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Paradoxical response to chest wall loading predicts a favorable mechanical response to reduction in tidal volume or PEEP

John Selickman^{1*}, Pierre Tawfik¹, Philip S. Crooke², David J. Dries^{3,4}, Jonathan Shelver⁵, Luciano Gattinoni⁶ and John J. Marini^{1,7}

Abstract

Background: Chest wall loading has been shown to paradoxically improve respiratory system compliance (C_{RS}) in patients with moderate to severe acute respiratory distress syndrome (ARDS). The most likely, albeit unconfirmed, mechanism is relief of end-tidal overdistension in 'baby lungs' of low-capacity. The purpose of this study was to define how small changes of tidal volume (V_T) and positive end-expiratory pressure (PEEP) affect C_{RS} (and its associated airway pressures) in patients with ARDS who demonstrate a paradoxical response to chest wall loading. We hypothesized that small reductions of V_T or PEEP would alleviate overdistension and favorably affect C_{RS} and conversely, that small increases of V_T or PEEP would worsen C_{RS} .

Methods: Prospective, multi-center physiologic study of seventeen patients with moderate to severe ARDS who demonstrated paradoxical responses to chest wall loading. All patients received mechanical ventilation in volume control mode and were passively ventilated. Airway pressures were measured before and after decreasing/increasing V_T by 1 ml/kg predicted body weight and decreasing/increasing PEEP by 2.5 cmH₂O.

Results: Decreasing either V_T or PEEP improved C_{RS} in all patients. Driving pressure (DP) decreased by a mean of 4.9 cmH₂O (supine) and by 4.3 cmH₂O (prone) after decreasing V_T , and by a mean of 2.9 cmH₂O (supine) and 2.2 cmH₂O (prone) after decreasing PEEP. C_{RS} increased by a mean of 3.1 ml/cmH₂O (supine) and by 2.5 ml/cmH₂O (prone) after decreasing V_T . C_{RS} increased by a mean of 5.2 ml/cmH₂O (supine) and 3.6 ml/cmH₂O (prone) after decreasing PEEP ($P < 0.01$ for all). Small increments of either V_T or PEEP worsened C_{RS} in the majority of patients.

Conclusion: Patients with a paradoxical response to chest wall loading demonstrate uniform improvement in both DP and C_{RS} following a reduction in either V_T or PEEP, findings in keeping with prior evidence suggesting its presence is a sign of end-tidal overdistension. The presence of 'paradox' should prompt re-evaluation of modifiable determinants of end-tidal overdistension, including V_T , PEEP, and body position.

Background

Airway driving pressure (DP) is used routinely to guide 'lung-protective' ventilation in acute respiratory distress syndrome (ARDS). The effect of tidal volume (V_T) on DP is determined by tidal compliance of the integrated respiratory system (C_{RS}), which is comprised of the lungs and chest wall. For a fixed V_T and positive end-expiratory pressure (PEEP), any net change in C_{RS} alters DP in the opposite direction. Because they share a common

*Correspondence: selic002@umn.edu

¹ Department of Pulmonary and Critical Care Medicine, University of Minnesota School of Medicine, 640 Jackson Street, Mail Stop 11203B, Minneapolis St. Paul, MN 55101, USA
Full list of author information is available at the end of the article



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volume, a decrease in compliance of either the lungs (C_L) or the chest wall (C_{CW}) simultaneously decreases C_{RS} unless there is a compensatory improvement in its counterpart.

In recent work, reducing lung volume and C_{CW} by external compression, or 'loading', has been noted to improve C_{RS} in patients with moderate to severe ARDS [1–6]; by immediate implication, loading must therefore result in improved C_L and lower transpulmonary pressure in these patients. The most appealing (but as yet unconfirmed) explanation for this counterintuitive mechanical 'paradox' (i.e., decreased C_{CW} resulting in improved C_{RS}) is relief of end-tidal overdistension that occurs in the unloaded state.

