EMERGENCIES AND SEPSIS

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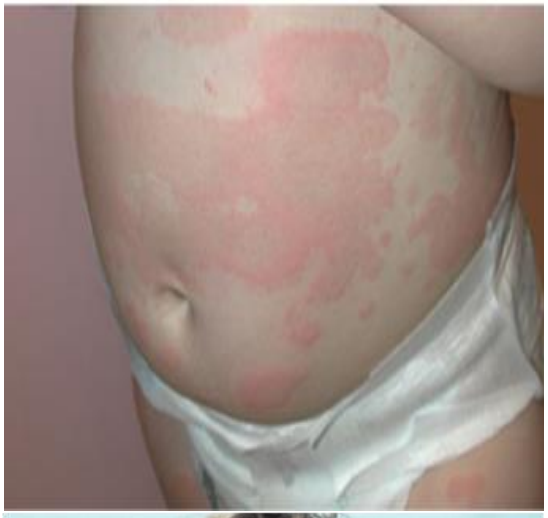
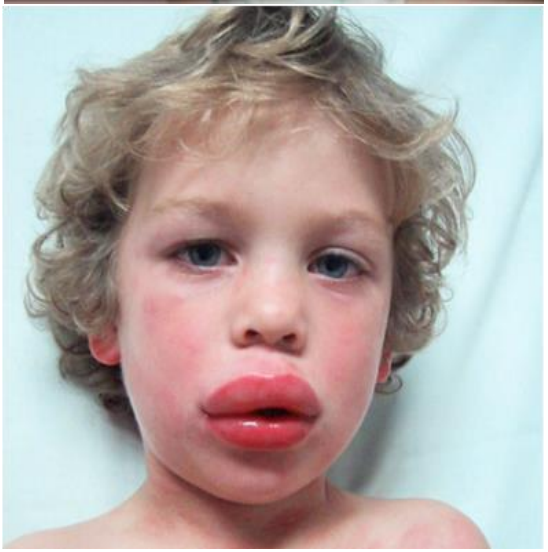
Anaphylaxis

Definition:

- Rapid-onset (minutes to hours) usually immunoglobulin E (IgE)-mediated systemic allergic reaction involving multiple organ systems, including two or more of the following:

1. **Cutaneous/mucosal** (**80% to 90%):** flushing, urticaria, pruritis, angioedema
2. **Respiratory** (70%): laryngeal edema, bronchospasm, dyspnea, wheezing, stridor, hypoxemia
3. **Gastrointestinal** (45%): vomiting, diarrhea, nausea, crampy abdominal pain
4. **Circulatory** (45%): tachycardia, hypotension, syncope





Anaphylaxis

Management:

1. **Stop exposure** to precipitating antigen.

2. While performing **A-B-Cs**, immediately give intramuscular **(IM) epinephrine**.
   - For child, administer **0.01 mg/kg of 1 mg/mL solution** up to a max dose of 1 mg/dose.
   - Repeat dosing every 5 to 15 minutes as needed.

3. Provide **oxygen and ventilatory assistance**. Consider early endotracheal intubation.

4. Obtain **IV access**. For management of shock, resuscitate with **20 mL/kg isotonic crystalloid fluid boluses and vasoactive agents** as needed.

5. Place patient in **Trendelenburg position** (head 30 degrees below feet).

6. Consider adjuvant pharmacologic agents:
   I. **Histamine receptor antagonist:** Diphenhydramine or chlorphenaramine **(H1-antagonism)** and ranitidine/famotidine (**H2-antagonism**)
   II. **Corticosteroid:** Methylprednisolone or dexamethasone
   III. **Inhaled β2 agonist:** Albuterol.

Remember


Anaphylaxis kills...
#EM3
East Midlands Emergency Medicine Educational Media
First line treatment is
IM adrenaline
NOT salbutamol or
antihistamines

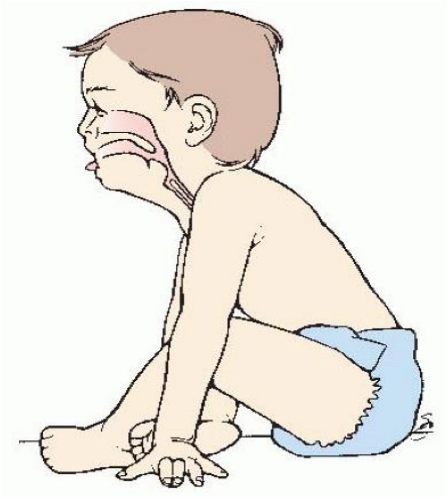
Anaphylaxis

- **Symptoms may recur** (“biphasic anaphylaxis”) **up to 72 hours** after initial recovery.
  - Observe for a minimum of 4 to 10 hours for late-phase symptoms.
  - Discharge with an epinephrine auto-injector and an anaphylaxis action plan.

Epiglottitis

Definition:

- Life-threatening, rapidly progressive inflammation (**usually infectious**) of the supraglottic region.
- May be caused by **infection, thermal injury, caustic ingestion, or foreign body**.
- Most common infectious organisms include ***Haemophilus influenzae*** **(unvaccinated)**, ***Streptococcus pneumoniae****,* group A streptococci, and *Staphylococcus aureus.*
- Patients often present **febrile, toxic-appearing, and tripoding in respiratory distress (Dyspnea)**. **Drooling, Dysphagia and inspiratory stridor (with Dysphonia)** are common. **Barky cough is absent**.



4Ds

Epiglottitis

Management:

1. **Avoid *any agitation*** of the child prior to securing airway to prevent impending complete obstruction.

2. Allow a **position of comfort**. Provide **oxygen**. Monitor with **pulse oximetry.**

3. Initiate **broad-spectrum antibiotic therapy** (e.g., **vancomycin and Ceftriaxone**).

Epiglottitis

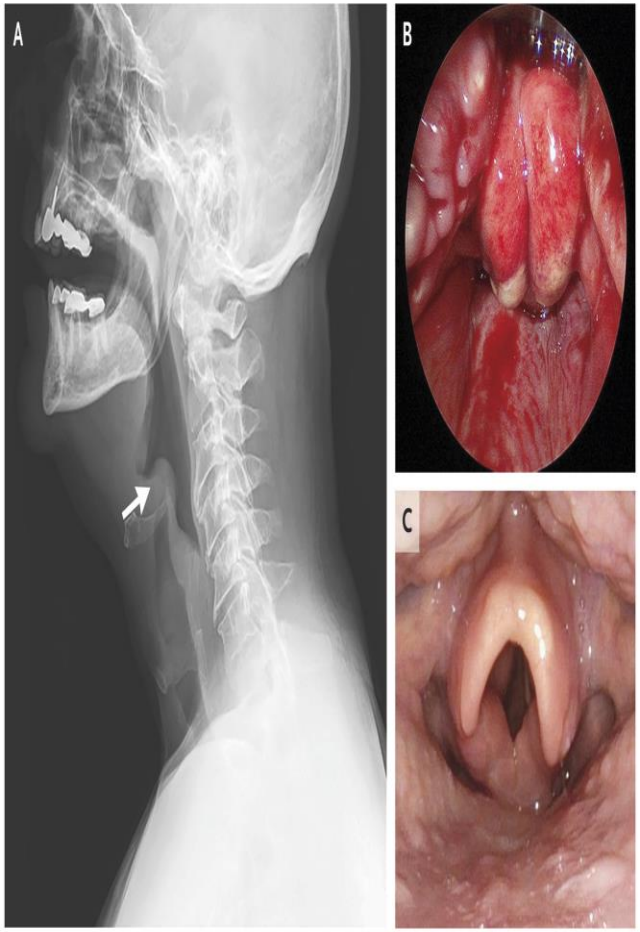
Management:

4. **To secure airway**, **emergently consult difficult airway personnel**.

1. If **unstable** (unresponsive, cyanotic, bradycardic), **emergently intubate**.

2. **If stable with high suspicion**, **send patient to Operation Room for laryngoscopy and intubation under general anesthesia**.

3. **If stable with moderate or low suspicion**, obtain **lateral neck radiograph** to assess for "**thumb sign**".



Croup

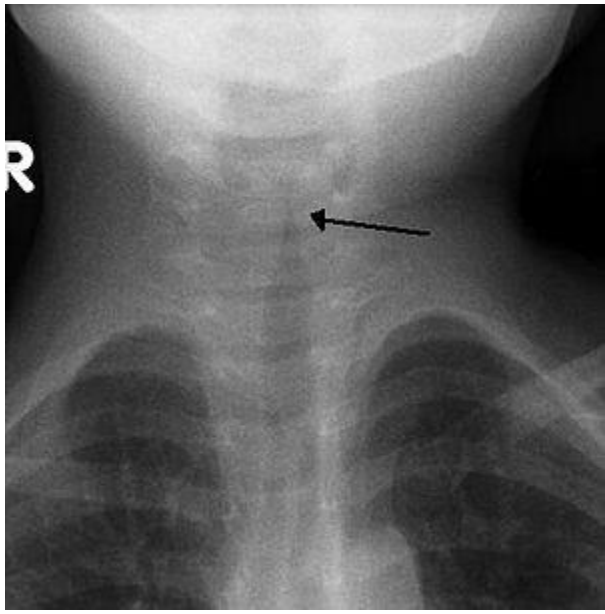
Definition:

- Common infectious inflammation of the subglottic area.
- Most common in infants **aged 6 to 36 months**.
- 75% of infections are caused by **parainfluenza virus**.
- Patients present with **fever, barking cough, inspiratory stridor, and increased work of breathing, often worse at night**.

