Shock and Bleeding

- A 35-year-old man is admitted with systolic blood pressure (BP) of 60 mm Hg and a heart rate (HR) of 150 bpm following a gunshot wound to the liver . What is the effect on the kidneys?
- (A) They tolerate satisfactorily ischemia of 3–4 hours duration.
- (B) They undergo further ischemia if hypothermia is present.
- (C) They can become damaged, even though urine output exceeds 1500 mL/d.
- (D) They are affected and cause an increased creatinine clearance.
- (E) They are prevented from further damage by a vasopressor.

- Immediate management of a patient with Multiple fracture and fluid loss includes the infusion -
- Blood
- Dextran
- Normal saline
- Ringer lactate

Hypotension

- **In Adults:**
  - **systolic BP ≤ 90 mm Hg**
  - **mean arterial pressure ≤ 60 mm Hg**
  - **⇩ systolic BP > 40 mm Hg from the patient's baseline pressure**

SHOCK

Inadequate perfusion (blood flow) leading to inadequate oxygen delivery to tissues

"Hypoperfusion can be present in the absence of significant hypotension."

Physiology

- Basic unit of life = cell
- Cells get energy needed to stay alive by reacting oxygen with fuel (usually glucose)
- No oxygen, no energy
- No energy, no life

Cardiovascular System

- Transports oxygen, fuel to cells
- Removes carbon dioxide, waste products for elimination from body

Cardiovascular system must be able to maintain sufficient flow through capillary beds to meet cell's oxygen and fuel needs

Flow = Perfusion

Adequate Flow = Adequate Perfusion

Inadequate Flow = Indequate Perfusion (Hypoperfusion)

Hypoperfusion = Shock

What is needed to maintain perfusion?

- Pump
- Pipes
- Fluid

Heart

Blood Vessels

Blood

How can perfusion fail?

- Pump Failure
- Pipe Failure
- Loss of Volume

Types of Shock and Their Causes

Cardiogenic Shock

- Pump failure
- Heart's output depends on
  - How often it beats (heart rate)
  - How hard it beats (contractility)
- Rate or contractility problems cause pump failure

Cardiogenic Shock

- Causes
  - Acute myocardial infarction
  - Very low heart rates (bradycardias)
  - Very high heart rates (tachycardias)

Why would a high heart rate caused decreased output?

Hint: Think about when the heart fills.

Neurogenic Shock

- Loss of peripheral resistance
- Spinal cord injured
- Vessels below injury dilate

What happens to the pressure in a closed system if you increase its size?

Hypovolemic Shock

- Loss of volume
- Causes
  - Blood loss: trauma
  - Plasma loss: burns
  - Water loss: Vomiting, diarrhea, sweating, increased urine, increased respiratory loss

If a system that is supposed to be closed leaks, what happens to the pressure in it?

Psychogenic Shock

- Simple fainting (syncope)
- Caused by stress, pain, fright
- Heart rate slows, vessels dilate
- Brain becomes hypoperfused
- Loss of consciousness occurs

What two problems combine to produce hypoperfusion in psychogenic shock?

Septic Shock

- Results from body's response to bacteria in bloodstream
- Vessels dilate, become "leaky"

What two problems combine to produce hypoperfusion in septic shock?

Anaphylactic Shock

- Results from severe allergic reaction
- Body responds to allergen by releasing histamine
- Histamine causes vessels to dilate and become "leaky"

What two problems combine to produce hypoperfusion in anaphylaxis?

OBSTRUCTIVE SHOCK

- Flow of blood is obstructed.
  - Cardiac tamponade
  - Constrictive pericarditis
  - Tension pneumothorax.
  - Massive pulmonary embolism
  - Aortic stenosis.

PATHOPHYSIOLOGY OF SHOCK SYNDROME

Cells switch from aerobic to anaerobic metabolism

↓

lactic acid production

↓

Cell function ceases & swells

↓

membrane becomes more permeable

↓

electrolytes & fluids seep in & out of cell

↓

Na+/K+ pump impaired
mitochondria damage
cell death

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- Stimulated by baroreceptors

- Increased heart rate
- Increased contractility
- Vasoconstriction (SVR-Afterload)
- Increased Preload

COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response

- **SNS - Hormonal:** Renin-angiotension system
  - Decrease renal perfusion →
  - Releases renin → angiotension I
  - angiotension II → potent vasoconstriction &
  - releases aldosterone → adrenal cortex
  - sodium & water retention →

COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response

- **SNS - Hormonal:** Antidiuretic Hormone
  - Osmoreceptors in hypothalamus stimulated →
  - ADH released by Posterior pituitary gland →
    - Vasopressor effect to increase BP
    - Acts on renal tubules to retain water

COMPENSATORY MECHANISMS:

Sympathetic Nervous System (SNS)-Adrenal Response

- **SNS - Hormonal:** Adrenal Cortex
  - Anterior pituitary releases adrenocorticotropic hormone (ACTH)
  - Stimulates adrenal Cx to release glucorticoids
  - Blood sugar increases to meet increased metabolic needs

Stages of Shock

- **Initial stage -** tissues are under perfused, decreased CO, increased anaerobic metabolism, lactic acid is building
- **Compensatory stage -** Reversible. SNS activated by low CO, attempting to compensate for the decrease tissue perfusion.
- **Progressive stage -** Failing compensatory mechanisms: profound vasoconstriction from the SNS → ISCHEMIA Lactic acid production is high → metabolic acidosis
- **Irreversible or refractory stage -** Cellular necrosis and Multiple Organ Dysfunction Syndrome may occur

DEATH IS IMMINENT!!!!

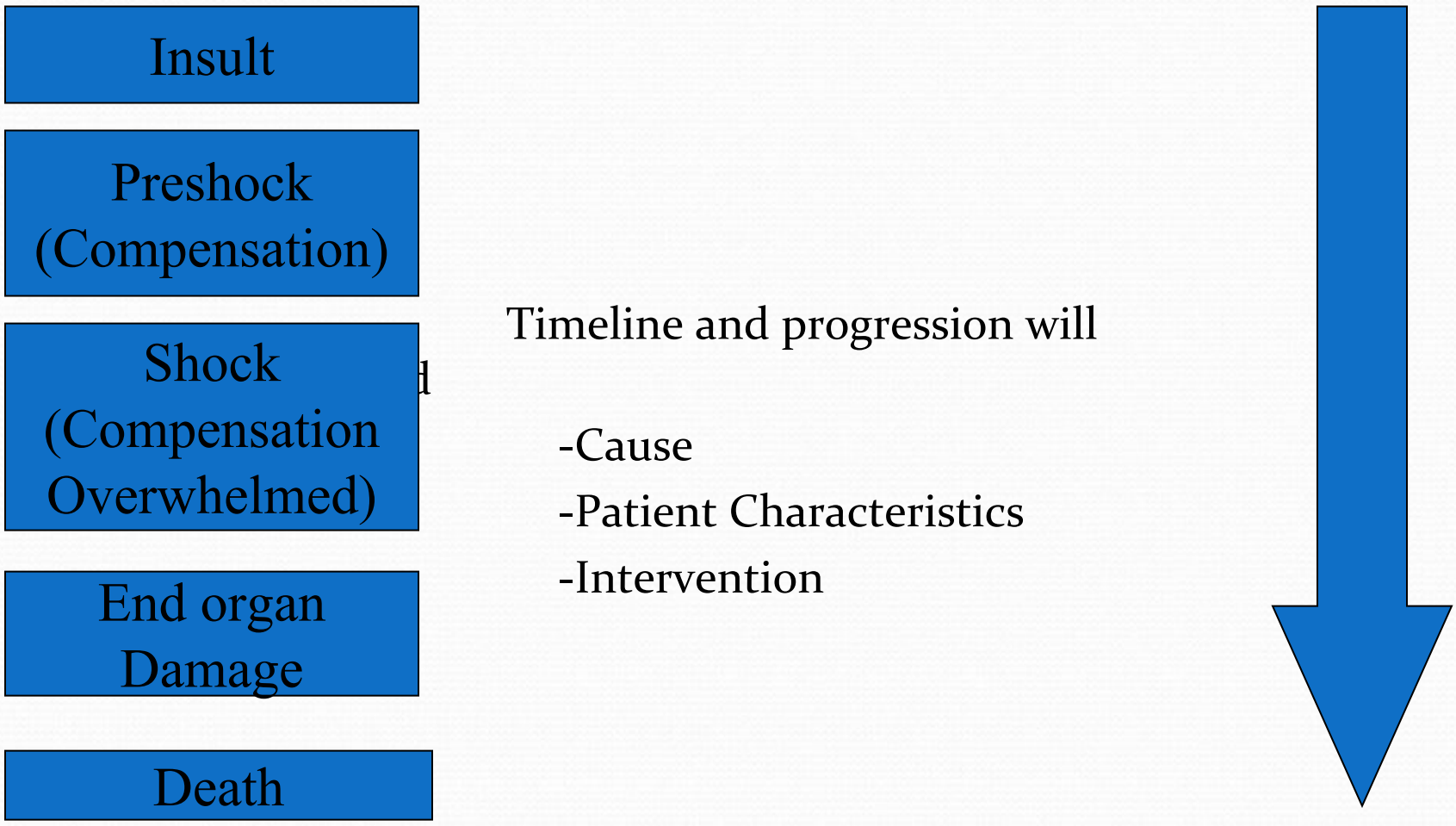
Net results of cellular shock:

- systemic lactic acidosis
- decreased myocardial contractility
- decreased vascular tone
- decrease blood pressure, preload, and cardiac output

Case 1

- 24 year old male
- Previously healthy
- Lives in a malaria endemic area (PNG)
- Brought in by friends after a fight - he was kicked in the abdomen
- He is agitated, and won't lie flat on the stretcher
- HR 92, BP 126/72, SaO2 95%, RR 26

Stages of Shock



Case 1: Stages of Shock

Stage	Pathophysiology	Clinical Findings
Insult	Splenic Rupture -- Blood Loss	Abdominal tenderness and girth

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Preshock	**Hemostatic compensation** MAP =↓**CO**(↑**HR** x↓**SV**) x↑**SVR** Decreased CO is compensated by increase in HR and SVR	MAP is maintained HR will be increased Extremities will be cool due to vasoconstriction

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Case 1: Stages of Shock

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Shock	Compensatory mechanisms fail	MAP is reduced Tachycardia, dyspnea, restlessness
End organ dysfunction	Cell death and organ failure	Decreased renal function Liver failure Disseminated Intravascular Coagulopathy Death

Is this Shock?

- Signs and symptom

Symptoms and Signs of Shock

- Level of consciousness
- Initially may show few symptoms
  - Continuum starts with
    - Anxiety
    - Agitation
    - Confusion and Delirium
    - Obtundation and Coma
- **In infants**
  - **Poor tone**
  - **Unfocused gaze**
  - **Weak cry**
  - **Lethargy/Coma**
  - **(Sunken or bulging fontanelle)**

Symptoms and Signs of Shock

- Pulse
  - Tachycardia HR > 100 - What are a few exceptions?
  - Rapid, weak, thready distal pulses
- Respirations
  - Tachypnea
  - Shallow, irregular, labored

Symptoms and Signs of Shock

- Blood Pressure
  - May be normal!
  - Definition of hypotension
    - Systolic < 90 mmHg
    - MAP < 65 mmHg
    - 40 mmHg drop systolic BP from from baseline
- Children
  - Systolic BP < 1 month = < 60 mmHg
  - Systolic BP 1 month - 10 years = < 70 mmHg + (2 x age in years)
- In children hypotension develops **late, late, late**
  - **A pre-terminal event**

Symptoms and Signs of Shock

- Skin
  - Cold, clammy (Cardiogenic, Obstructive, Hemorrhagic)
  - Warm (Distributive shock)
  - Mottled appearance in children
  - Look for petechia
- Dry Mucous membranes
- Low urine output <0.5 ml/kg/hr

Empiric Criteria for Shock

4 out of 6 criteria have to be met

- Ill appearance or altered mental status
- Heart rate >100
- Respiratory rate > 22 (or $PaCO_2$ < 32 mmHg)
- Urine output < 0.5 ml/kg/hr
- Arterial hypotension > 20 minutes duration
- Lactate > 4

Management of Shock

- History
- Physical exam
- Labs
- Other investigations
- Treat the Shock - Start treatment as soon as you suspect Pre-shock or Shock
- Monitor

Historical Features

- Trauma?
- Pregnant?
- Acute abdominal pain?
- Vomiting or Diarrhea?
- Hematochezia or hematemesis?
- Fever? Focus of infection?
- Chest pain?

