

# Effect of truncal obesity on airway resistance

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**Background and objective** Truncal obesity can influence respiratory mechanisms regardless of BMI. This study evaluated the impact of truncal obesity on both spirometry and impulse oscillometry (IOS).

**Patients and methods** The study included 102 patients recruited from the Outpatient Department of Ain Shams University Hospital. All patients were subjected to clinical examination, anthropometric measurements, IOS, and spirometry. According to BMI, the patients were categorized into the obese group (71 patients) and the nonobese group (31 patients). The obese group was subclassified into overweight, obese, and morbidly obese groups. The obese subgroup was reclassified according to waist to hip ratio (WHR) into the truncal obese group (41 patients) and the nontruncal obese group (30 patients). The data collected were comparatively analyzed using either the independent-samples *t*-test or one-way analysis of variance. Pearson's correlation coefficient was used to study the correlations between data. Multiple linear regression analysis was carried out to examine the simultaneous influences of anthropometric measures on the IOS data.

## Introduction

The common use of BMI as an obesity parameter is preferable for its easy calculation. However, BMI fails to provide information about the distribution of fat, which may be a more determining risk factor of morbidity [1,2]. Many studies have highlighted the effect of accumulation of abdominal fat and its effect on overall health [3]. Few community-based surveys have realized the effects of obesity on pulmonary function parameters, which are affected by the amount and distribution of body fat. Previous studies showed the impact of truncal obesity, which may be calculated by waist circumference or waist to hip ratio (WHR), on respiratory mechanisms regardless of BMI. The major respiratory impact includes an increased demand for ventilation, elevated effort of breathing, respiratory muscle inefficiency, and diminished compliance of the chest wall. The end results are hypoxemia, pulmonary arterial hypertension, and progressive increasing disability [4,5]. In addition, limitation of diaphragmatic expansion depends on waist circumference, which may affect the respiratory mechanisms [2,6]. This information becomes important for accurate interpretation of pulmonary function tests in an ever-increasing number of patients with truncal obesity. Thus, truncal obesity is most likely to affect pulmonary volumes, without direct effects on

**Results** There were significant differences in IOS and spirometry data between the obese and nonobese groups as well as among the four groups. Regarding WHR, there were significant correlations with IOS data and negative correlations with spirometry data except maximal mid expiratory flow. Using multiple linear regression analysis, it was revealed that WHR was the most powerful predictor for R5. BMI followed by WHR was the most powerful predictor for R5–R20.

**Conclusion** Truncal obesity significantly affects airway resistance, as evaluated by spirometry and IOS.

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**Keywords:** airway resistance, BMI, spirometry, truncal obesity, waist to hip ratio

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pulmonary obstruction, as was discussed in a review on the physiology of obesity and its effects on pulmonary function [7]. However, others have shown that obesity has adverse consequences on spirometry measures such as forced expiratory volume in the first second (FEV<sub>1</sub>) and forced vital capacity (FVC) [5].

The present study aimed to evaluate the impact of truncal obesity on both spirometry and impulse oscillometry (IOS) measures.

## Patients and methods

The study included 102 patients (76 male and 26 female), all of whom were nonsmokers, recruited from the Outpatient Department of the Chest Department, Ain Shams University Hospital. All patients had symptoms of acute upper respiratory infection.

Exclusion criteria included a positive history of smoking, presence of any chronic respiratory disease including generalized obstructive lung diseases, bronchial hyper-reactivity, interstitial lung diseases, pleural or chest wall diseases, right-sided or left-sided heart failure, unstable angina, recent myocardial infarction, or any other contraindication for performing a forced spirometry maneuver [8].

The study was approved by the review board of Pulmonary Medicine Department, Ain Shams University, and informed consent was obtained from all patients.

All patients underwent history taking and clinical examination, anthropometric measurements including height, weight, BMI, waist, hip and neck circumferences, and WHR, IOS, and prebronchodilator and postbronchodilator spirometry.

Patients were categorized into two groups on the basis of BMI: the obese group (BMI  $\geq 25$ ), which included 71 patients, and the nonobese group (BMI  $< 25$ ), which included 31 patients. These participants were further subclassified into the following four groups: normal (BMI = 18–24.9), which included 31 patients; overweight (BMI = 25–29.9), which included 28 patients; obese (BMI = 30–34.9), which included 19 patients; and morbidly obese (BMI  $\geq 35$ ), which included 24 patients [6].

