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Original Article

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Right Atrial Strain in Preterm Infants With a History of Bronchopulmonary Dysplasia

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The authors have no financial conflicts of interest.

ABSTRACT

BACKGROUND: Few studies have utilized right atrial (RA) strain to evaluate right ventricular (RV) diastolic dysfunction in preterm infants with bronchopulmonary dysplasia (BPD). We aimed to evaluate the associations of RA strain with BPD severity and respiratory outcomes in preterm infants with BPD.
METHODS: We retrospectively studied 153 infants with BPD born before 32 weeks of gestational age at CHA Bundang Medical Center. Peak longitudinal right atrial strain (PLRAS) was obtained using velocity vector imaging and compared among infants across BPD severity. Conventional echocardiographic parameters and clinical characteristics were also evaluated.
RESULTS: In infants with severe BPD, mean gestational age (27.4 ± 2.1 weeks) and mean birth weight (971.3 ± 305.8 g) were significantly smaller than in those with mild BPD (30.0 ± 0.9 weeks, 1,237.3 ± 132.2 g) and moderate BPD (29.6 ± 1.3 weeks, 1,203.2 ± 214.4 g). PLRAS was significantly lower in infants with severe BPD (26.3 ± 10.1%) than in those in the moderate BPD group (32.4 ± 10.9%) or mild BPD group (31.9 ± 8.3%). Tricuspid E/e′ and maximum RA volume index were similar across BPD severity. A decrease in PLRAS was significantly correlated with increased duration of mechanical ventilation duration; however, tricuspid E/e′ and maximum RA volume index were not.
CONCLUSIONS: Evaluating PLRAS with other parameters in infants with BPD might detect RV diastolic dysfunction. Longer follow-up and larger study populations may elucidate the association between PLRAS and respiratory outcomes in infants with BPD.

Keywords: Atrial function, right; Bronchopulmonary dysplasia; Ventilators, mechanical

INTRODUCTION

Preterm infants born under 32 weeks' gestational age are exposed to stressors, such as prolonged mechanical ventilation and oxygen therapy from birth, which hinder normal lung and heart development.[1)2)] As a result of these stressors, preterm infants are prone to bronchopulmonary dysplasia (BPD), a condition of arrested development of lung.[1)2)] In infants with BPD, hypoxia from structural and functional changes of lung could lead to elevation of pulmonary vascular resistance and altered right ventricular (RV) function and, in extreme cases, can progress to pulmonary hypertension.[3)]

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Author Contributions

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Apart from the detection of pulmonary hypertension in evaluating the association between BPD and respiratory outcomes,[4] recently, increasing number of studies have evaluated RV dysfunction in association with BPD severity.[3][5][6] However, these studies have mostly focused on evaluating RV systolic dysfunction, and mostly utilized conventional and tissue Doppler imaging, which is limited by angle dependency, and traction and translation from adjacent tissue.[7] Moreover, in infants with BPD who have isolated RV diastolic dysfunction and not RV systolic dysfunction, more sensitive echocardiographic methods besides the measurement of systolic RV dysfunction would be necessary to detect and treat abnormalities in RV function in these infants.

Atrial reservoir function has been associated with end-diastolic filling pressure in adults.[8] In addition, right atrial (RA) deformation analysis has been utilized to evaluate RV dysfunction in postoperative tetralogy of Fallot patients.[9] However, to date, studies evaluating RA function in preterm infants with BPD has been limited. We aimed to evaluate RA strain in preterm infants with BPD across BPD severity and evaluate associations between RA strain and clinical outcomes.

METHODS

This retrospective study was performed at CHA Bundang Medical Center. Infants born before 32 weeks' gestational age and hospitalized at the neonatal intensive care unit of CHA Bundang Medical Center from 2016–2018 were studied. BPD severity was assessed at 36 weeks gestational age through a review of medical records, by an investigator who was blinded to the echocardiography findings.[5]

Ethical approval

All procedures involving human participants in this study were performed in accordance with the ethical standards of the Institutional Review Board of CHA University Bundang Medical Center and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The study protocol was approved by the IRB of the institution, and patient consent for our study was waivered due to its retrospective nature.

