



## UNC Carolina Air Care Hyperkalemia

To identify and treat life-threatening hyperkalemia

**DEFINITION** The likelihood of developing significant alterations in physiology from elevations of serum potassium are a function of both the absolute level and the rate of rise. Very high levels may not result in adverse effects if the level rises over a prolonged period of time. The lowest levels associated with risk are  $> 6.5$  mEq/L in neonatal patients and  $> 5.5$  mEq/L in older pediatric patients.

**History: Patients at Risk:**

- Patients with renal failure
- Extremely premature infants
- Patients receiving nephrotoxic drugs
- Profound dehydration
- Cardiac patients

**Symptoms:**

- Muscle weakness
- Parasthesias
- Tetany

**PHYSICAL EXAM** No specific physical findings are associated with hyperkalemia, unless there is an associated arrhythmia or cardiovascular collapse.

**LABORATORY DATA** Serum potassium levels above 6.0mEq/L.

1. Level must be obtained from a specimen without hemolysis. Capillary specimens should always be suspected of being hemolyzed even if hemolysis is not present by visual inspection.
2. Results from capillary specimens should be confirmed by repeat analysis of venous or arterial blood before definitive therapy is initiated.
3. Progression of ECG changes:
  - T-wave elevation
  - Loss of P-wave
  - Widened QRS
  - Depressed S-T segment
  - Aberrant ECG complexes
  - Dysrhythmias: bradycardia, ventricular tachycardia, and ventricular fibrillation

### TREATMENT

For levels  $>6.0$  mEq/L: Remove potassium from all IV fluids.

For levels  $>6.5$  mEq/L (may be lower for neonates) Remove potassium from the body:

- Kayexalate: 1gm/kg PO or NG q 2-6 hours or 1gm/kg PR q 2- 6 hours as a retention enema. The enema should be retained for at least 60 minutes. 1gm/kg should lower potassium by 1 mEq/L.
- Diuresis when kidneys are functioning; Lasix 1mg/kg IV For hyperkalemia associated with ECG changes:

Shift potassium from extracellular to intracellular spaces rapidly but temporarily by the following therapies:

- Sodium Bicarbonate 1-2 mEq/kg IV over 5-10 minutes
- Calcium gluconate 10%, 0.5-1.0ml/kg IV over 5-10 minutes to correct cardiac changes if sodium bicarbonate not successful. Maximum single dose: 20ml (2.0g of calcium gluconate)
- Glucose/insulin drip may be needed if desired results have not been obtained. Mix 0.5 gm/kg glucose with 0.3 units insulin, infuse over 2 hours. (Contact MCO before beginning this therapy).
- Albuterol 0.4 mg/kg nebulized.



## UNC Carolina Air Care Hypernatremia

To identify and treat hypernatremia

**DEFINITION** Hypernatremia is defined as a serum sodium level of greater than 145 mEq/L. Hypernatremia results from either sodium overload or free water depletion.

**HISTORY** Infants at Risk:

Free water depletion

- extremely premature infant
- conditions characterized by water depletion (e.g., vomiting, diarrhea, etc.)
- diseases associated with excessive urine production (e.g. diabetes)
- conditions which may limit water intake (e.g. dependency states)

Sodium overload

- iatrogenic sodium intoxication; infants receiving medications containing sodium
- improper mixing of formula

**PHYSICAL EXAM** Free water depletion; signs of dehydration:

- tachycardia
- hypotension
- decreased urine output
- metabolic acidosis

General signs and symptoms:

- irritability
- lethargy alternating with marked hyperirritability when stimulated and awake
- hyperreflexia
- hypertonicity
- high-pitched cry

**LABORATORY DATA** Serum sodium >145 mEq/L Often accompanied by other abnormal serum levels (e.g., low bicarbonate, high BUN and creatinine, hyperglycemia, hypocalcemia)

**TREATMENT**

1. Restore vascular volume if volume depleted:

- Administer 10-20 ml/kg LR, NS, or 5% albumin solution (LR is preferable)
- Repeat once if evidence of volume depletion persists.
- Consult MCO if evidence of volume depletion persist after administering a fluid bolus twice.

2. After restoration of vascular volume, administer maintenance IV fluids. The concentration of sodium in the fluid should be adjusted to correct serum sodium over approximately 48 hours.

3. If sodium >150, contact MCO.

4. Replace ongoing gastrointestinal, urinary and insensible losses.

5. Monitor BP closely.

6. Quantitate urine output

## UNC Carolina Air Care Hyponatremia

To identify and treat hyponatremia

### DEFINITION

Hyponatremia is defined as a serum sodium level of less than 135 mEq/L. In newborns, hyponatremia may be defined as a serum sodium level of <130 mEq/L. Hyponatremia results from either sodium depletion secondary to inadequate sodium intake or excessive Na losses or dilution secondary to the presence of excess free water.

History Risk Factors:

Sodium depletion

- Abnormal losses through GI tract as with diarrheal illness or patients with ostomies
- Medications causing urinary loss of sodium (e.g., diuretics, methylxanthines)
- Cystic fibrosis (particularly during summer months)
- Inadequate dietary intake (e.g. improper mixing of infant formula)

Free water intoxication

- Renal failure
- Syndromes of inappropriate ADH secretion (e.g., asphyxia, meningitis)
- Congestive heart failure
- Congenital adrenal hyperplasia
- Iatrogenic free water overload

Historical Clues:

Sodium depletion

- Poor weight gain
- Non-specific symptoms such as anorexia, nausea and irritability

Free water intoxication

- Decreased urine output
- Rapid weight gain not attributable to increased caloric intake

PHYSICAL EXAM General signs and symptoms:

Appearance of central nervous systems signs somewhat dependent upon rapidity of decline of sodium level. Signs do not generally appear except at extremely low levels (< 120 mEq/L). Signs include irritability, altered level of consciousness and seizures. Free water intoxication: Peripheral edema but with minimal pitting

LABORATORY DATA Serum sodium < 135 mEq/L Other electrolytes and metabolites often abnormal; particular abnormality dependent upon etiology

### TREATMENT

If the serum sodium level is low enough to cause significant CNS symptoms, the serum sodium needs to be increased by 5-10 mEq/L acutely:

- Administer Lasix 0.5-1.0 mg/kg IV
- Administer concentrated NaCl solution IV (conc. NaCl = 4 mEq NaCl/ml)

To increase serum sodium by 5 mEq/L

0.75ml/kg IV add this amount to the volume of maintenance solution to be infused in one hour

To increase serum sodium level by 10 mEq/L

1.50ml/kg IV add this amount to the volume of maintenance solution to be infused in one hour

Specific therapy for free water intoxication

- Administer diuretic if urine output is low and circulation is compromised by volume overload (i.e. congestive heart failure).
- Restrict fluids to 60% of maintenance rate.



## UNC Carolina Air Care Hypocalcemia

To identify and treat the physiologic consequences of hypocalcemia

**DEFINITION** For the purposes of this protocol, hypocalcemia will be defined as a serum ionized calcium level of less than 4.0 mg/dL. If ionized calcium levels are not available, hypocalcemia may be defined as a total serum level of less than 8.0 mg/dL. This protocol will deal primarily with the identification and treatment of infantile hypocalcemia.

