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Impact of non-invasive ventilation on lung hyperinflation: A physiological study using electrical impedance tomography in COPD patients

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Abstract

High-pressure non-invasive ventilation (NIV) is the standard treatment for chronic obstructive pulmonary disease (COPD) patients with chronic hypercapnic respiratory failure. While the risk of lung hyperinflation under high-pressure ventilation is well established in acute settings, its impact in stable COPD patients remains unknown. The objective of this study is to assess the impact of NIV on lung volume in stable COPD using variation of end-expiratory lung impedance (EELI), a surrogate of functional residual capacity, obtain with electrical impedance tomography (EIT). During this prospective physiological study, we enrolled 30 patients, with a median forced expiratory volume in 1 second of 25% predicted [21–31]. There was a non-significant reduction in EELI with median changes of -50% [–115 to 51], p = 0.006, between the first and last minute of NIV. 17 patients (57%) had a decrease in EELI, and 12 patients (40%) had an increase in EELI, one patient (3%) showed no change in EELI. Patients who experienced a decrease in EELI under NIV reported better sleep quality and a more pronounced reduction in dyspnea.

Our data suggest that NIV can induce a reduction in lung volume, which is associated with improved sleep quality, a more pronounced reduction in dyspnea.

INTRODUCTION

Non-invasive ventilation (NIV) is now the standard treatment for chronic obstructive pulmonary disease (COPD) patients with severe chronic hypercapnic respiratory failure (1–3).

At present, high-intensity home NIV aimed at normalizing PaCO2 remains the standard treatment. However, in the intensive care setting during an acute exacerbation, high-pressure, high-volume ventilation has been associated with harm, especially an increased risk of hyperinflation (4–7), which has never been assessed using high intensity home NIV at a stable state.

Indeed, currently there is no direct analysis of hyperinflation during NIV. The expiratory flow limitation on screen curves from the ventilator remains the best way to follow it. Regarding chronic homecare ventilation, additional tools are needed, as these curves are not always available in real-time and their analysis lack of standardization or metric value. Electrical impedance tomography (EIT) has recently been used to assess changes in lung recruitment and ventilation distribution in patients receiving high-flow nasal therapy and NIV (8–10). EIT is a non-invasive bedside monitoring technique that enables real-time evaluation of changes in lung aeration and ventilation (11). It is a validated and reliable tool, demonstrating strong correlations with established reference methods (12,13). In particular, end-expiratory lung impedance (EELI) closely correlates with end-expiratory lung volume (EELV) and, indirectly, with functional residual capacity (FRC) (14). As lung hyperinflation is defined by the volume of air trapped in the thorax at the end of expiration—such as FRC—EIT represents an innovative approach for dynamically assessing hyperinflation, including during NIV sessions.

The objectives of this study was to evaluate if during an NIV session, EIT could detect changes in lung volume and therefore hyperinflation in stable COPD patients receiving long-term NIV and to explore how

it is associated with changes in ventilatory pattern and clinical features.

We hypothesized that EIT can reliably monitor dynamic changes in lung volume in stable COPD patients receiving long-term NIV and provide novel insights into high-pressure NIV and its impact on hyperinflation in this population.

RESULTS

Baseline characteristics

Between July 2023 and April 2024, 30 patients were included in the study, with 70% of women. Their characteristics are summarized in Table 1. The median age was 65 years [59–72]. Patients exhibited severe airflow obstruction, with a median forced expiratory volume in 1 s (FEV_1) of 25% predicted [21–31], and marked static hyperinflation, as indicated by a median residual volume (RV) of 222% predicted [186–275] and an inspiratory capacity/total lung capacity (IC/TLC) ratio of 17% [12–23]. The median daytime $PaCO_2$ was 51 mmHg [45–59].

Table 1
Patient characteristics

n	Total population	EELI increase	EELI decrease	p
	30	**12**	**17**	
Age	65 [59–72]	65 [62–73]	64 [67–71]	0.56
Sex, female	21 (70)	6 (50)	14(82)	
BMI (kg/m²)	24 [18–28]	23 [16–25]	24 [20–28]	0.21
Blood gas analysis				
pH	7.40 [7.37–7.43]	7.42 [7.41–7.43]	7.37 [7.36–7.41]	**0.03**
PaO_2 (mmHg)	67 [61–76]	65 [67–69]	71 [62–80]	0.25
$PaCO_2$ (mmHg)	51 [45–59]	52 [45–53]	52 [47–62]	0.34
HCO_3^- (mmol/L)	33 [30–36]	33 [30–34]	33 [30–36]	0.72
Pulmonary function test results				
FEV1 (% of predicted value)	25 [21–31]	24 [21–30]	25 [21–32]	0.89
FEV1 (mL)	540 [387–713]	685 [500–770]	510 [360–660]	0.17
FEV1/FVC (%)	34 [39–41]	35 [27–39]	33 [29–39]	0.87
TLC (% of predicted value)	127 [108–136]	117 [98–126]	133 [126–148]	**0.01**
TLC (mL)	6475 [4990–7822]	5470 [4545–8055]	6530 [5490–7290]	0.53
FRC (mL)	4980 [4110–6600]	4930 [3792–6630]	5150 [4430–6280]	0.66
FRC (% of predicted value)	183 [137–217]	163 [164–196]	195 [178–242]	0.10

