Eur Respir J
1991, 4, 180–187

Thoracic gas volume at functional residual capacity measured with an integrated-flow plethysmograph in infants and young children

F. Marchal*, C. Duvivier**, R. Peslin**, P. Haouzi*, J.P. Crance*

Thoracic gas volume at functional residual capacity measured with an integrated-flow plethysmograph in infants and young children. F. Marchal, C. Duvivier, R. Peslin, P. Haouzi, J.P. Crance.
ABSTRACT: Thoracic gas volume (TGV) was measured with an integrated flow plethysmograph in 15 infants aged 2–34 months. End-expiratory (TGVe) and end-inspiratory (TGVi) airway occlusions were compared, after correction of TGV for the occluded volume above functional residual capacity (FRC). The relationship between pressure at the airway opening (Pao) and volume displaced from the box during airway occlusion (Vg) was studied numerically by: 1) an algorithm including a correction for the drift of Vg and linear regression analysis (LR); and 2) Fourier analysis of the signals (FFT). TGVe was significantly higher than TGVi (256 *vs* 237 ml, 20.4 (square root of residual variance; p<0.002). The correlation coefficient of the Pao-Vg relationship was slightly but significantly higher for TGVi than for TGVe: 0.9968 (0.9937–0.9995) *vs* 0.9947 (0.9840–0.9990) (means and range). No difference was observed between LR and FFT, although the intra-individual coefficient of variation was lower for LR than FFT: 5.2% (1.6–11.3) *vs* 7.9% (1.9–21.0) (means and range). Model simulations suggested that the difference between TGVe and TGVi could be mainly attributed to gas compression in the instrumental deadspace and upper airway wall motion and/or to uneven distribution of alveolar and pleural pressure associated with chest wall distortion.
Eur Respir J., 1991, 4, 180–187.

* Laboratoire d'Explorations Fonctionnelles Pédiatriques, Hôpital d'Enfants, Centre Hospitalier Universitaire de Nancy, France.

** Unité 14 INSERM de Physiopathologie respiratoire, F-54500, Vandoeuvre-lès-Nancy, France.

Correspondence: F. Marchal, Laboratoire de Physiologie, Faculté de Médecine de Nancy, B.P. 184-F-54505 Vandoeuvre-lés Nancy cedex, France.

Keywords: Body plethysmography; gas compliance; infants; mechanical inhomogeneity; thoracic gas volume; uneven pleural pressure; upper airway wall motion.

Received: February 1, 1990; accepted after revision August 9, 1990.

In infants, thoracic gas volume (TGV) is usually measured in a pressure type plethysmograph [1–6]. Thoracic gas volume changes during respiratory efforts against occluded airways are measured by the corresponding pressure variation in the plethysmograph. These respiratory efforts occur at a spontaneous breathing rate (*i.e.* 0.4–0.6 Hz). In this range of frequency, the box pressure-volume relationship may be frequency dependent according to the relative magnitude of the period of the manoeuvre and of the box thermal time constant [7]. The calibration must then be performed at the same frequency as that adopted by the subject, as from isothermal to adiabatic compression, the box pressure-volume ratio is affected by a factor ranging from 1–1.4. However, the frequency content of the thoracic gas volume change during occluded breaths may vary between and within measurements. An alternative is to use a flow-type plethysmograph. The latter is particularly suited for studying slow events because the amount of gas compression is small. Furthermore, gas compression only influences the box frequency response, and that effect may be corrected for [7].

The drift of the volume signal represents a major drawback when TGV is read graphically from a pressure-volume plot, particularly with an integrated flow plethysmograph, as the small flow signal requires high amplification. The numerical transformation of airway pressure (Pao) and box volume (Vg) signals, allows the calculation of TGV by linear regression analysis of Pao and Vg including algorithms compensating for the drift [8]. An alternative is to use fast Fourier transforms of Pao and Vg, in order to determine their amplitude ratio and phase angle. The latter may give useful additional information in evaluating the validity of the measurement. Both methods yield similar results in adult subjects [8].

Previous studies have shown that, in infants, the value of TGV at functional residual capacity (FRC) may vary according to the lung volume at which the occlusion is performed [4, 5]. It has therefore been suggested that measurements of TGV should be obtained by occluding the airways both at end-expiration and end-inspiration [5].

The objective of this study was to assess the validity of measurements of TGV in infants using a flow plethysmograph. The resulting Pao-Vg relationships were studied by linear regression analysis (LR) and fast Fourier transform (FFT) and the values obtained by end-expiratory (TGVe) and end-inspiratory (TGVi) occlusions were compared.

Material and methods

Patients

Nine boys and 6 girls aged 2–34 months were studied during sleep induced by chloral hydrate (70 $mg \cdot kg^{-1}$). Measurements were performed during a morning session as the patients were referred to the laboratory for lung function tests. The individual biometric characteristics and diagnosis are presented in table 1. All infants were clinically stable and no infant recovering from bronchiolitis had wheezing at the time of the study.

