ELECTROLYTE

-WORKBOOK-

Organized by

Department of Pediatrics and Pediatric Nephrology,
Apollo Children's Hospitals, Chennai

Organizing

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NephKids 2024

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PROGRAMME

DAY 1

Saturday, September 14th, 2024

FLUID AND ELECTROLYTE WORKSHOP

Conveners: Dr. Thangavelu S, Dr. Sharada RC

TOPIC	MODERATOR	SPEAKERS	09:30- 10:30	10:30- 11:30	11:30-12:30
Hyponatremia	Dr Rajkumar PS	Dr Srinivasan G Dr Muthiah P Dr Swathi Kiran Shiri	Group A	Group B	Group C
Fluid in special situations	Dr Thangavelu S	Dr Sudarsan K Dr Murali T Dr Chidhambharam L	Group B	Group C	Group A
Metabolic Acidosis	Dr Poovazhagi V	Dr Sivaraman D Dr Dhakshayani R V Dr Naresh Kumar S	Group C	Group A	Group B
LUNCH	**12:30 hrs – 13:30 hrs**				
Hypernatremia	Dr Anitha VP	Dr Shyamala J Dr Priyavarthini V Dr Sharada R C	Group A	Group B	Group C
Hypocalcemia/ Hypomagnesemia	Dr Shanthi S	Dr Mani Kumar S Dr Anita Tarigopula Dr Mithuna Shree J	Group B	Group C	Group A
Hypo/ Hyperkalemia	Dr Gowrishankar NC	Dr Prem Kumar L K Dr Ekambaranath TS Dr Venkateswari R	Group C	Group A	Group B

Dr. B.R. Nammalwar

Organising Chairperson, NEPHKIDS

INTRODUCTION

Homeotasis is the fundamental basis for survival for all living creatures. Body water, electrolytes, acid bases and divalent ions have explicit role in maintaining the homeostasis. A solid foundation in understanding the composition of body fluid, fluid requirements, and regulations; assessing and managing dehydration, understanding the physiologic functions of electrolytes, and managing electrolyte derangements is critical. Study of water, electrolytes, acid bases and divalent ions is an important and difficult area of in medicine. Students, Residents (why not teachers) study it intensely, yet too often come away without clarity and confidence they want and need. Why is this? Body water, electrolytes, acid-bases and divalent ions cannot be seen, neither felt, nor palpated or auscultated. It is a mist. The nearer you go, the faster it disappears. In physiology it is taught as a pure science. In clinical medicine it is presented as a group of symptoms with solutions as per guidelines. No life in it. There is diversity in the methodology of teaching of this subject. What is needed is to teach this subject as an applied science. Pediatricians, Pediatric Nephrologists and Pediatric Intensivists with unbridled and non-exhausting enthusiasm for teaching with a team of similar minded colleagues from other teaching Institutions have been organizing "Workshop on fluids, electrolytes, acid-base and divalent ions" for last seven years. Partly it has been fulfilling the lacunae. With this booklet it is much more. To the readers, treasure it as a 'Rose' from your beloved. Express your feeling. If you are thankful, contribute your thoughts and knowledge. Knowledge is an ocean and no one person can fathom it. Someday, I know it will be a Monograph on Body water, electrolytes, acid bases and divalent ions. A dream will come true.

Faculty- Workshop

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HYPONATREMIA

Contributors: Dr. Rajkumar PS, Dr Srinivasan G, Dr Muthiah P, Dr Swathi Kiran Shiri

Physiology of Sodium balance:

- Sodium is the dominant cation of ECF. Maintains ECF osmolarity and thereby cell volume of billion cells in the body, including that of brain.
- Sodium intake: Infants receive sodium from breast milk (7 mEq/L) and formula (7-13 mEq/L). An average Indian consumes 10.98 grams of salt per day - 119 % > recommended limit of 5 grams/day by WHO (1 g NaCl = 394 mg, 17 mEq or 17 mmol of Na and Cl).

- Excretion: Occurs through urine, stools and skin. ▪ Control: Body sodium content is most intimately coupled with extracellular water content. Water and Na are like olden days couple. Water balance, not sodium balance, usually determines its concentration. When the sodium concentration increases, the resultant higher plasma osmolality causes increased thirst and increased secretion of ADH, which leads to renal conservation of water. During hyponatremia, the decrease in plasma osmolality stops ADH secretion, and consequent renal water excretion leads to an increase in the sodium concentration. Renal sodium regulation plays a major role in sodium homeostasis.

- **What is the priority in a conflicting situation?** Correction of volume depletion takes priority over osmolarity. Volume depletion stimulates ADH secretion even when there is hyponatremia. E.g. Hyponatremic dehydration in acute diarrhea. Once dehydration is corrected with NS, ADH is switched off, water retention ceases and serum sodium levels raises.

Algorithmic approach to hyponatremia

1. Does the child have neurological symptoms?

2. True or pseudo hyponatremia?

3. Is it acute or chronic?

4. Volume status?

5. Urine sodium levels

Explanatory notes for algorithm

Step 1 – Does the child have neurological symptoms?

The clinical presentation of hyponatremia is usually variable, nonspecific and when severe presents with symptoms of cerebral edema and those of underlying disease as noted below:

<130mEq - Apathy, anorexia, nausea, vomiting

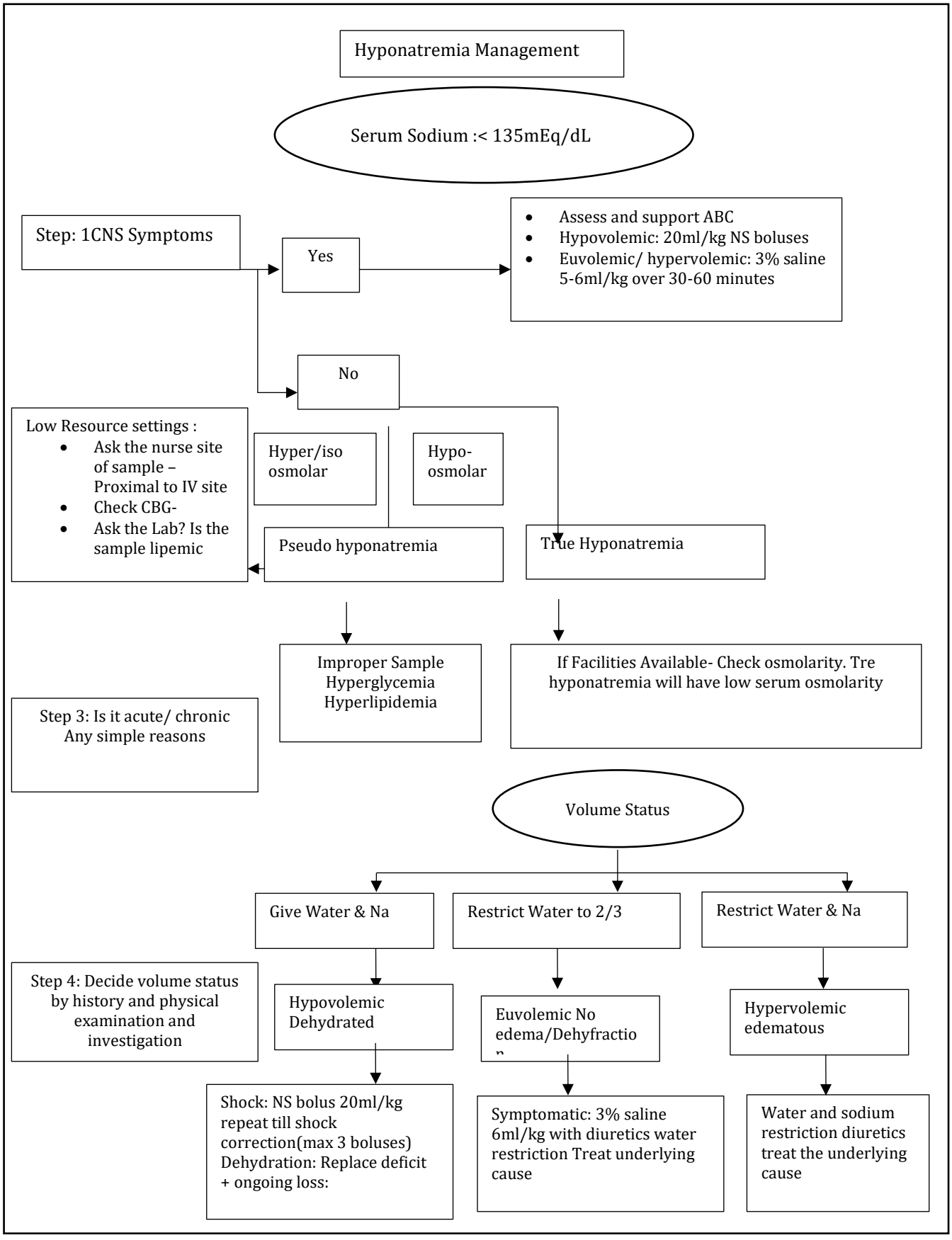
S. Sodium <120 mEq/dl - Muscular twitching, headache, seizures, coma.

Symptoms depend upon the degree and chronicity of hyponatremia. Patients with mild-to-moderate hyponatremia (greater than 120 mEq/L) or gradual decrease in sodium (greater than 48 hours) have minimal symptoms. Patients with severe hyponatremia (less than 120 mEq/L) or rapid decrease in sodium levels have multiple varied symptoms. Symptoms can range from anorexia, nausea and vomiting, fatigue, headache, and muscle cramps to altered mental status, agitation, seizures, and even coma.

Patients with neurological symptoms and signs need to be treated promptly to prevent permanent neurological damage.[18]

Symptomatic hyponatremia

When the child has neurological symptoms, regardless of underlying cause or volume status, or duration of the illness the deficit must be corrected to a safer level within 1-2 hours.


Hyponatremia Management
Serum Sodium :< 135mEq/dL
Step: 1CNS Symptoms
Yes
Assess and support ABC
Hypovolemic: 20ml/kg NS boluses
Euvolemic/ hypervolemic: 3% saline 5-6ml/kg over 30-60 minutes
No
Low Resource settings :
Ask the nurse site of sample – Proximal to IV site
Check CBG-
Ask the Lab? Is the sample lipemic
Hyper/iso osmolar
Hypo-osmolar
Pseudo hyponatremia
True Hyponatremia
Improper Sample
Hyperglycemia
Hyperlipidemia
If Facilities Available- Check osmolarity. Tre hyponatremia will have low serum osmolarity
Step 3: Is it acute/ chronic
Any simple reasons
Volume Status
Give Water & Na
Restrict Water to 2/3
Restrict Water & Na
Step 4: Decide volume status by history and physical examination and investigation
Hypovolemic
Dehydrated
Euvolemic No edema/Dehyfractio
Hypervolemic edematous
Shock: NS bolus 20ml/kg repeat till shock correction(max 3 boluses)
Dehydration: Replace deficit + ongoing loss:
Symptomatic: 3% saline 6ml/kg with diuretics water restriction Treat underlying cause
Water and sodium restriction diuretics treat the underlying cause

Safer levels mean 5 mEq above the current measured level. Eg. If measured Serum Sodium is 115mEq/dL, increasing it to 120mEq/dL is advised and definitely not raised to normal range immediately.

Clinical situation 1: In the presence of hypovolemic hyponatremia (acute diarrheal dehydration) and seizures -NS boluses in 20ml/kg aliquots is given, aiming at simultaneous volume and sodium correction. In the presence of dehydration/shock, both volume and sodium replacement is required. Once hypovolemia is corrected, ADH secretion is switched off and hyponatremia gets corrected.

Clinical situation 2: In normovolemic and hypervolemic hyponatremia with seizures, correction is done by using 3% saline 5mL/kg over 30 – 60 minutes. An increase of 5mEq/L over 2 hours is enough to tide over the crisis

Eg: 1 year old child, weighing 10 kg, serum Sodium is 110mEq/dL. Child is having seizures. In addition to management of seizures, we should raise the serum Sodium by 5 mEq/L. 60 ml of 3% saline is to be infused over 30-60 min. Close clinical monitoring and frequent (every 2-4 hrs) electrolyte estimation is mandatory. After the initial therapy, replacement is continued as that for an asymptomatic child.

Step 2 -If asymptomatic see whether it is pseudo hyponatremia? Confirm whether hyponatremia is true or false. Usually hyponatremia is associated with hypoosmolality. If facilities are available, check the serum osmolarity. If it is low it is true, if it remains normal or high it is pseudohyponatremia. If serum osmolarity is unavailable, simple history and examination can identify pseudohyponatremia.

Methods to identify pseudohyponatremia

- Improper sample – ask the nurse or resident whether sample was taken proximal to the IV cannula site. Blood obtained from a vein proximal to an infusion of hypotonic saline (1/2 GNS) will have a low sodium.
- Ask the lab persons whether the sample is lipemic. Presence of hyperlipidaemia can be identified as a cause of pseudohyponatremia. Pseudohyponatremia is a laboratory artifact seen in hyperlipidaemia and hyperproteinaemia where the serum osmolarity is normal. Hyperlipidaemia is suspected when serum is lipemic. Hyperproteinaemia is very rare in children.
- Check blood glucose by glucometer, which will identify hyperglycemia as a cause. Each 100mg raise of blood sugar will decrease serum sodium by 1.6 mEq/L Hyponatremia associated with hyperglycemia generally resolves as hyperglycemia is corrected. In hyperglycemia the serum osmolarity is high.

- Ask for the history of mannitol therapy, which also may be a cause for pseudohyponatremia. Step 3: Further evaluation when the child is asymptomatic and it is true hyponatremia If it is true hyponatremia, volume status needs to be evaluated based on history and physical examination

History: The following should be looked for: Diarrhea, vomiting, polyuria, oliguria, edema, breathlessness, altered level of consciousness or convulsions, any surgical procedure done, drugs and IV fluids administered.

Physical Examination: One has to look for signs of dehydration, features of shock, S3 gallop, respiratory distress, ascites, edema, pigmentation, stigmata of liver or renal disease and bony deformity suggestive of rickets. Genital examination is mandatory to look for signs of congenital adrenal hyperplasia.

Investigations needed: Serum electrolytes, glucose, urea, creatinine, chloride, x-ray chest, serum osmolarity, Urine osmolarity and urine sodium are the most useful investigations

Urine Na 20 mEq / L indicates extra renal loss

Urine Na 20 mEq / L indicates renal loss

HYPOVOLEMIC HYPONATREMIA

In hypovolemic hyponatremia, one should elicit history to identify cause of fluid loss. If child has vomiting/diarrhea/significant nasogastric tube aspirate - GI loss If child has polyuria or voiding urine despite dehydration-, consider renal loss. If the history does not point toward GI or renal loss – consider cutaneous loss, ask for excessive sweating.

a) GI LOSS:

If there is dehydration, correction needs replacement of water and sodium. So in a child with symptomatic hyponatremia and dehydration, correct with Normal saline bolus 20 mL/kg every 20-60 mins depending on the hydration status (20ml/kg aliquots will provide 5 mEq/kg).

- How? Eg – 10 kg child will receive 200 ml (20 mL/kg). Each 100 ml contains 15 meq and 200 mL will provide 30 mEq.
- How much sodium is needed to raise the serum level by 1 mEq? 10 x 0.6 x 1 = 6 mEq. Hence when we provide 200ml of NS, it will raise the serum sodium level by 5 mEq/L. For example, serum level increases from 115mEq/L to 120 mEq/L.
- In short, correct the dehydration with NS, hyponatremia will get corrected automatically

b) CUTANEOUS LOSS:

Because of excessive loss of sodium and chloride through the skin, from sweating, hyponatremia can occur and it can be worsened in those who consume plain water without electrolytes. This is more commonly seen in hot climates. In cutaneous loss as in cystic fibrosis and in marathon running, degree of dehydration will be mild and child will be mostly asymptomatic. In cystic fibrosis, metabolic alkalosis and hypokalemia are associated findings. Hence this can be corrected by replacing sodium by oral route either as salt containing oral fluid, dietary preparations such as butter milk, coconut water, vegetable soup, rasam (spicy soup containing salt, used with rice) or electrolyte solution such as ORS. Otherwise, it can be corrected by replacing maintenance fluid as isotonic fluid like normal saline in addition to oral supplements, periodically monitoring serum sodium levels. Drinking plain water should be replaced by electrolyte solution to prevent hyponatremia. Rapid

correction may not be necessary in this situation

c) URINARY LOSS:

Rapidity of correction depends on the severity of dehydration. If the child is hospitalised, child can be managed with normal saline depending on the severity. For example: mild dehydration 30-50 ml/kg, moderate dehydration 50-70ml/kg This deficit is to be combined with administration of maintenance fluid simultaneously and given over 24-48 hrs.

EUVOLEMIC/HYPERVOLEMIC DEHYDRATION

In the presence of symptomatic hyponatremia, hypertonic saline (3% sodium chloride) 5- 6 mL/kg is preferred.

Example: A 10 kg child will require 60 mL 3%saline infusion over 30 – 60 mins (depending on the urgency). This will not cause volume overload. ▪ How much sodium this will provide? 60 mL of 3% saline will provide 30 mEq (1 mL of 3% saline gives 0.5 mEq of sodium) How much sodium is needed to raise the serum level by 1 mEq? Eg. Baby weight 10 kg. 10 x 0.6 x1 = 6 mEq. Hence when we provide 30 mEq, it will raise the serum level by 5 mEq.

Hypovolemic Hyponatremia	Euvolemic Hyponatremia	Hypervolemic Hyponatremia
Dehydration present Causes 1.Extra renal loss – GI -Vomiting, diarrhea - significant nasogastric aspirate 2. Renal Loss -Renal Tubular Acidosis (RTA), osmotic diuresis (Diabetic ketoacidosis), diuretic therapy, adrenal insufficiency If history does not point toward GI or renal loss – consider cutaneous loss ask for excessive sweating/ manifesting in summer. 3. Cerebral salt wasting syndrome Investigation Urine Na >20mEq/L– Renal cause Urine Na < 20 mEq/L- Non Renal ↓Na ↓K ↑ cl – RTA ↓Na ↑K ↓glucose-Adrenal insufficiency	**No Dehydration no edema** Causes 1. Water intoxication (Use of 5% Dextrose in post operative Period). 2.Psychogenic water drinking 3.SIADH Investigation Urine Na > 20 mEq/L – SIADH Cerebral salt wasting Urine Na< 20 mEq/L – water intoxication Psychogenic water drinking	**Edema Present** Causes 1. Renal failure 2. Nephrotic syndrome 3. Congestive heart failure 4. Protein energy malnutrition 5. Cirrhosis liver Investigation Urine Na > 20 mEq/L – Renal failure Urine Na < 20 mEq – all

Most of the time hyponatremia is managed with clinical decisions supported by basic investigations.

Case Scenario 1

A 1-year-old developmentally normal, previously healthy baby boy is admitted with Lobar Pneumonia with fever, cough and tachypnea. CxR confirms Pneumonia. Baby is started on IV Ceftriaxone and IV maintenance fluids. On day 2 of admission, baby has decreased urine output (0.4 ml/kg/hr) for last 24 hours. Clinically perfusion is normal with no signs of dehydration and no h/o of vomiting/ diarrhoea. There is no edema/ hypertension. Urinary bladder is not distended. Rest of the examination is unremarkable apart from respiratory signs of Pneumonia. He has normal sensorium with no neurological deficit.

Lab Results:

Serum studies

Sodium 126 mEq/L BUN 4 mg/dL
Chloride 98 mEq/L Creatinine 0.4 mg/dL
Potassium 3.7 mEq/L Glucose 129 mg/dL
Bicarbonate 25 mEq/L

Urine studies
Specific gravity 1.035

Ultrasound Abdomen (KUB)

Normal

1. What is the likely cause of oliguria and hyponatremia?
2. What test will you do to confirm the diagnosis?
3. How will you manage the child further?

In spite of starting the correct management, baby develops generalised tonic clonic seizures refractory to IV Lorazepam and IV Phenytoin. ABG done shows Sodium of 116.

1. What will be your immediate management?
2. How will you manage the baby further after seizure stops?
3. What will you monitor (clinical and lab)?

What are the clinical features of the dreaded complication that can happen if you correct very fast?

Case scenario 2

3 year old female, weighing 10kg was brought to with complaints of fever for 3 weeks associated with bifrontal headache and GTCS refractory to lorazepam and levetiracetam and fosphenytoin, hence she was intubated and mechanically ventilated. CSF study and MRI was suggestive of Tubercular Meningitis. Antituberculosis treatment (ATT) along with intravenous steroids were started. Ventriculoperitoneal (VP) shunt was placed in view of obstructive hydrocephalus. Her admission serum sodium -132. On day 2 serum sodium -121meq/l). vitals HR-100/min, Spo2-99%, ABP-100/76 mmHg, UO-2.8ml/kg/hr.

Q: How will you approach this low sodium? What is your immediate management?

