

Vibratory Characteristics of Diplophonia Studied by High Speed Video and Vibrogram Analysis

Peak Woo, New York, New York

Summary: Diplophonia can occur in patients with polyps, atrophy, paralysis, or scars. Its vibratory patterns have not been well characterized. High-speed video (HSV) analysis can contribute to their understanding. Twenty subjects with a diplophonic voice quality were studied by HSV. Diplophonia was due to medical causes including vocal fold paresis (n = 7), vocal atrophy (n = 5), polyps (n = 5), and scars/sulci (n = 3). The HSV was analyzed using a multislice digital videokymography (DKG). The DKG tracing was analyzed qualitatively and then transformed into a vibrogram waveform signal for frequency analysis.

Results. Vibratory abnormalities seen on HSVs explained the diplophonia. Subharmonics to the fundamental frequency can be visualized by DKG. None could be resolved by stroboscopy. One can stratify diplophonia as symmetric or asymmetric based on the involvement of one or both vocal folds. Scars and atrophy showed symmetric subharmonic production with ectopic beats every 4–10 beats. Some subjects showed anterior and posterior independent vocal fold oscillators. Asymmetric causes of diplophonia are common in patients with paralysis. Two different oscillation frequencies of each vocal fold generate in and then out of phase interaction between the two sides. Vibrogram analysis documents the frequent presence of interharmonic energy peaks above the dominant fundamental frequency. Eighteen of the 20 subjects have obvious subharmonic peaks.

Conclusion. Patients with diplophonia have vibratory abnormalities arising from the vocal folds. HSV and vibrogram analysis followed by frequency analysis of the vibrogram can resolve vibratory abnormality into symmetric versus asymmetric causes and can document the type of vibratory abnormality.

Key Words: Diplophonia–Dysphonia–High-speed video–Vocal folds–Diagnosis.

INTRODUCTION

Diplophonia was first imaged by Ward and coauthors in a high-speed cinema of a single female with diplophonia. It showed two distinct oscillators with each vocal fold vibrating at a different frequency.¹ Two separate oscillators with quasiperiodic variations in the vocal fold vibration can result in the acoustic perception to two tonal qualities.

The condition of diplophonia is often transient and difficult to capture by high-speed imaging. Stroboscopy cannot resolve this issue. This is because stroboscopy depends on an acoustic filter to derive the fundamental frequency to time the strobe light. In diplophonia and chaotic vibration, this is not possible. It may occur at the onset of phonation before the steady state of phonation has been reached.² It is often ascribed to transient tension differences between two folds and thus resulting in vibration differences. Diplophonia can be observed in patients with unilateral vocal fold paralysis, vocal fold atrophy, vocal fold scars, and mass lesions.^{3,4} Acoustic analysis often shows a subharmonic of the fundamental frequency with the subharmonic strongly correlated with the perception of diplophonia. Despite this, some subjects with perceived diplophonia do not have acoustic subharmonics.⁵ Some authors have questioned whether the gen-

eration of subharmonics is commonly associated with asymmetrically oscillating vocal folds as the synthetic elastic vocal fold model can show diplophonia with symmetrical oscillations.⁶

Visualization of vocal fold vibration in patients during the production of diplophonia has been limited. This is because it requires a high-speed video (HSV) or kymography. As early as 2000, HSV and kymography were proposed as useful tools for the study of diplophonia and tremors.⁷ Since then, only a few anecdotal reports of biphonation or vibratory irregularities have been reported.^{8,9} In a large collection of patients with diplophonia studied by HSVs, some authors proposed an audio signal-processing algorithm for the detection of diplophonia but did not define the pattern of vibratory abnormalities in their patients with diplophonia.¹⁰

It is believed that diplophonia is the result of vibratory anomalies occurring at the vocal folds. These patterns may be due to vocal fold interactions between the normal and the pathological side (ie, vocal fold paralysis) or between the normal and abnormal portions of the vocal fold (ie, vocal fold scar). It is believed these vibratory anomalies occur with different patterns and may occur with the pathological involvement of vocal functions. These patterns may result in the acoustic perception of diplophonia. Different patterns of diplophonia and the study of these patterns may contribute to clinical care.

The purpose of this study is to use HSVs in diplophonia due to different pathological conditions. We propose to analyze those HSV images using objective imaging methods that include multislice digital kymography, vibrogram analysis, waveform extraction of the vibrogram, and fast Fourier transformation (FFT) signal analysis of the vibrogram waveform. In this way, we feel we can gain a better understanding of this poorly understood condition that contributes to dysphonia.

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From the The Department of Otolaryngology Head and Neck Surgery, Icahn School of Medicine, New York, New York.

Address correspondence and reprint requests to Peak Woo, Department of Otolaryngology Head and Neck Surgery, Icahn School of Medicine, 300 Central Park West, 1-H, New York, NY 10024. E-mail: peakwoo@peakwoo.com

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We hypothesize the following: (1) different diplophonia patterns are present and are dependent on the pathological involvement of vocal vibratory functions; (2) perceptual acoustic characteristics of subharmonics are due to phase interactions or vibratory interaction characteristics of the vocal folds at a higher-order frequency; and (3) the study of the spectral characteristic of the vibrogram derived from HSVs can characterize the vibrogram type by vocal pathology.