The purpose of this study was to define how choice of V_T and PEEP affect C_{RS} in ARDS patients demonstrating a paradoxical response to chest wall loading. When V_T operates in the linear ('middle') portion of the pressure–volume relationship, as intended for lung protection, small changes of V_T and/or PEEP should leave C_{RS} unaffected [7]. On the other hand, assuming the underlying mechanism of the loading paradox is relief of end-tidal overdistension, we reasoned that 'paradox positive' patients would demonstrate a disproportionate reduction in DP (and by extension, increased C_{RS}) following a small decrease of V_T or PEEP, both of which alleviate end-tidal overdistension. Conversely, we reasoned that such patients would demonstrate a disproportionate increase in DP (and decreased C_{RS}) following a small increase of V_T or PEEP—changes which would exacerbate any end-tidal overdistension.

Methods

This prospective, multi-center physiologic study was performed in two medical intensive care units (Regions Hospital, St. Paul MN, USA and Methodist Hospital, Minneapolis, MN, USA), with all data collected by the same investigative team between December 2021 and March 2022.

Patients

Consecutive patients with ARDS (as defined by the Berlin consensus criteria [8]) who demonstrated no signs of active breathing were enrolled and evaluated. All received invasive mechanical ventilation under controlled conditions, with passive breathing assured by either ongoing administration of neuromuscular blockers or deep sedation sufficient to suppress any evidence of active breathing.

Ventilatory strategy

All patients received mechanical ventilation in volume regulated, control mode (decelerating flow profile) using

one of two ventilators: Puritan Bennett 980 (Medtronic; Carlsbad, California, USA) or Maquet Servo-I (Siemens; Bloomfield, Connecticut, USA). Baseline measurements were performed using the V_T , PEEP and respiratory rate already prescribed by the clinical team for routine management prior to study enrollment.

Measurements

Measurements were performed in the position of care, either supine or prone, and this was not altered for the purposes of data collection. When possible, study measurements were repeated in the opposite position within 24 h, provided that the criteria for passive breathing were still met.

In the supine orientation, measurements were performed in a semi-recumbent position with the head elevated to 30°; in the prone orientation, measurements were performed with the bed flat (0°). The highest airway pressure during inflation was recorded as the peak pressure. Plateau pressure was measured at least two seconds after performing an end-inspiratory pause. Total PEEP (the sum of set PEEP and auto-PEEP) was measured at least three seconds after performing an end-expiratory pause, assuring that zero flow was achieved.

Measurement of tidal airway pressures were repeated after the following interventions: (1) increasing V_T by 1 mL/kg predicted body weight (PBW); (2) decreasing V_T by 1 mL/kg PBW; (3) increasing PEEP by 2.5 cmH₂O; and (4) decreasing PEEP by 2.5 cmH₂O.

Procedure for chest wall loading to detect paradoxical mechanical response

Following baseline measurements (obtained in an unloaded state), manual loading of the chest wall was performed. In the supine position, loading was accomplished by placing a hand over the patient's umbilicus perpendicular to the axis between the xiphoid process and the pubis; in the prone position, the hand was placed at the approximate mid-point between the inferior costal margin and the iliac crest, perpendicular to the lumbar spine. To gauge load adequacy, an end-inspiratory hold was then performed and manual pressure applied until there was an upward deflection of the pressure–time waveform of ≥ 2 cmH₂O, at which point chest wall loading was considered sufficient to influence transpulmonary pressure during tidal breathing. The inspiratory hold on the ventilator was then released, while continuing to apply sustained manual pressure on the abdomen or lumbar region. After five breaths had been delivered, measurements of tidal airway pressure were repeated, and manual pressure was then released.

Statistical analysis

A normality test was performed for all samples to verify a normal distribution. When normality was confirmed, the paired t-test was used to compare mean values of DP and C_{RS} at baseline, during chest wall loading, and following alteration of ventilator parameters as outlined above. In all instances where the normality assumption was not satisfied results were confirmed using the Mann–Whitney test. Differences at the level of a two-tailed P value < 0.05 were considered statistically significant.

Results

Nineteen patients with ARDS were studied, of whom seventeen had ARDS secondary to novel coronavirus (C-ARDS). Seventeen demonstrated a paradoxical response to chest wall loading. Of these, paired measurements were obtained in both the supine and prone positions in eight; in the remaining nine patients, five were evaluated in only the supine position, and four were evaluated in only the prone position. All but one patient had either moderate or severe ARDS, all were ventilated in accordance with lung protective principles for ventilation, and none received extracorporeal support for gas exchange or hemodynamics (Table 1). Mortality at thirty days from the time of data acquisition was 70.6% (12/17). Ventilator settings and gas exchange at baseline are reported in Table 2.

