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The Nature of Nasal Fricatives: Articulatory-Perceptual Characteristics and Etiologic Considerations

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Abstract

Nasal fricatives (NFs) are unusual, maladaptive articulations used by children both with and without palatal anomalies to replace oral fricatives. Nasal fricatives vary in articulatory, aerodynamic, and acoustic-perceptual characteristics with two generally distinct types recognized. One type is produced with velopharyngeal (VP) constriction that results in turbulent nasal airflow and, frequently, tissue vibration (flutter) at the VP port. Trost (1981) described these as posterior NFs that have a distinctive snorting quality. A second type of NF is produced without significant VP constriction resulting in turbulent airflow generated at the anterior liminal valve of the nose. Of importance, both types are “active” alternative articulations in that the speaker occludes the oral cavity to direct all airflow through the nose (Harding & Grunwell, 1998). It is this oral gesture that differentiates NFs from obligatory (or passive) nasal air escape that may sound similar due to incomplete VP closure. The purpose of this article is to (1) describe the articulatory, aerodynamic, and acoustic-perceptual nature of NFs, and (2) propose a theoretical framework for the acquisition of NFs by children both with and without cleft palate.

A NF is essentially an alternative articulation for an oral fricative in which aperiodic noise is generated either in the nasopharynx and/or nasal passage, thus bypassing the oral cavity. When NFs are produced in the nasopharynx, tissue vibration often accompanies the aperiodic noise. While NFs are typically substituted for sibilants, any of the oral fricatives or affricates can be affected, including derived affricates such as /tr/. Children both with and without cleft palate produce NFs. Children without cleft palate who produce NFs are an enigma to speech-language pathologists (SLPs). Why would a child learning the phonetics and phonology of his/her language make an already complex motor behavior such as fricative production even more complicated? What causes a child to learn NFs? One plausible theory involves conductive hearing loss related to frequent middle-ear disease as a mediating factor (Peterson-Falzone & Graham, 1990). That is, a child with conductive hearing loss may benefit from enhanced bone-conducted auditory feedback—and perhaps vibratory-tactile feedback—during production of NFs. While this explanation is attractive, it does not account for the initial event(s) that trigger nasal turbulence in a child without cleft palate that leads to accepting nasal frication as an auditory target for oral fricatives. Regardless of a known precipitating factor, most authors attribute the development of NFs in

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children without cleft palate to “faulty” VP learning (Kummer, 2008; Trost-Cardamone & Bernthal, 1993).

A potential trigger for nasal turbulence in a child with repaired cleft palate—or unrepaired submucous cleft palate—is easier to identify. In this case, there is the likely possibility of incomplete VP closure that precipitates nasal airflow during early linguistic development and attempts to produce oral fricatives. Consequently, it has been assumed that NFs produced by children with cleft palate are learned, compensatory responses to VP inadequacy. As discussed later in this article, there are both theoretical considerations and some empirical evidence to question this assumption, especially for posterior NFs that require near complete closure of the VP port.

Characteristics of Nasal Fricatives

In this article, the term NF is used exclusively to denote a learned, alternative production of fricative consonants (i.e., phonemic substitutions) and not obligatory co-occurrence of nasal airflow that may accompany either fricative and/or stop-plosive consonants as a result of incomplete VP closure. Understandably, because the perceptual consequences of obligatory nasal air escape may be similar or even identical to a NF, some authors and clinicians may describe obligatory nasal air escape as a NF. Sound scientific and clinical practice, however, demand the use of accurate and well-defined terms.

Two generally distinct types of NFs have been described in the literature. These descriptions are based primarily upon location of the constriction that generates turbulent airflow—either anterior or posterior—and secondarily upon the resulting perceptual nature of frication. Accordingly, the terms “anterior” and “posterior” will be used in this article to describe the articulatory, aerodynamic, and acoustic-perceptual characteristics of NFs. Anterior NFs will be described first as these productions are somewhat less complex in both articulatory and perceptual characteristics than posterior NFs and will set the stage for a better distinction between the two.

