Chapter 13
Nutrition Support

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Key Points

- Nutrition support is of critical importance to both the ischemic and hemorrhagic stroke patient population due to dysphagia, altered mental status, and the possible need for intubation.
- Enteral nutrition (EN) is clearly established as the preferential route of nutrition support for this patient population, which should commence as early as feasible. Parenteral nutrition (PN) is indicated for the patient with a non-functioning and/or inability to access the gastrointestinal (GI) tract.
- The extent of neurological damage and recovery will affect nutrition requirements and the nutrition care plan.

Keywords Nutrition assessment • Enteral nutrition • Parenteral nutrition • Dysphagia • Stroke • Transitional feeding • Stroke rehabilitation

Abbreviations

ACTH	Adrenocorticotropin Releasing Hormone
ASPEN	American Society for Parentearl and Enteral Nutrition
BMI	Body Mass Index
EN	Enteral Nutrition
GI	Gastrointestinal

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ICP	Intracranial Pressure
IL-1RA	Interleukin-1 Preceptor Antagonist
IL-6	Interleukin-6
IVFE	Intravenous Fat Emulsion
LBM	Lean Body Mass
NDD	National Dysphagia Diet
PPN	Peripheral Parenteral Nutrition
PN	Parenteral Nutrition
TBI	Traumatic Brain Injury
TEE	Total Energy Expenditure

Metabolic Response Post Stroke

The intense catabolic response that occurs in patients who have sustained a traumatic brain injury (TBI) is well documented, but not well defined in the stroke patient population. The earlier notion that stroke patients exhibit an initial hypermetabolic phase similar to TBI has been challenged over the past decade. Bardutzky and colleagues evaluated 34 sedated, mechanically ventilated patients with ischemia and hemorrhagic stroke and found that total energy expenditure (TEE) as determined by indirect calorimetry was low during the first 5 days after ICU admission [1]. In addition, the TEE did not differ between acute cerebral ischemia or intracerebral hemorrhage. Brain injury resulting from stroke has metabolic and physiologic consequences, and the presence of preexisting malnutrition and malnutrition that may develop after a stroke contributes to clinical outcomes [2–4]. The obligatory mobilization of lean body mass (LBM) observed with brain injury is estimated to be up to 25 g N per day, or triple the normal turnover rate, which would cause a 70 kg man to lose 10% of LBM in a week [5]. Chalela and associates examined nitrogen balance in patients who had acute stroke (either ischemic or hemorrhagic) where enteral nutrition (EN) was initiated within 2 days of injury and nitrogen balance was evaluated on day 5 [6]. The authors found that 44.4% of patients were in negative nitrogen balance and concluded that patients were underfed.

The injured brain stimulates the secretion of many hormones that affect metabolic function such as adrenocorticotrophin releasing hormone (ACTH), growth hormone, prolactin, vasopressin, and cortisol as a natural response to stress. In addition, glucagon and catecholamines are released in excess. Although catecholamines are released to help support blood pressure and cardiac output (and hence cerebral perfusion), the surge of catecholamines after an acute brain injury leads to hyperglycemia and hyperinsulinemia, which impairs ketogenesis and promotes protein catabolism. The resultant catabolic state can have significant detrimental effects on systemic organ function [7].

After excluding patients with evidence of infection, Beamer and colleagues [8] found higher levels of interleukin-6 (IL-6), a regulator of the acute phase response,

Table 13.1
Interpretation of BMI [9]

BMI	Interpretation
Below 18.5	Underweight
18.5–24.9	Normal
25–29.9	Overweight
30–34.9	Obesity (grade 1)
35–39.9	Obesity (grade 2)
Greater than 40	Obesity (grade 3)

and interleukin-1 receptor antagonist (IL-1RA), an anti-inflammatory mediator in the acute phase, in patients with acute stroke compared with healthy, community-living controls ($p < 0.001$). Both IL-6 and IL-1RA were significantly correlated with the C-reactive protein, an acute phase protein, suggesting that there is an acute phase response to brain infarction. Exogenous substrates provided by EN or PN may reduce the need for the liberation of endogenous substrate stores, and thus reduce these catabolic effects.

Nutrition Assessment

Components

Components of a comprehensive nutrition assessment include evaluation of anthropometrics, biochemical markers, clinical examination, and the patient's recent diet history. Although the same four components of nutrition assessment are utilized regardless of the patient's injury or disease state, the focus of these components can be tailored to the unique nutritional needs of the stroke patient. Nutrition assessment identifies patients requiring nutrition intervention and the ideal timing of intervention.

Assessment of body weight and composition guides nutrition assessment, estimation of nutritional requirements (macronutrient needs), and development of a nutrition care plan. Body mass index (BMI, {weight (kg)/height $(m)^2$}) is often a starting point to a nutrition assessment. See Table 13.1 for interpretation of BMI [9]. Caution must be taken when interpreting BMI as it may be overinflated by altered weight from fluid resuscitation or fluid retention in the ICU setting. A recent, premorbid weight or edema free admission weight should be used for BMI calculations. Assessment of the patient's weight history pre-neurological injury will guide the nutrition clinician in assessment of the nutritional status.

Unfortunately, there is no universally accepted definition of malnutrition. Currently The American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) defines malnutrition as "an acute, sub acute, or chronic state of nutrition, in which a combination of varying degrees of overnutrition or undernutrition and inflammatory activity have led to a change in body composition and diminished function" [10].

Table 13.2
Predictive equation

Formula	Male	Female
Harris-Benedict equation	BMR = 66 + (13.7 × weight (kg)) + (5 × height (cm)) − (6.8 × age (years))	BMR = 655 + (9.6 × weight (kg)) + (1.8 × height (cm)) − (4.7 × age (years))

A new A.S.P.E.N. Taskforce is proposing a new method to diagnose malnutrition by taking into account the impact of the inflammatory response on nutritional status [11]. Once validated, this will be a tool to help universalize the diagnosis of malnutrition.

Obtaining a detailed nutrition history including intake and recent weight history is critical for nutrition assessment. A weight history that reveals involuntary weight loss of greater than 10% of usual body weight in 6 months or loss of 5% of usual body weight in 1 month indicates malnutrition. Cognitive function or intubation status may limit the patient from providing an accurate history and nutrition professionals may need to seek this information from family members or other caregivers. A nutrition focused physical exam will identify edema, muscle wasting, signs of nutrient deficiencies, and assist in assessment of nutritional status.

