# Maintenance and replacement fluid therapy in adults

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# Water balance

- – Water losses lead to an increase in serum sodium and osmolality, resulting in stimulation of thirst and increased release of ADH.
- In normal individuals, these changes will lead to increased water intake and reduced water excretion, which will restore normal water balance.
- Patients who are alert, have an intact thirst mechanism, and access to water will not become hypernatremic.

- On a normal diet, the minimum water intake is estimated at 500 mL/day (assuming there are no increased losses).

- Individuals who can concentrate their urine to 1200 mosmol/L who excrete 600 mosmol of solute per day will have a minimum urine output of 500 mL (600 mosmol ÷ 1200 mosmol/L).

- There are two other sources of water in addition to fluid ingestion:

- the water content of food (fruits and vegetables are almost 100 percent water by weight)
- the water generated by oxidation of carbohydrates.

- There are also other sources of water loss in addition to the urine output:
- Insensible losses and sweat.

- Normal adults are considered to have a minimal obligatory water intake or generation of approximately 1600 mL per day, composed of the following:
- ● Ingested water – 500 mL
- ● Water in food – 800 mL
- ● Water from oxidation – 300 mL

- The sources of obligatory water output in normal adults are composed of the following:
- ● Urine – 500 mL
- ● Skin – 500 mL
- ● Respiratory tract – 400 mL
- ● Stool – 200 mL

- the water of oxidation and much of the water lost from the lungs during respiration are linked .

- The metabolic production of $CO_2$ and water occur in a 1:1 proportion during the oxidation of carbohydrates and fatty acids , if the arterial $pCO_2$ is close to 40 mmHg, these two end-products are eliminate together in alveolar air in a 1:1 proportion.

- Water and $CO_2$ are eliminated in parallel because the partial pressures of water vapor (47 mmHg) and $CO_2$ (40 mmHg) are virtually equal in alveolar air, and because both $CO_2$ and water are nearly absent in inspired air.

- Thus, the water of oxidation and most of the water normally lost from the lungs during respiration can probably be removed from estimates of water balance .

- In most patients, only the small amount of water evaporation from the upper respiratory tract results in a negative water balance.

- This does not apply to patients who are hyperventilating (which increases alveolar water losses) or are on a ventilator and inspiring humidified air warmed to body temperature (which decreases alveolar water losses).

- Evaporation of water from the skin as sweat (which usually has a sodium concentration of 15 to 30 mEq/L and is therefore mostly water) is required to dissipate heat.

- When additional heat loss is needed, there is an increase in evaporative water losses from the skin. On the other hand, these losses diminish during fasting and inactivity.

# Fluid therapy:

- – There are two components to fluid therapy:
- **Maintenance therapy** replaces the ongoing losses of water and electrolytes under normal physiologic conditions via urine, sweat, respiration, and stool.
- **Replacement therapy** corrects any existing water and electrolyte deficits. These deficits can result from gastrointestinal, urinary, or skin losses, bleeding, and third-space sequestration.

# MAINTENANCE FLUID THERAPY:

- — In the presence of normal or near-normal kidney function, maintenance fluid therapy is usually undertaken when the patient is not expected to be able to eat or drink normally for a prolonged period of time (eg, perioperatively or on a ventilator).

- The goal of maintenance fluid therapy is to preserve water and electrolyte balance and to provide nutrition.
- Patients expected to have inadequate energy or fluid intake for more than one to two weeks should be considered for parenteral or enteral nutrition.

- The serum sodium concentration provides the best estimate of water balance in relation to solute.
- A normal serum sodium concentration implies that the patient is in water balance in relation to sodium but does not provide any information on volume status.
- Weighing the patient daily provides the best means for estimating net gain or loss of fluid since gastrointestinal, urine, and insensible losses in hospitalized patients are unpredictable.
- The patient should also be monitored for clinical signs of either volume excess (edema) or volume depletion (eg, reduced skin turgor, fall in blood pressure).