The obese patients were reclassified according to WHR into the truncal obese group (WHR  $\geq 0.90$  in males and  $\geq 0.85$  in females), which included 41 patients, and the nontruncal obese group (WHR  $< 0.90$  in males and  $< 0.85$  in females), which included 30 patients [6].

### Spirometry

Spirometry was performed using Spirometrics ENC Flowmate (Spring Valley, New York, USA). The test was performed before and 20 min after  $\beta_2$ -agonist inhalation (salbutamol 400  $\mu\text{g}$ ) by means of a metered-dose inhaler. Prebronchodilator and postbronchodilator spirometry were measured according to American Thoracic Society/European Respiratory Society standards in all patients [8].

### Impulse oscillometry

IOS was measured using a Master Lab-IOU Unit (Master Screen IOS 2001, version 4.5; Erich Jaeger GmbH, Hoechberg, Germany). The test was carried out according to the main principles of the European Respiratory Society (ERS) Task Force recommendations [9]. As the patient breathes through a pneumotachograph, a sound wave generated by a loudspeaker is superimposed over their breathing. The patient's airflow and sound wave response is transmitted to the apparatus and used to calculate the various components of resistance to breathing. The actual values of airway resistance at 5 Hz (R5, total airway resistance), 20 Hz (R20, central airway resistance), and distal capacitive reactance at 5 Hz (X5) were recorded [10].

R5 and R20 are considered normal if they are greater than 150% of predicted values.

Resistance spectrum [ $R(f)$ ] is considered normal if it shows no frequency dependence.

X5 is considered normal if it equals X5 predicted (0.15).

Proximal obstruction is considered if the following conditions are fulfilled:

- (1) R5 is high ( $> 150\%$  predicted) (i.e. in the abnormal range).
- (2) R20 is elevated ( $> 150\%$  predicted).
- (3)  $R(f)$  is independent of frequency and almost horizontal (i.e. the R20 is similar to the R5).
- (4) X5 is completely normal, as the resonant frequency.

Peripheral obstruction is considered if:

- (1) R5 is abnormal (i.e.  $> 150\%$  predicted).
- (2) R20 is normal (i.e.  $< 150\%$  predicted).
- (3)  $R(f)$  is frequency dependent and becomes lower at higher frequencies.
- (4) X5 is reduced to the abnormal range, and the resonant frequency ( $f_{\text{res}}$ ) is shifted to the right (i.e. to higher frequencies) [11].

### Data analysis

Data were compared using analysis of variance, followed by post-hoc multiple comparisons by the Bonferroni method.

Simple correlations between data were tested using Pearson's correlation coefficient.

Multiple linear regression test was performed to examine the concurrent effects of anthropometric measures including calculated BMI, WHR, and measured neck circumference, with the IOS data R5, R20, R5–R20, and X5 as dependent variables.

The statistical software statistical package for the social sciences (SPSS, version 17; SPSS Inc., Chicago, Illinois, USA) was used for statistical analysis. All tests were considered significant at  $P$  value less than 0.05.

### Results

The study included 102 patients [76 (74.5%) males and 26 (25.5%) females]. Their mean age was  $45.88 \pm 11.54$  years. The patient characteristics of age, anthropometric measures, IOS values, and spirometry data are presented in Table 1. According to BMI, the patients were categorized into two groups: the obese group (BMI  $\geq 25$ ), which included 71 (69.6%) patients, and the nonobese group (BMI  $< 25$ ), which included 31 (30.4%) patients. The obese and nonobese groups were

compared using independent-samples *t*-test for IOS and spirometry data. There were significant differences between the two groups and they are outlined in Table 2.

The patients were further subclassified as follows: normal (BMI = 18–24.9), which included 31 (30.4%) patients; overweight (BMI = 25–29.9), which included 28 (27.5%) patients; obese (BMI = 30–34.9), which included 19 (18.6%) patients; and morbidly obese (BMI ≥35), which included 24 (23.5%) patients. The four groups were compared with regard to IOS and spirometry data using one-way analysis of variance followed by multiple comparisons with the post-hoc Bonferroni method; results are presented in Figs 1 and 2.