Chest wall loading

Chest wall loading reduced DP by a mean of 3.6 ± 2.3 cmH₂O in the supine position and by 2.7 ± 2.3 cmH₂O in the prone position ($P < 0.01$ for both). C_{RS} improved following chest wall loading by a mean of 6.1 ± 3.5 mL/cmH₂O in the supine position and 4.1 ± 3.0 mL/cmH₂O in the prone position ($P \leq 0.001$ for both) (Table 3 and Fig. 1).

Decreasing V_T and PEEP in the unloaded state.

Decreasing V_T resulted in a reduced DP and an improved C_{RS} in all seventeen patients. DP decreased by a mean of 4.9 ± 2.8 cmH₂O in the supine position and 4.3 ± 2.2 cmH₂O in the prone position ($P \leq 0.001$ for both). C_{RS} improved by a mean of 3.1 ± 2.1 mL/cmH₂O in the supine position and 2.5 ± 2.1 mL/cmH₂O in the prone position ($P \leq 0.005$ for both) (Table 3 and Fig. 2).

Decreasing PEEP similarly resulted in reduced DP and improved C_{RS} in all seventeen patients. DP decreased by a mean of 2.9 ± 1.9 cmH₂O in the supine position and 2.2 ± 2.2 cmH₂O in the prone position ($P \leq 0.008$ for both). C_{RS} improved by a mean of 5.2 ± 3.4 mL/cmH₂O in the supine position and 3.6 ± 3.2 mL/cmH₂O in the prone position ($P \leq 0.004$ for both).

Table 1 Patient characteristics

Subject	Diagnosis	Age (years)	Gender	BMI (kg/m ²)	LOH (days)	LOI (days)	NMB	30-day survival
1	C-ARDS	59	Male	35	9	1	Y	N
2	C-ARDS	45	Female	31.5	22	14	N	Y
3	C-ARDS	55	Male	25.2	24	5	Y	N
4	C-ARDS	65	Male	25.2	26	23	N	N
5	ARDS	50	Female	23.5	15	13	Y	Y
6	C-ARDS	54	Male	28.5	14	14	N	Y
7	C-ARDS	70	Male	37.4	11	11	N	N
8	C-ARDS	47	Male	28.9	50	41	Y	N
9	ARDS	67	Female	25.3	1	2	Y	N
10	C-ARDS	70	Female	42.7	1	1	Y	N
11	C-ARDS	76	Male	31.7	11	2	N	N
12	C-ARDS	54	Male	29.2	10	1	Y	N
13	C-ARDS	62	Male	26.9	14	4	N	N
14	C-ARDS	30	Male	29.6	22	14	Y	Y
15	C-ARDS	63	Male	29.4	1	1	N	Y
16	C-ARDS	76	Female	30.1	13	6	N	N
17	C-ARDS	61	Male	28.8	41	37	Y	N
Mean		59.1		29.9	16.8	11.2		
SD		11.9		4.8	13.3	12.3		

BMI Body mass index, LOH Duration of hospitalization, LOI Duration of intubation, NMB Neuromuscular blockade, C-ARDS COVID-related acute respiratory distress syndrome, ARDS Acute respiratory distress syndrome, Y Yes, N No, SD Standard deviation

Table 2 Ventilator settings and gas exchange at baseline

Subject	Pa _{O2} /Fi _{O2}	Pa _{CO2} (mmHg)	V _T (mL/kg PBW)	PEEP set (cmH ₂ O)	RR	Ventilatory ratio
1	85	50.8	7	16	22	2.1
2	137.5	101	4.9	12	30	4.0
3	109.3	66.7	6	7.5	24	2.6
4	83	81.3	5.4	6	32	3.7
5	87	62.6	3.9	6	27	1.8
6	157.2	65.3	5.9	9	32	3.3
7	245	63.4	5.5	12	22	2.0
8	82.2	63.2	5.9	5	30	3.0
9	86.9	61.5	5.4	8	32	2.8
10	147	48.1	7	14	34	3.1
11	191.4	47.4	5.9	12	28	2.1
12	160	74.3	6.6	10	24	3.1
13	98.3	49	4.6	10	32	1.9
14	102.5	45	5.2	8	28	1.7
15	65.6	59	4.6	12	34	2.5
16	185.5	58.7	6.7	12	26	2.7
17	73.4	92.7	4.7	6	34	4.0
Mean	123.3	64.1	5.6	9.7	28.9	2.7
SD	50.7	15.7	0.9	3.2	4.2	0.7

