Abstract

The management of fluid and electrolytes in the extremely low birth weight infant is an ongoing challenge during the first week of life. Each infant is different in their fluid and electrolyte requirements, which may be altered by perinatal events (asphyxia, sepsis) as well as perinatal medications (corticosteroids). A generalized approach to management will be discussed with an emphasis on individual modifications as common problems present during the initial days of life.

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Fluids and Electrolytes Management

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Fluid and electrolyte management in the extremely premature (ELBW) infant patient population is challenging. There is no cookbook approach that can be used for all ELBW infants during the first week of life. However, there can be a generalized management approach for each infant based on presenting problems coupled with an understanding of the ELBW infant's physiologic needs. Each management decision should not only address the infant's immediate fluid and electrolyte needs but should also address future needs and requirements as well. This article will attempt to address options in management of fluid and electrolyte needs of the ELBW infant as they typically present in the first days of life.

Fluid Requirements

The body composition of the ELBW infant is 85 to 90% water of which one-third is intracellular water (ICW) and two-thirds is extracellular water (ECW).^{1,2} The management of the fluid in the extracellular space becomes important in both the immediate management of the infant's volume status as well as how the management will affect the onset of postnatal diuresis and potential subsequent morbidities [intraventricular hemorrhage (IVH), patent ductus arteriosus (PDA), and bronchopulmonary dysplasia (BPD)] related to a delay in diuresis.³⁻⁹ During transition there is a redistribution of body water from the interstitial space to the extracellular space as well as reabsorbed lung fluid, resulting in an expansion of extracellular water volume and an increase in its sodium content.^{10,11} A serum sodium measured at this time before the establishment of postnatal diuresis may be low (130-134 mEq/L), reflecting an expanded extracellular fluid (ECF) volume. Adding sodium at this stage can prevent the postnatal diuresis and further expand the ECF and contribute to edema formation in the periphery as well as the lung.^{11,12} Infants cannot excrete a sodium load as well as an adult even when the fractional excretion of sodium (FENa) is maximized. The physiologic attempt to conserve sodium may be a developmental one in that the preterm infant needs sodium for growth and development.¹²⁻¹⁴ An adaptive response of the kidney is to elicit an antinatriuretic renal tubular response, resulting in an attempt to conserve sodium by the kidneys.¹² The FENa is higher in the preterm infant compared with the term newborn and adult (FENa = 1%). The FENa can be as high as 12.4% during the postnatal diuresis.¹⁰ The high FENa begins to fall as the postnatal diuresis abates followed by weeks of renal sodium conservation.^{11,15} A positive sodium balance is a requirement for growth.^{11,13,14} Exogenous sodium should not be added to intravenous fluids in the first days of life until the postnatal diuresis has been established, which occurs when the urine output drops and the specific gravity is >1.012.^{10,16}

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1527-3369/04/0402-0000\$30.00/0
doi:10.1053/j.nainr.2004.02.002

Six randomized controlled trials compare various fluid intakes during the first days of life and the effects on postnatal weight loss, incidence of PDA, necrotizing enterocolitis (NEC), BPD, IVH, and death.^{17–22} Four of these trials were reviewed by the Cochrane Library.²³ Three of these trials were published in the early 1980s and one was published in 1992.^{17–19} The mean gestational ages of the infants in these trials were 29, 31, and 34 weeks. Low fluid intake in three of the trials varied from 50 to 70 mL/kg/d versus a control group whose intake varied from 80 mL/kg/d (2 trials)^{18,19} to 150 mL/kg/d (1 trial).²⁰ All three trials used humidified incubators described as 50% or maximal humidity. The results of the four trials together demonstrate that restricted fluid intake significantly increases postnatal weight loss and significantly reduces the risks of PDA, NEC, and death. There was a trend toward an increased risk of dehydration and a reduced risk of BPD, however these trends were not of statistical significance.²³ There are two recently published trials that utilized even stricter fluid intakes of 40 mL/kg/d (restricted) versus 60 mL/kg/d (control).^{21,22} The first trial evaluated the effects of different fluid intakes on lung function [functional residual compliance (FRC)] and compliance.²¹ Infants in the restricted fluid group had a higher mean compliance on day three of life; however there was essentially no difference by day seven of life. There was, however, a negative correlation between colloid intake and lung compliance (FRC $P = 0.003$, compliance $P = 0.001$). The second trial utilized the same fluid intakes but evaluated the effects of these intakes on fluid balance, electrolyte, and metabolic adverse effects.²² They found no statistical differences in serum creatinine and arginine vasopressin during the first week of life as well as in the incidence of jaundice, hypotension, hypoglycemia, hypernatremia, or hyponatremia. Infants in the restricted group did have lower mean urine outputs and higher mean urine osmolalities than the control group. It would appear that a restricted fluid intake significantly increases postnatal weight loss and significantly reduces the risk of PDA and NEC without increasing the risk of adverse effects associated with dehydration.

