

## Scientific Letter

**Effects of Posture on the Inspiratory and Expiratory Components of Vital Capacity in Healthy Humans**


To the Director,

Comparing sitting and supine vital capacity (VC) helps identifying diaphragm dysfunction.<sup>1</sup> A 20–30% postural fall in VC ( $\Delta$ VC) suggests bilateral diaphragm weakness,<sup>2–4</sup> however, the  $\Delta$ VC threshold differentiating the normal from the pathological is ill-established. A 15%  $\Delta$ VC, chosen as representing twice the coefficient of variation of the measure in healthy subjects, has been proposed as the upper limit of normal,<sup>1</sup> seems appropriate to identify bilateral diaphragm paralysis<sup>2</sup> but may not be discriminatory enough to detect unilateral diaphragmatic paralysis.<sup>1–3</sup> This may stem from other inspiratory muscles compensating for diaphragm dysfunction, hence a preserved inspiratory capacity (IC).<sup>5</sup> The diaphragm, in addition to its inspiratory function, protects the lungs from compression by the abdominal content.<sup>6</sup> This protective purpose is compromised by diaphragm atrophy,<sup>6</sup> leading to infer that the abdominal content's cephalad movement when lying supine should reduce end-expiratory volume disproportionately in patients with diaphragmatic dysfunction, hence the corresponding reduction in expiratory reserve volume (ERV).<sup>7</sup> Yet gravity-related postural reductions in end-expiratory volume also occur in normal individuals.<sup>8</sup> Of note, standing upright involves applying a gravity-driven caudal force to abdominal viscera that could also modify lung volume and respiratory mechanics respective to their sitting values. Although it is usually accepted that the corresponding variations in lung volumes are trivial,<sup>9,10</sup> forced expiratory volumes are frequently higher when standing.<sup>11</sup> We therefore hypothesized that in normal individuals (1) sitting-to-supine postural variations in ERV, IC, or both (currently not described) would exceed, in percentage, the corresponding global VC changes; (2) standing-to-supine differences in VC, ERV and IC would deviate from sitting-supine such differences.

