SHOCK

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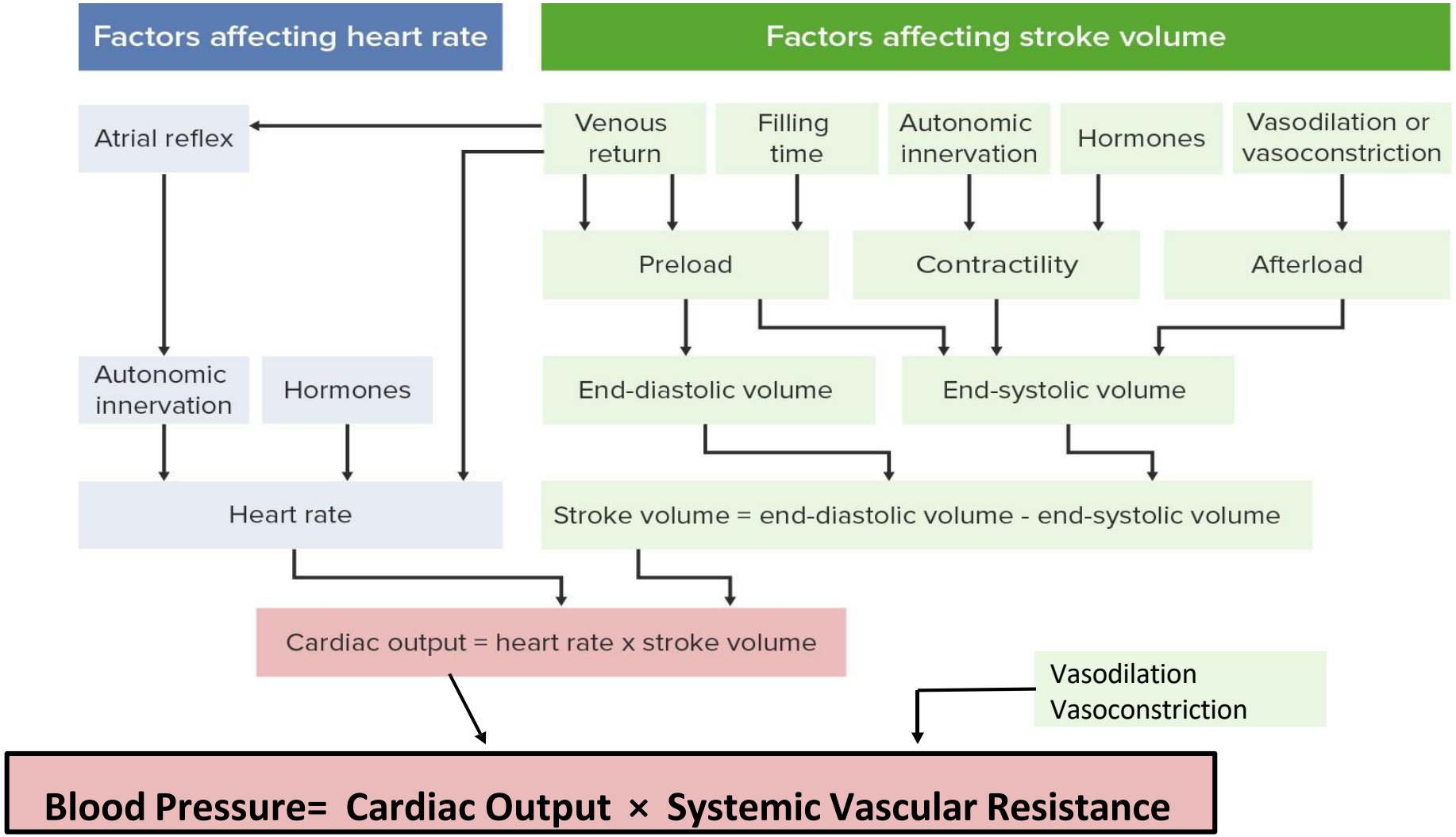
Objectives

- Definition
- Pathophysiology
- Stages
- Types
  - Hypovolemic
  - Cardiogenic
  - Obstructive
  - Distributive
    - Anaphylactic
    - Septic
    - Neurogenic

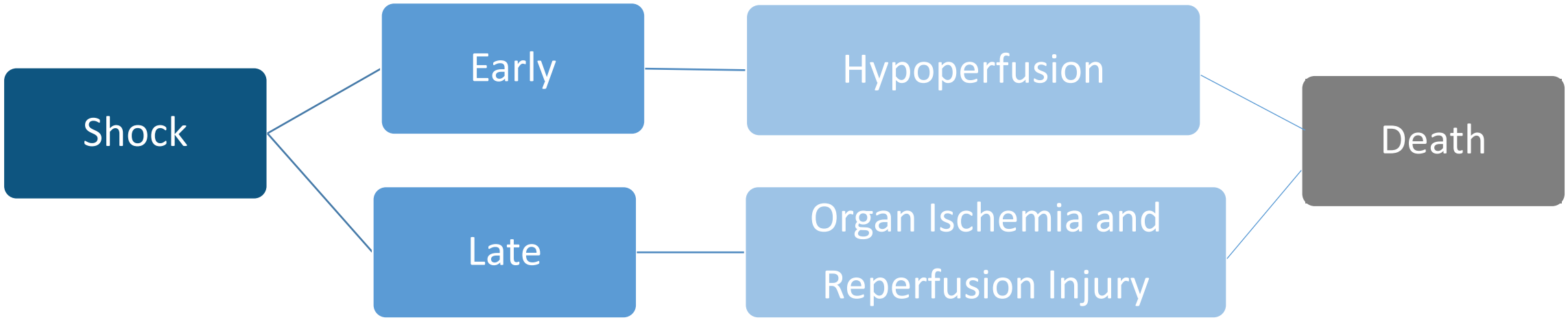
Physiology



https://www.lecturio.com/concepts/cardiac-mechanics/

Definition

- Shock: is a life-threatening condition of circulatory failure, causing inadequate oxygen delivery to meet cellular metabolic needs and oxygen consumption requirements, producing cellular and tissue hypoxia.
- The effects of shock are initially reversible, but rapidly become irreversible, resulting in multiorgan failure (MOF) and death.



