Pharyngeal flow simulations during sibilant sound in a patient-specific model with velopharyngeal insufficiency

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Dysfunction of the velopharyngeal valve in the human airway causes speech disorders because there is no separation between the oral and nasal cavities during normal oral speech. The speech literature hypothesizes that undesired sound is formed by turbulent flow in the nasal cavity in cases of small velopharyngeal openings. The aim is to determine the flow behavior and the sound-generating mechanism in the vocal tract using computational fluid dynamics in two patient-specific models with small and large velopharyngeal openings and contrast it with cases of complete velopharyngeal closure. The geometry for the models was reconstructed from computed tomography scans that were taken while the patients were sustaining a sibilant sound. The results for the turbulence are correlated with the broadband acoustic models of Proudman and Curle. The models show that turbulence in the vocal tract increases downstream of a constriction and that sound may be generated from it. Furthermore, most of the sound due to turbulence in the nasal cavity is governed by a dipole source where turbulence interacts with the nasal cavity walls. The generated sound power by turbulence itself in the nasal cavity (the quadrupole source) is two orders of magnitude less than the dipole source. © 2019 Acoustical Society of America. <https://doi.org/10.1121/1.5108889>

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I. INTRODUCTION

The velopharyngeal (VP) valve is an area in the pharynx that creates a physical barrier between the nasal and oral cavities. Nasal consonant sounds (/m/, /n/, and /ŋ/) are produced with the VP valve open, which results in an acoustic coupling between the oral and nasal cavities. For oral sounds, the degree of coupling between the cavities can vary based on the speech task. However, the production of fricatives typically involves no nasal airflow and thus a complete closure of the VP valve is required.

The term *velopharyngeal insufficiency* (VPI) is typically used to describe velopharyngeal dysfunction due to abnormal structure (Kummer, 2011). VPI occurs most often in children with cleft palate. In fact, an estimated one-third of children with cleft palate demonstrate VPI after cleft palate repair, i.e., 2650 cases per year according to CDC estimates (Parker *et al.*, 2010). Although cleft palate (including submucous cleft) is the major etiology, VPI can also occur for other reasons, including 1 in 10 000 post-adenoidectomy (Saunders *et al.*, 2004) or in some cases due to post-maxillary advancement (Sell *et al.*, 2002; Witzel and Munro, 1977).

VPI causes speech disorders because the VP valve does not adequately separate the oral and nasal cavities during speech. In a retrospective study, Kummer *et al.* (2003) noted that the opening size of the VP port was correlated with the type of speech disorder, but not with the perceived severity of the disorder. The classification of large/small opening size of the VP port was done in a qualitative manner based on

imaging data taken using nasopharyngoscopy. They observed that hypernasality was associated with a large VP opening while nasal turbulence was associated with a small VP opening.

In hypernasality, the distortion of speech is caused by undesired oral-nasal resonance due to the open VP valve. This particularly occurs with a relatively large VP opening and is perceived mainly on vowels and voiced consonants (Kummer *et al.*, 1992). The term “large opening” of the VP port is a qualitative description made by a clinician and typically indicates little to no elevation of the velum during speech. When the VP valve is open, acoustic energy from the glottis is transferred between the nasal and oral cavities and the result is speech with excessive nasal quality.

If flow escapes through the VP valve, there will be nasal emission. Nasal emission is inaudible with a large VP opening and barely audible with a medium-sized opening. With a small VP opening however, nasal emission is not only audible—it can also be very loud and distracting (Kummer *et al.*, 1992, 2003).

The speech disorder that occurs due to the loud and distracting form of nasal emission is commonly known as “nasal turbulence” as it was speculated that turbulent flow is the source of the undesired sound that is produced in the nasal cavity. Furthermore, in the literature for speech (Hixon *et al.*, 2014) and cleft palate speech (Peterson-Falzone *et al.*, 2016), the formation of turbulence and its sound in the nasal cavity is theorized to occur when the flow accelerates through the small VP opening and discharges into an expansion in the nasal cavity. However, because no studies have been done to show that such mechanisms actually exist, this theory remains an untested hypothesis.

