Approach to Shock

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TAAAC-EM Resident Teaching

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This session will be recorded

We are recording this Zoom session so that it can be watched again at your convenience, and so that we can share it with your colleagues who were not able to join us today.

If you would prefer that this recording **not** be shared with your EM colleagues, please email amcknight@ghem.ca within 24 hours of the session.

We will share the presentation slides and other materials (journal articles, etc.) by email; you will have access to all materials regardless of whether the recording is shared.

Please also note:

The information in this presentation and the video recording is up to date as of the date it was recorded (September 15, 2020).

It has not been updated to include any subsequent advances in practice, and the information presented in this video does not replace hospital, health centre, or governmental guidelines.

Acknowledgements

- Dr. Nazanin Meshkat
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Objectives

- Define shock
- Classify etiologies of shock
- Discuss clinical clues for shock and differentiating different types of shock
- Discuss resuscitation strategies for different etiologies of shock
- Focus on septic shock

My objectives for today are:
To talk about what shock is
And how to tell your patient is in shock clinically
Discuss clues on physical exam and investigations that might help you to think through and figure out the etiology of your patient's shock
Discuss different resuscitation strategies for different types of shock that we commonly encounter in the ER
And then go into greater detail specifically about septic shock since this is still one of the most common types of shock you will manage

I will be asking you lots of questions and hope you will participate. and feel free to ask questions at any time since I often find it works better than waiting until the end. Dr. Jennifer will keep an eye on the chatbox for those of you whose connection will only allow questions that way, or who would be more comfortable typing out your questions.

Definition of Shock

Inadequate perfusion (blood flow) to meet metabolic cellular demands

→An imbalance between O_2 delivery and demand

→Cellular dysfunction and damage

→Organ dysfunction and damage

Why should you care?

High mortality - 20-90%

Early on the effects of O_2 deprivation on the cell are *REVERSIBLE*

Early intervention reduces mortality

- Can prevent the detrimental effects of O_2 deprivation

Blood flow is dependent on

- Cardiac output
- Volume of blood
- Blood vessel diameter and length
- Compliance
- Blood viscosity

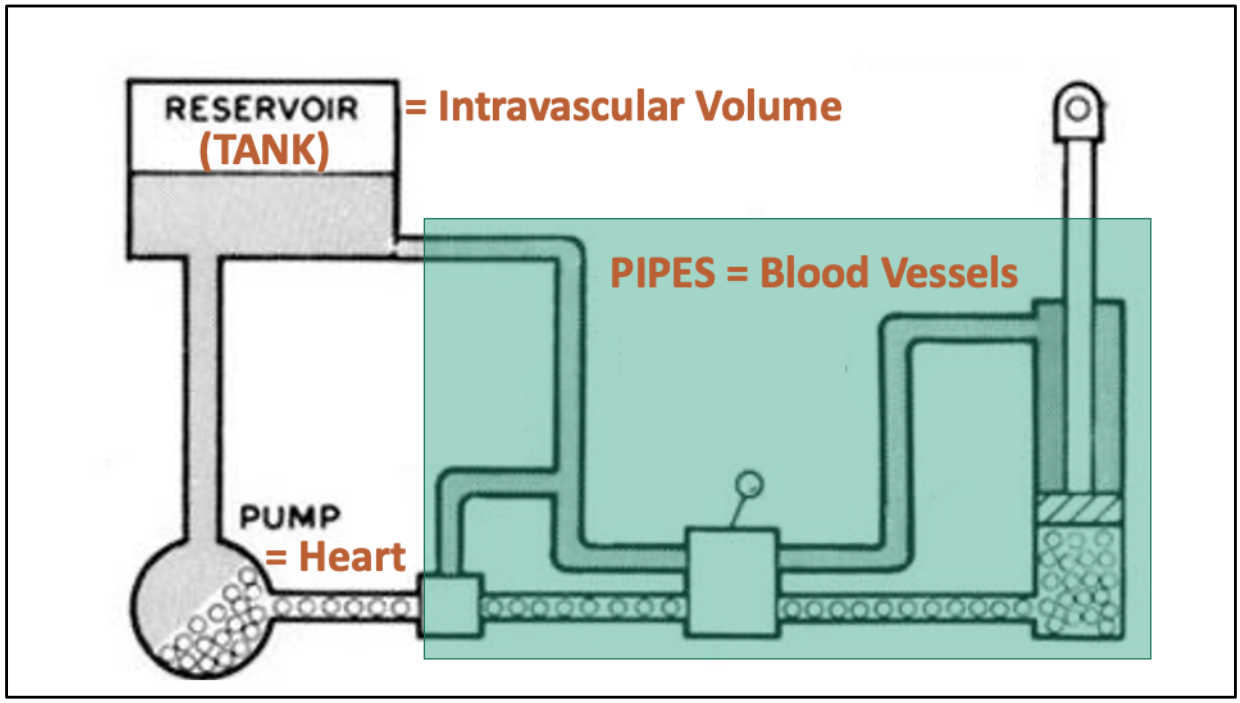
For our purposes in managing shock, it's really only the first 3 that matter, because for all intents and purposes, blood vessel length and blood vessel compliance are not factors that change acutely. When we're talking about blood viscosity, we're not talking about someone being dehydrated, we're talking about blood that is viscous with components such as proteins (like in multiple myeloma) or with cells like in polycythemia rubra vera, or hyperleukocytosis in leukemias.

Shock – A Circulatory Problem

Tank = Intravascular Volume
Pipes = Blood vessels
Pump = Heart

Now that we've defined Shock as a Circulatory Problem, we can use an analogy to think about the ways that poor perfusion can happen in circulation.

It's either going to be a problem with the Tank, the Pipes or the Pump.
When we talk about the tank, we mean Intravascular Volume,
When we talk about the pipes, we mean the Blood Vessels, and
When we talk about the pump, we mean the heart



For those of you who are more visual, this image might be a helpful way to think about and work through some of these concepts.

Blood flow is dependent on

- Cardiac output - **PUMP**
- Volume of blood - **TANK**
- Blood vessel diameter and length - **PIPES**
- Compliance
- Blood viscosity

If we were to map the concept of Pump, Tank and Pipes back onto the factors that affect blood flow or perfusion from the previous slide, this is what it would look like.

How does blood pressure fit in?

Tissue perfusion is determined by Mean Arterial Pressure (MAP)

$$MAP = \frac{(2 \times diastolic) + systolic}{3}$$

MAP = CO x SVR

HR x Stroke Volume

In any type of SHOCK tissue perfusion is determined by MAP - which is used as a measure of perfusion (MAP as a measure of perfusion is only a surrogate measure, and is not 100% accurate - however sometimes it's all we have to go by)

MAP = cardiac output multiplied by systemic vascular resistance = 2/3 systolic + 1/3 diastolic pressure
SVR is governed by the vessel length, blood viscosity, and vessel diameter
CO = heart rate (HR) multiplied by Stroke Volume (SV)

Etiologies of Shock

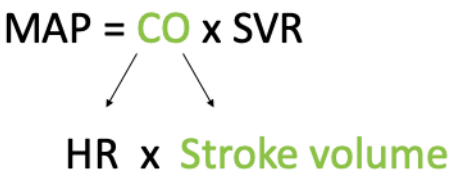
- Hypovolemic - TANK
- Distributive - PIPE
- Cardiogenic - PUMP (Intrinsic)
- Obstructive - PUMP (Extrinsic)

Can you list the different "types" of shock, or how shock can be classified?

