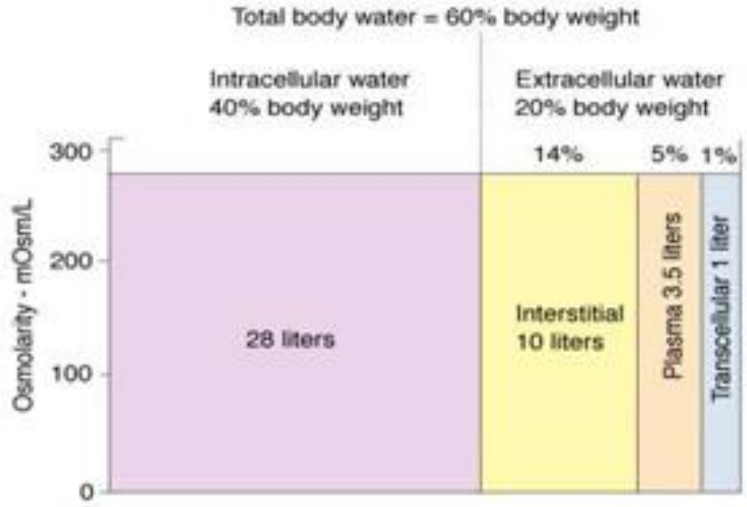
FLUID AND ELECTROLYTE THERAPY IN CHILDREN

Steven R. Alexander, M.D. Stanford, California; Michel G. Baum, M.D. UT Southwestern at Dallas, Texas

I. Distribution of Fluids and Electrolytes

Total body water constitutes 75% of the weight of the term infant and decreases to two- thirds of body weight after the neonatal period. Two-thirds of total body water is in the intracellular space and one-third is in the extracellular fluid space. Of the extracellular fluid (ECF), only 25% is intravascular. Thus, only about 7-8% of total body water is intravascular.



Note: These are approximate sizes of body composition in 70 kg adult

The composition of the ECF is what we measure when we obtain a set of electrolytes. The sodium content of the ECF is approximately 140 mEq/l, and the potassium is only 4-5 mEq/l. The predominant anions in the extracellular fluid are chloride and bicarbonate. The composition of the intracellular fluid (ICF) is dramatically different. Whereas there is a very small concentration of potassium in the extracellular fluid, the predominant cation of the intracellular fluid is potassium with an intracellular concentration of 140 mEq/l. The intracellular chloride concentration is very low in most cells. The bulk of the anions in the intracellular compartment is made up of charges on proteins and other impermeant molecules.

II. Basic Fluid and Electrolyte Therapy: Maintenance

The goal of maintenance therapy is the accurate replacement of ongoing water and electrolyte losses to maintain zero balance; that is: INTAKE = OUTPUT. In very unstable patients with abnormal or unpredictable losses, zero balance can be achieved only by frequent replacement of precisely measured losses. In more stable patients, it is clinically useful to begin fluid therapy by estimating normal maintenance requirements using the estimated caloric expenditure method.

A. Estimated Caloric Expenditure

Normal maintenance fluid and electrolyte requirements are, in general, determined by the child's metabolic rate. While numerous methods have been proposed for estimating the metabolic rates of hospitalized children, the method of Holliday and Seegar (Holliday MA, Segar WE: *Pediatrics* 1957; 19:823) has gained wide acceptance and has stood the test of time. It is easy to remember, and has proven to be sufficiently accurate for most clinical situations. Holliday and Seegar calculated the rate of caloric expenditure (i.e., the metabolic rate) of hospitalized children and found that it was proportional to the child's weight according to the following:

For the first:	3 Kg to 10 Kg allow:	100 Cal/kg/day;
plus, for:	11 Kg to 20 Kg allow:	50 Cal/kg/day;
plus, for any:	> 20 Kg allow:	20 Cal/kg/day.

For example:

Child's Weight	Estimated Caloric Expenditure
a. 9 Kg	900 Cal/day
b. 19 Kg	1000 + 450 = 1450 Cal/day
c. 29 Kg	1000 + 500 + 180 = 1680 Cal/day
d. 70 Kg	1000 + 500 + 1000 = 2500 Cal/day

The first step in maintenance fluids calculations is the calculation of the daily estimated caloric expenditure. From this number, all else follows logically.

B. Water – normal requirements

1. Insensible Water Loss (IWL) – evaporative losses from the skin and lungs which cannot be directly measured. (Does not include sweating).
   Skin losses = 30 cc/100 Cal/day
   Pulmonary losses = 15 cc/100 Cal/day
   IWL = 45 cc/100 Cal/day
   (In actual practice, the IWL of hospitalized children varies from 30 to 45 cc/100 Cal/day).

2. Renal Water Loss – the daily obligate urinary water loss is determined by the renal solute load and the concentrating ability of the child's kidneys. The renal solute load consists primarily of urea and major electrolytes (Na, K, Cl); under usual conditions seen in hospitalized children this is approximately 14.5 mOsm/100 Cal. Enough water should be provided for urine formation to avoid the need to either concentrate or dilute the urine, yielding a urine which is nearly isotonic with plasma (≅ 290 mOsm/L)
   Therefore obligate renal water loss = $\frac{\text{normal renal solute load}}{\text{desired urine concentration}}$

   = $\frac{\text{14.5 mOsm/100 Cal}}{\text{290 mOsm/L}}$ ≅ 50 cc/100 Cal

3. Stool water losses – stool water losses in absence of diarrhea are minimal (nl stool water loss = 5 cc/100 Cal/day)
   Remember that with diarrhea, stool water losses increase dramatically.
   Diarrhea water losses must be measured and replaced cc for cc.
   To summarize normal maintenance water requirements:

IWL	= 45
Renal	= 50
Stool	= 5
Total	100 cc/100 Cal/day

   Thus, one simply calculates child's estimated caloric expenditure (Holliday & Seegar) and provides 1 cc/1 Cal.
   Example: 25 Kg child
   Estimated caloric expenditure = 1000 + 500 + 100 = 1600 Cal/day
   Maintenance fluid requirement = 1600 cc/day = 67 cc/hr

The approach listed above assumes that for every hundred kilocalories metabolized, 100 ml of water is required. In truth, the actual water needs are approximately 120 ml of water per every 100 kilocalories, but 20 ml of water is obtained from the water of oxidation leaving us to provide the additional 100 cc's.

