Auris Nasus Larynx xxx (2018) xxx–xxx

Contents lists available at ScienceDirect

Auris Nasus Larynx

journal homepage: www.elsevier.com/locate/anl

# Is insufficient pulmonary air support the cause of dysphonia in chronic obstructive pulmonary disease?

Megahed M. Hassan [a,*], Mona T. Hussein [b], Ahmed Mamdouh Emam [a], Usama M. Rashad [c], Ibrahim Rezk [c], Al Hussein Awad [c]

[a] Phoniatrics Unit, Otolaryngology Department, Faculty of Medicine, Sohag University, 82524, Egypt
[b] Chest Department, Faculty of Medicine, Sohag University, 82524, Egypt
[c] Otolaryngology Department, Faculty of Medicine, Sohag University, 82524, Egypt

ARTICLE INFO

*Article history:*
Received 28 July 2017
Accepted 21 December 2017
Available online xxx

*Keywords:*
Dysphonia
Vocal function
Pulmonary function
Pulmonary support
COPD

ABSTRACT

*Objective:* Optimal pulmonary air support is essential pre-requisite for efficient phonation. The objective is to correlate pulmonary and vocal functions in chronic obstructive pulmonary disease (COPD) to find out whether the reduced pulmonary function per se could induce dysphonia.

*Methods:* In this prospective case-control study, sixty subjects with stable COPD underwent evaluation of pulmonary and vocal functions. The pulmonary functions measured include {Forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), FEV1/FVC ratio, peak expiratory flow (PEF), maximum mid-expiratory flow (MMEF)}. The vocal functions were {jitter, shimmer, noise-to-harmonic ratio, pitch perturbation quotient, amplitude perturbation quotient, maximum phonation time (MPT), sound pressure level, phonatory efficiency, resistance and power. A control group (n = 35) underwent the same measurements. These functions were compared between subjects and controls. Also, correlation of the vocal and pulmonary functions was conducted.

*Results:* Thirty five (58.3%) of COPD subjects have dysphonia. The pulmonary functions were lower in all COPD group than in the control group ($P < 0.001$ for all parameters). Also, the FVC, FEV1, PEF and MMEF % of predicted values were significantly lower in subjects with dysphonia (n = 35) than those without dysphonia (n = 25) with $P$ values 0.0018, <0.001, 0.0011 and 0.0026 respectively. In addition, the MPT in all subjects showed positive correlations to the 5 pulmonary functions ($P = 0.004$ for FEV1/FVC ratio and $P < 0.001$ for the rest). Also, the phonatory efficiency showed significant positive correlations with the pulmonary functions FVC, FEV1, PEF and MMEF ($P = 0.001$, 0.001, 0.002 and 0.001 respectively). Unlike efficiency, the phonatory resistance revealed significant negative correlations with these pulmonary functions in the same order ($P = 0.001$, 0.003, 0.002, 0.001 respectively).

*Conclusion:* Dysphonia is a common comorbidity with COPD which attributed to multifactorial etiologies. The lower the pulmonary function in COPD patients is the more likely to have dysphonia. Decreased pulmonary function was associated with reduced MPT and phonatory efficiency but with increased phonatory resistance. The reduced pulmonary functions in COPD can be the underlying cause of the altered vocal function and dysphonia. Great part of this dysphonia is functional, and hence, can be corrected by voice therapy in compensated subjects. Further researches are needed to evaluate the efficacy of voice therapy in these patients.



* Corresponding author.
*E-mail address:* megahed_hassan@med.sohag.edu.eg (M.M. Hassan).

https://doi.org/10.1016/j.anl.2017.12.002
0385-8146/

## 1. Introduction

Chronic obstructive pulmonary disease (COPD) is a common respiratory condition characterized by airflow limitation. This limitation makes it difficult to empty air out of the lungs which leads to shortness of breath. Dysphonia was reported extensively in COPD with controversy around its mechanism [1–6]. Many authors attributed the dysphonia in COPD to the associated mechanical irritations, inflammations, and medications [1,2]. The long-term use of inhaled corticosteroids [2], and anti-cholinergics [3] were accused because of their drying effect on the vocal tract. In addition, gastro-esophageal reflux diseases (GERD) cause acid-induced laryngeal mucosal changes. These mucosal changes may be presented by dysphonia even though the reflux is clinically occult [4]. Smokers have high prevalence of GERD, micro-aspiration and dysphonia [5]. So, dysphonia is probably multifactorial depending on medication, inflammation and mechanical irritation [2]. Moreover, hypoxemia and hypercapnia in COPD may induce vocal abnormalities. The soft voice quality might be explained by the narcotic effect of the hypercapnia [6]. The objective of this work is to correlate the vocal and pulmonary functions in COPD in order to find out whether the reduced pulmonary function per se could be a significant cause for dysphonia.

