Foundations of Emergency Medicine
Foundations III: Guided Small Group Experience

Session 20: Cardiogenic Shock and the LVAD
Unit: Critical Care

❖ **Agenda and Learning Objectives**:
- Case Part I – Diagnosis and stabilization of cardiogenic shock (20 min)
  - Correlate exam and POCUS findings to the pathology of cardiogenic shock
  - Make the diagnosis of recent STEMI as the cause for new onset cardiogenic shock
  - Stabilize the patient in cardiogenic shock
- Case Part II – Left Ventricular Assist Device (LVAD) Dysfunction (20 min)
  - Understand the normal function and components of a LVAD
  - Evaluate and examine a patient with a LVAD
  - Understand the most common LVAD-related complications
- Case conclusion (10 min)
  - Review session teaching points

❖ **Note to Facilitators**

This is a case in the critical care unit and covers cardiogenic shock and LVADs. It is a question guided 50 minutes session without small group activities. It may be useful to review which LVADs are used at your institution prior to the session but otherwise there is no special preparation. There are also no special materials needed to facilitate this session.

❖ **Case Begins – Diagnosis and stabilization of cardiogenic shock (20 min)**

- *A 66 year old male with a past medical history of CAD with stent placement 3 years ago, HTN, and DM presents with shortness of breath, leg swelling, and chest pain for two days.*

  On your evaluation of the patient, his vital signs are BP 75/47, HR 114, pO_2 89%, T 36.6, RR 24. You note that your patient is pale, with increased work of breathing, and can only communicate in 3-4 word sentences. On exam, you note crackles in the bilateral bases, JVD , a non-tender abdomen with abdominal wall edema, and 3+ pitting edema in both lower extremities.

  Immediately on arrival, you request an EKG (anterolateral STEMI):

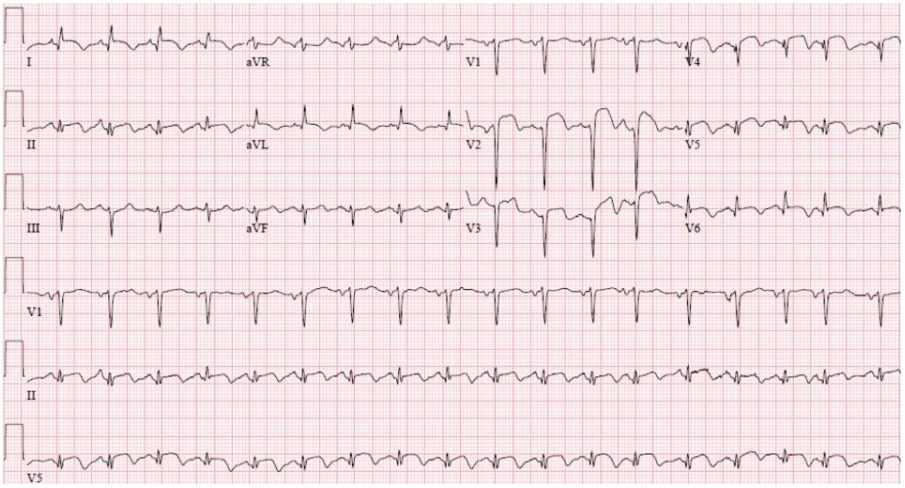
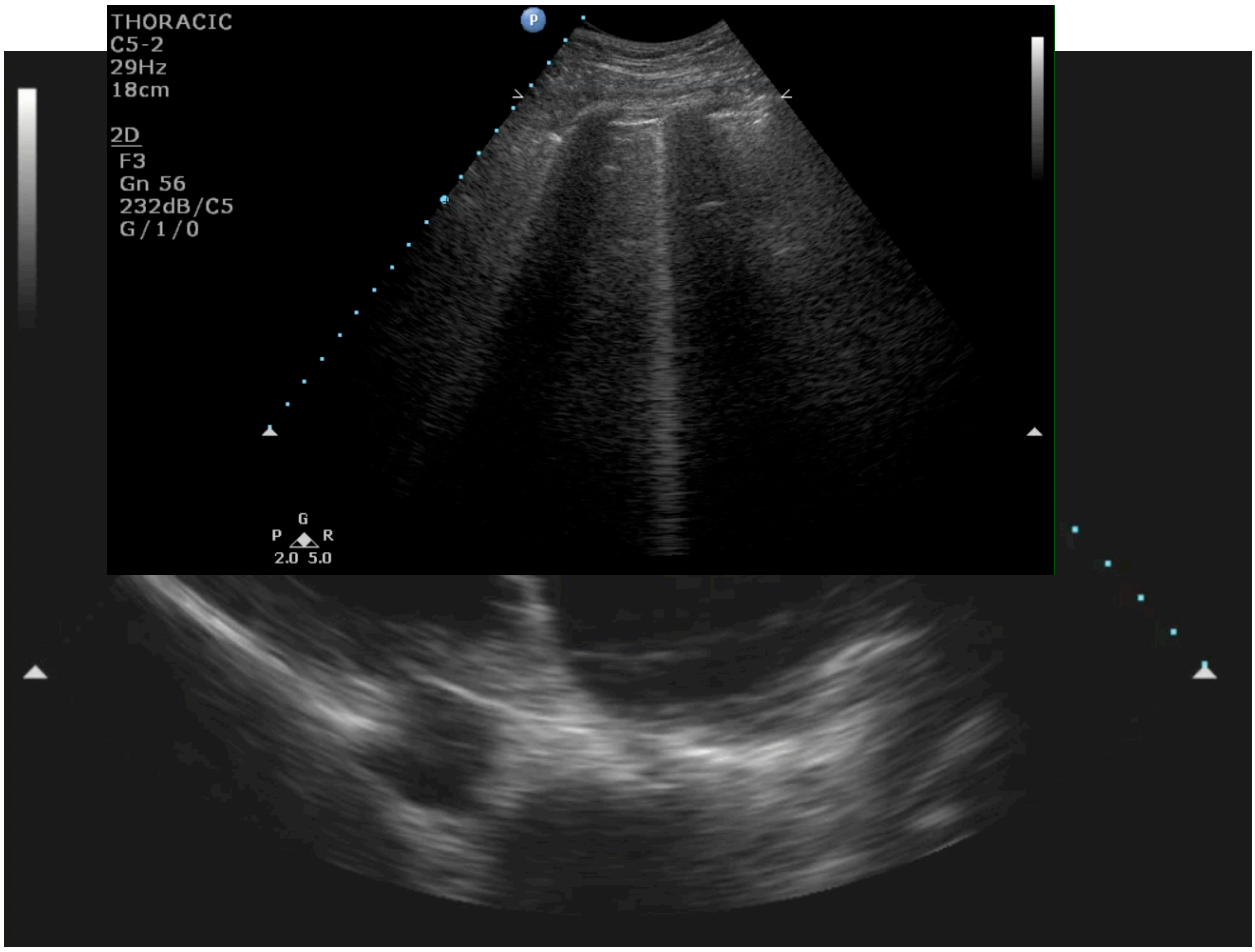


