Sepsis Management with Minimally Invasive Hemodynamic Monitoring

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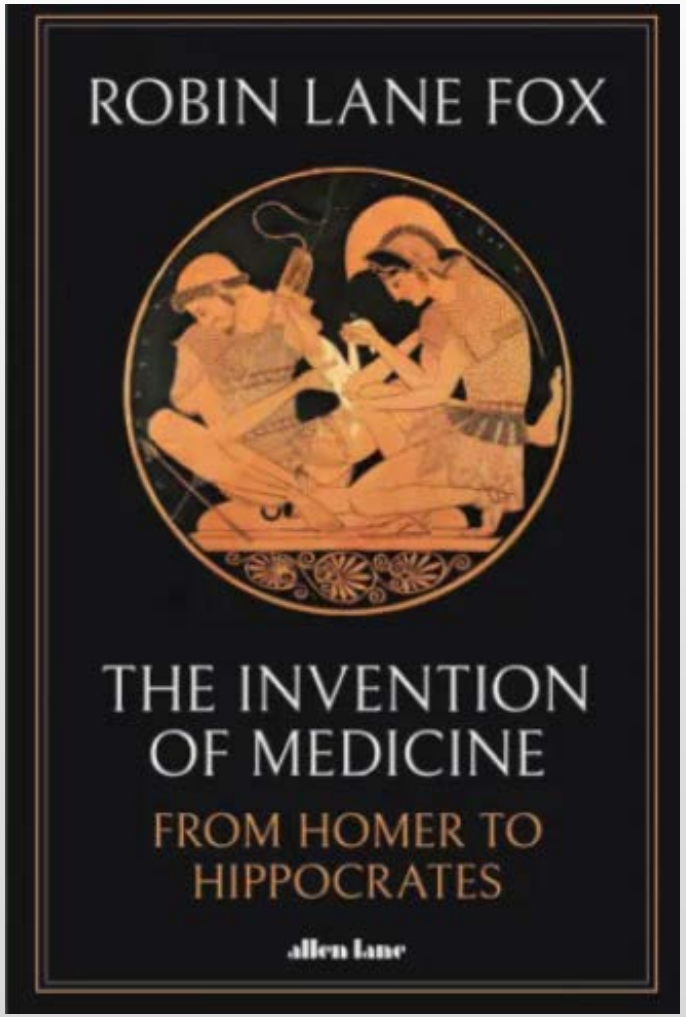
Learning Objectives

1. To identify the definition of sepsis
2. To recognize the progression of sepsis
3. To understand nursing role in identifying clinical signs and symptoms of sepsis
4. To understand minimally invasive monitoring of sepsis

Ancient Greece

- Sepsis dates back over *2,700* years ago when “SEPO” meant “ rot,” was mentioned in the ancient Greek poems of Homer.
- The term Sepsis was also used in the writings of Hippocrates, a Roman Physician. He viewed sepsis as a dangerous biological decay that could potentially occur in the body.

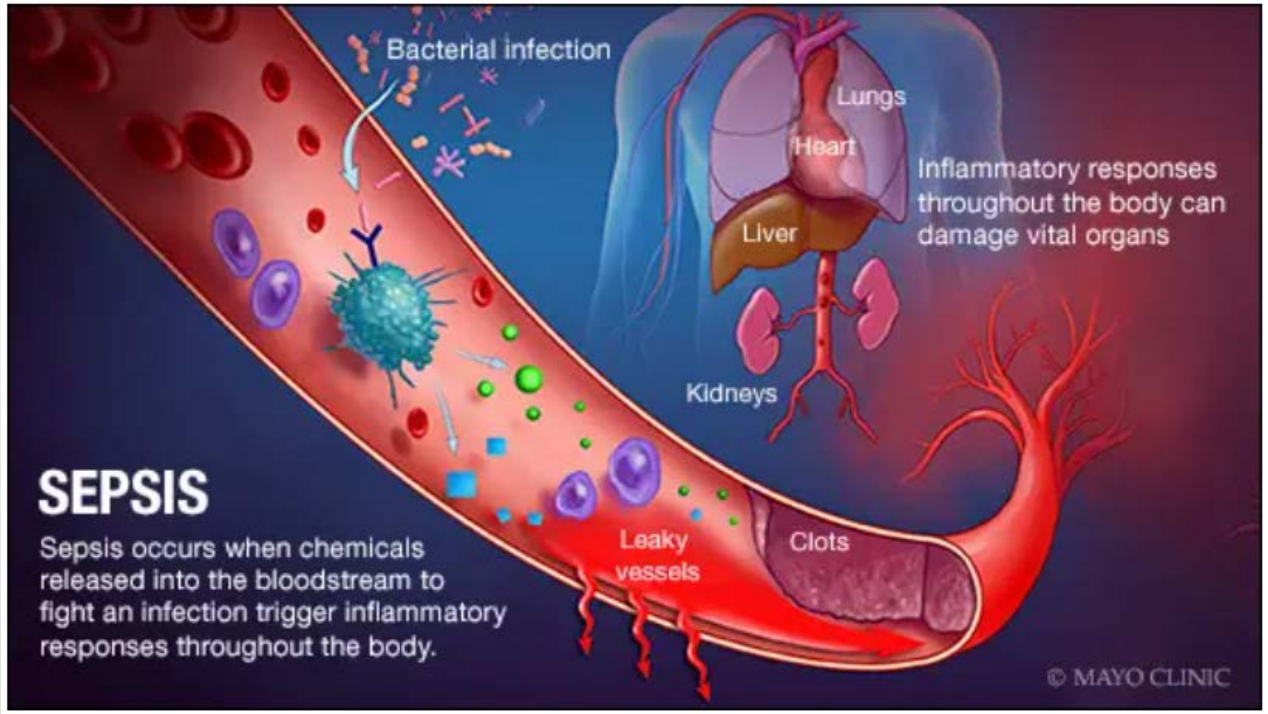


Sepsis Definition

- Sepsis is a life-threatening organ dysfunction caused by dysregulated host response to infection (Society of Critical Care Medicine, 2021).

