Lower Esophageal Sphincter Dysfunction Often Precludes Safe Gastric Feeding in Stroke Patients

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Objectives: To determine the relationship between stroke and lower esophageal dysfunction with vomiting and to identify an optimal nutrition protocol based on our findings.

Patients and Methods: The lower and upper esophageal sphincter functions were assessed in 35 patients who had an acute stroke to determine whether gastric or jejunal enteral feeding was the optimal route. Stroke was due to unilateral ischemia in 20 patients, unilateral intracerebral hemorrhage in 8 patients, and global ischemia in 7 patients. Our study consisted of 18 men and 17 women with an average age of 64 years.

Results: Using standard esophageal manometric definitions, the lower esophageal sphincter function was below normal in 24 patients: 3 had global anoxia, 5 had unilateral hemorrhage, and 16 had unilateral ischemia. The upper esophageal sphincter function was low in 30 patients: 6 had global anoxia, 7 had unilateral hemorrhage, and 17 had unilateral ischemia. Based on lower esophageal sphincter pressure, 7 patients underwent tube gastrostomy and 13 patients underwent tube jejunostomy placement. All tolerated enteral alimentation well. Prior to lower esophageal sphincter assessment, 4 patients had percutaneous endoscopy gastrostomy feedings that led to aspiration pneumonitis and consultation for tracheostomy; 2 terminally ill patients were referred to the ethics service, and 2 were converted to feeding via jejunostomy tube at the time of tracheostomy and did well.

Conclusions: Vomiting with aspiration due to lower esophageal sphincter dysfunction is common after acute strokes. Esophageal manometry serves as a guide to find the optimal feeding route.

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N ACUTE cerebral vascular stroke can lead to multiple problems, both short term and in convalescence. The first challenge is to overcome the life-threatening insults caused by the brain injury and the loss of many central regulatory systems. After surviving the acute phase, the patient enters the recuperative period when many other threats related to independent airway control, nutrition, and mobility must be faced. Success during this phase is strongly influenced by the restoration of an alimental diet.

Following stroke, oral deglutition is often impaired because of central hypopharyngeal dysfunction.1,2 Attempts at oral feeding are frustrated by aspiration; aspiration pneumonia is a common cause of death following stroke.3 This hazard may be circumvented by small intragastric feedings through narrow, specifically designed, gastric feeding tubes. The surgeon can be consulted later for placement of a more permanent intragastric feeding tube such as a percutaneous endoscopic gastrostomy.4,5 Vomiting with aspiration pneumonitis, however, is a frequent complication of intragastric feedings whether given by nasogastric tube or percutaneous endoscopic gastrostomy feedings,7,8 and is a common cause of death for patients in long-term care facilities.9

Prior studies have demonstrated that acute head injury causes a temporary reduction in lower esophageal sphincter (LES) function.10 Such patients are at high risk for aspiration pneumonitis. Our study was designed to assess esophageal function in patients who had a stroke and were referred to us for feeding tube placement and to identify an optimal nutrition protocol based on our findings. These patients can then be restored to a full alimental diet.

RESULTS

The esophageal manometry was well tolerated by all patients; there were no com-
PATIENTS AND METHODS

Esophageal function was assessed by esophageal manometry in 35 patients who had an acute stroke. All 35 patients were seen in consultation for performance of a tracheostomy for respiratory failure and pneumonia, for placement of a feeding tube for alimental nutrition, or for both. There were 18 men and 17 women with an average age of 64 years (range, 39-100 years). The cause of the acute stroke was unilateral ischemia in 20 patients, unilateral intracerebral hemorrhage in 8 patients, and global ischemia in 7 patients. All 35 patients had severe strokes, with complete unilateral deficits after focal ischemic insults and coma after global ischemia. Although modest neurologic improvement was present at the time of consultation in some patients, all had severe residual deficits. Esophageal manometry was performed during the second week after stroke in 14 patients, during the third week after stroke in 12 patients, and between the third and sixth week after stroke in 9 patients. All patients were receiving histamine2 blockers as prophylaxis for short-term, erosive gastritis. Twenty-six patients received antibiotics (often for aspiration pneumonia), 12 received antihypertensive medicines, and 7 received inotropic support. A detailed history of gastroesophageal reflux disease was difficult to obtain because of the communication impairment but 1 patient was treated for prior symptoms suggestive of reflux esophagitis.

