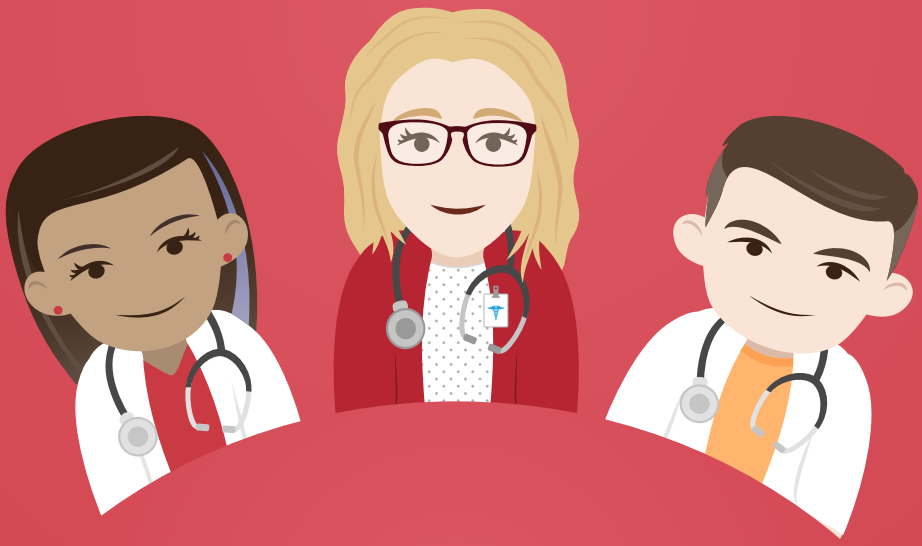
Cardiac Tamponade

Handbook



Lorrel E.B. Toft, MD

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Abbreviation list

Ao	aorta
BP	blood pressure
bpm	beats per minute
COPD	chronic obstructive pulmonary disease
ECG	electrocardiogram
echo	echocardiogram
IV	intravenous
LA	left atrium
LV	left ventricle
MV	mitral valve
RA	right atrium
RV	right ventricle

Chapter 1

The pathophysiology of cardiac tamponade

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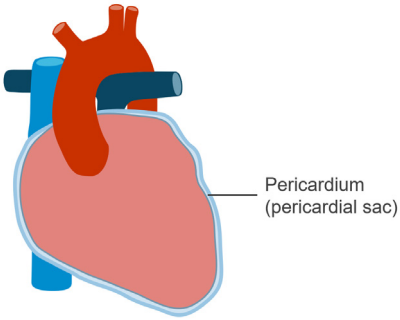
Understanding elevated intrapericardial pressure

Cardiac tamponade is elevated pressure inside the pericardial space that compromises cardiac output. But before we can dive into the details, we need to take a trip down memory lane to review some anatomy.

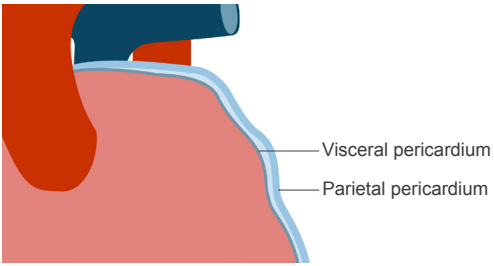
Heart anatomy

Pericardium

The heart sits in a double-layered membranous sack called the pericardium or pericardial sac.



The inner layer is called the visceral pericardium. It is closest to the heart muscle and so thin that it is almost non-existent, so you can just think of it as the outer surface of the heart. The outer layer is called the parietal pericardium and, in the words of my cardiac surgeon friends, it is like smooth, glistening, thick, wet leather.



Imagine the heart wrapped in a leather jacket!

Pericardial space

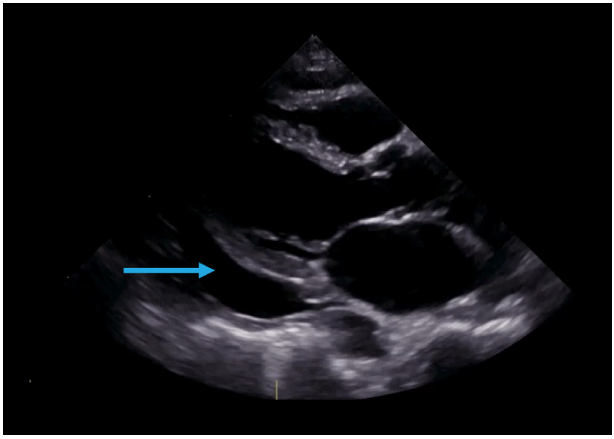
The pericardial space is the space between these two pericardial layers. In healthy circumstances, it contains a small amount of fluid, about 15–50 mL, which acts as a lubricating fluid.

The heart is a high-power machine, pumping 60–100 beats per minute (bpm). Pericardial fluid is there to decrease friction and allow for smooth movement of the heart.

Pericardial effusion and cardiac tamponade

A pericardial effusion is an accumulation of extra fluid in the pericardial space.

Many people will see a chest image or an echocardiogram with a large pericardial effusion, like the one shown below, and panic because they equate a pericardial effusion with cardiac tamponade. However, the increased *volume* of pericardial fluid is not the same as increased *pressure*.



Pericardial effusion

We care about increased *pressure* because that's what causes tamponade.

Aren't volume and pressure related?

Yes, volume and pressure are related. In general, if you increase the volume of fluid in a rigid container, you increase the pressure. But, if the container can stretch, then an increase in volume won't lead to the same sharp increase in pressure.

So, it's not really how *much* fluid accumulates, but more how *quickly* it accumulates that matters. Let me explain.

Remember I said the pericardium is like wet, glistening leather? Well, if you've ever owned a pair of leather shoes, you know that leather can stretch, but it doesn't stretch instantly like elastic does. It takes time to slowly stretch to fit your foot.

The pericardium is just like this. It can stretch to accommodate increases in fluid, but it does so slowly. And as it stretches, the extra fluid will not cause increased pressure. The pressure only increases when extra volume accumulates faster than the leathery pericardium can stretch.

That's why a large pericardial effusion, *if* it develops slowly, will not cause cardiac tamponade. However, a small amount of fluid accumulating rapidly, like in the case of chest trauma causing rapid arterial bleeding into the pericardial space, *will* cause tamponade. Sometimes 50 cc is all it takes.

Next, we will learn the consequences of increased pericardial pressure.

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Describing the consequences of elevated intrapericardial pressure

Now that we know how increased intrapericardial pressure develops, let's think about the effects that has on the heart.

In healthy circumstances, the pericardial pressure is effectively zero and has no effect on cardiac function. But when the pressure rises, it can start to compromise cardiac filling.

Let's review normal pressures inside the cardiac chambers.

Normal pressures

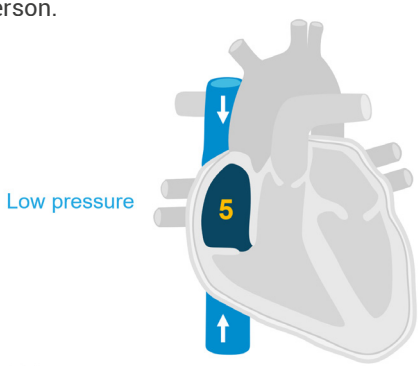
I think about the normal pressures inside the heart as American coins:

- Nickels are 5 cents.
- Dimes are 10 cents.
- Quarters are 25 cents.

If you aren't familiar with American coin denominations, you can just memorize those numbers.

Right atrium: 5 mmHg

The right atrium is a low-pressure chamber receiving the low-pressure venous blood return from the rest of the body and it sits around 5 mmHg in a well-hydrated person.

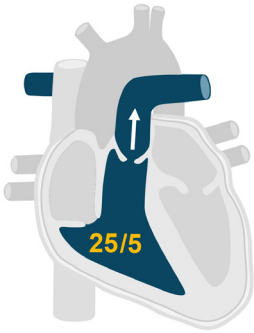


Right atrium

Right ventricle: systolic 25 mmHg, diastolic 5 mmHg

The right ventricle pumps blood into the pulmonary circulation, so it has two pressures, systolic and diastolic. The systolic pressure is around 25 mmHg, and the diastolic pressure is again 5 mmHg.

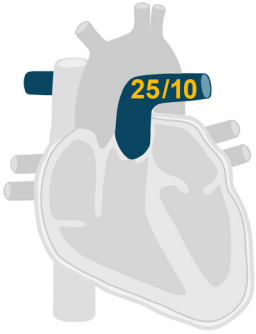
This makes sense because at the end of diastole when the tricuspid valve is open, the pressures in the right atrium and right ventricle equalize.



Systolic / diastolic pressure
Right ventricle

Pulmonary artery: systolic 25 mmHg, diastolic 10 mmHg

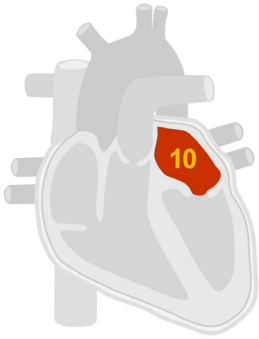
In the pulmonary artery, the systolic pressure is also 25 mmHg, but the diastolic pressure is 10 mmHg. This is because the pulmonary arteries narrow, so the diastolic pressure is higher than in the right ventricle due to the tighter space.