MATERIAL AND METHODS

Twenty subjects with diplophonia due to a variety of vocal pathologies were evaluated by stroboscopy and then with HS. All the subjects presented for an evaluation of dysphonia between 2015 and 2016. Diplophonia is defined here as an involuntary perceptual quality that is troublesome to the patient and is audible to the examiner (PW) as a rough low-frequency doubled sound simulating either a strong subharmonic to the fundamental frequency or two fundamental frequencies being produced simultaneously. None of the patients could switch between the diplophonia quality and a “normal” voice. No attempt to quantify or analyze the acoustic signature was done. Diplophonia had medical causes that included vocal fold paralysis, scars, atrophy, and polyps (vocal fold paralysis or paresis $n = 7$, vocal atrophy $n = 5$, polyps $n = 5$, and scars/sulci $n = 3$). We excluded diplophonia due to functional conditions as they could not be well characterized as being due to organic etiology. The condition of polyps, paralysis, and scars corresponded to the model of mass, tension, or stiffness pathology of the vocal folds. Those in the paralysis group and vocal atrophy group were labeled as having tension abnormalities. Those having polyps are labeled as mass abnormalities, and those with scars and sulci were labeled as having stiffness properties. The methodology for the capture of HSVs and digital analysis of the HSV has been previously published.¹¹

HSV recording

HSVs were acquired using the Kay Elemetrics High-Speed Digital Imaging (HSDI) system (*KayPentax Photronmotion*; Pentax Medical, Montvale, New Jersey), which consisted of a 70-degree rigid endoscope (Model 9100) coupled with a 300-Watt Xenon light source. The HSV system acquired gray-scale images at a rate of 2,000 frames per second with a spatial resolution of 256×120 pixels rotated to a vertical position for capture. HSVs were performed as in conventional videostroboscopy procedure. Following the instructions and practice trials, 8 seconds of data was captured during the production of “ee” at comfortable fundamental frequency and loudness. Once the audible diplophonia component was heard by the examiner, the trigger for capture was initiated. The trigger captures 2 seconds of video data before and 6 seconds after the trigger.

The video is played back and reviewed at 20 frames per second. The diplophonia segment during the steady-state phonation is identified by eye and selected for further analysis. Five hundred and twelve frames of the video showing diplophonia are selected for future digital image and vibrogram analysis. To qualify for the diagnosis of diplophonia and separate these from other vocal kinematic abnormalities such as transient voice breaks,

tremors, or simply chaotic vocal fold vibrations, the segment of the video must show any of the following: (1) repetitive pattern of vocal fold vibration with observable frequency differences between two folds; (2) repetitive differences in vibration pattern between different portions of the vocal folds; and (3) repetitive and alternative oscillators beyond the true vocal folds that can be observed in addition to true vocal fold oscillation.

Data analysis

Kymography image processing

Kymograph analysis of the vibratory samples was carried out. The images were adjusted to enhance brightness and contrast between the glottis and the vocal fold. A 512-frame video segment that demonstrated a full view of the vocal fold with minimal movement of the subject was extracted for analysis. *Kay's Image Processing Software* (Model 9181; Pentax Medical) was used to generate two kymograms. The first is by placing a transverse line across the glottis at the junction of the anterior one-third to mid-one-third of the membranous vocal fold. The second digital videokymography (DKG) line is placed at the junction between the middle one-third of the membranous vocal fold and the posterior one-third of the membranous vocal fold. The DKG image is then adjusted by brightening and or darkening the image prior to submitting it for edge detection. The adequacy of edge detection was verified by eye prior to further extraction of the vibrogram waveform. The edge detection software was subsequently applied to identify and trace the vocal fold edges from the DKG waveform (Figure 1). When the tracing delineated the vocal fold edges was complete and verified by eye, the Kymograph Edge Analysis function was applied on the kymogram edge to extract the waveform (Figure 2). The resulting values are the Kymograph Edge Data, which describe the coordinate values of the left and right edges of the vocal fold presented across time. From the four lines traced from the two DKG tracings, the waveforms were submitted for fast Fourier transformation (FFT) analysis. This results in right and left waveform spectral power plot (y-axis) versus frequency plot ranging from 0 to 1000 Hz (x-axis) at the two DKG lines (Figure 3). Figure 2 shows the waveform plot of a patient with diplophonia, and Figure 3 shows the FFT plot of the vibrogram waveform analysis for the two DKG lines traced from Figure 1.

Qualitative evaluation of the vibrogram and quantitative spectral data analysis

The video segment, the spectrogram, and the vibrogram for each subject were evaluated by eye. The site and type of involvement were classified as (1) symmetric versus asymmetric causes for diplophonia due to unilateral or bilateral vocal fold motion abnormality; (2) diplophonia due to DKG difference from the front DKG tracing versus the posterior DKG tracing; or (3) true vocal fold only or extra vocal fold oscillation as the cause for diplophonia.

From the FFT plots, the extraction of values was obtained from each DKG vibrogram spectrum for additional analysis: the frequency and peak power values of the fundamental ($F_0 = H_1$), second harmonic (H_2), and third harmonic (H_3). This was done for each fold for the two DKG plots. The interharmonic energy

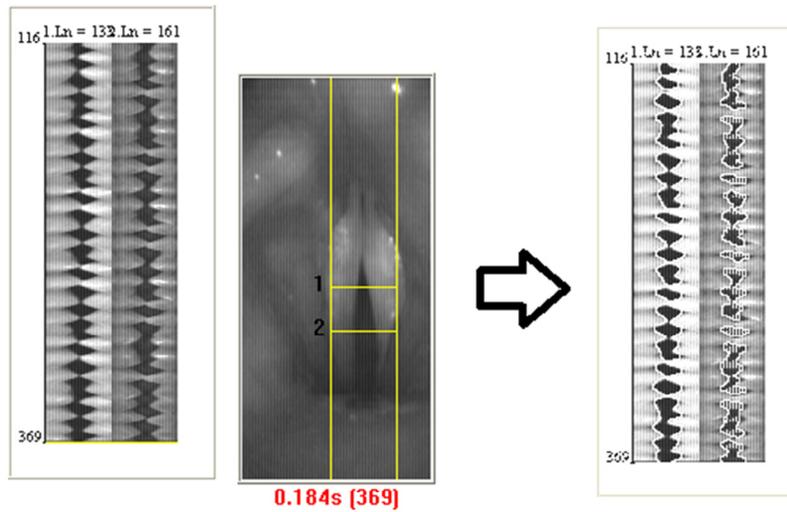


FIGURE 1. Schematic of the extraction of vibrogram waveform tracing from the HSV. The DKG is processed by edge detection to trace out the DKG vibrogram waveform.

and subharmonic energy for each spectrogram was noted for both DKG tracings. The data were entered into an Excel spread sheet, and an analysis was made by a correlation of right versus left fundamental frequencies and the presence of the interharmonic peak and the subharmonic energy peak.

