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Oesophageal pressure as a surrogate of pleural pressure in mechanically ventilated patients

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ABSTRACT

Background: Oesophageal pressure (P_{oes}) is used to approximate pleural pressure (P_{pl}) and therefore to estimate transpulmonary pressure (P_L). We aimed to compare oesophageal and regional pleural pressures and to calculate transpulmonary pressures in a prospective physiological study on lung transplant recipients during their stay in the intensive care unit of a tertiary university hospital.

Methods: Lung transplant recipients receiving invasive mechanical ventilation and monitored by oesophageal manometry and dependent and nondependent pleural catheters were investigated during the post-operative period. We performed simultaneous short-time measurements and recordings of oesophageal manometry and pleural pressures. Expiratory and inspiratory P_L were computed by subtracting regional P_{pl} or P_{oes} from airway pressure; inspiratory P_L was also calculated with the elastance ratio method.

Results: 16 patients were included. Among them, 14 were analysed. Oesophageal pressures correlated with dependent and nondependent pleural pressures during expiration (R^2=0.71, p=0.005 and R^2=0.77, p=0.001, respectively) and during inspiration (R^2=0.66 for both, p=0.01 and p=0.014, respectively). P_L values calculated using P_{oes} were close to those obtained from the dependent pleural catheter but higher than those obtained from the nondependent pleural catheter both during expiration and inspiration.

Conclusions: In ventilated lung transplant recipients, oesophageal manometry is well correlated with pleural pressure. The absolute value of P_{oes} is higher than P_{pl} of nondependent lung regions and could therefore underestimate the highest level of lung stress in those at high risk of overinflation.

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During controlled ventilation without respiratory muscle activity, absolute oesophageal pressure is higher than the pleural pressure of the nondependent lung regions and could therefore underestimate the highest level of lung stress in that lung https://bit.ly/3a95CUh

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Introduction

Mechanical ventilation for acute respiratory distress syndrome (ARDS) is still challenging. Recent guidelines have established strong recommendations for using low tidal volume (V_t) (4–8 mL·kg^{-1} predicted bodyweight) and limiting plateau pressure (P_{plat}) [1]. Concerning the level of positive end-expiratory pressure (PEEP) to apply, notably there is no well-established recommendation to use a high level of PEEP for patients with the most severe ARDS. Based on a previous pilot study [2], some experts recommend to set PEEP using oesophageal manometry by targeting the transpulmonary plateau pressure. Oesophageal pressure (P_{oes}) has been used for decades by physiologists as a surrogate of pleural pressure (P_{pl}) measurement and allows the calculation of the true lung distending pressure, the so-called transpulmonary pressure, $P_L=P_{aw}-P_{oes}$, where P_{aw} is the airway pressure [3]. However, there are controversies about using the absolute value of P_{oes} and some authors recommend to consider the tidal variation of oesophageal pressure which allows the calculation of the ratio of the elastance of the chest wall to the respiratory system [4].

Recently, in a ventilated lung-injured pig model and a human cadaver ventilated model, Yoshida *et al.* [3] have conciliated these two theories through comparisons of dependent and nondependent pleural pressures to oesophageal pressure. The main results of the Yoshida *et al.* [3] study are that P_{oes} accurately estimates the dependent pleural pressure both at inspiratory and expiratory pressures, and that elastance-derived inspiratory transpulmonary pressure accurately estimates the nondependent inspiratory transpulmonary pressure.

Therefore, the objective of this study was to compare P_{oes} with dependent and nondependent pleural pressures in lung transplant recipients receiving invasive mechanical ventilation during the post-operative period. Our hypothesis is that transpulmonary pressure calculated with P_{oes} could underestimate the regional P_L of the nondependent lung.

Methods

Study design, setting and participants

This study was conducted in the intensive care unit (ICU) of the North University Hospital (Marseille, France). According to French legislation, all patients gave their written informed consent to participate. The study was registered at ClinicalTrials.gov with identifier number NCT03179644 on 7 June 2017 and approved by the local ethics committee (Comité de Protection des Personnes Sud Méditerranée; 2016-A00567-44).

Patient were included if they fulfilled the following inclusion criteria: age ⩾18 years admitted in the ICU after double-lung transplantation and mechanically ventilated. Exclusion criteria were: age <18 years, pregnancy or breastfeeding, lack of medical insurance, deprivation of liberty by a judicial or administrative decision, those hospitalised without consent, single-lung transplantation and contraindication to placement of a nasogastric tube (oesophageal varices, oesophageal cancer, surgery of the oesophagus <1 year). Patients were not included in case of admission in the ICU with an open chest after surgery and/or high-flow air leaks (>10% of inspired volume) or if they had systemic sclerosis with oesophageal involvement.

Pleural pressure measurements

Before chest closure, the thoracic surgeon introduced multihole pleural catheters (Pleurocath; Plastimed, Le Plessis Bouchard, France) along the thoracic drains under direct view. The nondependent catheters were positioned at the surface of the anterior visceral pleura; the dependent catheters were positioned at the surface of the posterior visceral pleura (supplementary figure S1). According to surgical considerations, two or four pleural catheters were positioned on the right and/or left side, at least one to measure the dependent pleural pressure and one to measure the nondependent pleural pressure per patient. Before measurement, we verified catheter emptiness with 5 mL of air. Chest tubes were then clamped during measurements. Pleural catheters were thereafter connected to a pressure port of a Fluxmed monitor (MBMED, Martínez, Argentina). Good transmission of pleural pressure was assessed by an occlusion test. We performed 3–5-min recordings for each pleural tracing during the first 48 h post-operative period.

