Shock is a state of systemic hypoperfusion, with inadequate blood supply to the tissues. Unfortunately, this may occur in different ways. The most simple physiology of shock is cardiogenic shock, with low cardiac output to the entire body. However, septic shock can occur with an elevated cardiac output due to microvascular dysfunction at the tissue level (blood shunts through some vessels, while ignoring others).

Because shock has varying physiologies, it defies any simple operational definition at the clinical level. Shock is a bit like obscenity – after a while, you know it when you see it.
Shock is the final common pathway prior to death.

Shock is extraordinarily important because it is generally a final common pathway before death. Most serious diseases are capable of causing shock. Left untreated, shock will progress to multi-organ failure and death. However, shock is often reversible, thereby avoiding death.

**Diagnosis**

The importance of promptly diagnosing shock and discerning its cause cannot be overstated. Unfortunately, shock may present in a variety of ways, so diagnosis isn't always so simple. For example, shock is typically associated with hypotension and reduced cardiac output, but it can also occur with normal blood pressure and an increased cardiac output.

Different types of shock present differently. For example, the stereotypical patient with early sepsis and distributive shock will appear quite different from the patient with cardiogenic shock:

**Classic appearance of septic vs. cardiogenic shock**

<table>
<thead>
<tr>
<th></th>
<th>Sepsis</th>
<th>Heart failure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical appearance</strong></td>
<td>- Generally look sick.</td>
<td>- Often look deceptively normal.</td>
</tr>
<tr>
<td></td>
<td>- Often tachypneic, delirious, febrile</td>
<td>- May continue mentating, despite</td>
</tr>
<tr>
<td></td>
<td>(*&quot;toxic appearing&quot;)</td>
<td>profoundly low cardiac output.</td>
</tr>
<tr>
<td><strong>Skin examination</strong></td>
<td>- Initially flushed, warm extremities.</td>
<td>- Cool extremities.</td>
</tr>
<tr>
<td></td>
<td>(Later on may develop poor perfusion)</td>
<td>- Poor capillary refill, may have mottling</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>- Wide pulse pressure</td>
<td>- Narrow pulse pressure</td>
</tr>
<tr>
<td></td>
<td>- Very low diastolic pressure</td>
<td>- Both systolic and diastolic Bp are moderately reduced.</td>
</tr>
</tbody>
</table>

Urine output

|                        | Low | Low |


**Shock red flags**

There is no single diagnostic test for shock, but rather this diagnosis is made at the bedside on the basis of clinical judgement. Patients present differently, but this diagnosis will generally be suggested by the presence of more than one of the following features:

- **Hemodynamics** *(trends will usually be more helpful than a single abnormal value)*
  - **Hypotension** *(e.g. MAP<65 and/or significant drop from baseline)*.
  - **Tachycardia & elevated shock index**: the shock index (HR/SBP) is a useful way to understand tachycardia within the context of blood pressure. Shock index over ~0.8 suggests significant instability and possible shock.
• **Bradycardia**: Cardiac output is directly proportional to heart rate. Severe bradycardia (e.g., heart rate below ~45) should always raise concern for shock. Even if the blood pressure is maintained by compensatory systemic vasoconstriction, cardiac output and perfusion may still be poor (more on bradycardia [here](https://emcrit.org/ibcc/bradycardia/#why_bradycardia_is_dangerous)).

• **Low urine output (or dark urine)**: Urine output below 0.5 cc/kg/hr is worrisome for renal malperfusion. Immediately following Foley catheter placement the urine output won’t be known — in this situation scanty and dark urine is worrisome (more on the approach to oliguria [here](https://emcrit.org/ibcc/acute-kidney-injury/#approach_to_oliguria)).

• **Skin perfusion**:
  - **Cool hands and knees** are an early sign of vasoconstriction with reduced cardiac output. Normal people may have cool hands, but if all extremities are cool that’s more specific for hypoperfusion.
  - **Mottling** is less sensitive, but more specific for hypoperfusion and elevated mortality (figure below). Mottling suggests active endogenous vasoconstriction, implying that the patient would benefit from an increase in cardiac output (e.g., an inotroph) — not additional exogenous vasoconstrictors.
  - **Urticaria, angioedema, flushing, and pruritus** are suggestive of anaphylaxis; when in doubt start empiric therapy for anaphylaxis immediately.

• **Delirium**: New-onset delirium can be a sign of shock. However, this is neither very sensitive nor specific. Most new-onset delirium isn’t due to shock. Furthermore, patients with cardiogenic shock often maintain normal mentation (delirium tends to be a feature of septic shock rather than of cardiogenic shock).