Croup

Management:

- **oxygen** to children with hypoxemia or severe respiratory distress. Use **humidified air**, although current consensus suggests it is ineffective for mild to moderate disease.



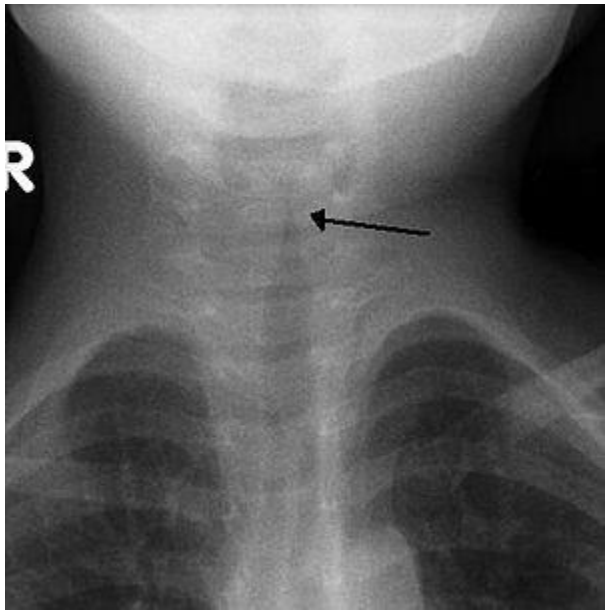
A. If **no stridor at rest**,
   1. give single dose **dexamethasone (IM or PO)**.
   2. **nebulized budesonide** in patients vomiting or who lack IV access.

B. If **stridor at rest**,
   1. give **dexamethasone and nebulized racemic epinephrine**.
   2. **Observe for 2 to 4 hours** given short duration of action of nebulized epinephrine.

Croup

Management

- Indications for hospitalization include
  1. >1 racemic epinephrine nebulization required,
  2. atypical age (<6 months),
  3. severe respiratory distress,
  4. dehydration.



- **Heliox** (helium and oxygen mixture) to improve turbulent airflow in moderate to severe croup, although benefit is controversial.

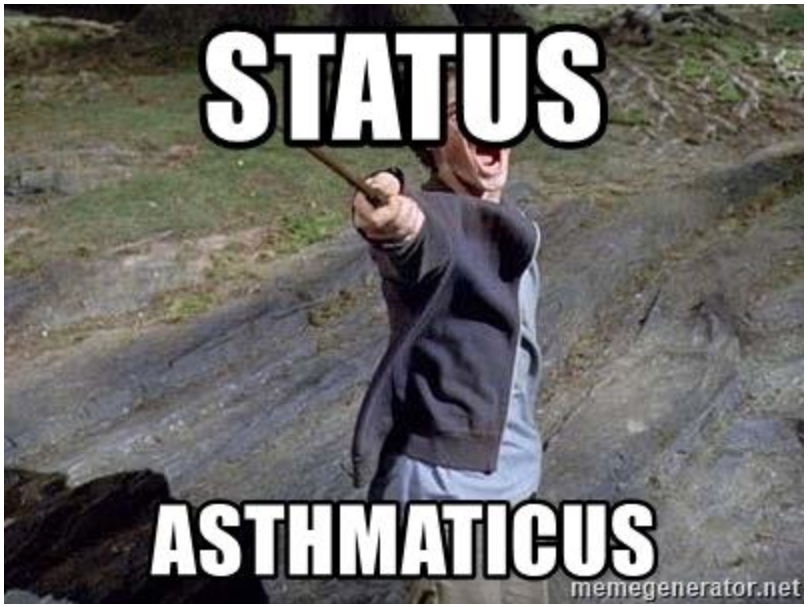
Status Asthmaticus

Definition:

It is the acute and sub-acute worsening in symptoms and lung function from the patient's usual status for an asthmatic patients.

Examination:

- Assess breathlessness,
- speech,
- alertness,
- respiratory rate,
- accessory muscle use,
- wheezing,
- HR,
- pulsus paradoxus,
- peak expiratory flow,
- SpO2 and pCO2.



Status Asthmaticus

Box 6-9. Initial assessment of acute asthma exacerbations in children 5 years and younger

Symptoms	Mild	Severe*
Altered consciousness	No	Agitated, confused or drowsy
Oximetry on presentation (SaO_2)**	>95%	<92%
Speech†	Sentences	Words
Pulse rate	<100 beats/minute	>200 beats/minute (0–3 years)
		>180 beats/minute (4–5 years)
Central cyanosis	Absent	Likely to be present
Wheeze intensity	Variable	Chest may be quiet

*Any of these features indicates a severe asthma exacerbation. **Oximetry before treatment with oxygen or bronchodilator.
† The normal developmental capability of the child must be taken into account.

Blood gas is important test to assess the severity of asthma exacerbation

Status Asthmaticus

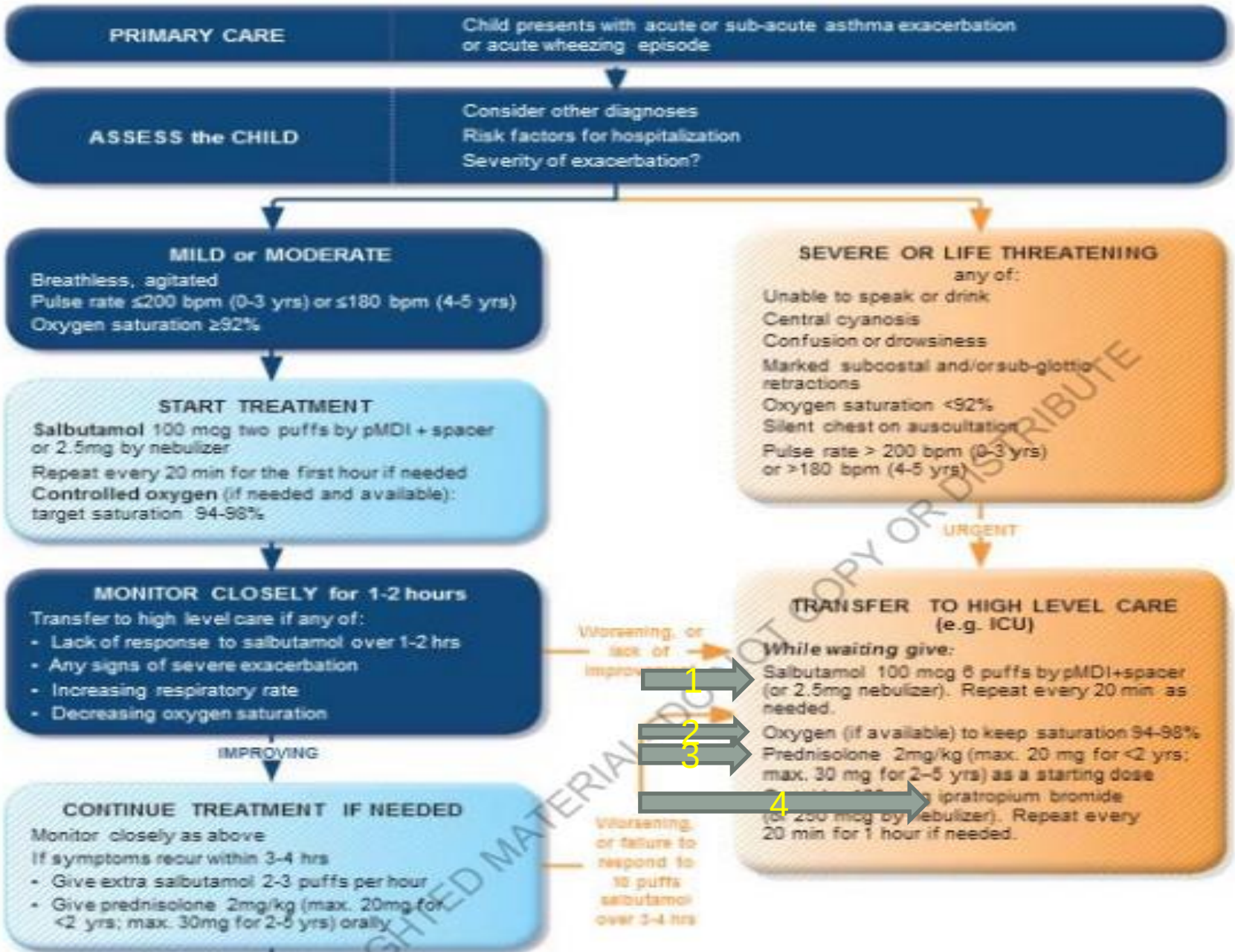
Management:

- Provide **oxygen** to achieve SpO2 ≥90%. If hypoxemia not readily corrected with supplemental oxygen, consider other complications.

- **Pharmacologic agents** used in acute asthma exacerbations.

