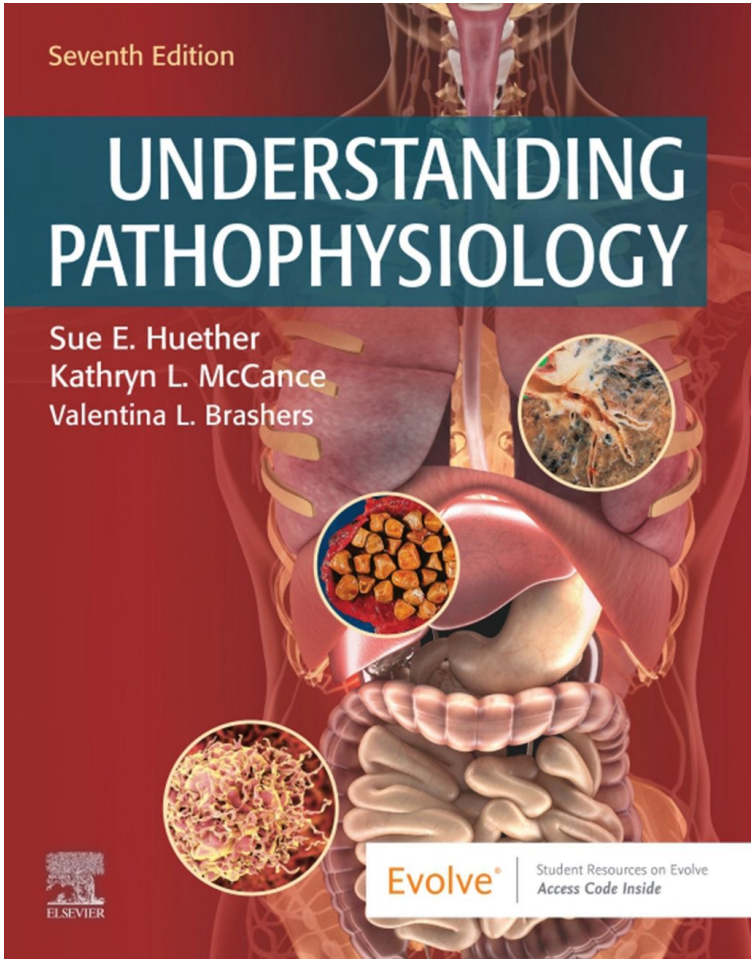
Using Pathophysiology as the Basis for Teaching in the EMS Classroom

Joseph J. Mistovich, M.Ed, NRP
Professor Emeritus
Chairperson & Professor (Retired)
Youngstown State University
Youngstown, Ohio
jjmistovich@gmail.com


Seventh Edition
UNDERSTANDING PATHOPHYSIOLOGY
Sue E. Huether
Kathryn L. McCance
Valentina L. Brashers
Evolve
Student Resources on Evolve
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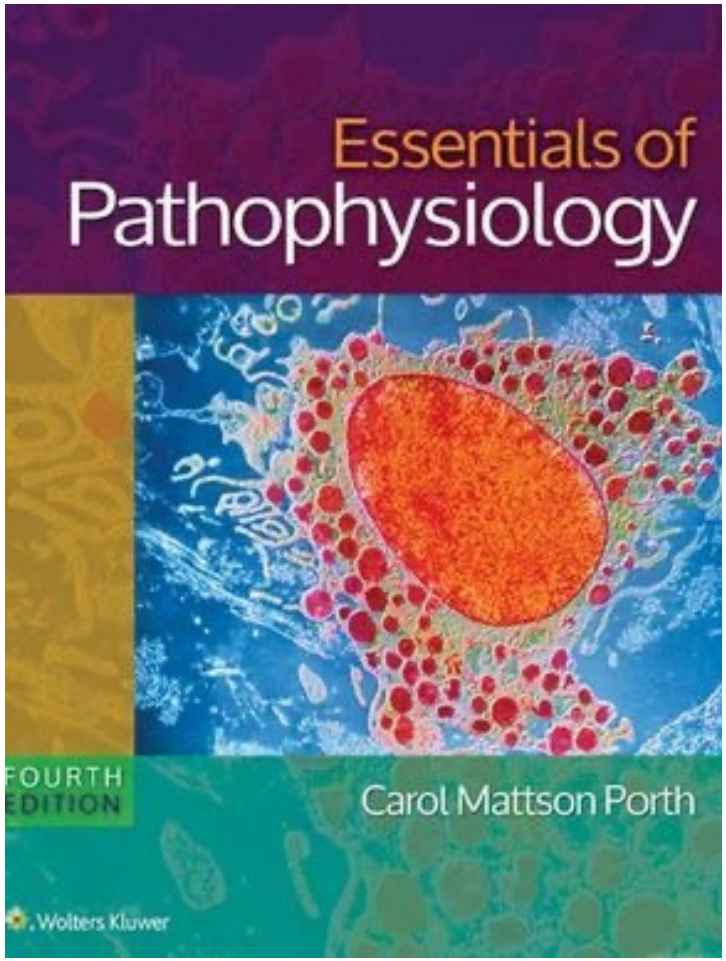

Essentials of Pathophysiology
FOURTH EDITION
Carol Mattson Porth
Wolters Kluwer

Pathophysiology in EMS Education

What pathophysiology is needed for the EMS student to understand the material that the educator will be or is presenting?

Is the pathophysiology relevant to EMS and the level of provider?

Can you as the educator apply the pathophysiology clinically to promote an understanding of emergency conditions?

- Assessment
- Treatment

Can the student use and apply the pathophysiology clinically to "figure out" assessment findings and treatment for other conditions?

Moving from a Teacher-Centered Education to a Student Centered-Education

My Personal Experience with Pathophysiology-Based EMS Education

Throw out PPT and use a whiteboard or chalkboard	Use in all clinical subject areas	Constantly explain the relevance and apply the lessons clinically	Provide foundational information	Facilitate student-led discussion	Build on previous knowledge – anatomy, physiology, pathophysiology
Encourage, praise, correct, direct, commend	Engage all students	Teach It and learn it forward and backwards	Understand - don't memorize	Constantly challenge students to use pathophysiology to “figure it out”	"It all makes sense"

What Pathophysiology Is Relevant to EMS? Top 12 Topics

1. Cellular metabolism (Aerobic vs. Anaerobic, By-products)

2. Composition of Ambient Air (Percentages and Pressures)

3. Patency of the Airway (Structures, Muscle Flaccidity, Adult vs. Pediatric)

4. Mechanics of Ventilation (Structures, Changes in Pressures, Boyle's Law, Water Glass Effect, Airway Resistance, Compliance, Active vs. Passive, Cardiothoracic Pump Effect, Valsalva Maneuver, Minute Ventilation vs. Alveolar Ventilation)