Pathophysiology

1- Cellular

- With insufficient delivery of oxygen and glucose, cells switch from aerobic to anaerobic metabolism. Anaerobic respiration increases lactic acid.
- After cell exhaustion, failure of anaerobic respiration, and cell death ensued by autodigestive enzymes and cell contents are released into the circulation leading to hyperkalemia.

2- Microvascular

- Hypoxia
- Acidosis
- Complement and Neutrophils
- ORS and Cytokines
- Leaky Endothelium
- Edema
- Immune and Coagulation

Pathophysiology

3- Systematic

- CVS: ↓in preload and afterload lead to activation of baroreceptors and ↑ catecholamines (tachycardia and systematic vasoconstriction)
- RS: compensatory respiratory alkalosis by ↑ respiratory rate
- Renal: low perfusion → low filtration (GFR) → low urine output
  low blood pressure → activation of RAAS → sodium and water retention.
- Endocrine: ADH release → vasoconstriction and reabsorption of water, cortisol release from adrenal cortex contributing to sodium and water reabsorption and sensitizes the cells to catecholamines.
- GI: ↓ blood flow hypoactive bowel sounds.

Pathophysiology

4- Reperfusion Injury

- Acid and potasium load that has built up can lead to myocardial depression and hypotension
  and further injury due to flushing of cellular and humeral response elements
  (complement and neutrophiles)

Stages of Shock

	Compensated Compensatory mechanisms are effective and homeostais is maintained	**Decompensated** Compensatory mechanisms start to fail continued decreased cellular perfusion and altered capillary permeability are the hallmarks of this stage			**Irreversable** Death is Inevitable
Severity		Mild	Moderate	Severe	• No response to vasopressors • Myocardial depression and loss of responsiveness • Due to improper resuscitation or severe shock
Lactic Acidosis	+	++	++	+++	
Urine Output	Normal	Normal	(<.5 ml/kg/hr) ↓	(0 ml/kg/hr) ↓	
Consciousnous Level	Normal	Mild Anxiety	Drowsy	Comatose	
Respiratory Rate	Normal	Increased ↑	Increased ↑	Labored ↓	
Heart Rate	Mild increase ↑	Increased ↑	Increased ↑	Increased ↑	
Blood Pressure	Normal	Normal But decreased Pulse Pressure ↓	Mild decrease ↓	Hypotensive ↓	
Key Labs	Glu (↑) PH (↑) PaO2(↓) PaCO2 (↓)	Liver enzymes (↑) thrombocytopenia bleeding times (↑)			Glu (↓), ammonia (↑), K(↑), PH (↓)

Limitations to Accuracy

- BP: young people may have a normal BP despite profound shock due to vasoconstriction and stroke volume, also elderly who are hypertensive may be normotensive relative to normal readings .
- HR: affected by beta blockers use and in patients with penetrating trauma where there is hemorrhage with little tissue damage may have paradoxical tachycardia.
- Cool extremeties and poor capillary refill: are not found in septic shock.

Hypovolemic Shock

- Most common type of shock (decreased intravascular volume)

Parameter	Effect
Cardiac Output	↓ Decreased
Vascular resistance	↑ Increased
Venous pressure	↓ Decreased
Mixed venous saturation	↓ Decreased: increased oxygen extraction by the cells.
Base deficit	↑ Increased

Hypovolemic Shock Stages

	Stage 1	Stage 2	Stage 3	Stage 4
Blood loss (%)	<15	15–30	30–40	>40
Blood loss (cm^3)	<750	750–1500	1500–2000	>2000
Pulse rate	<100	>100	>120	>140
Respiratory rate	14–20	20–30	30–40	>35
Blood pressure	Normal	Decreased	Decreased	Decreased
Mental state	Normal/slightly anxious	Mild anxiety	Confusion and Lethargy	Confusion

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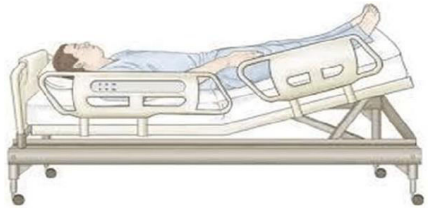
Hypovolemic Shock Causes

- 1- Hemorrhagic
  a. External bleeding
     (wounds)
  b. Internal bleeding
     (hemothorax, hemoperitoneum)
  c. Exteriorization of internal bleeding
     (melena, hematemesis, hemoptysis)

- 2- Non Hemorrhagic :
  a. Poor fluid intake
     (dehydration)
  b. More fluid loss
     (diarrhea, vomiting, renal losses)
  c. Increased evaporation
     (overheated enviroment, burns)
  d. Third spacing
     (bowel obstruction and pancreatitis)

