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Glottal source analysis of voice deficits in basal ganglia dysfunction: evidence from de novo Parkinson's disease and Huntington's disease

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Abstract

Dysphonia is a common speech disruption in people with Parkinson's (PD) and Huntington's (HD). Though the glottal source analysis (GSS) yielded promising results in PD, no study analyzed utility of the GSS in HD. In addition, the potential GSS sex-dependency remains unknown. This study examines sustained vowel phonations provided by 40 PD, 40 HD and 40 age- and sex-matched healthy participants using six GSS features including normalized amplitude quotient, quasi-open quotient, magnitude difference of first two spectral peaks, harmonic richness factor, maximum dispersion quotient (MDQ), and peak slope. Our results showed significant differences in HD men and women compared to the healthy counterpart, suggesting breathiness ($p < 0.01$), tension ($p < 0.001$), and decreased timbre ($p < 0.01$) in HD. Reported sex-related differences highlighted the sensitivity of the GSS towards the speaker's sex. The correlation analysis revealed significant relationship between disease severity and MDQ in HD men.

Index Terms: Glottal source analysis, Basal ganglia, Huntington's disease, Parkinson's disease, pathological voice

1. Introduction

The neurodegenerative disorders affecting basal ganglia, including Parkinson's disease (PD) and Huntington's disease (HD), have a profound effect on the quality of a patient's life [1], [2]. Because the basal ganglia are the critical element in voluntary motor planning, the damage results in a severe disruption of motor control, with disease specific alterations. In particular, PD is characterized by hypokinesia, bradykinesia, and rigidity, whereas choreatic moves and dystonia represent HD [1], [2].

With the expected population aging, the increase in prevalence of neurological disorders predicts a significant increase in society's burden. The resulting need for more effective disease management further underlines the need for early markers of neurodegenerative diseases. The current push for biomarkers reflecting early disease development as well as therapy effect puts forward digital technologies and brings new sources of information. The speech assessment seems to be one of the most promising areas enabling for robust and sensitive and yet non-invasive, easy-to-administer, cost-effective and largely scalable approaches [3].

Similarly, as the gross movement manifestations, speech inpairment in PD leads to hypokinetic dysarthria, and HD results in hyperkinetic dysarthria [4], [5], [6]. Hypokinetic dysarthria manifests by reduced vocal loudness, flattened loudness, pitch inflections, poor voice quality, variable and frequently increased speech rate, inappropriate silences, and breathiness [7]. Hyperkinetic dysarthria in HD demonstrates excess loudness and pitch variations, voice arrests, slow speech rate, inappropriate vocal noises, and intermittent breathy segments [7]. Interestingly, both dysarthric profiles include dysphonia as one of the hallmarks. However, it is hard to separate the impact of disrupted vocal fold control, disrupted movement of respiratory muscles, and dysregulation of upper articulators on phonation [7].

In recent years, the glottal source (GSS) analysis showed promising results in description of pathological voice [8], [9], [10], [11]. In the hypokinetic dysarthria the classification of de-novo PD and healthy speakers reached the area under curve (AUC) = 0.78 [10], and the implantation of glottal source analysis into the end-to-end systems led to $2 - 3\%$ improvement in classification performance [8]. The GSS method is based on the source-filter concept, with the vocal tract acting as a serial connection of resonator tubes and the vocal folds providing the source signal [12]. Using inverse filtering, the GSS method can separate the transfer function of the vocal tract and provide more direct insight into the glottal signal formation [12]. Therefore, the primary benefit of GSS analysis is the least acoustically disturbed insight into the vocal fold function, which may reveal distinct disruption patterns characteristic of different disorders.

Nonetheless, the GSS analysis has been currently applied only in the description of PD [8], [9], [10], and to the best of our knowledge, no studies focused on evaluation GSS characteristics in the hyperkinetic dysarthria of HD. Therefore, the main aim of the study was to assess the utility of GSS analysis in Huntington's disease. The second goal was to compare GSS in drug-naïve de-novo PD and HD as representatives of different dysarthric profiles. Moreover, because the phonatory characteristics are highly sex dependent, the third goal was to evaluate the sex dependency of GSS analysis performance.

2. Methods

2.1. Subjets

A total number of 120 Czech native speakers was recruited for the GSS analysis, including subgroups of PD, HD, and healthy control (HC) participants.

