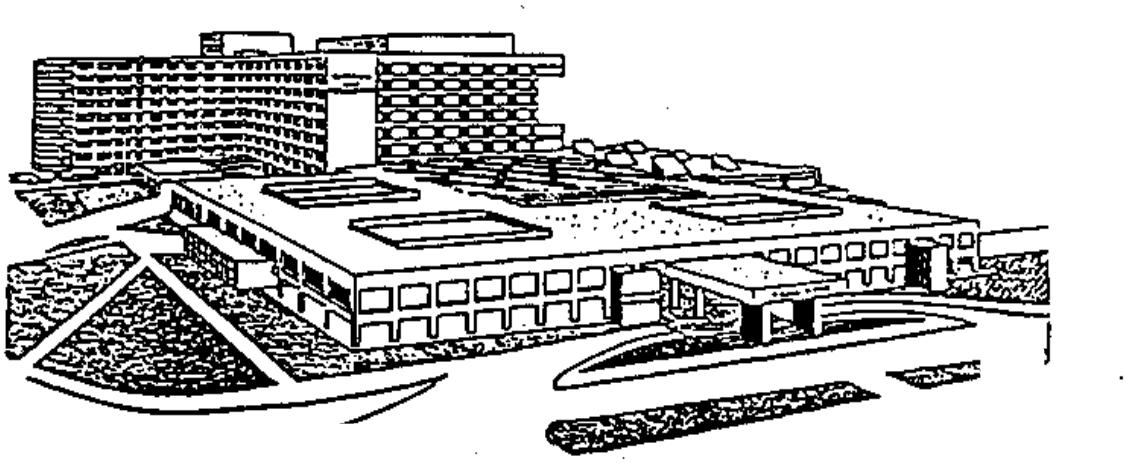
Pediatric Emergency Manual



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Department of Pediatrics

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PREFACE to the 2000 EDITION

This manual was developed for use by pediatric housestaff as a pocket-sized guide to the stabilization of potentially ill children. It is not a policy manual, but offers the developing physician one reasonable framework upon which knowledge and practice may be built. It is our hope that this manual, used in conjunction with other sources, will contribute to resident education and patient care by allowing rapid access to basic principles of the initial management of pediatric patients.

This manual was founded on the efforts of Dr. Jeff Butler and expanded upon by subsequent editors Drs. Bruce Banwart, Shelley McNair, and Keith Kerr. The current editor thanks them as well as the many other physicians and support staff, past and present, who have contributed to this work including: Drs. S. Berg, J. Brownlee, H. Cheu, D. Devoid, J. Doski, K. Dykstra, R. Fiser, B. Foley, A. Goins, M. Grimley, R. Haws, M. Kuskie, R. Moore, R. Morse, W. Rogers, J. Roscelli, L. Shaffer, and B. Wilson; Ms. V. Muraira, and Mr. R. Shick.

Erica A. Kirsch, MD

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ALGORITHMS

Remember: Doses of epinephrine given **via ETT** is 0.1 mg/kg in children and 0.01 mg/kg in neonates of the 1:1000 solution (1 mg max.).

Epi. doses: IV, IO first dose is 0.01 mg/kg of the 1:10,000 soln., second and subsequent doses: 0.1 mg/kg of the 1:1000 soln. (1 mg max.)

BRADYCARDIA

IF **SECONDARY** TO **HYPOXIA** AND **ISCHEMIA** OR IN A **NEONATE:**
100% OXYGEN AND VENTILATE, CHEST COMPRESSIONS IF INDICATED
EPINEPHRINE 0.01 MG/KG IV **(1:10,000),** REPEAT Q 3 - 5 MIN. PRN
IF NO RESPONSE CONSIDER INTUBATION AND REPEAT **LOW DOSE EPINEPHRINE**

IF **PRIMARY**,
100 % OXYGEN AND VENTILATE, CHEST COMPRESSIONS IF INDICATED
EPINEPHRINE 0.01 MG/KG IV **(1:10,000),** REPEAT Q 3 - 5 MIN. PRN
IF NO RESPONSE CONSIDER INTUBATION AND REPEAT LOW DOSE EPINEPHRINE
ATROPINE 0.02 MG/KG; MINIMUM OF 0.1 MG (1 CC)
REPEAT Q 5 MIN. PRN
(MAX. ATROPINE **SINGLE** DOSE CHILD: 0.5 MG; ADOLESCENT: 1.0 MG)
EXTERNAL PACEMAKER OR EPINEPHRINE OR ISOPROTERENOL DRIP

SUPRAVENTRICULAR TACHYCARDIA - STABLE

VAGAL MANEUVERS ETC. PER CARDIOLOGY PROBLEMS CHAPTER

SUPRAVENTRICULAR TACHYCARDIA - UNSTABLE

100 % OXYGEN, VENTILATE, ANALGESIA/SEDATION IF TIME
SYNCHRONIZED CARDIOVERSION, 0.5 - 1.0 JOULES/KG
(IF PATIENT IS DIGITALIZED, GIVE PRE-CARDIOVERSION LIDOCAINE BOLUS, 1 MG/KG)
IF IV AVAILABLE, MAY USE ADENOSINE 0.1 MG/KG RAPID IV BOLUS,
FOLLOW BY 5 - 10 CC FLUSH(DOUBLE SYRINGE TECH)
IF NO EFFECT, DOUBLE ADENOSINE DOSE (MAX. SINGLE DOSE: 12 MG)
EXPECT TRANSIENT ASYSTOLE AFTER AN ADENOSINE DOSE

ELECTROMECHANICAL DISSOCIATION
ALSO CALLED PULSELESS ELECTRICAL ACTIVITY (PEA)

CPR, CONTINUE UNTIL PULSE RETURNS
SECURE AIRWAY, 100 % OXYGEN, VENTILATE
EPINEPHRINE 0.01 MG/KG IV **(1/10,000)**
REPEAT EPINEPHRINE 0.1 MG/KG IV **(1/1000)** Q 3 - 5 MIN. PRN
CONSIDER: PNEUMOTHORAX, CARDIAC TAMPONADE, HYPOVOLEMIA, PULMONARY EMBOLISM, HYPOTHERMIA, SEVERE HYPOXIA OR ACIDOSIS

ASYSTOLE

UNRESPONSIVE, APNEIC, PULSELESS
START CPR; CONTINUE UNTIL PULSE RETURNS, EXCEPT TO CHECK PULSE
INTUBATE, 100 % OXYGEN, VENTILATE, START IV
EKG SHOWS ASYSTOLE
EPINEPHRINE 0.01 MG/KG IV **(1/10,000)**
REPEAT EPINEPHRINE 0.1 MG/KG IV **(1/1000)** Q 3 - 5 MIN. PRN

CHECK PULSE
? ATROPINE;(0.02 MG/KG; MINIMUM 0.1 MG (1 CC) IV)
CONSIDER PACEMAKER

VENTRICULAR TACHYCARDIA WITH PULSE (UNSTABLE)

100 % OXYGEN, VENTILATE, ANALGESIA/SEDATION IF TIME
CARDIOVERT 0.5 - 1.0 JOULES/KG **SYNCHRONIZED** (MAY GIVE LIDOCAINE FIRST)
CHECK RHYTHM
IF STILL V-TACH CARDIOVERSION 1.0 - 2.0 JOULES/KG
CHECK RHYTHM
IF STILL V-TACH CARDIOVERSION 2.0 - 4.0 JOULES/KG
CHECK RHYTHM
IF STILL V-TACH, INTUBATE (IF NOT ALREADY DONE), LIDOCAINE 1 MG/KG IV PUSH
CARDIOVERT, CHECK RHYTHM
IF STILL V-TACH, CONSIDER BRETYLIUM 5 MG/KG **SLOW** IV PUSH
CHECK RHYTHM
IF STILL V-TACH, CARDIOVERSION

VENTRICULAR FIBRILLATION & VENTRICULAR TACHYCARDIA WITH*OUT* PULSE

DEFIBRILLATE IF READILY AVAILABLE. IF NOT SECURE AIRWAY, 100 % OXYGEN, VENTILATE
START CPR; CONTINUE UNTIL PULSE RETURNS, EXCEPT TO CHECK PULSE OR TO SHOCK
EKG SHOWS V-FIB OR V-TACH WITHOUT PULSE
DEFIBRILLATE 2 JOULES/KG AND CHECK RHYTHM WITH PADDLES **QUICKLY(DO NOT LIFT PADDLES OFF CHEST)**
IF NO RHYTHM DEFIBRILLATE 4 JOULES/KG, CHECK RHYTHM WITH PADDLES **QUICKLY**
IF NO RHYTHM DO **NOT** WAIT TO CHECK PULSE OR DO CPR
DEFIBRILLATE AGAIN 4 JOULES/KG
IF NO PULSE CONTINUE CPR, INTUBATE (IF NOT ALREADY DONE), START IV
EPINEPHRINE 0.01 MG/KG IV **(1/10,000)**
DEFIBRILLATE 4 JOULES/KG
LIDOCAINE 1 MG/KG IV
IF NO PULSE DEFIBRILLATE 4 JOULES/KG, 30 - 60 SEC'S AFTER MEDICATIONS
CHECK PULSE
(IF CONVERTS START LIDOCAINE DRIP 20 - 50 **MCG**/KG/MIN. IV)
EPINEPHRINE 0.1 MG/KG IV **(1/1000),** REPEAT Q 3 - 5 MIN. PRN
DEFIBRILLATE 4 JOULES/KG
LIDOCAINE 1 MG/KG IV
IF NO PULSE DEFIBRILLATE 4 JOULES/KG, 30 - 60 SEC'S AFTER MEDICATIONS
CHECK PULSE, IF ABSENT EPINEPHRINE 0.1 MG/KG IV **(1/1000);** REPEAT Q 3 - 5 MIN.
DEFIBRILLATE 4 JOULES/KG
LIDOCAINE 1 MG/KG IV
DEFIBRILLATE 4 JOULES/KG
CONSIDER BRETYLIUM 5 MG/KG RAPID IV PUSH
IF NO PULSE DEFIBRILLATE 4 JOULES/KG, 30 - 60 SEC'S AFTER MEDICATIONS
CHECK PULSE
IF NO PULSE, BRETYLIUM 10 MG/KG RAPID IV PUSH
IF NO PULSE DEFIBRILLATE 4 JOULES/KG, 30 - 60 SEC'S AFTER MEDICATIONS

CHEST COMPRESSIONS IN CPR

(*1/3rd - 1/2 the chest diameter is an alternative depth for compressions*)

	Neonate (< 1 mos.)	**Infant (1 mos.- 1 yr.)**	**Child (1 yr.- 8 yrs.)**	**Adol./Adult (> 8 yrs.)**
Depth				

(inches)	1/2 - 3/4	1/2 - 1	1 - 1.5	1.5 - 2
Rate (per minute)	120	at least 100	100	80 - 100
Compressions: Ventilations	3:1 (NRC)	5:1	5:1	15:2
Position	1 finger breadth below nipple line	1 finger breadth below nipple line	1 finger breadth above xiphoid notch	1 finger breadth above xiphoid notch

PEDIATRIC ADVANCED LIFE SUPPORT

DRUG	**HOW SUPPLIED**	**SINGLE *DOSE/KG***	**MAX. or ADULT DOSE**
ADENOSINE	3 MG/CC	0.1 **MG/KG** = 0.03 **CC/KG**	DOUBLE DOSE IF NO EFFECT, MAX. DOSE = 12 MG
ATROPINE	0.1 MG/CC	0.02 MG = 0.2CC/KG (MIN: 0.1 MG = 1.0 CC= 5 KG OR UNDER)	0.5 MG = 5 CC (CHILD) 1 MG = 10 CC (ADOL.) 2 MG = 20 CC (ADULT)
BRETYLIUM	50 MG/CC	5-10 MG = 0.1 - 0.2 CC	30 MG/KG
CALCIUM~~ (elemental)	CHLORIDE 10% GLUCONATE 10%	20 MG = 0.2 CC 100 MG = 1.0 CC	INFUSE SLOWLY 500-800 MG = 5-8 CC
EPINEPHRINE#	1:10,000 (INITIAL IV,IO) (0.1 MG/CC), 1: 1000 (ETT, HIGH DOSE IV,IO) (1 MG/CC)	0.01 MG = 0.1 CC (IV) 1:10,000 0.1 MG = 0.1 CC (ETT) 1:1000	1.0 MG
GLUCAGON	1 MG/VIAL OR 10 MG MULTI-USE VIALS	<10 KG;0.1 MG/KG, >10 KG;1 MG/DOSE IV,IM,SQ	1 MG MAX, GIVE GLUCOSE AS NEEDED, FOLLOW DEX'S
GLUCOSE	D25W	0.5 - 1.0 GRAM = 2 - 4 CC	N/A
LIDOCAINE	1% SOLN. (10 MG/CC)	1.0 MG = 0.1 CC	1.0 MG/KG, MAX CUMULATIVE = 3 MG/KG
NaHCO3*	0.5 OR 1 MEQ/CC	1 - 2 MEQ	1.0 MEQ/KG
NALOXONE^	0.4 MG/CC 1.0 MG/CC	0.1 MG = 0.25 CC 0.1 MG = 0.1 CC	$\geq$ 5 YRS. OR $\geq$ 20 KG, USE 2.0 MG
VOLUME	LR, NS,5% ALB.	20 CC/KG	N/A

1. DO NOT GIVE CACL PERIPHERALLY UNLESS AN EMERGENCY. iF INFILTRATES USE WYASE.

2. # 0.1 - 0.2 MG/KG OF 1/1000 EPI AS SECOND DOSE FOR PULSELESS ARRESTS

3. * IF BASE DEFICIT KNOWN BY ABG: MEQ'S of BICARB = (BASE DEF. X 0.3 X KG)/2

4. ^ UNLESS USING FOR UNKNOWN COMA, TITRATE THE DOSE BASED ON CLINICAL STATUS. REVERSING PAIN MEDICATION TOO QUICKLY COULD CREATE A PAIN CRISIS.

5. ETT DRUGS: (NAVEL) - NALOXONE, ATROPINE, VALIUM, EPI., LIDOCAINE

6. WEIGHT ESTIMATES:

TERM NEWBORN	3.5 KG	BIRTHWEIGHT (BW)
6 MOS. OLD	7.0 KG	2 X BW
1 YEAR OLD	10 KG	3 X BW
4 YEAR OLD	16 KG	1/4 ADULT (70 KG)
10 YEAR OLD	35 KG	1/2 ADULT (70 KG)

6. ETT **SIZE -** internal diameter (ID) in mm:
INFANT/CHILD **<** 2 yrs : 4.0, or 4.5, or 5.0 ETT
CHILD > 2 yrs : (16 + age in yrs)/4 or (age in yrs/4) + 4
ESTIMATED DISTANCE (cm): **ALWAYS GET A CXR**
NEONATE: 1 kg = 7 cm, 2 kg = 8 cm, 3 kg = 9 cm, 4 kg = 10 cm
INFANT OR CHILD: ETT ID X 3 (eg. 3.5 ETT = 3.5 X 3 = 10.5 cm)

PEDIATRIC ADVANCED LIFE SUPPORT

DRUG	PEDIATRIC MIX (ALL IN D5W)	IV DOSE RANGE & COMMENTS
DOPAMINE 40 MG/CC	6 MG x WT (KG) IN 100 CC 1 CC/HR = 1 MCG/KG/MIN	1.0 - 20 MCG/KG/MIN
DOBUTAMINE 25 MG/CC	6 MG x WT (KG) IN 100 CC 1 CC/HR = 1 MCG/KG/MIN	1.0 - 20 MCG/KG/MIN
ISOPROTERENOL 0.2 MG/CC (1:5000)	0.6 MG x WT (KG) IN 100 CC 1 CC/HR = 0.1 MCG/KG/MIN	0.1 - 1.0 MCG/KG/MIN HR NOT TO EXCEED 180
EPINEPHRINE / NOREPI. 1.0 MG/CC (1:1000)	0.6 MG x WT (KG) IN 100 CC 1 CC/HR = 0.1 MCG/KG/MIN	0.1 - 1.0 MCG/KG/MIN HR NOT TO EXCEED 180
NITROPRUSSIDE 10 MG/CC	6 MG x WT (KG) IN 100 CC 1 CC/HR = 1 MCG/KG/MIN	0.5 - 4.0 MCG/KG/MIN PHOTOSENSITIVE, COVER BOTTLE
LIDOCAINE 2% = 20 MG/CC	60 MG x WT (KG) IN 100 CC 2 CC/HR = 20 MCG/KG/MIN	20 - 50 MCG/KG/MIN BOLUS FIRST (1 MG/KG) MAY NEED TO REDUCE DOSE IF HEPATIC DYSFUNCTION. FOLLOW LEVELS.
PGE1 500 MCG/CC	0.6 MG x WT (KG) IN 100 CC 1 CC/HR = 0.1 MCG/KG/MIN	.05-0.2 MCG/KG/MIN HYPOTENSION, APNEA

ALTERNATE METHODS TO MIX DRIPS BASED ON WEIGHT IN KILOGRAMS:
((6 X KG) X DESIRED DOSE (MCG/KG/MIN))/ INFUSION RATE (CC/HR) =
MG PER 100 CC OF SOLUTION

APPROACH TO ACID BASE PROBLEMS

Blood Gas Analysis--Rules To Live By:

1) Is there ACIDEMIA or ALKALEMIA

a. Alkalemia and Acidemia refer only to the net pH of blood, and do not describe the process that led to the alteration of the pH.

Acidemic pH < 7.35

Alkalemia pH >7.45

b. Whatever side of 7.4 the pH is on, the process that caused it to shift to that side is the primary abnormality. The body does not fully compensate for primary acid base disorders. After this step one should search for the underlying primary disorder. What processes, metabolic or respiratory brought the pH to either side of 7.4.

2) Identify abnormalities in PCO2.

a. Identify whether the PCO2 is normal, inappropriate, and whether there is a respiratory acidosis or alkalosis (i.e. the CO2 could be in the normal range but is the patient tachypneic and is CO2 inappropriately high).

For acute increases in PCO2 of 10 mm Hg there should be a rise of 0.08 in the pH.

For acute decreases in PCO2 of 10 mm Hg there should be a fall of 0.08 in the pH.

b. Winter's rule states that the last 2 digits of pH greater than 7 predicts the PCO2 in a compensatory respiratory alkalosis in a patient with a primary acidosis. So if a patient has a metabolic acidosis, say pH 7.25, one should expect a PCO2 of 25. If the CO2 is higher, than the patient is not having an appropriate compensation, suggesting respiratory dysfunction.

c. At this point one should decide whether the change in pH could be explained by your PCO2. If not, consider ordering a set of electrolytes to review the causes for a metabolic acidosis. One would evaluate the bicarbonate level (on the ABG the Bicarbonate level is a calculated value not a true value).

In addition, evaluating the anion gap can direct you toward the cause for an acidosis.

3) If pH change is appropriate for PCO2 change go to the evaluation of oxygenation.

4)Identify abnormalities in bicarbonate.

a. Normal bicarbonate is 22-24

If the value is lower than 22, there is a metabolic acidosis.

If the value is higher than 24, then there is a metabolic alkalosis.

b. Metabolic Acidosis is commonly divided into anion gap and nonanion gap acidosis. If the Bicarbonate value is less than 22 one must calculate the anion gap; if the anion gap is greater than 17, it suggests anionic gap acidosis.

Normal Anion Gap 12 +/- 2. If AG > 17, a metabolic acidosis is probably present. Anion Gap acidosis differential is remembered by stating this antonym: **CAT MUDPILES**. **C** for cyanide or carbon monoxide, **A** for alcohol, **T** for toluene, **M** for methanol, **U** for uremia, **D** for DKA, **P** for paraldehyde, **I** for iron and isoniazid, **L** for lactic acidosis, **E** for ethylene glycol, and **S** for strycnine. The body does not generate a large anion gap to compensate even for a chronic alkalosis. For each decrease in serum bicarbonate there should be an equal rise in the AG.

c. Calculate the excess anion gap. The excess anion gap = total anion gap minus the normal anion gap (12 mmol per liter). Add the excess anion gap to the measured bicarbonate concentration; if the sum is greater than a normal serum bicarbonate (> 30 mmol per liter) there is an underlying metabolic alkalosis; if the sum is less than a normal bicarbonate (< 23) there is an underlying nonanion gap metabolic

acidosis (.i.e. 1 mmol of bicarbonate titrates 1 mmol of acid titrates which forms Na acid which is a unmeasured cation which increases the anion gap). If the value is not less than 23 or greater than 30 the no additional process is going on.

d. If there is no anion gap but the bicarbonate is low, a urinary anion gap can help differentiate renal vs. gut as loss of bicarbonate. (Na + K)- Cl-correlates with ammonium excretion. As ammonium excretion increases the urinary gap narrows. So with diarrhea (gap is negative), ammonium excretion unimpaired and increases with acidosis. With RTA (gap is positive) the gap widens since they have lost the ability to absorb bicarb and form ammonium.

5) Assessment of Oxygenation

PaO2 of < 60 and Saturation < 90 is hypoxia.

A-a gradient is important to evaluate. One can estimate the Alveolar O2 by multiplying 5 X the FiO2.

Alveolar O2 = (760-47)FIO2 - PCO2/.8 or **5 X FIO2**.

If the predicted PaO2 is much different than the true PaO2 than further evaluation for causes should be done. The main lesson here is to understand that by looking at the ABG one can assess if there is an inappropriate FiO2 requirement. Normally the A-a is < 60 - 100. If it is larger the ability of the patient to oxygenate is compromised.

6) Venous Blood Gas

In patients with normal cardiac output, central venous pH is lower than arterial by an average of 0.03 units, with venous PCO2 being higher by about 6 mm Hg. With severe circulatory failure, the pH differs by 0.1 unit with pCO2 difference of 24. With a cardiopulmonary arrest, a venous blood gas could have a pH difference of 0.35 and a CO2 difference of 56 when compared to an arterial blood gas.

ANAPHYLAXIS

I. Definition/Pathophysiology

A systemic reaction (usually life-threatening) that occurs secondary to an IgE mediated antigen induced reaction (allergen) or exposure to mast cell degranulating agents (anaphylactoid). Both reactions cause mediator release (histamine, leukotrienes, PAF, etc.) which produce the symptoms. While there is often a history of prior exposure to a given antigen, in the non-IgE mediated (anaphylactoid) reactions, symptoms may occur during the first exposure.

II. Clinical Course

A. Symptoms usually occur within seconds to 60 min. of Ag exposure.

B. Variable: Initial symptoms may be mild or life threatening. Generally, the earlier the onset, the more severe the reaction.

C. Symptoms - cutaneous (urticaria/angioedema, pruritus), respiratory (bronchospasm, stridor, pulmonary edema, laryngeal edema), rhinitis, cardiovascular (hypotension, arrhythmias, myocardial ischemia, vaso-dilation, flushing), gastrointestinal (nausea, emesis, diarrhea, pain), asymmetric swelling of a limb or perioral area.

III. Most Common Etiologic Agents

A. Antibiotics (for instance penicillin, although any could be involved)

B. Insect (hymenoptera) stings

C. Foods (nuts, eggs, seafood)

D. Immunotherapy

E. Non-IgE (Anaphylactoid) mediated mast cell degranulation:

1. Morphine

2. Codeine

3. Polymyxins

4. Radiocontrast dye

IV. Risk Factors:

A. Personal history of previous allergic reaction.

B. Positive skin test.

C. Sick patient on multiple medications.

V. Therapy

A. ABC's

1. Establish airway if significant compromise.

2. May need intubation or trach. if no relief with epi.

3. Oxygen if respiratory distress or hypotension.

B. Stop antigen administration - if insect bite or allergy shot, isolate antigen site with tourniquets and inject 0.01 cc/kg epi. (1:1000) SQ into site after tourniquet applied. **Flick off (do not squeeze) any stinger present.**

C. Epinephrine:

1. Mainstay of treatment

2. SQ or IM 0.01 cc/kg of 1:1000 sol'n, max 0.3 cc, may repeat

3. Rarely IV 1:10,000 by drip and titrate to achieve response, begin at drip of 0.1 mcg/kg/min (only in refractory hypotension requiring CPR).

D. Immediate IV placement with IVF (LR/NS, bolus 20 cc/kg as needed for shock).

E. Continue to observe for 24 hrs, as symptoms may recur.

1. Subjective: SOB, anxiety.

2. Objective: stridor, retractions, wheezing, cyanosis, pallor.

3. BP: q 5-10 min initially, then q 1 hr.

4. Continuous EKG monitor or A-line as needed.

F. Other drugs as needed (**NOT** a substitute for epi.).

1. H1 Antihistamine - Benadryl 0.5-1.0 mg/kg po or slow IV push.

2. Steroids - 1-2 mg/kg methylprednisolone to prevent late phase response.

3. Cimetidine IV 5-10 mg/kg given over 5 min - given in association with H1 antihistamines may reverse profound hypotension unresponsive to fluids/pressors (this is controversial).

4. Glucagon may be effective in reversing hypotension in rare cases, especially if beta-blockade is present. (Dose: < 10 kg: 0.1mg/kg IM, > 10 kg: 1 mg/dose IM).

G. For cardiorespiratory arrest, continue with BCLS/ACLS algorithms.

VI. Differential diagnosis includes:

A. Insulin reaction

B. Vasovagal syncope

C. Arrhythmias

D. Hereditary angioedema

BITES AND STINGS

I. Mechanisms of Injury

A. Children may be at increased risk for severe reactions to stings and envenomations due to an increased dose of venom per kg.

B. There are several mechanisms for the toxic effects of bites and stings:

1. Immunologic, including IgE mediated anaphylaxis and serum sickness (Ab-Ag complex).

2. Hemotoxic

3. Neurotoxic

C. Most venoms are toxic due to a combination of the above factors

II. Purveyors of Venom

A. Snakes

Several different types of snakes must be differentiated due to the varying effects of their venoms.

1. Pit Vipers

a. These account for the majority of significant envenomations in the United States. Included are

(1). Rattlesnakes (60%)

(2). Water moccasins:

a. Copperheads (30%)

b. Cottonmouths (10%)

b. Many rattlesnake bites are provoked and thus involve the upper extremities

c. Pit viper venom is voluntarily injected by venom gland contraction. It generally contains digestive enzymes. Depending on the species there may be other components. The Mojave rattlesnake makes Mojave "toxin", a neurotoxin which may lead to paralysis and respiratory arrest. There may be varying hemotoxins which profoundly decrease platelet and clotting factors.

d. Treatment

(1). First Aid Management

- Initiate BLS as necessary (ABCs)

- Move the patient to a health care facility as rapidly as possible

- Minimize movement of an affected extremity and keep the extremity below the level of the heart

- Avoid ice, aspirin (coagulopathies possible), alcohol or sedatives

- Tourniquets not universally recommended; although constriction band in experienced hands may be useful when incision and suction are indicated or a long transport anticipated.

(2). Incision and Suction

These should be considered only if:

- Patient is more than one hour from a medical facility

- You have carefully considered the anatomy of the underlying area

- It has been less than 5-10 minutes since envenomation

The preferred method is suction with an extractor. If unavailable, use of the mouth is acceptable. The only incisions to be made are extensions of a fang mark, and no more than 1 cm long and 0.5 cm deep. DO NOT MAKE AN X SHAPED CROSS INCISION, and always keep in mind underlying structures.

(3). Tourniquets

A varying portion of venom, esp. rattlesnake, may be absorbed via the lymphatic system. Given this, if a medical facility is not nearby (W/IN 1 HOUR), a LIGHT, wide constricting band may be placed around an extremity. Use of a tourniquet is a controversial topic. A BP cuff at 15-20 mmHg is adequate. Otherwise the band should be wide and two fingers able to pass freely under it. The band should be tight enough to occlude lymphatic flow but loose enough to palpate pulses distal to the bite. If swelling occurs, place a second tourniquet above the first one before removal of the first band. Before this is done it is recommended that 2 IV’s are in place, fluid resuscitation is underway, and the antitoxin is given. At the trunk remove the last band. This will prevent a sudden release of venom into the systemic circulation.

(4). Hospital Management

- History of evenomation, PMH, previous snakebites and treatment

- The initial presentation of a snakebite victim may correlate poorly with subsequent outcome. Following a major envenomation a patient may develop profound circulatory shock. Hypotension is a late manifestation of this. ASSESS VITAL SIGNS. Observe for tachycardia, tachypnea, poor perfusion, alteration in consciousness. Circumferential measurements of the bitten extremity should be performed every 15 - 20 minutes to serve as an indication of the progression of the evenomation. Make **two** measurements - at the envenomation site and 5 - 10 cm above the lesion.

- When unknown, assume any snake may be involved in a snakebite case. Do not trust reports of amateur herpetologists. It is doubtful he/she will know the difference between a Mojave rattlesnake and a rattlesnake with less potent venom.

- Labs

(a). PT, PTT, Fibrinogen, Fibrin Split Products

(b). CBC, Platelets, blood for type and cross

(c). Lytes, BUN, Cr, CPK, glucose

(d). UA

(e). Skin testing for hypersensitivity to horse serum should be done only if it is known that antivenin will be administered

e. Staging

In approximately 10-20% of known rattlesnake strikes, no venom is released and only a fang mark will be present.

A staging system helps to provide a general scheme for intervention. Remember though, that what appears to be a minimal envenomation may develop severe manifestations.

(1). No envenomation or:

(2). Mild Envenomation:

A fang mark is surrounded by edema and tissue necrosis, but these are confined to the immediate segment of the extremity involved. No systemic effects are present.

(3). Moderate Envenomation:

A fang mark is present and surrounded by tissue breakdown including edema, bullae, and/or hemorrhagic blebs extending beyond the immediate area of the bite but not involving the entire extremity. No life threatening systemic effects are seen.

(4). Severe Envenomation:

A fang mark is present with obvious tissue breakdown, including edema, bullae, and/or hemorrhagic blebs. Tissue ecchymoses involving an entire extremity or systemic effects such as tachycardia, hypotension, poor perfusion, alteration in mentation or lab data showing coagulopathy or muscle breakdown are present.

(5). **Bites on the thorax or head/neck are all assumed to be severe. Early intubation should be considered for these patients as respiratory compromise is frequent.**

f. Basic Therapy

(1). Clean the wound area and administer tetanus prophylaxis

(2). Do q 15 minute measurements (circumferentials) of the involved extremity

(3). Use acetaminophen or mild opiates (codeine) for relief of pain

(4). All children, severity of bite aside, will be observed for 24 hours

(5). Establish rapid IV access and support shock as necessary with fluid and pressors

(6). Consider antivenin for moderate to severe envenomations (antivenin is most effective within the first 12 hours after the bite, antivenin is not indicated after 24 hours). Prior to initiation of antivenin therapy, volume expand with a 20 cc/kg fluid bolus, unless contraindicated by preexisting medical condition

(7). Antibiotics are controversial but may be appropriate esp. in dirty wounds.

g. Use of Antivenin

(1). For North American pit vipers (Crotalidae), the only approved antivenin is the polyvalent Crotalidae antivenin (Wyeth). It is active against all pit vipers, as well as

the Bushmaster, Fer de Lance and Mamushi.

(2). This is a horse derived product and has significant risk for anaphylaxis and serum sickness. Obtain consent prior to use. The anaphylactic reactions are not all IgE mediated and may occur on first exposure. Administer antivenin slowly.

(3). Prior to use attempt to ensure cardiovascular stability by correcting hypovolemia and using pressors as needed. During administration, continually monitor for changes in CV status. **Do not shake the vials** when mixed with fluid; stir them only by rocking in your hands - gently back and forth.

(4). After the decision is made to use antivenin, skin test, using an intradermal injection of antivenin. This is done by injecting 0.02 cc of saline diluted horse serum intradermally with observation for at least 20 - 30 minutes for the development of erythema, hives, pruritis or other adverse effects. If positive, the patient should be pretreated with benadryl and steroids prior to administration. A negative skin test does not mean no risk for anaphylaxis exists. Never skin test mild or non-venomous bites "just in case we need to use antivenin." You may sensitize the patient to horse proteins.

(5). Administration of antivenin is done in five vial aliquot increments. Moderate bites require an average of 10 vials of antivenin. In severe envenomations, as many as thirty vials of antivenin have been administered to halt symptom progression. For children, many experts recommend that the dose of antivenin be increased by 50 % because of a higher ratio of venom to body mass.

(6). Reconstituted antivenin is diluted with NS or D5W in a 1:4 to 1:5 dilution. Infuse antivenin slowly and if tolerated after 20 minutes increase the infusion to 1-5 vials/hr. Goal is to give entire antivenin dose within the first 4-6 hours to bind the venom and prevent further toxicity.5 vials are place in 125 ml for children less than 10 kg and in a bag of 250 ml for children over 10 kg. Infuse at 1 ml per hour initially, then increase to deliver 1 vial per hour.

(7). Infusion of one to two vials per hour is appropriate to minimize the side effects seen at higher infusion rates. Mild reactions (pruritus, urticaria etc.) can be treated with benadryl. With serious reactions consider D/Cing antivenin and treat with epinephrine SQ (0.01 cc/kg 1:1000 max 0.3-0.5 cc)

(8). After a five vial infusion, assess for changes in swelling, necrosis or systemic signs. If unimproved or

worsening administer five more vials.

(9). Serum sickness is almost universal in children and may occur up to 10 days post infusion. Parents should be informed of this likely occurrence.

h. Other Therapy

(1). If required use FFP for volume replacement

(2). It is unusual for active bleeding to occur

(3). Have a low threshold for intubation of a patient with a symptomatic neck bite

i. Controversies

(1). Wide fasciotomies of swollen extremities are not beneficial. Compartmental pressures may be measured in concerning situations, but it is rare that an increase occurs which requires fasciotomy. Consult orthopedics.

(2). Steroids are useful only in managing anaphylaxis or hypersensitivity to antivenin.

2. Coral Snakes

a. Are in the same family as the cobra and the mamba. The red on yellow are venomous (red on yellow - kill a fellow) as opposed to the non-venomous red on black (red on black - venom lack). Significant species are the Eastern and Western coral snakes.

b. Symptomatic bites are largely neurologic in their effect. Local hypesthesia may occur with changes in consciousness, muscle weakness, fasciculations and paralysis. Respiratory failure may ensue.

c. Lab work is not a reliable indicator of venom effects.

d. It is uncertain if venom effects can be reversed after onset of symptoms. Therefore, early treatment with antivenin (Micrurus antivenin, by Wyeth) is recommended (3-5 vials repeat as needed for signs of toxicity). Superficial scratches may not mean envenomation. A significant envenomation is usually heralded by local hypesthesia. A clear bite mark breaking the skin is serious.

e. Use same rules for antivenin administration as above.

3. Nonvenomous Snakes

a. May cause local reaction due to digestive enzymes.

4. Non-indigenous Snakes

a. A wide variety of exotic snakes are illegally imported, many poisonous.

b. The Arizona Poison Center (602-626-6016) keeps general inventory of antivenins maintained in zoos and hospitals throughout the country. The San Diego Zoo also has many antivenins available (619-231-1515). The San Antonio Zoo stocks some exotic snake antivenins (734-7183). **WHMC IV pharmacy** carries coral snake and pit viper antivenins and BAMC carries others (916-6638)

B. Scorpions

1. In general only the southwestern desert scorpion (Centruoides) produces mortality. The venom is a neurotoxin and primarily affects the autonomic and skeletal neuromuscular system. Classically envenomation leaves no significant local effect, but produces a tingling sensation which may progress up the extremity. Young children may develop fasciculations, agitation, opisthotonus or seizures.

2. Therapy

a. Mild envenomations may be treated with cool compresses and mild analgesics.

b. Severe Envenomation

(1). Basic supportive care: Increased parasympathetic tone may give increased secretions and loss of bulbar tone. Follow for airway compromise. Severe tachyarrhythmias may occur. These are best treated with propranolol slow IV 0.01 - 0.1 mg/kg over 5-10 minutes. Intubation may become necessary.

(2). Establish a firm diagnosis and consider sepsis/meningitis prior to a primary diagnosis of scorpion sting, especially with an unverifiable story.

(3). If symptoms are severe, an unlicensed antivenin is available in Arizona for the Centruoides species. Use the Arizona poison control number.

C. Hymenoptera Stings

The toxicity of ant, bee, and wasp stings is related primarily to their ability to produce anaphylactic and hypersensitivity reactions, Up to 80% of fatalities occur in individuals with no prior history of sensitivity. See anaphylaxis chapter. Fire ants deserve special consideration as its venom differs from venom of other hymenoptera. Fire ant venom has a direct toxic effect on most all membranes. The fire ant bites with its jaws then inflicts multiple stings which leave an immediate wheal and flare which may develop into the classic

white-cloudy pustule, surrounded by painful erythema. Systemic reactions can occur as with other hymenoptera.

1. Therapy

a. Local - Cool compresses and soap and water cleansing
The pustule of the fire ant sting is usually sterile, so there is no need for antibiotic creams or ointments.

b. Systemic - Benadryl 5 mg/kg/day divided q 6 hours prn itching or swelling.

c. Treat anaphylaxis as outlined in anaphylaxis chapter.

D. Spiders

All spiders possess venom. Highly toxic venom is limited to a few species. In North American two major patterns of bites occur, one associated with the black widow, the other with necrotizing spider bites (primarily associated with the brown recluse). Both live in Texas

1. Black Widow Spider

a. The female of the species delivers venom. It is a mixture of polypeptides that includes a neurotoxin that stimulates myoneural junctions, nerves and nerve endings.

b. Favorite locations are moist, dark areas away from sunlight

c. Signs and Symptoms

- Local pain with progression up an extremity.

- Increased motor tone with muscle spasm that may generalize. This may be very prominent over the abdomen and mimic an acute abdomen. Nausea and vomiting are often reported in children. Usually normal bowel sounds and x-rays.

- Diaphoresis, tachycardia, and periorbital edema.

- 4-5% mortality rate with death resulting from cardiovascular collapse. The mortality rate in young children may be as high as 50 %.

- Labs may show an increased CPK, also leukocytosis and hyperglycemia due to increased sympathetic tone.

d. Therapy

- Majority of cases are mild and resolve over 24 hours with mild analgesia.

- Young children are more seriously affected. Thus use of antivenin should be instituted in any child presenting with severe pain and muscle rigidity after a spider bite.

- Assess vitals (ABCs)

- Establish IV access

- Rule out other infectious and surgical conditions

- Severe muscle spasm may be treated, in an ICU setting, with diazepam (0.1 mg/kg IV). Calcium gluconate 10% soln. (0.1 cc/kg/dose IV slowly) can also be given for control of leg and abdominal cramps. Both of these agents are variably effective and short-lived.

- Impaired diaphragmatic excursion due to muscle spasm may necessitate intubation and ventilation.

- For severe envenomations a horse serum antivenin is available (from Merck, Sharp and Dome). Method of preparation, administration and cautions are similar to those for pit viper antivenin. The only difference is use of one vial in preparing the initial antivenin drip with additional vials added in one vial increments. One vial for children < 40 kg. The use of antivenin is controversial.

- The venom produces smooth muscle contraction, and the uterus is very sensitive. The result of a bite in a pregnant women may be spontaneous abortion. Antivenin should be administered early in the pregnant patient.

2. Necrotizing Spiders

a. The brown recluse (Loxosceles reclusa) is most common in the Southeast and Midwest U.S. There are several species of Loxosceles in the Southwest.

b. The venom of reclusa is extremely potent on a mg/kg basis. It contains multiple proteolytic enzymes and is cytotoxic.

c. Classically patients present with a target like skin lesion with a blackened necrotic center. Typically the patient is unaware an envenomation has occurred until an indurated area with a small vesiculated center develops. This ruptures and becomes a dark, necrotic area. This area may increase in size with continued necrosis for periods up to weeks. Then contraction of the wound with involution occurs.

- Systemic reactions are more common in young children and are usually seen 24-48 hours after the bite and include

fever, chills, malaise, nausea, vomiting, weakness or pain, rash, and renal failure.

d. There are case reports of hemolysis and DIC like syndromes associated with these bites. It is prudent to screen for these (CBC, PT/PTT, fibrinogen, and urine for hemoglobin).

e. Therapy

- Good local wound care and debridement

- Tetanus prophylaxis

- Consider treating intravascular hemolysis with steroids (controversial!)

- There are many suggested therapies of unproven and questionable value, these include local injection of phentolamine, dapsone and steroids. Wide excision seems unnecessary in view of the regression of these bites naturally into a smaller, final cosmetic lesion. **However**, excision and debridement of necrotic areas > 2 cm in diameter with antibiotic coverage is appropriate if the wound is being seen in the first 48 hours. For necrotic areas > 2.5 cm in diameter skin grafting may be necessary. Early involvement of our plastic surgery and pediatric surgery consultants is advised.

- Work is presently being done on the development of an antivenin.

CARDIOLOGY PROBLEMS

I. General approach to dysrhythmias:

A. First, rule out artifacts:

1. Flat line can be due to a loose or detached lead. If you are using the defibrillator monitor the “paddles” lead will be flat and may be the default lead when the defibrillator is turned on. Check the defibrillator set-up.

2. Tall T-waves may be perceived by the monitor as extra R-waves and be double counted. Run a strip and count for yourself.

3. Leads perpendicular to the P-wave axis may make P-waves appear absent. Check more than one lead to be certain.

4. Rhythmic movement artifacts (seizures, hiccoughs in neonates) may mimic dysrhythmias.

B. If hemodynamically stable obtain 12-lead ECG with rhythm strip prior to intervention.

C. Record rhythm strip during intervention.

D. As soon as feasible, ***repeat*** 12-lead ECG with rhythm strip after successful intervention.

II. Abnormal Rhythms: (**Cardiology should be consulted.**)

A. Principles of therapy:
- treat emergently only if cardiac output compromised (an unstable patient)or if rhythm has the potential to degenerate into a lethal (collapse) rhythm such as with V. fib.
- normal heart rates (HR) are given below.

Heart Rates in Normal Children

Age	Awake rate	Mean	Sleeping rate
Newborn to 3 mos.	85 - 205	140	80 - 160
3 mos. to 2 yr.	100 - 190	130	75 - 160
2 - 10 yr.	60 - 140	80	60 - 90
> 10 yr.	60 - 100	75	50 - 90

from : Gillette PC et al. Dysrhythmias. In Adams FH, Emmanouilides GC, Riemenschneider TA, eds. Moss’ Heart Disease in Infants, Children and Adolescents. 4th ed. Baltimore, Md: Williams & Wilkins; 1989: 725-741

B. Tachyarrhythmias:

1. Sinus tachycardia (ST): - therapy is directed to underlying cause: anxiety, fever, volume depletion, pain, stimulant medications, anemia, CHF, thyrotoxicosis.

a. Beat-to-Beat heart rate variability is common.

b. Acceleration and deceleration of heart rate is typical.

2. Supraventricular Tachycardia (SVT):

a. HR depends on patient age, usually 220 or greater (up to 300 bpm). Etiologies: idiopathic in approximately 50%, Wolff-Parkinson-White syndrome (WPW), congenital heart defects (Ebstein's anomaly, single ventricle, L-TGA), associated with hydrops fetalis, sepsis, trauma, central lines.

b. HR very regular, rarely associated with A-V block.

c. P-waves may not be visible at high rates.

d. QRS duration is normal in 90% of cases. Wide QRS may be indistinguishable from V-Tach.

e. May see ischemic ST-T changes in SVT of long duration.

f. Long-lasting SVT may cause a cardiomyopathy and/or congestive heart failure which may then lead to cardiomegaly; a CXR may be useful.

g. ECG criteria: ST vs. SVT.

(1). ST usually < 220 bpm, SVT > 220 bpm.

(2). In ST there is beat to beat variation in rate. No HR variation in SVT.

(3). Both onset and cessation of SVT are abrupt.

h. Therapy: ***depends on cardiac stability***. If the patient has a good BP, and cap. refill, is responsive and has no signs of CHF then he/she is probably stable. If the patient has weak or absent pulses, poor cap. refill and BP, is lethargic or unresponsive, has HSM, edema or rales then he/she is probably unstable.

(1). **Synchronized cardioversion** (0.5-1.0 joules/kg) for cardiovascular instability; may use adenosine if IV present and adenosine readily available (see below).

(2). If persists, repeat synchronized cardioversion at 2.0 joules/kg; SVT normally converts easily with synchronized cardioversion.

(3). If no success with cardioversion, consider sinus tachycardia, atrial fibrillation or atrial flutter. Review the ECG strips performed while you were attempting to cardiovert.

(4). In non-emergent cases attempt vagal maneuvers (stimulate gag reflex, ice bag to face (forehead to chin, ear to ear) Valsalva, carotid massage in **older** children. **Never press on the eyeballs.**

(5). Adenosine 0.1 mg/kg rapid IV push followed immediately with rapid fluid flush. Expect transient asystole at conversion (adenosine T 1/2 is seconds). May need higher dose if patient on theophylline. If first dose of 0.1 mg/kg has no effect, double the adenosine dose (maximum single dose is 12 mg).

(6). Consider digitalization. (see section at end of this chapter for dig. doses, side effects etc.) If WPW present, consult cardiology first. (dig + WPW deaths reported). Diagnose WPW when isoelectric PR segment is absent (i.e. short PR interval) and QRS is wide. May also see delta wave.

(7). Verapamil 0.1 mg/kg IV over 3-5 min. **only if patient stable and > 1 year old.** Contraindications : CHF present, age < 1 year, concomitant Beta-blocker therapy, myocardial depression, or if patient has a bypass tract. Always have calcium chloride available as antidote!

(8). Procainamide 2 - 6 mg/kg/dose IV over 5 minutes. Maintenance is 20 - 80 **mcg**/kg/min IV drip.

3. Ventricular Tachycardia (V-Tach):

a. Rate may vary from normal to 400 bpm (usually 150-250).

b. Majority of cases have underlying structural heart disease or a prolonged QT interval.

c. May be caused by hypoxia, acidosis, electrolyte imbalance, drugs (esp. methylxanthines, amphetamines, tricyclics, digitalis/ digoxin), myocarditis, poisons, long QT syndrome, or post surgery.

d. ECG criteria:

(1). Rate at least 120 BPM and regular.

(2). Wide QRS (> 0.08 sec).

(3). P-waves not seen, or dissociated

(4). T-waves opposite in polarity to QRS

(5). May resemble SVT with aberrant conduction; however, wide QRS tachycardia in children is V-Tach until proven otherwise!

e. Therapy:

(1). **Synchronized cardioversion** with cardiovascular instability 0.5 - 1.0 J/kg. This is done for unstable patients **with** a pulse. If patient has V-tach. with**out** a pulse then treatment is similar to V-fib. and uses defibrillation instead - **see algorithms !!**

(2). Lidocaine bolus (1.0 mg/kg) results in higher success rates of cardioversion.

(3). Lidocaine infusion (20 - 50 mcg/kg/min) will help maintain sinus rhythm after conversion.

(4). If cardioversion needs to be repeated use 2.0 J/kg

(5). If lidocaine is unsuccessful, use bretylium 5 mg/kg slow IV push and repeat cardioversion.

4. Atrial Fibrillation/ Flutter - mortality as high as 25% in infants

a. Flutter is defined by the morphology

(1). Flutter waves (sawtooth pattern) are seen in the inferior and right precordial leads. Rates of 300 or so are common but may be as high as 500 BPM.

(2). AV conduction is variable - it usually is 1:1 in infants

(3). Causes: In infants the heart is usually structurally normal. Flutter is also associated with congenital heart disease though, with 75% of episodes occurring post-op cardiothoracic surgery, especially after Fontans, Mustards, or Sennings. Also occurs from digitalis toxicity, myocarditis, and hypokalemia.

b. Fibrillation is characterized by an irregularly irregular rate and rhythm with variable “p” wave morphology. Ventricular rates are often > 200 BPM and atrial rates > 400 BPM.

(1). Causes: idiopathic, stimulants may precipitate episodes, congenital heart disease (Ebstein’s anomaly, mitral stenosis, tricuspid atresia etc.), rheumatic heart disease, hyperthyroidism, cardiomyopathies, myocarditis

c. Treatment of Atrial Fibrillation or Flutter:

(1). Synchronized cardioversion 0.5 - 1.0 J/kg.

(2). Transesophageal overdrive pacing at 125% of flutter rate if available.

(3). If the above maneuvers fail: **Make sure you do not have SVT.** Digitalize with 1/2 the digitalizing dose (see digoxin section at end of this chapter). 8 hours later give 1/4 the digitalizing dose, and 8 hours after that give the final 1/4 digitalizing dose. Digitalis slows the ventricular rate. Do not perform if patient is already on dig.

(4). If you have given the first dose of dig. and the patient has not converted in 30 minutes then load with procainamide: If the patient is < 1 year old: give 7 mg/kg IV followed by 3.5 mg/kg IV q 4 hours. If the patient is > 1 year old: give 15 mg/kg IV followed by 7 mg/kg IV q 4 hours.

(5). If the above steps fail repeat the cardioversion.

(6). If still not converted use IV amiodarone, 1 mg/kg/dose q 10 min. X 10. May repeat q 12 hours. Monitor the QTc on the ECG. If the QTc is > 0.53 seconds decrease the dose.

(7). Continue all drugs used for cardioversion, monitor procainamide and NAPA (n-acetyl-procainamide) levels, use continuous ECG monitoring.

C. Bradyarrhythmias:

1. ECG:

a. Slow rate is all that matters.

b. P-waves may be present or absent.

c. QRS is narrow or wide.

d. In context of CPR, the exact rhythm is not important.

e. Etiologies: normal athletes, vagal stimulation, increased ICP, hypothyroidism, hypothermia, hypoxia, hyperkalemia, digitalis, beta-blockers, acidosis, heart block, seizures, rapid calcium infusions, tricyclics.

2. Heart block:

a. Congenital, complete AV block:

(1). Patients are usually asymptomatic.

(2). QRS is usually narrow.

(3). Treatment is not urgent and often not necessary.

b. Acquired heart block:

(1). Notify the cardiologist.

(2). Most commonly occurs postoperatively.

(3). QRS is usually wide.

(4). May cause significant compromise of cardiac output.

(5). If patient is unstable, refer to bradycardia algorithm as isoproterenol infusions and cardiac pacing may be necessary.

(6). In older patients, transthoracic external pacemakers are available for temporary use in an ICU setting.

3. Therapy:

a. Ensure adequate ventilation.

b. Chest compressions.

c. Epi and other meds as per algorithms. Atropine.

d. Atropine.

D. Absent or disorganized rhythms:

1. Asystole:

a. Diagnosed by absence of auscultated or palpated pulse, straight line ECG, absent spontaneous respirations, and poor perfusion.

b. Therapy: appropriate ACLS steps as per algorithm.

2. Ventricular Fibrillation (V-fib):

a. Uncommon terminal event in pediatrics, very rare in infants. Etiologies: postoperative, hypoxia, hyperkalemia, digitalis or quinidine toxicity, myocarditis, infarctions, catecholamines, anesthetics.

b. No identifiable P, QRS, or T-waves.

c. Therapy: **Defibrillation** and treatment per ACLS algorithm.

3. Electromechanical Dissociation: (pulseless electrical activity)

a. Organized electrical activity on ECG, but ineffective or

absent cardiac contraction, absent peripheral pulses.

b. Causes include severe acidosis, pneumothorax, pulmonary embolism, pneumopericardium, hypovolemia, hypoxemia, cardiac tamponade, hyperkalemia, tricyclic antidepressants, Beta-blockers, Calcium channel blockers, and hypothermia.

c. Therapy: see algorithm

III. Defibrillation and Cardioversion:

A. Paddle size:

1. 4.5 cm for infants (usually < 1 yr, or < 10 kg)

2. 8 or 13 cm for older children (usually > 1 yr, or > 10 kg)

3. Largest paddle size possible is good rule of thumb.

B. Electrode interface: use electrode gel or redux cream, **NOT** ultrasound gel. Saline pads drip and cause short circuits.

C. Position: one on upper right chest below clavicle, other to left of the nipple in anterior axillary line. Try to "cup" the heart with the paddles. Reverse this position in cases of dextrocardia, i.e.. upper left chest and right anterior axillary line.

IV. Management of "Tet Spells" or Paroxysmal Hyperpnea in Cyanotic Heart Disease

A. Any patient with Tetralogy of Fallot may have "Tet spells". They may present with cyanosis, hyperpnea, restlessness, and agitation. On exam the pulmonic stenosis/ ventricular septal defect murmur may lessen or disappear as less blood flows to the lungs. However, other forms of cyanotic heart disease may have these spells as well so they are also known as cyanotic spells. Often these spells are initiated in otherwise healthy children with: fever, after defecation, warm baths, after crying, or after an increase in activity as these actions decrease systemic vascular resistance (SVR). They may also occur if pulmonary vascular resistance (PVR) is increased such as with a pulmonary infection.

B. **"Tet spells" may be caused by anything that reduces SVR or that increases PVR.**

1. SVR may be decreased by: dehydration, sepsis, sedatives or pain killers, loss of vascular tone, lack of cardiac contractility, anemia, anaphylaxis, or any shock state.

2. PVR may be increased by: viral or bacterial pneumonias, bronchiolitis, URI's, pneumothoraces, hypoxemia, and acidosis. Also, worsening pulmonic stenosis and right ventricular hypertrophy may

lead to diminished blood flow to the lungs resulting in pulmonary hypertension.

C. Tetralogy of Fallot has 4 associated findings:

1. Right ventricular hypertrophy (RVH)

2. Pulmonic stenosis (PS)

3. An overriding aorta

4. Large ventricular septal defect (VSD)

D. Physiology:

1. Spells are caused by shunting of blood away from the lungs to the body. Usually this is primarily an intracardiac shunt through the VSD from right to left. This causes deoxygenated blood to flow to the body causing hypoxemia, a resulting acidosis, and V/Q mismatch which may then aggravate the condition further by contributing to or causing pulmonary hypertension. (Usually though “Tet spells” are not classically thought of as having reactive pulmonary hypertension.)

2. With little blood flowing to the lungs the systemic venous return increases. The increased systemic venous return causes even more deoxygenated blood to flow right to left across the VSD and out to the body which further aggravates the hypoxemia and acidosis.

3. The patient gets anxious which further aggravates the condition and leads to a vicious cycle. **These spells are life threatening and a true emergency !** We attempt to break this cycle in 3 ways.

(1.) Re-establishing blood flow to the pulmonary bed. We do this by decreasing pulmonary hypertension if present, decreasing PVR by relaxing the infundibulum, and increasing SVR which forces more blood into the lungs by reducing the right to left shunt.

(2.) Decreasing systemic venous return which helps reduce the right to left shunt.

(3.) Improving oxygenation and correcting the acidosis.

E. **Treatment: Institute the first two steps below and call cardiology.**

1. Increase SVR to make the blood more likely to go to the lungs as you reduce the right to left intracardiac shunt. We may accomplish this by a knee-chest position. The knee-chest position, by pooling blood in the legs, also decreases systemic venous return which helps decrease the amount of deoxygenated blood flowing right to left. Having the parents hold the child in the knee-chest position also helps calm the child.

2. Increase SVR also by giving IV or oral fluids. The IV fluids should include glucose, such as D5 1/4 NS, to avoid hypoglycemia from increased glycogen utilization and decreased glycogen stores secondary to the stress of a “Tet spell”. Oxygen therapy should be provided as some blood is flowing through the lungs and may therefore be oxygenated. However, it may not help much until the right to left shunt is reduced.

3. If you are still encountering problems give morphine to decrease anxiety, relax the infundibulum, and decrease venous return. Dose is 0.1 mg/kg IV or IM but watch out for respiratory depression. Be ready to intubate. Morphine may reduce the SVR as well which will worsen the spell so be ready to give fluids. Do **not** use dopamine ! (see number 7 below)

4. If you are still encountering problems consider propranolol 0.10 - 0.25 mg/kg IV (max 10 mg) over 2 - 5 min, may repeat in 15 min. X 1. This has a negative inotropic effect on the infundibulum, which reduces the right ventricular obstruction and may allow more blood to flow into the lungs. The exact way propranolol helps in these spells is uncertain, however. It may stabilize peripheral vascular arteries thus preventing sudden reductions in SVR, or by reducing the HR may decrease the right to left shunt.

5. If you are still encountering problems consider bicarb 1 mEq/kg IV, and intubation and ventilation in an attempt to correct the acidosis. This helps abolish the hyperpnea as well. Hyperpnea may contribute to the increased venous return by making the negative thoracic pump more efficient. Abolishing the hyperpnea may therefore reduce the systemic venous return and lessen the right to left cardiac shunt.

6. If you are still encountering problems consider phenylephrine (Neosynephrine) a pure alpha agonist, to further raise the SVR. The dose is 0.1 mg/kg SC or IM, 0.01 mg/kg IV, or as a drip 0.1 - 0.5 **mcg**/kg/min.

