

Role of lung volumes in sleep apnoea-related oxygen desaturation

F. Sériès, Y. Cormier, J. La Forge

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ABSTRACT: We studied the influence of lung volumes on apnoea-induced desaturation in ten subjects with sleep apnoea syndrome. Lung volumes were measured by helium dilution in the sitting and supine position and closing volume with the single-breath nitrogen washout test. To characterize the severity of apnoea-induced desaturations, we determined a desaturation curve for each patient. This curve was obtained by plotting the fall in arterial oxygen saturation (SaO_2) reached at the end of each apnoea against the apnoea duration. From this curve we selected two indices: 1) the SaO_2 fall following 30 s of obstructive apnoea (ΔSaO_2 30); and 2) the desaturation surface between 10-30 s of obstructive apnoea (DS 10-30). Both the ΔSaO_2 30 and the DS 10-30 were significantly correlated with the expiratory reserve volume (ERV), measured in the sitting ($r=0.77$ and 0.65 , respectively; $p<0.05$) and the supine positions ($r=0.96$ and 0.87 ; $p<0.005$). A strong correlation was also observed with the difference between the supine ERV and the seated closing volume CV ($r=0.99$ with ΔSaO_2 30 and 0.89 with DS 10-30; $p<0.005$). Obesity influenced sitting and supine ERV values. We conclude that, among lung volumes, supine ERV and supine ERV-seated CV are the best indicators of the severity of apnoea-induced desaturation.

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Obstructive sleep apnoeas are usually associated with oxygen (O_2) desaturation [1]. The severity of these falls in arterial oxygen saturation (SaO_2) depends on several factors: the time spent in apnoea, the pre-apnoeic SaO_2 value, and the sitting expiratory reserve volume (ERV) [2]. In awake normal subjects, FINDLEY *et al.* [3] found that the SaO_2 falls following voluntary apnoeas increase when the apnoeas are initiated below closing capacity. Therefore ERV and closing volume (CV) appear to be important in the determination of the apnoea-induced desaturation. As obesity and supine position modify lung volumes [4, 5], we studied the relationship between the sitting and supine lung volumes and apnoea-induced desaturation. Our results show that supine ERV and supine ERV-seated CV are the lung volumes that best predict the severity of the desaturation following obstructive apnoeas.

Methods

Patients

Ten patients with sleep apnoea syndrome (SAS) were included in the study. The diagnosis of SAS was based on the clinical history and on the results of sleep studies (*cf infra*). All had an apnoea and hypopnoea index

(number of these events per hour of sleep) above 10. An SaO_2 value during sleep, in the absence of respiratory abnormalities, above or equal to 90% was also required for inclusion in this study.

Pulmonary function studies

Lung function tests were obtained by conventional techniques [6] the day following the polysomnographic studies. Lung volumes were measured with the helium dilution technique, in the sitting and supine position. Supine lung volume measurements were obtained after 15 min in this position. Predicted values were those of GRIMBY and SODERHOLM [7]. Closing volume was measured with the single-breath nitrogen washout technique [8].

Polysomnographic studies

Sleep studies included recordings of electroencephalogram (C_3A_2 , C_4A_1), electro-oculogram, submental electromyogram, electrocardiogram, nasal and mouth flows (thermistors), thoracic and abdominal movements with inductance plethysmography (Respirace, Ambulatory Monitoring, Ardsley, NY), SaO_2 (oximeter Biox IIA), and

intrathoracic pressures with an oesophageal balloon. Each signal was continuously recorded on a polygraph (Grass Instruments, model 78D, Quincy, MA), at paper speed of 10 mm·s⁻¹. Sleep stages and abnormal respiratory events were defined by standard criteria [9, 10]. Individual post-apnoeic desaturations were characterized by the relationship between the SaO_2 fall following an obstructive apnoea and the length of this apnoea. Collecting these two variables (SaO_2 fall and apnoea length) throughout the night's recording, we determined a desaturation curve for each patient (fig. 1). We only considered obstructive apnoeas where the pre-apnoeic SaO_2 was greater than or equal to 90% and the lowest SaO_2 was greater than or equal to 60% (limit of linearity of our oximeter). The SaO_2 fall following 30 s of obstructive apnoea (ΔSaO_2 30), and the desaturation surface between 10–30 s of obstructive apnoea (DS 10–30) were estimated from this desaturation curve. The desaturation surface represents the integrated desaturation between 10–30 s after the onset of an obstructive apnoea and is represented in figure 1 as the area under the desaturation curve between 10–30 s of apnoea.