Table 1. – Infant data

Subject	Sex	Age mth	Height cm	Weight kg	Diagnosis
1	M	5	67.5	7.4	Pneumonia
2	M	10	68	8.5	BMT
3	F	2	56	4.6	Apnoea
4	M	12	68	7.5	Bronchiolitis
5	M	2	55	4.2	Bronchiolitis
6	F	5	53	4.0	CLD
7	F	27	88	14.2	WS
8	F	24	78	9.2	CLD
9	F	4	61.5	6.5	Bronchiolitis
10	F	20	79	12.0	Pneumonia
11	M	17	73.5	9.7	Pneumonia
12	M	28	88	10.0	BMT
13	M	34	92.5	13.0	Pneumonia
14	M	9	71	13.0	Bronchiolitis
15	M	8	63	6.2	CLD

BMT: bone marrow transplantation; CLD: chronic lung disease; WS: white spirit intoxication.

Equipment

The plethysmograph was a home-made 80 *l* plexiglas box open to the atmosphere through a wire mesh screen of negligible inertance. The pressure at the airway opening was measured with a differential pressure transducer (Celesco LCVR ±50 cmH_2O). The time constant of the plethysmograph (τ), the product of gas compliance by screen resistance, was determined by applying to the box a sinusoidal flow generated by a loudspeaker from 2 to 20 Hz. The input flow was measured by a pneumotachograph calibrated by the integral method and corrected for its 2 ms time constant [9]. The pressure drop across the pneumotachograph (input flow) and the pressure in the box (output flow) were measured with identical pressure transducers (Celesco LCVR ±2 cmH_2O) that were matched within 1% of amplitude and 2° of phase up to 30 Hz.

The phase angle (ϕ) between input and output flow was calculated by Fourier analysis of the signals. At each frequency (*f*), the time constant was given by:

$$\tau = \tan \phi / \omega \qquad (1)$$

where ω is angular velocity ($2\pi f$) [7]. Measurements were repeated after adding known incompressible volumes to the box in order to determine τ under different values of gas compliance. This allowed to correct τ according to the infant's weight during the measurement of TGV. The time constant of the empty box was 80 ms and independent of frequency from 2–16 Hz. The screen flow signal ($\dot{V}$) was integrated to volume (V) which was corrected for τ, so that at any instant (t), the corrected volume (Vg) was:

$$Vg(t) = V(t) + \tau \cdot \dot{V}(t) \qquad (2)$$

The accuracy of the plethysmograph was tested by measuring the volume of a chamber compressed by a loudspeaker placed inside the box and comparing this value with that obtained by the volume displacement method. Both methods were found to agree within 5% of each other.

Measurements

The routine calibration of the plethysmograph was performed in the following way. A small pump varied the volume of the empty box at 1 Hz through the unheated pneumotachograph. τ was set at 80 ms and the gain of the box volume signal was then adjusted to obtain a slope of 1 for the box volume-pneumotachograph volume relationship. The time constant of the box was then modified according to the infant's weight.

The infant was then placed in the plethysmograph and a rigid face mask was applied on the nose and mouth and held in place with rubber straps. The face mask was connected to the pneumotachograph-occlusion system assembly. The occlusion system consisted of a pneumatically operated valve placed at the distal end of the pneumotachograph. The airtightness of the mask seal was checked by ensuring that airway pressure plateaued during airway occlusion at end-inspiration. The total added volume of gas (including the transducers connecting tubes) compressed during a TGV manoeuvre was 20 ml. However, as the mask could be moulded around the infant's nose and mouth, the effective deadspace during the measurement was probably lower.

For each infant, four measurements were performed by occluding the airways at end-expiration (TGVe) and four at end-inspiration (TGVi), in a random order. Box volume, airway pressure, ventilatory flow and volume were fed into a microcomputer and digitized at 100 Hz for 2–3 s, according to the infant's breathing rate, thus including 2–4 occluded ventilatory efforts for each measurement.

TGV was calculated by linear regression of airway pressure on box volume, using a previously described algorithm correcting for the drift of the volume signal (TGVr) [8] and the correlation coefficient (r) was calculated. The numerical data were stored on disk for later analysis by fast Fourier transform of volume and pressure allowing the calculation of their amplitude ratio (TGVf) and phase angle (ϕ) at the harmonic with the largest amplitude which probably corresponds to the fundamental frequency. The respiratory cycle immediately preceding the occlusion was used to define the end-expiratory position. TGVi was corrected for tidal volume. A similar correction was also made on TGVe when the occlusion did not occur exactly at end-expiration. Hence, after correction, both TGVi and TGVe should estimate TGV at FRC. Tidal volume was measured by the heated pneumotachograph and the calibration factor of the unheated pneumotachograph was used for measuring inspired tidal volume. It was found that this factor increased by about 10% when the flowmeter was heated, so that no correction to BTPS condition was done.

In 14 infants, the mean airway pressure (MAP) during the occlusion was also calculated, in order to assess its effect on TGV computation [10], according to:

$$TGV = Vg\ (PB + MAP - P_{H_2O})/\Delta P \qquad (3)$$

Although the error involved is likely to be small, there may be a systematic difference in MAP between end inspiratory and end-expiratory occlusions. This correction was performed on TGVr.