If we were to take our analogy further, we can break these down into Tank, Pipe, and Pump problems. So:
-hypovolemic shock is a TANK problem
-distributive shock is a PIPE problem
-cardiogenic shock is an intrinsic PUMP problem (something is wrong with the pump itself)
-obstructive shock is an extrinsic PUMP problem (the pump is fine but something is preventing it from pumping enough blood into the circulation)

Hypovolemic Shock: TANK problem

Heart pumps well, but not enough blood volume to pump



Notes:
-Hypovolemic shock is a consequence of decreased preload due to intravascular volume loss.
-The decreased preload diminishes stroke volume, resulting in decreased cardiac output (CO).

Hypovolemic Shock: Pathophysiology

Normal	Hypovolemic
MAP = CO x SVR	MAP = ↓CO x SVR MAP = ↓CO x ↑ SVR ↓MAP = ↓↓CO x ↑ SVR

Notes:

-The way our bodies compensate is the BaroRc sense the decreased cardiac output and lead to increased SVR in an effort to compensate for the diminished CO

-The vasoconstrictive mechanisms (I.e. the increase in systemic vascular resistance) compensate for decreased tissue perfusion by redirecting blood from the periphery to the vital organs, thereby maintaining coronary, cerebral, and splanchnic perfusion.

Hypovolemic Shock – Causes

- Hemorrhage (blood loss)
  - Traumatic
  - Non-traumatic – GI bleed, ruptured AAA, ruptured ectopic
- Dehydration (fluid loss)
- Adrenal crisis*

Hemorrhagic shock, which can be traumatic or non-traumatic.
Examples of non-traumatic blood loss can include upper and lower GI bleeds, ruptured AAAs, and ruptured ectopics. You want to keep some of these "invisible bleeds" at the back of your mind.
Dehydration. This is intravascular depletion from processes like gastroenteritis or DKA.
Adrenal crisis. Adrenal crisis can occur either because of an acute illness in someone known to have adrenal insufficiency, in someone with an acute illness who is on long-term steroids and needs extra stress steroids, or can occur as a first-time presentation in someone without any history of adrenal problems. It occurs because there is a lack of production of glucocorticoids and mineralocorticoids, and I have a star beside it because it can be both a tank and a pipe problem.

Distributive Shock: PIPE problem

Heart pumps well, but there is peripheral vasodilation due to loss of vessel tone

MAP = CO x SVR

HR x Stroke volume

Notes: Distributive (vasodilatory) shock is a consequence of severely decreased SVR.

Distributive Shock: Pathophysiology

Normal	Distributive
MAP = CO x SVR	MAP = CO x ↓ SVR MAP = ↑CO x ↓ SVR ↓MAP = ↑CO x ↓↓ SVR

Notes: The way our bodies compensate for the diminished SVR is the cardiac output increases, with increases in both heart rate and stroke volume

Distributive Shock – Causes

- Septic
- Anaphylactic
- Neurogenic
- Adrenal crisis

Under distributive shock, which is a PIPE problem, you can have septic, anaphylactic, and neurogenic shock. And as mentioned, adrenal crisis fits under here too.

Cardiogenic Shock: *intrinsic* PUMP problem

Heart fails to pump blood out

MAP = CO x SVR

HR x Stroke Volume

Notes:
Cardiogenic shock is a shock state that occurs as a consequence of cardiac pump failure, resulting in decreased cardiac output (CO). Pump failure can occur both as a result of an abnormality of the Heart rate or the Stroke volume

Cardiogenic Shock: Pathophysiology

Normal	Cardiogenic
MAP = CO x SVR	MAP = ↓CO x SVR
	MAP = ↓CO x ↑ SVR
	↓MAP = ↓↓CO x ↑ SVR

Notes:

-Baroreceptors sense the decreased cardiac output and leads to increased SVR in an effort to compensate for the diminished CO

-The vasoconstrictive mechanisms (I.e. the increase in systemic vascular resistance) compensate for decreased tissue perfusion by redirecting blood from the periphery to the vital organs, thereby maintaining coronary, cerebral, and splanchnic perfusion.

Cardiogenic Shock – Causes

- Valve
  - critical AS, advanced rheumatic heart dz, blown valve
- Electrical
  - arrhythmia
- Muscle
  - infarction, CHF, myocarditis, toxicologic

Any number of abnormalities with the heart could cause cardiogenic shock.
It could be valvular – as in critical aortic stenosis, critical congenital heart disease or advanced rheumatic heart disease, “blown valve” (a malfunctioning valve that was previously surgically repaired or replaced)
It could be electrical – which can include any brady or tachyarrhythmia causing impaired cardiac output.
Or it could be from the myocardium – such as a myocardial infarction. It could also be worsening heart failure, a cardiomyopathy that is acute (secondary to a virus, or Takotsubo), or it could be toxicologic (For example, someone who has overdosed on beta blockers).

Obstructive Shock: *extrinsic* PUMP problem

Heart pumps well, but the output is decreased due to an obstruction (in or out of the heart)

MAP = CO x SVR

HR x Stroke volume

The heart itself is functioning, but the cardiac output is decreased because of something blocking blood outflow from the heart.
Notes: The blood outflow from the heart is decreased either because there is decreased return to the heart (due to an obstruction) or "obstructed" as the blood leaves the heart the stroke volume diminishes, with the overall effect of decreasing the cardiac output

Obstructive Shock: Pathophysiology

Normal	Obstructive
MAP = CO x SVR	MAP = ↓CO x SVR
	MAP = ↓CO x ↑ SVR
	↓MAP = ↓↓CO x ↑ SVR

Notes:

-Like cardiogenic shock, the baroreceptors sense the decreased cardiac output and lead to increased SVR in an effort to compensate for the diminished CO

-The vasoconstrictive mechanisms (I.e. the increase in systemic vascular resistance) compensate for decreased tissue perfusion by redirecting blood from the periphery to the vital organs, thereby maintaining coronary, cerebral, and splanchnic perfusion.

Obstructive Shock – Causes

- Outflow obstructed
  - Cardiac tamponade
  - Massive pulmonary embolus
  - Aortic dissection
  - Tension pneumothorax
- Venous return obstructed
  - Vena cava syndrome
  - Sickle cell splenic sequestration

Entities that cause outflow obstruction would decrease stroke volume.
Entities that cause decreased venous return would decrease preload.

Shock – Clinical Presentation

- Altered mental status – anxiety, agitation, delirium, obtundation/coma
- Tachypnea
- Tachycardic
- Hypotensive
- Warm/Cool extremities
- Delayed capillary refill
- Mottled
- Low urine output (<0.5mL/kg/h)

Blood pressure may be normal!

Now let's talk about how your assessment can help you diagnose and differentiate between the different types of shock.

What are some of the clues that a patient may be presenting in shock?
Altered mental status
Tachypnea (from metabolic acidosis)
Tachycardic
Hypotensive
Cool extremities
Delayed capillary refill
Mottled
Decreased urine output
We're basically looking for signs of end organ dysfunction due to poor perfusion and oxygen delivery to tissues

Empirical Criteria for Shock

4 out of 6 criteria have to be met

1. Ill appearance or altered mental status
2. Heart rate >100
3. Respiratory rate > 20 (or PaCO2 < 32 mmHg)
4. Urine output < 0.5 ml/kg/hr
5. Arterial hypotension > 30 minutes duration, continuous
6. Arterial base deficit < -4 or Lactate > 4

Rosen's, 9th Edition

This is from Rosen's and is meant to help standardize the diagnosis of shock, but it is not evidence-based.
4 of these 6 criteria have to be met to define shock.