Clinical characteristics of infants with BPD

A preterm infant is said to have BPD when treated with oxygen > 21% for at least 28 days since birth. After a preterm infant is initially treated with > 21% oxygen for at least 28 days after being born, BPD is classified according to the amount of additional respiratory support required at either 36 weeks postmenstrual age, or at the time of discharge: 1) mild BPD, when requiring only room air; 2) moderate BPD, when needing less than 30% oxygen; and 3) severe BPD, when more than 30% oxygen and/or positive pressure ventilation is needed.[10]

Infants with chromosomal anomalies or congenital heart defects, except patent foramen ovale and patent ductus arteriosus (PDA), were excluded from our study. An infant was determined to have a hemodynamically significant PDA when a PDA greater than 2 mm was detected before 3 days postnatally, and when this resulted in any of the following; pulmonary hemorrhage, respiratory failure, or hypotension, as described previously.[11] The proportion of infants who developed grade III or higher intraventricular hemorrhage (IVH)[12] and/or grade IIA or higher necrotizing enterocolitis (NEC) by the modified Bell's staging criteria,[13] as well as the proportion of infants with sepsis, were evaluated across the BPD severity.[11]

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Echocardiography images were obtained using Siemens Acuson ultrasound equipment (ACUSON SC2000; Siemens Medical, Mountain View, CA, USA). Both conventional and deformation imaging analysis were done by investigators blinded to the BPD severity allocation of the infants. All echocardiography images were obtained at 40 weeks corrected age to assess biventricular function.

Conventional echocardiography

To estimate RV diastolic function, tricuspid E/e′ was obtained, as described previously.[14] Tricuspid regurgitation jet velocity (TRJV) and pulmonary artery acceleration time (PAAT) was measured as previously described,[15] and mean pulmonary artery pressure (MPAP) was estimated by the equation[15]:

$$MPAP = 48 - 0.28 \times PAAT$$

We utilized PAAT rather than TRJV in estimating pulmonary artery pressure, since tricuspid regurgitation jet has been reported to be not always obtainable even in the presence of pulmonary hypertension in neonates,[16] and this may lead to underestimation of pulmonary hypertension in BPD infants when relying on tricuspid regurgitation jet alone. Normal values for PAAT in preterm infants have been published, and PAAT less than 47 msec have been reported to be a reliable method of detecting pulmonary hypertension in preterm infants at 36 weeks postmenstrual age.[17] Left ventricular systolic function was estimated by measuring left ventricular ejection fraction. An infant was considered to have pulmonary hypertension when one or more of the following were present: 1) PAAT less than 47 msec, 2) evidence of bidirectional or right-to-left shunt, or interventricular septal flattening.[4][17]

Deformation imaging analysis

Deformation imaging analysis of infants with BPD in mild, moderate, and severe BPD groups was performed on prerecorded echo images obtained at 70 frames per second. Peak longitudinal right atrial strain (PLRAS) and RV longitudinal peak systolic strain was obtained by using vector velocity imaging as previously described.[18][19] Briefly, in the apical 4-chamber view, the endocardial borders of the RA and RV were manually traced and subsequently tracked using the velocity vector imaging software. PLRAS values of the lateral free wall, interatrial septum, and the roof of the RA were measured at end-systole and averaged. Measurements were all done in triplicate. Also at end systole, at the apical four-chamber view, RA volumes were measured by the area-length method,[14] and subsequently indexed to the body surface area. RV longitudinal peak systolic strain was measured at the onset of the QRS wave, as previously described.[18]

