Peak nasal inspiratory flow in chronic obstructive pulmonary disease*

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Abstract

Background: The nasal airflow in chronic obstructive pulmonary disease (COPD) is poorly characterized. Peak nasal inspiratory flow (PNIF) is a valuable instrument for assessing nasal airflow and the effect of pulmonary pathology such as COPD on PNIF remains unknown. To test the hypothesis that nasal airflow is reduced in COPD, we assessed airflow using PNIF in COPD and a control group. We also explored whether there is an association between COPD, chronic rhinosinusitis without nasal polyps (CRSsNP), and other predefined covariates with PNIF.

Methodology: Ninety patients with COPD and 67 controls underwent PNIF and spirometry. The associations between PNIF and COPD and pre-bronchodilator forced expiratory volume in the first second (FEV_1) (% predicted) were assessed by multivariable linear regression in two separate models.

Results: PNIF was significantly lower in the COPD group than in the control group. Multivariable linear regression showed that COPD and pre-bronchodilator FEV_1 (% predicted) were significantly associated with lower PNIF after adjustment for age, sex, CRSsNP, weight and height. CRSsNP was not associated with PNIF in either of the adjusted regression analyses.

Conclusions: PNIF is lower in COPD than in a control group. The finding of a low PNIF in the absence of disease in the upper airways may be due to obstructive lower airways diseases and special care should be taken when interpreting PNIF values in patients with COPD or reduced FEV_1.

Key words: COPD, PNIF, chronic rhinosinusitis, smoking, spirometry

Introduction

Chronic obstructive pulmonary disease (COPD) is ranked as the third leading cause of mortality and is expected to become the leading cause of death worldwide in 15 years [1, 2]. Associations with sinonasal symptoms and chronic rhinosinusitis without nasal polyps (CRSsNP) have been reported in epidemiological [3] and observational studies [4, 5], and a prevalence of 51% of CRSsNP was recently reported in an observational study of a larger sample of COPD subjects [5]. Moreover, nasal symptoms in COPD increase progressively over time [6].

The concept of united airways diseases (UAD) suggests that diseases of the upper airways coexist with diseases of the lower airways, and vice versa [7]. Upper airway inflammatory diseases such as allergic rhinitis (AR), chronic rhinosinusitis with nasal polyps (CRSwNP) and CRSsNP often have lower airway inflammatory diseases such as asthma, COPD, bronchiectasis and cystic fibrosis [8]. The former are characterized by nasal obstruction and a reduction of nasal airflow.

Airflow through the nasal cavity is influenced by physiologic and pathologic conditions. Nasal patency can be quantified with a peak nasal inspiratory flow (PNIF) meter. This technique is inexpensive, fast, portable, simple and has good reproducibility and is highly recommended for the evaluation of nasal obstruction in clinical practice and research [9]. PNIF is significantly associated with asthma [10] and is correlated with lower airway patency in asthma with AR [11].

We are not aware of any previous study that has evaluated nasal airflow in COPD with PNIF measurement. To better our understanding of the nasal airflow in patients with COPD, this study assesses PNIF in COPD and a corresponding control group with respect to age and sex and explores the association of PNIF with COPD, CRSsNP and other predefined covariates.

Materials and methods

Study design and setting

A cross-sectional study was conducted on 90 COPD patients and 67 controls, aged 40-80 years, between February 2016 and December 2017. The study sample has been previously described [5, 12, 13]. Briefly, COPD patients were recruited from the hospital respiratory outpatient and physical therapy clinics, general practitioner offices and a private pulmonology practice. Controls with no known disease of the upper and lower airways were recruited locally from nearby businesses, multiple retirement associations and via the hospital's social media page. Exclusion criteria for study participation were asthma (including Asthma on COPD overlap), pregnancy or breastfeeding, upper- and lower respiratory tract infection within the previous two weeks, previous sinonasal surgery, nasal polyps, cystic fibrosis, Parkinson's disease, Alzheimer disease, ongoing radio-chemotherapy or use of long-term oxygen therapy.

All subjects gave written informed consent, and all examinations and questionnaires were completed on the same day. The study was approved by the Regional Committee for Medical and Health Research Ethics, Central Norway, REC (reference number 2015/2017), and investigations were performed in accordance with the principles of the Declaration of Helsinki/Hong Kong.

