SHOCK

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Objectives:

1. Understand the importance of the subject.
2. Define shock from a pathophysiologic standpoint
3. Differentiate ,recognize and give examples of the four main types of shock: hypovolemic, cardiogenic, obstructive, and distributive.
4. Diagnose and treat different types of shock
5. Keep general goals of shock resuscitation in mind
6. Assess the degree of hypovolemic shock
7. Practice some examples

Why to learn about SHOCK?

*Shock is a life threatening emergency that may be reversible if appropriately recognized and treated.

**early recognition and appropriate management of shock are critical to avoid adverse outcomes, due to the high mortality rate of the disease process.

***basic knowledge about shock is universally important for all doctors, not just anesthesiologist!

****In fact, one of the core professional activities endorsed by the Association of American Medical Colleges that medical students should be able to perform by the time of graduation is the ability to recognize a patient requiring urgent or emergent care and to initiate evaluation and management .

IN GERMANY ALL HOSPITALS HAVE A SPECIAL TEAM(leadered by anesthesiologist) to manage shocked patients.

Definition:

- Shock is defined as the inadequate perfusion of tissue, such that the oxygen and blood volume delivery fails to meet the cellular metabolic and oxygen consumption needs.
- The pathophysiology underlying shock is related to the determinant of oxygen delivery which depends on: Cardiac output, the vascular integrity and resistance, and the oxygen content (determined by hemoglobin and oxygen saturation) .

Shock may originate via disturbances in the neural-hormonal regulation of heart rate and blood flow to the systemic vasculature, or with changes in preload, systemic resistive indices, or heart function .

Epidemiology / Pathophysiology:

Classically, four types of shock are described based on the physiologic disturbance causing
etiology .

1. ***Hypovolemic Shock:*** when the intravascular volume is depleted from blood or fluid losses .
2. ***Distributive Shock:*** occurs due to inappropriate vasodilatation of the peripheral blood vessels (sepsis, anaphylaxis, drug reactions, endocrine, and neurogenic abnormalities).
3. ***Obstructive Shock*** **:** is associated with obstruction of the heart or the great vessels (tension pneumo/ hemothorax and cardiac tamponade). leads to high pressure in the chest which effectively obstructs venous return and diminishes cardiac output leading to inadequate perfusion). Massive pulmonary embolism may impede outflow of the right heart and lead to ventilation and perfusion mismatch .
4. ***Cardiogenic Shock*** **:** is failure of the "pump" and may arise from Acute Coronary Syndrome (ACS), mechanical failure, and/or arrhythmias.

Patients who do not meet the above classification may be termed “undifferentiated shock”.
Alternatively, patients may have a combination of the above clinical scenarios; for instance, a
trauma patient with hemorrhagic shock may also have neurogenic shock, or a patient with septic
shock may sustain a myocardial infarction further complicating the clinical picture.
***obstructive,cardiac shock is an distributive one>

Signs , Symptoms and Physiological Responses:

In a patient presenting with hypotension and concern for shock, the clinician must evaluate for diagnostic clues to the underlying cause and type of shock.

*At the onset of shock, the process is compensated and often reversible .

**Preferential circulation is given to the vital organs, and peripheral and splanchnic vasoconstriction diverts blood flow to the essential organs. The body releases stress hormones such a catecholamines, cortisol, antidiuretic hormones, renin -angiotensin system among other adaptive responses to preserve fluid volume and to activate the “flight or fight” response !!!

- However, without prompt and aggressive treatment, a plasma volume loss of over 30% or a cardiac index less than 2.2 L/min may progress to end organ damage and cellular death.

I. Vital Signs: are important indicators of the patient's physiologic status .

1. *Temperature:* Fever or Hypothermia
2. *Heart rate:* heart rate is typically elevated in hypotension. To compensate for the low stroke volume while maintaining cardiac output per the equation:
   Cardiac Output = Heart Rate x Stroke Volume **

   Bradycardic or normal heart rates may be observed with neurogenic and cardiogenic shock.