Statistical analysis

Continuous data were presented as mean ± standard deviation, while categorical data were presented as counts(percentages), as appropriate. SAS 9.4 was used to analyze all data. Clinical and echocardiographic data were compared across BPD severity using the ANOVA test or Kruskal-Wallis test, for normally distributed and non-normally distributed variables. The χ^2 analysis was used to analyze categorical data. A p-value less than 0.05 was considered statistically significant for all analyses. Pearson's correlation was used to analyze the correlation between PLRAS and clinical and echocardiographic parameters. We have previously reported the intraobserver and interobserver variability of peak RV longitudinal strain in preterm infants to be 20% and 21%, respectively.[19] The mean percentage error[20] was calculated to obtain the intraobserver and interobserver variabilities of PLRAS, as described

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previously.[19] One investigator repeated deformation analysis of PLRAS 4 weeks apart on 30 infants to evaluate intraobserver variability. Two investigators who were blinded to BPD severity status and clinical information performed deformation analysis of PLRAS separately on 30 infants to determine interobserver variability.

RESULTS

The study population comprised 153 infants with BPD. The mean gestational age and mean birth weight were significantly lower in the severe BPD group than in the other two groups; however, the mean gestational age and mean birth weight were similar between moderate and mild BPD group (**Table 1**). Among the severe BPD group, 77.6% received antenatal steroids. Among the infants with severe BPD, 19 were dependent on oxygen, and 8 (16.3%) had pulmonary hypertension at 40 weeks' corrected age.

The proportion of infants with a history of hemodynamically significant PDA, IVH, and NEC was significantly higher in the severe BPD group than the moderate BPD or mild BPD groups. The duration of mechanical ventilation and the duration of oxygen support was longest in the severe BPD group and decreased according to decreased BPD severity.

Conventional echocardiography

At the time of echocardiography at 40 weeks' gestational age, there were no hemodynamically significant PDA in all infants. PAAT, and consequently, MPAP derived from PAAT, was significantly higher in the severe BPD group than in the mild BPD group; however, PAAT and MPAP did not differ significantly between the severe and moderate BPD groups, and between the moderate and mild BPD groups (**Table 2**).

The proportion of infants whose PAAT < 47 msec was significantly higher in the severe BPD group than the moderate BPD or mild BPD groups.

Table 1. Demographic and clinical characteristics of the study population

Characteristics	BPD severity			
	Mild (n = 60)	Moderate (n = 44)	Severe (n = 49)	p-value
GA (weeks)	30.0 ± 0.9	29.6 ± 1.3	27.4 ± 2.1	< 0.001*, < 0.001†, 0.15‡
BW (g)	1,237.3 ± 132.2	1,203.2 ± 214.4	971.3 ± 305.8	< 0.001*, < 0.001†, 0.81‡
BW < 1,000 g	1 (1.7)	9 (20.5)	35 (71.4)	< 0.001
Male	28 (46.7)	21 (47.7)	28 (57.1)	0.51
Antenatal steroids	16 (26.7)	19 (43.2)	38 (77.6)	< 0.001
5-min APGAR	7 (4–9)	7 (4–9)	6 (2–8)	< 0.001*, < 0.001†, 0.9451‡
hsPDA	11 (18.3)	18 (40.9)	25 (51.0)	0.001
NEC	0 (0)	3 (6.8)	18 (36.7)	< 0.001
IVH	0 (0)	3 (6.8)	13 (26.5)	< 0.001
Sepsis	5 (8.3)	17 (38.6)	36 (73.5)	< 0.001
Weight at echocardiography (kg)	3.52 ± 0.44	3.54 ± 0.76	3.24 ± 0.61	0.1383*, 0.0013†, 0.9779‡
Age at echocardiography (months)	2.46 ± 0.26	2.58 ± 0.32	3.12 ± 0.53	< 0.001*, < 0.001†, 0.1387‡
Duration of mechanical ventilation (days)	13.9 ± 4.4	35.3 ± 7.0	64.3 ± 21.5	< 0.001*, < 0.001†, < 0.001‡
Duration of oxygen support (days)	37.3 ± 5.3	49.8 ± 7.5	83.0 ± 24.8	< 0.001*, < 0.001†, < 0.001‡

