Surgical Management of Velopharyngeal Dysfunction

Jill Arganbright, MD

KEYWORDS

• Velopharyngeal dysfunction • Pharyngeal flap • Sphincter pharyngoplasty • Furlow palatoplasty • 22q11.2 deletion syndrome • Velopharyngeal insufficiency • Velopharyngeal inadequacy • Hypernasal speech

KEY POINTS

- Velopharyngeal dysfunction (VPD) is caused by inadequate closure of the velopharyngeal port. VPD can hinder a child's ability to communicate and impact his/her quality of life.
- Causes of VPD are multiple and include anatomic and neuromuscular causes, as well as behavioral/mislearning. It is important to identify the underlying cause as this can have implications for treatment options.
- Multiple surgeries exist for the management of VPD including pharyngeal flap, sphincter pharyngoplasty, buccal myomucosal flaps, Furlow palatoplasty, palate re-repair, and injection pharyngoplasty. Each speech surgery has its unique benefits and drawbacks associated with it.
- VPD evaluation and surgical recommendations are often made in a multidisciplinary setting. Deciding which surgery to choose for a specific patient often hinges on the type of closure pattern and gap size noted when visualizing velopharyngeal closure.
- Ultimately, the choice of speech surgery should be individually tailored to each child based on their specific needs and weighing the risk/benefit profile for their specific surgeries.

INTRODUCTION

Velopharyngeal dysfunction (VPD) is a term to describe any situation in which the space between the mouth and the nose (velopharynx) is not completely closing off during speech. This leads to an inappropriate leakage of air into the nasal passages during speech.[1] For example, in the English language, all sounds except the -m, -n, and -ng sound require complete closure of the space within the velopharynx which allows the air to be solely directed out of the mouth during speech production. For the velopharynx to close, a complex group of muscles act in unison, including the elevation of the soft palate and constriction of the lateral and/or posterior pharyngeal walls.[1,2]

Manifestations of VPD can include hypernasality, nasal air emissions, decreased vocal loudness, nasal grimacing, poor speech intelligibility, and resultant obligatory and compensatory misarticulation.[3] Additionally, children with VPD can frequently develop maladaptive articulations to compensate for their speech difficulties, called compensatory speech errors.[1] VPD is important to identify and treat because it impacts a child's quality of life[4–6] and affects the child's future ability to live independently and participate fully in society.[7]

WHAT CAUSES VELOPHARYNGEAL DYSFUNCTION?

Causes of VPD are multiple and include anatomic and neuromuscular causes, as well as behavioral/mislearning[1] (**Fig. 1**). VPD can be caused by abnormal *anatomic structure*. This type includes children with insufficient tissue/structure

Children's Mercy Hospital, University of Missouri-Kansas City, 2401 Gillham Road, Kansas City, MO 64108, USA
E-mail address: jarganbright@cmh.edu

Facial Plast Surg Clin N Am 32 (2024) 69–83
https://doi.org/10.1016/j.fsc.2023.06.007
1064-7406/24/© 2023 Elsevier Inc. All rights reserved.

Descargado para Biblioteca Medica Hospital Mexico (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

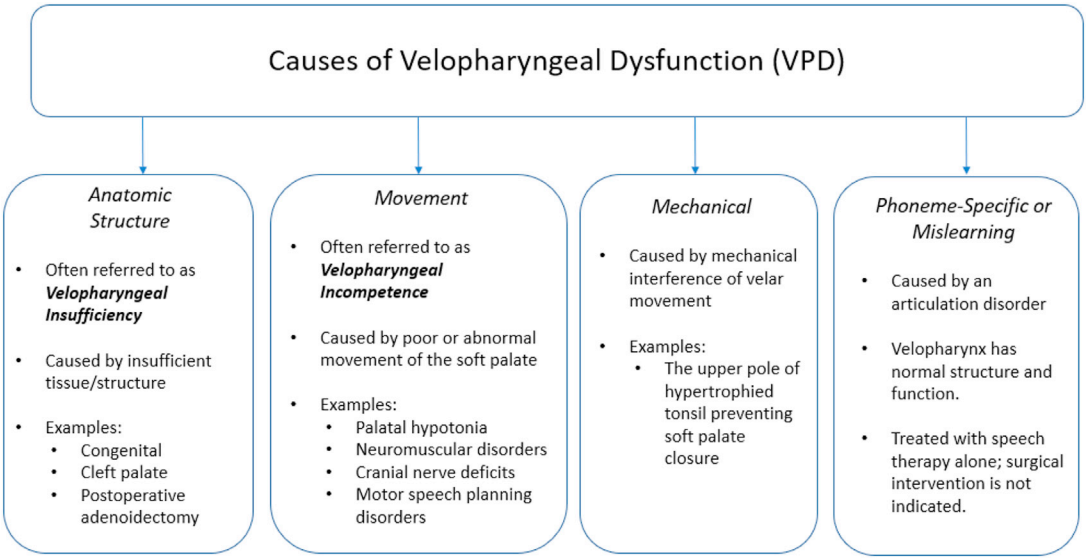

Fig. 1. Causes of velopharyngeal dysfunction. Original.

to obtain velopharyngeal closure and, as such, is often referred to as *velopharyngeal insufficiency* (VPI). Anatomic causes are the most common causes of VPD and include palatal clefting, whether overt cleft palate or submucous cleft palate (SMCP).[1] VPD caused by the abnormal/poor *movement* of the velopharyngeal structures is often referred to as *velopharyngeal incompetence*. This type includes children with palatal hypotonia, neuromuscular disorders (including 22q11.2 deletion syndrome), cranial nerve deficits, or motor speech planning disorders such as childhood apraxia of speech. Children may have one or a combination of these issues influencing or causing their VPD.[1]

VPD can also be caused by a *mechanical* obstruction, namely when the upper pole of a hypertrophied tonsil[8,9] acts as a stent and physically blocks the ability of the soft palate to close properly.[1] There is also a separate cause of VPD, which is *phoneme-specific or mislearning*. This type of VPD is an articulation disorder and a frequent source of VPD. In phoneme-specific VPD, the velopharynx has normal structure and function. However, the child has "learned" with certain sounds to keep the velopharynx open for sounds for which it should be closed. This type of VPD occurs only on specific sounds; for the remainder of speech sounds, the velopharynx is working appropriately. This phoneme-specific VPD is treated with speech therapy alone; knowing this highlights the importance of differentiating phoneme-specific VPD from other causes of VPD, as surgical intervention is not indicated for children with phoneme-specific VPD.[1]

WHO GETS VELOPHARYNGEAL DYSFUNCTION?

A wide array of children presents with VPD. One of the most common causes is cleft palate, including both overt cleft palate and SMCP. Even after palate repair, 20-50% of children with cleft palate will have persistent VPD.[7,10] VPD is also considered a hallmark of 22q11.2 deletion syndrome (22q11.2DS), independent of the presence or absence of overt palatal clefting. 22q11.2DS is the most common cause of syndromic palatal anomalies and VPD. Up to 75% of children with 22q11.2DS have palatal involvement, which includes overt cleft palate, SMCP, and VPD in the absence of clefting. Children with speech disorders such as apraxia, neuromuscular disorders, and cranial neuropathies can have difficulties with palatal movement and subsequent VPD. VPD is seen in various genetic differences and in children with generalized hypotonia.[3] While it is rare, VPD can occur following adenoidectomy with a frequency of 1:1,500-1:10,000.[11]

DIAGNOSING VELOPHARYNGEAL DYSFUNCTION

The evaluation of a child with concerns for VPD is best completed by a multidisciplinary team that includes a speech-language pathologist (SLP) and a surgeon trained in VPD. A combination of modalities should be used to perform a complete evaluation for VPD. First, a thorough history and physical should be completed (**Figs. 2** and **3**) as well as an audiogram. Second, a perceptual speech evaluation by

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

History

- Medial history (including cleft palate, syndromes, hypotonia, learning delays, cerebral palsy)
- Surgical history (prior palate surgeries, craniofacial, adenoidectomy, tonsillectomy, brain surgeries)
- Subjective speech concerns from patient/family
- Prior/current speech therapy
- Percent parents can understand of child's speech
- Percent a stranger can understand child's speech
- Nasal regurgitation
- Snoring and obstructive breathing at night
- Speech impact on quality life

Fig. 2. Patient history items to discuss when evaluating a patient with VPD. Original.

