
Nutrition Support in the Stroke Patient

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Stroke is a leading cause of disability in the elderly and a significant cause of disability in younger people as well. Nearly 3 million Americans have some degree of impairment from strokes, at an estimated annual economic impact of 30 billion U.S. dollars (1). Seventy-five percent of all cerebrovascular accidents occur in persons above the age of 65 years, and nutritional problems are commonly seen on admission to the hospital. Several worldwide studies have reported nutritional status of the elderly, with similar results showing a high prevalence of protein-energy malnutrition in institutionalized and home-bound elderly alike (2–4). We review general concepts and specifics concerning nutritional assessment and management of the stroke patient.

NUTRITIONAL ASSESSMENT

Determining nutritional status in humans is the object of numerous studies. Pertinent history taking (weight loss, edema, anorexia, vomiting, diarrhea, decreased or unusual food intake, and chronic illness), physical examination with emphasis on anthropometric indices (loss of subcutaneous fat, muscle wasting, and edema) and evidence of vitamin deficiencies are crucial. No rigid system of scoring is used but patients can usually be divided into one of three classes: (a) normal nutritional state, (b) mild malnutrition, (c) severe malnutrition (5). As adjuncts, anthropometric measurements such as weight and height derived using reference tables such as the Metropolitan Life Insurance reference weights (6) and the Health and Nutrition Examination Survey (HANES) normative values (7), can be employed.

The degree of malnutrition can be assessed comparing actual to ideal body weight as shown in Table 1. Visceral protein status markers include serum albumin, transferrin, and lymphocyte count. An albumin concentration of less than 3.5 g/dl is associated with poor nutritional status and increased mortality in nursing home residents and community dwellers alike (9,10). A total lymphocyte count less than 2000 cells/mm$^3$ and a transferrin concentration of less than 200 mg/dl are also considered markers of malnutrition as seen in Table 2.

Several caveats with regard to interpretation of these variables should be discussed. Albumin has a half-life of 20 days. Albumin concentration thus indicates
TABLE 1. Determination of malnutrition by percentage of ideal body weight (8)

<table>
<thead>
<tr>
<th>Percentage of ideal body weight</th>
<th>Actual weight/ideal body weight</th>
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<tbody>
<tr>
<td>Mild malnutrition</td>
<td>Body weight 85% of ideal</td>
</tr>
<tr>
<td>Severe malnutrition</td>
<td>Body weight 80% of ideal</td>
</tr>
<tr>
<td>Life-threatening malnutrition</td>
<td>Body weight 70% of ideal</td>
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Baseline visceral protein but is not adequate to assess rapid changes in nutritional status. In this situation, nutrition-sensitive serum proteins such as retinol binding protein with a half-life of 10–12 hours and thyroxin-binding prealbumin (transthyretin) with a half-life of 2–3 days are useful (12). Serum transferrin has a half-life of approximately 8 days, reflecting intermediate term changes in visceral protein status. Total lymphocyte count is altered by diverse factors: drug therapy, acute illness, stress, infection, and neoplastic disease (13). A nutritional marker indicative of high mortality rate in nursing home residents is a serum cholesterol concentration below 160 mg/dl (14).

Normal adults typically require 25 kcal/kg body weight to meet resting energy expenditure (REE). Stressed or post-injury patients require at least 1.2 times REE. Energy expenditures have been calculated for many clinical conditions with neurosurgical trauma having one of the highest requirements (up to 2.5 of predicted REE) (15).

Besides energy intake, protein nutrition is of major concern. Protein is required to prevent or ameliorate nitrogen losses brought about by the catabolic state of many disease processes. Age-related changes in the metabolism of protein include a reduction of both synthesis and turnover and an inability to synthesize additional albumin in response to a diet higher in protein (16). It appears that most patients will achieve nitrogen balance with diets supplying 1 to 2.5 g/kg body weight per day depending on metabolic condition, with those patients in hypercatabolic state requiring maximum supplementation. Potential factors limiting protein intake are liver and renal failure.

