وزارة الصحة

MINISTRY OF HEALTH
PAEDIATRIC COUNCIL
CLINICAL GUIDELINES COMMITTEE

GUIDELINES FOR THE MANAGEMENT OF COMMON PAEDIATRIC EMERGENCIES

KUWAIT 2016

GUIDELINES FOR THE MANAGEMENT OF COMMON PEDIATRIC EMERGENCIES

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Fluid Therapy in Diarrhea

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This Summery will address the Following issues:

1. Intravenous rehydrating fluids.
2. Dral rehydrating fluids.
3. Degrees of dehydration , Fluid & Sodium losses.
4. Principles of rehydration, Rapid Fluid resuscitation
5. Maintenance fluid therapy.
6. Example of Fluid therapy.
7. Hyponatremia.
8. Hypernatremia.
9. General Comments

Intravenous Rehydrating fluids

Make yourself familiar with the fluids you use, their sodium and Potassium Content , and their osmolality, (table 1)

Table (1) Composition of Intravenous fluids Solutions (Reference 1)

Solution	NA+ mmoL/L	K+ mmoL/L	glucose mmoL/L	Osmolality Mosmol/kg
0.9% Saline	154	0	0	308
0.45% Saline/Dextrose 5%	77	0	300	454
0.225% Saline/Dextrose 5%	38	0	300	377
Ringer Locate	130	0	4	280
4% Kcl -	500	-	-	1000
8.4 **Sod.bicarbonate**	1000	-	-	2000

Observe :

- Ringer locate & 0.225% saline are rarely used.
- One ml of 4% kcl contains 0.5 mmol/K+ (approx.).
- 100 ml of NS(0.9% Saline)Contains. 15 mmols NA+.
- One ml of Sod.bicarb contains one mmol NA+ &one mmol CO3-

(2) Oral Rehydrating Fluid

Tables (2) Depiets Commonly used Oral rehydrating salts follow manufactures' instructions of the volume of water to be added.

Table (2) Commonly used oral rehydrating salts (Ref 1)

Preparation	NA+ mmoL/L	K+ mmoL/L	glucose mmoL/L	HCO3- Equiralent mmoL/L	Osmolality Mosmol/kg
Who recommended ORS	90	20	110	30	330
Low Sodium ORS	60	20	110	30	270
pedialyte	45	0	140	30	270
Pepsi/seven-up	0	0	700	0	700
Apple juice	0	0	600	0	600

*Not to be used in rehydration . they Contain no sodium & Provide free water in Large Amounts ,and may lead to symptomatic Hypernatremia

(3) Degrees of dehydration (Reference 2)

	Mild	Moderate	server
Volume loss/kg (infants)	50	75	140
Volume loss/kg (Children)	30	60	90
pulse Normal	increased	Increased (weak)	
Capillary Refill (Seconds)	Below 2	2-4	Above 4
Fontanel/ (When open)	Normal	Normal	Depressed
Tears	Present	Decreased	Absent
Mucous Membranes	Normal	Dry	Cyanosed
Blood Pressure	Normal	Normal/low	Low-Shock

Important Consideration:

1. The degree of dehydration may be underestimated in overweight Children and in hypernatremia; and may be overestimated in malnourished children.
2. Shock develops early in hyponatraemia late in hypernatremia.
3. The most sensitive indicators of moderate dehydration are: tachycardia, and capillary refill of over 2 seconds (3)
4. Sever dehydration typically present with early signs of hypovolemic shock (3) , impaired Mental statues ,dry and cyanosed mucous membranes &absent tears.

(4) Principles of rehydration

a) Rapid fluid resuscitation (3-5)
b) Provision of maintenance & losses .
c) Attention to the clinical situation and Sodium Status
d) Rapid re alimentation (early Provision of food).

Table 4: Average Sodium Losses in Gastroenteritis

Na+ Status in dehydration	Approiroate Na+ loss in one liter of lost fluid
Acute with isonatraemia	100 mmo/s
Subaute with isonatraemia	80 mmo/s
Hyponatramia	120 mmo/s
Hypernatremia	50 mmo/s

*Widely variable. Of questionable clinical use.

Observe :-

With an acute stormy onset, sodium loss usually severe,as fluid is lost mainly from the extracellular fluid compartment , rich in Na+ with chronic diarrhea however,the intracellular fluid compartment contributes to water loss, and it is sodium poor.

Table 5: Maintenance fluid requirement the holiday-Segar formula (6)

Body weight	Daily water requirements
3-10 KG	100 ml/kg
11-20 KG	1000 ml + 50 ml/kg Above 10
21-30 KG	1500 ml + 50 ml/kg Above 20

-the same figures are true for caloric requirement.

-infants under 3 months need 125-150 mls/kg.

Segar and Holliday (6) estimated that for 100 calorie expenditure the body needs 100 mls of water, 60% of which is lost in wrin.

For each 100ml of water required, the body needs 3 mmo/s of Na+ and 2 mmo/s of K+.

One liter of maintenance fluid should contain 30 mmol/l (that is present in 0.18% sakine , As one liter of lost fluid in GE Contains 80-100 mmols /Na+ and as we give maintenance and deficit together then

Should we use isotonic (0.9%) Saline for maintenance?

This was associated with the development of hypernatremia (7) however , the cut-off point was below 136 mmol/l and none of the subjects developed encephalopathy (8). Isotonic saline imposes a Sodium load (9), here in this protocol we Adopted the use of a single formula for maintenance

Rapid Fluid Resuscitation

This has been recently adopted by the WHO; the COC, the American college care medicine (ACCM); high volume rapid fluid resuscitation should be the corner stone for the management of patients with circulatory compromise (3,4,5).

Volume : 40-60 m/s over 2 hour
Solution : 0.9% Salin

-it Applies for ISO ,HYPO, and Hypernatremia this has been found to (3,10) :-
Replenish intravascular fluid volume ;improve renal perfusion , rapid correction of electrolyte Abnormality and decreased morbidity & mortality .

Details of Rehydration

Mild dehydration :-

*aggressive feeding of oral rehydration solution containing 60-90 mmols sodium /l (table 2) the following was the outcome(1):

-90% of children did not require intravenous fluids.

-children with hypo or hypernatremia promptly recovered.

-despite being hypotonic ,oral solution did not cause hyponatraemia .

*an initial bolus of 20ml/kg of 0.9% (isotonic) saline may be needed if there is vomiting .

*in the uncomplicated patient , ondansteron, an anti -emetic may be used as a helpful adjunct to oral rehydration (3).

*Calculate the total amount of fluid needs from tables 3 and 5.

Moderate and severe dehydration

*Rapid fluid resuscitation : 0.9% (isotonic) 40mls (moderate) - 60mls (severe) over 2 hours.

*Calculate the deficit in volume (Table 3).

*Calculate the Maintenance in volume (Table 5).

*Consider ongoing losses (Weight, evaluation).

*Deduce the volume given by rapid fluid resuscitation from the sum of deficit and maintenance .

*Give half of the final amount over the next 8 hours.

*Give the other half over the following 14 hours.

a. in isonatremia

-DW5 0.45% Saline .

-Add 20-25 mmols K+ for one liter (i.e. 40-50 ml of 4% KCL to one liter or 20-25 ml of 4% KCL to 500 m/s.)

b. in asymptotic hyponatremia (Na 130-135)

-as in ISO natraemia .

-Check Na+.an additional bolus of 20ml isotonic Saline per kg may be needed

c.in asymptotic hyponatremia (125-129)

-0.9 % saline Alternating with DW5 . 0.45% Saline or mix equal proportions of the two solutions add KCL 4% as in isonatraemia

d.Symptomatic hyponatremia (NA below 125)

(seizures altered sensorium, neurologic signs)

-use 3-4 mls /kg of 3% saline (contains 500 mmols NA per liter).

-call your senior for repeat.

-aim at raising NA over 125 mmol/l

-then proceed as in asymptomatic hyponatremia.

Please Observe:

*NA levels below 125mmol/l are not common in gastroenteritis keep in mind other condition.

*Hyponatremia may change to isonatraemia after the initial phase of fluid resuscitation (3);the proceed as in isonatremia

Hypernatremic dehydration

Hypernatremia of acute onset leads to cell dehydration due to hyper osmolality of the plasma. Cell dysfunction results brain shrinks and there will be a confusional state.

In hypernatremia of chronic onset, the brain presents itself by breaking large molecules into smaller ones, called" idiogenic osmoles".these osmoles increase the cell osmolality, hence partially protecting the cell from dehydration.

Rapid infusion of hypotonic fluid results in sudden rush of water into brain cells resulting in sudden expansion , which may lead to convulsions and even brain damage (11).

Principles:

1.Circulartory compromise should corrected by rapid fluid resuscitation with isotonic saline .restoration of blood volume is a priority .

2.Improve renal & gastrointestinal perfusion.

3.Slow rehydration over 45 hours .

4.Aim at 0.5 -1 mmol/hour reduction in the serum sodium levels.

Protocol:

*Rapid fluid resuscitation if there is circulatory compromise (20-40 mls/kg of isotonic 0.9% saline over 2 hours)

- calculate deficit (table 3)
- calculate requirements (table 5) for 48 hours
- (Minus) Fluid volume given by rapid fluid resuscitation .

*infuse the estimated volume uniformly over 48 hours.

*when serum sodium is lowered to the isonatraomic range , proceed as in isonatraemia.

Early Alimentation

The American academy of pediatrics defines rehydration .

As (12): - Restoration of skin turgor & weight

-Tolerance to oral intake.

-Recovery of aerteness.

-Self c orrection of electrolyte abnormalities.

Principles

-goal is to quickly return the patient to an age appropriate, unrestricted formula (3)

-Special formula may be used on the suspicion of lactose intolerance or cow-milk Protein allergy

-Diluted milk and juices are to be avoided .

-Oral rehydration solutions Provide only 200 kcal per liter, compared to 700 kcal/l of milk.

-intravenous solution provide only 200 kcal per liter.

-Both oral & intravenous fluids will lead to negative calorie balance if alimentation is not started.

How to 'Prescribe 'Fluids

(1) look to them as drugs .know the composition (tables/end 2)

(2) your doses are in bottles. natnber them Cleary , with time

i.e. i.v.drip bottle (1)

400 cc 0.9% saline (8-10 am)

Rate 200 mls /hour.

i.e. i.v.drip bottle (2) (10 am-3 pm)

500 cc 0.45% saline in DW5

25 cc 4% kcl

Rate 100 mls/hour.

(we describe maintenance in 500 mls bottles (or bags)

(do not exceed 30 cc 4% KCL in a 500 m/bottle in the ward)

(3) Check your patient :-

-after initial rapid fluid resuscitation

-after the first bottle of maintenance

-for the need to repeat electrolytes (unnecessary if the patient is well).

-Persistence of losses.

-for unusual features : abdominal distension ? unwell ? in Pain ? confessional state?

-Ask for food : introduce it .

-can we change to oral

(A) Example : 10 kg , Circulatory Compromise, isonatromia

Prescription	comment
[1] IV Drip bottle 1 (8-10 Am) 0.9% Saline 400 mls Rate 200 mls/hour	Rapid fluid resuscitation (Please see text)
[2] IV Drip bottle 2 (10-4 Pm) 0.45% Saline,DW5 500 mls 4% KCL 25 mls Rate 80 mls/hour	Deficit in this child 1000 mls Maintenance 1000 mls Total 2000 mls Minus bottle 1 1600 mls
[3] IV Drip bottle 3 (4-10 Pm) 0.45% Saline,DW5 500 mls 4% KCL 25 mls Rate 80 mls/hour	Give 800 mls over 8-10 hours The other 800over 12-14 hours
[4] IV Drip bottle 4 (10-8 am) 0.45% Saline,DW5 500 mls 4% KCL 25 mls Rate 50 mls/hour	Re assess After Bottle 1 After Bottle 2 When necessary
[5] Re-evaluate. Continue Rate 50 mls/hour Allow hydrated start His usual fomula	If Oral rehydration is tolerated, discontinue IV fluid .

(B) Example : 10 kg , Circulatory Compromise, isonatromia

Prescription	comment
bottle 1 0.9% Saline 400 mls 200 mls/hour (over 2 hours) bottle 2 0.9% Saline DW5 250 mls	Rapid fluid resuscitation (see text) -this mixture gives : Na+.

0.45% Saline,DW5 250 mls 4% KCL 25 mls Rate 100 mls/hour See (Comment)	115 mmol/l -check Na+ after bottle 2 If above130, proceed as in example(A) -if below 130, proceed as bottle 2 (Or) add bolus of 0.9% saline 20 mls per kg

-if NA below 125 & symptomatic

3-5 mls/kg of 3% Saline

N.B:-

Rise of NA+ Level during rehydration should be less than 2 mmol/hour , preferably 1-1.5 mmol/h, to Avoid central protein myelosis.

(C) Example: 10 kg, Circulatory Compromise, NA+ over 160

Prescription	comment
bottle 1: 0.9% Saline 400 mls 200 mls/hour (over 2 hours)	Rapid fluid resuscitation (see text)
B 2 to B7(10 hours each/Approx) 0.45 % Saline DW5 500 mls 4% KCL 25 mls Rate 50 mls/hour See (Comment)	-Deficit 1000 minus bottle/=600 Maintenance for 48 h= 2000 Total 2500 Approx infuse over 48 hours Check electrolytes And calcium -when NA+ is lower than 145 proceed as with isonatraemia. -hypocalcaemia: add 10 Ml of 10% Calcium glucorate to each Bottle(2).

General Comments

* Any Protocol is As good the physician who us uses it, there is no substitute for Clinical judgment careful follow up.
* it is wise to underestimate than to overestimate volume of fluids. do not exceed 100ml/kg in deficit calculations .

  The total fluid volume should not exceed 200mls/kg.
* for older children use 30 ml/kg , 60 mls/kg, 80 ml/kg as deficit in mild , moderate and severe dehydration, respectively.
* A drowsy child with gastroenteritis is critical:

  Hypernatemia,septicaemia ,perforated appendix.
* Abdominal distension is always pathological in gastroenteritis :-

  Intussusception, perforated appendix, septicemia, hirscbrunges-very high mortality -,late necrotizing , enterocolitis ,milk intolerance, hypokalemia

  Ask yourself , is it really another GE case?

  Before a surgeon does that for you.

* underweight infants have poor tolerance to IV fluids signs of dehydration are exaggerated. He needs calories more than he needs water and sodium.
* overweight infants are misleading: signs of dehydration are easily missed. Fat is inert and should not be accounted for in maintenance calculation.
* An Acute stormy onset should a cause for concern:

-is it really gastroenteritis

-sodium losses are prominent and shock is early.

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Overview of management of
Pediatric Patient with

Acute Head Trauma

A clinical policy for pediatric departments,
MOH, Kuwait

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Head trauma is one of the most common causes of emergency room visits. E.g. annually in the US more than 470,000 visits a year with 35,000 requiring admission.

Intracranial injury is more frequent following falls from a height above three feet (91 cm, or twice the length/height of the individual), involvement in a motor vehicle accident (either as a passenger or a pedestrian) or impact from a high-velocity projectile.

Clinical symptoms of head trauma:

Including a variety of these
Headaches
Dizziness
Vomiting
loss of consciousness
blurred vision
seizures
amnesia.
Younger children: symptoms may not be as clear e.g.lethargy or irritability

Symptoms that are particularly associated with an intracranial injury:
-Prolonged loss of consciousness or impaired level of consciousness
-Disorientation, confusion or amnesia
-Worsening headache
-Repeated vomiting

Glasgow Coma scale is used to classify the severity of head trauma
Minor GCS 14 or 15
Moderate GCS 9 - 13
Severe? 8 - 3
Pediatric Glasgow coma scale: useful in preverbal children.
Score out of 15:(V+M+E)

Verbal V (out of 5)
5 coos or babbles
4 if irritable but consolable
3 if cries to pain
2 for emitting sounds, moans to pain
1 no response

Motor M (out of 6)
6 if moving purposefully
5 if withdraws to touch
4 if withdrawing to pain
3 if flexing to pain
2 if extending to pain
1 no response

Eye opening E (out of 4)
4 if spontaneous
3 if to voice
2 if to pain
1 if none
A regular Glasgow coma score GCS score for those pediatric patients that are verbal.
Score is out of 15

Verbal (out 5)
5 coherent
4 disoriented
3 words
2 sounds
1 none

Motor (out of 6)
6 follows command
5 localizing to pain
4 withdraw to pain
3 extending to pain
2 flexing to pain
1 none

Eye opening (out of 4)
4 spontaneous
3 to voice
2 to pain
1 none

Initial management is stabilizing vitals signs and following the ABCD (airway- breathing -circulation -disability) algorithm

For specific neurological assessment:

Calculate the GCS score

Check pupil size and reactivity to light

Assess muscle tone, reflex and movement of all four limbs

Fontanel fullness for infants

Check scalp for bogginess, areas of depressed skull fracture

Look for signs of basal skull fracture:

Periorbital ecchymosis ('raccoon eyes'),

ecchymosis over the mastoid bone (Battle's sign),

obvious leakage of CSF from the nose or ears,

hemotympanum.

If one or more of these signs is present thenno tube should be placed by the nasal route

Imaging:

1- Skull Xrays

Need not be performed routinely on all patients.

It has been shown to be useful in children less than 2 years old with a large boggy scalp and suspected skull fracture.

2- CT head

-All patients with moderate to severe head traumas require CT heads to rule out intracranial pathology.

-However there is some debate about when to do a CT head for a minor head trauma. It should be based on clinical judgment. Many studies have looked into to it. For e.g.

CATCH Canadian assessment of tomography for children (derived based on a prospective cohort study of 3886 children in 10 centers

A CT head is required for any child with a minor head injury:

i.e.(An injury sustained within the last 24 hrs. associated with witnessed loss of consciousness, definite amnesia, witnessed disorientation, persistent vomiting (more than one episode), persistent irritability in a child younger than 2 years, with a GCS (13-15)

+ plus any one of the following findings:

High risk (need for neurological intervention)

1 Glasgow Coma Scale score <15 at 2 h after injury
2 Suspected open or depressed skull fracture
3 History of worsening headache
4 Irritability on examination

Medium risk (brain injury on CT scan)

5 Any sign of basal skull fracture (eg, hemotympanum, 'raccoon' eyes, otorrhea or rhinorrhea of cerebrospinal fluid, Battle's sign)
6 Large, boggy hematoma of the scalp
7 Dangerous mechanism of injury (eg, motor vehicle collision, fall from a height ?3 feet (?91 cm) or down five stairs, falling from a bicycle without a helmet)

Other serious clinical indicators for need for CT

Seizures at the time of the event

A known coagulation disorder

Physical findings that indicate a need for CT head in a minor head trauma

Focal neurological deficit

Management after initial assessment:

1- Mild head traumas (GCS 14-15)

a- If patient is asymptomatic since initial presentation, neurological exam normal & with a negative CT head they may go home with instructions to the parents (worsening headache, repetitive vomiting, drowsiness, seizures…) to return to the hospital.

b- If patient was symptomatic,neurological exam normal, &with a negative CT head, then observe clinically every 2 hours (4-6hr). If become asymptomatic discharge home with instructions to the parents (worsening headache, repetitive vomiting, drowsiness, seizures…) to return to the hospital.

c- If patient was symptomatic, neurological exam normal, with a negative CT head, then observe clinically every 2 hours and remain so then admit for observation (24-48hr). e.g offer hydration for patients that are vomiting.

d- If patient was symptomatic or asymptomatic with a normal neurological exam, with a positive CT head, then consult Neurosurgery and admit to hospital for at least 24-48hr. A plan to repeat the CT head is based on clinical & radiological findings. Discharge and follow up should be discussed with Neurosurgery.

e- Children under 2 years old should be under higher suspicion for non accidental trauma and may require further workup and observation to rule that out (details out of scope of this policy)

2 - Moderate head trauma:

Ct head.
Admission to an intensive care setting or a highly monitored ward.
Elevate head of bed to 30 degrees
Consult with Neurosurgery (may require an urgent intervention)

3-Severe head trauma
CT head
After intubation for airway management.
Elevate head of bed to 30 degrees
Consult Neurosurgery (may require an urgent intervention)
Dehydrating measures to control ICP:
Give Mannitol (0.5 - 1 gm/kg) to a euvolemic patient with clinical signs of herniation defined as GCS <8 with one of the following:
fixed dilated pupil
posturing
bradycardia and hypertension.

An Option: hypertonics 3% range (0.1-1.0ml/kg/hr) continuous infusion

Pain management & sedation

Avoid hyponatremia
4- Seizures: early trauma seizures within 7 days of the injury can be treated with anti-epileptics (e.g. Phenytoin, Levetiracetam) administration for 7 days.

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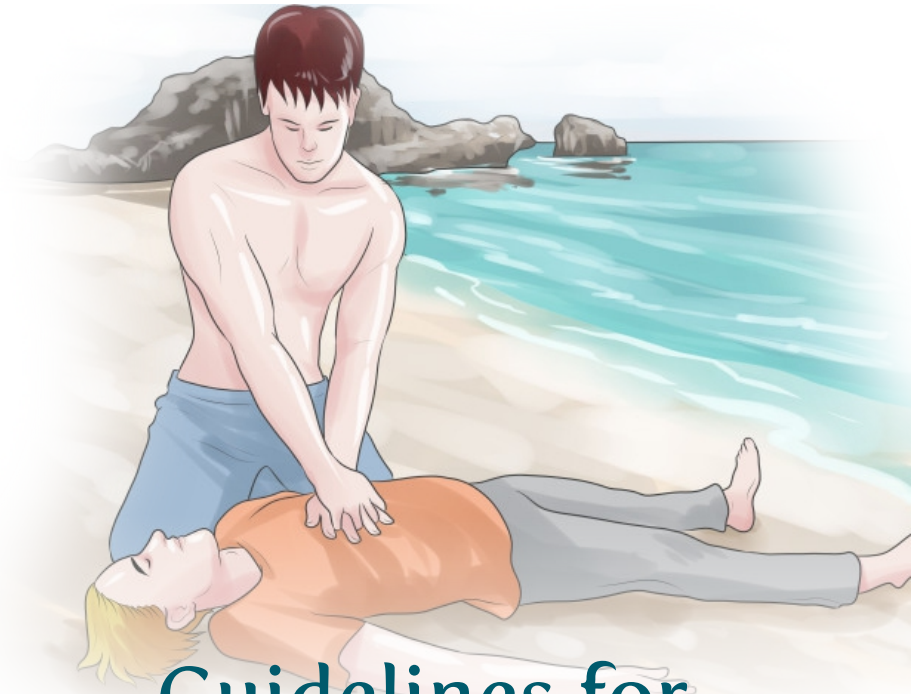

Guidelines for Management of Drowning

From PICU -Al Sabah
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Guidelines for management of Drowning

Definition

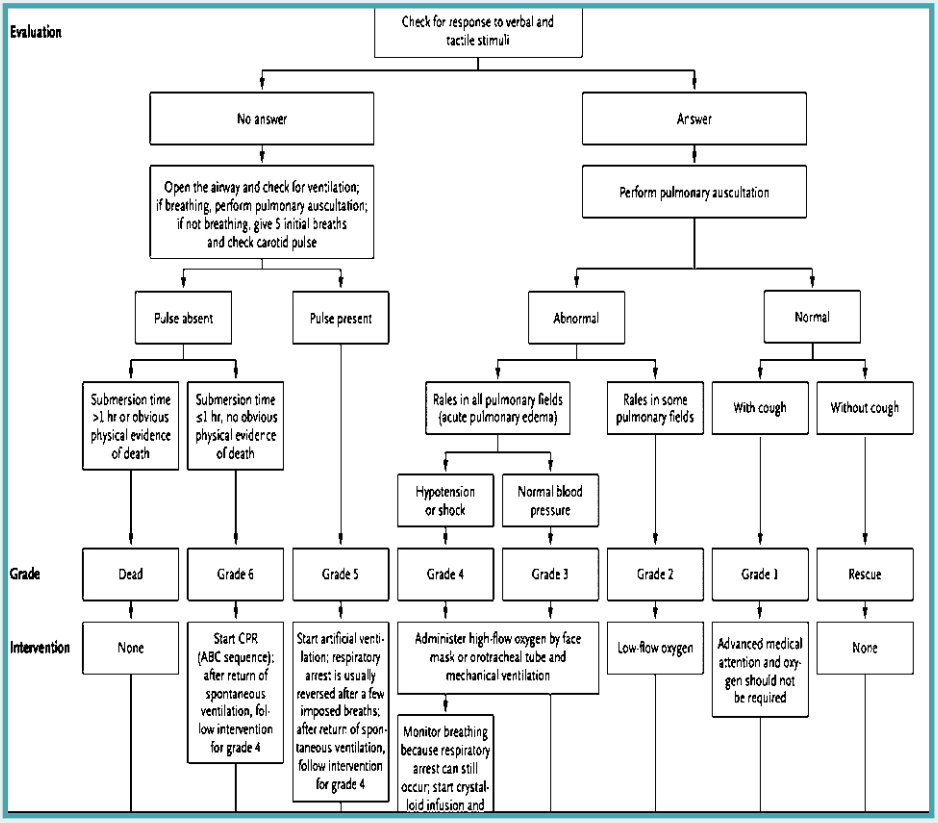
Drowning is the process of experiencing respiratory impairment from submersion/immersion in liquid."

The drowning process begins with respiratory impairment as the person's airway goes below the surface of the liquid (submersion) or water splashes over the face (immersion).

If the person is rescued at any time, the process of drowning is interrupted, which is termed a nonfatal drowning.

If the person dies at any time as a result of drowning, this is termed a fatal drowning.

Any submersion or immersion incident without evidence of respiratory impairment should be considered a water rescue and not a drowning.



Clinical features

Cough, Dyspnoea, Rales,Wheeze,Confusion,Coma, Asystole or pulseless electrical activity

INVESTIGATIONS

CBC,hematocrit ,Electrolytes, RFT,LFT,chest radiography, ABG,ECG

If the person remains unresponsive without an obvious cause, a toxicological investigation and

CT of the head and neck should be considered

MANAGEMENT

- If unresponsive follow BLS algorithm
- Secureairway ,Maintain adequate oxygenation
- Peripheral venous OR Intraosseousaccess
- If hypotension is not corrected by oxygenation, a rapid crystalloid infusion should be administered
- Hospitalization is recommended for all patients with a presentation of grade 2 to 6.
- Persons who have good arterial oxygenation without adjuvant therapy and who have no other associated morbidity can be safely discharged.
- For most patients with a grade 2 presentation, noninvasive oxygen administration results in normalization of clinical status within 6 to 8 hours, and they can then be sent home.
- Patients whose clinical status deteriorates are admitted to an intermediate care unit for prolonged observation.
- Patients with a presentation of grade 3 to 6, who usually need intubation and mechanical ventilation, are admitted to an intensive care unit (ICU).

COMPLICATIONS

- ARDS,
- Pneumonia
- Noncardiogenic pulmonary edema
- Permanent neurologic damage
- Systemic inflammatory response syndrome
- Sepsis and disseminated intravascular coagulation.
- Renal insufficiency or failure is rare but can occur as a result of anoxia, shock, myoglobinuria, or hemoglobinuria

Table 1. Important Facts and Predictors of Outcome in Resuscitation of a Person Who Has Drowned.

Early basic life support and advanced life support improve outcome[21,24,33,54]
During drowning, a reduction of brain temperature by 10°C decreases ATP consumption by approximately 50%, doubling the duration of time that the brain can survive[55]
Duration of submersion and risk of death or severe neurologic impairment after hospital discharge[19,21,24,32]
0–5 min — 10%
6–10 min — 56%
11–25 min — 88%
>25 min — nearly 100%
Signs of brain-stem injury predict death or severe neurologic sequelae[21,24,33,41]
Prognostic factors are important in the counseling of family members and are crucial in informing decisions regarding more aggressive cerebral resuscitation therapies[51]

Table 2. Guidelines for Prevention of Drowning.*

Keep yourself safe
Learn swimming and water-safety survival skills
Always swim with others
Obey all safety signs and warning flags
Never go in the water after drinking alcohol
Avoid inflatable swimming aids, such as "floaters"; know how and when to use a life jacket
Swim in areas with lifeguards
Know the weather and water conditions before going in the water
Always enter shallow or unfamiliar water feet first
Do not overestimate swimming capability[27]
Know how to stay away from rip currents, which are involved in more than 85% of drowning events at the beach[27]
Keep others safe
Help and encourage others, especially children, to learn swimming and water-safety survival skills
Swim in areas with lifeguards
Set rules for water safety
Always provide close and constant attention to children you are supervising in or near water
Know how and when to use a life jacket, especially for children and weak swimmers
Learn first aid and CPR
Learn safe ways of rescuing others without putting yourself in danger
Obey all safety signs and warning flags
Fence in a pool on four sides and install a self-closing, self-latching gate, measures that reduce the incidence of drowning by 50 to 70%[27]
Provide a warning sign for shallow water in a pool[27]

* The first eight messages in each section are from the International Open Water Drowning Prevention Task Force.[56]

Reference (Adopted)
ThDavid Szpilman, M.D., Joost J.L.M. Bierens, M.D., Ph.D., Anthony J. Handley, M.D., and James P. Orlowski,M.D.N Engl J Med 2012; 366:2102-2110May 31, 2012DOI: 10.1056/NEJMra1013317

Smoke Inhalation

Dr. Sujesh Chandran
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Introduction

Smoke is a mixture of heated particles and gases. Smoke inhalation is present in 2-30% of all flame burns presentations and in higher proportions if facial burns are present.

Inhalation injury may describe pulmonary trauma caused by inhalation of thermal or chemical irritants.

Smoke inhalation injuries are divided into three classes:

1) Heat injury which is restricted to upper airway structures
2) Local chemical irritation throughout the respiratory tract
3) Intoxication related with inhalation of carbon monoxide or cyanide

Type	Inhalant	Source	Injury/Mechanism
Irritant gases	Ammonia		Upper airway epithelial damage
	Chlorine		Lower airway epithelial damage
	Sulfur dioxide		Upper airway epithelial damage
	Nitrogen dioxide		Terminal airway epithelial damage
Asphyxiants (mitochondrial toxins)	Carbon monoxide		Competes for oxygen sites on hemoglobin, myoglobin, heme-containing intracellular proteins
	Hydrogen cyanide		Tissue asphyxiation by inhibiting intracellular cytochrome oxidase activity, inhibits ATP production, leads to cellular anoxia
	Hydrogen sulfide		Similar to cyanide, tissue asphyxiant by inhibition of cytochrome oxidase, leads to disruption of electron transport chain, results in anaerobic metabolism
Systemic toxins	Hydrocarbons		CNS narcosis, anesthetic stats, diffuse GI symptoms, peripheral neuropathy with weakness, coma, sudden death, chemical pneumonitis, CNS abnormalities, GI irritation, cardiomyopathy, renal toxicity
	Organophosphates		Blocks acetyl cholinesterase; cholinergic crisis with increased acetylcholine
	Metal fumes		Flulike symptoms, fever, myalgia, weakness.

Clinical features

- Cough and hyper secretion, Facial burns, Redness in eyes, Soot in the nostrils, Singed nasal hair, carbonaceous sputum, Skin color from pale to bluish to cherry red, Hoarseness, Dyspnea, Wheezing, Rales.
- Chemical asphyxiants and low levels of oxygen can lead to mental status changes.
- Headache, nausea, confusion and vomiting are symptoms of carbon monoxide poisoning.
- Confusion, fainting, seizures, and coma are all potential complications following smoke inhalation.

Investigations

- Pulse oximetry-Low saturation occurs with hypoxemia. Readings are falsely elevated by CO-bound hemoglobin and falsely decreased in methaemoglobinaemia.CO-oximetery can detect carboxy-hemoglobin and methemoglobin as well as hemoglobin and oxyhemoglobin.
- ABG, CBC, RFT, LFT, CK, Urine Myoglobin, ECG
- Chest radiography - Hyperinflation, ARDS, Radiographic evidence of pulmonary injury typically does not appear until 24-36 hours after the inhalation.
- Direct Laryngoscopy and Fiberoptic bronchoscopy

Carboxyhemoglobin level

0-10% - Usually no symptoms

10-20% - Mild headache, atypical dyspnea

20-30% - Throbbing headache, impaired concentration

30-40% - Severe headache, impaired thinking

40-50% - Confusion, lethargy, syncope

50-60% - Respiratory failure, seizures

>70% - Coma, death

Cyanide levels

Findings indicative of cyanide intoxication include the following :

- Persistent neurologic dysfunction unresponsive to use of supplemental oxygen
- Cardiac dysfunction
- Severe lactic acidosis, particularly in the presence of high mixed venous oxygen saturation
- "Arterialization" of the venous blood gas, with PO2 values similar to arterial levels due to lack of oxygen utilization by tissues.

Treatment

- Airway and Neurological assessment
- Provide 100% oxygen

- IV/IO access
- Attach pulse oximeter and cardiac monitor

Indications for admission

- Exposure in a closed space for more than 10 minutes.
- Thick, black sputum.
- PaO2 below 8 kPa (60 mm Hg)
- Metabolic acidosis.
- Carboxyhemoglobin above 15%.
- Arteriovenous oxygen difference (on 100% oxygen) greater than 13.33 kPa (100 mm Hg).
- Bronchospasm.
- Burns to the face

Medications

1. Aerosolized medications

- Nebulized B2 agonists-Albuterol
- Racemic epinephrine
- Nebulized Corticosteroids
- Nebulized Acetylcysteine

2. Analgesics

Specific therapy

1. **Carbon monoxide toxicity**

Hyperbaric oxygen therapy is indicated when

- Base excess lower than -2 mmol/L
- CO level greater than 25%
- Signs of cerebellar dysfunction
- Cardiovascular dysfunction
- Pulmonary edema
- Extremes of age

2. Cyanide Poisoning

- Hydroxocobalamin (Cyanokit)
- CN antidote kit containing amyl and sodium nitrite
- Sodium thiosulfate,

3. Methemoglobinemia

Methemoglobinemia in smoke inhalation is relatively rare and rarely requires treatment with methylene blue. Indications for treatment with methylene blue as follows:

- Alteration in mental status
- Acidosis
- Electrocardiographic (ECG) changes
- Ischemic cardiac symptoms

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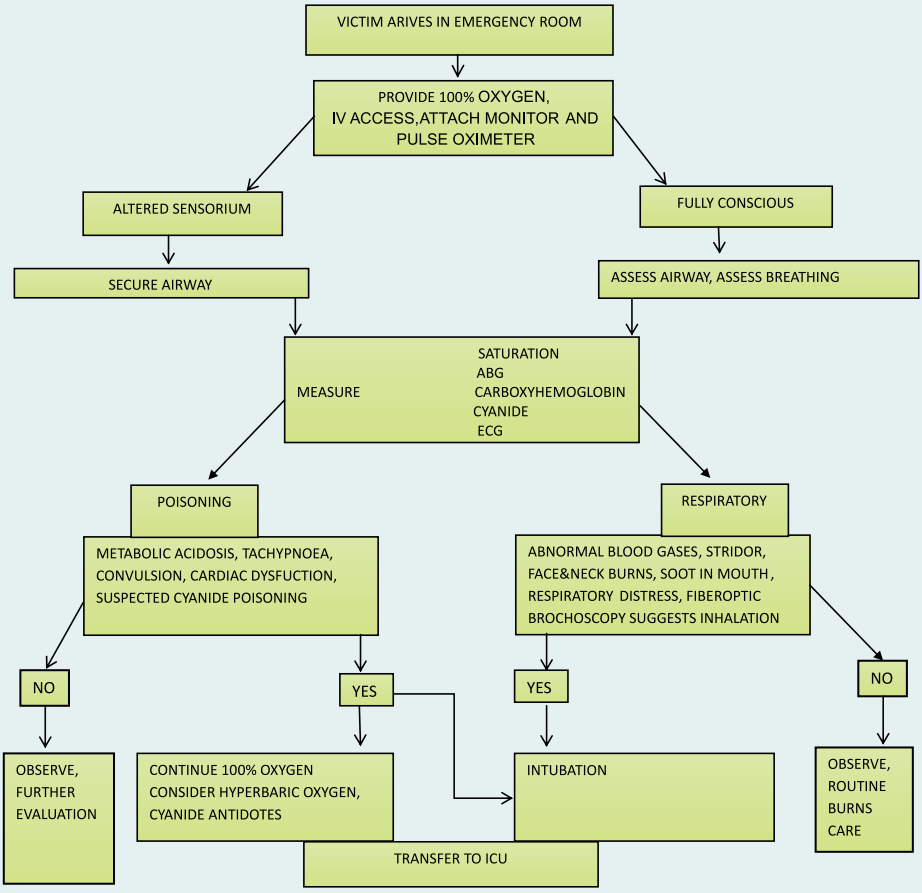

Algorithm for emergency management of smoke inhalation

Toxicology Guidelines

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Acute Poisoning

Initial Management Guidelines

General Principles

Assessment

- Type of ingestion (drug, preparation)
- Time of incident
- Amount of ingestion (include all medication that was potentially in the bottle or packet when calculating)
- Weight of child
- Is the ingestion potentially harmful?
- Beware of the possibility of
  - mixed overdose
  - Beware of the possibility of inaccurate dose reporting on history taking
- If mixed or undetermined ingestion Paracetamol level should be done.

Management

Airway

Breathing

Circulation

Removal of poison (if necessary)

Emesis

- No role in the hospital setting

Activated Charcoal

The treatment of choice for most ingestions.

Most effective when given within first hour.

Contraindications:

Patients with altered conscious state

The following agents:

Ethanol/glycols

Alkalis

Boric acid

Lithium

Iron compounds

Potassium and other metallic ions

Fluoride

Cyanide

Hydrocarbons

Mineral acids

Whole Bowel Irrigation has a limited role in treatment of some slow release preparations

Gastric Lavage has a very limited role in treatment and should not be used without consultation.

Specific antidotes may be available and serum drug levels may help in treatment decisions

All acts of deliberate self harm must be taken extremely seriously.

All intentional self poisonings in adolescents require admission under the adolescent unit after discussion with their on-call consultant.

If unexplained symptoms exist a urinary drug screen may be indicated.

ALKALIS POISONING

Alkalis include: Drain cleaners, Oven cleaners, Automatic dish washing liquids & powders Laundry detergents, Ammonia, Portland cement.

- pH of >11.5 is likely to cause significant GI ulceration.
- Attempt to obtain container to check contents and strength of substance.
- Corrosive potential varies with concentration of specific ingredients and preparations, ie liquid preparations are more likely to cause esophageal burns than powders.
- Check preparations with Poisons Information Centre to determine whether ingested substance is weak, strong, irritant or corrosive in nature.

Assessment

Toxicity

- Exposure may lead to severe burns of GIT, especially esophagus.
- Absence of mouth or pharyngeal ulcers does not preclude gastro-esophageal lesions.

Symptoms: May be minimal Pain.
Nausea & vomiting, drooling or refusing to eat and drink.
Stridor, respiratory distress.

Management

See also General Management of Acute Poisoning Guideline

- ~ ABCD
- ~ Activated charcoal is contraindicated
- ~ If asymptomatic treat with fluid dilution: 10ml/kg of water (max 250ml).
- ~ If asymptomatic after 4 hours and able to eat and drink the patient can be safely discharged home.
- ~ If any symptoms, contact the gastroenterologist, & admit for esophagoscopy.

ANTICONVULSANT POISONING

CARBAMAZEPINE, PHENYTOIN, SODIUM VALPROATE, PHENOBARBITONE.

Assessment

- ~ CNS: Ataxia, drowsiness, coma, convulsions
- ~ GIT: Nausea & Vomiting
- ~ CVS: Hypotension, Arrhythmias

Drug levels are available for some anticonvulsants eg carbamazepine, phenytoin, phenobarbitone

Patients Requiring Treatment

- ~ All symptomatic patients
- ~ Acute ingestion of unknown quantity
- ~ Carbamazepine ingestion of >20mg/kg (for patients not on maintenance treatment) or the greater of more than twice the daily dose or 20mg/kg for patients on maintenance treatment

Management

See also General Management of Acute Poisoning Guideline

- ~ **ABCD**
- ~ **Charcoal 1g/kg** unless altered conscious state (protect airway first)
- ~ **Mild symptoms** (ataxia, blurred vision) observe 4 hours, discharge if symptom free
- ~ **Moderate or persistent symptoms** (after 4 hours of observation)

  Admit for observation, contact the pediatric specialist.
- ~ **Severe symptoms**

  Depressed conscious state or cardiac arrhythmias contact I.C.U.

