2010

Vocal Fold Bowing in Elderly Male Monozygotic Twins: A Case Study

Kristine Tanner
The University Of Utah

Cara Sauder
University of Utah Hospitals and Clinics

Susan L. Thibeault
University of Wisconsin-Madison

Christopher Dromey
Brigham Young University, dromey@byu.edu

Marshall E. Smith
The University Of Utah

Original Publication Citation

Tanner, K., Sauder, C., Thibeault, S., Dromey, C. & Smith, M.E. (2010). Vocal fold bowing in elderly male monozygotic twins: A case study. Journal of Voice, 24, 470-476.

* Revised Manuscript

Vocal Fold Bowing in Elderly Male Monozygotic Twins: A Case Study

Kristine Tanner, Ph.D.
Department of Communication Sciences & Disorders and
Division of Otolaryngology—Head & Neck Surgery
The University of Utah
390 S. 1530 E., rm. 1310 BEH SCI
Salt Lake City, UT 84112-0252
kristine.tanner@hsc.utah.edu
(801) 633-7471
(801) 581-7955 (fax)

Cara Sauder, MA
Voice Disorders Center, University of Utah Hospitals and Clinics
Division of Otolaryngology-Head &Neck Surgery
Surgical Specialty Center
CAMT Building
729 Arapeen Dr.
Salt Lake City, UT 84108
cara.sauder@hsc.utah.edu
(801) 585-7946
(801) 587-3569 (fax)

Susan L. Thibeault, Ph.D.
Division of Otolaryngology—Head & Neck Surgery
The University of Wisconsin—Madison
5107 Wisconsin Institute of Medical Research
1111 Highland Ave
Madison, Wisconsin 53705-2275
thibeault@surgery.wisc.edu
(608) 263-6751
(608) 263-6199 (fax)

Christopher Dromey, Ph.D.
Communication Disorders
Brigham Young University
133 TLRB
Provo, UT 84602
dromey @byu.edu
(801) 422-6461

Marshall E. Smith, MD
Division of Otolaryngology—Head & Neck Surgery
The University of Utah and
Primary Children's Medical Center

50 N. Medical Drive
3C 120 SOM
Salt Lake City, UT 84132
(801) 588-2782
(801) 587-3569 (fax)

A portion of this study was presented at the Voice Foundation Annual Symposium, Philadelphia, PA, June 4, 2006.

Abstract

Objectives: This study examined case histories, diagnostic features, and treatment response in two 79-year-old male monozygotic (identical) twins with vocal fold bowing, exploring both genetic and environmental factors.

Study Design: Case study.

Methods: DNA concordance was examined via cheek swab. Case histories, videostroboscopy, auditory- and visual-perceptual assessment, electromyography, acoustic measures, and Voice Handicap ratings were undertaken. Both twins underwent surgical intervention and subsequent voice therapy.

Results: Monozygosity was confirmed for DNA polymorphisms, with 10 of 10 concordance for STR DNA markers. For both twins, auditory and visual-perceptual assessments indicated severe bowing, hoarseness and breathiness, although Twin 1 was judged to be extremely severe. Differences in RMS amplitudes were observed for TA and LCA muscles, with smaller relative amplitudes observed for the Twin 1 versus Twin 2. No consistent voice improvement was observed following surgical intervention(s), despite improved mid-membranous vocal fold closure. Marked reductions in Voice Handicap Index total scores were observed following behavioral voice therapy, coinciding with increased mid-membranous and posterior laryngeal (interarytenoid) glottal closure. No substantive differences in acoustic measures were observed.

Conclusions: Vocal fold bowing was more severe for Twin 1 versus Twin 2 despite identical heritability factors. Overall voice improvement with treatment was greater for Twin 2 than Twin 1. Environmental factors might partially account for the differences observed between the twins, including variability in their responsiveness to behavioral voice therapy. Voice therapy was useful in improving mid-membranous and posterior laryngeal closure, although dysphonia remained severe in both cases.

KEYWORDS: twins, bowing, EMG, breathiness, aging

Introduction

Presbylaryngis, or aging of the larynx, is a relatively common diagnosis in the elderly and is characterized by the laryngeal appearance of vocal fold bowing and dysphonia.[1] Incidence rates of bowing are as high as 30% in elderly clinical (treatment-seeking) populations,[2,3] and disagreement exists regarding whether vocal fold bowing is a normal part of the aging process or should be considered pathological.[4] Age-related changes in laryngeal structures and function are well-documented, although to what extent these might contribute to dysphonia and/or bowing is unclear.[5] The precise etiology of vocal fold bowing, including its progression, treatment responsiveness, and the relative contribution of genetic and environmental factors, remains unknown. Some evidence exists of both genetic and environmental influences on aspects of voice disorders and vocal aging, including auditory-perceptual features and disorder types, although no studies have specifically examined the relationship between these factors and vocal fold bowing. Recent twin studies have more closely examined the interaction between environmental factors, such as occupational voice demands, and genetics in dysphonia.[6] This case study provides a unique opportunity to explore the history, clinical presentation, and treatment response in two male geriatric monozygotic twins with vocal fold bowing.

Research suggests that neuromuscular, histological, and musculoskeletal changes occur with advanced chronological age, and bowing in the elderly population is presumed to reflect atrophic changes of the thyroarytenoid (TA) muscle. Although not specific to idiopathic vocal fold bowing, electromyography (EMG) research has compared TA motor unit durations, relative, average and absolute motor unit amplitudes in younger and older individuals, as well as those with Parkinson's Disease.[7] The results indicated that elderly speakers produced higher mean amplitudes than the younger group and lower mean amplitudes compared to the PD group. Maximum TA absolute values were highest in the elderly group, resulting in lower relative amplitudes compared to both the younger group and the PD group. Consistent with these findings of higher mean and maximum TA amplitudes, motor unit durations were also increased in elderly males compared to younger participants.[8] Collectively, EMG findings of increased motor unit amplitudes and durations presumably represent activation of denervated/reinnervated muscle fibers and greater motor unit recruitment. EMG findings corroborate histological findings of muscle fiber reorganization and composition alteration with age. An age-related reduction in length of type 1 slow twitch muscle fibers and greater numbers of co-localized fibers also support evidence of motor unit remodeling following cycles of denervation and reinervation.[9]