RESULTS

Qualitative analysis

Figure 4 is a montage of all 20 patients with two DKG tracings with the pathological condition labeled below each tracing.

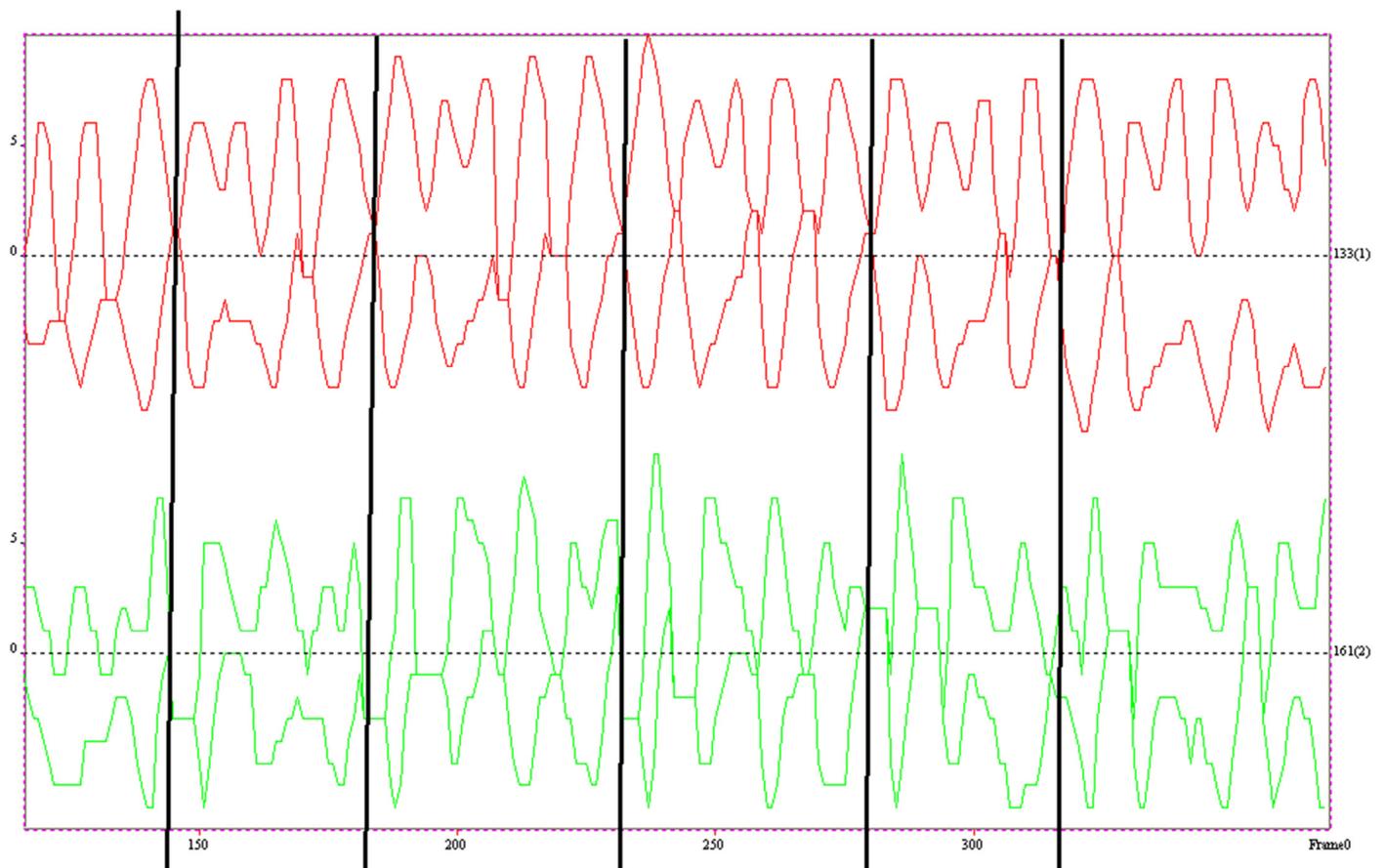


FIGURE 2. DKG waveform tracking plot with time on the x-axis and left and right folds on the y-axis. The regularly occurring diplophonia cycles every fourth beat indicated by the vertical black lines.

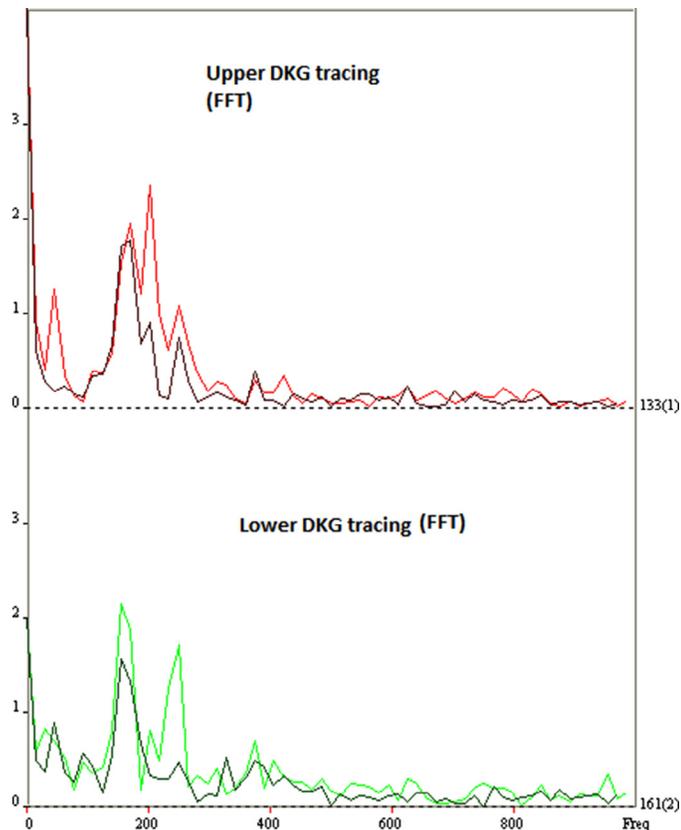


FIGURE 3. FFT of the waveform plot from Figure 2. Note that there are multiple interharmonic peaks above the fundamental frequency as well as a subharmonic peak.