Oesophageal pressures measurements

An oesophageal balloon catheter (Nutrivent; Sidam, Mirandola, Italy) was inserted and inflated with a minimal, nonstress volume (2–3 mL) of air as recommended [4]. Adequate positioning of the balloon in the lower part of the oesophagus was confirmed by the presence of cardiac artefacts on the oesophageal curve and a positive occlusion test (expiratory hold on the ventilator) under passive conditions with gentle chest compression [5]. Oesophageal pressure was recorded by the same device used for pleural pressure recordings. The occlusion test was considered positive if the relationship between ΔP_{pl} and ΔP_{aw}, as well as

between ΔP_{oes} and ΔP_{aw}, yielded a slope of 1.0±0.2. In case of a negative test, tracings and measurements were not analysed. Measurements were performed under static conditions (zero flow) during an end-inspiratory occlusion pause of 2 s allowing the measurement of P_{plat} and inspiratory P_{oes} ($P_{oes,insp}$), respectively, and following an end-expiratory occlusion pause of 5 s allowing the measurement of total PEEP ($PEEP_{tot}$) and expiratory P_{oes} ($P_{oes,exp}$), respectively.

Definitions and calculations

The following formulae were used for assessment of transpulmonary pressures.

Inspiratory transpulmonary pressure ($P_{L,insp}$) was determined using oesophageal pressure as $P_{L,insp,oes}=P_{plat}-P_{oes,insp}$ or using direct measurement of P_{pl} in the nondependent lung as $P_{L,ND,insp}=P_{plat}-P_{pl,ND,insp}$ and in the dependent lung as $P_{L,D,insp}=P_{plat}-P_{pl,D,insp}$.

Conversely, expiratory transpulmonary pressure ($P_{L,exp}$) was determined using oesophageal pressure as $P_{L,exp,oes}=PEEP_{tot}-P_{oes,exp}$ or using direct measurement of P_{pl} in the nondependent lung as $P_{L,ND,exp}=PEEP_{tot}-P_{pl,ND,exp}$ and in the dependent lung as $P_{L,D,exp}=PEEP_{tot}-P_{pl,D,exp}$.

Additionally, $P_{L,insp}$ was also calculated from the elastance ratio of the chest wall to the respiratory system [6] as $P_{L,insp,ER}=P_{plat}-(P_{plat}\times EL_{CW}/EL_{RS})$. Accordingly, respiratory system elastance (EL_{RS})=$(P_{plat}-PEEP_{tot})/V_t$ and chest wall elastance (EL_{CW})=$(P_{oes,insp}-P_{oes,exp})/V_t$.

All pressures were expressed in cmH_2O.

Statistical analysis

As this was an exploratory physiological study, no statistical power calculation was anticipated. However, the ethics committee approval allowed the enrolment of a maximum of 45 patients during a 2-year period. All presented results are part of the primary analysis of the data. All statistics were analysed by two-tailed tests. Continuous variables were reported as mean with standard deviation or median with interquartile range as appropriate. Comparisons were performed by the t-test or Mann–Whitney test as appropriate. Categorical variables were expressed as the absolute value and percentage. Comparisons were performed by the Chi-squared test. Normality of the distribution of variables was tested by Kolmogorov–Smirnov and Shapiro–Wilk tests. Correlations were determined with the Pearson correlation test with further Bland–Altman analysis for each correlation. A two-way repeated-measures ANOVA was performed to compare transpulmonary pressures at end-expiration and end-inspiration according to the modality of calculation and to the level of applied PEEP. The normality of the distribution of the residuals, the assumption of sphericity, and the interaction between transpulmonary pressures and PEEP were checked. Intra-group differences were evaluated by *post hoc* Bonferroni pairwise multiple comparisons. A p-value <0.05 was retained as significant. SPSS version 20.0 (IBM, Armonk, NY, USA) was used for all statistical analyses and figures.

Results

Patients and measurements

22 lung transplant recipients gave their informed consent before surgery (figure 1). Six patients were excluded; 16 lung transplant recipients were monitored. Two of these patients were not analysed because of a negative occlusion test (correlations between ΔP_{oes} and ΔP_{aw} and/or ΔP_{pl} and ΔP_{aw} <0.8). The main characteristics of the 14 remaining patients are shown in table 1. Illustrative tracings of volume, flow and pressures during an occlusion test with chest compression are shown in figure 2. 50% of patients were assisted by venovenous extracorporeal membrane oxygenation at ICU admission. All measurements were performed while patients were sedated and mechanically ventilated in volume-assisted controlled mode with a range of PEEP between 8 and 14 cmH_2O without spontaneous breathing effort. Among the 14 patients, four had daily serial measurements totalling 24 measurements. Details of gas exchange, mechanical ventilation settings and respiratory system mechanics are given in table 2.

Correlations between oesophageal and pleural pressures

Occlusions tests yielded 0.95±0.05 for $\Delta P_{oes}/\Delta P_{aw}$ and 0.94±0.06 for $\Delta P_{pl}/\Delta P_{aw}$. Dependent and nondependent expiratory pleural pressures were significantly correlated with expiratory oesophageal pressure (R^2=0.71 and R^2=0.77, respectively, p<0.01) (figure 3a and c). Dependent and nondependent inspiratory pleural pressures were significantly correlated with inspiratory oesophageal pressure (R^2=0.66 for each, p<0.05) (figure 4a and c). Oesophageal pressure was always found to be higher than nondependent pleural pressure. During expiration time, the mean±SD difference between oesophageal pressure and dependent pleural pressure was 0.48±2.87 cmH_2O and between oesophageal pressure and nondependent pleural pressure was 5.25±2.51 cmH_2O (figure 3b and d). During inspiration time, the mean±SD difference between oesophageal pressure and dependent pleural pressure was 0.98±2.90 cmH_2O