Physical Exam

- Vitals - HR, BP, Temperature, Respiratory rate, Oxygen Saturation
- Capillary blood sugar
- Weight in children

Physical Exam

- In a patient with normal level of consciousness - Physical exam can be directed by the history

Physical Exam

- In a patient with abnormal level of consciousness
  - Primary survey
    - Cardiovascular (murmers, JVP, muffled heart sounds)
    - Respiratory exam (crackles, wheezes),
    - Abdominal exam
    - Rectal and vaginal exam
    - Skin and mucous membranes
    - Neurologic examination

Laboratory Tests

- CBC, Electrolytes, Creatinine/BUN, glucose
- +/- Lactate
- +/- Capillary blood sugar
- +/- Cardiac Enzymes
- Blood Cultures
- Beta HCG
- +/- Cross Match

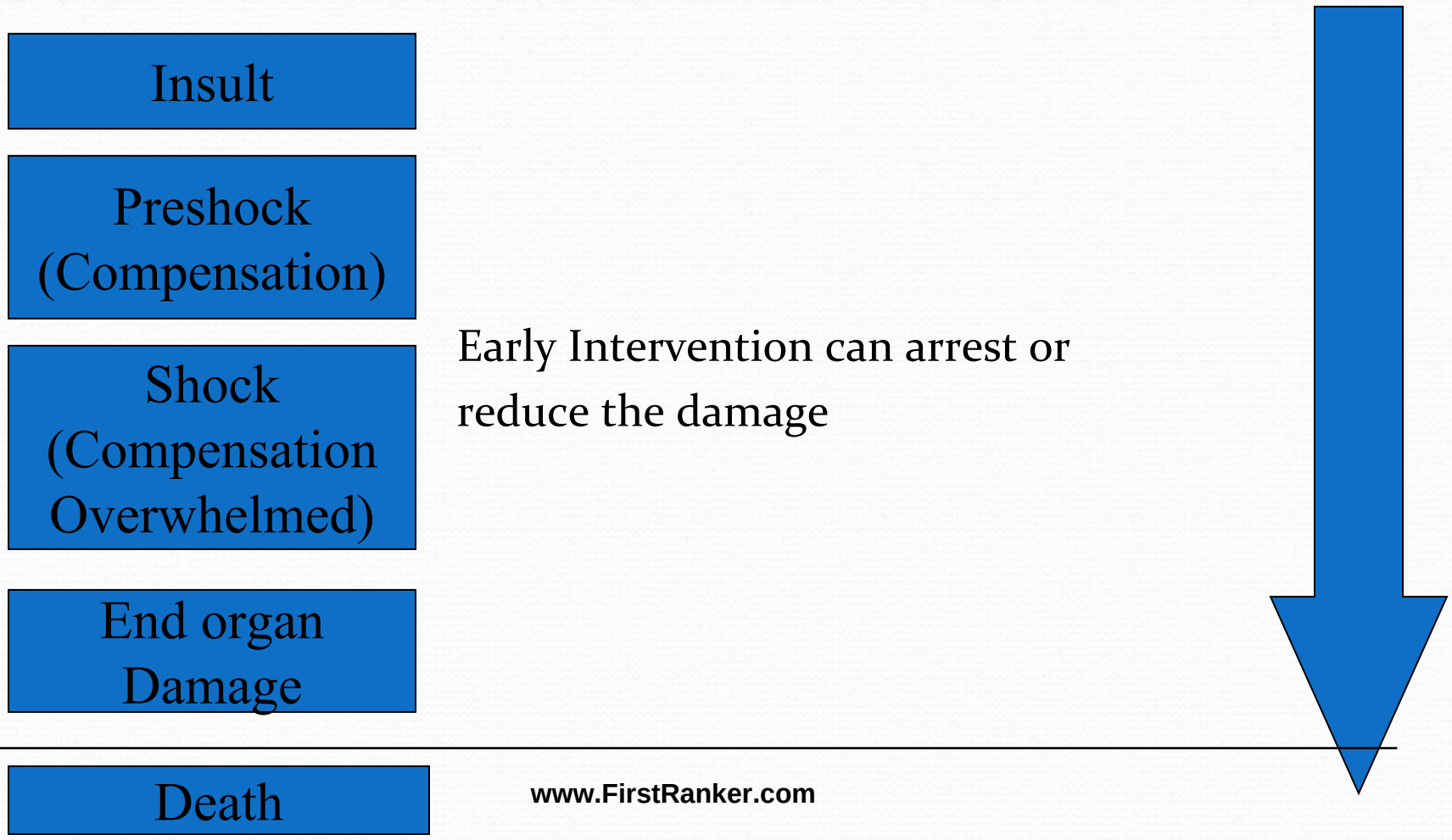
Other investigations

- ECG
- Urinalysis
- CXR
- +/- Echo
- +/- FAST

Treatment

- **Start treatment immediately**

Stages of Shock



Treatment

- ABC's "5 to 15"
  - Airway
  - Breathing
  - Circulation
  - Put the patient on a monitor if available
- Treat underlying cause

Treatment: Airway and Breathing

- Give oxygen

Treatment: Airway and Breathing

- Consider Intubation
  - Is the cause quickly reversible?
    - Generally no need for intubation
  - 3 reasons to intubate in the setting of shock
    - Inability to oxygenate
    - Inability to maintain airway
    - Work of breathing

Treatment: Circulation

- Treat the **early** signs of shock (Cold, clammy? Decreased capillary refill? Tachycardic? Agitated?)
- DO NOT WAIT for hypotension

Treatment: Circulation

- Start IV +/- Central line (or Intraosseous)
- Do Blood Work +/- Blood Cultures

Treatment: Circulation

- Fluids - 20 ml/kg bolus x 3
  - Normal saline
  - Ringer's lactate

Back to Case 1

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- Brought in by friends after a fight - he was kicked in the abdomen
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- HR 92, BP 126/72, SaO2 95%, RR 26

Case 1

- On examination
  - Extremely agitated
  - Clammy and cold
  - Heart exam - normal
  - Chest exam - good air entry
  - Abdomen - bruised, tender, distended
  - No other signs of trauma

Case 1: Management

- Hemorrhagic (Hypovolemic Shock)
  - ABC's
    - Monitors
    - O2
    - Intubate?
    - IV lines x 2, Fluid boluses, Call for Blood - O type
    - Blood work including cross match
  - Treat Underlying Cause

Case 1: Management

- Hemorrhagic (Hypovolemic Shock)
  - ABC's
    - Monitors
    - O2
    - Intubate?
    - IV lines x 2, Fluid boluses, Call for Blood - O type
    - Blood work including cross match
  - Treat Underlying Cause
    - Give Blood
    - Call the surgeon stat
    - If the patient does not respond to initial boluses and blood products - take to the Operating Room

Blood Products

- Use blood products if no improvement to fluids
  - PRBC 5-10 ml/kg
    - O- in child-bearing years and O+ in everyone else
  - +/- Platelets

Case 2

- 23 year old woman
- Has been fatigued and short of breath for a few days
- She fainted and family brought her in
- They tell you she has a heart problem

Case 2

- HR 132, BP 76/36, SaO2 88%, RR 30, Temp 36.3
- Appearance - obtunded
- Cardiovascular exam - S1, S2, irregular, holosytolic murmer, JVP is 5 cm , no edema
- Chest - bilateral crackles, accessory muscle use
- Abdomen - unremarkable
- Rest of exam is normal

Stages of Shock

Insult

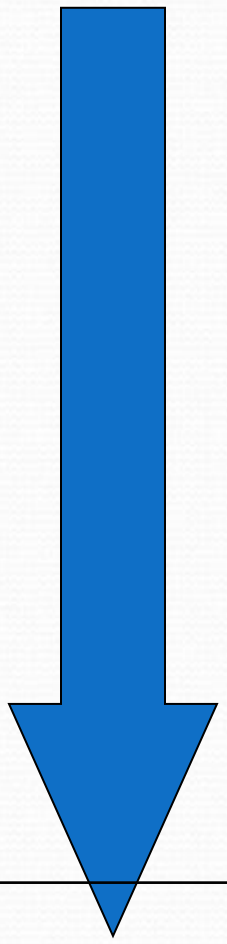
Preshock (Compensation)

Shock (Compensation Overwhelmed)

End organ Damage

Death

What stage is she at?



Case 2: Management

- Cardiogenic Shock
  - ABC's
    - Monitors
    - O2
    - IV and blood work
    - ECG - Atrial Fibrillation, rate 130's
  - Treat Underlying Cause

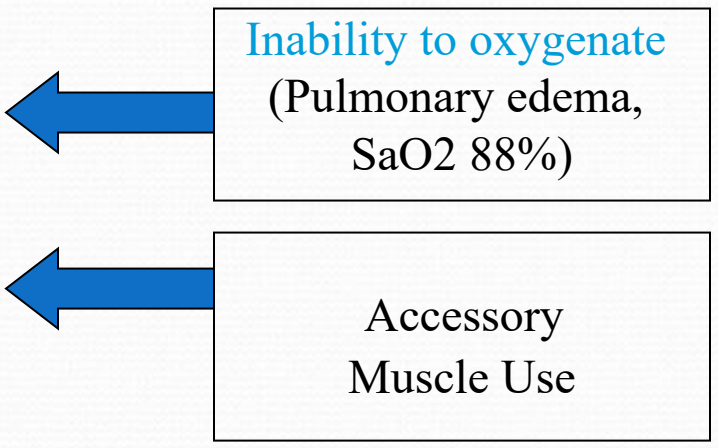
Case 2: Management

- Cardiogenic Shock
  - ABC's
    - Monitors
    - O2
    - IV and blood work
    - Intubate?
    - ECG - Atrial Fibrillation, rate 130's
  - Treat Underlying Cause

Case 2: Why would you intubate?

- Is the cause quickly reversible? **UNLIKELY**

- 3 reasons to intubate in the setting of shock
  - Inability to oxygenate
  - Inability to maintain airway
  - Work of breathing



Case 2: Why Intubate?

- Strenuous use of accessory respiratory muscles (i.e. work of breathing) can:
  - Increase O2 consumption by 50-100%
  - Decrease cerebral blood flow by 50%

Case 2: Management

- Cardiogenic Shock
  - ABC's
    - Monitors
    - O_2
    - IV and blood work
    - Intubate?
    - ECG - Atrial Fibrillation, rate 130's
  - Treat Underlying Cause

Case 2: Management

- Cardiogenic Shock
  - Treat Underlying Cause
    - Lasix
    - Atrial Fibrillation - Cardioversion? Rate control?
    - Inotropes - Dobutamine +/- Norepinephrine (Vasopressor)
    - Look for precipitating causes - infectious?