7. **Avoid dopamine, dobutamine, digitalis, and digoxin which have positive inotropic effects and may therefore, worsen “Tet spells”.**

8. Correct anemia if present. Tachyarrhythmias may decrease RV filling as well and should be treated.

V. Digoxin

Digoxin is a potent drug that has provided physicians with the ability to treat patients with a variety of cardiology problems. Prior to its discovery and use many of these patients died. However, while it is very useful, digoxin has numerous undesirable side effects. Only a physician with adequate knowledge concerning its actions, and potential complications may use it safely. Digoxin toxicity is one of the most common drug complications seen. This is especially true in neonates and small children, often from a miscalculation of its dose. To avoid this iatrogenic complication, all digoxin orders (indeed all medication

orders) should specify the patient's weight in kilograms, the mcg/kg (or mg/kg) desired and the mcg (or mg) dose. In this way the nurses and pharmacy may double-check the calculations to avoid mistakes. Because of its potential for misuse, it is mentioned in this manual.

A. Actions/ pharmacology:

1. Digitalis glycoside has positive inotropic and negative chronotropic actions. It increases myocardial catecholamine levels at low doses and inhibits sarcolemmal Na^+-K^+-ATPase at higher doses to enhance contractility. It indirectly increases vagal activity thereby slowing S-A node firing and A-V conduction.

2. It may reduce CSF production, and cause splanchnic, peripheral and pulmonary vasoconstriction.

3. Rapid absorption occurs after oral doses but this is diminished by antacids and rapid transit times.

4. Glomerular filtration and tubular secretion account for most of the clearance.

B. Monitoring:

1. Follow the heart rate and rhythm closely. Periodic ECG's should be performed to assess for toxicity.

2. For patients receiving diuretics or amphotericin which may cause hypokalemia, follow very closely as dig. toxicity is more likely.

3. Dig. toxicity is more likely in-patients with hypokalemia, hypercalcemia, or hypo **or** hypermagnesemia.

4. Drug interactions: indomethacin, spironolactone, quinidine, and verapamil all decrease digoxin clearance. Metoclopramide decreases digoxin absorption. Spironolactone interferes with the radioimmunoassay.

5. Therapeutic levels are 1 - 2 ng/ml. However, dig. like substances make such a determination inaccurate as the level may reflect these other substances and not just the drug.

C. Adverse effects and precautions:

1. GI manifestations: nausea, feeding intolerance, vomiting, diarrhea

2. CNS effects: lethargy

3. Cardiac effects: some of these are partially due to hyperkalemia which is seen with digoxin/ digitalis toxicity

Nontoxic Cardiac Effects	**Toxic Cardiac Effects**
shortening of Qtc interval	prolongation of PR interval
sagging ST segment	sinus bradycardia or SA block
diminished T-wave amplitude	atrial or nodal ectopic beats
slowing of heart rate	ventricular arrhythmias

D. Digoxin dosing:

1. Loading or "digitalizing" doses are usually only used when treating dysrhythmias. Oral doses are usually 25% greater than IV doses. These loading doses are given in 3 divided doses. Digitalize with 1/2 the digitalizing dose, 8 hours later give 1/4 the digitalizing dose, and 8 hours after that give the final 1/4 digitalizing dose.

	Total loading dose		**Maintenance dose**		
Post-conceptual age	**mcg**/kg/**24 hours**		**mcg**/kg/**dose**		interval
(weeks)	IV	PO	IV	PO	(hours)
≤ 29	15	20	4	5	q 24
30 - 36	20	25	5	6	q 24
37 - 48	30	40	4	5	q 12
49 weeks - 2 years	40	50	5	6	q 12
2 - 10 years	20 - 30	30 - 40	3 - 4	4 - 5	q 12
> 10 years	10 - 15	0.75 - 1.25 ***mg***	25% of IV loading dose	0.125 - 0.25 ***mg***	q ***24***

From: the 1995 Neofax and 13th edition of the Harriet Lane Handbook

E. Digoxin toxicity: use digoxin-immune "Fab" antibody (Digibind®), **stop the digoxin,** correct any electrolyte problems, hypoxia, and acid-base abnormalities.

1. Digibind® is a sterile powder of antigen binding fragments (Fab) derived from specific antidigoxin antibodies produced by sheep. Digibind® binds molecules of digoxin, making them unavailable for binding at their site of action on the cells. The Fab fragment-digoxin complex is excreted by the kidney.

2. It also has been used in cases of life threatening digitoxin overdose.

3. Digibind® will interfere with digoxin or digitoxin immunoassay measurements.

4. Dosage:

a. For **acute** ingestions of **unknown amounts** 20 vials is adequate to treat both adults and children. If the child is small and volume is a concern, 10 vials may be given to the child first and then they may be monitored for their response and an additional 10 vials be given as needed.

b. For toxicity occurring during **chronic** therapy a single vial will usually suffice if the patient weighs ≤ 20 kg. For larger patients 6 vials is usually adequate.

c. For **acute** ingestions of **known** amounts each vial of Digibind® contains 38 mg of the Fab fragments which will bind approximately 0.5 mg of digoxin or digitoxin. The dose of Digibind® may be based on the number of tablets or capsules ingested or the digoxin level in ng/ml (see following tables for adults and children).

Digibindâ Dose for Adults and Children Based on Number of Tablets or Capsules Ingested in a Single Large Digoxin Overdose

Number of digoxin tablets or capsules ingested (0.25 mg tablets or 0.2 mg Lanoxicapsâ capsules)	Digibindâ dose (number of vials)
25	10
50	20
75	30
100	40
150	60
200	80

Digibindâ Dose (mg) for Infants and Small Children Based on Steady State Serum Digoxin Concentration and the Patient's Weight

	Serum digoxin concentration (ng/ml)						
Weight (kg)	**1**	**2**	**4**	**8**	**12**	**16**	**20**
1	0.4 mg*	1 mg*	1.5 mg*	3 mg*	5 mg	6 mg	8 mg
3	1 mg*	2 mg*	5 mg	9 mg	14 mg	18 mg	23 mg
5	2 mg*	4 mg	8 mg	15 mg	23 mg	30 mg	38 mg
10	4 mg	8 mg	15 mg	30 mg	46 mg	61 mg	76 mg
20	8 mg	15 mg	30 mg	61 mg	91 mg	122 mg	152 mg

*** dilution of *reconstituted* vial by adding 34 cc of sterile isotonic saline to achieve a final concentration of 1 mg/ml may be desirable**

Digibindâ Dose (vials) for Adults and Large Children Based on Steady State Serum Digoxin Concentration and the Patient's Weight

	Serum digoxin concentration (ng/ml)						
Weight (kg)	**1**	**2**	**4**	**8**	**12**	**16**	**20**
40	0.5 v	1 v	2 v	3 v	5 v	7 v	8 v
60	0.5 v	1 v	3 v	5 v	7 v	10 v	12 v
70	1 v	2 v	3 v	6 v	9 v	11 v	14 v
80	1 v	2 v	3 v	7 v	10 v	13 v	16 v
100	1 v	2 v	4 v	8 v	12 v	16 v	20 v

v = number of vials

tables are from Digibind® package insert 1994

CHILD ABUSE

I. Physical Signs of Abuse

A. Bruises

1. < 12 mo. old unlikely to have multiple bruises except by non-accidental means

2. Sites suggestive of abuse:

a. Buttocks, thighs, lower back (paddling)

b. Genitalia, inner thighs (sexual abuse)

c. Cheek (slap)

d. Ear lobes (pinch)

e. Upper lip/frenulum, lingular frenulum (forced feeding or attempt to stop crying)

f. neck (choking)

3. Human hand marks

a. Oval grab marks (finger tips)

b. Trunk encirclement bruises (if present, also R/O frx ribs, pneumothorax, subdural hemorrhage, retinal hemorrhage from violent shaking)

c. Linear marks (fingers)

d. Hand print

e. Pinch marks

4. Human bite marks

a. Doughnut or double horseshoe shaped

b. 2-12 teeth impressions

c. Fade rapidly so especially important to get photo early

d. Salivary swabbings

e. May need forensic orthodontist to identify abuser

5. Strap marks

a. Linear bruises (belts or whips)

b. Belt buckle (C, U, or [] shaped)

c. Loop marks (doubled over cord)

6. Bizarre marks

a. Blunt instruments

b. Tattoos

c. Fork mark punctures

d. Circumferential marks (wrists, ankles)

e. Gag marks

7. Multiple bruises at different stages of healing, dating of bruises by appearance (estimates):

Age of bruise	Appearance
< 6 hrs	Red, swollen, tender
6-12 hrs	Blue, swollen, tender
4-10 days	Yellow to green
10-14 days	Brown
2-4 wks	Clearing

8. Normal bruises and external injuries

a. Facial scratches in infants with long fingernails

b. Knee, shin, forehead bruises (and bruises over other bony prominences) in ambulatory child

c. Excoriations in a child with Atopic Dermatitis

B. Burns

1. Inflicted:

a. Cigarette (7 - 10 mm - should all be the same size)

b. Match tip/incense

c. Iron/curling iron

2. Forced contact with:

a. Heating grate/radiator

b. Hot plate

3. Scalding from forced immersion:

a. Buttocks/perineum (look for doughnut of sparing)

b. glove/stocking burns

C. Head injuries

1. Scalp swelling/bruises

2. Traumatic alopecia

3. Subgaleal hematoma

4. Skull frx

5. Subdural hematoma: from direct blow or shaking, never spontaneous

6. "Shaken baby syndrome"

a. Retinal hemorrhages - if present make sure Staff ophthalmologist signs consult note as occasionally only residents perform ophthalmology consults.

b. Subdural hematoma.

c. Circumferential thoracic bruises, rib fractures- especially posterior.

d. Assoc. long bone frx in 25% - do skeletal survey!

D. Abdominal injuries- may cause shock from acute blood loss

1. Compression of viscus against vertebral column

2. Pummeling blows by blunt objects, may see abodominal bruises in periumbilical region, or over liver or spleen.

3. Rarely discolored

4. Injured sites:

a. Ruptured liver or spleen

b. Intestinal perforation

c. Intramural hematoma of duodenum (most common)

d. Ruptured blood vessel (rare)

e. Pancreatic injury (pseudocyst, traumatic pancreatitis)

f. Kidney injury (rare)

E. Bone injuries

1. Spiral fracture (fx.) or transverse fx. Spiral fx. more common in accidental as well as NAT. Consistent with described method of injury being a twisting injury.

2. Any fx. in a non-ambulatory child

3. Hx and mech of injury incompatible

4. Chip or cornier fx. of metaphysis (pathognomonic, especially if multiple), bilateral long bone fxs.

5. Subperiosteal bleeding, subperiosteal new bone formation, or bone bruise

6. Fx. in different stages of healing

7. Repeated fx. to same site

8. Rib fx. - highly suggestive in child < 2 yrs (post. > lat. > ant.)

F. Failure to thrive due to underfeeding

1. Underweight

2. Failure to gain weight at home

3. Rapid weight gain out of home

4. Ravenous appetite

5. Deprivation behaviors

G. Additional signs of physical abuse

1. Delay in seeking medical attention

2. History incompatible with injury or the history changes!! Or you receive different histories from different people and they don't make sense.

3. Injuries on the back

4. Injuries on the back of the arms (defensive)

5. Excessive clothing (attempt to hide wounds)

6. Child reports abuse: **do not ignore or minimize**

7. Repeated poorly explained trauma.

II. **Reporting**

A. Whom to call:

1. Child protective services 1-800-252-5400. For **Air Force** cases, consult with senior pediatric resident or staff, notify Dr. Goins WHMC pager 1306, home phone 681-8757, and call family advocacy (FA)@ ext. 2-5967, after hours WHMC pager #0932. If an active duty USAF member is involved, also call Office of Special Investigations (OSI)@ ext. 3-1852, after hours beeper number available by contacting the ER. If an on-base occurrence call Security Police 2-7135. For **Army** cases: contact LTC. Reginold Moore at pager 813-1727, home phone 651-6680, FA @ WHMC as above if pt at WH or Social Work at BAMC if pt. at BAMC, and if and active duty member is involved the Criminal Investigations Division (CID) at 221-1763/4. If occurrence on-post call For Sam Military Police 221-1221.

2. If the case is determined to be suspicious of abuse or is documented abuse:

a. Admit to Peds service if unable to determine if child has a safe place to go.

b. If patient is a dependent of retired Air Force, then family advocacy will not be involved and Child Protective Services (CPS) 532-2873, will need to be notified. If retired Army, both CPS and FA should be notified.

c. Remember, it is the job of FA or CPS to determine if the home is safe in questionable cases. They need to talk with guardians and make that determination - not pediatrics.

B. The individual who discloses that abuse may have occurred is required BY LAW to report

C. Explain to the parents in a non-judgmental manner that you're required by law to report any injury that may have been non-accidental regardless of your personal opinion about the injury or the parents.

III. Documentation

A. Carefully document in medical record:

1. Quotes from parent and child regarding circumstances of injury.

2. All injuries (use diagrams).

3. In all Army cases, complete a PCAN form.

B. Additional studies:

1. PT/PTT & CBC if indicated in cases of bruising

2. Skeletal survey is indicated in all children when ther is concern of physical abuse. A repeat skeletal survey should be performed a week later if the initial survey is concerning for subtle findings.

3. Head CT/MRI, other radiologic procedures, ophthalmology exam as indicated.

C. Photos

1. Call med photo; someone can be called in 24 hours a day.

2. Do not delay as evidence may fade

3. Get photos of all injuries, as well as a whole child picture

4. Serial photos of evolving injuries

IV. Disposition

A. CPS will place a legal hold prohibiting the parents from taking the child home if necessary. Again CPS will make that determination, not Pediatrics.

B. Hospitalize if medically indicated and Rx as appropriate (document injuries before Rx if possible)

C. If no medical indication for admission, but cannot send home, will need to be admitted until a proper placement can be made.

D. If no suspicion that child is in further danger (eg convincing story that perpetrator is not in home), consider sending child home with F/U. If in doubt, hospitalize. Clear the decision through CPS, attending staff, and child abuse staff (Dr. Goins-USAF/ Dr. Moore-USA).

E. If hospitalized and legal hold placed, DO NOT discharge until CPS has cleared child for discharge. A note to that effect must be placed on chart, containing name of CPS worker contacted and time. Discharge order must be specific in regards who is allowed to take child (eg: parents, name of foster parent, etc.) Foster parents must show identification.

V. Child Sexual Abuse

A. Acute - if said to have taken place in last 48-72 hours.

1. If female $\geq$ 12 years old - then should be evaluated by OB-GYN.

2. If female < 12 years old - then should be evaluated by Peds.

a. If there is a possibility of transmission of STD (i.e. penetration or oral sex) then rape kit should be completed.

b. Rape kits are in ER and self explanatory.

c. If no chance of STD (i.e. fondling breasts), then full exam with careful documentation needs to be done, but no need for cultures.

d. If offender is Active Duty, then FA, CPS, Military Police, and Office of Special Investigations (USAF) or CID (USArmy) need to be notified. (see #s sect II,A,1.)

e. As above, hospitalize if patient does not have a safe place to return.

B. Chronic or Distant - if abuse took place in the distant past.

1. If female $\geq$ 12 years - OB-GYN to evaluate. Defer to better setting than ED. If teenager, obtain informed consent from teen.

2. If female < 12 years or male - full evaluation (for subtle signs of old trauma) may be scheduled with child abuse staff. Defer exam to better setting than ED.

3. Again FA, CPS and possibly OSI or Military Police should be notified.

C. Sedation may be considered in a child unable to cooperate with an exam.

D. Any questions can always be directed to child abuse staff.

COAGULOPATHIES

I. DDX

A. Hemophilia A

B. Hemophilia B

C. von Willebrand’s disease

D. Other rare hereditary disorders:

1. Factor I: Afibrinogenemia, hypofibrinogenemia, and dysfibrinogenemia.
2. Factor II: Hypoprothrombinemia (8 cases) and dysprothrombinemia (12 cases).
3. Factor V: Parahemophilia. Autosomal (1q) recessive. Incidence: 1/500,000.
4. Factor VII: Very rare autosomal (13q) recessive disorder.
5. Factor X: Autosomal (chromosome 13) recessive, Incidence 1/500,000.
6. Factor XI: Hemophilia C Autosomal (chromosome 4) recessive.
7. Factor XII: Autosomal (chromosome 6) recessive.
8. Factor XIII: Very rare autosomal (subunit A on 6p, subunit B on 1q) recessive disorder.

E. Acquired bleeding disorders:

1. Liver disease:
   a. Site of synthesis of most coagulation factors, predisposing to clinical bleeding
   b. Prolonged PT, aPTT, TT. FVIII activity and antigen usually increased
   c. Presents with hemorrhage, HSM and jaundice.
2. Vitamin K deficiency:
   a. Cofactor for γ-carboxylation for C-terminus of protein, required for function.Factors II (prothrombin), VII, IX and X, and Proteins C and S.
   b. Requires normal bacterial flora for intestinal absorption.
   c. Hemorrhagic disease of the newborn, cystic fibrosis, biliary atresia, obstructive jaundice, small intestine dysfunction, and rarely with antibiotic therapy.
3. Cyanotic congenital heart disease: Polycythemia associated with poor coagulation function.
4. Renal failure:
   a. Platelet function is abnormal in uremia.
   b. DIC in acute renal failure, allograft rejection, and chronic renal insufficiency.
   c. Protein loss in nephrotic syndrome may associate with hemorrhage or thrombotic complications.
5. Anticoagulants:

a. Acquired hemophilia-in adults with autoimmune antibodies to factor VIII. Presents with severe life threatening hemorrhage, prolonged PTT which does not correct on mixing study.
b. Lupus anticoagulant in patients with SLE. Predisposes to DVT but not bleeding.

6. Disseminated intravascular coagulation (DIC):
   a. In the newborn: intrauterine infections, maternal toxemia, abruptio placentae, severe respiratory distress syndrome, and necrotizing enterocolitis.
   b. Most common causes in childhood: fulminant bacterial sepsis, disseminated viral diseases, and massive head injuries.
   c. Usually associated with:
      1) fragmented RBCs,
      2) low or decreasing platelet count,
      3) hemoglobinemia (pink plasma)
      4) prolonged PT and APTT, and low or decreasing fibrinogen. Confirm by measuring fibrin split products (FSP's), or D-dimers
      5) Factors V and VIII (decreased).
      6) Clinical manifestations: bleeding and/or thrombosis. Minimal until consumption of platelets and coagulation factors has occurred.
7. Thrombocytopenia:
   a. Platelet production failure: Generalized bone marrow failure due to leukemia, myelodysplasia, aplastic anemia, megaloblastic anemia, uremia or marrow infiltration.
   b. Selective megakaryocyte depression due to drugs, alcohol, chemicals or viral infections.
   c. Abnormal distribution of platelets: Splenomegaly.
   d. Increased destruction of platelets:
      1) Immune: neonatal alloimmune thrombocytopenia, post transfusion, ITP, SLE, post-infection, AIDS, post-BMT.
      2) Drug induced.
      3) DIC, including hemolytic uremic syndrome, and Kasselbach-Merrit syndrome.
   e. Dilutional loss: Massive transfusion of stored blood.

8. Qualitative platelet disorders (disorders of platelet function):
   a. Characterized by a prolonged bleeding time out of proportion to the platelet count.
   b. Hereditary forms include: Bernard-Soulier syndrome, Wiskott-Aldrich syndrome, Chediak-Higashi syndrome, gray platelet syndrome, von Willebrand's disease, and others
   c. Most common acquired form is secondary to drug use (e.g. aspirin, NSAIDs).

II. Diagnosis

A. History:
1. Trauma
2. Headache (consider intracranial bleed).
3. Prior bleeding (type, extent, and duration).

4. Careful family history
5. Medication use (particularly aspirin).

B. Pattern of bleeding: (see table 3)
1. Mucocutaneous bleeding (petechiae, ecchymoses, epistaxis, and GI & GU bleeding) suggestive of platelet disorder.
2. Purpura most cases are due to vasculitis with other systemic illnesses, not associated with thrombocytopenia.
3. Bleeding into potential spaces such as joints, between facial planes and into the retroperitoneum is characteristic of factor deficiencies or warfarin toxicity.
4. Bleeding from multiple sites to include both mucocutaneous and potential space bleeding may be seen in DIC.

C. Physical Examination:
1. Evaluate hemodynamic status.
2. Identify source of bleeding. Early hemarthrosis will show only pain with ROM, or bearing wt. All acute musculoskeletal pain is assumed secondary to occult bleed when factor level is low in a hemophiliac.
3. Look for other sites of blood loss (e.g. bruising, petechiae). Assess wound sites for oozing (IV sites, umbilical cord stump).

D. Screening assays:
1. CBC: check plt count, assess for microangiopathic anemia.
2. Platelet count usu adequate if> 50,000. Platelet counts < 10,000 may lead to spontaneous hemorrhage.
3. Prothrombin Time (PT): identifies coagulation abnormalities of the extrinsic and common pathways; requires factors VII, X, V, II, and I.
4. Activated Partial Thromboplastin Time (aPTT): identifies coagulation abnormalities of the contact phase and the intrinsic and common pathways; requires factors XII, XI, IX, VIII, V, II, and I
5. Bleeding Time: Unreliable test in children <4yrs. In expert hands, a bleeding time > 15 minutes is frankly abnormal and indicates:
   a. Severe impairment of platelet function.
   b. Very low blood levels of vWF.
   c. Afibrinogenemia or severe Factor V deficiency, or
   d. Platelet storage pool deficiency.
   e. Bleeding times between 8 and 15 minutes are usually associated with moderately low plasma vWF. Antiplatelet drugs, lupus-like anticoagulant, Factor XI deficiency.
6. The Tourniquet Test (Rumple-Leede Test): Identifies capillary fragility associated with viral infections, drug-induced vasculitis and collagen diseases (e.g., SLE).
7. Fibrinogen level, FSP’s, D-dimers. Nl fibrinogen level is > 150-200. FSP’s < 10.
8. Thrombin Clotting Time/Thrombin Time Assay (TCT/TT): Isolates the common pathway. Requires fibrinogen especially. Heparin will inhibit this test.
9. Thromboelastogram (TEG); a relatively new assay that observes the efficiency of whole blood in forming clot. Measurements reported allow information regarding the source of coagulopathy (contact, platelet, factor deficiency, inhibitor)

E. Confirmatory tests:

1. Factor Assays are reported as % activity compared to controls. These are usually functional assays- Clot-based coagulation assays (based on PT, APTT, TT)
2. Quantitative assays measuring factor levels, done only at reference labs. (I.e.: vonWillebrand antigen assay).
3. Gene analysis only available for known genetic abnormalities. Usually requires a proband have a defined abnormality, then the test can be applied to other affected family members as a screen.

III Treatment

A. General Principles:
1. Stabilize patient.
2. Utilize mechanical methods of hemostasis (e.g.: topical thrombin, cauterize bleeding vessels, oral surgery, etc.)
3. Give directed therapy for factor or platelet correction.
4. Pain control.
5. Avoid aspirin containing products.

B. Mucosal bleeds:
1. Local applications of GELFOAM®. Afrin for epistaxis, repeat every 20 min for bleeding as BP tolerates.
2. AMICAR® (epsilon aminocaproic acid) prevents clot dissolution in mouth bleeds. Contraindicated in hematuria or DIC. Dose = 100 mg/kg IV or PO q 6 hr (up to 24 gm/day).

C. Blood component therapy:
1. Fresh frozen plasma (FFP).
a. Contains all blood coagulation factors (by definition, 1mL = 1 unit of factor activity). Useful when diagnosis is unknown.
b. Indications. (WHMC 7/99)
1) Prophylaxis prior to procedures PT > 17, aPTT > 66
2) Bleeding with acquired coagulopathy due to a deficiency of coagulation factors.
3) Massive blood transfusion > 1 blood volume with evidence of a coagulation deficiency and bleeding.
4) Reversal of warfarin effect.
5) Congenital deficiency of Factor II, V, X, XI, or XIII.
c. Dose: 10-ml/kg-body weight. Monitor with PT and aPTT.
d. Remember: 6 units of platelets or 1 platelet pheresis unit delivers 1 unit of FFP.
e. Contraindicated as a volume expander (JAMA, March 9, 1994, p.778).

2. Cryoprecipitate:
a. 1 unit (9 to 16 mL) contains 80-120U Factor VIII and vWF, 200-300 mg fibrinogen, and 40-60U Factor XIII. Insignificant amounts of other factors.
b. Indications:
1) Hypofibrinogenemia. Most cases DIC so FFP also needed.
2) VonWillebrand disease if DDAVP not effective (Type II or III).
3) Hemophilia A when treated commercial factor VIII concentrates are not available.
c. Dose:

1) Congenital hypofibrinogenemia: 1 bag/5 kg body weight q o d.
2) Consumptive hypofibrinogenemia: 1 bag/5 kg body weight. Monitor fibrinogen level to guide frequency.
3) vonWillebrand disease: 1 bag/10 kg body weight.
4) Hemophilia A: Number of Bags of Cryo = [(Plasma/Volume in mL x % Increase in Factor VIII Needed)/100]/80.
5) See Factor VIII below for guidelines.

3. Platelets:
a. Indications (WHMC 7/99)
1) Decreased platelet production and/or increased platelet.
2) Bleeding prophylaxis for counts below 5000 ($5X10^9$).
3) Between 5000 and 20,000 give on the basis of significant bleeding risk.
4) Congenital platelet dysfunction: weigh risk of alloimmunization vs. bleeding Consider pharmacological methods of enhancing platelet functions (e.g., Desmopressin®, DDAVP).
5) Acquired platelet dysfunction (e.g., drug-related etc.).

b. Dose:

1 unit random platelets/10 kg body weight, or 4 units/M^2 body surface area incrementally raises platelet count by $50x10^9$/L.

D. Blood Derivative Therapy:
1. Human Factor VIII concentrate:
a. One unit/kg body weight of Factor VIII will raise plasma activity by 2%.
b. Biological half-life: 8 to 12 hours.
c. Prepared from as many as 22,000 donors.
d. Indications:
1) Severe Factor VIII deficiency, or mild to moderate Factor VIII deficiency with inadequate response to DDAVP.
2) Only one product, HUMATE-P®, (Armour, Kankakee, Illinois), contains adequate amounts of vWF for treatment of vonWillebrand disease, and is preferred over cryoprecipitate.
3) Dosage depends on type and severity of bleeding (see Table 1).
4) Available products: see Table 2.
2. Porcine Factor VIII concentrates: for patients with Factor VIII deficiency and inhibitors. Dose the same as human factor VIII.
3. Factor VIIA: FDA approved for use in patients with VIII inhibitors. Heme consult to set up and give (has very short half life and requires continuous infusion)
4. Prothrombin complex concentrate (Factor IX complex):
a. One unit/kg body weight of Factor IX will raise plasma level by 0.5% to 1.0%.
b. Biological half-life: 10.5 to 13.5 hours
c. Indications:
1) Factor IX deficiency.
2) Factor VIII deficiency with inhibitors (would be first choice of treatment)
3) Cautions:
a) Never administer simultaneously with AMICAR® (results in hypercoagulable state with risk of thrombosis).

b) Risk of thrombosis and DIC is high in patients with liver disease.
c) Dosage usually 75-80 units/kg every 12-24 hours.
d) Available products: see Table 2.

5. Activated prothrombin complex concentrates (AUTOPLEX®, FEIBA®):
   a. For use only in patients with inhibitors.
   b. Dose: 50 to 100 U/kg.
6. Purified and recombinant Factor IX concentrates.
   a. Indications: first line therapy for factor IX deficiency
   b. Recombinant Factor IX (Benefix®) requires 20% dose increase for equal efficacy.
   c. See tables 1 and 2.

E. Desmopressin (DDAVP, STIMATE®)
1. Transiently increases in all components of the FVIII, increases plasminogen activator.
2. Indications:
   a. Mild to moderate hemophilia A
   b. VonWillebrand disease with known previous response to DDAVP.
      1) Use in type IIb vonWillebrand disease will result in severe drop in platelets.
      2) Dose: 0.3 micrograms/kg diluted in 50cc normal saline, IV over 30 minutes.
      3) Also approved for intranasal administration 3 microgram/kg.
      4) Can be repeated at 12 hour intervals, BUT decreasing response is noted with each successive dose.
      5) Causes a rise in plasminogen activator.
      6) For oral bleeding use AMICAR®. 100 mg/kg q 6hr x 4-7 days.

Table 1: RECOMMENDED DOSAGES OF FACTOR VIII* and IX

Indications	Initial Dose (Factor VIII, U/kg)	Initial Dose (Factor IX, U/kg)	Other Treatment
Routine dose			
E.g.: sutures, joint, muscle, mouth	**20-25	30-40	Amicar with mouth bleeds
Major Dose			
E.g.: Head, GI, prior to invasive procedure (ex LP), and large joint bleeds (hip, knee)	40-50 Repeat (q12-24)	40-50 (Add 20% if using Benefix®) Repeat (q 24-48)	Ice packs, Non-weight bearing, light weight splint, Complete bed rest for iliopsoas hemorrhage

****In individuals who have mild hemophilia A, DDAVP (Desmopressin®) is the treatment of choice rather than factor VIII concentrates.**

Adapted from Lusher JM, Warrier I: Hemophilia. Pediatrics in Review 12:275, 1991. And Emergency Care for Patients with Hemophila, by Nursing Group of Hemophilia RegionVI, 1999

Table 2. PRODUCTS AVAILABLE FOR TREATMENT OF HEMOPHILIA A, HEMOPHILIA B, VON WILLEBRAND'S DISEASE.

Product	Manufacturer	Method of Viral Inactivation
	FACTOR VIII	
Intermediate Purity		
Koate Hp®	Alpha	Solvent/detergent
Humate-P®	Armour	Pasteurized (10 hr, 60°C)
Monoclonal Purified		
Monoclate-P®	Amour	Monoclonal antibody-purified plus pasteurization (10hr, 60°C)
Hemofil-M®	Hyland	Monoclonal antibody-purified plus solvent/detergent
Method-M®	American Red Cross	Monoclonal antibody-purified plus solvent/detergent
Porcine VIII		
Hyate C®	Porton	Porcine factor VIII
Recombinant VIII		
Recombinate®	Baxter-Hyland	None/ Synthetic (uses pasteurized human albumin in prep)
Bioclate®	Centeon	same
Kogenate®	Bayer	same
Fresh blood product		
Cryoprecipitate	Local blood blank	None (donor screening tests only)
DDAVP	Rorer	None/Synthetic preparation
	FACTOR IX	
Prothrombin Complex		
Konyne®	Cutter	Dry Heat (80 hr, 68°C)
Proplex T®	Baxter Hyland	Dry Heat (150 hr, 60°C)
Activated Prothrombin Complex		
Autoplex T®	Baxter Hyland	Dry Heat
FEIBA®		
Profilnine HT®	Alpha	Heated in an organic solvent [N-Heptane] (20 hr, 60°C)
Monoclonal IX		
Mononine®		
Alphanine ®	Alpha	Monoclonal antibody-purified plus heated in organic solvent
Recombinant IX		
Benefix®	Genetics Institute	None/ synthetic (No human albumin in prep or storage)
Fresh blood product		
FFP	Local blood bank	None (donor screening tests only)
	FACTOR VII A	
Recombinant VII A		
NovoSeven®	NovoNordisk	None/ synthetic (No human albumin in prep or storage)
	PRODUCTS FOR VON WILLEBRAND'S DISEASE	

DDAVP	Rorer	None/Synthetic preparation
Humate-P®	Armour	Pasteurized (10 hr, 60°C)
Cryoprecipitate	Local blood bank	None (donor screening tests only)

Adapted from Julius C, Westphal RG: The Safety of Blood Components and Derivations, Hematology/Oncology Clinics of North America 6:1071, 1992.

Table 3. DIFFERENTIATION OF PLATELET-VASCULAR ABNORMALITY FROM COAGULATION PROTEIN DEFICIENCY BY HISTORY AND PHYSICAL EXAMINATION

	Platelet or Vascular Abnormality	Coagulation Factor Deficiency
Ecchymoses	Small and superficial	Large and deep (often palpable)
Petechiae	Frequent	Never
Mucosal hemorrhage	Frequent	Uncommon in hemophilia: frequent in acquired disorders
Muscle, joint, or internal hemorrhage	Uncommon	Frequent
Prolonged bleeding from cuts and scratches	Frequent	Rare
Bleeding with trauma or surgery	Immediate; stops with pressure	Delayed (1-2 days later); does not stop with pressure

CROUP (LARYNGOTRACHEITIS)

I. Introduction

A. Viral infection causing subglottic and tracheal swelling.

B. Most common cause of stridor in children.

C. Parainfluenza virus (I) recovered in 50%. Additional causes: Parainfluenza (II & III), influenza, RSV, adenovirus, measles.

D. More common in fall and winter.

E. Usual age 3 mos - 5 years (mean = 18 months).

II. Pathophysiology

A. Virus invades epithelium of the nasopharynx with local spread to

larynx and trachea.

B. Epithelial damage causes mucous production and loss of ciliary function.

C. Edema of the subglottic larynx. A small amount of edema within the ring of the cricoid cartilage leads to a large decrease in air flow.

D. Fibrinous exudate partially occludes the lumen of the trachea.

III. Clinical Manifestations

A. Insidious onset of fever, coryza, cough and sore throat.

B. Stridor and barking cough develop on day 2-3.

C. May be unable to maintain PO intake.

D. Majority appear mildly to moderately ill.

E. Fever is quite variable(100 to 105 F).

F. Minimal to severe respiratory distress with varying amounts of tachypnea, dyspnea, stridor, flaring and retractions.

G. Lungs are clear with transmitted upper airway sounds; wheezes occur if there is concomitant lower airway involvement.

H. Rarely cyanotic.

IV. Lab/X-ray - rarely abnormal or of diagnostic value.

A. WBC mildly elevated with predominance of PMN’s.

B. The “steeple sign”, a manifestation of subglottic narrowing, seen on X-ray of the neck, is neither sensitive or specific.

V. Differential diagnosis includes:

A. Epiglottitis, foreign body, retropharyngeal or peritonsillar abscesses, bacterial tracheitis, congenital or acquired subglottic stenosis, paraquat poisoning and laryngeal diphtheria.

B. The history is <u>extremely</u> important. It may be the only source suggestive of another process such as a foreign body.

VI. Management

A. Make the patient comfortable. Avoid unnecessary procedures that will increase anxiety and worsen respiratory status.

B. Contact isolation. Pulse oximetry.

C. Rehydration. Account for ongoing losses such as tachypnea and fever.

D. Medical management.

1. Mist therapy:

a. Water droplets moisten mucosa and decrease viscosity of secretions.

b. Temperature not important (although cold mist may provoke bronchospasm in patient with RAD).

c. Administer with O_2 in hypoxic patient.

2. Racemic Epinephrine

a. Vasoconstrictive effects decrease mucosal edema.

b. 2.25% sol'n 0.05 cc/kg (0.5 cc max dose) in 3 cc NS.

c. Hold for HR > 180 (unless tachy from respiratory failure).

d. Potential rebound phenomenon: initial improvement followed by deterioration over the next 1-2 hours can occur and should be anticipated. The deterioration is usally not to a worse state than the child’s pre-racemic epinephrine baseline.

e. Contraindications: subvalvular aortic stenosis, pulmonary stenosis, Tetralogy of Fallot

3. Corticosteroids

a. Edema decreased by suppressing local inflammatory reaction, decreasing lymphoid swelling and decreasing capillary permeability.

b. Dexamethasone (Decadron) 0.6 mg/kg IV or IM x 1.

4. Antibiotics not indicated.

5. Sedation contraindicated.

E. Intubation in respiratory failure (< 5% of hospitalized patients).

1. Call ENT and anesthesia for help in intubation.

2. Preferably done in OR under controlled conditions.

3. Endotracheal tube (ETT) should be 1-2 sizes smaller than normally used.

4. ENT should be prepared for emergent bronchoscopy or tracheostomy.

5. Once safely intubated orally, may switch to more secure nasotracheal tube if desired.

6. CXR to verify tube position.

7. If needed may sedate or paralyze and use mechanical ventilation.

8. Extubation best done when significant air leak develops (2-6 days after intubation).

9. Decadron has been used to help decrease edema; 0.25 - 0.5 mg/kg IV q 6-12 hours prior to the extubation attempt and may be given again at extubation then prn.

F. Indications for admission (> 85% can be managed as outpatients).

1. Significant respiratory compromise.

2. Dehydration.

3. Recurrent Emergency Department or clinic visits in 24 hours.

4. Other situations which may require admission:

a. Patient < 1 year old.

b. Patient lives a long distance from the hospital or has inadequate transportation.

c. Inadequate observation or followup is likely.

d. Significant parental anxiety exists.

G. Criteria for discharge from the clinic or Emergency Department:

1. The episode must be the first visit to the hospital for croup.

2. If the patient has received a racemic epi. neb., steroids, and is in **no** respiratory distress 2.5 hours after the drugs were administered, then they may be considered for discharge home.

3. Phone followup **must** be assured a few hours later. Documentation of the phone call should be performed.

4. If any doubt exists for the fulfillment of **any** of these criteria the patient should be admitted.

DIABETIC KETOACIDOSIS

Definition: A metabolic state in an insulin dependent diabetic caused by insufficient insulin and excess of glucagon. It is characterized by hyperglycemia with blood glucose concentrations usually exceeding 300 mg/dl, ketonemia with total ketones positive at a 1:2 dilution in the serum or a positive sodium nitroprusside reaction with undiluted urine (Acetone, ketostix, etc.), acidosis with a pH of less than 7.30, and serum bicarbonate level of less than 15 mEq/l. The insulin lack allows unchecked ketogenesis and gluconeogenesis to occur with additional glucose released through unimpeded glycogenolysis. The elevated glucagon/insulin ratio promotes ketone body formation, glycogenolysis and gluconeogenesis.

Treatment Regimen for DKA in Children

I. Admission

A. History

1. Known diabetic? Missed insulin dose?

2. Emotional or physical trauma?

3. Infection? ASA or other ingestion? Known weight loss? Pregnancy? Appy?

B. Physical Examination

1. Level of consciousness

2. Respiratory status: depressed or Kussmaul? (fast and deep)

3. Extent of dehydration: BP, skin turgor, mucous membranes. (Most patients with DKA will be at least 10% dehydrated.) Patients are total body H_2O depleted as extracellular glucose causes an osmotic diuresis.

4. Flushed face? Acetone (fruity) breath?

5. Source of infection

6. Abdominal pain

a. Commonly seen as a non-specific symptom accompanying metabolic acidosis

b. If persistent, consider diff. dx. of "acute abdomen”: i.e. appy, pyelonephritis, PID, etc.

c. Severe abdominal pain may be associated with a transient elevation of pancreatic (? salivary) enzymes in the absence of documented pancreatitis.

C. Laboratory Data

1. Blood

a. Dextrostix--every 30 minutes until dextrose is added to IV fluids, then may go to every one hour until stabilized. **Watch rate of fall**--average rate is 75 mg/dl/hr, but may be as high as 300 mg/dl/hr. **We desire a fall of 50 - 100 mg/dl/hr**, add D5 as needed to maintain desired rate of fall.

b. Serum glucose--on admission and then every 1-2 hours

c. Serum ketones--on admission

d. Urine ketones--on admission and every void; may actually increase even as acidosis is improving due to a shift of Beta-hydroxybutyrate to acetoacetate and acetone.

e. pH--venous blood gas sufficient to follow level; follow every 2 hours. Occasionally ABG (A-line) is needed.

f. Serum electrolytes--follow every 2 hours X 2; then every 4 hours.

g. BUN/Creatinine--BUN every 2 hours X 2; then every 4 hours; creatinine on admission.

h. Serum calcium/phosphate--follow every 4 hours.

i. Serum magnesium--on admission and every 8 hours.

j. Serum osmolarity--follow every 4 hours by lab; and every 1-2 hours initially by calculation: 2Na + [glucose/18] + [BUN/3]; normal is < 300. Don't let serum osmolarity fall too fast, ie, too rapid a fall in glucose (see a. Dextrostix above).

k. Vital signs, including mental status exam every 30 min - 1 hour initially.

l. B-HCG if pregnancy is possible.

2. EKG - on admission and monitor throughout ICU stay. Screen for hyperkalemia (peaked T waves) and hypokalemia (flattened T waves).

3. Look for source of infection and consider:

a. Blood cultures

b. Chest X-ray

c. Urinalysis/Urine C&S

d. Lumbar Puncture

e. Other cultures as history and physical exam suggest

4. **If <u>NEW</u> diabetic, draw the following :**

a. Anti-insulin antibodies

b. Anti-islet cell antibodies

c. C-peptide

d. HbAlc

e. TFT’s, anti-thyroid antibodies.

5. Record all lab values on a flow sheet (flow sheet at end of chapter may be enlarged on a copy machine and used).

II. Treatment

A. Fluids and Electrolytes

1. Initial Bolus

a. 20 cc/kg of NS over an appropriate period of time. If in shock, get it in as rapidly as possible and give more as needed. If the patient is unstable remember your ABC’s and intubate patient.

b. This may be repeated once if patient still seems hemodynamically compromised due to dehydration

c. Further fluid boluses should be guided by the CVP, BP, etc.

2. Maintenance and deficit replacement

a. After the bolus, start 1/2 NS with 20 Meq/L KCl and 20 Meq/L KPhos at ~1.5 x maintenance plus ~8% deficit to be replaced evenly over 36-48 hours (maximum total rate of 2X maintenance as fluid overload may promote cerebral edema). As the patient’s acidosis corrects, hypokalemia and/or hypophosphatemia may occur and necessitate increasing the KCl and KPhos to 30 Meq/L each.

b. Add glucose (D5-D10) to the IV fluids as the patient's glucose approaches 250-300 mg/dl to prevent hypoglycemia and hypoosmolality.

(1). If patient is hypoglycemic but alert you may give orange juice.

(2). If patient is hypoglycemic and unconscious may give glucagon (< 10 kg use 0.1 mg/kg, > 10 kg use 1 mg/dose IV,

IM or SQ) and glucose, (0.5 g/kg of D25W) and increase glucose concentration in IV. Stop the insulin infusion temporarily, resuming it when soon as the hypoglycemia has resolved. (Insulin is needed to clear the acidosis, even with a normal glucose).

c. Replace deficit fluids over 48 hours.

d. Continue 1.5X maint. fluids until glucosuria and large UOP tapers off, then continue maint. fluids until the patient is ready to advance to an enteral diet.

e. Frequently reassess the patient's neurologic, fluid, and laboratory status. Gradual improvement in neurologic status, resolution of metabolic acidosis, and avoidance of iatrogenic hypoglycemia and electrolyte abnormalities are the main goals of monitoring and therapy.

f. A flow sheet of all labs and interventions, such as changes in IVF or insulin rates is an invaluble tool in following the patient's progress.

B. Insulin

1. Given as an IV solution piggybacked into main IV line

2. It has never been shown that an initial bolus of IV insulin beneficial prior to starting the continuous infusion of insulin.

3. Run infusion at 0.1 units/kg/hr until reassess, may increase if glucose not falling as expected.

4. Continue until patients serum $HCO3^-$ is > 15 mEq/L.

5. Record all changes on a flow sheet

6. Made as a solution of 125 units Regular insulin in 250 cc NS (0.5 units/cc) Flush line with insulin as insulin binds to IV tubing.

7. If glucose is < 200 then increase the glucose concentration and follow the glucose more closely. If the glucose is < 150 ***and*** the HCO3 is ≤ 15 increase the glucose more and follow the glucose even more closely. **You may need to increase the glucose rate again.** If the glucose is < 150 ***and*** the $HCO3^-$ is > 15 increase the glucose and decrease the insulin drip.

8. Aim to keep B.G. 150-250 until acidosis resolved, pH> 7.25, HCO_3 > 15.

C. Flow sheet

The trend of events during the course of treatment is often more important than is the patient's condition at any point in time. A flow sheet is the most important facilitator and gauge of proper management and should always be at the bedside.

Recommendations for frequency of lab tests, checks of vital signs, etc. are only a minimum requirement for most cases of DKA. There may be situations where more frequent testing and documentation is required.

D. Bicarbonate Therapy

1. The use of sodium bicarbonate in DKA is very controversial.

a. Arguments Against

(1). HCO_3 is produced endogenously with appropriate insulin therapy by oxidation of ketoacids.

(2). Alkalosis shifts the O_2-dissociation curve to the left decreasing oxygen release to the tissues and potentially increasing lactic acidosis.

(3). Increases potassium needs by accelerating the entry of potassium intracellularly.

(4). May lead to worsening of cerebral acidosis.

(5). Alkalosis decreases ionized Calcium.

b. Complications with pH at 7.0 or less

(1). Diminishes respiratory minute volume

(2). May produce hypotension with peripheral vasodilatation

(3). Impairs myocardial function and increases the risk of arrhythmias.

(4). May be a factor in insulin resistance

2. Consider using bicarbonate at pH of 7.10 or below

a. Give 1 mEq/kg over 1 hr IV.

b. Repeat as necessary to achieve a pH >7.10 as guided by ABG's.

III. Interpretation of Laboratory Tests

A. Ketones

1. Acetest measures acetoacetate (and acetone) only, whereas B-hydroxybutyrate is the major (80%) ketone body in early DKA.

2. In the severely hypoxic patient almost all ketone bodies may be B-OHB and Acetest negative

3. Urine ketones may paradoxically increase even in the face of improving serum acidosis as B-OHB is converted to acetoacetate.

B. Sodium

1. Hyperglycemia falsely lowers the serum Na: for each 100 mg/dl increase in serum glucose over 100 mg/dl, there is a decrease in serum sodium of 1.6 mEq/L.

2. Significant hyperlipidemia also falsely lowers the serum sodium. The Na decreases 2 mEq/L for each 1.0 gm/dL increase in triglycerides.

3. True Na = measured Na + 1.6 X [(glucose - 100)/100]. Failure of the “true” or “corrected” Na to increase with treatment may suggest that excess free water is present and may increase the risk of cerebral edema. Be especially concerned if hyponatremia was present at the start of treatment as the risk of cerebral edema may be increased further.

C. Potassium

1. Acidosis causes increase in serum potassium secondary to the intracellular to extracellular shifts. This scenario may not be a mechanism of relative importance in DKA. With a lack of insulin, potassium moves from the ICF to the ECF. With the osmotic diuresis, potassium can be lost in great quantities.

2. Normal plasma concentrations may be present which do not reflect the intracellular deficits

3. As the acidosis resolves, potassium shifts intracellularly, so potassium needs lessen. Frequent assessment is needed to avoid hypo or hyperkalemia.

D. Chloride

1. Falsely lowered with hyperglycemia and hyperlipidemia

2. Deficits are about 2/3 that of sodium, but since replaced as NaCl, a mild hyperchloremia (110-115 mEq/1) is frequently seen during the treatment of DKA

3. Hyperchloremia may decrease bicarbonate reabsorption which will delay recovery from the acidosis. One of the chief benefits of providing K as KPO4 is that it decreases Cl administration.

E. Amylase

1. Often elevated in DKA, does not always indicate pancreatitis as it is usually of salivary origin and plasma lipase is usually normal.

F. Phosphate

1. Acidosis causes intracellular to extracellular shifts which may cause elevated level initially. In addition, osmotic diuresis is responsible for phosphate losses.

2. Level frequently falls during the course of therapy

3. With replacement, can see a decrease in Mg and Ca. Rare to see tetany.

4. A low level of serum phosphate (< 1.0) may produce a tendency to prolong tissue hypoxia.

G. Creatinine

1. May be falsely elevated

2. Presence of acetoacetate in the blood interferes with the usual lab method of determining creatinine

3. If abnormal, recheck prior to discharge after acidosis and ketosis have resolved

H. Magnesium

1. May be low in DKA but treatment is rarely needed unless the patient is symptomatic

I. Leukocytosis

1. Be wary of elevated WBC count

2. Though leukocytosis may be attributable to increase intrinsic catecholamines associated with DKA, it may be due to infection.

IV. Transitioning from IV to SQ insulin

A. Preferable to do this at the morning or evening meal, once the acidosis has resoved (serum $HCO3^-$ > 15 mEq/L.

B. Assure that meal tray is at bedside. Give SQ insulin dose (do not stop IV insulin infusion):

1. Give usual dose if known diabetic

2. If new diabetic, calculate approximate insulin needs:

a. Start with 0.5-0.75 units/kg/day

b. Divide 2/3 of total dose as AM dose, and 1/3 as PM dose

c. AM dose is divided 2/3 NPH and 1/3 regular

d. PM dose is divided 1/2 NPH and 1/2 regular

e. Adjust dosing as necessary during hospital stay, but do not try to "fine tune"

C. Allow patient to eat.

D. Stop IVF and insulin 30 min after SQ insulin dose.

E. Monitor QAC and QHS dextrosticks (or more frequently if indicated)

V. Diabetic Coma

10% of patients with DKA will present in a comatose state. Diabetic Coma appears to correlate better with serum osmolality better than other laboratory values. A head CT Scan is indicated in patients presenting in coma to assess for evidence of CNS infarction secondary to hyperviscosity. The depressed mental status tends to improve with treatment. In an infant in DKA who presents with fever and a clouded sensorium, sepsis or meningoencephalitis should be considered. Spinal tap should probably be deferred until after a head CT is obtained to assess for ICP and the potential for herniation.

If the patient’s Glasgow coma score is $\leq$ 8, intubation is indicated and consultation for ICP monitoring should be sought. If, as the hyperglycemia is corrected, there is no improvement or a worsening of mental status, an ICP monitor is indicated.

Intubation of these patients should be performed with rapid sequence technique including the use of appropriate sedation and neuroprotective agents. Etomidate 0.3 mg/kg, lidocaine 1 mg/kg, vecuronium 0.2 mg/kg is one appropriate regimen. Atropine is added in children less that 1-2 years of age. Fentanyl and Versed is an acceptable alternative to Etomidate. Ketamine is **contraindicated.**

VI. Cerebral Edema

There are 75 deaths from DKA per year among 100,000 diabetics under 15 years of age in the United States, and neurologic collapse from cerebral edema accounts for a large proportion of these deaths. In contrast to a diabetic coma which is present at the time of presentation, the patient’s mental status

declines once treatment begins. It occurs more frequently in new onset diabetics.

A. Signs/symptoms

1. Severe Headache

2. Changes in arousal or behavior, lethargy, loss of consciousness

3. Incontinence

4. Fixed/dilated/sluggish pupils

5. Blood pressure changes, bradycardia, temperature instability.

6. Seizures

B. Occurs between 2.5-48 hours after starting therapy.

C. No clear etiology; possible etiologies include the presence of hyponatremia, overly aggressive fluid rehydration, and the use of Bicarbonate. (all are controversial)

D. Early treatment for increased ICP includes rapid sequence intubation, mannitol (0.25 gm/kg IV), avoiding excessive fluid administration, and possibly mild hyperventilation (pCO2 30-35).

E. Very important complication of DKA to anticipate, diagnose promptly, and treat immediately. Once CNS findings occur, CT may show diffuse cerebral edema at base of the brain, subarachnoid hemorrhage, or infarction.

F. Ways recommended to avoid herniation:

1. Avoid over aggressive fluid administration.

2. Avoid Na-bicarb unless necessary.

3. Extend fluid replacement for deficit over 48 hours especially if hyperosmolar.

4. Avoid rapid declines in Na or glucose. Patients with hypernatremia on presentation may be placed on 0.9 NS for maintenance fluids.

5. Treat for symptoms of increased ICP early.

VII. Shock

An ETT, central venous line, arterial line, and foley are indicated for patients in shock.

VIII. Pulmonary Edema

Pulmonary Edema is rare but occurs. The etiology is poorly understood. Some have suggested it could be from neurogenic edema in those with CNS manifestations.

IX. Acidosis

Delayed clearing of acidosis may be related to increased requirements for insulin, hyperchloremic acidosis, or further needs for fluid resuscitation.

ECTROLYTE AND FLUID ABNORMALITIES

Introduction: To understand fluid and electrolyte abnormalities we must first consider fluid compartments. Roughly speaking, we are comprised of two compartments, extracellular fluids (ECF) and intracellular fluids (ICF). The extracellular space makes up 1/3 of our body fluids and the intracellular space 2/3. The extracellular space refers to fluids outside our cells which may be interstitial or plasma. The intracellular space refers to fluids inside the cells themselves. Simply put, the extracellular compartment is influenced by the kidneys while the intracellular space is controlled by the extracellular space. Also, the extracellular space may undergo losses via sweat, respiration, urine output, and GI losses.

A. Our total body water = 0.6 X weight (kg), for children and adults
and 0.78 X weight (kg), for neonates

B. Maintenance fluids:

1.

BODY WEIGHT (kg)	**FLUIDS**
0 - 10	100 cc/kg/day
11 - 20	1000 cc + 50 cc/kg for each kg above 10
> 20	1500 cc + 20 cc/kg for each kg above 20

Fever: intermittent temperature elevations usually do not significantly increase daily fluid losses. If fever is present for a prolonged period, however, the maintenance fluid requirement is increased by 12% for each 1 degree above 37 degrees Centigrade.

2. Maintenance Na+ is 3 mEq/100 cc's.
Maintenance K+ is 2 mEq/100 cc's.
The normal intake of Na+ is between 1 and 3 mEq/kg/day. Homeostatic mechanisms maintain the [Na+] even if intake is reduced to 0.4 mEq/kg/day in normal patients. The amount of Na+ suggested here is adequate for most children but should be reduced to 1 mEq/kg/day or less in situations where Na+ may be retained (eg. acute or chronic renal failure).

3. Acute renal failure:
Patients with acute renal failure require meticulous management of their fluids and electrolytes. Management should include twice daily weights, strict I/O's, and close laboratory monitoring.
Oligo-anuric patients should receive fluid intake equal to their total output. Output must include insensible fluid loss ($400cc/M^2$/day in afebrile children), plus GI, urinary, and any other losses. Insensible fluid losses are essentially electrolyte free and should be replaced with D5W (or D10W). Stool and urine losses of electrolytes may be determined by lab measurement (see table in chapter describing composition of fluids lost). **Remember that this applies to children, not to neonates.** Insensible losses in neonates varies with gestational age and birth weight. Also, insensible losses in neonates may be dramatically increased by phototherapy or radiant warmers, or reduced by plethesmographs.
Children may also have acute renal failure with large urine losses. These patients typically have salt-wasting syndromes.
In both forms of acute renal failure a useful method of management should include the following:

1. Restore intravascular volume regardless of the degree of renal impairment. Use isotonic fluids such as NS or 5% albumin.
2. Use D5W at 400 cc/M^2/day for insensible losses.
3. For urine, GI, and other fluid losses, replace the fluid loss cc for cc with IV fluids chosen to contain electrolytes with a similar composition to the lost fluids.
4. Avoid administration of K+ containing fluids.
5. CALL A NEPHROLOGIST.

C. Dehydration may be classified as mild, moderate, or severe.
a. Mild - 3% deficit (child/adult), or 5% (neonate), slight thirst, flat fontanelle, good tears, moist mucous membranes, good skin turgor, normal urine output
b. Moderate - 6% deficit (child/adult), or 10% (neonate), irritable, moderate thirst, dry mucous membranes, +/- tears, +/- fontanelle, decreased skin turgor, oliguria
c. Severe - 9% deficit (child/adult), or 15% (neonate), lethargic, hyperirritable, intense thirst, dry mucous membranes, no tears, sunken fontanelle, poor turgor, oliguria or anuria

EXAMPLES OF FLUID MGT. FOR DEHYDRATION ARE LOCATED AT END OF CHAPTER.

D. Composition of commonly used IV fluids:

	(mEq/Liter)					
	Na	K	Cl	HCO3	Ca	CHO
NS, (0.9% NaCl)	154	-	154	-	-	-
1/2 NS, (0.45% NaCl)	77	-	77	-	-	-
1/4 NS, (0.2% NaCl)	34	-	34	-	-	-
3% saline	513	-	513	-	-	-
Ringer's lactate (LR)	130	4	109	28	3	0 - 10
Plasmanate	110	2	50	29	-	-
25% salt poor albumin	100-160	<1	<120	-	-	-
D5W	-	-	-	-	-	5
D10W	-	-	-	-	-	10

E. Composition of fluids lost:

	mEq/L			gm %
FLUID	Na	K	Cl	Protein
Gastric	20-80	5-20	100-150	-
Pancreatic	120-140	5-15	90-120	-
Small intestine	100-140	5-15	90-130	-
Bile	120-140	5-15	80-120	-
Ileostomy	45-135	3-15	20-115	-
Diarrheal	10-90	10-80	10-110	-
Sweat:				
Normal	10-30	3-10	10-35	-
Cystic fibrosis	50-130	5-25	50-110	-
Burns	140	5	110	3-5

F. Neonatal nuances: Newborns cannot concentrate their urine as well as adults and their GFR is lower too so they are more prone to fluid overload. Conversely, their response to ADH and atrial natiuretic

factor is blunted. As previously mentioned, insensible water losses varies with gestational age and size. Fluid management in these patients is even more challenging. In the next section urine Na+ values are mentioned as a diagnostic tool. Due to the newborn's renal physiology, urine lytes, while helpful, are not as precise.

