Original Article

Evaluation of Intra-Cranial Pressure Changes by Measuring the Optic Nerve Sheath Diameter During The Lung Recruitment Maneuver in Patients with Acute Respiratory Distress Syndrome: A Prospective Study

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Abstract

Background: The lung recruitment maneuver (LRM) applied in acute respiratory distress syndrome (ARDS) may increase the intra-cranial pressure (ICP). **Aims:** This study evaluated the effect of LRM on intra-cranial pressure changes in patients with ARDS by measuring the optic nerve sheath diameter (ONSD). **Patients and Methods:** LRM was applied to patients undergoing follow-up for ARDS, with a positive pressure of 30 cmH_2O for 30 s. ONSD on ultra-sonography, dynamic lung compliance (C_{dyn}), oxygen saturation (S_{pO2}), and hemodynamic parameters were measured before (T0), immediately after (T1), and 10 min after (T2) LRM. The primary endpoint was the effect of LRM on ONSD changes. The secondary endpoints included the effect of LRM on C_{dyn}, S_{pO2} change, and relationship between C_{dyn} and ONSD changes. **Results:** The study included 60 patients. ONSD was higher at T1 than at T0 (median [interquartile range]: 5.13 [0.4] vs. 5.3 [0.3] mm, $P < 0.001$) but was similar at T0 and T2 (5.13 [0.4] vs. 5.09 [0.37] mm, $P = 0.36$). C_{dyn} and S_{pO2} were significantly higher at T1 and T2 than at T0 (C_{dyn}: 22.3 [5.8] vs. 23.7 [7.5] vs. 19.4 [6.6] mL/cmH_2O, $P < 0.001$; S_{pO2}: 90[2] vs. 92[4] vs. 88[4] %, $P = 0.013$). A significant correlation existed between C_{dyn} and ONSD changes, which increased at T2 compared to T0 ($P < 0.001$). **Conclusion:** LRM applied in ARDS causes a short-term increase in ONSD. However, C_{dyn} increases 10 min after LRM and causes ONSD, thereby leading to a decrease in ICP.

Keywords: *Acute respiratory distress syndrome, intra-cranial pressure, optic nerve, recruitment maneuver, ultra-sonography*

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Introduction

Acute respiratory distress syndrome (ARDS) is one of the most common reasons for admission to the intensive care unit. Trauma, severe burns, sepsis, and diseases such as pancreatitis and pneumonia may cause ARDS.[1] Mechanical ventilation has become inevitable to ensure adequate oxygenation in patients with ARDS. However, mechanical ventilation practices may also increase lung damage.[2] Therefore, the therapeutic goal of mechanical ventilation is shifting from maintaining normal gas exchange to preventing ventilator-related lung damage.[3] The end-expiratory lung volume can be increased and lung damage can be reduced by preventing atelectasis with positive end-expiratory pressure (PEEP) and lung recruitment maneuvers (LRMs).[4]

LRM refers to the dynamic process of re-opening unstable atelectatic alveoli via a voluntary transient increase in trans-pulmonary pressure. LRMs are performed using

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various procedures.[5] LRM usually involves efforts to open the atelectatic lung areas by applying a positive airway pressure of 30 cmH_2O for 30 s and 40 cmH_2O for 40 s.[6,7] LRMs have become an important part of "protective lung ventilation" strategies.[8] Although LRMs improve oxygenation and reduce mortality, especially in patients with ARDS, they have some negative side effects. The most important side effects are the risk of barotrauma after the application of positive airway pressure, increased intra-thoracic pressure, hemodynamic instability, and increased intra-cranial pressure (ICP) following impaired venous return.[9]

The method considered the gold standard for monitoring ICP changes is the direct ICP measurement using a catheter placed in the ventricle.[10] However, as it is an invasive procedure that may cause potential complications, its routine use only for ICP monitoring is not recommended.[11] Optic nerve sheath diameter (ONSD) measurement using ultra-sonography (USG) is a safe, fast, and promising ICP measurement method that entails fewer complications than do invasive methods, and studies have suggested that it has a direct correlation with invasive methods.[12,13]

The present study aimed to evaluate the effect of an LRM on ICP by measuring ONSD using USG in patients with ARDS having impaired oxygenation and who required mechanical ventilator support.

