Evidence-based Approaches to the Management of Cardiogenic and Septic Shock: Clinical Updates

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Disclosure Statement

- No relevant financial or nonfinancial relationships to disclose.

- Case studies discussed are fictitious and are not meant to represent any patient(s) encountered in clinical practice.

Objective

- Learners will self-report an increase in knowledge about cardiogenic and septic shock management.

If you could describe Septic Shock in 3 words, how would you describe it?

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Types of Shock

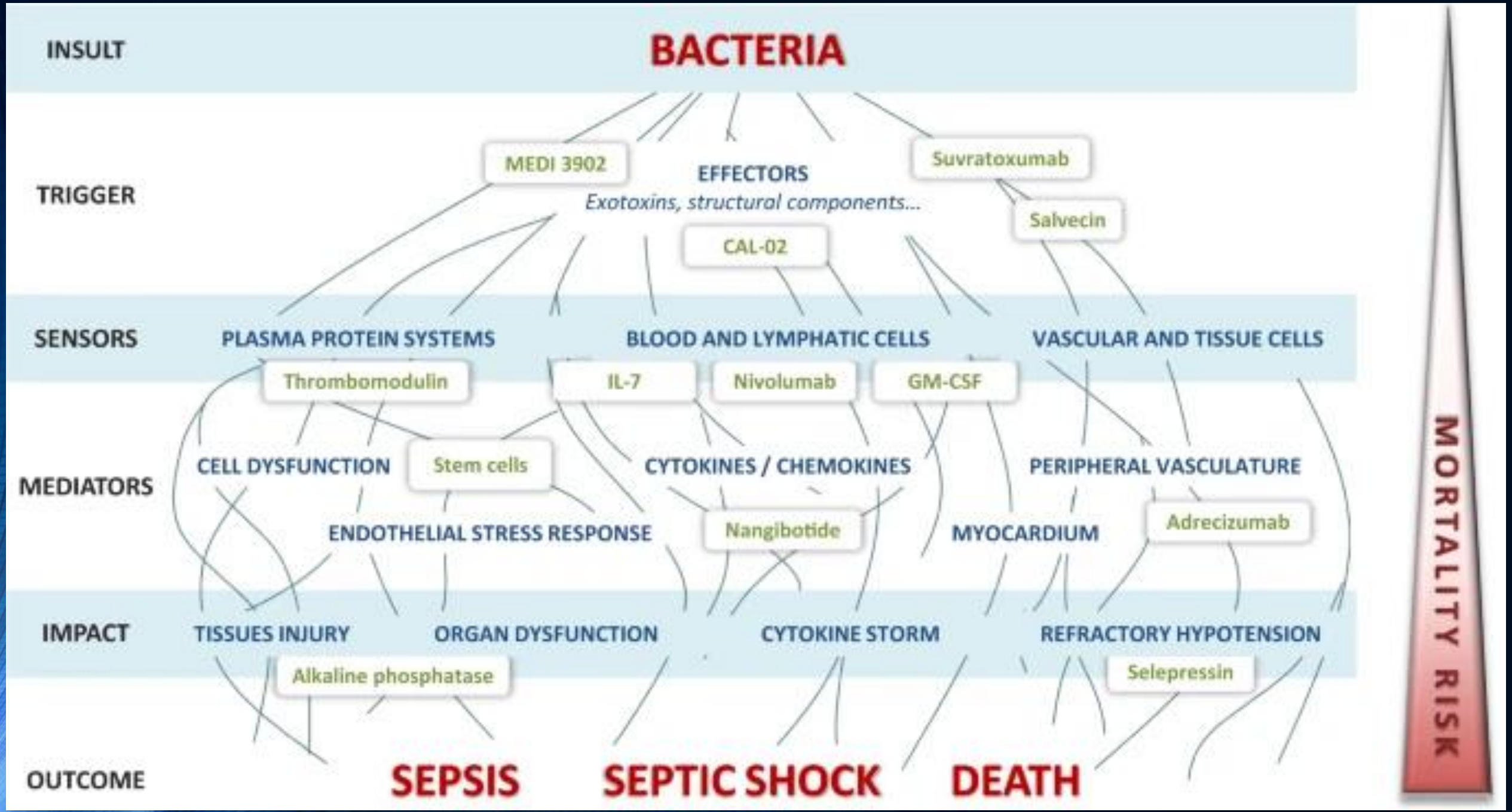
- Distributive (septic shock, systemic inflammatory response syndrome)
- Cardiogenic (myocardial infarction)
- Hypovolemic (fluid losses)
- Obstructive (pulmonary embolism, pulmonary hypertension)

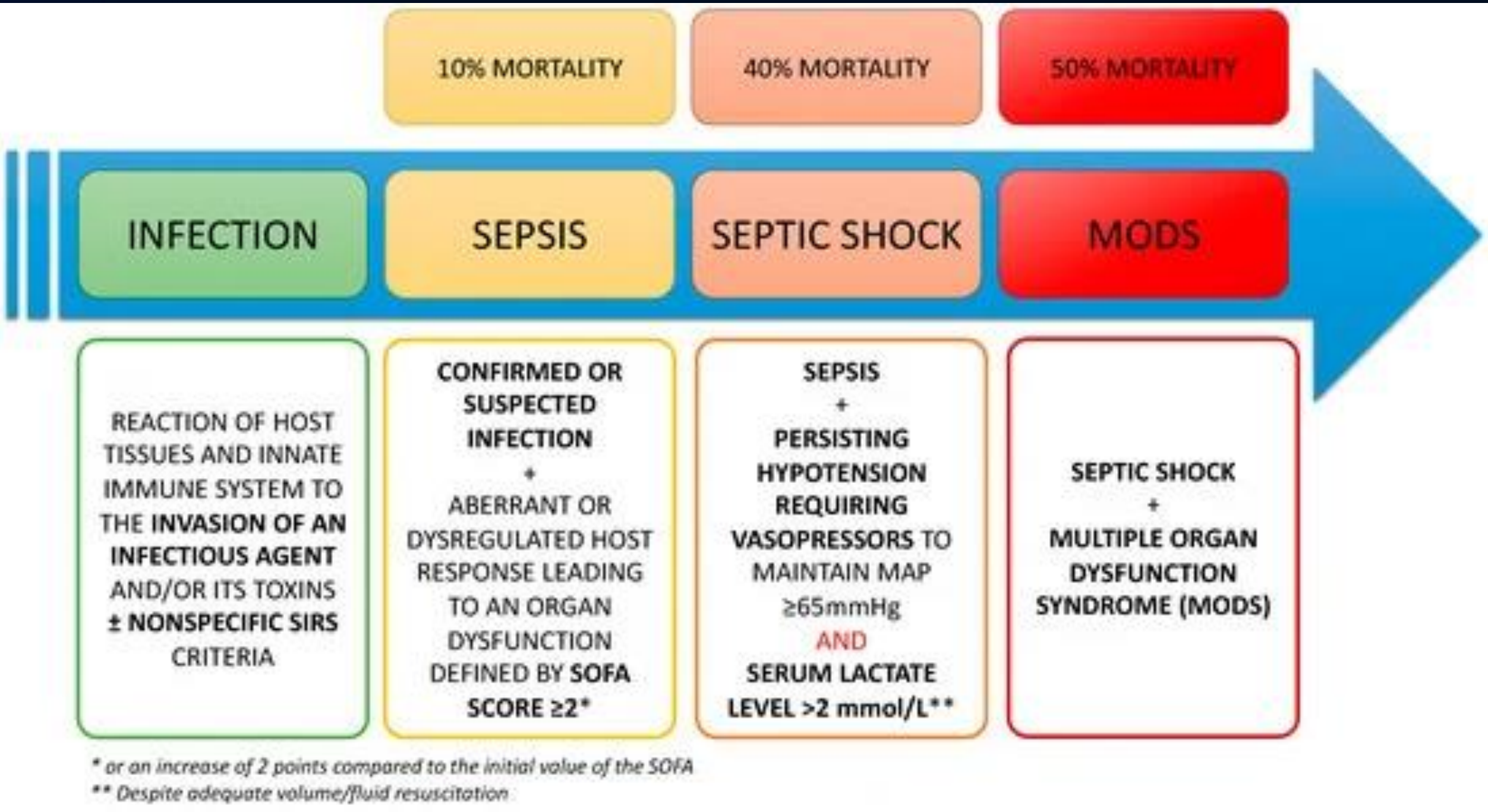
Significance of Sepsis and Septic Shock

- Sepsis is one of the leading causes of mortality worldwide
- Each year, more than 1.7 million U.S. adults receive hospital care for sepsis, and more than 270,000 American adults die of sepsis
- One out of every three patients who die in a hospital had sepsis
- Mortality increases 4-9% for every hour that treatment is delayed
- Primary reason for hospital readmission
- 30-50% greater risk of death for those who are diagnosed with Septic Shock
- Costs to treat sepsis total $62 billion annually in the U.S.