- The most common infections that trigger sepsis in the occur in the lungs, urinary tract, gut and skin (CDC, 2020).

- Hospitals admit patients experiencing one of these Pathways;
  - Systemic Inflammatory Response Syndrome (SIRS)
  - Sepsis
  - Severe Sepsis
  - Septic Shock



Mayo Clinic Online. (2023). Retrieved from Understanding Septic Shock

U.S. Sepsis Facts

- Yearly, more than **1.7 million** people develop sepsis.
- At least 500,000 of them die each year, making this the **leading cause of hospital deaths**.
- Nearly 80% of people develop sepsis symptoms outside of the hospital.
- It's the **most expensive condition** treated in hospitals costing about $62 billion annually.
- It is the #1 cause for hospital re-admissions.

https://www.endsepsis.org/what-is-sepsis/sepsis-fact-sheet-2/

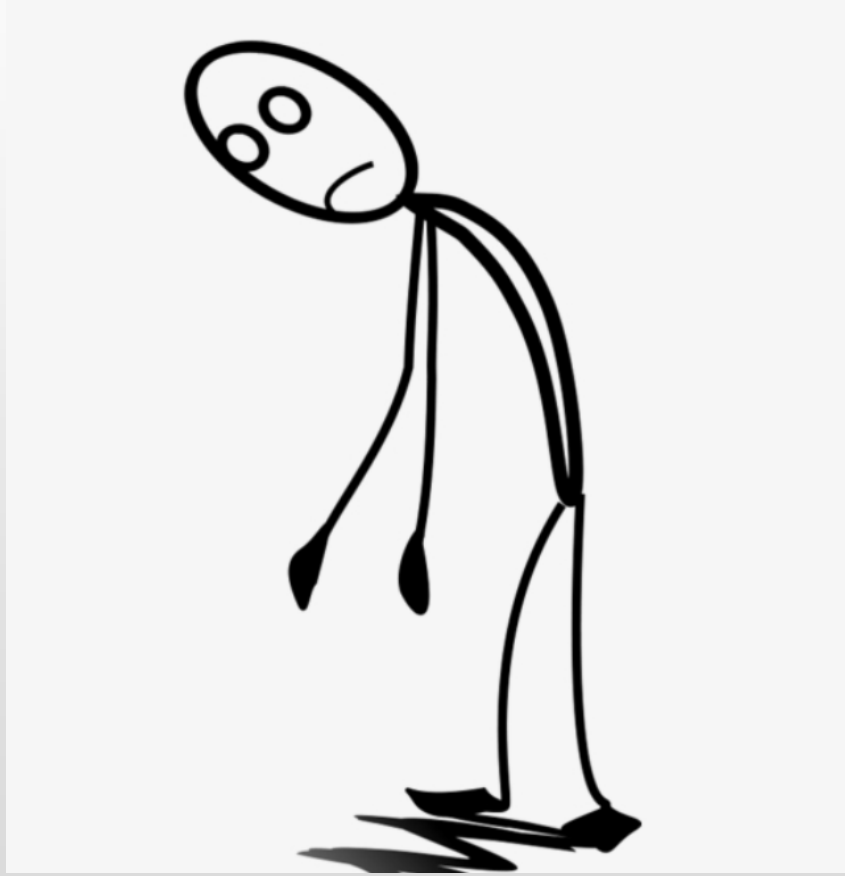
https://www.sepsis.org/sepsis-basics/what-is-sepsis/

Patient Population

It greatly affects:

- immunosuppressed patients (AIDS, transplant, genetic disorders)
- patients with severe illness (cancer)
- elderly and young people
- patients with multiple co-morbidities (HTN, HLD, DM)

Understanding the development of sepsis, recognizing symptoms, and monitoring oxygen balance can improve patient outcomes.



Systemic Inflammatory Response Syndrome (SIRS)

Sepsis begins with a systemic inflammatory response and presents with **two or more** of the following symptoms;

- hyperthermia >100.4F or (38C),
- hypothermia <96.8F or (36C),
  - More common w/ elderly, patients w/hepatic or renal failure
- tachycardia > 90 beats per minute
- tachypnea > 20 breaths per minute
- WBC >12,000 or < 4,000 or normal WBC with >10% bands.

