It is the most important problem in diarrhea &/or vomiting.

**TYPES OF DEHYDRATION**

- **According to the degree of dehydration:**
  1- Mild dehydration (wt loss is 3-5% in an infant): Such an infant is considered 5% dehydrated (water loss is 50 ml/ kg), normal or increased pulse rate, decreased urine output, thirsty, normal physical examination.
  2- Moderate dehydration (wt loss is 5–10% in an infant): tachycardia, little or no urine output, irritable/lethargic, sunken eyes and fontanel, decreased tears, dry mucous membranes, mild tenting of the skin, delayed capillary refill, cool and pale.
  3- Severe dehydration (wt loss is 10–15% in an infant): of body wt, this child is considered approximately 15% dehydrated. (150 ml/ kg), rapid and weak pulse, decreased blood pressure, no urine output, very sunken eyes and fontanel, no tears, parched mucous membranes, tenting of the skin, very delayed capillary refill, cold and mottled, renal shutdown, shock, coma, & death.

- In an older child or adult, the degree of dehydration is 3%, 6%, & 9% for mild, moderate, & severe dehydration, respectively.

- **According to the serum osmolarity & sodium level:**
  1- Isotonic dehydration: account about 70 % of cases, normal serum sodium & osmolarity, occur when the deficit of Na & water are equal.

  2- Hypertonic dehydration: S. Na is more than 150 mg/ dl. Occur when the loss of water is more, 20 % of cases. Plasma & interstitial hypernatraemia lead to shift of water from cells to the interstitial tissue causing intracellular dehydration & collapse of the brain cells. Children are often lethargic, but irritable when touched. Hypernatremia may cause fever, hypertonicity, and hyperreflexia; more severe neurologic symptoms may develop if cerebral bleeding occurs. The skin is doughy in nature & the tongue is parched,
shriveled & small. The condition is dangerous & may lead to permanent brain damage.

3- Hypotonic dehydration: about 10 %, Na level is below 130 mg/ dl with low serum osmolarity, occur when the sodium deficit is more than water deficit. Shift of water from interstitial tissue to the cells causing cellular distension, extracellular dehydration with more hypotension, severe dryness of mucous membrane, severe loss of skin turgor, sunken eyes, & more liability to shock & renal shutdown.

**TREATMENT**

In general:
Mild dehydration: at home by ORS (Dextrolyte)
Moderate dehydration: by ORS at hospital (ORS center), I. V. fluid in certain situation.
Severe dehydration: by I. V. fluid at hospital, & by ORS in certain situations.

**ORS (WHO)**
It contains 3.5 gm NaCl, 2.9 gm Na citrate, 1.5 KCl, & 20 gm glucose.
After dissolution in one liter of water: 90 mEq sodium, 80 mEq Cl, 20 mEq K, 30 mEq HCO3, & 111 mmol/l glucose.
It is given by spoon every 2-3 minutes, the baby fed in sitting position, & the rest of solution is discarded after 24 hr of dissolution. Vomiting may occur during the first 2 hr of administration of ORS, but it usually does not prevent successful oral rehydration if the ORS is given in small amounts at short intervals (a teaspoon every 1 to 2 min) & the emesis usually lessens over time.

**We have 2 methods for treatment of dehydration**

**CLASSICAL METHOD**

**Mild Dehydration:**
Give ORS 50 ml/ kg/ 4 hrs, then maintenance therapy should be started (100 mL of ORS/kg/24 hr until the diarrhea stops). Supplementary ORS is given to replace ongoing losses from diarrhea or emesis (an additional 10 mL/kg of ORS is given for each stool). Enhance breast feeding & fluid diet as rice water, fruit juice, and light tea.

**Moderate dehydration:**
Treated at hospital (ORS center) by 100 ml/ kg /4 hr ORS, then maintenance therapy should be started (100 ml of ORS/kg/24 hr until the diarrhea stops). Supplementary ORS is given to replace ongoing losses from diarrhea or emesis (an additional 10 mL/kg of ORS is given for each stool). Also enhance feeding & fluid diet.
For both the conditions above, if the patient develop peri-orbital edema or appear fully hydrated, the solution must be reduced.
I.V. fluid is indicated in:
1- Repeated persistent uncontrolled vomiting.
2- Severe gastric or abdominal distension.
3- Unable to drink due to extreme fatigue, stupours or coma.
4- Severe stomatitis & oral ulceration.
5- Primary glucose intolerance.

Severe dehydration:
Treated at hospital by I. V. fluid according to the rule
Total fluid requirement= deficit + maintenance + ongoing loss.

