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Association of right ventricular dysfunction on electrocardiogram with outcomes and ventilatory response in patients monitored by electrical impedance tomography: A cohort study

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ABSTRACT

Background: Mechanical ventilation is essential in critical care but can cause lung injury and hemodynamic compromise, particularly in patients with right ventricular dysfunction (RVD). Electrical impedance tomography (EIT) is increasingly used to guide ventilation, but its role in patients with RVD is not well defined.
Objectives: To evaluate how electrocardiographic (ECG) signs of RVD influence the application and effects of EIT-guided ventilation management.
Methods: This retrospective cohort study (2013–2023) included mechanically ventilated patients who underwent both ECG and EIT. Patients were grouped according to the presence of ECG signs of RVD. Demographic, clinical, and respiratory characteristics were compared. Airway pressures during EIT-guided recruitment maneuvers (RMs) and decremental positive end-expiratory pressure (PEEP) trials were analyzed using linear regression. Repeated ECG and EIT data were assessed using linear mixed-effects models.
Results: Of 285 patients, 38 (13 %) had ECG signs of RVD. They were more often male (89.5 % vs. 74.1 %, $p = 0.04$), older (68.2 vs. 63.5 years, $p = 0.02$), and had higher mortality (65.8 % vs. 48.6 %, $p < 0.05$). During EIT-guided RMs, they received lower maximum PEEP (–2.2 to –0.4 cmH_2O) and a narrower decremental PEEP range

Abbreviations: ABG, Arterial Blood Gas; APACHE IV, Acute Physiologic and Chronic Health Evaluation IV; ARDS, Acute Respiratory Distress Syndrome; Cdyn, Dynamic Compliance; CL, Alveolar Collapse; ECG, Electrocardiogram; EIT, Electrical Impedance Tomography; FiO2, Fraction of inspired Oxygen; ICU, Intensive Care Unit; MP, Mechanical Power; MUMC+, Maastricht University Medical Center+; MV, Mechanical Ventilation; OD, Alveolar Overdistension; PBW, Predicted Body Weight; PEEP, Positive End-Expiratory Pressure; Pinsp, Inspiratory Pressure; PO_2/FiO_2, partial pressure of oxygen-inspired fraction of oxygen ratio; RM, Recruitment Maneuver; RV, Right Ventricle; RVD, Right Ventricular Dysfunction; VILI, Ventilator-Induced Lung Injury; Vt, Tidal Volume; ΔP, Delta Pressure.

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(–2.5 to –0.9 cmH_2O, both $p < 0.01$). After EIT-guided optimization, dynamic compliance was higher in patients with ECG signs of RVD (43.6 vs. 38.4 mL/cmH_2O, $p = 0.04$).
Conclusion: ECG signs of RVD identified a high-risk group that appeared less tolerant of, yet more responsive to, EIT-guided PEEP titration. By integrating respiratory and cardiac monitoring, EIT may facilitate safer ventilation strategies.

Introduction

Mechanical ventilation (MV) is essential in the management of critically ill patients, but it carries both physiological and pathophysiological risks. While MV supports gas exchange, it can also cause ventilator-induced lung injury (VILI),[1] which has evolved from early concepts like barotrauma to more comprehensive mechanisms such as ergotrauma and biotrauma.[1,2]

Various tools and strategies are continuously being developed to improve the management and prevention of VILI. Electrical impedance tomography (EIT) enhances ventilation management by identifying optimal positive end-expiratory pressure (PEEP) levels during recruiting maneuvers (RMs) and decremental trials,[3] aiming to balance overdistension and collapse.[4]

However, these maneuvers may exacerbate the well-recognized hemodynamic effects of positive pressure ventilation.[5] This is particularly concerning in the presence of right ventricular dysfunction (RVD), which occurs in 10–25 % of patients with acute respiratory distress syndrome (ARDS) and is driven by factors such as hypoxia, hypercapnia, and pulmonary vascular alterations.[6] In both ARDS-related pulmonary vasoconstriction and primary cardiogenic dysfunction, the right ventricle (RV) has a limited capacity to tolerate excessive afterload. In the absence of chronic adaptation, as seen in longstanding pulmonary hypertension, the thin-walled RV compensates initially by increasing preload, but soon reaches a point where further increases in preload fail to augment contractility and instead impair left ventricular filling, thereby elevating pulmonary pressures.[7] In this setting, the combined effects of increased pulmonary vascular resistance and reduced venous return secondary to positive pressure ventilation may precipitate progression from RVD to overt RV failure, potentially culminating in hemodynamic collapse.[5,7]

Monitoring and preventing RV failure during MV is therefore essential. While echocardiography is the standard for cardiac assessment,[8] it requires equipment and expertise. Surface electrocardiogram (ECG), although more basic, can detect signs of RV strain and dysfunction.[9] However, the accuracy of ECG in diagnosing RVD, and how this may affect the safety and efficacy of EIT-guided PEEP titration, remains poorly investigated.