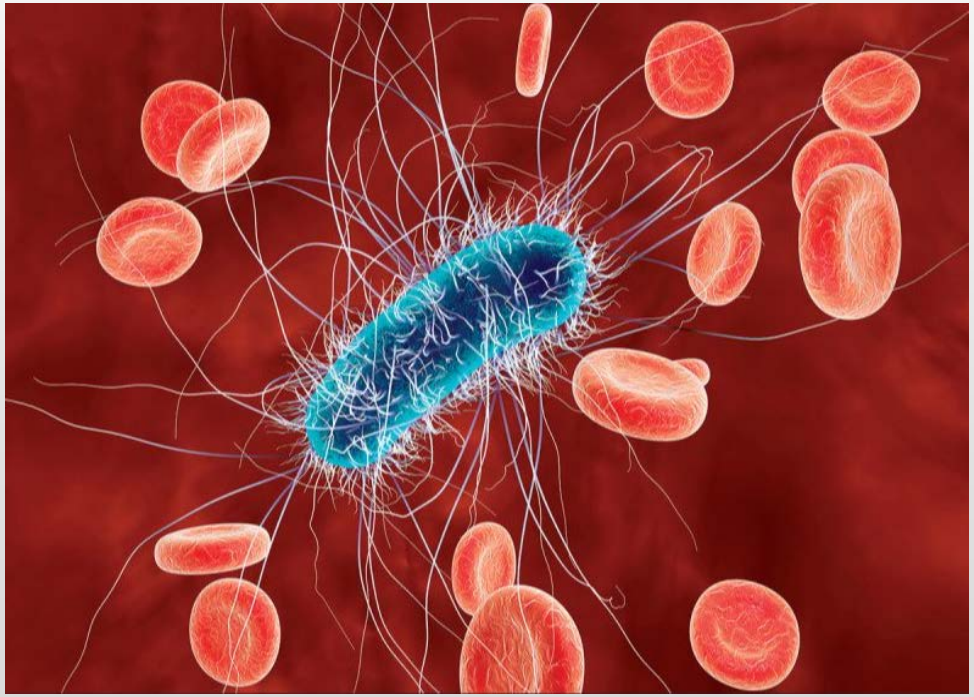
Chakraborty, R. K., & Burns, B. (2022). Systemic Inflammatory Response Syndrome. In *StatPearls*. StatPearls Publishing. [7]

Sepsis Diagnosis

Sepsis is confirmed when **two or more of the SIRS criteria** are present along with a documented infection validated by positive culture results.

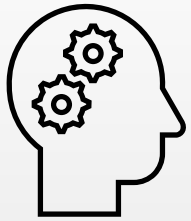
The symptoms of sepsis in its early stages can be vague and easy to overlook…if left untreated, sepsis can progress to severe sepsis or septic shock

(Sepsis Alliance, 2021).



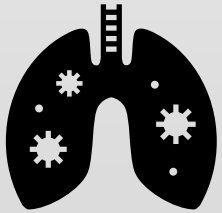
Severe Sepsis (1 / 2)

Severe Sepsis = Infection + sepsis symptoms + organ damage



- **Mental status** is affected by neuroinflammation and hypoperfusion causing confusion and/or lethargy.

- **Hypotension** occurs, contractility is impaired, which reduces cardiac output and ejection fraction.

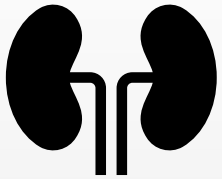


- Lung's vasculature becomes permeable inducing acute lung injury which causes **hypoxemia**.

- The increased demand for energy without sufficient supply of oxygen causes **lactic acid accumulation** in the blood. Lactate is elevated at **>2.0 mmol/L**

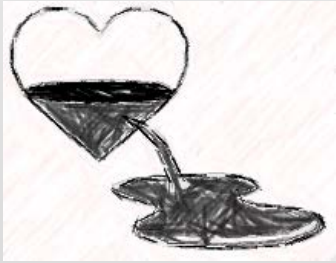
(Sepsis Alliance, 2023).

Severe Sepsis (2 / 2)



- **Kidneys** are affected, which is evident with elevated creatinine levels >2.0 mg/dL and **decreased urine output** <0.5mL/kg/hr. *Hypotension and DIC also contribute to kidney failure.

- There is also an alteration in the liver's ability **to transport bile acids and bilirubin** resulting in jaundice and elevated bilirubin levels > 2.0mg/dL.



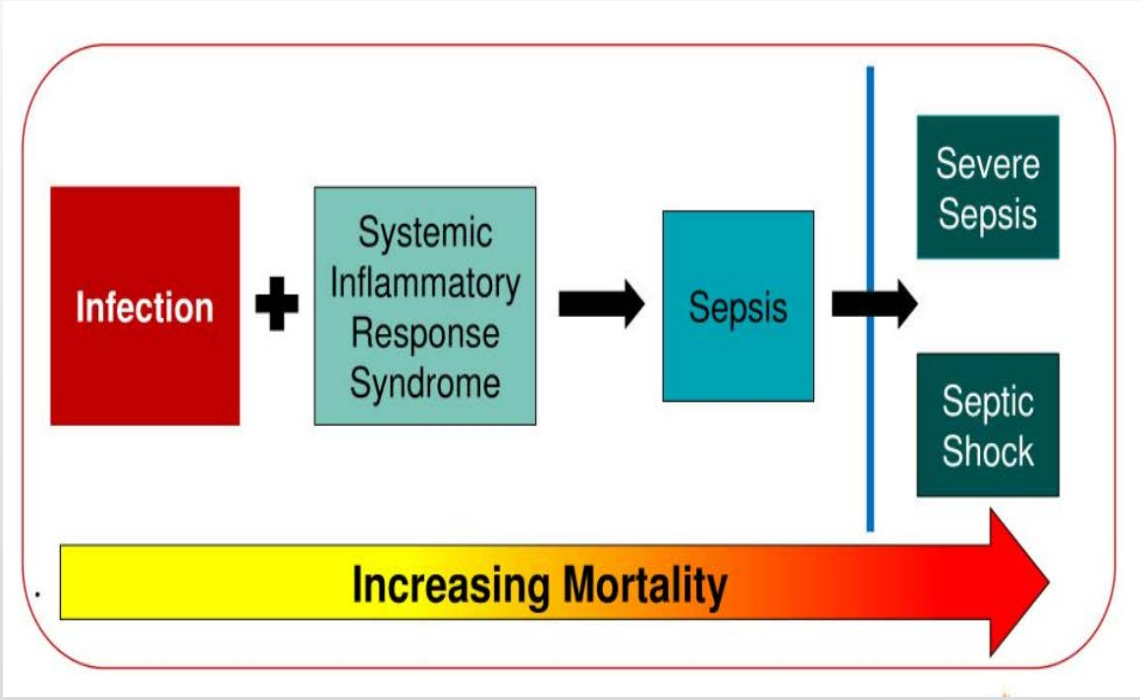
- Sepsis damages the lining of blood vessels making patients more susceptible to **coagulopathy** resulting in either **excessive clotting (DIC) or bleeding.** (INR > 1.5, a PTT, > 60 seconds, platelet count <100,000)