**Table 1 Patients' characteristic data**

Characteristics	Mean	SD	Minimum	Maximum
Age (years)	45.88	11.54	20	79
BMI (kg/m <sup>2</sup> )	30.35	9.01	18	57.80
NC (cm)	42.58	3.53	33	49
WHR	0.94	0.11	0.73	1.15
R5 (%predicted)	214.24	79.35	89.50	396
R20 (%predicted)	159.02	54.40	25	259
R5–R20 (%predicted)	56.36	45.14	11	226
predicted X5–x5 (kPa/l/s)	0.32	0.211	0.06	0.88
FVC (%predicted)	80.74	10.79	32	126
FEV <sub>1</sub> (%predicted)	80.17	6.37	50	109
FEV <sub>1</sub> /FVC ratio	85.1	4.50	50	98
MMEF (%predicted)	36.98	14.52	22.40	61.30

FEV<sub>1</sub>, forced expiratory volume in 1 s; FVC, forced vital capacity; MMEF, maximal mid expiratory flow; NC, neck circumference; R5, total airway resistance; R20, central airway resistance; WHR, waist to hip ratio; X5, distal capacitive reactance.

**Table 2 Comparison between the obese and nonobese groups regarding impulse oscillometry and spirometry parameters using the independent-sample *t*-test**

Parameters	Nonobese (mean ± SD)	Obese (mean ± SD)	<i>t</i>	<i>P</i>
R5 (%predicted)	127.29 ± 19.71	217.66 ± 75.68	6.54	0.000*
R20 (%predicted)	106.19 ± 13.54	140.39 ± 53.88	3.48	0.001*
R5–R20 (%predicted)	21.68 ± 16.30	78.93 ± 55.62	5.61	0.000*
X5 (kPa/l/s)	0.16 ± 0.08	0.39 ± 0.21	5.71	0.000*
FVC (%predicted)	87.04 ± 21.22	73.7 ± 22.57	9.74	0.000*
FEV <sub>1</sub> (%predicted)	87.22 ± 29.99	73.73 ± 20.81	4.75	0.000*
FEV <sub>1</sub> /FVC ratio	94.5 ± 11.95	78.34 ± 15.18	2.00	0.048*
MMEF (%predicted)	35.91 ± 15.22	37.45 ± 14.29	-0.49	0.624

There were significant differences between the two groups regarding all impulse oscillometry data and all spirometry except MMEF; FEV<sub>1</sub>, forced expiratory volume in 1 s; FVC, forced vital capacity; MMEF, maximal mid expiratory flow; R5, total airway resistance; R20, central airway resistance; X5, distal capacitive reactance; \*Significant at *P* < 0.05.

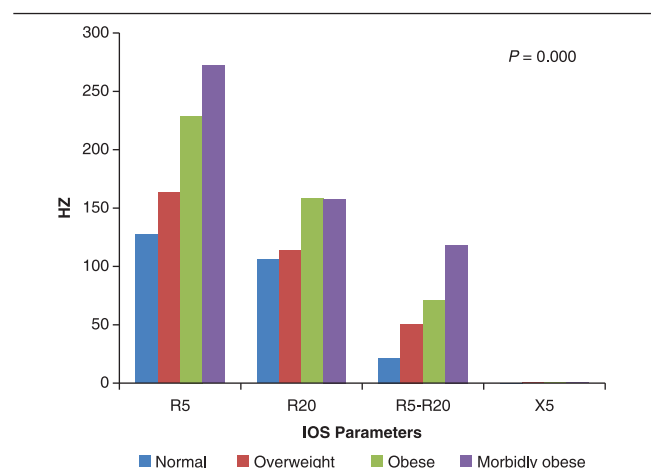
The obese patients were reclassified according to WHR into the truncal obese group (WHR ≥0.90 in males and ≥0.85 in females), which included 41 (57.7%) patients, and the nontruncal obese group (WHR <0.90 in males and <0.85 in females), which included 30 (42.3%) patients. The two groups were compared as regards the IOS and spirometry data using the independent-samples *t*-test. These results are presented in Table 3.

