Audiological assessment in patients with chronic obstructive pulmonary disease

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Background Chronic obstructive pulmonary disease (COPD), as a multisystemic disease, might have an impact on the auditory function. Thus, this study was designed for the audiological assessment of COPD patients to investigate the effect of smoking, and to further assess its possible correlation with the severity of COPD.

Patients and methods This prospective case–control study was conducted on 100 male patients with COPD with a mean age of 52.66±6.84 years. In addition, 25 healthy nonsmoker male participants with a mean age of 45.5±6.75 years were enrolled as the control group. For all COPD patients and controls, tympanometry and pure-tone audiometry at frequencies 250–8000 Hz were performed by an experienced audiologist.

Results Tympanometry type C was observed in the right ear of 30 COPD patients and in the left ear of 28 COPD patients. All low and high frequency tone audiometry differed significantly between COPD patients and controls ($P<0.001$), and the cutoff for changes in auditory function was 15 dB at both low and high frequency tones with 96% sensitivity and 100% specificity. Audiometry and tympanometry in COPD patients were not affected by either the smoking status or the type of smoking ($P>0.05$). Both low and high frequency tone audiometry correlated significantly and inversely with partial pressure of oxygen and forced expiratory volume in the first second, whereas the annual COPD exacerbations correlated significantly and directly.

Conclusion Changes in auditory function but not hearing loss is common in COPD and such audiological changes were not affected by smoking but correlates with the degree of airway obstruction and hypoxia as well as the rate of annual COPD exacerbation.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a multisystemic disease that often coexists with comorbidities that may have a significant impact on prognosis [1–8]. Some of these comorbidities arise independently of COPD, whereas others may be causally related, either with shared risk factors or by one disease increasing the risk or compounding the severity of the other [9]. Although many risk factors contribute to the development of COPD, cigarette smoking is still considered the most well-studied COPD risk factor [10]. Several studies reported the significant effect of smoking on hearing loss [11,12]. In a previous research, the impact of chronic hypoxemia secondary to COPD on the auditory function of these patients was investigated. The results showed a statistically significant difference for all auditory measures between patients with COPD and controls, but in general hearing impairment to date was not shown to be clinically relevant in patients with COPD [13,14]. Further, in stable patients with COPD and mild-to-moderate airflow obstruction, subclinical abnormalities of brainstem auditory evoked potentials have been observed [15].

In view of the above, this study was designed for the audiological assessment of patients with COPD in an attempt to investigate the effect of smoking on hearing, and to further assess the possible correlation between hearing impairment found with the severity of COPD.

Patients and methods

Study design and included patients

This prospective case–control study included 100 randomly selected COPD male patients selected from the outpatient clinic or inpatient admitted in the Chest Department at Ain Shams University Hospitals from June 2015 to February 2016. COPD was diagnosed according to the guidelines of the Global Initiative for Chronic Obstructive Lung Diseases [16]. In addition, 25 age-matched and sex-matched healthy nonsmoker male participants were enrolled in the study as the control group. Participants with a history of hearing loss, mental

impairment, intake of ototoxic drugs, diabetes mellitus, hypertension, frequent ear infection, ear surgery, familial deafness, and occupational exposure to noise were excluded from the study. All participants were clinically stable at the time of testing. The study was approved by the institutional ethical committee. Verbal consent was obtained from all included patients.

For all patients, the following were carried out: detailed medical history taking, thorough clinical examination, plain chest radiography, routine laboratory investi gations, arterial blood gases, and spirometry.

Lung function

Forced expiratory volume in the first second (FEV_1), forced vital capacity (FVC), FEV_1/FVC ratio, and forced expiratory flow over 25–75% part of FVC were measured using the spirometry system (Masterscreen 2001, version 4.5; Erich Jaeger GmbH, Germany). Readings were performed in triplicate, with the highest values recorded and expressed as a percentage of the predicted value according to the guidelines of the American Thoracic Society [17].

