

Determination of peak expiratory flow

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Determination of peak expiratory flow. S. Kano, D.L. Burton, C.J. Lanteri, P.D. Sly. ©ERS Journals Ltd 1993.

ABSTRACT: It is still unknown whether peak expiratory flow (PEF) is determined by "wave speed" flow limitation in the airways. To investigate the influences of airway mechanical properties on PEF, five healthy adults performed maximal forced expiratory effort (MFEE) manoeuvres, in the standard manner and following breathholds at total lung capacity (TLC) of 2 s and 10 s.

Oesophageal pressure (Poes) was measured as an index of respiratory effort. Subjects also performed a MFEE following a 10 s breathhold during which intrathoracic pressure was voluntarily raised by a Valsalva manoeuvre, which would increase transmural pressure and cross-sectional area of the extrathoracic airway. Additional MFEEs were performed with the neck fully flexed and extended, to change longitudinal tracheal tension. In separate studies, PEF was measured with a spirometer and with a pneumotachograph.

Breathholds at TLC (2 s and 10 s), and neck flexion reduced PEF by a mean of 9.8% (sd 2.9%), 9.6% (sd 1.6%), and 8.7% (sd 2.8%), respectively, when measured with the spirometer. The same pattern of results was seen when measured with the pneumotachograph. These reductions occurred despite similar respiratory effort. Voluntarily raising intrathoracic pressure during a 10 s breathhold did not reverse a fall in PEF. MFEE manoeuvre with neck extension did not result in an increase in PEF, the group mean % changes being -3.0% (sd 5.0%).

We conclude that these results do not allow the hypothesis that "wave-speed" (\dot{V}_{ws}) is reached at PEF to be rejected. A breathhold at TLC could increase airway wall compliance by allowing stress-relaxation of the airway, thus reducing the " \dot{V}_{ws} " achievable. *Eur Respir J.*, 1993, 6, 1347-1352

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The utility of measurements of forced expiration have come from the realization that much of the expiratory limb of the maximal forced expiratory flow-volume curve is effort-independent. HYATT *et al.* [1] initially estimated that the effort independent portion of the maximum expiratory flow-volume curves is between 30-50% of vital capacity (VC). This estimation was later increased up to about 80% of VC [2, 3]. Traditionally, peak expiratory flow (PEF) has been thought to be effort-dependent, as insufficient force is available to achieve flow limitation at total lung capacity (TLC). Recently, PEDERSEN [4] has argued that the PEF is likely to match "wave-speed". According to wave-speed (\dot{V}_{ws}) theory each bronchial cross-section can develop a choke-point, and flow can not exceed the lowest flow at which the local fluid velocity equals the local \dot{V}_{ws} , *i.e.* the speed at which a pressure wave can be propagated in that airway. \dot{V}_{ws} is given by:

$$\dot{V}_{ws} = (1/\rho)^{1/2} \times (1/(dA/dP_{tm}))^{1/2} \times A^{3/2}$$

where A is airway cross-sectional area, P_{tm} is transmural pressure, dA/dP_{tm} is airway compliance, and ρ is gas density. MELISSINOS and MEAD [5] proposed that the flow-limiting mechanism resides in the trachea at high lung volumes in normal subjects, and that increased maximum flow

(\dot{V}_{max}) with neck hyperextension reflects the effect of tracheal elongation, which stiffens the trachea under dynamic conditions and increases its tube-wave speed.

If PEF were to match " \dot{V}_{ws} ", the PEF obtained from maximal forced expiratory efforts (MFEE) following breathholds at TLC would be less than that obtained following standard MFEE. Breathhold could decrease PEF by a number of mechanisms, including: allowing stress relaxation to occur in the tissues of the airway walls resulting in an increase in airway compliance (C_{aw}); decreasing elastic recoil pressure *via* stress relaxation of the pulmonary parenchyma; and altering airway calibre. If, during maximal forced expiration, the choke-point resides in the trachea, PEF should be affected by changes in mechanical properties of the trachea, such as would be expected to occur with neck flexion or extension.

