Relationship Between Esophageal and Alveolar Pressure Variations During Occlusion

To the Editor: Failure of mouth pressure variations (ΔPm) to reflect alveolar pressure variations (ΔPA) during thoracic gas volume (TGV) measurements by Boyle’s law has been reported in asthmatic patients by Shore and Martin (1) and quite recently by Stânescu et al. (2). In both studies the conclusions were reached by comparing TGV’s obtained by relating plethysmographic volume changes (ΔVbox) to ΔPm (TGVm = ΔVbox/ΔPm) and by relating them to pressure variations in the esophagus (TGVes = ΔVbox/ΔPes), taken to represent pleural pressure variations (ΔPpl). How close ΔPpl is to ΔPA is the question. It has been discussed by Stânescu et al. (2) on the basis of a monoalveolar model featuring lung and airways properties, including the compliance of intrathoracic airway walls and the shunt impedance of the upper airways. This model study showed that for normal compliances of lung tissue (CL), alveolar gas (Cg), intrathoracic airway wall (Cw), and extrathoracic airway wall (Ce), ΔPpl was only 3% larger than ΔPA at a frequency of 2 Hz. Moreover ΔPpl/ΔPA, in contrast with ΔPm/ΔPA, was not substantially modified by increasing peripheral and central airway resistance and by decreasing the impedance of extrathoracic airways walls. Having made similar computer simulation both with the same and with a two-compartment model, I would like to add a few points.

Monoalveolar model. In static conditions, i.e., at infinitely low panting frequency, ΔPpl and ΔPA are in phase and ΔPpl/ΔPA = 1 + (Cg + Ce)/(CL + Cw). It may be seen that ΔPpl may be substantially larger than ΔPA when Cg is large and CL is small (when specific lung compliance is low, e.g., at high lung volume in normal subjects). ΔPpl/ΔPA is also directly related to upper airway wall compliance (Ce).

Although the relationship between ΔPpl and ΔPA is given by a high-order differential equation, their amplitude ratio and phase angle (φ) may be predicted to vary little with the panting frequency. In all the situations examined by Stânescu et al. (2) and up to 4 Hz, ΔPpl/ΔPA does not differ by more than 1% from its static value, as given above, and |φ| is less than 1°.

Bialveolar model. I assumed that the lung was made of two compartments arranged in parallel, each with its tissue compliance (C1, C2), gas compliance (Cg1, Cg2), and airway resistance (R1, R2). Tissue resistances and airway walls compliances were neglected. In such a model ΔPA will not be the same in the two compartments (ΔPA1 ≠ ΔPA2) except in static conditions or when their specific compliances are equal (C1/Cg1 = C2/Cg2). The accuracy of TGVes will depend on the ability of ΔPpl to reflect the weighted average of local ΔPA [ΔPA = (ΔPA1 · Cg1 + ΔPA2 · Cg2)/(Cg1 + Cg2)]. The relationship between ΔPpl and ΔPA is given by the differential equation

\[
\frac{d}{dt} \Delta P_{pl} + C \frac{d}{dt} \Delta P_{pl} = A \cdot \Delta P_{A} + B \cdot \frac{d}{dt} \Delta P_{A}
\]

where A = 1 + ((Cg1 + Cg2)/(C1 + C2)), B = (R1 + R2)/(Cg1 + Cg2)/(C1 + C2), and C = (R1 + R2) [(Cg1/Cg2)/(C1 + C2) + (Cg2/Cg1)/(C1 + C2)] + [C(Cg1/C1 + C2)]. At zero frequency the pressure drop through the resistances is nil, so that ΔPA1 = ΔPA2 and the system behaves as if it were homogeneous: ΔPpl and ΔPA are in phase, and their amplitude ratio tends toward A. At high frequency the pressure drop through the resistances becomes comparatively large, so that the system behaves as if the two compartments did not communicate through the airways: ΔPpl is again in phase with ΔPA, and their ratio is equal to B/C, which is greater than A. The larger the resistances, the sooner the high-frequency limit is reached. Between the two extremes, ΔPpl/ΔPA takes intermediate values and ΔPpl leads ΔPA. Examples of ΔPpl/ΔPA and φ at a frequency of 2 Hz are given in Table 1 for different ratios of local tissue and gas compliances and for two values of R1 + R2. It may be seen that even for strong nonhomogeneities, ΔPpl/ΔPA remains close to its static value (1.02 in the example), provided airway resistances are small. When they are high and a large part of the lung is very stiff, ΔPpl/ΔPA increases substantially with increasing frequency. Finally large-amplitude ratios may be obtained without much difference in phase.