(Sepsis Alliance, 2023).

Septic Shock

The final, most severe form of sepsis is difficult to treat, often called "blood poisoning".

- **With loss of vascular tone, fluid is detained in the venous space instead of returning to heart,** causing organs to fail as they are unable to receive enough blood
- **Hypotension (MAP <65mmHg) is refractory to fluid bolus, inotropes and vasopressors are required to maintain blood pressure**
- **Lactic Acid can be > 4mmol/L**
- **Multi-organ failure**



Signs and Symptoms of Septic Shock

- Hypothermia
- Lethargy or coma
- Respiratory rate (fast or slow)
- Hypotension
- Tachycardia (pulse weak and thready)
- Skin is cool and pale
- Anuria

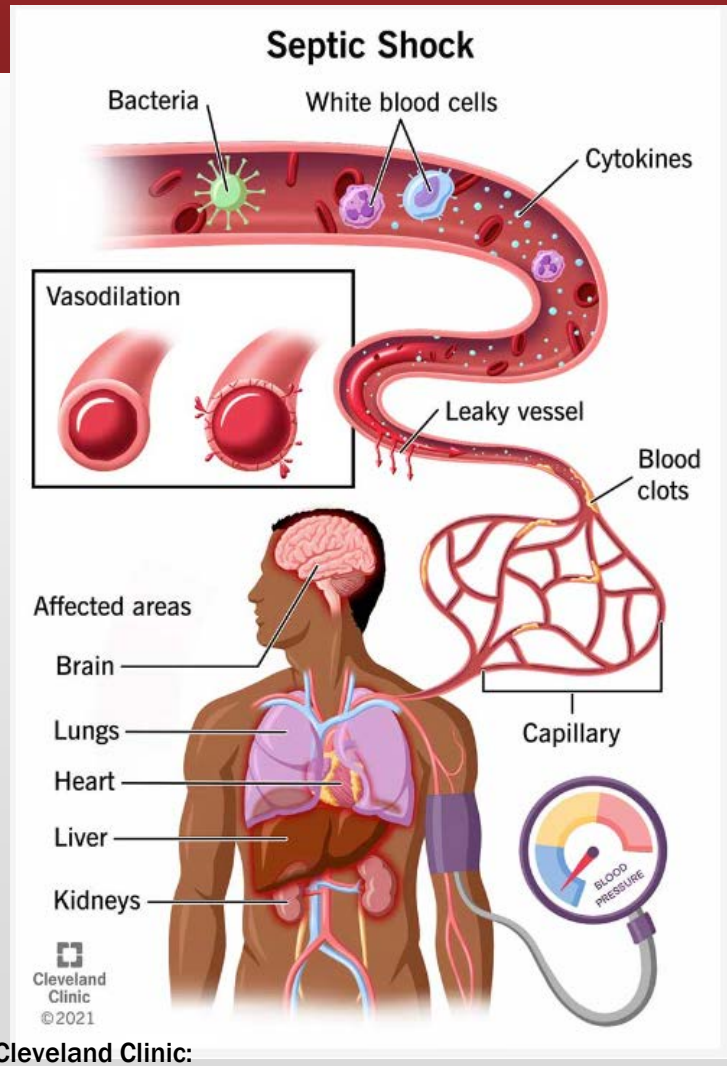


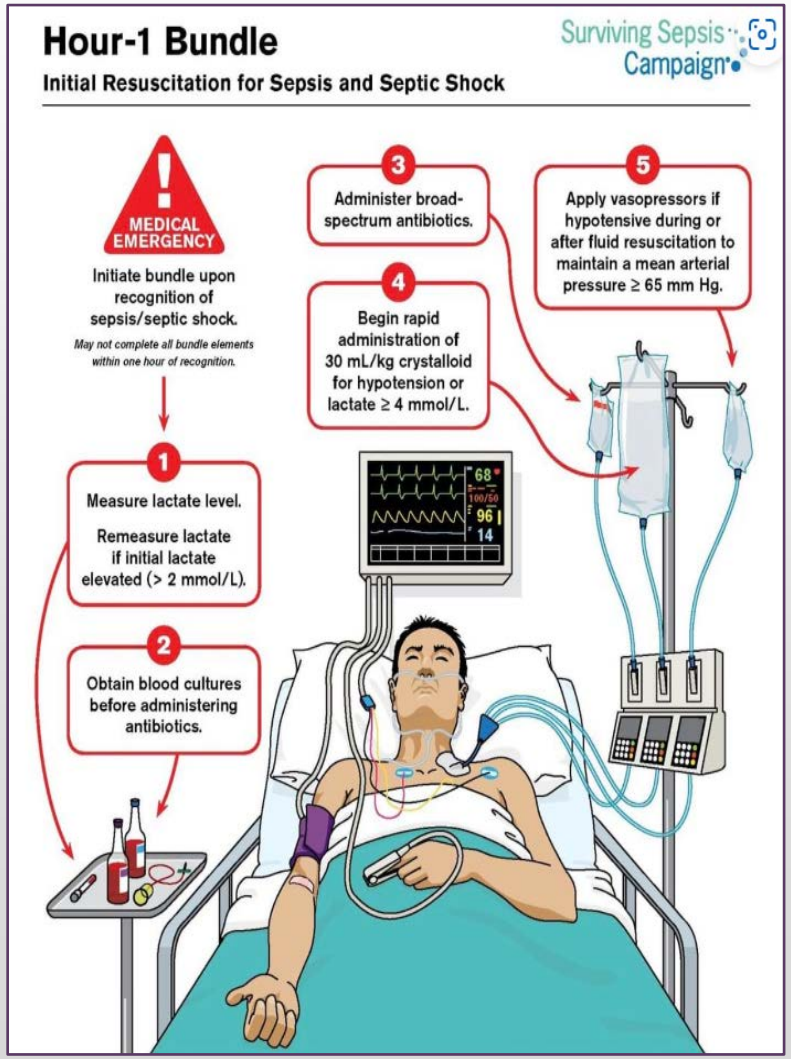
Photo by Cleveland Clinic: https://my.clevelandclinic.org/health/diseases/23255-septic-shock

2021 Surviving Sepsis Guidelines

Recommends reassessing volume status, tissue perfusion and lactate clearance

1. Measure Lactate (indicates oxygen balance)
2. Obtain Blood Cultures (finds microorganism's sensitivity to medication)
3. Administer STAT Antibiotics (do not delay if cultures are not easily obtained)
4. Begin fluid administration (30ml/kg)
5. Start vasopressors if fluid administration does not increase MAP by 10%

   (goal: MAP >65mm Hg)



Lactic Acidosis