ENERGY EXPENDITURE AND NUTRITIONAL STATUS IN STROKE PATIENTS

Current information on metabolic expenditure and supply during neurological disease is derived from work with head trauma subjects (17–19,22). There is an

TABLE 2. Nutritional markers of visceral protein (11)

<table>
<thead>
<tr>
<th>Marker</th>
<th>None</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
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<tbody>
<tr>
<td>Serum albumin (g/dl)</td>
<td>&gt;3.5</td>
<td>2.8–3.5</td>
<td>2.1–2.7</td>
<td>&lt;2.1</td>
</tr>
<tr>
<td>Serum transferrin (mg/dl)</td>
<td>&gt;200</td>
<td>151–200</td>
<td>100–150</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Total lymphocyte count (per mm³)</td>
<td>&gt;2000</td>
<td>1200–2000</td>
<td>800–1100</td>
<td>&lt;800</td>
</tr>
</tbody>
</table>
obvious direct effect between the severity of the neurologic injury and energy expenditure, especially when abnormal posturing and motor agitation occur. Values can reach 250% of that predicted. Outcomes in terms of mortality and lengths of stay for these neurosurgical patients are improved when early nutritional intervention is implemented. Data can be extrapolated to stroke patients who share similar neurologic abnormalities, especially during the acute phase. In 1988, Axelsson (20) reported the nutritional status of 104 patients admitted with stroke to a nonintensive care unit in a university hospital in Sweden. Six nutritional indicators were obtained: three anthropometric values (relative weight, triceps skinfold thickness, and arm muscle circumference) and three circulating proteins (albumin, transferrin, and prealbumin). On admission, 16% of the patients had two or more indicators below the normal range. During the hospital stay, a few patients—especially those under 65 years of age—improved their nutritional status but, overall, 22% of the original patients left the hospital with two or more indicators of malnutrition. Those identified as malnourished on admission had longer hospital stays and higher mortality than those with normal nutritional status.

Stroke itself places the patient at risk for protein-energy malnutrition because of several factors that are shown in Table 3. The neurological factors shown in Table 3 often impair independent oral intake (21). Dysphagia has obvious implications for inadequate nutritional intake and should be recognized early to prevent complications of aspiration and pneumonia (23). Dysphagia evaluation includes clinical examination of different phases of swallowing mechanism, video-fluoroscopic examination and occasionally, endoscopic studies (24). Dysphagia with aspiration occurs in up to half of patients with stroke, as determined by clinical and videofluoroscopic indices. Patients with combined cerebral-brainstem strokes with bilateral cranial nerve signs, weak cough, and dysphonia have a higher frequency of dysphagia and should be assessed promptly (25). About one third of patients with aspiration have "silent aspiration," not obvious without a video-fluoroscopic swallowing examination, whereby penetration of saliva or food below the level of the true vocal cords is detected in the absence of cough or other signs of respiratory distress. These individuals are also at an increased risk for aspiration and pneumonia (26). Dyspha-

<table>
<thead>
<tr>
<th>Neurological factors</th>
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<tbody>
<tr>
<td>Paralysis (involvement of the dominant side, poor mastication)</td>
</tr>
<tr>
<td>Dysphagia (with inadequate energy intake and risk of aspiration pneumonia)</td>
</tr>
<tr>
<td>Perceptual deficits (neglect syndrome, hemianopsia)</td>
</tr>
<tr>
<td>Motor apraxia (inability to feed self)</td>
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<table>
<thead>
<tr>
<th>Nutritional factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior impaired nutritional status</td>
</tr>
<tr>
<td>Elderly</td>
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<table>
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<tr>
<th>Metabolic factors</th>
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<tbody>
<tr>
<td>Depression</td>
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<td>Isolation</td>
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<table>
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<tr>
<th>Psychological factors</th>
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<tbody>
<tr>
<td>Depression</td>
</tr>
<tr>
<td>Isolation</td>
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</table>
TABLE 4. Sequelae of malnutrition in the stroke patient

- Impaired musculoskeletal system—loss of somatic protein/decreased skeletal muscle mass/weakness
- Impaired immune response—increased morbidity/mortality from septic complications
- Impaired wound healing—risk for decubitus ulcers

gia, however, is usually transient with 80% of patients recovering within 3 months after the initial event (27). Several rehabilitative exercises and training appliances have been designed for such patients with encouraging functional outcomes (28–30).