Diagnosing and Differentiating Types of Shock

- Hx
- Physical Exam
  - Point-of-care ultrasound (PoCUS)
- Investigations

The most important thing to help you identify what type of shock your patient has (hypovolemic vs. distributive vs. cardiogenic vs. obstructive) is still going to be your history. Looking at the person's history of presenting illness, their past medical history and risk factors for illness, and medications will give you a clue and starting point almost all of the time.

Your physical exam is going to include signs that we just talked about, essentially you're looking for are signs of lack of end organ perfusion.
Obviously, your vital signs will give you clues.
Most of your patients will present with tachycardia, though this won't be true with neurogenic shock, cardiogenic shock caused by a bradyarrhythmia, or if your patient is on a beta-blocker.
Hypotension or borderline systolic blood pressure. Remember that in certain patients, those who normally have hypertension, the blood pressure they have may be relatively hypotensive for them. You don't need a systolic blood pressure below 90 or a mean arterial pressure of 65 before you start suspecting or acting on a presentation of shock.
Fever may make you suspicious of septic shock, though infection is not the only reason for a fever.

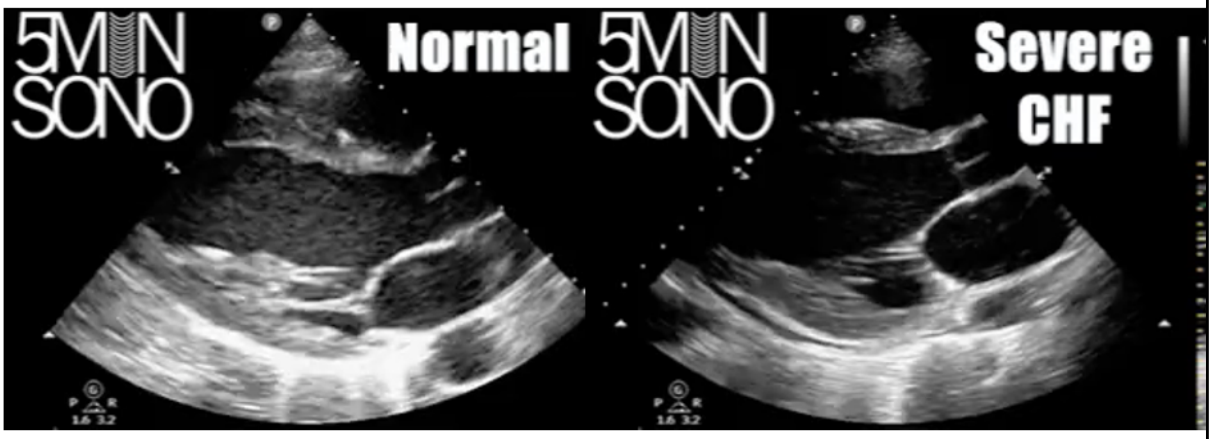
In addition, you want to do a thorough head-to-toe exam to look for other clues that might help you figure out what type of shock someone has. This includes a good skin exam, including turning patients with mobility issues to look for sacral or decubitus ulcers, which can be a source of infection and septic shock.

Point-of-care ultrasound (or PoCUS), because it really has in some ways revolutionalized the way we differentiate between types of shock in emergency medicine and critical care.
There is a caveat though: PoCUS should be considered not as an investigation, but as an extension of your physical exam and as another data point to help you figure out what your working diagnosis is. If you can't generate a good image or can't interpret the image, you should continue working up and managing your patient as you would if you didn't have ultrasound.

The scans I want to focus are the subxiphoid cardiac scan and the IVC which are most helpful to help us figure out whether we have a tank or pump problem.

PoCUS – Cardiac (PUMP)

How is the heart contracting? (Poorly vs. hyperdynamic)

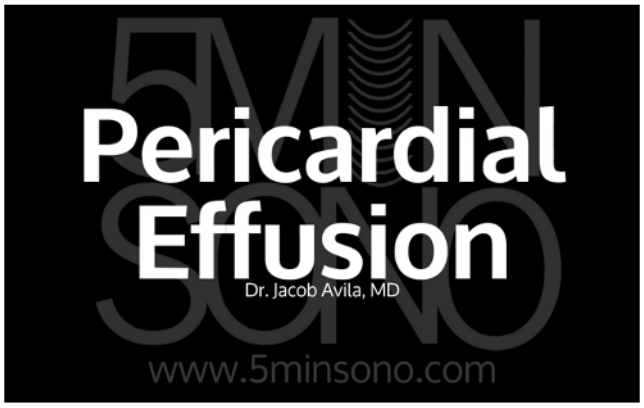


We'll first look at the subxiphoid scan of heart.
There are only 2 questions that we are going to ask with this scan. The first is whether or not the heart is grossly contracting normally. If not, be suspicious that there is a PUMP problem that is intrinsic to the pump.
(This is actually a parasternal long axis view of the heart, not a subxiphoid view – I wasn't able to find one, but they serve to illustrate gross cardiac function nonetheless.)

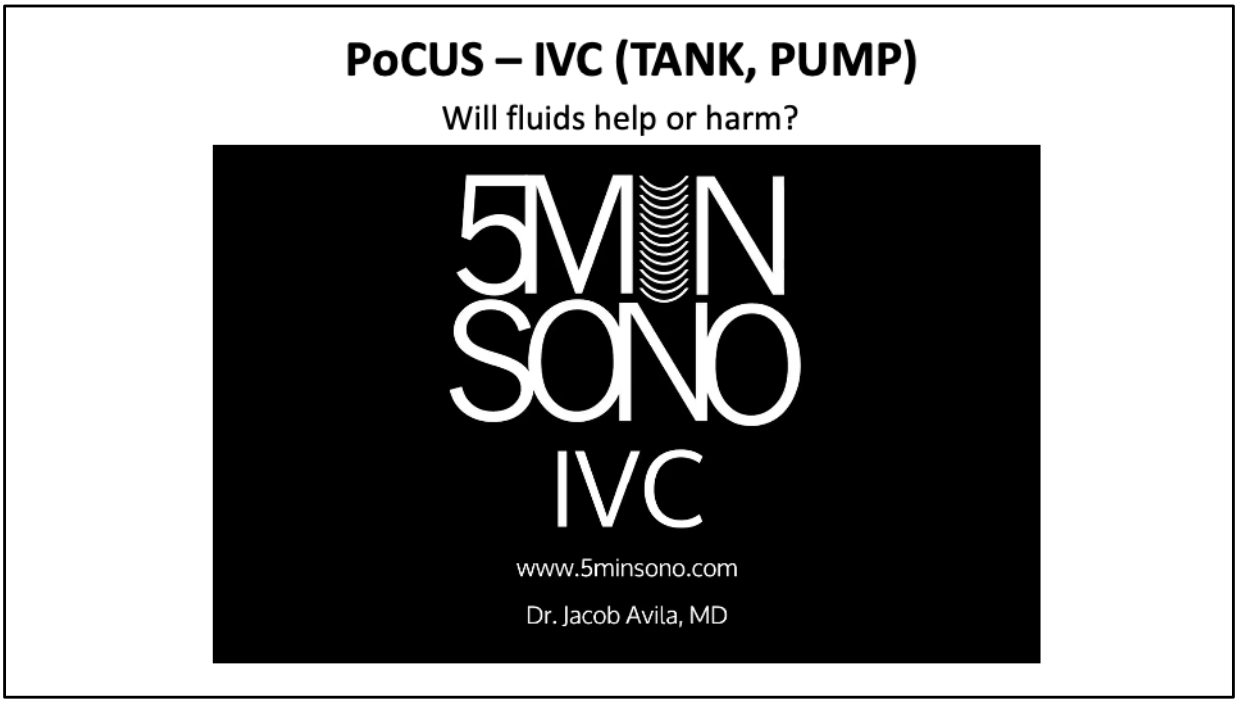
Links: no longer able to find the original videos, but here is another from 5 Min Sono about cardiac function:
https://www.coreultrasound.com/basic-cardiac-function/

PoCUS – Cardiac (PUMP)

Is there a pericardial effusion?