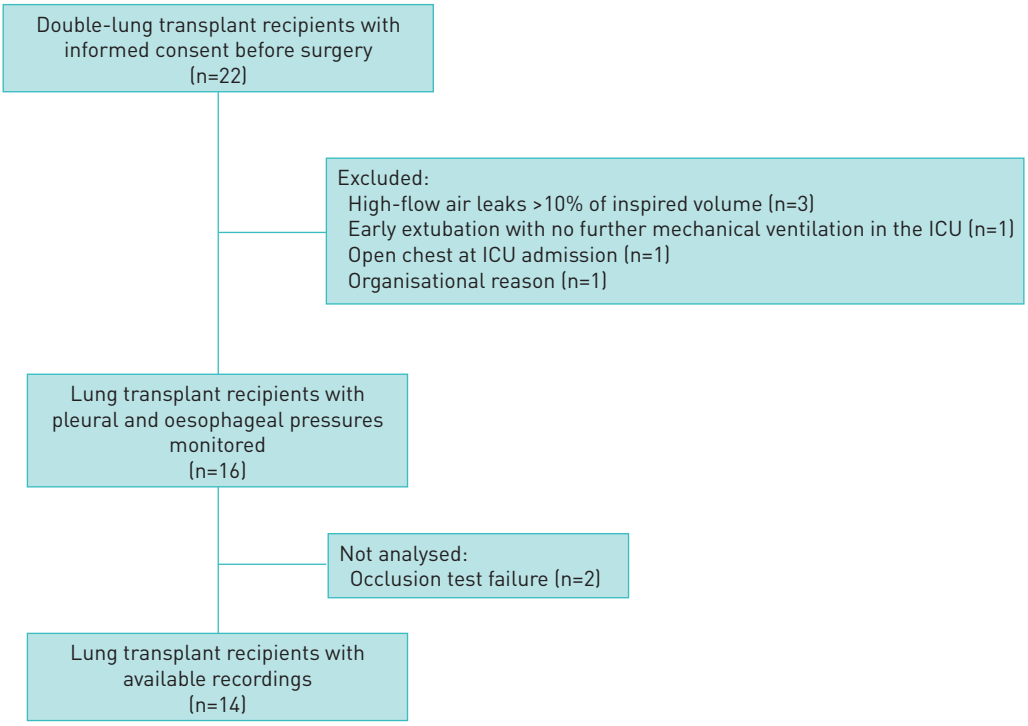

FIGURE 1 Flow diagram of the included patients. ICU: intensive care unit.

and between oesophageal pressure and nondependent pleural pressure was 6.09±2.90 cmH_2O (figure 4b and d). The mean±SD difference between dependent pleural pressure and nondependent pleural pressure was 4.76±2.94 cmH_2O at expiratory time and 5.38±2.11 cmH_2O at inspiratory time.

Correlations between transpulmonary pressures

Correlations and Bland–Altman analysis between inspiratory transpulmonary pressures according to four ways of calculation are presented in figure 5. Inspiratory P_L computed from oesophageal pressure was

TABLE 1 Patient characteristics

Subject	Age years	Sex	SOFA score at inclusion	SAPS II score at inclusion	Indication for DLT	vvECMO#	Duration of mechanical ventilation days	ICU length of stay days	ICU survival
1	61	Female	9	47	COPD	No	4	13	Yes
2	61	Male	7	55	Fibrosis	Yes	41	50	Yes
3	41	Male	7	40	Fibrosis	No	3	9	Yes
4	69	Male	6	39	Fibrosis	No	1	7	Yes
5	69	Male	8	53	Fibrosis	Yes	8	13	Yes
6	65	Male	8	34	Fibrosis	Yes	8	14	Yes
7	65	Male	11	58	Fibrosis	Yes	5	5	No
8	62	Male	5	46	Fibrosis	No	5	13	Yes
9	64	Male	7	39	Fibrosis	No	5	13	Yes
10	61	Female	11	51	COPD	Yes	90	90	Yes
11	53	Male	9	48	Fibrosis	Yes	6	10	Yes
12	62	Female	7	50	COPD	No	43	47	Yes
13	64	Male	8	47	COPD	Yes	14	14	No
14	64	Male	10	52	COPD	No	1	4	Yes
Mean±SD	61±7		8±2	47±7			17±25	22±24	

SOFA: Sequential Organ Failure Assessment; SAPS: Simplified Acute Physiology Score; DLT: double-lung transplantation; vvECMO: venovenous extracorporeal membrane oxygenation; ICU: intensive care unit; COPD: chronic obstructive pulmonary disease. #: at ICU admission.