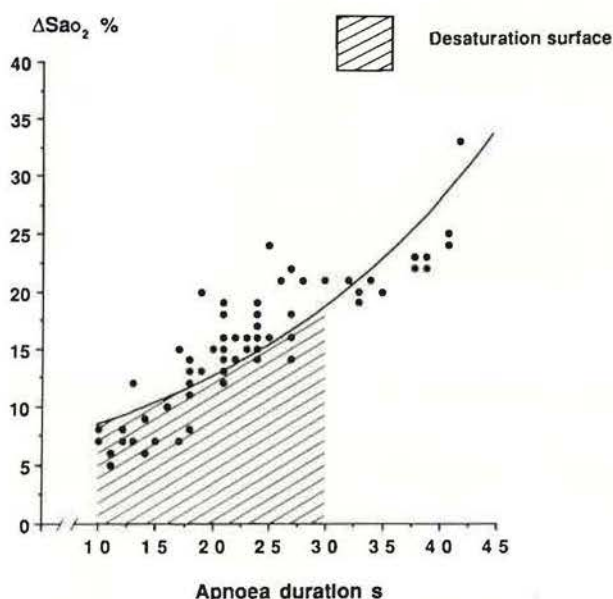


Fig. 1. – Representation of the desaturation curve in a typical subject. It was determined by plotting the fall in arterial oxygen saturation (SaO_2) against duration for each apnoea. For each patient we calculated the SaO_2 fall following 30 s of obstructive apnoea and the area under the desaturation curve between 10–30 s. This area represents the desaturation surface.

Statistical analysis

Since individual data were not normally distributed, statistical analysis was made using non-parametric tests. We used the Wilcoxon signed rank test to compare sitting and supine lung volumes. For each patient, the desaturation curve was determined by polynomial regression analysis from all data (SaO_2 fall and apnoea

duration) collected during the night. The Spearman correlation method was used to analyse correlations between ΔSaO_2 30, DS 10–30 and lung volumes, ΔSaO_2 30, DS 10–30 being the dependent variables.

Results

Patients' characteristics and pulmonary function tests

The mean age and weight of our ten men were 54 ± 8 yrs (mean \pm SD) and $124.8 \pm 4.5\%$ of ideal body weight (IBW). Four patients were ex-smokers, and the others nonsmokers. Results of the pulmonary function tests are reported in table 1. To isolate the effect of obesity on lung volumes, these were expressed as a percentage of the predicted value for the IBW. This was obtained by considering the IBW for the weight value in the regression equation of normal lung volumes. The expiratory flows were normal in all subjects. Forced expiratory volume in one second (FEV_1) ranged from 82–110% of predicted value, and FEV_1/FVC (forced vital capacity) from 75–85%. Sitting lung volumes were normal except for the expiratory reserve volume (ERV). By changing the body position from sitting to supine, functional residual capacity (FRC) fell from $101.2 \pm 27.9\%$ to $75.2 \pm 11.2\%$ ($p < 0.01$) and ERV from $71.8 \pm 26.6\%$ to $30.5 \pm 14.9\%$ ($p < 0.01$). The ERV sitting and supine values were influenced by the body weight, the greater the obesity, the smaller the ERV ($r = 0.72$ in the sitting and $r = 0.93$ in the supine position, $p < 0.05$). Closing volume (CV) values were 414.5 ± 39.5 ml (from 250–650 ml). In the sitting position ERV was greater than CV for all subjects (fig. 2). However, in five patients, supine ERV was lower than CV (fig. 2); in other words, in the supine position, their FRC was below the closing capacity (CV + residual volume).