Antihistamine poisoning

Including Antihistaminics, Decongestant preparations / Sympathomimetic Agents

Examples

- **Cough and cold preparations:**
  - Check formulation of drug - often a combination of substances
  - May contain paracetamol - calculate amount/kg ingested, and proceed as for paracetamol overdose
- **Psychostimulants:**
  - Dexamphetamine
  - Methylphenidate
- **Recreational drugs:**
  - Amphetamine
  - Cocaine
  - Ecstacy

Assessment

Symptoms:

- **Antihistamine**
  - drowsiness, restlessness, delirium
  - convulsion, coma
  - anticholinergic syndrome

- **Sympathomimetic**
  - tachycardia, palpitations, chest pain
  - hypertension, arrhythmias
  - tremor, agitation, hallucinations
  - convulsions

(Beware; **Ephedrine** may cause hypertensive crisis and cerebral hemorrhage)

Management

See also General Management of Acute Poisoning Guideline

Beware; slow release preparations:

- Onset of symptoms may be delayed many hours and patient may require repeated doses of activated charcoal

- **All patients ingesting =3 times the normal daily dose of slow release preparations should be admitted.**

a. Asymptomatic

- o Does not require treatment if dose ingested is < 3 times the normal daily dose.
- o If > 3 times the normal daily dose, or an unknown amount, give Charcoal 1g/kg (if ingestion within last 1 hours and for all slow release preparations).
- o Observe 6 hours.

b. Symptomatic

- o Immediate medical assessment.
- o Charcoal 1g/kg (if ingestion within last 1 hours and for all slow release preparations) unless altered conscious state.
- o Admit for observation.
- o If any cardiac arrhythmias, severe hypertension, altered conscious state, or convulsions contact I.C.U.

Benzodiazepine Poisoning

Often taken as part of mixed overdose in adolescents.

Assessment

Symptoms

- ~ CNS depression, drowsiness, coma
- ~ Respiratory depression
- ~ Hypotension

Beware; additive toxicity with other CNS & Respiratory depressants

Patients Requiring Observation

- ~ Ingestion of .3 times recommended dose for age.
- ~ All symptomatic patients.
- ~ Ingestion of unknown quantity.

Management

See also General Management of Acute Poisoning Guideline

- ~ ABC.
- ~ Charcoal is not usually of benefit.
- ~ If depressed state of consciousness, protect airway and contact ICU.
- ~ Antidote: Flumazenil, not indicated for ingestions and should only be used after discussion with consultant staff.

Camphor Poisoning

Common substances containing camphor:

Vicks VapoRub
Vicks Inhaler

Highly toxic.
Readily absorbed from skin but most toxic exposure is from ingestion.

Assessment

Symptoms

- Often a characteristic odour is present
- Neurologic complications are common acute seizures usually occur within 90 minutes of ingestion may be preceded by muscle fasciculation, confusion, restlessness
- Respiratory depression is common, often following seizure
- GIT involvement: oral/epigastric burning, nausea, vomiting

Management

See also General Management of Acute Poisoning Guideline

- Decontaminate: wash contaminated areas of skin and remove patient¡¦s clothes
- If ingestion within 1 hour, **Activated charcoal 1g/kg** unless conscious state depressed
- **If asymptomatic;**
  Observe for 4 hours & discharge if no symptoms develop. If symptoms develop treat as below.
- **If symptomatic;**
  Needs urgent medical assessment for airway control prior to charcoal administration
  Contact I.C.U.

Ethanol Poisoning

Ethanol Containing Preparations

- Light beer 2%
- Beer 5%
- Cider 5%
- Wine 10%
- Wine coolers 5%
- Spirits 45%
- Liqueurs 30%
- Perfumes& colognes >60%
- Aftershaves 80%
- Mouth washes (some) 25%

Assessment

Symptoms

~ Nausea, vomiting, abdominal pain
~ Hypoglycemia
~ Ataxia, lethargy, coma, convulsions
~ Respiratory depression
~ Hypothermia
~ Hypokalemia, metabolic acidosis

Unexplained drowsiness, hypothermia or hypoglycemia in adolescents may be ethanol induced. In adolescents ethanol ingestion often accompanies ingestion of other drugs.

Patients Requiring Treatment

~ Symptomatic patients

Management

See also General Management of Acute Poisoning Guideline

~ Charcoal is of no benefit
~ Check blood glucose in younger children

a. Asymptomatic or Mild Symptoms (mild incoordination)
   - o Observe for 2 hours
   - o Give frequent carbohydrate containing drinks
   - o Breath alcohol if possible
   - o If remains symptomatic or symptoms worsen admit

b. Symptomatic (more than mild symptoms or continued symptoms after 2 hours)
   - o Blood ethanol measurement, U& E, Glucose
   - o I.V. fluid
   - o Temperature regulation
   - o Admit.
   - o If unconscious or convulsions contact I.C.U.

Hydrocarbon Poisoning

Hydrocarbons Include:	Petrol	Lubricating Oil
	Kerosene	Furniture Polishes
	Lighter Fluid	2 Stroke Fuel
	Mineral Turpentine	Dielsel Fuel
	Paraffin Oil	White Spirit

Assessment

Main complication is Aspiration Pneumonitis
C.N.S. toxicity can be evident (either depression or excitement)

Symptoms:
coughing, choking, respiratory distress ataxia, drowsiness, coma, convulsions persistent burping (particularly seen after petrol ingestion)

Management

See also General Management of Acute Poisoning Guideline
Keep nil orally charcoal is contraindicated

a. Asymptomatic

- o Observe 6hours
- o Discharge if remains asymptomatic
- o Arrange review by LMO the following day

b. Symptomatic

- o If develops respiratory symptoms (aspiration), do CXR & O2 saturation.
- o Give O2 to maintain saturation > 94%
- o If stable, admit to general medical ward
- o If increasing O2 requirements or increased respiratory distress contact I.C.U.
- o If altered conscious state at any time contact I.C.U.

Iron Poisoning

Important ingestion is the amount of elemental iron not the iron salt

Patients Requiring Treatment

- ~ Ingestion of . 20 mg/kg elemental iron.
- ~ Ingestion of an unknown quantity.
- ~ Any symptomatic patients

Assessment

Symptoms

- ~ Initial symptoms are usually:
  nausea, vomiting, hematemesis, diarrhea
- ~ Fever is common

~ Other Symptoms are related to fluid shifts from intravascular to extravascular compartments and cellular hypoxia:
  - tachycardia, vasoconstriction, hypotension, shock
  - Metabolic Acidosis can occur
~ **Beware of the pale child.**

Investigations

~ **Asymptomatic patients:**
  o If tablet ingestion do AXR, if clear and asymptomatic for 8 hours no tests are needed.
  o If >60mg/kg ingested need serum iron levels (see below)

~ All symptomatic patients should have the following investigations:
  o ABG/CBG
  o Glucose
  o serum iron (serial levels every 4 hours)
  o FBE
  o U&E & Cr
  o X-match and clotting profile performed.

~ AXR is helpful in evaluating gastrointestinal decontamination after treatment if tablets have been ingested.

Management

See also General Management of Acute Poisoning Guideline

~ Charcoal is of no benefit.
~ Decontamination of choice is whole bowel irrigation with colonic lavage solution 30 ml/kg/hr until rectal effluent clear (only if bowel sounds present).
~ Chelating agent available Desferrioxamine (Desferrioxamine-iron complex usually turns urine orange/red) .
~ Supportive therapy to maintain adequate blood pressure and electrolyte balance is essential

a. Asymptomatic

o If tablet ingestion do AXR,
o If > 60 mg/kg ingested need serum iron levels 4 hourly until falling.
o If AXR reveals tablets, or capsules ingested, whole bowel irrigation with colonic lavage solution 30 ml/kg/hr until rectal effluent clear.
o Observe until 8 hrs post ingestion if asymptomatic discharge, if symptomatic treat as below.

b. Symptomatic

- o Whole bowel irrigation with colonic lavage solution 30 ml/kg/hr until rectal effluent clear
- o Investigations as above.
- o I.V. fluid resuscitation and potassium and glucose administration as necessary.
- o If altered conscious state, shock, severe acidosis (pH < 7.1), or worsening symptoms commence Desferrioxamine 15 mg/kg/hr I.V.

A Summary of Patients Requiring Chelation Therapy

1. Any patient with symptoms of altered conscious state, hypotension, tachycardia or tachypnea, or worsening symptoms irrespective of ingested dose or serum iron level.
2. Consider desferrioxamine in patients with symptoms who have serum iron levels > 60 micromol/l

All patients being considered for Desferrioxamine or with worsening symptoms

must be referred to ICU.

Paracetamol Poisoning

Patients Requiring Treatment (administration of charcoal)

- ~ Acute ingestion of 150 mg/kg or more
- ~ Ingestion of unknown quantity
- ~ Ingestion of 100mg/kg or more if known liver disease, anorexia, alcohol abuse, CF, or on anticonvulsant or barbiturate therapy, or recent high intake of paracetamol.

Management

See also General Management of Acute Poisoning Guideline

- ~ **Activated charco**al 1g/kg immediately if less than 1 hour since ingestion of tablets or capsules. (Not useful for liquid ingestions as fully absorbed within 20-30 mins).
- ~ Serum paracetamol level at (or as soon as possible after) 4 hours post ingestion will determine the need for N- acetyl cysteine administration (see nomogram below)
- ~ There is nothing to be gained by measuring serum paracetamol before 4 hours
- ~ N-acetyl cysteine treatment should not be started unless the nomogram indicates a potentially toxic paracetamol level
- ~ If N-acetyl cysteine treatment is required, do APTT/INR and baseline LFT's upon insertion of IV.

~N-Acetyl cysteine (see chart)

o Loading dose 150mg/kg in N/2 saline and 5% dextrose (10mls/kg) IV over 1hr.

o Infusion 10mg/kg/hr in N/2 Saline and 5% Dextrose (at half maintenance rate) for 20 hrs, longer if >10 hrs post ingestion or encephalopathic.

~ Monitor hydration and treat as indicated.

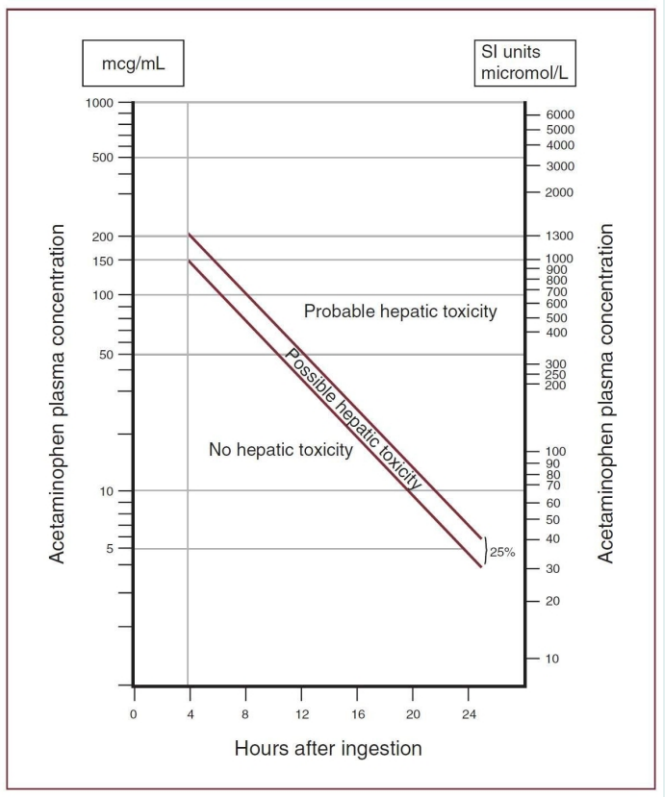
Note

~ Anaphylactoid reactions to N-Acetyl cysteine may occur (wheeze, rash): stop the infusion for 30 minutes & give promethazine (phenergan) 0.2 mg/kg i.v. then recommence infusion at half the previous rate. Increase the rate slowly over time until the desired rate is again reached.

Nomogram For Paracetamol Poisoning

Check you are using the correct units micromols/Litre

A level of over 1300 at 4 hours requires treatment (1000 for high risk patients) (for acute single dose ingestions only - multiple doses over time need an individualised approach - seek advice)



N-Acetyl cysteine (NAC) infusion chart

Weight(Kg)	Loading dose NAC (mg) In mls of N/2 saline · 5% dextrose Give over 1 hour	Infusion dose NAC (mg)in 1 litre of N/2 saline +5% Dextrose	Infusion rate (mls per hour) for 20 hours approx half maintenance rate
6	900mg in 60 mls	5000	12
8	1200mg in 80 mls	5300	15
10	1500mg in 100 mls	5000	20
15	2250mg in 150 mls	6000	25
20	3000mg in 200mls	6600	30
25	3750mg in 250 mls	8300	30
30	4500mg in 300 mls	8500	35
35	5250mg in 350 mls	10000	35
40	6000mg in 400 mls	10000	40
45	6750mg in 450mls	11250	40
50	7500mg in 500 mls	11100	45
55	8250mg in 500 mls	12200	45
60	9000mg in 500 mls	12000	50

Salicylates Poisoning

Examples: Aspirin and aspirin containing drug compounds Oil of Wintergreen

Assessment

Symptoms

~ Tinnitus, vomiting, hyperventilation, lethargy, coma, seizures, hyperthermia, dehydration, hypoglycemia, non cardiogenic pulmonary edema

~ Initial respiratory alkalosis (may be transient), followed by paradoxical aciduria (pH <6), then metabolic acidosis & hypokalemia (¡Ó ongoing respiratory alkalosis).

Patients Requiring Treatment

~ Acute ingestion 150 mg/kg

~ All symptomatic patients

~ Ingestion of unknown quantity

Investigations

~ Serum salicylate level at presentation (on patients requiring treatment), and 2 hrly if symptomatic or enteric coated preparation.

~ Urea & electrolytes, creatinine, acid-base, glucose.

Management

See also General Management of Acute Poisoning Guideline

a. Asymptomatic

- o Charcoal 1g/kg (if <1 hour since ingestion unless enteric coated preparation)
- o Observe 6 hours & discharge if still asymptomatic
- o If enteric coated preparations, serial salicylate levels (2 hourly)
- o Admit if levels have not plateaued at 6 hours post ingestion
- o I.V. bicarbonate infusion 1mmol/kg/hr to correct any acidosis (pH <7.3)

b. Symptomatic

- o All symptomatic patients require urgent medical assessment and investigations as above.
- o Charcoal 1g/kg unless altered conscious state (protect airway first)
- o I.V. fluid resuscitation to correct dehydration (use N. Saline)
- o I.V. bicarbonate infusion 1mmol/kg/hr, after initial slow bolus of 2mmol/kg, (keep urine pH >7.5)
- o Potassium replacement as required
- o Worsening symptoms, convulsion, coma, contact I.C.U. for respiratory support ¡Ó hemodialysis .
- o Salicylate level >7mmol/l following an acute poisoning contact I.C.U. for consideration of hemodialysis.

Theophylline Poisoning

Beware, many slow release preparations may cause toxicity after many hours Beware, sudden deterioration may occur with arrhythmias or convulsions

Assessment

~ CNS: Agitation, hyperventilation, headache, convulsions

~ Cardiovascular: Arrhythmias

~ GIT: Nausea & vomiting (may be intractable), thirst, diarrhea

Patients Requiring Treatment

- Acute ingestion of 10mg/kg
- Any ingestion while on maintenance theophylline
- Ingestion of unknown quantity
- All symptomatic patients

Investigations

- **Theophylline levels** should be determined on all patients requiring charcoal
- Serial levels are required at 2 hours then every 2 hours until peak reached or decline demonstrated.
- If slow release preparation has been taken: admit, continue levels at 4 hourly intervals after decline or plateau to ensure detection of secondary peak.
- Seizures are common at levels >330 micromol/L
- Hemoperfusion commonly needed at levels > 550 micromol/L.
- **U&E, Cr and Glucose on all patients.**

Management

See also General Management of Acute Poisoning Guideline

a. Asymptomatic

- Charcoal 1g/kg
- Observe 4 hours. If no symptoms, discharge if not slow release medication.
- If ingestion of slow release preparation, admit for observation and serial drug levels .

b. Symptomatic

- Charcoal 1g/kg initially unless altered conscious state (protect airway first) then 0.5g/kg 4 hourly, and whole bowel irrigation with colonic lavage solution 30ml/kg/hr.
- Cardiac monitoring
- I.V. fluid resuscitation & maintenance of adequate hydration is vital
- If depressed conscious state, arrhythmias or intractable vomiting contact I.C.U. as likely to need intubation
- Severe intoxication may require hemoperfusion
- If agitated, may need sedation with a benzodiazepine or phenobarbitone.

Tricyclic Overdose

Assessment

Symptoms

- **Anticholinergic**
  - Vomiting, blurred vision, ataxia, tachycardia, urinary retention
- Antiadrenergic
  - vasodilatation
- **Sodium Channel blockade**
  - widened QRS (>0.12 ms)
  - QT prolongation
  - reduced cardiac contractility & hypotension
- **CNS Depression**
  - drowsiness, coma, convulsions

Symptomatic patients require urgent medical assessment

Management

See also General Management of Acute Poisoning Guideline

- **Charcoal** 1g/kg unless altered conscious state (protect airway first)
- Require ECG, cardiac monitoring
- **Asymptomatic:** observe for 6 hours post ingestion and discharge if have a normal ECG just prior to discharge
- All **symptomatic** patients should be admitted
- If widened QRS on ECG commence **Sodium Bicarbonate infusion** 1mmol/kg/hr, after initial slow bolus of 2mmol/kg
- If altered conscious state, widened QRS or arrhythmia contact **I.C.U.** & protect airway.

Protocol for the management of Acute Bronchial Asthma

Revised and updated by:

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Protocol for the management of Acute Bronchial Asthma

These are quick practical guidelines written to aid the pediatricians when managing an acute exacerbation of bronchial asthma in hospital setting.

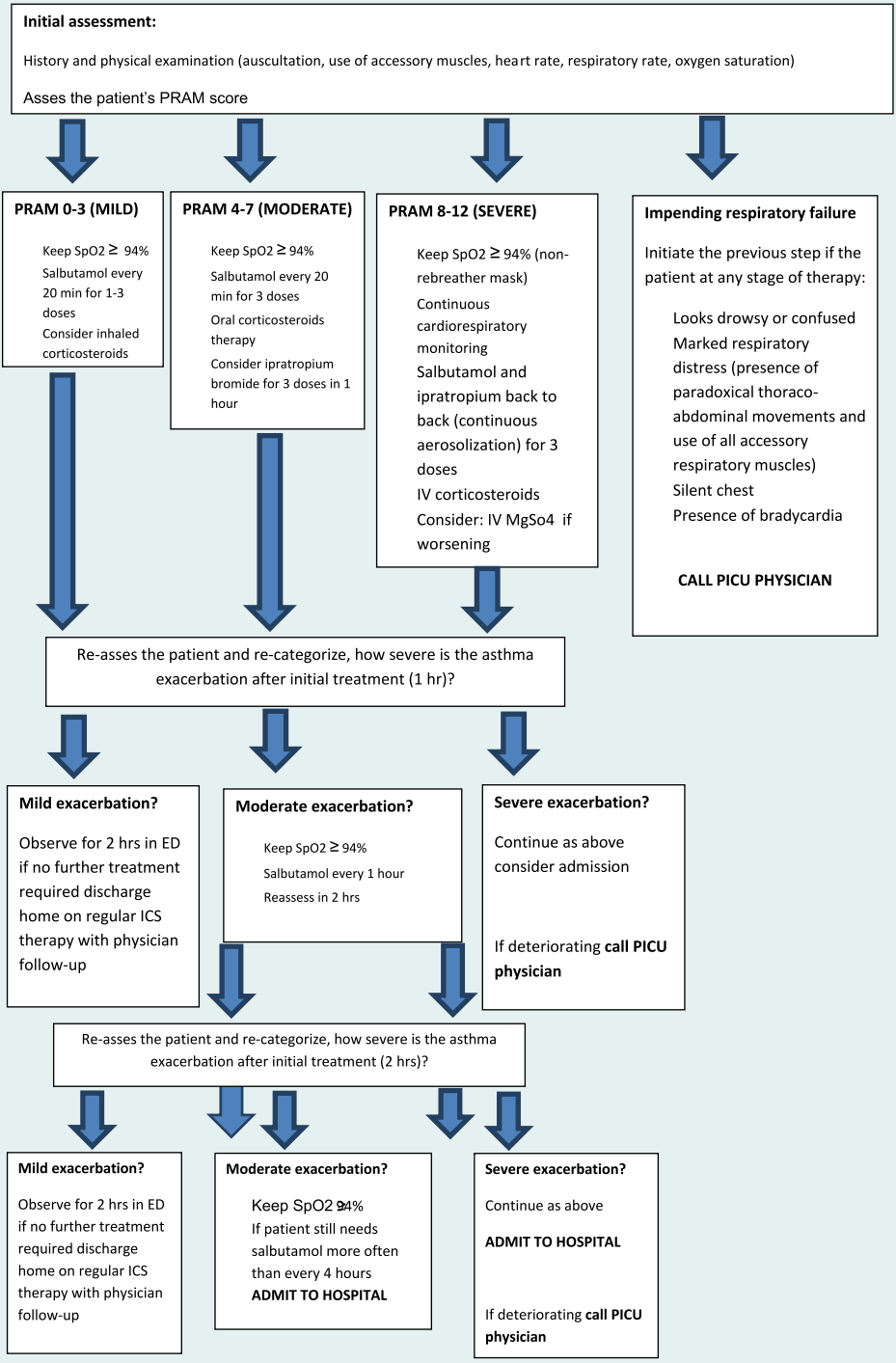
1. The Pediatric Respiratory Assessment Measure (PRAM SCORE): this tool is validated to asses acute asthma severity from toddlers to teenagers (2-17 years)

Sign	0	1	2	3
Suprasternal retractions	Absent	Absent	Present	Present
Scalene muscle contraction	Absent	Absent	Present	Present
Air entry*	Normal	Decreased at the bases	Widespread decrease	Absent/minimal
Wheezing	Absent	Expiratory only	Inspiratory and expiratory	Audible without stethoscope/silent chest with minimal air entry
O2 saturation	≥95%	92-94%	< 92%	<92%

* In case of asymmetry, rate the worst side.

~ A PRAM score from 0-3 indicate a mild attack
~ A PRAM score from 4-7 indicate a moderate attack
~ A PRAM score from 8-12 indicate a severe attack

2. Management of an asthma exacerbation in hospital:



3. Ancillary tests are not routinely recommended.
   a. **Chest x-rays** are very rarely indicated unless the clinician suspects complications (ie, pneumothorax), bacterial pneumonia, the presence of a foreign body, or in cases that fail to improve with maximized conventional treatment. In the absence of suggestive clinical features, there is a documented risk of over diagnosis of pneumonia
   b. **Blood gases** are not routinely required to treat a child with asthma exacerbation unless the patient has no clinical improvement with maximal aggressive therapy. A normal capillary carbon dioxide level despite persistent respiratory distress is a sign of impending respiratory failure.
4. Admission should be considered at any one of the following apply::
   - ~ A nongoing need for supplemental oxygen
   - ~ Persistently increased work of breathing
   - ~ s2-agonists are needed more often than q4 h after 4 to 8 h of conventional treatment
   - ~ The patient deteriorates while on systemic steroids.
   - ~ Other criteria may also be taken into consideration (eg, distance from home, comorbid conditions such as anaphylaxis).
   - ~ ICU admission should be considered if the patient requires continuous nebulized salbutamol and fails to improve on this therapy.

Risk factors for death from asthma:

ASTHMA HISTORY

- ~ Previous severe exacerbation (e.g intubation or PICU admission for asthma)
- ~ Two or more previous hospitalization for asthma in the past year
- ~ 3 o more ED visits fo asthma in the past year
- ~ Hospitalization or ED visit for asthma in the past month
- ~ Using > 2 canisters of SABA per month
- ~ Difficulty perceiving asthma symptoms or severity of exacerbation
- ~ Other risk factors: lack of written asthma action plan, sensitivity to Alternaria

SOCIAL HISTORY

- ~ Low socioeconomic status or inner-city residence
- ~ Illicit drug use
- ~ Major psychosocial problems

COMORBIDITIES

- ~ Cardiovascular disease
- ~ Other chronic lung disease
- ~ Chronic psychiatric disease

5. Medications and dosages for acute asthma treatment as per disease everity:

Asthma severity	Drug & route	Dose (maximum)	Risks	Comments
Mild	Salbutamol (MDI with spacer)	< 20 kg = 5 puffs (0.1 mg/puff) >20 kg = 10 puffs		Preferable route
	Salbutamol Intermittent nebulization	5 mg of in 2 ml NS		
	Inhaled corticosteroids on discharge			
Moderate	Salbutamol (MDI with spacer)	< 20 kg = 5 puffs (0.1 mg/puff) >20 kg = 10 puffs Every 20 min in the first hour		
	Salbutamol Intermittent nebulization	5 mg of in 2 ml NS every 20 min in the first hour		
	Oral corticosteroids	**Prednisone or Prednisolone** 1 to 2 mg/kg/day (max 60 mg) OR **Dexamethasone** 0.15-0.3 mg/kg/day (max 10 mg)	Prolonged course o frequently repeated courses can be associated with adrenal suppression	Recommend one daily morning dose to decrease risk of adrenal suppression
	Ipratropium bromide (MDI/spacer)	Puffs (20 mcg) every 20 min x 3 doses < 20 kg = 3 puffs > 20 kg = 6 puffs		Use with caution in children with soy allergy
Severe	Salbutamol continuous nebulization	0.3 mg/kg/hr. = 5 mg in 4 ml of NS	Tachycardia, hypokalemia, hyperglycemia	Monitor heart rhythm, glucose and electrolytes
	Ipratropium bromide nebulized	<20 kg = 0.25 mg >20 kg = 0.5 mg Every 20 min for maximum 3 doses		Can be mixed with salbutamol nebs
	IV corticosteroids	**Methylprednisolone:** 1-2 mg/kg/dose (maximum 60 mg every 6 hours) **Hydrocortisone** 5-7 mg/kg/dose (maximum 400 mg every 6 hr)		
Severe/impending respiratory failure	IV Magnesium sulfate	25-50 mg/kg IV bolus over 20 min (max 2 gm)	Hypotension	Consider if patient is not improving
	IV Salbutamol	Load: 7.5 mcg/kg over 2-5 min, followed by 1 mcg/kg/min (maximum 5 mcg/kg/min)	Tachycardia, hypokalemia, hyperglycemia	Monitor heart rhythm, glucose and electrolytes

6. Indications to referring patients with asthma to an asthma specialist (2):
   - ~ Patient has had a life-threatening asthma exacerbation.
   - ~ Patient is not meeting the goals of asthma therapy after 3.6 months of treatment. An earlier referral or consultation is appropriate if the physician concludes that the patient is unresponsive to therapy.
   - ~ Signs and symptoms are atypical, or there are problems in differential diagnosis.
   - ~ Other conditions complicate asthma or its diagnosis (e.g., sinusitis, nasal polyps, aspergillosis, severe rhinitis, VCD, & GERD).
   - ~ Additional diagnostic testing is indicated (e.g., allergy skin testing, rhinoscopy, complete pulmonary function studies, provocative challenge, bronchoscopy).
   - ~ Patient requires additional education and guidance on complications of therapy, problems with adherence, or allergen avoidance.
   - ~ Patient is being considered for immunotherapy.
   - ~ Patient requires step 4 care or higher (step 3 for children 0.4 years of age). Consider referral if patient requires step 3 care (step 2 for children 0.4 years of age).
   - ~ Patient has required more than two bursts of oral corticosteroids in 1 year or has an exacerbation requiring PICU hospitalization.
   - ~ there are difficulties achieving or maintaining control of asthma

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Protocol For Managing Croup In Hospital

Revised and updated by:

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PROTOCOL FOR MANAGING CROUP IN HOSPITAL

Croup occurs most commonly in children between 6 months and 3 years of age, but can also occur in children as young as 3 months and as old as 12 to 15 years of age. It has been reported very rarely in adults.

Croup occurs predominantly during late autumn and winter seasons

Most common causative viruses are: parainfluenza types1-3, followed by Enterovirus, Bocavirus, Influenza A & B, RSV and Rhinovirus

Croup symptoms most commonly occur in the late evening and at night and have an abrupt onset. They include:

- ~ Seal-like barky cough
- ~ Inspiratory stridor
- ~ Hoarseness
- ~ No to moderately high fever

Assessment of the severity of croup (Westley Modified Croup Severity Score):

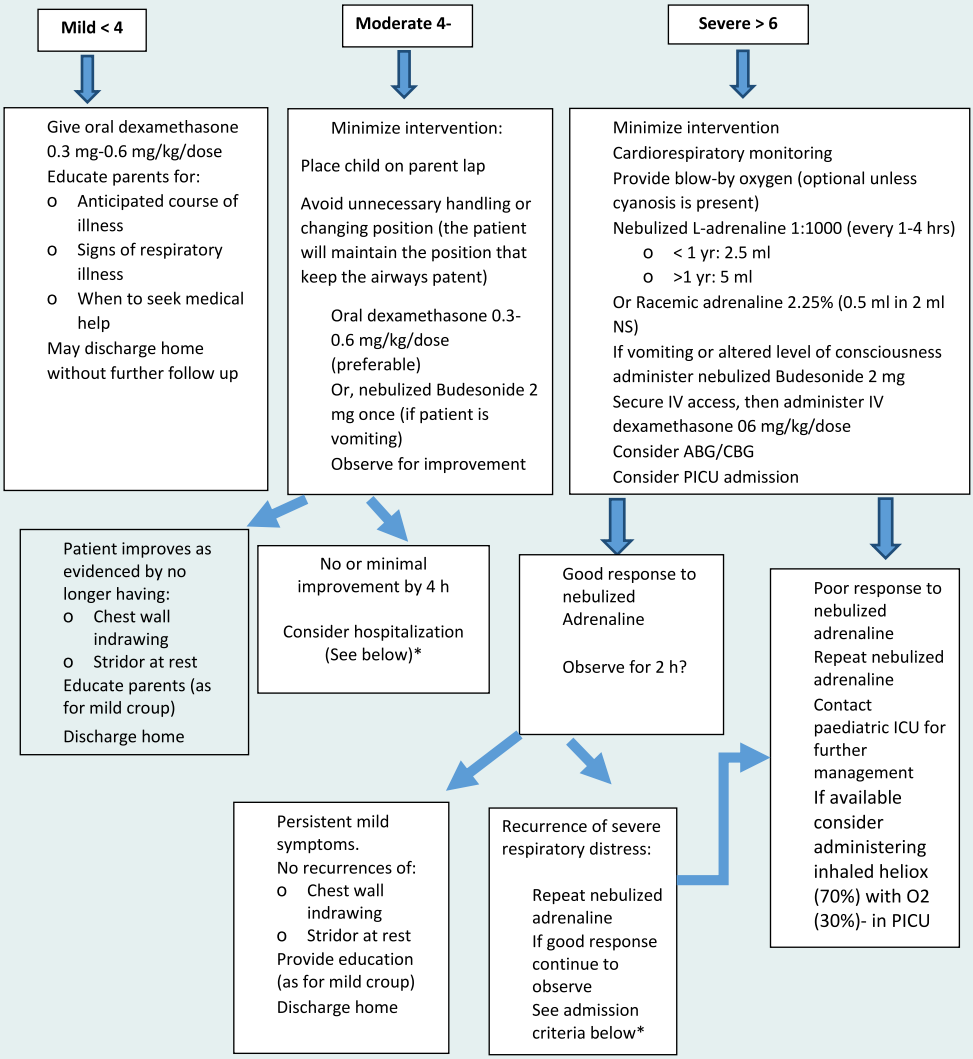
Clinical features	Degree	Score
Stridor	None	0
	With agitation only	1
	At rest	2
Recessions	None	0
	Mild	1
	moderate	2
	Severe	3
Air entry	Normal	0
	Decreased	1
	Severely decreased	2
Cyanosis	None	0
	With agitation	4
	At rest	5
Consciousness level	Normal	0
	Altered	5

Possible score 0-17

Mild croup < 4, moderate 4-6, and severe > 6

PROTOCOL FOR MANAGING CROUP IN HOSPITAL

Algorithm to the approach of a child with croup



PROTOCOL FOR MANAGING CROUP IN HOSPITAL

Consider hospitalization to the general ward if:

- Received steroids > 4 hrs ago and still symptomatic
- Continued respiratory distress(without agitation or lethargy)
  - tridor at rest
  - Chest wall indrawing

Treatment of croup with humidified air is not effective, despite its long history of use.

The majority of children resolve their croup symptoms within 48 hours, but a small proportion of children have symptoms that persist for up to one week

If the child presentation is not classic, other causes of airway obstruction should be considered

Other causes of upper airway obstruction:

Angioedema
Bacterial tracheitis
Epiglottis
Foreign body aspiration
Peritonsillar abscess
Retropharyngeal abscess
Spasmodic croup (recurrent croup)

Factors increasing the likelihood of hospitalisation:

1. History of severe obstruction before presentation
2. History of previous severe croup or known structural airway anomaly (e.g., subglottic stenosis)
3. Age less than 6 months
4. Degree of respiratory distress (stridor at rest = observation or admission)
5. Inadequate fluid intake
6. Parental anxiety
7. Proximity of home to the hospital/transport issues
8. Late evening or night-time presentation
9. Representation to the Emergency Department within 24 h of discharge
10 Poor response to initial treatment
1. 11. Uncertain diagnosis

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Foreign Body (FB) Aspiration In Children

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FOREGN BODY (FB) ASPIRATION IN CHILDREN

BACKGROUND:

Aspiration of foreign bodies results in significant morbidity and mortality in children.

The majority of foreign body aspirations occur in children less than 4 years of age. Immature dentition, poor food control, activity during feeding, and propensity to explore the environment orally are some of the reasons why children are susceptible to foreign body aspiration.

History of witnessed chocking is present in only 75% of cases of foreign body aspiration. 20% of patients with bronchial foreign bodies are totally asymptomatic this is why a high index of suspicion should guide the approach to foreign body aspiration

Table 1. Commonly aspirated foreign bodies
Organic
Food:
Peanuts
Popcorns
Seeds
Hot dogs
Vegetable matter
Non-food:
Bones
Inorganic
Toy parts
Crayons
Pen tops
Tacks
Pins
Nails
Screws
Bullets and casings

Evaluation:

The triad of positive history suggestive clinical signs and imaging are the key to decide on further intervention. The gold standard for diagnosis and FB removal is bronchoscopy.

Positive history suggestive of FB aspiration include: witnessed aspiration, acute chocking or acute coughing episode. High sensitivity when a positive history of FB aspiration exist (90-95%) but low specificity (45-75%).

The 3 most common clinical signs and symptoms reported in literature following a foreign body aspiration are persistent cough, localized decreased breath sounds and localized wheezing. Other signs seen on examination suggestive of FB aspiration include: stridor, localized crackles, and desaturations (usually significant for a large airway obstruction). Clinical signs when exist provide a sensitivity of 96% and a specificity of about 45%.

Table 2. Sign and symptoms of foreign body dislodgment in different locations:

Location	Signs and symptoms
Supra-glottis	Cough, dysphonia, salivation, dyspnea
larynx	Stridor, cough, dysphonia, severe difficulty of breathing
Intra-thoracic trachea	Expiratory wheeze, inspiratory ronchi
Extra-thoracic trachea	Inspiratory stridor, expiratory ronchi
Bronchus	Cough, localized or diffuse wheezing, difficulty of breathing

The primary imaging modality utilized is inspiratory and expiratory chest radiographs looking for air trapping on expiration or unilateral atelectasis. For younger children were inspiratory view are difficult to attain a bilateral decubitus views in addition to an AP films can provide a similar information. Chest x-rays have the sensitivity of 70-88% and a specificity of 45-70% for FB presence.

Table 3. Radiographic findings of aspirated foreign bodies
Normal findings
Air trapping
Mediastinal shift
Atelectasis
Pneumonia
Lobar collapse
Consolidation
Radiopaque foreign body

Complications of bronchial FB:

Significant complication result in FB retained in the bronchial tree for longer than 3 months, those complications can still result if the duration is shorter, further more special concern when the dislodged FB has a hazardous nature (e.g. sharps, batteries, peanuts...etc.)

Atelectasis, pneumonitis, bronchial granulomas, recurrent pneumonias, lung abscess, pneumomediastinum, bronchiectasis, plastic bronchitis, bronchocutaneous or bronchovascular fistulization are among the potential complications of untreated bronchial foreign bodies.

Prior to bronchoscopy era the mortality rate of FB aspiration in children was around 50% now this has reduced to < 1%.

Aim:

The purpose of this guideline is to establish consistency in evaluation of children with bronchial foreign bodies. The aim is to reduce the morbidity associated with delayed diagnosis of these patients.

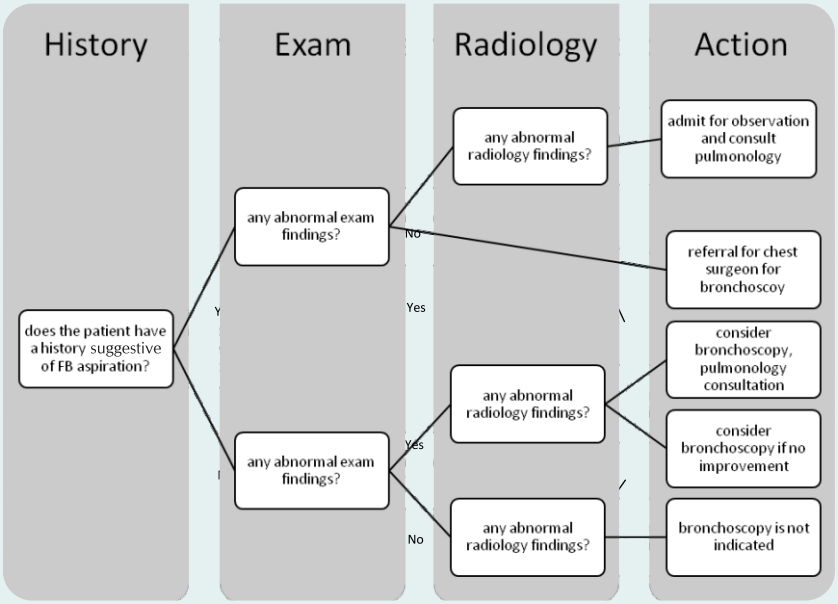
It includes guidelines to manage STABLE children suspected of unilateral foreign body aspiration. It excludes children with upper airway aspirations, children suspected of bilateral bronchial foreign body aspiration and clinically unstable children with decreased level of consciousness, airway compromise, respiratory failure (abnormalities of oxygenation and ventilation), and/or shock.

FOREGN BODY (FB) ASPIRATION IN CHILDREN

Approach to a child with bronchial FB aspiration:

- ~ It is crucial to realize that absence of history and/or normal CXR DOES NOT exclude foreign body aspiration
- ~ Assure stability of the patient (ABCs, cardiorespiratory monitoring)
- ~ Keep NPO and NO CHEST PHYSIOTHERAPY to avoid dislodgment or migration of the FB and compromising the airways
- ~ If the patient has a significant wheezing and/or signs of infection, airway edema or evidence of organic FB aspiration, then bronchodilators, antibiotics and systemic corticosteroids can be used.
- ~ Once stabilized, acquire an inspiratory/expiratory and AP chest x-rays (if young may get a bilateral decubiti views and AP view)
- ~ Please review the following algorithm (Figure 1) when deciding on the next

Figure1. Decision tree in the management of a child with suspected



FOREGN BODY (FB) ASPIRATION IN CHILDREN

Table 4. consider a bronchoscopy if any positive finding in 2 of the following 3 categories:
1. Acute history
• Witnessed aspiration
• Acute choking or coughing episode
2. Examination
• Unilateral diminished breath sounds
• Wheeze
• Stridor
• Crackles
• Increased work of breathing
• Desaturations
3. Radiology
• Radio-opaque FB
• Air trapping or hyperexpansion
• Mediastinal shift
• Atelectasis
• Consolidation

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Bronchiolitis Management in Infants Clinical Practice Guidelines

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Bronchiolitis Management in Infants Clinical Practice Guidelines-

This clinical practice guideline (CPG) has been adapted from the American Academy of Pediatrics (AAP) clinical practice guidelines in the diagnosis and management of bronchiolitis (2014).Not all recommendations from the AAP were adopted for Mubarak Hospital Recommendations that were relevant to the care offered within Mubarak Hospital were either adopted from AAP or adapted with slight modification.

Bronchiolitis an acute inflammatory injury of the bronchioles is the most common cause of lower respiratory tract infection in children aged younger than 12 months. It usually affects children aged younger than 2 years and peaks in infants aged between 3 and 6 months

- A number of viral pathogens can cause bronchiolitis, with respiratory syncytial virus (RSV) being the most common in children aged younger than 2 years and in children hospitalized for bronchiolitis.
- Other virus; HMPV, Rhinovirus, Influenza, adenovirus, and parainfluenza

- Typically bronchiolitis history starts with 2-3 days viral prodrome of fever, cough and rhinorrhea progression to tachypnea, wheeze and varying degree of respiratory distress Severity of symptoms peaks at 5-7 days of illness, symptoms may last for 2 to 3 weeks

Target population:

This CPG is intended primarily for use in children age 1-24 months, who are otherwise healthy and presenting with ronchiolitis typical in presentation and clinical course.

