Gastrointestinal Dysfunction in Stroke

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The importance of central nervous system control of gastrointestinal function has been known since Pavlov showed increased acid secretion following sham feeding of esophagostomized dogs. The effect of stroke on gastrointestinal function has gained little interest compared to its more apparent effects on cognitive, perceptual, and neuromotor function. Clinical knowledge relating stroke pathology to altered gastrointestinal physiology, like all medical knowledge of the late twentieth century, is a hodgepodge of carefully controlled clinical and physiologic experiments, astute clinical observations, good basic scientific research, and a decent number of myths, some based in reality, some waiting to be debunked. Our awareness of the effects of stroke on gastrointestinal function is increasing, with much yet to be learned. In this article, we describe associations between stroke and gastrointestinal function as learned from animal models and from clinical practice.

EFFECTS OF STROKE ON ORAL, PHARYNGEAL, AND ESOPHAGEAL FUNCTION

Dysphagia is the most important gastrointestinal complication following stroke. Large epidemiologic studies have shown that the frequency of dysphagia among patients admitted to an acute hospital for stroke approaches 45%.1 The more severe the stroke the greater the frequency of dysphagic complications.2 Suppression of sensorium is linked to the severity of dysphagia and is an obvious indicator of who is unsafe to manage oral feeding.3 The frequency of dysphagia is lowest for patients with unilateral ischemic hemispheric strokes, increases for those with bilateral hemispheric lesions, and is greatest for patients with brain stem lesions.2 Early studies indicated that oral-phase problems were more frequent among patients with left hemisphere lesions, and aspiration was more commonly seen with right hemisphere lesions.4 Subsequent work indicates that the frequency of dysphagia and aspiration is probably equal for patients with right and left hemispheric lesions.5,6

Dysphagia predisposes the patient to many complications: pneumonia, dehydration, calorie-nitrogen loss, and upper-airway obstruction.7,8 The severity of dysphagia and risk of complications are best defined by modified barium swallow (MBS) videofluoroscopy. This technique, first popularized by Logemann,9 utilized small volumes of test material to assess swallowing function: 1/3 tsp of liquid barium, 1/3 tsp of barium paste, or 1/4 of a cookie coated with barium. Small volumes were thought necessary to ensure safety when patients aspirated some of the barium. With time it became apparent that more physiologic amounts (20 to 30 ml) of barium were safe and that they more closely approximated the problems encountered with oral feeding.10

The severity of dysphagia can be assessed by the amount of material aspirated, by the consistency of material aspirated, or by the timing of aspiration. Simple penetration of the test bolus into the laryngeal vestibule occurs in about 5% of normal older volunteers.11 Aspiration of test material past the vocal folds is abnormal, and carries an increased risk of medical complications.7,11 The greater the amount of material aspirated, the greater the risk. Aspiration of thickened liquids or more solid consistencies indicates more severe dysphagia and has a greater risk of medical complications than aspiration of thin liquids only.7 There are no generally accepted criteria to define patients who are deemed to be unsafe oral feeders and in need of nonoral feeding systems. In our practice, patients who aspirate more than 10% of each test consistency and who cannot use compensatory swallowing strategies to eliminate aspiration are given nonoral feeding.12 Nonoral feeding is initiated with a medium-bore nasogastric tube, which allows liquid feeding and crushed medications to be given without clogging. Small-bore tubes have been advocated as less likely to be associated with gastroesophageal reflux, though this has not been shown in prospective trials. To our knowledge there are no data to show any advantage to small-bore tubes, which are more difficult to place and keep in position. If after 2 weeks the patient continues to require nonoral feeding, a percutaneous gastrostomy is placed.

It is inappropriate to refer all stroke patients for MBS evaluation before allowing oral feeding. A patient who is alert and can hold a cup in the unaffected hand and drink 3 oz of water without coughing or evidencing gurgling voice quality is probably safe with oral feeding.13 Such patients may need pureed diets to obviate
problems with buccal, lingual, and masticatory aspects of swallowing, but they are at low risk for medically significant aspiration. Patient-specific diet prescriptions and choice of appropriate compensatory swallowing techniques can await formal MBS evaluation and dysphagia therapy.