Audiometry and tympanometry

Audiometry and tympanometry were performed by an experienced audiologist. All included participants attended one hearing testing session in the audiology laboratory of Ain Shams University Hospitals. The audiologist, who was unaware of the smoking status of the study participant, performed an otoscopic examination. Pure-tone threshold audiometry was conducted in sound-isolated rooms or booths using a clinical audiometer in accordance with the Maximum Permissible Ambient Noise Levels for Audiometric Test Room [18]. Pure-tone audiometric air conduction testing is performed by presenting a pure tone (single frequency) to each ear through an earphone and the participant responding by pressing a button, raising hand, or saying 'yes' when stimuli were heard. Hearing thresholds were measured in each ear for the following frequencies: 250, 500, 1000, 2000, 4000, and 8000 Hz. Hearing threshold is defined as the lowest level in decibels at which a signal (tone) is heard 50% of the time according to standard clinical procedures [19]. Testing should begin with the better ear when identifiable, otherwise it is arbitrary. Test instructions were presented in the Arabic language.

The severity of the hearing loss (HL) was determined asfollows: 25±35 dB, mild impairment; 40±60 dB, moderate impairment; and greater than 65 dB, severe impairment.

Impedance tympanometer was used to evaluate the function of the middle ear system by applying the tip of a probe to seal off the entrance to the external ear canal; the air pressure within the enclosed ear canal cavity is gently changed from +200 to −200 mmH_2O, and the change in sound pressure level of a probe tone is graphed to verify the mobility of the eardrum.

Statistical analysis

Parametric numerical data were expressed as mean±SD, whereas nonparametric numerical data were expressed as number and percentage. The χ^2-test and/or Fisher exact test were applied to examine the comparison between two qualitative variables. The independent sample *t*-test was used to compare two groups as regards quantitative variables. One-way analysis of variance was used to compare more than two groups as regards quantitative variables. Receiver operating characteristic was plotted to identify the cutoff point for auditory changes. Spearman's correlation test was used to rank different variables against each other positively or inversely. Linear regression analysis was used to find the relationship between dependent and independent variables. Statistical significance was set at *P*-value less than 0.05. Statistical analyses were performed utilizing statistical package for the social sciences software (SPSS for Windows, version 20.0; SPSS Inc., Chicago, Illinois, USA).

Results

This study included 100 male patients with COPD ranging in age from 35 to 60 years with a mean age of 52.66±6.84 years. The control group comprised 25 healthy nonsmoker male participants ranging in age from 40 to 59 years with a mean age of 45.5±6.75 years. In the COPD group, 40 patients were current smokers, 30 patients were ex-smokers, and the remaining 30 patients were non smokers. On the basis of the severity of COPD, 66 (66%) patients had moderate COPD, 26 (26%) patients had severe COPD, and the remaining eight (8%) patients had very severe COPD. All low and high frequency tones of audiometry were significantly different between COPD and controls ($P<0.001$, Table 1).

Among COPD patients, tympanometry results showed type C (normal tympanic membrane mobility and negative middle ear pressure; consistent with Eustachian tube dysfunction) in the right ear of 30 patients and in the left ear of 28 patients (Table 2).In COPD patients, both audiometry and tympanometry of the right and the left ear did not differ statistically ($P>0.05$, Table 2).

The cutoff point for significant auditory changes in COPD was 15 dB at both low and high frequency tones with 96% sensitivity, 100% specificity, 100% positive predictive value, and 92.3% negative predictive value (Table 3 and Fig. 1).

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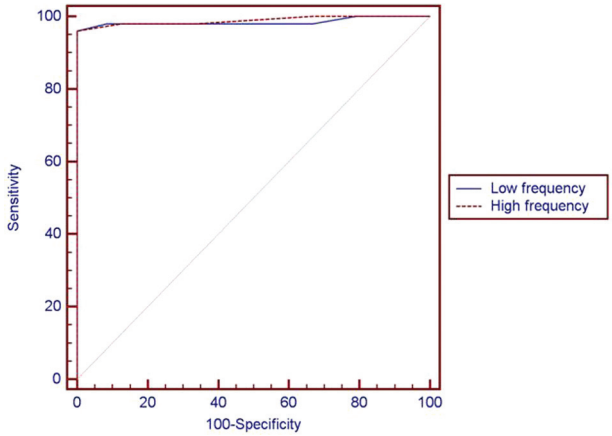
Results of audiometry and tympanometry in COPD patients were not affected by either the smoking status or the type of smoking ($P>0.05$, Tables 4 and 5).

