BMJ Open Respiratory Research

Exploratory study into the effect of abdominal mass loading on airways resistance and ventilatory failure

Raj S Dattani,[1,2,3] Casey B Swerner,[4] John R Stradling,[1,5] Ari RG Manuel[1,5]

To cite: Dattani RS, Swerner CB, Stradling JR, *et al*. Exploratory study into the effect of abdominal mass loading on airways resistance and ventilatory failure. *BMJ Open Resp Res* 2016;**3**:e000138. doi:10.1136/bmjresp-2016-000138

►Additional material is available. To view please visit the journal (http://dx.doi.org/10.1136/bmjresp-2016-000138).

ARGM is the senior author. RSD and CBS contributed equally.

Received 21 March 2016
Revised 4 May 2016
Accepted 13 May 2016

For numbered affiliations see end of article.

Correspondence to
C B Swerner;
caseyswerner@gmail.com

ABSTRACT

Objective: We hypothesised that the airway resistance during tidal breathing would correlate with a particular pattern of increasing obesity, particularly when supine, and would differ between participants with and without ventilatory failure.

Methods: In our cross-sectional cohort study, 72 morbidly obese patients (40 males, 32 females, mean body mass index (BMI) 47.2) had measurements of both airways resistance (by impulse oscillometry (IOS)) and adiposity (by dual-energy X-ray absorptiometry (DXA)).

Results: All measures of airways resistance increased in the supine position: total airways resistance (R5) +37% (p<0.0005); large airways resistance (R20) +29% (p<0.0005); and small airways resistance (R5–R20) +52% (p<0.0005). BMI was correlated with seated R5, seated R5–R20, supine R5 and supine R5–R20 (r=0.33 p<0.006, r=0.32 p<0.004, r=0.30 p<0.02 and r=0.36 p<0.04, respectively). Visceral adipose tissue mass was correlated with supine R5–20 (r=0.46 p<0.05). Supine measures of total airways resistance (R5) and large airways resistance (R20) differed between those with and without ventilatory failure, as did mean weight and BMI.

Conclusions: Our study identifies a potentially detrimental effect of the supine posture on tidal breathing airways resistance in obese patients. This change is correlated most with visceral adipose tissue mass and the small airways. We were able to demonstrate that supine increases in airways resistance during tidal breathing, within obese patients, are different between those with and without ventilatory failure.

Trial registration number: NCT01380418; pre-results.

KEY MESSAGES

In the largest study of respiratory mechanics (by impulse oscillometry) within an obese population to date, we identify an effect of abdominal mass loading on airways resistance. This effect is greatest in the small airways and is expressed differentially between those with and without ventilatory failure.

INTRODUCTION

'Pickwickian' syndrome or obesity hypoventilation syndrome (OHS) is defined as obesity-related chronic ventilatory failure, where ventilatory failure is defined as a daytime hypercapnia with an arterial $PaCO_2$ of >6 kPa.

OHS has been characterised as a 'restrictive' disease; whereby in obesity increased mass requires increased work to move the thoracic wall and abdomen during breathing. The inability to adequately compensate therefore results in hypoventilation and retention of carbon dioxide in OHS.

To some degree, classical spirometry results from obese patients support this hypothesis: these characteristically show a preserved forced expiratory volume in 1 s (FEV1)/forced vital capacity (FVC) ratio,[1–8] with FEV1 not depressed until at the extremes of morbid obesity,[4] implying a restrictive pattern. Furthermore, obese patients demonstrate reduced expiratory reserve volume (ERV)[5 9 10] and functional residual capacity (FRC) values.[5 9 10] However, total lung capacity (TLC)[3 5 7 11] and residual volume (RV)[4 11] are spared; others have shown that overall airways resistance is increased in obesity, implying an obstructive element in the pathophysiology.[12 13] Given that the mechanical properties of the airways, such as resistance, are dependent on lung volume itself, some have attempted to resolve the question by analyses of airways resistance that account for reduced lung volume.[14] However, two studies have found that increased resistance persists after this adjustment.[6 15] Furthermore, the finding that the frequency dependence of resistance increases with obesity has implicated the small peripheral airways as the site of obesity-related changes.[12]

In normal participants, there is a small reduction in FRC and increase in airflow resistance on adopting the supine posture.[16] Supine posture is a physiological variable,

thought to be due to the gravitational effects of the abdominal contents exerting mass loading, resulting in the relaxed diaphragm taking a more expiratory position; increasing mass when supine should amplify the tendency to lengthen the passive diaphragm.

