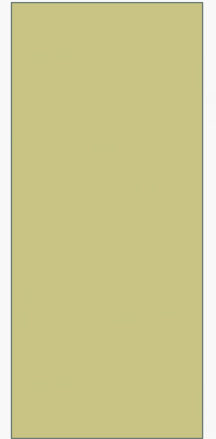




# HYPOTENSION IS DANGEROUS

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# MOST DANGEROUS EFFECTS OF LOW BLOOD PRESSURE

Low blood pressure can cause the following:



BLURRED VISION



LOSS OF  
CONCENTRATION



DIZZINESS OR LOSS OF  
CONSCIOUSNESS



NAUSEA

# OBJECTIVES

- Case-based overview of pressors
- Debunking pressor myths
- Utilizing push-dose pressors



# CASE 1

82-year old male, SOB, feeling unwell, generally weak.

T 38.2, RR 20 – 88% on RA, P 120, BP 75/45

Slightly depressed mentation

Tachycardic, no murmurs

Tachypneic, rales

Abdomen soft and nontender

Extremities with trace pedal edema

Skin warm to touch, dry

# NEXT STEPS IN ASSESSMENT?

Past Medical History

CHF, COPD, hypertension,  
hyperlipidemia

Medications

Lisinopril, Metoprolol, Lasix, ASA, Albuterol

# NEXT STEPS IN ASSESSMENT?

Adjunctive information to vitals?

ETCO<sub>2</sub>



# TREATMENTS?

T 38.2, RR 20 – 88% on RA, P 120, BP 75/45,  
ETCO<sub>2</sub> 22

**Fluid bolus** – how much?

Severe Sepsis and Septic Shock guidelines =  
30mL/kg

# CASE 1

- You administer high flow oxygen, transitioning to CPAP for respiratory support
- ETCO<sub>2</sub> is now 20
- Patient initially responds to a fluid bolus with a blood pressure of 100/79, then becomes hypotensive again.



# TREATMENTS?

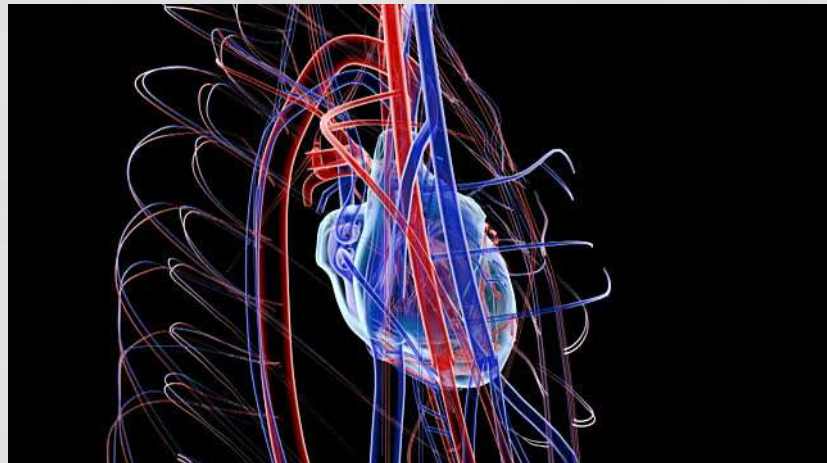
Fluid bolus – done

Pressors

Which one?