One patient showed no change in EELI during NIV and was therefore not classified in either the EELI increase or decrease EELI group.

Data are presented as median [IQR 25%–75%] for continuous variables and as n (%) for categorical variables.

BMI: body mass index; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; TLC: total lung capacity; VC: vital capacity; FRC: functional residual capacity; IC: inspiratory capacity; RV: residual volume; EELI: end-expiratory lung impedance.

P-values indicate comparisons between the EELI increase and EELI decrease groups using Fisher's exact test for categorical variables, and the Mann–Whitney U test for continuous variables.

n	Total population	EELI increase	EELI decrease	p
	30	**12**	**17**	
IC/TLC (%)	17 [12–23]	20 [16–23]	14 [11–20]	0.18
RV (% of predicted value)	222 [186–275]	207 [187–252]	255 [201–282]	0.22
RV (mL)	4500 [3335–5823]	4300 [3205–5747]	4740 [3840–6080]	0.60
One patient showed no change in EELI during NIV and was therefore not classified in either the EELI increase or decrease EELI group.				
Data are presented as median [IQR 25%–75%] for continuous variables and as n (%) for categorical variables.				
BMI: body mass index; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; TLC: total lung capacity; VC: vital capacity; FRC: functional residual capacity; IC: inspiratory capacity; RV: residual volume; EELI: end-expiratory lung impedance.				
P-values indicate comparisons between the EELI increase and EELI decrease groups using Fisher's exact test for categorical variables, and the Mann–Whitney U test for continuous variables.				

NIV parameters are presented in Table 2. Patients had a median nightly NIV usage of 8 h [6–9], with no evidence of obstructive events (AHI: 0.2 events/h [0.0–1.0]) or significant leaks (1 L/min [0–6]). The median PS was 14 cmH_2O [11–16], with a median IPAP of 22 cmH_2O [19–24], enabling adequate control of nocturnal $PTcCO_2$ (median $PTcCO_2$: 50 mmHg [44–55]). The median PSQI score was 6 [4–11]. A significant reduction in $PTcCO_2$ was observed under NIV, decreasing from 53.5 mmHg [47–58.5] at baseline to 46 mmHg [49.5–52] during NIV ($p < 0.001$) (Figure S-1). Finally, patients experienced a significant decrease in BORG scale during NIV, from 4 [3–6] before NIV to 1 [0–2] under NIV, with a median decrease of -3 [-4– -6] ($p < 0.001$).

Table 2
NIV parameters and monitoring data

n	Total population	EELI increase	EELI decrease	p
	30	**12**	**17**	
NIV monitoring data				
PS (cmH_2O)	14 [11–16]	13 [11–14]	14 [13–16]	0.12
IPAP (cmH_2O)	22 [19–24]	21 [18–22]	21 [20–24]	0.20
EPAP (cmH_2O)	8 [7–9]	8 [7–8]	8 [7–8]	0.86
Use (h)	8 [6–9]	7 [6–9]	8 [7–9]	0.60
AHI (/h)	0.2 [0.0–1.0]	0.2 [0.0–0.9]	0.2 [0.0–1.0]	0.77
Fractional use	11 (37)	5 (17)	6 (21)	0.73
Median leak (L/min)	1 [0–6]	0 [0–1]	3 [0–24]	**0.04**
Nocturnal $PTcCO_2$ (mmHg)				
Mean	50 [44–55]	51 [46–54]	48 [40–55]	0.76
Max	58 [50–63]	57 [50–61]	59 [50–65]	0.70
PSQI	6 [4–11]	9 [6–12]	2 [2–4]	**0.03**
BORG scale	3 [-4– -6]	−1 [−3 – 0]	−4 [−5 - – 3]	**0.001**

One patient showed no change in EELI during NIV and was therefore not categorized in either the EELI increase or decrease group.

Data are presented as median [IQR 25%–75%] for continuous variables and as n (%) for categorical variables. Use, AHI, and median leaks are presented as averages over the last 30 days. Fractional use is defined as two or more treatment interruptions during the night on > 50% of nights over the past 30 days.

