

Pediatric Critical Care Case Studies

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I have no conflicts of interest to disclose.

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Learning Objective

- Discuss the presentation and management of several disease processes seen in children requiring the PICU

Talk Outline

Case presentations

Pathophysiology

Therapies and Supportive Management

Pearls

Case 1



Case 1

- Previously healthy 11-year-old male presents with respiratory distress and dehydration
- On exam:
 - Afebrile. Patient is ill appearing with sunken eyes. Appears breathless. Cannot complete full sentence without taking another breath
 - Taking deep and fast respirations, RR 27. Lungs clear to auscultation
 - HR 136. No murmurs. Pulses 2+
 - Abdomen tender in epigastric region
 - Dry mucous membranes
- Further history reveals 2 months of thirst and excessive water intake. Past week with congestion/cough

Case 1

- Labs:

VBG **7.06/9/8/-20**

130	103	20	520
4.5	7	1.1	

	14.6	
11.2		328
	43.8	

Normal Anion Gap is ≤ 10 mEQ/L

Anion Gap = Corrected Na – (Serum Cl + Serum HCO_3^-)

$$\text{Corrected Na} = \text{Na (mEQ/L)} + 1.6 \left(\frac{\text{Glucose} - 100}{100} \right)$$

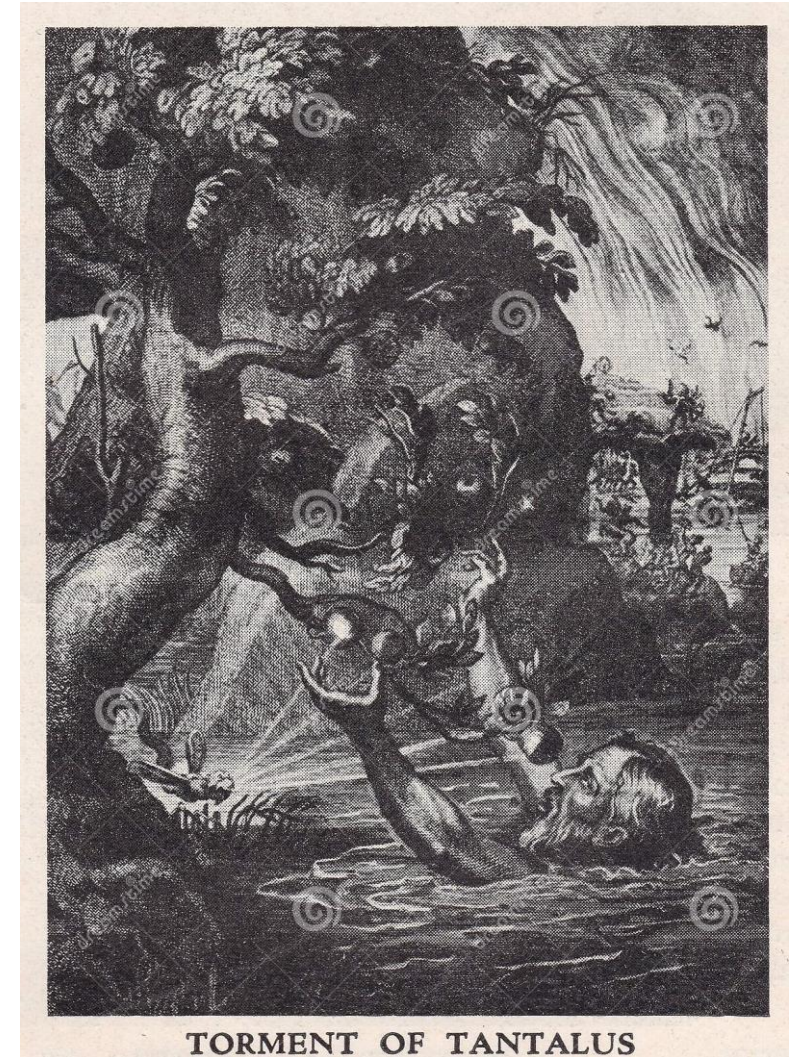
Corrected Na – (Serum Cl + Serum HCO_3^-) = Anion Gap

$$137 - (103 + 7) = \mathbf{27}$$

Diagnosis: New diagnosis diabetes mellitus presenting with severe diabetic ketoacidosis

Case 1- DKA- Pathophysiology

- Relative lack of insulin with stress response to obtain alternative forms of metabolic energy (lipolysis, ketogenesis, gluconeogenesis, glycogenolysis)
 - Hyperglycemia → osmotic diuresis → volume loss, depletion of K and Phos
 - Ketosis → anion gap
 - Respiratory compensation and Kussmaul breathing
 - Development of cerebral edema due to fluid and osmotic shift



Case 1- DKA- Management

CLINICAL PATHWAY



Diabetic Ketoacidosis (DKA) Treatment ALGORITHM

Algorithm for the Management of Diabetic Ketoacidosis (DKA)

Immediate Assessment:
Place patient on full cardio/respiratory (CR) monitor and obtain Vital Signs
Refer to ED/UC RN standing orders

Inclusion:
Suspicion of DKA
Exclusion:
Symptoms clearly attributed to other causes

Clinical History
Polyuria
Polydipsia
Weight loss
Abdominal pain
Fatigue
Vomiting
Confusion

Clinical Signs
Assess dehydration
Deep sighing respiration (Kussmaul)
Smell of ketones
Lethargy/drowsiness + vomiting

Obtain Initial Labs
RFP or BMP with phos, ketones (urine or blood), blood glucose, VBG, A1C
DKA diagnosis confirmed by initial labs:
Hyperglycemia with glucose greater than (>)200 mg/dL
pH less than (<)7.3 or HC03 less than (<)15, and
Ketonemia or ketonuria
Contact diabetes physician
* If patient is wearing an insulin pump, remove it after confirmation of DKA

If blood glucose is ≥ 600 mg/dL, calculate Serum Osmolality to evaluate for possible hyperosmolarity. Contact diabetes physician if calculated Serum Osmolality is ≥ 320 mOsm/kg for further guidance on management.
$$\text{Serum osmolality} = (\text{Na} \times 2) + (\text{Glucose}/18) + (\text{BUN}/2.8)$$

If not DKA, but the patient has diabetes or hyperglycemia, contact diabetes physician

!
If considering intubation, contact PICU physician and suspect cerebral edema

Case 1- DKA- Management

!
Intravenous
insulin boluses
and
sodium bicarbonate
are contraindicated
in DKA patients

Does the patient
have concern for shock:
hypovolemic instability,
decreased end organ
perfusion, altered mental
status, and/or
hypotension?

Resuscitation

- Maintain SpO2 at 100%
- 0.9% NaCl 10-20 ml/kg over 30 minutes and repeat until circulation is restored. Do not exceed 40mL/Kg unless patient is in shock
- Consider early pressors
- Avoid sedating drugs

If acidosis not improving or if deterioration, contact PICU physician

Re-evaluate

- IV fluid calculations
- Insulin delivery system and dose
- Need for additional resuscitation
- Consider sepsis

Initial Interventions
NaCl 0.9% 10-20 ml/kg bolus over 1 hour
Consider ECG if K is over 6 or under 3 mEq/L

- Obtain blood glucose (BG) after bolus complete and prior to starting insulin drip. Begin Q1 hour BG checks at this time
- Start regular insulin IV at 0.1 units/kg/hour after IV fluid bolus complete
Consider insulin drip rate as low as 0.05 units/kg/hour for the following situations: cerebral edema, altered mental status, difficulty in the past with higher rates, risk for hypoglycemia, hypokalemia, small body weight
- IV fluids at 1.5X maintenance
- Document strict I/O
- **Check neurological status at least hourly**

neurological WARNING SIGNS
Severe or worsening headaches, slowing heart rate more than expected from fluid resuscitation, irritability, irregular breathing, decreased level of consciousness, incontinence, or focal neurological abnormalities are present

Then, exclude hypoglycemia
Is it cerebral edema?