The PD group consisted of 40 (20 women) patients, with an average age of 50.4 ± 9.1 standard deviation (SD) (range 34 – 69) years. The recordings were obtained at the time of diagnosis and before the initiation of pharmacological treatment. The reported average symptom duration was 2.2 ± 1.5 SD (0.4 - 5.7) years. The diagnosis was performed according to the Movement Disorder Society clinical diagnostic criteria for PD [13]. The disease severity was estimated using the Unified Parkinson's Disease Rating Scale motor part III (MDS-UPDRS III) [14], reaching mean score of 28.8 ± 12.4 SD (6 – 56). Speech severity assessment was based on MDS-UPDRS III speech item 3.1, reaching an average score of 0.5 ± 0.5 SD (0 – 1).

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The HD group consisted of 40 (20 women) patients, with an average age of 49.5 ± 12.9 SD (25 – 69) years and a average disease duration of 5.5 ± 3.6 SD (1 – 16) years. The diagnosis was confirmed by genetic testing, with the average number of CAG triplets 44.3 ± 13.7 SD (40 - 51). Most of the patients were treated with monotherapy or a combination of benzodiazepines, antipsychotics, amantadine, and antidepressants. The disease severity was estimated using the Unified Huntington's Disease Rating Scale (UHDRS) [15], reaching mean score of 27.8 ± 12.6 SD (8 – 54). Speech severity assessment was based on UHDRS speech item, reaching an average score of 0.9 ± 0.4 SD (0 – 2).

The HC group consisted of 40 (20 women) speakers with an average age of 50.2 ± 12.0 SD (25 – 68) years. None of the HC participants had any history of neurological or speech disorders.

The study was approved by the Ethics Committee of the General University Hospital in Prague, Czech Republic, and all participants provided written informed consent. The data are not publicly available due to their containing of information that could compromise the privacy of study participants.

2.2. Recording

The recording was performed in a quiet room with low ambient noise using a head-mounted omnidirectional condenser microphone (Beyer-dynamic Opus 55, Heilbronn, Germany). The microphone was placed approximately 5 cm from the corner of the participant's mouth. The utterances were sampled at 48 kHz with 16-bit quantization. The recordings were obtained as a part of a more extensive speech examination protocol performed by a speech specialist in one session. During the examination, participants were asked to complete a sustained vowel /a/ twice at comfortable loudness and pitch. Participants were asked to perform sustained phonation on one breath for as long and as steady as possible.

2.3. Acoustic Analysis

The principal scheme of the glottal source analysis is depicted in Figure 1. During the GSS analysis, the recorded signal was split into 25 ms windows with a 5 ms window shift. Each window underwent the pitch asynchronous inverse adaptive filtering (IAIF). The transfer function of the vocal tract was predicted, and the inversion of the resulting 2-pole filter model was applied to the original signal. The resulting signal was used to more accurately predict the higher-order vocal tract transfer function, which was again applied to the original signal [16]. Parallelly, the glottal closure instants (GCI), defined as a transition between open and closed vocal fold phases, were detected.

The filtered signal representing the glottal flow, the glottal flow derivative, and the GCIs were used in the feature extraction step, during which the time and frequency domain parameters were extracted. The time domain was represented by the quasi-open quotient (QOQ), a ratio between quasi-open time and quasi-closed time of the glottis [17], and the normalized amplitude quotients (NAQ) a ratio between the maximal glottal flow amplitude and the maximal negative amplitude of the glottal flow derivative [18]. The frequency domain parameters were represented by the difference in magnitudes of the first two spectral peaks (H1H2) [19] and the harmonic richness factor (HRF), a ratio between the energy of all higher-order harmonics and the energy of fundamental frequency [20]. Additionally, two GSS parameters estimated directly from the original voice signal were included. These parameters included Maximum