NIV: non-invasive ventilation; PS: pressure support; IPAP: inspiratory positive airway pressure; EPAP: expiratory positive airway pressure; AHI: apnea–hypopnea index; PSQI: Pittsburgh Sleep Quality Index; EELI: end-expiratory lung impedance.

P-values represent comparisons between the EELI increase and EELI decrease groups using Fisher's exact test for categorical variables, and the Mann–Whitney U test for continuous variables.

EELI variation ventilatory pattern

The evolution of EELI under NIV is presented in Fig. 2. There was a reduction in EELI at every timepoint, with median changes of -21% [−67 − 16] at T2 (p = 0.064), -50% [−98 − 5] at T3 (p = 0.035), -39% [−106 −

36] at T4 (p = 0.146), -59% [–100 – 66] at T5 (p = 0.262) and – 50% [–115 to 51] at T6 the end of the session without statistical significance (p = 0.221).

Between T1 and T6, patients exhibited a significant decrease in TIV (ΔTIV: -21% [–28 to – 10], p < 0.001) and RR (ΔRR: – 10% [–18 to 0], p = 0.038) but with a significant increase in Te (ΔTe: +16% [3–26], p = 0.003), and Ti stay stable (ΔTi: +8% [-4–18], p = 0.084).

A one-unit increase in Te was associated with a -2.8 unit change in EELI (95%CI -4.8 to -0.9, p = 0.006 ; Fig. 3A) and one-unit increase in RR was associated with a 4.3 unit change in EELI (95%CI 1.5 to 6.8, p = 0.003 ; Fig. 3B)). No significant association was observed between ΔTIV and EELI (-1.8 (95%CI -4.1 to -0.4), p = 0.108).

Analysis of individual data revealed substantial variability in EELI responses among participants, ranging from a 327% increase to a – 414% decrease during the NIV session. Therefore, regarding EELI evolution 2 distinct groups emerged: those with a positive ΔEELI at T5 and T6 (n = 12 (40%), 83% [43–130]) and those with a decrease in lung volume (negative ΔEELI at T5 and T6) (n = 17 (57%), -104% [-164– -60]) (Fig. 2). One patient could not be categorized with a negative ΔEELI at T5 and a positive ΔEELI à T6. When comparing the characteristics of these two groups, patients with a decrease in EELI had significantly higher TLC values (133% [126–148] vs. 117% [98–126], p = 0.010) and better sleep quality, as indicated by lower PSQI scores (2 [1–4] vs. 9 [6–12], p = 0.030) (Table 1).

There were no significant differences between the groups in ventilator settings, ventilation quality, nocturnal alveolar hypoventilation (Table 4), or the effectiveness of NIV in correcting alveolar hypoventilation during the recorded session, as measured by the reduction in $PTcCO_2$ (–7.5 mmHg [–11.5 to – 4.5] in the EELI decrease group vs. – 8 mmHg [–11 to – 5] in the EELI increase group, p = 0.930). But there was a significantly better reduction in dyspnea in the group of patients who exhibited a decrease in EELI compared to those with an increase (–4 [–5 - – 3] vs. – 1 [–3 – 0], p = 0.001).

DISCUSSION

This study confirms that EIT can monitoring dynamic changes in lung volume in stable COPD patients performing NIV session. It is also possible to evaluate ventilatory parameters such as RR, reflects of tidal volume, Ti and Te. We showed that under NIV, there is a great variation in lung volume evolution among patients and some have a reduction of lung volume. In a second time, we found that a reduction of lung volume during NIV is associated with a greater dyspnea relief, a greater quality of sleep and also a reduction of RR and an increase of Te. Although these preliminary data suggest that EIT appears as a promising tool to evaluate lung volume under NIV, which might be useful for clinical adaptation, some points need to be discussed.

Regarding our study population and settings, our cohort consisted of hyperinflated COPD patients with severe alveolar hypoventilation, comparable to those reported in prior studies of COPD patients receiving NIV (1,2,15). However, the proportion of women in our study was slightly higher than that reported in

previous studies or French national databases (2,22), for which we were unable to determine the reason for this discrepancy. The hypoventilation in our cohort was somewhat less severe (median $PaCO_2$: 51 mmHg) compared to that reported by Köhnlein et al. (59 mmHg) (1) and Murphy et al. (58 mmHg) (2). This difference is likely attributable to the fact that our patients were already stable on NIV, with treatment initiated at least 6 months prior to inclusion. Indeed, the level of alveolar hypoventilation in our cohort was higher than that reported in a recent study of patients on NIV (median $PaCO_2$: 47 mmHg) (23), and similar to the levels observed 6 months after NIV initiation in other studies (15).