- **Water:**
- – Hospitalized patients who are afebrile, not eating, and physically inactive require less than one liter of electrolyte (sodium and potassium)-free water as maintenance fluid.
- Maintenance water requirements can be increased or decreased by a number of factors:

- Increased water intake is required if the patient has fever, sweating, burns, tachypnea, surgical drains, polyuria, or ongoing significant gastrointestinal losses.

- As an example, water requirements increase by 100 to 150 mL/day for each degree of body temperature elevation over 37°C.

- Decreased water intake is required in a number of clinical settings, including oliguric renal failure, the use of humidified air, edematous states, and hypothyroidism.

- In addition, sick patients may be unable to excrete excess water due to the presence of nonosmotic stimuli for the release of ADH (SIADH).

- the adequacy of water balance, as opposed to the adequacy of volume balance, is determined solely from the serum sodium concentration.
- A normal value means that the body has the proper amount of water for the amount of sodium but provides no information on volume balance.
- Because water deficits do not develop very rapidly in patients who do not have accelerated water losses, adjustment of the water prescription based upon frequent measurements of the serum sodium concentration is a logical strategy.

## Electrolytes:

- The majority of electrolyte losses (primarily sodium and potassium salts) are in the urine, with a lesser contribution from the skin and gastrointestinal tract.
- Electrolyte balance can be maintained over a wide range of intakes due to appropriate changes in urinary electrolyte excretion.
- If, for example, there is an increase in sodium intake, the ensuing increase in extracellular fluid volume will reduce the activity of RAS and increase the release of natriuretic peptides, resulting in an appropriate increase in sodium excretion.

- Since the maintenance requirement for electrolyte-free water intake is less than one liter per day, a reasonable approach is to begin with two liters per day of one half isotonic saline in 5 percent dextrose to which 20 mEq of potassium chloride is added per liter.

- This regimen provides 9 g of sodium chloride (3.4 g of sodium), which is similar to the sodium content of a hospital diet.

- Infusion of two liters of the dextrose-containing solution provides 400 kilocalories, enough to suppress catabolism.

- The original solution can be continued unless one of the following occurs:
- If the serum sodium starts to fall, a more concentrated solution should be given (eg, isotonic saline in 5 percent dextrose)
- If the serum sodium starts to rise due, for example, to increased insensible losses from high fever, a more dilute solution should be given (eg, one-quarter isotonic saline in 5 percent dextrose)
- ● If the serum potassium starts to fall, more potassium should be added and, should it rise above normal, potassium should be eliminated

# REPLACEMENT FLUID THERAPY:

- ***Volume deficit:***

- There is no formula that can be used to accurately estimate the total fluid deficit.
- If pre- and post-deficit body weight is known, then weight loss provides a reasonable estimate of fluid losses.
- If the degree of weight loss is not known, then the fluid deficit cannot be estimated.

- Clinical and laboratory parameters can be used to assess the possible presence of volume depletion, including:
- The blood pressure
- Jugular venous pressure
- Urine sodium concentration
- Urine output
- The hematocrit (if baseline values are available and bleeding has not occurred).

- These parameters should be followed to assess the efficacy of volume replacement.
- If, for example, the urine sodium concentration remains below 15 mEq/L , then the kidney is sensing persistent volume depletion and more fluid should be given.

- Use of the urine sodium concentration does **not** apply to edematous patients with heart failure or cirrhosis in whom the urine sodium concentration is a marker of effective circulating volume depletion but not of the need for more fluid or more salt.

# Rate of replacement:

- — The rate of correction of volume depletion depends upon its severity.

- With severe volume depletion or hypovolemic shock, at least 1 to 2 liters of isotonic fluids are generally given as rapidly as possible in an attempt to restore tissue perfusion.

- Fluid replacement is continued at a rapid rate until the clinical signs of hypovolemia improve (eg, low blood pressure, low urine output, and/or impaired mental status).

- In comparison, ***rapid fluid resuscitation is not necessary in patients with mild to moderate hypovolemia***.

