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Paradoxical Positioning: Does "Head Up" Always Improve Mechanics and Lung Protection?*

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OBJECTIVES: Head-elevated body positioning, a default clinical practice, predictably increases end-expiratory transpulmonary pressure and aerated lung volume. In acute respiratory distress syndrome (ARDS), however, the net effect of such vertical inclination on tidal mechanics depends upon whether lung recruitment or overdistension predominates. We hypothesized that in moderate to severe ARDS, bed inclination toward vertical unloads the chest wall but adversely affects overall respiratory system compliance (C_{RS}).

DESIGN: Prospective physiologic study.

SETTING: Two medical ICUs in the United States.

PATIENTS: Seventeen patients with ARDS, predominantly moderate to severe.

INTERVENTION: Patients were ventilated passively by volume control. We measured airway pressures at baseline (noninclined) and following bed inclination toward vertical by an additional 15°. At baseline and following inclination, we manually loaded the chest wall to determine if C_{RS} increased or paradoxically declined, suggestive of end-tidal overdistension.

MEASUREMENTS AND MAIN RESULTS: Inclination resulted in a higher plateau pressure (supineΔ: 2.8 ± 3.3 cm H_2O [$p = 0.01$]; proneΔ: 3.3 ± 2.5 cm H_2O [$p = 0.004$]), higher driving pressure (supineΔ: 2.9 ± 3.3 cm H_2O [$p = 0.01$]; proneΔ: 3.3 ± 2.8 cm H_2O [$p = 0.007$]), and lower C_{RS} (supine Δ: 3.4 ± 3.7 mL/cm H_2O [$p = 0.01$]; proneΔ: 3.1 ± 3.2 mL/cm H_2O [$p = 0.02$]). Following inclination, manual loading of the chest wall restored C_{RS} and driving pressure to baseline (preinclination) values.

CONCLUSIONS: In advanced ARDS, bed inclination toward vertical adversely affects C_{RS} and therefore affects the numerical values for plateau and driving tidal pressures commonly targeted in lung protective strategies. These changes are fully reversed with manual loading of the chest wall, suggestive of end-tidal overdistension in the upright position. Body inclination should be considered a modifiable determinant of transpulmonary pressure and lung protection, directionally similar to tidal volume and positive end-expiratory pressure.

KEY WORDS: acute respiratory distress syndrome; body position; chest wall loading; lung protection; mechanical ventilation

There is near-universal agreement among providers that head-upright positioning is beneficial for mechanically ventilated patients, and in most ICUs, a semirecumbent position (in which the head of bed is elevated 30–45°) is a standard of care, except when absolutely contraindicated. While this practice is driven in large part by published evidence that suggests its decreased incidence of ventilator-associated pneumonia (1), a physiologic rationale supporting head-upright positioning also exists: in more upright positions, the vector of abdominal weight shifts caudally (away from the diaphragm), increasing resting lung volume and

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KEY POINTS

- **Question:** In moderate to severe acute respiratory distress syndrome (ARDS), how does bed inclination toward vertical affect respiratory system mechanics and the tidal airway pressures targeted in lung-protective ventilation strategies?
- **Findings:** Unexpectedly, in such patients bed inclination towards vertical tends to decrease respiratory system compliance and increase tidal driving pressures. These changes are fully reversed with manual loading of the chest wall, suggesting end-tidal overdistension in the upright position as the underlying mechanism.
- **Meaning:** Body inclination should be considered a modifiable determinant of transpulmonary pressure and lung protection, similar to tidal volume and positive end-expiratory pressure.

reducing the tendency for basilar atelectasis to form (2, 3). In many patients, this caudal shift improves tidal compliance of the respiratory system as well (Crs) (4).

