax. *J Trauma*. 2011;70(2):510-518. doi: 10.1097/TA.0b013e31820b5c31.
73. Heffner JE. Diagnostic evaluation of pleural effusion in adults: Initial testing. In: Braddus VC, Finlay G, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/diagnostic-evaluation-of-a-pleural-effusion-in-adults-initial-testing. Updated Apr 2, 2018. Accessed July 23, 2019.
74. Sarko J, Stapczynski JS. Respiratory distress. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*. 8th ed. New York, NY: McGraw Hill; 2016:427-435.
75. AnatomyZone. Mediastinum - anatomy tutorial. YouTube. https://www.youtube.com/watch?v=Io4S7HmnNqE. Published November 14, 2011. Accessed January 16, 2018.
76. AnatomyZone. Muscles of the thoracic wall - 3D anatomy tutorial. YouTube. https://www.youtube.com/watch?v=mVLXqICrsdo. Published February 2, 2013. Accessed January 16, 2018.
77. Boylan AM, Broaddus CV. Pleural disease. In: Schraufnagel DE. *Breathing in America: Diseases, Progress, and Hope.* United States of America. American Thoracic Society; 2010:145-154.
78. Cadogan M. CXR interpretation. Life in the Fast Lane. https://lifeinthefastlane.com/investigations/cxr-interpretation/. Published August 29, 2017. Accessed January 16, 2018.
79. Casas A, Pavía J, Maldonado D. Respiratory muscle disorders in chest wall diseases. *Arch Bronconeumol*. 2003;39(8):361-366. doi: 10.1016/S0300-2896(03)75404-0.
80. Ellis SM, Flower C. Pleural disease. In: Ostensen H, Pettersson H, eds. *The WHO Manual of Diagnostic Imaging: Radiographic Anatomy and Interpretation of the Chest and the Pulmonary System.* Geneva: World Health Organization; 2006:105-118.
81. Jeung MY, Gangi A, Gasser B, et al. Imaging of chest wall disorders. *Radiographics.* 1999;19(3):617-37. doi: 10.1148/radiographics.19.3.g99ma02617.
82. Maskell N, Rahman N. Pleural disease. The Lancet Respiratory Medicine. http://www.thelancet.com/series/pleural-disease. Published July 7, 2015. Accessed January 16, 2018.
83. Mayo P. Bedside pleural ultrasonography: equipment, technique, and the identification of pleural effusion and pneumothorax. In: Broaddus VC, Finlay G, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/bedside-pleural-ultrasonography-equipment-technique-and-the-identification-of-pleural-effusion-and-pneumothorax. Updated Oct 26, 2018. Accessed July 23, 2019.
84. Nickson C. Trauma! Initial assessment and management. Life in the Fast Lane. https://litfl.com/trauma-initial-assessment-and-management/. Published February 28, 2019. Accessed July 30, 2019.
85. Pleural disease. Life in the Fast Lane. https://lifeinthefastlane.com/wp-content/uploads/2009/11/pleural-disease.pdf. Published November 2009. Accessed January 16, 2018.
86. Whitaker R, White J. Essential surface and related anatomy for clinical practice. LifeintheFastLane.com. https://lifeinthefastlane.com/wp-content/uploads/2010/10/surfaceanatomy.pdf. Published October 30, 2009. Accessed January 16, 2018.

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
Appendix A: Pneumonia	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of Pneumonia.	1. Define pneumonia and its diagnosis. 2. Understand the difference between community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP). 3. Identify common pathogens and unique features that may help with predicting a specific pathogen. 4. Discuss the risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*. 5. Describe why mortality is higher in elderly patients with pneumonia.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 4 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

Appendix B: Asthma and COPD	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of Asthma (pediatric and adult) and COPD.	1. Discuss the initial approach to management in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations. 2. Describe additional treatments for asthma/COPD exacerbations if the initial approach does not improve respiratory distress. 3. Discuss considerations when utilizing non-invasive ventilation and intubation in patients with asthma and COPD. 4. Describe the management of the intubated patient with acute asthma exacerbation. 5. Discuss the role of asthma action plans and discharge regimens in preventing recurrent emergency department (ED) visits for pediatric acute asthma exacerbations. 6. Discuss the role of antibiotics in patients with COPD exacerbation.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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
Appendix C: Pneumothorax	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of Pneumothorax, including tension physiology and traumatic vs. spontaneous.	1. Discuss the epidemiology, risk factors, and pathophysiology of traumatic, primary spontaneous and secondary spontaneous pneumothoraces. 2. Discuss various imaging modalities for the diagnosis of pneumothorax. 3. Describe differences in management of tension, traumatic, and spontaneous pneumothorax. 4. Discuss the difference between primary and secondary spontaneous pneumothorax. 5. List the potential complications of tube thoracostomy. 6. Discuss the appropriate disposition of patients with pneumothorax.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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
Appendix D: Pulmonary Hypertension	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of Pulmonary Hypertension.	1. Define pulmonary hypertension (PH). 2. Describe how to diagnose PH. 3. Discuss the most common causes of PH. 4. Describe the symptoms of a PH acute exacerbation. 5. Discuss treatment options, acute and chronic, for PH.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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
Appendix E: Acute Respiratory Distress Syndrome (ARDS) and Inhalants	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of acute respiratory distress syndrome (ARDS) and inhalant exposure/abuse.	1. Define acute respiratory distress syndrome (ARDS). 2. Name conditions that can be associated with ARDS. 3. Describe ventilator management in patients with ARDS. 4. Define conditions suggesting potential benefit of prone positioning. 5. List indications for extracorporeal membrane oxygenation (ECMO) in severe ARDS. 6. Discuss inhalation injuries and possible treatment modalities.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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
Appendix F: Septic Pulmonary Emboli	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of septic pulmonary embolism.	1. Describe the common clinical signs and symptoms in patients with septic pulmonary emboli. 2. List the common sources of septic pulmonary emboli and the most common culprit organisms. 3. Define what patient populations are at high risk of developing septic pulmonary emboli. 4. Discuss the diagnostic imaging modalities of choice. 5. Discuss what antibiotics should be initiated to cover the most common pathogens in patients with septic pulmonary emboli.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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
Appendix G: Pleural Effusions and Mediastinum Disorders	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of pleural effusions and mediastinum disorders.	1. Discuss a differential diagnosis for pleural effusions. 2. List criteria used to distinguish an exudative vs transudative pleural effusion. 3. Discuss indications and potential complications for ED thoracentesis for pleural effusions. 4. Discuss management of massive hemothorax. 5. Discuss causes and treatment for pneumomediastinum. 6. Discuss workup and management of esophageal perforation.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK) Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

Appendix A:
Pneumonia

Objectives

By the end of this small group session, the learner will be able to:

1. Define pneumonia and its diagnosis.
2. Understand the difference between community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP).
3. Identify common pathogens and unique features that may help with predicting a specific pathogen.
4. Discuss the risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*.
5. Describe why mortality is higher in elderly patients with pneumonia.

Case Studies

Case 1: A 25-year-old graduate student presents with a productive cough and fever that is getting worse. He has had a sore throat and a runny nose for 5 days, and also some ear pain. The cough started 2 days ago and he is bringing up green sputum. He has no other medical problems. Vital signs show a temperature (T) of 101°F and heart rate of 110 beats per minute, but normal respiratory rate, oxygen saturation, and blood pressure. Breath sounds are reduced on the left and chest radiograph (CXR) shows a left lower lobe infiltrate and a positive spine sign.

Question Prompts:

1. Describe the diagnostic criteria for pneumonia and how you would work up this patient.
    a. Common clinical features of pneumonia are:
        i. Cough (80% to 90%)
        ii. Fatigue (90%)
        iii. Fever (70% to 75%)
        iv. Dyspnea (67% to 75%)
        v. Sputum production (60% to 65%)
    b. Pneumonia findings on chest x-ray (CXR) vary:
        i. No infiltrate
        ii. Lobar infiltration (pneumococcal, *Staphylococcus aureus*)
        iii. Multiple areas of infiltration (*S. aureus*, *Pseudomonas*, *Haemophilus influenza*)
        iv. Diffuse infiltrates (*Moraxella catarrhalis*)
        v. Patchy infiltrates with hilar adenopathy (*Legionella*)
        vi. Parapneumonic effusion (*S. aureus*, *Haemophilus influenza*)
        vii. Empyema (*S. aureus*, *Pseudomonas*)
        viii. Abscesses (*Klebsiella*)

   c. This patient clinically fits a diagnosis of community-acquired pneumonia. The CXR confirms a lobar infiltrate. This should be treated with antibiotics. No further work up needs to be obtained. Remember, though, that pneumonia is a clinical diagnosis and not purely a radiographic diagnosis. Chest radiographs often lag behind the clinical course of pneumonia. Therefore, one should not depend on imaging findings to rule pneumonia in or out. Take vital signs, duration of illness, and physical exam into consideration when making this diagnosis and evaluating similar patients.

2. What are some associations between clinical presentations of pneumonia and specific pathogens?
   a. Sudden onset with fever, rigors, pleuritic pain + rust colored sputum + lobar = *Streptococcus pneumoniae* (pneumococcus)
   b. Similar presentation to above but with "currant jelly sputum" = *Klebsiella pneumoniae*
   c. Causes of pneumonia in chronic obstructive pulmonary disease (COPD) patients = *H. influenza* or *Moraxella*
   d. Pneumonia following influenza = pneumococcus or *S. aureus*
   e. Atypical pneumonia = *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae* and *Legionella*
   f. Pneumonia with gastrointestinal (GI) symptoms = *Legionella* and other atypical organisms
   g. History of alcoholism and pneumonia = pneumococcus, oral anaerobes, *Klebsiella* and tuberculosis (TB)
   h. Recently on a cruise ship (past 2 weeks) = *Legionella*
   i. Aspiration = anaerobes such as *Prevotella melaninogenica*, *Fusobacterium* species, *Peptostreptococcus* species) and/or oral streptococci (eg, *Streptococcus anginosus*)
3. What imaging modalities are available to help diagnose pneumonia?
   a. Keep in mind that pneumonia is ultimately a clinical diagnosis – not an imaging diagnosis. Chest radiograph (CXR) is most commonly used, although sensitivity of CXR for visualization of pulmonary opacities is low at 43.5% with specificity of 93%[1]. In many studies, computed tomography (CT) imaging is considered the criterion reference to compare other imaging modalities. Thus, CT is considered a reasonable imaging option in patients with an unclear clinical diagnosis with equivocal plain film X-rays.
   b. Point-of-care ultrasound (POCUS) is an emerging imaging modality for early and reliable diagnosis of pneumonia, with a sensitivity of 88.5% and specificity of 91.6%. In the setting of lobar pneumonia, lung tissue on ultrasound appears similar to that of liver tissue. This is due to consolidation of the affected lung; the appearance is referred to as "hepatization" of the lung tissue. It is also possible to see air bronchograms. This refers to the fact that small airways, surrounded by consolidated lung parenchyma, can be visualized. If these air bronchograms are dynamic, with active bubbling through the air bronchograms, it is most specific for pneumonia. Static air bronchograms could be present in the setting of pulmonary consolidation due to atelectasis.
   c. A reasonable approach might be to get a CXR first and consider using CT when a patient's CXR is not clear (presence of other prominent abnormalities such as congestive heart failure (CHF), pleural effusions or other lung pathology) or there are very prominent symptoms or signs despite a fairly unremarkable CXR (ie, hypoxia).
4. Describe the tools to determine whether this patient should be admitted or discharged?

a. The main tools are CURB65 and the Pneumonia Severity Index (PSI) and these are available through several medical applications on portable devices/phones and should be used for all these cases. Learners should know the scores because they provide risk stratification and assist with disposition and level of care decisions.
b. CURB-65 – Patients with 0 or 1 of the below criteria are considered low risk with 1.5% 30-day mortality (outpatient management). Patients with two of the below criteria are classified as moderate risk with 6.8% 30-day mortality (observation vs. inpatient). Any three or more of the below criteria are indicative of severe risk with 14% 30-day mortality (consider ICU). Any 4 or all is highest risk with 27.8% 30-day mortality (consider ICU).
    i. Confusion
    ii. Blood urea nitrogen (BUN) >19
    iii. Respiratory rate >29
    iv. Systolic BP <90mm Hg or diastolic BP <60 mmHg
    v. Age > 64

5. The pneumonia severity index (PSI)/PORT score is a more complicated scoring scale for community-acquired pneumonia that accounts for a patient's age, comorbidities (for example: cancer, CHF, cerebrovascular disease, renal disease, liver disease) and clinical findings (confusion, tachycardia, tachypnea, hypotension, and abnormal temperature). The PSI score may be more accurate in terms of ability to identify patients who would benefit from placement in the ICU. However, it is much more complex, making it more difficult to memorize. Facilitators should encourage students to use online calculators or other applications to determine this score.
6. What are the most likely organisms and appropriate empiric antibiotics for this patient?
    a. In healthy young patients, pneumococcus and mycoplasma should be considered or a viral etiology such as influenza or respiratory syncytial virus (RSV). *Legionella pneumoniae* and *H. influenza* are less likely but possible organisms as well.
    b. This patient would have a very low score (0) by CURB-65 and PSI and qualifies for outpatient management:
        i. Outpatient uncomplicated pneumonia can be treated with: a macrolide (azithromycin or clarithromycin) or tetracycline (doxycycline). Recommended duration is generally 5 days.
    c. If there are other comorbidities such as chronic heart, lung, liver, or renal disease, diabetes mellitus, alcoholism, malignancies, asplenia, immunosuppressive conditions or use of immunosuppressive drugs, use of antimicrobials within the previous three months AND it is safe to send the patient home then consider:
        i. A beta lactam (augmentin, amoxicillin, or cefdinir) PLUS azithromycin or doxycycline.
        ii. Consider monotherapy with a fluoroquinolone (levofloxacin or moxifloxacin).
            1. As with all medications, beware potential side effects. Fluoroquinolones have been associated with risk of tendonitis, QT prolongation, aortic aneurysm/dissection.