Pa_{O2} Partial pressure of arterial oxygen, Fi_{O2} Fraction of inspired oxygen, Pa_{CO2} Partial pressure of arterial carbon dioxide, V_T Tidal volume, PEEP Positive end-expiratory pressure, RR Respiratory rate, SD Standard deviation. Ventilatory ratio is defined as [minute ventilation (mL/min) x Pa_{CO2} (mmHg)]/(predicted body weight x 100 x 37.5)

Table 3 Response to chest wall loading, decreased tidal volume, and decreased positive end-expiratory pressure Baseline measurements were performed (column A) followed by chest wall loading (column B). In the unloaded state, V_T was decreased by 1 mL/kg PBW (column C) and PEEP was decreased by 2.5 cmH₂O (column D) in all seventeen patients.

		Baseline (A)	Loading (B)	↓ V _T (C)	↓ PEEP (D)	A to B (p value)	A to C (p value)	A to D (p value)
Supine	DP (cmH ₂ O)	17.7 ± 7.7	14.1 ± 6.5	12.8 ± 5.4	14.8 ± 6.5	0.0001	< 0.0001	0.0001
	C _{RS} (mL/cmH ₂ O)	25.7 ± 11.8	31.8 ± 13.2	28.9 ± 13	30.9 ± 14.6	< 0.0001	0.0002	0.0002
Prone	DP (cmH ₂ O)	17.2 ± 6.2	14.5 ± 5.2	11.9 ± 3.5	15.0 ± 5.5	0.003	0.0001	0.008
	C _{RS} (mL/cmH ₂ O)	24 ± 10.8	28.1 ± 11.8	28.1 ± 10.8	27.6 ± 11	0.001	0.005	0.004

DP Driving pressure, C_{RS} Respiratory system compliance, V_T Tidal volume, PEEP Positive end-expiratory pressure

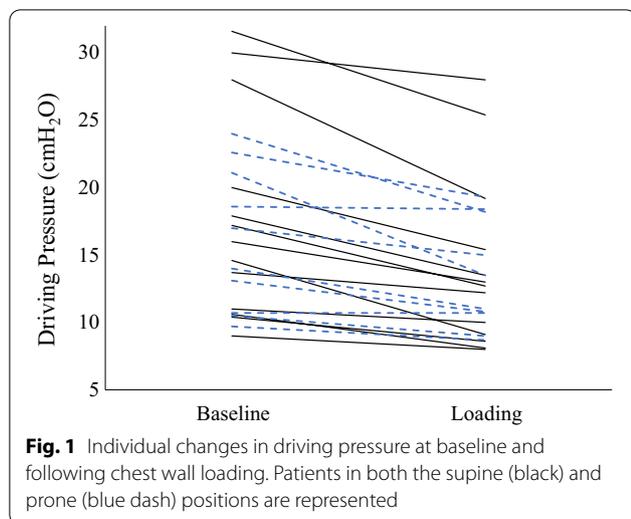
Increasing V_T and PEEP

In 6 patients, increasing V_T and/or PEEP resulted in an immediate rise of peak airway pressures to above 50 cmH₂O; in these cases, attempts to measure airway pressure after 5 tidal breaths had been delivered were not pursued out of concern for patient safety.

V_T was increased in twelve patients. An increased DP was observed in eleven of these patients; reduced C_{RS} was observed in eight. In response to the V_T increase, DP increased by a mean of 5.5 ± 5.7 cmH₂O in the supine position and 3.0 ± 1.3 cmH₂O in the prone position (P = 0.03 supine; P < 0.001 prone). C_{RS} deteriorated by a mean of 1.2 ± 4.1 mL/cmH₂O in the supine

position and 1.1 ± 2.8 mL/cmH₂O in the prone position (P = 0.44 supine; P = 0.32 prone) (Additional file 1 Table S1).

