COVID-19 Shock & Vasoactive Medications

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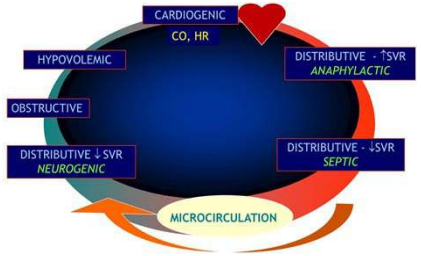
Shock

Inadequate tissue perfusion



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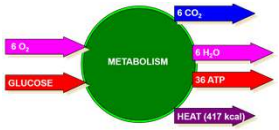
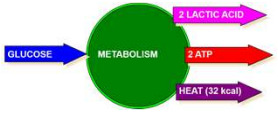
Cellular Metabolism

Aerobic



Anaerobic



- No oxygen = lactic acid production
- Reduction in number of ATP = reduced energy

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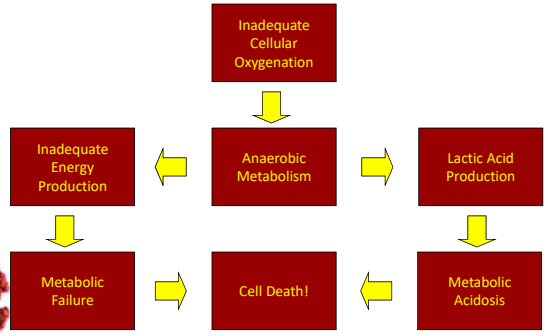
Anaerobic Metabolism – So What?



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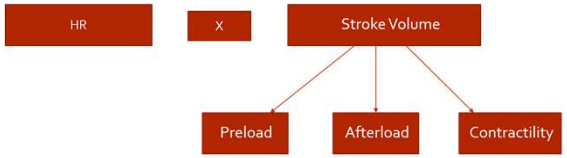
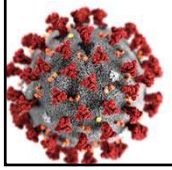
Cardiac Output Review





- Preload: amount of stretch at end of diastole (volume ready to be ejected)
- Afterload: resistant in which ventricle has to overcome to contract (vasoconstriction)
- Contractility: ability of the heart to contract

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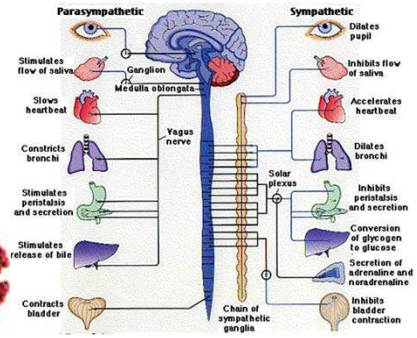
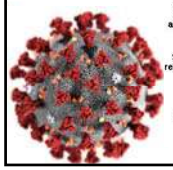
Autonomic Nervous System





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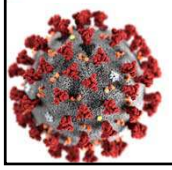
Effects of Sympathetic Nervous System Stimulation

Organ	Effect
Heart (muscle)	↑ force of contraction (+ inotropy)
Heart (rate)	↑ heart rate (+ chronotropy)
Peripheral vessels	Vasoconstriction
Pupils	Dilation
Sweat glands (cholinergic)	↑ secretion
Adrenal glands	↑ cortisol and medullary secretion
Bronchi	Dilation
Kidneys	↑ Renin secretion (↓ urine output)
Liver	Glycogenolysis (↑ blood sugar)



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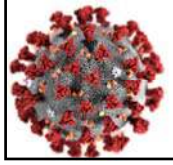
Stages of Shock



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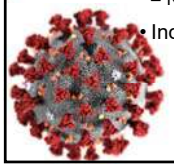
Compensated Shock

- Decreased cardiac output compensatory mechanisms
  - Tachycardia
- Activation of autonomic nervous system
  - Tachycardia
  - Vasoconstriction
- Activation of renin-angiotensin system
  - Vasoconstriction
  - Na/Water retention
- Increased rate and depth of respirations



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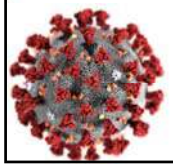
Compensated Shock – Clinical Findings

- Normal BP, narrow pulse pressure
- Sinus tachycardia
- Fast, deep respirations
- ↓ Urine Output
- ↑ Urine Specific Gravity
- Cool, clammy skin
- ↓ LOC
- Dilated pupils
- ↑ blood sugar
- Respiratory alkalosis with hypoxemia



