Bronchodiilatation induced by deep breaths in relation to transpulmonary pressure and lung volume

C J Duggan, J Chan, A J Whelan, N Berend

Abstract
Induced bronchoconstriction in normal subjects can be transiently reversed by a deep breath (airway hysteresis). The mechanisms of airway hysteresis are not fully understood. The aim of these studies was to determine whether the nature of the deep breath (slow or fast inspiration, five or 10 second breath hold) affects the resultant bronchodilatation. Bronchoconstriction was induced in 10 normal subjects by inhalation of histamine until specific airway conductance (sGaw) was halved (mean (SEM) post-histamine sGaw 0-09* (0-009) s0 cm H2O-1). A subsequent deep breath to total lung capacity (TLC) increased sGaw by 57% (13%) and neither the rate of inspiration to TLC nor periods of breath holding at TLC produced a significantly different degree of bronchodilatation. Reducing the volume of the deep breath produced progressively less bronchodilatation and this was no longer significant after a breath to 68% (2%) TLC. To determine whether the volume of the deep breath or the accompanying increase in transpulmonary pressure (PstL) was responsible for the effect on sGaw, subjects were studied with an oesophageal balloon in place with and without their chest strapped. Subjects took a deep breath to a PstL of 20 cm H2O after bronchoconstriction had been induced by histamine. The degree of bronchodilatation (mean (SEM) %) was not significantly different (strap on 25 (6), strap off 36 (5)) even though significantly larger lung volumes (as % TLC) were reached with the strap off (strap on 57 (2), strap off 78 (3)). These results suggest that PstL rather than lung volume during a deep breath determines airway hysteresis.

In 1948 Melville and Caplan1 reported that a maximal lung inflation could overcome induced bronchoconstriction in dogs. Nadel and Tierney2 subsequently found a similar phenomenon in man. They found that inspiration to total lung capacity (TLC) by normal (non-asthmatic) subjects reduced induced bronchoconstriction but had no effect on resting airway resistance. Fairshter,3 however, found an increase in specific conductance (sGaw) after a breath to TLC in the absence of an induced increase in resting airway tone. These observations are evidence for airway hysteresis—that is, airway calibre will vary according to whether a particular lung volume is reached by deflation from TLC or inflation from a lower lung volume.4 In keeping with these findings in vivo, smooth muscle contraction enhances hysteresis in dog airways in vitro.5

Several potential factors that could contribute to or be responsible for airway hysteresis have been considered. These include withdrawal of vagal bronchomotor tone, which appears important in the dog67 but less so in man.8 The effect of release of bronchodilator cyclo-oxygenase products has also been discounted.1 The most plausible explanation is an effect of stretch on airway smooth muscle. Gunst and Russell9 have shown that stretching of the dog trachealis muscle results in a diminution of muscle contraction.

In the present study we assessed the effect of the magnitude of inspired lung volume on the inspiration induced bronchodilatation that followed histamine induced bronchoconstriction in normal subjects. We also assessed the effect of breath holding and rapid and slow inspiration, analogous to different conditions of stretch applied to the airway smooth muscle.2 Finally, so that we could dissociate and investigate the effects of lung volume and transpulmonary pressure (PstL), the subjects performed inspiratory manoeuvres to an identical PstL but different lung volumes by carrying out the manoeuvres with and without the chest tightly strapped. The application of a chest strap is known to increase lung elastic recoil.10-12 The mechanism is not clear but does not include airway closure.13 11 It may be related to an alteration of the pleural pressure gradient or to changes in alveolar surface properties.12

Methods
EFFECTS OF LUNG VOLUME, BREATH HOLDING, AND RATE OF INSPIRATION ON HISTAMINE INDUCED BRONCHOCONSTRICTION
Ten normal subjects (age range 21–37 years), with no history of asthma or other respiratory disease, gave informed consent to the protocol, which was approved by the medical ethics review committee of the Repatriation General Hospital, Concord.

