SHOCK AND VASOPRESSORS: MEDICAL MANAGEMENT OF THE CRITICALLY ILL PATIENT

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Educational Objectives

1. To provide an overview and diagnostic approach to various shock states
2. To discuss the use and mechanism of vasopressors and inotropes

APPROACH TO SHOCK

What is shock?

Evidence of organ hypoperfusion

What does that look like?

Hypotension: sBP<90, MAP <70

Anything else?

Determinants of Mean Arterial Pressure (MAP)

BP/MAP = Cardiac Output (CO) X Systemic Vascular Resistance

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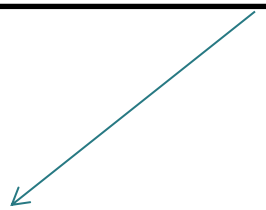
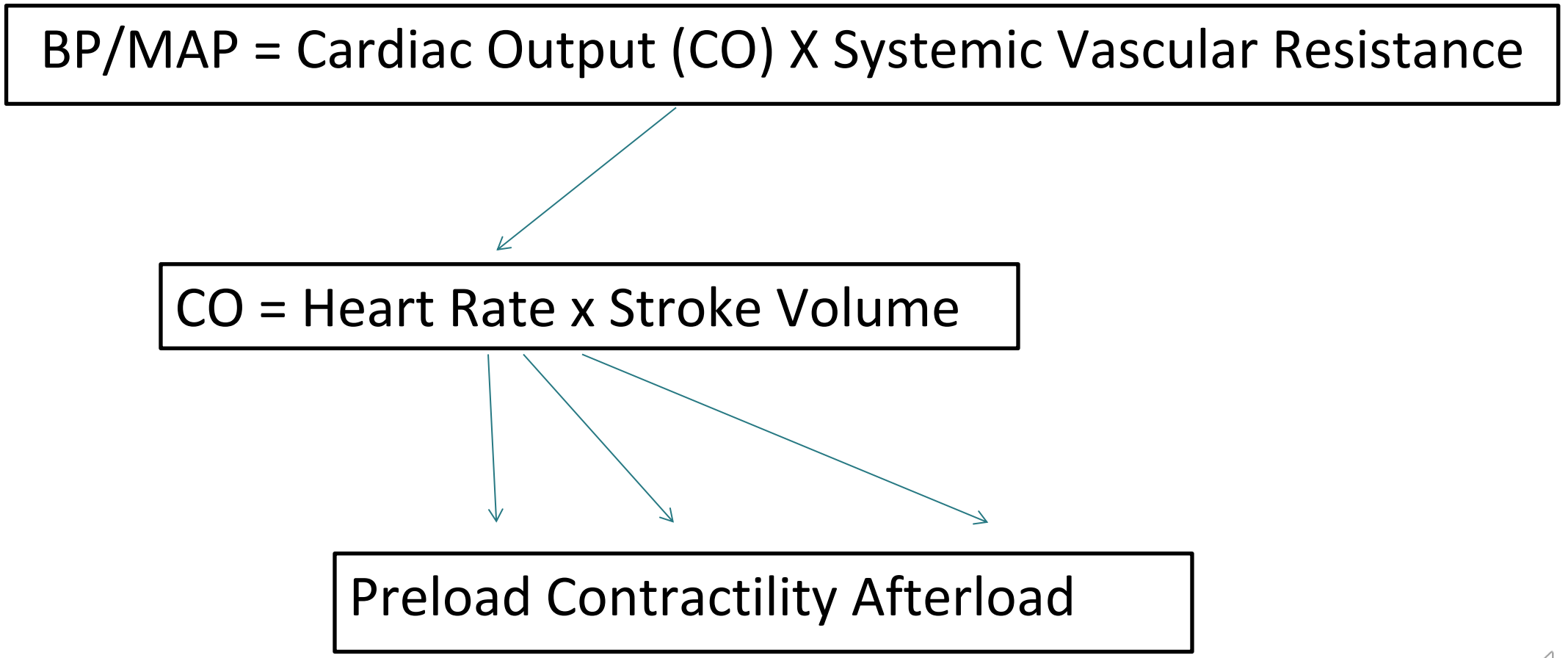


