BRIEF REPORT

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Dorsal Push and Abdominal Binding Improve Respiratory Compliance and Driving Pressure in Proned Coronavirus Disease 2019 Acute Respiratory Distress Syndrome

OBJECTIVES: We describe seven proned patients with coronavirus disease 2019-related acute respiratory distress syndrome in whom a paradoxical decrease in driving pressure reversibly occurred during passive, volume-controlled ventilation when compressing the lower back by a sustained "dorsal push." We offer a potential explanation for these unexpected observations and suggest the possible importance of eliciting this response for lung-protective ventilation of similar patients.

DESIGN/SETTING: Case series at a single teaching hospital affiliated with the University of Minnesota. Measurements were recorded from continuously monitored airway pressure and flow data.

PATIENTS: Nonconsecutive and nonrandomized sample of coronavirus disease 2019 acute respiratory distress syndrome patients who were already prone and paralyzed for optimized lung protective clinical management while inhaling pure oxygen.

INTERVENTIONS: Sustained, firm manual pressure applied over the lower back in all patients, followed by abdominal binding in a subset of these.

MEASUREMENTS AND MAIN RESULTS: Respiratory system driving pressure declined and respiratory system compliance improved in seven patients with the dorsal push maneuver. In a subset of four of these, abdominal binding sustained those improvements over >3 hours.

CONCLUSIONS: Sustained compressive force applied to the dorsum of the passive and prone patient with severe respiratory failure due to coronavirus disease pneumonia may elicit a paradoxical response characterized by improved compliance and for a given tidal volume, lower plateau, and driving pressures. Such findings, which suggest end-tidal overinflation within the aerated part of the diseased lung despite the already compressed anterior chest wall of prone positioning, complement and extend those observations recently described for the supine position in coronavirus disease 2019 acute respiratory distress syndrome.

KEY WORDS: abdominal pressure; acute respiratory distress syndrome; coronavirus disease 2019; dorsal pressure; paradoxical response; prone position

Supportive therapy for the acute respiratory distress syndrome (ARDS) prioritizes limiting further lung injury by using protective ventilation (1). To do so, clinicians often seek to constrain driving pressure (DP) and plateau pressures (Pplat), two values that for a given tidal volume (VT) are determined by compliance of the respiratory system (CRS). Because they are series-linked structures, both decreasing chest wall compliance and hyperinflating the lung normally reduce CRS.

Firas S. Elmufdi, MBBS¹ John J. Marini, MD²

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However, several recent reports of volume-controlled ventilation of supine patients with severe late-stage coronavirus disease 2019 ARDS (C-ARDS) describe the unexpected paradoxical response of decreased DP and improved CRs in response to sternal weighting or upper abdominal compression ("belly push") (2-4). We questioned whether compressive maneuvers might exert similar "paradoxical" effects on tidal mechanics in the already stiffened chest wall environment created by the prone position. Although abdominal suspension to "relieve" the abdominal compression that routinely occurs during prone positioning has previously been studied in ARDS (5-7), reported results are generally discouraging. To our knowledge, "increasing" the abdominal compression that characterizes the prone position has not been investigated-especially not in C-ARDS. We report here our experience with compressive maneuvers applied over the lower torso of proned C-ARDS patients.

METHODS AND RESULTS

We identified seven patients with severe C-ARDS (three females, 33 to 73 yr old: median age, 66 yr) managed directly by author (F.S.E.). **Table 1** summarizes patient demographics and ventilator settings at the time of data acquisition. Body mass index ranged

from 27 to 38 kg/m^2 (median, 31 kg/m^2). All patients were deeply sedated, paralyzed, and prone while inspiring pure oxygen and ventilated with "lung protective" VT (6 mL/kg or less). Because of illness severity and very low tidal compliance, Pplat could not be held below 30 cm H₂O without unduly compromising gas exchange, as evidenced by low pH and hypercapnia (Table 2). Initial PEEP values, set clinically by the ICU attending physician, were generally below those recommended in published ARDS network titration tables for non-COVID ARDS patients breathing pure oxygen. The specific reason was that attempts to try higher PEEP values than those in use at the time of our observations had failed because they did not improve oxygenation and invariably increased plateau and DPs further. Thus, lower back compression was undertaken after other routine options to safely improve ventilatory status and gas exchange had been attempted. Our compression procedures were performed during the course of routine clinical care intended to help guide management of each individual patient; our data were not collected as part of a research protocol. Approval to publish these data was obtained from the Institutional Review Board of Regions Hospital (Number A21-272). All patients received invasive support by Puritan Bennett 980 ventilators (Medtronic, Galway, Ireland) operated in assist control/volume control (AC/VC or AC/VC+) modes.

TABLE 1.

