Treatment option for chronic gag reflex with palate push-back osteotomy: case report

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Background: The pharyngeal reflex (gag reflex) serves as an airway protective mechanism, however, there are patients who suffer with chronic gagging secondary to abnormal anatomical interrelationships in the oropharyngeal structures. Chronic gag reflex can be a considerable disability and is usually treated non-surgically, often with ineffective results. This case report introduces a new surgical procedure to correct chronic gag reflex unresponsive to other treatment methods.

Case Description: A 47-year-old male developed chronic gag reflex immediately following a maxillary advancement surgery and geniohyoid suspension. A short hard palate displaced the soft palate too far forward impacting of the posterior aspect of the tongue triggering the severe gag reflex. A reverse geniohyoid procedure, additional maxillary osteotomes, and surgical alterations of the soft palate failed to eliminate the gag reflex. A new surgical procedure was developed, hard palate pushback osteotomy (HPPBO), and used to eliminate the gag reflex for this patient.

Conclusions: The HPPBO may eliminate the gag reflex for patients meeting the surgical criteria, by repositioning the mid-palatal bone and soft palate posteriorly, disengaging the soft palate from articulating against the posterior aspect of the tongue.

Keywords: Gag reflex; pharyngeal reflex; hard palate pushback osteotomy (HPPBO); short hard palate; case report

Introduction

Background

The pharyngeal reflex or gag reflex is a normal reflexive physiological mechanism associated with stimulation and contraction of the oropharyngeal muscles to minimize foreign material aspiration or choking (1-3). The gag reflex can be elicited by both psychological and physiological factors (4). The physiological factors can be visual, auditory, olfactory, or tactile (2,4). There are five trigger points known to illicit the tactile gag reflex located in the oropharynx (1,2,4). Stimulation of one of these points causes

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a signal to be conducted on the sensory limb mediated predominantly by cranial nerve (CN) IX (glossopharyngeal nerve) to the medulla. From the medulla oblongata efferent impulses are transmitted back through the motor limb by CN X (vagus nerve) causing rapid, uncoordinated brief oropharyngeal musculature contractures of gagging (2). This reflex is a maneuver by the body to block excessive amounts of noxious material from entering the oropharyngeal airway (OPA) that could subsequently lead to airway obstruction, pulmonary aspiration, or injury to the oropharyngeal structures (1-3).

Rationale and knowledge gap

While the pharyngeal reflex (gag reflex) serves as an airway protective mechanism, there are patients who suffer with chronic gag reflex secondary to abnormal anatomical interrelationships in the oropharyngeal structures creating a considerable disability. This type of chronic gag reflex can have an onset when tumors or other pathological processes in the oropharynx cause chronic swelling or enlargement of the soft palate (SP), adenoid tissues, tonsils, tongue, or abnormal anatomical positioning of the SP creating intimate contact with the base of the tongue (1-3).

Numerous treatment modalities have been suggested for hyperactive gag reflex with various successes from pharmacological intervention and behavioral modifications

to acupuncture and hypnosis (4). Unfortunately, these treatment options typically only provide temporary relief and fail to address the abnormal anatomical inter-relationships. While there are surgical options in the literature (5,6), albeit scarce, they have had limited success in resolving a debilitating chronic pharyngeal gag reflex.

Objective

A new surgical technique is introduced, hard palate push-back osteotomy (HPPBO), to address the chronic gag reflex when the SP is anatomically positioned too far anterior with constant articulation against the tongue base (TB) due to an abnormally short length of the hard palate in the presence of a normal OPA and no sleep apnea symptoms. This technique allows lengthening of the hard palate using bilateral parallel parasagittal palatal osteotomies connected by an anterior horizontal cut to posteriorly reposition the mid-portion of the palate. This displaces the SP posteriorly, disengaging it from articulating against the TB. The purpose of this paper is to introduce a surgical technique, HPPBO, that may eliminate chronic gag reflex for patients that fit the surgical criteria. The authors present this article in accordance with the CARE reporting checklist (available at https://fomm.amegroups.com/article/view/10.21037/fomm-22-39/rc).

Case presentation

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013).

History of the present illness

A 47-year-old male presented to the senior author (LM Wolford) with a chronic hyperactive gag reflex. The patient was unable to engage in normal conversation without gagging. The chronic gag reflex started immediately after the patient had his initial orthognathic surgery (04/2009) by another surgeon to advance the maxilla 8 mm and inferiorly reposition the incisors 4 mm to correct a Class III jaw mal-alignment and malocclusion in addition to a genio-hyoid advancement. This initiated his symptomatology, as the forward repositioning of the maxilla pulled the SP forward impacting against the posterior aspect of his tongue. Five months later he had a "reversal" chin procedure

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Highlight box

Key Findings
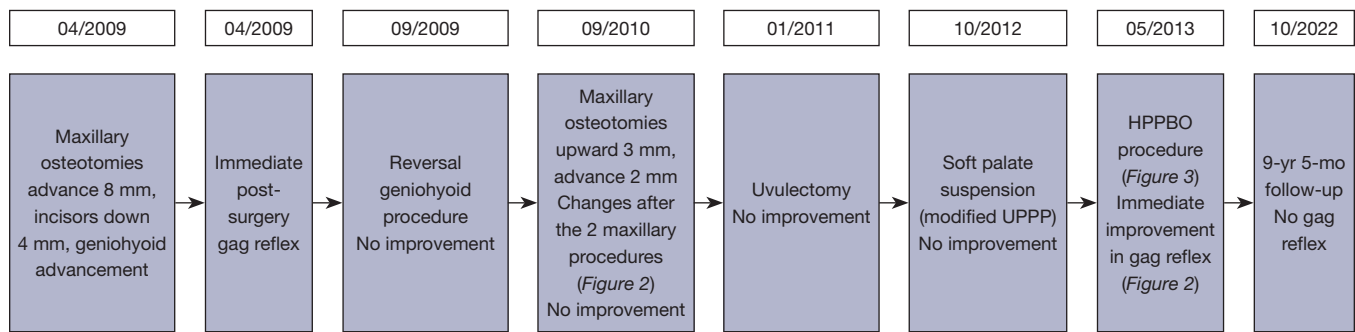- Hard palate pushback osteotomy (HPPBO) may be effective in eliminating chronic gag reflex for patients meeting the surgical criteria.