We have recently published cohort data demonstrating that visceral adipose tissue (VAT) mass is an independent correlate of chronic ventilatory failure,[17] and that, within this cohort, obese patients with a raised bicarbonate but no hypercapnia represent an early form of OHS.[18]

Here, we further investigate this cohort with the hypothesis that the VAT component within obese patients may specifically mediate respiratory pathophysiology. We use impulse oscillometry (IOS), a forced oscillation technique pioneered by DuBois *et al*,[19] which, in contrast to classical spirometry, provides an effort independent measure of lung function. In this technique, forced oscillations are superimposed over a range of frequencies on regular breathing allowing the extraction of certain derivatives to estimate resistance in different parts of the lung. Dual-energy X-ray absorptiometry (DXA) is an established method of evaluating body composition and distributions and has been validated by CT studies.[20 21]

METHODS

Study design and setting

An open cross-sectional cohort study of obese patients was undertaken between June 2011 and September 2013 at the Oxford Sleep Unit, Oxford Centre for Respiratory Medicine. The study was registered prospectively with a global trials registry site (ClinicalTrials.gov NCT01380418) and was approved by the Oxford Research Ethics Committee (Oxfordshire REC B 11/H0605/9).

Participants

A convenience sample of obese patients (defined as a body mass index (BMI) of >30 kg/m^2), with or without chronic ventilatory failure, was recruited following referral to the sleep and ventilation unit, or during assessment for possible bariatric surgery. Exclusion criteria included: being currently on any drug that might potentially stimulate/depress respiration, a diuretic or a theophylline; being diagnosed with obstructive lung disease (defined as an FEV1/FVC ratio <70%); or the presence of other severe comorbidities, for example, congestive cardiac failure, primary central nervous system or neuromuscular diseases, and untreated hypothyroidism; or being currently on treatment with continuous positive airways pressure (CPAP) or non- invasive ventilation.

Measures of airways resistance by IOS

Seated IOS measurements were taken in line with European Thoracic Society guidelines (ETS) Task Force recommendations,[22] using the Jaeger MS-IOS machine (Care Fusion, Germany). Forced oscillation was applied at the airway opening during normal tidal breathing to measure respiratory impedance. The participant supported the cheeks and floor of the mouth with the palms of the hands to minimise dissipation of the applied flow in the upper airway. The head and neck were kept in a neutral to slightly extended position, while the participant maintained tidal breathing via a large-bore mouthpiece and with a nose clip in place. In the supine measurements, participants lay in the standard anatomical supine position, with legs fully extended and arms fully extended supine. A nose clip was worn to avoid shunting via the nose during the measurement, and the cheeks were firmly supported by the experimenter to avoid shunting across the mouth. The participant was asked to breathe normally. Data were excluded if the participant began to swallow, breathe irregularly, hyperventilate or if glottis closure occurred. Furthermore, if there was leakage around the mouthpiece or a fault with the nose clip, or improper support of the cheeks, data were excluded. IOS measurements of overall resistance (R5) and large airways resistance (R20) were recorded, with the R5–R20 calculation used to determine small airways resistance. A Lilly-type pneumotachograph was used for flow measurements, following calibration with a 3 L syringe. Flow was kept to a maximum peak expiratory and peak inspiratory flow of 0.50 L/s (body temperature and pressure, saturated (BTPS)), thus simulating the flow range of spontaneous quiet breathing. Before and after testing, a 90 s impedance measurement was performed using a fixed impedance of 0.2 kPa/s/L to ascertain impulse pressure and flow accuracy.

Measures of adiposity and its distribution

In addition to basic anthropometric data (age, sex, height, weight, BMI), body fat and its distribution were assessed by DXA (Lunar system (GE Healthcare, Chalfont St Giles, UK)). DXA measures were of total body fat and trunk fat, with an estimation of VAT mass calculated only available in participants with a BMI≤40, as the algorithm has not been verified beyond this level of obesity.[23]

Participant grouping

Following arterial blood gas analysis, participants were divided a priori into two 'clinical' groups, representing those with ventilatory failure and those without. Those with a normal daytime $PaCO_2$ but elevated base excess were excluded as they may represent an early form of OHS[18] and therefore be confounding.

