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Respiratory input and transfer mechanical impedances in patients with chronic obstructive pulmonary disease

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ABSTRACT: Total respiratory input (Zin) and transfer (Ztr) mechanical impedances were measured from 4–30 Hz in 9 patients with severe chronic obstructive pulmonary disease (COPD) and in 12 healthy subjects. Zin was obtained by applying a pressure input around the head to minimize transmural pressure across extrathoracic airway walls, and Ztr was obtained with a pressure input at the chest. In agreement with previous studies total respiratory compliance and inertance were decreased in patients, while effective input resistance was increased and exhibited a negative frequency dependence. Effective transfer resistance (Re(Ztr)) was also increased at all frequencies, and, in some patients, the Re(Ztr)-frequency curve was sigmoid in shape, which was never seen in normals. When Ztr was analysed with a six-coefficient monoalveolar model featuring tissue properties, alveolar gas compliance, and airways properties, the model fitted the data less closely in patients than in normals and, in the former, provided unrealistic coefficients. Such was also the case with a bialveolar model. A better fit with more realistic values for the coefficients was obtained in selected patients with a model where central and peripheral (Rp) airway resistance were separated by a shunt representing airway wall compliance (Cb): Cb was found to range from 0.029–0.062 $l kPa^{-1}$ and Rp represented 44–81% of total airway resistance.

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A number of studies have been devoted to total respiratory mechanical impedance in patients with chronic obstructive pulmonary disease (COPD) [1–5]. In all of them, the pressure oscillations were applied at the mouth, as proposed by DuBois *et al.*[6], which provides the so-called respiratory input impedance (Zin). The real part of Zin or effective resistance (Re(Zin)), has been found to be increased in COPD patients, and to decrease as the frequency rises [1–5]. Its imaginary part (Im(Zin)), or reactance, has been found to be decreased [1, 3–5] and, when analysed with a simple second-order model, to provide abnormally low values of total respiratory compliance (Crs) and inertance (Irs) [3, 5]. Both the negative frequency dependence of Re(Zin) and the decreased Im(Zin) have been ascribed to mechanical non-homogeneity of the respiratory system [2, 4, 5] and various attempts have been made to explain the data with more sophisticated models [2, 4, 5, 7]. Such models include parallel pathways meant to represent different tissue compartments and/or airway wall properties [8, 9]. Several studies suggest that the data in COPD patients are best explained by an increased peripheral airway resistance shunted by the compliance of more central airway walls [4, 5]. That interpretation, however, has not yet been substantiated. Moreover, a problem in interpretating Zin in obstructive patients is that the negative frequency dependence of Re(Zin) and decreased Im(Zin) may be considerably exaggerated by the upper airway artefact [10], that is by vibrations of extrathoracic airway walls shunting part of the measured flow.

An alternative to measuring Zin is to apply the pressure oscillations around the chest, as proposed by Mead [11], which provides respiratory transfer impedance (Ztr). The latter differs from Zin because of alveolar gas compression [12]. Its measurement is technically less demanding than that of Zin because the flowmeter is not submitted to the input pressure swing [13]. Also, the upper airway artefact is minimal because transmural pressure across the cheeks is very small. The main advantage of Ztr, however, is that, provided thoracic gas volume (TGV) is known, the data of healthy subjects may be analysed with a six-coefficient monoalveolar model proposed by DuBois *et al.* [6] (fig. 1), and used to estimate airways and tissue properties separately [14]. As, to our knowledge, Ztr

measurements have not yet been reported in patients with COPD, the aim of this investigation was to assess their usefulness in analysing respiratory mechanical disorders in these patients. For this, we measured Ztr from 4–30 Hz in a small group of patients with severe airway obstruction. We also obtained Zin in the same patients using a method which eliminates the upper airway artefact [15]. It was unlikely that the data would be fully consistent with the simple monoalveolar model of DuBois *et al.* An objective of the study, therefore, was to find out if they could be better interpreted with models allowing for mechanical non-homogeneity and bronchial compliance.