Figure 1: Foundations EKG Course.

- **What immediate (next couple minutes) interventions do you want to pursue next?**
  - Cardiac monitor and pulse oximetry
  - Start 2 large bore IV's
  - Initiate BiPAP while avoiding hyperoxia, unless pre-oxygenating for anticipating impending intubation
  - Order pressors to bedside → in the meantime consider mixing push dose pressors to bedside in anticipation of needing them if there will be a delay in the drips
  - POCUS
  - Activate the interventional cardiology team

- **What are you looking for on POCUS? Describe your shock differential for this patient and how POCUS can help narrow it.**
  - Differential diagnosis includes: septic shock, tension pneumothorax, cardiac tamponade, massive PE, right heart failure, cardiogenic shock
  - Pertinent positives on this patient: cardiomegaly with low ejection fraction, an IVC that does not collapse with respiration, diffuse B lines

- Pertinent negatives: no pericardial effusion, normal LV/RV ratio, negative McConnell's sign, normal lung sliding, normal valve function



❖ **What is your most likely diagnosis? What are the criteria to diagnose cardiogenic shock and why does it happen?**

- Cardiogenic shock in the setting of an acute myocardial infarction.
  - This is the most common cause of cardiogenic shock, though it rarely occurs right away, as in this patient who is presenting 48 hours after his STEMI began
  - It is related here to reduced cardiac contractility from infarction → it is also important to evaluate for valve rupture on POCUS (or listen for new murmurs) because this would change management as it is a surgical emergency if found.
- Cardiogenic shock is defined as an acute state where decreased cardiac output results in end-organ hypoperfusion
- Criteria for cardiogenic shock vary by source but are generally agreed to be:
  - SBP <90mmHg, MAP <65mmHg, and/or vasopressors required to achieve SBP >90mmHg
  - Pulmonary congestion or elevated left-ventricular filling pressures
  - Signs of impaired organ perfusion either clinically or by lab evaluation
- Pathophysiology

- An acute myocardial infarction results in a decrease in myocardial contractility and subsequent hypoperfusion of tissues. → as a result of hypoperfusion, the myocardium has increasingly poor function leading to a negative feedback loop resulting in progressively worsening cardiac function
- In acute MI, the heart is thought to release nitric oxide, which further inhibits myocardial contractility, reduces responsiveness to catecholamines, and induces systemic vasodilation

❖ **What are the typical findings on physical exam and how do they relate to the pathophysiology of cardiogenic shock?**

- The exam is often characterized by hypoperfusion and volume overload, known classically as "cold and wet"
- Hypotension is often seen with a systolic blood pressure less than 90 mmHg or MAP less than 65 mmHg with pulse pressure less than 20 mm Hg → the narrow pulse pressure is secondary to a compensatory vasoconstriction and there is often a compensatory sinus tachycardia also → note that many of these patients are on beta blockers which might blunt this response
- Tachypnea is often present with the risk of progression to respiratory failure as a result of pulmonary edema → pulmonary edema is heard as crackles on the lung exam, seen as jugular venous distention, B lines on POCUS and can also be seen on CXR
- Peripheral edema suggests preexisting cardiac disease and is often associated with cyanotic, cool, and/or mottled extremities
- The cardiac exam can reveal a new murmur in the case of acute mitral regurgitation from ruptured chordae tendinea or papillary muscle dysfunction (POCUS can also aid in identification) → If this is suspected, immediate cardiothoracic surgery involvement is necessary

❖ **What are your first steps in stabilization in the ED?**

- Respiratory support
  - Titrate to an adequate oxygen saturation of mid-90's and reassess work of breathing
  - If oxygenation is difficult or work of breathing does not improve with adequate oxygenation, BiPAP may be needed
  - If a low likelihood of success with noninvasive ventilation is anticipated, then ensure maximal oxygenation in anticipation of intubation (see Session 14: Unstable Intubations)
    - BiPAP FiO2 to 100%
    - Nasal cannula under the BiPAP set to 15 L/min
  - Remember the hemodynamic effects of noninvasive and mechanical ventilation
    - They both change the intrathoracic pressure and may result in decreased preload and worsening hypotension
    - With BiPAP, and particularly intubation, a small fluid bolus or vasopressor therapy should be strongly considered since these patients are at high risk for hemodynamic decompensation or peri-intubation arrest → make sure to get these bedside before you proceed!
  - Circulatory support