For ARDS, however, the benefits of upright positioning remain undecided. In this setting, diaphragmatic descent in the head-upright position increases transpulmonary pressure (P_L) at end-expiration, which tends to recruit additional lung units and improve gas exchange (5–8). However, low-capacity "baby lungs" of ARDS may operate near the uppermost segment of their pressure-volume curves when inflated by customary levels of positive end-expiratory pressure (PEEP) (9). Consequently, the overall effect of increased P_L will depend upon whether recruitment of additional lung units outweighs overdistension of those that are already open. In the intubated patient with severe and unresolving ARDS, this balance between recruitment and overdistension may progressively shift over time toward the latter, as adhesive atelectasis, consolidation, and fibrosis gradually replace edema and compression.

We, along with others, have observed that passively ventilated patients with advanced ARDS frequently demonstrate "paradoxical" improvement of Crs in response to manual compression, or loading, of the chest wall, in both supine and prone body postures (10–15). In such "responders," Crs reproducibly returns to its lower value immediately upon off-loading of the chest wall. We ascribed these unanticipated behaviors to end-tidal overdistension in response to greater P_L in the unloaded state (16). Because standard head-upright positioning also raises the P_L with respect to the fully recumbent (0°) position, we reasoned that more horizontal body positioning than customary might also improve Crs in advanced-stage ARDS and consequently lower the plateau pressure and driving pressure (DP) that clinicians currently target in lung protective strategies. The purpose of the present study was to evaluate the effects of upright positioning on the bedside respiratory mechanics of patients with moderate to severe ARDS; we secondarily sought to evaluate the effects of chest wall loading in the upright position. We tested the hypothesis that increasing bed inclination adversely influences plateau pressure (P_{plat}), DP, and Crs and that these changes are reversed with chest wall loading.

MATERIALS AND METHODS

This prospective, multicenter study was performed in two medical ICUs (Regions Hospital, St. Paul, MN and Methodist Hospital, Minneapolis, MN), with all data collected by the same investigative team between December 2021 and February 2022. The study was approved by the Institutional Review Board governing both hospitals (HealthPartners Research Foundation, study number A21-280, approval date December 27, 2021). Informed consent was obtained from the surrogate decision-maker prior to inclusion in every case. Procedures were followed in accordance with the ethical standards of the responsible institutional committee on human experimentation and with the Helsinki Declaration of 1975.

Patients

Only patients with ARDS, as defined by the Berlin consensus criteria, were enrolled and evaluated (17). All received invasive mechanical ventilation under controlled conditions, with passive breathing assured either by ongoing administration of neuromuscular blockers or deep sedation adequate to suppress all physical and monitored evidence of active breathing.

Ventilatory Strategy

All patients received mechanical ventilation in volume regulated, control mode (decelerating waveform)

TABLE 1.
Patient Characteristics

Subject	Duration of Hospitalization (d)	Duration of Intubation (d)	Pao_2/Fio_2	Pco_2 (mm Hg)	Set Tidal Volume (mL/kg Predicted Body Weight)	Set Positive End-Expiratory Pressure (cm H_2O)
1	9	1	85	50.8	7	16
2	22	14	137.5	101	4.9	12
3	1	1	197	37	6.0	10
4	26	23	83	81.3	5.4	6
5	15	13	87	62.6	3.9	6
6	14	14	157.2	65.3	5.9	9
7	11	11	245	63.4	5.5	12
8	50	41	82.2	63.2	5.9	5
9	1	2	86.9	61.5	5.4	8
10	1	1	147	48.1	7	14
11	11	2	191.4	47.4	5.9	12
12	10	1	160	74.3	6.6	10
13	14	4	98.3	49	4.6	10
14	22	14	102.5	45	5.2	8
15	2	2	104.1	36.6	5	12
16	1	1	65.6	59	4.6	12
17	13	6	185.5	58.7	6.7	12
Mean	13.1	8.9	130.3	59.1	5.6	10.2
SD	12.3	10.7	52.1	16.2	0.9	3

using one of two ventilators: Puritan Bennett 980 (Medtronic, Carlsbad, CA) or Maquet Servo-i (Siemens, Bloomfield, CT). Baseline measurements were performed using the tidal volume (Vt), set PEEP, and respiratory rate prescribed by the clinical team prior to study enrollment, which were left unchanged for its duration. Whenever consistent with acceptable gas exchange, clinical teams used lung protective Vts (**Table 1**).