- Ventilation interventions:
  - Normalizing pCO2 can be a sign of impending respiratory failure.
  - NIPPV (e.g., BiPAP) may be used in patients with impending respiratory failure to avoid intubation.
  - Intubation should be approached cautiously. The Indications include:
    1. severe airway obstruction,
    2. markedly increased work of breathing,
    3. refractory hypoxemia,
    4. impending respiratory arrest.
  - Consider inhaled anesthetics or ECMO as rescue therapies.

Box 6-8. Primary care management of acute asthma or wheezing in children 5 years and younger



STATUS ASTHMATICUS MEDICATIONS[24-28]

Medication	Dose	Comments
Short-acting β_2 agonist		
Albuterol	**Mild to Moderate:** Administer up to 3 doses in the first hour MDI: 4—8 puffs (90 mCg/puff) q20 min—4 hr Nebulizer: 0.15 mg/kg (min 2.5 mg, max 5 mg) q20 min—4 hr **Severe:** Continuous nebulization: 0.5 mg/kg/hr (max 30 mg/hr)	Inhaler (with spacer) is preferred delivery method given equal or greater efficacy, fewer side effects, and shorter length of stay
Anticholinergics		
Ipratropium bromide	Administer q20 min for 3 doses with albuterol MDI: 4—8 puffs (17 mCg/puff) Nebulizer: 0.25—0.5 mg	No additional benefit shown in inpatient setting
Systemic corticosteroids		
Dexamethasone	**Mild to Moderate:** 0.6 mg/kg/day PO/IV/IM for 1—2 days (max 16 mg/day)	Equally as efficacious as prednisone or prednisolone with fewer side effects, better compliance and palatability
Prednisone, Prednisolone	**Mild to Severe:** 2 mg/kg/day PO for 5—7 days (max 60 mg/day)	Taper if course ≥7 days or bounce back from recent exacerbation
Methylprednisolone	**Severe:** Loading: 2 mg/kg IV (max 60 mg) Maintenance: 2 mg/kg/day IV divided q6—12hr (max <12 years 60 mg/day, ≥12 years 80 mg/day)	No known advantage in severe exacerbations for higher dosing or IV administration over oral therapy, provided normal GI transit and absorption
Injected β_2 agonist		
Epinephrine	0.01 mg/kg of 1 mg/mL IM (max 1 mg) q15—20 min for up to 3 doses	Consider for severe exacerbation with minimal air entry Consider quickly accessed autoinjector
Terbutaline	SC: 0.01 mg/kg (max 0.25 mg/dose) q20 min for up to 3 doses, then as needed q2—6 hr IV load: 2—10 mCg/kg IV IV continuous: 0.1—0.4 mCg/kg/min (doses as high as 10 mCg/kg/min have been used)	Consider for severe exacerbation with minimal air entry IV administration may decrease the need for mechanical ventilation
Adjunct therapies		
Magnesium sulfate	25—75 mg/kg/dose IV (max 2 g), infuse over 20 min	Smooth muscle relaxant May cause hypotension; consider simultaneous fluid bolus Reduces hospitalization rates in severe exacerbations

Medication	Dose	Comments
Ketamine	1–2 mg/kg IV bolus followed by 1 mg/kg/h infusion, titrated to affect	Used as a sympathomimetic adjuvant in effort to avoid endotracheal intubation Preferred induction-sedative agent for endotracheal intubation in asthma
Aminophylline	6 mg/kg IV bolus over 20 min followed by 0.5–1.2 mg/kg/h infusion (age-dependent, see formulary)	Use limited to severe exacerbations refractory to traditional interventions May improve lung function and oxygen saturation but is associated with greater length of stay and time to symptom improvement
Heliox	Optimal helium-oxygen ratio unknown, most commonly 70:30 or 80:20 mixture	Low density gas that promotes laminar airflow and improves β_2 agonist delivery to distal airways Useful in severe or very severe exacerbations
Inhaled anesthetics (e.g., halothane, isoflurane, sevoflurane)	Consultation with pediatric anesthetist recommended	Rescue therapy for intubated patients with life-threatening exacerbation Associated with prolonged length of stay and increased cost Isoflurane may cause hypotension Sevoflurane may cause renal tubular injury, hepatotoxicity, neuropathy

Status Epilepticus

Definition:

- Prolonged seizure (clinical or electrographic) or recurrent seizure activity without return to baseline lasting **5 minutes** or more.
  A. Common acute etiologies: febrile seizures, metabolic disturbances, sepsis, head trauma, stroke/hemorrhage, drug toxicity, inadequate antiepileptic therapy, hypoxia, hypertensive encephalopathy, autoimmune encephalitis

  B. Common chronic etiologies: preexisting epilepsy, tumor, stroke, inborn error of metabolism, ethanol abuse

Status Epilepticus

STATUS EPILEPTICUS TREATMENT GUIDELINE[33-34]

IMMEDIATE APPROACH (0–5 min)

Management:
Protect airway, intubate if needed
Assess vitals
Bedside fingerstick blood glucose

ABCs

Put in left lateral position

Establish peripheral IV access: administer emergent AED, fluid resuscitation, nutrient resuscitation (thiamine, dextrose)

Labs: laboratory blood glucose, CBC, BMP, calcium, magnesium, antiseizure medication drug levels, Na

Medication	Dose	Comment
Diazepam (Valium)	0.15–0.5 mg/kg IV (max 10 mg/dose) 2–5 years: 0.5 mg/kg PR (max 20 mg/dose) 6–11 years: 0.3 mg/kg PR (max 20 mg/dose) ≥12 years: 0.2 mg/kg PR (max 20 mg/dose) May repeat dose once in 5 min	Monitor for hypotension, respiratory depression Can be repeated 3 times 5 minutes apart
Lorazepam (Ativan)	0.1 mg/kg IV (max 4 mg/dose) May repeat dose once in 5–10 min	Monitor for hypotension, respiratory depression
Midazolam (Versed)	0.2 mg/kg IM/IN 0.5 mg/kg buccal Max: 10 mg all forms Single dose recommended	Monitor for hypotension, respiratory depression

Status Epilepticus

URGENT APPROACH (5–15 min)

Management:
Secondary AED control therapy
Initiate vasopressor support if indicated
Neurological examination
CT if indicated
Labs: Liver function tests, coagulation studies, toxicology screen, inborn error of metabolism screening
Neurologic consultation

Medication	Dose	Comment
Fosphenytoin	20 mg PE/kg IV/IM (max 1500 mg PE/24 hr) May give additional 5 mg PE/kg repeat dose	Monitor for arrhythmia, hypotension
Levetiracetam (Keppra)	20–60 mg/kg IV (max 4500 mg/dose)	Minimal drug interactions Not hepatically metabolized
Phenytoin	20 mg/kg IV (max 1500 mg/24 hr) May give additional 5–10 mg/kg repeat dose	Monitor for arrhythmia, hypotension, purple glove syndrome
Phenobarbital	15–20 mg/kg IV (max 1000 mg) May give additional 5–10 mg/kg repeat dose	Monitor for hypotension, respiratory depression
Valproic Acid	20–40 mg/kg IV May give additional 20 mg/kg repeat dose (max 3000 mg/dose)	Use with caution in TBI Monitor for hyperammonemia, pancreatitis, hepatotoxicity, thrombocytopenia