Vasopressors in Cardiogenic Shock

- Norepinephrine
- Dopamine
- Epinephrine
- Phenylephrine

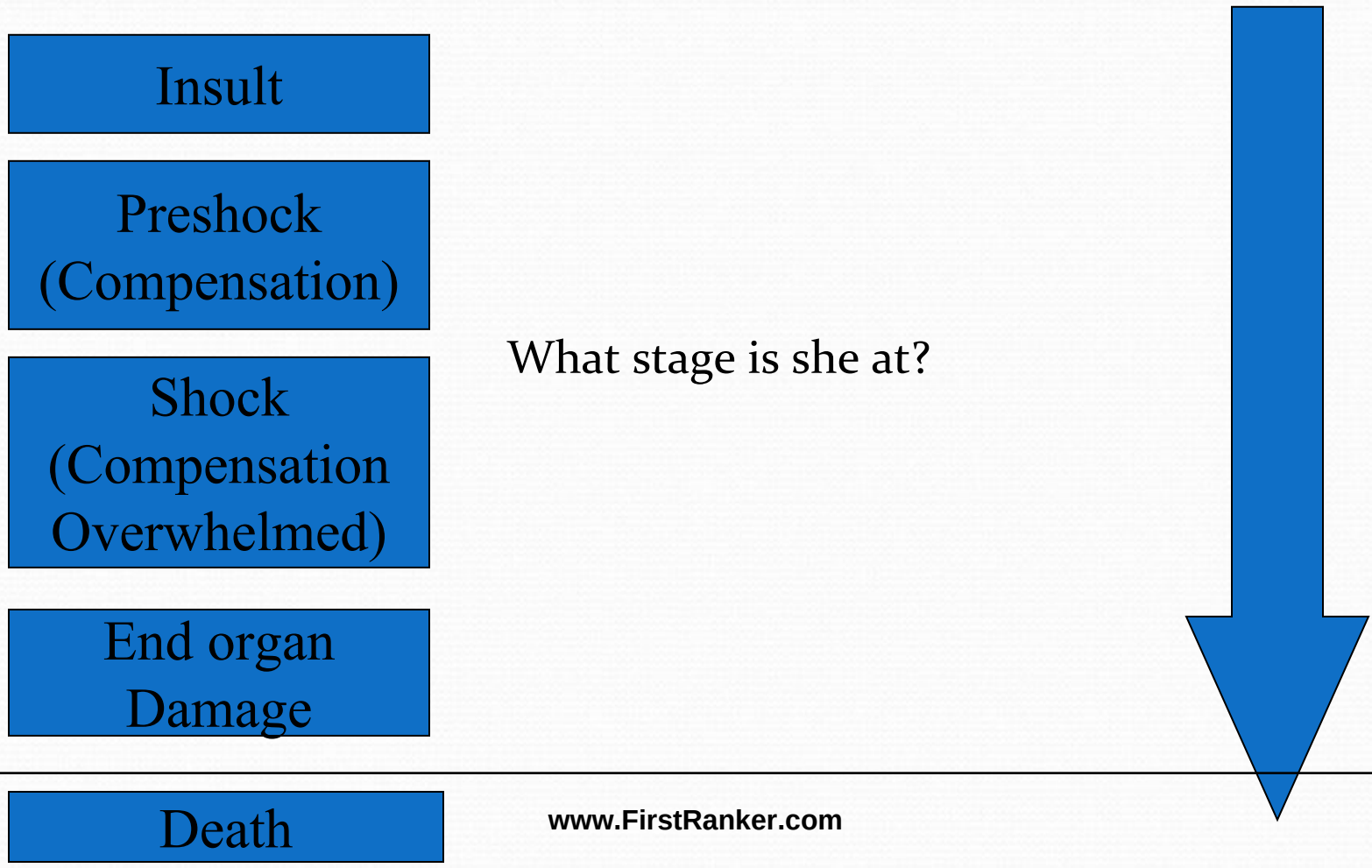
Case 3

- 36 year old woman
- Pedestrian hit by a car
- She is brought into the hospital 2 hrs after accident
- Short of breath
- Has been complaining of chest pain

Case 3

- HR 126, SBP 82, SaO2 70%, RR 36, Temp 35
- Obtunded, Accessory muscle use
- Trachea is deviated to Left
- Heart - distant heart sounds
- Chest - decreased air entry on the right, broken ribs, subcutaneous emphysema
- Abdominal exam - normal
- Apart from bruises and scrapes no other signs of trauma

Stages of Shock



Case 3: Management

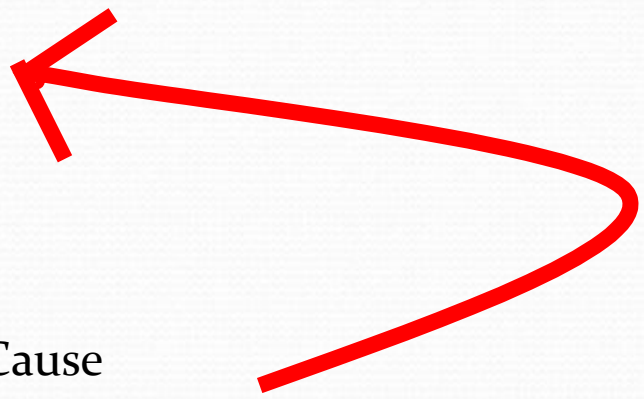
- Obstructive Shock
  - ABC's
    - Monitors
    - O2
    - IV
    - Intubate?
    - BW
  - Treat Underlying Cause

Case 3: Management

- Obstructive Shock
  - ABC's
    - Monitors
    - O2
    - IV
    - Intubate?
    - BW
  - Treat Underlying Cause
    - Needle thoracentesis
    - Chest tube
    - CXR

Case 3: Management

- Obstructive Shock
  - ABC's
    - Monitors
    - O2
    - IV
    - Intubate?
    - BW
  - Treat Underlying Cause
    - **Needle thoracentesis**
    - Chest tube
    - CXR
    - **Intubate if no response**



Case 3

- You perform a needle thoracentesis - hear a hissing sound
- Chest tube is inserted successfully
- HR 96, BP 100/76, SaO2 96% on O2, RR 26
- You resume your clinical duties, and call the surgeon

Case 3

- 1 hr has gone by
- You are having lunch
- The nurse puts her head through the door to tell you about another patient at triage, and as she is leaving "By the way, that woman with the chest tube, is feeling not so good" and leaves.

Case 3

- You are back at the bedside
- The patient is obtunded again
- Pale and Clammy
- HR 130, BP 86/52, SaO2 96% on O2
- Chest tube seems to be working
- Trachea is midline
- Heart - Normal
- Chest - Good air entry
- Abdomen - decreased bowel sounds, distended

Combined Shock

- Different types of shock can coexist
- Can you think of other examples?

Monitoring

- Vitals - BP, HR, SaO_2
- Mental Status
- Urine Output (> 1-2 ml/kg/hr)
- When something changes or if you do not observe a response to your treatment -

  re-examine the patient

Can we measure cell hypoxia?

- Lactate - we already talked about - a surrogate
- Venous Oxygen Saturation - more direct measure

Venous Oxygen Saturation

- Hg carries O2
- A percentage of O2 is extracted by the tissue for cellular respiration
- Usually the cells extract < 30% of the O2

Venous Oxygen Saturation

- Svo2 = Mixed venous oxygen saturation
  - Measured from pulmonary artery by Swan-Ganz catheter.
    - Normal > 65%
- Scvo2 = Central venous oxygen saturation
  - Measured through central venous cannulation of SVC or R Atrium - i.e. Central Line
    - Normal > 70%

PART 2

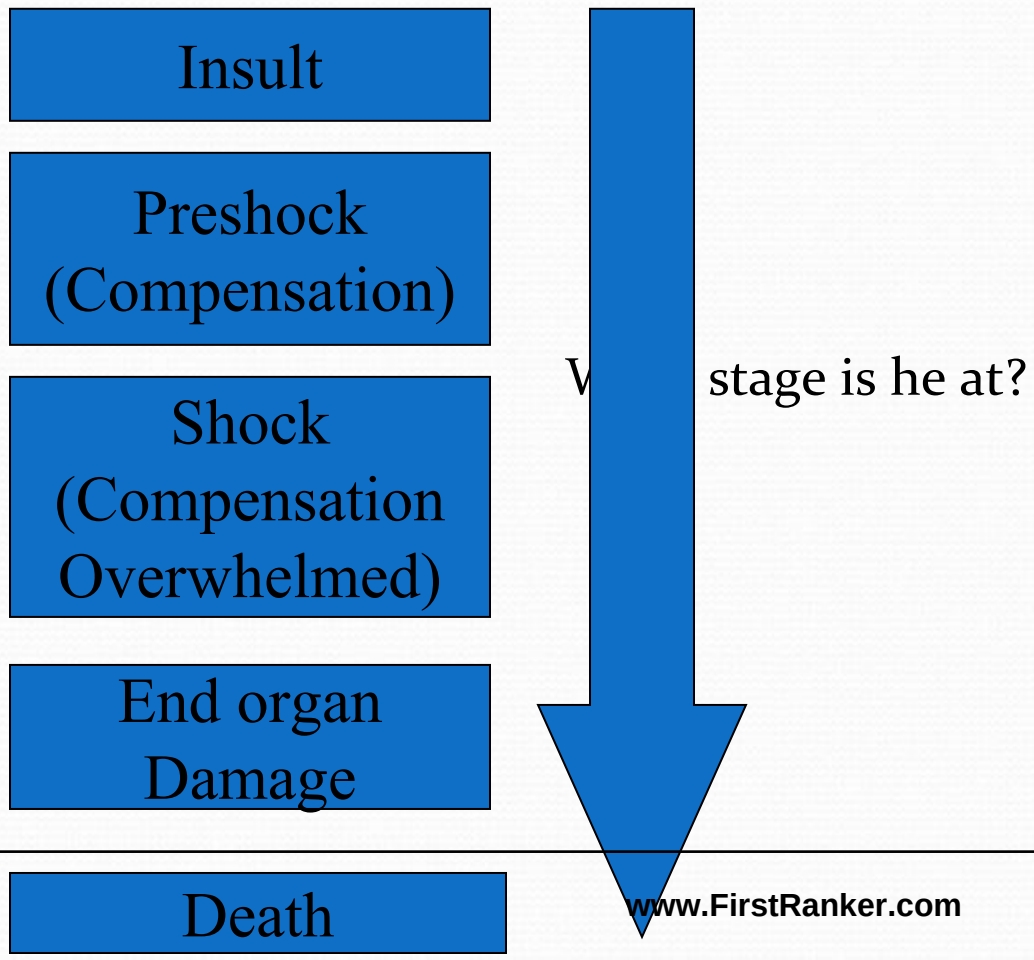
Case 4

- 40 year old male
- RUQ abdominal pain, fever, fatigued for 5-6 days
- No past medical history