# WHAT IS YOUR RESUSCITATION GOAL?

## End Organ Perfusion



# HOW TO MEASURE END ORGAN PERFUSION?

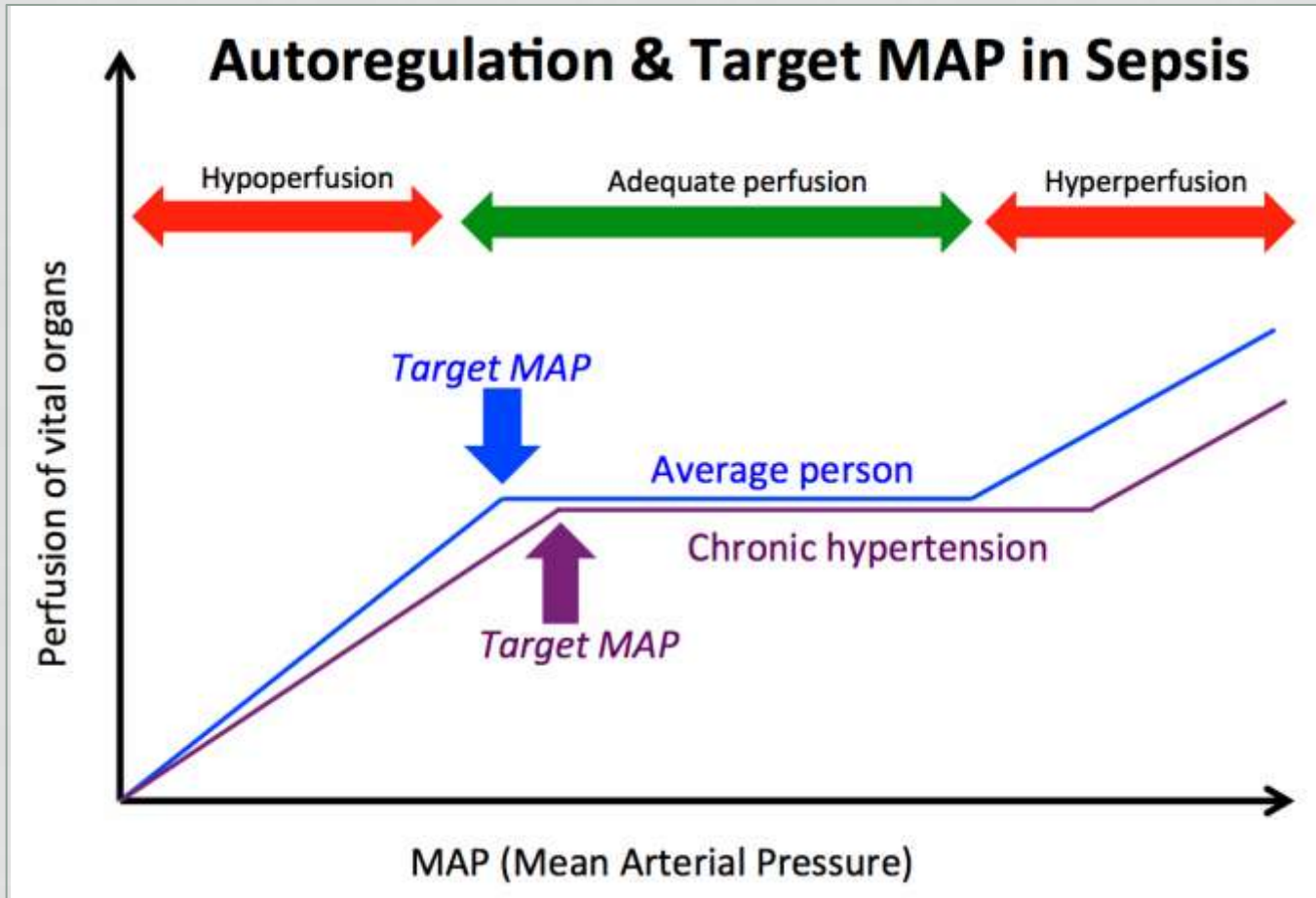
## **MAP:**

- Mean Arterial Pressure: Systolic – diastolic
- Linear relationship between MAP and blood flow to vital organs
- Goal in Sepsis = 65mmHg

