IPNA
International Pediatric Nephrology Association

RAMAIAH Medical College

INDIAN ACADEMY OF PEDIATRICS BENGALURU

# Water & salt
# Essence of life

## Workshop on Fluid & electrolytes in children

**Pre-conference workshop**

**12th IAP Bangalore Pedicon 2025**

**Course work book**

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## Disclaimer

This workbook is prepared to guide structured learning of fluid and electrolyte problems in children

This is not replacement for standard textbooks and guidelines

# Program schedule

**Date and time:** 13/06/2025 8.30am - 5pm

| Time | Topic | Speaker |
|---|---|---|
| **8.30am – 9am** | **Registration and Pretest** | |
| | **Morning session** | |
| 9 – 9.30am | Fluids - Physiology | Dr Hamsa |
| 9.30am – 10am | Fluid therapy in children | Dr Namita Ravikumar |
| **Workstation 1 - DIY – Solve fluid and electrolyte problems in children**<br>10 am – 10.45am | Fluids – a) Shock<br>b) Dehydration<br>c) AKI | Dr Namita/ Dr Hamsa/<br>Dr Shankar Prasad/<br>Dr Narayan/ Dr Hasmitha |
| **10.45 am – 11am** | **Break** | |
| 11am – 11.30am | Potassium balance and approach to potassium disorders | Dr Nivedita Kamath |
| **Workstation 2 - DIY – Solve fluid and electrolyte problems in children**<br>11.30 am – 12.15pm | Potassium a) Hypokalemia<br>b) Hyperkalemia | Dr Nivedita/<br>Dr Namita/ Dr Hamsa/<br>Dr Amruthvarshini/<br>Dr Srividya |
| 12.15pm – 12.45pm | Acid base physiology | Dr Shankar Prasad |
| 12.45pm – 1.15pm | Interpretation of ABG | Dr Rajashekar |
| **1.15pm – 2pm** | **Lunch** | |
| | **Post lunch session** | |
| 2pm – 3pm | Quiz on acid base disorders | Dr Shankar Prasad |
| 3pm – 4pm<br>**Online session** | Sodium balance and approach to sodium disorders | Dr Larry Greenbaum |
| **Workstation 3 - DIY – Solve fluid and electrolyte problems in children**<br>4pm – 4.45pm | Sodium a) Hyponatremia<br>b) Hypernatremia | Dr Shankar Prasad/<br>Dr Namita/ Dr Hamsa/<br>Dr Srividya / Dr Apoorva |
| **4.45pm – 5pm** | **Post test** | |

# Fluids – Physiology

Total body water content changes drastically from before birth until one year of age.

| Age | % TBW |
|---|---|
| Fetus | 85-90% |
| Neonate Term / Preterm | 75% / 80% |
| 1 year - puberty | 60% |
| Adolescent male | 60% |
| Adolescent female | 50% |

Total body water: Intracellular fluid + Extracellular fluid (Plasma water + Interstitial fluid)

Total body water varies with age (figure 1).

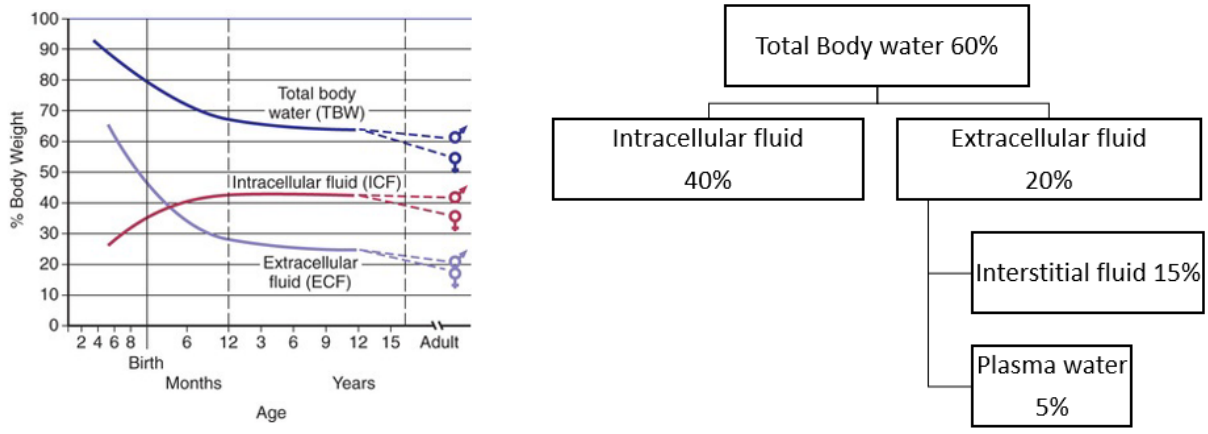



Figure 1: TBW, ECF and ICF as a percentage of body weight as a function of age and distribution of TBW.

## Constituents of ECF and ICF

Comparisons of the composition of the extracellular fluid, including the plasma and interstitial fluid, and the intracellular fluid are shown in Figure 2.

The composition of extracellular fluid is carefully regulated by various mechanisms, but especially by the kidneys.

*Understanding the fluid and electrolyte composition of various body fluids is important in deciding the choice of fluids in critically ill children.*

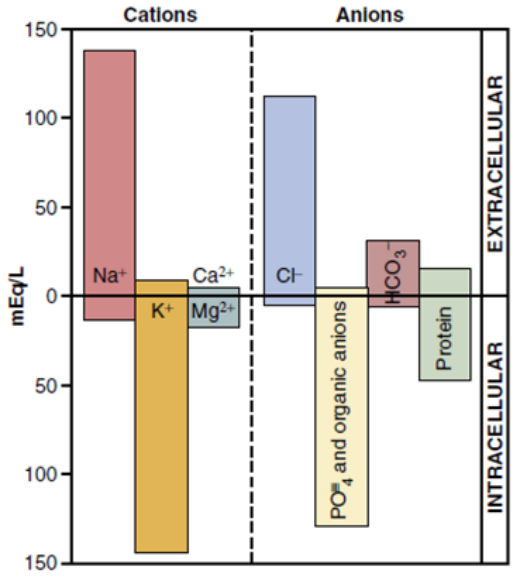



Figure 2 : Components of ECF and ICF

## Osmolality

The ICF and the ECF are in osmotic equilibrium because the cell membrane is permeable to water.

The ECF osmolality can be determined and usually equals ICF osmolality.

Plasma Osmolality can be measured using the formula:

$$\text{Osmolality} = 2\times[\text{Na}]+[\text{glucose}]/18+[\text{BUN}]/2.8$$

Normal plasma osmolality is 285-295 mOsm/kg.

Since urea is an ineffective osmole, the effective osmolality (also called the tonicity ) determines the osmotic force that is mediating the shift of water between the ECF and the ICF.

$$\text{Effective osmolality} = 2\times[\text{Na}]+[\text{glucose}]/18$$

## Osmolality vs Osmolarity vs Tonicity

- Osmolarity is the number of osmoles of solute per litre of solution.

  Osmolarity depends on the volume of the solution, and therefore on the temperature and pressure of the solvent

- Osmolality is the number of osmoles of solute per kilogram of solvent

  Osmolality depends on the mass of the solvent which is independent of temperature and pressure.

- Tonicity is the osmotic pressure between two compartments, and is related to the difference in the concentration of "effective" osmoles between them
- Effective osmoles are those substances which are unable to penetrate the membrane between compartments, and therefore they are effective in their contribution to the osmotic pressure gradient. Eg: Na, Glucose
- Ineffective osmoles are those that are able to equilibrate between compartments, and that are therefore unable to contribute to the osmotic pressure gradient. Eg: Urea.
- *In simple terms, hypothetically when a cell is immersed in a hypotonic solution, as the gradient exists across the semi-permeable membrane, the cell swells up as the water enters the cell. In a hypertonic solution, the cell shrinks as water is drawn out (Figure 3).*

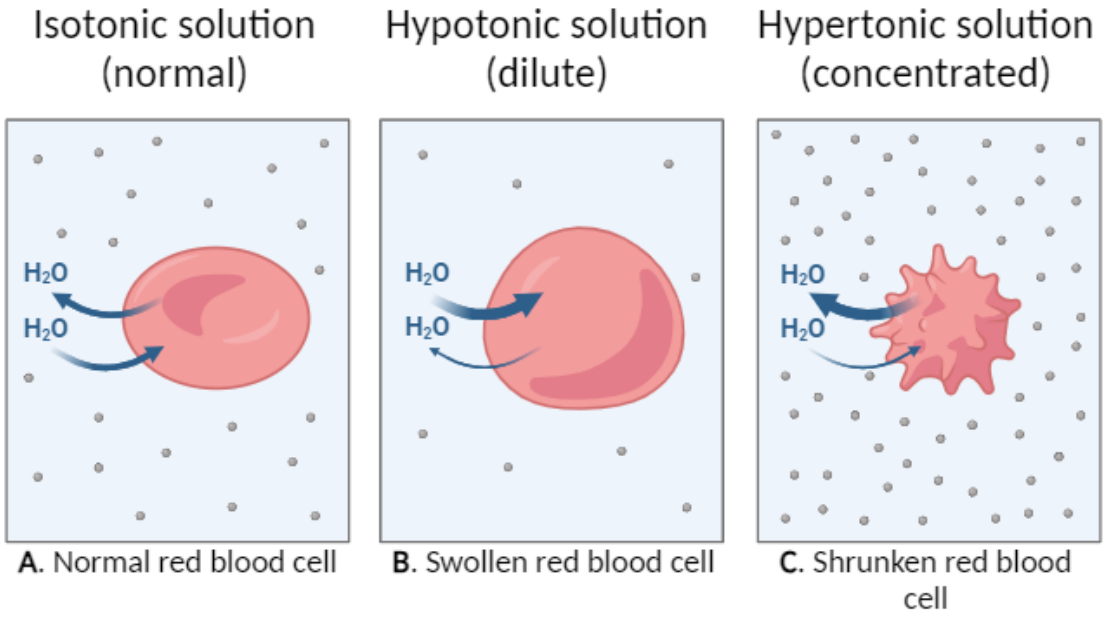



Figure 3: Cells immersed in solutions of variable tonicity

## Regulation of osmolality

The regulation of plasma osmolality and the intravascular volume is controlled by independent systems

a. Water balance determining the osmolality
b. Sodium balance determining the volume status.

***Control of osmolality is subordinate to maintenance of an adequate intravascular volume.***

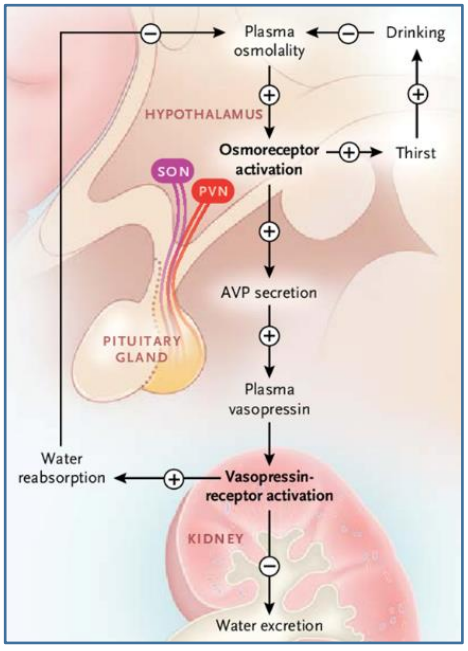
The plasma osmolality is tightly regulated by anti-diuretic hormone (ADH), water intake and glomerular filtration rate (GFR) and maintained at 285-295 mOsm/kg (figure 4).

ADH secretion is a graded adjustment as the osmolality changes. The regulation of ADH secretion is tightly linked to plasma osmolality, responses being detectable with a 1% change in osmolality. ADH secretion reduces to zero, when the plasma osmolality is low, allowing excretion of maximally dilute urine.

Water intake is regulated by hypothalamic osmoreceptors, which stimulate thirst even with a small increase in the serum osmolality. Sensation of thirst is stimulated with only 1-2% change in plasma osmolality, but requires moderate volume depletion.

The capacity of kidneys to maximally concentrate urine to 1200 mOsm/kg and pass maximally dilute urine up to 30-50 mOsm/kg in response to the volume status of the individual also helps in maintaining normal osmolality.

Figure 4: Regulation of serum osmolarity by thirst, ADH and kidneys



# Case 1

| Case details | | Case Analysis & Questions |
|---|---|---|
| 2-year-old boy | | Design a 6-hour resuscitation plan |
| Weight: 10 kg | | |
| presented with | | |
| fever, cough x 2 days | | |
| hurried breathing 1 day | | |
| On Examination child is irritable | | |
| **Vitals:** HR 165/min,bounding pulses | | |
| BP 74/40 mm/Hg | | |
| RR-70/min with retractions | | |
| SpO2 96% on NRBM | | |
| Temp 102F | | |
| RS: Reduced air entry in right infrascapular area, Bronchial breath sounds + | | What are your hemodynamic goals and monitoring targets? |
| Abdomen: No organomegaly | | |
| CNS: irritable | | |
| CVS: Normal heart sounds | | |
| Investigations | | |
| **VBG** | | |
| pH | 7.28 | |
| $pCO_2$ | 30 mm Hg | |
| $HCO_3$ | 14 mEq/L | |
| Lactate | 2.6 mmol/L | |
| | | |
| What are the specific investigations and interventions you would plan? | | |
| | | |
| | | |
| | | |
| | | |
| | | |
| | | |

# Fluid therapy

## Different indications of fluids

1. Resuscitation fluids - fluids given to replace intravascular volume in shock. In a child with shock, fluid administered needs to stay in the intravascular compartment, hence isotonic fluids are used. Isotonic crystalloids like 0.9% NaCl (normal saline) and Ringer's lactate are the choice of fluids in resuscitation.