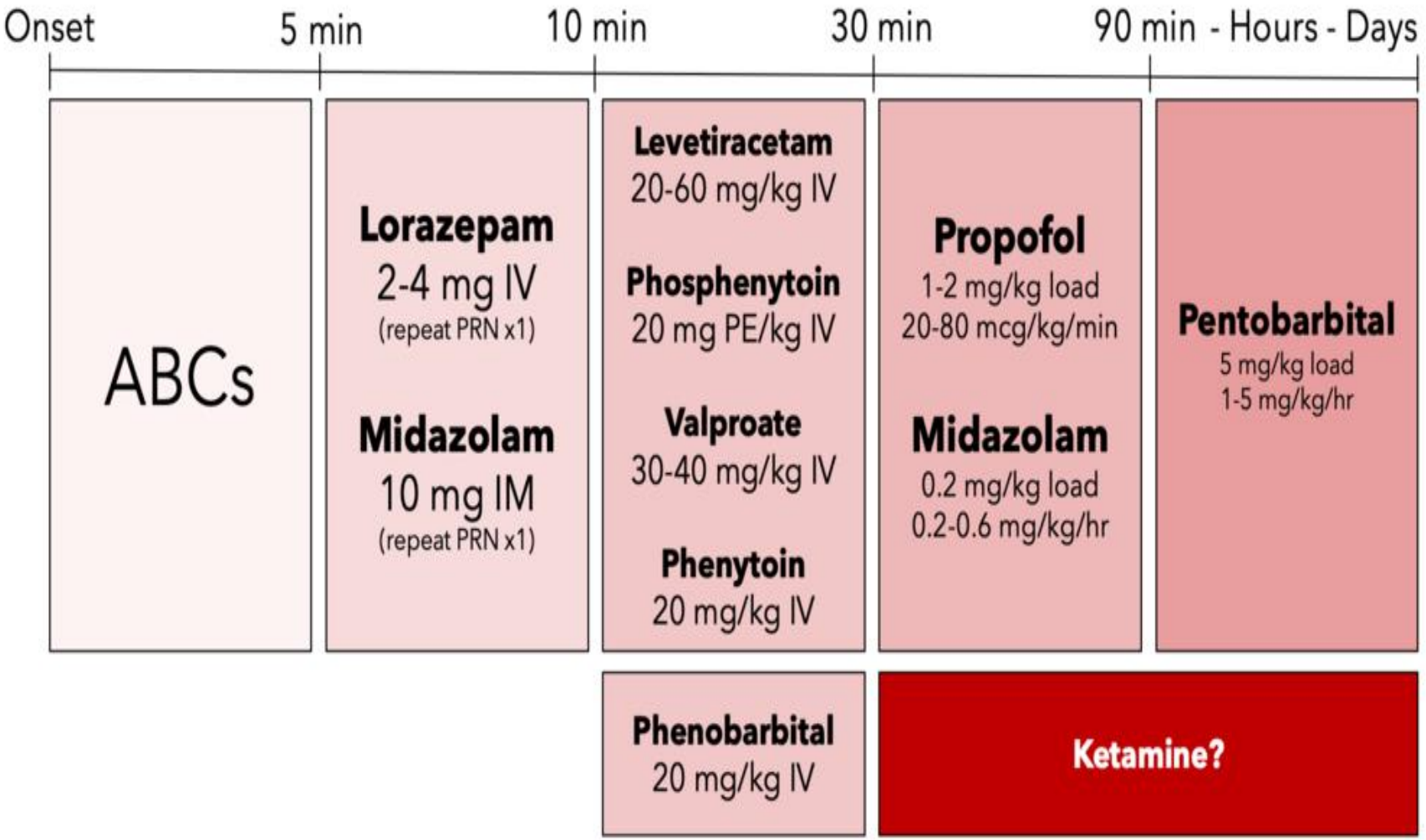
Status Epilepticus

REFRACTORY APPROACH (15–60 min)

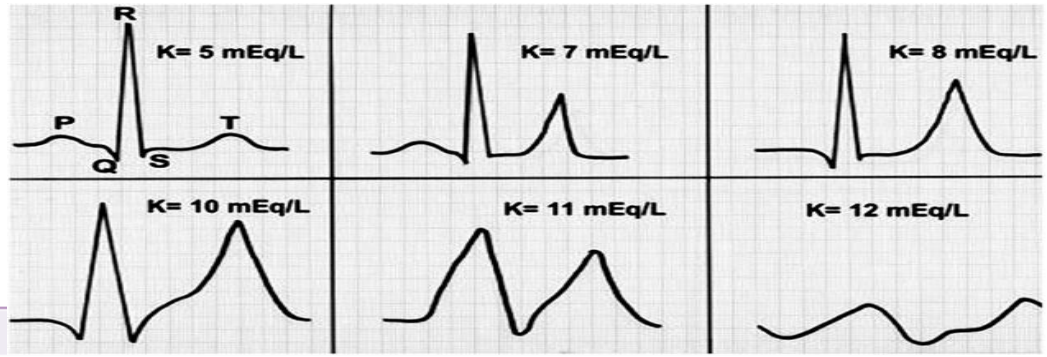
Management:
Refractory AED control therapy
Continuous EEG monitoring if indicated
MRI if indicated
Lumbar puncture if indicated
Consider broad-spectrum antibiotics and antivirals if indicated
Intracranial pressure monitoring if indicated
Urinary catheter

Medication	Dose	Comment
Midazolam (continuous infusion)	Load: 0.2 mg/kg Infusion: 0.05–2 mg/kg/hr Breakthrough: 0.1–0.2 mg/kg bolus	Tachyphylaxis with prolonged use Monitor for respiratory depression, hypotension
Pentobarbital	Load: 5–15 mg/kg Infusion: 0.5–5 mg/kg/hr Breakthrough: 5 mg/kg bolus	Monitor for hypotension, respiratory depression, cardiac depression, paralytic ileus
Propofol	Load: 1–2 mg/kg Infusion: 20–65 mCg/kg/min Breakthrough: 1 mg/kg bolus	Monitor for hypotension, respiratory depression, cardiac failure, rhabdomyolysis, metabolic acidosis, renal failure, hypertriglyceridemia, pancreatitis (propofol related infusion syndrome)

Status Epilepticus



Hyperkalemia



HYPERKALEMIA[7]

CLINICAL MANIFESTATIONS

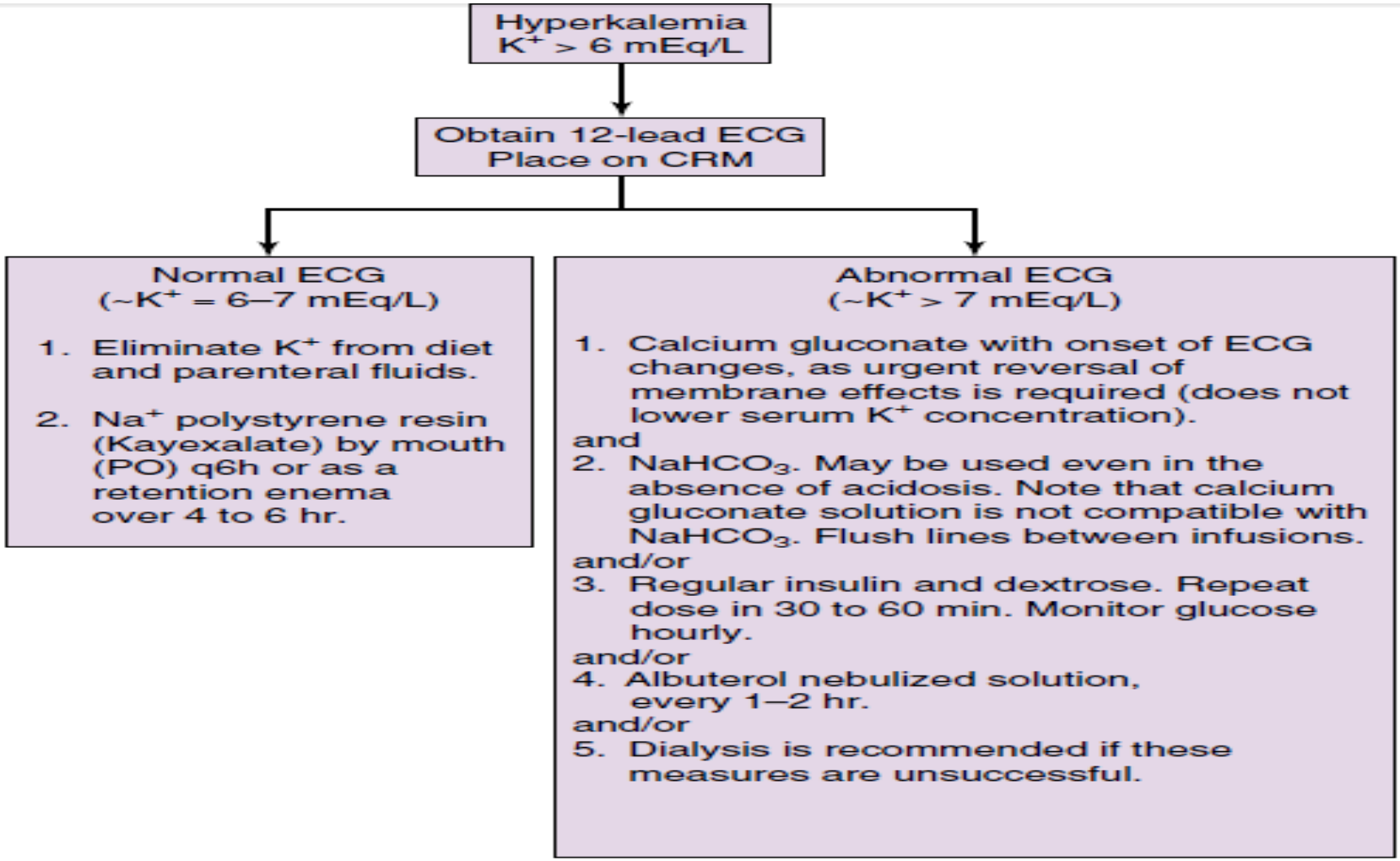
Skeletal muscle weakness, fasciculations, paresthesias, and ascending paralysis.
The typical ECG progression with increasing serum K^+ values:

1. Peaked T waves
2. Prolonged PR and widening of QRS
3. Loss of P waves
4. ST segment depression with further widening of QRS
5. Bradycardia, atrioventricular (AV) block, ventricular arrhythmias, torsades de pointes, and cardiac arrest

ETIOLOGIES

Increased total body K^+		Intracellular shifts (no change in total body K^+)
Increased urine K^+	**Decreased urine K^+**	
Transfusion with aged blood	Renal failure	Tumor lysis syndrome
Exogenous K^+	Hypoaldosteronism	Leukocytosis (>200 x $10^3/\mu L$)
Spitzer syndrome	Aldosterone insensitivity	Thrombocytosis (>750 x $10^3/\mu L$)[b]
	↓ Insulin causing hyperglycemia and/or DKA	Metabolic acidosis[a]
	K^+-sparing diuretics	Blood drawing (hemolyzed sample)
	Congenital adrenal hyperplasia	Rhabdomyolysis/crush injury
	Type IV RTA	Malignant hyperthermia
	Meds: ACE inhibitors, angiotensin II blockers, K sparing diuretics, calcineurin inhibitors, NSAIDs, heparin, TMX, drospirenone	Theophylline intoxication

