Dysphagia after anterior cervical spine surgery: pathophysiology, incidence, and prevention.

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412-400-5098**Introduction**

Post-operative dysphagia is a well-known complication of anterior cervical spine surgery. Despite its common occurrence, its incidence and risk varies widely in the published literature[2,3,5,12,14-17]. The reason for this, in part, is due the retrospective nature of most studies that examine this topic[3,12,14,16]. Furthermore, the pathophysiology of post-operative dysphagia is still unclear, and the treatment of persistent dysphagia is uncertain.

Recently, there have been a several prospective studies that have improved understanding of this complication. In addition, cadaver studies examining pathophysiology of dysphagia may allow possible preventive measures to be taken intra-operatively.

The purpose of this writing is to review recent literature on post-operative dysphagia related to anterior cervical spine surgery, the current understanding of its pathophysiology and risk factors, and possible means to minimize this common complication.

Anatomy and Pathophysiology of Dysphagia

Swallowing is divided into three neuroanatomical phases, consisting of oral, pharyngeal, and esophageal[13]. The oral phase begins with entry of the food into the oral cavity, and is dominated by voluntary musculature of the face and intrinsic musculature of the tongue. The tongue, innervated by the hypoglossal nerve, manipulates the bolus of food, and facilitates the mastication through series of rolling motion. The complex coordination of the soft palate, peristalsis of the tongue, salivary glands, and facial muscles are a result of both chemo and mechanoreceptors in the mouth that interchange information between facial, glossopharyngeal, and hypoglossal nerves.

The pharyngeal phase is an involuntary coordination of muscle contractions that begins when the leading edge of food bolus passes posterior to the faucial arch. The critical aspect of this phase include laryngeal elevation and inversion of the epiglottis and closure of true and false vocal folds that prevents seepage of food into the airway. The respiration momentarily ceases during this phase as powerful contractions from superior, middle, and inferior pharyngeal

constrictor muscles clear the bolus from the pharynx. The superior laryngeal nerve (SLN) and recurrent laryngeal nerves (RLN) play an important role in this phase of swallowing.

The esophageal phase begins with passage of the bolus through the upper esophageal sphincter, and ends with passage of food through the lower esophageal sphincter. This phase is also completely involuntary, occurring through the coordinated peristalsis of the esophageal musculature. The neural coordination of esophageal contractions occurs through the autonomous actions in myenteric plexus of Auerbach. This plexus lies between the longitudinal and circular muscle layers of the esophagus, and is triggered by the vagal nucleus.

This succinct review of the swallowing mechanism highlights several at-risk anatomic structures during anterior cervical spine surgery. These include glossopharyngeal, hypoglossal nerves, which are vulnerable in surgeries involving C3 level or above, superior laryngeal nerve in surgeries at C3-C4 levels, and recurrent laryngeal nerve at C6 level or below. The vagus nerve, although normally protected by the carotid sheath during anterior cervical exposures is vulnerable to retraction injury at any sub-axial cervical levels.

The analysis of patterns of dysphagia after anterior cervical surgery can suggest a particular etiology[8,9]. For example, impaired pharyngeal swallowing suggests disruption of pharyngeal plexus and the pharyngeal muscles, which can occur with injury to the superior laryngeal nerve[9]. On the other had, deficits in the oral stage point to the possibility of damage to hypoglossal or glossopharyngeal nerves. Martin et al.[9] evaluated 13 patients who had post-operative dysphagia with videofluroscopic swallow studies to analyze patterns of swallowing abnormality. They found a variety of patterns of dysphagia in all stages of swallowing. Some (3/13) had absent pharyngeal swallow pattern consistent with SLN injury, while others (4/13) had dysphagia with oral preparatory phase with impaired tongue propulsive action consistent with damage to the hypoglossal nerve. Two patients had dysphagia pattern consistent with prevertebral soft tissue swelling causing reduced pharyngeal wall movement, impaired UES opening, incomplete epiglottic deflection, and vallecular, pyriform sinus, and posterior pharyngeal wall residue.

Other studies have highlighted pre-existing swallowing dysfunction in many patients undergoing anterior cervical surgery. For example, Frempong-Boadu et al.[5] found that 66% of patients with myelopathy had pre-operative swallowing abnormality seen with barium swallowing study. This may suggest a centrally located mechanism of dysphagia, where spinal cord compression may cause swallowing dysfunction by interfering with the preganglionic, sympathetic outflow or spinal afferents that interrupts local reflex mechanisms. This suggests that a sub-group of surgical candidates are more susceptible to post-operative swallowing dysfunction.

Incidence and Natural History

The published incidence of dysphagia after anterior cervical surgery ranges from less than 2% to greater than 50% (Table 1). This may be due to the retrospective nature of many of these studies, which can be affected by recall and surgeon's bias. At this writing, three notable prospective studies have been published that have contributed to the understanding on incidence and natural history of post-surgical dysphagia[2,5,15].