Methods

All subjects were instructed to discontinue the use of systemic corticosteroids and antihistamines for 4 days and nasal decongestants 12 hours before the inclusion visit. Nasal corticosteroids were continued. COPD patients were instructed not to take their morning inhaled medication in accordance with published protocols [14].

Questionnaires on sinonasal- and allergic symptoms affecting the airways and smoking status were self-administered, and measures of weight and height were done by a research nurse. The diagnosis of COPD was confirmed by the presence of irreversible airflow obstruction defined as a post-bronchodilator forced expiratory volume in 1 second (FEV_1) to forced vital capacity (FVC) ratio of < 0.7, and the severity of airflow obstruction was graded according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2014 criteria [15]. The clinical diagnosis of CRSsNP was based on the presence of the symptomatic criteria of rhinosinusitis [5] and a positive nasal endoscopy and in accordance with the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS) 2012 definition [16].

Peak nasal inspiratory flow

Nasal patency was assessed with a portable PNIF meter (Incheck Nasal, Clement Clarke International, Harlow, Essex, UK). A forced maximum inhalation through the nose from residual volume was performed with the subject sitting in an upright position. Three satisfactory maximal inspirations were obtained and the highest of these results was taken as the PNIF value [17]. The scale on the PNIF meter was from 30-370 litres/minute (l/min).

Nasal obstruction

A question on nasal obstruction (NO-SNOT-22) over the past two weeks in the 22-item Sino-Nasal Outcome Test (SNOT-22) [18] was answered on a Likert scale with a response range from 0-5, where 0 equals no problem and 5 equals problem as bad as it could be. A response of 0-1 and 2-5 was defined as "symptom not present" and "symptom present", respectively [3].

The subjective degree of nasal obstruction during the previous two weeks was assessed on a 100 mm Visual Analog Scale (NO-VAS), with 0 mm as not troublesome and 100 mm as worst thinkable troublesome.

Nasal endoscopy

All subjects underwent a clinical ENT examination with nasal endoscopy (2.7mm, 0° True View II endoscope, Olympus, Japan) of the nasal cavity, to exclude tumours and nasal polyps. The endoscopic appearance of the nasal cavity was assessed using the modified Lund-Kennedy endoscopy score (MLK) [19] based on polyp extent (none with polyps were included in this study), oedema (0: absent; 1: mild; 2: severe) and discharge (0: none; 1: clear; 2: thick and purulent).

Spirometry

Pulmonary function tests were performed before and 10 minutes after administration of 0.4 mg salbutamol aerosol by a spacer and in accordance with European Respiratory Society (ERS) guidelines for spirometry [14]. The best FEV_1 in litres, percentage of predicted (% predicted) was recorded. Predicted normal values were based on reference values of Crapo et al. [20].

Allergy

Skin prick testing (SPT) with an allergen panel consisting of

Table 1. Subject characteristics.

	COPD		Control		P value
N	90		67		
Age years	66.2	(8.7)	63.5	(8.9)	0.06
Female	41	(45.6)	30	(44.7)	0.22
CRSsNP	46	(51.1)	NA	NA	
AR	5	(5.6)	NA	NA	
Smoking status					
Current	17	(18.9)	4	(6.0)	**<0.001**
Former	68	(75.5)	33	(49.3.)	
Never	5	(5.6)	30	(44.7)	
Height (cm)	172.1	(9.8)	172.8	(9.9)	0.70
Weight (kg)	80.6	(18.8)	81.4	(15.7)	0.75
MLK	2.8	(2.0)	1.0	(1.5)	**<0.01**
PNIF	131.6	(35.4)	159.3	(42.9)	**<0.001**
FEV_1 (% pred) pre	49.0	(18.1)	92.0	(13.2)	**< 0.001**
FEV_1 (% pred) post*	53.1	(18.7)	94.7	(12.5)	**< 0.001**
NO-SNOT-22[§]	54	(60)	14	(20.9)	**< 0.001**
NO-VAS	23.4	(25.9)	9.36	(15.1)	**<0.001**

Data presented as n (%) or mean (SD) unless otherwise stated. P-values refer to data comparisons between COPD and controls. * post-bronchodilator values with pre-bronchodilator values in 2 controls and 1 COPD. [§] Number (%) of participants with a SNOT-22 response of 2-5 defined as "symptom present".

