### **DIRECTOR'S FORUM**

# **APHAGIA: LOSS OF THE ABILITY TO SWALLOW**

H. Worth Boyce, M.D. Professor of Medicine and Radiology and Director

The medical dictionary defines aphagia as the refusal or loss of ability to swallow. There are several causes for this tragic medical condition. This presentation will discuss our experience with an increasingly common cause of this condition that often follows curative radiation and chemotherapy, and in some cases, combined with surgery, for cancer in the head and neck region.

Many remarkable advances in medical diagnosis and therapy have occurred over the past two decades. Most have been of benefit to a wide array of illnesses. These innovations in pharmacology, biochemistry, serology, imaging methods, surgical technique, endoscopic diagnosis and therapy have in rare instances, resulted either directly or indirectly, in harmful side effects. The purpose of this presentation is to review experience over the past twenty years with a condition occurring as the direct result of modern, effective, curative radiation/chemotherapy, with or without surgery, and an indirect effect of the success of percutaneous endoscopic gastrostomy (PEG), i.e., a feeding tube placed through the abdomen directly into the stomach, used as a route to restore and maintain adequate caloric intake in patients unable to swallow.

The increasing frequency of total obstruction of the swallowing passage and the magnitude of its deleterious result upon patients who have already endured the anxieties and side effects of therapy for cancer of the tongue, larynx and pharynx, including the upper esophagus, deserves emphasis. Recognition of this potentially reversible, but sometimes disastrous, occurrence by all physicians including otolaryngologists, surgeons, medical and radiation oncologists, gastroenterologists and patients, may help either in its prevention or early recognition and therapy.

The problem at hand is benign total pharyngoesophageal lumen occlusion following successful radiation/chemotherapy, and surgery in some cases, for head and neck cancer, i.e., squamous cell carcinoma of the tongue, pharynx, larynx, cervical esophagus and less often, other sites. The typical scenario is that of a patient who received a PEG tube prior to, or soon after, radiation and chemotherapy.

During therapy, as expected, the usual mucositis (inflammation of the surface tissue of mouth, pharynx and esophagus in the head/neck region), develops and results in odynophagia (painful swallowing) and dysphagia (difficult swallowing). This inflammatory response is not considered a complication, but is a regular potential occurrence with successful cancer therapy. The patient typically develops throat pain,

odynophagia, dysphagia, and receives appropriate standard symptomatic therapy with the exception of encouragement to try to swallow as much food as possible. This effort will help to maintain an open swallow passage and muscle function.

Another important consideration is early dilation (stretching) to keep the surfaces of the throat passage (lumen) from becoming adherent with adhesions that result in closing the lumen. When the dysphagia presents, or progresses to aphagia, during or soon after completion of the course of radiation therapy, the patient should be completely evaluated. Early swallowing therapy and possible dilation should be considered. When evidence of lumen narrowing is detected by barium x-ray contrast study, after termination of radiation/chemotherapy, a plan for dilation therapy should be instituted. Instead of this approach, some patients are reassured by their therapists who state, "Don't worry; your swallowing will improve in a few more weeks". It is difficult soon after cancer therapy to predict which patients will develop severe obstruction that requires dilation with or without neuromotor (nerve-muscle) dysfunction.

In the years before PEG nutrition was available, the combination of dysphagia and weight loss was much more ominous for the patient's overall health and nutrition and served to create sufficient concern to provoke prompt evaluation and therapy. The presence of dysphagia after therapy often led to early dilation and preservation of lumen patency sufficient for adequate oral intake unless there was also a concomitant loss of pharyngeal neuromotor function as a consequence of radiation and chemotherapy. The increasing frequency of this sequela, i.e., total or near total lumen occlusion with aphagia, justifies the need for concern.

Dysphagia or aphagia occurring under these circumstances must be treated early and adequately after radiation/chemotherapy regardless of the patient's healthy appearance that has been assured by the alternative feeding route via PEG with enteral nutrition. In our series, the average delay in referral of patients for evaluation of endoscopic lumen restoration (ELR) is 8.2 months (range 2 weeks to 27 months) after onset of aphagia.

The miseries of aphagia include the need to spit all saliva into a cup or tissue, the risk of bronchitis or pneumonia from throat contents being aspirated into the lungs, poor oral and dental hygiene, and the loss of the pleasures of eating with the associated social incapacities.

