Assessment of Lung Functions and Factors Affecting in Chronic Obstructive Pulmonary Disease (COPD) Patients

Anup Vegad[1], Maulik Varu[2], Vilas Patel[3], Varsha Joshi[4]

Abstract

Introduction: COPD is a major cause of chronic morbidity and mortality throughout the world. Its prevalence & mortality due to it are increasing day by day. Current prevalence in India is not well understood but condition is worsening. So the aim of present study is to compare and to assess the severity of lung dysfunctions in the patients with COPD & to study the effects of various factors like BMI, BSA, smoking, residence etc. on lung dysfunctions in COPD patients in rural & urban areas of Jamnagar district, Gujarat. **Material and Method:** Lung functions of 50 COPD patients of various age groups were measured by doing spirometry (Spiro-Analyzer St-90) in stable phase of disease & spirometric parameters are compared with their predicted values. Effects of smoking, BMI, BSA, residence, seasons etc. on severity of COPD were measured. The various data were collected; compiled, statistically analyzed and valid conclusions were drawn. **Observation and Results:** Study results showed the mean values of FVC, FEV1, FEV1/FVC%, FEF25-75, PEFR and MVV were greatly decreased in COPD patients (more profound in smokers) as compared with their predicted value, which were statistically significant. There was positive correlation of spirometric parameters with BMI and BSA. Majority of patients were in severity grade 2 (46%) and grade 3 (46%) (suggested by ATS). **Conclusion:** All spirometric parameters were greatly reduced in COPD patients. COPD was more prevalent in urban area and disease was more severe with smoking history & patients with low BMI & BSA values and worsens in winter. So by maintaining proper nutrition we can reduce the severity of the disease.

Key Words: Body mass index, Body surface area, chronic obstructive pulmonary disease, Spirometric parameters

[1, 2] Assistant professor, Physiology Department, Government Medical College, Bhavnagar, [3] Professor& Head, Physiology Department, GCS Medical College, Ahmedabad, [4]Professor & Head, Physiology Department, M.P. Shah Medical College, Jamnagar, Gujarat

Corresponding author mail: dranupvegad@yahoo.com

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. COPD is the fourth leading cause of death in the world[1] and further increases in its prevalence and mortality can be predicted in the coming decades[2]. Globally, COPD has emerged as the 5th leading cause of loss of 'Disability Adjusted Life Years' (DALYs) as per projection of the Global Burden of Disease Study (GBDS). The region wise projections for the developing countries including India were even worse[3]. Indeed, even the current prevalence is not well understood, although a series of studies collected by Murthy & Sastry[4] and Jindal[5] have suggested that it may average around 5% in the adult population with higher rates in smokers, males, rural areas, depending on the type of domestic fuel use and socioeconomic status. The global prevalence of physiologically defined chronic obstructive pulmonary disease in adults aged ≥40 yr is approximately 9-10 per cent[6]. The Burden of Obstructive Lung Disease (BOLD) study from 12 sites involving 9425 subjects who had completed post bronchodilator spirometry testing found that the overall prevalence of COPD of GOLD (Global Initiative for Chronic Obstructive Lung Diseases) stage II or higher was 10.1 per cent and the prevalence was 11.8 per cent for men and 8.5 per cent for women[7]. COPD has been defined by GOLD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases."[8] Airflow limitations is the slowing of expiratory airflow as measured by spirometry, with a persistently low forced expiratory volume in 1 second (FEV1) and a low FEV1/forced vital capacity (FVC) ratio despite treatment. The GOLD definition for airflow limitation is an FEV1/FVC ratio of less than 70% [8,9].COPD includes Emphysema and Chronic bronchitis. The development of COPD is multifactorial and the risk factors of COPD include genetic and environmental factors. Pathological changes in COPD are observed in central airways, small airways and alveolar space. The proposed pathogenesis of COPD includes proteinase-antiproteinase hypothesis,