Case scenario progresses:

Fluid restriction was done to 2/3rd maintenance and 3% saline was continued at 1ml/kg/hr to maintain normal serum sodium levels as a part of neuroprotective care. EEG monitoring revealed no seizures. Serum sodium was monitored 6hrly and Na transiently improved to 125 meq/l, but subsequently by day 4, the serum sodium gradually reduced to 123, 121 and 119 meq/l?

Q: What is the reason for hyponatremia despite 3% saline? What additional test will you do to confirm your diagnosis?

Q: Based on reports, How will you calculate fluid prescription for this child weighing 10kg to correct hyponatremia?

Q: What will you need to monitor?

Case scenario progression: She continued to have persistent hyponatremia with polyuria despite sodium replacement

Q: How will you manage refractory hyponatremia in this specific scenario?

Case progression:

After 10 days of hospitalisation, polyuria improved though serum sodium levels showed fluctuations requiring further fluid and medication adjustment. The child finally improved after 24days of treatment and got discharged.

Case scenario 3

A 5-year-old female child, known case of Steroid dependent nephrotic syndrome, on mycophenolate mofetil, presented with edema. Parents had stopped medication on their own for last 1 month. No e/o infection.

On examination, child irritable and anxious with strangers. Height - 108 cm(25-50 th centile), weight - 27 kg(> 97th centile), dry weight – 23 kg , BP - 114/84mmHg (95th centile), HR - 74 bpm, CFT < 2 sec, pulses well felt, RR - 33/min.

There is generalised anasarca. Respiratory system exam reveals reduced air entry on the right side. There is free fluid in the abdomen. Other systems are normal.

Investigations done on admission:

S.Creatinine	0.4
B urea	23
S. Sodium	28
S. Potassium	4.1
S. Chloride	102
S. Bicarbonate	22
S albumin	2.1
S. cholesterol	343
CBG	85
CBC	Normal

Q1. What is the metabolic abnormality in this child?

Q2. What further investigations will you order?

Q3. What are the steps in the management?

FLUIDS IN SPECIAL SITUATIONS

Contributors: Dr Thangavelu Dr Sudarsan K, Dr Murali T, Dr Chidhambharam L

Essential facts

- Fluid therapy is a dynamic process on constant flux
- Monitoring at the bedside and constant adjustments are necessary especiallyin a sick child
- Resuscitation is based on intravascular fluid status
- Restoration to baseline is based on ECF status and finally total body water

What is different in fluid Mx in SAM?

- Recognizing signs of dehydration
- Calculating volume of fluid to be replaced
- Determining the best electrolyte composition
- Associated comorbidities

STEP 1

Assessment of fluid status

- Change in weight if available is the best
- Clinical assessment
- Input / output charts
- Measurement of ongoing losses (Urine, drains)

If hypotensive → Resuscitation

STEP 2

Calculation of deficit

Determines Volume of replacement

- Expressed as percentage of body weight
- Based on all parameters in step 1
- Can add anticipated losses if deemed necessary

STEP 3

Identify associated electrolyte disturbance
Determines composition of replacement fluid

- Most important is sodium
- Potassium , Magnesium
- Phosphate

STEP 4

Attention to co morbidity / disease pathology
Determines rate of correction of replacement fluid

- Anemia
- Severe acute malnutrition (SAM)
- Cardiac function
- Renal function
- Cerebral edema
- Sepsis / Capillary leak / vasoplegia

STEP 5

Prescription based on all 3 above Volume, composition, rate

STEP 1: Assessment of fluid status

- Weight difference
- Calculate input and output meticulously
- Do not forget ongoing losses, losses through drains, urine
- Thorough clinical examination, frequently re-assess

Remember, in SAM.

- ✓ Edema may mask dehydration
- ✓ Altered skin turgor in v/o SAM
- ✓ Irritability, lethargy per se due to SAM

In a SAM child, Diarrhea + 2 of lethargy/sunken eyes/very slow skin pinch suggests **severe** dehydration

STEP 2: Calculating deficit

- % change in body weight
- Input-Output difference, estimate of dehydration, electrolyte changes
- Ensure all losses are taken into account

- Both over and under estimation of severity common

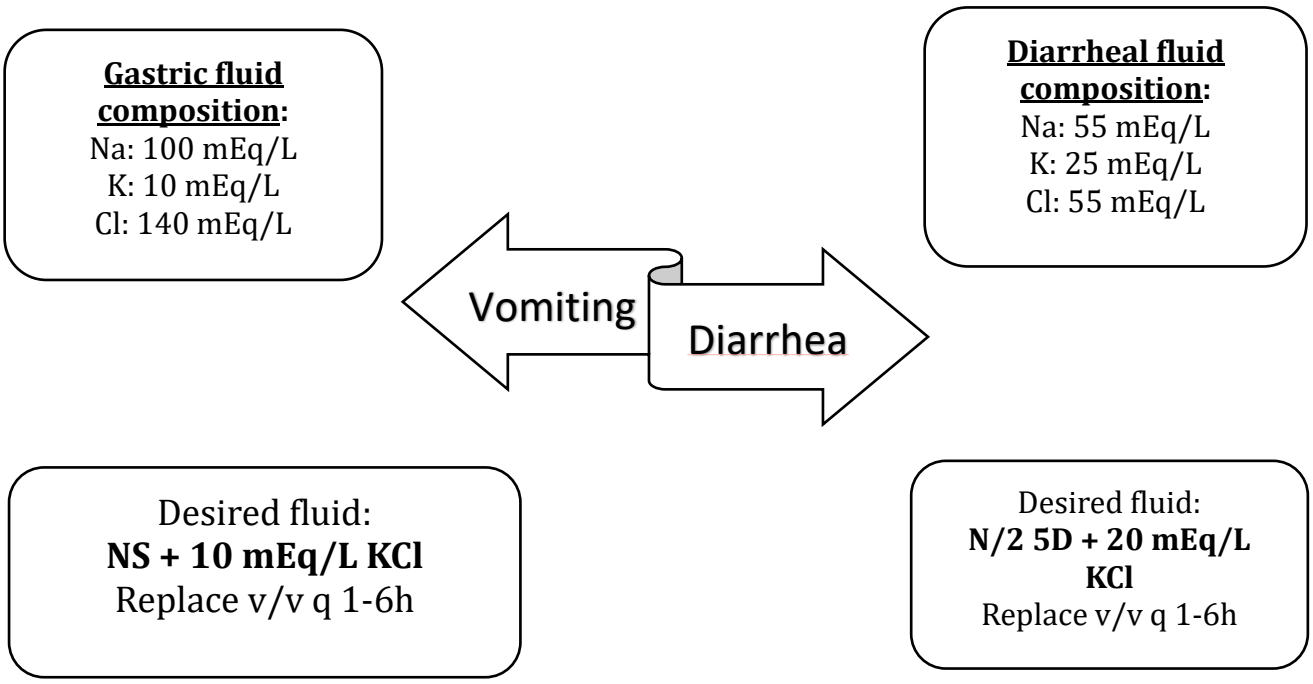
STEP 3: Identify electrolyte disturbances

- Actively look for electrolyte disturbances so that appropriate fluid can be chosen
    - ✓ Hypernatremia
    - ✓ Hypokalemia
    - ✓ Hypomagnesemia
    - ✓ Hypophosphatemia

Composition of commonly available fluids

IVF (/L)	Na (mEq/L)	Cl	K	Ca	Mg	Glucose(G/L)	Osm (mOsm/L)
NS	154	154	-	-	-	-	308
DNS	154	154	-	-	-	50	560
N/2S	77	77	-	-	-	-	155
N/2 5D	77	77	-	-	-	50	405
RL	130	109	4	3	-	-	273
Plasmalyte	140	98	5	0	3	-	294
Isolyte P	23	29	20	-	3	50	340
ORS	75	65	20	-	-	75	245
ReSoMak	45	70	40	-	3	25	300

Choice of replacement fluid



STEP 4: Associated comorbidities

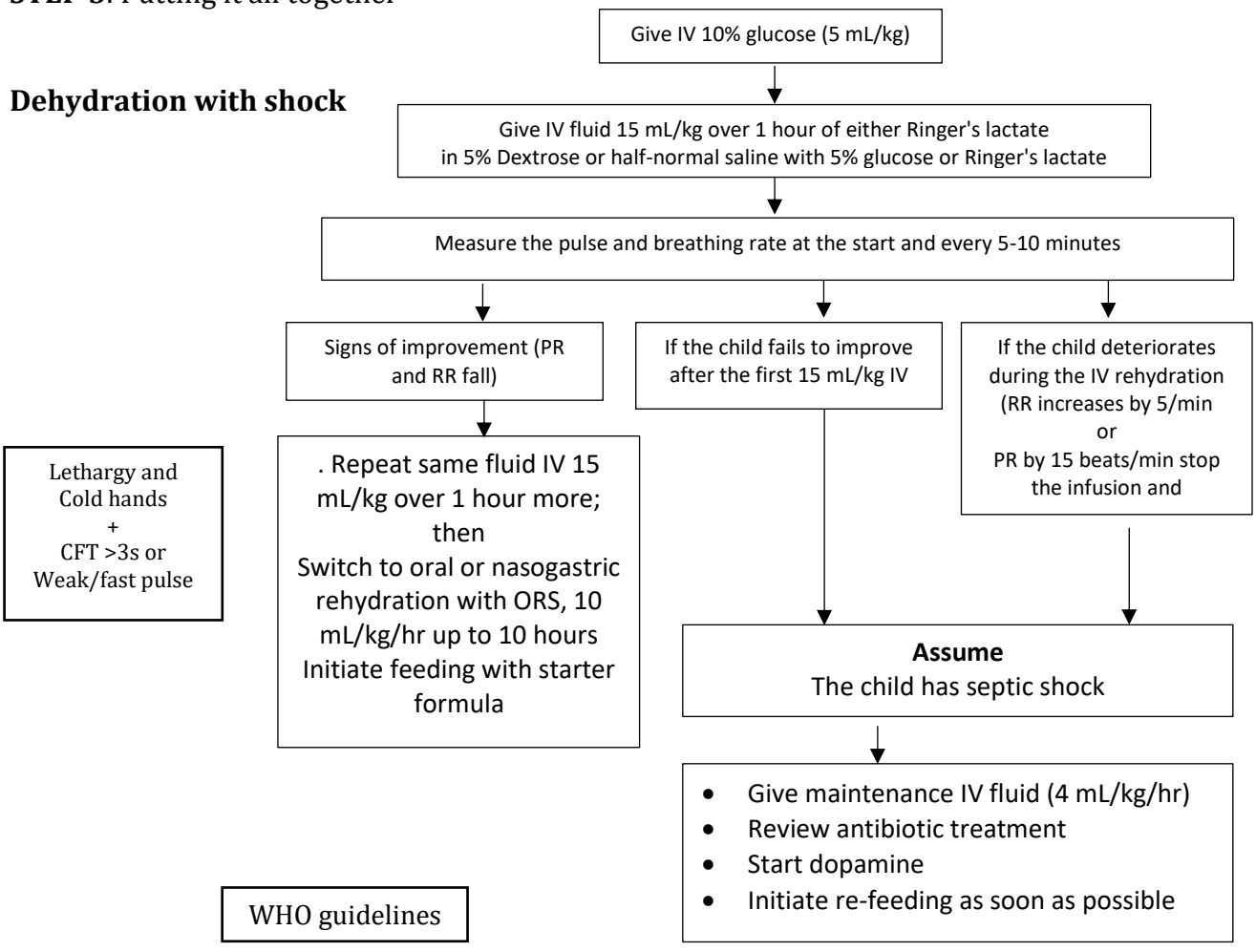
- Help determine the rate of correction
- Caution if associated
- Severe anemia
- CCF
- Renal dysfunction
- Cerebral edema
- Capillary leak

Evaluate for

- ✓ Hypoglycemia
- ✓ Hypothermia
- ✓ Infections

STEP 5: Putting it all together

Dehydration with shock



Dehydration with shock

- Empirical Abx: Ceftriaxone/Cefotaxime for 7-14d
- KCl: 3–4 mEq/kg/d for 14 days
- $MgSO_4$: 0.3 mL/kg (max 2 mL) im once then 0.2-0.3 mL/kg orally for 2 wks
- Food without added salt to avoid Na
- Do not treat edema with diuretics
- Multivitamin, Vit A, Zinc, Folate, Iron
- Feeding advise (F75 → F100)

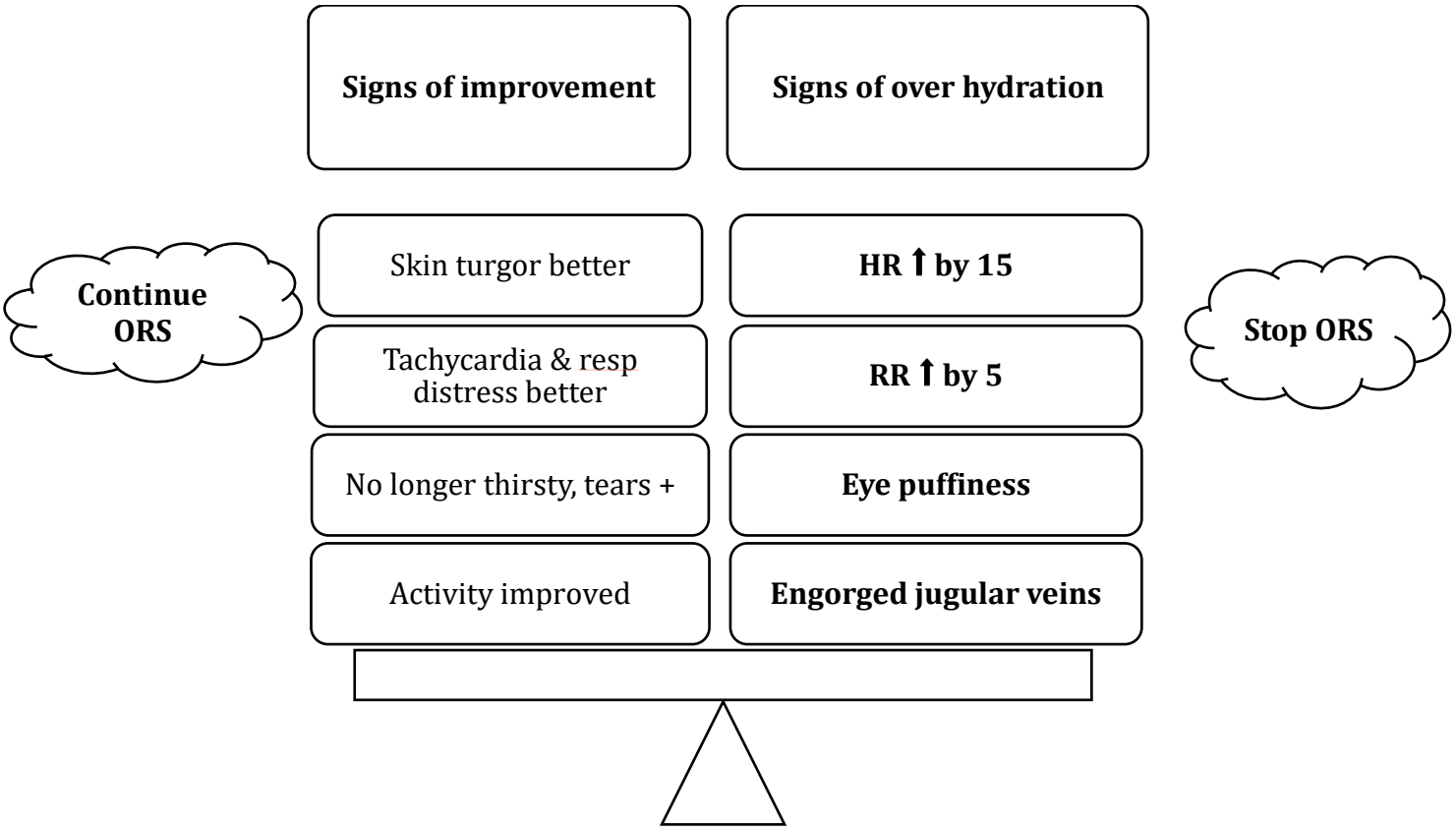
Dehydration without shock

- ORS 5 mL/kg q 30 min for first 2h f/b
- ORS 5-10 mL/kg q2h till rehydrated (max 10 h)
- Add 20 mEq/L (15 mL) KCl to 1L ORS
- Replace ongoing loss (30-50/100 mL per stool in <2/>2y)
- Continue breast feeding, start F-75 diet simultaneously

Home made ReSoMal
1 packet ORS (has 20 mEq K)
2L water
45 ml KCl syp (60 mEq K)

Monitoring

- Every 30 min initially till stabilized then q1-2h



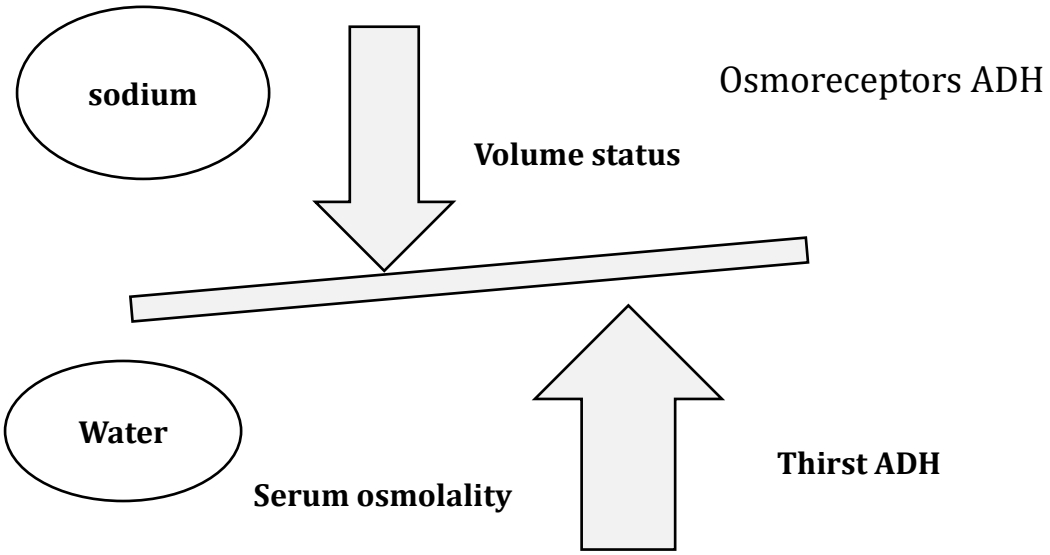
Case scenario

- 15 mo old Ramu is brought to OPD with diarrhea for the last 3 days- watery, 10 episodes/day, not blood stained. Mother says he has been less active. O/E: sunken eyes, cold peripheries, HR 172/min, CFT 4s. He has lustre less hair with protuberant abdomen and visible wasting
- Diagnosis with severity?
- Management?