Exclusion: This CPG is Not intended for use in:

- Children admitted to the intensive care unit, requiring assisted ventilation, and/or with sever co-morbid conditions such as;
- Primary Immunodeficiency and/or acquired immunodeficiency; HIV, Recipients of solid organ or hematopoietic stem cells transplants
- Underlying chronic respiratory illnesses: chronic lung disease, bronchopulmonary dysplasia, cystic fibrosis, Prematurity (GA <35wk), LBW, congenital airway anomaly
- Neuromuscular disorders
- Hemodynamically significant congenital heart disease: moderate to severe PHN, cyanotic CHD or acyanotic CHD that requires anti-failure medication

~ Diagnosis:

- ~ The diagnosis of bronchiolitis should be made clinically on the basis of history and physical examination
- ~ Clinicians should assess risk factors for sever disease, such as ; prematurity, underlying cardiopulmonary disease or immunodeficiency
- ~ The main goals when making the diagnosis is assess severity of the respiratory distress and to differentiate infant with viral bronchiolitis from those with other disorders such as viral myocarditis, congestive heart failure, mediastinal mass, vascular ring, asthma, foreign body aspiration or pulmonary embolism.

~ Assessment may require serial observation over time to assess clinical condition accurately

~ Clinical assessment should include:

- ~ Mental status (restless/lethargy may indicate hypoxemia or impending respiratory failure
- ~ Hydration status (ability to feed and signs of dehydration)
- ~ Respiratory status(tachypnea/ apnea, retractions, nasal flaring, grunting, wheezing or crackles
- ~ Pulse oximeter: Continuous saturation monitoring may be indicated for high-risk children in the acute phase of illness
- ~ intermittent monitoring or spot checks are appropriate for lower-risk children and patients who are improving clinically

~ Laboratory and radiological tests:

- ~ Chest radiograph (CXR) routine CXR is not recommended ,it should be considered when:

1. the diagnosis of bronchiolitis is unclear, localized finding or cardiac murmur
2. the rate of improvement is not as expected
3 Disease is severe enough to warrant ICU admission or raises other diagnostic possibilities/complications

- ~ Nasopharyngeal swabs for respiratory viruses PCR
- ~ Not routinely recommended unless required for infection control or when patient is on RSV prophylaxis.
- ~ Blood gas : only if concerns about potential respiratory failure
- ~ Complete Blood Count and bacterial cultures are not routinely recommended unless a concomitant bacteremia is suspected

~ Management:

- ~ Bronchiolitis is a self-limiting disease.
- ~ For those requiring admission, supportive care with assisted feeding

and oxygen therapy still forms the mainstay of treatment.

The main benefits of hospitalization of infants with acute bronchiolitis are:

- ~ The careful monitoring of clinical status with frequent reassessment
- ~ Maintenance of adequate hydration and oxygenation
- ~ Parental education(encourage breast feeding for all infants who are able to feed, for those who cannot feed during acute illness, expressed breast milk can be provided to the infant by nasogastric tube

~ The decision to admit to hospital should be based on clinical judgment taking into consideration;

- ~ Infants at risk for progression to severe disease with impending respiratory failure which includes:
- ~ Infants < 3 months at presentation
- ~ Born prematurely at < 35 weeks' gestation
- ~ Immunodeficiency
- ~ Hemodynamically significant congenital heart disease

- ~ the infant's respiratory status (indrawing, nasal flaring, grunting, RR >70/min, marked chest retractions)
- ~ Supplemental O2 requirement (there is lack of evidence to specify a target oxygen saturation by pulse oximetry below which a supplemental oxygen is indicated. Some data suggests, starting supplemental oxygen if the saturation persistently falls below 90% while awake for and below 88% during sleep
- ~ Cyanosis or apnea or bradycardia
- ~ Inability to maintain adequate hydration (dehydration or history of poor fluid intake, lethargy)
- ~ The family's ability to cope

~ Physicians should keep in mind that the disease tends to worsen over the first 72 h when deciding whether to hospitalize.

~ Oxygen:

- ~ Supplemental oxygen therapy is a mainstay of treatment in hospital
- ~ Oxygen should be administered if saturations fall below 90% and used to maintain saturations at ?90%
- ~ oxygen is usually administered via nasal cannula, face mask or a head box
- ~ A recent alternative is humidified high-flow nasal cannula therapy which may be better tolerated and potentially decrease the need for mechanical ventilation

- At this point, there is insufficient evidence to determine effectiveness

~ Hydration:

- Clinicians should administer nasogastric or intravenous fluids for infants who cannot maintain hydration orally
- Infants with a respiratory rate >80 breaths/min, nasal congestion or lethargy may have an increased risk of aspiration
- A recent RCT found nasogastric (NG) and intravenous (IV) routes to be equally effective, with no difference in length of hospital stay.
- Appropriate intravenous maintenance fluids (D5&0.9%NaCl) should be provided to infants who cannot tolerate oral or NG feeds.
- Monitor Na level for risk of hyponatremina with hypotonic fluids.
- The safety of resuming oral feeding should be frequently reassessed to avoid delays in restarting feeds.

Bronchodilators (salbutamol):

When the diagnosis of bronchiolitis is clear, a trial of salbutamol is not currently recommended as was considered in the 2006 AAP bronchiolitis guideline.

- Bronchodilators have not been shown to improve O2 saturation, reduce admission rates or shorten the hospitalization as concluded recently from an updated Cochrane systematic review assessing the impact of bronchodilators in bronchiolitis on oxygen saturation, the primary outcome measure, reported 30 randomized controlled trials involving 1992 infants in 12 countries Salbutamol may be considered as an option, when there is history for allergy, asthma, or atopy

Corticosteroids:

Clinicians should not routinely administer systemic corticosteroids to infants with bronchiolitis

Clear evidence showed that systemic and inhaled corticosteroids alone do not provide significant benefit to children with bronchiolitis.

A tentative Evidence for potential benefit of combined corticosteroid and epinephrine

Epinephrine:

Some studies have shown that epinephrine nebulization may be effective when combined with steroids in reducing hospital admissions. However, the evidence remains insufficient to support routine use of epinephrine in the emergency department.

It may be reasonable to administer a dose of epinephrine and carefully monitor clinical response; unless there is clear evidence of improvement, continued use is not appropriate.

There is insufficient evidence to support its routine use in admitted patients.

Combination Epinephrine and Dexamethasone

One publication from the Pediatric Emergency Research Canada group found

an unexpected synergism between the administrations of nebulized epinephrine with oral dexamethasone.

The combination appeared to result in a reduced hospitalization rate; owever, the results were not significant when adjusted for multiple comparisons.

Pending better definition of its risks and benefits, this combination is not recommended for the therapy of otherwise healthy children with bronchiolitis.

Nasal suctioning

There are insufficient data to make a recommendation about suctioning, but it appears that deep, infrequent suctioning may not be beneficial

Hypertonic Saline:

A Cochrane review of 11 trials found that nebulized hypertonic saline was associated with a reduced length of stay of one day in settings where the admission was longer than three days.

The optimal treatment regimen remains unclear.

The most commonly used regimen in most trials has been 3% saline nebulizer with or without bronchodilator every 8 hours

Nebulized 3% saline may be helpful in the inpatient setting; this treatment appears primarily to benefit patients with a longer length of stay.

Evidence does not currently support its routine use in the outpatient setting

Antibiotics:

- ~ Bacterial infection in otherwise healthy children with bronchiolitis is exceedingly rare
- ~ Antibiotics should not be administered to infants with bronchiolitis unless there is a clear documented a concomitant bacterial infection
- ~ Antibiotic therapy may be justified in some children with bronchiolitis who require intubation and mechanical ventilation for respiratory failure Antivirals:
- ~ Antiviral therapies, such as ribavirin, are expensive, provide limited benefit thus are not recommended for the routine treatment of bronchiolitis in otherwise healthy children.
- ~ In patients with or at risk for particularly severe disease, antivirals could be considered, but this decision should be made on an individual basis in consultation with appropriate subspecialists

Respiratory Care therapy:

Routine respiratory care therapy should not be used as they have not been found to be useful.

These include:

- ~ Chest physiotherapy
- ~ Cool mist therapy
- ~ Aerosol therapy with saline
- ~ Other therapies used for critically ill infants with severe bronchiolitis, such as helium/oxygen, nasal continuous positive airway pressure,

mechanical ventilatory and surfactant, are beyond the scope of thisprotocol

In Hospital Monitoring:

Monitoring should include assessment and documentation of respiratory rate, work of breathing, oxygen saturation, findings on auscultation and general condition, including feeding and hydration status

Thoughtful use of oxygen saturation monitoring in hospitalized patients is recommended.

Continuous saturation monitoring may be indicated for high-risk children in the acute phase of illness, and intermittent monitoring or spot checks (4-6 hr) are appropriate for lower-risk children and patients who are improving clinically.

Discharge Criteria:

Respiratory Status:

Respiratory status is consistently improving

Tachypnea and increased work of breathing are normal/mild

Oxygen saturation is in an acceptable range on room air (~94%)

Nutritional Status:

The patient is on oral feedings sufficient to prevent dehydration

Social Status:

Parent or guardian is competent and confident they can provide care at home

Follow-up:

Instructions of when to follow-up with own primary care provider, advisable within 48-72 hr post discharge

Parent Education and Infection Control measures:

The family should be educated on the following topics regarding prevention and the care of a child with bronchiolitis before discharge:

Basic pathophysiology and expected clinical course of bronchiolitis including lingering symptoms which may continue up to 21 days to disrupt child and family routines

Proper techniques for suctioning the nose and making breathing easier

To return to the hospital or to seek medical advice when the following signs are observed:

Increasing respiratory rate and /or work of breathing as indicated by accessory muscle use

(i.e breathing very fast and/or skin sucking in around the neck or ribs with each breath)

Inability to maintain adequate hydration

(i.e. unable to feed or drink)

Worsening general appearance

(i.e. has new symptoms not present while in the hospital such as

vomiting or fever, looks lethargic or does not respond normally to touch or sound, change in baby's colour)

Importance of hand washing by all caregivers before and after contact with the child to prevent spread of disease.

- Limiting exposure to contagious settings and siblings and wash clothing, toys, and eating utensils between uses by different children
- If hands are not visibly soiled, an alcohol-based rub is preferred according to The Centers for Disease Control and Prevention published recommendations
- Tobacco smoke exposure increases the risk and severity of bronchiolitis
- Respiratory infections were shown to be significantly less common in breastfed children for 6 months, thus the AAP presented a general policy on 2012 to encourage breast feeding

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Management of Heart Failure

Paediatric Cardiology Unit
Chest Hospital

Management of Heart Failure

Common causes of heart failure:

1.Congenital heart disease
   a. Large left to right shunt (VSD, AVSD, PDA)
   b.Obstructive lesions (coarctation, A.S)
   c. Anomalous left coronary artery from the pulmonary artery (ALCAPA)
2.Acute myocarditis.
3.Dilated cardiomyopathy (Familial, metabolic)
4.Restrictive cardiomyopathy

Physical Examination:

- Tachycardia
- Tachypnea with respiratory distress
- Arrhythmia particularly ventricular ectopy
- Weak peripheral pulses and or delayed capillary refill
- Heart sounds are often muffled with or without gallop rhythm
- Murmurs of the original disease
- Jugular venous distension may be observed in older children
- Pulmonary & systemic venous congestion are manifest by rales and hepatomegaly.

Investigations

- ECG: - low amplitude
  - sometimes abnormal axis
  - atrial or ventricular enlargement according to the original disease.
- Chest X-Ray: Cardiomegaly with pulmonary venous congestion.
- 2 D echocardiogram: To check for cardiac anomalies and ventricular function.
- Complete blood count with differential.
- Blood culture and ESR if fever & infection are suspected.
- Creatinine Kinase (CK – MB)
- Viral IgM antibody titres (in suspected viral myocarditis)
- Serum carnitine, Lactate, pyruvate in suspected metabolic or familial cardiomyopathy

General Management

- Maintain **ABC**s, give **oxygen** and connect to a cardiac monitor
- If in shock intubate and ventilate
- Secure an IV line, Do NOT give fluid bolus, consult cardiology
- Keep fluid input/output chart
- Fluid restriction 70% ml/kg/day
- If the baby is tachypnoic consider feeding via NG tube
- Monitor serum electrolytes frequently (specially Potassium)
- Consult a cardiologist

Management of heart failure due to acute myocarditis

Maintain ABCs
Admit to ICU
CVP monitoring

↓

Inotropic support

- **Dobutamine:** 2-20 µg/kg/min (the first drug used initially)

b IIf the patient is **hypotensive** add:

- **Dopamine:** 2-20 µg/kg/min (not used as first line inotropes unless the baby is hypotensive, (it causes tachycardia and subsequently reduced tissue perfusion)

↓

- **Phosphodiesterase inihibitors**

Milrinone - produces inotropic, vasodilator effect and after load reduction.
- best used as infusion in combination with Dobutamine. -
50 µg/kg/dose IV/IO over 10-60 min followed by continuous infusion 0.25-0.75 µg/kg/min

- **Diuretics:** Furosemide 1 – 2 mg/kg IV q6-12h(monitor electrolytes for hypokalemia).

↓

- **Sedation:** Needed in the initial stages in the form of morphine or fentanyl if the baby is ventilated to reduce exhaustion.
- **Anticoagulation:** Used when LVEF is below 20% for the fear of developing mural thrombi in left ventricle. Heparin is used initially.
- Other Drugs can be used according to the cause of heart failure like Immnosuppressive agents, gamma Globulins etc.

↓

After stabilization of the patient (may take few days):
- Improvement of tissue perfusion
- Disappearance of Gallop Rhythm
- Clearance of lung signs

↓

- Reduce IV inotropes gradually
- Start **digoxin** (maintenance): 0.003-0.005 mg/kg/dayPO
- Start **captopril**: (vasodilator/reduction of afterload)

Infant/children: 0.1 mg/kg/dose PO q8hr
titrate (if needed) to maximum 6 mg/kg/day (according to BP)
Adolescents/adults: 12 – 25 mg/dose PO TID
Increase weekly if necessary by 25 mg/dose to maximum 450 mg/day
Contraindicated in left ventricular outflow obstruction, e.g. critical aortic stenosis.

- Change IV furosemide to oral at the same dose and add **Spironolactone** 2 mg/kg/day q12h
- If LVEF is still below 30% give baby aspirin 3-5 mg/kg/day. Sometimes it is important to continue oral anticoagulants as warfarin.

Management of heart failure due to shunt lesions (VSD, PDA, AVSD)

Furosemide: 1 – 2 mg/kg IV/PO q6-12h
and add
Spironolactone: 2 mg/kg/day q12h

↓

Captopril: (vasodilator/reduction of afterload)
Infants/children: 0.1 mg/kg/dose PO q8hr
titrate (if needed) to maximum 6 mg/kg/day according to BP
Adolescents/adults: 12 – 25 mg/dose PO TID
increase (if needed) by 25 mg/dose to maximum 450 mg/day
Contraindicated in left ventricular outflow obstruction, e.g. critical aortic stenosis.

- **Digoxin** can be started in some cases of heart failure in infants
  maintenance dose: 0.01 mg/kg/day q12hr (No digitalization needed)
- When starting digoxin with diuretics (frusemide and spironalactone), the dose given as 0.01 mg/kg/day q12hr
- If captopril is given with digoxin and diuretics, then spironolactone should be reduced or stopped according to potassium level.
- Digoxin toxicity can occur if the above 4 drugs are given and lower doses of digoxin should be given (0.0075 mg/kg/day q12hr)
- It is advisable that antifailure therapy be started by **cardiologist** initially in conditions of large left to right shunt lesions and in obstructive lesions

Management of chronic heart failure
(with dilated cardiomyopathy and left ventricular dysfunction)

- Stable patients should be maintained on ACE inhibitors (e.g. Captopril, enalapril, Zestril) on long term. The doses are adjusted according to BP
- Diuretics are given in some patients as adjunctive therapy when left ventricular ejection fraction is < 40%
- Long acting Beta blockers have proven efficacy in patient with chronic heart failure. The drug used nowadays is Carvedilol 0.1 mg/kg/dose q12hr. increase slowly and monthly by 0.1 mg/kg/dose to 0.3-0.6mg/kg/dose q12 maximum dose of 6.25 mg q12h

References:

1. Treatment of heart failure in children. Current Paediatrics. 2005. 15, 539-548
2. Pediatric Cardiology. Robort Anderson. Second edition, 2002.
3. Pediatric Cardiac Intensive Care. Anthony Chang 1999
4. The Harriet Lane Handbook 2002

Acute Hemolytic Anemia Guideline Category

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Acute Hemolytic Anemia

Guideline Category

Diagnosis
Evaluation
Management
Risk Assessment
Treatment

Definition

Hemolysis is the destruction or removal of red blood cells from the circulation before their normal lifespan of 120 days.

Classification is based on:

- Site of hemolysis
- Intravascular : Red cell fragmentation syndromes, PNH (Paroxysmal Nocturnal Hemoglobinuria) and immediate transfusion reactions.
- Extravascular : RBCs destroyed by the reticuloendothelial system.

~ **Causes :**

Intrinsic (RBC defect) vs. an acquired extrinsic mechanism

~ **Intrinsic defects**: all are hereditary except PNH (Paroxysmal Nocturnal Hemoglobinuria)

- Membrane: Hereditary spherocytosis, hereditary elliptocytosis.
- Enzyme: e.g. G6PD deficiency (Glucose 6 Phosphate Dehydrogenase Deficiency) and Pyruvate Kinase Deficiency .
- Hemoglobinopathy : sickle cell, Thalassemias, Hgb C, Hgb E.
- Paroxysmal Nocturnal Hematuria : complement-mediated lysis. Consider with pancytopenia, unexplained thrombosis (especially intra-abdominal) and an elevated reticulocyte count and LDH.

~ Extrinsic causes

- Hypersplenism
- Antibody-mediated: warm (IgG) vs. cold (IgM)
- Trauma/ Red Cell fragmentation : heart valve, hemodialysis, vasculitis, cardiopulmonary bypass,
- Infection / toxin : malaria and clostridia

Diagnosis

History and Physical Examination

- When the onset of anemia is abrupt, children may present acutely with
  - pallor
  - fatigue
  - exercise intolerance.
- Jaundice and dark urine are often seen, and occasionally hemoglobinuria is present due to the rapidity of red blood cell (RBC) destruction.
- Physical examination findings often include:
- tachycardia with a flow murmur, with or without a gallop
- splenomegaly with or without hepatomegaly.
- With fulminate hemolysis, decreased oxygen-carrying capacity and severe anemia may be life-threatening, and the child may appear acutely ill, with tachycardia, tachypnea, signs of hypoxemia, and even cardiovascular collapse.

Labs

- Send CBC with retics , LDH, total and direct bilirubin, and possibly haptoglobin

Laboratory findings typically include

Normochromic, normocytic anemia with reticulocytosis or rarely reticulocytopenia.

White blood cell count and platelet count are normal or slightly elevated. An indirect hyperbilirubinemia is usually present, but hepatic and renal functions are normal unless the patient has suffered cardiovascular collapse. Haptoglobin level < 25 is 96% specific for hemolysis, but it is an acute phase reactant and a very sensitive test.

Once the diagnosis of hemolysis is made on the basis of laboratory and peripheral smear findings. it is necessary to determine the etiology. While most forms of hemolysis are classified as predominantly intravascular or extravascular.

Acquired Disorders

The age of onset, accompanying clinical presentation, and co-existing medical problems usually guide the clinician to consider either an acquired or a hereditary cause

IMMUNE HEMOLYTIC ANEMIA

Immune hemolytic anemias are mediated by antibodies directed against antigens on the red blood cell surface.

Microspherocytes on a peripheral smear and a positive direct antiglobulin test are the characteristic findings.

Immune hemolytic anemia is classified as autoimmune, alloimmune, or drug-induced, based on the antigen that stimulates antibody - or complement -mediated destruction of red blood cells.

1- AUTOIMMUNE HEMOLYTIC ANEMIA

Autoimmune hemolytic anemia (AIHA) is mediated by autoantibodies and further subdivided according to their maximal binding temperature.

Warm hemolysis refers to IgG autoantibodies, which maximally bind red blood cells at body temperature (37°C [98.6°F]). In cold hemolysis, IgM autoantibodies (cold agglutinins) bind red blood cells at lower temperatures

(0° to 4°C [32° to 39.2°F]).

The direct antiglobulin test (DAT), also known as the direct Coombs' test, demonstrates the presence of antibodies or complement on the surface of red blood cells and is the hallmark of autoimmune hemolysis.

2- ALLOIMMUNE (TRANSFUSION) HEMOLYTIC ANEMIA

The most severe alloimmune hemolysis is an acute transfusion reaction caused by ABO-incompatible red blood cells.

3- DRUGS INDUCED IMMUNE - MEDIATED HEMOLYTIC ANEMIA

Drugs that Cause Immune-Mediated Hemolysis: Streptomycin Fluorouracil, Penicillin , Ampicillin , Methicillin , Carbenicillin, Rifampin, Diclofenac , Interferon alfa , Sulfonamid , Apresoline are some examples.

INFECTION

Numerous mechanisms link infection and hemolysis.

Autoantibody induction (e.g., by M. pneumoniae and antimicrobial drugs (e.g., penicillin) are directly toxic to red blood cells.

Malaria is the classic example of direct red blood cell parasitization.

Plasmodium species, introduced by the Anopheles mosquito, invade red blood cells and initiate a cycle of cell lysis and further parasitization.

Hereditary Disorders

The mature red blood cell, while biochemically complex, is a relatively simple cell that has extruded its nucleus, organelles, and protein-synthesizing machinery. Defects in any of the remaining components-enzymes, membrane, and hemoglobin-can lead to hemolysis.

ENZYMOPATHIES

The most common enzymopathy causing hemolysis is G6PD deficiency (Glucose 6 Phosphate Dehydrogenase Deficiency)

G6PD is a critical enzyme in the production of glutathione, which defends red cell proteins (particularly hemoglobin) against oxidative damage. This X-linked disorder predominantly affects men.

Agents that Precipitate Hemolysis in Patients with G6PD Deficiency

Acetanilid*	Phenylhydrazine
Furazolidone (Furoxone)	Primaquine
Isobutyl nitrite	Sulfacetamide
Methylene blue	Sulfamethoxazole (Gantanol)
Nalidixic acid (NegGram)	Sulfapyridine
Naphthalene	Thiazolesulfone
Niridazole*	Toluidine blue
Nitrofurantoin (Furadantin, Macrobid, Macrodantin)	Trinitrotoluene (TNT)
Phenazopyridine (Pyridium)	Urate oxidase

Most patients have no clinical or laboratory evidence of ongoing hemolysis until an event-infection, drug reaction or ingestion of fava beans-causes oxidative damage to hemoglobin.

The oxidized and denatured hemoglobin cross-links and precipitates intracellularly, forming inclusions that are identified as Heinz bodies on the supravital stain of the peripheral smear. Heinz bodies are removed in the spleen, leaving erythrocytes with a missing section of cytoplasm; these "bite cells" can be seen on the routine blood smear.

Hemolysis occurs two to four days following exposure and varies from an asymptomatic decline in hemoglobin to a marked intravascular hemolysis. Even with ongoing exposure, the hemolysis usually is self-limited, as the older G6PD-deficient cells are destroyed.

G6PD activity levels may be measured as normal during an acute episode, because only nonhemolyzed, younger cells are assayed.

If G6PD deficiency is suspected after a normal activity-level measurement,

the assay should be repeated in two to three months, when cells of all ages are again present.

MEMBRANOPATHIES

Sickle cell anemia is an inherited disorder caused by a point mutation leading to a substitution of valine for glutamic acid in the sixth position of the ? chain of hemoglobin. Membrane abnormalities from sickling and oxidative damage caused by hemoglobin S, along with impaired

deformability of sickle cells, leads to splenic trapping and removal of cells. Some degree of intravascular hemolysis occurs as well. Hemoglobin electrophoresis reveals a predominance of hemoglobin S. Sickle cells are observed on the peripheral smear.

Treatment for Acute Hemolytic Anemia

i. Prophylactic folic acid 5 mg once daily is indicated because active hemolysis can consume folate and cause megaloblastosis.
ii. Blood transfusion if Hb dropped to less than 7 g/l in a dose of 15 ml/kg packed RBCs
iii. Corticosteroids are indicated in autoimmune hemolytic anemia (AIHA).
iv. Intravenous immunoglobulin G (IVIG) has been used for patients with AIHA, but only a few patients have responded to this treatment, and the responses have been transient.
v. Rituximab :
Rituximab belongs to a group of medicines known as monoclonal antibodies (Anti-CD20 Antibody).
It is designed to recognize specific proteins that are found on the surface of some lymphoma cells. The monoclonal antibody recognizes the protein and locks onto it (like a key in a lock). This may then trigger the body's immune system to attack and destroy the cancer cells.
Rituximab appeared to have efficacy in the treatment of AIHA (Auto-Immune Hemolytic Anemia) and Delayed hemolytic transfusion reaction
(DHTR),
vi. In more severe cases, the following treatments may be necessary and usually require hospitalization:

A- Exchange transfusion (similar to a blood transfusion but with more blood being given and an equal amount of hemolyzed blood being removed)

B- Surgical removal of the spleen

C- Immunosuppressive therapy

References

Great Ormond Street Hospital - UK - guidelines
American Society For Hematology clinical guidelines

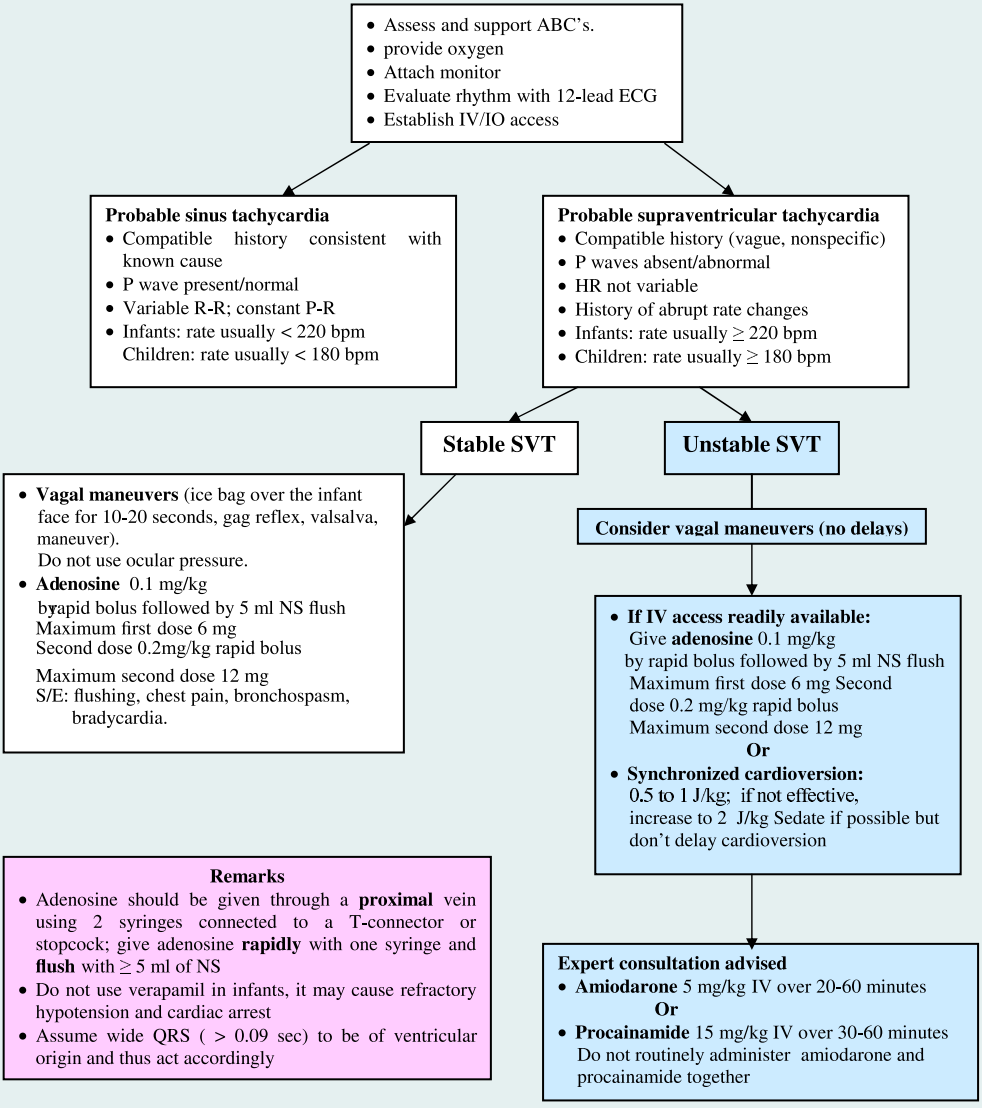
Supraventricular Tachycardia (SVT)

Paediatric Cardiology Unit
Chest Hospital

Supraventricular Tachycardia (SVT)

Signs & Symptoms

Irritability, vomiting, poor feeding, chest pain, palpitation. Poor perfusion, tachycardia, signs of heart failure, shock



References:

1. Moss and Adams; Heart Disease in Infants, children, and Adolescents, seventh edition.
2. Myung K. Park; Pediatric Cardiology for Practitioners, third edition.
3. Pediatric Acute Care, second edition.
4. The Harriet Lane Handbook, sixteenth edition.
5. American Heart Association Guidelines, 2001.

Medications for Pediatric Resuscitation and Arrhythmias

Medication	Dose	Remarks
Adenosine	0.1 mg/kg (max 6 mg) Repeat: 0.2 mg/kg (max 12 mg)	Monitor ECG Rapid IV/IO push
Amiodarone	5 mg/kg IV/IO over 20-60 minutes (maximum 300 mg) Repeat up to daily max 15 mg/kg (2.2 g in adolescents)	• Monitor ECG and BP • Adjust administration rate to urgency (give more slowly when perfusing rhythm present) • Use caution when administering with other drugs that prolong QT (consider expert consultation)
Atropine	0.02 mg/kg IV/IO (min dose 0.1 mg, max single dose child 0.5mg, adolescent 1 mg) Repeat once if needed, max total dose child 1 mg, adolescent 3 mg) 0.04 to 0.06 mg/kg ET*	Higher doses may be used with organophosphate poisoning
Calcium chloride (10%)	20 mg/kg IV/IO (0.2 ml/kg)	Push Slowly during arrest. Repeat PRN. Adult dose: 5-10 ml
Epinephrine	0.01 mg/kg (0.1 ml/kg 1:10 000)IV/IO 0.1 mg/kg (0.1 ml/kg 1:1000) ET* maximum dose: 1 mg IV/IO; 10 mg ET*	May repeat q 3-5 min
Glucose	0.5-1 g/kg IV/IO	D10W: 5-10 ml/kg D25W: 2-4 ml/kg D50W: 1-2 ml/kg
Lidocaine	Bolus: 1 mg/kg IV/IO Maximum dose: 100 mg Infusion: 20-50 µg/kg/min ET*: 2-3 mg/kg	
Magnesium sulfate	25-50 mg/kg IV/IO over 10-20 min; faster in torsades Maximum dose: 2 g	
Naloxone	<5 y or ≤20 kg: 0.1 mg/kg IV/IO/IM/SC ≥5 y or >20 kg: 2 mg IV/IO/IM/SC	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1-5 µg/kg)
Procainamide	15 mg/kg IV/IO over 30-60 min adult dose: 20 mg/min IV infusion up to total maximum dose 17 mg/kg	Monitor ECG and Bp Do not use routinely with amiodarone Use caution when administering with other drugs that prolong QT
Sodium bicarbonate	1 mEq/kg/dose IV/IO slowly	After adequate ventilation

* flush with 5 ml of normal saline and follow with 5 ventilations
(IV:intravenous-IO:intraosseous-ET:endotracheal-IM: intramuscular-SC:subcutaneous)

Medications to maintain cardiac output and for postresuscitation stabilization

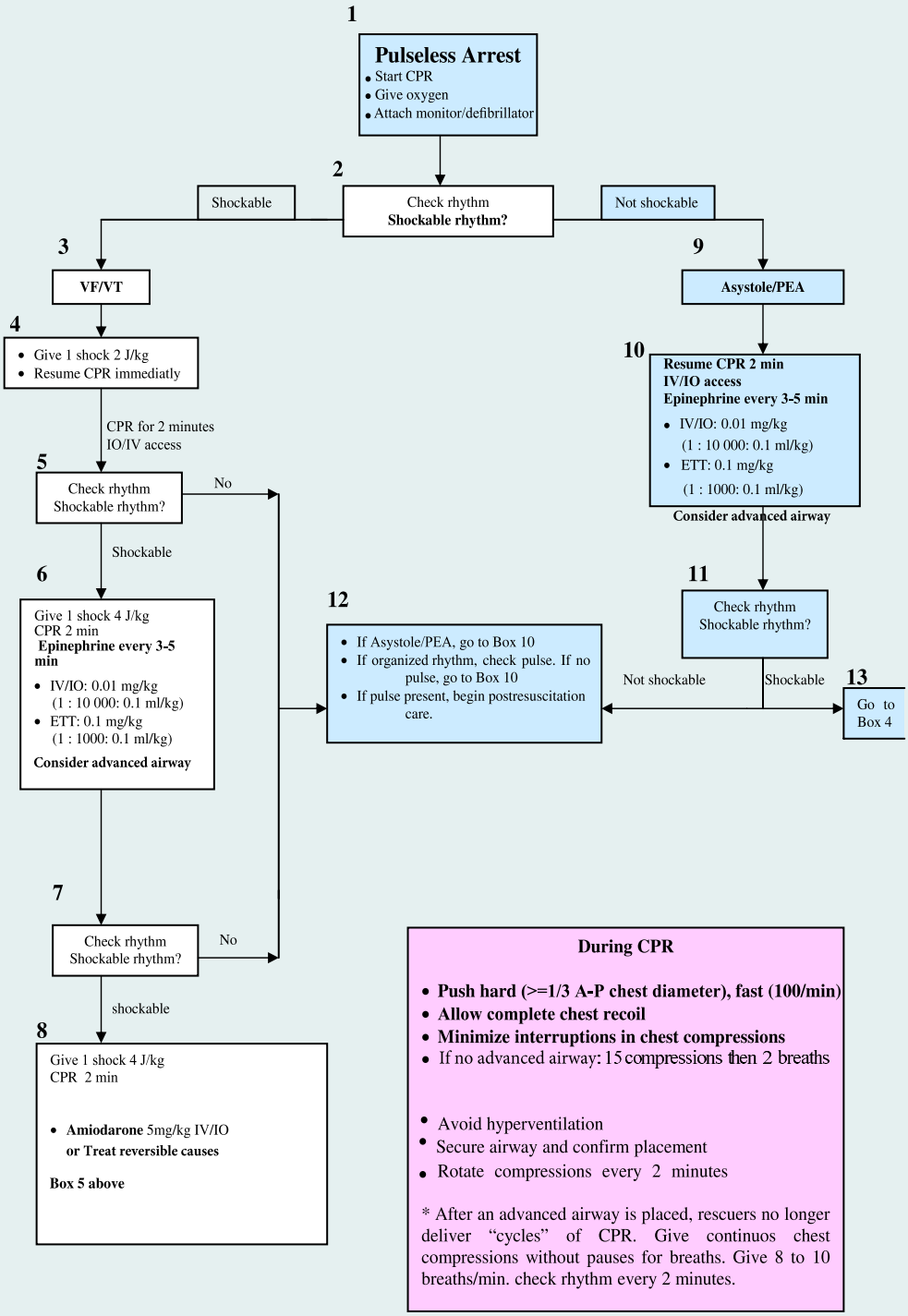
Medication	Dose	Comment
Dobutamine	2-20 µg/kg/min IV/IO‡	Inotrope; vasodilator
Dopamine	2-20 µg/kg/min IV/IO‡	Inotrope; chronotrope; renal and splanchnic vasodilator in low doses; pressor in high dose
Epinephrine	0.1-1 µg/kg/min IV/IO*	Inotrope; chronotrope, vasodilator in low doses; pressor in higher doses
Norepinephrine	0.1-2 µg/kg/min*	Inotrope; vasopressor
Sodium nitroprusside	0.3 to 1 µg/kg/min‡inital dose, increase up to 8 µg/kg/min	Vasodilator; prepare only in D5W

‡ 6 x body weight (in kg) = mg of drug to add to 100 ml D5W
then, an IV rate of 1 ml/h delivers 1 µg/kg/min of drug

* 0.6 x body weight (in kg) = mg of drug to add to 100 ml D5W
then, an IV rate of 1 ml/h delivers 0.1 µg/kg/min of drug

References:

1. American Heart Association in collaboration with International Liaison Committee on Resuscitation and European Resuscitation Council. From the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With treatment Recommendations, Part 6: Pediatric Basic and Advanced Life . Support. *Circulation* 2011;112:III -73-III-90.
2. American Heart Association. Part 12: Pediatric Advanced Life Support. *Circulation.* 2011; 112: IV-167-IV-187.
3. American Heart Association. Part 11: Pediatric Basic Life Support. *Circulation.* 2011; 112: IV-156-IV-166.