Hyperkalemia



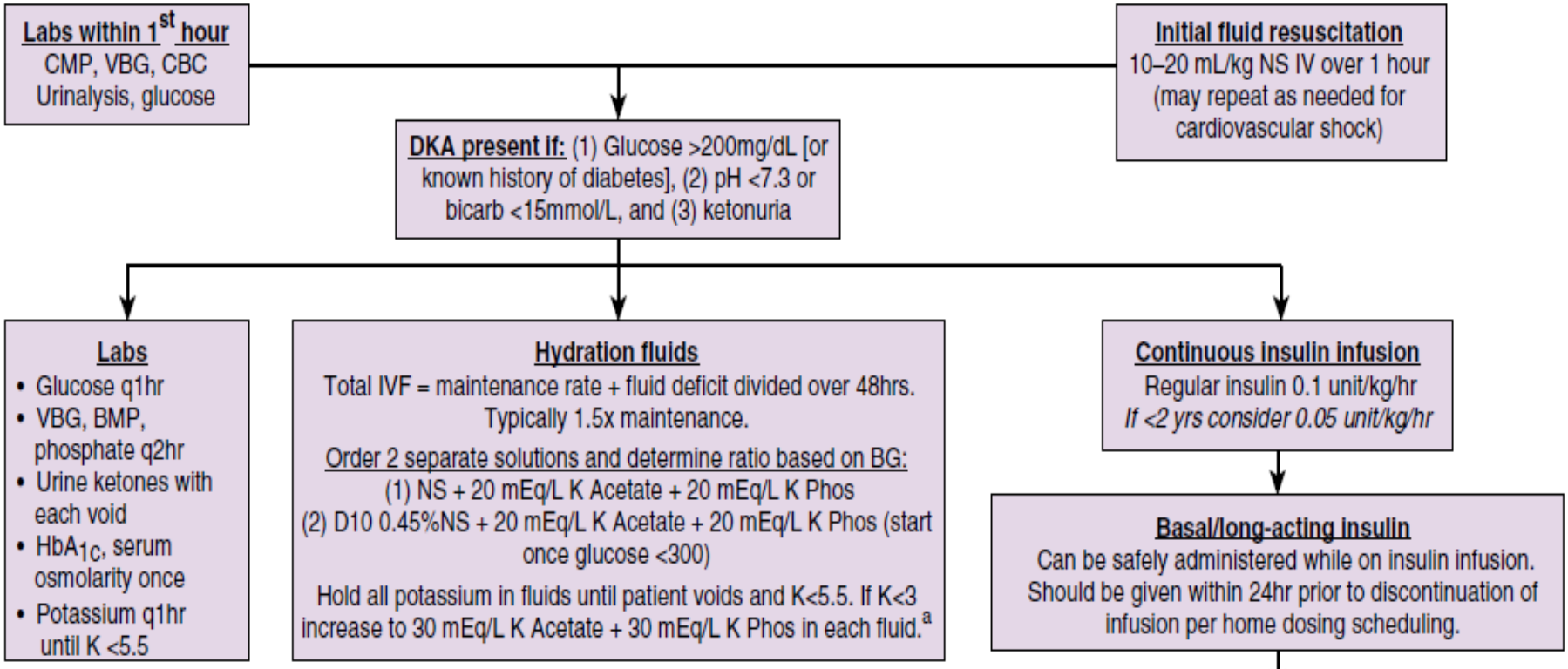
Diabetic ketoacidosis (DKA)

Definition:

Hyperglycemia (>200 mg/dL) with ketonemia, ketonuria, and metabolic acidosis (pH <7.30, bicarbonate <15 mEq/L)

- Blood Glucose reflects hydration status while pH reflects DKA severity
- Symptoms: Nausea, emesis, abdominal pain, fruity breath, altered mental status, Kussmaul respirations
- Precipitating factors: New-onset DM, known diabetes with missed insulin doses, insulin pump/infusion site malfunction, or physiologic stress due to acute illness.

Diabetic ketoacidosis (DKA)



BG blood glucose, SC subcutaneous

Note: This algorithm may need to be individualized depending on patient presentation.

a) Patients with DKA are total body K depleted and are at risk for severe hypokalemia during DKA therapy. However serum K levels may be normal or elevated as a result of the shift of K to the extracellular compartment in the setting of acidosis.

b) Appropriate to transition to SC insulin once pH >7.3, normal anion gap, normal physical exam, and patient ready to eat.

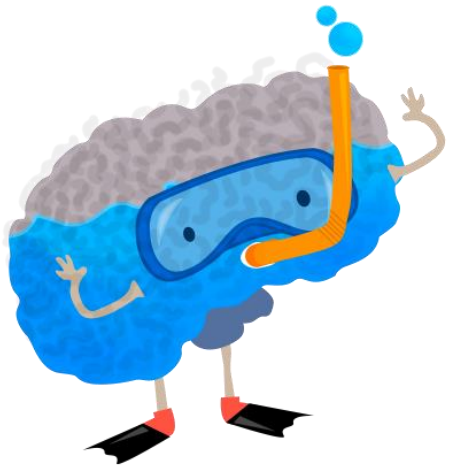
Diabetic ketoacidosis (DKA)

Management:

- Initial insulin administration may cause transient worsening of the acidosis as K+ is driven into cells in exchange for H+ ions.

- **Cerebral edema**: Most severe complication of DKA. Overly **aggressive hydration and rapid correction of hyperglycemia** may play a role in its development. Risk factors include
  1. severe acidosis,
  2. evidence of renal insufficiency,
  3. young age and new onset,
  4. use of bicarbonate
  5. early use of insuline (in the 1st hour).

- Management of cerebral edema is manitol.



SEPSIS

Definitions

- The **systemic inflammatory response syndrome** (**SIRS**) is an inflammatory cascade that is initiated by the host response to an **infectious or noninfectious trigger**. In neonates and pediatric patients, SIRS manifests as:
  1. temperature instability,
  2. respiratory dysfunction (altered gas exchange, hypoxemia, acute respiratory distress syndrome),
  3. cardiac dysfunction (tachycardia, delayed capillary refill, hypotension),
  4. perfusion abnormalities (oliguria, metabolic acidosis, decreased level of consciousness).

- **Sepsis** is defined as **SIRS** resulting from a **suspected or proven infectious etiology**.

- **Severe sepsis** (the presence of sepsis combined with organ dysfunction).

- **Septic shock** (severe sepsis plus the persistence of hypoperfusion or hypotension despite adequate fluid resuscitation or a requirement for vasoactive agents), that leads to multi-organ dysfunction syndrome (**MODS)**, and possibly death.

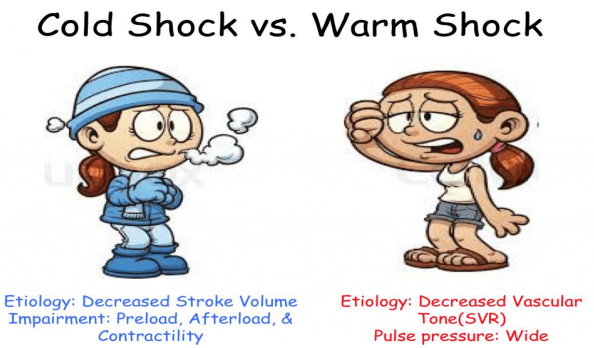
Table 70-7 International Consensus Definitions for Pediatric Sepsis

Infection	Suspected or proven infection or a clinical syndrome associated with high probability of infection
SIRS	Two of 4 criteria, 1 of which must be abnormal temperature or abnormal leukocyte count: 1. Core temperature >38.5°C (101.3°F) or <36°C (96.8°F) (rectal, bladder, oral, or central catheter) 2. Tachycardia: Mean heart rate >2 SD above normal for age in absence of external stimuli, chronic drugs or painful stimuli *or* Unexplained persistent elevation over 0.5-4 hr *or* In children <1 yr old, persistent bradycardia over 0.5 hr (mean heart rate <10th percentile for age in absence of vagal stimuli, β-blocker drugs, or congenital heart disease) 3. Respiratory rate >2 SD above normal for age or acute need for mechanical ventilation not related to neuromuscular disease or general anesthesia 4. Leukocyte count elevated or depressed for age (not secondary to chemotherapy) or >10% immature neutrophils
Sepsis	SIRS plus a suspected or proven infection
Severe sepsis	Sepsis plus 1 of the following: 1. Cardiovascular organ dysfunction, defined as: • Despite >40 mL/kg of isotonic intravenous fluid in 1 hr: • Hypotension <5th percentile for age or systolic blood pressure <2 SD below normal for age *or* • Need for vasoactive drug to maintain blood pressure *or* • 2 of the following: • Unexplained metabolic acidosis: base deficit >5 mEq/L • Increased arterial lactate: >2 times upper limit of normal • Oliguria: urine output <0.5 mL/kg/hr • Prolonged capillary refill: >5 sec • Core to peripheral temperature gap >3°C (5.4°F) 2. ARDS as defined by the presence of a PaO_2/FIO_2 ratio ≤300 mm Hg, bilateral infiltrates on chest radiograph, and no evidence of left heart failure *or* Sepsis plus 2 or more organ dysfunctions (respiratory, renal, neurologic, hematologic, or hepatic)
Septic shock	Sepsis plus cardiovascular organ dysfunction as defined above
MODS	Presence of altered organ function such that homeostasis cannot be maintained without medical intervention