During the time period of diuresis, the management of insensible water loss may be just as important as the management of fluid requirements. The water loss from the epidermis of an ELBW infant can be high due to the high body surface area to body mass ratio (up to 6 times that of an adult) and especially when the skin is gelatinous.^{24,25} It is important to minimize transepidermal water loss especially during the postnatal diuresis to prevent or minimize the incidence of a hyperosmolar extracellular space. The high osmotic pressure of hyperosmolar ECF can lead to a loss of fluid in the intracellular space and

result in intravascular depletion, and subsequent hypotension, hypernatremia, and hyperkalemia.²⁵

Efforts to minimize transepidermal water loss center around evaporative barriers and the use of high humidity (80 to 100%) in the incubator.^{25–32} Use of these barriers is best limited to the first week of life. Petrolatum-based barriers (skin emollients) such as Aquaphor® (Beiersdorf, Inc., Wilton, CT) have been associated with an increased risk for staphylococcus epidermidis infection as well as anecdotal concerns of fungal colonization.^{25,33} To avoid contamination with water-borne bacteria, humidified isolates should be changed routinely or dried thoroughly and the humidity chamber cleaned frequently. The effectiveness of these barriers will have a direct effect on how much exogenous fluid will be required to prevent dehydration and yet prevent overhydration and subsequent peripheral and pulmonary edema, which can interfere with the recovery from respiratory distress syndrome (RDS).

Recovery from RDS

Recovery from RDS is an important event that can determine the clinical course of an infant's time in the NICU. Recovery from RDS does not occur until the postnatal diuresis occurs. Postnatal diuresis is defined as a urine output >80% of volume input (IV fluid administration).³⁴ Diuresis can begin as soon as 12 hours after birth and typically peaks at about 48 hours of age. This diuresis precedes recovery from pulmonary disease.³⁴ Spitzer and coworkers⁸ demonstrated a longer duration of oxygen therapy in infants whose diuresis was delayed beyond 72 to 96 hours. In addition, BPD is more likely to occur in patients who demonstrate a late diuresis. This suggests that there may be a role for pulmonary edema or at least pulmonary fluid accumulation in the pathogenesis of BPD.^{21,25,35–37}

There may also be a relationship between the fluid volume administration during the first days of life and the development of a PDA. Gersony and coworkers⁶ published a large multicenter, collaborative trial on 1,600 infants and the effect of indomethacin in infants with a PDA. The incidence of a clinically significant PDA in infants with birth weights <1,000 g who received >90 mL/kg/d was nearly three times that of infants who received ≤90 mL/kg/d in the first 48 hours of life. Fluid volume restriction seems to be a recurring theme in the management of the ELBW infant for all of the above reasons. Fluids can be maintained at 60 to 80 mL/kg/d during the first days of life even in the smallest infants as long as serum sodium can be maintained <150 mEq/L.^{25,18–23} The successful use of evaporative barriers may determine the success of these lower daily fluid

deliveries. It is important for the infant to demonstrate a postnatal diuresis or extracellular water contraction to allow a timely recovery from RDS and perhaps minimize the incidence and severity of BPD.