        - Use your examination and POCUS to guide management
        - If EF is preserved, use norepinephrine for a MAP goal of 65
        - If EF is reduced, begin with an inotrope such as dobutamine
        - Reassess the MAP response after initiating dobutamine and add norepinephrine or epinephrine, if needed, for a goal MAP of 65
        - Dobutamine acts as a positive inotrope on the heart but also can be a vasodilator so be prepared for worsening hypotension and have a pressor hanging before initiating dobutamine
            - About 1/3 of patients will become *more* hypotensive with dobutamine so consider starting the norepinephrine or epinephrine WITH or BEFORE you start dobutamine
    - Other downstream therapies
        - Reperfusion and Revascularization
            - The AHA recommends early invasive revascularization (PCI or CABG) for all patients with an ACS-associated cardiogenic shock regardless of time delay from MI onset (SHOCK and SMASH trials)
            - This has been shown to have a reduction in mortality at one year
        - Fibrinolytics
            - This can be considered when an early invasive approach to revascularization is not available
            - For this option, involve your consultants either locally or at your receiving hospital if you are planning on transferring the patient

❖ **Despite medical therapy with vasopressors and inotropes, your patient continues to be hypotensive, what are other options?**
- *The following would be done in consultation with your cardiology and critical care consultants and would depend on availability at your institution. Catheterization first is the mainstay of treatment if available for ischemic LV failure.*
    - Intra-aortic balloon pump (IABP)
        - IABP has not been shown to decrease mortality (IABP-SHOCK II trial)
        - Use is considered in the following:
            - When a patient is revascularized by thrombolysis
            - When no revascularization has been done
            - When ECMO is unavailable or significantly delayed
    - Impella
        - This is a percutaneous trans-aortic valve device that simply pumps blood from the ventricle into the aorta
        - Used in high risk cardiac procedures (PCI) or in the setting of cardiogenic shock following acute MI
        - Unloads the left ventricle, decreasing end diastolic volume and pressure as well as LV wall stress, LV work, and myocardial oxygen demand
        - Can increase MAP, diastolic pressure, and cardiac output

- Veno-arterial Extracorporeal Membrane Oxygenation (V-A ECMO)
  - Used in the setting where hypotension continues despite medical therapy
  - ECMO is the preferred next step to provide complete cardiopulmonary support when the condition is not expected to rapidly improve. This is especially effective when the cause of cardiogenic shock is reversible.
- Temporary LVAD placement
  - Can also be considered for isolated LV failure without a significant oxygenation issue (which would then require V-A ECMO)

❖ **Case Part I Concludes**

- *Respiratory support*
  - *Your patient was placed on BiPAP. Your settings were iPAP of 10, ePAP of 5, and you set a FiO2 of 100% due to anticipating the need for endotracheal intubation*
  - *You placed a nasal cannula under the BiPAP mask set to 15 L/min*
- *Circulatory support*
  - *You initiated a dobutamine drip due to the reduced cardiac contractility evident on your POCUS*
  - *Repeat MAP was still under 65 so you also initiated norepinephrine*
- *Consultants*
  - *You paged the interventional cardiology team immediately upon diagnosis of cardiogenic shock*
- *Intubation was successful → blood pressure was adequately maintained throughout due to proper peri-intubation management*
- *Cardiology took the patient to the cath lab where they stented a proximal LAD lesion*

The patient had a prolonged hospital stay in the CCU before discharge home with a LVAD

❖ **Case Part II – Left Ventricular Assist Device Dysfunction (20 min)**

- *One year later you are caring for the same patient who now has a LVAD. He presents to the ED with complaints of nausea, vomiting, diarrhea, and chills for several days. He is accompanied by his wife who states that "he is just not acting right" so she brought him in. He has a grandchild with similar symptoms at home.*

  On exam, he is confused but awake. MAP 52, HR 110, pO_2 unobtainable, T 37.2, RR 28. His abdomen is soft and nontender. Rectal with no blood. Lungs are clear. LVAD is in place with lines appearing clean. 1+ bilateral lower extremity edema. Pulses cannot be palpated.

  POCUS shows a small left ventricle with right to left septal shift.

  The LVAD control unit has good battery power, a flow rate of 3, power of 3 watts, and a pulsatility index of 8.

- **What is the function of a left ventricular assist device and how do you measure a blood pressure?**
  - Left ventricular assist devices are used as a bridge to cardiac transplant, a bridge to recovery, a bridge to decision or as destination therapy for patients in end-stage congestive heart failure
  - Left ventricular assist devices have the main function of unloading the left ventricle throughout the entire cardiac cycle → they essentially work as a continuous one-way simple pump
  - The result is reduced myocardial work and oxygen demand leading to adequate maintenance of systemic perfusion and end-organ function
  - Since LVAD flow is continuous, there is usually no arterial pulse present (unless there is significant underlying native cardiac function) and there is one continuous pressure maintained (the MAP) → the MAP is measured using a blood pressure cuff and doppler
  - To measure the blood pressure, find blood flow using a Doppler ultrasound device over the radial artery. Then inflate a standard manual blood pressure cuff over the brachial artery until the flow stops. Deflate the cuff slowly and measure the cuff pressure at the point Doppler flow resumes in the radial artery. The pressure at which Doppler flow resumes is the MAPA normal MAP is between 70-90 mmHg, often the patients know their typical MAP