Holding V_T unchanged, PEEP was increased in fourteen patients; increased DP and reduced C_{RS} were observed in ten of these. In the remaining four, two had no change in DP and C_{RS}, and two had subtle improvements. The PEEP increment caused DP to increase by a mean of 3.4 ± 4.6 cmH₂O in the supine position and by 1.0 ± 1.2 cmH₂O in the prone position (P = 0.04 supine; P = 0.07 for prone). C_{RS} deteriorated by a mean of 2.2 ± 2.2 mL/cmH₂O in the supine position and 1.7 ± 2.4 mL/cmH₂O in the prone position (P = 0.01 supine; P = 0.11 prone) (Supplemental Table 1).



Discussion

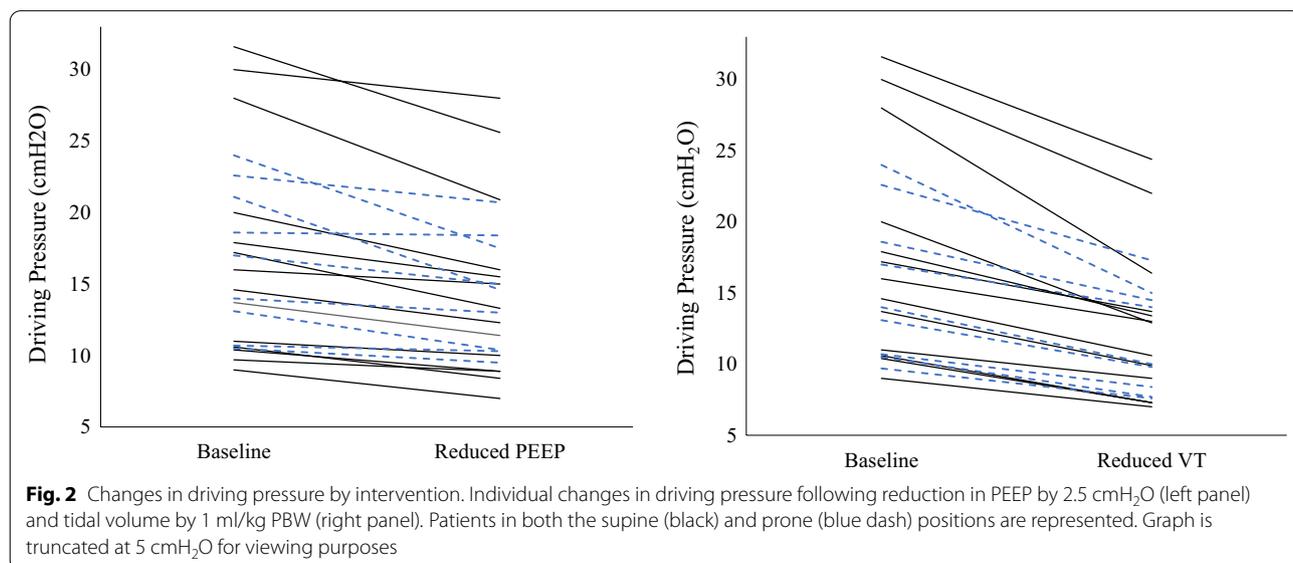
Among our cohort of patients with moderate-severe ARDS who demonstrated a paradoxical response to manual chest wall loading (17/19), small changes in tidal volume or PEEP exerted strong effects on C_{RS} and DP, despite the intent by their caregivers to follow ‘lung protective’ guidelines to the extent consistent with adequate gas exchange. The presence of mechanical paradox was associated with reduced C_{RS} at baseline; almost all (15/17) paradox positive patients had a baseline $C_{RS} < 40$ mL/cmH₂O, and the majority (11/17) had a baseline $C_{RS} \leq 30$ mL/cmH₂O. Assuming end-tidal overdistension as the most likely explanation for ‘paradox’, this finding is not unexpected. Because the low compliance

state of ARDS reflects primarily the reduced capacity of the ‘baby lung’, as opposed to altered elastic properties of remaining functional lung units [9], the risk of end-tidal overdistension would be expected to rise as compliance declines [10]. The severity of lung disease in our patient sample is also reflected by the CO₂ elimination data of Table 2 (PaCO₂ and ventilatory ratio).

Interestingly, the presence of paradox itself did not correlate well with duration of mechanical ventilation or hospitalization. While mean duration of hospitalization and of intubation were 16.8 and 11.2 days, respectively, at the time of data collection, over half (9/17) of our patients had been intubated for fewer than six days, and almost one third (5/17) had data collected within one day of intubation.