What Pathophysiology Is Relevant to EMS? Top 12 Topics

5. Control of Ventilation (Medullary Rhythm Centers – VRG &DRG, Chemoreceptors)

6. Local Regulation of Vessels & Bronchioles to Changes in Gas Transport (Haldane and Bohr Effect)

7. Ventilation/Perfusion (V/Q) Ratio (Hypoxia, Hypoxemia, Hypercarbia)

8. Blood Composition, Fluids, and Electrolytes (Osmosis, Diffusion, Active Transport)

What Pathophysiology Is Relevant to EMS? Top 12 Topics

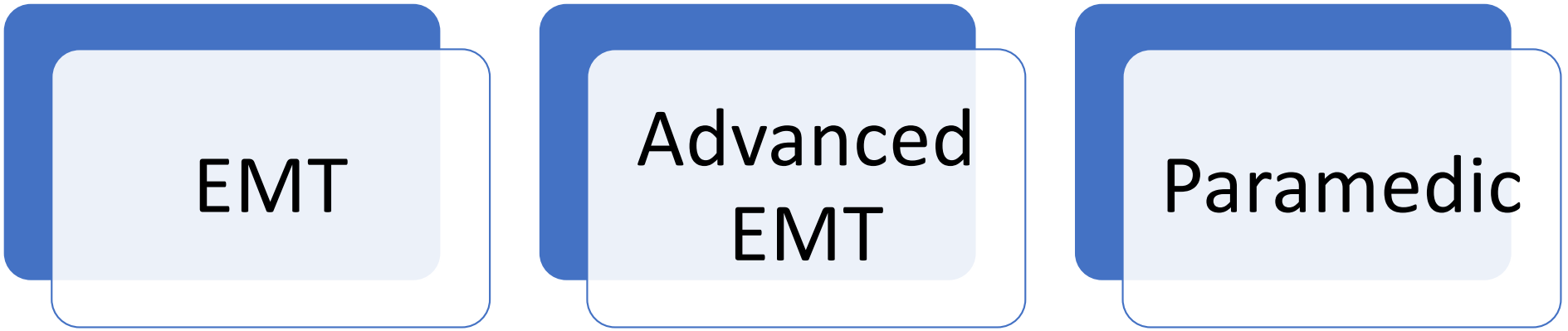
9. Transport of Gases by the Blood (O2, CO2, Plasma, Bicarbonate)

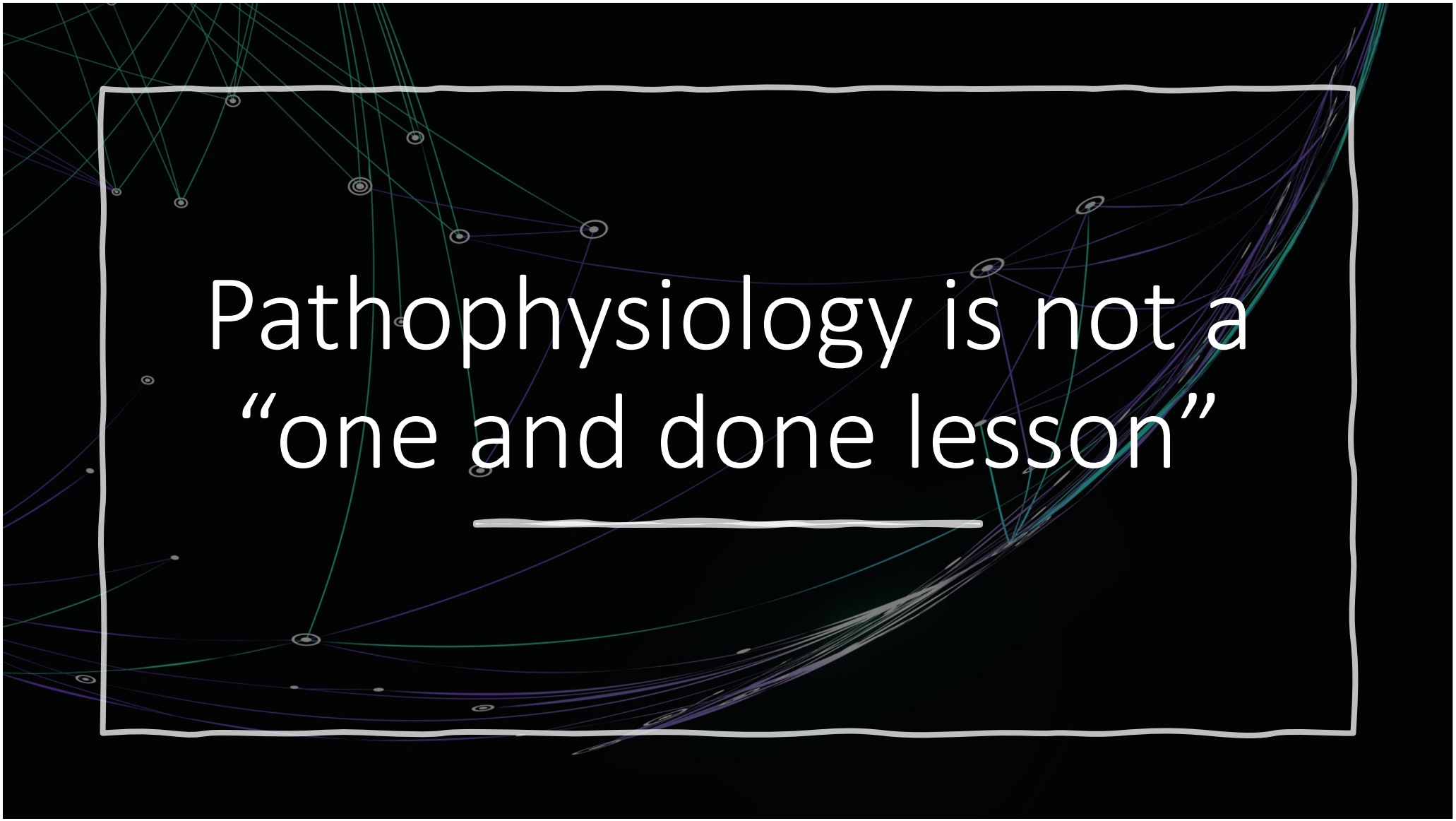
10. Hemodynamics (BP, CO, SV, Preload, Myocardial Contractility, Afterload, SVR)

11. Microcirculation (Local, Neural, and Hormonal Control)

12. Acid- Base Balance (Respiratory and Metabolic Components)

Depth and Breadth is Determined by the Level of Provider Education




Pathophysiology is not a
“one and done lesson”