Abbreviations: COPD: chronic obstructive pulmonary disease; CRSsNP: chronic rhinosinusitis without nasal polyps; AR: allergic rhinitis; MLK: modified Lund Kennedy endoscopy score; PNIF: peak nasal inspiratory flow; pre: pre-bronchodilator; post: post-bronchodilator; FEV_1% pred: forced expiratory volume in 1 second (% predicted); NO-SNOT-22: nasal obstruction SNOT-22; NO-VAS: nasal obstruction VAS. NA: not applicable.

birch, grass and mugwort pollen, cladosporium, house dust mite (*Dermatophagoides pteronyssinus*), and horse, dog, and cat epithelia, together with positive and negative controls (Soluprick SQ, ALK-Abello, Horsholm, Denmark) were performed. A diagnosis of AR was based on self-reported symptoms characteristic of rhinoconjunctivitis within the last 12 months on exposure to the specific allergen(s) found positive on SPT. All subjects allergic to pollens were included outside of the allergy season.

Sample size

A sample size analysis showed that 63 subjects were needed in each group to detect a difference of 25 l/min in PNIF between the groups with a significance level of 0.05 and a power of 80%.

Statistical analysis

For the statistical analysis, IBM SPSS 25.0 was used. Continuous variables are presented as means and standard deviations (SD). For group comparisons, an independent t-test was used for normally distributed data and the Mann–Whitney U test was used for non-normally distributed data. Categorical variables are presented as numbers (n) and proportions (%) and were analysed using Chi-Square tests or Fisher's Exact Test. Spearman's rank correlation coefficient was used to evaluate the relationship between PNIF and subjective variables and MLK.

As there was a strong association between COPD and FEV_1 (% predicted), we fitted two separate models for PNIF as a continuous variable to avoid multicollinearity. Pre-bronchodilator FEV_1 (% predicted) was chosen for linear regression in model 2 as it is more accessible than post-bronchodilator FEV_1 in clinical otolaryngological practice. After checking that the assumption of normality was fulfilled, multiple linear regression analysis was undertaken to investigate variables associated with PNIF and is presented with β and 95% confidence intervals (CI). Variables of interest that have been shown to influence PNIF in other published studies were age [21], sex [22], weight [23], height [21], AR [24] and CRSsNP [25]. AR was omitted from the regression analysis as a diagnosis of AR was made in 5 subjects in the COPD group and no subject in the control group. A difference was considered significant at a p-value of < 0.05.

Results

Data on age, sex, CRSsNP, AR, smoking status, height, weight and spirometry values for the two groups are summarized in Table 1. The MLK and NO-VAS scores were significantly higher