*For conversion to SI units for sGaw: 1 cm H2O = 0·1 kPa.
Airway resistance (Raw) and thoracic gas volume (TGV) were measured in a constant volume body plethysmograph from three to five pating breaths according to the methods described by DuBois et al.\(^\text{14,15}\); specific airway conductance (sGaw) was derived from these values. This was followed by an expiration to residual volume and inspiration to TLC. The subjects then inhaled a variable number of breaths of histamine in concentrations from 8 to 32 mg/ml using a dosimeter until a decrease in sGaw of at least 30% was achieved. They were then instructed not to take a deep breath, cough, or sigh before the subsequent controlled deep breath manoeuvre, which was followed by a repeat measurement of sGaw (that is, TGV and Raw) 10 seconds later. The controlled deep breath consisted of the following series of manoeuvres: (1) inspiration to TLC (inspiratory flow at subjects’ discretion); (2) inspiration to 80% TLC, with feedback on an oscilloscope; (3) inspiration to 60–70% TLC, again with the oscilloscope (originally a volume of 60% TLC was aimed for, but this was difficult as for some subjects a tidal breath already exceeded 60% TLC); (4) inspiration to TLC with a five second breath hold at TLC; (5) inspiration to TLC with a 10 second breath hold at TLC; (6) rapid inspiration to TLC (flow rates measured with a pneumotachograph); (7) slow inspiration to TLC, again with measured flow rates.

The total duration of the experiments was 1·5–3 hours for each subject. Because of the transitory nature of histamine induced bronchoconstriction more histamine had to be administered intermittently by dosimeter to maintain the bronchoconstriction.

**CHEST STRAPPING**

The chest strapping experiments were performed with eight further volunteers at the Royal North Shore Hospital. The subjects (age range 22–35 years) had no history of asthma or respiratory disease and gave informed consent to the protocol, which had been approved by the hospital’s medical ethics review committee.

On day 1 baseline Raw, TGV, and TLC were determined in a constant volume body plethysmograph as before. After baseline studies histamine (5 g/100 ml) was administered with a hand held DeVilbiss glass nebuliser. From 10 to 30 puffs were given until Raw doubled. An inspiration to TLC with re-measurement of sGaw followed. On day 2 an oesophageal balloon catheter was inserted via the nose to mid oesophagus and connected to one end of a differential pressure transducer (Validyne ±100 cm H\(_2\)O), the other end being connected to a side tap at the mouth so that Pstt. could be recorded.\(^\text{16}\) A corset like device was then tightly fastened around the thorax and sGaw and TLC were measured. Histamine was administered as on day 1. The subjects were then instructed to take a breath to a Pstt. of 20 cm H\(_2\)O, lung volume and Pstt. being recorded on a chart recorder. Ten seconds later (timed with a stop watch from deep breath to shutter closure) sGaw was re-measured. On day 3 the above protocol was repeated without chest strapping—that is, after inhaling histamine the subjects inspired to a Pstt. of 20 cm H\(_2\)O but the lung volume reached was now substantially higher than on day 2. The order of the manoeuvres on days 2 and 3 was randomised. All manoeuvres were performed four to six times and the mean results for all the subjects were analysed.

**ANALYSIS**

Changes in sGaw and TLC after the different manoeuvres were compared by analysis of variance with multiple comparisons between groups (Student’s-Newman-Keuls test). A p value below 0·05 was accepted as significant.

### Results

All subjects had a PD\(_{20}\)FEV\(_1\) for histamine of more than 8 mg/ml. Histamine inhalation was well tolerated, with minimal flushing and hoarseness.

### EFFECTS OF LUNG VOLUME, BREATH HOLDING AND RATE OF INSPIRATION ON HISTAMINE INDUCED BRONCHOCONSTRICTION

Mean baseline (SEM) sGaw for the 10 subjects was 0·205 (0·017) s\(^{-1}\) cm H\(_2\)O,\(^{1}\). After histamine inhalation sGaw decreased significantly to 0·099 (0·009) s\(^{-1}\) cm H\(_2\)O (p < 0·001)—that is, sGaw was approximately halved. There was no significant difference in the post-histamine

### Table 1 Post-histamine specific airway conductance (sGaw; mean (SEM) values) before the inflation manoeuvres

<table>
<thead>
<tr>
<th>% total lung capacity</th>
<th>sGaw (s(^{-1}) cm H(_2)O(^{1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>100%</td>
<td>0·099 (0·008)</td>
</tr>
<tr>
<td>80%</td>
<td>0·093 (0·007)</td>
</tr>
<tr>
<td>60–70%</td>
<td>0·107 (0·011)</td>
</tr>
</tbody>
</table>

For conversion to SI units: 1 cm H\(_2\)O = 0·1 kPa.
Table 2  Chest strapping study: baseline deep breath responses in terms of specific airway conductance (sGaw) in the eight subjects