Teach It Forward and Teach It Backward

Pathophysiology

Vasodilation SVR ↓

Vasoconstriction SVR ↑

→

Clinical Assessment Finding

Warm, red, flushed, dry skin ↓ DBP

Pale, cool, clammy skin ↑ DBP

Clinical Assessment Finding

Warm, red, flushed skin DBP ↓

Pale, cool, clammy skin DBP ↑

→

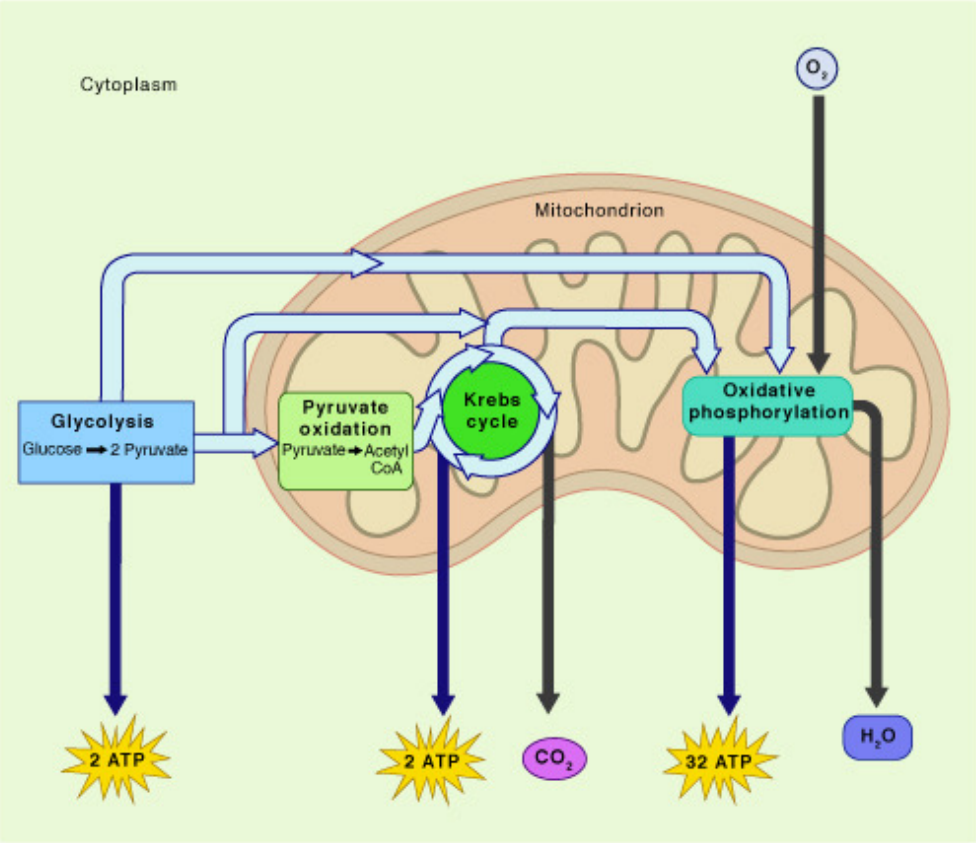
Pathophysiology

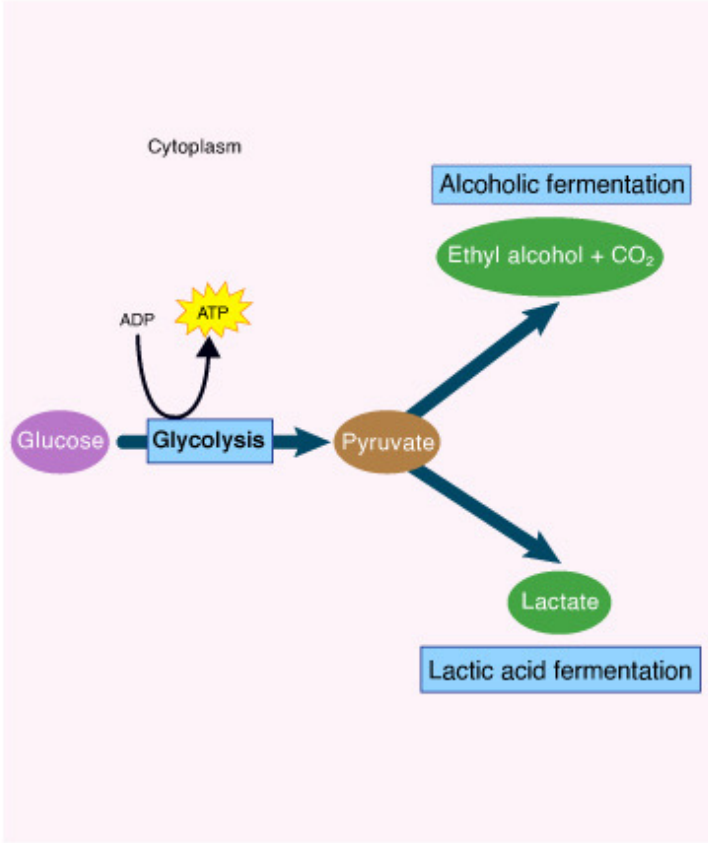
Vasodilation SVR ↓

Vasoconstriction SVR ↑

Apply the Pathophysiology Clinically

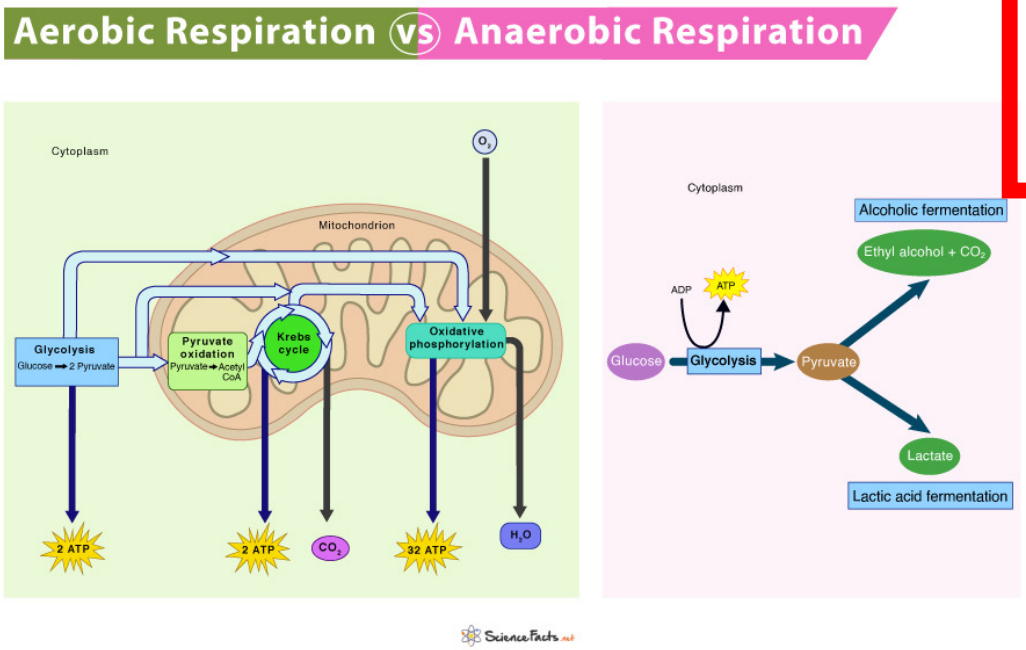
Aerobic Respiration vs Anaerobic Respiration