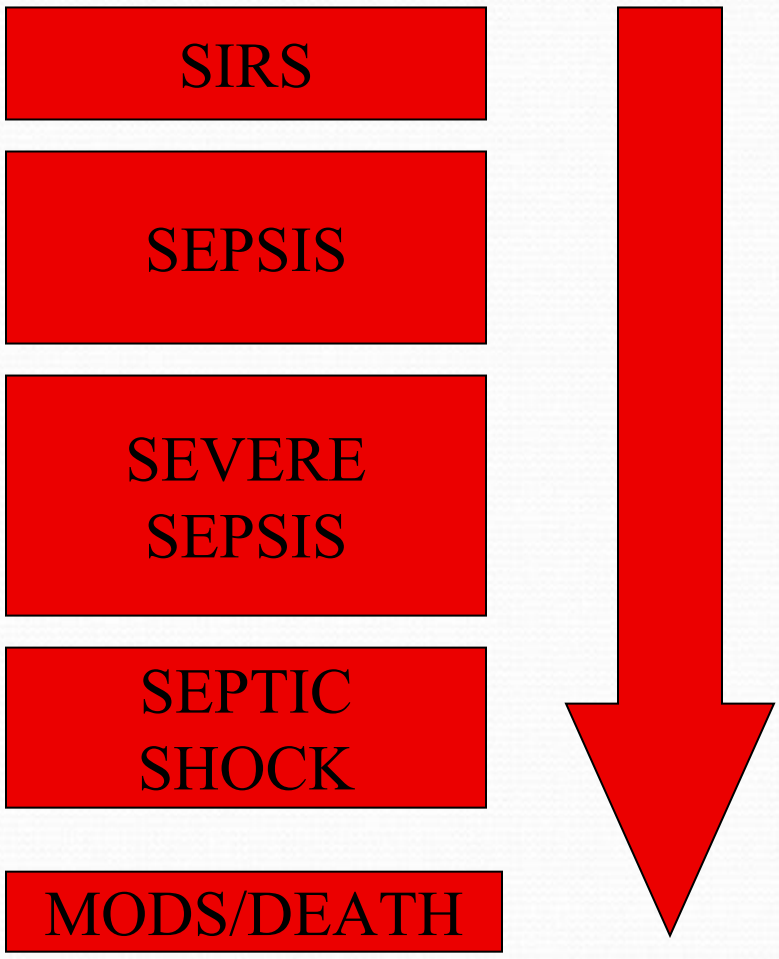
Case 4

- HR 110, BP 100/72, SaO2 96%, T 39.2, RR 26
- Drowsy
- Warm skin
- Heart - S1, S2, no Murmers
- Chest - good A/E x 2
- Abdomen - decreased bowel sound, tender RUQ

Stages of Shock



Stages of **Sepsis**



Definitions of Sepsis

- **Systemic Inflammatory Response Syndrome (SIRS)** – 2 or > of:
  - -Temp > 38 or < 36
  - -RR > 20
  - -HR > 90/min
  - -WBC >12,000 or <6,000 or more than 10% immature bands

Definitions of Sepsis

- **Sepsis** – SIRS with **proven or suspected microbial source**

- **Severe Sepsis** – sepsis with one or more signs of organ dysfunction or hypoperfusion.

Definitions of Sepsis

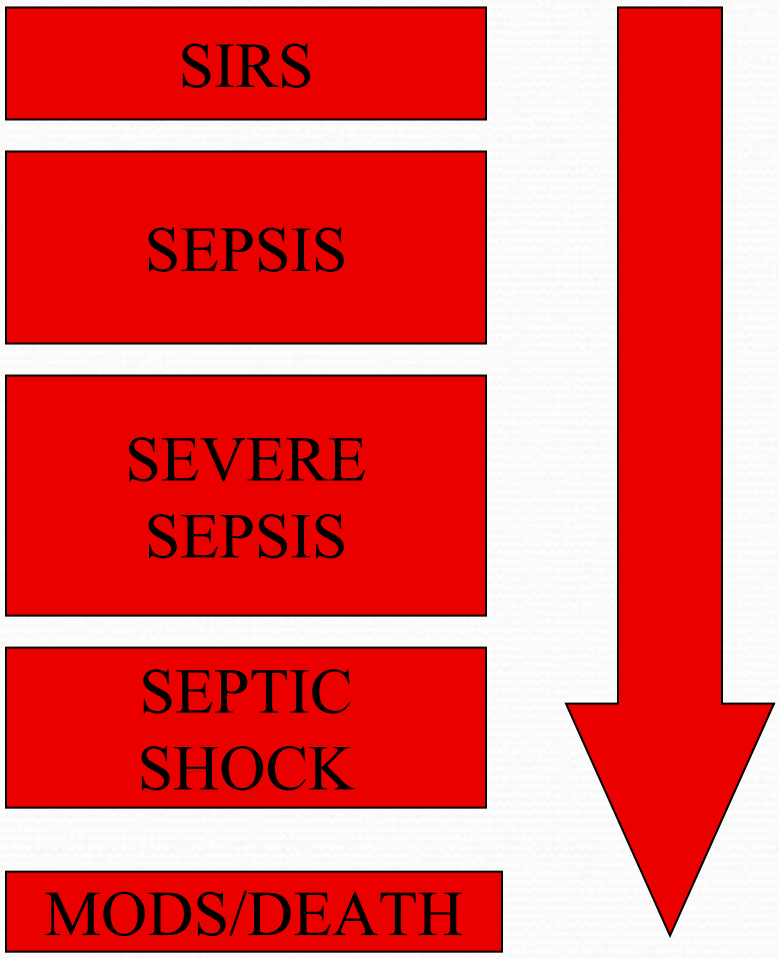
- **Septic shock** = Sepsis + Refractory hypotension

  -Unresponsive to initial fluids 20-40cc/kg – Vasopressor dependant

- **MODS** – multiple organ dysfunction syndrome

  -2 or more organs

Stages of Sepsis



Pathophysiology

- Complex pathophysiologic mechanisms

Pathophysiology

- Inflammatory Cascade:
  - Humoral, cellular and Neuroendocrine (TNF, IL etc)
- Endothelial reaction
  - Endothelial permeability = leaking vessels
- Coagulation and complement systems
  - Microvascular flow impairment

Pathophysiology

- End result = Global Cellular Hypoxia

Focus of Infection

- Any focus of infection can cause sepsis
  - Gastrointestinal
  - GU
  - Oral
  - Skin

Risk Factors for Sepsis

- Infants
- Immunocompromised patients
  - Diabetes
  - Steroids
  - HIV
  - Chemotherapy/malignancy
  - Malnutrition
- Sickle cell disease
- Disrupted barriers
  - Foley, burns, central lines, procedures

Back to Case 4

- HR 110, BP 100/72, SaO2 96%, T 39.2, RR 20
- Drowsy
- Warm skin
- Heart - S1, S2, no Murmers
- Chest - good A/E x 2
- Abdomen - decreased bowel sound, tender RUQ

Case 4: Management

- Distributive Shock (SEPSIS)
  - ABC's
    - Monitors
    - O2
    - IV fluids 20 cc/kg x 3
    - Intubate?
    - BW
  - Treat Underlying Cause

Resuscitation in Sepsis

- **Early goal directed therapy - Rivers et al NEJM 2001**
  - Used in pt's who have: an infection, 2 or more SIRS, have a systolic < 90 after 20-30cc/ml or have a lactate > 4.
  - Emergency patients by emergency doctors
  - Resuscitation protocol started early - 6 hrs

Resuscitation in Sepsis: EGDT

- The theory is to normalize…
  - Preload - 1st
  - Afterload - 2nd
  - Contractility - 3rd

BACK TO OUR EQUATION

MAP = CO x SVR

(HR x Stroke volume)

Preload

Contractility

Afterload

BACK TO OUR EQUATION

MAP = CO x SVR

(HR x Stroke volume)

Preload

Contractility

Afterload

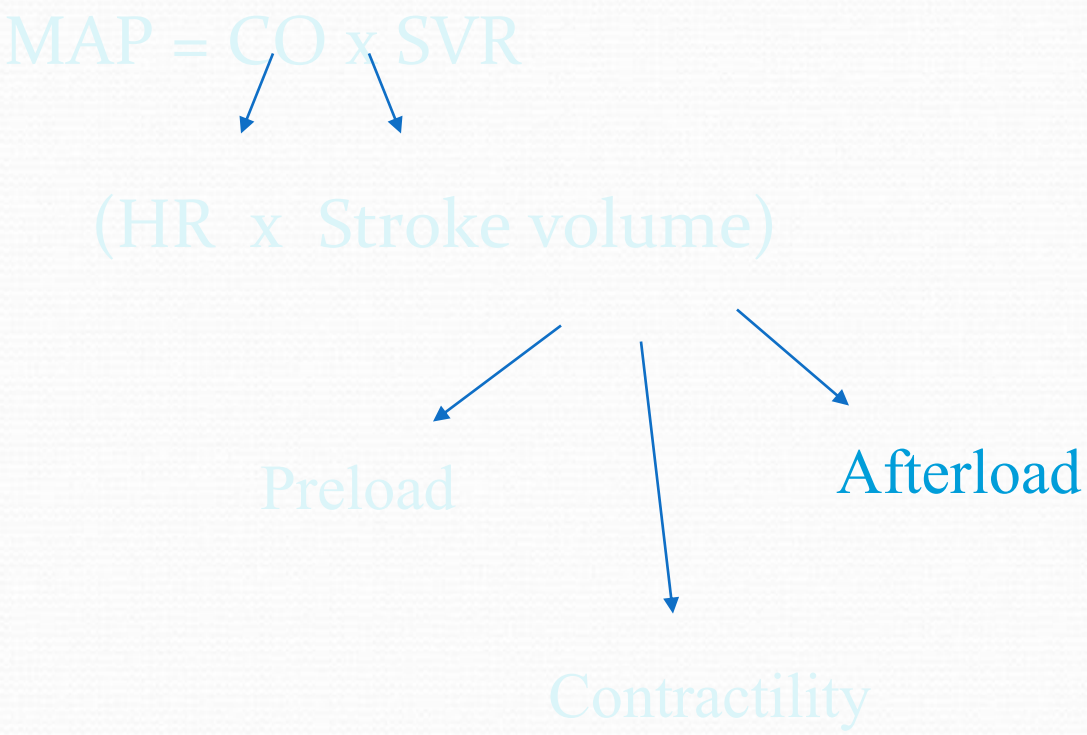
Preload

- Dependent on intravascular volume
  - If depleted intravascular volume (due to increased endothelial permeability) - PRELOAD DECREASES
- Can use the CVP as measurement of preload
  - Normal = 8-12 mm Hg

Preload

- How do you correct decreased preload (or intravascular volume)
  - Give fluids
  - Rivers showed an average of 5 L in first 6 hours
- What is the end point?

BACK TO OUR EQUATION



Afterload

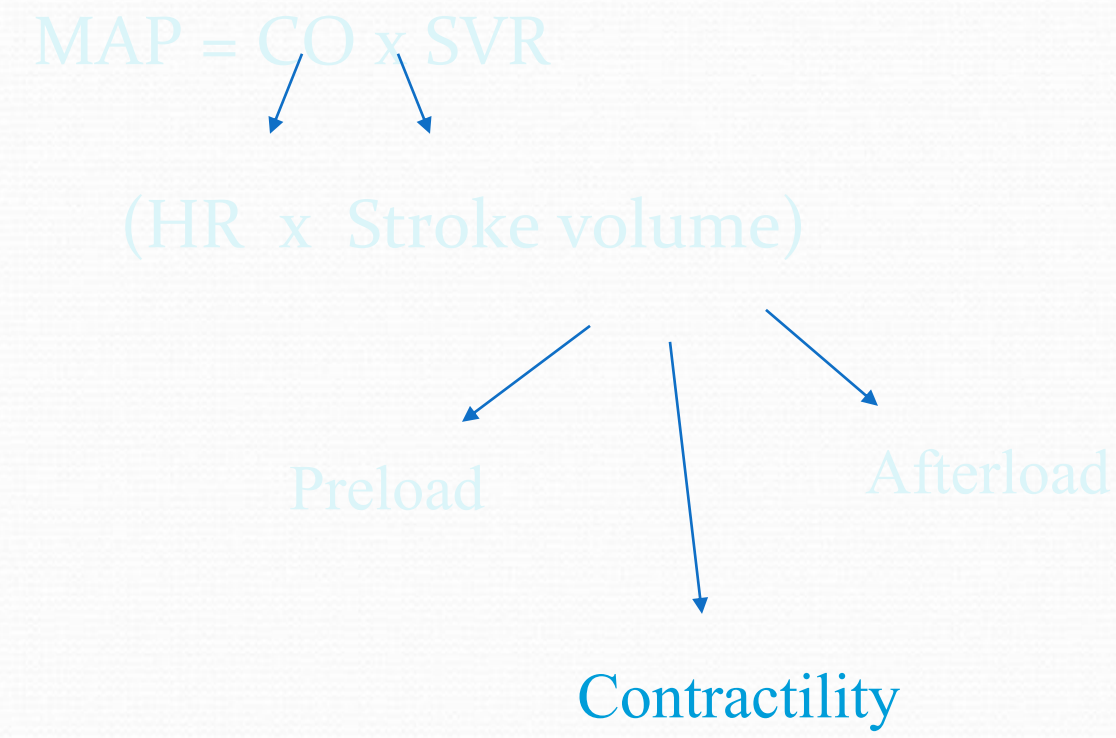
- **Afterload determines tissue perfusion**
  - Using the MAP as a surrogate measure - Keep between 60-90 mm Hg
  - In sepsis afterload is decreased d/t loss of vessel tone