a SLP should be performed, which provides the foundation for the diagnosis. The next step in evaluation is the visualization of the velopharynx, which has traditionally been accomplished using nasopharyngoscopy or multiview videofluoroscopy or both. Nasopharyngoscopy would ideally be completed in a child that is able to voluntarily repeat speech samples while the scope is in the nose. Variables assessed during these exams include velar length, velar structure (evidence of SMCP), pharyngeal width and depth, lateral and posterior pharyngeal wall movement, velar movement (asymmetric, sluggish, bouncy), velopharyngeal (VP) closure pattern, VP gap size, tonsil and adenoid size/positioning, and timing of VP closure.[3] Lipira and colleagues[12] evaluated the relative benefits of nasopharyngoscopy versus videofluoroscopy and concluded that the studies were best used in tandem to optimally evaluate children with VPD. Nasometry can also be performed; this is a computerized tool that can objectively quantify the degree of nasal air loss and compare it to normative data. Newer technology has allowed for magnetic resonance imaging (MRI) to be used as a tool to evaluate VPD. MRI allows for the visualization of the underlying VP structures and musculature during speech, which could impact surgical planning.[13]

Physical Exam

- Complete head and neck exam with focus on:
- Presence of nasal grimace
- Hard palate exam
- Soft palate:
  - Is there movement with phonation?
  - Is this movement symmetric?
  - Evidence of a submucous cleft palate?
    - Palpate for a hard palate notch
  - Are there any soft tissue defects?
- Tonsil exam
- Jaw position

Fig. 3. Pertinent physical exam when evaluating a patient with VPD. Original.

Each type of exam allowing the visualization of the velopharynx (nasopharyngoscopy, videofluoroscopy, MRI) has its benefits and limitations, and considerable variability currently exists among providers and institutions regarding their study (or studies) of choice for imaging the velopharynx. Regardless of which method is used, the direct visualization of the VP mechanism during speech is important for determining whether the child would benefit from speech surgery. Further, it assists in determining which speech surgery to recommend and allows the surgeon to tailor the specific surgery to the specific needs of the child.

Role of Speech Therapy in Children with Velopharyngeal Dysfunction

Regardless of VPD etiology, most children with VPD will benefit from an appropriate course of speech therapy to optimize their ability to communicate.[1] If the child is found to have phoneme-specific VPD, speech therapy is the preferred treatment; no surgical intervention is recommended. For children diagnosed with VPI and/or velopharyngeal incompetence, surgical options should be considered.

Surgical Treatment for Velopharyngeal Dysfunction: Choosing the Right Surgery

Once the decision has been made to move forward with a surgical intervention, the next step is deciding which speech surgery to recommend. Speech surgeries for cleft-related or non-cleft VPD include palatal procedures, pharyngoplasty, and pharyngeal wall augmentation. Classically, a child's closure type and gap size have been most impactful in determining surgical choice. Ultimately, the choice of speech surgery should be individually tailored to each child based on their specific needs and weighing the risk/benefit profile for their specific surgeries. This decision is best made in a multidisciplinary setting.

Velopharyngeal Dysfunction Surgical Outcomes

Several studies have been published assessing the outcomes of the various surgical procedures for the management of VPD. A systematic review was completed looking at outcomes after surgery for velopharyngeal dysfunction, which included 18 studies and 1,060 children.[14] The study found that for all children who had undergone surgery for VPD, 70.7% attained normal resonance and 65.3% attained normal nasal air emissions.[14]

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

Notably, regardless of procedure type (pharyngeal flap, sphincter pharyngoplasty, palatoplasty, and posterior pharyngeal wall augmentation), there was no difference in speech outcomes, need for revision surgery, or occurrence of obstructive sleep apnea (OSA). The study concluded that there is a lack of consensus in the literature to guide procedure selection, while highlighting the need for more uniform outcome reporting measures. A randomized trial looked at 97 patients ages 3-25 years with prior repaired cleft palate and persistent VPI for which they had undergone either pharyngeal flap or sphincter pharyngoplasty.[15] Their postoperative speech outcomes were reviewed by providers blinded to which procedure the patient had undergone. By 12 months post-surgery, no statistically significant differences in outcomes remained between the 2 procedures for resonance, nasalance, endoscopic outcomes, or surgical complications. Additionally, no difference was found between the 2 procedures in long-term incidence of OSA.

There is a validated instrument that allows providers to track VPI outcomes from a quality of life (QOL) standpoint. The VPI Effects on Life Outcomes (VELO) instrument was created to quantify QOL in VPD patients both before and after VPD surgery.[16] The VELO instrument continues to be a popular tool in measuring outcomes after VPD surgery.[17,18]

From a complications standpoint, Chen and colleagues (2020) used the American College of Surgeons National Surgical Quality Improvement Program-Pediatric (NSQIP-PEDS), where a total of 767 VPI cases were evaluated (191 palatal surgeries, 444 pharyngeal flap, and 132 sphincter pharyngoplasty). The evaluation showed no statistical difference in 30-day complications among any of the procedures.[19]

In summary, no one surgical procedure for VPD has been found to be significantly more effective than the others. While studies overall have shown a majority of the surgical options to be highly successful, studies have been limited by a lack of standardized speech and VPD outcome measures. Standardized measures of outcomes are necessary to allow accurate preoperative and postoperative comparisons, as well as comparisons among institutions and multi-institutional comparisons. The VELO instrument is a useful tool to quantify the "success" of the surgery from the parent/patient perspective and can be helpful for providers managing patients with VPD. Lastly, as each speech surgery impacts airflow dynamics to a certain degree, it is important to be screening and monitoring for OSA postoperatively (**Box 1**).

Box 1
Surgeries for the treatment of VPD include

1. Posterior pharyngeal flap
2. Sphincter pharyngoplasty
3. Buccal myomucosal flap
4. Furlow palatoplasty
5. Palate re-repair
6. Intravelar Veloplasty
7. Injection pharyngoplasty

Surgical positioning

For each of the following procedures, patients are positioned supine with a shoulder roll with the neck extended as tolerated. An oral ray endotracheal tube Dingman mouth retractor help facilitate maximal oropharyngeal exposure.

Posterior pharyngeal flap The posterior pharyngeal flap (PPF) is one of the oldest and most popular techniques of correction of VPD. The PPF uses tissue from the posterior pharyngeal wall to occlude the central nasopharyngeal area. Classically, this procedure is best used for children with sagittal or circular closure patterns, central gaps noted on endoscopy, and good lateral wall motion. PPF has also historically been the workhorse for children with little to no palatal movement (neurogenic soft palate) and subsequent large gaps as this is the only speech surgery that doesn't rely on palatal movement/elevation to be successful.

A staged adenotonsillectomy (AT) may be considered prior to the PPF should there be concerns that enlarged tonsil/adenoid tissue would interfere with ideal flap harvest or inset and/or if there are concerns preoperatively regarding airway obstruction. Due to the risk for worsening or creating obstructive sleep apnea following pharyngeal flap, if a patient has baseline symptoms consistent with airway obstruction at night (ie, snoring, apneas), staged AT should be strongly considered. Additionally, for patients who would require a very wide pharyngeal flap, AT may be completed regardless of preoperative symptoms to help mitigate the postoperative risk of OSA.

Procedure details

The PPF is a superiorly based myomucosal flap from the posterior pharyngeal wall which includes oral mucosa and underlying superior constrictor musculature, down to the prevertebral fascia (**Fig. 4**). Prior to flap elevation it is important that the surgeon be vigilant about watching for and

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

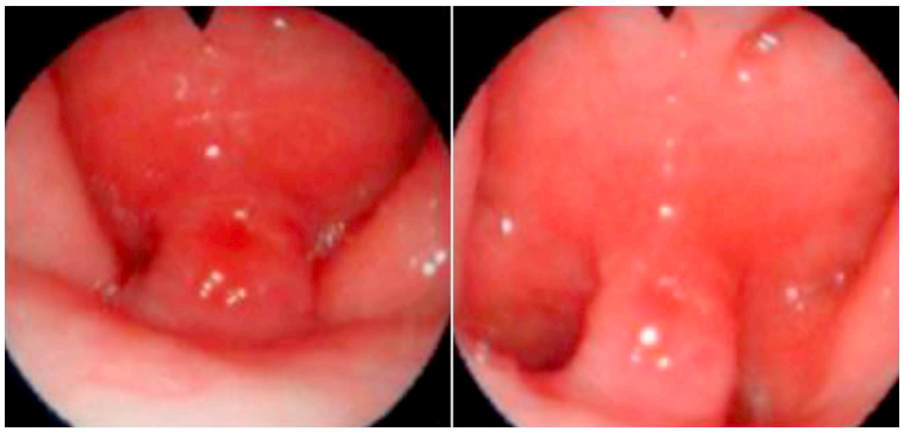

Fig. 4. Photo: pharyngeal flap. With active speech (*left*), lateral walls close around the flap to close off the velopharynx. At rest (*right*) the lateral ports are open. Original.