This study compared the execution and the effects of EIT-guided PEEP titration strategy between mechanically ventilated patients with and without ECG signs of RVD.

Methods

This report was written according to the “Strengthening the Reporting of Observational Studies in Epidemiology” (STROBE) guideline.[10]

Study design and population

This cohort study analyzed adult patients admitted to the Intensive Care Unit (ICU) of the Maastricht University Medical Center+ (MUMC+). The study covered a 10-year period from 2013 to 2023. From the full ICU cohort of 18,362 patients (as described elsewhere[11]), those who received MV and had at least one EIT assessment with a concurrent ECG were eligible for inclusion. Serial ECGs and EIT assessments performed during the patients' ICU stay were also included. Only patients ventilated in the supine position using pressure- or volume-controlled modes were included. EIT assessments were required to meet predefined quality criteria (intermediate or good quality), based on adherence to the expected pattern of decreasing alveolar overdistension (OD) and increasing alveolar collapse (CL) across the steps of the decremental PEEP trial, as previously described[12] (Fig. 1).

The study design was approved by the MUMC+ Institutional Review Board (METC 2021–2792), and the need for consent was waived.[11]

Data collection

Collected demographic and clinical variables included: age, sex, height, weight, admission diagnosis, Acute Physiology and Chronic Health Evaluation IV (APACHE IV) score, ICU and hospital mortality and extracorporeal membrane oxygenation support. Predicted Body Weight (PBW) and Body Mass Index were calculated using standard formulas (*Supplementary Materials*).

Electrical impedance tomography and respiratory data collection

Chest EIT measurements were performed following a standardized protocol described previously[13] (*Supplementary Materials*). For each PEEP level, EIT-derived variables—including dynamic compliance (Cdyn), OD, and CL—were extracted. In addition, ventilation parameters and arterial blood gas (ABG) data were recorded immediately before and after the EIT procedure. As all patients were managed with pressure-controlled ventilation, ventilator settings collected included PEEP, inspiratory pressure (Pinsp), respiratory rate, fraction of inspired oxygen, along with the resulting tidal volume (Vt). Due to missing data, static respiratory mechanics (e.g., total PEEP during expiratory hold, plateau pressure) were not analyzed. Additional derived variables included Cdyn, delta pressure (ΔP = Pinsp - PEEP), and mechanical power (MP). The latter was calculated using the simplified Becher's equation for pressure-controlled ventilation[14] (*Supplementary Materials*).

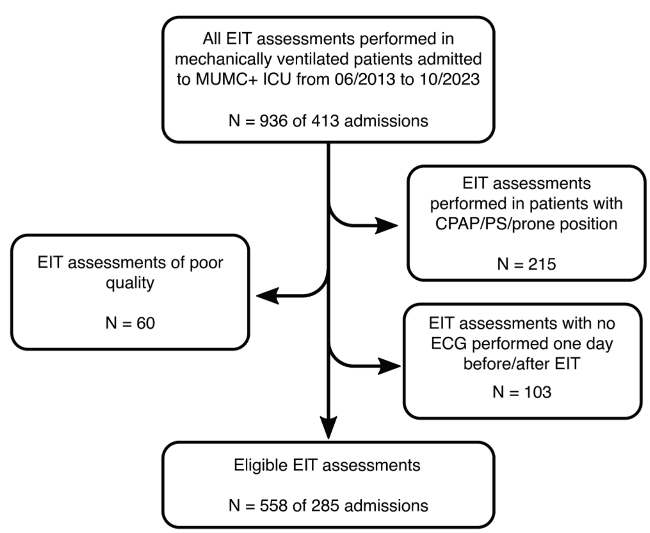

Fig. 1. Flowchart of the study.
(EIT, Electrical Impedance Tomography; MUMC+, Maastricht University Medical Center+; ICU, Intensive Care Unit; CPAP, Continuous Positive Airway Pressure; PS, Pressure Support; ECG, Electrocardiogram).

Electrocardiogram

ECGs were extracted from the hospital electronic medical record system and screened for signs of RVD according to a predefined protocol[15] (Fig. 2). ECG analysis was performed independently by two physicians: an emergency medicine specialist (AR) and an anesthesiologist (FM). In cases of disagreement ($n = 96$; 17 %), a final decision was made by a third reviewer, an expert cardiologist-intensivist (RD). Importantly, ECG evaluation was conducted independently of the EIT assessments, and clinicians performing EIT were blinded to the corresponding ECG interpretations.