**History Risk Factors:**

- Prematurity
- Birth asphyxia
- Maternal diabetes
- Maternal hyperparathyroidism
- Exchange transfusion
- Infantile hypoparathyroidism (e.g. with DiGeorge syndrome)
- Alkalosis

**Signs and Symptoms:**

- Vomiting
- Apnea
- Seizures

**PHYSICAL EXAM**

- Irritability
- Tremors
- Lethargy
- Tetany
- Laryngospasms
- Evidence of poor cardiac output (e.g. diminished pulses, poor capillary refill, low blood pressure)

**LABORATORY DATA** Calcium levels below 4.0 mg/dL (ionized) associated with symptoms should be treated. Calcium levels below 3.0 mg/dL (or below 6.0 mg/dL total) should be treated even in the absence of symptoms. Prolonged QT interval on ECG indicates physiologic effect.

**TREATMENT**

For seizures and tetany:

Administer Calcium Gluconate:

- Dose: 100mg/kg, max 2,000mg/dose
- Site: for peripheral IV, dilute 1:1 with sterile water
- Infuse carefully, infiltration produces severe necrosis
- Rate: administer over 10 minutes
- Monitor ECG complexes and rhythm

For shock and hypocalcemia in infants with severe systemic illness: Administer Calcium Gluconate:

- Neonates: 100-150mg/kg/dose IV Q 8 hr
- Infants, toddlers: 50-100mg/kg/dose IV Q 8 hr
- Preschoolers to adolescents: 20-50mg/kg IV Q 8 hr, (maximum single dose is 2,000 mg)

Consider Calcium Chloride for the child who is very ill but whose liver may not be able to metabolize the gluconate.

- Neonates: 30-50 mg/kg/dose IV Q 8 hr
  - Infants, toddlers: 15-33mg/kg/dose IV Q 8
  - Preschoolers to adolescents: 10-15 mg/kg IV Q 8 hr
-



## UNC Carolina Air Care Hypoglycemia

To prevent hypoglycemia and to maintain blood glucose in the normal range

Serum glucose levels in healthy individuals vary over a surprisingly wide range and may depend upon factors such as age, recent caloric intake and utilization and type of carbohydrate intake. The operational definition of hypoglycemia during transport will be: neonates < 40 mg/dl; older children < 60 mg/dl.

### History / Patients at Risk:

Neonates, premature infants, SGA infants, infants who have been stressed, hypoxemic, or hypothermic, infants with hyperinsulinism (i.e. infants of diabetic mothers), infants with congenital heart disease.

Older children: administration of insulin in diabetic without appropriate carbohydrate intake, sepsis

### PHYSICAL EXAM Signs and Symptoms:

- jitteriness, tremors
- irritability
- weak or high pitched cry
- irregular respirations, tachypnea, and/or apnea
- lethargy, decreased tone and/or poor feeding
- sweating, pallor and tachycardia
- seizures
- coma may result from severe or prolonged hypoglycemia

### Additional symptoms in older patients:

- confusion
- bizarre behavior
- visual disturbances
- headache

LABORATORY DATA Glucose meter results (utilizing I-Stat, Sure Step Pro or other instrument approved by UNC Air Care), showing serum glucose below the lower limits of the designated normal range for age.

### TREATMENT

1. Obtain glucose measurement with glucose meter or Chemstrip in all patients at risk, unless measurement has been performed by referring hospital within previous 30 minutes.
  2. Establish IV access and administer glucose if value is abnormal: Neonate: 2-4 ml/kg D10W over 2-5 minutes. Pediatric patient: 0.5 g/kg D50W (0.5g/ml) over 2-5 minutes.
  3. Provide continuous infusion of a glucose containing maintenance IV fluid following bolus infusion.
  4. Assess blood glucose 10-15 minutes after bolus infusion.
    - Reassess blood glucose every 30 minutes until stable
-



## UNC Carolina Air Care Sepsis

To identify and treat patients with sepsis

### History: Risk factors

- Early-onset Neonatal Sepsis
- Prematurity
- Male gender
- Multiple gestation birth
- Maternal genitourinary tract colonization and infection
- Chorioamnionitis
- Prolonged rupture of membranes

### Late-onset & older pediatric patients

- Indwelling catheters and other appliances
- Constitutional illnesses and diseases (e.g., Sickle cell disease, immune deficiency, bronchopulmonary dysplasia, etc.)
- Immunosuppressive therapies

### Historical clues

- Poor feeding
- Irritability
- Lethargy
- Temperature instability

### PHYSICAL EXAM

#### General

- Fever
- Hypothermia (more common than fever in neonate)
- Respiratory
- Grunting
- Nasal flaring
- Retractions
- Tachypnea
- Apnea

#### Neurologic

- Hypotonia
- Decreased spontaneous movement
- Seizures

#### Cardiovascular

- Bradycardia/tachycardia
- Hypotension/shock

#### Skin

- Petechiae
- Pustules
- Sclerema
- Hyperemia

### LABORATORY DATA

- Leukocytosis
- Neutropenia (much more common than leukocytosis in neonates)

- Thrombocytopenia
- Laboratory evidence of coagulopathy
- Hypoglycemia/hyperglycemia

### XRAY DATA

No specific radiographic findings; abnormalities on chest Xray present with associated pneumonia.

### TREATMENT

1. Maintain airway and oxygenate appropriately.
2. Obtain vascular access.
3. Place Foley catheter (in older patients).
4. Fluid resuscitate with NS or LR 10-20cc/kg if poor perfusion, hypotension or shock present.
5. If shock persists after two fluid boluses, contact MCO to discuss inotrope therapy.
6. Begin maintenance dextrose solution and follow blood glucose.
7. Obtain blood culture, and CBC (if time allows).



## UNC Carolina Air Care Sepsis

8. Administer antibiotics. For early-onset neonatal infection Ampicillin and Gentamicin. Administer Metronidazole if progressive NEC suspected. For late onset neonatal infection **consult** MCO For pediatric patients **contact** MCO
9. Maintain universal precautions



## UNC Carolina Air Care Meningitis

To outline the management of meningitis

### History

#### Neonates and Young Children

- Hypothermia in neonates
- Hypotension/shock
- Poor feeding
- Alternating drowsiness and irritability

#### Older Children

- Fever
- Irritability
- Vomiting
- Headache
- Photophobia
- Seizures

### PHYSICAL EXAM

#### General

- Lethargy, somnolence, or coma
- Petechial skin lesion (particularly with *N. meningitidis*)

#### Neonates and Young Children

- Jaundice
- Bulging fontanel

#### Older Children

- Nuchal rigidity
- Sensori-motor and/or autonomic disturbances

### TREATMENT

1. Begin respiratory isolation (mask for crew)
  2. Maintain airway and oxygenate appropriately.
    - May Keep PaO<sub>2</sub> >100, and PCO<sub>2</sub> 30-40.
    - May need to electively intubate and ventilate.
  3. Aggressively treat shock using colloid 10-20cc/kg, if shock persists, repeat administration of volume expander
  4. If shock persists after two fluid boluses, contact MCO to discuss inotrope therapy.
  5. Once shock is treated, begin maintenance intravenous fluids. Fluid restriction may be advisable; contact MCO.
  6. Treat seizures (see Altered Neurologic Status protocol).
  7. Administer antipyretics for hyperthermia.
  8. Administer antibiotics. (contact MCO for drug of choice.)
-



## UNC Carolina Air Care Metabolic Acidosis

To identify and treat metabolic acidosis and its underlying cause

### DEFINITION

Metabolic acidosis is the accumulation of excess hydrogen ions in the serum. Metabolic acidosis results from excessive production of hydrogen donors, inadequate excretion of hydrogen ions or excessive loss of buffer. Metabolic acidosis may result from a variety of causes including:

- hypovolemia
- other causes of shock
- renal tubular acidosis
- diabetic ketoacidosis
- poisonings/ ingestions
- inborn errors of metabolism
- miscellaneous causes

### HISTORY

The patient's history may reveal clues regarding etiology. These historical clues are very specific to each cause and are listed in the protocols for individual diseases and problems (e.g., see Hypovolemia/Shock and Poisoning).