Systolic / diastolic pressure
Pulmonary artery

Left atrium: 10 mmHg

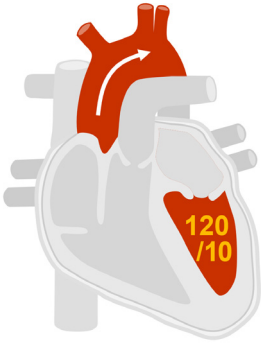
The left atrial pressure is right around 10 mmHg. It's actually 12 mmHg, but again the coin analogy breaks down, and we really just need to know the relative pressures to understand tamponade.



Left atrium

Left ventricle: systolic 120 mmHg, diastolic 10 mmHg

Finally, the left ventricular pressure is high in systole to pump the blood to the whole body, around 120 mmHg, and the end-diastolic pressure is again equal to the atrial pressure, so around 10 mmHg.



Systolic / diastolic pressures
Left ventricle

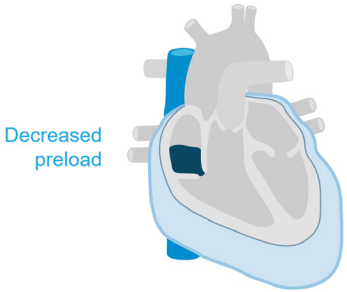
You can learn more in-depth about cardiac pressures in the Medmastery course Invasive Hemodynamic Monitoring.

What happens with increased intrapericardial pressure?

Imagine squeezing the heart from the outside. Which chambers will be affected first?

The low-pressure right-sided chambers will be the first to feel the pressure. As pericardial pressure rises, the pressure inside the right atrium and ventricle also starts to rise.

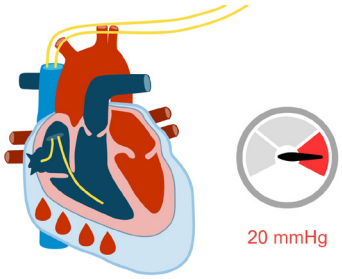
As this happens, blood from the low-pressure venous system has a harder time getting into the now high-pressure chambers, so the filling of the right side of the heart decreases. We call this decreased preload.



If there is less blood filling the right side of the heart, there is less blood being transmitted to the left side of the heart. Eventually, this leads to a drop in stroke volume (which is the amount of blood ejected into the aorta with each contraction). Therefore, the cardiac output, which is the amount of blood pumped per minute, also drops.

Let's review, this time using some numbers for the mathematically inclined.

Imagine a patient is having a pacemaker placed, and the right atrium is punctured during the procedure, causing bleeding into the pericardial space. The pericardial pressure rises from 0 mmHg up to 20 mmHg.



What happens to the right atrium and right ventricle? The pressures inside the chambers start to rise, eventually equalizing with the pericardial pressure.

Now that the right atrial pressure is 20 mmHg, the venous blood returning from the body can no longer fill the heart, eventually causing the reduced cardiac output as we just saw.

Summary

Overall, the hemodynamic consequences of elevated intrapericardial pressure that you need to remember are:

- Elevated right atrial and right ventricle pressures
- Decreased venous return to the heart
- Decreased right-ventricular and then left-ventricular filling
- Decreased cardiac output

Take a second to quiz yourself and see if you can remember that list. Next, we will move on to pulsus paradoxus.

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Explaining the finding of pulsus paradoxus

Possibly my favorite physical exam finding is pulsus paradoxus. I'll teach you how to measure it in the next chapter, but I want to use what we've learned about cardiac tamponade to explain this phenomenon.

Defining pulsus paradoxus

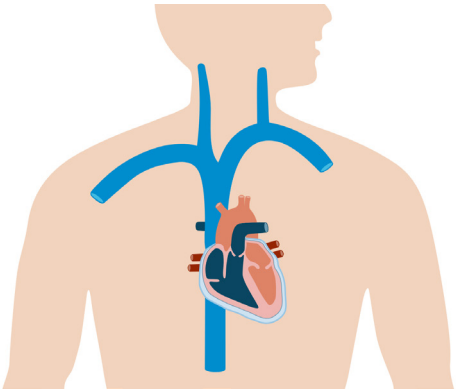
Pulsus paradoxus is misnamed, because it's not a *paradox* at all, but rather an exaggeration of normal physiology. We'll learn about it in the context of cardiac tamponade, but keep in mind it can occur in a few other clinical situations.

The definition is, "An exaggerated decrease of 10 mmHg or greater of the systolic blood pressure during inspiration." But that's quite complex, so let's think about it step by step.

What happens in a normal heart?

First, think about a normal, healthy heart. Normally, inspiration decreases intrathoracic pressure, which makes it easier for blood to enter the veins of the chest.

So, inspiration increases venous return. This increased filling of the right heart leads to an increased volume of blood in the right ventricle.



Inspiration increases venous return

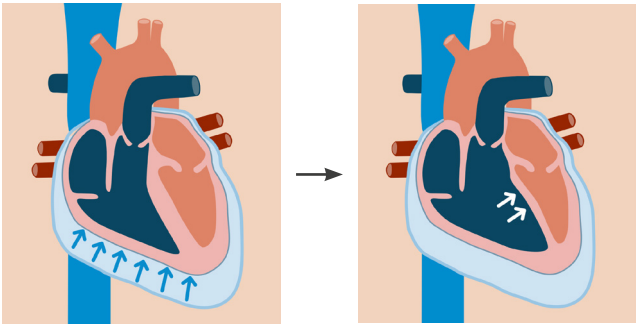
Here's where it gets exciting. How does the heart accommodate this extra blood volume?

In a healthy heart, the free wall of the right ventricle shifts outward slightly into the pericardial space, and all is well.

What happens in a heart under pressure (in tamponade)?

Again, inspiration decreases intrathoracic pressure, increasing venous return and increasing the right ventricular volume. But this time the free wall of the right ventricle (RV) is not free to expand into the pericardial space because the intrapericardial pressure is high.

So instead, the only way for the right ventricle to accommodate the extra blood volume is to shift the septum towards the left ventricle (LV), squishing it.



Increased intrapericardial pressure

Septum shifts toward LV during inspiration

This smaller LV cavity means a lower LV stroke volume, decreased cardiac output, and lower blood pressure.

So, going back to the definition of pulsus paradoxus, now we know that the reason why the blood pressure is lower during inspiration is that the left ventricle is getting squished by the right ventricle, which is getting squished by the pericardial pressure.

To help you remember this, think of sitting in the middle back seat of the car when you were young. Your sibling pushed into your personal space on purpose, squishing you, which caused you to squish your other sibling. That's basically how tamponade (the pushing sibling) causes decreased cardiac output (the squished sibling)!

Those of you who have never experienced this road-trip phenomenon are clearly not middle children!

Now that you've mastered the hemodynamics of cardiac tamponade, next we'll learn how to make the clinical diagnosis, and we'll use pulsus paradoxus to help us.

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Chapter 2

Diagnosing cardiac tamponade

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Making the clinical diagnosis

Cardiac tamponade is a life-threatening, can't-miss condition, but can be tricky to diagnose. We're going to integrate risk factors, clinical history, and physical exam findings to make the diagnosis.

You'll hear it said that tamponade is a *clinical* diagnosis. That just means that while imaging is helpful, you cannot rely on imaging alone and must make the diagnosis based on your clinical judgment.

Most but not all patients with tamponade will have hypotension (i.e., low blood pressure). So, for now, let's start by imagining a patient with hypotension, and I'll get to the exception later in this chapter.

Assessing risk factors

The first question I ask myself is, *How likely is this person to have tamponade?* That's another way of asking, *How high is my clinical suspicion based on their risk factors?*

So, what types of patients develop tamponade?

Chest trauma and cardiac procedures

Pretty obvious examples are a patient with chest trauma or a patient who has just had a cardiac procedure such as an angiography or pacemaker placement where, unfortunately, there can be perforation.

Any conditions associated with pericardial effusion

Anything that can cause a pericardial effusion can cause tamponade if the effusion accumulates quickly enough. So, conditions that can cause effusions and sometimes tamponade include the following:

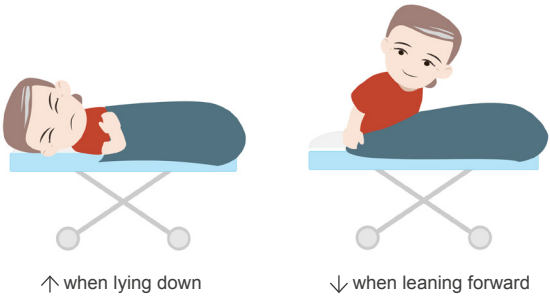
- Malignancies like breast and lung cancer
- End-stage renal disease (particularly in patients who have missed dialysis sessions)
- Tuberculosis (known for causing a bloody pericardial effusion)
- Prior chest radiation therapy (less frequent cause)
- Hypothyroidism (less frequent cause)

So, knowing your patient's history can help determine your level of suspicion. However, be aware that tamponade can sometimes develop without one of these causes, or it could be the first presentation of the underlying cause and, therefore, you won't yet know they have that predisposing condition.

Identifying the symptoms

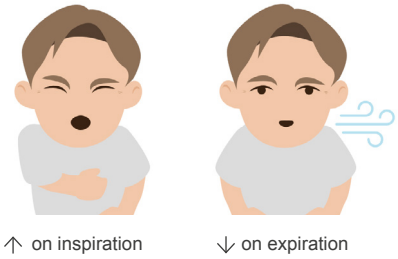
What symptoms might a patient complain of with tamponade?

1. **Shortness of breath**
   This is common, particularly with exertion.
2. **Chest pain**
   Chest pain can also be present, especially if the patient has an inflammatory pericardial effusion. This type of inflammatory chest pain is classically described as worse when they lie down and better when they lean forward.



