

Fluid and electrolyte management following open heart surgery

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Maintenance of correct fluid and electrolyte balance is important for the following reasons: (1) provision for optimal volemic status, which is a necessary prerequisite for stable hemodynamics; (2) maintenance of appropriate renal function; (3) optimization of electrolyte configuration of body fluids; and (4) achieving all above without increasing extravascular lung water or precipitating congestive heart failure.

Several factors affect the balance of body fluids and electrolytes following open heart surgery. (1) Amount of intraoperative fluid intake. This varies significantly from center to center. Criteria used for amount of and type of fluid used intraoperatively are not always supported by hard data. (2) The type of fluid used to prime the pump plays an important role, e.g., use of clear fluid instead of blood, use of mannitol, use of buffers.¹⁻³ (3) Loss of albumin from circulation during the perioperative period would affect the oncotic pressure of the plasma.⁴ (4) Inappropriate ADH production occurring during and following major surgery or in response to some anesthetic agents. (5) The impact of positive pressure ventilation on cardiac output, renal blood flow, and ADH production.

Quantitative consideration

1. Left ventricular filling pressure. Measured by left atrial pressure (LAP) or pulmonary capillary wedge pressure (PCWP) is a valuable indicator. Pressure of 8 to 10 mm Hg is appropriate for patients with no left ventricular failure, otherwise the pressure of 15 to 18 mm Hg is probably optimal. In the low output syndrome a combination of fluid intake and vasodilators could be helpful. Fluid intake resulting in left ventricular filling pressure higher than 16 to 18 mm Hg could be advocated by some at the risk of reducing subendocardial perfusion.

2. Cardiac index. Optimal cardiac index should be ≥ 2.2 L/m²/min. Low cardiac index associated with low LAP indicates the need for more volume up to a pressure of 15 to 18 mm Hg. Low cardiac index associated with high filling pressure could respond to volume and vasodilators or vasodilators and dopaminergic drugs.

3. Urine output. Empirically, one should expect 0.5 ml of urine per hour for every milliliter of fluid intake. In the first 24 to 48 hours urine volume is less than that above because of third spacing. After 48 hours, urine output is more than the figure shown above because of mobilization of extravascular fluid. Diagnostic fine tuning of above variations can be achieved by taking into account LAP/PCWP, serum osmolality, serum electrolyte data, and free water clearance.⁵ It is important to remember that low urine output in the immediate postoperative period is not always due to hypovolemia.

4. P_aO₂, alveoloarterial oxygen difference (A-aDO₂), shunt fraction Q_s/Q_t. Deterioration of pulmonary profile could indicate increasing interstitial pulmonary water and should indicate reduction of fluid intake or use of di-

uretics or both provided that hemodynamics and renal function are not too precarious.

Qualitative considerations

1. Albumin. Controversy over the use of albumin continues. The function of albumin is to help shift excess fluid from extravascular to intravascular sites as a prelude to diureses. It should be used with care if there is excess fluid, because of the danger of precipitating congestive failure and pulmonary edema. We would not recommend use of albumin in patients whose plasma protein oncotic pressure is more than 20 mm Hg.

2. Salt content. Patients who are in congestive failure should receive little salt. Low serum sodium is most often due to dilutional hyponatremia rather than to inadequate salt intake and should be treated by restricting fluid intake rather than increasing sodium intake. There is evidence to suggest that high salt intake in postoperative patients is associated with significant increase in Q_s/Q_t and A-aDO₂.⁶

3. Potassium. Attention to serum potassium levels is of particular importance in these patients, especially if they are receiving digitalis or diuretics to prompt the loss of excess fluids.

4. Sodium bicarbonate. This is needed to combat severe acidosis to improve reactivity of the cardiovascular system. The inherent problem of giving bicarbonate is the increase of sodium intake and the impact on serum potassium due to the intracellular shift of potassium.

5. Magnesium. Magnesium deficiency results from lack of intake and increased loss due to diuretics; 35 mg of magnesium sulfate in 500 ml of dextrose saline solution is suggested to correct this, provided no renal impairment exists.

References

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