Values are presented in means ± standard deviations, number (%) or median (range).
5-min APGAR: APGAR score at 5 minutes, BPD: bronchopulmonary dysplasia, BW: birth weight, GA: gestational age, hsPDA: hemodynamically significant patent ductus arteriosus, IVH: intraventricular hemorrhage, NEC: necrotizing enterocolitis.
*Comparison between severe BPD and moderate BPD; †Comparison between severe BPD and mild BPD; ‡Comparison between moderate BPD and mild BPD.

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Table 2. Echocardiographic parameters of infants with BPD

Parameters	BPD severity			
	Mild (n = 60)	Moderate (n = 44)	Severe (n = 49)	p-value
SBP (mmHg)	76 ± 6	76 ± 7	75 ± 6	0.9996*, 0.9059†, 0.8682‡
DBP (mmHg)	43 ± 5	42 ± 6	42 ± 5	0.9948*, 0.7443†, 0.6649‡
Heart rate (bpm)	134 ± 22	135 ± 17	136 ± 23	0.9629*, 0.9558†, 0.9999‡
PAAT (msec)	65 ± 9	61 ± 10	59 ± 14	0.7898*, 0.0248†, 0.0930‡
PAAT < 47 msec	0 (0)	1 (2.3)	8 (16.3)	0.0007
MPAP (mmHg)	30 ± 3	31 ± 3	32 ± 4	0.7898*, 0.0248†, 0.0930‡
TRJV (m/sec)	1.5 ± 0.4	1.8 ± 0.5	2.0 ± 0.8	0.2904*, 0.0035†, 0.0187‡
Tricuspid E/e′	6.3 ± 2.1	6.4 ± 2.5	6.8 ± 2.8	0.8095*, 0.7614†, 0.9670‡
Maximum RA volume indexed to body surface area (mL/m^2)	25.8 ± 9.4	30.6 ± 13.3	29.2 ± 10.3	0.9967*, 0.1951†, 0.1643‡
PLRAS (%)	31.9 ± 8.3	32.4 ± 10.9	26.3 ± 10.1	0.0192*, 0.0051†, 0.9946‡
RVPLS (%)	18.7 ± 2.5	18.7 ± 3.3	15.7 ± 4.2	0.0008*, 0.0001†, 0.8984‡
LVEF (%)	67.1 ± 7.6	64.7 ± 9.3	62.7 ± 10.6	0.6449*, 0.0766†, 0.3556‡

Values are presented in means ± standard deviations or number (%). Strain values are presented in absolute values.
BPD: bronchopulmonary dysplasia, DBP: diastolic blood pressure, bpm: beats per minute, LVEF: left ventricular ejection fraction, MPAP: mean pulmonary artery pressure, PAAT: pulmonary artery acceleration time, RA: right atrium, PLRAS: peak longitudinal right atrial strain, RVPLS: right ventricular peak longitudinal strain, SBP: systolic blood pressure, TRJV: tricuspid regurgitation jet velocity.
*Comparison between severe BPD and moderate BPD; †Comparison between severe BPD and mild BPD; ‡Comparison between moderate BPD and mild BPD.

Also, tricuspid E/e′ was similar between all BPD groups. TRJV were similar in infants with severe and moderate BPD; however, TRJV in both groups were significantly higher than those of infants with mild BPD. Maximum RA volume index and left ventricular ejection fraction did not differ significantly across the severity of BPD.