C. Electrolytes

Estimates of the normal requirements for major electrolytes are several times greater than actual minimum requirements; however, vigorously anabolic children may have even greater requirements.

To replace normal urinary electrolyte losses and provide additional electrolytes for growth, roughly 2-3 mEq of sodium and chloride, and 2 mEq of potassium are required for each 100 kilocalories of energy expended or 100 cc's of maintenance fluid. Thus, under ordinary conditions where a patient has a normal cardiovascular status, and normal renal function, adequate electrolytes will be provided using an intravenous fluid containing ¼ normal saline (Na = approx. 35 mEq/l), with 20 mEq of potassium per liter.

It should be remembered that these estimates of pediatric patient electrolyte requirements are based on the electrolyte composition of normal infant feedings (human breast milk, cow's milk, etc). Some authorities recommend higher sodium concentrations (eg., ½ normal saline) for older/larger children. However, in practice, the use of ¼ normal saline will suffice in most settings.

It must also be remembered that to arrive at these recommendations many assumptions are made in terms of the patient's normal renal and cardiovascular status. It is thus very important that you reassess any patient receiving IV fluids and determine serum electrolytes periodically.

D. Energy/Calories

As pediatricians we try to practice effective preventive medicine, and the same approach is necessary when considering maintenance fluids. One aspect of maintenance fluids which we have not considered is caloric/energy needs. While the use of D5 ¼ normal saline provides some calories in the form of dextrose, only 20% of maintenance caloric needs are being met this way. Whenever you consider providing IV fluid therapy, you need to make a nutritional assessment as well. If the patient is well nourished and will only be on intravenous therapy for a few days, the above maintenance fluids are satisfactory. However, if the patient is malnourished, or there is a potential for the patient to need intravenous fluids for a prolonged period of time, one must consider hyperalimentation. This should always be done sooner rather than later, for catch-up nutritional therapy does not work well in the sick child.

E. "Abnormal" – maintenance requirements

In unstable patients with abnormal requirements it is always best to replace measured losses cc for cc and mEq for mEq. The following guidelines may be helpful, but are only approximations.

1. Fever – Intermittent temperature elevations usually do not significantly increase caloric expenditure. If fever is present for a large part of the day the estimated caloric expenditure is increased by: 12% for each 1° above 37°.

   Example: 25 Kg child with average temperature of 38.5°C
   Est. caloric expenditure = 1600 x 1.18 = 1888 Cal/day

2. Sweat – to normal maintenance the following is added:
   + 30 cc/100 Cal/°C > 30.5°C ambient temp (87°F)

+ 0.5 to 1 mEq NaCl/100 Cal/day
(+ 2-3 mEq NaCl/100 Cal/day in cystic fibrosis pts.)

Remember – normal IWL contains no electrolytes. The electrolyte content of sweat is also variable according to the degree of acclimatization of the patient.

3. Abnormal Gastrointestinal losses
   a. Water – replace measured NG drainage, emesis, diarrhea, ostomy losses, etc. cc for cc. No other approach will do in the severely ill patient.

   b. Electrolytes – it is often best to directly measure electrolyte content of abnormal GI losses. The following approximations provide a point at which to begin:

Sources of Unusual Loss	Concentration of Electrolytes (mEq/L)			
	Na^+	K^+	Cl^-	HCO_3^-
Gastric	140	15	155	0
Small Bowel (ileostomy)	140	15	115	40
Diarrhea (non-secretory)	40	40	40	40

4. Chest tube drainage – replace cc for cc with 5% albumin or other colloid solution.
5. Hyperventilation – when respiratory rate increases, the pulmonary component of IWL increases proportionately. Thus, if RR is 40-50/min, IWL is increased by 15-25 cc/100 Cal/day to a total of 60-75 cc/100 Cal/day.
6. Mechanical ventilators – The humidified gases provided by ventilators and hoods greatly decrease pulmonary water loss. In some cases these devices actually deliver water to the patient. When the difference between observed and estimated requirements defies explanation, variable amounts of water gained from the ventilator may be the reason.

III. Dehydration

A. Factors producing dehydration:
Dehydration or contraction of the body fluid compartments will occur whenever the loss of water and salt exceeds the intake. Fever, sweating and diarrhea produce losses in excess of normal, but if intake remains good, patients will often be able to compensate for the increased losses. Anorexia and/or vomiting will impair this ability to compensate; in fact, due to continuing obligatory losses, dehydration can occur even in the absence of abnormal losses if anorexia is severe or prolonged.

B. Types of dehydration will depend on the relative losses of salt and water which occur and on the composition and volume of the intake received.

1. Isotonic:
   In this type of dehydration, the losses of water and electrolytes have been proportional and the ratio between solute and water in the body fluids remains normal although the total amounts of both salt and water are reduced. There are no shifts of fluid from ICF to ECF or vice-versa. The normal tonicity of body fluids is 275-295 mOsm/Kg. In isotonic dehydration, the serum sodium concentration is between 130 and 150 mEq/L. This is the most common type of dehydration and the type with the best prognosis.