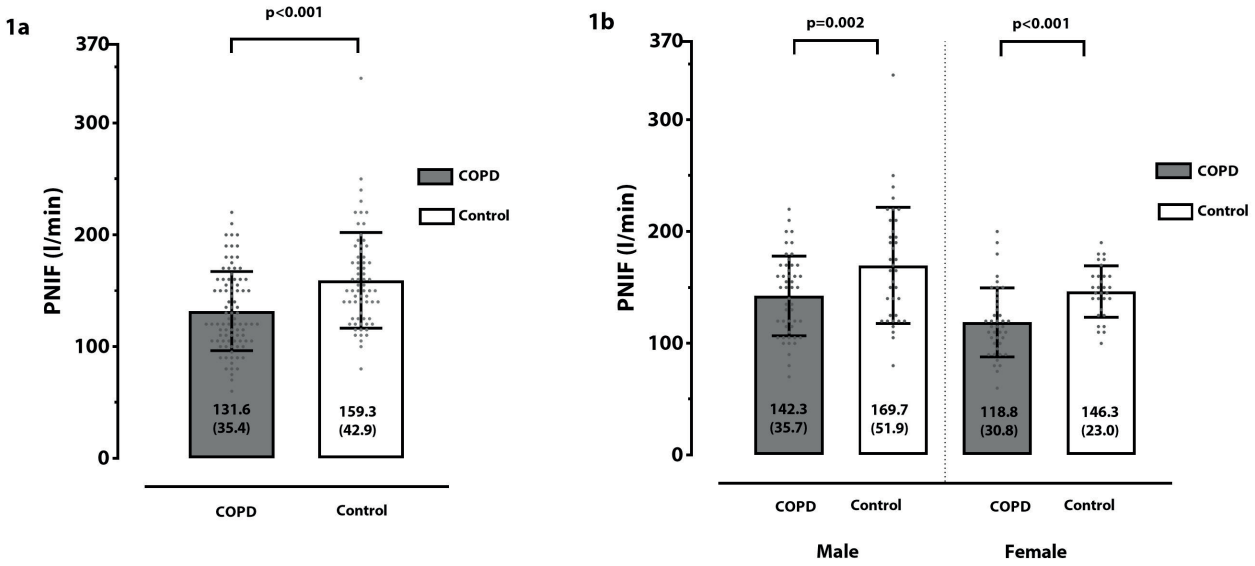

Figure 1. PNIF in COPD and control group (a), and in male and female sex (b). Data presented as mean (SD). Abbreviations: COPD: chronic obstructive pulmonary disease; PNIF: peak nasal inspiratory flow; l/min: litres/minute.

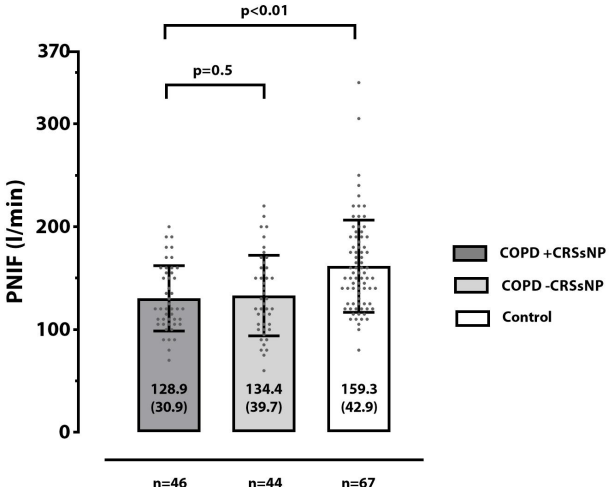

Figure 2. PNIF in COPD with and without CRSsNP and the control group. Data presented as mean (SD). Abbreviations: COPD: chronic obstructive pulmonary disease; +CRSsNP=with chronic rhinosinusitis sin nasal polyposis; -CRSsNP: without chronic rhinosinusitis sin nasal polyposis; PNIF: peak nasal inspiratory flow; l/min: litres/minute.

Table 2. Correlation between PNIF and NO-SNOT-22, NO-VAS and MLK.

Variable	PNIF			
	COPD		Control	
	Correlation coefficient	P-value	Correlation coefficient	P-value
NO-SNOT-22	-0.17	0.88	0.18	0.16
NO-VAS	-0.06	0.57	0.15	0.22
MLK	-0.10	0.93	0.03	0.80

Data presented with Spearman's pairwise rank correlation coefficient and P-value.
Abbreviations: PNIF: peak nasal inspiratory flow; COPD: chronic obstructive pulmonary disease; NO-SNOT-22: nasal obstruction SNOT-22; NO-VAS: nasal obstruction VAS; MLK: modified Lund Kennedy endoscopy score.

in the COPD group than in the control group. The prevalence of "symptoms present" of the NO-SNOT-22 was over threefold greater in the COPD group than in the control group.
PNIF was significantly lower in the COPD group than in the control group (Figure 1a).
On subgroup analysis, PNIF was significantly lower in males and females in the COPD group than in the control group (Figure 1b). In the COPD group, PNIF was not significantly different in the subgroup with CRSsNP than without CRSsNP (Figure 2) and in the subgroup with and without AR [mean (SD) 132.0 (26.6) vs 131.6 (36.0), p=0.9].
PNIF was not correlated with NO-SNOT-22, NO-VAS and MLK in COPD or controls (Table 2).
Of the two regression models which were computed, the association between COPD and PNIF is shown in model 1 (Table 3). COPD was significantly associated with lower PNIF after adjustment for age, sex, weight, height and CRSsNP.
The association between pre-bronchodilator FEV_1(% predicted) and PNIF is shown in model 2 (Table 3). Higher FEV_1 (% predicted) was significantly associated with higher PNIF after

Table 3. Linear regression with PNIF as the outcome variable.