### PHYSICAL EXAM

There are no specific findings associated with metabolic acidosis. Abnormal findings result from the underlying cause. Blood pressure should be recorded initially and monitored frequently in all patients with metabolic acidosis. Respiratory rate and depth may be increased when patients attempt to correct metabolic acidosis with respiratory alkalosis.

### LABORATORY DATA

1. Obtain an ABG in patients at risk. Significant metabolic acidosis in pediatric patients is usually defined as a pH less than 7.30 with a base deficit greater than 5
2. If the etiology of the metabolic acidosis is not known, the differential diagnosis may be narrowed by calculating the anion gap:

CALCULATING THE ANION GAP:  $GAP = \text{Serum Na} - [\text{serum HCO}_3 + \text{serum CL}]$ . Normal anion gap = 8 -15. Anion gap greater than 15; the patient has an increase in unmeasurable anions.

3. Causes of metabolic acidosis with an increased anion gap include:
  - diabetic ketoacidosis
  - uremia (phosphates and sulfates); acute renal failure
  - lactic acidosis (lactate)
  - aspirin poisoning (salicylate)
  - methyl poisoning (formic acid)
  - ethylene glycol poisoning (oxalic acid and formic acid)
  - inborn errors of metabolism
4. Causes of metabolic acidosis without an increased anion gap include:
  - loss of buffers, such as with diarrhea (loss of bicarbonate ions)
  - dehydration
  - uremia (the kidneys cannot excrete acids formed daily)
  - renal tubular acidosis
  - sepsis
  - shock from any cause



## UNC Carolina Air Care Metabolic Acidosis

### TREATMENT

1. The goal of therapy should be the identification and treatment of the underlying cause of the acidosis. If the etiology is identified and the problem corrected (e.g. treatment of hypovolemia), the administration of a buffer is often unnecessary.
2. Sodium bicarbonate therapy should be considered for persistent metabolic acidosis with a pH less than 7.25 and a base deficit greater than 7 that is not responding to correction of underlying cause.

### COMMENTS:

1. Metabolic acidosis occasionally accompanies respiratory acidosis. Treatment of the metabolic component, indicated by the base deficit, may obviate the need for more aggressive respiratory therapy.
2. Treatment with bicarbonate results in the production of CO<sub>2</sub>. Use cautiously if there is a concurrent respiratory acidosis. Rapid infusion of bicarbonate may necessitate an increase in respiratory support.
3. Severe metabolic acidosis (pH less than 7.20) may result in hypotension by decreasing myocardial contractility and cardiac output. Cardiac dysrhythmias and deep, rapid (Kussmaul's) respirations may result.
4. Buffer therapy should not be administered unless laboratory evidence of metabolic acidosis has been documented



## UNC Carolina Air Care Supraventricular Tachycardia

To identify and manage patients with supraventricular tachycardia

**DEFINITION** Supraventricular tachycardia (SVT) is a tachyarrhythmia characterized by a fixed elevated heart rate between 160 and 300, but usually around 230, with each beat originating from an abnormal focus in either an aberrant location in the atrium or in the node. When SVT persists beyond a brief period of time it is often called paroxysmal atrial tachycardia (PAT).

**HISTORY** is often nonspecific. First onset is usually in infancy, unless there is underlying cardiac disease complicated by SVT. Symptoms generally first appear after congestive heart failure develops. This may be hours to days after the onset of SVT in an otherwise healthy child. It may also result from Digoxin toxicity.

**PHYSICAL EXAM** SVT of short duration may not cause symptoms (stable SVT), but with time is accompanied by signs and symptoms of or significant CHF (unstable SVT). The period before onset of symptoms is variable and will depend upon the presence of underlying heart disease and age. (Curiously, infants appear to tolerate SVT for longer periods than older patients.)

### LABORATORY

- SVT is a narrow complex tachycardia with a QRS duration less than 0.1 second and an often unidentifiable P-wave.
- Heart rate is usually greater than 220
- Lack of variability in rate/rhythm is characteristic.

### TREATMENT

Stable SVT:

- Administer oxygen.
- Establish IV access.
- Consult with MCO regarding drug therapy.

Unstable SVT:

- Provide 100% oxygen and bag-mask ventilation if respiratory failure is present. Intubate and ventilate if signs of respiratory failure persist or are severe.
- Establish IV access. Consult with MCO regarding drug therapy. Drugs used in the management of SVT may include the following:
  - Adenosine
  - Digoxin
  - Propranolol
  - Verapamil

The following scheme will usually be recommended.

- Administer Adenosine 0.05mg/kg RAPID IV push. Repeat every 2 minutes to a maximum of 0.25mg/kg or until termination of SVT. Adenosine MUST be pushed very rapidly & flushed rapidly & immediately with NS very near the IV site. The half life is very short & loses efficacy if pushed slowly.
- If pharmacotherapy fails or cardiorespiratory failure upon presentation is life-threatening, perform synchronized cardioversion with 0.5-1.0 joules/kg. If unresponsive, repeat with 2 joules/kg.
- Once rhythm is restored, a drug, typically Digoxin, is usually initiated to maintain sinus rhythm.

Refer to Adult OB / Peds: Pediatric Tachycardia adequate or poor perfusion (Protocols 512 & 513) for wide complex tachycardia (QRS>.08 sec).



## UNC Carolina Air Care Congestive Heart Failure

To identify and treat congestive heart failure

**DEFINITION** Congestive heart failure is the condition in which the heart cannot provide adequate cardiac output for the body's demand. This condition can result from:

- Excessive demand (e.g. with hyperthyroidism, arteriovenous malformation, or a variety of structural abnormalities of the heart characterized by left-to-right shunting), sepsis
- Disease or dysfunction of the myocardium
- Structural abnormalities resulting in obstruction to outflow from the heart (aortic stenosis)
- Fluid overload
- Miscellaneous causes

### HISTORY

- decreased feeding
- poor weight gain
- easily fatigued
- decreased urine output
- may have history of congenital heart disease

**PHYSICAL EXAM** Clinical signs are variable and will depend upon the severity, age of patient and whether failure is predominantly left-sided or rightsided. Signs on physical exam may include:

- diaphoresis
- irritability
- tachypnea and dyspnea
- wheezing and/or rales
- tachycardia
- decreased peripheral pulses
- delayed capillary refill
- pulsus alternans
- jugular venous distention
- decreased blood pressure in the presence of cardiogenic shock
- peripheral edema
- hepatomegaly

**XRAY DATA** Chest xray:

- cardiomegaly
- abnormal cardiac silhouette with some lesions
- engorgement of pulmonary vasculature
- radiographic findings of pulmonary edema

**LABORATORY DATA** Arterial blood gas analysis should be performed to identify infants with acidosis and/or hypoxemia. Metabolic acidosis is often present, always with cardiogenic shock.

### TREATMENT

1. Administer oxygen to ensure normoxia.
  2. Intubate and ventilate if respiratory effort is excessive or cardiogenic shock is present.
  3. Sedate with Fentanyl.
  4. Administer Vecuronium when agitated after adequate sedation is administered.
  5. Reduce fluid intake to 60% of maintenance.
  6. Administer Lasix (1mg/kg IV) if pulmonary edema is present.
  7. Consider inotropic support if cardiac output does not improve with the measures outlined above (Dopamine, Dobutamine).
-



## UNC Carolina Air Care Respiratory Failure

To identify the presence of respiratory failure in a neonatal patient, to identify the underlying pathophysiology, and to treat respiratory failure with the necessary respiratory support.