As equations to estimate nutritional requirements are fraught with inaccuracies (of either under- or overestimation of caloric needs), indirect calorimetry is the gold standard for determining caloric requirements. Unfortunately, indirect calorimetry is not routinely available for use in many institutions for a variety of reasons (largely due to cost and the need for skilled clinicians to preform and interpret the readings). No single predictive equation is available or has been validated with a large patient sample size in the stroke population. The presence of obesity may also further complicate calculation of nutritional requirements when indirect calorimetry is unavailable.

Finestone and colleagues used indirect calorimetry on day 7, 11, 14, 21, and 90 days post stroke to study energy demands over time after stroke [12]. Resting energy expenditure (REE) was shown to be approximately 10% higher than predicted by the Harris-Benedict equation, but energy needs did not differ by type of stroke, and changes in resting energy expenditures were not statistically significant over time [12]. These results confirm findings from a smaller study that measured REE 24–72 h following stroke and a repeated measurement of REE 10–14 days after [13]. The authors suggested that energy requirements were not elevated due to decreased physical activity and changes in muscle tone due to the neurological injury [13]. See Table 13.2 for predictive equations for nutrition assessment. The caloric content of medications such as propofol (1.1 cal/mL) or dextrose containing intravenous fluids should be taken into consideration for the nutrition feeding prescription to prevent overfeeding.

Protein needs should be individualized, but 1–1.5 g/kg is recommended [14]. Protein needs may be higher in rehabilitation patients in the presence of wounds. Mobility limitations may also predispose patients for skin breakdown and

development of pressure ulcers. Routine skin assessment is required and provision of adequate protein is prudent.

Infection, mobility, activity levels, and weight status may alter caloric requirements leading to the need for frequent reassessment. When limitations in mobility or paralysis are present, caloric requirements will be lower due to decreased activity. Early medical treatments in the acute care setting with barbiturates or induced hypothermia as a method to decrease intracranial pressure also decreases caloric requirements [1, 15]. Indirect calorimetry is ideal for assessment of nutritional needs in these clinical scenarios.

Many professionals heavily rely on biochemical markers to quickly label nutritional status. Hepatic proteins such as albumin, prealbumin (transthyretin), and transferrin are commonly used as nutritional markers in clinical practice. Historically, the use of serum albumin was used to define malnutrition in hospitalized patients. Older studies, including the majority of those done in the stroke population, commonly placed a strong emphasis on equating low albumin levels to malnutrition without accounting for the role of the inflammatory cascade after injury. After neurological injury, metabolic demands are altered with elevation of peripheral plasma catecholamines, cortisol, glucagon, interleukin-6, interleukin-IRA, and acute phase proteins [12]. These alterations directly impact the way traditional biochemical markers of nutrition are assessed.

Now with the recent focus on the impact of inflammation on nutrition, clinicians understand that hepatic proteins are influenced by many non-nutritional factors and change rapidly in times of stress and in turn do not accurately reflect nutritional status. Mediators of inflammation have the largest effect on serum protein levels and contribute to an increase in net protein loss from catabolism [16]. Albumin, prealbumin, and transferrin are all negative acute phase proteins, thereby the liver decreases production of these components in the presence of inflammation regardless of pre-morbid nutritional status.

Albumin can be a good marker of nutritional status in the absence of inflammation and infection. Given the long half-life of albumin (20 days), it may only be ideal for long-term care or rehabilitation settings. Prealbumin (3 days) and transferrin (7 days) have much shorter half-lives compared to albumin and are more appropriately monitored in the acute care setting along with C-reactive protein levels for a decrease of inflammation. Following hepatic protein levels over time may be considered to be of more clinical significance by some clinicians than a single point in time measurement. Although commonly used in clinical practice, the use of prealbumin and C-reactive protein have not been validated.

Timing of Enteral Nutrition

Once it is determined that a stroke patient cannot be fed orally, EN should be initiated in the first 24–48 h, as it clearly becomes an important goal for the initial nutrition support plan. Early EN has been associated with beneficial effects such as

attenuation of the hypercatabolic response, gut atrophy, muscle mass loss, and infection in brain injury patients [17]. Chiang and colleagues found EN initiated within 48 h post injury in patients with severe TBI is associated with greater survival rate, Glasgow Coma Score (GCS) recovery, and a better clinical outcome at 1 month post injury [18]. The provision of early nutrition forestalls the breakdown of protein and fat stores, blunts the innate inflammatory response, promotes immune competence, decreases intensive care unit (ICU) infections, limits the risk of bacterial translocation, and improves neurologic outcome at 3 months after injury [16, 19].

Enteral Nutrition

The major effect on the GI tract following stroke is impairment of oral, pharyngeal, and esophageal functions manifested as dysphagia. Swallowing requires multiple neurologic inputs to perform correctly and damage to these circuits may occur as a result of stroke in as many as 60% of patients [20]. The decision on how to feed a stroke patient should be made shortly after hospital admission and will be partially dictated by the patient's presenting condition and medical/surgical history [21]. See the enteral stroke feeding algorithm in Fig. 13.1 [21]. The selected route of nutrition support should commence as soon as the stroke patient is stabilized and adequately resuscitated. In addition, a patient who sustained a massive stroke may be vented initially and require specialized nutrition support. Appropriate candidates for EN have functional GI tracts and no contraindications to placement of a feeding tube, such as coagulopathy following tissue plasminogen activator (t-PA) administration or previous medical/surgical procedures such as gastrointestinal (GI) surgery that may alter normal anatomy [22].

