OBJECTIVE AND SUBJECTIVE EVALUATION OF VOICE QUALITY IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

BY

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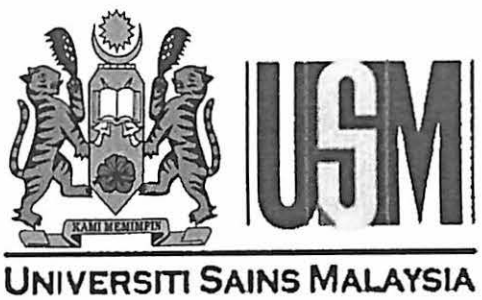

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LIST OF ABBREVIATIONS

COPD	-	Chronic obstructive pulmonary disease
GOLD	-	Global Initiative for Chronic Obstructive Lung Disease
PEFR	-	Peak expiratory flow rate
FEV1	-	Forced expiratory volume in one second
FVC	-	Forced vital capacity
F_0	-	Fundamental frequency
GERD	-	Gastroesophageal reflux disease
VHI	-	Voice Handicap Index
MPT	-	Maximum phonation time
HNR	-	Harmonic-noise-ratio
dB	-	Decibel
mls	-	Mililiter
Hz	-	Hertz
HUSM	-	Hospital Universiti Sains Malaysia
ORL-HNS	-	Otorhinolaryngology and Head and Neck Surgery
PPSP	-	Pusat Pengajian Sains Perubatan

ABSTRACT

BAHASA MALAYSIA VERSION

TAJUK: Objektif dan subjektif evaluasi kepada kualiti suara bagi pesakit-pesakit 'Chronic Obstructive Pulmonary Disease' (COPD).

OBJEKTIF: Menolak hipotesis null bahawa tiada perbezaan antara kualiti suara pesakit-pesakit COPD berbanding kumpulan kawalan.

KAEDAH: Kajian ini melibatkan 40 orang pesakit. 20 orang adalah daripada kumpulan COPD manakala 20 orang adalah daripada kumpulan kawalan, dengan purata umur 68.95 dan 58.40 setiap satunya. Kedua-dua kumpulan melalui proses kajian yang sama iaitu temuramah 'Vocal Symptom Questionaire', ujian ' Peak Expiratory Forced Rate' (PEFR), ujian kualiti suara, dan pemeriksaan telinga, hidung dan tekak. Ujian suara dianalisa menggunakan ' Dr Speech Vocal Assessment Version 4 (Tiger DRS,US). Keputusan setiap ujian dianalisa menggunakan ujian Mann Whitney dan analisis diskriptif.

KEPUTUSAN: Keputusan daripada 'Vocal Symptom Questionaire' menunjukkan purata symptom adalah signifikan kepada pesakit COPD (18.35+-9.8) berbanding kumpulan kawalan(4.65+-8.5; P<0.001). Purata PEFR adalah lebih besar untuk kumpulan kawalan (4.38.50+-139.75) berbanding kumpulan COPD (2.87+_123.11; P<0.001). Penilaian suara dianalisa menggunakan 'jitter', 'shimmer', nisbah

'Harmonic to Noise' (HNR), dan masa phonasi maksimum. Semua penilaian suara adalah lebih daripada julat normal dan keputusan Mann Whitney adalah tidak signifikan. Pemeriksaan ENT dan larinks juga tidak menunjukkan sebarang keputusan yang signifikan.

KESIMPULAN: Hipotesis null berjaya ditolak bagi analisis subjektif, tetapi bagi 'dysphonia' atau kualiti suara yang berubah, tidak dapat dibuktikan dengan penilaian objektif suara.

ABSTRACT

ENGLISH VERSION

TITLE: Objective and subjective evaluation of voice quality in patient with chronic obstructive pulmonary disease (COPD).

OBJECTIVE: To test the null hypothesis that there is no difference in voice quality between COPD group with the control group.