Pneumonia occurs in 8% of nondysphagic and 19% of dysphagic patients following stroke. It is usually seen during the first 2 weeks following stroke. In one study, 84% of patients who developed pneumonia did so within the first 5 days of hospital admission. Most of these had a significantly suppressed sensorium and had not been given oral feeding or liquids. Pneumonia due to such situations is not amenable to dysphagia treatment, but requires intubation for airway protection. Pneumonia developing after oral feeding has been initiated occurs in about 7% of dysphagic patients given diet consistencies and swallowing techniques prescribed after MBS evaluation. That pneumonia develops in 12% of patients receiving chronic nonoral feeding indicates that such measures offer only limited protection.

Aspiration of acid gastric contents is known to be a potent source for tracheal-bronchial bacterial contamination and chemical irritation. Since aspiration pneumonitis is usually not preceded by vomiting, it is probably due to contamination by oral-pharyngeal secretions, food, or liquids rather than by gastric contents. The airway is protected from oral secretions while subjects are awake, but a small amount of aspiration during sleep is common in normal people. Such aspiration may be more voluminous in stroke victims, who have altered sleep architecture and increased frequency of sleep apnea compared with age-matched controls. Many liquids have an acid pH (cola 2.4 and coffee 4.9, for example), and if aspirated while swallowing might be equally as irritating to the tracheal-bronchial tree as gastric contents. No one has suggested in the medical literature that dysphagic patients should be given buffered liquids to drink.

Dehydration, another complication of dysphagia, occurs in 19% of patients with acute stroke. It is also commonly seen in patients without dysphagia but who show suppression of sensorium, confusion, or aphasia. Such problems interfere with the patient’s ability to recognize or respond to thirst. Dehydration due to dysphagia is often improved by providing the patient with thickened liquids, which allow better control of the bolus as it passes from the oral cavity through the pharynx. The increased viscosity lessens premature spillage of the bolus over the epiglottis before it has had time to close the laryngeal vestibule. Urine specific gravity, in the absence of diuretic usage or primary renal insufficiency, is a convenient marker for fluid balance. It is a sensitive and reliable measure of response to fluid intake and can be checked daily without need for phlebotomy. Intake and output charts are difficult to estimate and are accurate only for patients with intravenous or nonoral fluid intake. Puree-consistency foods provide 75% of their weight as water and are a ready source for increasing water intake in dysphagic patients.

Calorie-nitrogen deficit and upper airway obstruction are seen with more severe dysphagia. Patients are often aware of their difficulty with swallowing and are frightened or exhausted by the struggle to eat. They may cough or choke with each swallow. Providing a chopped, soft-solid, or pureed diet obviates the need to chew and can remove the threat of upper airway obstruction. Patients with more than a 5 to 10 second delay in the swallow reflex in addition to their increased risk of aspiration require such prolonged feeding times that they become fatigued before adequate calories have been consumed.

Dysphagia following stroke is categorized as due to problems with one or more of the four phases of deglutition: anticipatory, oral and pharyngeal esophageal. Stroke affects the anticipatory phase of feeding by its effect on appetite. Loss of appetite may be due to depression (commonly seen following stroke), to inactivity with decreased energy expenditure, or to other as yet undefined neuroendocrine responses to brain injury. The linkage between depression and resistance of endogenous cortisol production to dexamethasone suppression is well established. Similar linkages have been observed with prolactin, luteinizing hormone, growth hormone, and depression following stroke.

There is reasonable evidence to implicate central perturbation in neurohormonal regulation as a causative factor in the development of appetite changes following stroke. We are not aware of clinical studies that have used appetite stimulants to improve the nutritional status of patients following stroke. Treatment with antidepressants results in reversal of anorexia and weight loss associated with depression.

Stroke affects the oral masticatory and oral transit phases of deglutition by interfering with facial, trigeminal, and hypoglossal nerve function. The deficits observed are usually due to hemispheric supranuclear lesions, with resultant upper motor neuron deficits. Buccal-labial dyspraxia may also be seen, but usually affects only oral articulation or performance of novel buccal-labial tasks to command. There is probably no true feeding apraxia, as sucking and swallowing motor patterns are present from birth and are part of primitive brain stem systems that persist even in chronic vegetative states. Pontomedullary lesions may also interfere with brain stem nuclear control of these structures. Dysphagia due to brain stem lesions is more severe, longer in duration, and more likely to show deterioration over time than dysphagia due to supratentorial lesions.