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An illuminating example to predict flow behavior in a speech disorder due to VPI is the scenario of turbulence going through an axisymmetric contraction, which serve as an idealized small VP opening. The funneling of the flow from a large to a small cross-section causes the flow to accelerate. Because the mean axial flow component increases in the converging section, the initial velocity fluctuation becomes less significant. Therefore the turbulence intensity within the velopharynx is reduced.

Understanding flow behavior in speech disorders due to VPI is important because sound can be generated from the flow, its turbulence, and its interactions with the boundaries. According to the work by Lighthill (1954), sound sources resulting from flow may be characterized by means of an acoustic analogy. The characterizations are commonly referred to as monopole, dipole, and quadrupole sound sources. As described in Lighthill's theory, turbulence acts as a sound source term in the acoustic analogy. Its acoustic intensity is proportional to the level of turbulence in the flow. The random nature of turbulent flow is identified as a broadband acoustic spectrum. Therefore, turbulent sound is commonly designated as "noise" without any tonal features.

The current study investigates the flow behavior in the upper airways in cases of small and large VP openings due to VPI. The hypothesis for the study is that formation of turbulence in the nasal cavity is higher in the case of a small VPI opening. This turbulence may contribute to the perceived form of distracting nasal emission. The magnitude and spatial distribution of the turbulence are used as inputs to the broadband correlation models, for the purpose of assessing the aeroacoustic properties with respect to the VP opening size. One motivation for the current study is to capitalize on the computed steady-state Reynolds Averaged Navier-Stokes (RANS) data, which provide an input to the broadband acoustic source models. This gives a relatively fast lead time at a feasible computational cost. One possibility is the availability of predictive flow-generated aeroacoustic sources via surface and volume-based correlations. Calculation of the acoustic power on surfaces or in the volume may give valuable insights in the comparison between a small and a large VP opening. In addition, a good insight is given to necessary grid refinements before a subsequent unsteady analysis such as large eddy simulation in order to capture frequency ranges of interest.

II. METHOD

The models in this study were based on an 8-year-old boy who was diagnosed with severe nasal emission (with normal resonance) and a 9-year-old girl who was diagnosed with hypernasality (with no audible nasal emission). Both patients had normal articulation placement. Their speech was diagnosed by a speech-language pathologist (SLP) who specializes in cleft-palate, resonance disorders, and VPI. The presence of a small VP opening for the boy and a large VP opening for the girl was confirmed using nasopharyngoscopy.

For the upper airway geometry rendering in the current model, CT scans were collected while the patients uttered the phoneme /z/. Voicing by the patient was needed to

ensure correct timing of the CT scans. The /z/ phoneme was selected because it is the voiced cognate of /s/, which is the continuant fricative sound where loud nasal emission is most prominent. This study protocol was approved by the Institutional Review Board (IRB) of Cincinnati Children's Hospital and Medical Center.

CT image sequencing was performed with an Aquilion ONE (Toshiba) with spatial capacity of 16 cm³. It provided adequate coverage from the nasal cavity to the laryngeal level. The collection comprised a total of 320 slices with a resolution of 0.5 mm. The scan procedure comprised a rotation of both tube and detector (350 ms) with 100 kVp and mA = 40. Thus, an ultra-low dosing technique was used.

The digital reconstruction of the respiratory (vocal) tract was performed with advanced imaging techniques, including masking and segmentation procedures (Mimics Research, Materialize NV, Leuven, Belgium). The CT images provide good contrast between tissue and air, which is favorable for delineation of the model geometry. The segmentation variability was calculated based on the method of Joskowicz *et al.* (2019) and its highest value of 15% was calculated at the aspect of the VP valve.

The reconstructed volume showed a small VP opening for the patient who was diagnosed with severe nasal emission and a large VP opening for the patient with hypernasality (Fig. 1). For each patient, a theoretical flow behavior for normal production of /s/ sound was predicted by removing the subvolume enclosing the nasal cavity (thus simulating full closure of the VP valve). The modification to the models was done in order to compare the flow behavior between a disordered and a (hypothetical) normal case using the same patient-specific geometry. Using this modification for comparison is considered valid because both patients were diagnosed with normal articulation; it is expected that the shape of their vocal tract during a sibilant sound would remain the same if their velopharynx could close normally.

