Academic Half Day: Sepsis

Facilitator Guide

1:05-1:20 Theory Burst
1:20-2:05 Case
2:05-2:15 Break
2:15-2:25 Questions for the expert
2:25-2:45 Group Lit Review Part 2
2:45-3:20 Case
3:20-3:30 Questions for the expert

While you are waiting for half-day to begin, please fill out the chart below with appropriate arrows (up, down, or equivocal):

	Preload	Pump	Afterload
	PCWP	CO	SVR
Hemorrhagic	↓	↓	↑
Cardiogenic	↑	↓	↑
Septic	↓	↑	↓

Before starting the case, review the chart above and discuss the underlying physiology.

Learning point: Can ask about sepsis-related decrease in cardiac output (i.e. cardiomyopathy), which portends a poorer prognosis. The presence of sepsis-related cardiomyopathy has a higher mortality rate (≈70-90%) than sepsis with preserved cardiac function (≈20%) (Citation: *Circulation*). Mechanisms for cardiovascular dysfunction are global ischemia, cytokines, and nitric oxide, with many unknown mediators. (Citation: *Chest*, 2019).

CASE: You are seeing a patient previously admitted to the floor

78 y.o. male with a PMH significant for HTN, HLD (on atorvastatin), HFrEF (LVEF of 40-45%, on metoprolol and losartan), type 2 DM (on metformin only), obesity, anemia of chronic disease, metastatic prostate cancer (well controlled with androgen deprivation therapy), and dementia who was admitted 24 hours ago due to failure to thrive. A nurse calls you to bedside to assess altered mental status and abnormal vitals (listed below). Prior to admission, the patient lived with his wife, who helped with ADLs. His wife is at bedside now and reports that for the past several days he has had a productive cough with yellow sputum. He now demonstrates some signs of shortness of breath. He also endorses chills and mild nausea. Denies headache, photophobia, neck stiffness, chest pain, abdominal pain, vomiting, diarrhea, dysuria, hematuria, joint swelling, or skin rashes/lesions. He has not required hospitalization or IV antibiotics within the past year.

Bedside evaluation reveals the following:

Vitals: Temp = 96.5 F, BP = 89/50 mmHg (MAP = 63 mmHg), HR = 98/min, RR 26/min, and SpO2 92% on 2 L of O2.

Exam: Patient is lethargic. He is oriented to person, but not place or time (previously oriented to person and place). He is able to answer simple questions and follow one-step commands. No focal neurologic deficit is appreciated. Mucus membranes are dry. Auscultation reveals borderline tachycardia with no murmurs/rubs/gallops, crackles in the left lung base, and decreased bowel sounds. Abdomen is soft, non-distended, and non-tender with no masses or organomegaly. Skin is warm, flushed, and moist. No rashes or wounds are noted.

1. Are you worried about sepsis in this patient? Why?

a. **Review the definition of sepsis and septic shock**

i. Sepsis

1. Life-threatening organ dysfunction caused by a dysregulated host response to infection (2016 SCCM/ESICM task force)
2. Organ dysfunction is defined as an increase of two or more points in the SOFA score
    a. **Specifics of SOFA score will be discussed below, but for reference:**
        i. Respiratory: < PaO2/FiO2
        ii. Hematology: < platelet count
        iii. Liver: > serum bilirubin
        iv. Renal: > serum creatinine (or < urine output)
        v. Brain: < Glasgow coma scale
        vi. Cardiovascular: hypotension/vasopressors
3. Pathogenesis: severe endothelial dysfunction leading to vessel dilation and a compromised barrier between the intravascular and extravascular spaces
4. NOTE: sepsis is part of a continuum (ranging from infection/bacteremia to sepsis and septic shock, ultimately leading to MODS and potentially death)

ii. Septic shock

1. Type of vasodilatory or distributive shock
2. Physiologic definition: circulatory, cellular, or metabolic abnormalities that are associated with greater risk of mortality than sepsis alone
3. Clinical definition: patients who fulfill the criteria for sepsis and require vasopressors to maintain a MAP ≥ 65 mmHg and a lactate > 2 mmol/L (despite adequate fluid resuscitation)
4. NOTE: with these criteria, hospital mortality exceeds 40% (PMID: 26903338)