Deformation imaging analysis

PLRAS was significantly lower in the severe BPD group than in the moderate BPD or mild BPD group (**Figure 1**). PLRAS in infants with severe BPD was also decreased, compared to the reported mean value of global RA strain in normal infants under 1 year of age estimated from the sum of global negative and positive RA strain, which was estimated to be approximately 30%.[20] Also, RV peak longitudinal strain was significantly lower in the severe BPD group than in the moderate BPD or mild BPD groups. However, there was no statistically significant difference in PLRAS and RV peak longitudinal strain between infants in the moderate BPD group and those in the mild BPD group.

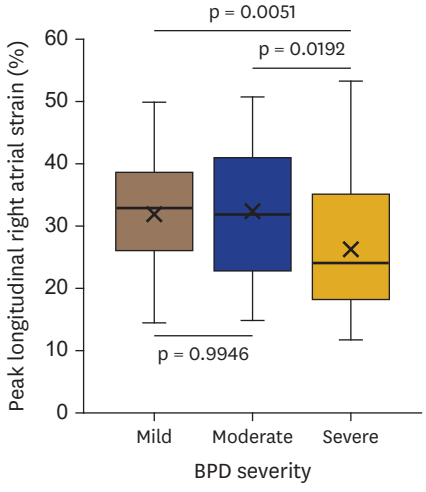

Figure 1. Peak longitudinal right atrial strain in infants with BPD.
BPD: bronchopulmonary dysplasia; line inside the box: median (50% percentile); 'X' inside the box: mean.

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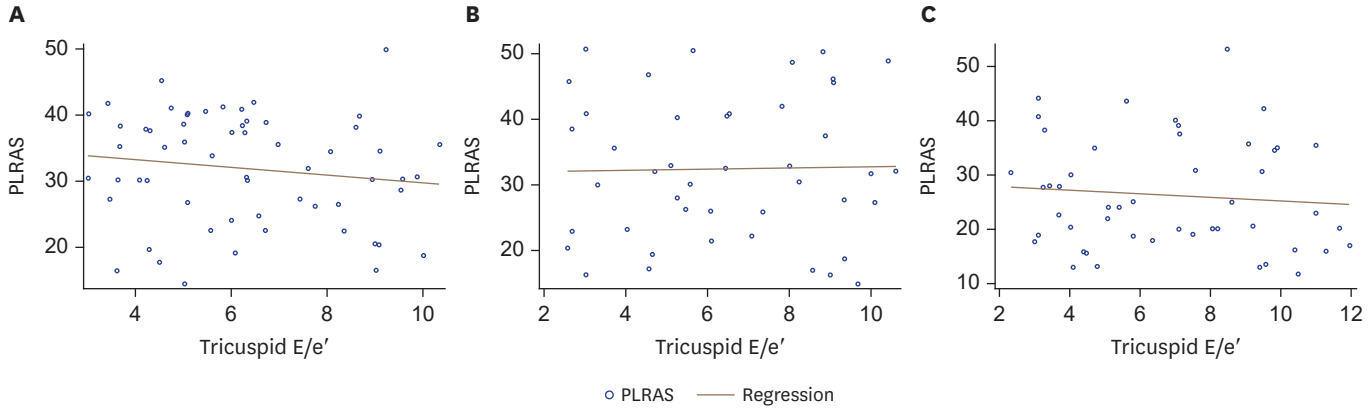

Figure 2. Scatterplot of tricuspid E/e′ and PLRAS across BPD severity. (A) Mild BPD, (B) moderate BPD, and (C) severe BPD groups.
X-axis represent tricuspid E/e′, y-axis represent PLRAS.
BPD: bronchopulmonary dysplasia, PLRAS: peak longitudinal right atrial strain.