Data analysis

A multi-factor analysis of variance (ANOVA) for repeated measures was used to assess the effect of each type of occlusion and calculation among subjects on TGV. Wilcoxon's signed rank test was used to compare mean airway pressure, regression coefficient and phase angle of the Pao-Vg relationship for expiratory and inspiratory manoeuvres. This test was also used to compare the coefficient of variation of TGVe and TGVi and of TGVf and TGVr. The coefficient of variation of TGV was calculated as the standard deviation to mean ratio percent.

A difference was considered as significant at a p value lower than 5%.

Results

The individual mean value and coefficient of variation of TGVi and TGVe obtained by FFT, LR and LR after correction for MAP (LRc) are reported in table 2. The coefficient of variation of the latter estimate of TGV is not reported as the value was virtually identical to that of LR. The analysis of the effect of type of calculation and occlusion on TGV is reported in table 3A. In addition to the expected difference among individuals, the ANOVA table shows that end-inspiratory occlusions yield significantly lower values of TGV than end-expiratory occlusions. The significant interaction indicated in table 3A suggests that the magnitude of the difference between TGVe and TGVi varies among

Table 2. – Individual mean data on TGV calculated by Fourier analysis, linear regression and LR corrected for MAP

Subject	TGVe			TGVi		
	FFT	LR	LRc	FFT	LR	LRc
1	185 (5.4)	180 (4.7)	-	177 (21.0)	157 (10.4)	-
2	244 (2.2)	223 (3.9)	221	241 (2.6)	245 (1.6)	247
3	111 (15.0)	111 (5.7)	110	89 (19.5)	95 (6.0)	94
4	237 (3.6)	240 (2.1)	238	196 (7.2)	204 (9.0)	204
5	122 (9.4)	117 (10.8)	116	116 (6.2)	113 (7.7)	113
6	136 (14.1)	132 (8.7)	131	99 (7.8)	103 (5.3)	103
7	362 (2.9)	362 (1.9)	357	316 (6.7)	322 (2.2)	333
8	373 (6.7)	371 (3.2)	368	345 (1.9)	345 (3.0)	347
9	170 (6.3)	169 (2.9)	167	159 (11.1)	156 (5.1)	157
10	278 (5.5)	287 (2.7)	285	268 (4.0)	264 (2.2)	266
11	238 (6.0)	235 (3.8)	233	230 (5.6)	229 (4.2)	231
12	400 (7.8)	370 (10.1)	366	372 (7.1)	376 (1.9)	376
13	457 (5.1)	430 (4.3)	428	394 (11.2)	386 (6.9)	389
14	338 (3.0)	347 (2.7)	342	356 (3.0)	362 (3.0)	361
15	227 (16.3)	224 (11.3)	223	196 (12.5)	203 (6.6)	204
Overall mean: (1)	258* (7.3+)	253* (5.3)		237 (8.5+)	237 (5.0)	
(2)	-	258	256	-	243	245

Numbers in brackets are coefficients of variation. (1) mean TGV for 15 subjects. *: significantly higher than corresponding TGVi; see table 3A. +: coefficient of variation significantly higher than corresponding LR value ($p<0.02$). (2) mean TGV for 14 subjects; see table 3B. TGV: thoracic gas volume (expiration and inspiration, respectively); FFT: Fourier analysis; LR: linear regression; LRc: LR corrected for mean airway pressure (MAP); TGVe and TGVi: end-expiratory and end-inspiratory airway occlusions, respectively.

Table 3. – Analysis of variance on TGV calculated by FFT and LR (A), and corrected for mean airway pressure (B)

A Source of variation	df	Variance	F ratio	p value
Subject	14	169491	407.6	0.0001
Occlusion	1	20948	50.4	0.0001
Interaction	14	1424	3.4	0.0002
Error	90	416		
FFT - LR	1	389	2.4	0.12

B Source of variation	df	Variance	F ratio	p value
Subject	13	172106	460.0	0.0001
Volume of occlusion	1	10308	27.6	0.0001
Interaction*	14	1540	4.1	0.0001
Error	84	374		
LR - LRc	1	2.0	0.3	0.6
Interaction+	1	193	28.9	0.0001

Only significant interactions are reported: *: significant interaction between "occlusion" and "subject" factors; +: significant interaction between "occlusion" and "treatment (*i.e.* correction for mean airway pressure)" factors; df: degree of freedom; TGV: thoracic gas volume; FFT: Fourier analysis; LR: linear regression.