Statistical analysis

The cohort was divided into two groups: patients with ECG signs of RVD and those without. Descriptive statistics were used to summarize demographic and clinical variables. Continuous variables were reported as mean ± standard deviation. Categorical variables were presented as frequencies and percentages. Group comparisons were performed using independent samples tests. The Student's *t*-test was used for continuous variables, and the Chi-square test was used for categorical variables. When expected frequencies were low, Fisher's exact test was used.

The primary analysis focused on the association between ECG signs of RVD and EIT execution, specifically examining the maximum PEEP applied during RM, the minimum PEEP at the end of the decremental trial, and the resulting PEEP range. These associations were first analyzed using linear regression models based on the first ECG and EIT assessment per patient. The same variables were subsequently assessed using linear mixed-effects models incorporating all serial ECGs and EITs, with random intercepts to account for within-subject variability.

EIT parameter trends (Cdyn, OD and CL) across the decremental PEEP trial were analyzed using polynomial regression models, as these parameters were expected to exhibit non-linear relationships with PEEP levels. The maximum and minimum values of these parameters, as well as the derived optimal PEEP, were described for both groups using data from the first and serial EIT assessments. Respiratory variables (e.g. ventilation parameters and ABG data) were compared between groups before and after the first EIT using independent samples Student's *t*-test. Within-group changes were assessed using paired samples Student's *t*-test.

For all analyses, normality assumptions for continuous variables and paired differences were assessed using Shapiro-Wilk tests and Q-Q plots; homoscedasticity and regression model assumptions (linearity, normality of residuals) were evaluated through residual diagnostics.

Statistical analyses were conducted using SPSS software (version 28; IBM SPSS Statistics), while mixed-effects and polynomial regression models were performed using R (version 4.1.1; R Foundation for Statistical Computing). The R packages *lme4* and *nlme* were used for mixed-effects modeling. β coefficients were reported with their 95 % confidence intervals, and a *p*-value < 0.05 was considered statistically significant. When a physiologically directional hypothesis was applied (e.g., expecting improvements in Cdyn or reductions in MP post-EIT), one-sided *p*-values were used for interpretation.

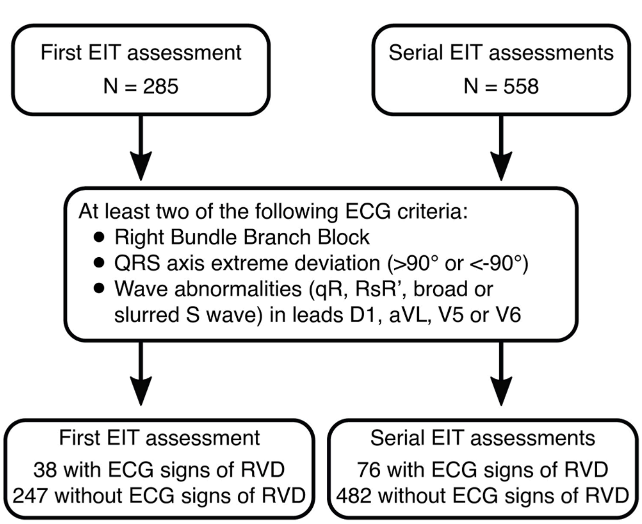

Fig. 2. ECG analysis.
(EIT, Electrical Impedance Tomography; ECG, Electrocardiogram; RVD, Right Ventricular Dysfunction).

Results

Patient characteristics and ECG classification

A total of 285 patients were included, yielding 558 paired EIT and ECG assessments. ECG signs of RVD were identified in 13.3 % of assessments, with consistent findings across serial measurements.

Patients exhibiting ECG signs of RVD were significantly older (68.2 vs. 63.5 years, $p = 0.02$), predominantly male (89.5 % vs. 74.1 %, $p = 0.04$), and demonstrated a higher in-hospital mortality rate (65.8 % vs. 48.6 %, $p < 0.05$) compared to those without ECG signs of RVD. Although the ECG signs of RVD group showed a higher frequency of cardiac-related admission diagnoses, this difference was not statistically significant (28.9 % vs. 17.1 %, $p = 0.08$) (Table 1).

EIT execution

During the RM, patients with ECG signs of RVD were subjected to a lower pressure strategy. Specifically, the maximum PEEP applied was significantly lower in this group (–2.2 to –0.4 cmH_2O, $p < 0.01$). In contrast, the minimum PEEP level at which the decremental trial was terminated did not differ significantly between groups. Consequently,

Table 1
Description of the entire cohort stratified by the presence or absence of ECG signs of Right Ventricular Dysfunction.