immunological mechanisms, oxidant-antioxidant balance, systemic inflammation, apoptosis and ineffective repair[10]. Tobacco smoking is single most important avoidable risk factor for COPD and smoking is associated with respiratory mortality and morbidity. Although smoking is the best-studied COPD risk factor, it is not the only one and there is consistent evidence from epidemiologic studies that nonsmokers may develop chronic airflow obstruction[11, 12]. Apart from tobacco smoke, occupational dusts and chemicals (vapors, irritants, and fumes) are also known causes of COPD[13, 14]. COPD is a polygenic disease and a classic example of gene-environment interaction. The genetic risk factor that is best documented is a severe hereditary deficiency of alpha-1 antitrypsin 4, a major circulating inhibitor of serine proteases[15]. In recent years numbers of physiological tests have been developed for the quantitative as well as qualitative evaluation of pulmonary function in patients with various lung diseases. Now-a-days pulmonary function tests are not restricted to research laboratory only but is also utilized widely in day to day clinical practice. Spirometry is simple, portable, noninvasive and less costly mean to assess the lung functions. It has a role in the early diagnosis and assessment of severity of lung dysfunctions in the patients with COPD.

BODE index (Body-mass index, Obstruction, Dyspnea, and Exercise capacity index) has become widely accepted as a staging tool to predict prognosis of COPD patients. BODE index is a strong predictor for mortality among COPD patients. The current study is done to compare and to assess the severity of lung dysfunctions in the patients with COPD & to study the effects of various factors like BMI, BSA, smoking, residence etc. on lung dysfunctions in COPD patients in our locality of rural & urban areas of Jamnagar district, Gujarat. **Aims & Objectives:** (1) To measure spirometric parameters in COPD patients & compare these values with their predicted values (2) To evaluate respiratory abnormalities in smoker and nonsmoker COPD patients by spirometry (3) To grade the severity of disease (4) To see the correlation of body mass index (BMI) & body surface area (BSA) with respiratory abnormalities in COPD patients.

Materials & Method: After taking permission from institutional ethical committee this observational clinical case study of 50 cases with COPD was carried out at Physiology department & T.B Chest department of Guru Gobindsinh Hospital, Jamnagar, Gujarat on outdoor or indoor basis with an aim to evaluate pulmonary functions by spirometry.

Inclusion Criteria: We studied 50 patients who had COPD diagnosed in accordance with American Thoracic Society Consensus Statement[16], which defines this illness as a disorder characterized by decreased maximum expiratory flow and slow forced emptying of the lungs that is slowly progressive, irreversible, and does not change markedly over several months. All patients were in a stable phase of their disease with optimal drug management (i.e. bronchodilator therapy and oxygen therapy, if necessary). The criteria for entry in to study were FEV_1<80% of predicted. Other inclusion criteria of these patients were a reported difficulty in performing at least one of the following activities as result of dyspnea: walking a city block, shopping, doing house hold works, lifting objects, walking upstairs and getting up out of bed.

Exclusion Criteria: Patients with ischemic heart disease, severe or uncontrolled systemic arterial hypertension, alterations in the thoracic cage, neuromuscular disorders, intermittent claudication or osteoarticular lesions in the lower extremity that could affect normal ambulation, receiving glucocortocoids, asthma were excluded. Patients suffering acute exacerbation in the course of the program were excluded.

50 patients of COPD according to criteria were selected. The height was measured in Cms with the subject standing barefoot with his back flat against a wall and the weight was noted in Kilograms on a single common scale throughout the study. Body Surface Area (BSA) was also calculated for each patient by deriving point of intersection with joining height and weight line with middle scale line of body surface (Nomogram by Bentley Laboratories). Detail clinical history was taken & general and systemic examination of each patient was done.

Procedure: The purpose and technique of spirometric measurements were explained to the subject with practical demonstration and

written informed consent was taken from all the patients. Results were taken with their willingness to undergo present investigation. Demonstration was given on how to carry out forced expiratory vital capacity maneuver with mouth piece of instrument firmly in mouth between lips so as to avoid air leakage and with a nose clip applied. All the tests were recorded in sitting and relaxed position in chair with no any tight clothing which substantially restricts full chest and abdominal expansion. In present study tabletop spirometer Spiro-Analyzer St-90 (Futuremed) was used for measuring FEV_1, FVC, FEV_1/FVC%, PEFR, MVV, and $FEF_{25-75.}$ Spirometry was performed that required three similar tracings, with the longest used for calculations. Prior to the use of this test in our study, we had studied its reproducibility, variability in a group of patients with COPD. We also recorded heart rate, blood pressure, respiratory rate before the test.