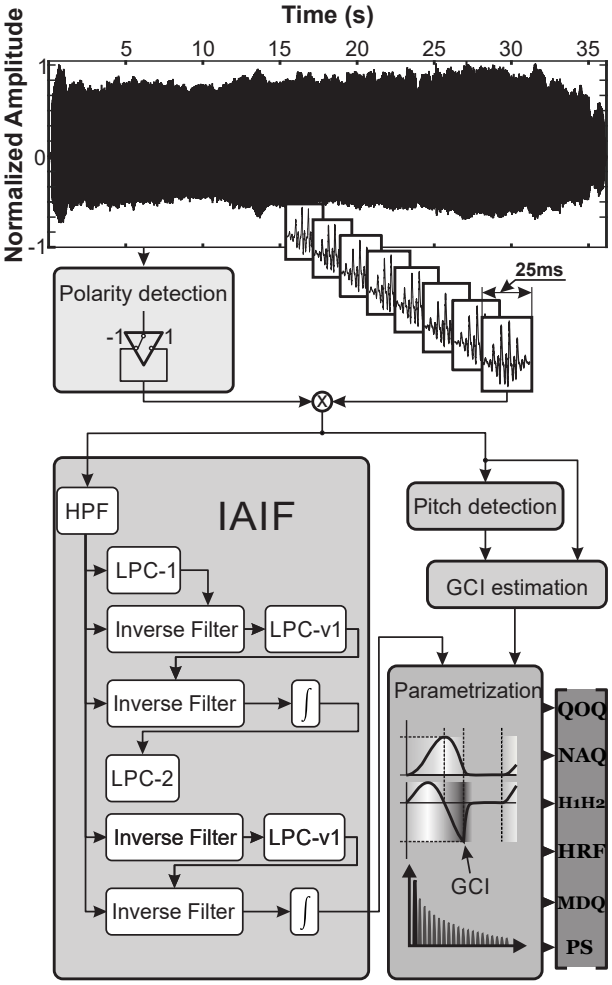

Figure 1: *Schematic diagram of speech glottal source analysis. IAIF =inverse adaptive filtering, HPF= high-pass filtering, LPC = linear predictive coding, GCI = glottal closure instants, NAQ = normalized amplitude quotient, QOQ = quasi-open quotient, H1H2 = difference of magnitudes of first two spectral peaks, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope. Adapted from [10].*

Dispersion Quotient (MDQ) and Peak Slope (PS). The MDQ captures the abruptness of vocal fold closure and is estimated by wavelet decomposition of linear predictive coding residual [21]. The PS parameter reflects the spectral slope of the GSS and is defined by a best-fitting regression line between maximal amplitudes in octave bands after wavelet decomposition [22].

2.4. Statistics

As participants provided two realizations of sustained phonations, an average value of both was used in a subsequent statistical analysis.

As the Anderson-Darling test of normality revealed non-normal distributions, the assessment of group differences was performed by Kruskal-Wallis one-way analysis of variance with post hoc Wilcoxon rank sum test assessment. Similarly, the relationships between GSS parameters and clinical assessment (UHDRS score, or MDS-UPDRS III score), were analyzed using the nonparametric Spearman's correlation.

3. Results

Results of the Kruskal-Wallis analysis for HC, PD and HD are listed in Table 1 for mixed sexes, Table 2 for women, and Table 3 for men. With regards to the mixed sexes, only peak slope parameter showed a significant group difference (PS: $\chi^2(2, 119) = 22.04; p < 0.001$). Similarly, the women subgroup revealed only differences in the peak slope parameter (PS: $\chi^2(2, 59) = 19.25; p < 0.001$). Finaly, the men subgroup revealed significant difference in quasi-open quotient (QOQ: $\chi^2(2, 59) = 7.93; p < 0.01$), harmonic richness factor (HRF: $\chi^2(2, 59) = 7.09; p < 0.01$), maximum dispersion quotient (MDQ: $\chi^2(2, 59) = 7.30; p < 0.01$), and peak slope (PS: $\chi^2(2, 59) = 6.17; p < 0.05$). Based on the the Wilcoxon rank-sum test study revealed statistical differences between HC vs. HD or PD vs. HD groups. The Wilcoxon rank sum analysis of the four GSS parameters reaching the significance level in at least one sub group are depicted in Figure 2 for women and Figure 3 for men. As the box plot with mixed sexes did not reveal additional significant GSS parameters only figures for separate genders are provided. The correlation analysis revealed a significant moderate correlation between MDQ and total UHDRS score in the Huntington's disease men subgroup with $r = 0.46$ and $p < 0.05$.

4. Discussion

The current study shows the utility of glottal source analysis in the objective acoustic assessment of voice pathology, especially in Huntington's disease. The glottal source characteristics obtained using an IAIF method revealed significant differences in phonation between women and men diagnosed with HD. Contrary, even though the analysis of PD shows some trends , especially in men, no significant differences were observed. Interestingly our study showed different dysphonia patterns in HD men compared to HD women.

Previous studies describe the voice in HD as harsh and tense, with increased pitch breaks and fluctuations. Recent objective acoustic analysis showed significantly distorted voice periodicity, irregular vibration of vocal folds, increased perturbations, and increased noise [23]. In accordance with the literature, the increased PS parameter suggests considerably increased tension in the voice. Moreover, in the men subset, the presence of increased tension in voice is supported by an increase in the QOQ parameter, while the MDQ suggests breathy quality, and decreased HRF indicates a decrease in voice timbre in HD men. The correlation analysis showed that the MDQ in men correlates (r = 0.46) with the disease severity represented by the overall UHDRS score.