What is Sepsis?

- Sepsis is a clinical syndrome characterized by a dysregulated host response to infection.
  - There is a continuum of severity ranging from sepsis to septic shock.
  - Wide-ranging and dependent upon the population studied, mortality has been estimated to be ≥10 percent and ≥40 percent when shock is present


INSULT
BACTERIA
TRIGGER
MEDI 3902
EFFECTORS
Exotoxins, structural components...
Suvratoxumab
Salvecin
CAL-02
SENSORS
PLASMA PROTEIN SYSTEMS
BLOOD AND LYMPHATIC CELLS
VASCULAR AND TISSUE CELLS
Thrombomodulin
IL-7
Nivolumab
GM-CSF
MEDIATORS
CELL DYSFUNCTION
Stem cells
CYTOKINES / CHEMOKINES
PERIPHERAL VASCULATURE
ENDOTHELIAL STRESS RESPONSE
Nangibotide
MYOCARDIUM
Adrecizumab
IMPACT
TISSUES INJURY
ORGAN DYSFUNCTION
CYTOKINE STORM
REFRACTORY HYPOTENSION
Alkaline phosphatase
Selepressin
OUTCOME
SEPSIS
SEPTIC SHOCK
DEATH
MORTALITY RISK


10% MORTALITY
40% MORTALITY
50% MORTALITY
INFECTION
SEPSIS
SEPTIC SHOCK
MODS
REACTION OF HOST TISSUES AND INNATE IMMUNE SYSTEM TO THE **INVASION OF AN INFECTIOUS AGENT** AND/OR ITS TOXINS **± NONSPECIFIC SIRS** CRITERIA
CONFIRMED OR SUSPECTED INFECTION + ABERRANT OR DYSREGULATED HOST RESPONSE LEADING TO AN ORGAN DYSFUNCTION DEFINED BY **SOFA SCORE ≥2***
SEPSIS + **PERSISTING HYPOTENSION REQUIRING VASOPRESSORS** TO MAINTAIN MAP ≥65mmHg AND **SERUM LACTATE LEVEL >2 mmol/L****
SEPTIC SHOCK + **MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)**
* or an increase of 2 points compared to the initial value of the SOFA
** Despite adequate volume/fluid resuscitation

World Health Organization

Who is at risk of sepsis

Anyone with an infection can develop sepsis but some are more at risk than others