- **What are the main components of the LVAD?**
  - The components of the LVAD include:
    1. Inflow cannula: from the left ventricle
    2. The pump: electrical and mechanical components
    3. Outflow conduit: anastomosed to ascending aorta
    4. Percutaneous lead (also called the Driveline)
    5. External components: controlled, monitors, and power source

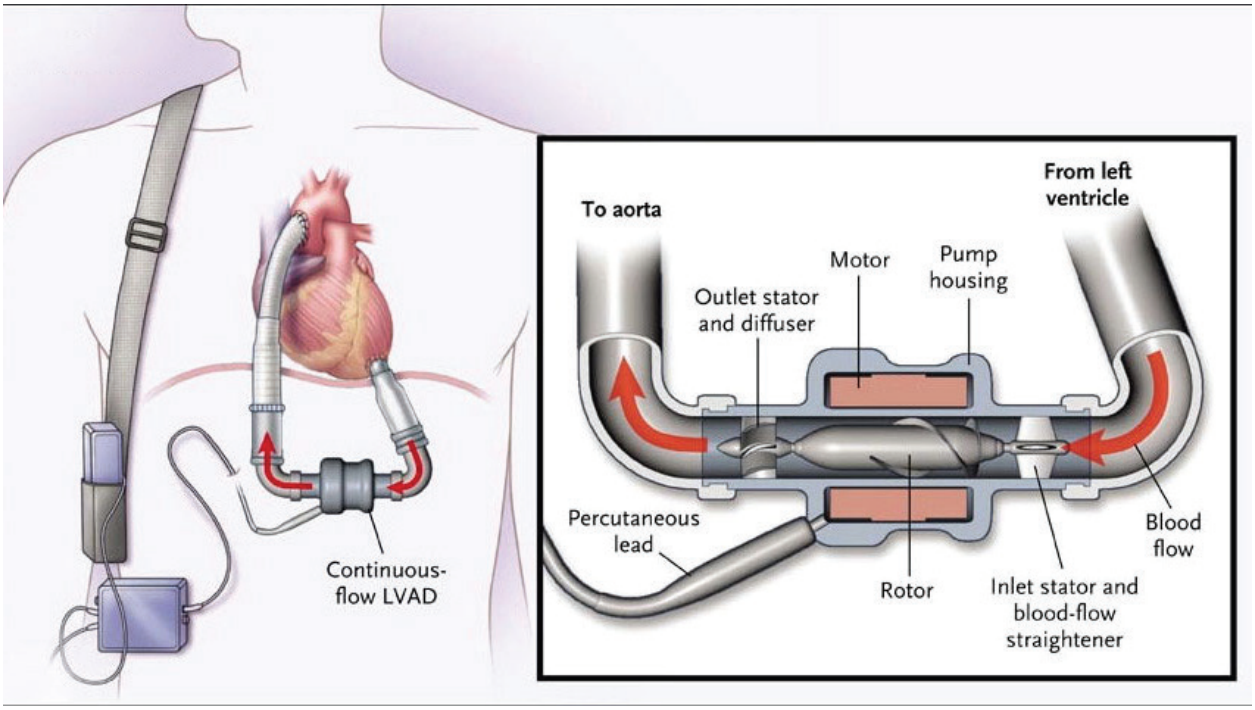


Figure 2: Continuous-flow left ventricular assist device.
Source: N Engl J Med. 2009;361(23):2241-2251

- **How might the initial evaluation and vital signs differ for a patient with a LVAD?**
  - Evaluating the patient with a left ventricular assist device starts with a basic primary survey
  - You should be able to auscultate a precordial/epigastric hum → this verifies function of the LVAD
  - Peripheral and distal pulses may be decreased or absent at baseline
  - EKG remains effective in evaluating for arrhythmia
  - Instead of a traditional blood pressure, the mean arterial pressure (MAP) is obtained
    - This is done with a manual blood pressure cuff and a Doppler ultrasound over the brachial or radial artery to find the number where you hear a return to flow → this number is the MAP
  - A normal MAP will be 70 mm Hg – 90 mm Hg → too low is a problem but so is too high
  - Hypotension and/or shock can be LVAD related or non-LVAD related
  - A quick POCUS and LVAD control device check can help narrow the differential diagnosis
    - POCUS
      - Focus on assessing relative sizes of the left and right ventricles (we'll tie this in diagnostically below)
      - Continue to assess for other findings that may indicate a non-LVAD related cause
    - Assess the LVAD control device readings (it is useful to review which LVADs are used at your institution and their normal functioning)
      - Power
        - How hard the pump is working
        - Normal is 4-6 watts
      - Flow
        - Flow rate of blood (ie cardiac output)
        - Normal is 4-6 L/min
      - Pulsatility Index (PI)
        - How much cardiac output is provided by the LVAD versus the patient. The higher the PI, the more work the native heart does versus the LVAD
        - Normal is 1-10
      - Battery power

- **How do POCUS and LVAD Pump findings relate to the potential differential diagnosis?**

  - Regarding POCUS and LVAD pump findings, here is a table that summarizes how they tie into some of your differential diagnosis. Have this available as a reference rather than memorizing. Keep it with your information on your normal LVAD control device readings.