Anterior Nasal Fricatives

Articulation of an anterior NF involves actively occluding the oral cavity and forcing air through the nasal cavity. Turbulence (frication) is created when airflow reaches the anterior nasal valve, the smallest cross-sectional area of the nose (Proctor, 1982). Anterior NFs can be considered as voiceless nasal consonants produced with turbulent nasal airflow to create frication (Harding & Grunwell, 1998; Sell, Harding, & Grunwell, 1999). As described by Sell et al. (1999), the production involves “complete oral closure at the place of articulation with the airstream directed exclusively nasally” (p. 22). Stopping of oral airflow can occur at the three typical places of nasal consonant articulation—bilabial, alveolar, and velar (see Figure 1)—or at atypical locations such as the hard palate, similar to production of a middorsum palatal stop.

As illustrated in Figure 1, anterior NFs are produced without significant closure of the VP port. Thus, if driving air pressure is high enough and the anterior nasal valve is small enough, aperiodic noise is generated as air passes through the nose.¹ Perceptually, an

anterior NF can be simulated by forcefully exhaling through the nose with the lips closed. Some speakers also exhibit a nasal grimace during production of anterior NFs. This gesture would further constrict the nasal valve and enhance generation of aperiodic noise (Zajac, Mayo, Kataoka, and Kuo, 1996). Indeed, as reported by Zajac et al. and shown below, overall spectral characteristics of anterior NFs are strikingly similar to the spectral components of oral fricatives.

Figure 2 illustrates the acoustic and spectral characteristics of anterior NFs. The figure shows the oral microphone signal, nasal microphone signal, and spectrogram generated from the nasal signal during production of anterior NFs substituted for /s/. The speaker was an 8 year-old girl with repaired bilateral cleft lip and palate who produced the phrase “say see see see again”. The oral and nasal signals were recorded using the headset and microphones of the Nasometer.

As seen in Figure 2, there are clear stop gaps in the oral signal with accompanying acoustic energy in the nasal signal during production of each NF. The phonetic symbol for /s/ is used in the figure and the three targeted /si/ syllables are labelled. Acoustic energies in the nasal spectrogram are prominent from approximately 3 to 12 kHz, a range that encompasses the frequencies expected for production of an oral /s/. Playback of the oral signal gives the clear perception of a stop—a cross between /d/ and /g/, or, most likely a palatal stop.² Playback of the nasal signal gives the clear perception of a fricative, very similar to /s/.

Posterior Nasal Fricatives

Trost (1981) described the posterior NF as having variable articulatory expression. Based upon radiographic imaging, Trost noted that (a) the velum approximated but did not contact the pharyngeal wall, and (b) the tongue articulated with the velum in some patients. Peterson (1975) and Peterson-Falzone and Graham (1990) also described a retracted tongue that appears to contact the soft palate. Additionally, Trost (1981) noted that the velum tended to flutter during production of the posterior NF, further contributing to turbulence. Indeed, as discussed below, it is the flutter component of the posterior NF that contributes most to its distinct perceptual quality, sometimes described as “snorting”.³ Peterson-Falzone, Trost-Cardamone, Karnell, and Hardin-Jones (2006) nicely summarized the posterior NF as “a turbulent velopharyngeal fricative articulation associated with small VP openings” (p. 29). Perceptually, many clinicians will describe the distinct quality of the posterior NF as nasal “snorting” and/or “turbulence.”

Two aspects of the above description of the posterior NF warrant further discussion. First, although Trost (1981) does not explicitly note oral stopping of airflow, it is clear from

¹Some authors have described anterior NFs as being “nonturbulent” in an apparent attempt to distinguish these articulations from posterior NFs (e.g., Peterson-Falzone, Trost-Cardamone, Karnell, & Hardin-Jones, 2006). Technically, this is inaccurate as audible frication results from turbulent airflow, regardless of the location of the constriction. Anterior NFs, therefore, might be better described as being “non-snorting” or “non-fluttering” as compared to posterior NFs.