Sleep studies results

For each patient the total sleep time (TST) was greater than 5 h (5.8 ± 0.6 h); the majority of the TST was spent in stage I–II ($85 \pm 3\%$), stage III–IV accounted for $4 \pm 2\%$ of TST and rapid eye movement sleep for $11 \pm 3\%$. The characteristics of apnoeas and apnoea-related desaturations are reported in table 2. The apnoea index varied from 18 to 71. Time spent in obstructive apnoea represented the most important part of the total apnoea time in all subjects. In every subject there was a strong correlation between the apnoea duration and the accompanying SaO_2 fall ($r > 0.85$; $p < 0.001$). The individual data of the ΔSaO_2 30 and the different lung volumes in the sitting and supine position are presented in table 3. The SaO_2 fall was correlated with sitting and supine ERV and with the absolute value of the supine ERV minus seated CV: the lower this volume the deeper the SaO_2 fall. Table 4 gives the results of the regression analysis between DS 10–30 and lung volumes measured in each position. The ΔSaO_2 30 and DS 10–30 were not correlated with any of the other respiratory functions.

Table 1. – Results of the pulmonary function tests in both positions.

	Position	
	Sitting	supine
Total Lung Capacity %	111.9±16.2	106.4±12.1
Vital Capacity %	93.0±13.8	92.3±13.9
Functional Residual Capacity (FRC) %	101.2±27.8	75.2±11.2 *
Expiratory Reserve Volume (ERV) %	71.8±26.5	30.5±14.8 *
Residual Volume %	131.2±41.4	117.6±20.5
Closing Volume (CV) ml	417.5±125.1	
FEV ₁ %	92.0±13.0	
FEV ₁ /Forced Vital Capacity %	83.1±10.1	

Lung volumes are expressed in percentage of the predicted value for the ideal body weight except for closing volume which is given in absolute values. Sitting and supine values were compared with the Wilcoxon signed rank test (mean±SD). *:p<0.05; FEV₁: forced expiratory volume in one second.

Table 2. – Characteristics of apnoea and post-apnoeic desaturation in the ten patients studied.

Apnoea index		44.8±18.2
Total apnoea time	% of total sleep time	19.0±8.2
Time spent in obstructive apnoea	% of total apnoea time	78.7±6.9
Time spent in mixed apnoea	% of total apnoea time	14.2±5.0
Time spent in central apnoea	% of total apnoea time	6.5±3.1
Baseline Sao ₂	%	94.5±1.4
ΔSao ₂ 30	%	12.8±2.6
DS 10–30	% Sao ₂ ·s ⁻¹ of apnoea	174.0±29.5

The baseline Sao₂ represents the Sao₂ value during a quiet breathing sleep. The ΔSao₂ 30 s is the Sao₂ fall following a 30 s obstructive apnoea and DS 10–30 the desaturation surface between 10–30 s of apnoea (mean±SD). Sao₂: arterial oxygen saturation.

Table 3. – Individual data of the arterial oxygen saturation (Sao₂) fall following a 30 s obstructive apnoea (ΔSao₂ 30) and the lung volumes measured in each position.

Subject	ΔSao ₂ 30 %	Weight % IBW	Sitting FRC %	Supine FRC %	Sitting ERV %	Supine ERV %	Supine ERV-CV ml
1	14.8	124	91	70	72	30	-70
2	15.2	130	69	58	49	15	-95
3	15.4	133	90	76	33	13	-100
4	9.1	119	139	91	100	48	340
5	11.1	116	115	89	68	32	100
6	16.1	157	140	75	70	10	-160
7	10.3	103	130	85	115	54	250
8	12.0	125	82	80	75	32	90
9	9.9	115	91	65	95	43	115
10	14.8	126	65	63	41	28	-75
r		0.90	0.28	0.50	0.77	0.96	0.99
p		0.01	>0.05	>0.05	0.02	<0.001	<0.001

Lung volumes are expressed as percentage of the predicted value for the ideal body weight except for supine ERV-CV which is given in absolute values. Correlations between ΔSao₂ 30 and the other variables were analysed with the Spearman correlation method. r is the correlation coefficient. Abbreviations are the same as in table 1. IBW: ideal body weight.

Table 4. — Regression analysis between the desaturation surface between 10–30 s of apnoea (DS 10–30) and the different lung volumes in the sitting and supine position.

Variables	DS 10–30	
	r	p
Weight	0.77	0.02
Sitting FRC	0.25	>0.05
Supine FRC	0.56	>0.05
Sitting ERV	0.65	0.05
Supine ERV	0.87	0.005
Supine ERV-CV	0.89	0.002

r is the correlation coefficient. Abbreviations are the same as in table 1.