POCUS Findings	Potential Causes	LVAD Pump Findings
Abnormally small LV (Right to left septal shift)	Suction event	Low power Normal or high PI High speed (rpm) Low pump flow
Large RV and LV	Pump thrombosis/obstruction	High power Low PI High speed (rpm) Low or no hum
Large RV and small LV	RV failure Pulmonary Hypertension	High power High PI
Small RV and LV	Hypovolemia GI Bleed Sepsis	Low flow

- **What are common complications and their treatments for patients with an LVAD?**
  - *Important note: Major treatment interventions need to be done in consultation with your hospital's LVAD team. These include aggressive fluid resuscitation, reversal of anticoagulation, thrombolytics, and any change to LVAD settings.*
  - When faced with an ill LVAD patient, determine who their LVAD doctor is and contact them early → this is contrary to our approach in EM generally which is to stabilize first and call later but this patient population is different and many stabilization/treatment options are entirely outside of our scope of practice
  - There can be pathology related to the LVAD directly, like specific hardware complications, and pathology related to downstream effects of the LVAD, like GI bleeding due to LVAD-related coagulopathy and AVM formation in the GI tract.

  - Direct LVAD related Causes
    - Battery Dead or Low
      - First step is to plug the unit in if you are able (most models have a backup plug) → ask the patient, their family, the VAD coordinator or the CCU charge RN
      - If battery power is low, the batteries will need immediate replacement
      - Whenever possible, do this with the LVAD team
      - Do not disconnect both batteries at once. Do one at a time, or the LVAD will completely power down
    - Pump thrombus
      - Thrombosis can occur in the pump or its conduits leading to LVAD dysfunction
      - Findings
        - Hemolysis: dark urine, elevated LDH, elevated bilirubin
        - POCUS may show LV and RV dilatation

            - The LVAD device control unit will indicate an increased in power and a decrease in flow
        - Treatment
            - In consultation with LVAD team
                - Heparin or thrombolytics (local vs. systemic)
                - Pump Exchange
    - Cannula malposition or obstruction
        - This can be caused by kinking, thrombosis, endocarditis, anatomic stenosis, or progressive migration of the inflow cannula
        - This is diagnosed with echo, CT, or angiography.
    - Suction events
        - This is when there is left ventricular collapse as a result of the large negative pressures generated in the left ventricle by the LVAD. → it can be precipitated by anything causing LV underfilling, such as hypovolemia, RV failure, tamponade, sepsis, and pulmonary hypertension
        - Findings
            - POCUS may show an abnormally small LV with right to left septal wall shift
            - The control unit may show low flow, low power, or normal/high PI
        - Treatment
            - Direct to the most likely diagnosis
            - Trial IVF bolus to increase LV filling, if indicated by diagnosis (e.g. dehydration/hypovolemia)
                - Take caution to reassess the RV!
                - RV enlargement may indicate RV failure and fluids could become counterproductive (see Session 11: RV failure)
            - In consultation with the LVAD team
                - Decrease pump speed (per cardiology and VAD technicians)
- Indirect LVAD Complications
    - Hypertensive Emergencies
        - We often think of low flow emergencies as these are common but high MAPs are also dangerous as they put excessive pressure on the LVAD and can burn out the motor
        - MAPs should generally not be above 85-90 mmHg → if they are much higher this is considered an emergency and must be treated!
        - Findings
            - The unit will often show high power (ie the pump is working very hard) with normal flow
        - Treatment
            - Treat the blood pressure → nicardipine is preferred
            - Attempt to determine why the blood pressures are suddenly high (ie AKI, med non-compliance etc)

- Bleeding
    - Gastrointestinal bleeding, epistaxis, and intracerebral hemorrhage are more common in LVAD patients due to them being anticoagulated and due to downstream risk factors related directly to the LVAD
    - LVADs can lead to AVMs in the GI tract and acquired Von Willebrand disease.
    - Findings
        - POCUS may show an abnormally small LV and RV due to hypotension
        - The control unit may show low flow
    - Treatment
        - IVF bolus
        - Hold anticoagulation
        - In consultation with the LVAD team
            - Consider blood transfusion
                - Risk: Allosensitization of a transplant candidate
            - Consider reversal of coagulopathy
                - Risk: Pump thrombosis due to anticoagulation reversal
- Right ventricular failure (see Session 11: RV failure)
    - This occurs in up to 10-15% of patients often soon after VAD implantation (even higher if you include those immediately post-op)
    - It can result from pulmonary hypertension, RV infarction, and pulmonary embolism
    - Findings
        - POCUS will show a large RV and small LV
        - LVAD device will indicate high power and high pulsatility index
    - Treatment
        - Treatment is tailored to the specific cause of RV failure
            - Considerations include:
                - IVF bolus (small – 250 mL and reassess) in the setting of RV infarction
                - Pressors (epinephrine or norepinephrine)
                - Fibrinolytics in the setting of massive PE
            - Avoid hypoxia and hypercarbia as both contribute to increased pulmonary vascular resistance and therefore worsen right heart dysfunction
- Arrhythmias
    - These result from postsurgical scarring from device implantation, the irritation from the device itself, and other non-LVAD related conditions would put the patient at risk for dysrhythmia
    - Treatment
        - These are treated normally with standard pharmacotherapy and/or cardioversion

        - Be aggressive with treatment → many LVAD patients still rely on some contribution of their native heart to maintain their MAPs so when they go into an arrhythmia they should be considered generally unstable
    - Infection
        - Any part of the LVAD can develop an infection that causes a severe increase in morbidity and mortality. Broad spectrum antibiotics are recommended early in the patient's course.
        - Treatment
            - IVF (cautiously)
            - Antibiotics
            - Additional sepsis/septic shock management per Surviving Sepsis Guidelines
    - Stroke
        - LVAD patients are at increased risk for both embolic and hemorrhagic stroke.
        - Acute neurological changes should prompt early neuroimaging.
    - Cardiac arrest
        - Simultaneous ACLS and evaluation for possible LVAD dysfunction causing the arrest should take place.
        - Chest compressions are controversial – check with your institution
            - If you are considering chest compressions make sure you know the LVAD is **not** working and there is **no** MAP → don't do chest compressions on an unconscious LVAD patient with a functioning device that "doesn't" have a pulse (because no LVAD patients do)!
            - There is a theoretical risk of damaging the LVAD with chest compressions but if the LVAD is truly not functioning you have little other choice
            - Be on the same page as your LVAD team in terms of chest compressions

