Maintenance and replacement fluid therapy in adults

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INTRODUCTION

A critical role of the kidneys is to maintain the effective circulating volume and plasma osmolality within relatively narrow limits, as well as to maintain electrolyte homeostasis. The normal homeostatic mechanisms that maintain the effective circulating volume and plasma osmolality are discussed elsewhere. (See "General principles of disorders of water balance (hyponatremia and hypernatremia) and sodium balance (hypovolemia and edema)".)

Under normal circumstances, the kidneys can adjust to wide variations in dietary intake by appropriate variations in water and electrolyte excretion [1], which is particularly important when discussing maintenance fluid requirements.

Water balance — Water losses lead to an increase in serum sodium and osmolality, resulting in stimulation of thirst and increased release of antidiuretic hormone (ADH). In normal individuals, these changes will lead to increased water intake and reduced water excretion, which will restore normal water balance. Thus, patients who are alert, have an intact thirst mechanism, and access to water will not become hypernatremic. (See "Etiology and evaluation of hypernatremia in adults".)

On a normal diet, the minimum water intake is estimated at 500 mL/day (assuming there are no increased losses). This value is based upon the balance of total water intake and production and the minimum rate of urinary loss. Individuals who can concentrate their urine to 1200 mosmol/L who excrete 600 mosmol of solute (sodium and potassium salts and urea) 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) and 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

However, the water of oxidation and much of the water lost from the lungs during respiration are linked [2]. The metabolic production of CO2 and water occur in a 1:1 proportion during the oxidation of carbohydrates and fatty acids and, if the arterial pCO2 is close to 40 mmHg, these two end-products are eliminated together in alveolar air in a 1:1 proportion. Water and CO2 are eliminated in parallel because the partial pressures of water vapor (47 mmHg) and CO2 (40 mmHg) are virtually equal in alveolar air, and because both CO2 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 [2]. 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 [15 to 30 mmol/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.

Sodium balance — Sodium intake and urinary sodium excretion are closely balanced. On a normal Western diet, the 100 to 250 mEq of sodium that are taken in are excreted in the urine. However, the kidney can excrete urine that is virtually free of sodium. As an example, in the era before antihypertensive drugs were available, most patients maintained sodium balance on the Kempner rice diet, which contained less than 10 mEq of sodium per day [3,4].

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 and replacement of mild fluid deficits will be reviewed here. The following discussion generally applies to patients with normal kidney function (and normal concentrating and diluting ability) and no underlying severe cardiac or hepatic dysfunction. Fluid and electrolyte balance are clearly different in patients with severe kidney disease or an edematous state. The treatment of severe hypovolemia or hypovolemic shock is discussed separately. (See "Treatment of severe hypovolemia or hypovolemic shock in adults".)

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. (See "Nutrition support in critically ill patients: An overview".)

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 and difficult to monitor.

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). (See "Etiology, clinical manifestations, and diagnosis of volume depletion in adults".)

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 kidney 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 antidiuretic hormone (ie, syndrome of inappropriate ADH secretion). (See "Pathophysiology and etiology of the syndrome of inappropriate antidiuretic hormone secretion (SIADH)".)

As mentioned above, 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 [5].

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 the renin-angiotensin-aldosterone system 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 (ie, 20 mmol) 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. The presence of dextrose in the solution does not alter its tonicity, and infusion of two liters of the dextrose-containing solution provides 400 kilocalories, enough to suppress catabolism. Patients with gastrointestinal or third-space losses may require a higher rate of saline (or blood) administration to maintain volume balance.

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).
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If the serum potassium starts to fall, more potassium should be added and, should it rise above normal, potassium should be eliminated.

In patients with normal or near-normal kidney function, hyperkalemia is a rare problem.

REPLACEMENT FLUID THERAPY

The goal of replacement therapy is to correct existing abnormalities in volume status and/or serum electrolytes. (See "Etiology, clinical manifestations, and diagnosis of volume depletion in adults".)