- To avoid worsening of the volume deficit, the rate of fluid administration must be greater than the rate of continued fluid losses, which is equal to the urine output plus estimated insensible losses (usually 30 to 50 mL/hour) plus any other fluid losses (eg, gastrointestinal losses) that may be present.

- One regimen that we have used to induce positive fluid balance in such patients is the administration of fluid at a rate that is 50 to 100 mL/hour greater than estimated fluid losses.

# Choice of replacement fluid:

- – The composition of fluid that is given is largely dependent upon the type of fluid that has been lost and any concurrent electrolyte disorders .

- Most patients are treated with isotonic or one-half isotonic saline but the choice of therapy can be influenced by concurrent abnormalities in serum sodium or potassium or the presence of metabolic acidosis.

- As examples:
- Hypotonic solutions should be used in hypernatremia,
- Isotonic or hypertonic saline should be used in hyponatremia,
- Isotonic saline and/or blood should be used in patients with blood loss.

- Potassium or bicarbonate may need to be added in patients with hypokalemia or metabolic acidosis.

# Hypernatremia:

- In infants, water deficits resulting in hypernatremia should generally be corrected slowly since overly rapid correction of chronic hypernatremia can cause cerebral edema, and water deficits usually develop gradually.
- When it is known that the water deficit developed in less than 48 hours, hypernatremia can and should be corrected rapidly.
- Increased water intake is required if the patient has fever, sweating, burns, tachypnea, surgical drains, polyuria, or ongoing significant GI losses.

- Fluid therapy can be planned by calculating how much dilute fluid (eg, 5 percent dextrose in water) should be given to lower the sodium at the desired rate.

- Dextrose in water can be given alone in patients with diabetes insipidus who have lost only water, but will not correct all of the hypovolemia if there has also been salt and water loss due, for example, to concurrent diarrhea.

- Sodium and/or potassium can be added to the IV fluid as necessary to treat concurrent volume depletion and/or hypokalemia (due, for example, to diarrhea).

- However, the addition of sodium and/or potassium decreases the amount of free water that is being given.

- If, for example, one-quarter isotonic saline is infused, then only three-quarters of the solution is free water.
- In this setting, approximately 1333 mL of ¼ isotonic saline must be given to provide 1000 mL of free water.
- If potassium is also added to the IV fluid, then less free water is present and a further adjustment to the rate of infusion must be made.
- These adjustments are only estimates that are then guided by serial monitoring of the serum sodium.

# Hyponatremia:

- — As with hypernatremia, overly rapid correction of hyponatremia is potentially harmful if there has been time for adaptation to the electrolyte disturbance(greater than 48 h).

- The administration of isotonic saline in hyponatremic patients will initially tend to raise the serum sodium since it has a higher sodium concentration than the serum.

- If the cause of hyponatremia is a hypovolemic stimulus to antidiuretic hormone (ADH) secretion, then once the volume deficit is largely repaired, the stimulus to ADH secretion will be removed.

- This will result in the excretion of a maximally dilute urine and possible overly rapid correction of the hyponatremia that can lead to severe neurologic dysfunction.

- If the cause of hyponatremia is SIADH, the urine will remain concentrated and the sodium contained in the intravenous isotonic fluid will be excreted in the urine at a higher concentration than in the infused intravenous fluid.

- This "desalination"phenomenon will result in a net gain of electrolyte-free water that may cause the serum sodium to fall during the infusion of isotonic saline.

# Addition of potassium:

- — Concurrent potassium replacement is indicated in patients who have developed potassium depletion as typically manifested by hypokalemia.
- There are also settings in which potassium depletion is present but the serum potassium is normal or even increased.
- A classic example is diabetic ketoacidosis or nonketotic hyperglycemia in which both hyperosmolality and insulin deficiency promote potassium movement out of the cells, masking the presence of potassium depletion.

