The Utility of Pulmonary Function Testing in Patients Presenting With Dysphonia

[1]Kirsten Meenan, Lisa Catanoso, [2]Julien Aoyama, [3]Stephen R. Stephan, [4]Ridley Chauvin, and Robert T. Sataloff, *Philadelphia, Pennsylvania*

Summary: Objective: We aimed to evaluate the utility of pulmonary function testing (PFT), particularly forced expiratory flow (FEF) 25−75%, in patients presenting with dysphonia.

Study Design: A retrospective chart review was carried out.

Methods: Records of 199 patients who presented with dysphonia were reviewed to determine whether in-office PFTs, which we perform routinely, lead to new pulmonary diagnoses or the need for additional pulmonary medications, after assessment by a pulmonologist. Of particular interest was evaluating if FEF25−75% of predicted values less than 80% can be used as a marker for occult pulmonary disease in patients presenting with dysphonia.

Results: Of the 199 patient charts reviewed, 129 were female and 70 were male. The age of patients ranged from 18 to 88 years, with a mean of 46.8 years. The body mass index ranged from 17.5 to 53.4 kg/m^2. One hundred five (52.8%) patients had FEF25−75% values less than 80% of predicted (poor midflow values). Of these patients, 76 (72.4%) were referred to a pulmonologist, 22 of 76 (28.9%) completed the referral, and 17 of 22 (77.3%) received a new pulmonary diagnosis or change in medications. Of the 155 patients without a history of pulmonary disease, 76 had poor midflow values, 57 (75%) of these patients were referred, and 12 of 57 (21%) completed the referral. Eight (67%) of these 12 patients were diagnosed with a previously unrecognized pulmonary disorder. Of the 44 patients with a prior history of pulmonary disease, 29 (65.9%) had poor midflow values. Nineteen (65.5%) of these patients were referred, and 9 (47%) received a new pulmonary diagnosis or a change in their medications. There were 51 classically trained singers and 148 nonclassically trained singers or nonsingers. There was no significant difference in average midflow values between the two groups (80.96 ± 24.7 and 80.73 ± 28.4, respectively) or in the percentage of classically trained singers with poor midflow values compared with nonsingers (53.5% vs. 49%, respectively).

Conclusion: This study suggests that patients with dysphonia may have unrecognized underlying pulmonary disease, and PFT should be considered as part of the routine initial voice evaluation for patients presenting with dysphonia.

Key Words: Pulmonary function tests−Dysphonia−Hoarseness−Voice−Voice evaluation.

INTRODUCTION

Dysphonia is a common patient complaint associated with many etiologies, including conditions that afflict the power source of the voice, but in current otolaryngology practice, it is not standard practice to assess pulmonary function. However, many papers have addressed the prevalence of dysphonia in asthmatics and patients with other obstructive lung diseases.[1−9] In obstructive lung disease, the voice may be affected by symptoms of the disease, such as coughing, increased mucous production, and shortness of breath, leading to a voice quality that is breathy or raspy.[7] Adequate pulmonary support is essential to produce an effective and sustained voice. In 1996, Carroll et al[1] addressed the need for adequate pulmonary support and coined the term "vocal athlete." They studied pulmonary function testings (PFTs) of 40 classically trained singers and found that male classically trained singers averaged 100% of predicted values for forced vital capacity (FVC) and 94% predicted values for forced expiratory flow 25−75% (FEF25−75%). Female classically trained singers averaged 103% of predicted values for FVC and 91% predicted values for FEF25−75%. The PFT results of these classically trained singers were better than normalized standards, and so the term vocal athlete was used to describe this phenomenon. In their study, Carroll et al[1] used decreased FEF25−75% values compared with predicted values (midflows) to refer four patients to a pulmonologist, all of whom were diagnosed with an obstructive lung disease. Finally, they described how performers (which may be extrapolated to include all professional voice users) might experience exercise-induced asthma due to hyperventilation associated with performance that leads to airway drying. They felt that changes only in FEF25−75%, and not other measures of PFTs, could lead singers to perceive a voice disturbance.