Case 2: A 66-year-old female presents from a nursing home for cough and fever for the past two days. She has been there since being discharged from the hospital two weeks ago for a CHF exacerbation. Paramedics note

that her oxygen saturation was 84% on room air. She also has a history of diabetes type 2, hypertension, renal failure on dialysis, and has a chronic wound on her foot that is being treated with topical therapy and seems clean. She has no orthopnea and her last dialysis was today. Her vital signs show a saturation of 100% on four liters of oxygen, blood pressure is 180/100 mmHg, temperature is 101°F, and pulse is 108 beats per minute. Exam reveals clear mental status with some rales and decreased breath sounds in both lower lobes. There is no jugular venous distension or peripheral edema. Her labs reveal a minimally elevated lactate and a white count of 13 x 1000/mm^3 and a hemoglobin of 9.4 g/dL. Sodium and glucose are 134 mEq/L and 222 mg/dL respectively. Chest X-ray shows bilateral lower lobe infiltrates.

Question Prompts:

1. What are the differences between CAP, HAP, and VAP?
    a. Community-acquired pneumonia (CAP) - the infectious causes come from the community.
    b. Hospital-acquired pneumonia (HAP) not acquired while on the ventilator. This is essentially a pneumonia that occurs 48 hours or more after admission and did not appear to be incubating at the time of admission.
    c. Ventilator-associated pneumonia (VAP) is a type of HAP that develops more than 48 to 72 hours after endotracheal intubation.
2. Of those above, what is the diagnosis in this patient and how should this patient be treated?
    a. Hospital-acquired pneumonia (HAP).
    b. CURB 65 = 2, and PSI of 136 or class V.
3. What are risk factors in this patient that should make the clinician broaden the coverage?
    a. This patient has recent admission, nursing home patient, dialysis patient and chronic wound as risks for needing broader coverage.
4. Should HAP patients be treated with CAP coverage or broader coverage?
    a. There seems to be an increasing amount of evidence that HAP patients admitted to the general wards and not to the ICU may be able to be treated with the same regimen as for CAP.
    b. Whereas patients admitted to the ICU or having many risk factors probably should receive broader coverage.
    c. The Infectious Diseases Society of America (IDSA) recommends each hospital regularly disseminate antibiograms and recommendations based on their local resistance patterns.

Case 3: A 55-year-old male renal transplant patient presents after being transferred from an outside hospital with possible bilateral pneumonia. He has a temperature of 99.9°F and was given intravenous (IV) azithromycin and transferred. He has a normal respiratory rate, oxygen saturation of 91%, and he is normotensive. His mental status is normal. He is coughing, feeling weak, and his CXR shows bilateral pneumonia versus pulmonary edema. He also has an acute kidney injury (AKI) with a blood urea nitrogen (BUN) and creatine (Cr) of 80 and 4.0, respectively (baseline Cr is 2.0). Glucose and sodium are normal. A chest CT is also performed without contrast and is interpreted as having evidence of necrotizing pneumonia.

Question Prompts:

1. Calculate CURB-65 and Pneumonia Severity Index (PSI).
    a. CURB-65 was 1 and PSI was 85 or class 3. See notes above for explanation of CURB-65 and PSI.
2. What additional questions should be asked in cases of necrotizing pneumonia and which organism in particular should you suspect?
    a. Since necrotizing pneumonia always requires broader coverage and should make one suspect MRSA, additional MRSA risk factors should be sought. From the Wunderink paper below these include:
        i. Rapid progression of infiltrates and effusions
        ii. Evidence of lung necrosis
        iii. "Dirty dishwasher" appearance of pleural fluid (if obtained via thoracentesis)
        iv. Gross hemoptysis
        v. History of MRSA skin lesions
        vi. Erythematous rash like toxic shock rash
1. Describe the risk factors for *Pseudomonas* pneumonia.
    a. Bronchiectasis
    b. Structural lung disease (chronic bronchitis, COPD, emphysema, interstitial lung disease and restrictive lung disease)
    c. Repeated antibiotic use
    d. Long term steroids during the past 3 months

Case 4: A 90-year-old female presents with mental status changes and seems delirious. The family says she is not making sense, cannot take care of herself, and in the past two days is getting weaker and more confused. She lives by herself but has had a home health nurse since she presented eight months ago with a urinary tract infection (UTI) and confusion. She is full code. She has a remote history of a myocardial infarction and a coronary artery bypass graft (CABG) 15 years ago. Now her temperature is 99°F, systolic blood pressure is 88, pulse oximetry is 90% on 4 liters of oxygen and her heart rate is 90 beats per minute. She only takes an aspirin and a beta blocker. She can follow simple commands but cannot answer orientation questions. Her mouth looks dry and she has poor skin turgor. She has decreased breath sound bilaterally and has mild diffuse abdominal tenderness

Question Prompts:

1. Discuss additional risk factors which can predispose elderly patients to pneumonia and impact prognosis
    a. Reduced mucociliary clearance
    b. Less effective cough reflex
    c. Low grade inflammatory state and diminished immune response
    d. Reduced response by macrophages to microbes
    e. Swallowing disorders with higher risk of aspiration

f. Many comorbid illnesses
g. High risk of cardiovascular disease after the infection
h. Frequently present with mental status changes
i. May not have typical respiratory symptoms
j. Slower GI motility and decreased absorption

2. As time allows, describe the controversies in using the following: Procalcitonin, d-dimer, blood cultures, urine antigens, steroids, viral studies.
    a. Procalcitonin: Although elevated procalcitonin helps predict a bacterial infection over a viral etiology (depending on the cutoff used) it is not clear that ruling out a bacterial infection based on a low procalcitonin can be used to withhold antibiotics. Atypical bacteria may not show an elevation and similarly combinations of bacterial and viral infection may not show a rise in procalcitonin. Also, levels which can be initially low may elevate within several hours. It seems that its greatest value may be in limiting the duration of antibiotic therapy.[5]
    b. D-dimer: May predict a more benign course if low. However, it should be used judiciously because these patients are more likely to have an elevated d-dimer. Providers should decide prior to sending the test whether they plan to follow up with a computed tomography angiography pulmonary embolism protocol study for elevated d-dimer levels. Consider the use of age-adjusted D-dimers.[6]
    c. Blood cultures: The IDSA recommends blood cultures and antigen testing in specific circumstances – ICU admission, cavitary infiltrates, leukopenia, active alcohol abuse, chronic severe liver disease, anatomic or functional asplenia, positive pneumococcal urine antigen, presence of pleural effusion.[7]
    d. Urine antigen testing for pneumococcus and legionella: These are more helpful than sputum and blood cultures with a sensitivity of nearly 40% to 50% compared to 10% of blood cultures. These tests can also be done within minutes.
    e. Steroids: Recent systematic review suggests that CAP patients who require hospitalization may benefit from corticosteroids (reduced mortality, need for mechanical ventilation, and hospital length of stay). However, the precise dose is not yet clear and studies have been small. A general approach can be to consider steroids in patients with septic shock refractory to vasopressors. Steroids are not a routine part of any pneumonia guidelines.[8]
    f. New on the horizon in the diagnosis and treatment of pneumonia:
        i. Use of swabs to detect multiple viral and atypical organisms.
        ii. Antivirals for respiratory viruses are being developed.

Suggested Readings:

Self WH, Courtney DM, McNaughton CD, Wunderink RG, Kline JA. High discordance of chest x-ray and computed tomography for detection of pulmonary opacities in ED patients: implications for diagnosing pneumonia. *Am J Emerg Med*. 2013;31(2):401-405. doi: 10.1016/j.ajem.2012.08.041.

Kalil AC, Metersky ML, Klompas M, et al. Management of adults with hospital-acquired and ventilator-associated Pneumonia: 2016 clinical practice guidelines by the Infectious Diseases Society of America and the American Thoracic Society. *Clin Infect Dis*. 2016;63(5): e61-e111. doi: 10.1093/cid/ciw353.

Wunderink RG, Waterer G. Advances in the causes and management of community acquired pneumonia. *BMJ*. 2017;358: j2471. doi: 10.1136/bmj.j2471.

Additional References:

Schuetz P, Branche A, Mueller B. Low procalcitonin, community acquired pneumonia, and antibiotic therapy – Authors reply. *The Lancet Infectious Diseases*. 2018;18(5):497-498. doi: 10.1016/s1473-3099(18)30226-3.

Adams D, Welch JL, Kline JA. (2014). Clinical utility of an age-adjusted D-dimer in the diagnosis of venous thromboembolism. *Annals of Emergency Medicine,* 64(3):232-234. doi: 10.1016/j.annemergmed.2013.12.018.

Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis*. 2007;44 Suppl 2: S27-72. doi: 10.1086/511159.

Dressler DD. NEJM Journal Watch: Summaries of and commentary on original medical and scientific articles from key medical journals. https://www.jwatch.org/na46012/2018/03/06/steroids-community-acquired-pneumonia-body-evidence-grows. Published February 1, 2018. Accessed July 16, 2019.

Prats M. Acute Dyspnea. Ultrasound GEL. https://www.ultrasoundgel.org/?search=pneumonia. Published May 22, 2017. Accessed June 27, 2019.

Aujesky D, Auble TE, Yearly DM, et al. Prospective comparison of three validated prediction rules for prognosis in community-acquired pneumonia. *Am J Med*. 2005;118(4):384-92. doi: 10.1016/j.amjmed.2005.01.006.

Bartlett JG. Diagnostic approach to patients with CAP. In: Ramirez JA, Bond S, eds. *UpToDate*. Waltham, MA: UpToDate Inc. https://www.uptodate.com/contents/diagnostic-approach-to-community-acquired-pneumonia-in-adults? Updated Jan 23, 2019. Accessed July 16, 2019.

Gottlieb M, Bailitz J. Do corticosteroids provide benefit to patients with community-acquired pneumonia? *Ann Emerg Med*. 2016;67(5):640-642. doi: 10.1016/j.annemergmed.2015.12.008.

Sharp AL, Jones JP, Wu I, et al. CURB-65 performance among admitted and discharged emergency department patients with community acquired pneumonia. *Acad Emerg Med.* 2016;23(4):400-5. doi: 10.1111/acem.12929.

Viasus D, Núñez-Ramos JA, Viloria SA, Carratalà J. Pharmacotherapy for community-acquired pneumonia in the elderly. *Expert Opin Pharmacother.* 2017;18(10):957-964. doi: 10.1080/14656566.2017.1340940.

Yandiola PP, Capelastegui A, Quintana J, et al. Prospective comparison of severity scores for predicting clinically relevant outcomes for patients hospitalized with community-acquired pneumonia. *Chest*. 2009;135(6):1572-9. doi: 10.1378/chest.08-2179.

Balk DS, Lee C, Schafer J, et al. Lung ultrasound compared to chest X-ray for diagnosis of pediatric pneumonia: a meta-analysis. *Pediatr Pulmonol.* 2018;53(8):1130-1139. doi: 10.1002/ppul.24020.

Appendix B:
Asthma and Chronic Obstructive Pulmonary Disease (COPD)

Objectives

By the end of this small group session, the learner will be able to:

1. Discuss the initial approach to management in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations.
2. Describe additional treatments for asthma/COPD exacerbations if the initial approach does not improve respiratory distress.
3. Discuss considerations when utilizing non-invasive ventilation and intubation in patients with asthma and COPD.
4. Describe the management of the intubated patient with acute asthma exacerbation.
5. Discuss the role of asthma action plans and discharge regimens in preventing recurrent emergency department (ED) visits for pediatric acute asthma exacerbations.
6. Discuss the role of antibiotics in patients with COPD exacerbation.

Case Studies

Case 1: A 25-year-old female with a history of severe asthma with many hospitalizations over the last few years presents for shortness of breath. Her respiratory rate (RR) is 24/min, oxygen saturation (O_2sat) is 93% on room air and end tidal carbon dioxide capnography ($ETCO_2$) is 40. On exam, she has diffuse expiratory wheezing.

Question Prompts:

1. What is your next step in management?
    a. This patient presents with an acute asthma exacerbation. Albuterol (beta$_2$ specific agonist) is the preferred initial starting agent. In an ED setting, nebulizer treatment is generally used over an inhaler, but delivery of medication via an inhaler with spacer is also effective (when done correctly, see more discussion in case 2 notes). Albuterol dosage depends on the severity of the asthma exacerbation, ranging from 2.5mg to 10mg.
    b. Levalbuterol (the levo-isomer) is more expensive and has no benefit over albuterol.[2]
    c. Salmeterol xinafaoate and formoterol are long-acting beta antagonists (LABAs), and they should NOT be used for acute exacerbations.
2. Discuss the role of non-invasive positive pressure ventilation (NIPPV) or bilevel positive airway pressure (BiPAP) in this asthmatic patient.

   a. Non-invasive positive-pressure ventilation will improve airflow → improve gas exchange → decrease work of breathing/fatigue → potentially reduce the need for intubation.
   b. Providers should move quickly to non-invasive ventilation if the patient is in respiratory distress and not immediately responsive to albuterol. Nebulized breathing treatments should be continued simultaneously with BiPAP.
3. Other than breathing treatments, what other medication should be given to all moderate/severe asthma exacerbations to improve airway inflammation?
   a. Steroids are indicated in all cases of moderate or severe asthma exacerbation. Generally oral and intravenous (IV) are equivalent, though IV administration may start to have effects slightly sooner than oral.
   b. The addition of ipratropium to albuterol may be additive and helpful in asthma exacerbations. Ipratropium is an anticholinergic agent that blocks the acetylcholine-mediated muscarinic receptor-driven bronchoconstriction, leading to bronchodilation.
4. The patient does not initially respond to BiPAP and 5mg of nebulized albuterol. Describe an algorithm for status asthmaticus.
   a. Continuous nebulized albuterol.
   b. Magnesium: 1-2 grams IV over 10-30 minutes, although some suggest that it is more effective when given quickly. Tintinalli suggests 95 mg of $MgSO_4$ in 4 divided doses, 20 minutes apart. A Cochrane review of the use of magnesium sulfate for treatment of asthma exacerbations in the emergency department describes improvement in peak expiratory flow rates and forced expiratory volume follow IV magnesium administration, but no change in admission rates.[3]
   c. Non-invasive positive-pressure ventilation: Start at inspiratory pressure of 10, expiratory pressure of 5 (10/5) and titrate carefully to watch for improvement. See this source for more information regarding the use of NIPPV in asthma.[4]
   d. Ketamine: Sub-dissociative doses of ketamine may help a patient to tolerate NIPPV and also help with bronchodilation by increasing circulating catecholamines. Also, in patients that are very agitated, fully dissociative ketamine can provide sedation while the team is able to initiate other treatments.
   e. Epinephrine: 0.3-0.5 mg intramuscular (IM). It can be given IV also in a carefully titrated, diluted fashion.
      i. Can mix an epinephrine infusion - take 1mg of epinephrine and add to 1 L of normal saline. Start the infusion at 1ml/min and titrate to effect. Max dose for adults is 20 mcg/min.
      ii. Can mix “push dose” epinephrine - take 1ml (1 mg/10 ml) from code epinephrine ampule and mix in 9ml of a saline flush. Dose is 1ml (0.1 mg) at a time every few minutes.
      iii. Be cautious with mixing push-dose or epinephrine drips, and do not do it if you are not comfortable with them because mathematical errors in mixing will often be 10- or 100-fold and can be dangerous.
   f. Terbutaline acts on $beta_2$ receptors to cause bronchial smooth muscle relaxation. It may also be considered for use in cases of severe asthma exacerbations. The recommended

dose is 0.25 mg administered subcutaneously, and may be re-administered every 20 min for a maximum of 3 doses if necessary. Since they have a very similar mechanism, usually one gives either epinephrine or terbutaline.