![Mottling Score](https://i1.wp.com/emcrit.org/wp-content/uploads/2017/06/mottling.png)

*Mottling isn’t particularly sensitive, but when present it is highly concerning. Image from Galbois A 2015 [here](http://www.sciencedirect.com/science/article/pii/S0168827814007417).*

**Labs can only suggest shock (not exclude it)**

- Lactate elevation (e.g., >4 mM) suggests shock, but this has a broad differential diagnosis. In practice lactate usually doesn’t reflect oxygen deficiency, but rather endogenous epinephrine ([here](https://emcrit.org/pulmcrit/understanding-lactate-in-sepsis-using-it-to-our-advantage/)) in response to physiologic stress. This explains why lactate can be normal in shocked patients who have inadequate sympathetic nervous function.
  - High lactate level is worrisome. This should be interpreted to represent shock or some other impending disaster until proven otherwise.
  - Normal lactate isn’t necessarily reassuring (can occur in shock).
  - Central venous oxygen saturation is sometimes used as a diagnostic test of systemic perfusion. This has poor performance and shouldn’t be used ([here](https://emcrit.org/pulmcrit/central-venous-saturation/)).

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**Causes of shock**

Numerous life-threatening processes can cause shock. These may be roughly categorized as shown below.

- **Arrhythmic shock**
  - Tachyarrhythmia (usually >> 150 b/m)
Bradyarrhythmia (usually < 45 b/m)

Hypovolemic shock
- Hemorrhage (external, GI bleed, retroperitoneal bleed, intra-peritoneal bleed, hemothorax, post-partum).
- Hypovolemic (e.g., vomiting, diarrhea, over-diuresis, post-ATN or post-obstructive polyuria).

LV failure (*cardiogenic* shock)
- LV systolic failure (e.g. MI, myocarditis, beta-blocker overdose)
- Acute aortic or mitral valve regurgitation (e.g. endocarditis, papillary muscle rupture, aortic dissection)
- Prosthetic valve thrombosis
- Dynamic LV outflow tract obstruction (LVOTO)

RV failure
- Pulmonary embolism
- Decompensated chronic pulmonary hypertension
- Right ventricular myocardial infarction

Obstructive
- Tension pneumothorax
- Tamponade
- Abdominal compartment syndrome
- AutoPEEP or high mean airway pressures

Vasodilatory shock (*distributive* shock)
- Sepsis
- Severe systemic inflammation (e.g. pancreatitis, post-cardiac arrest, post-MI)
- Anaphylaxis
- Adrenal crisis, thyroid storm
- Neurogenic shock (severe CNS/spinal trauma, spinal anesthesia)
- Liver failure
- Excess vasodilatory drugs

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evaluating the cause of shock

There is no single test to reveal the etiology of shock, but rather this depends on integration of information from numerous sources. For example, if a previously healthy woman develops shock in the postpartum state with an ejection fraction of 25%, post-partum cardiomyopathy is likely. Alternatively, if an elderly man with a baseline ejection fraction of 25% develops shock (with an unchanged ejection fraction of 25%), his acute deterioration probably isn't due solely to systolic heart failure.

**history & data review, with focus on:**
- ? Cardiac history (especially any prior information about cardiac structure/function such as EKG, echo, or even chest CT showing chamber size).
- ? Adrenal disease (noting: patients chronically on oral steroid may be assumed to be insufficient).
- ? History of venous thromboembolic disease.
- ? Immunosuppression, ? Invasive devices (e.g. hemodialysis catheters).
- ? Recent procedures or trauma.
- ? Current medications & changes in medication list
bedside shock evaluation

**monitor**
- Narrow pulse pressure (<25% systolic) suggests low cardiac output
- Wide pulse pressure with diastolic hypotension suggests high-output shock

**echocardiogram**
- RV size & function (if suspect PE, check for DVT)
- LV size & function
- Mitral & aortic valve function (exclude severe regurgitation)
- IVC (if unable to see IVC, evaluate internal jugular vein)
- Pericardial effusion?

**lung ultrasound**
- Absent lung slide suggests tension pneumothorax
- Bilateral & diffuse anterior B-lines suggest cardiogenic edema with elevated filling pressures
- Patchy B-lines and/or consolidation suggests PNA

**abdominal ultrasound**
- FAST exam to evaluate for peritoneal hemorrhage
- LUQ & RUQ views adequate to look for large volume hemorrhage
- Aorta evaluation for dissection flap

**skin perfusion**
- Warm extremities despite shock suggest vasodilatory shock
- Mottling or cool extremities suggests inadequate cardiac output

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**radiologic studies**
- **EKG** occasionally helpful (e.g. may reveal occlusive MI or RV strain)
- **CXR** (e.g. may reveal pneumonia or cardiogenic edema implying LV failure)
- **CT** depending on clinical scenario (e.g. CTA to evaluate for PE, CT A/P to exclude septic focus in abdomen)