PNIF	Unadjusted analysis β (95% CI)		P-value	Multivariable analysis β (95% CI) Model 1 with COPD		P-value	Multivariable analysis β (95% CI) Model 2 with FEV_1%		P-value
COPD [yes]	-27.6	-40.0 to -15.3	**<0.001**	-23.4	-37.5 to -9.3	**0.001**			
FEV_1 [%]	0.5	0.3 to 0.8	**<0.001**				0.5	0.3 to 0.8	<0.001
Age [years]	-0.9	-1.6 to -0.2	**0.01**	-0.7	-1.4 to -0.03	**0.04**	-0.7	-1.3 to -0.03	0.02
Sex [male]	23.7	11.2 to 36.2	**<0.001**	16.9	0.4 to 33.3	**0.04**	20.7	4.6 to 36.9	0.01
Weight [kg]	0.4	0.07 to 0.8	**0.02**	0.06	-0.3 to 0.4	0.7	0.09	-0.3 to 0.5	0.7
Height [cm]	1.3	0.6 to 1.9	**<0.001**	0.5	-0.4 to 1.4	0.3	0.4	-0.5 to 1.3	0.6
CRSsNP [yes]	-18.7	-32.6 to -4.7	**0.009**	-3.6	-18.9 to 11.8	0.6	-1.6	-16.3 to 13.2	0.8
Adjusted R^2				23.5			26.2		

Model 1: Including COPD and covariates; Model 2: Including pre-bronchodilator FEV_1 (% predicted) and covariates. The number of subjects in analysis=157. Abbreviations: PNIF: peak nasal inspiratory flow; COPD: chronic obstructive pulmonary disease; CRSsNP: chronic rhinosinusitis sin nasal polyps; FEV_1[%]: pre-bronchodilator forced expiratory volume in 1 second (% predicted).

adjustment for age, sex, weight, height and CRSsNP. CRSsNP was not associated with PNIF in either of the adjusted regression analyses.

Discussion

This study demonstrates that PNIF is lower in COPD than in the control group and in females than in males in both groups. Furthermore, PNIF is not significantly different in those with and without CRSsNP or AR in the COPD group. Regression analysis shows that a lower PNIF is associated with COPD in model 1, with pre-bronchodilator FEV_1 (% predicted) in model 2 and with female sex, and higher age in both models and is not associated with weight, height and CRSsNP in either model.

Our finding in model 1 that PNIF is lower in COPD compared to controls extends the findings for PNIF in other UAD such as asthma [10]. PNIF has also been reported to be lower in sleep-related breathing disorders [26, 27] and CRS [25]. Thus, it is important to be aware that a low PNIF in the absence of upper airway diseases may be due to obstructive diseases of the lower airways, such as asthma and COPD. As the PNIF measurement is made after exhalation to residual volume, the lower PNIF in COPD and asthma may be accounted for by the increase in residual volume that has been reported in those diseases [28, 29].

In model 2, we show that a lower pre-bronchodilator FEV_1 (% predicted) is associated with a lower PNIF in the multivariable regression analysis and that PNIF increases by 0.5 l/min per % increase in FEV_1 (% predicted). In comparison, in a similar study of patients with asthma and healthy controls, the increase in PNIF was 0.3 l/min per % increase in pre-bronchodilator FEV_1 (% predicted) [10]. Other studies have also shown an association between nasal patency assessed by PNIF and lower airway airflow limitation assessed by pre-bronchodilator FEV_1 [11] and by peak expiratory flow (PEF) [23]. In the former study, PNIF was assessed in children (6-12 years) with asthma and rhinitis and age-matched healthy controls. The latter study assessed PNIF in assumed healthy volunteers. However, this assumption was not confirmed by an extensive evaluation of the lower airways, such as spirometry and reversibility testing. In contrast, the present study includes both COPD and healthy controls, and the more robust measurement of FEV_1 was included as a covariate to enable a more precise assessment of the role of obstructive diseases of the lower airways on the PNIF measurements. A direct relationship of nasal patency with FEV_1 (% predicted) in COPD has been reported by Hurst et al. [30]. However, that study used the more expensive and complicated technique of acoustic rhinometry and did not have a control group. Their findings are confirmed in the present study which used the PNIF meter to assess nasal patency and included a control group. This instrument is inexpensive, easily available and simple to use.

PNIF was associated with the demographic factors of sex and age and was not associated with anthropometric factors of weight and height in both models. Our finding of an association of male sex and younger age with a higher PNIF is in accordance with past studies [21, 22]. With regard to anthropometric factors, there are divergent findings in the literature. The absence of an association of PNIF with weight in the present study is also in accordance with that of a large study of a general Swedish population [22]. PNIF was also correlated with height in that study and was associated with height in another study [21]. In contrast, no association of PNIF with height was found in a study of an

Italian general population [23].