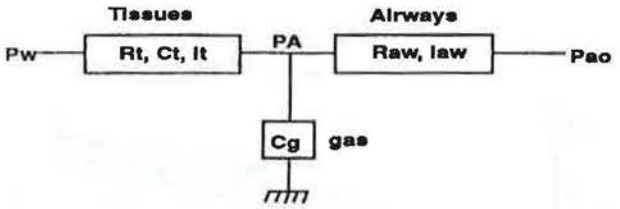

Fig. 1. – DuBois' six-coefficient monoalveolar model including tissue compliance (Ct), resistance (Rt) and inertance (It), alveolar gas compressibility (Cg), and airways resistance (Raw) and inertance (Iaw). Pw, PA, Pao: pressure around the chest, in the alveoli, and at the airway opening, respectively.

Input and transfer impedances were measured from 4–30 Hz as described previously [16]. Briefly, the subject was seated in a wooden box up to the neck, at which level a good seal without compression of the upper airway was obtained with a collar made of synthetic foam. The walls of the box were fitted with two 100 W loudspeakers which were used to apply pressure variations around the body during Ztr measurements.

For Zin, the box was kept open and pressure variations around the head were obtained using a plexiglass canopy equipped with a 100 W loudspeaker. With this type of pressure input, the transmural pressure across the cheeks and other upper airway walls is much smaller than with the conventional pressure input at the mouth, so that the upper airway artefact is almost completely eliminated [15]. For both Zin and Ztr measurements the pressure input generated by the loudspeakers was a pseudorandom noise [5], containing all the harmonics of 2 Hz from 4–30 Hz; the peak-to-peak pressure amplitude was kept below 0.2 kPa. Mouth flow was measured with a Fleisch no. 2 pneumotachograph connected to a Validyne MP45 ±0.2 kPa pressure transducer. Transrespiratory pressure was measured with a similar transducer matched to the first within 1% of amplitude and 2° of phase up to 30 Hz. The measurements were made during quiet breathing. The pressure and flow signals were digitized on-line for periods of 16 s by an Apple 2e computer system with a sampling rate of 128 Hz. They were processed by Fourier analysis as described by MICHAELSON *et al.* [4] so as to obtain the real and imaginary parts of impedance at the 14 investigated frequencies [16]. The analysis was made on 2 s time windows with 50% overlap between successive windows. The data were rejected when the coherence function [4] was below 0.9. The impedance values from 3–5 measuring periods were averaged.

Table 1. – Subjects' diagnosis, biometric characteristics and Spirometric data

Subject	Diagnosis	Age yr	Height cm	Weight kg	FVC % pred	FEV_1/FVC %	TGV *l*
ZA	CB + E	59	177	63	55	29	5.8
LA	CB + E	62	163	46	66	47	5.7
BR	CB + E	60	172	65	66	32	6.4
BI	CB	63	168	72	54	50	5.5
CH	CB + EE	69	170	65	93	37	5.3
DE	CB + E	62	172	70	38	33	8.0
AR	CB	69	172	85	38	48	3.4
SE	AB + E	35	167	55	53	31	6.5
PO	CB + Esp	50	166	80	68	50	3.7

CB: chronic bronchitis; E: emphysema; Esp: subpleural emphysema; AB: asthmatic bronchitis; TGV: thoracic gas volume; FVC: forced vital capacity; FEV_1: forced expiratory volume in one second.

Subjects and methods

The study was performed in nine male patients with severe chronic obstructive pulmonary disease (table 1). All had a forced expiratory volume in one second (FEV_1) below 50% of predicted and a FEV_1/forced vital capacity (FVC) ratio <50%. Two had chronic bronchitis and five had chronic bronchitis with functional and radiological signs of emphysema (CO transfer factor <60% predicted, loss of peripheral vascular markings, in two instances computerized tomographic (CT) scan evidence of centrolobular emphysema and, in one case, of panlobular emphysema with bullous lesions). One patient had asthmatic bronchitis with emphysema and the last had chronic bronchitis with subpleural emphysema. Impedance measurements were also performed for comparison in a group of 12 healthy males, 20–56 yrs old, with normal spirographic data.