To investigate these issues, five healthy adults performed MFEE in the standard manner and following breathholds of 2 and 10 s. Oesophageal pressure was measured to evaluate respiratory effort. To assess other factors which would influence PEF, subjects also performed a MFEE following a 10 s breath-hold, during which intrathoracic pressure was voluntarily raised by a Valsalva manoeuvre, to observe the influence of changes of cross-sectional area of the extrathoracic airway on PEF. Additional MFEEs were

performed with the neck fully flexed and extended to alter tracheal longitudinal tension. Studies were performed using a spirometer, as many pulmonary function tests performed for clinical reasons are performed on such instruments, and repeated using a pneumotachograph, to ensure an adequate frequency response for correctly measuring PEF.

Subjects and methods

Five normal subjects (aged 24–36 yrs) recruited from our laboratory personnel, with no history of respiratory diseases, were studied. Maximum expiratory flow-volume (MEFV) curves were collected with the subjects seated. Four types of MFEE manoeuvre were performed, in random order, as follows:

1. Standard MFEE manoeuvre ("control"). Subjects were instructed to maintain tidal breathing and inspire to TLC from an end-tidal expiration, followed immediately by a maximal exhalation to residual volume (RV), without a breathhold at TLC.
2. MFEE manoeuvre following breathhold at TLC of either 2 or 10 s ("2 s and 10 s breathhold"). During breathhold at TLC, subjects were instructed to close the glottis and to relax their respiratory muscles.
3. MFEE manoeuvre following 10 s breathhold with Valsalva manoeuvre ("Valsalva"). During 10s breathhold at TLC, intrathoracic pressure was voluntarily increased by Valsalva manoeuvre.
4. MFEE manoeuvre with neck flexion or extension ("neck flexion and extension"). MEFV curves were collected with no breathhold, with the neck fully flexed or extended, respectively, to alter the tracheal longitudinal tension.

Head position was kept constant during manoeuvres 1–3.

Measurements

Study 1 (spirometer)

Forced expiratory manoeuvres were recorded using a rolling seal spirometer (Morgan spirometer D58). The spirometer software was used to calculate PEF, forced vital capacity (FVC), expiratory flow at 50% of FVC (V_{50}), expiratory flow at 25% of FVC (V_{25}), and forced expiratory flow between 25–75% of FVC (FEF_{25-75}). During MFEE manoeuvres, pleural pressure (Ppl) was estimated from oesophageal pressure (Poes) measured relative to atmospheric pressure by the method of MILIC-EMILI *et al.* [6]. A pressure transducer (CTQH 360-5, linear ranges 0–350 cmH₂O, Kulite Semiconductor, USA) connected to a balloon system (8 cm balloon, 100 cm tubing, ID 1.4 mm), with a frequency response flat to 15 Hz with phase distortion of less than 1°, was used to measure Poes. The Poes signal was amplified (Applied Measurement, 043, Australia), low pass filtered at 10 Hz (4-pole Butterworth filters, Applied Measurement, 1260, Australia), and sampled at 200 Hz. Each manoeuvre was performed at least twice, and flow-volume curves were considered acceptable if the FVC was

within 5% of the maximum FVC obtained. To ensure that each manoeuvre was performed in a similar manner, respiratory efforts were estimated as follows: Δ Poes, calculated by subtracting baseline Poes (at end-tidal expiration) from peak Poes (fig. 1), and expired volume (%FVC) at which PEF was reached (%PEF-VOL) was calculated from flow-volume curves (fig. 2). Manoeuvres with matching respiratory effort were selected for analysis.

Study 2 (pneumotachograph)

Flow at the mouth was measured using a heated pneumotachograph (Fleisch No.4, P.K. Morgan, UK, amplitude response within 5% without phase shift to 1,000 Hz) and integrated to give volume. During MFEE manoeuvres,