II. HYPONATREMIA (Na^+ < 135 mEq/L)

A. Hypertonic hyponatremia, plasma Osm (POsm) > 295 *****

1. Etiologies:

a. hyperglycemia (1.6 mEq Na^+/L decrease for every 100 mg/dl glucose is increased over 100 mg/dl) eg. DKA

b. mannitol, glycerol

B. Isotonic pseudohyponatremia (POsm = 280-295)

1. Etiologies:

a. hyperproteinemia

b. hyperlipidemia

C. Hypotonic hyponatremia (POsm < 280)

1. hypovolemic (decreased total body Na+, and water), treat with volume expansion and Na+

a. Etiologies:

i. <u>renal losses, (urine [Na+] > 20 mEq/L):</u> diuretic excess, osmotic diuresis, obstructive uropathy, adrenal insufficiency, Fanconi syndrome, pseudohypoaldosteronism, Bartter's syndrome, interstitial nephritis, RTA (bicarb. loss)

ii. <u>GI losses, (urine [Na+] < 20 mEq/L):</u> vomiting, diarrhea, fistula, post-op tubes, gastrocystoplasty

iii. <u>sweat, (urine [Na+] < 20 mEq/L):</u> CF(hyponatremic, hypochloremic, metabolic alkalosis), heat stroke

iv. <u>third space, (urine [Na+] < 20 mEq/L):</u> effusions, ascites, burns, muscle trauma, pancreatitis, peritonitis

2. euvolemic (<u>+</u> total body Na+, increased total body water), treat with water restriction, <u>all show urine [Na+] > 20 mEq/L:</u>

a. Etiologies:

i. <u>water intoxication:</u> IVF's, tap water enema, psychogenic water drinking

ii. <u>excess ADH:</u> SIADH, pain, drugs (MSO_4, cytoxan, vincristine, TCA's, ASA, indomethacin, etc.)

iii. <u>glucocorticoid deficiency</u>

iv. <u>hypothyroidism</u>

v. <u>reset osmostat:</u> CVA, infection (TB), malnutrition

3. hypervolemic, (increased total body Na+, and water), treat with Na+ and water restriction

a. Etiologies:

i. <u>edema forming states, (urine [Na+] < 20 mEq/L):</u> CHF, cirrhosis, nephrotic syndrome, too much free water esp. in neonates

ii. <u>renal failure, (urine [Na+] > 20 mEq/L:</u> acute, chronic

D. Clinical Manifestations

1. Apathy, agitation, anorexia, nausea, vomiting, diarrhea, weakness, altered mental status, coma, hypotension, seizures

E. Treatment

1. Dependent on etiology
2. Euvolemic and hypervolemic states are treated with water restriction, hypervolemic states also may need Na+ restriction
3. Hypovolemic states are treated with correction of shock, saline replacement
4. **Symptomatic hyponatremia**
    a. usually an acute decrease in Na^+ < 120 mEq/L manifested by seizures or coma
    b. symptoms will usually resolve with increase in Na^+ by 3 mEq/L
    c. **mEq Na^+ needed = (desired Na^+ - current Na^+) x 0.6 x wt (kg)**
        i. **3% saline = 513 mEq/L (approximately 0.5 mEq/cc)**
        ii. give over 1-2 hrs (may give as rapidly as over 15 min)
5. Correct hyponatremia no faster than 0.5 mEq/L/hr
    a. osmotic demyelination syndrome may occur with too rapid of correction
6. Monitor Na^+ q 4 hrs until stable trend is noted
7. Correct underlying etiology
8. Correction of chronic hyponatremia should be done very slowly, replace 1/2 of the Na+ deficit in the first day and the remainder over the next 24 - 48 hours. NOTE: this does not apply to fluid deficits where in the treatment of shock fluids may be given more rapidly.

*** Calculated POsm = 2Na + (Glucose/18) + (BUN/2.8);** will be similar to measured osmolality in the absence of alcohols, mannitol, glycerol, or sorbitol

III. HYPERNATREMIA (Na^+ > 150 mEq/L)

A. Euvolemic hypernatremia (+ total body Na+, decreased total body water), urine [Na+] variable, treat with water replacement

1. Etiologies: exchange transfusion (infants < 1500 gms), dialysis (peritoneal or hemo for Na+ >180), iatrogenic (IVF's, or medications), increased skin or respiratory losses, DI (may also have hypovolemic hypernatremia)

B. Hypovolemic hypernatremia (decreased total body Na+, and water), treatment is volume expansion and hypotonic saline

1. Etiologies:
    a. increased extra-renal losses (urine [Na+] < 20 mEq/L), skin: [phototherapy, burns, sweating), or GI: AGE (viral or bacterial), diarrhea, hypertonic enemas, or lack of intake (breastfeeding problems, starvation)

b. renal losses (urine [Na+] > 20 mEq/L), DI (central or nephrogenic), diabetes mellitus, osmotic diuresis, renal dysplasia, obstructive uropathy

C. Hypervolemic hypernatremia (+ total body water, increased total body sodium), urine [Na+] > 20 mEq/L, treatment is use of diuretics, and water replacement or dialysis

1. Etiologies: hypertonic salt solutions, steroid excess, NaHCO3, primary hyperaldosteronism

D. Clinical Manifestations

1. Apathy, anorexia, thirst, vomiting, diarrhea, mental status changes, doughy skin, fever, seizures, tremulousness

E. Treatment

1. Identify etiology and stop the excessive free water loss
2. If Na^+ > 175-180 mEq/L consider rapidly correcting to 165-170 mEq/L
   a. CNS bleeding or venous thrombosis can occur with POsm > 335
   b. death from resp failure occurs when POsm approaches 430
3. Correct hypernatremia to normal range over 48 hrs
   a. rate of decrease should not exceed 0.5-1 mEq/L/hr
   b. rapid correction in hypernatremia may result in rebound cerebral edema
4. Estimate total body fluid deficit clinically or by change in weight
5. Calculate free water deficit
   a. **deficit (L) = 0.6 x wt (kg) x [1- (current Na^+/140)]**
   b. deficit will be replaced with free water (i.e. with D5 1/4 NS, D10 1/4 NS, in children, neonates may need D5, or D10)
   c. free water deficit may be estimated as 4 cc/kg of free water needed for every mEq/L the Na+ is over 145 mEq/L.
6. Remainder of deficit (total body fluid deficit - free water deficit) is replaced similar to isonatremic dehydration with NS
7. Fluid more hypotonic than D2.5 1/4 NS should be avoided as it may lyse cells and lead to cerebral edema
8. Monitor serum sodium q 4 hrs until stable trend
   a. if falling too rapidly add more sodium to IVF's
9. If patient shows initial neurologic improvement but later deteriorates, suspect cerebral edema and treat accordingly

IV. SIADH vs. DI

A. Laboratory data:

	SIADH	DI
1. serum [Na^+]	≤130	≥150
2. urine [Na^+]	≥60	≤40
3. serum osmol	≤275	≥305
4. urine osmol	≥500	≤250
5. urine spec grav	≥1.020	≤1.005
6. urine output (cc/kg/hr)	<1	>3
7. CVP (imprecise)	≥8	≤4

B. Pt's are to be transferred and treated in the ICU with continuous cardiorespiratory monitoring, frequent physical examinations with neurologic status checks, strict recording of fluid intake and output (consider foley catheter), frequent and serial laboratory data, and consider central venous pressure monitoring

C. Causes of SIADH: (**SIADH = inappropriately high secretion of ADH in presence of concentrated urine and euvolemic hyponatremia**)

1. CNS disorders: infection (meningitis, encephalitis, abscess), hypoxia-ischemia, trauma, CVA, tumors, psychosis, Guillain-Barre syndrome, vasculitis, shunt obstructions, cavernous sinus thrombosis
2. Pulmonary disorders: infections (bacterial, tubercular, viral, mycoplasma, fungal), decreased left atrial pressure (pneumothorax, atelectasis, asthma, bronchiolitis, CF)
3. Tumors: bronchogenic carcinoma, adenoca. of the pancreas or duodenum, ureter or prostate carcinoma, thymoma, ALL, lymphoma, lymphosarcoma, mesothelioma
4. Drugs: vasopressin, desmopressin, oxytocin, nicotine, barbiturates, narcotics, carbamazepine, colchicine, isoproterenol, vincristine, vinblastine, amitriptyline, ASA, acetaminophen,, NSAID’s, chlorpropramide, cyclophosphamide

D. Treatment of SIADH:

1. This is usually a euvolemic condition so is treated with fluid restriction (1/2 - 3/4 maintenance). Chronic SIADH that does not respond to fluid restriction may respond to therapy with lithium or demeclocycline which inhibit ADH action.

E. Causes of DI: [**excessive flow of dilute urine due to lack of ADH secretion (central DI) or failure of the kidney to respond to adequate amounts of ADH (nephrogenic DI)**]:

1. Central: primary (familial and non-familial forms), secondary to head trauma, CNS tumors, granulomas (sarcoid, TB, Wegener’s, syphilis), infections (meningitis, encephalitis), histiocytosis, vascular anomalies (aneurysms, thrombosis, hemorrhage, sickle-cell disease, vasculitis)
2. Nephrogenic: primary (X-linked, or autosomal recess.), secondary to renal disease (dysplasia, obstructive uropathy, reflux, cystic disease, nephritis, RTA, Fanconi’s syndrome), secondary to electrolyte imbalance (hypokalemia, hypercalcemia), secondary to systemic diseases with renal involvement (sickle-cell, amyloidosis, sarcoidosis, cystinosis)
3. Drugs that inhibit ADH release: alpha-adrenergic agonists, ethanol, naloxone, diphenylhydantoin, clonidine, carbon monoxide poisoning
4. Drugs inhibiting peripheral action of ADH: lithium, demeclocycline, methoxyflurane, ampho. B, cisplatin, cyclophosphamide, propoxyphene, angiographic dyes, osmotic diuretics (mannitol)
5. Others: septo-optic dysplasia, cleft lip/palate, familial cerebellar ataxia

F. Treatment of DI:

1. This is usually a hypovolemic, hypernatremic situation. Give fluids if shock is present. A water deprivation test diagnoses the condition. For an example on how to treat hypernatremic dehydration see the end of the chapter.

G. If uncertain whether pt. has DI or SIADH, or the pt. is alternating between the two (as may happen with head trauma):

1. A useful fluid regimen is: D5 1/2 NS + 10 - 20 mEq/L of KCl at 400 cc/M^2/day as maintenance fluids, then replace urine output cc for cc with D2.5 1/4 NS q 4 hours. Adding D5 1/4 NS would cause hyperglycemia. May add KCl to the urine replacement fluids by measuring the amount of K+ in the urine and adding this amount to the fluid used for urine replacement. **Make sure the patient does not get too much fluid. Monitor I/O's every few hours !** (see DKA chapter or Acute Tumor Lysis chapters for nomogram and calculation of body surface area)

V. HYPOKALEMIA (K^+ < 3.5 mEq/L)

A. Etiologies:

1. Decreased intake: intractable vomiting, starvation, malnutrition, kwashiorkor, anorexia nervosa, TPN error
2. GI losses: chronic diarrhea (10-80 mEq/L), fistula, laxative abuse, colostomy, nasogastric drainage, ureterosigmoidostomy
3. Renal losses: tubular diseases, Cushing's syndrome, hypomagnesemia, hyperaldosteronism, meds. (aminoglycosides, amphotericin, ticarcillin, NSAID's, diuretics), nephritis, licorice ingestion, Fanconi's syndrome, distal RTA, toluene sniffing, Bartter's syndrome, Gitelman's syndrome, Liddle's syndrome
4. Skin losses: cystic fibrosis, burns
5. Redistribution: metabolic alkalosis from bicarb. etc.(0.1 unit change in pH = 0.6-1.0 mEq/L change in K^+), insulin, $ß_2$ agonists (esp. albuterol), hypothermia
6. Respiratory alkalosis results in a much smaller generation of H^+ and thus minimal K^+ shifts

B. Clinical Manifestations

1. Weakness, paralysis, hyporeflexia, ileus
2. Atrial and ventricular premature contractions, ST depression, flattened T wave, presence of U wave, prolonged Q-U interval
3. Hypokalemia enhances digitalis toxicity

C. Treatment

1. Correct the underlying disorder
2. Oral route preferred: 1-3 mEq/kg/d; correction may take 5-7 days
3. If **K^+< 2.5 replace intravenously 0.5 mEq/kg over 1 hr**
4. Rates of 0.25 - 0.5 mEq/kg/hr are considered safe.
5. IVF concentration of $\leq$ 40 mEq/L considered safe

VI. HYPERKALEMIA (K^+ > 6.5 mEq/L)

A. Etiologies:

1. Impaired renal excretion: renal failure, adrenal insufficiency, obstructive uropathies (type IV RTA)
2. Increased intake: penicillin, K^+ salts, iatrogenic (IVF's), stored PRBC or platelet transfusions
3. Endogenous release: rhabdomyolysis (trauma, heat stroke), burns, tumor lysis syndrome, hemolysis, HUS, succinylcholine, neuromuscular blocking agents (pancuronium)
4. Ionic shift: metabolic acidosis (respiratory acidosis has very little effect on K^+), DKA
5. Drugs: digitalis overdose, glucose, mannitol, Beta-blockers, ACE inhibitors, NSAIDS, K^+ sparing diuretics, chemotherapy
6. Pseudohyperkalemia: hemolyzed specimen, thrombocytosis, leukocytosis

B. Clinical Manifestations

1. Weakness, paralysis, paresthesia
2. Increased PR interval, widened QRS, and peaked T waves leading to ventricular dysrhythmias

C. Treatment

1. **Treat the underlying condition, remove K^+ from IVF's or diet**
2. The acuity of the rise in K may be more important than the absolute level. Chronic K of 6.5 would not produce EKG changes.However, treatment of chronic elevation of K is different than treatment of acute changes.
3. An acute rise in K^+ with a level > 6.5 mEq/L needs immediate treatment. You may not see EKG changes at that moment.
    a. **Calcium chloride (10%)**
        i. 10-25 mg/kg or 0.1-0.25 cc/kg IV over 10 - 30 mins., may repeat dose x 1
        ii. antagonizes neuromuscular effects
        iii. onset is within minutes; duration for 30 min
    b. **Insulin and glucose**
        i. glucose (0.5-1 gm/kg) + insulin (0.1-0.2 units/kg) IV push with subsequent infusion established.
        ii. max 10 units insulin
        iii. monitor glucose during infusion; may repeat if glucose > 80 mg/dl
        iv. redistributes K^+ from ECF to ICF
        v. onset < 30 min; duration several hours
    c. **Sodium bicarbonate**
        i. 1-2 mEq/kg IV over 10-30 minutes
        ii. redistributes K^+ from ECF to ICF
        iii. may be used in conjunction with other treatment options, has little effect if used alone
        iv. onset 3-4 hrs
    d. **Kayexalate** (sodium polystyrene)
        i. 1 gm/kg (max 30 gms) in 70 % sorbitol po, ng, pr

ii. removes potassium from body and substitutes with Na+, follow lytes closely
iii. may repeat q 2-4 hrs
iv. onset 1 hr
v. 1 gm/kg removes approx. 1 mEq/L of K^+ if given po, 1 gm/kg removes approx. 0.5 mEq/L of K+ if given pr

e. Albuterol nebulization
i. dose for children: < 10 kg use 2.5 mg, 10 - 40 kg use 5 mg, > 40 mg use 10 mg (0.3 mg/kg/dose to max. of 10 mg)
ii. in adults, 10-20 mg/dose

f. dialysis if all else fails, call nephrology

VII. HYPOCALCEMIA (Calcium < 8 mg/dl in children, < 7 mg/dl in neonates)

Calcium in general: Total serum calcium is comprised of ionized or unbound calcium (40%), protein bound calcium - principally to albumin (50%), and 10% is complexed. A change in serum albumin of 1 gm/100 ml changes the serum calcium, in the same direction, by 0.8 mg/100 ml. Alkalosis decreases ionized calcium, and acidosis increases the ionized calcium. The ionized portion carries out the metabolic functions. In hypoproteinemic states a lower **total** serum calcium is tolerated as long as the ionized calcium is normal.

A. Etiologies of hypocalcemia:
1. Blood transfusion (chelation with citrate), sepsis, burns, toxic shock syndrome, respiratory alkalosis, hypoparathyroidism, post-neck surgery, DiGeorge Syndrome, vitamin D deficiency, hypomagnesemia, hyperphosphatemia, drugs (aminoglycosides, glucocorticoids, furosemide, phenobarbital, phenytoin, glucagon, calcitonin,mithramycin, bicarb, enemas, phosphates), renal insufficiency, nephrotic syndrome, pancreatitis

B. Clinical Manifestations
1. Muscle twitching, paresthesias, spasms, laryngospasm, seizures, Chvostek's and Trousseau's signs
2. Prolongation of QT interval, T wave inversion, bradycardia

C. Treatment
1. Correct underlying disorder (i.e. correct hypomagnesemia if present)
2. Single dose for cardiac arrest or severe hypocalcemia (Ca^{++}< 0.8 mmol/L) is below, give over 10 minutes:
a. **Calcium chloride (10%) 10-20 mg/kg or 0.1-0.2 cc/kg IV** (acts immediately, requires no metabolism)
b. **Calcium gluconate (10%) 100 mg/kg or 1 cc/kg IV** (may take longer to act as requires liver metabolism)
c. **Calcium gluceptate (22%) 110 mg/kg or 0.5 cc/kg IV** (may take longer to act as requires liver metabolism)
3. Maintenance therapy is with calcium gluconate (10%) 200-500 mg/kg/d given continuously or divided into 6 doses
4. Need for subsequent doses is guided by the ionized calcium

5. Pt's with acute renal failure and/or severe hyperphosphatemia usually tolerate hypocalcemia well and treatment should be reserved for those with CNS irritability

VIII. HYPERCALCEMIA (Calcium > 10.5 mg/dl)

A. Etiologies:

1. Malignancy (metastatic bone), hyperparathyroidism, granulomatous disorders (sarcoidosis, TB., histo., coccidio.), immobilization, milk alkali syndrome, parenteral nutrition, vitamin D or A intoxication, hyperthyroidism, William’s syndrome, thiazide diuretics, subcutaneous fat necrosis, hypophosphatasia, Jansen syndrome, multiple endocrine neoplasias

B. Clinical Manifestations

1. Weakness, anorexia, constipation, vomiting, hypertension, hypotonia
2. Shortened QT interval, U waves

C. Treatment

1. Hypercalcemia with total calcium of > 15 mg/dl or symptomatic is considered an emergency
2. Identify and treat the underlying cause
3. Saline hydration
   a. 200-250 ml/kg/d
   b. increases urinary excretion
   c. not to be used in CHF or renal failure
4. Furosemide
   a. 1-2 mg/kg/dose every 4 hrs
   b. this calciuretic agent enhances excretion
   c. fluid overload, hypokalemia and hypomagnesemia may result from aggressive use of IVF's and furosemide, follow lytes closely
   d. ionized calcium begins to decline within 4 hrs of therapy
5. Calcitonin
   a. may be necessary with severe or persistent hypercalcemia
   b. 4 units/kg q 12 hrs
   c. decrease in ionized calcium is generally not sustained
   d. inhibits bone resorption
6. Steroids
   a. used in hypercalcemia due to malignancy, granulomatous disease or vit. D toxicity
   b. 10 mg/kg/d of hydrocortisone equivalent
   c. onset of action is in 1-2 days
   d. decreases intestinal calcium absorption
7. Mithramycin
   a. effects are most significant in hyperparathyroidism or malignancy
   b. 15-25 mcg/kg IV over 3-8 hrs
   c. decrease in calcium begins within 24 hrs and peaks at 3-4 days
   d. decreases bone resorption
8. Hemodialysis or peritoneal dialysis with calcium free dialysate is effective

9. Binding of serum calcium with chelators and phosphate may be effective but has been associated with soft tissue deposition of calcium complexes and should be used as a last resort; (phosphate 0.15 - 0.3 mmol/kg IV over 6 hrs)

IX. HYPOPHOSPHATEMIA (Phosphorus < 2.5 mg/dl)

A. Etiologies:

1. Decreased tubular transport: hyperparathyroidism
2. Redistribution: alkalosis, insulin, Beta-agonists, increased glycolysis
3. Inadequate intake: patients on TPN, breast fed premies
4. GI losses: malabsorbtion, NG suctioning, phosphate binding agents, enteric fistulas
5. Renal losses: diuretics, tubular defects, DKA, Fanconi's
6. Increased utilization: refeeding after severe protein-calorie malnutrition
7. Other: hypothermia, Cushing's disease, burns, vit. D deficiency, gentamicin, thyrotoxicosis

B. Clinical Manifestations

1. Cardiomyopathy, respiratory muscle weakness, red blood cell dysfunction (hemolysis, decreased 2,3 DPG leading to tissue hypoxia), leukocyte dysfunction, thrombocytopenia, rhabdomyolysis, altered mental status

C. Treatment

1. Severe (phos < 1 mg/dl) or symptomatic hypophosphatemia:
   a. **0.15-0.33 mmol/kg/dose over 6 hrs**
   b. Potassium phosphate: 4.4 mEq K^+/ml and 3.0 mmol Phos./ml
   c. Sodium phosphate: 4.0 mEq Na^+/ml and 3.0 mmol Phos./ml
2. Maintenance requirements are 0.1-0.2 mmol/kg/d
3. Adverse effects of administration include hypocalcemia (especially if infused faster than over 6 hrs), metastatic calcification, hypotension, hyperkalemia or hypernatremia (depending on preparation used)

X. HYPERPHOSPHATEMIA (Phos. > 6.8 mg/dl in children, > 4.5 mg/dl in adults, > 9 mg/dl in neonates)

A. Etiologies:

1. Result of renal failure, overdose of phosphate enemas, tumor lysis, IV boluses of phosphate, hypoparathyroidism, rhabdomyolysis, severe hemolysis, endosteal hyperostosis, vit. D intoxification

B. Clinical Manifestations

1. Hypocalcemia, calcium phosphate soft tissue deposits, seizures, coma and death

C. Treatment

1. For life-threatening hyperphosphatemia: fluid administration to enhance excretion, IV calcium and dialysis

XI. HYPOMAGNESEMIA (Mg^{++} < 1.5 mEq/L)

A. Etiologies:

1. Nutritional deficiencies: starvation, TPN, prematurity, IUGR
2. GI losses: malabsorption, chronic diarrhea, NG suctioning
3. Renal losses: diuretics (furosemide), drug induced tubular dysfunction (aminoglycosides, ticarcillin, ampho B, cisplatin, cyclosporin, carboplatin, ifosphamide), Fanconi syndrome, pyelo, glomerulonephritis, Bartter's syndrome, Gitelman syndrome
4. Endocrine and metabolic: hyperaldosteronism, DKA, hyperparathyroidism, hyperthryoidism, hypophosphatemia, insulin,
5. Other: sepsis, burns, lipolysis, hypothermia, hepatobiliary disease

B. Clinical Manifestations

1. Apathy, delirium, coma, tremors, tetany, seizures, CHF
2. Prolonged PR, QT and QRS intervals; T wave inversion, ventricular arrhythmias

C. Treatment

1. Supplement diet with magnesium if serum level is > 1.2 mEq/L
2. For serum level < 1.2 mEq/L
   a. **Magnesium sulfate (10%) 25-50 mg/kg/dose**
   b. Should be given no more rapidly than 150 mg/min although can give 25 mg/min in emergent situations
   c. Rapid administration may cause hypotension, flushing, respiratory depression, and arrhythmias

XII. HYPERMAGNESEMIA (Mg^{++} > 2.0 mEq/L)

A. Etiologies:

1. Rare in the PICU except in children with renal failure or iatrogenic
2. Others: maternal magnesium therapy, magnesium laxatives, antacids, or enemas, lithium

B. Clinical Manifestations

1. Hyporeflexia, respiratory depression, coma, hypotension
2. Prolonged PR, QRS, and ST intervals, bradycardia
3. Symptoms seen with levels > 5 mEq/L

C. Treatment

1. Discontinuation of magnesium, administer normal saline and furosemide, IV calcium to reverse the neuromuscular and cardiovascular effects of magnesium

Dehydration examples follow.

XIII. **Dehydration examples:** (refer to maintenance fluid and electrolyte requirements at beginning of this chapter.) This is one reasonable approach, others exist as well. A nephrology consult should be considered.

A. Phased approach

1. PHASE 1 - Emergency restoration of volume in first 1 - 2 hours, 10 - 20 cc/kg of isotonic saline, LR, blood, etc. as rapidly as situation dictates (more may be needed in shock)

2. PHASE 2 - Replacement of 1/2 of the fluid loss (deficit and maintenance) in first 8 hours
3. PHASE 3 - Replacement of remaining 1/2 of the fluid loss (maintenance and remaining deficit) in next 16 hours. Replacement of potassium **after** voids with max. of 40 mEq/L. 1/2 of K+ replaced in first day.

B. **Example:** One year old child who weighs 10 kg on Monday and 9 kg on Thursday when he sees you.

Assume that in cases of rapid dehydration (< 2 days) the ratio of ECF to ICF deficits is 75%/25%.

In cases of moderately rapid dehydration (2 - 7 days) the ratio of ECF/ICF deficits is 60%/40%. This applies to our case.

In cases of slow dehydration (> 7 days) the ratio of ECF/ICF deficits is 50%/50%.

Most of our Na+ is located extracellularly at a concentration of 140 mEq/L, while most of our K+ is intracellular at 150 mEq/L.

1. **Isonatremic dehydration**:(You send lytes, the child has a Na+ of 135)

Total body fluid deficit = 10 kg - 9 kg = 1 kg or 1 liter.
ECF water deficit = 60% X 1 liter = 600 cc.
ICF water deficit = 40% X 1 liter = 400 cc.
ECF Na+ = 60% X 140 mEq/L = 84 mEq.
ICF K+ = 40% X 150 mEq/L = 60 mEq.

Fluids and electrolytes for this patient:

	Water (ml)	Na+ (mEq)	K+ (mEq)
Maintenance/day	1000	30 #	20 !
ECF water deficit	600	84	-
ICF water deficit	400	-	30 *
Totals	2000	114	50
per liter		57	25

maintenance is 3 mEq/100 cc's
! maintenance is 2 mEq/100 cc's
***** 1/2 of deficit replaced in first day
SO:
Phase 1 (first hour): 20 cc/kg of NS = 200 cc NS (= 200 cc water, 31 mEq Na+)

	Water (cc)	Na+ (mEq)	K+ (mEq)
Total	2000	114	not replaced
- phase 1	200	31	until voids
New total	1800	83	NA
per liter		46	

Phase 2 (hours 2 - 8): we will replace 1/2 of the fluid loss in the next 7 hours or 900 cc's in 7 hours which equals 129 cc/hr. We wish to add Na+ in a concentration of 46 mEq's/L which is roughly 1/3 NS so we can use D5, 1/3 NS at 129 cc/hr.

Phase 3 (hours 9 - 24): the patient voids X 2. We will replace the remaining fluid loss and add K+ now. This equates to 900 cc's over 16 hours of D5, 1/3 NS at 56 cc/hr. We will add potassium with a max. of 40 mEq/L and follow the electrolytes closely. As the patient has a 25 mEq/L K+ deficit and we are replacing 900 cc's (or roughly 1 Liter) of fluid we may choose to add 25 mEq/L of KCl. The IMPORTANT POINT is to follow the electrolytes closely!

2. **Hyponatremic dehydration** example with the same child and weight loss (he's having a bad year): The Na+ is measured and is 120.

Sodium deficit = (desired Na+ - current Na+) X 0.6 X weight (kg)
= (135 - 120) X 0.6 X 10
= 90 mEq

Fluids and electrolytes for this patient:

	Water (ml)	Na+ (mEq)	K+ (mEq)
Maintenance/day	1000	30	20
ECF water deficit	600	84	-
ICF water deficit	400	-	30
Na+ deficit	-	90	-
Totals	2000	204	50
per liter		102	25

Phase 1 (first hour): 20 cc/kg NS = 200 cc NS (= 200 cc water, 31 mEq Na+)

Phase 2 (hours 2 - 8):

	Water (cc)	Na+ (mEq)	K+ (mEq)
Total	2000	204	not replaced
- phase 1	200	31	until voids
New total	1800	173	NA
per liter		96	

We will replace 1/2 of the fluid loss (900 cc's) in the next 7 hours and we desire to add a Na+ concentration of 96 mEq's/L which is roughly equal to 2/3 NS. So, you might use D5, 2/3 NS at 129 cc/hr.

Phase 3 (hours 9 - 24): the patient voids X 2. We will replace the remaining fluid loss and add K+ now. This equates to 900 cc's over 16 hours of D5, 2/3 NS at 56 cc/hr. We will add potassium with a max. of 40 mEq/L and follow the electrolytes closely. As the patient has a 25 mEq/L K+ deficit and we are replacing 900 cc's (or roughly 1 Liter) of fluid we may choose to add 25 mEq/L of KCl. The IMPORTANT POINT, once again, is to follow the electrolytes closely!

3. **Hypernatremic dehydration** example with the same 10 kg ---> 9 kg weight loss from Monday to Thursday. Serum sodium is 180. This gets your attention.

Phase 1 (first hour): 20 cc/kg NS (or LR) over one hour which is 200 cc's water, 31 mEq's Na+. The next Na+ is 175.

Fluids and electrolytes for this patient:

Calculate maintenance fluids for the next **48 hours** and the free water deficit.

Free water deficit (Liters) =

0.6 X weight (kg) X [1 - (current Na+/140)]
= 0.6 X 10 X [1 - (175/140)
= - 1.5 liters , which means this patient is down 1500 cc's of free water.

Maintenance/**day**	Water (ml) 1000 but we want the requirement for 48 hrs (2 days) so:
	Water (ml)
Maintenance for 48 hrs	2000
Free water deficit	1500
Total	3500

We will replace this quantity over 48 hours so 3500 cc/48 hrs = 73 cc/hr.

The question in this case is what kind of fluids should we use ?
Never use a solution with less sodium than 1/4 NS as cell lysis and cerebral edema may occur!! We may initially use D5, 1/2 NS at 73 cc/hr for 4 hours and reassess our urine output and Na+ trend. Now, what about potassium?

	K+ (mEq)
Maintenance/day	20
ICF (as before)	30
Total	50
per liter	25

Once urine output is established we may add K+ (as Kphos or KCl) depending on the potassium, chloride and phosphorus status with a max. of 40 mEq's/L. We are down 50 mEq's of K+, so we may wish to add 20 mEq/L of K+ and reassess. Our IV rate will give us 1752 cc's/day (73 cc/hr X 24 hours) so at 20 mEq/L of K+ we will supply 35 mEq's of K+ in 24 hours, and replenish the full 50 mEq deficit in roughly 34 hours. This naturally depends on kidney function and hemodynamic status.

The IMPORTANT POINT is to reassess your Na+ often (q 4 hours to begin). If the Na+ is dropping too rapidly (>0.5 - 1 mEq/L/hr), decrease the infusion rate and/or increase the Na+ concentration. A useful guide is to try to drop the Na+ by 10 mEq/day.

EPIGLOTTITIS

I. Introduction

A. Definition: A life threatening bacterial infection of the epiglottis and aryepiglottic folds.

B. Etiology

1. Before the era of the HIB vaccine, Hemophilus influenza type B was the etiologic agent in > 75% of cases.

2. Group A Strep, Pneumococcus, S. aureus, H. parainfluenza - other etiologies.

C. Epidemiology

1. No seasonal predominance.

2. Most common between 2-6 y/o (mean = 40 months).

II. Pathophysiology

A. Pharynx often colonized with potentially pathogenic organisms.

B. Bacteria penetrate the mucosal barrier and invade the bloodstream.

C. Focal infection may occur at the epiglottis and surrounding structures causing inflammatory edema.

D. Edema leads to reduction in the caliber of the airway, which leads to turbulent air flow on inspiration and stridor.

III. Clinical Manifestations

A. Signs and symptoms

1. Abrupt onset - usual duration of illness before hospitalization < 24 hours.

2. Stridor and labored respirations.

3. Febrile - often > 103°F.

4. Sore throat.

5. Aphonia, hoarseness, muffled voice

6. Anxious appearance.

7. Prefer sitting position, with jaw thrust forward.

8. Cyanosis in later stages.

9. Drooling.

10. Tachycardia.

11. Tachypnea (but rarely > 40 b/min).

12. Retractions.

B. Visualization of the epiglottis is hazardous and should not be done in the child with suspected epiglottitis!! Agitating the child may precipitate complete airway obstruction!! Do **NOT** use a tongue depressor on these patients !!

C. Labs/X-ray - may be obtained only after airway is secured!

1. CBC - elevated WBC with bandemia.

2. Blood cultures - positive 80 - 90%.

3. Culture of epiglottis - positive in about 50%. Caution - make sure the lab does not set this up as a routine throat CX.

4. Lateral neck X-rays, usually time consuming, are often non-diagnostic and place the child in an area of the hospital where emergency airway care is suboptimal. If epiglottitis is suspected, radiographs are absolutely contraindicated until airway stable.

(1) Swollen epiglottis - assumes a configuration that is convex on both sides, commonly called the "thumb sign".

(2) Thickened aryepiglottic folds.

(3) Obliteration of the vallecula.

5. Meningitis occurs uncommonly (2-3% of cases). Consider LP in the OR in children with signs and symptoms of meningitis.

D. Complications

1. Sudden respiratory obstruction - most serious and potentially fatal.

2. Extraepiglottic spread of infection - eg. lungs, pericardium, soft tissues, synovium, and meninges.

3. Spread to susceptible contacts.

IV. Management Protocol - should be followed in ANY situation where the airway is acutely at risk or suspicion exists for impending airway compromise.

A. True Emergency - STAT anesthesia (Beepers #1023, #1085), ENT (Beeper #2822; Ext 5623/5605) and Pediatrics (Beeper #1030, ext 7715/7835) consults. Arrange for O.R. (ext 7467/5655). If O.R. is not immediately

available, patient will be taken to the PICU, or the recovery room if PICU bedspace is not available.

B. Anesthesia and ENT attending physicians to remain with patient at all times - be prepared to provide an emergency airway and resuscitate at any time.

C. Physical exam WILL NOT include attempts at visualization of pharynx by depressing the tongue!!

D. Keep child calm and avoid agitation. Parents should be allowed to stay with the child until just before intubation. Don't attempt to lay the patient down. The child may determine the position he/she is most comfortable in.

E. Oxygen may be given by mask, but not at expense of agitating patient. Usually child will allow parents to give O_2.

F. May see improvement with racemic epinephrine, therefore, an improvement with racemic DOES NOT always imply croup.

G. Prepare the child for transport.

1. Use gurney if possible.
2. Oxygen tank full and open.
3. Ambubag with appropriate size mask.
4. Laryngoscope.
5. Appropriate size ETT and smaller sizes.
6. Trach set.
7. Portable suction.
8. Atropine 0.02 mg/kg (min = 0.1 mg, max = 1.0 mg).
9. Succinylcholine (2 mg/kg) for use by anesthesia.
10. Culture swabs, blood culture bottles, IV equipment, LP tray (if needed), antibiotics.

H. Transport the child and parents only when a physician experienced in intubation is present with proper equipment.

I. Controlled intubation should occur by anesthesia in the O.R. with ENT in attendance in case of need for emergency bronchoscopy or tracheostomy.

J. Initial intubation should be orally, using an ET tube 1-2 sizes smaller than usual. Only after intubation should IV be started and bloods drawn for lab studies and cultures, LP etc.

K. Recommended antibiotic - Cefuroxime 150 mg/kg/day IV divided q 8 hours, Cefotaxime 200 mg/kg/day IV divided q 8 hours, or Ceftriaxone 100 mg/kg/day IV divided every 12-24 hours. First dose STAT in OR. If bacterial tracheitis is suspected add better gram ⊕ coverage - eg. Nafcillin, 150 mg/kg/day, IV, divided q 6 hours . **Respiratory isolation for 24 hours after initiation of antibiotics should be ordered.**

L. After stabilization, consider changing ETT to a more secure nasotracheal tube. Document position with CXR!

M. Humidified air, with or without supplemental 02 as required. ABG's as needed. Usually an A-line is required to adequately assess course.

N. CPAP if evidence of atelectasis. Suctioning Q1-2 hours and PRN.

O. Sedation - to prevent accidental extubation - Versed (0.1 mg/kg) or Morphine (0.1 mg/kg). Remember Morphine causes histamine release. Versed is relatively short acting. Sedation may be given as boluses or continuous infusion.

P. Soft restraints to prevent extubation.

Q. Sometimes necessary to sedate, paralyze and mechanically ventilate to maintain airway. Versed 0.01 mg/kg PRN or 0.1 mg/kg/hr and Vecuronium 0.1 mg/kg Q 1-3 hours prn or 0.1 mg/kg/hr is one acceptable regimen.

R. Extubate after objective evidence of improvement, i.e. development of an air leak. Usually 24-36 hours after intubation. Best done in O.R. with ENT present.

S. Observe in ICU for 12-24 hours post extubation. Should receive 7-10 day course of antibiotics (not necessarily all IV).

T. Robinul to decreased secretions as needed. Antisecretory dose is 0.004-0.010 mg/kg/dose IV q 4-8 hrs.

V. Prophylaxis of contacts of patient with H.influ. type B epiglottitis.

A. Individuals residing with the index case or individuals who spent 4 or more hours with the index case for at least 5 of the 7 days prior to admission need prophylaxis, irrespective of age, if household contains:

1. One incompletely vaccinated child < 48 months.

2. Child < 12 months.

3. Immunocompromised child of any age.

B. In families receiving prophylaxis, the index case also requires prophylaxis.

C. Use Rifampin 20 mg/kg (max = 600 mg) q d x 4 days.

FEBRILE SEIZURES

I. Introduction:

A. Definition: A seizure associated with fever in a child age 6 mos - 6 years without evidence of other cause (no evidence of trauma, CNS infection or metabolic cause, and no history of epilepsy).

B. Incidence: 3 -5% of all children

1. Recurrence Risk:

a. 30% will have a second seizure

b. 15% will have a third seizure

c. 9% will have more than three seizures

d. 70% of the recurrences are within one year, 90% within two years.

C. **Simple Febrile Seizure, Definition**:

1. Single event

2. Generalized, without focal onset.

3. Duration less than 15 minutes

4. No history of epilepsy

5. Patient ages 6 mos - 6 years

6. Risk factors for future epilepsy:

a. "Complex" febrile seizures are defined as lasting greater than 15 min., by being focal, or by being multiple seizures.

b. Family history of an afebrile seizure.

c. Neurologically abnormal before seizure.

d. When two or more factors are present, 10% will develop epilepsy.

II. Work Up:

A. History (as per status epilepticus).

B. Careful general exam, neuro exam - identify source of fever.

C. Lab

1. Lytes, BUN, creatinine, glucose, Ca, Mg

2. CBC with diff

3. Tox screen as indicated by history

4. Blood cultures if toxic appearing

5. Lumbar Puncture

   a. If less than 16 months and first seizure

   b. If greater than 16 mos and meningitis suspected clinically

   c. For new onset febrile seizure consider an LP. If child looks normal and F.S. was simple, an LP is probably not necessary.

6. EEG as an outpatient if "complex" febrile seizure

III. Hospitalize if:

A. Abnormal Exam

B. Abnormal Labs

C. Parental Concerns - it is not unusual to seize again in the first 24 hours

D. Pediatrician concerns - observation if you are not comfortable with patient.

IV. Treatment: **NO TREATMENT IS INDICATED FOR THE VAST MAJORITY OF FEBRILE SEIZURES**

A. Decreases the risk of recurrent febrile seizures.

B. Does not reduce the risk of epilepsy.

C. Only for more than 2-3 seizures, early age child.

D. Phenobarbital 4-5 mg/kg/day PO divided b.i.d.

   - may cause behavior changes (50% - hyperactivity).

   - discontinue (taper) medication after 1-2 years if no further seizures.

E. Phenytoin is ineffective for febrile seizures.

F. For motivated parents, prophylaxis at the time of a febrile illness is an effective alternative. Use Lorazepam 1 - 2 mg PO or PR q 8 hours until the child is without fever for 24 hours or for 3 days, whichever is less. **YOU MUST STRESS THAT SEIZURE PREVENTION MEDICATION IN NO WAY SUBSTITUTES FOR THE NECESSARY MEDICAL EVALUATION FOR FEVER !!**

GI BLEEDS

I. Initial Management

A. Clinical Assessment

1. Hemodynamic status

* Infants and children compensate well until they become critical. Indications of the hemorrhage extent may be subtle.

a. BP - not a reliable indicator of shock

b. Pulse

c. Orthostatic changes - see below

B. If hemodynamic compromise exists, immediate resuscitation:

1. Intravenous access - Class I (bleed that has stopped) one intravenous line. Class II-IV (active bleeding) two intravenous lines. Infant 20 gauge IV, Child 18 gauge IV, Adolescent 16 gauge IV.

a. Class I-IV, defined in table at end of chapter. These classes are defined for adults by the American College of Surgeons and ATLS but are generally applicable for children as well.

2. Fluids (LR or NS bolus).

a. 10 cc/kg/10 min until VS's normalized.

3. Blood

a. If needed urgently - O neg. blood

b. Warm blood if > 20 cc/kg needed.

c. Estimate 5 cc/kg will increase Hgb by 1, & HCT by 3.

d. Transfuse plt if < 50 K.

e. FFP if > 40 cc/kg of blood required.

C. Acid suppression:

1. Zantac (Ranitidine) 2-3 mg/kg/d IV, divided every 6 hours.

2. Carafate when taking PO (if UGI bleed).

D. Vasoactive agents.

1.**Somatostatin** produces a decrease in splanchnic flow by inhibiting the release of vasodilatory gastrointestinal peptides such as glucagon, vasoactive intestinal peptide, calcitonin gene-related peptide, and substance P. In adults the patient is bolused with 250 mcg followed by a continuous infusion of 250 mcg/hr. Bolus injections have been repeated for bleeding. **Octreotide** is a synthetic analogue of somatostatin, and has a longer half life. It has become the agent of choice when available. In adults this analogue has been used as a continuous infusion at 50 mcg/hour.Consult your GI physician for pediatric dosing.

2. Vasopressin 0.3 units/kg bolus followed by 0.2 units/1.73M^2/min. This can be increased by 0.1 units/1.73M^2/min every hour up to 0.6 units/1.73M^2/min maximum. Nitroglycerin infusion is often used in conjunction with vasopressin. Nitroglycerin infusion reduces the adverse hemodynamic effects of vasopressin and lowers portal venous resistance, futher decreasing portal pressures.The Nitroglycerin infusion dose starts at 0.5-1 mcg/kg/min and titrated upward to bring blood presssures into the normal range for patients age.

E. Vitamin K IM if previous history of liver disease (1 mg/year of age, IM or IV). Vit. K must be given slowly (over 1 hr.) if given IV.

F. Once stabilized, evaluation of bleeding episode

II. Evaluation of Bleeding Episode

A. History and Physical

1. Estimate of amount of bleeding

2. Color of bleeding

a. Bright red - rapid bleeding

b. Coffee ground - slower rate of bleeding/blood acted on by gastric acid

c. Hematochezia

- bright red blood per rectum

- most often lower GI bleed (only 10% massive UGI bleed)

d. Melena

- tarry stools, foul smelling

- bleed in upper GI/small bowel or right colon if partially obstructed.

- > 200 cc in older children is common blood loss.

B. Duration of bleeding

C. Circumstances surrounding bleeding

1. Febrile illness

2. Vomiting/abdominal distention/abdominal pain.

3. Ingestions

PMH: Previous abdominal surgery, hospitalizations, umbilical vein catheterization

D. FH: Peptic ulcer disease, liver disease, inflammatory bowel disease

E. ROS

F. PE

1. VS

2. Orthostatic changes - "tilt test": inc in pulse of > 20 from supine to sitting or standing or a dec in systolic BP > 10 from supine to sitting or standing is a positive tilt and suggests significant volume loss.

3. Signs or symptoms of chronic liver disease (coagulopathy, protein loss, jaundice, FTT etc.)

4. Hepatosplenomegaly, liver consistency and contour

5. Any cutaneous hemangiomas, palmar erythema, telangiectasias

6. HEENT: examine mouth, nose, throat for evidence of epistaxis, bleeding from tonsils, dental bleeding.

a. Insert NG tube and guaiac aspirate with gastrocult.

i. establishes level of bleeding ie. negative aspirate means bleeding distal to the ligament of Treitz.

ii. enables clearance of blood and clots.

iii. eliminates the possibility of substance mimicking blood such as:

(1). Bright red blood:

(a). food coloring
(b). beets
(c). jello
(d). kool aid
(e). antibiotics
(f). pyronium pamoate

(2). Melena:

(a). bismuth
(b). iron
(c). spinach
(d). blueberries
(e). grapes
(f). licorice
(g). Serratia marcesans in diapers

4. Rectal exam and guaiac

a. fissures

b. polyps

III. UGI bleed (NG aspirate guaiac positive) on gastroccult. (Gastric contents should be tested with gastroccult because of false negatives with hemoccult in acidic environment).

A. Resuscitate (ie treat for shock as needed). Transfuse if HCT < 25 with active bleeding, < 30 in patients with high oxygen requirements.

B. Labs:

1. CBC with platelets

2. Type and cross - CMV negative if patient is possible transplant candidate.

3. PT/PTT, fibrinogen

4. LFT's, Lytes, TP, Alb, Ca++, PO4--

5. UA

C. Saline Lavage

1. Elevate HOB

2. Insert NG tube: use Replogle (holes are located in distal portion of tube whereas a Sump tube has holes all along distal portion so some of the holes may be located in the esophagus in a child and hamper the lavage)

a. Infants - 10 F Replogle

b. 12 F in small children

c. 14-16 F in older children

3. Verify position by injection of 10 cc of air and auscultating over stomach

4. Place patient on left side so tube is in most dependent portion of stomach.

5. Infuse saline in aliquots using 60 cc catheter tipped syringe, ROOM temperature - **not** iced saline (causes hypothermia, coagulopathy).

a. 30 cc for infants

b. 60 cc for children

5. Allow to stand 2-3 minutes

6. Remove infusate

7. Repeat until return is clear or only slightly pink

8. Leave tube in place to gravity to allow reassessment of bleeding

E. Endoscopy

1. Emergent if life-threatening bleed (ie. copious bleeding requiring frequent transfusions or hemodynamic instability not responding to initial medical management)

2. Elective, 12-24 hrs after bleed for:

a. Hct < 30

b. Requiring continued transfusions

c. Hx of previous unexplained GI bleed

d. Should not have barium contrast for 24 hours prior to endoscopy.

F. Further W/U depends on specific etiology

G. In the newborn period, swallowed maternal blood is frequently mistaken for hemorrhage which can be ruled out by the Apt Test:

1. Mix one part guaiac positive stool or NG aspirate with 5 parts H20

2. Centrifuge

3. Separate supernatant and to 4 cc supernatant add 1 cc 1% NaOH

4. Maternal blood with adult Hgb remains yellow/brown while baby's blood with fetal Hgb turns pink

IV. Lower GI bleed (guaiac pos stool, neg NG aspirate)

A. Resuscitate (ie. treat for shock as needed)

B. Labs:

1. Previously mentioned labs

2. WBC/eos

3. Reducing substances

4. Culture for enteric pathogens

C. Acute Abdominal Series (R/O Obstruction)

D. Exploratory Laparotomy considered in cases of unremitting lower GI bleed requiring transfusion/resuscitation

E. Further W/U depends on specific etiology and may include:

1. Proctosigmoidoscopy

2. Meckel's scan

3. BE

4. UGI with small bowel follow through

V. Age Related Etiologies

A. Newborn:

1. Ingested maternal blood

2. Infectious diarrhea

3. Necrotizing enterocolitis

4. Vit K deficiency

5. Hirschsprung enterocolitis

B. Infancy to 2 yrs

1. Anal fissure

2. Infectious diarrhea

3. Meckel’s diverticulum

4. Intussusception

5. Milk colitis

6. Polyp

7. Esophageal Varices

C. 2 yrs to Preschool

1. Infectious diarrhea
2. Polyp
3. Anal fissure
4. Meckel’s diverticulum
5. Intussusception
6. Hemolytic uremic syndrome
7. Henoch-Schonlein purpura

D. Preschool to adolescence

1. Inflammatory bowel disease
2. Infectious diarrhea
3. Peptic ulcer disease
4. Esophageal varices
5. Polyp

HYPERTENSIVE EMERGENCIES

I. Introduction

A. Hypertensive emergencies exist when marked blood pressure elevation becomes a threat to function of vital organs including brain, heart, and kidneys. A major factor determining the outcome of children with hypertensive emergencies is the speed of initiating an effective plan to lower BP.

B. The objective of emergency treatment is prevention of hypertension-related adverse events (e.g., stroke, cardiac failure, encephalopathy). This usually requires only a modest reduction in BP. Attempts to rapidly achieve normal BP are contraindicated. In patients with chronic hypertension, a rapid lowering BP to “normal” can decrease needed perfusion to the heart and brain resulting in ischemia. For this reason, in individuals with longstanding hypertension or hypertension of unknown duration, an initial BP reduction (10-20%) followed by gradual normalization of BP is recommended. In acute hypertension, the therapy should be directed towards bringing the BP to normal values.

II. Causes of Pediatric Hypertensive Emergencies (partial listing)

A. Renal disease (80%)

1. Nephritides

a. Henoch-Schonlein Purpura

b. Postinfectious glomerulonephritis

c. Systemic lupus nephritis

d. Rapidly progressive glomerulonephritis

2. Vascular

a. Hemolytic-uremic syndrome

b. Renal artery stenosis & thrombosis*

c. Renal vein thrombosis*

d. Sickle Cell nephropathy

3. Congenital Malformations

a. Polycystic kidney disease*

b. Tuberous sclerosis*

c. Hydronephrosis*

d. Renal hypoplasia*

e. Obstructive uropathy*

4. Miscellaneous

a. Iatrogenic fluid overload*

b. Renal failure with fluid overload

c. Renal transplant (rejection or renal artery stenosis)

d. Reflux nephropathy

e. Renal tumors (Wilm’s, Tuberous sclerosis, congenital nephroma)*, perirenal masses*

B. Endocrine

a. Pheochromocytoma*

b. Congenital adrenal hyperplasia*

c. Hyperthyroidism

d. Neuroblastoma*

e. Cushings Syndrome*

f. Renin-secreting tumor

C. Neurological

a. Meningoencephalitis

b. Tumor

c. Guillain-Barre syndrome

d. Dysautonomia

e. Increased intracranial pressure (trauma)

f. Seizures*

D. Cardiovascular

a. Aortic thrombosis*

b. Aortic coarctation*

c. Aortic insufficiency

d. Subacute bacterial endocarditis

e. Takayasu's arteritis

E. Others

Leg traction
Burns
Neurofibromatosis
Williams Syndrome
ECMO*
Chronic lung disease*
Closure of an abdominal wall defect*
Drugs*: including corticosteroids, NSAIDs, cocaine, amphetamines, birth control pills, theophylline, pancuronium, phenylephrine

"Rebound hypertension" -occurs with abrupt withdrawl of clonidine, angiotensin converting enzyme inhibitors or beta-blockers

**** are causes of neonatal hypertensive emergencies.***

III. Immediate Evaluation (see normal BP tables at end of Chapter).

A. Admit, preferably to ICU-the need for an ICU is based on the severity of the HTN and if signs and symptoms are present.

B. Obtain a concise medical history, stressing renal, endocrine, cardiac and neurological systems, current medications or ingestions and hydration.

C. Perform physical examination, emphasizing: cardiovascular, genitourinary, dermatologic and neurologic systems. Insure that blood pressure measurement in all extremities is correctly performed with correct sized cuff and that pain and anxiety are minimized.

D. Gain vascular access for therapy.

E. Obtain baseline studies including: CBC and diff, BUN, creatinine, serum electrolytes, calcium, glucose, phosphorus, triglycerides, cholestrol, lipoprotein analysis (HDL, LDL), ESR, catecholamines, plasma renin activity (PRA), aldosterone, urinalysis and urine culture.
Note:
Catecholamines and PRA should be placed immediately on ice. These levels should be obtained before pharmacologic therapy is administered when possible.

F. Institute appropriate therapy based on initial assessment.

G. Request CXR, renal ultrasound, cardiac echocardiogram and depending on neurological status head CT. Obtain EKG with rhythm strip.

H. Consider intra-arterial continuous blood pressure monitoring. (A-line). Continuous parental therapy for BP mgt. should not be administered without continuous monitoring.

I. Provide adequate control of pain and anxiety.

IV. Treatment

A. General

1. Correct obvious causes (e.g. umbilical catheter, ICP, pain)

2. Start antihypertensive therapy. For severe HTN parenteral medications should be considered

3. May want to add further diagnostic labs:

a. 24 hr urine for VMA, catecholamines, and metanephrines (pheo, neuroblastoma), 17 ketosteroids, 17 hydroxycorticosteroids, aldosterone, pregnanetriol.

b. Serum aldosterone if not done already.

c. IVP, renal angiography, renal scan (anatomic pathology)

d. Renal biopsy

e. Thyroid function studies

4. When BP is under control with parenteral medications, gradually switch to oral antihypertensives

V. Therapy

A. Nifedipine - Most commonly used medication for asymptomatic children who can take medications. Calcium channel blocker that decreases BP by arteriolar vasodilatation. Nifedipine lowers both systolic and diastolic BP in children by 30-40 mm Hg within 30 minutes. Nifedipine comes in a gelatin capsule with 10 mg equal to 0.34 cc of solution. In older children the capsule should be chewed and swallowed to obtain optimal results. In small children the solution should be aspirated from the capsule and administered orally. (Minimal sublingual absorption).

1. Generic name: Procardia

2. Formulation: 10 or 20 mg capsule

3. Dose and route: 0.25 - 0.5 mg/kg, PO

4. Interval: q 30 min. X 1, then q 3 - 4 hours

5. Onset of action: 30 min.

6. Duration of action: 1 - 4 hours

7. Major acute problems: hypotension, dysrhythmias

B. Hydralazine-Vasodilator, although it is less potent than other vasodilatory drugs it can be effective. No longer commercially available in IV preparation.

1. Generic name: Apresoline

2. Formulation: 20 mg/ml

3. Dose and route: 0.1 - 0.5 mg/kg/dose; max. dose: 20 mg, IV or IM

4. Interval: repeat q 20 min. if no response, usual interval q 3 - 6 hours

5. Onset of action: 5 - 20 min.

6. Duration of action: 4 - 6 hours

7. Major acute problems: tachycardia, headaches, flushing, nasal congestion, palpitations, vomiting, diarrhea, hypotension, myocardial ischemia , sodium and fluid retention

C. Diazoxide-Vasodilator, very effective and rapid onset of action. Must be given by rapid iv push.

1. Generic name: Hyperstat

2. Formulation: 300 mg/ 20 ml

3. Dose and route: 1-3 mg/kg, **rapid IV push:**

4. Interval: **For rapid IV push:** may repeat in 5-15 min. if no response, or use prn (usual interval Q4-24 hours)

5. Onset of action: 1 - 3 min.

6. Duration of action: 30 min. to 24 hours

7. Major acute problems: hyperglycemia, hyperuricemia, hypotension, nausea, vomiting, flushing, dysrhythmias, sodium and fluid retention, tachycardia. Do not use > 10 days.

D. Nitroglycerin-Vasodilator, immediate onset of action, must be given as continuous infusion. Preferred above nitroprusside in patients with renal impairment due the risk of cyanide toxicty with nitroprusside. Requires continuous A-line monitoring.

1. Generic name: NitroBid

2. Formulation: 5 mg/ml

3. Dose and route: 0.5 - 5.0 **micrograms**/kg/min IV, 150 mcg max

4. Interval: continuous infusion

5. Onset of action: immediate

6. Duration of action: only effective during infusion

7. Major acute problems: hypotension, abdominal pain, dizziness, headache

E. Sodium Nitroprusside-Vasodilator, immediate onset of action, thiocyanate poisoning can occur within 12 hours in patients with renal failure. Check cyanide levels if used > 72 hrs. Requires continuous A-line monitoring. Reduces pre-load and after-load. Photosensitive, bottle and tubing must be wrapped in tin foil.

1. Generic name: Nipride

2. Formulation: 50 mg

3. Dose and route: 0.5 - 10 **micrograms**/kg/min., max. dose: 20 mg, IV

4. Interval: continuous infusion

5. Onset of action: immediate

6. Duration of action: only effective during infusion

7. Major acute problems: cyanide/thiocyanate toxicity which manifests as metabolic acidosis (maintain thiocyanate levels < 10 mg/dl if test available), chest pain, abdominal pain, headaches, rigidity, GI upset, seizures

F. Labetalol-alpha and beta blocker which can be given as IV bolus or continuous drip. Use with caution in patients with asthma or other lung disease, or cardiac disease. May mask hypoglycemic symptoms or s/s of pheochromocytoma. May have less cerebral vasodilating effects than diazoxide or hydralazine so may be better in cases of increased ICP

1. Generic name: Normodyne

2. Formulation: 100 mg/ 20 ml

3. Dose and route: 0.2 - 3 mg/kg/hour, IV

4. Interval: continuous infusion

5. Onset of action: 5 min.

6. Duration of action: only effective during infusion

7. Major acute problems: nausea, PVC's, rhinorrhea, hypotension, bronchospasm

G. Esmolol-beta blocker. More rapid onset of effect and more rapid clearance than labetalol, use with caution in patients with asthma or other lung disease.