❖ **What is your main concern for this patient? What are your management steps?**
- He is hypovolemic likely from a GI source (ie gastroenteritis)
- He is showing signs of a suction event likely due to hypovolemia
- You call his LVAD coordinator
- You initiate fluids with small boluses (250 mL) with frequent reassessment, administer broad spectrum antibiotics and admit in consultation with the LVAD team

❖ **Case Concludes (10 min)**

- *You give your patient a fluid bolus of 500mL of normal saline and his blood pressure and mental status start to slowly improve. His labs return with a new rise in creatinine and lactic acid but otherwise appear within normal limits. He is admitted to the hospital with hypotension secondary to gastroenteritis causing acute kidney injury, lactic acidosis, and transient altered mental status.*

- **Case Teaching Points Summary**
  - **Cardiogenic Shock**
    - Cardiogenic shock is defined as an acute state where decreased cardiac output results in end-organ hypoperfusion despite adequate fluid volume
    - Criteria for cardiogenic shock vary by source but are generally agreed to be:
      - SBP <90mmHg or MAP <65mmHg or vasopressors required to achieve SBP >90mmHg
      - Pulmonary congestion or elevated left-ventricular filling pressures
      - Signs of impaired organ perfusion with clinical (cold extremities, mental confusion) or laboratory (metabolic acidosis, elevated serum lactate) findings
    - Reperfusion and revascularization has been shown to have a reduction in mortality at one year → the AHA recommends early invasive revascularization (PCI or CABG) for all patients with a ACS-associated cardiogenic shock regardless of time delay from MI onset
    - Cardiothoracic Surgery should be consulted immediately upon suspicion of valve rupture
    - Dobutamine should be used in cases where there is decreased ejection fraction to augment cardiac output but anticipate worsening hypotension in some patients due to the vasodilatory effect
    - Epinephrine or norepinephrine should be used in cases of refractory hypotension once dobutamine has been initiated
    - Noninvasive ventilation may change the intrathoracic pressure and result in decreased preload and worsening hypotension → during the initiation of BiPAP or intubation, a fluid bolus or vasopressor therapy should be started since these patients are at high risk for peri-intubation arrest (see Session 14: Unstable Intubations)
    - In the setting where hypotension continues despite medical therapy, mechanical circulatory support (MCS) may be required → Impella devices or intra-aortic balloon pumps are often used to augment cardiac output, decrease myocardial oxygen demand, and decrease left ventricular work
    - Peripheral extracorporeal membrane oxygenation (ECMO) may be required if less invasive MCS fails in order to provide complete cardiopulmonary support when the condition is not expected to rapidly improve and the cause is reversible

- **LVAD Evaluation**
  - Left ventricular assist devices can be used a bridge to cardiac transplant, a bridge to recovery, a bridge to decision or as destination therapy for patient in end-stage heart failure
  - Left ventricular assist devices have the main function of unloading the left ventricle throughout the entire cardiac cycle → they essentially act as a continuous one-way pump
  - The result is reduced myocardial work and oxygen demand while maintaining adequate systemic perfusion and end-organ function

- The mean arterial pressure (MAP) is obtained instead of a traditional blood pressure → this can be done with a manual blood pressure cuff and a Doppler ultrasound over the brachial or radial artery to find the return of to flow
- MAPs should be between 70-90 mmHg → both too high and too low are problems!
- When faced with an ill LVAD patient, determine who their LVAD doctor is and contact them early → this is contrary to our approach in EM generally which is to stabilize first and call later but this patient population is different and many stabilization/treatment options are entirely outside of our scope of practice
- Most common LVAD issues:
    - Directly related to the LVAD
        - Pump thrombus
        - Cannula malposition or obstruction
        - Suction Events
    - Indirectly related to the LVAD
        - Hypertensive Emergency
        - Bleeding
        - Right ventricular failure
        - Arrhythmia
        - Infection
        - Neurological complications and strokes
        - Cardiac Arrest

❖ **Facilitator Background Information**

Cardiogenic Shock

Cardiogenic shock is defined as an acute state where decreased cardiac output results in poor tissue perfusion despite adequate or excessive fluid volume. The resulting decrease in myocardial contractility following AMI is the most common cause of cardiogenic shock. The resulting decrease in cardiac output and blood pressure results in hypoperfusion of the myocardium and therefore further worsening of contractility. The exact pathophysiology is unclear but it is thought to be related to an inflammatory response after AMI that causes nitric oxide release. Nitric oxide is thought to further inhibit myocardial contractility, reduce responsiveness to catecholamines, and induce systemic vasodilation. Early intervention by percutaneous coronary intervention or coronary artery bypass has been shown to improve outcomes but cardiogenic shock continuous to portend a high mortality rate.