Extra-cellular matrix (ECM) composition of the lamina propria is an important determinant of the viscoelastic properties of the vocal folds that are required for phonation. Histological analysis has revealed age-related changes in ECM components such as collagenous and reticular fibers, protein distributions, and hyaluronic acid (HA) levels.[10] An age-related reduction in interstitial space in the superior lamina propria as well as degradation and reduction in reticular fibers, which allow for greater tissue compliance compared to collagenous fibers, has also been observed.[11] These alterations impact elastic properties and might explain the characteristic glottal incompetence and alteration of vibratory characteristics observed in vocal fold bowing. In addition to potential changes in elastic properties of the vocal folds, viscosity is also associated with vocal effort, vibratory efficiency, and phonation threshold pressure.[13] Viscosity is influenced by the relative concentration of HA, which influences water content regulation. A trend towards less overall HA in the ECM has also been observed in older as compared to younger individuals.[10] In addition to reductions in HA concentration, laryngeal gland atrophy in the region of the false folds has also been documented in older adults.[14] Other signs of drying such as a reduction in secretory granules in serous cells and mucigen droplets have been reported.[11] There is also evidence that the decline in lung elasticity and respiratory strength associated with aging might further degrade phonatory efficiency.[12] Voice changes and their association with increasing chronological age have been well-documented, however there are often no distinctions within medical and voice-related histories to determine whether or not these changes are a normal part of the aging process or are pathological. However, the voice changes associated with increasing chronological age are compatible with many of the clinical features of vocal fold bowing.

Structural and physiological changes have been observed with increasing chronological age. However, the rate of aging varies such that chronological and physiological age might differ. Both genetic and environmental factors influence the aging process and might explain some of the changes observed in the aging voice. Differences in auditory-perceptual and acoustic voice characteristics of individuals of the same chronological age indicate some variability in the rate of vocal aging. Ramig and colleagues[15] studied two- and three-generation families to determine if other physiological measures of aging were similar to that of vocal aging. Listener-perceived age and some acoustic data correlated with measures of physiological aging, such as bone density and loss of visual acuity. Heritability patterns in physiological aging and its relationship with vocal aging has also been observed. Familial similarities in voice characteristics of monozygotic (MZ) and dizygotic (DZ) twins, as well as higher prevalence rates of voice disorders in those with a family history of voice disorders families have been reported.[16] Fairly high correlations between fundamental frequencies (F_0) of MZ and DZ twin pairs have been observed, with a greater correlation in MZ twins when controlling for height and weight.[17] Similarities in maximum phonation times and perceptual voice characteristics in MZ twins have also been identified.[18] Interestingly, a perhaps expected decline in the correlation between F_0 in twin pairs over time as a result of increased environmental influence has not been observed, suggesting a strong genetic influence.[19] The precise nature and relative contribution of environmental influences on vocal aging is unknown. Nevertheless, the role of occupational voice use in predicting voice disorders and voice complaints has been clearly established.[6, 16] A recent twin study investigated the effects of genetic and high occupational voice demands in MZ and DZ twins.[6] Surveys regarding specific voice problems were administered to MZ and DZ twin pairs with either high or low occupational voice demands. A moderate genetic effect was observed for the presence of voice problems, but environmental factors such as occupational voice demands had a greater effect. Although no age effect was observed, these twin pairs were relatively young (i.e., under 45 years of age).

The present investigation was undertaken in order to study in detail the case histories, diagnostic features, and responses to treatment in two 79-year-old male MZ twins with vocal fold bowing. A longitudinal, descriptive case study design afforded the opportunity to examine similarities and differences in the onset, progression, course, and response to treatment exploring both genetic and environmental factors. Auditory- and visual-perceptual, acoustic, and EMG measures were compared.

Methods

Participants

Two elderly male, monozygotic (identical) twins participated in this study (age = 79 years). Both twins were seen at The Voice Disorders Center at The University of Utah in May of 2005 for assessment and management related to complaints of hoarseness, vocal weakness and dysphagia. The diagnosis of vocal fold bowing was confirmed by a multidisciplinary team including a speech-language pathologist and an otolaryngologist following a detailed case history, auditory-perceptual analysis, and rigid videolaryngostroboscopy. Zygosity determination was undertaken using DNA polymorphisms, and the twins were concordant for 10 out of 10 STR DNA markers, indicating monozygosity with a greater than 99% probability (Affiliated Genetics, Salt Lake City, UT).

Case History

Environmental history. The twins were born and reared together, and have lived in close proximity to one another since birth in a rural area in Northern Utah. Both twins worked the majority of their adult lives as carpenters, were married in their mid-twenties, raised children, and never divorced. In their mid-sixties, both twins retired; however, Twin 1 was subsequently widowed (for 10 years prior to his initial evaluation at our clinic) and continued to live alone and work on a cattle ranch. Twin 2 continued to reside with his spouse throughout the duration of the study.

Medical history. With respect to the twins' medical histories, Twin 1 reported high cholesterol, high blood pressure, arrhythmia, and a previous of pneumonia. Twin 1's previous surgeries included hernia repair, rotator cuff repair, and a non-specified heart surgery for “irregular heart beat”, presumed to be pacemaker placement. Both twins were non-drinkers and non-smokers throughout their lifetimes. Medications included Lisinopril, Crestor, and aspirin. Twin 2’s medical history was unremarkable, with no health conditions or previous surgeries reported and no medications. However, Twin 2 reported that he experienced frequent acid reflux symptoms.

Two-channel 24-hour pH probe with manometry was undertaken, and reflux occurrences, durations, and levels (i.e., when $pH < 4$) were recorded for proximal and distal channels. Twin 1 had a significantly elevated Demeester score and was diagnosed with pathological gastroesophageal reflux. Twin 2’s pH probe results were unremarkable.

Voice history. Regarding voice and swallowing complaints, Twin 1 reported a 20 year history of a weak, breathy voice as well as effortful voice production that gradually worsened in severity. He rated his voice as 50% of normal at the time of his initial evaluation in May of 2005 (0% = no voice at all; 100% = completely normal voice). He reported a 10-year history of swallowing difficulties with both solid foods and liquids; however, he never sought medical attention for dysphagia.

Twin 2 reported a five-year history of a weak, hoarse voice. He rated his voice as 50% normal at the time of his initial evaluation. Twin 2 reported a three-year history of swallowing difficulties with solid foods, and a chin-tuck swallowing maneuver was recommended following modified barium swallow study performed at that time. However, speech/dysphagia therapy was not recommended at that time, and Twin 2 reported no improvement with the chin tuck maneuver.