CO = Heart Rate x Stroke Volume

Determinants of Mean Arterial Pressure (MAP)



CATEGORIES of Shock	Examples
DISTRIBUTIVE • warm, well perfused	Sepsis General Inflammation (pancreatitis/burns/toxic) Anaphylaxis Myxedema Coma (?cardio + loss of vascular tone) Adrenal insufficiency (+ other endo) Neurogenic
CARDIOGENIC • Cold, mottled, peripherally shut down, high JVP	Left ventricular failure (mi, myocarditis, septic cardiomyopathy) Right ventricular failure (pe, mi) Arrhythmia
HYPOVOLEMIC • Cold, peripherally shut down, low urine output, flat JVP	Diuresis Hemorrhagic
OBSTRUCTIVE	Tamponade Tension pneumothorax Abdominal compartment synd

Principles of Management

- Rapid recognition of SHOCK
- Rapid recognition of TYPE of SHOCK
  - Tension pneumothorax
  - Anaphylaxis

Support end organ perfusion
(vasopressors/inotropes/fluids)

Institution of particular therapies for underlying cause

Simultaneous initiation of interventions to support BP while thinking through differential diagnosis

The 12am ward call…

77 year-old male

- Admitted for SBO, found to have pyelonephritis
- Started on cipro, 2 peripheral IVs
- Fever is persistent despite Antibiotics
- last assessed by team at 7pm, BP 95/60, HR110 NSR

RN calls you STAT

SBP 69, HR 135, Sats 92% on 40%FM

Your 12am realities:

1. You don’t know much about patient
2. You need to have a good differential
3. You need to immediately rule out life threatening/rapidly reversible causes

The 12am ward call…

Elevator thoughts:

SEPTIC SHOCK? (fever, nosocomial infection)
ANAPHYLACTIC SHOCK? (new medication today)
ADRENAL INSUFFICIENCY? (did someone forget to order their steroids??)

CARDIOGENIC (did he have an MI)
CARDIOGENIC (right side) – has he been on DVT prophylaxis

HYPOVOLEMIC (does he have C diff? has he been getting diuresis)
HYPOVOLEMIC (?is he bleeding)

OBSTRUCTIVE (? Does he have a pneumothorax)
OBSTRUCTIVE (???Did he develop tamponade → unlikely)

The 12am ward call…

RAPID ASSESSMENT

- **Re-check vitals!**
- A - Airway
- B - Breathing
- C - IV access, BP, Fluids?

Head to Toe Exam:

- CNS → GCS
- H&N → angioedema/hives/tracheal deviated)
- GI/GU → get a foley
- ID (temp)

CALL FOR HELP

What do you think is going on?
What would you do next?

