Bouncebacks! Critical Care

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BOUNCEBACKS!
Critical Care

Bouncebacks! has done it again! Mike et al. bring a suspenseful and relatable series of cases...this time with a critical care focus. These cases grab your attention much like a "can't put down" thriller, the original documentation (with time stamps to boot) literally transport you to the bedside. Regardless of your level of training, Bouncebacks! Critical Care highlights and instructs on common pitfalls made along the way.

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I have been reading and recommending the Bouncebacks! series to colleagues and residents since the release of the first edition. The cases contain invaluable "back-to-the-basics" lessons on how to perform an exemplary H&P, how to think critically about diagnostic and management plans, and how to document responsibly and thoughtfully. The innovative format of this edition forces the reader to consider challenging clinical questions as they work through the case. The accompanying expert commentaries transport the reader into a virtual high quality Educational Grand Rounds environment. Highly recommended regardless of level of training!

Ramin Tabatabai, MD, MACM
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Bouncebacks is back, baby!!! This is a must read for any practicing emergency physician, at any point in their career. The authors provide story pauses at important decision points as possible management strategies are reviewed in a literature-based fashion, all while remaining concise and easily readable.

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SECTION C – CARDIAC/ VASCULAR

Chapter 11: A 63-year-old man with shortness of breath after a fall

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SECTION C / CHAPTER 11

A 63-year-old man with shortness of breath after a fall

The patient's story:
Ben Russ is a 63-year-old attorney. He has been successful in his career but has been unlucky with his genetics; despite maintaining his weight, exercising regularly, and stopping smoking in his mid-40s, he suffered his first deep vein thrombosis (DVT) at the age of 52 and was found to have factor V Leiden.

On a warm fall day, while standing on a ladder to clean his gutters, he falls onto his left side. His wife rushes outside, finding him still laying on the ground. She helps him to a sitting position, but after 30 minutes of significant pain with breathing, they decide he needs medical attention, so he is helped into the car, and his wife drives to the local emergency department. He is found to have 3 fractured ribs and is admitted for pain control, but during the night develops CO_2 retention, becomes confused and is intubated by the hospitalist physician.

He improves, is extubated, and is eventually discharged home, but only days later he is again short of breath. Waking from sleep with significant respiratory distress, his wife calls 911 and he is taken to a larger, regional hospital where he is diagnosed with pneumonia and a congestive heart failure (CHF) exacerbation. He undergoes a heart catheterization and is found to have 95% occlusion of the right coronary artery (RCA) which is stented. He is discharged in "good" condition, but six days later he is again experiencing chest pain and shortness of breath. EMS is activated.

ED VISIT #3

CC: Chest pain/shortness of breath

HPI (PA-C) (19:41): Patient is a 64-year-old male with history of hypertension, diabetes, DVT, COPD, atrial fibrillation, CHF and factor V Leiden. His recent history is notable for a fall resulting in multiple rib fractures requiring intubation for hypoventilation. He was then hospitalized 2 days later for shortness of breath and found to have pneumonia and pleural effusions for which was started on Levaquin. He also underwent cardiac catheterization and underwent PTCA of his RCA. He did require Foley placement for urinary retention. The patient was discharged from this hospital 6 days ago after being admitted for roughly 5 days. He is on Coumadin. Had an INR checked earlier this week and it was over 5. He was told to hold his Coumadin and reduce the dosage. He was discharged to home 6 days ago.

He returns today with chest pain/shortness of breath which worsened today. He states he feels dizzy and lightheaded. Has been nauseated but no vomiting. Family believes he is still on Levaquin for pneumonia. He denies any abdominal pain. States his blood sugars were in the 400s today. Normally poorly controlled but usually in the 200s. No fever, blood in urine/stool bruising, rash, extremity pain, paresthesias. He has not had his INR rechecked.

PMH:
Atrial fibrillation
CHF (congestive heart failure)
COPD (chronic obstructive pulmonary disease)
DM (diabetes mellitus)
DVT (deep venous thrombosis)
Factor 5 Leiden mutation, heterozygous
HTN (hypertension)
Macular degeneration
Recent rib fractures

PSH:
Stent in branch of right coronary artery
Gunther Tulip Vena Cava Filter
Ankle
Knee

Medications: Atorvastatin, budesonide-formoterol inh, metoprolol, montelukast, tamsulosin, tiotropium inh, colace, Januvia, klor-con, Lantus insulin, nitrostat 0.4mg PRN, Plavix, aspirin 81mg, ferrous sulfate, warfarin 4mg QD
SH: No smoking or alcohol

PE (PA):

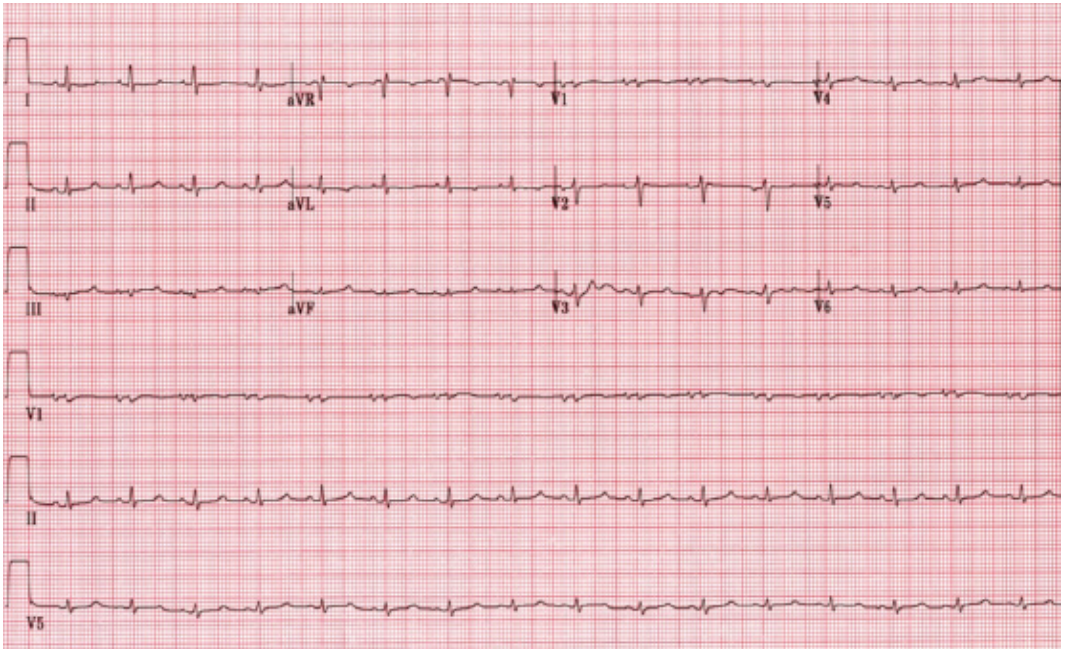
Vitals:						
Time	**Temp (F)**	**Pulse**	**Resp**	**Syst**	**Diast**	**Sat**
19:39	**95.7**	**121**	**18**	**78**	**58**	**97% (3L NC)**