Hypovolemic Shock Management

- Correct underlying cause: in patients who are actively bleeding it's counterproductive to start fluid therapy without controlling the site of hemorrhage.
  Increasing blood pressure merely increases bleeding from the site while fluid therapy cools the patient and dilutes available coagulation factors
- Warm fluids
- Supportive therapy with vasopressors
- Nursing care:
  - Ensure a patent airway
  - IV access Access should be through short, wide-bore catheters that allow rapid infusion of fluids as necessary.
  - O2
  - Modified trendelenberg position
  - Vitals every 5 minutes



Hypovolemic Shock Management

FLUID THERAPY:

- There’s no ideal resuscitation fluid.
- There’s no overt difference between crystalloids and colloids (which are more expensive with worse side effect profiles).
- Moreover there’s no oxygen carrying capacity of crystalloids and colloids so if the patient is bleeding the ideal replacement is blood.
- Hypotonic solutions (dextrose etc.) are poor volume expanders and should not be used in the treatment of shock unless the deficit is free water loss (eg. diabetes insipidus) or patients are sodium overloaded (eg. cirrhosis).

Hypovolemic Shock Management

Dynamic Fluid Response :

- After initial bolus of fluid (250-500ml/5-10 mins) the cardiovascular response is assessed through BP, CVP, HR.
- Responders: have an improvement in their cardiovascular status which is sustained. These patients are not actively losing fluid but require filling to a normal volume status.
- Transient responders: have an improvement which then reverts to the previous state over the next 10–20 minutes. These patients have moderate ongoing fluid losses (either overt hemorrhage or further fluid shifts reducing intravascular volume).
- Non-responders: are severely volume depleted and are likely to have major ongoing loss of intravascular volume, usually through persistent uncontrolled hemorrhage.

Hypovolemic Shock Management

End point of ressucitation:

- Using normal pulse, bp and UOP as an indicator to stop reessucitation may lead to occult hypoperfusion (state of normal vital signs and continued under perfusion) it is manifested only by a persistent lactic acidosis and low mixed venous oxygen saturation so …
- Resuscitation algorithms directed at correcting global perfusion end points (base deficit, lactate, mixed venous oxygen saturation) rather than traditional end points have been shown to improve mortality and morbidity in high-risk surgical patients

Hypovolemic Shock Management

Vasopressors and Ionotropic Support:

- Vasopressor or inotropic therapy is not indicated as first-line therapy in hypovolemia
- Administration of these agents in the absence of adequate preload rapidly leads to decreased coronary perfusion and depletion of myocardial oxygen reserves.
  Administration of inotropic or chronotropic agents to an empty heart will rapidly and permanently deplete the myocardium of oxygen stores and dramatically reduce diastolic filling and therefore coronary perfusion.
  Patients will enter the unresuscitatable stage of shock as the myocardium becomes progressively more ischaemic and unresponsive to resuscitative attempts.

Cardiogenic Shock

- Primary failure of heart to pump blood

Parameter	Effect
Cardiac Output	↓ Decreased
Vascular resistance	↑ Increased
Venous pressure	↑ Increased (associated with pulmonary and systemic edema)
Mixed venous saturation	↓ Decreased
Base deficit	↑ Increased

Cardiogenic Shock Causes

- Cardiomyopathic
  Myocardial infarction, Myocarditis, Drug-induced (eg, beta blockers)

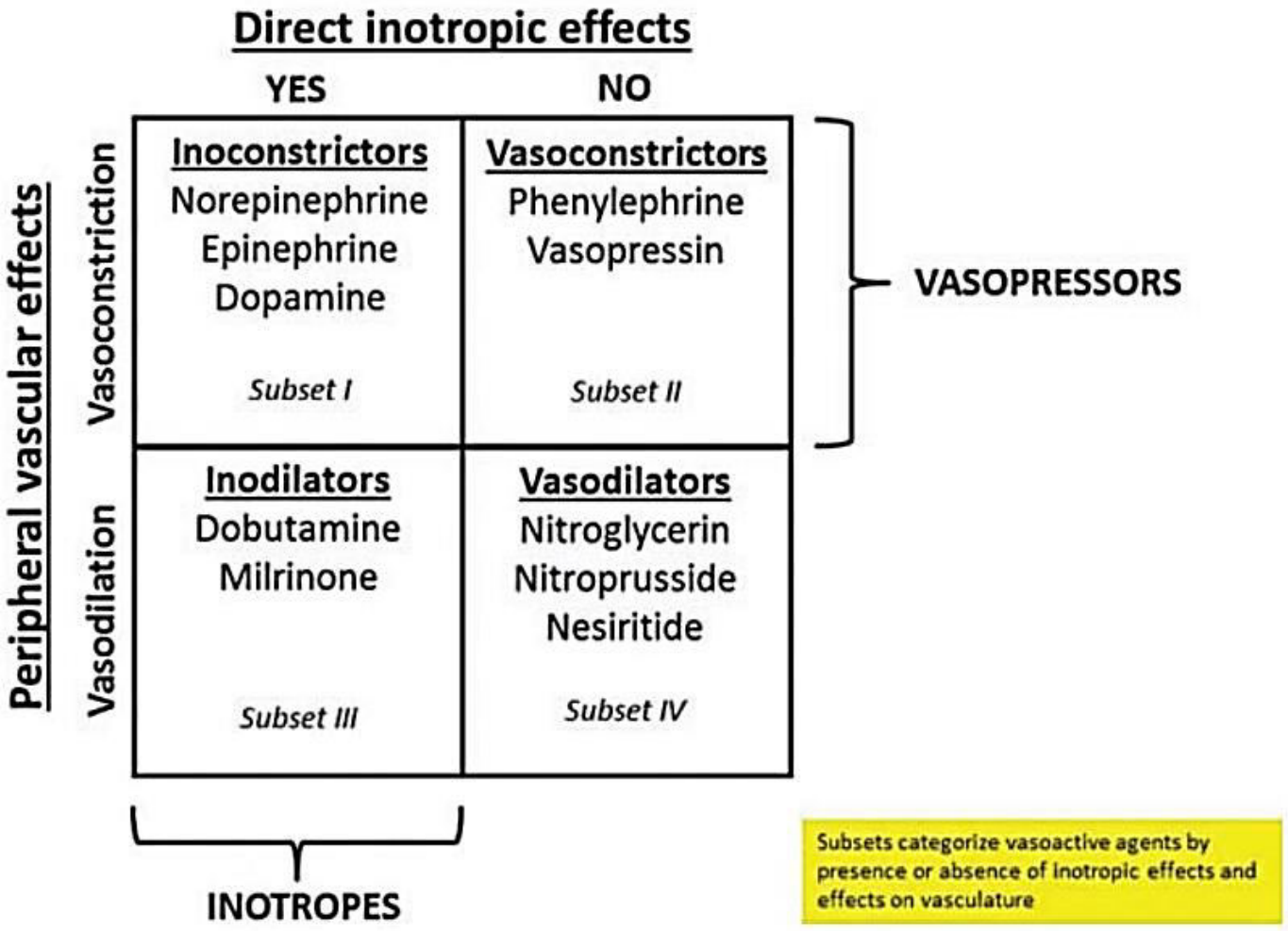
- Arrhythmogenic
  Tachyarrhythmia (A.fib, V.tach), Bradyarrhythmia (complete heart block)