*Fluid bolus dose and duration*

The initial bolus fluid of 10-20 ml/kg is given over 30-60 minutes while assessing therapeutic end points of shock like improvement in tachycardia, blood pressure (BP), pulse volume and capillary filling time (CFT).

In a child with hypotension and history of fluid loss like diarrhea, vomiting, blood loss etc, larger volumes of fluid can be given by push and pull technique to restore volume quickly.

In children with suspected cardiac cause, severe acute malnutrition (SAM) and chronic anemia, smaller aliquots of fluid bolus (5 ml/kg) over a prolonged duration (60 minutes) may be considered on a case-to-case basis while simultaneously monitoring for clinical signs of fluid overload like increase in respiratory rate, new onset crackles in the basal lung zones and hepatomegaly.

In centres with facility of point of care ultrasound (POCUS), IVC diameter, variation with respiration can be assessed prior to the next fluid bolus along with lung B-lines for fluid overload. Cardiac contractility by bedside echocardiography can also be assessed.

In certain conditions of extreme capillary leak like dengue shock, colloids are used when shock is refractory to crystalloids as they are retained for a longer duration in the intravascular compartment.

2. Replacement fluids

Different body compartment fluid losses are replaced with fluids of similar composition.

Stool losses in diarrhea are replaced with ORS by enteral route or Ringer lactate IV

Nasgastric or emesis losses are replaced with Ringer Lactate

Urinary losses in polyuria are replaced with 0.45%NaCl

3. Deficit replacement

Some dehydration - 75 ml/kg ORS or RL

Severe dehydration - 30 ml/kg RL over 1/2 hour in older children and over 1 hour in infants followed by 70 ml/kg over 2.5 hours in older children and over 5 hours in infants

4. Maintenance fluids

Estimates of maintenance fluid requirement in children are as per Holliday-Segar formula derived from normal children. However, in critically ill children, maintenance fluid requirements are about 60-70% of that estimated by the formula due to lesser insensible water losses in the ICU environment and risk of SIADH.

Holliday Segar Formula for maintenance fluid over 24 hours

100 ml/kg for first 10 kg

Add 50 ml/kg for next 10-20kg

Add 20 ml/kg for > 20 kg

**Composition of different intravenous fluids**

| Fluid type | Osmolality (compared with plasma) | Tonicity (with reference to cell membrane) | Na+ (mmol/ L) | Cl- (mmol/ L) | K+ (mmol/ L) |
|---|---|---|---|---|---|
| 0.9% sodium chloride (Normal saline) | Iso | Iso | 154 | 154 | 0 |
| Ringer Lactate | Iso | Iso | 131 | 109 | 5 |
| Plasmalyte | Iso | Iso | 140 | 98 | 5 |
| 0.9% sodium chloride with 5% glucose (DNS) | Hyper | Iso | 154 | 154 | 0 |
| 0.45% sodium chloride with 5% glucose(1/2 DNS) | Hyper | Hypo | 77 | 77 | 0 |
| 0.45% sodium chloride with 2.5% glucose | Iso | Hypo | 77 | 77 | 0 |
| 0.45% sodium chloride (1/2 NS) | Hypo | Hypo | 77 | 77 | 0 |
| 5% glucose | Iso | Hypo | 0 | 0 | 0 |
| 10% glucose | Hyper | Hypo | 0 | 0 | 0 |
| 3% NaCl | Hyper | Hyper | 513 | 513 | 0 |

# Case 2

| Case details | | Case Analysis & Questions |
|---|---|---|
| S, 2-year-old girl<br>Weight 12 kg<br>presents with<br>H/o large volume watery loose stools since 2 days, 12-15 times/day.<br>Initially she had good oral intake but not taking anything since this morning and lethargic.<br>On examination she has sunken eyes, dry oral mucosa<br>HR 145/min, thready peripheral pulse<br>BP 84/50 mmHg<br>RR 30/min<br>Systemic examination normal apart from lethargy. | | Identify the state of hydration<br>What are the goals of management?<br>Fluid therapy prescription for above case |
| **Investigations** | | |
| Hb (gm/dl) | 11.8 | |
| TLC (cc/$mm^3$) | 9,400 | |
| Plt (lakhs/$mm^3$) | 4.5 lakh | |
| Na+/ K+ /Cl- (mEq/L) | 134/3.5/98 | |
| Urea/Creat (mg/dL) | 68/ 0.7 | |
| GRBS | 98 | |
| VBG | pH- 7.3, Hco3- 16 mEq/l, Pco2- 28 mm Hg | |

## Assessment of dehydration

1. If pre-illness weight is known, then deficit = Pre-illness weight – present weight divided by pre-illness weight multiplied by 100
2. If weight is not known, estimation of probable deficit

| **Severity** | **Infants** | **>1year or >10kg** |
|---|---|---|
| Mild | 5% | 3% |
| Moderate | 10% | 6% |
| Severe | 15% | 9% |

- **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 deficits of fluid and electrolytes | Urgent replacement of existing deficits of fluid and electrolytes |
| **Fluid therapy** | Maintenance (oral) | Rehydration (oral) | Rehydration (IV) |

| Treatment plan | Plan–A | Plan–B | Plan–C |
|---|---|---|---|
| **Rehydration fluid** | Oral rehydration solution (ORS)/homemade solutions | ORS | RL* |
| **Amount of rehydrating fluid** | For every loose stool: 10 mL/kg<br>Age up to 2 months—5 teaspoons/purge<br>2 months to < 2 years → 50–100 mL<br>Age 2–10 years → 100–200 mL<br>Older child: As much as desired<br>Plus Free access to drinking water | 75 mL/kg Over 4 hours<br>Plus<br>Non-breastfed infants<br>100–200 mL of clean drinking water<br>Older children and adults: Free access to plain water in addition to ORS | IV Fluid<br><1 year:<br>30ml/kg over 1 hour<br>70ml/kg over 5 hours<br>>1 year:<br>30mL/kg over ½ hour<br>70mL/kg over 2½ hours<br>Plus ORS (5 mL/kg/h) start orally as soon as child is able to drink |

***Rehydrating fluids for severe dehydration:**

- RL is recommended fluid. Normal saline (0.9%) or half strength Darrows solution can be used if RL is not available
- In malnourished children, rehydration is done slowly over 6-12 hours

## Case 3

| Case details | Case Analysis & Questions |
|---|---|
| Ms A, 7-year-old girl is admitted to PICU with diagnosis of severe pneumonia with septic shock.<br><br>Weight 20kg, Height 125cm, BSA 0.8$m^2$<br><br>Her HR 140/min, feeble pulses, RR -34/min, BP 84/50mm Hg, increased work of breathing noted. Respiratory system examination revealed reduced air entry on the right infrascapular region with bronchial breath sounds in right mammary and axillary region.<br><br>At admission (8pm), she was given 20ml/kg NS bolus, repeated twice and was started Inj Adrenaline and started on HFNC. He was given IVF DNS at 80ml/hour and started on Inj ceftriaxone. In next 12 hours, child had one more episode of hypotension, 20ml/kg NS bolus was given and started on Inj Nor adrenaline.<br><br>Child was mechanically ventilated in view of refractory shock and increased respiratory distress.<br><br>At 24 hours BP noted at 50th centile. Her urine output since admission is 300ml. | What is your diagnosis?<br><br>List the problems in this child.<br><br>Write prescription for fluids in this child? |

| Investigations | | |
|---|---|---|
| Hb (gm/dl) | 10 | 9.8 |
| TLC (cc/$mm^3$) | 25,000 P85 | 18,200 |
| Plt (lakhs/$mm^3$) | 3.4 | 2.8 |
| Na+/ K+ /Cl- | 132/4.5/100 | 135/4.8 /118 |
| S Creatinine (mg/dl) | 1.8 | 2.3 |
| CRP (mg/dl) | 76 | |
| pH/pCO2/PO2/HCO3/BE | 7.21/43/65/12/ -10 | 7.31/34/102/18/ -4.2 |

## Fluid management in AKI

Fluid management in a child with AKI depends upon the volume status, etiology of AKI and the urine output.

If the etiology of AKI is prerenal or secondary to severe volume losses, the child has to be given fluids to correct the dehydration along with maintenance fluids, and monitoring of the urine output.

A child with established AKI as in septic AKI (not yet on RRT), fluids need to be restricted to insensible losses (400ml/$m^2$/day) plus urine output. The volume status, urine output, obligate fluid requirement, need for blood/blood products and nutritional needs of the child have to be kept in mind while prescribing fluids.

In a child with AKI and RRT, fluid restriction need not be very stringent as RRT can help in removal of ultrafiltration (based on hemodynamic status of the child). Percentage of fluid overload, hemodynamic status, urine output, daily fluid requirements should be considered while prescribing fluids in a child with AKI on RRT.

If the child has polyuria, the child will need maintenance fluids along with replacement for the excess urine output.

Fluid prescription in a child with AKI has to be reassessed once in 4-6 hours and prescription modified accordingly.

*Choice of fluids:*

Fluid should preferably be given orally or enterally and should be incorporated in the nutrition of the patient. When enteral fluid is not feasible intravenous fluids may be given. In a child who has severe oliguria and is not yet on RRT, half normal saline may be given.

Colloids, blood and blood products should be avoided in oliguric children who are not yet on RRT as they may increase blood pressure or cause respiratory distress due to pulmonary edema.

# Potassium Homeostasis

- Major intracellular cation
- Extracellular potassium is tightly regulated by intracellular shift mechanisms
- Intracellular shift is regulated by insulin and catecholamines (beta receptors)
- 90% of potassium is excreted by the kidneys
- In the kidney, potassium is freely filtered at the glomerulus and 90% is reabsorbed in the proximal tubule
- The aldosterone-sensitive distal nephron is the major segment of the nephron that causes potassium excretion
- Distal delivery of sodium and serum aldosterone are the major factors influencing potassium excretion