feeling for pulsations along the posterior pharyngeal wall that could suggest medialized carotid arteries, particularly for patients with 22q11.2 deletion syndrome. The flap length is designed to be slightly longer than the distance from the edge of the soft palate to the posterior pharyngeal wall; this slightly longer length ensures that the flap is long enough to reach the posterior velum without excessive tension or redundancy. The width of the flap is tailored to the child's needs based on the gap width and lateral pharyngeal wall motion on VP imaging. Some overcorrection is incorporated into the design of the flap width to take into account some shrinkage/contracture that will have occurred in the unlined flap during the healing phase. Alternatively, the flap's underside can be lined with nasal surface soft palate mucosa to reduce shrinkage. Using the child's VP exam (ie, nasopharyngoscopy) as guide, the flap is elevated superiorly into the nasopharynx to the match level of VP closure on the exam. The flap is then inset into the posterior margin of the soft palate. Multiple techniques are described for incorporating the flap into the soft palate, including those described by Argamaso[20,21] and Hogan.[22] Lateral ports need to be maintained to prevent nasopharyngeal stenosis and hyponasality. Some providers close the pharyngeal wall defect, while others let it heal by secondary intention.

Posterior pharyngeal flap outcomes

Among larger published series, the rates of objective improvement in resonance following PPF have ranged from 75 to 95%.[23] Concerns over potential postoperative complications with PPF are debated throughout the literature. A study by de Blacam and colleagues[23] (2022) reviewed 109 children managed with PPF with simultaneous dissection and repositioning of the velar muscles. Twelve-month follow-up showed 79.3% statistically significant improvement in hypernasality and an overall 30-day postoperative complication rate of 3.6%. Similarly, Swanson and colleagues[24] used the NSQIP-PEDS database and found a 5.3% rate of 30-day complications in 225 children undergoing pharyngeal flap pharyngoplasty.

Postoperative OSA has been a long-standing concern following VPI surgery given the obstructive nature of the procedure. Recent studies have shown this to be less of a concern than historically reported. Lee and colleagues[25] reviewed 40 children with 22q11.2DS undergoing PPF for VPD and evaluated pre- and post-procedure polysomnography results. This study showed mean OSA did not change significantly after PPF surgery and the prevalence of clinically significant OSA was identical pre-and post-operatively. A study by de Blacam (2022) and colleagues[23] reported that 7/109 patients developed OSA confirmed by sleep study following PFF. This study concluded that surgeons should not be dissuaded by historical concerns about high rates of perioperative complications and OSA and should keep PPF in their toolbox when managing children with VPD.

Of note, if postoperative OSA does occur, the PPF is reversible by dividing the flap at its base. Interestingly, in de Blacam and colleagues's study,[23] of the 4 children requiring PPF takedown, only 1 went on to need additional VPD surgery. This outcome is consistent with the findings of Katzel and colleagues,[26] who examined 64 children who underwent takedown of pharyngeal flap and found that 90% showed no clinically significant regression of their VPD. While the risk of developing postoperative OSA following PPF continues to be debated in the literature, it is the author's opinion that all children undergoing PPF

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

should have a postoperative sleep study at some point following this procedure to evaluate for OSA.

Sphincter pharyngoplasty Sphincter pharyngoplasty (SPP) uses two superiorly based myomucosal flaps and moves this tissue to the posterior pharyngeal wall, which, in turn, creates a "speed bump" at the level of pharyngeal closure, thus shortening the distance the soft palate needs to travel to close off the velopharynx. Historically, SPP is the most ideal for coronal closure patterns noted on endoscopy and is less reliant on lateral wall motion as compared to the pharyngeal flap. Many modifications to the SPP have been described since Hynes[27] introduced the concept of SPP in 1950, including those of Orticochea[28] and Jackson.[29] The technique described later in discussion is a modification from Sie and colleagues[30] that does not include posterior tonsillar pillars in the SPP to minimize the risk of postoperative hyponasality and airway obstruction. The position of the SPP is classically just below the level of the adenoid pad. However, if needed, adenoidectomy can be performed as a staged procedure prior to SPP to prevent having to sew the SPP flaps into the adenoid pad. Additionally, staged tonsillectomy is sometimes required if the tonsils are so enlarged that they impede the elevation of the myomucosal flaps. Of note, there have been claims that the sphincter itself may have some dynamic function due to its incorporation of muscle aiding in the VP closure; however, these claims have been largely unproven.

Procedure details

The uvula is retracted into the nasopharynx to optimize the visualization of the posterior pharyngeal wall (**Fig. 5**). The posterior tonsillar pillars are retracted laterally and anteriorly by silk suture secured to the Dingman. The posterior pillar is continuous with the inferior margin of the soft palate; it is essential to avoid including the posterior pillar into the myomucosal flaps to prevent the inferior margin of the soft palate from being included in the sphincter which can in turn make the velopharyngeal port too narrow and cause obstruction. The recipient site mucosa is incised transversely across the posterior pharyngeal wall to create a rectangularly shaped recipient site. It is important to avoid cutting through the underlying superior constrictor muscle and fascia to maintain the superior positioning of the SPP and avoid inferior migration. The recipient site is designed to be at the level of anticipated velopharyngeal closure. Two superiorly based myomucosal flaps are designed on each side of the posterior pharyngeal wall. These donor flaps will include the palatopharyngeus and the underlying constrictor muscle, with a depth superficial to the alar fascia. The length of the flaps is determined by the approximate width of the recipient site. The flaps are then transposed into a horizontal direction; the tips of the superiorly based donor flaps are secured to the contralateral lateral edges of the recipient site, thus overlapping the flaps. The superior flap is secured to the superior edge of the recipient site and the inferior flap is secured to the inferior edge of the recipient site. The 2 flaps are also secured to one another at their point of approximation. The donor sites are then closed.

Sphincter pharyngoplasty outcomes

A meta-analysis of patients with persistent VPD following cleft repair was performed by Grover and colleagues[31] and included 44 publications and 2,402 patients. The overall SPP success rate was 78.4%. The primary revision rate was 17.8%, most frequently due to persistent VPI and less frequently second to severe obstruction. After one revision, the success rate after SPP increased to 94.7%. Additional modifications to the SPP have been described including a combined Furlow palatoplasty and SPP.[32] In a study by Bohm and colleagues,[32] pediatric patients undergoing surgery for VPD were reviewed: 38 PPF, 20 SPP, and 38 undergoing a combined SPP and Furlow palatoplasty. The mean speech improvement was significantly greater in both the PPF and combined procedure group compared to the SPP alone group.

Buccal myomucosal flaps The buccal flap has historically had several uses in craniofacial surgery, including closure of palatal fistulas, and primary and secondary cleft palate repair in the wide cleft palate,[33] but it has recently been popularized for the management of VPD. The buccal flap procedure is a palatal lengthening procedure that introduces healthy tissue into the velum at the hard-soft palate junction and adds substantial length to the velum. This surgery is ideal for the management of VPD in children who have small-moderate gaps on endoscopy; adequate palatal movement or elevation is required for this surgery to be effective. Typically, this surgery does not rely on lateral wall motion to be successful. Benefits of the buccal flap include minimal donor-site morbidity and easily harvested flaps. Compared to pharyngoplasty, the buccal flap has the most "natural" change to the child's anatomy by lengthening the palate rather than adding tissue to pharyngeal wall/nasopharynx as well as a lower risk of postoperative OSA. The downside of the