It is noted that although CT scans were collected when the patient was uttering /z/, the inlet boundary conditions for the model were based on an /s/ sound. A first-line analysis would suggest that the only significant difference for this fricative pair is a vibration component of the vocal fold, which was shown using cineradiography (Subtelny and Oya, 1972).

A stagnation pressure of 6 cmH₂O was employed at the supraglottal inlet, which is extracted from published data for a child subject pronouncing a /s/ sound (Baken and Orlikoff, 2000). An acoustical free stream condition is employed at the outlet where atmospheric pressure prevails. All other boundaries of the model assume no-slip wall conditions. The Reynolds numbers (Re), Mach numbers (Ma), and reference velocities at the inlet, VP valve, and the oral constriction sections for all considered cases are summarized in Table I.

The flow in the model was predicted by solving the compressible RANS equations together with the shear stress transport (SST) *k- ω* equations in the computational fluid dynamics code STAR-CCM+. A full 3D simulation is considered. Since the supply flow is steady, the mean flow is conjectured to only change moderately compared with turbulent scales, which supports the use of Reynolds decomposition. Moreover, the flow

FIG. 1. (Color online) 3D reconstruction of pharyngeal airway from CT scans during sibilant sound. (a) Sagittal view from a subject with small VP opening. (b) Sagittal view from a subject with large VP opening. The bottom left legend shows the axis in which the sagittal plane was sampled. The reconstructed volume geometry and its modification to simulate full VP closure for each case are shown in the insert images. Zoomed-in views of the constrictions at the oral cavity and the VP valve are shown for (c) small VP opening and (d) large VP opening.

in the vicinity of the interesting VP valve area is subjected to thin shear flow, both near the no-slip wall but also in the jet wake region downstream of the VP valve opening. The two equation eddy viscosity model SST $k-\omega$ has been calibrated for these flow scenarios (Menter, 1994).

To determine the spatial localization and strength of the acoustic sources with respect to the pharyngeal airway, the steady-state broadband noise models of Curle (1955) and Proudman (1952) were applied. These methods can investigate turbulence-generated flow noise and how it contributes

TABLE I. Summary of boundary conditions for all considered cases. Reynolds and Mach numbers are indicated. Subscript *in* is the narrow constriction close to the glottal inlet, subscript *VP* is the VP valve constriction, and subscript *oral* is at the narrow oral constriction.

Case	P_{0in} (Pa)	P_{out} (Pa)	$Ma_{oral}(-)$	$Re_{oral}(-)$	U_{oral} (m/s)	$Ma_{in}(-)$	$Re_{in}(-)$	U_{in} (m/s)	U_{VP} (m/s)
Small VP	588.6	0	0.09	3100	30	0.02	1700	5.9	31
Large VP	588.6	0	0.03	1700	10	0.09	9400	29.6	5
Small VP closed	588.6	0	0.09	3300	33	0.01	800	2.8	N/A
Large VP closed	588.6	0	0.09	5100	32	0.03	2900	8.9	N/A

to the acoustic transmission. Both methods are cost-effective since flow field variables were obtained from RANS. In the formulation of these broadband correlation models, there is no mechanism to identify coherent flow structures such as vortex shedding. Moreover, determination of associated frequency information does not apply with these models. The major advantage, however, is that both models can be applied to steady-state flow fields, so simulations are relatively low-cost.

Broadband models based on synthesized fluctuations or stochastic noise generation and radiation are more accurate (Bailly and Juve, 1999; Bechara *et al.*, 1994). Both can account for refraction and convective effects in a sheared mean flow. The most accurate method for the simulation of turbulent flows is direct numerical simulation (DNS). In this approach, all temporal and spatial scales are resolved. Therefore, the computational effort for DNS is very high even at a moderately high Re number due to a very fine grid resolution and large memory requirements.

Computed results were verified using a grid sensitivity study (Fig. 2). Three different grids—coarse, medium, and fine—were considered. The fine grid indicates that the error is less than 5% for an integrated flow property, compared with 30% for the medium grid.