Stroke affects the pharyngeal phase of swallowing by interfering with the timing of laryngeal elevation and the strength of pharyngeal peristalsis. Delay in the swallowing reflex is the most commonly observed MBS abnormality following stroke. This interval is measured from the time the bolus passes the anterior aural pillars to the time the bolus passes through the cricopharyngeus muscle into the esophagus. As the food bolus is propelled into the oral pharynx, the base of the tongue is depressed and posteriorly rotated to allow the epiglottis to override the glottal opening. Elevation of the larynx against the descending epiglottis ensures an effective seal over the laryngeal vestibule. Elevation of the larynx also produces a mechanical pull and reflexive inhibition of the cricopharyngeus muscle, allowing the bolus to
pass into the esophagus. Spastic tightness of the crico-
opharyngeus muscle is a rare, but possible, cause of
dysphagia following stroke. It can be treated by crico-
opharyngeal myotomy, a minor procedure.24 The tem-
poral sequencing of the pharyngeal phase of swallowing is
critical for adequate airway protection and prevention of aspiration. Aspiration indicates pharyngeal-phase dysfunction with inadequate closure of the airway before, during, or after initiation of laryngeal elevation. Aspiration following laryngeal elevation, which is usually due to pharyngeal hypotonia, is demonstrated at the time of MBS evaluation by residual pooling of barium in the pyriform sinuses. This may be seen on the side opposite supratentorial lesions or on the side of nuclear brain stem lesions. Material pooled in the pyriform sinus is aspirated by venturi suction effect when air is drawn into the lungs.

The esophageal phase of swallowing is believed not to be affected by stroke.

EFFECTS OF STROKE ON STOMACH
AND DUODENUM

Central nervous system (CNS) activity has been shown on numerous occasions to impact upon gastric physiology. Gastric acid secretion, gastrin release, gastric contractions, and gastric emptying are all under the influence of varied CNS activities and CNS-derived neuropeptides.22 It would be anticipated that brain injury, as in stroke, would alter gastric function.

This has been shown to be the case in “stress” ulcers. Gastrointestinal bleeding from stress ulcers is a devastating complication of severe medical illness. The pathophysiology of this phenomenon has survived the paradigm shift in our thinking about peptic ulcer disease, which is now considered to be an infectious illness. Stress-related gastric mucosal injury appears to arise from interactions between the CNS, gastric circulation, gastric mucosa, and intraluminal contents.

Intraluminal acid, an important factor, probably facilitates the development of stress ulceration. Increased gastric acidity occurs in sepsis and head trauma, two conditions commonly associated with stress ulcer formation23,24; the mechanism behind the increased luminal acid, however, remains unclear. Impaired base secretion, too, may lead to stress ulceration. Water immersion and hemorrhagic shock, both of which are experimental models of “stress” leading to ulceration, result in a decrease in duodenal HCO₃⁻ secretion.25 Indeed, this may be why hypotension, which results in decreased acid secretion, might still predispose to stress erosions.26 H₂ blockade and nasogastric administration of antacids have been advocated for prophylaxis of stress ulceration in clinical trials.27,28

Despite these studies, many clinical entities in which luminal pH is not decreased are associated with stress ulcers.29 This has led to the theory that acidification of the gastric mucosa itself, and not just increased luminal acid, is necessary for the evolution of stress ulcers. Back diffusion of luminal acid, tissue hypoxia, and hypercapnia have been shown to contribute to a loss of cytoprotection and ulcer formation.30 In fact, parenteral NaHCO₃ prevents experimental ulceration.30 Other physiologic changes that may contribute to stress ulceration include regional decreases in gastric microcirculation (seen with stress) and impaired gastric mucosal blood flow.31,32 There is impaired gastric and duodenal secretion of HCO₃⁻ during ischemia,33 and gastric mucus secretion is decreased in animal models of stress.34 Roles for prostaglandins, epithelial renewal, and oxygen-derived free radicals remain under investigation.

Various studies have shown that there are multiple sites of brain activity, particularly in the hypothalamus and medulla, that influence gastric acid secretion. Several CNS peptides (bombesin, CRF, neuropeptides, and opioid-like peptides) enhance mucosal resistance. Others (histamine, acetylcholine, somatostatin, and TRH) increase acid secretion or mucosal vulnerability.35

The precise role of the CNS in stress ulceration remains unclear. Specific CNS diseases, however, have been clearly documented to increase the incidence of stress ulcers. In 1977, Kamada et al.36 reported the increase in gastrointestinal bleeding following head injury. In 1979, Tanaka et al.37 noted the increased frequency of significant gastrointestinal bleeding in cases of ruptured cerebral aneurysms. Cushing,38 of course, reported gastrointestinal bleeding now referred to as a “Cushing ulcer” accompanying a variety of brain lesions.