In 1997, another study was performed in which 20 adults with unexplained dysphonia were found to have abnormal PFTs or abnormal responses to methacholine challenge. After treatment for asthma, all of them experienced improvement in symptoms.[2] Another example of dysphonia resolving with asthma treatment was seen in a 2001 case report. In this report, a 21-year-old woman with dysphonia and a positive

methacholine challenge test showed a substantial improvement in her singing voice with albuterol and then immunotherapy. Two years after her diagnosis, she was taking albuterol only every 2 months, and her repeat methacholine challenge showed a 13% decline in forced expiratory volume in 1 second (FEV1) at 32 minutes, as opposed to a 20% decline after 5 minutes 2 years previously.[10]

More recently, Asnaashari et al[11] found a statistically significant difference in breathiness, harshness, hoarseness, normalized noise energy of glottal noise, and S/Z ratio between 34 patients with untreated mild-to-severe persistent asthma and age- and sex-matched controls. In 2016, Hamdan et al[5] published a cross-sectional study with 31 asthmatics and 19 age- and sex-matched controls. Allergy and smoking history were equivalent in both groups. The asthmatics reported a significantly higher prevalence of dysphonia, dyspnea, and cough. The perceptual grade of dysphonia, asthenia, and straining were also higher in the asthma patients. In the same year, in a population-based study published by Park and Choi,[8] 64 out of 568 (11.3%) asthmatics without laryngeal lesions reported subjective dysphonia, whereas 955 out of 17,408 (5%) controls without laryngeal lesions reported dysphonia. In that study, age, female sex, and higher stress also were associated with dysphonia. Both asthmatics who took asthma medications within 1 year and those who had not taken asthma medications within 1 year had higher adjusted odds ratios (AORs) than controls (1.97 and 1.62, respectively). Many studies suggest that, in addition to the lack of air support, the vocal symptoms seen in those with pulmonary disease might also be due to steroid inhaler use,[7,8,11,12] which can cause thyroarytenoid myopathy, laryngeal candidiasis, and steroid-induced laryngitis.[11]

To the authors' knowledge, there have been no studies addressing the utility of routine pulmonary function tests in patients presenting with dysphonia, regardless of laryngeal lesions and other potential etiologies. Because of personal experience in practice and the studies published in the 1990s,[1,2] the senior author (RTS) performs PFTs routinely as part of the initial voice assessment and usually refers patients for formal pulmonary assessment if the FEF25−75% predicted value is less than 80%. Midflow is more sensitive in detecting occult obstructive pulmonary disease, which may be leading or contributing to patients' symptoms, especially when the symptoms occur during or after prolonged voice use. Although many patients presenting with dysphonia have some laryngeal pathology, such as laryngopharyngeal reflux, mass lesions, muscle tension dysphonia, paresis, or paralysis, the authors believe it is important to assess for decreased pulmonary function, which may be a contributing factor or may even be the primary cause of the laryngeal lesion. This study's primary objective was to define the prevalence of pulmonary disease in patients presenting with dysphonia using midflows of less than 80% of predicted as the criterion for pulmonary referral. The authors believe that these patients' pulmonary disorders may be missed using a typical algorithm for diagnosing pulmonary disease like the American Academy of Family Physician's (AAFP) algorithm for interpreting pulmonary

function test results. Based on the AAFP's algorithm for interpreting PFT results, an obstructive defect is defined as FEV1/FVC < 70% and FVC >lower level of normal (LLN). A mixed pattern of pulmonary disease is defined as FEV1/FVC < 70% and FVC < LLN. A restrictive pattern of pulmonary disease is defined as FEV1/FVC > 70% and FVC < LLN.[13]

A secondary objective was to determine whether the FEF25−75% values differ between classically trained singers and the rest of the study population. The expected pulmonary function test results among classically trained singers are controversial in the literature. Some suggest that singers and wind-instrument players have superior pulmonary function compared with normal standard values,[1,14−16] while others suggest that they do not.[17−21]

MATERIALS AND METHODS

A retrospective chart review of the initial voice assessments of 199 subjects presenting with dysphonia between 2009 and 2015 was performed using office charts from the senior author's (RTS) practice. Any adult patient presenting with the complaint of dysphonia who completed the recommended pulmonary function test as part of the initial voice assessment during the stated time period was included in the study. Information about age, gender, body mass index (BMI), preexisting history of pulmonary disease, and whether the patient was a classically trained singer also were collected as part of the initial voice assessment and used in analysis. The PFT at the senior author's (RTS) practice between 2009 and 2015 was completed by the same two skilled technicians using EasyOne Plus Diagnostic Spirometer (NDD Medi/Interchnik AG, Zurich, Switzerland). The patients completed the spirometry testing three times, and their best scores for FVC, FVC% predicted, FEV1, FEV1% predicted, FEV1/FVC, FEF25−75, and FEF25−75% predicted were recorded. These values were collected for this retrospective analysis.

