Spirometry and Flow Volume Loops in Obstructive Sleep Apnea Patients

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ABSTRACT

Introduction: Obstructive sleep apnea syndrome is the most common sleep disordered breathing (SDB) and is associated with recurrent episodes of upper airway (UA) collapse during sleep. Arousal from sleep is required to re-establish the airway patency. Patients with OSAS have various structural and functional abnormalities of the upper airway during sleep, which may reflect on their pulmonary function tests.

Material and methods: We performed spirometry in 50 diagnosed OSAS patients. Spirometric indices like FEF50/FIF50 >1 and saw tooth appearance of FV loops were studied. Their association with OSA and grades of OSA was evaluated using statistical analysis.

Results: It was found that these spirometric indices were common in OSAS patients but their association with grades of OSA were not statistically significant.

Conclusion: Spirometry findings can point toward diagnosis of OSA but these findings are absent in most OSA patients.

Keywords: OSAS, Spirometry, Sleep, FV Loops

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS), also referred to as obstructive sleep apnea hypopnea syndrome (OSAHS) is a common sleep disorder that involves cessation or significant decrease in the airflow in the presence of breathing effort.[1] Approximately 4% men and 2% women suffer from obstructive sleep apnea syndrome. It is the most common sleep disordered breathing (SDB) and is associated with recurrent episodes of upper airway (UA) collapse during sleep. Arousal from sleep is required to re-establish the airway patency.[2] This distortion of sleep pattern and repetitive awakening contributes to excessive daytime sleepiness and other functional impairments like impaired cognition, memory and mood alterations. The nocturnal hypoxia may also lead to pulmonary and systemic hypertension as well as other cardiac and metabolic complications.[3]

Patients with OSAS have various structural and functional abnormalities[4-6] of the upper airway during sleep, which may reflect on their pulmonary function tests. Spirometry and flow-volume loop are simple and commonly used tests in patients with respiratory symptoms. Abnormalities in the flow volume curves and other spirometric indices have been reported to be present and related to OSAS. These abnormalities include (i) saw-tooth sign which consists of regular oscillations occurring on constant intervals at forced expiratory and inspiratory flow volume curves[17], (ii) increased the ratio of forced expiratory flow to forced inspiratory flow at 50% of vital capacity (FEF_{50}/FIF_{50}) >1. (iii) Restriction on spirometry. Restriction (decrease in forced vital capacity) is mostly due to obesity.

Current research aimed to study abnormalities in spirometric indices and flow volume loops and its relation with apnoea hypopnoea index in patients diagnosed with obstructive sleep apnoea syndrome.

MATERIAL AND METHODS

Patients attending Pulmonary medicine OPD / in-patients of Lilavati Hospital and Research centre diagnosed as Obstructive sleep apnoea (Apnoea hypopnoea index > 5 episodes/hour), were included in study after written informed consent. Patients with cardiac comorbidities (congenital heart diseases, valvular heart diseases, etc.) were excluded.

Patients were graded as mild, moderate and severe obstructive sleep apnea as per American academy of sleep medicine.

Patients were interviewed in detail and were clinically examined. Age, sex, height, weight, Body Mass Index (BMI) and neck circumference were noted. Presence of cardiovascular risk factors (systemic hypertension, dyslipidaemia, type 2 diabetes mellitus, coronary artery disease, obesity and smoking status) was noted.

Spirometry was analysed for flow volume curve abnormalities, extrathoracic airway obstruction (ratio of expiratory flow to inspiratory flow at 50 percent of forced vital capacity [FEF_{50}/FIF_{50}]) and spirometric indices. Spirometry was analysed for obstruction or restriction.

Appropriate statistical tests were applied and results were analysed. P value less than 0.05 is taken as significant level.