Table 70-8 Goal-Directed Therapy of Organ System Dysfunction in Shock

SYSTEM	DISORDERS	GOALS	THERAPIES
Respiratory	Acute respiratory distress syndrome Respiratory muscle fatigue Central apnea	Prevent/treat: hypoxia and respiratory acidosis Prevent barotrauma Decrease work of breathing	Oxygen Noninvasive ventilation Early endotracheal intubation and mechanical ventilation Positive end-expiratory pressure (PEEP) Permissive hypercapnia High-frequency ventilation Extracorporeal membrane oxygenation (ECMO)
Renal	Prerenal failure Renal failure	Prevent/treat: hypovolemia, hypervolemia, hyperkalemia, metabolic acidosis, hypernatremia/hyponatremia, and hypertension Monitor serum electrolytes	Judicious fluid resuscitation Establishment of normal urine output and blood pressure for age Furosemide (Lasix) Dialysis, ultrafiltration, hemofiltration
Hematologic	Coagulopathy (disseminated intravascular coagulation) Thrombosis	Prevent/treat: bleeding Prevent/treat: abnormal clotting	Vitamin K Fresh-frozen plasma Platelets Heparinization
Gastrointestinal	Stress ulcers Ileus Bacterial translocation	Prevent/treat: gastric bleeding Avoid aspiration, abdominal distention Avoid mucosal atrophy	Histamine H_2-receptor–blocking agents or proton pump inhibitors Nasogastric tube Early enteral feedings
Endocrine	Adrenal insufficiency, primary or secondary to chronic steroid therapy	Prevent/treat: adrenal crisis	Stress-dose steroids in patients previously given steroids Physiologic dose for presumed primary insufficiency in sepsis
Metabolic	Metabolic acidosis	Correct etiology Normalize pH	Treatment of hypovolemia (fluids), poor cardiac function (fluids, inotropic agents) Improvement of renal acid excretion Low-dose (0.5-2.0 mEq/kg) sodium bicarbonate if the patient is not showing response, pH <7.1, and ventilation (CO_2 elimination) is adequate

Pathophysiology



- The initial signs and symptoms of sepsis include alterations in temperature regulation (hyperthermia or hypothermia), tachycardia, and tachypnea.
  1. **Warm shock** in the **early stages (hyperdynamic phase, low SVR**), **cardiac output increases** in an attempt to maintain adequate oxygen delivery and meet the greater metabolic demands of the organs and tissues.
  2. **Cold shock** as septic shock progresses, **cardiac output falls** in response to the effects of numerous inflammatory mediators, leading to a compensatory **elevation in SVR**.

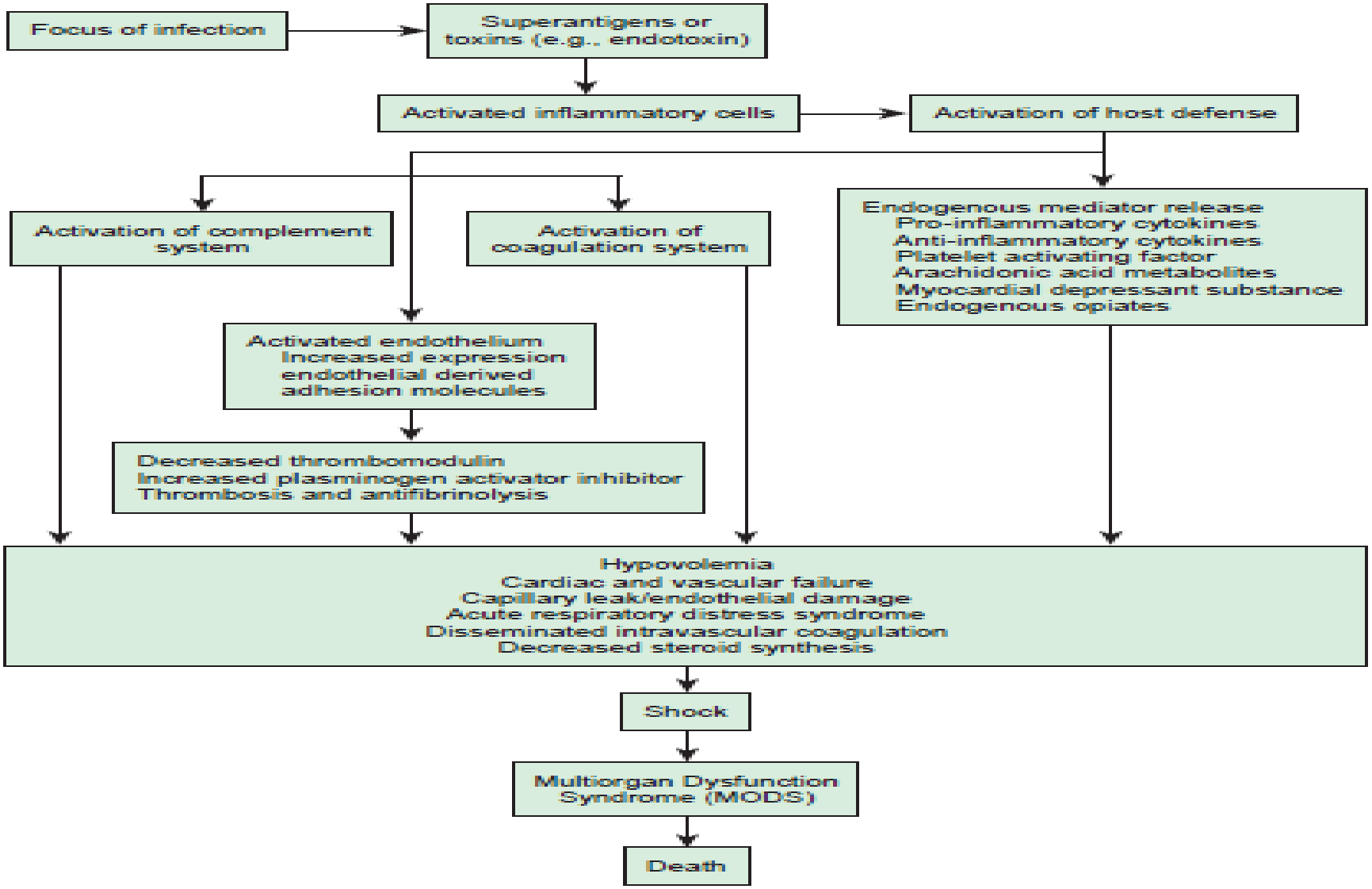
	Warm Shock	Cold Shock
Pulse pressure	Wide (≥30 mm Hg)	Narrow (<30 mm Hg)
Diastolic blood pressure	Decreased	Normal or Increased
Distal pulses	Bounding	Absent or Weak
Capillary refill	"Flash" or ≤2 seconds	"Delayed" or >2 seconds
Extremity temperature	Warm	Cool

doi:10.1371/journal.pone.0033355.t001

Pathophysiology

- **Septic shock** is often a unique combination of:
  1. **Distributive shock** is the result of decreased SVR and is **the primary mechanism**.
  2. **Hypovolemic shock** from intravascular fluid losses occurs through capillary leak.
  3. **Cardiogenic shock** results from the myocardial-depressant effects of sepsis.

Pathophysiology



Pathophysiology

Clinical Signs of Bacterial Sepsis

Table 109-5	Initial Signs and Symptoms of Infection in Newborn Infants

GENERAL
Fever, temperature instability
"Not doing well"
Poor feeding
Edema

GASTROINTESTINAL SYSTEM
Abdominal distention
Vomiting
Diarrhea
Hepatomegaly

RESPIRATORY SYSTEM
Apnea, dyspnea
Tachypnea, retractions
Flaring, grunting
Cyanosis

RENAL SYSTEM
Oliguria

CARDIOVASCULAR SYSTEM
Pallor; mottling; cold, clammy skin
Tachycardia
Hypotension
Bradycardia

CENTRAL NERVOUS SYSTEM
Irritability, lethargy
Tremors, seizures
Hyporeflexia, hypotonia
Abnormal Moro reflex
Irregular respirations
Full fontanel
High-pitched cry

HEMATOLOGIC SYSTEM
Jaundice
Splenomegaly
Pallor
Petechiae, purpura
Bleeding

DIAGNOSIS

- **Shock is a clinical diagnosis** based on a thorough history and physical examination.
- **The vital sign targets adjusted to pediatric-size patients.**