Afterload

- **How do you correct decreased afterload?**

- Use vasopressor agent
  - Norepinephrine
  - Alternative Dopamine or Phenylpehrine

BACK TO OUR EQUATION



Contractility

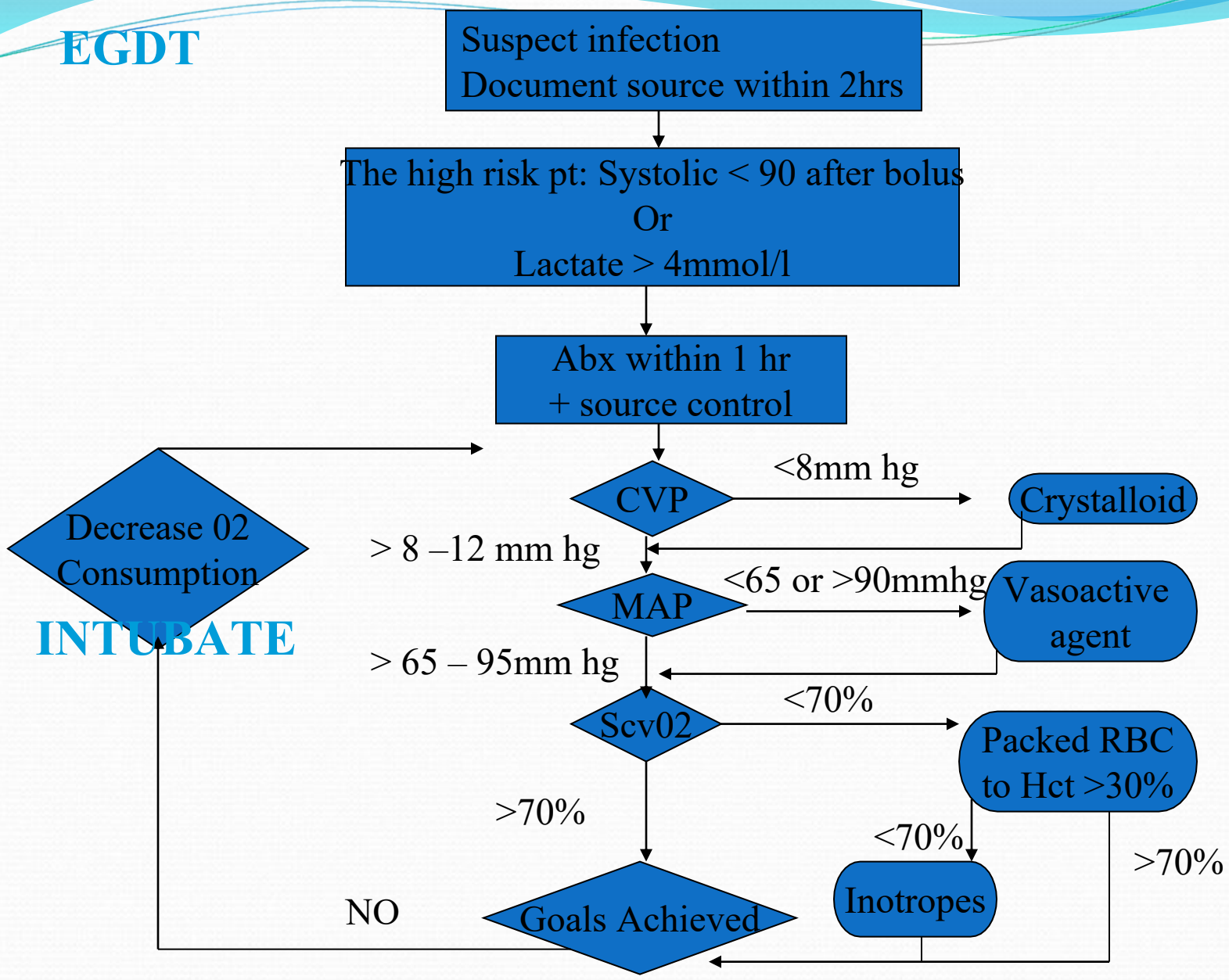
- Use the central venous oxygen saturation (ScvO2) as a surrogate measure
- Shown to a be a surrogate for cardiac index
- Keep > 70%

Contractility

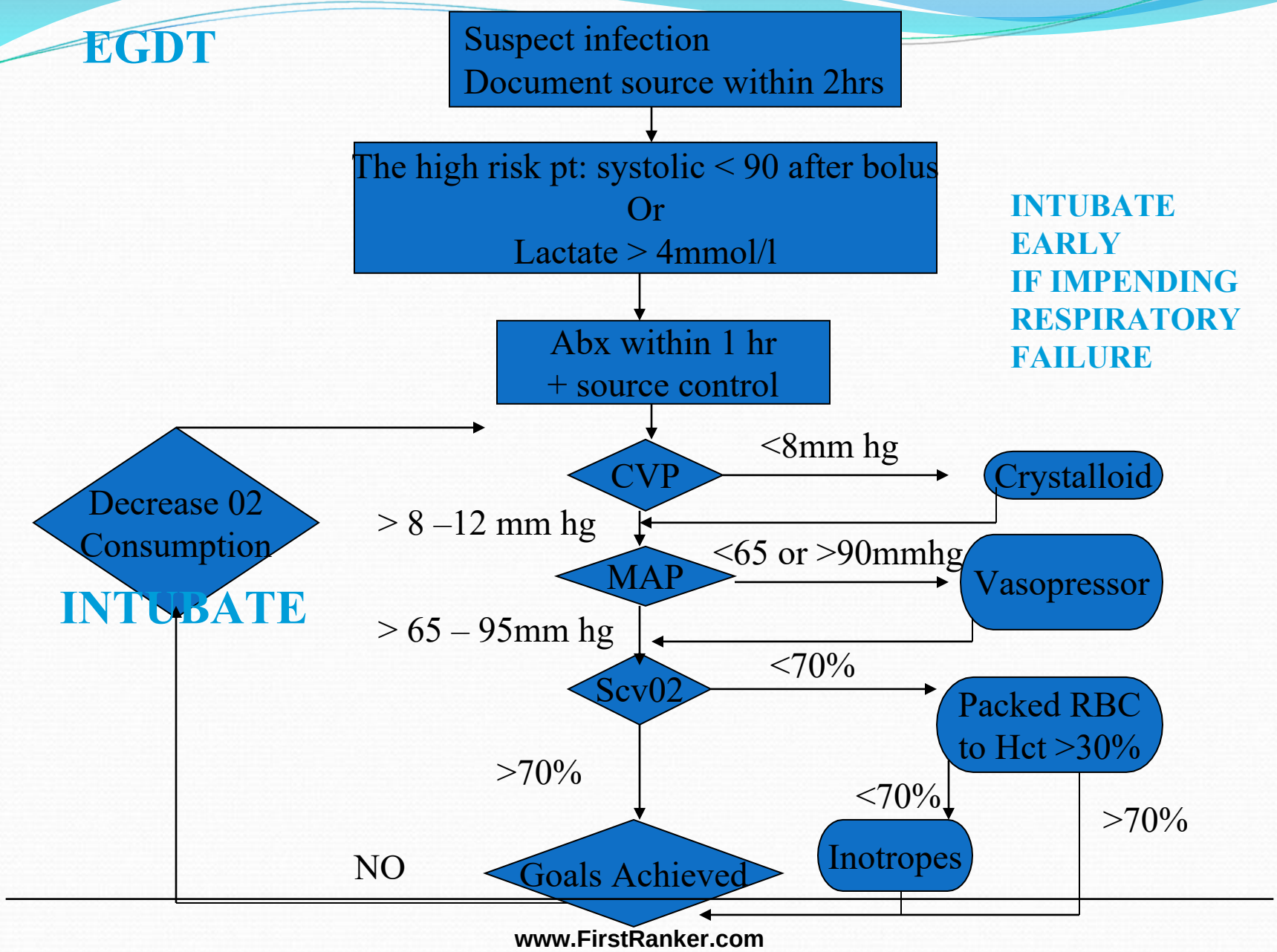
How to improve ScvO2 > 70%?

- Optimize arterial O2 with non-rebreather
- Ensure a hematocrit > 30 (Transfuse to reach a hematocrit of > 30)
- Use Inotrope - Dobutamine 2.5ug/kg per minute and titrated (max 20ug/kg)
- **Respiratory Support** - Intubation (Don't forget to sedate and paralyze)

EGDT



EGDT



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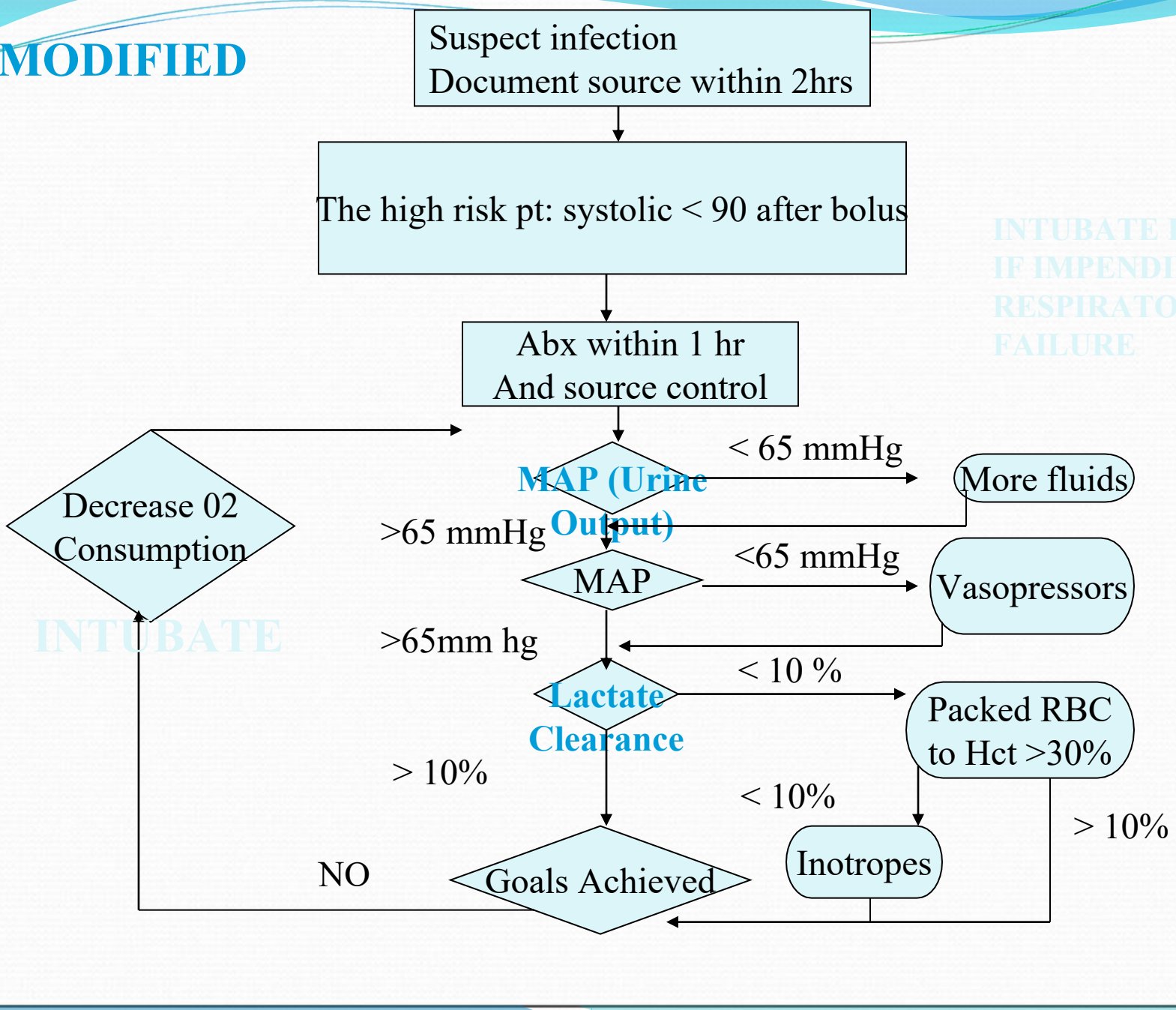

