Foundations of Emergency Medicine
Foundations III: Guided Small Group Experience

Critical Care
Session 11: "Right Heart Failure"

- **Agenda and Learning Objectives**
  - Case Part I – Understanding RV Failure and pHTN (20 min)
    - What is right ventricle failure? How is it different from left heart failure?
    - What is pulmonary hypertension? How is it important to ED providers?
    - Understanding how right ventricle failure patients decompensate
  - Case Part II – Management of RV Failure (20 min)
    - Learn how to manage hemodynamic status in RV Failure
    - Discuss principles in resuscitation in management of the RV Failure patient
    - Discuss adjuncts available for the management of RV Failure
  - Case Concludes (10 min)
    - Review Session Teaching Points

- **Case Part I – Understanding RV Failure and pHTN (20 min)**

55yoF with is BIB EMS after a witnessed syncopal episode at home.

She lives with her sister who has accompanied her in the ambulance. She says her sister has a history of pulmonary hypertension and takes a medication called sildenafil. For the last 3 days, she has been sick with "the flu." She has been trying to rest but walking to the bathroom this morning, she collapsed and passed out causing the sister to call 911. She was not shaking, did not bite her tongue, did not lose her urine and came to after a "minute or so." She has been having lightheadedness with walking the past two days but this is the first time she has collapsed.

Her initial vital signs are: 38.9F, 132, 28, 65/32, 80% RA however she improves to 93% on 15L NRB.

She is awake, alert, and oriented to place and situation in the resuscitation bay. She reports she just felt "weak and lightheaded" then passed out. She says she has been sick for a few days, does not know if she had a fever (hasn't checked). Has not really been coughing, just "weak and achy" all over. She reports she has had on and off chest pain that "only last a few minutes" only when she walks or "works herself up too much" but goes away if she calms down and rests. That's been going on for the past month.

She has "never smoked a day in her life." She denies alcohol or illicit drug use.

She denies leg swelling, orthopnea, paroxysmal nocturnal dyspnea. She denies nausea, vomiting, but "hasn't been eating too good for the past few days." She denies abdominal pain, changes in urination, burning with urination, back pain, changes in bowel movements.

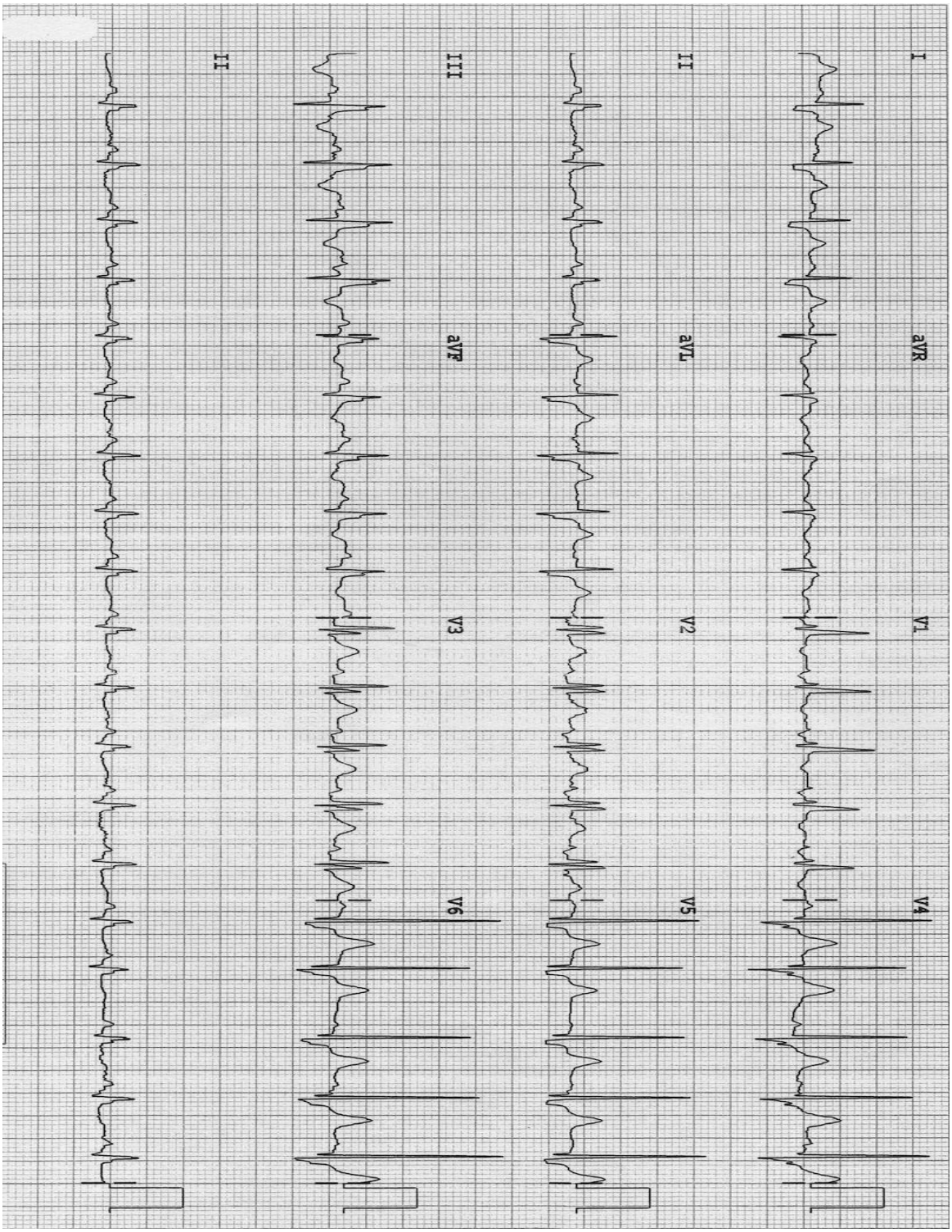
She has not recently had surgery. She has not been traveled anywhere or been immobilized for a long period of time.