Management
Elevate head of bed
Give hypertonic saline 5ml/kg or Mannitol 1 g/kg, max dose 50g
Restrict to maintenance fluid rate
Contact ICU and diabetes physician
Consider cranial imaging only after patient stabilized

Labs
BG Q1 hour
RFP (or BMP with phos) q2 hours
VBG initially, Q2 until pH at or above 7.15
Beta Hydroxybutyrate initially and as needed before transition to SC
Obtain ECG if K is over 6 or under 3

***Initial Potassium Supplementation Table**


Initial Serum Potassium	Potassium Supplementation
Greater than 5.5	None
4.5-5.5	20 mEq/L K-Acetate + 20 mEq/L Kphos*
Less than 4.5	30 mEq/L K-Acetate + 30 mEq/L Kphos* Hold Insulin drip until K above 3.

K supplementation is based on initial lab level. If K changes in management, patient may require repletion with potassium bolus

*40 mEq/L KCL may be used if K-Acetate and Kphos unavailable

Blood Glucose (mg/dL)	% Rate NS ± Electrolytes	% Rate D10NS ± Electrolytes	Final Dextrose Concentration
>300	100%	0%	0
251-299	50%	50%	5
200-250	25%	75%	7.5
151-199	0	100%	10
< 150	Either decrease insulin drip as low as 0.05unit/kg/hour and/or increase GIR by increasing D10NS fluid rate (up to 2X maintenance) or change to D12.5 NS at 100% total rate		

Case 1

- Patient is given single 20 ml/kg bolus of isotonic IV fluids before getting starting on insulin drip and 2 bag system of continuous IV fluids
- Communication handed to transport team for transfer to CHCO PICU
- 20 minutes into ground transfer, patient develops brief period of confusion (cannot recall name) and becomes very sleepy
- What should be done next?  Make sure head of bed elevated
Administer hypertonic saline or mannitol
Decrease IVF rate from 2x maintenance to 1x
Contact PICU
Avoid intubation if possible

Case 1- Pearls

- DKA itself can induce pancreatitis. Patients can have diffuse abdominal pain or epigastrically localized pain
- Risk factors for development of cerebral edema (Glaser, 2001):
 - **Bicarbonate supplementation**
 - Low pCO₂
 - High BUN
- Try to avoid sedating medications
 - Will lose neuro exam and take away critical drive of breathing
- Intubation generally contraindicated due to intrinsic respiratory compensation for metabolic acidosis
 - Very difficult to match physiologic hyperventilation
 - Cerebral vessels dilate at elevating pCO₂ levels

Case 2



Case 2

- Previously healthy 10 yo female presents with respiratory distress and dehydration
- On exam:
 - Ill appearing, diaphoretic with increased work of breathing, RR 28. Abdominal retractions.
 - Lungs diminished R>L
 - BP 80/50. HR 150. Cap refill 3-4 seconds
 - Abdomen diffusely tender
- Patient has had cough/congestion starting 2 weeks ago. Fevers in the last few days to 102°F. Family denies polyuria/polydipsia

Case 2

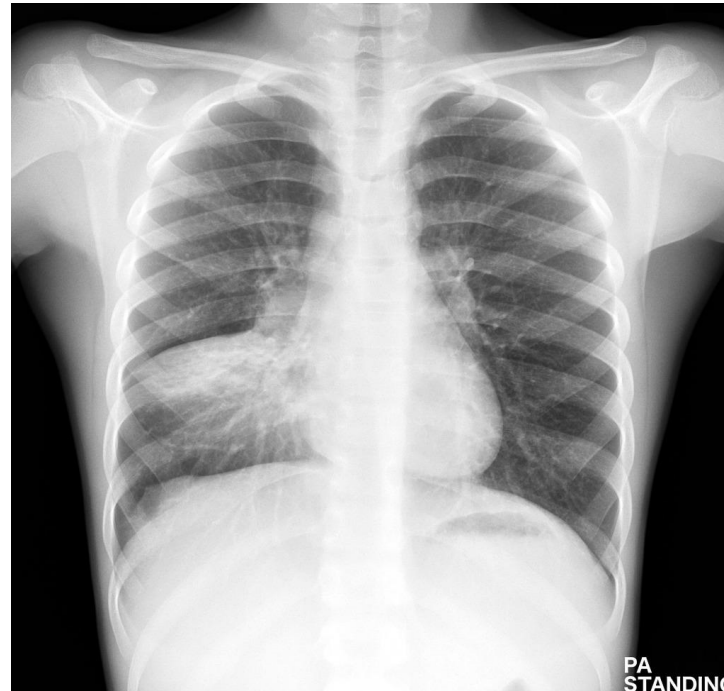
- Labs:

VBG **7.21/32/18/-8**

133	101	20	} 240
4.5	17	.76	

14.6	328
16.0	43.8

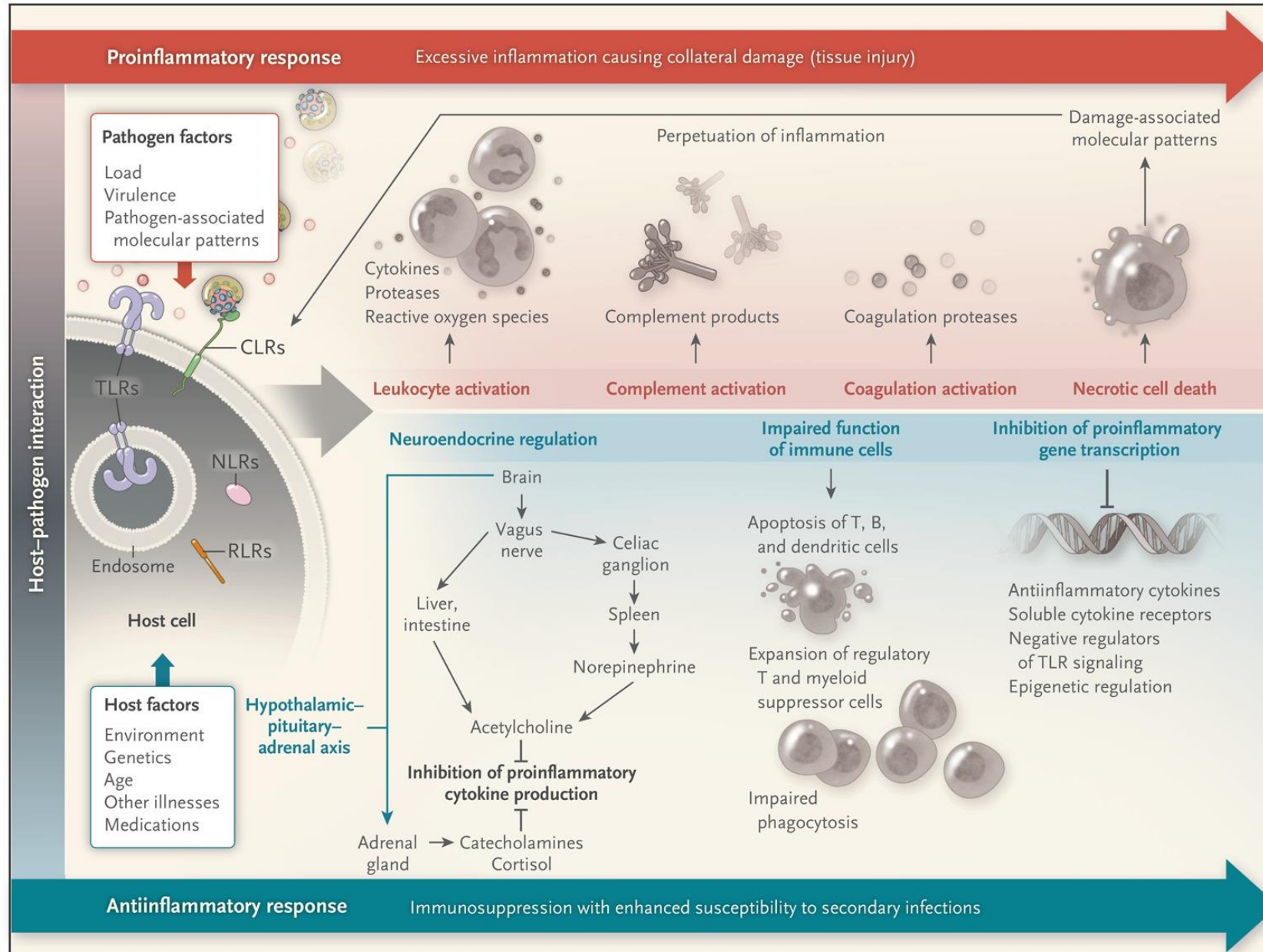
Differential diagnoses?



Lactate **5.6**

Diagnosis: RML pneumonia with septic shock

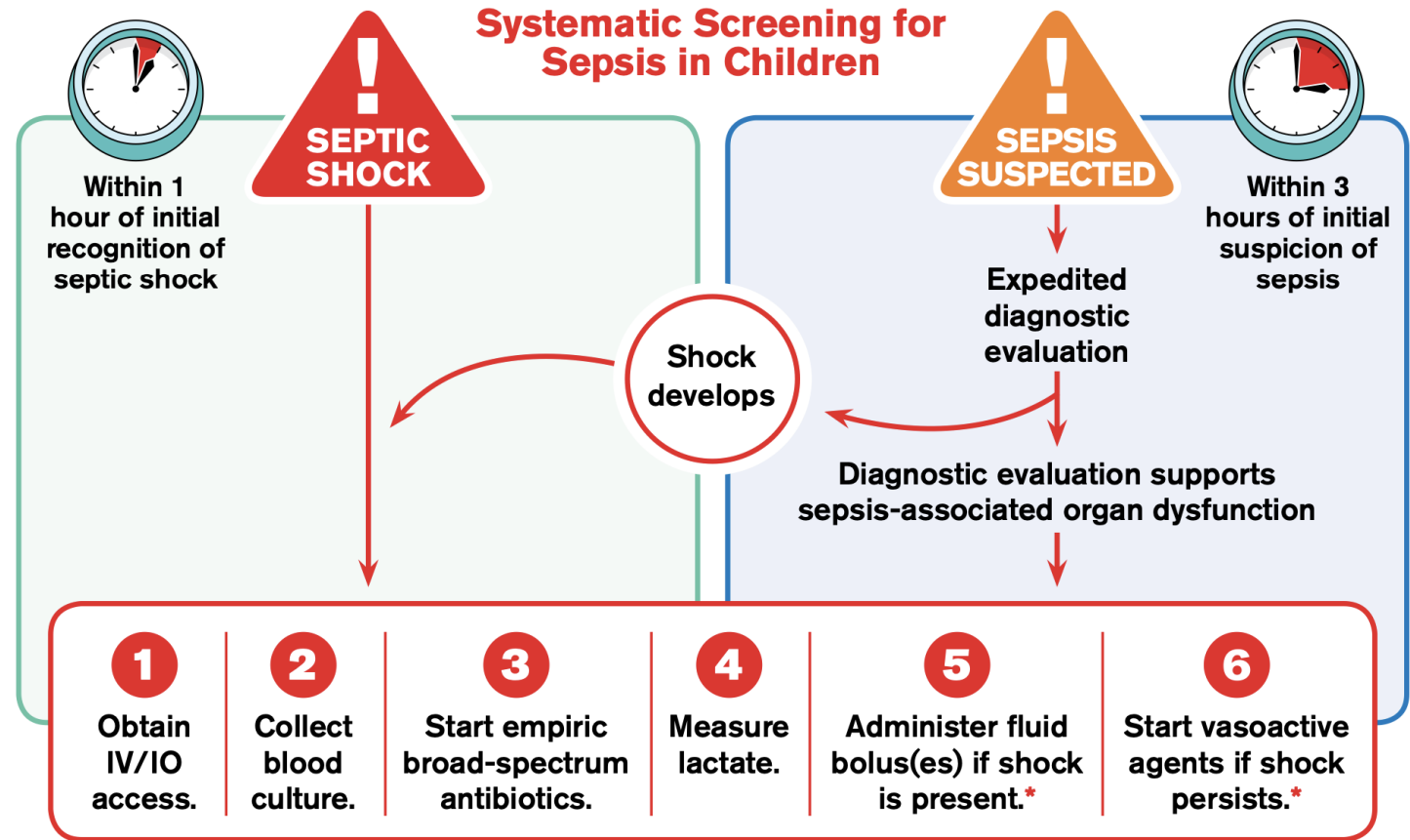
Case 2- Septic shock- pathophysiology and management



Case 2- septic shock management

Initial Resuscitation Algorithm for Children

Surviving Sepsis Campaign



Case 2- Septic shock- pearls

- Sometimes septic shock can look like DKA due to hyperglycemia and acidosis
 - DKA usually has higher degree of hyperglycemia compared to acidosis
 - History important
- Rapid antibiotic initiation can be forgotten during resuscitation for septic shock
- Hydrocortisone can be given for catecholamine refractory shock
 - Strongly consider in oncology patients
 - ALL, brain tumor, Hodgkin lymphomas

Case 3



Case 3

- Previously healthy 2 yo male presents after being found unresponsive in grandmother's pool. Last seen by mom ~10 minutes prior. Was given rescue breaths with chest compressions by dad and vomited some water. Unclear if patient was ever pulseless
- On exam:
 - Temperature 35.7°C. GCS 5 with decorticate posturing. Pupils sluggish but reactive
 - O2 saturations 86%. Disorganized breathing with RR 16. +Coughing intermittently with diminished breath sounds bilaterally
 - HR 110 without murmurs. BP 80/55. Delayed cap refill 4 seconds
- Saturations improved with low flow nasal cannula, but patient intubated due to depressed mental status