**IDENTIFICATION** Respiratory failure is present when any of the following conditions exist:

- Failure to adequately oxygenate
- Failure to adequately ventilate
- Airway obstruction
- Apnea

Pathology of the respiratory system results in functional abnormalities; the alteration in physiology causing these functional abnormalities in an individual patient represents the pathophysiology of respiratory failure in that patient and is the basis for therapy. The two major functional abnormalities in neonates requiring transport are failure to oxygenate and failure to ventilate.

Failure to oxygenate results from:

- Diffusion defects
- Hypoventilation
- Intrapulmonary shunting
- Extrapulmonary shunting

Failure to ventilate results from:

- Decreased central respiratory drive
- Upper airway obstruction
- Small airway obstruction
- Parenchymal (air space) disease

**HISTORY** Elements of the history may provide clues about the pathophysiology but are more helpful in determining the antecedent disease.

**PHYSICAL EXAM** Differential features of the physical exam include the following:

Failure to oxygenate:

Diffusion defects

- Rarely seen in the absence of an additional alteration in physiology
- No distinctive features on physical exam

Hypoventilation

- Decreased respiratory effort
- Retractions with airway obstruction
- Decreased air entry sounds

Intrapulmonary shunting

- “Abnormal” breath sounds, usually rales
- Physical evidence of non-compliance

Extrapulmonary shunting

- Relatively clear breath sounds
- Usually little or no decrease in compliance
- Heart murmur may be present if antecedent is cyanotic heart disease; usually not present if antecedent is PPHN

Failure to ventilate:

Decreased central respiratory drive

- Decreased respiratory effort; does not increase with stimulation
- Decreased depth or rate of respiration

Upper airway obstruction

- Stridor
- Other upper airway sounds

- Other findings of anatomical obstruction (e.g., choanal atresia, glossoptosis, laryngeal web)

Small airway obstruction

- Wheezing

Parenchymal disease

- Abnormal or absent breath sounds

**LABORATORY DATA** A blood gas analysis is the single most helpful laboratory test in the assessment of an infant with respiratory failure, and should be performed in the evaluation of all infants with respiratory distress.



## UNC Carolina Air Care Respiratory Distress Syndrome

To identify and treat the sequelae of surfactant deficiency in infants

### HISTORY

Infants at Risk:

- Premature infants (risk inversely proportional to gestational age; uncommon after 34 weeks)
- Infants of diabetic mothers
- Asphyxiated infants

Onset of symptoms at less than one hour of age. Progressive increase in severity during the first 48 to 72 hours of life (unless treated with surfactant)

### PHYSICAL EXAM

- Tachypnea (rate >60/min)
- Grunting
- Retractions (intercostal, subcostal, substernal)
- Nasal flaring
- Decreased thoracic volume
- Scattered rales heard throughout lung fields
- Cyanosis in room air

### XRAY DATA

Chest xray: (Prior to surfactant)

- Generalized reticulogranular pattern (ground glass appearance)
- Homogeneous density throughout lungs
- Air bronchograms
- Low lung volumes

### TREATMENT

Non-specific Therapies:

1. Discontinue enteral fluids and establish intravenous access.
2. Maintain adequate hydration & serum glucose.
3. Administer oxygen to keep PaO<sub>2</sub> 60-80 mmHg
4. Follow guidelines for treatment of respiratory failure.
5. Obtain CBC/blood culture & begin broad spectrum antibiotics (ampicillin and gentamicin).
6. Provide neutral thermal environment.

Specific Therapy:

Administer Surfactant if infants meets the following criteria:

- Diagnosis of RDS established with reasonable certainty
  - Mechanical ventilation required for treatment of RDS & oxygen requirement >30%.
  - Stable cardiorespiratory status with adequate oxygenation and ventilation
  - No contraindication to prolongation of time in referring hospital
-



## UNC Carolina Air Care Transient Tachypnea of the Newborn

To outline the management of patients with Transient Tachypnea of the Newborn (TTNB)

### HISTORY Risk Factors:

- Prematurity (although generally not extreme prematurity)
- C-section delivery
- Rapid vaginal delivery
- Mild birth asphyxia (particularly without labor)
- Maternal analgesia/anesthesia

Onset of symptoms within first hour of life. Distress usually not severe

### PHYSICAL EXAM

- Tachypnea (usually  $>80$ /min)
- Retractions, nasal flaring and grunting unusual
- Increased AP chest dimension
- Breath sounds clear but “distant”
- Mild cyanosis on room air

### LABORATORY DATA

Hypercarbia in first 8 hours of life common

### XRAY DATA Chest x-ray:

- Prominent vascular markings
- Fluid present in the fissures or pleural spaces
- Hyperinflation with flattened diaphragm on lateral view
- Chest x-ray usually clears within 24 -48 hours

### TREATMENT

1. Administer oxygen to keep PaO<sub>2</sub> 60-80mmHg.
  2. Follow Respiratory Failure Protocol for signs and symptoms of respiratory failure.
  3. Establish IV access.
  4. Discontinue enteral fluid
  5. Maintain adequate hydration & serum glucose.
  6. Administer antibiotics until pneumonia is ruled out (usually ampicillin and gentamicin).
  7. Provide neutral thermal environment
-



## UNC Carolina Air Care Meconium Aspiration Syndrome

To outline the management of patients with Meconium Aspiration Syndrome (MAS)

### HISTORY Infants at Risk:

- Any infant with meconium stained amniotic fluid during labor and delivery is at risk for aspiration. Aspiration of thick or particulate meconium places the infant at risk of developing MAS.
- Infants at risk for passing meconium in the intrapartum period include:
  - asphyxiated infants
  - post-term infants
  - infants of mothers with placental disease

### PHYSICAL EXAM

- Tachypnea
- Cyanosis often refractory to oxygen therapy alone
- Grunting, flaring, retractions
- Barrel chest; increased AP chest diameter
- Coarse breath sounds

### LABORATORY DATA

Severe hypoxemia refractory to oxygen therapy common; indicates presence of pulmonary hypertension. Hypercarbia and respiratory acidosis when airway obstruction by meconium present

### XRAY DATA

Chest x-ray:

- Non-uniform, coarse, patchy infiltrates radiating from the hilum into the peripheral field
- Focal areas of irregular aeration, some appearing atelectatic or consolidated, others emphysematous
- Hyperexpansion of the thorax with flattening of the diaphragm

### TREATMENT

1. Treat signs and laboratory evidence of respiratory failure: follow Respiratory Failure Protocol
  2. The goal of cardiorespiratory therapies should be to maintain pO<sub>2</sub> above 80 mmHg or oxygen saturation above 98%.
  3. If unresponsive to ventilatory management, consider the possibility of the pulmonary hypertension; if present follow Persistent Pulmonary Hypertension of the Newborn Protocol. Consider Surfactant administration for intubated hypoxemic infants.
  4. Obtain vascular access.
  5. Correct hypoglycemia, hypocalcemia and hypovolemia.
  6. Correct acidosis, either with buffer or ventilation depending upon the relative contribution of metabolic and respiratory acidosis, to maintain pH above 7.35.
  7. Sedate infant if agitated, particularly if agitation is associated with worsening cyanosis or desaturation.
  8. Paralysis may also be required if sedation alone is not effective.
  9. Obtain CBC and blood culture and administer antibiotics
-



To outline the management of patients with Persistent Pulmonary Hypertension of the Newborn (PPHN)

HISTORY Risk Factors:

- Term or near-term gestation
- Severe birth asphyxia
- Meconium aspiration
- Severe pulmonary parenchymal disease
- Lung Hypoplasia syndromes such as diaphragmatic hernia
- Sepsis
- Pneumonia
- Maternal use of non-steroidal anti-inflammatory agents at any time during gestation
- Respiratory distress within the first few hours of life

PHYSICAL EXAM Early signs include tachypnea and cyanosis, but work of breathing may not be markedly increased, unless there is coincidental pulmonary parenchymal disease (e.g. MAS). There is generally a rapidly increasing oxygen requirement to keep arterial oxygen tension in a satisfactory range. Cyanosis and hypoxemia usually respond poorly to increased ambient oxygen.