Inflammatory pericardial effusion chest pain

However, they could have pleuritic type chest pain instead which is worse on inspiration.



Pleuritic type chest pain

In fact, chest pain can be of any nature, so don't rule out tamponade just because the chest pain description isn't classical.

3. **Lightheadedness or confusion**
   This is due to the hypotension.

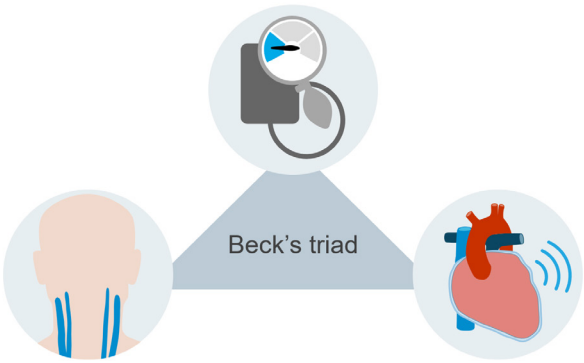
Physical exam findings

Now we get to the physical exam, which is critical for making this diagnosis.

Beck's triad

Beck's triad is the classic description of three signs that indicate cardiac tamponade:

1. Hypotension
2. Distended neck veins
3. Muffled heart sounds



Knowing the physiology of tamponade, this makes sense. Hypotension is a result of low cardiac output. Distended neck veins indicate the impaired venous return to the high-pressure right side of the heart, causing blood to back up and fill the neck veins. And muffled heart sounds are present because there is extra fluid between your stethoscope and the heart.

Pulsus paradoxus

Pulsus paradoxus is not part of Beck's triad, but it can be very helpful in making this diagnosis. Remember, pulsus paradoxus is a 10 mmHg or more decrease in

systolic blood pressure during inspiration. In the next lesson, you'll learn how to measure it yourself.

Tachycardia

Tachycardia is common as the heart rate increases to try to compensate for the low stroke volume and reduced cardiac output.

Describing the classic presentation

Why don't you try and describe a classic presentation of a patient with cardiac tamponade, and then I'll tell you mine? Try it, and then read my description below.

My patient is a 55-year-old female with breast cancer who has been feeling short of breath when walking between rooms in her house. She comes to the emergency department feeling light-headed.

Her blood pressure (BP) is 82/65 mmHg, her heart rate is 119 beats per minute (bpm), she's breathing rapidly, her external jugular vein is visible up to her earlobe when she is sitting upright, and her heart sounds are difficult to hear.

Did you imagine someone similar?

Next, let's learn how to measure pulsus paradoxus.

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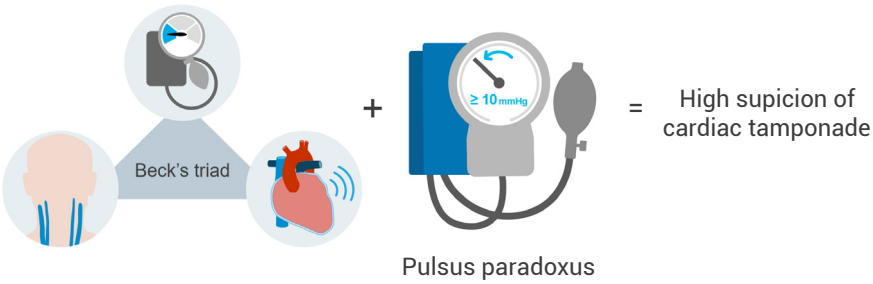
Obtaining a pulsus paradoxus measurement

Remember the patient we imagined presenting with cardiac tamponade? How can you confirm the diagnosis?

Here's where obtaining a pulsus paradoxus measurement is extremely useful. While there are a few other conditions that can cause pulsus paradoxus such as asthma or chronic obstructive pulmonary disease (COPD) exacerbations, the clinical picture of Beck's triad plus pulsus paradoxus is very indicative of tamponade.



Measuring pulsus paradoxus

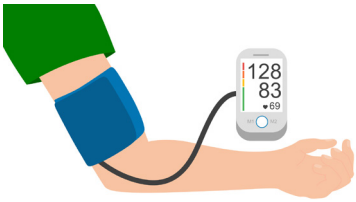
There are two different techniques to measure a pulsus paradoxus, but I'm going to teach you the one I find most practical and easiest to perform.

Preferred method

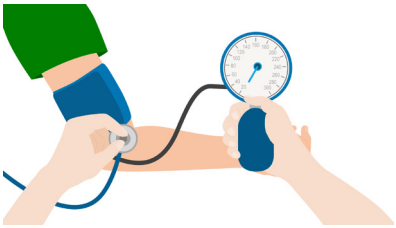
Remember that we are looking for a 10 mmHg or greater decrease in systolic blood pressure during inspiration compared to expiration.

1. **Take an automated BP measurement.**
   Start with getting an idea of the patient's average systolic blood pressure, usually from an automated measurement.
2. **Prepare to take a manual BP measurement.**
   Get out the manual sphygmomanometer (i.e., blood pressure or BP cuff) and prepare to take the blood pressure yourself. Note that you don't need to ask the patient to alter their breathing pattern in most cases.

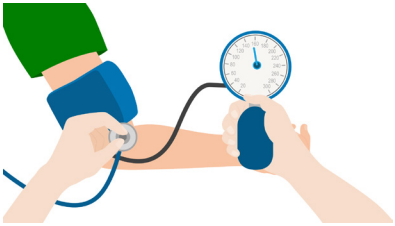
3. **Inflate the cuff approximately 20 mmHg *higher* than the automated systolic measurement.**
4. **Release the pressure very slowly and record the BP at the first Korotkoff sound.**
   Very slowly release the pressure while watching the needle and listening. Listen for the first time you hear a Korotkoff sound, which is the beat you hear with your stethoscope that is caused by blood rushing into the artery. Note the pressure corresponding to this first Korotkoff sound.
5. **Continue to very slowly release the pressure and record when you begin to hear a Korotkoff sound at every heartbeat.**
   You'll see the needle bounce with each heartbeat, but there will not be a sound with every beat, only intermittently. Wait until you hear a Korotkoff sound with *every* heartbeat or every bounce of the needle. Note the pressure when the sound with every heartbeat first occurred.
6. **Calculate the pulsus paradoxus measurement.**
   This is calculated by taking the BP you measured when you first heard a Korotkoff sound (i.e., the expiratory systolic pressure) minus the BP you measured when you began to hear Korotkoff sounds with every beat (i.e., the inspiratory systolic pressure).



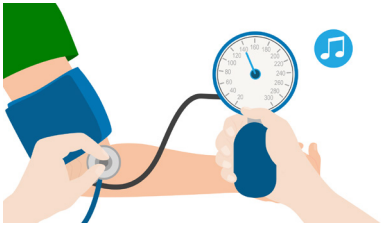
1. Take automated BP measurement.



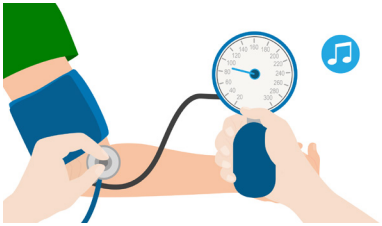
2. Prepare to take manual BP measurement.



3. Inflate ≈ 20 mmHg higher than the automated systolic measurement.



4. Release pressure very slowly and record BP at the first Korotkoff sound.



5. Continue to slowly release and record when Korotkoff sounds begin to occur at every heartbeat

Expiratory systolic pressure

– inspiratory systolic pressure

pulsus paradoxus

6. Calculate pulsus paradoxus

In the lesson video, we demonstrated what this process sounds like in reality.

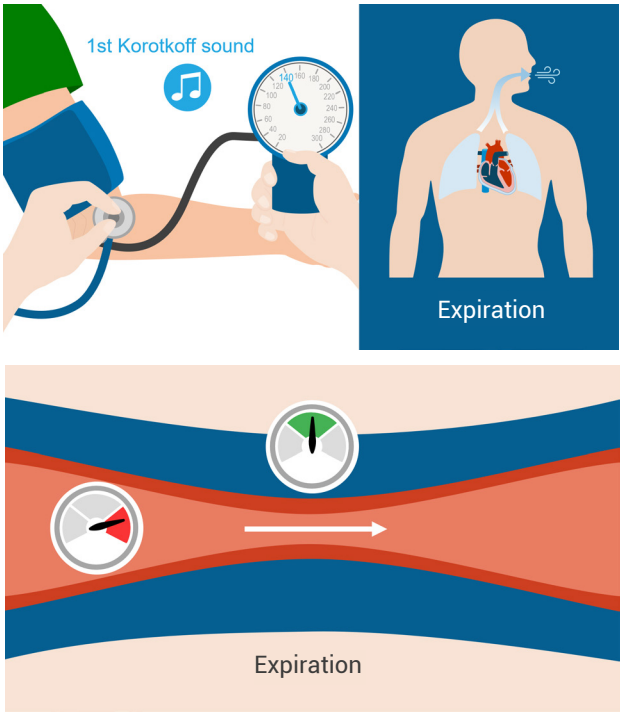
The first Korotkoff sound was heard at around 138 mmHg. Then we saw the needle bouncing but didn't hear another sound until around 110 mmHg. Then the needle continued to bounce but it was only when it reached 90 mmHg that we heard a sound with every beat.