The physical exam (including POCUS as an adjunct) is a critical step in the evaluation of a critically ill patient with suspected cardiogenic shock. This state is often characterized by hypoperfusion and volume overload, "cold and wet". Hypotension is often seen with a systolic blood pressure less than 90 mmHg or a MAP less than 65 mmHg. A resulting compensatory vasoconstriction may result in a pulse pressure less than 20 mm Hg as well as a compensatory sinus tachycardia. Initially, tachypnea is seen and patients can quickly progress to respiratory failure. The majority of these patients are fluid overloaded and pulmonary edema is often present. Peripheral edema suggests preexisting cardiac disease and is often associated with cyanotic and cool skin with mottled extremities. The cardiac exam can reveal a new murmur in the case of acute mitral regurgitation from ruptured chordae tendinea or papillary muscle dysfunction. POCUS can be invaluable in these cases to both help make the diagnosis as well as to monitor the patient for response to therapy.

The goal in the emergency department is to support the patient medically and consider if definitive treatment either with re-vascularization in the case of AMI or surgical repair in the case of acute valvular pathology is indicated. Oxygen support, noninvasive ventilation and even intubation can be required in severe cases. Keep in mind that any change in intrathoracic pressure will decrease preload and may cause worsening cardiac output and hypotension. If the patient develops hypotension without pulmonary edema or a right ventricular infarct is suspected, a small fluid bolus can be trialed for the treatment of hypotension. If the patient has pulmonary edema or does not improve with a small fluid bolus, the next step is vasopressors and inotropes. Dobutamine should be considered as it will increase cardiac contractility but be aware it has the potential for vasodilation. EPs should be ready to add a vasopressor, epinephrine or norepinephrine, if they are initiating dobutamine in a patient with cardiogenic shock.

In the setting where hypotension continues despite medical therapy, mechanical circulatory support (MCS) is often the preferred next step. Options include an intra-aortic balloon pump, an Impella or if complete cardiopulmonary support is needed after the prior therapies have failed, veno-arterial extracorporal membrane oxygenation (V-A ECMO).

LVAD

Left ventricular assist devices can be used as a bridge to cardiac transplantation, a bridge to recovery, a bridge to decision or as destination therapy for patients with end-stage heart failure. As the prevalence of heart failure continues to increase and LVAD therapy becomes more common, emergency department providers will undoubtedly care for patients with LVADs and their associated complications regardless of practice environment.

The components of the VAD include:

1. Inflow cannula: from the left ventricle
2. The pump: electrical and mechanical components
3. Outflow conduit: anastomosed to ascending aorta
4. Percutaneous lead (also called the Driveline)
5. External components: controlled, monitors, and power source

Left ventricular assist devices have the main function of unloading the left ventricle throughout the entire cardiac cycle, reducing myocardial work and oxygen demand, and maintaining adequate systemic perfusion and end-organ function. They essentially work as a simple, continuous one-way pump. Since flow is continuous, there is no arterial pulse present.

Evaluating the patient with a left ventricular assist device starts as always with a basic primary survey. Both peripheral and distal pulses may be decreased or absent. EKG remains effective in evaluating for arrhythmia as many LVAD patients are still somewhat reliant on their underlying cardiac rhythm to maintain their MAP. In exchange for a typical blood pressure, the mean arterial pressure (MAP) is obtained. This can be done with a manual blood pressure cuff and a Doppler ultrasound over the brachial or radial artery to find the return to flow, which will be the patient's MAP. MAPs should range between 70-90 mmHg and patients often know their normal MAP. Too low or too high is a problem and both should be treated aggressively. Finally, pulse oximetry may be unreliable or unobtainable.

As the primary survey is been completed, the LVAD settings and battery should be checked. In addition to LVAD related complications above, these patients are at high risk for GI bleeding, stroke, and infection. Early in the patient's presentation, the VAD-coordination team should be contacted. Any aggressive fluid resuscitation, thrombolysis, anticoagulant reversal, or device adjustments should be done in consultation with the LVAD team.

See the case above for VAD specific complications and their unique findings.

- **References**
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  - **Secondary Author:** Dr. George Leach
  - **Editor:** Dr. Katherine Nugent, Dr. Nicholas Mohr
  - **References:**

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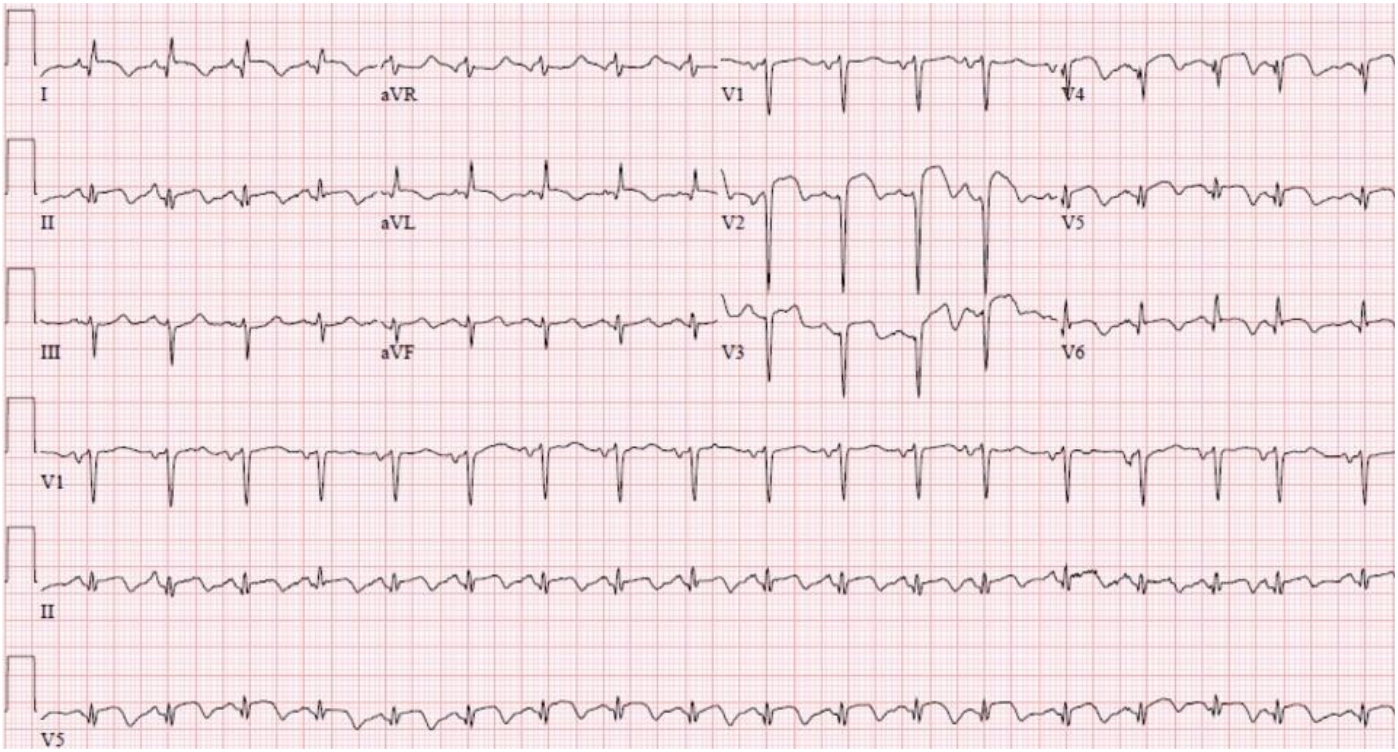