The reduction of hyperinflation under NIV has been a topic of interest since the 1990s, when it was first hypothesized (24). As lung hyperinflation is characterized by the volume of air trapped in the thorax at the end of expiration, EELI may represents an innovative metric for its dynamic assessment (14). Regarding our results, the decreases in EELI observed under NIV might be a marker of reduction in hyperinflation. This is supported by the association of EELI reduction and RR reduction and Te increase. Te increase is a well-recognized physiological mechanism, as one of the primary contributors to lung hyperinflation is incomplete lung emptying (25). Reducing RR and prolonging Te are established strategies for mitigating hyperinflation.

In addition to its impact on lung volumes, our findings offer important clinical insights by demonstrating a clear benefit of NIV on quality-of-life indicators such as dyspnea and sleep quality—effects that extend beyond the correction of alveolar hypoventilation in patient with a reduction in lung volume under NIV. Although reducing alveolar hypoventilation remains a well-established and essential goal in the management of COPD patients on NIV, our results suggest that targeting lung volume reduction may represent an additional key objective to further enhance patient comfort and overall quality of life. Regarding clinical phenotypes, patients who had a decrease of EELI under NIV have a higher TLC, which is the marker of an increase static hyperinflation. In these patients, NIV may provide a beneficial effect through the reopening and recruiting of distal (small) airways, a mechanism previously suggested in the literature for many years (26).

Although our results support the hypothesis that there is a link between RR reduction and lung volume reduction, this study is only observational. We did not perform comparison analysis between different NIV settings, such as IPAP and EPAP at different levels, and lung volume evolution. Indeed, some patients did not exhibit a decrease in EELI under NIV, but this may be related to suboptimal ventilator settings—particularly an insufficiently high IPAP, which may have failed to improve the load-capacity balance and adequately reduce RR (26). With regard to EPAP, patients with an EPAP set above or close to their intrinsic positive expiratory pressure may experience increased dynamic hyperinflation (27). Suh et al. (28) demonstrated that EPAP titration in COPD patients reduced neural respiratory drive, an indirect marker of lung hyperinflation, with considerable variability in optimal EPAP levels ranging from 4 to 13 cmH_2O. In our study, a higher range of EPAP was used which are consistent with those reported in other cohorts of COPD patients on NIV (29). However, we acknowledge that our study did not include data on the specific effect of EPAP on EELI in the stable state, particularly regarding the potential risk of increasing lung hyperinflation under NIV (27). As we were unable to determine whether EELI variations

were driven by NIV settings or COPD phenotypes, further research is needed to explore the impact of NIV setting adjustments on hyperinflation.

Another parameter not assessed in this study is the presence of asynchronies, particularly ineffective efforts that are common in COPD patients. Under NIV, the risk of asynchrony is considerable and is frequently associated with a decline in quality of life (30). In COPD patients, reducing lung hyperinflation can help lower the risk of ineffective efforts (5). A personalized approach to setting adjustments—based on the analysis of ventilation and flow–pressure curves, as recommended by the SOMNONIV working group (16)—may allow for more precise optimization and a potentially greater reduction in lung hyperinflation. Further studies are therefore warranted to determine whether individualized PS ventilation, potentially guided by EELI monitoring, could enhance the reduction of lung hyperinflation in a broader population of COPD patients receiving NIV.

Our study has also several limitations related to EIT imaging. First, EIT only provides analysis of a transversal thorax section, depending on the belt position, it represents approximatively 50% of the lung surface ; we positioned the thoracic electrode belt according to the literature to obtain the largest possible lung area for the analysis of lung volume variations (20,21). Additionally, several studies previously reported a strong correlation between lung volume variations measured with gold-standard technics and those observed by EIT (14,31). However, as severe COPD patient may exhibit strong heterogeneity in lung structure (i.e emphysema, atelectasis), some hyperinflated lung regions may have been missed by the imaging analysis that potentially affect the overall impact of NIV on EELI. This remains a current limit of EIT. As far as we know, there is no minimal clinically important difference (MCID) of the EELI metrics to precisely estimate the clinical impact of our results in stable COPD patient performing NIV session. EIT provides other interesting metrics, such as ventilation homogeneity changes or regional expiratory time constant analysis, which we were not able to exploit at present. This should be further explored in the future.

Finally, it is important to acknowledge that these data were collected during an afternoon NIV session and may not be fully generalizable to nighttime NIV, which is influenced by various physiological changes. However, the observed improvement in sleep quality among patients who showed a decrease in EELI suggests a possible nighttime effect as well—potentially reflecting a similar reduction in lung hyperinflation during sleep, consistent with the findings of our study.

