Published in final edited form as:

Otolaryngol Head Neck Surg. 2018 December ; 159(6): 1020–1027. doi:10.1177/0194599818800462.

Outcomes in Bilateral Vocal Fold Immobility: A Retrospective Cohort Analysis

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Abstract

Objective.—To test the hypothesis that the etiologies of bilateral vocal fold mobility impairment (BLVFI), bilateral vocal fold paralysis (BVFP), and posterior glottis stenosis (PGS) have distinct clinical outcomes. To identify patient-specific and procedural factors that influence tracheostomy-free survival.

Study Design.—Retrospective cohort study.

Setting.—Johns Hopkins Medical Center from 2004 to 2015.

Subjects and Methods.—Case series with chart review of 68 patients with PGS and 17 patients with BVFP. Multiple logistic regression analysis determined factors associated with airway prosthesis dependence at last follow-up and the procedural burden (defined as number of operative procedures per year).

Results.—PGS comprised the majority of BLVFI (76%). PGS injury arose primarily after endotracheal intubation (91%), while BVFP most commonly was due to iatrogenic surgical injury

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Disclosures

The content of this manuscript was presented as an oral presentation at Triological Society Combined Sections Meeting; January 19, 2017; New Orleans, Louisiana.

Competing interests: Lee M. Akst, Olympus, Inc (advisory board/consulting); Alexander T. Hillel, Olympus USA (consulting). Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

to bilateral recurrent laryngeal nerves (88%, $P < .001$). Overall in BLVFI, 66% were tracheostomy free at last follow-up (62% in PGS, 82% in BVFP). Of those who underwent an operative intervention to be decannulated, 88% were decannulated (90% PGS, 80% BVFP). Patients with PGS required higher procedural burden to achieve decannulation compared with the BVFP cohort (3.1 ± 5.2 vs 0.71 ± 1.4 , $P = .002$). In multivariate analysis of PGS, smoking was a risk factor for tracheostomy dependence ($P = .026$).

Conclusions.—BLVFI is primarily an iatrogenic complication. There are high rates of tracheostomy dependence in BLVFI, with procedural intervention needed for decannulation. Compared with BVFP, patients with PGS had a higher procedural burden overall and to achieve decannulation. Patients with PGS should be counseled that smoking, a modifiable risk factor, may increase the risk of tracheostomy dependence.

Keywords

bilateral vocal fold paralysis; vocal fold immobility; bilateral vocal fold immobility; glottic stenosis; posterior glottic stenosis; outcomes in bilateral vocal fold immobility

Bilateral vocal fold motion impairment (BLVFI) results from structural or neurologic injury to the cricoarytenoid joint complex, leading to compromised glottic mobility, with varying degrees of ventilatory obstruction. Neural injury (bilateral vocal fold paralysis [BVFP]) to the complex set of intrinsic laryngeal muscles results in reduced or absent vocal cord motion, with impaired abduction limiting the glottic airway. The most common cause of BVFP is surgery, usually thyroidectomy, and malignancy.¹ On the other hand, fibrotic remodeling and stenotic contracture of the posterior glottic mucosa, often involving the cricoarytenoid joint, similarly results in limitation of laryngeal luminal cross-sectional area, causing posterior glottic stenosis (PGS). Predictors of PGS include long-term intubation, diabetes, ischemic conditions, and large endotracheal tubes.²

The similar characteristics of the luminal compromise in BVFP and PGS have a significant impact on respiration, phonation, deglutition, and overall quality of life.³ Given the anatomic nature of airflow limitation, nearly all therapies aimed at improving airflow are surgical. However, surgical therapy remains an unsatisfactory compromise between voice, breathing, and swallowing. Current techniques restore airway patency at the cost of glottic closure, vocal quality, and at times swallowing. Most clinical treatment algorithms use minimally invasive initial treatment options, including endoscopic posterior cordotomy, arytenoidectomy, or suture lateralization.^{4,5} When these procedures fail to maintain a sufficiently patent airway, more invasive procedures such as a posterior cricoid split, laryngoplasty, or tracheostomy are undertaken to maintain a permanent airway and preserve ventilatory function.^{6–9}