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

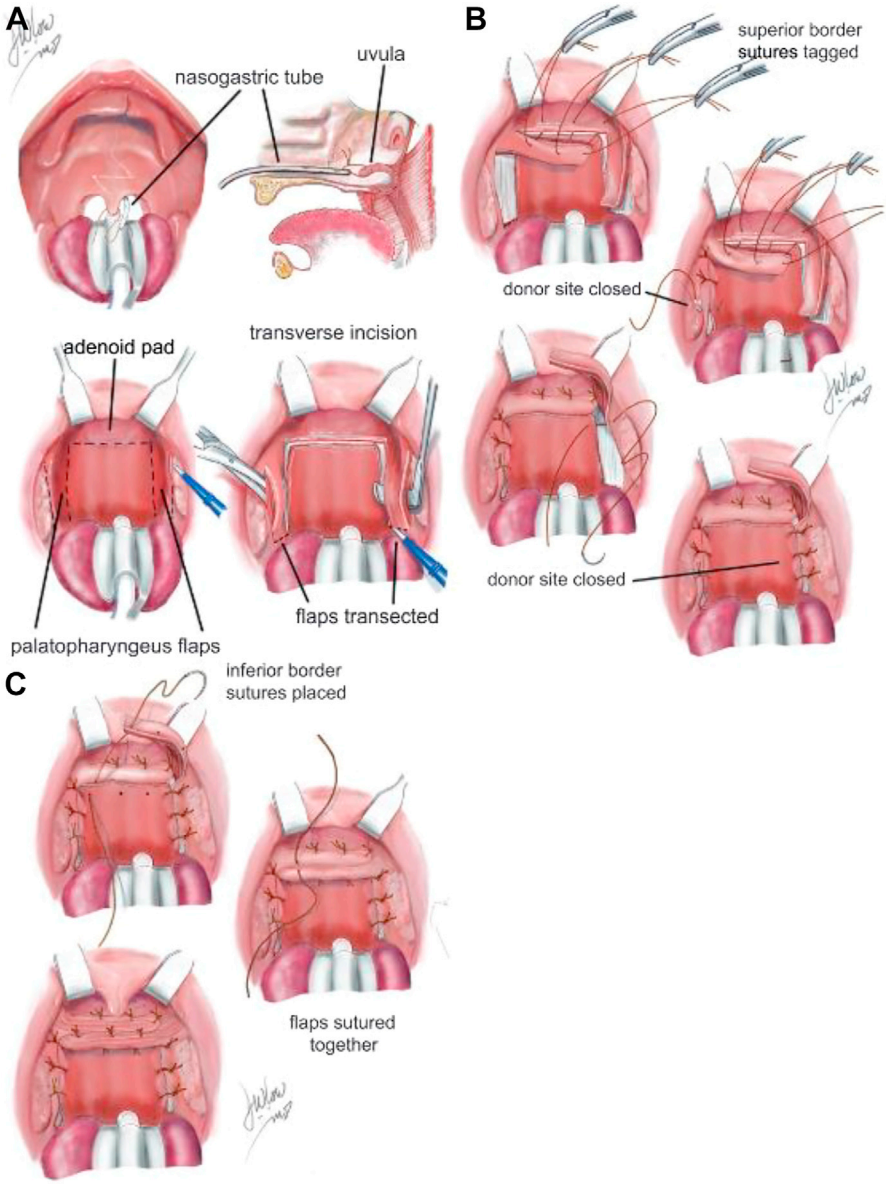

Fig. 5. (*A–C*) Illustration sphincter pharyngoplasty: (*A*) retraction of uvula and bilateral myomucosal sphincter flap design, incision of flaps and blunt undermining, (*B, C*) suturing on flaps and interrupted closure of donor site. (Jackson, O.A., Mehendale, F.V. (2021). Speech Surgery and Treatment of Velopharyngeal Insufficiency. In: Swanson, J.W. (eds) Global Cleft Care in Low-Resource Settings. Springer, Cham. https://doi.org/10.1007/978-3-030-59105-2_20.)

buccal flap is that it requires palatal movement to be effective; therefore, this procedure alone would not be the treatment of choice for a neurogenic soft palate (such as in 22q11.2DS) with minimal or no palatal elevation. Another downside is that it needs a second surgical procedure to divide the flap pedicles, although there is debate about the necessity to perform it.[4,34] Indications for the takedown of the flap pedicles include food getting trapped behind the pedicles or the child chewing/biting on the pedicles.

Procedure details

From a technique standpoint, the technique is similar to the buccinator sandwich pushback method[35] and more recently modified by RJ Mann (**Fig. 6**).[33] The initial curvilinear incision is made approximately 2.5 mm posterior to the hard palate/soft palate junction. Any inappropriately attached muscle fibers are released from the posterior hard palate. Once the thru-and-thru incision is made releasing the soft palate from the hard palate, the soft palate moves posteriorly.

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

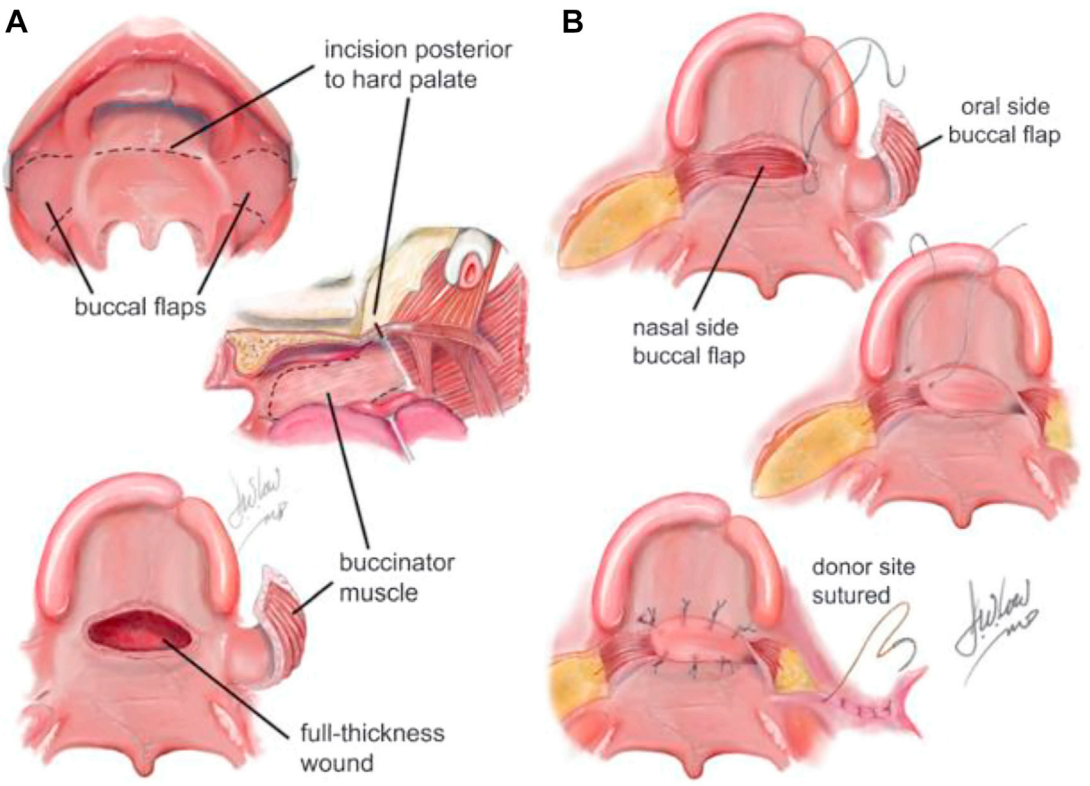

Fig. 6. (*A, B*) Illustration of palatal lengthening technique with bilateral buccal myomucosal flaps. (*A*) Buccal flap incision design and palatal incision near the hard-soft palate junction which is deepened full-thickness through the velum. (*B*) Inset of the buccal flaps in an overlapping fashion and closure of the buccal donor site with a running dissolvable suture. (Jackson, O.A., Mehendale, F.V. (2021). Speech Surgery and Treatment of Velopharyngeal Insufficiency. In: Swanson, J.W. (eds) Global Cleft Care in Low-Resource Settings. Springer, Cham. https://doi.org/10.1007/978-3-030-59105-2_20.)

Next, bilateral buccal flaps are designed with the flap tip at the oral commissure and its base at the retromolar trigone (RMT). The superior portion of the flap is defined by a transverse incision starting at the oral commissure, passing just below Stensen's duct and stopping at the RMT. The inferior aspect of the incision again starts at the oral commissure, then swings down inferiorly to include as much of the buccinator muscle as possible and is extended to the RMT. The base width of the flap is 17-18 mm.[33] When elevating the buccal flaps, only the buccal mucosa and the buccinator muscle are included, leaving buccal fat and the facial artery undisturbed. Unlike the axial pattern of a facial artery musculomucosal flap, this flap is designed randomly. One flap is interposed into the defect with the mucosal surface on the nasal side of the palate. The other flap gets rotated 180 degrees at its base and laid across the defect with the mucosal lining on the oral side of the palate (**Fig. 7**). The donor site is then closed, except for the area near the base of the flap to avoid tension on the pedicles. In older children who have their molars present, a splint may need to be considered to avoid biting and injuring the pedicle.