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**labs**
- Extended electrolytes (including Ca/Mg/Phos), CBC, Coagulation studies, LFTs
- Lactate
- Sepsis evaluation
  - Infectious workup if sepsis suspected (e.g. blood cultures, urinalysis and reflex culture, sputum culture if clinically indicated)
  - If a decision has been made to treat empirically with antibiotics, then check a procalcitonin as well.
- Type & cross-match blood if hemorrhage suspected
- Endocrine evaluation
  - Cortisol level if adrenal insufficiency is possible
  - TSH if thyroid storm suspected

---

25 YO female WITH undifferentiated shock.
EM ECHO tells the diagnosis @AbusinSalah @sharonmkay
@EMcardiac @EM_RESUS #FOAMed#SUDAN_EM

---

https://emcrit.org/ibcc/shock/
Findings on ultrasonography and physical examination may be integrated as shown below. This tends to work best in previously-healthy patients with a single mechanism of shock. Patients with multiple chronic problems or multi-factorial shock may defy categorization.

### Bedside classification of shock

<table>
<thead>
<tr>
<th>Classification</th>
<th>IVC or Jugular size</th>
<th>Lung POCUS</th>
<th>Pericardial effusion</th>
<th>RV dilation</th>
<th>LVEF</th>
<th>Mitral or aortic regurg?</th>
<th>Additional findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic shock</td>
<td>↑↑</td>
<td>A-lines</td>
<td>No</td>
<td>No</td>
<td>nl ↑</td>
<td>No</td>
<td>Hemoperitoneum or hemothorax in some cases</td>
</tr>
<tr>
<td>Distributive shock</td>
<td>↑↑ or normal</td>
<td>A-lines</td>
<td>No</td>
<td>No</td>
<td>nl ↑</td>
<td>No</td>
<td>Warm extremities (initially) - Low diastolic BP &amp; high pulse pressure - May be febrile or toxic-appearing</td>
</tr>
<tr>
<td>RV failure</td>
<td>↑↑</td>
<td>A-lines</td>
<td>No</td>
<td>Yes</td>
<td>nl ↑</td>
<td>No</td>
<td>DVT in legs</td>
</tr>
<tr>
<td>Tamponade</td>
<td>↑</td>
<td>A-lines</td>
<td>Yes</td>
<td>No</td>
<td>nl ↑</td>
<td>No</td>
<td>Pulmonary fibrosis</td>
</tr>
<tr>
<td>Tension pneumothorax</td>
<td>↑</td>
<td>No slide on affected lung</td>
<td>No</td>
<td>No</td>
<td>nl ↑</td>
<td>No</td>
<td>- May be impossible to see heart if L-sided PTX - sq emphysema</td>
</tr>
<tr>
<td>LV Failure</td>
<td>nl↑</td>
<td>B-lines everywhere</td>
<td>No</td>
<td>Possibly</td>
<td>nl ↑</td>
<td>Maybe</td>
<td>- Findings vary depending on etiology (e.g. diffuse vs. focal wall motion abnl)</td>
</tr>
<tr>
<td>Acute valve dysfunction</td>
<td>nl↑</td>
<td>B-lines everywhere</td>
<td>No</td>
<td>No</td>
<td>nl ↑</td>
<td>Yes</td>
<td>Endocarditis: embolic phenomena, fever - LVOTO: systolic anterior movement of mitral valve - Prosthetic valve: need formal study +/- TEE</td>
</tr>
</tbody>
</table>


### Stabilization

Stabilization must start immediately, often before the cause of shock is known. The following are common interventions to consider.

#### Volume resuscitation

- Often advisable, with the following exceptions:
  - Patients with bilateral B-lines may have left ventricular function and pulmonary edema.
  - Patients with elevated filling pressures (e.g. dilated IVC without respirophasic variation) are unlikely to benefit.
- Fluid should be provided in boluses (e.g. 500 ml) with attention to patient response. The total amount of fluid administered should generally be limited to <1-2 liters in the absence of a history suggesting substantial total-body volume depletion (e.g. severe gastroenteritis with a colostomy).
- Fluid administration can be diagnostic and therapeutic in confusing situations where hypovolemia is suspected:
  - If fluid resuscitation alone resolves shock, this supports a diagnosis of hypovolemia.
  - If fluid resuscitation fails, this suggests an alternative diagnosis. This is especially true if fluid resuscitation results in adequate filling pressures (e.g. full IVC) *without* resolving the shock.

#### Vasopressor administration

- Vasopressor administration should be started immediately if the blood pressure is inadequate (e.g. MAP<65 mm).
- Pressors may be administered via peripheral vein.
  - Norepinephrine may be given peripherally with careful monitoring of the IV site for limited periods of time.
  - Phenylephrine or epinephrine have a lower risk of extravasation and may be safer to use in situations with *less rigorous monitoring* (https://emcrit.org/pulcrit/phenylephrine-epinephrine-central-access/).

