into the subclavian vein or (less desirably) the internal jugular vein with advancement into the superior vena cava can be accomplished at the bedside by trained personnel using sterile techniques. Peripherally inserted central catheters (PICCs) can also be used, although they are usually more appropriate for non-ICU patients. Subclavian or internal jugular catheters carry the risks of pneumothorax or serious vascular damage but are generally well tolerated and, rather than requiring reinsertion, can be exchanged over a wire when catheter infection is suspected.

Although most SNS is delivered in hospitals, some patients require it on a long-term basis. At-home SNS requires a safe home environment, a stable clinical condition, and the patient's ability and willingness to learn appropriate self-care techniques. Other important considerations in determining the appropriateness of at-home parenteral or enteral SNS are that the patient's prognosis indicates survival for longer than several months and that the therapy enhances the patient's quality of life.

DISEASE-SPECIFIC NUTRITIONAL SUPPORT

The purpose of SNS is to correct and prevent malnutrition. Certain conditions require special modification of the SNS regimen. Protein intake may need to be limited in many stable patients with renal insufficiency or borderline liver function. In renal disease, except for brief periods, protein intakes should approach the required level for normal adults of at least 0.8 g/kg and should aim for 1.2 g/kg as long as severe azotemia does not occur. Patients with severe renal failure who require SNS need concurrent renal replacement therapy. In hepatic failure, protein intakes of 1.2–1.4 g/kg (up to 1.5 g/kg) should be provided as long as encephalopathy due to protein intolerance does not occur. In the presence of protein intolerance, formulas containing 33–50% branched-chain amino acids are available and can be provided at the 1.2- to 1.4-g/kg level. Cardiac patients and many other severely stressed patients often benefit from fluid and sodium restriction to 1000 mL of PN formula and 5–20 meq of sodium per day. In patients with severe chronic PEM characterized by severe weight loss, it is important to initiate PN gradually because of the profound antinatriuresis, antidiuresis, and intracellular accumulation of potassium, magnesium, and phosphorus that develop as a consequence of the resulting high insulin levels. This modification of parenteral SNS is usually accomplished by limiting daily fluid intake initially to ~1000 mL; limiting carbohydrate intake to 10–20% dextrose; limiting sodium intake; and providing ample potassium, magnesium, and phosphorus, with careful daily assessment of fluid and electrolyte status. Protein need not be restricted.

THE DESIGN OF INDIVIDUAL REGIMENS

FLUID REQUIREMENTS

Normal adults require ~30 mL of fluid/kg of body weight from all sources each day as well as the replacement of abnormal losses such as those caused by diuretic therapy, nasogastric tube drainage, wound output, high rates of perspiration (which can be several liters per day during periods of extreme heat), and diarrhea/ostomy losses. Electrolyte and mineral losses can be estimated or measured and need to be replaced **(Table 98e-2)**. Fluid restriction may be necessary in patients with fluid overload. Total fluid input can usually be limited to 1200 mL/d as long as urine is the only significant source of fluid output. In severe fluid overload, a 1-L central vein PN solution of 7% crystalline amino acids (70 g) and 21% dextrose (210 g) can temporarily provide an acceptable amount of glucose and protein substrate in the absence of significant catabolic stress.

Patients who require PN or EN in the acute-care setting generally have associated hormonal adaptations to their underlying disease (e.g., increased secretion of antidiuretic hormone, aldosterone, insulin, glucagon, or cortisol), and these signals promote fluid retention and hyperglycemia. In critical illness, body weight is invariably increased due to fluid resuscitation and fluid retention. Lean-tissue accretion is minimal in the acute phase of critical illness, no matter how much protein and or how many calories are provided. Because excess fluid removal can be difficult, limiting fluid intake to allow for balanced intake and output is more effective.

TABLE 98e-2 APPROXIMATE VOLUME AND COMPOSITION OF FLUID SECRETIONS[a]

Secretion	Volume (L/d)	Na	K	Cl	HCO_3	pH
Saliva	1–2	15	30	15	30	6–8
Gastric juice	1–2	50–90	5–30	90–130	0	1.5–3.5
Hepatic bile	0.5–1	130–150	5–10	80–120	30–50	7–9
Pancreas	0.5–1	130–150	5–10	70–100	90–110	8–9
Small intestine	1–3	120–140	10–20	80–120	20–40	7–9
Diarrhea	Varies	30–50	20–30		Varies	

[a]Concentration values are in mmol/L.

ENERGY REQUIREMENTS

Total energy expenditure comprises resting energy expenditure, activity energy expenditure, and the thermal effect of feeding **(Chap. 97)**. Resting energy expenditure accounts for two-thirds of total energy expenditure, activity energy expenditure for one-fourth to one-third, and the thermal effect of feeding for ~10%. For normally nourished, healthy individuals, the total energy expenditure is ~30–35 kcal/kg. Critical illness increases resting energy expenditure, but this increase is significant only in initially well-nourished individuals with a robust SRI who experience, for example, severe multiple trauma, extensive burns, sepsis, sustained high fever, or closed head injury. In these situations, total energy expenditure can reach 40–45 kcal/kg. The chronically starved patient with adapted PEM has a reduced energy expenditure and is inactive, with a usual total energy expenditure of ~20–25 kcal/kg. Very few patients with adapted PEM require as much as 30 kcal/kg for energy balance. Because providing ~50% of measured energy expenditure as SNS is at least as effective as 100% for the first 10 days of critical illness, actual measurement of energy expenditure generally is not necessary in the early period of SNS. However, in patients who remain critically ill beyond several weeks, in patients with severe PEM for whom estimates of energy expenditure are unreliable, and in patients who are difficult to wean from ventilators, it is reasonable to measure energy expenditure directly when the technique is available, targeting an energy intake of 100–120% of the measured energy expenditure.

Insulin resistance due to the SRI is associated with increased gluconeogenesis and reduced peripheral glucose utilization, with resulting hyperglycemia. Hyperglycemia is aggravated by excessive exogenous carbohydrate administration from SNS. In critically ill patients receiving SNS, normalization of blood glucose levels by insulin infusion reduces morbidity and mortality risk. In mildly or moderately malnourished patients, it is reasonable to provide metabolic support in order to improve protein synthesis and maintain metabolic homeostasis. Hypocaloric nutrition, with provision of ~1000 kcal and 70 g protein per day for up to 10 days, requires less fluid and reduces the likelihood of poor glycemic control, although a higher protein intake would be optimal. During the second week of SNS, energy and protein provision can be advanced to 20–25 kcal/kg and 1.5 g/kg per day, respectively, as metabolic conditions permit. As mentioned above, patients with multiple trauma, closed head injury, and severe burns often have greatly elevated energy expenditures, but there is little evidence that providing >30 kcal/kg daily confers further benefit, and such high caloric intake may well be harmful as it substantially increases the risk of hyperglycemia.

As a rule, amino acids and glucose are provided in an increasing dose until energy provision matches estimated resting energy expenditure. At this point, it becomes beneficial to add fat. A surfeit of glucose merely stimulates de novo lipogenesis—an energy-inefficient process. Polyunsaturated long-chain triglycerides (e.g., in soybean oil) are the chief ingredient in most parenteral fat emulsions and provide the majority of the fat in enteral feeding formulas. These vegetable oil–based emulsions provide essential fatty acids. The fat content of