The technique for esophageal manometry has been previously described.10 Esophageal manometry with direct testing of LES and upper esophageal sphincter (UES) tone was performed with a 3-channel probe connected through appropriate transducers to a computer-based (Sandhill, Sandhill Scientific, Highlands Ranch, Colo) manometric system.11,12 The esophageal manometric probe is a standard 3-transducer probe with the pressure sensors set at 5-cm intervals near the distal end of the probe. The probe was placed through the nose to a depth of 70 cm and a gastric baseline value was obtained. This measurement was considered the 0 point. The probe was then withdrawn at 5-cm intervals and both continuous and interval manometric measurements were obtained. All ancillary activity during the manometric testing was noted (ie, swallowing, coughing, and suctioning). These events caused a transient artifact in the ongoing measurements and were discounted. The continuous data measurements captured events that did not occur at a set interval,11,12 and the interval measurements allowed for study of an area to note any changes in tone over time.10 All 35 patients were given histamine2 blockers as prophylaxis against short-term, erosive gastritis, precluding pH monitoring of reflux from the stomach into the esophagus. All values are expressed as mean±SE.

The LES pressure and UES pressure were measured with a 3-channel probe connected through appropriate transducers to a computer-based (Sandhill, Sandhill Scientific, Highlands Ranch, Colo) manometric system.11,12 The esophageal manometric probe is a standard 3-transducer probe with the pressure sensors set at 5-cm intervals near the distal end of the probe. The probe was placed through the nose to a depth of 70 cm and a gastric baseline value was obtained. This measurement was considered the 0 point. The probe was then withdrawn at 5-cm intervals and both continuous and interval manometric measurements were obtained. All ancillary activity during the manometric testing was noted (ie, swallowing, coughing, and suctioning). These events caused a transient artifact in the ongoing measurements and were discounted. The continuous data measurements captured events that did not occur at a set interval,11,12 and the interval measurements allowed for study of an area to note any changes in tone over time.10 All 35 patients were given histamine2 blockers as prophylaxis against short-term, erosive gastritis, precluding pH monitoring of reflux from the stomach into the esophagus. All values are expressed as mean±SE.

Complications. The LES pressure averaged 13.2±3.01 mm Hg and the UES pressure averaged 23.9±3.75 mm Hg. Both the LES pressure (14 mm Hg) in 24 patients and the UES pressure (40 mm Hg) in 30 patients were below normal (Table 1). The 20 patients who had ischemic unilateral strokes had the lowest LES pressure, averaging 12.5±4.5 mm Hg, 4 of whom had an LES pressure above 14 mm Hg. The LES pressure in the 8 patients who had unilateral intracerebral hemorrhagic strokes averaged 17.8±4.6 mm Hg, 5 of whom had an LES pressure below 14 mm Hg (Table 1). The 7 patients who had global ischemia had an average LES pressure of 19.9±6.0 mm Hg, 3 of whom had a low LES pressure (Table 1). The LES and UES pressures were significantly (P<.05, unpaired Student t test) below those seen in normal volunteers (LES, 29±2.6; UES, 47±5.6) and in patients with head and neck cancer referred for feeding tube placement (LES, 27.5±5.6).

The UES pressure in the 20 patients who had ischemic strokes averaged 23.6±4.4 mm Hg, 17 of whom had a UES pressure below 40 mm Hg (Table 2). The LES pressure in the 8 patients who had unilateral intracerebral hemorrhagic strokes averaged 23.9±11.82; 7 of 8 patients had a UES pressure below 40 mm Hg (Table 2). The LES pressure in the 7 patients who had global ischemia averaged 25±6.04 mm Hg, 6 of whom had a low UES pressure (Table 2). These pressures were significantly lower (P<.05) than the UES pressure in normal volunteers.

The decision on the technique for alimental feedings was based on the LES manometric studies. Thirteen patients who had progressive illness died or were referred to the ethics service and never received an operative feeding tube, including 9 patients with low LES pressure and 4 patients with normal LES pressure. Two patients’ conditions improved and did not require a feeding tube. The remaining 12 patients with low LES pressure underwent an open feeding jejunostomy; all these patients tolerated their jejunal feedings without aspiration of the liquid diet. Many patients, however, exhibited a syndrome of increased gastric and biliary secretions that were sectioned from the nasogastric tube during the first 1 or 2 weeks of jejunal feedings. This common phenomenon of cryptic origin has been previously described; prevention of aspiration of the gastric and biliary commends requires continued nasogastric suction while the tube feedings are advanced.13 The addition of blue dye to the tube feedings confirmed that this nasogastric effluent had no blue discoloration and was not tube feeding.13 The remaining 8 patients with normal LES function were treated by feeding gastrostomy in 7 patients and by a feeding jejunostomy in 1 patient; this latter patient had prior vomiting of nasogastric tube feedings, biasing the surgeon to perform a feeding jejunostomy. The patients with gastrostomy, performed by either the open or percutaneous technique, tolerated the tube feedings without vomiting and aspiration. All 20 patients tolerated tube feeding and were transferred to a rehabilitation hospital or long-term care facility with tube feeding as the sole source of nutrition. Tolerance of the tube feeding persisted until discharge from the rehabilitation institution. Most patients’ conditions improved during the rehabilitation care. Four patients regained normal swallowing and had the feeding tube removed; none had a second esophageal manometry at this time. The other patients
were lost to follow up after discharge from the rehabilitation hospital.