Positioning

All patients were supported on identical critical care beds ("In Touch" Stryker, Kalamazoo, MI). Measurements were obtained in the positions (either prone or supine) used by the clinical team; except for inclination, these were not altered for the purposes of data collection. When possible, study measurements were repeated in the opposite position within 24 hours, provided that the criteria for passive breathing were still met. In supine patients, "baseline" measurements were obtained at a semi-recumbent angle with the head of bed elevated to 30° from horizontal and lower extremities parallel to the floor. In prone patients, baseline measurements were initially obtained horizontally (0°) with no elevation of either the head of bed or lower extremities. Following baseline measurements in either supine or prone position, the entire bed was "inclined" by tilting its plane relative to the floor (15° reverse Trendelenburg) without adjusting the existing angulation of the head of bed (**Fig. 1**). To minimize risk of excessive airway pressure or hemodynamic compromise, the inclined position was maintained only as long as needed for data collection.

Measurements

Baseline measurements were obtained as follows: Peak pressure (P_{peak}) was the highest airway pressure recorded during inflation; static airway pressure (P_{plat}) was measured at least two seconds after performing

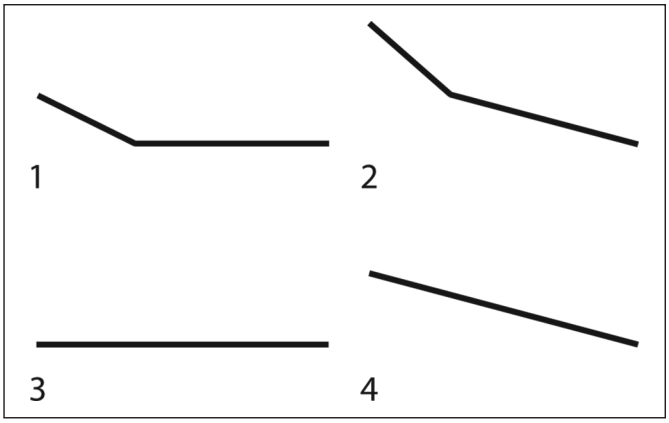

FIGURE 1. Bed orientation. 1) Supine baseline with head of bed 30°; 2) supine inclined with head of bed 30° and entire bed inclined 15°; 3) prone baseline with head of bed 0° and foot of bed 0°; 4) prone inclined with head of bed 0° and entire bed inclined 15°.

an end-inspiratory pause; and total PEEP ($PEEP_{tot}$, the sum of set PEEP and auto-PEEP), was measured at least three seconds after performing an end-expiratory pause, provided zero flow was achieved. The bed was then inclined by 15°, and all measurements were repeated after at least five breaths had been delivered. DP was calculated as the difference between P_{plat} and $PEEP_{tot}$. The Crs was calculated as the quotient of Vt and DP.

Chest Wall Loading

Manual loading of the chest wall was performed before and after bed inclination. 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 greater than or equal to 2 cm H_2O, at which point chest wall loading was considered sufficient to influence P_L 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

The paired *t* test was used to compare the values of P_{plat}, DP, and Crs in both the baseline and inclined positions, and before and after chest wall loading. Differences at the level of a two-tailed *p* value of less than 0.05 were considered statistically significant.