Patients and Methods

This prospective, quasi-experimental study was performed after obtaining approval from the local ethics committee (OMU/KAEK 2020/528) and in accordance with the guidelines of the Declaration of Helsinki. The protocol was registered at clinicaltrials.gov (NCT04935008). The study was conducted between July 1, 2021 and January 1, 2022. After obtaining written consent from the patients' representatives, patients aged over 18 years, who were receiving mechanical ventilator support at the intensive care unit, who were considered to have ARDS according to the Berlin criteria,[14] and who had oxygen saturation (S_{pO2}) ≤92% were included in the study. Patients with increased need for inotropic drugs in the last 2 h or a mean arterial pressure (MAP) lower than 65 mmHg; those with known intra-cranial hypertension, acute coronary syndrome, hypercapnia or known pneumothorax, subcutaneous emphysema, pneumomediastinum, or facial trauma; those whose ONSD could not be evaluated using USG; and those who used neuromuscular blocking agents (NMBAs) for severe ARDS were excluded from the study.

The patients who were receiving mechanical ventilator support owing to ARDS were monitored in a sedated state by administering an infusion of midazolam (0.05–0.3 mg/kg/h) and fentanyl (0.05–3 μg/kg/min). The Richmond Agitation Sedation Scale value was –2 or –3.

The patients' age, sex, body mass index, Acute Physiology and Chronic Health Evaluation II score, final Sequential Organ Failure Assessment score before LRM, additional systemic diseases, cause of ARDS, ARDS severity, mechanical ventilator support during LRM (mode), PEEP, airway peak pressure (Ppeak), and total fluid balance were recorded.

The patients were provided respiratory support using a GE Carescape R860 mechanical ventilator (GE Healthcare, Madison, WI, USA) in the pressure- or volume-controlled mode depending on a tidal volume (V_T) of 6–8 mL/kg, Ppeak <28 cmH_2O, a fraction of inspired oxygen <60%, and PEEP pressure and S_{pO2} values according to the protective mechanical ventilation strategy. LRM was applied to patients with S_{pO2} ≤92% in the 30-s continuous positive airway pressure mode with 30 cmH_2O positive pressure support and with the patient's head at 20–30° upward in the supine position. At the end of LRM, the patients were put back on mechanical ventilator support.

The changes in ONSD were recorded to evaluate the effect of LRM on ICP; the changes in the V_T, driving pressure (ΔP = Ppeak-PEEP), dynamic lung compliance (C_{dyn} = $V_T/\Delta P$), and S_{pO2} were recorded to evaluate their effect on lung mechanics, and the changes in MAP (mmHg) and heart rate (beat/min) were recorded to evaluate their effect on hemodynamics by performing measurements before LRM (T0), at the end of LRM (when the patients were put back on pre-LRM mechanical ventilator support) (T1), and 10 min after LRM (assuming that the effect of LRM on intra-thoracic pressure has ended) (T2).

For ONSD measurements, the patients' eyes were closed, and a thick conductive ultra-sound gel was applied after a sterile transparent cover was placed on the eyelids. Thereafter, a linear USG probe (GE Logiq V1, 8–13 MHz, General Electric Co., Jiangsu, China) was placed gently on the eyeball transversely, with the diameter on the horizontal plane and without applying pressure. The distance between the outer borders of the optic nerve and 3 mm posterior to the point where the optic nerve entered the eyeball was measured on the transverse plane [Figure 1]. To minimize observational variables, ONSD was measured three times in both eyes, and the mean value was considered the ONSD.

The ICP change (primary outcome) according to the changes in post-LRM ONSD, post-LRM C_{dyn}, and

post-LRM S_{pO2} and the relationship between C_{dyn} and ONSD changes (secondary outcome) were evaluated.