Case 4: Management

- Distributive Shock (SEPSIS)
  - ABC's
    - Monitors
    - O_2
    - IV fluids 20 cc/kg
    - Intubate
    - BW

  - Treat Underlying Cause
    - **Acetaminophen**
    - Antibiotics - **GIVE EARLY**
    - Source control - the 4 D's = Drain, Debride, Device removal, Definitive Control

Antibiotics

- **Early Antibiotics**

  Within 3-6hrs can reduce mortality - 30%

  Within 1 hr for those severely sick

  Don't wait for the cultures – treat empirically then change if need.

Other treatments for severe sepsis:

- Glucocorticoids
- Glycemic Control
- Activated protein C

Couple of words about Steroids in sepsis…

- **New Guidelines for the management of sepsis and septic shock = Surviving Sepsis Campaign**
  - Grade 2C – consider steroids for septic shock in patients with BP that responds poorly to fluid resuscitation and vasopressors

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Concluding Remarks

- Know how to distinguish different types of shock and treat accordingly
- Look for **early** signs of shock
- SHOCK = hypotension

Concluding Remarks

- Choose cost effective and high impact interventions
- **Do not need central lines and ScvO2** measurements to make an impact!!

Concluding Remarks

- ABC's "5 to 15"
  - Can't intubate?
    - Give oxygen
    - Develop algorithms for bag valve mask ventilation
    - Treat fever to decrease respiratory rate
  - Treat early with fluids - need lots of it!!

Concluding Remarks

- Monitor the patient
  - Do not need central venous pressure and ScvO2
  - Use the HR, MAP, mental status, urine output
  - Lactate clearance?

Concluding Remarks

- Start antibiotics within an hour!
  - Do not wait for cultures or blood work

- A 22 year old man was driving drunk and without his seatbelt fastened when he was involved in a
- single-vehicle automobile accident. When attended by EMT personnel, no information was
- available about the time of the accident. He was found agitated and complaining of abdominal
- pain. His airway was patent. At the scene, he was breathing at 20 per minute with a blood
- pressure of 90/60 and a pulse of 130. He was placed in a hard cervical collar and on a back board
- and transported to your emergency room. Upon arrival his vital signs are the same, with a
- temperature of 360C. His abdomen is markedly distended. His hands and feet are cold, his legs
- mottled. A nasogastric tube reveals green liquid. A urinary catheter reveals dark yellow urine. His
- hemoglobin is 7. His abdominal lavage reveals gross blood.

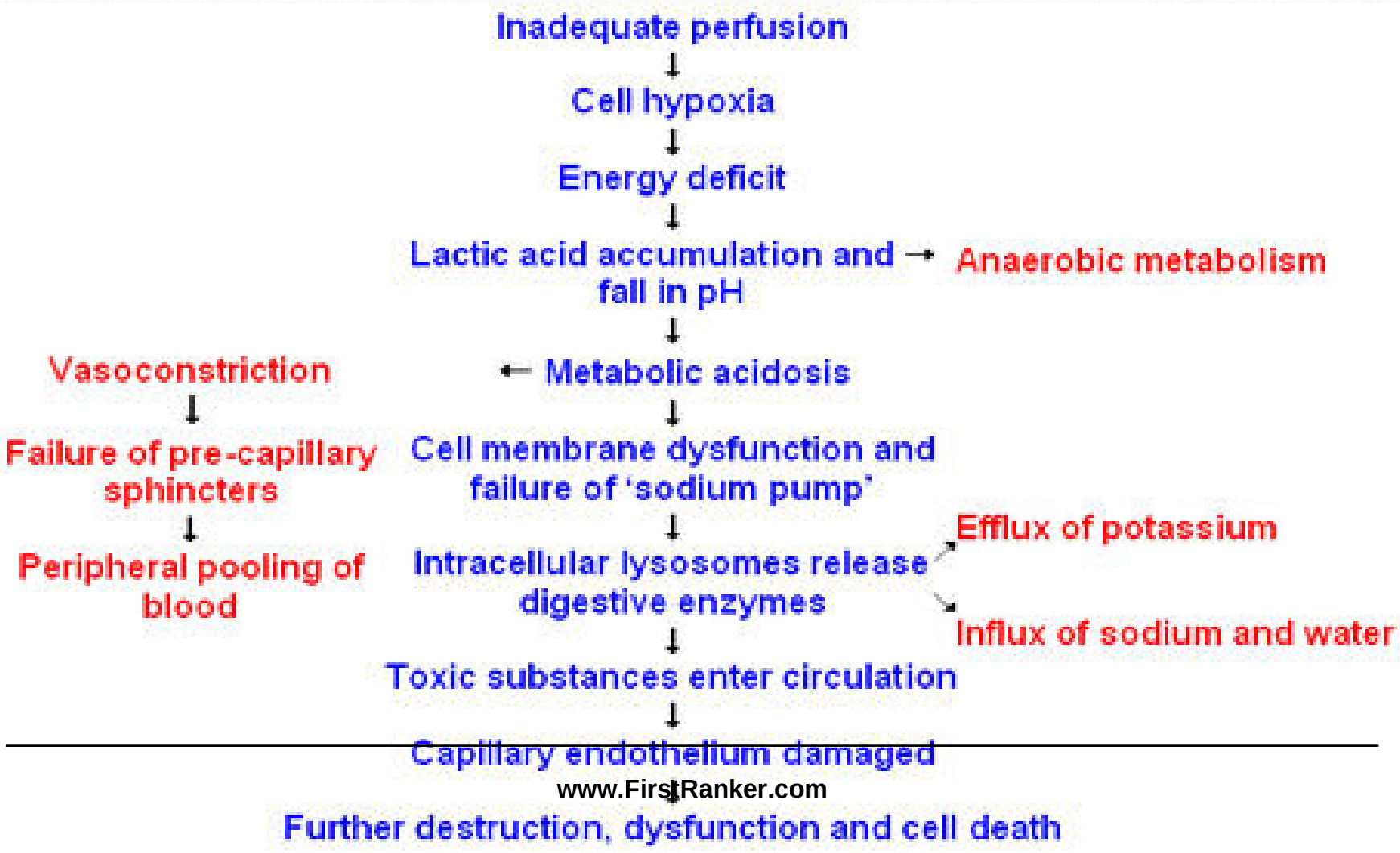
- **Study Questions:**
- What type of shock does this patient exhibit?
- What would be the cardiac output (low, normal, high)?
- What would be the systematic resistance (low, normal, high)?
- What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal,
- high)?
- What therapy would reverse the shock?

- A 65 year old man with known coronary artery disease (myocardial infarct three years earlier,
- currently taking a beta blocker) is admitted with acute left lower quadrant pain of six hours duration.
- His blood pressure is 90/50, pulse 120, respirations 18, temperature 390C. He is flushed with
- warm hands and warm feet, his legs are pink. Physical examination reveals findings consistent
- with peritonitis in the left lower quadrant.

- **Study Questions:**
- What type of shock does this patient exhibit?
- What would be the cardiac output (low, normal, high)?
- What would be the systemic resistance (low, normal, high)?
- What would be the central venous and/or pulmonary capillary occlusion pressure (low, normal,
- high)?
- What therapy would reverse the shock?

- A 35 year old man dove into three feet of water at a swimming pool, did not emerge and was
- rescued by friends who performed CPR. When the EMTs arrived they found the patient to have a
- blood pressure of 80/50, pulse 100, and no spontaneous respirations, although he was opening his
- eyes. They began ambu bag assistance of respiration and placed a hard cervical collar. He was
- placed on a back board and transported to your emergency room. Upon arrival he has the same
- vital signs with warm hands and feet and pink extremities.

STAGES OF SHOCK



Types of Shock and Their Causes

Cardiogenic Shock

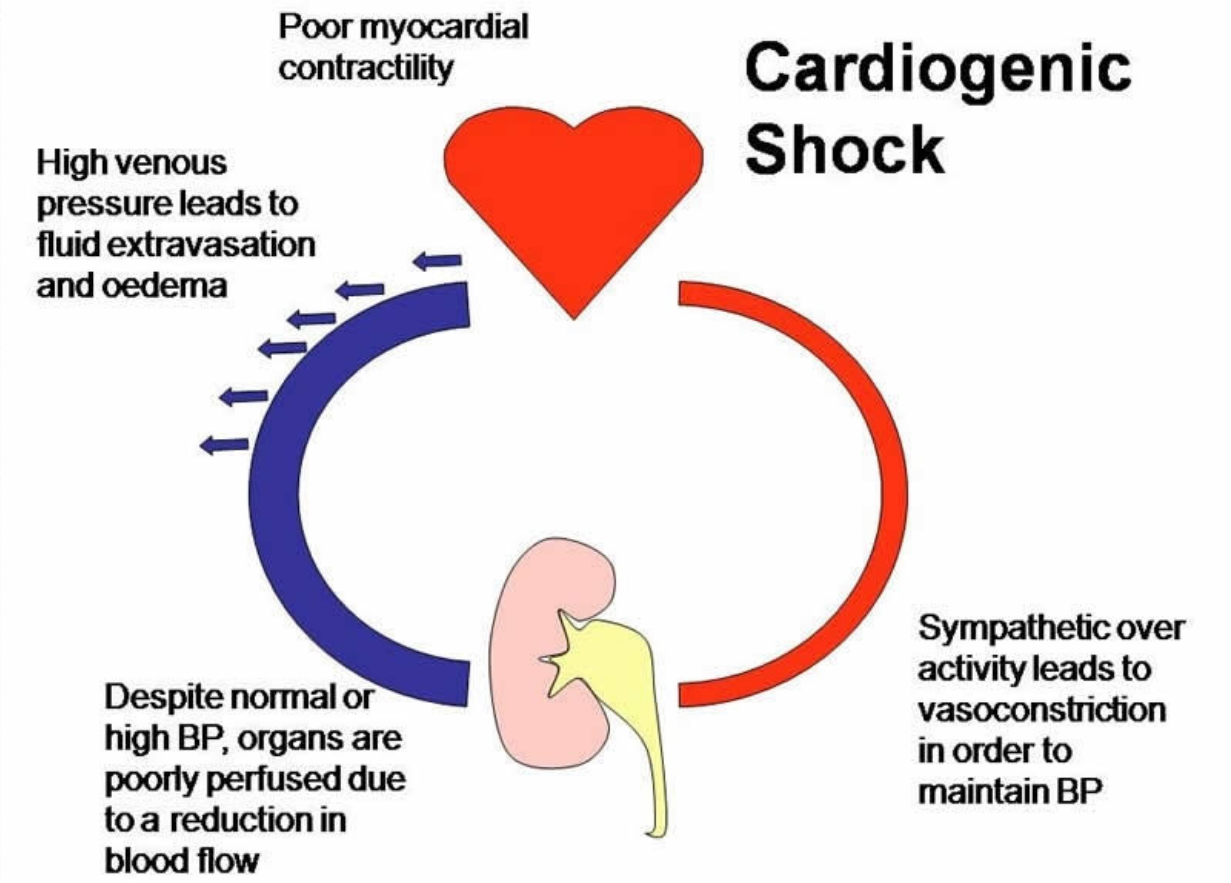
- Pump failure
- Heart's output depends on
  - How often it beats (heart rate)
  - How hard it beats (contractility)
- Rate or contractility problems cause pump failure

Cardiogenic Shock

- Causes
  - Acute myocardial infarction
  - Very low heart rates (bradycardias)
  - Very high heart rates (tachycardias)

Why would a high heart rate caused decreased output?