Figure 1



Predictive performance of audiogram in chronic obstructive pulmonary disease.

Table 1 Comparison between audiometry in chronic obstructive pulmonary disease patients and controls

Audiometry	Control (*N*=25) [mean±SD (range)]	COPD (*N*=100) [mean±SD (range)]	*P*-value
Low-frequency tone (Hz)			
250	10.20±3.67 (5–15)	29.40±7.26 (15–45)	0.000
500	11.20±3.32 (5–15)	29.08±8.74 (15–50)	0.000
1000	12.40±2.93 (5–15)	31.40±10.05 (15–70)	0.000
Low-frequency tones (250, 500, 1000)	11.11±2.23 (6.67–15)	29.96±7.77 (15–48.33)	<0.001
High-frequency tone (Hz)			
2000	13.00±2.50 (10–15)	29.90±12.02 (15–70)	0.000
4000	10.40±3.20 (5–15)	42.60±12.67 (10–85)	0.000
8000	11.40±3.39 (5–15)	45.30±15.99 (10–95)	0.000
High-frequency tones (2000, 4000, 8000)	11.67±2.14 (6.67–15)	39.27±10.54 (15–75)	<0.001

COPD, chronic obstructive pulmonary disease.

Table 2 Comparison between right and left audiometry and tympanometry in chronic obstructive pulmonary disease patients

COPD (*N*=100)	Right ear [mean±SD (range)]	Left ear [mean±SD (range)]	*P*-value
Low-frequency tone (Hz)			
250	29.40±7.26 (15–45)	29.20±8.35 (15–50)	0.828
500	29.08±8.74 (15–50)	28.30±8.24 (15–50)	0.376
1000	31.40±10.05 (15–70)	29.70±10.47 (15–70)	0.104
Low-frequency tones (250, 500, 1000)	29.96±7.77 (15–48.33)	29.07±8.45 (15–53.33)	0.229
High-frequency tone (Hz)			
2000	29.90±12.02 (15–70)	30.60±10.63 (10–60)	0.584
4000	42.60±12.67 (10–85)	39.20±11.04 (15–90)	0.063
8000	45.30±15.99 (10–95)	45.10±19.13 (15–110)	0.914
High-frequency tones (2000, 4000, 8000)	39.27±10.54 (15–75)	38.30±11.75 (15–86.67)	0.411
Tympanometry [*n* (%)]			
Type A	70 (70)	72 (72)	0.824
Type C	30 (30)	28 (28)	

COPD, chronic obstructive pulmonary disease; type A, normal tympanic membrane mobility and normal middle ear pressure; type C, normal tympanic membrane mobility and negative middle ear pressure consistent with Eustachian tube dysfunction.

Table 3 Predictive performance of audiogram in chronic obstructive pulmonary disease

	Cutoff point (dB)	AUC	Sensitivity	Specificity	PPV	NPV
Low frequency	>15	0.985	96	100	100	92.3
High frequency	>15	0.989	96	100	100	92.3

AUC, area under the curve; NPV, negative predictive value; PPV, positive predictive value.

Table 4 Comparison between smoking statuses in chronic obstructive pulmonary disease

Variables	Current smoker (*N*=40)	Exsmoker (*N*=30)	Nonsmoker (*N*=30)	*P*-value
Age [mean±SD (range)] (years)	52.80±7.53 (35–60)	55.00±4.61 (47–60)	50.13±7.28 (36–60)	0.150
BMI [mean±SD (range)]	26.75±5.72 (17–35)	28.35±4.01 (22–35)	29.4±4.75 (21–37)	0.292
Comorbidities [*n* (%)]				
No	28(70.0)	28 (93.3)	24 (80.0)	0.233
Yes	12 (30.0)	2 (6.7)	6 (20.0)	
FEV_1/FVC [mean±SD (range)]	54.73±9.20 (38–66)	58.00±6.77 (45–67)	58.40±8.09 (45–68)	0.346
FEV_1 [mean±SD (range)] (% predicted)	47.90±16.04 (21–73)	55.47±18.50 (16–77)	64.07±15.36 (21–75)	0.024
Exacerbation/year [mean±SD (range)]	4.25±2.12 (1–8)	3.80±2.43 (1–8)	2.53±2.45 (1–8)	0.098
PO_2 on RA [mean±SD (range)] (mmHg)	59.55±16.03 (42–92)	64.53±18.57 (40–93)	76.87±17.89 (44–98)	0.018
Low-frequency audiometry [mean±SD (range)] (Hz)	31.42±8.06 (18.33–48.33)	29.33±8.97 (18.33–45)	28.64±6.12 (15–38.33)	0.550
High-frequency audiometry [mean±SD (range)] (Hz)	39.25±11.91 (20–75)	38.56±7.26 (21.67–53.33)	40.00±11.92 (15–71.67)	0.935
Tympanometry [*n* (%)]				
Type A	24 (60)	20 (66.7)	26 (86.7)	0.221
Type C	16 (40)	10 (33.3)	4 (13.3)	

FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; PO_2, partial pressure of oxygen; RA, room air.

Table 5 Comparison of types of smoking in chronic obstructive pulmonary disease

Variables	Cigarette smoker (*N*=44)	Shisha smoker (*N*=26)	*P*-value
Audiometry low frequency [mean±SD (range)] (Hz)	29.24±8.19 (18.33–45)	32.69±8.62 (20–48.33)	0.099
Audiometry high frequency [mean±SD (range)] (Hz)	38.11±8.79 (21.67–56.67)	40.38±12.16 (20–75)	0.690
Tympanometry [*n* (%)]			
Type A	28 (63.6)	16 (61.5)	0.240
Type C	16 (36.4)	10 (38.5)	

Table 6 Correlation of audiometry and tympanometry with several variables in chronic obstructive pulmonary disease

Variables	Audiometry			
	Low frequency		High frequency	
	r	*P*-value	*r*	*P*-value
Age	0.179	0.213	0.061	0.676
Smoking index (packs/year)	0.279	0.050	0.176	0.221
Duration of smoking	0.132	0.359	0.036	0.805
FEV_1/FVC	–0.152	0.291	–0.109	0.451
FEV_1	–0.515	0.000	–0.330	0.019
Exacerbations/year	0.507	0.000	0.369	0.008
PO_2 on RA	–0.631	0.000	–0.468	0.001

FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; PO_2, partial pressure of oxygen; RA, room air.

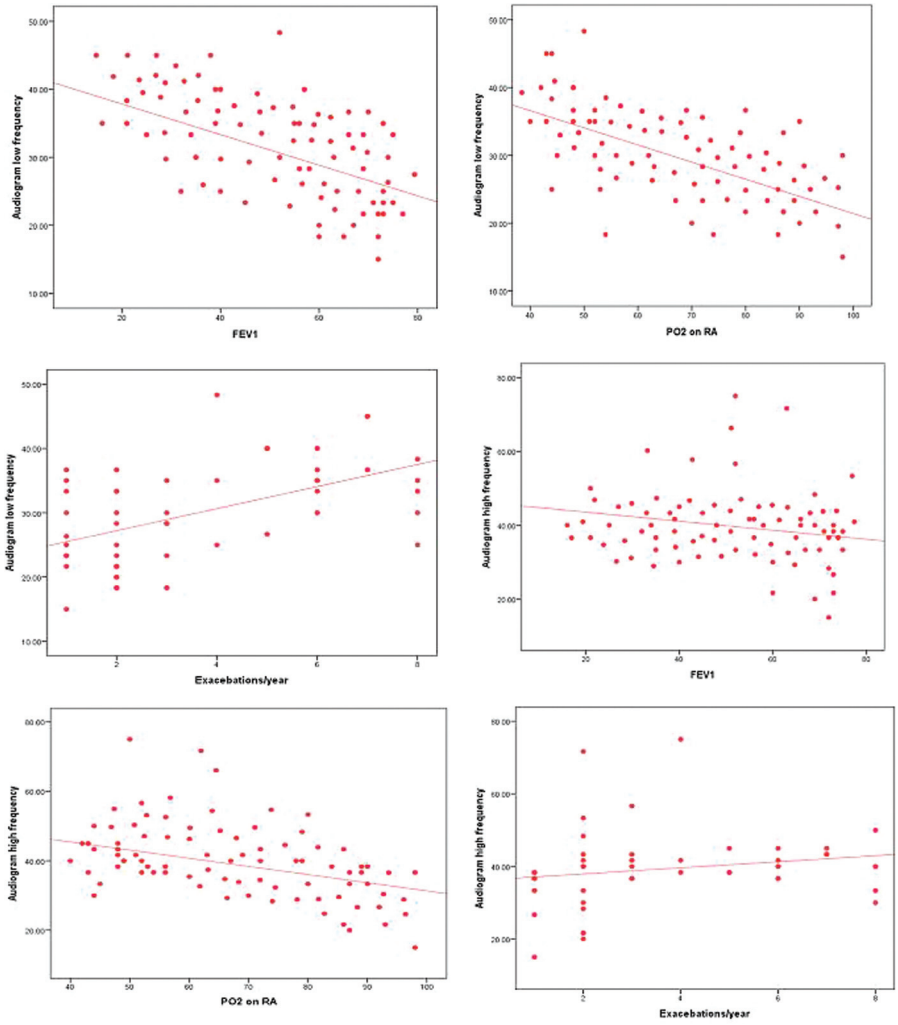
Logistic regression showed that both low and high frequency tone audiometry correlated significantly and inversely with partial pressure of oxygen (PO_2) on room air as well as FEV_1, whereas the exacerbations of COPD per year correlated significantly and directly (Table 6 and Fig. 2).