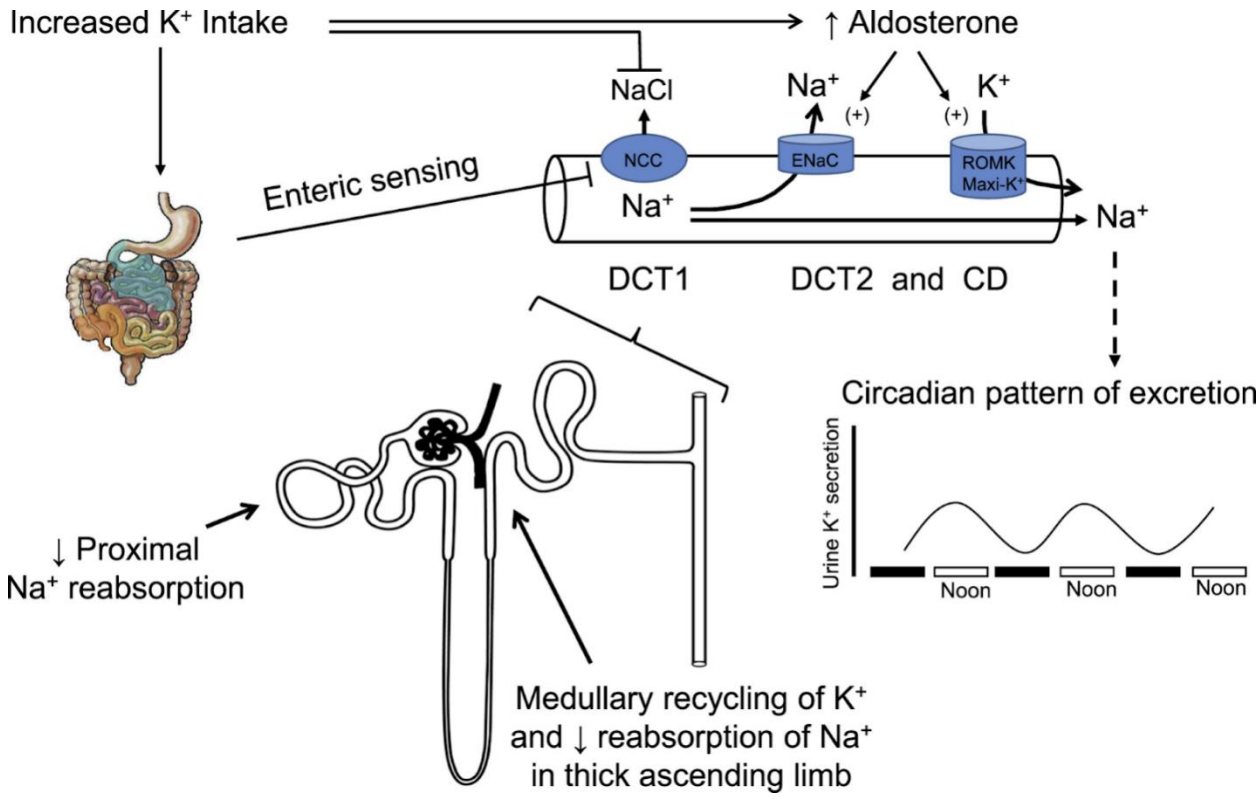



**Figure 5: Mechanisms underlying the normal kidney's capacity to excrete K+**

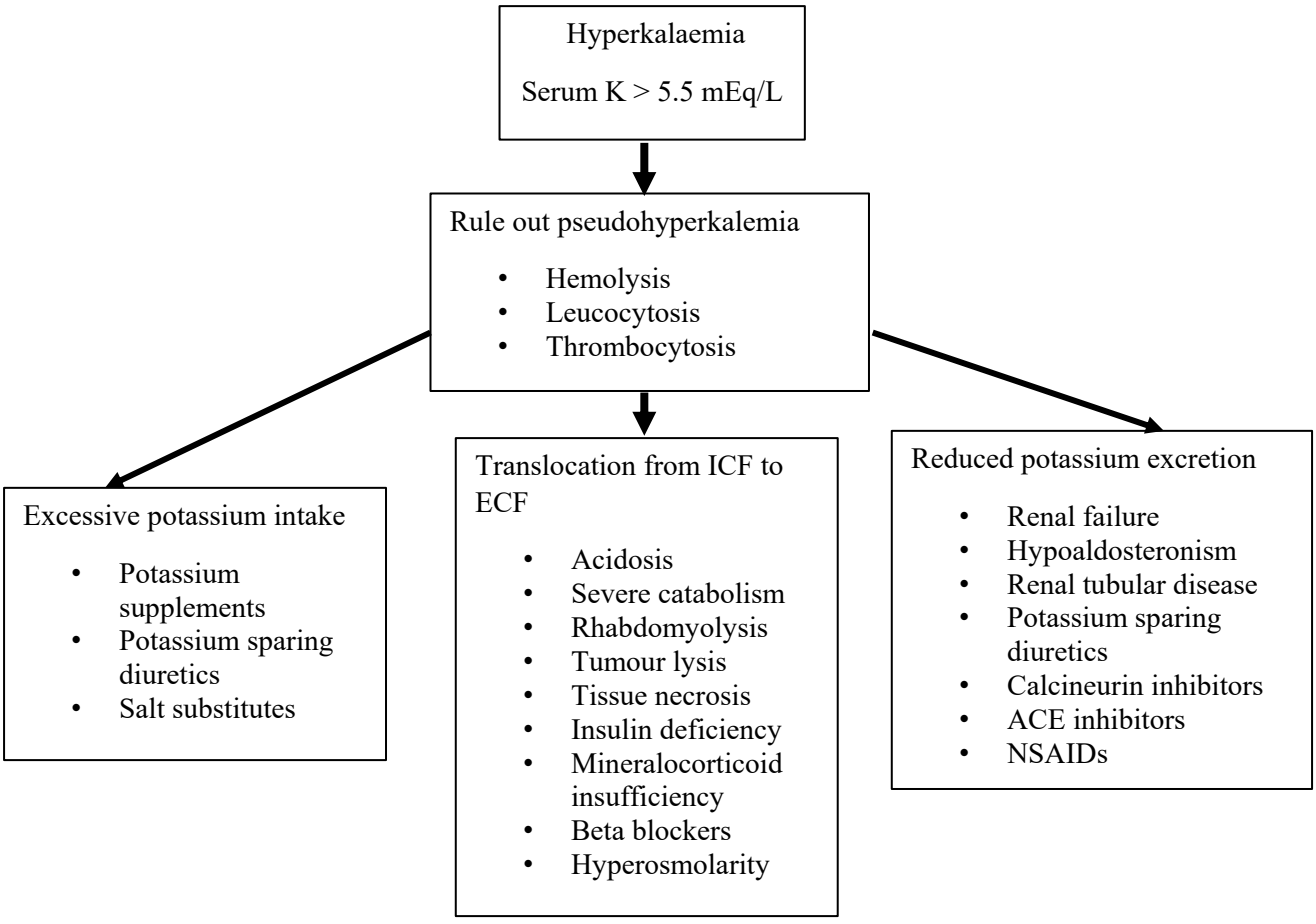
# Approach to Potassium disorders

## Hyperkalemia

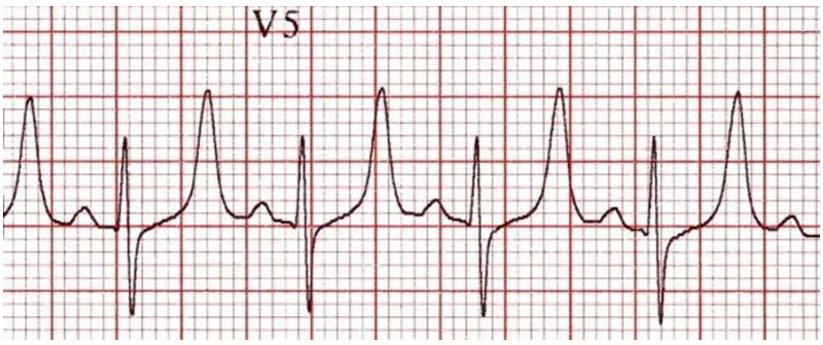
Serum potassium >5.5 meq/L in a child or >6meq/L in a neonate

Severe Hyperkalemia - Serum potassium >7 meq/L or >6.5 meq/L with ECG changes

## Approach to hyperkalemia



**ECG changes in hyperkalemia**




Tall peaked t waves

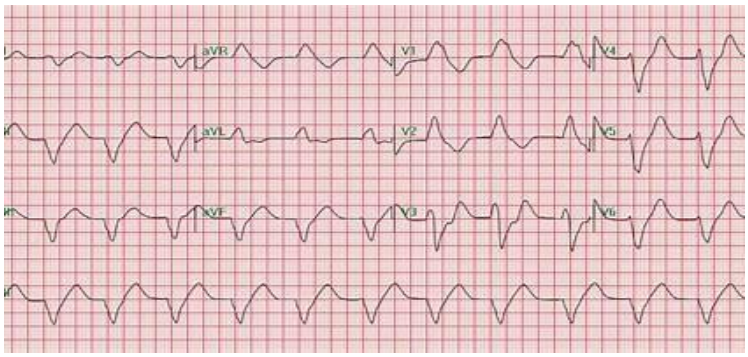
K >5.5 meq/L


Prolonged PR interval

K >6.5 meq/L


Absent P wave

K > 7 meq/L



Sine wave

K >7 meq/L

**Approach to Hypokalemia**

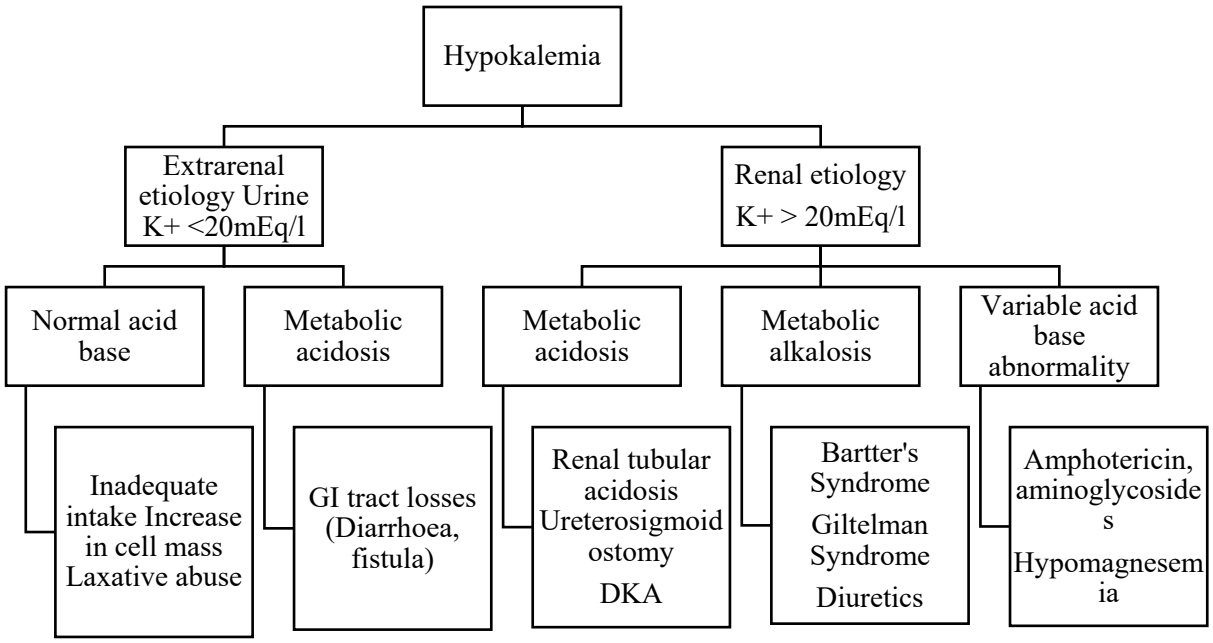
Definition: Serum potassium < 3.5 meq/L;

Severe hypokalemia - Serum potassium <2.5 me/L or <3 meq/L with ECG changes

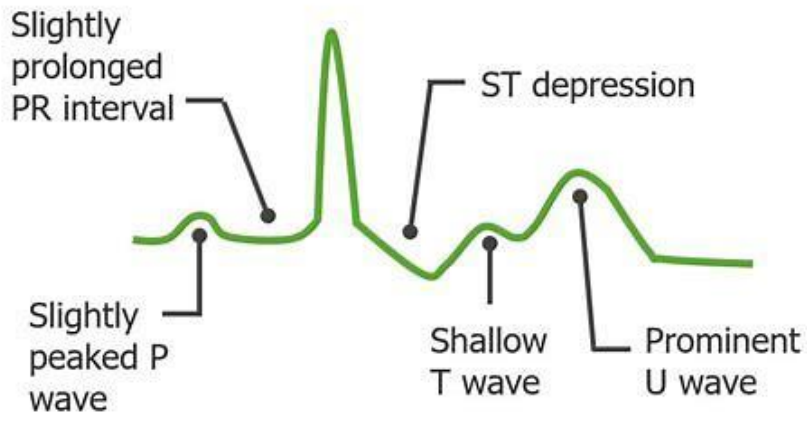
Etiology of hypokalemia

| Hypokalemia Without K Depletion | Hypokalemia With K Depletion |
|---|---|
| Spurious Hypokalemia<br>High WBC count | **GI loss**<br>• Vomiting, Diarrhea<br>• Laxative abuse |
| **Movement with ECF to ICF**<br>• Insulin<br>• Catecholamine excess<br>• Administration of beta-adrenergic excess<br>• Hypokalemic periodic paralysis<br>• Alkalosis | **Skin loss**<br>• Excessive sweating<br>• Burns |
| | **Renal Loss**<br>• Recovery from acute renal failure<br>• Use of loop, thiazide diuretics<br>• Renal tubular acidosis<br>• Bartter & Gitelman syndrome<br>• Syndromes of mineralocorticoid excess<br>• Interstitial Nephritis<br>• DKA |

Approach



**ECG changes in hypokalemia**



# Case 4

## Case details

A 14-year-old boy, known c/o type 1 diabetes mellitus since last 2 years presented to the emergency with fever for 2 days and vomiting for last 2days. Child has been not complaint with insulin.

O/E

Wt 40kg, Ht 135cm

HR 112/min, good volume; RR 34/min, acidotic breathing; BP 110/70mm Hg

Alert, active, signs of mild dehydration +

Systemic examination - normal

He was diagnosed as severe DKA. Child was started on fluids as per ISPAD guidelines.

After 6hours, child c/o weakness and has 1 episode of vomiting

## Investigations

| | At admn | After 6 hours |
|---|---|---|
| Hb (gm/dl) | 11.6 | |
| TLC (cc/$mm^3$) | 8,200 | |
| Plt (lakhs/$mm^3$) | 3.2 | |
| Na+/ K+ /Cl- (mEq/L) | 134/4.2/107 | 132/2.6/108 |
| S Creatinine (mg/dl) | 0.8 | |
| pH/pCO2/PO2/ HCO3/BE | 6.9/25/80/8.2/ -12 | 7.1/24/82/10.3/ -11 |
| Urine | Gluc 3+, KB ++ | |
| GRBS ( mg/dl) | 340 | 310 |

## Case Analysis & Questions

What is your diagnosis?