Statistical analysis: All the data were presented as mean ± S.D. and statastical analysis was done by unpaired t test - Mann-whitney test using graphpad Instat software. Also correlation coefficient (r) was calculated by applying linear (pearson) correlation test by using graphpad Instat software.

Results:

In present study there was 50% of the patients are in age group 51-60 years of age and 4% in the age group of above 60 years. The range is 27-70 years in present study with mean age being 47.5±10.8 years. In present study 68% of patients are male and 32% are female with male/female ratio of 2.13:1. 64% patients were from urban area and 92% were smokers in study group. Majority of patients were in old age and had low BMI and Law BSA. Main presenting complains were cough with expectoration and dyspnoea.

Table 1: Anthropological & other parameters of COPD patients

Parameters	Mean ± S.D.
Height (Cm)	161.24 ± 11.01
Weight (Kg)	54.50 ±11.40
BSA (m^2)	1.56 ± 0.19
BMI (Kg/m^2)	20.90 ± 3.35
Heart Rate (Per Minute)	100.06 ± 11.87
Systolic Blood Pressure (mmhg)	132.28 ± 18.62
Distolic Blood Pressure (mmhg)	81.12 ± 6.34
Respiratory Rate (Per Minute)	18.80 ± 2.50

Table 2: History and clinical features of COPD patients

History and clinical features	**No.**	**Percentage (%)**
Rural resident	18	36
Urban resident	32	64
Non smokers	4	8
Smokers	46	92
Seasonal variation of COPD exacerbations (in winter)	26	52
Patients not having seasonal variation	24	48
Cough with expectoration	47	94
Dyspnea	40	80
Fever	15	30
Edema	5	10
Cyanosis	5	10
Chest pain	3	6
Hemoptysis	3	6

Table 3: Computerised spirometric parameters in COPD patients

Parameter	Observed value (Mean ± SD) (n=50)	Predicted value (Mean ± SD) (n=50)	Percentage of Predicted (Mean ± SD) (n=50)	P value
FEV_1 (L)	1.07 ± 0.32	2.20 ± 0.46	46.76 ± 10.95	< 0.0001
FVC (L)	1.95 ± 0.50	2.65 ± 0.71	74.08 ± 14.75	< 0.0001
FEV1/FVC%	53.42 ± 11.01	85.92 ±20.12	65.50 ± 18.13	< 0.0001
PEFR (L/Min)	3.01 ± 2.25	4.42 ± 2.89	64.92 ± 19.85	0.0032
MVV (L/Min)	42.30 ± 24.67	57.09 ± 19.48	68.50 ± 22.68	0.0013
FEF_{25-75} (L/Sec)	1.55 ±0.44	1.98 ± 0.38	77.02 ± 14.92	< 0.0001

(P value < 0.05 is statisticaly significant)

Table 4: Computerised spirometric parameters in smokers and non-smokers

Parameter	Smokers (n=46)	Non-smokers (n=4)
FEV1 (L)	0.97 ± 0.4	1.26 ± 0.37
FEV1 (L) (Pred%)	43.16 ± 16.7	54 ± 9.89
FVC (L)	1.76 ± 0.72	2.09 ± 0.60
FVC (L) (Pred%)	70.13 ± 16.79	80 ± 9.93
FEV1/FVC %	48.76 ± 13.66	58.25 ± 14.68
FEV1/FVC % (Pred%)	55.35 ± 21.15	68.34 ± 16.74

In present study FEV1/FVC ratio, FEV1% and FVC all are significantly decreased in smokers than in non-smokers suggesting smokers have more severe COPD than non-smokers, statistically also this is quite significant having a value of $p < 0.05$.

Discussion: In our study when we compared values of spirometric parameters with their predicted values given by instrument we found highly significant statistical difference between observed values & their predicted values (Table-3). All values are greatly decreased in COPD patients. My study suggest that mean of FVC is 1.95 ± 0.50 L/Sec in COPD patients which is very low as compared to mean of FVC value in a study by J M Joshi et.al.[17] **(**2.5 ± 0.78 L/Sec). The forced expiration causes higher than normal transpulmonary pressure so that bronchiolar

Collapse, obstructive lesions and air trapping are all exaggerated. Similarly, mean of FEV_1 is 1.07 ± 0.32 in present study. This result is quite comparable with other study result of J M Joshi et.al. where this value is 1.53 ± 0.6(0.46-1.89) L/Sec. My results show that the mean value of FEV1% 46.76 ± 10.95%, which means it, is < 80%. This observation is comparable with work of some other studies[18, 19, 20].