²It should be noted that the stop component of a NF, either anterior or posterior, might be difficult to perceive when listening to a speaker, as the nasal frication will dominate.

³Morley (1966) described a “nasopharyngeal snort” as occurring when air is passed through “a sphincter which is closed but not so tightly as to prevent the passage of air as on snorting” (p. 175). Of interest relative to etiology, Morley also notes that the snort may occur (a) when adenoids are present, and (b) temporarily during speech development.

radiographic illustrations that oral occlusion occurs due to tongue-soft palate contact (see Figure 1, p. 135, Peterson-Falzone & Graham, 1990). To be sure, many SLPs routinely pinch a child's nose during production of suspected posterior (and anterior) NFs to confirm if oral closure has occurred. Figure 3 illustrates the complete stoppage of oral airflow during production of posterior NFs. The figure shows the audio, nasal airflow, and oral airflow signals obtained with a microphone and calibrated, oral-nasal pneumotachograph mask during repetition of three /si/ syllables. The speaker was an 8 year-old girl with enlarged tonsils but no cleft palate who substituted posterior NFs for /s/ and /z/ as part of a pattern of phoneme-specific nasal emission. The girl's articulation was otherwise normal and pressure-flow testing showed complete VP closure during production of /pi/ syllables. As illustrated in Figure 3, there is complete stoppage of oral airflow (bottom graph) during each posterior NF while nasal airflow peaks at approximately 200 mL/s. This oral-nasal airflow pattern is typical of posterior NFs. It should be noted that the final nasal airflow peak illustrated in Figure 3 is due to end-of-utterance exhalation. The potential significance of this normal nasal airflow event is discussed later in the article.

A second point to be made concerns the use of the term nasal "turbulence" to describe the distinct perceptual quality of posterior NFs. While turbulent airflow certainly occurs, it is the quasi-periodic flutter of tissue associated with a nearly closed VP port that contributes to the "loud and distracting" sound of posterior NFs.⁴ It should be noted that Kummer (2008) describes this sound as "rustle" and attributes it to the bubbling of secretions within a small VP port. Displacement of mucous within a partially closed and vibrating VP port can certainly contribute noise to a posterior NF. As shown below, however, it is the flutter component that is spectrally and perceptually prominent in a posterior NF.⁵ To be sure, it is this component that distinguishes a posterior NF from an anterior NF that has turbulence but little or no flutter.

Spectral evidence of VP port flutter is displayed in Figure 4. The oral signal, nasal signal, and nasal spectrogram are shown from a 7 year-old boy without cleft palate who substituted posterior NFs for /s/. The oral signal clearly shows stop gaps during targeted /s/ segments, similar to anterior NFs displayed in Figure 2. The nasal spectrogram in Figure 4, however, is "distinct" from anterior NFs in that aperiodic energy is modulated by a quasi-periodic component as evidenced by dark vertical striations that extend throughout the frequency range. An additional difference compared to the anterior NFs in Figure 2 is a prominent low-frequency component, well below 1 kHz. The quasi-periodic component in the spectrum reflects structural vibration, similar to vocal-fold vibration for a vowel. Although the spacing of the vertical striations are somewhat variable within and across the posterior NF segments, the average period in the figure corresponds to a frequency of approximately 80 to 100 Hz. Playback of the posterior NF segments from the nasal audio signal gives the clear perception of a flutter quality.

⁴Beebe (1946) noted that nasalization of /s/ may occur either with or without a flutter component and referred to it as "snoring." This certainly is an apt description of the distinct sound of the posterior NF.

⁵This author is not aware of any acoustic studies that have attempted to identify the noise components of bubbling of mucous.