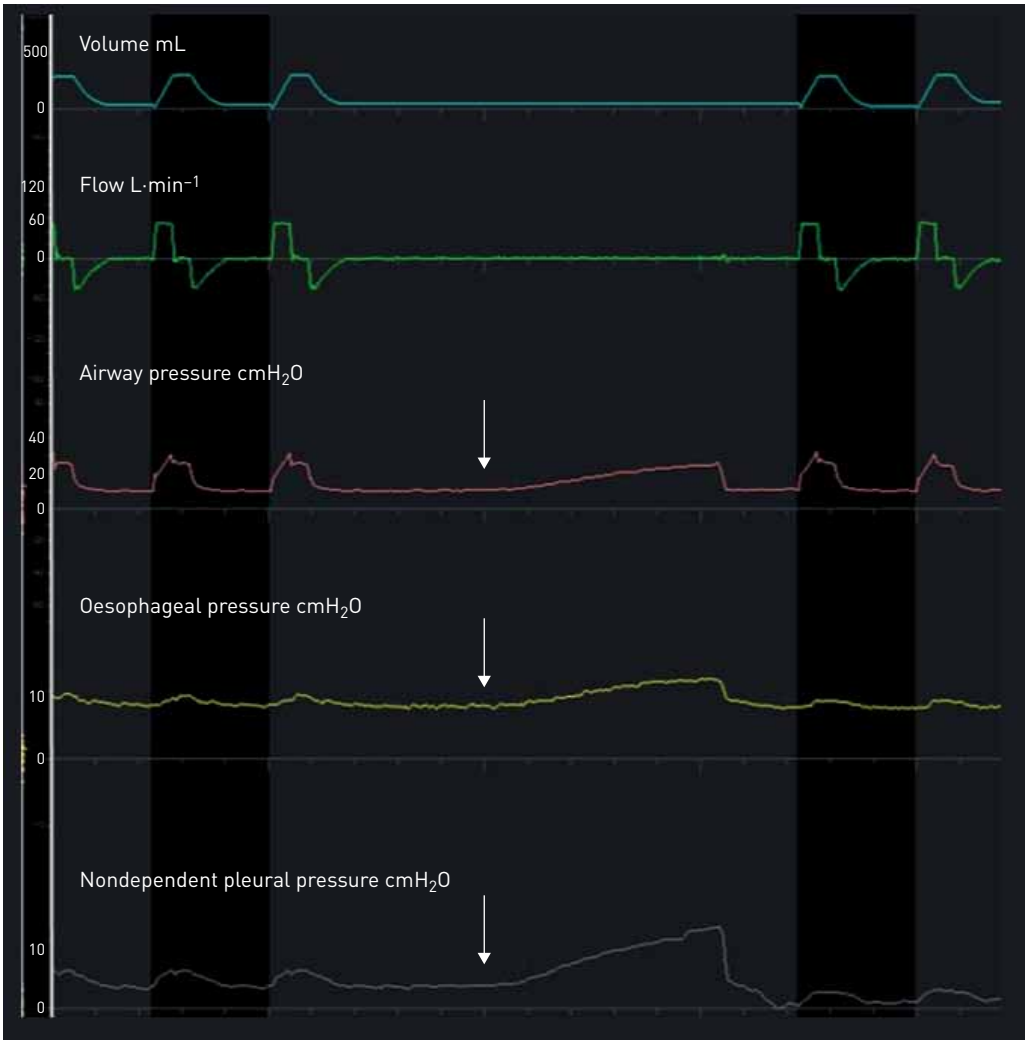

FIGURE 2 Representative tracings of volume, flow, and airway, oesophageal and nondependent pleural pressures during an occlusion test. The increase of airway, oesophageal and nondependent pleural pressures of the same magnitude during gentle thoracic compression (white arrows) ensures the correct placement of the pleural catheter and oesophageal balloon.

better correlated with inspiratory P_L calculated from dependent and nondependent pleural pressures than those calculated from the elastance ratio method (R^2=0.604, R^2=0.629 and R^2=0.45, respectively, p<0.05) [6]. However, the estimated bias was higher between $P_{L,insp,oes}$ and $P_{L,ND,insp}$ than between $P_{L,insp,oes}$ and $P_{L,D,insp}$ (−6.00±3.94 and −1.61±3.62 cmH_2O, respectively). Correlations and Bland–Altman analysis between expiratory transpulmonary pressures according to three ways of calculation are presented in figure 6. Expiratory P_L computed from oesophageal pressure was modestly correlated with expiratory P_L calculated from dependent and nondependent pleural pressures (R^2=0.479 and R^2=0.531, respectively, p<0.02). However, the agreement was better between $P_{L,exp,oes}$ and $P_{L,D,exp}$ than between $P_{L,exp,oes}$ and $P_{L,ND,exp}$ (estimated bias −1.34±3.32 and −5.55±3.36 cmH_2O, respectively).

Relationship between expiratory transpulmonary pressures at different PEEP levels

Expiratory transpulmonary pressures calculated using P_{oes} were close to those obtained from the dependent pleural catheter (figure 7a). Expiratory transpulmonary pressures calculated with the nondependent pleural catheter ($P_{L,ND,exp}$) were higher than those calculated from both the dependent catheter ($P_{L,D,exp}$) and oesophageal pressure ($P_{L,exp,oes}$) whatever the PEEP level. We also found a significant interaction between PEEP and $P_{L,exp}$ (R^2=0.301, p=0.02).

Relationship between inspiratory transpulmonary pressures at different PEEP levels

Inspiratory transpulmonary pressures calculated using P_{oes} were close to those directly measured by the dependent pleural catheter (figure 7b). Inspiratory transpulmonary pressures calculated from the elastance

TABLE 2 Gas exchange, mechanical ventilation settings and respiratory system mechanics

Subject	P_{aO_2}/F_{IO_2}	pH	P_{aCO_2} mmHg	Tidal volume mL	Plateau pressure cmH_2O	PEEP cmH_2O	Driving pressure cmH_2O	Respiratory system elastance $cmH_2O \cdot L^{-1}$	Chest wall elastance $cmH_2O \cdot L^{-1}$	Elastance ratio
1	60	7.35	40	340	28	12	16	47	7	0.15
2	346	7.26	30	270	21	10	11	41	21	0.51
3	184	7.41	41	334	22	10	12	36	17	0.47
4	388	7.39	35	443	22	8	14	32	8	0.25
5	200	7.29	34	250	27	14	13	52	11	0.21
6	157	7.49	33	383	25	10	15	39	3	0.08
7	65	7.30	50	284	31	15	16	56	9	0.16
8	150	7.30	78	358	26	10	16	44	4	0.09
9	160	7.34	49	417	16	5	11	26	6	0.23
10	90	7.36	39	200	24	10	14	70	18	0.26
11	126	7.30	37	222	26	14	12	54	8	0.15
12	140	7.36	41	321	29	14	15	47	5	0.11
13	225	7.42	32	460	23	14	9	20	12	0.60
14	250	7.38	36	400	25	12	13	32	8	0.25
Mean±SD	181±96	7.35±0.06	41±12	334±81	25±4	11±3	13±2	42±13	10±5	0.25±0.16

P_{aO_2}: arterial oxygen tension; F_{IO_2}: inspiratory oxygen fraction; P_{aCO_2}: arterial carbon dioxide tension; PEEP: positive end-expiratory pressure.