1. Generic name: Brevibloc

2. Formulation: 100 mg/10 ml

3. Dose and route: 500 mcg/kg IV load over 1 minute, followed by continuous IV infusion 100 - 300 **micrograms**/kg/min

4. Onset of action: immediate

5. Duration of action: only effective during infusion

6. Major acute problems: nausea, dizziness, bronchospasm, hypotension, bradycardia

Note: If rebound hypertension is suspected, reinstitution of the discontinued antihypertensive medication may rapidly and effectively control the hypertension.

The following 4 tables are from:

"The Report of the Second Task Force on Blood Pressure Control in Children 1987", PEDS, 79 (1), 1 - 25, 1987.

1. DEFINITIONS

TERM	DEFINITION
Normal BP	Systolic and diastolic BP's < 90th percentile for age and sex
High normal BP *	Average systolic and/ or average diastolic BP between 90th and 95th percentiles for age and sex
High BP (hypertension)	Average systolic and/ or average diastolic BPs ≥ 95th percentile for age and sex with measurements obtained on at least three occasions

* If the BP reading is high normal for age, but can be accounted for by excess height for age or excess lean body mass for age, such children are considered to have normal BP.

2. Classification of Hypertension by Age Group

Age Group	Significant Hypertension (mmHg)	Severe Hypertension (mmHg)
Newborn 7 day 8 - 30 days *	 Systolic BP ≥ 96 Systolic BP ≥ 104	 Systolic BP ≥ 106 Systolic BP ≥ 110
Infant (< 2 yr)	Systolic BP ≥ 112 Diastolic BP ≥ 74	Systolic BP ≥ 118 Diastolic BP ≥ 82
Children (3 - 5 yr)	Systolic BP ≥ 116 Diastolic BP ≥ 76	Systolic BP ≥ 124 Diastolic BP ≥ 84
Children (6 - 9 yr)	Systolic BP ≥ 122 Diastolic BP ≥ 78	Systolic BP ≥ 130 Diastolic BP ≥ 86
Children (10 - 12 yr)	Systolic BP ≥ 126 Diastolic BP ≥ 82	Systolic BP ≥ 134 Diastolic BP ≥ 90
Adolescents (13 - 15 yr)	Systolic BP ≥ 136 Diastolic BP ≥ 86	Systolic BP ≥ 144 Diastolic BP ≥ 92
Adolescents (16 -18 yr)	Systolic BP ≥ 142 Diastolic BP ≥ 92	Systolic BP ≥ 150 Diastolic BP ≥ 98

*** also see tables that follow for neonates !**

3. Historical Information to Elicit

Information	Relevance
Family history of hypertension, preeclampsia, toxemia, renal disease, tumors	Important in essential hypertension, inherited renal disease, and some endocrine diseases (eg. familial pheo. with multiple endocrine adenopathy II)
Family history of early complications of hypertension and/ or atherosclerosis	Suggests likely course of hypertension and/ or presence of other coronary artery disease risk factors
Neonatal history	Use of umbilical artery catheter suggests need to evaluate renal vasculature and kidneys
Headaches, dizziness, epistaxis, visual problems	Nonspecific symptomatology, usually not etiologically helpful
Abdominal pain, dysuria, frequency, nocturia, enuresis	May suggest underlying renal disease
Joint pains/ swelling, facial or peripheral edema	Suggests connective tissue disease and/ or other forms of nephritis
Weight loss, failure to gain weight with good appetite, sweating, flushing, fevers, palpitations	In combination, symptoms suggest pheochromocytoma
Muscle cramps, weakness, constipation	May suggest hypokalemia and hyperaldosteronism
Age of onset of menarche, sexual development	May be helpful in suggesting hydroxylase deficiencies
Ingestion of prescription and over-the-counter drugs, contraceptives, illicit drugs	Drug-induced hypertension

4. Findings to Look for on Physical Examination

Physical Findings	Relevance
<u>General:</u>	
Pale mucous membranes, facial or Pretibial edema	Renal disease
Pallor, evanescent flushing, Increased sweating at rest	Pheochromocytoma vs. hyperdynamic essential hypertension
Cafe au lait spots, neurofibromas	Von Recklinghausen disease
Moon face, hirsutism, buffalo hump, Truncal obesity, striae	Cushing syndrome
Webbing of the neck, low hairline, wide-spaced nipples, wide carrying angle	Turner syndrome
Elfin facies, poor growth, Retardation	Williams syndrome
Thyroid enlargement	Hyper- or hypothyroidism
<u>Cardiovascular:</u>	
Absent or delayed femoral pulses, low leg pressure relative to arm BP	Aortic coarctation
Heart size, rate, rhythm, murmurs, Respiratory difficulty, hepato-	Murmur - coarctation; tachycardia and/ or arrhythmia - pheochromocytoma; large

Megaly	heart or heart failure - prolonged or severe hypertension
Bruits over great vessels	Arteritis or arteriopathy
Abdomen:	
Epigastric bruit	Renovascular diseases isolated or associated with Williams or Von Recklinghausen syndromes, or arteritis
Unilateral or bilateral masses	Wilms tumor, neuroblastoma, pheochromocytoma, polycystic kidneys, other tumors
Neurologic:	
Hypertenive funduscopic changes	Chronic hypertension
Bell palsy	Chronic hypertension (HTN)
Neurologic deficits (eg. hemiparesis)	Chronic or severe acute HTN with stroke

Neonatal Systemic Hypertension:

The following 2 tables provide further information for neonates.

1. Systolic (S) and Diastolic (D) Blood Pressures in Low Birthweight Infants

Birthweight 600 - 999 g			**Birthweight 1000 - 1249 g**		
Day	**S (± 2 SD)**	**D (± 2 SD)**	**Day**	**S (± 2 SD)**	**D (± 2 SD)**
1	37.9 (17.4)	23.2 (10.3)	1	44 (22.8)	22.5 (13.5)
3	44.9 (15.7)	30.6 (12.3)	3	48 (15.4)	36.5 (9.6)
7	50 (14.8)	30.4 (12.4)	7	57 (14.0)	42.5 (16.5)
14	50.2 (14.8)	37.4 (12.0)	14	53 (30.0)	-
28	61.0 (23.5)	45.8 (27.4)	28	57 (30.0)	-
Birthweight 1250 - 1499 g			**Birthweight 1500 - 1750 g**		
Day	**S (± 2 SD)**	**D (± 2 SD)**	**Day**	**S (± 2 SD)**	**D (± 2 SD)**
1	48 (18.0)	27 (12.4)	1	47 (15.8)	26 (15.6)
3	59 (21.1)	40 (13.7)	3	51 (18.2)	35 (10.0)
7	68 (14.8)	40 (11.3)	7	66 (23.0)	41 (24.0)
14	64 (21.2)	36 (24.2)	14	76 (34.8)	42 (20.3)
28	69 (31.4)	44 (25.2)	28	73 (5.6)	50 (9.9)

from: Ingelfinger JR, Powers L, Epstein MF: Blood Pressure Norms in Low-birthweight Infants: Birth through 4 Weeks, Pediatr Res 17: 319A, 1983

2. Mean Arterial BP (MAP) in mmHg (± SD) in Preterm and Term Ill Neonates

Birthweight (kg)	**< 1.0 kg**	**1.0 - 1.5 kg**	**1.5 - 2.5 kg**	**> 2.5 kg**
Birth	32.9 ± 15.4	39.1 ± 18.2	42.4 ± 19.6	48.8 ± 19.4
7 days	41.4 ± 15.4	47.2 ± 18.2	50.4 ± 19.6	60.2 ± 19.4
14 days	44.6 ± 15.4	50.1 ± 18.2	53.2 ± 19.6	64.2 ± 19.4
28 days	47.6 ± 15.4	53.0 ± 18.2	56.1 ± 19.6	68.3 ± 19.4

from: Stork EK, Carlo WA, Kliegman RM, et al: Hypertension redefined for critically ill neonates. Pediatr Res 18: 321A, 1984

INCREASED INTRACRANIAL PRESSURE

I. Introduction

The Monro-Kellie doctrine, based on the concept of a fixed intracranial volume, states that an increase in the volume of one intracranial compartment (blood, brain, CSF) must be accompanied by a decrease in one of more of the other compartments if intracranial pressure is to remain unchanged. The CSF and cerebral blood volume are the two compartments best able to be manipulated to buffer changes in increased intracranial volume. When compensatory mechanisms become overwhelmed, life threatening intracranial hypertension can result from relatively small increases in volume. A pathologic increase in intracranial volume should be anticipated in predisposing conditions such as trauma, meningitis, diabetic ketoacidosis, and Reye's syndrome. Additionally, seemingly trivial physiologic abnormalities in atrial and venous pressure, PCO2, Na, and osmolality can result in catastrophic consequences in patients with severe compromise of intracranial pressure (ICP) homeostatic mechanisms.

II. Etiology

A. Diffuse brain swelling from:

1. Impaired autoregulation of cerebral blood flow (head injury, Reye's syndrome, encephalitis, asphyxia)

2. Cytotoxic edema (head injury, toxins, asphyxia)

B. Mass Lesion

1. Tumor

2. Bleeding (subdural, parenchymal, epidural, AVM)

3. Abscess

C. CSF obstruction (hydrocephalus, mass lesion, meningitis)

D. Hyperosmolar states (DKA, hypernatremia, non-ketotic hyperglycemic coma)

III. Presentation

A. Headache

B. Vomiting

C. Cushings Triad: Increased ICP, Hypertension, Bradycardia (tachycardia may be seen early). Bradycardia is usually a late sign and may herald an arrest.

D. Papilledema (very sensitive finding, but seen in < 50 % of patients early on in the course)

E. Depressed level of consciousness

F. Abnormal breathing pattern

G. Pupils dilated, unequal, unresponsive

H. Difficult to control seizure activity or prolonged seizures are especially associated with mass lesions, infections or hemorrhage.

I. Cranial Nerve findings (especially VI)

J. Glasgow Coma Scale (GCS) < 8. However, obtundation, confusion, restlessness, agitation and progressive unresponsiveness to environmental stimuli are early manifestations of increased ICP. (see neurologic assessment chapter for GCS's)

K. Decerebrate or decorticate posturing.

L. Bulging fontanelle.

IV. Emergency Management

A. As nothing can be done about the primary injury or neuronal damage already suffered, the goal centers on preventing secondary injury. In the immediate postinjury period, secondary insults will most likely occur as a result of **hypoxia**, ischemia from frank or realtive **hypotension**, or the detrimental effects of intracranial hemorrhage.

B. Airway (the "A" in ABC's)

1. Must establish quickly to avoid hypoxia. The threshold for intubation should be very low in a head injured child.

2. Any child unable to open his or her eyes or verbalize should be considered for intubation.

3. Patients with abnormal respiratory rate and rhythm, upper airway obstruction (loss of pharyngeal muscle activity, inability to clear secretions, foreign body, direct trauma, seizures), loss of protective airway reflexes, sign of ICP hypertension, or significant pulmonary or cardiovascular disease should be intubated.

4. C-spine precautions are paramount. The neck should not be extended and intubation medications should include a combination of sedation (for blunting of cardiovascular reflexes, and blunting of intracranial responses), lidocaine (for blunting of increased ICP response to laryngeal stimulation) and paralysis (for ease of intubation). The intubation should be done via rapid sequence protocol which includes the Sellick's maneuver (cricoid pressure) and preoxygenation with 100% FIO2 without bag ventilatiion (when possible). (See Rapid Sequence Induction chapter).

C. Breathing (B = breathing)

1. In the head injured patient, hypercapnia must be avoided. Carbon dioxide is a very potent cerebral dilator, which could increase cerebral blood volume and accordingly increase ICP.

D. Shock (C = circulation))

1. Once the airway and ventilation have been assured, the adequacy of circulation and perfusion must be assessed and restored as needed to prevent secondary injury.

E. Physical Assessment

1. A detailed history is needed to understand the mechanism of injury. If the patient looks uninjured but the mechanism of injury was significant, look carefully for occult injury.

2. Mechanism details: pt ejected?, amount of damage to the vehicles?, estimated speed?, distance of fall?, seatbelt/car seat?, surface ont which the pt. fell?

3. It also is important to ask how the patient responded at the scene: Was the patient conscious at the scene and did the patient have a seizure at the scene?

4. Past medical history; important for previous health problems, allergies, tetanus history, and current medications.

F. Complete physical exam - look for signs of occult trauma: blood behind TMs, CSF from ears/nose, retinal hemorrhages, broken ribs, unequal breath sounds, muffled heart sounds, chest/abd contusions, extremity swelling.

G. Labs:

1. ABG

2. dextrostix

3. CBC, type and cross (if signif blood loss suspected)

4. Chem 10, glucose, osmo

5. LFT’s, amylase, lipase

6. Ammonia (if Reye’s sydrome or metabolic disease suspected)

7. Toxicology screen if an ingestion is suspected

8. UA (for evidence of hematuia and renal injury)

9. Blood cultures, urine culture (if infection suspected)

H. Medical treatment of increased ICP (see below)

I. Consult Neurosurgery

J. CT Scan, continue aggressive brain resuscitation during scan. Perform emergently if hx, of LOC, seizure, coma, focal neuro exam, blood behind TM's, CSF rhinorrhea or otorrhea.

V. Medical Management

A. Intracranial Pressure Monitoring

1. The two major purposes for monitorin ICP are prevention of herniation and preservation of cerebral perfusion. Though ICP monitoring has not been proven to improve outcome, it is used extenisively to guide therapy and likely accounts partially accounts for the general improvement in outcome from traumatic brain injury that has occurred over the last 2 decades.

2. ICP monitoring is indicated in the following children:

a) Those with a GCS of < 8

b) Those with significant trauma requiring non-neurosurgical procedures

c) Those who require continued deep sedation or neuromuscular blockade for ventilatory management (or other in whom sequential neurologic exams cannot be adequately performed)

3. The two most common methods of ICP monitoring are the ventriculostomy and the intraparenchymal (Camino) catheter.

a) The ventriculostomy is silastic catheter that is placed through the frontal lobe into the anterior horn of the lateral ventricle. Advantages of this device include accurate measurement of ICP, ability to recalibrate the device, and ability to drain CSF in an attempt to lower ICP. Disadvantages include difficult placement in a patient with small ventricles, higher potential for ventriculitis (and worse outcome)in the case of catheter infection.

b) The intraparenchymal catheter contains a membrane, distortion of which deflects a mirror and attenuates a fiberoptically transmitted beam of light. This attenuation, detected by a sensor, provides a measure of the pressure on the transducer membrane. The device can be placed into the parenchyma, ventricle or subdural space. Advantages include easy placement and less risk of ventriculitis. Disadvantages include drift in readings over time and inabilibty to recallibrate the device as well as inability

to drain CSF throught the catheter. Intraparenchymal monitors are ~5X more expensive ($4,000-6,000) than ventriculostomies.

4. All monitors have a risk of infection. Infectious risk increases with duration of monitoring, particularly after the 5th day. The site of monitoring is typically changed every 5-7 days. Prophylactic antibiotics (Ancef) are indicated for the duration of ICP monitoring.

5. Intracranial waveforms resemble arterial waveforms. Plateau waves are sustained ICP elevations up to 40-50 mm H2O lasting 5-20minutes, falling quickly back to baseline. These appear to represent a "tight" brain and sudden increase in ICP, usually secondary to a fall in CPP and reflex increase in MAP until CPP is restored. (see below)

6. An arterial line is indicated in all patients with an ICP monitor.

B. Cerebral Perfusion Pressure (CPP)is the difference between the pressure of blood going to the brain (the mean arterial pressure or MAP) and the back pressure to this flow (the ICP). Thus:

CPP = MAP - ICP

A normal **ICP** is **10-15 mm Hg** or less. ICP will increase with agitation, coughing, etc., but should rapidly return to baseline. Patients with sustained increases in ICP should be treated in a stepwise fashion to try to decrease their ICP with the least invasive means possible being employed first. **Maintaining an adequate CPP is probably more important than keeping the ICP value normal.** Our goal should be to maintain cerebral perfusion pressure at **50-70mm Hg**.

F. Fluid Management

1. A central line is indicated to guide fluid management.

2. Patients with increased ICP need an adequate CVP and MAP to perfuse their brains. They should **not** be routinely fluid restricted. They should receive appropriate fluid resuscitation and be started on maintenance fluids with further management guided by clinical status, vital signs, CVP, and perfusion.

VI. Medical Manipulation of ICP

A. The art of ICP/CPP management is really related to the appropriate selection and timing of these therapeutic maneuvers and understanding their limitations. Though specific management varies somewhat between trauma centers, a stepwise approach to various interventions similar to the one below is attempted for patients with elevated ICP/low CPP:

1. Appropriate ABC's and fluid resuscitation and frequent monitoring of VS, exam, ICP, electolytes, osm.

2. Intubation using rapid sequence induction. Prophylactic hyperventilation is no longer routinely employed, however moderate hyperventilation (PCO2 ~ 30-35) may be used for suspected increase ICP in the field or prior to CT scanning.

3. Sedation using Versed or Ativan 0.1 mg/kg IV or 0.1 mg/kg/hour continuous infusion. Additional sedation and/or analgesia should be considered before suctioning, patient transfer, procedures, etc. In addition Lidocaine 1 mg/kg iv before suctioning may blunt the increased ICP associated with this intervention.

4. Elevation of the head of the bed 15-30 degrees and keeping the head midline may enhance cerebral venous drainage.

5. Removal of CSF if a ventriculostomy is in place.

6. Mannitol

   a) Decreases blood viscosity resulting in lower cerebrovascular resistance, reflex cerebral vasoconstriction and reduction in cerebral blood volume.

   b) May act as an osmotic agent to decrease brain water with subsequent osmotic diuresis.

   c) Dose is 0.25-0.5 gm/kg rapid IVP over ~ 5 min.

   d) Goal: reduction in ICP, improvement in CPP or maximum osmolality of 310-320. If multiple doses are administered, the patient can be placed on scheduled mannitol Q4-6 hours. Serum osm and Na are followed at a similar interval and mannitol is withheld if the osm are >310. Na typically are in the 150s.

7. Hyperventilation

   a) Hyperventilation causes cerebral arterial constriction with subsequent decreased cerebral blood flow and decreased ICP. It is a potentially harmful method of decreasing ICP as CPP may concomitantly be compromised. Moderate HV to a **PCO2 of 30-35** is typically well tolerated. More extreme hyperventilation is no longer practiced at most trauma centers.

   b) The beneficial effects of HV diminish after 24-48 hours, due to buffering, however rapid reversal of HV results in rebound increased ICP. Thus it is important that HV is weaned slowly (ie. over hours to days).

7. Neuromuscular blockade can be considered to decrease any muscle movement (coughing, shivering, etc.) that might contribute to increased ICP. Increased sedation alone can often accomplish this. If NMB is instituted a high level of suspicion for seizure activity must be maintained as tonic-clonic activity will not be apparent.

8. Barbiturate coma

a) Employed for increased ICP unresponsive to less invasive therapies.

b) Dose is 5 mg/kg bolus every hour X 3 and an infusion of 1-5 mg/kg/hour titrated up or down to achieve control of intracranial hypertension or to achieve burst suppression (2-9 bursts/minute on EEG), whichever comes first. Continuous EEG monitoring is necessary until burst suppression is achieved, intermittant monitoring may be appropriate once the patient has stabilized.

c) Hypotension due to cardiac depression is common and patients frequently require vasopressor support with epinephrine or norepinephrine. It is prudent to have vasopressor medications at the bedside before loading with pentobarb. A pulmonary artery catheter should be considered to guide management.

9. Other issues of monitoring and management.

a) Hyperthermia and seizures cause elevations in ICP and should be anticipated, prevented if possible, and treated aggressively. Patients with significant brain injury or mechanism of injury are routinely treated with Dilantin prophylactically for 5-10 days.

b) Hyperglycemia has been associated with worse outcome in stroke victims. This may or may not be relevant to trauma, but avoidance of hyperglycemia is probably prudent.

c) Patients with traumatic brain injury are at risk for DI and SIADH which can be identified by monitoring UOP and electrolytes.

d) Patients with traumatic brain injury are at risk for gastric ulcers. Prophylaxis with H2-blockers is recommended.

e) Patients with TBI are relatively immunnosuppressed. A high index of suspicion for infection (especially pneumonia) should be maintained and infections aggressively treated.

f) Steroids are not indicated in the management of traumatic brain injury. They may have a role in the management of edema surrounding a brain tumor (Decadron 1.0 mg/kg/day IV divided q 6 hours). Steroids are of benefit in spinal cord

injury (Solumedrol 30 mg/kg iv ASAP after injury, followed by 5.4 mg/kg/hour continuous infusion for 23 hours)

VI. Surgical Management

A. Mass lesions (hematomas, tumors) may require immediate surgical evacuation. With the initial surgical intervention, a bone flap can also beremoved, especially if it is anticipated that the patient will have tremendous difficulty with intracranial hypertension. Subsequent crainiotomy or frontal/temporal lobectomies are occaionally employed to treat refractory ICP.

B. An acute rise in ICP or change in neuro exam (ie. blown pupils, posturing, decreased responsiveness)warents consideration of a **stat CT scan** to rule out any surgically correctable lesions.

VII. Weaning of Interventions

Once a patient has stabilized (hours, days, or sometimes weeks into therapy), interventions are typically weaned in the reverse order that they were instituted. A significant number of children that require pentobarb coma will recover and be nerologically intact. Survival is best predicted by the patient’s GCS and cardiovascular status upon arrival to the ED.

VIII. Summary

Children with severe increased ICP requiring ICP monitoring should be managed by an intensivist, neruosurgeon, and ICU nurses experienced in the care of critically ill children. Important points to remember include: ABC's, cervical spine protection, consideration of increased ICP at all times, close attention to changes in VS and PE, prevention of secondary injury (hypoxia and hypotension), stepwise increase in interventions, knowledge of potential etiologies of deterioration and/or side effects of therapies.

NEUROLOGIC ASSESSMENT, ALTERED MENTAL STATUS, GLASGOW COMA SCALES

I. Neurologic assessment:

A. The signs of brain hypoperfusion depend on the severity and duration of the insult. In cases of sudden onset brain ischemia, few signs of neurologic compromise may precede loss of consciousness. Convulsions and pupillary dilation may occur with loss of muscular tone.

B. With shock, symptoms may be insidious. Confusion and lethargy may appear. Agitation alternating with lethargy is common. Failure to recognize parents is an early, ominous sign of cortical hypoperfusion.

C. In later stages of hypoperfusion deep tendon reflexes may be depressed, pupils may be small but reactive and a crescendo- decrescendo breathing pattern (Cheyne-stokes) may be present.

D. Hypotonia and intermittent flexor or extensor posturing may occur with prolonged cerebral hypoperfusion or extreme hypoxemia.

E. Always check electrolytes **and** a dextrostix on any neurologically impaired patient. (the dextrostix returns faster than a lab glucose)

F. The neurologic assessment should follow a simple, rational plan:

1. To determine the patient’s level of consciousness.

2. To determine the presence of localizing (abnormal) findings.

3. It also serves as a baseline for comparison later in the hospital course.

4. The exam needs to be tailored to the situation. For instance, in comatose patients the Glasgow Coma Scale may give you all the information you need. In other patients a more detailed exam will be both possible and necessary.

G. Approach:

1. Begin with the level of consciousness (the mental status equivalent).

2. Next, assess the cranial nerves from the top of the brainstem (pupils) to the bottom (respirations, BP, HR, gag reflex)

3. Perform a motor exam looking for weakness, asymmetry, or tone abnormalities.

4. DTR’s, including the Babinski reflex assessment, may help determine a level of asymmetry. A positive Babinski response

(extension of the great toe and abduction of the other toes instead of the normal flexion to plantar stimulation) is abnormal after the age of 12 months, and always abnormal if asymmetric. It is an “Upper Motor Neuron” finding that implies central (supranuclear) dysfunction.

5. Perform a sensory examination. This may be difficult in young children and comatose patients but their response to stimulation can be assessed.

6. **Neonates** should be assessed carefully for level of consciousness, cranial nerve and motor function. In addition, neonatal reflexes should be elicited.

Reflex	Appearance age	Disappearance age
Adductor spread of knee jerk	Birth	7 - 8 mos.
Babinski reflex (positive)	Birth	12 mos.(see above)
Landau reflex	10 mos.	24 mos.
Moro	Birth	5 - 6 mos.
Palmar grasp	Birth	6 mos.
Parachute	8 - 9 mos.	Persists
Plantar grasp	Birth	9 - 10 mos.
Rooting	Birth	3 mos.
Tonic neck response	Birth	5 - 6 mos.
Truncal incurvation	Birth	1 - 2 mos.

II. **Glasgow Coma Scale (GCS) (best for patients > 1 year old)**

Activity	Best Response Points
1. Eye opening	
No response	1
Response to pain	2
Response to voice	3
Spontaneously	4
2. Verbal Response	
No response	1
Incomprehensible Sounds	2
Inappropriate Words	3
Disoriented conversation	4
Oriented and appropriate	5
3. Motor Response	
No response	1
Decerebrate posturing	2
Decorticate posturing	3
Flexion withdrawal	4
Localizes pain	5
Obeys Commands	6

Maximum Score	15

Scores < 9 indicate severe injury, airway support usually required.

III. **Modified Glasgow Coma Scale (especially helpful for ages ≤ 1 year).**

Activity	Best Response
1. Eye Opening	
None	1
To pain	2
To speech	3
Spontaneous	4
2. Verbal Response	
None	1
Moans to pain	2
Cries to pain	3
Irritable cries	4
Coos, babbles	5
3. Motor Response	Points
None	1
Extensor Response	2
Abnormal Flexion	3
Withdraws to pain	4
Withdraws to touch	5
Normal Spontaneous Movements	6
Maximum Score	15

Scores < 9 indicate severe injury, airway support usually required.

IV. **Brief Neuro Exam: Follows general exam, GCS, and assessment for "ABC's".**

A. Cranial Nerves: pupils (size, symmetry), fundi, response to visual threat, extraocular muscles (use doll's eyes maneuver in comatose patient **with a stable neck**, cold calorics in those in whom the cervical spine has not been cleared), corneal reflex, facial grimace to pain, gag/swallow reflex.

1. Doll's eyes (oculocephalic response) **ONLY PERFORM IN PATIENTS WITH A STABLE, CLEARED C-SPINE !** : Grasp the head firmly and hold the eyelids open. Quickly turn the head to one side. Repeat by turning the head in the other direction.

a. If the eyes continue to gaze straight ahead the response is positive (the eyes actively deviated in the opposite direction of the turn). A positive response indicates an intact brainstem.

b. The lack of this response in **both** eyes is part of the picture of brain death. A low brainstem lesion will show a negative response. This means that the eyes will move in the direction of

the head turn, as if they were fixed in the orbits and were not mobile.

2. Cold calorics (oculovestibular reflex) may be performed in patients with C-spine injuries: First, examine the external auditory canal to make sure no obstruction or foreign matter is occupying the canal. Next, make sure there is no tympanic membrane perforation. Elevate the head of the bed 30 degrees. Gently instill 30 - 50 cc of ice water in the ear canal while holding the patients eyes open (may use an assistant). Observe both eyes for the response. Wait 5 min. to restabilize the vestibulo-ocular system and repeat in the other ear. This test is usually **NOT** performed in non-comatose patients as it is uncomfortable.

a. If the brainstem is intact the eyes should move towards the irrigated side. If done in an alert patient nystagmus occurs.

b. In the presence of severe brainstem damage the reflex is abolished and no deviation of the eyes occurs.

B. Motor - note posture at rest, spontaneous movements.
- tone assessment (resistance to passive movements).
- motor response to stimulation (posturing).

C. DTR's - including Babinski

D. Sensory - response to stimuli (localizes pain, withdraws, postures), other testing possible in more alert patient.

E. Coordination - rapid alternating movements
- finger-nose-finger
- heel-shin

F. Gait - width of base, stability, Romberg, heel/toe/tandem for more alert, cooperative patient.

V. Altered states of consciousness (altered mental status)

A. Pathologic alterations in consciousness are caused by a variety of disease processes. These processes may lead to decreased responsiveness to visual, auditory, and tactile stimulation. They are **NOT** all or none phenomena. Altered levels of consciousness may be divided into four categories.

1. **Lethargy**: This is a state of minimally decreased wakefulness where the primary deficit is attention. The patient is easily distracted and has faulty memory, but retains the ability to communicate by verbal or nonverbal means. Drowsiness is prominent.

2. **Obtundation**: This is a mild or moderate blunting of alertness accompanied by a lessened interest in or response to the environment. Communication is partially preserved.

3. **Stupor**: Stupor is clinically equivalent to deep physiologic sleep from which the patient can be partially or only temporarily aroused, and only by vigorous and repeated stimulation. Communication is minimal or nonexistent.

4. **Coma**: Coma can be defined as a reduction in neuronal function resulting from disruption of cerebral cortical or brain stem integrity. This is a state of unarouseable unresponsiveness in which the patient lies without spontaneous movement with the eyes closed. There is no intelligent speech. The patient may withdraw from noxious stimuli, but cannot localize pain with discrete, defensive movements.

B. These states are not to be confused with **delirium** which is an abnormal mental state characterized by disorientation, instability, delusions, or hallucinations. Delirium is prominent when toxic or metabolic disorders affect the cerebral hemispheres, either primarily or exclusively.

C. Distinguishing metabolic etiologies from structural damage provides the basis for developing and investigating the differential diagnosis of an altered state of consciousness.

D. Differential features:

1. Supratentorial destruction or mass lesions

    a. Initial signs are focal

    b. There is a rostral to caudal progression

    c. Hemispheric dysfunction (i.e. right or left hemisphere) occurs depending on handedness and site of injury

2. Infratentorial destructive or mass lesions

    a. You usually see a preceding brain stem dysfunction

    b. Onset of coma is sudden

    c. Cranial nerve palsies occur

    d. Respiratory disturbances may be seen early

3. Toxic, metabolic or infectious diseases

    a. Confusion or stupor precedes the motor signs

    b. Motor signs are symmetric

    c. pupillary reactions are preserved

    d. Asterixis, myoclonus, tremor, or seizures may be seen

    e. May see hyper or hypoventilation

E. Etiologic spectrum of diseases producing an altered state of consciousness:

1. Supratentorial lesions of the brain

    a. Extracerebral

        (1). Neoplasm

        (2). Epidural or subdural hematoma

        (3). Subdural empyema or effusion

    b. Intracerebral

        (1). Hemorrhage (parenchymal, intraventricular, or subarachnoid)

        (2). Infarction

        (3). Neoplasm, abscesses, granulomas

        (4). Expanding mass lesions

        (5). Edema

2. Infratentorial lesions of the brain

    a. Infarction

    b. Hemorrhage (brain stem or cerebellar)

    c. Expanding mass lesions

    d. Edema

3. Hydrocephalus

4. Toxic, metabolic, or infectious disorders

    a. Deprivation of oxygen, substrate or metabolic cofactors

        (1). Hypoxia

        (2). Ischemia

        (3). Seizure or postictal states

        (4). Cofactor deficiency (thiamine, niacin, pyridoxine)

        (5). Inborn error of metabolism

5. Other organ failure

    a. Liver (hepatic coma, or hyperammonemia)

    b. Kidney (uremic coma)

    c. Lung (CO_2 narcosis)

    d. Endocrine (thyroid, parathyroid, or adrenals)

6. Exogenous poisons

    a. Sedatives

    b. Acid poisons

    c. Heavy metals

    d. Cyanide

    e. Others (see poisoning chapter)

7. Abnormal ionic and acid-base balance

    a. Water

    b. Sodium

    c. Potassium

    d. Magnesium

    e. Calcium

8. Infectious and parainfectious diseases

    a. Meningitis

    b. Encephalitis

    c. Acute disseminated encephalomyelitis

    d. Toxic encephalopathy

9. Trauma

F. Important points of the history:

1. Duration and development of the coma

    a. Sudden onset: vascular catastrophe or seizure

b. Acute onset after period of normalcy: ingestion of drug, toxin, or poison

c. Coma developing over a period of hours to days: expanding intracranial mass, metabolic derangement, or infectious process

2. Recent illness or fever? Think of infectious process or Reyes

3. Trauma

a. Recent head injury? epidural? subdural?

b. Child abuse?

4. Recent travel?

a. Fungal, rickettsial, rabies, viral, plague, parasitic, Salmonella or Shigella infections

5. History of headache?

a. Chronic headache: brain tumors, vascular malformations, congenital anomalies, hydrocephalus, and other mass lesions

b. Headache of sinus origin: venous thrombosis, subdural empyema

c. Migraine can produce a reversible confusional state

6. History of other diseases?

a. Malignancy: think brain met.

b. Blood dyscrasia: think hemorrhage

c. Congenital heart disease: think infarct, embolization, or brain abscess

d. Renal disease: think uremia, or dialysis encephalopathy

e. Liver disease: think hyperammonemia

f. Diabetics: think hypo or hyperglycemia

G. Physical examination

1. ABC’s first !! Then assure vital signs are stable or being addressed. A careful evaluation for trauma, exanthems of the skin, and skin findings of the neurocutaneous syndromes or other systemic diseases should be performed.

2. Listen for cranial bruits. Check the character of the anterior fontanelle. Examine the ears and nose for the presence of blood or

leaking CSF. The retina of every child with an altered state of consciousness should be examined.

H. Laboratory evaluation

1. **The following is indicated for ALL patients with an altered state of consciousness:** glucose ***and*** dextrostix, BUN, creatinine, lytes, osm, calcium, phosphorus, magnesium, ammonia, CBC and diff, UA and ABG. (also, see number 5 below)

2. **The following is ALSO indicated for all patients with a suspected metabolic derangement:** urine metabolic screen, blood and urine amino and organic acids, thyroid screen, plasma cortisol, LFT's, urine porphyrins, plasma free fatty acids, ketones, and carnitine level.

3. **The following is ALSO indicated for all patients with a suspected toxic ingestion:** blood and urine drug screens, lead level, blood salicylate level, blood alcohol level

4. **The following is ALSO indicated for all patients with a suspected infection:** blood and other bodily fluid bacterial cultures, viral and/or fungal cultures or titers, ophthalmology exam if indicated for chorioretinitis etc., exams for parasites

   a. Lumbar puncture: An LP should be considered if a CNS infection is suspected. Meningitis and encephalitis are probably the only absolute indication for an LP. ***REMEMBER***: an LP may be hazardous in patients with increased intracranial pressure. A head CT should be performed before the LP. LP should also be deferred in patients with significant cardiovascular compromise or shock.

5. **EVERY** comatose patient should have a head CT unless the etiology has clearly been established.

I. General principles of management:

1. Maintain optimal HR, BP and respiratory status

2. Correct any systemic glucose, acid-base, or fluid and electrolyte imbalance

3. Manage hypo or hyperthermia

4. Treat increased intracranial pressure

5. Administer anticonvulsants for non-metabolic induced seizures

6. Perform frequent examinations and follow the patient's clinical status closely

NEWBORN RESUSCITATION

I. Introduction

A. General

1. 3.7 million infants are born in 5,000 hospitals in the U.S. each year. Only 5% have NICU's (Level III).

2. 6% will need life support in the Delivery Room. If under 1500 grams 80% will need resuscitation.

B. Basic Approach to CPR same as adult

1. Airway

2. Breathing

3. Circulation

a. Cardiac arrest is extremely uncommon in children or infants and when it does occur it is usually secondary to respiratory arrest. This makes airway and breathing even more important in infants and children.

C. Anticipation and Preparedness

1. If you are asked to attend a delivery and you feel it's worth your time to go, then it's worth going prepared and anticipating a disaster.

2. Know the equipment and know that it is functioning **BEFORE** the infant arrives.

3. NRC requires that at least 2 people with resuscitation skills be present at all deliveries, one must have intubation skills. The second person is absolutely necessary to evaluate the effectiveness of ventilation and to monitor the heart rate and if necessary to give chest compressions. If the delivery is considered high risk, two people must be dedicated only to the infant. If the resuscitation is prolonged, a third person will be required to insert lines and administer medications. **DO NOT GO BACK BY YOURSELF!!** Take a partner with you.

4. If you are alone and not with an established Resuscitation Team, the tasks should be divided among the available help **BEFORE** the infant arrives.

5. **Equipment List:**
Radiant warmer (on and warm)
Suction with manometer
Resuscitation bag (250-500cc)

Premature, and term face masks
Laryngoscope
Laryngoscope blades (straight 0 and 1)
Stethoscope
Towels
3-way stopcock(s)
Oral airways (newborn and premmie)
Bulb syringe
Meconium suction device
2.5, 3.0, 3.5 ETT's
Suction catheters (5,8,10 French)
ETT stylet
Syringes (10, 20 cc's)
Feeding tubes (5, 8 French)
Cord cutting scissors, clamp, gloves
3.5 and 5 Fr. umbilical catheter, sterile water
Medications: Epinephrine (1:10,000)
Sodium Bicarbonate 4.2%
Volume Expander - NS, plasminate
D10W

II. Basic Physiology

A. Fetal Circulation

1. Low flow (placenta)
2. High PVR
3. In parallel

B. Neonatal Circulation

1. Increased flow
2. Decreased PVR
3. In series

C. The pulmonary pressure is controlled in part by perivascular pH, paO2, paCO2. If ventilation and oxygenation are not established soon after birth, there is persistent pulmonary hypertension or persistent fetal circulation which will cause right to left shunting across the ductus arteriosus and the foramen ovale.

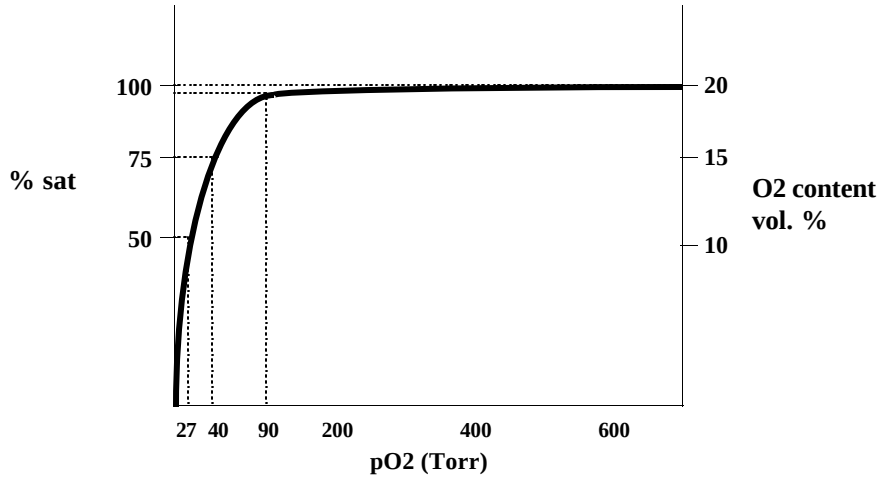
D. If ventilation and oxygenation are not soon established glycogen stores are used up, tissue hypoxia develops and anaerobic metabolism begins to produce large amounts of lactic acid. Cardiac output falls causing decreased perfusion to the organ systems and eventually the brain, heart, and adrenals.

E. The following graph depicts the ***adult*** oxyhemoglobin dissociation curve. ***Neonates have fetal hemoglobin which shifts the curve to the left.***

Shifts of this curve to the **left** are caused by: alkalosis, hypocarbia, decreased temperature, decreased 2,3-DPG, increased cardiac output, or fetal hemoglobin. These cause an increased oxygen affinity but a decreased oxygen release. Thus, the oxygen tension must drop lower than normal for the hemoglobin to release an equivalent amount of oxygen to the tissues compared to adult hemoglobin. However, fetal hemoglobin has a greater affinity for oxygen at lower paO2's (such as occurs with

placental blood perfusing the fetus) so more oxygen is bound in utero.

Shifts of this curve to the **right** are caused by: acidosis, hypercarbia, temperature elevations, increased 2,3-diphosphoglycerate (DPG), or decreased cardiac output. These cause a decreased oxygen affinity but increased oxygen release.



pO2, Saturation, and O2 Content

F. Oxygen content is the amount of oxygen contained in our blood. This oxygen may be in solution in the liquid portion of blood, or bound to hemoglobin inside the red cells.

1. Factors involved in calculating "oxygen content" include how much hemoglobin there is in the blood, how much oxygen a gram of hemoglobin can hold, the degree to which the hemoglobin is saturated, and finally how much oxygen is contained in the liquid portion of the blood. The equation which expresses the oxygen content of blood is:

$$0_2con = [Hgb] \times (\%\ sat) \times 1.36 + (p0_2 \times 0.0031)$$

where "0_2con" is oxygen content, "[Hgb]" is the hemoglobin concentration in grams per 100 ml of blood, "(% sat)" is the percent saturation of hemoglobin, 1.36 ml of oxygen per gram of hemoglobin is the oxygen carrying capacity of adult human hemoglobin, "$p0_2$" is the partial pressure of oxygen in the plasma in mmHg, and 0.0031 ml of oxygen per 100 ml per mmHg represents the solubility of oxygen in plasma. It is impressive to note from this equation that the magnitude of the contribution of dissolved oxygen in plasma, the only element affected directly

by the $p0_2$, to total oxygen content of the blood is quite minimal. Yet, pO_2 is the parameter most commonly followed clinically!

2. A moment's diversion on this point may be helpful. You may ask, "If the $p0_2$ is in fact such an unimportant parameter, why do we follow it so closely?" Historically, the $p0_2$ could be easily measured clinically. The hemoglobin saturation was either measured with a separate and specialized apparatus or was derived from the $p0_2$ and the pH. It also has to do with a fortuitous relationship between the $p0_2$ and hemoglobin saturation in the usual physiologic range as shown by the preceding diagram.

3. Between $p0_2$ values of approximately 40 and 90 torr, the relationship between the $p0_2$ and the hemoglobin saturation is fairly linear. Thus, a given increase in the $p0_2$ is accompanied by a roughly proportional increase in the hemoglobin saturation and thus in the oxygen content of the blood. However, it may also be clearly seen from the figure that below 40 torr and above 90 torr this is not the case. Below 40 torr, a small change in $p0_2$ is associated with a rather larger change in hemoglobin saturation. Thus, when the $p0_2$ decreases from 40 only to 27 torr the saturation falls from 75% to 50%. (Meaning that small changes in the $p0_2$ in this range can make a large difference in the well being of the patient's tissues!) The opposite is true above 90-100 torr where, even with dramatic increases in the $p0_2$, the saturation rises only very little. Thus, when the $p0_2$ rises from 90 to 600 torr the saturation goes only from about 99% to 100%. (Hemoglobin saturation cannot go any higher than 100%).

4. The relationship between changes in oxygen saturation and in changes in oxygen content are also depicted in the figure. It is important to note that these two parameters are related in a consistently linear fashion. That is to say that changes in one are accompanied by similar and proportional changes in the other. Because of this, hemoglobin saturation is a good indicator of oxygen content and is therefore a very useful clinical parameter to follow.

III. Infants at Risk

A. All infants are actually at risk for asphyxia during labor and delivery. Contractions decrease flow to the placenta and therefore gas exchange. A normal term infant without complications tolerates this without problems. There are four main mechanisms that can cause asphyxia to the fetus/neonate:

1. Interruption of umbilical blood flow i.e. cord compression.

2. Failure of exchange across the placenta because of separation i.e. abruption.

3. Inadequate perfusion on the maternal side of the placenta i.e. maternal hypotension, toxemia.

4. Neonatal asphyxia from failure to inflate the lungs from a variety of reasons:

a. Upper/lower airway obstruction i.e. meconium.

b. Inability to expand the lungs i.e. drugs, RDS/prematurity.

c. Inability to expand the lungs secondary to fetal asphyxia.

B. Maternal, perinatal, and postnatal complications and presentation identify the infants at risk.

1. Most asphyxia occurs prenatally when it is much more difficult to monitor. The best measure of in utero asphyxia is the cord blood gas pH. The arterial side is the side that reflects the fetus while the venous side reflects the placental well being. Obviously, we are more interested in the arterial or fetus side. The CO_2 is very helpful in determining the cause of the asphyxia. A high CO_2 on the arterial side out of proportion to the venous side is probably a cord compression causing CO_2 build up in the fetus. If utero-placental insufficiency is the problem, both gases would be abnormal.

VENOUS	Normal CORD GAS	ARTERIAL
7.29 (7.20 low normal)	pH	7.26 (7.15 low normal)
40	pCO_2	45-55
35	pO_2	20

2. Once the fetus/neonate has an abnormal gas, the neonate/fetus must be evaluated by a physician. A repeat blood gas from the newborn may be indicated.

3. It is very rare we are not forewarned of fetal distress and/or prematurity before the actual delivery. There is no excuse not to BE PREPARED BEFORE THE DELIVERY. CHECK THAT EQUIPMENT IS PRESENT, THE CORRECT SIZE AND PROPERLY FUNCTIONING!!!!

4. A high risk delivery should be anticipated if any of the following are present:

Antepartum Factors

Maternal diabetes	Post-term gestation
Pregnancy induced hypertension (PIH)	Multiple gestation
Chronic hypertension	Size-dates discrepancy
Preeclampsia	Previous stillbirth
Eclampsia	Drug therapy:

Previous Rh sensitization
Bleeding in 2nd or 3rd trimester
Maternal infection
Hydramnios
Oligohydramnios
Premature rupture of membranes
Decreased fetal movement
Reserpine
Lithium
Magnesium
Adrenergic blockers
Maternal drug abuse
No prenatal care
Fetal abnormality

Intrapartum Factors

Elective or emergency cesarean section
Abnormal presentation
Premature labor
Rupture of membranes > 24 hrs
Foul smelling amniotic fluid
Precipitous labor
Prolonged labor (> 24 hrs)
Prolonged 2nd stage of labor (>2 hrs)
Prolapsed cord
Abruptio placenta
Non-reassuring fetal HR patterns
General anesthesia use
Uterine tetany
Narcotics given to mom within 4 hrs of delivery
Meconium stained amniotic fluid
Placenta previa

IV. Assessment

A. APGAR SCORE SHOULD **NOT** BE USED TO DETERMINE THE NEED FOR RESUSCITATION

1. If the 5 minute score is less than 7 a score should be assigned every additional 5 minutes until greater than 7 or for a total of 20 minutes to assist in evaluating transition. The 15 and 20 minute scores are of more prognostic value than the 1 and 5 minute scores.

V. Steps in Neonatal Advanced Life Support

A. Temperature Regulation/Drying

1. Asphyxiated infants have an unstable thermoregulatory system and hypothermia delays recovery from acidosis.

2. Place the infant under a preheated radiant warmer.

3. Dry off amniotic fluid. Remove the wet towel.

4. The warmest place for a normal infant when a radiant warmer is not available is skin to skin against the mother.

B. Positioning

1. On back with neck in neutral or slightly extended position. If there are copious secretions, the head should be turned to the side.

2. A 1" blanket or towel may be place under the shoulders for maintaining the proper head and neck positions, esp. in babies with large occiputs from molding, edema etc.

3. Take care to prevent hyperextension or underextension of the neck.

C. Suctioning

1. First the mouth and then the nose. (The mouth is suctioned first to prevent aspiration should the infant gasp when the nose is suctioned). If secretions are copious turn the head so that secretions will pool in the mouth and not the posterior pharynx.

2. Use bulb syringe, or wall suction {**use no more than -100 mm Hg**,(80-100 mmHg) or -136 cm H_20}. Use 8 or 10 F. catheter.

3. Dangers:

Vagal bradycardia
Trauma
Hypoxia (suction no more than 5 sec.)
Vomiting and aspiration

D. Suctioning Meconium

1. Meconium staining of amniotic fluid occurs in up to 20% of all deliveries. It is thought to be a sign of fetal distress as well as a cause of pneumonitis.

2. If not adequately suctioned, 30% of infants with thick meconium (recent meconium and decreased fetal urine output another sign of fetal distress) will aspirate this fluid. Approximately 20% of all infants with moderate to thick meconium stained amniotic fluid will develop respiratory distress, pneumonitis, PPHN, air leaks.

3. The head should be delivered and the mouth, oropharynx, and hypopharynx thoroughly suctioned before delivery of the thorax and the first breath.

4. If the infant does not make any respiratory effort and will need positive pressure ventilation, the trachea should be intubated and suctioned before positive pressure ventilation is given. This is best done by connecting a wall suction meconium device to the endotracheal tube and suction while withdrawing the tube. **DO NOT USE** mouth suction through a mask or 4x4 or DeLee trap.

5. When thick or particulate meconium-stained fluid is present, tracheal suctioning should be completed as soon after delivery as possible. There is controversy regarding shether a vigorous baby with meconium-stained fluid requires tracheal suctioning. The difficulty of intubating a vigorous infant must be wighed against the advantages of full meconium removal.

6. Thin-watery or light meconium does not require endotracheal suctioning, unless the infant is depressed.

E. Stimulation

1. 2 flicks or slaps on the soles or 2 back rubs only. If no response proceed to ventilation. Further stimulation will only cause more hypoxia, and waste valuable time.

2. May rub head, body if baby is breathing and has a good HR.

VI. Assessment

A. The need for resuscitation should be determined by evaluating the:

Respiratory Activity
Heart Rate (HR for 6 sec X 10)
Color

VII. Ventilation/Oxygenation

A. If breathing:

1. Evaluate heart rate and then color.

2. If heart rate is above 100 and baby is centrally cyanotic give 100% blow by oxygen. Once baby is pink withdraw in increments, reassessing HR and breathing. (1/2" = 80%, 1" = 60%, 2" = 40%, then off.) Oxygen is not needed for peripheral cyanosis (acrocyanosis).

3. If HR > 100 and there is no central cyanosis, O_2 is not needed.

B. Indications for positive pressure ventilation. (4)

1. Apnea

2. Heart rate < 100

3. Persistent central cyanosis on 100% O_2

4. Gasping respirations

C. Maintain position of infant

D. Face masks are labeled preterm, and term infant. Should have no more than 5 ml of dead space. Establishing an airtight seal is the most critical step. Mask should be on bridge of the nose and cleft of the chin. The most common place to leak is between cheek and bridge of the nose. Do not put pressure on the trachea.

E. The person ventilating should be at the head of the bed and should have a clear view of the chest.

F. Remember the bag is 500-750 cc's and the infant's T.V. is only 20-30 cc's. Do not empty the bag with each squeeze. Use 5-8 liters/min of

100% O_2.

G. Give 40 to 60 breaths per minute. Count: squeeze, two, three, squeeze. Give enough to see chest rise a "normal easy breath". **LISTEN** over the upper chest or axilla and over the stomach.

H. If BVM applied for longer than 2 minutes insert an orogastric tube and leave in place during ventilation to prevent stomach distention. (Length = Bridge of nose --> earlobe --> xiphoid) Use 8 Fr. feeding tube and 20cc syringe. Tape to cheek. Consider intubation.

I. **IF NOT VENTILATING** check in order:

1. Reapply mask to obtain adequate seal

2. Reposition the head to correct a blocked airway

3. Check for secretions and suction if present to correct a blocked airway

4. Ventilate with infant's mouth open to correct for blocked airway.

   a. An oral airway may be used in this situation with choanal atresia or Pierre Robin Syndrome. Insert over tongue but don't force the tongue back as you insert it! It is not necessary to first reverse the position for insertion as in adults. Oral airways may cause gagging or vomiting so are usually not used in conscious patients. Often lying the baby on the stomach is sufficient to maintain airway patency.

5. Increase pressure delivered to correct for decreased compliance and inadequate tidal volume.

J. **Pressure**: May require 30-40 or even as high as 60 cm H_2O for initial inflation. How much pressure is 'enough'? 'Enough' means you observe the following:

1. Chest rising up and down

2. Bilateral breath sounds

3. Heart rate improving

4. Color improving. This may be 15-20 cm H_2O pressure or may be 20-40 cm H_2O <u>or higher</u> in lung disease with decreased compliance.

K. Bags

1. Self inflating i.e. Hope, Ambu, Laerdal

   a. Many have pop off valve at 30-35 cm H_2O

   b. Delivers only about 40% oxygen and 90-100% even with a

reservoir because of intake of room air which is mechanism of self inflation.

c. Can't give O_2 passively and usually no manometer in line for measuring PIP or PEEP.

d. Good to have for transports if out of gas.

2. Anesthesia Bags

a. Delivers 100% O2

b. Compliance detected

c. Pressure measured

d. Disadvantages: needs gas source, rebreathing of CO_2 if low flow and used for long periods.

L. After adequate ventilations established for 15-30 seconds, check the heart rate. Check the heart rate by listening with a stethoscope or feeling the umbilical or brachial pulse. The next step depends on the heart rate.

>100 and spontaneous respirations = O_2
<60 = continue ventilation, RECHECK VENTILATIONS and start chest compressions
60-100 and rising, ventilation only
60-100 and not rising RECHECK VENTILATION, if HR <80 initiate chest compressions

M. Signs of improvement:

1. Increasing heart rate

2. Spontaneous respirations

3. Improving color

VIII. Chest Compressions (External Cardiac Massage)

A. Recommendations by AHA/NRC

1. Never give a thump even with monitored fibrillation

2. Asphyxia leads to:

a. peripheral vasoconstriction

b. tissue hypoxia

c. acidosis

d. poor myocardial contractility

e. bradycardia

f. eventual cardiac arrest

3. Perform CC if **HR < 60**
HR < 80 and not rising despite adequate ventilation for 15 - 30 seconds

4. Position: ring and middle fingers one finger's breadth (index finger) below the line between the nipples.

5. Rate: 120 times per minute, do not remove your fingers from the chest between compressions

6. Compressions: 1/2 to 3/4 inch or 1/3 the chest AP diameter. If connected to blood pressure transducer, attempt to generate 75% of the systolic pressure present before the arrest.

7. Continue compressions until HR is 80 or greater. Continue ventilating until the HR is > 100.

8. Recheck Heart Rate every 30 seconds for 6 seconds (multiply X 10)

9. ALWAYS GIVE POSITIVE PRESSURE VENTILATION WITH 100% OXYGEN WHILE GIVING CHEST COMPRESSIONS!!

10. Compressions should be coordinated with ventilations in a 3:1 ratio so as not to compromise breaths. 120 “events” should occur each munute: 90 compresions and 30 breaths.

IX. Intubation

A. Indicated when bag and mask ineffective, for suctioning meconium, for prolonged ventilation or for known prenatal dx. of congenital diaphragmatic hernia.

B. Infant should be on a flat surface and in the sniffing position. May use a 1" towel roll under shoulders. DO NOT HYPEREXTEND because the cartilage of the trachea is collapsible.

C. The larynx is more cephalad, anterior, the epiglottis shorter and more U shaped, and the angle of the epiglottis with the cords more acute than in the adult.

D. Establish ventilation with bag and mask BEFORE ATTEMPTING INTUBATION. Most of the time intubation will be an elective procedure. Ventilation should not be interrupted for more than 20 sec. for intubation. Stop if the HR < 60 and bag. DON'T PANIC, an infant can

almost always be ventilated with a bag and mask. Exceptions:

1. ELBW <1200 grams with RDS

2. Diaphragmatic Hernias

3. Upper airway obstruction

E. Tubes

1. 2.5: <1000 grams, extremely premature, <28 weeks gestation

2. 3.0: 1000-2000 grams, premature, 28-34 weeks gestation

3. 3.5: 2000-3000 grams, term, 34-38 weeks gestation

4. 3.5 - 4.0: >3000 grams, large term, >38 weeks gestation

5. Use 0 laryngoscope blade for preterm and 1 for term infants

F. Technique

1. Laryngoscope is a left handed delicate instrument not a tire iron. A straight blade or Miller is preferred in infants. Check that the light functions!

2. Get down in the same plane as the infant's glottis.

3. Insert blade of laryngoscope and sweep tongue from right to left. Usually it is placed too deep into the esophagus. Pull back slowly, until the epiglottis comes in view, then lift epiglottis up. The cords will be posterior to or under the epiglottis. If they are not visible, apply cricoid pressure with your little finger or ask an assistant to apply it. While holding the blade in the direction of the handle, insert E.T. tube from the right side. An assistant may pull down the corner of the mouth for easier insertion. DO NOT SQUEEZE THE TUBE OR LET IT TOUCH THE GUMS!! This only directs the tube away from where you want it.

4. A stylet is not usually necessary. Those who think they need one should check and secure it so the tip does not go beyond the Murphy's eye. Remove the stylet once the ETT is in place.

5. If the cords are in spasm do not touch them. Ask someone to give the infant a gentle Heimlich maneuver and they will open. Never force the tube.

6. Monitor HR if it drops STOP, bag and try again.

7. If you don't see the cords, revisualize or have someone else do so. Attempting to intubate without adequate visualization rarely results in success and may lead to complications.

8. Distance from glottis to carina in term infant is 5 cm so place tube 2.5 cm below cords. Check breath sounds; **Lip to Tip measurement (cm = 6 + WT in kg), and CXR.**

9. Supervisors: if the baby is not responding to ventilation after intubation by a junior resident, he/she is not intubated until proven otherwise. **Look** to make sure the ET tube is between the cords.

10. Flexion of the baby's head causes tube to go down and extension causes it to move up.(the ETT goes the way the baby's nose moves)

11. Once in, note cm mark at upper lip and tape. Cut off tube so that no more than 4 cm of dead space exists.

12. ETT is correctly placed if: you hear bilateral breath sounds, see the chest rising with each ventilation, no air is heard entering the stomach, no gastric distention occurs.