Although both BVFP and PGS present with airway compromise, the unique pathophysiology driving anatomic compromise in the distinct patient populations has significant implications for developing patient-centered treatment. While a proportion of patients with bilateral vocal fold immobility are managed with tracheostomy, the rates of tracheostomy placement, dependence, and relative procedural benefit vs inherent procedural risk in bilateral vocal fold immobility patients have not been adequately analyzed.^{10–12}

Moreover, with the exception of a select few studies, the efficacy of procedural intervention in achieving decannulation is not well known.^{11,13,14} The present study was undertaken to test the hypothesis that BVFP and PGS have differing etiologies, unique natural histories, and distinct clinical outcomes. We also aimed to identify patient-specific and surgical techniques that influence tracheostomy-free survival. Contrasting the 2 etiologies, we hypothesized that patients with PGS had greater comorbidities that contributed to the tendency to form glottic fibrosis and were more likely to remain tracheostomy dependent. Finally, we aimed to assess various PGS staging systems (Bogdasarian, Sandhu) as predictors of tracheostomy outcomes.

Methods

Institutional review board approval was obtained from the Johns Hopkins University Institutional Review Board (NA00081469) prior to beginning this study. Medical records were examined from 2004 to 2015. All patients with glottic stenosis or BVFP were identified by *International Classification of Diseases, Tenth Revision* (ICD-10) codes J38.6 (*International Classification of Diseases, Ninth Revision* [ICD-9]: 478.74) and J38.02 (ICD-9: 478.30) and reviewed. Patients included in the study were 18 years old and had documented glottic narrowing on flexible or direct laryngoscopy that was the result of bilateral vocal fold paralysis or glottic stenosis. For assessment of outcomes, patients were required to have at least 2 follow-up visits. Patients with isolated subglottic or supraglottic stenosis independent of BLVFI were excluded from this study.

Data Collected

Patient characteristics (age, sex, race, follow-up duration, zip codes) and comorbidities were abstracted from the medical record. Patients were binned into poverty quintiles based on the percentage of residents living below the poverty line of their respective zip code based on the US Census 2010.¹⁵ Records were reviewed for etiology of glottic compromise, treatment approach (ie, endoscopic, open, and specific procedural type), and number of procedures. Comorbidities included gastroesophageal reflux disease (GERD), obstructive sleep apnea, diabetes mellitus, hypertension, chronic obstructive pulmonary disease (COPD), cerebrovascular accident, myocardial infarction, coronary artery disease, and smoking status. Operative details and office visit details, including flexible laryngoscopy, were recorded. Patients were staged with the established Bogdasarian and Sandhu classification systems as previously described.^{3–5} Patient tracheostomy status at initial presentation, time of decannulation, if decannulation occurred, and tracheostomy status at last follow-up were recorded.

Procedures

Treatments for BLVFI included cordotomy (with or without arytenoidectomy), suture lateralization, and posterior cricoid split. The treatment algorithm consisted of initial endoscopic evaluation for all patients. Patients were treated at the discretion of their surgeon following documentation and classification of stenosis.

Outcomes

Airway prosthesis status (either tracheostomy or T-tube) at last follow-up visit was the primary outcome. This represented failure of surgical management to correct laryngeal compromise. The procedural burden to accomplish decannulation (represented by the total number of operative procedures over the treatment course) was a secondary outcome. Operative interventions were defined as any procedure assessing or managing the laryngeal compromise. The number of procedures per year was calculated by dividing the number of procedures that the patient underwent during the study period by the total follow-up duration in years. Successful decannulation following specific surgical procedures was recorded.

Statistical Analysis

All statistical analysis was performed using STATA 12.0. (StataCorp, College Station, Texas).¹⁶ When testing between the 2 subgroups of BLVFI, continuous, nonparametric variables were analyzed using a Mann-Whitney *U* test, and χ^2 tests were used for categorical variables. Analysis of variance (ANOVA) tests were used to compare parametric continuous variables between etiology-specific groups, and Kruskal-Wallis 1-way ANOVA was used for nonparametric variables. A Holm-Sidak post hoc pairwise multiple-comparison analysis was used to further identify statistically significant subgroups for ANOVA tests, and Dunn's test of multiple comparison was used for post hoc analysis of Kruskal-Wallis tests. For multivariate regression models, all outcome variables were initially tested with bivariate analysis, and significant variables ($P < .20$) were included in multivariate analysis. Statistically significant associations were tested for collinearity, and relevant variables were subsequently included in the multivariate regression model. *P* values of less than .05 were considered statistically significant.