Table 70-3 Signs of Decreased Perfusion

ORGAN SYSTEM	↓ PERFUSION	↓↓ PERFUSION	↓↓↓ PERFUSION
Central nervous system	—	Restless, apathetic, anxious	Agitated/confused, stuporous, coma
Respiration	—	↑ Ventilation	↑↑ Ventilation
Metabolism	—	Compensated metabolic acidemia	Uncompensated metabolic acidemia
Gut	—	↓ Motility	Ileus
Kidney	↓ Urine volume ↑ Urinary specific gravity	Oliguria (<0.5 mL/kg/hr)	Oliguria/anuria
Skin	Delayed capillary refill	Cool extremities	Mottled, cyanotic, cold extremities
Cardiovascular system	↑ Heart rate	↑↑ Heart rate ↓ Peripheral pulses	↑↑ Heart rate ↓ Blood pressure, central pulses only

Table 109-7 Serious Systemic Illness in Newborns: Differential Diagnosis of Neonatal Sepsis

CARDIAC
Congenital: hypoplastic left heart syndrome, other structural disease, persistent pulmonary hypertension of the newborn (PPHN)
Acquired: myocarditis, hypovolemic or cardiogenic shock, PPHN
GASTROINTESTINAL
Necrotizing enterocolitis
Spontaneous gastrointestinal perforation
Structural abnormalities
Hepatic failure (inborn errors of metabolism, neonatal iron storage disease)
HEMATOLOGIC
Neonatal purpura fulminans
Immune-mediated thrombocytopenia
Immune-mediated neutropenia
Severe anemia
Malignancies (congenital leukemia)
Langerhans cell histiocytosis
Hereditary clotting disorders
Familial hemophagocytosis syndrome
METABOLIC
Hypoglycemia
Adrenal disorders: Adrenal hemorrhage, adrenal insufficiency, congenital adrenal hyperplasia
Inborn errors of metabolism: Organic acidurias, lactic acidoses, urea cycle disorders, galactosemia
NEUROLOGIC
Intracranial hemorrhage: spontaneous, caused by child abuse
Hypoxic-ischemic encephalopathy
Neonatal seizures
Infant botulism
RESPIRATORY
Respiratory distress syndrome
Aspiration pneumonia: amniotic fluid, meconium, or gastric contents
Lung hypoplasia
Tracheoesophageal fistula
Transient tachypnea of the newborn

Laboratory Testing

- **Septic work up:**

1. Blood culture
2. Lumber pumcture
3. Urine analysis and culture
4. CBC and differential
5. I:T ratio, absolute neutrophil count
6. Platelet count
7. CRP and procalcitonine (PCT). Other acute phase reactant (IL, TNF)
8. CXR

- **To assess severity and end organ damage:**

1. Lactic acid
2. DIC profile: PT, PTT, INR, fibrinogen and FDP.
3. Glucose levels.
4. Other electrolyte abnormalities are hypocalcemia, hypoalbuminemia, and metabolic acidosis (blood gas).
5. Renal and/ or hepatic function

Laboratory Testing

Blood Cultures

- The **gold standard for detection of bacteremia** with suspected sepsis is a positive blood culture result **(peripheral and central; aerobic and nonaerobic**).

- Most blood culture results are **detected within 24–48 hours** with use of the new technology.

- The use of **antibiotics can reduce the ability to detect bacteremia**.

- The decision to discontinue treatment with antibiotics should include **the assessment of the infant's clinical condition and should not rely solely on a negative blood culture result**. When the suspicion of sepsis is high, clinicians should consider continuing Abx.

Laboratory Testing

Urine Analysis and Cultures

- The frequency of positive urine culture results in infants with **early-onset sepsis is relatively low**.
- Infants with **late-onset sepsis tend to have a higher rate of positive urine culture results**.
- **In the first 72 hours of life**, because the yield from urine cultures is low, it is **not generally recommended to obtain urine specimens**.
- In the newborn **older than 72 hours**, a **urine sample collected by an aseptic technique** (**urinary catheter or suprapubic bladder aspiration**) is an essential part of the sepsis work-up.

Laboratory Testing

Cerebrospinal Fluid analysis and culturs

- The gold standard for diagnosis of meningitis is the analysis of the **CSF, including the WBC count, glucose and protein levels, viral PCR, Latex test, Gram stain, and culture.**

- Although an **increase is expected in the number of neutrophils** with bacterial meningitis, one **may see a predominance of lymphocytes within a conversion to PMNs**.

- **Lumbar punctures are deferred in infants with any instability or uncorrected bleeding disorders.**

Normal CSF analysis

	WBCs (/microL)	Protein (mg/dL)	Glucose (mg/dL)	RBCs
Normal in Children	0 - 5 (Lymphocytes)	20 – 40	50 – 100 (1/2 - 2/3) of serum glucose	0
Normal in Neonates	0-28 (60% Lymphocytes)	15-135	50 – 100 (1/2 - 2/3) of serum glucose	0

Laboratory Testing

White Blood Cell Count and Neutrophil Indices

- Normal white blood cell (**WBC**) counts range from **9000–32,000 cells** per microliter at **the time of birth**.

- The **absolute neutrophil count (ANC)** and **the ratio of immature neutrophils to total neutrophils (I/T)** are **more useful than total leukocyte counts in the diagnosis of neonatal sepsis.**

- **The optimal time to obtain WBC counts in neonatal sepsis is after 4 hours of age**, and most recommendations are to obtain the first counts at 6–12 hours of age.

- **Neutropenia is the best predictor of sepsis**, whereas neutrophilia does not correlate well.

- The **I/T ratio is considered to have the best sensitivity of all of the neutrophil indices (normal value < 20%).**

White Blood Cell Count and Neutrophil Indices

Table 1. Normal Blood Leukocyte Counts*

	Total Leukocytes		Neutrophils			Lymphocytes			Monocytes		Eosinophils	
Age	Mean	(Range)	Mean	(Range)	%	Mean	(Range)	%	Mean	%	Mean	%
Birth	18.1	(9.0 to 30.0)	11.0	(6.0 to 26.0)	61	5.5	(2.0 to 11.0)	31	1.1	6	0.4	2
12 h	22.8	(13.0 to 38.0)	15.5	(6.0 to 28.0)	68	5.5	(2.0 to 11.0)	24	1.2	5	0.5	2
24 h	18.9	(9.4 to 34.0)	11.5	(5.0 to 21.0)	61	5.8	(2.0 to 11.5)	31	1.1	6	0.5	2
1 wk	12.2	(5.0 to 21.0)	5.5	(1.5 to 10.0)	45	5.0	(2.0 to 17.0)	41	1.1	9	0.5	4
2 wk	11.4	(5.0 to 20.0)	4.5	(1.0 to 9.5)	40	5.5	(2.0 to 17.0)	48	1.0	9	0.4	3
1 mo	10.8	(5.0 to 19.5)	3.8	(1.0 to 9.0)	35	6.0	(2.5 to 16.5)	56	0.7	7	0.3	3
6 mo	11.9	(6.0 to 17.5)	3.8	(1.0 to 8.5)	32	7.3	(4.0 to 13.5)	61	0.6	5	0.3	3
1 y	11.4	(6.0 to 17.5)	3.5	(1.5 to 8.5)	31	7.0	(4.0 to 10.5)	61	0.6	5	0.3	3
2 y	10.6	(6.0 to 17.0)	3.5	(1.5 to 8.5)	33	6.3	(3.0 to 9.5)	59	0.5	5	0.3	3
4 y	9.1	(5.5 to 15.5)	3.8	(1.5 to 8.5)	42	4.5	(2.0 to 8.0)	50	0.5	5	0.3	3
6 y	8.5	(5.0 to 14.5)	4.3	(1.5 to 8.0)	51	3.5	(1.5 to 7.0)	42	0.4	5	0.2	3
8 y	8.3	(4.5 to 13.5)	4.4	(1.5 to 8.0)	53	3.3	(1.5 to 6.8)	39	0.4	4	0.2	2
10 y	8.1	(4.5 to 13.5)	4.4	(1.8 to 8.0)	54	3.1	(1.5 to 6.5)	38	0.4	4	0.2	2
16 y	7.8	(4.5 to 13.0)	4.4	(1.8 to 8.0)	57	2.8	(1.2 to 5.2)	35	0.4	5	0.2	3
21 y	7.4	(4.5 to 11.0)	4.4	(1.8 to 7.7)	59	2.5	(1.0 to 4.8)	34	0.3	4	0.2	3

*Numbers of leukocytes are in thousands/mcL ($\times 10^9$/L), ranges are estimates of 95% confidence limits, and percentages refer to differential counts. Neutrophils include band cells at all ages and a small number of metamyelocytes and myelocytes in the first few postnatal days.
From Dallman PR. Blood and blood-forming tissues. In: Rudolph AM, ed. *Rudolph's Pediatrics.* 16th ed. New York, NY: Appleton-Century-Crofts; 1977:1178, with permission.

Laboratory Testing

Platelet Counts

- Approximately 25%–30% of infants exhibit **thrombocytopenia** at the time of diagnosis of sepsis and **usually it is a late sign**.

- Accelerated platelet destruction and possibly depressed production caused by bacterial products on the bone marrow are the underlying mechanisms for thrombocytopenia in infected infants.

- **Disseminated intravascular coagulation** may be seen in some infants with severe sepsis.