Even though previous studies aimed at GSS analysis in PD showed promising results with AUC ranging between 0.71 to 0.97 [8], [10], [11], [24], the results of our analysis only suggested possible trends in the men speakers and did not reveal any statistically significant differences. There are a few possible reasons behind the discrepancy. First, the PD participants in our dataset are relatively young with predominantly early-

Table 1: *Results of the Kruskal-Wallis analysis for mixed sexes. NAQ = normalized amplitude quotient, QOQ = quasi-open quotient, H1H2 = difference of magnitudes of first two spectral peaks, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope.*

GSS	HC median (MAD)	PD median (MAD)	HD median (MAD)	KW χ^2(2,119) (p)
QOQ	0.52 (0.07)	0.53 (0.07)	0.48 (0.07)	4.45 (0.11)
NAQ	0.12 (0.02)	0.14 (0.03)	0.13 (0.03)	1.92 (0.38)
H1H2	6.77 (2.98)	6.86 (3.19)	6.49 (3.01)	0.77 (0.68)
HRF	20.92 (5.44)	19.71 (5.26)	19.98 (4.50)	0.85 (0.65)
MDQ	0.08 (0.01)	0.09 (0.02)	0.09 (0.01)	2.85 (0.24)
PS	**-0.54 (0.01)**	**-0.53 (0.01)**	**-0.51 (0.02)**	**22.04 (< 0.001)**

Table 2: *Results of the Kruskal-Wallis analysis for women. NAQ = normalized amplitude quotient, QOQ = quasi-open quotient, H1H2 = difference of magnitudes of first two spectral peaks, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope.*

GSS	HC median (MAD)	PD median (MAD)	HD median (MAD)	KW χ^2(2,59) (p)
QOQ	0.47 (0.05)	0.51 (0.05)	0.47 (0.05)	2.51 (0.29)
NAQ	0.13 (0.02)	0.16 (0.03)	0.14 (0.03)	4.98 (0.08)
H1H2	7.07 (2.79)	7.80 (2.74)	5.80 (3.65)	1.24 (0.53)
HRF	16.06 (2.29)	15.9 (1.46)	16.87 (2.74)	4.24 (0.12)
MDQ	0.08 (0.01)	0.09 (0.01)	0.09 (0.02)	1.68 (0.43)
PS	**-0.54 (0.01)**	**-0.54 (0.01)**	**-0.51 (0.02)**	**19.25 (< 0.001)**

Table 3: *Results of the Kruskal-Wallis analysis for men. NAQ = normalized amplitude quotient, QOQ = quasi-open quotient, H1H2 = difference of magnitudes of first two spectral peaks, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope.*

GSS	HC median (MAD)	PD median (MAD)	HD median (MAD)	KW χ^2(2,59) (p)
QOQ	**0.57 (0.06)**	**0.53 (0.07)**	**0.48 (0.06)**	**7.93 (< 0.01)**
NAQ	0.12 (0.02)	0.11 (0.03)	0.13 (0.02)	1.72 (0.38)
H1H2	6.12 (3.19)	5.97 (3.53)	6.83 (2.42)	0.01 (0.99)
HRF	**27.23 (3.00)**	**26.24 (3.04)**	**23.69 (3.21)**	**7.09 (< 0.01)**
MDQ	**0.07 (0.01)**	**0.07 (0.02)**	**0.08 (0.01)**	**7.30 (< 0.01)**
PS	**-0.54 (0.02)**	**-0.54 (0.02)**	**-0.53 (0.02)**	**6.17 (< 0.05)**

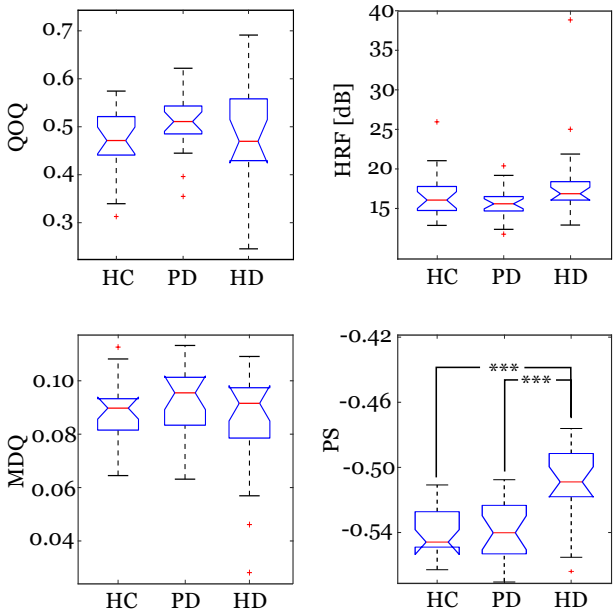

Figure 2: *The box plot of the women subgroup's four most sensitive GSS parameters. Asterisks denote statistically significant differences:* $***p < 0.01$. *QOQ = quasi-open quotient, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope.*