<table>
<thead>
<tr>
<th>Subject No</th>
<th>sGaw (s⁻¹ cm H₂O⁻¹)</th>
<th>After histamine</th>
<th>After deep breath</th>
<th>% Δ sGaw</th>
<th>TLC (l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0-289</td>
<td>0-097</td>
<td>0-136</td>
<td>40-2</td>
<td>7-01</td>
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<tr>
<td>2</td>
<td>0-159</td>
<td>0-078</td>
<td>0-126</td>
<td>61-5</td>
<td>8-28</td>
</tr>
<tr>
<td>3</td>
<td>0-213</td>
<td>0-089</td>
<td>0-166</td>
<td>86-5</td>
<td>7-50</td>
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<tr>
<td>4</td>
<td>0-316</td>
<td>0-112</td>
<td>0-186</td>
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<td>7-01</td>
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<tr>
<td>5</td>
<td>0-148</td>
<td>0-058</td>
<td>0-085</td>
<td>46-6</td>
<td>7-36</td>
</tr>
<tr>
<td>6</td>
<td>0-250</td>
<td>0-110</td>
<td>0-169</td>
<td>53-6</td>
<td>8-20</td>
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<tr>
<td>7</td>
<td>0-310</td>
<td>0-114</td>
<td>0-196</td>
<td>71-9</td>
<td>7-00</td>
</tr>
<tr>
<td>8</td>
<td>0-270</td>
<td>0-103</td>
<td>0-175</td>
<td>69-9</td>
<td>6-30</td>
</tr>
<tr>
<td>Mean</td>
<td>0-244</td>
<td>0-095*</td>
<td>0-159†</td>
<td>62-0</td>
<td>7-33</td>
</tr>
<tr>
<td>SEM</td>
<td>0-023</td>
<td>0-007</td>
<td>0-013</td>
<td>5-3</td>
<td>0-23</td>
</tr>
</tbody>
</table>

*Significantly different from baseline sGaw p < 0-001.
†Significantly different from post-histamine sGaw p < 0-001.
‡% change in sGaw after the deep breath to 100% total lung capacity (TLC).
Conversion to SI units: 1 cm H₂O = 0-1 kPa.

sGaw values before any of the deep breath manoeuvres (table 1). The lung volumes reached during the submaximal inspirations were 80-0 (0-8) and 68-2 (2-1)% TLC, both significantly less than TLC (p < 0-01). The rapid inspirations to TLC (peak flow 4-3 (0-4) l s⁻¹, mean inspiratory flow 2-7 (0-3) l s⁻¹) were significantly faster (p < 0-001) than the slow inspirations (peak flow 1-3 (0-2), mean inspiratory flow 0-6 (0-1) l s⁻¹).

The mean % Δ sGaw—that is, the difference between post-histamine sGaw and sGaw after each breathing manoeuvre—is plotted in figure 1. Only the bronchodilator response following a breath to 60–70% TLC differed significantly from the 100% TLC response (p < 0-05).

CHEST STRAPPING

The baseline deep breath responses are shown in table 2. Significant bronchoconstriction occurred after histamine inhalation (p < 0-001) with significantly subsequent bronchodilation following a deep breath to TLC (p < 0-001). After application of the chest strap (table 3) and inhalation of histamine sGaw did not differ from the post-histamine sGaw during the baseline responses. Inspiration to a PstL of 19-3 (0-6) cm H₂O increased lung volume to only 56-6% (1-8%) of baseline TLC. This was associated with an increase in sGaw of 25% (p < 0-01). With the chest strap off the post-histamine sGaw was slightly but significantly (p < 0-05) lower than with the chest strap applied.

Table 3  Deep breath responses in terms of specific airway conductance (sGaw) with chest strap applied in the eight subjects

<table>
<thead>
<tr>
<th>Subject No</th>
<th>sGaw (s⁻¹ cm H₂O⁻¹)</th>
<th>After histamine</th>
<th>After chest strap</th>
<th>Δ</th>
<th>% Δ sGaw</th>
<th>PstL of deep breath (cm H₂O)</th>
<th>Volume (l) of deep breath</th>
<th>% baseline TLC unstressed</th>
<th>Time (s)†</th>
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<tbody>
<tr>
<td>1</td>
<td>0-117</td>
<td>0-139</td>
<td>0-022</td>
<td>2-2</td>
<td>19-7</td>
<td>4-28</td>
<td>61</td>
<td>10-8</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0-097</td>
<td>0-103</td>
<td>0-006</td>
<td>6-9</td>
<td>20-2</td>
<td>5-25</td>
<td>63</td>
<td>8-4</td>
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<tr>
<td>3</td>
<td>0-094</td>
<td>0-141</td>
<td>0-047</td>
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<td>19-0</td>
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<td>60</td>
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<tr>
<td>4</td>
<td>0-098</td>
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<td>53</td>
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</tr>
<tr>
<td>5</td>
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<td>0-009</td>
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<td>19-8</td>
<td>4-28</td>
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<tr>
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<td>0-190</td>
<td>0-045</td>
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<td>15-9</td>
<td>3-67</td>
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<tr>
<td>8</td>
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<td>0-110</td>
<td>0-034</td>
<td>44-7</td>
<td>17-2</td>
<td>3-40</td>
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<tr>
<td>Mean</td>
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<td>0-23</td>
<td>1-8</td>
<td>0-3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from post-histamine sGaw (p < 0-01).
†From deep breath to measurement of sGaw.
Conversion to SI units: 1 cm H₂O = 0-1 kPa.
PstL—transpulmonary pressure; TLC—total lung capacity.