Volume deficit — There is no formula that can be used to accurately estimate the total fluid deficit [6]. 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, and, if baseline values are available and bleeding has not occurred, the hematocrit.

These parameters should be followed to assess the efficacy of volume replacement. If, for example, the urine sodium concentration remains below 15 mEq/L (15 mmol/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. (See "Pathophysiology and etiology of edema in adults", section on 'Renal sodium retention'.)

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). (See "Treatment of severe hypovolemia or hypovolemic shock in adults", section on 'Initial rate of fluid repletion'.)

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 [6]. 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, and 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.

Choosing a replacement fluid in patients with severe volume depletion, including a discussion about the use of crystalloid versus colloid, and the use of balanced crystalloid solutions versus isotonic saline are presented separately. (See "Treatment of severe hypovolemia or hypovolemic shock in adults", section on 'Choice of replacement fluid'.)

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. Because this complication has only been reported in infants, the proper rate of rehydration in adults is uncertain. If, however, it is known that the water deficit developed in less than 48 hours, hypernatremia can and should be corrected rapidly. The preferred rate of correction and the supportive data are reviewed elsewhere. (See "Treatment of hypernatremia in adults", section on 'Choosing a rate of correction'.)

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. (See "Treatment of hypernatremia in adults", section on 'Estimating the water deficit'.)

Sodium and/or potassium can be added to the intravenous 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 isotonic saline must be given to provide 1000 mL of free water. If potassium is also added to the intravenous fluid, then even 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 hours). 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.

On the other hand, if the cause of hyponatremia is the syndrome of inappropriate ADH secretion (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. (See "Osmotic demyelination syndrome (ODS) and overly rapid correction of hyponatremia" and "Overview of the treatment of hyponatremia in adults".)

Addition of potassium — Concurrent potassium replacement is indicated in patients who have developed potassium depletion as typically manifested by hypokalemia. (See "Clinical manifestations and treatment of hypokalemia in adults".)

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. (See "Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis", section on 'Serum potassium'.)

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 [7].

Potassium is as osmotically active as sodium. Thus, the addition of 40 mEq (ie, 40 mmol) of potassium to one liter of one-half isotonic saline (containing 77 mEq/L of sodium [ie, 77 mmol/L]) 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. (See "Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Treatment", section on 'Potassium replacement'.)

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 potassium replacement is no greater than 10 mEq per hour; in patients with life-threatening hypokalemia, the rate can be increased to 20 mEq per hour, although electrocardiographic monitoring is required. Thus, if 40 mEq of potassium has been added to a liter of intravenous (IV) solution, the rate of infusion should generally be limited to 250 mL per hour, or 500 mL per hour with electrocardiographic 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). (See "Approach to the adult with metabolic acidosis" and "Bicarbonate therapy in lactic acidosis" and

"Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Treatment", section on 'Bicarbonate and metabolic acidosis'.)

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 hypokalemia is the addition of three ampules of sodium bicarbonate (each containing 50 mEq of sodium and 50 mL of water) to one liter of 5 percent dextrose in water, which results in a nearly isotonic solution with a sodium concentration of approximately 130 mEq/L. In contrast, addition of the same three ampules of sodium bicarbonate to one-liter of half isotonic saline (containing 77 mEq/L of sodium) results in a hypertonic solution with a sodium concentration of 197 mEq/L, which will tend to raise the serum sodium concentration. Such a solution should not be used unless the patient is hyponatremic (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. (See "Hypoglycemia in adults with diabetes mellitus", section on 'Reversing hypoglycemia' and

"Treatment and prevention of hyperkalemia in adults", section on 'Insulin with glucose' and "Fasting ketosis and alcoholic ketoacidosis".)

- 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. (See "Clinical manifestations and treatment of hypokalemia in adults", section on 'Intravenous therapy'.)

Dextrose-induced hyperglycemia — The administration of large volumes of dextrose-containing solutions to critically ill patients can promote the development of hyperglycemia [8-10], 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 [8].