CONCLUSION

This exploratory study demonstrates that EIT can monitoring dynamic changes in lung volume and ventilatory patterns in stable COPD patients performing NIV session. Our data suggest that high-pressure NIV can induce a reduction in lung volume. This reduction is associated with improved sleep quality, a more pronounced reduction in dyspnea, and a greater reduction in respiratory rate (RR) during NIV in almost of patients. The use of EIT monitoring to guide individualized NIV settings warrants further

investigation, as real-time assessment of lung volume may represent the missing link in understanding and optimizing the beneficial effects of chronic NIV in COPD patients.

METHODS

This prospective, observational, physiological, single-center pilot study was conducted in the Pulmonology Department of Bordeaux University Hospital between July 2023 and April 2024.

This study was performed in accordance with the Declaration of Helsinki. This human study was approved by Ethics Committee CPP OUEST IV – NANTES - approval: 2023-A00975-40. All participants received information on the study and provided written informed consent prior to enrollment.

All consecutive patients admitted for their biannual assessment in our outpatient clinic were screened and prospectively included. Patients with COPD receiving long-term NIV, were eligible for inclusion if their treatment had remained stable for at least 6 months, defined as no adjustments to therapy during that period.

Patients were excluded if they were under 18 years of age, had a body mass index (BMI) > 35 kg/m², were experiencing for an exacerbation, or had contraindications to EIT use, such as implanted electrical medical devices, damaged skin, or the presence of dressings (12).

The monitoring and adjustment of long-term ventilation were conducted in accordance with current guidelines (3). All patients were treated with NIV in S/T mode. Inspiratory support was titrated to achieve at least a 20% reduction in alveolar hypoventilation or its normalization (1,15), the absence of nocturnal alveolar hypoventilation, as assessed by transcutaneous capnography ($PTcCO_2$) (2), daily use exceeding 4 h, absence of unintentional leaks, obstructive events, and optimal patient–ventilator synchronization (16,17).

The primary objective of this study was to describe lung volumes evolution under NIV. The primary outcome measure was defined as the change in EELI (ΔEELI) during NIV session (as described below).

Secondary objectives were to evaluate the association between EELI evolution and changes in the ventilatory pattern during the NIV session (characterized by the respiratory rate (RR), tidal volume (Vt), and inspiratory (Ti) and expiratory (Te) times assessed by the EIT), and also the association between lung volume evolution and $PTcCO_2$ during NIV, sleep quality, ventilation quality (compliance, median leaks, and average apnea–hypopnea index over the past 30 days), ventilator settings and clinical phenotype.

As part of routine follow-up, patients underwent annual spirometry and daytime arterial blood gas (ABG) on room air, with the most recent results included in this study. $PTcCO_2$ under NIV was measured at home within the month prior to hospitalization by the patient's homecare provider. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI).

During the NIV session, all patients underwent continuous $PTcCO_2$ monitoring using the Sentec device (Firmware 7.0, ResMed). Dyspnea was evaluated both before and during the NIV session using the BORG scale.

To assess lung volume variation during the NIV session, variation in EELI measured using the Pulmovista 500® (Dräger Medical, Lübeck, Germany) was used as the primary outcome measure.

An example of the signal obtained is shown in Fig. 1. The lowest point of each respiratory cycle corresponds to the impedance level at end-expiration, referred to as the EELI. The tidal volume variations was evaluated by the change in impedance between the trough and peak of each cycle—known as the tidal impedance variation (TIV)—(18). In addition, the device enables measurement of RR (19), Ti, and Te.

Each patient was positioned in a semi-sitting posture. The electrode belt, moistened beforehand to enhance electrical conductivity, was placed between the 4th and 6th intercostal spaces and remained in position throughout the measurement, in accordance with established recommendations (20). The midpoint of the belt's posterior section was aligned with the thoracic spinous processes, whereas electrodes 1 and 16 on the anterior section were positioned equidistant from the sternal line. For each patient, a belt size appropriate for their thoracic circumference was selected.

An additional moistened reference electrode was placed on the abdomen. To ensure optimal signal quality, patients were instructed to remain silent and minimize arm and trunk movements during the recording. Once the patient was stabilized, signal calibration was performed as recommended by the manufacturer to confirm high-quality data acquisition before initiating measurements.

For each patient, continuous 30-min EIT recording was performed, comprising 3 phases: 5 min of spontaneous breathing, 20 min of NIV, and a subsequent 5 min of spontaneous breathing.