ScienceFacts.net

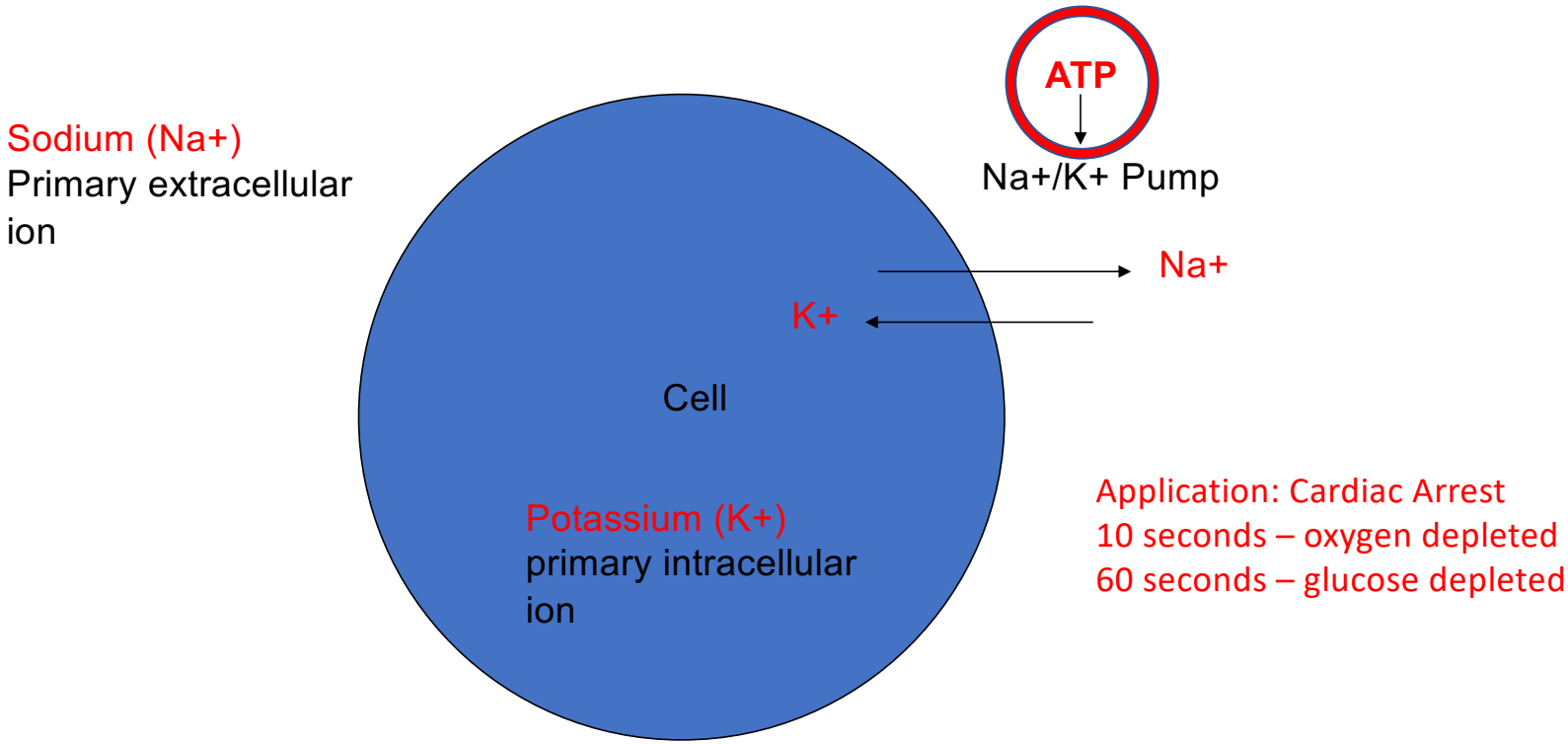
MVC with Two Patients



- Driver – screaming and yelling for help c/o open fracture of humerus and severe pain.
- Passenger – Sitting calmly and slow to respond to questions.

- ATP allows cell to function or change structure
- Mental status implication
- Why is patient warmth an issue?
- CO2 vs Latic acid
- CO2 + H2O = H2CO3
- Fever of 1 degree F or 0.6 degrees C increases HR by 10 bpm. (Fever of 101.6 = 30 bpm HR increase)

How Cells Die -**Sodium/Potassium Pump Failure**

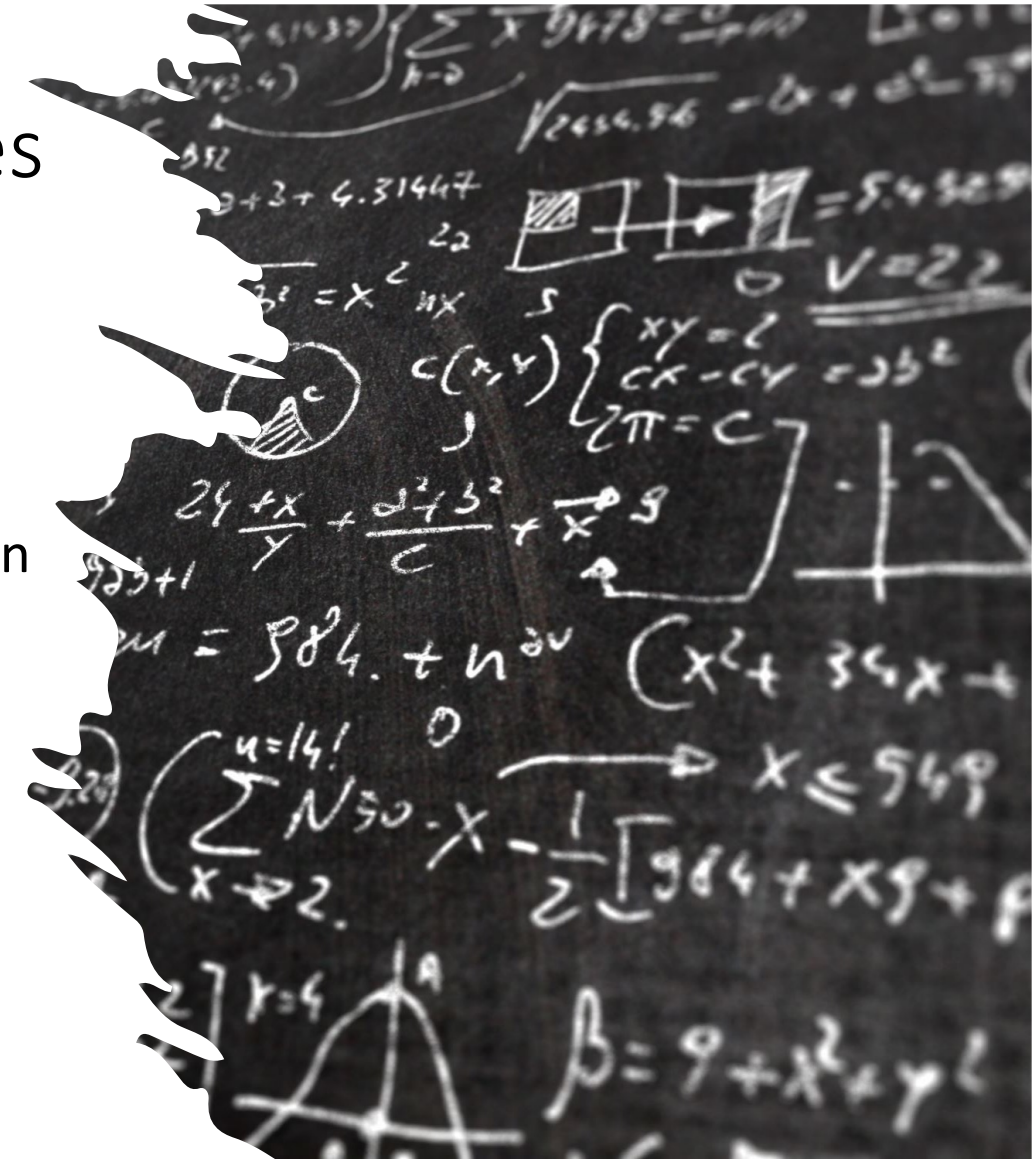


Aerobic metabolism = 36 ATP → Anaerobic metabolism = 2 ATP

How to Teach Anaphylactic Shock Using a Student – Centered Pathophysiology-Based Approach

Pathophysiologic Principles

- Student Responsibility - Preparation
  - Resources
    - Textbook
    - MyLab (exercises, quizzes)
- Teacher Facilitated – Lecture/Discussion of Foundational Information
  - NO PPT
  - Whiteboard or chalkboard
- Student-Centered and Teacher-Facilitated Discussion (Assessment Findings, Treatment)
  - Whiteboard or chalkboard



What Pathophysiology Is Necessary to UNDERSTAND Anaphylaxis?

1. Cellular metabolism

   Poor Perfusion and low ATP production

2. Composition of Ambient Air

   Need for oxygen therapy

3. Patency of the Airway

   Laryngeal edema and airway resistance (capillary permeability issue)

4. Mechanics of Ventilation

   Respiratory distress vs. Respiratory failure

   When to provide PPV vs. oxygen therapy

What Pathophysiology Is Necessary to UNDERSTAND Anaphylaxis?

5. Control of Ventilation

Respiratory Rate and Tidal Volume Changes

Chemoreceptor response to low O2 and high CO2

6. Local Regulation of Vessels & Bronchioles to Changes in Gas Transport

Metabolic acid vasodilates

Bronchioles with low CO2 constrict

Peripheral increase in CO2 vasodilates

Alveolar capillaries with high CO2 and low O2 constrict and shunt blood (effect of increase in hydrostatic pressure with an increased capillary permeability)

7. Ventilation/Perfusion

Explains the hypoxemia and cellular hypoxia and treatment

Ventilation disturbance

Perfusion disturbance

8. Blood Composition, Fluids, and Electrolytes

Increased capillary permeability

Interstitial edema

Intravenous fluid of choice in treatment

What Pathophysiology Is Necessary to UNDERSTAND Anaphylaxis?