Fever may point the examiner to search for signs of infection.
Hypothermia may accompany poor perfusion but may also be a paradoxical manifestation of infection or a symptom of endocrine dysfunction
**In distributive and hypovolemic shock
***&beta blockers or pace maker

3. B*lood pressure(BP)*: Hypotension defined as MAP <65 mm Hg is often a prominent feature of shock.

BP is related to cardiac output (CO) and systemic vascular resistance (SVR) by the following equation:

BP = CO x SVR

4. *Respiratory rate* :Tachypnea is commonly observed in patients with shock .why?

$CO_2 + H_2O \leftarrow\!\!-\!\!-\!\!-\!\!> HCO_3^- + H^+$

5. Oxygen saturation is typically preserved by increasing oxygen extraction. Saturations fall only at very late stages of hypoperfusion !

-However, patients may present with hypertension or swings of hyper and hypotension as their physiology decompensates
*or when ventilation perfusion mismatch occurs, as in pulmonary emboli or pneumothorax.

II. Examination: Applying the ABCDEs helps to efficiently evaluate the patient while also balancing management priorities concomitantly.

A. *Airway*: The airway should be assessed for patency

B. *Breathing*: The breath sounds should be equal on both sides of the chest on auscultation.

C. *Circulation*: is assessed with an evaluation of the peripheral pulses.

D. *Disability*: routinely assessed using the Glasgow Coma Scale (GCS) Low GCS (<8) is an indication for intubation, and a low threshold for intubation is required for any patient who is not protecting the airway .

E. *Exposure and secondary evaluation*: An exam of the patient's entire body is important in the critically ill patient. Evaluation of sources of infection, signs of bleeding, extremity perfusion and capillary refill, and volume status to determine the etiology of hypoperfusion

Care must be taken to secure an endotracheal tube in patient with inability to protect the airway or a Glasgow Coma Scale of <8.

c. Crystalloid infusion of at least 30cc/ kg should be administered to support the intravascular volume while assessment of acute blood loss is determined

c. Acral cyanosis of the extremities with a cold, clammy feel is consistent with obstructive, hypovolemic,
or cardiogenic shock. A warm dilated shock may be seen with distributive shock due to vasodilation of the peripheral vessels

Kussmaul's breathing: hyperventilation to compensate for metabolic acidosis manifesting as 'air hunger'

GLASGOW COMA SCALE(GCS)

Behaviour	Response
 Eye Opening Response	4. Spontaneously 3. To speech 2. To pain 1. No response
 Verbal Response	5. Oriented to time, person and place 4. Confused 3. Inappropriate words 2. Incomprehensible sounds 1. No response
 Motor Response	6. Obeys command 5. Moves to localised pain 4. Flex to withdraw from pain 3. Abnormal flexion 2. Abnormal extension 1. No response

→Clinical picture may include:

Signs of Organ Hypoperfusion

Multiorgan Dysfunction Syndrome (MODS)

Result is: end organ failure

Types of Shock:

HYPOVOLEMIC SHOCK :

Hypovolemic shock results from loss of the intravascular circulating volume from fluid loss or blood loss. It has four classes:

Class I shock (<500-750 ml): Small volumes of fluid loss are well tolerated due to the compensatory mechanisms of the body . Heart rate, blood pressure, and urine output are maintained.

Class II shock (750-1500 ml): As the body detects lower circulatory volumes, the heart rate increases to augment cardiac output. Blood pressure and urine output are maintained. Patients may experience mild anxiety.

-The body attempts to compensate for the lack of blood volume by diverting blood flow away from the extremities and intestinal circulation in favor of the heart and brain

Class III shock (1500-2000 ml): Ongoing volume loss greater than 1500-2000 ml overcomes the ability of the heart to maintain blood pressure, given that this equates to a 30-40% change in circulating volume, blood pressure decreases and urine output drops to preserve remaining circulatory volume . Patient is nearing irreversible shock and immediate, aggressive intervention with volume and blood replacement is necessary . Physical exam findings show peripheral vasoconstriction and cold, clammy extremities, dry mucous membranes, and pallor associated with extreme anemia .