Mean of FEV_1/FVC% is 53.42 ± 11.01 in present study and the similar finding (62 ± 10.04) was found in the study by J M Joshi et.al. , which is quite comparable. A normal individual can expire 50% to 60% of the FVC in 0.5 second, 75% to 85% in 1 second, 94% in 2 seconds, and 97% in 3 seconds. In obstructive pattern, a reduction in FEV_1/FVC%, a hallmark of obstructive pattern which is always accompanied by decrease in FEV_1 and there may or may not be decrease in FVC. In my study mean of FEV_1/FVC% is 53 ± 14, which is quite comparable with data of Kamat, S.R.Sharma, B.S.Rajiv, V.R.K. et.al.[21] 81.06% in normal and in COPD patients it is 55.6 ± 15.9 , i.e. there is marked reduction in FEV_1/FVC% with decrease in FEV_1 and also reduction in FVC. The first component of PFT report is spirometry, which mainly provides a measure of flow. This defines whether or not person is obstructed. We define obstruction as an FEV_1/FVC ratio of less than the 80% of the values obtained for normal. This value varies according to age and height of the patient but generally it is between 68-77%. In patients with a low FEV_1/FVC ratio, we define severity of obstruction as the absolute value of the FEV_1, the lower the EFV_1 (as a percent of predicted), the worse the obstruction. Thus the classification of obstruction suggested

by ATS should be followed. In asthma, there is no reduction in lung volumes and there is no impairment to gas transport from alveoli to the blood. Therefore the PFTs in asthma show a low FEV_1/FVC ratio, normal or even increased. The obstruction will typically improve with a bronchodilator challenge. On the contrary in emphysema, not only there is obstruction, but also there is hyperinflation and air trapping with impairment in gas transport from alveoli to the blood. Therefore, the PFTs in emphysema show a low FEV_1/FVC ratio, an elevated TLC. The obstruction is not reversible in pure emphysema; however, most patients with COPD have some bronchial hyper-reactivity as a component of their overall disease.

The mean value of PEFR is markedly reduced in COPD patients in present study (3.01 ± 2.25 L/Sec). Some other studies[18, 19, 20] also concluded that there is decrease in PEFR in obstructive lung diseases. Decreased PEFR is a hallmark of obstructive pulmonary disease and is a highly sensitive index. The peak flow mainly reflects the caliber of the bronchi and larger bronchioles which are subject to reflex bronchoconstriction due to airway obstruction; airway resistance is increased leading to decrease PEFR. Subjects with obstructive disease may develop an initially high flow rate before airway closing occurs but afterwards it may decrease.

Finally from the above data, the mean value of MVV is 42.3 ± 24.67 L/Min. MVV measures the status of the respiratory muscles, the compliance of lung-thorax system and the resistance offered by the airways and tissues. MVV is decreased greatly in subjects with moderate to severe obstructive disease. The maneuver exaggerates air trapping and exertion of the respiratory muscles.

In present study FEV1/FVC ratio, FEV1% and FVC all are significantly decreased in smokers than in non-smokers suggesting smokers have more severe COPD than non-smokers (Table 4). A study done by Chhabra et.al.[22] also shows the similar findings.

The findings of this study are quite relative and are comparable and indicate the nature of pulmonary ailment i.e. obstructive lung disorders and disorders reducing the actual lung capacity due to affection of lung tissue (i.e. emphysema). Besides these results access the severity of the disease and

currently suggested the severity classification by ATS has a key role in comprehensive management of COPD.

