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?

ETCO2
TREATMENTS?

T 38.2, RR 20 – 88% on RA, P 120, BP 75/45, ETCO2 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

• ETCO2 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?
HOW TO MEASURE END ORGAN PERFUSION?

- Skin color and temp
- Capillary refill
- Mental status
- Urine output
- Cardiac ischemia
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**

Refers to the amount of resistance the heart must pump against when ejecting blood.
increased afterload = reduced contraction
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

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DEEPER DIVE ON NOREPINEPHRINE

• Stimulates α-1 and α-2 receptors
  • Small amount of β-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?
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 β-1 and β-2 receptors
  • > inotropic effects than norepinephrine
  • Due to its β-agonism, epinephrine greatly increases heart rate and stroke volume, with a small amount of bronchodilation.
  • Moderate stimulatory effect on α-1 receptors

• Causes tachycardia and lactic acidosis
  • “dirty epi drip”
  • Push-dose pressors...
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CASE 4

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

P120, BP 105/40, RR 30, SpO2 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?!

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.
EPINEPHRINE

Has alpha and beta1/2 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 (10-50mgc 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, FiO2 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\textsuperscript{st} – volume pressures
  • Particularly in volume-dependent disease (Inferior MI)
  • Start vasoactive drugs when hypotensive after a rapid infusion of 2 L of crystalloid.

• 2\textsuperscript{nd} - 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.**
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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?