Class IV shock is reached with > 2000ml or blood or >40% of the circulating volume is loss.
*Patients are lethargic, with extreme tachycardia, profound hypotension, and oliguria. The patient may be moribund once stage IV shock commences.
** Children and pregnant patients will often guard their physiology until the point of collapse
*** Tachycardia will be prominent feature of severe shock before hypotension manifests in late class III to IV hemorrhage, just before circulatory collapse .

- On the other, extreme of age, the elderly may not be able to mount a tachycardic response to hemorrhage because of beta blockade, medications, and pacer dependence. Additionally, the elderly typically have baseline hypertension, thus, they may not manifest the traditional level of hypotension SBP <100 mm Hg or MAP <65 mmHg despite profound volume loss.

Classes of Hypovolemic Shock:

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	**> 100**	> 120	> 140
Blood Pressure	Normal	Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Resp. Rate	14 – 20	**20 – 30**	**30 – 40**	**> 40**
UOP	> 30	**20 – 30**	**5 – 15**	**negligible**
Mental Status	sl. Anxious	mildly anx	**confused**	**lethargic**
Fluid	crystalloid	crystalloid	**blood**	**blood**

- Hypovolemic shock may arise from bleeding due to trauma or atraumatic bleeding (such as an aortic aneurysm rupture or gastrointestinal bleed).
- Fluid losses from the GI tract from excessive vomiting or diarrhea , malabsorption, or hormone imbalances, such as diabetes insipidus can result in excessive volume loss that may lead to shock if left untreated.

Diagnosis:

History and physical may direct the diagnosis of hypovolemia. A history of trauma,
recent surgery, or evidence of bleeding may help diagnose acute blood loss .

On exam, the patient initially appears to have a cold shock picture, and pallor may be evident in
the setting of bleeding . Hgb and Hct may be decreased in acute blood loss . Thus, in early blood loss, the hemoglobin may remain preserved while the circulating blood volume may be significantly reduced. Trends in Hb , Hct are a better assessment of blood loss than a single value.

What do you think about Hb in fluid loss?

-Conversely with severe fluid loss, hemo concentration may occur elevating the concentration of the Hgb and Hct relative to circulating plasma volume .

Basic metabolic Laboratory:

1.Electrolyte assessment : K , Ca

2. Acid/ Base: Large volume loss leads to poor oxygen delivery to the tissue and a transition to anaerobic metabolism in the tissue bed. Lactate is produced and lactic acid level elevates leading to a metabolic acidosis.

Compare between excessive vomiting and diarrhea status?

Renal function: In severe hypovolemic shock the Urea level is elevated often > 20mg/dl. As shock progresses, renal failure may occur from acute tubular necrosis and cause further elevation of the renal function parameters .

- GI losses may lead to hypokalemia. Massive hemorrhage with transfusion of banked blood may lead to low ionized calcium
  levels that need aggressive repletion for hemostasis.
- BUN blood urea nitrogen test

Coagulation studies :(PT/ INR; PTT, fibrinogen, fibrin related markers): In severe hemorrhagic shock coagulopathy, secondary to an overactivation of clot breakdown (termed fibrinolysis) may occur.

SVO2: Mixed venous oxygen saturation will decreased.*

Imaging: history and examination guided.

. X-Ray CXR + pelvic x-ray

. bedside ultrasound

. CT Scan in stable patients

. Angiography may localize sources of bleeding

. Direct peritoneal aspiration/lavage (DPA/ DPL) : free blood go for emergent laparotomy.

-* As o2 extraction will increased

Treatment:

Aggressive **replacement of volume** while attending to the underlying etiology is the mainstay of treatment of hypovolemic shock .

In traumatic bleeding, patients should be triaged per the ABCDE's of Advanced Trauma Life Support (ATLS) and to identify the source of bleeding.