Physiologically, respiratory cycles during ordinary quiet respiration and during running speech are quite different [7]. The speaker rapidly inhales a markedly larger volume of air during running speech (1500–3000 cc), then slowly exhales the air in the uttered sentence. In quiet tidal respiration, the inhalation is slower and the air volume is lesser (500 cc). Although, the time of the respiratory cycle is equal in both, the time spent during inspiration is shorter in running speech (10% of the respiratory cycle) than in quiet respiration (40% of the respiratory cycle) [7]. Optimal pulmonary support is essential pre-requisite for normal voice production [8]. In this article, a correlation between vocal and pulmonary functions was made to show whether there is a relation between dysphonia and the degree of pulmonary function impairment per se.

## 2. Materials and methods

### 2.1. Subjects and control

This study was conducted in Sohag University Hospital during the period from February 2015 to March 2016. Ethics committee approved the study. Written informed consent was obtained from every patient. The study included 60 subjects; 54 males (90%) and 6 females (10%) with stable COPD diagnosed from outpatient clinic of the Chest Department according to Global Initiative for Chronic Obstructive Lung Disease guidelines (GOLD) [9]. The patients' age ranged from 50 to 85 years with average 67 and SD 9.5. Patients were considered to be in stable stage when they did not require changes in their regular medications for at least six weeks. Exclusion criteria were acute exacerbation of COPD, other lung diseases, regular inhalation therapy (inhaled corticosteroids, anti-cholinergics and/or β2 agonists) and other co-morbidities (gastro esophageal reflux disease and sinusitis). Control group included 35 apparently healthy non-smoker subjects; 31 (88.6%) males, and 4 (11.4%) females.

### 2.2. Methods

All subjects underwent complete history taking (age, sex, smoking index, drug history . . . etc.) and clinical examination. Chest X-ray was performed to confirm the diagnosis and exclude any associated complications. Spirometric test was made before and 15–30 min after the administration of 400 mcg of inhaled salbutamol according to the recommendations of American Thoracic Society (ATS) and European Respiratory Society (ERS) [10]. An electronic spirometer (Master Screen PFT, Jaeger) was used in the test with all subjects performed at least three acceptable maneuvers. Forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), FEV1/FVC ratio, peak expiratory flow (PEF), maximum mid expiratory flow (MMEF) and other spirometric measurements were obtained. The spirometric criteria for COPD is a post bronchodilator FEV1/FVC ratio $<70\%$. Arterial blood gases were measured using Nova biomedical (Phox S/N: UO/A 98010)-USA with the following parameters: arterial PH, arterial oxygen tension ($PaO_2$) in mmHg, arterial carbon dioxide tension (PaCO2) in mmHg, Oxygen saturation ($SaO_2$) and serum bicarbonate level ($HCO_3$) in mEq/L. Also, smoking index was calculated for all subjects. Body mass index (BMI) and duration of the disease in months were included in the study. The disease duration was estimated from the beginning of smoking cough. All subjects underwent ENT examination to exclude sinusitis, postnasal drips or any upper respiratory tract infections. All subjects underwent voice evaluation in the Phoniatrics Unit where clinical examination was done to exclude any organic laryngeal lesion. Also, auditory perceptual assessment (APA) of voice, acoustic and aerodynamic investigations were done. The APA was carried out by two experienced phoniatricians using overall Grade–Roughness–Breathiness–Asthenia–Straining (GRBAS) voice scale [8,11]. The mean score was considered. The acoustic analysis was performed by Multi-Dimensional Voice analysis Program (MDVP) model 5105 version 3.3.0 using Kay PENTAX-CSL Model 4500. Using Microphone Shure model SM 48 connected to the CPU, each subject was instructed to phonate the vowel/a/ with comfortable pitch and intensity at a distance of 20 cm from the microphone. The acoustic parameters measured were jitter, shimmer, noise-to-harmonic ratio, pitch perturbation quotient (PPQ), amplitude perturbation quotient (APQ). The noise-to-harmonic ratio (NHR) was converted into harmonic-to-noise ratio (HNR) using a specific formula ($HNR = 10 \times \log 1/NHR$) [12]. The aerodynamic measurements were carried out by Aerophone II. Two types of sample were taken for each subject. A connected speech sample (counting from 1 to 10) was taken while the aerophone mask is tightly sealed around the subject's mouth. Phonatory sound pressure level (SPL) in dBs was measured from this sample. Also,/ipi/sample was taken by instructing subjects to say/ipi/repeatedly while placing a fenestrated tube connected to the aerophone mask into the mouth. The aerodynamic parameters measured from

Please cite this article in press as: Hassan MM, et al. Is insufficient pulmonary air support the cause of dysphonia in chronic obstructive pulmonary disease? Auris Nasus Larynx (2018), https://doi.org/10.1016/j.anl.2017.12.002