Since the pressure was 138 mmHg when we heard the first Korotkoff sound and it was 90 mmHg when we started to hear a sound with every heartbeat, the pulsus paradoxus measurement would be calculated as follows:

$$138 - 90 = 48 \text{ mmHg}$$

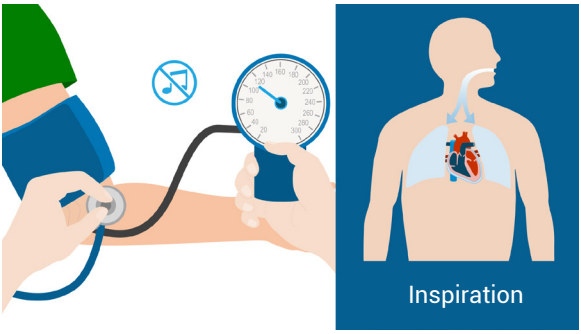
What does this have to do with inspiration and expiration?

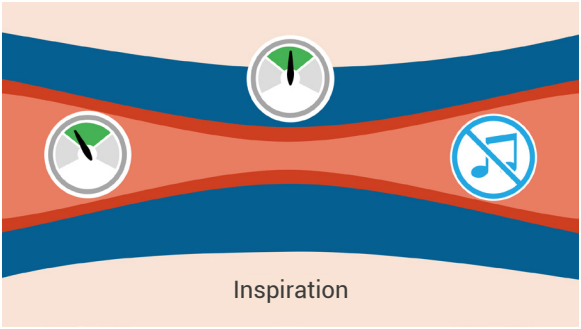
Well, that's exactly what you're seeing. The first Korotkoff sound, which happens at a high pressure, is during expiration. Remember, the sound from the blood rushing into the artery occurs once the pressure inside the artery is higher than the pressure from the blood pressure cuff.



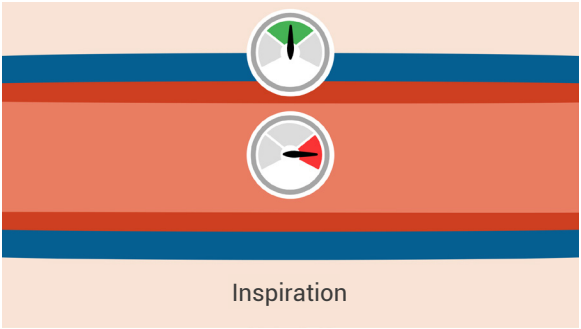
Then, the phase of intermittent sounds (i.e., the times when you don't hear any blood flow but see the needle move) indicates a heartbeat that is occurring during inspiration.

Remember that the blood pressure is lower during inspiration because of low cardiac output. So, during inspiration, the arterial pressure drops below the cuff pressure again, stopping blood from rushing in and resulting in no sound.





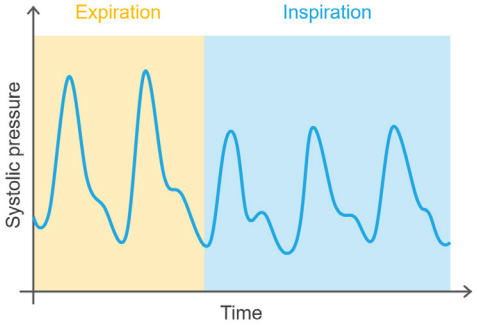
When you hear a sound with every heartbeat, that means you've got down to the point where the arterial pressure during inspiration now also exceeds the cuff pressure.



So, note the expiratory systolic pressure, which is when you first heard the sound, and then note the inspiratory systolic pressure, which is when you started hearing a sound with every beat. The difference is your pulsus paradoxus measurement!

How does this relate to pressure tracings?

If you were to look at the pressure tracing on an arterial line, it would look like the illustration below. You can see a lower systolic pressure during inspiration, and a higher systolic pressure during expiration.



If the patient has an arterial line in the intensive care unit, you might be able to see these fluctuations on the tracing. But I never rely on the arterial line; I always use my ears to make this measurement.

Alternate method

The other method of measuring pulsus paradoxus involves asking the patient to hold their breath during inspiration and taking a systolic blood pressure measurement, then taking a separate measurement during expiration. This can be tricky if the patient is short of breath or in distress, so I prefer the first method.

Troubleshooting

There are a few problems to avoid using my preferred method:

1. If the patient is breathing rapidly, you might not have much time to find the difference between inspiration and expiration.
2. If the pulsus paradoxus is really large (e.g., 20 mmHg), you may miss it if you do not inflate the BP cuff high enough.

I speak from experience when I recommend starting 20 mmHg higher than the automated measurements.

Next, we'll discuss electrocardiogram (ECG) and echocardiogram (echo) findings in tamponade.

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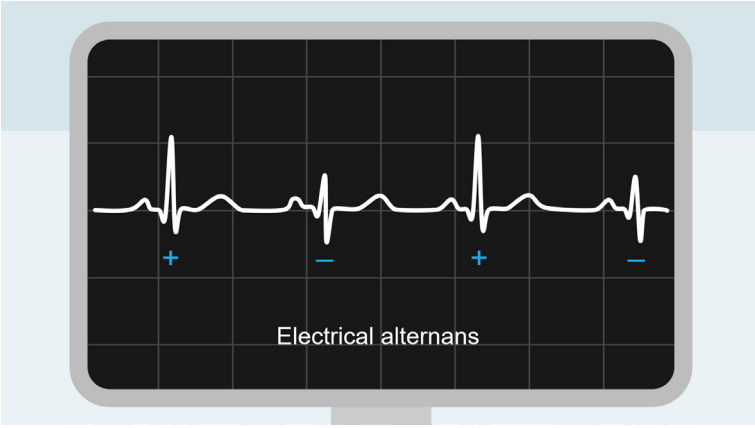
Interpreting ECG and echo findings

Remember I said that tamponade is a *clinical* diagnosis? That means you don't need an ECG or an echocardiogram to make the diagnosis, but they can be helpful, and it's important to know how to interpret the findings in tamponade.

ECG findings

In most cases of tamponade, the ECG will show sinus tachycardia (i.e., a regular rhythm greater than 100 bpm) and nothing more specific. However, there is a classic ECG finding called electrical alternans, which looks like the illustration below.

Alternans refers to the changing, or alternating, axis of the QRS complexes. See how at approximately every other beat the QRS switches from positive to negative axis? That's electrical alternans. This switch in axis is because the heart is swinging in the pericardial sack, which is now full of fluid.



In this lesson's video, we compared an echo of a heart with no effusion with an echo showing an example of a heart surrounded by a large pericardial effusion. We could see how the heart with effusion had a swinging motion. The heart changes positions from beat to beat relative to the fixed ECG leads, resulting in an alternating axis.

This can happen in any or all of the ECG leads, so there isn't a particular lead that is best to view electrical alternans.

But wait! Didn't I say that a patient can have tamponade *without* a large pericardial effusion? That's exactly right! Electrical alternans is not unique to cardiac tamponade, but rather to a moderate-size pericardial effusion.

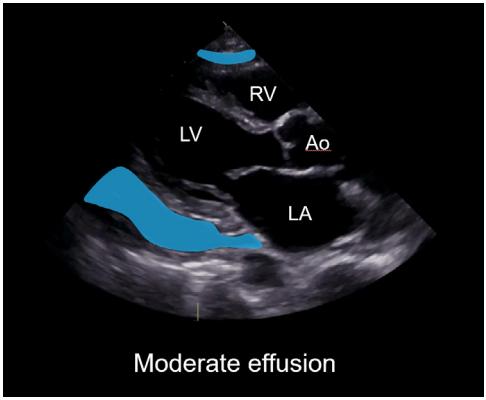
So, if a patient has tamponade caused by only a small effusion, you probably won't see this finding. But if the tamponade is caused by an effusion large enough, then electrical alternans might occur.

Echo findings

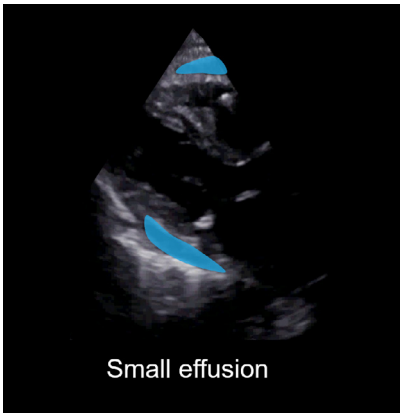
Many clinicians think of the echo as being definitive for tamponade but remember that it's just an added tool on top of your clinical diagnostic skills.

Size and location

An echocardiogram can show the size and location of a pericardial effusion. For example, below is a moderate effusion shown in blue. The chambers are pointed out for reference, LV for left ventricle, LA for left atrium, RV for right ventricle, and Ao for aorta.



Below is an echo demonstrating a small effusion.

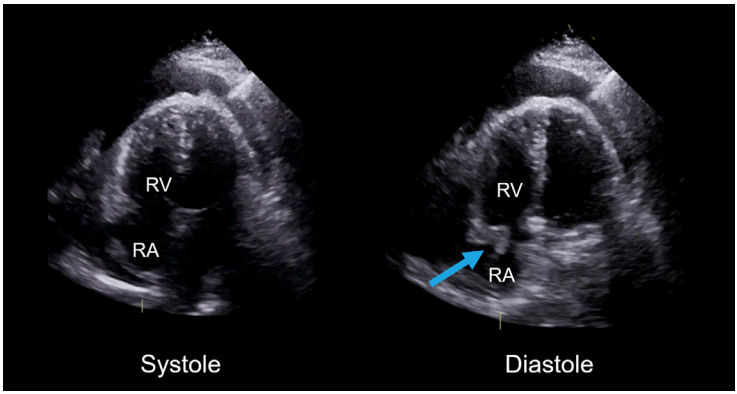


Increased intrapericardial pressure

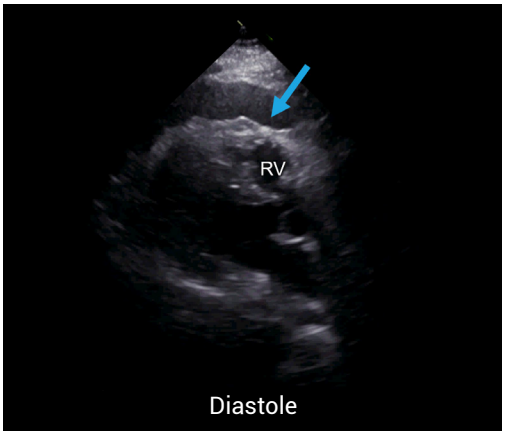
Echo can also detect signs of elevated intrapericardial pressure. Remember that as the intrapericardial pressure increases, the low-pressure right atrium (RA) and RV chambers get squeezed from the outside. They can even cave or buckle when the pressure inside the chambers is lower than the pressure outside in the pericardial space.