All forms of hemorrhage necessitate large bore IV access (14 or 16 gauge IV or short, large diameter resuscitation lines) (ATLS trauma et al) .

Crystalloid resuscitation :

The new guide lines recommend that Crystalloids administration should be limited to 1-2 liters of IV fluid, with a transition to early blood and plasma resuscitation when it is available.

- Colloids can be given.
- Hypovolemic shock from fluid loss must also aggressively repleted with like fluid . Oral rehydration may be attempted in early forms.
- In severe dehydration IV isotonic crystalloids can be given(Normal Saline (NS) or Lactated Ringers (LR)) as a bolus of 20-30ml/kg and repeated every 5-10 minutes, may quickly restore circulating volume .

- This recent paradigm shift in management has been guided by recent evidence suggesting that a 1:2 resuscitation (1 unit of plasma for every 2 units of packed blood cells) leads to less overall product transfused, improved coagulopathy, and a mortality benefit in trauma and non-trauma
- **Care must be taken to avoid fluid overload in certain patient populations.
  Patients with heart failure, severe malnutrition, diabetic ketoacidosis (DKA)

  patients (Holcomb, 2015; Holcomb , Cotton, Johansen).

End Points of Resuscitation "Goal-directed therapy"

Use objective hemodynamic and physiologic values to guide therapy:

1. Urine output > 0.5 mL/kg/hr
2. CVP 8-12 mmHg
3. MAP 65 to 90 mmHg
4. Central venous oxygen concentration > 70%

DISTRIBUTIVE SHOCK

- Distributive shock results from the inappropriate vasodilation of the peripheral vasculature(decreased SVR).
- Septic shock, anaphylactic shock, and neurogenic shock are all examples of this pathophysiology.

Septic shock:

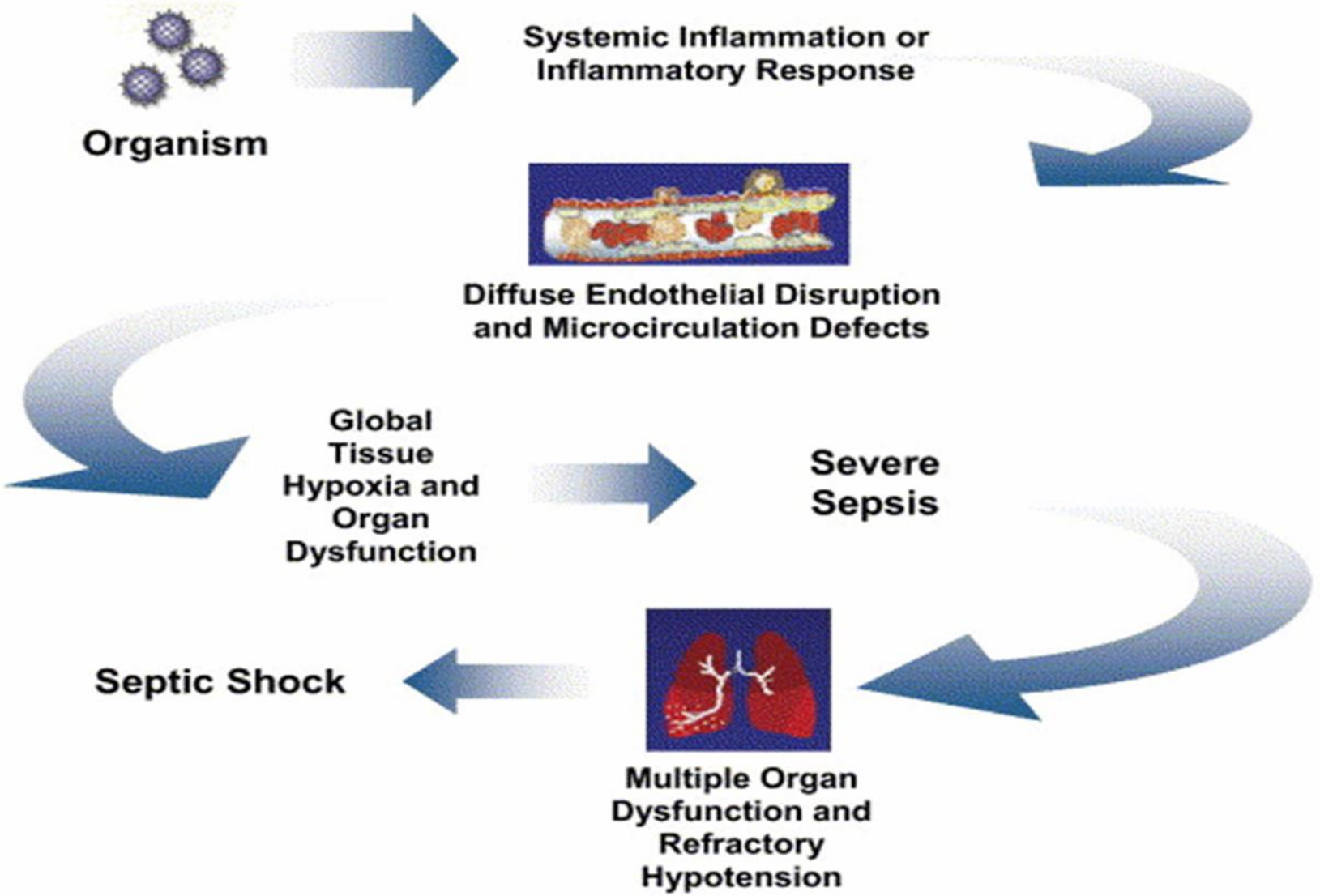
Severe sepsis and septic shock are highly lethal conditions that occur in response to infection.