RESULTS

Our study included 50 patients diagnosed with obstructive sleep apnea syndrome by polysomnography. Based on apnea

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Severity of OSAS	Frequency	Percentage
Mild OSAS	19	38.0
Moderate OSAS	12	24.0
Severe OSAS	19	38.0
Total	50	100.0

Table-1: Sex distribution in the study and distribution of study group as per BMI

hypopnea index, patients were graded as mild, moderate and severe obstructive sleep apnea syndrome.

Out of the 50 patients included in our study, 19 patients (38%) were of mild OSAS, 12 patients (24%) were of moderate OSAS and 19 patients (38%) were of severe OSAS. This was not in accordance to study of Young et al who investigated the association of sex, age, race, snoring, and obesity with

Sex		Severity of OSAS			Total
		Mild	Moderate	Severe	
Male	Count	8	7	14	29
	Percentage	42.1%	58.3%	73.7%	58.0%
Female	Count	11	5	5	21
	Percentage	57.9%	41.7%	26.3%	42.0%
Total	Count	19	12	19	50
	Percentage	100.0%	100.0%	100.0%	100.0%

Table-2: Sex distribution in the study

BMI (kg/m^2)		Severity of OSAS			Total
		Mild	Moderate	Severe	
<25	Count	2	0	0	2
	Percentage	10.5%	0.0%	0.0%	4.0%
25 to 30	Count	2	3	4	9
	Percentage	10.5%	25.0%	21.1%	18.0%
Above 30	Count	15	9	15	39
	Percentage	78.9%	75.0%	78.9%	78.0%
Total	Count	19	12	19	50
	Percentage	100.0%	100.0%	100.0%	100.0%
Chi-Square test		**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square		4.296	4	0.367	Not significant

Table-3: Distribution of study group as per BMI

FEF_{50}/FIF_{50}		Severity of OSAS			Total
		Mild	Moderate	Severe	
<1	Count	15	9	14	38
	Percentage	78.9%	75.0%	73.7%	76.0%
>1	Count	4	3	5	12
	Percentage	21.1%	25.0%	26.3%	24.0%
Total	Count	19	12	19	50
	Percentage	100.0%	100.0%	100.0%	100.0%
Chi-Square test		**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square		0.153	2	0.926	Not significant

Table-4: Association of FEF50/FIF50 with grades of severity of OSAS

BMI (in kg/m^2)		FEF_{50}/FIF_{50}		Total
		<1	>1	
<25	Count	2	0	2
	Percentage	100.0%	0.0%	100.0%
25 to 30	Count	9	0	9
	Percentage	100.0%	0.0%	100.0%
Above 30	Count	27	12	39
	Percentage	69.2%	30.8%	100.0%
Total	Count	38	12	50
	Percentage	76.0%	24.0%	100.0%
Chi-Square test	**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square	4.453	2	0.108	Not sig

Table-5: Association of FEF50/FIF50 with BMI

Sawtooth appearance		Severity of OSAS			Total
		Mild	**Moderate**	**Severe**	
Yes	Count	3	2	5	10
	Percentage	15.8%	16.7%	26.3%	20.0%
No	Count	16	10	14	40
	Percentage	84.2%	83.3%	73.7%	80.0%
Total	Count	19	12	19	50
	Percentage	100.0%	100.0%	100.0%	100.0%
Chi-Square test		**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square		0.768	2	0.681	Not significant

Table-6: Association of sawtooth appearance on flow volume loops with grades of severity of OSAS

BMI		sawtooth		Total
		Yes	**No**	
<25	Count	0	2	2
	Percentage	0.0%	100.0%	100.0%
25 to 30	Count	2	7	9
	Percentage	22.2%	77.8%	100.0%
Above 30	Count	8	31	39
	Percentage	20.5%	79.5%	100.0%
Total	Count	10	40	50
	Percentage	20.0%	80.0%	100.0%
Chi-Square test	**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square	0.534	2	0.766	Not sig

