Obesity: how pulmonary function tests may let us down

José Alberto Neder[1], Danilo Cortozi Berton[2], Denis E. O'Donnell[1]

BACKGROUND

There has been an exponential increase in the prevalence of obesity worldwide.[1] Consequently, there has been an increase in the number of obese individuals referred for pulmonary function tests (PFTs) prior to bariatric surgery, for example, as well as because of chronic wheezing, chronic breathlessness, and multiple comorbidities potentially explaining disproportionate dyspnea.[2] The pulmonologist in charge of interpreting the results of a PFT, as well as the physician requesting the test, should be acquainted with the peculiar effects that obesity has on lung function.

OVERVIEW

A 72-year-old male—smoking history = 32 pack-years; height = 159 cm; and body mass index (BMI) = 48.2 kg/m^2—was referred for a full PFT in a tertiary health care facility due to worsening dyspnea despite maximal therapy for suspected COPD. Office spirometry was, according to the referral note, "unremarkable". In fact, spirometry, whole-body plethysmography, and DLCO were all within the normal range. However, because the patient experienced severe dyspnea and distress after the tests, he was referred to the emergency department by the respiratory therapist. Upon arrival in the emergency

Chart 1. A non-exhaustive list of challenges and pitfalls in the interpretation of pulmonary function tests in obese patients. Note that these sources of confusion increase as does the body mass index, but they are also negatively affected by male sex, height, and abdominal obesity for a given body mass index.

Directional change	Putative mechanism(s)	Common misinterpretation and potential consequences
Spirometry		
↔ FEV_1/FVC in the presence of airway disease	↓ FVC due to early closure of small airways and/or due to ↓ TLC and/or ↑ RV/TLC	No airway disease is present. If the patient is a smoker, false reassurance; if he/she has asthma, undertreatment
↓ FEV_1/FVC in the absence of airway disease	Compression of central airways in the forced maneuver	Excessive pharmacological treatment (usually for asthma) of a patient who, fundamentally, should lose weight
↓ $FEF_{25-75\%}$ due to low FVC in the absence of airway disease	Flows commensurate with volumes	As above
Plethysmography		
↓ TLC in the absence of intraparenchymal restriction	↑ elastic recoil, including the chest wall; common if BMI > 50 kg/m^2, very common if BMI > 60 kg/m^2	Unfounded alert for ILD or another cause of restriction; underestimation of lung overdistension caused by obstruction
↓ FRC in the absence of intraparenchymal restriction	Downward displacement of the chest wall-parenchymal equilibrium point plus mass load effect	As above
↔ IC in the presence of expiratory flow limitation	↓ FRC but ↔ TLC	The effects of air trapping and lung hyperinflation on operating lung volumes are counterbalanced.
Gas exchange		
↔ DLCO in the presence of gas exchange impairment	↑ blood flow in areas of preserved ventilation-perfusion	No impairment in gas exchange
↔ K_{CO} in the presence of gas exchange impairment	K_{CO} ↑ exponentially as V_A ↓	As above
↓ SpO_2 on the six-minute walk test	↑ perfusion of poorly ventilated (dependent) airways with poorly oxygenated mixed venous blood	Overestimation of the impairment caused by any underlying respiratory disease

↔: preserved; ↑: high/increased; ↓: low/decreased; $FEF_{25-75\%}$: forced expiratory flow at 25-75% of FVC; BMI: body mass index; ILD: interstitial lung disease; FRC: functional residual capacity; IC: inspiratory capacity; K_{CO}: carbon monoxide transfer coefficient; and V_A: alveolar volume.

1. Pulmonary Function Laboratory and Respiratory Investigation Unit, Division of Respirology and Sleep Medicine, Kingston Health Science Center & Queen's University, Kingston (ON) Canada.
2. Unidade de Fisiologia Pulmonar, Hospital de Clínicas de Porto Alegre, Universidade Federal do Rio Grande do Sul, Porto Alegre (RS) Brasil.

department, the patient had a respiratory arrest. After endotracheal intubation, chest CT angiography showed bilateral massive pulmonary thromboembolism, as well as severe emphysema and diffuse airway plugging. After a prolonged stay in the ICU, the patient eventually died of ventilator-associated pneumonia. How could such dramatic, life-threatening abnormalities be missed by the PFTs?

Obesity may increase the expiratory flows due to increased lung/chest wall elastic recoil. FVC may underestimate slow VC because FVC is precociously "amputated" by early small airway closure in the forced maneuver, i.e., the FEV_1/FVC ratio tends to increase.[3] Although functional residual capacity decreases in comparison with that in the earlier stages of obesity,[4] volume "extremities"—RV and TLC—are only mildly affected (unless obesity is massive). It follows that expiratory reserve volume decreases and inspiratory capacity increases in tandem with BMI.[4] These changes are in opposite direction to those caused by obstruction with air trapping, leading to underestimation of or a false negative for airway disease. DLCO increases for a given alveolar volume (V_A) because lung perfusion and intrathoracic blood volume increase; moreover, V_A decreases more than does DLCO as the lung deflates. Therefore, carbon monoxide transfer coefficient (K_{CO} = DLCO/V_A) increases exponentially as V_A decreases.[5] Consequently, signs of impaired gas exchange efficiency (low DLCO and K_{CO}) might be obscured. A short height and abdominal obesity, as in our patient, tend to potentiate these effects of obesity. Chart 1 provides a non-exhaustive list of the most common pitfalls in the interpretation of PFTs in obese patients.

CLINICAL MESSAGE

BMI must be available in every PFT report: it is the third variable to look at (after age and sex) before any attempt to interpret the tests. This case illustrates that PFTs in obese patients can be relatively unaltered even in the presence of life-threatening conditions in the airways, lung parenchyma, or both. Special caution is advisable if little is known about the pre-test probability of abnormality (as is frequently the case). The final report should acknowledge these "shades of gray" rather than giving a rigid dichotomous "verdict": recognizing uncertainty always meets the best interests of the patient.

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