CONSTITUTIONAL: Alert and aware, chronic ill-appearing appearance, holding an emesis bag which is empty.
RESP: Normal chest excursion with respiration; breath sounds clear and equal bilaterally; no wheezes, rhonchi, or rales.
CARDIO: Regular rhythm, without murmurs, rub or gallop, distant heart sounds.
ABD: Non-distended; non-tender, soft, without rigidity, rebound or guarding.
SKIN: Normal for age and race; warm and dry; no apparent lesions
EXT: Chronic venous stasis changes.

MDM (MD @ 20:16): I discussed the case with [the PA who] saw the patient, spoke with him and examined him. Lungs are clear with equal breath sounds. Heart tones are distant. Abdomen is soft. No pulsatile abdominal mass but does have bilateral lower extremity edema which appears chronic. The patient had chest pain and shortness of breath during the day today. EKG shows low voltage QRS.

Author comments (Weinstock):

The ECG below is a reproduction but is not the actual patient's ECG. Note our patient's heart rate is 121 bpm but the ECG below shows a slower rate.



Author comments (Weinstock):

We are at the bedside of a hypotensive, tachycardic, anticoagulated patient with a concerning ECG who has just arrived per EMS. We are at:

DECISION POINT A – YOU ARE FACED WITH 4 QUESTIONS:

1. **What is the differential diagnosis?**
2. **Which tests should be done to start the work-up?**
3. **How should the hypotension be managed initially? How does the history of CHF change the indication for IV fluids?**
4. **How does the history of an elevated INR play into the ddx?**

Q#1: What is the differential diagnosis?

This patient is critically ill based on his presenting vital signs; HR 121 and BP 78/58. The differential diagnosis is broad given his history of chest trauma, heart catheterization and two recent hospitalizations during which he was immobilized and intubated. I recommend starting by organizing a differential based on his recent history.

Trauma:

The remote history of thoracic trauma and rib fractures puts him at risk for bleeding, which could be delayed given his recent history of elevated INR. Possible sites of bleeding are pleural (hemothorax), pericardial and intraperitoneal, particularly if this is a delayed presentation of splenic injury. Intubation in the setting of torso trauma and rib fractures also places him at risk for pneumothorax, which causes RV dysfunction and shock when tension pathophysiology is present.

Cardiac cath/Possible recent MI:

He had a recent cardiac catheterization with PTCA of his RCA, which places him at risk for reocclusion and myocardial ischemia. In-stent thrombosis causing RV infarct/hypotension usually occurs in the first 30 days. It is not clear from the history as to whether he sustained an MI during his most recent hospitalization, but if he had, he is also at risk for myocardial rupture, and pericardial effusion (Dressler's syndrome, hemorrhagic). Causes of hypotension in both scenarios include:

- Systolic dysfunction if ischemic etiology, diastolic dysfunction if effusion present
- Isolated RV dysfunction
- Pericardial tamponade
- Papillary muscle dysfunction—should be considered as the RCA is the sole coronary artery supplying the posteromedial papillary muscle. With dysfunction or rupture, the patient will have resultant acute mitral regurgitation, pulmonary edema, and cardiogenic shock.

Hospitalizations:

He has been hospitalized twice in recent weeks. Commonly encountered complications arising from hospitalization are infectious and thrombotic. Potential sources of infection in this patient are urine and lung. He reportedly had a pneumonia, pleural effusions and was recently intubated; he could have an empyema, parapneumonic effusion, abscess, or failed outpatient antibiotics. Hypotension in the setting of infection would be due to distributive shock.

Pulmonary embolism in a recently hospitalized hypotensive/tachycardic patient cannot be ruled out by his supratherapeutic anticoagulation; however, the suspicion is lower given that the patient has a secondary protection from an IVC filter.

Medications:

An elevated INR would not be surprising considering that he recently started levofloxacin,[1] placing him at risk for bleeding in all listed above locations plus other mucosal sites (e.g., GI tract). Other considerations of medication related conditions include uremic pericarditis from nephrotoxic medications such as levofloxacin, aspirin, contrast dye, or loop diuretics.

Summary:
Included in the differential are also mechanical causes of shock, specifically pericardial tamponade and tension pneumothorax, both of which require urgent needle decompression.

These varied scenarios create a "shock" differential that includes the following:
Cardiogenic, distributive and obstructive. The initial interventions are drastically different… Volume, inotropes or pressors? Each of these therapies holds certain risk if treating the wrong condition, such as erroneously giving a large fluid bolus when the patient actually has CHF. Delays in appropriate interventions are high-risk, particularly when considering hemorrhagic tamponade in a patient with a coagulopathy!

In the "old days" before the use of bedside sonography, pericardiocentesis was often considered only when the patient was in PEA arrest, and the chances of meaningful recovery at this point in the disease course were slim to none. This is the true value of point-of-care ultrasound (POCUS); early identification of life threatening conditions while there is still time to intervene.

Section editor comments (Greenwood):

Patients with low stroke volume as a result of external compression or reduced systolic function are fully dependent on an elevated heart rate to maintain their cardiac output. The patient is presenting with tachycardia and a particularly narrow pulse pressure, which is an important sign pointing towards hypovolemic, cardiogenic, or obstructive shock over a wide pulse pressure that's often present in septic shock as a result of severe vasoplegia.