Tips and tricks

- Presume severe dehydration even if moderate dehydration clinically in a SAM child
- Over and under estimation common; reassess frequently
- Give smaller volumes
- Correct slowly
- Prefer oral route
- Beware of electrolyte changes
- Continuous ongoing monitoring is vital

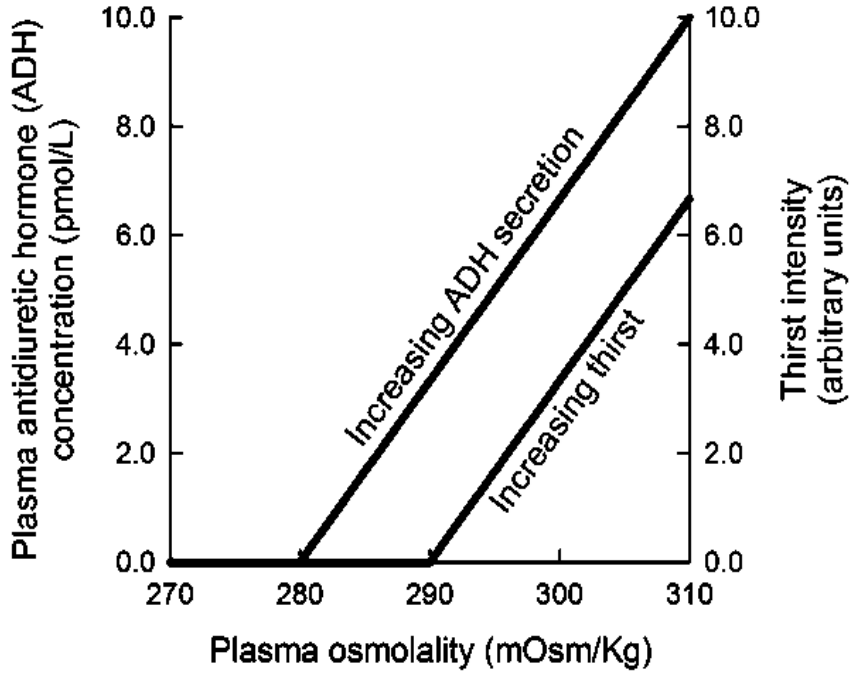
Hydration/ water homeostasis

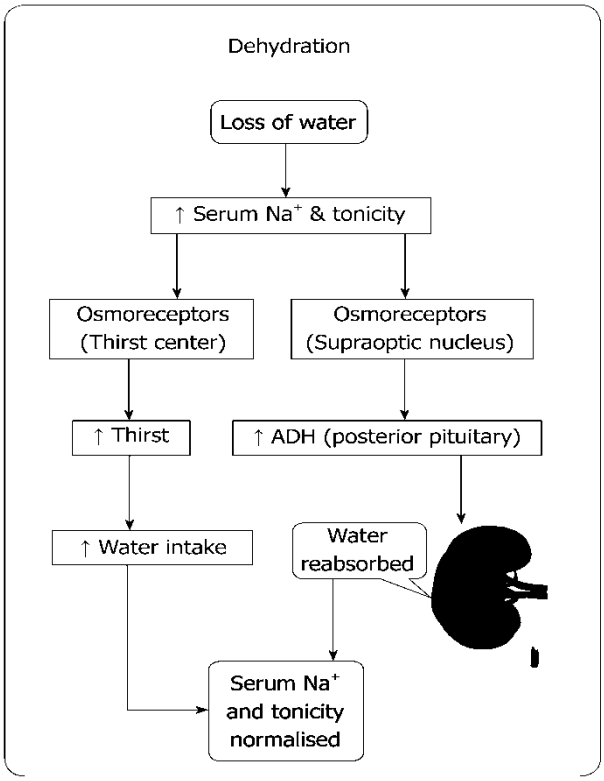


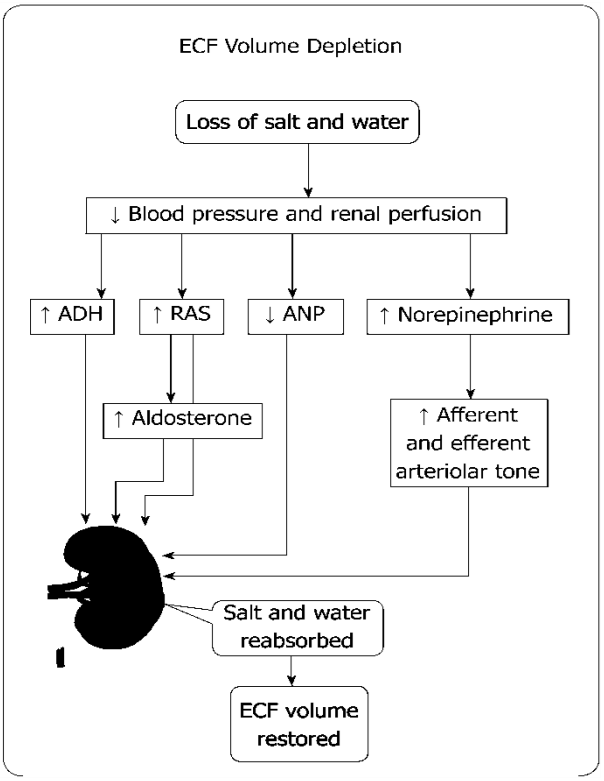
Dehydration

- Loss of normal fluid and electrolyte homeostasis

- Due to rapid and excessive fluid loss gastrointestinal tract (diarrhea and vomiting), skin (fever, sweat, burns), urine(glycosuria, diuretic therapy, obstructive uropathies, interstitial disease, neurogenic and nephrogenic diabetes insipidus).


Plasma antidiuretic hormone (ADH) concentration (pmol/L)
10.0
8.0
6.0
4.0
2.0
0.0
Increasing ADH secretion
Increasing thirst
Thirst intensity (arbitrary units)
270
280
290
300
310
Plasma osmolality (mOsm/Kg)


Dehydration
Loss of water
↑ Serum Na+ & tonicity
Osmoreceptors (Thirst center)
Osmoreceptors (Supraoptic nucleus)
↑ Thirst
↑ ADH (posterior pituitary)
↑ Water intake
Water reabsorbed
Serum Na+ and tonicity normalised


ECF Volume Depletion
Loss of salt and water
↓ Blood pressure and renal perfusion
↑ ADH
↑ RAS
↓ ANP
↑ Norepinephrine
↑ Aldosterone
↑ Afferent and efferent arteriolar tone
Salt and water reabsorbed
ECF volume restored

Isonatremic

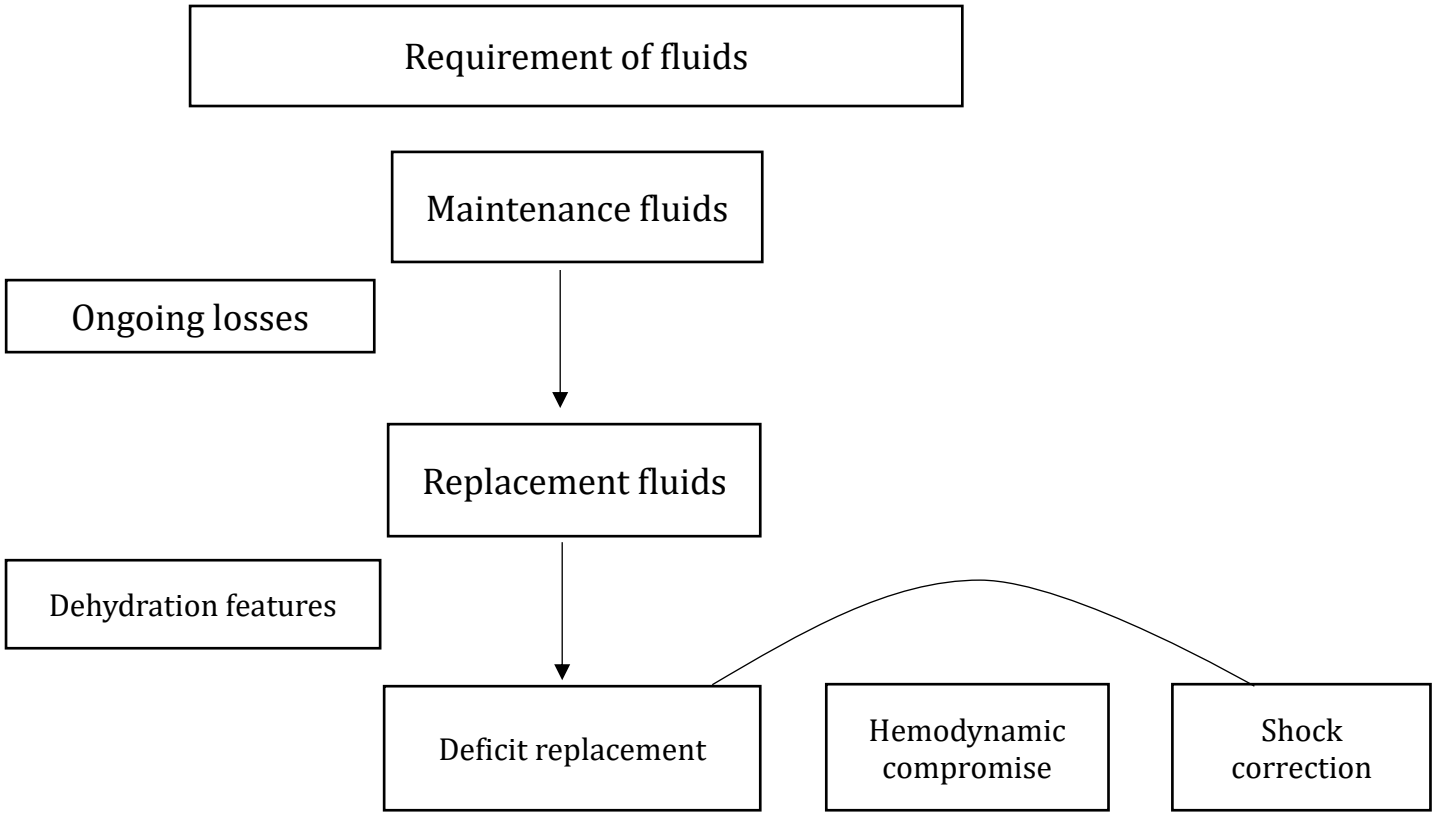
- Replace with isotonic Fluids – Rapid replacement

Hyponatremic

- Replace with isotonic fluids. Slow replacement (24 hrs)
- (Na < 120 – seizures – volume bolus – isotonic enough – hypertonic – Na overshoot)
- Post volume bolus – hypotonic fluids (N/2) to prevent rapid rise in Na

Hypernatremic

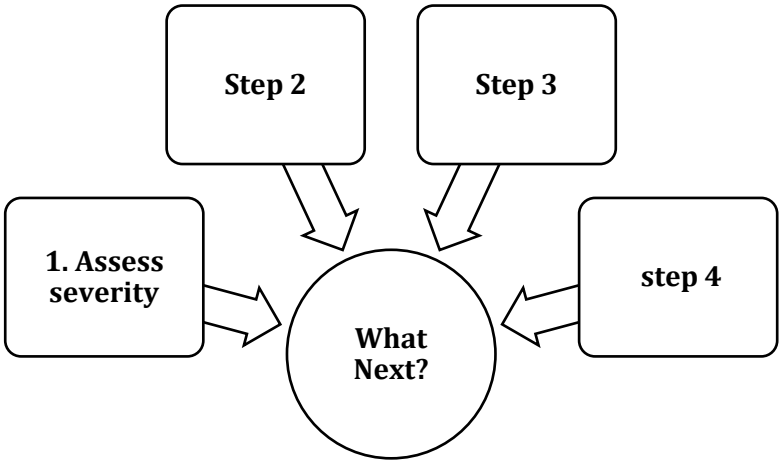
- Replace with isotonic fluids (to prevent rapid fall) . Slow replacement (24 hrs – 48 hours)
- If Na not falling with isotonic fluids as expected can use hypotonic fluids



CASE 1

- 6 years old
- Fever, vomiting 8-10 episodes
- Loose stools watery 5-6 episodes
- Poor oral intake, lethargic, urine output 2-3 times in last 10 hours
- Hr – 130 / min , BP – 100/80 , Radial pulse felt , RR – 20 / min, sats – 96% , afebrile
- CFT – 2 secs

What next?

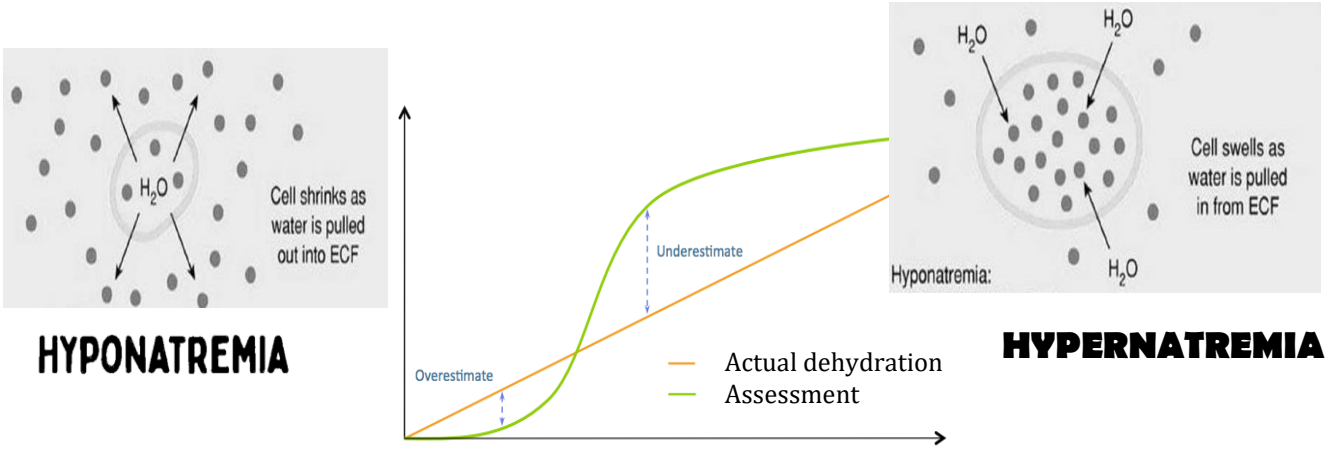


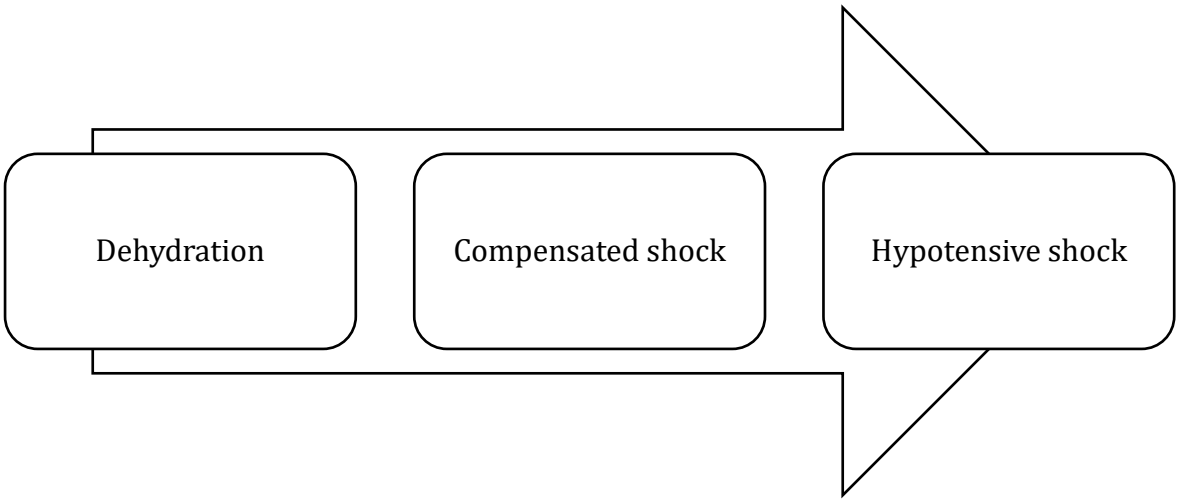
Step 1. Assess severity

Table 1. Degree of Fluid Deficit & Clinical Symptoms Associated with Dehydration

	Mild Dehydration	Moderate Dehydration	Severe Dehydration
Weight Loss Older child Infant	 3% (30 ml/kg) 5% (50 ml/kg)	 6% (60 ml/kg) 10% (100 ml/kg)	 9% (90 ml/kg) 15% (150 ml/kg)
Heart rate	Normal	Mildly increased	Marked tachycardia
Distal pulses	Normal	Slightly diminished	Weak, thready
Capillary refill	Normal	Approx. 2 seconds	>3 seconds
Urine output	Normal	Decreased	Anuria
Fontanelle	Flat	Soft	Sunken
Eyes	Normal	Normal	Sunken
Tearing	Normal	Diminished	Absent
Mucosa	Normal	Dry	Parched

Adapted from Gunn VL, Nechyba C. The Harriet Lane Handbook, 16th edition. 2002.

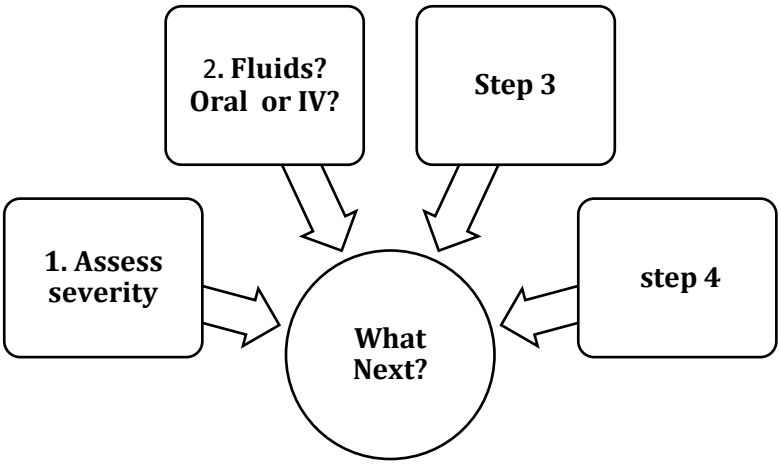




The index case

- Airway Stable
- Circulation: Tachycardic, perfusion good, Liver span N, BP normal, narrow pulse pressure
- Disability: lethargic
- Dehydration - moderate
- CBG: 62mg/dl
- Serum Na – 135

What Next?



Oral/ Intravenous?

1. SHOCK: Isotonic fluid IVF
2. MODERATE DEHYDRATION:
    - ORS
    - IVF as second choice.
    - Isotonic fluid throughout
3. NO/MILD: ORT
4. On admission– Check electrolytes
    - HCO3 < 16, hyponatremia, hypoglycemia
    - In hyperosmolar dehydration (DKA, Hypernatremia) signs of dehydration are absent, because dehydration is intracellular

Fluid replacement calculation for dehydration

Step 1: Deficit

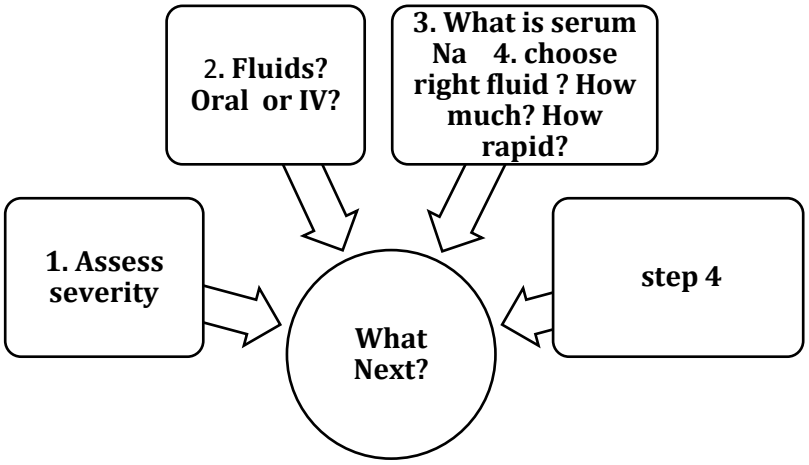
- Mild dehydration: 30-50ml/kg
- Moderate dehydration: 50-75ml/kg
- Severe dehydration: 70-100ml/kg

Step 2: Ongoing losses:

- replace one milliliter of fluid for every gram of output, stool, emesis, or urine.
- If measurements are not available, replacing 10 mL/kg body weight for each watery stool or 2 mL/kg body weight for each episode of emesis

Step 3: Maintenance

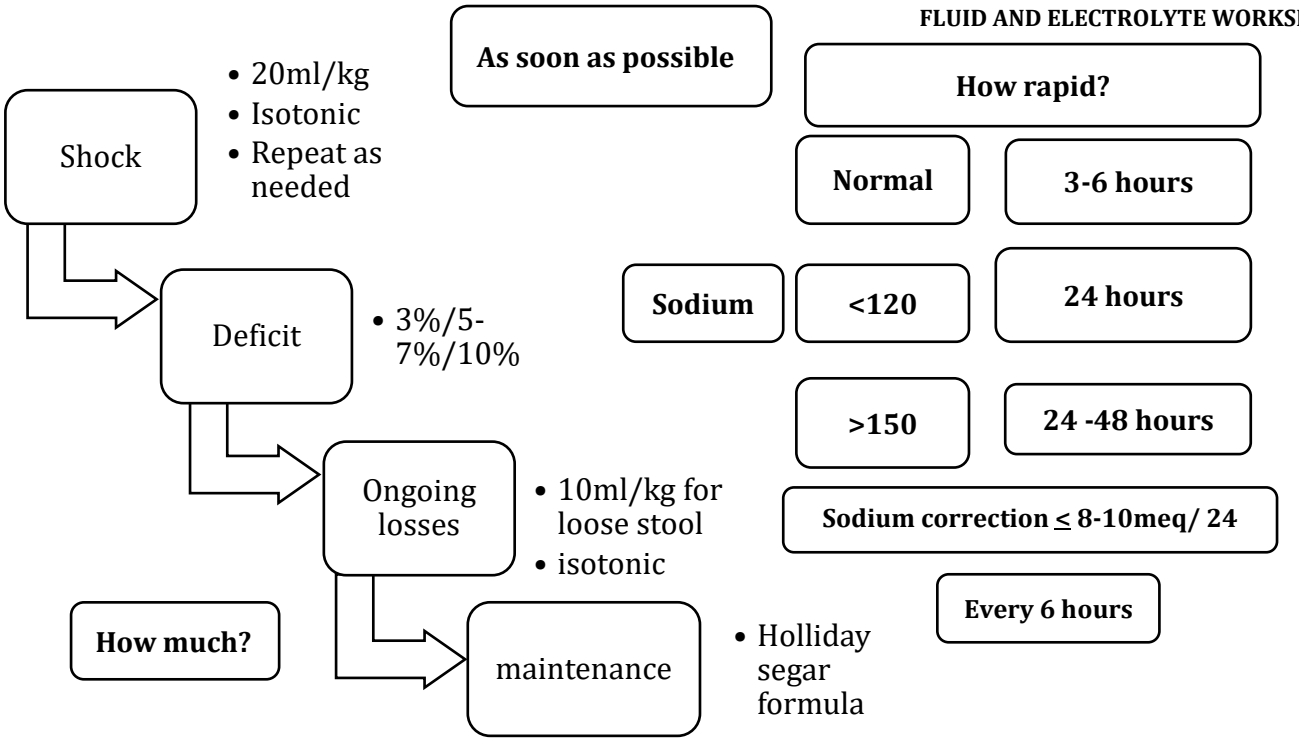
What next?