We recorded whether patients received a pulmonary referral based on their spirometry results, if they completed the referral, and if they received a new diagnosis, no pulmonary diagnosis, or a change in medications prescribed previously for a known pulmonary condition after pulmonary referral. The senior author typically refers patients for formal pulmonary assessment if FEF25−75% is less than 80% of the predicted value.

Patients were classified as obstructive, restrictive, or mixed lung disease based on the AAFP's algorithm for interpreting PFT results applied to PFT results obtained in the senior author's clinic before formal pulmonary referral.

After data collection, the information was analyzed using the Statistical Package for the Social Sciences (SPSS v24 IBM 2016). For most comparisons, FEF25−75% predicted was dichotomized as less than 80 versus 80 or more, as 80 was the cutoff used to determine potential need for referral. To compare these groups on numeric variables such as age and BMI, unpaired t tests or Mann-Whitney U tests were done, depending on the distribution. Nominal variables like gender, prior history of pulmonary disease, and being a

TABLE 1.
Male and Female Epidemiology in the Study Population

	Males	Females
N	70	129
Mean ± SD age (y)	50 ± 15.6	45.1 ± 17.1
Mean ± SD BMI (kg/m^2)	25.9 ± 4.6	25.9 ± 4.6

classically trained singer were compared between the FEF25−75% groups using chi-square. The percentages and confidence intervals (CIs) of those who completed pulmonary referral and received a new pulmonary diagnosis, no pulmonary diagnosis, or a change in pulmonary medication were calculated using a modified Wald method.

RESULTS

One hundred ninety-nine adult patients presenting with dysphonia were included in this study. The mean ± standard deviation (SD) age was 46.8 ± 16.7 years with a range of 18−88. The mean ± SD BMI was 25.9 ± 6 kg/m^2 with range of 17.5−53.4. Thirty-five percent (70) of the subjects were men, with a mean ± SD age of 50.0 ± 15.6 years and a mean ± SD BMI of 25.9 ± 4.6 kg/m^2. Sixty-five percent were women (129), with a mean ± SD age of 45.1 ± 17.1 years and mean ± SD BMI of 25.9 ± 4.6 kg/m^2. There were 40 (20%) cigarette smokers in the population. Smokers were identified as either current smokers or those who had quit less than 15 years before presentation. Forty-four (22.1%) of the patients had a known preexisting pulmonary disease, including asthma, chronic obstructive pulmonary disease (COPD), and pulmonary sarcoid, and 155 (77.9%) had no preexisting lung disease (Table 1).

TABLE 2.
Prevalence of Poor and Normal Midflow Values in Smokers Versus Nonsmokers

	FEF25−75% <80%	FEF25−75% >80%	
Smokers (n)	29 (27.6%)	11 (11.7%)	
Nonsmokers (n)	76 (72.4%)	83 (88.3%)	P = 0.005 (chi-square, two-tailed

Because the senior author uses FEF25−75% value less than 80% of predicted as a criterion to refer patients for a pulmonology evaluation, the authors were interested to see if age, BMI, gender, smoking history, or known preexisting lung disease relate to having poor midflows (defined as FEF25−75% value less than 80% of predicted). None of these potential predictors were associated with poor midflow values, except smoking. The average ages of subjects with midflows less than 80% and with midflows greater than or equal to 80% were 48.32 ± 16.7 and 45.16 ± 16.7, respectively (p = 0.18) and FEF25−75% values showed no significant correlation (r = −0.094, P = 0.19). BMI did not differ between midflow groups (26.0 ± 6.4 kg/m^2 in those with poor midflows vs. 25.7 ± 5.6 kg/m^2 with normal midflows, P = 0.9, by Mann-Whitney U test). The mean FEF25−75% predicted value for men was 77.89 ± 29.4 and for women was 82.36 ± 26.3 (P = 0.27). Chi-square also showed no significant difference in normal and abnormal midflow values between genders (P = 0.47). There was a significant difference between the rate of smokers and nonsmokers with poor midflows (27.6% and 72.4%, P = 0.005; Table 2).