Table 4  Deep breath responses in terms of specific airway conductance (sGaw) without chest strap applied in the eight subjects

<table>
<thead>
<tr>
<th>Subject No</th>
<th>sGaw (s⁻¹ cm H₂O⁻¹)</th>
<th>After histamine</th>
<th>After deep breath</th>
<th>Δ</th>
<th>% Δ sGaw</th>
<th>PstL of deep breath (cm H₂O)</th>
<th>Volume (l) of deep breath</th>
<th>% baseline TLC unstressed</th>
<th>Time (s)†</th>
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<td>0-015</td>
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<td>6-35</td>
<td>77</td>
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<tr>
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<td>0-102</td>
<td>0-031</td>
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<td>5-95</td>
<td>80</td>
<td>10-8</td>
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<td>0-009</td>
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<tr>
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<td>5-2</td>
<td>0-4</td>
<td>0-34</td>
<td>2-8</td>
<td>0-3</td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from post-histamine sGaw (p < 0-001).
†From deep breath to measurement of sGaw.
Conversion to SI units: 1 cm H₂O = 0-1 kPa.
PstL—transpulmonary pressure; TLC—total lung capacity.
applied (table 4). Inspiration to a Pstl of 21-0
(0-4) cm H2O (which did not differ from the
Pstl with the chest strap applied) was
associated with an increase in lung volume to
77.6% (2.8%) baseline TLC, which was
significantly higher ($p < 0.001$) than the
volume achieved with the chest strapped. This
manoeuvre was associated with an increase in
gaw of 36% ($p < 0.001$). There was no
significant difference between the % change in
gaw with and without the chest strap ($p = 0.08$). These results are depicted graphically in
figure 2. As the post-histamine sGaw values
were different with and without the chest strap,
the absolute changes in sGaw in response to the
breath were also compared. Again, there was no
significant difference ($p = 0.43$).

**Discussion**

This study has shown that in normal subjects
deep breath induced bronchodilatation depends on the lung volume reached, being
significantly smaller at volumes below 70%
TLC. The rate of inspiration to TLC and a
period of breath holding at TLC did not
influence the degree of bronchodilatation sign-
ificantly. Finally, the chest strapping study
suggested that the increase in Pstl rather than
lung volume is responsible for the broncho-
dilatation.

We had to consider whether airway conduc-
tance or specific conductance was the more
appropriate expression of airway calibre in
these studies. None of the bronchodilator re-
sponses that followed the deep breath man-
euvres was associated with a significant
change in FRC, suggesting that either
measurement would be reasonable. sGaw was
chosen because it reduced the interindividual
variability. The statistical results using Gaw,
however, were identical in pattern to those
using sGaw.

The main potential source of error in the
experiments lay in the possibility that subjects
would fail to attain the correct volumes and
Pstl on inspiration. With appropriate instruc-
tion and visual feedback accuracy proved to be
not too difficult and we obtained values close to

those desired. The 60–70% TLC inspirations
were a little more difficult as this was quite
close to the end of normal tidal breath for many
of the subjects. It has previously been shown
that the increase in sGaw after a deep breath is
maximum at the earliest possible measurement
(about seven seconds) after the deep breath and
that it then declines non-linearly with time
until it approaches the preinflation value by 60
seconds.17,18 The measurements were therefore
timed with a stopwatch and made as close as
possible to 10 seconds after the peak of the
inspiration.

