Cardiogenic Shock vs Septic Shock

Can you do it with a broken heart, or do you have bad blood?

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Introduction

Cardiogenic shock and septic shock are two conditions that we see often in the ICU. Both are shock states, but they present and look very different. Patients in these shock states can get very sick, very quickly, but the good news is, you can fix them (no really, you can)!

Cardiogenic shock

In this shock state, the main problem we need to fix is that the patient has a broken pump that's just not squeezing the way it used to. And due to this broken pump, the patient's perfusion and circulation are impaired. Myocardial ischemia with or without resulting infarction, myocarditis, papillary muscle dysfunction, a ventricular septal defect, ventricular aneurysm, and acute mitral or aortic insufficiency are all potential causes of cardiogenic shock. Cardiogenic shock is also a potential complication of peripartum cardiomyopathy, where a pregnant or recently postpartum patient with a perfectly good heart suddenly goes into heart failure for no determinable reason. Regardless of the cause, patients in cardiogenic shock will present with decreased cardiac output, left ventricular dysfunction, sympathetic nervous system compensation, and myocardial ischemia due to the drop in cardiac output and resulting decrease in perfusion. As it turns out, you cannot do it with a broken heart.

Septic shock

In this shock state, the main problem that needs to be fixed is an infection. We know all too well how quickly an untreated UTI in an elderly patient can send them into septic shock, but after the past few years, sepsis due to a bad case of community-acquired pneumonia is nothing new. Any pathogenic organism can cause an infection bad enough to lead to septic shock, but gram-negative bacteria (e. Coli, pseudomonas, salmonella, etc.) tend to be the most common culprit. Invading pathogenic organisms cause the activation of

chemical mediators, which cause massive widespread vasodilation and functional hypovolemia. Unfortunately, Band-Aids don't fix septic patients.

Vigileo Numbers and a Quick Refresher on Hemodynamics

We utilize hemodynamic monitoring in the ICU to assess a patient's cardiac function and the effectiveness of the current treatment regimen. You may think it's black and white, but the gold standard for measuring intracardiac pressures, arterial pressure, and cardiac output is through a Swan-Ganz catheter, the Vigileo is less invasive and still provides a good estimate of what's going on inside the patient's heart with the pressure gradients. By looking at numbers such as the central venous pressure (CVP), CO (cardiac output), systemic vascular resistance (SVR) and stroke volume (SV) we can determine what changes we need to make, if any.

Hemodynamic number (and normal range)	Cardiogenic shock	Septic shock
Heart Rate (60-100 bpm)	↑	↑
MAP (65-105 mmHg)	↓	↓
CO (4-8 L/min)	↓	↑ or ↓*
SV (60-100 ml/beat)	↓	↓
CVP (1-6 mmHg)	↑	↓
SVR (800-1200 dynes/sec/cm^2)	↑	↓ or ↑*

*depends on which phase of sepsis the patient is in

Cardiogenic shock: As you can see in the table, patients in cardiogenic shock will usually show an increase in HR, CVP, and SVR, but a decrease in MAP and CO. These changes are due to the broken pump not being able to effectively pump blood out of the heart, causing increases in preload and afterload. The heart rate increases because the tissues aren't getting perfused enough, so the heart tries to compensate by beating faster, thinking that will make blood move around quicker. But that only decreases stroke volume more, because the heart can't effectively pump blood at a normal rate, much less at a faster rate. Preload is increased, which is reflected by an increase in CVP, due to blood backing up in

the right side of the heart since the left side isn't pumping it out effectively. The increase in afterload, reflected by an increase in SVR, is due to the body thinking that constricting the blood vessels will help get the blood to the tissues faster. However, the trouble is with the pump, not necessarily the vessels. Increasing the SVR just increases the resistance that the already tired heart is trying to pump against. It makes sense that CO will be decreased, since cardiac output equals heart rate times stroke volume, and SV is severely decreased in cardiogenic shock due to the decrease in ejection fraction from the broken pump. MAP being decreased also makes sense, since the patient will be hypotensive thanks to impaired ventricular ejection.

Septic shock: Septic shock can be divided into two phases, the hyperdynamic/warm phase, or early septic shock, and the hypodynamic/cold phase, or late septic shock. In both phases, patients will present with a rapid HR, decreased MAP, decreased SV and decreased CVP. However, CO and SVR will vary depending on which phase of septic shock the patient is in. In the hyperdynamic phase, patients will present with decreased SVR due to the activation of chemical mediators that cause widespread peripheral vasodilation. Their CO will increase due to the body attempting to improve perfusion by pumping more blood out to the body; however, the blood has a problem getting to where it needs to go due to the widespread vasodilation. In the hypodynamic phase, the body isn't able to compensate much more, so CO drops, causing worsened hypotension and even more inadequate tissue perfusion. The blood vessels will attempt to constrict to improve perfusion, so SVR will increase as a result, but this compensatory effort isn't enough to prevent multiorgan system failure. This is why it's important to identify sepsis early, before your patient decompensates and everything spins out of control.