g. Heliox may be useful to help facilitate laminar flow in the narrowed airways.[5]

5. Despite everything you have provided above your patient continues to decompensate. What is your next step?
    a. Intubation: When you intubate an asthmatic, be aware that breath stacking on the ventilator can lead to cardiopulmonary arrest. Intubation does not fix the asthma/bronchospasm; it just rests the patient. In asthma, the pressure is delivered to the small airways, not the alveoli, so expect peak airway pressure will be high. But unless there is a problem in the alveoli such as pneumonia or pulmonary edema, the plateau pressure should be normal. The goals of intubation include decreasing work of breathing, improving oxygenation and preventing barotrauma while waiting for the other therapies to work.
        i. Peak inspiratory pressure (P_{IP}) is the highest level of pressure applied to the lungs during inhalation. In mechanical ventilation the number reflects a positive pressure in centimeters of water pressure (cm H_2O). Peak inspiratory pressure increases with any airway resistance. Things that may increase PIP could be increased secretions, bronchospasm, biting down on ventilation tubing, and decreased lung compliance.
        ii. Plateau pressure (P_{PLAT}) is the pressure applied to small airways and alveoli during positive-pressure mechanical ventilation. It is measured during an inspiratory pause on the mechanical ventilator.
        iii. Auto (Intrinsic) positive end-expiratory pressure (PEEP) results from incomplete expiration prior to the initiation of the next breath causes progressive air trapping (hyperinflation). This is called "breath stacking." This accumulation of air increases alveolar pressure at the end of expiration, which is referred to as auto-PEEP. Auto-PEEP = P_{PLAT} – extrinsic PEEP (your vent settings). The risk of this can be decreased by prolonging the expiratory phase on the ventilator.
    b. Ventilator management tips in asthma
        i. Settings – Respiratory rate (RR) 10-12, Tidal volume (TV) 5-7mL/kg, minute ventilation <115mL/kg, inspiratory flow rate of 80L/min, PEEP 5, fraction of inspired oxygen (FiO_2) at 100% and titrate down to keep oxygen saturation >90%.
        ii. Adjust ventilator settings to keep plateau pressure <30 and auto-PEEP <10.
        iii. Keep patient well sedated and potentially paralyzed to avoid fighting the vent.
        iv. Inspiratory flow rate may need to be increased to allow for a prolonged expiratory phase.
        v. Inspiration to expiration ratio (I:E) should be 1:3-1:5. This can first be accomplished by having a relatively low set respiratory rate and allowing for permissive hypercarbia.
        vi. If the patient does code on the ventilator, the first step should be to remove them from the ventilator circuit and allow for full exhalation, potentially actively compressing on the chest to fully exhale the patient.

c. If despite all your efforts, you still can't oxygenate the patient, consider extracorporeal membrane oxygenation (ECMO).

Case 2: A 2-year-old male with a history of eczema and several previous episodes of wheezing associated with colds presents to the ED. He has been previously well but today family noticed increased work of breathing. Vitals: heart rate (HR) 175, RR 45, SaO_2 91% on room air, temperature (T) 98.8°F, blood pressure (BP) 110/80 mmHg. Patient is noted to have tight aeration, global retractions, nasal flaring and expiratory wheezing.

Question Prompts:

1. What is your next step?
    a. Short acting bronchodilators are the mainstay of therapy for acute asthma exacerbation in children.
    b. The addition of ipratropium bromide is efficacious in decreasing hospital admission when used with beta agonists. They do not demonstrate this effect when used alone and are also associated with significant cost as a single agent. The advantage is demonstrated more in patients with moderate to severe exacerbations at presentation.[6]
2. What else can you give to help resolve the impending crisis?
    a. Early steroid administration is associated with decreased hospital admission.[7] This supports many large institutional practices of steroids in triage by standing order sets. There are multiple large studies which show decreased "bounce backs" when patients are discharged on steroids[8] although many of these studies included children and adults without breaking out the pediatric group separately.
    b. Route and specific steroid utilized is a topic of ongoing discussion and debate. In general, oral steroids have similar efficacy to IV steroids. Dexamethasone is a long-acting steroid with a half-life of approximately 50 hours, and has the added benefit of avoiding issues with medication compliance at home.
    c. Magnesium sulfate IV does demonstrate a reduction in hospital admissions particularly in those patients presenting with severe asthma. Nebulized magnesium does not demonstrate this outcome and currently is not recommended.
3. This patient improves dramatically with one nebulized albuterol-ipratropium and oral steroid. When discharging the patient, what is important to confirm with the parents?
    a. Confirming or developing an asthma action plan can help to empower parents/patients to manage their asthma symptoms at home, encourage compliance with recommended interventions, and prevent return visits to the ED.
    b. The plans give patients exact instructions on what medications to take (such as when to start using daily inhaled steroids), how much to take, when to take them, and when to seek help.
    c. These plans generally provide a stepwise approach to asthma management. They should include:
        i. Descriptions of what to do in normal (asymptomatic) circumstances – for example, avoid asthma triggers, use daily controller medications.

ii. Descriptions of what do to during mild, moderate, and severe asthma exacerbations – for example, instructions on how often to use inhalers, when to call one's own doctor, and when to call 911 and/or proceed to the nearest emergency department for help.

Case 2: A 56-year-old patient with a known history of COPD presents with complaints of worsening shortness of breath and dyspnea. He has had sputum production increased from baseline. Vitals: RR 26/min, BP 140/80 mmHg, HR 120/min, T 100.4 °F. Venous blood gas shows pH=7.25, pCO_2 =75.

Question Prompts:

1. What is your next step?
    a. Goals of treatment for a COPD exacerbation are to improve oxygenation, reverse reversible bronchospasm, and treat any underlying exacerbating factors.
        i. Oxygen: To keep oxygen saturation between 88%-92% while avoiding hyperoxia.
        ii. $Beta_2$-agonists: Frequent dosing.
        iii. Anticholinergics: Ipratropium may be as effective as beta agents and should be given along with albuterol.
        iv. Steroids: 60 mg of prednisone orally or 80 mg of methylprednisolone IV. Higher doses have not been shown to more effective.
2. When do you consider NIPPV in patients with COPD exacerbation?
    a. As in adult and pediatric asthma, NIPPV can have significant benefits in those with severe exacerbations.
    b. Non-invasive positive pressure ventilation provides symptomatic improvement → decreases intubation rates → improves short-term mortality rates → shortens hospitalization length.
    c. When patients are receiving NIPPV, maintain continuous cardiac monitoring and perform frequent reassessments.
3. What are the signs of impending respiratory failure?
    a. Uncorrected or worsening hypercapnia → worsening mental status/agitation → reduced respiratory drive → worsening hypoventilation → worsening hypercapnia.
4. Discuss the role of antibiotics in patients with COPD exacerbation.
    a. Antibiotics: If a concomitant respiratory infection is present, antibiotics are indicated. They should be directed at the most likely pathogens—*Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis.*
    b. Generally, macrolide or fluoroquinolone antibiotics are recommended.
        i. See Appendix A: Pneumonia for a discussion of risks of fluoroquinolones.
    c. Also consider antibiotics if there has been a change in sputum production from the patient's baseline with COPD exacerbation symptoms.

Suggested Readings:

Castro-Rodriguez, JA. Rodrigo GJ, Rodriguez Martinez, CE. Principal findings of systematic reviews of acute asthma treatment in childhood. *J Asthma*. 2015:52(10):1028-1045. doi: 10.3109/02770903.2015.1033725.

Additional References:

Jat KR, Khairwa A. Levalbuterol versus albuterol for acute asthma: A systematic review and meta-analysis. Pulmonary Pharmacology & Therapeutics. https://www.sciencedirect.com/science/article/pii/S109455391200171X?via=ihub. Published November 30, 2012. Accessed July 17, 2019.

Rowe BH, Bretzlaff J, Bourdon C, Bota G, Blitz S, Camargo Jr CA. Magnesium sulfate for treating exacerbations of acute asthma in the emergency department. https://www.cochrane.org/CD001490/AIRWAYS_magnesium-sulfate-treating-exacerbations-acute-asthma-emergency-department. Accessed July 17, 2019.

Soroksky A, Klinowski E, Ilgyev E, et al. Noninvasive positive pressure ventilation in acute asthmatic attack. European Respiratory Society. https://err.ersjournals.com/content/19/115/39. Published March 1, 2010. Accessed June 27, 2019.

Reuben AD, Harris AR. Heliox for asthma in the emergency department: a review of the literature. *Emergency Medicine Journal*. https://emj.bmj.com/content/21/2/131. Published March 1, 2004. Accessed July 17, 2019.

Walker G. Nebulized Ipratropium for Asthma Attack. TheNNT. http://www.thennt.com/nnt/nebulized-ipratropium-for-asthma-attack/. Accessed July 17, 2019.

Bhogal SK, Mcgillivray D, Bourbeau J, Benedetti A, Bartlett S, Ducharme FM. Early Administration of Systemic Corticosteroids Reduces Hospital Admission Rates for Children with Moderate and Severe Asthma Exacerbation. *Annals of Emergency Medicine*. 2012;60(1). doi: 10.1016/j.annemergmed.2011.12.027.

Kenyon, CC, Rubin DM, Zorc JJ, Mohamad Z, Faerber JA, Feudther C. Childhood asthma hospital discharge medication fills and risk of subsequent readmission. *J Pediatr*. 2015;166(5):1121-1127. doi: 10.1016/j.jpeds.2014.12.019.

Andrews AL, Russell WS, Titus MO, et al. Quality improvement methods improve inhaled corticosteroid prescribing in the emergency department. *J Asthma*. 2014;51(7)737-742. doi: 10.3109/02770903.2014.911885.

Ducharme, FM, Zemek RL, Chalut D, et al. Written action plan in pediatric emergency room improves asthma prescribing, adherence, and control. *Am J Resp Crit Care Med*. 2011;183(2):195-203. doi: 10.1164/rccm.201001-0115OC.

Koninckx M, Buysse C, de Hooq M. Management of status asthmaticus in children. *Paediatr Respir Rev*. 2013;14(2):78-85. doi: 10.1016/j.prrv.2013.03.003.

Yoos HL, Kitzman H, McMullen A, Sidora K. Symptom perception in childhood asthma: how accurate are children and their parents. *J Asthma*. 2003;40(1):27-39. doi: 10.1081/JAS-120017204.

Appendix C: Pneumothorax

Objectives

By the end of this small group session, learners will be able to:

1. Discuss the epidemiology, risk factors, and pathophysiology of traumatic, primary spontaneous and secondary spontaneous pneumothoraces.
2. Discuss various imaging modalities for the diagnosis of pneumothorax.
3. Describe differences in management of tension, traumatic, and spontaneous pneumothorax.
4. Discuss the difference between primary and secondary spontaneous pneumothorax.
5. List the potential complications of tube thoracostomy.
6. Discuss the appropriate disposition of patients with pneumothorax.

Case Studies

Case 1: A 24-year-old male presents as a trauma alert. The patient was a restrained driver and was T-boned on the driver's side. On arrival the patient has Glascow Coma Scale (GCS) of 15. He complains of severe left-sided chest pain and shortness of breath. His vital signs are blood pressure (BP) 89/50 mmHg, heart rate (HR) 122/minute, respiratory rate (RR) 26/min, oxygen saturation 93% on room air (RA), and temperature (T) 98.6°F. On exam you note marked left chest wall tenderness and decreased breath sounds on the left. The remainder of your exam reveals no additional injuries.

Question Prompts:

1. What is the most likely injury? Describe the pathophysiology that explains the patient's vital sign abnormalities.
    a. This patient is most likely suffering from a tension pneumothorax.
    b. The pathophysiology of traumatic pneumothorax is distinct from spontaneous pneumothorax. Blunt traumatic pneumothorax is caused by air entering the pleural space due to rib fracture with pleural penetration or from alveolar rupture due to sudden and forceful compression of the chest.
    c. When the patient inhales, air enters the pleural space through the injured alveoli or pleura. If the air is not able to exit the pleural space on exhalation the air is trapped in the pleural space. In these cases, with each breath the intrathoracic pressure rises. As the intrathoracic pressure rises it can compress the mediastinum, thereby impairing preload and diastolic filling and ultimately leading to hemodynamic instability. If not treated immediately, it can progress to cardiac arrest.

d. Of note, traumatic pneumothorax is often accompanied by hemothorax. Hypotension and tachycardia may also be due to hemorrhagic shock. The pleural cavity can hold more than four liters of blood!

2. What additional physical exam and findings might you expect to see in this patient given his likely diagnosis? What is the utility of chest radiography (CXR) and/or point of care ultrasound (POCUS) in this case?
    a. Physical exam: In addition to absent ipsilateral breath sounds, patients with tension pneumothorax may demonstrate asymmetry of chest wall movement, chest wall crepitus, chest wall deformities/flail segments with paradoxical chest wall movement, contralateral tracheal deviation and jugular venous distension.
        i. Be aware that 10%-23% of trauma patients with minimal physical exam findings will have significant intra-thoracic injuries!
    b. Chest radiograph: CXR will show a large pneumothorax (with or without rib fractures), contralateral mediastinal deviation, and may show a concomitant hemothorax.
        i. Pneumothorax is identified on CXR by visualizing air between the lung surface and chest wall. This is typically *detected on an upright film* by visualizing air at the apices.
        ii. However, in trauma patients who require spine immobilization upright films are not feasible. On a supine film look for hyperlucent lung bases and deep radiolucent costophrenic sulci (deep sulcus sign), see below.
        iii. *More importantly, a tension pneumothorax should never be diagnosed by CXR.* This is a clinical diagnosis and treatment should never be delayed to obtain diagnostic imaging.