METHOD: Subjects were 40 adult participants. Recruited 20 participants in COPD group and control group of 20 participants, with the mean age of 68.95 and 58.40 respectively. Both group underwent Vocal Symptoms Questionaire interview, Peak Expiratory Forced Rate (PEFR) test, voice recording and ear, nose and throat examination. The voice was recorded and analyzed with Dr Speech's Vocal Assessment Version 4 (Tiger DRS,US). The mean COPD and control group were subjected to Mann-Whitney test and descriptive analysis.

RESULT: Vocal Symptom Questionaire showed the mean total vocal symptom were significantly greater in COPD group (18.35 +_9.8) than for the control group (4.65+_8.5; P<0.001). The mean of

PEFR were greater for the control group (438.50+_139.79) than for COPD group (287.80 +_123.11; P<0.001). The vocal assessment were analyzed in term of jitter, shimmer, harmonic to noise ratio (HNR) and maximum phonation time. All the vocal assessment profile mean were more than the normal value and the Mann Whitney test were not significant (P >0.001). Full ENT examination did not reveal any significant finding. The laryngeal and ENT examination also shows no significant pathology to contribute to the voice change in study participant.

CONCLUSION: In the subjective analysis, the null hypothesis is rejected. COPD patients do complain of voice problem but the vocal assessment did not prove the dysphonia claimed by the study group.

CHAPTER 1
INTRODUCTION

1.0 INTRODUCTION

1.1 INTRODUCTION TO CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. COPD is characterized by chronic airflow limitation, a range of pathologic changes in the lung, some significant extrapulmonary effects, and important comorbidities that may contribute to the severity of the disease in individual patients (Klaus F. Rabe, 2007).

COPD is a preventable disease with some significant extrapulmonary effects such as cor pulmonale that may contribute to the severity in an affected patient. The pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases (Klaus F. Rabe, 2007).

COPD often develops in long time smokers in middle age and cigarette smoke is the most commonly encountered risk factor for COPD. Elimination of this risk factor is an important step toward prevention and control of COPD (Klaus F. Rabe, 2007). However, other risk factors for COPD should be taken into account where possible, including occupational dusts and chemicals, and indoor air pollution from biomass cooking and

heating in poorly ventilated dwellings, the latter especially among women in developing countries (Klaus F. Rabe, 2007).

Diagram shows summary of risk factor for COPD (Figure 1.1), adapted from Klause F. Rabe, 2007.

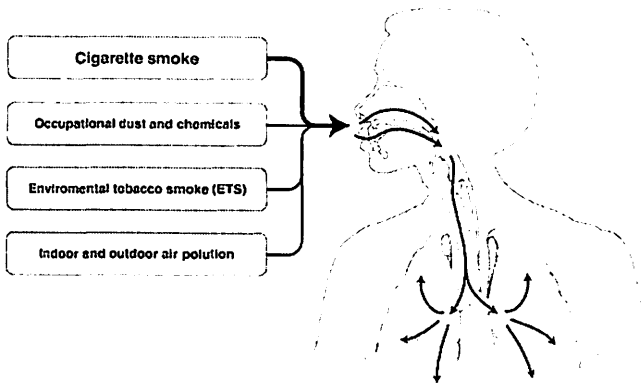

Figure 1.1: Risk factor for COPD

A clinical diagnosis of COPD should be considered in any patient who has dyspnoea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease (Klaus F. Rabe, 2007). In more severe condition, the symptoms are dyspnoea with poor exercise tolerance, and signs or symptoms of right-sided heart failure (Amir Qaseem, 2007). The diagnosis should be confirmed by spirometry (Klaus F. Rabe, 2007).

To consider COPD and perform spirometry, there are few indicators that may present in an individual. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD (Klaus F. Rabe, 2007).

1. Dyspnea that is progressive (worsens over time), usually worse with exercise, persistent (present every day), prescribed by the patient as an ''increased effort to breathe'', ''heaviness'', ''air hunger'', or ''gasping".
2. Chronic cough; may be intermittent and may be unproductive.
3. Chronic sputum production; any pattern of chronic sputum production may indicate COPD.
4. History of exposure to risk factor; tobacco smoke, occupational dusts and chemicals, smoke from home cooking and heating fuels.