Psychosocial history. With respect to psychosocial history, Twin 1 reported that he was fairly quiet and did not interact much with others since his wife died. He spoke with his brother often; however, this was generally the extent of his daily social interaction. He talked with his children occasionally over the telephone, and also interacted socially at church on Sundays. Twin 2 reported that he was more talkative than his twin brother, and spent the majority of his days at home with his wife and in his carpentry shop. He interacted with his brother and children over the telephone and at church on Sundays.

Initial Voice and Speech Assessment

The results from the twins' comprehensive voice and speech assessment in May of 2005 revealed severe vocal fold bowing with prominent auditory-perceptual features of severe breathiness and hoarseness, although Twin 1 demonstrated "extremely severe dysphonia". Maximum phonation time (MPT) for Twin 1 was one second, and was less than two seconds for Twin 2. No signs or symptoms of dysarthria were noted during alternating motion rates, diadokokinetic rates, oral reading, or during an oral mechanism examination. For both twins, rigid videolaryngostroboscopy revealed marked concavity of the medial edges of the vocal folds bilaterally during abduction with prominent vocal processes. Incomplete mid-membranous glottal closure was observed as well as a spindle-shaped vibratory pattern during all phonation attempts at modal and high pitches. A prominent and persistent posterior glottal gap (i.e., in the interarytenoid region posterior to the vocal processes) was observed. Generalized laryngeal erythema was also observed. Findings were similar for both twins, with the exception that both the midmembranous and posterior gaps were more severe for Twin 1. Additionally, a small varix was observed on the lateral margin of the anterior portion of the right fold (Figure 1).

Surgical Management

Twin 1 underwent a bilateral Hylaform medialization-injection procedure in June of 2005, followed by a bilateral Type 1 thyroplasty (3mm wedge implants) in August of 2005. Subsequently, Twin 1 received a bilateral adipose fat injection performed by a second otolaryngologist in December of 2005. Based on patient report, Twin 1 experienced temporary voice improvements with the Hylaform and fat injection procedures; however, these changes were modest and were not sustained. Twin 2 underwent a bilateral Hylaform medialization-injection procedure in August of 2005. He subsequently reported minimal, temporary voice improvements with the procedure. (It should be noted that videostroboscopic comparisons from initial evaluation, pre-therapy, and post-therapy indicated increases in mid-membranous closure following the medialization procedures, although a marked posterior glottal gap persisted).

Laryngeal Electromyography (LEMG)

In order to examine potential neuromuscular effects and/or etiologies related to the twins' severe vocal fold bowing, LEMG was undertaken. For each twin, the examination procedure was identical, and was performed in January of 2006 (i.e., following all surgical interventions/procedures). Prior to the LEMG procedure, a eutectic mixture of lidocaine (2.5%) and prilocaine (2.5%) was applied proximally on the neck in the region of the cricothyroid space. The otolaryngologist inserted modified bipolar hooked wire electrodes into the thyroarytenoid (TA) and lateral cricoarytenoid (LCA) musculature bilaterally. Electrode signals were verified using Valsalva and cough. EMG signals were captured and digitized using WINDAQ (version 2.44) acquisition software (DATAQ instruments, Akron, OH) simultaneously with the acoustic voice signal (headmounted microphone, AKG). The EMG signals were amplified and band-pass filtered, zero-meaned, rectified, and smoothed. The sample protocol included the following tasks, which were performed three times each (with the exception of quiet respiration): quiet respiration, swallow, cough, Valsalva, MPT (comfortable, soft, and loud), five-second (attempted) sustained /ɑ/ (comfortable, soft, and loud), and laryngeal DDKs.

For purposes of EMG signal analysis, maximum amplitudes for each task were identified and were used for relative amplitude calculations using a similar methodology previously described and reported by Baker and colleagues (1998).[7] In brief, EMG signals were downsampled from 10 kHz to 1 kHz, and RMS smoothed using Matlab (The Mathworks, Inc.). Prephonatory and phonatory segments were isolated 500 and 100 ms prior to and following mic onset, respectively (duration = 1000 ms). Maximum amplitudes were identified for each muscle by examining the entire EMG signals for right (R) TA, left (L) TA, RLCA, and LLCA. Relative amplitudes were calculated as the absolute mean (in μV), divided by the maximum signal, for each muscle.

Behavioral Voice Therapy

The twins underwent a four-session course of voice therapy from March to May of 2006. For each twin, the content and structure of the therapy sessions were identical, and were provided by the same clinician. Stimulability testing was undertaken involving a combination of manual circumlaryngeal techniques, resonance therapy training, and increased vocal effort and breath support was undertaken. The results from stimulability testing indicated a significant degree of hyperfunction and extralaryngeal muscle tension misregulation such that they were not judged to be suitable candidates for therapy involving the above treatment techniques. Because some recent evidence has been offered in the literature to suggest that Vocal Function Exercises (VFEs) might be useful in improving voice production in the elderly a short course of behavioral therapy involving these techniques was undertaken for each of the twins.[20]

Session one included videolaryngostroboscopic assessment and a pre-treatment audio-recording, followed by the introduction to VFE. Minimal voice facilitation techniques, including laryngeal reposturing and increased resonance, were briefly used to stimulate an "engaged" tone required for VFE productions. Instructional audio-recordings and written instructions were provided, and the twins were instructed to practice twice daily. Sessions two and three involved clinician model, guided practice, and corrective feedback related to performance of the VFEs. Session four included a post-treatment videostroboscopic assessment, an audio-recording, and instructions for a maintenance tapering program of the exercises. The Voice Handicap Index was administered during sessions one and four for purposes of documenting functional disability related to the twins' voice problems.[21]

