Driving Pressure is Not Predictive of ARDS Outcome in Chest Trauma Patients Under Mechanical Ventilation

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Research

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Abstract

Background: The relationship between the driving pressure of the respiratory system (ΔPrs) under mechanical ventilation and worse outcome has never been studied specifically in chest trauma patients. The objective of the present study was to assess in cases of chest trauma the relationship between ΔPrs and severity of acute respiratory distress syndrome (ARDS) or death and length of stay.

Methods: A retrospective analysis of severe trauma patients (ISS >15) with chest injuries admitted to the Trauma Center from January 2010 to December 2018 was performed. Patients who received mechanical ventilation were included in our analysis. Mechanical ventilation parameters and ΔPrs were recorded during the stay in the intensive care unit. Association of ΔPrs and ARDS with mortality and outcomes was specifically studied at the onset of ARDS (ΔPrs_{-ARDS}) by receiver operator characteristic curve analysis, Kaplan-Meier curves and multivariate analysis.

Results: Among the 266 chest trauma patients studied, 194 (73%) developed ARDS. ΔPrs was significantly higher in the ARDS group versus in the no ARDS group (11.6±2.4 cm H_2O vs 10.9±1.9 cm H_2O, p=0.04). Among the patients with ARDS, no difference according to the duration of mechanical ventilation was found between the high ΔPrs group (ΔPrs_{-ARDS} >14 cm H_2O) and the low ΔPrs group (ΔPrs_{-ARDS} ≤14 cm H_2O), (p=0.75). ΔPrs_{-ARDS} was not independently associated with the duration of mechanical ventilation (hazard ratio [HR], 1.006; 95% CI, 0.95–1.07; p=0.8) or mortality (HR, 1.07; 95% CI, 0.9–1.28; p=0.45).

Conclusion: A high ΔPrs_{-ARDS} was not significantly associated with an increase in mechanical ventilation duration or mortality risk in ARDS patients with chest trauma in contrast with medical patients.

Background

Traumatic chest injuries are responsible for significant morbidity and are the cause of trauma-related death in 20–25% of cases [1]. Following a trauma, patients affected by thoracic injuries are at risk of significant worsening of respiratory function, leading to mechanical ventilation need and acute respiratory distress syndrome (ARDS) in the most severe cases [2]. Causes of ARDS include direct injuries to the lungs and/or secondary mechanisms induced by the trauma setting (e.g. fat embolism, systemic inflammatory response and abdominal compartment syndrome). These physiopathological phenomena and the influence of harmful factors explain why many cases of ARDS occur more than 48 hours after admission [3]. In addition to these respiratory impairments related to trauma, lung injuries may also be aggravated by deleterious effects of mechanical ventilation, such as barotrauma or biotrauma [4, 5]. To prevent or minimize these expected complications, called ventilator-induced lung injury (VILI), current guidelines for lung-protective ventilation in patients with ARDS suggest the use of a low tidal volume (V_T), higher levels of total positive end-expiratory pressure (PEEP) and limitation of the inspiratory plateau pressure of the respiratory system (P_{PLAT}) under 30 cm H_2O [6, 7]. Based on the same rational, most experts strongly recommend this therapeutic approach in trauma patients [8]. However, this extrapolation

of the medical setting is based on populations affected by ARDS from multiple origins. Post-traumatic ARDS represented only 8–13% of these cohorts [3]. Specific studies focusing on populations of trauma patients are lacking.

One of the major determinants of VILI is the driving pressure of the respiratory system (ΔPrs), corresponding to the difference between P_{PLAT} and PEEP [9]. Many studies indeed demonstrated that ΔPrs in case of ARDS was associated with worst outcome [9, 10]. ΔPrs is a marker of alveolar collapse as well as stress and aggressive ventilation [11] and can be represented as the baby lung ventilation. Moreover, those findings remain to be confirmed in trauma patients.

The main goal of the present study was to assess in a population of severe trauma patients with chest injuries the relationship between ΔPrs observed at the onset of ARDS ($\Delta Prs_{\text{-ARDS}}$) and outcomes.

Methods

Study design

The charts of severe trauma patients (Injury Severity Score [ISS] > 15) admitted to Lapeyronie University Hospital (Level I Regional Trauma Center, Montpellier, France; Occitrauma network) over a 9-year period (January 2010 to December 2018) were reviewed retrospectively. We obtained approval from the local scientific and ethics committee of Montpellier University Hospital, Comité d'Organisation et de Gestion de l'Anesthésie Réanimation (COGAR), who stated that informed consent from the patient or next of kin was not required.

Inclusion criteria

Consecutive trauma patients with chest trauma (Abbreviated Injury Scale [AIS] ≥ 1) who required invasive mechanical ventilation for a minimum of 48 hours were included in the present study [12]. Exclusion criteria were as follows: minors, immediate death, admission from another hospital, absence of initial computed tomography (CT) scan and incomplete medical records.