A qualitative examination of the DKG vibrogram showed different patterns associated with different vocal pathologies.

Diplophonia patterns can be differentiated by eye as being due to unilateral involvement of one vocal fold or those with bilateral involvement. Unilateral involvement is most obvious in the patient with vocal fold paralysis. Figure 1 shows a kymography tracing of a patient with left vocal fold paralysis. For every four oscillations on the right fold, there are five beats on the left side. This results in vocal folds vibrating in phase for two cycles for every five cycles of oscillation. The other three cycles have each fold vibrating out of phase to each other. The paralyzed side is the side that has a higher fundamental frequency. This results in in-phase then out-of-phase vibrations, creating a subharmonic lower than either vocal fold fundamental frequency that is repeated every five glottal cycles. When analyzed by FFT, there is a spectral peak with two different fundamental frequencies for the F_0 (Figure 3), and a subharmonic peak is present at one-fifth of the fundamental frequency. This subharmonic peak, periodic but well below the fundamental frequency of either vocal fold, is what is perceived by the ear as an extra sound. The genesis for the subharmonic is due to a phase interaction between two folds vibrating at two different frequencies that then interact with each other as they come in and then out of phase on a quasiperiodic but subharmonic frequency. The subharmonic frequency is approximately one-fifth of the fundamental frequency

as the repetitive subharmonic cycle is repeated every five cycles on the left fold and six glottal cycles on the right fold.

Symmetric vocal fold oscillations can also result in diplophonia. This is seen best in patients with vocal atrophy. Figure 5 is a patient with vocal fold bowing and atrophy with an open-phase dominant pattern. On a sustained vowel production, diplophonia with a distinct low subharmonic is able to be heard. On HSVs, the more posterior DKG looks quite normal with the sinusoidal pattern of vocal fold oscillation. The anterior DKG shows a break in the periodic vibration with three normal beats followed by a break then four normal beats followed by a break. In between these aperiodic breaks, the vocal folds are in contact with good opposition of the other vocal fold. The normal oscillation continues for three or four glottal cycles, only to see another break with an incomplete closure. This break is a glottal cycle where there is no contact with a prolonged duration for the glottal cycle (Figure 5 arrows). This is not due to one vocal fold vibrating out of phase but due to a symmetric loss of vibration from both folds. Furthermore, this abnormality occurs only at the anterior aspect of the vocal fold and not along the full length of the vocal fold. This patient has vocal atrophy with an anterior gap on stroboscopy. HSVs defined the vibratory abnormality as coming from both vocal folds with regularly appearing aperiodic breaks due to anterior glottal incompetence. The subharmonic is occurring at one-fourth of the fundamental frequency. In this case of atrophy, the vocal fold vibration is completely symmetrical to the other fold. Both vocal folds slow down, fail to approximate, and then go back into a more steady-state oscillation. The FFT of the DKG plots shows there is a symmetric representation of power in the fundamental frequency from both folds (Figure 6). In the lower tracing there is now a new peak above the second harmonic that is not seen in the upper DKG FFT (Figure 6 down arrow). This is accompanied by a new subharmonic peak at one-fourth of the fundamental frequency (Figure 6 up arrow). HSV evidence of periodically occurring aperiodic voice breakdown at the anterior DKG site corresponds to the acoustic perception of diplophonia. In both symmetric and asymmetric vibration abnormalities, there are periodic subharmonic oscillations below the fundamental frequency. This results in a subharmonic peak on the FFT plot. In the paralysis case, this was due to two fundamental frequencies coming in and out of phase interaction. In the atrophy case, this was due to an incomplete closure at the anterior aspect of the true vocal fold with the breakdown of normal vibration. Both resulted in subharmonic diplophonia, but one can identify symmetric versus asymmetric patterns on visual inspection and by document on signal analysis. These two cases represent the asymmetric and symmetric breakdown of oscillation to create diplophonia. While the asymmetric breakdown may be due to asymmetric tension and mass effects, vocal atrophy and incomplete approximation of the fold can result in a breakdown of the aerodynamic viscoelastic properties needed for sustained periodic oscillation. Symmetric causes of a diplophonia vibratory pattern can occur without asymmetric mass, stiffness, or tension in one vocal fold.

In patients with vocal fold polyps, the vocal polyp can divide the fold into two oscillators, and this can result in diplophonia.