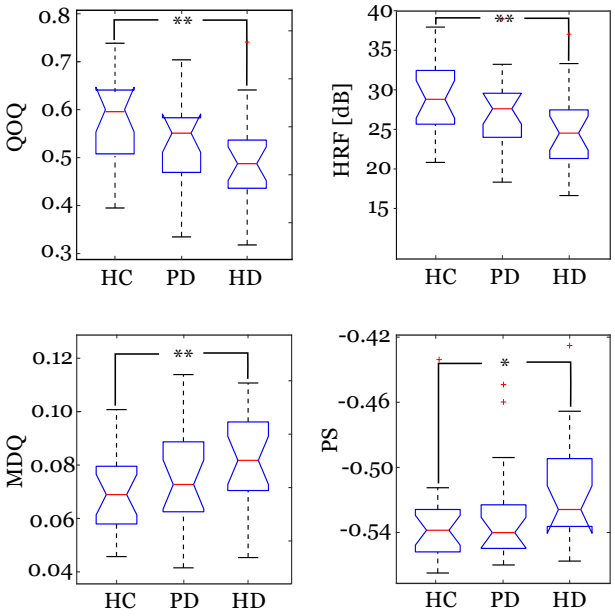

Figure 3: *The box plot of the men subgroup's four most sensitive GSS parameters. Asterisks denote statistically significant differences:* $**p < 0.01, *p < 0.05$. *QOQ = quasi-open quotient, HRF = harmonic richness factor, MDQ = maximum dispersion quotient, PS = peak slope.*

onset of disease. The recent study comparing early-onset and late-onset PD speakers showed that phonatory disturbances are present mainly in the late-onset PD phenotype [25]. Moreover, our analysis was aimed at the drug-naïve de-novo PD patients; therefore, we may assume that the phonatory disturbances will be less pronounced in these earlier disease stages. Nonetheless, the trends achieved by our study are like those reported previously on 80 de-novo PD men [10].

With regards to the sex-specific patterns, the HD women showed only one affected marker, i.e., PS. Nonetheless, the between-group difference was the most apparent, reaching $\chi^2(2, 59) = 19.25, p < 0.001$, and suggesting a shift towards tense voice quality. In comparison, the HD men showed four statistically significant GSS markers, including QOQ, HRF, MDQ, and PS, yet with the Kruskal-Wallis test results ranging between $\chi^2(2, 59) = 7.30, p < 0.01$ and $\chi^2(2, 59) = 6.17, p < 0.05$, the GSS markers in men did not reach the discriminative power of PS in women. Results obtained in the HD men subgroup, compared to women, suggest the presence of more but less pronounced dysphonia manifestations, including breathiness, tension, and a decrease in timbre. Overall presented results highlight high sex dependency of the GSS analysis in dysarthria evaluation, which agrees with previously published literature [26], [27].

Certain limitations of our study must be mentioned. The first limitation is the absence of more direct observation, such as laryngoscopy or electroglottography, connecting certain physiological phenomena with the acoustic measurements. Even though our results are in accordance with previously published literature, the direct observation would provide important insight, clarify possibly misleading aspects, and explain high variability in GSS parameters. Another possible way could be implementing synthetic pathological voice, which would enable a direct model of certain pathological aspects and observe how GSS parameters reflect them and whether there are possible confounding interactions [28]. The second limitation is that the sex-dependent differences may be caused by physiological differences between both sexes but also by sex-related differences in the disease manifestations. Therefore, by separation of those two aspects, the analysis may deepen understanding of the disease pathophysiology and increase the utility of the GSS.

5. Conclusions

In our study, we have presented statistically significant phonatory disruption in the HD group, demonstrating the possible utility of GSS parameters for the detection and description of hyperkinetic dysarthria-related dysphonia. With regards to PD, we observed only nonsignificant trends suggesting a lesser amount of phonatory disruption early disease stages. The between-sex comparison showed remarkable differences, which may have a confusing effect on voice pathology assessment and highlights the need for separate or at least well-sex-matched datasets.

6. Acknowledgements

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