Both the flutter and turbulent components of posterior NFs can be variable, even within the same speaker and utterance. As seen in Figure 4, while flutter appears to be prominent in the first NF segment, it is somewhat less prominent in the second NF segment. Clinically, this author has observed perceptual variability in the flutter component of posterior NFs to the extent that it may be absent in some productions of the same speaker, making it sound similar to an anterior NF. The variability in the occurrence and extent of flutter is likely dependent upon multiple anatomical and production factors. Anatomical factors may include length and thickness of the velum, presence of velar scar tissue (in speakers with repaired cleft palate), presence of a uvula, presence and size of tonsils and adenoids, and size of the VP gap. Production factors may include magnitude of driving oral air pressure, temporal position of the target in the breath group, and phonetic context related to high versus low vowels. While acoustic-spectral analyses may shed light on some aspects of flutter, high-speed imaging studies are needed to identify the specific anatomical tissues involved. Unfortunately, an inherent problem with clinical imaging procedures such as video endoscopy and/or fluoroscopy is limited sampling rate. Most clinical video is obtained at sampling rates no greater than 30 frames per second, a rate that results in the “blurring” of the VP port as noted by Trost (1981).⁶

Aerodynamic Characteristics of NFs

There is limited information on the aerodynamics of NFs. Zajac and Vallino (2015) studied eight children who produced posterior NFs and four who produced anterior NFs as substitutions for /s/. All of the children had either repaired cleft palate or unrepaired submucous cleft palate. Zajac and Vallino used oral-nasal audio recordings and spectral analysis to confirm NFs as either posterior or anterior. The pressure-flow method was used to determine the volume rate of nasal airflow during production of the NF segments. Velopharyngeal port size was estimated during production of /pi/ syllables. The median rate of nasal airflow for the children who produced posterior NFs was 174 mL/s while the median rate for the children who produced anterior NFs was 365 mL/s. Seven of eight children who produced posterior NFs had VP orifice areas under 5 mm² during production of /pi/. These findings are consistent with a small VP port that limits airflow during production of posterior as compared to anterior NFs. Of further importance, the majority of children who used posterior NFs had adequate VP closure as reflected by port areas under 5 mm². If these children also had adequate VP closure during early acquisition of posterior NFs, then one must question the assumption that these maladaptive articulations are compensatory to gross VP inadequacy. This issue is discussed below.

Etiology of Nasal Fricatives

As noted in the Introduction, the cause of NFs remains elusive, especially in children without history of structural palatal anomalies or oral-motor dysfunction. Peterson (1975) suggests that “The most tempting explanation is to assume an earlier, transient velopharyngeal incompetency” for children without known palatal anomalies (p. 112).

⁶An example of blurring of the VP port can be seen in a video endoscopy provided by Kummer (2008). In video 17-04, there is blurring (fluttering) of the marginal edges of a small, circular VP port associated with nasal “rustle” of a speaker. Although difficult to see at times due to mucous, flutter is most noticeable at the end of the video when the speaker is counting from “sixty”.

Peterson-Falzone, Hardin-Jones, and Karnell (2010) further state “[...]the variety of patients in whom this phenomenon has been observed certainly indicate that the etiology is heterogeneous” (p. 260). The principle of Occum’s razor, however, states that the fewest assumptions are to be preferred in explaining a phenomenon. As noted above, most authors agree that NFs are learned articulations regardless of the population. Is it possible, then, to identify a common underlying factor that is responsible for faulty learning of NFs in all children with specific triggering events that may vary from child to child?

To help propose a common framework that can account for the occurrence of NFs in children both with and without palatal anomalies, it is useful to review what is known about children who produce NFs. It cannot be denied that this information comes from a single published source and clinical observations. Nevertheless, it appears that children who use NFs also use multiple other phonological processes. Peterson-Falzone and Graham (1990) described patterns of phoneme-specific nasal emission in 19 children without palatal anomalies and 17 children with palatal anomalies. All of the children exhibited either nasal emission or posterior nasal frication, a distinction consistent with anterior and posterior NFs described in this article. Peterson-Falzone and Graham reported that the children in both groups exhibited a wide range of phonological processes with gliding and stopping occurring most frequently overall. It should be emphasized that the two groups of children did not differ in mean number of phonological processes used.