Q#2: Which tests should be done to start the work-up?

There is a growing body of data showing that early use of ultrasound is effective in reducing diagnostic uncertainty and narrowing the differential diagnosis. Additional benefits of early POCUS include an increase in the absolute proportion of patients with a definitive diagnosis, appropriate changes in the use of IV fluids, vasoactive agents, or blood products in addition to significant changes in major diagnostic imaging, consultation and emergency department disposition. [2–4]

This is a critically ill patient and he should be placed in an acute care resuscitation bed. Once IV access is established, blood should be sent for CBC to check for anemia, serum chemistries to measure electrolytes and serum glucose, renal function and anion gap and a VBG and lactate to assess tissue perfusion and hypoxia. Clearly his INR should be checked in addition to a type and screen, in the case that he will need blood. A troponin should also be sent and if positive, a CK-MB may also be helpful to determine timing of myonecrosis if he did have an MI within the past 14 days. The patient should be on telemetry and his BP frequently checked. A portable CXR should be performed to look for pulmonary infiltrate/consolidation, status of his known pleural effusions and to assess his cardiac contour. Importantly, the ECG is notable for a sinus tachycardia, low voltage and electrical alternans, so pericardial tamponade should be high on the differential.

This patient needs a diagnosis, now! The ultrasound machine should be brought to the bedside specifically looking for findings that would sufficiently narrow this broad differential diagnosis.

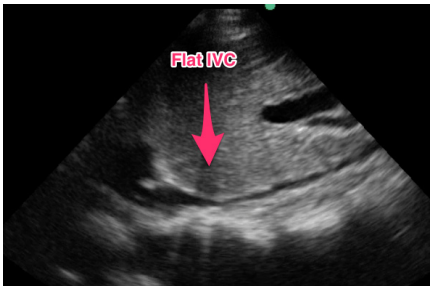
A number of US protocols have been proposed to organize the sonographic approach to the critically ill patient.[5-8] What they have in common is an organ-based approach to the patient with undifferentiated hypotension, assessing the heart, IVC, lung, and abdomen to identify causes of hypotension.

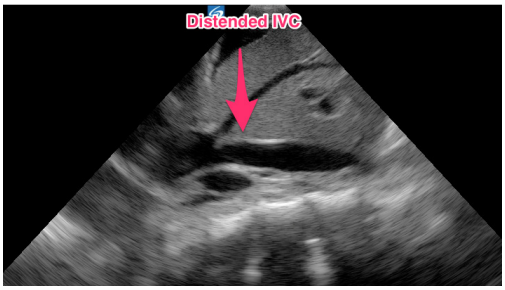
	CARDIOGENIC	RV DYSFUNCTION	TAMPONADE	HYPOVOLEMIC DISTRIBUTIVE	PNEUMOTHORAX
HEART	Poor systolic function, hypokinetic	Septal bowing, Enlarged RV	Effusion with RV end-diastolic collapse	Underfilled hyperdynamic	Likely underfilled and hyperdynamic
IVC	Plethoric	Plethoric	Plethoric	Collapsibility with respirations	Plethoric
LUNG	Diffuse B lines	(-)	(-)	(-)	No lung sliding, Barcode/stratosphere sign, A lines
AORTA Abdomen for free fluid	(-)	(-)	(-)	AAA Free IP fluid	(-)

1. **Cardiogenic shock:** LV systolic dysfunction, lung B lines, plethoric IVC with minimal respiratory variation.
2. **RV dysfunction (PE, Inferior MI):** RV free wall hypokinesis, McConnell's sign, RV dilatation, paradoxical RV septal systolic motion, dilated IVC with lack of respiratory collapse.
3. **Pericardial tamponade:** Pericardial effusion, RA systolic collapse, RV diastolic collapse.
4. **Hypovolemic shock (bleeding):** Hyperdynamic heart and flat IVC with exaggerated respiratory variation. AAA and/or free intraperitoneal fluid.
5. **Distributive shock (sepsis):** Note that unless the sepsis picture has affected the cardiac function, distributive and hypovolemic shock will appear similar on the RUSH ultrasound.
6. **Pneumothorax:** Absence of lung sliding, barcode/stratosphere sign on M-Mode, lung point.

Q#3: How should the hypotension be managed initially? How does the history of CHF change the indication for IV fluids?

POCUS is exceptionally useful when assessing volume responsiveness, particularly when the IVC is flat with exaggerated respiratory variation.[9] In this case, fluids are clearly of benefit. In patients with pulmonary or cardiac disease resulting in high RA/RV pressures, the respiratory collapsibility of IVC, while not perfect, may help predict fluid responsiveness. If there is any respiratory variation in the IVC, then a small bolus of fluids (500cc) may be helpful. The risk of a 500mL fluid bolus with history of CHF becomes a risk/benefit ratio, and the risk is lower when using a trial with a smaller volume.





In the patient who is not in florid pulmonary edema, tripoding and gasping for air, a fluid bolus trial is usually a safe and effective intervention. Fluid resuscitation is an appropriate and effective intervention in both hypovolemic and distributive shock.

POCUS has been shown to aid in assessing fluid responsiveness by assessing the IVC for diameter and respiratory variation with the patient supine and with straight raising.[9]

Section editor comments (Greenwood):

In general, IV fluids are an effective strategy in temporizing the shocked patient with pericardial tamponade. Fluids will increase intracardiac filling pressure relative to the intrapericardial pressure while arranging for pericardiocentesis.

Consulting editor comments (Weingart):

At our facility, we routinely temporize tamponade with volume loading.

Q#4: How does the elevated INR play into the differential?