Case 3- Submersion and Drowning- epidemiology

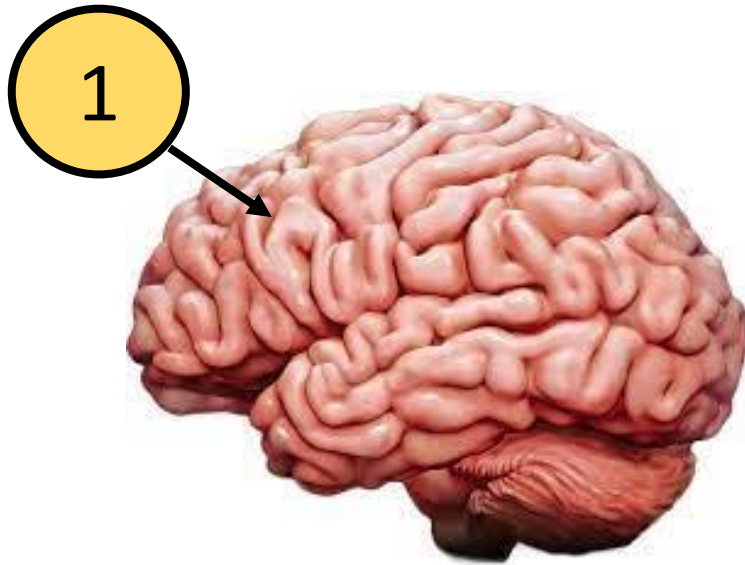
- Leading cause of accident-related childhood death and disability (WHO)
 - Prevention efforts
- Statistically, three groups of children at particular risk:
 - Very young
 - Male adolescents
 - African American children
- Also notable are children with cardiac channelopathies and prolonged QT syndrome

Case 3- Submersion and Drowning- Risk factors for poor prognosis (Quan L, *Resuscitation* 2014)

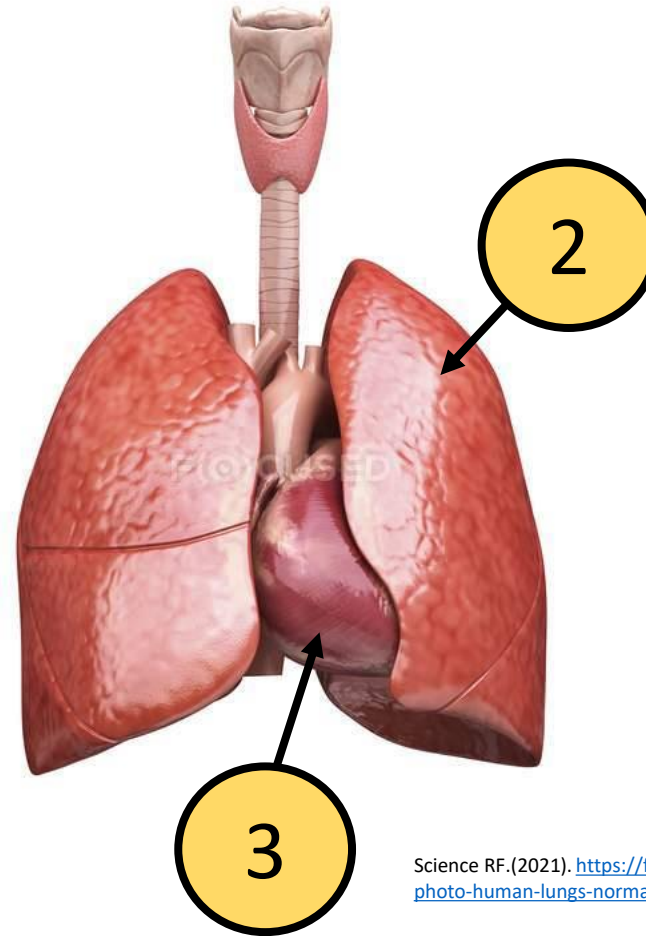
- Duration of submersion >5 minutes (most critical factor)
- Time to effective basic life support >10 minutes
- Resuscitation duration >25 minutes
- Age >14 yo
- Initial GCS <5
- Persistent apnea and CPR requirement in ED
- Initial pH <7.1
- Lack of purposeful movements at 24 hours

Case 3 – Submersion and Drowning- pathophysiology

- Three main organ systems impacted:



Brainline (2021). Interactive Brain.
<https://www.brainline.org/tbi-basics/interactive-brain>



Science RF.(2021). <https://focusedcollection.com/160225578/stock-photo-human-lungs-normal-anatomy.html>

Case 3 – Submersion and Drowning- pathophysiology

- Initial panic with disorganized breathing and struggle to not drown
- Reflex inspiratory effort with laryngospasm
- Laryngospasm and closed glottis may cause negative pressure injury to lungs, more hypoxemia and impede initial rescue efforts
- Loss of reflexes following struggle may result in aspiration of environmental fluid and surfactant washout. Could also aspirate gastric contents.
- Hypoxemia results in brain and other multi-organ injury

Case 3- Submersion and drowning- initial management

- Removal from water
- Activate EMS system
- Give rescue breaths
- Initiate CPR if no response to rescue breaths and patient still appears unresponsive
- Consider cervical collar for possible diving accidents
- Give supplemental oxygen vs. intubate if patient has low GCS or is unstable

Case 3-Submersion and drowning- additional management

Neurological

- Maintain normoxia and normocarbida as able
- Maintain normoglycemia
- Maintain normothermia
- Keep head of bed elevated
- Continuous EEG monitoring and neurocritical care consultation
- ICP goal-directed therapies (as in TBI) not generally recommended
 - e.g. hypertonic saline, targeting higher Na goals, pentobarbital gtt, etc.
 - Invasive ICP monitoring may be discussed in settings where ARDS complicates ability to maintain normal gas exchange. Can be used to determine impact of permissive hypercapnia and lower oxygen saturations

Case 3-submersion and drowning- additional management

Respiratory

- Maintain normal gas exchange through respiratory support
 - If patient suddenly develops rapidly worsening hypoxemia, consider negative pressure pulmonary edema ("Post obstructive pulmonary edema") as diagnosis
- Standard management for ARDS
 - Optimize PEEP for oxygenation while using low tidal volume strategy
 - Be cautious of pressure and volume induced lung injury
- No evidence for use of:
 - Steroids
 - Surfactant administration
- Antibiotics not prophylactically recommended
 - Can consider if water grossly contaminated or if patient had clear aspiration event

Case 3-Submersion and drowning- additional management

Cardiac (usually secondary effects from hypoxemia/lung disease)

- Use vasoactive support as needed to maintain perfusion
- EKG useful in evaluation for underlying hereditary arrhythmias
- Temperature regulation. Hypothermia can result in bradycardia and hypotension
 - Be wary of “rewarming shock”
 - Peripheral vasodilation and impaired cardiac output
 - Warm only enough to establish re-perfusing rhythm

Case 3- Submersion and drowning- pearls

- Up to 86% of drowning deaths in children < 18 yo can be mitigated through prevention efforts (including adequate adult supervision and pool gates)
- Therapy is focused on:
 - Restoration of oxygenation/ventilation and circulation at the scene
 - Rapid transfer to ED and PICU if patient does not recover
 - Supportive care and avoidance of secondary insults