Treatments

Cardiogenic shock: You don't need to be a mastermind to fix cardiogenic shock - fix the pump and the underlying cause of the broken pump! If the cardiogenic shock is due to a MI, then the patient needs cardiac catheterization and stent placement, or subsequent

CABG if multivessel disease is the cause. If the problem is a busted valve, replace the valve. But until then, support the system with vasopressors, inotropes, and osmotic diuretics. Nitroglycerin can be given to decrease preload, but giving vasopressors and vasodilators concurrently to a hypotensive patient is a delicate situation. Patients may need supplemental oxygen due to perfusion being altered and potentially may require intubation and mechanical ventilation. For severe cardiogenic shock, devices like an intra-aortic balloon pump (IABP) or Impella may be necessary to improve coronary perfusion and decrease afterload. An LVAD and/or ECMO could potentially be necessary.

Septic shock: Long story short, we need to treat the infection! Start IV antimicrobials (start with broad spectrum antibiotics and then narrow down based on culture and sensitivity results), fill the tank with IV fluid resuscitation, give oxygen as needed, and use vasopressors if indicated. Rember to draw blood cultures BEFORE starting the patient on IV antibiotics! Ideally, we'd like two sets of blood cultures to be drawn prior to administering the first dose of antibiotics, but if the patient's vasculature isn't ideal, so it goes. Since treating the infection is the priority, antibiotic administration shouldn't be delayed due to issues with obtaining blood cultures. The first dose of antibiotics should be administered within an hour of identifying that a patient is septic. If your patient has multiple different antibiotics ordered, make sure that you utilize Micromedex instead of navigating the labyrinth yourself to confirm compatibility (or incompatibility) before Y-siting them together! And if Micromedex shows conflicting compatibility, click on the details to check concentrations and dilutants, especially if Vancomycin is ordered, because Vanc will forever and always only be compatible in certain concentrations and dilutants. Also, keep an eye on the patient's pending culture and sensitivity results, because once the infectious organism is identified and sensitivities determined, that's when it's time to narrow the spectrum of antibiotics.

Before you start vasopressors on a septic patient, make sure that you've adequately fluid resuscitated them first! Trying to constrict vessels with nothing in them won't fix your patient's hypoperfusion or reverse their shock state, even in their wildest dreams. The goal for fluid resuscitation is to administer a total of 30 mls/kg of isotonic crystalloids like

lactated ringers or normal saline within three hours. If fluid resuscitation is successful, the patient's MAP should improve to greater than 65 mmHg and their lactic acid level should drop to less than 4. If your patient remains hypotensive despite adequate fluid resuscitation, it's probably time to change tactics and start some vasopressors.

Cardioactive drips for shock states: See chart for dosing and effects on numbers

Inotropes: These increase contractility of the heart, improve the squeeze, and are mostly used for treating cardiogenic shock.

- **Dobutamine** has inotropic (helps the squeeze), chronotropic (increases the heart rate), and vasodilatory effects. Though it's most useful in patients with low cardiac output due to decompensated heart failure and cardiogenic shock, it is also useful for patients with sepsis induced myocardial dysfunction and symptomatic bradycardia. Instead of boosting CO by increasing the patient's heart rate, dobutamine targets and raises the stroke volume instead. It should also improve renal blood flow and thus increase the patient's urine output. Due to its vasodilatory effects and potential to cause hypotension, dobutamine is most appropriate for patients who are normotensive with good peripheral vascular tone. Administering dobutamine concurrently with a vasopressor like levophed or neosynephrine will help offset its vasodilatory effects. Though dobutamine is less likely than dopamine or levophed to cause tachycardia, it isn't completely innocent either, since patients, particularly those that are already hypovolemic, will frequently have mild increases in heart rate.
- **Milrinone** has primarily inotropic and vasodilatory effects. It's useful for patients with right ventricular failure and low cardiac output, due to its ability to increase cardiac output without causing an increase in heart rate or myocardial oxygen demand. If your cardiogenic shock patient is already tachycardic, milrinone may be preferred over dobutamine. We forget that milrinone exists, because we don't use it often, but it is helpful for decreasing preload, afterload, and SVR. However, since

it's metabolized by the liver and excreted by the kidneys, it may not be the most appropriate choice for patients in hepatic or renal failure, due to the potential for the drug to accumulate.

Vasopressors: These constrict the blood vessels to raise blood pressure. They are used in both cardiogenic and septic shock, but some vasopressors are only indicated for one type of shock, not both. So here are some clinical pearls regarding vasopressor choice! Remember that the goal of maintaining a MAP greater than 65 mmHg isn't an arbitrary number; vital organs like the heart, brain, and kidneys require a patient's MAP be at least 60-70 mmHg for adequate perfusion.