Hypotension

The above approach to fluid management may seem reasonable in the “well” ELBW infant, however, the majority of preterm births result in an infant who is not “well.” Normal blood pressure limits for the ELBW infant have not been validated. “Normal” blood pressure values currently are gestational and postnatal age-dependent blood pressure values between the 10th and 90th percentile and have no proven physiologic relevance.³⁸ Acceptable mean arterial blood pressure during the first 12 to 24 hours of life is typically equivalent to the infant’s gestational age. By day three of life most ELBW infants have attained a mean blood pressure of 30 mm Hg.³⁹ If one considers that a normal physiologic blood pressure is one that supports perfusion (oxygen delivery) to the major organs, it is possible that “hypotension” in a *nonacidotic and nonhypoxemic* baby need not be treated immediately, especially in an infant who appears to be well perfused and whose mean arterial pressure continues to rise.^{40,41} Cautious observation and monitoring of this infant may result in the establishment of urine output (postnatal diuresis), assuming that the blood pressure values continue to rise over time. A recent retrospective analysis of serial mean arterial pressure values during the first three days of life in the ELBW infant population suggests that it is important for blood pressure to continue to increase and that between three and six hours of age may be a critical time period to aggressively treat hypotension.⁴² The hypotensive ELBW infant of most concern may be the infant demonstrating *fluctuations* in blood pressure. A recent trial is the first to demonstrate direct evidence that blood pressure *fluctuations* increase the incidence of IVH and periventricular leukomalacia (PVL) in preterm infants with low or absent cerebral blood flow autoregulation and that cerebral oxygenation is dependent on systemic blood pressure.⁴³ In addition, infants who developed severe IVH had significantly lower early right ventricular outputs, which was associated with left to right shunts through larger diameter patent ductus arteriosus and higher mean airway pressures.⁴⁴ Blood pressure is a function of blood flow and systemic vascular resistance.⁴⁵ Due to the dynamic changes in blood flow (ductal and atrial shunts) and systemic vascular resistance after birth, blood pressure may not be a good measure of cardiovascular status.^{44–46} Superior vena cava (SVC) flow may be a marker of systemic blood flow.⁴⁶ SVC flow represents flow to the upper body

of which 80% goes to the brain and is not affected by ductal and atrial shunting.^{46,47} Kluckow and Evans⁴⁶ measured SVC flow in infants 26 to 29 weeks during the first 48 hours of life to determine reproducibility and establish normal ranges. They found that an increase in SVC flow occurred during the first 48 hours of life, possibly as a result of improved myocardial function. A follow up study measured SVC flow and the association between hypoperfusion and development of IVH during the first 48 hours of life.⁴⁴ Their hypothesis was that the incidence of IVH relates to systemic and cerebral hypoperfusion–reperfusion cycle resulting from slow postnatal adaptation of the immature cardiovascular system. SVC flow was measured at 5, 12, 24, and 48 hours of age. Measurement of lowest SVC flow occurred at 5 and 12 hours of age in 80% of the studied infants (n = 126) but improved spontaneously over time. Low SVC flow was poorly predicted by mean arterial pressure (MAP). There was a strong association between low flow rates and subsequent IVH as the flow improved. The grade of IVH related significantly to severity and duration of low SVC flow. Low flow rates were significantly associated with lower gestation, higher upper body vascular resistance, larger diameter ductal shunts, and higher mean airway pressures.^{44,46} At the 5-hour echocardiogram, 50% of the ducts had constricted, but the other 50% were still hemodynamically significant. A recent randomized trial studied the effects of high frequency ventilation (HFOV) compared with conventional ventilation on SVC flow during the first 24 hours of life.⁴⁸ There were no significant effects on SVC flow associated with HFOV (higher MAP), however, there was a trend toward increased incidence of borderline SVC flows (<50 mL/kg/min) in the infants on HFOV with an increasing trend toward increased use of cardiovascular support. As a part of the study design, any infant with an SVC flow <50 mL/kg/min received dobutamine and, if that failed to increase the SVC flow, dopamine was added to therapy. The use of inotropes in this study does not duplicate current clinical practice, as SVC flow is not routinely measured. The previous studies highlight interesting observations and intriguing possibilities into the accurate assessment of systemic organ perfusion and as well as subsequent therapies to maximize organ perfusion in a timely manner after birth to minimize neurologic morbidity.

For any infant with hypotension, the cause needs to be determined so that the appropriate treatment may be initiated. The development of persistent hypotension may be the first physiologic indicator that an infant may be infected or entering into the uncompensated phase of shock.⁴⁹ The birth history can give us additional information as to the etiology of the hypotension (birth asphyxia and/or maternal infection). Birth asphyxia can result in

hypoxemia and metabolic acidosis resulting in peripheral vasodilatation and myocardial dysfunction (poor contractility). These events, which may be coupled with an inflammatory response due to infection, can result in fluid accumulation in the periphery (tissue edema).^{25,49} Management approaches should be centered around efforts to minimize further development of tissue edema without excessive fluid administration, correct metabolic acidosis, minimize peripheral vasodilatation, and augment cardiac contractility with the administration of pressors.