LABORATORY DATA Carbon dioxide excretion is generally not a serious problem. Acidosis is usually present but mild until profound hypoxia occurs. Marked liability of blood oxygenation with wide, rapid swings in PaO<sub>2</sub> are common and nearly pathognomonic of PPHN. The diagnosis of PPHN is strongly supported by observing a significant drop (15 torr) between the PaO<sub>2</sub> in the right radial artery and abdominal aorta. Also, saturation differences between pre-and post-ductal sites may be seen by pulse oximetry. Absence of this difference does not rule out PPHN, since the primary site of shunting may be at the foramen ovale.

XRAY DATA Pulmonary parenchymal densities are generally mild in comparison with the degree of clinical hypoxia. There may be scattered abnormal pulmonary densities but usually not the characteristic granularity and air bronchograms of RDS. Occasionally the scattered heavy densities of meconium aspiration syndrome with air trapping will be evident. Decreased vascular markings may be present, particularly if PPHN occurs in the absence of pulmonary parenchymal disease.

TREATMENT

Respiratory therapy:

1. All infants with PPHN should be placed in 100% oxygen throughout transport unless high PaO<sub>2</sub>'s can be maintained constantly in less oxygen.
2. If PaO<sub>2</sub> can be maintained in the 70 or above range by increasing ambient oxygen only, then ventilation should not be initiated.
3. If PaO<sub>2</sub> falls below this range in 100% oxygen, begin mechanical ventilation.
4. The goal of mechanical ventilation should be to reduce PCO<sub>2</sub> to the 30-35 range. Parameters of ventilation should be chosen to provide maximal ventilation with the least impact on mean airway pressure. Suggested initial settings are: FiO<sub>2</sub> 1.0, IMV 40, PIP 24, PEEP 4, Ti 0.3.
5. If improvement in oxygenation does not occur, increase PIP and IMV. IMV should not exceed 60/minute.

Pharmacologic therapy:

1. Agitation of an infant with PPHN should be avoided. Sedation is often helpful and necessary. Fentanyl should be administered to infants who become agitated and who are placed on mechanical ventilation.
2. Infants who are on mechanical ventilation and who do not respond to sedation with Fentanyl should be paralyzed with pancuronium or vecuronium.



### UNC Carolina Air Care Persistent Pulmonary Hypertension of the Newborn

3. The blood pH should be maintained in the 7.35 to 7.45 range. With adequate ventilation established, sodium bicarbonate may be needed to achieve this goal and should be administered by bolus or drip.
4. Blood pressure should be maintained in the normal range. The adequacy of tissue perfusion should be estimated. If blood pressure is low or tissue perfusion is inadequate, additional crystalloid or colloid should be administered. If volume expansion of 20ml/kg does not accomplish this goal, dopamine therapy should be initiated.
5. Infants who are not responding to conventional therapies may require treatment with inhaled nitric oxide; and when possible should be transported without interruption of treatment. Refer to the protocol for inhaled nitric oxide, pp.

COMMENTS Infants who have hypoxemia that is unresponsive to oxygen should be evaluated carefully for cyanotic congenital heart disease. Auscultation of the heart, palpation of the pulses, measurement of blood pressure, and examination of chest X-rays are useful. A pO<sub>2</sub> greater than 150mmHg at any time rules out nearly all types of cyanotic congenital heart disease. Infants with PPHN should be monitored continuously with pulse oximetry. Both pre- and post-ductal saturations may be helpful. The placement of an indwelling arterial catheter for blood pressure monitoring is strongly advised.

---



## UNC Carolina Air Care Diaphragmatic Hernia

To identify and treat cardiorespiratory dysfunction caused by congenital diaphragmatic hernia (CDH)

**HISTORY** There are no known risk factors for CDH. The majority of cases of CDH that present with acute respiratory distress result from hernias on the left side. Infants with CDH typically develop severe, early onset respiratory distress. Failure to respond to resuscitation immediately following delivery is common.

**PHYSICAL EXAM** Acute and severe respiratory distress; cyanosis may be unresponsive to increased ambient oxygen. Decreased or absent breath sounds on the ipsilateral side, usually the left. Bowel sounds may be audible on the ipsilateral side. Shift of heart sounds to the contralateral side. Scaphoid abdomen (may not be present after intubation and ventilation).

**XRAY DATA** Chest xray:

- Loops of intestine in the thoracic cavity
- Shift of the mediastinum to the contralateral side

**TREATMENT**

1. Immediate endotracheal intubation. DO NOT VENTILATE BY FACE MASK.
2. NG tube/repleg to low continuous suction- confirm placement by CXR.
3. Place pre-ductal pulse oximeter and transcutaneous CO<sub>2</sub> monitor.
4. Ventilation strategy:
  - a. Begin at 100 % oxygen, PIP of 20; PEEP at 4, IMV 40-50, I time 0.3.
  - b. Accept pre-ductal sats >80 %.
  - c. Accept PCO<sub>2</sub> 45-65.
5. For a base deficit >10, give 2 meq/kg of NaHCO<sub>3</sub>.
6. Sedate and paralyze.
7. Obtain vascular access (low UVC is acceptable).
8. IVF (D10W) at 80 cc/kg/day.
9. Judicious use of fluid boluses.
10. Use Dobutamine as needed for BP support.
- II. Minimize stimulation.

**COMMENTS** Diaphragmatic hernias usually result in lung hypoplasia most prominent on the ipsilateral side. Rupture of either lung is common and should be treated with a thoracostomy tube. Prevention of pneumothorax is essential. Low pressures, higher rates and short inspiratory time will help prevent pneumothoraces. Pulmonary hypertension also frequently accompanies diaphragmatic hernia as a result of lung hypoplasia.

**SAFETY:** Transilluminate prior to departure and enroute if deterioration.

---



## UNC Carolina Air Care Gastroschisis / Omphalocele

To identify and treat problems associated with congenital defects of the abdominal wall

### HISTORY

**Gastroschisis:** There are no known risk factors for gastroschisis. Because of the widespread use of prenatal fetal ultrasound, gastroschisis is now more often identified prenatally. Most cases are anticipated prior to delivery. Because of the high association with high GI obstruction, polyhydramnios is common, therefore, preterm delivery is common.

**Omphalocele:** There are no known risk factors associated with omphalocele except when associated with a syndrome (e.g. Beckwith- Wiedemann syndrome). Most large omphaloceles are identified during prenatal ultrasound. However, small lesions may go unrecognized.

### PHYSICAL EXAM

#### Gastroschisis

- Full thickness defect of the anterior abdominal wall, most commonly to the right and slightly below the umbilicus.
- The organs often located outside the abdomen can include stomach, small bowel, colon and, rarely, the liver.
- Exposed viscera may have fibrinous coating or may be under torsion.