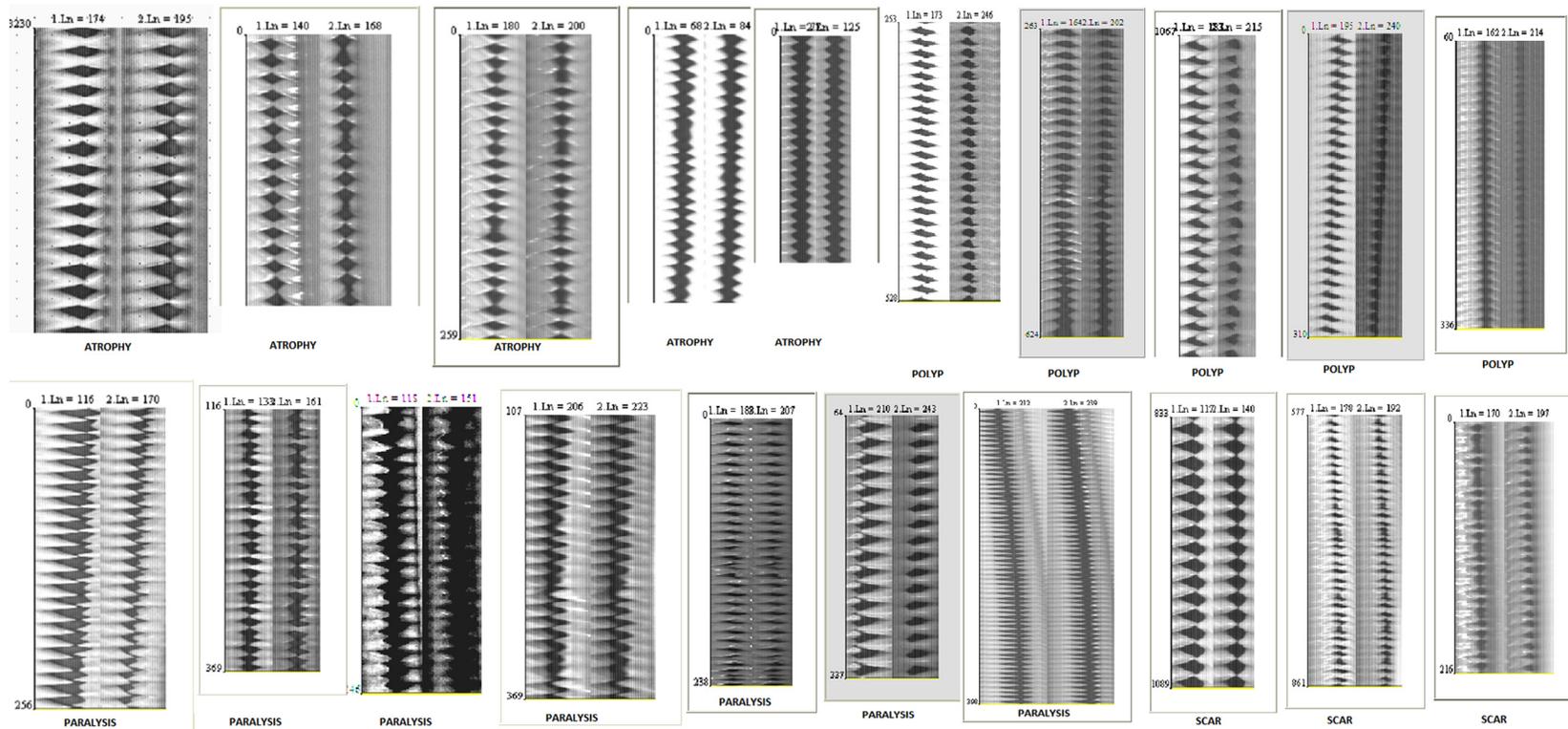


FIGURE 4. Montage of the DKG tracings for 20 subjects. Each is labeled below with the pathological condition.

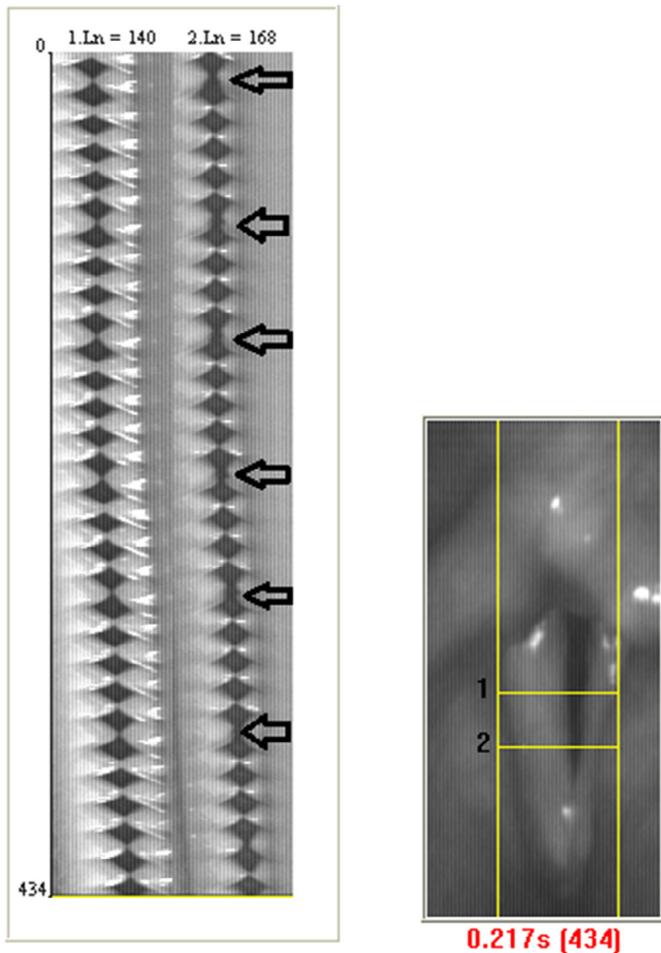


FIGURE 5. DKG display showing regular breaks in the vibration pattern in only the anterior part of the DKG tracing (arrow) in a patient with atrophy. This occurs every five beats and is symmetric as both vocal folds are oscillating at the same rate.

One part of the vocal fold may vibrate normally, while another can show vibratory anomalies. In [Figure 7](#), a polyp divides the vocal fold into two different parts. The posterior part appears to have quasiperiodic oscillation. The anterior DKG shows a dicrotic vibration pattern contributed mostly by an abnormal vibration from the side opposite the polyp. On the lower DKG tracing, each beat of the glottal cycle has a secondary smaller dicrotic beat to each glottal cycle. This is from the left vocal fold. This results in a dicrotic vibration ([Figure 7](#) arrows). The dicrotic vibrations are not uniform. Every second beat of the vibrogram appears to be attenuated, such that the dicrotic vibration patterns are repeated at regular intervals at every other glottal cycle. The dicrotic vibration then alternates in beats. It is not clear whether the perceived diplophonia is due to the dicrotic vibration or due to the alternating beats in the dicrotic vibration ([Figure 7](#) arrows).