She has no family history of early cardiac death or early myocardial infarction. She has no personal or family history of blood clots.

Exam

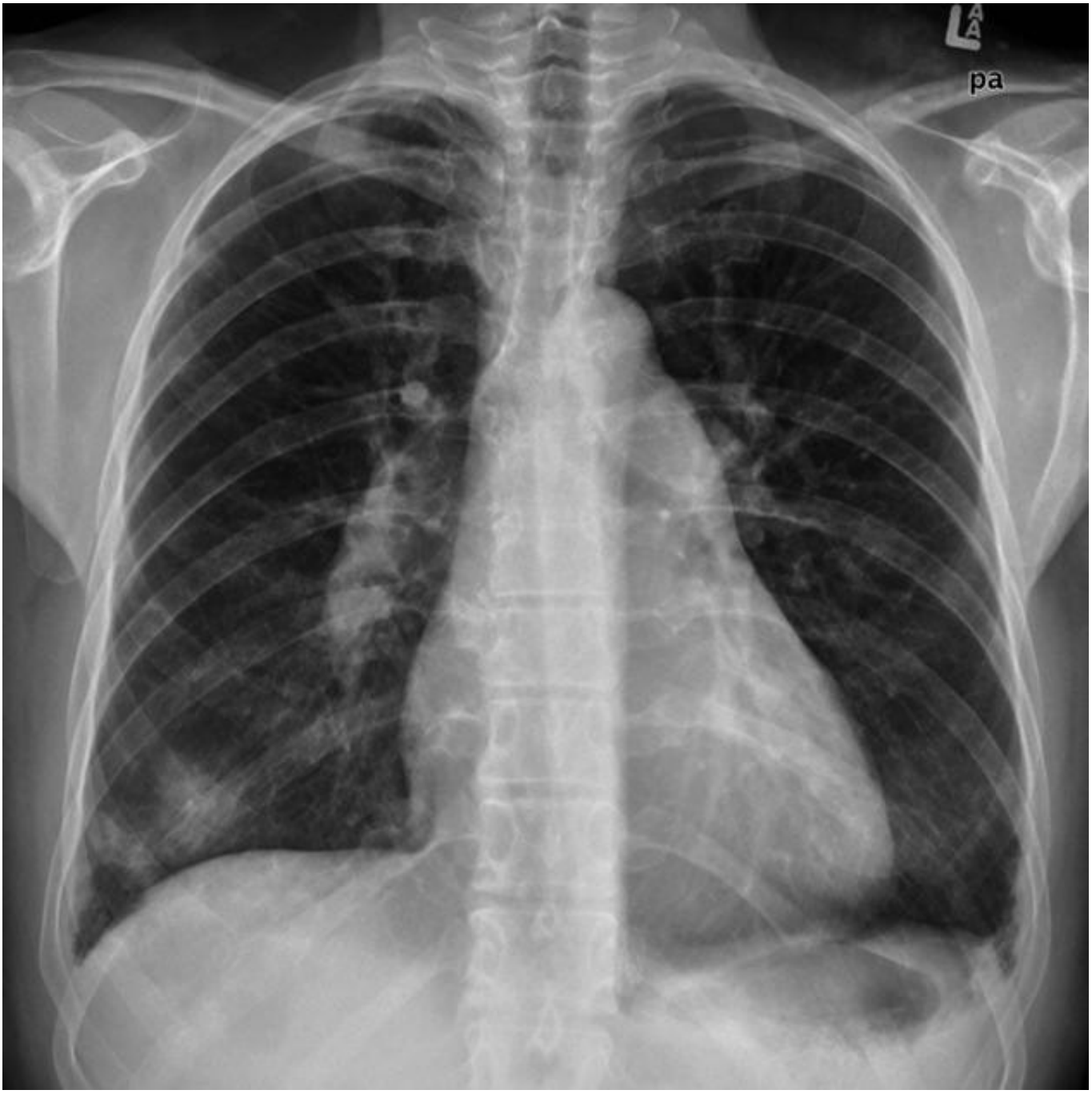
***General**: Diaphoretic, working to breathe, in distress*
***Neck**: Obese neck, unable to visualize JVD 2/2 to habitus*
***Cardiovascular**: Fast rate, regular rhythm without murmur*
***Pulmonary**: Tachypneic, bilateral equal clear breath sounds are present in full fields. No wheezes, no crackles, no rhonchi.*
***Abdomen**: Obese, but soft, nontender in all four quadrants. No rebound. No guarding.*
***Back**: No costovertebral angle tenderness bilaterally.*
***Extremity**: Large habitus, but no asymmetric swelling. No appreciable pedal edema.*
***Neuro**: Awake, alert, and oriented to place and situation. Gross motor and sensation intact in bilateral upper and lower extremities.*
***Skin**: There are no visible rashes.*

EKG (If asked, there is no old EKG to compare)