Hint: Think about when the heart fills.



Neurogenic Shock

- Loss of peripheral resistance
- Spinal cord injured
- Vessels below injury dilate

What happens to the pressure in a closed system if you increase its size?

Hypovolemic Shock

- Loss of volume
- Causes
  - Blood loss: trauma
  - Plasma loss: burns
  - Water loss: Vomiting, diarrhea, sweating, increased urine, increased respiratory loss

If a system that is supposed to be closed leaks, what happens to the pressure in it?

HYPOVOLEMIC SHOCK - CAUSES

HYPOVOLEMIC SHOCK RESULTS FROM EXCESSIVE FLUID LOSS AND INADEQUATE CIRCULATING VOLUME. THE CIRCULATORY SYSTEM COLLAPSES AND ORGANS SUCH AS THE KIDNEYS, BRAIN AND LUNGS ARE DEPRIVED OF BLOOD. THE HEART RATE BECOMES RAPID IN AN ATTEMPT TO MEET THE DEMANDS OF THE ORGANS FOR BLOOD. THE PATIENT BECOMES SHORT OF BREATH AND RESPIRATIONS INCREASE IN AN EFFORT TO MEET THE BODY'S NEEDS.

CAUSES OF HYPOVOLEMIC SHOCK INCLUDE:

BLOOD LOSS - BLUNT AND PENETRATING TRAUMA, CAUSING MASSIVE BLOOD LOSS, CARDIAC TAMPONADE
GI LOSS - DEHYDRATION DUE TO DIABETIC KETOACIDOSIS, EXCESSIVE VOMITING, DIARRHEA
GU LOSS - DIABETES INSIPIDUS, DIURETIC THERAPY WHICH RESULTED IN MASSIVE FLUID LOSS.

Psychogenic Shock

- Simple fainting (syncope)
- Caused by stress, pain, fright
- Heart rate slows, vessels dilate
- Brain becomes hypoperfused
- Loss of consciousness occurs

What two problems combine to produce hypoperfusion in psychogenic shock?

Septic Shock

- Results from body's response to bacteria in bloodstream
- Vessels dilate, become "leaky"

What two problems combine to produce hypoperfusion in septic shock?

Anaphylactic Shock

- Results from severe allergic reaction
- Body responds to allergen by releasing histamine
- Histamine causes vessels to dilate and become "leaky"

What two problems combine to produce hypoperfusion in anaphylaxis?

OBSTRUCTIVE SHOCK

- In this situation the flow of blood is obstructed which impedes circulation and can result in circulatory arrest. Several conditions result in this form of shock.
  - Cardiac tamponade in which fluid in the pericardium prevents inflow of blood into the heart (venous return). Constrictive pericarditis, in which the pericardium shrinks and hardens, is similar in presentation.
  - Tension pneumothorax. Through increased intrathoracic pressure, bloodflow to the heart is prevented (venous return).
  - Massive pulmonary embolism is the result of a thromboembolic incident in the bloodvessels of the lungs and hinders the return of blood to the heart.
- Aortic stenosis hinders circulation by obstructing the ventricular outflow tract

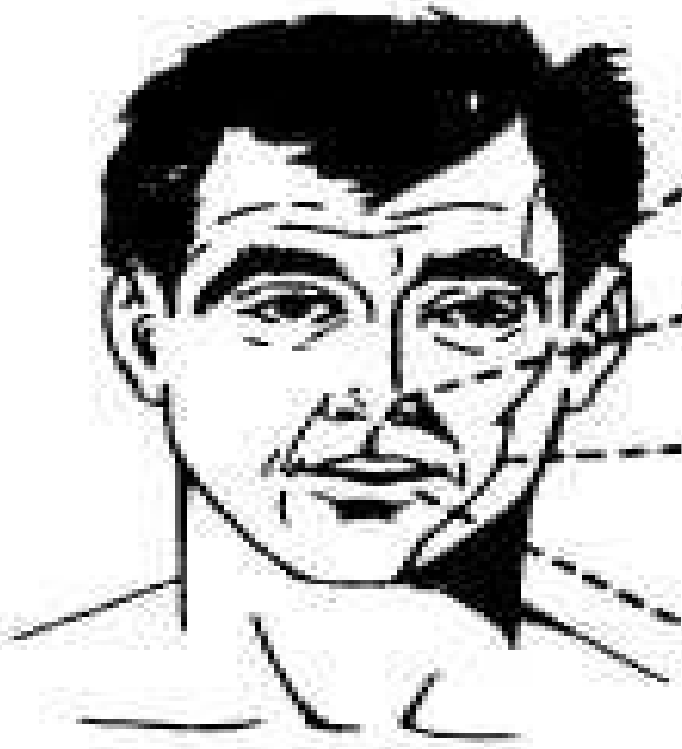
ENDOCRINE SHOCK

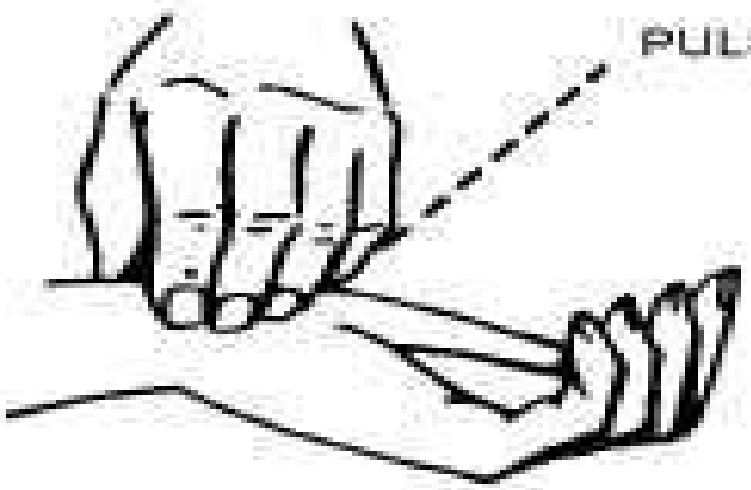
- Hypothyroidism, in critically ill patients, reduces cardiac output and can lead to hypotension and respiratory insufficiency.
- Thyrotoxicosis may induce a reversible cardiomyopathy.
- Acute adrenal insufficiency is frequently the result of discontinuing corticosteroid treatment without tapering the dosage. However, surgery and intercurrent disease in patients on corticosteroid therapy without adjusting the dosage to accommodate for increased requirements may also result in this condition.
- Relative adrenal insufficiency in critically ill patients where present hormone levels are insufficient to meet the higher demands .

Shock:
Signs and Symptoms

- Restlessness, anxiety
- Increased pulse rate
- Decreasing level of consciousness
- Dull eyes
- Rapid, shallow respirations
- Nausea, vomiting
- Thirst
- Diminished urine output

Why are these signs and symptoms present?
Hint: Think hypoperfusion





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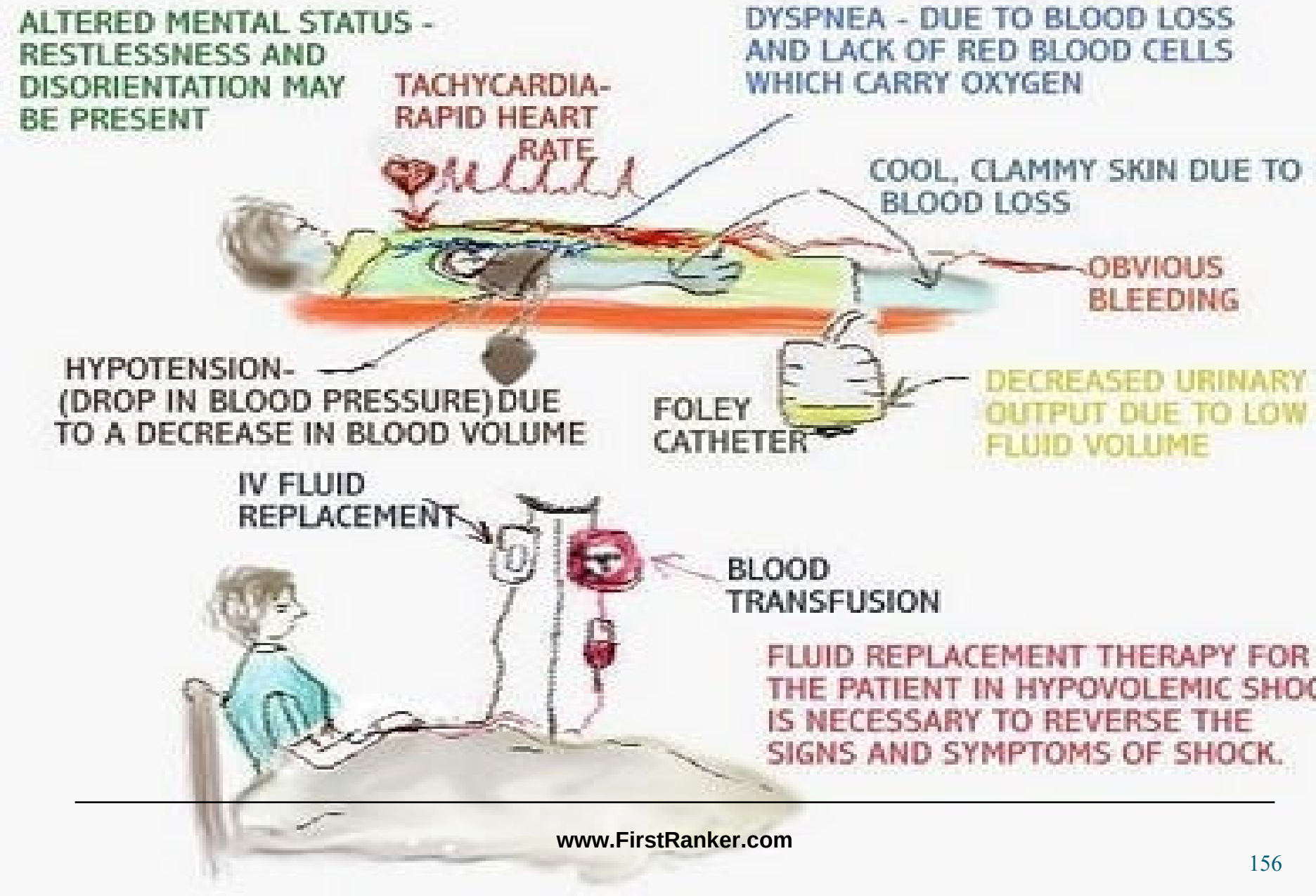
CLINICAL PICTURE OF A PATIENT IN HYPOVOLEMIC SHOCK



Shock: Signs and Symptoms

- Hypovolemia will cause
  - Weak, rapid pulse
  - Pale, cool, clammy skin
- Cardiogenic shock may cause:
  - Weak, rapid pulse or weak, slow pulse
  - Pale, cool, clammy skin
- Neurogenic shock will cause:
  - Weak, slow pulse
  - Dry, flushed skin
- Sepsis and anaphylaxis will cause:
  - Weak, rapid pulse
  - Dry, flushed skin

Can you explain the differences in the signs and symptoms?