Table 4. – Individual mean data on phase angle and correlation coefficient of the Vg-Pao relationship during the occlusion and of mean airway pressure during the occlusion

Subject	End-expiration			End-inspiration		
	ϕ °	r	MAP cmH_2O	ϕ °	r	MAP cmH_2O
1	-5.4	0.9935	-	-6.5	0.9950	-
2	+0.4	0.9975	-8.6	-2.0	0.9982	5.2
3	-4.4	0.9904	-6.4	-3.1	0.9947	5.3
4	-2.6	0.9953	-8.0	-3.4	0.9986	-0.2
5	-3.0	0.9933	-7.9	-4.7	0.9939	1.8
6	-10.8	0.9840	-4.8	-4.7	0.9941	0.7
7	-3.9	0.9990	-10.3	-1.5	0.9984	2.5
8	-5.9	0.9970	-6.8	-2.0	0.9969	3.2
9	+3.3	0.9968	-9.7	-3.6	0.9975	1.9
10	-3.1	0.9956	-6.6	-2.8	0.9994	4.6
11	+2.6	0.9960	-6.2	-1.7	0.9973	5.7
12	-0.5	0.9963	-9.1	-4.0	0.9964	1.3
13	-4.9	0.9989	-2.3	-4.1	0.9995	4.6
14	+0.5	0.9985	-11.3	-2.2	0.9985	-2.2
15	-0.9	0.9886	-3.6	-4.2	0.9937	2.3
Overall mean:	-2.6	0.9947*	-7.2*	-3.4	0.9968	2.6

*: significantly lower than corresponding end-inspiratory value, $p<0.01$; MAP: mean airway pressure.

infants. Finally, similar values of TGV were obtained by LR and FFT.

The correction for MAP does not suppress the difference between TGVi and TGVe. However, the interaction suggests that this correction tends to minimize the difference (table 3B).

The coefficients of variation of TGV were significantly lower for LR than FFT ($p<0.02$), with no significant effect related to the type of occlusion.

The data on phase angle, correlation coefficients of the Pao-Vg relationship and mean airway pressure during the occlusion are reported in table 4. Correlation coefficients are significantly higher for TGVi than for TGVe ($p<0.01$), although both values are close to 1. No significant difference is found for phase angles, and in all but one infant, the average phase was lower than 5°. During expiratory occlusions, MAP appears significantly more negative and greater in magnitude than during inspiratory occlusions ($p<0.01$) with an average difference of 9.7 cmH_2O.

Discussion

Measurement of thoracic gas volume with a flow plethysmograph in infants shows an overall short-term reproducibility similar to that previously reported with a pressure type box [2, 3]. Although averaging the data has little meaning in view of the heterogeneity of this population, the 27.1±5.4 ml (mean±SD) per kg body weight value is within the range usually reported in normal infants [1–3]. The finding of a lower value of TGVi than TGVe also is in agreement with previous measurements both in normal infants and in infants with a variety of respiratory diseases, with an average difference of about 10% [4–6]. The short-term reproducibility was slightly better for LR than FFT, whether occlusions were performed at end-inspiration or end-expiration. It may be due to the fact that the signals are far from being sinusoidal, so that the amplitude of the fundamental frequency is rather small. Thus, the energy available for Fourier analysis is lower than for linear regression which includes all components of the signals.

Metrological factors

A systematic error may be involved when correcting TGVi for the inspired tidal volume measured by the pneumotachograph. The temperature of inspired gas in the pneumotachograph is intermediate between room and body temperature so that the 10% increase in calibration factor related to the heating of the flowmeter could be an overcorrection to BTPS conditions. A 5% error on tidal volume estimation may reasonably be expected. For a tidal volume of 100 ml and TGV at FRC of 300 ml, the error on TGVi would be in the order of 2%, *i.e.*, lower than the average 8% difference observed between TGVi and TGVe.

Among other factors that could explain the difference between TGVi and TGVe is the difference in mean airway pressure between these types of occlusion. During inspiratory occlusions, the magnitude of MAP is smaller than during expiratory occlusions and is usually opposite in sign. The average difference is about 1% of PB. Correcting for MAP, as indicated in equation 3, does not suppress the overall effect of occlusion but tends to minimize the difference observed on the uncorrected data.

The better correlation of the Pao-Vg relationship calculated for TGVi than for TGVe could be attributed to a higher signal to noise ratio during end-inspiratory occlusions, as the volume measured is about 30% greater than during end-expiratory occlusions. On the other hand, it should be noted that the correlation coefficient between 2 periodic signals, in the noise free case, is related to their phase difference [8], so that:

$$r = \cos \phi \qquad (4)$$

For instance, the lowest r value reported in table 4 (0.9840, infant no. 6) is entirely explained by the phase shift of 10.8° (cos 10.8° = 0.9823). When ϕ is considered - Pao being used as the reference signal to determine its sign - no systematic difference is found between ϕe and ϕi. This suggests that the difference observed between TGVi and TGVe may be related to a combination of several mechanisms, that may differ from one infant to another.

Instrumental deadspace and upper airway artefact

Measurements of TGV are performed as the airway opening is connected to a face mask, and part of the volume displaced from the box is related to compression of the volume of gas in the deadspace (Vm, 20 ml in this study). The impedance of the alveolar gas (Zg) and that of the deadspace (Zm) are two parallel pathways separated by the impedance of the airways (Zaw).