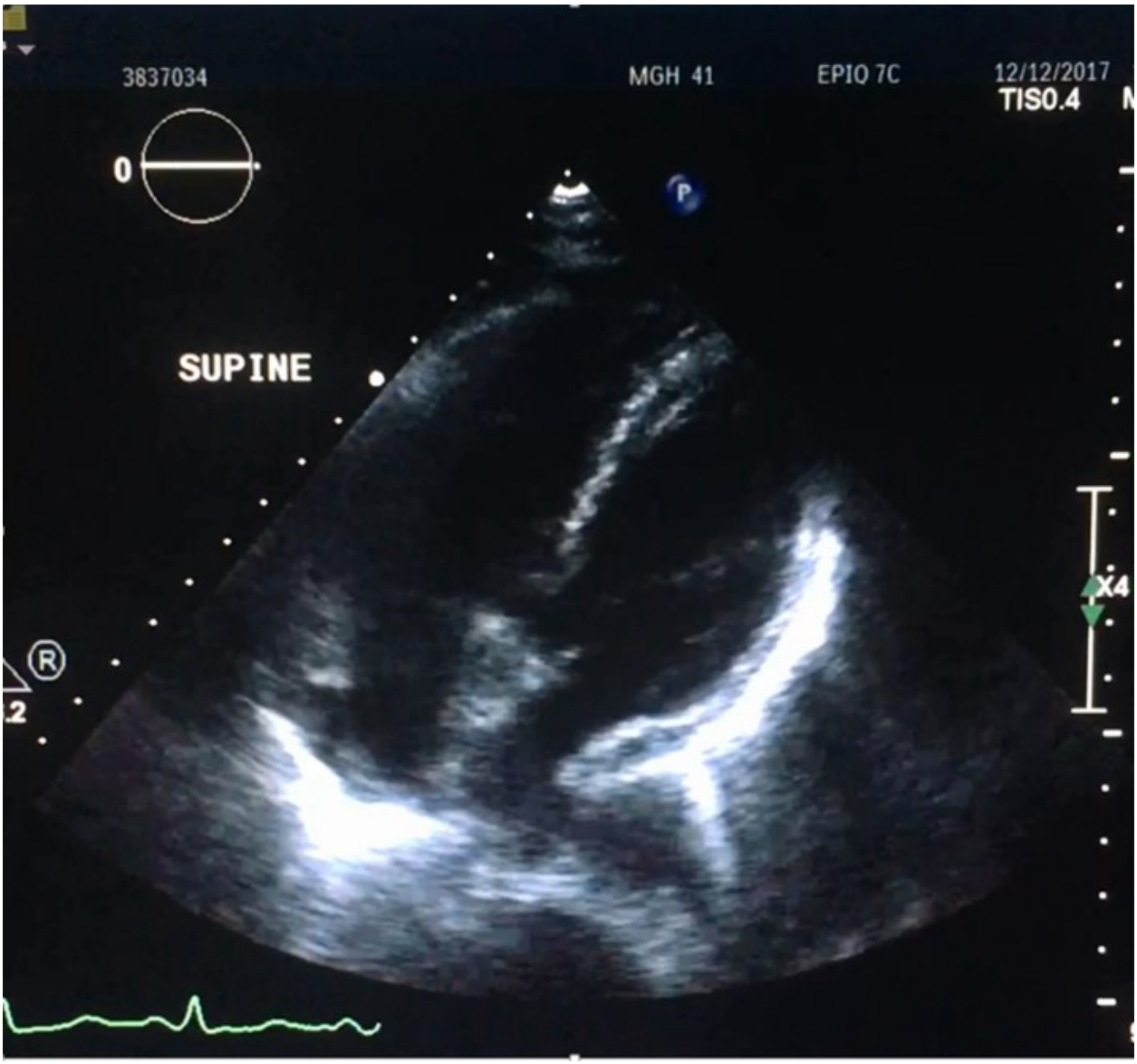
EKG Source: https://lifeinthefastlane.com/ecg-library/basics/vt_vs_svt/

Portable CXR



Source: Radiopaedia
https://radiopaedia.org/articles/pulmonary-hypertension-1

Bedside RUSH Exam (is otherwise normal)



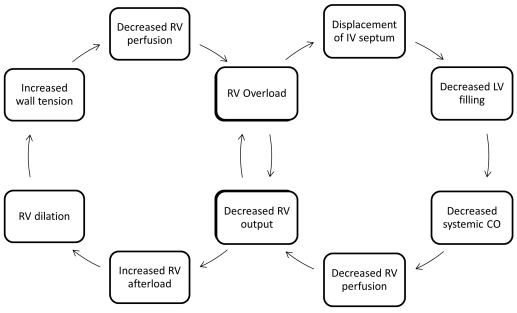
Echo courtesy of Dr. Susan Wilcox (Massachusetts General Hospital)

- **Discussion Questions with Teaching Points**
  - **Most providers would jump into resuscitation for a patient this ill without obtaining all of the history given but for education purposes, what is going on this with this patient? What is the diagnosis? What do the EKG, chest x-ray and echo show?**
    - This patient has a working diagnosis of severe sepsis/septic shock → however, there is a twist given her underlying pHTN
    - The EKG shows sinus tachycardia with a right bundle branch block
    - The chest x-ray shows an elevated cardiac apex due to right ventricular hypertrophy, prominent pulmonary outflow tract, and pruning of peripheral pulmonary vessels → it is suggestive, though not diagnostic, of pulmonary hypertension
    - Additional diagnostics will be needed to determine if the right heart strain is acute (ex. PE or RCA infarct) versus chronic (pulmonary arterial hypertension) versus acute on chronic (sepsis in a pulmonary hypertension patient) → the history above suggests the underlying right ventricle failure is more of a chronic process with a superimposed acute process (likely sepsis)
    - Point of Care Ultrasound will change your resuscitation of this patient
      - The RUSH exam is suggestive of right heart strain suggestive of right ventricle failure
      - The POCUS showing likely significant underlying right ventricle failure will change your resuscitation of this patient (as well as the work-up)