Concerning metabolic factors, there is a dearth of studies evaluating energy expenditure in stroke patients. It is known that 30% of severe head trauma patients have high metabolic rates (22). Whether some stroke patients have similarly high metabolic rates remains to be determined. Nevertheless, stroke places a patient at risk for a number of hypercatabolic conditions such as aspiration pneumonia and sepsis.

Psychological factors play an important role in long-term nutritional management and rehabilitation. For example, depression occurs in up to 44% of patients 3 months after stroke and 30% remain depressed by 12 months (1). Clinicians should, therefore, recognize psychiatric complications which are amenable to effective treatment.

As a result of the neurologic, nutritional, metabolic, and psychological factors mentioned, acquired physical manifestations of malnutrition may interfere with the rehabilitation process as depicted in Table 4.

METHODS FOR NUTRITIONAL SUPPORT

A rational approach to nutritional support of the stroke patient during the first few days (acute phase) should include provision of adequate fluid, energy, and nutrients, while minimizing potential complications of therapy. Close monitoring of fluid balance and electrolyte levels is mandatory in this phase of stroke because of raised levels of antidiuretic hormone with resulting hyponatremia, which can aggravate already impaired neurological status and brain edema (31). Another major metabolic abnormality—hyperglycemia—has been characterized as a predictor of mortality in these patients (32,33). Some investigators believe hyperglycemia is a cause of poor outcome because of animal studies showing larger areas of brain infarction in hyperglycemic cats with mechanically occluded cerebral arteries. It is hypothesized that there is a brain shift to anaerobic metabolism with larger amounts of lactic acid, subsequent acidosis, and neuronal death. Studies in rats and rabbits, however, show smaller areas of infarction with a similar protocol. At present, hyperglycemia is thought to be the result of the stroke and to be due to the release of stress-related circulating hormones such as cortisol and glucagon (34). From a practical standpoint, standard intravenous fluids should be given in the first hours after an acute stroke. Hyperglycemia can be managed with judicious doses of insulin and
fluid therapy. In head trauma patients, total parenteral nutrition (TPN) has not resulted in higher intracranial pressure or other neurological complications (35). In fact, several studies have reported enhanced recovery and increased survival in neurosurgical patients treated with TPN (18,19). Whether head trauma studies can be extrapolated to stroke patients is not clear, but we want to emphasize that in our view—as is discussed later—most stroke patients should obtain enteral rather than parenteral nutritional support, as the vast majority have an anatomically and functionally intact gastrointestinal system.

Once the acute phase is resolved and the stroke is completed, patients should be reassessed both nutritionally and functionally to evaluate their capacity for self-feeding and for meeting their calculated energy and nutritional requirements. Special attention should be given to the risk factor analysis presented in Table 3.

Recently, a clinical decision algorithm has been published by the American Society for Parenteral and Enteral Nutrition (ASPEN) to help the clinician determine the route and type of nutritional supplementation as shown in Fig. 1.

As just mentioned, enteral nutrition is preferable to parenteral therapy provided there are no contraindications and access can be safely attained, mechanical obstruction being the only absolute contraindication (42). Early enteral feeding of stroke patients results in a shortened hospital admission time (37), while parenteral nutrition carries a higher risk for both catheter-related and noncatheter-related infections (38). This excess of noncatheter-related infections could in part be related to the deterioration of the intestinal integrity in the absence of intraluminal fuels, resulting in translocation of gut bacteria (39,40).

The guidelines published by ASPEN (41), and more recently by the American Gastroenterological Association specifically for tube feedings (42), are helpful in deciding which enteral method of feeding to use when oral intake is not possible due to oropharyngeal dysphagia. These guidelines assume the gastrointestinal tract is available, as is the case in the great majority of these patients. Nasogastric or nasoenteric tubes are recommended for short-term feedings of less than 30 days. For those patients with a history of tube feeding related aspiration pneumonia, tubes should be placed in the third portion of the duodenum or beyond the ligament of Treitz to decrease such complication. Percutaneous endoscopic gastrostomy (PEG) tube placement is rapidly becoming the preferred method of long-term enteral nutritional support. Several papers show stroke as the indication for 30–80% of all patients receiving percutaneous endoscopic or surgical gastrostomies (43–47).