Following data acquisition, impedance values for each respiratory cycle were extracted using the dedicated Analysis Tool 6.1 software (Dräger Medical). The extracted data were then processed and analyzed using Excel (Microsoft Corp., Redmond, WA, USA) and JupyterLab (https://jupyter.org). Impedance values are usually reported in arbitrary units (a.u.).

For the primary objective, evolution of lung volume was described by EELI data expressed as percentage changes relative to a reference value, following the method proposed by Mauri et al. (21). This allowed us to compare the evolution of EELI under NIV between patients, as raw impedance values were expressed in arbitrary units and showed high inter-patient variability. The reference value was defined as the average EELI during the first minute of NIV (EELI T1), as illustrated in Fig. 1. Accordingly, EELI values at T2 (NIV + 2 min), T3 (NIV + 5 min), T4 (NIV + 10 min), T5 (NIV + 15 min), and T6 (NIV + 20 min) were averaged over the minute preceding each respective time point and expressed as percentage changes relative to EELI T1. In addition, data preprocessing included cleaning by removing outliers with absolute z-scores exceeding 3. For standardization, impedance values were normalized for each patient by subtracting the mean and dividing by the standard deviation of the first minute under NIV. To assess

changes in the ventilatory pattern during the NIV session, we analyzed variations in TIV, RR, Ti and Te assessed by EIT under NIV between T1 and T6 (First and last minute under NIV).

To assess the association between lung volume evolution and clinical data we compare patients with an increase in lung volume define by a positive ΔEELI at T5 and T6 (+ 15 and + 20min) and those with a decrease in lung volume define by a negative ΔEELI at T5 and T6 (+ 15 and + 20min).

As a pilot study, and due to the absence of prior data in the literature regarding our primary outcome measure in this population, no formal sample size calculation was performed. For feasibility reasons we included a sample size of 30 patients to address our study hypothesis, basis on previous physiological study regarding EIT and non-invasive ventilation therapies (8,9,21).

Categorical variables were presented as frequencies and percentages. Continuous variables were expressed as median and interquartile ranges (IQRs) [IQR, 25%–75%] for non-normally distributed data, and as mean ± standard deviation (SD) otherwise. Paired comparisons were performed using Wilcoxon signed-rank test for non-parametric data and paired t test otherwise. For comparisons between independent groups, Chi-2 or Fisher's exact test for non-parametric variable was used for categorical variables, and the Mann–Whitney U test or t test according to the variable distribution was used for continuous variables. A two-sided p-value of < 0.05 was considered statistically significant. Association between ventilatory pattern parameters changes (variations in TIV, RR, Ti and Te) and ΔEELI at T6 were estimated using linear regression models.

All statistical analyses were conducted using RStudio software (Integrated Development Environment for R, Posit Software, PBC, Boston, MA, USA).

Declarations

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Author contributions

Conceptualization: AG, CD, LG.

Methodology: AG, CD, JGB, HR, LG.

Software: not applicable

Formal analysis: AG, CD, SJ, JGB, HR, LG.

Investigation: AG, CD, PS, MG, AP, AM, LG.

Resources: AG, BR, MZ, JM, PG, LG

Data Curation: AG, CD

Data interpretation: all authors.

Writing - Original Draft: AG, CD, LG

Writing - Review & Editing: all authors

Visualization: AG, CD, PS, MG, AP

Supervision: LG

Project administration: AG, CD, LG

Data Availability Statement : The data that support the findings of this study are available from the corresponding author upon reasonable request.

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None

Conflict of interests statement

AG received support for attending meetings and travel from Dräger Medical

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MG has no competing interests

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Advisory Board for Sanofi and Vertex