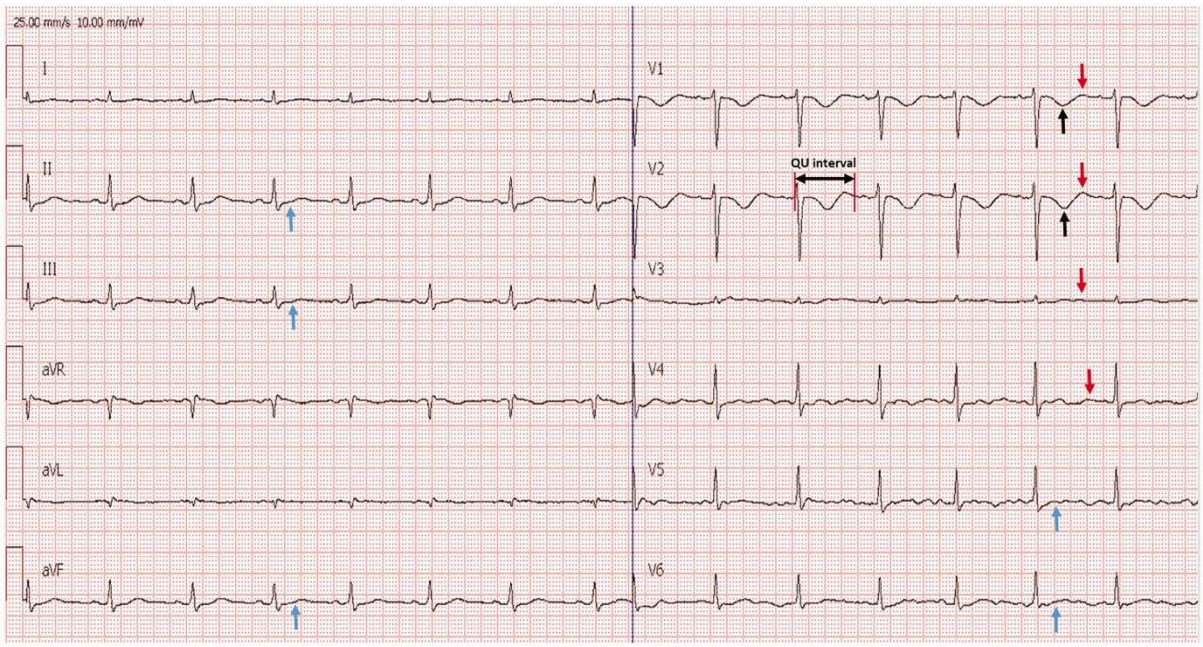
List the problems in this child.

Write prescription for management of hypokalemia

# Case 5

| Case details | Case Analysis & Questions |
|---|---|
| A 5-year-old girl (Wt 10kg, Ht 85cm) has presented with loose stools for 3 days followed by weakness of all 4 limbs noted for 1 day. No h/o loss of consciousness, seizures, slurring of speech noted.<br><br>There is history failure to thrive, polydipsia and polyuria with craving for salty foods, noted since early infancy.<br><br>History of two episodes of weakness in the past following diarrheal illness at 2 years and 3 years of age. These episodes of weakness resolved spontaneously after variable period of time.<br><br>On examination,<br><br>HR 80/min, RR – 22/min, BP 88/60mm Hg, SpO2 – 98% @room air<br><br>alert, active, mild pallor present, dry oral mucosa +, skin turgor prolonged, widening of wrists with genu valgum present,<br><br>CNS examination –<br><br>GCS 15/15, no cranial nerve deficits,<br><br>Hypotonia of all 4 limbs with absent reflexes motor in all limbs. | What is your diagnosis?<br><br>List the problems in this child.<br><br>Write prescription for management of hypokalemia. |

| Investigations | |
|---|---|
| Hb (gm/dl) | 9.6 |
| TLC (cc/$mm^3$) | 16,200 |
| Plt (lakhs/$mm^3$) | 4.8 |
| Na+/ K+ /Cl- (mEq/L) | 134/2.1/118 |
| S Creatinine (mg/dl) | 0.2 |
| pH/pCO2/PO2/HCO3/BE | 7.18/43/80/8/ -12 |
| S Ca/PO4/ALP | 8.4/3.1/980 |
| ECG | Next page |
| USG KUB | B/L medullary nephrocalcinosis |


25.00 mm/s 10.00 mm/mV
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
QU interval

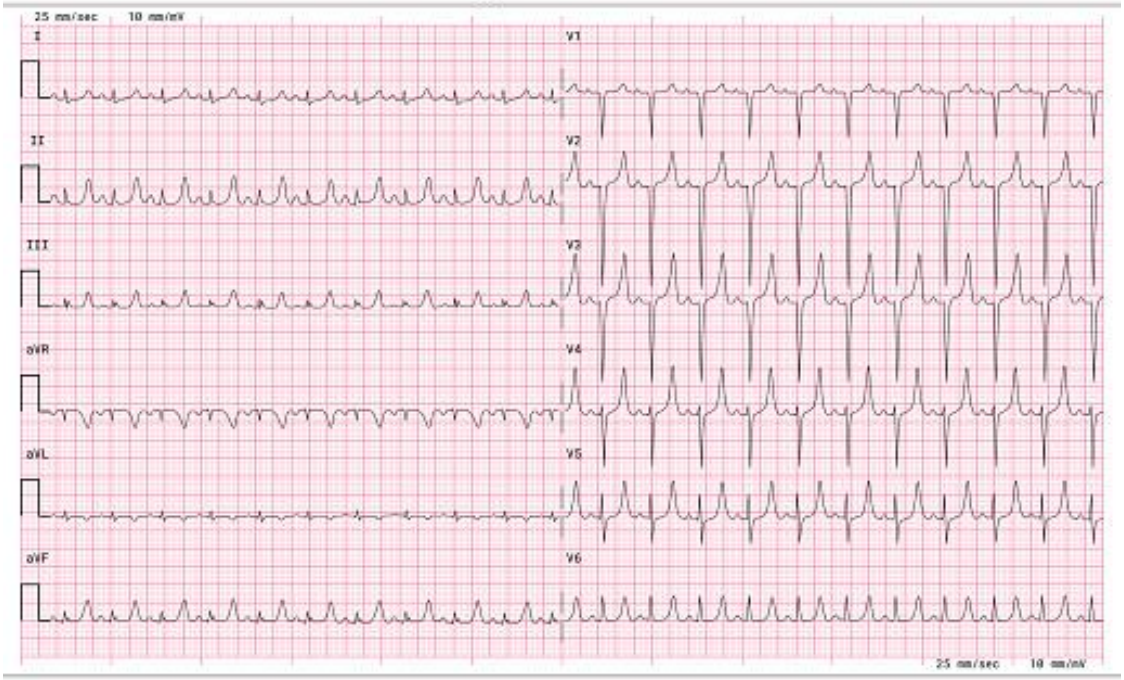
**Treatment of hypokalemia**

- Monitor ECG
- Check renal function – supplement potassium cautiously in children with oliguria/renal insufficiency
- Correct volume deficit if present.
- Potassium correction: oral or intravenous.
    - Oral - safest. Preferred route if the serum K+ is > 3.5 mEq/L, no life-threatening complications.
    - IV route – severe or symptomatic hypokalemia.
- K+ in intravenous fluids to 40 mEq/L (maximum K through peripheral line)
- Acute K+ correction: in children with ECG changes
    - 0.3 – 0.5 mEq/kg/hr infused by a syringe infusion pump preferably via a central line.
    - K deficit: 0.3(ml) x bodyweight (kg) x (normal potassium – measured potassium)
    - Infusion rate should not exceed 1mEq/kg/hr
- Treat the underlying cause
- Correct hypomagnesemia

# Case 6

| Case details | | Case Analysis & Questions |
|---|---|---|
| 8yr old male child with c/o progressive abdominal distension and pain since 1month, fever on and off for one month.<br>Wt 25kg, Ht 120cm<br>On examination, dull looking child,<br>pallor+, significant lymphadenopathy<br>P/A-Mass in RIF extending around umbilical region.<br>CECT abdomen + pelvis revealed mass in ileocecal region-possibility of neoplasm, IHC of biopsied lesion revealed-Burkitts lymphoma.<br>He was started on cytoreduction with prednisolone and started on hyperhydration, rasburicase. | | What is your diagnosis?<br><br><br><br><br><br><br><br>List the problems in this child.<br><br><br><br><br><br><br> |
| **Investigations** | | Write prescription for management of hyperkalemia |
| Hb (gm/dl) | 7.2 | |
| TLC (cc/$mm^3$) | 26,200 | |
| Plt (lakhs/$mm^3$) | 1.2 | |
| PS | Blasts + | |
| Na+/ K+ /Cl- (mEq/L) | 136/6.5/112 | |
| S Creatinine (mg/dl) | 0.8 | |
| pH/pCO2/PO2/HCO3/BE | 7.18/43/80/8/ -12 | |
| S Ca/PO4/Uric acid | 7.9/6.9/13.2 | |
| ECG | given in next page | |

# Hyperkalaemia



## Principles of therapy for hyperkalemia

- Acute hyperkalemia is potentially and unpredictably lethal.
- Verify values with a repeat lab draw
- Assess immediate risk
    - *Rapid rise of K+> 6.0-6.5 mEq/Litr*
    - *ECG changes*
    - *Associated acidosis*
    - *Hypocalcemia*
- Assess renal function, place child under continuous ECG monitoring

### Cardioprotective agents

| Agent | Dose/route | Onset, duration | Mode of action | Comments |
|---|---|---|---|---|
| Calcium gluconate (10%) | **0.5ml/kg over 10 mins X 1-2 doses**<br>given over 10 minutes with constant cardiac monitoring.<br>May repeat if no ECG changes in 5 mins | Onset: immediate<br>Duration: 30-60 mins | Myocardial protection.<br>Antagonizes membrane actions of $K^+$ | Max dose:10ml<br>Ca level unchanged<br>Monitor ECG.<br>Cease if bradycardia occurs |

### Drugs causing intracellular shift of K

| Agent | Dose/route | Onset, duration | Mode of action | Comments |
|---|---|---|---|---|
| $\beta_2$ – agonist ( salbutamol ) | 2.5 mg if <25 kg, 5 mg if >25 kg nebulised | O:10 –20min, D: 2-3hrs | Intracellular K+shift | |
| Glucose - Insulin | 1 gm/kg dextrose + 0.1Unit /kg insulin as infusion over 1 hr | O: 15 –30 mins<br>D: 4-6 hrs | Insulin causes intra –cellular $K^+$ shift | Monitor blood sugar |

Intracellular shift of K

| Agent | Dose/route | Onset, duration | Mode of action | Comments |
|---|---|---|---|---|
| Sodium bicarbonate | 1-2 mEq/kg over 10 mins | O: 20 mins<br>D: 2 hrs | Results in intra – cellular $K^+$ shift | controversial if no metabolic acidosis.<br>Ensure adequate ventilation.<br>Caution in pulmonary edema |

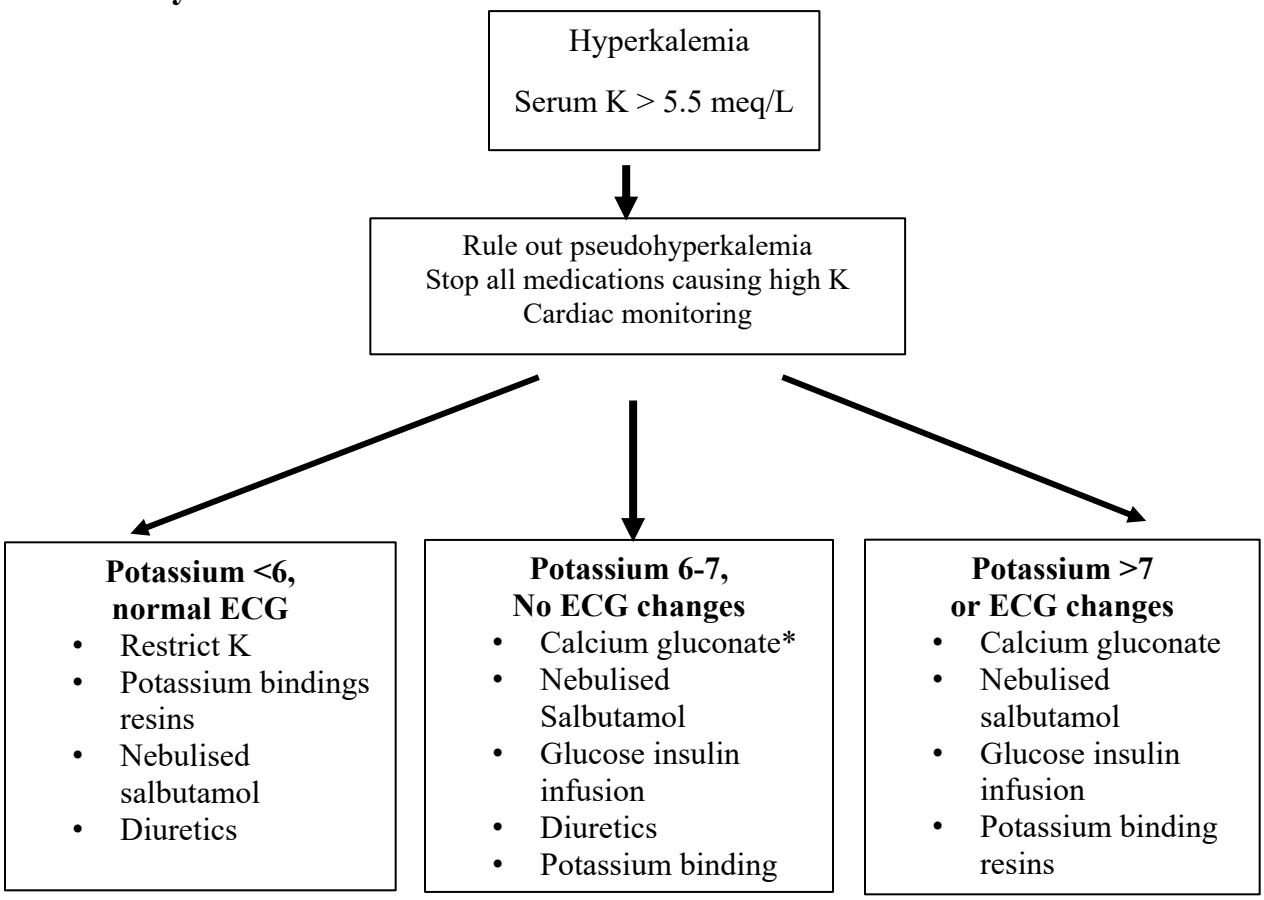
Reducing K absorption

| Agent | Dose/route | Onset, duration | Mode of action | Comments |
|---|---|---|---|---|
| Kayexalate sodium OR<br>Calcium resonium | 0.5 - 1gm/ kg PR or via nasogastric tube<br>q4-6hrs | 0: 30 –60mins<br>D: 4-6 hrs | Decreases body K+ by exchanging Na+ for K+ | Caution in postop patient (risk of intestinal necrosis)<br>Hypernatremia/ hypercalemia |