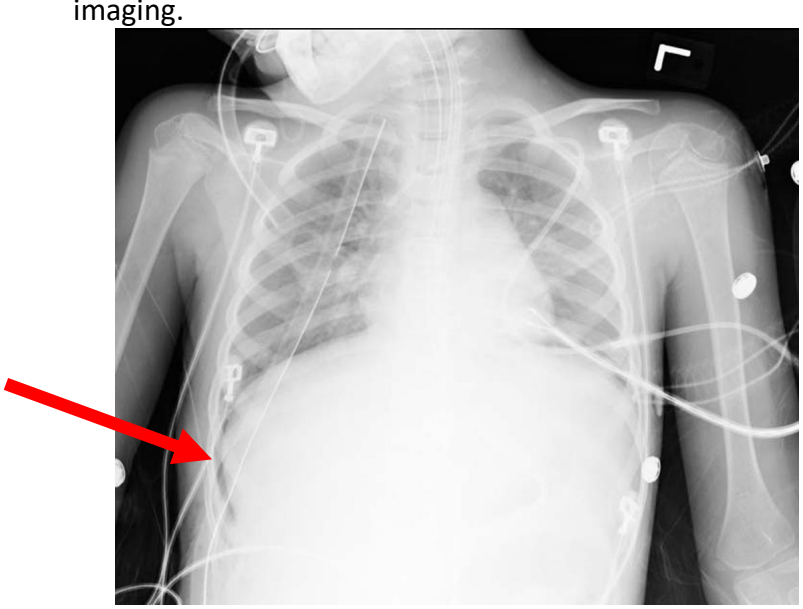

Image source: Radswiki. Pneumothorax: deep sulcus sign. In: Radiopaedia. rID: 11802. https://radiopaedia.org/cases/pneumothorax-deep-sulcus-sign. CC BY-NC-SA 3.0.

    c. POCUS: Ultrasound is 90.9% sensitive and 98.2% specific for detection of pneumothorax. Compare this to CXR which has a sensitivity of 50.2% and specificity 99.4%.[3]

3. What is the appropriate initial resuscitative measure in this patient? Describe the procedure.
    a. Immediate decompression with needle thoracostomy is indicated in this patient.
    b. The classically taught technique is to insert a 2.25 inch 14 or 16-gauge angiocatheter needle perpendicular to the chest wall at the 2nd or 3rd intercostal space, above the rib, in the midclavicular line. Then remove the needle leaving the angiocatheter in place to allow air to escape.
    c. Another technique involves inserting the needle in the anterior-axillary line at the 4th or 5th intercostal space – the similar location for chest tube insertion.
    d. See next questions for trouble shooting and placing the subsequent chest tube.
4. Your attempt at needle decompression is unsuccessful. What are the common pitfalls of this procedure and what are some ways to troubleshoot these issues?
    a. The most common pitfall of the classic needle thoracostomy procedure is that the standard 2.25-inch needle is not long enough to penetrate the pleural cavity. In order to successfully needle decompress obese patients a longer catheter is necessary.
        i. A meta-analysis found that a 2-inch catheter reached the pleural cavity in only 73% of patients, and a 2.52-inch catheter was required for a 95% successful rate.
    b. The chest wall is often actually least thick at the 5th intercostal space at the anterior axillary line. If needle decompression at the midclavicular line is unsuccessful, consider attempting the procedure at the anterior axillary line.
    c. If needle decompression is still unsuccessful in an unstable patient, the next step is to perform emergent finger thoracostomy. Finger thoracostomy is performed identically to the first steps of a tube thoracostomy and can be performed rapidly and requires only a scalpel and hemostat.
5. The needle is finally successfully placed and the patient's vital signs stabilize. What is the most appropriate next course of action?
    a. After needle decompression, patients with traumatic tension pneumothorax require chest tube placement.
    b. Given the high incidence of concomitant hemothorax, pigtail catheters are generally avoided in traumatic pneumothorax.
    c. Traditionally, larger tubes (36F - 40F) were used to help facilitate rapid drainage, drain blood, and prevent air leaks. However, a study in the *Journal of Trauma and Acute Care Surgery* recently compared the impact of small-bore chest tubes (28F - 32F) to large-bore chest tubes (36F - 40F). This study demonstrated no statistical difference in initial volume of blood drained, duration of chest tube placement, development of chest tube-related pneumonia and/or empyema, or persistent hemothorax. There was also no statistical difference in pain reported by patients at site of insertion.[4]
    d. In the polytrauma patient, resume advanced trauma life support (ATLS) protocol after the needle/finger thoracostomy to identify other injuries, and then place the chest tube during the secondary survey prior to computed tomography (CT) imaging.
    e. When appropriate, also consider local anesthesia with lidocaine, lidocaine with epinephrine, or sedation when placing a chest tube because this is a very painful procedure. If procedural

sedation is indicated, ketamine may be an ideal agent because it causes less airway compromise while not causing hypotension or other hemodynamic compromise.

6. What are the potential complications of tube thoracostomy?
    a. Chest tube malposition: Placement within the lung fissure or subcutaneous placement can result in a tube thoracostomy that does not work properly. Intraparenchymal (within the lung tissue) placement can cause major bleeding.
    b. Organ injury: Published injuries include lacerations of the lung, intercostal artery, esophagus, stomach, liver, spleen, diaphragm, pulmonary artery, and atrium as well as right ventricular compression.
    c. Infection: Pneumonia and empyema
    d. Re-expansion pulmonary edema is a potentially fatal complication of tube thoracostomy that can occur after rapid re-expansion of a lung that has been collapsed, due to either pneumothorax or effusion, for at least three days.

Case 2: An 80-year-old male with chronic obstructive pulmonary disease (COPD) on 3 liters home oxygen presents with a chief complaint of shortness of breath. The patient states that he has had worsening dyspnea, wheezing, and productive cough for the last three days. Last night during a particularly forceful coughing fit he developed a sudden onset of left-sided pleuritic chest pain. Vital signs are BP 150/90, HR 90, RR 24, SaO_2 90% on 4L nasal cannula (NC), and T 98.6°F. Physical exam reveals a cachectic male in moderate respiratory distress. There are decreased breath sounds on the left and diffuse wheezing. A portable CXR is shown below:

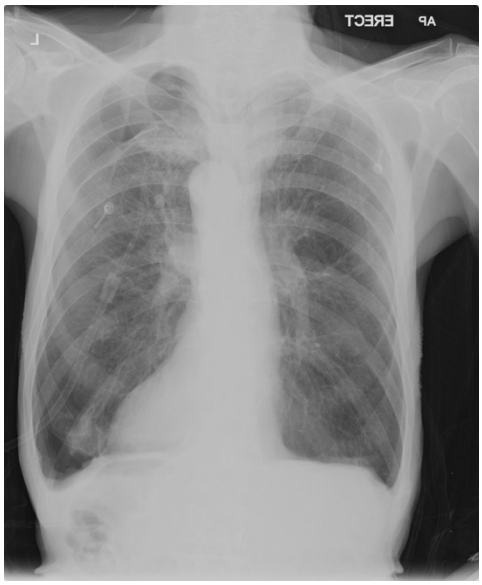

Image source: Knipe H. Spontaneous pneumothorax secondary to COPD. Radiopaedia. rID: 49811. https://radiopaedia.org/cases/spontaneous-pneumothorax-secondary-to-copd. CC BY-NC-SA 3.0.

Question Prompts:

1. What is the diagnosis? What is the pathophysiology of this disease process?

   a. This patient is suffering from a secondary spontaneous pneumothorax (SSP) in the setting of a COPD exacerbation.
   b. A secondary spontaneous pneumothorax is a pneumothorax that occurs as a complication of underlying lung disease. The most common cause of SSP is rupture of lung blebs in patients with COPD. The more severe the COPD the higher the risk of developing SSP.
      1. Other lung pathologies that predispose patients to SSP include cystic fibrosis, lung malignancy (due to endobronchial obstruction with air trapping and tumor necrosis), necrotizing pneumonia (bacterial, *Pneumocystis jirovecii*, tuberculosis), and Marfan's syndrome.
2. How does secondary spontaneous pneumothorax differ from primary spontaneous pneumothorax?
   a. Unlike SSP, primary spontaneous pneumothorax (PSP) occurs without an inciting event and without underlying lung pathology.
   b. Patients with SSP are often more symptomatic presumably because these patients have poor pulmonary reserve due to underlying lung disease.
   c. Treatment options differ between SSP and PSP (see below).
3. What studies can you employ to evaluate a patient for a possible pneumothorax?
   a. An upright CXR is usually the first imaging modality used when working up dyspnea in the emergency department. Visualizing the visceral pleural line that defines the interface of the lung and pleural air makes the diagnosis of pneumothorax. Point of care ultrasound and CT imaging are other imaging modalities which could be used.
   b. The diagnosis of SSP patients with COPD can be difficult for two reasons:
      1. It may be difficult to visualize the visceral pleural line because the emphysematous lung is hyperlucent and there is minimal difference in radiodensity of the lung and air in the pleural space.
      2. It may also be difficult to distinguish a thin walled bulla from the pneumothorax. As a general rule, the pleural line of a pneumothorax is convex whereas the pleural line of a bulla is concave relative to the chest wall.
   c. If the diagnosis of SSP is uncertain on CXR, CT of the chest is the gold standard diagnostic imaging modality and can define the presence, exact location, and size of the pneumothorax. Contrast is generally not necessary for the diagnosis of pneumothorax on CT. However, intravenous contrast is helpful in the evaluation of possible concomitant vascular thoracic injuries in cases of traumatic pneumothorax.
4. What is the most appropriate treatment for this patient?
   a. Supplemental oxygen: Treats hypoxia and helps facilitate absorption of air from the pleural space. In patients with COPD, however, hyperoxia should be avoided.
   b. Pleural drainage: In general, there are three options for treatment of any spontaneous pneumothorax without tension physiology: observation with supplemental oxygen, needle aspiration, and tube thoracostomy. Patients with SSP have underlying pulmonary disease such as COPD which make the likelihood of persistent air leak higher and are at high risk for further expansion of the pneumothorax. Thus, unlike the treatment of stable spontaneous primary pneumothorax (see below), all but the smallest SSP require pleural drainage.

1. Patients who are minimally symptomatic with a very small pneumothorax (<1cm between the visceral and parietal pleura at the level of the hilum on CXR) may be treated with supplemental oxygen and observed for 12 - 24 hours with frequent clinical reassessments and repeat CXR to monitor for expansion of the pneumothorax.
2. For all other SSPs, tube thoracostomy is preferred to needle aspiration due to the high risk of persistent air leak and pneumothorax expansion.

5. What are the benefits and possible risks of placing a pigtail catheter over a large bore tube?
    a. Studies comparing small bore (10F - 14F) and large bore (24F - 28F) chest tubes for drainage of SSP are all retrospective, and thus lower quality evidence. However, the available research seems to indicate that placement of small-bore chest tubes is non-inferior to the placement of large bore tubes. There does not appear to be any difference in rate of successful placement, length of hospital stay, recurrence rate, and complications. Anecdotally, and as one might expect, patients seem to tolerate placement of pigtail catheters better than large bore tubes.
    b. Traditionally, large bore tubes have been used to drain SSPs in patients who are intubated due to concerns that smaller tubes may not be adequate to drain large air volumes experienced during positive pressure ventilation. This is controversial and the decision should be made in conjunction with the surgical/critical care teams that will be assuming care of the patient.
6. You've successfully placed your chest tube and now wonder what to do with the free end of the tube. What are your options? What are the pros and cons of each? Suction or no suction?
    a. The two options are an underwater seal drainage device (ie, Pleur-evac) or a unidirectional flutter valve (ie, Heimlich valve). Both devices contain a valve mechanism that allows air to exit without allowing entry of air from the outside on inhalation. The underwater device has the benefit of allowing the physician to visualize and even quantify air leaks which may help guide further management. These devices are also readily attached to suction when/if needed. The Heimlich valve has the benefit of allowing the patient to be mobile. The decision can be made on a case-by-case basis considering patient preference and the wishes of the admitting team.
    b. Suction should not initially be used for management of tubes placed for SSP due to the increased risk of re-expansion pulmonary edema. Suction may be used if the pneumothorax does not resolve. Suction is applied using a high-volume, low-pressure system with pressures of -10 to -20 cm H_2O.

Case 3: A 20-year-old male with a one pack-per-day smoking history presents with left-sided chest pain. Other than his tobacco use he has no medical problems. He states that he was sitting on his couch watching television when he developed a sudden onset of pleuritic pain over the left side of his chest associated with mild dyspnea. He denies recent trauma or any other obvious precipitating factors. He has never experienced this pain before. He has no cardiac or pulmonary embolism risk factors. His vitals are BP 120/80 mmHg, HR 70/min, RR 14/min, O_2sat 97% on RA, and T 98.6°F. He has diminished breath sounds on the left but otherwise no focal exam findings. The chest radiograph shows a left-sided pneumothorax.