Patient's Initial Ventilator Settings at the Time of Obtaining Measurements

Patient No.	Age/ Gender (yr)	Body Mass Index (kg/m²)	Intervention	Pao₂/ Fio₂ ratio (mm Hg)	Respiratory Rate (Breaths per Minute)	Tidal Volume/ kg (mL/ kg)	Set Positive End- Expiratory Pressure (cm H ₂ O)	Peak Tidal Airway Pressure (cm H ₂ O)	End- Inspiratory Static (Plateau) Pressure (cm H ₂ O)
Case 1	50/male	37	Dorsal push	67	22	6	16	32	30
Case 2	73/male	27	Dorsal push	68	30	4	11	46	41
Case 3	58/male	29	Dorsal push then binder	101	34	5	15	41	35
Case 4	69/female	38	Dorsal push	101	33	5	16	36	33
Case 5	70/female	31	Dorsal push then binder	83	30	5.5	8	38	35
Case 6	66/male	29	Dorsal push then binder	50	32	4.5	7	34	31
Case 7	33/female	38	Dorsal push then binder	62	20	6	14	39	33

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TABLE 2.

Baseline Blood Gases, Ventilator Settings, and Changes in Tidal Volume With Dorsal Push

Patient No.	Auto Positive End- Expiratory Pressure ^a (cm H ₂ O)	Respiratory Rate (Breaths per Minute)	Vt (mL)	Vī/kg Ideal Body Weight mL/kg	рН	Paco ₂ (mm Hg)	Average Increase in VT in the First Tidal Cycle During Push ^b (Percentage Change From VT)	Average Decrease in VT in the First Tidal Cycle After Release ^b (Percentage Change From VT)
Case 1	0.0	22	510	6	7.37	44	269 mL (52%)	271 mL (53%)
Case 2	1.0	30	245	4	7.23	71		
Case 3	1.0	34	340	5	7.23	53	173 mL (51%)	100 mL (29%)
Case 4	0.0	33	280	5	7.30	77		
Case 5	0.8	30	220	5.5	7.15	78		
Case 6	1.3	32	340	4.5	7.30	75		
Case 7	0.0	20	360	6	7.32	91	100 mL (28%)	73 mL (20%)

 $V_T = tidal volume.$

^aAbove set positive end-expiratory pressure value.

^bData available in three patients. Average of 3 "dorsal push" maneuvers.

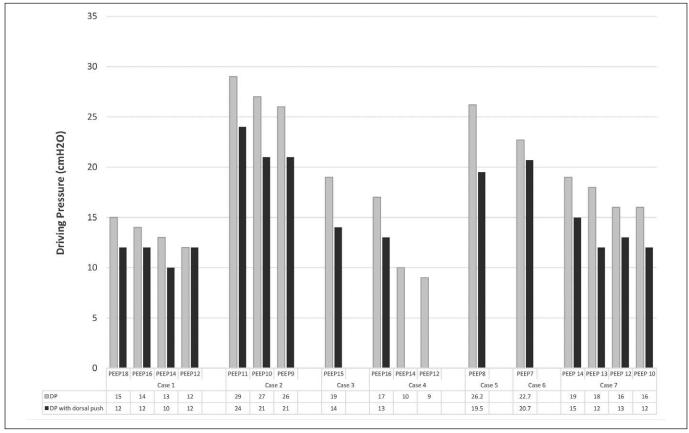


Figure 1. Driving pressure (DP) changes (cm H₂O) with dorsal push/abdominal binding, including changes at different positive endexpiratory pressure (PEEP) levels in cases 1, 2, 4, and 7. Note that both compression and PEEP reductions lowered DP, reflecting improved respiratory system compliance. Sustained external pressure that spanned both phases of each tidal cycle was applied by the author (F.S.E.) using a single extended arm and hand, manually compressing the lower back. At the onset of this maneuver, a drop in peak airway pressure was immediately visualized, despite an unchanged VT. Measurements were recorded after a minimum of three tidal cycles under external compression sustained through both phases of ventilation (**Fig. 1**).

Ventilator pressures returned to their initial values within one tidal cycle upon release of manual compression. Recorded auto-PEEP was universally minimal ($\leq 1.3 \text{ cm H}_2\text{O}$), and no significant changes of systemic blood pressure or heart rate were observed. In case number 6, bladder pressure was measured with a purpose-dedicated urinary catheter before and after binding the abdomen, changing from 4 to 14 cm H₂O. After cessation of these "dorsal push" diagnostic maneuvers, PEEP was titrated downward in four patients (numbers 1, 2, 4, and 7; Fig. 1), and DP decreased with each drop in PEEP, suggesting further release of lung overdistension.

In four patients, we followed the diagnostic maneuver by wrapping a binder (Comfor 9" width; Bird & Cronin, St. Paul, MN) around the abdomen. This wide elastic wrap was tightened until similar "paradoxical" ventilator pressure changes were elicited as had been observed during the preceding (diagnostic) manual maneuver. Binder position was then marked to ensure similar pressure was subsequently applied. In each patient, lower plateau and DPs persisted for the period the binder was in place, and arterial oxygen saturation monitored by pulse oximetry remained stable or marginally improved. As during the preceding dorsal push, no significant changes of systemic blood pressure, heart rate, or urine output were observed. The abdominal binder was released every 2-6 hours to prevent skin injury, per nursing staff discretion.