Paralysis

Two different frequencies with one fold vibrating faster were most obvious in the patients with paralysis ([Figures 1–4](#)). The paralyzed fold looks more floppy and seems to vibrate with extra

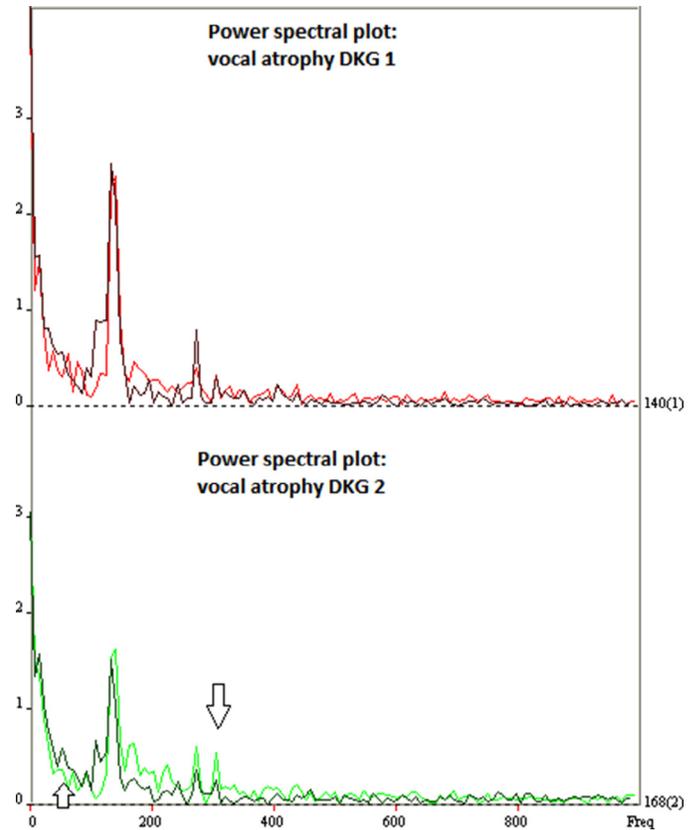


FIGURE 6. FFT plot of the waveform extracted from [Figure 5](#). Note the symmetry of F0, but on the second DKG tracing there is both an interharmonic peak (down arrow) and a subharmonic peak present (up arrow).

beats. When FFT is applied to the DKG vibrogram, the paralyzed side has a higher fundamental frequency. Sometimes the paralyzed side will have an extra beat added as it appears quite floppy compared with the sinusoidal pattern from the innervated fold. The higher frequency from the paralyzed side creates phase shift differences such that the folds come into and then out of phase. This creates the subharmonics below the fundamental frequency of vibration of each vocal fold. This example is illustrated in [Figure 1](#) for the DKG, and [Figure 3](#) shows the FFT characteristic of diplophonia in paralysis patients.

Atrophy

Atrophy patients will show a symmetric pattern of diplophonia with both folds vibrating at the same frequency. The vocal folds will show a breakdown of vibration with a visible repetition in loss of normal vibration every few glottal cycles. Unlike the asymmetric pattern often seen in paralysis, both vocal folds show this pattern of breakdown with each fold having an exact mirroring of the opposite fold. The symmetric pattern of diplophonia is characteristic of the patients with vocal fold bowing and atrophy ([Figures 4–6](#)).

Scar

Vocal fold stiffness due to scar or sulcus formation can result in diplophonia on only one side or both sides. Periodic vibration

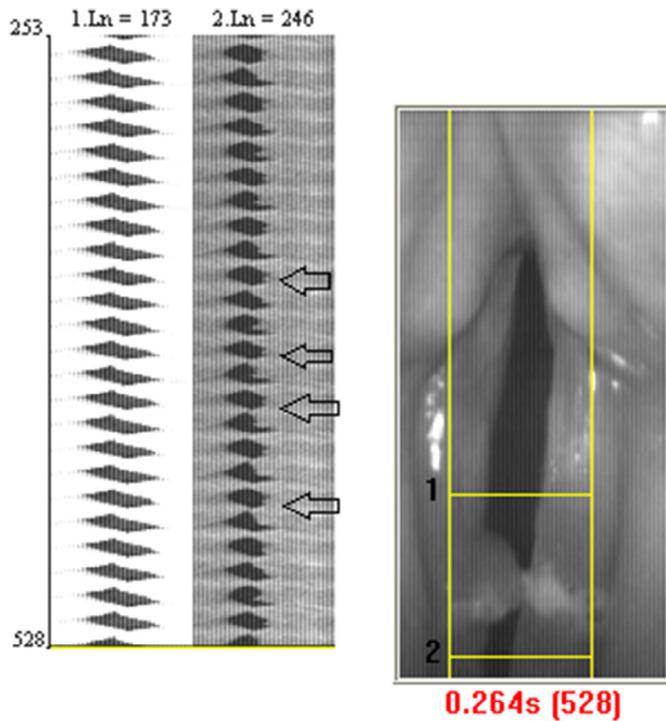


FIGURE 7. DKG tracing of a patient with a polyp. At the more anterior DKG trace, there is an alternating beat with a dicrotic pattern (arrows). This is on the side without the polyp.

may be present with a dicrotic vibration pattern, where the vocal folds may show alternate large and then small glottal cycles. One such example is shown in Figure 8. There are chaotic vibrations with alternate beats that change every five to six cycles and result in the bilateral degradation of the DKG. The abnormal vibration can come from the stiff fold as well as from the more pliable fold. Figure 9 is a DKG showing vibrations that show every sixth beat alteration from the posterior DKG tracing in a subject with a sulcus. The abnormal DKG is occurring in both folds and is not limited to the scarred side.

Quantitative analysis of the vibrogram is performed by FFT analysis of the DKG waveform. In subjects with different fundamental frequencies, the right and left spectral peaks will not overlap and will have a separate F_0 . The side with paralysis will show a higher-frequency spectral peak. A common finding is the presence of a subharmonic energy peak. The subharmonic peak is well below the fundamental frequency of either vocal fold. This is interpreted as either due to the phased interaction that results in every third to fifth cycle coming in and then out of phase to each other or due to regularly occurring aperiodic beats in the vibration pattern creating a distinct subharmonic below the F_0 .