Table 5: FEV1% results to assess the severity of COPD as per criteria suggested by ATS[23]

Stage	Present study (by PFTs)	Present study (by symptoms & signs)
Grade 0	0	
Grade 1	0	
Grade 2	23	Mild: 20
Grade 3	23	Moderate: 32
Grade 4	4	Severe: 8
Total	50	

Table 6: Correlation of BMI and BSA with spirometric parameters (Correlation Coefficients=r)

Spirometric Parameters	**BMI (Kg/m^2)**	**BSA (m^2)**
FEV_1 (L)	0.112 (r)	0.034 (r)
FVC (L)	-0.023 (r)	-0.014 (r)
FEV1/FVC%	0.165 (r)	0.048 (r)
PEFR (L/Min)	0.232 (r)	-0.087 (r)
MVV (L/Min)	0.017 (r)	0.167 (r)
FEF_{25-75} (L/Sec)	0.145 (r)	0.094 (r)

Majority of patients were in severity grade 2 (46%) and grade 3 (46%) as suggested by American Thoracic Society.

In our study we found positive correlation between values of FEV_1, FEV1/FVC%, PEFR, MVV & FEF_{25-75} with BMI (Table 6). Previous studies shows that in low BMI patients efficiency of lungs is more compromised and various spirometric parametrs are low and BMI decreases further with increase in severity of COPD.[24,25] Low BMI could be a consequence of COPD. Basal metabolic rate is increased in moderate to severe COPD.[26] Decramer et al have proposed that the pathogenesis of nutritional depletion in COPD is high energy expenditure and low energy intake.[27] Importantly, low BMI is one of the independent predictors for mortality in patients with COPD.[28] Clinic-based studies suggest that low BMI is an independent prognostic factor of poor long-term survival following diagnosis of

COPD[29,30]. Cachexia is common in long term disease, so by maintaining proper nutrition we can increase the respiratory efficiency and reduce the severity of the disease. We also found positive correlation between values of FEV_1, FEV1/FVC%, MVV & FEF_{25-75} with BSA (Table 6). Here 70% of patients had body surface area (BSA) < 1.5 $sq.m^2$ and 30% had BSA > 1.5 $sq.m^2$, so with lower BSA patient develop poor effort of breathing and so severity of COPD increases with increase in BSA[24].

Conclusion: In patients of COPD there was great statistically significant reduction in values of FVC, FEV_1, PEFR, FEV_1/FVC%, FEF_{25-75} and MVV mean values which were suggestive of an increase airway resistance. Majority of patients were male (68%) and of old age (51-60 yrs). Disease was more prevalent in urban area suggesting air pollution is a risk factor in COPD and in smokers. Spirometric results suggested severe COPD in smoker group than non-smoker confirming smoking was an important avoidable risk factor. Majority of patents were with lower BMI and BSA. Severity of COPD increases with decrease in BMI and BSA. So by maintaining proper nutrition we can reduce the severity of the disease. History of patients suggests that exacerbation of COPD were more frequent in winter months which are frequently associated with upper respiratory tract infection. So COPD patients have to take special precautions during winter months to avoid exacerbation of disease.

References:

1. World Health Report. Geneva: World Health Organization. Available from URL: http://www.who.int/whr/2000/en/statistics.htm; 2000.
2. Lopez AD, Shibuya K, Rao C, Mathers CD, Hansell AL, Held LS, et al. Chronic obstructive pulmonary disease: current burden and future projections. Eur Respir J 2006;27(2):397-412.
3. Murray CJL, Lopez AD. Alternative projection of mortality and disability by cause 1990-

2020: Global Burden of Disease Study. Lancet 1997;349:1498-504.

4. Murthy KJR, Sastry JG. Economic burden of chronic obstructive pulmonary disease. National Commission on Macroeconomics and Health Background Papers – Burden of Disease in India. National Commission on Macroeconomics and Health, Government of India, 2005. Available from: http://www.who.int/macrohealth/action/NCMH_Burden%20of%20disease_%2829%20Sep%202005%29.pdf
5. Jindal SK. Emergence of chronic obstructive pulmonary disease as an epidemic in India. *Indian J Med Res* 2006;124:619-30.
6. Halbert R11. J, Natoli JL, Gano A, Badamgarav E, Buist AS, Mannino DM. Global burden of COPD: systematic review and meta-analysis. *Eur Respir J* 2006; *28* : 523-32.
7. Buist A12. S, McBurnie MA, Vollmer WM, Gillespie S, Burney P, Mannino DM, *et al*. BOLD Collaborative Research Group. International variation in the prevalence of COPD (the BOLD Study): a population-based prevalence study. *Lancet* 2007; *370* : 741-50.
8. Pauwels RA, Buist AS, Calverley PM, et al. Global Strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. Am J Respir Crit Care Med 2001;163:1256–76.
9. World Health Organization. The GOLD global strategy for the management and prevention of COPD. Available at http://www.goldcopd.com. Accessed 13 August 2001.
10. V.K. Vijayan, Chronic obstructive pulmonary disease. Indian J Med Res 137, February: 2013, pp 251-269
11. Celli BR, Halbert RJ, Nordyke RJ, Schan B. Airway obstruction in never smokers: results from the Third National Health and Nutrition

Examination Survey. Am J Med 2005;118:1364-72.