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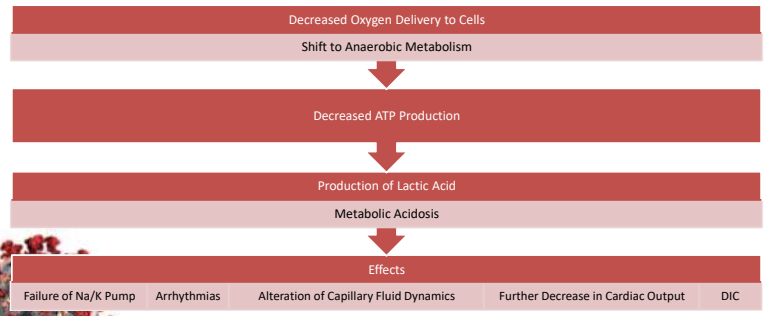
Decompensated (progressive) Shock



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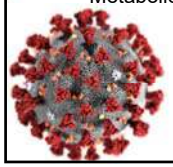
Decompensated (progressive) Shock – Clinical Findings

- ↓ BP with narrow pulse pressure
- Continued tachycardia
- Acute renal failure
- Continued decreasing LOC
- Interstitial pulmonary edema
- Peripheral edema
- Metabolic and respiratory acidosis with hypoxemia



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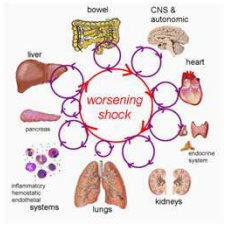
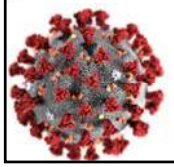
Irreversible Shock – Multiorgan Dysfunction Syndrome

- Microvascular and organ damage are now irreversible
- There is often a "last ditch" effort from the ischemic midbrain with an enormous discharge of endogenous catecholamines and this can create a last spike of sinus tachycardia





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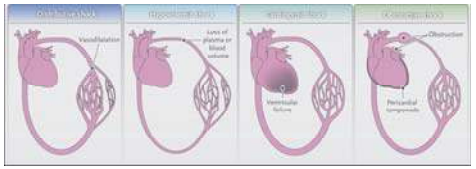
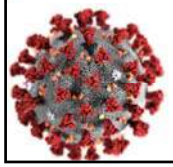
Classification of Shock

- Hypovolemic
- Distributive
- Cardiogenic
- Obstructive





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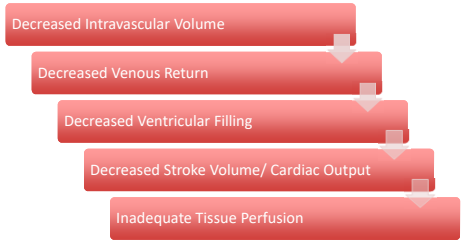
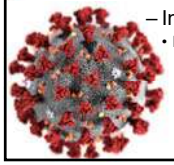
Hypovolemic Shock

Causes
- Dehydration
  - Vomiting
  - Diarrhea
  - Sweat
  - Decreased Oral Intake
  - Excessive Urine Output
- Hemorrhage
- Interstitial Fluid Loss
  - Burns





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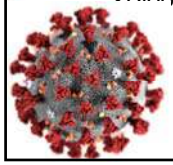
Hypovolemic Shock Treatment – Fluid Resuscitation

- Ideally, the volume that is lost is replaced

- Crystalloids
- Colloids
- Blood/Blood Products



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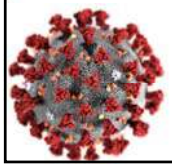
End Points to Fluid Resuscitation

- Traditional
- Invasive Hemodynamic Monitoring
- Metabolic Parameters



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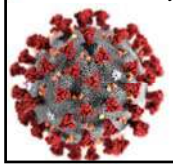
End Points to Fluid Resuscitation - Traditional

- Vital Signs
  - Blood pressure is **not** a good predictor of tissue perfusion
- Urinary Output
  - < 0.5 ml/kg/hour is an early sign of inadequate perfusion
- Mental Status
  - May also be affected by pre-existing conditions, alcohol, or drugs



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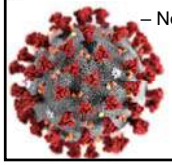
End Points to Fluid Resuscitation – Invasive Hemodynamic Monitoring

- CVP
  - Measures right ventricular preload (norm 2-6 mmHg)
- Wedge Pressure
  - Measures left ventricular preload (norm 8-12 mmHg)
- Cardiac Index
  - Normal 2.5-4 L/min/m^2



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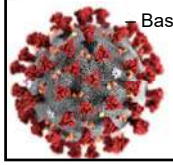
End Points to Fluid Resuscitation – Metabolic Parameters