9. Transport of Gases by the Blood

Hypoxemia

Hypercarbia

Cellular Hypoxia

10. Hemodynamics

Poor perfusion state – Hypotension due to decreases in SVR and Preload

BP = CO x SVR

CO = HR x SV (preload, myocardial contractility, afterload)

11. Microcirculation

Peripheral vasodilation effect on BP & perfusion

SVR changes

Skin signs and blood pooling

12. Acid- Base Balance

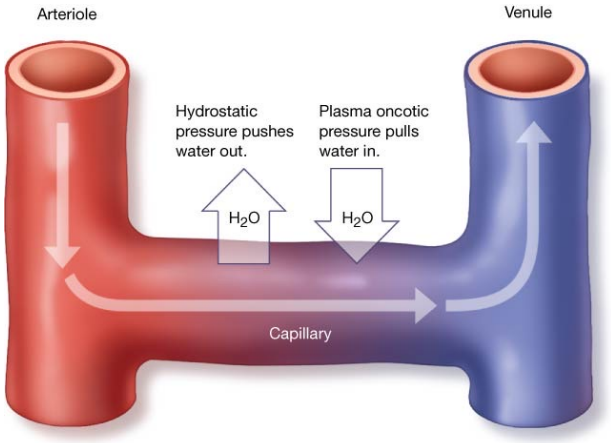
Respiratory acidosis

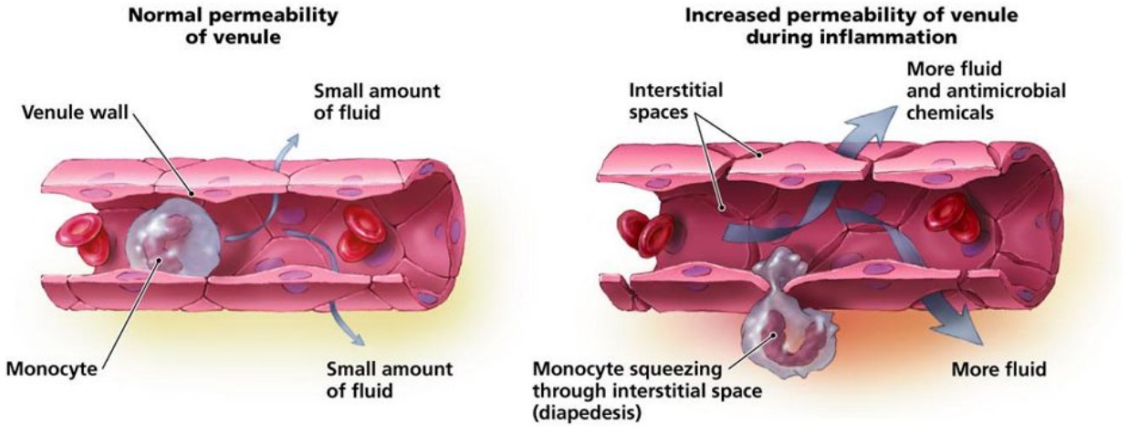
Metabolic acidosis

pH and H+ ion concentration

- Decrease in plasma oncotic pressure
- Increase in hydrostatic pressure
- Increase in capillary permeability
- Lymphatic channel obstruction

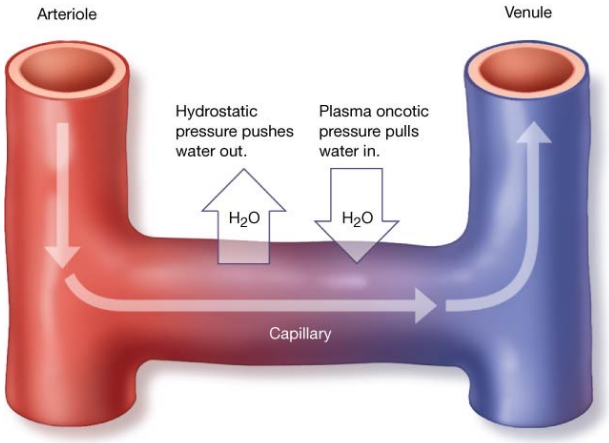
EDEMA

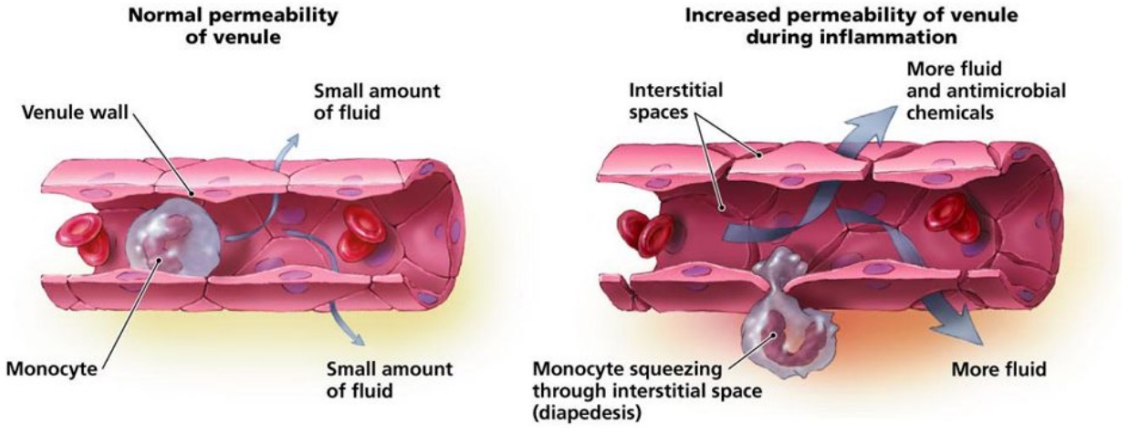




- Decrease in plasma oncotic pressure
- Increase in hydrostatic pressure
- Increase in capillary permeability
- Lymphatic channel obstruction

