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 Stánescu 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 Stánescu 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 (CJCg1 = CJCgz).

The accuracy will not be the same in the two compartments except in the specific case where their specific compliances are equal (CJCg1 = CJCgz). The accuracy will be low, e.g., at high lung volume in normal subjects.

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 Stánescu 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°.

Monoalveolar model. In static conditions, i.e., at infinitely low panting frequency, ΔPpl and ΔPA are in phase and ΔPpl/ΔPA = 1 + (Cg1 + CE)/(C1 + CB). It may be seen that ΔPpl may be substantially larger than ΔPA when Cg is large and CB 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).

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 Stánescu'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 compliance, or when the latter is low, may one question the validity of taking TGVes as a reference.


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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 (R = 2)</th>
<th>ΔPpl/ΔPA (R = 20)</th>
<th>φ (°) (R = 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>R = 2 R = 20</td>
<td>R = 2 R = 20</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>50</td>
<td>1.027 1.053</td>
<td>0.73 0.37</td>
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<td>50</td>
<td>1.022 1.147</td>
<td>1.35 7.75</td>
<td></td>
</tr>
<tr>
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<td>0.34 0.06</td>
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<tr>
<td>50</td>
<td>1</td>
<td>1.032 1.040</td>
<td>0.32 0.08</td>
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</tr>
<tr>
<td>90</td>
<td>10</td>
<td>1.046 1.153</td>
<td>2.91 1.40</td>
<td></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-'s. In all cases C1 + Cg1 = 0.2·1·cmH2O-' and Cg2 + Cg = 0.004·1·cmH2O-' (A = 1.02) and frequency = 2 Hz.
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 (∆Ppl), as measured by variations in esophageal pressure (∆Pes), accurately reflect ∆PA and can be used, instead of ∆Pm, to compute TGV (TGVes). Not only in healthy subjects, but also during induced (5) or naturally occurring (6) airflow obstruction, ∆Pes [and, if extrathoracic airways are bypassed, also ∆Pm (2, 5)] faithfully reflects ∆PA.

The point raised by Peslin is whether ∆Ppl/∆PA has a fixed relationship under different conditions. Based on simulations made on a one- and two-compartment model, Peslin shows that ∆Ppl/∆PA 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 ∆Ppl/∆PA (∆Pes/∆PA) increases, as Peslin suggests, then for a given ∆Ppl (∆Pes), ∆PA, and therefore the corresponding change in plethysmographic volume (∆Vbox), would be smaller. A decrease in ∆Vbox for a given ∆Ppl (∆Pes) 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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