Despite these findings, cerebrovascular accidents (lumped together as one diagnostic entity) may not be a meaningful risk factor in stress ulcer formation or gastrointestinal bleeding. Of 16,612 consecutive stroke patients admitted to the Mayo Clinic between 1976 and 1994, only 17 developed a gastrointestinal hemorrhage as diagnosed by hematemesis, nasogastric return of gross blood or coffee-grounds material, or melena. (Patients with aneurysmal subarachnoid hemorrhage, chronic alcohol abuse and cirrhosis, and coagulopathies were excluded from the study.)39 Of these 17 patients, 15 had significant risk factors for gastrointestinal bleeding: 6 were long-term users of nonsteroidal antiinflammatory drugs, 3 were taking aspirin at a dose greater than 325 mg/day, 4 had grossly prolonged coagulation, and 2 had biopsy-proven Helicobacter pylori (anastral biopsy was performed in only 3 of the 17 cases of documented bleeding). One patient was on corticosteroids, and one patient had no identifiable risk factors for gastrointestinal bleeding. The rate of significant or clinically apparent gastrointestinal bleeding in this population is close to 0.1%, far less than the percentage of patients who are transferred to our institution on H₂ blockers or sucralfate for ulcer prophylaxis. Although theoretical links between head trauma, neurosurgery, and rupted cerebral aneurysms can be made, no formal association between gastrointestinal bleeding and stroke has been documented. Additionally, recent studies indicate that prophylaxis against stress ulceration is needed only in patients who are mechanically ventilated, have an underlying coagulopathy, or have an illness previously shown to have an association with stress ulcer formation: burns over more than 30% of the body surface area, organ transplantation, endoscopic or radiographic
diagnosis of peptic ulcer or gastritis in the 6 weeks prior to ICU admission, or gastrointestinal bleeding 3 to 6 weeks prior to admission.40

Nevertheless, many stroke patients are put at risk for gastrointestinal bleeding as part of their therapy, when given warfarin (often resulting in over-anticoagulation) or aspirin. In this manner, gastrointestinal bleeding does accompany stroke.

As for other disorders of gastric function, it is clear that the CNS plays an important role in regulating (or at least influencing) gastric emptying via vagal efferents and via vagovagal reflexes, organized in the medulla (nucleus of the tractus solitarius) and modified by the hypothalamus.22 Currently, there are no data linking stroke to alterations in gastric motility.

STROKE EFFECTS ON THE COLON

Constipation, defined as a decrease in frequency of bowel movements, is often a complaint of elderly patients and is commonly associated with stroke. Hippocrates noted that “it is a general rule that the intestines become sluggish with age,” though the mechanism for this association even today remains unclear. Even as our understanding about the physiology of colonic transit and the interplay between the central and enteric nervous systems grows, the specific pathophysiological basis for constipation and fecal impaction remains elusive. Although medical reviews frequently claim an association between constipation and stroke, supporting data are limited at best.

The pathogenesis of constipation is clearly multifactorial. Normal colonic motility results from the interaction of intraluminal contents with colonic smooth muscle. Segmentation waves, peristaltic waves, and mass movements, under the influence of an array of neurotransmitters, peptideergic neurocrines, and a host of unidentified endocriines, paracriines, and autocriines, act in an organized fashion to propel the contents aborally for defecation. Systemic and local factors can interfere with this system and thus impair motility and cause constipation (or, conversely, diarrhea). A number of these constipating factors have been identified or proposed.

Medications frequently interfere with normal bowel activity and are cited as a frequent cause of constipation. Opiate analgesics, aluminum- and calcium-containing antacids, anticholinergics, anticonvulsants, antidepressants, drugs for the treatment of Parkinson’s disease, ganglionic blockers, diuretics, iron, anti hypertensives, antipsychotics, and irritant laxatives (in chronic use) can all cause constipation.41 Looking at this list, it is not hard to imagine that patients with newly diagnosed strokes would be exposed to many of these agents during their acute hospital stays (and thereafter). It would not be surprising, then, that constipation would ensue as a secondary effect of appropriately prescribed medications.

Additionally, immobility and bed rest often give rise to constipation. Indeed, work done by Kinnunen42 suggests that walking less than 0.5 km/day increases the risk of constipation in the elderly. Deconditioning, which accompanies bed rest and immobility, may result in impaired or inadequate force to defecate.