PREGNANT WOMEN

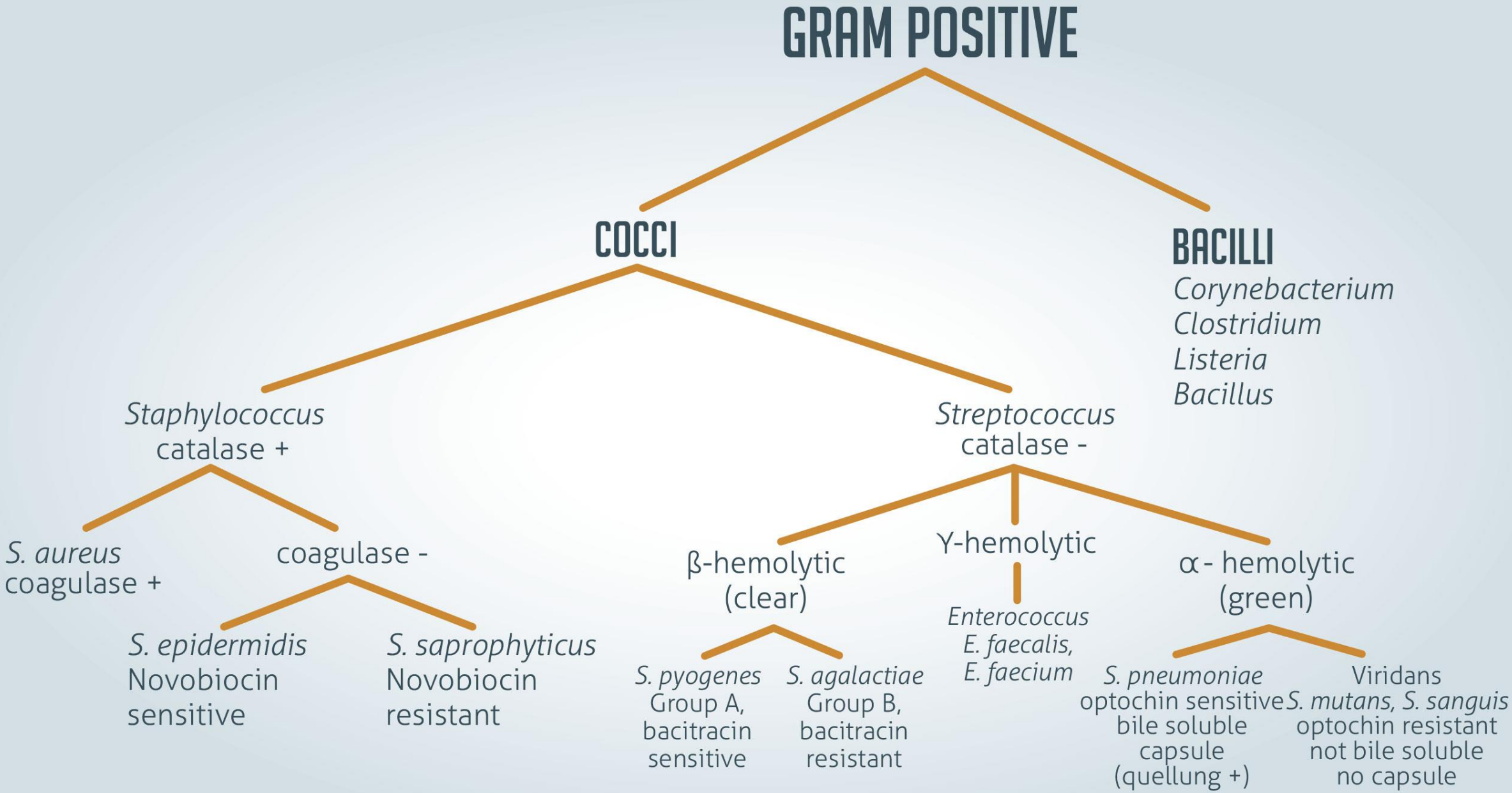
NEONATES

THE ELDERLY

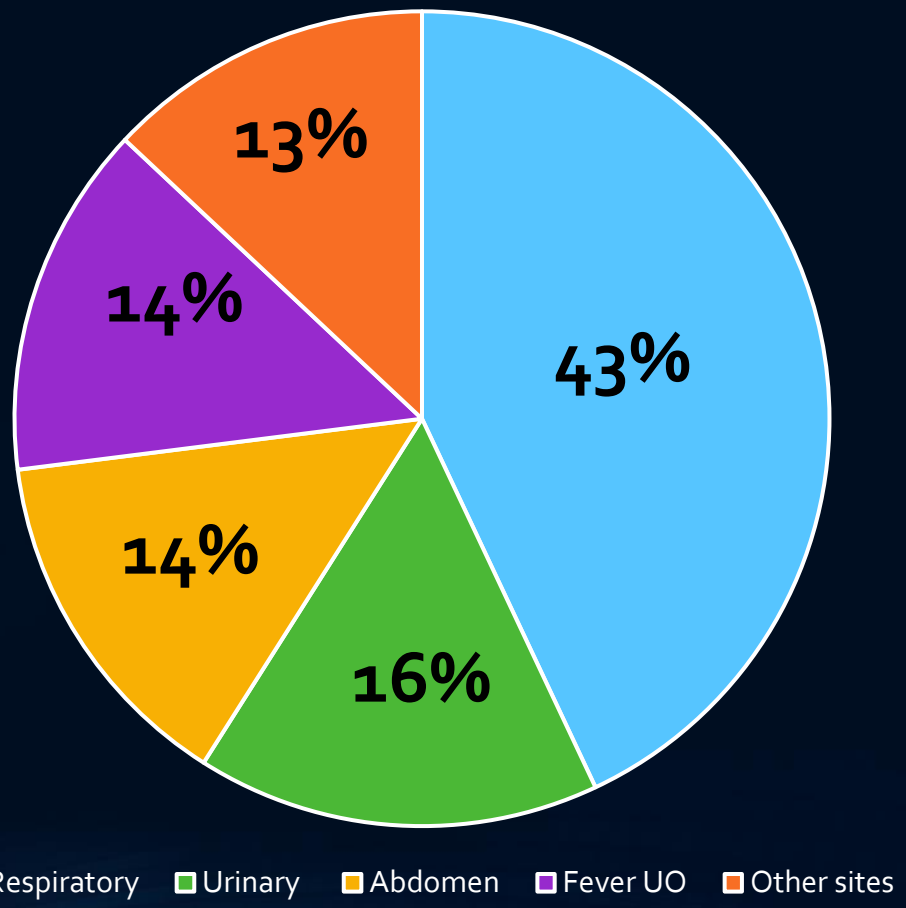
THE IMMUNOSUPPRESSED

PATIENTS WITH CHRONIC DISEASES

HOSPITALIZED PATIENTS


GRAM POSITIVE
COCCI
BACILLI
Corynebacterium
Clostridium
Listeria
Bacillus
Staphylococcus
catalase +
Streptococcus
catalase -
S. aureus
coagulase +
coagulase -
S. epidermidis
Novobiocin
sensitive
S. saprophyticus
Novobiocin
resistant
β-hemolytic
(clear)
Y-hemolytic
α - hemolytic
(green)
S. pyogenes
Group A,
bacitracin
sensitive
S. agalactiae
Group B,
bacitracin
resistant
Enterococcus
E. faecalis,
E. faecium
S. pneumoniae
optochin sensitive
bile soluble
capsule
(quellung +)
Viridans
S. mutans, S. sanguis
optochin resistant
not bile soluble
no capsule

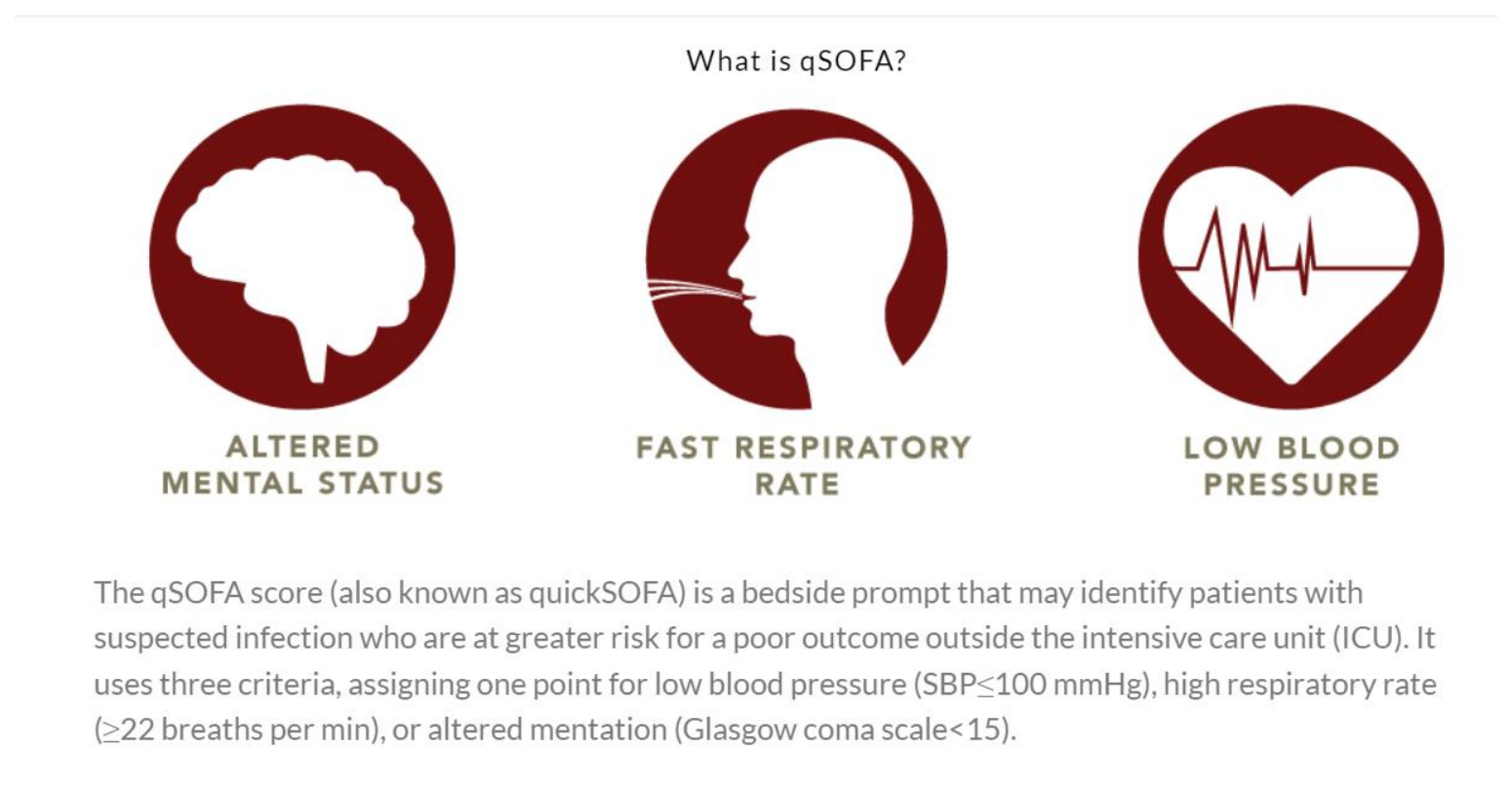
Proportion of Infectious Sites



Septic Shock Clinical Manifestations

- Hypotension
- Tachycardia
- Oliguria
- Abnormal mental status
- Tachypnea
- Cool, clammy, cyanotic skin
- Metabolic acidosis
- Hyperlactatemia

Organ Failure Assessment Tool: qSOFA Score



- qSOFA :: quick Sepsis Related Organ Failure Assessment

What's New?

- The Centers for Medicare & Medicaid Services make SEP-1 sepsis care bundle a pay-for-performance measure.
- Social Determinants of Health and Health Equity
- Society of Critical Care Medicine – Pediatric
  - Initial-Resuscitation-Algorithm-for-Children.pdf (sccm.org)
- Septic Shock Management, updated clinical research

What percentage of your work week do you initiate or manage a Septic Shock protocol?

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OUR EVIDENCE SHOWS HOW TO IMPROVE SEPSIS CARE ACROSS THE DELIVERY SYSTEM

More rapid sepsis identification and treatment saves lives.

Post-ICU clinics and peer support can help patients and families recover from sepsis.

More intensive sepsis treatment in intensive care units (ICUs) can save lives without increased costs.