. Severe sepsis is defined as a systemic host response to infection that leads to organ dysfunction. a mortality of 10%

. Septic shock is termed when the response to the infection leads to hypotension requiring vasopressors to maintain a mean arterial pressure (MAP) of >65mm Hg with concomitant lactic acidosis (>2 mmol/L) mortality up to 18-46% .

. In the United States, leading infective sources remain gram positive cocci from respiratory sources followed closely by gram negative bacterial pathogens .

The clinical sequela of sepsis is mediated by the host response to the pathogen.
Infection leads to the release of pro and anti-inflammatory cytokines. Tumor necrosis factor α, interleukins (IL) such as IL-2 ,IL-6,IL-8,and IL-10 lead to the recruitment of macrophages, neutrophils, and monocytes which compounds the responses by secreting more cytokines such as leukotrienes and prostaglandins that lead to systemic alterations in perfusion, microcirculation, cell death, and organ dysfunction. Each system of the body is affected by the host response to infection.


Organism
Systemic Inflammation or Inflammatory Response
Diffuse Endothelial Disruption and Microcirculation Defects
Global Tissue Hypoxia and Organ Dysfunction
Severe Sepsis
Multiple Organ Dysfunction and Refractory Hypotension
Septic Shock

Diagnosis:

Two or more of SIRS criteria

- Temp > 38 or < 36 C
- HR > 90
- RR > 20
- WBC > 12,000 or < 4,000

Plus

presumed existence of infection

Treatment

- 1. Infection source control : Broad spectrum antibiotics are strongly recommended within 1 hour of sepsis recognition .
- Identification of the infectious source with cultures, preferably drawn before antibiotic
  administration, is recommended to assist in diagnosis and to inform antibiotic stewardship
  2. Resuscitation and life support in critically ill patients :

- Fluid management - a 30ml /kg crystalloid infusion in the first 3 hours of the identification of sepsis is strongly recommended to support blood pressure

To date, a simple bedside test, the passive leg raise(PLR), remains one of the most clinically relevant, evidence based bedside evaluations to cardiac output → BP (Monnet).