The second question we ask is whether there is a pericardial effusion.
Of course, you have to take this into clinical context. Not all pericardial effusions are acute, and not all of them cause tamponade.
Link to: Pericardial Effusion – play from start until 1min 30sec
(https://www.coreultrasound.com/pericardial-effusion/)



The interpretation of IVC is one of the most controversial in point of care ultrasound and the literature continues to evolve. However, it can still give us some good information.
The IVC is going to tell you about the tank (just as the JVP on your clinical exam is going to). However, just like the JVP, it can also tell you about the pump.
It basically tells you whether the patient would 1) benefit from more fluids, and 2) whether it is safe to give the patient more fluids.

If the IVC is under 1.2cm at its widest in the AP diameter or it collapses by more than 40% with respiration, then that patient is considered to be volume deplete. That is, giving them fluids is going to help improve their shock.

If the IVC is greater than 2.3cm at its widest in the AP diameter or collapses by less than 15% during respiration, then that patient is considered to be volume-overloaded and is unlikely going to benefit from more IV fluids, and fluids may in fact harm them. For example, you might see this in a patient with cardiogenic shock who is in heart failure. **That is a patient in whom you will likely choose to start vasopressors in with minimal fluid resuscitation.**

If the IVC is between 1.2cm and 2.3cm and the respiratory variability is between 15-40% when you're eyeballing it, it essentially is considered "normal" and doesn't really give you additional information.

Link to IVC video: https://www.coreultrasound.com/pericardial-effusion/ (Play from 2min 48sec to 3min 19sec, or watch whole video)

Remember take these findings in clinical context. A plethoric IVC may not be an acute finding – for example, in Ethiopia, there is lots of rheumatic heart disease, so you may have a patient wit triscuspid regurgitation leading to an IVC that is always plethoric and may not be related to why that person is in shock.

Other PoCUS exams to consider

Lung

- Hemo/pneumothorax?
- Pleural effusion?

Abdominal part of FAST

- intra-abdominal free fluid?

DVT

PoCUS findings for shock

RUSH exam	Hypovolemic shock	Distributive shock	Obstructive shock	Cardiogenic shock
Pump	Hyperdynamic heart	Hyperdynamic heart (early sepsis) Poor contractility (late sepsis)	Pericardial tamponade RV strain Poor contractility	Poor contractility
Tank	Small, collapsing IVC Peritoneal or pleural fluid	Normal/small IVC Pleural or peritoneal fluid	Large, non-collapsing IVC Absent lung sliding	Large, non-collapsing IVC Lung rockets Pleural effusion
Pipes	AAA or dissection	Normal	DVT	Normal

ALiEM. https://www.aliem.com/rush-protocol-rapid-ultrasound-shock-hypotension/

Investigations – Be liberal

- CBC
- Electroytes, Ca/Mg/PO4
- Liver enzymes, LFTs
- Renal function tests
- Cultures
- Troponin
- Lactate
- Coagulation profile
- ABG/VBG
- EKG
- CXR

These are sick patients, so they are not the patients you need to skimp on for investigations. Be liberal here!
You are trying to figure out what exactly is causing their shock and how you are going to fix it. There can be multiple issues going on.
I want to draw special attention to lactate because it features so prominently in approaches to diagnosing and managing shock.

Lactate is a byproduct of anaerobic cellular metabolism due to lack of perfusion and oxygenation.
So it is a useful marker of shock if it's abnormal. And it can be trended to help you see how well you are resuscitating a patient.

However, lactate can also be abnormal for a host of reasons not necessarily related to a person being in shock. These include diagnoses related to poor perfusion or anaerobic cellular metabolism even if the patient is not in shock like ischemic bowel or carbon monoxide poisoning.

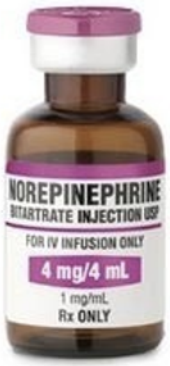
There was also a recent RCT (ANDROMEDA-SHOCK) comparing 28 day mortality between two resuscitation strategies in adult patients presenting in septic shock. The

first was trending lactate clearance, the second was assessing capillary refill time in adult patients presenting with septic shock. While the results did not reach the level of statistical significance, there was a trend toward decreased mortality in the capillary refill time group.

So the bottom line with lactate is: it can be useful if you have it. But you certainly don't need it to diagnose shock, nor do you need it to assess how effectively you are resuscitating your patient. Instead, look at other things – heart rate (is it coming down?), blood pressure (is it going up?), capillary refill time, peripheral pulses, mental status, urine output (are they improving?)

Shock Management "Toolkit"

AB +



Start treatment immediately if you suspect shock

Let's switch gears now and consider interventions and management of your patient in shock.
Before we delve deeper into each type of shock, there are some universal tools that you should consider for most patients in shock.

The first intervention is IV access. This seems like a silly thing to talk about. But there are 3 things that I think are important to stress here.
1. Get at least 2 large bore IVs in the antecubital fossa if possible. Large bore means 14G or 16G which in reality is difficult to find unless you are in a trauma centre. However, you should recognize that a 22G IV in your patient's hand is not going to be effective for resuscitation. Push for more than one point of IV access and for it to be at least 18G or 20G wherever possible.
2. If you cannot get IV access, use an IO. Whatever you can give through a central line, you can give through an IO.
3. Do not waste your time getting a central line into a patient you need to resuscitate. It can often take a long time and is not a good tool for resuscitation when you think about it. The catheter is long and skinny which does not lend itself to high flow, compared to an IO which is short and fat.

The 2nd intervention is fluids. Most patients in shock (except for those in cardiogenic shock) will benefit from IV fluids. You should consider an initial bolus of 20-30mL/kg. Two important points here:
1. Use crystalloids (like normal saline or Ringer's lactate) over colloids (like albumin, starches, etc.)
2. Use Ringer's lactate over normal saline when possible. While there's no evidence of mortality benefit between one over the other, there is a reduction in patients who will go on to require dialysis when using Ringer's lactate over normal saline. The reason for is the high chloride load in normal saline – chloride is nephrotoxic. And large volumes of normal saline leads to a hyperchloremic metabolic acidosis in a patient who already likely has a metabolic acidosis from their shock.