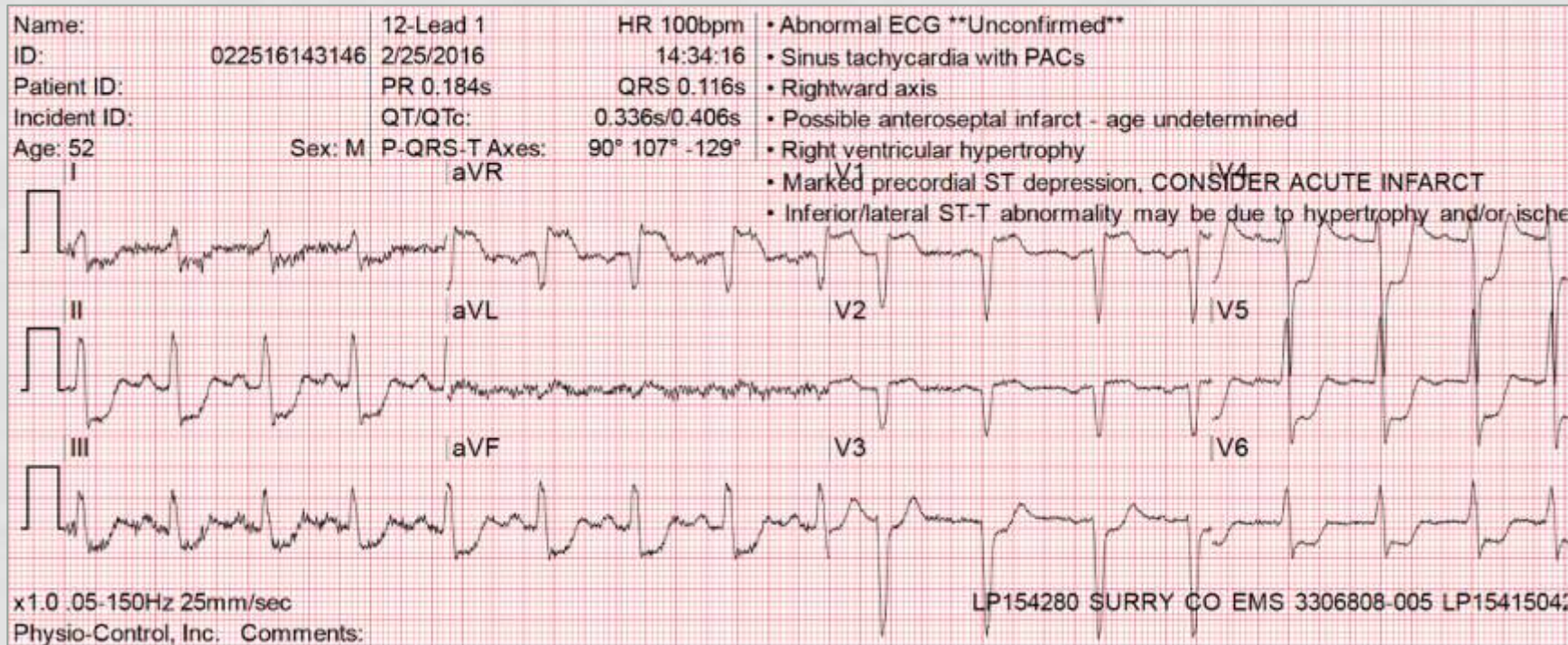
**What if my patient has chronic hypertension?**

**What is the right MAP?**

# MAP IN SEPSIS



# HOW TO MEASURE END ORGAN PERFUSION?



# CASE 1

Getting back to our poor patient...

BP 88/40, increasingly tired appearing  
Becoming obtunded  
Poor skin color

You start .....what pressor?



# VASOPRESSORS

Which one is best for my patient?

