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In Reference to "Partial Epiglottoplasty for Pharyngeal Dysphagia due to Cervical Spine Pathology"

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No sponsorships or competing interests have been disclosed for this article.

Jamal et al recently described the role of epiglottoplasty in the treatment for pharyngeal dysphagia caused by anterior cervical osteophytes.[1] The article suggests that large cervical osteophytes prevent the complete retroflexion of the epiglottis, causing vallecular pooling and dysphagia; thus, removal of a portion of the epiglottis will improve these symptoms, without necessitating more complex and debilitating spine surgery. While this is a simple yet interesting concept, we do have some comments on the study.

First, of the 9 patients who received epiglottoplasty, 3 had previous anterior cervical spine (c-spine) surgery. Dysphagia following anterior c-spine surgery is a well-described complication and can persist for up to a year following surgery.[2] Dysphagia may be due to swelling and prominent hardware, as posited in the paper. However, dysphagia may also be due to disruption of vagal afferents and motor fibers to the pharynx or to decreased laryngeal elevation as a result of instrumentation, among others. Patients who had previous c-spine surgery should be excluded from the study, as it is difficult to assess the true nature of their dysphagia and whether epiglottoplasty is truly of benefit.

Second, no information in the study is reported regarding the level, size, or number of osteophytes when the epiglottoplasty was performed. One can imagine that large osteophytes closer to the larynx or esophageal inlet may cause dysphagia by causing narrowing in this area, rather than by vallecular pooling. Therefore, epiglottoplasty may not be of benefit and may cause worsened dysphagia by removing its protective function.

Overall, we congratulate the group for this intriguing concept and novel treatment for dysphagia. We feel that this procedure would be of most benefit in the correctly selected patient—that is, a patient without previous c-spine surgery where the main source of dysphagia is due to impaired epiglottic retroversion. It may be best suited as a procedure for patients unwilling or unable to undergo anterior c-spine surgery due to medical issues. Further study including a larger number of patients and more complete information regarding the nature of the dysphagia is clearly needed before widespread adoption of the procedure is considered.

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1. Jamal N, Erman A, Chhetri DK. Partial epiglottoplasty for pharyngeal dysphagia due to cervical spine pathology. *Otolaryngol Head Neck Surg*. 2015;153:586-592.
2. Riley LH 3rd, Vaccaro AR, Dettori JR, Hashimoto R. Postoperative dysphagia in anterior cervical spine surgery. *Spine (Phila Pa 1976)*. 2010;35(9):S76-S85.

Authors' Response to Letter: "In Reference to 'Partial Epiglottoplasty for Pharyngeal Dysphagia due to Cervical Spine Pathology'"

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We appreciate the comments by Drs Amit Patel, Christopher G. Tang, and Andrew Blitzer regarding our article "Partial Epiglottoplasty for Pharyngeal Dysphagia due to Cervical Spine Pathology."[1] As the title suggests, the unifying pathophysiology of the epiglottic dysfunction reported in the patients was pharyngeal crowding due to cervical spine abnormalities,

causing an inability of the epiglottis to retroflex adequately during deglutition. The epiglottis then acted as an impediment to swallowing, instead of moving out of the way of the food bolus. Cervical osteophytes were one cause of pharyngeal crowding. However, pharyngeal crowding was also seen after anterior cervical spine surgery due to an increase in pharyngeal wall thickness. We therefore included both groups under "cervical spine pathology."

Dysphagia after anterior cervical spine surgery may occur from a variety of factors, including disruption of the pharyngeal plexus, scarring of the fascial planes, and prominent hardware. However, a subset of these patients continues to have symptomatic dysphagia due to epiglottic dysfunction despite swallow therapy and an adequate time given for healing, with no therapeutic alternatives. Thus, for patients with food bolus obstruction due to epiglottic dysfunction from pharyngeal crowding and with minimal to no laryngeal penetration during the swallow, partial epiglottoplasty is a low-risk alternative to anterior cervical spine surgery.

In regard to the level of the osteophytes causing epiglottic dysfunction, these are present at the level of the epiglottis and are evident upon review of the modified barium swallow study or fiberoptic endoscopic evaluation of swallowing (Figure 1 in article[1]). Certainly, osteophytes may cause dysphagia without causing epiglottic dysfunction. These cervical osteophytes and those that are located lower, around the level of the cricoid cartilage, are better treated with an open resection of the osteophytes. With regard to those osteophytes causing epiglottic dysfunction, however—based on our personal, unpublished observations—the size and number of osteophytes do not appear to be as important as the functional impairment of the epiglottis due to them. Thus, we carefully review the preoperative swallow study and focus on the functional impairment caused by osteophytes at the level of the epiglottis. If the osteophytes do not cause epiglottic dysfunction (or dysphagia in some other way), then they do not need treatment, no matter their size or number.

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A Revision in Evaluating the Results of Intratympanic Otoprotective Injections against Cisplatin-Induced Ototoxicity

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This letter is written in response to the article entitled "Cisplatin-Induced Ototoxicity and the Effects of Intratympanic Diltiazem in a Mouse Model," by Naples and Parham.[1] Recently, some researchers have focused on evaluating the effectiveness of intratympanic injections of agents such as N-acetyl cysteine, D-l-methionine, lactate, vitamin C, and diltiazem against cisplatin-induced ototoxicity in patients receiving chemotherapy. The possibility of cytokine-related mechanism of this side effect has evolved researchers to also try intratympanic injections of corticosteroids such as dexamethasone and betamethasone.[2,3] Here, I want to discuss the methodological shortcomings of such studies and to criticize their design.

First, almost all of these studies usually use 1 of the 2 ears as the control and the other as the case to compare the otoprotective effect of the injections before and after the course of chemotherapy with cisplatin. Although this design seems logical and the idea is praiseworthy, some evidence implies that the ototoxicity of cisplatin may not necessarily be the same bilaterally; therefore, it is not comparable in 2 ears as case and control.[4]

Second, the last audiodiagnostic test in such studies is usually done immediately or a short while after the last injection of the otoprotective agent, while there is evidence that cisplatin-induced ototoxicity can occur a long time after the last dose of cisplatin. As Peleva et al showed a rate of 70% ototoxicity in a 60-month follow-up, it was 48% immediately after the last dose of cisplatin.[5]

Third, pure tone audiometry and distortion product otoacoustic emission, which are the most common audiodiagnostic tests used in such studies, are able to detect changes in only a limited spectrum of frequencies, while there is evidence showing that cisplatin-induced ototoxicity may occur in very high frequencies, something between 10 and 20 kHz, which needs special tests, such as extended high-frequency pure-tone audiometry. For example, in one study in 2014, 7 patients out of 10 showed ototoxic changes after receiving cisplatin, from whom 4 patients