Numerous challenges exist in providing adequate EN to critically ill stroke patients such as impaired gastric emptying due to vagus nerve damage, elevated levels of endogenous opioids and endorphins, or medications such as pentobarbital or narcotics, altered levels of consciousness, and overall neurologic function [7, 23, 24]. Patients who are in the ICU can have elevated intracranial pressure (ICP), which can delay gastric emptying. Thus, initial attempts to feed via a nasogastric tube may not be successful, and small bowel feeding should be considered. However, careful consideration of clinical status is warranted before endoscopically placing a feeding device in patients with ICP elevations [7, 25]. Monitoring the ICP is important during endoscopic procedures in critically ill stroke patients to minimize secondary insults to the brain (intracranial hypertension, seizures, and cerebral edema) [7]. As the ICP improves, patients often tolerate gastric feeding [21]. Tube feeding should be started as a continuous drip, full strength at 10–20 mL/h, and advanced to goal rate by 10–20 mL every 4–8 h. In the stable stroke patient who is tolerating gastric feedings, tube feeding regimens may be adjusted on an individual basis to a bolus or gravity controlled regimen to facilitate initiation of oral intake or to avoid food–drug interactions.

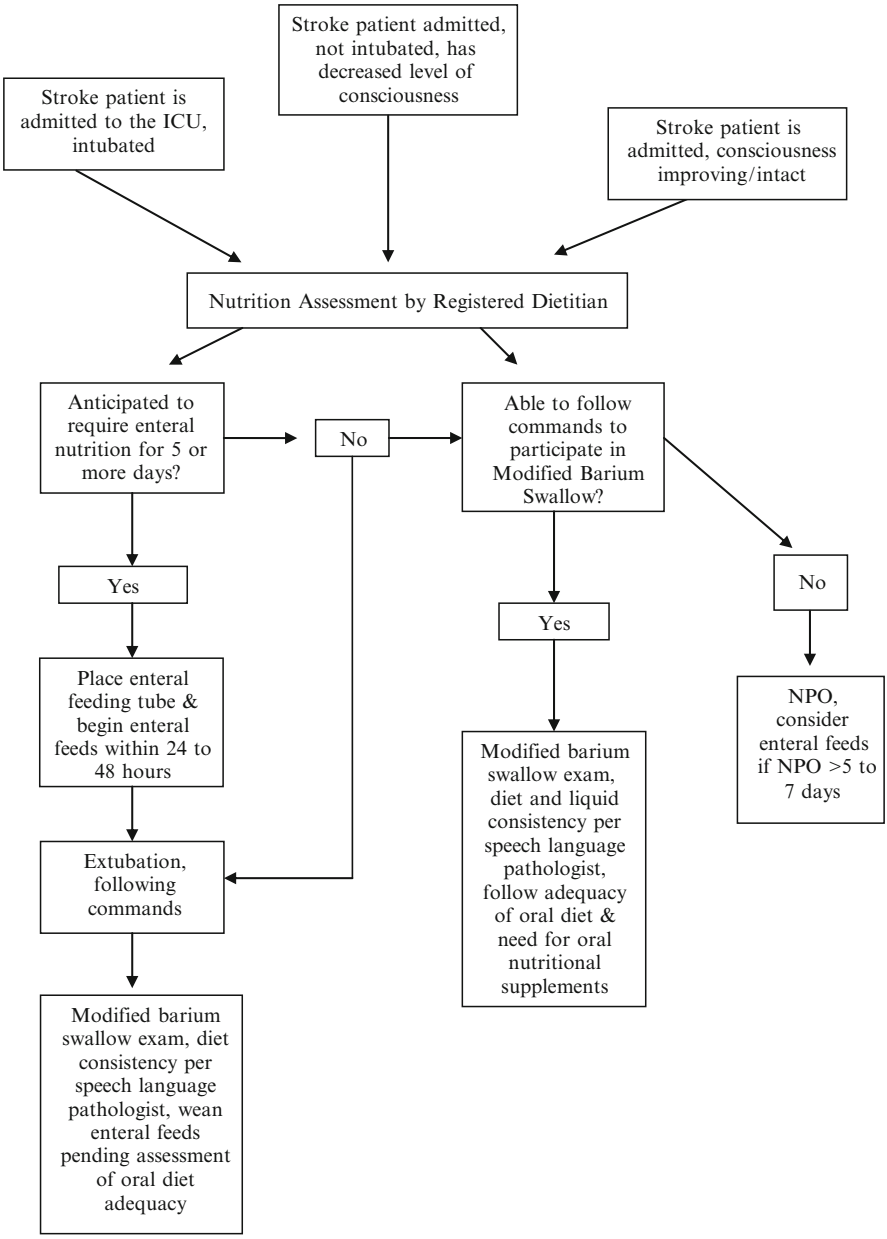

Fig. 13.1 Enteral stroke feeding algorithm (Reprinted with permission from Sage Publications [21])

Caution is also warranted in patients receiving EN and high doses of vasopressors due to case reports of ischemic bowel. EN should be withheld in patients who are hypotensive (mean arterial pressure <60 mmHg), particularly if clinicians are initiating use of catecholamine agents (norepinephrine, phenylephrine, epinephrine, dopamine) or escalating the dose of such agents to maintain hemodynamic stability. It appears that provision of EN with caution to patients on stable low doses of pressor agents is safer than initially thought. If a patient is on stable or declining doses of vasopressors, start trophic or trickle feeds of 10–20 mL/h of a polymeric, fiber-free solution and leave at that rate for 24 h then reassess. But any signs of intolerance (abdominal distention, increasing nasogastric tube output or gastric residual volumes, decreased passage of stool and flatus, hypoactive bowel sounds, rising lactate, increasing metabolic acidosis and/or base deficit), should be scrutinized as possible early signs of gut ischemia [26]. If the patient is tolerating slow rate EN and the clinical condition is improving, start to increase the feeding rate. A recent large scale, multicenter, observational study evaluated mechanically ventilated, vasopressor dependent patients who were classified as to whether they were fed within 48 h (early group) or after 48 h (after group). They used sophisticated statistical strategies (propensity scores) to adjust for potential confounding variables and showed that vasopressor dependent patients fed early (within 48 h) had a significant survival advantage compared to the delayed group (after 48 h) [27].

Nasally inserted small bowel feeding access devices are generally safe and easily inserted at bedside. Adequate anchoring of the tube is essential to prevent the tube from getting dislodged due to patient agitation, discomfort, or inadequate sedation. In these instances, use of a commercial retention system or nasal bridle has been associated with fewer displaced tubes [28–30]. Acosta-Escribano and associates evaluated the efficacy of small bowel feeding compared with gastric feeding in patients with severe TBI and found a decrease in the incidence of ventilator-associated pneumonia, low rate of GI complications, and tube malfunction [31]. Implementation of feeding protocols that specifically address the timing of establishing enteral access, tube placement confirmation, and route of feeding (EN or PN) could assist clinicians so patients are fed as quickly as possible [26].