Dehydration (or, more appropriately, hypovolemia) is another common problem in the stroke population, whether secondary to restrictions on thin liquid ingestion because of dysphagia, lack of access to water because of motor abnormalities, or an impaired thirst mechanism. Dry mucous membranes, dry axillae, and a rising BUN-to-creatinine ratio are frequently seen on the stroke ward. These abnormalities give rise to increased ADH and aldosterone synthesis and release, with resultant increased hardness of stool.

Diet also plays an important role in the pathogenesis of constipation. Increased dietary fiber has been shown to increase stool volume, decrease colonic transit time, and increase the frequency of defecation.43 Its precise role in the etiology and treatment of constipation remains murky, with several studies failing to demonstrate an association between fiber intake and either the prevalence or course of constipation.44 Nevertheless, most stroke patients at risk for constipation are placed on a high fiber diet providing at least 35 g of fiber per day.

To study the association between stroke and constipation, Johanson et al45 reviewed records of all Medicare hospitalizations during 1987 that resulted in a diagnosis with the ICD code for constipation (564.0). The frequency distribution of all three-digit ICD codes and the code for constipation was tested with a significance level of P = 0.0001. Results indicated that many neurologic and psychiatric disorders were associated with constipation (odds ratios ranging from 1.4 to 3.9): multiple sclerosis, mental retardation, Parkinson’s disease, myelopathies, and paralytic syndromes. Hemiplegia and intracerebral hemorrhage were not. One explanation for the apparent lack of association between hemiplegia and stroke and constipation may be that only five ICD codes per admission were allowed. A follow-up study by Sonnenberg et al46 reviewed ICD codes for VA patients between 1985 and 1989 (with up to 10 codes allowed). This study was performed to test whether the entity “institutional colon” truly existed. As controls, four diagnoses associated with immobility were chosen. Cerebrovascular disease (ICD 430–438) was one of these diseases. No association between cerebrovascular disease and constipation was found, though the ICD code for “hemiplegia, unspecified” (342.9) was associated with constipation, fecal impaction, and volvulus.

For a long time, without supporting data, physicians have taken it on faith that stroke is associated with constipation. Yet the above two comprehensive studies failed to demonstrate such a concordance. One possible explanation is that physicians and nurses use appropriate prophylactic measures to prevent constipation in stroke patients, and thus constipation is no longer seen frequently enough in the stroke population to be significant in the epidemiologic studies cited. Another possible explanation lies in the coding of constipation, which may not make the top five or ten ICD codes for stroke patients with many other preexisting illnesses and comorbidities.
Fecal impactions are also frequently associated with stroke. The mechanism usually cited is immobility with increased colon transit time and increased time for desiccation in the rectosigmoid colon. There may be many other predisposing factors: inadequate dietary fiber, dehydration, use of anticholinergic or opioid medications, depression, or autonomic dysfunction. Stroke may also affect the force of contraction of the striated muscles of the diaphragm and abdominal wall, resulting in a decrease in abdominal pressure assistance with defecation. Lesions affecting the pontine defecatory center may disrupt the sequencing of sympathetic and parasympathetic components of defecation and impede the coordination of the peristaltic wave and relaxation of the pelvic floor and external anal sphincter. Stroke may impair rectal sensation with resultant rectal volumes exceeding 500 cc (normally not greater than 200 cc), a volume noted to predispose to impaction in those with sensory deficits of the involved region. In such situations, rectal contents become too massive to expel. Such problems are commonly seen with spinal cord and cauda equina lesions. Stroke, thus, has been alleged to give rise to fecal impaction: but, as with constipation, there are no definitive studies in the literature to document the connection.

Lesions affecting prefrontal association areas inhibiting reflex defecation are expected to cause fecal incontinence rather than impaction. Cortical stroke syndromes resulting in dementia, object agnosia, or visuospatial disorientation interfere with adherence to social conventions concerning the timing and location of defecation and may lead to fecal incontinence.

**STROKE AND NUTRITION**

Patients with stroke typically are elderly and suffer the comorbid problems of the elderly. One of these problems is malnutrition. Single elderly adults will often not bother to prepare three balanced meals each day. Devoid of the stimulation of preparing a communal meal, they often find it more expedient to settle for a meal of tea and cookies. Stroke victims are, therefore, expected to be at least as malnourished as age-matched controls in the community.

The most dramatic effects of stroke on nutrition relate to dysphagia, as described. Stroke may also affect sympathetic and parasympathetic medullary and hypothalamic centers regulating gut motility, secretory status, and appetite. Such lesions, however, usually involve adjacent vital cardiopulmonary centers and permit few to survive.