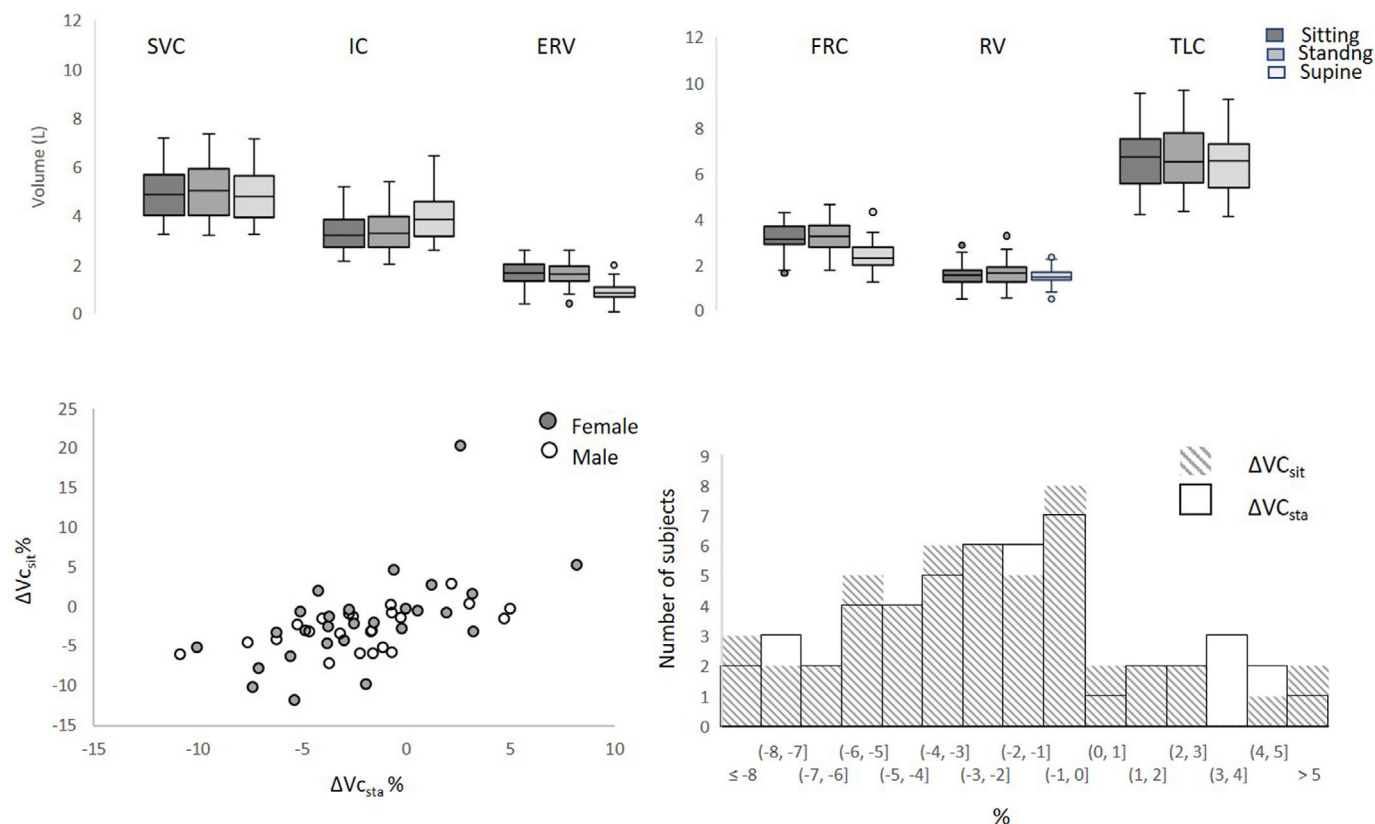
This study was approved by the *Comité de Protection des Personnes Ile-de-France VI*. Participants provided written consent. Inclusion criteria were: age over 18, non-obese, non-smoker, no history of respiratory, cardiac or neuromuscular disease, normal clinical examination. Lung function tests were performed according to the current recommendations and reference values of the *European Respiratory Society*<sup>10,12,13</sup> in sitting, standing and supine positions in a random order (Fig. 1, Table 1) using a SpiroAir device running the Exp'air 1.28.20 software (Medisoftware, Belgium). Total lung capacity (TLC) and residual volume (RV) were calculated using the measurement of functional residual capacity (FRC) using the Helium dilution technique<sup>13</sup> and a slow VC maneuver with TLC

equal to FRC plus IC and RV equal to FRC minus ERV. Maximal inspiratory (MIP) and expiratory (MEP) pressures and sniff nasal inspiratory pressure (SNIP) were measured while sitting,<sup>1,14,15</sup> to rule out respiratory muscle dysfunction. Data are summarized as median and ([Q1; Q3]). A Friedman non-parametric test compared the three positions ( $p < 0.05$  for all volumes variations). Then sitting and standing positions were separately compared to the supine position using a Wilcoxon's signed rank test, giving access to  $\Delta$ VC<sub>sit</sub>,  $\Delta$ IC<sub>sit</sub> and  $\Delta$ ERV<sub>sit</sub> (sitting-to-supine variations) and  $\Delta$ VC<sub>sta</sub>,  $\Delta$ IC<sub>sta</sub> and  $\Delta$ ERV<sub>sta</sub> (standing-to-supine variations). The significance level was set to 0.05.

Fifty participants (34 years [26; 48]; 22 female, 172 cm [165; 176]; 71 kg [61; 79]; 24 kg/m<sup>2</sup> [21; 26]) were included and the complete dataset was analyzed (no missing data) (Table 1). All measured variables were within their normal range, including forced expiratory volume in 1 s (FEV<sub>1</sub>) (3.84 L [3.23; 4.54]; 109% predicted [101; 117]), FEV<sub>1</sub>/SVC (77% [71; 82]), MIP (104 cm H<sub>2</sub>O [83; 128], 99% predicted [83; 121]), MEP (153 cm H<sub>2</sub>O [129; 180], 80% predicted [68; 90]), SNIP (97 cm H<sub>2</sub>O [79; 115], 94% predicted [87; 111]).