In the development of tamponade, RA and RV collapse occurs first during diastole. This is because diastolic pressures inside these chambers are lower than systolic pressures, and so it takes less pressure in the pericardial space to collapse them during diastole.

So, an early sign of elevated intrapericardial pressure you might see on echo is the diastolic collapse, or inward folding, of the right atrial or right ventricular walls. For example, next are echo images taken during systole and diastole, showing the right atrium and the right ventricle. The wall of the right atrium is buckling inward during diastole.



In this lesson, we watched a video of this echo showing the buckling of the right atrium and another echo video showing the right ventricular wall buckling inward during diastole. A screenshot from that second image is featured below.



This is the most important echo finding for evidence of elevated intrapericardial pressure and tamponade and it is essentially the echo equivalent of pulsus paradoxus. We can simplify this finding as different ventricular filling in inspiration versus expiration.

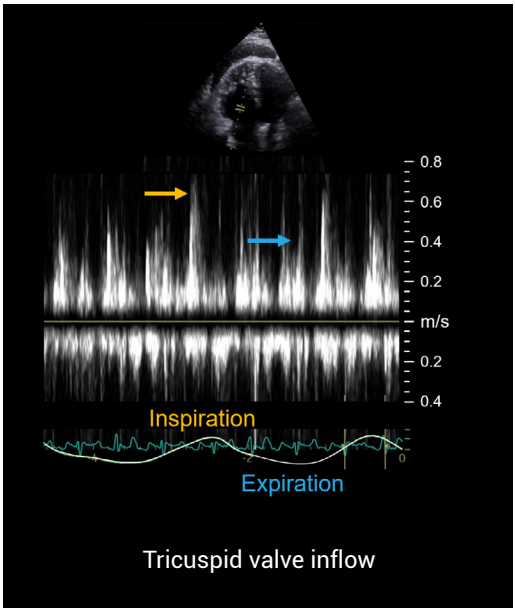
On echo, we can estimate ventricular filling by looking at the flow across the tricuspid and mitral valves. This is called tricuspid inflow or mitral inflow.

Estimating ventricular filling using tricuspid inflow

Below is what tricuspid inflow looks like. You can see at the top of the screen that the ultrasound probe is focused on the tricuspid valve on the right side of the heart.

The white tracing in the middle shows the flow velocity in meters per second across the tricuspid valve. This is a surrogate measure of right ventricular filling. There is an ECG across the bottom of the screen and overlaid on the ECG is a wave showing respirations, with inspiration at the peak and expiration in the valley.

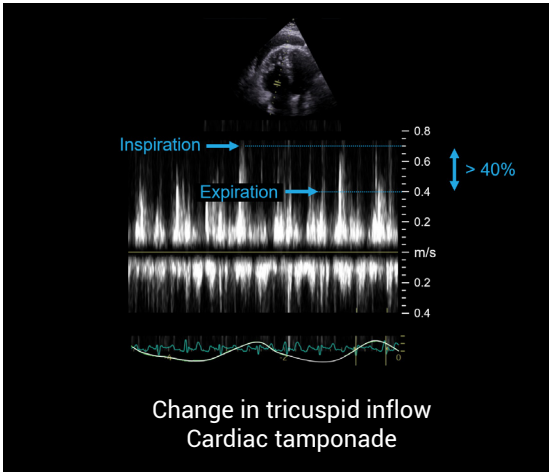
Because this is the tricuspid valve on the right side of the heart, we expect to see right ventricular filling (measured by velocity) higher during inspiration and lower during expiration



Now, remember that pulsus paradoxus is just an exaggeration of normal physiology. The definition used when looking for tamponade is a 40% or greater change in tricuspid inflow velocity between inspiration and expiration. In other

words, the velocity during expiration is at least 40% lower than the velocity during inspiration.

In the example below, see how the velocity during inspiration is 0.7 m / s but during expiration, it's only 0.4 m / s, which is just over 40% lower. Therefore, this meets the criteria for tamponade.

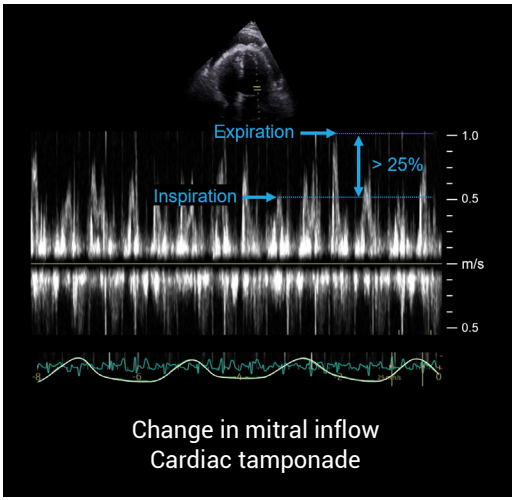


Estimating ventricular filling using mitral inflow

You can also look at mitral inflow on the left side. But on the left side, we expect ventricular filling to be lower during inspiration and higher during expiration. Also, because the left side chambers are more muscular and are higher pressure chambers in general, they are less sensitive to pericardial pressure changes.

So, the definition used on the left side when looking for tamponade is a 25% or greater change in mitral inflow velocity between expiration and inspiration. In other words, the velocity during inspiration is at least 25% lower than during expiration.

In the next echo showing mitral valve (MV) inflow, the velocity during expiration is around 1 m / s and the inspiratory velocity is just above 0.5 m / s, which is a greater than 25% decrease. Therefore, this also meets the criteria for tamponade.



Unless you're someone who reads echos routinely, it's not important to memorize those definitions. However, it is important to understand how the echo findings demonstrate the underlying pathophysiology of cardiac tamponade.

And now you understand why ECG and echo can be helpful but are not definitive for diagnosing cardiac tamponade!

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Diagnosing tamponade without hypotension

You are now armed with clinical skills and pathophysiology knowledge so you're ready to diagnose cardiac tamponade!

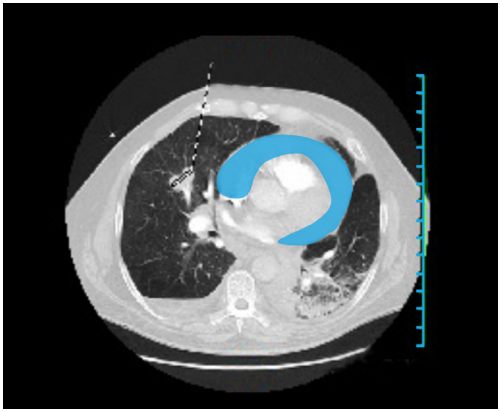
However, I mentioned earlier that cardiac tamponade presents with hypotension most of the time, but not always. This is just unfair since Beck's triad starts with hypotension!

Let me tell you a story of how understanding cardiac tamponade physiology helped me diagnose cardiac tamponade in a patient with elevated blood pressure.

A 48-year-old female presented to the emergency department with shortness of breath. She was from the Philippines and spoke Tagalog with limited English.

When asked, "How are you," she responded in English by saying, "I feel fine." But then when asked any other questions, such as, "Why did you come to the hospital?" she would just repeat, "I feel fine," over and over.

Her blood pressure was 135/90 mmHg, her heart rate 106 bpm, she was afebrile, and she was breathing 22 times per minute. A CT of the chest was obtained due to her shortness of breath and demonstrated a large pericardial effusion, shown below in blue.



At this point, the emergency department resident called me since I was on call overnight in the cardiac intensive care unit. He was practically holding a needle over her chest, eager to perform pericardiocentesis.

"She's got a *huge* effusion, so she must be in tamponade, right?" he said.

"She's normotensive!" I replied. "And tamponade is a clinical diagnosis, starting with hypotension." I clearly thought I was *so* smart, with all of 2 years' experience as a physician!

When I examined her, it was unclear whether her communication was limited by a language barrier or by altered mental status. Her blood pressure was indeed elevated. But I felt her legs, and they were cool to the touch, an indication of poor perfusion and low cardiac output.

This really seemed like tamponade! And sure enough, her pulsus paradoxus measured at 15 mmHg!

At this point, we activated the interventional team to perform urgent pericardiocentesis. This was performed in respiratory isolation because a possible cause of her effusion was tuberculosis, which is endemic in the Philippines and a common cause of effusion and tamponade.

Nearly 1 L of fluid was removed, her blood pressure skyrocketed to 160/100 mmHg, and she communicated fluently in English as she was actually bilingual! So, in this case, her altered mental status was the cause of the initial language barrier.

Keep in mind that when there are language issues, appropriate medical translation services should be obtained to ensure an accurate history and physical exam.