Enteral Formula Selection

Enteral tube feeding formulas are generally well tolerated in the stroke patient population. Depending on calorie and protein needs, a 1 kcal/mL to a 2 kcal/mL, polymeric, standard intact protein formula is appropriate. Very limited data exists regarding any benefits or detriments of specialty products in the stroke patient population. The role of elemental and semi-elemental enteral tube feeding formulations is not well defined in this patient population. The use of fiber must be considered in light of the medical plan of care. Fiber is avoided in cases where pressors or paralytic agents are being used. Fiber-containing formulation is generally recommended for patients requiring long-term enteral feedings, especially during rehabilitation.

Most 2 kcal/mL formulas do not meet estimated protein requirements for this population. Therefore, patients will require use of a protein supplement to meet needs. Monitoring protein supplementation is difficult as albumin and prealbumin are both negative acute phase proteins and are more indicative of inflammation and not nutrition deprivation or recovery. Protein supplementation should be routinely reviewed and adjusted during the course of stroke recovery. A 2 kcal/mL formula may also be warranted during fluid restriction for control of the syndrome of inappropriate antidiuretic hormone (SIADH).

Complications of Enteral Nutrition

Diarrhea is the most commonly reported complication of enteral feeding. No universally accepted definition of diarrhea exists. However, a clinically useful definition is any abnormal volume or consistency of stool. Normal stool water content is 250–500 mL/day. Diarrhea has also been defined as >500 mL every 8 h or >3 stools per day for at least 2 consecutive days [32, 33]. Most standard EN formulas are lactose free, not excessively high in fat, and are not exceedingly hypertonic to cause diarrhea. Brain-injured patients often receive elixirs (containing sorbitol), electrolyte supplements, and other medications that are extremely hypertonic compared to the EN [7]. Patients with diarrhea should be evaluated and treated in a stepwise approach before using parenteral nutrition (PN). Once infection and medication-related factors have been eliminated as causes of diarrhea, changing to an enteral formula with added fiber or addition of soluble fiber to the medication regimen may lessen diarrhea. A semi-elemental EN product may improve absorption and minimize diarrhea. Antidiarrheal agents can be added once *Clostridium difficile* has been ruled out or is being treated. Constipation is common in the stroke patient receiving narcotics and should be given a standing bowel regimen when not experiencing diarrhea.

Gastric residuals have traditionally been used to assess tolerance to tube feeding and aspiration risk. Research has shown no correlation between the presence of high gastric residual volumes and gastric emptying, regurgitation, vomiting, and pneumonia [34]. An isolated incidence of a high gastric residual volume should not prompt enteral feeding to be held without other signs and symptoms of intolerance. Evidence suggests that a decision to stop tube feeding should be based on a trend in serial measurements and not on a single isolated high volume [35].

Implementation of a feeding protocol has been recommended by clinical practice guidelines as a key strategy to standardize the delivery of EN, maximize the benefits, and minimize the risks of EN in the critically ill patients [26]. Utilizing feeding protocols or algorithms not only improve the delivery of EN, but have also been shown to improve outcome. Results of a multicenter observational study showed that sites that used feeding protocol use more EN, started EN earlier, and used more motility agents in patients with high gastric residual volumes compared to sites that did not use a feeding protocol [36]. In addition, adequacy of EN was higher in sites

with protocols compared to non-protocol sites. Components of feeding protocols may include orders for early initiation of EN, use of motility agents, gastric residual volumes, head of the bed elevation, and use of small bowel feeding tubes.

If delayed gastric emptying is suspected, appropriate interventions include elevating the head of bed by 30° to 45° to decrease reflux of gastric contents into the pharynx and esophagus and X-ray confirmation of feeding tube placement [26]. The use of small bowel feeding tubes (ideally the tip of the feeding tube located in the third portion of the duodenum or further distal) allows for increased feeding tolerance and to minimize pulmonary aspiration [37]. Small bowel access can be attained at the bedside, and a number of techniques that will be described on Chap. 14 (endoscopically, fluoroscopically, etc.). Continuous infusion of EN is better tolerated early in neurologic illness compared to bolus feedings. If nausea or vomiting occur as the rate of administration of the EN increases, the rate should be reduced to the last tolerated amount, with an attempt to increase the rate again after the symptoms abate. Promotility agents such as metoclopramide or erythromycin should also be considered to promote peristalsis and EN tolerance. Promotility agents are not without adverse effects, so these agents should be used for a short duration until the desired effect is obtained and maintained [7].

Abdominal distention and its associated symptoms of bloating and cramping, usually occurs as a result of ileus and obstruction. Critically ill stroke patients frequently require paralytic agents and narcotics which can contribute to poor GI motility and may prevent the patient from reaching their EN goal. Discontinuation of EN and initiation of PN may be necessary if the motility is poor or if the bowel is markedly dilated.

Parenteral Nutrition

The use of PN in the stroke population is rare, but when indicated is not without risk. The use of small bowel feeding tubes has alleviated many cases of inappropriate PN use. Common complications of PN used on a short-term basis include hyperglycemia, catheter related blood stream infection, and electrolyte imbalances.

Indications for Parenteral Nutrition

Delivery of enteral nutrients provides multiple benefits over delivery of nutrients via the parenteral route. Benefits of EN include decreased length of stay, lower risk of infection, decreased cost, and fewer hyperglycemia and infectious complications compared to PN, and also maintains integrity of the gut mucosa, preserves gut barrier function, and gut associated immune function [38–42].

Indications for PN require the patient to present with a nonfunctioning GI tract or unable to ingest or absorb adequate nutrients from EN (either orally or via a

feeding tube). Indications for PN include diffuse peritonitis, intestinal obstruction, intractable vomiting or diarrhea, prolonged ileus, and GI ischemia [39], radiation enteritis, malabsorption from short bowel syndrome, high output GI fistulas, severe GI bleed, and pseudo-obstruction [38, 43]. These medical conditions necessitating use of PN are not routinely seen following stroke.