Respiratory management of patients

All patients included in the study were ventilated in our unit using a lung-protective mechanical ventilation protocol as defined in the literature: low tidal volume between 6 and 8 ml/kg of ideal body weight, limited P_{PLAT} and PEEP [13]. Ventilatory parameters were set to avoid intrinsic PEEP. Thoracic drainage was performed if necessary, with surgical dissection by an anterior approach for pneumothoraces (14 Fr) and axillary approach for haemothoraces (24 or 28 Fr). In specific settings such as severe bronchopleural fistulae, unilateral lung injuries, or refractory hypoxemia, an alternative to conventional mechanical ventilation may be used; for example, separated lung ventilation in the prone position, high-frequency percussive ventilation, or veno-venous extracorporeal membrane oxygenation in extreme cases.

Data collection

The following data were extracted from the medical records: age, sex, mechanism, characteristics of injuries, mortality, duration of mechanical ventilation, length of stay in the intensive care unit (ICU) and hospital length of stay. In addition, ISS and AIS scores were obtained for each body region as well as the Simplified Acute Physiology Score (SAPS) II. Prone position, pH, partial pressure of carbon dioxide in arterial blood ($PaCO_2$), PaO_2/F_IO_2, lactate, haemoglobin, base deficit, respiratory rate, V_T, PEEP, P_{PLAT}, respiratory system compliance (Crs), elastance of respiratory system (Ers), ΔPrs and PaO_2/F_IO_2 were recorded. Mechanical power (MP) was also computed as the product of ΔPrs in Newtons (cm H_2O × 0.098), tidal volume and respiratory rate. It was expressed as J/min and was included in the analysis [14]. For the non ARDS group respiratory parameters were collected the day of admission following initial stabilisation. For the ARDS group these parameters were collected on the onset day of ARDS.

In addition, specific data on chest trauma were collected based on the CT scan on admission (size of pulmonary contusions, pneumothoraces, haemothoraces, rib fractures, flail chest and atelectasis) allowing the Thoracic Trauma Severity (TTS) score on admission to be determined on a scale ranging from 0 to 25 points [15, 16].

Study definitions

ARDS was defined using the Berlin criteria consensus definition [17]: (1) timing: onset within 1 week of a known clinical insult or new or worsening respiratory symptoms; (2) chest imaging: bilateral opacities, not fully explained by effusions, lobar/lung collapse or nodules; (3) origin of oedema: respiratory failure not fully explained by cardiac failure or fluid overload. The ARDS diagnoses were retrospectively reviewed by two physicians (SR and MA). Patients were thus categorized into two groups according to the occurrence (ARDS group) or the absence of ARDS (no ARDS group). Timing was also considered: early ARDS (onset in the first 48 h after admission) and late ARDS (onset 48 h after admission). In the subgroup of ARDS patients, $\Delta Prs\text{-}_{ARDS}$ was the ΔPrs value that was observed at the onset of ARDS. The duration of mechanical ventilation, length of stay in the ICU and hospital length of stay for the ARDS group were extracted from medical records.

Statistical analysis

The study population was first divided into two groups according to the occurrence of ARDS (ARDS and no ARDS groups) and compared. Quantitative variables were expressed as the mean (standard deviation [SD]) or median (interquartile range, 25–75%) and compared using the Student t test or Wilcoxon-Mann-Whitney test as appropriate. Qualitative variables were expressed as number (%) and compared using the chi-squared test or Fisher test as appropriate. A descriptive analysis of the ARDS group focused on respiratory parameters was thus performed. Box plots was used to expose these data. In the case of missing data, the number of missing values is clearly stated for each variable. An a priori threshold for ΔPrs was defined based on previous results in the field (> 14 mmHg) [9, 18]. Similarly, the threshold for MP was defined a priori as ≥ 12 J/min [14].

Thereafter, a specific analysis on predicting severity of ARDS using ΔPrs and ROC curve analysis in the ARDS subgroup was performed. The area under the ROC curve (AUC) was expressed with the 95% confidence interval (CI). A survival analysis for weaning from mechanical ventilation was performed. Weaning from mechanical ventilation is censored by death before extubation. To draw Kaplan-Meier curves, the ARDS subgroup was divided according to the value of ΔPrs or MP. The thresholds defined a priori for ΔPrs and MP (> 14 mmHg and ≥ 12 J/min respectively) were conserved in this part of the analysis. The log-rank test was used to compare these curves. False-positive and false-negative rates were determined.

Finally, the relationship between ΔPrs or MP and different outcomes (duration of mechanical ventilation, occurrence of death, length of stay in the hospital and in the ICU) were assessed using a multivariate Cox regression analysis. In this analysis, the occurrence of ventilator-associated pneumonia was particularly considered as a confounding factor with the duration of mechanical ventilation. The statistical analysis was performed by CD and KB from the Clinical Research and Epidemiology Unit of Montpellier University Hospital using SAS statistical software (version 9.4; SAS Institute; Cary, NC). Statistical significance was set at a bilateral alpha risk < 0.05.