13. An intubated patient who desaturates should have the ETT carefully evaluated. 6 problems occur in this situation:

1. Patient is extubated with ETT in the esophagus.
2. ETT is in the right mainstem bronchus.
3. ETT is plugged.
4. Patient has a pneumothorax.
5. Patient has decreased compliance so requires more pressure.
6. Ventilator, gas supply, or equipment is malfunctioning. Check ETT is not kinked !

X. Medication/Volume

A. Myocardial dysfunction and shock in the neonatal period are usually the result of profound hypoxia, acidosis, and/or hypovolemia.

B. Shock may also be secondary to hypovolemia.

1. Place UVC and measure CVP. If low consider hypovolemia. If normal or high, no help.

2. Usually there will be a history of blood loss, abruption, placenta previa, cord compression (especially if venous occlusion occurred only-cord gases will be helpful), fetal distress with fetal to placental transfusion.

3. Signs of hypovolemia:

a. Decreased B.P. - may not be present. BLOOD PRESSURE DOES NOT EQUAL PERFUSION. Shock can be present with normal or even high blood pressure.

b. Poor response to resuscitative efforts

c. Rapid heart rate with weak pulses, cap. refill (on chest) > 2 sec.

d. Pallor persisting despite oxygenation

e. Decreased CVP - but may not be decreased secondary to increased RV and pulmonary pressures.

4. Give volume over 5-10 minutes

a. 10 cc/kg 0 neg. blood crossmatched with mother if indicated.

b. 10 cc/kg N.S. or L.R.

c. 10 cc/kg 5% Albumin or plasmanate

5. Check Dextrostix and give 2 cc/kg of $D_{10}W$ if glucose < 40.

C. Medication should be administered if, after adequate ventilation with 100% oxygen and chest compressions, the heart rate remains < 80.

1. AHA/ACLS "There is no current evidence that two previously recommended drugs atropine and calcium, are useful in the acute phase of neonatal resuscitation. Sodium bicarbonate may be useful in the acute phase of neonatal resuscitation to help correct a documented metabolic acidosis but its' use is discouraged in brief arrests or episodes of bradycardia.

2. Epinephrine

a. An endogenous catecholamine with potent B adrenergic stimulating properties; in a cardiac arrest setting, the Alpha adrenergic mediated vasoconstriction may be the more important action.

b. Vasoconstriction elevates the perfusion pressure during chest compression, enhancing the delivery of oxygen to the heart.

c. It also enhances the contractile state of the heart, stimulates spontaneous contractions, and increases HR.

d. Indications

(1). Asystole, or hypotension unrelated to hypovolemia.

(2). HR <80 despite adequate ventilation for a minimum of 30 sec's with 100% oxygen and chest compressions.

e. Dose

(1). 0.01-0.03 mg/kg (0.1-0.3 cc/kg) 1:10,000

(2). May repeat q. 5 min

(3). I.V. or E.T. (May dilute 1:1 with NS if given via ETT)

3. Sodium bicarbonate will not improve blood pH in the absence of adequate ventilation, therefore the initial approach to resuscitation concentrates on oxygenation and ventilation to correct hypoxemia and acidosis. If needed, based on documented metabolic acidosis:

a. 2 mEq/kg over at least 2 minutes. (1 mEq/kg/min)

b. Remember that hypovolemia causing a metabolic acidosis should be treated with volume initially.

4. Naloxone

a. Indications
(1). For reversal of respiratory depression induced by narcotics given to the mother within 4 hours of delivery.

b. Ventilate and oxygenate first

c. Must admit to NICC on a monitor if mother or baby given naloxone, as the duration of action of naloxone is 1-4 hours and narcotics have a longer duration of effect.

d. **May induce withdrawal symptoms in baby if mother addicted !!**

e. Dose: 0.1 mg/kg of 0.4 mg/ml solution of Narcan rapidly

f. Route: I.V., E.T., if good perfusion I.M., S.Q.

XI. Vascular Access

A. UMBILICAL VEIN IS THE PREFERRED VASCULAR ACCESS

B. Place a 3.5-5.0 F. inserted so the tip is just below the skin level but has free flow (2-4 cm). Avoid placing the line higher in the acute phases of a resuscitation to avoid infusing hypertonic solutions into the liver.

C. Peripheral veins may be used

D. E.T. tube may be used for:
Narcan
Atropine
Valium > NAVEL
Epi
Lidocaine

E. Same dose but may dilute it with 1 cc of N.S. to aid in delivery. Place via feeding tube down E.T. and stop compressions during bagging.

XII. Countershock and Cardioversion

A. Defibrillation

1. Asystole and bradyarrhythmias are responsible for 90% of the arrhythmias in Pediatric cardiac arrest. Ventricular arrhythmias make up the remaining 10%.

2. If fibrillation present, DO NOT THUMP, oxygenation and ventilation with chest compressions usually is all that is necessary.

3. Paddle size: Infants 4.5 cm (< 10 kg, < 1 yr)
Children 8.0 cm (> 10 kg, > 1 yr)

4. Dose: 2 Joules/kg (follow algorithm)

B. Cardioversion

1. 1.0 Joules/kg (follow algorithm)

References: JAMA 255:2961-2973,1986, American Heart Association Newborn Resuscitation Textbook, 1994

ONCOLOGIC EMERGENCIES

ACUTE TUMOR LYSIS SYNDROME

I. CLINICAL FEATURES

A. Definition: Acute tumor lysis syndrome is a phenomenon of cellular death causing electrolyte abnormalities and renal dysfunction which occurs after malignant cell degradation.

B. Onset: may occur before therapy or 1 to 5 days after start of cytotoxic therapy (induction).

C. Most commonly seen in Burkitt's lymphoma and T-cell leukemia/ lymphoma. Also associated with bulky abdominal disease.

D. Associated with elevated pretreatment serum uric acid (see below), LDH (an indirect marker of tumor burden), and serum creatinine. Uric acid levels > 7.0, LDH > 250 and creatinine > normal for age place the patient at risk for tumor lysis syndrome.

E. Poor urine output.

II. PATHOPHYSIOLOGY

A. Degradation of malignant cells causes the release of phosphates and potassium during tumor lysis.
1. PO_4^{2-} concentration is 4-fold higher in lymphoblasts.
2. Calcium phosphate crystals precipitate in the microvasculature and renal tubules when $[PO_4^{2-}]x[Ca2^{++}]$ > 60 mg/dL.

B. Development of hyperuricemia occurs.
1. Purines are released by fragmented tumor nuclei. This causes increased substrate for uric acid production.
2. Xanthine oxidase is responsible for the enzymatic conversion of hypoxanthine to xanthine and xanthine to uric acid:

purine ® hypoxanthine ® xanthine ® uric acid

C. Patients become symptomatic at uric acid levels > 10 mg/dL. Symptoms include lethargy, nausea and vomiting, uric acid calculi, oliguria, anuria, and hematuria. A neurologic syndrome with lethargy, seizures and paresthesias may be observed in severe cases. (It also is associated with gouty arthritis in patients with solid tumors or chronic leukemia.)

D. The excretory capacity of the kidneys is exceeded resulting in inadequate renal function. Oliguric renal failure may ensue, resulting in hyperkalemia and hyperphosphatemia.

III. EVALUATION/MONITORING

A. Monitor electrolytes (especially K^+, PO_4^{2-}, Ca^{2+}, uric acid) and renal function (BUN, creatinine) every 4 to 6 hours.

B. Obtain an ECG if the K^+ is elevated. Widened QRS complexes and peaked T waves will precede malignant arrhythmias or asystole.

C. Strict volume status monitoring is essential. Evaluate I/Os q 6 hours.

D. Patients with tumor lysis syndrome are admitted to the PICU for constant monitoring of cardiac rhythm and BP.

E. Placement of central venous/ pulmonary artery catheters and arterial lines to facilitate hemodynamic monitoring is usually required.

IV. THERAPY (see Figure 1 which follows)

A. Establish metabolic stability before treatment.

B. Management of hyperkalemia, hyperphosphatemia and hypocalcemia are discussed in the chapter on electrolyte and fluid abnormalities. Avoid severe alkalosis which potentiates precipitation of calcium phosphate crystals in the microvasculature and renal tubules.

C. Hyperuricemia

1. Decrease uric acid production with allopurinol, a xanthine oxidase inhibitor.
2. Promote uric acid excretion, keeping urine pH alkaline (>6.5 and ≤ 7.5). Start with $NaHCO_3$ 100 mEq/L.
3. Decrease the uric acid concentration in the urine. Hydrate at 1½ to 2 times maintenance (2400 to 3000 cc/M^2/day - see body surface area formula below).
4. Treat renal insufficiency. Early insufficiency can be managed with lasix 1 mg/kg or mannitol 0.5 to 1.0 g/kg. MANNITOL IS CONTRAINDICATED IN CASES OF OLIGURIA (urine output < 0.5 cc/kg/hr). Acute or severe oliguric renal failure is managed by Pediatric Nephrology with hemoperfusion, hemodialysis or peritoneal dialysis.

D. Body surface area in M^2 = $\sqrt{\frac{\text{height (cm) X weight (kg)}}{3600}}$

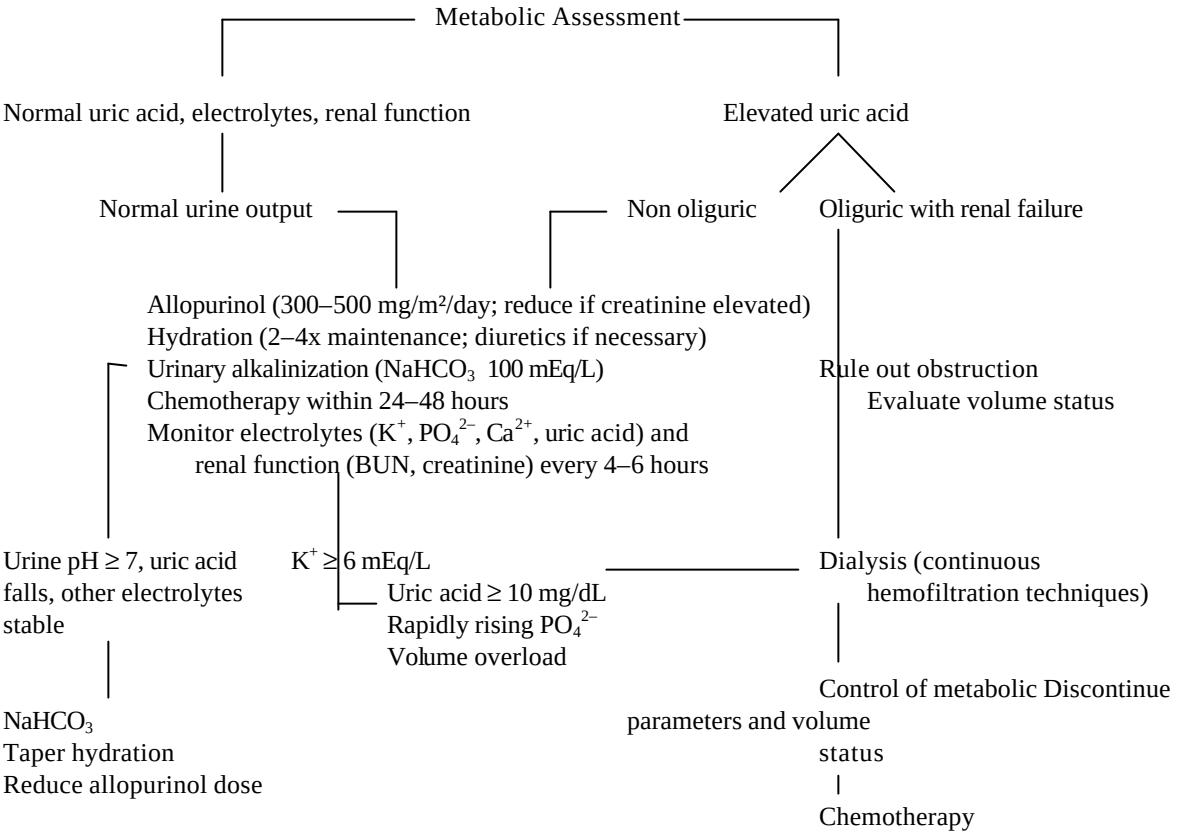


Figure 1. Management of patients with tumor lysis syndrome (Burkitt lymphoma or lymp hoblastic leukemia). Adapted from Ognibene, F. P. and Pizzo P. A., "Oncologic Issues" in Holbrook, P. R., ed., Textbook of Pediatric Critical Care, W. B. Saunders Company, Philadelphia, 1993, p. 809.

LEUKEMIA

I. Introduction

A. Leukemia occurs when a single progenitor cell undergoes malignant transformation giving rise to poorly differentiated blasts which do not differentiate further but replace the normal marrow cells. Acute Lymphoblastic Leukemia (ALL) accounts for 80% of childhood leukemias with approximately 2,500 new cases being diagnosed per year in the United States. Acute Nonlymphocytic Leukemia (ANLL), previously referred to as Acute Myelogenous Leukemia (AML), accounts for the other 20%. ALL is of lymphoid origin while ANLL is of myeloid origin.

II. Clinical presentation

A. Signs and symptoms

1. Fever

2. Pallor - the mean Hgb at diagnosis is 7.0

3. Bruising/ Petechiae - the mean platelet count at diagnosis is 50,000

4. Neutropenia - leukocytosis may be seen secondary to the increased numbers of blasts but this causes a functional neutropenia

5. Hepatosplenomegaly

6. Bone pain

7. Lymphadenopathy

8. Mediastinal mass

B. The most common signs and symptoms are secondary to the marked pancytopenia.

C. The ANC at diagnosis is usually < 1,000.

D. ANLL may present with skin findings.

1. Chloromas are solid collections of myeloblasts. They may occur anywhere but are most common in the epidural space, retroorbital areas, and the skin. When they occur in the skin they are called leukemia cutis or granulocytic sarcoma.

III. Laboratory evaluation

A. Screening labs and tests

1. CBC with differential. **If the patient is neutropenic and has fever he/she needs broad spectrum IV antibiotics (see neutropenia and fever chapter).**

2. Chem 20

a. Creatinine: this serves as a screen for tumor lysis (see that chapter) and renal involvement.

b. LDH as an indirect marker of tumor burden and lysis.

c. Uric acid as a marker of tumor lysis.

d. LFT’s as a screen for hepatic involvement.

e. PT, PTT, fibrinogen: M3 subtype of ANLL associated with DIC.

f. Type and cross for PRBCs and platelets if needed (**ALWAYS** give CMV negative, irradiated and leukofiltered blood products as the patient may be a bone marrow transplant candidate in the future)

g. CXR: T-cell ALL is associated with mediastinal masses.

IV. Complications

A. The most common complications after diagnosis are:

1. Bleeding: treat the patient if the platelet count is < 20,000. Treat DIC if present.

2. Infection: if fever and neutropenia are present treat with the appropriate antimicrobials.

3. Tumor lysis syndrome

4. Leukostasis: this is rare unless the WBC count is > 200,000. Organs at risk are the brain and lungs. CNS symptoms vary from somnolence to stroke. The first pulmonary sign is usually tachypnea, often accompanied by a decreased oxygen saturation. CXR changes are late findings. The treatment is the rapid initiation of chemotherapy. Leukapheresis or exchange transfusion will quickly lower the WBC count. The effect is transient though, and chemotherapy should be started as soon as possible. Prophylactic use of leukapheresis or exchange transfusion should be considered in patients with very high WBC counts.

5. Mediastinal mass: if signs of airway compromise occur the patient may need emergent radiation therapy prior to diagnosis.

NEUTROPENIA AND FEVER

I. PHYSIOLOGICAL ALTERATIONS IN CHILDREN WITH CANCER

A. Altered cellular immunity predisposes patients to infections with intracellular bacteria, fungi, herpes virus group, and protozoa.

B. Altered humoral immunity predisposes patients to encapsulated bacteria.

C. Neutropenia predisposes patients to gram-negative bacilli and *Staphylococcus* species.

D. Mass effect of tumors, tumor invasion, mucositis, medical devices and medical interventions violate physical barriers which normally protect against infection.

II. EVALUATION OF A CHILD WITH FEVER AND NEUTROPENIA

A. History
1. Cough, dyspnea, tachypnea, chest pain.
2. Diarrhea, pain with defecation.
3. Skin sores.
4. Pain with swallowing, sore throat.
5. Exposure to persons with infections (particularly varicella and other herpes viruses).
6. Date of last chemotherapy.
7. Expected interval until the granulocyte count returns to normal.

B. Complete Physical Examination

C. Laboratory Evaluation
1. CBC with differential and platelets.
2. BUN and creatinine.
3. Liver function tests.
4. Cultures all ports of catherters and obtain one peripheral cx.
5. Urinalysis and urine culture. Do not catheterize patient.
6. If patient has meningeal signs discuss LP with heme-onc staff ***prior*** to performance.

D. Radiographic Evaluation
1. Posterior and lateral chest X-rays, if pulmonary symptoms present.
2. Other radiographic studies as clinically appropriate.

III. PRINCIPLES OF ANTIBIOTIC THERAPY

A. Initial therapy must have a broad spectrum of coverage. Options include:

1. **An aminoglycoside plus a semisynthetic penicillin**: Gentamicin, tobramycin, or amikacin, plus ticarcillin or piperacillin. This synergistic combination provides greater bactericidal activity. ***This is the most commonly used regimen.***

2. **Oxicillin and Cefipime.**

3. **Vancomycin, aminoglycoside, and a** b**-lactam**. Expands coverage for gram-positive organisms. **Discouraged as a front line therapy due to emergence of vancomycin-resistant organisms such as *Enterococcus*.** Delaying the addition of vancomycin or nafcillin until confirmation of a gram-positive infection doesn't appear to influence the outcome of therapy.

4. If psuedomonas is documented or likely, double gram negative coverage is indicated.

B. Bacterial isolates and antibiotic susceptibility vary greatly. Familiarize yourself with antibiotic susceptibilities in your institution.

C. Therapy should be safe, with minimal toxic effects.

D. The rate of superinfection or development of resistant organisms should be minimized.

IV. RE-EVALUATION OF INITIAL EMPIRIC THERAPY

A. 24 to 72 hours:
1. Evaluate response to therapy.
2. Optimize treatment based on results of pre-treatment cultures.
3. Modify therapy if persistent bacteremia or development of new symptoms or signs occurs.
4. Continue broad antimicrobial therapy until neutropenia resolves.

B. 5 to 7 days:
1. **Fever and neutropenia resolving**: If infection is documented, give 10 to 14 days of antibiotics. If no infection demonstrated, discontinue antibiotics.
2. **Neutropenia persists**: Continue antibiotics until neutropenia resolves. Because this is associated with an increased risk of bacterial or fungal superinfection, an alternative is to continue antimicrobial therapy for a week, or for 5 days after the fever subsides, whichever is longer, and reinstitute therapy at the first sign of any deterioration in clinical status.
3. **Fever and neutropenia persist**: A diligent search for the source of infection is important. Add amphotericin B and continue throughout the period of neutropenia.

C. Breakthrough bacteremia.
1. Gram-positive isolate: add vancomycin.

2. Gram-negative isolate: assume resistant organism. Switch to a new regimen.

D. Catheter-associated infections.
1. **No evidence of local infection**: Use vancomycin as well as coverage for gram-negative organisms such as an aminoglycoside. Remove catheter if cultures remain positive after 48 hours of appropriate antibiotic coverage. If cultures become negative, do not remove catheter, complete 10 to 14 day antimicrobial course.
2. **Exit site infection without fever or bacteremia**: Culture all ports, peripheral blood and the local catheter site. Treat with vancomycin, an aminoglycoside and a β-lactam. If parenteral therapy fails, remove catheter.
3. **Exit site with fever or bacteremia**: Culture all ports, peripheral blood and local site. If normal ANC, administer vancomycin and aminoglycoside. If neutropenic, administer vancomycin, aminoglycoside and β-lactam. Reassess at 48 to 72 hours. Remove catheter if cultures still positive.
4. **Tunnel infection**: Remove catheter.

E. Severe oral mucositis or necrotizing gingivitis.
1. Cover anaerobic organisms with clindamycin or metronidazole.
2. Perform viral cultures and consider Acyclovir therapy for herpes.

F. Esophagitis.
1. Institute a trial of oral clotrimazole, ketoconazole, fluconazole or IV amphotericin B.
2. Perform viral cultures and start acyclovir.

G. Pulmonary infiltrates.

1. **Patchy or localized**: Continue antibiotics expectantly if ANC rising. If neutropenia persists, perform broncho-alveolar lavage (BAL) or open biopsy and treat according to findings. If a diagnostic procedure cannot be tolerated, begin empiric Amphotericin B.

2. **Diffuse or interstitial**: In patients with neutropenia, perform a BAL or open biopsy, or begin empiric therapy with trimethoprim-sulfamethoxazole, erythromycin and amphotericin B. If normal ANC, institute a trial of TMP-SMZ and erythromycin and do diagnostic procedure only if patient does not improve.

H. Perianal tenderness. In addition to broad spectrum antibiotics, add anaerobic coverage with clindamycin or metronidazole.

SPINAL CORD COMPRESSION

I. ETIOLOGY

A. Compression caused either by local tumor extension or by tumor metastasis.

B. Tumors: lymphoma, neuroblastoma or soft tissue sarcoma most common.

II. PRESENTATION

A. Radicular back pain and motor weakness.

B. Sensory deficits are seen less frequently.

C. Bladder and bowel dysfunction. Incontinence is typical of lesions below L2.

III. DIAGNOSIS

A. Diagnosis is based on demonstration of vertebral lesion with dural compression.

B. Imaging techniques: spinal MRI, contrast myelography if MRI not available.

C. Tissue diagnosis via biopsy.

IV. THERAPY

A. Once compression is documented, urgent therapy aimed at nerve decompression should be initiated.

B. High-dose dexamethasone: 50 mg/M^2 bolus followed by 10 mg/M^2 every 6 hours.

C. Emergency radiation therapy consult. Lymphomas and neuroblastomas are particularly radiosensitive.

D. A neurology consult should be considered to carefully assess the pre-treatment neurologic function. Therefore, any post-treatment improvement will be apparent.

E. Laminectomy is indicated if the etiology is hemorrhage, the tumor is not radiosensitive, or if radiation therapy fails to produce neurologic improvement. Consult neurosurgery.

SUPERIOR VENA CAVA SYNDROME / SUPERIOR MEDIASTINUM SYNDROME

I. FEATURES

A. Extrinsic compression of the superior vena cava usually caused by the mass effect of an anterior mediastinal tumor. Often associated with tracheal compression.

B. Hodgkin's disease, non-Hodgkin lymphoma, and T-cell leukemia are the most common etiologies. Also associated with rhabdomyosarcoma and neuroblastoma when mediastinal involvement occurs.

C. Presentation:
1. Plethora or facial cyanosis.
2. Cyanosis of neck and upper chest.
3. Upper extremity edema.
4. Distended neck veins.
5. Stridor, dyspnea and anxiety if there is associated airway obstruction.
6. Neurologic symptoms due to increased ICP: headache, stupor, coma, or seizures.
7. Cardiac compromise with shock due to decreased venous return and/or decreased ventricular volume.

II. EVALUATION

A. History and physical exam.

B. Radiographic evaluation:
1. Chest X-ray – reveals clinically significant mediastinal mass.
2. Chest CT – determines the extent of the mass. Must be performed without sedation.
3. Echocardiogram - to evaluate for thrombi from stasis of flow

C. Tissue diagnosis is necessary to implement specific therapy.
1. Non-sedated bone marrow biopsy and/or superficial lymph node biopsy.

2.High risk of death during intubation, especially if greater than 50% reduction in trachea size documented on CT scan. **A skilled bronchoscopist must be on hand if intubation is attempted. Call pediatric pulmonology, pediatric surgery, or ENT.**

3.Establish diagnosis with least invasive measures. Be aware of potential for curculatory and respiratory failure associated with anesthesia or sedation.

4.Empiric therapy is initiated if no tissue is available and the risk of intubation is too great. It may include radiation, steroids, and/or chemotheray.

PEDIATRIC RESUSCITATION

I. Shock: a clinical state characterized by inadequate delivery of oxygen and metabolic substrates to meet the metabolic demands of tissues. May occur with normal, increased or decreased blood pressure. In the first two cases shock is compensated.

II. Etiologies

A. Blood loss/Volume loss

1. Whole blood loss

2. Hemorrhage

a. External

b. Internal (GI bleed, splenic/hepatic laceration, fractures, intracranial bleed, major vessel injury).

3. Relative loss

a. Fluid/Electrolyte loss (vomiting, diarrhea, DKA, DI, etc).

b. Plasma loss (burns; sepsis with capillary leak; 3rd spacing from peritonitis, intestinal obstruction, hypoproteinemia).

c. Vasodilatory drugs (morphine, valium, dilantin, etc).

d. Endocrine (adrenal insufficiency).

e. Renal (post obstructive diuresis - esp. with posterior urethral valves).

B. Cardiogenic Shock - decreased cardiac output.

1. Drug intoxication

2. Dysrhythmia

3. Acidosis

4. Pericardial effusion

5. Pneumothorax

6. Congenital heart disease

7. Cardiomyopathy

8. Ischemia

C. Anaphylactic shock

D. Spinal shock - secondary to spinal cord injury with resultant vasodilation and flaccid paralysis.

E. Septic shock- a combination of hypovoemic, cardiogenic, and vasodilatory shock.

III. Evaluation

A. A rapid cardiopulmonary assessment is necessary if any of the clinical signs of early shock or conditions predisposing to shock are present:

1. Respiratory rate > 60 **(normal RR's are in Status Asthmaticus chapter)**

2. Heart rate **(normal HR's are in Cardiac Dysrhythmias chapter)**

a. > 180 or < 80 (under 5 years)

b. > 160 or <60 (over 5 years)

3. Respiratory distress

4. Trauma

5. Burns

6. Cyanosis (peripheral or central)

7. Failure to recognize parents

8. Diminished consciousness

9. Seizures

10. Fever with petechiae

B. Evaluation of Respiratory Performance:

1. Possible respiratory arrest should be anticipated in infants and children who have:

a. Increased respiratory rate or respiratory effort

b. Diminished breath sounds

c. Diminished level of consciousness or pain response

d. Poor muscle tone

e. Cyanosis

f. Tachypnea without distress may be a compensatory mechanism in:

(1). Septic shock

(2). DKA

(3). Inborn errors of metabolism

(4). Severe diarrhea

(5). Salicylate overdose

(6). Chronic renal failure

g. A slow respiratory rate in an ill patient is ominous and may indicate:

(1). Hypothermia

(2). Fatigue

(3). CNS depression

h. A decreasing RR may indicate a tiring child.

2. Respiratory Mechanisms:

a. Increased work of breathing (nasal flaring; intercostal, subcostal and suprasternal retractions).

b. Grunting

c. Head bobbing

d. Stridor

e. Prolonged expiratory phase

f. All are important signs of impending respiratory failure.

3. Cyanosis: A late and important sign of respiratory failure best seen in the oral mucosal membranes and the nail beds. Cyanosis of the extremities is usually due to circulatory failure.

C. Evaluation of Cardiovascular Response: Organ perfusion is determined by cardiac output and perfusion pressure. HR and BP are easily determined, while stroke volume and peripheral vasculature resistance must be assessed by examining pulses and tissue perfusion. Remember septic and anaphylactic shock may be associated with increased cardiac output and normal BP. **NORMAL BP DOES NOT EXCLUDE SHOCK!** When vascular resistance is low, perfusion will appear normal with bounding pulses and wide pulse pressure.

1. Heart rate

a. Tachycardia - may be a response to hypoxia, hypercapnia, hypovolemia or fever. Newborns increase cardiac output mainly by increasing HR.

b. Bradycardia is an ominous sign of arrest

2. Blood pressure - can be maintained by vasoconstriction, tachycardia, and increased cardiac contractility. When these fail, hypotension ensues. It is a late sign of cardiovascular decompensation.

a. A formula to estimate the 50th percentile of systolic BP in children (over 5 yoa) is 90 + (2 X age in years)

b. The lower limit of systolic BP is
70 + (2 X age in years)

c. If systolic BP falls 10 mg Hg then begin prompt, frequent evaluations for shock.

d. **For normal BP values for age see Hypertensive Emergency chapter**

3. Peripheral circulation - discrepancies between central (carotid, brachial, femoral) and peripheral (radial, post tib, DP) pulses may represent vasoconstriction from hypothermia or redistribution of blood flow due to decreased CO.

D. End Organ Perfusion - skin, brain, kidneys.

1. Skin: decreased perfusion is an early sign of shock, and should be assessed with the following signs:

a. Usually the extremities are warm and pink to the distal phalanx. As output drops, a line between warmth and coolness will ascend to the trunk.

b. Slow capillary refill (> 2 sec) with the extremity elevated above the level of the heart.

c. Mottling, pallor, and peripheral cyanosis.

2. Brain: Signs of sudden decrease in perfusion include:

a. Failure to recognize or be consoled by parents.

b. Decreased muscle tone

c. Seizures

d. Pupillary dilatation

3. Kidney: History of decreased output is useful, but is more

important in the long term management. Adequate output is 1cc/kg/hr (minimum) in infants, and 300 cc/m2/24 hrs in older children.

IV. Labs

Initial evaluation is clinical, treatment is initiated BEFORE lab results are available. Initial studies on shocky patients should include:

A. ABG (see Golden Rules below)

B. Hematocrit (spun) then CBC, diff, PLTS

C. Lytes, BUN, Cr, Ca, Glucose

D. Immediate dextrostix - only takes a drop of blood and you get the result faster than a lab glucose.

E. Other labs (LFT's, ammonia, drug levels, etc) as dictated by situation.

F. PT/PTT/fibrinogen if sepsis/bleeding are considered.

G. Determine Anion Gap: A.G. = Na^+ - (Cl^- + HCO_3^-) = unmeasured cations minus unmeasured anions. Normal = 12 $\pm$ 2 mEq/Liter. (see poisoning chapter as well)

1. Causes of increased A.G: hypokalemia, hypocalcemia, hypomagnesemia (decreased unmeasured cations) or lactate, ketones (DKA), phosphate, sulfate, transient hyperalbuminemia, salicylates, formates, nitrates, penicillins, paraldehyde, inborn errors, ethylene glycol, methanol, uremia, hyperosmolar hyperglycemic nonketotic coma (increased unmeasured anions), alcohol, toluene, carbon monoxide, iron, isoniazid, strychnine, cyanide or lab errors: falsely increased Na^+ or decreased Cl^- or HCO_3^-.

2. Causes of decreased A.G: hyperkalemia, hypercalcemia, hypermagnesemia, IgG, THAM, lithium (increased cation), hypoalbuminemia (decreased unmeasured anion) or lab error: falsely decreased Na^+, increased Cl^- or HCO_3^-.

3. Causes of normal A.G. acidosis: loss of HCO_3^- (diarrhea, pancreatic fistula, enterostomies, renal tubular acidoses, carbonic anhydrous inhibitors, hyperchloremic metabolic acidosis.

V. Therapy

A. Airway

1. Provide maximal 02

2. Protect the airway - intubate early if any concerns for compromise (seizures, coma, etc). Remember to pass an OG/NG tube if you use BVM > 2 minutes.

3. High PEEP may be needed to oxygenate some patients, especially in septic shock with capillary leak.

4. In the awake patient intubation is most successful using rapid induction (see that section).

5. Airways:

a. Nasal cannula - only used if modest O_2 requirement is present. Flows > 6 liters/min are irritating to nasopharynx. Humidification is problematic.

b. Oxygen Hood - usually used in children $\leq$ 1 year. Well tolerated. Humidification is possible.

c. Oxygen mask - delivers 35-60% O_2 at flow rates of 6-10 liters/min.

d. Face Tent - often better tolerated than a face mask. Not reliable for FiO_2 > 0.40.

e. Oral airways - holds tongue away from posterior pharynx. Only used in unconscious patients (occasionally used for Pierre-Robin or choanal atresia in conscious neonates but may induce gagging). Size: place flange at level of central incisors with bite block parallel to the hard palate. The tip of the airway should reach the angle of the jaw. Invert as insert (not necessary in neonates), then rotate as airway approaches the posterior pharynx. Do not obstruct airway by pushing the tongue posteriorly. May also be inserted without inverting the oral airway by using a tongue depressor. This is probably preferable to avoid trauma. Proper head and jaw position must be maintained even once oral airway is in place. Causes gagging and may lead to vomiting in conscious patients which may cause aspiration. Remove once patient can guard airway.

f. Nasopharyngeal airway - soft rubber or plastic tube. Tolerated well. May be used in responsive patients. Outside diameter should not be so large that it causes sustained blanching of ala nasae. Length: measure distance from tip of nose to tragus of ear. Lubricate when insert. May damage adenoidal tissue resulting in a nose bleed that may compromise airway.

g. Esophageal Obturator Airway - not recommended for pediatric patients.

B. Cardiac/Vascular

1. Unless real concerns exist for cardiogenic shock, "prime the pump" with 20 cc/kg of isotonic fluid (LR, NS or 5% albumin.) Remember that in liver disease LR causes increased lactate levels and acidosis.

2. Large amounts of fluid may be necessary for resuscitation. Children with septic shock may require > 100 cc/kg in the first 24 hours. Give whatever is needed (or as one esteemed intensivist was heard to say- stop counting, keep pushing).

a. If no change in status after initial bolus then repeat as necessary.

b. Place a CVP lines and follow CVP to guide therapy (if low, give fluid, if high and still hypotensive or porrly perfused, consider inotropes). Also, assess for physical signs of volume overload such as rales, increased liver size, distended neck veins, and gallop. A CXR will help to assess heart size.

c. If CVP rises without improved perfusion after fluids, then consider pump failure and need for inotropes/pressors. An echo can be helpful to assess cardiac function. Consider placement of a pulmonary artery catheter to better assess left heart preload and cardiac output.

3. IV access- see PALS manual. **Don't forget intraosseous access in emergencies, especially in children < 6 years old!**

a. To place an intraosseous cannula: use sterile technique, identify the tibial tuberosity, insert the intraosseous cannula 1 - 3 cm below (distal) to the tuberosity on the medial side of the tibia, direct the needle perpendicular to the long axis of the bone or slightly caudal (toward the toes) to avoid the epiphysial plate, use a firm twisting motion. **Do NOT use an IO in a fractured bone.**

b. Stop advancing the needle when you feel a sudden decrease in resistance as the needle enters the marrow cavity. The needle is in the marrow cavity if:

(1). You felt the decrease in resistance as you entered the marrow cavity.

(2). The needle can remain upright without support.

(3). Marrow can be aspirated (looks red and grainy) - not always achievable.

(4). Fluid flows freely without subcutaneous infiltration.

VI. Specific Agents - (**for doses see algorithms and code drug sections inside front and back covers**).

A. Inotropes/Vasopressor

1. Indicated in patients with hypotension or poor perfusion despite volume resuscitation.

2. Most agents are sympathomimetic and work via alpha or beta receptors.

a. Beta-1 receptors: found mostly in cardiac myocytes, stimulation resoults in increased HR and contractility.

b. Beta-2 receptors: found primarily in blood vessels and bronchi, stimulation results in dilation.

c. Alpha-1 receptors: found primarily in blood vessels, stimulation results in constriction.

3. Specific Agents

a. Dopamine

(1). Acts on alpha, beta, and dopaminergic receptors at different dosage ranges.

(a). dopaminergic effects: 1-3 mcg/kg/min, increased renal perfusion and splanchnic blood flow.

(b). beta-1 effects: 4-10 mcg/kg/min, increased HR and contractility.

(c). alpha-1 effects: 10-20 mcg/kg/min, vasoconstriction.

(2). Uses

(a). Hypotension or poor perfusion unresponsive to fluid resuscitation.

(3). Precautions

(a). Side effects include tachycardia, dysrhythmias, hypertension, extravasation necrosis (treat with Regitine).

(b). $\geq$ 50% of dopamine's action is indirect via nerve uptake and conversion to norepi. Sick children may not have adequate intrinsic catecholamine stores to release. Thus dopamine may be ineffective, and epinephrine may be a better drug in children with shock.

b. Epinephrine-The drug of choice in post arrest situations and in most pediatric pateints with severe shock.

(1). Actions – Acts on alpha and beta receptors

(a). beta-1: 0.05-0.3 mcg/kg/min, increased HR and contractility.

(b). alpha-1: 0.3-5 mcg/kg/min, vasoconstiction

(2). Uses

(a). Similar to dopamine. May be effective when dopamine is not, as all actions are direct.

(b). Symptomatic infant or child with bradycardia unresponsive to ventilation and oxygen administration.

(3). Precautions

(a). Similar to dopamine. Also causes increased myocardial oxygen consumption. May cause decreased splanchnic perfusion.

c. Norepinephrine

(1). Actions - primarily alpha effect, increases systemic vascular resistance.

(2). Used in shock with hypotension despite volume resuscitation and adequate inotropic support. Sometimes used as a first line vasopressor in septic shodk

(2). Precautions - potential for decreased regional perfusion.

d. Dobutamine - Actions

(1). Actions- primarily Beta 1 effect, 5-20 mcg/kg/min, increased HR, increased contractility, vaso**dilation.**

(2). Used for congestive heart failure or to treat hypoperfusion associated with high vascular tone. Contraindicated in patients with hypotension.

d. Milrinone

(1). Actions- a phosphodiesterase inhibitor, Milrinone exhibits both inotropic and vasodilatory effects by inhibiting phosphodiesterase III resulting in increases in myocyte and vasular smooth muscle cAMP.

(2). Used for CHF, cardiac dysfuction and afterload reduction s/p cardiac surgery, and in shock with cardiac dysfuction and high vascular tone.

(3). Dose: 50 mcg/kg load, followed by infusion of 0.5-1mcg/kg/min.

(4). Precautions: Rarely causes thrombocytopenia. Potential for hypotension.

B. Other agents

1. Lidocaine

a. Most useful drug for ventricular ectopy as it decreases automaticity.

b. Indications - VFib, Vtach or symptomatic ventricular ectopy.

c. Precautions:

(1). reduce the infusion rate in shock, CHF, cardiac arrest, liver dysfunction.

(2). toxicity = myocardial/circulatory depression, CNS symptoms include seizures.

2. Bretylium

a. Effective in VFib unresponsive to usual ACLS support.

b. Not well studied in pediatrics.

3. Atropine

a. Actions - parasympatholytic

(1). Accelerates sinus/atrial pacemakers

(2). Increases AV conduction

b. Indications

(1). Symptomatic bradycardia

(2). Vagally mediated bradycardia (from intubation).

c. Precautions

(1). Smaller than vagolytic dose may cause paradoxical bradycardia (min dose 0.1 mg).

(2). May mask hypoxia induced bradycardia.

4. Calcium - indicated only with documented hypocalcemia, and in treatment of hyperkalemia, hypermagnesium, and Ca-channel blocker

overdose. **GIVE SLOWLY to avoid bradycardia.**

5. NaHCO3

a. Use with caution, preferably with ABG as a guide.Consider use when acidosis is not correcting with other methods of resuscitation. Base deficit X weight (**in kg**.) X 0.3 = the mEq's of bicarb to completely correct the acidosis.

b. Adequate ventilation must be assured or it will aggravate respiratory acidosis.

c. Results in intracellular and CSF acidosis. Shifts oxyHgb curve to the left with impaired tissue oxygen delivery.

d. Don't mix with Ca or catecholamine infusions.

6. Antimicrobials - in suspected septic shock give antibiotics immediately. PUSH the antibiotics and initially give half the full daily meningitic dose. **The following are first doses** in septic patients, to be followed by maintenance doses:

a. Neonate: Ampicillin 100 mg/kg and Gentamicin (1.25 to 2.5 mg/kg depending on weight) or instead of Gent., Cefotaxime 100mg/kg. Meningitis doses for Amp. and Cefotaxime are 200 mg/kg/day.If already on antibiotics or has indwelling lines, consider Vancomycin and Amikacin.

Neonatal Dosages (mg/kg) and Intervals

Antibiotic	**Route**	**Wt < 1200 g**	**Weight 1200 - 2000 g**		**Weight > 2000 g**	
		Age 0-4 wks	**Age 0-7 days**	**> 7 days**	**Age 0-7 days**	**> 7 days**
Amikacin	IV, IM	7.5 q 18-24 hrs	7.5 q 12-18 hrs	7.5 q 8-12 hrs	10 q 12 hrs	10 q 8 hrs
Vancomycin	IV	15 q 24 hrs	15 q 12-18 hrs	15 q 8-12 hrs	15 q 12 hrs	15 q 8 hrs
Ampicillin	IV, IM	50 q 12 hrs	50 q 12 hrs	50 q 8 hrs	50 q 8 hrs	50 q 6 hrs
Cefotaxime	IV, IM	50 q 12 hrs	50 q 12 hrs	50 q 8 hrs	50 q 12 hrs	50 q 8 hrs

b. Infants 1-2 mos: Ampicillin 100 mg/kg and Cefotaxime 100 mg/kg. If already on antibiotics or has lines in, consider Vancomycin and Amikacin (see following table). **Consider Acyclovir 25 - 50 mg/kg/day IV divided q 8 hrs.**

c. Children > 2 mos use:

a. Ampicillin 100 mg/kg and

b. Cefotaxime 100 mg/kg or

c. Ceftriaxone 100 mg/kg

e. If already on antibiotics or has lines in consider Vancomycin and Amikacin (see table below). **Consider Acyclovir 25 - 50 mg/kg/day IV divided q 8 hrs.**

Dosages (mg/kg) and Intervals for Children and Older Infants

Antibiotic	Route	Dosage	Interval
Amikacin	IV, IM	15 - 22.5 mg/kg/day	q 8 hrs
Vancomycin	IV	40 mg/kg/day (meningitis 60mg/kg/day)	q 6 hrs
Ampicillin	IV, IM	100 - 200 mg/kg/day (meningitis 200 - 400 mg/kg/day)	q 6 hrs
Cefotaxime	IV, IM	100 - 150 mg/kg/day (meningitis 200 mg/kg/day)	q 6 - 8 hrs
Ceftriaxone	IV, IM	50 - 100 mg/kg/day (meningitis 100 mg/kg/day)	q 12 - 24 hrs (q 12 hrs for meningitis)

7. FFP for bleeding difficulties (see that section).

8. 0 neg blood can be used for emergent blood replacement if no crossmatched blood is available.

POISONING - GENERAL MANAGEMENT

The clinician should maintain a high index of suspicion to be able to arrive at the often difficult diagnosis of poisoning. **Strongly consider an ingestion in any patient with an unexplained loss of consciousness. For any questions or for a discussion with a toxicologist call the Poison Control Center at 1 - 800 - POISON - 1**

I. Seven Phases of Poisoning Management. **Emergency stabilization of the patient comes first.**

A. First, treat the patient, not the poison !!

B. ABC's of resuscitation then add "D" for:

C. Disability;

1. Perform a brief neurologic exam, establish the level of consciousness (Glasgow Coma Scale), determine pupillary size and reactivity.

2. Institute drug therapy: oxygen, dextrose, naloxone as indicated.

3. Consider decontamination: ocular, dermal, GI, etc.

II. Clinical evaluation (see specific chapters in this manual as well)

A. Symptom complexes (toxidromes) may give clues to an unknown poisoning. **(also see tables at end of chapter). Not all findings may be present ! Mixed ingestations may present with confusing findings.**

Drug Class	Level of Consciousness	Pupils	Vital Signs	Miscellaneous
1. Anticholinergics (atropine, cyclic antidepressants, antihistamines)	Delirium, psychosis, seizures, coma	Mydriasis (dilated)	↑HR, ↑T, ↑BP, Arrhythmias - cyclic anti-depressants	Flushing, hot skin, dry skin, hyperreflexia, urinary retention
2. Sympathomimetics - (amphetamines, cocaine)	Agitated, tremors, psychosis, hyper-active, seizures	Dilated	↑HR, ↑BP, ↑T	Sweaty, delirium
3. Opiates (narcotics, clonidine)	Euphoria, coma	Pinpoint	↓RR ,↓HR, ↓BP, ↓T	Shallow respirations, hyporeflexia
4. Organophosphates	Sedated, coma	Miosis (pupill-lary constric-tion)	↑↓HR, ↑↓BP	"S.L.U.D." - salivation, lacrimation, urination, defecation, also fasciculation, bronchorrhea
5. Barbiturates,	Confusion, coma,	Miosis or	↓RR, ↓BP,	Ataxia, nystagmus,

sedatives-hypnotics, ethanol	slurred speech	Mydriasis	↓T	hyporeflexia
6. Phenothiazines-Haloperidol	Sedated, coma, tremor, seizures	Miosis	↓BP, ↓T, ↑HR	Dystonic rxns, ataxia, back arching, trismus, torticollis
Drug Class	**Level of Consciousness**	**Pupils**	**Vital Signs**	**Miscellaneous**
7. Salicylates	Lethargy, seizures, coma	-	↑RR, ↑T	Vomiting, tinnitis, met. acidosis
8. Theophylline	Agitation, tremor, seizures	-	↑RR, ↑HR, ↓BP	Nausea, vomiting, hypokalemia, met. acidosis
9. Methanol, ethylene glycol	sedated, visual disturbances	-	↑ RR	oxalate crystals in urine (ethylene glycol), osmolal gap

Signs or symptoms may also provide valuable clues to identifying the agent.

Sign or Symptom	Associated Drugs
Bradycardia	β-blockers, cyclic antidepressants, calcium channel blockers, clonidine, digoxin, thioridizines, mesoridizine, nicotine, carbamates, organophosphates, opiates
Tachycardia and myocardial irritants	amphetamines, sympathomimetics, cocaine, cyclic antidepressants, caffeine, theophylline, propoxyphen, beta agonists, digoxin, thioridizines, mesoridizine, anticholinergics, procainamide, carbon monoxide, cyanide, freon solvents, organophosphates, phenothiazines
Hypotension	diuretics, β-blockers, ACE inhibitors, calcium channel blockers, clonidine, imidazoles, serotonin reuptake blocking antidepressants, cyclic antidepressants, thioridizines, mesoridizine, caffeine, theophylline, propoxyphen, beta agonists, quinine, quinidine, NSAIDS, isuprel, nicotine, carbamates, organophosphates, carbon monoxide, cyanide, nitrites, opiates, barbiturates, ethanol, phenothiazines
Hypertension	amphetamines, sympathomimetics, cocaine, MOI, phencyclidine, clonidine, imidazoles, nicotine, anticholinergics, carbamates, organophosphates
Hypoglycemia	insulin, alcohol, oral hypoglycemic agents, aspirin, β-blockers
CNS depression	opiates, sedative - hypnotics, anticonvulsants, antipsychotics, antidepressants, anticholinergics, iron, phencyclidine, lithium, carbamates, organophosphates, freon, carbon monoxide, cyanide, salicylates, ethanol, methanol, ethylene glycol
Seizures	NSAIDS, amphetamines, sympathomimetics, cocaine, tegretol, antidepressants, anticholinergics, iron, theophylline, lithium, phencyclidine, organophosphates, isoniazid, phenothiazines, carbon monoxide, cyanide, camphor, strychnine, salicylates
CNS agitation	anticholinergics, amphetamines, sympathomimetics, caffeine, theophylline, salicylates, cocaine,

	phencyclidine, carbon monoxide

B. History - focused and complete.

1. Substance or substances - including ingredients. Meds in house.

2. Maximum possible amount (number in bottle originally - number left)

3. Estimate ingestion - usually grossly underestimated.

4. Estimated time of ingestion.

5. Symptoms.

6. Home treatment.

7. Significant PMH: hobbies (glue exposure ?), recurrent episodes etc.

8. Parents vocation.

C. PE - Vitals, level of consciousness (GCS), motor function, eyes (pupils, EOM, fundi), mouth (lesions, odors) heart (rate, rhythm), lungs (rate, pattern), skin, odors (breath, clothing), **can the patient maintain the airway ?, does the patient have a gag ?.**

D. Major modes of presentation - COMA, cardiac arrhythmias, metabolic acidosis, GI disturbances, seizures.

E. Lab exam (individualize) CBC, lytes, BUN, creat, glucose, calcium, dextrostix, LFT's serum osmolality, ABG, EKG, CXR, KUB, urine and blood for TOX screen, drug levels if intoxicant known.

F. Calculate anion gap:

1. $[Na^+]$ - $([HCO3^-]$ + $[C1^-])$ = A.G.

2. Normally 12 - 14 mEq/Liter

3. Causes of increased anion gap (see Peds. Resus. chapter as well), Mnemonic: "CAT MUD PILES"

C - Cyanide, carbon monoxide
A - Alcohol
T - Toluene
M - Methanol
U - Uremia
D - DKA
P - Paraldehyde
I - Iron, Isoniazid, Inborn errors of metabolism

L - Lactic Acidosis
E - Ethylene glycol
S - Salicylates, Strychnine

G. Calculate osmolality:

1. $2[Na^+] + [glucose/18] + [BUN/2.8] = Osm.$

2. **Osmolal gap** = lab osmolality - calculated osmolality

3. Normally < 10 mOsm

4. **Osmolal gap** is increased with acetone, ethanol, ethylene glycol, isopropyl alcohol, mannitol, methanol, propylene glycol

III. Elimination of the poison from the GI tract, skin and eyes

A. Gastric emptying

1. Syrup of Ipecac - usually used at home, rarely used after presenting to medical facility.

a. Dose - < 1 year = 1 cc/kg
1 to 12 years = 15 ml
> 12 years = 30 ml

b. Follow with water or juice (induction of emesis will be delayed if given with milk) - may repeat once if no emesis in 30 minutes- keep emesis for analysis

c. Contraindications:

- lost gag reflex, decreased level of consciousness, seizures

- ingestion of agent that rapidly depresses mental status (cyclic antidepressants, hypnotics, strychnine)

- ingestion of caustic agent

- petroleum distillate/hydrocarbon ingestion

- Nissen fundoplication

- < 6 months of age

2. Gastric lavage - usually used for extremely toxic substances, in cases of unknown ingestions or when loss of consciousness is present

a. When patient is unable to protect their own airway, intubate before proceeding

b. Place large bore OG/NG tube (16 - 36 Fr)

c. Confirm placement by auscultation, place patient in left lateral decubitus position (left side down) with head lowered. Consider the use of a bite block in older patients.

d. Warm saline is instilled in aliquots until stomach contents are clear.

e. Contraindicated - alkalis, sharp objects, pills larger than lavage hose.

B. Activated Charcoal

1. Almost irreversibly absorbs drugs and chemicals, preventing absorption.

2. Consider for all significant toxic ingestions; poorly binds Fe and Lithium, not to be used in caustic ingestions because of poor binding and makes endoscopy difficult.

3. Dose = 1 gm/kg or 30-60 gm for children and 60-100 gm in adults. Prepared as a slurry with a ratio 1:4 charcoal to water. Goal is to have a charcoal to toxin ratio > 10:1 .

4. Repetitive doses of charcoal (1 gm/kg q 4-6°) will help clear enterohepatic circulation of some drugs (carbamazepine, digoxin, phenobarb, salicylates, theophylline).

5. Cathartics such as sorbitol (5 ml/kg) can be used with first dose of charcoal to prevent constipation.

6. Cathartics should not be used repetitively as it will cause fluid and electrolytes disturbances.

IV. Antidotes **(see tables at end of chapter)**

A. Use of specific antidotes is invaluable; unfortunately few poisons have antidotes

B. Contact poison control for specific antidotes and doses

V. Elimination of the Absorbed Substance

When indicated, the following 5 modalities can be used:

A. Supportive therapy (while the patient metabolizes the particular poison). Intubation, ventilation, even ECMO!

B. Forced diuresis (falling out of favor)

1. When used with pH modification, patient needs close monitoring for toxicity.

2. 1 1/2 - 2 X maintenance (3000 cc/M^2/day) (see DKA or Acute Tumor Lysis chapters for M^2 nomogram or calculation)

3. U.O. should approach 3-6cc/kg/hr

C. Alkalinization

1. Ingestions of phenobarbital, salicylate.

2. 0.5-2 mEg/kg/hour IV NaHCO3 - titrate to keep urine pH 7.5-8.0.

D. Acidification

1. Used for ingestions of amphetamine, chloroquine, lidocaine, quinidine

2. Ammonium chloride 75 mg/kg/day ÷ q 4-6° p.o. (contraindication - hepatic insufficiency)

3. Keep urine pH 5.5 -6.0

E. Adsorbent hemoperfusion and dialysis (consult nephrology)

1. Dialysis has been used for many substances, **some** of which are: ammonia, amphetamines, anilines, antibiotics, barbiturates, boric acid, bromides, calcium, chloral hydrate, ethylene glycol, fluorides, iodides, isoniazid, meprobamate, methanol, paraldehyde, potassium, quinidine, quinine, salicylates, strychnine, thiocyanates

VI. Supportive Therapy and Observation

The support of respiration, circulation, and other vital functions takes priority over all other aspects of therapy.