Second, it appears that children who use NFs have histories of frequent middle ear disease. While Peterson-Falzone and Graham (1990) were unable to adequately document early episodes of otitis media in the children of their study, they nonetheless noted “suspicious” histories of middle ear disease in 64% of the children without palatal anomalies and 94% with palatal anomalies. The high rate of middle ear disease in children with palatal anomalies should not be surprising. Indeed, it has long been known that infants with unrepaired cleft palate experience almost universal Eustachian tube dysfunction (e.g., Paradise, Bluestone, and Felder, 1969). Even following palate repair, middle ear disease tends to persist at relatively high levels. Nunn, Derkay, Darrow, Magee, and Strasnick (1995), for example, reported that approximately 70% of children who underwent early palate repair by 1 month of age still required ventilation tubes by 3 years of age. It is also known that approximately half of children without cleft palate will have experienced at least one episode of acute otitis media by 3 years of age (Willging & Kummer, 2008).

Third, children who use NFs tend to have histories of nasal airway obstruction, which may be related to middle ear disease. Unfortunately, Peterson-Falzone and Graham (1990) did not report histories of airway obstruction of the children in their study. A frequent clinical observation of this author, however, is the presence of enlarged tonsils and/or adenoids or histories of tonsillectomy and/or adenoidectomy in children who produce NFs, especially those without palatal anomalies. Zajac and Vallino (2015) also noted that 6 of 12 children with cleft palate who exhibited NFs had cleft lip and palate. It is well known that nasal deformities associated with cleft lip and palate tend to reduce the size of the anterior nasal valve by 20–30% compared to the non-cleft population (e.g., Warren, Duany, & Fischer, 1969). There is also growing evidence to suggest that individuals with cleft palate are more

likely to have obstructive breathing and sleep apnea than non-cleft individuals due to small and narrow airways (e.g., Abdel-Aziz, 2012).

Based upon the above, a model for the development of NFs by children both with and without cleft palate can be proposed with a minimum of assumptions. As illustrated in Figure 5, the model proposes that the common underlying condition in all children is frequent and/or fluctuating conductive hearing loss that drives acceptance of nasal frication as auditory targets for oral fricatives. The model further assumes that either posterior and/or anterior constrictions in the nasal airway cause turbulent airflow that is triggered by either respiratory and/or speech events. If a child has cleft palate, then it is more likely that nasal turbulence will occur during speech that may lead to the development of NFs. The model does not assume that cleft palate (or VP insufficiency), however, is the cause of NFs, only that the two may occur together. As described below, the model is elegant in that it appears to account for the variable expression of NFs in children both with and without cleft palate and leads to testable predictions.

As shown in the model, there are several possible triggering events for the occurrence of nasal turbulence as a precursor to the development of NFs—normal respiration and specific speech behaviors. For any of the triggering events, the model assumes that audible frication is generated at either posterior and/or anterior sites of nasal obstruction. The first triggering event is normal respiration. During exhalation, the presence of enlarged tonsils, enlarged adenoids, and/or a constricted anterior nasal valve may create audible frication that a child with conductive hearing loss may accept as an auditory target for production of oral fricatives. As illustrated in Figure 3, nasal airflow associated with end-of-utterance exhalation is a common phenomenon. The magnitude and duration of this airflow may create nasal turbulence if a constriction is present. Of interest, the speaker in Figure 3 was an 8 year-old girl with enlarged tonsils. While the end-of-utterance exhalation illustrated in Figure 3 was not audible, it was at other times for this speaker. Audible respiratory noise might be more likely to occur in younger children who have smaller airways. One clinical case was a 2-year-old boy with submucous cleft palate who produced posterior NFs. He also produced loud audible noise during both inhalation and end-of-utterance exhalation. In this boy, the respiratory noise was remarkably similar to his posterior NF—a fluttering quality.