Type A: With Tissue Hypoxia
Type B: Without Tissue Hypoxia

LACTATE > 2

START THINKING

LACTATE > 4
START WORRYING

Is enough O2 being delivered to organs?

- Hemoglobin
- Oxygen saturation (SaO2)
- Partial Pressure of Oxygen (PaO2)

Is there sufficient cardiac output?

- Severe acidosis impairs cardiac function and causes hypo-responsiveness to vasopressors

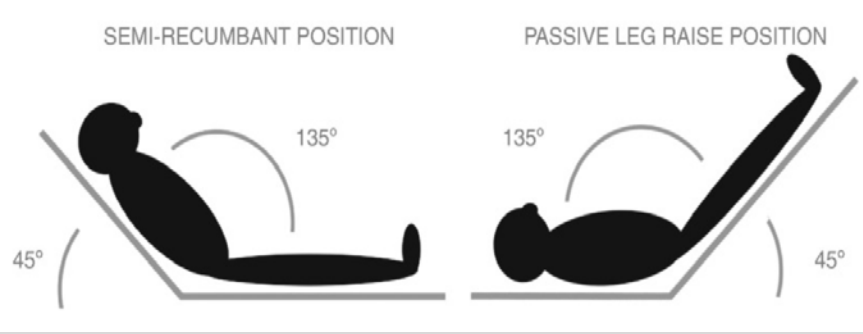
How can I improve O2 delivery?

- O2 delivery
- RBCs
- Improve CO with fluids or inotropes

Fluid Administration

PLR TEST:

- Passive Leg Raise Test: (PLR) raises the patient's legs (45 degrees) to induce a gravitational transfer of venous blood from the legs into the central circulation.
- The resulting effect is a transient increase in cardiac preload of ~150-300 mL.
- If PLR test increases CO or SV within 30-60 seconds, patient is fluid responsive



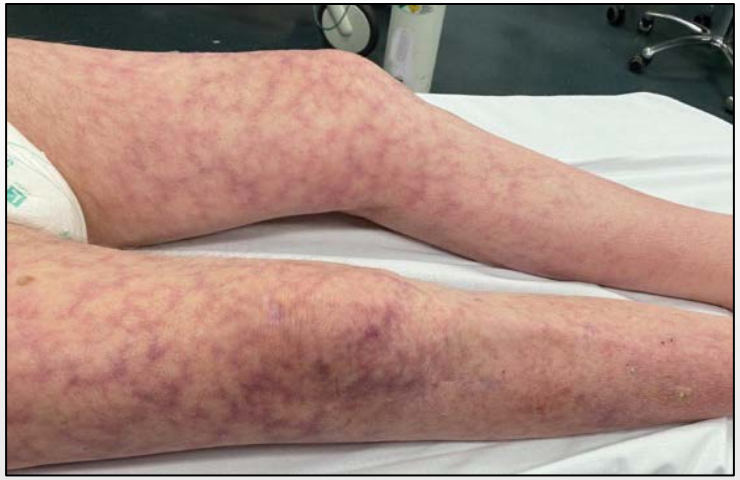
FLUID BOLUS:

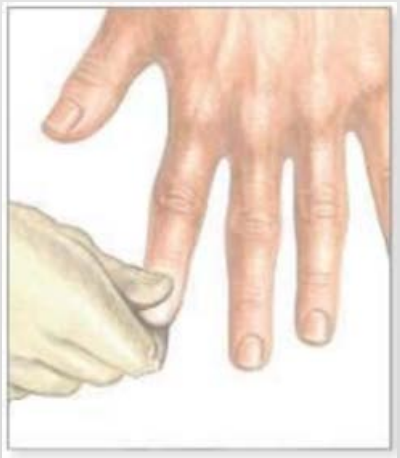
- Aimed at increasing cardiac output and tissue perfusion.
- Rapidly administer 250-500 mL fluid over 5-10 minutes.
  - Usually crystalloids, colloids or blood products
- Greater than 10% increase =fluid responder
- Less than 10% increase =non-fluid responder
- Good idea to connect CVP monitoring
- If the patient is fluid responsive, there will be an increase in cardiac output.