b. **What factors increase this patient's risk for sepsis?**

i. Community acquired PNA

1. Severe sepsis (a now retired term that was previously defined as sepsis-related hypoperfusion or organ dysfunction) develop in approximately 48% of hospitalized patients with CAP (PMID: 16608946)

ii. Advanced age (≥ 65 years old)
    1. Also an independent predictor of mortality in the setting of sepsis
iii. Cancer
    1. Cancer of all types increases the risk of developing sepsis almost 10-fold (PMID: 15469571)
iv. Diabetes
v. Obesity
vi. Other risk factors:
    1. ICU admission
        a. Approximately 50% of ICU patients have a nosocomial infection (PMID: 7637145)
    2. Bacteremia
        a. One study suggested that 95% of positive blood cultures were associated with sepsis (PMID: 8759492)
    3. Previous hospitalization
        a. Though to induce alteration in the human microbiome
    4. Immunosuppression
    5. Genetic factors

c. **Review tools for sepsis identification and risk-stratification**
    i. 2021 Surviving Sepsis Guidelines recommend **Against using a single** sepsis screening tool like qSOFA or SIRS—Ultimately, diagnosis still requires a clinical assessment and evaluation, while these screening tools can help as a **component** of the clinical evaluation.
        1. Interesting side note: despite this, the SOFA score is still part of the *definition* of sepsis mentioned above.
    ii. **SOFA (Sequential Organic Failure Assessment)**
        1. Criteria (based on surrogate markers mentioned above)
            a. Glasgow coma scale
            b. PaO2/FiO2
            c. MAP (or administration of vasopressors)
            d. Platelet count
            e. Bilirubin
            f. Creatinine
        2. Calculator (link):
    iii. **qSOFA (Quick SOFA)**
        1. Criteria (can be obtained on initial exam)
            a. RR > 22/min
            b. Altered mental status
            c. SBP ≤ 100 mmHg

2. Interpretation

qSOFA Score	Risk group
0-1	Not high risk for in-hospital mortality
2-3	High risk for in-hospital mortality (3- to 14-fold increase)

3. NOTE: Mixed evidence regarding validity. One prospective cohort study in Europe showing similar efficacy to the complete SOFA score, with regards to predicting in-hospital mortality. It also demonstrated superior accuracy when compared to SIRS or severe sepsis criteria (PMID: 28114554). Subsequent retrospective studies have questioned this finding, including a retrospective analysis of nearly 200,000 ICU patients in 2017. A 2018 meta-analysis (PMID: 29404582) calculated a sensitivity or 61% for qSOFA (versus 88% for SIRS) and a specificity of 72% (versus 26% for SIRS).

iv. **NEWS (National Early Warning Score)**

1. Criteria
    a. RR
    b. SpO2
    c. O2 supplementation
    d. Temp
    e. SBP
    f. HR
    g. Mental status
2. Note that this is another Sepsis screening tool (not to be used alone without clinical interpretation)

d. **What is our patient's diagnosis?**

i. Sepsis (cannot label as septic shock until resuscitation is attempted)

2. You begin writing orders. The patient's wife makes it clear that he should remain full code. What level of care do you anticipate? What labs do you want to order?

a. **Disposition:** Patients with a change in clinical status should be re-triaged. Some tools that could help with disposition are listed below.

i. Pneumonia: CURB-65 vs PSI/PORT

1. **Spaced learning from last week. Have group pull up MDCalc and calculate Curb-65 or PSI/PORT score to help determine disposition.**

ii. Sepsis: qSOFA vs SOFA (need labs before the latter can be applied)

1. qSOFA score = 3

iii. Disposition: Patient should be transferred to the MICU or MSD.