Overall, 105 (52.8%) subjects had midflows less than 80% of predicted in the otolaryngology office. Seventy-six (72.4%) of these patients were referred to a pulmonologist, and 22 (28.9%) of these patients completed the referral. Seventeen (77.3%) of the patients who completed the pulmonary referral received a new pulmonary diagnosis or a change in pulmonary medications (95% CI = 56%−90%). Thus, 17 of the 76 referred patients (22.4%) received a new pulmonary diagnosis or change in medications (95% CI = 14%−33%).

As expected, preexisting lung disease was correlated significantly with FEF25−75% values less than 80% of predicted (uncorrected chi-square P = 0.048). Of those without lung disease, 76 of 155 (49.0%) subjects had poor midflows versus 29 of 44 (65.9%) of those with preexisting lung disease. The average midflow value for those without lung disease was 83.52 ± 25.88 and was 71.18 ± 30.81 for those with preexisting disease (P = 0.004; Table 3). Of the patients with poor midflow values, there was no significant difference in the prevalence of smokers and nonsmokers between the two groups (chi-square, two-tailed P = 0.63; Table 4).

Of the 76 patients with poor midflow values who did not have a history of pulmonary disease, 57 (75%) were referred, but only 12 (21%) completed the referral. Three of these patients had evidence of obstructive lung disease in the clinic based on the AAFP algorithm, while the remaining nine had normal PFTs other than FEF25−75% predicted values.

TABLE 3.
Midflow Values in Patients Without a History of Pulmonary Disease Versus Patients With a History of Pulmonary Disease

	Mean ± SD FEF25−75% Predicted Value	FEF25−75% Predicted Value <80%, N	FEF25−75% Predicted Value ≥80%, N	Total
No history	83.52 ± 25.88	76 (49%)	79 (51%)	155
History	71.18 ± 30.81	29 (65.9%)	15 (34.1%)	44
	P = 0.004	P = 0.048	P = 0.048	

TABLE 4.
Prevalence of Smokers and Nonsmokers in Patients With Pulmonary Disease Versus Patients Without Pulmonary Disease Who Have Poor Midflow Values

	Smokers, N	Nonsmokers, N
Pts with lung Dz Hx and poor midflows, n = 76	20 (20/76 = 26.3%)	56 (56/76 = 73.7%)
Pts without lung Dz Hx and poor midflows, n = 44	9 (9/29 = 31%)	20 (20/29 = 69%)
		P = 0.63 (chi-square, two-tailed)

TABLE 6.
Prevalence of New Diagnoses/Change in Medications in Smokers Versus Nonsmokers in Patients With Poor Midflows

Pulmonary Disease	New Diagnosis or Change in Pulmonary Medications		
	Smokers, n	Nonsmokers, n	
No history	3 (3/20 = 15%)	5 (5/56 = 8.9%)	P = 0.73
History	1 (1/9 = 11.1%)	8 (8/20 = 40%)	P = 0.26

Eight of these 12 (67%) patients were diagnosed with a pulmonary disorder (95% CI = 39%−86%) by the pulmonologist, despite the fact that only 2 of them had obstructive lung disease based on the AAFP algorithm in the office (Table 5). Thus, 8 of 155 (5%) patients without prior pulmonary disease received a new diagnosis after referral (95% CI = 3%−10%). Of these 8 patients, 2 were smokers and 5 were nonsmokers. There was no significant difference in the rate of smokers who were diagnosed with a pulmonary disease compared with nonsmokers (15% and 8.9%, respectively, P = 0.73; Table 6). There were two additional patients without a prior history of pulmonary disease with midflows in the normal range who were referred based on clinical judgment.

There were 44 patients in the study population with a pre-existing pulmonary disease, 29 (65.9%) of whom had FEF25−75% predicted values less than 80%. Based on the AAFP algorithm for interpreting PFTs, 11 of these 29 (38%) patients would have qualified as having either obstructive, restrictive, or mixed lung disease based on otolaryngology office assessment (95% CI = 23%−56%). Seven of these 11 (64%) patients were referred, but only 3 completed the referral. All three of these patients had evidence of obstructive lung disease in the clinic based on the AAFP algorithm. Two of the seven (29%) received a new diagnosis or change in pulmonary medications (95% CI = 8%−65%), and one was not diagnosed with a pulmonary disorder upon

referral. Thus, two (66.7%) of the three who completed the referral received a new diagnosis or change in medications.