2. Hypertonic:
   As a result of the balance of intake (volume and composition) and output (volume and composition), the losses of water exceed the losses of solutes so that the osmolarity of the

body fluids increases (serum osmolarity in excess of 300 mOsm/Kg or serum sodium of over 150 mEq/L). This type of dehydration is seen more commonly in infants under 6 months of age, suggesting that renal immaturity may be a factor. A history of continued intake of relatively high solute fluids such as undiluted milk or concentrated oral electrolyte solutions may be obtained but not necessarily so. There is often a history of markedly reduced oral intake of any fluids. Fluid losses may be from both ECF and ICF spaces are usually predominantly from the intracellular space (intracellular dehydration). Possibly due to the intracellular dehydration, CNS signs and symptoms are common (i.e., stupor, coma, hypertonia, convulsions). Vascular volume is usually maintained until the degree of dehydration is quite severe.

3. Hypotonic:
   The loss of salt over a period of time exceeds the loss of water (a balance between the volume and composition of intake and the volume and composition of renal, G.I. and sweat losses) so that the tonicity of the body fluids diminishes (osmolarity less than 270 mOsm/kg; serum sodium less than 130 mEq/L). Acute hypotonic dehydration may be seen in older infants and children with the severe diarrhea associated with bacterial G.I. infection (i.e., shigella, salmonella) in which the stool volumes may be large and contain a fairly high concentration of salt.

   This type of dehydration may occur when patients have received as their only intake very low salt containing fluids such as water, rice water, or tea over a period of time and is also seen in malnourished and chronically ill patients. Hypotonic dehydration is also seen in adrenal insufficiency.

   Not only is fluid lost to the outside of the body but there is also a shift of fluid from the ECF to the ICF. Due to the predominant loss of extracellular fluid in hypotonic dehydration, vascular collapse is seen more often and earlier than in the other types of dehydration.

C. Estimation of the antecedent deficit (expressed in relation to the body weight of the patient):
   Since accurate weights prior to onset of illness are not often available, it is necessary to estimate the degree of weight loss by careful appraisal of the physical status of the patient.

Signs and Symptoms of Volume Depletion

VOLUME DEPLETION	SIGNS AND SYMPTOMS
Mild (3-5% Volume Depletion)	Thirst, decrease in urine output, ± dry mucous membrane.
Moderate (6-10% Volume Depletion)	Postural changes in blood pressure and heart rate, dry mucous membranes, sunken eyes and fontanel, skin tenting, listlessness, tachycardia.
Severe (>10% Volume Depletion)	Poor perfusion, tachycardia, hypotension, lethargy and coma.

D. Fluid therapy of dehydration:

Isotonic Dehydration

The losses of fluid in most cases of dehydration do not come equally from the intracellular and extracellular fluid volumes. Most cases of volume depletion are due to a loss of extracellular fluid volume. Only in cases of prolonged dehydration will there be substantial losses from the intracellular fluid compartment as well. It is thus appropriate to think about dehydration as a shrinkage of the extracellular fluid compartment. Repletion of those losses should be performed with fluids which resemble the extracellular fluid compartment. In most cases of dehydration, the fluid deficit is replaced with normal saline. Thus, if one calculates the amount of fluid required to replace the deficit with normal saline, plus the maintenance fluids in the form of ¼ normal saline,

most cases of isotonic dehydration will utilize ½ normal saline with 20-30 mEq/l of potassium as the intravenous fluid.

The repair of a deficit can be broken down into two phases. In the initial phase of fluid therapy one needs to restore the intravascular volume. This phase should be reserved for patients who are significantly dehydrated or have any signs of vascular instability. In most cases, the patient should receive 1-2% of their body weight (10-20 cc's/kg) of an isotonic fluid in the form of normal saline or Ringer's lactate. If the patient is hypotensive or shocky, one may need to give more fluid than this, and 3-5% of their body weight in the form of normal saline or lactated Ringer's can be given in an emergency situation. If the patient is shocky or hypotensive, 5% albumin should also be considered. If the patient is having excessive blood loss one should utilize blood as well, but by no means wait for blood to arrive before instituting aggressive fluid therapy.

The second phase of fluid therapy is to provide maintenance plus deficit replacement. In the first 8 hours one should give 1/3 of the normal maintenance plus replacement of ½ of the estimated deficit. One should replace the other 50% of the deficit plus deliver the required maintenance solution over the next 16 hours. The fluid that should be utilized during the period of deficit repair is again a combination of the isotonic fluid required to replete the volume deficit, plus ¼ normal saline. Thus, one usually uses D5 ½ NS with 20-30 mEq K/l. The concentration of potassium should not ordinarily exceed 40 mEq/l (4 mEq/100 cc's) nor should the rate of infusion of potassium be >0.5 mEq/Kg/hr.

Potassium should never be added to IV fluid therapy unless one is sure that the patient is not in renal failure. Thus, one should have a serum creatinine and be sure that the patient is voiding prior to the institution of potassium therapy. Once volume depletion has been corrected one can then go back to simple maintenance fluids as described above.

Hypotonic Dehydration

Most cases of dehydration in children and adults are isotonic. However, there are patients who have either hyponatremic or hypernatremic dehydration, but it must be emphasized that it is of extreme importance that one determines the etiology of the hyponatremia or the hypernatremia during the course of the patient's hospitalization.

If the patient has hyponatremic dehydration, one can use the formula outlined below to calculate the amount of sodium that would be necessary to increase the serum sodium to the desired level. This sodium deficit is in addition to the other deficits outlined above.

$$Na_{deficit} = (Na_{desired} - Na_{current}) \times (0.6) \times (\text{Body wt. in Kg})$$

The rate at which the serum sodium should be corrected had been under some debate. However, it is now generally agreed that the serum sodium should be corrected slowly to prevent central pontine myelinolysis. Thus, the serum sodium should not increase by more than 15 mEq/l in a 24-hour period. If a patient has hyponatremic dehydration, the serum sodium needs to be measured frequently.