Better follow-up care is needed to improve outcomes and prevent rehospitalizations.

Requirements for hospitals to adopt evidence-based sepsis response plans (such as New York's "Rory's Regulations") have contributed to reduced deaths from sepsis, lengths of stay, and average time to treatment.

The Sepsis Kentucky Consortium

Sepsis in Kentucky - KYHA

Evidence-based Management of Septic Shock- Antimicrobials

- Empirical antimicrobial treatment (1hr versus 3h)
  - Multidrug antimicrobial regimens with a wide spectrum of activity (e.g., carbapenems and anti-Gram-negative antimicrobials with dual coverage)
    - 1 hour highly suspected and shock detectable
    - 3 hours if concern for infection

Fluids

- Fluid (crystalloids) replacement (according to fluid responsiveness)
- Fluid boluses are the preferred method of administration
- Infusion of intravenous fluids (30 mL/kg)
  - Start within the first hour and complete within the first three hours of presentation
  - Repeat until blood pressure and tissue perfusion are improved (watch for pulmonary edema)

Vasopressor Management

- Vasoactive agents (e.g., norepinephrine) to maintain mean arterial pressure > 65 mmHg
- Norepinephrine remains the first-choice vasopressor in patients with septic shock
  - Vasopressin and epinephrine represent second-line vasopressor therapies and dopamine should be avoided
  - Refractory shock, vasopressin (rather than epinephrine) should be combined with NE to reach an acceptable level of pressure control

Peptide Precursors

- Procalcitonin (PCT) is widely used for differentiating bacterial vs. non-bacterial infections or other inflammatory conditions
- Recently, Presepsin (PSP), a soluble N-terminal fragment of the cluster of differentiation marker protein 14 (CD14), has been proposed as an alternative biomarker to PCT

Additional Measures

- If mechanical ventilation is indicated, keep tidal volume ~6 mL/kg.
- LMWH rather than UFH should be used to prevent VTE
- Glycemic control is recommended with insulin
- Hydrocortisone may be considered in patients with vasopressor-resistant, inadequate MAP.
- The efficacy of other treatments (e.g., proton-pump inhibitors, sodium bicarbonate, etc.) is largely debated, and used on a case-to-case basis.

Severe Sepsis and Septic Shock Management Bundle (SEP-1)

- The first step of the Severe Sepsis and Septic Shock Management Bundle (SEP-1) calls for:
  - Lactate measurements (every 6 hours)
  - Blood cultures
  - Broad-spectrum antibiotics administration within three hours of sepsis diagnosis.
  - The Centers for Medicare & Medicaid Services (CMS) has required hospitals to report on SEP-1 compliance since the 2017 fiscal year.
  - The inclusion of SEP-1 in CMS' Hospital Value-Based Purchasing Program makes the bundle a pay-for-performance measure.

Sepsis Bundle Management

- Airway, Correct hypoxemia, establish appropriate vascular access
- Laboratory studies (complete blood count, electrolyte panel, liver function, coagulation studies, D-dimer)
- Serum lactate
- Arterial blood gases
- Blood cultures (aerobic and anaerobic) from two distinct venipuncture sites and from all indwelling vascular access devices; blood cultures before the initiation of antibiotics
- Cultures from easily accessible sites (i.e., sputum, urine)
- Imaging of suspected sources

Septic Shock Bundle (continued)

- 30 mL/kg of IV fluids within three hours
- Vasopressors within five hours for persistent hypotension
- Repeat volume assessment within six hours

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Original Article

Compliance with SEP-1 guidelines is associated with improved outcomes for septic shock but not for severe sepsis

Shelly N.B. Sloan[1], Nate Rodriguez[1], Thomas Seward[1], Lucy Sare[1], Lukas Moore[1], Greg Stahl[2], Kerry Johnson[3], Scott Goade[4], Robert Arnce[1,*]

Critical Care Explorations

Society of Critical Care Medicine
The Intensive Care Professionals

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Social Determinants of Health Associated With the Development of Sepsis in Adults: A Scoping Review

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SEPTEMBER IS
SEPSIS AWARENESS
MONTH

The Future of Sepsis and Septic Shock

- Emerging antibiotics against most frequent pathogens
- Concern for bacterial resistance which requires new therapeutic approaches
- Monoclonal antibodies (mAbs) targeting virulence factors of causative bacteria - either preventive or as adjunctive to antibiotic therapy

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Thank you!

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Cardiogenic Shock

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Purpose

- To improve the morbidity and mortality in patients who present with or develop cardiogenic shock.
- The goal of therapy is to quickly identify the patients, initiate the cardiogenic shock algorithm, and provide the best patient-specific care based on the patient's condition.

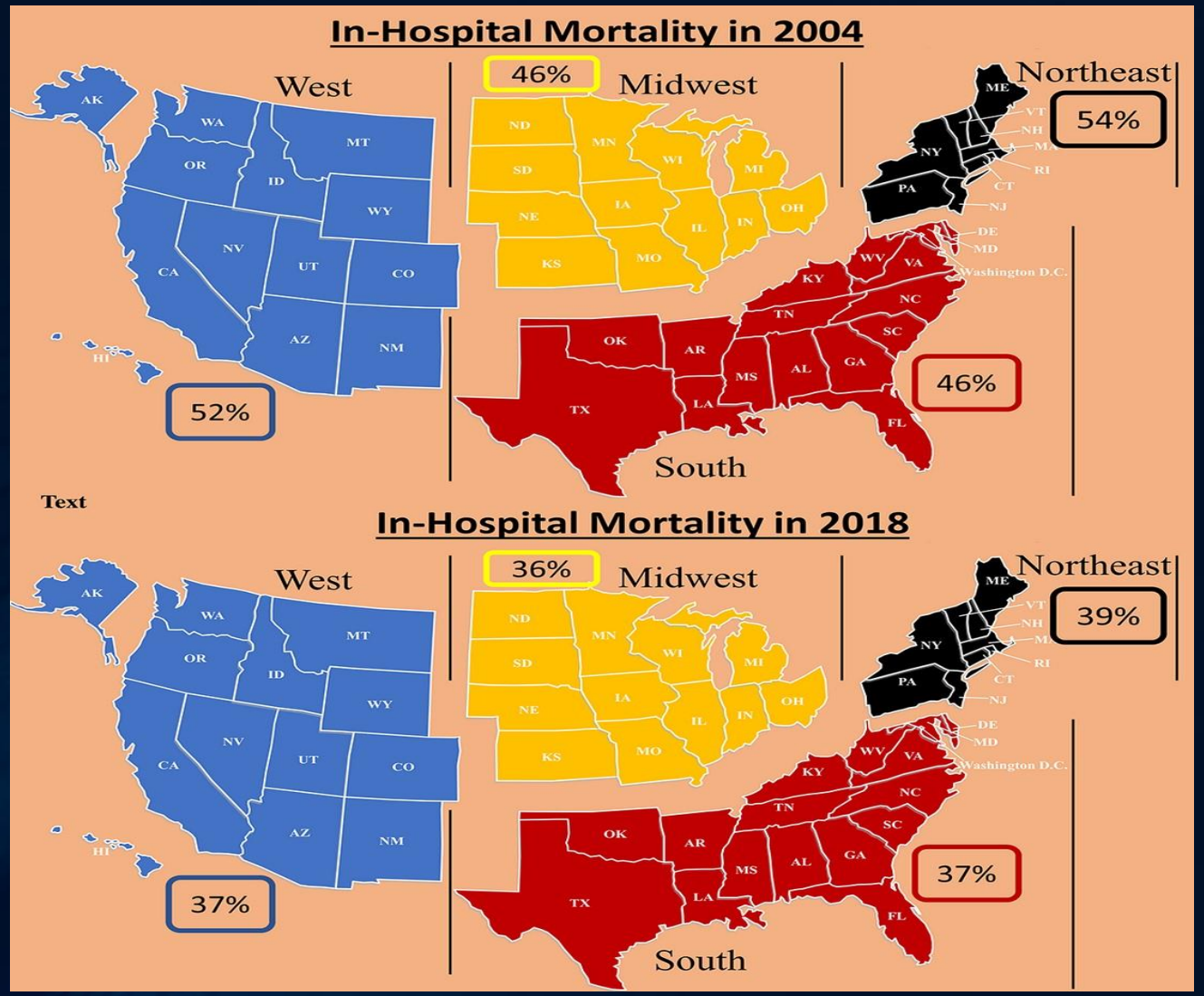
Statistics

- DESPITE ADVANCES IN MEDICATIONS AND MECHANICAL SUPPORT DEVICES, THE MORTALITY FOR CARDIOGENIC SHOCK REMAINS AT 30%.
- IMPLEMENTING A MULTIDISCIPLINARY CARDIOGENIC SHOCK TEAM HAS BEEN SHOWN TO DECREASE MORTALITY.