An abnormal spectral peak in both the interharmonics area and the subharmonic area was present in the majority of subjects but not all. Overall, 14 of 20 subjects in this study had both subharmonic peaks and interharmonic peaks that could be identified, but six subjects did not have both. Figure 10 graphs the spectral energy of the subharmonic peaks and the interharmonic peaks for each subject. In 14 of the 20 subjects, both subharmonic and interharmonic peaks were easily tabulated. Eighteen of the

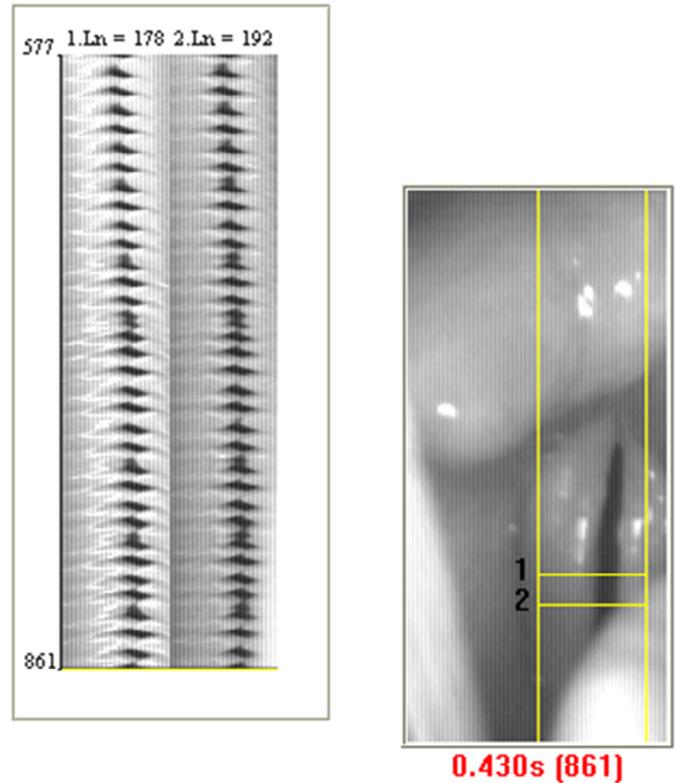


FIGURE 8. DKG tracing of a patient with a vocal fold scar. The regularly occurring breakdown of vocal fold oscillation is from both vocal folds as it skips beats every three to five glottal cycles.

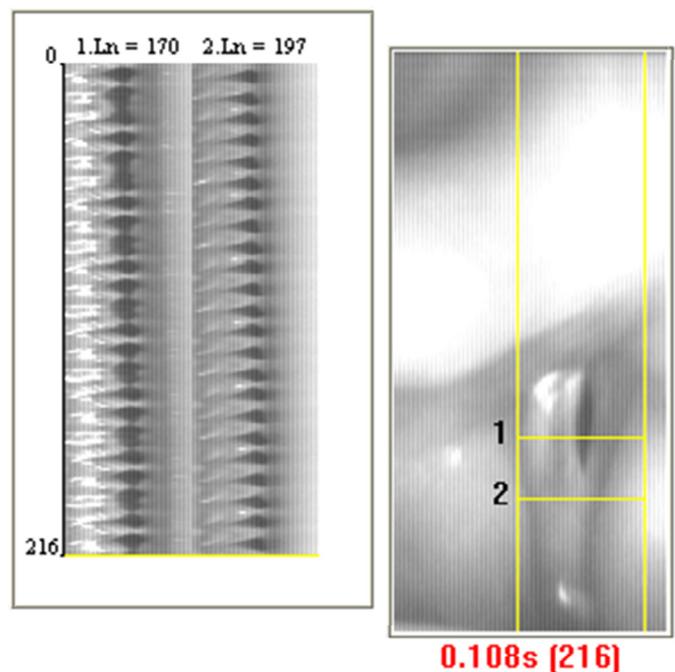


FIGURE 9. DKG tracing from a patient with a sulcus. Note that only the upper tracing shows diplophonia breaks with in-phase oscillation every sixth beat. This is a symmetric pattern.

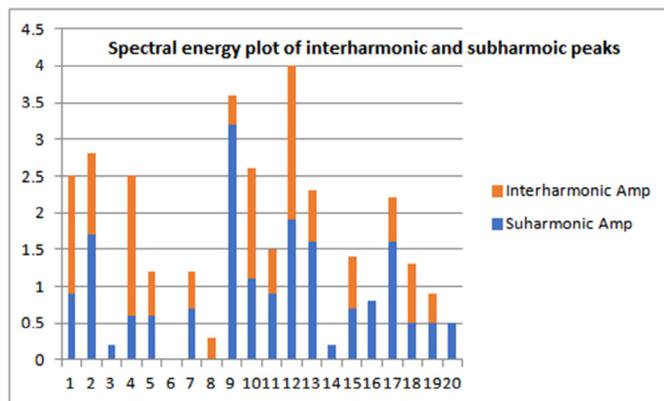


FIGURE 10. Spectral energy peak plots for subharmonic and interharmonic energy peaks for the 20 subjects. Note that the majority of subjects have both a subharmonic peak and an interharmonic peak that could be calculated.

20 subjects had a subharmonic peak, and 16 of the 20 subjects had an easily identified interharmonic peak. When the HSVs without subharmonic peaks were then further evaluated by playback, the vibration patterns were more consistent with a dicrotic vibration and did not have regularly occurring subharmonic variations. This resembled the vibration patterns of vocal fry and not of typical diplophonia. This may point to the possibility that perceived acoustic quality to the examiner as diplophonia may not be due to a subharmonic interaction but may be due to a dichrotic vocal fold vibration similar to vocal fry.

DISCUSSION

This paper examines the vibratory characteristics of 20 patients with disparate causes of diplophonia. The paper uses available edge tracking software to analyze the vibrogram from a multislice DKG of HSVs to evaluate the spectral characteristics of patients with diplophonia. The advantages of a spectral analysis of the vibrogram over that of a simple qualitative interpretation is the ability to characterize the spectral components of the vibrogram waveform.

We have identified multiple types of vibratory spectral anomalies that can result in diplophonia. The vibrogram may be different between the two folds, they may be different between the anterior DKG vibrogram and the posterior DKG vibrogram, and they may result in periodic ectopic beats without any specific vocal fold pathology other than atrophy. While the patterns of vocal fold vibratory abnormalities may appear to be complex and the spectrograms may seem difficult to categorize, we can confidently state that most of the abnormalities in vibrograms of patients with diplophonia are due to vocal fold abnormalities. The 20 subjects did not show supraglottis or pharyngeal vibrators to account for their vibratory abnormality. Compared with our understanding of normal fold DKGs studied using similar techniques,¹¹ we can see important differences between these patients compared with normal patients. Diplophonia vibrograms and their spectral characteristics differ from normal vibrograms in the following: (1) there is primary energy in the fundamental frequency with loss of second and third harmonic energy

compared with normal vibrograms; (2) there is elevated interharmonic energy with often a specific spectral peak in the energy above the fundamental frequency; and (3) both symmetric and asymmetric patterns of diplophonia will often show a subharmonic energy peak.