- Mechanical
  Severe valvular insufficiency, acute valvular rupture (papillary or chordae tendineae rupture), valvular stenosis, ventricular septal wall defect (severe)

Cardiogenic Shock Management

- ABC: (IV fluid are likely to be harmful , instead we give them diuretics “ This is relative according to patient case”)
- Vasopressor: dopamine (dose dependent adrenergic agonist effects) to improve cardiac contractility
- Inotrope: dobutamine (Pure Beta agonist) / Milrinone (increase the heart's contractility and decrease pulmonary vascular resistance)
- Treat underlying cause.
- The intra-aortic balloon pump (IABP): is a mechanical device that increases myocardial oxygen perfusion and indirectly increases cardiac output through afterload reduction

Vasoactive Agents Classification



Obstructive Shock

- Decreased preload due to mechanical obstruction of filling

Parameter	Effect
Cardiac Output	↓Decreased
Vascular resistance	↑Increased
Venous pressure	↑Increased
Mixed venous saturation	↓Decreased
Base deficit	↑Increased

Obstructive Shock Causes

- 1- cardiac temponade
- 2- pneumothorax
- 3- massive pulmonary embolus
- 4- air emboli

Obstructive Shock Management

- Pain relief and referral to CCU
- Bed rest and O2 adminestration
- ECG monitoring
- Drugs like ionotropic vasodialators
- Specific
  tension pneumothorax : needle aspiration at 2nd intercostal space along midclavicular line
  pulmonary embolism : high flow oxygen intubation
  temponade : pericordiocentesis

Obstructive Shock Management

- General strategy :

- Assessment
- Analysis
- Planning/implentation of intervention : If there is initial doubt about the cause of shock, it is safer to assume the cause is hypovolemia and begin with fluid resuscitation, and then assess the response.
- Evaluation and continuous monitoring
- Documentation of interventions.

Obstructive Shock Management

- assessment and analysis

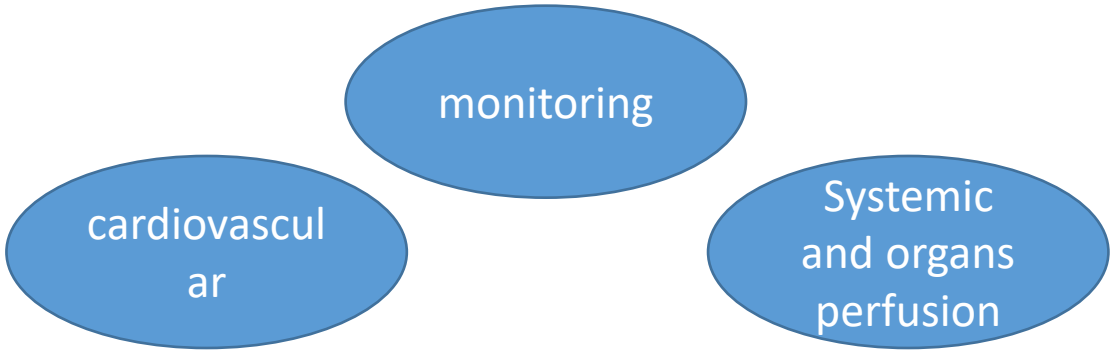
- Monitor vitals
- Monitor mental status
- Monitor lab values

- ECG
- chest Xray.
- continuous pulse oximetry.

