EDITORIAL

https://doi.org/10.5125/jkaoms.2018.44.1.1
pISSN 2234-7550 · eISSN 2234-5930

Management of velopharyngeal dysfunction: what is the role of oral and maxillofacial surgeons?

Seung-O Ko, D.D.S., Ph.D.

Department of Oral and Maxillofacial Surgery, School of Dentistry, Chonbuk National University, Jeonju, Korea

The nasal and oral cavities must be completely closed off during swallowing, vomiting, blowing, sucking, whistling, and talking. This velopharyngeal (VP) closure is especially important when producing pressure sensitive sounds. During normal VP function, the posterior third of the soft palate moves posterior-superiorly, while the pharyngeal wall moves anteriorly and medially to form a sphincter to close the oral and nasal cavities. In a small group, the formation of a Passavant ridge on the posterior pharyngeal wall may contribute to the closure. Four basic types of closure patterns are used to describe velar closure: coronal, sagittal, circular, and circular with a Passavant ridge. Defining the closure pattern is important for surgical intervention for correction of VP dysfunction (VPD). It is generally agreed that three muscles contribute to VP closure. The levator veli palatine, when contracted, pulls the velum up and back against the posterior nasopharyngeal wall. The sphincter complexity of the superior constrictor acts as a pseudosphincter and can close the VP port circumferentially. Finally, the muscularis uvula provides thickness to the post. A third of the velum helps to occlude the VP port[1].

VPD is a term describing inappropriate functioning of the VP port, which consists of lateral and posterior pharyngeal walls and the soft palate. This muscular valve can control the air passage between the oro- and nasopharynx. The impairment of VP function can be attributed to structural, neurologic, and speech causes. Even though there is sufficient soft tissue to close the VP port with normal anatomical structures, VP function can be disrupted (VP incompetence, VPI) due to neuromuscular disorders such as cerebral palsy, myotonic dystrophy, and cerebral vascular accidents. Soft tissue deficiency for closing the VP port as a result of surgical removal or congenital loss of the normal structure separating the nasal and oral cavities can lead to a state called VPI.

The most common cause of VPI is overt cleft palate. Despite successful palatoplasty, the incidence of VPI after surgery has been reported to be as high as 20% to 50%. VPI may also be seen in patients with a submucous cleft palate or an occult submucous cleft palate. VPIs rarely occur after adenoidectomy, while tonsillar hypertrophy may restrict VP closure, causing VPI[2]. Also, several syndromes may manifest with VPI with or without cleft palate. Most commonly, velocardiofacial syndrome (VCFS) has a very large spectrum of phenotypes and often includes facial dysmorphisms, cardiac anomalies, and VPD. The effects of VPD on a patient's speech include hypernasality, nasal emissions, and decreased speech intelligibility. The severity of speech impairment depends on several factors including the amount of gap with a closed velum, the patient's articulation and oral motor ability, and compensatory strategies the patient may have developed to decrease nasal emissions or hypernasality. The diagnosis of VPD, identifying a critical cause of the dysfunction, can be carried out through physical and oral examinations, perceptual speech assessment, radiographic multiplanar videofluoroscopy, and nasendoscopy.

Treatment options of VPD include surgical and prosthetic interventions in combination with speech therapy. Speech therapy is the mainstay of treatment of patients with VPD. It is also essential to treat compensatory misarticulations resulting from VPI. It is important that speech therapy continues after surgical correction of an anatomic defect when misarticulations exist. Surgical treatment is typically the first line of treatment for VPI. Various surgical techniques, such as pha-

Seung-O Ko
Department of Oral and Maxillofacial Surgery, School of Dentistry, Chonbuk National University, 567 Baekje-daero, Deokjin-gu, Jeonju 54896, Korea
TEL: +82-63-250-2211 FAX: +82-63-250-2089
E-mail: omfskso@chonbuk.ac.kr
ORCID: http://orcid.org/0000-0001-7504-7557

ryngeal flap surgery, sphincter pharyngoplasty, and Furlow palatoplasty, have been used[3], but the success rate is approximately 50%. Prosthetic devices for VPD can be used as an alternative treatment method when the surgical approach is not considered. Widely used types of these devices, called speech aids, include the palatal lift appliance and speech bulb. Palatal lifts serve to elevate the neurogenic palate with reduced motion or decreased accurate timing of soft palate elevation to achieve VP closure. Generally, children requiring these treatments will have adequate velar length, but poor muscle tone. In contrast, speech bulbs are fashioned to fill the open space between the soft palate and posterior pharyngeal wall in cases of insufficient palate length. The lateral and posterior walls of the pharynx can then close against the obturator.

Speech aids can be implemented to treat VPI due to cleft palate and can also be effectively applied to VPI caused by neuromuscular disorders, adenoidectomy, and idiopathic pharyngeal hypotonia[4]. Many researchers recommend that prosthodontists or orthodontists be the ones to fabricate the speech aid. Frankly, speech aids are simple devices that any dentist can fabricate. However, the design of the functional part must reflect the anatomical features and function of the velopharynx and the characteristics of the soft palate after surgical treatment. Therefore, a dentist who understands the anatomy and action of the velopharygeal area with extensive knowledge of VP insufficiency should be in charge of creating the speech aid.

Management of VPD is most accurately performed through evaluation within the context of a multidisciplinary team including a VPD surgeon, an experienced speech and language pathologist, and an otolaryngologist.

Oral and maxillofacial surgeons are a distinctive group of dentists who not only possess the knowledge of dentition, occlusion, and prosthetics, but also thoroughly comprehend the anatomy and function of the velopharynx and the cause, symptoms, and surgical treatment of VPI. Therefore, oral and maxillofacial surgeons should pay close attention to not only VPI due to cleft palate, but also VPI with other etiologies.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References

1. Glade RS, Deal R. Diagnosis and management of velopharyngeal dysfunction. Oral Maxillofac Surg Clin North Am 2016;28:181-8.
2. Brigger MT, Ashland JE, Hartnick CJ. Diagnosis and treatment of velopharyngeal insufficiency. In: Hartnick CJ, Boseley ME, eds. Clinical management of children's voice disorders. San Diego: Plural Publishing; 2009:225-244.
3. Setabutr D, Senders C. Surgical management of velopharyngeal dysfunction. Operative Techniques in Otolaryngology 2015;26:33-8.
4. Shin YJ, Ko SO. Successful and rapid response of speech bulb reduction program combined with speech therapy in velopharyngeal dysfunction: a case report. Maxillofac Plast Reconstr Surg 2015;37:22.