Minimally Invasive Monitoring

Monitoring Patient

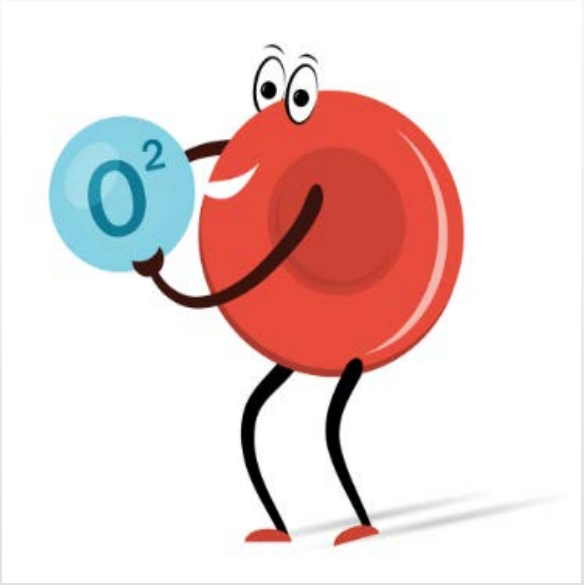
1. Evaluate oxygen saturation (SaO^2) with pulse ox (>95%)
2. Monitor Blood Pressure (arterial catheter preferred)
3. Assess perfusion
    - **Skin Mottling**: Patchy skin discoloration reflects blood flow reduction and skin hypoperfusion.
    - **Capillary Refill Time**: (CRT) Evaluates intravascular volume in critically ill patients. Goal is <3 sec.
5. Monitor Lactate concentration
6. If patient has arterial line, connect Flotrac





Components of Oxygen Delivery

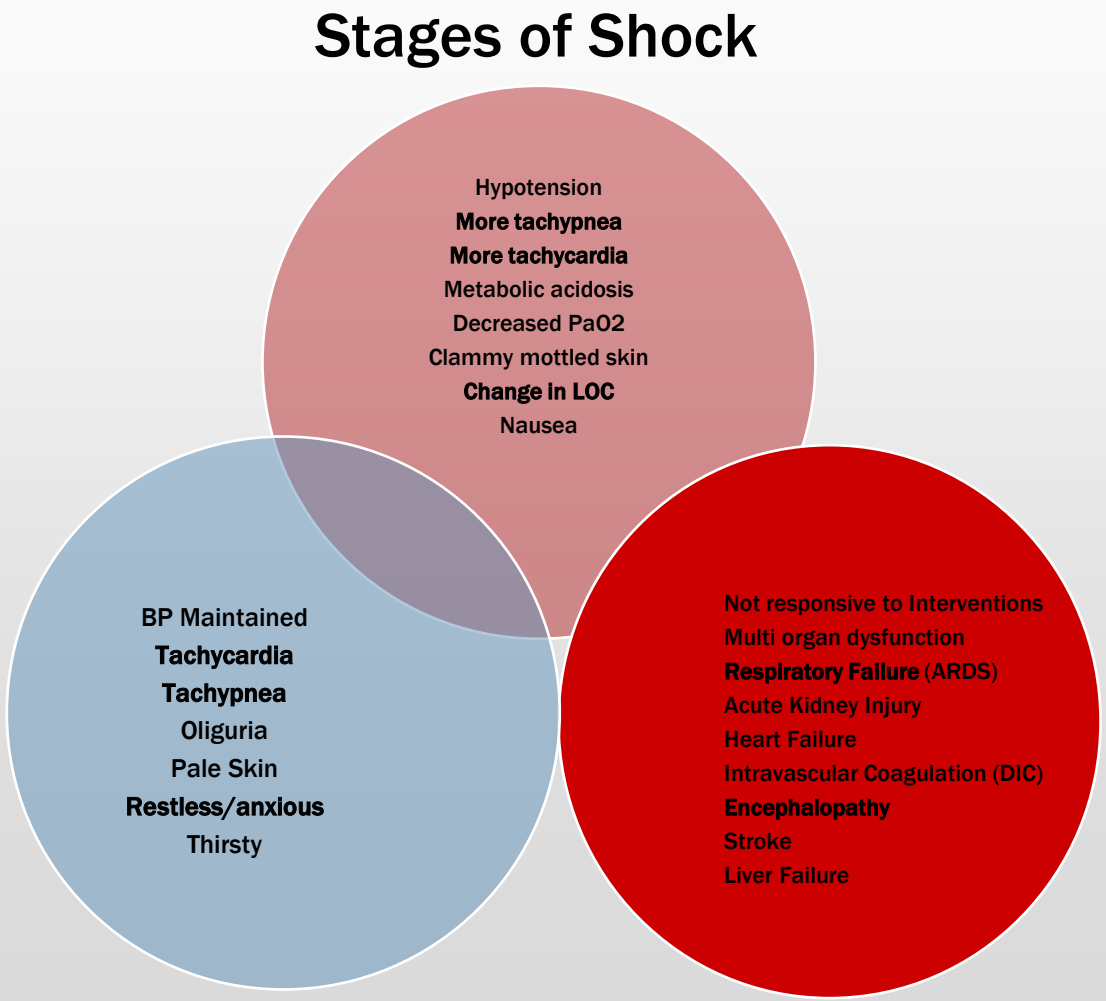
1. **Cardiac Output** (HR x SV)
2. **Total Hemoglobin**
3. **Saturation of arterial oxygen** (SaO2)