Furthermore, the upper airway wall impedance (Zuaw) may be responsible for inequalities between alveolar pressure and pressure at the airway opening [11–13]. It may be particularly important in infants where Zaw is likely to be high compared with Zuaw [14]. If the impedance of the lower airway (Zawi) increases from inspiration to expiration [15], TGVe may be expected to differ from TGVi, because of gas compression in the deadspace and upper airway wall motion, according to the model described in figure 1 and developed in the appendix. Simulating the frequency response of this model shows that the overestimation of TGV and the positive phase shift between Vg and Pao are frequency dependent. TGVe also leads to higher values than TGVi above 0.5 Hz, for the set of chosen parameters (fig. 2). The difference between TGVe and TGVi will increase with increased compliance of the upper airway wall and of the instrumental deadspace. Larger variations of Rawi and lung volume, and a difference in the rate of respiratory efforts between inspiration and expiration may also increase this difference. The rate of respiratory efforts may indeed be slower during end-inspiratory occlusions as a result of the inhibitory-inspiratory Hering Breuer reflex which has been shown to persist beyond the neonatal period [16].

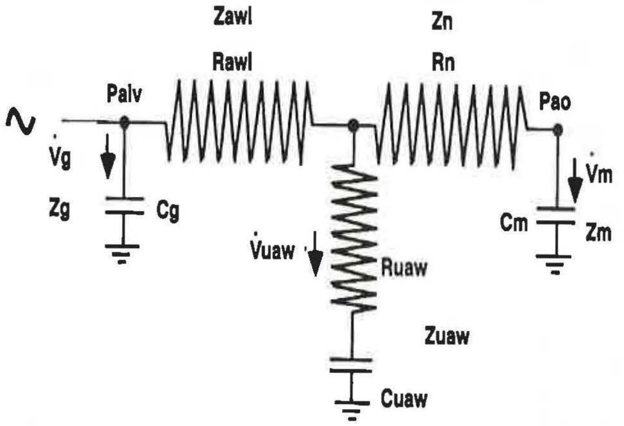

Fig. 1. – Model accounting for deadspace gas (Cm) compression and upper airway wall (Ruaw, Cuaw) motion during thoracic gas volume (TGV) measurement. The thoracic gas (Cg) is in parallel with Cm and upper airway wall which is located between the resistance of the nose (Rn) and that of the remaining airway (Rawi). V̇g, V̇uaw and V̇m, respectively, are flow in the lung, upper airway wall and face mask during the occlusion. Palv: alveolar pressure; Pao: pressure at the airway opening.

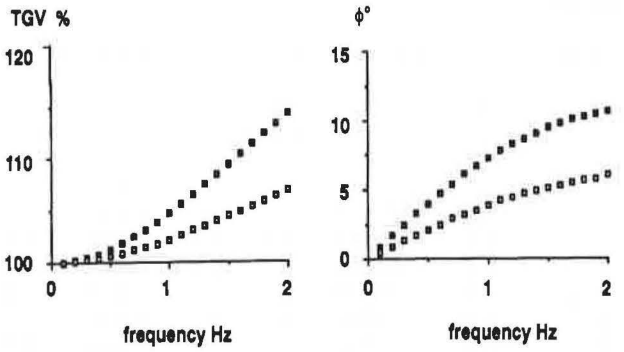

Fig. 2. – Error on TGV expressed as percentage of expected value: (Cg + Cm)·(PB - P_{H_2O}) (left panel) and phase shift between Vg and Pao (right panel), as a function of breathing frequency, according to the model described in figure 1. The following values have been ascribed to the parameters: Ruaw = 100 $cmH_2O \cdot l^{-1} \cdot s$; Cuaw = 0.5 $ml \cdot cmH_2O^{-1}$; Rn = 17 $cmH_2O \cdot l^{-1} \cdot s$; Cm = 0.02 $ml \cdot cmH_2O^{-1}$. For TGVi (□): Cg = 0.28 $ml \cdot cmH_2O^{-1}$, Rawi = 24 $cmH_2O \cdot l^{-1} \cdot s$ and for TGVe (■): Cg = 0.21 $ml \cdot cmH_2O^{-1}$, Rawi = 48 $cmH_2O \cdot l^{-1} \cdot s$. For abbreviations see legend to figure 1.

Mechanical inhomogeneity

Non-uniform alveolar pressure may be present because of airway closure near FRC. Bohadana *et al.* [11] simulated the effect of non-uniform alveolar pressure in a two compartment model where each compartment is characterized by its lung tissue (Cl_1, Cl_2) and gas compliances (Cg_1, Cg_2) and airway resistances (Raw_1,

Raw_2), as described in figure 3. This study showed that the error on TGV depended on: 1) the distribution of gas in the two compartments (Cg_1/Cg_2); 2) local specific lung compliances (Cl_1/Cg_1, Cl_2/Cg_2); and 3) local specific airway resistances ($Raw_1 \cdot Cg_1$, $Raw_2 \cdot Cg_2$) [11]. However, unless dramatic differences in specific Raw or specific Cl, or large inequalities in the distribution of gas between the two compartments were present, the error on TGV did not exceed 5%.