  - **What is right ventricle failure? How is it different from left heart failure? Need to review (briefly) a few principles from physiology….**
    - Right heart failure is defined as dysfunction of the right ventricle resulting in hypotension or other evidence of end organ hypoperfusion[2]
    - Unlike the left ventricle which is very strong and pumps blood through the entire body, the right ventricle is weak (1/6 the thickness of LV) and relies on pushing against a very low resistance to perfuse the pulmonary vasculature to promote gas exchange and fill the left heart
    - To manage the right ventricle failure patient properly, it is important to understand 3 physiology concepts of the right heart.
      1. Frank Starling concepts still apply to the right heart. Preload only helps improve cardiac output until a certain point, then will plateau and cause cardiac output to decrease. With tricuspid regurgitation, the overload may be more prominent
      2. Intraventricular Dependence. The left ventricle and right ventricle live within a fixed space – the pericardium. The higher pressures of left ventricle usually push the septum against the right which helps left ventricle filling and maintain ejection fraction. If RV failure is severe, this bowing may be reversed.
      3. RCA perfusion. The right ventricle is perfused in both systole and diastole unlike the left ventricle which is perfused only in diastole.

- **What is pulmonary hypertension? When should I consider it in the ED?**
  - Pulmonary hypertension is a hemodynamic condition defined as a mean pulmonary artery pressure by right-sided heart catheterization of at least 25 mm Hg at rest
  - Can either be primary (without a discernable cause) or secondary from diseases such as COPD, collagen vascular diseases or left sided cardiac disease
  - Increases in pulmonary vascular pressures (either acute or chronic) ultimately lead to increase RV load and RV failure
  - 20-30% of patients with unexplained exertional dyspnea with a negative workup for acute pathology will end up being diagnosed with pulmonary hypertension and right ventricle failure[3,4]
  - One in five patients with pulmonary arterial hypertension (Group 1) will have symptoms for 2 years before being diagnosed and when they are, it is at a late stage of disease which correlates to poor outcomes[5,6]
  - The ED provider should be on the look-out for possible undiagnosed right ventricle failure/pulmonary hypertension in those patients with unexplained exertional dyspnea especially as it can significantly change management in the ED especially in terms of fluid resuscitation
  - If a patient already carries a diagnosis of pHTN this will likely impact your resuscitation!

- **How do patients with pHTN decompensate? What is the "right heart spiral of death"?**
  - In a patient with known pulmonary hypertension, the physiology is very tenuous at baseline. An acute stressor (ex. sepsis) can trigger a quick **spiral towards cardiopulmonary collapse → the goal is to reverse the spiral before its too late!**
  - An increase in RV afterload (due to some stressor) → worsen RV function → worsen perfusion → increase ischemia → further worsen function → increase pressure pushing on the septum → decrease cardiac output → worsen ischemia → worsen function
  - Cardiovascular collapse and arrhythmias are the two most common causes of mortality



(Courtesy of Dr. Susan Wilcox)

Case Part II – Resuscitation (20 min)

Based on the vital signs, the nurse has activated a "sepsis" alert on this patient prompting a rapid protocol of drawing blood cultures, hanging fluids, and starting antibiotics. The nurse begins to hang a 30cc/kg bolus of crystalloid fluids.

- **Discussion Questions with Teaching Points**
  - **This patient is in shock but should she get fluids?**
    - The goal of fluids in septic shock is to help improve end organ perfusion
    - Given her RV Failure and pHTN, fluids in this patient may not help perfusion and may make the RV Failure worse, thus worsening end organ perfusion
    - The physical examination in patients with pulmonary hypertension is often unreliable for determining volume status[1]
      - Ultrasound, CVP and pulmonary catheters all have a role, but have downsides
    - The problem acutely with right ventricle failure patients is rarely a problem of preload but rather a problem of increased afterload
    - If you as a provider determine the patient may benefit from additional preload based on history or physical (though remember this is unreliable!), use small boluses (no more than 250cc at a time) with very close monitoring
    - **If unsure about fluid status in a patient with right ventricle failure (which you generally should be), assume they are fluid overloaded because most of the time they are[1]**
    - Too much fluids can increase RV pressures and trigger the right heart spiral of death
    - Patients who are volume overloaded and not hypotensive may benefit from diuresis to improve cardiac output as it will decrease the RV stress from the overload (Frank Starling) and improve LV output (intraventricular dependence)
      - In advanced/severe disease, the pulmonary pressure becomes "fixed" so diuresis does not improve cardiac output and can make hypotension worse

  - **So… if not fluids, I guess I need to start a vasopressor to improve perfusion. Which one should I select?**
    - An ideal vasopressor would improve cardiac output, decrease pulmonary vascular resistance, while at the same time, not cause any arrhythmias, ischemia, hyperlactatemia, or hypercoagulability → there is no agent to achieve all these goals but some agents are better choices than others
    - Bottom line is that norepinephrine (at doses < 0.5 mcg/kg/min) or epinephrine are viable choices (choice is largely dependent on how the critical care MD trained as there is little data) and phenylephrine and dopamine are bad choices
    - Can also consider the addition of vasopressin
    - Some information about each pressor:
      - Norepinephrine
        - Norepinephrine improves the systemic vascular resistance and will improve coronary perfusion and perfusion to other organs