Table 5 gives results from two prospective randomized studies comparing nasogastric versus PEG tubes on patients with neurologic dysphagia. Both studies show important advantages of PEG over nasogastric tube feedings. PEG patients receive a significantly greater proportion of their prescribed feeds with attendant improvement in nutritional variables. Both studies also demonstrate that PEG tube feedings have fewer treatment failures. Even more important, an article by Norton and colleagues (48) indicated that PEG is associated with a lower 6-week mortality than nasogastric feedings. Finally, gastrostomy patients are more likely to be discharged earlier.
ROUTES TO DELIVER NUTRITION SUPPORT IN ADULTS

NUTRITION ASSESSMENT

Decision to initiate nutritional supplementation

FUNCTIONAL GI TRACT

YES

ENTERAL NUTRITION

Long term
Gastrostomy
Jejunostomy

Short term
Nasogastric
Nasoduodenal
Nasojejunal

PARENTERAL NUTRITION

Short term
Peripheral PN*

Long term or fluid restriction
Central PN*

GI FUNCTION RETURNS

YES

NO

GI Function*

Normal
Intact Nutrients (c)

Compromised
Defined formula (b)

NUTRIENT TOLERANCE

Adequate
Progress to Oral feedings
supplementation

Inadequate
PN supplementation

Adequate
Progress to more complex diet and oral feedings as tolerated

FIG. 1. (36) Reprinted from the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.), Guidelines for the Use of Parenteral and Enteral Nutrition in Adult and Pediatric Patients, JPEN; 17(4):1SA-52SA. A.S.P.E.N. does not endorse this material in any form other than its entirety.

(a) Feedings may be more appropriate distal to the pylorus if the patient is at increased aspiration risk.

(b) Elemental low/high fat content, lactose free, fiber-rich and modular formulas should be provided according to the patient's GI tolerance. See specific conditions for formula guidelines.

(c) Polymeric, complete formulas, or pureed diets are appropriate.

PN—Parenteral Nutrition
GI—Gastrointestinal
### TABLE 5. Studies comparing nasogastric tube with percutaneous endoscopic gastrostomy tube feedings in patients with neurological dysphagia

<table>
<thead>
<tr>
<th>Variable Studied</th>
<th>Norton B et al (48)</th>
<th>Park RHR et al (49)</th>
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<tbody>
<tr>
<td></td>
<td>PEG (n = 16)</td>
<td>PEG (n = 20)</td>
</tr>
<tr>
<td></td>
<td>NG (n = 14)</td>
<td>NG (n = 20)</td>
</tr>
<tr>
<td></td>
<td>p Value</td>
<td>p Value</td>
</tr>
<tr>
<td>Six-week mortality (%)</td>
<td>12</td>
<td>N/A</td>
</tr>
<tr>
<td>% Of prescribed feeding given</td>
<td>100</td>
<td>93</td>
</tr>
<tr>
<td>Change in nutritional status</td>
<td>-0.3*</td>
<td>-1.4†</td>
</tr>
<tr>
<td>Treatment failure (%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>% Discharged at 6 weeks</td>
<td>42</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>57</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td>71</td>
<td>94</td>
</tr>
<tr>
<td></td>
<td>-0.8*</td>
<td>-0.6†</td>
</tr>
<tr>
<td></td>
<td>-0.003</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>N/A</td>
<td>N/A</td>
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* measured as changes in albumin concentration expressed in g/dl; † weight gain in kg after 1 week of treatment; N/A = not applicable.

Complications may occur as a result of nasogastric and PEG tube insertion. Common nasogastric tube complications include nasal trauma, acute and chronic sinusitis, tube clogging, tube dislodgement, gastroesophageal reflux, and aspiration. Rare nasogastric tube complications include bronchial intubation with feeds administered into the lungs, pneumonia, pneumothorax, empyema, visceral perforation, otitis media, arrhythmia, myocardial infarction, and gastrointestinal bleeding (42). Nasogastric tubes are frequently dislodged, with mean tube lifespan of 10 days, underscoring their use as short-term devices (42,50). Their replacement and repositioning only increase the probability of complications.

PEG tubes are also subject to complications. In the largest single center study to date, PEG placement was successful in 95% of 314 consecutive patients. Procedure-related mortality was 1%. Three percent had major complications and 13% minor complications (51). In general, complications of PEG tubes are minor and easily handled. Clinicians should be aware of two infrequent but serious complications of PEG placement, namely, necrotizing fasciitis and colocutaneous fistula.