## Cochrane Review 2016

Except for increased arrhythmia risk with dopamine, there is **no significant difference** in mortality between vasopressors and “evidence of any other differences between any of the six vasopressors examined is insufficient”

# VASOPRESSORS VS INOTROPES

## Vasopressors:

the goal is to **increase afterload** via vasoconstriction and increased arterial pressure

## Inotropes:

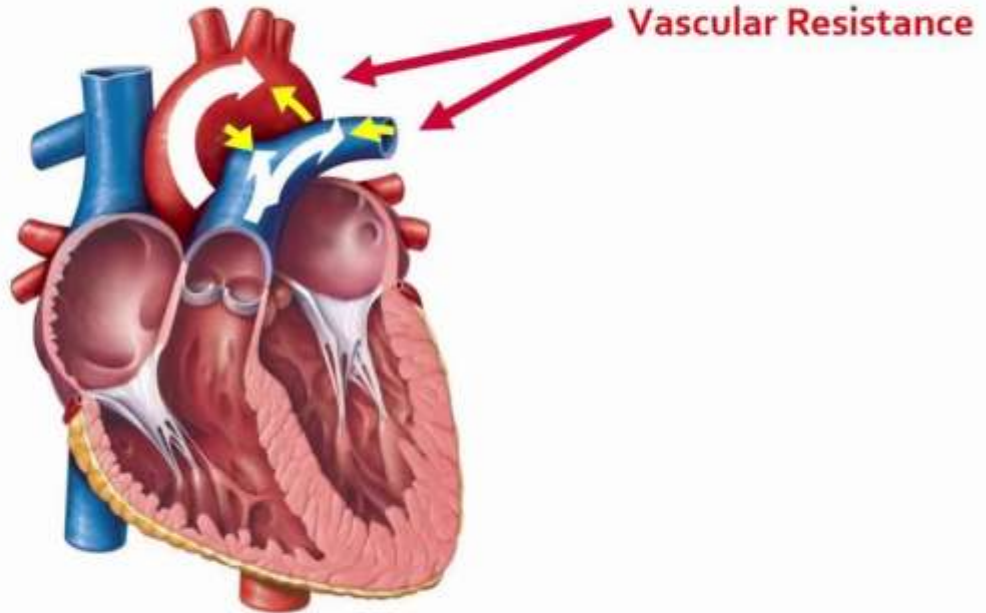
**increase cardiac contractility**, thereby improving stroke volume and cardiac output



# AFTERLOAD

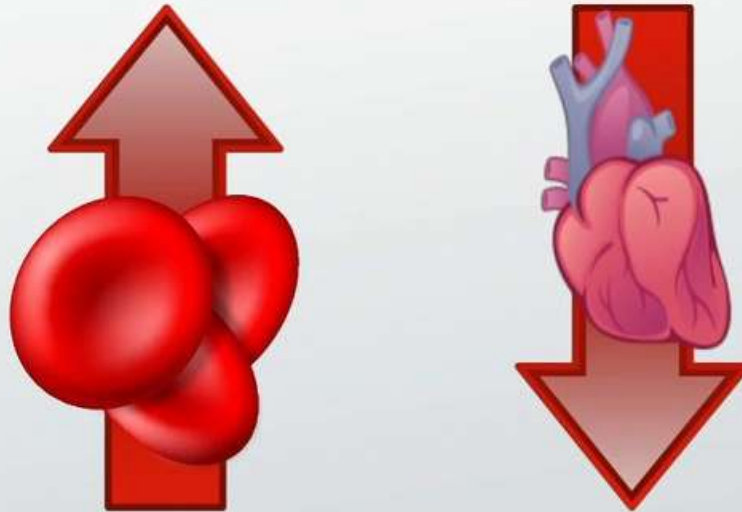
## Afterload

Refers to the amount of resistance the heart must pump against when ejecting blood