Results

Demographic characteristics and comorbid conditions of study participants stratified by PGS or BVFP status are shown in Table 1. BVFP participants were significantly older than patients with PGS (60 ± 13.7 vs 48.5 ± 16.1 , $P = .002$). There were no significant differences between the BVFP and PGS groups in sex, race, body mass index (BMI), socioeconomic status, or comorbidities, with the exception of motor vehicle accidents (MVAs), which were more common in the PGS cohort (25% in PGS cohort vs 0% in the BVFP cohort, $P = .011$). Participants in the PGS cohort had significantly increased procedures per year compared with the BVFP cohort (3 ± 5 vs 0.7 ± 1 , $P = .040$). Tracheostomy dependence at last follow-up was not significantly different between the 2 cohorts. Intubation was the most common cause of PGS (91%), while surgical nerve injury was the most common cause of BVFP (88%) (Figure 1), which was significantly different between the 2 groups ($P < .001$). Of the surgical nerve injury subgroup of BVFP, 63% of iatrogenic injuries occurred during thyroid surgery. None of the staging systems used to classify PGS predicted tracheostomy-free status at last follow-up.

To investigate patient-specific risk factors for decannulation failure, we stratified both PGS and patients with BVFP according to airway prosthesis status. Bivariate analysis showed that a significantly higher proportion of smokers were found to be airway prosthesis dependent

(61% prosthesis dependent vs 28% prosthesis free, $P = .002$) in the PGS cohort (Table 2). African American ethnicity (42% prosthesis dependent vs 25% prosthesis free; $P = .032$) was also negatively associated with dependence on an airway prosthesis. Factors that were significantly associated with prosthesis dependence were included in a multivariate model (Table 3). Multivariate analysis included tobacco usage, age, ethnicity, sex, BMI, COPD, and hypertension as variables for examining prosthesis dependence at last follow-up. For PGS, only tobacco use (odds ratio [OR], 0.23; 95% confidence interval [CI], 0.06–0.84; $P = .026$) remained associated with tracheostomy dependence when controlling for factors significant on bivariate analysis. In the BVFP cohort, only high Charlson Comorbidity Index (CCI) (1.7 ± 1.6 prosthesis dependent vs 1.2 ± 0.71 prosthesis free) was significantly associated with tracheostomy dependence. However, this was not significant on multivariate analysis.

To investigate procedure-specific success for decannulation, we combined both cohorts to study all patients with BLVFI according to airway prosthesis status. Post hoc analysis was performed to calculate the success rate of the procedure leading to decannulation in the patient cohort who had a tracheostomy at the beginning of the study period for their treatment for BLVFI. Figure 2 depicts the procedures and results for all patients who began the study with a tracheostomy. Only 1 patient of the 18 who underwent no procedural intervention was decannulated without procedural intervention (6%), compared to 92% (23/25) who were decannulated after 1 or more procedural interventions ($P < .001$). Assessing specific procedures, there was a 22% (2/9) decannulation rate for patients who underwent excision of scar, all of whom had PGS. Six of the 7 patients who failed to be decannulated with scar excision went on to have a cordotomy. A total of 20 patients had a cordotomy, which was 60% (12/20) effective at decannulation. The 8 patients who were unable to be decannulated with cordotomy went on to have framework surgery, which was 90% successful at decannulation (9/10) for BLVFI in total. Figure 1 stratifies the patients in each cohort of tracheostomy-dependent patients.

The extent of surgical history was assessed in each cohort of tracheostomy-free patients. Of 32 patients who were not tracheostomy dependent at initial presentation in the PGS cohort, 50% underwent procedural intervention. Of 10 patients who were not tracheostomy dependent at initial presentation in the BVFP cohort, 30% underwent a procedural intervention to enlarge the narrowed glottic airway. In addition, 1 patient in the BVFP group underwent a tracheostomy for treatment.