Within the first few hours of birth, the most common causes of hypotension are related to immature vasoregulation and myocardial dysfunction, sometimes referred to as “distributive shock.”^{49,50} Hypovolemia does not appear to be a common cause of hypotension in the newly born and yet most infants receive at least one course of volume administration to treat hypotension.⁴⁹ There appears to be no relationship between blood pressure and blood volume and dopamine is at least twice as effective as volume administration in treating hypotension in the first hours after birth.^{49,51} One may argue that, since objective assessment of intravascular volume is not practical at the bedside, and there is evidence that up to 23% of preterm infants may be hypovolemic, administration of a single bolus of 10 to 20 mL/kg of volume may not be unreasonable in a hypotensive patient without evidence of acute volume loss.⁴⁰ However, in view of recent evidence that the severity and duration of low SVC flow correlates with more severe grades of IVH, it may be prudent to initiate pressor support as a first line therapy for hypotension in patients in high-risk categories for IVH or to begin volume support and pressor support at the same time to augment cardiovascular function. In older infants (28 to 29 weeks’ gestational age) at lesser risk without other IVH risk factors, if normal blood pressure is not achieved after a single volume bolus, then dopamine should be initiated.^{40,41,49} The type of volume, colloid versus crystalloid, can also be of some controversy. However, it has been demonstrated that it is the volume infused, not the protein content of the fluid that is important in sustaining blood pressure.^{45,49} Normal saline (0.9% sodium chloride) is as effective as 5% albumin in increasing blood pressure and causes less fluid retention after its administration.^{49,52,53} The administration of 5% albumin can impair gas exchange due to leaking of protein in patients with increased capillary permeability such as the infected or asphyxiated infant.^{21,49,53} Due to this permeability, albumin is not preferentially retained in the intravascular space and could cause a shift of fluid out of the intravascular space.^{49,54} The sudden shift in fluid could potentiate brain injury or lead to IVH. In a patient in whom there is an identifiable volume loss (preload), the volume should be replaced with the type of fluid that was lost, such as blood with packed

red blood cells, platelets, or fresh frozen plasma (fibrinogen). The circulatory capacity for preload is estimated to be between 30 to 50 mL/kg in the premature infant and can guide the total volume of fluid administered for volume expansion.²⁵ A common mistake in fluid management is to increase the maintenance fluids to facilitate urine output. However, it is likely that fluids should remain restricted until kidney function and circulation are established to prevent further tissue edema from occurring.²⁵ Birth is associated with renal innervation stimulation that can increase renal vascular resistance (sympathetic stimulation), which results in a suppressed glomerular filtration rate (GFR) and urine output (prediuretic phase first 12 hours of life). As the ECF volume expands, there is an inhibition of sympathetic activity and subsequent decreased renal vascular resistance resulting in increased urine output (diuretic phase).¹⁰

One study has retrospectively evaluated the relationship between mean blood pressure and neonatal death and morbidity in infants <1,500 g and found no relationship between mean blood pressure, death, and PVL. However, there was a relationship between IVH and MAP lows, highs, and extremes in mean blood pressure variability.^{39,55} Early and effective treatment of hypotension to obtain a stable cardiovascular status may increase the chance of improved neurological outcome and survival.⁴⁹

Indomethacin

Indomethacin is a nonsteroidal antiinflammatory drug used commonly in the NICU to chemically close a symptomatic PDA or as prophylaxis to prevent IVH. The primary mechanism of action is the inhibition of cyclooxygenase, which results in a decrease in circulating prostaglandins, which are potent vasodilators. Inhibition of these prostaglandins can alter filtration pressure within the kidney, increasing the risk for renal failure. The newborn kidney has a low GFR, maintained by a balance of vasoconstriction and vasodilatation. This balance is very important in a kidney with minimal renal blood supply resulting from low mean arterial blood pressure and a high intrarenal vascular resistance.¹¹ Because of these characteristics, the newborn kidney is vulnerable to any form of stress (sepsis, anoxia, nephrotoxic drugs). When these stresses occur in combination and sometimes solo, the result can be oliguric renal failure, which is reversible but presents its own morbidities and management challenges. Dopamine may have some benefit in preventing or mitigating the detrimental effects of indomethacin on the kidney. Dopamine increases total renal blood flow and GFR via stimulation of dopamine receptors in the renal vasculature.^{39,56,57} It also increases blood pressure by in-

creasing peripheral vascular resistance. These characteristics of dopamine maintain perfusion pressure within the kidney and may mitigate the adverse effects of indomethacin on the kidney.⁵⁷