Results

Among the 1669 patients admitted with chest trauma to our trauma ICU during the study period, 761 (46%) met the inclusion criteria. Of these, 460 (60%) were not eligible for analysis due to duration of mechanical ventilation less than 48 h in 432 cases and a lack of data in 28 cases Finally, 301 patients were included in the study. Of these, 35 patients (11%) were excluded from our analysis due to early death (traumatic brain injury or early haemorrhagic shock). The 266 remaining patients were thus included in our analysis. The flowchart of the study is presented in Fig. 1. Among the study population, 204 patients were male (77%), the mean age was 43.0 (± 19.3) years, and the mean ISS was 32 (± 10). One-hundred and fifty patients (56%) were classified as AIS ≥ 3 thoracic trauma, 84 (31%) were AIS ≥ 3 abdominal trauma and 135 (51%) were AIS ≥ 3 traumatic brain injury. The mean TTS score was 9.5 (± 4.9). The mortality rate was 6%; 15 patients died during the hospital stay. The mean PaO_2/FiO_2 ratio on admission was 317 (± 173). A total of 194 patients (73%) experienced ARDS in the first week after admission; 52% early ARDS and 48% late ARDS **(Appendix. 1)**. The mean duration of mechanical ventilation was 16.3 (± 13.9) days. The main demographic characteristics of the patients are presented in Table 1.

Table 1
Baseline characteristics

Variable		Total population	ARDS		*p*
			No	Yes	
		N = 266	n = 72	n = 194	
Sex, n (%)	Men	204 (76.69)	50 (69.44)	154 (79.38)	0.09
Age (years)		43.60 (± 19.28)	34.92 (± 16.56)	46.82 (± 19.27)	< 0.01
BMI (kg/m^2)		25.04 (± 4.60)	23.69 (± 4.88)	25.54 (± 4.40)	< 0.01
SAPS II		39.87 (± 15.61)	35.40 (± 12.13)	41.53 (± 16.44)	0.01
ISS		31.70 (± 9.95)	31.36 (± 9.39)	31.83 (± 10.17)	0.81
TTS		9.49 (± 4.86)	5.73 (± 3.29)	10.88 (± 4.62)	< 0.01
Thoracic trauma, n (%)					0.03
	Mild (AIS 1 or 2)	116 (43.61)	41 (56.94)	75 (38.66)	
	Moderate (AIS 3 or 4)	133 (50.00)	28 (38.89)	105 (54.12)	
	Severe (AIS 5 or 6)	17 (6.39)	3 (4.17)	14 (7.22)	
Pneumothoraces, n (%)					0.05
	No	129 (48.50)	34 (47.22)	95 (48.97)	
	Mild (AIS 1 or 2)	58 (21.80)	23 (31.94)	35 (18.04)	
	Moderate (AIS 3 or 4)	30 (11.28)	7 (9.72)	23 (11.86)	
	Severe (AIS 5 or 6)	49 (18.42)	8 (11.11)	41 (21.13)	
Hemothoraces, n (%)					0.09
	No	177 (66.54)	56 (77.78)	121 (62.37)	
	Mild (AIS 1 or 2)	28 (10.53)	5 (6.94)	23 (11.86)	

Data are expressed as mean ± SD, or as number of patients (percentage) as appropriate

BMI, body mass index; SAPS II, Simplified Acute Physiology Score 2; ISS, Injury Severity Score; TTS, Thoracic Trauma Severity score; AIS, Abbreviated Injury Score

Variable		Total population	ARDS		*p*
			No	Yes	
	Moderate (AIS 3 or 4)	36 (13.53)	8 (11.11)	28 (14.43)	
	Severe (AIS 5 or 6)	25 (9.40)	3 (4.17)	22 (11.34)	
Traumatic brain injury, n (%)					0.70
	No	106 (39.85)	28 (38.89)	78 (40.21)	
	Mild (AIS 1 or 2)	25 (9.40)	9 (12.50)	16 (8.25)	
	Moderate (AIS 3 or 4)	112 (42.11)	28 (38.89)	84 (43.30)	
	Severe (AIS 5 or 6)	23 (8.65)	7 (9.72)	16 (8.25)	
Abdominal trauma, n (%)					0.30
	No	130 (48.87)	31 (43.06)	99 (51.03)	
	Mild (AIS 1 or 2)	52 (19.55)	12 (16.67)	40 (20.62)	
	Moderate (AIS 3 or 4)	68 (25.56)	24 (33.33)	44 (22.68)	
	Severe (AIS 5 or 6)	16 (6.02)	5 (6.94)	11 (5.67)	
Hemoperitoneum, n (%)					0.08
	No	186 (69.92)	43 (59.72)	143 (73.71)	
	Mild (AIS 1 or 2)	37 (13.91)	16 (22.22)	21 (10.82)	
	Moderate (AIS 3 or 4)	24 (9.02)	8 (11.11)	16 (8.25)	
	Severe (AIS 5 or 6)	19 (7.14)	5 (6.94)	14 (7.22)	
pH at entry		7.34 (± 0.09)	7.37 (± 0.08)	7.34 (± 0.09)	0.01