**Necrotizing fasciitis** involves a necrosis of the superficial layers of the abdominal fascia. If not diagnosed and treated aggressively with surgical debridement, associated mortality is high. The condition usually becomes evident 3–14 days after the procedure with fever, skin edema, cellulitis, and eventually crepitus. Possible contributing factors are lack of prophylactic antibiotics and technical problems such as small abdominal incision and excessive traction on the tube (42). A single pre-procedure dose of parenteral antibiotics has been effective in reducing wound infections in a prospective, randomized double-blind study (52).

**Colocutaneous fistula** is an uncommon PEG complication usually manifested as persistent diarrhea, and in most cases, the fistula closes upon removal of the catheter.

Two common problems associated with tube feedings are bronchial aspiration and diarrhea (42). To minimize aspiration, the following practical considerations are useful:
Bed elevation (30°–45°) during feeding
Intermittent or continuous feeding rather than rapid bolus
Gastric residual should be checked periodically
Consider jejunal access in patients with recurrent tube feeding (not oropharyngeal) aspiration or in critically ill patients with high risk for gastric motility dysfunction

Diarrhea is a common but poorly defined complication of enteral feeding. Multiple etiologies have been identified (53), some of which are not directly related to the tube feedings but rather are complications of other treatments (antibiotics, magnesium-, and sorbitol-containing preparations). Other causes are related to the impaired nutritional status such as severe protein-energy malnutrition inducing decreased absorptive and digestive capacity. Severely reduced serum albumin (below 2 g/dl) is thought to be partially responsible for diarrhea in critically-ill patients. Factors related to the formula itself include rapid rates of infusion, high osmolality, and microbial contamination. Colocutaneous fistula has already been mentioned as a rare complication of PEG placement.

Regarding the type of feed, the standard isotonic polymeric formulas are appropriate for most patients with stroke and will meet their nutritional requirements when given in suitable amounts. Elemental formulas should be reserved for the exceptional patient with intestinal disease resulting in malabsorption. Specialty formulations have no role in these patients.

ETHICS

There are ethics considerations to be taken into account when considering nutritional support (54,55). These include the following:

The patient's wishes should always be paramount.
The benefits and burdens of tube feeding should be assessed for the individual, the patient being the final arbiter.
A therapeutic trial of enteral feeding should be discontinued when burdens outweigh benefits.

Furthermore, gastrostomy probably should not be indicated if life expectancy is less than 30 days from the time of placement.

POTENTIAL ROLE OF GROWTH FACTORS
AS NUTRITIONAL ADJUNCTS

The patient with stroke is at particular risk for rapid loss of skeletal muscle and other components of lean body mass due to immobilization as a result of neurological impairment. This situation is aggravated when catabolic complications such as aspiration pneumonia occur in an older individual, who most probably will already have an age-related decline in lean body mass. It has been shown that the long-term administration of human growth hormone (hGH) to healthy elderly individuals with declining growth hormone secretion is capable of reversing some of the age-related
changes in body composition by increasing lean body mass and decreasing adipose tissue mass (56). In short-term studies, the administration of hGH has been shown to improve the anabolic effect of parenteral nutrition in medically stable individuals (57), decrease the catabolic response to major surgery (58), and improve serum protein concentration and other indices of nutritional repletion in enterally fed, immobile trauma patients (59). Thus, it would be of great interest to evaluate the effects on body composition and function of short-term hGH administration in immobilized stroke patients combined with appropriate nutritional support and physical therapy.

CONCLUSION

To conclude, this review describes significant progress in nutrition support. These advances have been successfully applied to the patient with stroke. Areas of specific interest to these patients that need further research include the optimum nutritional management of the acute phase of stroke in relation to energy expenditure and to brain edema; outcome studies are also needed both in the acute phase as well as in the long-term nutritional management. The potential role of human growth hormone and other growth factors in preventing loss of lean body mass and enhancing rehabilitation should also be investigated.