Hypertonic Dehydration:

Hypernatremic dehydration is extremely unusual. At the time of presentation one needs to make a determination of the cause of the hypernatremic dehydration. To repair the hypernatremic dehydration one has an additional free water deficit in addition to the deficits outlined above. The free water deficit needs to be repaired slowly. Under no circumstance should the serum sodium decrease by more than 15 mEq/l in a 24-hour period. Should this occur cerebral edema and death can follow.

The equation to estimate the free water deficit is shown below.

$$FreeWaterDeficit = \frac{Na_{observed}}{Na_{desired}} x(0.6)x(wt.inKg) - (0.6)x(wt.inKg)$$

As you can see, the equation is simply a calculation of the total body water times the ratio of the observed sodium divided by the desired sodium. This will give you the amount of total body water that you want your patient to have. From this you need to subtract the amount of total body water which you have at the time of observation. That difference is the free water deficit.

As with hypotonic dehydration, it is extremely important that the serum electrolytes be measured frequently during the course of correction of the hypernatremic dehydration. One should err on the side of correcting the hypernatremic dehydration too slowly rather than too rapidly.

E. Examples

At this point, a few examples will be provided to show you an approach to calculating fluids and electrolytes. It is instructive to break down each component of fluids and electrolytes individually and then come up with a composite fluid. Let's take the example of a 15 kg child who presents with a 5% fluid deficit. This child will not only need his maintenance IV fluids, but will also need to have his deficit replaced. To determine the amount of volume and the sodium composition of that solution to be administered, figure out how much fluid this child will need, the sodium composition in mEq/l of each of these fluids, and then the total amount of sodium. Finally, divide the total sodium by the volume to get the sodium composition of the fluid to be administered. This is illustrated in the table below.

	Water (ml)	Na (mEq/L)	Total Na (mEq)
Maintenance/24 hrs	1250	35	44
Deficit	750	140	105
Total	2000		149
	or 75 mEq/l Na		

The maintenance volume is calculated using the standard formula. One uses ¼ normal saline containing approximately 35 mEq/l, and from our calculation, one needs 1250 cc's of fluid per 24 hours. The total amount of sodium to be administered in that maintenance fluid is 44 mEq. Now, this child also has a 5% fluid deficit. This will require administration of 750 cc's (5% of this child's body weight) as an isotonic fluid containing approximately 140 mEq/l of Na. The total sodium that this child will receive from this fluid is 105 mEq. Thus, in total, we are going to give 149 mEq of sodium in 2000 ml, or approximately 75 mEq/l sodium. One would administer D5 ½ NS with 20 mEq K/L (once we are sure that this child has normal renal function) at a rate to equal 2000 ml in the next 24 hours. If one replaces one-half of the deficit in the first 8 hours one would administer this fluid at 100 ml/hr for the first 8 hours and then 75 ml/hr for the next 16 hours. If the child is then volume replete one can then administer maintenance fluids.

With the second example, let us say we have the same 15 kg child who now presents with a 5% volume deficit, but has a serum sodium of 120 mEq/L. One would only want to increase the serum sodium by 15 mEq/L in the next 24 hours. This child will still require his maintenance fluids, and will still have a volume deficit just as we have calculated before. In addition this child also has a sodium deficit which is calculated as described above. This sodium deficit is 135 mEq

(15 x 0.6 x 15) and will have to be added to the total sodium that we administer to this patient, as shown below.

	Water (ml	Na mEq/L	Total Na (mEq)
Maintenance	1250	35	44
Deficit	750	140	105
Na Deficit			135
Total	2000		284
	or 142 mEq/L Na		

The amount of maintenance fluid and the fluid deficit of isotonic solution has not changed. What we have added at this point is enough sodium to replete an additional sodium deficit. We are now going to administer 2000 ml's and 284 mEq of sodium. This child will get D5 NS with the addition of 20 mEq/L of KCl when normal renal function is established.

It is best to administer these fluids evenly over 24 hours to avoid too-rapid rises in serum sodium concentration during the early phases of therapy. However, patients with hypotonic dehydration may have significant cardiovascular instability due to reductions in ECF volume (see above). Aggressive therapy of shock with isotonic fluids always takes precedence over concerns about too-rapid rises in serum sodium concentration.

The next problem is the treatment of hypernatremic dehydration. This is conceptually a little bit more difficult to understand. First, consider a simplistic example: Let us take a 5 liter beaker that has a sodium content of 165 mEq/l. If we would like to drop the sodium content to 150 mEq/l by adding more water, we must calculate the final volume needed to drop the sodium concentration to 150 mEq/l and then subtract the volume that we have. This will give us the volume of water to be added.

The volume we have in the case of our hyernatremic beaker is quite simple. It is 5 liters. How do we calculate the volume that we want? This is simply the ratio of the sodium that we have divided by the sodium that we want [(165/150) X 5 liters] as shown in the figure below. The volume that we want is 5.5 liters. The volume that we have is 5 liters. The additional water we need to add to drop the sodium by 15 mEq is 0.5 liters. The same principle holds true when one does this to a

patient.
Hypernatremia is extremely rate, and one must determine the reason for hypernatremia in any patient. Probably the greatest risk to a patient with hypernatremia is that someone is going to correct the hypernatremia too rapidly. **Never correct hypernatremia by more than 15 mEq in 24 hours; otherwise cerebral edema will ensue.**

We are now going to use the same principles that we have learned before, to deal with a 15 kg patient who presents with a serum sodium of 165 mEq/l. We are going to want to bring this serum sodium down to 150 mEq/l during the first 24 hours of fluid therapy. Strictly speaking, we are not only bringing down the serum sodium, but we are actually correcting the osmolarity of the extracellular fluid compartment and the intracellular fluid compartment at the same time since water is freely permeable across cell membranes. Thus, in all of our calculations we use the percentage 0.6 or the percentage of our body which is made up of water, as shown below.