In a pilot study we conducted on 20 patients, ONSD was measured before and after LRM (mean ± standard deviation: 5.00 ± 0.175 vs. 5.08 ± 0.169), and the data were evaluated using G*Power (Version 3.1.9.4, Dusseldorf, Germany). The minimum number of patients was determined as 53 when the effect size was 0.46, the type 1 error was 0.05, and the test power was 0.90. Accounting for potential data loss, we decided to complete the study by including 60 patients.

Data were analyzed using IBM SPSS Statistics for Windows/Macintosh, Version 26.0 (IBM Corp., Armonk, NY, USA). Normal distribution analyses of the groups were performed using the Kolmogorov–Smirnov test, and sub-group analyses for sample sizes of 30 and below were performed using the Shapiro–Wilk test.

Continuous variables were expressed as mean ± standard deviation or median [interquartile range (IQR): 25th–75th percentiles], and nominal variables were expressed as frequencies and percentages.

The paired-samples t-test or Wilcoxon tests were applied depending on the results of the normality test for the variables measured before and after LRM. The severity of ARDS, which was categorized into three groups as mild, moderate, and severe depending on ONSD change, was evaluated using the Kruskal–Wallis test according to the normality test results.

The correlation between C_{dyn} and ONSD changes was evaluated using Spearman's correlation test according to the results of the normality tests, and P value < 0.05 was considered significant for all tests.

Results

LRM was applied to 81 patients during the study period. Among them, 12 patients were excluded as ONSD could not be evaluated because of suspected traumatic brain injury and intra-cranial hypertension, five were excluded as ONSD could not be evaluated because of facial trauma, and four were excluded as they were administered NMBAs for ARDS treatment. Finally, 60 patients were included in the study and analyzed [Figure 2]. The demographic data, systemic diseases, causes and severity of ARDS, the mechanical ventilation mode applied, pressure support, and total fluid balance of the patients included in the study are summarized in Table 1.

ONSD was higher at T1 than at T0 in 53 patients (88.3%), lower at T1 than at T0 in five patients (8.3%), and similar at T1 and T0 in two patients (3.3%). However, ONSD was higher at T2 than at T0 in 22 patients (36.7%), lower at T2 than at T0 in 31 patients (51.7%), and similar at T2 and T0 in six patients (11.7%). When all patients were evaluated, the mean ONSD at T1 was significantly higher than that at T0 (median [IQR]: 5.13 [0.4] vs. 5.3 [0.3] mm, $P < 0.001$). The mean ONSD at T2 was similar to that at T0 (median [IQR]: 5.13 [0.4] vs. 5.09 [0.37] mm, $P = 0.36$). C_{dyn} (mL/cmH_2O) was significantly higher at

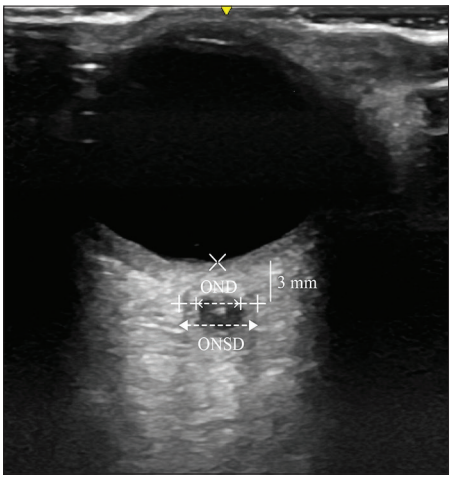

Figure 1: Measurement of the optic nerve sheath diameter by USG. ONSD, optic nerve sheath diameter; OND, optic nerve diameter