Acoustic Analysis

Audio-recordings were collected in May 2005 (initial evaluation), January of 2006 (LEMG recordings), March of 2006 (pre-therapy) and May of 2006 (post-therapy) using a Sony digital video camera (DCR-TRV 350 NTSC Capture) and a Shure SM48 multi-directional microphone (mouth-to-mic distance held constant at 2 in), and including *The Rainbow Passage*, three sustained /ɑ/ tokens, maximum phonation time, and ascending and descending pitch glides. For purposes of acoustic analysis, audio signals were captured using the Computerized Speech Lab (version 4300B, Kay Elemetrics, Lincoln Park, NJ). Long-Term Average Spectrum (LTAS) analyses of the central sentences of the rainbow passage were undertaken using a wide analysis bandwidth (128-bit) and Hamming window weighting, with a frequency range of 0 to 8,000 Hertz, and percent jitter, shimmer, and harmonic-to-noise ratio were calculated from the central 1 sec of the second sustained /ɑ/ tokens using the Multidimensional Voice Range Profile (MultiSpeech, version 3.1.1, Kay Elemetrics, Lincoln Park, NJ).[22]

Results

Relative EMG Amplitudes

Relative EMG amplitudes (RA) means and maximums for prephonatory and phonatory segments during sustained /ɑ/ tokens at each loudness level are presented in Table 1. RA means for laryngeal DDKs (i.e., /ɑ ɑ ɑ/ versus /hɑ hɑ hɑ/) are also presented. In general for Twin 1, prephonatory RAs were greater than phonatory segment RAs. This pattern was most obvious during loud sustained vowel attempts, and was fairly consistent across muscles and speaking tasks. Additionally, RAs for RLCA were notably greater than for RTA, LLCA, and LTA, across all speaking tasks. During loud phonation, maximum RAs were much greater than the mean RAs for all muscles. In general, Twin 2 had greater RA means and maximums as compared to Twin 1 (Figure 1), and prephonatory RAs were smaller than phonatory RAs. This finding was particularly notable during laryngeal DDKs. Consistently, for both twins, RA means for LTA are greater than RTA, and RLCA are greater than LLCA (Figure 2).

Acoustic Analysis

Percent jitter, shimmer, and harmonic-to-noise ratio data for sustained vowel productions, as well as spectral mean and standard deviation (SD) from the LTAS, based on the reading passage, from audio-recordings during the initial assessment, the LEMG session, pre-therapy, and post-therapy are presented in Table 2. Maximum phonation time (MPT) is also provided. In general, no substantive and consistent differences were observed based on acoustic data from the initial assessment (observation 1) to the post-therapy assessment (observation 4) for either twin, nor were differences observed between the twins. In general, all acoustic measures reflected the severity/aperiodicity of the voice signal (i.e., the twins remained severely dysphonic based on acoustic data throughout the case study report).

Voice Therapy

Videolaryngostroboscopic and self-perceived patient handicap data were obtained immediately prior to and following voice therapy. Midmembranous and posterior laryngeal glottal closure was observed to increase following voice therapy. Maximum adduction (closed phase) during modal phonation during videostroboscopic assessment is illustrated in Figures 2 and 3, pre- and post-therapy, respectively. Overall severity scores based on the Voice Handicap Index decreased markedly for Twin 1, less so for Twin 2 (Table 3).

Discussion

This case study provided a unique opportunity to closely examine the distinctions and similarities in elderly MZ twins with a diagnosis of vocal fold bowing and with slight variations in severity. Detailed case histories, diagnostic features, and treatment responsiveness were explored and compared to previous research related to vocal aging, including genetic and environmental factors. Auditory- and visual-perceptual, acoustic, and EMG measures were compared.

In general, the present study findings are consistent with previous research related to vocal aging and familial/heritability factors. The visual-perceptual features of prominent vocal processes, concavity of the vocal fold medial edges, and incomplete glottal closure observed in these MZ twins were consistent with those diagnostic phenomenological features previously described in the literature.[1, 2] Perceptual characteristics of breathiness, weakness, and strain, in addition to severely reduced MPTs were also consistent with previous reports, although the twins in this study were judged to be very severely dysphonic. EMG results, including increased relative amplitude (RA) means and maximums from the present study, particularly those for Twin 1 (i.e., the severely dysphonic twin), are similar to those reported elsewhere.[7]

Previous studies related to genetic/heritability factors indicate that familial traits have been correlated with perceived vocal age, and F_0 and MPT have been shown to be highly correlated in twin pairs, more so for MZ than DZ.[6] Audio-recording data for Twins 1 and 2 from the present investigation support these findings. It should be noted, however, that many twin studies involving the voice rely on registries for participant identification and recruitment, and therefore do not adequately reflect the clinical populations to which findings might be generalized. Additionally, the nature and severity of Twin 1 and 2's dysphonia in the present investigation provide strong evidence for the influence of genetic factors, specifically those related to the aging process. Individuals with voice disorders have been shown to have a family history of dysphonia and shared genetics might partially account for the age-related structural and functional voice changes observed here.[16] Although it is unclear whether or not these changes are the result of the normal aging process or are pathological, the likely genetic influence on bowing observed in Twin 1 and 2 in this study is apparent.

Interestingly, differences between Twin 1 and Twin 2 with respect to environmental factors, as indicated in the psychosocial interview, were observed. Although the twins were reared together and spent the majority of their lifetimes in close geographic proximity, and had similar occupations and social histories, Twin 1 (i.e. extremely severe) had a self-reported substantial reduction in voice use since his wife passed away 10 years prior to his evaluation. Twin 1 also had markedly pathological gastroesophageal reflux, which has been theorized to influence or perhaps worsen dysphonia.[23] It is possible that this reduction in voice use, in addition to differences in reflux and other medical history, contributed to the discrepancy in the relative voice disorder severity, onset and progression of symptoms, and the degrees of dysphonia, bowing, glottal incompetence, and self-perceived voice handicap. In any case, this study provides evidence for the possible influence of environmental factors, in addition to heritability, on vocal fold bowing.

Perhaps the most striking finding in this study was the influence of behavioral voice therapy in improving glottic configuration/closure and reducing self-perceived voice handicap in these twins. On initial evaluation, surgical intervention, including medialization-injection procedures and, in the case of Twin 1, thyroplasty, was considered to be an optimal primary treatment approach due to the severity of the mucosal changes and glottal closure patterns associated with vocal fold bowing. However, the lack of significant functional improvement following surgery prompted the recommendation for additional behavioral intervention. Although admittedly both twins in this study remained severely dysphonic after voice therapy, improvements in glottal closure configuration, as well as marked reductions in self-perceived voice handicap, were observed. This finding was somewhat surprising given that the option of behavioral voice therapy was initially rejected due to the severity of glottal incompetence. However, treatment response was consistent with some previous reports related to surgical and behavioral management of severe vocal fold bowing.[24] Due to significant age-related changes in the distinct organization and layered structure of the vocal folds, as well as general health characteristics that may influence vibratory properties, gross improvement in glottal closure alone might not result in optimal voice improvement in patients with bowing. Thus, regardless of bowing severity, behavioral voice therapy might be considered a primary or adjunctive treatment approach.