These data support Stânescu’s opinion that in general ΔPpl will not differ much from ΔPA. Provided ΔPpl is uniform and accurately measured in the esophagus, only in patients with large resistances and severe maldistribution of lung specific compliances, or when the latter is low, may one question the validity of taking TGVes as a reference.

Table 1. Influence of maldistribution of specific compliance on the relationship between Ppl and PA during occlusion

<table>
<thead>
<tr>
<th>C1, %</th>
<th>Cg1, %</th>
<th>ΔPpl/ΔPA</th>
<th>φ,°</th>
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<tr>
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<td>R = 2</td>
<td>R = 20</td>
</tr>
<tr>
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<td>1.053</td>
</tr>
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</tr>
<tr>
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<td>1</td>
<td>1.032</td>
<td>1.040</td>
</tr>
<tr>
<td>90</td>
<td>10</td>
<td>1.046</td>
<td>1.153</td>
</tr>
</tbody>
</table>

Ppl, pleural pressure; PA, alveolar pressure; φ, phase angle; C1, tissue compliance of compartment 1 as percent of total tissue compliance; Cg1, gas compliance (or volume) of compartment 1 as percent of total gas compliance (or volume); R = R1 + R2 in cmH2O·l−1·s. In all cases C1 + C2 = 0.2 1·cmH2O−1, Cg1 + Cg2 = 0.004 1·cmH2O−1 (A = 1.02), and frequency = 2 Hz.


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REPLY

To the Editor: We have shown that in moderate-to-severe airflow obstruction, thoracic gas volume (TGVm) is overestimated by the method of DuBois et al. (1) because of the failure of mouth pressure (Pm) changes to faithfully reflect alveolar pressure (PA) changes. Overestimation of lung volume is related to the impedance of extrathoracic airways, airway resistance, and rate of panting (4-6). According to the predictions of the model we used, in healthy subjects, changes in pleural pressure (ΔPpl), as measured by variations in esophageal pressure (ΔPes), accurately reflect ΔPA and can be used, instead of ΔPm, to compute TGV (TGVes). Not only in healthy subjects, but also during induced (5) or naturally occurring (6) airflow obstruction, ΔPes [and, if extrathoracic airways are bypassed, also ΔPm (2, 5)] faithfully reflects ΔPA.

The point raised by Peslin is whether ΔPpl/ΔPA has a fixed relationship under different conditions. Based on simulations made on a one- and two-compartment model, Peslin shows that ΔPpl/ΔPA is in most cases close to unity. Only under extreme conditions, i.e., very high airway resistance and inhomogeneity of lung compliance in the two-compartment model, does this ratio increase.

The extreme cases Peslin refers to in the two-compartment model require the presence of either a very small but highly compliant compartment or two compartments of equal volume with very unequal compliances. These situations are a priori unrealistic, because both compartments, submitted to the same Ppl, would tend to modify their volume and thus their gas compliance.

Our data in severe airway obstruction, known to result in inhomogeneous distribution of lung compliance and airway resistance, show that the hypothetical cases Peslin refers to actually are not occurring. We have measured TGVes and esophageal total lung capacity (TLCes), and TGVm and TLCm, respectively, before and during induced airway obstruction and seen no decrease in either TGVes or TLCes (3, 4, 6). If during obstruction ΔPpl/ΔPA (ΔPes/ΔPA) increases, as Peslin suggests, then for a given ΔPpl (ΔPes), ΔPA, and therefore the corresponding change in plethysmographic volume (ΔVbox), would be smaller. A decrease in ΔVbox for a given ΔPpl (ΔPes) results in a decrease in TGVes and TLCes. This was never observed (3-6).

In conclusion, the hypothesis advanced by Peslin, though theoretically possible, seems to be unlikely in vivo.


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