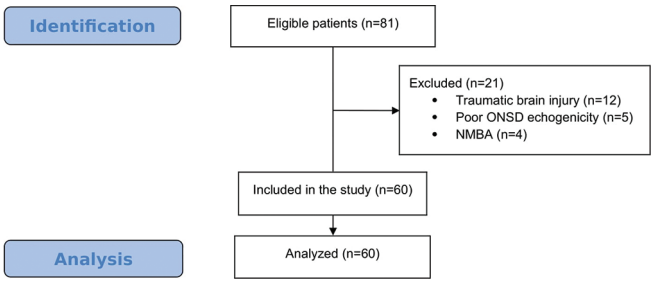

Figure 2: Flow diagram of patients' data distribution

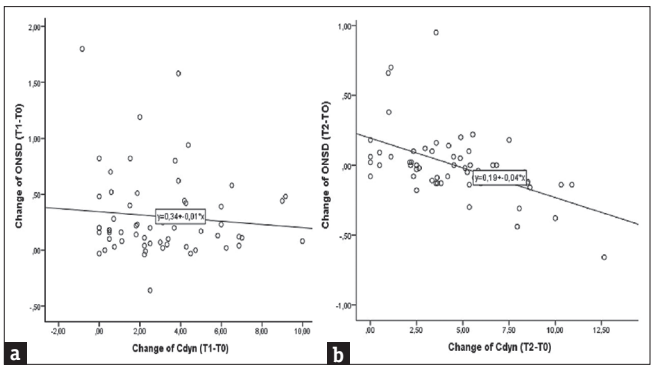

Figure 3: (a): Change of ONSD and Cdyn at the end of the recruitment maneuver, (b): Change of ONSD and Cdyn at 10 minute later of the recruitment maneuver; ONSD: optic nerve sheath diameter, Cdyn: dynamic compliance, T0: times start of recruitment maneuver, T1: end of recruitment maneuver, T2: recruitment maneuver 10 minute [T1-T0 (a), $P = 0.583$; T2-T0 (b), $P < 0.001$]

T1 (median [IQR]) (22.3 [5.8]) and T2 (23.7 [7.5]) than at T0 (19.4 [6.6], $P < 0.001$), and S_{pO2} (%) was significantly higher at T1 (median [IQR]) (90 [2]) and T2 (92 [4]) than at T0 (88 [4], $P = 0.013$). MAP (mmHg) decreased at the T1 (median [IQR]) (67.5 [14]) measurement time compared to the T0 (70 [12]) measurement time ($P < 0.001$). The T2 (71 [15.5]) measurement time was similar to the initial value ($P = 0.613$). ONSD measurements and respiratory and hemodynamic variables at T0, T1, and T2 are summarized in Table 2. No correlation was found between the change in ONSD (T0-T1) (mm) following LRM and the severity of ARDS (median [IQR]) (mild, 5.12 [0.35] vs. 5.23 [0.2]; moderate, 5.12 [0.56] vs. 5.32 [0.67]; severe, 5.22 [0.1] vs. 5.38 [0.14], $P = 0.138$). The C_{dyn} (T0-T1) (mL/cmH_2O) change after LRM was found to be associated with ARDS severity (median [IQR]) (mild, 19.1 [6.1] vs. 21.8 [7.6]; moderate, 21 [6.7] vs. 22.5 [3.7]; severe, 16 [1.7] vs. 18 [2.9], $P = 0.012$). However, a negative correlation was observed between the change in C_{dyn} and ONSD, which was higher at T1than at T0, even though not statistically significant. A statistically significant negative correlation was also observed between the change in C_{dyn} and ONSD, which was higher at T2 than at T0 [Figure 3a and 3b; $P = 0.583$ and $P < 0.001$].