Removal of K from the body

| Agent | Dose/route | Onset, duration | Mode of action | Comments |
|---|---|---|---|---|
| Frusemide | 1-2mg / kg IV | 0: 30 –60mins<br>D: until diuresis lasts | Removes $K^+$ from the body | Saline better than diuretic in volume depleted states.<br>Diuretics may be ineffective in AKI |
| Dialysis (HD or PD ) | | | Removes $K^+$ from the body | HD more efficient than PD |

## Summary


Hyperkalemia
Serum K > 5.5 meq/L
Rule out pseudohyperkalemia
Stop all medications causing high K
Cardiac monitoring
Potassium <6, normal ECG
Restrict K
Potassium bindings resins
Nebulised salbutamol
Diuretics
Potassium 6-7, No ECG changes
Calcium gluconate*
Nebulised Salbutamol
Glucose insulin infusion
Diuretics
Potassium binding
Potassium >7 or ECG changes
Calcium gluconate
Nebulised salbutamol
Glucose insulin infusion
Potassium binding resins

Notes

# Sodium homeostasis

*(Please read this in conjunction with fluid homeostasis)*

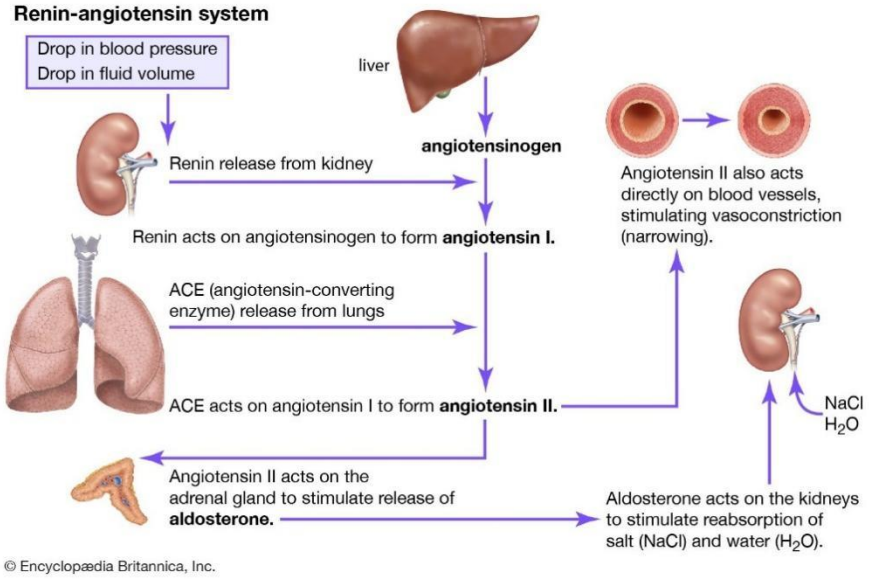
- Sodium is the dominant cation of the ECF.
- It is the principal determinant of extracellular osmolality, thus necessary for the maintenance of intravascular volume.
- A child's diet determines the amount of Na+ ingested
- Sodium excretion occurs in stool and sweat, but the kidney regulates Na+ balance and is the principal site of Na+ excretion. Stool Na+ loss in minimal, unless diarrhea is present.
- Serum Na+ levels are determined by water balance, not Na+ balance.

Maintenance of normal osmolality depends on control of water balance. Control of volume status depends on regulation of sodium balance. When present, volume depletion takes precedence over regulation of osmolality. ***Control of osmolality is subordinate to maintenance of an adequate intravascular volume***.

The kidney determines sodium balance because there is little homeostatic control of sodium intake. The kidney regulates Na+ balance by altering the percentage of filtered Na+. Normally, the kidney excretes <1% of the Na+ filtered at the glomerulus. Kidney has the capacity to adapt to large variations in sodium intake. When necessary, urinary sodium excretion can be reduced to virtually undetectable levels or increased dramatically.

The effective intravascular volume is the most important determinant of renal Na+ excretion.

Decreased intravascular volume activates **renin angiotensin aldosterone system** in the kidney.




Increase in effective circulatory volume causes right atrium distension stimulating the synthesis of **atrial natriuretic peptide (ANP)**. which is produced by the atria in response to atrial wall distention. ANP inhibits Na+ resorption in the collecting duct, causing an increase in urinary Na+ excretion.

# Approach to sodium disorders

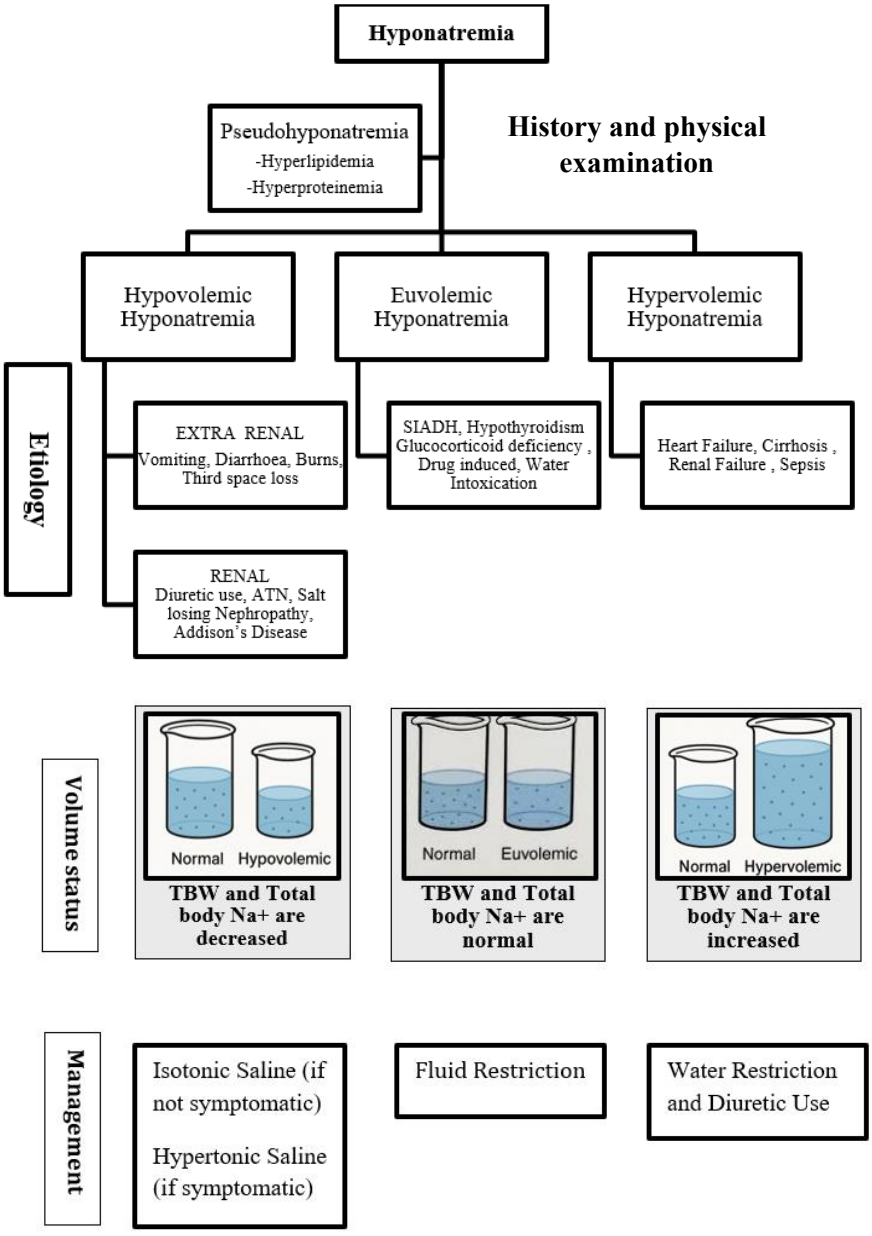
## Approach to Hyponatremia

Definition: serum sodium level <135 mEq/L

Both total body sodium and TBW determine the serum sodium concentration.

Hyponatremia exists when the ratio of water to Na+ is increased.

Clinical manifestations of hyponatremia are mostly the result of the low plasma osmolality