Table-7: Association of sawtooth appearance of flow volume loop with BMI

Spirometry			Severity of OSA			Total
			Mild	**Moderate**	**Severe**	
Normal		Count	6	7	7	20
		Percentage	12.0%	14.0%	14.0%	40.0%
Restriction	mild	Count	2	0	2	4
		Percentage	4.0%	0.0%	4.0%	8.0%
	moderate	Count	4	2	0	6
		Percentage	8.0%	4.0%	0.0%	12.0%
	severe	Count	6	2	6	14
		Percentage	12.0%	4.0%	12.0%	28.0%
	total	Count	12	4	8	24
		Percentage	24.0%	8.0%	16.0%	48.0%
Obstruction + restriction		Count	1	1	1	3
		Percentage	2.0%	2.0%	2.0%	6.0%
Obstruction		Count	0	0	3	3
		Percentage	0.0%	0.0%	6.0%	6.0%
Chi-Square test			**Value**	**df**	**P Value**	**Association is**
Pearson Chi-Square			8.0154	6	0.236	Not significant

Table-8: Spirometric findings in OSAS patients

SDB in community-dwelling adults, using data from 6119 participants in the Sleep Heart Health Study (SHHS), a multicentre cohort study of SDB and cardiovascular disease. They found prevalence of mild, moderate and severe disease among the patients of SDB as 53%, 29% and 18%, respectively.

The higher prevalence of severe disease in this study is arguably due to the hospital based model of the study and the fact that with the presently prevalent knowledge and awareness of SDB among patients and referring physicians, a large number of cases who are at a risk of severe OSAS are subjected to polysomnography and detected. Also patients surface only when the disease is severe. In our study, 29 (58%) were males as compared to 21 (42%) females.

In our study, 4% (2 out of 50) patients had normal BMI (BMI < 25 kg/m^2), 18% (9 out of 50) patients were overweight (BMI 25-30 kg/m^2) and 78% (39 out of 50) patients were obese (BMI > 30 kg/m^2). The prevalence of obstructive sleep apnea syndrome is more in obese patients. The association of grades of severity of OSAS and BMI was not statistically significant (table-2,3).

Association of FEF$_{50}$/FIF$_{50}$ with grades of severity of OSAS

It was observed that only 24% (12 patients) have FEF$_{50}$/FIF$_{50}$

ratio more than 1. 21.1% (4 out of 19) patients with mild OSAS, 25% (3 out of 12) with moderate OSAS and 26.3% (5 out of 19) patients with severe OSAS ratio less than 1. The association of grades of severity of OSAS and $FEF_{50}/FIF_{50} >$ 1 was not statistically significant (table-4).

In our study all patients (24%) with $FEF_{50}/FIF_{50} > 1$ had BMI > 30kg/m^2. However association of BMI and $FEF_{50}/FIF_{50} > 1$ was not statistically significant. Similarly severe obstructive sleep apnea syndrome patients had more prevalence of sawtooth appearance on flow volume loop (table-5).

Association of sawtooth appearance on flow volume loops with grades of severity of OSAS and BMI

In our study, 20% (10 out of 50) patients had sawtooth appearance on flow volume loop of spirometry. 15.8% (3 out of 19) patients with mild OSAS, 16.7% (2 out of 12) patients with moderate OSAS and 26.3 (5 out of 19) patients with severe OSAS had sawtooth appearance of flow volume loop on spirometry (table-6,7).

Spirometric findings in OSAS patients

In our study 40% patients had normal spirometry, 48% had restriction, 6% had obstruction plus restriction and 6% has obstruction on spirometry (table-8).

DISCUSSION

Study showed increased prevalence of disease amongst males. This was in accordance with the results of other studies.[7-9] The reasons for gender differences in the prevalence and severity of sleep apnea are multifactorial. Some of the most commonly proposed hypothesis include differences in the effect of weight, differences in body fat distribution, abnormalities in upper airway mechanics, control of breathing, and structural differences in upper airway dimensions. In the study conducted by Sharma and colleagues[7], male gender was associated with a 10-fold higher risk of having OSAS.