        - Norepinephrine also has a β-1 effect which provides a small amount of inotropic support which increase cardiac output
        - Some may argue against norepinephrine because it is an alpha agonist and that works to constrict the pulmonary vasculature and increase the pulmonary vascular resistance → studies have only shown this affect with norepinephrine at **higher doses** (greater than 0.5 mcg/kg/min) and some other small cohort studies which has shown norepinephrine has lowered the PVR/SVR ratio[2]
        - A definitive trial showing the impact of norepinephrine on PVR and a comparison trial against other vasopressors has yet to be done
    - Epinephrine
        - Epinephrine and the pure β receptor properties are also used as a potential first line agent for right ventricle failure
        - Increases cardiac output by providing inotropic support
        - Epinephrine also has no impact on the pulmonary vascular resistance but increases the systemic vascular resistance thus lowering the PVR/SVR ratio
        - Some studies showed it increases the mean pulmonary artery pressure[7]
        - Epinephrine has been associated with increased tachyarrhythmias which needs to be avoided in right ventricle failure patients → treat these aggressively (usually with electricity)
        - Epinephrine is often used for right ventricle failure after cardiac pulmonary bypass surgery in many cardiothoracic surgery ICUs and is favored by many cardiac anesthesiologists
    - Vasopressin
        - Works on V1 receptor and favorable because it increases systemic vasoconstriction but also dilates the pulmonary vasculature (through a NO based mechanism)
        - Reduces the PVR/SVR ratio
        - May also have a diuretic effect and result is less tachycardia compared to norepinephrine and epinephrine
        - Downsides:
            - It is not titratable
            - Vasopressin may cause bradycardia at higher doses 0.4 U/min (possible direct myocardial effects - coronary vasoconstriction)
- Vasopressors to generally avoid in right ventricle failure
    - Phenylephrine
        - Generally avoided because it increases pulmonary vasculature resistance through direct alpha stimulation
    - Dopamine
        - Generally avoided because of a strong association with an increase in tachyarrhythmias
        - Increases the PVR/SVR ratio

- **Stepping back, what other general principles should I keep in mind for the resuscitation of the Right Heart Failure patient? What else should I be looking for and doing for these patients? What additional etiologies contribute to their ultimate death?**
  - The two most common reasons why these patients arrest are arrhythmias and cardiopulmonary collapse.
  - From a physiology standpoint, the goal is to reduce pulmonary vascular resistance which will help reestablish the trans septal gradient which will help perfusion
  - This includes **avoiding intubation as much as possible** as the positive pressure decreases venous return, increases pulmonary vascular resistance, and can precipitate cardiopulmonary collapse
  - This also means using non-invasive positive pressure (BiPAP) modalities with caution as it has the same effect
  - High flow nasal cannula may be a first line non-invasive option to improve oxygenation in a crashing patient without too much of an increase in positive pressure
    - HFNC has been shown to improve oxygenation but may also improve hypercapnia some
    - It is also required in order to deliver NO
  - Inotropes may also be helpful but usually results in systemic hypotension (secondary to decreased SVR) and will need to be used alongside a vasopressor (see below)
  - In general, inhaled agents (if available) are preferred in these patients over systemic agents (theory, some small case studies, but unproven → achieves effects with less systemic side effects)
  - Correct any metabolic abnormalities such as hypocalcemia, sepsis, acidemia, etc which may be further stressors on the physiology
  - It is important to recognize arrhythmias early and pursue cardioversion as these patients' physiology is very tenuous → it is wise to treat all these patients as unstable
  - β blockers and calcium channel blockers may make the problem worse!

- **This patient is on sildenafil at home. What do I do about that? What about medications that are given continuous IV infusion?**
  - If they are on pulmonary HTN medications, it is important to keep them on their regime → otherwise, they are at risk for rebound pulmonary HTN which can trigger a spiral towards hemodynamic collapse
  - If there is an indwelling line with a continuous prostanoid infusion (ex. Treprostinil), the infusion must continuously run with changes made in conjunction with the patient's pulmonary hypertension specialist → do not discontinue!
    - A common scenario is for these patients to presents to the emergency department with a catheter occlusion or pump malfunction → you should temporarily infuse the medication through a peripheral IV catheter until a new central line is in place or the issue is resolved

- Patients may present to the emergency department with side effects of these medicines, but the medication should only be changed in junction with the patient's specialist.

- **Besides vasopressors, what other treatments are available?**
  - **Generally, if available, inhaled agents are preferred over IV (see below for a discussion of specific agents) due to less systemic side effects**
  - Inotropes
    - Best agent would be inhaled milrinone if available, otherwise IV milrinone is also an option
      - For IV milrinone, the recommended dosing is 50mcg/kg over 10 minutes followed by a maintenance dosing of 0.1-0.75 mcg/kg/min (starting at 0.1 mckg/kg/min with very slow titration) → there is debate in the literature of the need for a loading dose
      - Inhaled milrinone is still very new and the dosing is controversial
        - There is a registered trial of dosing with the NIH being conducted at Vanderbilt University and the University of Kansas hoping to answer this question with results expected in 2019 or 2020
        - Published pilots have reported safety with 5 mg over 5 minutes in 10L/min of oxygen with no discussion of maintenance [8,9]
      - Milrinon (especially IV) usually results in systemic hypotension and will need to be used alongside a vasopressor (vasopressin is often used as first line as an adjunct agent)
      - Nebulized (versus systemic) milrinone has been associated with less systemic hypotension and less VQ mismatch in cardiothoracic ICU settings[10,11]
    - Low dose dobutamine may help RV performance with pulmonary vascular dysfunction because of the β 1 Effect
      - May have a synergistic effect with nitric oxide in patients with pulmonary hypertension[12]
      - Usually results in systemic hypotension (secondary to decreased SVR) and will need to be used alongside a vasopressor
      - Has been associated with tachyarrhythmias at higher doses (though less than dopamine)
  - Inhaled Nitric Oxide
    - Inhaled nitric oxide acts as pulmonary vasodilator to reduce pulmonary vascular resistance
    - Studied to improve RV failure secondary in the ARDS population, acute PE, pulmonary artery hypertension, right RV infarction
    - Though not specifically studied in the ED population, may be beneficial in acute resuscitation for the correct patient
    - Dosing starts at 5 ppm up to 10 ppm