. Additionally, urine output greater than 0.5cc/kg in the non-oliguric patients is also a helpful determination of adequate volume status

- Pressors are often needed to support blood pressure in septic shock patients Norepinephrine , Epinephrine , Vasopressin

- Typically, after 3 pressors, refractory shock consideration of corticosteroids is often empirically started to treat Critical Illness-Related Corticosteroid Insufficiency (CIRCI)

- Mechanical ventilation is often needed to support patient with sepsis and sepsis related acute respiratory distress ARDS.

- Renal replacement therapy may be required in cases of severe acute kidney injury with hyperkalemia, severe acidosis, uremia or volume overload.

Neurogenic shock:

Neurogenic shock describes the hemodynamic changes resulting from a sudden loss of autonomic tone due to spinal cord injury. It is commonly seen when the level of the injury is above T6.

. This is estimated to occur in up to 20% of cervical spine injuries .

.While Spinal shock, refers to loss of all sensation below the level of injury and is not circulatory in nature.

- Diagnosis:
- In traumatic injury, the physical exam may suggest a high level spinal cord injury. Which can be confirmed by imaging CT or MRI.
- Treatment: (supportive).

After ruling out other concomitant forms of shock, fluids and pressor use may be required to support the patient.

-Mean arterial blood pressure should be sustained to 85-90 mmHg to aid in spinal perfusion

Anaphylactic shock:

- Allergic reactions(Exaggerated immunological responses to antigenic(eg, antibiotic***, muscle relaxants*, latex**) stimulation in previously sensitized persons) liberate vasodilatory substances such as histamine through immunoglobulin E (Ig-E)(Anaphylaxis). Or a direct activation of the complement may trigger mast cell degranulation and basophil to release of histamine leading to "anaphylactoid reactions".
- Incidence is about 1:3000 to 1:20000(mortality is 4%).
- Despite different mechanisms BOTH are clinically indistinguishable and equally life-threatening depending on the severity of the reaction.
- Massive systemic vasodilation occurs leading to cardiovascular collapse, facial and tongue swelling leading to airway compromise, and bronchospasm of the airways.
- Diagnosis is largely clinical(exposure to an antigen via respiratory system, eyes, skin, IV, IM, or peritoneal).

-Immunological IGE or non-immunological reaction.

Treatment: ABC's

Stop the exposure to the trigger while assessing the patient's airway and hemodynamic stability is equal to therapy .

1. Airway: Lip and tongue swelling, called angioedema, as well as pharyngeal and glottic swelling may compromise the airway.
   a. For signs of impending airway compromise, securing an endotracheal tube early is a priority.
   b. Supplemental O2 and continuous monitoring are necessary.

2. Epinephrine injection(0.01-0.5 mg IV or IM) depending on the severity, with repeat dosing every 5-15 minutes as needed. A drip(IVInfusion) can be prepared for refractory response.

3. IV access should be emergently obtained and normal saline administered to support the patient to normotension.

4. Albuterol is a bronchodilator for bronchospasm and can be given as a nebulizer.

5*. Both H1 and H2 antihistamines should be administered to effectively block histamine receptors.

6*. Consider steroid for airway edema and severe reactions. 125mg of IV methylprednisolone is administered.

7. Epinephrine is the primary vasopressor of choice; however, others may be added to maintain MAP >65 mm Hg.

* Can be given prophylactically to decrease the severity of the reaction.

Adrenal insufficiency (AI):

- Acute adrenal crisis(Addisonian crisis) occurs if the adrenal gland is deteriorating (Addison's disease, primary adrenal insufficiency), if there is pituitary gland injury (secondary adrenal insufficiency), or if adrenal insufficiency is not adequately treated. Risk factors for adrenal crisis include physical stress such as infection, dehydration, trauma, or surgery, adrenal gland or pituitary gland injury, and ending treatment with steroids such as prednisone or hydrocortisone too early.

Clinically: Headache, fatigue, nausea, vomiting, fever and hypotension

Diagnosis: usually precipitated by external stress ↖
. An ACTH (cortrosyn) stimulation test shows low cortisol, is the "gold standard" diagnostic tool

Treatment : An intravenous or intramuscular injection of hydrocortisone must be given immediately. Supportive treatment of low blood pressure with intravenous fluids is usually necessary.