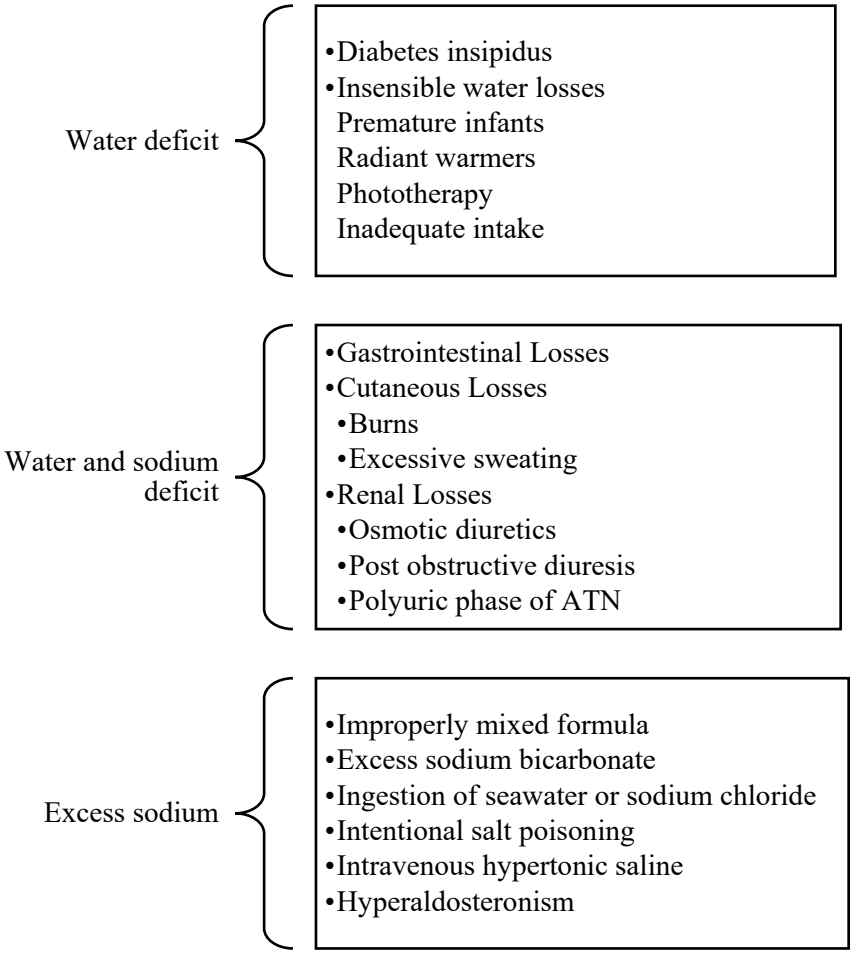


# Approach to hypernatremia

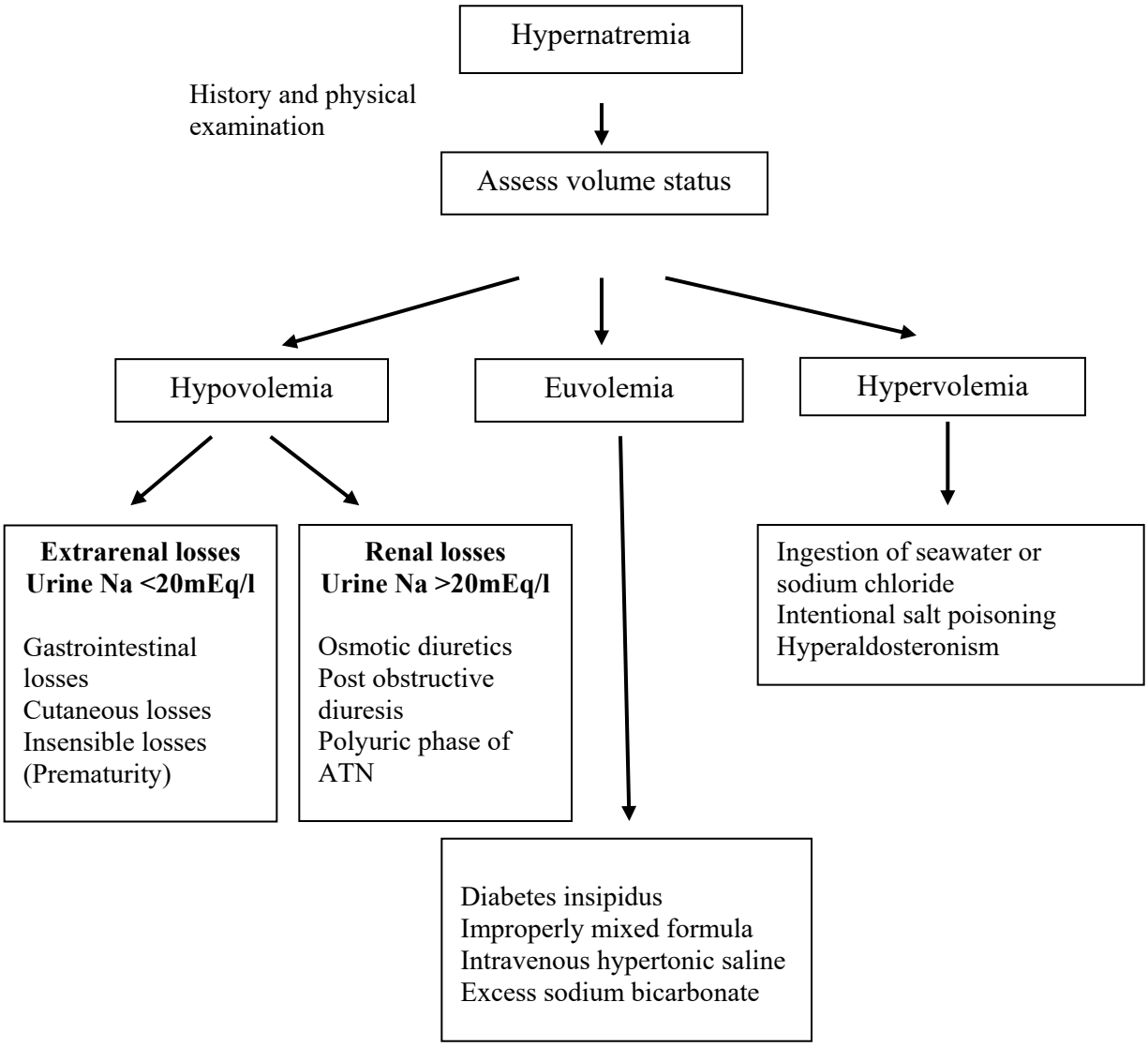
Definition: Serum sodium levels >145 mEq/l

It is due to the relative deficit of water in relation to serum sodium.

Etiology



**Approach**


Hypernatremia
History and physical examination
Assess volume status
Hypovolemia
Euvolemia
Hypervolemia
Extrarenal losses
Urine Na <20mEq/l
Gastrointestinal losses
Cutaneous losses
Insensible losses (Prematurity)
Renal losses
Urine Na >20mEq/l
Osmotic diuretics
Post obstructive diuresis
Polyuric phase of ATN
Ingestion of seawater or sodium chloride
Intentional salt poisoning
Hyperaldosteronism
Diabetes insipidus
Improperly mixed formula
Intravenous hypertonic saline
Excess sodium bicarbonate

# Case 7

| Case details | | Case Analysis & Questions |
|---|---|---|
| B, 2 year old girl<br><br>Weight: 15 kg, Height: 89 cm<br><br>Previously healthy, presented with a 4-day history of facial puffiness and progressive lower limb swelling. Urine output was reduced over the last 3 days.<br><br>Pain abdomen and loose stools for last 2 days. One day prior to admission, she had 2 episodes of vomiting.<br><br>There is no history of seizures or altered consciousness.<br><br>On Examination child is irritable, has periorbital edema<br><br>**Vitals:** HR: 120/min, RR: 28/min, BP: 112/65 mmHg ($90^{th}$-$95^{th}$ centile), Temp: 36.8°C<br><br>Abdomen: Distended with rebound tenderness, shifting dullness present, no organomegaly<br><br>CNS: irritable but comforted by mother, no focal neurological signs<br><br>CVS: Normal heart sounds<br><br>RS: Normal breath sounds | | What is the likely diagnosis?<br><br>Enlist the specific issues to be addressed<br><br>How will you approach hyponatremia in index child? |
| **Investigations** | | What is the cause of hyponatremia? |
| Hb | 9.8g/dl | |
| TLC | 20,200/ mcL | |
| Plt | 6.2 lakh/ mcL | |
| Na+/ K+ /Cl- | 125/ 4 /90 meq/L | |
| Creat | 0.3 mg/dL | |
| CRP | 42 mg/dL<br>(<0.6 normal) | |
| Urine routine | Protein 4+<br>RBC/ ketones nil | |
| Urine protein/creat | 10 | |

# Case 8

| Case details | | Case Analysis & Questions | |
|---|---|---|---|
| N, 8 year old boy<br>Weight: 25 kg, height: 125 cm<br><br>Previously healthy child presented with:<br>Fever for 5 days, no chills or rigors<br>Generalized headache for 3 days<br>Multiple episodes of non-bilious vomiting for past 2 days<br>Mother noticed increasing drowsiness, irritability, and confusion for 1 day<br>GTCS 1 hour prior to presentation<br><br>General Examination:<br>Drowsy, responds to painful stimuli<br>Temperature: 101.2°F<br>PR: 90/min, RR: 22/min<br>BP: 110/70 mmHg<br>SpO2: 98% on room air<br><br>No signs of dehydration or peripheral edema.<br><br>CNS : GCS: 11 (E3V4 M4)<br>Pupils 3 mm equal and reactive to light<br>Nuchal rigidity<br>No focal neurological deficits<br>CVS: Normal heart sounds, no murmur<br>Respiratory: normal breath sounds<br>Abdomen: soft, non-tender | | What is the likely diagnosis? | |
| | | What is the next investigation? | |
| | | **Further investigation** | |
| | | Serum Osmolarity | 260 mOsm/kg |
| | | Urine Sodium | 60 mmol/L |
| | | Urine Osmolarity | 600 mOsm/kg |
| | | Serum Glucose | 90 mg/dL |
| | | What is the likely cause of hyponatremia in this child? | |
| **Investigations** | | | |
| Hb | 9.4 g/dl | | |
| TLC | 27,324 | | |
| Plt | 2.3 lakh | | |
| Na+/ K+ /Cl- | 120/ 3.8/ 99 meq/L | How do you manage severe symptomatic hyponatremia? | |
| Creat | 0.5 | | |
| CRP | 10 | | |
| BUN | 3.5 | | |
| Uric Acid | 3 | | |
| CT Brain | Mild cerebral edema, no intracranial bleed | | |

**SIADH (Syndrome of Inappropriate Anti-Diuretic Hormone secretion)**

SIADH is a diagnosis of exclusion.

In SIADH, the secretion of ADH is not inhibited by either low serum osmolality or expanded intravascular volume leading to retention of free water. This results in dilution of the serum Na+ and hyponatremia.

***Diagnostic criteria:***

| | |
|---|---|
| Serum osmolality <280 mOsm/kg and serum sodium <135 mEq/L<br>Clinical Euvolemia<br>Urine osmolality >100 mOsm/kg (usually > plasma<br>Urine sodium >30 mEq/L with normal dietary Na intake<br>Normal thyroid, adrenal and renal functions<br>Exclude use of diuretic within last 1 week<br>No hypokalemia and acid base disorders | *Supporting evidence*<br>Serum uric acid level < 4mg/dl<br>Blood urea nitrogen <10mg/dl<br>FeNa <1%, Fe Urea > 55%<br>Failure to improve or worsening of hyponatremia with 3% NS<br>Improvement of hyponatremia with fluid restriction |

## Management of severe symptomatic hyponatremia

- 4-6ml/kg of 3%NS over 1-2 hours rapidly increases serum Na, thus increasing serum osmolality and reduction of cerebral edema (Each ml.kg of 3% saline increases serum sodium by 1mEq/L)
- Further increase till 135 is done slowly over next 24 hours using the formula
  Na deficit = 0.6 x weight in kg x (Desired Na – Current Na)
- Rapid correction can precipitate cerebral edema or pontine myelinolysis, so increase in Na should not be >10-12 mEq/day or >18 mEq/L/48 hr.

# Case 9

| Case details | | Case Analysis & Questions |
|---|---|---|
| D, 1.5-year-old girl<br><br>Preillness weight 10kg<br><br>Presented with acute watery diarrhoea - 10-12 episodes per day for 2 days.<br><br>Was advised zinc and ORS on OPD basis. Mother has been mixing ORS with a glass of water everytime she is giving<br><br>Next 24 hours, child continues to have loose stools, became drowsy. The last void urine was four hours back.<br><br>On examination- child was irritable<br><br>Weight at admission: 9kg<br><br>HR -144/min, pulse volume normal; RR- 38 /min, BP 80/60 mm Hg<br><br>Irritable child, oral mucosa is dry with absent tear film, skin pinch normal, with doughy consistency | | Enlist the issues and possible diagnosis<br><br>Describe the risk factors<br><br>How would you calculate the deficit, maintenance fluids and replacement?<br><br>What is the choice of fluid for each and duration of correction? |
| Investigations | | |
| Hb | 10.8g/dl | |
| TLC | 10,400 | |
| Plt | 4.2 lakh | |
| Na+/ K+ /Cl- | 165/4.5/128 meq/L | What are the aspects to monitor during treatment? |
| Urea/Creat | 65/ 0.8 | |
| GRBS | 90 | |
| VBG | pH- 7.25, Hco3- 12meq/l, Pco2- 25 | |

**Management of hypernatremic dehydration**

1. Assess hemodynamic status
2. Restore intravascular volume: If the child is in shock, give 20ml/kg of Normal saline bolus (repeat bolus until tachycardia, hypotension and poor perfusion is restored
3. Serum Na has to be corrected slowly to prevent osmotic disequilibrium
4. Determine time for correction based on initial serum sodium concentration

| Intial S Na | Duration of correction |
|---|---|
| 145 – 157 mEq/L | 24 hr |
| 158-170 mEq/L | 48hr |
| 171-183 mEq/L | 72 hr |
| 184-196 mEq/L | 84 hr |

5. Calculate Total fluid deficit (TFD) = Free water deficit (FWD) + Solute fluid deficit (SFD)

6. Total fluid deficit is calculated using the degree of dehydration

| Dehydration % | TFD |
|---|---|
| Mild 5% | 50ml/kg |
| Moderate 7.5% | 75ml/kg |
| Severe 10% | 100ml/kg |

7. Free water deficit is calculated using the formula
    a. FWD = 4 mL/kg x pre-illness weight (kg) x (Serum Na - Desired Na)
    b. The difference between serum Na+ and desired Na+ should be no greater than 10 mEq/24 hr to avoid rapid correction of Na
    c. If S Na > 170mEq/L, take the correction factor as 3ml/kg.
    d. Rate of replacement of FWD will be dependent on the initial S Na (Table above).

8. Solute fluid deficit is TFD minus FWD, replacement has to be given over 24 hours.

9. Calculate Solute deficit (SD) using the formula: SD = 0.6 x SFD (l) x 145 (normal Na value in ECF)
10. Calculate maintenance fluid using Holiday Segar formula
11. Calculate maintenance Na = 3mEq/kg/day
12. Total fluid requirement (ml) = maintenance fluids + FWD + SFD
13. Total Na requirement = Solute deficit + maintenance Na
14. Choose a fluid based on total fluid and Na requirement per litre of fluid
15. Replace ongoing losses (stool losses/ polyuria) as they occur

To minimize the risk of cerebral oedema during the correction of hypernatremic dehydration, the serum sodium concentration should not decrease by >10 mEq/L every 24 hr.