Data analysis

The real part of input impedance was characterized by its mean value ($\bar{R}e(Zin)$) over the entire frequency range, and its frequency dependence by the slope (S in $kPa \cdot l^{-1} \cdot s^2$) of a straight line fitted to the

resistance-frequency curve by linear regression. The imaginary part (Im(Zin)) was analysed, as in previous studies [3, 5] in terms of total respiratory compliance (Crs) and inertance (Irs) according to:

$$Im(Zin) = Irs \cdot \omega - 1/(Crs \cdot \omega) \quad (1)$$

where $\omega = 2\pi f$. Irs and Crs were obtained by least squares non-linear regression.

Transfer impedance data were first analysed using the six-coefficient monoalveolar model of DuBois *et al.* [6]. The model (fig. 1) includes a tissue compartment featuring tissue compliance (Ct), resistance (Rt) and inertance (It), alveolar gas compressibility (Cg), and an airway compartment with airway resistance (Raw) and gas inertance (Iaw). The analysis required entering the value of Cg [14]. The latter was computed from plethysmographic thoracic gas volume ($Cg = TGV/P_B$ where P_B is barometric minus alveolar water vapour pressure). A general parameter estimation algorithm [17] was used to find the set of tissue and airways coefficients which minimized the root mean squared (rms) difference (D) between measured (Ztr_m) and computed (Ztr_c) transfer impedances:

$$D = \left(\frac{1}{n}\sum_{1}^{n}|Ztr_m - Ztr_c|^2\right)^{1/2} \quad (2)$$

where n is the number of investigated frequencies. In subjects selected for the quality of their data, Zin and Ztr were also analysed using two other models allowing for parallel pathways; they will be described later on. The coefficients of these models giving the best fit to both Zin and Ztr were found using a similar minimization criterion:

$$D = \left(\frac{1}{2n}\sum_{1}^{n}|Ztr_m - Ztr_c|^2 + |Zin_m - Zin_c|^2\right)^{1/2} \quad (3)$$

Results and discussion

Average input and transfer impedance curves in the group of patients are compared in figure 2 to the data in 12 healthy subjects. The corresponding indices are presented in tables 2 and 3. Re(Zin) (fig. 2 top) was significantly increased at all frequencies ($p<0.001$) and, on average, decreased significantly with increasing frequency ($p<0.001$ by variance analysis), which was not the case in normal subjects. Im(Zin) was significantly lower than normal over the entire frequency range ($p<0.001$), and in three patients it was still negative at 30 Hz. Both the total respiratory compliance and inertance, computed from the curves, were significantly decreased ($p<0.001$). These findings are qualitatively similar to those made by others [1–5].

Re(Ztr), like Re(Zin), was significantly larger than normal at all frequencies ($p<0.001$). Also, on average, Re(Ztr) was larger than Re(Zin) up to 27 Hz, which was only the case up to 10 Hz in healthy subjects. Transfer reactance, contrary to input reactance, was only significantly decreased in patients at the lowest frequencies. The quality of the fit of the monoalveolar

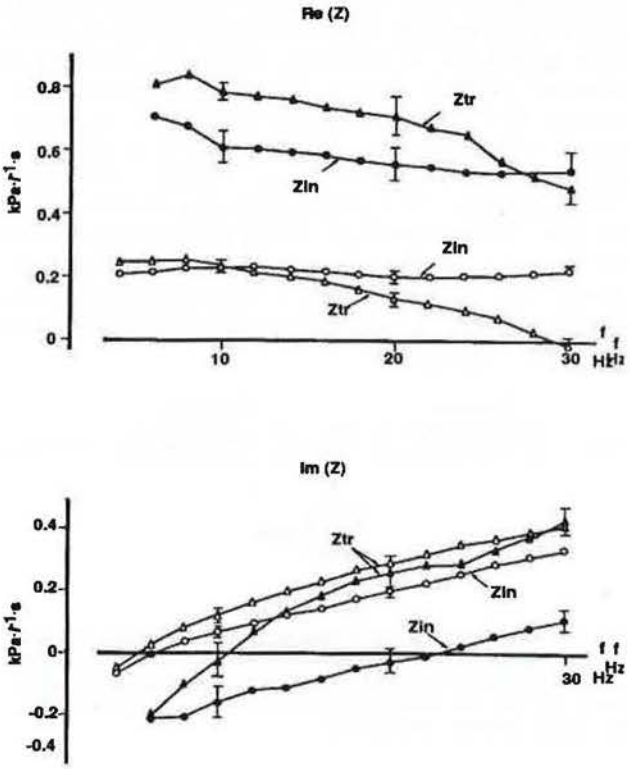

Fig. 2. – Mean values of real (Re) (top) and imaginary (Im) (bottom) parts of transfer impedance (triangles) and input impedance (circles) in 9 COPD patients (closed symbols) and in 12 healthy males (open symbols). Vertical bars: standard errors. Zin, Ztr: respiratory input and transfer impedance, respectively.