Cardiogenic Shock (CGS) Statistics

- The most common cause of cardiogenic shock is secondary to acute myocardial infarction (AMI) 60-80%
- However, there is argument that non-AMI cardiogenic shock is under diagnosed and could account for 70% of total cardiogenic shock cases.

CGS Mortality



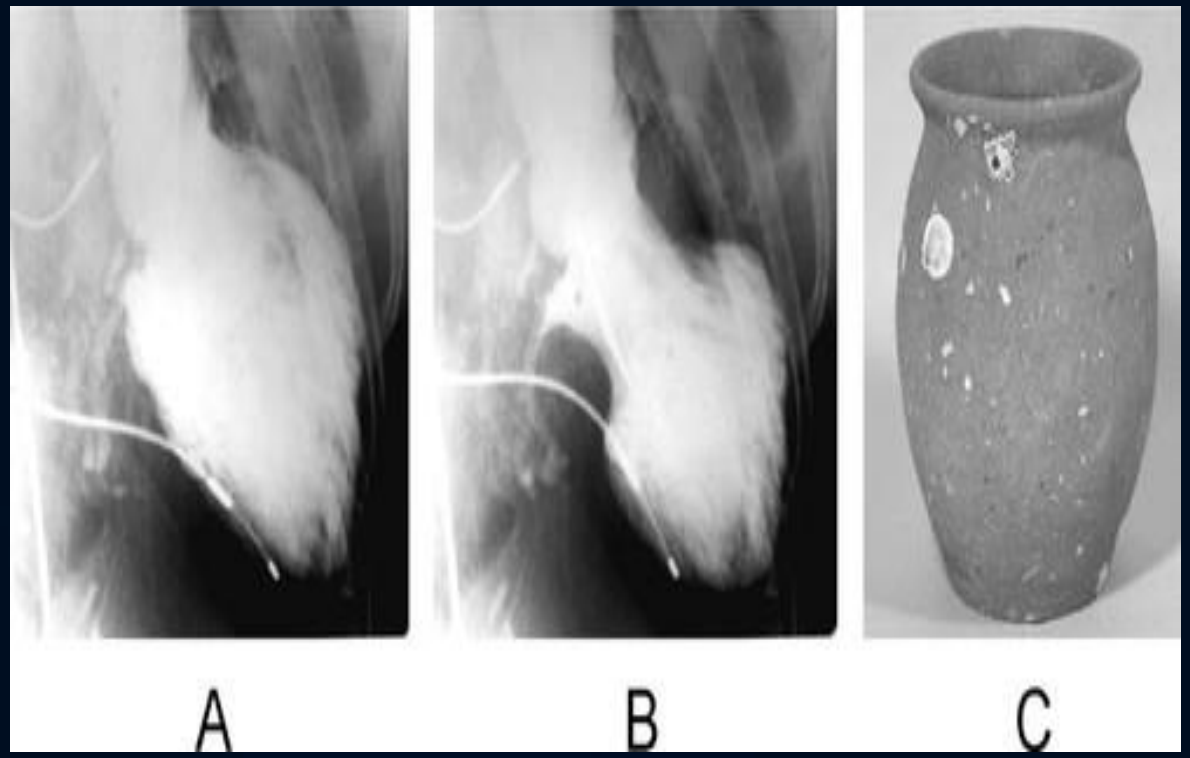
Source: (Osman et al., 2021)

Detroit Cardiogenic Shock Initiative

- STARTED BY HENRY FORD HEALTH IN 2016
- INCREASED SURVIVAL RATE TO 72% THROUGH THE USE OF CARDIOGENIC SHOCK PROTOCOLS FOCUSING ON EARLY MECHANICAL CIRCULATORY SUPPORT
- BEGAN WITH AMI PATIENTS AND PROGRESSED TO NON-AMI PATIENTS
- WHEEL/SPOKE/HUB MODEL

Non-AMI Cardiogenic Shock

- Free Wall Rupture
- Acute severe mitral regurgitation
- Right Heart Failure
- Takotsubo Cardiomyopathy
- Postpartum Cardiomyopathy (10.3 patients per 10k live births)
- Massive Pulmonary Embolism



Cardiogenic Shock

- DOOR TO SUPPORT
- DOOR TO MECHANICAL SUPPORT TIME <1.25 HOURS – SURVIVAL 66%
- DOOR TO MECHANICAL SUPPORT TIME 1.25-4.25 HOURS- SURVIVAL 37%
- DOOR TO MECHANICAL SUPPORT TIME >4.25 HOURS- SURVIVAL 26%

For every hour of delay in escalation of care is a 10% increase in mortality

Cardiogenic Shock Phenotypes

		Volume Status	
		Wet	Dry
Peripheral Circulation	Cold	Classic Cardiogenic Shock (↓CI; ↑SVRI; ↑PCWP)	Euvolemic Cardiogenic Shock (↓CI; ↑SVRI; ↔PCWP)
	Warm	Vasodilatory Cardiogenic Shock or Mixed Shock (↓CI; ↓/↔SVRI; ↑PCWP)	Vasodilatory Shock (Not Cardiogenic Shock) (↑CI; ↓SVRI; ↓PCWP)

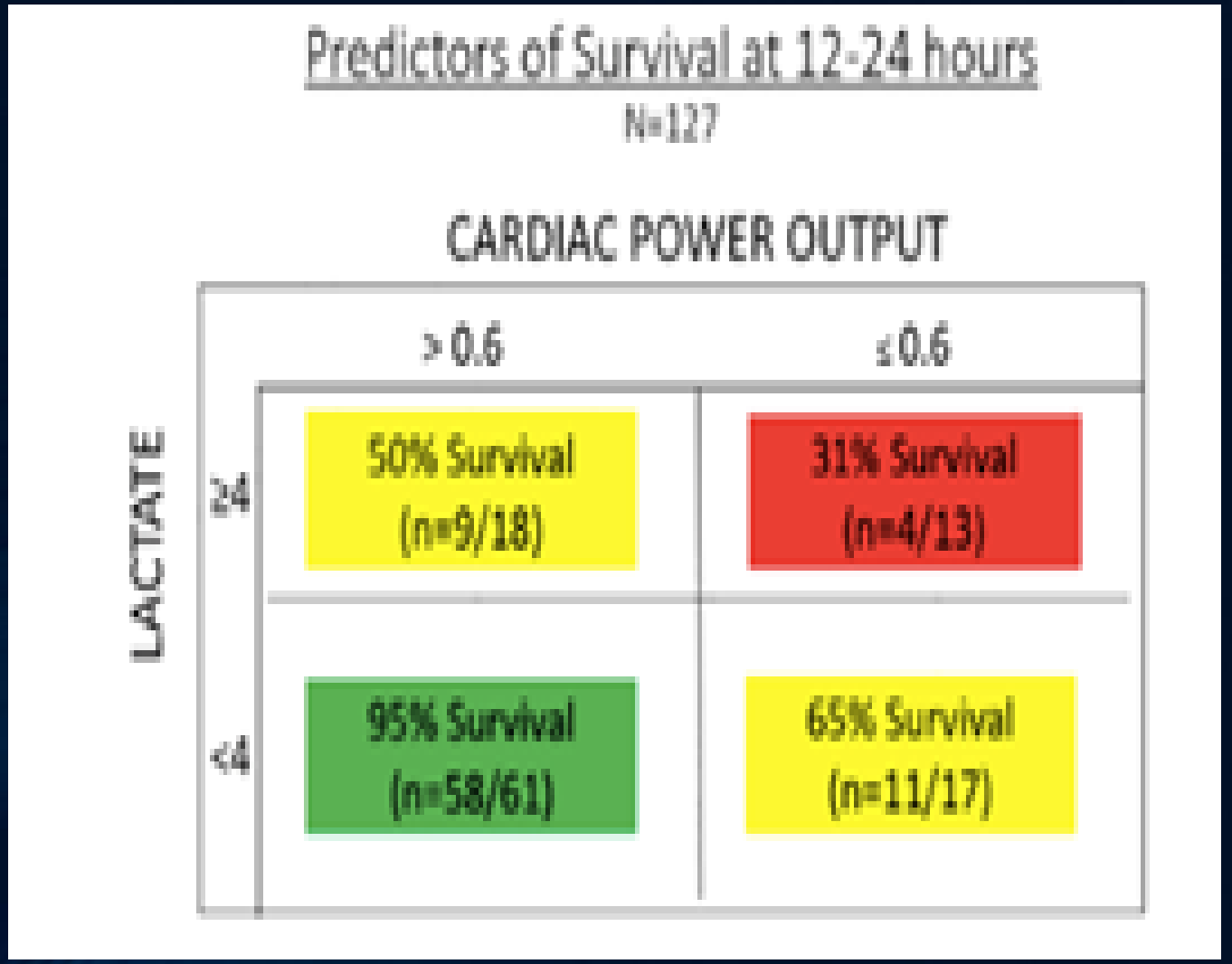
Cardiac Power (CP)