The 3rd intervention is vasopressors and inotropes, which we will talk about next
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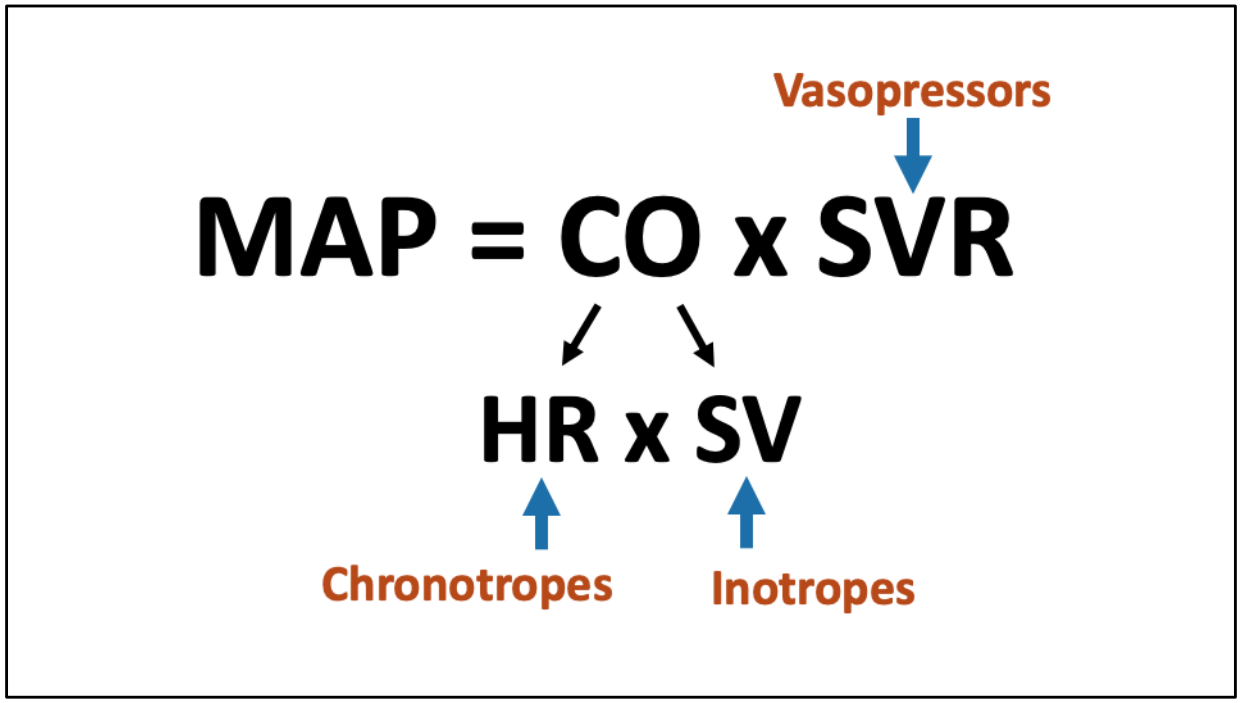
Fluid resuscitation
- Balanced crystalloid (Ringer's lactate vs NS) if available – significantly reduced composite of death from any cause, need for new dialysis, persistent renal dysfunction
    - Recommend use unless neurologic impairment (since Ringer's lactate is hypotonic) or significant metabolic alkalosis
    - Balanced Crystalloids Versus Saline in Noncritically Ill Adults (SALT-ED) – Self et al., NEJM 2018
    - Balanced Crystalloids versus Saline in Critically Ill Adults (SMART) Semler et al., NEJM 2018

Vasopressors - squeeze the PIPES

Inotropes – squeeze the PUMP

Chronotropes – make the PUMP work faster

We often refer to vasopressors and inotropes interchangeably, and while many of the drugs that we use act as both, they are not the same thing.
Vasopressors constrict → they squeeze the pipes.
Inotropes increase the contractility of the heart → they squeeze the pump.
Chronotropes increase the heart rate → they make the pump work faster.



So vasopressors work on increasing SVR
Chronotropes work on increasing HR to increase CO
Inotropes work on increasing SV to increase CO

Vasopressors and Inotropes

	Vasopressor	Inotropy and Chronotropy	Dosing range
Norepinephrine	++++	++	0.01-3mcg/kg/min
Epinephrine	++++	++++	0.01-0.1mcg/kg/min
Dopamine	++ 10-20mcg/kg/min	++ 3-10mcg/kg/min	--
Dobutamine	+/- (Dose-dependent)	++++	2-20mcg/kg/min
Isoproterenol	vasodilation	+++++	2-10mcg/kg/min

I have included this chart for your reference (Dr. Finot is giving a separate lecture on Vasopressors and Inotropes), but I do want to highlight some of the main points from this slide.

The 5 drugs here, norepinephrine, epinephrine, dopamine, dobutamine and isoproterenol, represent the 5 vasopressors and inotropes that you will see most often used in the ER.

First, inotropy and chronotropy effects are grouped together because the same receptors (the beta-1 receptors) activate both.

Second, there is usually a trade-off between inotropic and chronotropic effects and vasoconstriction effects. What I mean by that is, usually, the more vasoconstriction a drug causes, the less inotropy and chronotropy it causes and vice-versa.

I've ordered them so that the most vasoconstrictive drug out of the 5, that's the norepinephrine, is at the top, while the most inotropic and chronotropic, the isoproterenol is at the bottom.

Third, the effects of some of these drugs are dose-dependent. Take dopamine for instance.

The bottom line here is: the first 2 drugs – norepinephrine and epinephrine are usually used as vasopressors, while the bottom 2 – dobutamine and isoproterenol are used as inotropic and chronotropic agents. Dopamine's effects are dose-dependent (though it usually is used as a vasopressor in the ER setting).

Putting it all together…

And now for some cases to apply an approach to managing patients in shock, including some of what we've just learned

Case 1

23 yo M from Addis
3 d of fatigue and shortness of breath
Brought in by family after a syncopal episode
They tell you he has a “heart problem”

Case 1

HR 132, BP 76/36, SaO_2 88%, RR 30, Temp 36.3

- Appearance: obtunded
- Cardiovascular exam: N S1,S2, irregular, holosytolic murmur, JVP is 5 cm ASA, no edema
- Resp: bilateral crackles, accessory muscle use
- Abdomen: unremarkable
- PoCUS: poor cardiac contractility, IVC plethoric with no respiratory variation, multiple B-lines bilateral lung fields

Shock?

Pump, Tank, or Pipes?

Is this patient in shock and why?
What type of shock?
- Cardiogenic, intrinsic pump problem

What could the underlying cause could be?
-Rheumatic heart disease with mitral valve regurgitation with decompensation likely due to a secondary insult (such as infection or non-compliance with meds)
-cardiomyopathy secondary to chronic regurgitation with decompensation likely due to a secondary insult
-endocarditis
-also consider atrial fibrillation as the cause - however caution as the rapid rate can be a compensatory mechanism

Empiric Criteria for Shock

4 out of 6 criteria have to be met

1. **Ill appearance or altered mental status**
2. **Heart rate >100**
3. **Respiratory rate > 20** (or PaCO2 < 32 mmHg)
4. Urine output < 0.5 ml/kg/hr
5. **Arterial hypotension > 30 minutes duration**
6. Lactate > 4

Assuming hypotension has been > 30 min, meets criteria for shock

Patient meets 4 of the following 6 criteria:
Ill appearance or altered mental status
HR >100
RR > 22 or PaCO2 < 32 mmHg
UO < 0.5 ml/kg/hr
Arterial hypotension > 20 mins duration
Lactate > 4

Case 1: Management

Cardiogenic Shock

- ABC's
  - Monitors
  - O_2
  - IV and blood work
  - ECG
    - Atrial Fibrillation, rate 130's

What are your initial actions going to be?