Data are expressed as mean ± SD, or as number of patients (percentage) as appropriate

BMI, body mass index; SAPS II, Simplified Acute Physiology Score 2; ISS, Injury Severity Score; TTS, Thoracic Trauma Severity score; AIS, Abbreviated Injury Score

Variable	Total population	ARDS		*p*
		No	Yes	
Base deficit at entry	5.72 (± 3.59)	5.64 (± 3.89)	5.75 (± 3.48)	0.43
Lactates at entry	3.58 (± 2.59)	3.58 (± 2.40)	3.59 (± 2.66)	0.94
Haemoglobin at entry	11.54 (± 2.64)	11.33 (± 2.84)	11.62 (± 2.56)	0.47
Data are expressed as mean ± SD, or as number of patients (percentage) as appropriate				
BMI, body mass index; SAPS II, Simplified Acute Physiology Score 2; ISS, Injury Severity Score; TTS, Thoracic Trauma Severity score; AIS, Abbreviated Injury Score				

ΔPrs was significantly higher in the ARDS group than in the no ARDS group; 11.8 (± 2.9) cm H_2O vs 12 (± 2.03) cm H_2O ($p = 0.04$). P_{PLAT} and MP showed the same behaviour; 18 (± 3) cm H_2O vs 16 (± 2) cm H_2O, ($p < 0.001$) and 11.5 (± 3.6) J/min vs 9.6 (± 3.0) J/min ($p < 0.001$), respectively. The characteristics of the ventilatory parameters and outcomes of the patients are presented in Table 2.

Table 2
Respiratory characteristics and outcome

Variable		Total population	ARDS		*p*
			No	Yes	
		N = 266	n = 72	n = 194	
PAO_2/FiO_2 at entry		317.03 (± 173.30)	494.54 (± 166.90)	251.14 (± 121.95)	< 0.01
Worst PaO_2/FiO_2		232.49 (± 120.65)	397.13 (± 75.90)	171.39 (± 63.26)	< 0.01
ARDS severity, n (%)					
	Mild	64 (24.06)		64 (32.99)	
	Moderate	100 (37.59)		100 (51.55)	
	Severe	30 (11.28)		30 (15.46)	
Early ARDS, n (%)					
	< 48 h	101 (37.97)		101 (52.06)	
	≥ 48 h	93 (34.96)		93 (47.94)	
Day of occurrence of ARDS		2.22 (± 2.41)		2.22 (± 2.41)	
Driving pressure (ΔPrs; cm H_2O)		11.58 (± 2.70)	10.96 (± 2.03)	11.81 (± 2.89)	0.04
Driving pressure, n (%)					0.27
	< 14 cm H_2O	230 (86.47)	65 (90.28)	165 (85.05)	
	> 14 cm H_2O	36 (13.53)	7 (9.72)	29 (14.95)	
Mean Driving pressure over the first 5 days (cm H_2O)		11.47 (± 2.24)	10.93 (± 1.84)	11.68 (± 2.34)	0.02
Plateau pressure (P_{PLAT}; cm H_2O)		17.26 (± 3.09)	16.23 (± 2.31)	17.64 (± 3.26)	< 0.01
Static compliance (Crs ; cm H_2O)		47.53 (± 11.45)	47.69 (± 8.42)	47.47 (± 12.40)	0.53

Data are expressed as mean ± SD, or as number of patients (percentage) as appropriate.VAP, ventilator-acquired pneumonia

Variable		Total population	ARDS		*p*
			No	Yes	
Mechanical power (MP; cm H_2O)		11.00 (± 3.50)	9.60 (± 2.98)	11.52 (± 3.55)	< 0.01
VAP, n (%)					< 0.01
	No	110 (41.35)	47 (65.28)	63 (32.47)	
	Yes	156 (58.65)	25 (34.72)	131 (67.53)	
Death (, n (%)					0.08
	No	251 (94.36)	71 (98.61)	180 (92.78)	
	Yes	15 (5.64)	1 (1.39)	14 (7.22)	
Length Duration of mechanical ventilation (days)		16.32 (± 13.89)	12.30 (± 12.34)	17.81 (± 14.16)	< 0.01
28 Free days free of ventilation		13.05 (± 9.95)	16.78 (± 9.36)	11.67 (± 9.83)	< 0.01
Length of stay in ICU (days)		26.32 (± 20.43)	20.72 (± 18.02)	28.40 (± 20.92)	< 0.01
Length of hospital stay (days)		36.22 (± 28.57)	30.47 (± 19.08)	38.37 (± 31.15)	0.03
Data are expressed as mean ± SD, or as number of patients (percentage) as appropriate.VAP, ventilator-acquired pneumonia					