Buccal myomucosal flap outcomes

Mann and colleagues[33] treated 27 children using double-opposing buccal flap procedure for palatal lengthening in persons with VPD. The level of intelligibility and resonance improved significantly postoperatively. They reported 2 children with distal flap necrosis and 1 with superficial dehiscence of the buccal flap. No donor site complications were reported. There were no reports of postoperative OSA. One child went on to require pharyngeal flap due to persistent VPD. Chauhan and colleagues[4] reviewed 50 children undergoing the double-opposing buccal flap procedure for palatal lengthening in persons with VPD. The palatal length was increased between 10 and 19 mm for all children. They found significant improvement in hypernasality and intelligibility in all age groups. No children showed hyponasality. Mild complications occurred more in the teenage

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

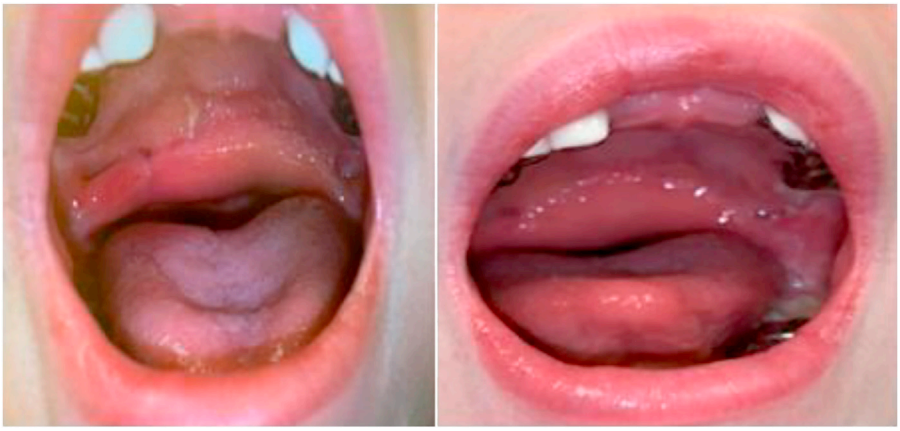

Fig. 7. Buccal myomucosal flap for VPD: 4 weeks postoperative (*left*); 3 months postoperative (*right*). Original.

and adult groups and included difficult mastication, tubing of the flap, and marginal necrosis of the flap.

Denadai and colleagues[34] reported 37 children with preoperative moderate-severe hypernasality who underwent the double-opposing buccal flap procedure. Results showed that hypernasality was significantly lower than at preoperative assessment and that children improved regardless of velopharyngeal gap size/pattern. The authors highlighted the improvement in speech results over time with children's 12-month postoperative hypernasality scores markedly improved from even their 3-month postoperative scores. They emphasized that a period of at least 1 year should be considered ideal for final speech evaluation.

Furlow palatoplasty (double opposing Z-plasty) In 1986, Leonard Furlow described a double opposing Z-plasty technique that reoriented the muscle fibers of the levator sling, lengthened the soft palate, and created a non-linear scar (limiting future shortening of the soft palate secondary to scar contracture).[36] This procedure re-establishes healthy soft palate anatomy and, in many cases, restores normal function.[37] Classically, Furlow palatoplasty has been used to repair submucous cleft palate (SMCP).

Classic SMCPs are characterized by a triad of bifid uvula, notching of the posterior order of the hard palate, and a deficiency of muscle in the midline of the soft palate (zona pellucida). SMCP has a reported incidence of approximately 1:1,200.[38] Anatomically in patients with SMCP, the levator veli palatini musculature is inappropriately attached to the posterior edge of the hard palate, creating a sagittal orientation rather than forming a complete muscular sling in the normal transverse orientation. This muscle malorientation limits the velum's ability to fully contact the pharyngeal wall and results in VPI. Of note, 5-8% of children with isolated SMCP have 22q11.2DS. Providers should strongly consider genetic testing for children who present with isolated SMCP.[3]

Procedure details

Furlow palatoplasty is performed by first designing the oral layer Z-plasty (**Fig. 8**). A posteriorly based myomucosal flap is elevated on the left and an anteriorly based oral mucosal flap is elevated on the right. The second (nasal layer) Z-plasty is designed to again allow for the elevation of a right-sided posteriorly based myomucosal flap and a left-sided anteriorly based nasal mucosal flap. The nasal layer flaps are rotated in Z-plasty fashion and secured into position followed by the oral layer flaps. This results in positioning the levator musculature into its proper transverse location, recreating the levator sling. Many modifications have been made to the classic Furlow palatoplasty.

Furlow palatoplasty outcomes

Sommerlad and colleagues[39] (2004) prospectively followed 40 children with SMCP repaired by Furlow palatoplasty. Their results showed a highly significant improvement in hypernasality, nasal air emissions, and velopharyngeal closure. Brooker and colleagues[40] reviewed 351 children undergoing Furlow for SMCP. They reported an 82% success rate, with the only complication being the need for a secondary speech surgery. Sie and colleagues[30] reported 48 children undergoing Furlow palatoplasty for SMCP and VPI. They concluded that most children had complete resolution or substantial improvement of VPI postoperatively. The main complication in this cohort was palatal fistula, which occurred in 2 cases.

Palate re-repair As discussed above, the Furlow, as well as its subsequent modifications, has been

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

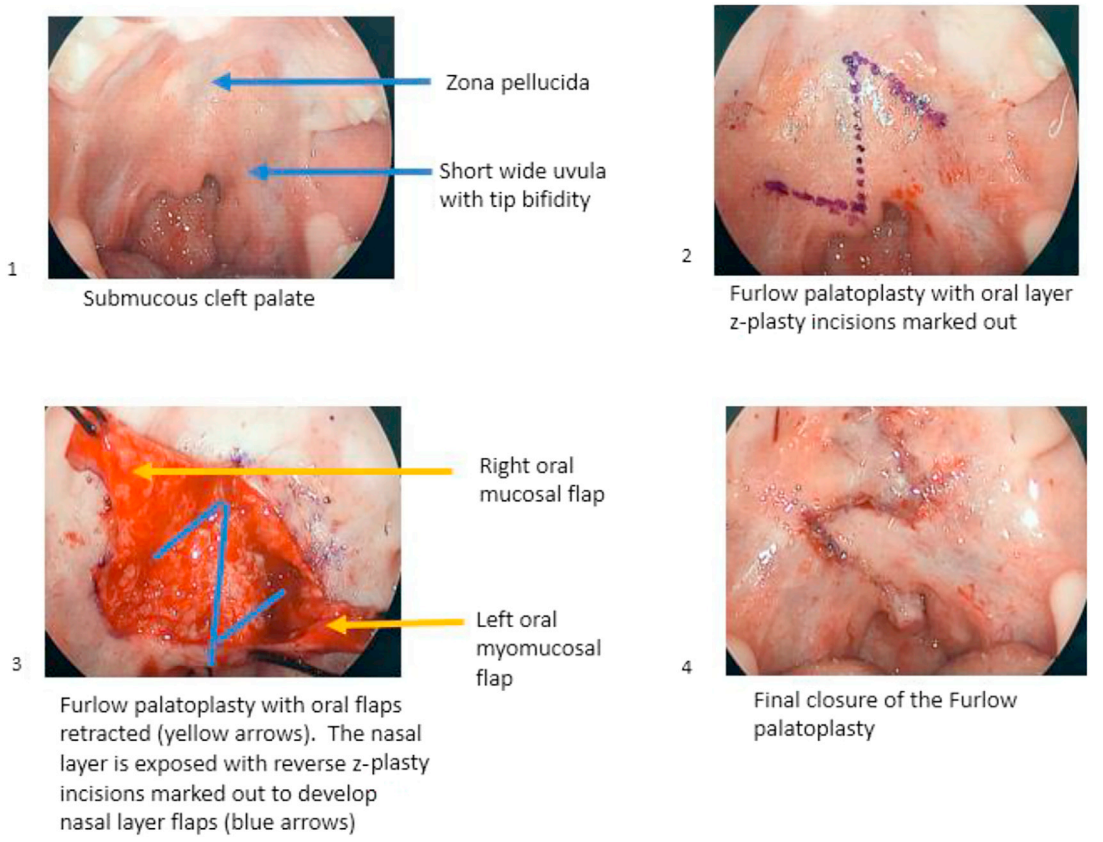

Fig. 8. Furlow palatoplasty. Original.

a mainstay of cleft repair and, from a VPD perspective, the treatment of choice for most children with a SMCP. For persons who have had prior Furlow palatoplasty but continue to have persistent VPD, another option for surgical treatment is performing a "palate re-repair"[41] or a secondary Furlow palatoplasty. The palate re-repair[10] aims to reposition and reconstruct the velar muscles and can be performed using a straight-line incision in the oral mucosa of the palate[42] or by using a double opposing Z-plasty, as discussed in Chen and colleagues (1994)[43] and Randall and colleagues[44]

This procedure has been considered ideal for children with small VP gaps,[43] allowing for acceptable speech outcomes while incurring less surgical morbidity,[45] namely a lower risk of OSA.[10,46] It is also considered to be more physiologic than pharyngoplasty. Sommerlad and colleagues[42] (2002) showed re-repair to be up to 82% effective at treating VPI. However, despite the apparent benefits, the palate re-repair has not been a widely adopted procedure.[10] Critics have a theoretical concern that the secondary Furlow would disorganize the normal orientation of the levator sling which had been previously established during the primary Furlow palatoplasty.[45] The secondary Furlow does re-orient the levator muscle fibers in a more sagittal direction, and concerns exist that this misalignment, distortion, and disorganization of the velar musculature would lead to poor outcomes. Despite these concerns, studies have reported somewhat favorable outcomes following this procedure[45,47,48] perhaps due to further palatal lengthening from the geometry of the Z-plasty.