Atraumatic bleeding in patients with an elevated INR can occur anywhere but most commonly in the GI tract, lung, and brain. In the setting of thoracic trauma, as in this patient, we have additional concerns regarding bleeding in the pleural, pericardial, and intraperitoneal cavities. Hypotension caused by bleeding within the pericardial space is most often due to myocardial contusion, wall rupture, or laceration of an epicardial vessel. These often cause hemodynamic instability with small volumes because of the speed of accumulation within the pericardial space. Slowly accumulating effusions can become quite large before becoming symptomatic. These tend to be due to malignancies that have involved the pericardium, uremia, rheumatologic diseases that cause inflammation of the serosal surfaces, (e.g., SLE, familial Mediterranean fever and indolent infections such as tuberculosis). Pericardial effusion due to any one of these conditions can also be hemorrhagic, particularly in the setting of an elevated INR.[10,11]

We know that the patient had a supratherapeutic INR and that his warfarin dose was reduced. We would have to assume that INR is still elevated, increasing the chance that bleeding may be contributing to his hypotension, either into his pericardium, pleural spaces and/or GI tract.

DECISION POINT A – ***What did the provider actually do?***

MDM (20:31): One consideration is pericardial tamponade and for that reason I will ask for a stat echocardiogram. Patient may have congestive heart failure and a saline well will be started. Additional labs are pending. Chest x-ray ordered. I did speak with the echocardiogram technician who asked that I call [the cardiologist]. I spoke with him and he agrees with an echocardiogram and he will call the echocardiogram technician to ask her to come in from home to do a stat echocardiogram. His lungs are clear, and he still could have congestive heart failure as etiology of his symptoms; however, the concern would be for fluid around the heart.

The ED physician was correct in being concerned about a pericardial effusion. It is not clear if he/she had access to a bedside ultrasound and/or training to screen for a pericardial effusion. ED physicians are now expected to have the ability to screen for a pericardial effusion, since this is one component of the FAST exam![12,13] If there was a machine then why wasn't it used? The 30 seconds it would take to look for a pericardial effusion would provide a diagnosis and shorten time to definitive care.

The diagnosis and treatment of a hemodynamically significant pericardial effusion is nearly impossible to treat without US at the bedside. Although it is something you might suspect, you are not going to blindly stick a needle in, unless the patient is actually in arrest. Any ED treating sick patients should have a US capable of performing the RUSH exam in any patient who presents hypotensive or in shock.

If there was no machine, then the ED physician needed to appreciate the severity of this patient's condition. The patient is hypotensive and tachycardic, and if the cause is a pericardial effusion, then it is causing tamponade physiology. While the potential causes of pericardial effusions are numerous, those conditions that present as *tamponade* are usually due to trauma or malignancy; we can be fairly certain that in addition to all his other problems, he is not likely to have developed cancer in the interim! He did suffered a significant blow to his torso breaking ribs, is anticoagulated and recently underwent a cardiac procedure. One must be concerned that he has a hemopericardium that is already of sufficient size to create hemodynamic instability to the point that it is imminently life threatening.

Section editor comments (Greenwood):

Rapid bedside echocardiography could also provide time sensitive information to get the correct consultant team assembled. In the setting of a recent AMI (approximately 3–7 days post-MI), one must be concerned about free wall rupture. Visualization of simple/clear fluid could not only represent a serious or inflammatory effusion but also fresh blood. A complex fluid collection, concerning for pericardial clot, may lead the EP to consider early surgical evaluation as well.

ED course:

The nurse approaches the physician, "I rechecked the blood pressure and it is now 72. Has the cardiologist called back?"

You respond, "Yeah. I just spoke with him and we are calling in the ECHO tech to see if there is pericardial tamponade."

"Do you want me to give a fluid bolus," asks the nurse. "We only have a saline well right now."

"How does the patient look?" you ask, hoping for a positive response.

"Sleeping…" responds the nurse.

DECISION POINT B – YOU ARE FACED WITH 4 QUESTIONS:

1. **What is the sensitivity and specificity of physical exam and ECG findings for pericardial effusion/tamponade?**
2. **Which POCUS findings support tamponade vs. effusion?**
3. **Should the EP perform a pericardiocentesis or wait for the cardiologist?**
4. **What is the best option for the EP to emergently drain the effusion/tamponade?**

Q#1: What is the sensitivity and specificity of physical exam and ECG findings for pericardial effusion/tamponade?

In medical school we learned that the "classic" findings for pericardial tamponade are Beck's Triad (Muffled heart + JVD + hypotension) and pulses paradoxus; measuring Korotkoff sounds when first heard compared to when they are heard throughout the respiratory cycle. When the difference is > 10mmHg, there is a 98% sensitivity and 70% specificity with + LR of 3.3.

A 2007 *JAMA* review described the presence of physical findings in pericardial tamponade as reported in 8 studies.[14] Five features were found in most patients with tamponade:

- dyspnea (sensitivity range, 87%–89%).
- tachycardia (pooled sensitivity, 77%; 95% confidence interval [CI], 69%–85%).
- pulsus paradoxus (pooled sensitivity, 82%; 95% CI, 72%–92%).
- elevated jugular venous pressure (pooled sensitivity, 76%; 95% CI, 62%–90%).
- cardiomegaly on chest radiograph (pooled sensitivity, 89%; 95% CI, 73%–100%).

The most common ECG finding is tachycardia (sensitivity 77%)! The three other findings associated with pericardial effusion are low voltage QRS, electrical alternans, and PR depression. These findings have good specificity but poor sensitivity for pericardial effusion/tamponade.[15]

The bottom line is that physical findings associated with tamponade are nonspecific and present in a variety of conditions that include congestive heart failure, pulmonary embolus and COPD exacerbation. Pulsus paradoxus is the only exam finding unique to the diagnosis of tamponade.

Q#2: Which POCUS findings support tamponade vs. effusion?