The most commonly seen strokes are unilateral hemispheric lesions. These are associated with hypometabolism, as documented by positron emission tomography scanning techniques, at the site of lesion as well as at sites functionally related but distant from the lesion (diaschisis). Diaschisis is most prominent during the acute phase of stroke and diminishes with time. Appetite, swallowing, and autonomic homeostasis are primitive functions that are bilaterally represented, but potentially affected by diaschisis.

As described earlier, with anticipatory-phase dysphagia there is a linkage between appetite loss and poststroke depression. There are also documented poststroke changes in cortisol, prolactin, and growth hormone homeostasis associated with clinical depression following stroke. The relationship of these alterations and gut-related neuropeptides regulating feeding behavior (cholecystokinin, bombesin, somatostatin, calcitonin) has not been studied. Cholecystokinin is released by the proximal small intestine in response to feeding. It has the effect of turning off feeding behavior, decreasing stomach motility and gastric acid secretion, and increasing pyloric sphincter tone. Cholecystokinin released from the small intestine does not penetrate the blood-brain barrier. Its effect on the CNS is probably mediated by vagal afferents from receptors in the pyloric sphincter, pancreas, and vagus nerve terminals. Cholecystokinin is, however, found presynaptically in the hypothalamus. It is released by neuronal microelectrode stimulation, causes depolarization of parameirene hypothalamic neurons, and produces cessation of feeding when given in physiologic concentrations intraven-tricularly. Bombesin administered parenterally in the rat also causes cessation of feeding. It has a local effect of causing gastric smooth muscle contraction, which mimics gastric distention associated with feeding. It also has a central hypothalamic effect on appetite suppression that is not mediated by vagal afferents. Somatostatin, calcitonin, and other gut-related neuropeptides produce a recurrence of migrating motor complexes associated with feeding behavior in the small intestine of fed animals. It is not clear to what extent their stimulating effects on feeding behavior are due to direct versus vagally mediated hypothalamic effects.

Stroke is a major medical illness and, like pneumonia or myocardial infarction, is associated with weight loss. The mean weight loss following middle cerebral artery infarction has been estimated at 30 lb. The mean change in weight for patients with versus without dysphagia has been estimated as $-0.23 \pm 0.09$ SEM kg/week versus $+0.03 \pm 0.06$ kg/week during the rehabilitation phase following stroke. Since there is no known increase in basal metabolic rate following stroke, it is assumed that weight loss is due to a combination of dysphagia (discussed earlier), appetite loss, and decreased muscle mass due to immobility. Appropriately prescribed dysphagia diets, compensatory swallowing techniques, diet supplements, and graded exercise are reasonable treatment strategies. Appetite stimulants and anabolic steroids are not, as yet, of proven value.

There is a direct linkage between the severity of hypoalbuminemia and duration of acute hospitalization. This association is very robust and has been duplicated in many studies. A similar phenomenon has been observed following stroke. There is also a linkage between hypoalbuminemia and decreased self-care and mobility function following head injury. There are no studies that control for the severity of dysphagia; therefore, it is difficult to determine to what extent hypoalbuminemia is due to decreased protein intake rather than protein catabolism as a result of chronic illness. In no
studies was hypoalbuminemia treated in an effort to decrease mortality or improve functional recovery following stroke.

During the acute stroke phase there is reasonable attention to defining the patient's nutritionally related stroke risk factors: diabetes, hypertension, hypercholesterolemia. The dietary prescription is often an 1,800 calorie American Diabetes Association diet with 2 mg of sodium and 300 mg of cholesterol. If there is associated dysphagia or concern for constipation, additional dietary constraints may be applied: pureed, high fiber supplementation. The resultant diet is quite bizarre and unappetizing. Perhaps it is more prudent to forego most dietary restrictions, realizing that it is more beneficial for patients to savor their meal than to adhere to an "optimal diet." Use of a diuretic can allow the patient to consume a more palatable liberalized sodium diet. It has been estimated that a 7% reduction in serum cholesterol at age 60 probably adds only 2 months to the patient's life expectancy.54

There is a need for dietary management of stroke risk factors, but the window of opportunity is often closed by the time patients present with their stroke. Nutritional consultation might be better focused on the patient's children or younger siblings or deferred until the patient has reestablished metabolic homeostasis.

REFERENCES

42. Kinnunen O. Study of constipation in a geriatric hospital, day hospital, old peoples home and at home. Aging 1991;3:162-70