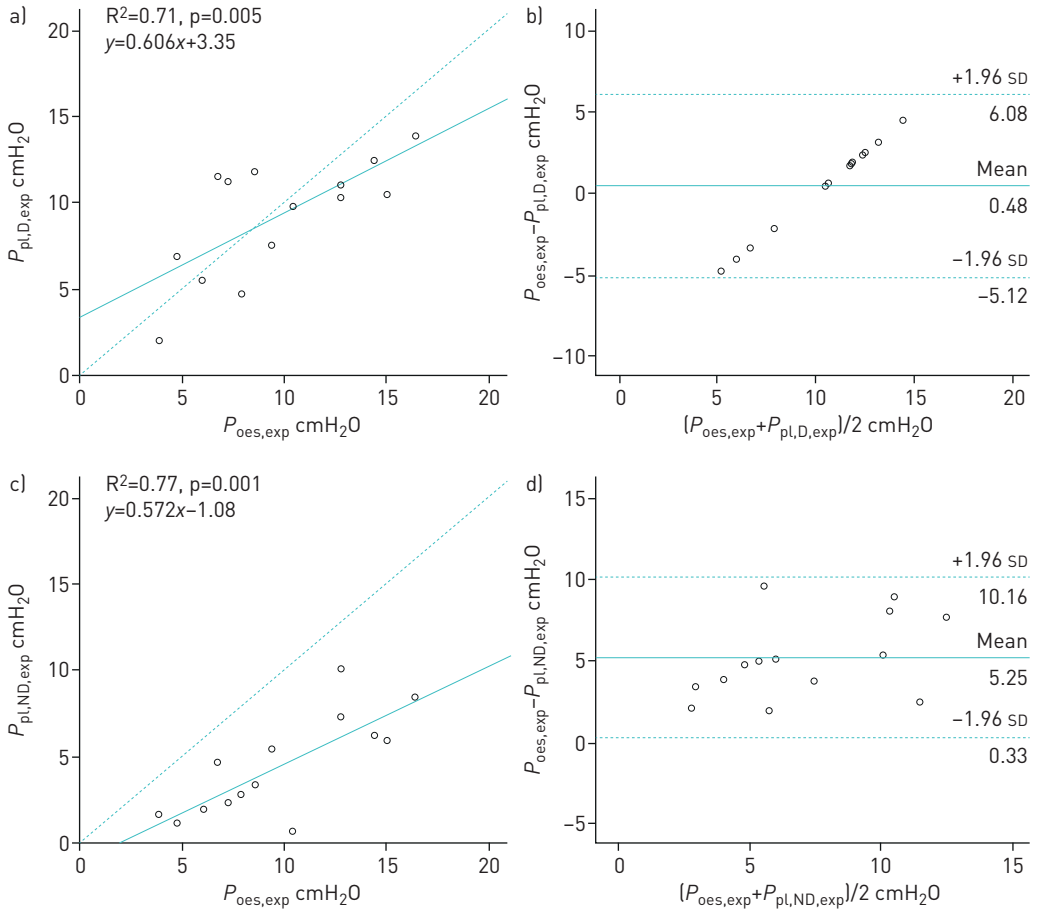

FIGURE 3 a, c) Correlations and b, d) Bland–Altman analysis between a, b) dependent and c, d) nondependent pleural pressures and oesophageal pressure at end-expiration. a, c) Dashed line represents the identity line. b, d) Solid and dashed lines represent mean±1.96 SD of the differences. Each circle represents a different patient.

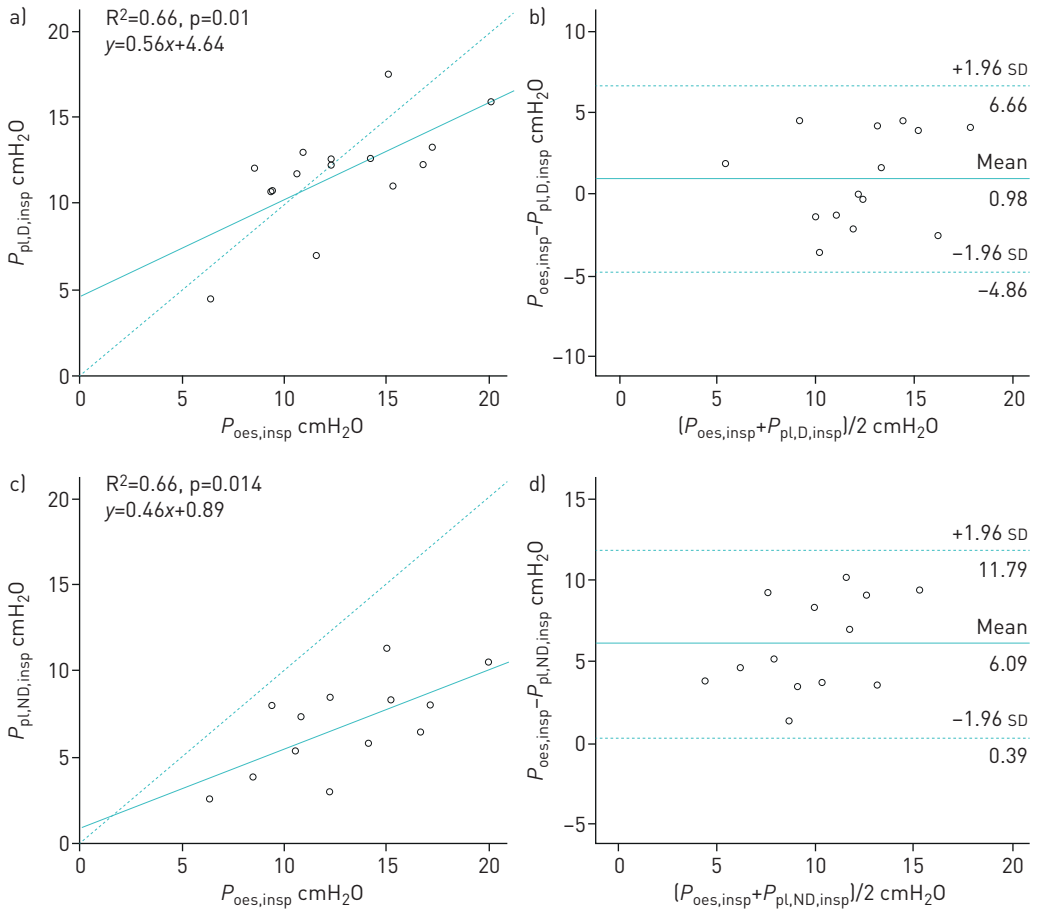

FIGURE 4 a, c) Correlations and b, d) Bland–Altman analysis between a, b) dependent and c, d) nondependent pleural pressures and oesophageal pressure at end-inspiration. a, c) Dashed line represents the identity line. b, d) Solid and dashed lines represent mean±1.96 SD of the differences. Each circle represents a different patient.

ratio of the chest wall to the respiratory system ($P_{L,insp,ER}$) were also close to those measured using the nondependent pleural catheter ($P_{L,ND,insp}$). In our model, $P_{L,insp,oes}$ underestimates the true regional transpulmonary pressure of the nondependent lung region ($P_{L,ND,insp}$). We did not find any interaction between PEEP and $P_{L,insp}$ (R^2=0.132, p=0.203).