Discussion

BLVFI is a devastating disease that results in loss of laryngeal function with resultant impacts on voice and breathing. In this study, BLVFI was separated into structural and neural etiologies in PGS and BVFP, respectively, to evaluate causes of injury, procedural outcomes, and comorbidities that affect outcomes for each cohort. A subgroup analysis examined tracheostomy decannulation for those patients who had a tracheostomy at the beginning of the study period; however, all patients, regardless of tracheostomy status, were included in the study. Similar to other studies, the overwhelming cause of BVFP was due to surgical injury, while PGS resulted from endotracheal intubation almost exclusively.^{4,5,17,18} At our institution, PGS was far more common, as the rate of PGS was over 3 times that of

BVFP. This higher than expected ratio can be attributed to the status of the study institution as a broad tertiary care referral center. In addition, this study demonstrated the high rate of tracheostomy in patients with BLVFI (51%, 43/85) and the need for procedural intervention to successfully decannulate patients. Other studies have shown similar tracheostomy rates, ranging from 34% to 46%.^{2,19,20} BFVP and PGS represent distinct disease entities with differing wound-healing physiology that contribute to respiratory impairment. In this study, patients with PGS had a significantly higher procedural burden compared with patients with BVFP and trended toward a greater number of patients who were tracheostomy dependent at last follow-up. Finally, patients with PGS who used tobacco were significantly less likely to be tracheostomy free than patients with PGS who did not have a smoking history.

The differences between the BFVP and PGS cohorts manifest in the comorbidities that affected tracheostomy status. In the relatively small BVFP group, diabetes and a high CCI were univariate predictors of tracheostomy dependence that did not remain significant in the multivariate model. These comorbidities may contribute to poor wound healing following endoscopic or open procedures to increase the glottic airway, or perhaps because surgical attempts to decannulate were avoided in these sicker patients. In the PGS cohort, tobacco use and African American race proved to be significant variables predicting tracheostomy dependence in univariate analysis. In the multivariate model, only tobacco use remained significant. The lower overall rate of tracheostomy-free survival in PGS tobacco users may also be due to smoking-induced damage to the laryngeal epithelium. Tobacco use was the only risk factor that remained significantly associated with tracheostomy status at last follow-up in multivariate analysis. In COPD, chronic tobacco use results in pathologic wound repair, depleted vascular supply to the mucosa, and increased inflammation, making the lower airway mucosa more susceptible to injury.^{21–23} We hypothesize that, as a majority of airflow that reaches the lower airways crosses the posterior glottis, it is to be expected that the mucosa in the posterior glottis would be exposed to similar levels of tobacco smoke.²³ The mechanism by which tobacco smoke-induced damage primes the mucosa for intubation injury remains to be elucidated.

Assessment of the outcomes of procedural intervention in our cohort demonstrates that patients with BLVFI with a tracheostomy were unlikely to be decannulated without surgery to open the glottic airway. Furthermore, this study shows decannulation rates increased with more invasive surgical procedures. All but 1 patient in both cohorts required procedural intervention to achieve decannulation in all patients who had tracheostomy during the study period. In addition, this study demonstrated that a stepwise approach is used during surgical planning to achieve decannulation. Patients who failed excision and dilation procedures were most commonly decannulated with cordotomy, and those who failed decannulation with cordotomy were most commonly successfully decannulated with open-framework surgery. While cordotomy and/or partial arytenoidectomy was the most commonly performed procedure, it was not as successful in decannulation as cricoid split and transcervical laryngoplasty with laryngeal stent placement. These results support most airway surgeons' progression toward more invasive procedures if earlier and less invasive procedures are not successful at achieving decannulation.

The procedural rate, or number of procedures per year, also differed between cohorts, with the BVFP cohort having significantly fewer surgeries than the PGS cohort. Furthermore, for patients who were decannulated, BVFP patients had significantly fewer procedures to achieve decannulation than PGS patients who were successfully decannulated. This finding may be due to the neurologic etiology, which has less structural remodeling to the posterior glottis and cricoarytenoid joints than the resultant fibrosis in PGS. Because of this, we hypothesized that the BVFP cohort would have higher rates of decannulation and tracheostomy-free survival, which was shown to be the case in this study.²⁴

The disparity between rates of surgical intervention in the 2 cohorts may be explained by a few factors. Primarily, the BVFP cohort was relatively small. The small size of the BVFP cohort contributes to the low rate of surgical intervention in the BVFP cohort. One other factor that was investigated was whether the patients with BVFP had shorter follow-up times and therefore may have not had surgery at the time of study completion. However, that hypothesis was disproven as the mean follow-up time for the BVFP cohort (42.3 ± 42 months) was higher than that for the PGS cohort (32.3 ± 46.9 months). In addition, excision procedures were not an option for the BVFP cohort, limiting the opportunities for surgical intervention.