Correlation of anthropometric measures with IOS and spirometry data was performed using Pearson's correlation coefficient. There were significant positive correlations between BMI and IOS data — namely, R5 (*r* = 0.706, *P* = 0.000), R20 (*r* = 0.352, *P* = 0.000), R5–R20 (*r* = 0.695, *P* = 0.000), and X5 (*r* = 0.469, *P* = 0.000). There were significant negative correlations between BMI and spirometry data — namely, FVC (*r* = -0.861, *P* = 0.000), FEV<sub>1</sub> (*r* = -0.618, *P* = 0.000), and FEV<sub>1</sub>/FVC ratio (*r* = -0.555, *P* = 0.000), but there was no significant correlation with maximal mid expiratory flow (MMEF) (*P* > 0.05).

As regards neck circumference, there were significant positive correlations with IOS data — namely, R5 (*r* = 0.323, *P* = 0.001), R20 (*r* = 0.258, *P* = 0.009), R5–R20 (*r* = 0.230, *P* = 0.02), and X5 (*r* = 0.307, *P* = 0.002). There were significant negative correlations between neck circumference and spirometry data — namely, FVC (*r* = -0.407, *P* = 0.000) and FEV<sub>1</sub> (*r* = -0.213, *P* = 0.032), but there were no significant correlations with FEV<sub>1</sub>/FVC ratio and MMEF (*P* > 0.05).

Regarding WHR there were significant positive correlations with IOS data — namely, R5 (*r* = 0.712,

**Fig. 1**



Comparison between the four groups with respect to impulse oscillometry (IOS) data using one-way analysis of variance followed by multiple comparisons with the post-hoc Bonferroni method. There were significant differences between the four groups regarding total airway resistance (R5) (*F* = 42.47, *P* = 0.000), central airway resistance (R20) (*F* = 10.93, *P* = 0.000), R5–R20 (*F* = 26.25, *P* = 0.000), and X5 (*F* = 11.87, *P* = 0.000).

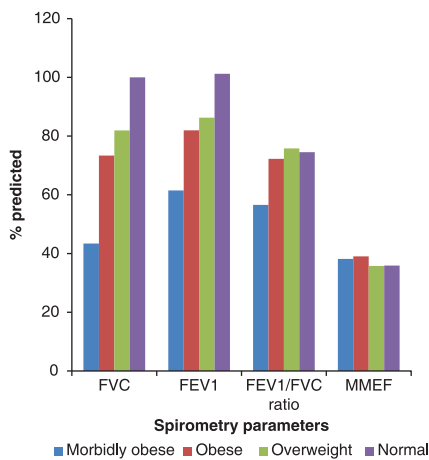
$P = 0.000$ ),  $R20$  ( $r = 0.351, P=0.000$ ),  $R5-R20$  ( $r = 0.677, P = 0.000$ ), and  $X5$  ( $r = 0.257, P=0.009$ ). There were significant negative correlations between WHR and spirometry data — namely, FVC ( $r = -0.606, P = 0.000$ ),  $FEV_1$  ( $r = -0.472, P = 0.000$ ), and  $FEV_1/FVC$  ratio ( $r = -0.394, P=0.000$ ), but there was no significant correlation with MMEF ( $P > 0.05$ ). These correlations are illustrated in Figs 3–6.

**Table 3 Comparison between truncal obese and nontruncal obese groups regarding impulse oscillometry and spirometry parameters using the independent sample *t*-test**

Parameters	Nontruncal	Truncal	<i>t</i>	<i>P</i>
R5 (%predicted)	161.51 ± 62.64	211.10 ± 79.10	-3.405	0.001*
R20 (%predicted)	123.51 ± 50.70	134.72 ± 46.03	-1.164	0.247
R5-R20 (%predicted)	40.93 ± 31.16	76.55 ± 62	-3.459	0.001*
X5 (kPa/l/s)	0.32 ± 0.21	0.32 ± 0.22	-0.268	0.790
FVC (%predicted)	88.27 ± 35.3	75.25 ± 26	2.146	0.034*
$FEV_1$	90.06 ± 30.54	79.88 ± 22.16	1.951	0.054
$FEV_1/FVC$ ratio	72.38 ± 15.43	68.63 ± 13.7	1.297	0.198
MMEF (%predicted)	34.98 ± 14.69	38.44 ± 14.34	-1.192	0.236

There were significant differences between the two groups regarding R5 and R5-R20 and there was no significant difference regarding R20 and X5. Also there were significant differences between the two groups regarding FVC and there were no significant differences regarding  $FEV_1$ ,  $FEV_1/FVC$  ratio, and MMEF;  $FEV_1$ , forced expiratory volume in 1 s; FVC, forced vital capacity; MMEF, maximal mid expiratory flow; R5, total airway resistance; central airway resistance; X5, distal capacitive reactance; \*Significant at  $P < 0.05$ .