VII. Disposition

A. May involve medical and/or psychiatric follow-up (if inpatient psychiatric treatment is necessary the psych. residents will arrange)

B. Consider social service involvement

C. "Preach" preventive medicine

SUMMARY OF ANTIDOTES

POISON	ANTIDOTE(S)
Acetaminophen	N-Acetylcysteine (Mucomyst) initial dose of 140 mg/kg PO in water, cola, juice or soda: then, 70 mg/kg q 4 hr for 68 hrs (17 doses, 18 total doses), see chapter
Anticholinergics	Physostigmine (adult, 2 mg; child, 0.5 mg) IV; may repeat in 15 min. until desired effect is achieved; subsequent doses q 2 - 3 hrs. prn. (**CAUTION:** may cause seizures, asystole, cholinergic crisis)
Anticholinesterases	Atropine 2-5 mg (adults); 0.05-0.1 mg/kg (in children) IM or IV, repeated q 10-15 min until atropinization is evident;
Organophosphates	Pralidoxime chloride 1-2 grams (adults);25-50 mg/kg (in children) IV; repeat dose in 1 hr prn, then q 6-8 hrs for 24-48 hrs. Consider constant infusion.
Carbamates	Atropine as above; pralidoxime for severe cases
Benzodiazepines	Flumazenil 0.01 mg/kg IV, max. dose 3 mg (estimated pediatric dose)
Beta-adrenergic blockers	Glucagon 50 micrograms/kg IV
Calcium channel blockers	Calcium chloride 10%, 10 ml (adult); 0.2 ml/kg (pediatric) IV or Calcium gluconate 10%, 30 ml (adult); 0.6 ml/kg (pediatric) IV Glucagon 50 micrograms/kg IV
Carbon monoxide	Oxygen 100% inhalation, consider hyperbaric for severe cases
Cyanide	Adult: Amyl nitrate inhalation (inhale for 15-30 sec every 60 sec) pending administration of 300 mg sodium nitrite (10 ml of a 3% solution) IV slowly over 2-4 min., follow immediately with 12.5 grams sodium thiosulfate (2.5-5 ml/min of 25 % solution) IV Children: (Na nitrite should not exceed recommended dose because fatal methemoglobinemia may result, see below)

Hemoglobin	Initial dose 3% Na nitrite IV	Initial dose 25% Na thiosulfate IV
8 g	0.22 ml (6.6 mg)/kg	1.10 ml/kg
10 g	0.27 ml (8.7 mg)/kg	1.35 ml/kg
12 g (nl)	0.33 ml (10 mg)/kg	1.65 ml/kg
14 g	0.39 ml (11.6 mg)/kg	1.95 ml/kg

Digitalis	Fab antibodies (Digibind): dose based on amount ingested and/or digoxin level (see pkg. insert)
Ethylene glycol	(see methanol)
Fluoride	Calcium gluconate 10%, 0.6 ml/kg IV slowly until symptoms abate, serum calcium normalizes, repeat prn

POISON	ANTIDOTE(S)
Heavy metals/usual chelators	
Arsenic / BAL	BAL (dimercaprol): 3-5 mg/kg/dose deep IM q 4 hours for 2 days, every 4-6 hours for an additional 2 days, then every 4-12 hours for up to 7 additional days
Lead / BAL, EDTA,(+ penicillamine), DMSA, (see chapter)	EDTA 50-75 mg/kg/24 hours deep IM or slow IV infusion given in 3-6 divided doses for up to 5 days,; may be repeated for a second course after a minimum of 2 days; each course should not exceed a total of 500 mg/kg body weight Penicillamine 100 mg/kg/day (max. 1 gram) PO in divided doses for up to 5 days; for long term therapy do not exceed 40 mg/kg/day DMSA (succimer) 350 mg/M^2 (10 mg/kg) PO every 8 hours for 5 days, followed by 350 mg/M^2 (10 mg/kg) PO every 12 hours for 14 days
Mercury / BAL, DMSA	
Iron	Deferoxamine: 5-15 mg/kg/hr IV; use higher dose for severe symptoms and decrease as patient recovers (see chapter)
Isoniazid	Pyridoxine 5-10%, 1 gram per gram of INH ingested IV slowly over 30-60 min.
Methanol and Ethylene Glycol	Ethanol, loading dose to achieve blood level of 100 mg/dl Adult: 0.6 grams/kg + 7-10 grams to be infused IV over 1 hour Children: 0.6 grams/kg to be infused over 1 hour Maintenance doses should approximate 10 grams/hour in adults and 100 mg/kg/hour in children, to be adjusted according to measured blood ethanol levels **ALSO:** Folate 50 -100 mg IV every 6 hours (methanol) Thiamine 0.5 mg/kg and pyridoxine 2 mg/kg for ethylene glycol
Methemoglobinemic agents	Methylene blue 1%, 1-2 mg/kg (0.1-0.2 ml/kg) IV slowly over 5-10 min if cyanosis is severe or methemoglobin level is > 40%
Opioids	Naloxone 0.1 mg/kg IV, IM sublingual or via ETT
Phenothiazines (dystonic reaction)	Diphenhydramine 1-2 mg/kg slow IV or IM Max. dose 300 mg/day
Warfarin (and superwarfarin	Vit. K adult: 10 mg; children: 1-5 mg, slow IV,

__rat poisons)_____IM, SQ, or PO_____ __

 # Adapted from: Fleisher, GR et al: Textbook of Emergency Medicine (3rd ed.) Williams and Wilkins, Baltimore, 1993, pg's 756-757

TOXIC SYNDROMES

SYNDROME	MANIFESTATIONS	TYPES
Anticholinergic	**"mad as a hatter, red as a beet, blind as a bat, hot as a hare, dry as a bone"** **Parasympatholytic:** dry skin/ mucous membranes, thirst, dysphagia, blurred vision (near objects), fixed, dilated pupils, tachycardia, hypertension, flushing, scarletiniform rash, hyperthermia, abdominal distention, urinary urgency and retention **Central:** lethargy, confusion, delirium, hallucinations, delusions, ataxia, respiratory failure, cardiovascular collapse, extrapyramidal movements	Belladonna alkaloids, atropine, scopolamine, plants (jimson weed, nightshade, mushrooms, Jerusalem cherries), phenothiazines Synthetic:Glycopyrrolate Others: Antihistamines, cyclic antidepressants
Anticholinesterase	**Muscarinic:** sweating, constricted pupils, lacrimation, wheezing, cramps, vomiting, diarrhea, tenesmus, bradycardia, hypotension, blurred vision, urinary incontinence, excessive salivation **Nicotinic:** Striated muscle: fasciculations, cramps, weakness, twitching, paralysis, respiratory compromise, cyanosis, cardiac arrest **Sympathetic ganglia:** tachycardia, hypertension **Central:** anxiety, restlessness, ataxia, convulsions, insomnia, coma, absent reflexes, Cheyne-Stokes breathing, respiratory/ circulatory depression	Organophosphates, carbamate insecticides
Cholinergic	see anticholinesterases;	Acetylcholine, betel nuts,

	nicotinic and muscarinic	bethanecol, muscarine, pilocarpine
Extrapyramidal	**Parkinsonian:** dysphonia, dysphagia, oculogyric crisis, rigidity, tremor, torticollis, opisthotonos, shrieking, trismus	Chlorpromazine, haloperidol, perphenazine, promazine, thioridazine, trifluoperazine
Hemoglobinopathy	disorientation, headache, coma, dyspnea, cyanosis, cutaneous bullae, gastroenteritis	Carboxyhemoglobin (carbon monoxide), methemoglobin, sulfhemoglobin
Metal fume fever	chills, fever, nausea, vomiting, muscular pain, throat dryness, headache, fatigue, weakness, leukocytosis, respiratory distress	**Fumes of oxides:** brass, cadmium, copper, iron, magnesium, mercury, nickel, titanium, tungsten, zinc
Narcotics	CNS depression, pinpoint pupils, slowed respirations, hypotension Response to naloxone: pupils may dilate and excitement may predominate	Codeine diphenoxylate (Lomitil), fentanyl, heroin, morphine, opium, oxycodone
Narcotic **withdrawl**	diarrhea, mydriasis, goose bumps (piloerection), hypertension, tachycardia, insomnia, lacrimation, muscle cramps, restlessness, yawning, hallucinosis	Cessation of: alcohol, barbiturates, benzodiazepines, chloral hydrate, glutethimide, meprobamate, methaqualone, narcotics, opioids, paraldehyde
Sympathomimetic	CNS excitation, convulsions, hypertension, tachycardia	Aminophylline, amphetamines, caffeine, cocaine, dopamine, ephedrine, epinephrine, fenfluramine, levarterenol, methylphenidate, pemoline, phencyclidine

Ref: Adapted from Done AK: Poisoning- A Systematic Approach for the Emergency Department Physician, in Emergency Medicine, Tintihalli et al. eds. McGraw Hill, 1985

ACETAMINOPHEN POISONING

I. Metabolism

A. Sulfation, glucuronidation, CP-450 pathway

1. Only CP-450 pathway produces toxic metabolites (especially as glutathione stores are depleted in toxic ingestions).

2. Main toxicity is to the liver, children (< 6 y/o) are less susceptible to liver damage than adults.

B. Most common toxic ingestion in U.S.A.

C. Readily absorbed.

II. Clinical Course

A. Stage 1 (0-24 hrs post-ingestion).

1. Nausea, vomiting, diaphoresis, anorexia.

2. CNS depression is rare unless a coingestion exists.

B. Stage 2 (24-48 hrs) initial manifestation of liver injury.

1. Clinically improved.

2. SGOT, SGPT, bili, PT begin to rise.

C. Stage 3 (48-96 hrs), maximal hepatotoxicity

1. Asymptomatic to fulminant hepatic failure.

2. AST values > 20,000 not unusual.

3. Fatalities occur between 3 and 5 days; survivors reach stage IV.

D. Stage 4 (4 days - 2 weeks)

1. Resolution of hepatic dysfunction usually complete by one week.

E. Patients who are treated late or who are untreated and eventually die, maintain high levels of liver enzymes beyond 72-96 hrs.

F. Miscellaneous (unusual)

1. Hematologic - coagulopathy secondary to liver dysfunction (anemia, thrombocytopenia, agranulocytosis, methemoglobinemia with phenacetin)

2. Hypersensitivity (maculopapular rash, laryngeal edema, urticaria, angioedema, anaphylaxis)

3. Renal (ATN, ADH effect, acute renal failure) occurs in 25% of cases with documented hepatotoxicity

4. Cardiomyopathy/myocarditis

5. Metabolic (hypoglycemia, metabolic acidosis, hyperammonemia)

6. Dermatitis

7. Neurologic (cerebral edema, herniation)

III. Management

A. Assessment of toxicity of ingestion

1. History

a. Toxicity unlikely for dose < 150 mg/kg in children or 7.5 grams in adults.

b. History often unreliable (lack of knowledge, faulty recollection, active deception).

2. Acetaminophen level.

a. Draw at least 4 hrs post ingestion.

b. If you can get level back quickly, then await results and plot on nomogram, treat for level above lower line. (see R.M. nomogram at end of chapter)

c. If level not available quickly, then begin therapy and continue or discontinue **depending on result of 4 hour level.**

d. Only initial (4 hour) blood level is used.

e. Subsequent blood levels that may fall below the line are NOT an indication to terminate treatment.

B. Specific therapy

1. Empty stomach via Ipecac or lavage as indicated.

2. Treat any other coingestant with the standard approach.

3. Draw baseline LFT's, PT, lytes, BUN, Cr, Glucose, tox. screen.

4. Activated charcoal should be used if less than 4 hours have elapsed since ingestion or if a mixed ingestion is suspected.

a. Activated charcoal will bind N-acetylcysteine (NAC, mucomyst) but with the large doses of NAC administered, there’s only a small decrease in bioavailability. Gastric lavage of the charcoal **IS NOT** necessary prior to administration of NAC.

b. NAC is rapidly absorbed in stomach and proximal small bowel so most efficacious if given a short time after charcoal.

c. NAC (N-Acetylcysteine, , Mucomyst) promotes glucuronyl system hopefully preventing CP-450 toxin accumulation.

5. Prepare NAC as 5% solution in water, grapefruit juice or a cola beverage (it will taste and smell terrible).

6. NAC dose - 140 mg/kg loading followed by 70 mg/kg q 4 hrs x 17 doses (18 doses total).

a. No difference exists in the incidence of hepatotoxicity if NAC is given anytime within 8° after ingestion.

b. Standard of care has been to treat as late as 24 hours after ingestion.

c. May be beneficial to treat later than 24 hours especially if still can detect acetaminophen level. (indicates initial elevation)

7. If any dose is vomited within one hour of administration, repeat the dose.

8. If emesis becomes a constant problem, the NAC may be administered by DUODENAL intubation or may give antiemetics (eg. metoclopramide 1 - 2 mg/kg/dose q 2 - 6 hours IV, metoclopramide may cause extrapyramidal sx. so premedicate with diphenhydramine 5 mg/kg/day divided q 6 hours PO, NG, or use another antiemetic such as phenergan 0.25 - 0.5 mg/kg/dose PO, NG, or PR q 4 - 6 hours prn, adult dose is 12.5 - 25 mg q 4 - 6 hours).

9. SGOT, SGPT, Bili, PT should be followed daily for at least 4 days if a significant ingestion occurred.

a. If abnormal, follow till normalize.

b. If normal, then repeat at 8 days.

ASPIRIN POISONING

I. Pharmacokinetics

A. Absorption

1. Therapeutic doses are rapidly absorbed in stomach; gastric emptying is **delayed** in overdoses. In large ingestions the tablets may form concretions that can remain in the stomach for many hours. They may slowly release salicylates and prolong the toxicity. Gastric lavage with epigastric or even endoscopic manipulation may be necessary to break up and remove such masses.

2. Enteric-coated preps delay absorption even longer; peak serum salicylate levels can occur up to 60 hours after an excessive ingestion.

B. Metabolism and Excretion

1. 80 % of a normal dose is conjugated with glycine and glucuronic acid in liver.

2. These pathways are rapidly saturated, even at therapeutic plasma salicylate concentrations.

3. After their saturation, renal excretion becomes increasingly important.

C. Distribution

1. Unbound, nonionized fraction of salicylate crosses membranes including the blood - brain barrier. The ionized form does **not** cross the BBB.

2. As pH decreases, nonionized salicylate increases (fall in pH from 7.4 to 7.2 doubles the nonionized form).

3. This is an important consideration during treatment with alkalinization.

II. Clinical Features

A. Pathophysiologic Effects

1. Direct CNS respiratory center stimulation --> hyperpnea and respiratory alkalosis.

2. Uncoupling of oxidative phosphorylation causes:

a. increased heat production ---> hyperpyrexia

b. failure to produce high energy phosphates (e.g. ATP)

c. increased oxygen utilization and CO2 production due to increased skeletal muscle metabolism

d. increased tissue glycolysis

e. CNS particularly sensitive; may have decreased glucose in CNS even in the face of normal blood glucose levels!!

3. Stimulation of lipid metabolism --> ketone body formation

4. SIADH --> oliguria, fluid overload

5. Hemostatic effects

a. Decreased prothrombin and factor VIII formation

b. Decreased platelet aggregation

c. Actual hemorrhagic manifestations are in practice very uncommon

6. Electrolyte abnormalities

a. Hypernatremia common

b. Assume total body K+ depletion, even if [K+] normal

B. Signs/Symptoms

1. Asymptomatic - No objective signs

2. Mild

a. **CLASSIC TRIAD:** vomiting, hyperpnea, hyperpyrexia
ALWAYS consider the possibility of a salicylate ingestion when these coincide in a patient.

b. Tinnitus

c. Hypocapnia without frank acidosis

3. Moderate

a. Severe hyperpnea and marked lethargy and/or excitability

b. Usually no coma or convulsions

c. Compensated metabolic acidosis in child

4. Severe

a. Coma, possibly convulsions

b. Uncompensated metabolic acidosis in child after 12 hours

5. Children under 4 y.o. almost invariably have Acidosis. Acidosis may alter the estimation of the severity of the intoxication.

III. Management

A. Assessment of severity of poisoning

1. Altered consciousness is the most important indicator of severe salicylate intoxication.

2. By history: (although clinical condition is usually the best guide)

a. < 150 mg/kg is usually non-toxic

b. 150 - 300 mg/kg: mild to moderate toxicity

c. 300 - 500 mg/kg: severe toxicity

d. > 500 mg/kg: potentially lethal

3. "DONE" nomogram (see at end of chapter)

a. Only useful in single dose, acute ingestion

b. **Not useful** if ingestion occurred over several hours or days, if the preparation is enteric coated or sustained release, if the compound is Oil of Wintergreen, if the patient has renal insufficiency, or if the patient is acidemic.

c. An initial “nontoxic” level may evolve into severe ASA poisoning. **GET MORE THAN ONE LEVEL !!** In large overdoses salicylate levels should be obtained q 4 - 6 hours to determine that no further absorption is occurring.

B. Specific Measures

1. Induce emesis or lavage - significant amounts may be present in stomach up to 12 - 24 hours (longer with time released products). Enteric coated pills are larger and may not be removed with lavage. X-rays may help as *some* preps are radiopaque - however, if X-rays are negative it does not rule out an ingestion.

2. Activated charcoal - multiple doses may be superior to a single dose

a. Dose: 1 - 2 grams/kg followed by 20 - 60 grams q 3 - 4 hours until passage of charcoal stool.

3. Saline cathartic

4. Blood for - salicylate level (6 hrs after ingestion), lytes, glucose, ABG, PT/PTT, ionized Ca++

   a. Monitor labs frequently (q 4 - 6 hours), consider arterial line

   b. Repeat salicylate levels until downward trend established, draw q 1 - 2 hours until levels are declining and the patient's clinical condition stabilizes.

5. Treat shock if present, use fluid resuscitation as needed, condition is likened to DKA except glucose containing fluids are used.

6. Forced alkaline diuresis (urine output of 3 - 6 cc/kg/hour) to increase ASA excretion.

   a. Use D5 1/2 NS + 15 mEq NaHCO3/L at 3000 cc/M^2 /day (see DKA chapter or Acute Tumor Lysis chapters for nomogram and calculation of body surface area)

   b. Add 30 mEq KCL/L after good urine output obtained.

   c. HCO3 must be used with caution, especially in children >4 y.o. who will often have respiratory alkalosis and blood pH > 7.40, even in the face of low HCO3 levels.

   d. Aim of HCO3 therapy is to maintain blood pH 7.45 - 7.50 and urine pH > 7.5.

   e. Watch for metabolic alkalosis, increased Na, decreased K, decreased Ca with tetany, hypoglycemia.

7. Hemorrhagic tendency

   a. Vit K, 5-10 mg IM

   b. Guaiac all stool/emesis

   c. FFP for severe bleeding

8. Hyperthermia - sponging, cooling blanket

9. If none of your interventions succeed, consider exchange transfusions, or hemodialysis. Obtain a nephrology consult early on in the course. This is especially true in cases where the salicylate level is rising, or if evidence of end-organ damage has developed such as pulmonary edema, CNS deterioration, persistent acidemia, or coagulopathy.

CAUSTIC INGESTIONS

I. Epidemiology

A. Most commonly seen between 1 - 3 years (peak 18 - 24 mos.)

B. May be accidental in children and the mentally retarded - volume is generally small because of immediate and severe pain with the ingestion.

C. Deliberate in adults as suicide attempt with large ingested volumes.

D. Rarely a cause of child abuse.

E. Most common exposure is to household bleaches - relatively neutral pH makes them less irritating than other alkalis or acids.

F. Other frequent exposures include automatic dishwasher agents, laundry detergents, swimming pool products, toilet bowl and oven cleaners. (see table at end of chapter)

II. Pathogenesis

A. Esophageal burns account for the most serious injuries and complications - liquid Drano™, lye, and ammonia are the most caustic

B. Injuries to lips, oropharynx and upper airway occur.

C. Solids adhere to mucosa producing deep burns of the oral cavity and esophagus - less likely, however, to reach stomach

D. Powders tend to injure upper airway causing stridor and epiglottitis

E. **Alkaline agents - (pH > 7)**

1. Liquefaction necrosis and early disintegration of the mucosa cause deep penetration leading to perforation

2. Depth of injury is related to concentration of the agent and duration of contact with the mucosa

3. Small amounts of alkaline substances with pH > 11 may cause severe burns

4. Agents with pH’s of 9 - 11 rarely cause serious injury unless large amounts are ingested

5. In the first week inflammation and vascular thrombosis cause additional destruction

6. In the second week granulation tissue forms with weakening of the esophageal wall making it susceptible to perforation

7. Beyond the third week fibrogenesis and stricture formation occurs which makes perforation less likely

F. **Acidic agents, also called corrosives - (pH < 7)**

1. Coagulation necrosis forms a coagulum on the mucosa limiting deeper penetration

2. Alkaline pH of the squamous epithelium of the esophagus is protective of esophageal damage but not totally in all cases (see II,F,5. below)

3. Greater amounts reach the stomach causing gastric injury and perforation

4. Pooling in the antrum with antral spasm causes injury in the "prepyloric" area

5. Up to 20 % of acid ingestions also cause esophageal burns

G. Batteries - small button type

1. Most measure 7.9 - 11.6 mm and pass spontaneously

2. Larger ones ($\geq$ 15.6 mm) may lodge in the esophagus and leak their caustic contents

3. They contain silver or mercuric oxide, manganese dioxide, zinc, or lithium

III. Clinical presentation

A. **The presence or absence of oral lesions or symptoms does NOT predict the presence or severity of esophageal or gastric burns.**

B. Dysphagia is the most common symptom. It results from alterations in peristalsis secondary to esophageal irritation.

C. Drooling

D. Retrosternal or abdominal pain

E. Stridor, hoarseness, nasal flaring and retractions

F. Epiglottitis may be severe (especially in children < 2 y.o.) and may require intubation !!

G. Vomiting and hematemesis

IV. Initial management - (see flow sheet at end of chapter also)

A/B. AIRWAY and BREATHING

1. Respiratory distress requires oral intubation

2. Cricothyroidotomy or tracheotomy may be necessary if tracheal visualization is impossible

C. Circulation

1. Hypotension and shock (? perforation) require fluid resuscitation

2. GI hemorrhage may require blood products

D. Gastric decontamination is **contraindicated**

1. Induction of vomiting (ipecac) will reexpose the esophageal mucosa to the caustic agent

2. Blind placement of a nasogastric tube for purposes of lavage may cause additional injury or even perforation of the injured mucosa

E. Neutralizing substances are **discouraged**

1. Neutralization may cause heat production and further injury

F. Dilution with milk or water is **not** recommended

1. The volume needed to dilute the caustic agent is too great

2. Additional vomiting may occur with large amounts of a diluting agent

3. Clearly **CONTRAINDICATED** in the presence of respiratory distress

4. Not beneficial if $\geq$ 1 hour after ingestion

G. Labs: ABG, CBC with diff, electrolytes, Ca++, BUN, creat, type and cross

H. X - rays - of chest and abdomen

1. Perforation - see subdiaphragmatic free air, mediastinal emphysema

2. Impending perforation - gastric dilatation, intramural air

3. Pulmonary aspiration

I. Early consultation with Peds GI or Peds surgery is **MANDATORY**

V. Endoscopy

A. Usually performed in any suspected ingestion although observation may be considered if the ingestion was questionable or if agent was household bleach

B. Done within first 24 - 48 hours after the ingestion

C. If done < 12 hours, may not see full extent of injury but does not change treatment

D. Done under general anesthesia with severe burns, resp. distress, or if rigid scope used

E. Flexible scope may be placed past damaged esophageal mucosa into stomach

F. Rigid scope is not advanced beyond first significant burn to avoid risk of causing perforation

G. Allows grading of esophageal burns

1. First degree: limited to edema and erythema

2. Second degree: linear ulceration and necrotic tissue with white plaques

3. Third degree: circumferential injury with sloughing of the mucosa

VI. Treatment

A. Nasogastric tube

1. **<u>ONLY</u> to be placed under direct visualization at time of endoscopy**

2. Used with extensive circumferential burns or in possibilities of perforation

3. Provides route for nutritional support

4. Maintains lumen during stricture formation

5. Serves as guide for esophageal dilatation after stricture forms

B. Corticosteroids are controversial (used to prevent strictures)

1. May be beneficial in first or second degree burns

2. Dose used is equivalent to prednisone 1 - 2 mg/kg/day (max. 60 mg/day) for 3 - 4 weeks; given IV until patient taking medication orally

3. NOT indicated in third degree burns since strictures are inevitable; any mediastinitis or infection secondary to perforation would be masked as well

4. NOT indicated in acid ingestions since gastric injury is more common; would mask gastric necrosis and perforation

5. Prophylactic antibiotics are generally used with steroids in second degree burns because of an increased risk of infection, especially bacterial spread to the mediastinum

C. Antibiotics

1. Used in second or third degree burns even if steroids are not used

2. Recommendation: Unasyn 50 mg/kg/day divided q 6 hours

3. Gentamicin use is +/-, 5 mg/kg/day divided q 8 hours

D. UGI is done 3 - 4 weeks after the injury, earlier if obstruction or dysphagia are present

E. Batteries

1. Evaluated by X-ray

a. Prompt removal if in esophagus

b. If battery is past the esophagus F/U X-ray after 3 - 4 days

VII. Early complications

A. Systemic

1. Airway obstruction, ARDS

2. Shock

3. Nutritional failure

4. Infection

B. Gastrointestinal

1. Perforation

2. Pyloric obstruction from edema

3. Hemorrhage

VIII. Delayed complications

A. Strictures

1. Primary complication

2. Occur in most third degree burns, less likely in second degree burns

3. 80 % will have obstructive symptoms by 2 months

4. Repeated dilatation is required

5. < 50 % will have success with dilatation and will require colonic interposition

B. Pyloric stenosis

1. Occurs with both acids and alkalis

2. Sx’s develop over 3 to 10 weeks

3. Treatment is surgical bypass or balloon dilatation

C. Esophageal carcinoma

1. Incidence approximately 5 %

2. Detection of cancer has occurred as late as 16 - 42 years after the ingestion

CYCLIC ANTIDEPRESSANT OVERDOSE

I. Introduction:

A. Cyclic antidepressants include bicyclics, tricyclics and tetracyclic forms. While showing undeniable clinical efficacy, their narrow therapeutic and toxic range creates a dilemma for physicians. Cyclic antidepressants include the tricyclics amitriptyline, doxepin, imipramine, trimipramine, amoxapine, desipramine, nortriptyline and protriptyline, the tetracyclic maprotiline, the monocyclic bupropion, the triazolopyridine trazodone and the serotonin reuptake inhibitors fluoxetine, sertraline, and paroxetine.

B. Death rate from overdose 2.2%.

C. Accounts for 25% of all drug overdose hospital admissions.

D. Most common cause of death from prescription drug overdose.

II. Pharmacology - 3 ring aromatic nucleus with aliphatic aminoprophyl side chain.

A. Absorption

1. Rapidly absorbed in alkaline small intestine with rapid distribution.

2. Absorption delayed in overdose because of delayed gastric emptying and decreased GI motility from the anticholinergic drug effect.

3. Significant enterohepatic circulation leading to decreased excretion.

4. Up to 95% of the drug is protein bound at physiologic pH and is inactive. Hypoalbuminemia and acidemia increase free drug.

B. Metabolism

1. Almost entirely metabolized in liver.

2. Half-life variable - 10 to 81 hrs. This long half-life is due to an extremely large vol. of distribution from extensive tissue distribution of the drug and its' active metabolites. This is why neither dialysis, nor hemoperfusion are effective in removing significant quantities.

C. Mechanism of action

1. Peripheral and central anticholinergic effect. Block muscarinic - cholinergic and alpha-adrenergic receptors.

2. Block reuptake of released norepinephrine and serotonin at

presynaptic adrenergic nerve endings. Leads to biphasic effect with initial excess of catecholamines followed by depletion.

a. Initial accumulation of norepi --> tachycardia and hypertension (alpha effects). This may precipitate dysrhythmias.

b. Later norepi depletion --> bradycardia and hypotension (alpha blockade) or depletion. May see decreased contractility and/or decreased vasomotor tone.

3. Inhibits fast Na^+ channel resulting in slowing of depolarization.

4. Direct effect on myocardium - with above --> disturbances in excitability and conduction defects --> almost any arrhythmia! Often see a quinidine - like conduction delay (wide QRS tachycardia indistinguishable from V. Tach). Bradydysrhythmias are ominous. AV blocks may also occur.

5. Earliest toxic sign is probably a QRS > 0.10 sec.

6. Death is usually due to cardiac complications.

III. Clinical Presentation

A. Anticholinergic effects (less pronounced with doxepin, desipramine, amoxapine and maprotiline while trazodone has little or none).

1. Tachycardia, mild HTN, blurry vision, mydriasis (often poorly reactive), urinary retention, pulmonary edema, hypoventilation. Priapism is common with trazodone.

2. Dry mouth, fever, hallucinations, agitation, ataxia, absent bowel sounds (ileus).

3. Myoclonic twitching, seizures (especially with amoxapine or maprotiline) coma, flaccid paralysis hyperreflexia. Seizures more common if QRS > 0.16 sec. in one study.

4. Diff. Dx. of these effects includes many other antihistamines, antispasmodics, plants, etc.

5. Earliest signs usually tachycardia, dry mouth, ileus, mydriasis, and altered mental status.

B. Cardiotoxic effects (see above) - include cardiac arrhythmias, hypotension, and pulmonary edema. More common if QRS > 0.16 sec.

IV. Management

A. Assessment of Severity of Ingestion

1. Toxic effects are not necessarily dose dependent. Any ingestion should be considered life threatening!!

2. Ingestion of $\geq$ 1000 mg generally assoc. with severe toxicity.

3. Toxic symptoms may be seen with a dose of 10 mg/kg but are the rule when ingestion >20 mg/kg.

4. As mentioned, QRS > 100 msec (0.10 sec.) => severe toxicity.

B. Specific Measures

1. ICU, "ABC's"!! BEWARE of RAPID onset of s/s! A decrease in level of consciousness and loss of airway control may produce a respiratory acidosis leading to increased free drug concentrations!

2. Continuous cardiac monitoring, pulse oximetry. Make sure you have secure IV access. EKG.

a. At least 24-48 hrs of monitoring.

b. Continue until dysrhythmia free for at least 24 hrs.

3. Labs: Stat lytes, BUN, creat, glucose, ABG, drug screen for coingestants, and EKG. Drug levels not helpful as no correlation exists between plasma levels and symptoms of serious poisoning.

4. Consider foley

5. Drug removal

a. Gastric lavage - can be helpful even 24 hrs after ingestion. Ipecac not recommended as patients are frequently lethargic and may rapidly deteriorate to coma, seizures or dysrhythmia.

b. Charcoal - Delayed gastric emptying and enterohepatic recirculation make GI decontamination very important. Dose 1 gram/kg initially then 1 gram/kg every 4-6 hours for 24-48 hours if GI motility is present and patient is symptomatic.

c. Cathartic - sorbitol.

d. Left lat. decub. position may decrease gastric emptying.

e. Again, because of marked protein binding and large volume of distribution, forced diuresis, dialysis, exchange transfusion and hemoperfusion are not indicated.

6. Drug reversal

a. Physostigmine - acetylcholinesterase inhibitor, previously touted to reverse cholinergic symptoms, **NOT RECOMMENDED** due to potential side effects including seizures, heart block,

hypotension, bradycardia/asystole, respiratory distress, excess salivation, sweating, diarrhea

7. Management of BP

a. Hypertension rarely necessitates treatment

b. For hypotension, correct acidosis, vasopressors (phenylephrine, norepinephrine) for refractory hypotension.

8. Correction of arrhythmias

a. **NaHCO3** is the treatment of choice. A bicarb drip 1-2 meq/kg/hour is initiated and titrated to keep the serum pH 7.45-7.55 in comatose pts, pts with QRS > 100 msec or arrhytmias. Alkalization should also be considered in pts with a suspected large ingestion. The drip is continued until the pt is stable and has a normal EKG for at least 24 hrs.

9. Ventricular arrhythmias

a. First try NaHCO3 bolus (1 mEq/kg).

b. Lidocaine if NaHCO3 fails (1 mg/kg then drip 20-50 micrograms/kg/min).

c. Propranolol, dose 0.1 mg/kg, slow IV - may lead to hypotension, bradycardia or asystole in cyclic antidepressant overdoses so only use for life-threatening tachycardias after other measures have failed.

d. Phenytoin (Dilantin) use controversial. Has been used successfully for vent. dysrhythmias and conduction delay. Dose 15 mg/kg over 15-20 min. (max. 50 mg/min).

10. Bradycardia

a. Atropine ineffective due to muscarinic receptor blockade and has been associated with onset of asystole.

b. Complete heart block, Mobitz II heart block, and refractory bradycardia are indications for insertion of a temporary pacemaker. However, the patient may be resistant to pacemaker capture.

11. Other:

a. If all these measures fail consider ECMO to support the patient until detoxification occurs. WHMC was the first ECMO center to report this <u>successful</u> application.

b. Resuscitation efforts should continue for a minimum of one hour. Full recovery of cyclic antidepressant induced cardiac arrest after five hours is documented.

12. Contraindicated - (cyclic antidepressants cause conduction delays which these drugs may worsen).

a. Quinidine

b. Procainamide

c. Disopyramide

d. Flecanide

e. Encainide

f. Propafenone

13. Correction of seizures - may be intractable especially with amoxapine.

a. Valium or lorazepam drug of choice. Flumazenil contraindicated (associated with increased incidence of seizures).

b. Can also use phenobarb, paraldehyde.

c. Use of naloxone, thiamine, dextrose in patients with coma or altered mental status in ED is considered standard of practice prior to antiepileptic treatment.

14. Scheme for monitoring.

a. Asymptomatic patient who has had stomach emptied and charcoal - monitor 24 hrs in ICU.

b. Non-cardiac S/Sx.s - monitor till Sx. free.

c. EKG changes - monitor for at least 24-48 hrs after normal EKG.

HYDROCARBON INGESTIONS

I. Introduction

A. Petroleum distillates are comprised of aliphatic and aromatic hydrocarbons. Contamination with heavy metals or toxic chemicals is common. Most hydrocarbons are petroleum distillates, however, some such as turpentine are derived from pine oil. The large number of hydrocarbon containing products, bright labels and ‘fruity’ fragrances all increase children’s exposure to these products.

II. Pathogenesis:

A. Incidence of pulmonary complications differs depending on hydrocarbon derivative. High volatility, (volatility means the tendency of a liquid to form a gas) decreased viscosity and low surface tension distillates are more likely to be aspirated leading to respiratory injury (see chart at end of chapter). Lower viscosity enhances distal airway penetration. Lower surface tension facilitates spread.

B. Pulmonary injury usually occurs either from aspiration or direct installation into the lung not from GI absorption. (Some GI absorption occurs but is usually minimal.)

C. Small ingestions may result in aspirations with significant pathology.

D. The intentional abuse by inhalation of gasoline or paint thinners is usually not associated with significant pulmonary toxicity.

III. Pathology

A. Pulmonary injury is of principle importance.

B. Lungs show interstitial inflammation, atelectasis, hyperemia, vascular thrombosis, bronchial and bronchiolar necrosis, intra-alveolar hemorrhage, edema, bronchospasm and emphysema.

C. Hydrocarbons directly injure pulmonary tissue and capillaries and inhibit surfactant activity.

D. Lipoid pneumonias which are usually more localized, may occur with higher viscosity agents.

E. Bacterial pneumonias may supervene but are less frequent than once thought. (probably result from aspiration of oral microorganisms).

F. Local mouth or pharynx irritation, nausea and emesis occur most frequently with fuel or furniture polish ingestions.

G. Intravascular hemolysis and hemoglobinuria have been reported with gasoline ingestions.

IV. Clinical Effects:

A. Respiratory signs/symptoms predominate.

1. Choking, coughing, hemoptysis, SOB, dyspnea, cyanosis, tachycardia, tachypnea, nasal flaring, grunting, retractions, rhonchi, wheezes, rales all may be present.

2. Cyanosis may occur rapidly as alveolar gas is displaced by hydrocarbon vapors. Hypoxia, hypercarbia may occur.

3. Prolonged, persistent cough is suggestive of aspiration and requires evaluation.

B. CNS

1. Somnolence is the chief neurologic manifestation. Coma and convulsions may occur.

2. Lethargy, dizziness, stupor, coma or seizures may indicate the presence of toxic additives (insecticides or aromatic hydrocarbons), a large ingestion or serious aspiration.

C. Gastrointestinal

1. Spontaneous vomiting is common (up to 40% of cases) and is associated with aspiration.

2. Local irritation to mouth etc. is common. Melena is rare.

3. Bloating, flatus, abdominal pain, liquid feces, all may occur.

4. Minimal, if any, absorption by the GI tract occurs and the agent is usually eliminated in the stools.

5. Oral antacids or H2 - receptor antagonists may precipitate vomiting and are usually not given in the unintubated patient.

D. Fever

1. High fever is common. It is usually due to chemical pneumonitis, but may be due to occur with secondary infection (pneumonia).

E. Other

1. Hepatosplenomegaly is rare. Etiology unclear. May be due to a toxic additive effect.

2. Cardiac dysrhythmias are rare.

V. Evaluation and Treatment

A. ABC's FIRST!! Perform complete history and physical exam next.

B. All patients who vomited spontaneously after the exposure or are in respiratory distress or whose parents cannot observe the patient or who you are not sure about should be admitted to the PICU. TOTALLY asymptomatic children with a normal CXR after 6 hours may be followed as an outpatient only if frequent telephone calls (every 2 hours x 6) are possible, the parents are reliable and will return immediately if symptoms occur and if okayed by ED or pediatric staff. Twenty-four hour observation in the PICU is preferred in the vast majority of cases.

C. Patients with respiratory distress should receive oxygen and an ABG. Serial CXR's (every 6 hours), IV access and continuous cardiorespiratory monitoring should all be obtained. An arterial line should be placed if frequent ABG's are needed.

D. Labs: CBC with differential, liver function tests and electrolyte panels should be drawn. If the ingestion was deliberate consider tox screens. If the patient is intubated, serial tracheal aspirates for gram stain and culture should be considered if a bacterial pneumonia is suspected.

E. CXR: Positive findings in > 65% in the first 6 hours after ingestion. Abnormalities may occur as early as 30 minutes after exposure or as late as 12 hours. Resolution of radiographic findings usually lags behind improving clinical status.

1. Most common findings: bilateral basilar infiltrates (65%), right basilar infiltrates (30%), fine, punctuate perihilar densities (5%).

2. Patchy densities that may coalesce to form larger areas of consolidation, atelectasis, emphysema, pleural effusions, pneumatoceles, pneumothoraces, pneumomediastinum and sub Q emphysema are all reported.

3. CXR may be markedly abnormal even in the presence of a normal exam!

F. Intubation: Consider if:

1. Moderate to severe respiratory distress.

2. ABG abnormalities; paO2 <60 on 6 liters O_2 or paCO2 >50mmHg.

3. Deteriorating mental status

4. Absent breath sounds

5. Cyanosis on 40% FiO2

6. Exhausted patient leading to decreased respiratory effort.

G. Most authorities usually recommend AGAINST gastrointestinal decontamination in hydrocarbon ingestions to reduce the chances of

lavage induced emesis and subsequent aspiration. Activated charcoal does not bind most petroleum distillate products or other hydrocarbons so is of little benefit.

1. Ipecac induction of emesis is likewise usually not indicated to avoid aspiration. **HOWEVER:**

2. Products contaminated with pesticides, heavy metals or toxins such as anilene or nitrobenzene should be evacuated. In the alert child who can guard his airway (see Poisoning - General Management Chapter for dose) some people use syrup of Ipecac, but an elective intubation and lavage may be safer. In the unconscious or stuporous patient protect the airway first with a cuffed endotracheal tube then perform gastric lavage.

H. Bronchodilators as needed.

I. Steroids have repeatedly been shown to be ineffective in preventing the development of pneumonitis or in its' treatment.

J. Prophylactic antibiotics are not routinely prescribed.

1. Antibiotics may be necessary later in the course in the face of persistent fever (> 36 hours), leukocytosis (> 36 hours), clinical deterioration or a positive tracheal gram stain or culture.

2. Antibiotics should cover for mouth and GI flora: H. influenza, Staph aureus, Strep pneumoniae etc. Usual choices are Cefuroxime, Ceftriaxone, Clindamycin or Penicillin G.

3. HFOV or ECMO have both been used successfully in severe cases. The longest ECMO air transport in history was performed by WHMC for a hydrocarbon ingestion/aspiration.

VI. Prognosis

A. Majority recover fully.

B. May predispose to increased risk of future respiratory infections.

C. 75% may have PFT abnormalities.

IRON POISONING

I. Pathophysiology

A. Local effects:

1. Direct corrosive mucosal lesions of stomach and proximal small bowel (early events)

2. Mucosal irritation, ulceration, bleeding, ischemia, infarction and perforation may occur. Perforations are usually late events.

3. Produces profound fluid losses and hypotension

B. Systemic effects:

1. Venodilation

a. Results from elevated serum and tissue iron levels

b. Causes secondary hypotension with low CVP

2. Increased capillary membrane permeability leads to edema

3. Inhibition of serum proteases

a. Iron directly inhibits thrombin thereby prolonging PT unrelated to liver dysfunction

b. The coagulopathy in later stages is secondary to hepatic dysfunction

4. Metabolic acidosis

a. Results from poor perfusion with increased anaerobic metabolism secondary to hypotension, anemia from GI bleeds, and hypovolemia

b. Liberation of H+ from conversion of iron from ferrous to ferric state

c. Poisoning disrupts oxidative phosphorylation with resulting anaerobic glycolysis and lactic acidosis

5. Cellular damage

a. Liver - greatest risk for injury because of first pass effect

b. Renal failure

c. Coma / seizures

C. Liver effects:

1. Cloudy swelling, portal damage, fatty degeneration, Kupffer/parenchymal deposition of iron

2. Once iron accumulates in the hepatic mitochondria it cannot be removed nor can the pathology be reversed

D. Other (much less frequent) effects:

1. Pancreatic injury

2. Bleeding in lungs/kidney

3. Fatty degeneration of heart and renal tubules

II. Clinical Presentation

A. Three stages of acute phase (in severe ingestions)

1. Early (< 6 hrs)

a. Abdominal cramps, vomiting, diarrhea, and GI hemorrhage

b. Hypotension, pallor, lethargy

c. Leukocytosis, metabolic acidosis, hyperglycemia

2. Intermediate (6 to 48 hrs)

a. Transition between resolution of GI symptoms and overt systemic toxicity

b. May continue to recover or progress to stage three

3. Late (2 to 3 days) - multiple organ failure

a. Cerebral dysfunction and coma

b. Myocardial dysfunction with vascular collapse or shock

c. Massive hepatic failure with jaundice, increased transaminases, coagulopathy, and hypoglycemia

d. Renal failure

e. Ischemic bowel

f. Pulmonary edema

4. Sequelae

a. Intestinal scarring, gastric outlet and small bowel obstruction

b. Hepatic cirrhosis, rare in children

III. Management

A. Assess severity of ingestion

1. Assess amount of **elemental iron**

a. Ferrous fumarate = 33%

b. Ferrous sulfate = 20% hydrated, desiccated or dried = 30%

c. Ferrous gluconate = 11.6%

d. Ferrous chloride = 28%

2. Average lethal dose = 180 mg elemental Fe/kg

a. Minimum lethal dose as little as 600 mg

b. Total doses of 200-400 mg have caused severe symptoms in young children

c. **Toxic dose generally > 20 mg/kg**

3. Plasma level peaks at 2-6 hrs post-ingestion (mean = 4 hrs). After 6 hours the iron has been rapidly cleared from the serum, primarily by the liver.

- later with enteric coated preparations

4. Iron levels - if readily available. Vomiting, diarrhea, hyperglycemia (> 150 mg/dl), and leukocytosis (> 15,000) that develop in the 6 hours after ingestion and an abdominal radiograph that demonstrates the presence of radiopaque material are highly predictive of and specific for a serum iron level $\geq$ 300 mcg/dl. Vomiting is considered to have the highest sensitivity and predictive value, so it is unlikely that after an asymptomatic period of more than 6 hours after the ingestion that serious toxicity will occur.

   a. < 300 mcg/dl --> no specific treatment

   b. 300-500 mcg/dl --> chelation therapy

   c. 500-1000 mcg/dl --> chelation therapy and aggressive support

   d. > 1000 mcg/dl --> vigorous support and prolonged chelation therapy, significant increase in mortality

5. Deferoxamine Challenge

   a. Chelates with Fe to form soluble/excretable complex

   b. In the past this challenge was performed to determine the need for chelation therapy. After the deferoxamine injection one would observe the urine for a “vin rose wine” color as an indication for continuation of therapy. **This is NOT now recommended** as some patients will have a toxic ingestion without production of the urine color changes.

B. Specific Therapy

1. Observation alone with ingestions < 20 mg/kg and a negative KUB, Not all preps radiopaque, however.

2. If asymptomatic for 6 hours following the ingestion they do not require admission.

3. Ipecac - is not contraindicated in the awake, alert child but may mask the vomiting produced by the iron ingestion resulting in an underestimation of the toxicity

4. Gastric lavage

   a. Abd x-ray will help determine if lavage successful (again, not all preps radiopaque)

   b. Use large bore OG, 1/2NS (not Fleet's) for lavage

   c. Sodium bicarb. or deferoxamine are **NO LONGER** recommended as part of the lavage.

   d. Examine for Fe tablets

5. Activated charcoal is not indicated as it does not prevent iron absorption and is not used UNLESS a coingestion exists.

6. Chelation therapy **(Deferoxamine)**

a. Indications

(1). All symptomatic patients exhibiting more than transient minor symptoms

(2). Patients with lethargy, coma, seizures, significant abdominal pain, hypovolemia, or acidosis

(3). Patients with a positive radiograph showing multiple radiopacities (retained pill fragments will lead to further absorption and toxicity)

(4). Patients with serum iron levels $\geq$ 300 mcg/dl

b. Route

(1). Given IV, IM or SQ, **IV preferable, and is most appropriate route.**

(2). IM contraindicated if patient is hypotensive, as won't be absorbed well. IM route is painful, especially as repeated injections required. It has erratic absorption, obtains higher peak deferoxamine levels and shows a higher incidence of side effects.

c. Dose

(1). 15 mg/kg/hour IV, no maximum dose although package insert states 6 grams/day

d. Side Effects (rare if given at above rate)

(1). Hypotension / shock seen at rates > 45 mg/kg/hr.

(2). **NOT to be given with compazine as coma can result!**

e. Cautions

(1). The iron-deferoxamine complex is excreted in the urine so the patient must be making adequate urine for this to work

(2). If oliguric/anuric then use chelation and dialysis, (hemodialysis, continuous arteriovenous hemofiltration, or peritoneal dialysis)

f. Most patients require no more than 24 hours of therapy

g. Treatment is continued until all the following criteria are satisfied:

(1). Patient is free of s/s of iron toxicity

(2). Serum iron level is normal or low (< 100 mcg/dl)

(3). KUB shows no radiopacities

(4). If patient developed “vin rose” colored urine, the urine color has normalized

7. Supportive therapy

(1). ICU, CVP, A-line, etc. as indicated

(2). Adequate fluid resuscitation can not be over emphasized

8. Other considerations

a. Severe coagulopathies - draw **baseline** PT, PTT, fibrinogen levels, for lab evaluation see below

b. Hepatic failure

c. Intestinal infarction

- Due to retained pill fragment or hypotensive episode

9. Other therapies: whole bowel irrigation, gastrotomy, especially if iron containing aggregates occur in the stomach or intestines on X-ray.

C. Lab Evaluation

1. Serial serum Fe’s and TIBC’s

a. Run Fe levels **STAT** with first level drawn 2-4 hrs after ingestion if possible

b. Follow until within safe range

c. Some labs are unable to measure serum Fe accurately in the presence of deferoxamine

2. Electrolytes, BUN, creat, glucose. Follow serum glucoses carefully as sudden hypoglycemia may develop after a period of hyperglycemia.

3. CBC with diff (leukocytosis, anemia)

4. ABG to assess respiratory status and metabolic acidosis
5. Type and Cross
6. Clotting Studies, (PT, PTT, fibrinogen)
7. LFT's,- baseline and serially
8. Assess urine output
9. Guaiac stools and emesis

LEAD POISONING

I. Introduction

A. Lead is absorbed through the GI tract (ingestion) and the lungs (inhalation).Increased intake results in deposition in the soft tissues, especially kidney, liver and brain.

B. Poisoning is usually from prolonged, excessive exposure; 1/2 life is approximately 20 years.

C. Sources

1. Lead pigmented paints (greatest hazard)

2. Contamination of food and beverages by lead glazed ceramic pitchers and lead soldered cans

3. Ingestion and retention in the stomach of fishing weights, shot, jewelry painted with lead paint, pencil coatings

4. Sniffing fumes of leaded gasoline containers

5. Mexican and oriental folk medicine (azarcon, greta, paylooah)

II. History of High Risk Children

A. High risk environment

1. Dilapidated housing

2. Exposures as above

B. Pica

C. Sibs with lead intoxication

III. Lab / X-ray

A. Venous lead level - fingerstick levels are inaccurate

B. CBC with peripheral smear (microcytic anemia, basophilic stippling)

C. UA (glycosuria, proteinuria), BUN, creat to evaluate for renal damage

D. KUB (flecks of lead in GI tract)

E. X-rays of long bones (lead lines in children age 2-5 yrs) - lines of increased density in metaphyseal plates represent growth arrest

F. FEP, (free erythrocyte protoporphyrin) - until recently was used to screen children for lead poisoning; it has a poor sensitivity for Pb

levels < 25 micrograms/dl and is not specific as it is increased in iron deficiency anemia, levels > 190 almost exclusively lead poisoning

IV. Clinical Picture and Management

A. Acute Lead Encephalopathy

1. Onset may be sudden

2. Coma, seizures, bizarre behavior, ataxia

3. Any of these sx. with an elevated blood lead level constitutes a medical emergency

4. Almost always assoc. with levels > 100 although reported at lower levels

5. Diagnosis can usually be made without LP which is dangerous because of inc ICP, which may be present in the absence of any of the usual sx. If LP necessary for differential, consider pre LP CT scan!

6. Treatment

a. Supportive

- NPO until significant improvement

- Fluid restrict to maintenance plus losses

b. Chelation (see below)

c. Seizures

- Treat initially with lorazepam 0.1 mg/kg IV q 2 min. to total initial dose of 1 mg/kg.

- If resistant use phenobarbital 15 - 20 mg/kg IV.

- Further therapy with either valium 0.1mg/kg IV, or phenytoin 10 mg/kg slow IV.

7. Cerebral Edema: Mannitol 0.25 - 0.5 grams/kg slow IVP. Steroids may be helpful dexamethasone 0.1 - 0.2 mg/kg q 4 hours IV

8. Renal and hepatic function, lytes, blood lead monitored daily

B. Symptomatic without encephalopathy

1. Lethargy, anorexia, vomiting, abdominal pain, constipation

2. Usually assoc. with lead levels > 70 (if level < 50, consider

other cause)

3. All symptomatic children potentially have lead encephalopathy, therefore treat immediately.

a. Supportive as above

b. Chelation (see below)

C. Asymptomatic with increased lead level

1. Although asymptomatic, have metabolic effects and subclinical neurobehavioral effects

2. Essential to have diagnosis based on lead level

3. If blood lead level 25-44 micrograms/dl, perform the CaNa2-EDTA provocation test; this is expensive and usually done only at centers where large numbers of lead poisoned children are treated. To perform:

a. Obtain a baseline lead level

b. Patient empties bladder

c. CaNa2-EDTA 500 mg/M^2 IV in 250 cc/M^2 D5W over one hour (see DKA chapter or Acute Tumor Lysis chapters for nomogram and calculation of body surface area)

d. Collect all urine over 8 hrs in lead free equipment

e. Results: $\frac{\text{urine lead excreted (mcg/cc) X total volume (cc)}}{\text{CaNa2-EDTA given (mg)}}$

Test is positive if > 0.6

V. Prevention

A. Do not return children to lead environment (consult social services for assistance in obtaining safe housing)

B. Screen all family members

C. Report all cases to public health for investigation and abatement of lead hazards

D. Extended follow up to at least age 6 yrs

VI. Chelation Therapy for Lead Poisoning

A. Symptomatic

1. Acute Encephalopathy, (usually for Pb > 100 micrograms/dl)

a. BAL (dimercaprol) 450 mg/M^2/day and CaNa2-EDTA 1500 mg/M^2/day

1. Start with BAL 75 mg/M^2 IM q 4 hrs

2. After 4 hrs, start continuous infusion of EDTA 1500 mg/M^2/day

3. Continue BAL and EDTA for 5 days

4. Interrupt therapy for 2 days

5. Treat for 5 additional days with EDTA and BAL, if Pb level remains high (> 70 micrograms/dl)

6. Measure rebound venous Pb level 7 - 10 days after therapy.

a. Other cycles will be needed if Pb rebounds > 50 micrograms/dl.

2. If symptomatic at **any** level without acute encephalopathy **OR** Pb level > 70 micrograms/dl

a. BAL 300 mg/M^2/day and CaNa2-EDTA 1000 mg/M^2/day

1. Start with BAL 50 mg/M^2/day IM Q 4 hrs

2. After 4 hrs, start continuous infusion of EDTA 1000 mg/M^2/day

3. Continue EDTA for 5 days

4. BAL may be DC'd after 3 days if Pb level < 50 micrograms/dl

5. Interrupt therapy for 2 days

6. Treat for 5 additional days with EDTA and BAL if Pb level remains high (> 70 micrograms/dl)

7. Other cycles may be needed depending on Pb rebound for levels > 50 micrograms/dl as above.

3. Asymptomatic - Before treatment measure blood Pb level:

a. Level of 45 - 69 micrograms/dl:

1. CaNa2-EDTA 1000 mg/M^2 /day by continuous infusion for 5 days

2. FDA recently approved (1991) DMSA (succimer) for asymptomatic children with Pb levels of 45 - 69 micrograms/dl. Dose is 350 mg/M^2 (or 10 mg/kg) q 8 hours X 5 days PO, then 350 mg/M^2 (or 10 mg/kg) q 12 hours X 14 days PO. Its' use has been limited.

b. Level of 25 - 44 micrograms/dl: CaNa2-EDTA provocation test as above then:

1. If ratio > 0.6 : CaNa2-EDTA 1000 mg/M^2/day IV for 5 days

RAPID CARDIO-PULMONARY ASSESSMENT

I. Introduction:

A: Selected Conditions Requiring a Rapid Cardiopulmonary Assessment (partially from PALS, Chapter 1.)

1. The purpose of this assessment is to determine in < 30 seconds which patient has cardiac or pulmonary failure that may lead to an arrest. **Any of the following selected conditions require a rapid cardio-pulmonary assessment:**

Respiratory rate > 60
Heart rate > 180 or < 80 (under 5 years)
> 160 or < 60 (over 5 years)
Respiratory Distress - increased work of breathing (retractions, nasal flaring, grunting)
Trauma
Burns totaling > 10 % of surface area
Cyanosis
Failure to recognize parents
Diminished level of consciousness - unusual irritability, or lethargy
Seizures
Fever with petechiae
Admission to an ICU

II. Airway Patency - is the airway patent, maintainable or non-maintainable?

III. Breathing

A. Respiratory Rate (<u>also see Status Asthmaticus chapter</u>)

Normal Newborn	Normal 1 year	Normal 18 years	
< 40 - 60	24	18	> 60 <u>always</u> abnormal

- a slow or irregular RR in an acutely ill child or infant is ***<u>OMINOUS</u>*** ! This may indicate the patient is deteriorating rather than improving secondary to hypothermia, fatigue or CNS depression.

B. Air Entry

1. Chest rise?
2. Breath sounds bilaterally?
3. Stridor? - indicates upper (extrathoracic) airway obstruction such as the tongue, laryngomalacia, vocal cord

paralysis, hemangioma, tumor, cysts, infection, edema, or aspiration of a foreign body

4. Wheezing? - indicates intrathoracic obstruction such as bronchiolitis, asthma, pulmonary edema, or an intrathoracic foreign body

C. Mechanics

1. Retractions - intercostal, subcostal, suprasternal
2. Grunting - to preserve functional residual capacity (FRC)
3. Flaring
4. Gasping - often a sign that bag-valve mask is needed esp. in neonates.
5. Head bobbing - indicates increased respiratory effort

D. Baseline Color - if patient is cyanotic does color improve with O_2, bagging, intubation? Anemic patients may not exhibit cyanosis even though they are hypoxemic.

IV. Circulation

A. Heart Rate (also see Cardiac Dysrhythmia chapter)

	Newborn - 3 mos.	3 mos. - 2 yr.	2 - 10 yr.	> 10 yr.
Normal:	140	130	80	75

Abnormal: < 5 yr.: > 180 or < 80, > 220 consider SVT
> 5 yr.: > 160

B. Blood Pressure (BP)- remember hypotension is a LATE and often sudden sign of cardiovascular decompensation. (also see tables in Hypertension chapter)

Normal	Newborn	1 yr.	> 1 yr.
Systolic	> 60	>70	> 70 + (2 x yr.) - lower limit (5th percentile)of normal 90 + (2 x yr.) - 50th percentile

Blood volume: Neonate 85 cc/kg, infants 80 cc/kg, children 75 cc/kg

C. Peripheral/Central Pulses

1. Present/absent
2. Volume/ strength
3. Pulse pressure (systolic - diastolic BP), as Cardiac Output decreases the PP narrows and the pulse becomes thready, as Cardiac Output increases such as in septic or anaphylactic shock the PP

widens and pulses are bounding. A wide PP does ***not*** mean that perfusion is adequate !

D. Skin Perfusion

1. Capillary refill time: Normal is < 2 sec.
2. Temperature
3. Color - cyanosis, pallor?
4. Mottling

E. CNS Perfusion

1. Recognition of parents
2. Quick assessment of responsiveness
   - Awake
   - Responds to voice
   - Responds to pain
   - Unresponsive
3. Muscle tone
4. Pupil size
5. Posturing

F. Shock - ***definition***: The failure of the cardiovascular system to adequately perfuse vital organs. Shock is a clinical state characterized by inadequate delivery of oxygen and metabolic substrates to meet the metabolic demands of tissues. It produces signs of inadequate organ and tissue perfusion and function such as oliguria and lactic acidosis. Shock may occur with a normal, increased, or decreased Cardiac Output and a normal, increased, or decreased BP. In **compensated** shock the BP is normal. In **uncompensated** shock we see hypotension and often a low Cardiac Output. Note: This definition fails to stress blood pressure!

1. Five basic types:
   1. Hypovolemic - usually low Cardiac Output (C.O.)
   2. Cardiogenic - usually low C.O.
   3. Septic - usually high C.O. secondary to low systemic vascular resistance, although tissue perfusion is still inadequate
   4. Neurogenic - usually low C.O.
   5. Anaphylactic - usually high C.O. secondary to low systemic vascular resistance, although tissue perfusion is still inadequate

2. Patients must be reevaluated after **every** intervention !!! For example; if a fluid bolus has been given then assess the child for any improvement as indicated by:
   1. Improved cap. refill
   2. Stonger pulses
   3. Improved urine output
   4. Lower HR
   5. Improved mental status

When giving fluid beware of cardiac decompensation or signs of cardiogenic shock such as distended neck veins, enlarged liver, rales, or cardiomegaly on CXR. If these signs are not evident then consider further fluid boluses as needed but **reassess after each and every intervention !!**

G. If the patient is intubated and experiences problems the endotracheal tube may be the reason! Check:

1. Is the ETT in the airway? Listen for breath sounds bilaterally, and over the stomach. Is the chest rising? Is the color improving? Is the HR improving? If in doubt look!

2. Is the ETT plugged? Suction the tube. Remember, however, that a ETT plugged with sticky mucous may not clear with suctioning.

3. Is the ETT in the right main stem bronchus? Listen for breath sounds bilaterally! If no breath sounds on the left consider right main stem intubation. Although rare, left main stem intubations occur as well.

4. Does the patient have a pneumothorax? - If unstable needle the chest. If stable get a CXR, prepare for a chest tube.

5. Does the patient need more pressure or volume? eg. decreased compliance with drowning victims, RDS etc. or too small a ETT.

6. Is the equipment malfunctioning? Check the BVM, ventilator, gas source, ETT - is it kinked?

RAPID SEQUENCE INDUCTION

I. Rapid sequence induction (RSI) for intubation implies the use of sedation and nueromuscular blokade, avoidance of positive pressure BVM ventilation prior to intubation, and use of cricoid pressure. These measures are an attempt to decrease the cardovascular and CNS responses to intubation and decrease the risk of aspiration.

II. Use of paralysis should may be contraindicated if intubation is anticipated to be difficult due to anatomic abnormality or pathology. If airway problems anticipated, Anesthesia should be consulted.

III. RSI is performed in any patient with a full stomach or possible increased intracranial pressure.

V. Rapid sequece induction:

A. Preparation, preoxygenation, premedication, sedation, cricoid pressure, paralysis, intubation, verify.

B. Preoxygenation involves 2-5 minutes of 100% O2 via nonrebreather in spontaneously breathing patients or 1-2 minutes in apneic patients via BVM.