What is known and what is new?
- Chronic gag reflex is a debilitating condition that may be difficult to eliminate. There are many non-surgical methods of management in the literature with inconsistent effectiveness, but little information for surgical treatment.
- This case report presents a new surgical protocol for treatment of chronic gag reflex that was effective in eliminating the condition in this patient. Patients that meet the surgical criteria may benefit from this surgical approach.

What is the implication, and want should change now?
- Patients with chronic gag reflex that meet the specific surgical criteria may benefit from this treatment protocol. However, larger studies are necessary to validate the efficacy of this surgical treatment in eliminating chronic gag reflex.

04/2009	04/2009	09/2009	09/2010	01/2011	10/2012	05/2013	10/2022
Maxillary osteotomies advance 8 mm, incisors down 4 mm, geniohyoid advancement	Immediate post-surgery gag reflex	Reversal geniohyoid procedure No improvement	Maxillary osteotomies upward 3 mm, advance 2 mm Changes after the 2 maxillary procedures (*Figure 2*) No improvement	Uvulectomy No improvement	Soft palate suspension (modified UPPP) No improvement	HPPBO procedure (*Figure 3*) Immediate improvement in gag reflex (*Figure 2*)	9-yr 5-mo follow-up No gag reflex

Figure 1 This timeline illustrates the surgical interventions the patient encountered that created the gag reflex, and the procedures performed during his pursuit to eliminate the gag reflex (*Figures 2,3*). UPPP, uvulopalatopharyngoplasty; HPPBO, hard palate push-back osteotomy; yr, year; mo, month.

(09/2009) to revise the genio-hyoid advancement, but this had no effect on the chronic gag reflex. One year after the "reversal" chin procedure, another surgeon performed (09/2010) a second maxillary operation for esthetics to reposition it superiorly 3 mm at the incisors and advance it forward 2 mm to maintain the occlusion but did not resolve his chronic gag reflex issue (*Figure 1*, Timeline). The patient was then referred to the senior author with the chief complaint of chronic gag reflex.

Clinical exam

The patient had reasonable facial balance and a Class I occlusion. He had a hyperplastic uvula. He had bilateral temporomandibular joint (TMJ) arthritis with crepitation, but asymptomatic. Incisal opening was 41 mm and excursions were 3 mm to the right and 2 mm to the left. He otherwise was in good health.

Radiographic evaluation

Lateral cephalometric radiographs were used for hard and soft tissue analysis for this patient (*Figure 2*). At initial presentation, radiographic evaluation showed the patient had a Class I skeletal and occlusal relationship with bone plates from his previous maxillary surgery. The SP rested against the posterior aspect of the tongue. Cephalometric analysis was used to determine the dimensions of the hard and SPs for comparison to normal anatomical structures (*Figure 2A*). *Table 1* includes definitions and abbreviations for the anatomical landmarks used for evaluation. *Table 2* presents the normal anteroposterior dimension of the hard palate measuring from anterior nasal spine (ANS) to the

posterior nasal spine (PNS) for males is 61.6±3.0 mm and for females 57.0±4.0 mm and from Point A (subspinale) (PtA—the deepest midline point on the premaxilla) to the PNS for males is 55.6±4 mm and for females 51.8±4.0 mm (7). The normal pharyngeal depth measuring from PNS to posterior pharyngeal wall (PPW) for males is 26.2±5.0 mm and for females is 27.0±3.0 mm. The normal SP length measuring from the PNS to the tip of the uvula (TU) in males is 38.0±2.0 mm and in females 36.2±3.0 mm. The normal SP thickness for males is 9.8±1.0 mm, and for females 9.0±2.0 mm (8). The normal antero-posterior dimension of the OPA measuring from the PPW to the SP and PPW to the posterior TB for males and females is 11±2 mm (9) (*Figure 2A*; *Table 2*).