**Fig. 2**



Comparison between the four groups with respect to spirometry data using one-way analysis of variance followed by multiple comparisons with the post-hoc Bonferroni method. There were significant differences between the four groups regarding forced vital capacity (FVC) ( $F = 75.86, P = 0.000$ ), forced expiratory volume in 1 s ( $FEV_1$ ) ( $F = 14.50, P = 0.000$ ), and  $FEV_1/FVC$  ratio ( $F = 12.84, P = 0.000$ ). There was no significant difference regarding maximal mid expiratory flow (MMEF) ( $P > 0.05$ ).

Simultaneous effects of different anthropometric data including BMI, neck circumference, and WHR were analyzed by multiple linear regression analysis, with the IOS data (R5, R20, and R5-R20) as the dependent variables, and independent variables being selected by the stepwise procedure.

The first linear regression model used R5 as the dependent variable; the model was significant ( $F = 57.412, P = 0.000$ ) and accounted for 63% of R5 estimation. The most powerful predictor was the WHR [95% confidence interval (CI) 199.31–418.39], followed by BMI (95% CI 2.16–4.78). The multiple linear regression model is shown in Fig. 7.

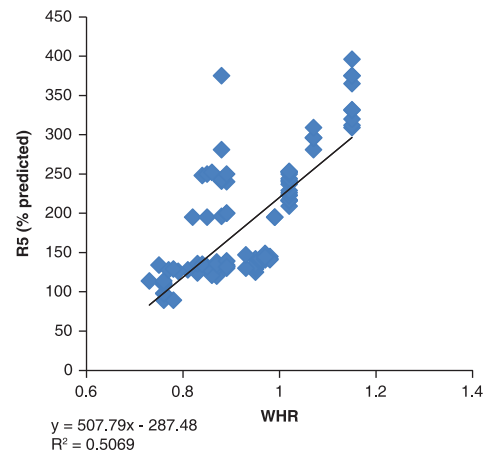
The second linear regression model used R5-R20 as the dependent variable; the model was significant ( $F = 45.915, P = 0.000$ ) and accounted for 58% of R5-R20 estimation. The most powerful predictor was BMI (95% CI 1.66–3.65), followed by WHR (95% CI 118.39–284.48). The multiple linear regression model is shown in Fig. 8.

A third multiple regression model was tried using R20 as the dependent variable. The model accounted for only 18% of estimation of R20 and none of the anthropometric variables were significant contributors in this estimation.

**Discussion**

The effect of obesity and overweight on pulmonary mechanics and airway resistance is the main concern of this study. As expected, our data showed that there were significant differences between the two groups, obese and nonobese, as regards spirometric data

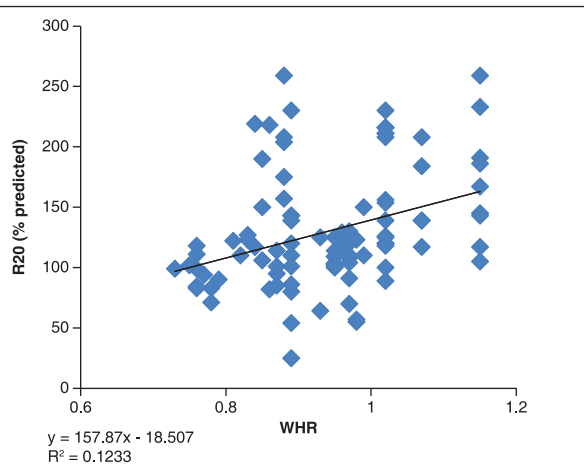
**Fig. 3**



There was a significant positive correlation between waist to hip ratio (WHR) and total airway resistance (R5) ( $r = 0.712, P = 0.000$ ) on using Pearson’s correlation coefficient.