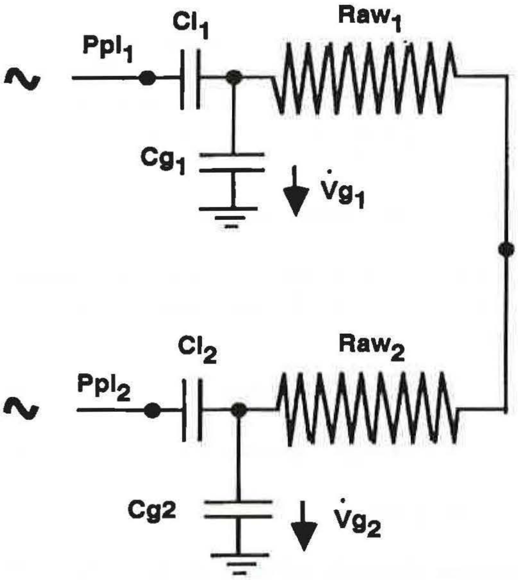

Fig. 3. – Two-compartment model describing inhomogeneity of alveolar pressure and where different pleural pressure (Ppl_1, Ppl_2) may be applied to each compartment. Each compartment is defined by its own gas (Cg_1, Cg_2) and tissue compliance (Cl_1, Cl_2) and airway resistance (Raw_1, Raw_2). For abbreviations see legend to figure 1.

In infants, chest wall distortion may be responsible for local differences of pleural pressure [5] which enhance uneven distribution of alveolar pressure [17]. We used the model described in figure 3, where the amplitude ratio ($K = \Delta Ppl_2/\Delta Ppl_1$) and phase angle ($\theta$) between the pressures applied to both compartments may be varied [11]. The error on TGV when K and θ are varied are given in table 5. Both over- and underestimation of TGV may result, according to K and θ. It is interesting that when K = 1 and θ = 0, no appreciable error is found, despite the difference between specific airway resistances and compliances and amount of gas of the two compartments. In the general case, TGV may be over- or underestimated. This model may be relevant in explaining the striking underestimation of TGV that has been reported in infants with acute bronchiolitis [6]. It may also account for the effect of abdominal gas compression if one compartment is identified as the abdominal gas exposed to gastric pressure. However, the study by Godfrey *et al.* [6] suggested that the usually low ratio of gastric pressure to esophageal pressure change would be a minimal source of error in the measurement of TGV.

Table 5. – Error on TGV (TGV %) and phase shift between Vg and Pao (ϕ) according to the model described in figure 3 when the amplitude ratio (K) or phase angle (θ) of pleural pressures applied to each compartment are varied

K	θ	TGV %	ϕ	K	θ	TGV %	ϕ
1	0	100	0	-	-	-	-
1	+30	106	-6.0	1	-30	95	+5.6
1	+60	113	-19.5	1	-60	90	+11.4
1	+90	124	-25.6	1	-90	84	+18.6
0.5	0	129	+7.5	2	0	66	-11.8

Breathing frequency is set at 0.5 Hz. The following values have been respectively ascribed to the parameters of each compartment: Raw: 125 and 62 cm $H_2O \cdot l^{-1} \cdot s$; Cl: 9 and 5 $ml \cdot cmH_2O^{-1}$; Cg: 0.12 and 0.09 $ml \cdot cmH_2O^{-1}$. TGV: thoracic gas volume. For other abbreviations see legend to figure 1.

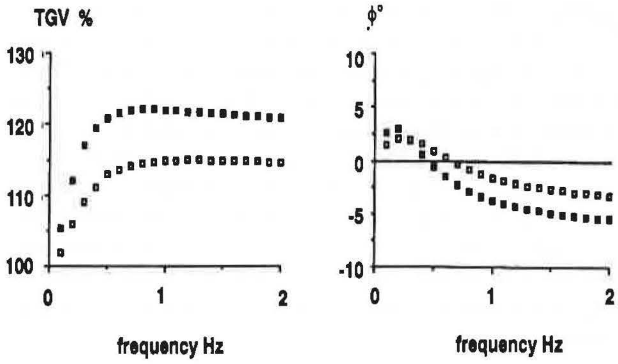

Fig. 4. – Error on thoracic gas volume (TGV) expressed as percentage of expected value (($Cg_1 + Cg_2$)$\cdot$(PB - P_{H_2O})) (left) and phase between Pao and Vg (right) as a function of breathing frequency, according to the model in figure 3, when K = 0.5 and θ = +30°. For TGVi (□) the parameters in the two compartments, respectively, are: Cg = 0.15 and 0.13 $ml \cdot cmH_2O^{-1}$, Raw = 80 and 50 $cmH_2O \cdot l^{-1} \cdot s$, Cl = 9 and 5 $ml \cdot cmH_2O^{-1}$. TGVe (■) as indicated in table 5. For abbreviations see legend to figure 1.

The effect of inspiratory or expiratory occlusion on the model of figure 3 is simulated in figure 4, assuming a 30% increase in total lung volume during end-inspiratory occlusion and a 53% decrease in total airway resistance, with θ = +30° and K = 0.5, *i.e.* the pressure with the larger amplitude (Ppl_1) is applied to the compartment with the higher specific resistance and lags Ppl_2. This would roughly mimick the experimental data of Brown *et al.* [17] who demonstrated that pleural pressure change was greater with regard to an occluded lung lobe than to other territories. Also, specific airway resistances and compliances are assumed to tend toward equalization during inspiration. It appears that the overestimation of TGV is frequency dependent. Furthermore, the difference between TGVe and TGVi also depends on frequency and TGVe may be about 10% higher than TGVi. The phase shift between Pao and Vg does not exceed 5° and may be either positive or negative, depending on frequency. Note that the value of the correlation coefficient of 0.95 which is usually set to attest to the quality of the Pao-Vg relationship would barely detect this error because it already corresponds to a phase shift of 18° (equation 4).