COMMON NAME	Normal Saline[1]	Hartmann's[2]	Plasma-Lyte[3]	D5W[4]	D10W	0.9% Saline with 5% Glucose	0.45% Saline with 5% Glucose	0.18% Saline with 4% Glucose	3% Saline
CHEMICAL NAME	0.9% Sodium Chloride	Compound Sodium Lactate	PL 148	5% Dextrose	10% Dextrose				
Na	154	129	140	0	0	154	77	31	513
K	0	5	5	0	0	0	0	0	0
Cl	154	109	98	0	0	0	77	31	513
Ca	0	2	0	0	0	0	0	0	0
Mg	0	0	1.5	0	0	0	0	0	0
Bic	0	29 (as lactate)	27 (acetate) 23 (gluconate)	0	0	0	0	0	0
Gluc	0	0	0	50 g	100g	50 g	50 g	40 g	0

What fluid-RL/NS

- Numerous normal saline fluid boluses may result in a hyperchloremic non-anion gap metabolic acidosis, which may obscure acidosis secondary to poor tissue perfusion.
- Lactated Ringer's solution has the theoretical benefit of producing bicarbonate from lactate, provided that liver function is normal



IAP – STG Watery diarrhea(isonatremic dehydration)

Table 1: Rehydration therapy in acute diarrhea

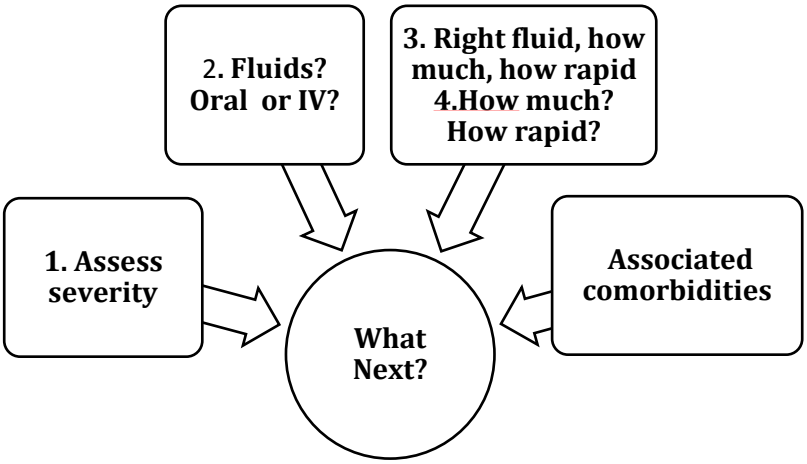
Treatment Plan	**Plan – A**	**Plan – B**	**Plan – C**
State of hydration	No dehydration	Some dehydration	**Severe dehydration**
Percentage of body weight loss	<5	5-10	>10
Estimated fluid deficit (mL/kg)	<50	50- 100	>100
Goals of management	Replacement of ongoing losses of fluid and electrolytes	Correction of existing deflicts of fluid and electrolytes	Urgent replacement of existing deflcits of fluid and electrolytes
Fluid therapy	Maintenance (oral)	Rehydration (oral)	Rehydration (intravenous (IV))
Treatment facility	Home	Health facility	Health facility
Rehydration fluid	Oral rehydration solution (ORS)/homemade solutions	ORS	RL*
Amount of rehydration fluid	For every loose stool: 10mL/kg Age up to 2 months- 5 teaspoons/ purge 2 months to < 2 years- 50 – 100 ml Older child: As much as desired Plus Free access to drinking water	75 mL/kg Over 4 hours Plus Non- breastfed infants <6 months- 100-200mL of clean drinking water. Older Children and adults: Freeaccess to plain water in addition to ORS	IV Fluid Infants 30ml/kg over 1hour 70ml/kg over 5 hours Age>1 year 30 ml/kg over ½ hour 70ml/kg over 2 ½ hours Plus ORS (5 ml/kg/h) start orally as soon as child is able to frink
Monitoring	Watch for vommiting, early signs of dehydration, blood in stools, etc	Monitor every hour and reassess after 4 hours • If still in plan B repeat as above • If rehydration, shift to plan A	Monitor ½ hourly and reassess after 6 hours(infants) 3 hours(older children) • If still in plan C, repeat as above • If rehydrated , shift to plan B/A

Normal saline (0.9% NacL) or half strength Darrow's solution may be used if Ringer Lactate (RL) is not available. Severely malnourished children rehydration slowly over 6- 12 hours.
In children who fail on oral rehydration administration of rehydration fluids either by nasogastric(NG) tube or intravenously (IV) is effective and recommended

Maintenance fluids

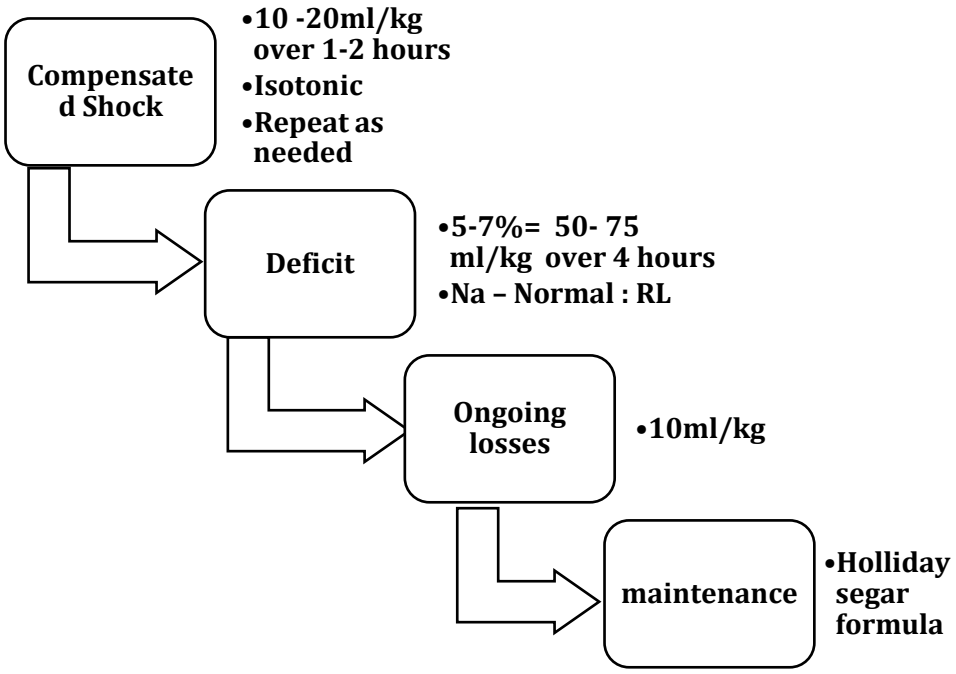
Body weight	Fluid per day
0-10kg	100ml/kg
11-20kg	1000ml+50ml/kg for each kg>10kg
>20kg	1500ml+ 20ml/kg for each kg>20kg

Body weight	Fluid per day
0-10kg	4ml/kg/hr
11-20kg	40ml/hr+2ml/kg/hr x (wt-10 kg)
>20kg	60ml/hr+ 1 ml/kg/hr x (wt-20kg)



Associated co-morbidities

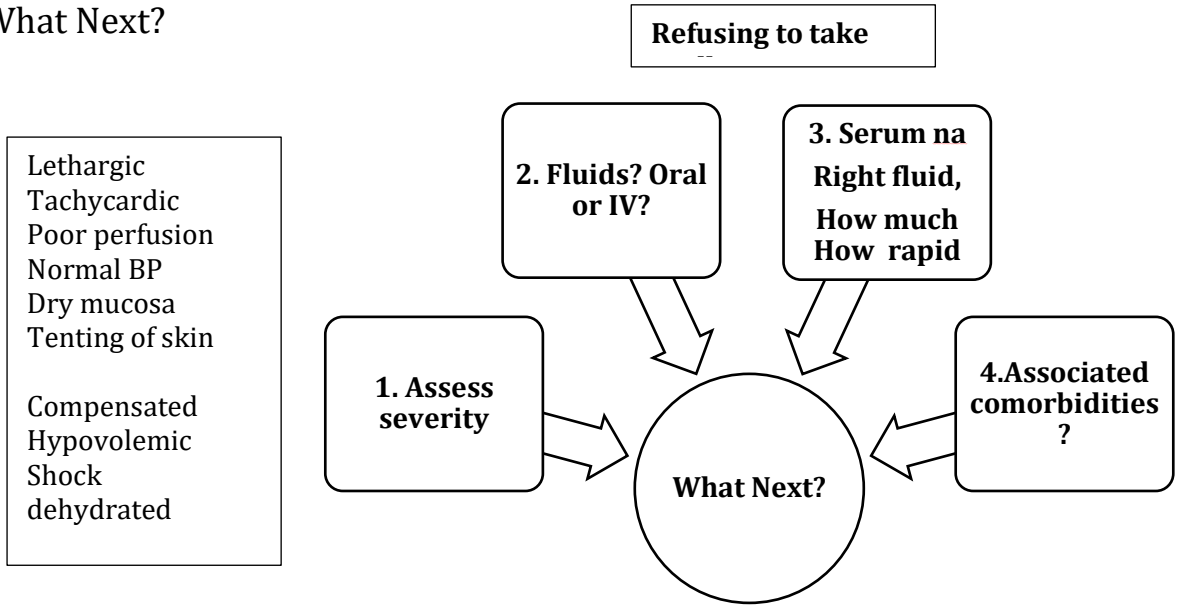
- Look for sepsis
- Adequacy of fluids
- Calculative errors
- Failure to thrive
- CNS abnormalities

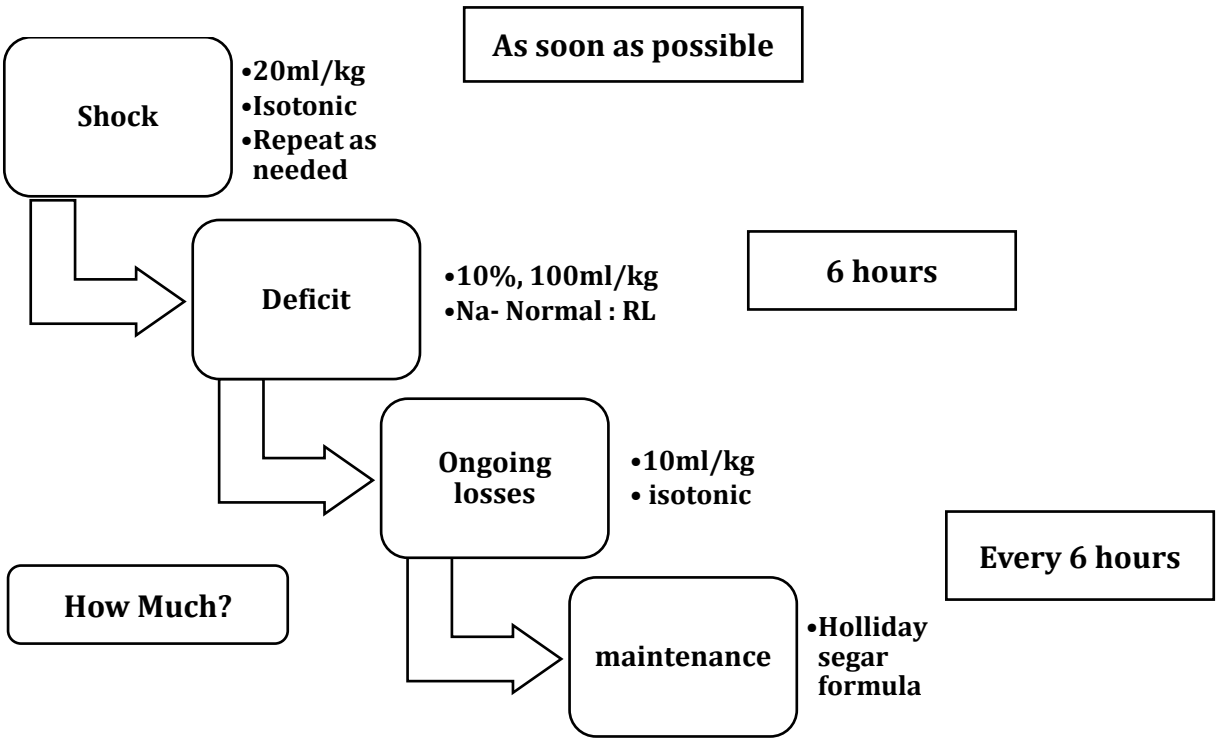


Case 2

A 10-month-old infant presents to the emergency department wita 4-day history of frequent watery stools. He is now refusing to drink. He is listless in his mother's arms. On physical examination, his mucous membranes are dry and the skin on his abdomen is tenting. His heart rate is 160 beats/min and blood pressure is 80/40 mm Hg. His current weight is 9 kg. One weck ago, when he was seen in clinic for a routine examination, he weighed 10 kg. His serum sodium measures 138 mEq/dL (138 mmol/L). After failing a trial of oral therapy, intravenous access is obtained and he is given 20 mL./kg (200 mL) of normal saline. Following the infusion, his heart rate, perfusion, and mental status improve.

What Next?

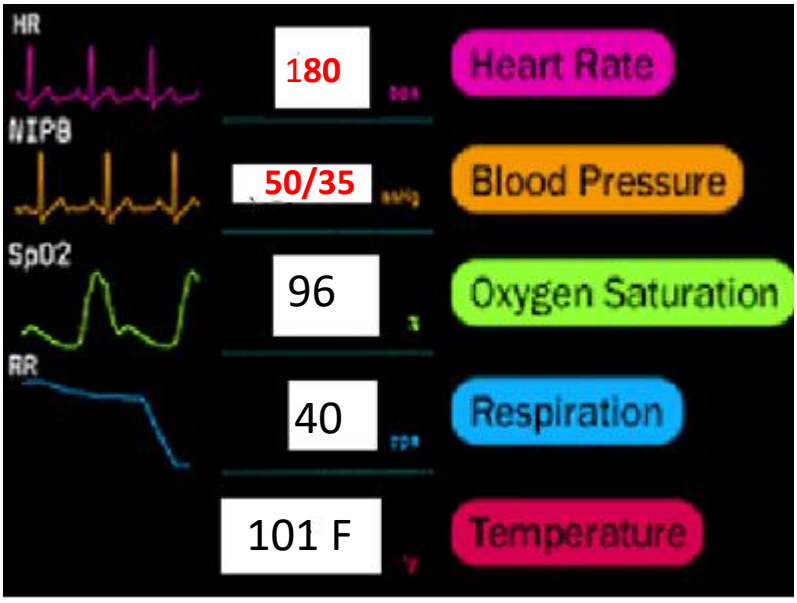




Case 3

8 Month male infant history of fever, loose stools, weight loss, posturing, irritability, reduced urine output

Irritable, bulging anterior fontanelle, intermittent posturing, doughy skin, systematic examinaton-normal.



Mother Revealed improper mixing of 2gm ORS packet in 200 ml water

Diagnosis : Hypernatremic dehydration , in shock , with altered sensorium ? CNS bleed

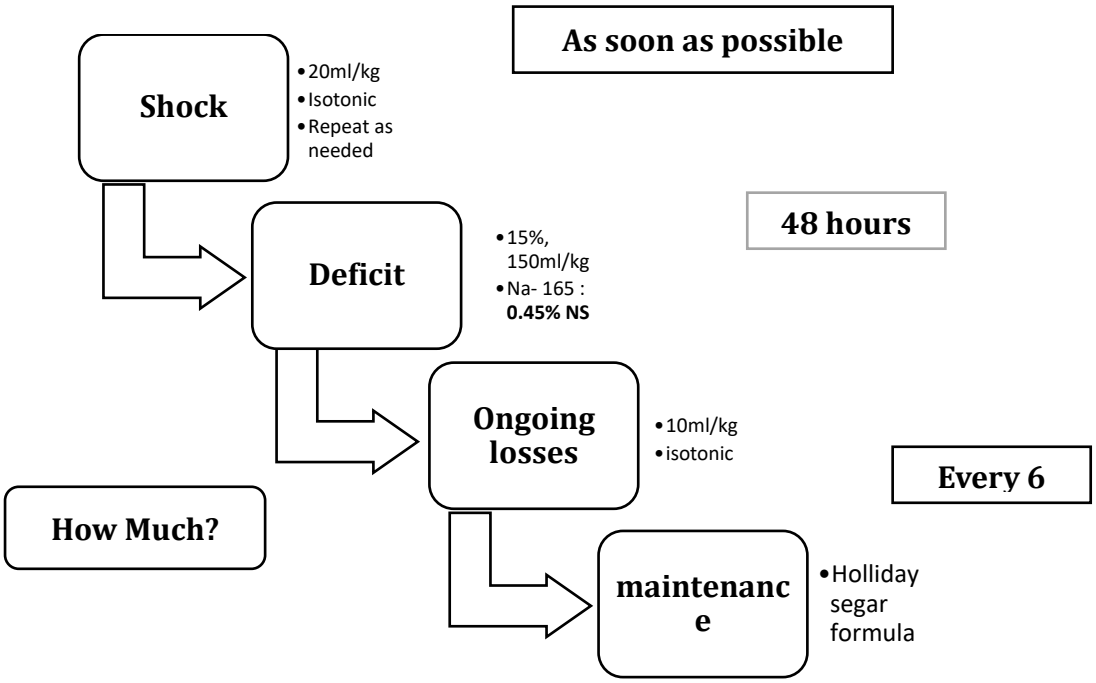
CBC : HB/TLC/PLATELET	**14/17700/3 LAKH**
Serum Sodium / K	**165 / 3**
Blood cultures sent	
INR/APTT	**1.3, 34/27**
Urea/ Creatinine	**60/0.5**

Weight at last vaccine visit : 10 kg

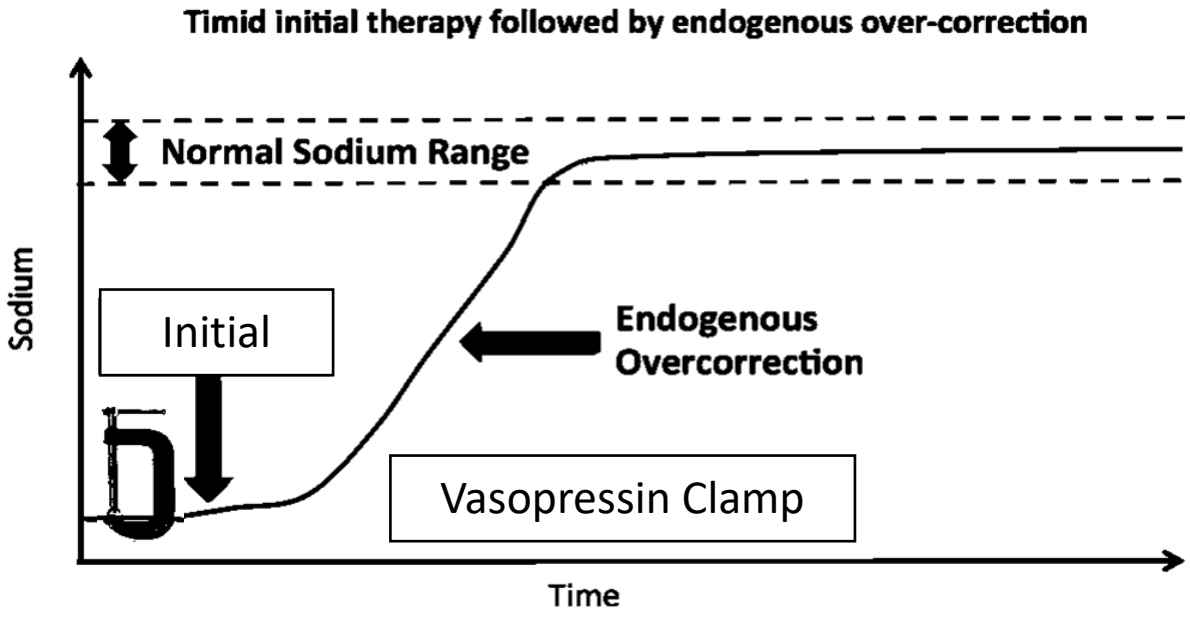
8 Month infant 10 Kg

RATE OF FLUID ADMINISTRATION

- Total fluid requirement = deficit and maintenance fluid , and expected drop of Na per day
- deficit = 15 % Severe Dehydration (because infant in shock) = 150*10 = 1500 over 48 hours
- Maintenance = 1000*2 = 2000 ml over 48 hours
- Total = 3.5 L – bolus 200 ml = 3.3 L over 48 hrs = 69 ml/hr



Endogenous overcorrection of Hyponatremia in hypovolemic hyponatremia



METABOLIC ACIDOSIS

Contributors: Dr Poovazhagi V, Dr Sivaraman D, Dr Dhakshayani R V, Dr Naresh Kumar S

A fall in pH is termed acidemia, and the underlying disorders that lead to acidemia is acidosis. A primary metabolic acidosis is a pathophysiologic state characterized by an arterial pH of less than 7.35 (acidemia) in the absence of an elevated PaCO2.