The 12am ward call…

Investigations

- Routine bloodwork
- ABG, lactate
- CXR, ECG

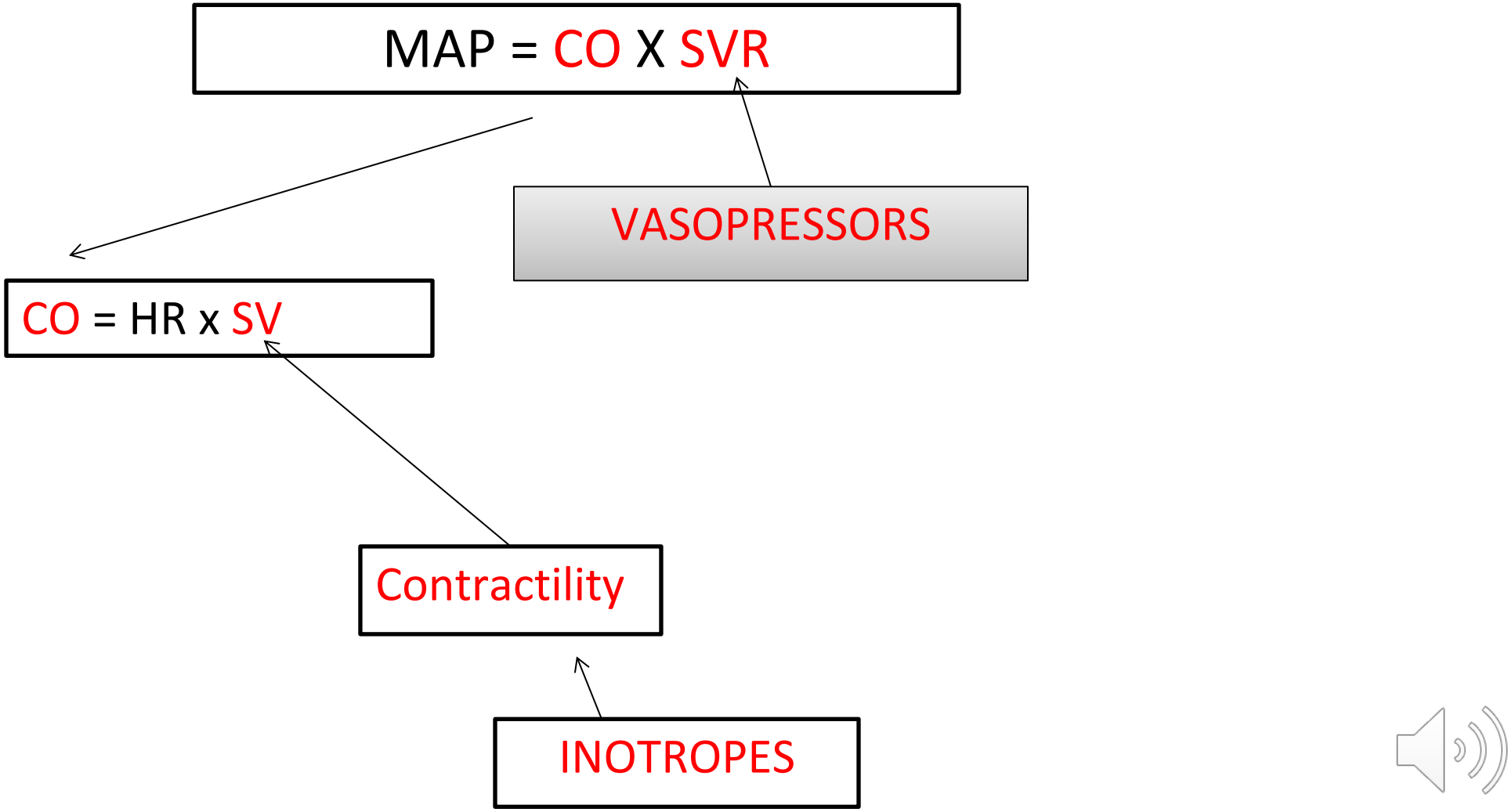
Management

- Fluids
- Vasopressor?
- Antibiotics?

What do you think is going on?
What would you do next?

VASOPRESSORS:
The ABC's or α-β-δ

Vasopressors vs. Inotropes



RECEPTOR	ACTION
Alpha-1	Vascular smooth muscle Mediates vasoconstriction
Alpha-2	CNS Mediates sedation, analgesia and platelet aggregation
Beta-1	Located in the heart Mediates increased contractility and HR (inotropy, chronotropy)
Beta-2	Mainly Smooth muscle of bronchi Mediates bronchodilatation Blood vessels • Dilation of coronary vessels, dilation of vessels to skeletal muscle

Mechanism of Action

Vasopressor	Alpha	Beta	Others	Effect
Norepinephrine (0.01-0.5mcg/kg/min)	+++	++	------	Increased SVR, increased HR at higher doses
Epinephrine (0.01-0.4 mcg/kg/min)	++++	++++	-------	Increased SVR, increased HR at higher doses, may increase lactate production
Dopamine (5-20 mcg/kg/mi)	>10	5-10	N/A	Lower dose – mostly ionotropy Higher doses - vasopressor
Phenylephrine (100mcg bolus)	++++	------	-------	Increased SVR, may cause reflex bradycardia
Vasopressin (0.04u/min or 2.4U/hr)	-----	-----	VP recep-tor	Replace physiology Vasopressin; may redirect splanchnic blood flow,