- **Patient's 1st compensatory mechanism is to increase HR and venoconstrict in order to increase cardiac output and deliver oxygen to tissues.**
- **To decrease tissue O_2 demand we must control fever, pain, decrease work of breathing, and shivering.**
- **Supplemental of Oxygen should be given if O_2 level is inadequate.**

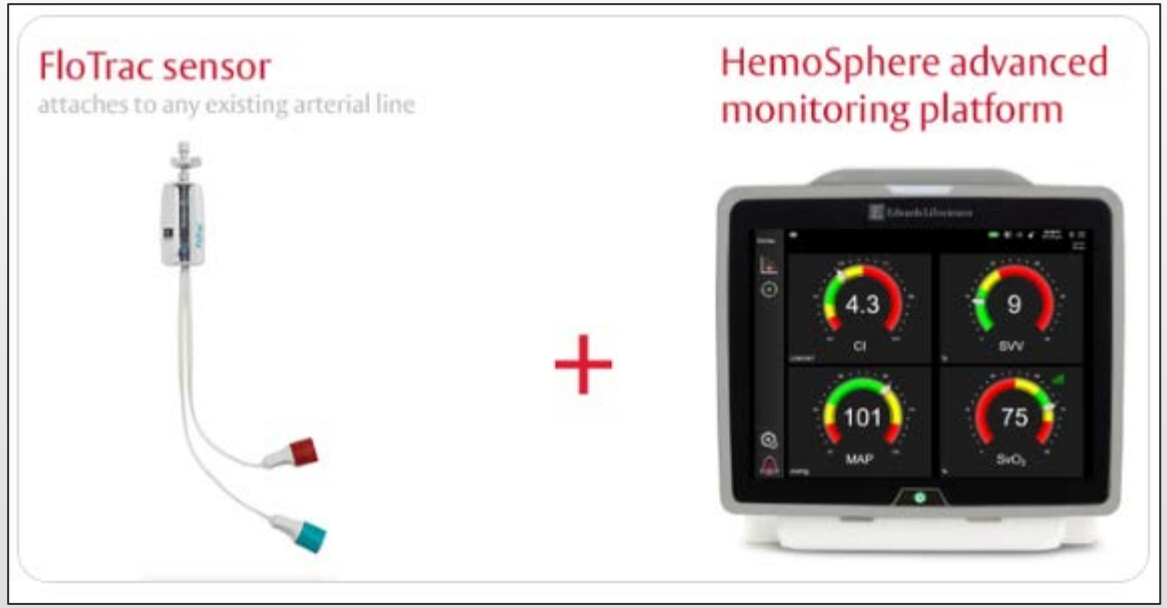
Summary

- Monitoring patient symptoms can provide early warnings of changes before complexity develops into irreversible organ damage.
- Close patient monitoring helps track whether interventions are effective.
- Most monitoring does not require devices.
  - Mental status changes, respiratory rate, and blood pressure are all important parameters to watch.
  - Inspect skin for color, temp and abnormality in circulation, cap refill/ mottling
  - Listen to patient's bowel sounds, lungs and heart