1. Normal daytime $PaCO_2$ (≤6 kPa) and base excess <2 mmol/L
2. Elevated daytime $PaCO_2$ (>6 kPa).

Statistical analyses

Data analyses were recorded using MS Excel and analysed using SPSS (V.22, IBM Corporation Ltd, USA). Descriptive data are presented as the mean and SD for normally distributed data. Data were tested for normality and parametric analyses conducted when appropriate.

Paired t tests were performed to analyse any differences between seated and supine measures of airways resistance generated by the IOS method (R5, R20 and R5–R20). Pearson's correlations were performed between measures of adiposity (weight, BMI, DXA total fat, DXA trunk fat and DXA VAT mass) and both seated and supine IOS measures of airways resistance. A series of unpaired t tests were used to test for differences in mean values of airways resistance between our two participant groups.

RESULTS

Seventy-two morbidly obese patients (40 males, 32 females) had anthropometric measurements, measures of airways resistance and adiposity as described. Descriptive results for these data-points are presented in table 1. Mean measures for all components of airways resistance were elevated when compared with previously described values for non-obese patients in the supine position (reference data available in online supplementary table S1).

Paired t tests demonstrated that all measures of airways resistance increase significantly when measured in the supine position versus the seated position. Supine R5 values were higher (0.69±0.21 kPa/s/L) than seated values (0.50±0.19 kPa/s/L), a statistically significant increase of 0.19 kPa/s/L (37%) (95% CI 0.14 to 0.23, t(71)=7.994, p<0.0005, d=0.95). Supine R5-R20 CI 0.06 to 0.12, values were higher (0.43±0.15 kPa/s/L) than seated values (0.33±0.12 kPa/s/L), a statistically significant increase of 0.10 kPa/s/L (29%) (95% CI 0.06 to 0.12, t(70)=7.122, p<0.0005, d=0.85). Supine R5–R20 values were higher (0.26±0.15 kPa/s/L) than seated values (0.17±0.11 kPa/s/L), a statistically significant increase of 0.09 kPa/s/L (52%) (95% CI 0.61 to 0.12, t(71)=6.310, p<0.0005, d=0.75).

Pearson's correlation coefficients between measures of adiposity and airways resistance, both in the seated and supine positions, are presented in table 2. Significant p values are marked with an asterisk. BMI was significantly correlated with seated and supine measures of total airways resistance (R5) and small airways resistance (R5–R20), with the strength of this correlation varying between r=0.30 and r=0.36. VAT mass was significantly correlated with a supine measure of small airways resistance, with the strength of this correlation being r=0.46. Since VAT mass could only be validly measured in the subgroup of our cohort with a BMI<40, descriptive data demonstrating similar characteristics to the overall cohort are presented in online supplementary table S2.

Student's t tests between our two participant groups are presented in table 3. Significant p values are marked with an asterisk. Weight, BMI, supine measures of total airways resistance (R5) (as p = 0.050 exactly and is NOT less than 0.05) were statistically different between those with and without ventilatory failure.

DISCUSSION

This exploratory study provides data on airways resistance in the largest cohort of obese patients studied by IOS until now. That respiratory resistance is increased in the supine posture is in concordance with previously

Table 1 Descriptive statistics for anthropometric measures, measures of adiposity and IOS measures of airways resistance

	n	Minimum	Maximum	Mean	SD
(A) Anthropometric measures					
Age (years)	72	26	74	52.0	8.8
Height (cm)	72	149	188	170.0	9.3
Weight (kg)	72	78	229	136.0	29.5
(B) Measures of adiposity					
BMI (kg/m^2)	72	32.3	73.9	47.2	9.8
DXA total fat (kg)	55	32.3	86.2	58.2	13.5
DXA trunk fat (kg)	55	22.0	55.7	35.7	8.0
DXA visceral adipose tissue mass (kg)	21	1.8	5.2	3.2	1.0