An initial lateral cephalometric radiograph of this patient was obtained prior to the two maxillary osteotomy procedures (*Figure 2B*). The anatomical measurements of the hard palate length measured 52 mm from ANS to PNS and 51 mm from PtA to PNS (normal 61.6 and 55.6 mm, respectively), indicating a pre-existing anatomically short hard palate (*Table 2*). The SP length was also relatively short measuring 32 mm. The pharyngeal depth and OPA dimensions were within normal limits. The patient subsequently had two maxillary osteotomy procedures; one surgery to advance the maxilla 8mm and down-graft 4 mm, and the second surgery to reposition the maxilla superiorly 3 mm and advance an additional 2 mm. Comparing the initial measurements to those from the pre-HPPBO cephalogram (*Figure 2C*) showed the hard palate length from ANS to PNS and PtA to PNS significantly decreased from 52 to 45 mm and 51 to 45 mm, respectively. A major factor in this change was the ANS and PtA were removed and/or resorbed from the previous two maxillary surgeries, resulting in 4
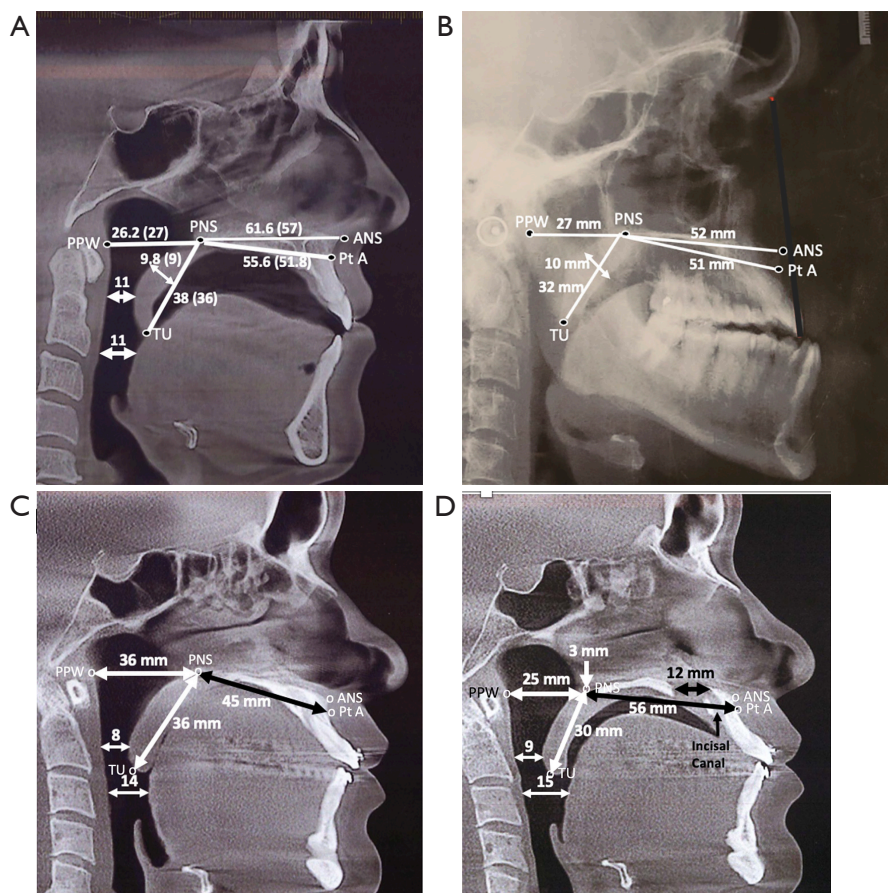
Figure 2 Provides the cephalometric radiographic analyses used in this case presentation. (A) An anatomically normal lateral cephalometric sagittal tomogram is used to identify the anatomical landmarks used for the lateral cephalometric analysis. The specific anatomical landmarks and abbreviations used in the analysis are described in *Table 1*. The normal male and female (in parentheses) dimensions of the hard palate, soft palate, and oropharyngeal structures are illustrated. Normal values are recorded in *Table 2*. (B) Lateral cephalogram of the patient prior to any orthognathic surgical procedures. Hard palatal length indicates it is significantly shorter than normal. The pharyngeal depth is within normal range, although the soft palate length is shorter than normal (*Table 2*). There was no history of chronic gag reflex prior to surgery. (C) The lateral cephalometric radiograph was acquired following the two previous maxillary osteotomies but prior to the HPPBO surgical procedure. The hard palate length is significantly shorter than normal, due to pre-existing short hard palate as well as removal and/or bone resorption at the ANS and PtA areas of the maxilla. The pharyngeal depth is significantly greater than normal indicating the PNS is positioned further forward relative to the cranial base and tongue than normal. Note that the soft palate rests directly against the posterior aspect of the tongue. (D) One-year post HPPBO follow-up lateral cephalometric radiograph shows the 12-mm posterior repositioning of the mid-palatal structures noted at the anterior bone gap. The PNS moved posteriorly 11 mm and pharyngeal depth developed a normal dimension. The hard palate was lengthened significantly to 56 mm and within normal range. PPW, posterior pharyngeal wall; PNS, posterior nasal spine; TU, tip of uvula; ANS, anterior nasal spine; PtA, Point A, HPPBO, hard palate push-back osteotomy.

to 5 mm of additional bone loss, thus further contributing to the already short hard palatal dimension. SP length increased from 32 to 36 mm. The pharyngeal depth prior to any surgery was within normal limits (27 mm), but the maxillary advancement increased it to an abnormal 36-mm length which is compatible with the short hard palate.

Interestingly, the OPA decreased 2 mm at the SP level while at the TB increased 4 mm.

Following the two maxillary surgeries, the cephalometric measurements demonstrated the length of the hard palate from ANS to PNS was 45 mm and from PtA to PNS was 45 mm, indicating the hard palate was significantly shorter

than normal (*Figure 2C*). The pharyngeal depth was 36 mm; significantly greater than the normal range, related to the short hard palate. The pre-HPPBO SP length was 36 mm and the thickness 10 mm, both within normal range. The OPA, measured from PPW to the SP and PPW to the TB were 8 and 14 mm, respectively (*Figure 2C*). The foreshortened length of the hard palate implicated the SP was positioned anatomically further anterior than normal, thus increasing the articulation of the SP against the posterior aspect of the tongue, creating the chronic gag reflex.

Following referral to the senior author (LM Wolford), two soft tissue surgical procedures were tried first to eliminate the gag reflex. A uvulectomy was performed (01/2011) to remove the elongated uvula, without improvement. A modified uvulopalatopharyngoplasty (UPPP) (10) was then performed (10/2012) to suspend the SP upward, but this was also unsuccessful (*Figure 1*). The HPPBO procedure was then considered and performed (05/2013).

Surgical technique

The premise for the HPPBO procedure is to lengthen the hard palate by extending the mid-palatal bone posteriorly in order to displace the SP also posteriorly to disengage it from constant contact with the posterior aspect of the tongue to eliminate the chronic gag reflex. The procedure includes bilateral parallel parasagittal palatal osteotomies, an anterior horizontal connecting osteotomy, and then reposition the mid-palatal bone posteriorly with fixation to provide a stable bony scaffold to support the SP vertically and posteriorly. A maxillary anterior vestibular incision is made, piriform rims identified, and the nasal mucosa dissected off the floor of the nose from the piriform rim area to the posterior border of the palate, as well as the inferior portion of the nasal septum. A nasal septal osteotome is used at the floor of the nose to separate the septum from the palate. The incision is closed.