- Lactate
  - Byproduct of inadequate tissue perfusion
  - Patients who lactate levels do not normalize have a higher mortality rate
  - Lactate > 4 mmol/L indicates widespread tissue hypoperfusion
- Base Deficit
  - Measures buffering capacity of the blood reflecting metabolism and depth of hemorrhagic shock
  - Base deficit > 6 mmol/L is a marker of severe injury



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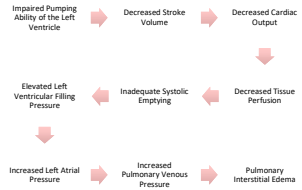
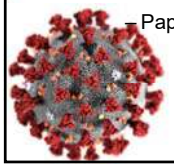
Cardiogenic Shock

- Severe dysfunction of the right or left ventricle that results in inadequate pumping
- Causes:
  - Myocardial Infarction
  - Cardiomyopathy
  - Blunt Cardiac Injury
  - Papillary Muscle Dysfunction





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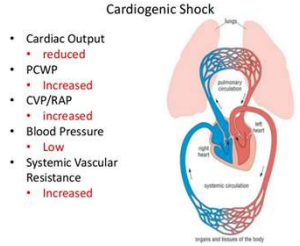
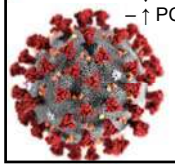
Cardiogenic Shock

- Symptoms:
  - ↓ BP (may be normal initially)
  - ↑ HR
  - ↑ RR
  - ↓ Urine Output
  - Normal Temp
  - Cool, Pale Skin
  - ↓ CO/CI
  - ↑ PCWP/PA Pressures





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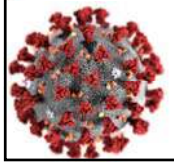
Cardiogenic Shock - Treatment

- Hemodynamic Support
- Identify & Treat Cause

Right Sided Failure	Left Sided Failure
Volume Expansion + Inotropes	Vasodilators Diuretics Afterload Reducers + Inotropes IABP Ventricular Assist Device



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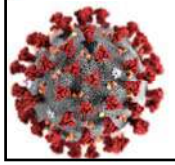
Obstructive Shock

- Hypoperfusion of tissue due to an obstruction in either the vasculature or heart

Cause	Treatment
Pregnancy	Roll patient to her side
Tension Pneumothorax	Chest Tube/Needle Decompression
Cardiac Tamponade	Pericardiocentesis
Pulmonary Embolism	Thrombolytics
Aortic Aneurysm	Surgical Intervention
Aortic Stenosis	Surgical Intervention
Excessive Positive End Expiratory Pressure	Readjust Ventilator Settings



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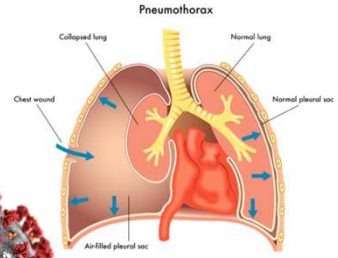
Obstructive Shock – Tension Pneumothorax



↑ intrathoracic pressure → Displacement of vena cava → ↓ preload → Obstruction to atrial filling → ↓ cardiac output

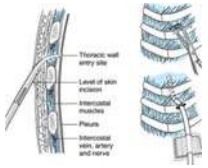
Obstructive Shock Treatment – Tension Pneumothorax

Needle Decompression

- Insert 14 g x 3.25 in angiocath into chest wall
  - 2nd intercostal space
  - Midclavicular line
  - Above 3rd rib (to avoid nerves, vein, artery that are located under ribs)

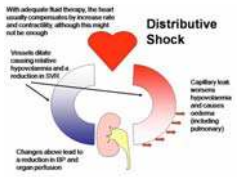
Chest Tube Placement

- Thoracostomy tube placed 4th - 5th intercostal space, midaxillary line



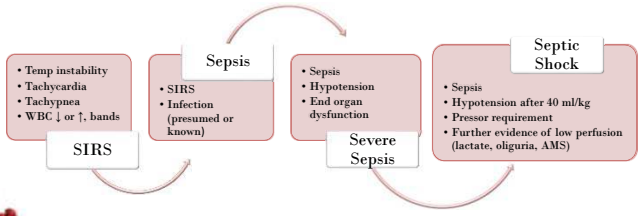
Distributive Shock

- Abnormality in the vascular system that produces a maldistribution of blood flow.
- Occurs when blood vessels dilate without subsequent increase in volume



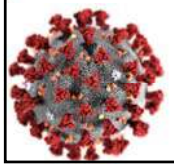
3 Types:
- Septic
- Neurogenic
- Anaphylactic

Septic Shock (Distributive)