In summary, the present case study provides evidence to support both genetic and environmental influences on the aging voice. Although bowing is a frequent visual finding in patients diagnosed with presbylaryngis, incidence rates and evidence of differences between chronological and physiological aging might be the result of environmental factors, such as voice use patterns, in addition to heritability. Future studies should continue to explore the relative contributions of genetic and environmental influences on the aging voice.

Acknowledgements

Genetic testing was funded by Grant R01 DC4336 from the National Institutes of Health.

Figure Captions

Figure 1 (a,b). Maximum closed phase during rigid videolaryngostroboscopy for Twin 1 and Twin 2 during their initial evaluations, May 2005.

Figure 2 (a,b). Maximum closed phase during rigid videolaryngostroboscopy for Twin 1 and Twin 2 pre-therapy, March 2006.

Figure 3 (a,b). Maximum closed phase during rigid videolaryngostroboscopy for Twin 1 and Twin 2 post-therapy, May 2006.

References

1. Honjo I, Isshiki N. Laryngoscopic and voice characteristics of aged persons. *Arch of Otolaryngol*. 1980;106:149-150.
2. Reulbach TR, Belafsky PC, Blalock PD, Koufman JA, Postma GN. Occult laryngeal pathology in a community-based cohort. *Otolaryngol Head Neck Surg.* Apr 2001;124(4):448-450.
3. Hagen P, Lyons G, Nuss D. Dysphonia in the Elderly: Diagnosis and management of Age-Related Voice Changes. *Southern Medical Journal.* 1996;89(2):204-207.
4. Hendridks J, Achenbaum A. A historical development of theories of aging. In Berngston and Schaie, Eds. Handbook of Theories of Aging. New York: Springer; 1999:21-39.
5. Omori K, Slavitt, D, Matos, C, Kojima H, Kacker A, Blaugrund, S. Vocal fold atrophy: Quantitative glottic measurement and vocal function. *Ann Oto Rhino Laryngo.*1997;106: 514-518.
6. Simberg S, Santtila P, Soveri A, Varjonen M, Sala E, Sandnabba K. Exploring genetic and environmental effects in dysphonia: A twin study (in press). *J Speech Lang Hear Res.*
7. Baker K, Ramig L, Luschi E, Smith M. Thryoartenoid muscle activity associated with hypophonia in Parkinson Disease and aging. *Neurology.* 1998;51;1592-1598.
8. Luschei E, Ramig L, Baker K, Smith M. Discharge characteristics of laryngeal single motor units during phonation in young and older adults and in persons with Parkinson Disease. *J Neurophysiology.* 1999; 81: 2131-2139.
9. Malmgen L, Fisher P, Bookman L, Uno T. Age-related changes in muscle fiber types

in the human thyroarytenoid muscle: An immunohistochemical and stereological study using confocal laser scanning microscopy. *Otol Head Neck Surg.* 1999;121(4):441-451.

10. Sato K, Hirano M, Nakashima T. Age related changes of collagenous fibers in the human vocal fold mucosa. *Ann Oto Rhino Laryngo.* 2002;111:15-20.

11. Sato K. Reticular fibers in the vocal fold mucosa. *Ann Oto Rhino Laryngo*. 1998;107:1023-1028.

12. Hixon TJ, Goldman MD, Mead J. Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *J Speech Hear Res*. 1973;16:78-115.

13. Verdolini K, Min Y, Titze I, Lemke J, Brown K, Mersbergen M, Jiang J, Fisher K. Biological mechanisms underlying voice changes due to dehydration. *J Speech Lang Hear Res.* 2002;45(2):268-281.

14. Butler J, Hammond T, Gray S. Gender-related differences of hyaluronic acid distribution in the human vocal fold. *Laryngoscope*. 2001;111:907-911.

15. Olson-Ramig L, Gray S, Baker K, Corbin-Lewis K, Buder E, Luschei E, Coon H, Smith M. The aging voice: A review, treatment data, and familial and genetic perspectives. *Folia Phon Logo*. 2001; 53:252-265.

16. Roy N, Merrill RM, Thibeault S, Parsa RA, Gray SD, Smith ME. Prevalence of voice disorders in teachers and the general population. *J Speech Lang Hear Res.* 2004; 47: 281-293.

17. Przybyla BD, Horii Y, Crawford MH. Vocal fundamental frequency in a twin sample: Looking for a genetic effect. *J Voice.* 1992:6;261-266.

18. Decoster, W, Van Gysel A, Vercammen J, Debruyne F. Voice similarity in identical twins. *Acta Oto Rhino Laryngo Belgica*. 2001;55:49-55.

19. Van Lierde KM, Vinck B, De Ley S, Clement G, Van Cauwenberge P. Genetics of voice quality characteristics in monozygotic twins: A multiparameter approach. *J Voice*. 2005;19:511-518.

20. Gorman S. Senile Laryngis. In Stemple, Ed. Voice Therapy: Clinical Studies. California: Singular Publishing Group; 2000:192-188.

21. Jacobson B, Johnson A, Grywalski C, Silbergleit A, Jacobson G, Benninger M, Newman, C. The voice handicap index (VHI): development and validation. *Am J Speech Lang Path.* 1997;6:66-70.

22. Tanner K, Roy N, Ash A, Buder EH. Spectral moments of the long-term average spectrum: Sensitive indices of voice change following voice therapy? *J Voice*. 2005;19: 211-221.

23. Koufman J, Milan R, Amin MD, Panetti M. Prevalence of reflux in 113 consecutive patients with laryngeal and voice disorders. *Oto Head Neck Surg*. 2000;123:385-388.

24. Lu F, Lundy D, Casiano R, Xue J. Vocal evaluation of thyroplasty type I in the treatment of nonparalytic glottal incompetence. *Ann Oto Rhino Laryngo*. 1998;118(4):113-119.

Table 1

Relative Amplitude (RA) means and maximums for prephonatory and phonatory segments during sustained /ɑ/ and laryngeal DDKs during normal pitch and loudness (NPNL), soft phonation, and loud phonation.