A circum-palatal incision is made approximately 5 to 6 mm below the gingival crest, lateral to the greater palatine foramen down onto bone (*Figure 3A*). A mucoperiosteal flap is reflected to the posterior border of the hard palate maintaining the integrity of the greater palatine arteries. Approximately a 20-mm anterior horizontal osteotomy

Table 1 Abbreviations and definitions for cephalometric analysis

Abbreviation	Definition
ANS	Anterior nasal spine
PNS	Posterior nasal spine
PtA	Point A
TU	Tip of uvula
PPW	Posterior pharyngeal wall
OPA	Oropharyngeal airway
SP	Soft palate
TB	Tongue base

Table 2 Anatomical measurements of hard and soft palatal structures normal dimensions verses case presentation

Variable	Normal female	Normal male	Patient prior to any surgery	Patient pre-HPPBO	Patient post-HPPBO	Pre- and post-HPPBO changes
Hard palate length						
ANS-PNS (mm)	57.0±4.0	61.6±3.0	52	45	56	11
PtA-PNS (mm)	51.8±4.0	55.6±3.0	51	45	56	11
Soft palate length (PNS-TU) (mm)	36.2±3.0	38.0±2.0	32	36	30	−6
Soft palate thickness (mm)	9.0±2.0	9.8±1.0	10	10	10	0
Pharyngeal depth (PPW-PNS) (mm)	27.0±3.0	26.2±5.0	27	36	25	−11
OPA						
PPW-SP (mm)	11±2	11±2	9	8	9	+1
PPW-TB (mm)	11±2	11±2	10	14	15	+1

Normal values from references (7-9). HPPBO, hard palate push-back osteotomy; ANS, anterior nasal spine; PNS, posterior nasal spine; PtA, Point A; TU, tip of uvula; PPW, posterior pharyngeal wall; OPA, oropharyngeal airway; SP, soft palate; TB, tongue base; mm, millimeters.
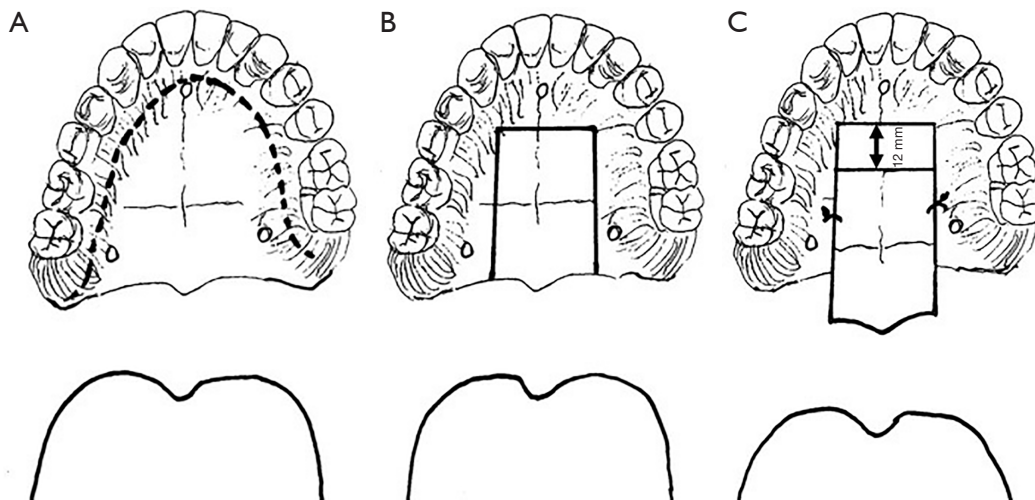
Figure 3 Surgical procedure for HPPBO. (A) Illustrated is the palatal incision (dashed line). (B) The palatal osteotomy segment is made as wide as possible. A horizontal osteotomy is made about 4 mm posterior to the incisive canal approximately 20 mm in width connected to parallel parasagittal osteotomies made just medial to the greater palatine foramens and extending through the posterior border of the palate. (C) The palatal segment is mobilized, moved posteriorly, and stabilized with interosseous wires or small thin bone plates. The palatal incision is closed. HPPBO, hard palate push-back osteotomy.

is completed with a 702 bur about 4 mm posterior to the incisive foramen. A reciprocating saw is used to make bilateral parallel parasagittal palatal osteotomies, approximately 20 mm apart, connecting with the anterior horizontal osteotomy and extended through the posterior border of the palate, staying medial to the greater palatine foramen (*Figure 3B*). The palatal bone is then mobilized and repositioned posteriorly the predetermined amount (in this case 12 mm). Interosseous holes are placed just medial and anterior to the greater palatine foramen and through the anterior and lateral aspect of the palatal bone. A 28-gauge stainless steel wire is placed through these holes bilaterally and twisted to secure the palatal bone in its new position (*Figure 3C*) or thin bone plates can be used. The palatal flap is sutured back into position with 4-0 PDS suture (Ethicon, Somerville, NJ, USA). This completes the surgical procedure. A gauze pack is placed between the tongue and the palatal soft tissues and left in position for approximately 6 hours for hemostasis and readapt the soft tissues to the palate. The anterior palatal bone defect for the most part will remain and be covered with normal healthy soft tissue.