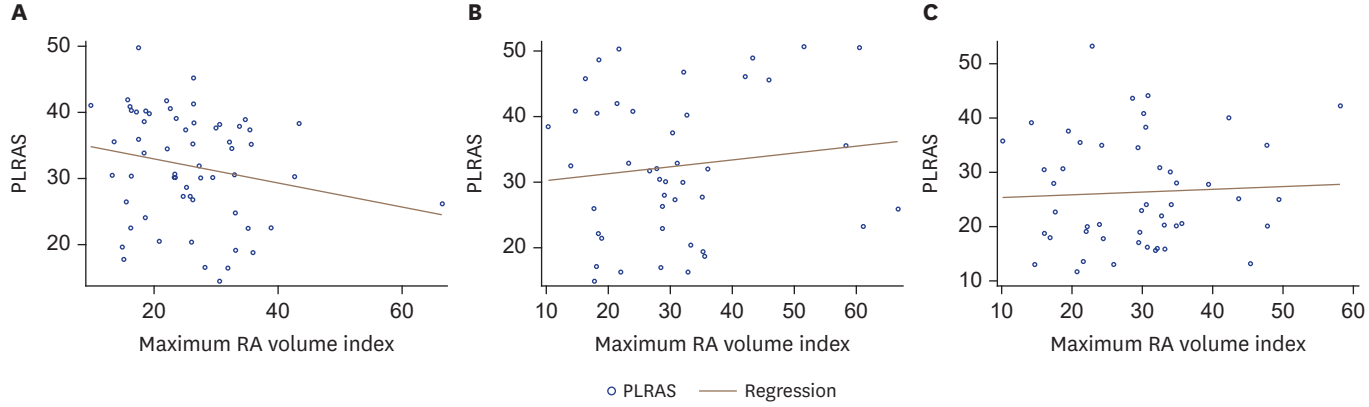

Figure 3. Scatterplot of maximum right atrial volume indexed to BSA (maximum RA volume index) and PLRAS across BPD severity. (A) Mild BPD, (B) moderate BPD, and (C) severe BPD groups.
X-axis represent maximum RA volume index, y-axis represent PLRAS.
BPD: bronchopulmonary dysplasia, BSA: body surface area, PLRAS: peak longitudinal right atrial strain.

Association between PLRAS and conventional RV diastolic parameters

There was no significant correlation between PLRAS and tricuspid E/e′, and between PLRAS and maximum RA volume index across BPD severity (**Figures 2** and **3**).

Association between PLRAS and respiratory outcomes

PLRAS was significantly correlated with gestational age, birth weight, antenatal steroid use, mechanical ventilation duration and oxygen support duration. A decrease in PLRAS was significantly correlated with increased duration of mechanical ventilation duration; however, tricuspid E/e′ and maximum RA volume index were not significantly correlated with the mechanical ventilation duration (**Table 3**).

Table 3. Correlation of PLRAS and conventional echocardiographic parameters of RV diastolic function with duration of mechanical ventilation of infants with BPD

Echocardiographic parameters of RV diastolic function	Correlation coefficient (r)	p-value
PLRAS	−0.45625	< 0.001
Tricuspid E/e′	0.13318	0.1008
Maximum RA volume indexed to body surface area	0.08820	0.2783

BPD: bronchopulmonary dysplasia, PAAT: pulmonary artery acceleration time, PLRAS: peak longitudinal right atrial strain, RA: right atrial, RV: right ventricular.

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Intraobserver and interobserver variability

Intraobserver and interobserver variability for PLRAS were 13.3% and 16.0%, respectively.

DISCUSSION

We found that PLRAS was significantly decreased in severe BPD infants than in the other BPD groups; however, there was no significant difference in PLRAS between moderate BPD infants and mild BPD infants. PLRAS in infants with severe BPD was also decreased, compared to the reported mean value of global RA strain in normal infants under 1 year of age estimated from the sum of global negative and positive RA strain, which was estimated to be approximately 30%.[20] Also, RV strain was significantly decreased in severe BPD infants than in both mild and moderate BPD infants. However, the difference in RV strain between mild and moderate BPD infants was not statistically significant. A decrease in PLRAS was significantly correlated with increased duration of mechanical ventilation duration; however, tricuspid E/e′ and maximum RA volume index were not.