The free water deficit is, once again, the volume of fluid in the body that we want, minus the volume of fluid in the body that we have. The volume of body fluid that we have is 0.6 X the

patient's weight in kg. The volume of fluid that we want is that fraction multiplied by the ratio of sodium that we have divided by the sodium that we want, as shown below.

Free water deficit = $\frac{165}{150}$ X (0.6)(15) - (0.6)(15) = 0.9 liters

To calculate the fluid requirements of this patient, one has to again give this child maintenance. The deficit is no longer made up of of normal saline, but rather is made up of the free water deficit that we calculated above. Note that the free water deficit (900 ml) exceeds the clinical estimate of 5% dehydration (750 ml). This is not uncommon in hypernatremic dehydration where increased ECF tonicity supports ECF volume and masks the clinical severity of dehydration. The portion of the free water deficit to be replaced during the first 24 hours is 900 ml. The TOTAL free water deficit is actually 1.6 liters! (165/140 X 0.6 X 15) – (0.6 X 15).

	Water (ml)	Na mEq/L	Na mEq Total
Maintenance	1250	35	44
Deficit-Free water	900	--	--
Rest of deficit	--	140	--
Total	2150		44
	or 20 mEq/L Na		

As you can see in this case we are going to want to administer 2.15 liters of D5 1/8 normal saline (with 20 mEq of KCl/L once we are sure that the child has normal renal function). In the case of hypernatremic dehydration, it is essential to administer fluids at an even rate to avoid abrupt changes in serum sodium concentration.

Of course, your patient is not a beaker, and many factors will influence the rate at which the serum sodium falls. It is essential to measure serum sodium concentration at least every two hours and adjust therapy accordingly. It may be easier in some cases to add a second IV of D5W that can be adjusted frequently to fine tune the rate of hypernatremia correction.

Finally, the most important thing to remember is that all of what we have discussed reflects approximations. You will need to observe your patients frequently to assess their volume status and to reevaluate their serum electrolytes to make sure that your calculations have come close to the patient's requirements.

F. Evaluation of adequacy of therapy:
   a) During the <u>first day</u>, fluid orders should not be written for more than 4-6 hours in advance. Constant re-evaluation of fluid estimates is necessary.
   b) Clinical improvement of the patient should occur with rehydration, particularly in regard to general appearance, color, vital signs, sensorium status, urine output and activity.
   c) Patients should be weighed frequently. Weigh diapers. Precise I & O is essential.
   d) The establishment of adequate urinary output should be achieved by 4 to 6 hours.
   e) Beware of ongoing, underestimated stool losses or third space losses into the bowel lumen.
   f) Failure of clinical response in 6 to 10 hours may indicate severe metabolic, renal, adrenal, or posterior pituitary dysfunction which may require specific treatment.
   g) The guides to fluid therapy given in this outline can in no way substitute for sound clinical judgment.

IV. Sodium Problems

A. Basic considerations

Because sodium is primarily concentrated in the ECF, to which we have ready access, most abnormalities of sodium concentration lend themselves to rational and reliable therapy. Sodium "behaves" as if it were distributed evenly throughout the total body water. Thus the "sodium space: may be estimated simply: Wt (Kg) X 0.6 = "sodium space"

1. A 10 Kg infant with known WPW is seen in the ER for an episode of SVT refractory to usual conversion techniques. His serum Na is 130 mEq/L. A "keep open" IV of D_5W is started in preparation for transfer to the ICU. On arrival in the PICU about an hour later the infant has several generalized seizures. During the efforts to perform emergency DC cardioversion it is not recognized that approximately 750 cc of his D_5W IV bottle has been infused during transfer from the ER. Why is this the more likely etiology of his seizures than cerebral hypoxia from SVT?

   Answer: His serum Na is now 115 mEq/L.
   10 kg x 0.6 = 6 L "sodium space"
   6 L x 130 mEq/L = 780 mEq of Na
   Following 750 cc D_5W infused rapidly, new "sodium space" is 6.75 L
   780 ÷ 6.75 = 115 mEq/L

2. A 5 kg infant receives sodium bicarbonate during CPR. If his serum Na was 140 at the time of his arrest, what dose of sodium bicarbonate will it take for his serum Na to exceed 150 mEq/L?

   Answer: 30 mEq or six 1 mEq/Kg doses.

B. Hyponatremia: treatment strategies

1. Acute, severe, symptomatic, hyponatremia:
   a. Usually seen when serum Na < 115 mEq/L; develops <u>rapidly</u>, in an euvolemic pt. with access to free water.
   b. Cerebral edema can be severe, rarely life-threatening.
   c. Infants and young children most often present with seizures; older patients more often obtunded or comatose.
   d. Hypertension and bradycardia are inconsistent findings, especially in infants who may only have hypertension as ICP rises secondary to cerebral edema.
   e. Goals of treatment:
      1. Control seizures with anticonvulsants.
      2. Protect airway, support ventilation.
      3. Correct serum Na to 125 mEq/L in 8 hours. Do not calculate emergency correction beyond 125 mEq/L.
         a. First, calculate sodium deficit:
            (125 – pt.'s serum Na) x Wt (Kg) x 0.6 = mEq of Na required.
         b. Choose repair solution
            0.9% (Normal) saline = 154 mEq/L
            3% saline = 530 mEq/L
            5% saline = 884 mEq/L
         c. Rate of repair depends on severity of cerebral edema. In severe cases give ½ the Na deficit in the first 2 hours, then remainder in next 6 hours. <u>This rapid correction is reserved for pts. with symptomatic cerebral edema secondary to hyponatremia.</u>