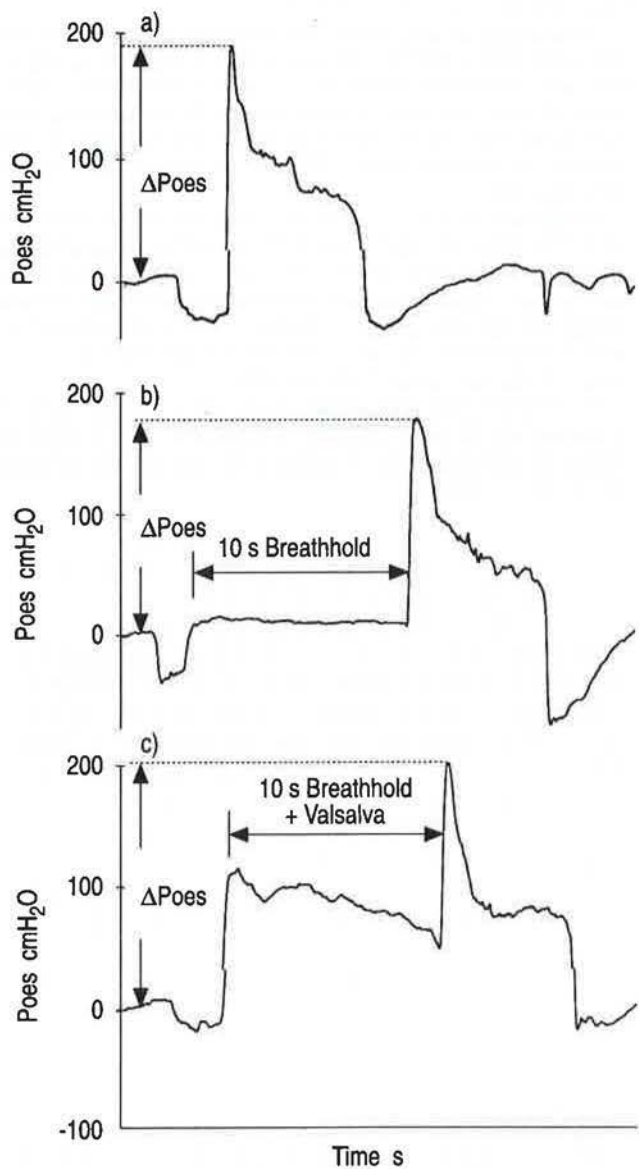


Fig. 1. — Estimation of respiratory effort from oesophageal pressure in study 1. Δ Poes: calculated by subtracting baseline Poes (at end-tidal expiration) from peak Poes. a) Control breathhold; b) 10 s breathhold; c) Valsalva manoeuvre. Poes: oesophageal pressure; Ppl: pleural pressure.

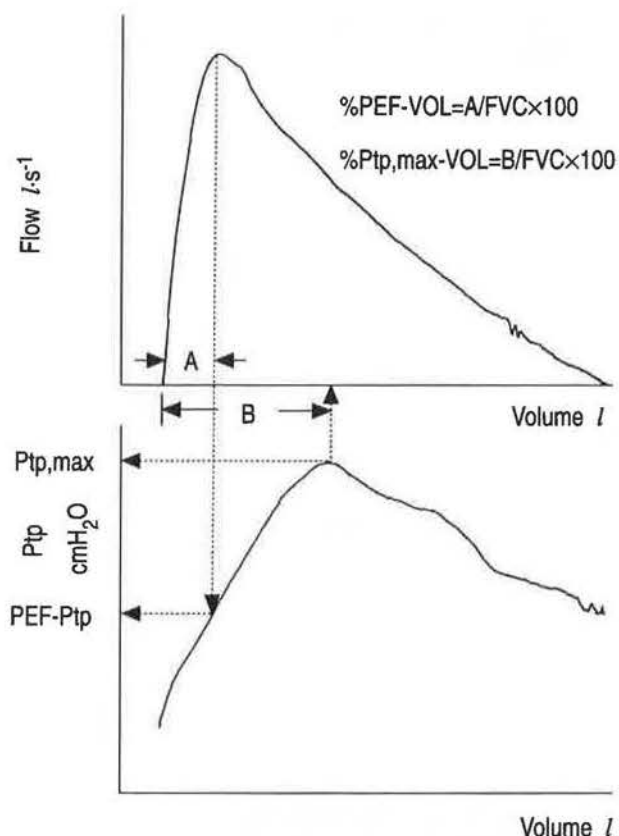


Fig. 2. - Estimation of respiratory effort from flow-volume curve and transpulmonary pressure-volume curve. %PEF-VOL: expired volume (%FVC) at which PEF was reached; %Ptp,max-VOL: expired volume (%FVC) at which Ptp reached maximum. PEF-Ptp: transpulmonary pressure at PEF; Ptp,max: maximum transpulmonary pressure; FVC: forced vital capacity; PEF: peak expiratory flow.