- If you choose to initiate inhaled NO in the ED, do not abruptly discontinue it → defer to your colleagues in the ICU as there is a risk of rebound pulmonary HTN and RV Failure without weaning
- Downsides: Inhaled NO is expensive and needs monitoring for toxicity (ex. Methemoglobinemia)

▪ Prostanoids
- Epoprostenol (Flolan) is the most common inhaled agent likely available in your hospital. You may have to call your anesthesiologist or your OR to get it
  - Also works to reduce PVR
  - Short term equivalence to inhaled NO though no clear head to head trial favors one agent over the other
  - Should only be used with PA catheter monitoring real time RH pressures with the consultation of the PH Specialist

- **My patient is crashing in front of me and she appears to be peri-arrest. What other options do I have depending on my institution?**
  - Mechanical Support
    - Usually would mean ventricular assist devices (Impella or less often IABP) or venous-arterial extracorporeal membrane oxygenation (ECMO)
    - Considered in patients with right ventricle failure refractory to volume management, vasopressors/inotropes, and pulmonary vasodilators as a bridge to transplant or possible destination therapy

- **Changing the scenario, my patient has known pulmonary hypertension but arrives with normal vital signs and feels better. Can I send them home?**
  - An important test before discharge is an ambulation challenge with pulse oximetry
  - If they are able to walk around in the emergency department without desaturation or significant worsening of symptoms, then they may be able to be discharged to follow up closely with their pulmonary hypertension specialist.[1]
  - Otherwise, they need admission for likely repeat echocardiogram, medication titration, or mechanical intervention
  - If the patient had symptoms of exertional syncope or near syncope, admit them for further evaluation!

❖ **Case Concludes (10 min)**
- *The patient is stabilized in the ED with a combination on inhaled milrinone and vasopressin and norepinephrine. The patient was able to avoid intubation and transferred to the ICU for further management.*

- **Case Teaching Points Summary**
  - Diagnosing/Considering Right Heart Failure
    - Consider pulmonary arterial hypertension in a patient with unexplained, progressive, chronic exertional dyspnea
    - Bedside ultrasound can help guide your resuscitation → don't forget your RUSH exam and look for RV dysfunction!
    - Right heart failure exacerbation triggers a spiral of death → it is important to understand the physiology to offer appropriate treatment
    - Knowing if the right ventricle failure is acute, chronic or acute on chronic will help the provider know how much the right heart can handle

  - Resuscitation of the Right Heart Failure Patient
    - If you are not sure about volume status, assume the right ventricle failure patient is volume overloaded because most of the time they are. **Be very judicious about fluids!**
    - There is no perfect vasopressor to meet all the hemodynamic considerations but first line agents are norepinephrine and epinephrine → can consider adding vasopressin as a second line
    - Keep at lower doses to avoid side effects and may consider adding a second agent before maximizing a single agent
    - Inotropes (milrinone or dobutamine) may help improve cardiac output in right ventricle failure but likely will need a concurrent vasopressor
    - Pulmonary vasodilators such as inhaled nitric oxide and prostacyclins (Flolan) may be beneficial in an acute resuscitation setting → however, no there is no data from the ED supports this to date
    - Inhaled agents (ie milrinone and Flolan) are generally favored over systemic agents
    - If the patient presents on pulmonary hypertension medications, keep the patient on those medications and only change in conjunction with the patient's specialist
    - Check the pump and line if they are on a chronic IV maintenance medication
    - Consider cardioversion in any new arrhythmia → consider them unstable!

- **Facilitator Background Information**

Right Heart Physiology in Failure

The pulmonary circulation is a high flow, low pressure (normal 15 mmHg), low resistance system. The right ventricle has a thin wall which pumps against thin pulmonary vessels and a large number of un-perfused vessels. The right ventricle can acutely tolerate large changes in preload but does not tolerate changes in afterload ie PVR (even small ones).

The right heart fails acutely most commonly because of an increase in pulmonary vascular resistance (afterload) from an insult such as PE, pneumonia, AMI or sepsis among others. An increase in pulmonary vascular resistance (PVR) will push against the RV and reduce RV stroke volume. The increase in right ventricle end diastolic volume will cause RV dilation and failure (Frank Starling) and will increase tricuspid regurgitation creating a cycle further reducing cardiac output. This creates a spiral that worsens upon itself. Another important concept to understand is the interventricular dependence between the right ventricle and the left ventricle. At baseline, the higher pressures of the LV push the septum against the LV allowing LV filling and LV systolic function. In right ventricle failure, increased volume of the right ventricle will push on the intraventricular septum into the left ventricle. This further decreases LV filling and also reduces overall cardiac output.