Case 1: Management

HR 132, BP 76/36, SaO_2 88% on max O_2, RR 30

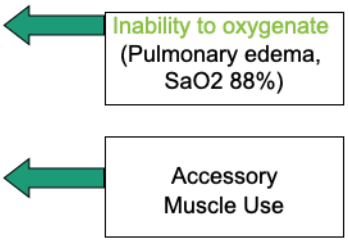
Would you intubate this patient?

Case 1: Why would you intubate?

Is the cause quickly reversible? **UNLIKELY**

3 reasons to intubate in the setting of shock

- ☐Inability to oxygenate
- ☐Inability to maintain airway
- ☐Work of breathing



Case 1: Management

- Cardiogenic Shock
  - Treat Underlying Cause
    - Lasix for fluid overload
    - Atrial Fibrillation: Cardioversion vs Rate control?
    - Pressors/Inotropes – ideally Dobutamine +/- Norepinephrine if available
    - Look for precipitating causes

Notes:
-start with diuresing the patient with lasix (decreasing the pulmonary edema, will decrease the work of breathing, and likely lead to improved cardiac output)
- in a recent trial low dose lasix may be just as good as high dose lasix (see below for the trial by Felkner et al) in pts with decompensated heart failure
-It is often challenging to distinguish what is the primary cause in these patients --> AF causing heart failure or heart failure causing atrial fibrillation
-afib may be a chronic condition in these patients, and the high heart rate may be compensatory
-one approach is to diurese the patient first - if the heart rate slows down, it suggests that the rapid afib was secondary to the heart failure. If the diuresis does not work, then attempt electrical cardioversion or rate control
-if the left atrium is dilated, cardioversion will likely not work (at least in the early stages)
-for rate control choose a short acting agents such as esmolol (rarely available) or diltiazem (preferable to verapamil because it is less of a negative inotrope), or long acting agent such as digoxin (which will have a slow onset 6-12hrs but also has inotropic activity)

-amiodarone is an option if available
-don't forget to anticoagulate this patient with warfarin (and heparin if cardioverting!) - they have a very high risk of stroke
-in the next slide we discuss the use of inotropes

Felker et al NEJM 2011;364;797
-pts presenting with acute decompensated heart failureweithin 24 hrs (with previous known HF, and previously on diuretics)
-pts were randomized to receive furosemide at low or high dose, and by IV or continuous infusion (High dose = doubling the amount of lasix pt was on before presentation)
-primary outcome was global assessment of symptoms at 72 hrs
-primary safety endpoint was 72 hr change in serum creatinine
-308 participants
-primary end point did not differ between any group, although the primary outcome was slightly greater in high dose versus low dose groups (P= 0.06)
-however, more high dose than low dose pts had an increase in CR level of > 0.3 mg/dL (23% vs 14% p=0.04)

Vasopressors in Cardiogenic Shock

- **Norepinephrine:** *drug of choice when available*
- Dopamine
- Epinephrine
- Phenylephrine

Notes: This slide discusses the choices of vasorpessors and inotropes available
-Dopamine has fallen out of favour as the vasopressor of choice in cardiogenic shock, NE had become pressor of choice in most shock situations
-Dobutamine 2-10 micrograms/kg/minute +/- Norephinephrine 0.01-3 micrograms/kg/minute (usual range 8-30 micrograms/minute)
-Epi and Dopamine likely not a good idea - can cause increased HR

De Backer et al *N Engl J Med* 2010;362;779
-1679 pts with shock (hypovolemic, cardiogenic, septic shock) were randomized to either dopamine or norepinephrine if still hypotensive after fluids
-primary end point was death at 28 days
-Dopamine and NE no difference in mortality when used in all-comers with shock
-however dopamine increased mortality in cardiogenic shock!
-also, significantly more patients on dopamine developed arrythmias (24 vs 12%)
***HOWEVER, consider whether these results apply to your patient (no patients from low-middle income countries were enrolled in this trial,

and the effect of dopamine was based on a subgroup analysis) so if dopamine is what is accessible, it is perfectly acceptable to use***

Levy B et al. *Crit Care Med* 2011 Mar; 39:450.
-small open randomized trial study (approximately 30 pts in each arm) in pts with cardiogenic shock
-norepinephrine/dobutamine versus epinephrine
-10/15 in epi group and 11/15 in NE/D group survived
-epi was associated with significantly mean higher HR and mean lactate level, and new arrythmias were observed in 2 pts in epi group
-small study - therefore hard to draw any conclusions from it

Monitoring

- Vitals - BP, HR, SaO2
- Mental Status
- Urine Output (> 1-2 ml/kg/hr)

- When something changes or if you do not observe a response to your treatment, re-examine the patient

Notes: These are indirect measures

Case 2

- 40 year old female
- Urinary symptoms for a week
- Fever, flank pain, fatigued for 3 days
- No past medical history

Case 2

HR 110, BP 100/72, SaO2 96%, T 39.2, RR 28

Drowsy

Warm skin

Heart - S1, S2, no murmurs

Chest – good air entry bilat

Abdo- soft, non tender, +bilateral CVA tenderness

PoCUS:

Cardiac – hyperdynamic, no pericardial effusion

IVC – 1cm, collapsing 50% with respiration

Shock?

Type of Shock? (Tank, Pump or Pipes?)

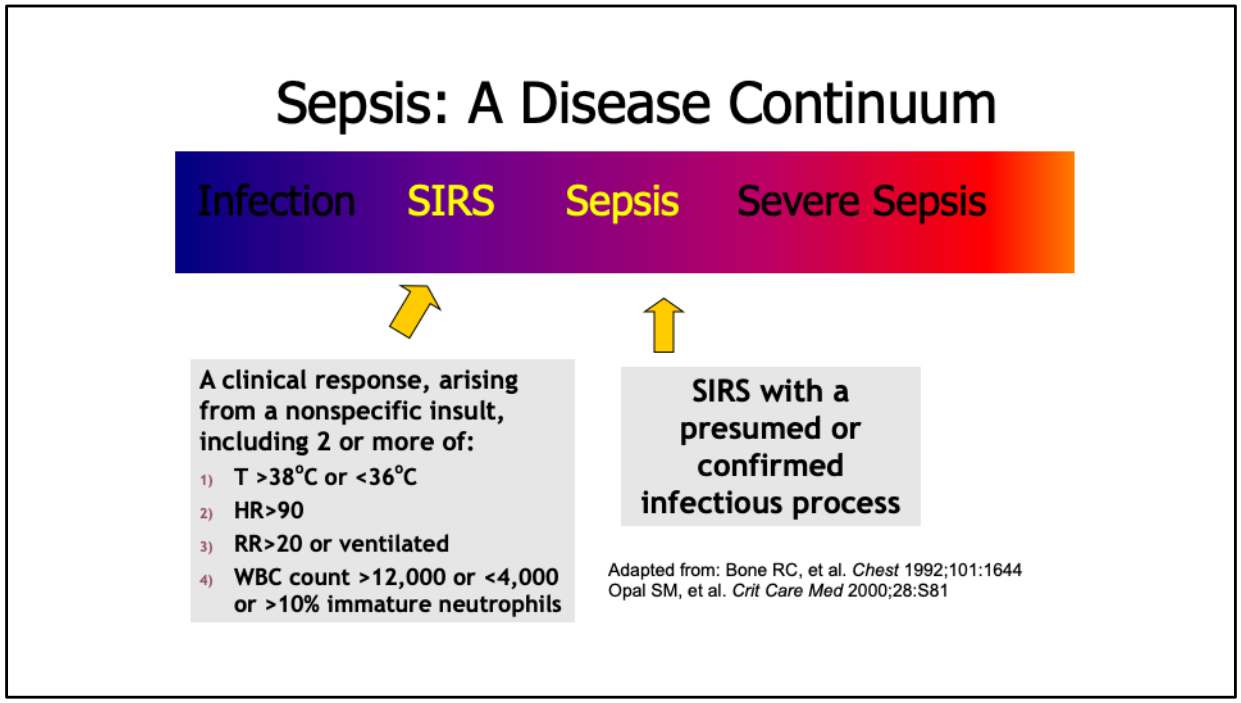
Instructions: Ask the learner Is he in shock? Why?
Answer: Pt is in early shock - Pt meets 3 of 6 criteria (see next slide)
Instructions: What kind of shock is this?
Answer: SEPTIC – primarily a pipe problem, but also a tank problem (so to treat, you need to fill the tank AND squeeze the pipes)