Laryngeal edema



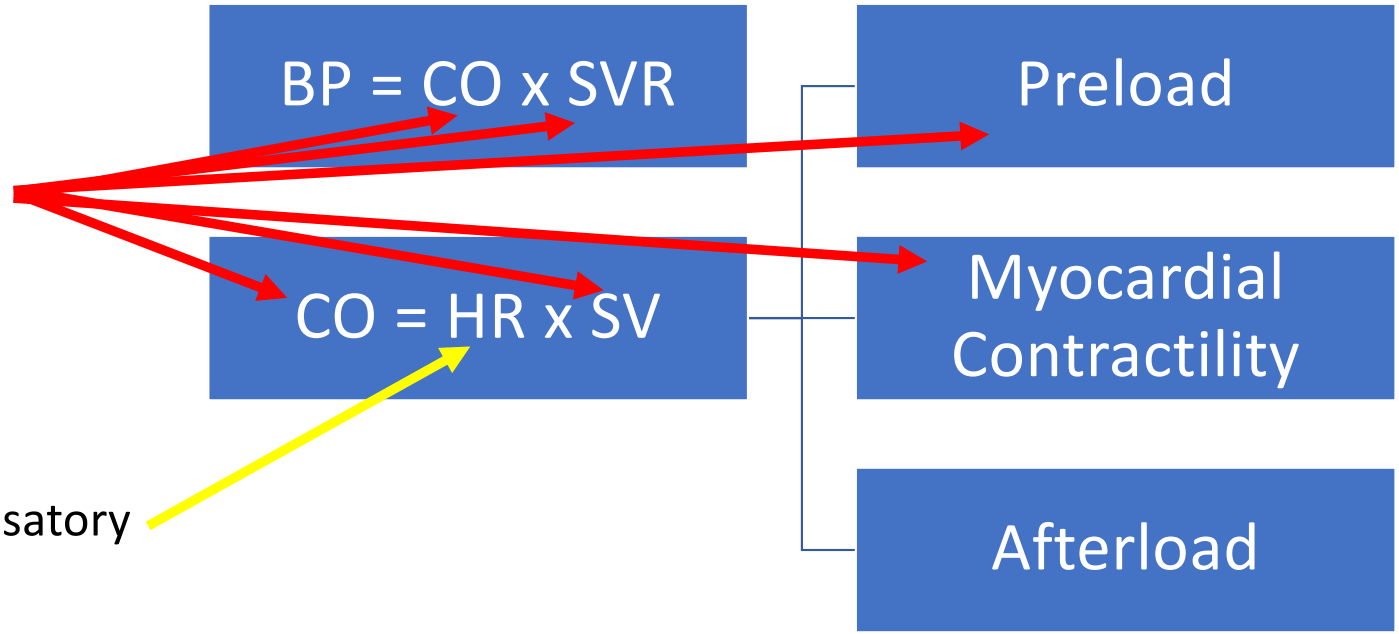


Hemodynamics of Blood Pressure

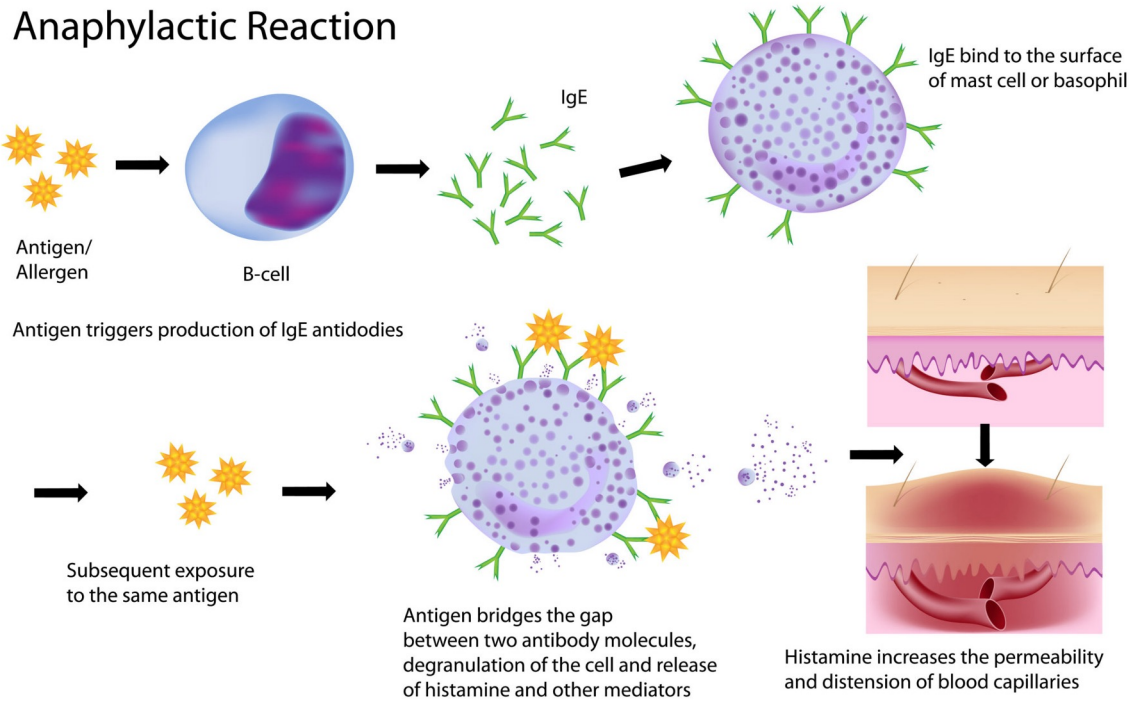
Explains:

Etiology of poor perfusion and hypotension

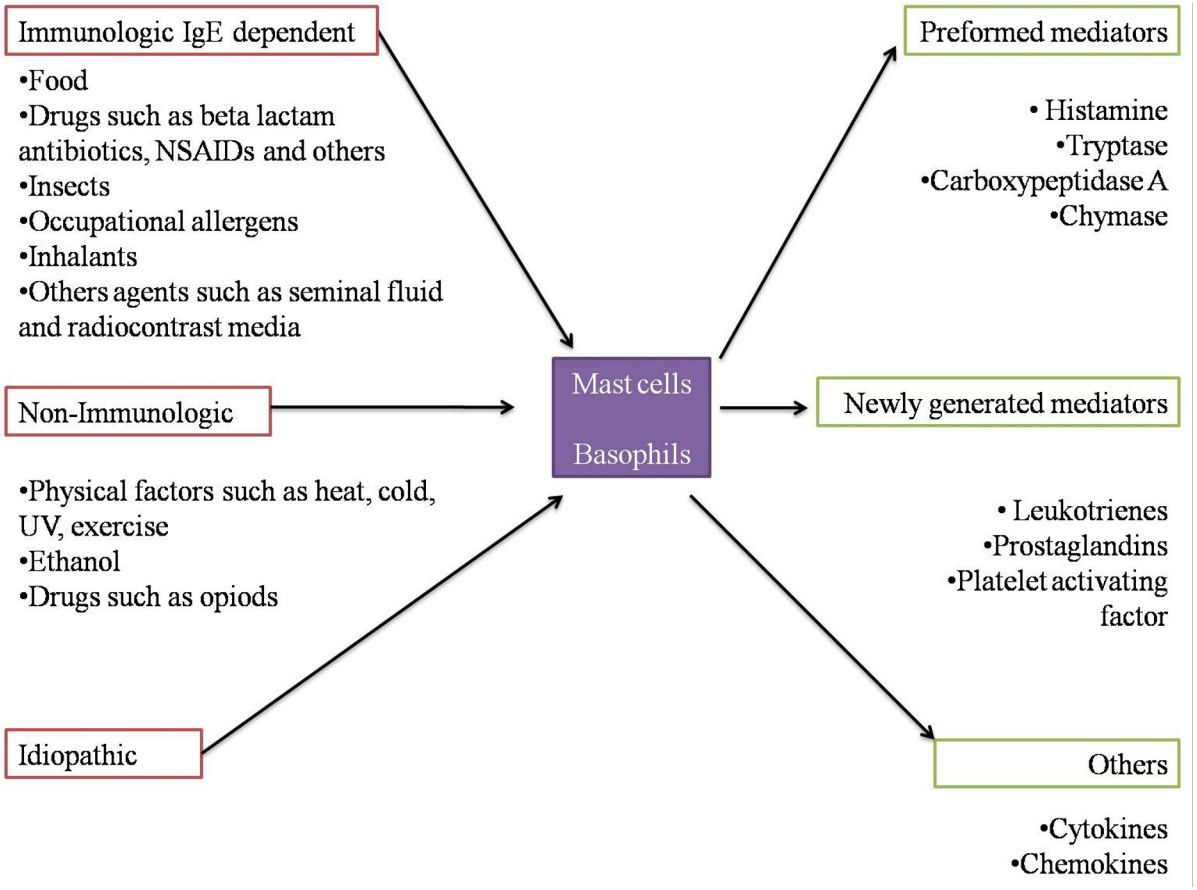
Expected compensatory signs



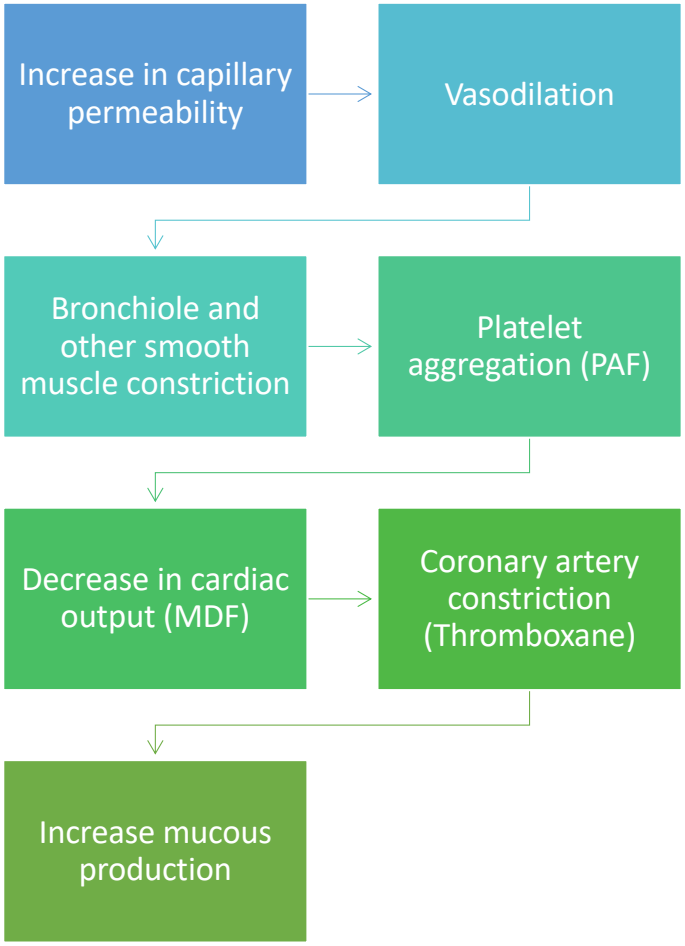
Foundation Information



Foundational Information



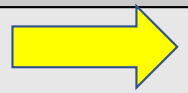
Student-Led Activity – List Pathophysiologic Effects of Chemical Mediators



Next Activity – Determine Assessment Findings Related to Pathophysiology

- Pathophysiology → Assessment Finding
- Increased Capillary Permeability → Peripheral Edema
  Stridor

- Pathophysiology ← Assessment Finding
- Bronchiole Smooth Muscle Contraction → Wheezing
- Increased capillary permeability ← Bronchiole Mucosal Inflammation ← Cough

Pathophysiology	**What Are the Assessment Findings?**
Increase in capillary permeability	The student provides the ~~pathophysiology and then~~ determines the assessment finding related to that pathophysiology
Vasodilation	
Bronchiole Smooth Muscle Contraction	
Other Smooth Muscle Contraction	Pathophysiology  Assessment Finding
Platelet Aggregation (PAF)	
Decrease in CO (MDF)	
Coronary Artery Constriction (Thromboxane)	
Increase mucous production	

Pathophysiology	What Are the Assessment Findings
Increase in capillary permeability	Rhinitis, laryngeal edema (stridor), hoarseness, throat tightness, tachycardia, urticaria, pruritis, angioedema, diarrhea
Vasodilation	Nasal congestion, tachycardia, hypotension, lightheadedness, weakness, syncope, hypotension, chest pain, tingling, warmth sensation, flushed skin, anxiety, headache,
Bronchiole Smooth Muscle Contraction	Wheezing, retrosternal tightness, tachypnea, dyspnea, cyanosis, respiratory distress, decreased SpO2, anxiety, restless, agitated, sleepy
Other Smooth Muscle Contraction	Dysphagia, GI cramping, nausea, vomiting, tenesmus, urinary incontinence, uterine cramping, pelvic pain
Platelet Aggregation (PAF)	Bruising, abnormal bleeding, DIC, vaginal bleeding
Decrease in CO (MDF)	Tachycardia, hypotension,
Coronary Artery Constriction (Thromboxane)	Chest pain, dysrhythmias,
Increase mucous production	Cough, rhonchi,
Stimulation of Nerve Endings	Sneezing, ocular itching, increased lacrimation, red eyes

Assessment Finding	What is the Pathophysiology	Assessment Finding	What is the Pathophysiology	Assessment Finding	What is the Pathophysiology
Urticaria	#1 or ICP				
Flushed Skin	#5 or V				

The student provides the assessment finding and then must determine the associated pathophysiology

Pathophysiology ← Assessment Finding

1. Increase in capillary permeability (ICP)
2. Platelet aggregation (PA)
3. Increase mucous production (IMP)
4. Bronchiole/other smooth muscle constriction (BOSMC)
5. Vasodilation (V)
6. Decrease in cardiac output (DCO)
7. Coronary artery constriction (CAC)
8. Nerve ending stimulation (NES)

Assessment Finding	Pathophysiology	Assessment Finding	Pathophysiology	Assessment Finding	Pathophysiology
Urticaria	1 or ICP	Dysrhythmias	7, 4	N/V	4, 3
Flushed Skin	5 or V	Hypotension	1, 5, 6,	Dyspnea	4
Sneezing	8 or NES	Abnormal bleeding	2	Headache	5
Cough	3	GI cramping	4	Vaginal bleeding	2
Chest pain	7	Retrosternal tightness	4	Tingling	5
Tachycardia	6, 5, 1	Tachypnea	4	Urinary incontinence	4
Bruising	2	Uterine cramping	4	Agitated	4
Dysphagia	4	Lightheadedness	5	Syncope	5
Wheezing	4	Tenesmus	4	DIC	2
Nasal congestion	5	Warmth feeling	5	Sleepy	4
Rhinitis	1	Hoarseness	1	Pelvic pain	4
Laryngeal edema (stridor)	1	Throat tightness	1	Cyanosis	4
Ocular itching	8	Increased Lacrimation	8	Red eyes	8, 5
Rhonchi	3	Anxiety	4, 5,6	Decreased SpO2	4
Urticaria	1	Pruritis	1	Angioedema	1
Diarrhea	1, 4				

1. Increase in capillary permeability (ICP)
2. Platelet aggregation (PA)
3. Increase mucous production (IMP)
4. Bronchiole/other smooth muscle constriction (BOSMC)
5. Vasodilation (V)
6. Decrease in cardiac output (DCO)
7. Coronary artery constriction (CAC)
8. Nerve ending stimulation (NES)

Based on Pathophysiology and the Related Assessment Findings the Student Must Then Determine Treatment

What Are the Immediate Life Threats?

- Airway closure/obstruction
- Respiratory Failure
- Severe Hypoxemia
- Poor Perfusion

Reverse the Pathophysiology and Eliminate the Immediate Life Threats and Signs and Symptoms

What Are the Immediate Life Threats Based on the Pathophysiology?

Airway closure/obstruction

- Laryngeal edema due to increased capillary permeability

Respiratory Failure

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability

Severe Hypoxemia

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability
- Vasodilation

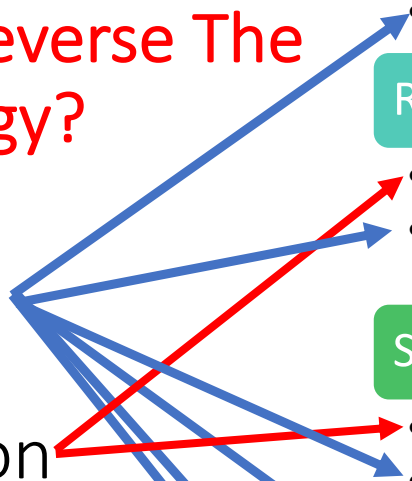
Poor Perfusion

- Vasodilation
- Increased capillary permeability

How Do You Reverse The Pathophysiology?

Vasoconstrict

Bronchodilation



Airway closure/obstruction

- Laryngeal edema due to increased capillary permeability

Respiratory Failure

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability

Severe Hypoxemia

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability
- Vasodilation

Poor Perfusion

- Vasodilation
- Increased capillary permeability

Why Not Just Give Diphenhydramine?

MEDIATORS OF ANAPHYLAXIS

- Histamine : stimulates vasodilation and increases vascular permeability, heart rate, cardiac contraction, and glandular secretion.
- Prostaglandin D_2 : is a bronchoconstrictor, pulmonary and coronary vasoconstrictor, and peripheral vasodilator.
- Leukotrienes : produce bronchoconstriction, increase vascular permeability, and promote airway remodeling.
- Platelet-activating factor : is also a potent bronchoconstrictor and increases vascular permeability.
- Tumor necrosis factor: activates neutrophils, recruits other effector cells, and enhances chemokine synthesis.