- ABGs and lactate
- CBC : RBC normal HCT decrease HGB increase
- Coagulation panel : PTT/PT/INR/ D-dimer
- Troponin/ CKMB / BUN / TCK
- Glucose initially elevated then decrease after glycogen depletion

Obstructive Shock Management

- Evaluation and continuous monitoring
- The **minimum** standard for monitoring of the patient in shock is continuous heart rate and oxygen saturation monitoring, frequent non-invasive blood pressure monitoring and hourly urine output measurements. Most patients will need more aggressive invasive monitoring, including central venous pressure and invasive blood pressure monitoring



Obstructive Shock Management

- CARDIOVASCULAR:
- Cardiovascular monitoring at a minimum should include continuous heart rate (ECG), oxygen saturation and pulse waveform and non-invasive blood pressure. Patients whose state of shock is not rapidly corrected with a small amount of fluid should have central venous pressure monitoring and continuous blood pressure monitoring through an arterial line
- 1- CVP
  There's no normal cvp we monitor the response after fluid bolus for 10-20 mins
  The normal CVP response is a rise of 2–5 cmH2O which gradually drifts back to the original level over 10–20 minutes. Patients with no change in their CVP are empty and require further fluid resuscitation. Patients with a large, sustained rise in CVP have high preload and an element of cardiac insufficiency or volume overload.

Obstructive Shock Management

- 2- COP
- A- invasive : pulmonary artery catheter
  B- noninvasive: Doppler ultra- sound, pulse waveform analysis and indicator dilution methods
- allows not only assessment of the cardiac output but also the systemic vascular resistance and, depending on the technique used, end diastolic volume (preload) and blood volume. Early consideration should be given to instituting cardiac output monitoring on patients who require vasopressor or inotropic support.

Obstructive Shock Management

- Systemic and organ perfusion
- Systemic perfusion :
  - base deficit and lactate
  importance : diagnosis, monitoring, prognosis (Patients with a base deficit over 6 mmol/L have a much higher morbidity and mortality than those with no metabolic acidosis.), end point of resuscitation
  how ? : measured from arterial blood gas analyses (the frequency of measurements is limited, and they do not provide minute-to-minute data)

Obstructive Shock Management

- Mixed venous oxygen saturation: normal = 50-70 percent
- How ? : Accurate measurement is via analysis of blood drawn from a long central line placed in the right atrium , Estimations can be made from blood drawn from lines in the superior vena cava, but these values will be slightly higher

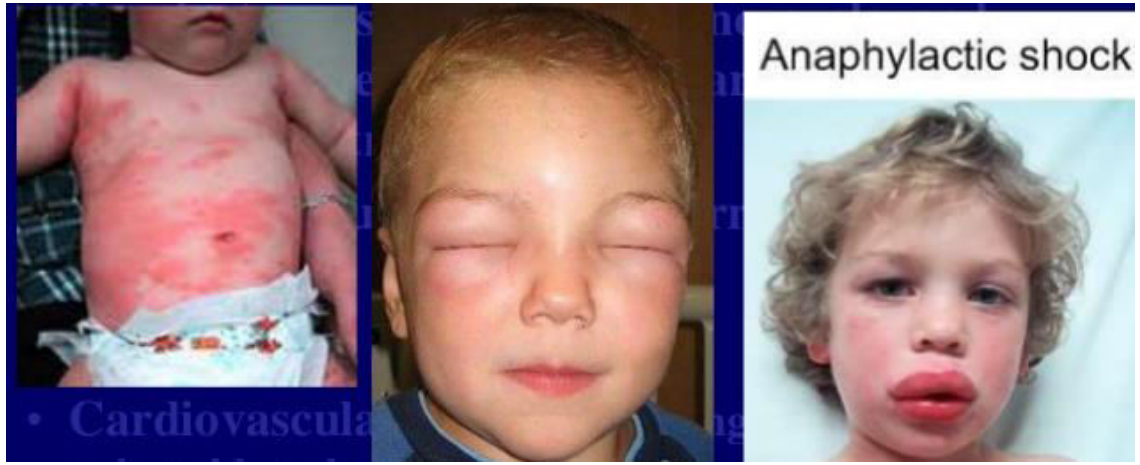
Distributive Shock

- A type of shock in which Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload and a resulting abnormally high cardiac output.

Includes :

- 1- anaphylactic shock
- 2 - septic shock
- 3- neurogenic shock

Anaphylactic Shock



- Exposure to previous sensitized antigen diffuse hypersensitivity reaction
- histamine production vasodilatation , bronchospasm and edema .
- Antigen examples : 1- food 2-Insect stings 3- drugs : Penicillin
- Specific sign:
- – circulatory: Significant and sudden drop in BP and Tachycardia
  – cautanous :Frequent coetaneous signs: rash,erythema,itching, urticaria, angioedema
  – Respiratory signs: dyspnoea, stridor ,wheezing

- ***Most common sign → skin reaction***
- ***Most dangerous sign (laryngeal edema)***

Treatment

Non pharmacotherapy :

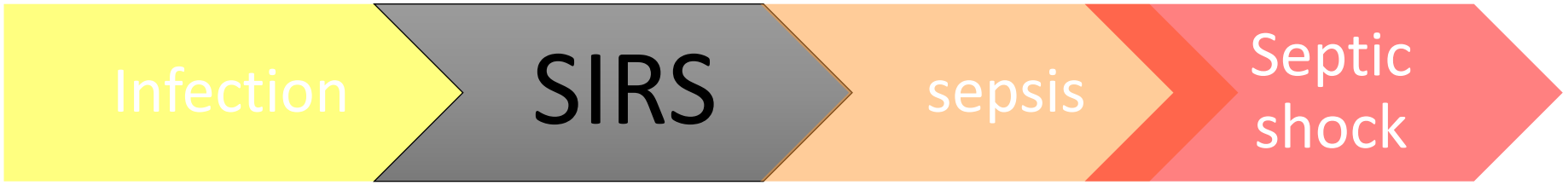
- Ensure adequate airway and maintain O2
- Remove the source of allergy
- Cardiac monitoring and/or pulse oximetry
- High-flow oxygen
- Fluid resuscitation with isotonic crystalloid solution
- Supine position (or position of comfort if dyspneic or vomiting) with legs elevated