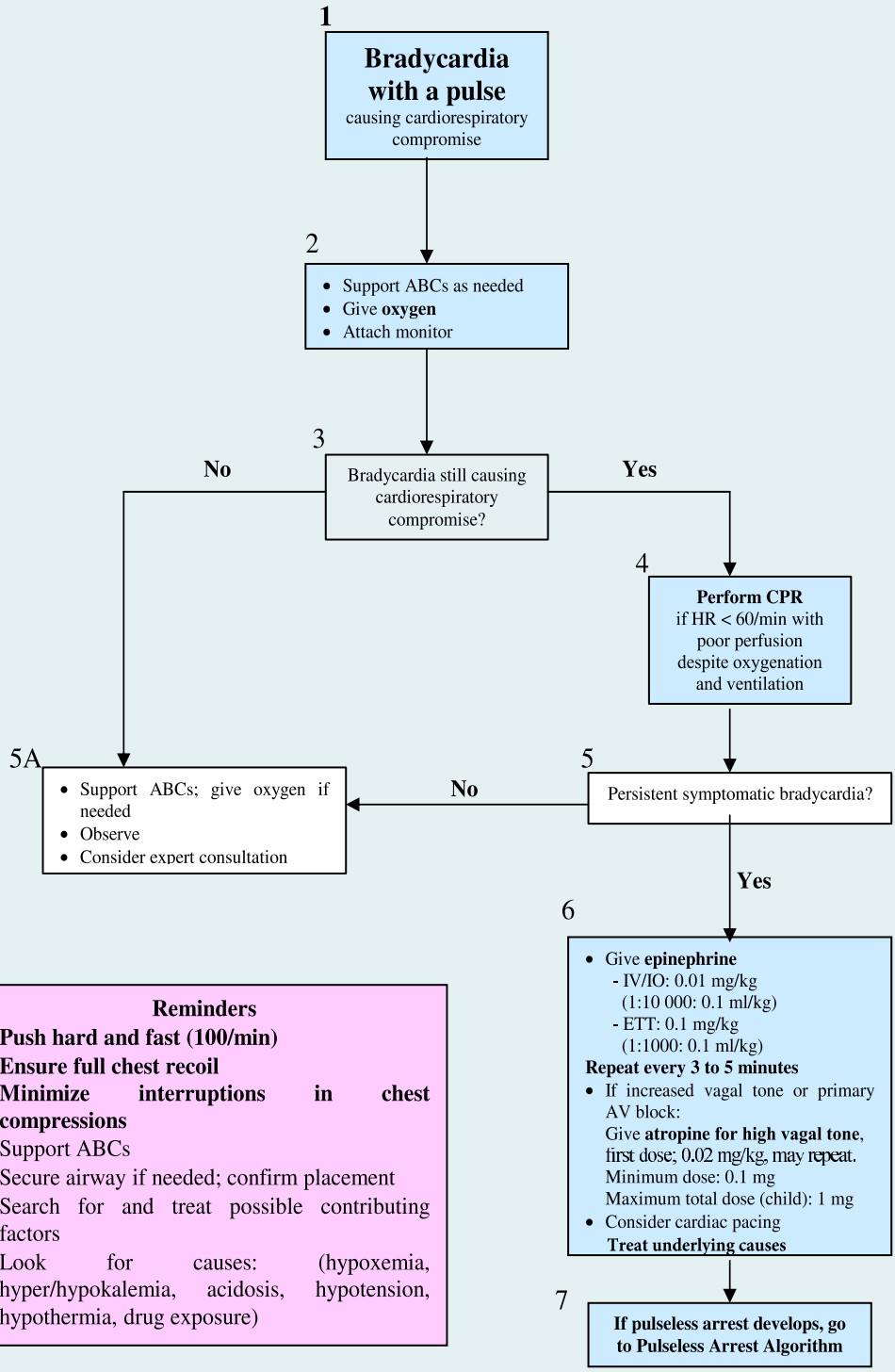


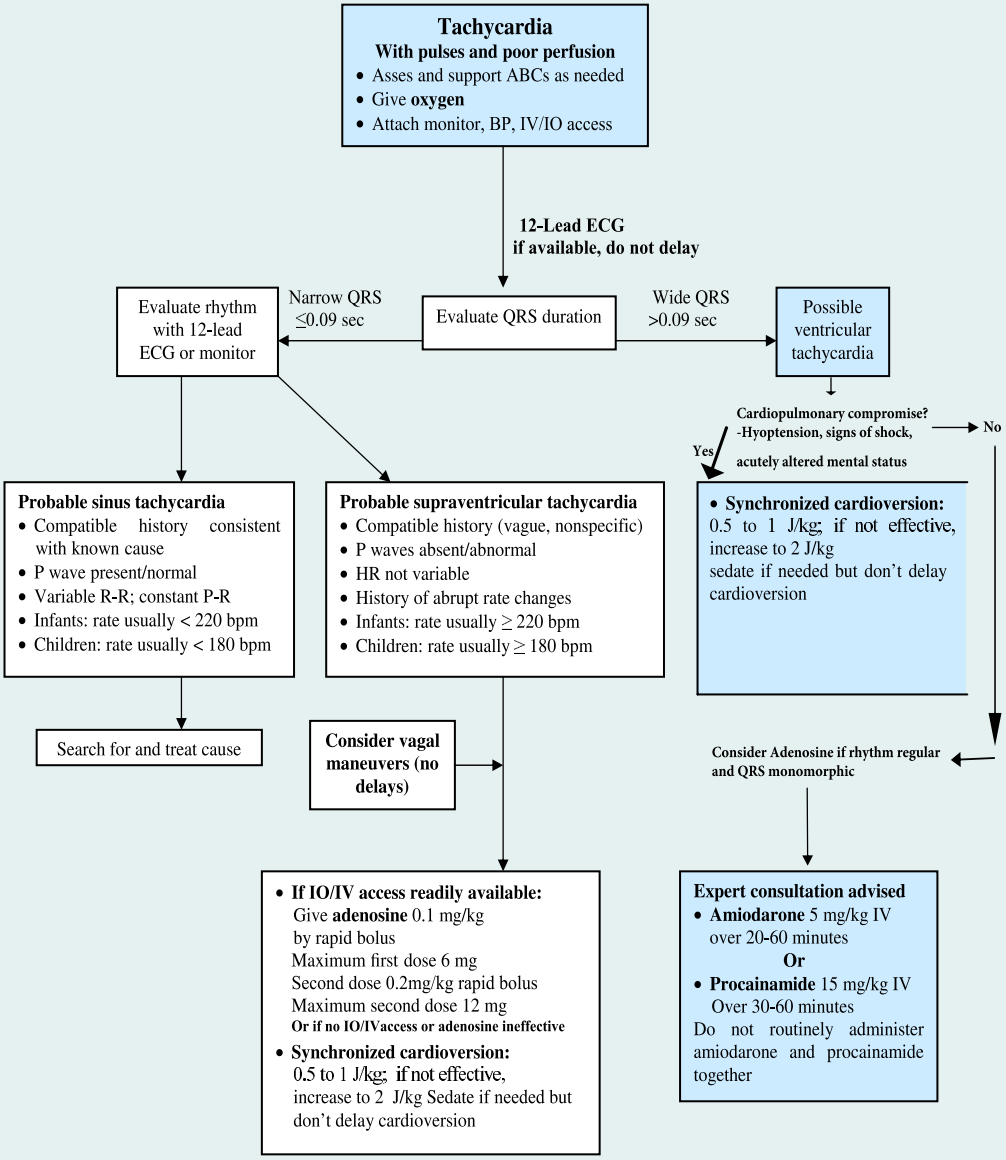
During CPR

- **Push hard (>=1/3 A-P chest diameter), fast (100/min)**
- **Allow complete chest recoil**
- **Minimize interruptions in chest compressions**
- If no advanced airway: 15 compressions then 2 breaths

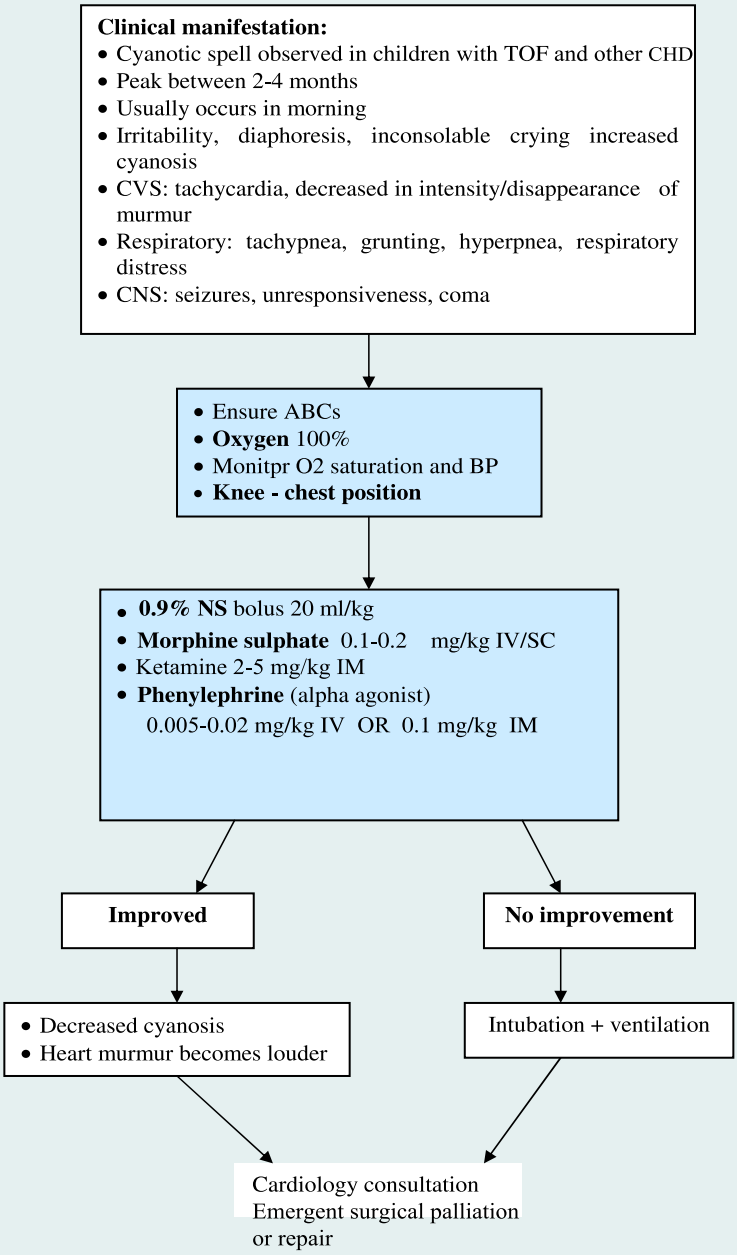
- Avoid hyperventilation
- Secure airway and confirm placement
- Rotate compressions every 2 minutes

* After an advanced airway is placed, rescuers no longer deliver "cycles" of CPR. Give continuos chest compressions without pauses for breaths. Give 8 to 10 breaths/min. check rhythm every 2 minutes.


1
Bradycardia with a pulse
causing cardiorespiratory compromise
2
Support ABCs as needed
Give oxygen
Attach monitor
3
Bradycardia still causing cardiorespiratory compromise?
No
Yes
4
Perform CPR
if HR < 60/min with poor perfusion despite oxygenation and ventilation
5
Persistent symptomatic bradycardia?
No
5A
Support ABCs; give oxygen if needed
Observe
Consider expert consultation
Yes
6
Give epinephrine
- IV/IO: 0.01 mg/kg (1:10 000: 0.1 ml/kg)
- ETT: 0.1 mg/kg (1:1000: 0.1 ml/kg)
Repeat every 3 to 5 minutes
If increased vagal tone or primary AV block:
Give atropine for high vagal tone, first dose: 0.02 mg/kg, may repeat.
Minimum dose: 0.1 mg
Maximum total dose (child): 1 mg
Consider cardiac pacing
Treat underlying causes
7
If pulseless arrest develops, go to Pulseless Arrest Algorithm
Reminders
Push hard and fast (100/min)
Ensure full chest recoil
Minimize interruptions in chest compressions
Support ABCs
Secure airway if needed; confirm placement
Search for and treat possible contributing factors
Look for causes: (hypoxemia, hyper/hypokalemia, acidosis, hypotension, hypothermia, drug exposure)


Tachycardia
With pulses and poor perfusion
• Asses and support ABCs as needed
• Give oxygen
• Attach monitor, BP, IV/IO access
12-Lead ECG
if available, do not delay
Evaluate rhythm with 12-lead ECG or monitor
Narrow QRS ≤0.09 sec
Evaluate QRS duration
Wide QRS >0.09 sec
Possible ventricular tachycardia
Cardiopulmonary compromise?
-Hyoptension, signs of shock,
acutely altered mental status
Yes
No
Probable sinus tachycardia
• Compatible history consistent with known cause
• P wave present/normal
• Variable R-R; constant P-R
• Infants: rate usually < 220 bpm
• Children: rate usually < 180 bpm
Probable supraventricular tachycardia
• Compatible history (vague, nonspecific)
• P waves absent/abnormal
• HR not variable
• History of abrupt rate changes
• Infants: rate usually ≥ 220 bpm
• Children: rate usually ≥ 180 bpm
• Synchronized cardioversion:
0.5 to 1 J/kg; if not effective, increase to 2 J/kg
sedate if needed but don't delay cardioversion
Search for and treat cause
Consider vagal maneuvers (no delays)
Consider Adenosine if rhythm regular and QRS monomorphic
• If IO/IV access readily available:
Give adenosine 0.1 mg/kg
by rapid bolus
Maximum first dose 6 mg
Second dose 0.2mg/kg rapid bolus
Maximum second dose 12 mg
Or if no IO/IVaccess or adenosine ineffective
• Synchronized cardioversion:
0.5 to 1 J/kg; if not effective,
increase to 2 J/kg Sedate if needed but don't delay cardioversion
Expert consultation advised
• Amiodarone 5 mg/kg IV over 20-60 minutes
Or
• Procainamide 15 mg/kg IV Over 30-60 minutes
Do not routinely administer amiodarone and procainamide together

Management of Cyanotic Spell



References:

1. Moss and Adams; Heart Disease in Infants, children, and Adolescents,seventh edition.
2. Myung K. Park; Pediatric Cardiology for Practitioners, third edition.
3. Pediatric Acute Care, second edition.
4. The Harriet Lane Handbook, sixteenth edition.
5. American Heart Association Guidelines, 2001.

2013 Clinical Practice Guideline on the

Evaluation and Management of Immune Thrombocytopenia (ITP)

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Guideline on the Evaluation and Management of Immune Thrombocytopenia (ITP)

Definition

Immune Thrombocytopenia (ITP) is an acquired immunemediated disorder characterized by isolated thrombocytopenia, defined as a peripheral blood platelet count less than 100 x 109/L, and the absence of any obvious initiating and/or underlying cause of the thrombocytopenia.

New Terminology

Newly diagnosed ITP Replaces acute ITP
Persistent ITP --- ITP (plats <100 for 3 to 12 months)
Chronic ITP ------ ITP (plats <100 for over 12 months)

Diagnosis

- **History:** Isolated bleeding symptoms consistent with thrombocytopenia without constitutional symptoms (e.g. significant weight loss, bone pain, night sweats).
- **Physical examination**: Bleeding symptoms in the absence of hepatosplenomegaly, lymphadenopathy, or stigmata of congenital conditions.

Investigations

- Complete blood count: Isolated thrombocytopenia (platelet count <100 109/L).
- Peripheral blood smear: Identified platelets should be normal to large in size. Red and white blood cell morphology should be normal.
- Bone marrow examination is unnecessary in patients with the typical features of ITP outlined above, irrespective of the age of the patient.

- **Bone marrow examination is done in ITP If :**

1- prior to initiation of treatment with corticosteroids.
2- prior to splenectomy
3- In patients who fail intravenous immunoglobulin (IVIg) therapy.

There is insufficient evidence to support the routine use of anti-platelet, antiphospholipid, and anti-nuclear antibodies, thrombopoietin levels, or platelet parameters obtained on automated analyzers in the evaluation of patients with suspected ITP.

Management

The goal of all treatment strategies for ITP is to achieve a platelet count that is associated with adequate hemostasis, rather than a normal platelet count. The decision to treat should involve a discussion with the patient and consideration of the severity of bleeding, anticipated surgical procedures, medication side effects, and health-related quality of life.

1. Assessment of Disease Status:

- What bleeding is the patient experiencing?
- Determine the timing, location, and severity of bleeding symptoms.
- Does this patient have any additional risk factors for bleeding such as use of antithrombotic agents or high-risk occupation?
- Is a surgical procedure anticipated?

Patients with no bleeding or mild bleeding (defined here as skin manifestations only, such as petechiae and bruising) can be treated With observation alone regardless of platelet count.

In Case of troublesome bleeding

corticosteroids (Give 4mg/Kg Prednisolone for maximum 4 days or of preferred
low dose 1-2 mg / Kg prednisolone for max 14 days, do not tail a/c to platelets, do not give maintenance

If a rapid increase in Platelets count are needed

A single dose of IVIg (0.8-1.0 g/kg

- Platelets are only indicated in life threatening bleeding along with immune suppression.

High dose Dexamethasone

May be considered for children or adolescents with ITP who have significant ongoing bleeding
30-mg single daily dose for 4 consecutive days, every 28 days.
A total of 3 cycles.

Rituximab

If no response to steroids and IVIG
AND STILL HAVE TROUBLESOME BLEDING
- Single 375mg/m dose induces remission in 40% adults at 1 year

Immunosuppressant

Multiple agents have been reported; however data for any one specific agent remain insufficient for specific recommendations.

References

1- ASH (American Hematology Association) ITP guidelines 2012
2- NHT (National Health Trust) , Great Ormond Street , London- UK

Management of fever & isolated neutropenia in previously healthy children

Dr. Entesar Husain & Dr. Mona Bo Rhama

Management of fever & isolated neutropenia in previously healthy children

Dr. Entesar Husain & Dr. Mona Bo Rhama

Neutropenia is defined as an absolute neutrophil count (ANC) ≤1.5 x 10^9 /L. ANC is the percentage of bands plus mature neutrophil x the total white blood cell count. It is divided into:

Mild	>1.0–1.5 x 10^9 /L
Moderate	>0.5–1.0 x 10^9 /L
Severe	≤ 0.5 x 10^9 /L

Patients with isolated neutropenia and otherwise normal immune systems would be expected to have lower risks of infection at any ANC.

Transient neutropenia (acute): lasting less than 2 weeks

Chronic neutropenia (persistent): more than 2 weeks in duration

Etiologies of transient isolated neutropenia in children

1. Viral : CMV, EBV, influenza, Parvovirus B19, HIV
2. Bacterial: Brucella, typhoid fever, paratyphoid, TB
3. Protzoa: malaria
4. Drugs: anticonvulsants (carbamazepine, valproate), antimicrobial (sulfonamides, penicillins, trimethoprim/sulfamethoxazole), antirheumatic (Gold, penicillamine), immune (neonatal isoimmune, autoimmune)

Clinical evaluation

History:

- Frequency of illness, the severity of fever
- Presence or absence of oral inflammation (mouth ulcers, gingivitis, periodontitis, tooth loss and replacement)
- Presence or absence of other common infectious problems of patients with neutropenia (cellulitis, sinusitis, otitis, pharyngitis, pneumonia, gastrointestinal symptoms, perirectal infections and sepsis or evidence of deep tissue infections).
- Medication history
- Family history of immune deficiency or unexplained infantile deaths

Physical examination

- Mouth: ulcers, abnormal teeth and gums
- Acute or chronic sinusitis, pharyngitis, or otitis.
- Careful examination of the chest and
- Abdominal tenderness & guarding
- The perirectal and anal area (tenderness or collection)
- Cellulitis, including necrotizing cellulitis

Investigations (if admitted)

- CBC + blood film
- ESR
- Blood culture
- Urine culture
- RFT & LFT

Management

- Healthy children with acute transients neutropenia + the rest of CBC is normal
  - If there is a focus of infection
    Treat the focus
  - No focus of infection
    - Mild & moderate neutropenia: can be treated as an outpatient. Perform a blood culture. Patients should be followed up with another CBC in 3 days
    - Severe neutropenia: admit to hospital and start on ceftazidime 150 mg/k/day.
  - If the child is ill looking, admit regardless of the ANC count and should be treated with ceftazidime
- Children with neutropenia > 2 weeks: Consult with hematology

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Alarioi AJ. Management of the febrile, otherwise healthy child with neutropenia. Pediatr Infect Dis J. 1994 Feb;13(2):169-70.

Sickle Cell Disease In Childhood

Standards And Guidelines For Clinical Care

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Conditions to be treated

Haemoglobin SS (Sickle Cell Anemia)
Haemoglobin SC
Haemoglobin SD (Punjab)
Haemoglobin SE
Haemoglobin S/ß thalassaemia
Haemoglobin SOArab

Presentation

1- Infant sudden death before diagnosis (pneumococcal sepsis due to splenic hypofunction or acute splenic sequestration)
2- No symptoms (till HB F is switch at 6 month)
3- Dactylitis is a common presentation (In infants between 9-18 months)
4- Sickle cell crisis which includes :

A- Vaso-Occlusive Crises

is caused by sickle-shaped red blood cells that obstruct capillaries and restrict blood flow to an organ, resulting in ischemia, pain, necrosis and often organ damage. The frequency, severity, and duration of these crises vary considerably.

B- Splenic Sequestration Crises :

Splenic sequestration crises: are acute, painful enlargements of the spleen. The sinusoids and gates would open at the same time resulting in sudden pooling of the blood into the spleen and circulatory defect leading to sudden hypovolaemia. The abdomen becomes bloated and very hard. markedly elevated reticulocyte count

Splenic sequestration crises are considered an emergency. If not treated, patients may die within 1-2 hours due to circulatory failure

C- Aplastic Crises

Aplastic crises are acute worsening of the patient's baseline anaemia, producing pallor, tachycardia, and fatigue. This crisis is triggered by parvovirus B19, which directly affects erythropoiesis . Reticulocyte counts drop dramatically during the disease

D - Hemolytic Crises :

Haemolytic crises are acute accelerated drops in haemoglobin level. The red blood cells break down at a faster rate. This is particularly common in patients with co-existent G6PD deficiency.

E - Acute chest syndrome :

It is an important cause of morbidity and mortality in SCD

Common in early childhood although, at that time, the clinical features are generally more typical of pneumonia. In later childhood and adulthood, the syndrome can develop during a painful crisis or after anesthesia. The disorder is most common in the 2 to 4 year age group and gradually declines in incidence with age

Characterized by a combination of some of these symptoms :

- Fever - sever chest pain (pleurisy pain)
- Difficulty in breathing
- cough, sputum production
- Abnormality in chest auscultations
- Low oxygen saturation (less than95 %)
- CXR shows pulmonary infiltrates (new), Serial

chest x-rays are needed to assess the extent and course of the pulmonary changes

AUDIT STANDARDS

Penicillin prophylaxis

Penicillin V (or Erythromycin if sensitive) should be prescribed by 3 -6 months for Infants.

Penicillin v prophylaxis should be started while waiting for clarification of diagnosis if this is delayed.

Penicillin v should be given lifelong.

Refusal to start Penicillin by parents should be documented.

<1yr 62.5 mg/ po/Bd

1-5yr 125 mg/po/bd

>5yr 250 mg/po/bd

Pneumococcal immunization

Pneumovax (polysaccharide antigen) at 2 years of age and five-yearly thereafter

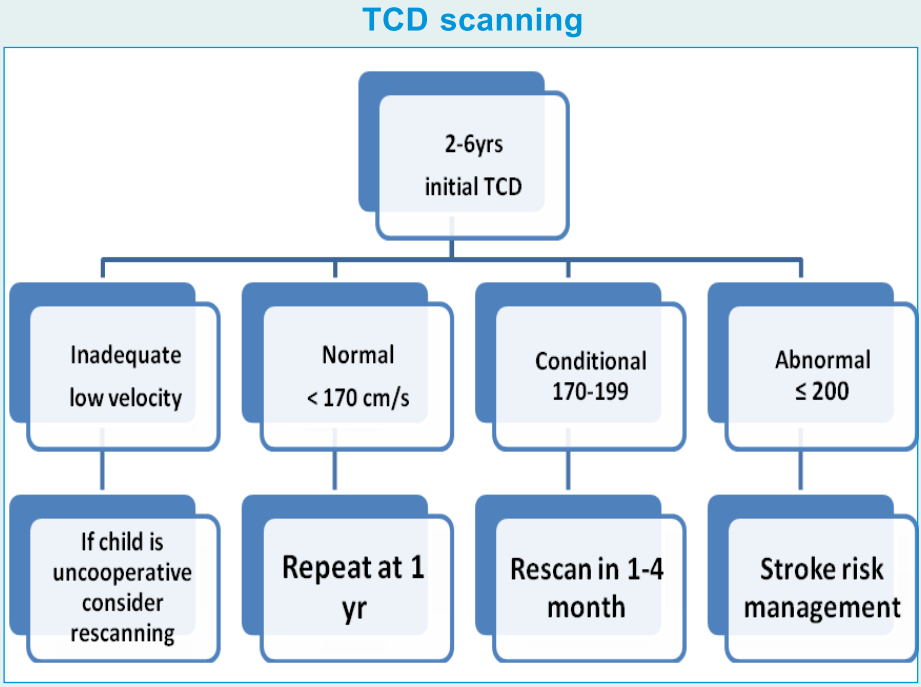
Vaccinations

A course of Hepatitis B immunization should be offered to non immune at the age of 1 yr - 3 doses -(12 month , 13 month and 18 month)

Annual Influenza vaccination should be offered

TCD scanning

Children with SCD (Hb SS and HbS/?0 thalassaemia) should be offered annual TCD scans from the age of 2 to 16 years



The OPD Follow up

Frequency

Minimum of 3 monthly during the first 2 years
6 monthly until the age of 5 yrs
annually thereafter

History

Current symptoms and a review of painful episodes,
Hospital admissions since the last consultation
Adherence to penicillin prophylaxis
How pain and fever is managed at home
Regularity of school attendance and reasons for absence

Examination

Weight & Height)) Assessment of growth and development
A general physical examination that should take particular note of any pallor,

jaundice, spleen size, presence of heart murmur
Oxygen saturation

At the first consultation, investigations should include

Full blood count.
Haemoglobin electrophoresis.
Reticulocyte count.
Blood group and extended red cell phenotype.
G6PD assay
Liver , Renal profiles
Urine Routine

Follow up Investigations

Full Blood count with retics
Liver, Renal profiles
Or as clinical concern

Management of pain at home

Paracetamol and ibuprofen are the analgesics of choice in mild-to-moderate pain.

Can be given together (alternative with 3 hrs interval)

Codeine phosphate can be added for more severe pain, but it should be recognized that at least 20% of cases will not respond due to the lack of the enzyme needed to convert it to morphine.

If there is no response to these, the child should be assessed in hospital to start Morphine.

non- pharmacological therapies for pain, such as good hydration , massage. Children should be encouraged to use psychological coping strategies, including distraction techniques such as games, computers and television

avoid factors that regularly trigger acute pain, such as exposure to cold weather, excessive physical activity and dehydration.

Zinc vitamins Should be given if growth retarded

Nutrition and Growth

Impaired growth, poor nutritional status and delayed skeletal and sexual maturation are common in children with SCD.

Check height & weight each visit

Zinc Supplementation should be given if there is growth retardation

High caloric diet (1.5 Kcal/Ml) to help catching the growth is of great help.

Nocturnal enuresis

If nocturnal enuresis is present over the age of 6 years, this should be

documented and parents should be given information and advice on treatment

Liver disease

Annual steady-state liver function tests should be carried out.

Recurrent episodes of abdominal pain should be investigated with an ultrasound of liver and biliary tree).)

Elective cholecystectomy should be carried out in symptomatic biliary disease

Kidney disease

Renal complications are relatively common in SCD, particularly with increasing age

Any child with a urinary tract infection should be treated and then investigated

Macroscopic haematuria should be fully investigated

Blood pressure, urea, creatinine and electrolytes should be measured on a yearly basis and renal investigations initiated if hypertension is present or if there are raised creatinine and urea levels

Cerebrovascular disease

Annual TCD scans should be performed on all children with SCD from aged 2 years

For those children who are considered to be "high risk", the risks and benefits of starting regular blood transfusions and/or other treatments should be fully discussed

Appropriate imaging studies to assess if

Evidence of cerebral vessel narrowing on TCD, learning difficulties, atypical symptoms such as unusual behavior during acute pain, frequent headaches, fits or other unexplained neurological, psychiatric or psychological symptoms

Blood pressure should be measured and recorded annually

Transfusion therapy should be offered throughout childhood for the secondary prevention of stroke.

Avascular necrosis of the femoral and humeral head

An MRI scan should be carried out where there is persistent pain in the hip or shoulder.

Referral to an orthopedic surgeon to be done.

Priapism

All boys and their parents should be warned early in childhood about priapism being a complication of SCD

For minor events, complete bladder emptying before sleep, pain relief and warm baths should be recommended.

Management of acute complications

Management of the febrile child

The most common organisms are encapsulated pneumococcus due to aspleen and Salmonella .

Admission to hospital if fever more than 38 c

Cultures of blood, urine and other possible sites of infection should be routinely done on any child presenting with acute pain and fever

Malaria films should be sent if there is any suspicion of malaria or if a patient has returned from a malarial region in the previous year.

Empirical antibiotics should be given appropriate for the range of likely infectious agents. An agent active against pneumococcus should always be included.

Suspected chest infection should include agents against atypical organisms

Prophylactic penicillin should always be continued in hospital if different antibiotic is not prescribed to treat an acute infection

Acute chest syndrome

The acute sickle chest syndrome is characterised by

pleuritic chest pain, fever, abnormal chest examination and new pulmonary infiltrates on the chest X-ray.

It is an important cause of morbidity and mortality in SCD

Common in early childhood although, at that time, the clinical features are generally more typical of pneumonia. In later childhood and adulthood, the syndrome can develop during a painful crisis or after anesthesia.

Early intervention with :

IV fluids at a rate equal to one and a half times the expected maintenance requirements

analgesia, (Paracetamol and ibuprofen) -- avoid narcotics

oxygen, to keep oxygen saturation more than 95 %

Bronchodilators

physiotherapy,

antibiotics (includes high dose third generation cephalosporin's plus a macrolide antibiotic to cover atypical community acquired pneumonia.

Blood Transfusion or exchange transfusion is essential

PICU ventilatory support to be arranged if needed.

Serial chest x-rays are needed to assess the extent and course of the pulmonary changes.

incentive spirometry performed regularly ever 2 hrs reduces the risk of acute chest syndrome in patients with chest and back pain

Children with either two or more episodes of acute chest syndrome in the last 2 years, or one episode requiring ventilatory support, should be offered hydroxyurea

Hydroxyurea

Hydroxycarbamide))

Indications

Hydroxyurea should be considered in patients who:

1- have recurrent episodes of acute pain (more than three admissions in the previous 12 months,
2- or are symptomatic in the community
3- or who have had two or more episodes of acute sickle chest syndrome.

Treatment Details

~ Baseline measurements- At least two months of baseline information on the hematologic status of patients should be available before starting treatment

~ Starting dose- hydroxyurea can be started at a dose of 10 mg/kg orally, on a daily basis.

~ Dose escalation- The dose of hydroxyurea can be increased at a rate of 5 mg/kg/wk as long as the hematologic values remain in an acceptable range, and the patient shows no other evidence of side-effect

~ Maximum dose- the maximum dose of hydroxyurea used in most patients is 25 mg/kg/day.

~ Trial period- Patients should remain on hydroxyurea for six to nine months before any decision is made on the efficacy of the treatment.

~ Limiting effects
1. platelet count of less than 80,000.
2. neutrophil count (not white count) of less that 2,500
3. hemoglobin of less than 6 g/dl.
4. hair loss, GI upset, rash.

~ Monitoring hydroxyurea
1. hydroxyurea blood levels- Blood levels of hydroxyurea are difficult to interpret .
2. MCV- The MCV rises in many patients treated with hydroxyurea.
3. Fetal hemoglobin levels- Fetal hemoglobin levels rise in many patients treated with hydroxyurea.

~ Contraindications to hydroxyurea Treatment
1. Pregnancy.
2. Poor or erratic follow-up.
3. allergies to hydroxyurea

This discussion should be documented in the patient's notes

Use of Blood Transfusion therapy

Indications for acute transfusion

Acute anemia (of an acute fall in hemoglobin of more than 2g/dl below steady-state hemoglobin) as in :

- aplastic crises - sequestration crises

Acute chest syndrome - early top-up transfusion may avoid the need for exchange transfusion

Stroke or acute neurological deficit - exchange transfusion is usually necessary to reduce the HbS to less than 30%, Hb10-11g/dl.

Multiorgan failure

Preparation for urgent surgery

Indications for regular, long-term transfusion

Primary and secondary stroke prevention

Recurrent acute chest syndrome not prevented by hydroxyurea

Progressive organ failure

Iron chelation should be started in all children on regular blood transfusions

Immunization against hepatitis A and B should be offered to all those on long term transfusion

Children receiving regular monthly blood transfusion should have a specific annual review

Bone marrow or stem cell transplantation

Bone marrow/stem-cell transplantation is the only treatment for SCD which is potentially curative with 92-94% survival rate

There is no recurrence of clinical vaso-occlusive events in 75 - 84 % of patients with stable engraftment, but 10% of patients experience rejection or recurrent SCD.

The majority of patients have an excellent quality of life after bone marrow transplantation (BMT

All patients or families with a child with SCD should be offered the opportunity to discuss BMT as a treatment option. This should not depend upon the family having an available donor at the time

Splenectomy

Recurrent splenic sequestration is an indication for splenectomy.

References

1- ASH (American Society of Hematology) guidelines

2- GOSH (Great Ormond Street Hospital)-London - UK guidelines

Clinical Practice Guidelines

febrile seizures*

Neurology unit
Sabah Hospital

Febrile Seizures

1. Definitions:

1.1. Simple febrile seizure (SFS)

A short (<15 min) generalized seizure, occurring during a febrile episode

(temperature > 38°C) not caused by an acute disease of the nervous system, in a child aged 6 months to 5 years, with no neurologic deficits, with normal psychomotor development, and with no previous afebrile seizures.

1.2. Complex febrile seizure (CFS)

A focal, or generalized and/or prolonged (>15 min) seizure, and/or occurring more than once in 24 h, and/or or occurring in a child with previous neurologic deficits. It can be associated with postictal (usually transient) neurologic abnormalities. Seizure stopped with anticonvulsive therapy (i.e., diazepam) before the 15th minute should also be classified within this group.

Febrile status epilepticus is a complex febrile seizure (CFS) characterized by a duration of more than 30 min, or by shorter serial seizures, without consciousness being regained in the interictal state.

2. Diagnosis and criteria for hospital admission

History and physical examination should direct clinician in direction of CNS infection (meningitis, encephalitis), gastroenteritis (shigella, salmonella), ingestions of toxic substances, hypoglycemia, electrolyte abnormalities, head injury and other conditions excluding febrile seizures.

2.1. Simple febrile seizures

~ First episode

~ Age > 18 months: admission is unnecessary if the patient is clinically stable, and the source of fever does not require hospitalization for investigations or treatment; parents should be adequately educated. Significant caregiver anxiety and concerns of coping with a possible recurrent seizure at home might be indication for admission.

~ Age < 18 months: admission is recommended for observation (at least 24h) for possible performance of lumbar puncture (see 3.1)

~ Already diagnosed simple febrile seizures: admission is unnecessary if patient is clinically stable and the source of temperature doesn't require investigations or treatment; parents' education must be verified. However, a history of simple febrile seizure (SFS) does not exclude that the ongoing seizure may be the symptom of another

wide variability of conditions underlying this event.

2.3. Febrile seizure in children without a reliable familiar context: admission is recommended

3. Investigations

3.1 Simple febrile seizure

- ~ Routine laboratory tests: the decision on whether or not to perform the tests should exclusively aim at identifying the cause of fever (search for fever etiology)
- ~ Routine electroencephalogram: not recommended because of limited diagnostic value in a child with a first SFS
- ~ Routine neuroimaging: not recommended
- ~ Lumbar puncture (LP) (usual contraindications apply):
- ~ In the presence of meningeal signs: to be performed
- ~ In patients receiving antibiotic treatment during the days before the seizure: recommended, owing to the possible masking of meningitis signs and symptoms
- ~ In patients of age <18 months: clinical signs and symptoms of meningitis tend to be less sensitive with lower age: LP should not be delayed , if there's any suspicion of meningitis
- ~ In infants between 6 and 12 months of age, a lumbar puncture should be strongly considered when the child is deficient in Haemophillus influenzae type b or Streptococus pneumoniae immunizations or when immunization status cannot be determined (increased risk of bacterial meningitis)
- ~ In patients of age >18 months: LP should not be considered as a routine procedure, as the clinical signs of CNS infection are usually identifiable

3.2. Complex febrile seizure

- ~ Search for fever etiology : recommended
- ~ Performance of blood chemical tests: decision is related to clinical condition
- ~ Search for a possible underlying brain lesion: recommended in order to differentiate the symptomatic forms of FS from those based on a genetic predisposition
- ~ Electroencephalogram: recommended (in case of suspected viral encephalitis as early as possible; if not postpone 2-3 weeks
- ~ Neuroimaging: computed tomography (CT) scan and/or nuclear magnetic resonance (NMR) are highly recommended
- ~ Lumbar puncture: in patients with a suspected CNS infection

4. **Treatment of an acute episode:**
In most cases, SFS spontaneously ceases within 2-3 min, and does not require treatment. If FS lasts longer than 3 min pharmacologic treatment is recommended (see 6.4).
5. **Recurrence risk and prevention** The general risk of febrile seizure recurrence is estimated at around 30-40%. The risk factors for recurrence, similar for simple and complex FS: early age of onset (<12 months), febrile seizures or epilepsy in first-degree relatives, febrile seizures in first-degree relatives, frequent febrile illnesses, low temperature at the onset of the febrile seizure

Due to benign prognosis **prophylaxis for SFS recurrences is not recommended**. Give parents exhaustive information, including instructions for diazepam SOS administration in the event of seizures > 3 min; on clinical follow up monitor the natural evolution of seizures.
In a restricted group of patients with SFS for whom the seizures are considered as "unacceptable" because of their high frequency (3 or more in 6 months, 4 or more in 1 year), or in patients with febrile status epilepticus or recurrent CFS

prophylaxis may be considered:

~ Intermittent therapy: oral (0.3 mg/kg) or rectal (0.5 mg/kg) diazepam, administered at the onset of fever, to be repeated after 8 - 12 h for 1 day if fever persists

~ In case of failure of intermittent therapy, and, in particular, when parents

are unable to promptly recognize the onset of fever, continuous anticonvulsive therapy with (preferably) valproic acid (20-30mg/kg/day in 2-3 intakes) or phenobarbiton (3-5mg/kg/day in 1- 2 intakes) may be used

6. Essential issues for family education

6.1. Describe to parents the features of febrile seizures

6.2. Verify that the instructions for fever control are well understood

6.3. Educate parents how to manage possible recurrences and administer rectal diazepam (0.5mg/kg, max 10mg), or **buccal midazolam (0.3 mg/kg, max 10mg)** in case of seizure lasting over 3 min

6.4. Instruct on the appropriateness of anticonvulsive therapy, when prescribed, including the relevant side effects.

6.5. Inform parents that a medical intervention is necessary in case:

~ Seizures of a duration >10 min or not remitting after treatment
~ Recurrent seizures during same febrile illness
~ Focal seizures
~ Presence of postictal palsy or patient not regaining consciousness soon after FC

~ **The recommendations do not indicate an exclusive course of management; variations, taking into account individual circumstances, may be appropriate.**

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AAP: Febrile seizures: Clinical practice guideline for the long-term management of the child with simple febrile seizures. Pediatrics 2008; 121: 1281 - 1286

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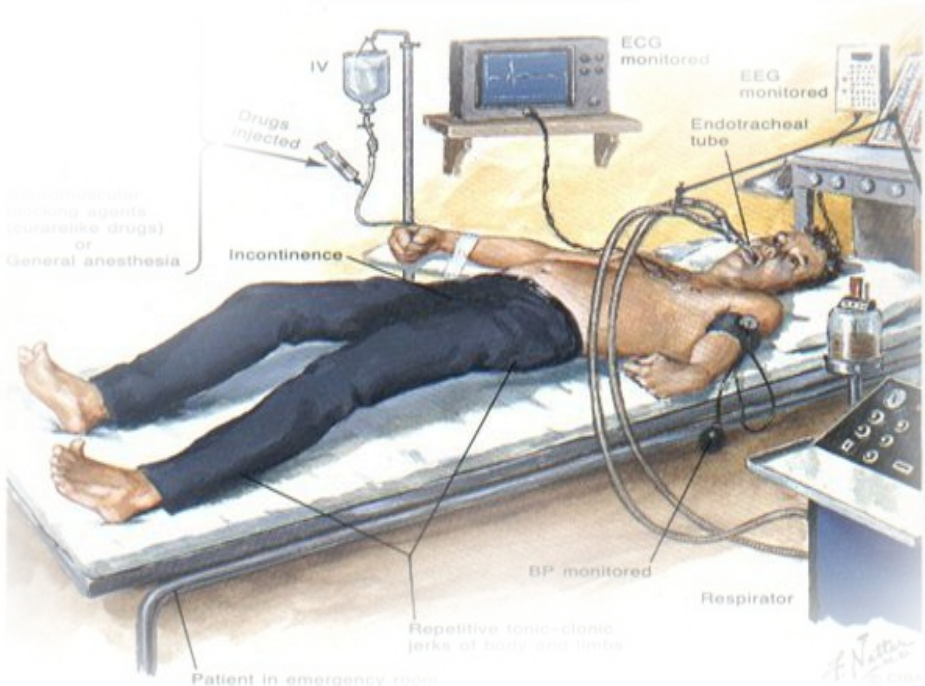

Generalized Convulsive Status Epilepticus (se) Protocol

Paediatric Neurology Unit
Al-Sabah Hospital

GENERALIZED CONVULSIVE STATUS EPILEPTICUS (SE) PROTOCOL

A. Resuscitation and stabilization:

- ~ Check ABCDE, manage accordingly, apply 100 % oxygen
- ~ Establish IV (IO) access
- ~ Labs for initial studies (CBC, BGA, glucose, electrolytes including Ca, Mg, P, liver and renal profile, consider CK, ammonia, lactate, septic work up, anticonvulsant level, toxicology, metabolic disease screen)
- ~ If (or suspected to be) hypoglycemic give 2ml/kg of 10% dextrose IV

Continue monitoring and management of ABCDE accordingly; if patient is shocked or cyanosed at any stage call PICU

B. Anticonvulsants:

Stage I: impending SE (5 - 10 minutes from the beginning of the attack)

~ at 5 minutes benzodiazepine: (one of the following)

Diazepam 0.3 - 0.5mg/kg (max 10mg) rectal
Midazolam 0.3mg/kg (max 10mg) buccal (nasal)

If IV access:
Lorazepam 0.05 - 0.1mg/kg (max 4mg) IV over 2-3 minutes
Diazepam 0.2 - 0.3 mg/kg (max 10mg) IV over 2 minutes
Midazolam 0.1 - 0.2mg/kg IV (max 10mg)
Midazolam 0.2mg/kg IM (max 10mg)

For children under 18 months, consider **Pyridoxine** (Vit B6) 30 mg/kg IV.

Stage II: early / established SE (10 - 30 minutes from the beginning of the attack):

- ~ at 10 minutes: repeat benzodiazepine
- ~ at 15 minutes:

Phenytoin (PHT) 15-20 mg/kg (max 1000mg) IV infusion over 20 min (max 50mg/min) under ECG monitor (prepare infusion as 10mg phenytoin/ml NS)
If seizure control is achieved continue with maintenance dose of PHT 5 - 8 mg/kg/day in 2 divided doses, monitor blood level and adjust the dose.

- ~ In 20 - 30 minutes start management of brain edema (20% Mannitol - section D)

Stage III: refractory SE (alert PICU)

~ at 35 minutes:

Phenobarbital (PB) 15-20 mg/kg (max 1000mg) IV over 10 min.
If seizure control is achieved continue with maintenance dose of PB 5mg/kg/day, monitor blood level and adjust the dose
In children younger than 18 months, PB should be considered before PHT

~ Other options:
Levetiracetam (Keppra) 20-40 mg/kg IV, over 10 - 15 min (prepare infusion as 5 ~ 15 mg levetiracetam / ml NS or D5%) followed by maintenance dose of 40 - 60 mg/kg/day (max 3000mg/day) in 2 divided doses

Na Valproate 25 - 30mg/kg IV infusion over 10 - 15 minutes followed by 20 - 40 mg/kg/day (max 2500mg) in 2 divided doses, monitor blood level and adjust the dose

~ At 45 minutes (PICU care):
Midazolam 0.1mg/kg - 0.2 mg/kg (max 10 mg) IV bolus over 2 minutes followed by 0.1mg/kg/hour IV infusion; if needed, can be titrated up to 0.5mg/kg/h (exceptionally higher or additional boluses can be used, be ready for endotracheal intubation and ventilation)
If possible start EEG monitoring

~ If seizure is not controlled with Midazolam infusion in 1-2 hours induce barbiturate coma (obligatory intubation and ventilation short acting muscle relaxant)
Thiopental: 2-5 mg/kg IV bolus over 5 min followed by 2-8 mg/kg/hr IV infusion
Discontinue Midazolam and Phenobarbital when thiopental infusion started, maintain phenytoin
Besides listed other anticonvulsants (PB)/anesthetics (propofol, lidocaine, ketamine…..) may be used

When seizures have been controlled for 12 - 24 hours, the drug dosage should be slowly reduced over further 12 - 24 h. If seizures recur, the drug infusion should be given again for another 12h and afterwards withdrawal attempted again.