Table 2. – Parameters derived from input impedance curves

Subject	Re kpa·l⁻¹·s	S Pa·l⁻¹·s²	Crs l·kPa⁻¹	Irs Pa·l⁻¹·s²	f_n Hz
Patients					
ZA	0.41	-3.7	0.13	0.76	18
LA	0.26	-5.9	0.16	0.56	18
BR	0.60	-10.2	0.06	-0.10	>30
BI	0.49	-1.4	0.11	0.71	20
CH	0.45	1.4	0.25	1.01	11
DE	0.32	-15.1	0.04	0.09	>30
AR	0.64	-14.1	0.11	0.79	20
SE	0.46	-8.6	0.12	0.35	23
PO	0.67	-1.4	0.12	-0.32	>30
Mean	0.48	-6.5	0.12	0.43	
SD	0.14	5.8	0.06	0.45	
Normal subjects (n = 12)					
Mean	0.19	-1.0	0.33	1.62	6.6
SD	0.04	0.9	0.05	0.22	0.5
p	<0.001	<0.02	<0.001	<0.001	

Re: mean value of real part of input impedance from 4–30 Hz; S: slope of linear regression of Re *vs* frequency; Crs, Irs: total respiratory compliance and inertance derived from imaginary part of input impedance (equation 1); f_n: resonant frequency at which imaginary part of impedance is zero; p: statistical significance of differences between data in COPD patients and in normals (t-test); COPD: chronic obstructive pulmonary disease.

Table 3. – Coefficients derived from transfer impedance curves using the 6-coefficient monoalveolar model of DuBois *et al.*

Subject	Raw kPa·l⁻¹·s	Iaw Pa·l⁻¹·s²	Rt kPa·l⁻¹·s	Ct l·kPa⁻¹	It Pa·l⁻¹·s²	Dr kPa·l⁻¹·s
Patients						
ZA	0.48	<0	0.066	0.22	0.30	0.033
LA	0.36	<0	0.061	0.15	0.26	0.040
BR	0.55	<0	0.040	0.08	0.32	0.039
BI	0.50	2.11	0	0.21	0.15	0.064
CH	0.36	0.16	0.143	0.31	0.13	0.063
DE	0.19	<0	0.055	0.03	0.81	0.042
AR	0.67	2.18	0.035	0.10	0.25	0.067
SE	0.44	<0	0.053	0.13	0.40	0.059
PO	0.76	0.61	0.042	0.12	0.40	0.099
Mean	0.48	-	0.055	0.15	0.34	0.056
SD	0.17	-	0.038	0.08	0.20	0.021
Normal subjects (n = 12)						
Mean	0.13	1.56	0.094	0.35	0.19	0.014
SD	0.04	0.25	0.025	0.06	0.07	0.003
p	<0.001		<0.02	<0.001	<0.05	<0.001

Raw, Iaw: airway resistance and gas inertance in airways; Rt, Ct, It: tissue resistance, compliance and inertance; Dr: residual root mean square difference between observed impedance and best-fitting model impedance.