Children with repaired cleft lip and palate may be especially susceptible to respiratory turbulence generated by the anterior nasal valve. As part of research project sponsored by the National Institute of Health (NIH) studying the physiologic development of stop consonants, we have observed the onset of anterior NFs in an 18-month-old child with repaired bilateral cleft lip and palate. The mother reported that the child has experienced congestion and difficulty breathing through the nose “since birth.” As part of her participation in the study, we obtained longitudinal nasal ram pressure (NRP) data during respiration and speech every 2 months beginning at 14 months of age. The child’s production of stop consonants at 16 months of age was characterized by incomplete VP closure as evidenced by positive NRP during approximately 50% of stops. At 18 months of age, the child began producing anterior NFs for fricative targets in word final position, often with an accompanying nasal grimace. Figure 6 illustrates the audio, NRP, and spectrogram of the child producing “fish” two times. As shown in the figure, the child exhibited stopping

for the /f/ segments and used anterior NFs for the /sh/ segments. The anterior NFs were produced with excessive NRP, indicating an open VP port. This child also exhibited frication-like noise as part of end-of-utterance exhalation that was perceptually and spectrally similar to the NFs shown in Figure 6.

As further shown by the model, speech behaviors may also trigger nasal turbulence. One behavior might be passive nasal air escape during production of stop consonants due to large tonsils and/or adenoids. Large tonsils may protrude behind the velum and inhibit complete closure. Similarly, large and/or irregular adenoids may prevent complete closure of the VP port (see Figure 7–14, p. 195, Kummer, 2008). If a child has repaired cleft palate, then incomplete—but not necessarily inadequate—VP closure may also trigger passive nasal air escape during production of stop consonants. Indeed, Peterson-Falzone et al. (2006) note that “passive” posterior NFs can be co-produced with any or all of the high-pressure consonants. Passive nasal air escape is an attractive triggering event for several reasons. First, it is known that most children acquire stop-plosives before fricatives. Second, as reported by Peterson-Falzone and Graham (1990), the phonological process of stopping was frequently used by children who produced NFs.

Another speech behavior that might trigger nasal turbulence is frictionalized stops. These are stops produced with a fricative quality due to “failure of stop formation” (Shriberg and Kent, 2003). Shriberg and Kent note that velar stops are often frictionalized due to “sliding” of the tongue against the soft palate. Children who have large tonsils and/or repaired cleft palate might be more likely to frictionalize stops given their structural anomalies.

A final speech behavior that might trigger nasal turbulence is phonological assimilation. Assimilatory processes that simplify word production such as consonant harmony are a hallmark of young children’s speech. One of our graduate students recently worked with a 19-month-old child without cleft palate and no known history of airway obstruction or middle ear disease. The child was developing speech normally and used some phonological processes to simplify word productions. Unexpectedly, the child started to exhibit anterior NFs with a nasal grimace. The NFs occurred only in words such as “snip” involving fricative-nasal sequences—apparently a result of nasal consonant harmony. The child produced all singleton /s/ segments orally. The child experimented with NFs for a brief time and soon produced “snip” and other fricative-nasal sequences normally. Given that this child did not have known hearing loss, it can be hypothesized that she was able to distinguish nasal from oral auditory targets and did not persist in using NFs.⁷

If the above theory is valid, then the following predictions can be made: First, a child may engage in nasal frication but not develop NFs if hearing is adequate; Second, children with structural anomalies such as cleft palate and/or enlarged tonsils and adenoids may be at increased risk for the development of NFs; and Third children with repaired cleft palate might be at increased risk for anterior versus posterior NFs if there is gross VP inadequacy. Obviously, clinical research is needed to test these and other predictions.