Task	**Twin 1**				**Twin 2**			
	RTA Mean (Max)	LTA Mean (Max)	RLCA Mean (Max)	LLCA Mean (Max)	RTA Mean (Max)	LTA Mean (Max)	RLCA Mean (Max)	LLCA Mean (Max)
Prephonatory (vowel)								
NPNL	1.2 (4.4)	3.5 (11.8)	28.9 (53.4)	2.2 (5.2)	7.4 (28.4)	34.3 (66.2)	19.9 (33.0)	18.7 (55.5)
Soft	0.7 (1.3)	1.4 (8.6)	11.7 (19.7)	1.9 (3.1)	4.0 (6.5)	20.7 (32.3)	11.7 (28.1)	7.6 (15.3)
Loud	13.9 (59.1)	23.6 (80.6)	23.2 (83.2)	6.4 (30.0)	7.9 (33.8)	22.4 (33.1)	32.1 (55.7)	15.6 (31.5)
Phonatory (vowel)								
NPNL	1.0 (2.1)	2.2 (9.9)	11.7 (29.7)	2.3 (4.2)	12.0 (15.7)	25.6 (56.9)	26.1 (36.5)	11.1 (18.8)
Soft	0.6 (0.9)	0.4 (1.2)	6.8 (17.2)	1.6 (4.4)	15.7 (30.8)	37.5 (64.3)	22.7 (49.2)	14.3 (38.1)
Loud	3.7 (41.3)	5.9 (35.2)	15.4 (37.3)	2.6 (5.3)	14.0 (32.7)	24.9 (37.5)	28.2 (52.1)	18.5 (35.2)
Prephonatory (DDK)	5.9 (47.9)	10.2 (75.5)	15.4 (31.6)	2.8 (4.5)	7.6 (18.2)	8.9 (23.0)	6.5 (12.6)	3.2 (7.2)
Phonatory (DDK)	1.6 (5.3)	2.3 (13.6)	12.6 (35.9)	3.6 (6.4)	13.4 (24.1)	16.3 (25.2)	10.9 (25.1)	5.5 (9.6)

Table 2
MPT, percent jitter, shimmer, and harmonic-to-noise ratio, as well as spectral mean and SD of the LTAS based on audio-recordings during the initial assessment, LEMG session, pre-therapy, and post-therapy.

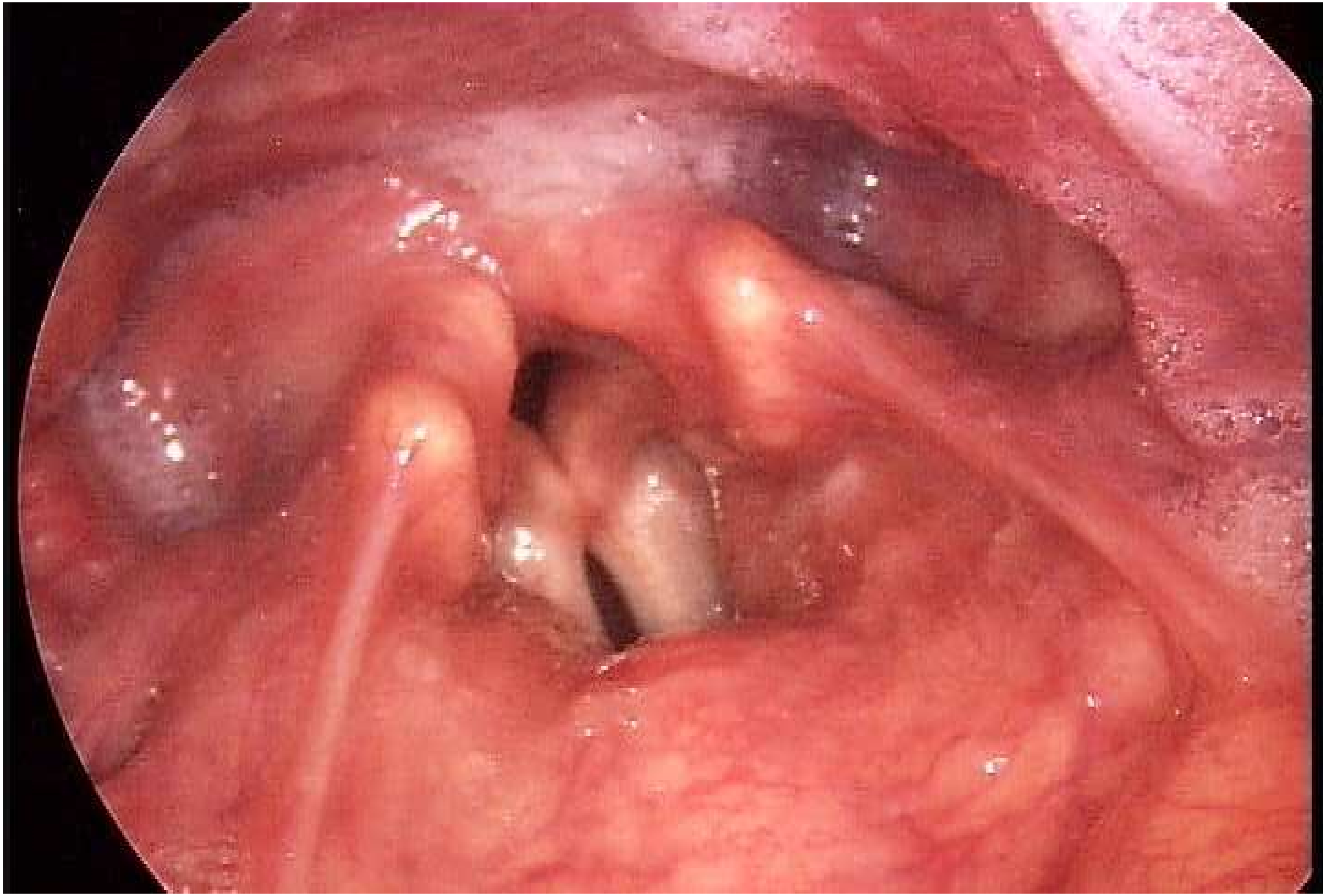
Measure	**Twin 1**				**Twin 2**			
	5/05	1/06	3/06	5/06	5/05	1/06	3/06	5/06
F_0 (reading)	150	165	155	136	137	178	158	157
MPT (s)	1.0	1.0	1.3	1.1	1.7	3.6	3.4	3.8
% Jitter	4.1	5.2	2.6	3.8	3.7	1.2	1.4	2.0
% Shimmer	6.4	8.6	8.6	6.8	9.5	12.0	4.8	3.8
HNR	0.2	0.2	0.2	0.2	0.2	0.2	0.2	0.2
Spectral Mean (Hz) (LTAS)	367.5	424.0	532.72	411.64	310.5	270.5	295.6.7	325.9.0
Spectral SD (LTAS)	4.16	827.5	1070.0	693.2	382.0	287.8	456.0	527.6