The GOLD (Global Initiative for Chronic Obstructive Lung Disease) report emphasizes the importance of the functional assessment of COPD; the functional assessment including the progression of the disease and the non fully reversible of the airflow limitation. The non fully reversible of airflow limitation are measured as a ratio between forced expiratory volume in 1 s (FEV1) and forced vital capacity (FVC) after bronchodilator below 0.7 (Klaus F. Rabe, 2007).

Spirometry is the gold standard because it is the most reproducible, standardized, and objective way of measuring airflow limitation (Klaus F. Rabe, 2007).

The spirometric classification of severity of COPD includes four stages: stage I, mild; stage II, moderate; stage III, severe; stage IV, very severe (Klaus F. Rabe, 2007).

Stage I: mild COPD: Characterized by mild airflow limitation (FEV1/FVC , 0.70, FEV1 > 80% predicted). Symptoms of chronic cough and sputum production may be present, but not always. At this stage, the individual is usually unaware that his or her lung function is abnormal.

Stage II: moderate COPD: Characterized by worsening airflow limitation (FEV1/FVC , 0.70, 50% < FEV1 , 80% predicted), with shortness of breath typically developing on exertion and cough and sputum production sometimes also present. This is the stage at which patients typically seek medical attention because of chronic respiratory symptoms or an exacerbation of their disease.

Stage III: severe COPD: Characterized by further worsening of airflow limitation (FEV1/FVC , 0.70, 30% < FEV1 , 50% predicted), greater shortness of breath, reduced exercise capacity, fatigue, and repeated exacerbations that almost always have an impact on patients' quality of life.

Stage IV: very severe COPD: Characterized by severe airflow limitation (FEV1/FVC , 0.70, FEV1 , 30% predicted or FEV1 , 50% predicted plus the presence of chronic respiratory failure). Respiratory failure is defined as an arterial partial pressure of O2 (PaO2) less than 8.0 kPa (60 mm Hg), with or without an arterial partial pressure of CO2 (PaCO2) greater than 6.7 kPa (50 mm Hg) while breathing air at sea level. Respiratory failure may also lead to effects on the heart such as cor pulmonale (right heart failure).

Clinical signs of cor pulmonale include elevation of the jugular venous pressure and pitting ankle edema.

Patients may have stage IV COPD even if their FEV1 is greater than 30% predicted, whenever these complications are present. At this stage, quality of life is very appreciably impaired and exacerbations may be life threatening.

After a conclusive diagnosis made, effective COPD management should be planned. The planning must includes four components that is;

1. to assess and monitor the disease.
2. to reduce risk factor.
3. to manage stable COPD.
4. to manage exacerbations.

And also, the management should be aimed at the following goals; relieve symptoms, prevent disease progression, improve exercise tolerance, improve health status, prevent and treat complication, reduce mortality. Treatment benefits for COPD are primarily related to reduced exacerbations among patients who are more likely to have exacerbations, dyspnea that limits activity (Wilt TJ, 2007).

Pharmacologic therapy is used to prevent and control symptoms. Unlike asthma, the airflow limitation of COPD is poorly reversed with bronchodilating agents (Celli BR, 2004). Currently available treatment options provide only limited symptomatic relief, but

do nothing to forestall the inexorable course of disease progression and accelerated decline in pulmonary function (Nestor A. Molfino, 2007).

Bronchodilator medications are central to the symptomatic management of COPD (Klaus F. Rabe, 2007). They are given either on an as-needed basis for relief of persistent or worsening symptoms or on a regular basis to prevent or reduce symptoms.

Table 1.1: Example of beta2-agonist and anti-cholinergic use in COPD patient (Table adapted from the official website for Chronic Obstructive Pulmonary disease by the Chronic Obstructive Pulmonary Disease Association, Singapore).