Consistent with our hypothesis that interventions alleviating end-tidal overdistension would lead to improved mechanics, there was universal improvement in C_{RS} following a minor decrease in either V_T or PEEP from the baseline value. Conversely, there was a clear trend toward increased DP and reduced C_{RS} following increases of either machine setting. Indeed, extreme rises in airway pressures in response to small increments of V_T or PEEP prevented data from being collected in several patients out of concern for safety; as a result, our data understate the adverse response in our patients with mechanical paradox to increasing either V_T or PEEP.

All patients in this study were receiving ventilation with low V_T and low to moderate levels of PEEP at baseline; mean V_T was 5.6 ± 0.9 mL/kg PBW and mean PEEP was 9.7 ± 3.2 cmH₂O. Nonetheless, decreasing V_T by 1 mL/kg PBW or PEEP by 2.5 cmH₂O resulted in a disproportionate reduction in DP and, therefore, improved C_{RS} .



These findings are consistent with those of prior studies demonstrating radiographic evidence of significant hyperinflation in as many as one third of patients treated with a ventilatory strategy targeting 6 mL/kg PBW V_T and $P_{plat} < 30$ cm H₂O [11]; they further demonstrate that no generalized ventilatory strategy, even those generally considered ‘lung-protective,’ can be employed indiscriminately without further concern regarding safety for the individual under care.

The paradoxical response to chest wall loading, in which C_{RS} unexpectedly improves following a decrease in chest wall volume, has been described in several recent reports. In these cases, C_{RS} improved not only in response to direct compression of the chest wall [3, 4, 6], but also in response to interventions that resulted in cephalad displacement of the diaphragm, including abdominal compression [2, 5]; compression of the lumbar region (while prone) [1]; and placement in a less upright position [2, 4, 5]. Considered collectively and in association with the data reported here regarding ventilatory pattern, the most plausible unifying explanation for mechanical paradox is that tidal ventilation infringes on the upper flat portion of the lung’s pressure volume inflation curve. Compression of the chest wall results in a forced volume reduction of lung units otherwise overdistended at end inspiration, leading to descent along the pressure–volume curve to a position more favorable to tidal excursions [12]. Limited data from studies that have used electrical impedance tomography and computed tomography with quantitative density analysis support this hypothesis, even though the precise mechanism remains unconfirmed [4, 6]. An alternative explanation may be that, in the setting of ARDS, heterogeneity gives rise to *unaltered* lung units that are buttressed by zones of inflammatory debris and edema; as a result, these fortified lung units may be exposed to high transpulmonary pressures without being subjected to injurious strain. In this scenario, volume reduction of such units may still lead to improved C_{RS} , but without the same implications regarding end-tidal overdistension of those embedded individual units.

Limitations

While these findings suggest end-tidal overdistension in patients with mechanical paradox, our study was not designed to be mechanism defining, but rather to focus selectively on the diagnostic value of detecting a paradoxical response to chest wall compression as it pertains to the ventilatory prescription. As such, we did not measure esophageal pressure for the purpose of partitioning C_{RS} into its individual components (C_{CW} and C_I). For safety concerns, changes in V_T or PEEP were sustained only for brief periods of time and, as noted, in several cases not

pursued when small increases were attempted. Although we observed no decreases in oxygen saturation during any loading maneuver or parameter change, the effects of altering the ventilatory prescription on gas exchange or hemodynamics could not be evaluated.

We did not perform interventions in a randomized sequence, primarily because we felt that doing so would compromise the consistency and efficiency of data collection; in each case, however, the duration of alterations was short lived, and the baseline was restored between interventions. As such, we think it is unlikely that randomizing V_T and PEEP would have had a significant effect on our findings. Our sample was also drawn primarily from patients affected by C-ARDS, many of whom had been intubated for over a week. Therefore, our results might differ quantitatively (but we suspect not qualitatively) from patients with other forms of severe ARDS. Finally, we emphasize that no conclusions can be drawn regarding the effect of decreasing V_T or PEEP on clinical outcomes of paradox positive patients on the basis of our findings.