Table 1: Demographic and clinical characteristics of patients

	Study Patients (n=60)
Age (years)	63.5 (25.5)
Sex (female) (%)	29 (48.3)
BMI (kg/m^2)	31±6.1
Apache II	26 (11.5)
SOFA	6.5 (3)
Systemic diseases (%)	
Diabetes mellitus	28 (46.7)
Hypertension	31 (51.7)
Coronary artery disease	13 (21.7)
COPD	10 (16.7)
Chronic renal failure	5 (8.3)
Cause of ARDS (%)	
Pneumonia	39 (65)
Non-pulmonary sepsis	14 (23.3)
Trauma	7 (11.7)
ARDS severity (%)	
Mild	22 (36.7)
Moderate	31 (51.7)
Severe	7 (11.7)
Mechanical ventilation mod (%)	
P-SIMV	48 (80)
V-SIMV	8 (13.3)
CPAP	4 (6.7)
PEEP (cmH_2O)	8 (2)
Ppeak (cmH_2O)	28 (5.7)
Fluid balance (mL)	3190±1551

APACHE II: Acute Physiology and Chronic Health Evaluation score, ARDS: acute respiratory distress syndrome, BMI: body mass index, COPD: chronic obstructive pulmonary disease, CPAP: continious positive airway pressure, PEEP: positive end expiratory pressure, Ppeak: peak pressure, P-SIMV: pressure synchronized intermittent mandatory ventilation, V-SIMV: volume synchronized intermittent mandatory ventilation

The patients included in the study did not develop pneumothorax, pneumomediastinum, subcutaneous emphysema, or any other pulmonary complications during LRM.

Discussion

The present study demonstrated that the LRM applied to patients with ARDS increased ONSD. However, this was a short-term effect. The S_{pO2} and C_{dyn} of the patients increased after LRM. At the 10th minute after LRM, the C_{dyn}, which increased when compared to the baseline value (thus decreased intra-thoracic pressure), resulted in a decrease in ONSD and ICP.

The question of whether the decrease in atelectatic areas and the application of short-term high PEEP, which improves oxygenation during LRM, causes an increase in ICP has not yet been answered definitively. Bein *et al.*[15] demonstrated that ICP increased after LRM applied to patients with ARDS and cerebral damage. Similarly, Nemer *et al.*[16] revealed that ICP increased after the LRM applied to patients with ARDS

Table 2: Recruitment maneuver and change of ONSD, respiratory, and hemodynamics

	T0	T1	*P*	T0	T2	*P*
ONSD (mm)	5.13 (0.4)	5.3 (0.34)	< 0.001	5.13 (0.4)	5.09 (0.37)	0.366
V_T (mL)	340 (77.5)	400 (90)	< 0.001	340 (77.5)	400 (83.7)	< 0.001
ΔP (cmH_2O)	18 (3)	18 (4)	0.005	18 (3)	16 (3)	< 0.001
C_{dyn} (mL/cmH_2O)	19.4 (6.6)	22.3 (5.8)	< 0.001	19.4 (6.6)	23.7 (7.5)	< 0.001
SpO_2 (%)	88 (4)	90 (2)	0.013	88 (4)	92 (4)	< 0.001
MAP (mmHg)	70 (12)	67.5 (14)	< 0.001	70 (12)	71 (15,5)	0.613
HR (pulse/min)	107 (17)	108 (18)	< 0.001	107 (17)	102 (14.2)	0.548

C_{dyn}: dynamic compliance, HR: heart rate, MAP: mean arterial pressure, ONSD: optic nerve sheath diameter, SpO_2: oxygen saturation, V_T: tidal volüme, ΔP: driving pressure, T0: start of the recruitment maneuver, T1: end of the recruitment maneuver, T2: recruitment maneuver 10 minute

and cerebral damage. However, other studies have demonstrated that LRM improves oxygenation without causing an increase in ICP.[17,18] Moreover, a few studies have examined the effect of the LRM applied to patients without intra-cranial pathologies on the increase in ICP by measuring ONSD using USG.[19] To our knowledge, no other study similar to ours has investigated the change in ICP following LRM in patients with ARDS without intra-cranial pathologies.