Of the 29 patients with a prior history of pulmonary disease and poor midflow values, 18 (62%) of them did not have obstructive, restrictive, or mixed lung disease in the otolaryngology office based on the AAFP algorithm for interpreting PFT results (95% CI = 44%−77%). Twelve (67%) patients in this subpopulation were referred. Seven of the 12 (58%) completed the referral, and all 7 of these patients had a change in their pulmonary medications (regimen or class of drug) (95% CI = 65%−100%).

Thus, in the subgroup of patients with a history of pulmonary disease and poor midflow values, 19 (65.5%) patients were referred, but only 10 (52.6%) completed the referral. Of these 10 patients, 9 (90%) of them received a new pulmonary diagnosis or a change in medications (Table 5); that is, 9 of the 19 (47%) referred patients with a prior history of pulmonary disease and poor midflow values received a new pulmonary diagnosis or a change in pulmonary medications from the pulmonologist (95% CI = 27%−68%). In this subgroup of patients, there was also no significant difference in the rate of smokers who were diagnosed with a new pulmonary disease or had a change in their pulmonary medications compared with nonsmokers (11.1% and 40%, respectively, P = 0.26; Table 6).

In the study population, there were 51 classically trained singers and 148 patients who did not identify themselves as classically trained singers. The mean FEF25−75% values for classically trained singers and nonclassically trained singers/nonsingers were 80.96 ± 24.7 and 80.73 ± 28.4, respectively (P = 0.96). There were 79 (53.5%) nonclassically

TABLE 5.
Rate of New Pulmonary Diagnoses or Change in Medications Upon Referral in Patients With a History of Pulmonary Disease Versus Those Without a History of Pulmonary Disease

	N, Total	N, Poor Midflow Value	N, Referred	N, Referral Completed	N, New Diagnoses or Change in Medication
No history	155	76 (49%)	57 (75%)	12 (21%)	8 (67%) (95% CI = 39%−86%)
History	44	29 (65.9%)	19 (65.8%)	10 (52.6%)	9 (90%) (95% CI = 27%−68%).

TABLE 7.
Midflow Value Comparison of Classically Trained Singers and Nonclassically Trained Singers/Nonsingers

	Classically Trained	Not classically Trained	
N	51	148	
Mean FEF25−75% predicted value	80.96 ± 24.7	80.73 ± 28.4	*P* = 0.96
N, poor midflow values	25 (49%)	79 (53.5%)	*P* = 0.59

trained singers/nonsingers with poor midflow values and 25 (49.0%) classically trained singers with poor midflow values. The small difference between these two groups is not statistically significant (*P* = 0.59; Table 7).

DISCUSSION

Dysphonia is a common difficulty for many professional voice users. In one study, dysphonia was reported in 57% of active teachers.[22] The etiologies of dysphonia are commonly classified into three groups, organic, functional, and neurologic, which drive the clinician's assessment and plans in making a diagnosis.[23] However, all three of these categories may affect the lungs and the vocal tract. Damaged cilia and mucosa from smoking, allergic rhinitis, and nasal polyps are just a few of the changes that occur in the vocal tract that have been shown to correlate with an increased incidence of dysphonia.[4,24] While there have been several reports of increased prevalence of dysphonia in asthmatics,[4,5,7,8,11,25] there is a lack of literature correlating poor pulmonary function tests and dysphonia.

Inadequate pulmonary function undermines voice support and may be responsible for or contribute to the laryngeal pathology for which patients seek otolaryngologic help. The aim of this study was to determine whether there is utility in using PFT routinely to screen patients presenting with dysphonia to identify a possible underlying etiology of their voice complaints. To the best of the authors' knowledge, the senior author (RTS) is the only clinician who recommends pulmonary function screening routinely on all new patients who present with voice complaints, regardless of physical examination findings. The reason for this is to identify pulmonary problems that have gone misdiagnosed but that might affect voice. Usually, pulmonary disorders are diagnosed using FEV1/FVC, FVC, bronchodilator response, carbon monoxide diffusing capacity, and sometimes bronchoprovocation.[13] The senior author assesses these values but uses FEF25−75% values less than 80% of predicted for referring patients to a pulmonologist, because of the belief that midflow values are useful to identify occult pulmonary disease in voice patients, especially when the FEV1 and FVC are within normal limits. Additionally,

some dysphonic patients may only experience symptoms with extended voice use, such as singing or teaching.