Nonoliguric Hyperkalemia

Nonoliguric hyperkalemia is not uncommon in some ELBW infants during the first few days of life even in the absence of exogenous potassium intake. A postnatal diuresis may be established and yet the serum potassium continues to rise to above 5 to 6 mEq/L. Recent data suggest this phenomenon may have multiple etiologies, most of which are centered on renal tubular function. Na^+/K^+ ATPase (sodium/potassium adenosine triphosphatase) is an enzyme protein that is important in the tubular handling of sodium and water, as well as in blood pressure.¹¹ There are significantly lower amounts of Na^+/K^+ ATPase activity in the erythrocytes of ELBW infants who develop hyperkalemia versus matched infants who do not develop hyperkalemia.⁵⁸ Gruskay and colleagues⁵⁸ showed that infants with hyperkalemia had a normal GFR, but a higher fractional sodium excretion and diminished potassium secretion in the distal tubule. In addition there also appears to be a shift of potassium from the intracellular space to the extracellular space. Increased extracellular potassium coupled with diminished excretion would likely increase the risk for hyperkalemia. Infants at a higher risk for hyperkalemia are those with severe bruising, IVH, or any other event that may require the infant to break down red blood cells. Omar and coworkers⁵⁹ evaluated the effect of prenatal steroids on potassium balance in the ELBW infant. The peak serum potassium was significantly lower in the prenatal steroid group (5.3 ± 0.2 versus 6.2 ± 0.4 mMol/L) with similar potassium excretion rates and zero potassium intake in both groups. They concluded that prenatal steroids upregulate Na^+/K^+ ATPase in the fetus. The prenatal steroid group also had a significantly less negative potassium balance at the end of the first week of life (-1 versus -7 mMol/kg). The authors concluded that the difference in negative potassium balance could be accounted for by a stabilization of cell membrane (greater Na^+/K^+ ATPase) that may result in a decrease in potassium from the intracellular space to the extracellular space. The recent widespread use of prenatal steroids may explain the decreased incidence of nonoliguric hyperkalemia. Potassium should not be added to an infant's parenteral fluid until serum potassium levels have returned to normal range or have begun to demonstrate a decreasing trend within the normal serum limit range. When this occurs, potassium should be added at a dose of 1 mEq/kg/d.

Importance of Sodium Chloride Administration and Onset of Late Hyponatremia of Prematurity

Previously, we have discussed the importance of limiting the amount of exogenous sodium chloride administered to the infant to enhance the onset of the postnatal diuresis. Now we will discuss the role of sodium chloride after the postnatal diuresis. So when is the optimal time to add sodium to the parenteral fluids? It has been suggested that sodium can be added after the postnatal diuresis is established, yet others suggest waiting until there is a documented weight loss. When a diuresis is established it may take a day or two until significant weight loss is assessed via a scale.¹⁰ Adding sodium or increasing fluids before a documented weight loss could delay the loss of ECF volume. Hartnoll and coworkers¹³ randomized preterm infants 25 to 30 weeks to receive sodium 4 mMol/kg/d beginning either on the second day after birth (early) or when weight loss of 6% of birth weight (delayed) had been achieved. They measured sodium intake, total fluid intake, calorie intake, urine volume, and urine sodium excretion. On days 1, 7, and 14, total body water was measured and on days 1 and 14, ECF volume was measured. There were no differences between the groups on day one in body water compartments. In the delayed group there was a significant loss of total body water during the first week of life compared with the early group (delayed -44 mL/kg, $P = 0.048$; early 6 mL/kg, $P = 0.97$). By day 14 of life the delayed group also had a significant loss in ECF volume (delayed -53 mg/kg, $P = 0.01$; early -37 mL/kg, $P = 0.2$). These changes resulted in a significant difference in body composition by the end of the first week of life (delayed TBW = 791 mL/kg; early TBW 849 mL/kg, $P = 0.013$).¹³ A small change in the prescribing of sodium supplements in the preterm infant had measurable effects on body composition by the end of the first week of life. This study shows the importance of individualizing clinical management of sodium supplementation in the preterm infant during postnatal adaptation. Routine sodium supplementation should not occur until there is both a postnatal loss of body water as well as weight loss of at least 6% of birth weight. This again emphasizes the importance of restrictive management of exogenous fluids during the first days of life as well optimal timing of the administration of sodium.