Traditional Henderson-Hasselbalch theory - [H+] = 24 x PaCO2/[HCO3 -]. a change in either HCO3 - or PaCO2 changes the other variable in the same direction (compensation) within certain limits. Limitations of this approach:

- Though it does show the change that occurs in Paco2 and Hco3 it does not necessarily state that they are the cause of the underlying acid base abnormality.

- The role of plasma proteins, specifically albumin, in acid–base balance is neglected.

The Modern Stewart Approach – The modern physical- chemical approach introduced in 1980 by Peter Stewart states that there are only 3 independent variables controlling H+ concentration and that changes in Hco3 and Paco2 are the consequence of these in an attempt at maintain pH in the normal range .

- PaCo2

- SID- Strong Ion Difference (strong cations – strong anions)
  (Na+K+Ca+ Mg) – (Cl+lactate) Normal – 40 mmol/L
- Narrowing of SID causes Acidosis and Widening of SID causes Alkalosis

- Atot – weak acids (albumin, phosphate)
  Albumin and phosphate act as weak acids the latter contributing to acidosis in renal failure. Hypoproteinemia causes a base excess.

Eg: Metabolic Acidosis occurring with large volume saline administration is because of excess chloride administration and narrowing of SID. When large volumes of saline are administered it has a proportionally greater effect on total body chloride than on sodium.

Metabolic Alkalosis with Vomiting occurs due to loss of Chloride. Replacement with Saline or Ringers Lactate corrects its

Classification of Lactic Acidosis (Cohen & Woods):

TYPE A: It occurs in hypoperfusion and hypoxia.

- Tissue hypoxia is seen in carbon monoxide poisoning, severe asthma and severe anemia.
- Hypoperfusion occurs in state of shock- cardiogenic, hemorrhagic, septic, regional

  (mesentric , limb) ischemia) , cardiac arrest

TYPE B: It occurs when there is NO clinical evidence of hypoperfusion.

It is further subdivided into 3 subtypes:-

- **B1 -** Acquired diseases - diabetes mellitus, seizures, ARDS , sepsis, malignancies, pheochromocytoma, post cardiopulmonary bypass, renal failure, thiamine deficiency, thyroid storm (all causing increased production)hepatic failure, (decreased clearance) etc.
- **B2 -** Medications and Toxins - acetaminophen, epinephrine, isoniazid, nitroprusside etc.
- **B3** - is due to Inborn errors of metabolism

Laboratory assays estimate only L- lactic acidosis. D-lactic acidosis is rare and is caused by d-stereo isomer of lactic acid which is synthesized by pathological gut flora

In Metabolic Acidosis, check for Anion Gap, Delta Gap, Osmolar Gap

Anion Gap (correct for low albumin)

NAGMA - Normal Anion gap acidosis (Cl high)

(diarrhoea, renal tubular acidosis, saline, acetazolamide)

HAGMA -Wide Anion Gap acidosis > 12 (Cl -N)

GOLDMARK mnemonic G- glycols,O- oxoproline, L- lactic acidosis, D- D lactate , Mmethanol, A-aspirin, R-renal failure, K-Ketoacids, Oxoproline - Acetaminophen use

Low Albumin - a correction factor of 2.5 must be multiplied to every 1mg/dl reduction of albumin below 4 mg/dl and this added to Anion Gap (AG) to get the **True anion gap.**

eg- Albumin – 1.6 mg/dl and Anion Gap - 12

Corrected (True) AG = (4 - 1.6) × 2.5 + AG = 6 +12 = 19

Another formula- Na – (Chloride + Bicarbonate) + 2.5 (4 – Sr.albumin)

When the anion gap is not corrected in hypoalbuminemic pts, abnormally elevated anion gaps could be missed.

DELTA RATIO & DELTA GAP

Universal rule- The increment in anion gap (AG-12) = decrease in bicarbonate (24- NaHco3)

DELTA RATIO. (Delta AG / Delta HCo3)

(AG – 12 / 24 - NaHCo3)

Metabolic Alkalosis

Change in **AG** > drop in **HCO3** (24 - NaHco3)

(For the given anion gap increase , bicarbonate did not fall as much)

N anion gap metabolic acidosis

Change in AG < drop in HCO3 (For the given anion gap increase , bicarbonate fell much more)

Values -

< 0.4 - NAGMA
1.4-0.8 – NAGMA + HAGMA
1- 2 - HAGMA
>2 - HAGMA + Metabolic Alkalosis

DELTA GAP = (AG - 12) + HCo3

If Delta Gap < 18 = Non Anion Gap Metabolic Acidosis
If Delta Gap > 30 = Metabolic Alkalosis

OSMOLAR GAP

Osmolar Gap = Measured Osmolarity – Calculated Osmolarity. (Normal = < 10 meq/L)
Calculated Osmolarity = 2xNa + Glucose/ 18 + Urea/ 6

Treatment of Metabolic Acidosis –

Correction of acidosis with bicarbonate may be warranted in patients of myocardial dysfunction as acidosis can cause catecholamine refractoriness. Adverse effects of bicarbonate can be reduced by giving slow infusions in preference to rapid boluses, by correcting hypocalcemia and ensuring adequate ventilation

Bicarbonate side effects

- Hypernatremia, Hyperosmolality (osmolality is 2,000 mOsm/L equal to 5.8% NaCl.
- Impaired oxygen unloading due to left shift of the oxyhaemoglobin dissociation curve
- Hypercapnia with paradoxical intracellular & CSF acidosis -Ventilation must be adequate to eliminate CO2 produced from. HCo3
- Ionized hypocalcemia & Low K due to alkalosis causing shift into cells decreasing myocardial contractility.

Always treat the underlying cause

- Shock - Restore perfusion with fluids and adequate tissue oxygenation / ventilation, vasoactives, early antibiotic treatment, source control (surgical debridement , central line removal , ischemic gut)

- Status asthmaticus - taper high dose of beta 2 agonist to reduce lactate levels

- Changing from Normal Saline to Balanced fluids (Ringer Lactate, PlasmalyteA) to reduce hyperchloremic normal anion gap acidosis. Normal Saline- Na- 154 meq/l, Cl- 154meq/l Ringer Lactate – Na-130 meq/l, Cl- 110 meq/l

- Antidotes for toxins, drugs (Paracetamol even in normal doses can be toxic in hepatic dysfunction).

- Institute dialysis early in Renal failure for persistent acidosis

Analysis with ABG

Measured values - pH, paCo2 , paO2, tCO2 : **Calculated values –** Hco3, BE, SBE

1. History & Physical. gives an idea of what acid base disorder might be present

2. Look at the pH

   • If pH < 7.35, then academia
   • if pH > 7.45, then alkalemia
   • pH may be normal in the presence of a mixed acid base disorder, particularly if other parameters of the ABG (PCo2, HCo3)are abnormal.

3. Look at PCO2, HCO3-. What is the acid base process (alkalosis vs acidosis) leading to the abnormal pH? Are both values normal or abnormal?

   - One abnormal value will be the initial change (side of the pH change) and the other will be the compensatory response.
   - The direction of compensatory variable is on the same side as the primary variable.
   - Remember compensation never over shoots the pH.

4. If respiratory process, is it acute or chronic?

   - To assess if acute or chronic, determine the extent of compensation.
     10 mmHg change in PaCo2 – Bicarbonate changes by 1 (Acute)
     10 mmHg change in PaCo2 – Bicarbonate changes by 4 (Chronic)

5. If metabolic process, is degree of compensation adequate?

   - Calculate the estimated PCO2, this will help to determine if a separate respiratory disorder is present. In a primary metabolic acidosis, the degree of acute respiratory compensation (pCo2 rise) can be predicted by the following relationship:

     Expected PaCO2 = (1.5 X [HCO3-]) + 8 ± 2 Winters Formula

     If the measured PaCO2 is higher than the expected PaCO2, a concomitant respiratory acidosis is also present. Another formula.....

     1 mEq/L change in HCo3 – PaCo2 changes by 1 (Acute)
     1 mEq/L change in HCo3 – PaCo2 changes by 4 (Chronic)

6. If metabolic acidosis, then look at the Anion Gap.
   - If elevated (> 12), then acidosis due to. (Ketoacidosis, Uremia, Lactic acidosis, Toxins)
   - If anion gap is normal, then acidosis likely due to diarrhea, RTA, saline

7. If anion gap is elevated, then calculate the Delta-Ratio (Δ/Δ) to assess for other disorders.

- Δ/Δ compares the change in the anion gap to the change in bicarbonate.
- If ratio between 1 and 2, then only wide anion gap acidosis
- If < 1, then there is a coexistent Normal anion gap acidosis
- if > 2, then there is a coexistent Metabolic alkalosis present (or rarely a compensated chronic respiratory acidosis.)

8. If normal anion gap and cause is unknown, then calculate the Urine Anion Gap (UAG).

- In RTA, UAG is positive.
- In diarrhea and other causes of metabolic acidosis, the UAG is negative. (neGUTive in diarrhea)

Metabolic acidosis is the pathological state with an arterial pH of less than 7.35 in the absence of an elevated PaCO2
Traditional Henderson- Hasselbalch theory $H^+ = 24 \times PaCO_2 / HCO_3$. Change in any one variable changes the other variable within limits. However They do not state the cause of the underlying abnormality. This does not consider the role of albumin.
The modern Stewart approach is based on the 3 independent variables which control the H^+ concentration to maintain the pH. Partial pressure of CO2, the strong-ion difference (SID), and the total amount of weak acids. SID is the difference between strong cations and anions. (Na+K+Ca+Mg)– (Cl+ lactate) Normal – 38-42 mEq/L. Narrowing is acidosis and widening is alkalosis. Weak acids are Albumin and phosphate A tot- total concentration of weak acids. increase in ToT is metabolic acidosis and decrease results in metabolic alkalosis.

Approach to Acid base analysis

1. What is the pH? 7.35-7.45 normal but check pCO_2 and Base excess
   pH <7.35 acidemia
   pH >7.45 alkalemia

2. What is the primary disorder?
   In acidosis check for low HCO3 (metabolic) or High $PaCO_2$(respiratory)
   In alkalosis check for high HCO3 (metabolic) or low $PaCO_2$ (respiratory)
   pCO_2 and pH move in the opposite directions in respiratory and in the same direction in metabolic

3. Look for compensation in metabolic events
   Metabolic acidosis - winters formula to know the $PaCO_2$ compensation
   1.5 (HCO_3) +8 ± 2 or 40 ± SBE
   Metabolic alkalosis - pCO_2 changes by 0.7 for every1 mEq/L HCO3

4. Compensation in respiratory events

Acute respiratory acidosis Hco3 changes by 1 for every 10 change in $PaCO_2$
pH changes by 0.08 for every 10 change in $PaCO_2$
Chronic respiratory acidosis Hco3 changes by 3 for every 10 change in $PaCO_2$
pH changes by 0.03 for every 10 change in $PaCO_2$
Acute respiratory alkalosis Hco3 changes by 2 for every 10 change in $PaCO_2$
pH changes by 0.08 for every 10 change in $PaCO_2$
Chronic respiratory alkalosis Hco3 changes by 4 for every 10 change in $PaCO_2$
pH changes by 0.03 for every 10 change in $PaCO_2$

5. Check for anion gap (AG).
AG = (Na+ K)– (Cl+HCO_3) =12 ± 2 mEq/L
Correct for low albumin= observed AG + 2.5(4 - albumin in g/dL)

6. Delta Gap Increment in anion gap should be the same as decrement in bicarbonate Delta ratio is Delta AG/Delta HCO_3
When change in AG is more it is metabolic alkalosis
When change in HCO_3 is more it is non anion gap metabolic acidosis.
if <.4 NAGMA
1-2 HAGMA
1.4-1.8 NAGMA+HAGMA
>2 HAGMA + metabolic Alkalosis
Delta Gap – AG-12 +HCO_3 <18 non anion gap metabolic acidosis
>30 metabolic alkalosis
Osmolar Gap=measured osmolality –calculated osmolality (n =<10mEq/L)

7. Non anion gap metabolic acidosis (Normal anionic gap metabolic acidosis)
Calculate urine anionic gap (UAG) = (urinary Na + Urinary K)- urinary chloride
If positive UAG: Renal etiology (Type I, II, IV renal tubular acidosis)
Urinary pH>6: Type I RTA
Urinary pH <5.5: Hypokalemia: Type II RTA, Hyperkalemia: Type IV RTA
if negative UAG: Extra renal etiology

Lactic acidosis–Cohen and woods classification.

Type A in tissue hypoperfusion and hypoxia

Type B in situations without tissue hypoperfusion

B1 – acquired conditions-Sepsis, seizures, Diabetes, ARDS, renal failure, malignancy, thyroid storm, post cardio pulmonary by-pass (all with excess production) Liver failure (decreased excretion)
B2 medications - Epinephrine, acetaminophen, isoniazid, nitroprusside.

B3-IEM related

Management of metabolic acidosis:

Correction is warranted if myocardial dysfunction is encountered

Given as slow infusions / Correct hypocalcaemia/Ensure adequate ventilation

Causes of metabolic acidosis

Anion gap acidosis	Non -Anion Gap Acidosis
Lactic acidosis DKA Aki IEM Tumour lysis Rhabdomyolysis TPN Exogenous sources Poisoning – methanol, Glycols, Ethanol, Salicylates	Hyperchloremic acidosis TPN GI loss Renal causes Drug induced tubulopathies

Approach to metabolic acidosis:

1. History- GI loss, medications, renal issues
2. Examination – for aetiology dehydration in DKA, hyperventilation
3. ABG confirmation – to know metabolic acidosis ± respiratory acidosis
4. Anion gap – look for normal and high anion gap
5. Check compensation by winters formula
6. Look for additional metabolic disturbances by Delta gap
7. Treat the underlying cause /bicarbonate therapy/ dialysis

DKA- Insulin and fluids
Distal RTA – bicarbonate and citrate
CKD-oral sodium bicarbonate
Methanol. Ethanol poisoning - Fomepizole
Sepsis – fluid resuscitation and electrolyte correction
Sodium bicarbonate increases the arterial pH only if there is adequate alveolar ventilation

Bicarbonate replacement is done using SBE or bicarbonate levels
SBE x body weight in Kg x 0.3
HCO_3 deficit (mEq/L) = 0.3 x bodyweight in Kg x HCO_3(Expected – Observed)
Adverse effects of sodium Bicarbonate
Hypervolemia, hyperosmolarity, hypernatremia, hypocalcaemia
Bicarbonate may worsen lactic acidosis
Can decrease blood pressure and cause raised ICP due to hypertonicity

References:

1.Nichols DG & Shaffner DH. Rogers text book of pediatric Intensive care (2016)
2. Robert M, Kliegman & Joseph W. St Geme III.Nelson Textbook of Pediatrics International Edition 21st edition
3.Zimmerman J.J Clark R.S.B, Fuhrman B.P, Rotta A.T, Kudchadkar S.R, Relvas M.S & Tobias J.D. Fuhrman & Zimmerman's Pediatric Critical Care (Sixth edition)
4. Sood P, Puri S. Interpretation of arterial blood gas. Indian J Crit Care Med 2010; 14 (2):57-64.

1. 1year male child (6 Kg) with acute watery diarrhoea 10-12 episodes /day, lethargy, vomiting and seizures Treated with IVF outside. Clinical examination revealed verbal responsive, sunken eyes, dry tongue and mucosa, pallor, increased skin turgor. Heart rate was 160/mt. RR49/mt. ++/+ prolonged CRT, BP 80/66mm Hg. Cool below ankle SpO_2 96% in room air. urine output is 4ml/kg/hr. Blood glucose was 35 mg/ dl. ABG showed the following –pH 7.2, pCO_2 35 HCo3 14 BE -7

   Na 122 K 2 Cl 96 lactate 3.6
   - What is the physiological status?
   - Interpret the ABG
   - Outline the fluid therapy and management?

2. 3month male child admitted with persistent vomiting and seizures. History revealed previous sib death at 3 months of age. examination revealed lethargy, tachypnoea, Dusky extremities. Primary assessment revealed stable airway, RR 60/mt, no retractions, bil air entry, no added sounds. HR 160.mt +++/+ Poor distal pulses, CRT >3 seconds, cool below knee, BP 70/? Verbal responsive, PERRL. CBG was 40mg/dl,
   ABG pH 6.9 Pco2 25 Hco3 10 BE -8 lactate 6 Na 130 K 3 CL 100

   - What is the physiological status?
   - Interpret the ABG
   - Discuss the investigations and management

3. 10 year old female child was admitted with altered sensorium and pain abdomenhematemesis. She was lethargic, and pink. Primary assessment revealed the following maintainable airway, RR of 40/mt, bil air entry was equal, no retractions, no added sounds, saturation was 96% in room air. HR was 140/mt, central and peripheral pulses were +++/+, CRT >3 seconds, cool below knee, BP 80/40mm Hg.GCS 10. PERRL. Head to foot examination was not contributory. Her urine was red coloured.

   CBG was 250mg/dl. Venous blood gas revealed the following pH 6.8Pa02 85 Paco2 28 Hco3 7 Na 140 K 5.0Cl 100 BE -20 lactate 3

   - What is the physiological status

- Interpret the VBG
- Anion gap and delta gap
- What are the possible diagnosis?

4. 2year female child weighing 14 kg was admitted with history of fever vomiting 5 days afebrile since morning. History of facial puffiness and rashes over the body. She was breathless and lethargic with reduced urine output. Treated with paracetamol and ceftriaxone. Assessment revealed the following stable airway, RR5/mt bil air entry no added sounds, no retractions, HR 160/mt ++/0 BP not recordable. Cool below knee CRT > 3 sec anuric for 4 hours. GCS 8 PERRL. Petechial rashes and ecchymosis all over the body with distended abdomen.

   BG was 40mg/dl. Na 128 K 5 Cl 90 lactate was 3.6 mmol VBG showed the following pH7.0 HCo3 12 PCo2 18 Po2 80 BE -6 lactate 3
   - What is the physiological status?
   - Interpret the ABG
   - Likely diagnosis investigations and management

5. 8-year-old female child (16kg) with vomiting, breathlessness, lethargy and pain abdomen. Treated outside as wheeze with nebulisation for 2 days. history revealed recent weight loss. She was lethargic but responsive to commands, dehydrated, pink. Airway was stable, RR36/mt, no retractions, bil airentry normal, no added sounds.HR 120/mt ++/+ cool below knee, CRT 3 seconds unequal pupils, BP90/60 mmHg, sats 96% room air Urinary Catheter revealed 120 ml urine output.

   Her CBG was 350mg/dl. Ph 7.104 Pco2 18.5 po2 60.5 Hco35.7 BE -23.9NA 154 K 3 .29 Ca 1.09 Cl 128 Hb 18.2 A gap 23.9lac 2.55
   - What is the physiological status
   - Interpret the ABG
   - Outline the management

6. 1 year boy child with cyanosis. Airway- maintainable. Breathing - RR 30/min, WOB Normal, No added sounds, Cyanosis present, SPO2 83% with O2 NRM. Circulation – HR 156/min, NIBP – 92/66 Perfusion good. Disability – unresponsive, Exposure – Temp – 98 F, No abnormal smell, Central Cyanosis present

   ABG -pH 7.2 Pco2 28.7 o2 167 Hco3 14 BE -1.2 HCt 40Na 142 k 4 ca 1.0 cl 109sO2 99 FO2HB 63.2 FCOHB 0.8 FMetHB 53.5 Lactate5 Gl200

   - What is the physiological status
   - Interpret the ABG Calculate Delta Gap
   - Write the Management
   - List the likely causes for this scenario

7. 10-year female kid with history of vomiting, lethargy and pain abdomen. Treated outside with normal saline boluses as acute abdomen. Referred for surgical opinion. She is lethargic /Primary assessment showed maintainable airway, RR 40/mt, no retractions, no added sounds, bill air entry saturation with room air 99/Hr 120.mt, +++/++ peripheries warm, CRT <3 sec BP 100/70. DEM intact PERRL.