The subharmonic peak can be small due to dicrotic vibration patterns or large with ectopic beats occurring at regular intervals at every three to seven beats. These subharmonic peaks result in the typical subharmonic oscillation pattern that can be traced by DKG alone and can be identified by the eye. Subharmonic oscillations that occur with regularity may be due to the symmetric oscillatory breakdown seen in patients with scars and atrophies or due to asymmetric vibratory differences as seen in patients with paralysis or mass lesions. Based on this, we feel the resulting vocal fold vibratory breakdowns that were recorded in this clinical series resulted and contributed in some measure to the production of acoustic signatures so characteristic of the “diplophonia” voice quality. Further study will be necessary to link the acoustic signature of acoustic subharmonics in perceived diplophonia to the subharmonic spectral peaks seen in the DKG vibrogram from this study.

Using HSVs for the study of diplophonia voices by HSVs of vocal fold vibrations may have a clinical utility. We can now clearly recognize several patterns of diplophonia. How these patterns are associated with different disease processes are of interest for further investigation. The clearest patterns are those asymmetric patterns more associated with unilateral paralysis versus those symmetric patterns more associated with atrophy. Unilateral paralysis is associated with the asymmetric pattern of diplophonia, while the atrophy patient has symmetric diplophonia. This may simply be due to an asymmetric tension in paralysis versus a symmetric tension anomaly in atrophy patients due to tissue atrophy or poor breath support. This pattern is sufficiently distinct that we believe the diplophonia pattern associated with paralysis is indicative of high-grade paralysis due to loss of motor tone, resulting in the higher fundamental frequency due to loss of the two-mass model of vocal fold oscillation associated with innervated vocal folds. Additional data will be needed to see if these impressions hold true. Such stratification based on diplophonia patterns may have clinical applications. While the patient with paralysis may benefit from unilateral augmentation or reinnervation, the patient with the atrophic pattern should be considered for bilateral augmentation by an injection laryngoplasty or medialization laryngoplasty. Recognition that asymmetric patterns are associated with unilateral disease processes may prompt the clinician to seek additional diagnostic and therapeutic treatments based on information acquired from HSVs. Additional analysis will be needed in the scar and sulcus patterns to better define whether voice therapy, augmentation, or scar lysis will change the oscillatory properties that contribute to the production of diplophonia.

An understanding of the diplophonia character can change the management of the patient. For example, in patients with glottis insufficiency with paresis, one can encounter diplophonia without obvious vocal fold paralysis. The finding of a symmetric pattern of diplophonia would then favor the diagnosis of atrophy, while the finding of two independent frequencies from each fold would

favor paralysis or paresis. This was used in an early report of a paresis in a young woman with vocal fold paresis and had a thymoma found after an HSV indicated a paresis pattern.¹² In patients with polyps or nodules in the midmembranous vocal fold, the removal of the lesion results in the entire vocal fold vibrating in synchrony, especially in the contralateral, normal-appearing vocal fold that was vibrating with a dicrotic pattern.

This is an early attempt to tabulate DKG patterns in a variety of diplophonia voice disorders. While most patients with dysphonia can be studied by stroboscopy due to the quasiperiodic nature of their dysphonia, HSVs remain the only method to visualize vocal fold vibratory abnormalities in this fascinating phonation disorder. We have demonstrated that diplophonia vibration patterns differ depending on the pathology. The patterns that are due to pathological conditions due to mass lesions, tension abnormalities, and stiffness result in different vibration patterns. These patterns can now be recorded using HSVs and analyzed using spectral analysis of the vibrogram. Using digital DKGs, the vibration patterns contributing to diplophonia can be analyzed qualitatively, and using a digital image analysis, a spectrogram analysis of the vibrogram can be achieved to gain insight on the spectral characteristics of the diplophonia gesture. A valid concern in using DKG analysis with HSVs is the use of a two-dimensional imaging technique (DKG) to evaluate a three-dimensional event of vocal fold oscillation. This is especially true in patients with vocal fold paralysis, where level differences are common. The interaction of vocal folds at different levels results in phase shift anomalies that can be easily appreciated by stroboscopy. Level differences probably also contribute to the instability of the oscillators at different heights, resulting in asynchronous and variable frequencies between the two sides, thereby producing asymmetric patterns of diplophonia. The presence of symmetric diplophonia may be more indicative of a more global physiological deficiency such as atrophy and poor breath support, while an asymmetric pattern may be more indicative of a localized anatomic pathology.

The challenges of such a study are characterized by the often transient nature of diplophonia. The 20 patients with diplophonia were captured over 2 years of a busy clinical office practice. Although the clinician can hear diplophonia many times, HSV capture of the diplophonia gesture is not always possible. Once captured, the edge detection and waveform extraction can be time intensive and tedious. HSVs are often not of sufficient quality to render for objective edge tracking and analysis. The soft-

ware requires human visual validation before waveform extraction and spectral analysis. Lastly, there are no standardized norms for the comparison of abnormal calculations obtained from pathological cases to normal vibrogram values. Until normative data can be established in terms of frequency, gender, and loudness, the observations made in this type of study will continue to be largely qualitative and observational in nature. Despite this, there is optimism that additional investigation using tools outlined in our study will continue to contribute to the clinicians' understanding of this fascinating voice disorder.

CONCLUSION

HSVs and spectral analysis of the DKG vibrogram allow the clinician to characterize the patterns of diplophonia. From a study of 20 subjects, we believe most patients with diplophonia have the pathology limited to vibratory anomalies arising from the vocal folds. These different patterns of diplophonia may be associated with different pathological states. Further clinical correlations of diplophonia patterns are worthy of future study.

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