The outcomes of treatment of aphagia after radiation and

## **APHAGIA: LOSS OF THE ABILITY TO SWALLOW: (continued)**

chemotherapy vary from superbly gratifying successes to sad failures. Lumen restoration procedures can, and should be attempted, especially in those patients with no evidence of residual or recurrent cancer. The lumen obstruction may be caused by a very thin web-like membrane, or a dense fibrotic scar up to 2.5 cm (1 inch) or more in length. In rare instances, an extremely narrow (1 to 2 mm) residual opening that prevents oral food intake or even passage of saliva, is found at the initial evaluation. This finding makes lumen restoration easier since dilation can be started promptly.

Lumen restoration between pharynx and esophagus usually can be accomplished by combined antegrade - retrograde endoscopic procedures with fluoroscopy to assist with axis orientation from two directions. This stage of the ELR procedure is performed by the esophagologist in conjunction with an otolaryngologist in the surgical suite under general anesthesia. Accessories used to establish an opening between pharynx and esophagus have included biopsy forceps, rigid suction cannulus, blunt tipped probes, large gauge needles on a long stainless steel tube, and the rigid (reverse) end of a flexible guide wire. The otolaryngologist member of the team approaches the occlusion via the mouth, while the esophagologist-gastroenterologist approaches the site of blockage from below, using a very thin endoscope through the site of the PEG tube (which is temporarily removed for this purpose). The two operators align their instruments using direct vision through their endoscopes under simultaneous viewing by fluoroscopy.

Once the strictured segment is penetrated, a flexible guide wire is passed and a strong silk suture is pulled from mouth through the esophagus, stomach and PEG stoma. Retrograde dilation is then performed using a series of small diameter Tucker (spindle-shaped) dilators, attached to each other, by loops of silk suture on the end of each dilator, like links of sausages. After completing the initial dilation, the proximal end of the suture is retrieved from the pharynx using a transfer catheter through the nostril and then looped over the ear and tied firmly to the distal end of the suture and taped loosely to the upper chest. Future dilations are done in a similar manner from stomach up through the stricture until a guide wire can be easily passed from the mouth through the stricture for a normal type or antegrade dilation. After several sessions of

gradually larger antegrade dilations, a guide wire can be passed with ease. The indwelling suture can then be permanently removed with assurance that future dilations can be performed in the usual manner and lumen patency can be maintained.

A program of serial dilations, usually over 3-6 months, is continued at 2 to 4 week intervals until lumen dilation is increased to 15 to 17 mm. At this level of dilation, a program of neuromotor swallowing rehabilitation is initiated under direction of a speech pathologist with expertise in swallowing disorders.

In our experience with 13 patients, successful lumen restoration after total lumen occlusion and aphagia has been possible in 9 patients able to resume a modified regular or soft diet, 1 patient is taking small volumes of liquids only, but still undergoing initial dilation therapy. One patient, who has completed initial dilation therapy was only able to swallow saliva and small volumes of liquids. This failure was primarily due to residual inadequate oropharyngeal neuromuscular function. Two patients had successful lumen restoration, but were subsequently treated in other institutions. In 2 patients, ELR failed on the first attempt and succeeded on the second.

Adequate lumen restoration is necessary before residual pharyngeal neuromotor function can be assessed. When patients have been aphagic for weeks, months or years, the determination of adequate neuromotor function is not possible with certainty until adequate lumen patency has been restored. Swallowing rehabilitation over a matter of weeks should be conducted under supervision by a Speech Pathologist who has experience with these techniques.

If total loss of pharyngeal neuromotor function is confirmed and the patient aspirates during swallowing attempts, after a totally occluded lumen has been restored to 17 mm or 51 French diameter over several weeks, the risk of any oral intake is high and the prognosis is poor.

Patients with aphagia or total pharyngoesophageal obstruction following radiation-chemotherapy for head and neck cancer deserve an attempt at ELR. This technique offers the best opportunity for restoring the ability for, and pleasures of, nutrition by mouth with a low risk if an adequate lumen is restored and a functional level of oropharyngeal neuromotor function returns with a swallow rehabilitation program.

#### **AVOID HOLIDAY HEARTBURN**

During the holidays, rich foods, large meals and alcohol can lead to attacks of heartburn.

Heartburn is a term that is sometimes misleading. It has nothing to do with your heart. Heartburn results from a backup of acid-containing stomach contents into your esophagus (feeding tube).