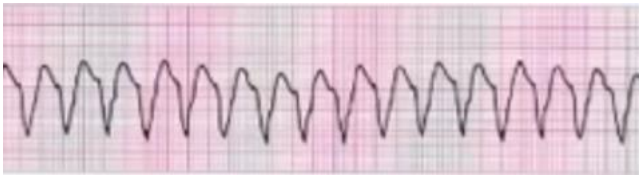
   ABG pH 6.99 Hco3 4 Pco2 30 Po2 97 CBG 300 Na 121 K 5.6 Cl 108 lactate 2

   - Interpret the ABG
   - What is the fluid of choice
   - How to treat acidosis
   - After 8 hours of therapy the ABG
   - pH 7.23 Pco2 35 Hco3 18 Po2 100 Na 146 K 4 CL 128 BE - 3
   - What is your interpretation of ABG
   - Management strategy?

8. 2year old female child weighing 7 kg admitted with fever loose stools and vomiting 3 days Breathlessness one day. Wt for age and Wt for HT below -3SD She is lethargic, dehydrated, acidotic breathing. RR40/mt bil air entry equal no added sounds mild Subcostal retractions saturation 96% with NRM HR 160 central pulses good peripheral pulses weak CRT> 3 sec Cool below knee Bp 78/50. Received NS bolus outside

   CBG 50mg/dl ABG Ph 7.1 Hco3 10 Pco2 38 Po2 160 Na 130 K3.5 CL 112 SGOT 50 SGPT 35 Bil 0.3 Albumin 2.

   - Interpret the ABG calculate the anion gap
   - What is the fluid management

9. 5yr female child brought to the emergency department, afebrile, Unresponsive HR 200/mt, BP120/80mmHg, Seizures at ER, Dilated pupils, sluggish reaction. No significant history. No previous medical illness.
   ABG Ph 7.1 Pco2 28 HCo3 12 Po2 90 Na 135 K 4 Cl 110 BG 100
   ECG 

   - Interpret the ECG and Blood gases
   - What is the likely diagnosis?
   - What is the management?

10. 10 months female with history of fever breathlessness vomiting loose stools

    Lethargic, responsive to calls, RR 65/mt retractions, bil equal air entry, no added sounds, sats at 96% with NRM HR 170/mt ++/+ cool below knees BP not recordable. catheter showed 20 ml clear urine DEM intact PERRL.
    ABG Ph 7.2 Paco2 50 Pao2 79 Na 139 K 4. Cl 110 Hco3 10
    - What is the physiological status
    - Interpret the ABG
    - Management

11. 6year boy with disorientation and drowsy for 2 hours. Was playing at the Garage with the father. Later brought by the neighbour as found alone and drowsy in the car. Examination revealed drowsy child GCS 6/15 , no evidence of trauma pallor or cyanosis or fang mark or bite marks. No shock No history of previous seizures. Intubated in view of low GCS.
    Post intubation Blood gases are as follows -pH 7.3 Po2 110 Pco2 18 HCO3 18 BE -11 Lac 4 Na 140 K 4.5 CL 110 BG 100 serum osmolarity 311.

- Interpret the ABG
- Calculate the AG delta Gap and osmolar Gap
- What are the underlying causes you would think in this type of ABG

HYPERNATREMIA

Contributors: Dr. Anitha VP, Dr Shyamala J, Dr Priyavarthini V, Dr Sharada R C

Definition: Serum Na > 145 mmol/ L or mEq/L. Some references mention as > 150 mEq/L

Salt and water physiology

60% of body weight is constituted by water. Between extracellular fluid (ECF) and intracellular fluid (ICF) is the cell membrane. Only water permeates through the cell membrane. This transport is from lower tonicity to higher tonicity which means in hypernatremia it is from intracellular to extracellular compartment. The electrolyte composition of ECF and ICF has almost equal osmolality but electrolyte concentration is different. ECF: Na 135-145 mEq/L K 3.5 -4.5 mEq/L and ICF: Na 10-20 mEq/L K 120 - 150 mEq/L. Sodium is closely related to water balance. Thirst and ADH release are the major defences associated with hypernatremia. Threshold for thirst begins at 5-10 mosm/kg higher than that for ADH release

Causes of hypernatremia (Table 1)

Hypernatremia develops only by two mechanisms

1. Loss of free water (most common) – could be as loss of water or hypotonic fluids leading to hypovolemic hypernatremia

2. Gain of sodium (less common) – mostly iatrogenic; rarely accidental leading to hypervolemic hypernatremia

Table 1 showing the causes for hypernatremia

Hypotonic fluid or electrolyte free water loss	Excess salt or gain of salt
GIT : Diarrhea	Improper ORS or formulae
Renal: osmotic agents Diabetes Insipidus Chronic kidney disease Acute tubular necrosis	Iatrogenic Bicarbonate or hypertonic saline
Skin :Burns , increased sweating	Salt poisoning Child abuse Salt with water instead of sugar Pica Hyperaldosteronism

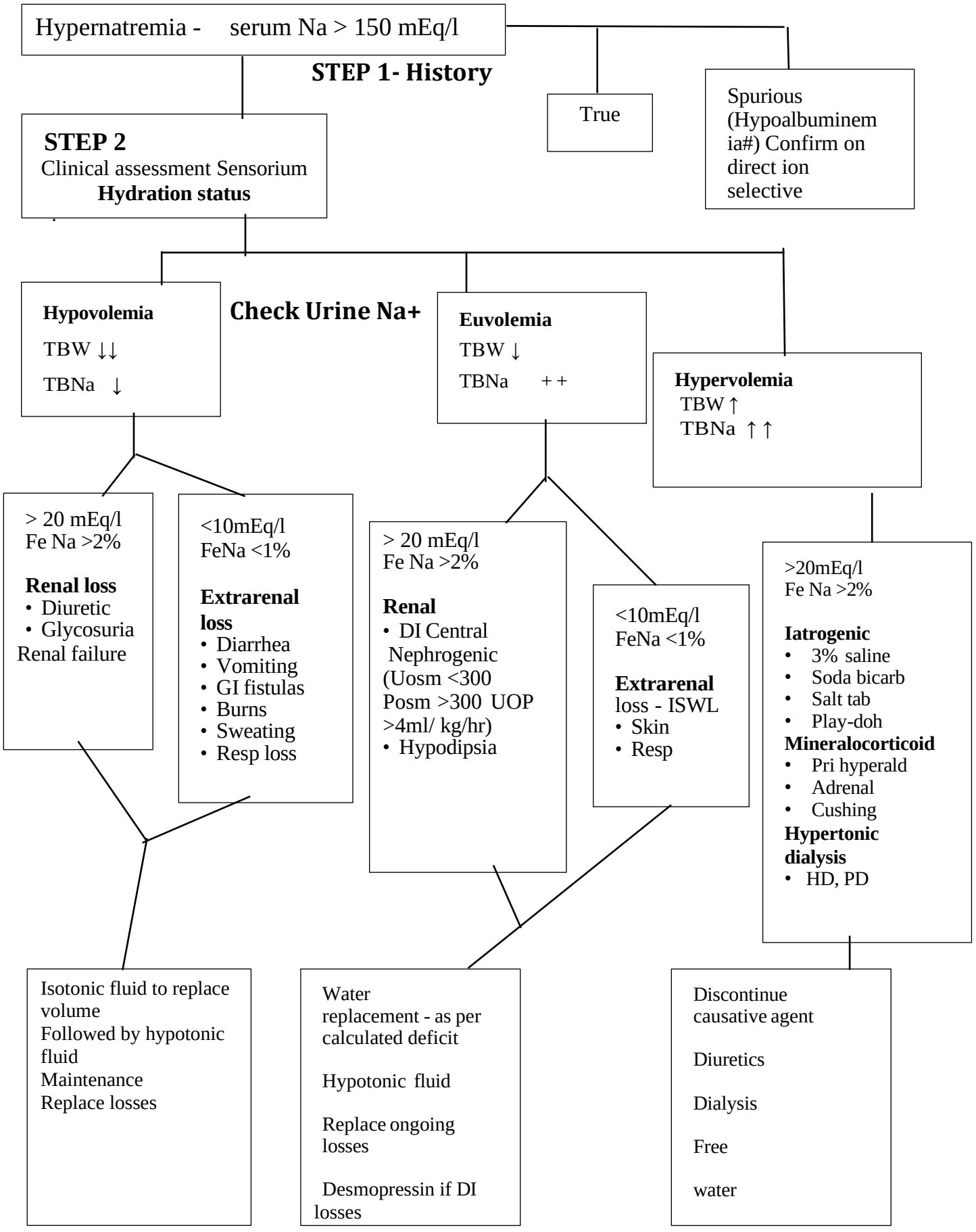
Clinical features

1. Dehydration and shock: Not obvious as ECF water content is near normal. Subtle findings – thirst and "doughy" skin, tachycardia, wt loss
2. CNS symptoms: Convulsions, irritability, high-pitched cry and. Some alert infants are very thirsty.

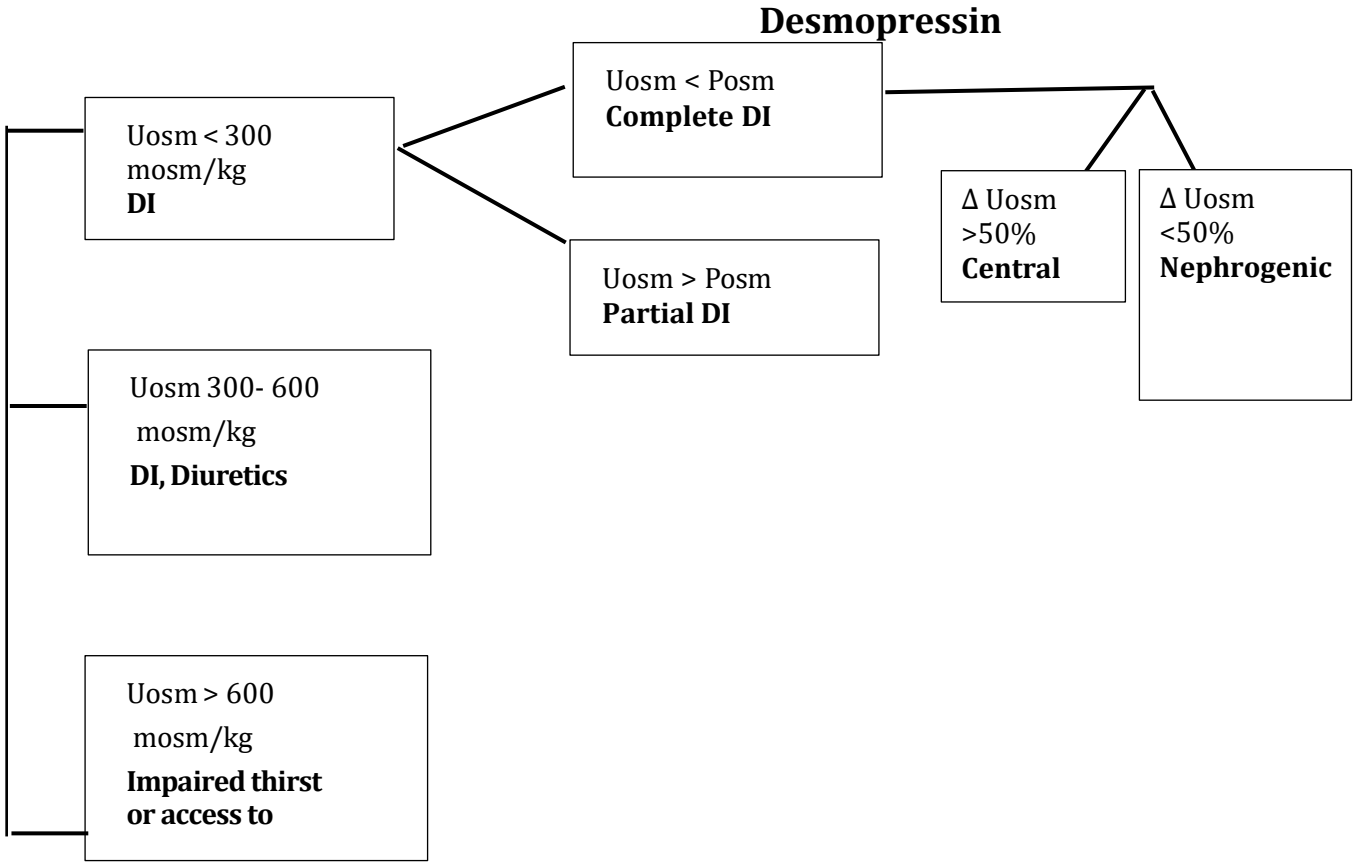
   a) Brain haemorrhage : Brain shrinks, results in tearing of bridging blood vessels - subarachnoid, subdural, parenchymal bleed .
   b) Thrombosis: Stroke, dural sinus thrombosis, peripheral thrombosis, and renal vein thrombosis - possibly due to hypercoagulability.

3. Lab investigations

- CBC: high HCT favours dehydration
- CXR: Can identify volume overload-pulmonary congestion, pleural fluid USG Lungs: presence of B lines. IVC filling will indicate the volume status
- Urine Sodium: < 10 favours dehydration > 20 – Salt excess or renal losses FENA: < 1.0 – hypovolemia > 2.0 Salt excess and hypervolemia
- Blood sugar, urea, creatine, other electrolytes, calcium and magnesium. High serum chloride – Salt excess
- Serum and urine osmolarity
- Serum AVP level and response to AVP
- Neuroimaging: For cause in DI and CNS complications
- ABG: In a setting of edema, hypertension, hypokalemia, hypernatremia and metabolic alkalosis – hyperaldosteronism


Hypernatremia - serum Na > 150 mEq/l
STEP 1- History
True
Spurious (Hypoalbuminemia#) Confirm on direct ion selective
STEP 2
Clinical assessment Sensorium
Hydration status
Check Urine Na+
Hypovolemia
TBW ↓↓
TBNa ↓
Euvolemia
TBW ↓
TBNa + +
Hypervolemia
TBW ↑
TBNa ↑↑
> 20 mEq/l
Fe Na >2%
Renal loss
• Diuretic
• Glycosuria
Renal failure
<10mEq/l
FeNa <1%
Extrarenal loss
• Diarrhea
• Vomiting
• GI fistulas
• Burns
• Sweating
• Resp loss
> 20 mEq/l
Fe Na >2%
Renal
• DI Central Nephrogenic (Uosm <300 Posm >300 UOP >4ml/ kg/hr)
• Hypodipsia
<10mEq/l
FeNa <1%
Extrarenal loss - ISWL
• Skin
• Resp
>20mEq/l
Fe Na >2%
Iatrogenic
• 3% saline
• Soda bicarb
• Salt tab
• Play-doh
Mineralocorticoid
• Pri hyperald
• Adrenal
• Cushing
Hypertonic dialysis
• HD, PD
Isotonic fluid to replace volume
Followed by hypotonic fluid
Maintenance
Replace losses
Water replacement - as per calculated deficit
Hypotonic fluid
Replace ongoing losses
Desmopressin if DI losses
Discontinue causative agent
Diuretics
Dialysis
Free water

STEP 5- Check urine osmolality



- Useful to do UEC, calcium, magnesium, phosphate and glucose. These may need concurrent management
- In the presence of hypoalbuminaemia (albumin <30 g/L), a blood gas sodium level is more reliable
- Initial paired serum and urine sodium, creatinine and osmolality is ideal, but if results will be delayed, a urine dipstick for specific gravity will give an indication of urinary concentration and treatment should not be delayed

Acute hypernatremia, of < 48 hours duration and symptomatic, is managed to achieve a rise in Na of 3-5 mEq/l immediately with a rise of 10-12 mEq/l in 24 hours
Chronic hypernatremia, of > 48 hours duration is corrected more gradually, with a rate of rise of not more than 0.5 per hour or 8-10 mEq/l per day.

Free water deficit in milliliters = Current total body water x ([current plasma Na/140] - 1)
Free water deficit in milliliters = (4 mL/kg) x (weight in kg) x (desired change in plasma Na)
Adrogue Madias formula predicts the change in Na achieved with 1 litre of chosen fluid

Change in serum (Na^+)

$$= \frac{\text{infusate } (Na^+) + \text{infusate}((K^+) - \text{serum } (Na^+)}{\text{Total body water}+1}$$

Case scenario

A late preterm neonate born at 36 + 3 weeks, B wt – 2.48 kg with uneventful antenatal and early neonatal period. Brought for first postnatal review only on D 18 with weight of 1.573kg, decreased urine output, loose stools-watery and foul smelling. He was exclusively breast fed. On admission he was dull and lethargic with sunken eyes and depressed AF. Systemic examination revealed HR of 180/min, doughy feel of skin, proximal and distal pulses equally felt, legs a little cold to touch. There was 36.8 % weight loss.Systolic BP – 60 (NIBP). Septic work up done. Treatment initiated USG abdomen- bilateral renal cortical echoes, echogenic medullary pyramids, no hydronephrosis What are the salient points in the history? What may be the cause? What tests would you like to order? What is the probable diagnosis? Outline the management

Investigations done on admission:

S.Creatinine	2.7
B urea	217
S. Sodium	180
S. Potassium	5.8
S. Chloride	102
S. Bicarbonate	22
CBG	85
CBC	Normal

Case history:

11 months old female infant weighing 10 kg was brought to ER with acute watery diarrhoea and vomiting of 2 days duration. She also had an episode of sudden vacant stare followed by up rolling of eyeball yesterday which lasted for a period 2 minutes. Child had fever for which she was treated with ORS and some medications On assessment at your ED on arrival

- Found to be pain responsive. febrile. weight -9kg
- RR 40/m9n, no increased WOB, BAE: equal, SpO2 of 98-99% in room air
- HR – 162/min, BP- 70/48 mm Hg, CFT- >3s. Weak peripheral pulses. Normal urine output
- POCT: RBS-295mg%, BLOOD Gas electrolytes: Na- 156, K- 4.2, Cl – 125, HCO3 – 17

Discuss the following questions and write your management plan.

Questions

1. What is your diagnosis?
2. What are the principles in management in this child?
3. List the various steps in management in order of priority.
4. What is the underlying mechanism of hypernatremia based on history and volume status? What etiological factors have contributed to hypernatremia?
5. What is the duration over which the correction of sodium levels should be done based on current serum sodium values?
6. How will you calculate the maintenance fluid requirement in this child?
7. How will you estimate the electrolyte free water loss in this child?
8. What will be the final fluid prescription after correction of shock? Choice of fluids & rate of correction?
9. How will you address the ongoing losses?

Case scenario:

A 10-year-old boy weighing 30 kg is undergoing treatment for severe traumatic brain injury after a road traffic accident in the PICU (day 5 of PICU stay). He had severe cerebral edema and underwent decompressive craniectomy on day 2 of admission. There is ongoing discussion regarding poor neuroprognosis given the CT Brain and clinical features such as non-reactive pupils (4 mm each). He has no response to pain, absent deep tendon reflexes. He is invasively ventilated and on ketamine and midazolam infusions. His vitals are as follows:

- HR 150/min, BP 86/44 (55), SpO2 98% (FiO2 30%.
- His peripheral perfusion is poor and CVP is 2 mmHg.
- He is on 3% saline infusion at 30 ml/hr. A routine review of charts shows increasing sodium trends over the past 24 hours as follows:

- Na (in mmol/L) every 6 hours: 145 – 150 – 153 – 164
- Other labs: K 4 mmol/L, HCO3 – 24 mmol/L, Cl- 110 mmol/L, Urea 30 mg/dl, Creatinine 0.6 mg/dl
- Are you concerned about the electrolyte trends?
- Could 3% saline be the cause of this rising sodium trends?
- What is the one clinical parameter that will guide you to the possible diagnosis?
- How will you approach this situation?

Hypocalcemia/ Hypomagnesemia

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Calcium is essential for nerve conduction, muscle contractility and coagulation.

Normal values of calcium:

Ionized calcium – 4.65 to 5.25 mg/dL (1.2 to 1.3 mmol/L) (1 mg/dL = 0.25 mmol/L).

Total calcium – 8.5 to 10.5 mg/dL (2.12 to 2.62 mmol/L)

Hypocalcemia is defined as corrected serum total calcium levels <2.12 mmol/l (8.5 mg/dl).

The serum calcium falls by 0.8 mg/dL (0.2 mmol/L) for every 1 g/dL (10 g/L) fall in the serum albumin concentration

The formula for albumin-corrected serum calcium is as follows:

total serum Ca concentration (mg/dL) + 0.8 × [4 – serum albumin concentration (g/dL)

Ionised calcium is not affected by albumin levels. However, it can be affected by change in pH. Alkalosis increases the amount of albumin-bound calcium and decreases the level of ionized calcium, in acute respiratory alkalosis, the level of ionized calcium falls to 0.16 mg/dL for each 0.1 unit increase in pH

Hypocalcemia in Newborns

1) For term infants or preterm infants weighing >1500 g at birth

   Total serum calcium <8 mg/dL (2 mmol/L) or ionized calcium <4.4 mg/dL (1.1 mmol/L)

2) For very low birth weight infants weighing <1500 g.