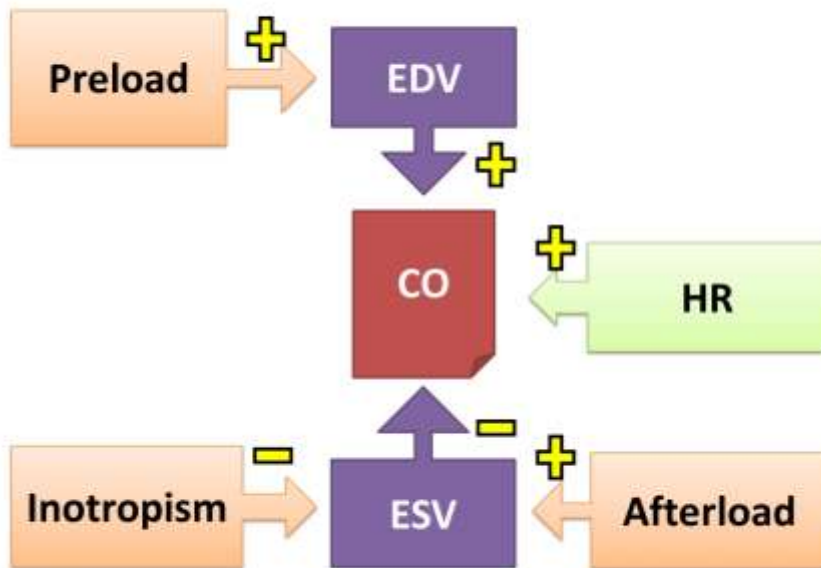
# AFTERLOAD AND CONTRACTILITY

***increased afterload = reduced contraction***



# CONTRACTILITY

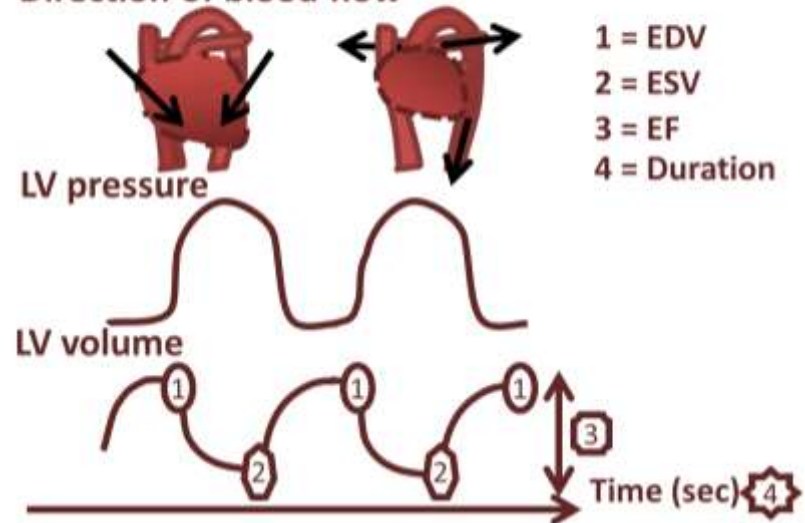
## Determinants of Cardiac Output



$$CO = HR \times SV$$

## Determinants of Stroke Volume

Direction of blood flow



# BEST USE FOR VASOPRESSORS

Vasopressors are best for hypotension due to distributive or obstructive shock

sepsis, anaphylaxis, PE, tamponade

## WHY?

**Pressors increase afterload**

Cardiac disease + increased afterload =  
**decreased cardiac output**

# BEST USE FOR INOTROPES

**Inotropes** are usually preferred when there is suspicion for poor cardiac function

Examples:

1. cardiogenic shock
2. septic shock in the setting of CHF

# INOPRESSORS

Most medications used in emergency  
medicine = “inopressors”

# CASE 2

35-year old male, helmeted motorcycle collision. AMS with GCS 8, deteriorating on scene. Multiple superficial abrasions, flail chest evidence by paradoxical motion, tachypneic.

T 35.6, P 140, BP 100/40, RR 40



# CASE 2

Walk through your assessment & interventions

Primary survey

ABCs

Life threatening injuries

Secondary survey

C-collar and BB

DCAP-BTLS



# CASE 2

SBP drops to 80/50, HR 120

## Treatments

- Fluids??
- Pressors??