d. Example: Comatose 10 Kg. Infant with generalized seizures poorly controlled with anticonvulsants; dilated pupils; unstable VS: (BP 130/90, HR 100, requiring ventilatory support for intermittent apnea); serum Na 110 mEq/L; Na deficit = (125-110) x (10 Kg) x (0.6) = 90 mEq.
Repair solutions: 0.9% saline = 584 cc required
3% saline = 170 cc required
5% saline = 102 cc required
Rate: 3% saline @ 43 cc/hr x 2 hrs, then 14 cc/h for 6 hr.
5% saline @ 25 cc/hr x 2hr, then 9 cc/hr for 6 hrs.
Do not use 0.90 saline in this patient. The volume required for correction is too great. N.B. Hypertonic saline causes a chemical necrosis if the I.V. infiltrates. During hypertonic saline
infusions, IV site must be checked q15 min. to assure patency and absence of infiltration.

e. Some authorities recommend Mannitol over hypertonic saline in such patients. Mannitol is an effective therapy for cerebral edema, but it will worsen hyponatremia by dilution and increased urinary Na losses, making eventual correction of hyponatremia more difficult. If hypertonic saline is not readily available, Mannitol (1 gram per kg of 25% Mannitol solution over 1 hour) is an acceptable alternative in an emergency.

f. Some authorities recommend routine use of a potent diuretic, such as Furosemide, during the treatment of hyponatremia with hypertonic or isotonic saline. Furosemide causes a net increase in free water excretion by kidneys. However, urinary sodium losses are also increased to an unpredictable degree. If furosemide is used, urinary sodium and water losses must be precisely monitored and replaced appropriately.

2. Subacute, chronic, asymptomatic hyponatremia:
   a. When hyponatremia develops slowly, cerebral edema does not occur b/c brain cell volume is maintained by active depletion of intracellular solutes (Na, K, Cl, amino acids). Total brain water content remains constant despite serum Na levels as low as 100 mEq/L when these levels are achieved slowly (i.e., over several days).
   b. The absence of signs and symptoms of cerebral edema in a patient with severe hyponatremia is evidence for a chronic process to which the brain has successfully adapted.
   c. Rapid correction of severe <u>chronic</u> hyponatremia is unnecessary and potentially harmful, because rapid correction causes acute dehydration of the brain and may result in permanent brain injury.
   d. <u>Osmotic Demyelination Syndrome:</u> Brain injury due to too-rapid correction of chronic hyponatremia is often associated with delayed neurological deterioration (onset of symptoms 1 to 4 days after serum Na is increased by > 12 mEq/L in less than 24 hours.)
   e. Example: Asymptomatic 10 Kg infant with serum Na = 110 mEq/L
   Rate of repair = 0.5 mEq/L/hr
   Repair solution: 0.9% saline = 1.54 mEq/10 cc = 20 cc/hr x 30 hours
   Ongoing urinary Na losses must be measured and replaced mEq for mEq, usually with a separate IV.

3. If it is not clear whether the individual patient has acute or chronic hyponatremia, correct as if the patient has chronic hyponatremia to avoid the osmotic demyelination syndrome.

V. Potassium Problems

A. Factors affecting serum [K+]

1) 98% of total body potassium is intracellular. It is therefore impossible to calculate potassium deficits or excesses as can be done with sodium. Potassium balance is primarily maintained by the kidney, largely the distal tubule and collecting duct where K is excreted in exchange for Na. Aldosterone potentiates this exchange as does anything which increases distal Na delivery. Insulin and catecholamines are also important in minute to minute regulation of ECF potassium concentration.

2) Common sources of abnormal K+ input:
   a) Diet – Starvation often leads to hypokalemia. High dietary K intake can transiently exceed secretory capacity of very young infants but is not a problem in older patients with normal kidney function.
   b) IV fluids – remember the first step in the hyperkalemic patient is to remove K from IV fluids.
   c) Salt substitutes are largely KCL.
   d) Hemolysis and tissue trauma release vast amounts of potassium into the ECF.

3) Common sources of abnormal K+ output:
   a) Renal insufficiency (including "pre-renal"). The ability to excrete K drops in proportion with fall in GFR.
   b) Mineralcorticoid imbalance. Potassium wasting in hyperaldosteronism (1° or 2°), Bartter's Syndrome. Potassium retention in Addison's, Congenital Adrenal Hyperplasia.
   c) Increased distal Na delivery: Diuretics; obstructive uropathy (after relief of obstruction); osmotic diuresis (Diabetes Mellitus, urea); non-oliguric acute renal failure; Type II (proximal) RTA; volume expansion.
   d) Chronic hypomagnesemia causes potassium wasting
   e) GI losses (diarrhea, emesis, NG suction, ileostomy drainage, etc.)

4) Alterations in serum [K+] unrelated to I & O are the result of :
   a) Rapid changes in pH
   "Alkalosis drives K into cells" "Acidosis pulls K out of cells." In general, a 0.1 unit change in pH yields a 0.6 mEq/L change in serum [K+]. If pH <u>rapidly</u> rises from 7.2 to 7.4, serum [K+] will decrease ≅ 1.2 mEq/L.
   b) Hyperglycemia in presence of insulin, drives potassium into cells. IV solutions with hypertonic dextrose (D_{10} or greater) can produce hypokalemia (and hypophosphatemia).
   c) Insulin in IV fluids profoundly lowers serum [K].

5) Sources of spurious serum [K+] values:
   a) Too high: hemolysis of sample; polycythemia (blood drawn through ≤ 21 ga. Needle)
   b) Too low: dilutional sampling through a line, hep. lock, etc.