The association between obesity and OSA has long been appreciated. There is graded increase in OSA prevalence with increasing BMI as shown by cross sectional studies[10-14] and population based studies.[15-17] All most all have found significant association between OSA and measures of excess body weight.

Haponik et al.[4] studied the FEF50%/FIF50% and the PIF in two groups of patients, 27 with OSA and 25 without OSA, and observed that 44% patients with OSA had FEF50%/FIF50% >1.0 and only 8% patients without OSA had ratio less than 1.

Hoffstein et al.[18] analyzed 405 patients showed, that the FEF_{50}/FIF_{50} ratio did not differ between groups. Rauscher et al.[19] showed that the FEF_{50}/FIF_{50} ratio and sawtooth sign are of limited value for predicting OSA and Katz et al.[20] reported similar results.

Ashraf et al.[21] found no significant difference between BMI and FEF_{50}/FIF_{50} ratio and sawtooth appearance in OSAS and non OSAS patients.

Conceptually, the UA is a compliant tube and, therefore, is subject to collapse.[22] OSA is caused by soft tissue collapse in the pharynx.

Trans-mural pressure is the difference between intraluminal pressure and the surrounding tissue pressure. If trans-mural pressure decreases, the cross-sectional area of the pharynx decreases. If this pressure passes a critical point, pharyngeal closing pressure is reached. Exceeding pharyngeal critical pressure (Pcrit) causes a juggernaut of tissues collapsing inward. The airway is obstructed. Until forces change trans-mural pressure to a net tissue force that is less than Pcrit, the airway remains obstructed. OSA duration is equal to the time that Pcrit is exceeded.

Most patients with OSA demonstrate upper airway obstruction at either the level of the soft palate (nasopharynx) or the level of the tongue (oropharynx).

Anatomic factors (e.g., enlarged tonsils; volume of the tongue, soft tissue, or lateral pharyngeal walls); length of the soft palate; abnormal positioning of the maxilla and mandible) may each contribute to a decrease in the cross-sectional area of the upper airway and/or increase the pressure surrounding the airway, both of which predispose the airway to collapse.[23,24]

Neuromuscular activity in the UA, including reflex activity, decreases with sleep, and this decrease may be more pronounced in patients with OSA.[25] Reduced ventilatory motor output to upper airway muscles is believed to be the critical initiating event leading to UA obstruction; this effect is most pronounced in patients with a UA predisposed to collapse for anatomical reasons.

Central breathing instability is a well-established factor contributing to the development of CSA, particularly in patients with severe congestive heart failure (CHF).[26] Evidence also indicates that central breathing instability contributes to the development of OSAS.

Both static factors and dynamic factors are involved in the development of OSA. Static factors include surface adhesive forces, neck and jaw posture, tracheal tug, and gravity. Any anatomic feature that decreases the size of the pharynx (e.g., retrognathia) increases the likelihood of OSA. Gravitational forces are felt simply by tilting one’s head back to where the retro-position of the tongue and soft palate reduce the pharyngeal space. For most patients, OSA worsens in the supine sleeping position.

Dynamic factors include nasal and pharyngeal airway resistance, the Bernoulli’s principle and dynamic adherence. The Bernoulli’s effect plays an important dynamic role in OSA pathophysiology. In accordance with this effect, airflow velocity increases at the site of stricture in the airway. As airway velocity increases, pressure on the lateral wall decreases. If the trans-mural closing pressure is reached, the airway collapses. The Bernoulli’s effect is exaggerated in areas where the airway is most compliant. Loads on the pharyngeal walls increase adherence and, hence, increase the likelihood of collapse.

Given these principles, it is understandable why the likelihood of OSA is increased among obese patients, why weight loss decreases the risk of OSA, and why physical examination helps in predicting the presence of OSA. However, the clinical situation is complex because of the interplay of known static and dynamic factors and because of unknown factors.