Pharmacotherapy

- The primary drug treatments for acute anaphylactic reactions are epinephrine and H1 antihistamines.
- -Adrenergic agonists (eg, epinephrine)
- -H1 Antihistamines (eg, diphenhydramine, hydroxyzine) -H2 receptor antagonists (eg, cimetidine, ranitidine, famotidine) -Bronchodilators (eg, albuterol) -Corticosteroids (eg, methylprednisolone, prednisone) to prevent reccunt atta -Vasopressors (eg, dopamine)

Septic Shock

- Septic shock is the most common cause of distributive shock. Caused by an overwhelming systemic infection resulting in vasodilation leading to hypotension. The primary infection is most commonly caused by bacteria, but also may be by fungi, viruses or parasites.
- Sepsis can rang from infection and bacteremia to sepsis and septic shock, which can lead to multiple organ dysfunction syndrome (MODS) and death.
- The systemic inflammatory response syndrome (SIRS) is no longer included in the definition since it is not always caused by infection .



Inflammation In the blood stream

Two or more of the following conditions:

- Temperature > 38°C or <36°C
- Heart rate > 90 beats per min
- Respiratory rate > 20 breaths per min
- White blood count > 12,000 / mm³ or < 4,000 / mm³

Systemic inflammatory response syndrome (2 or more of the following)	
Finding	Value
Temperature	<36 °C (96.8 °F) or >38 °C (100.4 °F)
Heart rate	>90/min
Respiratory rate	>20/min or PaCO2<32 mmHg (4.3 kPa)
WBC	<4x10⁹/L (<4000/mm³), >12x10⁹/L (>12,000/mm³), or 10% bands

Keep Clinical Picture in mind

Systemic response to infection:

- SIRS
- Documented or highly suspected Infection Confirmed infection

SEPSIS SPECTRUM

Sepsis associated abnormalities:

- Sepsis Reversible
- Evidence of organ dysfunction, hypoperfusion, or hypotension

elevated lactate, or decreased urine output

Sepsis induced hypotension:

- Systolic BP < 90 mmHg despite adequate fluid resuscitation → 30ml/kg
- Evidence of organ dysfunction, hypoperfusion, or hypotension

Multiorgan dysfunction syndrome Septic shock + Irreversible Organ failure and mostly liver failure

SOFA score:

The sequential organ failure assessment score (SOFA score), → known as the sepsis-related organ failure assessment score, is used to track a person's status during the stay in an intensive care unit (ICU) to determine the extent of a person's organ function or rate of failure.

Symptoms and signs

- Symptoms and signs specific to an infectious source (eg, cough and dyspnea may suggest pneumonia, pain and purulent exudate in a surgical wound may suggest an underlying abscess).
- Arterial hypotension (eg, systolic blood pressure [SBP] <90 mmHg, mean arterial pressure [MAP] <70 mmHg, an SBP decrease >40 mmHg, or less than two standard deviations below normal for age). Because a sphygmomanometer may be unreliable in hypotensive patients, an arterial catheter may be needed
- Temperature >38.3 or <36ºC.
- Heart rate >90 beats/min or more than two standard deviations above the normal value for age.
- Tachypnea, respiratory rate >20 breaths/minute.

Signs Of End-organ Perfusion:

- Warm, flushed skin may be present in the early phases of sepsis. As sepsis progresses to shock, the skin may become cool due to redirection of blood flow to core organs. Decreased capillary refill, cyanosis, or mottling may indicate shock.
- Additional signs of hypoperfusion include altered mental status, obtundation or restlessness, and oliguria or anuria.
- Ileus or absent bowel sounds are often an end-stage sign of hypoperfusion

Laboratory signs

- Similarly, laboratory features are nonspecific and may be associated with abnormalities due to the underlying cause of sepsis or to tissue hypoperfusion or organ dysfunction from sepsis. They include the following:
- Leukocytosis (white blood cell [WBC] count >12,000 microL–1) or leukopenia (WBC count <4000 microL–1)
- Normal WBC count with greater than 10 percent immature forms.
- Hyperglycemia (plasma glucose >140 mg/dL or 7.7 mmol/L) in the absence of diabetes.
- Plasma C-reactive protein more than two standard deviations above the normal value.
- Acute oliguria (urine output <0.5 mL/kg/hour for at least two hours despite adequate fluid resuscitation).
- Creatinine increase >0.5 mg/dL or 44.2 micromol/L.

Laboratory signs

- Coagulation abnormalities (international normalized ratio [INR] >1.5 or activated partial thromboplastin time [aPTT] >60 seconds).
- ●Thrombocytopenia (platelet count <100,000 microL–1).
- ●Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 micromol/L).
- ●Adrenal insufficiency (eg, hyponatremia, hyperkalemia), and the euthyroid sick syndrome can also be found in sepsis.
- ●Hyperlactatemia (higher than the laboratory upper limit of normal) – An elevated serum lactate (eg, >2 mmol/L) can be a manifestation of organ hypoperfusion in the presence or absence of hypotension and is an important component of the initial evaluation, since elevated lactate is associated with poor prognosis . A serum lactate level ≥4 mmol/L is consistent with, but not diagnostic of, septic shock.
- **Additional laboratory studies** that help characterize the severity of sepsis include a low platelet count, and elevated international normalized ratio, creatinine, and bilirubin. Although arterial and venous lactate correlate, arterial lactate measurements are more accurate and preferred.