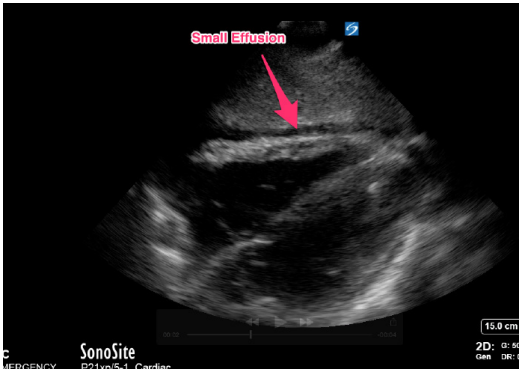
Early signs of tamponade reflect the fact that pressure within the pericardial sac has reached a point at which ventricular filling is restricted by the surrounding fluid/blood. During diastole, LV filling begins slightly before the RV. As the muscular LV fills, the less robust RV is essentially compressed and cannot completely fill. The rate of accumulation is more significant in cardiac

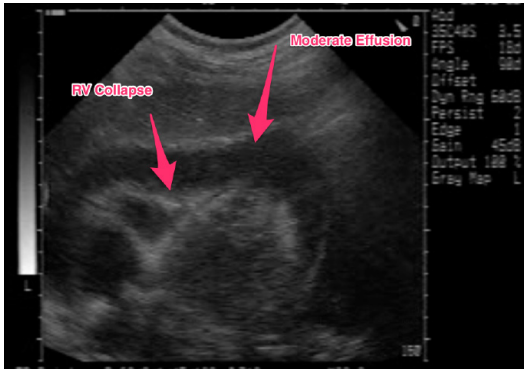
tamponade than the absolute size or composition of the pericardial fluid. Rapidly developing pericardial effusions can abruptly increase the intrapericardial pressure and produce cardiac tamponade with as little as 100–200 mL of pericardial fluid. But slowly accumulating effusions, as seen in malignancy or connective tissue diseases, can allow pericardial distention with tamponade not seen until the accumulation of 1–2L of pericardial fluid.

Transudative effusions as seen in rheumatic disease, hypothyroidism and uremia will be echo free, but those effusion related to trauma, infection or inflammatory conditions may contain echogenic debris (blood, pus, fibrin).[16]

The size of the effusion roughly correlates with the pericardial fluid volume as follows:

- Trace effusion: < 0.5cm
- Small effusion: < 1cm (roughly corresponds to < 100cc)
- Moderate effusion: 1–2cm (roughly corresponds to 100–500cc)
- Large effusion: > 2cm (roughly corresponds to > 500cc)





Major sonographic signs of tamponade are:

- Large pericardial effusion with swinging heart.
- Systolic collapse of RA.
- Diastolic collapse of RV.
- Inferior vena cava (IVC) plethora (dilatation > 20mm and < 50% reduction in the diameter of IVC with respiratory phases).
- Variations in E velocities during respiration across the mitral valve, tricuspid valve, and pulmonary outflow that are greater than 25, 50, and 30%.

Figure 1. Subxiphoid view of the heart showing a small pericardial effusion due to chest wall trauma that is already hemodynamically compromising as seen by RA collapse.

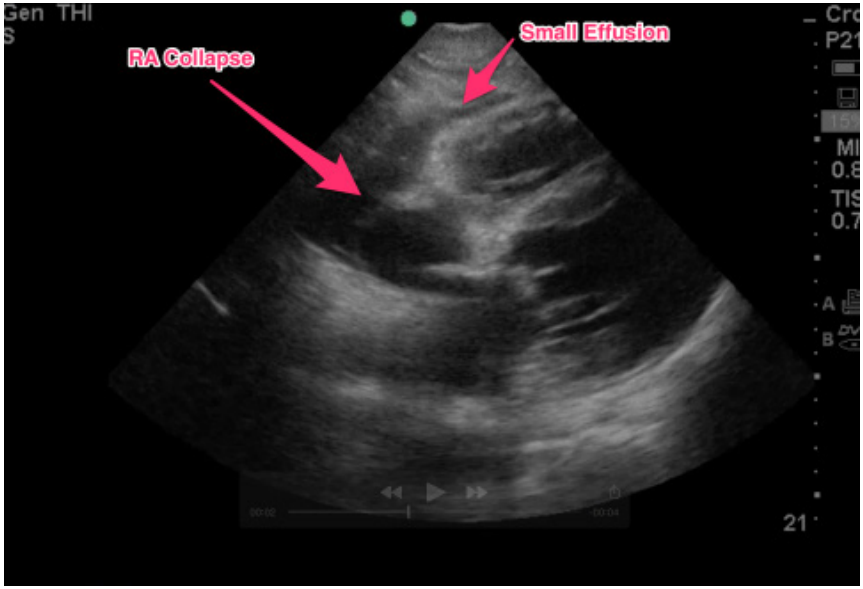


Figure 2. Large pericardial effusion in a patient with lung cancer that is now hemodynamically significant as seen by end-diastolic RV collapse.

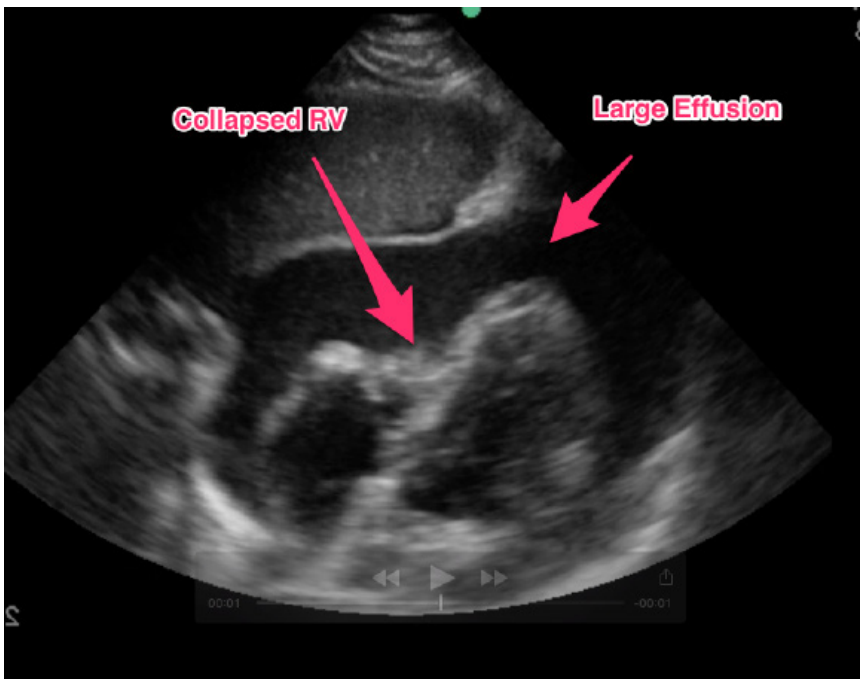
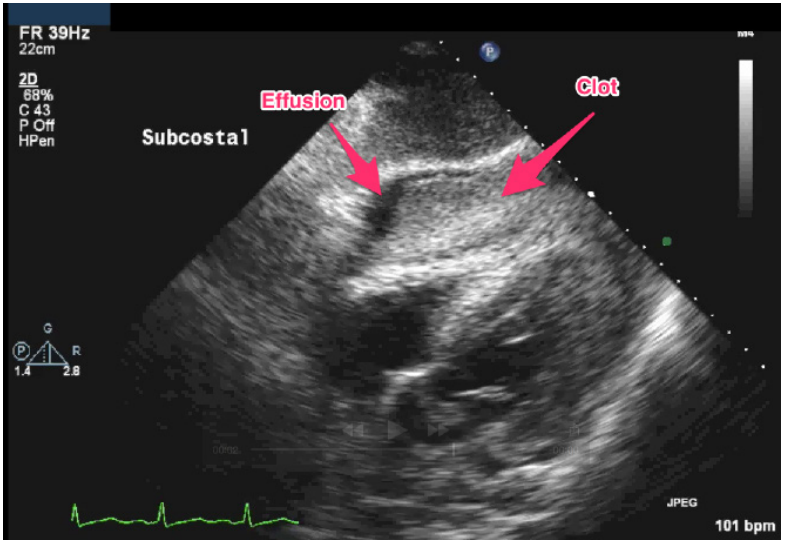