## CASE 2

- You give LR 2 liters
  - Persistent hypotension
- Partner asks about sedation – patient moving arms as if to grab for tube
  - No lower extremity movement noted

## CASE 2

- How are you going to reassess the patient?
- How are you going to treat the persistent hypotension?
- What is your treatment goal in this case?

# INOPRESSOR COMPARISON

Pressor	Mechanism	Risk	Indication
Norepinephrine	$\alpha_1\alpha_2(\beta_1)$	Safest, myotoxicity, arrhythmia, ischemia	<b>1<sup>st</sup> line for sepsis, neurogenic, cardiogenic</b>
Epinephrine	$\beta_1\beta_2(\alpha_1)$	Tachy, lactic acidosis	<b>1<sup>st</sup> line: anaphylaxis 2<sup>nd</sup> line: sepsis</b>
Dopamine	dopa $\beta_1\alpha_1$	Dysrhythmia, incr mortality	Refractory shock
Vasopressin	vasopressin R	Digital ischemia	<b>2<sup>nd</sup> line: sepsis</b> <i>Pure pressor</i>
Phenylephrine	pure $\alpha_1$	Reflex brady, ischemia	Adjunct to norepi <i>Pure pressor</i>

# DEEPER DIVE ON NOREPINEPHRINE

- Stimulates  $\alpha$ -1 and  $\alpha$ -2 receptors
  - Small amount of  $\beta$ -1 agonism (*inotropic*)

## **Balanced venous and arterial vasoconstrictor**

- Arteries: increased coronary blood flow and afterload
- Veins: increased physiologic venous reserve = increased preload

# DEEPER DIVE ON NOREPINEPHRINE

- Number needed to treat = 9, compared with dopamine
- Compared to epinephrine, phenylephrine, vasopressin, it is superior in improving:
  - central venous pressure
  - urinary output
  - arterial lactate

# WORD OF CAUTION ON NOREPI

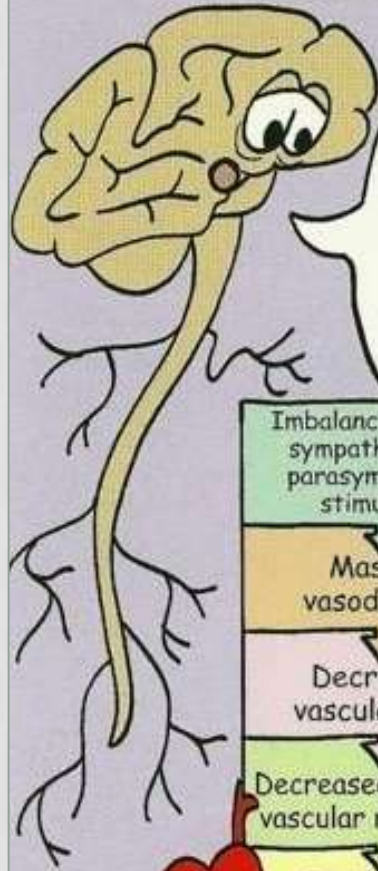
- Norepinephrine demonstrates
  - NO mortality benefit
  - NO improvement in hemodynamic endpoints
  - May NOT improve end-organ flow
- Risks of norepinephrine include
  - Cardiac myocyte toxicity
  - Cardiac arrhythmias
  - Arterial vasoconstriction to digits = ischemia