B. Hypokalemia

1) Diagnosis
   a) Symptoms: Musculoskeletal = weakness, cramps, paresthesias, paralysis; GI = nausea, anorexia, vomiting, diarrhea; CNS = lethargy, confusion.
   b) Physical findings: hyporeflexia
   c) Lab findings: low urine specific gravity; low serum K+ (<3.5 mEq/L requires therapy).
   d) EKG findings: flat or inverted T waves; ST depression. Less commonly: QT Long, wide QRS, U wave. With digitalis toxicity: all of the above plus various AV blocks, bradycardias, and arrhythmias of any type.

2) Treatment
TREAT AS IF THERE IS AN EMPTY TANK INTO WHICH YOU CAN'T SEE
a) When patient is symptomatic, or on Digitalis, rapid correction is needed.
b) P.O. potassium therapy is safest and should be used whenever possible. You can give 1-2 meq/Kg. po every few hours, checking serum K+ q 4 hrs.
c) IV correction when patient is NPO for other reasons.
1. 40 meq/l. is generally safe at usual IV fluid rates.
2. 60 meq/l. can be given when on cardiac monitor.
3. ABSOLUTE UPPER LIMIT FOR K+ ADMINISTRATION RATE:
0.5 meq/Kg./hr. THIS REQUIRES FREQUENT SERUM POTASSIUM LEVELS.
4. NEVER GIVE POTASSIUM AS BOLUS IV INJECTION
5. When potassium level is still dangerously low despite already giving IV fluids with KCl at 60 meq/l., it is probably better to give intermittent bolus doses no more than 0.5 mEq KCl/Kg/hr until the serum K>3.0. Check serum [K]q 1-2 hours when using this replacement approach.
6. Back off on K+ replacement when [K+] is > 3.5.

C. Hyperkalemia
1) Diagnosis
a) Symptoms: weakness, paralysis
b) Physical findings: none specific
c) Lab: serum K+ > 5.5 suspect acidosis
d) EKG findings (in approximate order of appearance as K+ rises): peaked, tall T waves
PR interval lengthens - 1° heart block
QRS, QT lengthens, QRS may blend into T in a diphasic curve
P widens and flattens
R widens and flattens
S deepens
Ectopic rhythms
Ventricular fibrilation
Asystole

2) Treatment of Hyperkalemia
a) Stabilize myocardium
1. $CaCl_2$ – 0.2 – 0.3 cc/Kg of a 10% $CaCl_2$ solution very slow IV push.
Measure serum Ca after each dose to avoid hypercalcemia; serum ionized Ca level is preferred.
2. Na – usually accomplished when $NaHCO_3$ is given.
b) Drive K+ into cells
1. Increase pH if acidosis is present
a. Hyperventilation
b. $NaHCO_3$ (see section on Bicarbonate therapy for dose calculations or give 1 meq/Kg and repeat blood gases).
c. Do not give bicarbonate to patient whose pH is > 7.4.
2. Dextrose plus insulin – most potent treatment for hyperkalemia short of hemodialysis.
a. Add 6 μ reg insulin to 100 cc $D_{25}W$ for a ≅ 1 μ/4 grams dextrose solution; give in 2 cc/Kg IV doses
b. For infants < 6 mos. Give a 1 μ/8 grams dextrose solution to avoid hypoglycemia.
3. Albuterol – continuous inhalation therapy as for RAD: extremely effective. The IV albuterol preparation is not available in the U.S.
c.) Remove K+ from the patient
1. Kayexalate: Na/K exchange resin

a. Give as enema in 20% sorbitol or p.o. as powder mixed in fruit juice
b. Enema is more effective since diarrhea it causes increases K losses.
c. Kayexalate dose: 1 gm/Kg will lower serum K ≅ 0.8 meq/L
d. Tape buttocks to keep kayexalate in at least ½ hour
e. Complications: hypocalcemia
hypomagnesemia
sodium overload
hypokalemia
f. When giving kayexolate p.o. avoid laxatives and antacids

3) Hemodialysis – most effective treatment of hyperkalemia
4) Peritoneal dialysis – may be too slow in very catabolic patients
5) Exchange transfusion (neonates only) using washed or very fresh pRBC's re-suspended in 5% albumin or FFP or saline
6) CVVH – not a good initial treatment for hyperkalemia – start with HD, then continue with CVVH or CVVHD.

VI. Rational Use of Sodium Bicarbonate in the Treatment of Metabolic Acidosis

A. Principles of bicarbonate therapy
1) Do not give as IV push except during CPR
2) Calculate deficit conservatively as follows: Goal: pH . 7.25
a) The blood HCO_3 concentration needed to achieve this pH rarely exceeds 15 mEq/L if patient is capable of maintaining $pCO_2 < 35$ mmHg. (See acid-base nomogram).
b) Bicarbonate deficit = (15 – plasma [HCO_3]) x Wt (kg) x 0.3
Note that bicarbonate given IV distributes acutely in only about ½ the total body water (TBW = Wt (kg) x 0.6). While this calculation underestimates total replacement, it is the only safe approach to acute correction.
3) Give calculated deficit SLOWLY; may give ½ of deficit over 1st hour if pH < 7.15, remainder over 2-3 hours
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B. Monitor with frequent blood gases, (venous or capillary will be as good as arterial for acid-base status monitoring).

C. Complications of bicarbonate therapy
1) Hypokalemia
2) Hypo-ionized-calcemia
3) Hypernatremia
4) Over-shoot metabolic alkalosis
5) Cerebral edema – paradoxial CSF acidosis (?)
6) Increasing pCO_2 in patients with respiratory failure, resulting in worsened acidosis.