Table 1. - Estimation of respiratory efforts for maximal forced expiratory manoeuvre performed with the spirometer

	Control	2 s-BH	10 s-BH	Valsalva	Neck flexed
Δ Poes cmH ₂ O	158.1 (± 48.6)	162.0 (± 54.4)	156.2 (± 43.6)	150.7 (± 54.3)	162.4 (± 58.7)
%PEF-VOL	14.5 (± 1.5)	15.0 (± 2.3)	14.1 (± 0.7)	14.0 (± 2.1)	13.2 (± 1.8)

Data are presented as mean (\pm SD) in parenthesis. No significant changes from control (no breathhold) were observed. 2 s-BH: 2 s breathhold; 10 s-BH: 10 s breathhold, Δ Poes: difference between peak oesophageal pressure and baseline oesophageal pressure; %PEF-VOL: expired volume (%FVC) at which peak expiratory flow was reached; FVC: forced vital capacity.

Poes and airway opening pressure (Pao), measured proximal to the pneumotachograph, were measured, thus providing simultaneous recordings of transpulmonary pressure (Ptp = Pao - Poes) and flow. Poes was measured by the method used in study 1. Before measurement of each manoeuvre, occlusion tests were performed to validate Poes [7]. Each signal was amplified (Validyne, CD15, Northridge, Ca,

USA, for flow and Pao; Applied Measurement, 014, Australia, for Poes), low pass filtered at 100 Hz (Applied Measurement, 1260, Australia) and sampled at 200 Hz. Respiratory efforts were estimated as follows: %PEF-VOL, as for study 1, expired volume (%FVC) at which Ptp reached maximum (%Ptp,max-VOL), transpulmonary pressure at PEF (Ptp-PEF), and maximum transpulmonary pressure (Ptp,max) (fig. 2). Each measurement was performed three times and averaged for analysis.

The signals were processed using Anadat and Labdat software (RHT Info-dat, Montreal, Canada). Paired t-tests were used to test for significant changes with each manoeuvre. Statistical significance was accepted at the 5% level.

Results

Spirometer

Table 1 shows the estimation of respiratory efforts in each manoeuvre. There were no significant differences in either Δ Poes or %PEF-VOL between manoeuvres. No significant changes were observed in FVC between manoeuvres (table 2). Breathhold (either 2 or 10 s) and neck flexion reduced PEF significantly from control by a mean of 9.8% (SD 2.9%), 9.6% (SD 1.6%) and 8.7% (SD 2.8%), respectively, (table 2 and fig. 3). With a Valsalva manoeuvre, a significant decrease in PEF was also observed (table 2, fig. 3); i.e. voluntarily raised intrathoracic pressure during 10 s breathhold did not reverse the fall in PEF seen with the breathhold, suggesting that the extrathoracic airway did not play a part in this. There were no significant changes in \dot{V}_{50} , \dot{V}_{25} , and FEF₂₅₋₇₅ with any manoeuvre (table 2).

Pneumotachograph

Table 3 shows FVC and the estimation of respiratory efforts for each manoeuvre recorded with the pneumotachograph. No significant changes in FVC and %PEF-VOL from control were observed. With neck flexion, small but statistically significant falls in %Ptp,max-VOL were seen. With both the Valsalva and neck extension manoeuvres, Ptp,max decreased significantly from a mean of 156.3 cmH₂O (control) to a mean of 133.7 cmH₂O and 131.6 cmH₂O, respectively. The transpulmonary pressure at PEF (PEF-Ptp) was considerably lower than the maximal transpulmonary pressure (Ptp,max), and occurred at higher lung volume, indicating that the driving pressure continued to increase after PEF was reached, as indicated in figure 2. It was not lower than control with any of the MFEE manoeuvres, in fact PEF-Ptp tended to be higher, reaching significance for the 2 s breathhold and neck flexion manoeuvres (table 3).