Procedure details

The same technique as the Furlow palatoplasty but on a palate that has already undergone a prior Furlow palatoplasty.

Palate re-repair outcomes

Hsu and colleagues[45] looked at 13 children with prior Furlow and marginal VPI who underwent a palate re-repair with redo-double opposing Z-plasty. This study discussed at length the concerns about the potential disruption of the levator musculature. However, the authors found that all children improved, leading them to hypothesize that the velar-lengthening effect outweighs the possible problems of muscle disorientation. Rudnicki and

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

colleagues[37] looked at palate motion on nasopharyngoscopy following primary and secondary Furlow palatoplasty procedures. This study did not show significant differences between the two procedures and suggested that there are no major deleterious effects on palate motion following secondary palatoplasty. Kurnik and colleagues[10] performed a systematic review, including 18 studies, looking specifically at palate re-repair in children with VPI following primary cleft palate repair. The overall incidence of achieving no consistent hypernasality following palate re-repair was 61%, and the incidence of no consistent nasal air emission was 78%. The incidence of additional speech surgery for persistent VPI symptoms was 21%. The incidence of OSA following re-repair was 28%.

Intravelar veloplasty Intravelar veloplasty (IVV) involves the restoration of the muscular sling within the soft palate.[49] This is accomplished by freeing the abnormal muscle attachment from the posterior border of the hard palate and repositioning the levator sling into its proper orientation.[50,51] While the muscle repositioning is similar to the Furlow palatoplasty, this procedure can be done using a single vertical incision on the oral surface of the soft palate; by avoiding the interruption of the nasal mucosal layer, benefits of this procedure may reduce the risk of oronasal fistula.[52] IVV is an option for the surgical management of VPD in patients with a SMCP[52,53] or as a secondary cleft repair where inappropriate alignment of the levator musculature is suspected.[36,37]

The term "intravelar velopalsty" was first coined by Kriens in 1969,[54] the concept of a radical IVV was reported by Cutting and colleagues,[55] and the procedure further modified by Sommerlad.[56] The addition of an oral Z-plasty to the radical IVV has also been described.[57] There is wide variability in the extent of dissection and repositioning of the muscle done during IVV; a classification system of IVV was proposed by Andrades and colleagues[58] (**Table 1**).

Table 1
Classification of IVV[50,58]

Type	Magnitude of Levator Muscular Dissection
0	No muscle dissection or suturing of muscle
1	No muscular dissection, parallel suturing of the muscle
2a	Partial dissection: release of muscle from the hard palate but with minimal dissection from nasal and oral mucosa
2b	Partial dissection: release of muscle from the hard palate and dissection from the nasal mucosa but not oral mucosa
3 (radical IVV)	Complete dissection: release of the muscle from the hard palate and dissection from both nasal and oral mucosa creating a transverse muscle sling

Procedure details

Classically done using a single midline vertical incision from the junction of the hard-soft palate to the base of the uvula. The levator veli palatine musculature is released from the posterior edge of the hard palate and separated from the surrounding muscular attachments. The levator muscles are dissected, and both ends of the muscle are anchored to the body of the opposite muscle, thus recreating the levator sling.

Intravelar veloplasty outcomes

Woo and colleagues[57] compared Furlow palatoplasty to a single oral Z-plasty with overlapping IVV (Woo palatoplasty) for the correction of VPI in cleft patients. Thirty patients underwent Furlow and 22 Woo palatoplasty. Results showed a larger portion of the Woo palatoplasty (95%) did not require a secondary speech surgery compared to the Furlow palatoplasty (63%) and they concluded this technique is a viable alternative for the management of persistent VPI in cleft patients.

Barbosa and colleagues[59] reviewed 78 patients with history of cleft palate repair and persistent VPI, 40 underwent pharyngeal flap and 38 IVV. Results showed absence of hypernasality occurred in 70% of the pharyngeal flap group and only 34% of the IVV group and this study concluded pharyngeal flap was more effective than IVV to reduce hypernasality.

IVV has also been completed in combination with other VPD surgeries.[60,61] Ezzat and colleagues[60] describes 15 with VPI following repaired cleft palate who underwent the combination of posterior pharyngeal flap and IVV; where an overall success rate of VP competence of 93.4% was achieved. Nam and colleagues[61] reports 15 patients undergoing a sphincter pharyngoplasty combined with IVV with 100% achieving satisfactory results and no patients required additional speech surgery.

Injection pharyngoplasty (posterior pharyngeal wall augmentation) Another surgical technique for the management of VPD is injection pharyngoplasty, whereby materials are injected into the posterior velopharynx to add bulk and reduce the

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

velopharyngeal gap. Many materials[62,63] have been used, including autologous fat, cartilage, fascia, paraffin, silicone, acellular dermis, exogenous fillers such as hyaluronic acid and calcium hydroxyapatite, and dextranomer/hyaluronic acid (Dx/HA). Studies have shown promising improvements in VPD outcomes with less morbidity than with classic surgical repairs.[62,63] Shortcomings of injectable materials include migration, foreign-body granulomatous reactions, resorption, and donor site morbidity (for fat). While becoming a popular choice for injection pharyngoplasty, Dx/HA is approved by the Food and Drug Administration for human use in urologic procedures and the treatment of anal incompetence; it is considered an off-label use in the pharynx.

Procedure details

For adults, the injection can be done in-office with the person awake; however, children typically require some degree of anesthesia[62] for the procedure. The injection is completed trans orally, and the material is placed into the specific location of the velopharyngeal gap as identified on preoperative endoscopy. The amount of the injected material is typically tailored to the patient's needs based on endoscopy.

Injection pharyngoplasty outcomes

Peck and colleagues[62] reported 25 children undergoing injection pharyngoplasty with Dx/HA. The amount of Dx/HA used depended on the gap size: small gaps required a mean of 2.5 mL compared to moderate-large gaps averaging 4.1 mL. For this cohort, 76% showed improvement in their perceptual nasal resonance. Complications reported included 2 children with retropharyngeal fluid collections requiring transoral incision and drainage. Six children required repeated injections. This study suggested Dx/HA injections were most effective in children with small to moderate sized VP gaps and those with a better degree of lateral pharyngeal wall motion.

Brigger and colleagues[63] reviewed 12 children who received calcium hydroxyapatite injection pharyngoplasty. Injection amount ranged from 1 to 4 mL. Eight children demonstrated success after 3 months; of these, 4 had long-term follow-up at 24 months and were found to have sustained stable outcomes. All 4 failures occurred in children with associated craniofacial anomalies, and no complications were reported. This study reported that injection pharyngoplasty was most successful for children with mild VPD and small central VP gap; the authors specifically highlighted a child who has undergone adenoidectomy with a resultant small central gap as an ideal candidate.