b. **What labs and/or imaging would you order?**

i. CBC, renal, LFTs, ABG/VBG, lactate, INR, blood cultures (x2), UA(?), CXR

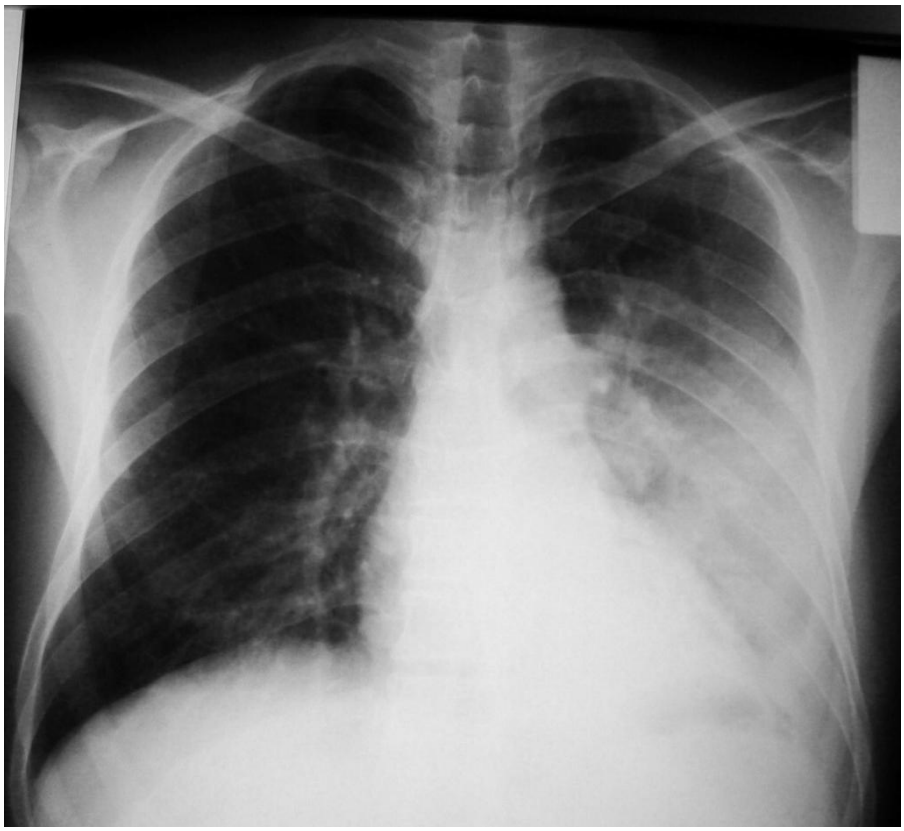
CASE: Continued…

Labs:

- CBC: WBC = 14,000, Hgb = 9.5, Hct = 32, Plt count = 120,000
- Renal: Na = 142, K = 3.8, Cl = 105, Bicarb = 12, BUN = 40, Cr = 1.5 (baseline 1.1), glucose = 210
- LFTs: AST = 35, ALT = 42, Alk phos = 120, total bilirubin = 1.1
- ABG: pH = 7.24, pCO2 = 28, pO2 = 68
- Lactate = 4.2
- Procalcitonin = 0.6
- Blood cultures = pending
- UA = spec gravity = 1.035, glucose = 0 mg/dL, protein = 100 mg/dL, ketones = 0 mg/dL, WBC = 2, RBC = 0, leuk esterase = negative, nitrite = negative, bacteria = none

Imaging:

- CXR:



3. Calculate patient's SOFA score. Determine his acid-base status.

a. **SOFA**
   i. Score = 6 points (GCS scored as 14 for confusion, FiO2 estimated as 29% using the rule of 4% for each L of flow via nasal cannula)

b. **Acid-Base Status**
   i. SPACED REPITITION: Acid-base review from week 1
   ii. Answer: Anion gap metabolic acidosis
   iii. Winter's Formula

1. pCO2 = (1.5 x HCO_3) + 8 ± 2
    a. 28 = (1.5 x 12) + 8 ± 2 = 26 ± 2
2. Interpretation: appropriate respiratory compensation

iv. Delta Ratio

1. Delta Ratio = Δ (increase) in Anion Gap / Δ (decrease) in HCO_3-
    a. Delta Ratio = (25 – 12) / (24 – 12) = 13/12 = 1.08
2. Interpretation: pure anion gap metabolic acidosis
    a. < 0.4 due to a pure NAGMA
    b. 0.4 – 0.8 due to a mixed AGMA + HAGMA
    c. 0.8 – 2.0 due to a pure AGMA
    d. > 2.0 due to a mixed AGMA + metabolic alkalosis