	ECG RVD (*N* = 38)	No ECG RVD (*N* = 247)	*p*-value
Demographic			
Age, mean (SD) - yr	68.2 (10.3)	63.5 (11.8)	0.02
Male Sex, N (%)	34 (89.5)	183 (74.1)	0.04
Height, mean (SD) - cm	173.8 (10)	174.3 (10.2)	0.69
Weight, mean (SD) - Kg	87.1 (14.3)	84.9 (16.7)	0.44
BMI, mean (SD) - Kg/mˆ2	29.1 (6.5)	27.8 (4.9)	0.16
Clinical			
SARS-CoV2 Pneumonia, N (%)	20 (52.6)	137 (55.7)	0.72
Pneumonia, Aspiration, and Bacterial Pneumonia, N (%)	3 (7.9)	29 (11.8)	0.59
Pneumocystis Jirovecii Pneumonia and Neutropenia, N (%)	2.5 (5.3)	7 (2.8)	0.34
Other Respiratory Causes, N (%)	1 (2.6)	12 (4.9)	1
Cardiac Surgery and other Cardiac Causes, N (%)	11 (28.9)	42 (17.1)	0.08
Miscellaneous Extra-Pulmonary Cause	1 (2.6)	18 (7.3)	0.47
APACHE IV score, mean (SD)	72.9 (27.9)	77.1 (28)	0.42
ICU Death, N (%)	16 (42.1)	87 (35.2)	0.41
Total in-hospital Death, N (%)	25 (65.8)	120 (48.6)	<0.05
ECMO	2 (5.3)	15 (6.1)	1

Data are presented as mean (SD) or N and percentages. *p* values were derived from the Student's *t*-test, Chi-square test, and Fisher's exact test. (ECG, Electrocardiogram; ECG RVD, ECG signs of Right Ventricular Dysfunction; No ECG RVD, No ECG signs of Right Ventricular Dysfunction; BMI, Body Mass Index; APACHE IV, Acute Physiology and Chronic Health Evaluation IV; ICU, Intensive Care Unit; ECMO, Extracorporeal Membrane Oxygenation).

patients with ECG signs of RVD were assessed over a significantly narrower PEEP range throughout the procedure (–2.5 to –0.9 cmH_2O; $p < 0.01$). This finding was consistently observed in both the initial and serial EIT assessments (Table 2).

EIT parameters and optimal PEEP

Polynomial modeling demonstrated a higher Cdyn during EIT assessment in patients with ECG signs of RVD (46.1 vs. 40.1 mL/cmH_2O). Additionally, this group exhibited a higher optimal PEEP level compared to patients without ECG signs of RVD (10.9 vs. 9.3 cmH_2O) (Table 3, Fig. 3). These findings indicate a more pronounced effect of RM and PEEP titration in patients with ECG signs of RVD, indicating that their respiratory mechanics were more influenced by alveolar collapse than by overdistension.

EIT effect: respiratory mechanics

Prior to the EIT assessment, no significant differences in ventilation parameters were observed between groups. Following EIT, both groups demonstrated a comparable and statistically significant increase in PEEP levels (Table 4, and Table 5 Supplementary Materials).

In patients with ECG signs of RVD, there was a trend toward increased Cdyn, whereas patients without ECG signs of RVD showed a significant reduction in ΔP along with a lower Vt/PBW delivered. These changes were indicative of a more protective ventilation strategy implemented across the entire cohort, characterized by a lower ΔP, reduced Vt/PBW, and, consequently, a decrease in MP, reflecting reduced respiratory workload. Overall, the cohort exhibited a general trend toward improved Cdyn (post-EIT vs. pre-EIT: 39 vs. 38.2 mL/cmH_2O, one-sided $p = 0.07$) (Table 5 Supplementary Materials). Notably, as supported by polynomial modeling, the effects of RM and PEEP titration were more pronounced in patients with ECG signs of RVD. Following EIT, these patients exhibited significantly higher Cdyn values compared to those without ECG signs of RVD (43.6 vs. 38.4 mL/cmH_2O, $p = 0.04$) (Table 4).

EIT effect: gas exchanges

Following EIT, oxygenation significantly improved across the entire cohort, as evidenced by an increase in partial pressure of oxygen-inspired pressure of oxygen ratio (PO_2/FiO_2) (post-EIT vs. pre-EIT: 202 vs. 187 mmHg, $p < 0.01$). In contrast, the other ABG parameters showed no statistically significant changes (Table 5 Supplementary Materials).

