Diagnosis and Management of Velopharyngeal Dysfunction

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KEYWORDS

• Velopharyngeal dysfunction • Velopharyngeal insufficiency • VPI • VPD • Nasoendoscopy • Nasopharyngoscopy • Videofluoroscopy • Palatoplasty

KEY POINTS

- Velopharyngeal dysfunction (VPD) describes any condition whereby the velopharyngeal valve does not properly close during the production of oral sounds, with multiple causes, including velopharyngeal mislearning (nasopharyngeal sound substitution for an oral sound), velopharyngeal incompetence (neurolophysiologic dysfunction causing poor pharyngeal movement), and velopharyngeal insufficiency (a structural or anatomic defect prevents velopharyngeal closure).
- Evaluation for VPD is best performed within the context of a multidisciplinary team and consists of history and physical examination, perceptual speech evaluation, and instrumental assessment of speech with either video nasoendoscopy or multiview speech fluoroscopy.
- Speech therapy is the mainstay in treatment of velopharyngeal mislearning, while velopharyngeal inadequacy and insufficiency may require surgical intervention after a trial of speech therapy.
- Surgical correction of VPD is based on the size and location of the velopharyngeal gap seen during instrumental assessment and includes posterior wall augmentation, Furlow palatoplasty, sphincter pharyngoplasty, and pharyngeal flap.

Video content accompanies this article at http://www.oralmaxsurgery.theclinics.com/

INTRODUCTION

With the exception of /m/, /n/, and /ng/, all phonemes within the English language are produced orally and require complete or nearly complete closure of the velopharyngeal mechanism to be perceived as normal. If air or sound is allowed to leak through or resonate in the nasal chamber during production of the nonnasal sounds, speech will be marked by hypernasal resonance and nasal air emission.

ANATOMY

It is generally agreed that 3 muscles contribute to velopharyngeal closure. The levator veli palatini is a paired muscle that originates on the inferior surface of the temporal bone near the torus tubarius (where the eustachian tube exits the temporal bone) and along the medial lamina of the eustachian tube cartilage. The fibers course downward and forward, intertwining with fibers of the superior constrictor muscle in the lateral nasopharyngeal wall. They ultimately insert into the velum at a 45° angle where they meet the contralateral fibers. Most of the fiber pairs are found in the mid one-third of the velum, where they form a slinglike structure. When contracted, levator pulls the velum up and back against the posterior nasopharyngeal wall (above the level of the atlas) while also

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collapsing the side walls of the nasopharynx medially. In this manner, the entire velopharyngeal port can be closed. The sphincter complex of the superior constrictor is paired and originates at the pharyngeal raphe. Its fibers course forward and medially to enter the velum from either side. On contraction, the sphincter complex acts as a pseudosphincter and can close the velopharyngeal port circumference. In some patients, the posterior pharyngeal wall is pulled anteriorly by this muscle complex, creating an anterior bulging of the posterior pharyngeal wall during velopharyngeal closure. This phenomenon is called Passavant pad (Video 1). Curiously, sometimes it occurs too low in the posterior pharyngeal wall to aid velopharyngeal closure during speech. Finally, muscularis uvula is paired and originates on either side of the posterior nasal spine. The fibers course posteriorly, approaching the velum. On contraction, they add thickness to the posterior third of the velum, helping to occlude the velopharyngeal port.

TERMINOLOGY

Several terms have been used interchangeably to describe the multiple causes responsible for inappropriate airflow through the nasopharynx during speech; this has led to redundancy and confusion in both medical literature and communication between practitioners. In this article, the following commonly accepted definitions are used. Velopharyngeal dysfunction (VPD) describes any condition in which the velopharyngeal valve does not close completely and consistently during the production of oral sounds. VPD has multiple causes, which are broadly grouped into 3 distinct subgroups based on root cause (**Fig. 1**). These causes include velopharyngeal mislearning (VPM), velopharyngeal incompetence, and velopharyngeal insufficiency (VPI). VPM describes the creation of certain sounds within the nasopharynx as a substitution for oral sounds. This behavior is learned, with no anatomic or neurophysiologic source. Velopharyngeal incompetence refers to a neurophysiologic disorder resulting in poor velopharyngeal movement. Palatal structure and anatomy are normal, including length, but poor movement prevents complete closure of the velopharyngeal valve. VPI describes a structural or anatomic defect that prevents closure of the velopharyngeal mechanism, such as overt or submucous cleft palate.

Distinguishing the precise cause of VPM is essential, because the treatment of VPM, velopharyngeal insufficiency, and VPI varies.

CAUSE

The most common cause of VPI is children with an overt cleft palate. Despite successful palatoplasty, the incidence of VPI after surgery has been reported to be as high as 20% to 50%.[1,2] VPI is also seen in submucous cleft palate, where no overt cleft is seen, but the levator muscle fibers fail to fuse in the midline. These VPIs classically manifest with the triad of a bifid uvula, diastases in the midline (caused by insertion of the levator muscles onto the hard palate rather than into a midline raphe), and hard palate notch. Interestingly, many children with submucous cleft will have no evidence of VPI during their lifetime, and management is only required when symptoms exist.[3] In an occult submucous cleft palate, similar to submucous cleft palate, the levator muscles insert onto the posterior hard palate but a bifid uvula or midline diastasis is not present.[4] Occult submucous cleft palate is best diagnosed by video nasopharyngoscopy, where a sagittal orientation of the levator muscles is noted with an absence of the muscularis uvulae (Video 2). VPI is rarely

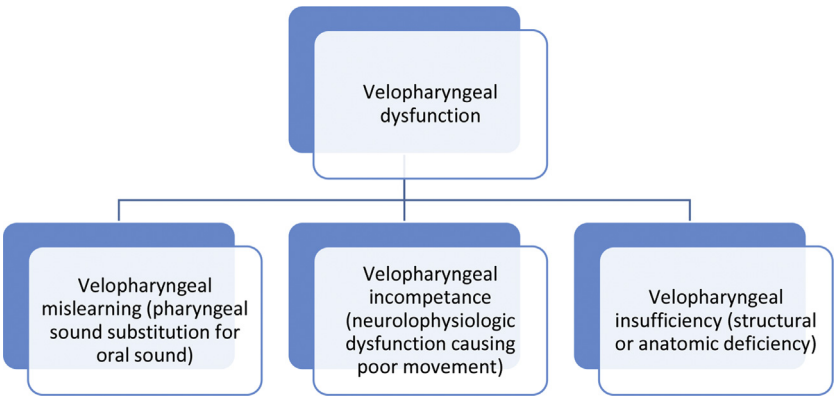

Fig. 1. Classification of VPD.