#### Omphalocele

- Full thickness defect of the abdominal wall at the umbilicus which includes the base of the umbilical cord
- Contents depend primarily upon the size of the defect
- No exposure of viscera if membrane intact.

#### General Clinical Signs

- Hypothermia often present, particularly with gastroschisis
- Hypovolemia/hypotension if fluid loss not replaced adequately (rarely occurs with intact omphalocele)
- Potential for respiratory distress which is usually linked to the degree of prematurity

**LABORATORY DATA** Metabolic acidosis if hypovolemia present. Hypoglycemia if in association with Beckwith-Wiedemann syndrome.

### TREATMENT

1. Assure adequate oxygenation and ventilation
2. Bowel should be positioned and supported, if necessary, so that no tension or torsion is applied to the viscera. IF BOWEL HAS BEEN WRAPPED PRIOR TO ARRIVAL OF THE TEAM, IT SHOULD BE UNWRAPPED AND OBSERVED FOR TORSION AND REWRAPPED.
3. Cover exposed viscera with warm sterile saline dressing, turban-style. Put infant in a sterile bowel bag secured at the level of the axilla.
4. Place infant on side and support abdomen with diapers if necessary.
5. Repleg to low suction.
6. Administer parenteral fluids at 1.5 times maintenance with salt containing fluid (usually D<sub>10</sub>W 1/4NS).
7. Correct acid-base balance.
8. Provide neutral thermal environment.
9. Begin broad spectrum antibiotics.
10. Monitor serum glucose.

**COMMENTS** Omphaloceles are usually easily distinguished from gastroschisis by the covering membrane and the involvement of the umbilical cord seen in the former. A ruptured omphalocele should be treated like a gastroschisis. An intact membrane will decrease fluid loss. Gastroschisis is rarely associated with other congenital defects, although commonly associated with preterm birth. Omphaloceles, however, have a high incidence of accompanying anomalies: Cardiac, renal, neurological and chromosomal.

---



## UNC Carolina Air Care Neural Tube Defect

To support a patient with a neural tube defect, including patients with meningocele, meningocele, and encephalocele

**HISTORY** Because of the availability of maternal AFP screening and prenatal ultrasound, neural tube defects are usually identified prior to delivery and some are repaired in-utero. Maternal history may include poor fetal growth, decreased fetal movement or polyhydramnios.

### PHYSICAL EXAM

- Note size, shape, and contents of lesion.
- Note presence of covering membrane and any leakage of CSF.
- Measure the head circumference.
- Assess intracranial pressure by palpating the anterior fontanel.
- Palpate the abdomen for bladder size and emptying ability using the crede maneuver.
- Note position and movement of lower extremities.

**XRAY DATA** Radiographic evaluation of infants with neural tube defects is critical in planning appropriate therapies, but is not necessary during transport.

### TREATMENT

1. Position prone and prevent sac from rupture.
  2. Apply sterile non-adherent dressing (e.g. Telfa), moistened with saline over the defect.  
DO NOT USE BETADINE!! IT KILLS NERVE CELLS!!
  3. Apply 4x4 over dressing and secure with Kerlex.
  4. For open lesions, obtain a swab for culture.
  5. Do not use sterile isolation bag. May wrap with Kerlex.
  6. Prevent contamination of defect with urine or stool.
  7. Place IV and begin maintenance fluids.
  8. Begin broad spectrum antibiotics (usually ampicillin and gentamicin).
-



## UNC Carolina Air Care Necrotizing Enterocolitis

To identify and treat problems associated with necrotizing enterocolitis (NEC)

### HISTORY Infants at Risk:

- Preterm infants
- Infants exposed to cocaine
- Associated with other complications of prematurity (e.g., PDA, RDS, asphyxia, hypothermia, exchange transfusions)
- Term infants with a history of sepsis, acidosis and/or hypoxemia
- Enteral feeding (feeding with breast milk protective)
- Unusual in first three days of life; rarely seen after three weeks

### Historical Clues:

- Poor feeding
- Temperature instability
- Lethargy
- Irritability
- Apnea
- Diarrhea
- Gastric residuals

### PHYSICAL EXAM

- Abdominal distention and tenderness
- Rubor or bluish discoloration of the abdominal wall
- Absent bowel sounds
- Poor skin perfusion
- Hypotension/shock
- Bile stained gastric aspirate
- Respiratory compromise

### LABORATORY DATA

- Occult and/or gross blood in stools
- Stools with increased reducing substance (over I+ on a Clinitest tape)
- Metabolic acidosis
- Thrombocytopenia
- Neutropenia
- Hyponatremia
- Laboratory evidence of coagulopathy

### XRAY DATA

- Dilation of the bowel
- Persistent nonmotile loops of bowel
- Thickened bowel wall
- Pneumatosis intestinalis (diagnostic)
- Free air within the peritoneal cavity
- Air in the portal venous system

### TREATMENT

1. Assure adequate oxygenation and ventilation.
2. Obtain IV access.
3. Treat hypovolemia or shock.
4. NPO.
5. Place replegic to low intermittent suction.
6. CBC, blood culture, stool culture (if possible), ABG.
7. Broad spectrum antibiotics (ampicillin, and gentamicin).
8. Metronidazole should be added to antibiotic coverage if perforation has occurred or if perforation is likely as evidenced by a rapidly deteriorating condition.
9. Maintenance IV fluids should be adjusted to individual needs:
  - Because of capillary damage, third spacing and excessive fluid loss into the gut or peritoneum, an increase in maintenance fluids above normal may be required, usually 1.5 times maintenance. D5W with electrolytes is satisfactory (omit KCl if the infant is acidotic or oliguric).
  - The goal for urine output should be 1-1.5cc/kg/hr.

COMMENTS Necrotic bowel causes marked increase capillary permeability



## UNC Carolina Air Care Necrotizing Enterocolitis

throughout the entire body with the resulting sequestration of large volumes of fluid in the extravascular spaces.



## UNC Carolina Air Care EA / TEF

To identify and treat the complications of esophageal atresia and tracheoesophageal fistula (EA/TEF)

**DEFINITION** A congenital interruption of the esophagus (esophageal atresia) may be associated with fistulous connections between the tracheal/bronchial tree or distal GI tract.

**HISTORY** There are no known risk factors for EA/TEF. These lesions are not easily identified during prenatal ultrasound unless EA is present in the absence of TEF. With this anomaly, polyhydramnios is invariably present and a stomach “bubble” is not seen on fetal ultrasound. Birth is more likely to be premature.

### PHYSICAL EXAM

- Excessive saliva production, drooling
- Inability to pass NG tube into stomach
- Choking, cyanotic episodes, tachypnea, wheezing
- Scaphoid abdomen if EA present without TEF

### XRAY DATA

- Radiographic documentation of coiling of the NG tube in the esophagus
- Esophageal pouch (often filled with air) is visible
- Air in GI tract confirms presence of TEF; absence implies EA without TEF

### TREATMENT

1. Provide airway and/or ventilatory support as needed.
  2. Place infant on right side and elevate head and chest to a 45 degree angle.
  3. Place a Replogle in esophageal pouch and place to low-intermittent suction.
  4. NPO; provide maintenance IV fluids.
  5. Avoid agitation. During valsalva associated with agitation, air is forced into the stomach through the fistula. The stomach distends and reflux of gastric content into the lungs may occur and cause severe respiratory distress.
-



## UNC Carolina Air Care Cyanotic Congenital Heart Disease

To outline the care of an infant with suspected or confirmed cyanotic congenital heart disease

**DEFINITION** Cyanotic congenital heart disease occurs whenever there is an anatomic abnormality which:

- decreases pulmonary blood flow
- causes circulation of venous return directly into systemic outflow
- causes separation of the systemic and pulmonary circulation.