Validation of the models was also done by comparing the computed sound pressure level (SPL) with the SPL computed from nasometry (Nasometer II: model 6450, PENTAX Medical) measurements taken prior to the CT scans as part of

the clinical evaluation of each subject. A sample of the audio recording from the nasal microphone during the SNAP test (Kummer, 2005; MacKay and Kummer, 2005) for each subject during the sequence “Suzy sees...” is attached to this manuscript. The comparison between the predicted and measured SPL values is done after correcting for the fact that the physical location for the audio measurement and the model is different (Lighthill, 1952, 1954). The supplemented multimedia files are referred to as Mm. 1 for the subject with the small VP opening and Mm. 2 for the subject the large VP opening.

Mm. 1. Recorded audio for the subject with the small VP opening. This is a file of type “wav” (179 KB).

Mm. 2. Recorded audio for the subject with the large VP opening. This is file of type “wav” (261 KB).

III. RESULTS

It is realized from the reconstructed geometry in Fig. 1 that the flow must undergo a rather complicated route through a variety of airway passages and cavities. The first bifurcation of the flow is found at the oropharyngeal junction where the flow path divides into an oral cavity and a nasal cavity. Further downstream in the nasal cavity there is a second bifurcation at the back nose openings (choanae) where the nasopharyngeal cavity is separated into left and right passages terminating in nostril openings. The delineate geometry of the nasal tract showed a noticeable difference between the subjects due to the patient-specific anatomy.

The flow simulations provide both qualitative and quantitative details of the flow characteristics in the vocal tract (Fig. 3). The velocity contours indicated with color, show the velocity magnitude normalized by the mass flow averaged velocity at the inlet boundary condition, as given in Table I. Streamlines are overlaid to clarify the flow directionality. Following the Bernoulli principle, the velocity of the flow in the VP valve is higher for the case with the small VP opening [Fig. 3(a)] compared with the large VP opening [Fig. 3(b)]. The size of the oral constriction is about the same size in both models, yet the predicted velocity is lower for the model with large VP opening. The difference stems from the greater oral pressure buildup that occurs in the case of the small VP opening. There is significantly less oral pressure buildup in the large VP opening case because more flow is discharged through the nasal cavity.

When both cases are simulated with full VP closure [Figs. 3(c)–3(d)] the velocity magnitude and distribution is

FIG. 2. Grid dependency study based on the integrated pressure drop from the inlet to the nares. The averaged cell edge length is indicated with h . The normalized length is obtained by $y^+ = yu_\tau/\nu$, where u_τ is the friction velocity at the VP valve and ν is the kinematic viscosity of the fluid. Streamwise and cross-flow directions are indicated with x and z , respectively, and y is the wall normal direction. The error is computed using the extrapolation of Richardson (1911), assuming existence of an infinite grid solution. The error is seen to drop two orders of magnitude for each grid refinement order, $O(x^{+2})$ is indicated with the dashed line.

FIG. 3. (Color online) Velocity magnitude distribution with superimposed streamlines. (a) Small VP opening. (b) Large VP opening. (c) Same model as (a) with the nasal cavity removed (thus simulating full VP closure). (d) Same model as (b) with the nasal cavity removed.

fairly similar. The remaining differences stem from the small variations in the patient-specific geometries of the vocal tracts. Another interesting observation is the effect of simulating full VP closure on the inlet velocity of both cases; for the small VP opening there is little change in the Re_{in} , while this value drops by more than 60% for the case large opening. The change in Re_{in} stems from the significant change in pharyngeal flow resistance difference. The velocity contours also show that in both cases of normal and disorder sibilant, a large circulation zone is predicted in the oropharyngeal cavity. Some of the delivered flow from the supraglottal level is thus trapped in this zone and being subjected to a longer residence time.