   Total serum calcium <7 mg/dL (1.75 mmol/L) or ionized calcium <4 mg/dL (1 mmol/L)

Etiology of Hypocalcemia

	Parathyroid mediated	**Non parathyroid mediated**
Genetic	• Familial isolated hypoparathyroidism • Syndromes associated with hypoparathyroidism: −22q11.2 deletion (DiGeorge) syndrome −Hypoparathyriodism, Sensory Neural Deafness, Renal Dysplasia Syndrome (HDR) −Kearns–Sayre syndrome −Kenny–Caffey syndrome type 1 and 2 −Mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes (MELAS) syndrome −Sanjad Sakati syndrome (SSS) −Mitochondrial trifunctional protein (MTP) deficiency syndrome • Autosomal Dominant Hypocalcemia (ADH) 1 and 2 • Pseudohypoparathyroidism 1A and 1B • Wilson's disease • Hemochromatosis	• Vitamin D dependent rickets (VDDR) type 1 and 2 • Heriditary vitamin D-resistant rickets (HVDRR) • Osteopetrosis • Maternal hyperparathyroidism
Acquired	• Post-surgical hypoparathyroidism • Hypomagnesemia • Autoimmune polyendocrine syndrome type 1 (APS1) • Blood transfusion(haemosiderosis) • Radiation therapy • Sclerotic metastases	• Vitamin D deficiency • Malabsorption chronic kidney disease Hungry bone" syndrome End-stage liver disease • Critical illness • Acute pancreatitis Citrate (blood trasfusion) Drugs: −Loop diuretics −Phosphate −Foscarnet −EDTA −Anti-convulsants −Magnesium sulfate −Calcitonin, bisphosphonates −Cinacalcet

Adopted from Pepe, J., Colangelo, L., Biamonte, F., Sonato, C., Danese, V. C., Cecchetti, V., et.al

2020. *Diagnosis and management of hypocalcemia. Endocrine.*

Evaluation:

Obtain a complete history and physical examination to detect clinical features of hypocalcemia and findings that may help in identifying etiology.

History:

- Dietary history (particularly calcium, vitamin D intake)
- History of neck surgery/radiation
- Family history of calcium disorders
- Feeding problems, nausea, vomiting, delayed eruption of teeth
- Apnea, jitteriness, irritability
- Cardiac abnormalities or recurrent infections
- Muscle cramps, twitching, or spasms
- Circumoral or distal paresthesias
- Seizures

Suspect hypocalcemia when a child presents with any of the following features

- Numbness and tingling sensation in the circumoral region
- Paresthesias of the hands and feet, muscle cramps especially after exercise
- Carpopedal spasm (tetany). (Signs of latent tetany: Chvostek sign: twitching of the orbicularis oris muscle with light tapping of the facial nerve at the anterior external auditory meatus. Trousseau sign: carpopedal spasm when BP cuff maintained 20 mm Hg above SBP for 3 minutes)
- Laryngospasm with stridor
- Convulsions
- Features of Rickets: Widening at the wrists, knees, and/or ankles, bowing of the extremities

- Cardiovascular manifestations: hypotension, heart failure, arrhythmias
- ECG changes: prolonged QT interval

Diagnostic Tests:

Step 1: Check Total and ionized calcium levels – confirm hypocalcemia

Step 2: Check serum phosphorus, magnesium, alkaline phosphatase, creatinine

Step 3: Check Intact PTH level. If intact PTH is low, evaluate for hypoparathyroidism

Step 4: Assess Vitamin D status – check 25-hydroxyvitamin D level. If vitamin D level is in deficiency range, evaluate. Blood for 1,25 dihydroxy vitamin D should be stored before initiating treatment. 1,25 (OH)2 D levels will be needed to detect problems in Vitamin D metabolism. In Vitamin D dependent rickets type I levels are low and in Vitamin D dependent rickets type II levels are increased.

Step 5: Check urine calcium and creatinine. A timed 24hr urinary calcium excretion (collected in containers with hydrochloric acid to prevent precipitation of calcium salts) can be obtained in older children. Hypercalciuria is suggested by values greater than 0.1mmol/kg/day. Timed urinary collection may be difficult in young children and a random spot urine calcium creatinine ratio repeated on 2–3 occasions at the same time of day is the most appropriate way of assessing urine calcium excretion. The calcium creatinine ratio on the second voided urine sample of the day after an overnight fast is most closely related to 24-hour urine calcium level. In the presence of hypocalcaemia a urine calcium/creatinine ratio greater than 0.3 on spot samples suggests inappropriate excretion and indicates hypocalcemic hypercalciuria. This is due to activating mutations of the calcium sensing receptor which downshift the set point for calcium responsive PTH release.

Step 6: Hand/wrist/knee x-ray if rickets is suspected

Step 7: Measure maternal calcium and vitamin D levels in the case of hypocalcaemia in infancy because of the link with maternal vitamin D deficiency and hyperparathyroidism

Other investigations that may be needed for rare causes are karyotyping (22q11&10p13 deletion and chest radiograph for thymic hypoplasia (Di-George syndrome), renal ultrasonogram for nephrocalcinosis and renal dysplasia (syndrome of hypoparathyroidism, deafness, renal dysplasia HDR), assessment of autoantibodies (antiparathyroid antibodies) for autoimmune causes, hearing test for deafness. Maternal and family screening may be needed for familial forms of hypocalcemia. DNA testing may be necessary to identify genetic causes of hypocalcemia.

Treatment

- Acute symptomatic hypocalcemia should be treated immediately
- Calcium gluconate (preferred) 100 to 200 mg/kg/dose (max 1 to 2 g/dose) IV (0.5ml-1 ml/kg/dose) over 5 to 10 minutes with cardiac monitoring
- (Calcium chloride 20 mg/kg/dose (max 2 g/dose) can alternatively be given if readily available).
- Bolus should be immediately followed by a continuous infusion of calcium gluconate: 500 to 800 mg/kg/24 h or an intermittent infusion of calcium chloride of 10 to 20mg/kg/dose (max 1 g/dose) q4–6h PRN.

10% solution of calcium gluconate contains about 9.3 mg of elemental calcium / mL

- Calcium gluconate can be given via peripheral IV. It is diluted in equal amount of NaCl or 5%dextrose or distilled water and given over 10 minutes in children and

over 20 minutes in newborns. Calcium chloride should only be given via central line due to risk of tissue necrosis with extravasation.

- Continue the IV infusion or intermittent doses until patient is on an effective oral regimen.
- Oral calcium 25 to 50 mg/kg/24 h elemental calcium (max 1 g elemental calcium per 24 hours) divided 3 to 4 times daily. Calcium carbonate contains 40% of elemental

  calcium and is the drug of choice.
- For patients with hypoparathyroidism, calcitriol should be initiated as soon as possible:

  infants 0.04 to 0.08 mcg/kg/24 h divided twice daily, > 1 year 0.25 mcg/24 h and increase as needed up to a maximum of 2 mcg/24 h
- Magnesium supplements should be given as needed to correct hypomagnesemia.
- For patients with vitamin D deficiency, treat with high-dose oral cholecalciferol

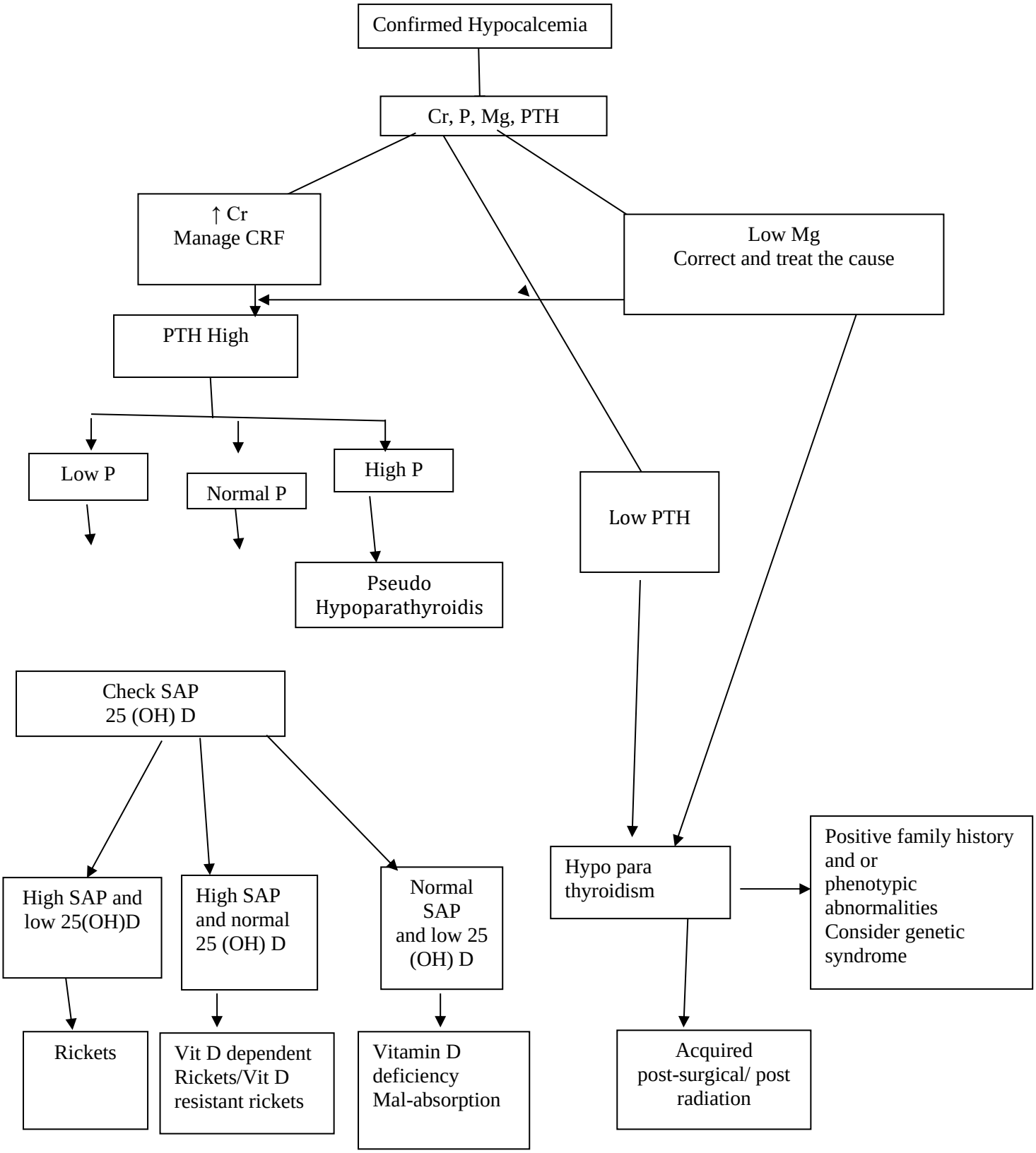
  (vitamin D 3) over 8 to 12 weeks (goal total of ~200,000 to 400,000 IU).

  Infants < 1 month: 1,000 IU daily

  Infants and children 1 month to 5 years: 1,000 to 2,000 IU daily

  Children 5 years to adult: 5,000 to 6,000 IU daily

IV Calcium is hyperosmolar and can cause severe tissue necrosis if extravasation occurs. Select a large vein. Monitor IV site frequently. Rapid IV administration can cause vasodilation, hypotension, bradycardia, syncope, cardiac arrhythmias, and cardiac arrest. Continuous cardiac monitoring is essential during infusion.



Modified from Pepe, J., Colangelo, L., Biamonte, F., Sonato, C., Danese, V. C., Cecchetti, V., Cipriani, C. et.al Diagnosis and management of hypocalcemia. Endocrine. 2020

CASE 1 :

A 2-year-old boy was brought because of absent teeth development and failure to walk. The patient appeared to be well nourished and content. His body mass index was 19.1 kg/m2 (90th percentile), he was 86 cm long (25th percentile) and he weighed 13.6 kg (75th percentile). Palpation of the patient's extremities revealed prominent, flared distal radii, humeri and femurs. The result of a total serum calcium test was 1.4 (normal 2.1–2.6) mmol/L

1.What further history should you elicit?

2.What are the investigations needed?

3.How will you manage this child?

CASE 2 :

13 days/male/ term/2.5 kg admitted with right focal seizures since 2 days.

Born to a 29 year old primi mother who had no pre-existing medical or surgical illness / drug intake. Spontaneous conception. Antenatal period was uneventful. Born by normal vaginal delivery at term. Birthweight was 2.5 kg. Apgar was 8/10 at one minute. Baby was discharged on second day of life. She is on exclusive breast feeds. Serum calcium was 3.6 mg/dL

1.What will be your initial management?

2. How will you clinically evaluate this child?

3. What other investigations will you do?

Hypomagnesemia

Magnesium is a co-factor in many biochemical reactions and essential for cellular function and nerve conduction. Magnesium also affects the electrical activity of the myocardium and vascular tone,
Normal serum magnesium levels are between 1.5 and 2.3 mg/dL(1.2- 1.9mEq/L).
Hypomagnesemia is serum magnesium less than 1.5 mg/dL. There may be variations among clinical laboratories

Etiology of hypomagnesemia

Gastrointestinal	Renal losses	Redistribution of magnesium from the extracellular to the intracellular space
Reduced intake Reduced absorption • Malabsorption • Short bowel syndrome Increased losses Chronic diarrhea Laxative abuse Excessive gastric suctioning or vomiting Hypomagnesemia with secondary hypocalcemia (HSH)	Inherited • Gitelman syndrome • Bartter syndrome Familial hypomagnesemia with hypercalciuria and nephrocalcinosis (FHHNC) • Autosomal-dominant hypocalcemia with hypercalciuria (ADHH) • Isolated dominant hypomagnesemia (IDH) with hypocalciuria • Isolated recessive hypomagnesemia (IRH) with normocalcemia • Hypomagnesemia with secondary hypocalcemia (HSH)	• Hungry bone syndrome • Treatment of diabetic ketoacidosis • Refeeding syndrome • Acute pancreatitis
	Drugs: Loop diuretics,cisplatin, amphotericin B, cyclosporine, tacrolimus, and pentamidine,	
	Others Aldosterone excess Hypercalcemia Hypophosphatemia	

Hypomagnesemia is commonly seen in critically ill children often associated with other electrolyte abnormalities like acidosis, hypocalcemia and hypokalemia.

Clinical features include neuromuscular irritability(tremors,tetany, hyperreflexia seizures) and cardiac abnormalties(ventricular tachycardia, torsades de pointes)

Approach to hypomagnesemia includes a detailed history, clinical examination and relevant investigations. Since most patients are asymptomatic and magnesium is not included in routine electrolyte estimations, a high index of suspicion is needed. Consider hypomagnesemia in any child with refractory hypocalcemia or hypokalemia.

Invstigations

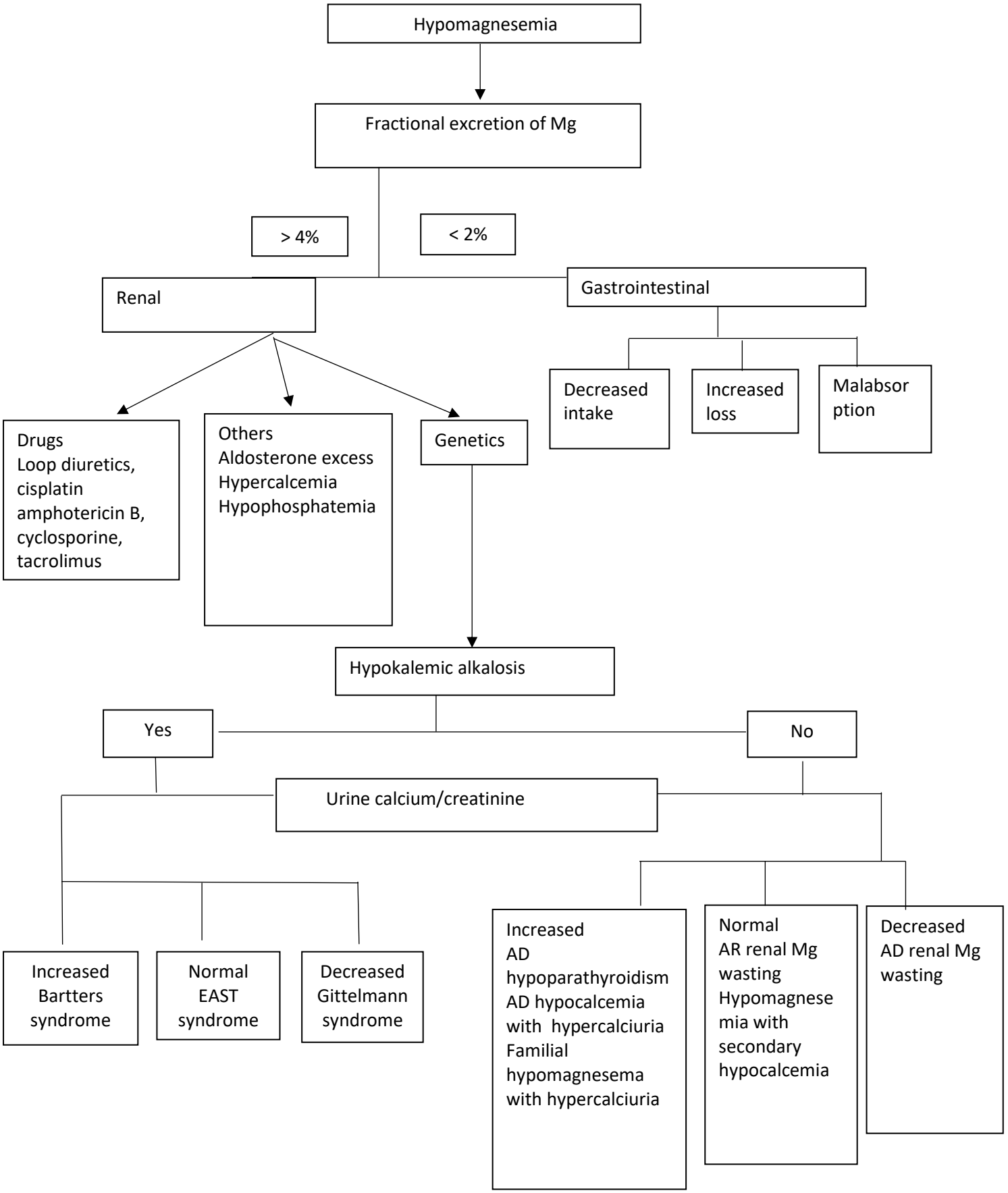
Serum Mg, Ca, Phosphate, sodium, potassium , bicarbonate, serum creatinine, fractional excretion of Mg, urine calcium, creatinine, ECG

FEMg = [(UMg x PCr) / (PMg x UCr x 0.7)] x 100 where UMg is urinary magnesium concentration, PCr is plasma creatinine concentration, PMg is plasma magnesium concentration and Ucr is urinary creatinine concentration. Plasma magnesium concentration is multiplied by 0.7 since 30% is bound to albumin and not filtered at the glomerulus.