Immediate Evaluation And Management

- The sepsis six :
- 1. Deliver high-flow oxygen
- 2. Take blood cultures
- 3. IV antibiotics
- 4. Measure serum lactate and send CBC
- 5. IV fluid replacement
- 6. Measurement of urine output

Management

Respiration

- Securing airway and correcting hypoxemia

- Supplemental oxygen should be supplied and oxygenation should be monitored continuously

- -Intubation and mechanical ventilation may be required to support the increased work of breathing that typically accompanies sepsis, or for airway protection since encephalopathy and a depressed level of consciousness frequently complicate sepsis

Management

- Establish venous access

- Central venous access should be established as soon as possible in patients with suspected sepsis.
- However, the insertion of a central line should not delay the administration of resuscitative fluids and antibiotics.

- A central venous catheter (CVC) can be used to infuse intravenous fluids, medications (particularly vasopressors), and blood products, as well as to draw blood for frequent laboratory studies. While a CVC can be used to monitor the therapeutic response by measuring the central venous pressure (CVP) and the central venous oxyhemoglobin saturation (ScvO2)

Initial investigations

- Quickly obtaining the following is preferable (within 45 minutes of presentation) but should not delay the administration of fluids and antibiotics:
- **-Complete blood counts** with differential, chemistries, liver function tests, and coagulation studies including D-dimer level. Results from these studies may support the diagnosis, indicate the severity of sepsis, and provide baseline to follow the therapeutic response.
- **-Serum lactate** – An elevated serum lactate (eg, >2 mmol/L or greater than the laboratory upper limit of normal) may indicate the severity of sepsis and is used to follow the therapeutic response
- **Peripheral blood cultures** - (aerobic and anaerobic cultures from at least two different sites), urinalysis, and microbiologic cultures from suspected sources (eg, sputum, urine, intravascular catheter, wound or surgical site, body fluids) from readily accessible sites.
- **-Arterial blood gas (ABG) analysis** – ABGs may reveal acidosis, hypoxemia, or hypercapnia.
- **-Imaging targeted at the suspected site of infection is warranted (eg, chest radiography, CT of chest and/or abdomen).**

Initial Resuscitative Therapy

- **The cornerstone of initial resuscitation is the rapid restoration of perfusion and the early administration of antibiotics**

- Tissue perfusion is predominantly achieved by the aggressive administration of intravenous fluids (IVF), usually crystalloids (balanced crystalloids or normal saline) given at 30 mL/kg (actual body weight), started by one hour and completed within the first three hours following presentation
- Empiric antibiotic therapy is targeted at the suspected organism(s) and site(s) of infection and preferably administered within the first hour.

Intravenous fluids (first three hours)

- **Volume** — Intravascular hypovolemia is typical and may be severe in sepsis. Rapid, large volume infusions of IVF (30 mL/kg) are indicated as initial therapy for severe sepsis or septic shock, unless there is evidence of significant pulmonary edema.
- **Choice of fluid** — Evidence from randomized trials and meta-analyses have found no convincing difference between using albumin solutions and crystalloid solutions (eg, normal saline, Ringer's lactate) in the treatment of sepsis or septic shock
- In our practice, we generally use a crystalloid solution instead of albumin solution because of the lack of clear benefit and higher cost of albumin.

Treating metabolic acidosis

- -The role of exogenous bicarbonate therapy in patients with lactic acidosis is controversial. Most experts believe that it is appropriate to use bicarbonate in acutely ill patients with profound lactic acidosis that has generated an arterial pH less than 7.1 and a serum bicarbonate of 6 mEq/L or less.

Empiric antibiotic therapy (first hour)

Initial evaluation of common sources of sepsis

Suspected site	Symptoms/signs*	Initial microbiologic evaluation ¶
Upper respiratory tract	Pharyngeal inflammation plus exudate ± swelling and lymphadenopathy	Throat swab for aerobic culture
Lower respiratory tract	Productive cough, pleuritic chest pain, consolidative auscultatory findings	Sputum of good quality, rapid influenza testing, urinary antigen testing (eg, pneumococcus, legionella; not recommended in children), quantitative culture of protected brush or bronchoalveolar lavage
Urinary tract	Urgency, dysuria, loin, or back pain	Urine culture and microscopy showing pyuria
Vascular catheters: arterial, central venous	Redness or drainage at insertion site	Culture of blood (from the catheter and a peripheral site), culture catheter tip (if removed)
Indwelling pleural catheter	Redness or drainage at insertion site	Culture of pleural fluid (through catheter)
Wound or burn	Inflammation, edema, erythema, discharge of pus	Gram stain and culture of draining pus, wound culture not reliable
Skin/soft tissue	Erythema, edema, lymphangitis	Culture blister fluid or draining pus; role of tissue aspirates not proven
Central nervous system	Signs of meningeal irritation	CSF cell count, protein, glucose, Gram stain, and culture Δ
Gastrointestinal	Abdominal pain, distension, diarrhea, and vomiting	Stool culture for Salmonella, Shigella, or Campylobacter; detection of Clostridium difficile toxin
Intra-abdominal	Specific abdominal symptoms/signs	Aerobic and anaerobic culture of percutaneously or surgically drained abdominal fluid collections
Peritoneal dialysis (PD) catheter	Cloudy PD fluid, abdominal pain	Cell count and culture of PD fluid
Genital tract	Women: Low abdominal pain, vaginal discharge Men: Dysuria, frequency, urgency, urge incontinence, cloudy urine, prostatic tenderness	Women: Endocervical and high vaginal swabs onto selective media Men: Urine Gram stain and culture
Bone	Pain, warmth, swelling, decreased use	Blood cultures, MRI, bone cultures at surgery or by interventional radiology
Joint	Pain, warmth, swelling, decreased range of motion	Arthrocentesis with cell counts, Gram stain, and culture

CSF: cerebrospinal fluid; PD: peritoneal dialysis; MRI: magnetic resonance imaging.
*Fever is frequently seen with all conditions.
¶ Suggested initial tests are not considered to be comprehensive. Additional testing and infectious disease consultation may be warranted.
Δ Bacterial antigen and/or molecular testing may also be appropriate in selected patients. Refer to UpToDate topics on diagnostic testing for meningitis.