**HISTORY** Infants are usually term. There is usually no history of prenatal/intrapartum complications.

**PHYSICAL EXAM:**

- Profound cyanosis in an infant who may otherwise appear well
- No improvement in cyanosis with 100% oxygen
- Usually not initially acidotic, but metabolic acidosis may develop with prolonged hypoxemia
- Typically ventilation is normal with clear breath sounds
- Heart murmur or other auscultative findings may or may not be present

**XRAY DATA** Abnormal size or shape of heart or decreased pulmonary vascular markings may be present. Pulmonary vascular markings can be increased as with truncus arteriosus. Lung fields are usually clear.

**TREATMENT**

1. Place infant in 100% oxygen.
2. Intubate and ventilate as needed for apnea or labored respiratory effort. Infants on prostaglandin infusions should be considered for intubation prior to transport due to the possible side effect of apnea. The risk of apnea is greatest in the first hours of prostaglandin infusion.
3. Consult MCO to discuss use of prostaglandins. Consider prostaglandin therapy if:
  - $PO_2 < 30-40$  mmHg and/or oxygen saturation  $< 80\%$
  - Femoral pulses diminished or absent with poor perfusion
  - Metabolic acidosis persists with good ventilation and volume/ inotrope support.
  - There is a high likelihood of cyanotic congenital heart disease based on history/exam/data
4. Sedate if agitated.
5. Consider bicarbonate administration to correct metabolic acidosis if adequately ventilating.
6. Monitor metabolic parameters (glucose, calcium) and correct if necessary.



To outline management of patients with upper airway obstruction

***CROUP (INCLUDES BACTERIAL TRACHEITIS AND SUBGLOTTIC STENOSIS)***

1. Signs, symptoms: mild elevation of temperature, malaise, rhinitis, loss of appetite.
2. After 2-3 days, development of hoarseness, inspiratory stridor, barking cough, tachypnea, retractions.

**TREATMENT**

1. Minimize agitation.
2. Provide supplemental oxygen in a manner patient will tolerate.
3. Avoid respiratory depressants or sedatives.
4. Administer racemic epinephrine(0.25cc in 2cc NS by nebulization).
5. Consider dexamethasone 0.5-0.6 mg/kg - contact MCO.
6. Discontinue any known drying agents antihistamines, atropine
7. Decision to intubate is generally made on clinical grounds, not with ABG's.
8. If intubation is needed, use ETT one size smaller than usual for age.
9. Administer IV fluids at 1.5 times maintenance.
10. Discontinue oral intake.

***EPIGLOTTITIS***

1. Short prodrome of 6-10 hours with high fever (38-40 degrees Celsius).
2. Signs, symptoms: sore throat, malaise, rapid development of dysphagia, inability to swallow, drooling, inability to lie down, dyspnea.

**TREATMENT**

1. Maintain close observation.
  2. Do not aggravate the patient(no labs, no ABG's, no IV, do not place in horizontal position).
  3. Administer humidified oxygen.
  4. Controlled intubation in OR with anesthesiologist and ENT.
  5. Secure endotracheal tube.
  6. Sedate after airway secured.
  7. Muscle relaxants may be needed.
  8. Respiratory isolation.
  9. Maintenance IV fluids.
  10. Antipyretics for temperature>38.5C.
  11. Tracheal aspirate culture post intubation.
  12. Obtain blood culture.
  13. Begin antibiotics.
-



To outline care of patients with lower airway obstruction

***PNEUMONIA/BRONCHIOLITIS***

1. Mild upper respiratory infection for several days, abrupt fever >39.0, productive or dry cough.
2. Signs/symptoms: respiratory distress, tachypnea, retractions with use of accessory muscles, cyanosis.
3. Auscultation: rales, wheezes.
4. Chest x-ray: indicative air trapping, thickening of bronchial wall, interstitial pneumonia, atelectasis.

**TREATMENT**

1. Establish airway, ventilate as required.
2. Administer oxygen with humidity.
3. Bronchodilators may be needed for bronchospasm & wheezing.
4. Respiratory isolation techniques.
5. Sedation, muscle relaxants.
6. Administer IV maintenance fluid.
7. Obtain blood culture.
8. Initiate antibiotic therapy.

***ASTHMA***

1. Signs, symptoms:
  - d. Respiratory distress
  - e. Cough
  - f. Expiratory wheeze
  - g. Prolonged expiratory phase
  - h. Prolonged inspiratory phase (if severe)
  - i. Dehydration
2. Labs: ABG, CBC
3. Chest x-ray: infiltrates, patchy atelectasis, hyperinflation
4. Note quality of cry or ability to talk (indicators of patient's forced expiratory volume)

**TREATMENT**

1. Keep a calm atmosphere, allow to assume a position of comfort.
  2. Administer oxygen 2-4 liters per minute by mask or nasal cannula.
  3. Aerosolized bronchodilators:
    - Albuterol-(0.5% solution) 0.03ml/kg; max=5mg(1cc). May use hourly. Alternatively, give continuous nebulization of 0.4-0.8 mg/kg/hr; max of 15 mg/hr.
    - Atropine-(0.5 mg/ml) 0.075 mg/kg (max 2 mg every six hours)
    - Glycopyrrolate-(0.2 mg/ml) 0.025-0.05 mg/kg every 6 hours. (preferred with atropine)
  4. Adrenergic agents:
    - Epinephrine -I:1000 SQ: 0.01cc/kg/dose (max=0.03cc); may repeat two times at 20 minute intervals
    - Terbutaline -1mg/cc SQ: 0.01cc/kg/dose(max= 0.3cc); may repeat once in 30 minutes
    - Aminophylline (for those not on chronic theophylline therapy) Load: 6 mg/kg over 20 minutes  
Maintenance: 1 mg/kg/hr If a dose is missed, give 2.5mg/kg and then start an aminophylline drip of 1mg/kg/hr Therapeutic level is 10-20mcg/ml
    - Steroids: Methylprednisone - Load: 2 mg/kg, maintenance: 1 mg/kg every six hours
  5. Administer antibiotics as indicated
  6. Mechanical ventilation.
-



To outline the care of patients with diabetic ketoacidosis

### SUPPORTIVE DATA

1. Serum glucose >300
2. Metabolic acidosis with pH <7.2
3. Serum bicarbonate <15
4. Dehydration
5. Ketonuria
6. Lab values: decreased Na, K, CO<sub>2</sub>, PO<sub>4</sub>, and pH; increased BUN, glucose

### TREATMENT

1. Manage airway appropriately.
2. Establish IV access.
3. Assume 10% dehydration.
4. If shock present, bolus with 10-20cc/kg NS/LR and repeat as needed.
5. Maintenance fluid:
  - Glucose >300 = 0.45 NS with KCl if +urine output.
  - Glucose <300 = D10% 0.45NS with KCl if +urine output.
6. Rate = 2 times maintenance rate.
7. Insulin drip: 0.1 U/kg/hr. Mix I:I in NS using Regular insulin and remember to flush the tubing.
8. Sodium bicarbonate only by MCO order.
9. Monitor serum glucose every hour.
9. Be observant for cerebral edema and increased ICP.