Discussion

In this mechanically ventilated *in vivo* human model, P_{oes} is close to the pleural pressures of the dependent lung region. However, we found overestimation by P_{oes} using nondependent lung region pleural pressures. Therefore, the limitation of inspiratory lung stress using P_{oes} may lead to underestimating lung stress in nondependent lung regions. Rather, inspiratory P_L calculated with the elastance ratio ($P_{L,insp,ER}$) may reflect local lung stress in nondependent lung regions, which are usually the overinflated lung regions.

From previous clinical and experimental studies, we know that 1) because of the weight of the heart and the increase of the gravitational gradient of pleural pressure during ARDS, P_{oes} is higher in supine patients ventilated for ARDS than those of nonventilated healthy subjects in the upright position [2, 7, 8], and 2) from an experimental study in dogs [9], and recently in humans [10], it was demonstrated that absolute pleural pressures are ~7 cmH_2O lower than P_{oes} in the nondependent regions and ~5 cmH_2O higher in the dependent regions at low intrathoracic pressure. Therefore, some authors have proposed to apply a correction subtraction of between 2.5 and 5 cmH_2O to the actual measured oesophageal pressure to calculate the transpulmonary pressure [8, 9, 11]. However, the utility of a fixed correction of absolute transpulmonary pressure is still debated [12, 13].

A previous experimental study has demonstrated that in anesthetised pigs and human cadavers 1) P_{oes} was midway between P_{pl} in the dependent region and P_{pl} in the nondependent region, and 2) elastance-derived transpulmonary pressure matched the directly measured transpulmonary pressure from nondependent regions [3].

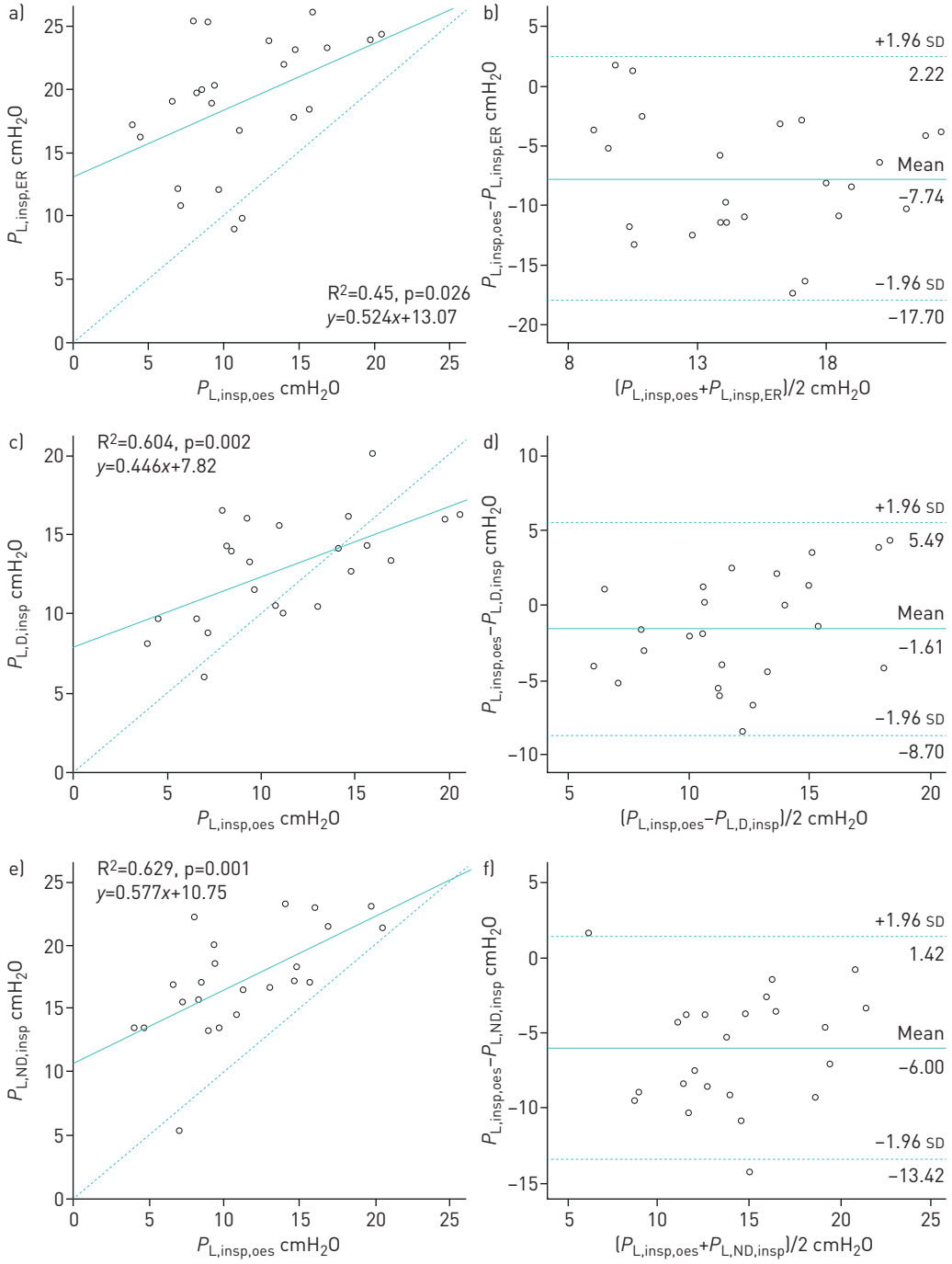

FIGURE 5 a, c, e) Correlations and b, d, f) Bland–Altman analysis between transpulmonary pressures calculated using a, b) elastance ratio, c, d) dependent pleural pressure and e, f) nondependent pleural pressure during end-inspiration. a, c, e) Dashed line represents the identity line. b, d, f) Solid and dashed lines represent mean±1.96 SD of the differences. Each circle represents a different patient.