$$CP = \frac{\text{MEAN ARTERIAL PRESSURE (MAP) X CARDIAC OUTPUT (CO}}{451}$$

CP IS THE STRONGEST HEMODYNAMIC CORRELATE TO MORTALITY IN CARDIOGENIC SHOCK

NORMAL RANGE >.6

Cardiac Power/Lactate



- How accurately is the average provider able to diagnose cardiogenic shock on assessment?

PAPi- pulmonary artery pulsatility index

$$PAPI = \frac{\text{PA Systolic Pressure} - \text{PA Diastolic Pressure}}{CVP}$$

Normal Range= >0.9

PAPI < 0.9 100% sensitivity and 98% specificity for predicting hospital mortality and requirement of RV support

Diagnosing Cardiogenic Shock

- How accurately is a provider able to diagnose Cardiogenic Shock on physical assessment alone?

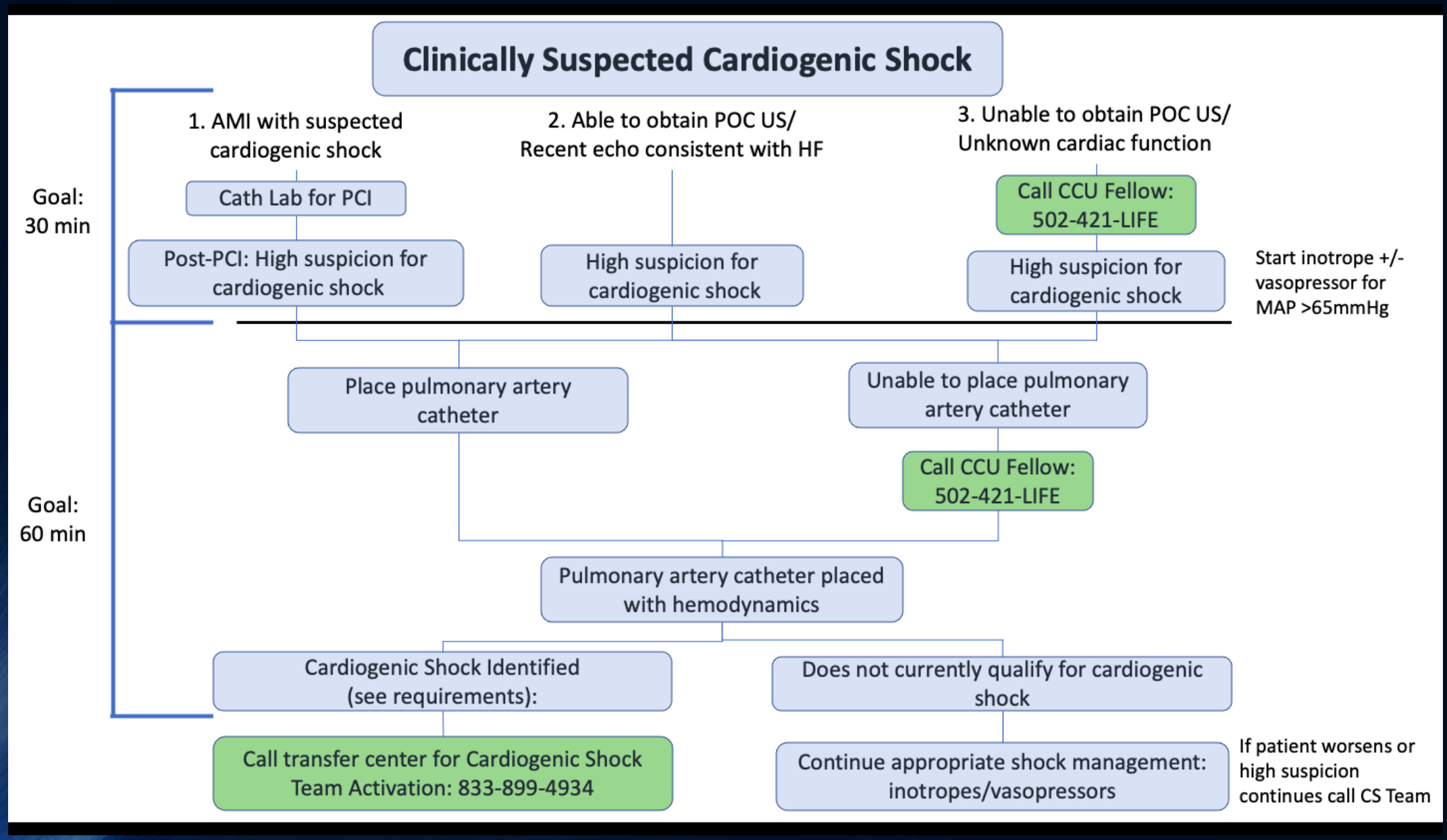
Clinical Diagnosis of Cardiogenic Shock

- SBP < 90 X 30 MIN
- MAP < 60 X 30 MIN
- VASOPRESSOR USE TO ACHIEVE SBP > 90 OR MAP > 60
- LACTIC ACID > 2
- SIGNS OF ORGAN MALPERFUSION- ALTERED MENTAL STATUS, COLD EXTREMITIES, DECREASED URINE OUTPUT

- DEPENDING ON CARDIOGENIC SHOCK PHENOTYPE, PATIENTS CAN BE IN THE EARLY STAGES OF SHOCK AND HAVE A NORMAL BLOOD PRESSURE

CGS relative exclusion criteria

- Anoxic brain injury
- Unwitnessed out of hospital cardiac arrest or cardiac arrest with ROSC>30 min and no neurological recovery
- Distributive or hypovolemic shock
- Active uncontrolled bleeding
- Immediately post cardiac surgery
- LVAD patient