	Seated (n=70)			Supine (n=69)			Supine vs Seated	
	Minimum	Maximum	Mean (SD)	Minimum	Maximum	Mean (SD)	Difference	p Value
(C) IOS measures of airways resistance								
R5 (kPa/L/s)	0.18	1.01	0.50 (0.18)	0.10	1.17	0.69 (0.21)	0.19 (+37%)	<0.0005
R20 (kPa/L/s)	0.13	0.70	0.33 (0.12)	0.10	0.79	0.43 (0.12)	0.10 (+29%)	<0.0005
R5–R20 (kPa/L/s)	0.00	0.50	0.17 (0.11)	–0.02	0.69	0.26 (0.15)	0.09 (+52%)	<0.0005

R5, overall airways resistance; R20, large airways resistance; R5–R20, small airways resistance; BMI, body mass index; DXA, dual-energy X-ray absorptiometry; IOS, impulse oscillometry.

Table 2 Correlations between measures of adiposity and measures of airways resistance

	n	Pearson's r	p Value
(A) Measures of adiposity vs seated R5 (total airways resistance)			
Weight (kg)	70	+0.13	0.28
BMI	70	+0.33	0.005*
DXA total fat (kg)	54	+0.15	0.28
DXA trunk fat (kg)	54	+0.09	0.51
DXA visceral adipose tissue mass (kg)	21	−0.11	0.62
(B) Measures of adiposity vs seated R20 (large airways resistance)			
Weight (kg)	70	−0.01	0.99
BMI	70	+0.18	0.13
DXA total fat (kg)	54	−0.01	0.31
DXA trunk fat (kg)	54	−0.07	0.48
DXA visceral adipose tissue mass (kg)	21	−0.36	0.11
(C) Measures of adiposity vs seated R5–R20 (small airways resistance)			
Weight (kg)	69	+0.22	0.07
BMI	69	+0.32	0.003*
DXA total fat (kg)	54	+0.27	0.048*
DXA trunk fat (kg)	54	+0.24	0.08
DXA visceral adipose tissue mass (kg)	20	−0.17	0.49
(D) Measures of adiposity vs supine R5 (total airways resistance)			
Weight (kg)	69	+0.24	0.05
BMI	69	+0.30	0.01*
DXA total fat (kg)	53	+0.09	0.50
DXA trunk fat (kg)	53	+0.11	0.43
DXA visceral adipose tissue mass (kg)	21	+0.36	0.11
(E) Measures of adiposity vs supine R20 (large airways resistance)			
Weight (kg)	69	+0.13	0.30
BMI	69	+0.23	0.06
DXA total fat (kg)	53	+0.02	0.92
DXA trunk fat (kg)	53	+0.03	0.98
DXA visceral adipose tissue mass (kg)	21	+0.10	0.67
(F) Measures of adiposity vs supine R5–R20 (small airways resistance)			
Weight (kg)	69	±0.22	0.07
BMI	69	±0.36	0.03*
DXA total fat (kg)	53	+0.12	0.39
DXA trunk fat (kg)	53	+0.16	0.27
DXA visceral adipose tissue mass (kg)	21	+0.46	0.04*

R5, overall airways resistance; R20, large airways resistance; R5–R20, small airways resistance; BMI, body mass index; DXA, dual-energy X-ray absorptiometry.

published data,[6 14 24] though our results further this knowledge by demonstrating a differential effect by airway calibre: there is a large effect in small airways (52%) with a smaller effect in larger airways (29%), with total airways resistance lying in between these two values (37%).

We also demonstrate a novel correlation between BMI and supine VAT mass with airways resistance. Our previous work[17] has demonstrated that VAT mass is the best independent correlate of ventilatory failure, while BMI is a comparatively poor correlate. Taken together, these two results suggest that fat within the abdominal cavity is significant in obesity-related respiratory pathophysiology. This hypothesis is supported with classical spirometry results within the literature: a negative correlation between VAT and FEV1/FVC is documented in the literature. While this correlation was initially discovered in studies using waist circumference as a surrogate marker for VAT mass, the result has also been replicated using more precise techniques: Rossi *et al*[25] (CT), Park *et al*[26] (CT), Inomata *et al*[27] (Bioimpedance) and Thijs *et al*[28] (MRI).