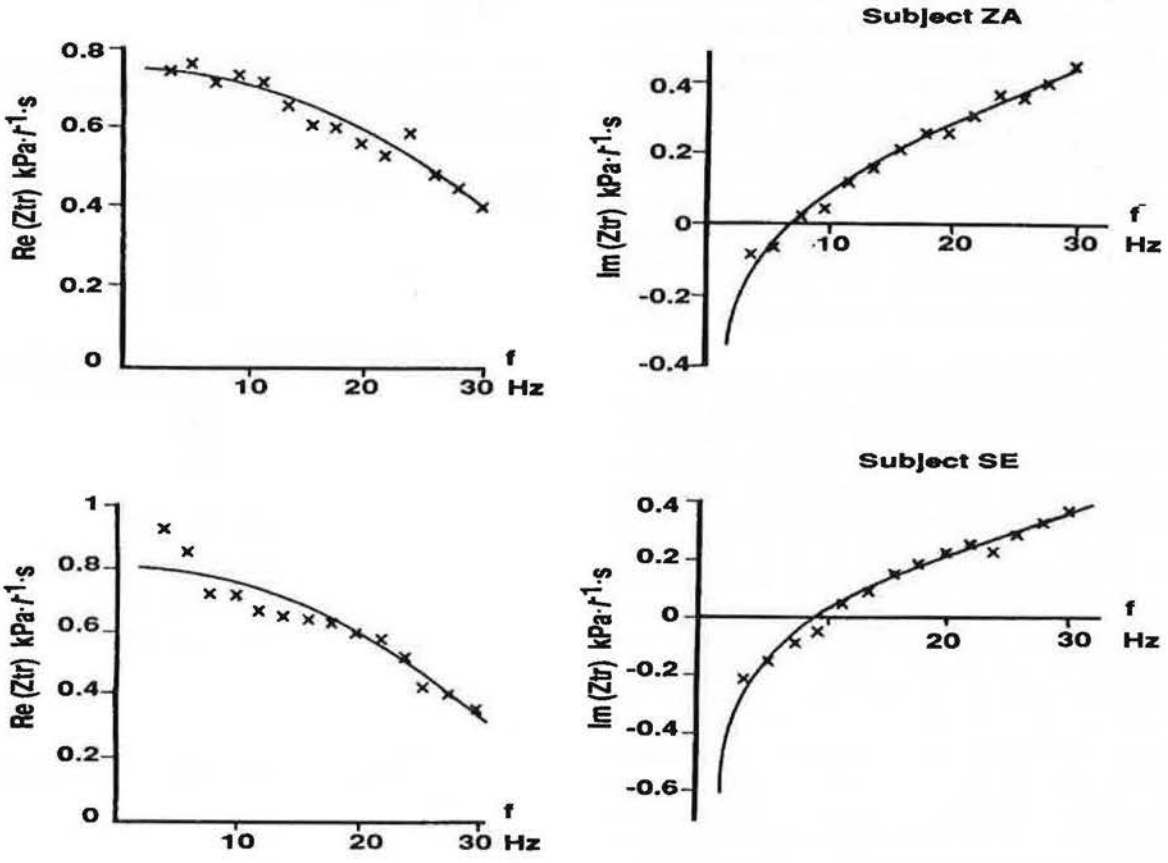

Fig. 3. – Examples of fit between measured impedance (crosses) and impedance computed from DuBois' six-coefficient monoalveolar model (continuous line). Abbreviations as in figure 2.

model of DuBois *et al.* [6] to transfer impedance data in two representative patients is illustrated in figure 3. On average, the residual rms difference between model and observed impedances (Dr) was much larger in patients than in normals (table 3). This was due, to some extent, to the larger value of the impedance and to a larger amount of experimental noise, but also, in many instances, to systematic differences in shape between the observed and computed impedances. For instance, in several patients (including the case in figure 3 bottom), the resistance-frequency curve was sigmoid, which could never be the case with the model. The values of Raw and It giving the best fit to the data were, on average, significantly higher than in normal subjects (table 3), while the values of Rt and Ct were significantly lower. In five out of nine patients Iaw was negative, which is physically meaningless and was not observed in any of the healthy subjects. It is for that reason, and because of the differences in shape between computed and observed Ztr in some patients, that the data were also analysed with alternative models including parallel pathways. This model analysis was only performed in three patients who had both negative Iaw values and comparatively noise-free Zin and Ztr data. Two of them had a clearly sigmoid Re(Ztr)-frequency curve. As the models had a large number of coefficients, we thought it better to test them on all the available information, that is simultaneously on Zin and Ztr data, using the combined minimization criterion defined in equation 3. The same value of Cg was used as previously. For comparison, this was also done with the model of DuBois *et al.* [6] where it always led to a larger residual rms error than when the analysis was done on Ztr alone (0.046, 0.204 and 0.088 kPa·l^{-1}·s, compared to 0.040, 0.042 and 0.059 kPa·l^{-1}·s respectively, in the three subjects). This may reflect the limitations of the model, but also biological variability since Zin and Ztr were not measured simultaneously. An example is shown in figure 4 top.