Figure 3. This is a patient with recent minor thoracic trauma, on warfarin, who presented with syncope. There is blood within the pericardial space that has partially clotted. Clot has the same echogenicity as the adjacent myocardium, which can lead to underdiagnosis of the effusion or size estimates.



Q#3: Should the EP perform a pericardiocentesis or wait for the cardiologist?

This is a tough call, because it is a procedure that is rarely performed and usually only under stressful conditions! I would venture to say that even a consultant cardiologist would have limited experience in performing emergent pericardiocentesis. Yet the performance of procedures in the setting of life-threatening conditions is what emergency physicians do, even if done infrequently. Pericardiocentesis falls in the same category as cricothyrotomy, an uncommon procedure that is essential and time-dependent when needed. As with emergent "crics," all ED physicians should take advantage of educational opportunities to maintain skills learned in residency through simulation and a wide variety of online instructional materials.

Consulting editor comments (Weingart):

This is a critical time to assess the confidence of the emergency physician to successfully perform US guided pericardiocentesis; if you can see a big pocket that you can reach with no intervening structures, the barrier to pull the trigger should be much smaller. If there is only the ability to perform a Hail-Mary/blind pericardiocentesis, I would wait until the last moment if a consultant is coming in, and they can do this under fluoroscopic guidance or US at the bedside.

Consulting editor comments (Weingart):

Note: Blind placement of a needle into a suspected pericardial effusion is very likely to just drain the ventricle and not the effusion. There should be no blind pericardiocentesis short of cardiac arrest—this is potentially deadly.

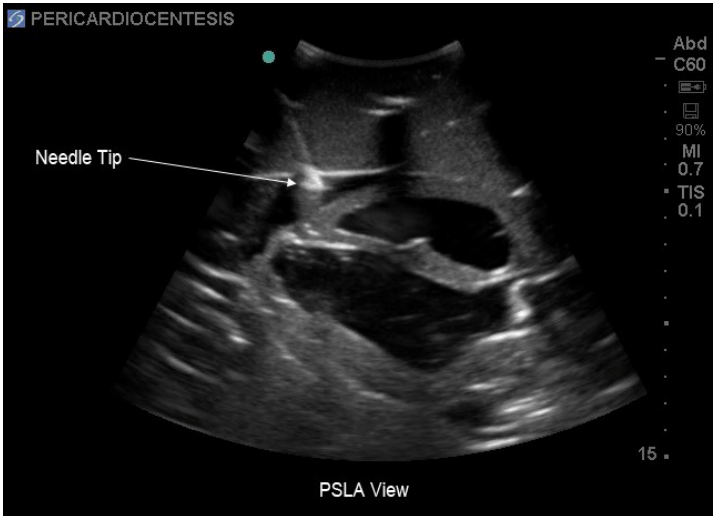
Q#4: What is the best option for the EP to emergently drain the effusion/tamponade?

Breathe, use POCUS and believe that you are the best person to save this person's life!

Pericardiocentesis procedural pearls:

1. Prep: Clean skin, elevate the head of bed to angle 30–45 degrees if possible to draw the heart more anteriorly. Use an 18Ga spinal needle, 20mL syringe. Methods for ongoing drainage include using a 3-way stopcock with tubing and also placing a catheter (8fr.pigtail/catheter).
2. Approach to pericardiocentesis when using sonographic guidance:
   a. The optimal approach is at the level of the 5th intercostal space, lateral to where you imagine the internal mammary artery.
   b. Apical, 1 interspace below and 1cm lateral to PMI toward right shoulder.
   c. Blind pericardiocentesis (PEA arrest) : The subxiphoid approach is 45 degrees along subxiphoid and left costal margin aiming towards left shoulder.
3. Procedure: Enter skin, aspirate continuously. Upon blood return, can use 3-way stopcock to drain.
4. After procedure: Use ultrasound if available to evaluate for improvement in effusion. Get chest x-ray to evaluate for pneumothorax and hemothorax.

Figure 4. This is a phantom view of needle aspiration of a pericardial effusion that closely represents the images seen in "real-life."