SPECIAL CONSIDERATIONS: 22Q11.2 DELETION SYNDROME

22q11.2DS is the most common cause of syndromic palatal anomalies.[3] VPD and SMCP (both overt and occult) are seen in 67% of children with 22q11.2DS.[3,64] Differences in the velopharyngeal structure and function specific to children with 22q11.2DS place them at higher risk for developing VPD and include hypoplasia and hypotonia of the velopharyngeal musculature, a wide and/or deep pharynx, platybasia (obtuse anterior cranial base angle), cervical spine abnormalities, reduced adenoid volume, asymmetric muscle function, and cranial nerve abnormalities.[3,65,66] Additionally, the timing of the velopharyngeal closure may be slower or poorly coordinated.[67] For these reasons, surgical management of VPD in children with 22q11.2DS can be quite challenging, and reports have shown these children have poorer outcomes and a higher need for secondary speech surgery. A study by Sommerlad and colleagues[39] (2004) found poorer speech outcomes after SMCP repair for larger VP gaps (>13 mm) and for those with 22q11.2DS. With these outcomes in mind, some surgeons will repair the SMCP in children with 22q11.2DS with Furlow palatoplasty to best optimize palatal function; accepting the need for secondary pharyngoplasty is common. Others, though, will move directly to pharyngoplasty.[3]

Medialized and/or aberrant vasculature is common and should be evaluated during video endoscopy. Some providers obtain imaging (computerized tomography angiogram or magnetic resonance angiography) prior to performing surgical procedures that involve the pharynx, including SPP and PPF. Whether imaging is obtained or not, it is imperative that the surgeon be aware of these risks and frequently palpate/observe for pulsations during SPP or PPF in children with 22q11.2DS.[68]

With increased risk for OSA at baseline for children with 22q11.2DS, it is important to screen children for obstructive breathing following VPD surgery, and to maintain a low threshold for performing polysomnography.[68] Finally, postoperative hypocalcemia can occur in children with 22q11.2DS. The updated guidelines for the management of children with 22q11.2DS recommend monitoring serum calcium perioperatively.[68]

NONSURGICAL OPTIONS FOR THE MANAGEMENT OF VELOPHARYNGEAL DYSFUNCTION

While outside of the scope of the current discussion, it is worth noting that some prosthetic devices

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

can be designed and worn by the patient to help facilitate the closure of the velopharynx. As these are custom-made, they do require frequent adjustments as children grow, and they can be difficult for some children to tolerate. While this option exists, it has traditionally been used when surgical treatment is not an option.[69]

SUMMARY

A multidisciplinary team is essential for the diagnosis and management of children with VPD. Multiple surgical options exist to treat VPD. Currently, there is no consensus as to the preferred or best surgical option. Patient factors, endoscopy findings, procedure risks and benefits, and surgeon preference and comfort level with the procedure should all be considered when deciding on which surgery to recommend. Standardized speech and VPD outcome measures should continue to be developed to allow for more pre- and postoperative comparisons.

CLINICS CARE POINTS

- Velopharyngeal dysfunction (VPD) is an overarching term that describes any situation where air is inappropriately escaping through the nose during speech. Velopharyngeal (VP) insufficiency, VP inadequacy, mechanical preventing soft palate closure (ie, enlarged tonsils), and phoneme-specific are the 4 main causes (or types) of VPD.
- Multiple surgeries exist for the treatment of VPD. A large systematic review found that for all children who had undergone surgery for VPD, 70.7% attained normal resonance.
- Special considerations should be made for children with 22q11.2 deletion syndrome undergoing VPD surgery including monitoring for medialized and/or aberrant vasculature and postoperative hypocalcemia.

DISCLOSURE

The authors have nothing to disclose.

REFERENCES

1. Woo AS. Velopharyngeal dysfunction. Semin Plast Surg 2012;26(4):170–7.
2. Muntz H.R., Velopharyngeal dysfunction. cummings pediatric otolaryngology, 2015, Saunders, an imprint of Elsevlier, Inc., Philadelphia, PA: 123–133, chap 9.
3. Solot CB, Sell D, Mayne A, et al. Speech-language disorders in 22q11.2 deletion syndrome: best practices for diagnosis and management. Am J Speech Lang Pathol 2019;28(3):984–99.
4. Chauhan JS, Sharma S, Jain D, et al. Palatal lengthening by double opposing buccal flaps for surgical correction of velopharyngeal insufficiency in cleft patients. J Cranio-Maxillo-Fac Surg 2020;48(10):977–84.
5. Smith B, Guyette TW. Evaluation of cleft palate speech. Clin Plast Surg 2004;31(2):251–60, ix.
6. Kummer AW. Speech evaluation for patients with cleft palate. Clin Plast Surg 2014;41(2):241–51.
7. Bhuskute A, Skirko JR, Roth C, et al. Association of velopharyngeal insufficiency with quality of life and patient-reported outcomes after speech surgery. JAMA Facial Plast Surg 2017;19(5):406–12.
8. MacKenzie-Stepner K, Witzel MA, Stringer DA, et al. Velopharyngeal insufficiency due to hypertrophic tonsils. A report of two cases. Int J Pediatr Otorhinolaryngol 1987;14(1):57–63.
9. Abdel-Aziz M, El-Fouly M, Nassar A, et al. The effect of hypertrophied tonsils on the velopharyngeal function in children with normal palate. Int J Pediatr Otorhinolaryngol 2019;119:59–62.
10. Kurnik NM, Weidler EM, Lien KM, et al. The effectiveness of palate re-repair for treating velopharyngeal insufficiency: a systematic review and meta-analysis. Cleft Palate Craniofac J 2020;57(7):860–71.
11. Saunders NC, Hartley BE, Sell D, et al. Velopharyngeal insufficiency following adenoidectomy. Clin Otolaryngol Allied Sci 2004;29(6):686–8.
12. Lipira AB, Grames LM, Molter D, et al. Videofluoroscopic and nasendoscopic correlates of speech in velopharyngeal dysfunction. Cleft Palate Craniofac J 2011;48(5):550–60.
13. Perry JL, Snodgrass TD, Gilbert IR, et al. Establishing a clinical protocol for velopharyngeal MRI and interpreting imaging findings. Cleft Palate Craniofac J 2022. https://doi.org/10.1177/10556656221141188. 10556656221141188.
14. de Blacam C, Smith S, Orr D. Surgery for velopharyngeal dysfunction: a systematic review of interventions and outcomes. Cleft Palate Craniofac J 2018;55(3):405–22.
15. Abyholm F, D'Antonio L, Davidson Ward SL, et al. Pharyngeal flap and sphincterplasty for velopharyngeal insufficiency have equal outcome at 1 year postoperatively: results of a randomized trial. Cleft Palate Craniofac J 2005;42(5):501–11.
16. Skirko JR, Weaver EM, Perkins JA, et al. Validity and responsiveness of VELO: a velopharyngeal insufficiency quality of life measure. Otolaryngol Head Neck Surg 2013;149(2):304–11.
17. Li L, Strum D, Larson S, et al. Quality of life outcomes following velopharyngeal insufficiency surgery. Int J Pediatr Otorhinolaryngol 2019;127:109643.