The results of the descriptive analysis are shown in Fig. 2 for the subgroup analysis of the ARDS group. The AUC of the ROC curve for ΔPrs_{-ARDS} in predicting the severity of ARDS was 0.59 (95% CI, 0.48–0.71; p = 0.06) for moderate and severe ARDS **(Appendix. 2).** and 0.60 (95% CI, 0.58–0.61; p = 0.04) for severe ARDS. A ΔPrs_{-ARDS} value > 14 cm H_2O had a sensitivity of 20.8% (95% CI, 13.8%–27.7 %), a specificity of 88.9% (95% CI, 81.1–96.6%), a predictive positive value of 79.4 (95% CI, 65.8%–93 %) and a negative predictive value of 35.2 (95% CI, 27.8%–42.6 %) for predicting severe ARDS. In the survival analysis using the a priori ΔPrs_{-ARDS} threshold > 14 cm H_2O, the duration of mechanical ventilation did not differ between the high and the low driving pressure groups, HR = 0.82 (95%CI, 0.57–1.2), p = 0.3 Fig. 3. This was also observed for ICU length of stay (p = 0,78) and hospital length of stay (p = 0.75). Kaplan-Meier curves using MP are presented in **(Appendix. 3).** In the multivariate analysis, ΔPrs_{-ARDS} was not found to be independently associated with the duration of mechanical ventilation (hazard ratio [HR], 1.01; 95% CI, 0.95–1.07; p = 0.8) or mortality (HR, 1.07; 95% CI, 0.9–1.28; p = 0.45) Table 3. When considering ΔPrs_{-ARDS} in two groups (≤ 14 cm H_2O and > 14 cm H_2O) no association was found for mortality or duration of

mechanical ventilation (Table 3). Similarly, no other respiratory parameters, such as P_{PLAT} (HR, 0.98; 95% CI, 0.93–1.02; $p = 0.33$), Crs (HR, 0.98; 95% CI 0.98–1.00; $p = 0.12$) and MP (HR, 0.98; 95% CI, 0.94–1.02; $p = 0.22$), were found to be independently associated with duration of mechanical ventilation duration or mortality **(Appendix. 4).**

Table 3
Multivariate cox regression analysis results on duration of mechanical ventilation

Variable	Modality	Hazard ratio	95% confidence interval	p
Duration of ventilation				
Age (years)	+ 1 year	0.99	0.99-1.00	< 0.04
SAPS II	+ 1 point	0.97	0.96–0.99	< 0.0001
ISS	+ 1 point	0.99	0.97–1.01	0.25
Chest injury				
Reference	AIS < 2			
	≥ 2 AIS ≤ 4	0.95	0.7–1.29	0.75
	AIS > 4	0.51	0.26–0.97	0.04
Head injury				
Reference	No injury			
	AIS < 2	0.91	0.56–1.48	0.7
	≥ 2 AIS ≤ 4	0.84	0.6–1.18	0.31
	AIS > 4	0.45	0.25–0.81	0.007
ΔPrs_{-ARDS}	+ 1 cm H_2O	1.01	0.95–1.08	0.84
Group outcome[a]				
Mortality	ΔPrs_{-ARDS} ->14 cm H_2O	1.42	0.44–4.54	0.56
Ventilation duration	ΔPrs_{-ARDS} >14 cm H_2O	0.9	0.61–1.33	0.59

Discussion

This is the first study to assess the association of ΔPrs with outcomes in a specific population of chest trauma patients. The main finding is that the ΔPrs value at the onset of ARDS was not associated with the duration of mechanical ventilation, mortality risk or ICU length of stay either in survival analysis or multivariate analysis. Our analysis demonstrates therefore that ΔPrs is an unreliable predictor of outcomes in this specific population.

Nowadays, ΔPrs, usually called the driving pressure, is used universally in clinical practice to reflect the mechanical stress generated by V_T on the ventilated lung [11]. ΔPrs also means respiratory system compliance for a given V_T. Thus, a high ΔPrs may indicate impairment of respiratory system compliance by a decrease in the functional size of the lung. This phenomenon, called baby lung within context of ARDS, is related to alveolar collapse.

Many studies have demonstrated that ΔPrs is associated with mortality in patients with ARDS [9, 10, 18]. However, these previous studies focused on ARDS in medical settings. The morbid association between ΔPrs and mortality may nevertheless be altered in different clinical situations. Thus, De Jong et al [18] have reported that in obese patients with ARDS, ΔPrs was not associated with mortality. Similarly, our data suggest the absence of a relationship between ΔPrs and outcomes in the case of patients with ARDS related to trauma. One of the main physiopathological explanations for this difference observed between the medical and trauma contexts could be significant modifications of chest wall compliance related to traumatic injuries. Traumatic parietal dehiscence increases chest wall compliance, which may lead to an increase in ΔPrs [19]. Thus, ΔPrs is a reflection of both chest wall and lung compliance and it may be directly modified by strong variation in chest wall compliance in cases of severe chest trauma. ΔPrs = chest wall driving pressure, ΔPcw + transpulmonary driving pressure, ΔPl; consequently, ΔPl would offer a better reflection of actual lung compliance and alveolar collapse [20]. Similarly to obese patients, in chest trauma patients, much of the pressure provided by the ventilator will be used to distend the chest wall rather than the lung. Hence, there may not be an increase in transpulmonary pressure with accompanying lung overdistension. The only way to monitor the ΔPl of real interest from bench to bedside is to measure oesophageal pressure, which is a surrogate for ΔPcw. Thus, physicians may quickly differentiate a high ΔPrs due to an increase in ΔPcw, without a real lung injury, or inversely a high ΔPrs with a normal ΔPcw, a sign of lung pathology.