In the stroke population, the use of PN may be indicated with prolonged inability to access the GI tract depending on the patient's pre-morbid nutritional status. Every attempt to achieve enteral access should continue to be made after PN is initiated. The use of small bowel feeding tubes (ideally the tip of the feeding tube should be located in the third portion of the duodenum or beyond—see Chap. 14) can avoid the inappropriate use of PN for ileus or gastroparesis following stroke. Patients receiving PN therapy should exhibit presence of significant malnutrition. For the benefit of PN to be realized, PN should be used for at least 7 days [26]. Clinically, it may be challenging for clinicians to predict the length of use of PN therapy as each patient case and progress is unique.

The timing of PN initiation is important and is determined based on the patient's nutritional status. Published ICU nutrition guidelines from the Society for Critical Care Medicine (SCCM) and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N) in 2009 specifically address use and timing of PN in the critically ill population. In the presence of severe malnutrition (as identified from nutrition assessment), if EN is not possible, PN should be started early after adequate resuscitation to reduce complications compared to just providing standard therapy (intravenous fluids and no nutrition support) [41, 44]. Standard therapy (intravenous fluids and no nutrition support) in severely malnourished ICU patients leads to a higher risk of mortality and also a trend towards a higher rate of infection [44].

In the absence of malnutrition prior to ICU admission, where EN is not an option or indicated, standard therapy (intravenous fluids and no nutrition support) is superior to PN until after 7 days [26]. Standard therapy was associated with significantly less infectious morbidity, and a trend towards overall reduction in complications compared to use of PN [45]. When PN was provided in patients who were not malnourished, an increase in mortality and a trend towards increased rate of complications occurred [44].

Contraindications for Providing Parenteral Nutrition

The main contraindication to PN is a functional GI tract. Other contraindications include unstable cardiopulmonary parameters, aggressive therapy is no longer desired due to DNR status, or unstable fluid and electrolytes. Initiation of PN should be delayed until fluid and electrolyte disturbances are fully corrected as evidenced by normal serum values. Careful ongoing monitoring and correction of electrolyte abnormalities and glycemic control is prudent in patients receiving PN.

Vascular Access and Types of Parenteral Nutrition Solutions

There are a variety of vascular access devices through which PN may be infused. The choice for which vascular access device to use is dictated by the length of PN therapy. Peripheral catheters can be used on a short-term basis to provide peripheral PN (PPN); however, the patient's full nutritional requirements will not be met. PPN is generally only used for a maximum of 1–2 weeks due to the increased amount of fat provided. Fat contributes least to the osmolarity of a PPN solution leading to the higher amount of fat dosed in PPN. A PPN solution with a maximum of 900 mOsm can be infused through a peripheral catheter (see Chap. 19 for calculation of peripheral PN and osmolarity).

A peripheral catheter providing PPN may be useful while awaiting placement of central access or confirmation of the location of the tip of the catheter. Peripheral PN should not be used in patients with cardiac or renal dysfunction necessitating fluid restriction or in any patient requiring a low volume PN solution. Peripheral PN solutions deliver a large percentage of calories as isotonic fat emulsions and a smaller percentage of dextrose and protein calories, and are less hypertonic than central PN solutions (less than 900 mOsm).

Hospitalized patients commonly have temporary central venous catheters (either via the internal jugular or subclavian veins) or Peripherally Inserted Central Catheters (PICCs). When a vascular access device is used for PN, the tip of the central venous catheter should be placed with the tip located at the cavo-atrial junction to reduce the risk of catheter associated venous thrombosis [38, 46–48]. Other acceptable positions for the tip of the vascular access device include the mid to lower third of the superior vena cava. It is critical in the stroke patient especially to take any measure to avoid thrombosis as anticoagulation may be contraindicated in certain patients after a stroke [21]. A central PN solution has no limit on osmolarity as does peripheral PN and therefore can be customized to meet the patient's full nutritional requirements. For calculation of central PN solutions see Chap. 19.

Parenteral Nutrition Solutions

The three basic components of a PN solution include dextrose (as dextrose monohydrate providing 3.4 cal/g), protein (in the form of crystalline amino acids providing 4 cal/g), and intravenous fat emulsion ([IVFE] providing 10 cal/g). It is a general recommendation that IVFE be limited to provide no more than 1 g/kg of body weight of fat per day. PN solutions providing only dextrose and protein are called two-in-one PN solutions whereas three-in-one PN solutions contain dextrose, protein, and IVFE. The decision to incorporate IVFE into the PN solution is individualized based on the clinical judgment. SCCM and A.S.P.E.N. Guidelines suggest IVFE free PN solutions for ICU patients during the first week of therapy [26].

Additives

Electrolytes are added as salts for maintenance of serum electrolytes. Calcium, magnesium, potassium, sodium, and phosphorus may be added to PN solutions. Potassium and sodium salts come in the form of acetate or chloride and are manipulated based on that patients laboratory studies (serum bicarbonate and chloride levels). Standard intravenous multivitamin (MVI) and multiple trace element preparations are added to PN solutions daily. Some MVI solutions contain a small amount of vitamin K (150 μg), but the MVI should not be omitted from the PN solution when on anticoagulation therapy as the amount of vitamin K is consistent from day to day.

Iron is not routinely added to PN solutions and cannot be added to a PN solution containing IVFE due to the risk of destabilizing the solution [49]. The only form of iron compatible with fat free PN solutions is iron dextran and a test dose must be given outside the PN solution before it can be added due to the risk of anaphylaxis. Iron therapy via PN solutions is rarely used in the ICU setting and only with long-term PN patients outside the ICU setting. Iron should not be given in times of infection or sepsis [49].

Medications may be added to PN solutions; however, if the PN solution is stopped for any reason the patient does not receive the full dose of the medication. Commonly added medications include H2 blockers, octreotide, insulin, hydrocortisone, or heparin.

Parenteral Nutrition Administration

PN solutions are infused over 24 h in acute care settings. Calories are gradually increased over a few days to provide the patients full nutritional requirements while monitoring electrolytes and blood sugars. Cyclic PN solutions are typically only used in the home or long-term care setting.