C. Diagnostic workup & specific treatment:

Majority of patients with SE are not known to be epileptics, 30 - 50 % of cases have acute CNS insult (e.g. trauma, infection); after stabilization consider further work up including CT scan head and LP and EEG to identify and treat possible underlying cause (antibiotics, antipyretics).

D. Symptomatic & Supportive management:

~ Management of brain edema including 20% Mannitol 5ml/kg over 20 minutes (first dose to be started 30 minutes from the beginning of the attack) Dexamethasone 0.5mg/kg IV, then 0.15mg/kg/6 hr
~ Monitor clinical condition for possible deteriorations & complications of underlaying etiology and side effects of treatment such as hypotension, arrhythmia, myocardial depression
~ Monitor blood sugar, electrolytes, BGA, renal, liver, coagulation profile, antiepileptic drug level

Guidelines for the Management of

Diabetic Ketoacidosis in Children

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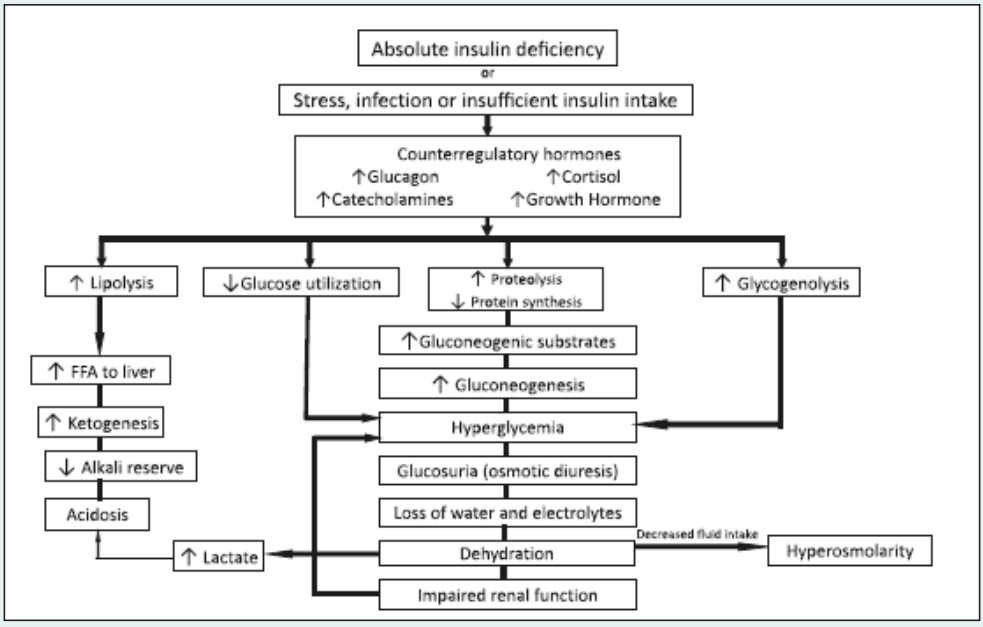
Guidelines for the Management of Diabetic Ketoacidosis in Children

Diabetic ketoacidosis (DKA) results from deficiency in circulating insulin and/or increased levels of counter-regulatory hormones: catecholamines, glucagon, cortisol and growth hormone. DKA can occur in the following conditions:

1. Newly diagnosed type 1 diabetic patients (especially the very young ones)
2. Insulin omission in already diagnosed patients:
   i. Deliberate insulin omission
   ii. Mismanagement during inter-current illness
   iii. Insulin pump mal-function



Pathophysiology of DKA (ISPAD 2014)

Definition of DKA:

Biochemical features of DKA include:

1. Blood glucose more than 11mmol/L
2. Venous pH < 7.2 or HCO3 < 15.
3. Ketonemia and Ketonuria.

The severity of DKA is categorized by the severity of acidosis

1. Mild DKA: pH< 7.3 or bicarbonate < 15 mmol/L
2. Moderate DKA: pH< 7.2or bicarbonate < 10 mmol/L
3. Severe DKA: pH< 7.1 or bicarbonate < 5 mmol/L

Clinical manifestation of DKA:

1. Dehydration
2. Tachypnea; deep respiration (Kussmaul breathing)
3. Nausea, vomiting, and abdominal pain that may mimic an acute abdominal condition
4. Confusion, drowsiness, may proceed to progressive loss of consciousness

Management of DKA:

A: Emergency Assessment: Should follow the guidelines of PALS

1. Perform a clinical assessment to confirm the diagnosis and determine a possible cause, look for signs of infections
2. Assess the clinical severity of dehydration:
    a. Patients with DKA are at least 5-7.5% dehydrated.
3. Weigh the patient:
    a. This weight will be used for all calculations
4. Assess level of consciousness: Glasgow Coma Scale
5. Obtain blood samples for:
    a. Plasma glucose: Bedside meter measurement can be used to start treatment (especially in know diabetics), but need to confirm by laboratory measurement.
    b. Serum electrolytes (including bicarbonate), blood urea nitrogen, creatinine
    c. Venous pH, pCo2
    d. Complete blood count: Note that increased white cell count may not be indicative of infection but a response to stress.
    e. Albumin, calcium, phosphorous and magnesium if possible
6. Urine analysis for ketones.
7. Obtain appropriate specimens for cultures: blood, urine if indicated.
8. ECG if serum potassium levels are delayed.

B: Additional measures

1. Secure airway, and empty stomach by continuous NG tube
2. Peripheral intravenous line (s) should be placed for painless repetitive blood sampling
3. Give oxygen for patients with severe circulatory impairment or shock
4. Place cardiac monitor
5. Give antibiotics to febrile patients after obtaining appropriate cultures
6. Catheterization of the bladder may be needed for unconscious patients or those unable to void on demand.

C: Where should the child with DKA be managed?

1. In a unit with :
    a. Experienced nursing staff
    b. Written guidelines for DKA management
    c. Access to a laboratory that can provide results for frequent measurements.
2. A specialist /consultant should be informant and plan should be discussed and approved

- ADMISSION to PICU:
    - o Children less than 2 years of age.
    - o Children with signs of cerebral edema

* Feel free to consult PICU if you have any concerns especially if the child is below 5 years of age.

D: Clinical and biochemical monitoring:

Successful management of DKA mandates careful and repeated monitoring

1 Documentation on a flow-chart of hourly clinical assessments, IV and oral medications, fluids.

2. Monitoring should include:
    a. Vital signs
    b. Neurological exam should be done regularly, initial examination is not enough. This should include:
    i. Restlessness, irritability
    ii. Increased drowsiness
    iii. Incontinence
    iv. Headache
    c. Signs of cerebral edema:
    i. Change of CNS status
    ii. Rising blood pressure
    iii. Decrease O2 saturation
    iv. Rapidly rising
3. Degree of dehydration
4. Capillary blood glucose hourly; crossed checked by laboratory at least every 2 hours (depending on the lab).
5. Other laboratory test: electrolytes and blood gas every 2-4 hours.

E: Goals of therapy:

1. Correct dehydration
2. Correct acidosis and reverse ketosis
3. Restore blood glucose to near normal
4. Monitor for complications of DKA and its treatment
5. Identify and treat any precipitating factors

F:Fluid and salt replacement

1. Patients with severe volume depletion/shock volume expansion should start with 0.9% saline 5 to 10 ml/kg/hour over 1-2 hours and repeated as need to restore circulatory status
2. Fluid management (deficit replacement) should continue with 0.9% saline for at least 4-6 hours.
3. The sodium content of the fluid may need to be increased or decreased according to the serum Na and to maintain and avoid drop of effective osmolality.
4. Potassium should be added after the initial bolus (see below)
5. The rate of IV fluids should be capsulated to rehydrate evenly over 48 hours.
6. Example of volumes of maintenance + 10% deficit, to be given over 48 hours:

Weight (kg) Infusion rate (ml/kg/hr)

Weight (kg)	Infusion rate (ml/kg/hr)
4-9	6
10-19	5
20-39	4
40-59	3.5
60-80	3

Example: A 6 year old boy weighing 20 kg will be given 80ml/hr or a total volume of 1920 my per 24 hrs for 48 hours.

7. Urinary losses should not be routinely added to the calculation of replacement fluid
8. When oral fluid is tolerated, IV fluid should be reduced accordingly
9. Avoid using hypotonic saline unless necessary. Try not to go below 0.45% NaCl fluid.

G: Insulin Therapy:

1. Start Insulin infusion 1-2 hours after starting fluids replacement therapy
2. Insulin therapy should be started in the ER/ Casuality, if the patient is still there, and should not be delayed.
3. Correction of insulin deficiency:
   1.Dose: 0.1 unit/kg/hr. (e.g dilute 50 units of **Regular** insulin in 50 my normal saline, 1 unit= 1m or prepare by adding 25u of insulin to 250 cc of normal saline (so 1u=10ml). Some use 10 units with 100 cc, it is the same.)
4. Route of administration : I V
5. IV bolus insulin is not needed

6. Initially, plasma glucose may fall 5-10 with rehydration even before insulin is started. However, it should not fall more than 5 mmol/hr with the insulin therapy.

7. Initial dose of insulin is 0.1 u/kg/hr. Consider reducing the insulin dose in case of the following:
    a. Rapid drop of glucose (>5 mmol/hr). Remember to add glucose.
    b. Intractable Hypokalemia. (To avoid central IV line)
    c. Intractable drop of osmolality (after managing Na concentration)
8. The dose of insulin should be between 0.5- 0.1 u/kg/hr until the resolution of DKA (pH > 7.3, bicarbonate > 15 mmol/L).

9. To prevent rapid decrease in plasma glucose, 5% glucose should be added to the IV fluids (ie 5% glucose with 0.9 saline) when plasma glucose falls to 15 mmol/l or sooner if the rate of fall is rapid. (ie more than 5 mmol/hr)

10. It may be necessary to use 10% or even 12.5% dextrose to prevent hypoglycemia, while continuing to infuse insulin to correct the metabolic acidosis.
    a. 7.5%D= add 25ml of 50%D to 500ml of 5%D.
    b. 10%D= add 50ml of 50%D.

11. If biochemical parameters of DKA (pH, bicarbonate) do not improve, reassess the patient hydration status, review insulin therapy (preparation, dose calculation, expiry dates) and the presence of other causes like infection. (Consider Changing the IV circuits and mixing a new insulin)

12. Keep NPO, until acidosis is under control (PH > 7.25), anorexia and delayed gastric emptying occur with acidosis.
13. Start with clear fluids, and go from there according to the patient's tolerance.
14. Keep mannitol 1gm/kg ready at the bedside.

H: Potassium (K) replacement:

1. Replacement is required regardless of serum potassium concentration
2. If the patient is hypokalemic, start potassium replacement at the time of initial fluid therapy and before staring insulin therapy.
3. Otherwise, start after the initial restoration of fluid and with starting insulin therapy.
4. Dose of K:
    a. Start with 40 mmol/L
    b. Subsequent dose is bases on serum K measurement
5. K replacement should continue throughput IV fluid therapy.
6. Given either as potassium chloride or phosphate
7. The maximum recommended rate of IV potassium replacement is usually 0.5 mmol/kg/hr

: Phosphate:

1. Studies have not shown clinical benefits from phosphate replacement
2. Severe hypophosphatemia with unexplained weakness should be treated.
3. Can be replaced safely as potassium phosphate

J: Acidosis:

1. Bicarbonate administration should not be routinely administered, unless in severe cases with shock.

Introduction of Oral fluids and transition to subcutaneous (SC) insulin:

1. Oral fluids should be introduced only when clinical improvement in hydration and neurological status is observed, and the patient is willing to eat (mild acidosis / ketosis/ketonuria may still be present)

2. When oral fluid is tolerated, IV fluid should be reduced

3. To prevent rebound hyperglycemia. The first SC injection should be given 15-30 min (with rapid acting analogues) or 30-60 min (with regular insulin) before stopping insulin infusion.

Cerebral Edema

A: Warning signs and symptoms:

1. Headache and slowing heart rate
2. Change in neurological status (restlessness, irritability, increased drowsiness, incontinence)
3. Specific neurological signs:
    a. Unreactive pupils
    b. Cranial nerve palsies
4. Rising blood pressure
5. Decreased O2 saturation

A: Treatment of Cerebral Oedema:

1. Initiate treatment as soon as the condition is suspected. DO NOT WAIT **FOR CT SCAN**

2. Reduce the rate of IV fluids to 75% the required

3. Give mannitol 0.5-1 g/kg IV over 20 minutes and repeat if there is no initial response in 30 minutes. To 2 hours

4. Hypertonic saline (3%), 5ml/kg over 30 minutes, may be an alternative to mannitol, especially if there is no response to mannitol
    a. Mannitol or Hypertonic saline should be available at the bedside

5. Elevate the head of the bed

6. Intubation may be necessary for the patient with impending respiratory failure, but aggressive hyperventilation (to a $pCO_2 < 2.9$ Pa) has been associated with poor outcome and is not recommended.

7. After treatment for cerebral edema has been started, a cranial CT scan should be obtained to rule out other possible causes for the neurological deterioration like IC hemorrhage, thrombosis.

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Hypoglycemia in infant and children

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Hypoglycemia in infant and children

Hypoglycemia is a medical emergency that may result in seizures, permanent brain damage, or even sudden death. This section presents an approach to disorders of hypoglycemia in infant and children based on the endocrine and metabolic systems involved in successful adaptation to fasting. Since the integrity of these systems is reflected in plasma levels of critical fuel and hormones the time of hypoglycemia, the most important specimens for diagnosis are the ones obtained at the time of hypoglycemia. These plasma and urine specimens are known as the " Critical samples" and should be obtained immediately before treatment of hypoglycemia. For approach of neonatal hypoglycemia (see neonatal hypoglycemia protocol).

Definition

Hypoglycaemia is defined as a blood sugar level (BSL) less than 2.6mmol/l.

The fasting systems

Three metabolic systems (hepatic glycogenolysis, hepatic gluconeogeneis, hepatic ketogenesis) are coordinated by endocrine system, consisting of insulin (which suppresses all 3 metabolic system) balanced by a set of counter regulatory hormones (cortisol, glucagon, epinephrine and growth hormone) that activates one or more of the three metabolic systems (Table.1) .

Normal BSL depends upon:

- Intact endocrine system.
- Normal enzymes for glycogenolysis, glycogen synthesis, glycolysis, gluconeogenesis, and protein and fat metabolism.
- Adequate supply of fat, glycogen, and protein.

Table 1. Hormonal regulation of fasting metabolic system

	Glycogenolysis	Gluconeogesis	Lipolysis	Ketogenesis
Insulin	-	-	-	-
Glucagon	+	+		
Epinephrine	+		+	+
Cortisol		+		
Growth hormone			+	

Eatiology

Overutilization of glucose

a) Invariably results from hyperinsulinemia. Possible causes include maternal

diabetes in pregnancy, persistent hyperinsulinemic hypoglycemia of infancy, insulin- producing tumors, and child abuse.

b) Metabolic disorders:

- Fatty acid oxidation defects
- Amino acidopathies (Maple Syrup urine disease)
- Organic acidemias
- Classical Galactosemia with liver failure

Disorders of glucose underproduction

a) Inadequate glucose stores are associated with:

- Prematurity
- Small for gestational age (SGA)
- Malnutrition
- Ketotic hypoglycemia

b) Disorder of hepatic glucose production through various defects:

I. Disorders of gluconeogenesis: inhibition or decreased glucose production

- Fructose1,6diphosphatase deficiency
- Hereditary fructose intolerance
- Rare causes: PEPCK deficiency (phosphoenolpyruvatecarboxykinase deficiency) and PC deficiency (pyruvate carboxylase deficiency). These are associated with neurodegeneration and lactic acidosis.
- Glycerokinase deficiency
- Metabolic disorders causing liver failure: Tyrosinemia type I, Classical galactosemia, mitochondrial disorders, GSD IV

II. Disorders of glycogenolysis:blockage of glucose release

- Glycogen synthase deficiency (GSD type 0)
- Glycogen storage disorder types, I, III, VI, IX, and XI

Hormonal abnormalities

Panhaypopitutarism, growthhormone deficiency and cortisol deficiency.

Toxins & other illnesses

e.g Malaria; Ethanol, salicylates, propranolol.

Clinical Clues

- Hyperinsulinism is an important cause of hypoglycaemia under two years old, characterized by absence of ketonuria.
- "Accelerated starvation" (ketotic hypoglycaemia) classically presents between 18 months and 5 years, and generally remits spontaneously before 8 or 9 years of age. A presumptive diagnosis is made by documenting a low blood sugar in association with ketonuria and/or ketonaemia. Definitive diagnosis requires exclusion of other metabolic and endocrine causes.
- Always check the blood sugar in an acutely unwell child.

- Signs of hypoglycemia in infants can be non-specific.
- Hypoglycemia may be an early manifestation of other serious disorders (sepsis, RDS, congenital heart disease, Brain hemorrhage.)

Assessment

Clinical features can be divided into:

Autonomic features (warning signs)

- sweating, hunger, tingling around the mouth.
- tremor, tachycardia, pallor, palpitations and anxiety.
- These warning signs may be lost in patients with repeated or prolonged hypoglycemia.

Neurological features

- Lethargy, tiredness, change in behavior.
- Headache, visual disturbance, slurred speech, dizziness.
- Altered level of conscious, coma, convulsions.

Approach to hypoglycemia in non-diabetic patients

Past history

- Neonatal history of hypoglycemia.
- Episodes suggestive of hypoglycemia e.g. undiagnosed seizure disorder.

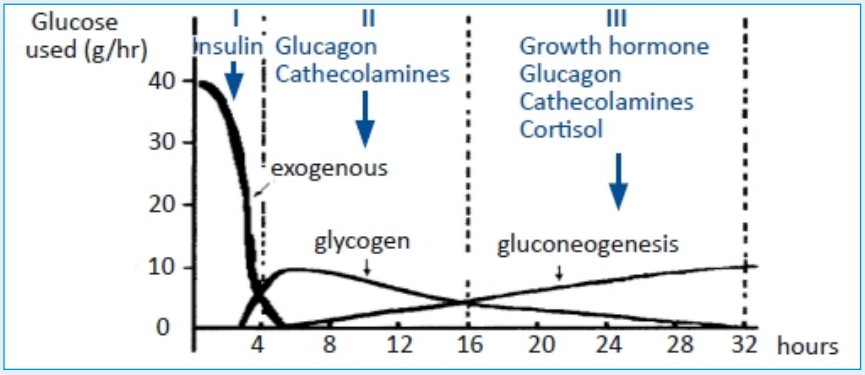
Age of onset

- Neonatal period and first two years: inborn errors of metabolism and congenital hormone deficiencies.
- After one year of age: ketotic hypoglycemia, isolated growth hormone deficiency, and cortisol deficiency.
- Toddlers and young children: also consider accidental ingestion of alcohol, oral hypoglycemic agents, aspirin, beta blockers, or toxins.

Fasting tolerance

When does hypoglycemia occur in relation to last meal?

Figure 1. Glucose homeostasis time course



Relation to food

- Milk products (galactosemia)
- Fructose e.g. juices (hereditary fructose intolerance)
- Protein (amino acid or organic acid disorders)

Family history

- unexplained infant deaths (may be from inborn errors of metabolism)
- Hormonal deficiencies and hyperinsulinism may run in families

Physical examination

- Weight and Height
  - o Short stature(hypopituitarism or growth hormone deficiency).
  - o Failure to thrive (disorders of amino acid, organic acid, and carbohydrate metabolism).
- Midline facial defects (eg, a single central incisor, optic nerve hypoplasia, cleft lip or palate) and microphallus or undescended testicles in boys may indicate hypopituitarism and/or growth hormone deficiency.
- Macrosomia, hepatosplenomegaly, and umbilical hernia (Beckwith-Wiedemann syndrome).
- Hepatosplenomegaly and hypotonia (glycogen storage disease, defects in gluconeogenesis, galactosemia, or hereditary fructose intolerance.)
- Hyperventilation (metabolic acidosis from an inborn error of metabolism).
- Hyperpigmentation, especially dorsal aspect of interphalangeal joints, areolae, genitals (adrenal insufficiency).

Investigations

Critical Blood samples (Table.2)

Should only be taken during hypoglycaemia (BSL <2.6mmol/l).

Ask about hypoglycemia critical sample package.

Table 2. Critical blood sample

Test	Type of Tube	Amount of blood	Special precautions
Glucose and lactate	Fluoride oxalate (grey top)	**1ml***	Put tube on ice
Insulin Ketones (beta-hydroxybutarate) Cortisol Growth hormone	Plain tube (red top)	**5 ml***	
Free fatty acid			
C-peptide			
Ammonia	EDTA	**1 ml***	Put tube on ice
Plasma amino acid	Heparinized (purple top)	**3 ml***	
Acid-base	Capillary sample		
Acyl carnitine profile	Blood drops onto a filter paper test card		

*according to Hormonal Lab Sabah Hospital, minimum total volume of blood required is 10 ml

Urine (first voided urine)

1. Ward test for ketones, glucose, reducing substances
2. 10-20ml saved if indicated for amino acids and organic acids, and toxicology.

Other tests can be collected after the acute management of hypoglycemia include:

1. Liver function and electrolytes.
2. CK, uric acid, lipid profile.
3. Others if required e.g. Toxicology studies (Salicylates, ethanol, sulfonylurea)

If the critical samples are not available at the time of initial presentation, hypoglycemia must be reproduced using a closely monitored fast which should be conducted in a centre that can respond quickly and appropriately if significant hypoglycemia consequences develop. Conservative recommendations for the maximum length of fast depends on the age of the child and are as follows:

<6 months	8hrs
6-8 months	12 hrs
8-12 months	16 hrs
1-2 yrs	18 hrs
2-7 yrs	20 hrs
> 7 yrs	24 hrs

Interpretation of critical sample

- Metabolically, free fatty acid (FFA) levels should increase to > 0.5mmol/L and beta-hydroxybutarate levels > 1 mmol/L. Failure of both to increase suggest hyperinsulinemia.
- Increase in FFA >3 mmol/L without increase in beta-hydroxybutarate levels suggest FFA oxidation defect.
- High plasma lactate levels suggest gluconeogenesis, glycogenolysis, or respiratory-chain defects.
- Plasma cortisol level should be increases(>550 nmol/L) and growth hormones level should be increased (>6mcg/L). (Fig.2)

Further information on determining the cause of hypoglycemia are listed in table 3

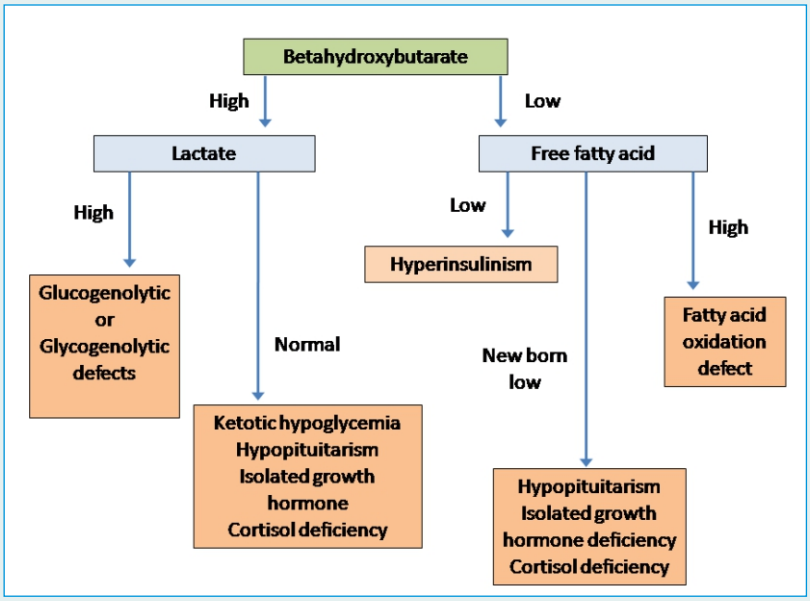


Figure 2. Interpretation of the critical sample

Management

- If the patient is fully conscious and able to drink and swallow safely, a rapidly-absorbed carbohydrate (eg, glucose tablets, glucose gel, table sugar, or fruit juice) should be given by mouth. If hypoglycemia does not improve within 10-15 minutes, parenteral glucose must be administered.
- Infants and children with altered consciousness and/or are unable to safely swallow a rapidly-absorbed carbohydrate should be treated with intravenous (IV) dextrose.
- Initial bolus of dextrose 2.5 mL/kg of dextrose 10% (0.25 grams/kg of body weight) administered slowly (2 to 3 mL/min) to avoid acute hyperglycemia.
- Don't use dextrose concentrations above 10% for acute management.
- After IV bolus, commence infusion at 3-5ml/kg/hr of 10% dextrose (6-8mg/kg/min) to maintain BGL above 4.0mmol/l
- Aim for blood glucose level 4.0-8.0mmol/l
- Glucose requirement >12 mg/kg/min indicates hyperinsulinism.

Rate of infusion (mg/kg per min) = (Percent dextrose in solution x 10 x rate of infusion [mL per hr]) ÷ (60 x weight [kg])

Checking blood glucose level (BSL)

- After 30 min initially and then every 60 minutes and the dextrose infusion adjusted accordingly until BSL > 5 mmol/l in two consecutive hours.
- Then Check BSL every 2-4 hrs.
- All patients with hypoglycemia of unknown cause require admission.
- Consult endocrinology and metabolic for further management of the patient.

Note:

- hypoglycemia due to metabolic disorders is easily corrected with IV glucosebut may recur if the underlying metabolic defect is not treated.
- in contrast, hypoglycemia due to endocrine disorders especially hyperinsulinism is persistent and difficult to control requiring other medications . (Table 4)

Table 3. Determining the cause of hypoglycemia

Ketone level	Timing of hypoglycemia	Other clues	Diagnosis
Hypoketotic	No specific timing	↑Insulin (glucose <2.6 mmol/l), ↑ammonia in HIHA	Hperinsulinism
Ketotic	No specific timing	↓GH, ↓ cortisol, ↓TSH, micropenis, midline defect	Hypopituitarism
Hypoketotic	Infant: <3 hrs	Permanent hepatomegaly, ↑↑lactate, ↑ uric acid, ↑ TG, ↑ cholesterol, ↑ALT, ↑AST	GSD type I
Ketotic	3-8 hrs	Hepatomegaly, ↑ lactate, ↑ uric acid, ↑ TG, ↑cholesterol, ↑ ALT, AST,↑ CK (some GSD III) ↑ urine tetraglucoside, (hypoglycemia usually mild compared to GSD I)	GSD III, VI,IX
Ketotic	>8hrs	Hepatomegaly in acute phase, ↑ lactate, ↑ urine glycerol/ 2-ketoglutarate	Gluconeogenesis defects
Hypoketotic	Infant: > 8hrs older children: > 16hrs	Hepatomegaly during acute phase, mild ↑NH3, mild ↑ lactate, mild ↑AST/ALT, abnormal acylecarnitine profile, ↓free fatty acid, urine organic acid- dicarboxyluria	Fatty acid oxidation disorders
Ketotic	1-2 hr	Jaundice, liver dysfunction , GI symptoms, positive urine reducing sugar, galactosemia: ↑ Gal-1-P-uridyl transferase (GALT), ↓ Gal-1-P	Sugar intolerance (Galactosemia, Fructosemia)
Ketotic	3-8 hr	No hepatomegaly; Fasting:↓ glucose, ↓ lactate, ↓alanine, ↑ ketones Postprandial: hyperglycemia, glucosuria, ↓ ketones, ↑ lactate, mild ↑ AST/ALT; Liver biopsy: absent glycogen	GSD 0
Ketotic	During crisis	Metabolic acidosis, ↑lactate, abnormal acylcarnitine and urine organic acid. Ketolytic defects: urine ketones persistently positive when patient is well	Organic acidemias / ketolytic defects

Abbreviations: HIHA, Hyperinsulinism-hyperamoniaemia syndrome; GSD, Glycogen storage disease; GH, Growth hormone; TSH, Thyroid stimulating hormone; TG, triglyceride; CK, creatine kinase; NH3 ammonia

Table 4. Drug therapy for hypoglycemia

Selective Drugs for Hypoglycemic Disorders

Intravenous Glucose Rescue doses
- Dextrose emergency bolus: IV push 0.2 g/kg (2 ml/kg dextrose 10%), followed by D10 continuous infusion 5ml/kg/hr
- If plasma glucose not corrected after 15 minutes, bolus 2ml/kg D10 and increase continuous infusion by 25 to 50%

Glucagon (Emergency Treatment Only in Case of Insulin-induced hypoglycemia)
- 1 mg intramuscular or intravenously
- Side effects: vomiting and rebound hyperglycemia

Diazoxide (Use in Hyperinsulinism or Sulfonylurea Overdose
- 5-15 mg/kg/day divided into 2 or 3 doses
- Start with maximum dose (15 mg/kg to test) then lower possible (responders usually require 10 mg/kg or less)
- Side effects: fluid and sodium retention, hypertrichosis

Octreotide
- Start at 2 to 5 mg/kg/day and increase to 20 mg/kg/day SC divided q6-8h
- Side effects: transient diarrhea, abdominal discomfort, gallstones, transient growth impairment

Cornstarch
- 1-2 g/kg/dose (freshly prepare each dose by suspending in cold sugar-containing liquid)
- Effect last 4 to 6rh
- Not well absorbed in infancy
- Side effect: diarrhea

Carnitine (For Plasma Membrane Carnitine Transporter Defect)
- 100 mg/kg/day divided into 3 or 4 doses
- Side effects: diarrhea and fishy body odor

IV, intravenous and SC, subcutaneous

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Inpatient Management of diagnosed and suspected 21 OH deficiency (CAH)

Pediatric Endocrine Division ,
Mubarak Al-Kabeer Hospital

Inpatient Management of diagnosed and suspected 21 OH deficiency
CAH Pediatric Endocrine Division , Mubarak Al-Kabeer Hospital

Consideration of CAH:

In the following circumstances :

- neonate with ambiguous genitalia
- high 17-OH-progesterone on newborn screening
- salt wasting crisis : low serum Na, High serum K

If any of the above , immediately consult a pediatric endocrinologist

Workup To Be Collected at Initial Assessment:

Blood Tests:

Serum glucose, electrolytes, renal function
Random cortisol
ACTH
17-OH-P
Androstenedione
Aldosterone
Plasma renin activity PRA
Karyotype AND FISH for Y
DNA sample for CYP21 mutation testing
ACTH stimulation

ACTH stimulation : if child had received hydrocortisone, hold HC for at least 12 hours then do test , OR do test while child has been switched to Dexamethasone

Ref : Endo text

Urine Tests:

Urine Electrolytes

Imaging:

US to assess the internal genitalia AND the adrenal glands

Treatment Consideration in Patients with CAH

Unstable Child

- Management should be according to the latest PALS guidelines published bythe AHA
- PLUS : IV hydrocortisone 5-10 mg/kg STAT

IF SUSPECT ADRENAL INSUFF DO ACTH STIMULATION

I FEEL : in OPD - N patients so I can depend on early am cortisol , if inpatient - I don't know how stress they r or sick , here I might go directly to ACTH stimulation

He does not take aldosterone - as it's difficult to measure and unreliable - so PRA better

Stable Child

Treatment Components :

1- Glucocorticoids GC

2- Mineralocorticoids MC

3- others

UK people : not like canada
The highest dose in the morning (50-60%) if the total as am dose
The younger age group QID

1- Glucocorticoids GC:

Optimal Dosing

- DO NOT USE SUSPENSION , use crushed tablets PO
- Dose: 10-15 mg/m2/day divided three times daily (in infancy the dose can reach up to 25 mg/m2/day)

- Preferred steroids:

Infancy and childhood - **Hydrocortisone**

Older children at or near completion of linear growth - Prednisone or Prednisilone (2-4 mg/m2/day divided twice daily) OR Dexamethasone 0.25-0.375 mg/m2/day once daily

Monitoring while on GC

BP

Weight

Labs like glucose

2- Mineralocorticoids MC:

Fludrocortisone

Dosing:

- in early infancy 0.05 - 0.3 mg/day
- maintenance dose 0.05 - 0.2 mg/day

Monitoring while on MC:

BP

PRA

Electrolytes

3-Others:

Sodium chloride Supplementation

Used in Neonates and infants

Dosing:

1-3 g/day distributed in several feedings

(3% saline = 3 gm in 100 ml solution)

Discharge of A Newly Diagnosed Child with CAH

- Completed education
- taught stress dosing of GC and sick day management
- prescribed GC, MC, NaCl
- prescribed and taught emergency hydrocortisone IM use
- arrange follow up with endocrinology

Stress Dosing of GC:

1-Major illness, major trauma, major surgery
IV HC 100 mg/m2 at admission or oncall to OR
Then 25 mg/m2/dose Q6H
While on the above regimen HOLD Mineralocorticoids

2- Other circumstances to stress dose

Fever > 38.5
Respiratory symptoms
Minor procedures
Vomiting

Dose : x 2-3 maintenance dose used at home until back to normal
(if within 2 weeks the stress dosing was done- no need to taper down the dose, just directly go back to maintenance dose)

Other practical regimens used in hospital for stress dosing go GC

< 3 yrs - 25 mg then 25-30 mg/day divided into three times aday
3-12 50 mg then 50-60 mg/day divided into three times aday
Adolescence 100 mg then 100 mg/day divided into three times aday

WHILE IN HOSPITAL

Monitor the following :
Blood sugar
BP
Maintain IV saline and dextrose

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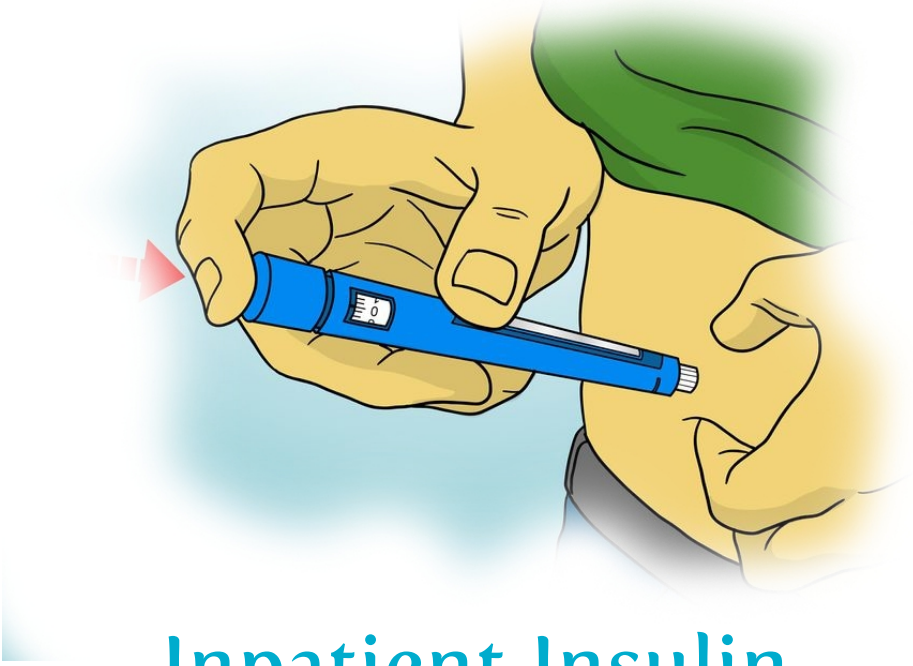
May, 2012
Approved by Dr Majedah AbdulRasoul and Dr Iman Busairi
Adapted from the Consensus Statement on 21-Hydroxylase Deficiency from The European Society for Paediatric Endocrinology and The Lawson Wilkins Pediatric
Endocrine Society. 2002



Inpatient Insulin Pump Guidelines

Mubarak Al-kabeer Hospital

Sick Day Rules & DKA On Insulin Pump
Pediatric Endocrine and Diabetes Unit

Risk Of DKA On Insulin Pump

People who use an insulin pump are at greater risk of DKA than people who give themselves injections. This is because pumps do not use long-acting insulin. Pumps use only rapid-acting (short-acting) insulin that starts working in 10 to 15 minutes and lasts 4 to 5 hours in most people.

Blood glucose levels can start rising very quickly and cause DKA if:

1) A blocked catheter:

- Is your pump delivering insulin? Check this by disconnecting the tubing from the infusion set. Then give a bolus and watch to see if a few drops of insulin appear at the end of the tubing
- make sure to change your catheter every two to three days.
- If possible, change your catheter before giving a bolus (meal or correction) and check your blood sugar 2 hours later to make sure it's working.
- never do your catheter change just before bed - if it blocks, you might not realize it until the next morning when it's too late.
- always check your blood sugars a minimum of 4 times per day!

2) Air in the line:

- check the tubing for air bubbles regularly.
- keep your opened insulin vial at room temperature; this will cause less bubble formation in the reservoir and tubing.
- make sure all connections are appropriately tightened.
- don't forget to PRIME or FILL CANULA after a new catheter insertion.

3) Illness:

- colds, fever, sore throat, gastro, etc. will often cause ketones.
- check for ketones early in order to get treatment started as soon as possible, even if your blood sugars are not elevated.
- broken bones, sprains, or other physical stressors can bring on ketones too.

4) Your insulin has degraded:

- it has passed it's expiry date.
- the bottle has been opened more than one month.
- it has been exposed to summer heat or winter cold.
- The insulin has been in the pump reservoir too long (ie more than a week)

5) Human error:

- forgetting to give your meal boluses!
- leaving the pump disconnected for too long without checking

Sick Day Rules-Insulin Pump

Blood glucose	Urinary ketones	Blood ketones	Action
Less than 5.5 mmol/l	Negative or trace	< 0.6 mmol/l	Treat low blood glucose if hypoglycemic. if experiencing recurrent hypos,set temp basal for 2 hrs (50% or -50%) . decrease bolus doses by 10-20%. may need to consider minidoses of glucagonIf BG4 mmol/l.
Less than 5.5 mmol/l	Positive : Small, moderate, large	0.6 - 3 mmol/l	Starvation ketosis. Encourage the child to drink sugar containing fluid, and 20 g CHO. Check blood glucose and ketones in 2 hrs. Repeat above steps again If ketones are less than 3 mmol/l If remain unchanged after 4 hrs ,go to A&E May need IV glucose if child cant eat or drink, as there is risk of DKA.
5.5-9.9 mmol/l	Negative or trace	<0.6mmol/l	Give the usual insulin covering carbs Test again in 2-4 hrs
5.5-9.9 mmol/l	Small-moerate-large	0.6-3 mmol/l	Starvation ketosis Extra CHO (20g) and clear sugar free fluids are needed. Give the usual insulin dose ,as programmed by pump calculator with BG and 20 g CHO. Test BG and ketones in 2 hrs.Repeat above steps again if ketones are less than 3 mmol/l And if remained unchanged after 4 hrs go to A&E
10-13.9 mmol/l	Negative or trace	< 0.6 mmol/l	Give the usual insulin by pump ,at usual time Recheck BG and ketones in 2 hrs
10-13.9 mmol/l	Small -moderate-large	0.6-3 mmol/l	" Give sugar free fluid . " Change infusion set.

			" Program pump calculator with BG and give correction bolus by pen. " Set increased temp basal for 2 hrs (+20-50%) " Check BG and ketones after 2 hrs . if ketones are less than 3 mmol/l ,and did not change,give another correction dose by pen may need to jncrease temp basal further if still BG and ketones unchanged then go to A&E.
Blood glucose	**Urinary ketones**	**Blood ketones**	**Action**
>14 mmol/l	Negative/ trace or Trace/small Small/moderate	0.6-3 mmol/l Blood ketones <0.6 mmol/l 0.6-0.9 mmol/l 1.0- 1.4 mmol/l	Check BG and ketones 1-2 hrly . Drink extra low carb fluid,(1-2 cups/ hr). Encourage exercise.if ketones negative or trace **INSULIN DOSE :** " Give correction insulin dose as calculatedby the pump. " Test BG in 2 hrs to make sure BG has dropped at least 50mg/dl or 3mmol/l " If after 2 hrs BG lowered, decide if another correction bolus is needed by the pump until BG reaches the target. B) If after 2 hrs from first correction dose by the pump the BG did not improve or at least drop by 50 mg/d or 3mmol/l ,then follow the steps below: **First:** calculate correction dose and give it by pen injection.(use your pump to calculate correction dose) **Next:** change the infusion set and reservoir. **Then**set increased TBR for 4 hrs (120%-150%). **Finally:** Recheck BGand ketones after 2 hrs, if no improvement,calculate correction dose and give as pen injection. oIf BG and ketones remain unchangedafter 4 hrs, go to A&E.
>14 mmol/l	Moderate - large	>1.5-3mmol/l	" Check BG and ketones 1-2 hrly

			" Drink extra low carb fluid,(1-2 cups/ hr). " discourage exercise. " Treat underlying sickness. " **INSULIN DOSE :** " **First:**Take correction dose by pen or syringe . " If ketones are moderate, give the correction bolus equal to 10% of TDD. " If ketones are large, give correction bolus equal to 20% of TDD . " Or by using the individual correction bolus using ISF or ascalculated by pump, and multiply the dose by 1.5.* " (TDD can be calculated from the pump ,either by adding both total basal and total boluses in the pump set, or taking the average of 3 days TDD). **Next: Disconnect the infusion set and site.** " program the correction you just gave (by syringe or pen) into the pump. " Let the insulin drip out (remember the pump is NOT connected to you). This is a "fake" bolus to enable the pump to keep track of insulin on board.**. " Then:Change the infusion set and site. " **Finally:** Program (120%-150%) temporary basal rate for 4 hours and re-evaluate both blood sugar and need for further adjustment. " If BG remain high, and ketones persist after 2 hrs,or developed nausea ,vomiting or abdominal pain , contact the diabetes pump team ,or go to A&E .