Deficit:
\[
= \text{wt (gm)} \times \% \text{ of body loss.}
\]
e. g. 10 % dehydration in 8 kg wt baby
\[
= 8 \times 1000 \times \frac{10}{100} = 800 \text{ ml.}
\]

Maintenance fluid:
The daily requirement of fluid which is physiologically lost through metabolism, urine, skin, lung, & stool.
According to the wt of the child:
1st
\[
10 \text{ kg} = 100 \text{ ml/kg/ day}, \text{ then add } 50 \text{ ml/kg/day for every kg above 10 kg}, \text{ then add } 20 \text{ ml/kg/day for every kg above 20 kg body wt.}
\]

Ongoing loss:
The fluid lost during the state of disease, including by diarrhea & vomiting,
- Replace stool & vomiting mL/mL every 1-6 hr

Solution: 500 ml (1/5 Dextrose Saline) + 15 mEq/L bicarbonate + 20 mEq/L KCl

Approach to a child with severe dehydration:
- Admit to the hospital, measure the weight of the child.
- Rapid assessment of the level of consciousness, pulse, blood pressure, breathing, urine output, & skin perfusion.
- Insert cannula & aspirate blood for urea, creatinine, serum electrolytes, pH, bicarbonate, in addition to PCV & WBC.
- Restore intravascular volume by 20 ml/ kg normal saline ( IRT: initial rehydration therapy) within 20 min or faster, repeat this dose till improvement of heart rate, BP., skin perfusion, & consciousness, then start 1/2 dextrose saline, wait the result of S. Na, add 20 KCL/l after voiding.
- if the s. Na 130- 150 mEq/ l, continue the same fluid, give one half of the total fluid during the1st 8 hr (subtract shouts of NS from this amount), give the rest within the next 16 hr.
- If the s. Na was more than 150: The treatment of hypernatremic dehydration may cause significant morbidity and mortality. There is generation of osmoles within the brain during the development of hypernatremia. These osmoles increase the osmolality within the cells of the brain, providing protection against brain cell shrinkage caused by movement of water out of cells into the hypertonic extracellular fluid. However, these osmoles dissipate slowly during correction of hypernatremia. With overly rapid lowering of the extracellular osmolality during correction of hypernatremia, there may be an osmotic gradient created that causes water movement from the extracellular space into the cells of the brain, producing cerebral edema. Symptoms of the resultant cerebral edema can range from seizures to brain herniation and death, so the most important point is slow rehydration & slow decrease of s. Na of no more than 12 mEq/ l/ day, this by give slow correction of the deficit within 36- 48 hr with monitoring of s. Na & the fluid is not run at a faster rate during the first 8 hr.
If the patient develop convulsion during correction, this is due to cerebral edema, acutely, increasing the serum concentration via an infusion of 5 ml/ Kg 3% sodium chloride can reverse the cerebral edema.
- if the s. Na less than 130 mEq/l, rapid increase of s. Na or rapid rehydration will precipitate central pontine damage, s. Na must not be increased more than 12 mEq/l/day. So continue on the same regimen but take care for this note. Patients with neurologic symptoms (e.g. seizures) from hyponatraemia need to receive an acute infusion of hypertonic (3%) saline (5 ml/ kg) to rapidly increase the serum sodium concentration.

* The formulation of a plan for correcting a child’s dehydration is only the beginning of management. All calculations in fluid therapy are only approximations. This is especially true with the assessment of percent dehydration. It is equally important to monitor the patient during treatment and to modify therapy based on the clinical situation.

**WHO METHOD**
More easy, more applicable, money saving, the patient classified into:
**Group A**: called Diarrhea with no dehydration.
**Group B**: called D. with some dehydration, mild - moderate dehydration.
**Group C**: called D. with severe dehydration.
Group A:
Treated by ORS:
- <2yr: 50- 100 ml/ motion.
- 2- 10 yr: 100 – 200 ml/ motion.
- > 10 yr: as much as he can drink.
Enhance breast feeding & fluid diet as rice water, fruit juice, light tea.

Group B:
Treat him in ORS center by 75 ml/ kg within 4 hr, then check for dehydration, if improved, shift to group A treatment, if still, repeat group B treatment, if deteriorate shift to group C treatment. Indications of I. V fluid are the same in classical method.

Group C:
Give the initial rehydration therapy 30 ml/kg Normal saline (for less than 1 yr within 1 hr, for more than 1 yr within 1/2 hr), then continue the therapy with Ringer lactate 70 ml/ kg (for child less than 1 yr within 5 hr, for more than 1 yr within 2.5 hr), then assess the state, if no improvement repeat the dose till improvement, if improved, shift to group B, & so on.

Gluten-Sensitive Enteropathy (Celiac Disease)
Clinical disorder results from gluten sensitivity of the intestine, it predominantly affects the proximal part of small intestine result in malabsorption. It is a permanent intolerance to gluten & withdrawal of gluten results in complete remission.