Unlike the left ventricle, the right ventricle is perfused both during systole and diastole. However, as the pressure in the RV rises closer to the aortic root pressure, the gradient between the two no longer exists making the RCA only fill during diastole (when the aortic pressure is still higher diastole RV pressure). This results in under-perfusion of the RCA leading to further RV ischemia. This ischemia causes worsening RV contractility and thus worsening RV failure, continuing the spiral of worsening failure.

Types (and causes) of Right Heart Failure

There are multiple frameworks to classify right ventricle failure. For the emergency provider, it may be best to classify based on acute causes, chronic causes and acute on chronic (exacerbation) as this conceptually may be the most relevant to emergency practice.

Acute Failure

The most common acute causes of right ventricle failure are massive PE and RV infarction, which requires management of the right ventricle failure as well as management of the acute cause (thrombolytics, cath lab activation etc). Also, the emergency provider would be wise to consider less common alternative causes of acute right heart strain, such as acute tricuspid regurgitation secondary to endocarditis in an intravenous drug user.

Chronic Failure and pHTN

Chronic right failure results from gradual pressure increases in the pulmonary vasculature (pulmonary hypertension) resulting in a gradual increased load to the right heart resulting in right heart hypertrophy and decreased function of the right heart. Pulmonary hypertension is a hemodynamic condition defined as a mean pulmonary artery pressure by right-sided heart catheterization of at least 25 mm Hg at rest. It can either be primary, without a discernable cause, or secondary from diseases such as COPD, collagen vascular diseases or left sided cardiac disease. Increases in pulmonary vascular pressures (either acute or chronic) ultimately lead to increase RV load and RV failure

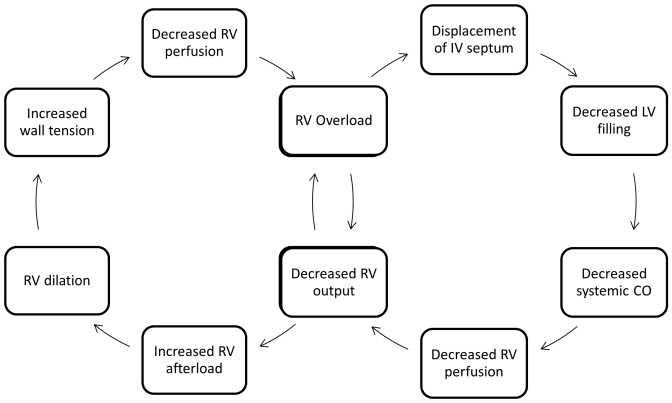
Pulmonary hypertension is never formally diagnosed in the ED as the diagnosis requires a right heart catheterization but certain findings can be suggestive. The most common presenting complaint is progressive, unexplained dyspnea. Often patients have symptoms for years prior to be diagnosed leading to end-stage diagnoses with poor overall outcomes. Consider this diagnosis in patients with unexplained exertional dyspnea especially with risk factors for secondary disease processes.

Acute on Chronic Causes

This is the scenario most likely to be encountered by the average emergency provider. This is a patient with known right ventricle failure presenting with an acute stressor to their physiology. Common stressors include: sepsis, metabolic (ex. DKA), MI or rapid changes in volume status.

The Right Heart Failure "Spiral of Death"

Given the above physiological considerations, patients with underlying RV Failure are extremely difficult to manage when acutely ill. This is because they enter what is commonly called the right ventricle failure "spiral of death" ending in cardiopulmonary collapse. This, often with concurrent arrhythmia, is the most common cause of mortality in these patients. The spiral of death often starts with an acute increase in RV afterload (ie PVR) due to an outside stressor (ie sepsis, MI, DKA etc). This increase in PVR leads to worsening RV function due to falling off the Frank-Starling curve which leads to even worsening RV function, worsening RV perfusion due to right sided coronary under-perfusion, increased RV ischemia, worsening LV functioning and systemic hypoperfusion due to bowing of the inter-ventricular septum into the LV leading to systemic hypoperfusion. This in turn decreases RV filling and worsens function also. This spiral feeds into itself and will quickly lead to cardiopulmonary collapse if not intervened upon.



Right Heart Spiral Towards Cardiopulmonary Collapse – The "Spiral of Death"
(Provided by Dr. Susan Wilcox)

Resuscitation and Management of the Right Heart Failure Patient

Fluid management in these patients is extremely difficult and EPs should avoid large fluid boluses. These patients are often in fact fluid overload (or have fluid in the wrong places) and if given typical sepsis fluid boluses (30 cc/kg) will further decompensate. It is generally recommended not to proceed with significant volume resuscitation without close monitoring though there is significant argument about the best way of monitoring these patients (in short, there is no great way to monitor these patients):

Ultrasound. There is a lot of debate amongst the ultrasound community, from outflow indexes to color artery flow with respiratory variation about different techniques to measure fluid responsiveness. There is no universally agreed upon gold standard. Further discussion is outside the scope of this lesson.

Central Venous Pressure – Trends in CVP may be helpful, but a single value will unlikely provide much diagnostic benefit as higher baseline right heart pressures and the presence of likely tricuspid regurgitation will artificially increase the CVP regardless of volume status.

Pulmonary Artery Catheter ("Swan"). The need for one is debated amongst critical care experts, but for right ventricle failure patients, the data can be extremely valuable to guide management. Critics will argue pulmonary catheters have not changed clinical outcomes, and there have been complications. Also, it is unlikely available as an option quickly in the emergency department in most centers to guide acute resuscitation.

In an unstable right ventricle failure patient, the emergency provider should generally avoid significant fluid administration and should be quick to reach for vasopressor and inotropic support. There is no perfect agent. Norepinephrine (in doses < 0.5 mcg/kg/min) and Epinephrine are generally preferred and dopamine and phenylephrine are not recommended.