# CASE 2 - TREATMENT

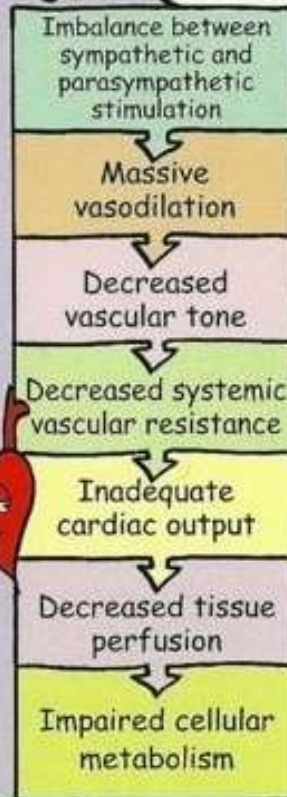
- You reassess patient:
  - No abdominal distension
  - No obvious hemorrhage
  - Equal bilateral breath sounds
- You now suspect spinal injury/neurogenic shock
- Start norepinephrine at 8mcg/min with improvement in blood pressure



# NEUROGENIC SHOCK



Any factor that stimulates parasympathetic activity or inhibits sympathetic activity of vascular smooth muscles can cause neurogenic shock, which results in widespread and massive vasodilation.



## Causes of neurogenic shock:

- Spinal cord injury above T5
- Spinal anesthesia
- Vasomotor center depression (e.g., severe pain, drugs, hypoglycemia)

Symptoms of neurogenic shock consist of low systemic vascular resistance, excessive parasympathetic activity, and bradycardia.



## CASE 3

26-year old female with SOB, throat tightening, nausea and vomiting. Multiple food/med allergies, unknown exposure.

P120 BP120/50 RR 16 – SpO2 95% RA

Anxious appearing

Lungs with scattered wheezes

Tachycardic, no murmur

Nontender abdomen

# CASE 3

Differential?

Treatment?

# CASE 3 - TREATMENT

Patient becomes increasingly anxious.

## Treatment

- Benadryl 25-50mg IV
- Duoneb
- Solumedrol



# CASE 3

BP 70/40 P 140 RR 40

## Treatment

- Epi 1:1000
- How many IM epi dosages before we move to an epi infusion?

# EPINEPHRINE

- Stimulates  $\beta$ -1 and  $\beta$ -2 receptors
  - > inotropic effects than norepinephrine
  - Due to its  $\beta$ -agonism, **epinephrine greatly increases heart rate and stroke volume, with a small amount of bronchodilation.**
  - Moderate stimulatory effect on  $\alpha$ -1 receptors
- Causes tachycardia and lactic acidosis
  - “dirty epi drip”
  - Push-dose pressors...

# INOPRESSOR COMPARISON

Pressor	Mechanism	Risk	Indication
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## CASE 4

55-year old male with a history of CHF/COPD and fever.

P120, BP 105/40, RR 30, SpO<sub>2</sub> 75% on RA

Anxious, tachypneic

Rales bilaterally

Tachycardic, no murmur



## CASE 4

Initially responds to CPAP with oxygen saturations improved to 95%.

Patient fatiguing

You consider emergent intubation...but what about his blood pressure?

# PUSH DOSE PRESSORS



# PUSH DOSE PRESSORS?!

Used by anesthesiologists for decades to reverse transient hypotension.

Safe, effective

Mostly ephedrine, phenylephrine

Epinephrine now more widespread and acceptable.

# PROS OF PUSH DOSE EPI

- Good for ***short-term pressor needs***, transient hypotension, or as a bridge to fluid resuscitation or vasopressor drips.
- Best for 10-15 minute situations, long transport – need a pressor drip.

# PROS OF PUSH DOSE EPI

- Easy to mix from readily available 1:10,000 epinephrine and normal saline. The dosing is relatively straightforward.
- May need more than one syringe mixed up.

# EMCRIT

## EPINEPHRINE

Has alpha and beta<sub>1/2</sub> effects so it is an inopressor

Do not give cardiac arrest doses (1 mg) to patients with a pulse

### Mixing Instructions:

- Take a 10 ml syringe with 9 ml of normal saline
- Into this syringe, draw up 1 ml of epinephrine from the cardiac amp (Cardiac amp contains Epinephrine 100 mcg/ml)
- Now you have 10 mls of Epinephrine 10 mcg/ml

**Onset**-1 minute

**Duration**-5-10 minutes

**Dose**-0.5-2 ml every 2-5 minutes (5-20 mcg)



# DOSING OF PUSH DOSE EPI

- Slow IV push for hypotension or bradycardia
- 2-10mcg per minute
  - Cardiogenic shock – 0.1-0.5 mcg/kg/minute (10-50mcg per minute for 100kg patient).
  - Severe anaphylaxis 100-250mcg IV every 3-5 minutes followed by continuous IV infusion.