Studies regarding pharynx geometry in awake patients with OSA have shown that the pharyngeal diameter of these subjects is smaller than normal. Studies of the pharyngeal muscles of awake patients with OSA have demonstrated that in these patients added inspiratory resistive loads do not provoke an adequate muscular response in pharyngeal zones, where greater collapsibility is found. These results suggest that there may be an altered regulation of these structures. Other studies on the upper airway resistance (UAR) of patients with OSAS, using transducers placed in different pharyngeal zones, have shown that the UARS is higher in these patients than in normal subjects. These data suggest that the causes of OSAS may be primarily anatomic, and that some muscular regulation factor may be altered, leading to an increase in upper airway resistance even in the awake state. During sleep these alterations may be much more pronounced causing periodic occlusions of the pharynx.[27]

The flow-volume curve, mainly the inspiratory phase, has been the parameter most frequently used to look for a simple way to pinpoint patients with OSAS, since the PSG is a difficult and expensive exam. The sawtooth signal and an FEF50/FIF50 ratio >1.0 have been the most used variables, but there are controversies about their reliability. Haponik et al.[4] studied the FEF50/FIF50 and the PIF in two groups of patients, 27 with OSA and 25 without OSA, and observed that the group with OSAS had FEF50/FIF50>1.0 and lower PIF. The authors concluded that the flow-volume curve was useful for the diagnosis of OSAS. Tammelin et al.[28] studied the flow-volume curve and performed fiberoptic nasopharyngoscopy in 22 patients with OSA. They observed that, in the presence of endoscopic alterations of the upper airway, the flow-volume curve was often abnormal (saw-tooth, FEF50/FIF50 >1.0 or both).

Hoffstein et al.[18] analyzed 405 patients referred to the sleep laboratory with the major complaint of snoring. According to the PSG, the patients were divided into 207 with OSA and 198 without OSA. The authors showed that the FEF50/FIF50 ratio did not differ between groups. Rauscher et al.80 showed that the FEF50/FIF50 ratio and sawtooth sign are of limited value for predicting OSA and Katz et al.[20] reported similar results.

CONCLUSION

Spirometric indices like FEF50/FIF50 > 1 and saw tooth appearance on FV loops are common in OSAS patients than in non OSAS patients. However it appears that it is more associated with obesity and BMI than OSAS itself. The grades of severity of OSAS and these indices are not statistically associated. Also most of patients have normal spirometry or restriction.

We conclude that all patients of OSAS should be evaluated with spirometry and FEF50/FIF50 > 1 and saw tooth appearance on FV loops can point towards confirmation of OSA but normal indices and FV loops does not exclude OSA.

REFERENCES

1. Ramirez JM, Garcia AJ, 3rd, Anderson TM, Koschnitzky JE, Peng YJ, Kumar GK, et al. Central and peripheral factors contributing to obstructive sleep apneas. Respiratory physiology & neurobiology. 2013;189:344-53.
2. Guilleminault C, Tilkian A, Dement WC. The sleep apnea syndromes. Annual review of medicine. 1976;27:465-84.
3. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet. 2005;365:1046-53.
4. Haponik EF, Bleecker ER, Allen RP, Smith PL, Kaplan J. Abnormal inspiratory flow-volume curves in patients with sleep-disordered breathing. The American review of respiratory disease. 1981;124:571-4.
5. Levent E, Sariman N. Analysis of obstructive sleep apnea patients with "sawtooth sign" on the flow-volume curve. Sleep & breathing = Schlaf & Atmung. 2011;15:357-65.
6. Kosmas EN RT, Toukmatzi S, Michaelides S, Polychronopoulos V. Chest 1998; 114: 371S. Significance of the inspiratory flow-volume loop in the diagnosis of obstructive sleep apnea syndrome. Chest. 1998;114:371.
7. Sharma SK, Kumpawat S, Banga A, Goel A. Prevalence and risk factors of obstructive sleep apnea syndrome in a population of Delhi, India. Chest. 2006;130:149-56.
8. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. Jama. 2002;288:1723-7.
9. Zamarron C, Gude F, Otero Y, Alvarez JM, Golpe A, Rodriguez JR. Prevalence of sleep disordered breathing and sleep apnea in 50- to 70-year-old individuals. A survey. Respiration; international review of thoracic diseases. 1999;66:317-22.
10. Bixler EO, Vgontzas AN, Ten Have T, Tyson K, Kales A. Effects of age on sleep apnea in men: I. Prevalence and severity. American journal of respiratory and critical care medicine. 1998;157:144-8.
11. Ip MS, Lam B, Tang LC, Lauder IJ, Ip TY, Lam WK. A community study of sleep-disordered breathing in middle-aged Chinese women in Hong Kong: prevalence and gender differences. Chest. 2004;125:127-34.
12. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. American journal of respiratory and critical care medicine. 2002;165:1217-39.
13. Wong ML, Sandham A, Ang PK, Wong DC, Tan WC, Huggare J. Craniofacial morphology, head posture, and nasal respiratory resistance in obstructive sleep apnoea: an inter-ethnic comparison. European journal of orthodontics. 2005;27:91-7.
14. Shepertycky MR, Banno K, Kryger MH. Differences between men and women in the clinical presentation of patients diagnosed with obstructive sleep apnea syndrome. Sleep. 2005;28:309-14.
15. Newman AB, Nieto FJ, Guidry U, Lind BK, Redline S, Pickering TG, et al. Relation of sleep-disordered