Parenteral Nutrition Complications

Complications of PN include infectious (catheter sepsis or site infections), mechanical (catheter occlusions, breaks), and metabolic (hyperglycemia, electrolyte abnormalities, metabolic bone disease). Other complications include thrombosis and PN associated liver disease. Short-term PN complications seen in the acute care setting with short-term PN use are mainly hyperglycemia and electrolyte abnormalities, but also can include catheter sepsis and mechanical complications.

Hyperglycemia seen with initiation of PN solutions can be managed in many ways. Initiating PN with a gradual increase of the dextrose load is prudent along with frequent monitoring until blood sugars are stable on the goal calorie PN

Table 13.3
Parenteral nutrition monitoring for the hospital setting

Baseline (before PN initiation)	First 3 days (or until normal values on goal PN solution)	Weekly
CMP[a]	BMP[b]	CMP[a]
Magnesium and phosphorus	Magnesium and phosphorus	Magnesium and phosphorus
Complete blood count		Complete blood count
Glucose, serum	Every 6 h with continuous PN	On CMP

[a]Complete metabolic panel (sodium, potassium, chloride, bicarbonate, blood urea nitrogen, creatinine, glucose, total protein, albumin, AST, ALT, Alk phos, bilirubin)
[b]Basic metabolic panel (sodium, potassium, chloride, bicarbonate, blood urea nitrogen, creatinine, glucose)

solution. Insulin drips can be utilized in the ICU setting for severe hyperglycemia and insulin can be added to the PN solution to assist with glycemic control. Great debate continues on the ideal range to maintain glycemic control. Blood sugars may be elevated due to reasons other than PN solutions such as medications (steroids), past medical history (diabetes, pancreatitis), and in the presence of infection.

Refeeding syndrome or a shift of serum electrolytes from the serum to intracellular space may occur in malnourished patients when initiating a large number of calories through initial PN solutions. Hypomagnesemia, hypokalemia, and hypophosphatemia are frequently observed with excessive dextrose in initial PN solutions [50]. Gradual advancement of calories via PN solutions is prudent to avoid this metabolic complication.

Parenteral Nutrition Monitoring

See Table 13.3 for suggested monitoring of PN therapy in the hospital setting.

Dysnatremia

Disturbances of sodium balance, referred to as dysnatremia, are frequently observed in neurocritical patients. In the past, fluid restriction was a common practice in patients with elevated ICP to reduce ICP by decreasing total circulating volume. Today, however, most data suggest resuscitation to a normal intravascular volume, with the avoidance of hyponatremia and hypo-osmolarity [51]. Hyponatremia can occur due to the syndrome of inappropriate antidiuretic hormone (SIADH) or cerebral salt wasting syndrome (CSWS). The physiological response elicited by SIADH results in renal conservation of water and a dilutional hyponatremia that is treated with a water restriction. To facilitate a negative fluid balance, concentrated enteral or parenteral formulations should be provided.

The primary defect in CSWS is due to the kidney's inability to conserve salt due to intracranial disease, resulting to hyponatremia and volume depletion. CSWS is predominantly associated with subarachnoid hemorrhage (SAH), but has also been described in conjunction with traumatic brain injury, glioma, and tuberculous or carcinomatous meningitis [52]. Once the presence of CSWS is confirmed, treatment consists of volume and salt replacement which may be accomplished by intravenous (IV) normal saline, or rarely, hypertonic saline, and/or oral/enteral salt provision [53]. The initial finding of an abnormal sodium level should always prompt specific investigation into the underlying cause before management is initiated. The speed of the onset of the hyponatremia, as well as the presence of symptoms, is most important because patients with the most rapid onset are more likely to become symptomatic [54]. A differentiation must be made between hypervolemia with normal total body sodium (suggesting SIADH) and hypovolemia with disproportionately low total body sodium (suggesting CSWS). This differentiation is crucial because the managements of these two conditions are diametrically opposed, but will not be considered further here [53].

Hypernatremia and hyperosmolality due to diabetes insipidus (DI) may frequently develop in patients with severe cerebral disease or injury. DI is characterized by urinary output of more than 300 mL/h with a specific gravity of <1.005, along with a rising serum sodium [51]. These patients often require the aggressive use of desmopressin acetate (DDAVPP) via the intravenous (IV) or subcutaneous route.

Medication Considerations

Impaired absorption of phenytoin with patients on enteral feeding is probably the most commonly known drug–nutrient interaction. Phenytoin is an anticonvulsant often used for the prevention and treatment of posttraumatic seizures. Holding EN for 1 h before and after each dose of the acid suspension (adequately shaken to ensure even particle distribution) appears to be feasible and effective option for circumventing this interaction [7]. If EN is to be held for phenytoin delivery, adjust the EN infusion rate to avoid underfeeding and verify by checking actual EN delivered into the patient (see Chap. 19 for modifying EN schedule with oral/enteral phenytoin). To avoid routine EN interruptions, the IV route for phenytoin administration may be a better option.

Propofol, a short-acting anesthetic and sedative, is commonly used in neurocritical patients because it is easily titrated to desirable clinical effects, and its actions are rapidly terminated with drug discontinuation [55]. In vitro, propofol has been shown to be neuroprotective against oxygen and glucose deprivation brain injury [56]. The oil source is soybean, composed of long-chain triglycerides (LCTs) and omega-6 fatty acid. This 10% lipid vehicle provides 1.1 kcal/mL (0.1 g/mL) calories in the form of fat, primarily linoleic acid. Because propofol also contains egg lecithin, it should be used with caution in patients who have known allergy to soy or eggs. The extra calories provided should be considered when recommending nutrition support

regimens. Institutions with sedation protocol in place should include a nutrition component, such as routine measurement of triglycerides and adjustment of nutrition support formulations to avoid overfeeding and ensure protein adequacy (see Chap. 19 for modifying EN regimen with propofol administration).