FRC significantly decreased by a median 25.8% (0.79 L) from sitting to supine and 27.3% (0.86 L) from standing to supine. VC also decreased significantly compared to sitting VC or standing VC (Table 1, Fig. 1), but  $\Delta$ VC<sub>sit</sub> and  $\Delta$ VC<sub>sta</sub> remained low, with a median reduction of –2.4% only for both and a maximal reduction never exceeding –12% (Fig. 1). None of the participants had both  $\Delta$ VC<sub>sit</sub> and  $\Delta$ VC<sub>sta</sub> exceeding –8% (Fig. 1). The conjunction of large FRC changes and small VC changes resulted in supine ERV and IC changes being noticeably more marked than their VC counterparts. Likewise, ERV significantly decreased in the supine position (median  $\Delta$ ERV<sub>sit</sub> –43.9%; median  $\Delta$ ERV<sub>sta</sub> –46.7%) while IC significantly increased (median  $\Delta$ IC<sub>sit</sub> 18.9%; median  $\Delta$ IC<sub>sta</sub> 18.7%). Expressed in volume, the IC increases exactly compensated the ERV decreases, explaining the stability of VC (median  $\Delta$ ERV<sub>sit</sub> +  $\Delta$ IC<sub>sit</sub> = –110 mL to be compared with median  $\Delta$ VC<sub>sit</sub> = –110 mL; (median  $\Delta$ ERV<sub>sta</sub> +  $\Delta$ IC<sub>sta</sub> = –90 mL to be compared with  $\Delta$ VC<sub>sta</sub> = –90 mL).

This study shows that lying supine markedly modifies the expiratory and inspiratory components of VC, in relationship with the postural decrease in FRC. Opposite ERV and IC changes result in a quasi-preserved VC. We observed a supine reduction in FRC of roughly 25%, amounting to an approximate median of 0.8 L. This corresponds to observations made in healthy humans long ago<sup>4,16</sup> and multiple times.<sup>8</sup> We also confirmed that standing and sitting lung volumes were comparable (with individual differences) and that  $\Delta$ VC was minimal between these two positions.<sup>8</sup> The maximal  $\Delta$ VC recorded in our participants was –12%, which is below the 15% threshold proposed to suspect an anomaly in the context



**Fig. 1.** Lung volumes according to posture. Above: slow vital capacity; IC: inspiratory capacity; ERV: expiratory reserve volume; FRC: functional residual capacity; RV: residual volume; TLC: total lung capacity.  $p < 0.05$  Wilcoxon's signed rank test for all comparisons excepted for RV sitting–supine. Below:  $\Delta VC_{sit}$ : variation of vital capacity between supine and sitting positions;  $\Delta VC_{sta}$ : variation of vital capacity between supine and sitting positions; left: corresponding values of  $\Delta VC_{sit}$  and  $\Delta VC_{sta}$  by participant in females and males (standing–sitting  $-0.04$  L [ $-0.14$ ;  $0.05$ ] in females and  $0.03$  [ $-0.13$ ;  $0.17$ ] in males (ns, Mann–Whitney test); right: distribution of  $\Delta VC_{sit}$  and  $\Delta VC_{sta}$  per 1% class.

**Table 1**  
Lung Function and Lung Volume Variations According to Posture.

Lung Function		Lung Volume Variations According to Posture		
Sitting Position			Sitting–Supine ( $\Delta_{sit}$ )	Standing–Supine ( $\Delta_{sta}$ )
FRC (L)	3.13 [2.90; 3.69]	(L)	-0.79 [-0.92; -0.63]*	-0.86 [-1.04; -0.58]*
% pred	103 [92; 112]	% variation	-25.8 [-29.5; -19.6]	-27.3 [-31.2; -20.4]
SVC (L)	4.88 [4.00; 5.67]	(L)	-0.11 [-0.24; -0.03]*	-0.09 [-0.23; -0.01]*
% pred	114 [104; 125]	% variation	-2.4 [-5.0; -0.6]	-2.4 [-4.5; -0.2]
IC (L)	3.21 [2.71; 3.87]	(L)	0.59 [0.36; 0.76]*	0.56 [0.34; 0.80]*
		% variation	18.9 [10.8; 23.8]	18.7 [11.7; 28.0]
TLC (L)	6.72 [5.57; 7.52]	(L)	-0.16 [-0.30; 0.08]*	-0.29 [-0.49; -0.04]*
% pred	103 [97; 114]	% variation	-2.3 [-5.8; 1.1]	-4.4 [-6.9; -0.7]
ERV (L)	1.67 [1.32; 2.02]	(L)	-0.75 [-0.91; -0.56]*	-0.69 [-0.94; -0.47]*
		% variation	-43.9 [-54.3; -36.8]	-46.7 [-55.0; -36.0]
RV (L)	1.54 [1.23; 1.78]	(L)	-0.04 [-0.21; 0.17]	-0.16 [-0.42; 0.03]*
% pred	88 [73; 96]	% variation	-4.5 [-10.5; 10.2]	-9.0 [-23.6; 2.1]