Table 3

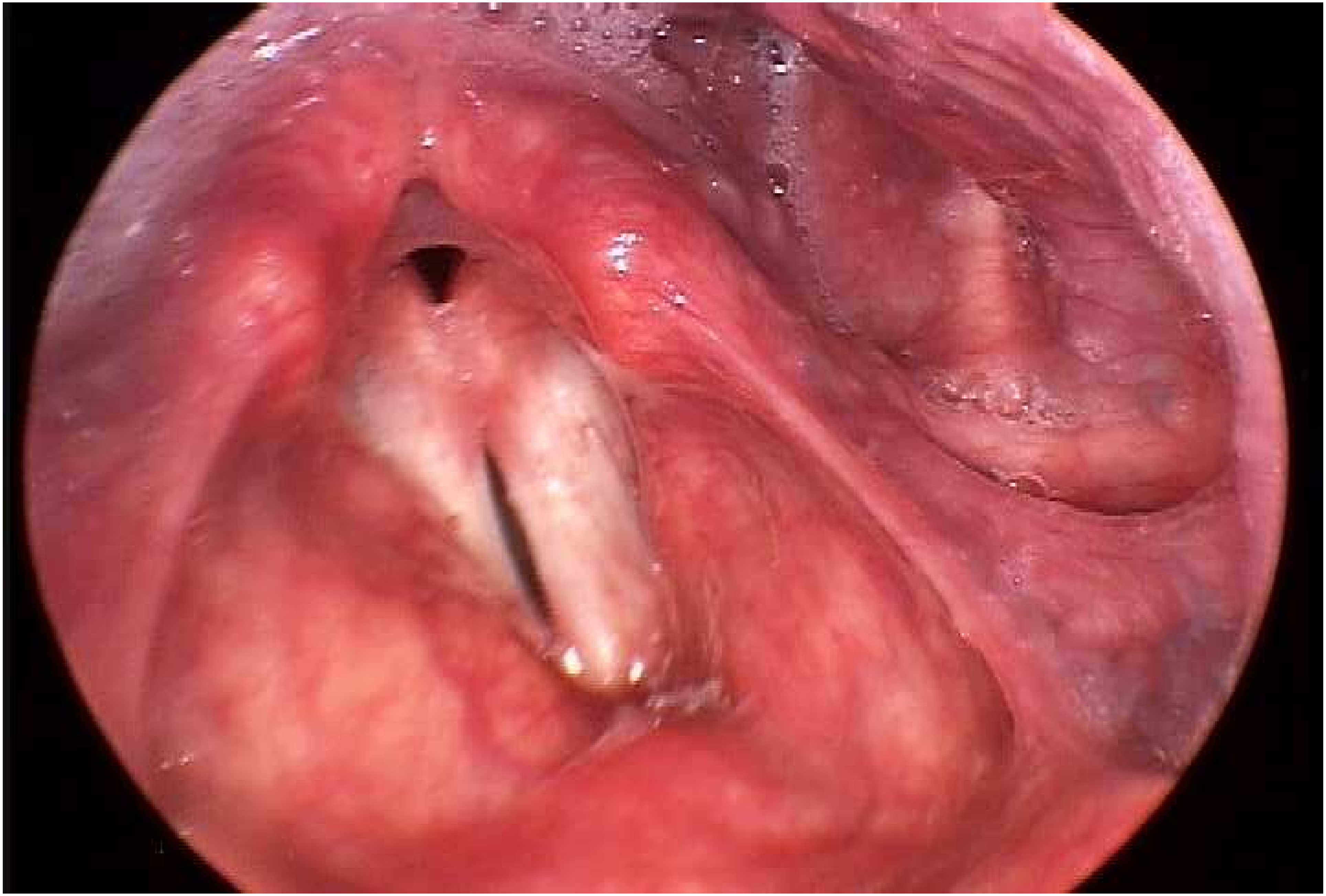
Voice Handicap Index scores during the LEMG session, pre-therapy, and post-therapy.

Date	**Voice Handicap Index: Total Score**	
	Twin 1	**Twin 2**
1/06	60	73
3/06	80	67
5/06	76	18