Post-operative course

The patient was seen on the first post-surgery day, showing good adaptation of the soft tissues against the palate, and

the patient noticed relief of his chronic gag reflex. A 1-year post-surgery lateral cephalometric evaluation demonstrates posterior positioning of the palate 11 mm at the PNS compared to pre-surgery. The SP was displaced posteriorly and disarticulated off the tongue creating separation (*Figure 2D*). The 12 mm gap in the anterior palate can be seen correlating to the amount of posterior movement of the palate. The patient continued to have significant relief of his gag reflex at the 2-year and 9-year 5-month post-surgery follow-ups. The changes that occurred anatomically are seen in *Table 2* and include the following: (I) hard palate lengthened 11 mm at PNS; (II) SP shortened 6 mm (36 to 30 mm), but this in part was related to the uvulectomy and SP suspension procedure performed after the pre-HPPBO imagining was acquired and prior to the HPPBO surgery; (III) the pharyngeal depth decreased 11 mm from 36 to 25 mm regaining the normal range; (IV) the OPA increased 1 mm at both the SP and TB; and (V) the PNS was positioned 3 mm inferior compared to pre-HPPBO. The PNS positional change had minor effects on the measurements. Although the palatal segment was posteriorly positioned 12 mm as measured at the anterior surgical site, the 3-mm inferior post-surgery position shortened the distance from PtA to PNS by 1 mm and as well as increased the pharyngeal depth by 1 mm. The dropping of the PNS may have been related to soft tissue tension created by the previous soft palatal

procedures. Using thin bone plates to stabilize the palatal shelf instead of wire fixation could minimize this shift. The patient was evaluated at 9 years 5 months post-HPPBO surgery. Patient perspective; he reported no significant gag reflex episodes since the HPPBO procedure.

Written informed consent for publication of this case report and accompanying images was not obtained from the patient or the relatives after all possible attempts were made. However, no means of patient identification are evident in the materials presented.

Discussion

Key findings

A new surgical procedure HPPBO is presented that was successful in significantly reducing chronic gag reflex for the patient in this case report. The patient had a short hard palate and a hypoplastic maxilla. The initial maxillary advancement procedure pulled the SP forward impacting on the posterior tongue stimulating the chronic gag reflex. The HPPBO procedure posteriorly positioned the hard and SPs disarticulating the SP from the posterior aspect of the tongue, eliminating the chronic gag reflex. For this procedure, there are important criteria that must be met to avoid complications.

(I) Short hard palate positioning the SP forward against the posterior tongue.

(II) Chronic gag reflex from SP contact on the posterior aspect of the tongue.

(III) Normal OPA since the procedure will displace the SP posteriorly and may decrease the OPA space.

(IV) Non-surgical procedures failed to eliminate the chronic gag reflex.

(V) The anterior palatal bone defect for the most part will remain and be covered with soft tissue. Similar to the bony defects as seen in cleft palatal repair or closure of oronasal fistulas, except with normal healthy tissue. As observed in this patient, there should be no long-term impact or alteration in eating, speaking, or other functions involving the palate and oral cavity.

There have been extensive publications on the non-surgical management of chronic gag reflex, but minimal reports on surgical approaches.

Strengths and limitations

The HPPBO procedure eliminated chronic gag reflex for this patient when other surgical treatment options failed and may be beneficial for other patients who met the surgical criteria. Limitations include the narrow criteria for application of this technique, single case report, potential risks and complications, and the requirement of further studies to verify the efficacy of this surgical protocol. Potential risks and complications could include: velopharyngeal insufficiency, decreased OPA creating partial airway obstruction, sleep apnea, snoring, chocking, nasopharyngeal reflux, dysphagia, delayed bolus clearance, palatal flap necrosis oronasal fistula development, adverse effect on speech, and continued chronic gag reflex. Although we did not encounter any complications, close observation post-operatively would allow for early intervention if a complication were to occur. For this procedure it is very important that the OPA dimensions are within normal range prior to surgical intervention.

The gag reflex

The gag reflex is an involuntary, physiological neuromuscular protective mechanism that thwarts noxious irritants from entering the respiratory system to minimize pulmonary aspiration and choking (1-3). The gag reflex is believed to be an inborn reflex, like swallowing, which regresses during the first 4 years of life and continues to move posteriorly to the tonsil pillars by the time the primary dentition is present (11). Assuming that swallowing is a smooth, coordinated action of muscle peristalsis signaled by stimulation of the mouth, tongue, palate, pharynx and larynx; the gag reflex occurs when the peristalsis of the same muscles is uncoordinated, spasmodic, and occurring in the opposite direction (1,2,12).

The gag reflex can typically be elicited by either somatogenic (physical stimuli) or psychogenic sources (4), but this can be difficult to discern clinically (1). There are five zones in the oral cavity that can elicit the tactile gag reflex in an adult. These trigger zones are comprised of the glossopalatine and palatopharyngeal arches, posterior third of the tongue, the SP, the uvula and the PPW (1,2,4). While tactile stimulation can activate the gag reflex, other physiological factors such as visual, auditory, olfactory or psychic (fear/anxiety) stimuli may also elicit the gag reflex (4,13).

When a trigger zone is stimulated, the afferent fibers of the reflex arc originate from CN V (trigeminal), IX (glossopharyngeal) and X (vagus) to transmit a stimulus to the gag reflex center in the medulla oblongata (2). The

medulla oblongata is responsible for several functions of the autonomic nervous system; most notable are the reflex centers for gagging (gag reflex), vomiting (emetic nucleus) and swallowing (palatal reflex) (14). The gag reflex center is in close approximation to the chemoreceptor trigger zone situated in area postrema (15), as well as the salivary and cardiovascular centers of the medulla. The intimate proximity of all these centers may explain why gagging is often associated with sialorrhea, epiphora, hyperhidrosis, or syncope (4,15-17). The gag reflex also receives input from the cerebral cortex, permitting the gag reflex to be instigated by imagination or suppressed by distraction (18). From the medulla oblongata efferent impulses are transmitted through CN V (trigeminal), VII (facial), IX (glossopharyngeal), X (vagus), XI (accessory), and XII (hypoglossal) causing the spasmodic, uncoordinated, ejector mechanism of the muscles in an attempt to expel the irritant (1,18-20).