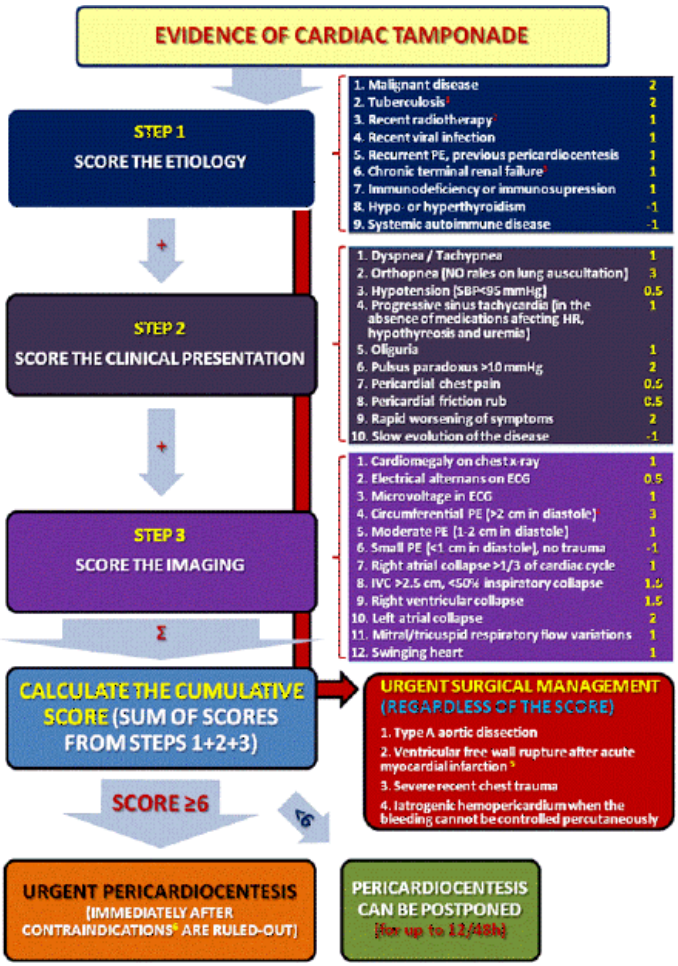


http://www.bluephantom.com/product/Transthoracic-Echocardiography-and-Pericardiocentesis-Ultrasound-Training-Model.aspx?cid=411. Image provided courtesy of CAE Healthcare.

The key question here is whether the EP should "wait." A consensus committee by the European Society of Cardiology in 2015 developed a scoring system to guide the timing of the intervention.[11]

Our patient has a score of 7 without ECHO! Consideration of his physical findings, CXR and ECG, mandates urgent pericardiocentesis.

- Dyspnea (1)
- Hypotension (0.5)
- Progressive tachycardia (1)
- Rapid worsening of sx (2)
- Cardiomegaly on CXR (1)
- Electrical Alternans (0.5)
- Microvoltage on ECG (1.5)



Ristić AD, Imazio M, Adler Y, et al. Triage strategy for urgent management of cardiac tamponade: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. Eur Heart J. 2014 Sep 7;35(34):2279-84. Copyright © 2014, Oxford University Press. Used with permission.

DECISION POINT B – ***What did the actual provider do?***

MDM 20:38: I did review the chest x-ray from today. This was done as a stat portable chest x-ray. Does show an enlarged heart which sometimes can be present with the portable chest x-rays. I did compare to previous. Today's film does show a large heart as has been seen in the past; however, the patient's lungs do appear mostly clear and do appear improved compared to previous x-ray. The blood pressure did now decrease to 72 systolic. Will give a 500mL normal saline bolus. I will go back and see the patient. He has been moved to the trauma room. Labs have returned:

Results review:		
WBC Count	**13.7thou/mcL**	**HI**
Hemoglobin	**9.9gm/dL**	**LOW**
Platelet Count	284thou/mcL	
Sodium Level	**123mmol/L**	**LOW**
Potassium Level	**6.1mmol/L**	**CRIT**
Chloride Level	**91mmol/L**	**LOW**
Carbon Dioxide Level	**16mmol/L**	**LOW**
Anion Gap	16.0mMol/L	
Glucose Level	**438mg/dL**	**HI**
BUN	**171mg/dL**	**HI**
Creatinine	**5.61mg/dL**	**HI**
Lactic Acid Level	**2.6 mmol/L**	**HI**
B Type Natriuretic Peptide	**131Picogram/ml**	**HI**
Prothrombin Time (PT)	**96.9Sec**	**HI**
INR	**8.43**	**CRIT**
Troponin I POCT	0.01ng/mL	

MDM (continued): I did review the patient's labs. Lactate elevated 2.6. INR is elevated at 8.4. I did speak with [the cardiologist] as the echocardiogram is being done at this time. The patient does have a large pericardial effusion. He is coming in to perform emergent pericardiocentesis.

Expert commentary: Was the action of the EP appropriate? What could have been done better?

As written, this physician's actions do not suggest an appropriate urgency in managing this very sick patient! Even if unable to perform a bedside ECHO, they should be at the bedside and watching the technician. On seeing the effusion, it should have been clear that this was the cause of the patient's hypotension (no surprise) and the physician should be concerned that it may be hemorrhagic because of the markedly elevated INR. They should have been on the phone with

the blood bank to order PCC, type and cross-matched blood, and then called the cardiologist to inform them of the findings and to drive to the hospital faster! At the same time a needle and syringe with alligator clamps should be clamped to the leads on the ECG in readiness to perform an emergent pericardiocentesis if the patient were to lose pulses (PEA arrest). In the majority of mechanical causes of PEA arrest (tamponade, tension pneumothorax, pulmonary embolism), RV filling is compromised. Chest compressions directly over the RV may further limit RV filling and hence LV filling! I would suggest that they have a 500mcg dose of phenylephrine at hand for pressor support until the pericardium is drained.

DECISION POINT C – YOU ARE FACED W ITH 2 QUESTIONS:

1. **Should the patient undergo emergent pericardiocentesis or be sent to surgery for a pericardial window?**
2. **What is the quickest way to reverse the anticoagulation?**

Q#1: Should the patient undergo emergent pericardiocentesis or be sent to surgery for a pericardial window?

The patient is in obstructive shock and is decompensating while in the ED. They have a problem which can be temporized by a procedure within the scope of practice of the EP. In the setting of his coagulopathy, it would be advised to leave a drain in place (i.e., 8 fr. catheter or catheter from pericardiocentesis kit) in case it was to reaccumulate.