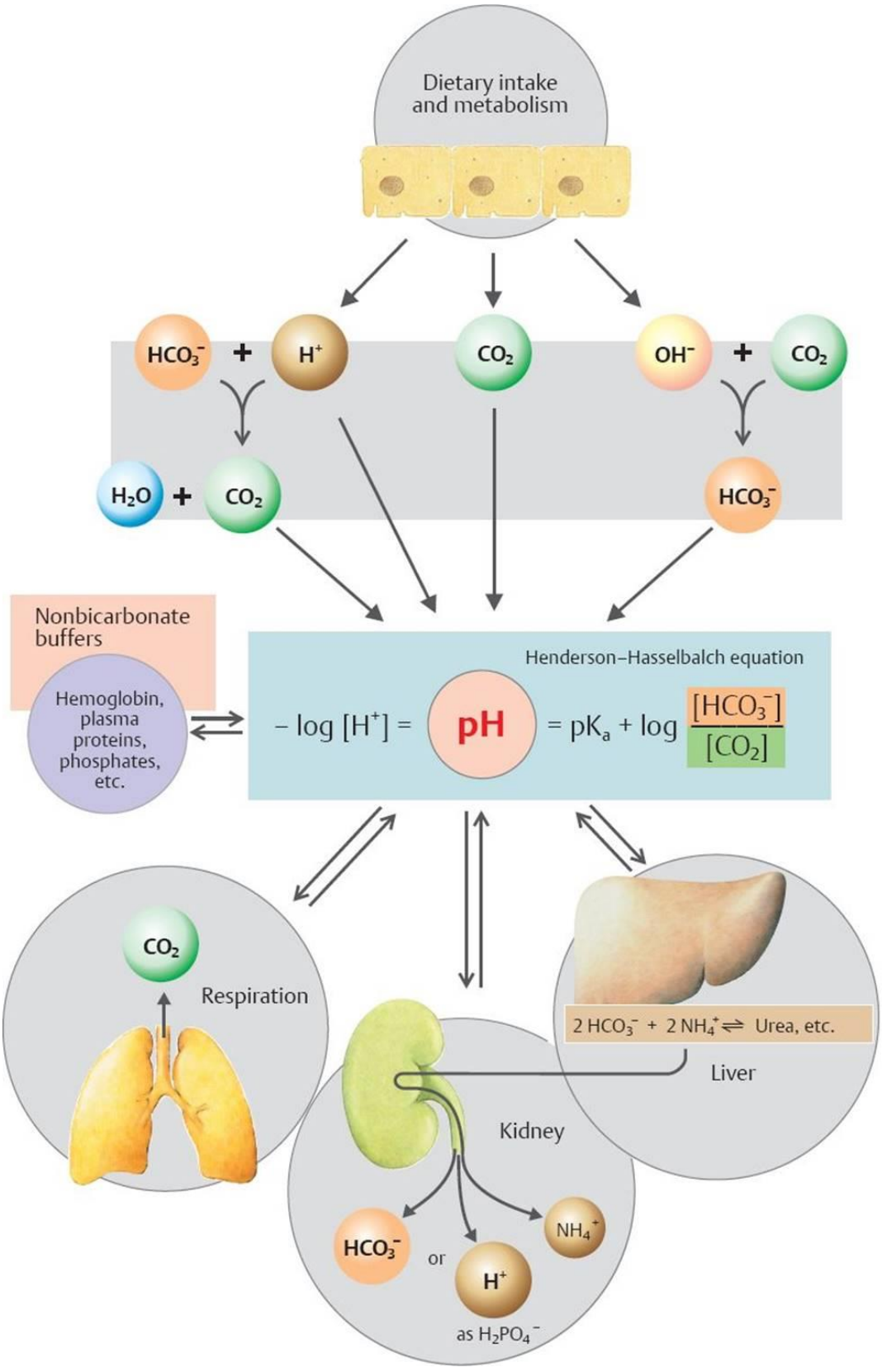
Rough rule of thumb: Rate of fluid is 1.5times maintenance and type of fluid 1/2 NS

Monitor serum sodium and clinical status every 4-6$^{th}$ hourly and adjust the fluid.

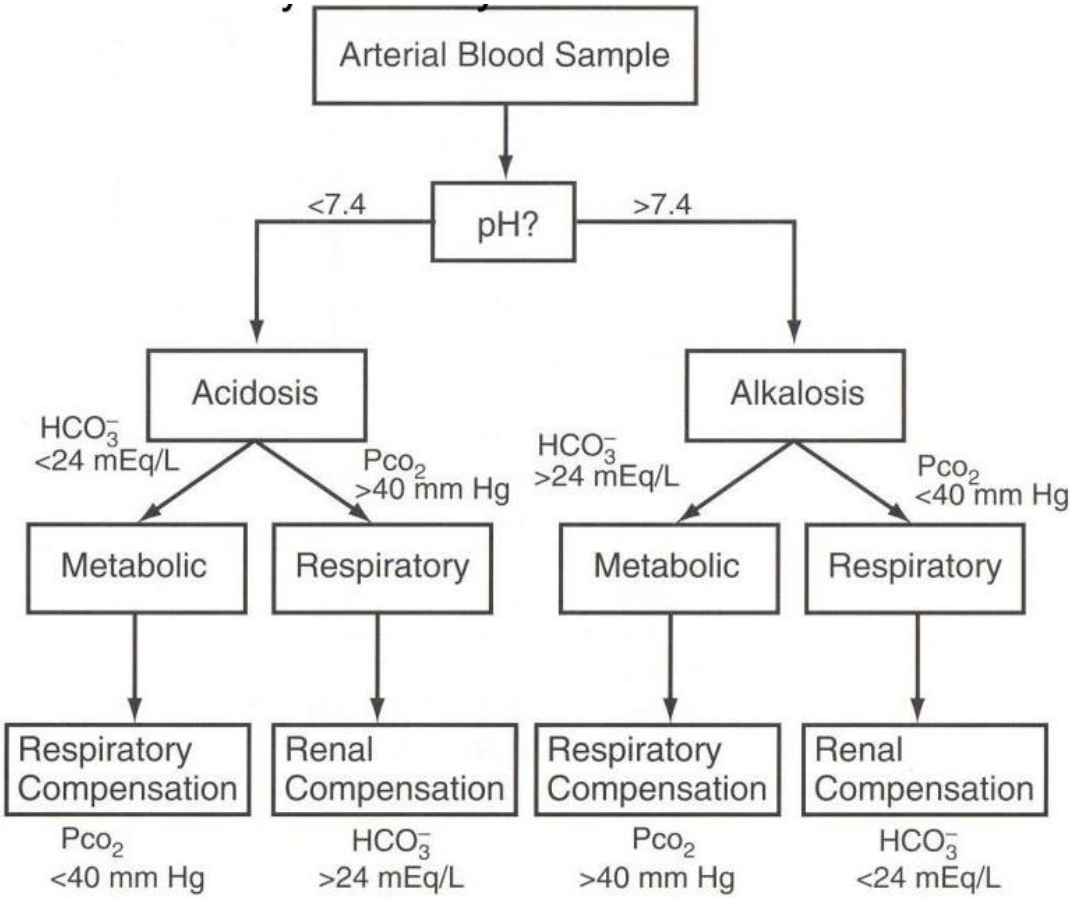
- Signs of volume depletion: administer normal saline (20 mL/kg)
- Sodium decreases too rapidly; either:
    - Increase sodium concentration of IV fluid
    - Decrease rate of IV fluid
- Sodium decreases too slowly; either:
    - Decrease sodium concentration of IV fluid
    - Increase rate of IV fluid

## Notes

# Acid -base physiology



# Approach to acid-base disorders



## Steps of blood gas interpretation

1. Check internal accuracy
2. Look at pH to see acidosis or alkalosis
3. Identify primary disorder: metabolic /respiratory
4. Measure compensation
5. Calculate anion gap
6. Look for additional disorder: simple vs mixed
7. Calculate delta gap

**Step 1:** Assess the internal consistency of the values using the Henderseon-Hasselbach equation:

$$[H^+] = \frac{24(PaCO_2)}{[HCO_3^-]}$$

If the pH and the [H+] are inconsistent, the ABG is probably not valid.

| **pH** | **Approximate [H+] (nmol/L)** |
|---|---|
| 7.00 | 100 |
| 7.05 | 89 |
| 7.10 | 79 |
| 7.15 | 71 |
| 7.20 | 63 |
| 7.25 | 56 |
| 7.30 | 50 |
| 7.35 | 45 |
| 7.40 | 40 |
| 7.45 | 35 |
| 7.50 | 32 |
| 7.55 | 28 |
| 7.60 | 25 |
| 7.65 | 22 |

**Step 2:** Is there alkalemia or acidemia present?

pH < 7.35 acidemia

pH > 7.45 alkalemia

This is usually the primary disorder

**Remember:** an acidosis or alkalosis may be present even if the pH is in the normal range (7.35 – 7.45)

Check the PaCO2, HCO3- and anion gap

**Step 3:** Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the PaCO2? In primary respiratory disorders, the pH and PaCO2 change in opposite directions; in metabolic disorders the pH and PaCO2 change in the same direction.

| | | | |
|---|---|---|---|
| Acidosis | Respiratory | pH ↓ | $PaCO_2$ ↑ |
| Acidosis | Metabolic | pH ↓ | $PaCO_2$ ↓ |
| Alkalosis | Respiratory | pH ↑ | $PaCO_2$ ↓ |
| Alkalosis | Metabolic | pH ↑ | $PaCO_2$ ↑ |

**Step 4:** Is there appropriate compensation for the primary disturbance? Usually, compensation does not return the pH to normal (7.35 – 7.45)

| **Disorder** | **Expected compensation** |
|---|---|
| Metabolic acidosis | PaCO2 = (1.5 x [HCO3-]) + 8 ± 2 |
| Metabolic alkalosis | Pco2 increases by 7 mm Hg for each 10 mEq/L increase in serum [$HCO_3^-$]<br>Increase in PaCO2 = 40 + 0.7 (ΔHCO3-) |
| Acute respiratory acidosis | [HCO3−] increases by 1 for each 10 mmHg increase in Pco2<br>Increase in [HCO3-] = Δ PaCO2/10 ± 3 |
| Chronic respiratory acidosis<br>(3-5 days) | HCO3 −] increases by 3.5 for each 10 mm Hg increase in Pco2<br>Increase in [HCO3-]= 3.5(Δ PaCO2/10) |
| Acute respiratory alkalosis | [HCO3−] falls by 2 for each 10 mm Hg<br>decrease in Pco2<br>Decrease in [HCO3-]= 2 (Δ PaCO2/10) |
| Chronic respiratory alkalosis | [HCO3−] falls by 4 for each 10 mm Hg<br>decrease in Pco2<br>Decrease in [HCO3-] = 4 (Δ PaCO2/10)<br>can be upto 7(Δ PaCO2/10) |

**Step 5:** Calculate the anion gap (if a metabolic acidosis exists):

$$AG = [Na+]-( [Cl-] + [HCO3-])-12 \pm 2$$

- A normal anion gap is approximately 12 meq/L.
- In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 gm/dL would be approximately 7 meq/L.)
- If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations.

    - Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure
    - Toxic ingestion is suspected

- Osmolal gap = measured Osm – (2[Na+] - glucose/18 – BUN/2.8

    - The Osmolal gap should be < 10

**Step 6:** If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in [HCO3-].

Assess the ratio of the change in the anion gap (Δ AG) to the change in [HCO3-] (Δ[HCO3-]): ΔAG/Δ[HCO3-]

*This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present. (Pure high anion gap metabolic acidosis)*

If this ratio falls outside of this range, then another metabolic disorder is present:

- If ΔAG/Δ[HCO3-] < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present.

- If ΔAG/Δ[HCO3-] > 2.0, then a concurrent metabolic alkalosis is likely to be present.

It is important to remember what the expected "normal" anion gap for your patient should be, by adjusting for hypoalbuminemia.

**Case 11**

1year old boy, 1st born to3rd degree consanguineously married couple has been brought to OPD with failure to thrive, vomiting and 2 episodes of seizures. There is h/o two previous sibling deaths. On examination, child has altered sensorium, Systemic examination shows hepatomegaly.

ABG shows pH - 7.1, pco2 - 23, hco3 - 8. Na - 140, cl - 106

Interpret the ABG.

How will you approach this acid base abnormality?

**Case 12**

A 5-year-old child, k/c/o Nephrotic Syndrome with relapse and anasarca. Parents are self-medicating the child with previously prescribed medications for last 5-6 days.

ABG shows Ph - 7.48, pco2 – 46, pO2 65, HCO3 - 34

Interpret the ABG.

What drugs can cause this acid base abnormality?

## Case 13

A 2-year-old boy is admitted with accidental consumption of sleeping pills. On examination, child is drowsy with hypopnea.

ABG shows Ph - 7.1, pCO2 - 80, HCO3 - 28, pO2 – 42

Interpret the ABG.

Calculate compensation for the acid base abnormality.

## Case 14

A 7-year-old boy is brought to ER with h/o fall from cycle followed by swelling and bleeding over right knee. Child has been excessively crying.

ABG shows Ph - 7.54, pco2 - 23, hco3 – 21

Interpret the ABG.

Calculate compensation for the acid base abnormality.

## Case 15

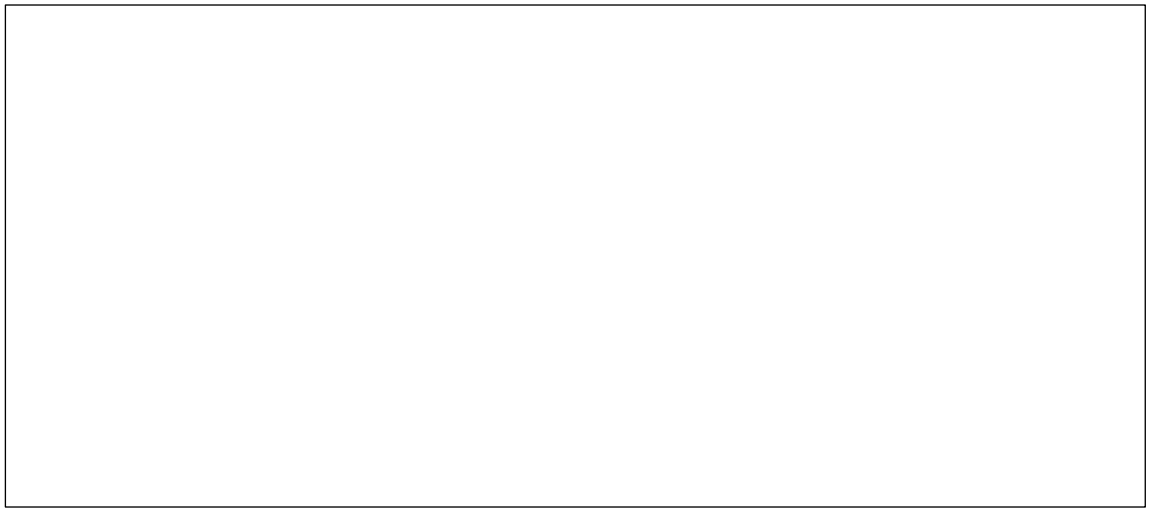
A2 year old boy, case of drowning with post cardiac arrest state, ROSC achieved.

