Reviewer's report

Title: Thoracic gas compression during forced expiration in patients with emphysema, interstitial lung disease and obesity

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Reviewer: Tomasz Golczewski

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The manuscript presents interesting experimental results which seem to confirm results that could be derived theoretically by biomedical physicists or engineers. Thus, the work could be valuable for such scientists as experimental proof of the theoretical knowledge. On the other hand, the work could be valuable as a 'direct experimental knowledge' for physicians who are not familiar with mathematics and physics, which is more important here as the Journal is a medical journal.

Unfortunately, interpretation and discussion of results are not perfect and, in consequence, they can mislead those readers who cannot interpret results by themselves.

Major Compulsory Revisions

1. The 1st sentence (Introduction) might suggest that compression is a result of forces developed by respiratory muscles and elastic properties of the lungs and thorax; but it is not the whole true because the compression is caused mainly by the forces and existence of airway resistances. For example, if there was no airway resistance i.e. if the airway resistance was equal to zero (air was superfluid), no compression would exist; only some compression-decompression oscillation related to the resonance system composed from air inertance and compliance (=compressibility) could appear.

2. It is not clear whether the term 'gas compression' means here the effect (increased pressure, compressed gas) or act (pressure alteration) of compressing. However, the discussion of results seems to suggest the first possibility, i.e. that the 'compression' means here the effect.

If it is not true, the text should be reedited to eliminate this doubt.

If it is true, treating the difference between the thoracic and mouth flows as a quantification of compression (=effect of compressing) is a mistake because this difference reflects rather changes of the compression than the compression per se. For example, if the airway resistance was infinite (airways closure), the whole thoracic flow would result in compression change; and if significantly increased alveolar pressure balanced the forces, a significant constant gas compression would still exists despite that the difference between thoracic and mouth flows
would be equal to zero (since both flows would be equal to zero).

These two fundamental aspects, i.e. (a) compression as a result of the airways resistance existence and (b) the difference between the flows as a quantification of compression alteration, are not taken into account in the discussion. Instead of that, the discussion contain several not justified statements and general ones related to well-known facts but having no direct relation to the discussed results. For example, there are description of tobacco smoking influence on the respiratory system (well-known facts not connected by the authors with their results) and the statement that ‘gas compression must be low when MEF25 is low in patients with emphysema’ (neither justified nor explained statement; moreover, usually the lower the mouth flow is because of elevated airway resistances, the greater the compression, whether it is treated as the effect or act of compressing).

3.
Presentation of “uncomfortable negative compression” proofs authors’ honesty. Unfortunately, the proposed explanation of such a compression with some imperfections in measurement procedures seems to be not very correct. On the one hand, "negative compression" exists only in the case of ILD patients (and one? healthy subject) whereas measurement procedures are the same for the all subjects (I suppose). On the other hand, such a ‘negative compression’, i.e. mouth flow bigger than thoracic flow, has to appear in some ILD patients soon after the beginning of the forced expiration (MEF75 or MEF82 or ... etc.) because of reasons mentioned above.

Minor Essential Revisions
1. The 2nd paragraph: there are 7-8 and 10-11 in references; what is it?
2. Usually, 25<BMI<30 is treated as overweight; why here ‘lean’ means BMI<30?
3. Why is COPD sometime used for the emphysema group? - e.g. in figs. 2 and 5 is COPD; the same is in the text. COPD and emphysema are not the same: COPD can be related to either bronchial obstruction or emphysema (or often to coexistence of both). Thus, emphysema is COPD but COPD is not emphysema.
4. Although the fig.1 presents results in a schematic, approximate form, it should not suggests that the ‘distance’ between PEF and MEF75 is the same as between MEF75 and MEF50 or MEF50 and MEF25, i.e. it is equal to 25% of FVC; in fact, PEF is usually close to MEF75.

Discretionary Revisions
In general, tables and figures well present the results. However: (a) why MEF75 is not presented in the table 2? (b) there is TLCb in this table while its caption contains TGVb; (c) table 3 - since the paired t-test was used, the mean, SD, and range of differences between flows could be (also) presented;

Presentation of all investigated groups and then description of methods used to determine these groups may be a little confusing. For example, the emphysema
group is presented as patients with FEV1/FVC<0.7, which is not precise since FEV1/FVC<0.7 can be caused only by bronchial obstruction. Detailed presentation of methods used to created the emphysema group is three paragraphs below. Perhaps it would be better to present each group and corresponding methods in a separate paragraph for this group.

**Level of interest:** An article of importance in its field

**Quality of written English:** Not suitable for publication unless extensively edited

**Statistical review:** No, the manuscript does not need to be seen by a statistician.

**Declaration of competing interests:**

I declare that I have no competing interests