There were no statistically significant differences in ABG values between patients with or without ECG signs of RVD, either before or after the EIT assessment (Table 4).

Table 2
Association of ECG signs of Right Ventricular Dysfunction with PEEP values applied across first and serial EIT assessments.

	ECG RVD vs. No ECG RVD	PEEP max	PEEP min	PEEP range
First EIT	β	−0.7	0.6	−1.3
	95 % Confidence Interval	−2 – 0.5	−0.4 – 1.7	−2.5 – −0.2
	p-value	0.25	0.24	0.03
Serial EITs	β	−1.3	0.5	−1.7
	95 % Confidence Interval	−2.2 – −0.4	−0.3 – 1.3	−2.5 – −0.9
	p-value	<0.01	0.26	<0.01

(ECG, Electrocardiogram; PEEP, Positive End-Expiratory Pressure; EIT, Electrical Impedance Tomography; ECG RVD, ECG signs of Right Ventricular Dysfunction; No ECG RVD, No ECG signs of Right Ventricular Dysfunction).

Table 3
EIT-derived respiratory mechanics across first and serial EIT assessments.

	Respiratory mechanics	ECG RVD	No ECG RVD
First EIT	Cdyn, min – max - ml/cmH_2O	34.1 – 67.5	30.2 – 47
	OD, min – max - %	1.4 – 35.2	3.1 – 36.6
	CL, min – max - %	0.3 – 15.2	0.4 – 8.3
	Optimal PEEP - cmH_2O	12	10.3
Serial EITs	Cdyn, min – max - ml/cmH_2O	32 – 46.1	29 – 40.1
	OD, min – max - %	2.3 – 35.9	3 – 36.4
	CL, min – max - %	0.2 – 7.1	0.4 – 6.5
	Optimal PEEP - cmH_2O	10.9	9.3

(EIT, Electrical Impedance Tomography; ECG, Electrocardiogram; ECG RVD, ECG signs of Right Ventricular Dysfunction; No ECG RVD, No ECG signs of Right Ventricular Dysfunction; Cdyn, Dynamic Compliance; OD, Overdistension; CL, Recruitable Alveolar Collapse; PEEP, Positive End-Expiratory Pressure).

Discussion

In this retrospective cohort study, patients with ECG signs of RVD exhibited a distinct response to EIT-guided PEEP titration. During the RM, clinicians applied lower maximum PEEP—and consequently lower inspiratory airway pressures— to these patients, resulting in a narrower range of PEEP values assessed throughout the procedure.

Notably, the decremental PEEP trial tended to be terminated earlier in patients with ECG signs of RVD, associated with the identification of higher optimal PEEP levels. This finding aligns with the greater impact of EIT assessment on respiratory mechanics observed in this group, as evidenced by the increase in Cdyn following EIT. Importantly, this improvement occurred exclusively in patients with ECG signs of RVD, despite similar increases in PEEP across both groups. Overall, EIT-guided PEEP titration was effective in improving oxygenation across the entire cohort.

EIT-Applied pressures and heart-lung interactions

The differences in EIT execution between the two groups, although not directly confirmed by hemodynamic monitoring, are consistent with current physiological understanding of heart–lung interactions. The lower inspiratory pressure threshold observed during RMs in patients with ECG signs of RVD may reflect hemodynamic compromise secondary to reduced RV contractile reserve in the setting of increased afterload. It is well established that positive intrathoracic pressure during MV affects RV function.[5] Elevated right atrial pressure decreases the pressure gradient for venous return, reducing RV preload and, by the Frank-Starling mechanism, cardiac output. Simultaneously, RV afterload rises with increasing pulmonary vascular resistance caused by higher transpulmonary pressures during lung inflation.

These two mechanisms influence RV performance differently, depending on baseline loading conditions, volume status, and RV contractility. Under physiological conditions, PEEP titration exerts a biphasic effect: at lower levels, it primarily reduces preload, whereas at higher levels, it may impose excessive afterload, leading to RV strain and dysfunction.[7,16] Moreover, PEEP titration can unmask latent RV impairment, provoking progressive dilation and failure, even at relatively low PEEP levels.[17] As previously mentioned, the volume status also plays a critical role, influencing not only the upstream pressure driving venous return[18] but also the recruitment and distention of the pulmonary microvasculature.[19,20]

Despite the potential adverse effects of positive airway pressure, recent evidence indicates that individualized PEEP titration based on respiratory mechanics—assessed by EIT or other methods—can yield beneficial hemodynamic effects. Specifically, optimal PEEP mitigates pulmonary vascular compromise by preventing lung overdistention at high PEEP levels, which would otherwise compress intra-alveolar vessels. Conversely, inadequate PEEP may promote atelectasis-related