Echocardiography or fluoroscopy-guided pericardiocentesis is the usual method of choice for drainage of pericardial effusions, with surgery is reserved for the following 4 scenarios:

1. Type-A aortic dissection.
2. Free wall rupture after a myocardial infarction.
3. Recent and severe chest trauma.
4. Iatrogenic hemopericardium when bleeding cannot be controlled through percutaneous access.[17-19]

This patient has had both trauma and a catheterization in the recent past and is anticoagulated. He is at great risk for hemopericardium, so that a catheter (rather than needle aspiration) should be placed within the pericardium to allow for continued drainage and CT surgery notified as back-up.

Section editor comments (Greenwood):

In the setting of hemopericardium from type-A dissection or free wall rupture, one additional consideration of the EP would be to perform a controlled pericardial drainage as a bridge to the OR instead of a complete pericardial drainage. The goal is to improve the patient's hemodynamics enough to maintain systemic perfusion but not to excessively increase the patient's blood pressure to encourage re-bleeding. An interval drainage of 5–10 mls of pericardial fluid to achieve a systolic blood pressure goal between 80–90mmHg, should be sufficient to improve the patient's clinical status and prevent further deterioration.[20]

Consulting editor comments (Weingart):

I agree!! I took 3mLs off of a dying type A dissection recently and the patient was rock solid stable through 60-minute transfer after the procedure.

Q#2: What is the quickest way to reverse the anticoagulation?

Patient is having tamponade which is likely hemorrhagic and an INR > 8. The INR needs to be urgently corrected prior to performance of pericardiocentesis. Once a drain/window has been established, there will be the ongoing concern for continued bleeding. He should receive vitamin K 10mg IV and 4-factor prothrombin complex concentrate (PCC) which is dosed based on INR and body weight. For this patient with an INR of 8.0, he should receive 50U/kg; max dose 5,000U capped at 100 kg body weight for VKA reversal. Per unit volume, 4F-PCCs contain approximately 25 times the concentration of vitamin K–dependent factors as compared with plasma (1U/mL). Therefore, PCC can be given in a much smaller volume at a much faster infusion rate (8×) compared with plasma.[21]

DECISION POINT C – *What did the actual provider do?*

MDM (21:08): I did speak with the pharmacist and we will do a prothrombin complex concentrate and they will send that down emergently. His blood pressure is now 60/palp and he is lethargic and diaphoretic. I asked for 250mcg of phenylephrine to be administered and a spinal needle to perform an emergent pericardiocentesis. I was able to withdraw 50cc of bloody fluid with improvement the patient's BP to 110 systolic. He had a brief run of VT and is now more alert.

Expert commentary: This is where the EP physician got lucky. A blind pericardiocentesis was performed and the observed VT was likely due to minor myocardial injury. There was improvement in the patient's BP which is a good thing and may have been due to the phenylephrine as well as the small volume drained.

MDM (22:23): [The cardiologist] did arrive and placed a catheter in the pericardial space and at this point has removed 300mL of dark red hemorrhagic fluid. The patient is receiving prothrombin complex concentrate (PCC) and IV vitamin K. The patient did receive Versed 2mg and morphine 2mg prior to the procedure. I have reviewed the labs. For the hyperkalemia he did receive dextrose, insulin and sodium bicarbonate. Patient does have significant hyperkalemia and significantly worsened renal failure.

Critical care time: 98 minutes

Final diagnosis:
Pericardial tamponade secondary to hemorrhage secondary to over coagulation from Coumadin, tachycardia, hypotension, acute on chronic renal failure, anemia, severe hyponatremia, hyperkalemia.

The patient is admitted to the intensive care unit with a consult to the intensivist and the cardiologist.

Final discussion/chapter author expert opinion:
This is a patient who presented in triage at 19:39 with vital signs showing a HR of 121 and BP 78/58. Patient presumptively was seen by a PA where H&P was obtained. Forty minutes later the hypotensive patient is first seen by MD at 20:16. This seem to be an excessively long delay in MD notification when seeing a patient with unstable vital signs at triage. MD notes that heart tones are distant, and the ECG shows low voltage QRS with a rate of 104. An ECHO is then ordered "stat" at 20:31. The CXR is reviewed and shows an enlarged heart. Labs are reviewed at 20:40 and show that he is hyperglycemic, acidotic, coagulopathic and uremic. The patient then becomes more hypotensive, and a fluid bolus is given. Over the next 2 hours the patient was treated with fluids, PCC, and Vitamin K awaiting the arrival of the echocardiographer and cardiologist. Fortunately, the patient survived despite what can be interpreted as delays in care owing to the failure to perform a bedside echo on the patient's arrival.

Timeline (a look back at this case): Arrival 19:39 > Pericardiocentesis @ 22:23 = 2 hours and 44 minutes

An alternative scenario including the use of echocardiography would be the following:

19:39 - Pt arrives with unstable VS.

20:00 Physician made aware and obtains history of trauma, recent instrumentation and medications. Requests immediate assistance from staff to start IV fluids and send off INR, lactate, VBG, Type and Screen. ECG obtained showing sinus tachycardia and low voltage.

20:15 Bedside ECHO performed by physician that identifies pericardial effusion with tamponade physiology.

20:30 Physician reviews labs and sees elevated INR. Orders PCC.

21:00 PCC given. Equipment assembled to perform pericardiocentesis (Betadine, Lidocaine and 18-gauge needle). Aspiration of hemorrhagic fluid until aspiration is dry and/or BP improves.

Timeline (optimal/improved): Arrival 19:39 > Pericardiocentesis @ 21:00 = 1 hour 21 minutes

Inpatient course:
Cardiology consultation: Patient with recent hospitalization for CP/PNA with an echo showing a trivial pericardial effusion presented with elevated INR/distant heart tones/low voltage ECG per ED. Stat echo was ordered showing a large pericardial effusion. Bedside pericardiocentesis was performed in the setting of coagulopathy due to hypotension and tamponade. Improved hemodynamics after procedure. Effusion likely a combination of uremia and coagulopathy. Resume warfarin when effusion and renal function stabilized. Will follow.

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