Treatment

Severe hypomagnesemia is treated with IV magnesium sulphate at a dose of 25-50mg/kg(0.05 -0.1ml/kg of a 50% solution given slowly. The dose is repeated every 6 hours . After 2 or 3 doses Mg levels are rechecked. Rapid infusion can cause hypotension.The dose is reduced in renal insufficiency. 1ml of 50% MgSo4 contains 500mg of Mg

Long term therapy is often given orally. 10-20mg/kg given 3-4 times a day


Hypomagnesemia
Fractional excretion of Mg
> 4%
< 2%
Renal
Gastrointestinal
Decreased intake
Increased loss
Malabsorption
Drugs
Loop diuretics, cisplatin amphotericin B, cyclosporine, tacrolimus
Others
Aldosterone excess
Hypercalcemia
Hypophosphatemia
Genetics
Hypokalemic alkalosis
Yes
No
Urine calcium/creatinine
Increased
Bartters syndrome
Normal
EAST syndrome
Decreased
Gittelmann syndrome
Increased
AD hypoparathyroidism
AD hypocalcemia with hypercalciuria
Familial hypomagnesema with hypercalciuria
Normal
AR renal Mg wasting
Hypomagnesemia with secondary hypocalcemia
Decreased
AD renal Mg wasting

Hypo/ Hyperkalemia

Contributors: Dr Gowrishankar NC, Dr Prem Kumar L K, Dr Ekambaranath TS, Dr Venkateswari R

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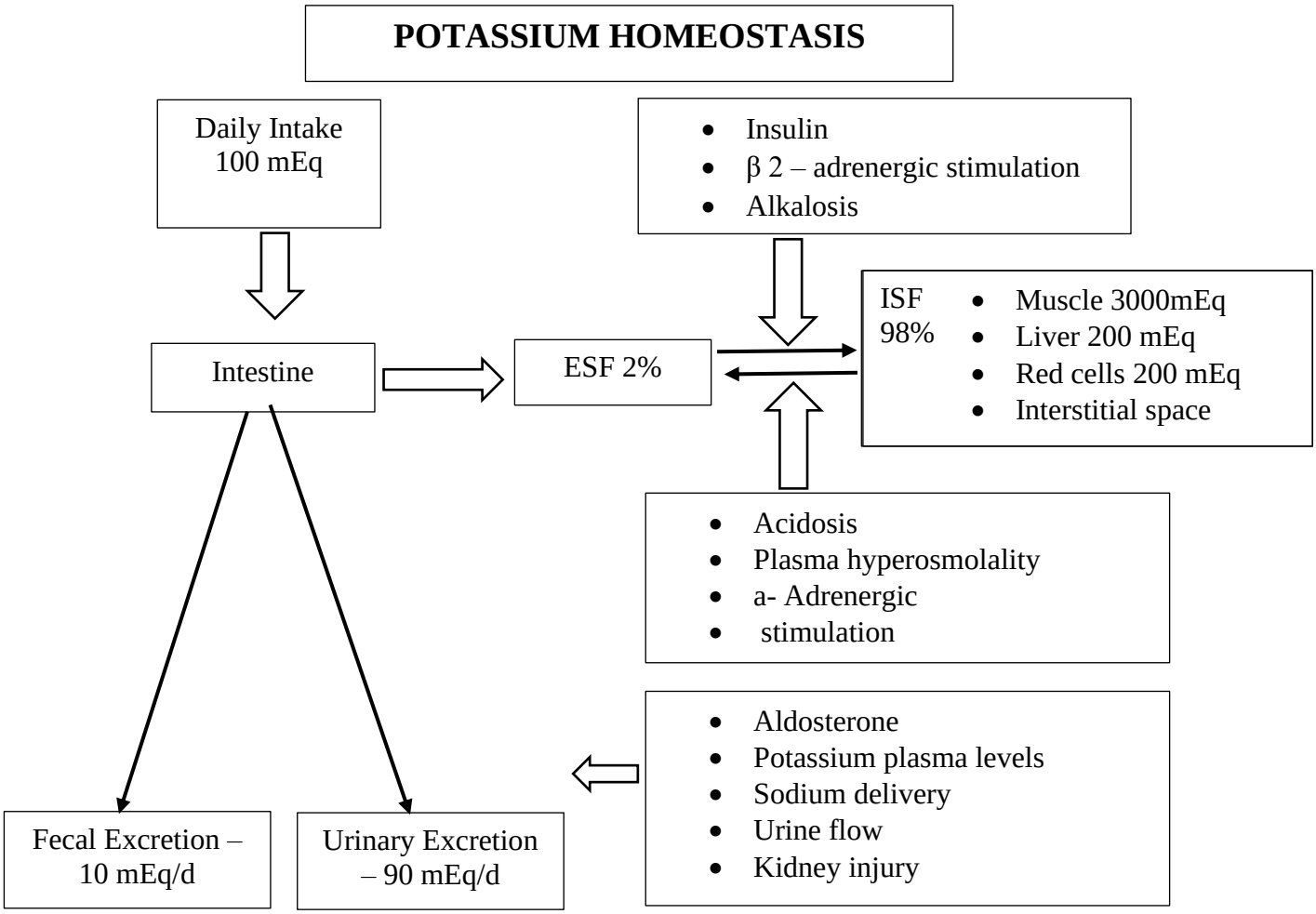


Potassium

- Total body K -50mEq/Kg. Predominantly an intracellular cation. 98% is in the intracellular compartment and majority in the skeletal muscle.
- The normal serum level is 3.5-5.5mEq/L. Higher levels may be seen in newborns and young infants.
- Potassium is essential for growth, to maintain the resting membrane potential of skeletal, smooth, cardiac muscle and nerves. It also helps to regulate cellular volume as well as intracellular calcium content.
- 90% excreted in urine and 10% GIT, sweat. Most of the filtered K is absorbed in the distal convoluted tubule and cortical collecting duct. K is secreted into the tubular lumen in exchange with Na and H ions.

Hyperkalemia

Definition: Serum or plasma concentration of K >5.5mEq/L; in neonates > 6mEq/L (serum K is 0.1-0.7 mmol/L higher)

- Mild hyperkalemia - 5.5-6 mEq/L.
- Moderate hyperkalemia - 6 -7 mEq/L.
- Severe hyperkalemia - >7 mEq/L and or presence of ECG changes

Causes of hyperkalemia

1. Increased K intake- IV/Oral, blood transfusions, parenteral nutrition
2. Increased production followed by transcellular shift-Tumor lysis syndrome, Excessive trauma, rhabdomyolysis, hemolysis, malignant hyperthermia
3. Transcellular shifts- metabolic acidosis, drugs-succinyl choline,beta blockers, digoxin, hyperosmolality(mannitol), hyperkalemic periodic paralysis

Symptoms

May be asymptomatic. Symptoms can range from muscle weakness to ascending flaccid paralysis, palpitations, syncope, arrhythmia and sudden cardiac arrest. Respiratory depression, ileus and paresthesia can occur

ECG changes include tall, peaked T waves, prolonged PR interval, progressive widening of QRS, Sine wave((fusion of QRS and T wave),VT, VF, asystole

A normal ECG does not exclude risk for arrhythmia, as life threatening arrhythmia can occur without warning

HYPOKALEMIA

Definition

- Severe hypokalemia – Potassium level less than 2.5 mEq/L
- Moderate hypokalemia – Potassium level between 2.5 and 3 mEq/L
- Mild hypokalemia – Potassium level between 3 and 3.5 mEq/L

Etiology of hypokalemia

Decreased intake	Increased intracellular uptake	Increased loss-extra renal	Increased Loss-renal	Endocrine
Severe acute malnutrition Anorexia	Metabolic alkalosis Insulin Beta adrenergic agents Heavy metals(barium) Anti -psychotic drugs Hypokalemic periodic paralysis	Diarrhea Emesis Cystic fibrosis	Diuretics DKA Tubulo interstitial disease Bartter syndrome Gitelman syndrome Renal tubular acidosis Amphotericin Liddle syndrome Hypomagnesemia	Aldosterone secreting adenoma Glucocorticoid remediable aldosteronism Apparent mineralocorticoid excess (AME) 11-beta-hydroxylase deficiency 17-alpha-hydroxylase deficiency Thyrotoxicosis

Clinical features

Many patients are asymptomatic. If severe hypokalemia, can present with muscle weakness (headlag, hypotonia, paralysis, respiratory failure, death) cramps, fasciculation and arrhythmias

ECG changes

PR prolongation, flattening of T waves, ST depression, U waves can emerge after the T waves (best seen in the precordial leads).

Management

Emergent treatment is needed in symptomatic patients, or those with ECG changes or severe hypokalemia

- Potassium chloride IV 0.5 to 1 mEq/kg of body weight per hour. The goal is to raise the potassium level by 0.3 to 0.5 mEq/L. May be associated with pain and phlebitis when administered through a peripheral vein. Choose a large vein . External jugular vein is a good option. Maximum adult dose is 40 mEq.

- Do NOT administer undiluted or by IV push . It must always be diluted in infusion fluid (RL or 0.9% sodium chloride).

- Rapid intravenous administration or overdose may cause cardiac arrest. Administer via an infusion pump.
- An infusion with a potassium concentration of no more than 40 mEq/L is given in most situations. Occasionally a higher concentration of 60mEq/L may be needed.
- When adding potassium chloride to an IV fluid bag, mix well by inverting the bag at least 10 times
- Clearly label all bags, syringes, pumps and lines that contain potassium to avoid inadvertent flushing
- Continuous ECG monitoring is needed.
- Serum concentrations should be evaluated 1 to 2 hours after completion of infusion
- May repeat dose as needed based on lab values
- Watch for rebound hyperkalemia
- Iv fluids should not contain dextrose as it can stimulate insulin secretion.

Note: 1 ml of KCl contains 2 mEq.

Asymptomatic patients

Stop diuretics/laxatives and drugs which result in hypokalemia. Use potassium-sparing diuretics if diuretic therapy is required. Treat underlying cause - Diarrhea or vomiting.
Replace ongoing excessive losses.

Moderate hypokalemia: Oral replacement. IV only for those who are unable to take oral medications.

Mild hypokalemia

Increase dietary potassium. Oral K supplements For those who are unable to take enteral potassium, the addition of a maintenance amount of potassium to IV fluids 20mEq/L is sufficient

In asymptomatic patients with chronic hypokalemia (RTA) potassium supplementation may be needed

Oral potassium is preferred over IV potassium in asymptomatic patients. Potassium chloride, phosphate, potassium acetate, potassium citrate-citric acid, and potassium bicarbonate are the various salts available. Potassium chloride is commonly used. Patients with acidosis can be given potassium acetate or citrate.

Dose:
Initial: 1-2 mEq/kg/day in divided doses. Titrate to desired clinical response. Usual range: 1 to 5 mEq/kg/day. Not to exceed 1 to 2 mEq/kg as a single dose up to 40 mEq/dose

Note:

- The strength of K in most commonly available potassium chloride syrup is 20 mEq in 15 ml
- Oral and parenteral potassium can safely be used simultaneously.
- Best taken with or soon after food to reduce gastrointestinal irritation.

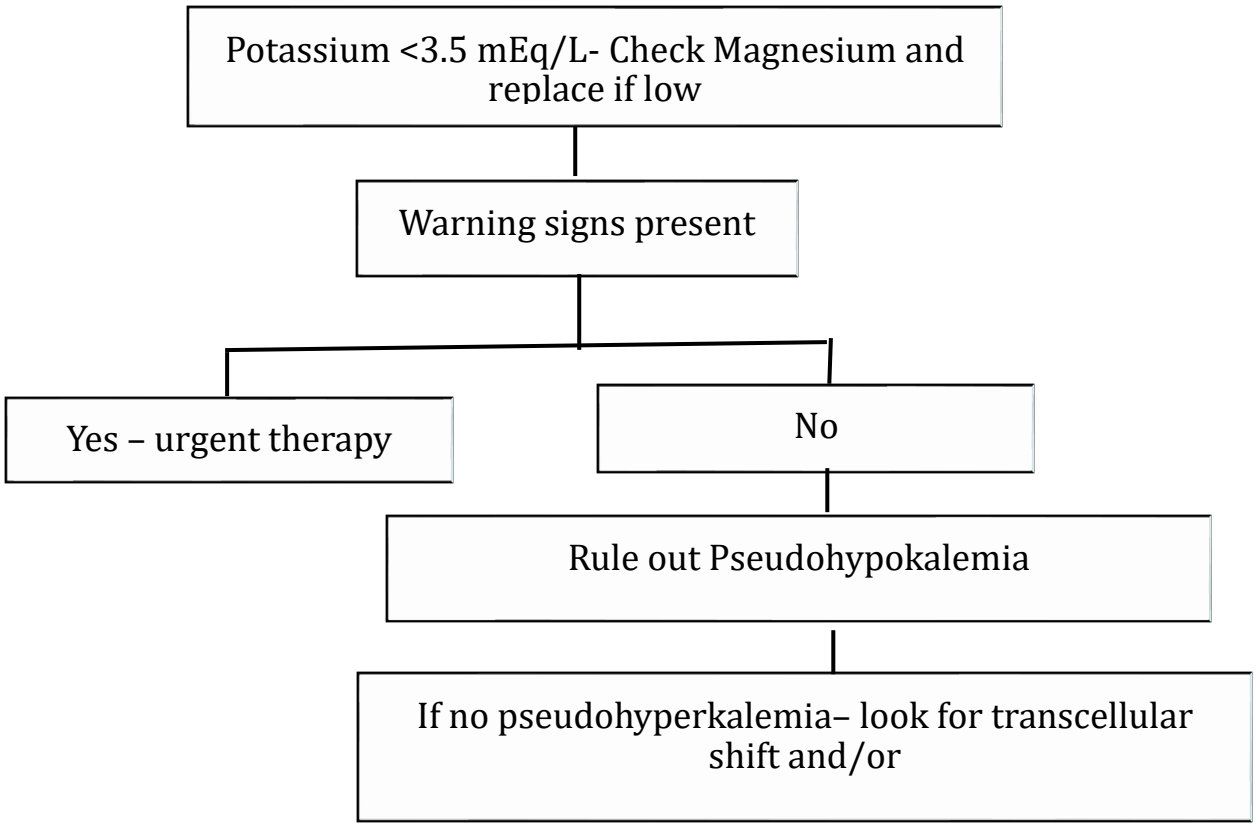
Other treatment:

Magnesium sulphate if hypomagnesemia is the cause.(25-50mg/kg IV over 30 minutes)

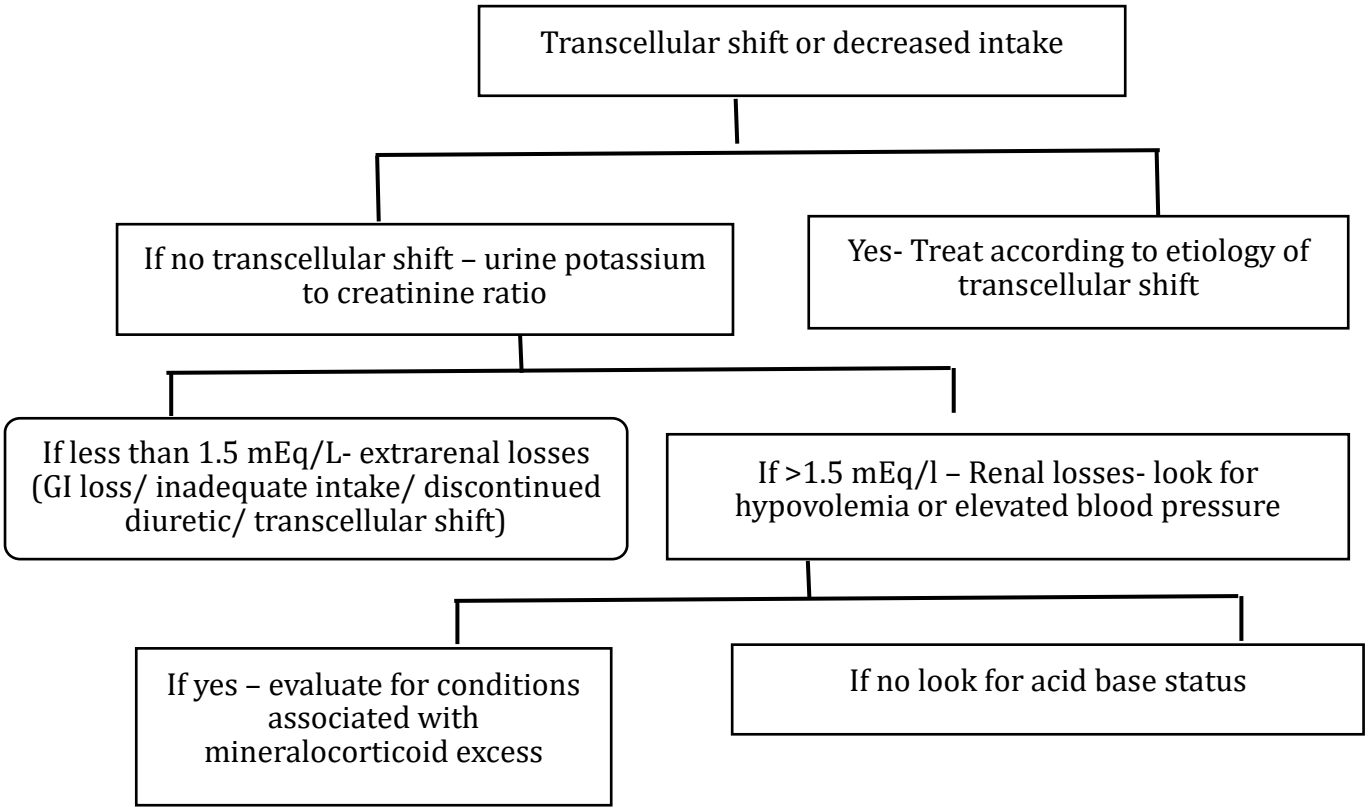
Potassium-sparing diuretic such as amiloride in Bartter, Gitelman

Spironolactone or eplerenone in hyperaldosteronism

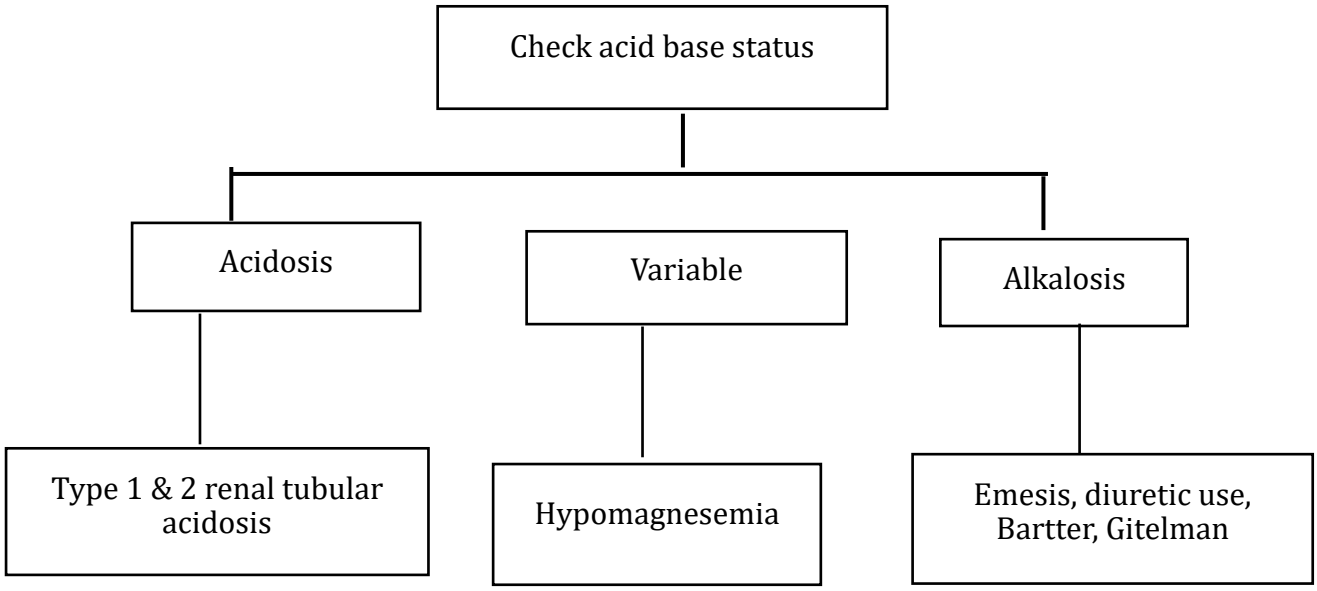
STEP 1- APPROACH TO HYPOKALEMIA



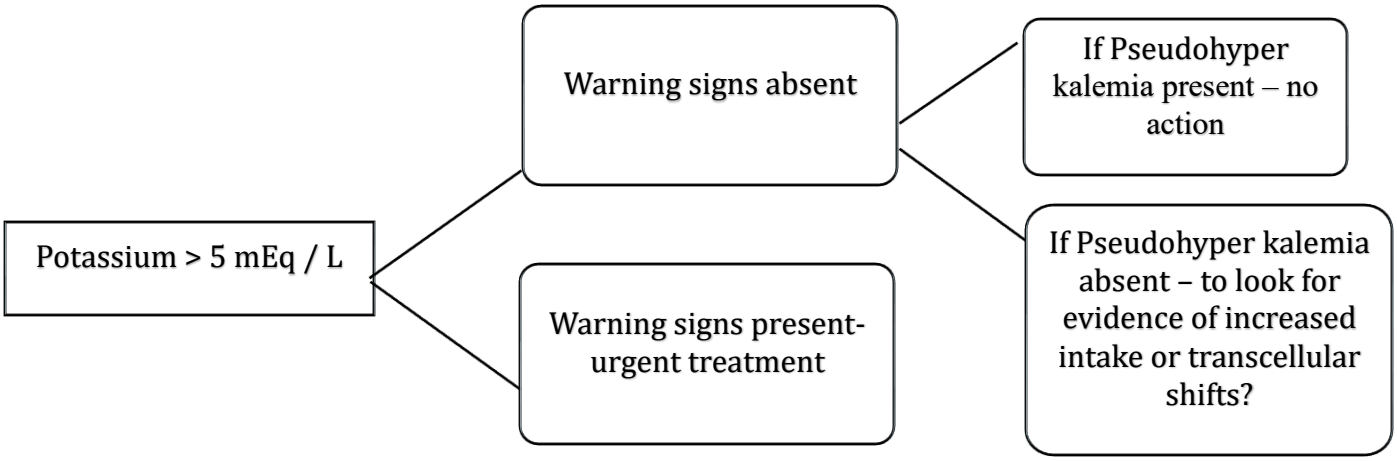
STEP 2- APPROACH TO HYPOKALEMIA



STEP 3- APPROACH TO HYPOKALEMIA



STEP 1- APPROACH TO HYPOKALEMIA



STEP 2- APPROACH TO HYPOKALEMIA