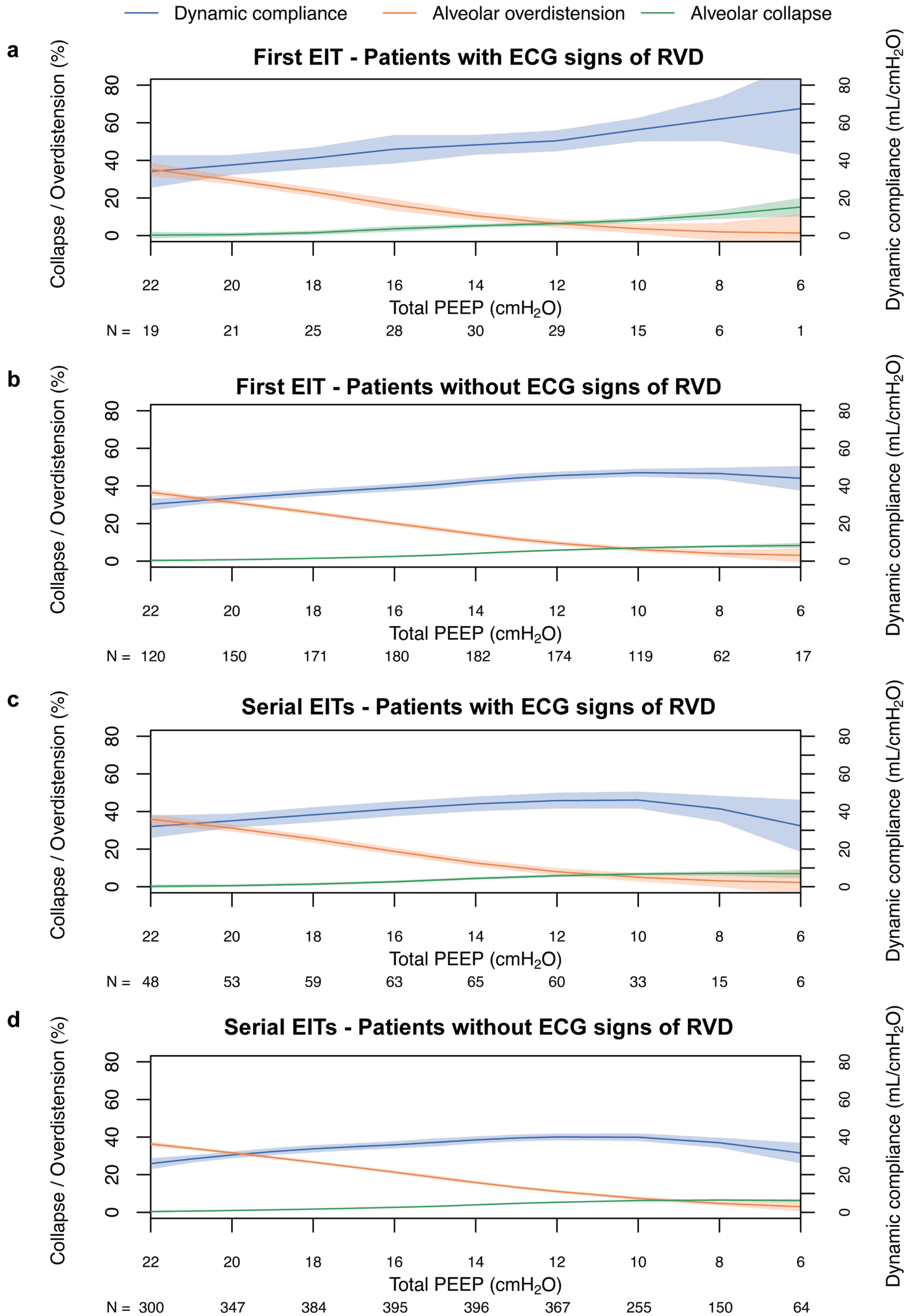

Fig. 3. EIT-derived respiratory mechanics across first and serial EIT assessments. Straight lines represent the mean values of EIT-derived parameters, and shaded areas indicate the 95 % confidence intervals. *N* denotes the number of EIT assessments performed at each PEEP step. (EIT, Electrical Impedance Tomography; ECG, Electrocardiogram; RVD, Right Ventricular Dysfunction; PEEP, Positive End-Expiratory Pressure).

Table 4
Comparison of respiratory characteristics between patients with and without ECG signs of Right Ventricular Dysfunction, before and after EIT assessment.