Case 4



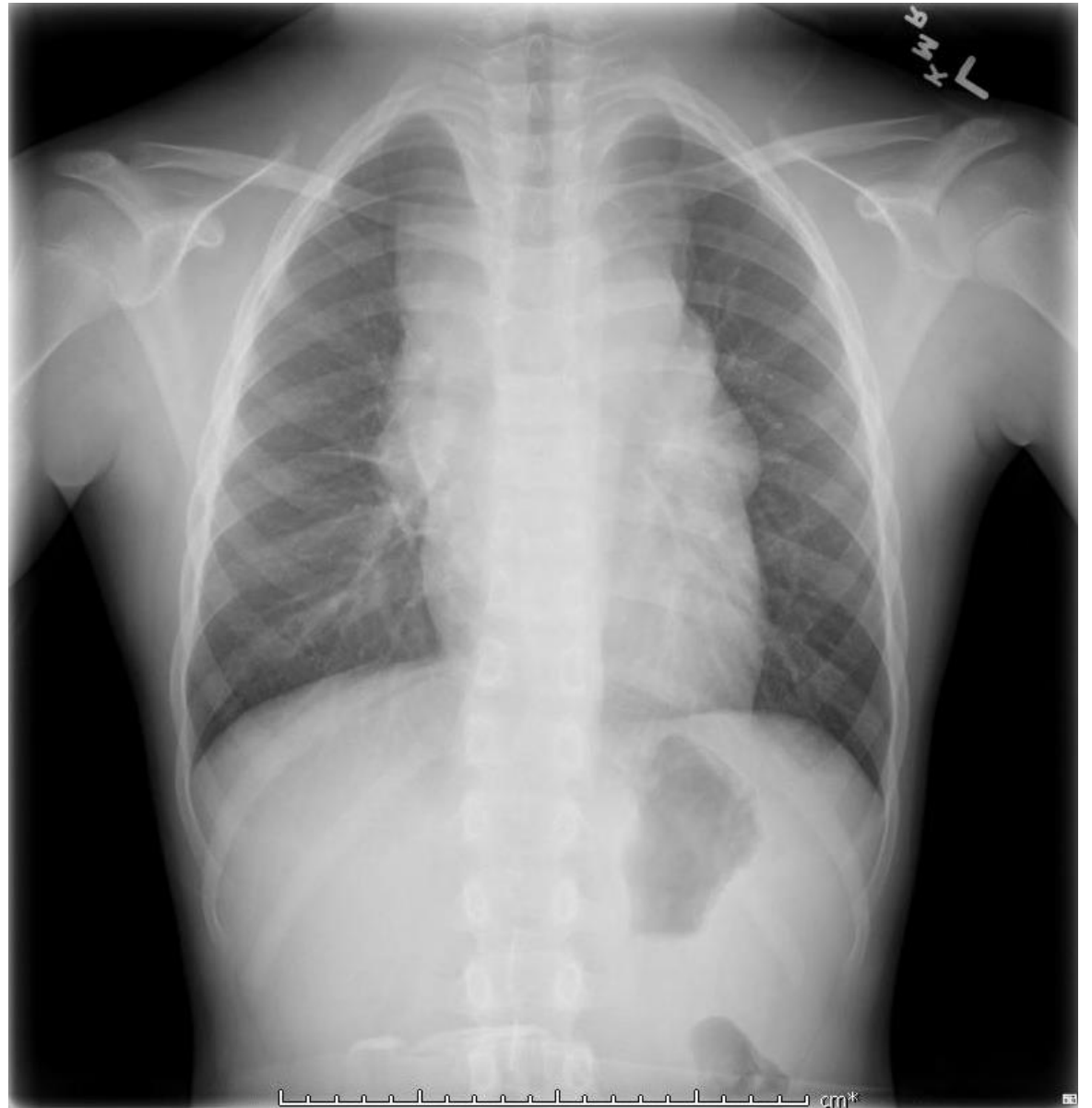
Case 4

- 10 yo male with h/o eczema presents with swollen face that seems to be more prominent today. Parents deny any known food allergies. They did attend a family birthday party yesterday, but patient asked to go home early and seemed tired.
- On exam:
 - Afebrile and anxious appearing
 - RR 24, Lung aeration diminished L>R
 - HR 120s, BP 100/74 with soft heart tones. No murmurs. Pulses equal throughout
 - Abdomen non tender and soft
 - Swelling to neck and face, spares rest of body



Case 4

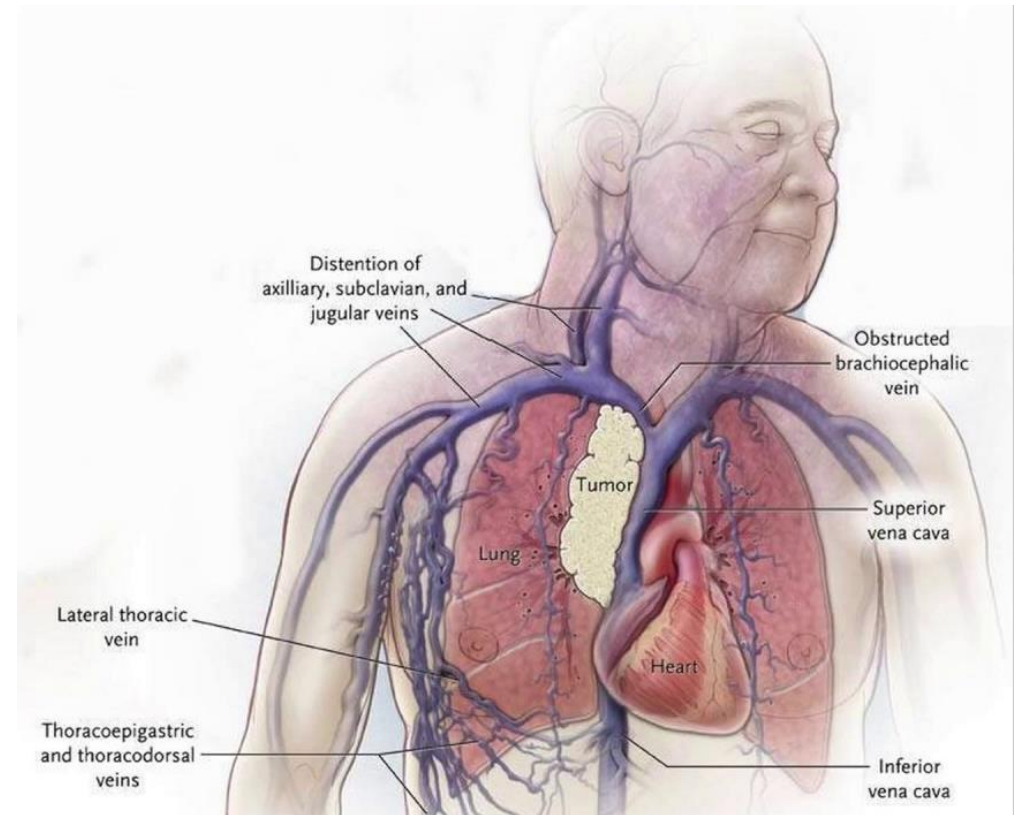
- IM epinephrine administered with no improvement in swelling
- CXR obtained due to differential lung exam
- CT chest ordered, but patient has trouble with increased anxiety laying supine in scanner



Case 4 – Pathophysiology

Diagnosis: Superior Vena Cava (SVC) syndrome in setting of anterior mediastinal mass

- Displacement/compression of anatomical structures vs. thrombus/tumor within the SVC
 - Airway compression
 - Compression of great vessels
- Impaired venous return



Wilson, L. et al. (May, 2007). *New England Journal of Medicine*.