Question Prompts:

1. Compared to the patient in Case 2, how would you classify this patient's pneumothorax? What is the incidence and possible risk factors for this disease process?
    a. Because there is no underlying pulmonary disease, the pneumothorax would be classified as a primary spontaneous pneumothorax. In contrast, the patient in Case 2 has severe underlying COPD making the pneumothorax secondary spontaneous.
    b. The incidence of PSP in the US is 7.4 cases per 100,000 population per year in men and 1.2 cases per 100,000 per population per year in women. The reason for the strong male predominance is unknown. Interestingly, the incidence of PSP in the UK is 5x higher in men and 12.5x higher in women than in the US.
    c. Cigarette smoking is the most significant risk factor for PSP. In a study that reviewed four articles, 91% of patients diagnosed with PSP were cigarette smokers. The incidence of PSP is also directly proportional to the amount of cigarette smoking. Other risk factors include family history, tall stature, thoracic endometriosis, and anorexia nervosa.
2. What are the treatment options for this patient? How do they differ from the patient in Case 2?
    a. The initial treatment options for patients with PSP include observation and supplemental oxygen, needle aspiration, and tube thoracostomy. The choice depends on patient characteristics and clinical circumstances. Unlike SSP, patients with PSP are more often candidates for observation or needle aspiration rather than tube thoracostomy due to a much lower rate of persistent air leak and recurrence.
    b. Small pneumothorax (<2 - 3cm) and minimally symptomatic: These patients are candidates for supplemental oxygen and observation. If after 6 hours of observation their symptoms have not progressed and repeat CXR shows stable or improving pneumothorax, these patients may be discharged with strict return precautions.
    c. Larger pneumothorax (>3cm) or significant symptoms: These patients should undergo needle aspiration. Needle aspiration is performed using a thoracentesis kit. Using CXR or ultrasound to identify the air pocket the catheter is inserted and air is manually withdrawn until no more air can be aspirated. A persistent air leak is presumed if there is still no resistance after aspirating 4L of air. At this time a small-bore chest tube should be placed.
    d. If aspiration is successful you can proceed one of two ways:
        1. The catheter is left in place and a stopcock is attached to the free end and the tube secured to the chest wall. The patient is observed for four hours at which time a chest x ray is obtained. If the lung remains expanded the catheter may be removed and observed for an additional two hours. If a repeat chest x ray shows no recurrence the patient can be discharged home.
        2. The catheter is left in place and attached to a Heimlich valve and the patient is discharged home with 48-hour follow up.
    e. Aspiration is preferred over tube thoracostomy in management of PSP. Aspiration has been shown to have similar outcomes to tube thoracostomy with the benefit of limiting admissions and hospital length of stay.

f. Failure of observation or needle aspiration is an indication for pigtail chest tube placement.

Suggested Readings:

Nicks BA, Manthey D. Pneumothorax. In: Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM. eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide,* 8th ed. New York, NY: McGraw-Hill; 2016. http://accessemergencymedicine.mhmedical.com/content.aspx?bookid=1658&sectionid=109429615. Accessed July 19, 2019.

Valle P, Sullivan M, Richardson H, Bivins B, Tomlanovich M. Sequential treatment of a simple pneumothorax. *Ann Emerg Med.* 1988;17(9):936-942. doi: 10.1016/S0196-0644(88)80676-0.

Additional References:

Alrajhi K, Woo MY, Vaillancourt C. Test characteristics of ultrasonography for the detection of pneumothorax: a systematic review and meta-analysis. *Chest*. 2012;141(3):703-708. doi: 10.1378/chest.11-0131.

Inaba K, Lustenberger T, Recinos G, et al. Does size matter? A prospective analysis of 28 - 32 versus 36 - 40 French chest tube size in trauma. *J Trauma Acute Care Surg*. 2012;72(2):422- 427. doi: 10.1097/TA.0b013e3182452444.

Andrivet P, Djedaini K, Teboul JL, Brochard L, Dreyfuss D. Spontaneous pneumothorax. comparison of thoracic drainage vs immediate or delayed needle aspiration. *Chest*. 1995:108(2):335-339. doi: 10.1378/chest.108.2.335.

Bense L, Eklund G, Wiman LG. Smoking and the increased risk of contracting spontaneous pneumothorax. *Chest*. 1987;92(6):1009-1012. doi: 10.1378/chest.92.6.1009.

Carfarotti S, Dall'Armi V, Cusumano G, et al. Small-bore wire-guided chest drains: safety, tolerability, and effectiveness in pneumothorax, malignant effusions, and pleural empyema. *J Thorac Cardiovasc Surg*. 2011;141(3):683-687. doi: 10.1016/j.jtcvs.2010.08.044.

Carson-Chahhoud KV, Wakai A, van Agteren JE, et al. Simple aspiration versus intercostal tube drainage for primary spontaneous pneumothorax in adults. *Cochrane Database Syst Rev*. 2017;9:CD004479. doi: 10.1002/14651858.CD004479.pub3.

Clemency BM, Tanski CT, Rosenberg M, May PR, Consiglio JD, Lindstrom HA. Sufficient catheter length for pneumothorax needle decompression: a meta-analysis. *Prehosp Disaster Med*. 2015;30(3):249-253. doi: 10.1017/S1049023X15004653.

Devanand A, Koh MS, Ong TH, et al. Simple aspiration versus chest-tube insertion in the management of

primary spontaneous pneumothorax: a systematic review. *Respir Med*. 2004;98(7):579-80. doi: 10.1016/j.rmed.2004.04.006.

Inaba K, Ives C, McClure K, et al. Radiologic evaluation of alternative sites for needle decompression of tension pneumothorax. *Arch Surg*. 2012;147(9):813-818. doi: 10.1001/archsurg.2012.751.

Light RW. Pneumothorax. In: *Pleural Diseases*. 5th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott, Williams and Wilkins; 2013: 306-339.

MacDuff A, Arnold A, Harvery J, BTS Pleural Disease Guideline Group. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. *Thorax*. 2012;65 Suppl 2: ii18-31. doi: 10.1136/thx.2010.136986.

Millo NZ, Plewes C, Rowe BH, Low G. Appropriateness of CT of the chest, abdomen, and pelvis in motorized blunt force trauma patients without signs of significant injury. *AJR Am J Roentgenol*. 2011;197(6):1393-1398. doi: 10.2214/AJR.11.6536.

Radhi S, Alexander T, Ukwu M, Saleh S, Morria A. Outcome of HIV-associated *Pneumocystis* pneumonia in hospitalized patients from 2000 through 2003. *BMC Infect Dis*. 2008; 8:118. doi: 10.1186/1471-2334-8-118.

Tsai WK, Chen W, Lee JC, et al. Pigtail catheters vs large-bore chest tubes for management of secondary spontaneous pneumothoraces in adults. *Am J Emerg Med*. 2006;24(7):795-800. doi: 10.1016/j.ajem.2006.04.006.

Wilkerson RG, Stone MB. Sensitivity of bedside ultrasound and supine anteroposterior chest radiographs for the identification of pneumothorax after blunt trauma. *Acad Emerg Med.* 2010;17(1):11-17. doi: 10.1111/j.1553-2712.2009.00628.x.

Appendix D:
Pulmonary Hypertension

Objectives

By the end of this small group session, learners will be able to:

1. Define pulmonary hypertension (PH).
2. Describe how to diagnose PH.
3. Discuss the most common causes of PH.
4. Describe the symptoms of a PH acute exacerbation.
5. Discuss treatment options, acute and chronic, for PH.

Case Studies

Case 1: A 48-year-old female presents to the emergency department with complaints of exertional dyspnea. She says that the shortness of breath started about eight months ago and has gotten progressively worse when she tries to do physical activity. She denies any chest pain or tightness. She has also had lower extremity swelling that has been getting worse over the last few months. On exam she has normal sounding lungs and 2+ pitting edema in the lower extremities bilaterally.

Question Prompts:

1. What is the differential diagnosis for this patient?
    a. Very broad! A few to consider – congestive heart failure, acute coronary syndrome/angina, PH, liver disease, renal disease, Budd-Chiari (hepatic vein obstruction), malignancy, anemia.
2. What testing is indicated?
    a. Complete blood count (CBC), basic metabolic panel, brain natriuretic peptide, chest radiograph, electrocardiogram (ECG), troponin, cardiac echocardiography.
    b. Echocardiography is the best initial diagnostic test for PH in the ED. Pulmonary artery systolic pressure can be estimated, and decreased right ventricular function, right atrial hypertrophy, and right ventricular hypertrophy can be seen.
3. What defines pulmonary hypertension? How is it diagnosed?
    a. The pulmonary circulation is normally a high flow, low resistance system. Pulmonary hypertension is defined as a mean pulmonary arterial pressure >25 mm Hg at rest or >30 mm Hg during exertion.
    b. The diagnosis of pulmonary hypertension (PH) is often subtle in the early stages and symptoms are often attributed to deconditioning or a coexisting medical condition. As the disease progresses, the symptoms become worse and a patient may present with overt findings of right heart failure. It is often diagnosed when it has already become severe. The average time to diagnosis is two years.

   c. Diagnostic tests may include echocardiogram, chest radiograph (CXR), other imaging studies, and ECG. Ultimately, a right heart catheterization is performed to confirm a diagnosis of PH.
4. What are the symptoms of pulmonary hypertension?
   a. Exertional chest pain – often due to subendocardial hypoperfusion from increased right ventricular wall stress and myocardial oxygen demand. It can also happen if the enlarged pulmonary artery compresses the left main coronary artery.
   b. Exertional syncope – often due to the inability to increase cardiac output during physical activity.
   c. Peripheral edema – due to right ventricular failure and fluid volume expansion.
   d. Anorexia and/or right upper quadrant (RUQ) pain – this is caused by passive hepatic congestion.
5. What are some possible findings on the ECG of a patient with severe PH?
   a. Right axis deviation (most common finding)
   b. R/S ratio >1 in lead V_1
   c. An R/S ratio <1 in leads V_5 and V_6
   d. A qR complex in lead V_1
   e. $S_1Q_3T_3$
   f. Right atrial enlargement in the inferior leads
   g. Incomplete or complete right bundle branch block
6. How is pulmonary hypertension classified and what are the reported causes?
   a. Group 1 – Pulmonary arterial hypertension (idiopathic pulmonary hypertension). Vasoconstriction results from endothelial dysfunction caused by an imbalance between endogenous vasodilators (*eg*, prostacyclin) and vasoconstrictors (*eg*, endothelin-1). In situ thrombi form. Additional problems include microvascular permeability, abnormal hypoxic vasoconstriction, microvascular thrombosis, leading to vascular remodeling.
   b. Group 2 – Due to left heart disease. One of the most common causes of pulmonary hypertension.
   c. Group 3 – Due to chronic lung disease and/or hypoxemia: chronic obstructive pulmonary disease (COPD), interstitial lung disease, obstructive sleep apnea, high altitude.
   d. Group 4 – Chronic thromboembolic pulmonary hypertension (CTEPH), can develop chronically after pulmonary embolism.
   e. Group 5 – Associated with hematologic disorders, systemic disease other than connective tissue disorders, metabolic or miscellaneous disorders, or unclear multifactorial mechanisms. Causes can include lymphatic obstruction, myeloproliferative disorders, sarcoidosis, neurofibromatosis, glycogen storage disease and thyroid disorders.

Case 2: A 53-year-old male presents in severe respiratory distress. He has known pulmonary hypertension and is chronically on an epoprostenol sodium (Flolan) infusion. He had his pump filled a few days ago and is not sure if it is working correctly.

Question Prompts:

1. What is the mechanism that accounts for why abrupt discontinuation of Epoprostenol is problematic?
    a. If continuous epoprostenol (Flolan) infusions are stopped acutely, pulmonary vascular resistance can acutely rise and the weak right ventricle cannot suddenly compensate. This results in acute right ventricular heart failure.
2. What should be done for this patient?
    a. Get the epoprostenol infusion restarted immediately!
3. Describe the common medications used to treat PH.
    a. Prostanoids: epoprostenol [also known as Flolan; intravenous (IV) formulation], treprostinil (aka Romodulin; available in inhalation, oral, subcutaneous, and IV formulations), iloprost (aka Ventavis; inhalation formulation). All are potent vasodilators.
    b. Endothelin Receptor Antagonists: bosentan (aka Tracleer; oral formulation) and ambrisentan (aka Letairis; oral formulation).
    c. Phosphodiesterase type 5 (PDE-5) inhibitors: sildenafil (aka Revatio; oral formulation) and tadalafil (aka Adcirca; oral formulation).

Case 3: A 50-year-old male with severe pulmonary hypertension on epoprostenol infusion presents to the emergency department with hypoxia and hypotension. He does not appear to be clinically volume overloaded. He has been vomiting for the last few days and has noted fever, body aches, and headache. His infusion pump appears to be working.

Question Prompts:

1. Outline a management strategy for this patient.
    a. Provide supplemental oxygen to keep oxygen saturation > 90%. Beware of intubating a patient with PH. Although it may be necessary in some cases, be careful to minimize the increase in intrathoracic pressure with positive pressure ventilation. Elevated intrathoracic pressure will lead to decreased preload and worsening of right heart function.
        i. Use a tidal volume of 6 mL/kg of ideal body weight.
        ii. Use the lowest positive end-expiratory pressure to maintain the oxygen saturation above 90%).
        iii. Avoid hypercapnia to prevent increased pulmonary vascular resistance, pulmonary artery pressure, and right ventricular (RV) strain.
    b. The hypovolemic patient should receive small boluses of fluid but use caution to not cause RV volume overload because it will displace the septum, impairing left ventricular (LV) output. Use boluses of 250-500ml at a time, then reassess for changes in vital signs as well as signs of volume overload.
        i. Dobutamine or milrinone can improve RV output. Avoid doses of dobutamine > 10 mcg/kg/min because it can increase pulmonary vascular resistance.

c. To perfuse the right coronary artery (RCA), the pressure at the aortic root must be higher than the pulmonary arterial pressure. Hypotensive patients can be given norepinephrine to improve perfusion. Avoid high doses to prevent a rise in pulmonary vascular resistance; a Pulmonology expert should be consulted as soon as possible when managing these patients because imbalance in oxygenation, pulmonary and systemic vascular resistance can lead to acute decompensations.
    i. Avoid agents that can produce direct or reflex tachycardia such as dopamine and phenylephrine.
d. Medications can be used to reduce RV afterload.
    i. Prostanoids: epoprostenol, treprostinil, iloprost. All are potent vasodilators.
    ii. Endothelin Receptor Antagonists: bosentan and ambrisentan.
    iii. PDE-5 inhibitors: sildenafil and tadalafil.
e. Nitric oxide (NO) can be useful as a temporizing measure while other therapies are implemented. Be aware that a "rebound effect" can occur with NO when it is abruptly discontinued.
f. Exracorporeal membrane oxygenation (ECMO) can be used as a bridge to starting definitive therapy or as a bridge to transplant.

Suggested Readings:

Greenwood JC, Spangler RM. Management of crashing patients with pulmonary hypertension. *Emerg Med Clin North Am*. 2015;33(3):623-643. doi: 10.1016/j.emc.2015.04.012.

Wilcox SR, Kabrhel C, Channick RN. Pulmonary hypertension and right ventricular failure in emergency medicine. *Ann Emerg Med*. 2015;66(6):619-628. doi: 10.1016/j.annemergmed.2015.07.525.

Additional References:

McLaughlin VV, Archer SL, Badesch DB, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension, a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. *Circulation*. 2009;119(16):2250-2294. doi: 10.1161/CIRCULATIONAHA.109.192230.

New 2014 pulmonary hypertension guidelines released. PulmCCM. https://pulmccm.org/review-articles/new-2014-pulmonary-hypertension-guidelines-released/. Published September 5, 2014. Accessed March 19, 2018.