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

18. Crockett DJ, Goudy SL. Update on surgery for velopharyngeal dysfunction. Curr Opin Otolaryngol Head Neck Surg 2014;22(4):267–75.
19. Chen AD, Kang CO, Tran BNN, et al. Surgical approaches and 30-day complications of velopharyngeal insufficiency repair using american college of surgeons national surgical quality improvement program-pediatric. J Surg Res 2020;250:102–11.
20. Argamaso RV. Pharyngeal flap surgery for velopharyngeal insufficiency. Operat Tech Plast Reconstr Surg 1995;2(4):233–8.
21. Argamaso RV, Shprintzen RJ, Strauch B, et al. The role of lateral pharyngeal wall movement in pharyngeal flap surgery. Plast Reconstr Surg 1980;66(2):214–9.
22. Hogan VM. A clarification of the surgical goals in cleft palate speech and the introduction of the lateral port control (l.p.c.) pharyngeal flap. Cleft Palate J 1973;10:331–45.
23. de Blacam C, O'Dwyer A, Oderoha KE, et al. The case for the pharyngeal flap pharyngoplasty in the management of velopharyngeal dysfunction. J Plast Reconstr Aesthetic Surg 2022;75(9):3436–47.
24. Swanson JW, Johnston JL, Mitchell BT, et al. Perioperative complications in posterior pharyngeal flap surgery: review of the national surgical quality improvement program pediatric (NSQIP-PEDS) database. Cleft Palate Craniofac J 2016;53(5):562–7.
25. Lee A, Chang BL, Solot C, et al. Defining risk of postoperative obstructive sleep apnea in patients with 22q11.2DS undergoing pharyngeal flap surgery for velopharyngeal dysfunction using polysomnographic evaluation. Cleft Palate Craniofac J 2020;57(7):808–18.
26. Katzel EB, Shakir S, Naran S, et al. Speech outcomes after clinically indicated posterior pharyngeal flap takedown. Ann Plast Surg 2016;77(4):420–4.
27. Hynes W. Pharyngoplasty by muscle transplantation. Br J Plast Surg 1950;3(2):128–35.
28. Orticochea M. Construction of a dynamic muscle sphincter in cleft palates. Plast Reconstr Surg 1968;41(4):323–7.
29. Jackson IT, Silverton JS. The sphincter pharyngoplasty as a secondary procedure in cleft palates. Plast Reconstr Surg 1977;59(4):518–24.
30. Sie KC, Tampakopoulou DA, Sorom J, et al. Results with Furlow palatoplasty in management of velopharyngeal insufficiency. Plast Reconstr Surg 2001;108(1):17–25 [discussion: 26-9].
31. Grover R, Barnett A, Rampazzo A, et al. Outcomes of sphincter pharyngoplasty in the cleft population. J Craniofac Surg 2018;29(4):908–13.
32. Bohm LA, Padgitt N, Tibesar RJ, et al. Outcomes of combined Furlow palatoplasty and sphincter pharyngoplasty for velopharyngeal insufficiency. Otolaryngol Head Neck Surg 2014;150(2):216–21.
33. Mann RJ, Neaman KC, Armstrong SD, et al. The double-opposing buccal flap procedure for palatal lengthening. Plast Reconstr Surg 2011;127(6):2413–8.
34. Denadai R, Sabbag A, Amaral CER, et al. Buccinator myomucosal flap for the treatment of velopharyngeal insufficiency in patients with cleft palate and/or lip. Braz J Otorhinolaryngol 2018;84(6):697–707.
35. Hill C, Hayden C, Riaz M, et al. Buccinator sandwich pushback: a new technique for treatment of secondary velopharyngeal incompetence. Cleft Palate Craniofac J 2004;41(3):230–7.
36. Furlow LT Jr. Cleft palate repair by double opposing Z-plasty. Plast Reconstr Surg 1986;78(6):724–38.
37. Rudnicki PA, Tsang C, Vecchiotti MA, et al. Palatal motion after primary and secondary furlow palatoplasty. JAMA Otolaryngol Head Neck Surg 2017;143(2):111–5.
38. Wales CJ, Corsar K, Devlin MF. Submucous cleft palate. Br Dent J 2009;207(6):254.
39. Sommerlad BC, Fenn C, Harland K, et al. Submucous cleft palate: a grading system and review of 40 consecutive submucous cleft palate repairs. Cleft Palate Craniofac J 2004;41(2):114–23.
40. Brooker JE, Bykowski M, Cray JJ, et al. Outcomes of Furlow Double-Opposing Z-Plasty Palatoplasty for the Treatment of Symptomatic Overt and Occult Submucous Cleft Palate: A Comparison Study. Plast Reconstr Surg 2021;147(5):1141–8.
41. Gosain AK, Chim H, Sweeney WM. Double-opposing Z-plasty for secondary surgical management of velopharyngeal insufficiency following primary furlow palatoplasty. Cleft Palate Craniofac J 2018;55(5):706–10.
42. Sommerlad BC, Mehendale FV, Birch MJ, et al. Palate re-repair revisited. Cleft Palate Craniofac J 2002;39(3):295–307.
43. Chen PK, Wu JT, Chen YR, et al. Correction of secondary velopharyngeal insufficiency in cleft palate patients with the Furlow palatoplasty. Plast Reconstr Surg 1994;94(7):933–41 [discussion: 942-3].
44. Randall P, LaRossa D, Solomon M, et al. Experience with the Furlow double-reversing Z-plasty for cleft palate repair. Plast Reconstr Surg 1986;77(4):569–76.
45. Hsu PJ, Wang SH, Yun C, et al. Redo double-opposing Z-plasty is effective for correction of marginal velopharyngeal insufficiency. J Plast Reconstr Aesthetic Surg 2015;68(9):1215–20.
46. Mehendale FV, Lane R, Laverty A, et al. Effect of palate re-repairs and hynes pharyngoplasties on pediatric airways: an analysis of preoperative and postoperative cardiorespiratory sleep studies. Cleft Palate Craniofac J 2013;50(3):257–67.
47. Elsherbiny A, Amerson M, Sconyers L, et al. Outcome of palate re-repair with radical repositioning of the levator muscle sling as a first-line strategy in postpalatoplasty velopharyngeal incompetence

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

management protocol. Plast Reconstr Surg 2018; 141(4):984–91.
48. Araújo B.M.A.M., Da Silva A.S.C., Bertier C.E., et al., Palate re-repair for velopharyngeal insufficiency treatment: a long-term auditory-perceptual assessment of speech. Perspectives of the ASHA Special, Interest Groups, 2022, 1–10, https://doi.org/10.1044/2022_PERSP-22-00079.
49. Marsh JL, Grames LM, Holtman B. Intravelar veloplasty: a prospective study. Cleft Palate J 1989; 26(1):46–50.
50. Jayarajan R, Natarajan A, Nagamuttu R. Intravelar veloplasty: A review. State of the art. Journal of Cleft Lip Palate and Craniofacial Anomalies 2018 2018; 5(2):68–73.
51. Nam SM. Surgical treatment of velopharyngeal insufficiency. Arch Craniofac Surg 2018;19(3): 163–7.
52. Sung KP, Lee SH. Intravelar veloplasty in submucous cleft palate without lateral relaxing incision or nasal mucosal incision. Int J Pediatr Otorhinolaryngol 2023;170:111575.
53. Lee SK, Nam SM, Cha HG, et al. Overlapping intravelar veloplasty improves the speech outcomes in submucous cleft palate. Ann Plast Surg 2023; 90(5):451–5.
54. Kriens OB. An anatomical approach to veloplasty. Plast Reconstr Surg 1969;43(1):29–41.
55. Cutting CB, Rosenbaum J, Rovati L. The technique of muscle repair in the cleft soft palate. Operat Tech Plast Reconstr Surg 1995;2(4):215–22.
56. Sommerlad BC. A technique for cleft palate repair. Plast Reconstr Surg 2003;112(6):1542–8.
57. Woo AS, Skolnick GB, Sachanandani NS, et al. Evaluation of two palate repair techniques for the surgical management of velopharyngeal insufficiency. Plast Reconstr Surg 2014;134(4):588e–96e.
58. Andrades P, Espinosa-de-Los-Monteros A, Shell DHt, et al. The importance of radical intravelar veloplasty during two-flap palatoplasty. Plast Reconstr Surg 2008;122(4):1121–30.
59. Barbosa DA, Scarmagnani RH, Fukushiro AP, et al. Surgical outcome of pharyngeal flap surgery and intravelar veloplasty on the velopharyngeal function. Codas 2013;25(5):451–5.
60. Ezzat AE, Khalifa RA, Akel MM, et al. Functional and speech outcomes of superiorly based flap pharyngoplasty combined with radical intravelar veloplasty. J Cleft Lip Palate Craniofacial Anomalies 2015; 2(1):41–8.
61. Nam SM, Kim YB, Park KC, et al. Speech outcomes of treatment for velopharyngeal insufficiency: combined use of an overlapping intravelar veloplasty and dynamic sphincter pharyngoplasty. Ann Plast Surg 2022;88(5):518–23.
62. Peck BW, Baas BS, Cofer SA. Injection pharyngoplasty with a hyaluronic acid and dextranomer copolymer to treat velopharyngeal insufficiency in adults. Mayo Clin Proc Innov Qual Outcomes 2017; 1(2):176–84.
63. Brigger MT, Ashland JE, Hartnick CJ. Injection pharyngoplasty with calcium hydroxylapatite for velopharyngeal insufficiency: patient selection and technique. Arch Otolaryngol Head Neck Surg 2010;136(7):666–70.
64. Campbell IM, Sheppard SE, Crowley TB, et al. What is new with 22q? An update from the 22q and you center at the children's hospital of Philadelphia. Am J Med Genet 2018;176(10):2058–69.
65. Kirschner RE, Baylis AL. Surgical considerations in 22Q11.2 deletion syndrome. Clin Plast Surg 2014; 41(2):271–82.
66. Dyce O, McDonald-McGinn D, Kirschner RE, et al. Otolaryngologic manifestations of the 22q11.2 deletion syndrome. Arch Otolaryngol Head Neck Surg 2002;128(12):1408–12.
67. Baylis AL, Watson PJ, Moller KT. Structural and functional causes of hypernasality in velocardiofacial syndrome. A pilot study. Folia Phoniatr Logop 2009;61(2):93–6.
68. Oskarsdottir S, Boot E, Crowley TB, et al. Updated clinical practice recommendations for managing children with 22q11.2 deletion syndrome. Genet Med 2023;25(3):100338.
69. Tuna SH, Pekkan G, Gumus HO, et al. Prosthetic rehabilitation of velopharyngeal insufficiency: pharyngeal obturator prostheses with different retention mechanisms. Eur J Dent 2010;4(1):81–7.

Descargado para Biblioteca Medica Hospital México (bibliomexico@gmail.com) en National Library of Health and Social Security de ClinicalKey.es por Elsevier en febrero 12, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.