Case 4- SVC syndrome in setting of anterior mediastinal mass

- Progressive venous congestion and airway compression leads to:
 - Facial engorgement
 - Headache
 - Plethora
 - Cyanotic facies
 - Dyspnea/orthopnea
 - Stridor/hoarseness or dysphagia with or without effusions

Case 4- Anterior mediastinal mass

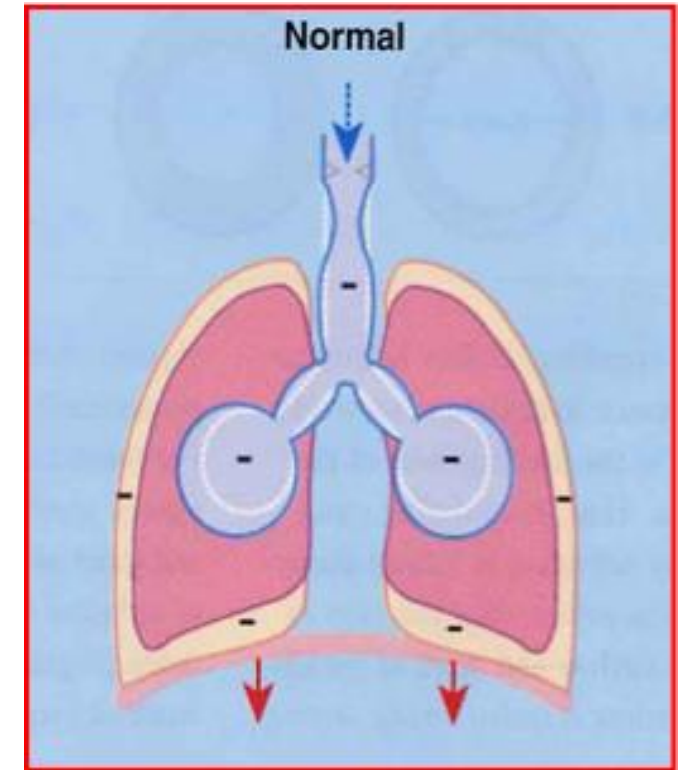
- Similar pathophysiology as SVC syndrome and not mutually exclusive
- 50% of mediastinal masses are anterior
- Can present with pericardial and pleural effusions
- Lethality arises from compression of vital structures
 - Great vessels
 - Trachea, carina and main bronchi



Singh, B. (2021). Radiopaedia.org, rID: 8593

Case 4- Complications with intubating the patient with anterior mediastinal mass with or without SVC syndrome

- Physiology
 - Loss of airway tone with sedating agents
 - Elimination of normal transpleural pressure gradient that stents airways
- Risk factors for airway compromise:
 - >50% decrease in cross sectional area of trachea on CT
 - Peak expiratory flow <50% of predicted value
 - Compression at level of the carina or significant bronchus compression
- Intubation with paralytic/ deep sedation can risk immediate irreversible cardiorespiratory collapse