C. Standard Medication Regimen

1. Atropine 0.02 mg/kg with a minimum of 0.1 mg IV,if less than 2 years of age

2. Fentanyl 1-2 mcg/kg and Versed (midazolam) 0.1- 0.2 mg/kg IV

3. Paralysis if Able to Bag Mask Ventilate and No Anatomic Abnormalities or other contraindictions to Paralysis:

a. Vecuronium 0.2-0.3 mg/kg IV or Rocuronium 1 mg/kg:

b. Succinylcholine 1 - 2 mg/kg IV. Succ. has many side effects (see below) and probably is not as safe in inexperienced hands as vecuronium. Infants tend to need a larger dose of succinylcholine (2-3 mg/kg).

c. Preoxygenation is continued until the pt. is fully relaxed.

d. **In children older than 4 years of age,** if succinylcholine is used, a defasciculating dose of a nondepolarizing muscle relaxant (vecuronium 0.01 mg/kg) may precede the depolarizing agent to prevent muscle fasciculations and elevation of intragastric pressure, which may be associated with regurgitation.

e. If continued paralysis is required after patient is intubated, a non-depolarizing NMB (vecuronium) is used.

VI. There are patients in which sedation and paralysis is contraindicated (unstable airway - epiglottitis, mediastinal mass, anatomic abnormalities making it difficult or impossible to use the usual methods of intubation).

VII. Each patient should be evaluated on a case by case basis, especially since there may be contraindications to using certain medications for intubation. IN GENERAL:

A. **For sepsis/ shock/ asthma:**

1. Consider using ketamine for sedation rather than versed (maintains BP, bronchodilator). Dose is 2.0 mg/kg IV. Ketamine increases secretions so atropine 0.02 mg/kg (min 0.1 mg) IV or Robinul 5 mcg/kg is given as well. Ketamine can also cause nightmares/emergence reactions. Versed 0.1 mg/kg given with Ketamine should prevent this.

2. May consider Fentanyl 1-2 mcg/kg for sedation rather than Ketamine if shock is severe.

3. Paralyze fast with Vecuronium 0.3 mg/kg IV, **or** Rocuronium 1 mg/kg.

B. **For head trauma or other diseases with increased ICP:**

1. Use lidocaine (1.0 mg/kg IV) 1 minute prior to intubation to buffer the ICP spike in response to laryngeal stimulation.

2. Etomidate 0.3 mg/kg is the drug of choice as it is cerebral protective and has minimal hemodynamic effects. Thiopental (2-4 mg/kg IV) is an alternative, but may result in hypotension.

3. Use atropine for children younger than 2 years of age 0.02 mg/kg.

4. Use vecuronium (0.3 mg/kg IV) or Rocuronium 1 mg/kg.

5. Ketamine causes a rise in ICP - should **not** be use for sedation in this situation.

VIII. Unwanted Effects of Succinylcholine

A. Cardiovascular - bradycardia, hypertension or hypotension. Atropine prior to administration may prevent bradycardia.

B. Hyperkalemia – life threatening hyperkalemia is most likely to occur following: burns (24-48 hours post burn), spinal cord injury, tetanus, severe intraabdominal infections, encephalopathy, polyneuropathy including Guillain- Barre syndrome, and chronically ill children.

C. Increased intraocular pressure- avoid in globe injury.

D. Hypersensitivity reactions - anaphylactic reactions

E. Muscle pains - ameliorated with pancuronium

F. Rhabdomyolysis and myoglobinuria

G. Malignant hyperthermia

H. Pulmonary edema and pulmonary hemorrhage; several infants in whom pulmonary edema (increased SVR, decreased PVR, leaky capillaries) formed only minutes after 4 mg/kg of IM succinylcholine were given are reported.

I. Increased ICP - can be attenuated with prior administration of nondepolarizing agent and thiopental or lidocaine. Increased cerebral blood flow.

J. Increased gastric pressure and increased risk of regurgitation.

K. Not effective in patients with Myasthenia Gravis.

IX. Why use drugs.

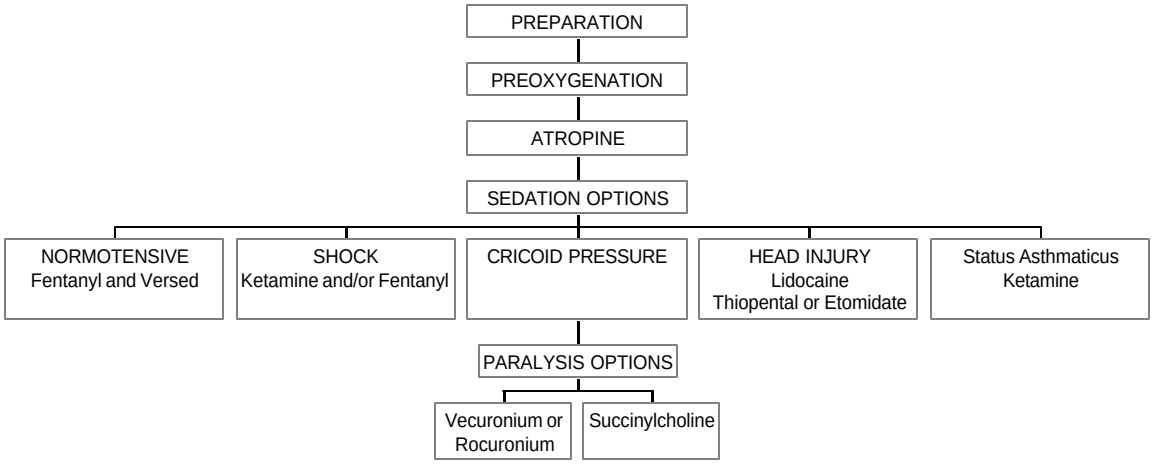
A. AVOID STRUGGLING & TRAUMA

B. AVOID HYPOXEMIA

C. AVOID UNDESIRABLE PHYSIOLOGICAL RESPONSES -Hypertension, tachycardia, increased ICP, etc

D. PAIN CONTROL & AMNESIA

E. TO FACILITATE INTUBATION



SICKLE CELL ANEMIA

I. DEFINITION AND PATHOPHYSIOLOGY - sickle cell anemia comprises several distinct disorders caused by abnormal hemoglobin (Hgb) which results in polymerization and formation of long hemoglobin chains. These chains distort the shape of red cells causing changes in blood viscosity and in the red cell membrane.

A. The mutation in sickle cell disease occurs when the sixth amino acid in the beta chain (usually glutamic acid) is replaced by valine. This causes the hemoglobin to form polymers when deoxygenated.

B. Pathophysiologic mechanisms:

1. Oxygen saturation: deoxygenated hemoglobin S polymerizes to form long rods (tactoids) that distort the shape of the red cell. Sickling is reversed by oxygenation but repeated sickling and unsickling damages the cell membrane.

2. Hemoglobin concentration: the polymerization rate changes in proportion to the thirtieth power of the hemoglobin concentration.

3. Temperature: polymerization of sickle hemoglobin is potentiated by fever or hypothermia.

4. Blood pH: acidosis increases polymerization. Reperfusion of the tissue results in lactic acidosis. Renal tubular defects seen in patients with sickle cell disease reduce the capacity of the kidneys to excrete acid. Oversedation may result in respiratory acidosis.

II. GENERAL MANAGEMENT

A. IDENTIFICATION

1. Hemoglobinopathy screening of newborn infants.

a. Nearly 100% Hgb S, some Hgb F, no Hgb A:
i. Sickle cell anemia — homozygous (Hgb SS)
ii. Sickle β^{o}-thalassemia

b. Hgb A, Hgb S, ± Hgb F:
i. Sickle trait - heterozygous
ii. Sickle β^{+}-thalassemia

c. Hgb S, Hgb C: Sickle hemoglobin C disease

2. Consultation with a pediatric hematologist is advised.

B. ROUTINE AND PREVENTIVE HEALTH CARE MEASURES

1. Treat as normal children. Provide all routine immunizations, good nutrition, and other preventive health care measures.

2. Must have a primary health care provider with whom there is a mechanism for prompt emergency care when the need arises.

3. Infection prevention:

a. By 3 to 4 months of age (when the fetal hemoglobin declines to below 50% of the total), clinically significant hemolytic anemia and impairment of splenic function develops.

b. Therefore, children with sickle cell anemia are at risk of overwhelming septicemia, often without a primary focus, due to the encapsulated organisms *Streptococcus pneumoniae* and *H. influenzae* type b. Without preventive measures, 15-20% of infants and young children with sickle cell anemia die before 5 years of age.

c. Penicillin. Either tablets or suspension can be utilized. Since the suspension necessitates a prescription refill every 2 weeks, tablets, which can be crushed and given in a spoonful of formula or food, are preferred.

i. Prior to 2 years of age: Pen-VK 125 mg BID po.
ii. After age 2: Pen-VK 250 mg BID
iii. Continue until 5 years of age.

d. Vaccinations.

i. Conjugated *H. influenzae* type b vaccine early in infancy.
ii. The 23-valent pneumococcal vaccine at 24 months of age. Earlier immunization is ineffective. A "booster" dose of pneumococcal vaccine should be given 3 years after the first dose.

III. FEVER

A. ETIOLOGY

1. Fever is usually the initial manifestation of sepsis. Prompt antibiotic therapy can be life-saving.

2. Septicemia due to *Streptococcus pneumoniae* or *Hemophilus influenzae* type b is the most common cause of death in young children with sickle cell anemia

B. EVALUATION

1. **Whenever a child with sickle cell anemia or sickle b^0-thalassemia has a temperature > 101.5 °F, he or she should be evaluated <u>at once</u>.**

2. Rapid but complete history and physical examination.

3. Immediately place an intravenous catheter or needle. Obtain a CBC (including differential, WBC count and reticulocyte count) and blood culture.

4. Immediately administer intravenous (or intramuscular if the intravenous route is impractical) ceftriaxone (Rocephin) 50-75 mg/ kg, maximum 2 grams. If ceftriaxone is unavailable, give cefuroxime (Zinacef, Kefurox) 50 mg/kg. Ampicillin 75 mg/kg or another antibiotic effective against S. pneumonias and H. influenzae type b may also be given.

5. Parenteral antibiotics should be given even if there is an obvious focus of infection (otitis, URI, etc.).

6. A chest X-ray should be considered when the child has a fever, cough, tachypnea, chest pain or other physical findings suggesting a pneumonia or other pulmonary process.

7. Other laboratory tests may be indicated (e.g., type and cross match for possible red blood cell transfusion, CSF analysis and culture, urine culture, etc.) depending upon the clinical findings.

8. Prompt and careful physical assessment and administration of IV antibiotics should have high priority. Do **not** wait until after the chest X-ray or blood count results return.

C. CRITERIA FOR HOSPITALIZATION

1. IF THE CHILD IS **"TOXIC"** OR IF THEY HAVE AN **ALTERED MENTAL STATE**, HE OR SHE SHOULD BE PROMPTLY ADMITTED FOR OBSERVATION AND ADDITIONAL PARENTERAL ANTIBIOTIC THERAPY. **IF IN DOUBT,** ADMIT THE PATIENT TO THE HOSPITAL.

2. ALL PATIENTS RECEIVING **CEFUROXIME** OR **AMPICILLIN** RATHER THAN CEFTRIAXONE SHOULD BE ADMITTED IN VIEW OF THE SHORT PLASMA HALF-LIFE OF THESE AGENTS.

3. Temperature greater than or equal to 103°F.

4. Recent doses of prophylactic penicillin missed.

5. Child under 12 months of age.

6. Lobar infiltrate on chest X-ray.

7. Respiratory distress.

8. WBC count > 30,000 per mm^3 or shifted to the left and/or other hematologic parameters greatly altered from baseline values (e.g., hemoglobin < 6 gm/dl, platelets < 150,000 per mm^3).

9. Admit if follow-up (telephone contact, return visit, etc.) is uncertain or unlikely because of distance, inconvenience, or poor compliance.

D. INPATIENT MANAGEMENT

1. Administer intravenous Cefuroxime (preferably) or Ampicillin 150 mg/kg/day in 3 or 4 divided doses x 48 hr until cultures are sterile and clinical status improves.

2. Observe closely for deterioration in clinical status that may indicate septicemia or development of acute chest syndrome.

3. Mycoplama coverage if pt. fails to improve with initial antibiotics.

E. OUTPATIENT MANAGEMENT

1. If the evaluation suggests that outpatient management is possible, a short period of observation is advised, followed by reevaluation prior to discharging the patient. Reevaluation should include assessment of vital signs, level of consciousness, and ability to take oral fluids and medications.

2. Blood cx, CXR if indicated, Rocephin 50 mg/kg X1 dose.

3. **Follow-up by telephone or a repeat outpatient visit within 24 hours is necessary in all cases.**

4. **Blood culture results must be checked daily and patient recalled at once if positive.**

IV. VASO-OCCLUSIVE EPISODE/PAIN CRISIS (see drug doses below)

A. DEFINITION/ ETIOLOGY

1. Vaso-occlusive crisis is the most common complication of sickle cell disease. It is characterized by episodic attacks of ischemic pain and infarcts of various organs.

2. Precipitating factors include infection, fatigue, fever, dehydration, or exposure to cold.

B. MILD PAIN

1. The Child with MILD pain may not appear to be uncomfortable but complains of pain.

2. Treatment:

a. Acetaminophen or ibuprofen every 4 hours.

b. Hydration with maintenance fluids.

c. Send home with prescription for at least 20 doses of acetaminophen with codeine (1 mg/kg/codeine/dose) for more severe pain. Ibuprofen may be alternated with the acetaminophen or acetaminophen with codeine; so that some medication is given every two hours.

C. MODERATE PAIN

1. The child with a MODERATE episode evidences discomfort by facial grimacing, unhappiness, irritability, a poor appetite, and has not responded to home treatment.

2. If the child has not received codeine, give an appropriate dose of acetaminophen with codeine (1 mg/kg/codeine/dose).

3. If pain does not improve, give IV fluids at maintenance and a bolus injection of morphine (0.1 - 0.15 mg/kg to a maximum of 8 mg). Fluid bolus as appropriate if evidence of dehydration.

4. If the child has received codeine in an appropriate dose at home, treat as severe crisis (see below).

D. SEVERE PAIN

1. The child with severe pain is extremely uncomfortable, may be agitated, crying, screaming and cannot be consoled.

2. If the patient has been taking codeine at home in appropriate doses then immediately start IV fluids at maintenance, give an IV bolus injection of morphine (0.1 - 0.15 mg/kg/dose to a maximum of 8 mg per dose) and observe for pain relief.

3. If the patient is comfortable for more than two hours after an IV morphine bolus, then give a dose of oral medication such as acetaminophen with codeine and observe the child for at least one more hour. Send home with a prescription for 20 doses of acetaminophen with codeine. Remind parent to call the hematology/oncology clinic the next day. Reassure family that pain is not dangerous and will resolve in a few days.

4. If pain returns 1 or 2 hours after an IV morphine bolus, repeat the dose (0.1 to 0.15 mg/kg/dose to a maximum of 8 mg per dose) and observe for another hour. If a child receives 2 IV boluses with continued discomfort, then admit to hospital. Decrease IV fluids to maintenance rate if admitted to hospital.

5. In a child with poor venous access consider SQ or IM injection of pain medication.

E. INPATIENT MANAGEMENT OF ACUTE PAIN EPISODE

1. Exclude a precipitating complication or other cause of pain.

2. Bed rest and oral or intravenous hydration.

3. Tailor the analgesic used, dosage, route and frequency of administration to the severity of pain and the patient's reported response. Use of a pain scale may be beneficial.

3. Always provide the pain medication on a fixed schedule of administration, **NOT PRN**, based on the duration of drug action. Periodic adjustments should be based on pain control and level of sedation.

4. Intermittent, intravenous administration is not recommended because of high acute blood levels, excessive euphoria, respiratory depression, and short duration of action. As pain improves, reduce the amount of medication per dose, not the frequency of administration. A **PCA** pump should be considered.

5. If treatment is required for more than 5 days, physical withdrawal may occur so the medication should be tapered over several days.

F. DRUG DOSES

1. Severe Pain:

a. Morphine: 0.1 to 0.15 mg/kg/dose; IV (preferred; repeat q 1-3 hours. Maximum 8 mg per dose. If using PCA: basal rate of 0.03 mg/kg/hour with PCA doses of 0.015 mg/kg Q 10 min with 4 hour lockout 0.3mg/kg is a reasonable regimen to start with.

b. Hydromorphone (Dilaudid): 0.02 mg/kg/dose (IM, IV) q 3-4 hr. or 0.04 mg/kg/dose po q 4 hr.

c. Oxycodone: 5-10 mg/dose po q 4 hr.

2. Moderate Pain:

a. Codeine: 1 mg/kg po q 4 hr.

3. Mild Pain:

a. Acetaminophen: 8 mg/kg/dose po q 4 hr.

b. Ibuprofen: 10 mg/kg po q 4 to 6 hr.

c. Naproxen: 250 mg/dose po q 12 hr.

d. Indomethacin: 25 mg/dose po q 4 to 8 hr. Contraindicated in psychiatric, neurologic and renal diseases.

e. Tolmetin: 400 mg po q 8 hr.

4. Toradol is a NSAID that can be given IV and may be beneficial in mild-moderate pain as well as an adjuct to narcotics in severve pain (it may decrease the dose of narcotics needed to control pain).

5 **CONTRAINDICATED**:

a. Aspirin: associated with Reyes syndrome.

V. ACUTE CHEST SYNDROME

A. DEFINITION/ETIOLOGY.

1. An acute respiratory illness characterized by cough, fever, pleuritic chest pain, respiratory distress, leukocytosis and pulmonary infiltrate on chest x-ray.

2. Etiology is uncertain, but in most cases it probably represents a combination of infection and intrapulmonary sickling (infarction).

3. Less common etiologies of chest pain include myocardial ischemia, chest wall pain from bone infarction, part of the diffuse pain of a pain crisis, esophageal disease, peptic ulcer disease, and gallbladder disease.

B. CRITERIA FOR HOSPITAL ADMISSION: Respiratory distress (tachypnea, dyspnea, retractions, flaring, prominent cough, etc.) and/or:

1. Moderate or severe chest pain.

2. Fever greater than 39°C or 102°F.

3. Oxygen saturation more than 3% below baseline or <90% in room air.

C. INPATIENT MANAGEMENT

1. Blood culture initially: repeat after 48-72 hours for persistent temperature over 38.5°C (101.2° F).

2. Intravenous fluids at maintenance rate; watch for volume overload.

3. Daily weight, strict I & 0s, frequent vital signs.

4. Supplemental oxygen by mask should may be given; the level of oxygenation should be continuously monitored by pulse oximetry, and maintained at or above baseline value (see data base) or if no baseline available > 90%.

5. Chest X-ray initially and then every 2 to 3 days, or as clinically indicated.

6. CBC with differential and reticulocyte count initially, then daily Hgb/HCT/Retic. The hemoglobin often falls by 2-3 gm/dl during severe acute chest syndrome.

7. Antibiotic coverage for *S. Pneumoniae*, *H. influenzae* type b and possible concomitant mycoplasma infection. Current recommendations: Cefuroxime, 150 mg/kg/day IV plus Erythromycin 40 mg/kg/day po both divided TID.

8. Cautious use of narcotics for chest wall pain due to the risk of hypoventilation, however pain may lead to splinting and hypoventilation as well. Tordol may be a good option for control of mild-moderated pain.

9. Chest physiotherepy to involved area of lung when consolidation, atelectasis,or abundant secretions are present; bronchodilators by inhalation may be of use as well.

D. MANAGEMENT OF SPECIFIC COMPLICATIONS

1. Monitor arterial blood gases for increasing respiratory distress and falling O_2 saturation. Cyanosis may not be obvious in such patients, and if pO_2 is less than 60 mm Hg, generalized sickling may occur.

2. Give packed red blood cell transfusion of 7-10 ml/kg each for declining hemoglobin and worsening clinical condition (to increase oxygen carrying capacity). Post transfusion hemoglobin should not exceed 11 gm/dl. Do not routinely transfuse stable patients as there is no evidence that correction of the anemia hastens recovery. Adjust IV rate during transfusion to avoid volume overload.

3. Transfer to ICU for a 1 to 2 blood volume exchange transfusion (see below) using whole blood (to remove sickled cells} in patients with a pO_2 less than 60 mm Hg despite maximal oxygen therapy and/or clinical deterioration. If the patient is over 20 to 25 kg consider use of automated pheresis with packed red blood cells (see below).

4. Intubation and mechanical ventilation as indicated for hypoxemic respiratory failure or repiratory failure due to fatigue.

VI. APLASTIC CRISIS

A. DEFINITION AND PATHOPHYSIOLOGY

1. A rapid decline in hemoglobin concentration resulting from transient cessation of erythropoiesis.

2. Etiology: direct cytotoxic effect of parvovirus B19 on erythroid precursors in the bone marrow. Erythroid precursors disappear from the bone marrow for about 10 days following parvovirus infection, resulting in a reticulocyte count usually less than 0.1% and a reduction in hemoglobin values, often by 3-5 gm/dl.

3. Patients with aplastic crisis do not usually exhibit the other manifestations of parvovirus B19 infection (such as the "slapped cheeks rash of erythema infectiosum or arthralgia/arthritis).

4. Approximately one week following the onset of erythroid aplasia, patients develop an antibody response (initially IgM and then IgG), which results in viral neutralization, resumption of marrow erythroid activity, and a rapid rise in the reticulocyte count and hemoglobin that is often heralded by a large number of of nucleated red blood cells on the peripheral blood smear.

B. CLINICAL MANIFESTATIONS/ LABORATORY FEATURES:

1. Occurs between 2 and 15 years of age.

2. Patients usually present with fever, malaise, lethargy, and possibly syncope.

3. Physical examination shows pallor and tachycardia. Patients with severe anemia may exhibit congestive heart failure. The spleen is not larger than usual.

4. Laboratory abnormalities include severe anemia (hemoglobin usually 2 to 6 gm/dl) and reticulocyte count < 1.5 % (usually < 0.1 % unless the patient is already in the recovery phase). The WBC count is usually normal or slightly elevated. Platelet count is generally normal.

C. MANAGEMENT AND OUTCOME

1. Type and crossmatch for a total of 15-20 ml/kg of packed RBCs. For very young patients ask the blood bank to divide a unit of blood into aliquots.

2. Transfuse patient with 5-7 ml/kg of packed RBCs over 3 to 4 hours. After assuring that the patient is stable (i.e., not in heart failure), repeat the transfusion until a total of 15-20 ml/kg is administered. Patients with profound anemia who are in heart failure may require an exchange transfusion (see below). Do not over transfuse (Hb~11).

3. Patients with parvovirus should be placed in contact isolation since parvovirus Bl9 is highly contagious. They should have no contact with pregnant care-givers or ward personnel. Patients must wear a mask when out of their rooms.

4. Serum parvovirus antibody and antigen studies are usually not required.

5. Many patients with acute parvovirus infection have high fever and require blood cultures and intravenous antibiotics. Fever during an aplastic crisis is usually due to parvovirus Bl9 infection but bacteremia and the risk of sepsis cannot be initially excluded.

6. Following transfusion the patient may be promptly discharged from the hospital. Hemodynamically stable patients with less severe anemia (i.e., over 4.0 gm/dl) may be managed in the outpatient setting.

7. Hemoglobin levels should be followed every 2-3 days in the outpatient unit until reticulocytosis resumes and hemoglobin increases. When seen in clinic, patients should wear a mask to avoid the spread of the virus.

8. Siblings with sickle cell anemia (or other patients with sickle cell anemia with whom the patient has come in dose contact) should have a hemoglobin, hematocrit, and reticulocyte count immediately and again 10-14 days later to be sure that they, too, are not infected.

9. Recurrent aplastic events are rare since life-long humoral immunity against parvovirus Bl9 follows an aplastic crisis.

VII. ACUTE SPLENIC SEQUESTRATION CRISIS (ASSC)

A. DEFINITION AND PATHOPHYSIOLOGY:

1. Characterized by sudden enlargement of the spleen and decline in the hemoglobin concentration. Large quantities of sickled erythrocytes are pooled (sequestered) in the splenic red pulp.

2. A large percentage of the patient's blood volume accumulates in the spleen.

3. At one time, ASSC was one of the most common causes of death in infants with sickle cell anemia.

B. CLINICAL FEATURES:

1. Most common in infants and young children with sickle cell anemia between 6 months and 5 years of age.

2. Also seen in patients with preexisting chronic splenomegaly.

3. May affect teenagers and young adults with sickle C disease.

4. Usually no obvious triggering event is known.

5. Signs and symptoms are nonspecific, including lethargy or irritability, pallor, tachycardia, and sometimes pain in the left upper quadrant (especially in older patients). Occasionally, only an increase in spleen size is appreciated.

6. Patients with severe ASSC may present in frank cardiovascular collapse.

7. Physical examination:

a. Signs of anemia and/or hypovolemia.

b. The spleen is larger than the baseline, sometimes massively so.

C. LABORATORY FEATURES:

1. The hemoglobin is at least 2 gm/dl below the baseline steady state value. In some severe cases, the hemoglobin declines to life threatening levels.

2. Reticulocyte counts are elevated (usually 10 to 30%), and nucleated RBCs are almost always present on the blood smear.

3. The WBC count usually remains normal or slightly elevated.

4. The platelet count often declines to 50,000 to 150,000/mm3.

D. MANAGEMENT AND OUTCOME:

1. Identify that ASSC is occurring. Parents of most patients with sickle cell anemia have been taught to palpate the spleen and may present to the emergency department or clinic with the chief complaint of the spleen being larger than usual. Consulting the patient's database or hospital chart will confirm the patient's usual hemoglobin value and spleen size.

2. In mild cases of ASSC (i.e., spleen only slightly larger than usual, hemoglobin 2-3 gm/dl below baseline, patient hemodynamically stable), the child may be followed as an outpatient with daily physical examinations and blood counts. Consult peds heme-onc.

3. Patients with moderate or severe ASSC should be hospitalized and require the following:

   a. Careful and repeated physical assessments for spleen size and vital sign stability.

   b. Type and crossmatch for PRBCs.

   c. Serial hemoglobin determinations.

   d. If hemoglobin decline is substantial (i.e. to below 4.5-5.5 gm/dl),transfuse with 10 ml/kg packed RBCs (repeated as necessary) to raise the hemoglobin and maintain cardiovascular stability.

4. ASSC usually resolves within 2-5 days. Often, especially following blood transfusion, hemoglobin values rise to above steady state levels since the blood that had been pooled in the spleen is redistributed in the circulation. When the hospitalized patient shows stable or rising hemoglobin values and smaller spleen size, he or she should be discharged, with close outpatient follow-up.

5. Following an episode of ASSC, some patients have persistent splenomegaly and hypersplenism, with lower than usual hemoglobin and platelet values lasting weeks or months. All children who experience an episode of ASSC are at risk of repeat events.

6. Even though the spleen is enlarged, its reticuloendothelial function is defective. Therefore, children with ASSC are still as risk of overwhelming pneumococcal septicemia and require the usual preventive

measures, such as penicillin, pneumococcal vaccine, and prompt antibiotic therapy in event of fever.

7. Recurrent episodes of ASSC that require transfusion should be treated with splenectomy. Many patients, however, will not require splenectomy but exhibit gradual diminution in spleen size, with eventual autoinfarction.

VIII. TRANSFUSION THERAPY

A. INDICATIONS FOR TRANSFUSION

1. Definite indications

   a. Acute neurologic event.

   b. Splenic sequestration with substantial decline in hemoglobin (i.e. to below 4.5-5.5 gm/dl), or hemodynamic instability.

   c. Severe pneumonia or pulmonary infarction with declining hemoglobin or worsening clinical condition. Exchange transfusion indicated if pO_2 less than 60 mm Hg despite maximal oxygen therapy.

   d. Severe anemia with cardiac decompensation. Aplastic crisis with severe anemia.

   e. Acute arterial hypoxia (S_aO_2 < 90%).

   f. Hyperhemolytic crisis with enlarging liver/spleen.

   g. Ophthalmological surgery.

2. Relative indications:

   a. Symptomatic anemia.

   b. Hepatic sequestration.

   c. Leg ulcers refractory to conservative management.

   d. Priapism, recurrent, or resistant to acute treatment.

   e. Severe or prolonged pain episodes.

   f. Frequent pain episodes.

   g. Chronic respiratory insufficiency.

   h. High dose intravascular contrast studies.

   i. Surgery with general anesthesia.

   j. Pregnancy.

B. SIMPLE TRANSFUSION

1. Indicated for severe anemia, aplastic crisis, hyperhemolytic crisis, and in chronic transfusion programs. Also used in patients with a relative indication and a hematocrit < 20%.

2. **ALWAYS USE SICKLE-CELL (SICKLE DEX) NEGATIVE BLOOD.** All patients with a history of previous transfusions should be carefully screened for the presence of autoantibodies. Use washed or reconstituted frozen blood in patients with a history of allergic transfusion reactions.

3. Chronic transfusion:

   a. Give PRBCs to raise the hematocrit to 30%.

   b. Then transfuse q week until the % Hgb S is < 50%.

   c. After the % Hgb S is < 50%, the hematocrit can be raised to 35%.

   d. Transfuse every 3 to 4 weeks to maintain hematocrit > 30%, % Hgb S < 30% and the reticulocyte count < 4%.

4. Acute transfusion:

   a. Give PRBCs to raise the hematocrit to 28 - 33%.

   b. Further transfusions are administered based on symptoms.

5. Useful approximations:

   a. Total Blood Volume (TBV) = 70 cc X weight in kg.
      Total Blood Volume (in chronic anemia) = 75 cc X weight in kg.
      (see below for using the TBV in calculating the transfusion volume)
   b. Red Cell Volume of Patient = TBV X Hematocrit.

   c. To raise Hgb by 1 gm/dl: give 3 cc/kg of PRBCs.
      To raise Hematocrit by 10%: give 10 cc/kg of PRBCs.

   d. **Transfusion volume =** $\dfrac{\text{TBV X (HCT desired - HCT current)}}{\text{HCT of donor unit}}$

   e. Average values in transfused units
      i. Whole Blood: Hematocrit = 35%.
      ii. PRBCs: Hematocrit = 70%.
      iii. PRBCs with AdSol: Hematocrit = 60%.

C. EXCHANGE TRANSFUSION

(THIS DESCRIPTION ONLY APPLIES TO PATIENTS WITH SICKLE CELL ANEMIA – other algorhythms may be inidicated in neonates or in other scenarios where exchange transfusion is indicated)

1. Indicated in patients with acute neurologic events, severe pneumonia or pulmonary infarction, acute arterial hypoxia, ophthalmological surgery, high dose intravascular contrast studies, surgery with general anesthesia, and pregnancy.

2. Preparation

   a. Insert venous and arterial catheter OR two large-bore venous catheters OR a double-lumen hemodialysis catheter.

   b. Send blood to laboratory for:
      i. Complete blood count.
      ii. Quantitative sickle cell preparation (correlates well with the quantity of Hb SS noted at electrophoresis).
      iii. Electrolytes and calcium determination.
      iv. Cross-match with PRBC (**sickle negative**).

   c. Calculate and prepare volume to be transfused (see below).
      i. PRBCs are assumed to have a hematocrit of 60% in AdSol.
      ii. The whole blood equivalent is whole blood with a hematocrit of 40% or PRBCs reconstituted to a hematocrit of 40% with saline or FFP.

3. Procedure

   a. If the patient's hematocrit is ≤ 19% (Hgb < 6.5 g/dL). Give PRBCs equal to 30 cc/kg of body weight while removing an equal volume of the patient's blood. Then give donor whole blood equivalents cc for cc while removing an additional 40 cc/kg of patient blood.

   b. If the patient's hematocrit is between 20 and 30% (Hgb 6.6 to 10 g/dL). Give PRBCs equal to 10 cc/kg of body weight while removing an equal volume of patient blood. Then give donor whole blood equivalents cc for cc while removing an additional 70 cc/kg of patient blood.

   c. If the patient's hematocrit is > 30% (Hgb > 10 g/dL). Remove 10 cc/kg of patient blood and exchange with 10 cc/kg of normal saline. Then give donor whole blood equivalents cc for cc while removing an additional 80 cc/kg of patient blood.

   d. Rate of exchange transfusion and aliquots:

      i. Adjust the intravenous rate so the exchange transfusion occurs over 4 to 6 hours (increase the exchange transfusion time to 8 - 10 hours if over 1000 ml are to be exchanged).

      ii. Withdraw blood at 10 to 15 minute intervals from the arterial line or a large-bore venous catheter. The aliquot

for each draw will be determined by dividing the exchange transfusion volume by the total exchange transfusion time.

iii. If only a single catheter is available, exchange 2.5 cc/kg every 10 minutes. Attempts to establish a second line should continue after the exchange transfusion begins.

4. Monitoring

a. Heart rate and blood pressure (continuously).

b. Hematocrit and/or hemoglobin every 2 hours and at the last hour. Hemoglobin levels of greater than 12 g/dL (hematocrit > 36%) during the exchange may be associated with increased blood viscosity and complications.

c. Electrolytes, calcium every 2 hours.

5. Endpoint. This protocol should give a Hgb S level of about 30% and a final hemoglobin level between 10 and 12 g/dL.

STATUS ASTHMATICUS

I. Definition: Status asthmaticus (SA) is a life threatening form of asthma that is defined as a condition in which a progressively worsening attack is unresponsive to the usual appropriate therapy that leads to pulmonary insufficiency. The primary mechanical event in status asthmaticus is a progressive increase in airflow resistance. Mucous plugging and mucosal edema or inflammation are the major causes for the delayed recovery in status asthmaticus. The combination of hypoxia, hypercapnia, and acidosis may result in cardiovascular depression and cardiopulmonary arrest.

II. History:

A. Known asthmatic?

B. Asthma meds? Compliance? Time of last dose/nebulizer Tx?

C. Previous clinic/ED visits?

D. Previous hospitalizations, intubations, steroid courses?

E. When did current wheezing/resp distress begin?

F. Precipitating factors.

III. Physical Exam:

A. Vital Signs:

1. T: Fever may indicate URI, atelectasis or pneumonia

2. P: Usually elevated, especially if treated w/epi

3. R: Often tachypneic {see tables below for normal respiratory rates (RR)}

Respiratory Rates (breaths/min) of Normal Children Age 6 mos. to 8 years

	SLEEPING		AWAKE	
Age	**Mean**	**Range**	**Mean**	**Range**
6 - 12 mos.	27	22 - 31	64	58 - 75
1 - 2 yrs.	19	17 - 23	35	30 - 40
2 - 4 yrs.	19	16 - 25	31	23 - 42
4 - 6 yrs	18	14 - 23	26	19 - 36
6 - 8 yrs	17	13 - 23	23	15 - 30

Respiratory Rates (breaths/min) of Normal Children Age 8 - 18 years

	Mean	Range
8 - 10 yrs.	19.5	17 - 22
10 - 12 yrs.	19.5	17 - 22
12 - 14 yrs.	19	16 - 22
14 - 16 yrs.	18	15 - 21
16 - 18 yrs.	17	14 - 20

4. BP: Pulsus paradoxus (a decrease in systolic BP during inspiration of > 10 mmHg in children or > 15mmHg in adolescents) correlates well with moderate to severe disease. It can be measured with sphygnomanometer and a stethoscope: Inflate the cuff, deflate it slowly. At a certain pressure, you will hear pulse sounds during expiration, but not inspiration. As the cuff is deflated further, you will be able to hear sounds during inspiration and expiration. The difference in systolic BP between these two phenomina is the pulsus paradoxus. Pulsus paradoxus is readily apparent in patients with arterial lines where it is observed as a dampening of the arterial wave form during inspiration.

5. The use of accessory respiratory muscles correlates with the severity of airway obstruction (abdominal paradoxic breathing, sternocleidomastoid use, nasal flaring, intercostal retractions). Wheezing is the least sensitive indicator of obstruction. Crepitus indicates air leak in subcutaneous tissues.

B. **Asthma score:**

		0	1	2
1.	PaO2	70-100 in air	$\leq$ 70 in air	$\leq$ 70 in 40%
2.	Cyanosis	none	in air	in 40%
3.	Inspir. BS	none	unequal	Dec. to absent
4.	Access. mus. use	none	moderate	maximal
5.	Exp. wheeze	none	moderate	marked
6.	Cerebral function	normal	depressed or agitated	Coma

1. Clinical asthma score > 5 signifies impending respiratory failure, > 7 plus PCO2 > 65 signifies existing respiratory failure. **May substitute saturations for PaO2: > 95, 90-95, < 90.**

2. Be aware of the possibility of sudden deterioration in patients condition (mucous plugging, pneumothorax, worsening bronchoconstriction).

C. Must have air movement in order to wheeze, so lack of wheezing does **NOT** necessarily mean everything is fine!!

D. I:E ratio normally 5:2; may be 1:2 with severe attack

E. Symmetry of breath sounds:

1. Some asymmetry may be heard with asthma alone

2. Increased wheezing unilaterally may indicate foreign body

3. Decreased breath sounds unilaterally may indicate pneumonia or pneumothorax.

F. Mental Status: severe attack causes hypoxia and hypercarbia, causing confusion and decreased consciousness.

IV. Lab/X-ray

A. Pulmonary function tests: Obtain peak flow with Wright's Peak flow Meter on children old and well enough to cooperate.

B. CXR:

1. A CXR should be obtained on every child admitted to the hospital with SA to define the extent of the associated parenchymal disease; any complications, and to differentiate other disease entities.

a. The EKG may show acute RAD “p” pulmonale and a right ventricular strain pattern. During a severe attack hypoxemia and hyperinflation may lead to increased pulmonary vascular resistance. Also, negative pleural pressures become even more negative which, with lung hyperinflation, may lead to increased LV afterload.

2. Usually shows hyperinflation (diaphragm flat or everted)

3. May see pneumomediastinum, pneumothoraces, pneumoperitoneum, and/or subcutaneous emphysema

4. R/O pneumonia

5. Consider bilateral decub. films or insp./exp. films to R/O foreign body (unilateral hyperinflation, tracheal shift, radio-opaque foreign body).

C. ABG:

1. Usually not necessary in children who have responded partially to initial treatment and continue to improve.

2. An ABG should be obtained on patients:

a. With moderate - severe respiratory distress

b. Not responding to therapy

c. Serial ABG's may be necessary to evaluate progress/ deterioration

3. Because of air trapping, oxygenation is impaired and PaO2

decreases. Initially, hyperventilation leads to a decreased PaCO2. However, with further air trapping, work of breathing increases and lung compliance decreases leading to hypoventilation and increased PaCO2. Thus, a "normal" PaCO2 of 40 is abnormally high in the face of the increased respiratory rate and indicates a moderately severe attack. Similarly, the initial hyperventilation causes a respiratory alkalosis and increased pH. However, as hypoventilation ensues, a respiratory acidosis occurs as well as a metabolic acidosis from work of breathing and poor tissue oxygenation.

4. Blood Gases: Acidemia in excess of that predicted from measured PaCO2, accompanied by an abnormally large serum anion gap and high plasma lactate level, has been shown to occur in those severe asthmatic patients who require intubation. PaO2's less than 60 mmHg are an additional danger signal. Hypoxemia during severe asthma exacerbations occurs because of mismatching of ventilation and perfusion and can persist for days. If PEFR < 25%, you may see alveolar hypoventilation and a rise in PCO2.

5. Blood Gases:

		pO2	pCO2	pH	Severity
Example on RA pH/pCO2/pO2					
7.4/39/89	Stage I	↓	normal	normal	mild
7.45/35/78	Stage II	↓	↓	alkalotic	mild but worsening
7.4/38/68	Stage III	↓↓	normal	normal	moderate
7.3/48/55	Stage IV	↓↓	↑	acidotic	severe

V. Differential Diagnosis of Wheezing:

A. Wheezing in a known asthmatic is almost always an asthma attack.

B. First time wheezing in an infant may be bronchiolitis, or pneumonia (the last two may also occur in asthmatics).

C. Congenital Malformations

D. Laryngotracheomalacia, vocal cord paralysis, tracheal or bronchial stenosis, GER, vascular ring

E. Enlarged lymph nodes from infection or tumor.

F. Foreign bodies in trachea, bronchus, or esophagus.

G. Infections

H. Acute bronchiolitis

I. Bronchitis and asthmatic bronchitis

J. Vocal cord paralysis

K. Cystic Fibrosis

L. Aspergillus

M. Anaphylaxis (hx. of insect sting or drug?)

N. Toxic fume induced bronchospasm

VI. Treatments in Clinic or Emergency Department:

A. Acute:

1. Beta-agonist nebulizer treatment, may give 5-10 mg nebs back-to-back if minimal initial response.

a. Albuterol 0.15 mg/kg (max 10 mg/dose)

2. Atropine 250mcg (<2 y.o.), 500 mcg (>2 y.o.), add to 2nd or 3rd beta-agonist neb

3. Subcutaneous injection - subcutaneous epi. rarely done today as nebulizers are so commonplace. Terbutaline also may be used.

a. Epinephrine: 1:1000 0.01 cc/kg/dose (0.3 cc max)

b. Terbutaline 0.05% solution, 0.01 mg/kg/dose with a max. of 0.25 mg q 20 - 30 minutes.

4. Steroids: Solumedrol 2 mg/kg IV, then 1-2 mg/kg IV Q 6 hours. If mild exacerbation or quick response to above, may consider oral prednisone 2 mg/kg.

5. Oxygen

a. In younger patients, the distress caused by fighting the mask may only make the wheezing worse.

b. Humidified 02 should be placed on all patients who show evidence of hypoxia (O2 sat <90% on sat monitor/pulse oximeter) or respiratory distress. Remember the sat monitor gives no information regarding ventilation and pCO2.

6. A child who goes home after a clinic/ED visit needs additional therapy usually. If not on meds, begin albuterol orally or via inhaler. If on theo and subtherapeutic but yet claim good compliance, increase theo dose and add Albuterol. If therapeutic on theo, add another med (Albuterol - orally, or inhaler, or steroids). Arrange f/u to assess interventions.

VII. Treatments in the PICU

A child with a low paO2, high paCO2, decreased breath sounds, severe wheezing, retractions or altered mental status is in danger of respiratory failure and

should be considered for admission to the PICU. A-line monitoring should be considered and is indicated in most intubated patients. Patients requiring neb more frequently than Q1 hour for a prolonged period should probably be admitted to the PICU. Once in the PICU, patients must continue to be followed closely - (VS, mental status). Use continuous cardio-respiratory monitors. Beware of sudden deteriorations in the patient's condition.

A. Beta-agonists

1. Epinephrine - Has both B1 and B2 adrenergic agonist effects. Dose 1:1000, 0.01 ml/kg/dose SC, max 0.5 ml every 15-20 min. Generally not used over the selective B2 agonists.

2. Selective B2 agents

a. Albuterol

(1). If a patient is not optimally responding to standard doses of albuterol (0.15 mg/kg), one can increase the hourly dose and/or increase the frequency. Intermittent doses of 0.3 mg/kg (to max of 10 mg) every 20 min for 1-2 hours is acceptable. Tachycardia is generally mild with its greatest degree occurring during the first 60-80 minutes. Gradual improvement in the tachycardia despite continuation of therapy usually occurs.

(2). Continuous nebulized dose is 0.5-2.0 mg/kg/hour to a maximum of 20 mg/hr. **Administer the neb as close to the patient as possible if administering into the ventilator circuit.** Doses higher than 20 mg/hr may be needed if the drug is adhering into the ventilator tubing. Make sure that at least 6 liters of gas flow administer the nebulized dose.

(3). One will want to use a nebulizer with at least 8 liter/min of O2 flow if patient is not intubated. A Maxiheart nebulizer delivers 20 ml/hour of mist at 8 liters of flow. It delivers 30 and 50 ml/hour of mist at 10 and 15 liters of flow.The miniheart nebulizer is only used on the ventilator circuit. It uses 2 liters of flow to deliver 8 ml/hour of mist.

(4). Side effects - tremor, hypokalemia, headache, arrhythmias, anaerobic metabolism leading to met. acidosis with overdoses, chest pain.

b. Terbutaline

(1). Nebulized terbutaline has safely been administered to children with severe asthma (up to 0.4 mg/kg/dose) as well as by continuous nebulization (0.4 mg/kg/hr) with good results. Usual nebulizer dose: 0.03 mg/kg (up to 1 mg) in 1 cc NS.

(2). Terbutaline SC dose - 0.05% solution 0.01 mg/kg/dose (max 0.25 mg) every 20-30 min.

(3). May consider using continuous IV infusion if patient does not improve with inhaled beta-agonists. The recommended dose: 10 **mcg**/kg bolus over 15 minutes followed by 0.4 **mcg**/kg/min infusion. This can be increased by 0.2 **mcg**/kg/min at a time to a maximum of 6 **mcg**/kg/min. Tachycardia greater than 200 would warrant lowering the dose.

(4). There is a recommendation of reducing the maintenance dose of terbutaline by 50% in patients who are already receiving theophylline.

(5). Side effects: tremor, nervousness, headache, nausea.

B. Aminophylline

1. Studies of the emergency department management of asthma indicate that methylxanthines do not significantly enhance the bronchodilator response to B2 agonists when the latter are repetitively given at short intervals. However, when the dose and frequency of inhaled B2 agonists are reduced, methylxanthines and B2 agonists may maintain bronchodilation better than B2 agonists alone. **Metabolism varies - if you think you are subtherapeutic or toxic, check a level!**

Suggested mixing of IV Aminophylline: mix 1000 mg in 100 cc of **total** volume (10 mg/cc) or 500 mg in 500 cc of **total** volume (1 mg/cc) depending on the patients size and fluid requirements.

Suggested dosing of IV Aminophylline for continuous infusion:

0-1 month	0.15 mg/kg/hr
1-6 month	0.5 mg/kg/hr
6 month-1 year	1.0 mg/kg/hr
1-9 years	1.0-1.5 mg/kg/hr
10-16 years	0.8-1.2 mg/kg/hr
> 16 years	0.5-0.7 mg/kg/hr

2. The therapeutic level is 10-20 mg/liter and the desired level is 8-12 mg/liter. It is **not** necessary to keep levels as close as possible to the upper limit (20 mg/liter). Levels > 20 mg/liter are toxic. If the patient is not on theophylline one may begin the infusion by loading with 6 - 7 mg/kg over 20 minutes and then starting the appropriate infusion rate (see the above table). One then checks a post-loading dose at one hour after the bolus, and at 4 hours after the bolus. If the post bolus level is low one may reload with the appropriate amount **(1 mg/kg increases the level by 2 mg/liter)**. If the 4 hour level is lower then the post-bolus level, that suggests the infusion rate is too low and the patient should get rebolused, have the infusion increased by 10%, and have another 1 hour post-bolus level and 4 hour post-bolus level checked. If the 4

hour level is higher then the post-bolus level, that suggests the infusion rate is too high and the patient should have the infusion decreased by 10%, and another level should be checked in a few hours. A 12 - 16 hour level indicates steady state. If the patient is on theo. already, check a level and give an appropriate amount to reach a level of 12-14. So: the 1 hour post-bolus level indicates how the bolus did, the 4 hour post-bolus level indicates how well the drip is maintaining the bolus, and the 12-16 hour level indicates steady state.

3. Meds that alter theo clearance:

1. Increased theo metabolism:

a. Barbiturates

b. Phenytoin

c. Isoproterenol

2. Decreased theo metabolism:

a. Allopurinol

b. Cimetidine

c. Erythromycin

d. Propranolol

e. Oral Contraceptives

C. Isoproterenol

1. Nonselective B-adrenergic agonist once used for asthma unresponsive to Aminophylline is **NO LONGER INDICATED** in asthma as it causes a increased myocardial oxygen consumption and may put patients as risk for myocardial ischemia. There are more selective beta-2 agonists that provide all of the advantages of Isoproteranol with less risk.

D. Anticholinergic Agents - The anticholinergic agents are believed to act by blocking the irritant receptors and inhibiting cGMP metabolism which results in bronchodilation. The higher the parasympathetic tone in the patient, the more responsive to anticholinergics they will be.

1. Ipratropium Bromide (Atrovent)

a. Synthetic quaternary ammonium derivative of atropine.

b. When given by inhalation, its' peak effect appears in 30 minutes, and lasts 4-6 hours.

c. Dose - 250 **mcg** for children less than 2 years of age, and 500 **mcg** for older children. The side effects seem fewer than atropine; there is no CNS effect. There is no significant effect on the tracheal mucous transport rate.

d. An Ipratropium Bromide dose of 250 mcg via neb. given along with repeat doses of nebulized Albuterol at 0.15 mg/kg q 20 minutes may reduce hospitalizations from the ER.

E. Corticosteroids (Solumedrol, methylprednisolone)

1. Anti-inflamatory

2. Initial improvement occurs within 6 hours, but a longer time is needed for full affect.

3. Helps speed the resolution of severe asthma refractory to bronchodilator therapy.

4. Optimal dose is not known - 2 mg/kg IV for the initial dose than 1 mg/kg IV every 6 hours thereafter is one recommendation, 2 mg/kg IV every 6 hours x 4 then 1 mg/kg IV every 6 hours thereafter is another. Observe for hyperglycemia and hypertension.

F. Magnesium

1. Theoretical reasons explaining MgSO4's bronchodilatory effect include its' calcium channel blocker capabilities, sedative action, and its' effect in decreasing acetylcholine release from nerve terminals. MgSO4 levels greater than 10 have been associated with cardiac arrest and respiratory insufficiency. Levels must be followed.

2. Suggested dose 25 mg/kg of MgSO4 to a maximum of 2 grams delivered over 20 minutes in children to obtain levels of 4-7 mg/dl. Consider using in patients who are in moderate to severe distress despite usual therapy.

G. Ketamine

1. A dissociative anesthetic which causes amnesia with potent bronchodilator properties.

2. May be administered as a bolus dose of 0.5-1.0 mg/kg followed by a continuous infusion of 0.5 - 1.0 mg/kg/hr. Excersise extreme caution in non-intubated patients due to risk of respiratory depression.

3. Caution is recommended when using ketamine in patients suspected of having increased PVR, especially during spontaneous ventilation without a protected airway. Ketamine increases ICP and should be avoided after head trauma and in other patients at risk for elevated ICP. Also increases secretions.

4. The emergence phenomenon in older patients can be decreased with the concurrent administration of benzodiazepines.

H. Heli-ox

1. Helium is an inert gas with lower density than ambient air. The addition of helium to inhaled gases enhances the diffusibility of the gases by decreasing resistance in areas of turbulent flow.

2. 60-80% helium, 40-20% O2 (Heliox) has been reported to decrease airway pressures and CO2 retention in intubated patients. A trial of Helium-oxygen mixtures should be considered for patients in moderate to severe distress or those mechanically ventilated asthmatics with a respiratory acidosis who fail conventional therapy and who may tolerate 20-40% FiO2.

I. Antibiotic Therapy

1. Respiratory infections are usually caused by viruses so antibiotics have no role in treatment.

2. Think about RSV and mycoplasma. Erythromycin increases theo levels (decreases clearance) - follow theo levels carefully.

J. Hydration and Correction of Acidosis

1. If dehydration is present, it should be corrected. High insensible losses occur due to decreased po intake and increased respiratory losses.

2. Overhydration must be prevented. Monitor for hyponatremia, SIADH and evidence of water intoxication. The more negative intrapleural pressure during severe asthma favors fluid accumulation in the interstitial space around the bronchiole.

3. Patients who can **not** guard their airway sufficiently due to respiratory distress (especially small children), should be kept NPO to avoid aspiration.

4. IV fluids of 1 X maintenance are usually adequate. Tachycardia may occur from asthma drugs. UOP and cap. refill are better indicators of hydration status.

5. Monitor for hypkalemia secondary to beta agonists.

6. If you wish to correct a metabolic acidosis, Tham is the agent of choice, as ti will not further compromise ventilation.

(Administration of HCO3 will result in conversion to CO2 and may further compromise ventilation)

K. Chest Physical Therapy

1. In selected patients who manifest severe mucous hypersecretion, postural drainage, chest vibration and percussion may be beneficial.

L. Oxygen

1. Administer humidified O2 to keep saturations at $\geq$ 90%.

2. Hypoxemia is associated with air hunger, anxiety, bronchoconstriction and increased bronchial reactivity.

M. Mechanical Ventilation

1. Due to high airways resistance, patients with severe asthma are difficult to manage on mechanical ventilation. Complications occur at nearly 3 times the usual rate. Endotracheal intubation may intensify the degree of bronchospasm.

2. Indications for mechanical ventilation

a. The presence of apnea or near apnea, diminished level of consciousness with inability to protect the airway and/or progressive exhaustion.

b. Hypercarbia--although initial hypercapnia in an acute asthmatic is worrisome, these patients should be evaluated individually and most will **not** require intubation. Elevated CO2 may respond to aggressive treatment. If the patient is oxygenating and his/her level of consciousness is reasonable, ther is not a specific CO2 level that mandates intubation.

c. Clinical deterioration despite aggressive therapy as evidenced by paradoxical abdominal movement, cyanosis on 40% FiO2, paO2 < 60 on 6 liters O2, exhaustion, absence of breath sounds, increasing WOB.

3. Rapid sequence induction with Ketamine (bronchdilating properties)and neuromuscular blockade is recommended for intubation. Maintenance of sedation with Ketamine or benzodiazepines during mechanical ventilation is usually adequate, though some patients will benefit from NMB. Continuous NMB in the face of steroids places the patient at risk for stroid myopathy, a state of prolonged muscular weakness that may result in the need for prolonged ventilator support and rehabilitation.

4. The approach to mechanical ventilation generally employs use of low rates to allow for long inspiratory and expiratory time with an I:E ration of 1:2-1:4 to allow pts time to exhale and prevent air trapping; tidal volumes of 8-10 cc/kg in an attempt to limit pead inspiratory pressures, and PEEPs of 2-4. Permissive hypercapnea and

hypoxia are used to limit barotrauma, volutrauma, and oxygen toxicity. ensues. A typical goal is to achieve a pH > 7.20, allowing elevated pCO2's and correcting acidosis with Tham. However, if the patient has a poorly functioning heart or suffered an anoxic cerebral injury, this strategy may be contraindicated.

N. Anesthetics - call Anesthesia.

1. Halothane

a. Concentrations of 0.5-2%.

b. The duration of treatment depends on the clinical response and PaCO2. Once PaCO2 is stabilized and clinical airway obstruction is diminished, as indicated by decreased PIP or decreased wheezing, halothane inhalation can be discontinued.

(1). Side effects

(a). Myocardial depression

(b). Arrhythmias

2. Isoflurane

a. Has fewer side effects than halothane.

b. Isoflurane causes systemic vasodilation secondary to arterial smooth muscle relaxation.

STATUS EPILEPTICUS

I. Initial Management: position on side, protect from injury, loosen clothing.

A. **Airway**

1. Jaw lift

2. Bite block or oral airway if able (no tongue blade or fingers in mouth)

3. Suction secretions or emesis

4. Roll on side.

B. **Breathing**

1. O2 by mask

2. Intubate if needed

a. Address seizures first - will be difficult to intubate and ventilate if patient is seizing (unless you paralize them).

b. May need to intubate for respiratory depression secondary to meds given.

C. **Circulation** - start IV, monitor BP, O_2 SAT.

II. Quick assessment, pertinent history - ask yourself: Why is this patient seizing now? Trauma? Toxin? History of past seizure? Infection? Has the seizure pattern changed?

A. Seizure

1. Description: [precipitating event, onset: focal/generalized, duration], was child post-ictal ?

2. Fever? S/S illness?

3. Previous seizures? (degree, control, etc.)

4. Chronic seizure meds? (dose, compliance, levels)

5. Hx. trauma? (Accidental or non-accidental)

6. Toxin ingestion?

7. Chronic medical problems? Hx. of syncope?

8. Behavior changes?

9. Vomiting? / diarrhea? - Consider inborn error of metabolism in infants.

III. Physical Exam

A. Vital Signs

1. **Evidence of increased ICP / herniation ?** {increased BP, tachycardia (early), bradycardia (occurs late and is an ominous sign), dilated pupils, papilledema}

2. Decreased BP from sepsis, toxins

3. Fever from meningitis

B. Mental Status / Level of Consciousness, Glasgow Coma Scale (see Neurologic Assessment chapter)

C. Respiratory Pattern

1. Assure good air exchange

2. Abnormal patterns with worsening level of consciousness

D. HEENT

1. Pupils (size, reactivity)

2. Fundi (papilledema, hemorrhage)

3. Signs of head trauma

4. Signs of meningismus

E. Neuro. Exam - focal signs, level of consciousness

IV. Labs/X-ray

A. STAT dextrostix - remember, it only takes a drop of blood and you get the result quicker than a lab glucose !

B. STAT anticonvulsant levels

C. STAT lytes, BUN, creatinine, glucose, Ca++, Mg++, calculate anion gap (Anion Gap described in Pediatric Resus. chapter)

D. CBC with diff (sepsis) and blood cultures as indicated.

E. Serum Tox screen ("coma/dangerous drug panel")

F. Other studies (if indicated): LFT's/ammonia (Reyes), lactate and pyruvate esp. in infants, FEP (lead poisoning), inborn error w/u., consider long QT syndrome especially if the patient has a history of recurrent syncope, syncope with exercise, is an athlete or has a positive family history for sudden death, syncope, or cardiomyopathy.

G. Lumbar puncture (LP)

1. This is to evaluate for meningitis - antibiotics should **not** be held if you think the patient has a CNS infection!! You have about 4 hours to tap the patient after antibiotics and still be able to evaluate the CSF, so consider early treatment.