ARDS related to chest trauma represents a low percentage of case of ARDS and has its own specific properties [21]. Its incidence is estimated to be between 10% and 30% of critically ill trauma patients, mainly depending on the severity of the trauma in severely injured patients. Surprisingly, trauma-related ARDS is known to be to twice less likely to lead to death than medical ARDS [22]. Our cohort presents similar results with a low mortality rate in the ARDS group (7%). Traumatic ARDS is characterized by a typical immunological profile different from ARDS in a medical setting. The early phase following trauma is thus characterized by an important and uncontrolled inflammatory response in the lung tissues. The importance of this inflammatory response depends on the intensity of the initial trauma but also on the genetic profile of the patient. A previous work by Xiao et al. [23] in a severe trauma population showed that the early leukocyte genomic response was simultaneously associated with increased expression of genes involved in the systemic inflammatory, innate immune, and compensatory anti-inflammatory responses, as well as in the suppression of genes involved in adaptive immunity. All these modifications induce a massive release of damage-associated molecular pattern molecules from injured tissues [24]. Massive release of epithelial biomarkers such as sRAGE, for example, is responsible for dysfunction of the capillary-alveolar barrier, increase in inflammation, and oxidative stress favouring fibrosis in cases of

ARDS [25, 26]. These specific characteristics explain the specificities of ARDS after chest trauma, its lower lethality rate, and maybe the absence of association between ΔPrs and outcomes in the trauma context.

The present study has some limitations. First, its design was monocentric with a retrospective analysis, which limits extrapolation of the results. However, the data were collected prospectively with the ICU software, and the management of patients with ARDS was standardized according to international recommendations. Only a few data are missing regarding the driving pressure (28 of 329; 8%) in trauma patients under mechanical ventilation. Second, no threshold analysis was performed according to the ΔPrs. Patients who died from traumatic brain injury or from haemorrhagic shock in first 48 h after admission were excluded from our study. Consequently, only 15 non-survivors are present in our final analysis. This may lead to a lack of power and threshold analysis is impossible. However, previous work also used a threshold of 14 cm H_2O for ΔPrs [9, 18]. Third, patients in our cohort were severely injured with a mean ISS of 32, and about 25% of the cohort also had abdominal injuries. Increase in abdominal pressure may also be involved in the change in chest wall compliance and create an analysis bias. It is therefore impossible to exclusively attribute our results only to chest injuries. However, multiple trauma is a frequent situation, which makes our study close to real life.

Conclusion

The results of the present study demonstrate that driving pressure in a specific population of chest trauma patients was a poor predictor for ARDS severity and had no correlation with worse outcomes. Changes in chest compliance due to traumatic injuries could explain this unexpected observation. Therefore, our results suggest that the use of transpulmonary pressure would be relevant in chest trauma patients to directly monitor injured lungs and guide an optimal mechanical ventilation strategy. Further studies will be necessary to prove the positive impact of transpulmonary pressure on chest trauma management.

Declarations

Ethics approval and consent to participate

We obtained approval from the local scientific and ethics committee of Montpellier University Hospital, Comité d'Organisation et de Gestion de l'Anesthésie Réanimation (COGAR), who stated that informed consent from the patient or next of kin was not required.

Consent for publication

Informed consent from the patient or next of kin was not required.

Availability of data and materials

All data are extracted from medical record.

Competing interests

The authors declare no competing interests.

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Authors' contributions

S.R., M.A, and K.B collected clinical data for this study; S.R., M.A, and K.B collected

pharmaceutical and blood requirements. J.C, S.R, A. DJ, C.D prepared the draft of the study and conducted the statistical analysis. S.R. and M.A. wrote the article, which was revised by S.J, T.L, A.DJ and X.C.