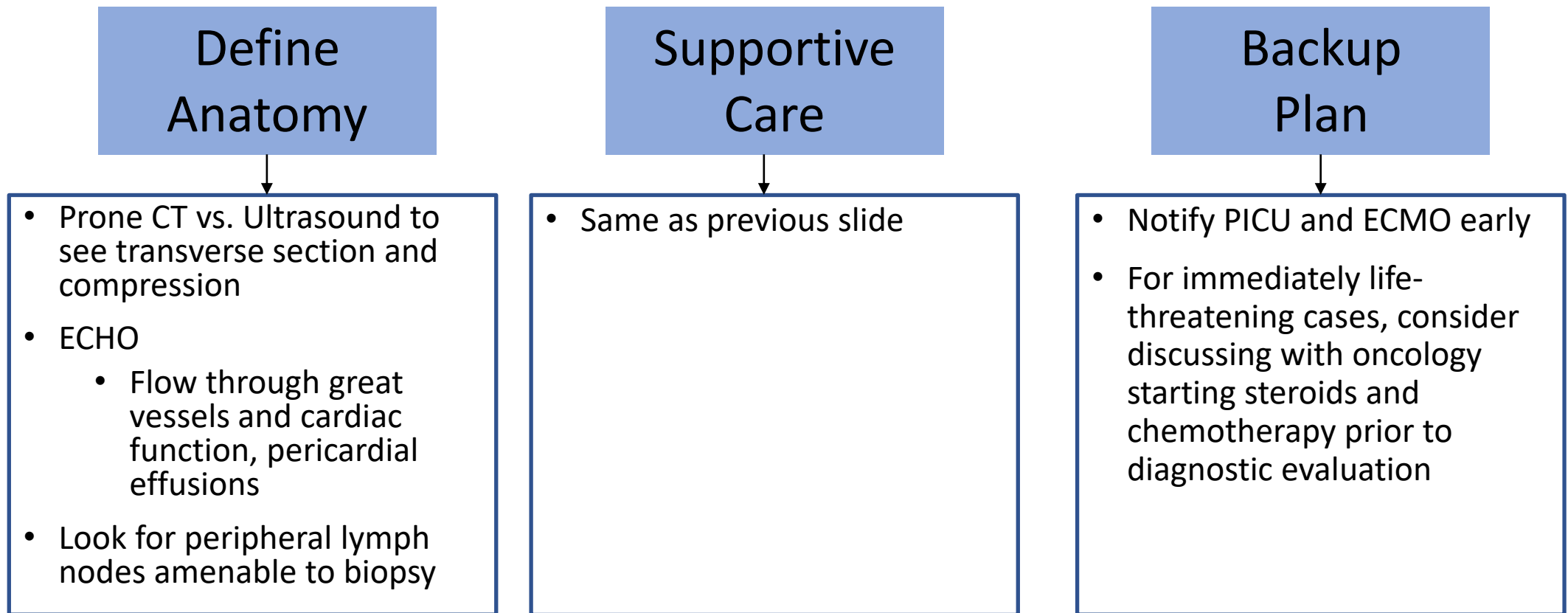


Case 4- Anterior Mediastinal Mass/SVC Syndrome- Supportive Measures

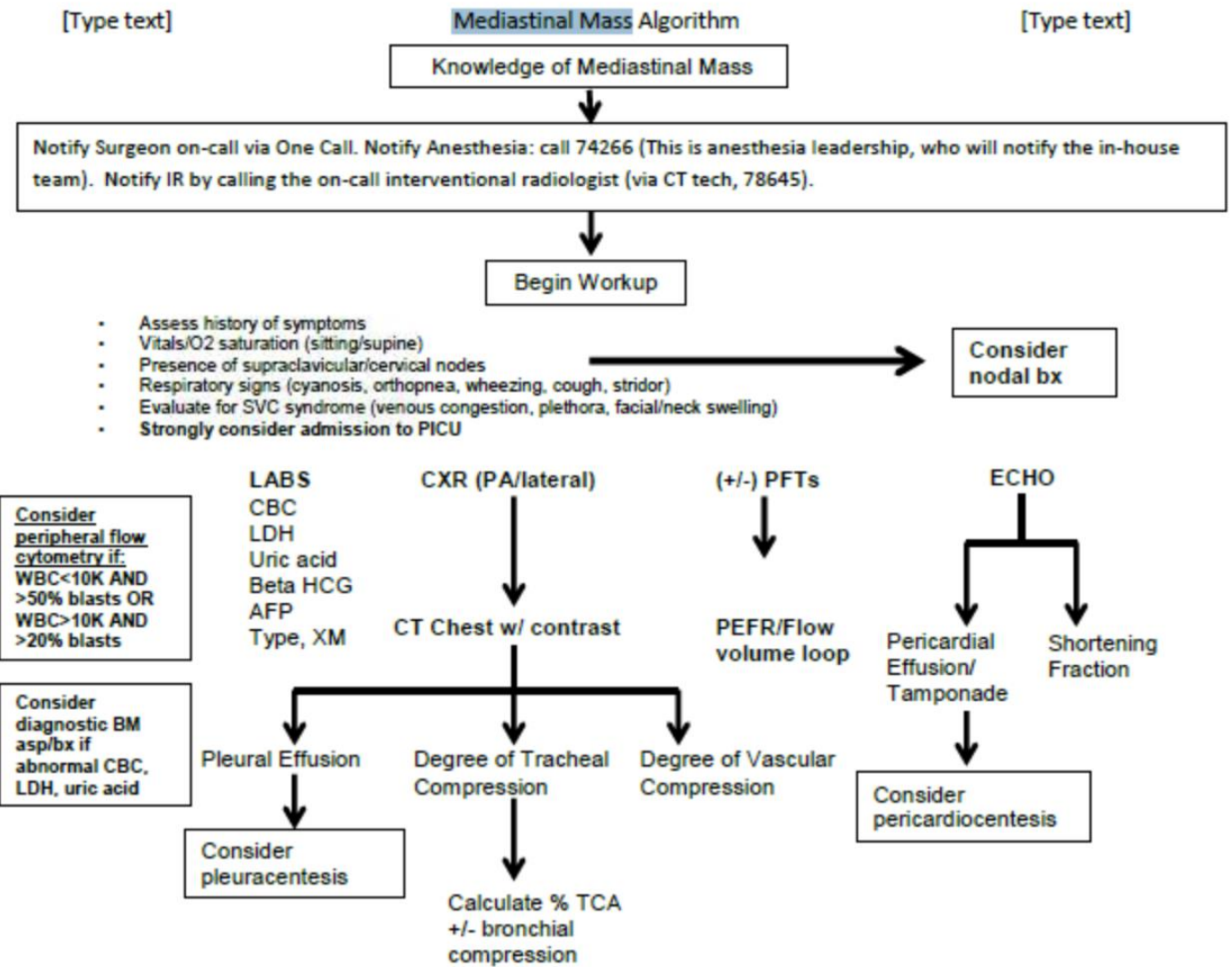
- Keep head of bed elevated
- Avoid agitation
- Keep patient awake- **do not sedate**
 - Do not take away patient's intrinsic effort to breathe
- Avoid hypotension and fluid overload
- Access in upper and lower extremities
- PICC if central access needed

Case 4- Anterior Mediastinal Mass- Further management

- Three main steps in initial management upon admission to PICU
 - Notify mediastinal mass algorithm/team



Children's Hospital Colorado Mediastinal Mass Algorithm



Anesthetic Technique	Reassuring; GA if needed	Concerning; Avoid GA
Symptoms	Asymptomatic	Significant, esp if orthopnea/SVC syndrome
CT Findings	Minimal tracheobronchial compression	Tracheobronchial diameter < 50% normal regardless of symptoms
Echo Findings	No tamponade physiology or compression of major vessels	Echo showing tamponade physiology or compression of major vessels

*other combination of symptoms and test abnormalities will be considered at anesthesia's discretion in discussion with multi-disciplinary team

Case 4- Anterior mediastinal mass and SVC syndrome- pearls

- If unable to be supine for CT scan, can adjust to prone or side positioning to obtain images
- Avoid intubation
 - Survival physiology and patency of airways rely on patient's intrinsic respiratory drive and negative pressure breathing
- Keep patient as calm as able while avoid sedating medications
- Involve ECMO, PICU and mediastinal mass team as soon as possible