When we talked to her further, we discovered that she had chronic hypertension but had run out of medications recently. Further workup revealed the etiology of

her effusion and tamponade to be a large malignant thymoma (a tumor of the thymus gland) for which she underwent treatment.

The fascinating aspect of her case was that she had tamponade with relative hypotension since she normally lived with a much higher blood pressure. Her decreased cardiac output was causing her confusion and altered mental status, but we only knew so after fluid removal.

It was a lesson I'll never forget in the importance of measuring a pulsus paradoxus and understanding individual patient physiology.

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Chapter 3

Treating cardiac tamponade

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Stabilizing the patient

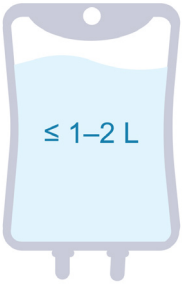
You've diagnosed a patient with cardiac tamponade. What now?

The definitive treatment is to drain the fluid by performing pericardiocentesis, which we'll cover in the next lesson. But while you wait (or set up) for that procedure, there are some temporizing measures to stabilize the patient.

Intravenous (IV) fluid

This is first and foremost. I prefer normal saline, but lactated ringer's solution is also acceptable. Any isotonic fluid to try and bolster intravascular volume works.

The problem in tamponade is underfilling of the right and therefore, left ventricles. By simply flooding the venous system with extra fluid, you can slightly increase venous pressure and get more fluid into the right side of the heart and therefore, the left.

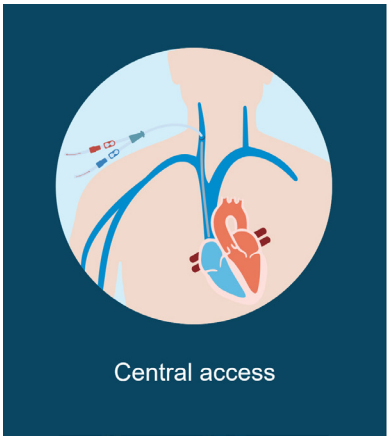


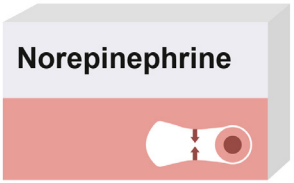
This is truly a temporary measure, and usually, 1–2 L of fluid is the limit.

The goal is a central venous pressure of around 15 mmHg. With higher pressures due to excessive fluid, the intrapericardial pressure will start to increase.

Vasopressors

If the IV fluids don't bring up the blood pressure, you can start vasopressors only as a stabilizing measure. If the patient has a central venous catheter, then norepinephrine is my vasopressor of choice.





Without central access, many providers like to use dopamine as it causes potentially less local vasoconstriction. However, dopamine is also going to increase the heart rate, and, in general, has not shown favorable effects in cardiovascular causes of shock.

I also do not recommend delaying definitive treatment in order to obtain central access. But if you have the time to quickly insert a central line, it can be useful.

The point is you must support the blood pressure any way you can until the tamponade is relieved. Vasopressors can acutely support blood pressure and tissue perfusion, but ultimately the increased afterload on the heart will further compromise cardiac output.

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Performing a pericardiocentesis

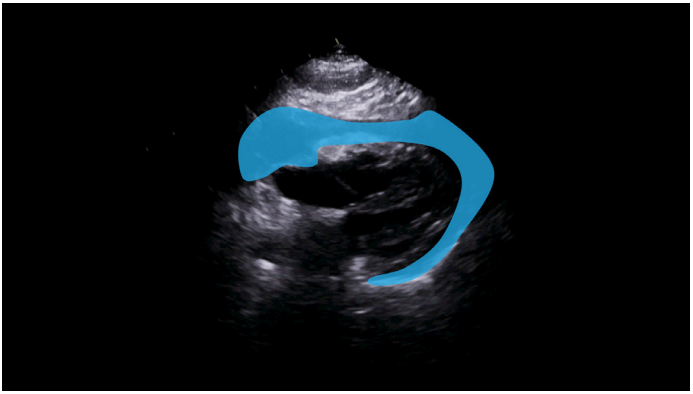
Pericardiocentesis is a procedure to remove fluid from the pericardium. The primary goal is to alleviate the elevated pressure and the secondary goal is to avoid damaging any vital structures in the process.

Depending on your practice location, the pericardiocentesis may be performed by different types of providers such as an interventional cardiologist, an emergency room physician, or a cardiovascular surgeon.

It's always preferred to perform the procedure in a controlled, sterile environment by an experienced team. But, in an emergency situation, pericardiocentesis can be lifesaving. So, let's learn how it's done.

Preparing for a pericardiocentesis

If available, ultrasound guidance is key to a successful procedure, as you can directly visualize the needle entering the pericardial space. The echocardiogram (echo) below shows a pericardial effusion. Not all effusions are circumferential like this one.



If the effusion is mostly posterior to the heart, bedside pericardiocentesis will not be possible or effective.

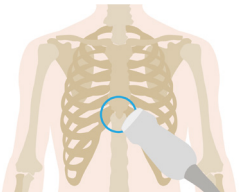
Prepare your supplies, including the following:

- Spinal needle or pericardiocentesis needle with a stylet in place
- 10 mL syringe
- 3-way stopcock
- Chlorhexidine or other sterilizing scrub
- Local anesthetic in a small syringe with needle
- Sterile drape
- Tubing connected to a collection bag for collecting the fluid

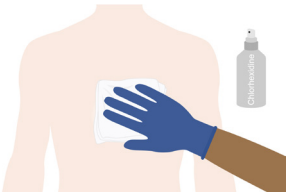
Steps to performing pericardiocentesis

If using ultrasound, first use the probe to find the pericardial space. The standard approach for pericardiocentesis is subxiphoid.

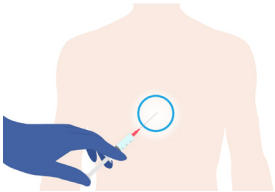
1. Locate the xiphoid process and, if using ultrasound, use the probe to find the pericardial space.
2. Sterilize the area with chlorhexidine or whatever is standard in your practice.
3. Use local anesthesia such as lidocaine to numb the anticipated path of the needle.
4. Insert the spinal or pericardiocentesis needle with the stylet in place. The approach is immediately below the xiphoid process, aiming the needle toward the patient's left shoulder and inserting it at a 45° angle to the skin.
5. Once the skin is punctured, remove the stylet.
6. Attach a 3-way stopcock and syringe.
7. Then, under ultrasound guidance, advance the needle towards the left shoulder while gently aspirating continuously with the syringe.
8. When the needle punctures the pericardium and passes into the pericardial space, the syringe will begin to fill with fluid.
9. Withdraw fluid into the syringe.



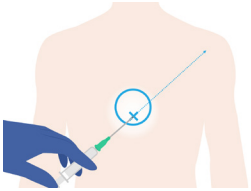
1. Locate xiphoid and pericardial space



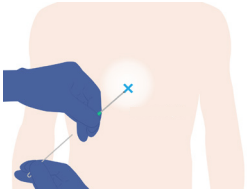
2. Sterilize



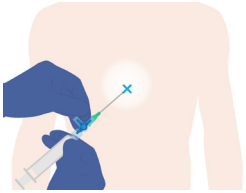
3. Anesthetize



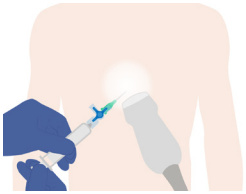
4. Insert needle with stylet in place



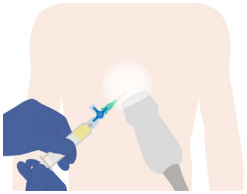
5. Remove stylet



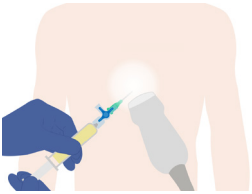
6. Attach 3-way stopcock and syringe



7. Advance while gently aspirating



8. Syringe will begin filling after pericardium is punctured



9. Withdraw fluid

With even a small amount of fluid removal, hemodynamics will often improve. It is sometimes recommended that if you are performing this procedure emergently but have an experienced provider on the way, you can remove 50 mL of fluid, or just enough to improve the hypotension, and then stop to wait for the experienced provider to reduce the risk of complications.

Send the fluid for testing

Removal of fluid addresses the immediate need to reduce the pressure, but don't forget this is a diagnostic as well as therapeutic procedure. Therefore, any fluid specimen should be evaluated for diagnostic clues.

The most important tests to perform include the following:

- Cell count to examine the number of red and white blood cells in the fluid
- Gram stain to identify any bacteria
- Acid-fast staining to look for tuberculosis
- Hematocrit to identify whether the effusion is bloody
- Cultures to see if any bacteria grow from the fluid

Other tests to evaluate for transudative versus exudative effusion include the following:

- Lactate dehydrogenase
- Total protein
- Glucose

In general, transudative effusions are pressure phenomena due to excess volume, like in the case of renal failure with missed dialysis sessions. Exudative effusions, on the other hand, indicate an inflammatory, infectious, or malignant process causing the effusion.

Pericardiocentesis is indicated for cardiac tamponade. It is not routinely recommended to drain pericardial effusions if the patient is stable. There are many ways the pericardial space can cause trouble, so there needs to be an important therapeutic reason to enter the space. Pericardiocentesis for diagnostic reasons is rare, and every attempt should be made to reach a diagnosis with alternate testing.

Leave it to experienced providers whenever possible!

There is a reason this procedure is only done by an experienced provider OR in a life-threatening emergency. The complication rate is 5–40% and includes serious things like puncturing a coronary artery, pneumothorax, inducing arrhythmia, or puncturing nearby structures like the liver or stomach!