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Figures

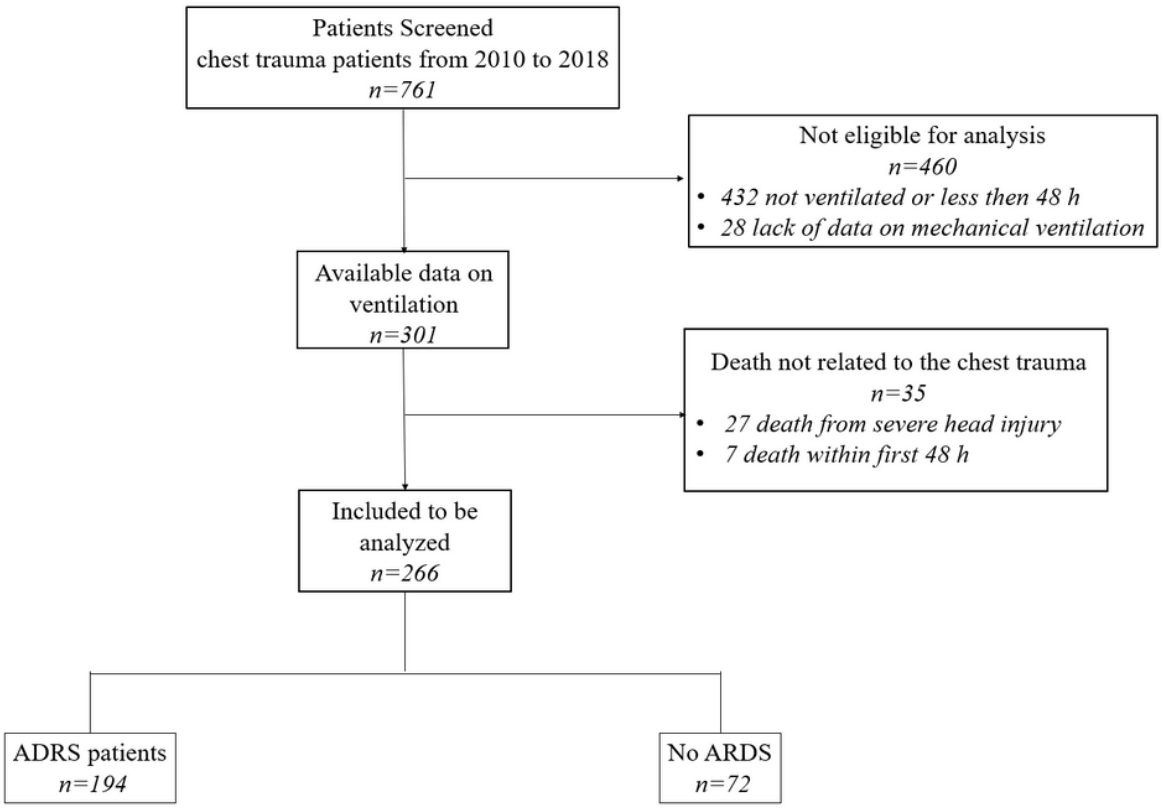

Figure 1. Flowchart of the study

Figure 1

Flowchart of the study

Figure 2. Correlation between driving pressure on the day of ARDS (ΔPrs_{-ARDS}) and the worst PaO2/FiO2 in chest trauma patients