It doesn’t present until gluten products have been introduced into the diet, it may present in infancy, children & adult, but the most common period of presentation is between 6 mo and 2 yr of age, and

Etiology
The gluten present in wheat, barley, rye, & oat, the activity of gluten resides in the gliadin fraction which is composed of repetitive amino acid component (motifs) that sensitize the lymphocytes of the lamina propria & damage the intestinal mucosa.

Theories of etiology
A- Immunological theory:
- High titer of Abs.
- Complement activation.
- response to steroid.
- Prevalence with other immunologic disease.
B- Genetic theory:
- Autosomal dominant with incomplete penterance.
- 80% of cases have associated HLA type B8, DR 7 & DR 3.
- 98% of patients associated with histocompatibility class 2 alleles.
- Concordance in monozygotic twins approaching 100%. 2-5% of 1st-degree relatives have symptomatic gluten-sensitive enteropathy, and as many as 10% of first-degree relatives have asymptomatic damage to small bowel mucosa consistent with this disorder.

**Pathophysiology**
Long term dietary exposure to gluten cause sensitization of the lamina propria lymphocytes lead to inflammatory process & damage of the mucosa with characteristic microscopical changes showing villus atrophy, deep crypts & crypt hyperplasia, irregular vacuolated surface epithelium with increased numbers of lymphocytes in the epithelial layer. The lesion takes few wk – 2 yr of exposure to gluten to develop, & few month- 2 yr to change to normal when put on gluten free diet.

**Screening & incidence**
Screening test by serological markers show the incidence of celiac disease varies with population, e.g. 1/4000 in Denmark & 1/150 in Ireland. Screening tests indicate the asymptomatic cases form 5-7 times the symptomatic patients.

**Clinical features**
The mode of presentation is variable;
- Most patients present with diarrhea (constipation in some).
- Children can have failure to thrive or vomiting as the only manifestation, 10% of children referred to endocrinologists for growth retardation without an endocrine or overt gastrointestinal disorder have gluten sensitivity.
- Anorexia is common (in contrast to infants with cystic fibrosis) and may be the major cause of weight loss or lack of weight gain.
- Infants with gluten-sensitive enteropathy are often clingy, irritable, unhappy children who are difficult to comfort.
- Pallor and abdominal distention are common.
- Digital clubbing can occur.
- Other presentation like alopecia, bone pain, lymphocyte gastritis, rectal prolapse, asymptomatic hepatitis, dermatitis herpiformis, & neurological manifestation (like ataxia, developmental delay or epileptic convulsion).
- It is more commonly associated with other conditions like type 1 DM, autoimmune thyroiditis, rheumatoid arthritis, pernicious anemia, isolated IgA deficiency, Addison disease, & Down syndrome.
Investigation
- Hb, blood film (mostly show microcytic or dimorphic anemia), serum albumin, stool pH & reducing substance, D-xylose test.
- Serological markers:
  . Anti-gliadin IgA and IgG and anti-reticulin IgA antibody tests are no longer recommended tests due to lack of specificity.
  . The anti-endomysium IgA antibody and anti-tissue transglutaminase IgA antibody tests are highly sensitive and specific. The anti-endomysium IgA antibody test is relatively expensive; interpretation is operator dependent and prone to errors so that it has largely been replaced by anti-tissue transglutaminase IgA antibody tests, which are simpler to perform and have similar sensitivity and specificity.

- Intestinal biopsy is the gold standard for diagnosis.

**Diagnostic Regimen of Celiac Disease**

If + ve 1st biopsy, serum markers, & clinical, start GFD for 2 yrs, & then do 2nd biopsy:
(1) if normal mucosa then do gluten rechallenge:
   (a) if the symptoms relapsed ......then do another biopsy to confirm that the condition is celiac disease.
   (b) if no symptoms after rechallenge also do a third biopsy (after 2 yrs) to confirm the exclusion of the disease, if the third biopsy is normal, so it is not celiac disease.
(2) if abnormal mucosa, then the condition is either other pathology or due to mixed diet.

Patient above 2 yr no need for re-challenge & further biopsies if gluten free diet result in clinical, histological, & serological marker response.

Patient less than 2 year re-challenge must be done for response follow up because other diseases may cause flat intestinal mucosa in this age, e.g. Infections such as rotavirus enteritis & Giardia lamblia, undernutrition, cow's milk protein or soy protein intolerance.

**Treatment**
Long life gluten free diet, response occurs within a week with change in mood, increase appetite & wt & improvement of diarrhea, in older children, the response may be delayed for several weeks. No long-term complications from a gluten-free diet have been recognized.

Patient is more liable to develop malignancy of the intestine (lymphoma), and other forms of cancer, especially adenocarcinoma of the small intestine, of the pharynx, and of the esophagus. The long life strict GFD will reduce the risk of all these cancers.