breathing to cardiovascular disease risk factors: the Sleep Heart Health Study. American journal of epidemiology. 2001;154:50-9.

16. Ferini-Strambi L, Zucconi M, Palazzi S, Castronovo V, Oldani A, Della Marca G, et al. Snoring and nocturnal oxygen desaturations in an Italian middle-aged male population. Epidemiologic study with an ambulatory device. Chest. 1994;105:1759-64.
17. Jennum P, Hein HO, Suadicani P, Gyntelberg F. Cardiovascular risk factors in snorers. A cross-sectional study of 3,323 men aged 54 to 74 years: the Copenhagen Male Study. Chest. 1992;102:1371-6.
18. Hoffstein V, Wright S, Zamel N. Flow-volume curves in snoring patients with and without obstructive sleep apnea. The American review of respiratory disease. 1989;139:957-60.
19. Rauscher H, Popp W, Zwick H, inventorsFlow-volume curves in obstructive sleep apnea and snoring patent. Lung. 1990;168:209-14.
20. Katz I, Zamel N, Slutsky AS, Rebuck AS, Hoffstein V. An evaluation of flow-volume curves as a screening test for obstructive sleep apnea. Chest. 1990;98:337-40.
21. Ashraf M SS, BaHammam SA. Spirometry and flow-volume curve in patients with obstructive sleep apnea. Saudi Med J 2008;29:477-81.
22. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. Chest. 2007;132:325-37.
23. Schwab RJ, Pasirstein M, Pierson R, Mackley A, Hachadoorian R, Arens R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. American journal of respiratory and critical care medicine. 2003;168:522-30.
24. White DP. Sleep apnea. Proceedings of the American Thoracic Society. 2006;3:124-8.
25. White DP. The pathogenesis of obstructive sleep apnea: advances in the past 100 years. American journal of respiratory cell and molecular biology. 2006;34:1-6.
26. Leung RS, Bradley TD. Sleep apnea and cardiovascular disease. American journal of respiratory and critical care medicine. 2001 Dec 15;164:2147-65.
27. Amado VM, Costa AC, Guiot M, Viegas CA, Tavares P. Inspiratory flow-volume curve in snoring patients with and without obstructive sleep apnea. Brazilian journal of medical and biological research = Revista brasileira de pesquisas medicas e biologicas / Sociedade Brasileira de Biofisica [et al]. 1999;32:407-11.
28. Tammelin BR, Wilson AF, Borowiecki BB, Sassin JF. Flow-volume curves reflect pharyngeal airway abnormalities in sleep apnea syndrome. The American review of respiratory disease. 1983;128:712-5.

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