Furthermore, a cross-sectional population-based study of nearly 122 000 participants showed that abdominal obesity was the strongest correlate of impaired lung function[29] while early work in this area identified a reduced ERV as a possible provoker of obesity-related hypoventilation.[30] Sutherland *et al*[31] used a wide range of body fat variables (including DXA to determine the effect of fat distribution on lung volumes), to demonstrate that in healthy adults lung volumes were only loosely associated with BMI. BMI is regarded as an unreliable method of evaluating causal associations between obesity and disease, a view based on the finding that the distribution of adipose tissue has been shown to be more predictive in the development of metabolic disorders.[29 32]

In a clinical context, this preferential abdominal obesity is a plausible contributory cause to the gradual development of daytime hypercapnia. The effect of a raised intra-abdominal pressure on ventilation is well recognised in individuals with other pathologies that raise intra-abdominal pressure (refer to Hedenstierna and Larsson[33] for review). This is particularly seen in acutely ill patients in intensive care, where additional obesity has been shown to be a significant risk factor for ventilatory failure.[34]

Additionally, our results provide evidence for small airways as the anatomical location of obesity-related resistive pathophysiology, a conclusion corroborated by the finding that the frequency dependence of resistance increases with obesity.[12] A simple explanation for this finding might be that in the supine position the effect of mass loading to the thoracic cavity, and thus lung volume, is amplified. In our study, hypercapnic obese individuals (ie, those meeting the criteria for OHS), had, on average, greater airways resistance in the supine position when compared with obese individuals who were not in ventilatory failure. This result supports the suggestion that supine mass loading may mediate obesity-related respiratory pathophysiology to the extent that it causes disease.

Mass loading may affect respiratory mechanics by other mechanisms such as altering chest wall compliance

Table 3 Student's t tests of anthropometric and airways measures between participant groups

	Non-ventilatory failure group n=31	Ventilatory failure group n=15	t-Statistic (df)	Difference between means	Percentage of difference	p Value of t-statistic
Age (years)	53.2 (9.5)	54.6 (8.1)	0.492 (44)	+1.41	+2.65	0.625
Weight (kg)	130.6 (29.4)	153.3 (36.8)	2.256 (44)	+22.7	+17.3	0.029*
BMI (kg)	45.1 (9.1)	53.0 (12.3)	2.201 (21.7)†	+7.9	+17.5	0.039*
R5 seated	0.50 (0.22)	0.58 (0.16)	1.325 (44)	+0.083	+16.8	0.192
R20 Seated	0.33 (0.14)	0.35 (0.10)	0.538 (44)	+0.021	+6.45	0.593
R5–R20 seated	0.167 (0.12)	0.22 (0.12)	1.645 (44)	+0.062	+37.6	0.107
R5 supine	0.64 (0.20)	0.79 (0.24)	2.253 (44)	+0.15	+23.6	0.029*
R20 supine	0.40 (0.12)	0.46 (0.10)	2.016 (44)	+0.070	+17.7	0.05
R5–R20 supine	0.24 (0.13)	0.32 (0.19)	1.488 (20.05)†	+0.080	+33.1	0.152

*p<0.05. Data are presented as mean values with SDs in brackets.
†Levene's Test for homogeneity of variances violated (p<0.05), therefore t test not assuming equal variances performed.
R5, overall airways resistance; R20, large airways resistance; R5–R20, small airways resistance; BMI, body mass index.

directly by restricting expansion and causing the respiratory system to operate on a less compliant part of the pressure-volume curve. Interestingly, one study reports the compliances of the thorax and total respiratory system in obese patients as low as 20% of normal.[35] Alternatively, mass loading preventing descent of the diaphragm inferiorly might change thoracic pressure-volume characteristics, an effect seen by Steier *et al*.[36] Sharp *et al*[37] demonstrated that overstretching of the diaphragm reduced muscle fibre contraction efficiency.

Clear limitations of our results and conclusions relate to the exploratory nature of the study. While our results report the largest obese cohort studied by IOS, this cohort lacks sufficient power to definitively prove our conclusions. In the context of repeated correlations, a stringent Bonferroni corrected α would have resulted in statistical insignificance for all results and a likely type 2 error. A linked limitation, most likely due to insufficient power, is that small airways resistance did not significantly vary between our two participant groups. In this case, a large SD and positive Levene's test demonstrate unequal variance, further decreasing the power. Second, DXA can only provide estimations of visceral adiposity for participants with a BMI<40, as the algorithm has not been validated above this BMI.[23] This technical limitation can be overcome by the use of CT or MRI studies, but these are costly and labour intensive. We therefore strongly advocate replication of our study with a large cohort of obese patients, including patients with ventilatory failure to conclusively show a differential effect of VAT on respiratory mechanics. However, given that, in addition to a large cohort, this approach would ideally use CT/MRI to estimate VAT across the full range of BMI values, this further work would need to be of a different scale to our current exploratory study.

