

the efficacy of low-dose warfarin therapy. A “no vitamin K” version is available for patients receiving this therapy. Catheters can become occluded due to mechanical factors; by fibrin at the tip; or by fat, minerals, or drugs intraluminally. These occlusions can be managed with low-dose alteplase for fibrin, with indwelling 70% alcohol for fat, with 0.1 N hydrochloric acid for mineral precipitates, and with either 0.1 N hydrochloric acid or 0.1 N sodium hydroxide for drugs, depending on the pH of the drug.

Metabolic The most common problems caused by parenteral SNS are fluid overload and hyperglycemia (Table 98e-7). Hypertonic dextrose stimulates a much higher insulin level than meal feeding. Because insulin is a potent antinatriuretic and antidiuretic hormone,

hyperinsulinemia leads to sodium and fluid retention. Consequently, in the absence of gastrointestinal losses or renal dysfunction, net fluid retention is likely when total fluid intake exceeds 2000 mL/d. Close monitoring of body mass as well as of fluid intake and output is necessary to prevent this complication. In the absence of significant renal impairment, the sodium content of the urine is likely to be <10 meq/L. Provision of sodium in limited amounts (40 meq/d) and the use of both glucose and fat in the PN mixture will reduce serum glucose levels and help reduce fluid retention. The elevated insulin level also increases the intracellular transport of potassium, magnesium, and phosphorus, which can precipitate a dangerous re-feeding syndrome if the total glucose content of the PN solution is advanced too quickly in severely malnourished patients. To assess glucose tolerance, it is generally best to start PN with <200 g of glucose/d. Regular insulin can be added to the PN formula to establish glycemic control, and the insulin doses can be increased proportionately as the glucose content is advanced. As a general rule, patients with insulin-dependent diabetes require about twice their usual at-home insulin dose when receiving PN at 20–25 kcal/kg, largely as a consequence of parenteral glucose administration and some loss of insulin to the formula’s container. As a rough estimate, the amount of insulin provided can be proportionately similar to the number of calories provided as total parenteral nutrition (TPN) relative to full feeding, and the insulin can be placed in the TPN formula. Subcutaneous regular insulin can be provided to improve glucose control as assessed by measurements of blood glucose every 6 h. About two-thirds of the total 24-h amount can be added to the next day’s order, with SC insulin supplements as needed. Advances in the TPN glucose concentration should be made when reasonable glucose control is established, and the insulin dose can be adjusted proportionately to the calories added as glucose and amino acids. These are general rules, and they are conservative. Given the adverse clinical impact of hyperglycemia, it may be necessary to use intensive insulin therapy as a separate infusion with a standard protocol to initially establish control. Once control is established, this insulin dose can be added to the PN formula. Acid-base imbalance is also common during parenteral SNS. Amino acid formulas are buffered, but critically ill patients are prone to metabolic acidosis, often due to renal tubular impairment. The use of sodium and potassium acetate salts in the PN formula may address this problem. Bicarbonate salts should not be used because they are incompatible with TPN formulations. Nasogastric drainage produces hypochloremic alkalosis that can be managed by attention to chloride balance. Occasionally, hydrochloric acid may be required for a more rapid response or when diuretic therapy limits the ability to provide substantial sodium chloride. Up to 100 meq/L and up to 150 meq of hydrochloric acid per day may be placed in a fat-free TPN formula.

Infectious Infections of the central access catheter rarely occur in the first 72 h. Fever during this period is usually attributable to infection elsewhere or another cause. Fever that develops during parenteral SNS can be addressed by checking the catheter site and, if the site looks clean, exchanging the catheter over a wire, with cultures taken through the catheter and at the catheter tip. If these cultures are negative, as they usually are, the new catheter can continue to be used. If a culture is positive for a relatively nonpathogenic bacterium like *Staphylococcus epidermidis*, a second exchange over a wire with repeat cultures or replacement of the catheter can be considered in light of the clinical circumstances. If cultures are positive for more pathogenic bacteria or for fungi like *Candida albicans*, it is generally best to replace the catheter at a new site. Whether antibiotic treatment is required is a clinical decision, but *C. albicans* grown from the blood culture in a patient receiving PN should always be treated with an antifungal drug because the consequences of failure to treat can be dire.

Catheter infections can be minimized by dedicating the feeding catheter to TPN, without blood sampling or medication administration. Central catheter infections are a serious complication, with an attributed mortality rate of 12–25%. Fewer than three infections per 1000 catheter-days should occur in central venous catheters dedicated to feeding. At-home TPN catheter infections may be treated through

TABLE 98e-7 SELECTED METABOLIC DISTURBANCES CAUSED BY PN AND THEIR CORRECTION

Disturbance	Cause	Corrective Action with PN
Hyponatremia	Increased total body water or decreased total body sodium	Decrease free water or increase sodium.
Hypernatremia	Occurs commonly with excessive isotonic or hypertonic fluid followed by diuretic administration with free water clearance; can also occur with dehydration and normal total body sodium	Increase free water to produce net positive fluid balance, maintaining sodium and chloride balance.
Hypokalemia	Inadequate intake relative to need	Use supplements.
	Excessive diuresis, tubular dysfunction	Use supplements.
	Magnesium deficiency	Increase PN magnesium.
	Metabolic alkalosis	Correct alkalosis.
Hyperkalemia	Excessive provision	Reduce supplements.
	Metabolic acidosis	Evaluate acidosis. Treat with PN acetate salt, and decrease potassium.
	Renal deterioration	Evaluate patient and adjust PN as indicated.
Hypocalcemia	Reciprocal response to phosphorus repletion	Increase calcium.
	Critical illness effect	Increase calcium.
	Severe malabsorption	Supplement calcium.
Hypercalcemia	Excessive administration or pathology (cancer, hyperparathyroidism)	Reduce or eliminate calcium.
Hypomagnesemia	Increased requirements due to diuretic use, alcoholism, malabsorption, malnutrition	Supplement magnesium.
	Critical illness	Supplement magnesium.
Hypophosphatemia	Inadequate intake relative to needs related to malnutrition, alcohol use	Supplement phosphorus.
	Increased calcium intake	Use supplements.
Hyperphosphatemia	Excessive administration or worsening renal function	Reduce phosphorus.
Azotemia	Excessive amino acid infusion or worsening renal function	Reduce amino acid level if feasible, but use renal replacement therapy if 1 g of protein/kg cannot be provided for prolonged periods.

Abbreviation: PN, parenteral nutrition.