There is also concern that delaying sodium administration may compromise postnatal growth. The amount of intracellular fluid can be taken as a measure of cell mass (body solids) so that increasing ICF volume indicates cell growth (TBW-ECW = ICW). Singhi and coworkers⁶⁰ showed a gradual increase in body solids up to the time of regaining birth weight that was associated with a fall in

TBW and ECW and an increase in ICW, suggesting growth can begin immediately after birth. It would appear that the loss of ECW and TBW is an important postnatal adaptation.

Adequate sodium supplementation may also have a role in neurodevelopmental outcomes. Al-Dahhan and coworkers¹⁴ assessed outcomes in infants ≤ 33 weeks fed a low (1-1.5 mMol sodium/d) or supplemented sodium (4-5 mMol sodium/d) diet during the first 14 days of life and evaluated these patients at 10 to 13 years of age. Outcomes assessed were motor function, assessment of intelligence, memory and learning, language and executive skills, and behavior. Children in the supplemented group performed better in all modalities than those from the unsupplemented group.¹⁴ Lucas and coworkers also demonstrated a difference in children who were fed their mothers' milk or enriched preterm formula versus standard term formula.^{61,62} The enriched group had significantly higher verbal performance full scale IQs than those fed standard formula. Sodium is required for growth and development of the central nervous system. Sodium should be supplemented as soon as a weight loss of 6% occurs and should not be delayed until a drop in serum sodium is appreciated. Early and adequate sodium supplementation may prevent or at least mitigate the onset and severity of late hyponatremia of prematurity.²⁵ Sodium supplementation to the IV fluids should begin at 2 to 3 mEq/kg/d and advance as necessary. Small for gestational age (SGA) infants have higher requirements as they have greater nutritional needs. SGA infants will require a minimum of 4 to 5 mEq/kg/d of sodium added to their IV fluids and may require enteral sodium supplementation once full feeds are attained.²⁵

Conclusion

There are many clinical aspects to consider when determining an optimal fluid and electrolytes prescription for the ELBW infant. There are a few general guidelines that can be recommended:

1. Fluids should be initiated at 60 to 80 mL/kg/h. Infants initiated at >90 mL/kg/hr had three times the incidence of clinically significant PDA. Fluid administration above an infant's requirement can delay postnatal diuresis and subsequent recovery from RDS.
2. All fluids including fat emulsion and medication continuous infusions should be documented in the total daily input. Intermittently dosed medications and flushes can add clinically significant contributions to the total fluid intake. Documentation of medication and flush volumes on the nursing flow sheet will assist in determination of the total volume administered.
3. Consider prescribing a total IV rate for instances when rates

of continuous infusions of medications can vary throughout the day. This can prevent the unintentional delivery of additional fluid. It is important to calculate glucose infusion rates during this time, especially when there is a rapid wean of pressor support in the infant recovering from shock. This can prevent the incidence of unintentional hypo- or hyperglycemia.

4. Fluid restriction should be maintained until the postnatal diuresis occurs and at least a 6% weight loss is documented. This may require weight assessment every 12 hours to evaluate changing fluid requirements. This can be especially important in the infant in shock to minimize peripheral edema formation.
5. Initiation of an evaporative barrier from birth to about day seven of life or until the skin has cornified. A humidified incubator may be most effective in the prevention of trans-epidermal water loss and subsequently allow lower exogenous fluid administration.
6. Fluids should be calculated on the birth weight until day seven of life or when the skin has adequately keratinized and a humidified incubator or evaporative barrier is no longer considered necessary. The use of a common denominator (weight) on a daily basis eases the assessment of fluid requirements.
7. Correct metabolic acidosis. Cardiovascular status will remain unstable until this is accomplished.
8. Infants whose mothers did not receive prenatal steroids may be at a higher risk of nonoliguric hyperkalemia.
9. Hypotension defined as a mean blood pressure less than the 10th percentile (GA = 25 weeks, 10th percentile MAP = 25 mm Hg) should be carefully evaluated. Probable etiology of the hypotension will guide initial therapy. A nonacidotic, nonhypoxemic infant who appears well perfused may not need immediate intervention assuming the MAP continues to rise. If the blood pressure does not show a consistent rise or is labile, dopamine may be the treatment of choice to assist in the stabilization of the cardiovascular status and possibly to facilitate organ flow/perfusion (SVC flow). For patients who have a documented reason for volume loss, volume should be administered in the form in which it was lost (packed red cells or fresh frozen plasma). For the $\sim 20\%$ of infants who may be hypovolemic, a single dose of volume in the form of crystalloid (0.9% normal saline) may be administered, however, if ineffective in raising the blood pressure, dopamine should be initiated or, if currently administered, the dose should be increased. The goal of the treatment of hypotension/shock is to attain cardiovascular stability as soon as possible.