PEF data recorded with the pneumotachograph are also shown in table 3. Individual coefficients of variation for PEF from three consecutive measurements were less than 5%, except for one subject with the Valsalva manoeuvre (CV 11.9%). The changes in PEF followed the same pattern as seen with the spirometer (fig. 3), i.e., 2 s breathhold, 10 s breathhold and neck flexion decreased PEF by a

Table 2. – Peak expiratory flow (PEF) and flow-volume parameters from each manoeuvre performed with the spirometer

	Control	2 s-BH	10 s-BH	Valsalva	Neck flexed
PEF $l \cdot s^{-1}$	11.62 (± 2.26)	10.48* (± 2.05)	10.53** (± 2.19)	10.25* (± 2.05)	10.61* (± 2.07)
FVC l	5.07 (± 0.67)	5.01 (± 0.66)	4.98 (± 0.63)	4.98 (± 0.65)	4.92 (± 0.62)
\dot{V}_{50} $l \cdot s^{-1}$	5.13 (± 1.11)	4.96 (± 1.01)	5.00 (± 0.91)	5.00 (± 1.02)	5.08 (± 0.84)
\dot{V}_{25} $l \cdot s^{-1}$	2.27 (± 0.42)	2.17 (± 0.41)	2.15 (± 0.39)	2.24 (± 0.60)	2.15 (± 0.60)
FEF ₂₅₋₇₅ $l \cdot s^{-1}$	4.00 (± 0.69)	3.90 (± 0.67)	3.83 (± 0.57)	3.91 (± 0.69)	3.92 (± 0.74)

Data are presented as mean (\pm SD) in parenthesis. Statistically significant differences from control (paired t-test: *: $p < 0.01$; **: $p < 0.001$. \dot{V}_{50} : forced expiratory flow at 50% FVC; \dot{V}_{25} : forced expiratory flow at 25% of FVC; FEF₂₅₋₇₅: forced expiratory flow between 25–75% of FVC. For further abbreviations see legend to table 1.

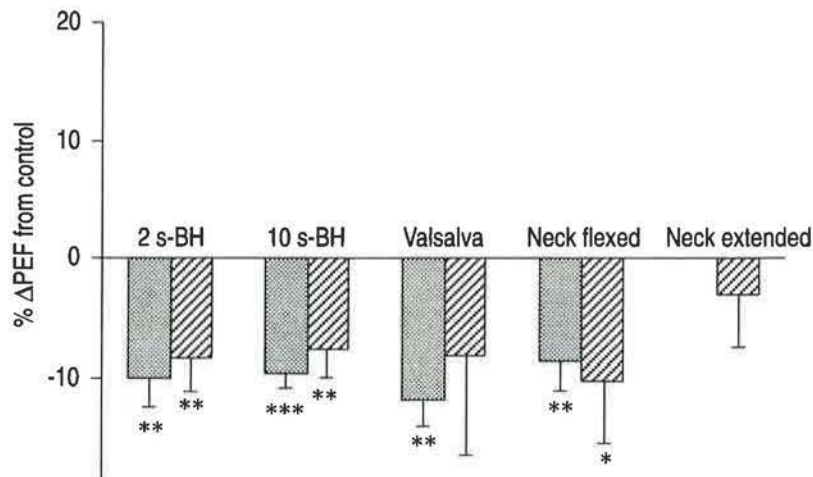


Fig. 3. – Percentage in group mean PEF from standard manoeuvre (control). Breathhold (BH) and neck flexion decreased PEF significantly. Neck extension and Valsalva manoeuvre did not result in an increase in PEF. There were no significant differences between 2 and 10 s breathholds. ■■■: Study 1; ▨▨▨: Study 2. Statistically significant difference from control: *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$. Error bars indicate SD.

Table 3. – Peak expiratory flow (PEF), forced vital capacity (FVC) and respiratory effort for each manoeuvre performed with the pneumotachograph

	Control	2 s-BH	10 s-BH	Valsalva	Flexed	Extended
PEF $l \cdot s^{-1}$	11.52 (2.45)	10.52* (2.13)	10.60* (2.11)	10.71 (2.99)	10.35* (2.34)	11.15 (2.35)
FVC l	5.09 (0.63)	5.03 (0.61)	5.13 (0.64)	5.12 (0.69)	5.04 (0.64)	5.03 (0.58)
*PEF-VOL	10.9 (1.0)	12.3 (2.0)	11.4 (1.3)	10.2 (1.8)	10.5 (1.4)	11.8 (1.8)
%Ptp,max-VOL	41.9 (5.3)	42.5 (9.6)	47.5 (9.6)	37.8 (3.8)	38.9** (5.8)	42.0 (6.7)
PEF-Ptp cmH_2O	48.6 (14.6)	74.0* (15.3)	65.4 (29.2)	77.9 (35.5)	62.8* (22.8)	48.3 (23.9)
Ptp,max cmH_2O	156.3 (39.6)	146.8 (32.6)	148.6 (43.0)	133.7** (35.8)	151.5 (39.8)	131.6** (39.7)