REFERENCES


36. ASPEN Board of Directors. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. *JPEN* 1993; 17 (suppl): 7SA.


DISCUSSION

Dr. Guesry: We have heard how a high blood glucose may induce lactic acid production in the brain together with acidosis, which would put the patient into a vicious circle of acidosis, edema, and increased lesion size. You mentioned that if the patient is malnourished and hypercatabolic, you are inclined to start the TPN immediately. Could you suggest any substitute for glucose because the brain has to be fed but is only able to use glucose or ketones, which are also acidogenic?

Dr. Feller: What I said is we try to use enteral nutrition as much as possible. If the gastrointestinal tract is available and intact, we stay away from TPN. Maybe I was not sufficiently clear: we try not to use TPN but we do use enteral nutrition. I think the solution is to try to control the hyperglycemia as much as possible. The solution I see as an internist is to give insulin if necessary. Probably these patients are under severe stress. Severe stress means increased glucagon, catecholamines, corticoids, and growth hormone, and in most cases, this is the reason for the hyperglycemia. If the patient is not malnourished and is not hypercatabolic, I would have no problem in just giving saline for 2 or 3 days.

Dr. Ginsberg: Experimentally, it has been shown that the period of risk for hyperglycemia is in the first few hours and probably only after an ischemic stroke. After that, the acidosis mechanism has probably played itself out unless there is a recurrence of the stroke, so clinical
caution should probably dictate that one restrict glucose in the first day or two, but not chronically. The question I would like to ask is about growth hormone. What are the risks and disadvantages of the use of growth hormone for muscle atrophy, in elderly people for example?

Dr. Feller: The potential value of this in the stroke patient is in the initial phase in order to prevent muscle atrophy at the same time as starting rehabilitation. If you use it for 1 or 2 weeks, or perhaps a month, there should not be any problems. The main adverse effect in the short-term is salt retention. That could be a problem but it is easily taking care of. Growth hormone is of course also diabetogenic, so you have to be careful about that. However, none of our patients was diabetic. When we supplemented growth hormone for 12 months, we had long-term complications, but these do not occur in the initial stage of treatment.

Dr. Milon: In your study on the growth hormone, did you test the functionality of the increased muscular mass?

Dr. Feller: Yes, we did. Muscle strength increases, but obviously we are aware that exercise also increases muscle strength in nonagenarians. However, at 3 months, 50% of the effect had been lost, which means that the way to maintain the muscle mass is probably to exercise. Exercise induces release of growth hormone, so we think that a way of maintaining that is to increase exercise.

Dr. Milon: These subjects were not exercising?

Dr. Feller: No, most of them were sedentary elderly people.

Dr. Hennerici: Are you aware of any data to suggest that patients with strokes of different brain regions, say brain stem versus hemisphere or insular region, vary in the degree to which they get obvious endocrine problems or gastrointestinal problems?

Dr. Feller: No, I don’t know any studies on this, but I think it is an important concern. For instance, one should recall that patients with brainstem stroke have a greatly increased incidence of dysphagia compared to other strokes. Some types of strokes are also more hypercatabolic than others.

Dr. Leweling: Some patients have to be fed intravenously. Most amino acid solutions contain glutamate. Can we use these solutions?

Dr. Feller: I don’t know, but I do know that patients who have a stroke and need TPN are very rare, for example, a patient with short bowel syndrome who also has a stroke will be the exceptional case. Most patients have an intact GI tract and in this case, there is no need for TPN.

Dr. Leweling: But some have to be mechanically ventilated and then, they have to be relaxed. In this case, gut motility is absent or greatly reduced and we have to feed them parenterally.

Dr. Feller: Why not do a gastrostomy?

Dr. Lavel: The food is not transported in the gut because the gut is atonic.

Dr. Feller: That must be very exceptional. I think I have seen one case in 5 years where TPN was necessary and in that case, we gave him the usual TPN formula without problem. However, the glutamate problem didn’t occur to me. Maybe Dr. Ginsberg has an answer to that. What do you think?

Dr. Ginsberg: Glutamate is normally excluded by the blood-brain barrier. The original observations had to do with glutamate producing toxicity in zones of the brain that lack the barrier—in the circumferential organs and so on—or in pathologic state in which the barrier has broken down. There is blood-brain barrier linkage in stroke and so that might be a reason to avoid a high glutamate intake in the early post-stroke period. To my knowledge, this has not been studied but I don’t think dietary glutamate poses a risk for the brain.