- Patients with anaphylaxis will:
  - Develop hives (urticaria)
  - Itch
  - Develop wheezing and difficulty breathing (bronchospasm)

What chemical released from the body during an allergic reaction accounts for these effects?

Shock:
Signs and Symptoms

Shock is NOT the same thing as a low blood pressure!

A falling blood pressure is a LATE sign of shock!

Shock:
Signs and Symptoms

- Obscure/Less viewed symptom of shock
  - Drop in end tidal carbon dioxide ($ETCO_2$) level
  - Indicative of respiratory failure resulting in poor oxygenation, therefore, poor perfusion or Shock

Severity of shock

- ***Compensated shock***
- body's cardiovascular and endocrine compensatory responses reduce flow to non-essential organs to preserve preload and flow to the lungs and brain.
- Apart from a tachycardia and cool peripheries (vasoconstriction, circulating catecholamines) there may be no other clinical signs of hypovolaemia.

- ***Decompensation***
- Further loss of circulating volume overloads the body's compensatory mechanisms and there is progressive renal, respiratory and cardiovascular decompensation.
- In general, loss of around 15% of the circulating blood volume is within normal compensatory mechanisms.
- Blood pressure is usually well maintained and only falls after 30–40% of the circulating volume has been lost.

- ***Mild shock***
- Initially there is tachycardia, tachypnoea and a mild reduction in urine output and mild anxiety.
- Blood pressure is maintained although there is a decrease in pulse pressure.
- The peripheries are cool and sweaty with prolonged capillary refill times (except in septic distributive shock).

- ***Moderate shock***
- As shock progresses, renal compensatory mechanisms fail, renal perfusion falls and urine output dips below 0.5 ml kg–1h–1.
- There is further tachycardia and now the blood pressure starts to fall.
- Patients become drowsy and mildly confused.

- ***Severe shock***
- In severe shock there is profound tachycardia and hypotension.
- Urine output falls to zero and patients are unconscious with laboured respiration

Treatment

- Secure, maintain airway (ABC's)
- High concentration oxygen
- Assist ventilations
- Control obvious bleeding (consider TraumaDex®)
- Stabilize fractures
- Replace Fluids
- Prevent loss of body heat
- Transport rapidly to appropriate facility

Treatment

- Elevate lower extremities 8 to 12 inches in hypovolemic shock (Trendelenberg Position)
- Do NOT elevate the lower extremities in cardiogenic shock

Why the difference in management?

Treatment

- Administer nothing by mouth, even if the patient complains of thirst

TREATMENT

- Immediate intervention, even before a diagnosis is made.
- Re-establishing perfusion to the organs is the primary goal.
- Restoring and maintaining the blood circulating volume ensuring oxygenation and blood pressure are adequate, achieving and maintaining effective cardiac function, and preventing complications.)
- Intubation and mechanical ventilation may be necessary.

- In hypovolemic shock, caused by bleeding, it is necessary to immediately control the bleeding and restore the casualty's blood volume by giving infusions of isotonic crystalloid solutions. Blood transfusions, packed red blood cells (RBCs), Albumin (or other colloid solutions), or fresh-frozen plasma are necessary for loss of large amounts of blood (e.g. greater than 20% of blood volume), but can be avoided in smaller and slower losses. Hypovolemia due to burns, diarrhea, vomiting, etc. is treated with infusions of electrolyte solutions that balance the nature of the fluid lost. Sodium is essential to keep the fluid infused in the extracellular and intravascular space whilst preventing water intoxication and brain swelling. Metabolic acidosis (mainly due to lactic acid) accumulates as a result of poor delivery of oxygen to the tissues, and mirrors the severity of the shock. It is best treated by rapidly restoring intravascular volume and perfusion as above. Inotropic and vasoconstrictive drugs should be avoided, as they may interfere in knowing blood volume has returned to normal

TREATMENT

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- Hypovolemia due to burns, diarrhea, vomiting, etc. is treated with infusions of electrolyte solutions that balance the nature of the fluid lost.

TREATMENT

- Opinion varies on the type of fluid used in shock. The most common are:
- Crystalloids - Such as sodium chloride (0.9%), or Lactated Ringer's. Dextrose solutions which contain free water are less effective at re-establishing circulating volume, and promote hyperglycaemia.
- Colloids - For example, polysaccharide (Dextran), polygeline (Haemaccel), succunylated gelatin (Gelofusine) and hetastarch (Hepsan). Colloids are, in general, much more expensive than crystalloid solutions and have not conclusively been shown to be of any benefit in the initial treatment of shock.
- Combination - Some clinicians argue that individually, colloids and crystalloids can further exacerbate the problem and suggest the combination of crystalloid and colloid solutions.
- Blood - Essential in severe hemorrhagic shock, often pre-warmed and rapidly infused.

TREATMENT-HAEMORRHAGIC SHOCK

- It is to be noted that NO plain water should be given to the patient at any point, as the patient's low electrolyte levels would easily cause water intoxication, leading to premature death.
- An isotonic or solution high in electrolytes should be administered if intravenous delivery of recommended fluids is unavailable.

TREATMENT-HAEMORRHAGIC SHOCK

- Vasoconstrictor agents have no role in the initial treatment of hemorrhagic shock, due to their relative inefficacy in the setting of acidosis.
- Definitive care and control of the hemorrhage is absolutely necessary, and should not be delayed.

TREATMENT-CARDIOGENIC SHOCK

- In cardiogenic shock, depending on the type of myocardal infarction, one can infuse fluids or in shock refractory to infusing fluids, inotropic agents.
- Inotropic agents, which enhance the heart's pumping capabilities, are used to improve the contractility and correct the hypotension.
- Should that not suffice, an intra-aortic balloon pump can be considered (which reduces the workload for the heart and improves perfusion of the coronary arteries) or a left ventricular assist device (which augments the pump-function of the heart.)

TREATMENT CARDIOGENIC SHOCK

- The main goals of the treatment of cardiogenic shock are the re-establishment of circulation to the myocardium, minimising heart muscle damage and improving the heart's effectiveness as a pump.
- This is most often performed by percutaneous coronary intervention and insertion of a stent in the culprit coronary lesion or sometimes by cardiac bypass.

TREATMENT

- The main way to avoid the deadly consequence of death is to make the blood pressure rise again with:
- fluid replacement with intravenous infusions
- use of vasopressing drugs (e.g. to induce vasoconstriction);
- use of anti-shock trousers that compress the legs and concentrate the blood in the vital organs (lungs, heart, brain).
- use of blankets to keep the patient warm - metallic PET film emergency blankets are used to reflect the patient's body heat back to the patient

TREATMENT

- In distributive shock caused by sepsis the infection is treated with antibiotics
- Supportive care is given (i.e. inotropica, mechanical ventilation, renal function replacement).
- Anaphylaxis is treated with adrenaline to stimulate cardiac performance and corticosteroids to reduce the inflammatory response.
- In neurogenic shock because of vasodilation in the legs, one of the most suggested treatments is placing the patient in the Trendelenburg position, thereby elevating the legs and shunting blood back from the periphery to the body's core. However, since bloodvessels are highly compliant, and expand as result of the increased volume locally, this technique does not work. More suitable would be the use of vasopressors.

TREATMENT

- In obstructive shock, the only therapy consists of removing the obstruction.
- Pneumothorax or haemothorax is treated by inserting a chest tube.
- Pulmonary embolism requires thrombolysis (to reduce the size of the clot), or embolectomy (removal of the thrombus).
- Tamponade is treated by draining fluid from the pericardial space through pericardiocentesis.

TREATMENT

- In endocrine shock the hormone disturbances are corrected.
- Hypothyroidism requires supplementation by means of levothyroxine.
- In hyperthyroidism the production of hormone by the thyroid is inhibited through thyreostatica, i.e. methimazole (Tapazole) or PTU (propylthiouracil).
- Adrenal insufficiency is treated by supplementing corticosteroids

TREATMENT

Definitive therapy

Hypovolaemic shock
- Maintain or increase intravascular volume.
- Decrease any future fluid loss via i.v. fluid regimen.
- Give supplementary O_2 therapy.

Cardiogenic shock
- O_2 therapy.
- Administration of cardiac drugs.
- Increase heart's pumping action through medication.

Septic shock
- Restore intravascular volume via i.v. fluids.
- Give supplemental oxygen therapy.
- Identify and control source of infection.
- Administer antibiotics.
- Remove risk factors for infection.

Anaphylactic shock
- Identify and remove causative antigen.
- Administer counter-mediators such as anti-histamine.
- O_2 therapy and i.v. fluid replacement.

Supportive therapy

- Maintain airway and respiratory effort.
- Maintain the cardiac pump.
- Restore metabolic equilibrium.
- Reverse metabolic acidosis.

Monitoring for patients in shock

Minimum
- Electrocardiogram
- Pulse oximetry
- Blood pressure
- Urine output

Additional modalities
- Central venous pressure
- Invasive blood pressure
- Cardiac output
- Base deficit and serum lactate

PROGNOSIS

- The prognosis of shock depends on the underlying cause and the nature and extent of concurrent problems. Hypovolemic, anaphylactic and neurogenic shock are readily treatable and respond well to medical therapy. Septic shock however, is a grave condition and with a mortality rate between 30% and 50%. The prognosis of cardiogenic shock is even worse.

Bleeding

Identification of External Bleeding

- Arterial Bleed
  - Bright red
  - Spurting
- Venous Bleed
  - Dark red
  - Steady flow
- Capillary Bleed
  - Dark red
  - Oozing

What is the physiology that explains the differences?

Control of External Bleeding

- Direct Pressure
  - gloved hand
  - dressing/bandage
- Elevation
- Arterial pressure points

Arterial Pressure Points

- Upper extremity: Brachial
- Lower extremity: Femoral

Control of External Bleeding

- Splinting
  - Air splint
  - Pneumatic antishock garment (MAST)

Control of External Bleeding

- Tourniquets
  - Final resort when all else fails
  - Used for amputations - sometimes
  - 3-4” wide
  - Write “TK” and time of application on forehead of patient
  - Notify other personnel

Control of External Bleeding

- Tourniquets
  - Do not loosen or remove until definitive care is available
  - Do not cover with sheets, blankets, etc.

Epistaxis

- Nosebleed
- Common problem

Epistaxis

- Causes
  - Fractured skull
  - Facial injuries
  - Sinusitis, other URIs
  - High BP
  - Clotting disorders
  - Digital insertion (nose picking)

Epistaxis

- Management
  - Sit up, lean forward
  - Pinch nostrils together
  - Keep in sitting position
  - Keep quiet
  - Apply ice over nose

Internal Bleeding

- Can occur due to:
  - Trauma
  - Clotting disorders
  - Rupture of blood vessels
  - Fractures (injury to nearby vessels)

Internal Bleeding

Can result in rapid progression to hypovolemic shock and death

Internal Bleeding

- Assessment
  - Mechanism?
  - Signs and symptoms of hypovolemia without obvious external bleeding

Internal Bleeding

- Signs and Symptoms
  - Pain, tenderness, swelling, discoloration at injury site
  - Bleeding from any body orifice

Internal Bleeding

- Signs and Symptoms
  - Vomiting bright red blood or coffee ground material
  - Dark, tarry stools (melena)
  - Tender, rigid, or distended abdomen

Management

- Secure, maintain airway (ABC's)
- High concentration oxygen
- Assist ventilations
- Control obvious bleeding (consider TraumaDex®)
- Stabilize fractures
- Replace Fluids
- Prevent loss of body heat
- Transport rapidly to appropriate facility