Clinical implications

Repeated exposure to tidal cycles that cause excessive strain of lung parenchyma is believed to be a proximate stimulus for ventilator-induced lung injury in ARDS [13]. Once a strain threshold is exceeded and mechanical forces disrupt structural microelements, previously functioning lung units will begin to drop out, initiating a positive feedback cycle whereby inflation energy (and power) concentrate among fewer and fewer units [10]. Lung heterogeneity exacerbates this process further, leading to the amplification of stress at the interface of open and closed lung units [14]. Tidal volumes operating in the ‘upper inflection zone’ not only encourage damaging strain—both global and regional, but also risk barotrauma, regional small airway remodeling, and distortion of vulnerable lung units that are hyperinflated at end-inspiration. Such mechanical processes may help explain the highly regionalized emphysematous changes [15], noteworthy incidence of pneumothorax [16], and rapidly evolving bronchiectasis encountered in C-ARDS [17].

In our study, not only was mechanical paradox encountered in all but two of the nineteen patients with ARDS who met our inclusion criteria, but also it was present in spite of consistent adherence to ventilator settings widely regarded as lung-protective; it was frequently encountered early in the course of invasive mechanical ventilation; and the mechanics of all patients with paradox responded favorably to even small reductions in V_T and PEEP. The use of manual compression to detect paradox may thus serve as a valuable tool for revealing otherwise undetected excessive tidal strain, and its presence should

prompt re-evaluation of modifiable determinants of end-tidal overdistension, including PEEP, V_T , and positioning [18, 19]. In some patients with severe and unresolving ARDS, however, protection of the entire lung may simply be impossible without extracorporeal gas exchange as excessive end-tidal strain may be the unavoidable consequence of adequate ventilation.

Conclusions

A paradoxical response to chest wall loading is frequently observed in the setting of moderate to severe ARDS, particularly in the setting of low C_{RS} . Our data demonstrate that paradox can be present early in the course of mechanical ventilation and occur despite conservative application of V_T and PEEP. Paradox-positive patients demonstrate uniform improvement of C_{RS} following minor reduction in either V_T or PEEP, findings in keeping with prior evidence suggesting that paradox is a sign of tidal overdistension.

Abbreviations

ARDS: Acute respiratory distress syndrome; C-ARDS: Acute respiratory distress syndrome secondary to COVID-19; C_l : Lung compliance; C_{RS} : Respiratory system compliance; C_{CW} : Chest wall compliance; DP: Driving pressure; PEEP: Positive end-expiratory pressure; PBW: Predicted body weight; V_T : Tidal volume.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s13054-022-04073-2>.

Additional file 1: Table S1 Response to increased tidal volume and positive end-expiratory pressure V_T was increased by 1 mL/kg PBW in a total of twelve patients (baseline values for these twelve patients in column A); PEEP was increased by 2.5 cmH₂O in a total of fourteen patients (baseline values for these fourteen patients in column B). DP driving pressure, C_{RS} system compliance, V_T tidal volume, PEEP positive end-expiratory pressure.

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Not applicable.

Author contributions

JS, PT, DJD, JSh, LG and JJM contributed to study conception and design. JS and PT performed data acquisition. PSC and JS performed the statistical analysis. JS, PSC, DJD, LG, and JJM interpreted the data. JS, LG and JJM wrote the manuscript. All authors provided critical review of the manuscript and approved the final version.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or

comparable ethical standards. The study (A21-280) was approved by the HealthPartners Institutional Review Board. Written and verbal consent was obtained from legal surrogate decision makers in all cases prior to enrollment.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Not applicable.

Author details

¹Department of Pulmonary and Critical Care Medicine, University of Minnesota School of Medicine, 640 Jackson Street, Mail Stop 11203B, MinneapolisSt. Paul, MN 55101, USA. ²Department of Mathematics, Vanderbilt University, Nashville, TN, USA. ³Department of Surgery, Regions Hospital, St Paul, MN, USA. ⁴Department of Surgery, University of Minnesota School of Medicine, Minneapolis, MN, USA. ⁵Department of Pulmonary and Critical Care Medicine, Methodist Hospital, St. Louis Park, MN, USA. ⁶Department of Anesthesiology, Medical University of Göttingen, University Medical Center Göttingen, Göttingen, Germany. ⁷Department of Critical Care Medicine, Regions Hospital, St. Paul, MN, USA.

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