4. What is the utility of procalcitonin?

a. Sepsis: Evidence does not currently support routine use in the setting of sepsis
b. 2 exemplar studies:
    i. 2011 open-label RCT in *Critical Care Medicine* (PMID: 21572328):
        1. Intervention:
            a. "Procalcitonin arm" supplemented standard of care with a drug-escalation algorithm and intensified diagnostic based on daily procalcitonin measurements
            b. Any procalcitonin level > 1 ug/L on daily labs automatically triggered broader antibiotic coverage and increased imaging in the intervention arm
        2. Conclusion:
            a. Procalcitonin-guided antimicrobial escalation in the ICU did not improve survival, while leading to organ-related harm (i.e. kidney injury) and prolonged admission
    ii. 2017 Cochrane Review:
        1. Inclusion criteria:
            a. Reviewed 10 randomized control trials that studies PCT-guided decisions in at least one comparison arms for adults with sepsis, severe sepsis, or septic shock
        2. Analysis:
            a. Meta-analysis of the primary outcomes of mortality, time spent receiving antimicrobial therapy, time on broad-spectrum antibiotics (before narrowing), and time on mechanical ventilation
        3. Conclusions:
            a. Low-quality evidence showed no significant differences in mortality; time spent on antibiotics was -1.28 days in the PCT intervention arms (statistically significant, but considered very low-quality evidence based on the design of the trials)
c. Per Surviving Sepsis 2021 Guidelines:

i. "For adults with suspected sepsis or septic shock, we **suggest against** using procalcitonin plus clinical evaluation to decide when to start antimicrobials, as compared to clinical evaluation alone."
ii. "For adults with an initial diagnosis of sepsis or septic shock and adequate source control where optimal duration of therapy is unclear, we **suggest** using procalcitonin AND clinical evaluation to decide when to discontinue antimicrobials over clinical evaluation alone."

5. After calling a rapid, the patient is transferred to the MICU. What is your initial resuscitation plan?

a. **Access?**
   i. Two large-bore (18G or greater) IVs
      1. NOTE: two large-bore IVs are actually preferred over CVC or PICC lines for rapid fluid/blood infusions (PMID: 20581377); CVC and PICC lines are often placed in the MICU to establish secure access, manage caustic meds, and allow for hemodynamic monitoring.

b. **Monitors?**
   i. If not already applied, the patient should be placed on HR/BP monitors and continuous pulse ox with frequently recycled BP measurements

c. **What is the goal time for administration of antibiotics?**
   i. 1 hour for all patients with possible septic shock or a high likelihood of sepsis.
      1. Of note, the 2021 Surviving Sepsis Guidelines recommend: "for patients with possible sepsis without shock, we **suggest** a time-limited course of rapid investigation and if concern for infection persists, the administration of antimicrobials within 3 hours from the time when sepsis was first recognized." This represents a change from the 2016 guidelines which recommend 1 hour to antibiotics for everyone with concern for sepsis.
   ii. What is the associated mortality increase with each hour of delay in antibiotics? 7-12%

d. **What factors influence your choice of antibiotics?**
   i. Source of infection
   ii. Personal risk factors for MRSA, Pseudomonas, and MDROs
      1. MRSA coverage in patients with any risk factors for MRSA, including hospital acquired infections, previous cultures
      2. Pseudomonas coverage in high-risk populations discussed in the PNA AHD such as structural lung disease, intubation/trach, previous cultures
   iii. From 2021 Guidelines: "For patients with sepsis or septic shock and high risk for multidrug resistant organisms, we **suggest** using two antimicrobials with gram-negative coverage for empiric treatment over one gram-negative agent" **Very low-quality evidence.** For example, as we discussed in PNA AHD, consider double covering for pseudomonas, but not commonly done in practice.