Comparing Vasopressors

Inotropes	Alpha	Beta	Others	Effect
Dobutamine	----	+++ b1 >b2		Increased HR, Pulm vasodilation
Milrinone	------		cGMP	Increased contractility, slow effect, long half life, pulm vasodilation and LV relaxation
Isoproterenol	-----	++++ b1		Increased contractility

Comparing Inotropes

Phenylephrine

- Mechanism:
  - Pure vasoconstriction effect

- When to use:
  - Good for intermittent peripheral pushes to temporize BP in setting of decreased SVR
    - 10mg/100cc mini bag
    - Administer 1cc (100mcg) at a time

- Because of potent vasoconstriction may reduce SV/CO, often not used as infusion vasopressor of choice. Consider only if:
  - NE causes major arrhythmias
  - Cardiac output known to be high → Do not use in cardiogenic shock or significant bradycardia
  - Salvage therapy

ALPHA 1 Vasoconstriction	BETA 1 Inotropy/Chronotropy	BETA 2 Vasodilation
+++++		

Norepinephrine

- Mechanism:
  - Predominant vasoconstriction (arterial/venous)
  - Some inotropic/chronotropic activity

- When to use:
  - Good drug for most shock states that have a vasodilatory component
    - Exception = anaphylaxis
  - First line for septic shock
  - Bridge vasopressor for other shock states

ALPHA 1 Vasoconstriction	BETA 1 Inotropy/Chronotropy	BETA 2 Vasodilation
+++	++	

Epinephrine

- Mechanism:
  - Low doses → predominant an inotropic/vasodilatory effects
  - High doses → predominant vasoconstrictive effects

- When to use:
  - First line for anaphylaxis
  - Second/third line for septic shock, mixed shock (cardiogenic + vasodilatory)

- Side Effects:
  - Arrhythmias
  - Elevated lactate: Potent splanchnic vasoconstrictive effects/metabolic stimulation

DOSE	ALPHA 1 Vasoconstriction	BETA 1 Inotropy/Chronotropy	BETA 2 Vasodilation
0.01-0.05 mcg/kg/min	+	++++	++
0.05-1.0 mcg/kg/min	++++	++	++

Dopamine

- Mechanism:
  - Low doses → predominant inotropic/chronotropic /vasodilatory effects
  - High doses → predominant vasoconstrictive effects

- When to use:
  - Low BP + low HR
  - Renal dose to preserve kidney function does not work
  - Second or third line agent for septic shock

- Side effects:
  - High risk for arrhythmias, more arrhythmias than NE

DOSE	ALPHA 1 Vasoconstriction	BETA 1 Inotropy/Chronotropy	BETA 2 Vasodilation
5-10 mcg/kg/min	+	++++	++
>10 mcg/kg/min	+++	++	0

Vasopressin

- Mechanism:
  - Endogenously released peptide hormone, adjunct to catecholamines
  - Acts on V1 smooth muscle receptors

- When to use:
  - Vasodilatory shock
  - Second line agent in refractory vasodilatory shock states such as sepsis (<2.4 units/hr)

- Side Effects:
  - Higher doses → digital, splanchnic cardiac ischemia
  - Theoretical pulmonary vasodilatory effect

Dobutamine

- Mechanism:
  - Predominantly inotropic, chronotropic, vasodilatory effect

- When to use:
  - Good drug for cardiogenic shock
  - Good drug if cardiogenic component to septic shock (once volume replete)

- Side Effects:
  - Arrhythmias (particularly >15 mcg/kg/min)
  - If under resuscitated or volume deplete, vasodilation may unmask and drop BP

ALPHA 1 Vasoconstriction	BETA 1 Inotropy/Chronotropy	BETA 2 Vasodilation
0/+	+++	++

Milrinone

- Mechanism:
  - Phosphodiesterase inhibitor
    - Breaks down cAMP
    - Increases intracellular calcium → Increased myocardial contractility

- When to use:
  - Cardiogenic shock
  - May improve cardiac relaxation

- Side Effects:
  - Accumulates in renal failure
  - Long duration of action (4 hours)

Summary

- Shock is a heterogenous disease with multiple causes
- The cause of shock is critical to determine to guide ongoing management
  - The clinical exam and history can provide important clues
- Norephinephrine is the best first line vasopressor for most shock states → When in doubt, start with this agent