### COMMENTS

1. Initial rehydration will decrease glucose and increase pH even without insulin therapy.
  2. Glucose should not drop more than 50% in the first six hours.
    - Titrate insulin drip for glucose drop of 50-100 mg/dl/ hour.
    - If admission glucose over 800, drop only by 50mg/dl/ hour to prevent cerebral edema.
-



To outline the care of patients with altered level of consciousness, increased intracranial pressure, and status epilepticus.

### ***ALTERED LEVEL OF CONSCIOUSNESS***

#### **SUPPORTIVE DATA**

History: diabetic imbalance, drug/alcohol ingestion, trauma, infection, heat stroke, cardiac dysrhythmias.

#### **TREATMENT**

1. Manage airway and oxygen appropriately.
2. Stabilize spine: in line cervical stabilization must be applied if trauma has not been ruled out. [Refer to adult team Pediatric Head Trauma if suspected].
3. Establish IV access.
4. Volume resuscitation as needed.
5. If there is an obvious overdose of narcotics, give Narcan 0.1mg/kg IV or IM.
6. Begin maintenance IV fluids.
7. Follow neurological exam. If condition deteriorates, begin measures to lower the ICP.
8. After facial trauma ruled out, place a nasogastric tube and decompress the stomach.
9. Consider activated charcoal.
10. Contact MCO for pharmacologic agents.

### ***STATUS EPILEPTICUS***

#### **SUPPORTIVE DATA**

1. History-recurrent seizures without awakening.
2. Neonatal causes - congenital or acquired metabolic disorders, hypoxia, congenital malformation, CNS infections, hemorrhage.
3. Infant/child causes - head trauma, child abuse, toxins, meningitis, idiopathic.
4. Adolescent causes - metabolic disorders, drug withdrawal, uremia, toxins, CNS tumors, head injury, idiopathic, drug overdose.

#### **TREATMENT**

1. Maintain airway and oxygenate appropriately.
2. Establish vascular access.
3. Correct electrolyte abnormalities.
4. Anticonvulsants to stop seizures:
  - \* Lorazepam 0.1 mg/kg or Valium 0.2-0.4mg/kg. Then load with Phenobarbital 20mg/kg or Phenytoin 18mg/kg.
5. Fluid resuscitate as needed.
6. Begin maintenance IV fluids, use D5NS or D51/2NS.
7. Follow neurological exam.
8. NGT to suction.
9. Antipyretics for hyperthermia.
10. Antibiotics as indicated.



II. Anticonvulsant levels.

#### COMMENTS

1. Apnea may result from the use of some medications to treat seizures, especially when phenobarbital is combined with a benzodiazepine (diazepam, lorazepam, midazolam).
2. At night, the blinking lights in the helicopter or ambulance may induce seizure activity, cover the patient's eyes.

#### *INCREASED INTRACRANIAL PRESSURE*

#### SUPPORTIVE DATA

History of impaired CSF dynamics, increased volume of brain tissue, increased cerebral blood volume, mass lesion.

#### TREATMENT

1. Maintain airway and oxygenate appropriately.
2. Intubate and ventilate if necessary. Lidocaine can be used as an adjunct to intubation or for suctioning.
3. Ventilate - PCO<sub>2</sub> 30-35, minimize PEEP, maintain PaO<sub>2</sub> >100.
4. Maintain Mean Arterial Pressure 50mmHg greater than ICP, or at least 70.
5. Establish vascular access.
6. Treat seizures.
7. If volume is needed, expand with colloids.
8. Sedate if agitated.
9. Antipyretics for hyperthermia.
10. Consider Mannitol (0.25-1gm/kg), or Lasix (0.5-1.0 mg/kg).
11. Place foley catheter.
12. Elevate head of bed, keep head in neutral position.

#### COMMENTS

1. Muscle relaxants will mask seizure activity; if needed, consider loading with anticonvulsant.
  2. Sedation will alter neurologic exam.
  3. Noise, vibration, light, and noxious stimuli may increase the intracranial pressure.
-



To identify and manage pathology associated with ingestion or other exposures to poisonous substances

### ASSESSMENT

1. Refer to Assessment Protocol.
2. Note burns in mouth, nose, etc.
3. Note any odors.
4. Note any material around mouth.
5. Treat the patient not the poison.

### SUPPORTIVE DATA

Obtain history:

- Define product name, ingredients, amount consumed, and current symptoms.
- If there is any question regarding a medication, request the prescription number, dispensing pharmacy, or prescribing physician.
- Always request that the product be brought in with the child.

### TREATMENT

1. Manage airway and oxygenate appropriately.
2. Establish IV access.
3. Treat shock.
4. Gather available laboratory data: urine, blood, gastric aspirate, ABG, electrolytes, glucose, ammonia, liver enzymes, CXR.
5. *Decontamination:* for dermal or ophthalmic exposure, flush with copious amounts of water for at least 15 minutes.
6. Ingested poisons-the goal is to delay absorption and enhance elimination.
7. *Emesis:*
  - A. Syrup of Ipecac (15ml) PO or NG, may repeat one time if no emesis.
  - B. Save product of emesis.
  - C. Contraindications:
    - the unconscious patient.
    - LOC has or may deteriorate.
    - absent gag reflex.
    - ingestion of caustic agents.
    - ingestion of most antiemetic drugs.
    - ingestion of most hydrocarbons(gasoline, kerosene, etc.).
8. *Gastric lavage*
  - A. Indicated when patient is unconscious, rapidly losing consciousness, seizing, or has no gag reflex.
  - B. Intubate prior to lavage.
  - C. Use a large Ewald tube.
  - D. Continue lavage until absence of solid material.



## UNC Carolina Air Care Poisoning

E. Position patient left side with head down.

9. *Medications and Antidotes*

A. Catharsis - MgSO<sub>4</sub>(Epsom salts) 250mg/kg

Contraindications:

- Magnesium-containing cathartics in patients with renal failure.
- Oil based cathartics in pesticide poisoning.

B. Activated charcoal - binds with most ingested drugs -dosage 1gm/kg (use sorbitol for first dose).

C. Toxins which require immediate antidotes:

- Carbon monoxide - 100% oxygen.
- Cyanide:
  - Amyl nitrate valproate-crush and have patient inhale 30 out of every 60 seconds
  - Sodium nitrate-10 mg/kg IV push.
  - Sodium thiosulfate-1.65cc/kg of 25% solution.
  - Nitrites/nitrates-give 0.2mg/kg of a 1% solution of methylene blue IV over 5 minutes.

## 10. Specific Poison

A. **Acetaminophen**

Signs/symptoms:

- diaphoresis
- anorexia, nausea, vomiting
- increased AST (SGOT)
- increased prothrombin time, increased bilirubin
- oliguria
- jaundice
- encephalopathy

B. **Caustic substances** (lyes, strong bleaches, dishwasher and laundry powders)

Signs/symptoms:

- Drooling, vomiting, stridor, respiratory distress, abdominal pain.
- Do Not perform lavage or administer fluids charcoal, or ipecac to children with caustic ingestion.

C. **Tricyclic antidepressants**

Signs/symptoms:

- flushing, dry skin and mouth,
- dilated pupils, urinary retention, sedation, confusion, agitation.

D. **Salicylates**

Signs/symptoms:

- deep, driven respirations, decreased level of consciousness, vomiting, tinnitus.

E. **Narcotics and barbiturates**

Signs/symptoms:

- CNS depression, respiratory depression, miotic pupils, hypotension, seizures.
- Treatment:



### UNC Carolina Air Care Poisoning

- Narcotics-Naloxone give 0.1mg/kg, repeat the dose every 5-10 minute prn.
- Barbiturates-gastric emptying.

II. Alert MCO of changes in patient status.

---