	Pre-EIT assessment			Post-EIT assessment		
	ECG RVD (*N* = 38)	No ECG RVD (*N* = 247)	*p*-value	ECG RVD (*N* = 38)	No ECG RVD (*N* = 247)	*p*-value
Ventilation parameters						
PEEP, mean (SD) - cmH_2O	11.9 (3.5)	12.3 (2.9)	0.49	13 (3.1)	13.1 (3.3)	0.87
Pinsp, mean (SD) - cmH_2O	24.7 (4.9)	25.6 (4.2)	0.24	25 (3.7)	25.7 (4.1)	0.35
ΔP, mean (SD) - cmH_2O	12.8 (3.8)	13.3 (3.4)	0.40	11.9 (3)	12.6 (3.4)	0.27
Vt/PBW, mean (SD) – $ml*Kg^{-1}$	6.9 (1.5)	6.9 (1.6)	0.90	6.9 (1.5)	6.6 (1.5)	0.32
Cdyn, mean (SD) – ml/cmH_2O	40.6 (14.6)	37.7 (13.6)	0.24	43.6 (17.5)	38.4 (13.1)	0.04
MP, mean (SD) - $J*min^{-1}$	24.3 (10)	25.7 (9.8)	0.43	24.5 (7.7)	25.1 (8.8)	0.71
RR, mean (SD) - breaths per minute	21.2 (6.9)	21.3 (5.2)	0.43	21.9 (8)	22.3 (5.2)	0.73
FiO_2, mean (SD)	0.57 (0.21)	0.58 (0.19)	0.60	0.53 (0.21)	0.53 (0.21)	0.27
ABG analysis						
pH, mean (SD)	7.36 (0.07)	7.37 (0.09)	0.39	7.36 (0.09)	7.37 (0.09)	0.86
PCO_2, mean (SD) – mmHg	43 (7)	44 (11)	0.33	44 (10)	45 (11)	0.82
PO_2, mean (SD) – mmHg	100 (30)	94 (34)	0.34	97 (37)	95 (34)	0.74
HCO_3^-, mean (SD) – mmol/l	24 (5)	26 (5)	0.25	25 (5)	25 (5)	0.70
PO_2/FiO_2, mean (SD) - mmHg	207 (102)	182 (101)	0.19	217 (105)	195 (116)	0.29

Data are presented as mean (SD). (ECG, Electrocardiogram; EIT, Electrical Impedance Tomography; ECG RVD, ECG signs of Right Ventricular Dysfunction; No ECG RVD, No ECG signs of Right Ventricular Dysfunction; PEEP, Positive End-Expiratory Pressure; Pinsp, Inspiratory Airways Pressure; ΔP, Delta Pressure; Vt, Tidal Volume; PBW, Predicted Body Weight; Cdyn, Dynamic Compliance; MP, Mechanical Power; RR, Respiratory Rate; FiO_2, Fraction of Inspired Oxygen; ABG, Arterial Blood Gas; PCO_2, Partial Pressure CO_2, PO_2, Partial Pressure O_2, HCO_3^-, Bicarbonate; PO_2/FiO_2, Partial Pressure O_2-Fraction of Inspired O_2 Ratio).

complications such as vascular leakage, capillary folding, and hypoxic pulmonary vasoconstriction, all of which contribute to RV failure.[21]

Respiratory mechanics and gas exchanges

The differing effect of EIT assessment on respiratory mechanics—despite a comparable increase in PEEP in both groups—can be explained by the important distinction between two often-confused concepts: PEEP and recruitability. PEEP represents the pressure required to maintain alveolar inflation and is considered an intensive property of the lungs, meaning it is independent of the number of potentially recruitable alveolar units. In contrast, recruitability is an extensive property that reflects the amount of lung tissue that can be reopened, depending on factors such as disease severity, lung size, and the degree of pulmonary edema.[22] Consistent with previous literature,[23] the effects of PEEP in our study were more pronounced in patients with higher recruitability—specifically those whose respiratory mechanics improved with recruitment and PEEP titration. Since recruitability correlates with lung weight and edema, the response to PEEP also parallels the severity of underlying disease. Although this relationship has been extensively characterized in ARDS,[24] our findings suggest that it may also apply to pulmonary dysfunction of cardiac origin. In our cohort, patients with ECG signs of RVD—including both ARDS and cardiogenic cases—demonstrated greater recruitability and a worse clinical prognosis, further supporting the link between disease severity and responsiveness to recruitment strategies.