Sildenafil worked in pulmonary hypertension due to CHF with preserved EF (RCT). PulmCCM. https://pulmccm.org/randomized-controlled-trials/sildenafil-worked-in-pulmonary-hypertension-due-to-chf-with-preserved-ef/. Published December 26, 2011. Accessed March 19, 2018.

Taichman DB, Ornelas J, Chung L, et al. Pharmacologic Therapy for pulmonary arterial hypertension in adults: CHEST guidelines and expert panel report. *Chest*. 2014;146(2):449-475. doi: 10.1378/chest.14-0793.

Appendix E:
Acute Respiratory Distress Syndrome and Inhalants

Objectives

By the end of this small group session, the learners will be able to:

1. Define acute respiratory distress syndrome (ARDS).
2. Name conditions that can be associated with ARDS.
3. Describe ventilator management in patients with ARDS.
4. Define conditions suggesting potential benefit of prone positioning.
5. List indications for extracorporeal membrane oxygenation (ECMO) in severe ARDS.
6. Discuss inhalation injuries and possible treatment modalities

Case Studies

Case 1: 45-year-old female with a past history of diabetes and hypertension presents to the emergency department with a chief complaint of fever and altered mental status. Today, her family noted that she was febrile to 101.3°F, somnolent, and confused. She was intubated upon arrival due to hypoxic respiratory failure. Chest radiograph shows diffuse bilateral infiltrates. Over the next day, her partial pressure of oxygen (PaO_2) decreases to 38 mmHg despite ventilator support with a fraction of inspired oxygen (FiO_2) of 1.0 and positive end-expiratory pressure (PEEP) of 20cm of water.

Question Prompts:

1. What is acute respiratory distress syndrome?
    a. Berlin definition of ARDS
        i. Acute – lung injury within one week of clinical insult.
        ii. Bilateral lung opacities on imaging, not fully explained by effusions, lobar/lung collapse, or nodules.
        iii. Respiratory failure not fully explained by heart failure or volume overload.
        iv. Decreased PaO_2/FiO_2 ratio - indicative of reduced arterial oxygenation from available inhaled gas.
            1. Mild: 200 mmHg < $PaO_2/FiO_2 \leq$ 300 mmHg with PEEP or continuous positive airway pressure (CPAP) $\geq$ 5cm H_2O.
            2. Moderate: 100 mmHg < $PaO_2/FiO_2 \leq$ 200 mmHg with PEEP $\geq$ 5cm H_2O.
            3. Severe: $PaO_2/FiO_2 \leq$ 100 mmHg with PEEP $\geq$ 5cm H_2O
2. What conditions can be associated with ARDS?
    a. Sepsis
    b. Pulmonary contusion/trauma
    c. Aspiration

   d. Upper airway obstruction
   e. Pneumonia
   f. Stem cell transplant
   g. Drug reaction
   h. Burn
   i. Pulmonary embolism
   j. Blood product transfusion (transfusion-related acute lung injury (TRALI))
   k. Amniotic fluid embolism
   l. Cardiopulmonary bypass
   m. Neurogenic pulmonary edema
   n. Pancreatitis
   o. Acute eosinophilic pneumonia
   p. Drug overdose (after naloxone administration)
   q. Bronchiolitis obliterans organizing pneumonia
   r. Drowning
   s. Smoke inhalation
3. Describe ventilator management in patients with ARDS.
   a. Strategies for ventilator management in patients who have ARDS are generally described as lung protective strategies and are aimed at improving lung recruitment while avoiding barotrauma.
   b. Low tidal volumes of 6-8 mL/kg based on predicted body weight are recommended.
   c. Maintain plateau pressure <30 cm H_2O.
   d. Read more about this at ardsnet.org.
      i. There is a "Ventilator Protocol Card" PDF available.[3]
4. What conditions suggest potential benefit of prone positioning?
   a. ARDS with severe hypoxemia.
      i. PaO2:FiO2 < 150 mmHg.
      ii. FiO2 ≥0.6.
      iii. PEEP ≥ 5cm H_2O.
   b. It has been suggested that there is benefit from prone treatment when it is used early and in relatively long sessions.
5. What are the indications for ECMO in severe ARDS?[4]
   a. Severe hypoxemia despite high levels of PEEP (15-20 cm H_20) for at least 6 hours.
   b. Uncompensated hypercapnia with acidemia (pH<7.15).
   c. Excessively high end-inspiratory plateau pressure (>35-45 cm H_2O).
   d. Potentially reversible respiratory failure.
   e. Relative contraindications.
      i. High pressure ventilation (plateau pressure >30 cm H_20) or high oxygen requirements (>0.8) for over seven days.
      ii. Limited vascular access.
      iii. Other medical conditions that limit the likelihood of ultimate survival.
   f. Absolute contraindication.

i. Any condition for which anticoagulation is contraindicated.

Case 2: A 20-year-old college student presents to the emergency department in respiratory distress. A friend who accompanies your patient reports that the patient and his roommates are moving into off-campus housing today. The patient was attempting to clean the bathroom with multiple cleaning products when he began to complain of rhinorrhea, cough, and difficulty breathing. The friend brought the cleaning products to the emergency department – bleach & toilet bowl cleaner. Your patient is unable to provide further history due to cough and increased work of breathing. Pulmonary auscultation reveals bilateral wheezing.

Question Prompts:

1. What is your differential diagnosis?
    a. Inhalation injury such as chlorine gas exposure which resulted from the mixture of bleach, sodium hypochlorite, and hydrochloric acid, commonly found in relatively high concentrations in toilet bowl cleaners.
    b. Asthma exacerbation causing bronchospasm.
    c. Vocal cord dysfunction.
2. Discuss inhalation injuries and possible treatment modalities. What are the types of inhalants?
    a. Inhalation injuries can come in many forms, from injury related from fire (direct thermal injury, carbon monoxide poisoning, cyanide poisoning), intentional inhalant abuse, and unintentional inhalant exposure.
    b. For intentional inhalant abuse, chemical vapors are breathed that produce mind altering effects. Examples include rubber cement, hair spray, fabric protector, chloroform, paint thinner, White Out, whippets, toxic markers, lighter fluid, gasoline, octane booster, nitrous oxide, room deodorizer, and helium.
    c. There are three types of inhalants – solvents, gases, and nitrites. Abuse can cause serious injury to the central nervous system.
    d. Inhalation injury → bronchospasm and irritation of nasal & oral mucosa
        i. Supplemental oxygen as needed to keep oxygen saturation above 90%.
        ii. Nebulized bronchodilators and steroids.
            1. Limited evidence supports initiation of inhaled glucocorticoids with gradual taper.
        iii. Start with airway and breathing: Supplemental oxygen, airway support necessary due to respiratory depression from central nervous system effects of inhaled chemicals.
        iv. Circulation – use advanced cardiac life support (ACLS) protocols for ventricular arrhythmias. Avoid epinephrine or other catecholamines because these can cause or worsen arrhythmias. Consider use of amiodarone or b-adrenergic antagonists (such as labetalol) instead. Consider cardioversion/defibrillation as appropriate in case of unstable arrhythmias.
        v. Other actions:
            1. Place patient on a pulse ox and telemetry monitor.

2. Order electrocardiogram (ECG), complete blood count (CBC), complete metabolic panel (CMP), urinalysis, screen for other drugs of abuse.
3. Chest radiograph.
4. Screen for depression and suicidal intent.

3. What are potential complications of this condition?
    a. Pneumomediastinum secondary to coughing.
    b. Respiratory distress necessitating initiation of non-invasive positive pressure ventilation (NIPPV) or intubation.
    c. Reactive airways dysfunction syndrome (RADS) & irritant induced asthma (IIA).
    d. Acute respiratory distress syndrome.
    e. Secondary pneumonia in setting of lung injury.
    f. For abused inhalants, there are many short and long-term complications. These can include central nervous system disorders, liver damage, hearing loss, kidney damage, bone marrow suppression, and sudden sniffing death.
        i. Sudden sniffing death is described as acute cardiotoxicity thought to be due to increased myocardial sensitization that promotes arrhythmia in the setting of a catecholamine surge.
4. Bonus info: Chlorine gas was used as a chemical weapon during World War I. What is recommended in the setting of a chlorine gas attack/environmental exposure?
    a. Recognize the situation – strong, irritating odor; may be able to detect gas that is yellow-green in color.
    b. Leave the area.
    c. Chlorine gas is heavier than normal environmental "air," so seek higher ground.
    d. Decontamination – remove exposed clothing but avoid pulling contaminated clothing over the head if possible, risking further airway/ocular exposure. Wash in soap & water.

Case 3: A 25-year-old man is brought to the emergency department after spraying spray paint into a bag and inhaling it in an attempt to get high. He is altered and tachycardic.

Question Prompts:

1. What are street (or colloquial) terms for this type of behavior?[5]
    a. Several terms can be used to describe inhalant abuse, generally when associated with an attempt to become high, or intoxicated:
        1. "Sniffing" or "snorting" describes inhaling chemical fumes directly.
        2. "Bagging" involves inhaling chemical fumes after spraying them into a bag
        3. "Huffing" describes inhalation from a cloth which is saturated in a substance then held over one's nose and/or mouth.
2. What are the major risks of this behavior?
    a. Hypoxia, asphyxia, arrhythmia, "sudden sniffing death."
3. Describe the mechanism of "sudden sniffing death" and its management.

a. Sudden sniffing death is believed to be caused by a sudden catecholamine surge which leads to myocardial hyperexcitability, which subsequently leads to arrhythmia, and death.
b. Management is supportive. Consider administration of beta blockers. Respiratory and cardiac status must be monitored carefully. Treatment can be directed at minimizing further catecholamine surge and arrhythmia treatment as indicated.
c. Screen for mental health risks including suicidality and opportunities to intervene on addiction when the patient is medically stabilized.

Suggested Readings:

Inhalant abuse. *Emergency Physicians Monthly*. http://epmonthly.com/article/inhalant-abuse. Published May 13, 2011. Accessed January 24, 2018.

Nickson C. Acute Respiratory Distress Syndrome (ARDS). Life in the Fastlane: https://lifeinthefastlane.com/ccc/acute-respiratory-distress-syndrome-ards/. Accessed January 24, 2018.

Additional References:

ARDSnet NIH NHLBI ARDS Clinical Network Mechanical Ventilation Protocol Summary. NHLBI ARDS Network. http://ardsnet.org/files/ventilator_protocol_2008-07.pdf. Published July 2008. Accessed July 30, 2019.

Extracorporeal Membrane Oxygenation for ARDS in Adults: *NEJM*. https://www.nejm.org/doi/full/10.1056/NEJMct1103720. Accessed July 19, 2019.

Baydala L. Inhalant abuse. *Paediatrics and Child Heath*. 2010;15(7):443-448. doi: 10.1007/springerreference_44310.

Ranieri VM, Rubenfeld GD, Thompson BT, et al, ARDS Definition Task Force. Acute respiratory distress syndrome: The Berlin Definition. *JAMA*. 2012;307(23):2526-2533. doi: 10.1001/jama.2012.5669.

Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. *N Engl J Med*. 2011;365(20):1905-1914. doi: 10.1056/NEJMct1103720.

Centers for Disease Control and Prevention. Facts about Chlorine. Centers for Disease Control and Prevention. https://emergency.cdc.gov/agent/chlorine/basics/facts.asp. Updated April 10, 2013. Accessed January 24, 2018.

Gorguner M, Aslan S, Inandi T, Cakir Z. Reactive airways dysfunction syndrome in housewives due to a bleach-hydrochloric acid mixture. *Inhal Toxicol*. 2004;16(2):87-91. doi: 10.1080/08958370490265004.

Guérin C, Reignier J, Richard JC, et al. Prone positioning in severe acute respiratory distress syndrome. *N Engl J Med*. 2013;368(23):2159-2168. doi: 10.1056/NEJMoa1214103.

Henderson WR, Griesdale DE, Dominelli P. Does prone positioning improve oxygenation and reduce mortality in patients with acute respiratory distress syndrome? *Can Respir J*. 2014;21(4):213-215. doi:10.1155/2014/472136.

Lemière C, Boulet LP, Cartier A. Reactive airways dysfunction syndrome and irritant-induced asthma. In: Barnes PJ, Hollingsworth H, eds. UpToDate, Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/reactive-airways-dysfunction-syndrome-and-irritant-induced-asthma. Updated May 31, 2018. Accessed July 19, 2019.

Przybysz TM, Heffner AC. Early treatment of severe acute respiratory distress syndrome. *Emerg Med Clin North Am*. 2016;34(1):1-14. doi: 10.1016/j.emc.2015.08.001.

Rogers R, Herbert M, Swadron S. Episode 125 C3 Project: Asthma, Pneumothorax and ARDS. EM: RAP. https://www.emrap.org/episode/february2012/c3projectasthma. Published February 2012. Accessed January 24, 2018.

Walker PF, Buehner MF, Wood LA, et al. Diagnosis and management of inhalation injury: an updated review. *Crit Care*. 2015;19:351. doi: 10.1186/s13054-015-1077-4.

Anderson CE, Loomis GA. Recognition and prevention of inhalant abuse. *Am Fam Physician*. 2003;68(5):869-874.

Appendix F:
Septic Pulmonary Emboli (SPE)

Objectives

By the end of this small group session, learners will be able to:

1. Describe the common clinical signs and symptoms in patients with septic pulmonary emboli.
2. List the common sources of septic pulmonary emboli and the most common culprit organisms.
3. Define what patient populations are at high risk of developing septic pulmonary emboli.
4. Discuss the diagnostic imaging modalities of choice.
5. Discuss what antibiotics should be initiated to cover the most common pathogens in patients with septic pulmonary emboli.

Case Studies

Case 1: A 35-year-old female presents with fever, chest pain and cough. She has been sick for a few weeks. She felt warm intermittently during this time but did not take her temperature. She admits to using intravenous heroin for several months. Her chest pain is pleuritic. She has noted occasional wheezing and at times mild shortness of breath (SOB). She has no risk factors for primary cardiac or pulmonary problems. She has no extremity problems and has not been to an emergency department in years. Her temperature is 101°F, heart rate of 120/min, oxygen saturation of 94% on room air. Her exam is remarkable for track marks from her recent injections, a rash and occasional wheezing on exam although good air flow. Chest radiograph (CXR) shows patchy bilateral infiltrates.

