Dialysis as Treatment of End-Stage Renal Disease

INDICATIONS AND CONTRAINDICATIONS FOR USE OF SODIUM MODELING (HIGH/LOW PROGRAMS)

Indications
- Intradialysis hypotension
- Cramping
- Initiation of hemodialysis in setting of severe azotemia
- Hemodynamic instability (e.g., intensive care setting)

Contraindications
- Intradialysis development of hypertension
- Large interdialysis weight gain induced by high-sodium dialysate
- Hypernatremia

Use of a low-sodium dialysate is more often associated with intradialysis hypotension as a result of several mechanisms. The drop in serum osmolality as urea is removed leads to a shift of water into the intracellular compartment that prevents adequate refilling of the intravascular space. This intracellular movement of water, combined with removal of water by ultrafiltration, leads to contraction of the intravascular space and contributes to the development of hypotension. High-sodium dialysate helps to minimize the development of hypo-osmolality. As a result, fluid can be mobilized from the intracellular and interstitial compartments to refill the intravascular space during volume removal. Other potential mechanisms whereby low-sodium dialysate contributes to hypotension are indicated. Na—sodium; BUN—blood urea nitrogen; PGE2—prostaglandin E2.

Dialysate Buffer in Hemodialysis

ACID CONCENTRATE

- NaCl
- CaCl
- KCl
- MgCl
- Acetic acid
- Dextrose

NaHCO3 CONCENTRATE

- NaHCO3

PURE H2O

- H2O

Final dialysate

- Na 137 mEq/L
- Cl 105 mEq/L
- Ca 3.0 mEq/L
- Acetate 4.0 mEq/L
- K 2.0 mEq/L
- HCO3 33 mEq/L
- Mg 0.75 mEq/L
- Dextrose 200 mg/dl

MECHANISMS BY WHICH ACETATE BUFFER CONTRIBUTES TO HEMODYNAMIC INSTABILITY

- Directly decreases peripheral vascular resistance in approximately 10% of patients
- Stimulates release of the vasodilator compound interleukin 1
- Induces metabolic acidosis via bicarbonate loss through the dialyzer
- Produces arterial hypoxemia and increased oxygen consumption
- Decreased myocardial contractility

There has been interest in varying the concentration of sodium (Na) in the dialysate during the dialysis procedure so as to minimize the potential complications of a high-sodium solution and yet retain the beneficial hemodynamic effects. A high sodium concentration dialysate is used initially and progressively the concentration is reduced toward isotonic or even hypotonic levels by the end of the procedure. The concentration of sodium can be reduced in a linear, exponential, or step pattern. This method of sodium control allows for a diffusive sodium influx early in the session to prevent a rapid decline in plasma osmolality secondary to efflux of urea and other small-molecular weight solutes. During the remainder of the procedure, when the reduction in osmolality accompanying urea removal is less abrupt, the dialysate is sodium level is set lower, thus minimizing the development of
hypertonicity and any resultant excessive thirst, fluid gain, and hypertension in the interdialysis period. In some but not all studies, sodium modeling has been shown to be effective in treating intradialysis hypotension and cramps [5-11].

Indications and contraindications for use of sodium modeling (high/low programs). Use of a sodium modeling program is not indicated in all patients. In fact most patients do well with the dialysate sodium set at 140 mEq/L. As a result the physician needs to be aware of the benefits as well as the dangers of sodium remodeling.

**FIGURE 2-3**

**FACTORS RELATED TO DIALYSIS THAT AFFECT DISTRIBUTION OF POTASSIUM BETWEEN CELLS AND THE EXTRACELLULAR FLUID**

Factors that enhance cell potassium uptake
- Insulin
- \( \beta_2 \)-adrenergic receptor agonists
- Alkalemia

Factors that reduce cell potassium uptake or increase potassium efflux
- \( \beta_2 \)-adrenergic receptor blockers
- Acidity (mineral acidosis)
- Hypertonicity
- \( \alpha \)-adrenergic receptor agonists

**FIGURE 2-4**

The current utilization of a bicarbonate dialysate requires a specially designed system that mixes a bicarbonate and an acid concentrate with purified water. The acid concentrate contains a small amount of lactic or acetic acid and all the calcium and magnesium. The exclusion of these cations from the bicarbonate concentrate prevents the precipitation of magnesium and calcium carbonate that would otherwise occur in the setting of a high bicarbonate concentration. During the mixing procedure the acid in the acid concentrate reacts with an equimolar amount of bicarbonate to generate carbonic acid and carbon dioxide. The generation of carbon dioxide causes the pH of the final solution to fall to approximately 7.0–7.4. The acidic pH and the lower concentrations in the final mixture allow the calcium and magnesium to remain in solution. The final concentration of bicarbonate in the dialysate is approximately 33–38 mmol/L.
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Mechanisms by which acetate buffer contributes to hemodynamic instability. Although bicarbonate is the standard buffer in use today, hemodynamically stable patients can be dialyzed safely using as acetate-containing dialysis solution. Since muscle is the primary site of metabolism of acetate, patients with reduced muscle mass tend to be acetate intolerant. Such patients include malnourished and elderly patients and women.

Dialysate Potassium in Hemodialysis