Management

Source Control	Examples
Drainage	• Intra-abdominal abscess
	• Thoracic empyema
	• Septic arthritis
Debridement	• Pyelonephritis, cholangitis
	• Infected pancreatic necrosis
	• Intestinal infarction
	• Mediastinitis
Device removal	• Infected vascular catheter
	• Urinary catheter
	• Infected intrauterine contraceptive device
Definitive control	• Sigmoid resection for diverticulitis
	• Cholecystectomy for gangrenous cholecystitis
	• Amputation for clostridial myonecrosis

Management

Choosing a regimen

- most patients with sepsis without shock, we recommend empiric broad spectrum therapy with one or more antimicrobials to cover all likely pathogens

-Many patients with septic shock, particularly those suspected to have gram negative sepsis, should receive combination therapy with at least two antimicrobials from two different classes (ie, combination therapy) depending on the organisms that are considered likely pathogens

Duration – The duration of antibiotics should be individualized. For most patients, the duration of therapy is typically 7 to 10 days

Patients Who Fail Initial Therapy

- Patients having persistent hypoperfusion despite adequate fluid resuscitation and antimicrobial treatment should be reassessed for fluid responsiveness, adequacy of the antimicrobial regimen and septic focus , as well as the accuracy of the diagnosis of sepsis and/or its source and the possibility that unexpected complications or coexisting problems have occurred (eg, pneumothorax following CVC insertion)

- Other options for treatment of persistent hypoperfusion such as the use of vasopressors, glucocorticoids, inotropic therapy, and blood transfusion

Vasopressors

- Maintain MAP ≥ 65 mm Hg.
- Norepinephrine or dopamine centrally administered are the initial vasopressors of choice.
- Use epinephrine as the first alternative agent in septic shock when BP is poorly responsive to norepinephrine or dopamine.
- In patients requiring vasopressors, insert an arterial catheter as soon as practical.
- Glucocorticoids — Guidelines recommend against the routine use of glucocorticoids in patients with sepsis. However, corticosteroid therapy is appropriate in patients with septic shock that is refractory to adequate fluid resuscitation and vasopressor administration
- Inotropic therapy — A trial of inotropic therapy may be warranted in patients who fail to respond to adequate fluids and vasopressors, particularly those who also have diminished cardiac output
- **Give RBC when Hb < 7.0 g/dl to target HB 7.0–9.0 g/dl in adults.**
- -Exceptions include suspicion of concurrent hemorrhagic shock or active myocardial infarction

Supportive Therapies

- Nutrition
- Stress ulcer prophylaxis
- Neuromuscular blocking agents
- Venous thromboembolism prophylaxis
- Intensive insulin therapy =**Keep blood glucose < 150 mg/dl**
- External cooling or antipyretics

Neurogenic Shock

- Occurs secondary to spinal cord injuries from vertebral body fractures of the cervical or high thoracic region that disrupt sympathetic regulation of peripheral vascular tone (there is failure of sympathetic outflow and adequate vascular tone)
- The classic symptoms include a slow heart rate due to loss of cardiac sympathetic tone and warm skin due to dilation of the peripheral blood vessels.
- Low blood pressure occurs due to decreased systemic vascular resistance as a result of lacking sympathetic tone which in turn causes pools of blood staying within the extremities and not being redirected to the core body. The slowed heart rate results from unopposed vagal tone activity.
- result in bradycardia, hypotension, cardiac dysrhythmias, reduced cardiac output, and decreased peripheral vascular resistance.
- The classic description of neurogenic shock consists of decreased blood pressure associated with bradycardia (absence of reflexive tachycardia due to disrupted sympathetic discharge), warm extremities (loss of peripheral vasoconstriction), motor and sensory deficits indicative of a spinal cord injury, and radiographic evidence of a vertebral column fracture.

Spinal v/s neurogenic shock

	Spinal shock	Neurogenic shock
Definition	Immediate temporary loss of total power, sensation and reflexes below the level of injury	Sudden loss of the sympathetic nervous system signals
BP	Hypotension	Hypotension
Pulse	Bradycardia	Bradycardia
Bulbocavernosus reflex	Absent	Variable
Motor	Flaccid paralysis	Variable
Time	48-72 hrs immediate after SCI	
Mechanism	Peripheral neurons become temporarily unresponsive to brain stimuli	Disruption of autonomic pathways → loss of sympathetic tone and vasodilation

Management

1-Hypotension

- 1. crystalloid (250mL boluses) and IVI – may not improve BP despite massive infusion (beware fluid overload and pulmonary oedema)
- 2. vasopressors eg noradrenaline, dopamine - after trial of volume replacement to Maintain organ perfusion.
- Consider CVP monitoring

2. Bradycardia

- 1. atropine
- 2. avoid over vagal stimulation with suction/NGT and ETT placement

THANK YOU