In studies of patients without diabetes who are treated with total parenteral nutrition (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 [9-11]. 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.

The usual safety of lower glucose infusion rates was demonstrated in a report of TPN in hematopoietic stem cell transplant recipients, who are typically highly stressed [12]. The proportion of hyperglycemic days was not increased compared to patients not treated with TPN at an average glucose infusion rate of 2.7 mg/kg per min (range 1.3 to 3.9 mg/kg per min).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Hyponatremia" and "Society guideline links: Fluid and electrolyte disorders in adults".)

SUMMARY AND RECOMMENDATIONS

Maintenance therapy — The goal of maintenance fluid therapy is to preserve water and electrolyte balance and to provide nutrition among patients who are not able to eat or drink.

- 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. Requirements are increased if there are increased fluid losses as with fever, surgical drains, or ongoing significant gastrointestinal losses. Water requirements are decreased by oliguric kidney failure, the use of humidified air, edematous states, hypothyroidism, and the presence of nonosmotic stimuli for the release of antidiuretic hormone (ie, syndrome of inappropriate ADH secretion). (See 'Maintenance fluid therapy' above.)

- For patients who require maintenance fluid and have normal or near-normal kidney function and are otherwise stable, we suggest beginning with two liters per day of one-half isotonic saline in 5 percent dextrose to which 20 mEq (ie, 20 mmol) of potassium chloride is added per liter (**Grade 2C**). (See 'Electrolytes' above.)

    - Patients with gastrointestinal or third-space losses may require a higher rate of saline (or blood) administration to maintain volume balance.

- The original maintenance regimen can be continued unless one of the following occurs (see 'Electrolytes' above):

    - If the serum sodium starts to fall, a more concentrated solution should be given (eg, isotonic saline).

    - If the serum sodium starts to rise, a more dilute solution should be given (eg, one-quarter isotonic saline).

- If the serum potassium starts to fall, more potassium should be added, and, if it rises above normal, potassium should be eliminated.

Replacement therapy — The goal of replacement therapy is to correct existing abnormalities in volume status and/or serum electrolytes. (See 'Replacement fluid therapy' above.)

- The total fluid deficit can be estimated from pre- and post-deficit body weight. 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 and urine sodium concentration. (See 'Volume deficit' above.)

- The rate of correction of volume depletion depends upon its severity. Patients with severe volume depletion or hypovolemic shock are generally administration 1 to 2 liters of isotonic fluids 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). (See 'Rate of replacement' above.)

- A less rapid rate of correction should be used in patients with mild to moderate hypovolemia. In such cases, 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. (See 'Rate of replacement' above.)

- The choice of replacement fluid is dependent upon the type of fluid that has been lost and any concurrent electrolyte disorders. Most patients are initially treated with balanced crystalloid solutions or isotonic saline. (See 'Choice of replacement fluid' above.)

- Hypernatremia and hyponatremia should usually be corrected slowly since overly rapid correction is potentially harmful. (See 'Hypernatremia' above and 'Hyponatremia' above.)

- Potassium replacement is indicated in patients with potassium depletion. This is typically manifested by hypokalemia, but may occur in the setting of a normal or

even increased serum potassium in patients with diabetic ketoacidosis or nonketotic hyperglycemia. (See 'Addition of potassium' above and "Diabetic ketoacidosis and hyperosmolar hyperglycemic state in adults: Clinical features, evaluation, and diagnosis", section on 'Serum potassium'.)

- The addition of sodium bicarbonate may be required in patients with metabolic acidosis if the acidemia is severe (arterial pH less than 7.2) or bicarbonate losses persist (as with severe diarrhea). (See 'Addition of bicarbonate' above and "Approach to the adult with metabolic acidosis".)

- In general, there is little evidence that adding or omitting dextrose from saline has any benefit or harms. However, there are settings in which dextrose should or should not be used. (See 'Saline alone or with dextrose' above.)

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