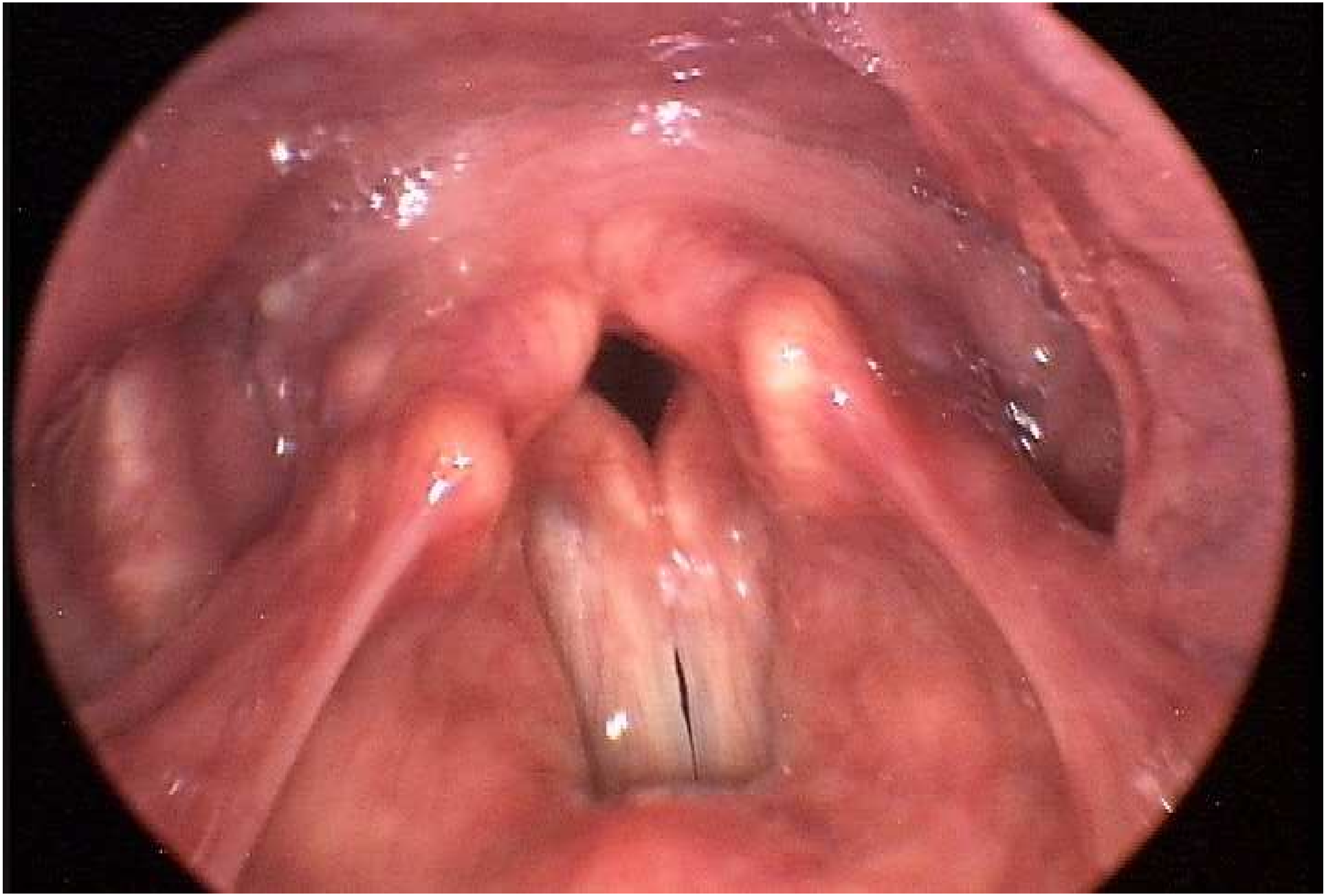
Figure(s)
Click here to download high resolution image



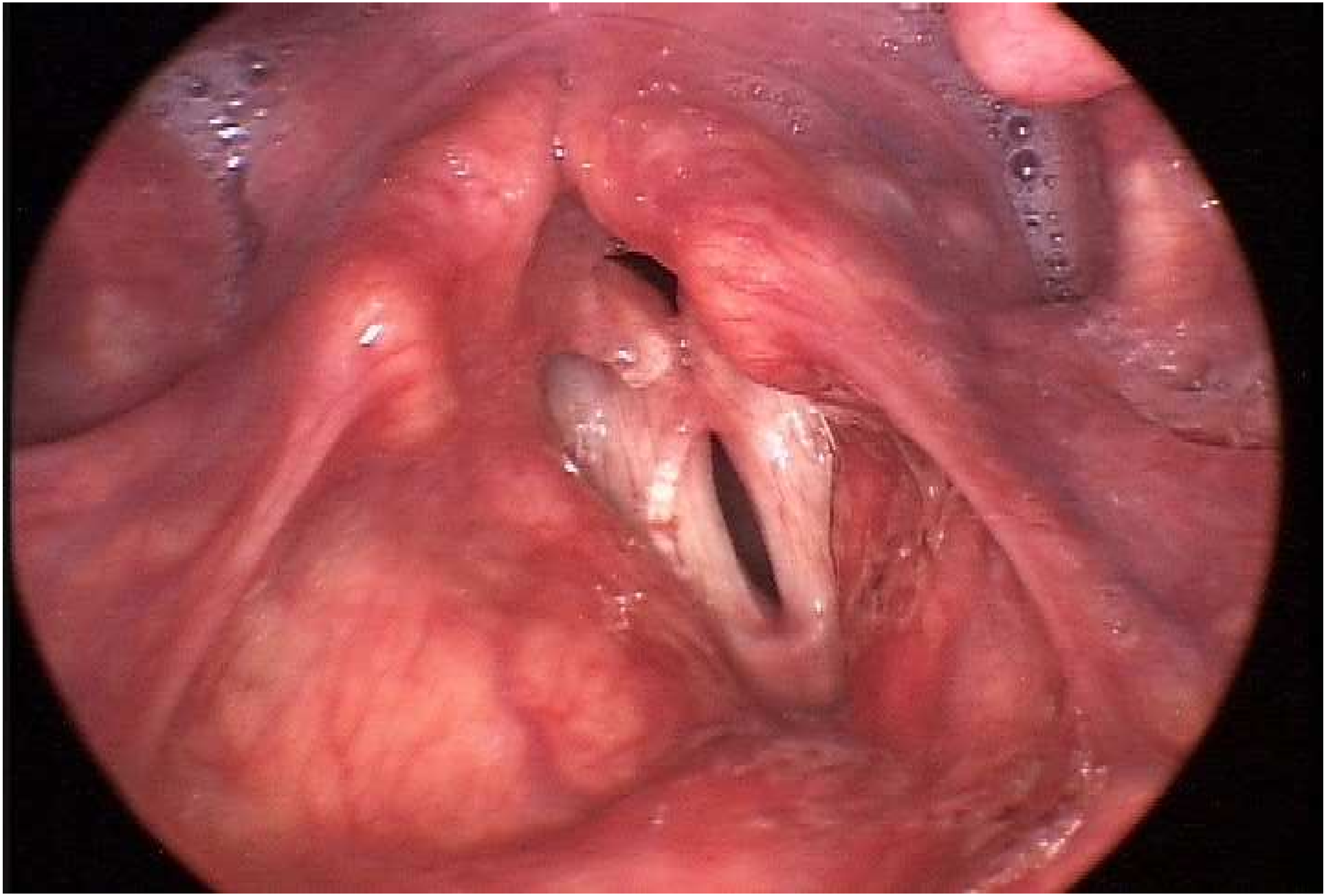
Figure(s)
Click here to download high resolution image



Figure(s)
Click here to download high resolution image



Figure(s)
Click here to download high resolution image



Figure(s)
Click here to download high resolution image

Figure(s)
Click here to download high resolution image