Data are presented as mean (\pm SD) in parenthesis. Statistically significant difference from control: *: $p < 0.05$; **: $p < 0.001$. %Ptp,max-VOL: expired volume (%FVC) at which transpulmonary pressure was maximal; PEF-Ptp: transpulmonary pressure at PEF; Ptp,max: maximum transpulmonary pressure. For further abbreviations see legends to tables 1 and 2.

mean of 8.4% (SD 3.0%), 7.6% (SD 2.9%), and 10.3% (SD 6.1%), respectively. Valsalva and neck extension did not result in a significant increase in PEF.

Discussion

The importance of the mechanical properties of the airways in the determination of maximal flow has long been recognized [2, 8–10]. JONES *et al.* [11, 12] proposed that maximal flow is dependent on the compliance of the flow limiting segment. DAWSON and ELLIOTT [13] proposed the "wave-speed" (\dot{V}_{ws}) theory, *i.e.* flow in a compliant tube could not exceed the lowest flow at which the fluid velocity equalled the local wave speed, and expiratory flow limitation could be explained by " \dot{V}_{ws} ". MELISSINOS *et al.* [5] reported that the flow limiting segment resides in the central airways at high lung volume in normal subjects, and that neck hyperextension resulted in increased \dot{V}_{max} , due to a decrease in tracheal compliance.

Traditionally, PEF has not been thought to be flow-limited because a plateau is not seen on iso-volume pressure flow (IVPF) curves, presumably because of inability of the respiratory muscles to generate sufficient force. Recently, PEDERSEN [4] argued that PEF is likely to match \dot{V}_{ws} if forced expiratory flow has a well-defined peak. He calculated \dot{V}_{ws} from measurements of dA/dP_{tm} and cross-sectional area (A), measuring intrabronchial pressure using a pitot static probe. He proposed that, using a speed index (SI) defined as the actual velocity (\dot{V}/A) divided by the \dot{V}_{ws} (\dot{V}_{ws}/A), PEF is just limited with a SI of 1 in the central airways. This does, not, however, mean that PEF is independent of effort. The magnitude of PEF depends on how this maximal flow is reached. If expired volume from TLC (%FVC) at which PEF is reached (%PEF-VOL) is small, PEF will be higher, because, at higher lung volume, the higher elastic recoil pressure and lower upstream resistance result in a greater \dot{V}_{ws} and a higher PEF. In any interpretation of changes in PEF, the magnitude of effort and the volume at which PEF is reached is critical. The values of %PEF-VOL achieved in the present study ranged from 10 to 15%. No significant differences were observed between the various manoeuvres performed. Therefore, it is likely that each manoeuvre was performed with similar effort, and that the changes in PEF reported here are not due to the manner in which the MFEE was recorded.

The studies were initially performed using a spirometer to determine whether clinically significant reductions in PEF would be obtained with an instrument in every day clinical use. The 10% reduction in PEF seen with breathholds at TLC is clinically relevant, and likely to be encountered in clinical practice. The changes in PEF measured with the pneumotachograph were similar to those seen with the spirometer, suggesting that these changes are real and not an artifact of the measurement equipment.

The results of the present study are consistent with an increase in C_{aw} during breathhold at TLC, possibly by allowing stress-relaxation to occur in the tissues of the airway walls. COBURN and PALOMBINO [14] reported the presence of stress relaxation in the airways, the magnitude of which was increased after stimulation of the cervical vagosympathetic trunk. Also, SASAKI and HOPPIN [15] measured

bronchial pressure-volume relations under various conditions, to observe the parenchymal effect on dynamic bronchial compliance. They found that the presence of lung parenchymal attachments reduced dynamic collapsibility more than static collapsibility. When the airway was separated from parenchymal attachments, dynamic compliance was still less than static compliance. These results may be explained by viscoelasticity of the airway tissues. Thus, it is feasible that C_{aw} may be increased during breathhold, as time is allowed for stress-relaxation to occur. However, a breathhold at TLC may have other effects which would influence PEF. End-inspiratory breathhold is known to decrease elastic recoil, by allowing stress-relaxation of the pulmonary parenchyma. This would result in a lower driving pressure and a lower PEF, if \dot{V}_{ws} is reached at PEF. Also, a breathhold at TLC may be expected to decrease airway calibre, by allowing the "distending" influence of the previous deep inspiration to "wear off" prior to the MFEE manoeuvre being performed.