OBSTRUCTIVE SHOCK

Impediment to the flow of blood in the cardiopulmonary circuit results in obstructive shock

Classic examples of obstructive shock are from

1. Tension pneumothrax :A history of trauma ,hypoxemia
2. tamponade : Anxiety is prominent
3. pulmonary Emboli: Pulmonary emboli are blood clots that obstruct the pulmonary venous circulation. Which may lead to significant shunt
4. pericarditis
5. and restrictive cardiomyopathies

Treatment:

- The mainstay of treatment is directed at the underlying etiology of the obstruction to cardiopulmonary flow.

. In tension pneumothorax, placement of a needle in the midclavicular line in the second intercostal space will change a tension pneumothorax to a pneumothorax.

Pressurized hemothorax and moderate to large pneumothoraces should also be treated with a placement of a chest tube in the 4th intercostal space in the anterior axillary line.

Pulmonary emboli are treated with anticoagulation .

CARDIOGENIC SHOCK

- Cardiogenic shock results from failure of the cardiac "pump." Failure of forward delivery of blood and, therefore, oxygen to the tissues, leads to shock.
- When pressures elevate on the left side, pulmonary edema may occur, when there is right heart dysfunction, systemic congestion may result.
- in response to the reduction in the cardiac output, the systemic resistance increases in response to catecholamine stimulation and angiotensin II. Increased oxygen extraction in the tissue bed .
- **Causes:** myocardial infarct (MI); valvular insufficiency, and arrhythmias .

- **Diagnosis** :

Diagnosis of myocardial infarction per the WHO guidelines requires:
a. Detection of increase and/or decrease of cardiac biomarkers (preferably troponin) with at least 1 value above the 99th percentile of the upper reference limit
b. Evidence of myocardial ischemia with at least 1 of the following: symptoms, ECG changes, or supportive imaging

- ECG: ST elevation MI(STEMI)or Non-ST elevation MI (NSTEMI) and Unstable Angina (UA)
- Typically : crushing substernal chest pain, upper extremity, back, epigastric or jaw pain

Treatment:

- ABCs of resuscitation is essential to the management of cardiogenic shock. Intubation and ventilatory support (where appropriate) and obtaining excellent IV access and invasive blood pressure monitoring is crucial to effectively supporting the critical patient.

1. Medication administration
a. Aspirin 325 mg and heparin IV should be administered expediently.
b. Glycoprotein IIb/IIIa inhibitor with NSTEMI may be beneficial.
2. Pressor support to a MAP of 65 mmHg Norepinephrine and dopamine are first line agents.
3. Percutaneous Coronary Intervention (PCI) is the mainstay of therapy and should be
administered to all STEMI within 12 hours of symptoms and, all patients with cardiogenic
shock regardless of time of onset
4. Alternatively, if PCI cannot be administered within 120 minutes of arrival, then fibrinolytic therapy should be administered in absence of contraindication. Return of symptoms or failure to improve is an indication for emergent transfer to a PCI center.
6. Coronary Artery Bypass Grafting (CABG) is indicated in patients with STEMI with difficult PCI
6. Mechanical support
a. Intra-Aortic Balloon Pump (IABP) is a type of mechanical circulatory support

Summary

Type	PAOP	C.O.	SVR
HYPOVOLEMIC	↓	↓	↑
CARDIOGENIC	↑	↓	↑
DISTRIBUTIVE	↓ or N	**varies**	↓
OBSTRUCTIVE	↑	↓	↑

Goals of Shock Resuscitation

- Airway patency
- Control Work of Breathing
- Optimizing Circulation
- End Points of Resuscitation
  Goal-directed therapy: Use objective hemodynamic and physiologic values to guide therapy

1. Urine output > 0.5 mL/kg/hr
2. CVP 8-12 mmHg
3. MAP 65 to 90 mmHg
4. Central venous oxygen concentration > 70%

In general, support and treat the cause…

Thank you