Sepsis Guidelines

- Initial Resuscitation
- Screening for Sepsis
- Diagnosis
- Antimicrobial Therapy
- Source Control
- Fluid Therapy
- Vasoactive Medications
- Corticosteroids
- Blood Products
- Immunoglobulins
- Blood Purification
- Anticoagulants
- Mechanical Ventilation
- Sedation & Analgesia
- Glucose Control
- Renal Replacement Therapy
- Bicarbonate Therapy
- VTE Prophylaxis
- Stress Ulcer Prophylaxis
- Nutrition
- Goals of Care



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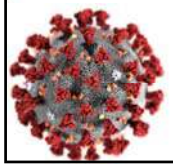
Septic Shock–
Initial Resuscitation

Intervention	Goal	Notes
Fluid	30 ml/kg crystalloid within 1st 3 hours	• Additional fluids guided by frequent reassessment • Albumin may be considered
Hemodynamic Assessment	Maintain MAP ≥ 65 mmHg if requiring vasopressors (use arterial line if need vasopressors)	• Norepinephrine – 1st choice • Vasopressin or Epinephrine next • Dobutamine for persistent hypoperfusion (adequate volume and vasopressors)
Lactate	< 4	• Marker to guide evidence of tissue hypoperfusion
Antibiotics	Empiric broad-spectrum therapy (typically 7-10 days)	• Therapy narrowed once pathogen identified
Corticosteroids	200 mg / day	• Only to be used if hemodynamic stability not achieved with adequate fluid/vaso
Hgb	≥ 7.0 g/dL	



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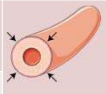
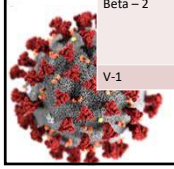
Vasopressors

Receptor	Graphic	Location	Function
Alpha – 1		Vascular Smooth Muscle	• Vasoconstriction • Increased blood pressure • Dilation of pupil • Increased contractility
Alpha – 2		Skeletal Blood Vessel	• Constriction of blood vessels in muscle tissue
Beta – 1		Heart	• Increased heart rate • Increased conductivity • Increased contractility
Beta – 2		Bronchi & Skeletal Blood Vessel	• Relaxation of bronchi • Vasodilation • Pupil dilation • Activation of glycogenolysis
V-1		Vascular Smooth Muscle	• Vasoconstriction



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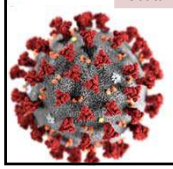
Vasoactive Medication Receptor Activity

Drug	Alpha – 1	Beta – 1	Beta – 2	V-1	Effects
Phenylephrine	*****	0	0	0	SVR ↑ ↑, CO ↔/↑
Norepinephrine	*****	***	**	0	SVR ↑ ↑, CO ↔/↑
Epinephrine	*****	****	***	0	CO ↑ ↑, SVR ↓ (low dose) SVR↑ (higher dose)
Vasopressin	0	0	0	*****	SVR ↑↑
Dobutamine	*	***	***	0	CO ↑, SVR ↓

MAP Goal 60-65 mmHg



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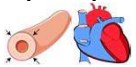
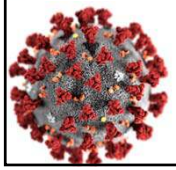
Norepinephrine



- First line vasoactive agent
- Typical Dosing: 1-30 mcg/min
- Goal is for MAP 60-65 mmHg



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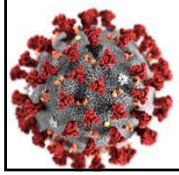
Vasopressin

- 2nd line vasoactive agent
- Typical Dosing: 0.04 units/min (not titrated)
- Can cause coronary and splanchnic vasoconstriction



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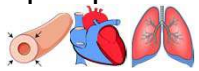
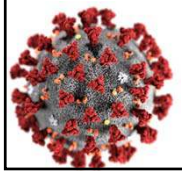
Epinephrine



- Can be used as 1st line agent if norepinephrine is not available
- Typical Dosing: 0.5-30 mcg/min



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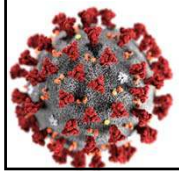
Phenylephrine

- 3rd or 4th line for Septic Shock
- Typical Dosing: 10-300 mcg/min
- Can cause reflex bradycardia



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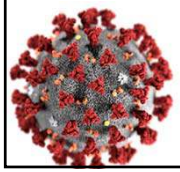
Dobutamine

- Patients with cardiac dysfunction & persistent hypoperfusion despite fluids/norepinephrine
- Can cause tachyarrhythmias or worsen hypotension
- Typical Dosing: 2.5-20 mcg/kg/min



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References

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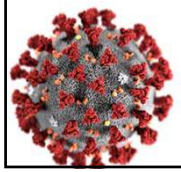

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