Several authors have previously studied the time dependence of flow volume curves. GREEN and MEAD [16] reported that breathhold produced a fall in maximal flow at a fixed lung volume in partial expiratory flow volume (PEFV) curves, but not in maximal expiratory flow volume (MEFV) curves. HIGENBOTTAM and CLARK [17] reported that a 15 s breathhold caused reductions in maximal flow both in PEFV curves and MEFV curves. Interestingly, they also measured PEF, which decreased from a mean value of 11.18 ($l \cdot s^{-1}$) without breathhold to 10.28 ($l \cdot s^{-1}$) with 15 s breathhold, consistent with the results of the present study. These results, however, could be explained by an action on either airway or lung tissues. GREEN and MEAD [16] speculated that the main mechanism of reducing maximal flow was related to increased airway resistance, because the reduction in maximal flow with time was abolished after nebulized isoproterenol.

Recently, D'ANGELO *et al.* [18] reported that breathhold significantly reduced expiratory flow at any given lung volume. They proposed that this time dependency might be explained by the presence of viscoelastic elements within the pulmonary tissues; *i.e.* expiratory flow was increased by an increase in the effective elastic recoil pressure with no breathhold manoeuvre because of stretching of the viscoelastic element. However, end-inspiratory breathhold results in a decrease in elastic pressure from dynamic to static, which would be expected to reduce effective driving pressure, reducing expiratory flow at any given lung volume. Stress relaxation of the airway tissues would also explain these results, by increasing C_{aw} and allowing more dynamic airway compression during the MFEE manoeuvre.

In the present study, the MFEE manoeuvre performed with neck flexion resulted in a decrease in PEF. Neck flexion results in a decrease in longitudinal tracheal tension, which could increase tracheal compliance. Thus, this reduction in PEF is consistent with the hypothesis that \dot{V}_{ws} is reached at PEF and that the flow limiting segment resides in the trachea. Alternatively, neck flexion may increase extrathoracic airway resistance [19], which may shift the flow-limiting segment proximally and, thus, alter PEF. Although we expected that MFEE manoeuvres performed with the neck extended would increase PEF,

as has been reported previously [5], we were not able to demonstrate this. This may be explained by failure to decrease tracheal compliance sufficiently. Alternatively, neck extension may have resulted in a decrease in tracheal cross-sectional area, due to longitudinal traction masking the expected increase in PEF.

During a Valsalva manoeuvre intra-airway pressure increases as Ppl increases. The Ptm of the intrathoracic airway should not be altered, because alveolar pressure equals intra-airway pressure in this circumstance. However, the change in Ptm occurring in the extrathoracic airway should result in an increase in cross-sectional area and a decrease in upper airway resistance. If \dot{V}_{ws} is achieved at PEF and the choke-point resides in the intrathoracic airway, PEF would not be affected by the mechanical properties of the extrathoracic airway (upper airway). In the present study, a Valsalva manoeuvre during a 10 s breathhold did not increase PEF. This suggests that flow-limitation is achieved, and that the flow limiting site resides in the intrathoracic airway.

Finally, the demonstration that the Ptp,max far exceeds the Ptp recorded at the volume at which PEF occurs (table 3) suggests that flow-limitation is achieved at PEF. Whilst the true driving pressure is equal to Ptp plus the static elastic recoil at that volume, Ptp,max exceeded PEF-Ptp by 70–100 cmH₂O in all cases. The effort-dependence of PEF can be explained as follows: if the driving pressure that produces PEF (i.e. PEF-Ptp) is higher, PEF will be reached at a higher lung volume (i.e. %PEF-VOL will be smaller), resulting in a higher PEF as \dot{V}_{ws} increases with lung volume.

In conclusion, we have demonstrated that MFEE manoeuvres performed following breathhold, and with neck flexion, resulted in a fall in PEF. This fall is consistent with an increase in Caw, secondary to stress relaxation of the trachea and changes in longitudinal tension, respectively, reducing the \dot{V}_{ws} achievable. Based on these results, one can not reject the hypothesis that \dot{V}_{ws} is reached at PEF.

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