- Rarely, the serum potassium may be low in the absence of potassium depletion, as may be seen in patients with thyrotoxic or familial hypokalemic periodic paralysis; potassium replacement in such patients can lead to hyperkalemia .

- Potassium is as osmotically active as sodium. Thus, the addition of 40 mEq of potassium to one liter of one-half isotonic saline (containing 77mEq/L of sodium creates a solution that is essentially three-quarters isotonic saline and therefore contains less free water.

- This could be important in patients with an elevated plasma osmolality due to hypernatremia or uncontrolled diabetes mellitus.

- If potassium is added to isotonic saline or one-half isotonic saline, it limits the potential rate of infusion.

- In most cases, the desired rate of K replacemen is no greater than 10 mEq per hour; in patients with life-threatening hK, the rate can be increased to 20 mEq per hour, although ECG monitoring is required.

- Thus, if 40 mEq of potassium has been added to a liter of IV solution, the rate of infusion should generally be limited to 250 mL per hour, or 500 mL per hour with ECG monitoring if the patient has life-threatening hypokalemia.

# Addition of bicarbonate:

- — A more complex solution may be required in patients with metabolic acidosis.

- In this setting, sodium bicarbonate may be added, particularly if the acidemia is severe (arterial pH less than 7.15 to 7.2 or less than 7 in diabetic ketoacidosis) or bicarbonate losses persist (as with severe diarrhea).

- Suppose a patient with diarrhea presents with mild hypernatremia, mild hypokalemia, and a serum bicarbonate concentration of 10 mEq/L.

- An appropriate dilute replacement fluid in this setting might be one-quarter isotonic saline in 5 percent dextrose (containing 38.5 mEq of sodium chloride) to which 20 mEq of potassium chloride and 25 mEq (one-half ampule) of sodium bicarbonate have been added.

- The total cation concentration is 83.5 mEq/L, roughly equivalent to one-half isotonic saline.

- It is important to add potassium to the intravenous fluid in hypokalemic patients since both the administration of bicarbonate and increased insulin secretion induced by dextrose will tend to drive potassium into the cells, which will further reduce the serum potassium concentration.

- An alternative regimen that can be used in patients with metabolic acidosis without hk is

- the addition of three ampules of $NaHCO_3$ (each containing 50 mEq of sodium and 50 mL of water) to one liter of DW5%, which results in a nearly isotonic solution with a Na concentration of approximately 130 mEq/L.

- In contrast, addition of the same three ampules of $NaHCO_3$ to one-liter of HS (containing 77 mEq/L of sodium) results in a hypertonic solution with a Na concentration of 197 mEq/L, which will tend to raise the serum Na concentration.

- Such a solution should not be used unless the patient is hNa (such as in a patient with short bowel syndrome who has mild hyponatremia and metabolic acidosis).

# SALINE ALONE OR WITH DEXTROSE:

- — There is little evidence that a dextrose-saline solution has any benefit or harm compared to a saline solution alone for most patients.
- However, there are some exceptions to this general rule:
- Dextrose-containing solutions **should be used** in patients with hypoglycemia or alcohol or fasting ketoacidosis and should be given with insulin in patients with hyperkalemia and no hyperglycemia since insulin-mediated entry of potassium into cells will lower the serum potassium concentration.

- Dextrose-containing solutions **should not be used** in patients with uncontrolled diabetes mellitus or hypokalemia.
- With respect to hypokalemia, the administration of dextrose stimulates the release of insulin, which promotes potassium entry into cells with possible worsening of the hypokalemia.

# Dextrose-induced hyperglycemia:

- – The administration of large volumes of dextrose-containing solutions to critically ill patients can promote the development of hyperglycemia ,

- which is in part mediated by both administering dextrose at a rate that exceeds the maximum rate of metabolism and by the counterregulatory hormone response (eg,increased epinephrine secretion) and perhaps cytokine responses .