Empiric Criteria for Shock

4 out of 6 criteria have to be met

1. **Ill appearance or altered mental status**
2. **Heart rate >100**
3. **Respiratory rate > 22** (or $PaCO_2$ < 32 mmHg)
4. Urine output < 0.5 ml/kg/hr
5. Arterial hypotension > 30 minutes duration, continuous
6. Lactate > 4

Defining sepsis



Previous definition of Sepsis (Sepsis -2)
No longer used.

Sepsis: New Definition

Life-threatening organ dysfunction caused by a dysregulated host response to infection

Suspected or documented infection

AND

Acute increase of >/= 2 SOFA points

(SOFA= Sequential Organ Failure Assessment)

Practically speaking, sepsis is ”suspected or documented infection AND an acute increase of 2 or more SOFA points)

SOFA Score

- A measure of organ dysfunction
- Assumes a baseline of zero in pts without pre-existing organ dysfunction
- SOFA score >/= 2 reflects overall mortality risk of ~10%

Tool for identifying pts at high risk for in hospital death or prolonged ICU stay
Does not define sepsis, used as a risk assessment tool

qSOFA (Quick SOFA) Criteria

- RR >/= 22/min
- Alteration in mental status (GCS<15)
- Systolic BP </= 100 mm Hg

qSOFA = quick SOFA score is used outside of the ICU
Provides simple bedside criteria to identify pts with suspected infection who are likely to have poor outcomes
Positive qSOFA criteria should also prompt consideration of possible infection infection in pts not previously recognized as infected, that is new or worsening organ dysfunction should raise the possibility of an underlying infection.
qSOFA is also a predictor of mortality, not a “test” for sepsis.

Septic Shock: New Definition

Subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality

In lay terms: sepsis is a life threatening condition that arises when the body's response to an infection injures its own tissues and organs
The term "severe sepsis" is no longer used.

Septic Shock

Persisting hypotension requiring vasopressors to maintain MAP > 65 mm Hg

AND

Serum lactate > 2 mmol/L despite adequate volume resuscitation

- *with these criteria => mortality >40%*

New Definition Controversies

- Subjective
  - "Life threatening", "dysregulated"
- SOFA and qSOFA are mortality predictive indicators, not a test for sepsis
- SOFA calculation requires a lot of data (ICU)
- qSOFA is not specific for sepsis
- Scores derived on ICU pts and may not be applicable to EM

Many critically ill but not infected pts may have an elevated SOFA score. With fewer elements than previous definition (SIRS criteria), SOFA may be less specific in identifying infection
The score has only been used as a predictive score and is not prospectively validated
New sepsis definitions have not been endorsed by and Emergency Medicine groups. However it is important that we are aware of the new definitions

The working/practical definition is not very useful for EM since it is, for ER docs, a retrospective definition – and the trigger to start treatment early is not captured by this definition (you should not wait for risk-stratification based on SOFA score to start treatment)

So what are we left with to start suspecting sepsis?
1. qSOFA should be a trigger to think of sepsis.
2. Look for signs of organ failure as suggested by the empirical criteria and clinical presentation that we talked about previously.

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Back to Case 2

HR 110, BP 100/72, SaO2 96%, T 39.2, RR 28
Drowsy
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PoCUS:
Cardiac – hyperdynamic, no pericardial effusion
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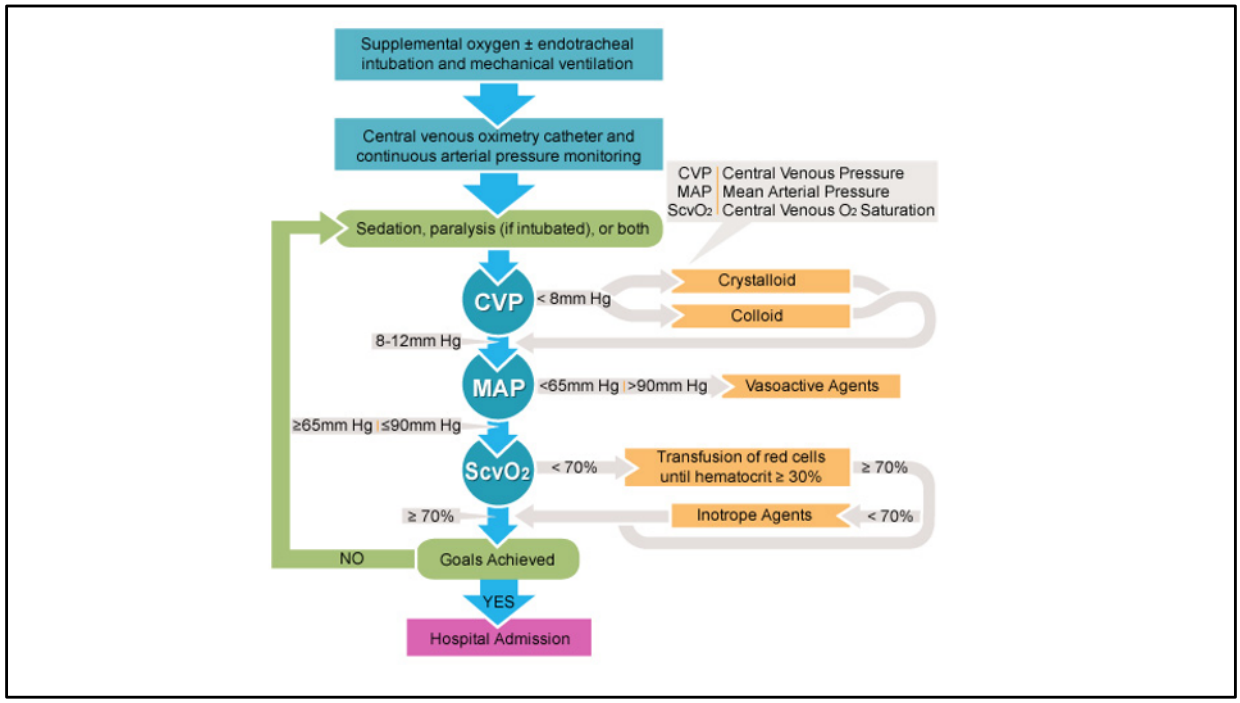


Early Goal Directed Therapy – used to be a concept in how we treated sepsis, with target measures and invasive monitoring. This is no longer used, but is mentioned here because you might still come across this in older resources.