Four patients were initially consulted for tracheostomy for pneumonitis that developed from aspiration of percutaneous endoscopic gastrostomy feeding. The percutaneous endoscopic gastrostomy, in each instance, had been placed without manometric studies. All 4 patients had low LES tone. The tracheostomy was done in conjunction with percutaneous endoscopic gastrostomy tube removal and placement of an open feeding jejunostomy in 2 patients who rapidly recovered from pneumonitis when they were no longer aspirating intragastric feedings. Both patients tolerated jejunal feedings without vomiting. The other 2 patients had progression of the brain deficit after the onset of aspiration pneumonitis and were transferred to the ethics service.

**Table 1. Lower Esophageal Sphincter (LES) Studies**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>LES, mm Hg</th>
<th>LES ≥ 14 mm Hg</th>
<th>LES &lt; 14 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Patients, No.</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Patients, total No.</td>
<td>15.17 ± 3.01</td>
<td>35</td>
<td>32.29 ± 7.26</td>
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<tr>
<td>Patients with</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global anoxia</td>
<td>19.89 ± 5.96</td>
<td>7</td>
<td>28.01 ± 8.41</td>
</tr>
<tr>
<td>Focal hemorrhage</td>
<td>17.75 ± 4.82</td>
<td>8</td>
<td>32.09 ± 4.48</td>
</tr>
<tr>
<td>Focal ischemia</td>
<td>12.45 ± 4.52</td>
<td>20</td>
<td>36.73 ± 19.74</td>
</tr>
</tbody>
</table>

**Table 2. Upper Esophageal Sphincter (UES) Studies**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>UES, mm Hg</th>
<th>UES ≥ 40 mm Hg</th>
<th>UES &lt; 40 mm Hg</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SE</td>
<td>Patients, No.</td>
<td>Mean ± SE</td>
</tr>
<tr>
<td>Patients, total No.</td>
<td>23.90 ± 3.75</td>
<td>35</td>
<td>65.23 ± 10.44</td>
</tr>
<tr>
<td>Patients with</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global anoxia</td>
<td>24.99 ± 6.04</td>
<td>7</td>
<td>44.40 ± 0.00</td>
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<tr>
<td>Focal hemorrhage</td>
<td>23.93 ± 11.82</td>
<td>8</td>
<td>103.10 ± 0.00</td>
</tr>
<tr>
<td>Focal ischemia</td>
<td>23.58 ± 4.40</td>
<td>20</td>
<td>59.56 ± 6.01</td>
</tr>
</tbody>
</table>

Many patients who have acute strokes die within 96 hours from the extensive brain injury or associated diseases complicated by the acute stroke. Other patients recover quickly and are able to tolerate a carefully administered and slowly advanced oral diet. The remaining patients require multidisciplinary support as they enter the recuperative phase. A key element in the successful treatment of these patients is the successful institution of alimental nutrition.

During the early poststroke period, when central nervous system dysfunction, adynamic ileus, abdominal distention, and pulmonary distress are paramount, nutrition typically is provided by the intravenous route. Once this acute phase has passed and the adynamic ileus has resolved, the patient should be assessed for hypopharyngeal function and swallowing proficiency. Some patients exhibit adequate swallowing and oral feeding may be carefully started. Presumably, these patients have normal esophageal manometries, although no measurements of LES and UES pressures have been reported in patients who had a stroke and with normal swallowing. Acute stroke often impairs swallowing due to the injurious effects on the cranial nerves that modulate deglutition. When doubt exists about tolerance of oral feedings, a swallowing test can determine if there is evidence of aspiration associated with swallowing. There may also be patients who do not seem to aspirate during a swallowing test but then develop the clinical findings of aspiration pneumonitis. When oral feedings are not tolerated because of hypopharyngeal dysfunction, intragastric feedings should be instituted. Patients who tolerate the intragastric feedings will probably tolerate gastric tube feedings. The benefits of the intragastric route relate both to the maintenance of immunological competence of alimental feedings and the ability to give bolus feedings. Nasogastric tube feedings with monitoring residual volume predicts successful tolerance of gastric tube feedings; however, cryptic vomiting with aspiration may recur and be evidenced by the development of recurrent or worsening pneumonitis. This sequence presages LES dysfunction and can be quickly and safely identified by esophageal manometry. If the LES function is low, the patient requires intrajejunal feedings that can be instituted by an endoscopically placed tube or by open laparotomy. The manometric assessment of LES function, identified herein, seems to accurately define whether a patient should receive a tube gastrostomy or a tube jejunostomy.

These studies identify an interesting but small subgroup of patients who have a normal LES function, but a low UES function. The current level of experience with this subgroup of patients is too small to predict what type of feeding is most desirable. Clearly, more study is needed to identify whether these patients are candidates for intragastric or intrajejunal feeding.
REFERENCES