Class	**Medicine**
Beta2 - agonists (short - acting)	Fenoterol Salbutamol
Anti-cholinergics (short - acting)	Ipratropium
Anti-cholinergics (Long - acting)	Tiotropium
Beta2- agonists (Long - acting)	Formoterol Salmeterol
Combination of beta2-agonist and anti-Cholinergic	Fenoterol and Ipratropium Salbutamol and Ipratropium

Inhaled corticosteroid is also a treatment in COPD. But regular treatment with inhaled glucocorticosteroids does not modify the long-term decline of FEV1 in patients with COPD (Klaus F. Rabe, 2007). The literature showed that monotherapy with long acting inhaled agonists, a long-acting inhaled anticholinergic, or inhaled corticosteroids was superior to placebo or short-acting anticholinergics in reducing exacerbations (Amir Qaseem, 2007). However, adding an inhaled corticosteroid to a long-acting agonist may reduce exacerbations compared with long-acting agonist monotherapy (Wilt TJ, 2007).

Pooling of the data from the 47 trials with 13,139 people showed that although inhaled steroids resulted in a small improvement in breathing tests initially, there was no long term benefit in the rate of decline in breathing capacity (Yang IA, 2007). Inhaled steroids were beneficial in slowing down the rate of decline in quality of life, and reducing the frequency of exacerbations (Yang IA, 2007).

The understanding of the risk factor is key point in management of COPD. Health care awareness and prevention is important in preventing COPD. Smoking cessation programme should be included in overall management of COPD. With the increasing trend of COPD, awareness of the health worker is important for early diagnosis of COPD. This is to to prevent life threatening exacerbations and improve quality of life.

1.2 DEFINITION OF VOICE DISORDER

A person is considered as having disorders of voice when the person's voice quality, pitch, or loudness differ significantly from others of same age, gender, cultural background, and geographic location, thereby drawing attention to the speaker (Coyle, 2001). Voice disorders may be caused by vocal misuse, overuse and abuse voice, medical or physical conditions, psychological factors or a combination of these factors (Ramig L. O., 1998).

Voice disorders may involve an abnormality of the larynx, which causes the laryngeal mechanism fail to meet the functional vocal needs of the speakers (Lehto, 2008). Symptoms of voice disorders often revealed by vocal fatigue, hoarseness, aphonia, strained voice, poor pitch and loudness modulation, and abnormal throat sensation during speech (Mattiske, 1998).

Voice disorders can be divided into two categories, which are organic voice disorders and functional voice disorders. Organic voice disorders are those disorders that have a known physical cause (Shipley, 2004). Functional voice disorders arise when the voice is used improperly. They typically results from vocal abuse and vocal misuse.

1.3 DEFINITION OF VOCAL SYMPTOMS

Symptoms refer to the individual's subjective complaint, either real or imagined (Aronson, 1990). Thus, vocal symptoms refer to the voice complaints reported by the respondents or patients. Generally, vocal symptoms starts slowly and gradually over time from sporadic to permanent lesions (Tavares, 2007). Vocal symptoms can be categorised into voice-related symptoms or phonatory symptoms (example: hoarseness, tired voice, low speaking voice, high note difficulty, weak voice, low noted difficulty, breathy, voice spasm) and physical discomfort or laryngopharyngeal symptoms (for example: tiring, effortful, ache, uncomfortable, chronic throat dryness, frequent throat clearing) (Roy, 2004).

The term hoarseness commonly used when a patient came with voice complaint. 'Hoarseness' is actually limited to deviant voice 'quality' (or timbre), and excludes pitch, loudness and rhythm factors (Philippe H. Dejonckere, 2001). Two main components of hoarseness have been identified, as shown by principal component analysis (PH Dejonckere, 1996):

(1) Breathiness (B): audible impression of turbulent air leakage through an insufficient glottic closure may include short aphonic moments (unvoiced segments).

(2) Roughness or harshness: audible impression of irregular glottic pulses, abnormal fluctuations in fundamental frequency (F_0), and separately perceived acoustic impulses (as in vocal fry), including diplophonia and register breaks.