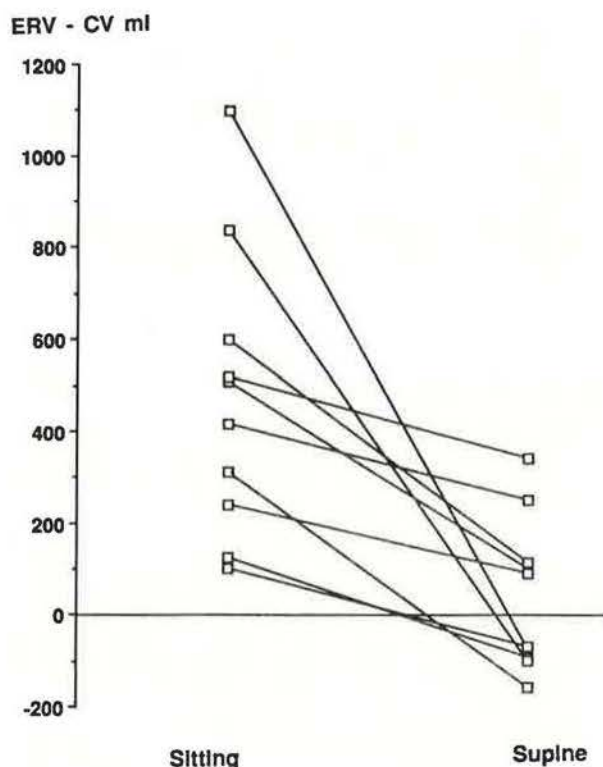


Fig. 2. — Influence of body position on lung volumes. For all subjects, the closing volume (CV) was below the expiratory reserve volume (ERV) in the sitting position (ERV-CV>0). Lying supine, ERV was close to CV in five subjects, and lower than CV in five others.

Discussion

Our results suggest that the depth of O₂ desaturations following an obstructive apnoea are largely influenced by lung volumes when the pre-apnoeic Sao₂ is greater than 90%.

Because we wanted to measure lung volumes in the sitting and supine positions, we could only use a helium dilution technique. Since none of our patients had obstructive lung disease, this technique can be considered

reliable. We have limited our study to obstructive apnoeas because these events were the most frequent in our patients, and we wanted to characterize the desaturations of one type of apnoea.

The baseline Sao₂ during sleep (Sao₂ value during normal breathing) was greater than or equal to 90% in all subjects; furthermore, we only considered obstructive apnoeas whose pre-apnoeic Sao₂ was greater than or equal to 90%. These criteria excluded the apnoeas whose pre-apnoeic Sao₂ value was on the steep part of the haemoglobin dissociation curve. Therefore, any influence of the pre-apnoeic Sao₂ level on the rate of desaturation was small.

For each patient, the post-apnoeic desaturations were evaluated by a desaturation curve. We believe that the determination of this curve accurately characterizes individual post-apnoeic desaturations; there was always a strong correlation between the apnoea duration and the secondary Sao₂ fall, O₂ desaturation increasing exponentially with apnoea length (fig. 1). From the equation of the desaturation curve, we calculated the Sao₂ fall after 30 s of apnoea and the desaturation surface between 10–30 s of apnoea. We considered the Sao₂ falls following 30 s of apnoea because the Sao₂ falls over this period of apnoea were always significant, and because the desaturations related to longer apnoeas could not be evaluated in some patients since it fell below 60%. The desaturation surface is interesting since it reflects the severity of the desaturation between 10–30 s during an apnoea and not only the Sao₂ fall at a particular apnoea duration.

We found a strong correlation between the ΔSao₂ 30, the DS 10–30 and the sitting ERV, the supine ERV and the ERV-CV difference. BRADLEY *et al.* [2] found that during SAS the main factors determining the mean nocturnal Sao₂ are the diurnal arterial oxygen partial pressure, the time spent in apnoea, and the ERV. Like these authors we did not find any significant correlation between the apnoea-related desaturation and the FRC; our results confirm the important role of the ERV in apnoea-related desaturations. The lack of correlation between FRC and the post-apnoeic Sao₂ falls can be explained by the influence of obesity (which tends to decrease FRC) and of small airways disease (which tends to increase residual volume and then FRC) on FRC value. This probably accounts for the stronger correlation between apnoea-related desaturation and ERV that we found.