The predicted turbulence behavior is shown using turbulent intensity (Fig. 4). Turbulence intensity is defined as $\sqrt{2k/3}/U_{in}$, where U_{in} is the mass flow averaged inlet velocity and k is the turbulent kinetic energy. In the case of the small VP opening, the behavior of the turbulence intensity is markedly different from the velocity distribution, specifically around the area of the VP valve. Unlike the velocity contour where the highest magnitude occurs within the VP valve, the turbulence intensity reaches its maximum in the distal section of nasopharyngeal cavity [Fig. 4(a)]. Moreover, turbulence intensity is actually reduced inside the VP valve. After it reaches a maximum, the turbulence slowly decreases towards the nostrils. The increase in turbulence for the small VP opening case stems from the jet wake that develops in the nasopharyngeal cavity. By contrast, such a wake does not develop in the case where there is a large VP opening because the expansion of the flow into the nasopharyngeal cavity is not sudden [Fig. 4(b)]. In both models, a jet

wake develops near the supraglottal region. The supraglottal recirculation zone also locally increases the turbulence intensity level in this region, giving rise to associated flow losses. Just like the velocity distribution, the turbulence intensity is distributed similarly for both VP cases when a full VP closure is simulated [Figs. 4(c) and 4(d)]. The highest level of turbulence is predicted just past the oral constriction and the difference in magnitude stems from the slight geometrical differences.

Profiles of the velocity and the Reynolds shear stress component are extracted in the lateral-sagittal plane to further characterize the jet-like behavior of the pharyngeal flow (Fig. 5). The profiles are extracted in the oropharynx (VP_{in}), at the narrowest constriction of the VP valve (VP_{valve}), and the nasopharynx (VP_{out}). The approximate locations for these profiles are marked in Fig. 1. For the smaller VP opening case, the streamwise velocity component gradually increases into the VP opening. The velocity reaches a maximum at the point of minimum VP valve cross-sectional area [Fig. 5(a)]. From the minimum area of the VP valve, the flow must diffuse into a larger cross-sectional area and hence the velocity magnitude decreases. The jet-like behavior is also shown by the increased magnitude of the Reynolds shear stress of the nasopharyngeal flow [Fig. 5(c)]. By contrast, the velocity profile for the larger VP opening case shows a fairly constant bulk magnitude [Fig. 5(b)]. This is because there is less change in the cross-sectional area and no jet wake development downstream of the VP valve. In terms of the shearing Reynolds stress, a larger peak is seen coinciding with the supraglottal jet wake [Fig. 5(d)]. This feature is more suppressed for the small VP opening case

FIG. 4. (Color online) Turbulent intensity distribution. Same key for figures as Fig. 3.

[Fig. 5(c)], which on the other hand shows a larger stress level in the nasopharyngeal jet wake.

Flow data are correlated with broadband acoustics using the Curle's surface integral model (Fig. 6). The Curle model

is based on Lighthill's acoustic analogy to give information about contribution of the solid wall to the sound. Simply stated, the Curle model predicts acoustic contribution from dipole-like sources (e.g., from the flow interacting with a

FIG. 5. Streamwise velocity normalized with the VP valve bulk axial velocity. The Reynolds shear stress component is normalized with the friction velocity u_τ . (a) and (c) small VP opening, $u_\tau = 1.4$ m/s. (b) and (d) large VP opening, $u_\tau = 0.9$ m/s. See Fig. 1 for line profile locations.

FIG. 6. (Color online) Surface acoustic pressure with Curle's correlation model. Same key for figures as Fig. 3.

wall boundary). Higher acoustic power is generated where the jet wake interacts with tissue wall and in areas subjected to larger velocity gradients adjacent to the viscous no-slip wall [Figs. 6(a) and 6(b)]. This is because the flow in those locations are subjected to higher wall shear stress, which is a component influencing Curle's equation.

Using Curle's surface integral model on the modified baseline geometries to produce a normal /s/ sound show that the areas generating acoustic power are now shifted towards the oropharyngeal cavity as well as to the mouth opening [Figs. 6(c) and 6(d)], which is expected for the /s/ sound.

Another way to predict acoustic data based on the flow simulations is using Proudman's model (Fig. 7). Proudman's model uses acoustic analogy to predict the acoustic volume sources based on the assumption that the acoustic power scales with the turbulent kinetic energy. Simply stated, the Proudman model predicts acoustic contribution from quadrupole-like sources (e.g., interactions within the flow). Areas with inflection points in the flow, as well as in a recirculation zone (e.g., in the oropharyngeal cavity), generate more acoustic power, because the flow in those locations are subjected to higher turbulent kinetic energy. The elevated areas of the acoustic pressure are primarily seen in three locations for the small VP opening case. At the supraglottal level after the inlet, in the nasopharyngeal cavity jet wake, and where the tongue and the teeth form a small opening [Fig. 7(a)]. For the larger VP opening, most of the acoustic power is generated at the supraglottal level [Fig. 7(b)], which corresponds to the turbulent kinetic energy distribution in Fig. 4(b).