RESULTS

Seventeen patients with ARDS were studied. Paired measurements in both the supine and prone positions were obtained in seven; of the remaining 10: seven patients were evaluated only in the supine position, and three were evaluated only in the prone position. Chest wall loading was performed in all patients in the baseline position and in 14 patients while inclined. All patients had either severe or moderate ARDS, with the exception of one, and the majority had ARDS secondary to COVID (C-ARDS) (14/17). The mean age was 57.9 years (± 13.2 yr) and mean body mass index was 30.2 kg/m^2 (± 5.7 kg/m^2).

Inclination

Following inclination, P_{plat} and DP were significantly higher, and Crs was significantly lower, compared with baseline values (**Table 2**). These were consistent findings in both the supine position and the prone position (**Supplemental Figs. 1** and **2**, http://links.lww.com/CCM/H176). In the supine position, P_{plat} increased by a mean of 2.8 cm H_2O (± 3.3 cm H_2O; $p = 0.01$); DP increased by a mean of 2.9 cm H_2O (± 3.3 cm H_2O; $p = 0.01$); and Crs decreased by 3.4 mL/cm H_2O (± 3.7 mL/cm H_2O; $p = 0.01$) following inclination. In the prone position, P_{plat} increased by a mean of 3.3 cm H_2O (± 2.5 cm H_2O; $p = 0.004$); DP increased by a mean of 3.3 cm H_2O (± 2.8 cm H_2O; $p = 0.007$); and Crs decreased by a mean of 3.1 mL/cm H_2O (± 3.2 mL/cm H_2O; $p = 0.02$) following inclination.

Chest Wall Loading

Chest wall loading in both the baseline and inclined positions resulted in significant increases in Crs, regardless of whether it was performed in the prone or supine orientation (Table 2). In the noninclined baseline state, chest wall loading in the supine position decreased P_{plat} by a mean of 1.4 cm H_2O (± 2.3 cm H_2O; $p = 0.06$) and DP by a mean of 1.8 cm H_2O (± 2.1 cm H_2O;

TABLE 2.
Ventilatory Parameters

Measurements	A	B	C	D	A to B	A to C	C to D
Patients in the supine position							
P_{peak} (cm H_2O)	29.3 ± 5.2	28.7 ± 4.7	32.4 ± 7.3	27.2 ± 7.2	NS	[a]	[b]
P_{plat} (cm H_2O)	25.5 ± 5.1	24.0 ± 4.1	28.3 ± 7.7	24.1 ± 4.2	NS	[b]	NS
$PEEP_{tot}$ (cm H_2O)	11.6 ± 2.7	12.0 ± 3.1	11.6 ± 2.6	12.4 ± 4.2	NS	NS	NS
DP (cm H_2O)	13.9 ± 6.2	12.1 ± 5.5	16.7 ± 9	11.7 ± 6.4	[b]	[b]	[a]
Crs (mL/cm H_2O)	31.9 ± 10.2	36.2 ± 11	28.8 ± 12.3	39.6 ± 11.7	[b]	[b]	[a]
Patients in the prone position							
P_{peak} (cm H_2O)	31.3 ± 5.6	29.5 ± 4.8	34.7 ± 7.3	28.2 ± 4.7	NS	[a]	[b]
P_{plat} (cm H_2O)	27.7 ± 5.9	25.2 ± 4.8	31.0 ± 7.1	24.7 ± 5.6	[b]	[a]	[b]
$PEEP_{tot}$ (cm H_2O)	11.1 ± 3	11.3 ± 3.1	11.1 ± 3.2	10.2 ± 2.3	NS	NS	NS
DP (cm H_2O)	16.6 ± 6.6	13.9 ± 5.5	19.9 ± 8.4	14.5 ± 6.8	[b]	[a]	[b]
Crs (mL/cm H_2O)	25.4 ± 11.6	29.8 ± 12.5	22.2 ± 11.7	28.1 ± 13.6	[a]	[b]	[a]

Crs = compliance of the respiratory system, DP = driving pressure, NS = not significant, PEEPtot = total positive end-expiratory pressure, Ppeak = peak airway pressure, Pplat = plateau pressure.

