The goal of dialysis for patients with chronic renal failure is to restore the composition of the body’s fluid environment toward normal. This is accomplished principally by formulating a dialysate whose constituent concentrations are set to approximate normal values in the body. Over time, by diffusional transfer along favorable concentration gradients, the concentrations of solutes that were initially increased or decreased tend to be corrected. When an abnormal electrolyte concentration poses immediate danger, the dialysate concentration of that electrolyte can be set at a nonphysiologic level to achieve a more rapid correction. On a more chronic basis, the composition of the dialysate can be individually adjusted in order to meet the specific needs of each patient.

**Dialysate Composition for Hemodialysis**

In the early days of hemodialysis, the dialysate sodium concentration was deliberately set low to avoid problems of chronic volume overload such as hypertension and heart failure. As volume removal became more rapid because of shorter dialysis times, symptomatic hypotension emerged as a common and often disabling problem during dialysis. It soon became apparent that changes in the serum sodium concentration—and more specifically changes in serum osmolality—were contributing to the development of this hemodynamic instability. A decline in plasma osmolality during regular hemodialysis favors a
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fluid shift from the extracellular space to the intracellular space, thus exacerbating the volume-depleting effects of dialysis. With the advent of high-clearance dialyzers and more efficient dialysis techniques, this decline in plasma osmolality becomes more apparent, as solute is removed more rapidly.

Use of dialysate of low sodium concentration would tend further to enhance the intracellular shift of fluid, as plasma tends to become even more hyposmolar consequent to the movement of sodium from plasma to dialysate. The use of a higher sodium concentration dialysate (>140 mEq/L) has been among the most efficacious and best tolerated therapies for episodic hypotension [1-3].

The high sodium concentration prevents a marked decline in the plasma osmolality during dialysis, thus protecting the extracellular volume by minimizing osmotic fluid loss into the cells.

In the early 1960s acetate became the standard dialysate buffer for correcting uremic acidosis and offsetting the diffusive losses of bicarbonate during hemodialysis. Over the next several years reports began to accumulate that linked routine use of acetate with cardiovascular instability and hypotension during dialysis. As a result, dialysate containing bicarbonate began to re-emerge as the principal dialysate buffer, especially as advances in biotechnology made bicarbonate dialysate less expensive and less cumbersome to use. For the most part, the bicarbonate concentration used consistently in most dialysis centers is 35 mmol/L. Emphasis is now being placed on individually adjusting the dialysate bicarbonate concentration so as to maintain the predialysis tCO2 concentration above 23 mmol/L [12-16]. Increasing evidence suggests that correction of chronic acidosis is of clinical benefit in terms of bone metabolism and nutrition.

Dialysis assumes a major role in the maintenance of a normal serum potassium concentration in patients with end-stage renal disease. Excess potassium is removed by using a dialysate with a lower potassium concentration, so that a gradient is achieved that favors movement of potassium. In general, one can expect only up to 70 to 90 mEq of potassium to be removed during a typical dialysis session. As a result, one should not overestimate the effectiveness of dialysis in the treatment of severe hyperkalemia. The total amount removed varies considerably and is affected by changes in acid-base status, in tonicity, in glucose and insulin concentration, and in catecholamine activity [17-20].

The concentration of calcium in the dialysate has implications for metabolic bone disease and hemodynamic stability. Like the other constituents of the dialysate, the calcium concentration should be tailored to the individual patient [21]. Some data suggest that lowering the dialysate calcium concentration would exacerbate hemodynamic instability during the dialysis procedure [21]. In this regard, the intradialysis drop in blood pressure noted in patients dialyzed against a low-calcium bath, while statistically significant, is minor in degree [22,23]. Nevertheless, for patients who are prone to intradialysis hypotension avoiding low calcium dialysate concentration may be of benefit. On the other hand, the use of a lower calcium concentration in the dialysate allows the use of increased doses of calcium-containing phosphate binders and lessens dependence on binders containing aluminum. In addition, use of 1,25-dihydroxyvitamin D can be liberalized to reduce circulating levels of parathyroid hormone and, thus, the risk of inducing hypercalcemia. With dialysate calcium concentrations below 1.5 mmol/L, however, patients need close monitoring to ensure that negative calcium balance does not develop and that parathyroid hormone levels remain in an acceptable range [24].