Our study showed that PNIF was not significantly different in COPD patients with and without CRSsNP (model 1). To our knowledge, there are to date no studies of PNIF in COPD patients with CRSsNP, and the absence of an association between CRSsNP and PNIF in our study may be due to a type 2 error. Furthermore, in the study by Araújo-Martins et al. [25] PNIF was lower in CRSsNP than in healthy controls. However, that study did not evaluate the lower airways of the CRSsNP patients and comorbid obstructive lung disease could potentially cause the lower PNIF values.

Nasal obstruction assessed by the participant with SNOT-22 and VAS was not correlated with PNIF in either the COPD or the control group. The sensation of nasal obstruction is multifactorial and may explain the absence of a correlation. These factors include intranasal anatomical geometry, the autonomic nervous system and various physiological and pathological factors [32], the patient's psychological status, and expectations of the patient. Thus, the subjective evaluation of nasal obstruction may be a synthesis of these different factors. Despite this, some studies have found such a correlation [33] and others have not [34]. Ottaviano et al. [35] found a weak but significant negative correlation ($r = -0.13$, $P = 0.001$) between PNIF and VAS nasal obstruction, and Ta et al. [36] found the strongest correlation between PNIF and patient-reported outcome measures and as such advocating for its use both for clinical and research purposes. Nevertheless, it could be favourable to use other questionnaires such as the NOSE [37] to evaluate nasal obstruction in COPD patients. Past studies have not found a correlation between nasal endoscopic findings and PNIF in CRS with and without NP [38, 39]. In the present study, we did not find any correlation of PNIF with MLK in either the COPD or the control group. To date, there are no studies that have examined the relationship between PNIF and nasal endoscopic findings in COPD.

Strengths and limitations

The main strengths of this study are the large sample size of patients with COPD and the inclusion of control subjects from a comparable population for the assessment of PNIF. We also had an almost equal distribution of the sexes in the COPD and control groups, and we performed regression analysis to investigate variables that could affect the PNIF measures. Further, the use of standardized criteria for diagnosis of COPD and CRSsNP ensures that the results are not attributable to asthma and nasal polyps. Some limitations should also be addressed. The participants in the present study were of white Caucasian descent, and as such the generalizability of our results to other populations may be limited. Further, the low number of non-smokers in the COPD group and current smokers in the control group precluded the exploration of a possible interaction between smoking and PNIF. As smoking is the leading cause of COPD [40] and may affect nasal airflow [41] it would have been desirable to have had statistical strength to include an interaction term. Further, the absence of an association between CRSsNPand PNIF may be due to a type 2 error as discussed earlier. Past studies have found that PNIF is lower in subjects with AR than in subjects without AR [24] and is closely related to signs of rhinitis in an adult general population [31]. However, we were unable to investigate the association between AR, PNIF and FEV_1 due to the low number of participants with AR in the COPD group. Lastly, possible reasons for inaccuracy of the PNIF measures include loose-fitting face masks or incompletely closed mouths, but these errors were minimalized as the measurements were performed by the same trained operator throughout the study.

Clinical implication

Although PNIF was significantly lower in COPD than in the control group, the clinical implication of our finding is uncertain. The absolute value for PNIF does not differentiate a healthy subject from a COPD subject (Figure 1a) or a COPD subject with CRSsNP from a COPD subject without CRsNP (Figure 2). Normative values for PNIF have been reported, but these vary considerably [21, 42, 43]. Moreover, in the Ottaviano et al. [21] study, considerable residual variability between individuals due to factors other than age, sex and height precluded the derivation of % predicted PNIF. Ideally, PNIF should be expressed as % predicted to allow comparison between individuals and thus identification of individuals with an abnormal PNIF value. Larger population studies that include a broader range of age groups, anthropometric factors and possibly FEV_1 or PEF are needed to define a more robust set of normative values and consequently enable the comparison of PNIF values in healthy individuals and patients with obstructive pulmonary diseases.

Conclusion

PNIF is lower in COPD than in a control group. The finding of a low PNIF in the absence of disease in the upper airways may be due to obstructive lower airways diseases, and special care should be taken when interpreting PNIF values in patients with COPD or reduced FEV_1.

Authorship contribution

WMT: Study design, data collection, statistical analysis, paper drafting; MRØ: Study design, data collection, paper drafting; MSC: Study design, paper drafting; SKS: Study design, paper drafting; ASH: Study design, statistical analysis, paper drafting.

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Conflict of interest

None declared.

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