Despite the fact a normal gag reflex serves as an airway protective mechanism, it can be a considerable disability especially when an individual is afflicted with an exaggerated or hyperactive gag reflex. As mentioned, the gag reflex can be activated secondary to any of the five types of stimuli: tactile, visual, auditory, olfactory or psychic (fear/anxiety). In particular, when an abnormal anatomical interrelationship exists within the oral structures, it can cause stimulation of any of the five trigger points previously discussed. A discrepancy of the oropharynx anatomical interrelationship can lead to activation of a trigger point. The pathological etiologies may include onset of tumors in the SP, base of tongue, tonsils, adenoids, oropharyngeal area or chronic swelling of the tonsil or adenoid tissues (1-3). An excessively elongated or thickened SP can likely contribute to chronic gag reflex. Likewise, an enlarged tongue could potentially contribute by allowing displacement of the posterior aspect of the tongue in a posterosuperior direction leading to articulation of the tongue against the SP. Patients with a significantly retruded maxillo-mandibular complex with high occlusal plane angle facial morphology that displaces the tongue posteriorly against the SP and PPW can create a gag reflex (21,22). Another possible etiology, as seen in this patient, could occur when the hard palate is abnormally short preventing the SP from maintaining its normal anatomical relationship, allowing the SP to occlude on the posterior aspect of the tongue. Prior to the first maxillary surgical procedure, this patient did not complain of a gag reflex. The patient had an abnormally short hard palate, but the retruded maxilla enabled clearance of the SP from

articulating against the base of the tongue. The maxillary advancement brought the tongue forward to occlude against the TB initiating the gag reflex.

Numerous attempts to quantify the gag reflex in dental patients have been proposed in the literature (3,18,23-30). The most notable index was introduced by Dickinson and Ficke in 2005. They devised a Gagging Severity Index (GSI) in order to classify the severity of the patient's gagging into five grades in ascending severity with recommended management (26).

- ❖ Grade I: normal gagging reflex—can be controlled by the patient;
- ❖ Grade II: mild gagging—infrequent gagging during treatment, controlled easily by the patient;
- ❖ Grade III: moderate gagging—routine gagging during treatment, gagging prevention methods required;
- ❖ Grade IV: severe gagging—gagging with all procedures including visual exam, routine treatment is impossible without interventions;
- ❖ Grade V: very severe gagging—spontaneously occur without physical contact, treatment impossible without appropriate interventions.

Comparison of treatment modalities

The nonsurgical treatment modalities offered in the literature for management of gag reflex is considerably expansive with varied success. The nonsurgical treatments for a hyperactive gag reflex can be classified as behavioral modifications, pharmacological interventions and non-pharmacological interventions (4). Behavioral modifications attempt to lessen anxiety and activities that promote gagging (31). These modifications embrace a wide scope of techniques to include various methods such as cognitive behavioral therapy (32), errorless learning (33), hypnosis/suggestion (34,35), relaxation techniques (36), and distraction (30). Pharmacological interventions can range from local anesthetics to interrupt the afferent fibers of the gag reflex arc to other short-term solutions with centrally acting drugs such as sedatives/tranquillizers, antihistamines, and various parasympatholytic agents (37). Additional pharmacological interventions are also suggested in the literature such as trimethobenzamide (38), nitrous oxide (39), granisetron (18), as well as general anesthesia (1). Non-pharmacological interventions typically include transcutaneous electric nerve stimulation (TENS) device (40), acupuncture (41), management with various oral prostheses (4,42), and sucking
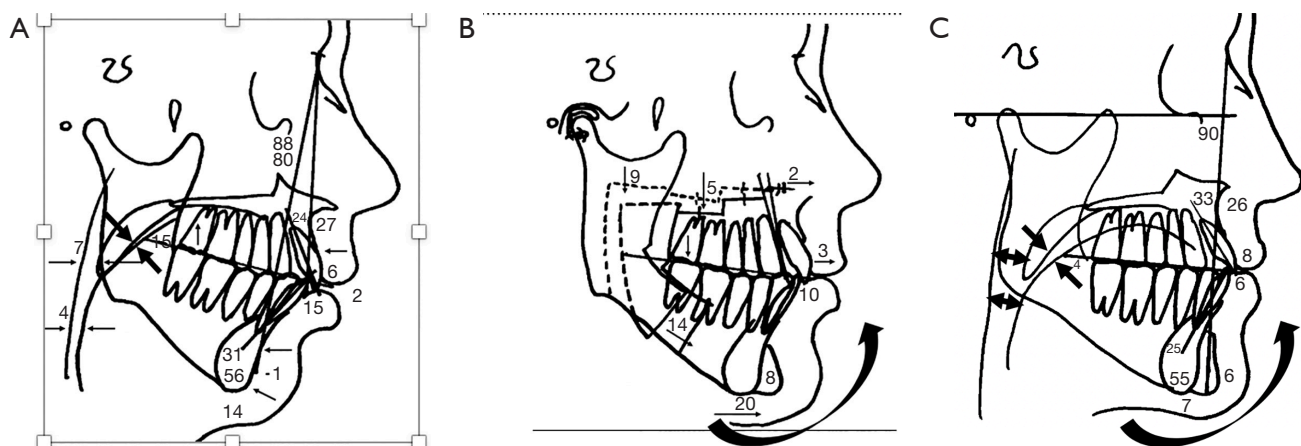
Figure 4 Effect of high occlusal plane angle facial morphology on tongue and soft palate relation. (A) A lateral cephalometric radiograph of a male patient with a high occlusal plane angle facial morphology resulting in a retruded mandible and maxilla. This displaces the tongue posteriorly against the soft palate as well as significantly decreases the oropharyngeal airway. These patients can also experience difficulties swallowing, choking, breathing, gagging, and sleep apnea because of the decreased oropharyngeal airway. These types of patients are not amenable to the HPPBO technique as this could further decrease the functional airway and worsen the other symptoms. Numbers are linear and angular cephalometric analysis values, confirming high occlusal plane angle facial morphology. (B) Maxillary and mandibular osteotomies to advance the maxillomandibular complex in a counterclockwise direction will improve breathing, jaw function, and esthetics and may eliminate other symptoms such as difficulties swallowing, choking, and gagging. The mandible advances further forward than the maxilla with this movement. Numbers indicate the surgical movements in millimeters. (C) This counterclockwise rotation of the maxillo-mandibular complex increases the oropharyngeal airway, can create separation between the soft palate and tongue, and may decrease presurgical symptoms such as the gag reflex. Numbers are the post-surgical cephalometric linear and angular values. HPPBO, hard palate push-back osteotomy.

on marbles throughout the day (43).