iv. Local antibiogram - **do they know how to look this up?** Click on antibiogram link in the order of any antibiotic (may need to log in using uid_____@uchealth.com email account)
v. Severity of illness

e. **What antibiotics does this patient need?**
   i. CAP coverage (ceftriaxone + azithromycin). Spaced learning from PNA AHD
f. **How much fluid should be given?**
   i. Per Surviving Sepsis Guideline initial goal of 30 cc/kg in first 3 hours
      1. Now somewhat debated, most will approach by giving aliquots (500-1000 mL) and reassessing after each bolus up to 30 cc/kg
   ii. Consider a more conservative approach in patients with underlying cardiomyopathy.
   iii. Reassess for fluid responsiveness after **each** bolus
g. **What kind of fluid?**
   i. Crystalloids versus colloids
      1. Answer: Crystalloids preferred over colloids (*Surviving Sepsis Guideline)
      2. Studies/trials:
         a. ALBIOS Trial (PMID: 24635772): Multi-center, open-label trial performed in 100 ICUs. 1818 patients with severe sepsis were randomly assigned to 20% albumin solution or crystalloid arms. Albumin replacement did not improve the rate of survival at 28 and 90 days.
      3. Caveats: Consider colloids (including blood products) if Hgb < 7 or if sepsis is secondary to SBP
   ii. Balanced fluids versus saline
      1. Answer: Balanced fluids may improve outcomes
      2. Studies/trials:
         a. SMART-MED Trial (PMID: 29485925): Cluster-randomized, multiple-crossover trial performed in 5 ICUs at Vanderbilt. Total of 15,802 adults enrolled. Using balanced fluids resulted in a lower rate of persistent renal dysfunction, new renal replacement therapy, and the composite outcome of death.
      3. Theory: Based on animal studies, a chloride load seems to result in renal vasoconstriction and decrease renal blood flow / GFR
      4. Caveats: Need to consider potassium load in patients with renal failure.
         a. Realistically, this is not a significant K load even in renal failure

6. How will you assess for fluid responsiveness? Should we assess fluid responsiveness before giving 30 mL/kg of crystalloids?

a. Answer:
   i. Repeat vitals, exam, and lactate after the initial fluid challenge

ii. NOTE: There is controversy about whether fluids should be administered aggressively in early resuscitation efforts or if patients should be placed on pressors faster
    1. The CLOVERS Trial (PMID: 36688507) (in which UC was one of the participating centers) examined restrictive versus liberal fluid strategies during the first 24 hours of resuscitation for sepsis-induced hypotension. The primary outcome was 90-day in-hospital mortality (mortality before discharge or within 90 days if still admitted). There was no difference between the two groups (published 2/2023).

b. Strategies for gauging fluid responsiveness prior to administering large-volume fluids boluses include dynamic and static variables:
    i. Dynamic variables or small volume challenges should be used instead of static parameters to predict fluid responsiveness
    ii. Dynamic variables (Google ICU One Pager: Fluid Responsiveness and Tolerance)
        1. IVC diameter variation with respiration (only reliably studied in mechanically ventilated patients)
        2. pulse pressure variation with respiration (e.g.. Vigeleo)
        3. stroke volume variation with respiration (e.g. CHEETAH)
        4. plethysmographic variability index
        5. variation in aortic blood flow peak velocity

        NOTE: these measurements examine variations in surrogate markers of cardiac output during the respiratory cycle when preload naturally oscillates, giving the investigator some sense of how cardiac output will respond to more volume

    iii. Static variables
        1. CVP
        2. pulmonary artery occlusion / wedge pressure
        3. IVD diameter
        4. left ventricular diastolic area
        5. global end diastolic volume

        NOTE: CVP (8-12 mmHg) was used to help guide Early Goal-Directed Therapy (EGDT) strategies for resuscitation in the early 2000s, before subsequent trials showed no benefit (with some post-hoc analysis suggesting potential harm with aggressive fluid resuscitation)

    iv. Fluid “challenge” strategies
        1. (1) Mini-bolus of 300-500 mL, (2) passive leg raise

        NOTE: passive leg raise accurately predicts fluid responsiveness, with a positive LR of 11 and a pooled area under the ROC curve of 0.95 (PMID: 26741579)