⁷Similarly, Harding and Grunwell (1998) noted the resolution of NFs in some but not all young children with repaired cleft palate.

A New Perspective on Nasal Fricatives

If one accepts the above etiologic model, then one must also question the commonly held assumption that NFs—especially posterior NFs—are compensatory to VP inadequacy in children with cleft palate. In the framework of the model, all productions of NFs regardless of palatal status of the child are considered to result from faulty phonological learning. While structural anomalies such as cleft palate have a role in the model, that role is only facilitative. That is, while a child with cleft palate may be more likely to develop NFs than a child without cleft palate, they both develop NFs for the same underlying reason. As stated by Moller (1990), “Perhaps we have focused on the problems of the cleft palate child for too long and have failed to recognize that the child is a normal child trying like any other to master the language, to sound like others” (p. 129).

Finally, the above perspective has diagnostic and management ramifications for children with cleft palate. Obviously, speech therapy is the only ethical treatment for children without cleft palate who exhibit NFs. For children with cleft palate, clinicians need to be especially vigilant and guard against assuming that all cases of NFs, especially posterior, reflect gross VP inadequacy and the need for secondary surgery. To be sure, gross VP inadequacy may exist in some cases and surgery will be needed to eliminate severe obligatory symptoms. Some children with repaired cleft palate who use NFs, however, may have only mild obligatory symptoms in the presence of adequate but not complete VP closure. In these cases, successful speech therapy that eliminates NFs may reduce the overall impact of mild obligatory symptoms and, perhaps, eliminate the need for additional surgery.

Summary

Nasal fricatives are learned, maladaptive articulations for oral fricatives that are produced by children both with and without palatal anomalies. Nasal fricatives vary in articulatory and acoustic-perceptual characteristics with two generally distinct types recognized, posterior and anterior. Both types of NFs are produced with articulatory gestures that completely inhibit oral airflow and distinguish these productions from passive nasal air emission. A model was proposed that accounts for the development of NFs based upon conductive hearing loss and specific triggers that generate nasal turbulence in the presence of airway obstruction. The model appears to account for the occurrence of NFs in all children regardless of palatal status.

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References

- Abdel-Aziz M. Hypertrophied tonsils impair velopharyngeal function after palatoplasty. *The Laryngoscope*. 2012; 122(3):528–532. [PubMed: 22252734]
- Beebe HH. Sigmatisms nasalis. *Journal of Speech Disorders*. 1946; 11:35–37. [PubMed: 21023147]
- Harding A, Grunwell P. Active versus passive cleft-type speech characteristics. *International Journal of Language & Communication Disorders*. 1998; 33:329–352. [PubMed: 10326043]