We observed a very high correlation between ΔSao₂ 30 and the supine ERV-CV difference. This confirms that in SAS, apnoea-related desaturations largely depend on CV. Similar results were reported by FINDLEY *et al.* [3] who studied the desaturations following voluntary apnoeas at different lung volumes: when realized above closing capacity (CC) the desaturations were the same whatever the pre-apnoeic lung volume, but the post-apnoeic desaturation increased exponentially when the pre-apnoeic lung volume was below CC with an increasing rate of fall of Sao₂ during the apnoea. The increased severity in apnoea-related Sao₂ falls observed at low lung volumes can be explained by the combination of the small

quantity of O_2 present in the lungs and ventilation/perfusion mismatches [11, 12]. The pre-apnoeic lung volume represents the only oxygen store, and this will be reduced by any reduction in the lung volume, therefore increasing the rate of desaturation during an apnoea [3]. Furthermore, when FRC is below the CC, airways closure results in ventilation/perfusion mismatches and shunting, producing deeper SaO_2 falls [12]. This is in accordance with the results of STROHL *et al.* [13] who reported the importance of the pre-apnoeic SaO_2 level in the rate of oxygen saturation fall during an apnoea.

The important role of lung volumes in the rate of desaturation in SAS has been confirmed by several reports, a pulmonary inflation secondary to positive expiratory airways pressure [14] or negative extrathoracic pressure [15] being accompanied by an improvement in post-apnoeic desaturations. Continuous nasal airflow has been reported to decrease the severity of apnoea- and hypopnoea-related desaturations [16]; this can also be related to an increase in FRC.

We found that the sitting and supine ERV values were related to the degree of obesity. The ΔSaO_2 30 and the DS 10–30 were also correlated with weight, the rate of desaturation increasing with obesity. These results probably represent the effects of obesity on lung volume [5]. This could explain the effect of weight loss on the improvement in apnoea-related desaturation [17, 18].

The severity of the desaturations related to obstructive apnoeas whose pre-apnoeic SaO_2 is >90% appears to be closely dependent on lung volume, particularly with the supine ERV and the CV. These parameters can be considered as reliable indices in predicting the severity of post-apnoeic desaturations in SAS.

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Désaturation artérielle des apnées du sommeil. F. Sériés, Y. Cormier, J. La Forge.

RÉSUMÉ: Au cours du syndrome des apnées du sommeil (SAS), la sévérité des désaturations secondaires aux apnées du sommeil est variable d'un patient à l'autre. Il a été établi que les chutes de SaO_2 sont influencées par le volume pulmonaire auquel survient l'apnée. Nous avons évalué, chez 10 patients porteurs d'un SAS, avec quels volumes pulmonaires étaient le mieux corrélées les désaturations par apnée. Les volumes pulmonaires ont été mesurés par dilution à l'hélium en position assise et couchée, le volume de fermeture a été déterminé par respiration unique à l'azote. Pour chaque patient, la sévérité des désaturations a été caractérisée grâce à une courbe de désaturation représentant la chute de SaO_2 consécutive à chaque apnée enregistrée en fonction de la durée de l'apnée correspondante. La chute de SaO_2 après 30 secondes d'apnée (ΔSaO_2 30) et la surface de désaturation entre 10 et 30 secondes d'apnée (SD 10–30) ont été déterminées à partir de cette courbe de désaturation. La ΔSaO_2 30 et la SD 10–30 étaient en corrélation avec le volume de réserve expiratoire (VRE) mesuré en position assise ($r=0.77$ et 0.65 respectivement; $p<0.05$) et en position couchée ($r=0.96$ et 0.87 ; $p<0.005$); la ΔSaO_2 30 et la SD 10–30 étaient aussi corrélées avec la différence entre le VRE couché (VRE_c) et le volume de fermeture (VF) ($r=0.99$ et 0.89 ; $p<0.005$). La surcharge pondérale influençait la valeur de VRE dans chaque position et la chute de VRE accompagnant le passage en décubitus dorsal. Nous concluons que parmi les paramètres fonctionnels respiratoires, le VRE_c et le VRE_c-VF permettent le mieux de prévoir la sévérité des désaturations au cours du SAS.

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