        2. Technique:
            a. Place patient in a semi-recumbent (if possible)
            b. Lower head of bed to 0˚
            c. Raise the foot of the bed to 45˚

d. Measure a surrogate marker of cardiac output before and after 30-90 seconds
    i. pulse pressure change (on arterial line)
    ii. stroke volume on (cardiac output monitor)
    iii. aortic blood flow velocity (TTE)
e. Increase of > 10-15% suggests the patient will respond to fluids
f. NOTE: Moving from a semi-recumbent to a modified Trendelenburg position (i.e. torso flat, feet raised) provides a temporary fluid bolus of 300-500 mL, while raising the legs from a recumbent position equates to a 150-300 mL bolus

BREAK (please regroup at 2:15 PM; type any questions you have for the expert into the chat box)

7. **Mini Literature Review** ***(Depending on pace of progress, consider skipping this section)***
    a. **Instructions:** Have each learner briefly look up 1-2 of the following studies and summarize for the group
    b. **Studies**
        i. **Rivers (2001)**:
            1. Description: Early Goal Directed Therapy (EGDT) in the Treatment of Severe Sepsis and Shock
            2. Conclusion: Targeting a MAP > 65 mmHg, CVP between 8-12 mmHg, ScvO2 >70%, and UOP > 0.5 mL/kg/hr conferred a 16% ARR (with a NNT of 6)
        ii. **ARISE (2014)**:
            1. Description: EGDT vs physician directed care
            2. Conclusion: No change in mortality at 90 days
        iii. **PROCESS (2014)**:
            1. Description: EGDT vs noninvasive hemodynamic monitoring vs usual care
            2. Conclusion: No change in mortality at 60 days
        iv. **PROMISE (2015)**:
            1. Description: EGDT vs usual care
            2. Conclusion: No mortality benefit at 90 days
        v. **PRISM (2017)**:
            1. Description: Meta-analysis of all the trials list above
            2. Conclusion: No mortality benefit with EGDT regardless of sepsis severity
        vi. **TRISS (2014)**:
            1. Description: RCT of liberal (<9) vs restrictive (<7 Hgb) transfusion strategies in critically ill patients
            2. Conclusion: No mortality benefit at 90 days (but 50% less transfusions with restrictive transfusion strategy)
        vii. **ALBIOS (2014)**:
            1. Description: Randomized trial assessing 20% albumin vs crystalloid resuscitation in septic patients
            2. Conclusion: Albumin provided no benefit over crystalloids
        viii. **SMART-MED (2018)**:

1. Description: Cluster-randomized, multiple-crossover trial comparing outcomes with balanced fluids versus normal saline in critically ill patients
2. Conclusions: Using balanced fluids resulted in a lower rate of persistent renal dysfunction, new renal replacement therapy, and the composite outcome of death

ix. **SOAPII (2010)**:
1. Description: Dopamine vs norepinephrines as first line vasopressors in septic shock
2. Conclusions: No difference in 28-day mortality, but dopamine is associated with 2x more arrhythmias

x. **VASST (2008)**:
1. Description: RCT comparing addition of vasopressin to NE (to maintain a low rate of NE) versus escalating doses of NE only
2. Conclusions: Addition of vasopressin does not decrease overall mortality, but may confer a small survival benefit at 28 days in less severe cases of sepsis

xi. **SEPSISPAM (2014)**:
1. Description: RCT comparing MAP goal of 80-85 mmHg to a more liberal goal of 65-70 mmHg
2. Conclusions: MAP goal of 80-85 mmHg had no mortality benefit over MAP 65-70 mmHg, but did improve renal outcome in patients with chronic HTN

xii. **CORTICUS (2008)**:
1. Description: RCT comparing addition of hydrocortisone in sepsis to placebo
2. Conclusions: Hydrocortisone hastens the reversal of shock, but does not confer a survival benefit among patients with septic shock.

xiii. **ADRENAL (2018)**:
1. Description: RCT comparing week-long administration of 200 mg/day of hydrocortisone to placebo in the septic shock
2. Conclusions: In ventilated patients on pressors, continuous hydrocortisone infusion did not reduce 90-day mortality, but was associated with shortened reversal of shock / time to extubation / length of ICU stay / blood transfusions

xiv. **NICE-SUGAR (2009)**:
1. Description: RCT comparing intensive glycemic control to conventional control
2. Conclusions: Intensive glycemic control (80-110) has a higher mortality compared to liberal glycemic control (140-180)