A few other hypotheses were disproven in this study. We hypothesized that patients with PGS (relative to the BVFP group) would have greater comorbidities, especially those that predispose to pathologic wound healing such as diabetes. However, there was no association with BMI or CCI in tracheostomy-free survival in the PGS cohort. Socioeconomic status and demographics are hypothesized to be a factor in PGS because of the increased number of comorbidities and higher smoking prevalence in lower socioeconomic groups, which would theoretically lead to worse outcomes, poor wound healing, and lower baseline health.^{25,26} Despite the belief that differences in race, and subsequently genetics, may contribute to altered inflammatory responses to wound healing, race was not associated with outcome on multivariate analysis in this study.²⁷ In addition, none of the PGS staging systems were predictive of outcomes.^{23,28,29} While cohort size may be a factor, this suggests a need for a predictive staging system in PGS.

This study has several additional limitations. First, the retrospective nature of our study design limits drawing causal relationships between risk factors and outcomes, particularly given the small size of the BVFP cohort. Second, we included only patients with glottic stenosis or BVFP and their treatment courses occurring in 1 institution. The status of the hospital as a broad tertiary care referral institution may skew the demographics of the treatment-seeking population. Third, intubation history details of the particular hospitalization creating PGS are difficult to obtain given the delayed nature of referrals to tertiary care hospitals after initial injury, the unclear nature of which intubation was directly associated with damage to the glottis, and poor documentation of both tube sizes and time of extubation. In addition, one outcome that was not assessed in this study included perceptual voice and subjective quality-of-life voice outcomes, which should be accounted for when judging the success of glottic airway surgery. While studies have looked at voice outcomes following cordotomy, voice outcomes have not been directly compared between posterior cricoid split and other procedures that improve the glottic airway.³⁰ Finally,

a limitation in determining the procedural algorithm leading to decannulation was that patients who presented to the study institution may have received procedural intervention prior to presentation at outside institutions. Capturing these algorithms accurately requires prospective analysis. Further prospective studies examining decannulation rates and voice outcomes will better establish long-term treatment plans for newly diagnosed patients with BLVFI.

In conclusion, this study explores surgical treatment efficacy for patients with BLVFI and provides important baseline outcome data with one of the largest series of adult BLVFI patient cohorts to date. The health care impact of BLVFI is evident with high rates of tracheostomy dependence and the need for surgery to successfully decannulate many of these patients. While a progression from least to most invasive surgery was seen, more invasive procedures were more successful at decannulation. Compared with BVFP, patients with PGS had a higher procedural burden overall and required more surgeries to achieve decannulation. Patients with PGS should be counseled accordingly to manage expectations, and the type of procedure chosen for each patient should be targeted to maximize opportunity for decannulation.

Acknowledgments

Sponsorships: None.

Funding source: Research reported in this publication was supported by the National Institute of Deafness and Other Communication Disorders of the National Institutes of Health under award number 1K23DC014082 and NIDCD: 5K23DC013569. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. In addition, research reported in this publication was supported by the Triological Society and American College of Surgeons.

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Figure 1. Etiology of injury. Comparison of percentage of each cohort caused by specific etiologies. BVFP, bilateral vocal fold paresis; PGS, posterior glottic stenosis. $P<.001$ between cohorts.

Figure 2.

Decannulation outcomes stratified by surgical intervention. The total number of patients who underwent the procedure for decannulation in the excision/dilation group, cordotomy group, and open framework surgery group was 9, 20, and 10, respectively. BVFP, bilateral vocal fold paresis; PGS, posterior glottic stenosis.