A computed tomography (CT) for pulmonary embolism shows infiltrates but also nodules and cavitary lesions with bilateral pleural effusions. Eventual cardiac echocardiogram reveals tricuspid valve vegetations.

Question Prompts:

1. What is the usual clinical presentation of septic pulmonary emboli (SPE) and what is the mortality rate?
    a. Fever, dyspnea, chest pain and cough are the most common symptoms in this order. Hemoptysis may occur if the septic embolic result in pulmonary infarction. Consider the diagnosis in cases where the patient has history of intravenous drug abuse (IVDA), an indwelling line, a history of endocarditis or valvular heart disease including valve replacement. Other sources are soft tissue infections or infection related to thrombus formation. Symptoms are commonly present for 2 to 3 weeks before a diagnosis is made.
    b. Patients may have endocarditis and pleural effusions or empyema.
    c. Consider the diagnosis if there are multiple pulmonary nodules or infiltrates on imaging in the right clinical setting.

d. Mortality ranges from 2% to 10%

2. What are the most common sources?
    a. Intravenous drug abuse (IVDA) is the most common source in most large series of SPE patients, but over the years the proportion of IVDA seems to be decreasing with many sources attributing this decrease to the increased use of clean needles. Intravenous drug abuse commonly involves SPE of the tricuspid valve or much less likely of the pulmonic valve. At a tertiary medical center, catheter related infections and thrombus formation are relatively common sources.
    b. The triad of an active extrapulmonary source of infection, adjacent venous thrombosis and SPE verify an association of SPE and septic thrombophlebitis. The extrapulmonary infection needs treatment before the condition can improve. This could mean draining an abscess or treating a thrombus with anticoagulation or even surgical removal.
    c. Large suppurative infections in muscles or in the liver have also been described as sources of SPE.
    d. Endocarditis can lead to SPE.
        i. Further evaluate the risk of endocarditis in patients for which clinical suspicion is high using the Duke criteria[3,4,5]:
            1. Requires 2 major and 1 minor criterion OR 1 major and 3 minor criteria or 5 minor criteria
        ii. Major criteria:
            1. Two positive blood cultures with microorganisms typical of infective endocarditis
            2. Evidence of endocardial involvement
        iii. Minor criteria:
            1. Predisposition: intravenous drug use or heart condition predisposing patient to endocarditis
            2. Fever
            3. Vascular abnormality – SPE, intracranial hemorrhage, conjunctival hemorrhages, mycotic aneurysm, arterial emboli, Janeway lesions (nonpainful lesions of the palms of hands and/or soles of feet)
            4. Immunologic abnormality – Osler nodules (painful, tender lesions on the hand/feet), Roth spots (red spots with pale center, visualized on retinal examination), glomerulonephritis.
            5. Microbiologic abnormality – positive blood culture that does not meet description of the major criteria
3. What is the pathophysiology of SPE?
    a. Usually infected emboli containing pathogens embolize to pulmonary arteries, leading to focal lung infections and abscess formation.
    b. For septic thrombophlebitis to occur, usually there are extrapulmonary infectious sources leading to extravasation to nearby veins.
    c. Bacteria cause endothelial damage and thrombosis, which leads to propagation of bacteria in fibrin/platelet matrix. This can lead to metastatic spread to lung.

   d. In addition, as discussed later, some bacteria are more likely to precipitate thrombus formation.
4. Discuss the diagnostic work up for a patient with suspected SPE.
   a. Blood cultures, CXR and labs that include inflammatory parameters such as white blood cell count (WBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and lactate are recommended.
   b. The key to the diagnostic work up seems to be the recognition of suggestive CXR findings such as bilateral pulmonary infiltrates and possibly nodular infiltrates with or without effusion(s) and possibly cavitation in the right clinical setting.
   c. Plain radiographs can show ill-defined nodular pulmonary opacities, cavities, abscesses, infarction, and pulmonary gangrene but often only bilateral pulmonary infiltrates are seen on CXR with or without effusions.
   d. Nearly all studies describe case series of patients with CXRs showing multiple or bilateral infiltrates with or without effusions, but the CTs show multiple pulmonary nodules with many having cavitation diagnostic of SPE. Thus, CT is much more sensitive and the test of choice to diagnose SPE.
   e. In one study, CT findings included multiple peripheral nodules ranging from 0.5 to 3.5cm (83%), a feeding vessel sign (67%), cavitation (50%), wedge-shaped peripheral lesions abutting the pleura (50%), air bronchograms within the nodules (28%), and extension of the lesion into the pleural space (39%).[6]
   f. Finally, many of these patients will eventually need a transesophageal echocardiogram to rule out valvular involvement and vegetations.
   g. Although ultrasound has not been well studied in comparison to CXR and CT, the fact that most of these nodular lesions are more peripheral often with cavitation suggests that ultrasound may have a role for these patients.
5. What are the most common organisms? What are the empiric antibiotics for suspected cases of SPE?
   a. In cases of SPE that are due to complications of peripheral vein suppurative thrombophlebitis, *Staphylococcus aureus* [methicillin sensitive (MSSA) and methicillin resistant (MRSA)] are the most common pathogens.
   b. In cases of catheter-related infections with catheter-related septic thrombophlebitis and involvement of the superior vena cava or inferior vena cava, the most likely organisms are *S. aureus* and Enterobacteriaceae. For this reason, standard sepsis protocols designed to include vancomycin and ceftriaxone (or piperacillin-tazobactam) should cover both of these groups of organisms. Candida should also be considered in a patient with leukemia or lymphoma and therefore antifungal therapy may need to be initiated.
   c. In one series where IVDA was the most common risk factor, MSSA and MRSA were the most common organisms cultured with *Fusobacterium*, *Klebsiella* and Candida as the next most common organisms.
   d. In cases of suppurative thrombophlebitis associated with the oropharynx and the throat and tonsils, *Fusobacterium necrophorum* is the most likely organism but other anaerobes have also been cultured. Empiric therapy for jugular vein suppurative thrombophlebitis

should include a beta-lactamase resistant beta-lactam antibiotic, since treatment failure with penicillin and *F. necrophorum* beta-lactamase production has been reported. Acceptable regimens include ampicillin-sulbactam, or piperacillin-tazobactam, or ticarcillin-clavulanate, or monotherapy with a carbapenem once *Fusobacterium* has been confirmed.

Case 2: A 55-year-old male on dialysis presents with fever, minor cough and some shortness of breath. The patient also has a clotted shunt and had a dialysis catheter placed a month ago. The catheter site is slightly red and painful. He was transferred from dialysis after he complained of fever and SOB and was noted to have a temperature of 102°F. Chest radiograph showed very subtle infiltrates bilaterally with a pleural effusion. Computed tomography of the chest showed multiple pulmonary nodules with some showing cavitation.

Question Prompts:

1. How can one diagnose the central line catheter as the source and how should this be treated in this patient?
    a. High resolution CT is the best way to diagnose catheter-related septic thrombosis because a filling defect can be seen. Ultrasound can also be used.
    b. Coverage of *S. aureus* with appropriate antibiotics such as vancomycin as well as Enterobacteriaceae with piperacillin-tazobactam should be considered. Also, the infected catheter should be removed.
    c. Surgical removal of a large focus of infected thrombus should be considered. Anticoagulation could be considered if there is a large infected thrombus.

Case 3: A 25-year-old male presents with a sore throat and high fever for about two to three weeks. He has noticed some swelling on the right side of his neck. He now reports cough and chest pain with persistent fever. He complained that the chest pain was pleuritic, and he was short of breath. His pulse oximetry is 90%, and he is febrile and tachycardic. Chest radiograph shows bilateral infiltrates.

Question Prompts:

1. How would one make the diagnosis in this patient?
    a. In a patient with sore throat, neck pain and these respiratory symptoms, the patient should have a CT of the neck and chest with contrast protocoled to screen the internal jugular and pulmonary vessels for septic pulmonary emboli.
2. What is the pathophysiology unique to this infection?
    a. Septic thrombophlebitis of the internal jugular vein is also known as Lemierre's syndrome.
    b. *Fusobacterium necrophorum* can be cultured from the throat and often is felt to cause a superinfection of the throat. Other organisms include strep and *S. aureus*. These organisms can produce thrombogenic toxins. For example, *S. aureus* produces a heat stable leukocidin that has thrombogenic effects. *S aureus* also produces a specific coagulase that helps coagulate fibrin. Both are more likely to form a thrombus.

c. Because of the penicillinase production by *Fusobacterium*, piperacillin-tazobactam or a carbapenem with or without metronidazole has been recommended.
d. Use of anticoagulants can be considered.

Suggested Readings:

Cook RJ, Ashton RW, Aughenbaugh GL, Ryu JH. Septic pulmonary emboli. Presenting features and clinical course of 14 patients. *Chest.* 2005;128(1):162-166. doi: 10.1378/chest.128.1.162.

Brenes JA, Goswami U, Williams DN. The association of septic thrombophlebitis with septic pulmonary embolism in adults. *Open Respir Med J*. 2012; 6:14-9. doi: 10.2174/1874306401206010014.

Additional References:

Nickson C. Infective endocarditis. Life in the Fast Lane Medical Blog. https://litfl.com/infective-endocarditis/. Published April 24, 2019. Accessed July 30, 2019.

Duke criteria for infective endocarditis. MDCalc. https://www.mdcalc.com/duke-criteria-infective-endocarditis. Accessed July 30, 2019.

Smith C. Infective endocarditis – spoon feed. Journal Feed. https://journalfeed.org/article-a-day/2018/infective-endocarditis-spoon-feed-version. Published July 28, 2018. Accessed July 30, 2019.

Kuhlman JE, Fishman EK, Teigen C. Pulmonary septic emboli: diagnosis with CT. *Radiology*. 1990;174(1):211-231. doi: 10.1148/radiology.174.1.2294550.

Chow AW. Deep neck space infections in adults. In: Durand ML, Sullivan M, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/deep-neck-space-infections-in-adults. Updated June 6, 2019. Accessed August 26, 2019.

Holm K, Bank S, Nielsen H, Kristensen LH, Prag J, Jensen A. The role of Fusobacterium necrophorum in pharyngotonsillitis – a review. *Anaerobe.* 2016:42:89-97. doi: 10.1016/j.anaerobe.2016.09.006.

Johannesen KM, Bodtger U. Lemierre's syndrome: current perspective on diagnosis and management. *Infect Drug Resist.* 2016; 9:221-227. doi: 10.2147/IDR.S95050.

Karpel JP. Overview of pulmonary disease in people who inject drugs. In: King TE Jr, Hollingsworth H, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/overview-of-pulmonary-disease-in-people-who-inject-drugs. Updated August 5, 2019. Accessed August 26, 2019.

Spelman, D. Suppurative (septic) thrombophlebitis. In: Sexton DJ, Sullivan M, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/suppurative-septic-thrombophlebitis. Updated Apr 4, 2018. Accessed July 23, 2019.

Stubington TJ, James P. Lemeirre's syndrome: a pain in the neck with far-reaching consequences. *BMJ Case Rep.* 2018;1-4 pii: bcr-2017-222723. doi: 10.1136/bcr-2017-222723.

Wong KS, Lin TY, Huang YC, Hsia SH, Yang PH, Chu SM. Clinical and radiographic spectrum of septic pulmonary embolism. *Arch Dis Child.* 2002;87(4):312-315. doi: 10.1136/adc.87.4.312.

Ye R, Zhao L, Wang C, Wu X, Yan H. Clinical characteristics of septic pulmonary embolism in adults: a systematic review. *Respir Med.* 2014; 08(1):1-8. doi: 10.1016/j.rmed.2013.10.012.
Pickens A, Koyfman A, Long B, Singh M. EM in 5: Lemierre's Disease. *EmDocs.* https://emin5.com/2013/10/26/lemierres-disease/. Published October 26, 2013. Accessed October 16, 2018.

Appendix G:
Pleural Effusions and Mediastinum Disorders

Objectives

By the end of this small group session, learners will be able to:

1. Discuss a differential diagnosis for pleural effusions.
2. List criteria used to distinguish an exudative vs transudative pleural effusion.
3. Discuss indications and potential complications for emergency department (ED) thoracentesis for pleural effusions.
4. Discuss management of massive hemothorax.
5. Discuss causes and treatment for pneumomediastinum.
6. Discuss workup and management of esophageal perforation.

Case Studies

Case 1: A 55-year-old female with a history of ovarian cancer presents to the emergency department for dyspnea. She reports her shortness of breath (SOB) has been progressive over the last several weeks. It is worse when she lays flat in bed. A chest radiograph (CXR) shows an opacity overlying the right lung base extending superiorly; it involves about two-thirds of the area of the right lung and a meniscus is present at the superior margin.

Question Prompts:

1. What is the differential diagnosis for pleural effusions?
    a. Intrathoracic: congestive heart failure, parapneumonic effusion, malignant pleural effusion, pulmonary embolism, viral disease, post coronary artery bypass, disease, tuberculosis, mesothelioma, empyema, hemothorax.
    b. Extrathoracic: hepatic failure, renal failure, ovarian carcinoma, pancreatitis, subphrenic abscess, gastrointestinal disease.
2. How does one make the diagnosis of a pleural effusion?
    a. Recovered fluid should be sent for cell counts, protein, lactate dehydrogenase (LDH), glucose, and pH. Pleural fluid cytology should be considered if undiagnosed malignancy is a consideration.
    b. Light's Criteria is the most popular test to evaluate pleural fluid. If one or more criterion is met, the fluid is defined as exudate: [3]
        i. fluid/serum protein > 0.5
        ii. fluid/serum lactate dehydrogenase (LDH) > 0.6
        iii. fluid LDH > ⅔ upper limits of normal serum LDH
    c. Two-test rule. If both are positive, then more likely exudative.

i. pleural fluid cholesterol > 45mg/dl
ii. pleural fluid LDH > 0.45X the upper limit of normal serum LDH

d. Three-test rule. If all are positive, then more likely exudative.
i. pleural fluid protein > 2.9g/dl
ii. pleural fluid cholesterol > 45mg/dl
iii. pleural fluid LDH > 0.45 times the upper limit of the normal serum LDH

e. The benefit of the two and three test rules is that they do not require a venipuncture or simultaneous blood draw.