Table 5 Pulmonary vascular properties of vasoactive agents

	CI	PVR	SVR	PVR/SVR	Tachycardia	Renal[a]/metabolic
Vasopressors				Dose related		
NE	+	+	++	+/-	+	Lactic acidosis
PHE	-	++	+	+	-	-
Low-dose AVP	+/-	+/-	++	-	-	Diuresis ++
Inotropes						
Dobutamine	++	-	-	-	+	
<5 μg/kg/min						
Dopamine	+	+/-	+	+	++	Natriuresis
Epinephrine	++	-	++	-	++	Lactic acidosis
Inodilators						
PDE IIIs	++	-	-	-	+/-	-
Levosimendan	++	-	-	-	-	-

AVP, arginine vasopressin; NE, norepinephrine; PDE IIIs, phosphodiesterase inhibitors; PHE, phenylephrine. [a]Renal blood flow is likely to improve with increased cardiac output and systemic blood pressure with all agents.

Source: Price et al. Critical Care, 2010.[2]

Inotropes may also help with forward flow but will likely need to be used in conjunction with vasopressors because they can cause systemic hypotension. The best agent would be inhaled milrinone if available, otherwise IV milrinone is also an option. Milrinone is a phosphodiesterase (PDE) III inhibitor which improve RV performance and reduce PVR in patients with pulmonary vascular dysfunction (increasing cAMP so indirectly increasing inotropy while also resulting in vasodilation – notably pulmonary vasodilation)**.** Milrinone is mentioned because it is the most commonly used and widely studied but other PDE III inhibitors such as enoximone and amrinone also have similar properties**.** PDE III inhibitors may result in tachycardia and will result in tachyphylaxis if used for a prolonged period of time**.** Low dose dobutamine may also be an option to improve RV performance because of the β-1 Effect and may have a synergistic effect with nitric oxide in patients with pulmonary hypertension[12]. However, this needs to be used with caution as dobutamine has been associated with tachyarrhythmias at higher doses (though less than dopamine).

Inhaled nitric oxide, prostacyclins and PDE-5 inhibitors are all options to reduce the pulmonary vascular resistance. Inhaled nitric oxide has been studied in the acute setting and seen to improve RV failure secondary to ARDS, acute PE and right RV infarction but is expensive, not readily available, needs to be titrated and at toxic doses may lead to methemoglobinemia. There are experimental protocols ongoing for PE, ARDS for the use of Epoprostenol (Flolan) in an acute setting, which may be beneficial, but a definitive study has not proven the efficacy of its use. As a last resort, emergency mechanical support (ex. ECMO) may have a role to support patients as a bridge to cardiopulmonary transplant.

A key point that should be emphasized is that **every effort should be made to avoid intubation in these patients**. Acute hemodynamic collapse is well described in these patients likely from a combination of factors: decreased venous return from positive pressure ventilation, decreased SVR from sedation medication, increased PVR further decreasing cardiac output. However, the risks of intubation need to be closely weighed against increasing PVR from persistent hypoxia, hypercarbia, and metabolic acidosis (which feeds into the spiral of death) and ultimately intubation might be the best choice. BiPAP may be an option; however, remember that NIV positive pressure will also decrease venous return which may further impair cardiac output and worsen perfusion. High flow nasal cannula may be the best first line non-invasive option in crashing patient that helps improve oxygenation but with only a minor increase in positive pressure. If intubation is unavoidable, it is recommended to use lung protective strategy with low tidal volumes and focus on keeping plateau pressure low (high intrathoracic pressure mechanically compresses the pulmonary vessels and further increase the PVR). Be prepared for hypotension and possible cardiovascular collapse during intubation; have push dose epinephrine and/or a norepinephrine/epinephrine drip up and hanging before you start and prepare your team! Intubation, and positive pressure ventilation, further strains the right heart and will likely precipitate the need for further support from a vasopressor or inotropic agent (or both) so be prepared.

Some final points (in case you haven't had enough yet)! It is import cardiovert any tachyarrhythmias early (such as Afib with RVR). Given the tenuous physiology (and because in these patients the LV often depends on the atrial kick for filling), it is wise to consider these patients unstable even if they appear stable initially. β-blockers and calcium channel blockers may further impair right ventricular function and should generally be avoided. [13]

Finally, PDE-5 Inhibitors (such as sildenafil) are generally not used in an acute setting but are used for outpatient management. If patients arrive on these medications it is important to keep them on their scheduled regiment. If they are on a continuous infusion, check that the infusion is working (pump and line)!

Right sided heart failure is complicated but it is important that emergency physicians have a basic understanding of the physiology and how the resuscitation of these patients may be different so as to avoid (or abort) the right sided heart failure "spiral of death". The bottom line for resuscitation is that these patients are often volume overloaded and tolerate large fluid boluses extremely poorly. EPs should reach for pressors quickly (either epinephrine or norepinephrine) and consider other adjuncts such as milrinone (inhaled or IV), nitric oxide or Flolan. Avoid intubation if possible but weigh this with the understanding that respiratory distress, hypoxia, hypercarbia and acidosis also worsens these patients' physiology. If intubating, anticipate cardiovascular collapse and prepare as best you can. Finally, ensure patients are maintained on their home medications regiments (including IV infusions) and reach out to their doctors early and often. Ultimately, a healthy respect for the tenuous nature of these patients' underlying physiology, and an understanding of how to manage it, can save their lives!

- **References**
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