Table 1.Comparison of Glottic Stenosis and Bilateral Vocal Fold Paralysis Patients.^a

Patient Characteristics	PGS (n = 68)	BVFP (n = 17)	P Value ^b
Age, mean ± SD, y	48.5 ± 16.1	60.0 ± 13.7	.006
Sex, male	34 (50.0)	7 (33.3)	.180
Race			
White	43 (63.2)	10 (47.6)	.158
African American	20 (29.4)	6 (28.6)	
Asian	2 (2.9)	1 (4.8)	
Other	3 (4.4)	4(19.1)	
BMI, mean ± SD, kg/m ²	31.9 ± 16.1	26.7 ± 9.3	.098
Comorbidities			
Charlson Comorbidity Index, mean ± SD	1.1 ± 1.4	1.2 ± 1.2	.733
GERD	14 (20.6)	4(19.1)	.855
OSA	6 (8.8)	3(14.3)	.455
DMII	18 (26.4)	5 (23.8)	.834
HTN	28 (41.2)	8 (31.8)	.764
COPD	12(17.6)	4(19.1)	.862
CVA	7(10.3)	2 (9.5)	.934
MI	8(11.8)	0(0)	.102
CAD	11 (16.2)	2 (9.5)	.464
Burn/electrocution	6 (8.8)	0(0)	.159
MVA	17 (25.0)	0(0)	.011
Autoimmune disease	9 (13.2)	1 (4.8)	.282
Psychiatric disease	19 (27.9)	2 (9.5)	.082
Tobacco use	22 (32.4)	4 (22.2)	.237
Stenosis characteristics Location			
Glottis only	37 (54.4)	14 (66.7)	.381
Multiple sites	31 (45.6)		
Associated SGS	31 (45.5)		
Bogdasarian staging	(n = 66) ^c		

Patient Characteristics	PGS (n = 68)	BVFP (n = 17)	P Value ^b
Class 1	1 (1.5)		
Class 2	9(13.6)		
Class 3	7(10.6)		
Class 4	49 (74.2)		
Sandhu staging	(n = 66) ^c		
Class 1	12(18.2)		
Class 2	39 (59.1)		
Class 3	2 (3.0)		
Class 4	13 (19.7)		
Poverty quintile	(n = 65) ^d	(n=17)	.289
Q1	11 (16.9)	7 (41.2)	
Q2	14 (21.5)	3 (17.6)	
Q3	13 (20.0)	3 (17.6)	
Q4	14 (21.5)	2 (11.8)	
Q5	13 (20.0)	2 (11.8)	
Procedural outcomes			
No. of procedures per year, Mean ± SD	3.1 ± 5.2	0.71 ± 1.4	.002
Tracheostomy free at first presentation	32(47.1)	10 (58.8)	.801
Tracheostomy free at last follow-up ^e	42 (61.8)	14 (82.3)	.056
Decannulation ^f	(n = 36) 19 (52.8)	(n = 8) ^g 5 (62.5)	.21

Abbreviations: BMI, body mass index; BVFP, bilateral vocal fold paralysis; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DMII, type II diabetes mellitus; GERD, gastroesophageal reflux disease; HTN, hypertension; MI, myocardial infarction; MVA, motor vehicle accident; OSA, obstructive sleep apnea; PGS, posterior glottic stenosis; SD, standard deviation; SGS, subglottic stenosis.

^aValues are presented as number (%) unless otherwise specified.

^b $P < .05$ considered significant (significant values are bolded).

^cTwo patients did not have adequate information for staging by the Bogdasarian and Sandhu staging systems.

^dThree patients did not have adequate information for stratification by poverty quintile.

^eTracheostomy free at last follow-up included all patients in the study at last follow-up.

The decannulation percentage was calculated by dividing the number of patients in each cohort who safely had their tracheostomy tubes removed divided by the number of patients who had a tracheostomy at any time during the study.

One patient in the BVFP cohort who did not have a tracheostomy at first presentation underwent tracheostomy as part of his or her treatment.