Ten percent of healthy people have at least one episode of heartburn each week. Here's what you can do to reduce heartburn:

Eat smaller meals – Too much food expands your stomach and puts pressure on a band of muscle (esophageal sphincter) that helps keep food in your stomach.

Avoid alcohol, fatty foods, chocolate, spearmint and peppermint – these foods can relax your esophageal sphincter and promote upward flow of stomach contents.

**Use an antacid** – People most often take over-the-counter antacids to relieve symptoms of heartburn. But, an antacid also can help prevent symptoms. Take it after meals and before bedtime.

Don't eat before sleeping – Wait two to three hours after eating before lying down. This allows enough time for increased stomach acid produced by your meal to taper off.

Wear loose clothes – A tight belt, girdle, or waistband can cause hearthurn.

**Stop smoking** – The nicotine from cigarettes can relax your esophageal sphincter.

**Elevate the head of your bed** – Raise the head of your bed 4 to 6 inches. This helps keep stomach acid in your stomach where it belongs.

When the holidays are over, if you are overweight, make a commitment to lose weight. Trimming down helps reduce the pressure your abdomen puts on your stomach when you are lying down.

If you are taking acid suppressing medication such as PPI drugs (AcipHex, Nexium, Prevacid, Protonix, Zegerid) or H2 blockers (Pepcid, Zantac), be sure to take them on schedule as directed. **PPI** drugs should be taken 30 or more minutes before a meal.

## NON-CARDIAC CHEST PAIN: DIAGNOSTIC DILEMMAS AND THERAPY

David S. Estores, Jr., M.D.

Chest pain is a common symptom leading to an emergency room visit. The patient who has chest pain experiences a lot of anxiety and there is a substantial negative impact on quality of life. This would be true even after the patient is told that heart disease is not the cause of chest pain. Recurrent chest pain in a person who has had the appropriate studies to exclude heart disease is given a label of non-cardiac chest pain (NCCP).

Approximately 75 million Americans are expected to have experienced NCCP at any point in time. Most of these patients go to their primary care provider, who in turn, refer them for a complete cardiac evaluation. The prevalence of NCCP among men and women is the same. Among the estimated 600,000 who undergo a cardiac catheterization every year, approximately 30% or 180,000 have no significant coronary artery disease and thus are said to have NCCP. Among these patients, approximately half or 90,000 may have chest pain attributable to the esophagus.

The most commonly accepted explanation for NCCP from the esophagus centers around an increase in sensitivity of the esophageal wall to acid. Esophageal motility disorders or "muscle spasms" are another category of esophageal disorders which can cause NCCP. There are a variety of non-esophageal causes of NCCP, among which are: psychological (panic disorder, anxiety or major depression); musculo-skeletal (inflammation in the chest wall); microvascular heart disease (syndrome X, not seen on routine coronary artery imaging); and lung disorders (pneumonia, pulmonary embolism).

There is no highly reliable or accurate way to differentiate between chest pain from your heart or from your esophagus based on characteristics of the chest pain or other patient-related variables (age, sex, risk factors for heart disease, family history). Thus, there is a great need to have an appropriate diagnostic work-up

done to ensure that the pain is indeed not from the heart. The reason why it is difficult to differentiate whether the pain signal is coming from the heart, or esophagus, is because these structures share some common nerve pathways and have a similar type of muscle that can respond to nitrate medications in the same manner.

The most prudent approach to exclude gastroesophageal reflux would be to administer a trial of higher than usual dose of a PPI (proton pump inhibitor) for at least two weeks while monitoring symptoms of NCCP. The proton pump inhibitor class of drugs includes AcipHex, Nexium, omeprazole generic, Prevacid, Prilosec OTC, Protonix, and Zegerid. If the chest pain persists while the patient is under acid suppression with a proton pump inhibitor, it is reasonable to pursue additional testing in the form of esophageal pH monitoring (to test for acid reflux) and an esophageal manometry to evaluate for esophageal spasm or other so-called motility disorders.

Medical therapy in the form of antidepressants has also been useful for patients with NCCP. There have been favorable reports of non-traditional medical treatments such as hypnosis and behavioral therapy for patients with NCCP. Treatment of NCCP may be frustrating for both the patient and the physician since this entity is not completely understood.

NCCP is a condition with which a sound physician/patient relationship can play a vital role. The issue becomes even more complicated in patients with coronary artery disease who may have chest pain provoked by acid exposure in the lower esophagus or due to other esophageal disorders. Treatment should be tailored to each individual's unique needs based on a thorough medical evaluation.