An experienced provider will remove all the pericardial fluid, and then exchange the needle for a pericardial drain to provide continuous drainage under observation, in case the effusion recurs acutely.

Next, we will learn how to manage a pericardial drain.

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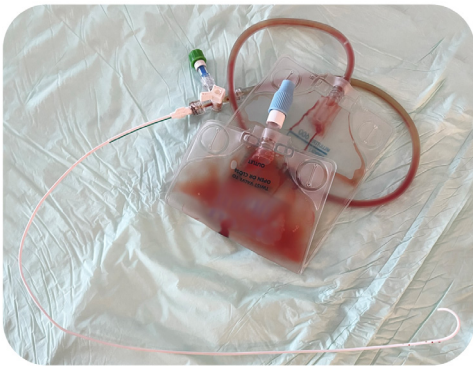
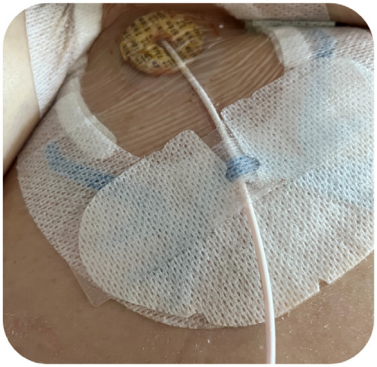
Managing a pericardial drain

Phew! You successfully diagnosed, stabilized, and treated a patient with cardiac tamponade. But the work isn't over yet.

After pericardiocentesis, many patients will have a pericardial drain placed by replacing the needle with a small-diameter catheter. The photo on the left shows what a pericardial drain looks like, after removal from a patient of course. The image on the right shows what the catheter looks like in place.



After removal



In place

Pericardial drain

The drain is placed in case the effusion reaccumulates. Once you've accessed the pericardial space, you don't want to do it a second time!

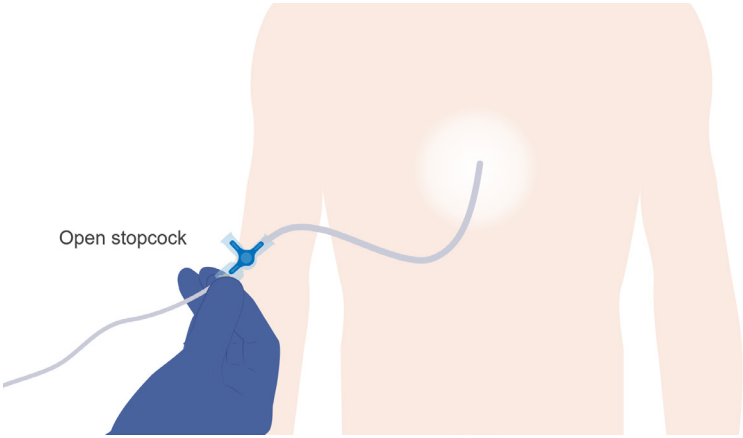
Pericardial drain management

Apart from being relatively rare, is a pericardial drain just like any other drain? The answer is no. This drain requires specialized care, usually in a unit with nursing expertise and experience with pericardial drains.

Different cardiologists or surgeons will have unique approaches to their drains, so I'll share my approach which I think represents best practice.

Allow it to freely drain

The drain can be allowed to freely drain with gravity with an open stopcock and tube continuously draining into a bag, which is placed lower than the level of the patient.



Fluid can also be manually drained with a syringe attached to the stopcock, although this is usually done only at the time of initial placement or if there is concern that fluid has reaccumulated or the drain is not functioning properly.

Do not flush the catheter routinely

I do not recommend flushing the catheter routinely, as there is a risk of introducing bacteria into the pericardial space even when using a sterile technique. I only recommend flushing the catheter if there is high suspicion it is clogged, such as when the effusion is bloody and clots are seen in the tubing.

Consider antibiotics

Antibiotics to protect from skin flora are sometimes given when the drain is in place.

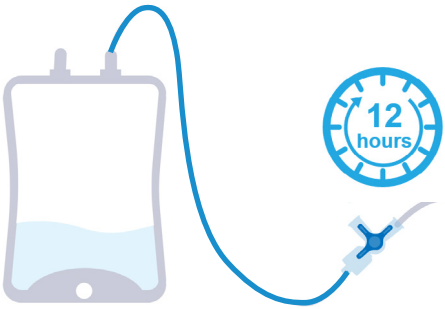
Record the volume drained

The amount of fluid draining should be recorded meticulously. It tells you the rate of fluid accumulation in the pericardium.

When to remove a pericardial drain

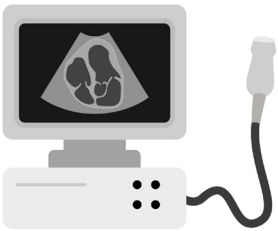
There is no established protocol, but in my practice, I recommend the following:

1. Notice once the total fluid drained in 24 hours is less than 50 mL.
2. Close the stopcock for 12 hours.
3. Repeat an echocardiogram to see if an effusion reaccumulates. You may end up doing many echos in this process.
4. If the drain is closed for 24 hours with no reaccumulation of fluid, then I recommend the removal of the catheter.

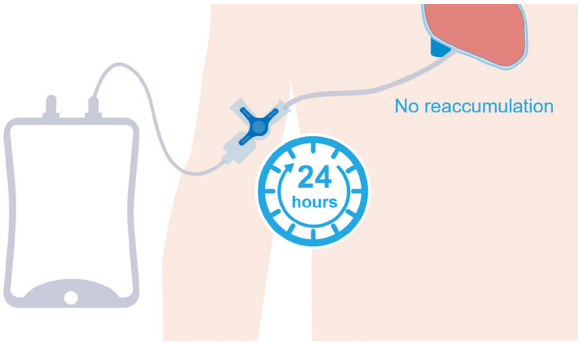


1. Notice if < 50 mL has accumulated in 24 hours

2. Close stopcock for 12 hours

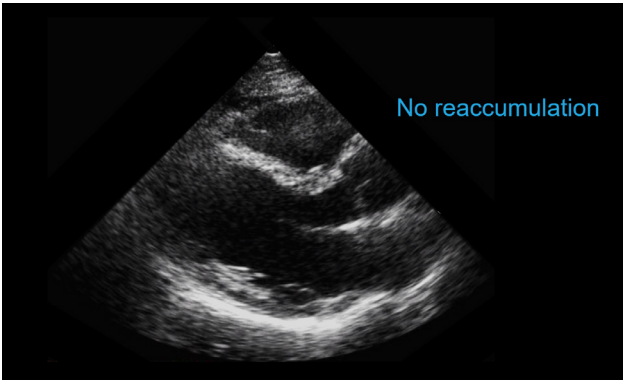


3. Repeat echo to check for effusion



4. Remove the catheter once there is no reaccumulation after 24 hours

The echo image below shows an example where there was no reaccumulation.



Timing of a pericardial catheter removal is a balance between the risk of infection from an indwelling catheter and the risk of repeat tamponade from fluid reaccumulation.

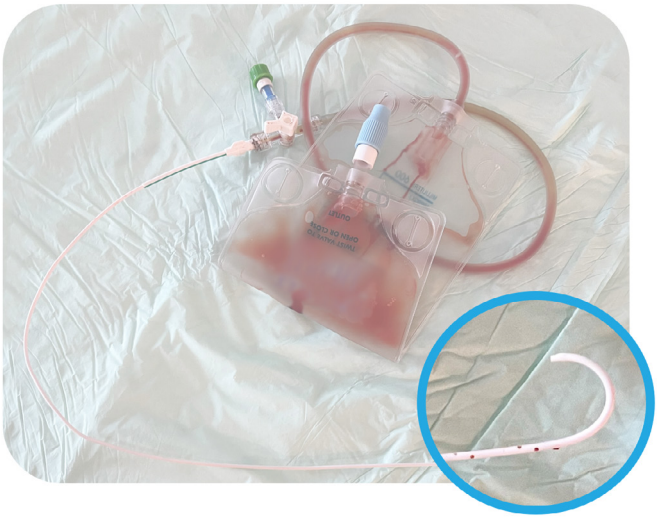
Pericardial drain removal

Removal of the catheter is relatively straightforward.

1. Cut any sutures keeping it in place.
2. Tug gently to ensure no fibrotic tissue has formed.
3. Remove the catheter in a single fluid motion, not too quickly to cause irritation of the pericardium but not too slowly.

It's very important to never readvance the catheter once it is pulled out, even by a centimeter. Pushing the unsterile catheter into the pericardial space is a great way to introduce infection.

If the catheter is a pigtail (i.e., it has a curve on the end), it can feel a little sticky during removal as the curved portion straightens at entry sites. Removal should not be painful but can cause a slight irritation in the chest.



Pigtail catheter

And that's it! Well done! You've learned the physiology, diagnosis, and management of cardiac tamponade. You're ready to save a life!

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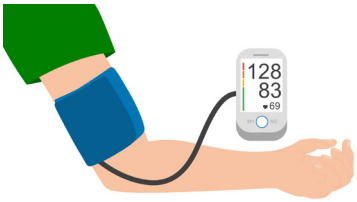
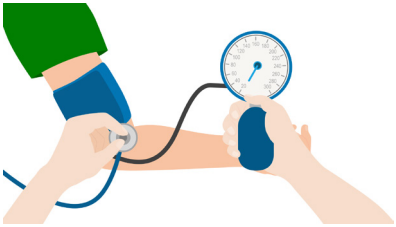
Appendix

Steps to measuring pulsus paradoxus

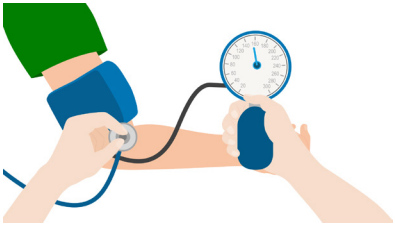
Remember that for a diagnosis of pulsus paradoxus, we are looking for a 10 mmHg or greater decrease in systolic blood pressure (BP) during inspiration compared to expiration.