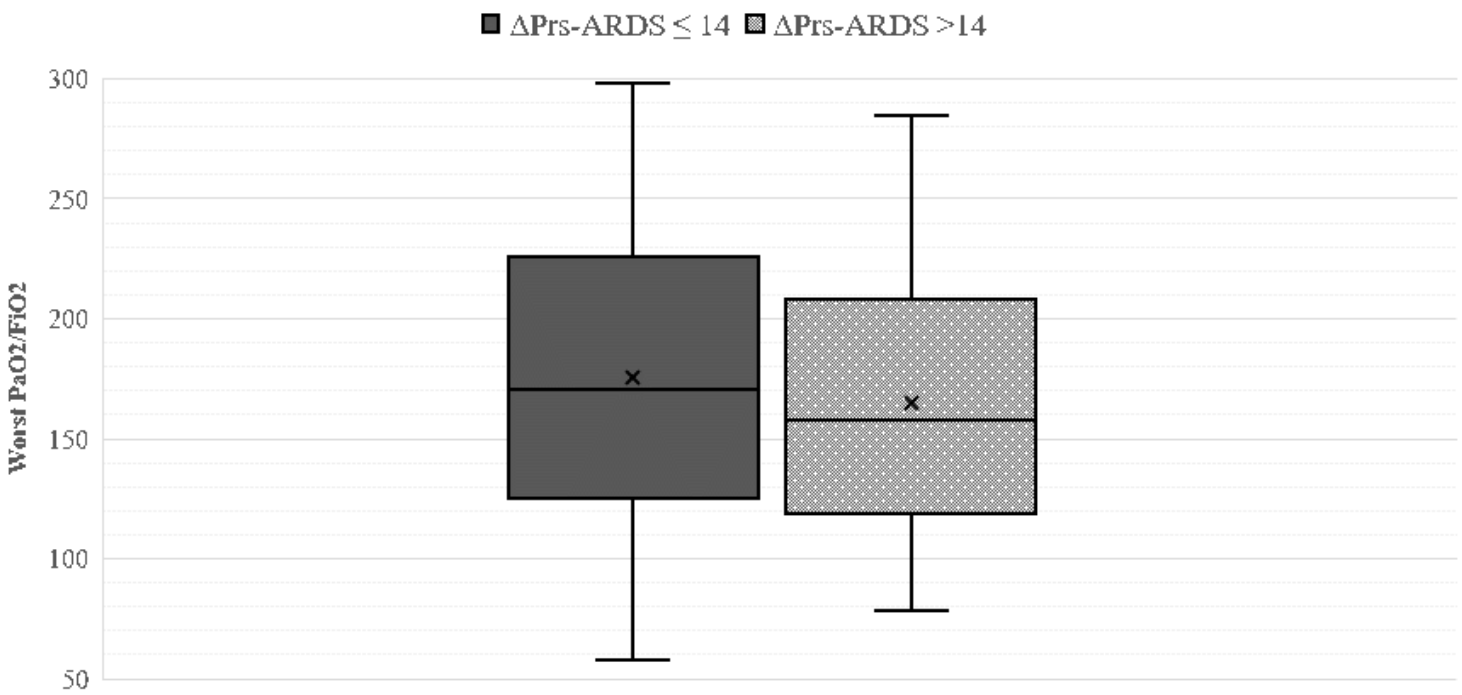

Figure 2

Correlation between ΔPrs-ARDS and the worst PaO2/FiO2 in ARDS patients.

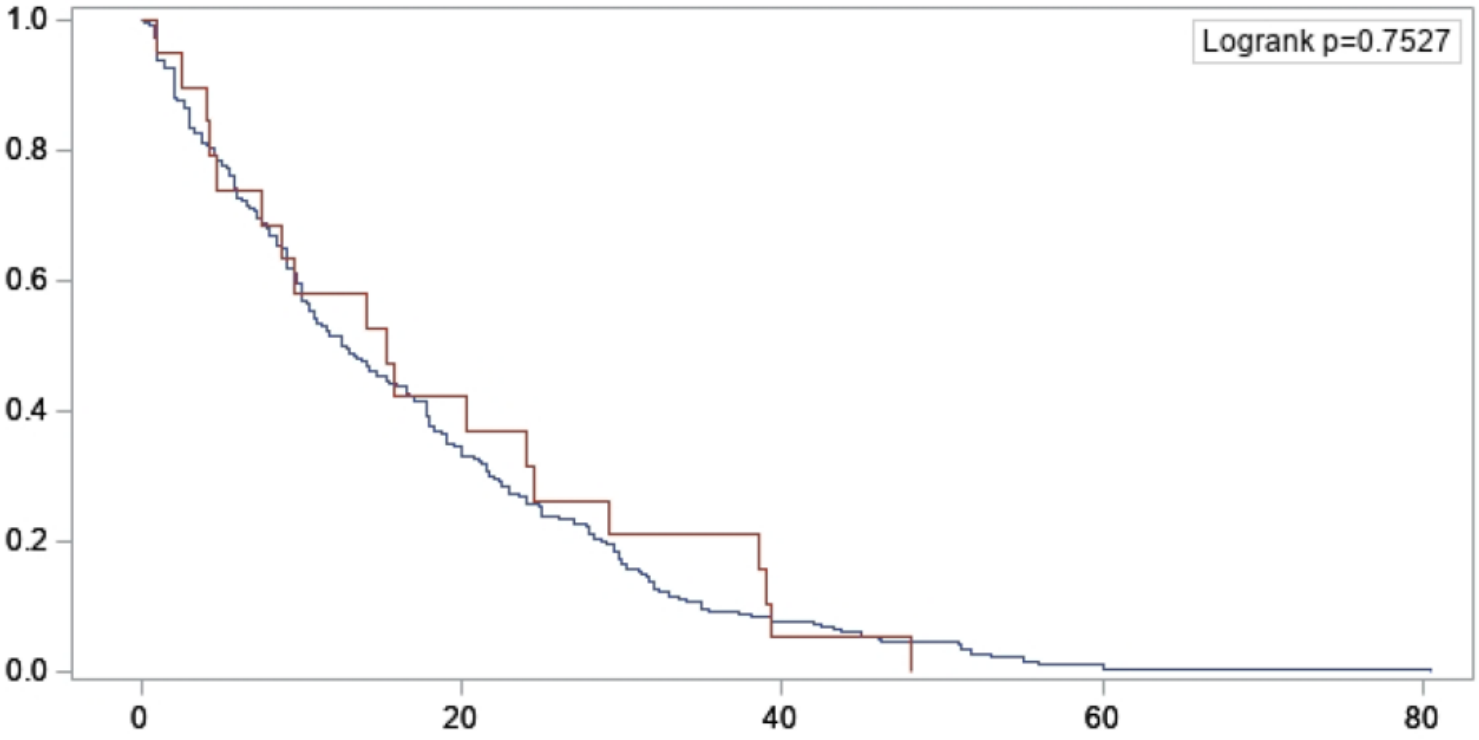

Figure 3

Kaplan-Meier curves for time to weaning from mechanical ventilation according to driving pressure level

Supplementary Files

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