Although the nonsurgical treatment interventions offered is vastly documented, the literature concerning surgical options for management of chronic gag reflex is scant, to say the least. One of the first surgical techniques proposed was published in 1940 by Leslie (6) who noted persistent gagging in certain patients unable to tolerate a complete denture. He suggested the persistent gagging occurred secondary to an atonic and relaxed SP that no longer maintained its normal suspension allowing the uvula to have contact with the tongue and the SP with the pharyngeal walls. He advocated a procedure to shorten and tighten the SP. The senior author was unsuccessful utilizing a modified UPPP technique (10) to elevate the SP off the tongue for managing this patient's chronic gag reflex.

Kim *et al.* (5) noted an intense gag reflex activated by any contact at the junction of the pillars of the fauces and the tongue in a patient who underwent a UPPP with tonsillectomy for Obstructive Sleep Apnea (OSA). They successfully used the medical procedure of radiofrequency ablation for this patient to overcome the trigger-point hypersensitivity gag reflex after an unfortunate attempt with botulinum toxin.

Patients with a retruded maxilla and mandible may present with sleep apnea and can experience difficult swallowing, chocking, and sometimes increased gag reflex, particularly those with a high occlusal plane facial morphology where the maxilla and mandible are significantly retruded, displacing the tongue posteriorly against the SP and PPW creating these symptoms (*Figure 4A*). The surgical treatment for these patients is advancement of the maxillo-mandibular complex with the counter-clockwise rotation that predictably advances the jaws, base of tongue and SP forward to increase the OPA and eliminate the sleep apnea, difficulty swallowing, chocking, and gag reflex (*Figure 4B*) (21,22,44-49). For this counterclockwise rotation of the maxillo-mandibular complex, the mandible and base of the tongue advance further forward than the maxilla and SP, naturally creating separation between the SP and tongue (*Figure 4C*). In fact, this counter-clockwise rotation of the maxillo-mandibular complex is generally the most predictable method to eliminate a decreased OPA and sleep apnea. However, this patient presented herein, had a relative normal OPA and

already had his maxilla advanced, but never complained of sleep apnea symptoms, even prior to his first surgery. Therefore, further advancement of the maxillo-mandibular complex would not improve his gag reflex. The inherent shortness of the hard palate was a primary contributing factor since this anatomical variant positioned the SP against the back of the tongue creating the gag reflex.

Implications and actions needed

The HPPBO procedure may be indicated to treat patients with chronic gag reflex who met the specific inclusion criteria. Further studies are necessary to verify the efficacy of this surgical technique.

Conclusions

The gag reflex is an important mechanism for protection of the OPA. Patients with hypersensitive gag reflexes related to anatomical interferences, as seen in the case presented, may exacerbate the symptoms of this pathological process. When traditional non-surgical treatment methods are ineffective, surgery may be indicated. The HPPBO procedure was devised to eliminate chronic gaging caused by the SP articulating against the TB. Surgically moving the hard and SPs posteriorly disengages the SP from continuous contact with the base of the tongue, resolving the chronic gag reflex. The patient presented herein met the surgical criteria for HPPBO with resultant significant gag reflex relief, maintained at a 9-year 5-month follow-up. Additional studies are necessary to validate the efficacy of this technique.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://fomm.amegroups. com/article/view/10.21037/fomm-22-39/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent for publication of this case report and accompanying images was not obtained from the patient or the relatives after all possible attempts were made.

References

1. Bassi GS, Humphris GM, Longman LP. The etiology and management of gagging: a review of the literature. J Prosthet Dent 2004;91:459-67.
2. Conny DJ, Tedesco LA. The gagging problem in prosthodontic treatment. Part I: description and causes. J Prosthet Dent 1983;49:601-6.
3. Nuaimy KMT. Gag Problem in Dental Treatment Assessment and Methods to Control it. Al-Rafidain Dental Journal 2010;10:287-91.
4. Eachempati P, Kumbargere Nagraj S, Kiran Kumar Krishanappa S, et al. Management of gag reflex for patients undergoing dental treatment. Cochrane Database Syst Rev 2019;2019:CD011116.
5. Kim EY, Courey MS, Kezirian EJ. Treatment of trigger-point hypersensitivity of gag reflex following surgical treatment of obstructive sleep apnea. Otolaryngol Head Neck Surg 2011;145:1055-6.

6. Leslie SW. A new operation to overcome gagging as an aid to denture construction. J Can Dent Assoc 1940;6:291-4.

7. Riolo ML. An atlas of craniofacial growth: cephalometric standards from the University School Growth Study, the University of Michigan. 1974:105-106. Available online: https://catalog.hathitrust.org/Record/000036423

8. Khoja A. Soft Palate Dimensions and Nasopharyngeal Depth (Need's Ratio) In Different Sagittal and Vertical Skeletal Patterns: A Lateral Cephalometric Study. Adv Dent & Oral Health 2018;8:555726.

9. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea syndrome: a surgical protocol for dynamic upper airway reconstruction. J Oral Maxillofac Surg 1993;51:742-7; discussion 748-9.

10. Wolford LM, Mehra P. Modified uvulopalatopharyngoplasty: the lateral inversion flap technique. J Oral Maxillofac Surg 2001;59:1242-3.