FRC: functional residual capacity; SVC: slow vital capacity; IC: inspiratory capacity; TLC: total lung capacity; ERV: expiratory reserve volume; RV: residual volume.  
\*  $p < 0.05$  Wilcoxon's signed rank test for sitting versus supine of standing versus supine.

of suspected diaphragm dysfunction.<sup>1</sup> In addition, when  $\Delta VC_{sta}$  fell between  $-8\%$  and  $-12\%$ ,  $\Delta VC_{sit}$  was always of  $-8\%$  or less, and vice versa. This means that considering both  $\Delta VC_{sta}$  and  $\Delta VC_{sit}$  can bring the lower limit of normal  $\Delta VC$  to  $-8\%$ . Nevertheless, establishing a  $\Delta VC$  threshold below the proposed  $15\%$  value<sup>1</sup> may still not suffice to identify unilateral diaphragmatic palsy. In a study where  $\Delta VC_{sit}$  was tested against ultrasound indicators of diaphragm dysfunction, no single threshold of  $\Delta VC$  could accurately predict unilateral diaphragm dysfunction.<sup>2</sup>

A thorough analysis of the  $\Delta VC$  literature indicates that the differential effects of postural changes on the expiratory and inspiratory components of VC have not been described before in normal individuals. Our study, therefore, brings novel information

in this respect. In our participants, the supine drops in FRC and the supine drop in ERV – of similar magnitudes – likely resulted from the gravity-related cephalad movement of the abdominal content displacing the diaphragm cranially despite its elasticity and tonic activity.<sup>6</sup> Such a decrease in lung volume is associated with a deteriorated respiratory system impedance that proceeds from reduced lung compliance<sup>6,17,18</sup> increased bronchial and upper airway resistance.<sup>9,11,19</sup> Our participants were, however, able to maintain or near-maintain VC in the supine posture through compensatory increases in IC. Therefore, in their cases, the strength of the inspiratory muscles – diaphragm included – was sufficient to overcome the posture-related added inspiratory load. Such a finding is not surprising giving the large reserve that exists between

the maximal inspiratory strength available and the strength necessary to bring normal lungs to their TLC.<sup>1,20</sup> In summary, in supine normal humans, inspiratory muscle recruitment allows VC to be maintained despite the corresponding fall in FRC.

A study conducted in 13 patients with diaphragm dysfunction showed that the occurrence of orthopnea coincided with that of expiratory flow limitation, leading the authors to consider the postural deterioration of respiratory mechanics as possibly causative of supine respiratory discomfort.<sup>7</sup> In this study, the patients exhibited a median supine ERV reduction of 35%, similar to that we observed in our healthy population. However, the supine ERV reductions in the diaphragm weakness patients occurred on top of reduced sitting FRC values (leading some patients to exhibit a nearly zero ERV) that probably resulted from an atrophy-related loss of the barrier function of the diaphragm. In addition, contrary to what occurred in our participants, the diaphragm weakness patients did not exhibit compensatory changes in IC in the supine posture, probably because of a diaphragm weakness-related insurmountable load-capacity imbalance.

From the above, we submit that the value of supine spirometry to infer diaphragm dysfunction should be revisited and go beyond the mere determination of  $\Delta VC$ . We acknowledge that this should be verified on a large cohort of patients with well documented diaphragmatic weakness.

#### Authors' Contributions

Conception and design of the study: VA, PL, CS, TS.

Data acquisition: VA, PL, RV, BS, CS, TS.

Analysis of the data: VA, PL, RV, BS, CS, TS.