In addition, TERZI *et al.* [14] showed in a ventilated pig model that in the supine position, the mean difference between P_{oes} and $P_{L,D}$ was 2.2 cmH$_2$O and between P_{oes} and $P_{L,ND}$ was 7.2 cmH$_2$O at 10 cmH$_2$O PEEP. Interestingly, whereas the prone position did not modify the gradient between P_{oes} and $P_{L,D}$, the gradient between P_{oes} and $P_{L,ND}$ decreased to 1.8 cmH$_2$O.

PASTICCI *et al.* [10] have recently investigated pleural pressures in humans, through a chest tube on the surgery side immediately after lung resection of the nondependent lung region in the lateral and supine positions. The main finding of the PASTICCI *et al.* [10] study was that oesophageal pressures were 7.3±2.8 cmH$_2$O higher than nondependent pleural pressures in the supine position; however, because the

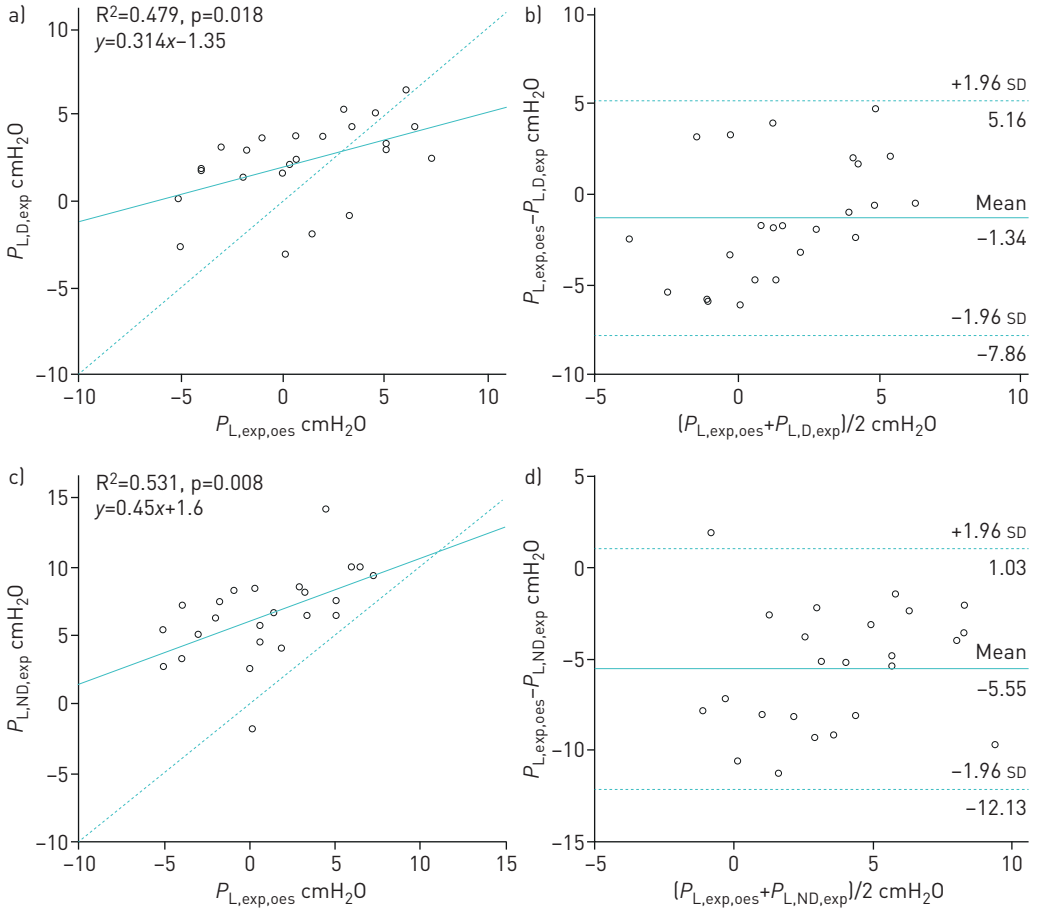

FIGURE 6 a, c) Correlations and b, d) Bland–Altman analysis between transpulmonary pressures calculated using a, b) dependent and c, d) nondependent pleural pressure during end-expiration. a, c, e) Dashed line represents the identity line. b, d, f) Solid and dashed lines represent mean±1.96 SD of the differences. Each circle represents a different patient.

change of pleural pressure induced an identical change in oesophageal pressure, the transpulmonary pressures calculated with the elastance ratio methods were perfectly correlated.

Therefore, the principal strength of our study is to confirm and duplicate in a human *in vivo* setting the results from previous experimental and clinical studies [3, 10, 14] with the unique characteristic to simultaneously investigate dependent and nondependent pleural pressures and oesophageal pressure.

Minimal discrepancies could be explained by some differences between the models. First, the anatomy of the oesophagus differs in pigs and humans, with a more posterior location in pigs. Second, different cardiac and vascular filling pressures may explain the differences in absolute values of oesophageal pressure observed in lung transplant recipients and cadavers. Third, the pleural pressure sensors were different.

Despite some differences between our model and previous experimental models (animal and cadaver), they also share some common results. In the supine position, the median (IQR) dorsal-to-ventral pleural gradient from the dependent to nondependent lung region was 5.0 (2.7–6.4) cmH_2O at inspiration and 4.4 (1.9–5.6) cmH_2O at expiration in our study, which is very close to the values measured in pigs (4.4 (2.4–6.8) cmH_2O) [14], but lower than those measured in cadavers (n=3; mean±SD 10.0±3.1 cmH_2O) [3]. In this latter experiment, despite the "Thiel method" to restore elasticity of the tissues, it is possible that the model affects chest wall recoil force compared with humans.