The overall improvement in oxygenation observed following EIT may be explained by the re-expansion and stabilization of previously atelectatic or poorly aerated alveolar units, resulting in a reduction of intrapulmonary shunting and ventilation–perfusion mismatch. However, as noted in prior studies,[25] oxygenation alone is not a reliable surrogate for lung recruitment, as it can be influenced by variations in cardiac output. It is therefore plausible that the observed improvement in oxygenation was, at least in part, mediated by enhanced cardiac output following RV afterload optimization, which would increase mixed venous oxygen content and systemic oxygen delivery.

Pills for the intensivist and future directions

The findings of our study reinforce the value of EIT-based monitoring in critically ill patients. As previously demonstrated,[3] patients with extended lung damage, like ARDS, benefit the most from recruitment and alveolar stabilization strategies, as their respiratory mechanics are characterized more by alveolar collapse than overdistension. At the same time, more severe lung disease is associated with secondary RVD.

Therefore, even if these patients are more susceptible to the hemodynamic impact of RM, they are also the ones who respond most favorably to afterload modulation, which is improved not only through adequate alveolar inflation but also by enhanced gas exchanges. Because RV function is affected both by preload and afterload conditions, in cases of suspected RVD (e.g. ECG signs of RVD), developing EIT-echocardiogram integrated protocols would be valuable, enabling clinicians to detect early signs RV dilation indicative of impending systolic dysfunction.

Strengths and limitations

This study has several strengths. The large sample size provided robust statistical power, enhancing the reliability and generalizability of the findings, which align with existing literature. The inclusion of a comprehensive pulmonary evaluation allowed for a detailed analysis of respiratory mechanics, highlighting the clinical utility of EIT in critically ill patients. This was particularly evident in the group with ECG signs of RVD, where EIT-guided management improved respiratory system mechanics. Additionally, the accuracy of ECG interpretation was ensured by a predefined protocol, applied independently by two senior physicians, with discrepancies adjudicated by a consultant cardiologist.

However, this study has important limitations. Its observational design limits causal inference, and the absence of direct hemodynamic monitoring restricts the interpretation of physiological responses. As previously mentioned, several variables — such as volume status and vasoactive drug use — could have influenced RV performance and, consequently, tolerance to EIT-guided ventilation. Moreover, since arterial pressure is a proxy rather than a direct measure of cardiac output, more advanced hemodynamic monitoring would be valuable in future studies. Right heart catheterization directly measures the ventricular output, allowing an accurate assessment of the effects of positive airway pressure on RV function. Nevertheless, only echocardiography can distinguish whether a hemodynamic alteration results from reduced preload, with hypovolemic right chambers, or from RV failure due to increased afterload, characterized by progressive dilation and contractile dysfunction.

Conclusion

In this large cohort of mechanically ventilated patients undergoing

EIT-guided PEEP optimization, we found that ECG signs of RVD were associated with distinct respiratory mechanics and a differential response to PEEP titration. These findings suggest that the pathophysiological condition of RVD, despite heterogeneity in the underlying etiologies, alters cardiopulmonary interactions and respiratory patterns, with both expected benefits and caveats from EIT assessments.

Our results underscore the importance of considering cardiovascular dynamics, particularly right heart function, when performing and interpreting EIT assessments for PEEP optimization. Further research is warranted to better integrate the hemodynamic impact of airway pressures into EIT-based strategies for personalized mechanical ventilation.

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The authors declare that they have not received any financial support for the conduct of this study. Furthermore, the authors have no financial relationships or conflicts of interest to disclose that could have influenced the outcomes or interpretation of the research presented in this manuscript.

Data availability

The clinical datasets supporting the findings of this study are stored in a secure institutional computer file. Due to patient privacy regulations, the data are not publicly available but can be shared by the corresponding author upon reasonable request and with appropriate data use agreements if required.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used ChatGPT/OpenAI to improve language and readability. After using this ChatGPT/OpenAI, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the publication.

CRediT authorship contribution statement

Adriano Rossi: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Frederik J. Mooi:** Writing – review & editing, Data curation. **Eda Aydeniz:** Writing – review & editing, Formal analysis. **Teun Timmermans:** Writing – review & editing. **Serge J.H. Heines:** Writing – review & editing, Investigation. **Frank van Rosmalen:** Writing – review & editing, Data curation. **Jip de Kok:** Writing – review & editing, Visualization, Data curation. **Iwan C.C. van der Horst:** Writing – review & editing. **Jan-Willem E.M. Sels:** Writing – review & editing. **Dennis C.J.J. Bergmans:** Writing – review & editing. **Marco Giani:** Writing – review & editing. **Giuseppe Citerio:** Writing – review & editing. **Bas C.T. van Bussel:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Rob G.H. Driessen:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.hrtlng.2025.102706.

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