The Correction Formula For High Blood Sugar With Ketones is:

$$\frac{\text{Current blood glucose - Target blood glucose}}{\text{Insulin sensitivity factor}} \quad \text{XI 5}$$

" When you have ketones the correction formula us larger than the usual one.

Extra insulin is needed to clear the ketones

" This means you are giving 1.5 times the usual correction (50% more insulin) and sometimes you can give double the correction if the blood glucose and ketones didn't improve.

" Example:

If blood glucose is 20.4, and ketones ++ ,your ISF is 4, and target blood glucose is 6.

20.4 - 6 X 1.5 .4 units of extra insulin.

4

**The correction dose given ,should take into consideration the residual effect of previous meal or correction bolus given. A useful guide is to use unused bolus rule(around 30% of rapid acting insulin is absorbed each hour).the correction dose should be reduced accordingly.

e.g if 5 units had been given as meal bolus,2 hr back,then60%would have been used and remaining 40% or 2 units would still be active. This should be subtracted from any correction dose.

DKA And Insulin pump:

The use of insulin pump is contraindicated in DKA.The insulin pump must be stopped once the patient develop DKA,and the patient should be started on insulin IV infusion as per DKA protocol.

Once the patient is out of DKA ,and is ready to eat, in appropriate time ,the patient can go back on insulin pump and basal rate will be started, with the meal bolus given just before the food ,and then the insulin infusion can be stopped .

Use of Pumps in Inpatient Settings:

GUIDELINE

As statedin the American Diabetes Association's 2014 Standards of Medical Care and supported by others (64-66).

"Patientswho use CSII pump therapy in the outpatient setting can be candidates for diabetes self-management in the hospital, provided that they have the mental and physical capacity to do so. [The] availability of hospital personnel with expertise

in CSII therapy is essential. It is important that nursing personnel document basal rates and bolus doses on a regular basis (at least daily)."

1.0 Competency

Any patient who is admitted to hospital using an insulin pump must be assessed for theircompetency to use their device. If they can demonstrate their physical and mental competency to manage the device, the patient should be allowed to continue on their insulin pump.

On admission to hospital, either to a ward or Emergency Department, the patient mustdemonstrate to the satisfaction of the assessing health professional that they have the ability touse the management program of the device and understand how to modify the program. It isacknowledged that the assessing health professional may have no competency in the practical management of the insulin pump. The role of the health professional is to assess the competencyof the patient to use the insulin infusion pump.

This will involve asking the patient to demonstrate that they:

1.1 Can open the management menu of the device.

1.2 Are able to adjust the basal rate.

1.3 Are able to adjust the bolus dose.

1.4 Can re-site their pump cannula. This could involve discussing how it is done,rather than actually undertaking the activity at this initial assessment.

1.5 Can demonstrate technical competency regarding cannula sites / how theywould manage infusion line obstructions / site leaks.

1.6 Can undertake appropriate problem solving actions if BGLs are high or low.

1.7 Have adequate supplies of infusion sets, spare batteries and the insulin used inthe insulin pump available for the anticipated duration of the admission.

1.8 Have been performing regular blood glucose monitoring tests (for example, fourtests per day).

The diabetes educator or diabetes resource person for the hospital should be notified uponadmission of a patient with an insulin pump (Section 4). An urgent consultation should be obtained if there is a concern about competency of the patient to continue on pump therapy. Theresource person may be able to advise or rectify any issues or concerns, allowing the patient to continue on their insulin pump.

If the patient (or parent/guardian) cannot competently demonstrate or describe the actions above,the insulin pump should be discontinued. The patient should be placed on an IV insulin infusion orsubcutaneous insulin regime (eg, basal/bolus insulin regimen) during their hospitalisation.

2.0 Contra-indication

The use of the CSII is contra-indicated in situations where the patient's safety may becompromised by the physical illness or mental state of the patient.

Absolute contra-indications for CSII using an insulin pump are:-

2.1 Patients with an impaired level of consciousness.

Note: insulin pump therapy can be continued during anaesthesia (with decreased levelof consciousness) as long as the anaesthetist is aware of and willing to manage thepump during anaesthesia.

2.2 Patients with critical illness requiring intensive care.

2.3 Patients with major psychiatric disturbance.

2.4 Diabetic ketoacidosis.

2.5 Patients refusing or unwilling to participate in self care.

2.6 Lack of infusion sets, spare batteries and other equipment required to maintain patienton CSII therapy.

2.7 Any other medical circumstance deemed unsuitable by the supervising medical officer.

If the patient presents with any of Items 2.1-2.6 the insulin pump must be discontinued and thedevice managed according to the hospital's policy for storage of patient valuables. When storingthe pump be sure to remove the battery from the device to prevent ongoing pump alarms. The

patient should be placed on an IV insulin infusion or subcutaneous insulin regime (eg, basal/bolusinsulin regimen) during their hospitalisation.

In the case of Item 2.7, an urgent discussion of the patient's condition and management with thediabetes specialist, diabetes educator or diabetes resource person for that hospital should beconsidered.

3.0 Documentation

Before a patient continues on CSII as an in-patient, the following criteria must be documented:

3.1 It must be clearly written in the medical record and on the blood glucose monitoring formthat the patient is on an insulin pump.

3.2 The brand name and model of the pump must be written in the medical record.

3.3 The type of insulin used in the insulin pump must be identified and recorded in theblood glucose monitoring form.

3.4 The current basal and bolus insulin doses must be documented in the medical record.

Ideally the pump data would be downloaded and the print out stored in the medical chartfor reference.

3.5 That competency has been assessed and deemed satisfactory, as per Section 1.

3.6 The patient agrees to notify the medical staff of any changes they make to their insulinpump.

4.0 Consultations

The following health professionals should be consulted.

4.1 Endocrinologist or Physician with interest in diabetes.

4.2 Diabetes Educator or diabetes resource person trained in insulin pump management.

4.3 Dietitian.

5.0 Insulin Adjustment

5.1 Changes to the patient's insulin therapy may be made at anytime by the patientprovided the change is notified to the medical staff, as per Section 3.6.

5.2 Any change to the insulin regimen recommended by the medical staff will bedocumented in the medical record and confirmed by the patient at the time ofimplementation, as per Section 3.6.

6.0 Blood Glucose Monitoring

6.1 Patients on an insulin pump should perform a minimum of four blood glucose tests perday.

6.1.1 A minimum of four tests per day may be performed in patients with satisfactorycontrol. These should be performed before each main meal and before going tosleep at night.

6.1.2 In patients with less satisfactory control, six tests per day should be performed

(one test before and two hours after each of the three main meals).

6.1.3 An overnight test (eg, 02.00hrs) may be necessary.

6.2 Additional blood glucose levels may be undertaken at any time by the patient.

6.3 Additional tests may be performed at the request of the medical officer or nursing staffwhen clinically indicated.

6.4 The number of tests performed each day can only be reduced on the orders of the medical officer and can NEVER be reduced to less than four tests per day (6.1.1).

7.0 Device management

7.1 The patient or guardian is responsible for ensuring the correct operation of the insulinpump.

7.2 The patient or guardian will rotate the infusion set consistent with the recommendationsfor the device. This will be every three days, unless other documentation is provided.

7.3 The patient or guardian will make the adjustments to the insulin pump program.

7.4 The patient or guardian will be responsible for all bolus dose administration.

If the patient is not capable of undertaking these actions, the insulin pump must be discontinuedas per Section 1.

8.0 Operations and Procedures.

The use of the CSII in operating theatres, procedure rooms, etc is not contra-indicated. Its usemust be considered carefully in consultation between the anaesthetist, surgeon, physician,diabetes educator and patient.

Potentially the insulin pump, by delivering stable and consistent

insulin administration over hours can provide excellent peri-operative blood glucose control.

In thebasal infusion mode only, it can be considered "equivalent" to very long acting insulin.

As with all patients with diabetes undergoing surgery, patients who are unconscious need to bemonitored carefully during and after their surgical procedure. Their blood glucose should bemeasured frequently while their conscious state is impaired.

8.1 Patients continuing on CSII peri-operatively

8.1.1 The patient (or parent/guardian) must consent to continuing on insulin pump therapyperi-operatively.

8.1.2 CSII and IV insulin therapy should not run at the same time.

8.1.3 The infusion site must be placed away from the operation site with consideration alsogiven to where a diathermy pad may be placed. Ensure the insertion cannula isplastic not metal. If the pump is to be used during surgery, the patient must replacemetal cannulas with plastic insertion cannulas before any surgical procedures thatmay involve diathermy are performed.

8.1.4 An identification tag must be attached to the patient that states that the patient isusing an insulin pump. This should be sited in a readily visible position appropriatefor the procedure to be undertaken.

8.1.5 Theanaesthetist must have access to the insulin pump during surgery to enable it tobe turned off or disconnected, if necessary.

8.1.6 The patient's BGLs must be monitored every hour peri-operatively until they havesatisfactorily regained consciousness and the patient (or parent/guardian) is capableof making decisions regarding managing their insulin pump.

8.1.7 In the event of the blood glucose levels increasing to an unsatisfactory level perioperatively,

an IV insulin infusion should be commenced and the insulin pump turned

off, or disconnected.

8.1.8 In the event of the BGL levels falling below 4mmol/l peri-operatively, the insulinpump must be turned off and/or disconnected. The hypoglycaemia should be treatedwith IV glucose.

Once euglycaemia is restored, there are three choices regardingrecommencementof the CSII:

1. Recommence the pump at a lower insulin infusion rate (if the medical staff is ableto program the device). Consideration should be given to using a temporarybasal rate rather than adjusting the usual basal rate. This offers

more flexibilityand may avoid confusion if basal settings are not restored to usual when thepatient has recovered.

2. Recommence the pump at the usual basal rate with a higher IV glucose infusionrate to prevent further episodes of hypoglycaemia.

3. Leave the pump off and commence an IV insulin infusion to control the patient'sBGLs.

8.1.9 The use of CSII in major procedures should be only considered in rare circumstances due to the strong probability that an adjustment to the patient's insulintherapy will be required during the prolonged peri-operative period.

Discontinuation of the insulin pump and commencement of IV insulin therapy isrecommended in this situation.

8.2 Patients NOT continuing on CSII peri-operatively :

8.2.1 Patients whose insulin pump is discontinued prior to surgery will require either anintravenous insulin infusion or subcutaneous therapy according to the hospital's perioperativetype 1 diabetes management guidelines. Discontinuation of the insulinpump for even short periods of time with no alternative source of insulin will result inthe rapid development of hyperglycaemia.

8.2.2 The CSII can be re-commenced when (a) the patient has regained full consciousness and (b) it is considered medically appropriate.

9.0 Other circumstances

9.1 The insulin pump may need to be discontinued temporarily during a number ofcircumstances during hospitalisation. In this situation, discontinuation of the insulinpump for more than 30 minutes may result in significant hyperglycaemia.

Such circumstances where the insulin pump needs to be temporarily disconnectedinclude:-

- Any radiological investigation (pump must be removed)
- CT Scan (pump must be removed)
- MRI scan (pump must be removed, including metal cannula)
- Physiotherapy (depending on the therapy)
- Hydrotherapy (even if the pump is labeled as water-proof)

9.2 Patients whose insulin pump needs to be discontinued for longer than one hour mayneed to be considered for an injection of subcutaneous insulin, eg subcutaneoussoluble insulin (Actrapid, Humulin R, Humalog, Novorapid or Apidra) to cover theirshort term requirements.

Alternatively, the pump can be discontinued for up to twohours at the discretion of the treating doctor if the patient is clinically stable and blood glucose levels are being monitored regularly.

Upon recommencement of thepump, blood glucose should be rechecked and if needed a correction bolus can begiven.

9.3 Patients needing to be regularly disconnected from their insulin pump should beconsidered for basal/bolus subcutaneous insulin injection therapy.

10.0 Pediatric Population

10.1 The continuation of CSII in a child in hospital needs to be considered carefully inconsultation between the patient, their parent(s) or guardian and their medical team.

10.2 In the circumstances that the guardian or parent is responsible for the management ofthe insulin pump, the medical officer must be satisfied that the responsible person cansatisfy all the criteria as per Section 2. This decision may be made in consultation withdiabetologist,or a credentialed diabetes educator, when appropriate.

10.3 Additionally, the parent or guardian must be able to stay with the patient at all timesduring the admission so that adjustments to the insulin pump can be made at any time.

10.4 If the above conditions cannot be met, the insulin pump should be discontinued and subcutaneous insulin injection should be commenced.

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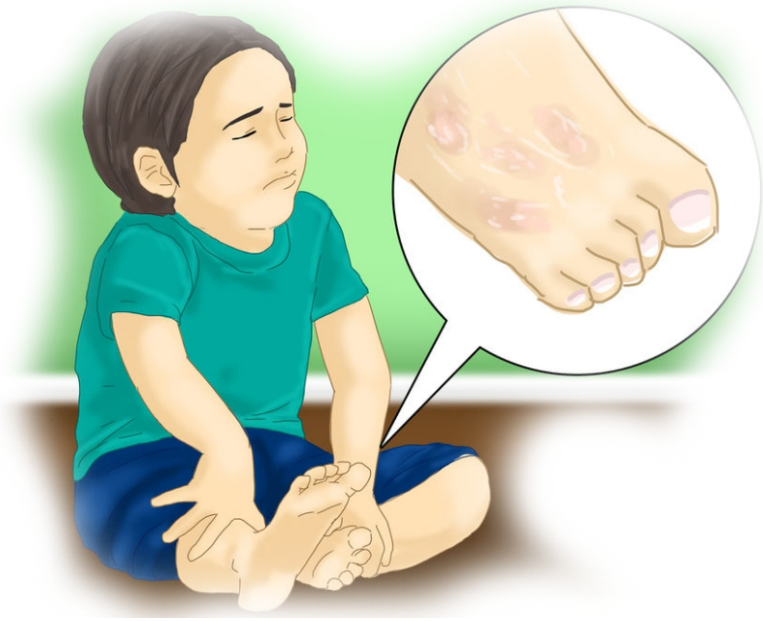

Guidelines to the Acute Limping Child

Rheumatology Unit
Mubarak Al-Kabeer Hospital

Guidelines to the acute limping child

1 Introduction

Limping following obvious trauma poses little diagnostic challenge and will not be dealt within this guideline.
Two common patterns of presentation exist, namely the **painful hip** and the **acutely swollen joint.**

It is important to remember a few general points when approaching the limping child:

- Limping may be due to pain referred from somewhere else (genital/spinal pain referring to the hip and pain from the hip referring to the thigh or knee).
- Certain causes of limping are age dependant.
- Can be caused by mild,self-limiting event or can be a sign of serious life-threatening condition

2 History and examination

The **history** should include questions about trauma, preceding infections or drug exposure, the presence of fever, rashes, features of systemic disease with joint involvement and the rate of onset of the problem.

On **examination** assess:

- Gait: Antalgic gait, running or jumping usually accentuates any pathological features.
- Standing: Back and spine (deformity and tenderness), pelvic tilt.
- Supine: examine each joint separately for tenderness, swelling, effusion, erythema, warmth and range of movement:
- Hip rotation is the most sensitive test and should be symmetrical if there is no pathology.
- Examine abdomen and groin and look for muscle atrophy, weakness and abnormal tendon reflexes.

2.1 Differential diagnosis

Common causes of limping in children		
All ages		
•Trauma (fracture, haemarthrosis, soft tissue) •Infection (septic arthritis, osteomyelitis, discitis) very important •Secondary to various viral illnesses •Tumor •Sickle cell disease •Serum sickness		
Toddler (1-3 years)	**Child (4-10 years)**	**Adolescent (11-16 years)**
•Transient synovitis •Toddler's fracture •Child abuse •Developmental dysplasia of the hip	•Transient synovitis •Juvenile arthritis (pauciarticular) •Perthes disease •Rheumatic fever	•Slipped upper femoral epiphysis •Overuse syndromes •Osteochondritis dissecans

•Juvenile arthritis (pauciarticular) •Neuromuscular disease •Haemophilia •Hennoch-Schoenlein purpura	•Haemophilia •Hennoch-Schoenlein purpura	

2.1.1 The painful hip

There are four common and important diagnoses to be aware of:

Septic Arthritis can destroy a joint within 24 hours. Diagnosis is by exclusion.
Important signs to watch for are:

- Marked pain/spasm
- High fever
- Systemic upset++
- Raised ESR >20 , CRP

Remember that not all of the features may be present and that the younger the child, the more subtle the presentation can be!

Transient synovitis is a relatively common problem, especially in children between the ages of 3 and 6 years old which is usually self-limiting within approximately one week. There is a higher incidence in boys than girls.

- Rapid onset of hip pain and limping in an otherwise well child
- +/- history of preceding viral illness
- Hip held in flexion and abduction, limitation of internal rotation
- Only mild reduction of hip movements

Perthe's disease (osteonecrosis of the femoral head) tends to be found in the age group of 4 to 12 years and is due to an avascular necrosis of the femoral head. Diagnosis is radiological but x-ray changes may be absent early in the illness.

- Boys : Girls = 4 :1
- 15% may be bilateral
- no systemic features

Slipped upper femoral epiphysis tends to occur in 10 – 15 year olds, often with body weight above the 90th centile. Boys are affected slightly more often than girls and nearly a quarter of patients have bilateral disease. There may or may not be a history of minimal trauma. Diagnosis is radiological.

- Boys : Girls = 2 : 1
- 25% may be bilateral
- AP views alone may miss subtle changes therefore bilateral 'frog view' is required

2.2 Management

Specific management depends on diagnosis.

Ensure adequate analgesia.

2.2.1 Discharge and Follow-up:

If no specific cause found, or suspected transient synovitis:

- Bed rest is important for children with transient synovitis.
- Analgesia; NSAID (eg ibuprofen,naproxen) +/- paracetamol,
- Return to hospital if febrile, unwell or getting worse
- Patients with prolonged symptoms can be referred to rheumatology clinic

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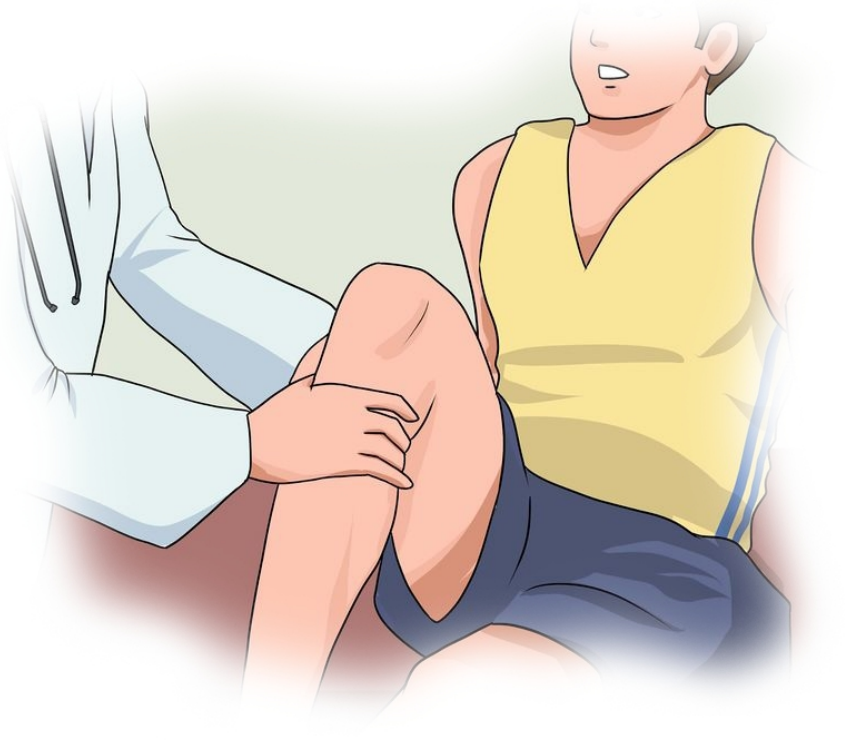

Guidelines to the Acutely Swollen Joint

Rheumatology Unit
Mubarak Al Kabir Hospital

1.1.1 Guidelines to the acutely swollen joint

The differential diagnosis of an acutely swollen and painful joint is large and initial investigation is aimed at excluding conditions which require urgent treatment.
If the diagnosis is still unclear after initial investigations it is essential to organize appropriate follow up.
In the vast majority of cases this will be at the pediatric rheumatology outpatient clinics at Sunday,Monday and Wednesday mornings.

The following table shows some common reasons for acute arthritis in children and specific management diagnostic issues.

Management of the painful joint

	Suggestive features	Investigations	Disposal
Septic arthritis/ osteomyelitis	"Fever, systemic upset, severe limitation of joint movement. "Beware of subtle presentation	" CBC, CRP, ESR " Ultrasound and guided joint aspiration may be possible " X-ray of the joint may show signs of osteomyelitis (late sign	" Needs urgent orthopaedic in-put " intravenous antibiotics
Joint trauma	"History of trauma	" X-ray	"Refer to fracture clinic
Irritable hip	"Systemically well	" Bloods as above " Ultrasound if easily available " +/- X-ray	" Follow up in Paediatric OPD clinic " Advise regular analgesia for 48 hrs
Hennoch-Schoenlein Purpura	"Purpuric rash in typical distribution "Abdominal pain "Haematuria	" Urine dipstick and microscopy " Blood pressure " Be aware of complications	" Paediatric referral and follow-up as "? consultations
Haemarthrosis	" If spontaneous or after minor injury consider haemophilia	" Coagulation studies	" Refer to hematologist if clotting abnormal
Rheumatic fever	" Carditis " Erythema marginatum " Migrating polyarthritis " Subcutaneous nodules " Chorea	" ECG/ECHO " Bloods as above +ASOT, DNase B	" Refer to Pediatric rheumatology
Serum sickness	"Chorea " History of medication, e.g. Cefaclor " Rash	" Bloods as above	" Follow up in OPD
Reactive arthritis	" History of recent viral illness " Well child	" Exclude septic arthritis (see above)	" Follow up in Paediatric rheumatology OPD

clinical features associated with joint pain/swelling tell you about the underlying diagnosis?

If sign/symptom present…	consider these disorders
Severe joint pain	Infection-related, malignancy, trauma, AVN, chronic regional pain syndrome (CRPS)
Pinpoint tenderness	Osteomyelitis, trauma, AVN, malignancy, enthesitis, CRMO
Night pain	Malignancy, osteoid osteoma, "growing pains"
Redness	Septic arthritis, acute rheumatic fever, reactive arthritis
Migratory joint pain	Leukemia, acute rheumatic fever
Not walking	Infection, malignancy, discitis, myositis, CRPS
Hip pain	Infection-related, AVN, SCFE, malignancy, chondrolysis, toxic synovitis, JIA (particularly enthesitis related arthritis)
Back pain	Usually benign, but consider bone or spinal cord tumour, discitis, spondylolysis/spondylolisthesis, JIA (especially enthesitis related arthritis), myositis, osteoporosis, fibromyalgia
Periarticular pain	Malignancy, hypermobility, CRPS, fibromyalgia
Dactylitis	JIA (particularly enthesitis related arthritis and psoriatic arthritis), sickle cell, trauma
Clubbing	CF, IBD, malignancy (especially lung), familial
Weight loss	Malignancy, connective tissue diseases, IBD
Muscle weakness	Myositis, malignancy, pain-related weakness
Rash	Connective tissue disease, vasculitis, JIA (particularly systemic arthritis, enthesitis related arthritis and psoriatic arthritis),

Oral ulcers	Vasculitis
Nail or nail fold changes	Connective tissue diseases, psoriasis, subacute bacterial endocarditis
Raynaud phenomenon	Connective tissue diseases, vascular obstruction
	Fibromyalgia, chronic fatigue
School withdrawal	Infection-related (e.g. tuberculosis, Lyme disease)
Consanguinity	Genetic or metabolic diseases

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Drug dosing in renal Impairment

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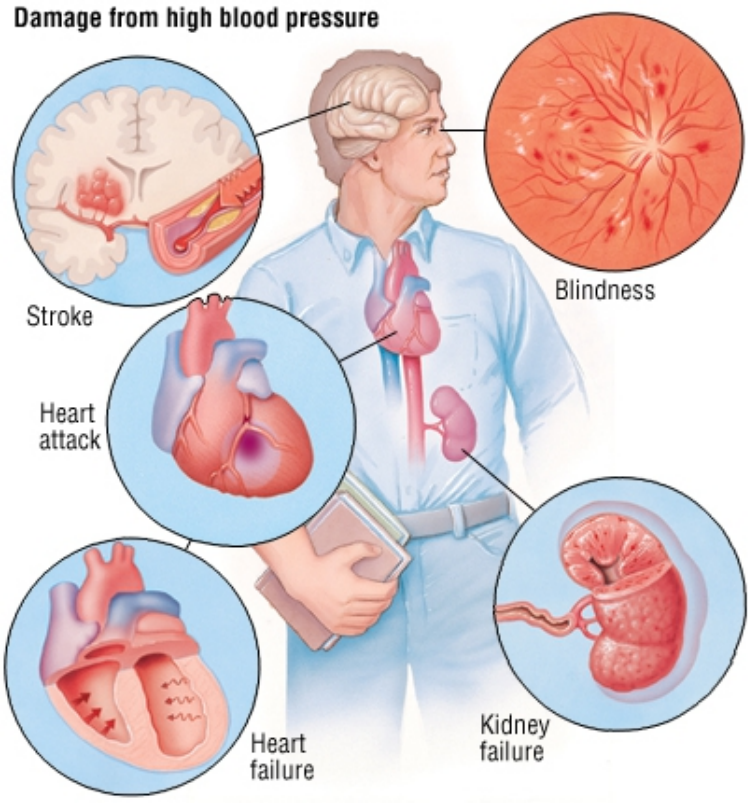
Drug Dosing in Renal Impairment

Some of the medications need dosing adjustment (frequency, dose or both) in patients with renal impairment. This depends mainly on drug that is renally excreted. In order to choose the appropriate dose, glomerular filtration rate (GFR) need to be known. Measuring GFR is not practical, so using modified Schwartz equation **{36.5 x height (cm) / serum creatinine (micromol/L)}** to estimate GFR is the practical way if your lab measure creatinine but does not measure cystatin C. The following are the common drugs that are used by pediatricians and need adjustments. You can refer to any drug formulary book or consult nephrology physicians or pharmacist for any other drug(s) adjustment.

Drug	GFR (ml/min/1.73m^2) 30-50	10-29	< 10
Acyclovir	10 mg/kg/dose q12h	10 mg/kg/dose q24h	5 mg/kg/dose q24h
Amikacin*	q12-18h*	q18-24h*	q48-72h*
Ampicillin	q6h	q 8-12h	q12h
Cefotaxime	q8-12h	q12 h	q24h
Ceftriaxone	100 %	100 %	100 %
Meropenem	20-40 mg/kg/dose q12h	10-20 mg/kg/dose q12h	10-20 mg/kg/dose q24h
Piperacillin-tazobactam	100 % q6h	75 % q8h	50% q8h
Teicoplanin	1-4 mg/kg/dose q24h	1-4 mg/kg/dose q24h	1mg/kg/dose q24h
Vancomycin*	10 mg/kg/dose q12h*	10 mg/kg/dose q18-24h*	10 mg/kg/dose q24-96h*

* Dose should be guided by trough level.
* Notes:
- Abbreviations: (q: every; h: hours)
- q refer to frequency of dosing & % refer to percentage of regular dose



Management Of Hypertensive Crises Guidelines

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Hypertension Systolic and/or diastolic blood pressure (BP) ?95th percentile measured upon three or more occasions.

Divided into the two stages:

~ **Stage 1 hypertension -** Systolic and/or diastolic BP between the 95th percentile and 5 mmHg above the 99th percentile.

~ **Stage 2 hypertension -** Systolic and/or diastolic BP ?99th percentile plus 5 mmHg.

Severity - Evaluation and treatment varies with severity of the hypertension.

Stage 2 needs immediate evaluation and pharmacologic treatment, while stage 1 hypertension allows more time for evaluation and nonpharmacologic therapy (unless the patient is symptomatic or has hypertensive target-organ damage).

Hypertensive emergency - An acute severe symptomatic high BP WITH evidence of potentially life-threatening symptoms or target organ damage. The absolute level of BP reading is less important than whether symptoms and/or target end organ damage is present. The most commonly involved organs are the brain, kidneys, eyes, and heart.

Hypertensive urgency - an acute severe elevation in BP WITHOUT severe symptoms or evidence of acute target organ damage

Approach

Start with confirmation of significant BP reading, using auscultation and appropriate cuff size and placement.(Look at choosing appropriate cuff) Quick history and physical examination that might reveal causes of hypertension that need different management like aortic coarctation, head trauma, intracranial mass, sympathomimetic drug overdose, and severe pain.

Physical examination:

Shouldfocus on airway, breathing, and circulation.

Four-extremity BP measurementand pulse palpation

Rapid assessment of severity of the disease (end organ damage symptoms/ signs)

Symptoms of target organ damage:

CNS: confusion, seizures, focal weakness

CVS: heart failure with tachypnea and pulmonary edema, S3 / S4 gallop

Renal:Flank pain, hematuria, proteinuria, oliguria

Visual: visual changes, papilledema, retinal hemorrhage, exudate

Investigation:

~ CBC , reticulocyte, and blood film (anemia and thrombocytopenia may indicate SLE, schistocyte indicate hemolysis)

~ Urea, creatinine, and electrolyte

~ CXR, ECG to assess cardiac hypertrophy and heart failure

~ Whenever possible obtain:

Cardiac echo

Renal U/S

Head CT if encephalopathy present

If physical examination andinvestigation resultsdemonstrated presence of end organ damage, the patient is classified as hypertensive emergency

If no evidence of end organ damage, the patient is classified as hypertensive urgency

Hypertensive emergency

Admit to ICU with BP and cardaic monitoring

Draw blood for the basic investigation

Obtain CXR and ECG

Involve pediatric nephrology service

Start IV antihypertensive medication

The aim is to lower blood pressure in a controlled fashion by no more than 25 percent over the first eight hours of treatment

this can be accomplished by one to two intravenous boluses of an antihypertensive medication (eg, labetalol 0.2 mg/kg per dose, hydralazine 0.2 mg/kg per dose) followed by a continuous infusion of labetalol (0.25 - 3.0 mg / kg / hr) .

The infusion rate should be titrated to maintain a blood pressure no more than 25 percent below the presenting value during the first eight hours of treatment and to prevent additional symptomatic blood pressure increases.

Avoid labetalol in pt with Asthma, Pulmonary edema, uncompensated heart failure.

If labetalol not available or contraindicated, use hydralazine boluses (0.2 mg /kg/dose Q 4-6 hours) to a maximum of 3.5 mg / kg/day

Further reduction to reach BP reading below the 95% BP should be done over the next 48 hours

Once initial control of severe hypertension is achieved, gradual conversion to oral antihypertensive medications should start.

Plan for further diagnostic evaluation should be arranged

Hypertensive Urgency

Admit patient to general ward

Take history and physical examination

No need for IV access

Draw blood for basic investigations

CXR

ECG

Start Oral antihypertensive medication, choices include :

Hydralazine: initial dose 0.75-1 mg/kg/day (max 5 mg /kg/day)

Captopril: initial dose 0.3-0.5 mg/kg/dose Q6-12 hr (maximum 6mg/kg/day)

Amlodipine : initial dose 0.1mg/kg/dose QD or BID (maximum dose 0.6mg/kg/day)

Further investigation should be arranged while patient in hospital

Treating the underlying cause is the main aim.

Neonatal hypertension

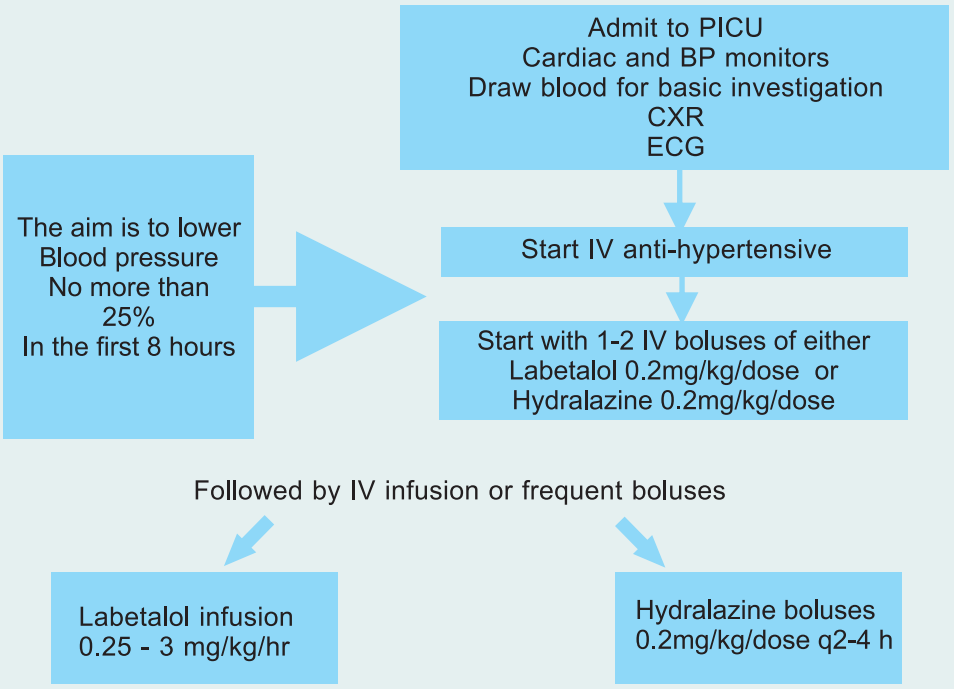
Limited experience about the drug of choice for this age group , but some studies suggest Nicardipine

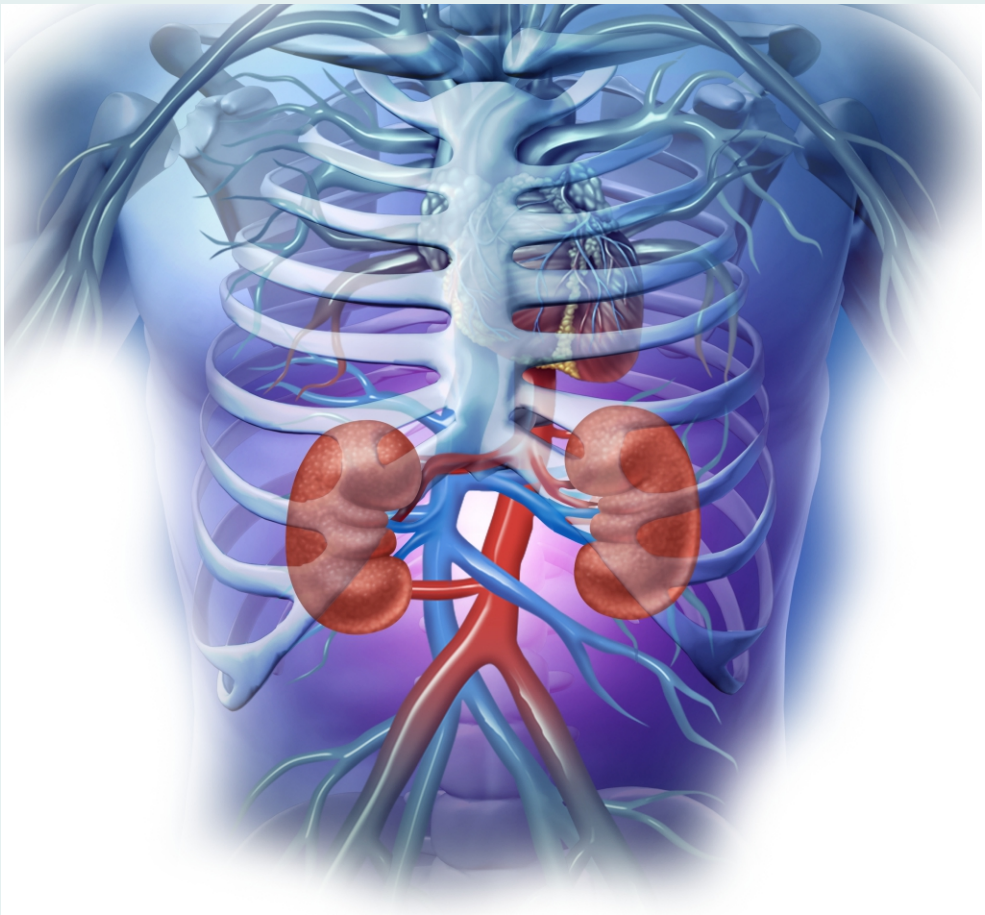
Nicardipine is reported as effective in reducing hypertension caused by many different etiologies in neonates, including renal artery thrombosis, coarctation of the aorta, bronchopulmonary dysplasia, polycystic kidney disease and renal vein thrombosis

(Nicardipine is not available in Kuwait)

Hypertensive emergency

Severe hypertension with evidence of end organ damage





Acute Kidney Injury (AKI) Protocol

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Acute Kidney Injury (AKI) Protocol

If a patient developed AKI (rise of creatinine >1.5 x baseline or urine output < 0.5 ml/kg/hr for > 6 hours), follow the following steps (see algorithm):

Treatment:

1. If evidence of intravascular depletion, flid resuscitation as per PALS (initial flid bolus with isotonic saline 20 ml/kg). If patient is euvolemic, give 1x maintenance flids (100 mls/kg/day). Close monitoring for evidence of flid overload.
2. No potassium or phosphorus supplement unless became low.
3. Manage electrolytes abnormalities (hyperkalemia, hyperphosphatemia, dysnatremia) and metabolic acidosis.
4. Treat other underlying causes (antibiotics for sepsis, relieve obstruction, stop nephrotoxic medications if possible).
5. Insure adequate protein (1.5-2.5 g/kg/day) and caloric intake (100-150% of healthy children requirement).
6. Adjust medication dosage as per current GFR (at least daily assessment of GFR).
7. Avoid further insults:
    a. Avoid intravascular depletion (dehydration) and hypotension
    b. Avoid nephrotoxic medications or medications that affect GFR if possible (NSAID, ACE-I/ARBs, Aminoglycosides, Vancomycin, ...etc)
    c. Monitor for levels if using nephrotoxic medications
    d. Avoid contrasts

Monitor:

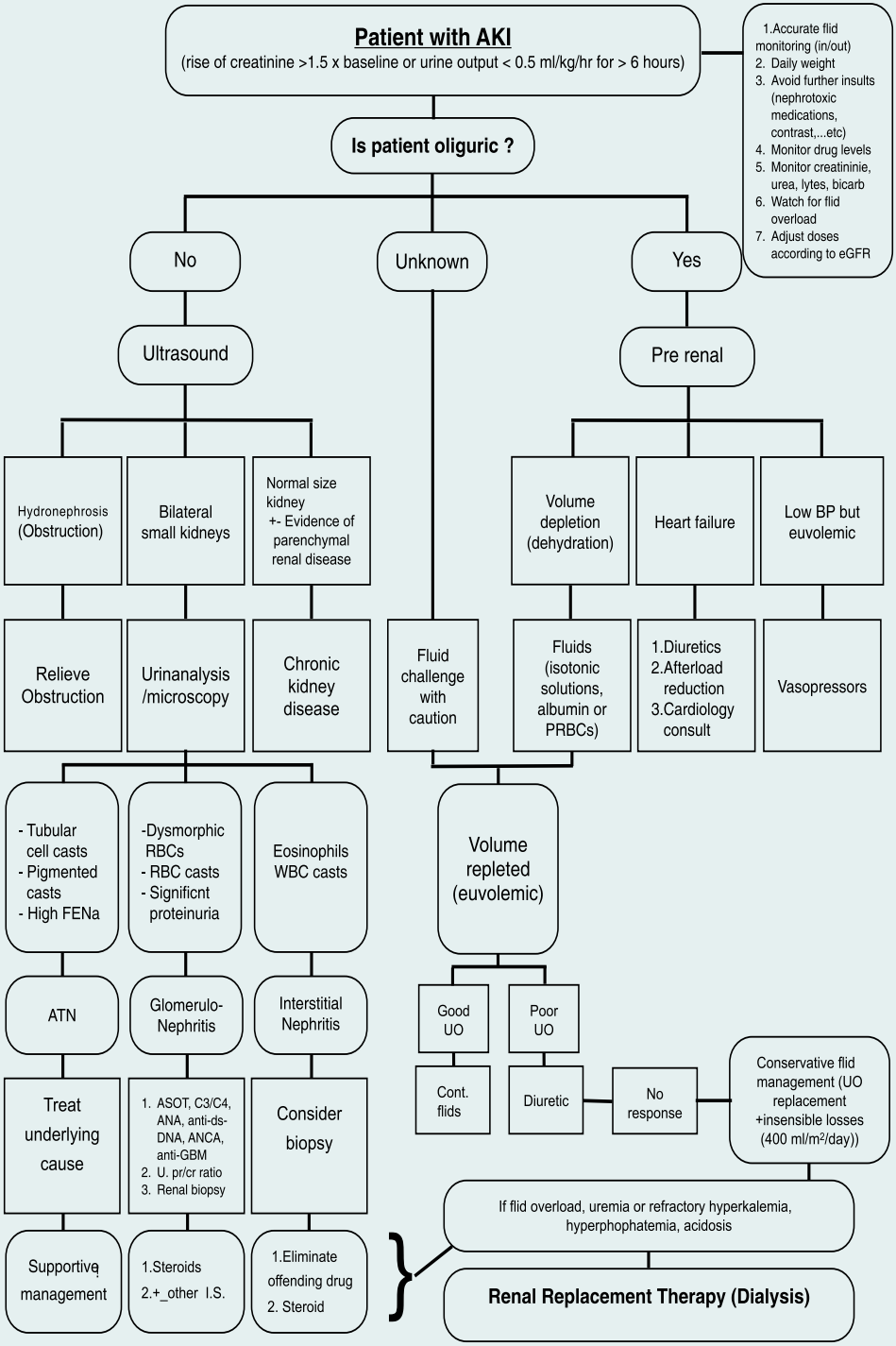
1. Accurate monitoring of all flid in/out (preferably to use foley catheter)
2. At least daily weight.
3. Monitor creatinine, urea, electrolytes/minerals, bicarbonate level at least daily.
4. Re-evaluate flid status (in/out/balance/weight, signs of edema, ...etc)

Investigations (do the following unless the cause of AKI is obvious):

1. Urine dipstick (hematuria, proteinuria). If proteinuria present, do urine protein/creatinine ratio.
2. Urine microscopy
3. Renal ultrasound
4. Liver enzymes (hepatorenal), CRP, CK (rhabdomyolysis).
5. If anemia and thrombocytopenia (HUS), do blood fim/LDH/bilirubin/retics/haptoglobin
6. If hematuria (RBC casts) and proteinuria, do glomerulonephritis work up (ASOT, C3/C4, ANA, Anti-ds-DNA, ANCA, anti-GBM, +- renal biopsy)

Complications:

Consider renal replacement therapy (dialysis) for refractory hyperkalemia, acidosis, pulmonary edema/flid overload, dysnatremia, uremia, hyperphosphatemia, or hypocalcemia to medical management.