Metabolic suppression agents such as pentobarbital are used to induce a pharmacologic coma in an effort to decrease cerebral metabolism. Neurocritical patients receiving pentobarbital for refractory elevated ICP may have diminished energy requirement due to decreased cerebral and peripheral energy demands [57]. The use of these medications for the treatment of elevated ICP should be considered when estimating nutrition needs and plan of care. Pentobarbital-induced coma decreases the tone and amplitude of contractions of the GI tract; therefore, patients are more likely to not tolerate EN [58]. Routine use of bowel regimens with stool softeners and stimulants may delay or eliminate the development of a drug-induced ileus [7].

Nutrition in Stroke Rehabilitation

Enteral Nutrition

Swallowing difficulties that require placement of an enteral feeding tube to safely maintain adequate nutrition and hydration are common following a severe stroke. EN may represent a sole or supplemental source of nutrition support after stroke. Generally, EN as the sole source of nutrient intake is reserved for dysphagic patients for whom oral feeding is considered unsafe. However, failure to thrive non-dysphagic stroke patients may also be candidates for EN in the presence of prolonged and inadequate oral intake [59]. The use of feeding tubes in these stroke patients has been shown to reverse malnutrition [60]. A nasoenteric tube is often placed in patients who are expected to return to a full oral diet within 1 month. A permanent feeding access is indicated when a prolonged period of non-oral intake (>1 month) is anticipated (see Chap. 14).

There is a growing practice trend to refer more stroke patients who have high levels of medical severity and complexity to rehabilitation programs, a change that has shifted the management of many acute medical problems into the rehabilitation setting. This trend has brought to rehabilitation units more patients who need external support for nutrition, hydration, ventilation, and other physiological functions. One study showed that the presence of ≥1 medical tubes (enteral feeding tube, tracheostomy, or indwelling urethral catheter) is associated with more severe and disabling strokes, an increased number of medical complications, longer acute and rehabilitation hospitalizations, and greater resource use [61].

Ickenstein and colleagues investigated the predictors for removal of a feeding tube from stroke patients during rehabilitation and identified three negative predictors (bilateral stroke, aspiration during videofluoroscopic swallowing study, and age) [62]. A follow-up study by Ickenstein et al. reported that 11.6% of stroke patients admitted to a rehabilitation hospital required an enteral feeding tube secondary to

dysphagia, and only 45% of the patients were able to resume oral diets and have their feeding tubes removed [63]. In the same study, the authors gave a conservative estimate that a large percentage of patients with a feeding tube placement in the acute period after stroke will return to oral feeding within 3 months of stroke onset [63]. In another study, 47% of stroke patients were able to return to three meals daily within 5.5 weeks from onset of stroke, and 20% of the patients were able to have their feeding tubes removed prior to discharge from inpatient rehabilitation [64]. Factors associated with returning to three meals daily included gender (i.e., female), longer inpatient rehabilitation length of stay, and higher admission Functional Independence Measurement (FIM) scores [64]. The removal of the feeding tube was associated with patients being more likely to be discharged to the home environment.

Specialized Diets for Dysphagia

Nutrition therapies for dysphagia have been standardized by the Academy of Nutrition and Dietetics through the National Dysphagia Diet Task Force. The National Dysphagia Diet (NDD) was instituted in April 2002 to standardize food consistencies and terminology throughout the health care continuum. Dietitians, speech-language pathologists, and food researchers from this task force developed the NDD. It includes three levels of solid foods (dysphagia pureed, dysphagia mechanically altered, dysphagia advanced) and four levels of fluids (thin, nectar-thick, honey-thick, spoon-thick) [65]. See Table 13.4 for an overview of the three levels of the NDD [21].

Liquids may need to be thickened to the appropriate consistency as recommended by the speech language pathologist. Some patients may be on a regular solid consistency but still require altered liquid consistencies. Commercially available gel and powder thickening agents can be added to liquids to achieve the recommended consistency (nectar-thick, honey-thick, or spoon-thick). See Chap. 19 for a list of commercial and household liquid thickening agents. Pre-thickened beverages are also available and provide less variation in consistency compared with gel or powder thickening agents. Of concern, is that the thicker the liquid is made, the less fluid is usually consumed by the patient. Dehydration can be a potentially overwhelming problem for patients with dysphagia. Many of these patients will require an additional source for hydration either by intravenous fluids or by a feeding tube.

The level 1 NDD (dysphagia pureed) is designed for patients who have moderate to severe dysphagia with poor oral phase abilities and reduced ability to protect their airway. The diet consists of pureed, homogenous, and cohesive foods that have "pudding-like" texture. Foods that require bolus formation, controlled manipulation, or mastication are excluded.

The level 2 NDD (dysphagia mechanically altered) is indicated for patients with mild to moderate oral and/or pharyngeal dysphagia. This level consists of foods that are moist, soft-textured, and easily formed into bolus. This diet is used in transition from the pureed texture to a more solid texture. All foods from NDD level 1 are appropriate at this level.

Table 13.4
The national dysphagia diet

Food type	Breads	Cereals/grains	Dairy/desserts	Fats	Fruits	Meat/meat substitutes	Soups	Vegetables	Other
Level 1 Pureed *Recommend*	Pureed breads (pancakes, muffins, bread, etc.)	Smooth cooked cereals (farina type), pureed noodles	Smooth pudding, custard, pureed desserts	Butter, strained gravy, mayo, cheese sauces	Pureed fruits, thickened juices (no pulp, seeds, chunks)	Pureed meat or eggs, hummus, legumes	Pureed, thickened soup broth	Pureed vegetables, mashed potatoes, tomato sauce	Sugar, salt, spices, ketchup, honey, smooth jelly
Level 1 Pureed *Avoid*	Non-pureed breads	Dry cereals, oatmeal, cooked cereal with lumps	Ice cream, gelatins, non-pureed baked goods, chewy candy	Fats with coarse, chunky additives	Whole fruit (frozen, fresh, canned, or dried)	Whole or ground meat, non-pureed eggs	Non-pureed soups or broth with chunks	Non-pureed vegetables, tomato sauce with seeds or chunks	Coarse pepper, jams with seeds, nuts, sticky foods
Level 2 Mechanically altered *Recommend*	Soft pancakes with syrup, slurried breads	Cooked cereal with texture (oatmeal), dumplings with gravy, noodles in sauce	Pudding, custard, cake with icing, canned fruit, soft chocolates	Butter, gravy, sour cream, mayo, cheese sauce	Soft canned or cooked fruit without skin, ripe bananas, gelatin with canned fruit	Moistened ground meats in gravy/ sauce, pasta, scrambled eggs	Soup with veggies or meat (<½in. in size)	Well-cooked vegetables, boiled or baked potatoes	Jams and preserves without seeds, salsa
Level 2 Mechanically altered *Avoid*	Dry breads	Dry, course cereals, cereals with flax, seeds, or nuts	Chewy candy, dry cakes/ cookies, yogurt with nuts	Fats with coarse, chunky additives	Pineapple, fruit with skin or seeds, dried fruit	Dry meat, bacon, sausage, hot dogs	Large chunks of meat, rice, corn in soup	Broccoli, asparagus, celery, corn, peas, potato skins	Nuts, seeds, sticky foods, coconut