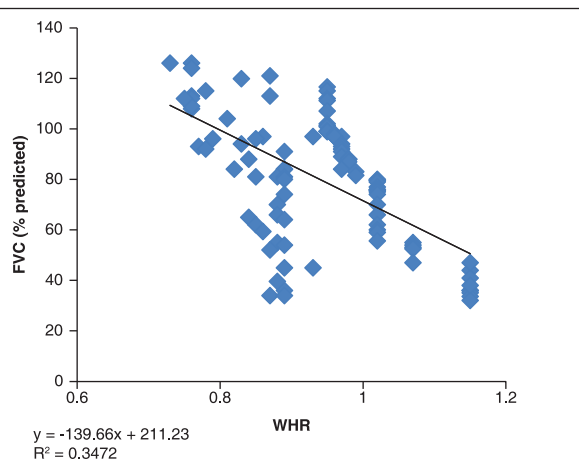
including FVC, FEV<sub>1</sub>, and FEV<sub>1</sub>/FVC data except for MMEF. There was significant difference between the two groups as regards IOS data, especially R5, which gives information about the entire respiratory tract, as well as R20, R5–R20, which indicates the central and the peripheral airway resistance, and X5, the distal capacitive reactance elastic lung and thorax components affecting compliance and therefore in turn affecting small airway resistance. These results were similar to those studies that proved that obesity decreases the total respiratory system compliance by two-thirds and also increases airway resistance. Moreover, these studies proved that FEV<sub>1</sub> is lower in obese patients compared with nonobese patients; obese patients also had their flow rates at 50 and 75% of exhaled vital capacity decreased. Further, airway resistance was significantly greater in obese patients [12,13].

Fig. 4



There was a significant positive correlation between waist to hip ratio (WHR) and central airway resistance (R20) ( $r = 0.351$ ,  $P = 0.000$ ) on using Pearson's correlation coefficient.

Fig. 6

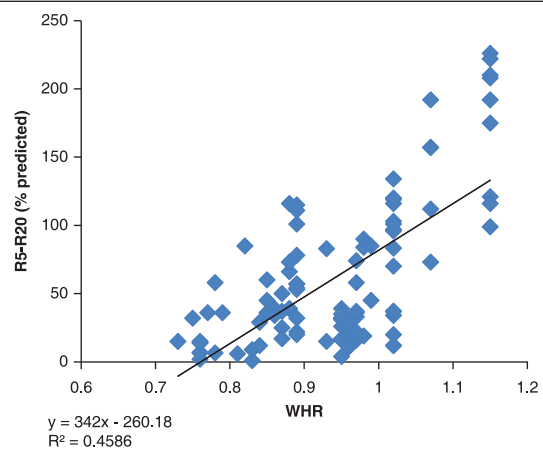


There was a significant negative correlation between waist to hip ratio (WHR) and forced vital capacity (FVC) ( $r = -0.606$ ,  $P = 0.000$ ) on using Pearson's correlation coefficient.

The results of this study showed that IOS detected peripheral airway resistance in the obese group, and diagnosing small airway disease gives it an advantage over spirometry. This agrees with those studies that concluded that IOS provides additional information not obtained by simple spirometry and that airflow resistance measurements were not well predicted by spirometry [14,15].

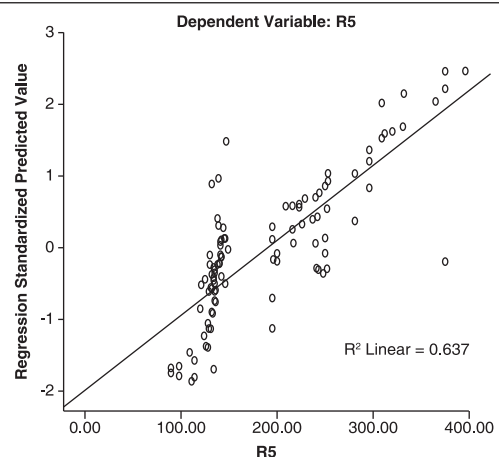
The four subgroups of normal, overweight, obese, and morbidly obese were compared with regard to IOS and spirometry data. There were statistically significant differences between the four groups with respect to all data IOS and spirometric data. This was previously proved by various studies: for example, the observations of Zerah *et al.* [16] who examined airway resistance in a