Patient with AKI
(rise of creatinine >1.5 x baseline or urine output < 0.5 ml/kg/hr for > 6 hours)
1.Accurate flid monitoring (in/out)
2. Daily weight
3. Avoid further insults (nephrotoxic medications, contrast,...etc)
4. Monitor drug levels
5. Monitor creatininie, urea, lytes, bicarb
6. Watch for flid overload
7. Adjust doses according to eGFR
Is patient oliguric ?
No
Unknown
Yes
Ultrasound
Pre renal
Hydronephrosis (Obstruction)
Bilateral small kidneys
Normal size kidney +- Evidence of parenchymal renal disease
Volume depletion (dehydration)
Heart failure
Low BP but euvolemic
Relieve Obstruction
Urinanalysis /microscopy
Chronic kidney disease
Fluid challenge with caution
Fluids (isotonic solutions, albumin or PRBCs)
1.Diuretics 2.Afterload reduction 3.Cardiology consult
Vasopressors
- Tubular cell casts - Pigmented casts - High FENa
-Dysmorphic RBCs - RBC casts - Significnt proteinuria
Eosinophils WBC casts
Volume repleted (euvolemic)
ATN
Glomerulo-Nephritis
Interstitial Nephritis
Good UO
Poor UO
Treat underlying cause
1. ASOT, C3/C4, ANA, anti-ds-DNA, ANCA, anti-GBM
2. U. pr/cr ratio
3. Renal biopsy
Consider biopsy
Cont. flids
Diuretic
No response
Conservative flid management (UO replacement +insensible losses (400 ml/m2/day))
If flid overload, uremia or refractory hyperkalemia, hyperphophatemia, acidosis
Supportive management
1.Steroids 2.+_other I.S.
1.Eliminate offending drug 2. Steroid
Renal Replacement Therapy (Dialysis)

Guidelines for management of Upper Gastrointestinal (GI) Bleeding in Children

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Guidelines for management of upper gastrointestinal (GI) bleeding in children

Definition

Upper gastrointestinal bleeding is blood loss proximal to the suspensory muscle of the duodenum (ligament of Treitz) presents as

~ Hematemesis: vomiting of bright red blood

~ Coffee ground emesis: vomiting of acid denatured blood giving the coffee ground effect

~ Melena: passing black, tarry stools, due to bacterial oxidation of blood proximal to colon

Causes of Upper GI Bleeding

Some clinical signs associated with specific diseases

Newborn	
Non GI causes Swallowed maternal blood Hemorrhagic disease of the newborn Vascular anomaly Coagulopathy Milk protein sensitivity	**GI causes** Stress gastritis Peptic ulcer disease
Children	
Non GI causes Epistaxis Tonsillitis/sinusitis Drugs (e.g.NSAIDs) Tumors Hematologic disorders Munchausen by proxy Caustic ingestion Foreign body	**GI causes** Mallory-Weiss tear Esophagitis/Gastritis Gastric/duodenal ulcer, stress ulcers Dieulafoy's lesion Esophageal/Gastric varices Crohn disease Vascular anomaly ,telangiectasia vasculitis (HSP) Hemobilia (bleeding from the biliary system) Gastric volvulus/gastrointestinal duplications

Diagnosis

Some clinical signs associated with specific diseases

Disorder	Sign
Surgical abdomen	Borborygmi
Vasculitis (e.g. HSP)	Petechiae, purpura
Hemangioma, telangiectasia	Presence of extra intestinal haemangiomas
Esophageal varices	Caput medusae, spider angioma, jaundice, splenomegaly , low platelets
Epistaxis	Blood in hypopharynx
Genetic polyposis syndromes (e.g.Peutz-Jeghers syndrome)	Hyperpigmented lesions on gums and lips

Evaluation

Clinical

1. Vital signs: heart rate (HR), blood pressure (BP), respiratory rate
2. Cardiovascular stability: capillary refill, orthostatic changes (HR increased by 20/min, drop systolic BP >10 mmHg)
3. Level of consciousness

Laboratory

CBC, reticulocyte count, blood type, crossmatch& reserve blood
Coagulation profile, liver function tests, renal and chemistry panel

Management

This management protocol is intended to be used as guidelines, please contact your paediatric gastroenterologist to direct management toward specific patient needs

~ Manage airway breathing and circulation and stabilize patient
~ Fluid and blood resuscitation
~ Correct shock, anemia, coagulopathy andmetabolic/electrolyte abnormality
~ Nasogastric tube insertion and continuous drainage maybe useful to assess site of bleeding and whether it stopped (if patient have gastrostomy tube open tube for drainage).
~ Always keep patient NPO during bleeding

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Pharmacologic therapy

~ Proton pump inhibitors
Aim is to maintain gastric PH >6 and prevent re-bleeding
Omeprazole 2mg/kg/day (max 20mg IV twice a day)
~ Somatostatin/octeriotide
Reduces splanchnic & portal blood flow therefore it is indicated in known cases or highly suspected of portal hypertension with bleeding from esophageal varices
Somatostatin dose: 1mcg/kg IV bolus, followed by 2mcg/kg/hour continuous infusion

Upper Endoscopy or esophagogastroduodenoscopy (EGD)

~ Preferred diagnostic and therapeutic modality
~ Identifies mucosal lesions & source of bleeding
~ Control the bleeding

Arrange for EGD

~ Should be performed in hemodynamically stable patient therefore

1. Stabilize the patient
2. Order necessary investigations as per endoscopy protocol
3. Contact gastroenterologist on-call

Imaging modalities

Imaging should be carefully selected with the help of the on-call gastroenterologist to be booked for each individual patient according to clinical scenario

1. Plain X-ray chest/abdomen
2. Upper gastrointestinal contrast study (if cause of bleeding is suspected to be radiolucent foreign body)
3. Abdominal ultrasound to assess for portal hypertension
4. Nuclear medicine, Tc-99m-labeled erythrocyte scan can diagnose actively lower GI bleeding flow as low as 0.1 mL/min. but of limited value in upper GI bleeding
5. Angiography, detect active bleeding > or = 0.5 mL/min. used when endoscopic management can¡¦t control the bleeding and therapeutic coiling/embolization is indicated.

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Guidelines for management of
Acute Liver Failure in Children

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Guidelines for management of acute liver failure in children

Definition

Acute liver failure (ALF) in children is defined as a rare multisystem disorder in which severe impairment of liver function occurs in association with hepatocellular necrosis in a patient with no recognized underlying chronic liver disease, definition of liver dysfunction is

INR .1.5 with encephalopathy

OrINR .2 with or without encephalopathy (1)

ALF corresponds to a high mortality rate of up to 70% without liver transplantation.(2)

Clinical presentation

~ Flu-like prodromawith malaise, myalgia, nausea,vomiting, and jaundice.
~ Increased transaminases, hyperbilirubinemia, coagulopathy, hypoglycemia and encephalopathy.
~ Rapidly falling enzymes with worsening coagulopathy suggests exhaustion of hepatocytemass.

Poor prognostic markers(3)

~ INR > or = 4
~ Bilirubin> or = 235 ƒÊmol/L
~ Age <2 years
~ White blood cell count >9X109/L

Etiology and investigations (2,4)

The table below is meant to be used as a guideline,orderinginvestigations should be selective and directed bythe attending gastroenterologist / hepatologist

Category	Etiology	Investigations
Indeterminate etiology	Diagnosis of exclusion affect about 47% of children presenting with ALF(3)	
Toxins & medications	Acetaminophen, anticonvulsants,isoniazid, mushroom	History and drug level
Infections	Herpes simplex virus, EBV, enteroviruses, CMV, HHV-6, and parvovirus HAV, HBV, HCV, HEV, HDV	Viral PCR, blood / CSF cultures and IgM level
	Influenza virus	Nasal wash for influenza virus
Metabolic	Wilson disease	Ceruloplasmin&24h urinary copper

	Galactosemia	Urine reducing substances, GAL-1-PUT level
	Hereditary fructose intolerance	History and enzyme level
	Tyrosinemia	Urine succinyl acetone, Serum amino acid
	Urea cycle defect	Serum ammonia, urine orotic acid
	Fatty acid oxidation defects	Plasma acylcarnitine profile
	(FAO) Mitochondrial cytopathy	Muscle biopsy, skin fibroblast and liver biopsy for respiratory chain enzyme
	Neonatal hemochromatosis	Transferrin saturation & ferritin level
	Niemann-Pick disease type C	Bone marrow aspirate
	Congenital disorders of glycosylation	Transferrinisoeclectro phoresis
Immune	Giant cell hepatitis with coombspositive hemolytic anemia	Positive Coomb's test
	Autoimmune hepatitis	ANA, ASMA, LKM, IgG
Ischemic	Shock, Budd-Chiari syndrome, and congenital heart disease	Echocardiogram and ultrasound abdomen
Infiltrative	Leukemia, lymphoma and hemophagocyticlymphohistio-cytosis	CBC and blood film, Bone marrow aspirate

Management(2)

This management protocol is intended to be used as guidelines, please contact your

pediatric gastroenterologist to direct management toward specific patient needs

The goals in the evaluation of any child with ALF are

~ Determine etiology of ALF.

~ Assess the severity of liver failure & need for transfer to a liver transplant

center, liver transplantation is not available in Kuwait.

~ Provide hepatic support till spontaneous recovery or liver transplantation.
~ Anticipate & prevent complications.

General management

~ Nursing in a quite environment with head of the bed at 30..
~ Avoid stimulation, pain, and sedation. If patient needs sedation then electively intubate & ventilate.
~ Fluid restriction to 2/3rd maintenance.
~ Prevention of hypoglycaemia (maintain glucose levels >4.0 ƒÊmol/l).
~ Correct metabolic acidosis with maintaining intravascular volume &consider using bicarbonate.
~ Ranitidine 2-4 mg/kg/day IV q6-8h (maximum 200mg/day).
~ Prevention of sepsis using broad spectrum antibiotics & antifungal.
~ Keep protein intake tominimum of 1 g/kg/day via enteral routes.
~ Start lactulose-only if irregular bowel movement-to produce 2.3 stools per day.
~ Maintainplatelet count> 50 X 109/dL.
~ Anticipate and prevent renal failure by maintaining circulating volume with colloid or fresh frozen plasma.
~ N-acetylcysteine continuous infusion (100 mg/kg/24 hours) until INR < 1.4 was reported as safe and of some benefit in non-acetaminophen over dose ALF.(6).

Management of complications

Encephalopathy (2)

~ Monitor clinically for signs of encephalopathy.
~ Children with signs of agitation or Grade III or IV encephalopathy should be electively ventilated.

Cerebral edema (2)

~ Invasive intracranial pressure (ICP) monitoring is controversial
Maintain ICP < 20-25 mm Hg & cerebral perfusion pressure > 50 mm Hg.
~ Management of raised ICP
1 Mannitol, rapid bolus of 0.5 g/Kg as a 20% solution of mannitol over 15 minute. Target serum osmolarity= 320 mOsm/L.
2 Sodium thiopental (mannitol-resistant cerebral edema), bolus 2 - 4 mg/Kg over 15 minute followed by a slow intravenous infusion 1 - 2 mg/Kg/h.(7)

Coagulopathy(3,5)

~ INR is an indicator of the severity of the liver damage.
~ Risk of hemorrhage correlates with the severity of thrombocytopenia rather than coagulopathy, keep platelets > or = 50x109.

~ Correction of coagulopathy with fresh frozen plasma & activated factor VIIais indicated only if

~ Active bleeding

~ Prior to invasive procedures (e.g. insertion of central line, ICP monitor)

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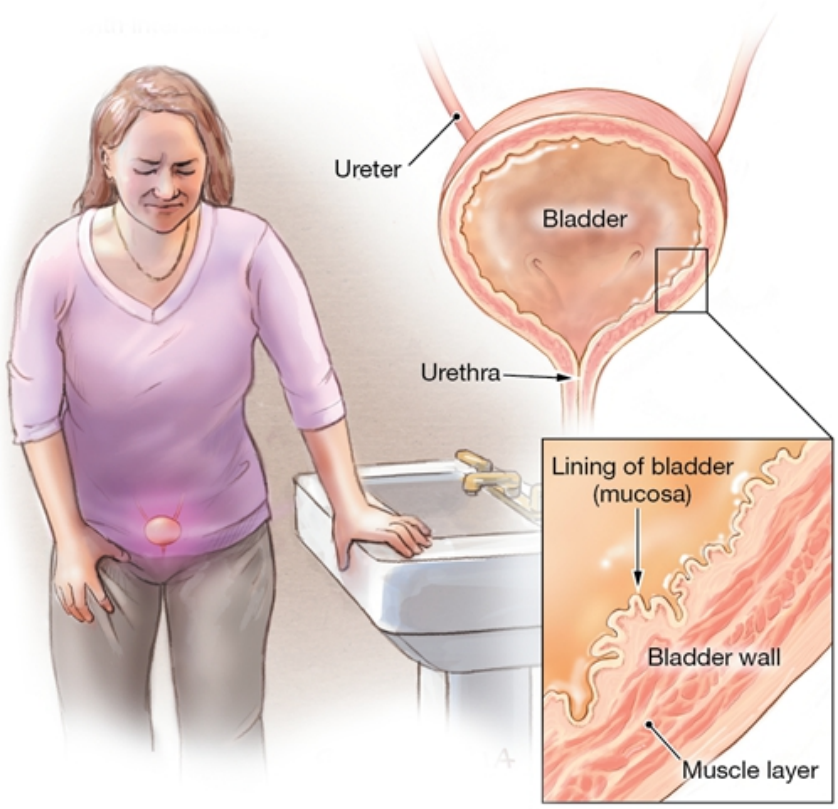

2012 Kuwait Guidelines for management of

Pediatric Urinary Tract Infection

Prepared by

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2012 Kuwait Guidelines for management of Pediatric Urinary Tract Infection

There have been major changes in the new guideline for the management of pediatric urinary tract infection especially those concerning the use of prophylactic antibiotics and the need for renal scan and MCUG after the first urinary tract infection. Our guideline is based on the AAP (American Academy of Pediatrics) and NICE (National institute for health and clinical excellence) guidelines with some modifications to suit the settings in Kuwait.

Clinical Manifestations and classification.

- ~ **Acute pyelonephritis**: Abdominal or flank pain, fever, Chills, vomiting, diarrhea. Some newborn and infants may show jaundice, poor feeding, irritability, and weight loss.
- ~ **Acute cystitis**: Dysuria, urgency, frequency, suprapubic pain, incontinence, malodorous urine, +/- fever.
- ~ **Asymptomatic bacteriuria (ABU)**: Individuals who have a positive urine culture without any manifestations of infection and occurs almost exclusively in girls.

Physical exam ¡V points to cover

Growth parameter
Temperature (Fever in pyelonephritis), always check blood pressure
Abdomen (kidney, bladder, stool)
Neurological exam
Spinal exam ¡V dimple, hair etc
External genitalia (External abnormalities, Sexual abuse)

Investigations:

~ CBC
~ ESR, CRP (if possible)
~ Urine Analysis:
~ Dip Stick
~ Leukocyte esterase test.
~ Nitrite test.
~ Microscopy:
~ WBC > 5 cells / HPF or 25 WBCs per £gL.
~ bacteriuria
~ Urine Culture:

Urinary Sampling Methods:

1. Midstream clean catch

a. Preferable method for toilet trained children.

2. Bladder catheterization:

a. Used for non toilet trained infants and children.
b. In sick infants and children who require urgent antibiotic use.

3. Suprapubic aspiration. Use only in limited circumstances

1. Labial adhesion
2. Tight foreskin
3. Anatomical abnormality

NB.

1) Bagged Urine is only useful for urine analysis and SHOULD NOT BE USED for urine culture. Bagged urine culture results are ONLY useful if NEGATIVE. Better results if perineum cleaned before bag placed and removed as soon as voids.
2) When urinary tract infection is suspected in a stable infant or non toilet trained child, in whom immediate antibiotic coverage is not required, Urine bag can be used initially for URINE ANALYSIS ONLY. If urine analysis suggests UTI then another urine sample by catheter should be obtained.
3) Colony Count: No definitive cut-off values of bacterial count exist. In most instances, an appropriate threshold to consider bacteriuria ¡§significant¡¨ in infants and children is the presence of at least 50 000 CFUs per ml of a single urinary pathogen. Colony count should be interpreted within the context of other symptoms and signs of urinary tract infection.
   i.e. a colony count of 50 000 CFU per ml in a febrile symptomatic child is significant whereas a higher colony count in a non febrile non symptomatic child could mean contamination or asymptomatic bacteriuria

Bacterial Pathogen of urinary tract infection.

The most common urinary pathogen is Escherichia coli, other bacteria are Klebsiella spp., Proteus, Staphylococcus saprophyticus, Entrococcus spp., Psuedomonas aeroginosa

Organisms NOT considered urinary pathogens in otherwise healthy children such as Lactobacillus spp, coagulase-negative staphylococci, and Corynebacterium spp

TREATMENT:

Since most oral antibiotics have the same efficacy as the intravenous ones, the indication for admission can be minimized to the following conditions:

Indication for admission:

~ Neonates and infants below 3 months of age. (3-6 months are preferably admitted but not a must).
~ Children of any age with high fever and / or flank pain, sepsis or shock.
~ Known complex underlying urological pathology.
~ Persistent vomiting, dehydration or inability to take oral medication
~ Known / suspected causative organism resistant to oral medication.
~ Psychosocial issues: inability of family to care for child appropriately.

Antibiotic Therapy

Bacterial antibiotic resistance patterns are geographically determined and should be reviewed at each hospital to determine the best initial oral antibiotics. Current

Note: Agents that are excreted in the urine but do not achieve therapeutic concentrations in the bloodstream, such as Nalidixic acid or Nitrofurantoin, should NOT be used to treat UTI in febrile infants and young children in whom renal involvement is likely.

Some antimicrobial for oral treatment of UTI

E-Coli is the main urinary pathogen. Since E-Coli has a high rate of resistance to Trimethoprim sulfamethoxazole (Septrin) and Cephalexin (keflex) 45% and 47 % respectively # . Therefore, it is **Not Recommended** to use those antibiotics

empirically unless the pathogen¡¦s sensitivity is known.
(Mubarak hospital Antibiogram 2010)

Antimicrobial	Dosage
Amoxicillin/clavulanate (Augmentin)	20-40 mg of amoxicillin / kg/day q 12 hrs
Cefprozil (Cefzil)	30 mg / kg/d in 2 doses
Cefuroxime axetil (Zinnat)	30 mg / kg/d in 2 doses
Cefpodixime (orelox)	10 mg / kg / d in 2 doses
Cefixime (suprax)	8mg / kg/d in 2 doses
Trimethoprimsulfamethoxazole (Septrin) *	6-12 mg TMP, 30-60 mg SMX/kg/d q12 hrs
Cephalexin (keflex) *	50-100 mg / kg/d in 4 doses
* Not recommended for empiric treatment	

Some antibiotics for parenteral treatment of UTI

Antibiotic	Daily Dosage
Ceftriaxone	75 mg/ kg every 24 hour
Cefotaxime	150 mg/kg/day divided every 6 h
Ceftazidime	150mg / kg/ day divided every 6 h
Gentamycim	7.5mg/kg/day divided every 8 h
Amikacin	15 mg/kg/day divided every 8 h
Ampicillin	100 mg / kg/ day divided by every 6 h

Intravenous antibiotics can be switched to oral antibiotics once the causative agent and the Antibiotic sensitivities were identified. The patient can be discharged home if:

~ > 2 months of age
~ Afebrile for > 24 hours
~ Tolerating oral fluids and medications.

Duration of therapy:

~ Seven to ten days treatment regimens are recommended for pyelonephritis. Longer duration up to 14 days might be necessary in some cases.
~ Three to five days treatment is usually adequate for simple lower urinary tract infection (cystitis) in older children.

There have been Major changes in the guidelines regarding prophylactic antibiotics and imaging studies after first urinary tract infection.

Prophylactic antibiotics:

~ No need to routinely start prophylactic antibiotics after the first UTI. If
~ Non complex urinary tract infection.
~ Normal ultrasound abdomen.
~ If prophylaxis to be used, the following antibiotics can be used. The dose

Prophylactic antibiotics	Dosage
Trimethoprimsulfamethoxazole	2 mg TMP QHS
Nitrofurantoin	1 mg/ kg dose QHS
Cephalexin (Keflex)	25mg/kg/dose QHS
Amoxicillin/clavulanate(Augmentin)	10 mg/kg of amoxicillin QHS
Cefuroxim axetil	7.5 mg/kg QHS

Follow up investigation:

1) Ultrasound abdomen to rule out major urinary tract structural pathology:

~ There has been no proof or strong evidence that medical or surgical intervention for vesicoureteric reflux, especially low grades I-III, changes the outcome and the long term prognosis of infants and young children with vesicoureteric reflux. Therefore, routine MCUG and radionuclide studies after the first urinary tract infection are no more recommended. This step will dramatically reduce unnecessary invasive investigations. It will also reduce the cost and the burden on radiology departments. The use of prophylactic antibiotics will also decline leading to less antibiotic resistance.

~ After the first UTI, close clinical follow up monitoring should be maintained to permit prompt diagnosis and treatment of recurrent infections to avoid long term complications of UTI.

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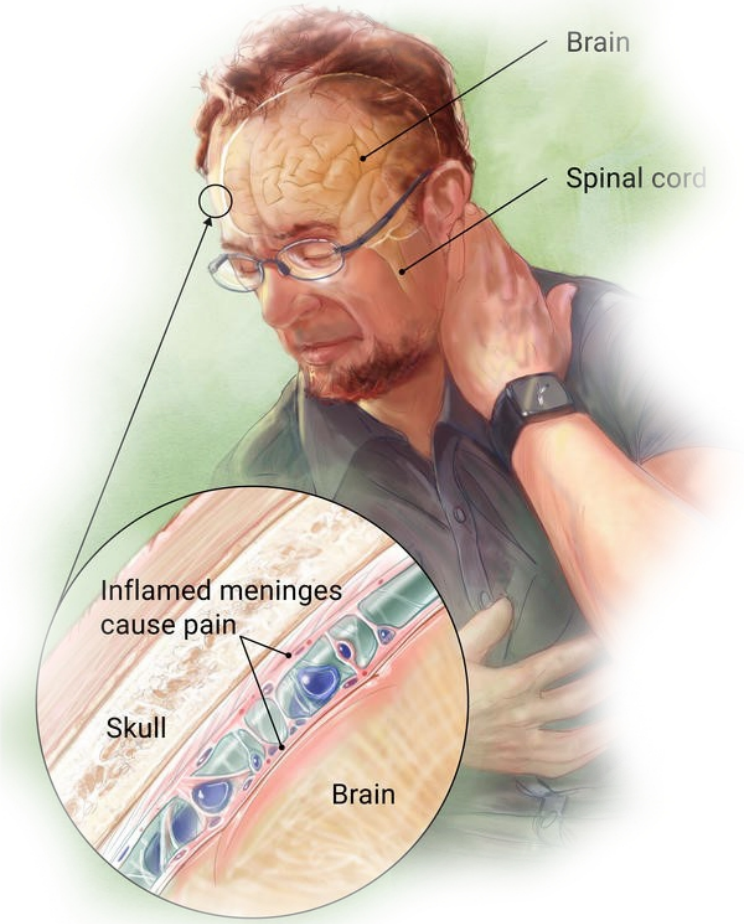

Acute Bacterial Meningitis

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Acute Bacterial Meningitis

~ Acute bacterial meningitis is a neurologic Emergency associated with high morbidity and mortality.
~ Parental anxiety should not be discounted, even if the child does not appear especially unwell.
~ If HSV encephalitis is suspected in newborn, aciclovir (20mg/kg/dose IV , 8 hourly) should be started until further clinical information is available.
~ Do not delay antibiotics or supportive care to undertake or wait for CT Scan.

Table 1 Common Causes of Acute Bacterial Meningitis According to Age

Age	Pathogen
0-4 wk	Streptococcus agalactiae Escherichia coli Listeria monocytogenes Streptococcus pneumoniae
1-3 mo	E. coli L. monocytogenes Neisseria meningitidis S. agalactiae S.pneumoniae Haemophilus influenzae
3 mo to 18 yr	N. meningitidis S.pneumoniae H.influenzae

Investigations:

~ CBC, ESR/CRP , Blood C/S, S.electrolytes , Bl.Glucose
~ Coagulation profile, FDP if suspected DIC
~ Lumbar Puncture for CSF
  - gram stain
  - cell count (WBC & differential , RBC)
  - Chemistry (protein & Glucose compared to blood G)
  - Culture & sensitivity
  - If suspected TB : AFB stain and mycobacterium TB culture
  - If suspected Viral Encephalitis: PCR for HSV, VZV, CMV, and enterovirus
  - Always keep extra tube of CSF sample

Broad categories	Specific indications to delay LP
Local site for LP	• Skin infection at site of LP • Anatomical abnormality at the LP site
Patient instability	• Respiratory or cardiovascular compromise • Continuing seizure activity
Suspicion of space occupying lesion or raised intracranial pressure	• Focal seizures • Focal neurological signs • Reduced conscious state (some suggest a GCS of <8) and especially if the patient is comatose • Decerebrate or decorticate posturing • Fixed dilated or unequal pupils • Absent dolls eye movement • Papilloedema • Hypertension or bradycardia • Irregular respirations
Haematological	• Coagulopathy

CT head is indicated if

~ Focal neurological
~ Prolonged convulsions
~ Impaired level of consciousness
~ Evidence of increased intracranial pressure

	Polymorphs (PMN) (x 10^6/L)	Mononuclear cells (lymphocytes) (x 10^6/L)	Protein (g/L)	Glucose (mmol/L)	Glucose (CSF:Blood ratio)
NORMAL ≤ 1 month of age	0*	< 20	< 1.0	≥ 2.5	≥ 0.6
NORMAL > 1 month of age	0	≤ 5	< 0.4	≥ 2.5	≥ 0.6
Bacterial meningitis	100 - 10,000 (but may be normal)	Usually < 100	> 1.0 (but may be normal)	Usually decreased	< 0.4 (but may be normal)
Viral meningitis	Usually <100	10 - 1000 (but may be normal)	0.4 - 1 (but may be normal)	Usually normal	Usually normal

Management :

~ check ABCs
~ restore circulating volume and urinary output as priority
~ monitor vital signs, hydration and neurological status
~ If impaired consciousness keep patient NPO
~ Start antibiotics after collecting cultures as soon as possible

Duration of therapy

As a guide, the duration of antibiotic therapy in 'uncomplicated' cases of acute bacterial meningitis are:

- Group B streptococcus, 14 days;
- Gram negative rods, 21 days;
- Listeria monocytogenes, 21 days;
- *Neisseria meningitidis*, 7 days;
- *Haemophilus influenzae* type b, 10 days;
- *Streptococcus pneumoniae*, 14 days;
- 'Culture negative' but significant CSF pleocytosis present, minimum of 7 days recommended.

Supportive Care & Monitoring

- Input & Output chart, weight
- Daily head circumference
- Watch for SIADH (urine sp. Gravity, s-Na)
- IVF maintenance (if SIADH 2/3 maintenance)
- Anticonvulsant if convulsions

TABLE 5 : Recommendations for Empiric Antibiotic Therapy of Bacterial Meningitis

Patients and Special Modifying Circumstances	Antibiotic	Dosage (IV)
Neonate/infant <3 mo	Ampicillin *plus* Cefotaxime *or* Gentamicin	50–100 mg/kg every 6–8 hr 100 mg/kg every 8–12 hr 2.5 mg/kg every 8 hr
Neonate preterm, low birth weight	Vancomycin *plus* Ceftazidime	15 mg/kg every 8–24 hr 100 mg/kg every 8–12 hr
>3 mo to <50 yr	Ceftriaxone *or* Cefotaxime	100 mg/kg every 24 hr (max. 4 g every 24 hr) 100 mg/kg every 8 hr (max. 12 g per day)
Drug-resistant *Streptococcus pneumoniae*	Ceftriaxone *plus* Rifampicin *or* Vancomycin	100 mg/kg every 24 hr (max. 4 g every 24 hr) 10–20 mg/kg every 24 hr (max. 600 mg every 24 hr) 15 mg/kg every 6 hr (max. 500 mg every 6 hr)
Neurosurgery, cerebrospinal fluid shunt, or head trauma	Ceftazidime plus Nafcillin *or* Flucloxacillin (*or* Vancomycin *plus* Aminoglycoside)	100 mg/kg every 8 hr (max. 12 g per day) 50 mg/kg every 6 hr (max. 12 g per day) 15 mg/kg every 6 hr (max. 500 mg every 6 hr) 2.0–2.5 mg/kg every 8 hr

Adjunctive Therapy . Corticosteroids

- Current evidence suggests that early steroids (first dose given just after antibiotics) in children with acute bacterial meningitis reduce the risk of hearing loss & neurological sequelae.
- Steroids are recommended early in children (older than 3 months of age) with acute bacterial meningitis , provided that they have not been pre-treated with parentral antibiotics.
- The dosing regimen is 0.15 mg/kg/dose IV ,every 6 hours for 4 days
- If CSF results are not consistent with bacterial meningitis & the child is clinically improving , the recommendation is to stop steroids

but continue antibiotics for at least 48 hours until negative CSF cultures are confirmed

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Hyperbilirubinemia

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Hyperbilirubinemia

INTRODUCTION

Jaundice occurs in most newborn infants. Most jaundice is benign but, because of the potential toxicity of bilirubin, newborn infants must be monitored.

DEFINITIONS

- **Bilirubin encephalopathy** are the clinical central nervous system findings caused by bilirubin toxicity to the basal ganglia and various brainstem nuclei
- **Acute bilirubin encephalopathy** is used to describe the acute manifestations of bilirubin toxicity occured in the first weeks after birth
- **Kernicterus** describes chronic and permanent clinical sequelae of bilirubin toxicity

RISK FACTORS

- Major
    - Jaundice within the first 24 hours
    - Blood group incompatibility
    - Gestational age, 35-36 weeks
    - Previous sibling requiring phototherapy for hemolytic disease
    - Cephalhematoma or significant bruising
    - Weight loss greater than 10% of birth weight (may be associated with ineffective breast-feeding)
    - Family history of red cell enzyme defects
- Minor
    - Jaundice occurring before discharge
    - Gestational age 37-38 weeks
    - Previous sibling with jaundice
    - Macrocosmic infant of a diabetic mother
    - Male gender
- Avoid risk of hyperbilirubinemia by:
    - Promoting and supporting successful breast feeding
    - Measure total serum bilirubin (TSB) in jaundiced infants at least once per 24 hours in nonhemolytic jaundiced babies
    - Infants < 38 weeks gestation, particularly those who are breastfed, are at higher risk of developing hyperbilirubinemia and require closer monitoring
    - Follow table below for laboratory evaluation of jaundiced infant

Laboratory evaluation of the jaundiced infant of 35 or more weeks gestation

Indications	Assessments
Jaundice in the first 24 h **Jaundice appears excessive for infant's age** **Infant receiving phototherapy or TSB rising rapidly and unexplained**	x TSB level x Blood type and Coombs' test x Complete blood count smear x Direct conjugated bilirubin x Reticulocyte count, G6PD, albumin x Repeat TSB every 2, 4 or 12 h depending on infants age and TSB level
Elevated direct (or conjugated) bilirubin level	x Do urinalysis and urine culture x Reticulocyte count, G6PD, albumin x Evaluate for sepsis if indicated depending on history and physical examination x Liver function tests x Abdominal ultrasound
Jaundice present at or beyond age 3 wk **Sick infant**	x Total and direct bilirubin level x If direct bilirubin elevated, evaluate for causes of cholestasis x Liver function tests x Check results of newborn galactosemia screen, and evaluate infant for signs or symptoms of hypothyreoidism

- Interpret bilirubin level according to the infant's age and available graphs at the end of this guideline depending on weight and clinical condition of the baby
- Apply phototherapy or exchange transfusion if needed
- For clinical pathway for infants readmitted in the hospital for phototherapy or exchange transfusion, follow table below

Hyperbilirubinemia

Clinical pathway for infants readmitted in the hospital for phototherapy or exchange transfusion

x	Use intensive phototherapy and/or exchange transfusion as indicated
x	Laboratory tests
	¾ TSB and direct bilirubin levels
	¾ Blood type (ABO, Rh)
	¾ Direct antibody test (Coombs)
	¾ Serum albumin
	¾ Complete blood cell count with differential and smear for red cell morphology
	¾ Reticulocyte count
	¾ G6PD if suggested by ethnic or geographic origin or if poor response to phototherapy
	¾ Urine for reducing substances
x	In case of suspected sepsis, perform: ¾ Blood culture ¾ Urine culture ¾ Cerebrospinal fluid analysis

PHOTOTHERAPY

- Exposure of free or bound bilirubin to blue light with wave lengths of 400 – 500 nm causes changes in configuration from the normal Z form to the E form which is:
  - Non- toxic to CNS tissues
  - Excreted in bile without configuration
- Phototherapy works in three ways
  - Configurational isomeraization
  - Structural isomeraization
  - Photooxidation
- Effectiveness of phototherapy depends on
  - Wavelength
  - Dose-response relationship
  - Distance between light and baby
  - Skin surface area exposed
  - Character of the light source
    - With standard single light around (12-15 mw/cm^2/nm) there will be 6 -20% decline in first 24 hours
    - Intense phototherapy (around 30 mw/cm^2 /nm) cause 30 - 40 % decline in TSB in first 24 hours mainly first 4 - 6 hours
- Types of phototherapy lights
  - Green light
  - Quartz halide spotlights
  - Blue florescent tube
  - White (daylight) florescent lamps
  - Fiberoptic light

INTRAVENOUS IMMUNOGLOBULIN - **IVIG**

- Hyperbilirubinemia in Rh and ABO sensitized infants results from destruction of RBCs coated by maternal isoantibodies
- They cause extravascular destruction of RBCs mediated by Fc receptors-bearing cells within the reticuloendothelial system
- IVIG blocks and inhibiting hemolysis, so reducing formation of bilirubin. Also, they may accelerate the rate of immunoglobulin G (IgG) catabolism, thereby reducing the circulating pathogenic autoantibodies
- In hemolytic disease of newborns (HDN), IVIG can be given when hyperbilirubinemia is refractory to phototherapy and reaching values close to exchange transfusion level (e.g. 30-50 Pmol/L below the level of exchange)
- Dose: 0.5-1 g/kg over 2-4 hours and repeat in 12 h if necessary. However, dose of 2 g/kg are used as well
- Until today the only complication reported with the use of IVIG in newborns is NEC which can occur in 2.2% compared with 0.3% of those who did not receive IVIG

EXCHANGE TRANSFUSION (ET)

- Exchange transfusion (ET) may be used to treat severe hyperbilirubinemia but it is also used to manage severe anemia at birth, particularly in the presence of heart failure
- In the treatment of hemolytic disease of the newborn (HDN), the aim is to remove both the antibody-coated red cells and the excess bilirubin
- ET is an invasive procedure associated with a potential risk (especially in preterm babies) of serious adverse events. As such, it should be undertaken only by staff who are experienced in the procedure
- Meticulous care must be taken throughout, especially with volume balance, the rate of the exchange, the vital signs and any signs of air in the lines
- All exchanges are to be conducted in NICU Level 3
- There must be at least one doctor and one nurse in attendance
- Before ET is undertaken informed written consent must be obtained However, if ET is urgently needed and consent could not be taken, consultant should sign
- Resuscitation equipment and drugs must be checked and ready for use including adrenaline
- Ventilator must be set up ready for use in the unit
- The infants cardiorespiratory status and oxygen saturation will be monitored continuously
- Aspirate stomach contents prior to commencement of procedure
  - Once it has been started, ET should not be stopped or interrupted unless baby needs resuscitation

BASICS IN ONE CATHETER PUSH PULL TECHNIQUE

- It should be done in sterile conditions
- This can be done through an umbilical venous catheter
- For high UVC placement, the position of the catheter should be checked by an X ray. This is not always necessary for a low position
- A low positioned catheter is usually removed after blood exchange
- Follow table below to estimate appropriate aliquots of blood according to the patient weight

Weight	Milliliters (ml)
<1000gr	5 ml
1000-2000gr	10 ml
>2000gr	15 ml

- Withdraw blood over 2 minutes, infuse slightly faster. Duration of the procedure should take 1½ -2 hours
- Usually use two blood volumes (180 ml/kg)

Monitoring and documentation during ET

Steps	Action
1	Record baseline observations prior to commencing exchange transfusion ¾ Temperature ¾ Heart rate ¾ Respiratory rate ¾ Blood pressure ¾ Oxygen saturation and colour
2	Continuously monitor and record at 15 minutes intervals on the Record of Exchange Transfusion sheet, the following observations: ¾ Skin temperature ¾ Heart rate ¾ Respiratory rate ¾ Oxygen saturation ¾ Blood Pressure (non-invasive)
3	Observe for any changes in neurological status - drowsiness, irritability
4	Record blood results on the Exchange Transfusion Results Sheet
5	Vital signs should be monitored continuously for at least 6-8 hours post transfusion or longer if the baby's condition is not stable

*Have serum electrolytes before starting exchange transfusion as baseline
*This may be repeated after ET has been completed

In the event of collapse during or after exchange

During exchange

Air embolus	¾ Ensure the lines are correctly set up ¾ Watch the lines constantly for air. Be ready to turn off a line instantly if air is seen
Volume imbalance	¾ The nurse should record the volume balance throughout the exchange
Arrhythmias	¾ Watch the ECG screen frequently and set up a monitor to have a beep with the QRS
Acidosis	¾ Blood is preserved in CPD (citrate, phosphate, dextrose) and can be acidotic ¾ Check the baby's pH at the beginning, and then at least once during the exchange (more frequently for a sick, unstable or small baby) and also after exchange is completed
Respiratory distress	¾ Monitor respiration and SpO_2 constantly
Unexpected collapse	¾ Watch the baby carefully ¾ Have resuscitation equipment ready

After exchange

Infection	¾ Prophylactic antibiotics are not required routinely if ET done under complete sterile conditions during procedure
Hypocalcemia	¾ May occur post exchange. Monitor [Ca^{++}] and give replacement Ca^{++} in IV
Hypoglycemia	¾ Unlikely during the exchange ¾ However, if this develops a 10% dextrose solution should be started
Polycythemia, Anemia Thrombocytopenia	¾ Check CBC ¾ Thrombocytopenia is common after ET
Polycythemia or Anemia	¾ From poorly mixed or packed blood. Check Hgb
Coagulopathy or Neutropenia	¾ More likely if more transfusions are done
Necrotising enterocolitis	¾ May occur
Blood transmitted infections	¾ Make sure that blood for ET is properly tested (for example HIV, Hep B, C…)
Graft Versus Host Disease	¾ More likely with more preterm infants, intrauterine transfusions, multiple exchanges, and related donors ¾ This can be prevented by using irradiate donor blood