It is concluded that flow plethysmography in infants provides estimates of TGV similar to those previously reported with a pressure type box, although no comparative measurements of TGV by barometric and debitmetric methods are available in infants. When the data are evaluated in view of the correlation coefficient of the Pao-Vg relationship, it is suggested that a minimal value of 0.98 should be selected as this value would attest to a phase shift of at most 10°. LR and FFT yield similar estimates of TGV. The slightly better reproducibility of LR would favour its routine use. On the other hand, it may be of interest to apply FFT on the data from infants with acute bronchiolitis to determine whether any systematic phase shift between Vg and Pao is present. There exists a difference of about 8% between TGVe and TGVi. Up to 3% may readily be explained by metrological factors: the BTPS correction of the volume occluded above FRC (2%) - when no BTPS conditioning of the inspiratory gas is available - and the difference in MAP during end-inspiratory and end- expiratory occlusions (1%). Another 2% may be attributed to compression of the gas in the deadspace and upper airway wall motion. Finally, an unpredictable amount of error appears to depend on the degree of lung inhomogeneity and of uneven distribution of pleural pressure.

Appendix

Deadspace compression and upper airway wall motion (fig. 1). Total gas compression flow ($\dot{V}g_{tot}$) is given by:

$$\dot{V}g_{tot} = \dot{V}g + \dot{V}m \qquad (1)$$

Where $\dot{V}g$ and $\dot{V}m$, respectively, are flow in alveoli (Zg) and deadspace (Zm). During nasal breathing the upper airway wall impedance (Zuaw) is a pathway in parallel with Zm and Zg, located between the impedance of the nose (Zn) and that of the remaining airway (Zawi).

From the parallel arrangement of: 1) Zm and Zn + Zuaw; and 2) Zg and the remainder of the system, the following relationships may be established, respectively, between Vm and the flow in the upper airway wall (Vuaw, equation 2) and among Vg, Vm and Vuaw (equation 3):

$$Zuaw \cdot \dot{V}uaw = (Zn + Zm) \cdot \dot{V}m \qquad (2)$$

$$Zg.\dot{V}g = Zawi \cdot (\dot{V}m + \dot{V}uaw) + (Zn + Zm) \cdot \dot{V}m \qquad (3)$$

Equations 2 and 3 may be solved for $\dot{V}g$ and $\dot{V}m$ and these values replaced in equation 1, leading to the measured impedance:

$$\frac{\Delta Pao}{\Delta \dot{V}g_{tot}} = \frac{Zg \cdot Zuaw \cdot Zm}{Zuaw\,(Zg+Zn+Zm) + Zawi\,(Zuaw+Zn+Zm)} \qquad (4)$$

At low frequencies, Zawi and Zn are assimilated to pure resistances: Rawi and Rn, respectively. Zg and Zm are given by:

$$Zg = -j/Cg \cdot \omega \qquad (5)$$

$$Zm = -j/Cm \cdot \omega \qquad (6)$$

where j is the unit of imaginary numbers, Cg and Cm are gas compliances, respectively, equal to TGV/(P_B-P_{H_2O}) and Vm/(P_B-P_{H_2O}). Finally, Zuaw is assimilated to a resistance (Ruaw) and a compliance (Cuaw) arranged in series, so that:

$$Zuaw = Ruaw - j/Cuaw \cdot \omega \qquad (7)$$

Equation 4 may be transformed to express the volume/pressure ratio and its real (R) and imaginary part (X) may be calculated:

$$R = -(A/Cuaw \cdot \omega + B \cdot Ruaw)/D \qquad (8)$$

$$X = (A \cdot Ruaw - B/Cuaw \cdot \omega)/D \qquad (9)$$

A, B and D are given by:

$$A = Ruaw \cdot Rn + Raw\,(Ruaw+Rn) - 1/Cuaw \cdot \omega\,(1/Cm \cdot \omega + 1/Cg \cdot \omega) \qquad (10)$$

$$B = -Rn/Cuaw \cdot \omega - Ruaw\,(1/Cm \cdot \omega + 1/Cg \cdot \omega) - Raw\,(1/Cuaw \cdot \omega + 1/Cm \cdot \omega) \qquad (11)$$

$$D = (Ruaw^2 \cdot Cuaw^2 + 1)/(Cg \cdot Cm \cdot Cua^2 \cdot \omega^3) \qquad (12)$$

Finally the amplitude ratio (Vg_{tot}/Pao) and phase angle (ϕ) are given by:

$$Vg_{tot}/Pao = (R^2 + X^2)^{0.5} \qquad (13)$$

$$\phi = \tan^{-1}(X/R) \qquad (14)$$

The error is then expressed as the ratio of the measured compliance (Vg_{tot}/Pao) to the actual one (Cg + Cm) per cent.