#### Antibiotics

- If sepsis is possible, cultures should be performed and empiric antibiotics should be started without delay.
- In patients with possible sepsis, you don't necessarily need to go extremely broad with the antibiotics. A single broad-spectrum agent may be reasonable (e.g. piperacillin-tazobactam).

**steroids**

- Indicated for patients whom you suspect have adrenal crisis, for example:
  - Patients with known adrenal insufficiency
  - Patients taking chronic steroids who recently missed doses

- When in doubt about adrenal insufficiency, a reasonable approach is to give 6 mg dexamethasone and check a cortisol level simultaneously.

- Dexamethasone doesn't interfere with the cortisol level, allowing you to perform an ACTH-stim test later on if indicated.

- More on the approach to adrenal crisis [here](https://emcrit.org/ibcc/adrenal-crisis/).

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**checklist**

[Checklist](https://emcrit.org/wp-content/uploads/2019/07/shockalg04.swf)

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**Approach to undifferentiated shock/hypotension**

**Data review**

- Vital sign & lab trends
- Recent procedures or medication changes?
- Relevant history (e.g. cardiomypathy, adrenal dysfunction)

**Bedside exam including:**

- General appearance & skin perfusion
- Echocardiogram
- Lung US to exclude pneumothorax
- Abdominal exam (tender? tense? Consider FAST)

**Cause identified**

**Cause remains unclear**

**Labs, potentially including:**

- Basic labs (chemistries, CBC, coags, LFTs)
- Lactate
- If infection suspected: cultures, urinalysis
- If infected suspected & ABX started: procalcitonin
- If hemorrhage possible: type & cross-match blood
- Cortisol level if adrenal insufficiency possible
- TSH if thyroid storm suspected

**Radiologic studies**

- EKG occasionally helpful
- Chest X-ray (Evidence of heart failure or PNA)
- Formal echocardiogram if bedside exam equivocal
- POCUS scan for DVT if PE remains a concern
- CT scan depending on scenario, for example:
  - CT angiogram if PE suspected
  - CT abdomin/pelvis to look for septic focus
  - CT angigram abdomen to look for hemorrhage

**Stabilizing maneuvers to consider**

(even before diagnosis is clear):

- Vasopressors (e.g. peripheral norepinephrine)
- Epinephrine drip (possible anaphylaxis)
- Fluid (depending on scenario & echocardiogram)
- Empiric antibiotics (if infection suspected; ideally after obtaining cultures)
- Empiric steroid (6 mg dexamethasone if adrenal crisis is suspected)
- Improved vascular access (central line and/or arterial line)

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**podcast**

[Listen](https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/apps.40518.14127333176902609.7be7b901-15fe-4c27-863c-7c0dbfc26c5c.5c278f58-912b-4af9-88f8-a65ff2da477.mp3)


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The Podcast Episode

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**questions & discussion**

[Back to contents](https://emcrit.org/ibcc/shock/)
Patients who have suffered cardiac arrest or MI will often develop a post-arrest or post-MI distributive shock due to cytokine release and systemic inflammation. This may lead to a confusing multifactorial picture, where distributive shock may obscure the primary problem.

- There is no sign, symptom, or lab which is entirely sensitive for shock. Therefore, no single investigation can exclude shock (e.g. “the patient is mentating well so she cannot be in shock” or “the lactate is normal so that excludes shock” are both incorrect statements).
- Patients in distributive shock may have a normal blood pressure, particularly if they have chronic hypertension.
- Diagnostic algorithms for shock (like any diagnostic algorithms) work best among patients with a single disease process who were previously normal. Unfortunately, many patients have multifactorial shock on an abnormal baseline (e.g. chronically reduced ejection fraction) – so simple algorithms will fail these patients.
- The most common cause of shock of unclear etiology is septic shock. However, other causes should be carefully excluded prior to reaching an empiric diagnosis of septic shock (e.g. echocardiography to evaluate for massive pulmonary embolism or pericardial tamponade).
- Don’t forget to evaluate archival data (e.g. old EKGs and CT scans). These may help sort out chronic pathology versus acute pathology.

**Going further:**

- [Cognitive approach to shock using POCUS](https://emcrit.org/pulmcrit/cognitive-approach-to-shock-diagnosis-using-ultrasonography/) (PulmCrit)
- [Shock – Do we know it when we see it?](https://lifeinthefastlane.com/challenging-the-assessment-of-shock/) (Michelle Johnston, LITFL)
- [Shock](https://lifeinthefastlane.com/ccc/shock-ddx/) (LITFL, Chris Nickson)
- [Undifferentiated hypotension](https://first10em.com/undifferentiated-hypotension/) (Justin Morgenstern, First 10 in EM)
- [The hypotensive ED patient: A sequential systematic approach](http://www.emdocs.net/hypotensive-ed-patient-sequential-systematic-approach/) (Manpreet Singh, emDocs)