In this study, slightly greater than half of the patients presenting with dysphonia had FEF25−75% values less than 80% of predicted, and most of these patients (72.4%) were referred for formal pulmonology assessment. This high percentage may be clinically justifiable, given that of the 22 patients (28.9%) who completed the referral, 77.3% received a new diagnosis or had a change in their current pulmonary medication. The number referred is not 100% because some patients, while advised, refused further pulmonary assessment at the time of visit so they were not included. Others were not referred because they already had a pulmonologist or had conditions being treated by a family doctor. It is unlikely that the high rate of noncompliance had to do with a difference in subjective pulmonary function as the majority of those who were compliant, and all of those who had a new diagnosis, were unaware of any lung-related symptoms. Because the group was subjectively asymptomatic except for voice complaints that were present in both groups, it is possible but unlikely that compliant patients saw a pulmonologist because of a pulmonary function difference compared with the noncompliant group. Therefore, it seems likely that if more patients completed the referral, more pulmonary disease likely would have been identified, although possibly not at a rate of 77%. Unfortunately, one of the limitations of this retrospective study is that there was no long-term follow-up of the patients who were diagnosed with a new pulmonary disease or had a change in their medication to ascertain whether their dysphonia improved with pulmonary treatment. This will be investigated in a prospective study.

Several studies have provided evidence that people with pulmonary disease, such as asthma and COPD, have a significantly higher rate of dysphonia.[4,5,7,8,11,25] In addition to the authors' explanation that poor "breath support" may contribute to dysphonia, there are several other potential explanations for these higher rates of dysphonia in this population; these include the use of inhaled corticosteroids (ICS), symptoms associated with these diseases such as coughing, prior or current smoking history, and common comorbidities of pulmonary disease, such as allergic rhinitis, gastroesophageal reflux disease, and xerostomia. In a cross-sectional study of 31 patients with asthma (41% controlled, 59% uncontrolled, and 58% on steroid inhalers) and 19 age- and sex-matched controls, 32.3% of asthmatics and only 5.3% of the control group reported dysphonia (*P* = 0.025). Additionally, 80% of the asthmatic patients compared with only 6% of the controls reported cough (*P* < 0.05). The grade of dysphonia, asthenia, and strain were also significantly greater in the asthmatic population.[5] A limitation of that study was that it did not address whether uncontrolled asthma or the use of steroid inhalers contributed to the higher rates of dysphonia and other symptoms in the asthmatic population, which one might expect. Govindaiah et al[4] found a higher prevalence of dysphonia in asthma patients (44%), and inhaled corticosteroids and excessive voice use were both positively associated with his finding

(odds ratio [OR] 3.13, $P < 0.025$, and OR 2.15, $P < 0.1$ respectively). In a study by Park and Choi,[8] only 64 of 568 (11.3%) asthma patients without laryngeal pathology reported dysphonia. However, this was still greater than the 955 of 17,408 (5%) controls who reported dysphonia. Lastly, Mohamed and El Maghraby[7] identified 25 of 50 (50%) COPD patients with dysphonia, based on high jitter %, shimmer%, grade of dysphonia, and low fundamental frequencies. They also found significant inverse correlations between FVC, FEV1, and maximum mid-expiratory flow percent of predicted values with jitter%, shimmer%, and grade of dysphonia. There was a significant positive correlation between these PFTs and fundamental frequency.

Smoking leads to pulmonary disease, which may lead to dysphonia, and smoking itself may be an etiology of dysphonia in people with normal pulmonary function. It was no surprise that in our study, a history of smoking was associated significantly with poor PFTs. However, the prevalence of smoking was the same in patients with pulmonary disease and patients without pulmonary disease who had poor midflow values. Furthermore, smoking history was not significantly related to obtaining a new diagnosis or change in medications after referral. Therefore, clinicians should not use smoking history as a means of predicting which dysphonic patients will have underlying pulmonary disease or require a change in their medications.