# CASE 5

- 65-year old hypertensive, diabetic with cardiac arrest
- Epinephrine x3, Shock x4, Amio
- Obtain ROSC
  - VS: BP 80/40, P 100, RR bagged 12 bpm, FiO<sub>2</sub> 100%

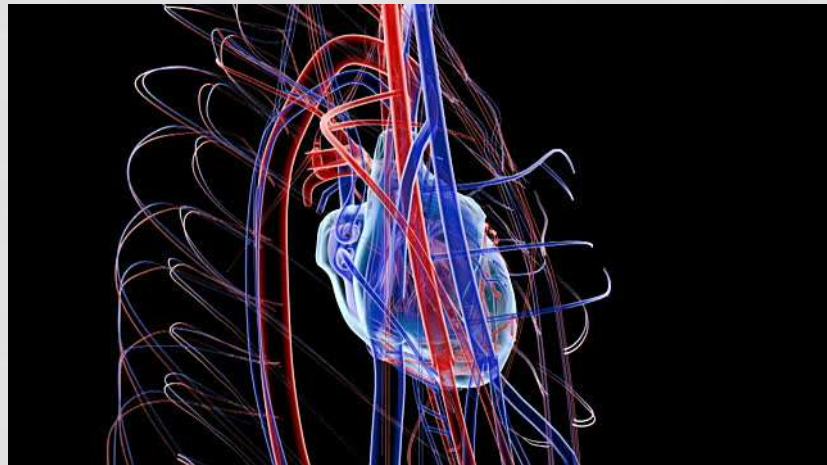


# CASE 5

- Do you need to treat post-ROSC hypotension?
- Do you start with fluids?
- What pressors are best?

# WHAT IS YOUR RESUSCITATION GOAL?

End Organ Perfusion



# POST-ROSC HYPOTENSION

- Need to balance the metabolic needs of an ischemic brain with overstressing a decompensated heart.
- 1<sup>st</sup> – volume pressures
  - Particularly in volume-dependent disease (Inferior MI)
  - Start vasoactive drugs when hypotensive after a rapid infusion of 2 L of crystalloid.
- 2<sup>nd</sup> – Pressors

# POST-ROSC HYPOTENSION

- Inotropes and vasopressors can mitigate the myocardial dysfunction after cardiac arrest.
- No evidence demonstrating superiority of any vasopressor after cardiac arrest
- Septic patients: No difference in dopamine and norepinephrine with regard to mortality
- Dopamine = arrhythmogenic

**Norepinephrine is the first line inopressor for an undifferentiated post-arrest patient**

# SUMMARY

Pressor	Mechanism	Risk	Indication
Norepinephrine	$\alpha_1\alpha_2(\beta_1)$	Safest, myotoxicity, arrhythmia, ischemia	<b>1<sup>st</sup> line for sepsis, neurogenic, cardiogenic</b>
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# SUMMARY

- Most drugs used in EMS and Emergency Medicine are truly inopressors (alpha and beta effects)
- Norepinephrine (Levophed) is our first line inopressor for most situations
- Dopamine actually demonstrates INCREASED mortality in studies
- Epinephrine is first line for anaphylaxis

# SUMMARY

- Dirty epi drips are effective and safe
- Push-dose epinephrine is a useful tool for preventing peri-intubation cardiac arrest in hypotensive or borderline hypotensive patients.
- Post-arrest patients should get fluids, then norepinephrine for hypotension

THANK YOU!

Questions?