Fig. 5



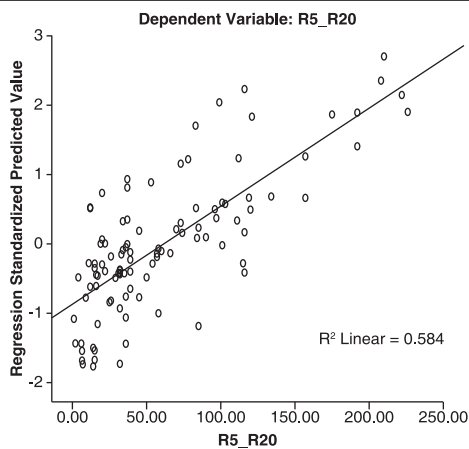
There was a significant positive correlation between waist to hip ratio (WHR) and total airway resistance–central airway resistance (R5–R20) ( $r = 0.677$ ,  $P = 0.000$ ) on using Pearson's correlation coefficient.

Fig. 7



Multiple linear regression model with total airway resistance (R5) as the dependent variable and the anthropometric measures BMI, neck circumference, and waist to hip ratio as independent variables selected by the stepwise procedure. The model is significant ( $F = 57.412$ ,  $P = 0.000$ ) with a coefficient of determination of 63%.

Fig. 8



Multiple linear regression model with total airway resistance–central airway resistance (R5–R20) as the dependent variable and the anthropometric measures BMI, neck circumference, and waist to hip ratio as independent variables selected by the stepwise procedure. The model is significant ( $F = 45.915$ ,  $P = 0.000$ ) with coefficient of determination of 58%.

group of obese patients who were subdivided into mildly obese, moderately obese, and morbidly obese groups; in their study airway resistance increased significantly with the degree of obesity, and was inversely related to changes in the functional residual capacity and also to the elastic load. Obese individuals must overcome the increased airway resistance resulting from the decrease in lung volume due to obesity [16].

In this study the obese patients were reclassified according to WHR into truncal obese (WHR  $\geq 0.90$  in males and  $\geq 0.85$  in females), which included 41 (57.7%) patients, and nontruncal obese (WHR  $< 0.90$  in males and  $< 0.85$  in females), which included 30 (42.3%) patients. The two groups were compared as regards the IOS and showed a statistically significant difference between them with respect to R5 and R5–R20 but no significant difference with respect to R20 and X5, thus indicating increased peripheral airway resistance. The primary reason is obviously due to a decrease in chest wall compliance, which results from the accumulation of fatty adipose tissues in the abdomen, diaphragm, and around the lower ribs of obese individuals. Thus, it was previously proved that the total respiratory compliance is markedly decreased in obese patients having truncal obesity compared with obese individuals without truncal obesity; this reduction is mostly due to the decreased compliance, although it may also be due to an increase in airway resistance [17].

In this study, there was an insignificant difference with respect to R20; thus, peripheral resistance is the main factor causing significant increase in airway resistance in the group with truncal obesity. With regard to

WHR, there were significant correlations with IOS data — namely, R5, R5–R20, R20, and X5. Also there were significant negative correlations between WHR and spirometry data — namely, FVC, FEV<sub>1</sub>, and FEV<sub>1</sub>/FVC ratio, but there was no significant correlation as regards MMEF, which may be less sensitive than IOS in detecting airway resistance formed by the peripheral airway affection.

When the truncal and nontruncal obese groups were compared for spirometry data, there was a significant difference between the two groups only for FVC.

Simultaneous effects of different anthropometric data including BMI, neck circumference, and WHR were analyzed using multiple linear regression analysis, with the IOS data (R5, R20, and R5–R20) as the dependent variables, and independent variables selected by the stepwise procedure. The most powerful predictor of R5 was the WHR, whereas the most robust predictor of peripheral resistance was BMI, followed by WHR. However, the study of the simultaneous effects of BMI, neck circumference, and WHR on R20, which measures the R20, revealed that they accounted for only 18% of estimation of R20 and none of the anthropometric variables were significant contributors in this estimation. This confirms the effect of truncal obesity on the peripheral and consequently R5 without a direct effect on R20. All of the above results confirm those reached by a previous research that concluded that truncal obesity is most likely to affect pulmonary volumes, without direct effects on pulmonary obstruction [7].

In conclusion, truncal obesity significantly affects airway resistance, as evaluated by spirometry and IOS.

## Acknowledgements

### Conflicts of interest

None declared.

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