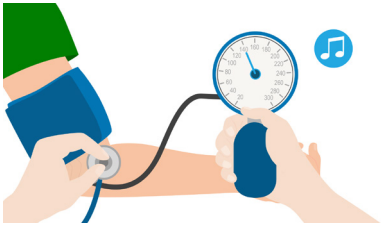
1. Take automated BP measurement.



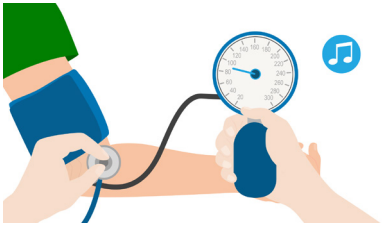
2. Prepare to take manual BP measurement.



3. Inflate ≈ 20 mmHg higher than the automated systolic measurement.



4. Release pressure very slowly and record BP at the first Korotkoff sound.



5. Continue to slowly release and record when Korotkoff sounds begin to occur at every heartbeat

Expiratory systolic pressure

– inspiratory systolic pressure

pulsus paradoxus

6. Calculate pulsus paradoxus

1. **Take an automated BP measurement**
   Start with getting an idea of the patient's average systolic blood pressure, usually from an automated measurement.
2. **Prepare to take a manual BP measurement**
   Get out the manual sphygmomanometer (i.e., BP cuff) and prepare to take the blood pressure yourself. Note that you don't need to ask the patient to alter their breathing pattern in most cases.
3. **Inflate the cuff approximately 20 mmHg *higher* than the automated systolic measurement.**
4. **Release the pressure very slowly and record the BP at the first Korotkoff sound.**
   Very slowly release the pressure while watching the needle and listening. Listen for the first time you hear a Korotkoff sound, which is the beat you hear with your stethoscope that is caused by blood rushing into the artery. Note the pressure corresponding to this first Korotkoff sound.
5. **Continue to very slowly release the pressure and record when you begin to hear a Korotkoff sound at every heartbeat.**
   You'll see the needle bounce with each heartbeat, but there will not be a sound with every beat, only intermittently. Wait until you hear a Korotkoff sound with *every* heartbeat or every bounce of the needle. Note the pressure when the sound with every heartbeat first occurred.
6. **Calculate the pulsus paradoxus measurement.**
   This is calculated by taking the BP you measured when you first heard a Korotkoff sound (i.e., the expiratory systolic pressure) minus the BP you measured when you began to hear Korotkoff sounds with every beat (i.e., the inspiratory systolic pressure).

In the lesson video, we demonstrated what this process sounds like in reality.

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Treating cardiac tamponade cheat sheet

Stabilize the patient

1. Intravenous (IV) fluid
   - ≤ 1–2 L of any isotonic fluid (normal saline preferred).
2. Vasopressors
   - Norepinephrine preferred if the patient has a central venous catheter.
   - Dopamine can be used if placing a central venous catheter will delay treatment.

Perform pericardiocentesis

See *Steps to performing pericardiocentesis* resource for more information.

Send the fluid for testing

- Cell count to examine the number of red and white blood cells in the fluid
- Gram stain to identify any bacteria
- Acid-fast staining to look for tuberculosis
- Hematocrit to identify whether the effusion is bloody
- Cultures to see if any bacteria grow from the fluid

Other tests to evaluate for transudative versus exudative effusion include the following:

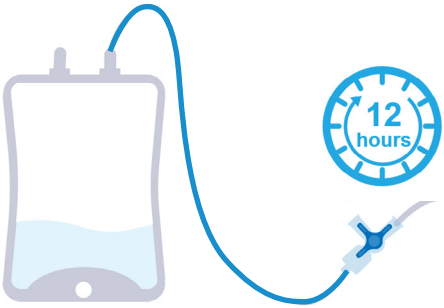
- Lactate dehydrogenase
- Total protein
- Glucose

Managing a pericardial drain

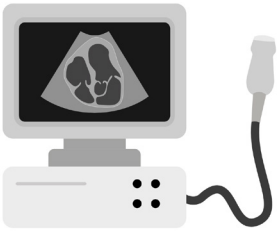
1. Allow it to freely drain with gravity with an open stopcock.
2. Do not flush the catheter routinely.
3. Consider antibiotics.
4. Record the volume drained.

Removing a pericardial drain

There is no established protocol, but in my practice, I recommend the following:

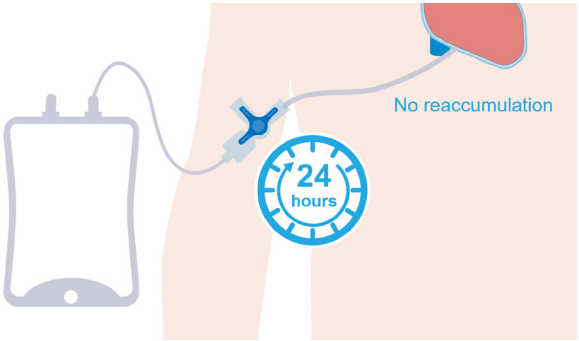


1. Notice if < 50 mL has accumulated in 24 hours



2. Close stopcock for 12 hours

3. Repeat echocardiogram (echo) to check for effusion



4. Remove the catheter once there is no reaccumulation after 24 hours

1. Notice once the total fluid drained in 24 hours is less than 50 mL.
2. Close the stopcock for 12 hours.
3. Repeat an echocardiogram to see if an effusion reaccumulates. You may end up doing many echos in this process.
4. If the drain is closed for 24 hours with no reaccumulation of fluid, then I recommend the removal of the catheter.

Steps

1. Cut any sutures keeping it in place.
2. Tug gently to ensure no fibrotic tissue has formed.
3. Remove the catheter in a single fluid motion, not too quickly to cause irritation of the pericardium but not too slowly.

It's very important to never readvance the catheter once it is pulled out, even by a centimeter. Pushing the unsterile catheter into the pericardial space is a great way to introduce infection.

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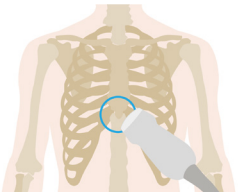
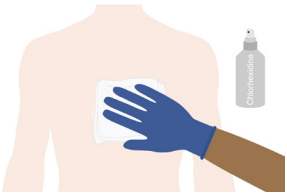
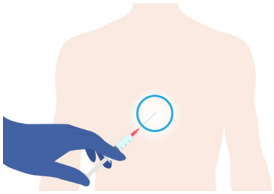
Steps to performing pericardiocentesis and drain placement guide



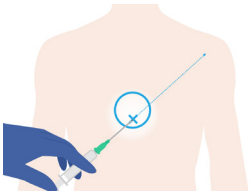
1. Locate xiphoid and pericardial space



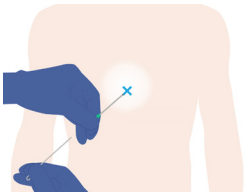
2. Sterilize



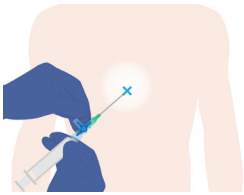
3. Anesthetize



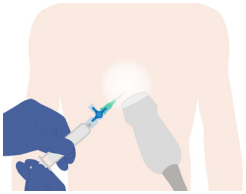
4. Insert needle with stylet in place



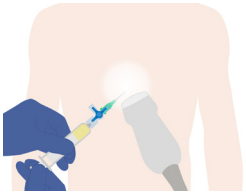
5. Remove stylet



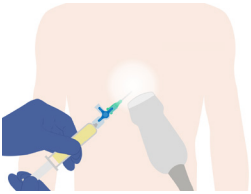
6. Attach 3-way stopcock and syringe



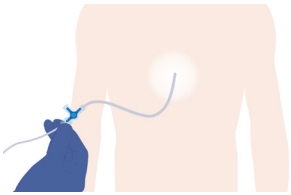
7. Advance while gently aspirating



8. Syringe will begin filling after pericardium is punctured



9. Withdraw fluid



10. Place pericardial drain

Supplies

- Ultrasound if available
- Spinal / pericardiocentesis needle with stylet
- 10 mL syringe
- 3-way stopcock
- Chlorhexidine or other sterilizing scrub
- Local anesthetic in small syringe with needle
- Sterile drape
- Tubing connected to a collection bag

Steps

1. Locate the xiphoid process and, if using ultrasound, use the probe to find the pericardial space.
2. Sterilize the area with chlorhexidine or whatever is standard in your practice.
3. Use local anesthesia such as lidocaine to numb the anticipated path of the needle.
4. Insert the spinal or pericardiocentesis needle with the stylet in place. The approach is immediately below the xiphoid process, aiming the needle toward the patient's left shoulder and inserting it at a 45° angle to the skin.
5. Once the skin is punctured, remove the stylet.
6. Attach a 3-way stopcock and syringe.
7. Under ultrasound guidance, advance the needle towards the left shoulder while gently aspirating continuously with the syringe.
8. When the needle punctures the pericardium, the syringe will begin to fill with fluid.
9. Withdraw fluid into the syringe.
10. Replace the needle with a small diameter catheter to allow the effusion to continue to drain.

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Reference list

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