Clinically Suspected Cardiogenic Shock
1. AMI with suspected cardiogenic shock
2. Able to obtain POC US/ Recent echo consistent with HF
3. Unable to obtain POC US/ Unknown cardiac function
Goal: 30 min
Cath Lab for PCI
Call CCU Fellow: 502-421-LIFE
Post-PCI: High suspicion for cardiogenic shock
High suspicion for cardiogenic shock
High suspicion for cardiogenic shock
Start inotrope +/- vasopressor for MAP >65mmHg
Place pulmonary artery catheter
Unable to place pulmonary artery catheter
Call CCU Fellow: 502-421-LIFE
Goal: 60 min
Pulmonary artery catheter placed with hemodynamics
Cardiogenic Shock Identified (see requirements):
Does not currently qualify for cardiogenic shock
Call transfer center for Cardiogenic Shock Team Activation: 833-899-4934
Continue appropriate shock management: inotropes/vasopressors
If patient worsens or high suspicion continues call CS Team

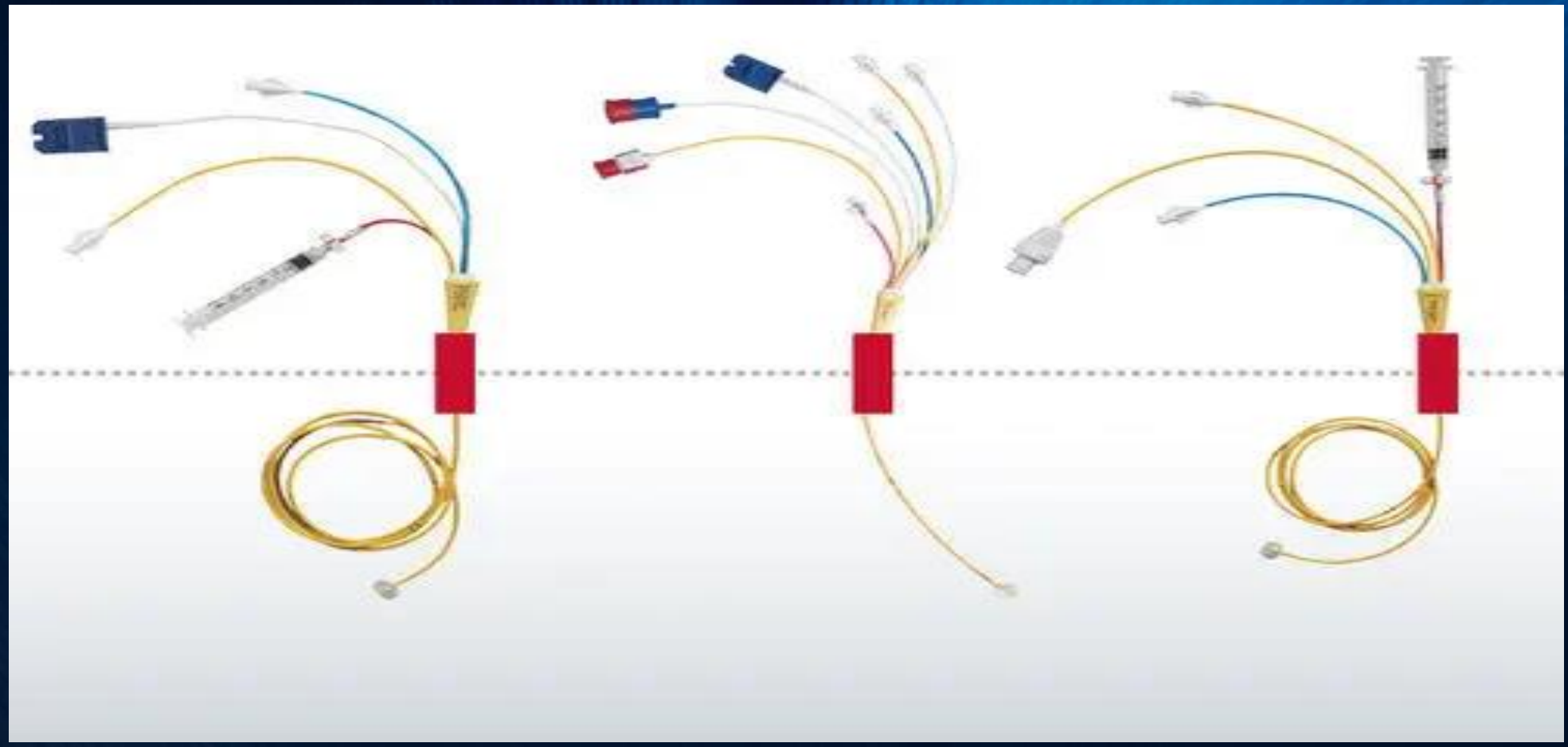
Cardiogenic Shock Team

- Heart Failure Cardiologists
- CT Surgery
- Interventional Cardiology
- CVICU Intensivists

Identification phase- echo

GOAL TIME <90 MINUTES
CGS SUSPECTED, ECHO OBTAINED
-CI ESTIMATION
-LV SYSTOLIC FUNCTION
-RV SYSTOLIC FUNCTION USING TAPSE OR RV STRAIN
-RVSP= PASP ESTIMATION
-IMAGING OF IVC= RAP ESTIMATION
-VALVULAR DEFECTS, VSD, WALL RUPTURES
-RV OUTFLOW TRACT FOR PE

If echo is consistent with CGS, place Swan Ganz Catheter- goal < 90 minutes



To swan or not to swan?

- The ESCAPE trial in 2006 did not show a mortality benefit in using SGC with shock patients and resulted in decreased use of SGC in clinical practice. However, this trial did not enroll CGS patients.
- Swan-Ganz Catheters are required to determine hemodynamics and CS phenotype and aid in deciding which device is best for the patient.
- Meta-analysis in 2017 showed 20% decrease in mortality in CGS patients with a SGC and more likely to escalate to mechanical circulatory assist devices.

Assessment

- AMS
- EKG CHANGES/ARRHYTHMIAS
- HR <50, > 120
- URINE OUTPUT < 0.5 ML/KG/HOUR
- HYPOTENSION
- CI < 2.2
- RISE IN LACTATE OR CREATININE

Diagnosing CGS with SGC

- CI < 1.8 OR CI < 2.2 REQUIRING INOTROPES OR PRESSORS
- AND ORGAN DYSFUNCTION- LACTIC ACIDOSIS, OLIGURIA, AMS, DYSPNEA, HYPOTENSION