- In studies of patients without diabetes who are treated with TPN or in normal individuals given glucose infusions, hyperglycemia is primarily seen when glucose is given at a rate exceeding 4 to 5 mg/kg per minute,

- A rate that exceeds the body's ability to metabolize glucose, even with maximum doses of insulin .

- In a patient weighing 70 kg, a glucose dose of 4 to 5 mg/kg per minute translates into an infusion rate greater than 5.6 to 7 mL/min (336 to 420 mL/hour) with 5 percent dextrose solutions and

- Greater than 0.8 to 1 mL/min (48 to 60 mL/hour) with the 25 to 35 percent glucose solutions that may be used in TPN.

| Solution | Solute | Concentrations, g/100 mL | Ionic concentration, meq/L $[Na^+]$ | $[K^+]$ | $[Ca^{2+}]$ | $[Cl^-]$ | $[HCO_3^-]$ | Total mosmol/ |
|---|---|---|---|---|---|---|---|---|
| Dextrose in water | | | | | | | | |
| 5.0% | Glucose | 5.0 | — | — | — | — | — | 278 |
| 10% | Glucose | 10.0 | — | — | — | — | — | 556 |
| Saline | | | | | | | | |
| Hypotonic (0.45%, half-normal) | NaCl | 0.45 | 77 | — | — | 77 | — | 154 |
| Isotonic (0.9%, normal) | NaCl | 0.90 | 154 | — | — | 154 | — | 308 |
| Hypertonic | NaCl | 3.0 | 513 | — | — | 513 | — | 1026 |
| | | 5.0 | 855 | — | — | 855 | — | 1710 |
| Dextrose in saline | | | | | | | | |
| 5% in 0.225% | Glucose | 5.0 | — | — | — | — | — | — |
| | NaCl | 0.225 | 38.5 | — | — | 38.5 | — | 355 |
| 5% in 0.45% | Glucose | 5.0 | — | — | — | — | — | — |
| | NaCl | 0.45 | 77 | — | — | 77 | | 432 |
| 5% in 0.9% | Glucose | 5.0 | — | — | — | — | — | — |
| | NaCl | 0.90 | 154 | — | — | 154 | — | 586 |
| Alkalinizing solutions | | | | | | | | |
| Hypertonic sodium bicarbonate (0.6*M*) | $NaHCO_3$ | 5.0 | 595 | — | — | — | 595 | 1190 |
| Hypertonic sodium bicarbonate (0.9*M*)[b] | $NaHCO_3$ | 7.5 | 893 | — | — | — | 893 | 1786 |
| Polyionic solutions | | | | | | | | |
| Ringer's | NaCl | 0.86 | — | — | — | — | — | — |
| | KCl | 0.03 | 147 | 4 | 5 | 156 | — | 309 |
| | $CaCl_2$ | 0.03 | — | — | — | — | — | — |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Lactated Ringer's | NaCl | 0.60 | — | — | — | — | — | — |
| | KCl | 0.03 | — | — | — | — | — | — |
| | $CaCl_2$ | 0.02 | 130 | 4 | 3 | 109 | 28[c] | 274 |
| | Na lactate | 0.31 | — | — | — | — | — | — |
| Potassium chloride[d] | KCl | 14.85 | — | 2 | — | 2 | — | — |

[a] Adapted from A. Arieff, *Clinical Disorders of Fluid and Electrolyte Metabolism*, 2d ed, Maxwell MH, Kleeman CR (eds). New York, McGraw-Hill, 1972.

[b] The 0.9*M* solution of $NaHCO_3$ usually is available in the clinical setting in 50-mL ampuls containing 44 meq of $Na^+$ and 44 meq of $HCO_3^-$. This solution can be infused intravenously or added to other solutions.

[c] Lactated Ringer's solution contains 28 meq/L of lactate, which is converted in the body to $HCO_3^-$.

[d] The KCl solution is available in 20- to 50-mL ampuls, which can be added to other solutions to provide $K^+$. The $K^+$ concentration in this solution is 2 meq/mL.

***THANKS FOR YOUR ATTENSION***