Figure 1

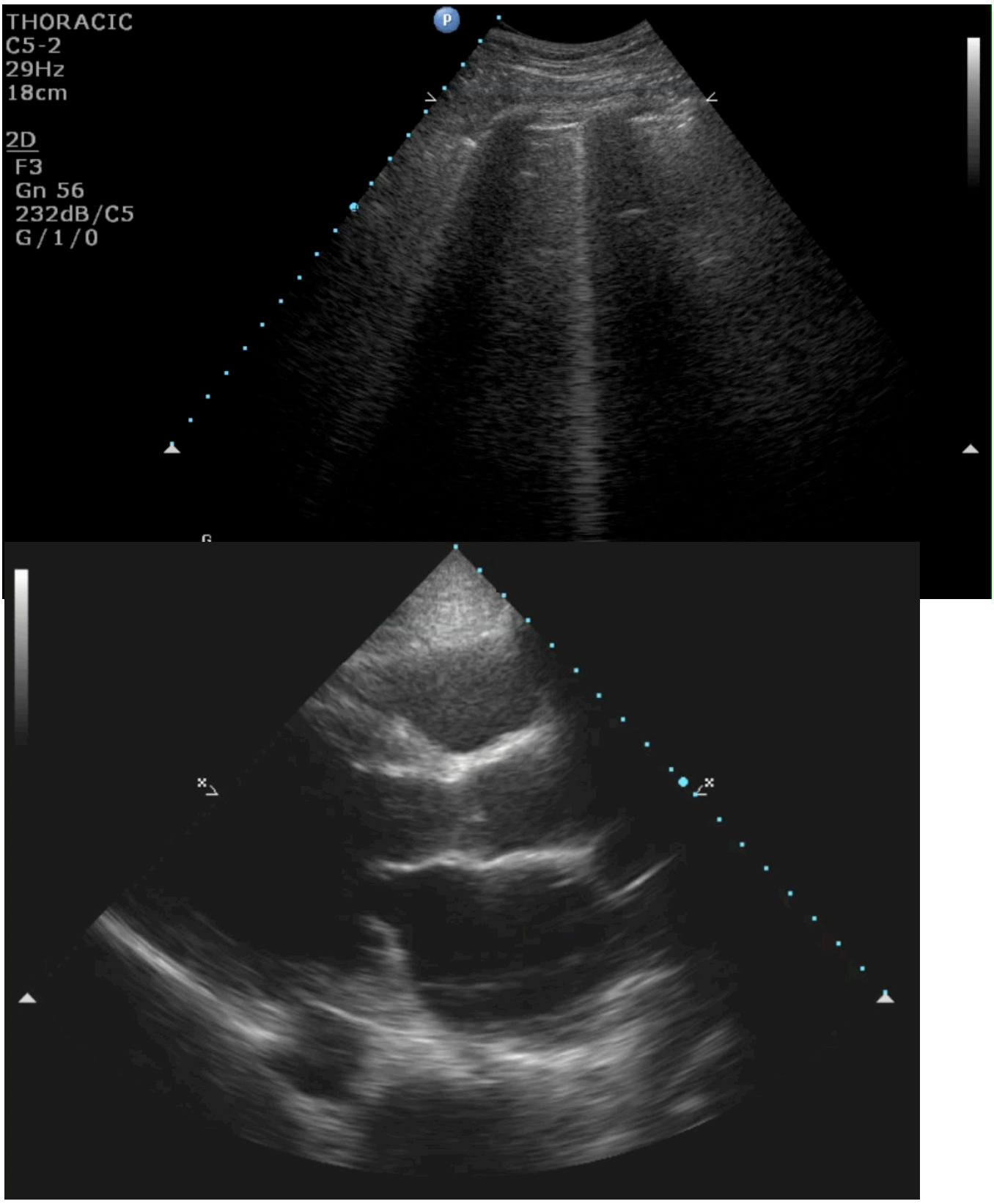

Figure 2