Cerebral perfusion pressure (CPP) is the net pressure gradient that supplies oxygen to brain tissues. It is the difference between the MAP, measured in millimeters of mercury, and ICP. Maintaining appropriate CPP is critical to the management of patients with intra-cranial pathologies, including traumatic brain injury, and hemodynamic instabilities, such as shock.[20] The LRM applied with high airway pressures increases intra-thoracic pressure and may cause a decrease in MAP and S_{pO2} as a result of impaired venous return. Increased ICP and decreased MAP during LRM negatively affect CPP. Simultaneously, the S_{pO2} that decreases as a result of the decreased MAP may lead to a decrease in oxygen supply to the brain. Li *et al.*[21] showed that the LRM applied during carotid endarterectomy reduces cerebral oxygen saturation and cerebral blood flow. Similarly, in the present study, at the end of the LRM applied with a positive pressure of 30 cmH_2O for 30 s, ICP, and consequently ONSD increased and MAP decreased compared to their pre-recruitment values. CPP was negatively affected as a result of increased ICP and decreased MAP. The mean ONSD measured 10 min after LRM was lower than the baseline values in 51.7% of the patients, even though the difference was not statistically significant. MAP, which decreased at the end of LRM, increased to the initial value 10 min after LRM. Decreased ONSD (decreased ICP) and increased MAP compared to the pre-LRM values suggested that the possible decrease in CPP at the end of LRM lost its effect 10 min after LRM. We think that the significant increase in the S_{pO2} value measured at the end of and 10 min after LRM compared to that at the baseline and the possible normalization of CPP at the 10^{th} minute after LRM increased cerebral oxygen supply.

The PEEP applied during LRM owing to lung heterogeneity in ARDS is associated with the hyper-inflation of open lung parts (i.e., "baby lung").[5] Hyper-inflation increases the risk of barotrauma and makes respiration difficult. It may also reduce cardiac pre-load and cardiac output. Therefore, the risk/benefit ratio of LRM in ARDS is debatable. Although LRM is recommended for selected patients because of potential risks in the guideline published by Papazian *et al.*[22] for ARDS management, the details regarding which patients can be treated using LRM remain unclear. Therefore, determining the factors predicting the LRM response in patients with ARDS is still necessary. Generally, the increase in lung compliance after opening the atelectatic areas indicates that patients would benefit from LRM.[23] However, which patients may experience hyper-inflation because of LRM and the consequences of hyper-inflation are unclear. ARDS severity is also associated with lung heterogeneity.[24] Therefore, the LRM applied to patients with severe ARDS may cause hyper-inflation and a further increase in intra-thoracic pressure, leading to an increase in ICP. In the present study, no association was observed between the increased ONSD following the LRM and ARDS severity or C_{dyn} before LRM. This result shows that the suspicion that the LRM applied to patients with severe ARDS or low lung compliance may cause more severe lung hyper-inflation is incorrect. To our knowledge, the changes in C_{dyn}, ONSD, and ICP following the LRM applied to patients with ARDS have not been investigated in previous studies. In the present study, a negative correlation was observed between increased C_{dyn} and decreased ONSD 10 min after LRM. Although ONSD increased at the end of LRM, the restored C_{dyn} would decrease intra-thoracic pressure and consequently lead to a decrease in ONSD and ICP, thereby contributing to the improvement in cerebral perfusion.

Most of the studies investigating ONSD and ICP increase compared ONSD with ICP measured using a different method. No study has investigated the correlation between the extent of increase in ONSD and the increase in ICP. Therefore, we are unsure to what extent ONSD, which increased in the present study, caused an increase in ICP. CPP at the end of LRM decreased because of both the increase in ONSD and the decrease in MAP at T1.

The limitations of our study are that changes in cardiac output and central venous pressure, essential for perfusion, were not evaluated in the present study. Second, despite the potential change in perfusion pressure, we could not calculate the level of difference in oxygen supply because of the increase in S_{pO2}. In addition, the clinical consequences of ONSD change after LRM were not evaluated.

The LRM applied to patients with ARDS caused an increase in short-term ONSD and a decrease in MAP. LRM could be considered to affect brain perfusion negatively because of the increased ONSD and consequently increased ICP and decreased MAP. However, the effect of LRM on brain perfusion and oxygen supply should be investigated in larger patient populations because of ONSD and MAP, which return to normal values within a short period, and the increased S_{pO2} after the LRM.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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