Author affiliations
[1]Oxford Respiratory Trials Unit, Experimental Medicine Division, Nuffield Department of Medicine, University of Oxford, Oxford, UK
[2]St Peter's College, New Inn Hall Street, Oxford, UK
[3]Centre for Altitude, Space and Extreme Environment Medicine (CASE Medicine), Institute of Sport Exercise and Health, University College London, London, UK
[4]University College London, London, UK
[5]Oxford Centre for Respiratory Medicine, NIHR Oxford Biomedical Research Centre, The Churchill Hospital, Oxford University Hospitals NHS Trust, Oxford, UK

Contributors ARGM and JRS contributed to the cohort inception. ARGM contributed to the data collection. RSD, CBS, ARGM and JRS contributed to the study conception and research question. RSD and CBS contributed to the analysis. RSD and CBS participated in the manuscript writing, and ARGM and JRS edited it. All the authors read and approved the manuscript.

Funding Open access publication costs were defrayed by University College London's institutional subscription to the BMJ open access scheme, which was utilised via C.B.S's status as a research student at UCL. This work was supported by the Oxford Health Services Research Committee, the Oxford NIHR Biomedical Research Centre and the Oxford Radcliffe Hospital Charitable Funds.

Competing interests None declared.

Patient consent Obtained.

Ethics approval Oxford Research Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement The authors are happy to collaborate with any group wishing to use data from their cohort within the bounds of ethical approval and confidentiality.

REFERENCES

1. Schachter LM, Salome CM, Peat JK, *et al*. Obesity is a risk for asthma and wheeze but not airway hyperresponsiveness. *Thorax* 2001;56:4–8.
2. Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. *Arch Intern Med* 2002;162:1477–81.
3. Burki NK, Baker RW. Ventilatory regulation in eucapnic morbid obesity. *Am Rev Respir Dis* 1984;129:538–43.
4. Biring MS, Lewis ML, Liu JT, *et al*. Pulmonary physiologic changes of morbid obesity. *Am J Med Sci* 1999;318:293–7.