CASE: Continued…

The patient shows no improvement despite aggressive IVF resuscitation and antibiotics. He requires intubation in the MICU for worsening respiratory failure. Central venous and radial arterial lines are placed for administration of vasopressors and hemodynamic monitoring. There is no change in pulse pressure (e.g. cardiac output) based on proprietary Vigileo algorithms with a passive leg raise. Despite

receiving a total of 5 liters of LR, patient's MAP remains below 65 mmHg with a lactate of 4.1 and urine output of 10 mL/hr.

8. What does this patient have now?

a. **Answer:** Septic shock

b. **What objective data can we follow to capture worsening sepsis?**

   i. Blood pressure, heart rate, UOP, lactic acid

9. What is the next step?

a. Answer: If patient has been adequately volume resuscitated but remains hypotensive, administer vasopressors

b. What is our MAP goal?

   i. MAP > 65-70 mmHg (*Surviving Sepsis Guideline)

c. If you're called to the bedside of a patient how can you quickly calculate a patient's MAP in your head using their SBP/DBP? If on the floor during rapid/code can be very helpful to quickly calculate a MAP in your head.

   i. Subtract your DBP from your SBP

   ii. Divide that number by 3

   iii. Add that to your DBP- you now have your MAP!

   iv. Example using BP of 65/50

      1. SBP (65) minus DBP (44): 21
      2. 21/3 = 7
      3. DBP + 7 = 51 = MAP!

d. Which vasopressor(s)?

Vasopressor / Inotrope	Receptor: alpha-1	beta-1	beta-2	Dopamine Receptor	Physiologic Effects
Norepinephrine	+ + +	+ +	0	0	↑↑ SVR +/- CO
Dobutamine	+/-	+ + +	+ +	0	↑ CO ↓ SVR
Epinephrine	+ + +	+ + +	+ +	0	↑↑ CO ↓ SVR (low dose) ↑ SVR (high dose)
Dopamine (mcg/kg/min)					
1 to 3	0	+	0	+ +	CO
5 to 10	+	+ +	0	+ +	↑ CO ↑ SVR
> 10	+ +	+ +	0	+ +	↑↑ SVR
Phenylephrine	+ + +	0	0	0	↑↑ SVR +/- CO

CO: Cardiac Output
SVR: Systemic Vascular Resistence
+++ Strong effect ++ Moderate Effect + Weak Effect 0 No effect

   i. Norepinephrine recommended as a first-line agent, with the SOAPII trial showing increased arrhythmias with dopamine (*Surviving Sepsis Guideline)

   ii. Can add vasopressin (VASST) to help decrease overall NE dose

1. NOTE: vasopressin had a different MOA than catecholamines (i.e. V1 mediated vasoconstriction)

iii. Generally epinephrine is the 3rd vasopressor we add on, but this can sometimes vary based on the underlying physiology and clinical picture.

10. Despite max pressor support, patient continues to be hypotensive with little UOP, what is a possible next step?

a. **Reassess shock type** (e.g. bedside echo)
   i. Consider adding dobutamine if there is a cardiac component
b. **Consider corticosteroids** (patients with overwhelming sepsis may have relative adrenally insufficiency)
   i. Review ADRENAL and CORTICUS studies (above)
   ii. Add hydrocortisone 200mg/day in divided doses if MAP goal not attained - often dose 50 mg q6h here (*Surviving Sepsis Guideline)
c. **Should we do ACTH stim first**?
   1. Answer: No (PMID: 25521173)

11. After starting steroids, the next two FSBS levels are 260 and 285. What is your next course of action?

a. If >2 consecutive FSBS > 180, start insulin gtt with a goal glucose level of 140-180 (*Surviving Sepsis Guideline) Review the NICE-SUGAR trial (above)

BREAK (please regroup at 3:20 PM; type any questions you have for the expert into the chat box)