## **EFFECT OF DECAFFEINATION OF COFFEE OR TEA ON GASTROESOPHAGEAL REFLUX**

Coffee and tea are believed to cause gastroesophageal reflux; however, the effects of these beverages and their major component, caffeine, have not been quantified. The aim of a study several years ago was to evaluate gastroesophageal reflux induced by coffee and tea before and after a decaffeination process, and to compare it with water, and water containing caffeine.

Three hour ambulatory pH monitoring (measurement of acid in the esophagus) was performed on 16 healthy volunteers who received 300 ml of regular coffee, decaffeinated coffee or tap water. Six volunteers received normal tea, decaffeinated tea, tap water, or coffee adapted to normal tea in caffeine concentration. There were 8 who received caffeine-free and caffeine-containing water together with a standardized breakfast.

**Results:** regular coffee induced a significant gastroesophageal reflux compared with tap water and normal tea, which were not different from each other. Decaffeination of coffee significantly reduced gastroesophageal reflux, whereas decaffeination of tea or the addition of tea to water had no effect. Coffee adapted to normal tea in caffeine concentration significantly increased gastroesophageal reflux.

Conclusion: coffee, in contrast to tea, increased gastroesophageal reflux, an effect that is less pronounced after decaffeination. Caffeine does not seem to be responsible for gastroesophageal reflux which must be attributed to other components of coffee.

Aliment Pharmacol Ther 1994; 8(3):283-7.

## **Lecture Presentations by CSD Staff**

Dr. Boyce served as moderator of a symposium on Types, Techniques and Complications of Dilation in Benign Pharyngeal and Esophageal Disorders, and also presented a lecture on Endoscopic Lumen Restoration at the 8th World Congress of OESO in Avignon, France, September 3-6, 2006.

Dr. Boyce was a discussant at the First International Gastrointestinal Eosinophil Research Symposium in Orlando on October 17-18, 2006.

## **Professional Activities by CSD Staff**

Dr. Boyce has been appointed a member of the Permanent Scientific Committee, Section of Endoscopy, of OESO, the World Organization for Specialized Studies on Diseases of the Esophagus.

## DAVID S. ESTORES, JR., M.D.

The Swallowing Center is pleased to announce the addition of David S. Estores, Jr., M.D., Assistant Professor of Medicine, as the third physician on our medical staff. Dr. Estores completed a Fellowship in Gastroenterology and Hepatology at the University of Pittsburgh Medical Center in Pittsburgh, Pennsylvania in 1992. He joined our faculty from the University of Miami School of Medicine, where he served for six years and will serve here as the Director of our Esophageal Physiology Laboratory. He is certified by the American Board of Internal Medicine in the subspecialty of Gastroenterology.

#### THINGS TO REMEMBER

- 1. OFFICE HOURS: 8:00 a.m. until 5:00 p.m. Monday through Friday. Telephone hours: 8:00 a.m. until 5:00 p.m.
  - Our telephone number is 813-974-3374. Also, our after hours emergency telephone number is 813-974-2201.
- 2. **BILLING:** Payment for services rendered is due at the time of your visit. Please be prepared to pay any copayments due at the time of your visit to the Center.

Patients who have problems with their facility or physician fee bills should contact Gayle Stephens, Financial Specialist, at the University of South Florida Medical Clinics at 813-974-3575 between the hours of 9:00 a.m. and 4:00 p.m. Monday through Thursday.

For those patients who are from out-of-town, a new toll-free number has been added for any billing questions. The number is 1-888-873-3627. This number is for calls originating in Florida and is only for billing questions and help with insurance authorizations.

3. HAS YOUR INSURANCE COMPANY OR PRIMARY CARE PHYSICIAN CHANGED? With an ever changing medical insurance market (shopping for the best contract, companies merging, others closing their doors, etc), you may have changed your insurance. If you have changed your insurance company, you may have a new primary care physician. Maybe you have moved and had to choose a new doctor closer to your home. Regardless of the circumstances, we would very much appreciate your contacting our office to let us know at 813-974-3374. This will not only insure we can obtain the necessary authorizations/pre-certifications and that your medical bills go to the right insurance company, but it will help us make sure your medical records are forwarded to the right doctor. Thank you for helping us keep the records straight.

### 8th ANNUAL

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