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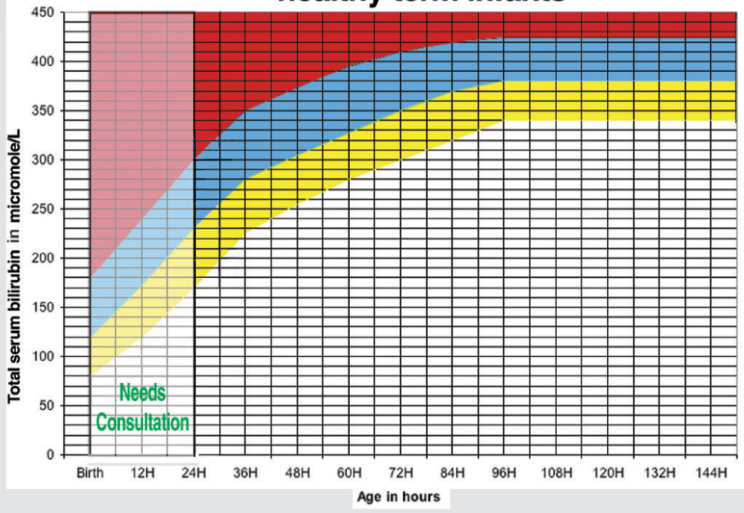
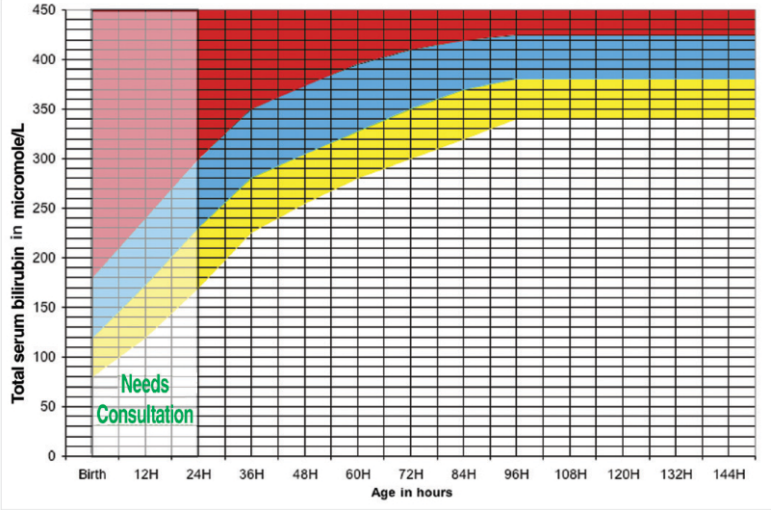
Management of hyperbilirubinemia in the healthy term infants



Management of hyperbilirubinemia in the sick term newborn infant or with hemolytic disease

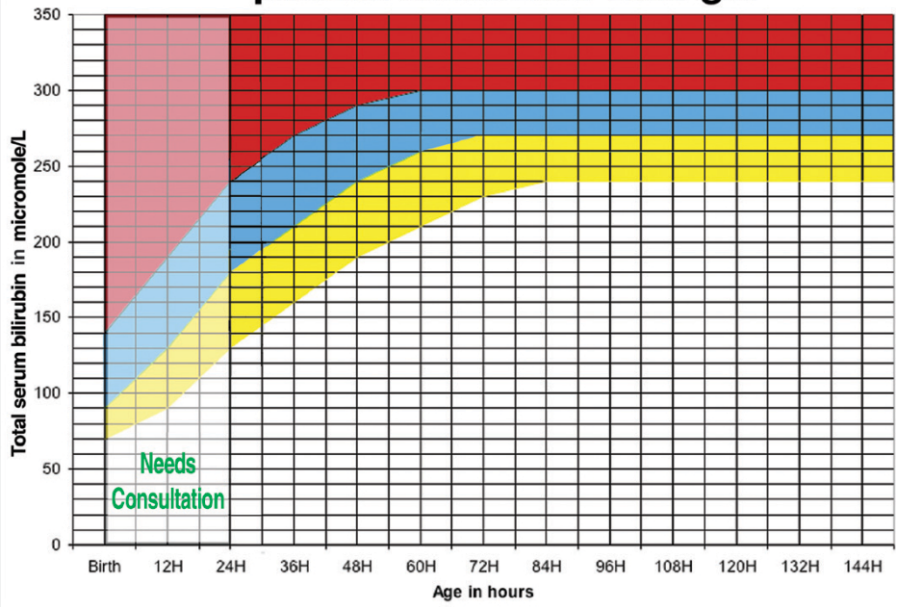


Exchange transfusion

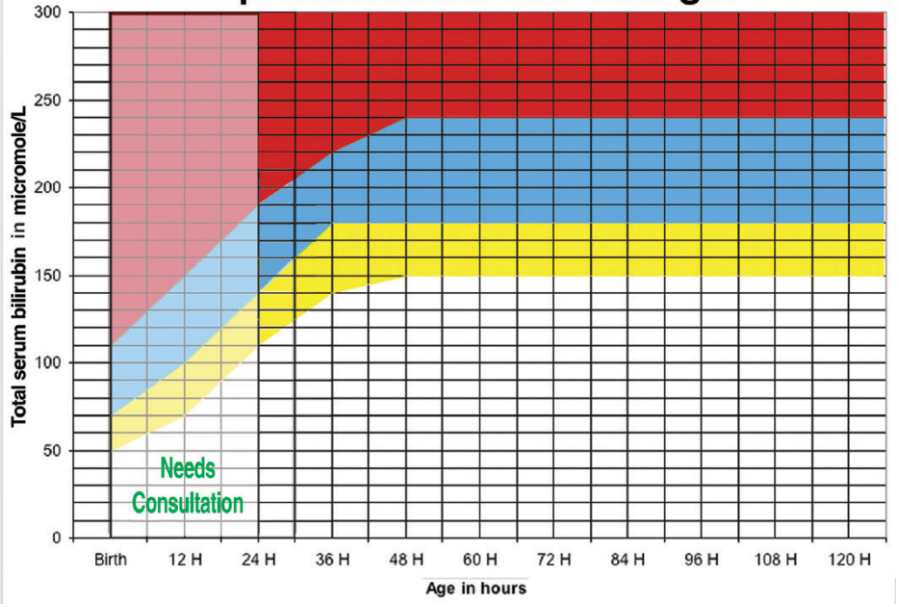
Double phototherapy

Single phototherapy

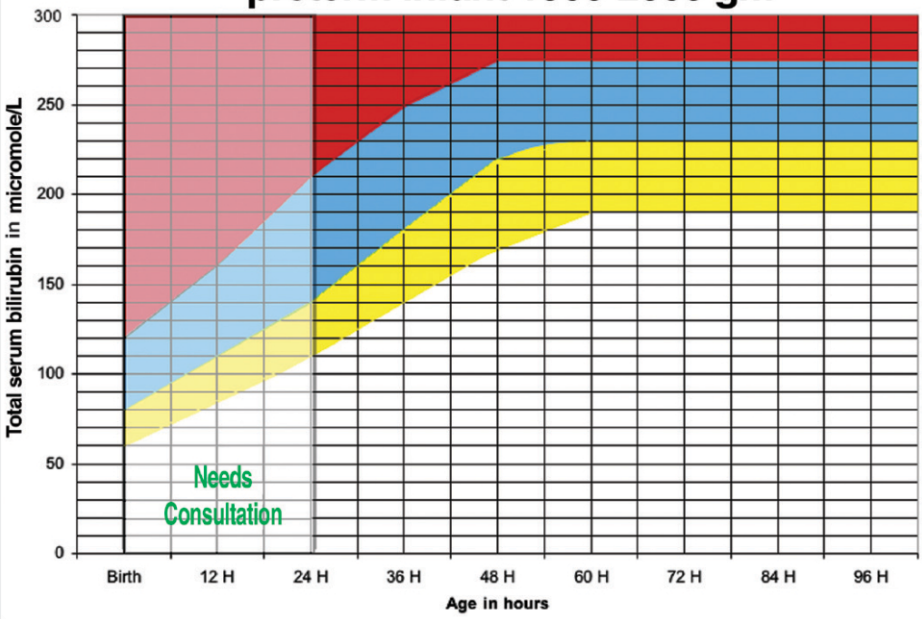
Management of hyperbilirubinemia in the preterm infant 2000-2500 gm



Management of hyperbilirubinemia in the preterm infant 1000-1500 gm



Management of hyperbilirubinemia in the preterm infant 1500-2000 gm



Hyperbilirubinemia in preterm infants 500 – 1000 gm

- Prophylaxis phototherapy from birth for 4-5 days
- Exchange transfusion when levels reach 150 – 240 umol/L depends on the GA, weight and whether sick or healthy. To be discussed with a consultant

Appendix

Vital signs in children

Vital signs in children

HEART RATE (PER MINUTES)

Age	Awake Rate	Sleeping Rate
Newborn – 3 months	85 – 205	80 – 160
3 months – 2 years	100 – 190	75 – 160
2 – 10 years	60 – 140	60 – 90
>10 years	60 – 100	50 – 90

RESPIRATORY RATE (PER MINUNTES)

Age	Rate
0 – 3 months	25 – 66
3 – 6 months	24 – 64
6 – 9 months	23 – 61
9 – 12 months	22 – 58
12 – 18 months	21 – 53
18 – 24 months	19 – 46
2 – 3 years	18 – 38
3 – 4 years	17 – 33
4 – 6 years	17 – 29
6 – 8 years	16 – 27
8 – 12 years	14 – 25
12 – 15 years	12 – 23

Hypotension by Systolic Blood Pressure

AGE	SYSTOLIC BLOOD PRESSURE (MM HG)
Term Neonate (0 – 28 days)	< 60
Infant (1 – 12 months)	< 70
Children (1 – 10 years)	< 70 + (age in years x 2)
Children >10 years	< 90

Modified Glasgow Coma Scale for Infants and Children

	CHILD	INFANT	SCORE
Eye opening	Spontaneous	Spontaneous	4
	To speech	To speech	3
	To pain	To pain	2
	None	None	1
Best Verbal Response	Oriented, appropriate	Coos and babbles	5
	Confused	Irritable, cries	4
	Inappropriate words	Cries in response to pain	3
	Incomprehensible sounds	Moans in response to pain	2
	None	None	1
Best Motor response*	Obeys commands	Moves spontaneously and purposely	6
	Localizes painful stimulus	Withdraws in response to touch	5
	Withdraws in response to pain	Withdraws in response to pain	4
	Flexion in response to pain	Abnormal flexion posture to pain	3
	Extension in response to pain	Abnormal extension posture to pain	2
	None	None	1

*If the child's intubated, unconscious, or preverbal, the most important part of this scale is motor response.

Endotracheal tube (ETT) size for children

Age	Uncuffed	Cuffed
GA < 28 wks (or < 1 kg)	2.5	---
GA 28 – 34 wks (or 1 – 2 kg)	3	---
GA 34 – 38 wks (or 2 – 3 kg)	3.5	---
Term 0 – 28 days (> 3 kg)	3.5 – 4	---
Infant (1 – 12 months)	3.5	3
Child (1 – 8 years)	4+(Age in yrs/4)	3.5+(Age in yrs/4)

*GA = Gestational age

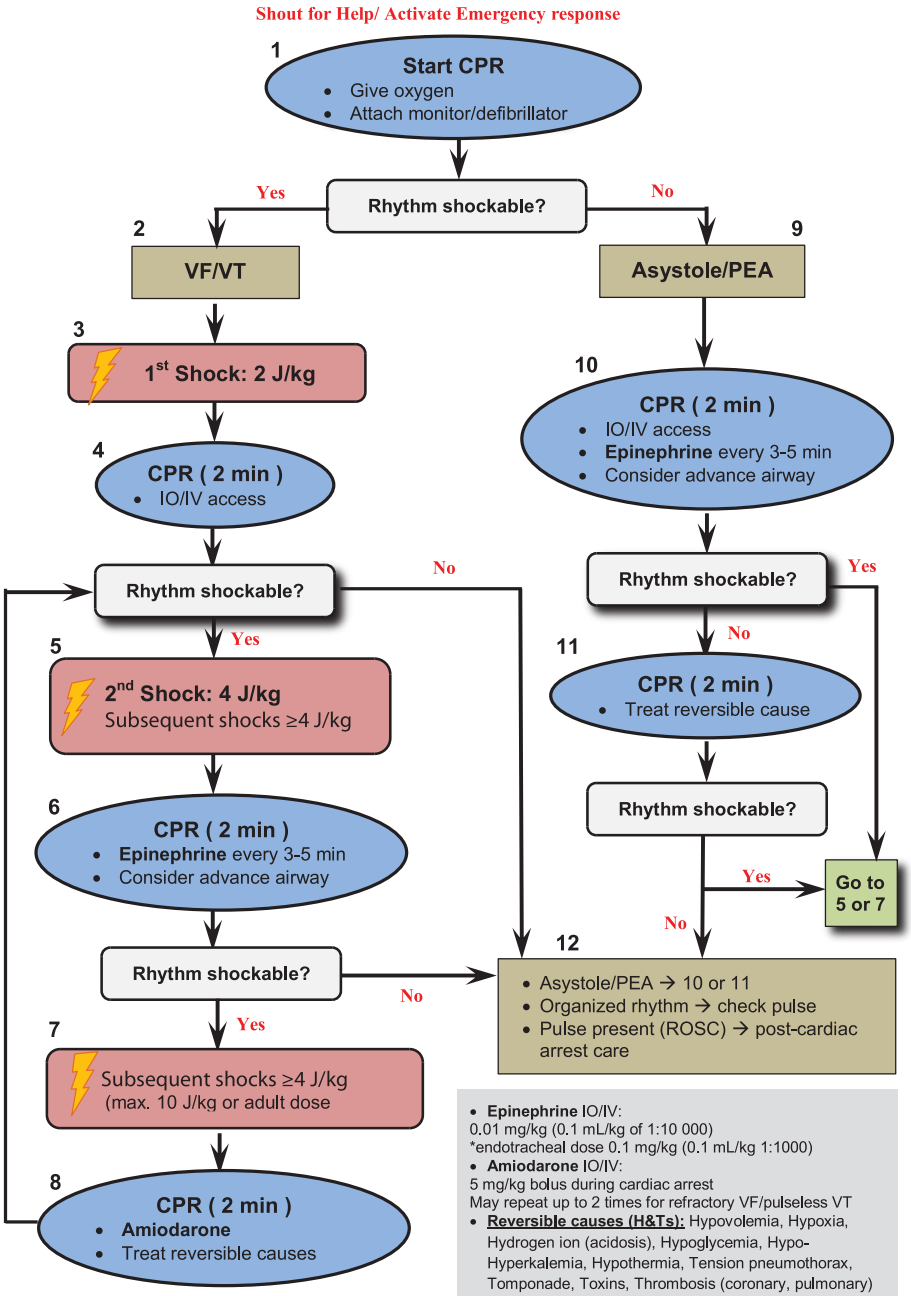
Surface Area

Body surface area

$$= \sqrt{\frac{hight\ \ cm\ X\ wt\ \ kg}{3600}}$$

- Ref: 2010 American Heart Association, 2013 UpToDate, Neonatal Resuscitation Textbook 5th edition

Pediatric Cardiac Arrest

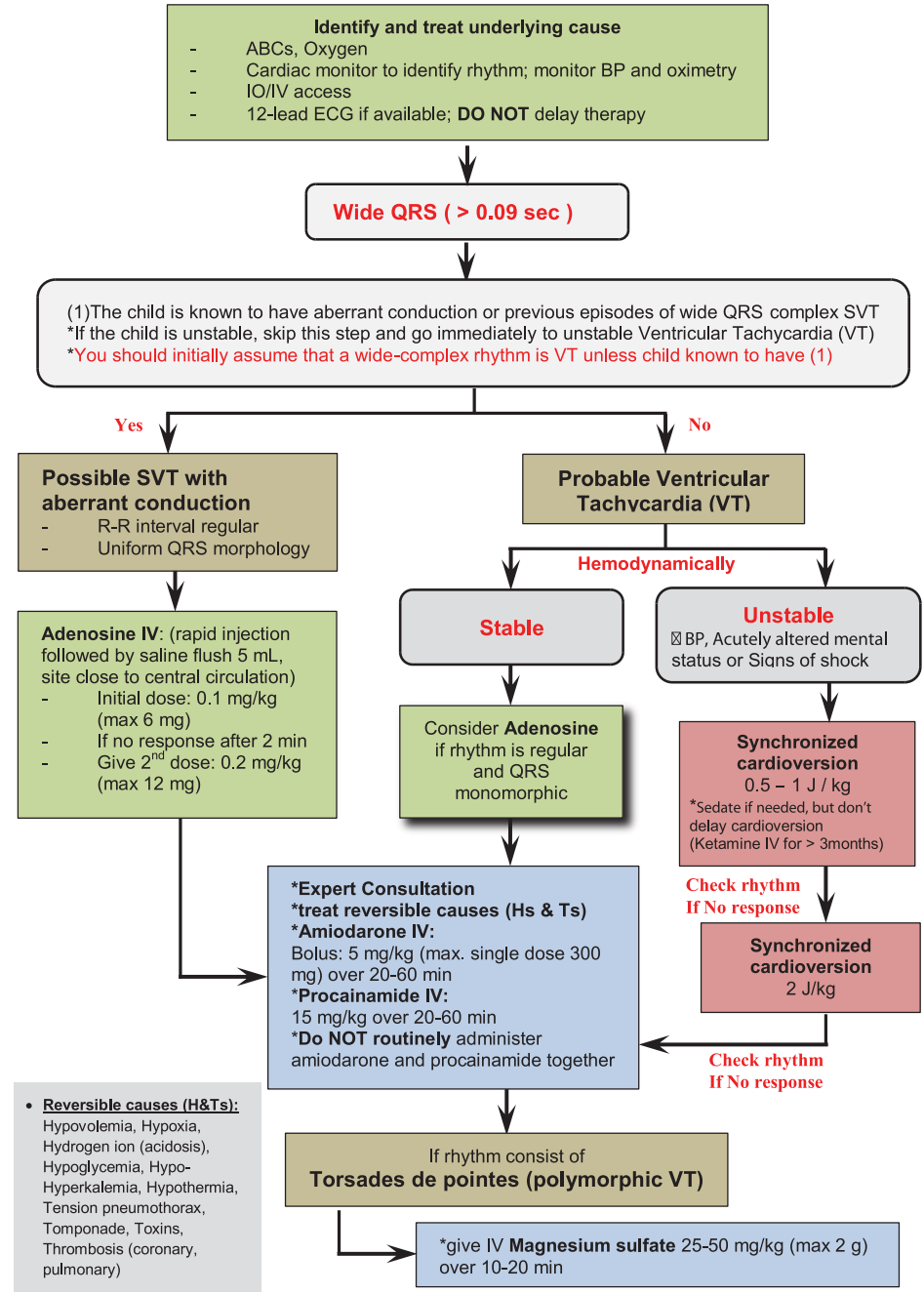


- **Epinephrine** IO/IV:
0.01 mg/kg (0.1 mL/kg of 1:10 000)
*endotracheal dose 0.1 mg/kg (0.1 mL/kg 1:1000)
- **Amiodarone** IO/IV:
5 mg/kg bolus during cardiac arrest
May repeat up to 2 times for refractory VF/pulseless VT
- **Reversible causes (H&Ts):** Hypovolemia, Hypoxia, Hydrogen ion (acidosis), Hypoglycemia, Hypo-Hyperkalemia, Hypothermia, Tension pneumothorax, Tomponade, Toxins, Thrombosis (coronary, pulmonary)

- Ref: 2010 American Heart Association (PALS)

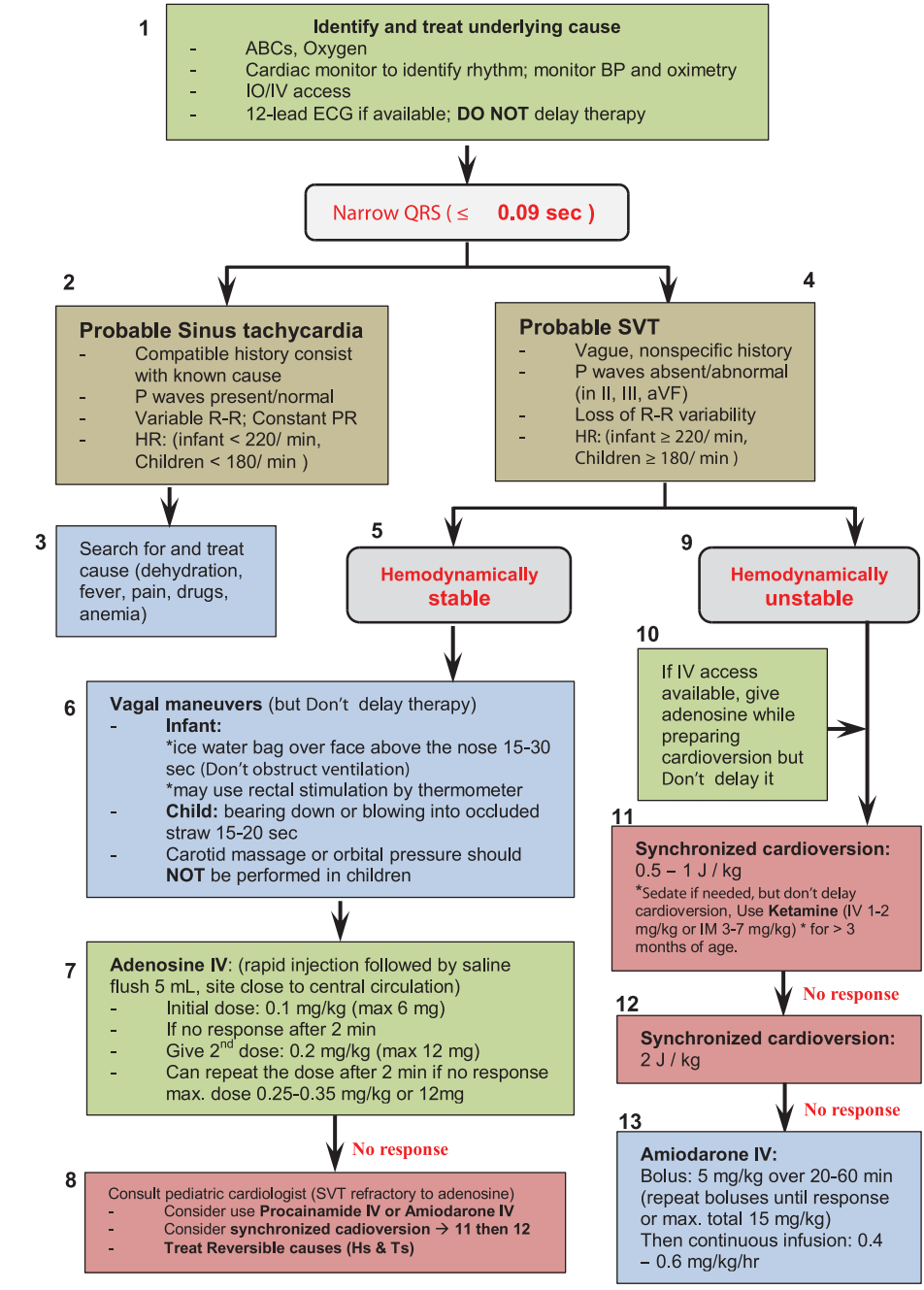
Kuwait Pediatrics KIMS 2015

Wide Complex Tachycardia with a pulse



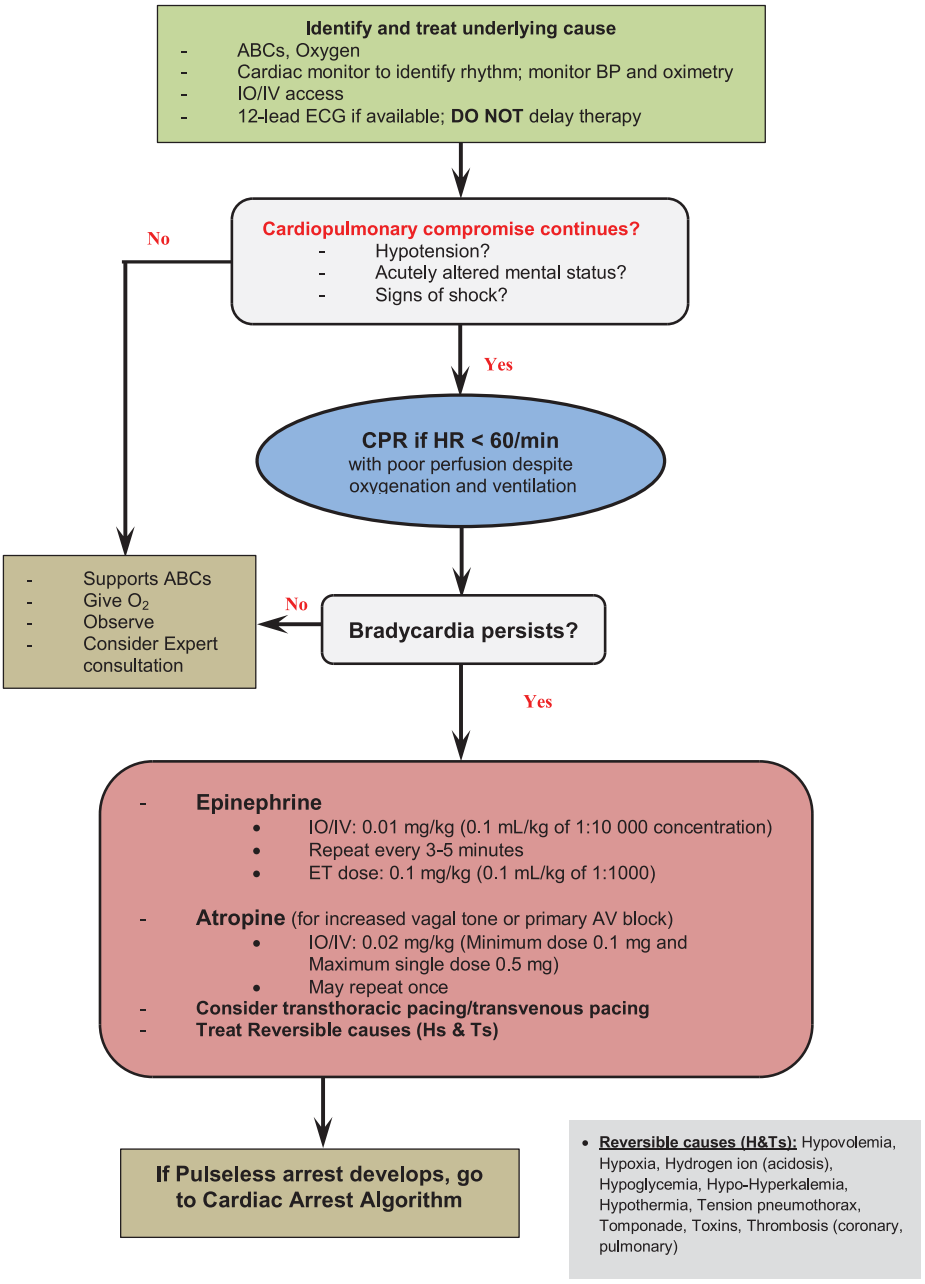
- Ref: 2010 American Heart Association (PALS) , 2013 UpToDate

Narrow Complex Tachycardia with a pulse



- Ref: 2010 American Heart Association (PALS) , 2013 UpToDate

Bradycardia with a pulse & Poor Perfusion



- Ref: 2010 American Heart Association (PALS)

Septic Shock

Define as sepsis with **CVS dysfunction** (ie, ⊠ BP, reliance on vasoactive drugs to maintain a normal BP, or 2 of the following: prolonged capillary refill, oliguria, metabolic acidosis, ⊠ arterial Lactate)**that persists despite the administration of ≥ 40 ml/kg NS in 1 hour.**

1st hour

- Recognize altered mental status and perfusion
- ABC, O2 and support ventilation, establish vascular access
- Consider CBC, BGA, Lactate, glucose, ionized calcium, cultures

First hour: Push repeated 20 ml/kg 0.9% NS to treat shock. Give up to 3, 4, or more boluses unless rales, respiratory distress or hepatomegaly develops.
Additional therapies:

- Correct hypoglycemia and hypocalcemia
- Give 1st dose antibiotics STAT
- Consider ordering vasopressor drip and stress-dose **hydrocortisone***
- Establish 2nd vascular access site if vasoactive infusion anticipated

Fluid responsive (ie, normalized perfusion/hemodynamics)?

Yes → **Consider PICU monitoring**

No

Begin vasoactive drug therapy and titrate to correct hypotension/poor perfusion; consider establishing arterial and central venous access

Normotesive:
Begin **Dopamine** 2 – 20 mcg/kg/min IV/IO infusion; titrate to desired effect

Hypotensive vasodilated (worm) shock:
Begin **Norepinephrine** 0.1 – 2 mcg/kg/min IV/IO infusion; titrate to desire effect

Hypotensive vasoconstricted (cold) shock:
Begin **Epinephrine** 0.1 – 1 mcg/kg/min IV/IO infusion (consider higher doses if needed)

Evaluate ScvO2; goal ScvO2 sat ≥ 70%?

ScvO2 ≥70%, ⊠ BP **(warm shock)**
-Additional fluid boluses
-**Norepinephrine** +/-
-**Vasopressin** (0.2-2 milliunits/kg/min)

ScvO2<70%, normal BP but poor perfusion:
-Transfuse to Hgb >10 g/dl
-Optimize arterial oxygen sat.
-Additional fluid boluses
-Consider **Dobutamine** (2-20 mcg/kg/min IV/IO; titrate to desire effect)
-Consider **Milrinone** or **Nitroprusside**

ScvO2<70%, ⊠ BP& poor perfusion (cold shock):
-Transfuse to Hgb >10 g/dl
-Optimize arterial oxygen sat.
-Additional fluid boluses
-Consider Epinephrine or Dobutamine + Norepinephrine

*Fluid-refractory and Dopamine- or norepinephrine-dependent shock defines pt at risk for adrenal insufficiency (AI)
-Draw baseline cortisol; consider ACTH stimulation test if unsure of need for steroids
- If adrenal insufficiency is suspected, Give **hydrocortisone 2 mg/kg bolus IV** (Max 100 mg)

- Ref: 2010 American Heart Association (PALS)

Rapid Sequence Intubation (7 Ps)

1. Preoxygenation (5 minutes before intubation):
- Administer O_2 at highest concentration available
- For spontaneously breathing child, use non-rebreathing mask for at least 3 min.
- If apnic or desaturating, use bag-mask ventilation +/- Cricoid pressure

2. Preparation (AMPLE)
- Identify conditions that will affect choice of medications
- Identify conditions that will predict difficult intubation or bag-mask ventilation
- Assemble equipment & check function
- Develop contingency plan for failed intubation
- If cardiac arrest or deeply comatose child, sedation & paralytic agents are unnecessary prior to intubation

(AMPLE)
- A = Allergy
- M = Medications
- P = Past Medical
- L = Last Meal
- E = Events

3. Pretreatment
- **Atropine** IV: 0.02 mg/kg (minimum 0.1 mg – max 0.5 mg)
  - All infant < 1 yr
  - 1-5 yrs receiving succinylcholine, and > 5 yrs with 2nd dose succinylcholine
- **Lidocaine** IV: optional for ↑ ICP, 2-3 min before intubation
  - 1.5 mg/kg (max. dose 100 mg)

Sedation

Hx of asthma, bronchospasm, ↓BP **or Septic shock**
- **Ketamine (> 3 months)** IV: 1-2 mg/kg IM: 3-7 mg/kg

↑ ICP or head **injury (if stable or** ↓BP)
- **Etomidate** IV: 0.3 mg/kg

Status epilepticus *(stable Bp)
- **Midozolam** IV: 0.2-0.3 mg/kg (max 2mg)

*(↓ Bp)
- Etomidate IV: 0.3 mg/kg

Uncomplicated child

Etomidate IV: 0.3 mg/kg

4. Paralytic
- **Succinylcholin:**
  - IV: infant & younger children 2 mg/kg, older children 1-1.5 mg/kg
  - IM: 3-5 mg/kg
  - **AVOID in:** neuromuscular disease, organophosphate poisoning, 48-72 hrs after burn, crush or denervation injury, malignant hyperthermia, pre-existing ↑ K^+.
- **Rocuronium (if succinylcholine contraindicated) dose IV 1 mg/kg**

5. Protection & Positioning
- Sniffing position
- Cricoid pressure

6. Placement & confirmation

7. Post-intubation
CXR, Infusion Midozolam, Infusion fentanyl (1 mic/kg/hr)

- Ref: UpToDate 2013

Emergency Drugs

Drug	Indications/Dosages	Max . dose
Adenosine	**SVT** • 0.1 mg/kg IV/IO (1st dose) rapid push, 2nd dose 0.2 mg/kg IV/IO rapid push	1st dose 6 mg 2nd dose 12 mg
Albumin	**Shock, Trauma, burns** • 0.5 – 1 g/kg (10-20 mL/kg of 5% solution) IV/IO rapid infusion	
Albuterol	**Asthma, Anaphylaxis (bronchospasm), hyperkalemia** • MDI: 4-8 puffs via inhalation q 20 min PRN with spacer (or ET if intubated) • Neb: 2.5 mg/dose (wt <20 kg) or 5 mg/dose (wt >20 kg) via inhalation q 20 min PRN • Continuous Neb: 0.5 mg/kg/hr via inhalation	Max 20 mg/hr
Amiodarone	**SVT, VT (with pulse)** • 5 mg/kg IV/IO load over 20-60 min **Pulseless arrest (ie, VF/pulseless VT)** • 5 mg/kg bolus	*Single dose: 300 mg *Daily boluses max. 15 mg/kg (2.2g in adult)
Atropine sulfate	**Bradycardia (symptomatic)** • 0.02 mg/kg IV/IO (min dose 0.1 mg) • May repeat the dose once • 0.04 – 0.06 mg/kg ET	*Single dose 0.5 mg (child) 1 mg (adult) *total dose (if repeated) 1 mg (child) 3 mg (adult)
Calcium chloride 10%	**Hypocalcaemia, Hyperkalemia, Hypermagnesemia** **Calcium channel blocker overdose** • 20 mg/kg (0.2 mL/kg) IV/IO slow push during arrest, repeat PRN	
Dexamethasone	**Croup** • 0.6 mg/kg PO/IV/IM	10 mg
Dextrose (glucose)	**Hypoglycemia** • 0.5 – 1 g/kg IV/IO ($D_{25}W$ 2 - 4 mL/kg; $D_{10}W$ 5 -10 mL/kg)	
Diphenhydramine	**Anaphylactic shock** • 1 – 2 mg/kg IV/IO/IM q 4 to 6 hours	Single dose 50 mg
Dobutamine	**Congestive heart failure, cardiogenic shock** • 2 to 20 mcg/kg/min IV/IO infusion; titrate to desired effect	
Dopamine	**Cardiogenic shock, distributive shock** • 2 to 20 mcg/kg/min IV/IO infusion; titrate to desired effect	
Epinephrine	**Pulseless arrest, bradycardia (symptomatic)** • IV/IO: 0.01 mg/kg (0.1 mL/kg of 1:10 000) q 3 - 5 min. • ET: 0.1 mg/kg (0.1 mL/kg of 1:1000) q 3 - 5 min **Hypotensive shock** • 0.1 – 1 mcg/kg/min IV/IO infusion (consider higher doses if needed) **Anaphylaxis** • IM autoinjector 0.3 mg (wt ≥30 kg) or IM junior autoinjector 0.15 mg (wt 10 – 30kg) • IM: 0.01 mg/kg (0.01 mL/kg of 1:1000) q 15 min PRN • IV/IO: 0.01 mg/kg (0.1 mL/kg of 1:10 000) q 3 – 5 min (max single dose 1 mg) if hypotensive **Asthma** • SC: 0.01 mg/kg (0.01 mL/kg of 1:1000) subcutaneously q 15 minutes **Croup** • 0.25 to 0.5 mg racemic solution(2.25%) mixed in 3 mL NS via inhalation • 3 mL of 1:1000 sol. Mixed with 3 mL NS via inhalation	Single dose IV/IO 1 mg Single IM dose 0.3 mg SC: 0.3mg or 0.3 mL

- Ref: 2010 American Heart Association

Emergency Drugs *(continued)*

Drug	Indications/Dosages	Max . dose
Etomidate	**RSI** • 0.2 – 0.4 mg/kg IV/IO infused over 30 -60 seconds will produce rapid sedation that lasts for 10 to 15 min	20 mg
Hydrocortisone	**Adrenal insufficiency** • 2 mg/kg IV bolus	100 mg
Ipratropium bromide	**Asthma** • 250 – 500 mcg via inhalation q 20 min PRN x 3 doses	
Lidocaine	**VF/pulseless VT, wide-complex tachycardia (with pulses)** • 1 mg/kg IV/IO bolus • Maintenance: 20 – 50 mg/kg/min IV/IO infusion (repeat bolus dose if infusion initiated >15 min after initial bolus) • ET: 2 – 3 mg/kg	
Magnesium sulfate	**Asthma (refractory status asthmaticus), Torsades de pointes, hypomagnesemia** • 25 – 50 mg/kg IV/IO bolus (pulseless VT), or over 10 – 20 min (VT with pulses) or slow infusion over 15-30 min (status asthmaticus)	2 g
Methylprednisolone	**Asthma (status asthmaticus), anaphylactic shock** • Load: 2 mg/kg IV/IO/IM; only use acetate salt IM • Maintenance: 0.5 - 1 mg/kg IV/IO q 6 hours	60 mg / day Max in adult 125 mg
Milirnone	**Myocardial dysfunction and increased SVR/PVR** • Loading: 50 mcg/kg IV/IO over 10 – 60 min followed by 0.25 – 0.75 mcg/kg/min IV/IO infusion	
Naloxone	**Narcotic (opiate) reversal** • Total reversal required (for narcotic toxicity secondary to overdose): 0.1 mg/kg IV/IO/IM/SC bolus q 2 min PRN • Total reversal not required (eg, for respiratory depression associated with therapeutic narcotic use): 1-5 mcg/kg IV/IO/IM/SC; titrate to desired effect • Maintain reversal: 0.002 – 0.16 mg/kg/hr IV/IO infusion	2 mg (bolus)
Nitroglycerin	**Congestive heart failure, cardiogenic shock** • Initiate at 0.25 – 0.5 mcg/kg/min IV/IO infusion; titrate by 1 mcg/kg/min q 15-20 min as tolerated. Typical dose range 1- 5 mcg/kg/min	10 mcg/kg/min
Nitroprusside	**Cardiogenic shock (ie, associated with high SVR), severe hypertension** • 0.3-1 mcg/kg/min IV/IO initial dose; then titrate up to 8mcg/kg/min as needed	
Norepinephrine	**Hypotensive (usually distributive) shock (ie, low SVR and fluid refractory)** • 0.1-2 mcg/kg/min IV/IO infusion; titrate to desire effect	
procainamide	**SVT, atrial flutter, VT (with pulses)** • 15 mg/kg IV/IO load over 30-60 min (Do not use routinely with amiodarone)	
Prostaglandine E_1 (PGE_1)	**Ductal-dependent congenital heart disease (all forms)** • 0.05 – 0.1 mcg/kg/min IV/IO infusion initially, then 0.01 to 0.05 mcg/kg/min IV/IO	
Sodium bicarbonate	**Metabolic acidosis (severe), hyperkalemia** • **1 mEq/kg IV/IO slow bolus**	
Vasopressin	**Cardiac arrest** • 0.4 to 1 unit/kg bolus **Catecholamine-resistant hypotension** • 0.0002 to 0.002 unit/kg/min (0.2 to 2 milliunits/kg/min)	40 units (bolus)

• Ref: 2010 American Heart Association

Infusions

Drug	Infusion Rate/ Preparation
Dopamine	**5 - 30 mcg/kg/min** • 3 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 1 mcg/kg/min • 15 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 5 mcg/kg/min
Dobutamine	**5 - 30 mcg/kg/min** • 3 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 1 mcg/kg/min • 15 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 5 mcg/kg/min
Epinephrine	**0.1 - 1 mcg/kg/min** • 0.3 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 0.1 mcg/kg/min
Prostaglandin E1	**0.05 - 0.1 mcg/kg/min** • 0.3 x wt (kg) = mg added to 50 ml NS → 1 ml/hr = 0.1 mcg/kg/min • 500 mcg in 80 ml of NS → 1 ml/kg/hr = 0.1 mcg/kg/min
Morphine	**10 - 40 mcg/kg/hr** • 0.5 x wt (kg) = mg of morphine added to 50 ml NS → 1 ml/hr = 10 mcg/kg/hr

Weight formula:
2 x age (yrs) + 8 = wt in (kg)

- Ref: 2010 American Heart Association

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