Management phase

- TRANSFER CENTER CALL
- PHYSICIAN CONFERENCE
- DECISION

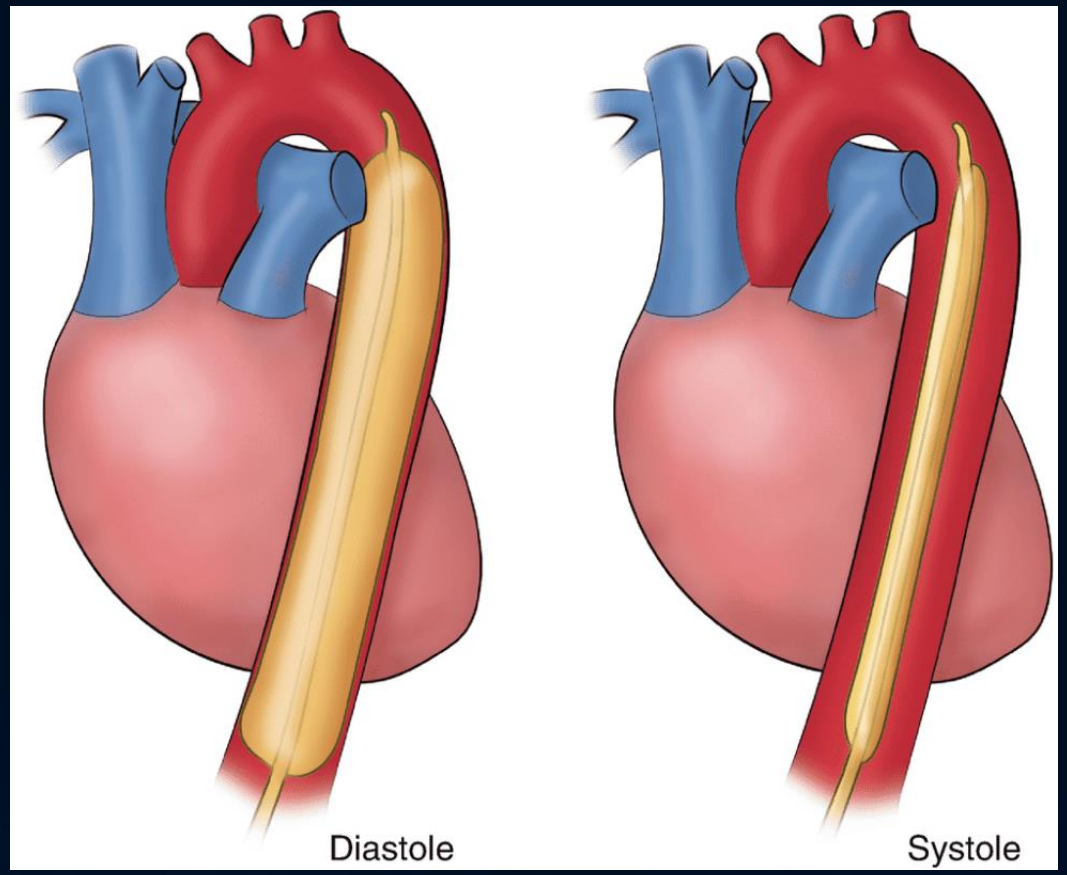
Inotropes/vasopressors

Table 2: Inotrope/Vasopressor Use in Cardiogenic Shock	
Inotrope/Vasopressor	**Max dosing before escalation**
Norepinephrine	0.1 mcg/kg/min
Dobutamine	5 mcg/kg/min
Milrinone	0.25 mcg/kg/min
Epinephrine	0.06 mcg/kg/min
Vasopressin (only in RHF or vasoplegic CS)	0.04 units/min

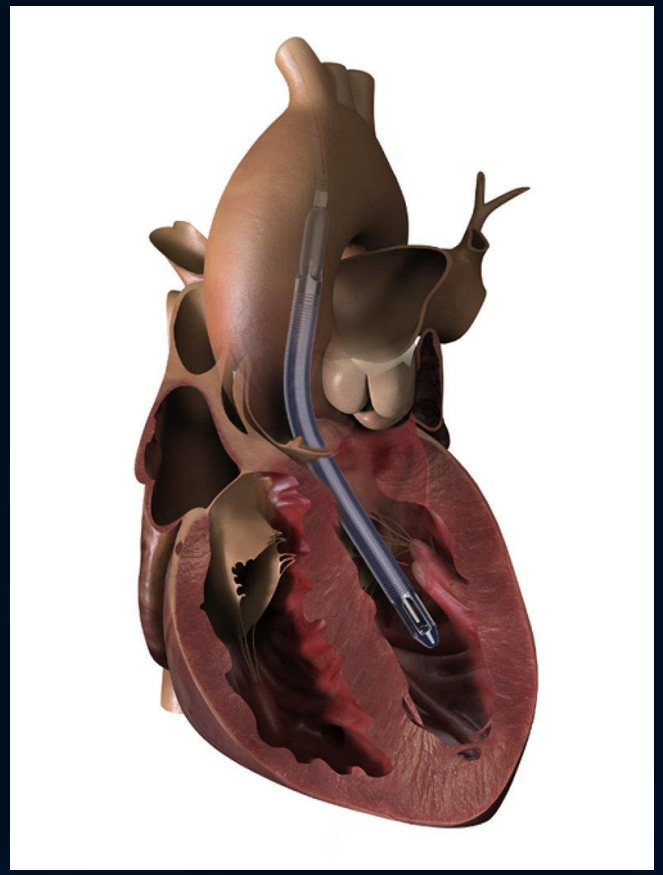
Types of MCS devices

-IMPELLA CP AND 5.5
-IABP (FEM + AXILLARY)
-ECMO (VA)

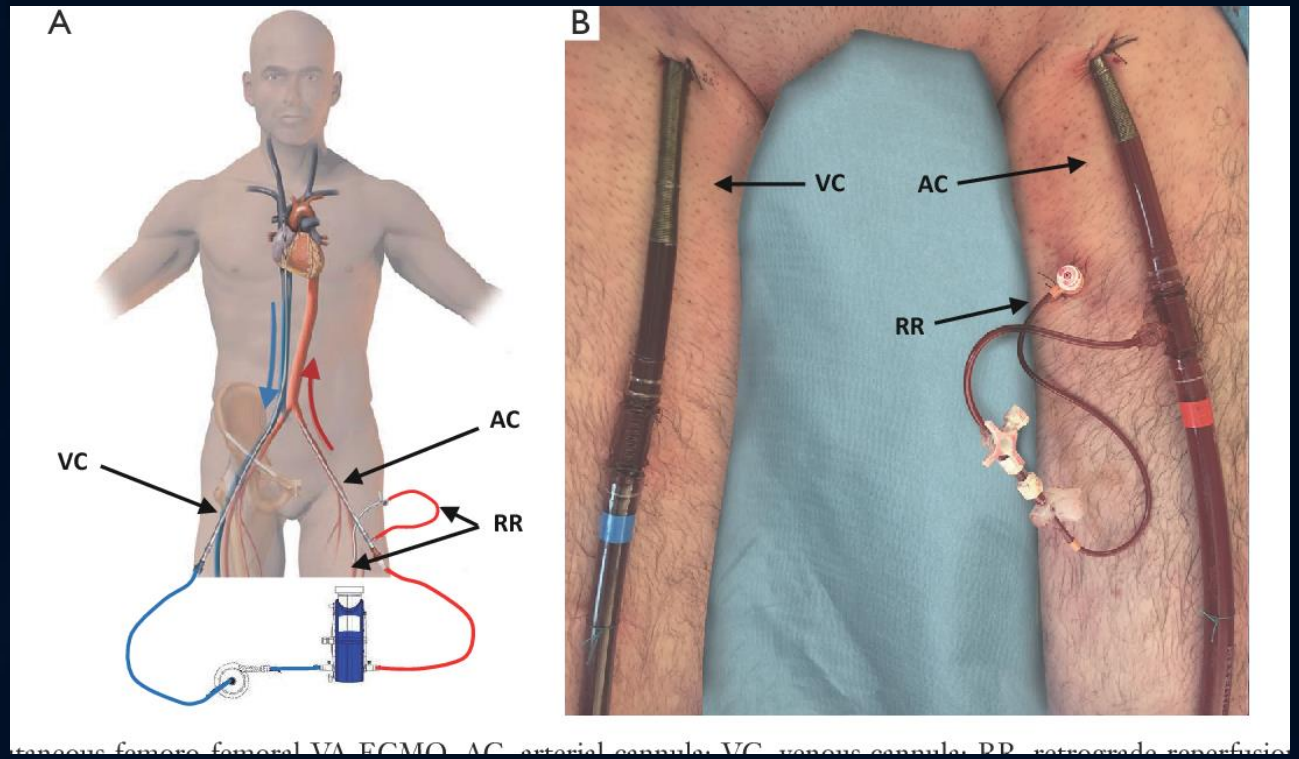
Intra-Aortic Balloon Pump



Impella 5.5



VA ECMO



taneous femoro-femoral VA-ECMO. AC, arterial cannula; VC, venous cannula; RR, retrograde reperfusio

The Future of Cardiogenic Shock

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- UL Cardiogenic Shock Policy (2022)

Case Study#1 -CGS vs Septic Shock

- 64-year-old male with PMH HF with rEF and afib who presented to the ER with hypotension. On home inotropic support.
- In the ER, WBC 25k, fever, required levophed and vasopressin drips.
- Discussion

Case Study #2- Cardiac Versus Septic Shock

- A 63-year-old female with PMHx of CAD s/p CABGx3v 4 weeks ago was brought to the ED with reports of tachycardia and chest pain as well as a wrist laceration after a fall while walking her dog.
- Vital Signs : BP 76/54 mmHg, P 83 bpm, RR 30/min and temperature is 101°F
- Discussion

Thank you!!

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