Using Proudman's model on the cases simulating full VP closure yields a drastic change of the generated acoustic power distribution. For the case with a small VP opening, closing the VP valve eliminates the jet wake in the

nasopharynx, and the circulation zone in the oropharynx is reduced. In both cases, a more typical sound distribution for a (voiceless) sibilant sound emerges where most of the acoustic power is generated at the mouth opening [Fig. 7(c) and 7(d)].

Integrating the distribution of the acoustic power based on both surface and volume models (Figs. 6 and 7) shows that contributions from dipole sources is two orders of magnitude higher than contributions from quadrupole sources (Table II). According to the current data, the majority of the sound is therefore generated by the flow interacting with solid boundaries (e.g., cavity walls, tongue, teeth, etc.). The results also show that if a small VP opening is formed during a normal "s" sound, the dipole source contribution decreases, but the quadrupole contribution is increased. The increase in the quadrupole contribution is directly related to the increase in turbulent flow in the nasal cavity. The decrease of the dipole source contribution correlates with loss of pressure buildup in the pharyngeal and oral cavities because of flow leakage through the VP opening.

For the large VP opening case the loss of pressure is more gradual since the constriction size is larger. It is identified that the product of the wall shear stress and the turbulent velocity that approximates the mean-square time derivative of the source surface pressure in Curle's equation is larger with the large VP opening. This correlates with a higher Reynolds number for the larger VP opening case (recall Table I) and motivates an increased dipole source contribution compared to the small VP opening case. The values within parenthesis in Table II are the integrated sound power level for the "s" sounds obtained from the nasometry measurements for the sequence "Suzy see" during the speech tasks for sibilant sounds. The difference is 1 dB between the numerical and the experimental data for both patients.

FIG. 7. (Color online) Volume acoustic pressure with Proudman's correlation model. Same key for figures as Fig. 5.

IV. DISCUSSION

The models in the current study identify flow behavior during the production of sibilant sound in patients who were diagnosed with speech disorders due to VPI. The results show how in a case with small VP opening, the turbulence increases in the nasal cavity due to the sudden cross-sectional expansion (and not due to the flow acceleration). For the case with the larger VP opening, the results confirm that turbulence does not develop in the nasal cavity; thus, a minimal contribution for the undesired sound in the nasal cavity is coming from the flow.

The main finding from the current study is that in case of a small VP opening, the majority of the sound is generated by dipole sources as the flow interacts with a solid boundary. The formation of sound from turbulence (i.e., quadrupole source) exists, but is minimal in comparison to the magnitude of the dipole source. Larger contributions from such dipole sources were also predicted by Shadle (1991) and Krane (2005) for sibilant sounds during normal speech.

The term “nasal turbulence” suggests that the undesired sound that is formed in the nasal cavity is produced by the

turbulence in the flow. The results of the current study confirm that turbulence in the nasal cavity indeed generates sound and also further show that the actual mechanism that produces the sound is the turbulence interacting with the solid nasal walls (as opposed to sound generated within the airflow).

V. LIMITATIONS

There are limitations with broadband acoustic correlation models. Neither the model of Proudman or Curle indicates the frequency of the generated noise. The noise associated with large-scale (or resolved) flow features, such as vortices, is not subordinate to this category and is therefore inappropriate to be quantified by these correlations. The steady-state RANS methodology is limited to single sibilant sounds instead of compound speech. In order to quantify the relationship in the perceptual turbulence intensity of nasal emissions, it is natural to transition towards a dynamic simulation that can simulate different types of the speech sounds, e.g., fricative and plosive.

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TABLE II. Surface and volume integrated acoustic power from the Curle and Proudman models. Comparison with nasometry data for the small and large VP opening cases are given within the parenthesis.

	Small VPI	Small VPI, closed	Large VPI	Large VPI, closed
Curle [dB]	40	38	47	41
Proudman [dB]	−8	−8	−9	−9
Sum [dB]	40 (39)	39 (38)	47	41

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