The initial part of this study clearly showed
that the change in sGaw was related to the
depth of the previous inspiration. Although
Butler et al in 19609 showed in man that lung
elastic recoil was responsible for the relation
between airway conductance and lung volume,
the emphasis subsequently has in general been
on the effects of lung volume. Bouhuys and
colleagues20 suggested that the use of partial
expiratory flow-volume curves initiated from
60% TLC may be a more sensitive index of
bronchoconstriction than measurements made
from manoeuvres begun at TLC. This tech-
nique, which has subsequently become widely
used, focuses attention on the relation of flow to
lung volume. Previous work, however, sug-
gests that Pstl and the stretch applied to airway
smooth muscle rather than lung volume is the
cause of the observed airway hysteresis.14,21

The present study clearly shows that inspira-
tion to the same Pstl with widely different lung
volumes and with a chest strap on and off
results in similar increases in sGaw. The slightly
(but not significantly) larger change in
sGaw (36% versus 25%) with the chest strap
removed raises the possibility of a type II error.
The first part of the study, however, showed
that inspiration to only 60–70% TLC does not
lead to significant bronchodilatation, yet with
the chest strap applied inspiration to only
56.6% TLC resulted in a significant increase in
sGaw. Thus the group data point to Pstl as the
determining factor and make the possibility of a
type II error less likely.

Closer inspection of the individual responses
with the chest strap applied (table 2) shows that
the subjects can be divided into two groups.
Subjects 1, 2, 5, and 7 had minimal broncho-
dilator responses whereas subjects 3, 4, 6, and 8
had responses similar to the ones they had with
the chest strap removed (table 3). We could
find no feature of these subjects to explain these
different responses. With the extreme degree of
chest constriction induced by the strap,
however, considerable chest wall and lung
distortion must have resulted. This may have
varied, depending on the chest configuration
and chest wall compliance of each subject, and
may serve to explain some of these differences
by introducing regional inhomogeneities of
transmural airway pressures.11

The effects of rate of inspiration (or rate of
change of Pstl) have been examined in vitro
and in vivo previously. Sasaki and Hoppin’s
cycled precontracted dog airways through a
range of simulated breath frequencies and
volumes. The range of frequencies was from

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**Figure 2** Percentage increase in specific airway conductance (sGaw) after inspiration to various lung volumes. Open circles indicate values without a chest strap; the closed circle indicates the value following inspiration to a transpulmonary pressure (Pstl) of 20 cm H2O with a chest strap applied; and the square indicates the value following inspiration to a Pstl of 20 cm H2O without a chest strap. Conversion to SI units: $1 \text{ cm H}_2\text{O} = 0.1 \text{ kPa}$. 

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0.0017 to 1 Hz. They showed that the degree of hysteresis of airway cross sectional area depended on cycling frequency, with the greatest hysteresis observed at a slow 0.0017 Hz. By contrast, Hida et al.22 and Beaupré and Orehek23 found greater bronchodilatation in vivo after a rapid inspiration than after a slow inspiration. This difference may well be explained by the relatively greater transmural airway pressures generated by rapid inspiration, particularly across narrowed airways. It may also explain the observation that the magnitude of the deep breath effect depends on the degree of induced airway narrowing.23 One further explanation for the difference in deep breath responses between dog and human lungs may be related to the different contributions of airway resistance and tissue viscoance. After bronchoconstriction has been induced in the dog a deep breath results in a pronounced decrease in tissue viscoance,24 whereas in man it is the airway resistance that falls.25 This could contribute to different responses at different cycling frequencies.

In this study there was a trend for the fast inspiration to produce a greater change in sGaw but this did not reach statistical significance. This may in part be due to the smaller difference between the fast and the slow flow rates in our study than in that of Hida et al.22 Because of the lack of statistical difference in sGaw responses between the slow and the fast inspirations, inspiratory flow rates were not tightly controlled in the chest strapping study. Possibly the subjects inspired more slowly with the chest strap applied, and this may also have contributed to the trend for the change in sGaw to be less with the chest strap applied. The PstL of 20 cm H2O in the strapping study was static at the end of the inspiration. Transmural airway pressures during the dynamic manoeuvre would have been higher with a fast inspiration.

Several previous studies have shown that the degree of bronchodilatation decreases with time of breath holding before expiration,24,25 though the studies of Beaupré and Orehek23 were performed in asthmatic subjects. The difference in sGaw in our study between a 5 second and a 10 second breath hold was minimal and not significant. These results are of some practical importance because a considerable variation in inspiratory flow and breath holding, within the ranges that may be found in normal individuals and those with airflow limitation, does not produce large changes in sGaw, minimising a further potential complexity in the interpretation of the results of lung function tests.

In the normal lung volume and PstL are linked by a defined pressure-volume relation. The practical importance of the findings may be in relation to conditions such as acute asthma and reactions to inhaled bronchoconstricting agents, when the pressure-volume curve may shift and inhalation to a given lung volume may result in a different PstL. This may be one reason for the different deep breath responses shown by patients with asthma.26,27

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