[a] $p < 0.01$.

[b] $p < 0.05$.

Mean ± SD for: (A) ventilatory parameters at baseline; (B) at baseline with chest wall loading; (C) in the inclined position; (D) and in the inclined position with manual loading of the chest wall. Data obtained in supine orientation is in the dark gray rows; data obtained in prone orientation is in the light gray rows. Paired *t* test values for change in ventilatory parameters between baseline position and chest wall loading in baseline position (A to B); between baseline position and vertical (A to C); and between vertical position and chest wall loading in the vertical position (C to D).

$p = 0.02$); Crs increased by a mean of 4.3 mL/cm H_2O (± 5.5 mL/cm H_2O; $p = 0.03$). In the prone position, chest wall loading decreased P_{plat} by a mean of 2.5 cm H_2O (± 2.5 cm H_2O; $p = 0.02$) and DP by a mean of 2.7 cm H_2O (± 2.5 cm H_2O; $p = 0.01$). Crs increased by a mean of 4.4 mL/cm H_2O (± 3.3 mL/cm H_2O; $p = 0.004$).

When inclined, chest wall loading in the supine position decreased P_{plat} by a mean of 4.2 cm H_2O (± 5.5 cm H_2O; $p = 0.05$) and DP by a mean of 5.1 cm H_2O (± 4.9 cm H_2O; $p = 0.008$); Crs increased by a mean of 9.9 mL/cm H_2O (± 7.0 mL/cm H_2O; $p = 0.002$). In the prone position, chest wall loading decreased P_{plat} by a mean of 5.1 cm H_2O (± 3.8 cm H_2O; $p = 0.02$) and DP by a mean of 5.3 cm H_2O (± 4.0 cm H_2O; $p = 0.02$); Crs increased by a mean of 6.5 mL/cm H_2O (± 3.6 mL/cm H_2O; $p = 0.003$).

DISCUSSION

These data, collected in a cohort of patients with moderate to severe ARDS, primarily due to COVID, demonstrate that compliance of the respiratory system consistently decreased with bed inclination in both supine and prone orientations, thereby increasing both P_{plat} and DP for an unchanged Vt and PEEP. After inclination, Crs was restored to its baseline (less upright) value following manual loading of the chest wall. Our results regarding the "paradoxical" effects on tidal mechanics of chest wall loading in both supine and prone positions are consistent with our previous work (11, 15) but extend the principles of diagnostic chest wall loading into the practical domain of using bed and body angulation for its innate diagnostic value and potential for improving lung protection.

Loading the chest wall, either by direct application of pressure to the thoracic cage or by interventions that displace the diaphragm cephalad (e.g., abdominal compression), decreases local chest wall compliance. As the lungs and chest wall share a common volume, any decrease in chest wall compliance secondary to loading leads to a parallel decrease in Crs, unless there is a simultaneous and significant improvement in lung compliance, which is not generally encountered. As recently reported by several groups (10–15), however, stiffening the chest wall through the application

of external weight, manual pressure, or abdominal binding may "paradoxically" improve Crs in patients with advanced ARDS. Our current study indicates that using a less upright posture may confer a similar mechanical benefit.

Inclining the head of the bed, a default clinical practice, is a form of chest wall "unloading" that predictably increases P_L and lung volumes in all patients. Increased P_L in the upright position tends to recruit additional lung units while further distending those that are already open. The net result may either improve or deteriorate lung and system compliances, as well as their associated airway pressures, depending on whether recruitment or overdistension predominates. In the advanced stages of ARDS, increased P_L associated with upright positioning may fail to recruit additional lung units, resulting instead in end-tidal overdistension of the baby lung and decreased Crs. Loading the chest wall in this scenario, or decreasing bed angulation, could force volume reduction of open lung units, thereby placing them in a more favorable region of their individual pressure-volume curves and leading to paradoxical improvement in compliance of both the lungs and respiratory system (16).