The first of the alternative models was meant to account for mechanical non-homogeneity of the airways and of the tissues and was made of two DuBois' six-coefficient units in parallel (fig. 5, model A). However, to keep the number of coefficients within reasonable limits, it was imposed that the two units had the same Iaw, It and Rt. This was thought to be acceptable because It and Rt are probably mostly located in the chest wall, which had no reason to be abnormal in these patients, and most of Iaw is in central airways [18] common to the rest of the system. In addition, to avoid multiple solutions, it was imposed that the two units had the same alveolar gas volume. With these restrictions, the model had 7 instead of 11 unknown coefficients, and the two units could only differ by their airway resistance and tissue compliance. The results are shown in table 4. In the three subjects this model gave a significantly lower residual rms difference between observed and computed impedances than the monoalveolar model ($p<0.001$ by the F-test proposed by Eyles *et al.* [19]). However, in none of them was the solution physiologically meaningful: Iaw was too low

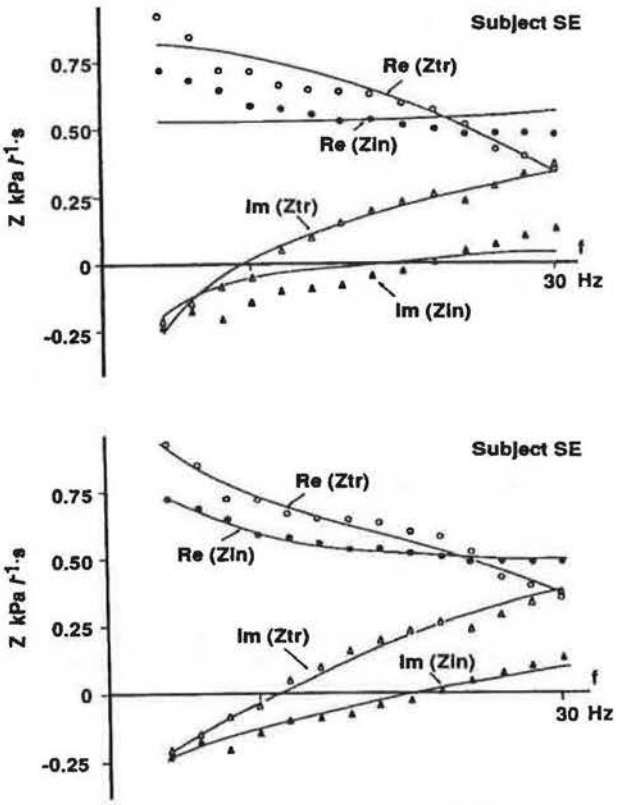

Fig. 4. – Example of fit of DuBois' monoalveolar model (top) and of model (fig. 5, model B) with airway wall compliance (bottom) to measured input (closed symbols) and transfer (open symbols) impedances. Abbreviations as in figure 2.