VA, PL and TS drafted the initial manuscript. VA, PL, RV, BS, CS, TS contributed to the data interpretation and edited the manuscript for important scientific content. All the authors agree to be accountable for all aspects of the work in regard to accuracy and integrity.

#### Artificial Intelligence Involvement

Not applicable.

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#### Conflict of Interest

The authors declare not to have any conflicts of interest that may be considered to influence directly or indirectly the content of the manuscript.

#### References

1. Laveneziana P, Albuquerque A, Aliverti A, Babb T, Barreiro E, Dres M, et al. ERS statement on respiratory muscle testing at rest and during exercise. *Eur Respir J*. 2019;53(6).
2. Brault M, Gabrysz-Forget F, Dube BP. Predictive value of positional change in vital capacity to identify diaphragm dysfunction. *Respir Physiol Neurobiol*. 2021;289:103668. Epub 2021/04/04.

3. Laroche CM, Mier AK, Moxham J, Green M. Diaphragm strength in patients with recent hemidiaphragm paralysis. *Thorax*. 1988;43:170–4.
4. Allen SM, Hunt B, Green M. Fall in vital capacity with posture. *Br J Dis Chest*. 1985;79(3):267–71. Epub 1985/07/01.
5. Attali V, Mehiri S, Straus C, Salachas F, Arnulf I, Meininger V, et al. Influence of neck muscles on mouth pressure response to cervical magnetic stimulation. *Am J Respir Crit Care Med*. 1997;56:509–14.
6. Perry SF, Similowski T, Klein W, Codd JR. The evolutionary origin of the mammalian diaphragm. *Respir Physiol Neurobiol*. 2010;171(1):1–16. Epub 2010/01/19.
7. Redolfi S, Straus C, Ninane V, Similowski T. Postural lung volume reduction, expiratory flow limitation, and orthopnea in diaphragmatic weakness: preliminary observations. *Pulmonology*. 2023. Epub 2023/09/24.
8. Katz S, Arish N, Rokach A, Zaltzman Y, Marcus EL. The effect of body position on pulmonary function: a systematic review. *BMC Pulm Med*. 2018;18(1):159. Epub 2018/10/12.
9. Badr C, Elkins MR, Ellis ER. The effect of body position on maximal expiratory pressure and flow. *Aust J Physiother*. 2002;48(2):95–102.
10. Miller MR, Crapo R, Hankinson J, Brusasco V, Burgos F, Casaburi R, et al. General considerations for lung function testing. *Eur Respir J*. 2005;26(1):153–61. Epub 2005/07/05.
11. Castile R, Mead J, Jackson A, Wohl ME, Stokes D. Effects of posture on flow-volume curve configuration in normal humans. *J Appl Physiol Respir Environ Exerc Physiol*. 1982;53(5):1175–83. Epub 1982/11/01.
12. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen R, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. *Eur Respir J*. 1993;6 Suppl. 16:5–40.
13. Wanger J, Clausen JL, Coates A, Pedersen OF, Brusasco V, Burgos F, et al. Standardisation of the measurement of lung volumes. *Eur Respir J*. 2005;26(3):511–22. Epub 2005/09/02.
14. Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationship to age and sex. *Am Rev Respir Dis*. 1969;99(5):696–702. Epub 1969/05/01.
15. Uldry C, Fitting J-W. Maximal values of sniff nasal inspiratory pressure in healthy subjects. *Thorax*. 1995;50:371–5.
16. Moreno F, Lyons HA. Effect of body posture on lung volumes. *J Appl Physiol*. 1961;16(1):27–9.
17. Estenne M, Yernault JC, De Troyer A. Rib cage and diaphragm-abdomen compliance in humans: effects of age and posture. *J Appl Physiol*. 1985;59(6):1842–8.
18. Lim TP, Luft UC. Alterations in lung compliance and functional residual capacity with posture. *J Appl Physiol*. 1958;14(2):164–6.
19. Wallace JL, George CM, Tolley EA, Winton JC, Fasanella D, Finch CK, et al. Peak expiratory flow in bed? A comparison of 3 positions. *Respir Care*. 2013;58(3):494–7. Epub 2012/08/22.
20. De Troyer A, Borenstein S, Cordier R. Analysis of lung volume restriction in patients with respiratory muscle weakness. *Thorax*. 1980;35(8):603–10.

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