12. Behrendt CE. Mild and moderate-to-severe COPD in nonsmokers. Distinct demographic profiles.Chest 2005;128:1239-44.
13. Becklake MR. Occupational exposures: evidence for a causal association with chronic Obstructive pulmonary disease. Am Rev Respir Dis 1989;140(3 Pt 2):S85-91.
14. Trupin L, Earnest G, San Pedro M, Balmes JR, Eisner MD, Yelin E, et al. The occupational burden of chronic obstructive pulmonary disease. Eur Respir J 2003;22(3):462-9.
15. Stoller JK, Aboussouan LS. Alpha1-antitrypsin deficiency. Lancet 2005;365(9478):2225-36.
16. Celli BR, Claudia G Cote et.al. The BMI, airflow obstruction, Dyspnea and Exercise capacity Index in
21. Kamat S R, Sharma B S, Rajiv et.al. Indian Norms of Pulmonary function: Observed values, Prediction equations and Intercorelations. JAPI 1997:531-540.

COPD. N Engl J Med 2004; 350: 1005-12.

17. J M Joshi, Spirometry: All you need to know. Medicine Update 2005:98, 493-496.
18. Deborah Leader, RN. Obstructive and Restrictive Lung Diseases: Comparing Differences Between Obstructive and Restrictive Lung Diseases. About . com guide 2012.
19. Joshil Kumar Behera, Sushma Sood, Naresh Kumar, Kirti Sharma, Reshmi Mishra and Prasanta Saha Roy. Heart Rate Variability and its Correlation with Pulmonary Function Test of Smokers. Heart Views 2013 Jan-Mar; 14(1): 22–25.
20. Rubeena bano, Nadeem ahmad, Mahagaonkar. Study of lung functions in smokers and non-smokers.Indian j physiol pharmacol 2011; 55 (1) : 84–88
22. Chhabra S K, S Rajpal, R Gupta et.al; Pattern of smoking in Delhi and Comparison of chronic respiratory morbidity among

smokers. Indian J. Chest. Dis. Alied Sci.2001:43, 19-26.

23. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. American Thoracic Society. [Review]. Am J Respir Crit Care Med 1995;152(5 Pt 2):S77–121
24. E Ogawa,Y Nakano, T Ohara, S Muro, T Hirai, S Sato et al. Body mass index in male patients with COPD: correlation with low attenuation areas on CT, Thorax 2009;64:20–25.
25. Sajal De, Body mass index among patient with chronic obstructive pulmonary diseases, Indian J Physiol Pharmacol 2012; 56(4)
26. Hugli O, Schutz Y, Fitting JW. The daily energy expenditure in stable chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1996;153:294–300.
27. Decramer M, De Benedetto F, Del Ponte A, et al. Systemic effects of COPD. Respir Med 2005;99:S3–10.
28. Celli BR, Cote CG, Marin JM, et al. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. N Engl J Med 2004;350:1005–12.
29. Schols AM, Slangen J, Volovics L, Wouters EF. Weight loss is a reversible factor in the prognosis of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 1998;157:1791–97.
30. Ringbaek TJ, Viskum K, Lange P. BMI and oral glucocorticoids as predictors of prognosis in COPD patients on long-term oxygen therapy. Chron Respir Dis 2004;1: 71–78.

Abbreviations: Chronic Obstructive Pulmonary Disease (COPD), BMI (Body mass index), BSA (Body surface area), FVC (forced vital capacity), FEV1 (forced expiratory volume in 1 second), FEF25-75 (Mid expiratory flow rate), PEFR (Peak expiratory flow rate) and MVV (Maximum voluntary ventilation), ATS (American thoracic society)