Frempong-Boadu et al.[5] evaluated 23 patients undergoing elective ACDF prospectively during 1-year period. The patients were evaluated with barium swallow study pre-operatively and 1-month post-operatively. Their analysis showed that 48% of their cohort (66% with myelopathy) had *pre*-operative swallowing abnormality. Post-operatively, 67% of patients who did not have pre-existing radiographic findings demonstrated new barium swallow abnormalities.

Overall incidence of dysphagia at one month was 48%, and age diabetes, hypertension, smoking, nutritional status were not associated with post-operative swallowing abnormalities. Also, post-operative prevertebral or pharyngeal swelling was observed in 61% of patients, and 86% of these patients had abnormal swallowing tests.

Bazaz et al.[2] prospectively analyzed 249 patients who had anterior cervical surgery. They contacted the patients at 1, 2, 6, and 12 months and rated the dysphagia as "mild", "moderate", or "severe" based on a scoring system devised by the authors (Bazaz-Yoo dysphagia score). Risk factors of age, gender, procedure type, hardware use, and number/location of the levels were evaluated. At one month, 50.3% (99/197) had some level of swallowing difficulty, and 5.6% (11/197) were severe. At 6 months, 17.7% (37/208) experienced dysphagia, and by 12 months 12.5% (19/152) had dysphagia. Female gender was a risk factor at 6 months, but age, primary vs. revision, corpectomy vs. discectomy, or placement of hardware was not a risk factor at any time points. Multiple-level surgery was a risk factor at one month, but by 6 months, this statistical difference disappeared.

Smith-Hammond et al.[15] performed a 3-year prospective study to evaluate incidence of dysphagia on anterior cervical patients. Notably, this study used patients who underwent posterior cervical or posterior lumbar procedures during the same time period as a control group. Swallowing was assessed pre-and post-operatively by subjective questions as well as videofluoroscopic swallow evaluation (VSE) or fiberoptic endoscopic evaluation of swallowing (FEES). Post-operative evaluations were performed 2.0+/-1.5 days after the index surgery. A total of 83 patients were evaluated. The incidence of dysphagia in the anterior cervical surgery group was nearly 50% (18/38). The majority (71%) of these patients with dysphagia returned to regular diet within 2-months. Age (>60 years) was found to be a statistically significant risk factor, whereas other co-morbidities including hypertension, diabetes mellitus, smoking, gastroesophageal reflux disease, chronic obstructive pulmonary disease, alcohol abuse, substance abuse, coronary artery disease, cancer, and myelopathy were not significant. Since the study was based at a Veteran's Affairs Medical Center, a majority of the patients were male, and gender could not be analyzed as a risk factor. None of the surgical variables, including the use of instrumentation, duration of surgery, operated spinal levels, and number of spinal levels were significant risk factors. Interestingly, 20% (4/19) patients who had posterior cervical surgery had post-operative dysphagia, but the authors did not feel that endotracheal intubation itself was a risk factor in their cohort.

The results of these prospective studies indicate that surgeons should expect approximately 50% of patients to have some degree of swallowing dysfunction peri-operatively. By 6-months, a majority of the patients should improve, with about 10% of patients experiencing dysphagia at 12-months. Female gender, age, pre-existing swallowing dysfunction, and multiple-level surgery may be risk factors for post-operative dysphagia.

Studies that analyze long-term (> 12-months) dysphagia are rare. One recent study by Yue et al. evaluated 74 patients who returned for follow-up at an average of 7.2 years after anterior cervical spine procedures. At final review, a varying degree of dysphagia was present in 35% of patients. Only 12 of 74 patients (16.2%), however, had "moderate" dysphagia as scored by Bazaz-Yoo dysphagia index, and one patient (1.4%) had severe dysphagia. These authors also found female gender as a risk factor. This study may indicate that dysphagia that does not resolve by 12-months may persist throughout the patient's lifetime.

Prevention

Recent findings on preventive measures for post-operative dysphagia suggest that certain intra-operative routines may minimize this complication. Apfelbaum and Kiscovich et al.[1,6] performed a cadaveric study in order to minimize endotracheal tube (ET) related injury to the recurrent laryngeal nerve. Their hypothesis was that fixed ET with inflated cuff and tape at the mouth could exert asymmetric pressure to the trachea after a cervical retractor is placed. This can cause pressure related damage to the recurrent laryngeal nerve. To test this hypothesis, a cadaver study was done where the ET cuff was inflated to 15 mmHg and taped, and the cuff pressure was measured after the retractor was placed. They found that the cuff pressure increased up to 52 mmHg after the retractor placement. A simple maneuver of releasing and re-inflating the cuff after retractor placement allowed a more central positioning of ET within the endolarynx. Using this data, they performed 650 anterior cervical procedures in patients where this re-inflation maneuver was done. They found that with this simple maneuver, the overall rate of RLN palsy fell to 2.0% from 6.8%.