In our study population, the average midflow values for those with pulmonary disease were significantly lower than for those without a history of pulmonary disease, and more people with preexisting pulmonary disease had poor midlows. This may be evidence of poorly managed obstructive disease in these people.

Should patients with known pulmonary disease and poor midflows be referred for further evaluation? Our results suggest that they should be. Eleven of the 29 patients with a history of pulmonary disease and poor midflows had obstructive, restrictive, or mixed lung disease based on the AAFP algorithm. Two of the three of these patients who completed the referral received either an additional pulmonary diagnosis or had additional pulmonary medications added to his regimen by the pulmonologist. These two patients represent a larger population who obviously had inadequate control of their pulmonary disease, which might otherwise not have been recognized in a timely fashion. Eighteen patients out of 29 with a history of pulmonary disease and poor midflow values did not have obstructive, restrictive, or mixed pulmonary disease based on the AAFP algorithm. Seven of the 12 referred patients in this group were given new medications or a change in their current regimen upon pulmonary referral. This was 100% of those who completed the referral. These seven patients raise concerns about a larger population of people whose pulmonary disease might be controlled suboptimally and for whom the AAFP algorithm may not have been sufficient to recognize their diminished pulmonary function.

Furthermore, 8 of 12 (66.7%) patients without a history of pulmonary disease who followed-up with a pulmonologist were diagnosed with a pulmonary disease. Of particular interest is that six of these patients had normal pulmonary function according to the AAFP algorithm. Without the strict standard of referring patients with midflow values less than 80%, these six patients would likely have remained undiagnosed, at least until their current symptoms worsened or new symptoms developed.

The literature on pulmonary function differences in professional singers is minimal and outdated. Some have found that trained singers have superior PFTs,[1,15,26] while others have found no difference compared with normative data.[17,18,21] In 1973, Gould and Okamura[15] compared trained professional singers, students of voice, and a group with no vocal training. While they found no significant difference in the total lung capacity (TLC) among the three groups, the residual volumes (RV) were lower in those with more training. Thus, the professional singers had the lowest RV/TLC ratio, followed by students, and lastly by those without vocal training.[15] Two years later, Gould[26] published a follow-up study with similar findings, which showed that more years of training correlated directly with a lower RV/TLC ratio.[26] Conversely, in 1985, Schorr-Lesnick et al[18] found no significant difference in peak expiratory pressure, peak inspiratory pressure, maximum voluntary ventilation percent of predicted, FEV1 percent of predicted, FVC percent of predicted, FEV1/FVC, or FEF25−75% of predicted when comparing singers, wind instrumentalists, and a control group. In 2000, Lundy et al[21] found that pulmonary function tests in singing students were within normal limits of the general public. In our study, we also found no statistically significant difference in FEF25−75% values between classically trained singers and nonclassically trained singers/nonsingers. However, the mean midflow values and percentage of those with normal midflow values were greater in classically trained singers. A future study should evaluate all parts of a standard pulmonary function test to assess potential differences between these two groups.

The authors are aware of additional limitations to this study. First, the presence of structural laryngeal anomalies was not taken into account. It might be that some of these structural anomalies resulted from poor technique due to inadequate breath support. This hypothesis is one that needs further testing. In the recent study by Park and Choi,[8] there was no significant difference in the amount of organic laryngeal lesions in asthmatics compared with nonasthmatics. However, they only assessed three organic lesions: vocal fold nodules, vocal fold polyps, and laryngitis. Second, we did not control for the presence or absence of allergies in our population. Allergies may cause changes in the larynx and upper airway, which may contribute to voice changes.