The above suggestions in fluid and electrolyte management are guidelines and should be modified to meet individual patient needs and to conform to various clinical situations.

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Objectives: At the end of this article the reader will be able to: 1) Describe the basic fluid requirements of the very low birthweight infant. 2) Discuss how certain conditions such as Respiratory Distress Syndrome (RDS) affects fluid management. 3) Outline key elements of an infants history, disease course, and medications that impact on fluid and electrolyte management.

Article: Fluid & Electrolyte Management

- The percentage of body composition of the extremely low birth weight (ELBW) infant that is composed of water is
 - 65-70
 - 75-80
 - 85-90
- The compartment that contains the majority of water in the ELBW infant is the
 - extracellular
 - intracellular
 - intravascular
- During transition to extrauterine life and prior to establishment of the postnatal diuresis, serum sodium in the ELBW infant is expected to be
 - decreased (<135 mEq/L)
 - elevated (>145 mEq/L)
 - normal (135-145 mEq/L)
- Compared to the term infant, the fractional excretion of sodium (FENa) in the preterm infant is
 - higher
 - lower
 - the same
- Exogenous sodium should not be added to intravenous fluids in the ELBW infant until
 - forty-eight (48) hours of age
 - postnatal diuresis has been established
 - weight loss equals 10% of birth weight
- According to clinical trials comparing various fluid intakes in the first days of life in ELBW infants, the risk for a patent ductus arteriosus (PDA) in the presence of a restricted fluid intake is
 - decreased
 - increased
 - unchanged
- Use of barriers to decrease insensible water loss in preterm infants should be limited to day of life
 - 7
 - 14
 - 21
- Postnatal diuresis is defined as urine output that exceeds what percentage of the intravenous fluid administered?
 - 20
 - 50
 - 80
- The postnatal age at which postnatal diuresis peaks in the ELBW infant is
 - 24 hours
 - 36 hours
 - 48 hours
- Delayed postnatal diuresis is linked with an increased incidence of
 - bronchopulmonary dysplasia (BPD)
 - intraventricular hemorrhage (IVH)
 - necrotizing enterocolitis (NEC)
- According to a recent retrospective study of hypotension in the ELBW infant, the critical time period for aggressive treatment of hypotension is
 - between three and six hours of postnatal age
 - immediately after birth during resuscitation
 - when the mean blood pressure is less than the gestational age
- According to the article, the best marker of systemic blood flow is
 - blood pressure
 - cerebral blood flow
 - superior vena cava blood flow
- The most common cause of hypotension in newborns shortly after birth is
 - cardiogenic shock
 - distributive shock
 - hypovolemic shock

14. According to the article, the most effective treatment of hypotension in the first hours after birth is the administration of
 - A. dobutamine
 - B. dopamine
 - C. fluids
15. The most important factor in sustaining blood pressure in the ELBW infant who is undergoing fluid resuscitation is
 - A. protein content of the fluid used
 - B. speed of administration of the fluid
 - C. volume of fluid infused
16. The preload circulatory capacity of the preterm infant is
 - A. 10-30 cc/kg
 - B. 30-50 cc/kg
 - C. 50-70 cc/kg
17. The decreased incidence of non-oliguric hyperkalemia in ELBW infants is attributed to widespread use of
 - A. antenatal steroids
 - B. double-walled incubators
 - C. topical emollients
18. Routine sodium supplementation in the ELBW infant should occur after the postnatal diuresis and when the infant's weight loss from birth weight is at least
 - A. 6%
 - B. 10%
 - C. 14%
19. The minimum amount of sodium to be added to the intravenous fluids of small for gestational age (SGA) infants is
 - A. 2-3 mEq/kg/day
 - B. 4-5 mEq/kg/day
 - C. 6-7 mEq/kg/day
20. The risk of developing a clinically significant patent ductus arteriosus (PDA) increases when the intravenous fluids are provided at a rate of
 - A. 60 ml/kg/day
 - B. 75 ml/kg/day
 - C. 90 ml/kg/day