3. Discuss indications and potential complications for ED thoracentesis for pleural effusions.
a. Indications of ED thoracentesis:
i. Significant pleural effusion with respiratory compromise (causing hypoxia, increased in work of breathing)

b. Potential complications:
i. Postprocedural pneumothorax
ii. Intercostal artery injury
iii. Lung injury, liver injury, peritoneal penetration
iv. Infection, bleeding, pain
1. Re-expansion pulmonary edema
a. Risk factors include:
b. Pulmonary collapse for >1 week
c. Rapid removal of pleural fluid (removal of 1-2 L pleural fluid every 2 hours should not be exceeded)[4]

Case 2: A 40-year-old male presents as a trauma alert due to motor vehicle vs tree collision. Estimated speed 65 mph. The patient was an unrestrained driver. Paramedics report the steering wheel and column appeared bent. The patient was speaking to paramedics, complaining of chest pain, just prior to ED arrival, but now appears more somnolent. He is unable to provide further history. He is intubated for airway protection in the setting of altered mental status. A left-sided chest tube is placed due to inability to auscultate breath sounds on that side. Over 1200 mL blood immediately drains from the chest tube.

Question Prompts:

1. Review the initial steps which were used in the management of this patient. After blunt trauma, the patient became more somnolent, then unresponsive. What is your differential for what may be causing this change in mental status?
a. Intracranial hemorrhage, skull fracture, hypoxia, internal vs external hemorrhage (including hemorrhagic shock), arrhythmia, acute coronary syndrome, substance use, underlying infectious process, electrolyte derangement (hypoglycemia or other), encephalopathy.
b. While the differential diagnosis for what caused this change in mental status is quite broad, priority should be given to protecting the patient's airway, and continuing to work through airway, breathing and circulation (ABCs) in order to stabilize the patient.
2. What is your differential diagnosis regarding the patient's unilateral absent breath sounds?

   a. Pneumothorax, tension pneumothorax, hemothorax.
   b. In an intubated patient, also consider whether this could be caused by a mainstem endotracheal tube (ETT), meaning the ETT is too deep, and is positioned in the left or right mainstem bronchus, therefore making it difficult to hear breath sounds on the contralateral side of the chest.
3. What defines a massive hemothorax?[5]
   a. A massive hemothorax is defined by the rapid accumulation of greater than 1000–1500 ml of blood or one-third or more of the patient's blood volume in the chest cavity.
   b. Most commonly, a massive hemothorax is secondary to penetrating injury disrupting pulmonary or systemic blood vessels.
   c. In hemothorax associated with great vessel injury, 50% die immediately, 25% live 5–10 minutes, and 25% live 30 minutes or longer. Respiratory insufficiency is dependent on how much blood is lost. In massive injury, the affected lung is collapsed producing a right to left shunt. The loss of blood also leads to circulatory compromise
4. How do you manage a massive hemothorax?
   a. Indications for thoracotomy in the setting of hemothorax: immediate loss of 1,500 mL or more of blood accumulating in chest tube, or persistent bleeding into chest tube of 150-200 mL/hour for two hours or more.
   b. While mobilizing the operating room (OR), initiate massive transfusion protocol. If available at your institution, can consider auto-transfusion.

Case 3: A rapid response alert is called after development of hypoxia and agitation in a 37-year- old male who had been undergoing upper endoscopy for presumed impacted food bolus. The team caring for this patient describe the procedure as being difficult and report that the patient's oxygen saturation dropped during attempts to push the food bolus past the gastroesophageal junction. The oxygen saturation is in the mid-80s on room air. You are unable to place the patient on supplemental oxygen due to agitation. The patient is clutching his chest.

Question Prompts:

1. What is your differential diagnosis for this case?
   a. Iatrogenic esophageal rupture, pneumomediastinum, pneumothorax, tracheobronchial rupture, acute coronary syndrome, anaphylaxis/reaction to medications given during endoscopy.
2. What are causes and treatment for pneumomediastinum?
   a. Causes: esophageal perforation that is intraluminal (Boerhaave, foreign body ingestion, food impaction) or extraluminal (neck trauma), tracheobronchial injury, iatrogenic (surgery, endoscope/bronchoscope, forceful intubation, barotrauma), gas-forming infective organisms, tumors, aortic pathology.
   b. The treatment of pneumomediastinum is always directed at the underlying disorder whether it be asthma, trauma, or drug abuse.

i. Esophageal rupture (Boerhaave's syndrome) is a potentially fatal disorder without prompt thoracotomy, esophageal repair, and wide mediastinal drainage. Surgery should be consulted emergently.
ii. Tracheobronchial rupture must be recognized quickly because continued attempts to ventilate the lungs may force additional air into the mediastinum with resultant compression and displacement of other mediastinal structures.
iii. Give antibiotics if concerned about pneumomediastinitis. Cover gastrointestinal flora, resistant organisms and *Pseudomonas*. Consider use of piperacillin/tazobactam or ciprofloxacin and metronidazole.
iv. Management of pneumomediastinum which develops as a result of an asthma exacerbation is different. You should treat the patient's asthma exacerbation (see appendix B) and the pneumomediastinum will self-resolve with time. No specific treatment is needed for asthma-related pneumomediastinum.

3. What is the workup and management of esophageal perforation?
    a. Chest X-ray has the following common findings:
        i. Pneumothorax
        ii. Widened mediastinum
        iii. Left sided pleural effusion
        iv. Mediastinal air with or without subcutaneous emphysema
    b. Esophagram using water-soluble Gastrografin is preferred because it is less likely to cause pulmonary irritation/inflammation as compared to barium. If chest X-ray is equivocal, consider computed tomography of the chest.
    c. If patient unstable, proceed to rapid resuscitation, including intubation if needed for airway stabilization. Start broad spectrum intravenous antibiotics and surgical consultation early.

Suggested Readings:

Eldridge L. What is the mediastinum and three main regions? Contents and abnormalities of the mediastinum. VeryWell.com. https://www.verywell.com/mediastinum-definition-anatomy-and-conditions-2249125. Updated December 4, 2017. Accessed January 16, 2018.

Loyola University of Chicago Stritch School of Medicine. Mediastinum. MedDean.luc.edu. http://www.meddean.luc.edu/lumen/MedEd/Radio/curriculum/Pulmonary/mediastinum_teach.htm. Accessed January 16, 2018.

Additional References:

PACCM MICU Education. Pleural disease diagnostics. https://paccm.pitt.edu/MICU/PACCM_Fellows_Ed_2017/handouts/Pleural Disease Diagnostics.pdf. Published 2015. Accessed July 23, 2019.

Petiot A, Tawk S, Ghaye B. Re-expansion pulmonary oedema. *Lancet*. 2018:392(10146):507. doi: 10.1016/S0140-6736(18)31722-7.

Mowery NT, Gunter OL, Collier BR, et al. Practice management guidelines for management of hemothorax and occult pneumothorax. *J Trauma*. 2011;70(2):510-518. doi: 10.1097/TA.0b013e31820b5c31.

Heffner JE. Diagnostic evaluation of pleural effusion in adults: Initial testing. In: Braddus VC, Finlay G, eds. UpToDate. Waltham, MA: UpToDate, Inc. https://www.uptodate.com/contents/diagnostic-evaluation-of-a-pleural-effusion-in-adults-initial-testing. Updated Apr 2, 2018. Accessed July 23, 2019.

Sarko J, Stapczynski JS. Respiratory distress. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 8th ed*. New York, NY: McGraw Hill; 2016:427-435.

AnatomyZone. Mediastinum - anatomy tutorial. YouTube. https://www.youtube.com/watch?v=Io4S7HmnNqE. Published November 14, 2011. Accessed January 16, 2018.

AnatomyZone. Muscles of the thoracic wall - 3D anatomy tutorial. YouTube. https://www.youtube.com/watch?v=mVLXqICrsdo. Published February 2, 2013. Accessed January 16, 2018.

Boylan AM, Broaddus CV. Pleural disease. In: Schraufnagel DE. *Breathing in America: Diseases, Progress, and Hope.* United States of America. American Thoracic Society; 2010:145-154.

Cadogan M. CXR interpretation. Life in the Fast Lane. https://lifeinthefastlane.com/investigations/cxr-interpretation/. Published August 29, 2017. Accessed January 16, 2018.

Casas A, Pavía J, Maldonado D. Respiratory muscle disorders in chest wall diseases. *Arch Bronconeumol*. 2003;39(8):361-366. doi: 10.1016/S0300-2896(03)75404-0.

Ellis SM, Flower C. Pleural disease. In: Ostensen H, Pettersson H, eds. *The WHO Manual of Diagnostic Imaging: Radiographic Anatomy and Interpretation of the Chest and the Pulmonary System.* Geneva: World Health Organization; 2006:105-118.

Jeung MY, Gangi A, Gasser B, et al. Imaging of chest wall disorders. *Radiographics.* 1999;19(3):617-37. doi: 10.1148/radiographics.19.3.g99ma02617.

Maskell N, Rahman N. Pleural disease. The Lancet Respiratory Medicine. http://www.thelancet.com/series/pleural-disease. Published July 7, 2015. Accessed January 16, 2018.

Mayo P. Bedside pleural ultrasonography: equipment, technique, and the identification of pleural effusion and pneumothorax. In: Broaddus VC, Finlay G, eds. UpToDate. Waltham, MA: UpToDate, Inc.

https://www.uptodate.com/contents/bedside-pleural-ultrasonography-equipment-technique-and-the-identification-of-pleural-effusion-and-pneumothorax. Updated Oct 26, 2018. Accessed July 23, 2019.

Nickson C. Trauma! Initial assessment and management. Life in the Fast Lane. https://litfl.com/trauma-initial-assessment-and-management/. Published February 28, 2019. Accessed July 30, 2019.

Pleural disease. Life in the Fast Lane. https://lifeinthefastlane.com/wp-content/uploads/2009/11/pleural-disease.pdf. Published November 2009. Accessed January 16, 2018.

Whitaker R, White J. Essential surface and related anatomy for clinical practice. LifeintheFastLane.com. https://lifeinthefastlane.com/wp-content/uploads/2010/10/surfaceanatomy.pdf. Published October 30, 2009. Accessed January 16, 2018.

Small Group Evaluation

Date:	**Appendix A: Pneumonia**
Presenters:	
Educational Objectives: 1. Define pneumonia and its diagnosis. 2. Understand the difference between community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP). 3. Identify common pathogens and unique features that may help with predicting a specific pathogen. 4. Discuss the risk factors for methicillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas*. 5. Describe why mortality is higher in elderly patients with pneumonia.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix B: Asthma and COPD**
Presenters:	
Educational Objectives: 1. Discuss the initial approach to management in patients with acute asthma and chronic obstructive pulmonary disease (COPD) exacerbations. 2. Describe additional treatments for asthma/COPD exacerbations if the initial approach does not improve respiratory distress. 3. Discuss considerations when utilizing non-invasive ventilation and intubation in patients with asthma and COPD. 4. Describe the management of the intubated patient with acute asthma exacerbation. 5. Discuss the role of asthma action plans and discharge regimens in preventing recurrent emergency department (ED) visits for pediatric acute asthma exacerbations. 6. Discuss the role of antibiotics in patients with COPD exacerbation.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix C: Pneumothorax**
Presenters:	
Educational Objectives: 1. Discuss the epidemiology, risk factors, and pathophysiology of traumatic, primary spontaneous and secondary spontaneous pneumothoraces. 2. Discuss various imaging modalities for the diagnosis of pneumothorax. 3. Describe differences in management of tension, traumatic, and spontaneous pneumothorax. 4. Discuss the difference between primary and secondary spontaneous pneumothorax. 5. List the potential complications of tube thoracostomy. 6. Discuss the appropriate disposition of patients with pneumothorax.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix D: Pulmonary Hypertension**
Presenters:	
Educational Objectives: 1. Define pulmonary hypertension (PH). 2. Describe how to diagnose PH. 3. Discuss the most common causes of PH. 4. Describe the symptoms of a PH acute exacerbation. 5. Discuss treatment options, acute and chronic, for PH.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix E: ARDS and Inhalants**
Presenters:	
Educational Objectives: 1. Define acute respiratory distress syndrome (ARDS). 2. Name conditions that can be associated with ARDS. 3. Describe ventilator management in patients with ARDS. 4. Define conditions suggesting potential benefit of prone positioning. 5. List indications for extracorporeal membrane oxygenation (ECMO) in severe ARDS. 6. Discuss inhalation injuries and possible treatment modalities.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix F: Septic Pulmonary Emboli (SPE)**
Presenters:	
Educational Objectives: 1. Describe the common clinical signs and symptoms in patients with septic pulmonary emboli. 2. List the common sources of septic pulmonary emboli and the most common culprit organisms. 3. Define what patient populations are at high risk of developing septic pulmonary emboli. 4. Discuss the diagnostic imaging modalities of choice. 5. Discuss what antibiotics should be initiated to cover the most common pathogens in patients with septic pulmonary emboli.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

Small Group Evaluation

Date:	**Appendix G: Pleural Effusions and Mediastinum Disorders**
Presenters:	
Educational Objectives: 1. Discuss a differential diagnosis for pleural effusions. 2. List criteria used to distinguish an exudative vs transudative pleural effusion. 3. Discuss indications and potential complications for emergency department (ED) thoracentesis for pleural effusions. 4. Discuss management of massive hemothorax. 5. Discuss causes and treatment for pneumomediastinum. 6. Discuss workup and management of esophageal perforation.	

Evaluator: (circle) **Faculty** **Resident** **Student** **Other**

DIRECTIONS: Circle the number that reflects each evaluation statement. Please use the comment section to provide feedback on evaluation statements rated 3 or below or for additional comments related to the statements.

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

EVALUATION STATEMENTS:

1. The moderator demonstrated adequate knowledge of subject.
   5 4 3 2 1
2. The moderator's facilitation of the conference facilitated my learning.
   5 4 3 2 1
3. The overall discussion was relevant to the stated topic(s).
   5 4 3 2 1
4. The discussion teaching methods (slides, handouts, videos, etc.) were effective.
   5 4 3 2 1
5. List one thing you learned from this discussion.

6. What practice problems are you experiencing that you would like addressed at future presentations?

7. Comments, (**required**):

8. Was there any evidence of commercial bias/influence in the program content?
   ☐ Yes ☐ No

Evaluator: Faculty/Resident (Name Required) _____ _____
Print Name Signature

Completion of this form, including the comments, is mandatory in order to receive credit for attending this conference. Once your attendance has been confirmed, your name is separated from the evaluation form. The presenter will only see the aggregation of the evaluation forms and will not see the individual forms.

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