Early Goal Directed Therapy

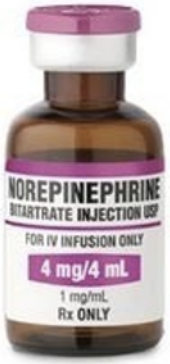
- Predefined resuscitation end points to help guide resuscitation of patients in septic shock within first 6 hours
- No mortality benefit of original EGDT guidelines over institutional guidelines
- Practically available end points
  - MAP, U/O, IVC assessment on U/S as a surrogate for CVP?

Previous parameters required invasive monitoring to obtain central venous pressures and central venous oxygenation and are not practical in most settings
As well, 3 major randomized trials following institution of guidelines (ProCESS, ARISE, ProMISE) showed no mortality benefit using the original EGDT guidelines
Practical end points will be discussed in more detail shortly
Guidelines now focus on less invasive clinical parameters with the goals of care focusing on early IV fluids and antibiotic therapy

Distributive Shock - Septic

Management Priorities

AB +



Steroids?

So let's talk about Management Priorities.
In the ER, we always start with ABC's.
Let's talk about this in order

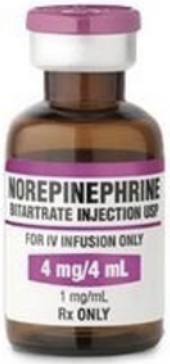
Case 2: Airway - Would you intubate?

- ☐ Is the cause quickly reversible? **LIKELY**
- ☐ 3 reasons to intubate in the setting of shock
  - ☐ Inability to oxygenate
  - ☐ Inability to maintain airway **POSSIBLY**
  - ☐ Work of breathing **POSSIBLY**

Distributive Shock - Septic

Management Priorities

AB +



Steroids?

Breathing – supplemental O2

Circulation – 20mL boluses of Ringer's lactate (large volume Normal Saline resus → hyperchloremic metabolic acidosis), end point – clinical improvement: perfusion, mentation, U/O (aim 1-2mL/kg/h)


Antibiotics

Antibiotics in Sepsis

- **Early Antibiotics**

  Within 3-6hrs can reduce mortality - 30%

  Within 1 hr for those severely sick

  Don't wait for the cultures – treat empirically then change if needed

Notes:
Choice of antimicrobials will depend on age, cause, comorbidities, Gram stain data, local resistance patterns
Remember enteric coverage - GI or GU coverage and Listeria monocytogenes and HSV in infants < 28 days

For adults
-vanco + 3rd cephalo or pip-tazo or carbapenem
-vanco + ceftazidime or imipenem/meropenem or pip tazo or cipro or aminoglycoside if suspect pseudomonas

For children > 28 days
-vancomycin 15 mg/kg max 1-2 g (to cover for MRSA)
-cefotaxime 100mg/kg max 2g or ceftriaxone
-aminoglycoside for GU source
-clindamycin or metronidazole for GI source
-if immunosuppressed and at risk for pseudomonas - switch cephalosporins for Cefepime or ceftazidime

For children < 28 days
-add ampicillin 50 mg/kg and gentamicin 2.5 mg/ kg to vancomycin and cefotaxime
-add acyclovir 20 mg/kg if suspect HSV

Empiric Abx for severe infections

- Cellulitis – cefazolin
- Diabetic foot infections - ceftriaxone + metronidazole
- Pneumonia – ceftriaxone + azithromycin
  - If Pseudomonas colonized – piperacillin-tazobactam/other anti-Pseudomonal
- Urinary source – ceftriaxone
- Intra-abdominal source – (ceftriaxone + metronidazole) or pip-tazo
- Biliary source – ceftriaxone + ampicillin
- Meningitis – ceftriaxone 2g IV + vancomycin
  - If <1m/o or >50y/o – add ampicillin, consider acyclovir to cover HSV

Choice of antimicrobials will depend on age, cause, comorbidities, Granm stain data, local resistance patterns, but these are the empiric antibiotics that I would consider based on suspected source.

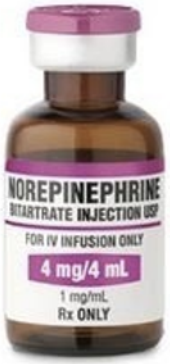
Source Control

- Surgical infections require **IMMEDIATE** surgical drainage/removal
- 4 D's
  - Drain it
  - Debride it
  - Device removal
  - Definitive control

Distributive Shock - Septic

Management Priorities

AB +



Steroids?

Vasopressors and inotropes?

Vasopressors

- Persistent hypotension
  - Despite fluid resuscitation
  - Unable to tolerate fluid resuscitation
- Where available, norepinephrine is Rx of choice
- Dopamine (caution re: chronotropic effects)
- Phenylephrine (if profound tachycardia)

Phenylephrine has pure alpha adrenergic activity and therefore may be a reasonable choice in pts with sever tachycardia precluding the use of agents with beta adrenergic activity

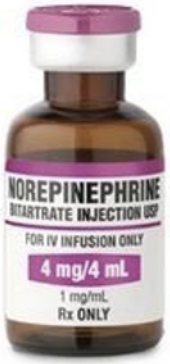
Inotropic Support

- In patients with refractory shock with diminished cardiac output can consider addition of dobutamine

Distributive Shock - Septic

Management Priorities

AB +



Steroids?

Steroids?

Steroids in sepsis?

Maybe?

Consider use in fluid- and vasopressor-refractory shock

Cochrane systematic review published in 2019 (Source: https://doi.org/10.1002/14651858.CD002243.pub4) that included 58 trials comparing steroids to no steroids in adult and children with sepsis found there is probably reduced 28-day mortality and in-hospital mortality, but less clear about mortality after 90 days.
So, you could consider using it in fluid- and vasopressor-refractory shock, but it's also okay not to give it.

-ADRENAL (3658 patients who had septic shock) found no statistically significant difference in 90 day mortality between the hydrocortisone and placebo groups.[1]
-APROCCHSS (1241 patients who had septic shock) found that hydrocortisone plus fludrocortisone reduced 90 day mortality.[2]

References In Adults:
Sprung CL et al - Hydrocortisone therapy for patient with septic shock **(CORTICUS)** - NEJM 2008 Jan 10 358:111
-No significant difference in 28 day mortality (34 vs 32%)
-BP improved more quickly with hydrocortisone but there were more episodes

of superimposed infections
-No difference in cosyntropin responders and non-responders - disputing earlier results in Annan's study JAMA 2002

References In children
-1 RCT in dengue shock showed improved outcomes with hydrocortisone
-1 retrospective cohort study showed mortality increased in septic shock
-consensus - in those with catecholamie resistant shock who have or are suspected to have adrenal insufficiency should receive glucocorticoids
-a total random serum cortisol level < 18ug/dL (496 nmol/L) defines absolute adrenal insufficiency and indicates the need for continued glucocorticoid therapy (preliminary evidence suggests that calculation of free cortisol and free cortisol index is more accurate - derived from total cortisol and cortisol binding globulin

Concluding Remarks

- Shock = inadequate perfusion to tissues, *not low BP*
- Start treatment early – IV access, fluids, vasopressors, antibiotics
- Consider: Tank, Pipes or Pump?
  - Tank = fluids or blood
  - Pipes = vasopressors
  - (Intrinsic) Pump = vasopressors, inotropes, chronotropes
  - (Extrinsic) Pump = remove obstruction

Concluding Remarks

Choose cost effective and high impact interventions

Do not need central lines and ScvO2 measurements to make a big impact!!