- **Dopamine** is indicated for septic shock, but it should NOT be your first choice. It's actually more useful for bradycardia-induced hypotension and is effective in treating heart failure that's causing cardiogenic shock, as well as correcting hemodynamic imbalances during shock. If you have a patient that is still hypotensive despite fluid resuscitation, starting dopamine may be indicated. However, be aware that dopamine does have a reputation for causing arrhythmias and tissue necrosis if it infiltrates.
- **Epinephrine's** effects will change based on the dosage. At both low and high doses, both cardiac output and the patient's heart rate will increase, and tachycardia is likely. However, SVR will not always be affected by low doses; it will increase with higher doses due to the alpha-adrenergic effects. Use caution when putting a patient prone to arrhythmias on an Epi drip, they may not tolerate it! In addition, even though Epi will boost CO and increase myocardial oxygen delivery, it also increases myocardial oxygen consumption and may not be the most appropriate option for patients with active or suspected myocardial ischemia and/or infarction. You may also want to think twice before putting a patient with known or suspected gastrointestinal ischemia on an Epi drip though, because Epi may cause new GI ischemia, or worsen already existing GI ischemia. However, combining it with a dobutamine drip may diminish this effect.

- **Levophed** (norepinephrine) is the first line option for septic shock because it primarily vasoconstricts and increases SVR. However, because it does increase contractility and slightly increase cardiac output, it is indicated for cardiogenic shock as well. If your patient is hypotensive with a decreased SVR, levo may be the one you want to try first. Levo works on alpha1 receptors more than beta1 receptors, and due to its effects on heart rate, tachyarrhythmias are possible. If your patient goes into a tachyarrhythmia, then it may be time to say so long, Levo.
- **Neosynephrine** (phenylephrine) is a sympathomimetic adrenergic agent, it activates the sympathetic nervous system, and as a result, constricts blood vessels and raises blood pressure. Keep your eyes open and watch your patient's heart rate though, because Neo can cause reflex bradycardia due to increased vagal tone from direct baroreceptor stimulation. Neo should not be used in patients with heart failure, because the increases it causes in SVR may result in a decrease in CO. It also should not be utilized for patients with known/suspected bowel ischemia.
- **Vasopressin** is the second line option for septic shock, and particularly useful for patients that don't respond to catecholamines like Levo, Epi, Dopamine, or Dobutamine. If your septic patient's blood pressure is in the tank despite high doses of Levo, start them on some Vaso, and you might be able to get yourself out of the woods and in the clear. Vaso is also helpful for patients with upper GI bleeds due to gastric and esophageal varices due to its ability to reduce portal pressures. Using vasopressin independently of other vasopressors does not provide significant effects, as it's actually a relatively weak vasoconstrictor, but it hits different and has additive effects when used as a secondary or supplemental agent alongside a catecholamine.

Vasodilators: It may seem contraindicated to give a vasodilator to a patient in a shock state, but for patients in cardiogenic shock, sometimes a vasodilator is needed to help decrease preload and take some strain off the busted pump by decreasing SVR and afterload.

- **Nitroglycerin** is used to decrease preload and afterload in patients with cardiogenic shock due to left ventricular failure. If the cardiogenic shock is due to an underlying MI, Nitro will also help dilate the coronary vasculature and improve coronary perfusion. You might think, "this is absurd" when you see an order for a Nitro drip when your cardiogenic shock patient is already on a vasopressor, and though it may seem counterintuitive, two is better than one in situations where you need to attack a problem from different angles to best support the patient's circulation. Infusing nitroglycerin and neosynephrine concurrently in the setting of acute myocardial ischemia is a combination proven to decrease preload and vasodilate the coronary vasculature while also maintaining arterial blood pressure.
- **Nipride** (nitroprusside) is another vasodilatory agent we can utilize to help decrease preload and afterload. It acts on both veins and arteries and can actually increase cardiac output due to decreasing left ventricular afterload and thus the strain on the left ventricle itself.

The Great War: A septic patient with chronic congestive heart failure

No, this isn't a hoax, since literally anyone can become septic. Call it what you want, but I'll just call it what it is, a nightmare scenario for intensivists and cardiologists alike. Because what are you supposed to do when Grandma Janie with an ejection fraction of 25% shows up from the SNF next door, covered in feces, with a raging UTI turned urosepsis due to e. Coli (which you won't be aware of for a few days until the cultures result). You don't want to aggressively fluid resuscitate this patient, she already sounds a little crackly and has +2 pitting edema at baseline, but her MAP is 55 mmHg when she's awake, and 45-50 when she's asleep. Not to mention that her lactic acid level is 5.3 mmol and her WBC count is 18k. Oh and it's right after shift change and the ED just brought her up without really calling report because they want to go home. Looks like you're on your own, kid, but you can face this!

If you've made it this far, and you thought to yourself, "hmm, this seems a little more entertaining than usual," and you think you've picked up on a few things…what if I told you none of it was accidental?? Let me know if you can pick out 20+ references, and you can get a prize ;)

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Reference List

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