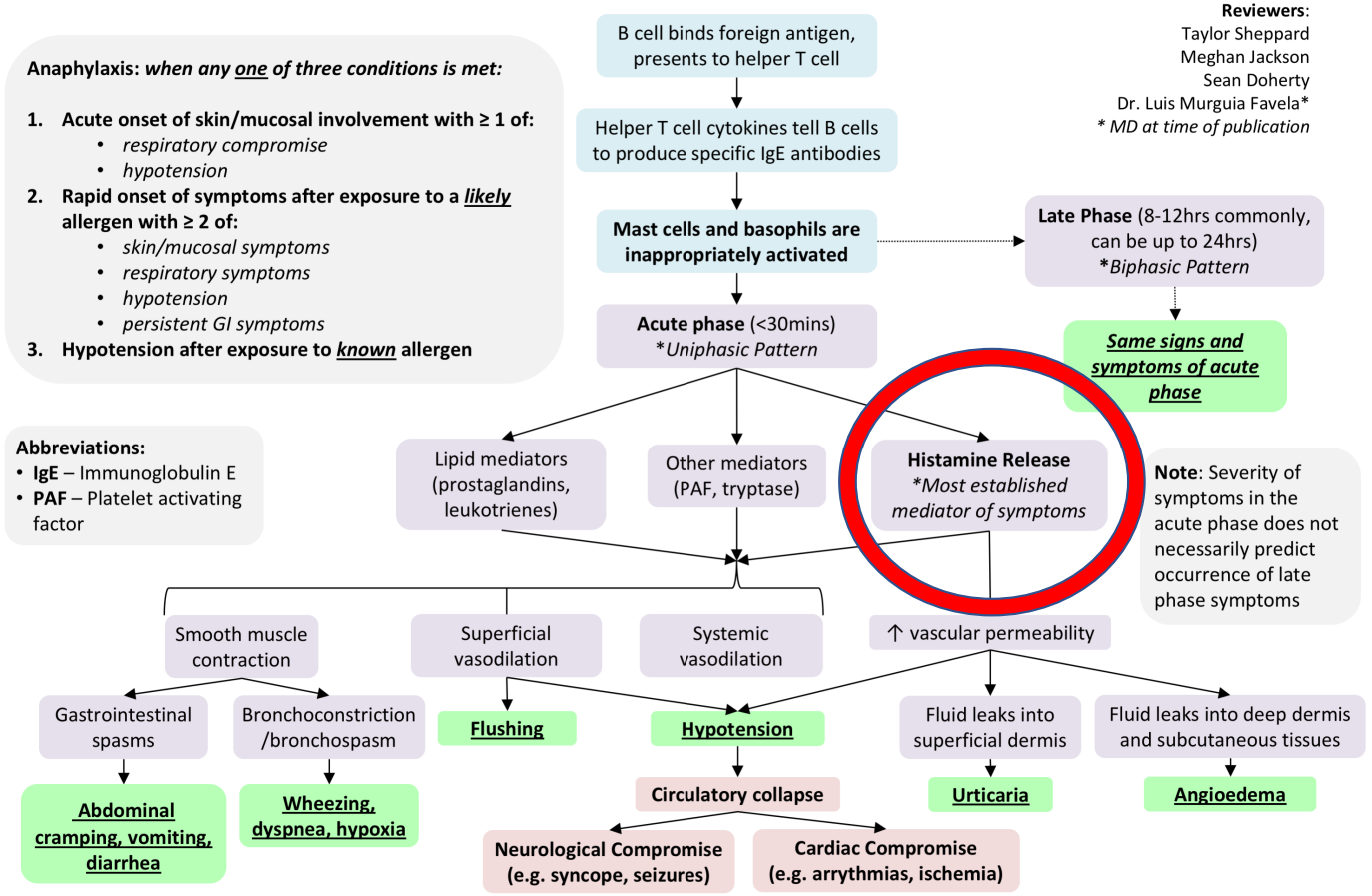
Anaphylaxis: *Signs and Symptoms*

Authors:
Matt Piaseczny
Reviewers:
Taylor Sheppard
Meghan Jackson
Sean Doherty
Dr. Luis Murguia Favela*
** MD at time of publication*

Anaphylaxis: *when any <u>one</u> of three conditions is met:*

1. **Acute onset of skin/mucosal involvement with ≥ 1 of:**
   - *respiratory compromise*
   - *hypotension*
2. **Rapid onset of symptoms after exposure to a ***<u>likely</u>*** allergen with ≥ 2 of:**
   - *skin/mucosal symptoms*
   - *respiratory symptoms*
   - *hypotension*
   - *persistent GI symptoms*
3. **Hypotension after exposure to ***<u>known</u>*** allergen**

Abbreviations:
- **IgE** – Immunoglobulin E
- **PAF** – Platelet activating factor



Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

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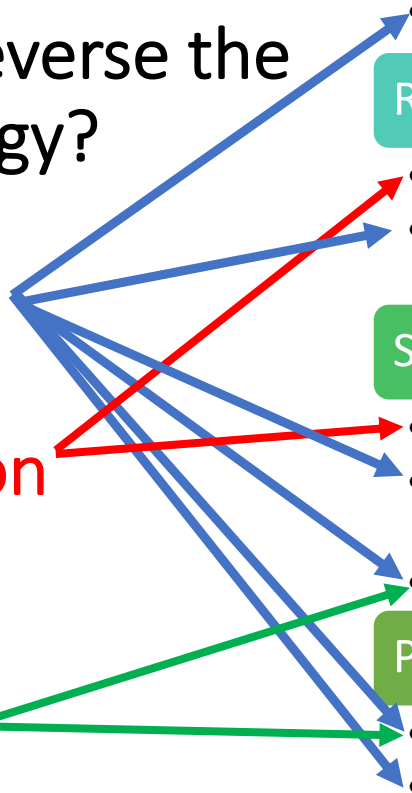
How do you reverse the pathophysiology?

Vasoconstrict
Alpha 1 & 2

Bronchodilation
Beta 2

Isotonic fluid



Airway closure/obstruction

- Laryngeal edema due to increased capillary permeability

Respiratory Failure

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability

Severe Hypoxemia

- Bronchoconstriction
- Bronchiole inflammation due to increased capillary permeability
- Vasodilation

Poor Perfusion

- Vasodilation
- Increased capillary permeability

Epinephrine – Drug of Choice

- The stridor is still severe indicating the laryngeal edema is persistent and the patient is still at risk for airway closure/obstruction?
  - Why?
    - Increased capillary permeability
  - Action Needed?
    - Reverse the capillary permeability
  - How?
    - $Alpha_{1\ \&2}$
  - Treatment?
    - Administer another dose of epinephrine for the alpha stimulation
    - Consider advanced airway management
    - Maximize oxygenation

Epinephrine – Drug of Choice

- The patient remains hypotensive with signs of poor perfusion?
  - Why?
    - Persistent vasodilation
  - Action Needed?
    - Increase the intravascular pressure
  - How?
    - Vasoconstrict -Alpha$_{1\ \&2}$
    - Fill the vascular space with volume
  - Treatment?
    - Increase the SVR by administering another dose of epinephrine for the alpha stimulation and vasoconstriction
    - Initiate two large bore IVs with NS or LR infusion WO

Treatment Choice

- The patient has persistent wheezing with no other signs.
  - Why?
    - Persistent bronchoconstriction
  - Action Needed?
    - Bronchodilate
  - How?
    - $Beta_2$ Specific agonist
    - Nebulize?
  - Treatment?
    - Direct deposition of the $Beta_2$ stimulant at the site of smooth muscle contraction with limited side effects
  - Patient has muscle tremors after treatment
    - Concern? Why?
      - No. $Beta_2$ stimulates skeletal smooth muscle

“IT ALL MAKES SENSE”

Questions or Comments?

jjmistovich@gmail.com