Discussion

On the basis of the above results, our study proved a significant difference in auditory measures but not hearing loss in COPD patients in comparison with normal controls. The cutoff point detected in our study for significant changes in auditory function in COPD was 15 dB at both low and high frequency tones with 96% sensitivity and 100% specificity. This cutoff point is lower than the lower limit for hearing loss identified in our study; thus, the audiological assessment of COPD patients in our study demonstrated changes that were far below the threshold limit for the occurrence of hearing loss. These results are in accordance with several reports documenting that hearing impairment was clinically irrelevant in patients with COPD [13,14,20].

Although smoking is regarded as the main risk factor for the development of COPD, our study

Figure 2



Linear regression analysis of low-frequency and high-frequency tone audiometry.

revealed that neither the smoking status nor the type of smoking affected the degree of impairment in auditory measures. On reviewing the literature for studies addressing the possible effect of smoking on hearing, the results were conflicting; some studies reported a significant association between smoking and increased risk for hearing loss [12,21–28], whereas other studies demonstrated no correlation between hearing loss and smoking [29,30].

Auditory changes correlated directly with the rate of COPD exacerbation/year where the increase in the number of these exacerbations significantly increased the degree of auditory changes. Both FEV_1 and PO_2 also correlated with auditory changes but in an indirect way, wherein the increase in airway obstruction and hypoxia had a significant effect on audiological measures. Several previous studies have documented that the transduction mechanism of the inner ear is highly dependent upon the cochlear oxygen supply, such that hypoxia locally will be accompanied by loss of sensitivity [31–33]. In another study, the results suggested poorer central auditory function in hypoxemic patients than in normally oxygenated individuals [34]. One recent study showed a statistically significant difference for all audiological measures between the control group and a COPD subgroup – the presumptive hypoxic patients (partial oxygen tensions, PO_2, <75 mmHg). Furthermore, PO_2 correlated with the changes observed in all audiological measures [13]. Our results showed no correlation between the changes in audiological measures and several variables, including age, smoking index, and the duration of smoking. In contrast to our results, studies found a significant statistical association between hearing loss and the number of cigarettes smoked and the duration of smoking [11,21,23,24,35–37]. Other studies demonstrated a correlation between age and hearing loss [27,37].It is worth mentioning that this study has some limitations: smoking was the only risk factor for COPD tested; the included COPD patients were all male, and thus a selection bias cannot be excluded; and the level of

hypoxia was not severe enough to test the possible effect of severe chronic hypoxia on the inner ear.

In conclusion, changes in auditory function but not hearing loss is common in COPD patients in comparison with normal controls and such audiological changes were not affected by smoking but correlates with the degree of airway obstruction and hypoxia as well as the rate of annual exacerbation of COPD.

Hopefully, this study might pave the way for large-scale studies investigating thoroughly the effect of various risk factors for COPD on auditory function in an attempt for early detection of any hearing impairment among COPD patients to further carry out a timely intervention for its correction.

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Conflicts of interest
There are no conflicts of interest.

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