What do you expect in blood gas?

pH 7.21 paO2 119 paCO2 45 HCO3 18

Na-145 K- 4.7 Cl- 107

Interpret the ABG.



# Notes

# Integrated cases

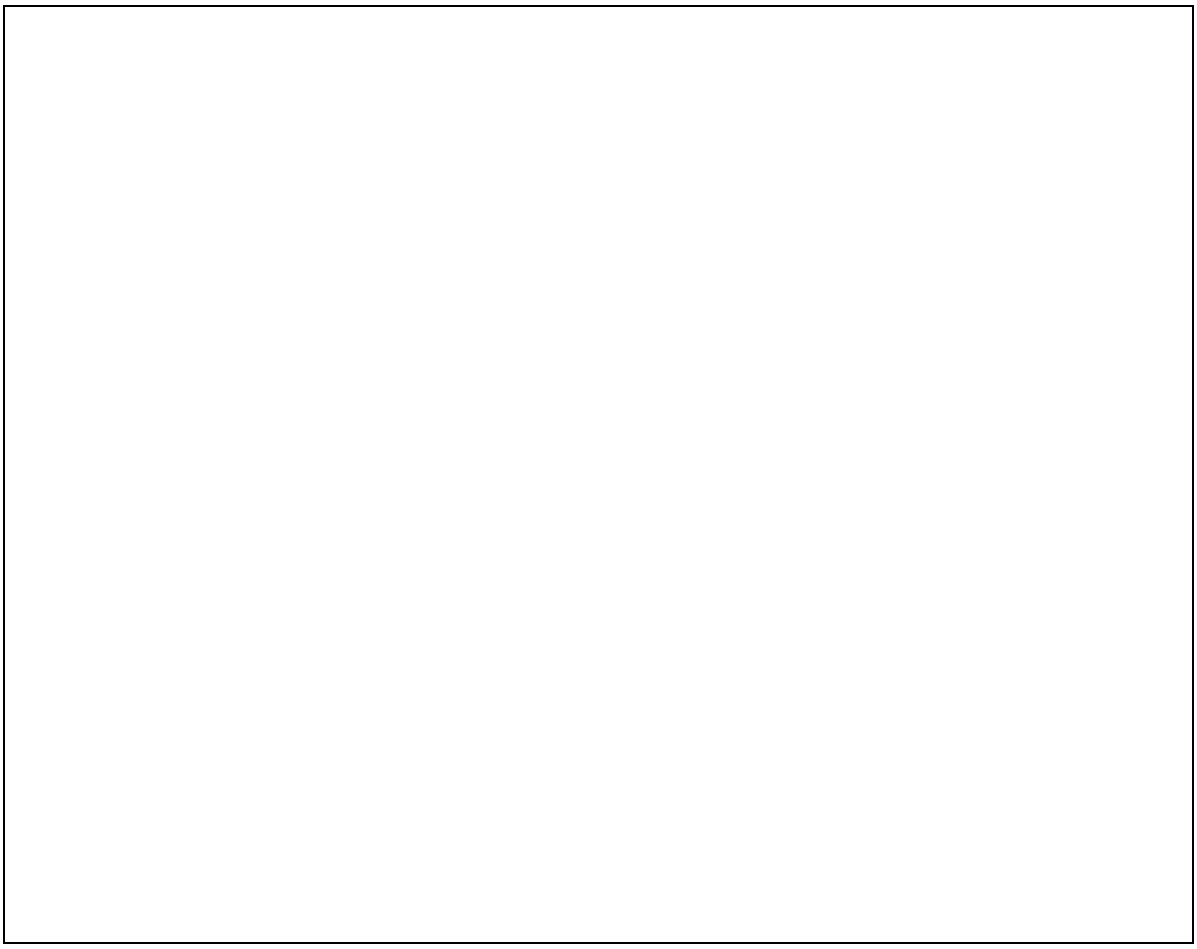
**Case 16**

A 13 year boy presents to ER at 7pm with history of pain abdomen and vomiting since 3 days. Mother has noticed lethargy and fast breathing since few hours. No loose stools / fever

On examination, he appears dehydrated, HR 154/min, afebrile, thready pulse, BP 112/72, acidotic breathing. Systemic examination normal.

**Questions:**

- What investigations you order?
- How do you correct his dehydration? (weight- 25kg)



**Key points for discussion:**

- Identification of specific etiology for dehydration
- Fluid therapy prescription in DKA

**Diagnostic criteria to define DKA are**

- Hyperglycemia with random blood glucose (BG) > 200 mg/dL
- Venous pH<7.3 OR bicarbonate <18 mmol/L
- Ketonemia (>3mmol/L) and/or ketonuria (>2+)

**Fluid therapy in DKA:**

The deficit can vary from 5-10% in various guidelines. Slow and even rehydration without major osmolar shifts is the key.

**i) Fluid calculation (volume of fluids)**

| | |
|---|---|
| Hypotensive shock | 20 ml/kg bolus of 0.9% saline over 20-30 min |
| Compensated shock | 10-20 ml/kg bolus of 0.9% saline over 20-30 min |
| Hemodynamically stable | **Deficit calculation (A)**: % dehydration assumed x body weight<br>Deficit is taken as a rough estimate of 6.5 to 8.5% (A) A higher estimate is chosen in severe DKA (10%).<br>**Maintenance fluid (B)**: From the Holiday-Segar equation which roughly estimates fluid requirement based on weight.<br>48-hours maintenance fluid is calculated (B)<br>bolus fluids given in first hour if any (C) subtracted.<br>**Hence, Total fluid requirement = (A + B) – C** |

- Total fluid requirement is calculated for 48 hours and given as hourly infusion
- Urinary losses need not be replaced routinely
- Calculation of fluid infusion rates for obese or large children should be similar to those of other children, use of ideal body weight or restricting fluids is not necessary
- If child develops features of cerebral edema, restrict fluids to 100%maintenance along with anti-raised ICP measures

**ii) Type of fluid to be used**

- Resuscitation fluid: Normal saline (0.9%) is preferred for initial resuscitation (bolus fluid)
- Deficit replacement fluid: 0.45% saline or a balanced salt solution (Ringer's lactate, Hartmann's solution or Plasmalyte)
- Deficit replacement should be with a solution that has a tonicity in the range of 0.45-0.9% saline, with added potassium chloride, potassium phosphate or potassium acetate
- Add 5% Dextrose to fluid (NS or N/2 saline) when blood glucose is < 250mg/dl.

**DKA severity assessment:**

| **Parameter** | **Mild** | **Moderate** | **Severe** |
|---|---|---|---|
| Arterial pH | 7.3- 7.2 | 7.2 - 7.1 | <7.1 |
| Serum bicarbonate(mmol/L) | 10-18 | 5-10 | <5 |
| Level of consciousness | Alert | Alert/drowsy | Stupor/coma |

**Some calculations used in DKA:**

| | **Formula** |
|---|---|
| Anion gap (AG) | AG= Na − (Cl+HCO3) |
| Corrected sodium | Measured Na + 2[(plasma glucose−100) /100] mg/dL |
| Effective osmolality | 2 × (plasma Na) + plasma glucose/ 18 |

## Case 17

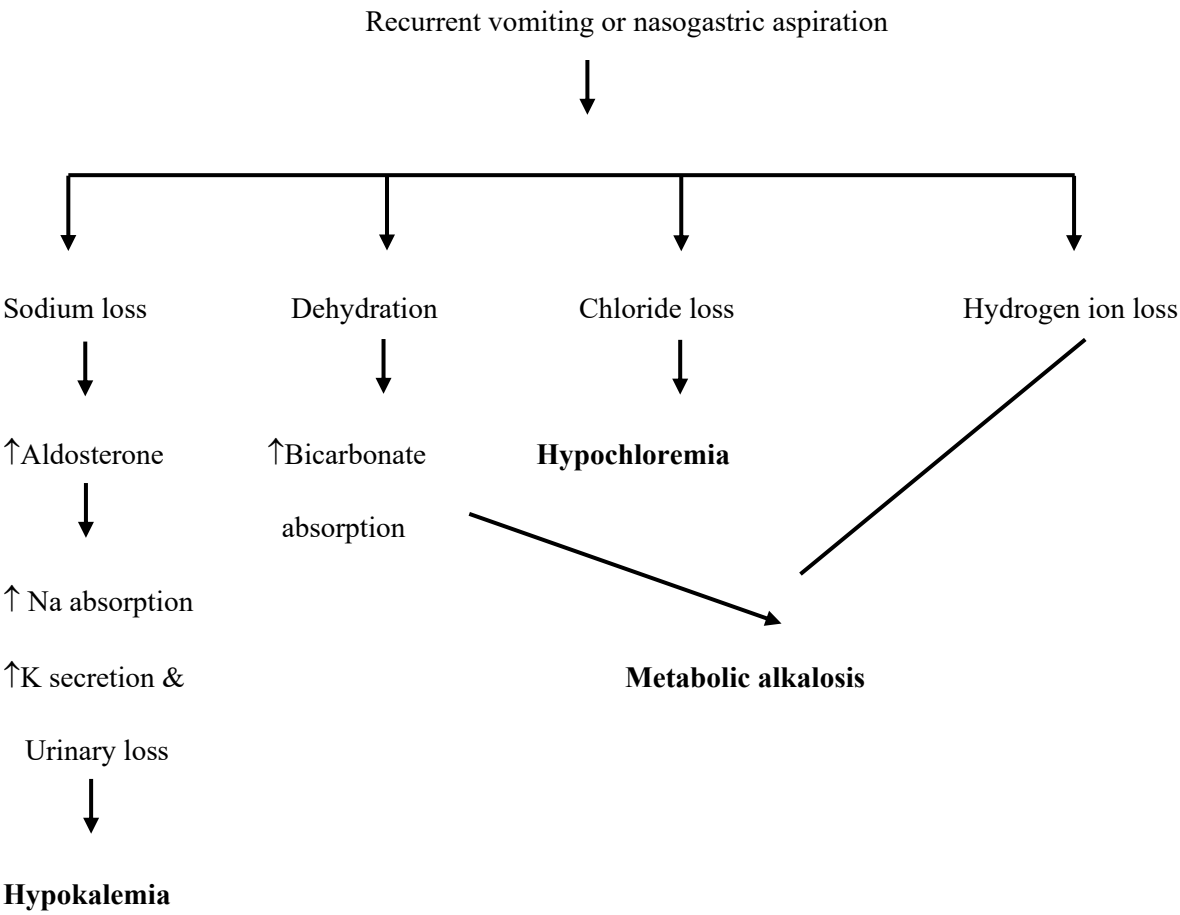
3 months infant is brought to ER with recurrent episodes of projectile vomiting since 1 week. Mother gives history of vomiting after each feed. On examination her weight was less than previous visit by 200g, she was dehydrated and irritable.

**Questions:**

- What fluid and electrolyte abnormalities you expect in the index case?
- Fluid of choice and rationale?
- How to assess the efficacy of fluid therapy in upper GI loss?

**Key points for discussion:**

A. Fluid and electrolyte abnormalities in upper GI loss




Blood gas: **Hypokalemic hyperchloremic metabolic alkalosis**

B. Fluid therapy in upper GI loss

- Volume by volume replacement of fluid loss should be made
- 0.9% normal saline with addition of 20-40mEq/L of potassium is used to restore the ongoing loss. Isolyte G is an alternative fluid

  Rationale: Infusion of isotonic saline corrects the hypovolemia and hypochloremia thereby decreasing renal bicarbonate absorption and increases the bicarbonate secretion respectively correcting the underlying metabolic alkalosis

- Dehydration to be assessed and corrected over 6hours in index case (3 hours in age>1year)

C. Assessment of the response to fluid therapy
    - Urinary pH is important tool in assessing the response. Acidic urine suggests need for more aggressive treatment and alkaline urine suggests response to therapy

**Case 18**

A 2 -year boy weighing 12kg accidently tipped a pan of hot oil over himself and sustains second and third degree burns to about 37% involving right upper limb, upper and lower chest, right lower limb sparing the genitalia. (41% of TBSA)

Questions:

- What is the optimum composition of resuscitating fluid?
- How much fluid to administer in initial 24 hours?
- When to consider colloids?

**Key points for discussion:**

**A. Fluid of choice in burns resuscitation**

- Crystalloids isotonic fluids are preferred for burn resuscitation. Hypotonic fluids are avoided as they can exacerbate edema
- Lactated Ringer solution is recommended is the preferred choice (avoids hyperchloremic metabolic acidosis)
- Dextrose may be added in infants

**B. Fluid calculation in burns**

Modified Parkland formula is commonly used in children to calculate the fluid requirement in first 24 hours

**(4ml/kg x %TBSA burn) + Maintenance fluids**

*Maintenance fluids calculated as per holiday segar formula

- Half of the resuscitation volume is given over first 8 hours and rest half in next 16 hours
- In children weighing >40kg, maintenance fluids are not included in the calculation

Ongoing changes in resuscitation fluid is determined by urine output and fluid status which is evaluated every 1-2 hours

Target urine output: 1ml/kg/hr

If urine output is below target, increase the fluid rate by 10-20% and monitor fluid status response

**C. Role of colloids**

- 5% albumin may be considered in patients >40% TBSA burns, low serum albumin levels, poor response to crystalloid resuscitation
- Non protein colloids such as Hetastarch and dextran are avoided