2. Needed on some patients, but not emergently. R/O increased ICP with a head CT before LP!! **Focal exam findings or focal seizures are especially important indications of possible CNS mass lesions.**

3. An LP is needed for febrile patients with S/S infection, and for comatose patients **(after CT).** If septic or unstable, push antibiotics STAT, and LP later. Consider Acyclovir (10 mg/kg/dose) IV every eight hours if encephalitis is suspected,(send viral and herpes cultures on the CSF, and do nasopharyngeal, rectal, eye, and urine viral cultures)

4. Obtain CSF opening and closing pressures.

H. CT of Head

1. If evidence increased ICP, head trauma, or comatose.

2. Also CT if: new onset seizure, suspicious circumstance or focal findings on exam, or focal seizure.

I. EEG once stable or at Peds neurology discretion. Arrange for immediately if seizures do not respond to standard anticonvulsants.

V. Treatment

A. Glucose if hypoglycemic, 0.5-1.0 gm/kg = 1-2 cc/kg D50.

B. Lorazepam (Ativan)

1. 0.1 mg/kg IV, over 1-2 min, up to 2 mg - repeat after 5-10 mins. if seizures continue. May be given PR.

2. Alternative: Diazepam (Valium) - 0.5 mg/kg IV over 1-2 mins., q 10 - 30 minutes, up to 10 mg. May be given PR or via ETT.

3. Major problems:

a. Short half life - (especially Lorazepam), may need longer acting medications.

b. Respiratory depression, esp. in combination with phenobarb (be prepared to intubate).

C. Phenytoin (Dilantin)

1. 20 mg/kg IV over 20 min. 1 gm max.

2. Major side effects are hypotension and arrhythmia. May cause sedation or ataxia.

3. Phospheytoin, newer derivative, same dose in phenytoin equivilents, may be given IM iv necessary.

C. Phenobarbital

1. 20 mg/kg IV over 20 mins.

2. Can be given IM if no IV established

3. Requires several minutes to work

4. Major side effects are sedation and respiratory depression.

D. If patient is on chronic phenobarb or phenytoin: use small boluses of same drug (5mg/kg) until levels are available.

E. Pyridoxine 100 mg IV should be given if standard anticonvulsants are ineffective in a child under 1 year with unexplained status. May cause respiratory depression if given too rapidly.

F. If above measures are ineffective, proceed to pentobarbital coma - call neurology, EEG, admit to ICU, have dopamine and fluids ready, and intubate the patient (if not already done).

SYNCOPE

I. INTRODUCTION:

A. Syncope may be defined as a sudden fall in blood pressure or failure of cardiac systole resulting in cerebral hypoperfusion and subsequent transient loss of consciousness. It is commonly called fainting.

B. Fainting is not a rare event. It is most often the result of vasovagal reactions which are usually benign. However, some causes of syncope are life-threatening.

C. A patient with a history of syncope deserves careful evaluation because syncope, especially if it recurs, places the patient and others at risk for injury. This is especially true if the episode occurred while crossing a street, swimming, driving, climbing or operating machinery.

D. In addition, when heart disease is the etiology for syncope, a risk of serious dysrhythmias and sudden death exists.

E. Syncope may occur at least once in up to 50% of adolescents and accounts for 1 - 3% of all Emergency Dept. visits. The pediatricians task is to identify the small subset with significant disease.

II. ETIOLOGIES:

A. Causes of syncope in children and adolescents (6 major categories):

1. Abnormal circulatory control, vascular volume or tone:
   a. Vasovagal syncope - i.e. occurs in a hot crowded room, tight collar etc.
   b. Pallid infantile syncope
   c. Acute volume depletion i.e. hemorrhage, dehydration
   d. Chronic hypovolemia i.e. diuretic abuse, idiopathic
   e. Orthostatic hypotension i.e. idiopathic, postexercise, familial dysautonomia
   f. Situational syncope i.e. micturition, swallowing cold fluids, defecation, coughing
   g. Pregnancy

2. Cardiac abnormalities:
   a. Tachyarrhythmias:
      1. Long QT syndromes i.e. familial dominant (Romano-Ward syndrome), familial recessive deafness assoc. (Jervell Lange-Nielsen syndrome), idiopathic nonfamilial long QT syndrome, drug associated long QT/torsades de pointes/ventricular tachycardia from quinidine, procainamide, amiodarone, terfenadine (potentiated by IV macrolides - erythromycin, troleandomycin, or ketoconazole), astemizole, cyclic antidepressants, IV pentamidine
      2. Supraventricular tachycardias i.e. rapid reentrant supraventricular tachycardia with hypotension, Wolff-Parkinson-

White syndrome with atrial fibrillation, exercise-induced SVT associated with Wolff-Parkinson-White syndrome
3. Ventricular tachycardia (VT) (unassociated with long QT syndromes) i.e. arrhythmogenic right ventricular dysplasia, exercise-associated (catecholamine sensitive) VT, “sleep death” in young Southeast Asian males (especially from Cambodian or Laotian descent), rapid monomorphic sustained VT after repair of Tetralogy of Fallot

b. Bradyarrhythmias:
1. AV block (heart block) i.e. congenital, tumor of the AV node, L-transposition (congenitally corrected transposition), idiopathic, neuromuscular disorders with AV block, Lyme disease
2. Sinus node disease i.e. after atrial repair of transposition, idiopathic, sick sinus syndrome

c. Structural heart disease:
1. Left ventricular outflow obstruction i.e. aortic stenosis, carotid artery stenosis, hypertrophic cardiomyopathy (idiopathic hypertrophic subaortic stenosis or IHSS)
2. Right ventricular outflow obstruction i.e. pulmonic stenosis, pulmonary embolism, primary pulmonary hypertension
3. Atrial myxoma
4. Dilated cardiomyopathy i.e. idiopathic, caused by coronary anomalies, caused by myocarditis
5. Mitral valve prolapse
6. Pericardial effusion or pericarditis with tamponade
7. “Tet” spells

d. Myocardial infarction (rare)

3. Metabolic:
a. Hypoglycemia
b. Hypocalcemia
c. Hypomagnesemia
d. Hypoxia
e. Imbalances in sodium, potassium, or chloride

4. Central nervous system abnormalities:
a. Seizures
b. Trauma
c. Cerebral vascular accidents (strokes), rare
d. Migraines especially with involvement of vertebrobasilar system
e. Obstruction to cerebrovascular blood flow i.e. cervical spondylosis, Takayasu’s disease, rare
f. Vertigo
g. Central autonomic insufficiency, rare

5. Psychological disorders:
a. Hysteria/ conversion
b. Malingering
c. Hyperventilation
d. Panic disorder
e. Munchausen’s syndrome (by proxy)

f. Laryngeal syncope (Charcot's vertigo)

6. Drugs (the following agents are *not* usually associated with QT prolongation. This contrasts them with the drugs listed above in the long QT section):

a. Antihypertensive agents
b. Cocaine and other illicit drugs
c. Alcohol
d. Barbiturates
e. Phenothiazines
f. Nitrates

III. PHYSIOLOGY:

A. Cardiac output (CO) = heart rate (HR) X stroke volume (SV)
Blood pressure = CO X Total peripheral resistance (TPR) so,
BP = HR X SV X TPR

B. Determinants of HR: sinus node function, autonomic control (neural and humoral) i.e. vagal tone (inhibitory), catecholamines (stimulatory), and sympathetic tone (stimulatory)

C. Determinants of SV: circulatory blood volume, venous return (which is altered by muscle tone, tissue pressure, pregnancy, respirations)

D. Determinants of TPR: baroreceptor tone (carotid sinus, aortic arch), arteriolar tone (altered by electrolyte balance, oxygenation status, catecholamines, autonomic tone), drugs, ventricular "C" receptors

E. For example:

1. Stimulation of the carotid sinus may induce bradycardia, decreased peripheral resistance, reduce BP and cause syncope

2. Stimulation of the upper respiratory tract, GI tract, eyes, or skeletal muscles may induce a vagal response and therefore cause bradycardia, decreased peripheral resistance, reduced BP and also cause syncope

IV. DESCRIPTIONS OF SOME OF THE TYPES OF SYNCOPE:

A. Vasovagal syncope (also known as vasodepressor syncope, neurocardiogenic syncope, neurally mediated syncope, a simple faint and ventricular syncope). This is the most common cause of fainting in children and adolescents. Has an abrupt onset of systemic hypotension (the vasodepressor response) when sitting or most often when standing, followed by bradycardia with a slow junctional rhythm or period of asystole (the cardio-inhibitory response). Some patients manifest more of one of these responses than the other. A prodrome may occur - see the next section. The period of unconsciousness is usually brief, < 1 min. Pallor prior to the event may be noted. May be avoided if the person assumes a Trendelenberg or supine position. Headache or fatigue may occur after the event.

B. Pallid infantile syncope (also known as anoxic reflexive seizure, vagal attack, white breath holding spell or pallid breath holding spell). This is one of two types of breath holding attacks.

1. Cyanotic breath holding attacks. These begin with an event that is distressing to the child such as being frustrated, scared, angry such as with temper tantrums, or in pain. The event is usually unexpected, the child cries vigorously, gasps, ceases breathing, becomes cyanotic and loses consciousness. If the apnea was long enough a seizure may be provoked. Ages 1 week to 6 years, but most common in the 16-24 month age range.

2. Pallid infantile syncope is less common than cyanotic breath holding attacks. It also is precipitated by stress or unexpected pain. A few seconds after the triggering event the child falls limply. Crying, pallor or sweating may or may not precede the loss of consciousness. Seizures may also be provoked but are rare. Usually there is no cyanosis or breath holding. Ages 3 mos. to 14 years.

C. Situational syncope. Fainting here occurs in specific circumstances such as during micturition, swallowing cold fluids, defecation, blood drawing, or coughing. Vagally mediated.

D. Tachyarrhythmias - SVT, long QT syndrome etc. Syncope in the long QT syndrome is caused by paroxysmal episodes of VT often in a polymorphic pattern called “torsades de pointes”. Sudden death may ensue. May occur during sudden exertion, after a startle response from a loud noise or fright, or after an emotional upset. When the patient is in sinus rhythm the QT may be only mildly prolonged therefore a high index of suspicion is needed.

E. Bradyarrhythmias. Syncope from heart block is more common in children post-op from cardiac surgery (even when years earlier) or with a prior history of lesser degrees of heart block. Children with a history of VSD, Tetralogy, or double outlet right ventricle repairs are at increased risk. Syncope from bradyarrhythmias also may occur in any child with structural heart disease or in neonates born to mothers with lupus.

F. Neurologic causes - seizures may be confused with syncope **and vice versa**. Usually syncope manifests nausea, pallor, or sweating (vasovagal) while seizures may show cyanosis of the face (not pallor), frothing of the mouth, tongue biting, postictal sleepiness, and more prolonged unconsciousness (> 5 min.). The setting in which the spell occurred, the family history, and the patient’s memory of feeling “faint” may help distinguish the majority of cases.

G. Psychological causes - hysteric faints are unassociated with decreased BP, decreased HR, or pallor. They may occur in the supine position. Fluttering of the eyes behind half-closed eyelids may be noted. Hyperventilation syncope is believed to be the result of cerebral vasoconstriction from self induced hypocapnia. Frequent symptoms are tachypnea, anxiety, and breathlessness, blurred vision and lightheadedness.

V. MANAGEMENT APPROACH:

A. HISTORY: Details of the history are particularly useful to evaluate the cause of syncope. Up to 25% of children who die suddenly had a prior syncopal episode. Syncope that occurs with exercise, occurs in an athlete, is recurrent, or that occurs in a patient with a positive family history of syncope, sudden death, or cardiomyopathy should be closely evaluated.

1. Age - gives clues to the most common etiologies

a. Syncope in an infant should always be aggressively investigated. Myocardial tumors, outflow obstructions, myocarditis, cardiomyopathies, long QT syndrome, and seizures are all possible.

b. Syncope in adolescents may be caused by IHSS, mitral valve prolapse, coronary artery abnormalities, pulmonary hypertension, anemia, pregnancy, psychological problems, or drug abuse. The most common cause of syncope in adolescents is vasovagal syncope or orthostatic hypotension.

c. Any age may suffer from dysrhythmias, sick sinus syndrome, AV block, seizures, electrolyte imbalances, volume depletion, hypoxia or hypoglycemia.

2. Family history:

a. Be especially aggressive in evaluating patients with a positive FH of cardiomyopathy, “enlarged hearts”, sudden death, syncope associated with exercise, recurrent syncope, deafness, or syncope occurring in trained athletes. **All of these patients should be evaluated by a cardiologist as they constitute high-risk subgroups !**

3. Did a prodrome occur ? What was the patients position ?

a. Vasovagal syncope occurs while standing or sometimes while sitting, but is virtually unheard of in the supine or prone positions. It often (but not always) is associated with the following prodrome. This prodrome is **NOT** the same as the aura perceived by some patients with seizures or migraines.

PRODROME IN VASOVAGAL SYNCOPE	
Psychophysiologic Factors	**Signs/ Symptoms**
Hunger	Pallor or clammy skin
Fatigue	Sweating
Illness	Dilated pupils
Hot, humid or crowded room	Blurred vision, visual gray-out

Pain	Nausea or epigastric distress*
Anxiety*	Lightheadedness*
Perceived threat*	Dizziness*
Sight of blood	Weakness
Tight collar	Sensation of warmth

* may occur in seizure patients as well

b. Syncope that occurred after a startle reaction such as after a loud noise, may indicate long QT syndrome or pallid infantile syncope.

4. Is a witness available ? Was there focal neurologic activity ? Incontinence ? How long did the patient remain unconscious ? What was the quality of the recovery ?

a. An abrupt loss of consciousness suggests epilepsy or a cardiac electrical disturbance. **Some patients with cardiac etiologies for syncopal episodes may show focal or generalized neurologic activity as well !**

b. Pallor that persists and cyanosis may be seen with dysrhythmias.

c. Prolonged unconsciousness suggests severe left or right ventricular outflow tract obstruction.

d. If upon regaining consciousness the patient shows diminished alertness, abnormal tone or motor movements, slurred speech, impaired memory, sore muscles or focal neurologic deficits a seizure disorder should be suspected.

5. Certain questions help clarify the etiology of syncopal episodes.

a. Affirmative answers to the following questions suggest psychiatric causes for the syncope. This may be unassociated with cerebral hypoperfusion.

1. Was the patient recumbent at the time ?

2. Was there any odd behavior prior to the episode ? - hyperventilation or hallucinations ?

6. Ask about the past medical history. Was this the first episode ? Remember that recurrent syncope requires careful evaluation. Is there known: heart, kidney, lung, or liver disease ? Is there a history of a metabolic disorder ? Diabetes ? Thyroid disease ? CNS disease ? Use of medications ? Fever ? Emotional stress or fatigue ?

7. Was the child coughing ? Laryngeal syncope is a paroxysmal neurosis characterized by attacks of coughing with unusual sensations such as tickling in the throat followed by loss of consciousness. Severe coughing which leads to syncope is also seen with situational syncope which is probably vagally mediated. Situational syncope is

also seen with straining during micturition, swallowing hard, swallowing cold fluids, and defecation.

8. For females of the appropriate age: have they missed their periods (pregnancy) or had heavy menses (anemia) ?

9. Was the patient exercising ? **This is very important to ask !**

a. Vasovagal syncope is rarely associated with exercise. Sudden death in the high-risk groups previously mentioned is provoked by exercise. Exercise develops the cardiovascular system and is associated with increased vagal tone and lower resting heart rates. Athletes may have first degree AV block or Wenckebach (Mobitz 1) block which may be benign. Progression to higher order blocks is rare unless there is underlying heart disease. Some athletes do show progression which usually resolves upon discontinuation of the training. Others, however, may continue to manifest bradycardia and have true sinus node dysfunction. Athletes with resting heart rates < 40 BPM, dizziness, recurrent syncope, or a near-miss sudden death experience may require pacemakers.

b. Exercise modulates autonomic discharge. It increases catecholamine levels and may lead to myocardial irritability and dysrhythmias - {exercise-associated (catecholamine sensitive) VT}

c. Patients with mitral valve prolapse may have dysrhythmias with exercise and sudden death. They also are at risk for cerebrovascular ischemic episodes, bacterial endocarditis and resulting cerebral emboli.

d. Children with a long QT syndrome may develop ventricular dysrhythmias with exercise or emotional excitement which triggers autonomic nervous system imbalances. They may require β-blockers or left stellate ganglion ablation.

e. Exercise induced syncope may occur in patients with IHSS, anomalous coronary arteries, or primary pulmonary hypertension.

10. Palpitations before syncope are sometimes associated with tachyarrhythmias or hyperventilation syncope.

B. PHYSICAL EXAMINATION:

1. Begin with a **complete** physical examination and full set of vital signs. Include a measurement of the BP while supine, sitting, and standing. A child with acute volume depletion from a GI disturbance, blood loss, dehydration, or with autonomic dysfunction (true orthostatic hypotension) may have a > 20 mm Hg decrease in the systolic BP or an abnormal increase in the HR upon standing.

2. Pulses - quality of all pulses including the carotids ?

3. Pallor ?

4. Masses in the ears, nose, throat, abdomen ?

5. Bruits ?

6. Location of apical impulse ?

7. Heart murmurs or extra sounds ? Changes in heart sounds upon standing (decreasing venous return) or squatting (increasing venous return) ?

8. Focal neurologic findings ?

9. Mental status ?

10. Possibility of pregnancy ?

11. Ocular compression or vagal maneuvers should only be performed with cardiology input.

12. To check the autonomic nervous system:

a. Submerge a hand in cold water for several minutes. Normally the BP increases.

b. Ask the patient to Valsalva, i.e. grunt or bear down. Normally the BP increases, followed by a mild decrease then a prominent increase with slowing of the HR. Do **not** perform this in pregnant patients or those placed at risk from increased intracranial pressure.

c. Patients with abnormal autonomic nervous systems will not increase their BP with cold water hand immersion. They will decrease their BP then increase their BP but not have a change in HR with the Valsalva maneuver. These findings suggest primary dysfunction of their autonomic nervous systems such as occurs with Riley-Day syndrome and may suffer repeated syncopal episodes from orthostatic hypotension. Children with Riley-Day syndrome are usually recognized in infancy with other manifestations such as chronic aspiration from poorly coordinated swallowing, excessive salivation, sweating, decreased tear formation, labile hypertension, and diminished pain sensation.

C. LABS / TESTS:

1. ECG, continuous monitoring while evaluating the patient

2. CBC and differential

3. Lytes, $HCO3^-$, BUN, creat, glucose **and** dextrostix, calcium, phosphorus, magnesium. Calculate the anion gap.

4. If indicated: ABG, pregnancy test, tox. screen, drug levels

5. Echocardiography if a cardiac etiology is suspected, syncope is recurrent of an undetermined origin, in an athlete, or with a positive family history of cardiomyopathy, sudden death, or high risk factor

6. EEG if a neurologic basis is suspected, or syncope is recurrent of an undetermined origin

7. Holter monitor/ telemetry in athletes, child with suspected heart disease, recurrent syncope of an undetermined origin, or dysrhythmia

8. Others: electrophysiology studies, tilt testing, exercise stress testing, cardiac catheterizations are per cardiology. CNS imaging studies (MRI, CT, etc.) per neurology or if CNS etiology is suspected.

D. TREATMENT - depends on the type of syncope. CPR training may be required by parents or other family members especially if the patient has a type of syncope placing them at high risk for sudden death. For vasovagal syncope, the patient should lie down with their feet elevated if the prodrome occurs. (Usually adolescents balk at this until you tell them that this will be less embarrassing than a full faint.) Avoidance of the situations which cause the syncopal episode is important. Certain athletes may need to restrict training. For orthostatic hypotension, a more gradual shift from lying down to sitting or standing may help. Cardiac syncope will be treated by the cardiologists and may require drugs, pacemakers, ablation procedures, ganglionectomies, or surgery. Drugs have been used for many types of syncope including recurrent vasovagal syncope, and cardiac syncope. Naturally, if anemia, electrolyte imbalance, hypovolemia, hypoglycemia, or other easily treatable cause is found, the treatment will be replacement of the deficient factor and an investigation as to why the condition developed.

E. RESTRICTIONS:

1. Protect the child and those around them.

2. Sports may have to be limited in athletes or in cases of heart disease to curtail syncopal episodes.

3. Driving, climbing, operating machinery, swimming, crossing streets unaided and other potentially dangerous activities may have to be restricted depending on the etiology for the syncope.

F. FOLLOWUP:

1. Required if the syncope is recurrent or in a high risk category.

2. May need neurology, or psychiatry consults as applicable.

3. The potential for a poor outcome is highest if a cardiac etiology is found. These children need especially careful re-evaluation.

TRANSFUSION REACTIONS AND BLOOD COMPONENT THERAPY

I Transfusion Complications

A. Hemolytic transfusion reactions

1. Signal Symptoms:

a. Anxiety
b. Red or black urine, flank pain
c. Nausea, vomiting, pain, cheat tightness, headache
d. Chills, shaking
e. Fever
f. Shock with subsequent renal and other end organ failure

B. Non-Hemolytic transfusion reactions

1. Allergic: urticaria, pruritus, flushing, rarely angioedema or anaphylaxis
2. Isolated Fever/ Infection- fever, headache, hypotension, flushing, emesis, diarrhea
3. Circulatory overload: hypertension, sx of CHF
4. Air Embolus: shortness of breath, chest pain, anxiety
5. Hypothermia chills, low temperature, irregular heart rhythm, possible cardiac arrest.
6. Hyperkalemia: nausea, diarrhea, muscular weakness, paralysis, extremity paresthesias, bradycardia, apprehension, arrest.
7. Acidosis
8. Depletion of clotting factors: bleeding symptoms
9. Hypocalcemia: myoclonus, tetany

II. Treatment

A. STOP TRANSFUSION IMMEDIATELY:

1. Assess and treat from evidence of shock, observe urine for color change
2. Give 0.9% NaCL through IV at rapid rate (1-2X maint)for 1-2 hr.
3. Consider oxygen
4. Treat symptoms (Tylenol, Benadryl, epinephrine)
5. Verify correct product was given
6. Laboratory evaluation (suspected hemolytic reaction, or if transfusion is discontinued.)

B. Hemolytic Reaction: assume if hemoglobinuria, shock, or clerical error identified, treat ASAP

1. Give mannitol 0.25 grams/kg IV to force diuresis, Desired UOP after mannitol:
   a. Infant: 10 cc/hr
   b. Child < 20 kg: 40 cc/hr
   c. Child > 20 kg: 60 to 100 cc/hr
2. Give $NaHCO_3$ 3 meq/kg/12 hours, to urine to pH > 7

3. Begin treatment for acute renal failure. IV rate should match urine output and insensible losses.

C. Febrile Reactions:
   1. Defined as 2 degrees Fahrenheit increase during transfusion.
   2. Administer tylenol (not aspirin or NSAID's to patients with thrombocytopenia.)
   3. Discontinue transfusion, then Lab evaluation with cultures.
   4. Start antibiotics in neutropenic patients, and other patients at risk.

D. Allergic reactions:
   1. Treat mild urticaria or pruritis with benadryl 1 mg/kg IV or po
   2. Can restart after 15-20 min if mild symptoms, alleviated with meds
   3. Anaphylaxis: Administer epinephrine, discontinue transfusion, lab evaluation.

E. Rigors
   1. Especially problematic, most common platelet reaction in patients repeatedly transfused
   2. Assume this could be a hemolytic reaction or infected blood component
   3. Treat as above for fever.

F. Circulatory overload:
   1. Place child upright with feet in dependent position. Lasix 1 mg/kg IV.
   2. Start O2
   3. Consider PEEP, pressors etc

III Laboratory evaluation

A. Required by blood bank: (new products will not be released until the transfusion w/u is completed)
   1. Red top: 5 to 10 cc of patient blood
   4. The blood bag, unused donor blood and transfusion set up. (a repeat crossmatched is performed)
   5. Urine: A UA is done for hemoglobin
   6. Purple top: A CBC is done

B. Consider doing:
   1. PT/PTT, fibrinogen.
   2. Serum and urine hemoglobin.
   3. Bilirubin (Total/Direct) . If the reaction is hemolytic, the indirect bilirubin often rises in the first few hours and is usually normal by 24 hours.
   4. If sepsis is suspected, get blood cultures from both the patient and the donor blood.

IV Prevention of Transfusion Reactions

A. Blood products must be completed before expiration (4 hours-PRBC's, 1 hr- Platelets)

B. Most lethal transfusion errors occur because of human clerical error.
   1. Verify patient identification.
   2. Verify correct blood product.
   3. Identifying donor and recipient blood types before transfusion is begun.

C. Transfuse blood slowly for the first 15 to 20 minutes. Someone should remain with the patient during this time to monitor for acute transfusion reaction.

D. Allergic Reactions:
   1. Premed future transfusions: Benadryl (0.5-1mg/kg), consider Solumedrol (1mg/kg)

E. Febrile Reactions
   1. May give acetaminophen for prophylaxis.
   2. Leukocyte-poor (leuko-depleted) platelets are less likely to cause reaction.

F. Rigors
   1. For patients with repeated history: plasma removal and/or premed with Solumedrol (1mg/kg)

G. Circulatory Overload
   1. Transfuse blood slowly (max 5cc/kg/hr for routine transfusion, max 1cc/kg/hr for chronic Hb <5)
   2. Consider ordering a "split pack", which is a half unit and giving over 2 four hour aliquots.
   3. Platelets are given rapidly, be cognizant of volumes
   4. Platelet volumes ea. random unit = 50 cc("6-Pack"= 300cc), (pheresis unit = 300-400 cc)

H. Air Emboli:
   1. May occur when blood is transfused under pressure.
   2. Normalize pressure before container is empty when infusing with pressure cuff.
   2. Clear tubing of air by aspirating air with syringe at nearest Y connector. If air is observed in tubing, disconnect tubing and allow blood to flow until air has escaped.

I. Hypothermia:
   1. From rapid infusion of refrigerated blood
   2. Must use blood warmer (PICU and NICU only) if anticipated > 20cc/kg PRBC over 12 hours
   3. Never use microwave oven

J. Hyperkalemia:
   1. Use washed PRBCs or fresh blood if patient is at risk
   2. Observed with massive transfusions, exchange transfusions or patients with renal insufficiency.

V. Selection of Therapeutic Blood Components

A. Whole blood
   1. Fresh whole blood is unavailable
   2. Blood can be reconstituted to a desired Hct using FFP or saline by the blood bank only, on request. (i.e. priming pheresis or ECMO circuit)
   3. Blood reconstituted in FFP can be used in treating symptomatic large volume deficits.
   4. Practical use in trauma: PRBCs are run cc/cc with NS. Remember to give FFP if approaching one blood volume transfusion.

C. Packed red blood cells (PRBCs)
   1. Most commonly used form of RBCs.
   2. Indications: symptomatic anemia.
   3. Criteria for transfusion varies (preprinted orders have institutional recommendations)
      a. A hemoglobin level of 8 g/dL is not a hard and fast indication for transfusion.
      b. Dose is usually 10 ml/kg. Round to the nearest unit (approx 380cc) or consider ordering a split pack.
      c. If patient has long-standing severe anemia it is safer to give only lmL/(Hgb in g/dL)/kg.
         For example: if a patient with iron deficiency anemia has a hemoglobin of only 5 g/dL and weighs 20kg, give: lmL X 5 X 20 = 100cc.

D. Leukopoor RBC’s (leukocyte filter)
   1. This is not the same as the particulate filter that is within the infusion tubing.
   2. Generally recommended in patients who will receive repeated transfusions
   3. Will reduce chances of CMV infection if donor is CMV positive
   4. Almost all peds patients (transplants, chronic illnesses, infants, cancer, sickle)

E. CMV negative RBC’’s
   1. Used in CMV negative patients who are transplant candidates.
   2. Also used in neonates and ECMO candidates.
   3. Leukofilter may come to substitute for CMV testing in the near future.
   4. No proof that CMV- blood is necessary in a Patient who is + for CMV

F. Irradiated Blood products
   1. Inactives T-cells to block a GVH response
   2. All immunocompromised patients (including all neonates)
   3. All “fresh” products, (ie not FFP)

G. Granulocytes /WBCs
   1. Rarely used due to severe transfusion reactions due to leukocyte antigens.

2. Used only for severe infections in patients with prolonged neutropenia.

H. Fresh frozen plasma FFP)—See chapter on coagulopathies.

I. Cryoprecipitate— See chapter on coagulopathies.

J. Random donor platelets— See chapter on coagulopathies.

1. Pheresis platelets-Indicated if multiple units of platelets are expected to be given or if alloimmunization has occurred.
2. One pheresis unit equals approximately 4 random donor units.

K. Factor VIII concentrate (anti-hemophilic factor) and Porcine factor VIII concentrate— See chapter on coagulopathies.

L. Prothrombin complex concentrate (factor IX complex) and Activated prothrombin complex concentrate— See chapter on coagulopathies.

M. Donor Directed Blood donation:
1. This service is available to patients on request (Autologous and allogeneic).
2. Requires minimum of 3 business days to arrange
3. There are very few autologous indications in pediatrics
4. Be advised that infectious risks are dramatically increased
   a. Potential donors are not usually as educated as the random volunteer donor pool regarding infectious risks
   b. potential donors are relatively coerced on the behalf of the ill patient
   c. potential donors may not be frank especially with family members about behavioral risks

TRAUMA

I. Introduction:

A. **Trauma is the leading cause of death and disability in the pediatric age group.** While principles of resuscitation for trauma are the same as for nontraumatic pediatric patients, some aspects of the stabilization phase are unique to the trauma setting. This chapter does **NOT** take the place of the Advanced Trauma Life Support class (ATLS). Its' purpose is to expose the pediatric resident to fundamental concepts of trauma care. The information in this chapter is taken primarily from the ATLS and PALS courses.

B. A trimodal distribution of death from injuries occurs. The first peak of death is within minutes of the injury. Deaths during this time are usually due to lacerations of the brain, brain stem, higher spinal cord, heart or great vessels. The second peak occurs within minutes to several hours of the injury. This is the period that ATLS focuses upon. Deaths here occur secondary to subdural or epidural hematomas, hemopneumothoraces, a ruptured spleen, liver lacerations, pelvic fractures and/ or multiple injuries with significant blood loss. The third death peak occurs several days to weeks after the initial injury. These deaths are usually secondary to sepsis, multiple system organ failure (MSOF), or unrecoverable pulmonary injury.

C. The child with multisystem trauma may have **both** cardiorespiratory failure and shock. A rapid cardiopulmonary system evaluation must be performed as well as a rapid thoracoabdominal examination to detect life threatening chest or abdominal injuries which may interfere with a successful resuscitation. For instance, ventilation, oxygen and perfusion therapies may be ineffective until a tension pneumothorax is treated.

D. Basic ATLS concepts include:

1. Treat the greatest threat to life first.

2. The lack of a definitive diagnosis should never impede the application of an indicated treatment.

3. A detailed history is not an essential prerequisite to begin evaluating an acutely injured patient.

E. Improper resuscitation has been identified as a major cause of preventable pediatric death. Common errors in resuscitation include failure to:

1. Open and maintain the airway.

2. Provide appropriate and adequate fluid resuscitation to head injured children.

3. Recognize and treat internal hemorrhage.

F. A qualified surgeon should be involved ASAP in the resuscitation.

G. A child with a pediatric trauma score of 8 or less **or** a child with a revised trauma score of 11 or less should be transported to a pediatric trauma center. (***see following tables***)

PEDIATRIC TRAUMA SCORE

	Coded value		
Patient characteristics	**+ 2**	**+ 1**	**- 1**
Weight (kg)	> 20	10 - 20	< 10
Airway	Normal	Maintained	Unmaintained
Systolic BP (mm Hg)	> 90	50 - 90	< 50
Central nervous system	Awake	Obtunded	Coma
Open wound	None	Minor	Major
Skeletal trauma	None	Closed	Open, multiple

REVISED TRAUMA SCORE

Glasgow Coma Scale Score *	Systolic Blood Pressure (mm Hg)	Respiratory Rate (breaths/ min)	Coded Value
13 - 15	> 89	10 - 29	4
9 - 12	76 - 89	> 29	3
6 - 8	50 - 75	6 - 9	2
4 - 5	1 - 49	1 - 5	1
3	0	0	0

* ***Glasgow Coma Scales (GCS) are in the Neurologic Assessment, ... chapter.***

H. At WHMC, pediatric trauma patients are primarily managed by a trauma team. Usually our pediatric surgeons head these teams. We assist in the initial stabilization of the patient but remain under the primary guidance of the trauma team. The pediatric dept. PICU team **consults** on all of these patients and facilitates fluid, electrolyte, and ventilation management. We also offer **suggestions** concerning other problems or organ system management.

II. General approach to the trauma patient

A. Trauma patients should be taken to the closest appropriate facility. Once triaged, the patient is assessed and assigned a treatment priority which is based on their injuries, stability, and the injury mechanism.

B. The **Primary Survey** is performed next to identify and **simultaneously** manage life threatening conditions. (ABC's plus D and E)

A = Airway maintenance **with cervical spine control**
B = Breathing and ventilation
C = Circulation with hemorrhage control
D = Disability: neurologic status
E = Exposure/ Environmental control: **completely** undress the patient, but prevent hypothermia

1. Airway - assess for signs of airway obstruction such as foreign bodies or facial, mandibular, or tracheal/ laryngeal fractures.

Cervical spine protection must be insured (use chin lift/ jaw thrust). Do not hyperextend, hyperflex, or rotate the cervical spine. Cervical immobilization should be achieved.

2. Breathing - auscultation, percussion, inspection, and palpation should be performed to assess for tension pneumothoraces, flail chest, pulmonary contusions, open pneumothoraces, fractured ribs etc.

3. Circulation with hemorrhage control - hypotension after trauma should be considered as hypovolemic in origin until proven otherwise. Level of consciousness if reduced, may be from cerebral hypoperfusion. Skin color especially if ashen gray or white are signs of hypovolemia. Rapid, thready pulses are early signs of hypovolemia. Rapid external blood loss should be managed initially by direct manual pressure on the wound. Pneumatic splints may also lessen hemorrhage. Tourniquets should **not** be used as they crush tissue and cause distal ischemia.

4. Disability - (Neurologic evaluation) - the **"AVPU"** method is used to assess the level of consciousness, and pupillary size and reactiveness. The Glasgow Coma Scale (GCS) may be performed in lieu of the AVPU. The GCS is always performed in the Secondary Survey.

A = Alert
V = responds to Verbal stimuli
P = responds only to Painful stimuli
U = Unresponsive

a. An altered level of consciousness should prompt an immediate reevaluation of oxygenation, ventilation, and perfusion. If these are adequate assume trauma is the etiology for the decreased level of consciousness. Alcohol or drugs may also do this but are a diagnosis of exclusion in the trauma patient.

5. Exposure/ Environmental control - completely undress the patient but protect them from hypothermia. **Warm blankets, warmed IV fluids, conchas for ventilators and a warm environment must be provided.**

C. Resuscitation:

1. The airway must be protected and maintained at all times. Nasopharyngeal airways may be used in conscious patients.

2. Patients with compromised airways, those with ventilatory problems or who are unconscious should be endotracheally intubated. If oral or nasal intubation is contraindicated a surgeon should place a surgical airway. (decision tree follows)

3. Oxygen should be given to all trauma patients. ABG's should be freely used.

4. A minimum of 2 large bore IV's should be placed. Remember if an IV cannot be placed promptly, place an intraosseous (IO) catheter, especially in patients < 6 years old (described in pediatric resuscitation chapter). If patient is in severe shock go directly to an IO. **Do NOT use an IO in a fractured bone.**

5. All patients should receive a Type and crossmatch, CBC, UA, baseline electrolytes and an amylase. Females of child bearing age should receive a pregnancy test.

6. If colloid is needed, and Type specific blood is not immediately available, then O neg. blood may be used. Shock should be assumed to be hypovolemic in origin and should be treated aggressively with fluids rather than pressors, steroids or bicarb.

7. Continuous ECG monitoring must be used.

a. Dysrhythmias, tachycardia, A-fib, PVC's, and ST segment changes may all indicate cardiac contusion.

b. EMD may indicate cardiac tamponade, tension pneumothorax, profound hypovolemia, severe acidosis, pulmonary embolism, pneumopericardium, hypoxemia, hyperkalemia, tricyclic antidepressants, Beta-blockers, Calcium channel blockers, and hypothermia.

c. Bradycardia, premature beats or aberrant conduction patterns may indicate hypoxia, hypothermia, or hypoperfusion

8. Urinary catheters should be placed unless urethral transection or injury is suspected. Contraindications to placing a foley are: 1) blood at the urethral meatus, 2) blood is in the scrotum, or 3) the prostate is high riding or cannot be palpated. Therefore, an exam of the genitalia and rectum is required prior to urinary catheter insertion.

9. Gastric tubes should be placed to reduce stomach distention and decrease the risk of aspiration. If the cribiform plate is fractured or suspected to be fractured, the gastric tube should be placed orally or through a properly placed nasopharyngeal airway to prevent intracranial passage. Such a fracture may be suspected in patients with otorrhea, rhinorrhea, Battle's sign, hemotypanum, or raccoon eyes. However, sometimes these findings do not appear until hours after the injury.

10. The following X-rays are always obtained: 1) lateral C-spine, 2) AP chest, and 3) AP pelvis films. Later, complete radiographs of the neck and other areas, CT's etc. should be obtained.

D. **The Secondary Survey:**

1. The secondary survey begins once the primary survey (ABC's) is completed, resuscitation has commenced and the patient's ABC's have

been reassessed. The secondary survey is a head-to-toe evaluation including a full set of vital signs, and complete history and physical examination. A complete neurologic evaluation and the GCS, X-rays, labs and peritoneal lavage (if indicated) are performed here.

2. The history should be complete especially for the mechanisms of the injury, and whether it was blunt or penetrating. The **"AMPLE"** mnemonic is useful.

A = Allergies
M = Medications
P = Past illnesses
L = Last meal time
E = Events/ Environment related to the injury

a. A history in blunt trauma such as from automobile collisions, falls, etc. should include questions concerning seat belt use, infant car seat use, steering wheel deformations, the direction of the impact, damage to the vehicle, speed of the vehicle, mortalities associated with the accident, loss of consciousness, and whether the patient was ejected or not.

b. A history in penetrating trauma such as from firearms, stabbings, or from impaling objects should address the region of the body injured, the velocity of the missile, caliber of the bullet, trajectory, and the distance from weapon to wounded.

c. Histories should include exposure to burns, cold injuries, toxin exposure, chemical exposure, radiation exposure, duration of any such exposure, and extraction time from the vehicle.

III. Specific considerations:

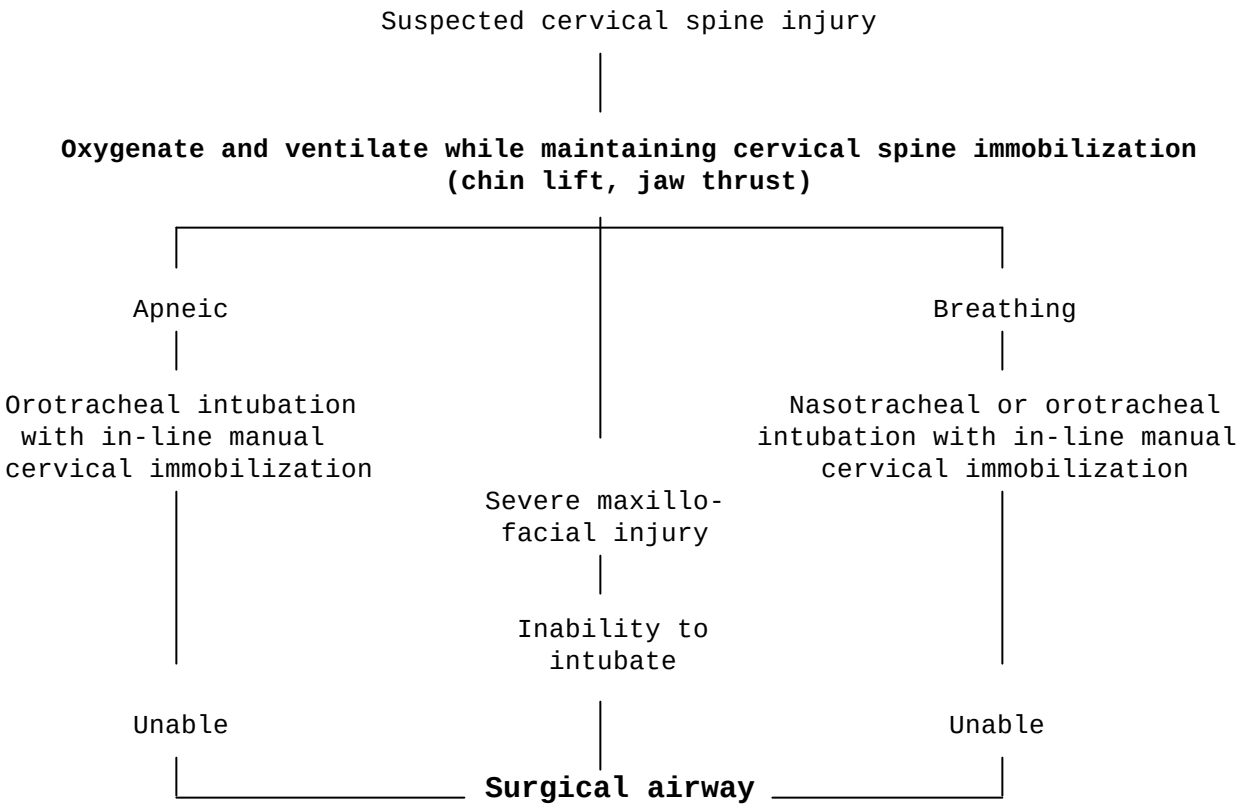
A. Airway - the airway must frequently be assessed for patency and adequacy of ventilation. Failure to heed this advice is one of the most common reasons for failure in trauma evaluation and resuscitation ! Oxygen should be freely used.

1. **A definitive airway is necessary for: apnea, inability to protect the airway with other means, to protect the lower airway from aspiration of blood or vomitus, when potential or impending compromise of the airway may occur such as with facial fractures, inhalational injuries, or sustained seizures, for patients with closed head trauma requiring hyperventilation, for failure to maintain adequate oxygenation by other means, and for GCS's < 9.**

2. The following decision tree helps determine airway appropriateness for apneic patients in whom a cervical spine injury is suspected. **A surgeon will perform a cricothyroidotomy if needed**.

IMMEDIATE NEED FOR DEFINITIVE AIRWAY

Unconscious patient with blunt trauma



B. Shock - this is covered in the GI bleed, newborn and pediatric resuscitation chapters so will only be touched on here. **Remember**, shock should be assumed to be hypovolemic in origin and should be treated aggressively with fluids rather than pressors, steroids or bicarb. This is because almost all shock from trauma is hypovolemic in origin. Neurogenic shock and cardiogenic shock are rare in pediatric trauma patients. Specifics concerning trauma are:

1. Pneumatic Antishock Garment (PASG) - this raises systolic pressure by increasing peripheral vascular resistance and myocardial afterload. **Not used in pediatric trauma patients unless an unstable pelvic fracture is present.**

a. Indications:

(1). Splinting and control of pelvis fractures **with** continuing hemorrhage and hypotension.

(2). Intra-abdominal trauma with severe hypovolemia **only** in patients en route to the OR or another facility.

b. Contraindications:

(1). Pulmonary edema

(2). Known diaphragmatic rupture

(3). Uncontrolled hemorrhage outside the confines of the garment

(4). If inflation of the garment causes an increase in respiratory rate or increased respiratory distress it must be deflated regardless of BP. Assume a diaphragmatic rupture.

C. Abdominal trauma

1. At WHMC abdominal trauma patients always get a CT scan unless going immediately to the OR for a celiotomy.

a. CT scan indications include:

(1). Abdominal distention

(2). Abdominal findings such as: tenderness, masses, or inability to assess the abdomen such as in a unconscious patient

b. Pediatric CT scans are done with enteral, IV and rectal contrast. The dose for pediatric patients follows.

(1). ***Oral*** contrast is dilute gastrograffin (4 cc's in 8 oz. of water). The oral dose of this mixture is 10 cc/**kg**.

(2). ***IV*** contrast is 1 cc/**pound** of weight of non-ionic contrast. This is usually given as a bolus in < 1 minute.

(3). ***Rectal*** contrast is dilute gastrograffin (4 cc's in 8 oz. of water). This is given via a pediatric rectal tube and is delivered via gravity by hanging a bag above the CT table. The exact dose is per the radiologist

2. Diagnostic Peritoneal Lavage (DPL)**, rarely used in pediatric patients** because most intra-abdominal injuries, even if they cause bleeding, are monitored expectantly without surgical intervention. Additionally, CT scans can be obtained relatively quickly. Howver, if a pt is being taken to the OR for another indication and a CT is not practical, DPL may have a role in pediatric trauma.

a. May be 98 % sensitive for intraperitoneal bleeding.

b. The abdomen may sequester large amounts of blood without appearing abnormal.

c. The only **absolute** contraindication for a DPL is an already existing indication for celiotomy (exploration).

d. **Relative** contraindications include: previous abdominal operations, morbid obesity, advanced cirrhosis, or pre-existing coagulopathy. Use in pregnancy is controversial.

e. A positive DPL, and therefore, need for surgical intervention is indicated by:

(1). ≥ 100,000 RBC's/mm^3 (controversial in children)

(2). > 500 WBC's/mm^3

(3). Bacteria on gram stain

(4). Fecal material

3. Indications for celiotomy

a. Hypotension with evidence of abdominal injury (i.e. gunshot or stab wounds, blunt trauma with gross blood on DPL)

b. Peritonitis - early or subsequent

c. Recurrent hypotension despite adequate resuscitation.

d. Extraluminal air

e. Injured diaphragm

f. Intraperitoneal perforation of the urinary bladder on cystography.

g. A positive contrast study of the upper or lower GI tracts.

h. Persistent amylase elevation with abdominal findings.

D. Head trauma

1. Approximately 50% of all trauma deaths are associated with head injury.

2. Anatomy - 5 tissue layers cover the skull bone. "SCALP"

a. Skin

b. subCutaneous tissue

c. galea Aponeurotica

d. Loose areolar tissue

e. Periosteum

- the loose areolar tissue is subject to subgaleal hematomas, scalping injuries, and large flaps from injury

- due to the generous blood supply, a laceration may result in a major blood loss, especially in children

3. Skull - comprised of the cranial vault (calvarium) and the base. The base is irregular and rough therefore allowing injury to occur as the brain moves within the skull during acceleration and deceleration.

4. Meninges - the tough dura adheres to the internal surface of the skull. A potential space under it (the subdural space) exists before you encounter the arachnoid. Hemorrhage can occur into this space, usually from trauma to veins (bridging veins) that traverse the space and cause a subdural hematoma. Meningeal arteries lie between the dura and the internal surface of the skull (epidural space). Laceration of these arteries causes an epidural hematoma. Under the arachnoid is the pia which firmly attaches to the brain cortex. Between the arachnoid and the pia is the subarachnoid space where CSF circulates. Hemorrhage into this space causes a subarachnoid hemorrhage.

5. The brain is comprised of the right and left cerebral hemispheres with the left usually dominant and controlling language, frontal lobe (emotions, motor function), occipetal lobe (vision), parietal lobe (sensory function), temporal lobe (memory, may be relatively silent on the right side), cerebellum and brain stem.

6. **An alteration of consciousness is the hallmark of brain injury.**

7. Examination is based on the “AVPU” mnemonic and a mini-neurologic exam (GCS, assessment of pupillary function and assessment for any lateralized extremity weakness).

a. GCS - if ≤ 8 the patient is considered comatose
- if > 8 patient is not in coma
- if ≤ 8 the patient has a severe head injury
- if 9 - 12 the patient has a moderate head injury
- if > 12 the patient has a minor head injury

b. A difference in pupil diameters of ≥ 1 mm is abnormal.

c. A delay in onset of movement to a painful stimuli, less movement than expected, or need for more stimulus on one side is significant. A lateralized weakness suggests an intracranial mass lesion.

d. A patient has a severe head injury **irrespective of the GCS** if any of the following conditions are present:

(1). Unequal pupils

(2). Unequal motor examination

(3). An open head injury with leaking CSF or exposed brain tissue

(4). Neurologic deterioration (a decrease in the GCS by $\geq$ 2 points)

(5). Depressed skull fracture

e. If headache increases, pupil size increases, or weakness develops on one side then the neurologic status may be deteriorating !! The initial neurologic evaluation is only the beginning. Exams must be repeated !

f. Patients with significant loss of consciousness or the above findings get an immediate head CT without contrast. Depending on the neurologic evaluation these patients may be admitted to the PICU for observation and frequent neuro checks. The treatment of increased intracranial pressure is discussed in its chapter.

8. Types of head injury

a. Skull fracture - common, may not be associated with severe injury, higher likelihood the patient will have intracranial hematoma exists with skull frx., therefore all patients require a neurosurgical consult

(1). Linear, nondepressed frx. - if lies across vascular arterial grooves or suture lines it should raise the suspicion of epidural hemorrhage

(2). Depressed skull frx. - requires elevation

(3). Open skull frx. - dura is torn, requires early surgical intervention

(4). Basal skull frx. - otorrhea, rhinorrhea, eccymosis in mastoid region (Battle’s sign), hemotypanum, raccoon eyes (periorbital eccymoses) are all associated with cribiform plate frx’s.

b. Diffuse brain injuries - associated with acceleration/ deceleration injuries

(1). Concussion - brief loss of neurologic function, confusion, amnesia, or a temporary loss of consciousness may be present. Observation is required

in the PICU.

(2). Diffuse axonal injury - prolonged coma (days - weeks), mortality is up to 33%, results in microscopic damage scattered widely throughout the brain, does not require surgery

c. Focal injuries - contusions, hemorrhages, hematomas

(1). Contusion - may be single or multiple, small or large, associated with severe concussions, coup/ contrecoup forms, commonly in frontal or temporal lobes, edema may result

(2). Intracranial hemorrhages

(a). Meningeal hemorrhage

((1)). Acute epidural hemorrhage - usually from a tear in the dural arteries especially the middle meningeal artery, rapidly fatal, associated with a loss of consciousness, intervening lucid period, then a secondary depression of consciousness, and development of a hemiparesis on the opposite side, and sometimes a fixed and dilated pupil on the same side (Hallmark sign)

((2)). Acute subdural hemorrhage - also life threatening and more common than epidurals, usually due to a rupture of bridging veins between the cortex and the dura

((3)). Subarachnoid hemorrhage - bloody CSF associated with photophobia, headache

b. Brain hemorrhages and lacerations

((1)). Intracerebral hematomas

((2)). Impalement injuries - leave the object in place until a neurosurgeon arrives

((3)). Bullet wounds

E. Spine and spinal cord trauma **(BEWARE OF spinal cord injury with*out* radiographic abnormality (SCIWORA). See below under pediatric nuances.**

1. A vertebral column injury should be presumed and **total** spinal immobilization of the entire patient should be maintained until

screening X-rays are obtained and fractures or fracture-dislocations are excluded for any patient:

a. With an injury above the clavicle or

b. With a head injury resulting in an unconscious state

c. This is especially true if the injury resulted from high speed vehicles

2. Conscious patients can usually identify pain at the injury site and will have a loss of sensation below this level.

3. Unconscious patients may have the following clinical signs suggesting a cervical cord injury:

a. Flaccid areflexia - especially with a flaccid rectal sphincter

b. Diaphragmatic breathing

c. Ability to flex but not extend the elbow

d. Grimaces to pain above, but not below the clavicle

e. Hypotension with bradycardia especially without hypovolemia

f. Priapism

4. Neurologic assessment - 3 spinal cord tracts may be assessed clinically (all are paired tracts which may be singly or dually injured)

a. Corticospinal tract (posterolateral aspect of cord) - controls motor power on the same side of the body. Tested by voluntary muscle contraction or involuntary responses to painful stimuli.

b. Spinothalamic tract (anterolateral aspect of cord) - transmits pain and temperature from opposite side of body. Test by pinch or pin-prick.

c. Posterior columns - carry proprioceptive impulses from the same side of the body. Tested by position sense of fingers and toes or tuning fork vibration.

5. Neurogenic shock results from impairment of the descending sympathetic pathways in the spinal cord. This leads to loss of vasomotor tone and loss of sympathetic innervation of the heart. The patient is vasodilated and hypotensive without hypovolemia but may not become tachycardic and may even be bradycardic. Pressors usually help maintain the BP. Once again, neurogenic shock is rare so trauma patients in shock are treated as hypovolemic initially.

6. Spinal shock occurs soon after the spinal cord injury. Function may not be apparent although all areas are not permanently destroyed and only time will demonstrate return of function. You see flaccidity and loss of reflexes.

IV. Pediatric Trauma Nuances

A. Nearly 22,000,000 children are injured in the U.S.A. each year.

B. Encounters with motor vehicles (as an occupant, pedestrian, or cyclist) account for the largest fatally injured group followed by drownings, house fires, and homicide.

C. Multisystem injury is the rule rather than the exception.

D. The order and priorities of pediatric trauma management are the same for injured children as adults, however, their unique anatomic characteristics deserve special consideration:

1. Because of smaller body mass, energy from linear forces (fenders, bumpers, falls) results in greater force applied per unit body area. Children have less fat, less elastic connective tissue, and close proximity of organs which leads to more multisystem organ injuries.

2. The skeleton is incompletely calcified and is more pliable. Internal organs may be damaged without evidence of overlying bone fractures. If bones are broken assume a massive amount of energy must have been applied.

3. The body surface area to volume ratio is highest at birth so hypothermia may develop quickly.

4. The child’s ability to interact and cooperate with care-givers is limited making history and physical examinations difficult.

5. Shock resuscitation fluids are LR or NS, with 20 cc/kg boluses.

6. Solid abdominal organ injuries (spleen, liver) in a pediatric patient may be observed in the PICU with bedrest and frequent HCT checks rather than taken directly to the OR as with adults.

E. Airway:

1. The smaller the child, the greater is the disproportion between the size of the cranium and midface. This produces a greater propensity for the posterior pharyngeal area to buckle as the relatively large occiput forces passive flexion of the c-spine. To prevent this assume a “sniffing” position, while maintaining c-spine control.

2. Visualization of the larynx may be difficult as the soft tissues (tongue, tonsils) are large compared to the oral cavity.

3. The larynx is more antero-caudal and is naturally shorter so right mainstem intubations occur more easily.

4. **Orotracheal** intubation under direct visualization while maintaining c-spine immobilization is the preferred method of establishing airway control, especially in children 8 years of age or younger.

F. Chest trauma:

1. The child's chest wall is very compliant which allows for the transfer of energy to intrathoracic soft tissues, frequently without any evidence of external chest wall injury. Consequently, pulmonary contusions and intrapulmonary hemorrhages are commonly seen.

2. Mobility of mediastinal structures makes the child more sensitive to tension pneumothoraces and flail segments.

G. Head trauma:

1. Children are particularly susceptible to the secondary effects of brain injury which are produced by hypoxia, hypotension, seizures and hyperthermia. Shock resuscitation and avoidance of hypoxia is critically important to a good outcome.

2. Young children with open fontanelles and mobile cranial suture lines are more tolerant of expanding intracranial mass lesions. They may not decompensate until the mass lesion has become large. A bulging fontanelle or suture diastases should prompt neurosurgical evaluation.

H. Spinal cord injury:

1. Children may sustain spinal cord injury without radiographic abnormality (**SCIWORA**). **Normal spine X-rays do not exclude significant spinal cord injury !!** This is due to the pediatric spine being so much more elastic and mobile than the adult spine. The interspinous ligaments and joint capsules are more flexible, the facet joints are flatter, and the relatively large head allows for more angular momentum to be generated during flexion and extension, resulting in greater energy transfer. Spinal precautions must be maintained (Philadelphia c-spine collar and logrolling for examinations).

2. **Neurosurgical evaluation should be obtained if there is any doubt that a spinal cord injury exists.**

I. Abdominal trauma: Doses of contrast for pediatric abdominal CT's are listed in the previous abdominal trauma section.

J. Shock:

1. Hemorrhagic shock may be classified based on systemic signs as listed in the following table:

Hemorrhagic Shock Classification

	Class I	Class II	Class III	Class IV
Degree of hemorrhage	very mild	mild	moderate	severe
Blood volume loss	< 15 %	15 - 25 %	26 - 39 %	≥ 40 %
Cardiovascular	HR normal or mildly ↑'d, normal pulses, normal BP	Tachycardia, peripheral pulses may be ↓'d, normal BP	significant tachycardia, thready peripheral pulses, ↓'d BP	severe tachycardia, thready CENTRAL pulses, significantly ↓'d BP
pH	normal	normal	metabolic acidosis	significant acidosis
Respiratory	RR normal	tachypnea	moderate tachypnea	severe tachypnea
CNS	slightly anxious	irritable, confused, or combative	irritable, lethargic, or diminished pain response	lethargic, coma
Skin	warm, pink, capillary refill brisk	cool extremities, mottled, delayed cap. refill	cool extremities, mottled or pallor, prolonged cap. refill	cold extremities, pallor or cyanosis
Kidneys	normal urine output	oliguria, increased specific gravity	oliguria, increased BUN	anuria

From: the ATLS manual