Similarly, Ratnaraj et al.[11] performed a study where the intra-operative ET cuff pressure was strictly monitored throughout the procedure. In the experimental cohort, the ET cuff pressure was constantly measured and adjusted to be about 20 mmHg, whereas, in control cohort, the ET cuff pressure was not monitored or adjusted. A blinded observer questioned the patients post-operatively for sore throat, hoarseness, and dysphagia at 1-hour, 24-hours, and 1-week post-operatively. At 24-hours the experimental group has significantly less sore throat compared to the control group (51% vs. 74%). At 24-hours, longer retraction time correlated with development of dysphagia. They concluded that maintaining low (~20 mmHg) ET cuff pressure and minimizing retraction times may decrease post-operative sore throat and dysphagia.

Lee et al[7]. recently showed that anterior cervical plate design may be a factor in causing dysphagia. In their prospective study, patients who had anterior cervical arthrodesis using the Zephir cervical plate (Medtronic-Sofamor Danek, Memphis Tennessee) had significantly less dysphagia at 1-year follow-up than those with Atlantis cervical plate, made by the same manufacturer. The authors attributed this difference to the lower profile and smoother surface of the Zephir system. These findings suggest that at least some persistent long-term dysphagia can be attributed to adhesions between posterior esophagus and the plate. Along these lines, Fogel et al.[4] reported on retrospective analysis of 44 patients referred to the otolaryngology service with persistent dysphagia. These patients ultimately underwent removal of anterior cervical plating and lysis of adhesions. Operative findings at the instrumentation removal included extensive adhesions attaching the esophagus-trachea midline structures to the prevertebral fascia and anterior aperture in the plate. Comparison of Bazaz-Yoo dysphagia score after instrumentation removal showed significant improvement overall, although mild dysphagia continued in 36% of the patients.

Preventive methylprednisolone infusion has recently been investigated. Pedram et al.[10] prospectively infused 78 patients with no history of upper gastrointestinal tract disorder and good tolerance to anti-inflammatory treatment with 1-mg/kg methylprednisolone at the end of operation, at 12-hours, and at 24-hours. In comparison to the control group, methylprednisolone *did not* reduce the incidence of subjective dysphagia, but it did seem to decrease objective findings of abnormal pharyngeal wall, arytenoids and vocal cords during post-operative fiberoptic examination. Because of the complications that can occur with intravenous steroid infusion, the decision to preemptively treat patients with methylprednisolone should be made cautiously.

Summary

Given above discussions, following summary can be made regarding risk factors and preventive measures of post-operative dysphagia.

- An extensive pre-operative discussion should take place with the patient regarding this common complication. The patient should be warned that approximately 50% of the patients will experience some degree of dysphagia peri-operatively, and that about 10% may continue to have dysphagia at long-term. This holds especially true of female patients.
- Older (>60-years) patients, patients undergoing multilevel surgery, and patients with pre-existing swallowing dysfunction are especially at risk for persistent dysphagia.
- Know the anatomy of at-risk structures: glossopharyngeal and hypoglossal nerves above C3, superior laryngeal nerve at C3-4, and recurrent laryngeal nerve and vagus trunk at lower cervical areas.
- Meticulous tissue dissection and hemostasis should be performed intra-operatively to prevent scar tissue formation.
- Endotracheal tube cuff deflation and re-inflation after placement of retractors is recommended to minimize recurrent laryngeal nerve damage. Maintaining ET cuff pressure < 20 mmHg throughout the procedure may be beneficial.
- Keep retraction time to a minimum. Relaxing the retractors during surgical pauses (ie. during bone graft preparation) will minimize traction injury to neural and muscular structures.
- Use of smoother contour and lower profile plates may decrease incidence of dysphagia.
- Use of methylprednisolone as a preventive measure remains controversial.

Table 1. A sample of published literature on incidence of dysphagia

Author (year)	No. of Patients	Incidence of dysphagia
Caspar et al. (1989)[3]	60	1.7%
Saunders et al. (1991)[12]	40	2.5%
Stewart et al. (1995)[16]	73	45%
Martin et al. (1997)[9]	247	6.5%
Schneeberger et al. (1999)[14]	35	5.7%
Frempong-Boadu et al. (2002)[5]*	23	48%
Bazaz et al. (2002)[2]*	249	50.3% (at one month)
Smith-Hammond et al. (2004)[15]*	83	50%
Yue et al. (2005)[17]	74	35.1% (average 7.2 year follow-up)

*Prospective Study

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