Acknowledgements: The authors thank N. Bertin, B. Chalon, C. Chone, G. Colin for their skilful technical assistance.

References

1. Doershuk CF, Downs TD, Matthews LW, Lough MD. – A method for ventilatory measurements in subjects 1 month–5 years of age: normal results and observations in disease. *Pediatr Res*, 1970, 4, 165–174.
2. Radford M. – Measurement of airway resistance and thoracic gas volume in infancy. *Arch Dis Child*, 1974, 49, 611–615.
3. Stocks J, Godfrey S. – Specific airway conductance in

relation to postconceptional age during infancy. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1977, 43, 144–154.
4. Beardsmore CS, Stocks J, Silverman M. – Problems in measurements of thoracic gas volume in infancy. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 995–999.
5. Helms P. – Problems with plethysmographic estimation of lung volume in infants and young children. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 53, 698–702.
6. Godfrey S, Beardsmore CS, Maayan C, Bar-Yishay E. – Can thoracic gas volume be measured in infants with airways obstruction? *Am Rev Respir Dis*, 1986, 133, 245–251.
7. Peslin R. – Body plethysmography. *In*: Techniques in Respiratory Physiology. Part II. A.B. Otis ed., New York, 1984, pp. 1–26.
8. Peslin R, Gallina C, Rotger M. – Methodological factors in the variability of lung volume and specific airway resistance measured by body plethysmography. *Bull Eur Physiopathol Respir*, 1987, 23, 323–327.
9. Peslin R, Morinet-Lambert J, Duvivier C. – Etude de la réponse en fréquence des pneumotachographes. *Bull Eur Physiopathol Respir*, 1972, 8, 1363–1376.
10. Begin P, Peslin R. – Mesure pléthysmographique du volume gazeux intrathoracique. Un retour aux hypothèses. *Bull Eur Physiopathol Respir*, 1983, 19, 247–251.
11. Bohadana AB, Peslin R, Hannhart B, Teculescu D. – Influence of panting frequency on plethysmographic measurements of throracic gas volume. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 739–747.
12. Rodenstein DO, Stanescu DC, Francis C. – Demonstration of failure of body plethysmography in airway obstruction. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 949–954.
13. Stanescu DC, Rodenstein D, Cauberghs M, van de Woestijne KP. – Failure of body plethysmography in bronchial asthma. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1982, 52, 939–948.
14. Marchal F, Peslin R, Duvivier C, Gallina C, Crance JP. – Measurement of ventilatory mechanical impedance in infants using a head pressure generator. *Pediatr Pulmonol*, 1987, 7, 209–216.
15. Wohl MEB, Stigol LC, Mead J. – Resistance of the total respiratory system in healthy infants and infants with bronchiolitis. *Pediatrics*, 1969, 43, 495–509.
16. Marchal F, Crance JP. – Measurement of ventiltory system compliance in infants and young children. *Respir Physiol*, 1987, 68, 311–318.
17. Brown RS, Scharf S, Ingram HR. – Nonhomogeneous alveolar pressure swings in the presence of airway closure. *J Appl Physiol: Respirat Environ Exercise Physiol*, 1980, 49, 398–408.

Volume gazeux thoracique à la capacité résiduelle fonctionnelle, mesuré par pléthysmographie débit métrique chez le nourrisson et le jeune enfant. F. Marchal, C. Duvivier, R. Peslin, P. Haouzi, J.P. Crance.

RÉSUMÉ: Le volume gazeux thoracique a été mesuré au moyen par pléthysmographie débit métrique chez 15 petits enfants âges de 2 à 34 mois . L'on a comparé les occlusions des voies aériennes en fin d'expiration (TGVe) et en fin d'inspiration (TGVi), après correction pour la différence de volume par rapport à la capacité résiduelle fonctionnelle pendant l'occlusion. La relation entre la pression à l'ouverture des voies aériennes (Pao) et le volume déplacé pendant l'occlusion (Vg) a été étudiée par: 1) un algorithme incluant une correction pour la dérive de Vg et une analyse de la régression linéaire (LR); et 2) une analyse de Fourier des signaux (FFT). TGVe est significativement plus élevée que TGVi (256 *vs* 237 ml, 20.4)*. Le coefficient de corrélation de la relation Pao-Vg est légèrement mais significativement plus élevé pour TGVi que pour TGVe: 0.9968 (0.9937–0.9995) *vs* 0.9947 (0.9840–0.9990). Aucune différence n'a été observée entre LR et FFT, quoique le coefficient de variation intra-individuel fut plus bas pour LR que pour FFT: 5.2% (1.6–11.3) *vs* 7.9% (1.9–21.0)°. Des simulations modélisées ont suggéré que la différence entre TGVe et TGVi pouvait être attribuée principalement à la compression des gaz dans l'espace mort instrumental et au montement des parois des vois aériennes supérieures, ainsi que/ou à une distribution inégale des pressions alvéolaires et pleurales associée à la distorsion des parois thoraciques. (*) moyennes et racine carrée de la varience residuelle, $p<0.002$. (°) moyennes et extrême, $p<0.02$.

Eur Respir J., 1991, 4, 180–187.