DISCUSSION

Our results replicate for the prone position the unexpected and potentially beneficial "paradoxical" improvements of DP and CRs previously observed when pressure is increased over the chest or abdomen in the supine patient with severe C-ARDS (2, 3) and demonstrate that these effects have potential to persist for hours if sustained by abdominal binding. Prone positioning reconfigures the lung and stiffens the anterior surfaces of the abdomen and chest. In most patients, modest increases occur in both intra-abdominal and esophageal pressures (4), but the changes in CRs, DP, and lung volume with proning have been variable (8–10). Our data show that in prone patients with C-ARDS, further loading of the chest wall by imposing a dorsal push and/ or binding the abdomen affects total respiratory mechanics by means other than simple stiffening of the chest wall.

Although several mechanisms may theoretically explain or contribute to these unexpected responses, perhaps the most likely is that the push and binding "reduced" the end-expiratory lung volume but eased end-inspiratory lung overdistension sufficiently to offset the reduction of chest wall compliance (9). This is supported in available recorded data from three patients by the increase in the exhaled VT measured by the ventilator in the first tidal cycle after applying dorsal push and decrease of the exhaled VT in the cycle immediately after release of the dorsal push (Table 2). Another influence may be amplification of proning's tendency to beneficially redistribute global transpulmonary pressures associated with VT delivery, moderating mechanical nonhomogeneity and in the process improving and stabilizing regional recruitment (10). Reducing occult gas trapping (not detected by the end-expiratory occlusion maneuver) is also conceivable, if less likely.

Interestingly, we detected no clear relationship between duration of symptoms and the emergence of the paradoxical response. The median number of days between onset of symptoms or positive COVID test to intubation was 14 (11-43 d), and the number of days between intubation and obtaining the measurements was 7 (0-8 d). On average, this differs from our prior experience with the supine belly push, in which the majority of push-responding patients with severe C-ARDS were in the late stage (2). We speculate that because the lungs of early phase C-ARDS patients are more gas-filled than at later stages, they, too, may be subject to end-inspiratory overdistension (10), the prime candidate to explain why tidal respiratory system compliance improves with maneuvers that diminish global lung volume (e.g., PEEP reduction).

The simple maneuvers we describe can be very quickly performed at any bedside without weights or the need for additional equipment. They produce, however, unknown and likely variable increases of pleural pressure and reductions of functional residual capacity. Yet, it is the "directional" rather than quantitative responses of plateau and DPs-increase or decrease-that are the key observations. Assuming controlled ventilation delivering unchanging VT, the DP change that occurs during a dorsal push would appear to be an attractive diagnostic indicator of endinspiratory overdistention. If pressure control were the mode in use, the directional change in VT should hold the same significance. We note here that most severely impaired C-ARDS patients we have subsequently encountered after this initial observational study have exhibited these same "paradoxical" responses to prone dorsal compression.

Although the "diagnostic" potential of brief, manual applications of external force is appealing, numerous questions and limitations remain regarding the present data: Our small sample size does not allow generalizability to patients with different body morphologies (e.g., massive obesity), positions, or illnesses; no specific protocol was used to regulate PEEP titration, and the lack of other measurements such as esophageal pressures, electric impedance tomography, or sequential CT imaging limits our ability to confirm or refute our proposed explanations. Furthermore, we emphasize that the "therapeutic" benefit of long-term chest wall restriction by abdominal binding also is not proven, and its impact on gas exchange remains unclear. Yet, in theory, well-monitored chest wall compression in "push-responsive" patients may allow safer use of higher PEEP required for oxygenation in some, as well as avoidance of tidal recruitment with less risk of regional overdistension. Indeed, binding may not be needed if other indicated measures are undertaken without impairing oxygenation (e.g., using less PEEP). Nonetheless, our experience suggests that abdominal binding in push-responsive patients may be physiologically well tolerated.

Further research is clearly needed to determine the occurrence rate of "paradoxical" response among intubated and passive patients with ARDS of varying severity and body habitus, as well as to better standardize the "dorsal push" maneuver itself.

CONCLUSIONS

Sustained compressive force applied to the dorsum of the passive and prone patient with severe respiratory failure due to COVID pneumonia and severe respiratory failure may elicit a paradoxical response characterized by improved compliance and lower plateau and DPs during volume-controlled ventilation. Such findings suggest but do not confirm an opportunity to further improve the distribution of ventilation and to moderate the risk of ventilator-induced lung injury by using less PEEP and/or reducing VT, even under what otherwise appear to be appropriate ventilatory conditions. Although intriguing, these initial observations are clearly exploratory and demand additional confirmation by carefully designed prospective studies. Furthermore, investigation should be directed toward longer-term benefits or consequences of abdominal binding in responders to the "dorsal push."

- 1 Pulmonary Medicine and Critical Care, Regions Hospital and University of Minnesota, St. Paul, MN.
- 2 Pulmonary Medicine and Critical Care, University of Minnesota and Regions Hospital, Minneapolis/St. Paul, MN.

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For information regarding this article, E-mail: marin002@umn. edu

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