Do not treat respiratory acidosis with bicarbonate.
Remember, proceed cautiously and correct slowly once pH > 7.15. Many with metabolic acidosis have generated large amounts of lactic acid which they will metabolize to bicarbonate once they are rehydrated and given glucose and oxygen.

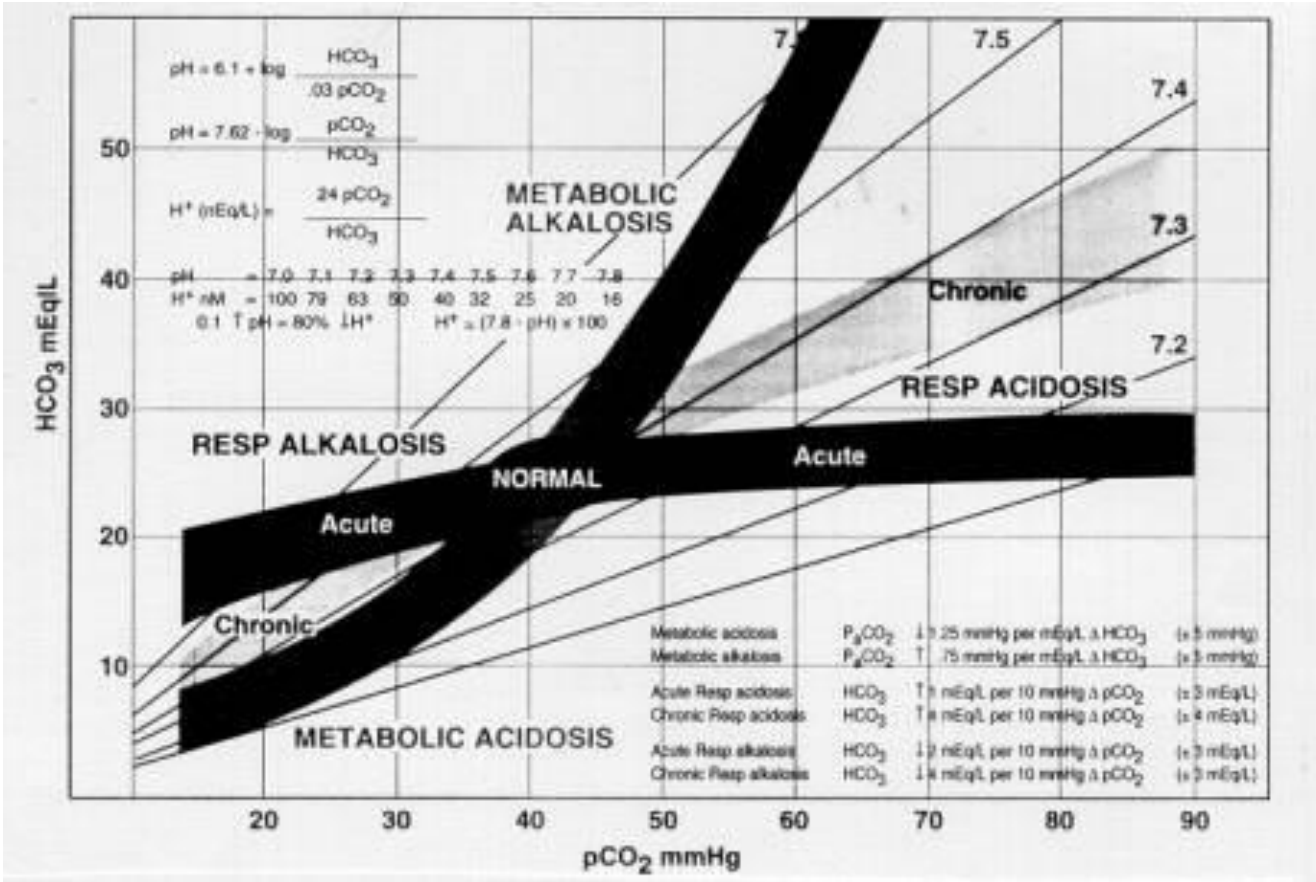

TABLE 10-19

COMPOSITION OF FREQUENTLY USED PARENTERAL FLUIDS

Liquid	CHO (g/100 mL)	Protein* (g/100 mL)	cal/L	Na^+ (mEq/L)	K^+ (mEq/L)	Cl^- (mEq/L)	$HCO_3^{-\dagger}$ (mEq/L)	Ca^{2+} (mEq/L)	mOsm/L
D_5W	5	—	170	—	—	—	—	—	252
$D_{10}W$	10	—	340	—	—	—	—	—	505
NS (0.9% NaCl)	—	—	—	154	—	154	—	—	308
½ NS (0.45% NaCl)	—	—	—	77	—	77	—	—	154
D_5 ¼ NS (0.225% NaCl)	5	—	170	34	—	34	—	—	329
3% NaCl	—	—	—	513	—	513	—	—	1027
8.4% sodium bicarbonate (1 mEq/mL)	—	—	—	1000	—	—	1000	—	2000
Ringer's	0-10	—	0-340	147	4	155.5	—	≈4	
Lactated Ringer's	0-10	—	0-340	130	4	109	28	3	273
Amino acid 8.5% (Travasol)	—	8.5	340	3	—	34	52	—	880
Plasmanate	—	5	200	110	2	50	29	—	
Albumin 25% (salt poor)	—	25	1000	100-160	—	<120	—	—	300
Intralipid‡	2.25	—	1100	2.5	0.5	4.0	—	—	258-284

CHO, Carbohydrate; *HCO_3^-,* bicarbonate; *NS,* normal saline.

*Protein or amino acid equivalent.

†Bicarbonate or equivalent (citrate, acetate, lactate).

‡Values are approximate; may vary from lot to lot. Also contains <1.2% egg-phosphatides.