Table 2.
Comparison of Factors Associated with Tracheostomy-Free Survival on First Presentation.^a

Characteristic	PGS		BVFP	
	Tracheostomy Free (n = 32)	Tracheostomy Dependent (n = 36)	Tracheostomy Free (n = 10)	Tracheostomy Dependent (n = 7)
Age, mean ± SD, y	47.8 ± 15.7	49.1 ± 16.0	59.8 ± 15.6	58.5 ± 18.5
Sex, male	14 (43.8)	22 (61.1)	2 (20.0)	2 (28.6)
Race				
White	24 (75.0)	16 (44.4)	4 (40.0)	2 (28.6)
African American	8 (25.0)	15 (41.7)	1 (10.0)	4 (57.1)
Asian	0(0)	2 (5.6)	1 (10.0)	0(0)
Other	0	3 (8.3)	4 (40.0)	1 (14.2)
BMI, mean ± SD, kg/m ²	33.4 ± 18.4	29.3 ± 11.5	27.8 ± 3.2	25.4 ± 9.1
Comorbidities				
Charlson Comorbidity Index, mean ± SD	1.2 ± 1.2	1.3 ± 1.6	1.2 ± 0.71	1.7 ± 1.6
GERD	6 (18.8)	8 (22.2)	1 (10.0)	2 (28.5)
OSA	3 (9.4)	4 (11.1)	1 (10.0)	2 (28.5)
DMII	9 (28.1)	8 (22.2)	0	3 (43.8)
HTN	11 (34.3)	19 (52.8)	1 (10.0)	4 (57.1)
COPD	4 (12.5)	10 (27.8)	0	2 (28.5)
CVA	4 (12.5)	3 (8.3)	2 (20.0)	1 (14.2)
MI	4 (12.5)	3 (8.3)	0	0
CAD	8 (25.0)	4 (11.1)	0	1 (14.2)
Burn/electrocution	3 (9.4)	3 (8.3)	0	0
MVA	4 (12.5)	14 (38.9)	0	0
Autoimmune disease	3 (9.4)	5 (14.0)	1 (10.0)	0
Psychiatric disease	11 (34.4)	10 (27.8)	2 (20.0)	0
Tobacco use	9 (28.1)	22 (61.1)	0	3 (43.8)
Bogdasarian staging (n = 66)				
Class 1	1 (2.7)	0 (0)		
Class 2	5 (15.6)	3 (8.3)		

Characteristic	PGS			BVFP		
	Tracheostomy Free (n = 32)	Tracheostomy Dependent (n = 36)	P Value ^b	Tracheostomy Free (n = 10)	Tracheostomy Dependent (n = 7)	P Value ^b
Class 3	4 (12.5)	4 (11.1)				
Class 4	22 (68.8)	29 (80.6)				
Sandhu staging (n = 66)			.818			
Class 1	7 (21.9)	5 (13.9)				
Class 2	18 (56.3)	23 (63.9)				
Class 3	1 (3.1)	1 (2.8)				
Class 4	6 (18.8)	7 (19.4)				
Poverty quintile	(n = 32)	(n = 36)		(n = 10)	(n = 7)	
Q1	7 (22.0)	3 (8.3)	.419	5 (50.0)	3 (44.4)	.347
Q2	8 (25.0)	6 (16.7)		1 (12.5)	1 (14.3)	
Q3	6 (18.8)	7 (19.4)		1 (10.0)	1 (14.3)	
Q4	6 (18.8)	9 (25.0)		2 (25.0)	1 (14.3)	
Q5	5 (15.6)	11 (30.6)		1 (12.5)	1 (14.3)	

Abbreviations: BMI, body mass index; BVFP, bilateral vocal fold paralysis; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DMII, type II diabetes mellitus; GERD, gastroesophageal reflux disease; HTN, hypertension; MI, myocardial infarction; MVA, motor vehicle accident; OSA, obstructive sleep apnea; PGS, posterior glottic stenosis; SD, standard deviation.

^aValues are presented as number (%) unless otherwise specified.

^bP < .05 considered significant. (significant values are bolded)

Table 3.

Multiple Logistic Regression Model of Tracheostomy-Free Survival at Last Follow-up for Glottic Stenosis Patients.^a

Tracheostomy-Free Survival Multivariate Regression Model		
Variable	Odds Ratio (95% CI)	P Value ^c
Tobacco use	0.23 (0.06, 0.84)	.026
African American ethnicity ^b	1.3 (0.21, 7.4)	.800

Abbreviation: CI, confidence interval.

^a Adjusted for age, sex, body mass index, chronic obstructive pulmonary disease, and hypertension.

^b White ethnicity used as reference.

^c $P < .05$ is considered significant (significant values are bolded).