PG has no competing interests

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Figures

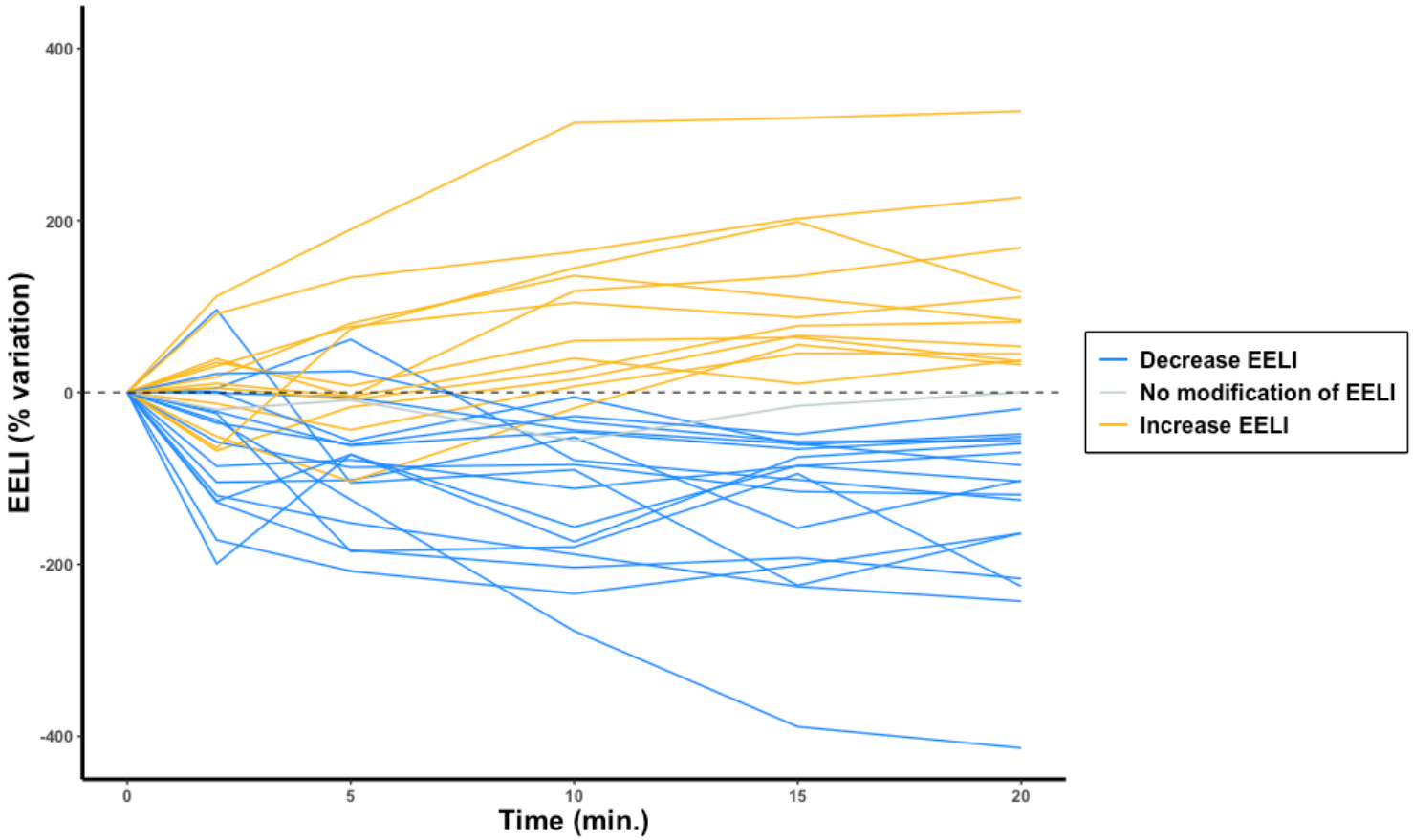


Figure 1

Individual trajectories of EELI during the NIV session.
Data are expressed as percentage change relative to the first minute of NIV (T1).

EELI: end-expiratory lung impedance; NIV: non-invasive ventilation.