- Kummer, AW. Cleft palate and craniofacial anomalies: Effects on speech and resonance. 2nd ed.. Clifton Park, NY: Thomson Delmar Learning; 2008.
- Moller, KT. Early speech development: The interaction of learning and structure. In: Bardach, J.; Morris, HL., editors. Multidisciplinary management of cleft lip and palate. Philadelphia, PA: W.B. Saunders; 1990. p. 357-380.
- Morley, ME. Cleft palate and speech. Baltimore, MD: The Williams & Wilkins Company; 1966.
- Nunn DR, Derkay CS, Darrow DH, Magee W, Strasnick B. The effect of very early cleft palate closure on the need for ventilation tubes in the first years of life. *Laryngoscope*. 1995; 105:905–908. [PubMed: 7666722]
- Paradise JL, Bluestone CD, Felder H. The universality of otitis media in 50 infants with cleft palate. *Pediatrics*. 1969; 44:35–42. [PubMed: 5795401]
- Peterson SJ. Nasal emission as a component of the misarticulation of sibilants and affricates. *Journal of Speech and Hearing Disorders*. 1975; 40(1):106–114. [PubMed: 1123917]
- Peterson-Falzone SJ, Graham MS. Phoneme-specific nasal emission in children with and without physical anomalies of the velopharyngeal mechanism. *Journal of Speech and Hearing Disorders*. 1990; 55(1):132–139. [PubMed: 2299829]
- Peterson-Falzone, SJ.; Hardin-Jones, MA.; Karnell, MP. Cleft palate speech. 4th ed.. St Louis, MO: Mosby; 2010.
- Peterson-Falzone, SJ.; Trost-Cardamone, JE.; Karnell, MP.; Hardin-Jones, MA. The clinician's guide to treating cleft palate speech. St Louis, MO: Mosby Elsevier; 2006.
- Proctor, DF. The upper airway. In: Proctor, DF.; Anderson, IB., editors. The nose: Upper airway physiology and the atmospheric environment. New York, NY: Elsevier Biomedical Press; 1982.
- Sell D, Harding A, Grunwell P. GOS.SP.ASS.'98: An assessment for speech disorders associated with cleft palate and/or velopharyngeal dysfunction (revised). *International Journal of Language & Communication Disorders*. 1999; 34(1):17–33. [PubMed: 10505144]
- Shriberg, LD.; Kent, RD. Clinical phonetics. 3rd ed.. Boston, MA: Allyn & Bacon; 2003.
- Trost JE. Articulatory additions to the classical description of the speech of persons with cleft palate. *Cleft Palate Journal*. 1981; 18(3):193–203. [PubMed: 6941865]
- Trost-Cardamone, JE.; Bernthal, JE. Articulation assessment procedures and treatment decisions. In: Moller, KT.; Starr, CD., editors. Cleft palate: Interdisciplinary issues and treatment. Austin, TX: Pro-Ed; 1993. p. 307-336.
- Warren DW, Duany LF, Fischer ND. Nasal pathway resistance in normal and cleft lip and palate subjects. *Cleft Palate Journal*. 1969; 6:134–140. [PubMed: 5253575]
- Willging, MD.; Kummer, AW. Ear, nose, and throat anomalies: Effects on speech and resonance. In: Kummer, AW., editor. Cleft palate and craniofacial anomalies: Effects on speech and resonance. 2nd ed.. Clifton Park, NY: Thomson Delmar Learning; 2008. p. 214-237.
- Zajac DJ, Mayo R, Kataoka R, Kuo J. Aerodynamic and acoustic characteristics of a speaker with turbulent nasal emission: A case report. *Cleft Palate Craniofacial Journal*. 1996; 33:440–444. [PubMed: 8891377]
- Zajac, DJ.; Vallino, LD. Velopharyngeal status of children with cleft palate who produce nasal fricatives; Poster presented at the Annual Meeting of the American Cleft Palate-Craniofacial Association; Palm Springs, CA. 2015 Apr.

Figure 1.

Articulation of Anterior NFs.

From Peterson-Falzone, S. J., Trost-Cardamone, J. E., Karnell, M. P., and Hardin-Jones, M.

A. (2006). *The clinician's guide to treating cleft palate speech*. St Louis, MO: Mosby Elsevier. Reproduced with permission.

Figure 2. Oral Microphone Signal (Top), Nasal Microphone Signal (Middle), and Spectrogram of the Nasal Signal (Bottom) During Production of Anterior NFs Substituted for /s/.

Figure 3. Audio (Top), Nasal Airflow (Middle), and Oral Airflow (Bottom) During Production of Posterior NFs. Complete stoppage of oral airflow occurs during production of posterior NFs.

Figure 4. Oral Microphone Signal (Top), Nasal Microphone Signal (Middle), and Nasal Spectrogram (Bottom) During Production of Posterior NFs Substituted for /s/.

Figure 5.
Theoretical Model for the Development of NFs by Children With and Without Cleft Palate.

Figure 6. Audio (Top), Nasal Ram Pressure (Middle), and Spectrogram (Bottom) During Production of Anterior NFs in Two Productions of “Fish”.