The elastance-derived method to assess transpulmonary pressure ($P_{L,insp,ER}$) found very close values to those directly measured by $P_{L,ND,insp}$. These findings are concordant with experimental results and therefore suggest that $P_{L,insp,ER}$ could be a valuable target to prevent regional stress and strain of nondependent lung regions [3].

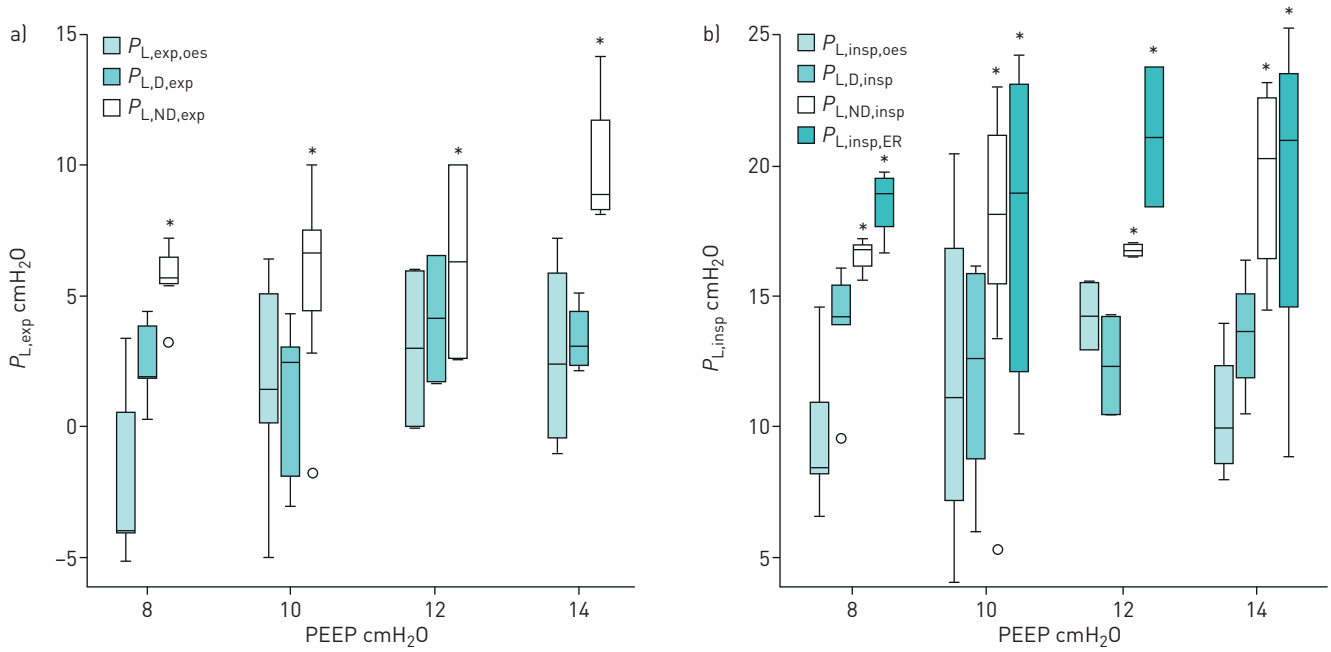

FIGURE 7 Relationship of transpulmonary pressures calculated from oesophageal pressure and pleural pressures in mechanically ventilated human lung transplant recipients during a) expiratory time and b) inspiratory time at different positive end-expiratory pressure (PEEP) levels. *: p<0.05 compared with P_{oes} and the dependent catheter by the *post hoc* Bonferroni test. Boxes represent median and interquartile range; whiskers represent minimum–maximum range. Outliers are represented by circles. See Methods for definitions and calculations.

There are several limitations to the present study. First, we used a very specific *in vivo* model of mechanically ventilated patients with some of them presenting acute lung injury following lung transplantation (primary graft dysfunction). Second, after open chest surgery, the presence of chest tubes, even clamped with no vacuum, may have created some artefacts in the pleural pressure signal. Third, we used common pleural catheters to measure pleural pressure and not specific flat balloon pleural sensors, which have only been used for animal studies so far. However, this was the only device allowed by the French drug safety administration for the study.

Finally, even if oesophageal pressures were well correlated with pleural pressures, we found a significant bias of agreement between oesophageal pressures and nondependent pleural pressures of 5.25±2.51 cmH_2O at expiration time and 6.09±2.90 cmH_2O at inspiration time. Of note, a noninferior bias of agreement of 7.2±5.56 cmH_2O was also reported in a pig model under strict experimental conditions [14].

Although of potential clinical interest, oesophageal manometry is still very underused in clinical practice in ARDS patients (0.8% in the cohort of all ARDS patients in the LUNG SAFE study and 1.2% for severe ARDS patients) [15]. Recently, the largest trial (EPVent-2 study) using oesophageal manometry in ARDS patients failed to demonstrate outcome benefit targeting expiratory transpulmonary pressure compared with a strategy of high PEEP based on a PEEP/inspiratory oxygen fraction table [16].

Oesophageal manometry may be still of clinical interest in specific ARDS clinical vignettes, notably when abdominal or chest wall elastance is increased [17] or in unrecognised harmful strong respiratory efforts [18, 19]. Oesophageal manometry also remains useful to diagnose patient–ventilator asynchrony which may worsen the outcome [20–22].

In conclusion, in ventilated lung transplant recipients, oesophageal manometry was well correlated with direct measures of pleural pressure with nonspecific sensors and the absolute values were close to those from the dependent lung. During controlled ventilation without respiratory muscles activity, the absolute value of P_{oes} is higher than the pleural pressure of nondependent lung regions and could therefore underestimate the highest level of lung stress in nondependent lung regions. In addition, the elastance-derived method seems useful to prevent this pitfall.

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