11. Ardelean L, Bortun C, Motoc M. Gag reflex in dental practice-etiological aspects. TMJ 2003;53:312-5.

12. Jurel SK, Singh P, Singh M. Gagging: aetiology and management. Indian Journal of Medical Specialities 2011;2:127-32.

13. Fleece L, Linton P, Dudley B. Rapid elimination of a hyperactive gag reflex. J Prosthet Dent 1988;60:415-7.

14. Hornby PJ. Central neurocircuitry associated with emesis. Am J Med 2001;111 Suppl 8A:106S-112S.

15. Miller AD, Leslie RA. The area postrema and vomiting. Front Neuroendocrinol 1994;15:301-20.

16. Kaira L, Dabral E, Kukreja H. Gagging a Review. NUJHS 2014;4:149-55

17. Blumenfeld H. Neuroanatomy through clinical cases. Sunderland: Sinauer Associates, Inc.; 2010.

18. Barenboim SF, Dvoyris V, Kaufman E. Does granisetron eliminate the gag reflex? A crossover, double-blind, placebo-controlled pilot study. Anesth Prog 2009;56:3-8.

19. Larner AJ. A Dictionary of Neurological Signs. Springer; 2016.

20. Bhanot R, Sharma A, Bansal P, et al. Gagging-Causes and Management in Prosthodontic Treatment: A Review of Literature. Indian Journal of Dental Sciences 2010;2:25-8.

21. Wolford LM, Chemello PD, Hilliard F. Occlusal plane alteration in orthognathic surgery—part I: Effects on function and esthetics. American Journal of Orthodontics and Dentofacial Orthopedics 1994;106:304-16.

22. Wolford LM, Chemello PD, Hilliard FW. Occlusal plane alteration in orthognathic surgery. J Oral Maxillofac Surg 1993;51:730-40; discussion 740-1.

23. Faigenblum MJ. Retching, its causes and management in prosthetic practice. Br Dent J 1968;125:485-90.

24. Morstad AT, Petersen AD. Postinsertion denture problems. J Prosthet Dent 1968;19:126-32.

25. Saita N, Fukuda K, Koukita Y, et al. Relationship between gagging severity and its management in dentistry. J Oral Rehabil 2013;40:106-11.

26. Dickinson CM, Fiske J. A review of gagging problems in dentistry: I. Aetiology and classification. Dent Update 2005;32:26-32.

27. Akarslan ZZ, Biçer AZ. Utility of the gagging problem assessment questionnaire in assessing patient sensitivity to dental treatments. J Oral Rehabil 2012;39:948-55.

28. Van Linden van den Heuvell GF, Ter Pelkwijk BJ, Stegenga B. Development of the Gagging Problem Assessment: a pilot study. J Oral Rehabil 2008;35:196-202.

29. Bilello G, Fregapane A. Gag reflex control through acupuncture: a case series. Acupunct Med 2014;32:24-7.

30. Krol AJ. A new approach to the gagging problem. J Prosthet Dent 1963;13:611-6.

31. Klepac RK, Hauge G, Dowling J. Treatment of an overactive gag reflex: two cases. J Behav Ther Exp Psychiatry 1982;13:141-4.

32. Barsby MJ. The control of hyperventilation in the management of 'gagging'. Br Dent J 1997;182:109-11.

33. Wilks CG. Functional analysis of the gag reflex. Br Dent J 1985;159:72.

34. Eitner S, Wichmann M, Holst S. A long-term therapeutic treatment for patients with a severe gag reflex. Int J Clin Exp Hypn 2005;53:74-86.

35. Weyandt JA. Three case reports in dental hypnotherapy. Am J Clin Hypn 1972;15:49-55.

36. Neumann JK, McCarty GA. Behavioral approaches to reduce hypersensitive gag response. J Prosthet Dent 2001;85:305.

37. Murthy V, Yuvraj V, Nair PP, et al. Management of exaggerated gagging in prosthodontic patients using glossopharyngeal nerve block. BMJ Case Rep 2011;2011:bcr0720114493.

38. Grace LE, Hackney RD, Dobbs EC. A clinical evaluation of trimethobenzamide (Tigan) as an antigagging drug. Oral Surg Oral Med Oral Pathol 1963;16:422-5.

39. Chidiac JJ, Chamseddine L, Bellos G. Gagging prevention using nitrous oxide or table salt: a comparative pilot study. Int J Prosthodont 2001;14:364-6.

40. Morrish RB Jr. Suppression and prevention of the gag reflex with a TENS device during dental procedures. Gen Dent 1997;45:498-501.

41. Rosted P, Bundgaard M, Fiske J, et al. The use of

acupuncture in controlling the gag reflex in patients requiring an upper alginate impression: an audit. Br Dent J 2006;201:721-5; discussion 715.

42. Conny DJ, Tedesco LA. The gagging problem in prosthodontic treatment. Part II: Patient management. J Prosthet Dent 1983;49:757-61.

43. Singer IL. The marble technique: A method for treating the "hopeless gagger" for complete dentures. J Prosthet Dent 1973;29:146-50.

44. Coleta KD, Wolford L, Gonçalves JR, et al. Maxillo-mandibular counter-clockwise rotation and mandibular advancement with TMJ Concepts® total joint prostheses. Int J Oral Maxillofac Surg 2009;38:126-38.

45. Gonçalves JR, Gomes LC, Vianna AP, et al. Airway space changes after maxillomandibular counterclockwise rotation and mandibular advancement with TMJ Concepts® total joint prostheses: three-dimensional assessment. Int J Oral Maxillofac Surg 2013;42:1014-22.

46. Goncalves JR, Buschang PH, Goncalves DG, et al. Postsurgical stability of oropharyngeal airway changes following counter-clockwise maxillo-mandibular advancement surgery. J Oral Maxillofac Surg 2006;64:755-62.

47. Mehra P, Downie M, Pita MC, et al. Pharyngeal airway space changes after counterclockwise rotation of the maxillomandibular complex. Am J Orthod Dentofacial Orthop 2001;120:154-9.

48. Mehra P, Wolford LM. Surgical management of obstructive sleep apnea. Proc (Bayl Univ Med Cent) 2000;13:338-42.

49. Chemello PD, Wolford LM, Buschang PH. Occlusal plane alteration in orthognathic surgery--Part II: Long-term stability of results. Am J Orthod Dentofacial Orthop 1994;106:434-40.