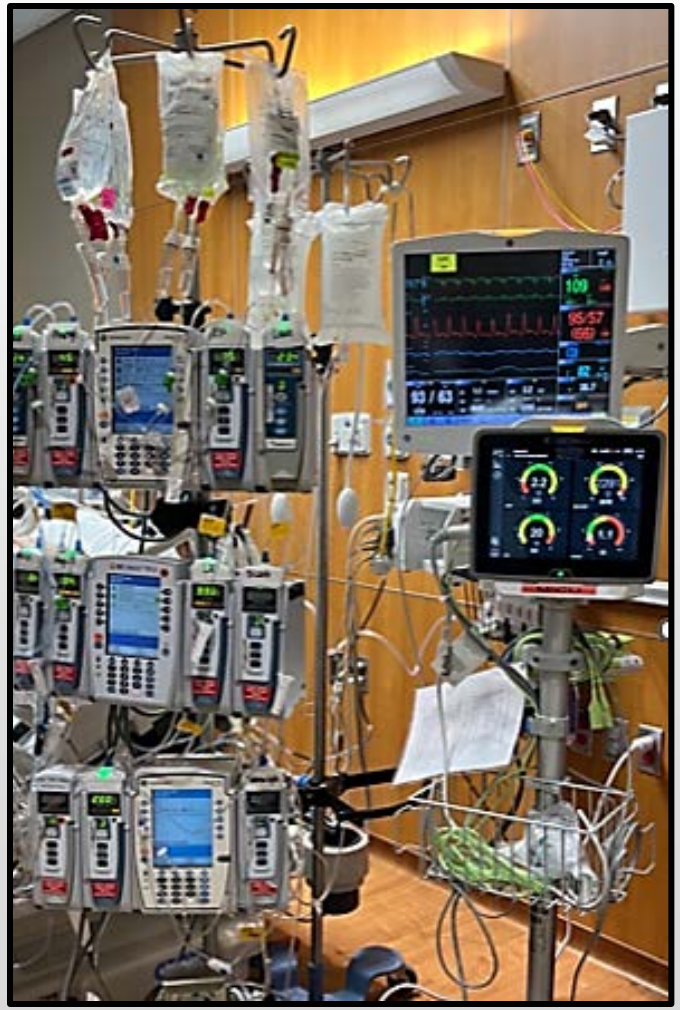
Edwards HemoSphere

- The FloTrac Sensor connects to an existing arterial line and automatically updates every 20 seconds, reflecting rapid physiological changes.
- Parameters Obtained with Sensor
  - Stroke Volume & Stroke Volume Index (SV & SVI)
  - Systemic Vascular Resistance (SVR)
  - Cardiac Output & Cardiac Index (CO & CI)
  - Stroke Volume Variation (SVV)
  - Contractility reflection (Dp/Dt)
  - Arterial Elastance (EaDYN)
  - *Hypotension Predictive Index (HPI)
    - *A hypotension event is defined as MAP<65 mmHg for a duration of at least one minute. (Defined in context of the software)



Hemodynamic Monitoring

Medication Blue: vasopressor Green: inotrope	Receptor: α (blood vessels)	Receptor: ß1 (heart)	Receptor: ß2 (bronchial and vascular smooth muscle)	Receptor V1: (Vascular smooth muscle)	Response Preload: CVP Afterload: SVR Contractility: CO
Norepinephrine (Levophed) 1st line	++++	++			HR ↑ CO ↑ MAP↑ SVR ↑
Vasopressor (Vasopressin) 2nd line				+	HR 0 CO 0/↓ MAP↑ SVR ↑
Phenylephrine (Neosynephrine) 3rd line	++++				HR 0 CO 0/↓ MAP↑ SVR ↑
Angiotensin II (Giapreza) 3rd line				+	HR ↑/0 CO ↑/0 MAP ↑ SVR ↑
Epinephrine (adrenaline)	++++	++++	++	++	HR ↑ CO ↑ MAP ↑ SVR ↑
Dopamine (Inotropin)	++ (<5mcg/kg/min)	++++ (<10mcg/kg/min)	+	+	HR ↑ CO ↑ MAP↑ SVR ↑
Dobutamine (Dobutrex): improves contractility	+	++++	++		HR 0/↑ CO ↑ MAP 0/↑ SVR ↓



Hemodynamic Parameters

Patient #1: BP 106/45 MAP 65 HR 104

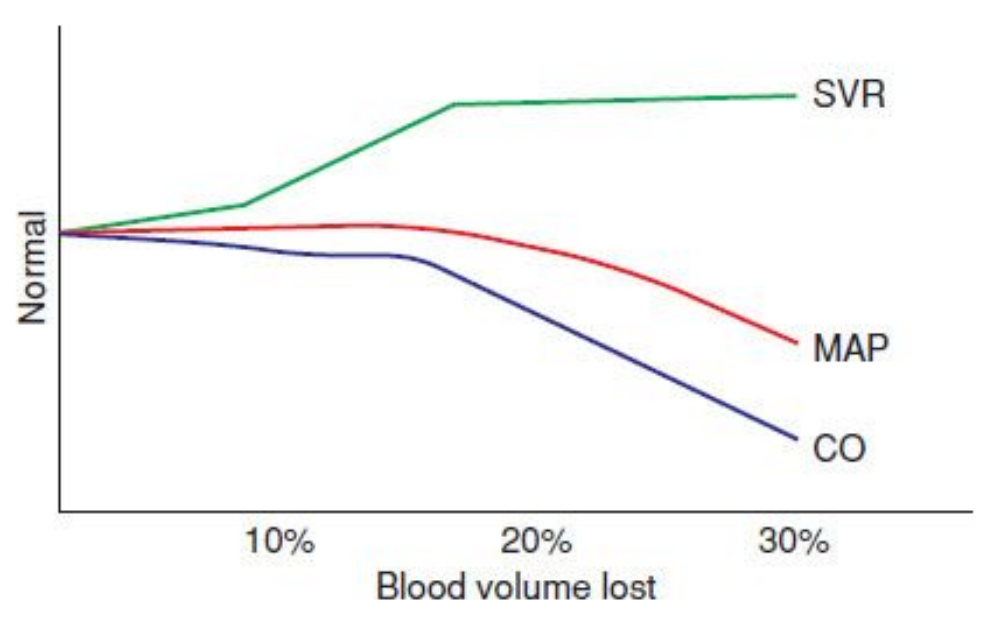
- CI: 1.9, SV 48, SVR 620

Patient #2: BP 104/46 MAP 65 HR 120

- CI: 1.2, SV 50, SVR 2130

Patient #3: BP 105/45 MAP 65 HR 103

- CI: 3.8, SV 68, SVR 1010



HemoSphere Screen

Stroke Volume

- Amount of blood pumped by left ventricle during each beat (systolic contraction) 60-100
- Changes by 10% if pt is fluid responsive

Systemic Vascular Resistance

- Measures total peripheral resistance, resistance left ventricle overcomes (800-1200 dynes)
- Medications that vasoconstrict will increase SVR

Cardiac Output Cardiac Index

- CO= SV x HR (4-8 L/min)
- CI is cardiac output divided by body surface area (2.5-4L/min/m²)

SVV

- Stroke volume variation: arterial pressure response to volume expansion in hypotensive ventilated patients, measure in %. Goal is value less than 13%

dP/dt

- Indicator of left ventricular global contractility performance not influenced by afterload (>1200mmHg/s)

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