Previous studies evaluating the effects of inclination in ARDS have observed a favorable effect on gas exchange, with variable effects on respiratory system mechanics (5–8). Recent studies, performed on passively ventilated patients with more severe ARDS, however, have consistently demonstrated reduced Crs and elevated airway pressures following placement in a more upright position (18, 19). We confirm, and extend those observations, by demonstrating that such changes respond favorably to sustained manual loading of the chest wall, strongly indicating that the underlying mechanism is end-tidal overdistension of an unloaded baby lung already distended by moderate PEEP.

These findings bear immediate clinical implications. Ventilator-induced lung injury is believed to result from excessively elevated P_L, stress, and strain. However, measurement of P_L is rarely performed in clinical practice, as it requires the skilled use of equipment not widely deployed (20). At the bedside, lung protective ventilatory strategies focus instead on using low-range Vt and avoiding excessive PEEP to prevent P_{plat} and DP from exceeding certain numerical thresholds (21). While the absolute differences we observed in response to bed angle and chest wall loading for P_{plat} and DP were moderate, such differences may be clinically relevant for patients who are approaching (or above) the numerical thresholds commonly targeted for P_{plat} and DP at the bedside (e.g., 30 cm H_2O and 15 cm H_2O, respectively). Whether supine or prone, body inclination should thus be considered a modifiable determinant of P_L in this population, similar to Vt and PEEP.

As the data reported here and in previous work indicate, chest wall loading is an informative diagnostic maneuver, and the presence of a paradoxical response should prompt reevaluation of both positioning and ventilatory prescription. However, while chest wall loading may be effective for modifying P_L even when sustained over hours (e.g., by abdominal binding, weighting, or by pressure-regulated mechanical devices), its effects on the regional distribution of ventilation and pulmonary blood flow, its optimal duration, and its ultimate clinical value or adverse consequences remain largely undefined.

Our study focused selectively on immediate changes in tidal mechanics of the passive respiratory system in late-stage ARDS and consequently has several clear limitations. The effects of bed inclination on gas exchange were not evaluated. For safety concerns, patients were held in the inclined position for only brief periods of time. Apart from ongoing electrocardiographic and blood pressure monitoring, the hemodynamic response to inclination was not systematically monitored; we did, however, note a clinically significant decrease in systemic blood pressure in multiple patients following inclination, which returned to baseline immediately once inclination was decreased. Any regional alterations of lung volume, ventilation, or perfusion were not evaluated. Additionally, we did not evaluate for the presence of complete airway closure (22), which can result in falsely elevated calculations of DP and Crs. However, we would expect that if complete airway closure was present to a significant degree in our study, the increased P_L associated with inclination would have exceeded critical opening pressure in at least some patients, resulting in improved measurements of respiratory mechanics in the upright position. Based on the near uniform decrease in Crs we observed following inclination, we think it unlikely that the presence of complete airway closure affected our overall findings. Finally, our sample was drawn primarily from patients affected by C-ARDS, many of

whom had been intubated for over a week, and might differ quantitatively (but we suspect not qualitatively) from patients with other forms of severe ARDS.

CONCLUSIONS

At the bedside, most clinicians attempt to restrain passive tidal pressures below defined numerical thresholds for the purposes of lung protection. Indeed, targets such as a P_{plat} less than 30 cm H_2O and DP less than 15 cm H_2O are currently encoded into many hospital and society guidelines. In severe ARDS, a more upright position "off-loads" the chest wall, adversely affecting respiratory mechanics and said numerical thresholds; these changes appear to be indicative of end-tidal overdistension and are fully reversed by chest wall loading. As such, body angulation should be considered a modifiable determinant of P_L, directionally similar to Vt and PEEP. When P_{plat} and DP exceed a desired threshold, paradoxical improvement in respiratory mechanics following manual loading of the chest wall should prompt reevaluation of positioning and the ventilator prescription.

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