Level 3 Advanced *Recommend*	Moist breads (butter, jam, syrup)	Moist cereal, rice, pasta	All others except those in avoid category	All others except those in avoid category	Canned or cooked fruit, peeled fresh fruit	Ground tender meats and tender fish	All others except those in avoid category	Tender vegetables, shredded lettuce, fried potatoes	Seasoning, sauces, jelly, jam, honey
Level 3 Advanced *Avoid*	Dry bread, toast, crackers	Dry cereals, shredded wheat bran	Dry baked goods, nuts, taffy, coconut	Course spreads with nuts	Dried fruit, fruit snacks, fruit with pulp/ skin	Tough dry meat, fish with bones, chunky	Chowders, large pieces of vegetables	Raw, rubbery, stringy vegetables	Chunky peanut butter

Recommendations by type of food and consistency (Reprinted with permission from Sage Publications [21])

The level 3 NDD (dysphagia advanced) is a transition to a regular diet. This level consists of food of nearly regular textures except for very hard, crunchy, or sticky foods. The foods still need to be moist and should be in bite-size pieces. At this level, patients should be assessed for ability to tolerate mixed textures. This diet level is appropriate for patients with mild oropharyngeal dysphagia [65]. See Chap. 19 for dysphagia diet sample menus. Eating frequent, small meals may help increase food intake. After patients demonstrate the ability to tolerate these foods safely, the diet can be advanced to a regular diet without restrictions.

Weaning Enteral Nutrition

The goals of rehabilitation include enhancing function and returning to normal living, which includes oral eating. Weaning stroke patients from tube to oral feeding is a primary nutrition goal and can take place in the acute setting, during stroke rehabilitation, or at home. Transitional feeding requires an interdisciplinary approach involving the speech language pathologist, dietitian, nurse, and physician. Regularly scheduled evaluation of tube-fed patients by a speech language pathologist is necessary to identify positive changes in swallowing function that can permit transition from tube to oral feeding [21]. Feeding the stroke patient requires not only routine nutrition assessment by the dietitian, but also assistance from nursing for patients who cannot feed themselves, as well as assistance from occupational therapists for evaluation of assistive feeding devices. After clinical, diagnostic, and instrumental evaluations, the speech language pathologist will recommend the appropriate food and liquid consistencies for the initial oral diet. A modified barium swallow (MBS) study can help guide decisions about feeding regimens and estimate the stroke patient's risk of respiratory complications from oral feeding. It allows the speech-language pathologist to determine whether some type of compensatory swallowing strategy can circumvent a problem and actually experiment with that strategy during the examination to assess its benefit. In addition, an MBS study may also allow the patient to continue eating safely, even full nutrition and hydration cannot be maintained by mouth.

The weaning process, nutrition goals, and plan of action should be discussed thoroughly with the patient, family members, and the health care team. Continuous tube feedings are often transitioned to cyclic feedings at night and oral feedings during the day to potentially stimulate the hunger sensation. EN can infuse between 8 and 20 h and provide anywhere from 25 to 75% of the requirement during the time of transition. A calorie-dense EN formula may be needed to achieve this amount of calories in a shortened time frame. Bolus or intermittent feeds can be implemented to offer a more normal timing of meals and to accommodate other therapies. Oral nutrition supplements can be given between meals or as part of a meal to achieve caloric goals. Adequacy of oral intake dictates adjustment in tube feeding volume and consequent success of feeding tube removal [21]. Documenting the patient's intake during the transitional phase can be cumbersome, yet it is critical in deciding

when to discontinue the tube feedings. The total time to wean from tube to oral feeding is patient dependent, and may not be a goal shared by all patients [66]. Crary and Groher described that the process of transitioning from tube to oral feeding can be challenging both cognitively and physically for some patients [67]. At a minimum, tube-fed patients with dysphagia who are candidates to return to oral feeding must be able to consume adequate oral nutrition and demonstrate a safe and efficient swallow on a consistent basis [67].

Buchholz proposed a 2-phase clinical algorithm, specific to patients with stroke or acquired brain injury, for transitioning tube-fed patients to oral feeding during acute rehabilitation [66]. The initial phase, termed the *preparatory phase*, focuses on medical and nutrition stability, swallowing assessment, and implementation of an intermittent tube feeding schedule. The *weaning phase* is a described as gradual progression in oral feeding with corresponding decreases in tube feeding. Once a patient is able to consume ≥75% of his or her nutrition requirements consistently for 3 days, all tube feedings are discontinued. Weight, hydration, and swallowing ability are closely monitored during these stages with a specific focus on respiratory complications.

A safe swallow function does not guarantee that the stroke patient, especially the elderly, will be able to ingest adequate oral nutrition. As with any change, the goal of transitional feeding is long-term success. If at any time in the progression toward oral intake a stroke patient is unable to maintain adequate consumption, other measures should be considered. In such cases, it is recommended that the patient's nutrition be maintained through partial oral feeding with supplemental tube feeding as required.

Summary

Malnutrition is a common finding following a stroke and nutrition support is common in patients with severe neurological injury. Whether the patient requires modified oral diets for dysphagia, enteral tube feedings, or PN, a registered dietitian is integral in managing the nutrition care of stroke patients. In addition to the dietitian, implementing the nutrition care plan relies on the interdisciplinary team of nurses, physicians, pharmacist, SLP, and occupational therapy as meeting nutritional goals relies on many non-nutritional factors.

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