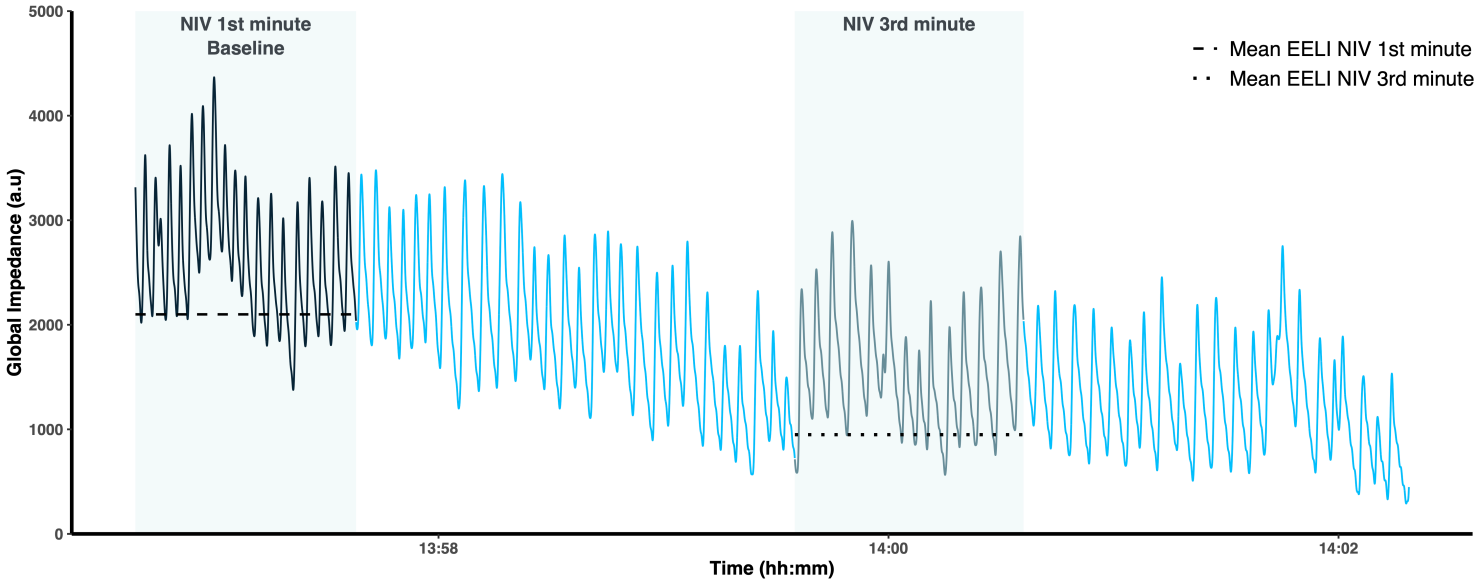


Figure 2

Example of a global impedance signal recorded after a few minutes of spontaneous breathing following the initiation of NIV.

EELI: end-expiratory lung impedance; NIV: non-invasive ventilation.

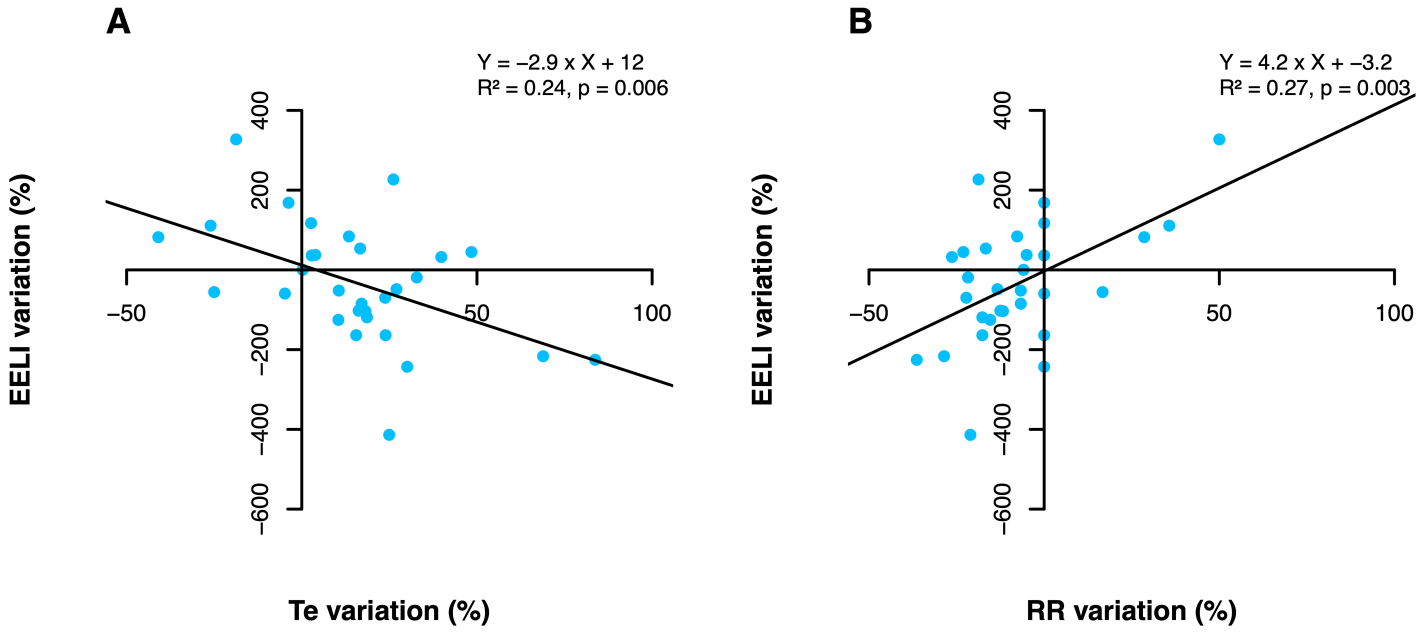


Figure 3

Linear regression between change in EELI and (A) change in Te, and (B) change in RR for all patients.

EELI: end-expiratory lung impedance; Te: expiratory time; RR: respiratory rate.

Supplementary Files

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- FigureS1.png
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