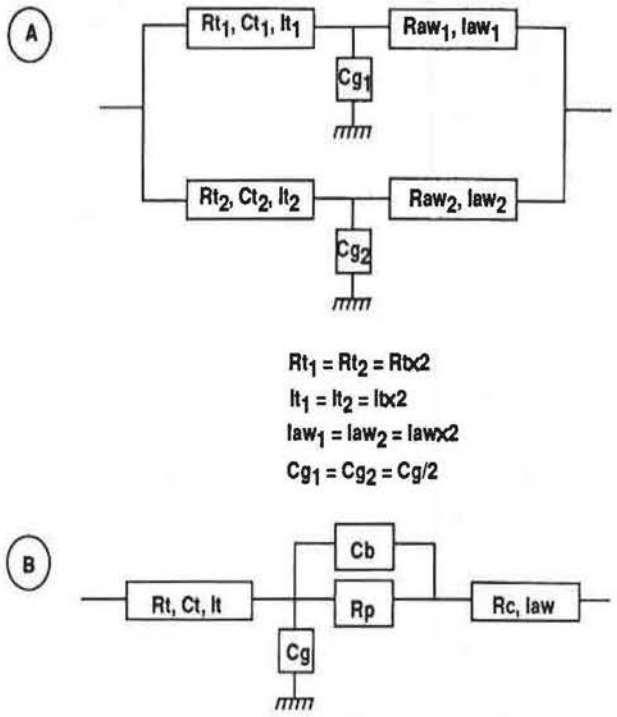

Fig. 5. – Models including parallel pathways. A: non-homogeneity of airways and tissue properties obtained by placing two DuBois' six-coefficient units (fig. 1) in parallel. B: model with airway wall compliance (Cb) in parallel with peripheral airway resistance (Rp). Rc is central airway resistance; other symbols as in figure 1.

Table 4. – Coefficients obtained in three subjects with two-compartment model

	Subject		
	LA	DE	SE
Iaw $Pa \cdot l^{-1} \cdot s^2$	0.81	<0	0.32
Raw_1 $kPa \cdot l^{-1} \cdot s$	0.53	0.37	1.77
Raw_2 $kPa \cdot l^{-1} \cdot s$	0.86	>3.00	0.70
Rt $kPa \cdot l^{-1} \cdot s$	0.025	0.018	0.035
Ct_1 $l \cdot kPa^{-1}$	0.019	0.024	>9.99
Ct_2 $l \cdot kPa^{-1}$	1.90	0.105	0.039
It $Pa \cdot l^{-1} \cdot s^2$	0.08	0.51	0.33
Dr $kPa \cdot l^{-1} \cdot s$	0.032	0.161	0.071

Raw_1, Raw_2, Ct_1, Ct_2: airway resistance and tissue compliance of compartments 1 and 2 (model A, fig. 5). Other symbols as in table 3.

Table 5. – Coefficients obtained in three subjects with model featuring airway compliance

	Subject		
	LA	DE	SE
Rc $kPa \cdot l^{-1} \cdot s$	0.21	0.26	0.43
Iaw $Pa \cdot l^{-1} \cdot s^2$	1.35	0.86	1.03
Rp $kPa \cdot l^{-1} \cdot s$	0.19	1.12	0.34
Cb $l \cdot kPa^{-1}$	0.029	0.029	0.062
Rt $kPa \cdot l^{-1} \cdot s$	0.039	0.062	0.048
Ct $l \cdot kPa^{-1}$	0.126	0.302	0.306
It $Pa \cdot l^{-1} \cdot s^2$	0	0.02	0.14
Dr $kPa \cdot l^{-1} \cdot s$	0.029	0.062	0.034

Rc, Rp: central and peripheral airway resistance; Cb: bronchial compliance (model B, fig. 5). Other symbols as in table 3.

(expected value for the large airways of $\cong 1$ $Pa \cdot l^{-1} \cdot s^2$ [18]) or even negative, and in two subjects total compliance (Ct_1+Ct_2) was too large. Also, Rt was smaller than found in normal subjects with the monoalveolar model (table 3).

The second model (fig. 5B) was meant to represent the type of parallel pathway described by MEAD [8] and included a bronchial compliance (Cb). The latter partitioned the airway between a central segment with a resistance (Rc) and a gas inertance (Iaw), and a purely resistive peripheral segment (Rp). On the other side Cb was connected to the alveolar space, rather than to the pleural space, so that it was not necessary to represent lung tissue and chest wall properties separately. This was done to limit the number of coefficients and was shown by computer simulation to make little difference. Indeed, lung impedance is low at the frequencies at which the shunt effects of Cb becomes important. In practice, the role of Cb is to shunt the peripheral resistance, and its effect is greater when Rp is large and when the frequency increases (wall impedance is proportional to 1/f). In the three subjects this model fitted the data much better than DuBois' model, as illustrated in figure 4 (bottom), and also better than the two-compartment model, although it had the same number of parameters. The corresponding coefficients are shown in table 5. Depending on the subject Rp represented from 44–81% of total airway resistance. Iaw was still lower than in normals (table 3), but substantially larger than with the other models. Cb averaged 0.04 $l \cdot kPa^{-1}$, which is of the order of magnitude mentioned by MEAD [8]. In two subjects Ct was substantially higher than with DuBois' model, and similar to that found in normals; Rt, however, remained rather low. On the whole, these results are much more acceptable than those obtained with the two-compartment model. They suggest, in agreement with computer simulation of Zin [4, 5] that, in the 4–30 Hz frequency range, mechanical non-homogeneity of the respiratory system of COPD patients is mostly due to airway wall compliance in parallel with a high peripheral airway resistance.