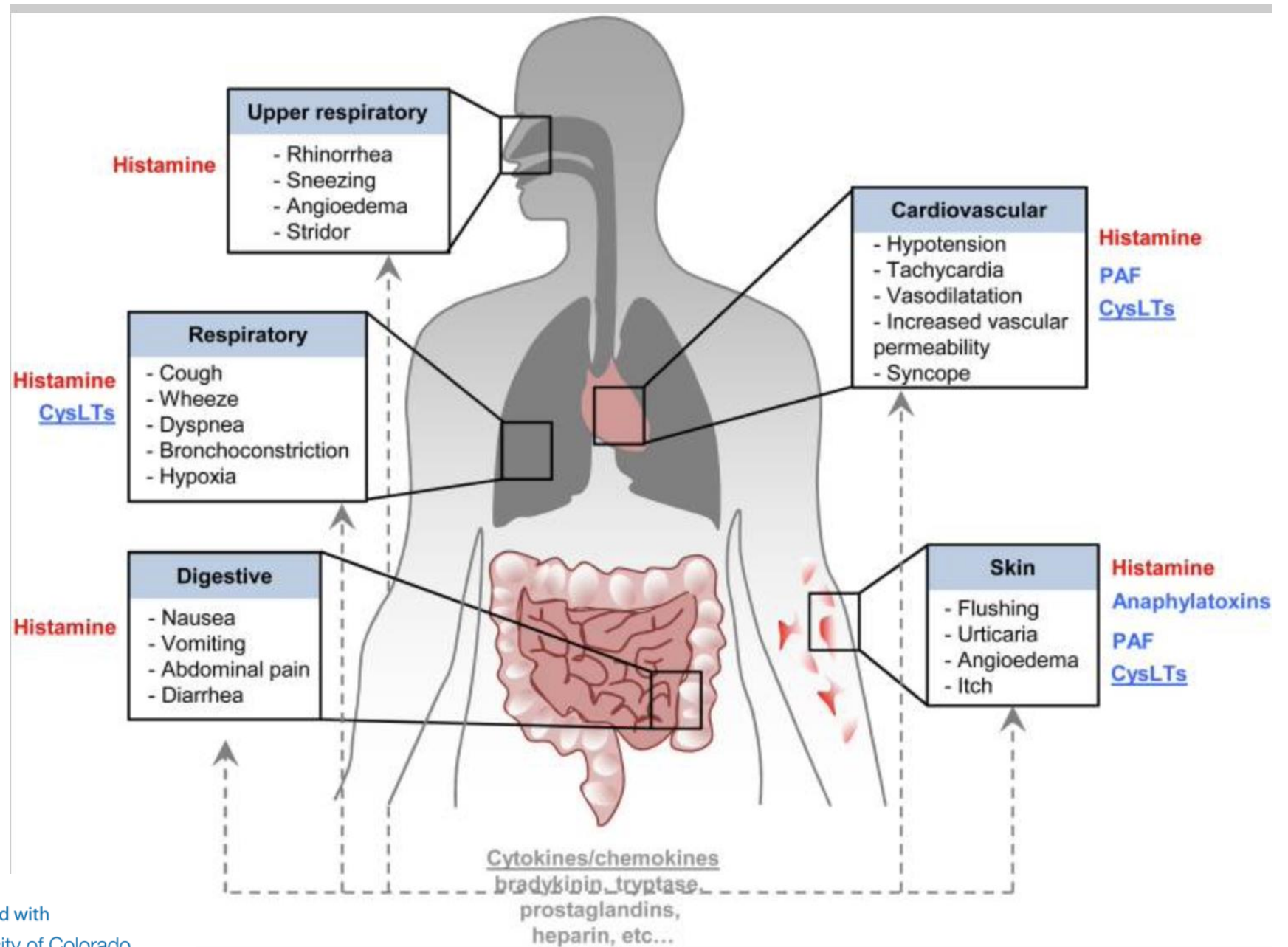
Case 5



Case 5

- 6 yo female with eczema and allergy to tree nuts presents with vomiting and swollen eyes and ears after attending Halloween trunk or treat event at school.
- On exam:
 - Anxious and uncomfortable appearing
 - RR 24 with occasional wheeze on auscultation. Otherwise with good air movement. Mildly increased work of breathing
 - HR 130s. BP 72/50 with warm extremities and flash cap refill in finger tips
 - Diffuse flushing over trunk
 - Abdomen diffusely tender to palpation

Case 5- Anaphylaxis Pathophysiology



Case 5- Anaphylaxis-diagnosis

Anaphylaxis is highly likely when any ONE of the following three criteria is fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula)

AND AT LEAST ONE OF THE FOLLOWING:

A. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, hypoxemia)

B. Reduced BP* or associated symptoms of end-organ dysfunction (eg, hypotonia, collapse, syncope, incontinence)

2. TWO OR MORE OF THE FOLLOWING that occur rapidly after exposure to a LIKELY allergen for that patient (minutes to several hours):

A. Involvement of the skin mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula)

B. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, hypoxemia)

C. Reduced BP* or associated symptoms (eg, hypotonia, collapse, syncope, incontinence)

D. Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)

3. Reduced BP* after exposure to a KNOWN allergen for that patient (minutes to several hours):

A. Infants and children - Low systolic BP (age-specific)* or greater than 30% decrease in systolic BP

B. Adults - Systolic BP of less than 90 mmHg or greater than 30% decrease from that person's baseline

Case 5- Anaphylaxis- Causes

Allergens (IgE-dependent immunologic mechanism)
Foods, especially peanut, tree nut, crustacean shellfish, finned fish, cow's milk, hen's egg
Insect stings (eg, Hymenoptera venom) and insect bites (eg, kissing bugs)
Medications (eg, antibiotics, NSAIDs)
Biologic materials, including allergen immunotherapy, monoclonal antibodies, chemotherapy agents, and vaccines*
Natural rubber latex
Food additives, including spices, insect-derived colorants (eg, carmine), and vegetable gums
Inhalants (rare; eg, horse dander, cat dander, grass pollen)
Human seminal fluid (rare trigger of anaphylaxis in women)
Occupational allergens (eg, stinging insects, natural rubber latex)
Immunologic triggers (IgE-independent mechanism)
IgG dependent (rare; eg, to high-molecular-weight dextran, infliximab)
Coagulation system activation (eg, heparin contaminated with oversulfated chondroitin sulfate)
Idiopathic anaphylaxis
Consider the possibility of a hidden or previously unrecognized trigger
Consider the possibility of a mast cell activation syndrome, including systemic mastocytosis
Nonimmunologic triggers (direct activation of mast cells and basophils)
Physical factors (eg, exercise [¶] , cold, heat)
Medications (eg, opioids, NSAIDs)
Radiocontrast agents
Alcohol (ethanol; may augment, rarely induces)

Case 5- Anaphylaxis- management

- Early treatment is critical
 - Anaphylaxis more responsive to treatment in early phases
 - In case series of 164 anaphylaxis fatalities, time interval between symptoms and cardiorespiratory arrest was 5-30 minutes (Pumphrey, R. August, 2000. Clinical & Experimental Allergy)
- Key components of early management
 - Removal of trigger
 - Call for additional help if needed
 - IM injection of epi immediately followed by additional dosages of epi as needed (IV epi PRN or gtt also options)
 - Supplemental O2, albuterol if needed
 - Volume resuscitation with IV fluids

Case 5- Anaphylaxis- management

Rapid overview: Emergency management of anaphylaxis in infants and children*

Diagnosis is made clinically:

The most common signs and symptoms are cutaneous (eg, sudden onset of generalized urticaria, angioedema, flushing, pruritus). However, 10 to 20% of patients have no skin findings.

Danger signs: Rapid progression of symptoms, evidence of respiratory distress (eg, stridor, wheezing, dyspnea, increased work of breathing, retractions, persistent cough, cyanosis), signs of poor perfusion, abdominal pain, vomiting, dysrhythmia, hypotension, collapse.

Acute management:

The first and most important therapy in anaphylaxis is epinephrine. There are **NO absolute contraindications to epinephrine** in the setting of anaphylaxis.

Airway: Immediate intubation if evidence of impending airway obstruction from angioedema. Delay may lead to complete obstruction. Intubation can be difficult and should be performed by the most experienced clinician available. Cricothyrotomy may be necessary.

IM epinephrine (1 mg/mL preparation): Epinephrine 0.01 mg/kg should be injected intramuscularly in the mid-outer thigh. For large children (>50 kg), the maximum is 0.5 mg per dose. If there is no response or the response is inadequate, the injection can be repeated in 5 to 15 minutes (or more frequently). If epinephrine is injected promptly IM, patients respond to one, two, or at most, three injections. If signs of poor perfusion are present or symptoms are not responding to epinephrine injections, prepare IV epinephrine for infusion (see below).

Place patient in recumbent position, if tolerated, and elevate lower extremities.

Oxygen: Give 8 to 10 L/minute via facemask or up to 100% oxygen, as needed.

Normal saline rapid bolus: Treat poor perfusion with rapid infusion of 20 mL/kg. Re-evaluate and repeat fluid boluses (20 mL/kg), as needed. Massive fluid shifts with severe loss of intravascular volume can occur. Monitor urine output.

Albuterol: For bronchospasm resistant to IM epinephrine, give albuterol 0.15 mg/kg (minimum dose: 2.5 mg) in 3 mL saline inhaled via nebulizer. Repeat, as needed.

H1 antihistamine: Consider giving diphenhydramine 1 mg/kg (max 50 mg IV, over 5 minutes) or cetirizine (children aged 6 months to 5 years can receive 2.5 mg IV, those 6 to 11 years of age can receive 5 or 10 mg IV, over 2 minutes).

H2 antihistamine: Consider giving famotidine 0.25 mg/kg (max 20 mg) IV, over at least 2 minutes.

Glucocorticoid: Consider giving methylprednisolone 1 mg/kg (max 125 mg) IV.

Monitoring: Continuous noninvasive hemodynamic monitoring and pulse oximetry monitoring should be performed. Urine output should be monitored in patients receiving IV fluid resuscitation for severe hypotension or shock.

Case 5- Anaphylaxis- pearls

- Treat early
- If slight suspicion of anaphylaxis, give IM epi!
 - Risk of death with anaphylaxis outweighs the few temporary side effects from IM epi
- All the hard work and treatment is usually administered by the time the patient arrives to the PICU

Patient cases

- All patients featured in the case presentations survived to be discharged from the hospital

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Thank you!