Another limitation of this study was that it did not consider the vocal effects of inhaled corticosteroids (ICSs) in patients taking these medications. Williamson et al[9] performed a study in which a questionnaire was used to identify the rate of dysphonia or other voice symptoms in people who used ICSs. They found that 58% of patients taking ICSs reported dysphonia, whereas only 13% of the control group reported voice symptoms. In Park and Choi's[8] study, asthma

patients who took their ICSs in the past year had a higher AOR for dysphonia than asthma patients who had not taken their medications in the past year (AOR = 1.97, 95% CI = 1.28%−3.02%); these patients not on medication had higher AORs than patients without asthma (AOR = 1.62, 95% CI = 1.0%−2.42%). Thus, the presence of asthma was related to dysphonia, and the use of ICSs increased the odds of having dysphonia. However, it is not clear whether the dysphonia is related to steroid inhalers or to worse asthma that led to the prescription of steroid inhalers, or to some other cause. Numerous other studies have evaluated the relationship of ICSs and dysphonia. In a questionnaire study by Ihre et al,[25] both the degree of asthma trouble and ICS dose were significantly positively associated with voice complaints. Govindaiah et al[4] found similar results and reported ICS use and excessive voice use were both positively associated with dysphonia in asthma and allergic rhinitis patients (OR = 3.12, $P < 0.025$, and OR = 2.15, $P < 0.1$, respectively).

In the study by Bhalla et al,[13] the vocal effects of regular use of ICSs (at least once a day) were compared with those of infrequent use of ICSs (about once a week or during summer months). They found that regular users rated their vocal performance via a questionnaire significantly lower than infrequent users ($P < 0.001$). Regular users also had more side effects such as hoarseness, throat discomfort, vocal weakness, aphonia, and cough ($P < 0.001$). They also exhibited significantly more jitter, shimmer, and closed-phase quotient scores. It is important for otolaryngologists to consider not only pulmonary impairment an etiology of dysphonia but also pulmonary treatment and to question whether changing the medications or using the medications less frequently would control pulmonary symptoms adequately.

While most of the studies evaluating the prevalence of dysphonia and effects of ICSs on the voice have been in asthmatic populations, Mohamed and El Maghraby[7] evaluated 50 patients with COPD and found a 50% prevalence of dysphonia in these patients. High doses of ICSs showed significant positive correlation with abnormal jitter, shimmer, and grade of dysphonia, whereas low-to-moderate dose of ICSs showed a significant inverse correlation with jitter, shimmer, and the grade of dysphonia. High doses also showed a significant inverse correlation with fundamental frequency, and low doses showed a significant positive correlation with fundamental frequency.

Despite its limitations, our study found a significant amount of new pulmonary disease and unsuspected inadequately controlled pulmonary disease in patients who complied with pulmonary referral. This suggests pulmonary screening is valuable for dysphonia patients and supports more aggressive efforts in assuring compliance with pulmonary referrals. Future studies should examine a higher compliance rate to see if the results remain congruent with those of this study. A future study also should provide long-term follow-up of the patients who complete pulmonary referral to see whether their voice complaints improve with treatment.

There would also be benefit from future studies that evaluate whether subjective and objective voice measures correlate with poor pulmonary function tests and pulmonary disease. Dogan et al[3] used subjective and objective voice measures to demonstrate that lower airway disease can disturb phonation. In their study, the patients with mild-to-moderate asthma had significantly higher ratings in grade of dysphonia, roughness, and breathiness ($P < 0.001$, $P < 0.007$, and $P < 0.001$, respectively). More asthma patients also had abnormal Voice Handicap Index scores than controls ($P < 0.0001$). Shimmer was significantly greater in asthma patients, and jitter and noise-to-harmonics ratio were greater in female asthma patients than in controls. The mean phonation times were significantly less in asthma patients than in controls. The S/Z ratio, vital capacity, and phonation quotients were similar in both groups. Asnaashari et al[11] performed a similar study in patients with mild-to-severe persistent asthma. They found significant differences between asthma patients and controls in breathiness, harshness, hoarseness, normalized noise energy, and S/Z ratio. There was no difference in fundamental frequency, jitter, or shimmer between the two groups. Although some of these results differ from those in the study by Dogan et al,[3] both studies show that pulmonary disease affects phonation. Further, larger scale studies would be beneficial to resolve conflicting data. These future studies would be even more beneficial if they could explore a relationship between abnormal subjective and objective voice measures and abnormal pulmonary function tests.

CONCLUSION

Routine PFT in patients presenting with dysphonia should be considered in otolaryngology practice to identify underlying pulmonary disease that might be causing or contributing to voice complaints. The authors believe pulmonary function screening is important in dysphonic patients because most of these pulmonary disorders are treatable, and treatment is likely to help in optimizing laryngological outcome. Further study appears warranted.

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