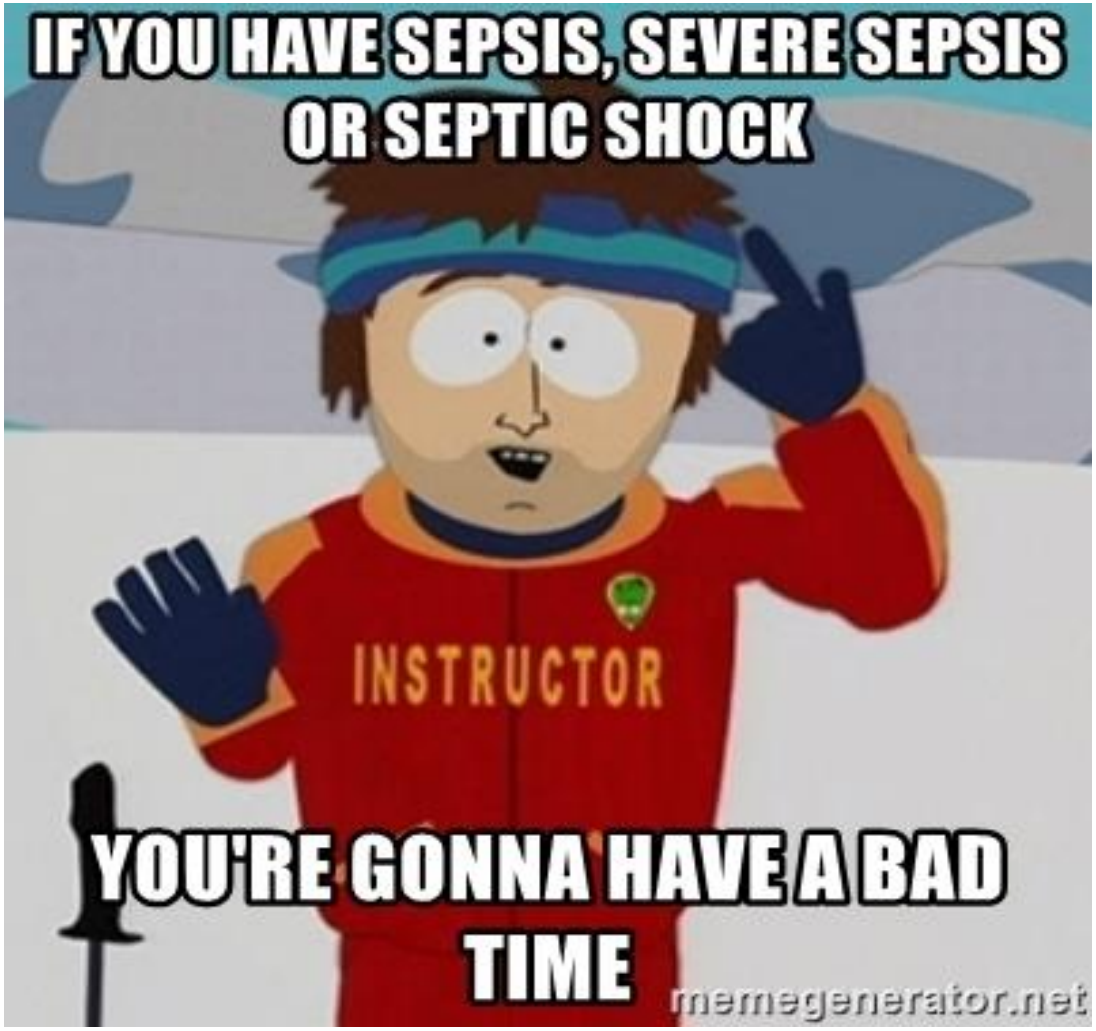
Laboratory Testing

Acute-Phase Reactants

- **C-reactive protein (CRP)**
  - **Produced by the liver** and induced by proinflammatory cytokines, and **its level rises to a maximum at 12–24 hours**.
  - **CRP value of 5 mg/dL is considered the upper limit of normal.**
  - **Monitoring CRP levels** has been widely used **to diagnose infections and to adjust the duration of antibiotic therapy** in infants with suspected versus proven sepsis.

- **Procalcitonin (PCT)**
  - Produced by cells such as **hepatocytes, nephrons, and monocytes.**
  - **PCT concentrations rise much faster than CRP concentrations; rises at 4 hours**, peaks at 6 hours, and plateaus 8–24 hours after a stimulus.
  - Most srudies have concluded that **PCT levels are superior to CRP levels in the early diagnosis of neonatal sepsis.**

TREATMENT


IF YOU HAVE SEPSIS, SEVERE SEPSIS OR SEPTIC SHOCK
INSTRUCTOR
YOU'RE GONNA HAVE A BAD TIME
memegenerator.net

TREATMENT

- **Early recognition and prompt intervention** are extremely important in the management of all forms of shock.
- **Stabilization of airway, breathing, and circulation**.
- Establishment of **intravenous (IV) or intraosseous access**.
- **Rapid IVF of 20 mL/kg isotonic fluid** can be repeated quickly **up to 60-80 mL/kg**.
- If shock **remains refractory following volume resuscitation**, **vasopressor therapy (norepinephrine, or epinephrine**) should be instituted.
- If **vasopressor resistant shock** give **cortiosteroids**.
- Admitted to an **intensive care** unit with continuous monitoring.
- **Start empiric antibiotic treatment within the first hour of diagnosis.**

Septic	↑	↓↓	Normal or ↓	↓	**Within 1st hour:** Administer isotonic crystalloid boluses, broad-spectrum antibiotics, and consider stress-dose hydrocortisone **Warm:** Support with norepinephrine or high-dose dopamine **Cold:** Support with epinephrine or dopamine

0 min

INFANTS/CHILDREN

Recognize decreased mental status and perfusion.
Begin high flow O_2 and establish IO/IV access according to PALS.

5 min

If no hepatomegaly or rales/crackles then push 20 mL/kg isotonic saline boluses and reassess after each bolus up to 60 mL/kg until improved perfusion. Stop for rales, crackles or hepatomegaly. Correct hypoglycemia and hypocalcemia. Begin antibiotics.

15 min

Fluid refractory shock?

Begin peripheral IV/IO inotrope infusion, preferably Epinephrine 0.05–0.3 µg/kg/min
Use Atropine/Ketamine IV/IO/IM if needed for Central Vein or Airway Access

Titrate Epinephrine 0.05–0.3 µg/kg/min for Cold Shock.
(Titrate central Dopamine 5–9 µg/kg/min if Epinephrine not available)
Titrate central Norepinephrine from 0.05 µg/kg/min and upward to reverse Warm Shock.
(Titrate central Dopamine ≥ 10 µg/kg/min if Norepinephrine not available)

60 min

Catecholamine-resistant shock?

If at risk for Absolute Adrenal Insufficiency consider Hydrocortisone.
Use Doppler US, PICCO, FATD or PAC to Direct Fluid, Inotrope, Vasopressor, Vasodilators
Goal is normal MAP-CVP, $ScvO_2$ > 70* and CI 3.3–6.0 L/min/m^2

Normal Blood Pressure
Cold Shock
$ScvO_2$ < 70%* /Hgb > 10g/dL
on Epinephrine?

Begin Milrinone infusion.
Add Nitroso-vasodilator if CI < 3.3 L/min/m^2 with High SVRI and/or poor skin perfusion.
Consider Levosimendan if unsuccessful.

Low Blood Pressure
Cold Shock
$ScvO_2$ < 70%* /Hgb > 10g/dL
on Epinephrine?

Add Norepinephrine to Epinephrine to attain normal diastolic blood pressure. If CI < 3.3 L/min/m^2 add Dobutamine, Enoximone, Levosimendan, or Milrinone.

Low Blood Pressure
Warm Shock
$ScvO_2$ < 70%*
on Norepinephrine?

If euvolemic, add Vasopressin, Terlipressin, or Angiotensin. But, if CI decreases below 3.3 L/min/m^2 add Epinephrine, Dobutamine, Enoximone, Levosimendan.

Persistent Catecholamine-resistant shock?

Evaluate Pericardial Effusion or Pneumothorax,
Maintain IAP < 12 mmHg

Refractory shock?

ECMO

TREATMENT

Antibiotic therapy

- **Early administration of broad-spectrum antimicrobial agents is associated with a reduction in mortality.**

- Prompt initiation of empiric antimicrobial therapy based on **patient age, underlying disease, bacterial resistance and geographic location**.

TREATMENT

Antibiotic therapy

- Neonates ----- ampicillin plus cefotaxime and/or gentamicin.
- *Neisseria meningitidis and Haemophilus influenzae*---------- 3rd-generation cephalosporin (ceftriaxone or cefotaxime).
- Resistant *Streptococcus pneumoniae,* Methicillin-resistant *Staphylococcus aureus and* presence of catherters ------- vancomycin.
- Intraabdominal process, aspiration pneumonia, anaerobic coverage ------- metronidazole, clindamycin, or piperacillin-tazobactam.
- Nosocomial sepsis ------ resistant gram positive (vancomycine) + extended gram negtaive coverage (meropenem).
- Herpes simplex virus ---------- Acyclovir.
- Fungal infections ----- immunocompromised patients, preterms prolonged Abs use.

THANKS