To summarize our findings, we observed that in patients with severe COPD, the real part of transfer impedance was increased at all frequencies and that, in some of the patients, the Re(Ztr)-frequency curve exhibited a sigmoid shape, which was never the case in normal subjects. On the other hand, contrary to what was seen for Zin, the imaginary part of Ztr was only significantly decreased at low frequencies. Analysing the data with DuBois' six-coefficient monoalveolar model almost invariably led to unrealistic values of one or several of the coefficients. The most obvious shortcomings of this model, when applied to COPD patients, are that alveolar pressure is assumed to be homogeneous, and that airway walls are assumed to be stiff. The data of representative subjects with little experimental noise were therefore further analysed with two models allowing for mechanical non-homogeneity of alveolar pressure, and for intrathoracic airway wall compliance, respectively. It was found that the second of the models fitted Zin and Ztr data better than the first, and provided more realistic coefficients. This finding, however, should be considered cautiously because the number of subjects with Zin and Ztr curves smooth enough to be analysed with a sophisticated model was very small. It is, nonetheless, encouraging enough to stimulate further work in that direction. Indeed, the contribution of forced oscillation measurements to mechanical investigations in COPD patients would be much more valuable if they could supply information on peripheral airway resistance and bronchial wall elasticity. A step in that direction would be to improve the quality of the measurements, which may be accomplished in a number of ways: increase the number of sampling periods and their duration; optimize the input pressure signal in terms of relative amplitude of the components; increase the value of the coherence function below which the data are discarded; alternate input and transfer impedance sampling periods so as to avoid any time bias. A second step would be to substantiate the validity of the model by studying the consistency of the data obtained in various experimental situations, for instance mechanical loading at the mouth or at the chest, breathing of foreign gases, or bronchomotor challenge.

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Impédance d'entrée et impédance de transfert dans les maladies pulmonaires obstructives chroniques. Y. Ying, R. Peslin, C. Duvivier, C. Gallina, J. Felicio da Silva.

RÉSUMÉ: L'impédance d'entrée (Zin) et l'impédance de transfert (Ztr) du système mécanique ventilatoire total ont été mesurées de 4 à 30 Hz chez 9 malades souffrant d'une affection pulmonaire obstructive chronique et chez 12 sujets sains. Zin a été obtenue en appliquant des variations de pression autour de la tête afin de minimiser la pression transmurale au niveau des parois des voies aériennes extrathoraciques; Ztr a été mesurée avec une entrée en pression au niveau du thorax. En accord avec les données de la littérature, la compliance et l'inertance respiratoires totales étaient diminuées chez les malades, tandis que la résistance déntrée effective était augmentée et diminuait avec la fréquence. La résistance de transfert effective (Re(Ztr)) était également augmentée à toutes les fréquences et, chez quelques malades, la relation Re(Ztr) - fréquence présentait une forme sigmoïde, qui n'est pas observée chez le sujet sain. Les valeurs de Ztr ont été analysées avec un modèle monoalvéolaire représentant les propriétés tissulaires, la compliance gazeuse alvéolaire et les propriétés des voies aériennes. Chez les malades, l'adjustement du modèle aux résultats était moins bon que chez l'homme sain et les coefficients obtenus peu réalistes. Tel était aussi le cas avec un modèle bi-alvéolaire. Un meilleur ajustement avec des valeurs de coefficients plus vraisemblables a été obtenu chez quelques sujets sélectionnés avec un modèle incluant la compliance bronchique (Cb): suivant les sujets, Cb était compris entre 0.029 et 0.062 $l \cdot kPa^{-1}$ et la résistance périphérique représentait 44 à 81% de la résistance totale des voies aériennes.

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