Dialysate Composition for Peritoneal Dialysis

To meet the ultrafiltration requirements of patients on peritoneal dialysis, the peritoneal dialysate is deliberately rendered hyperosmolar relative to plasma, to create an osmotic gradient that favors net movement of water into the peritoneal cavity. In commercially available peritoneal dialysates, glucose serves as the osmotic agent that enhances ultrafiltration. Available concentrations range from 1.5% to 4.25% dextrose. Over time, the osmolality of the dialysate declines as a result of water moving into the peritoneal cavity and of absorption of dialysate glucose. The absorption of glucose contributes substantially to the calorie intake of patients on continuous peritoneal dialysis. Over time, this carbohydrate load is thought to contribute to progressive obesity, hypertriglyceridemia, and decreased nutrition as a result of loss of appetite and decreased protein intake. In addition, the high glucose concentrations and high osmolality of currently available solutions may have inhibitory effects on the function of leukocytes, peritoneal macrophages, and mesothelial cells [25]. In an attempt to develop a more physiologic solution, various new osmotic agents are now under investigation. Some of these may prove useful as alternatives to the standard glucose solutions. Those that contain amino acids have received the most attention.

The sodium concentration in the ultrafiltrate during peritoneal dialysis is usually less than that of extracellular fluid, so there is a tendency toward water loss and development of hyponatremia. Commercially available peritoneal dialysates have a sodium concentration of 132 mEq/L to compensate for this tendency toward dehydration. The effect is more pronounced with increasing frequency of exchanges and with increasing dialysate glucose concentrations. Use of the more hypertonic solutions and frequent cycling can result in significant dehydration and hyponatremia. As a result of stimulated thirst, water intake and weight may increase, resulting in a vicious cycle.

Potassium is cleared by peritoneal dialysis at a rate similar to that of urea. With chronic ambulatory peritoneal dialysis and 10 L of drainage per day, approximately 35 to 46 mEq of potassium is removed per day. Daily potassium intake is usually greater than this, yet significant hyperkalemia is uncommon in these patients. Presumably potassium balance is maintained by increased colonic secretion of potassium and by some residual
renal excretion. Given these considerations, potassium is not routinely added to the dialysate.

The buffer present in most commercially available peritoneal dialysate solutions is lactate. In patients with normal hepatic function, lactate is rapidly converted to bicarbonate, so that each mM of lactate absorbed generates one mM of bicarbonate. Even with the most aggressive peritoneal dialysis there is no appreciable accumulation of circulating lactate. The rapid metabolism of lactate to bicarbonate maintains the high dialysate-plasma lactate gradient necessary for continued absorption. The pH of commercially available peritoneal dialysis solutions is purposely made acidic by adding hydrochloric acid to prevent dextrose from caramelizing during the sterilization procedure. Once instilled, the pH of the solution rises to values greater than 7.0. There is some evidence that the acidic pH of the dialysate, in addition to the high osmolality, may impair the host’s peritoneal defenses [25,26].

To avoid negative calcium balance—and possibly to suppress circulating parathyroid hormone—commercially available peritoneal dialysis solutions evolved to have a calcium concentration of 3.5 mEq/L (1.75 mmol/L). This concentration is equal to or slightly greater than the ionized concentration in the serum of most patients. As a result, there is net calcium absorption in most patients treated with a conventional chronic ambulatory peritoneal dialysis regimen. As the use of calcium-containing phosphate binders has increased, hypercalcemia has become a common problem when utilizing the 3.5 mEq/L calcium dialysate. This complication has been particularly common in patients treated with peritoneal dialysis, since they have a much greater incidence of adynamic bone disease than do hemodialysis patients [27]. In fact, the continual positive calcium balance associated with the 3.5-mEq/L solution has been suggested to be a contributing factor in the development of this lesion. The low bone turnover state typical of this disorder impairs accrual of administered calcium, contributing to the development of hypercalcemia. As a result, there has been increased interest in using a strategy similar to that employed in hemodialysis, namely, lowering the calcium content of the dialysate. This strategy can allow increased use of calcium-containing phosphate binders and more liberal use of 1,25-dihydroxyvitamin D to effect decreases in the circulating level of parathyroid hormone. In this way, development of hypercalcemia can be minimized.

**Dialysate Na in Hemodialysis**