5. Jones RL, Nzekwu MM. The effects of body mass index on lung volumes. *Chest* 2006;130:827–33.
6. Watson RA, Pride NB. Postural changes in lung volumes and respiratory resistance in subjects with obesity. *J Appl Physiol (1985)* 2005;98:512–17.
7. Watson RA, Pride NB, Thomas EL, *et al*. Reduction of total lung capacity in obese men: comparison of total intrathoracic and gas volumes. *J Appl Physiol* 2010;108:1605–12.
8. Lazarus R, Sparrow D, Weiss ST. Effects of obesity and fat distribution on ventilatory function: the normative aging study. *Chest* 1997;111:891–8.
9. Ladosky W, Botelho MA, Alubuquerque JP Jr. Chest mechanics in morbidly obese non-hypoventilated patients. *Respir Med* 2001;95:281–6.
10. Collet F, Mallart A, Bervar JF, *et al*. Physiologic correlates of dyspnea in patients with morbid obesity. *Int J Obes (Lond)* 2007;31:700–6.
11. Collins LC, Hoberty PD, Walker JF, *et al*. The effect of body-fat distribution on pulmonary function tests. *Chest* 1995;107:1298–302.
12. Zerah F, Harf A, Perlemuter L, *et al*. Effects of obesity on respiratory resistance. *Chest* 1993;103:1470–6.
13. Rubinstein I, Zamel N, DuBarry L, *et al*. Airflow limitation in morbidly obese, nonsmoking men. *Ann Intern Med* 1990;112:828–32.
14. Yap JC, Watson RA, Gilbey S, *et al*. Effects of posture on respiratory mechanics in obesity. *J Appl Physiol (1985)* 1995;79:1199–205.
15. King GG, Brown NJ, Diba C, *et al*. The effects of body weight on airway calibre. *Eur Respir J* 2005;25:896–901.
16. Duggan CJ, Watson RA, Pride NB. Postural changes in nasal and pulmonary resistance in subjects with asthma. *J Asthma* 2009;41:701–7.
17. Manuel AR, Hart N, Stradling JR. Correlates of obesity-related chronic ventilatory failure. *BMJ Open Respir Res* 2016;3: e000110.
18. Manuel ARG, Hart N, Stradling JR. Is a raised bicarbonate, without hypercapnia, part of the physiologic spectrum of obesity-related hypoventilation? *Chest* 2015;147:362–7.
19. DuBois AB, Brody AW, Lewis DH, *et al*. Oscillation mechanics of lungs and chest in man. *J Appl Physiol* 1956;8:587–94.
20. Lane JT, Mack-Shipman LR, Anderson JC, *et al*. Comparison of CT and dual-energy DEXA using a modified trunk compartment in the measurement of abdominal fat—Springer. *Endocrine* 2005;27:295–9.
21. Glickman SG, Marn CS, Supiano MA, *et al*. Validity and reliability of dual- energy X-ray absorptiometry for the assessment of abdominal adiposity. *J Appl Physiol* 2004;97:509–14.
22. Oostveen E, MacLeod D, Lorino H, *et al*. The forced oscillation technique in clinical practice: methodology, recommendations and future developments. *Eur Respir J* 2003;22:1026–41.
23. Ball SD, Altena TS, Swan PD. Comparison of anthropometry to DXA: a new prediction equation for men. *Eur J Clin Nutr* 2004;58:1525–31.
24. Navajas D, Farre R, Rotger MM, *et al*. Effect of body posture on respiratory impedance. *J Appl Physiol (1985)* 1988;64:194–9.
25. Rossi AP, Watson NL, Newman AB, *et al*. Effects of body composition and adipose tissue distribution on respiratory function in elderly men and women: the health, aging, and body composition study. *J Gerontol A Biol Sci Med Sci* 2011;66:801–8.
26. Park YS, Kwon HT, Hwang SS, *et al*. Impact of visceral adiposity measured by abdominal computed tomography on pulmonary function. *J Korean Med Sci* 2011;26:771–7.
27. Inomata M, Kawagishi Y, Taka C, *et al*. Visceral adipose tissue level, as estimated by the bioimpedance analysis method, is associated with impaired lung function. *J Diabetes Investig* 2012;3:331–6.
28. Thijs W, Alizadeh Dehnavi R, Hiemstra PS, *et al*. Association of lung function measurements and visceral fat in men with metabolic syndrome. *Respir Med* 2014;108:351–7.
29. Leone N, Courbon D, Thomas F, *et al*. Lung function impairment and metabolic syndrome: the critical role of abdominal obesity. *Am J Respir Crit Care Med* 2009;179:509–16.
30. Cullen JH, Formel PF. The respiratory defects in extreme obesity. *Am J Med* 1962;32:525–31.
31. Sutherland TJT, Goulding A, Grant AM, *et al*. The effect of adiposity measured by dual-energy X-ray absorptiometry on lung function. *Eur Respir J* 2008;32:85–91.
32. Arner E, Westermark PO, Spalding KL, *et al*. Adipocyte turnover: relevance to human adipose tissue morphology. *Diabetes* 2010;59:105–9.
33. Hedenstierna G, Larsson A. Influence of abdominal pressure on respiratory and abdominal organ function. *Curr Opin Crit Care* 2012;18:80–5.
34. Holodinsky JK, Roberts DJ, Ball CG, *et al*. Risk factors for intra-abdominal hypertension and abdominal compartment syndrome among adult intensive care unit patients: a systematic review and meta-analysis. *Crit Care* 2013;17:R249.
35. Naimark A, Cherniack RM. Compliance of the respiratory system and its components in health and obesity. *J Appl Physiol* 1960;15:377–82.
36. Steier J, Jolley CJ, Seymour J, *et al*. Neural respiratory drive in obesity. *Thorax* 2009;64:719–25.
37. Sharp JT, Henry JP, Sweany SK, *et al*. Effects of mass loading the respiratory system in man. *J Appl Physiol* 1964;19:959–66.