To date, our study is the first to evaluate PLRAS in infants with BPD. Our results of decreased PLRAS in infants with severe BPD compared to infants with moderate BPD and infants with mild BPD may indicate RV diastolic dysfunction in severe BPD infants. We can speculate from our results that RV diastolic dysfunction is present in infants with severe BPD, since PLRAS in this group was decreased compared to reported mean value of global RA strain in normal infants under 1 year of age.[20] In infants with BPD, increased pressure overload to the RV due to increased lung pressure would result in increased RV stiffness, causing RV diastolic dysfunction.[21] Impaired RV filling would impact RA reservoir function, shown by the existing evidence of abnormal RA function in abnormal RV diastolic functions in repaired tetralogy of Fallot patients.[9] RA reservoir function has been considered as a predictor of adverse outcomes in adults who have pulmonary hypertension.[22] Since PLRAS mirrors the RA reservoir function, screening for PLRAS may be beneficial in predicting adverse outcomes possibly stemming from elevated pulmonary artery pressure in severe BPD infants.

In the presence of decreased RV systolic function, decreased movement of the TV annulus in systole from base to the apex could also decrease PLRAS.[18] In our study, decreased RV strain in severe BPD infants might have contributed to the decreased PLRAS in infants with severe BPD. In our study, infants with severe BPD had significantly lower gestational age and birth weight, and significantly lower RV strain than in the other two groups. The maturation pattern of RV mechanics in preterm infants has been reported to be affected by body weight and age,[23] therefore the lower gestational age and birth weight might have affected the more significant decrease in RV strain in the infants with severe BPD.

MPAP, as estimated by PAAT, differed significantly between severe BPD and mild BPD patients in our study, and the proportion of infants whose PAAT < 47 msec and therefore have pulmonary hypertension, was significantly higher in the severe BPD group than the moderate BPD or mild BPD groups. However, PAAT is limited by technical issues such as angle dependency, and PAAT values could be prolonged by the presence of RV dysfunction, thereby leading to underestimation of MPAP and pulmonary hypertension.[17] As such, our results of decreased PLRAS in severe BPD infants may be a more sensitive parameter to accurately reflect abnormal RV diastolic function than PAAT. Our results are in accord with other studies which have reported abnormal RV function in infants with BPD in the absence of documented elevation in pulmonary artery pressure.[24]

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Our results showed that maximum RA volume index did not differ significantly across the severity of BPD, while in infants with severe BPD, PLRAS was reduced compared to moderate and mild BPD infants. As such, the addition of RA strain to conventional methods of assessing RV diastolic function such as maximum RA volume index would be beneficial. When compared with mean global RA strain values in normal infants,[20] our findings of decreased PLRAS in the severe BPD group may imply elevated RV filling pressure presumably due to RV diastolic dysfunction. However, when RV diastolic dysfunction is present, RA volume alone is reportedly not considered a definitive tool for assessing increased RA pressure.[25] The reason for this is that, in the setting of RV diastolic dysfunction, changes in RA volume in this setting would be dependent on RA compliance.[25] When RA compliance is reduced, RV diastolic dysfunction might cause an excessive increase in RA pressure than in the increase in RA volume; conversely, when RA compliance is preserved, RV diastolic dysfunction would cause RA volume to be more increased in relation to RA pressure.[25] Therefore, measuring RA volume alone may not always accurately detect RV diastolic dysfunction. We speculate that supplementing RA strain in addition to maximum RA volume index and tricuspid E/e′ would help increase sensitivity in detecting RV diastolic dysfunction.

In our study, tricuspid E/e′ did not differ significantly across the BPD severity. Tricuspid E/e′ has been used to estimate RV filling pressure[26]; however, the E/e′ ratio is reportedly correlated to filling pressures on the condition that impaired relaxation of ventricles exists, which might not often be present in children.[27] Also, the tricuspid E/e′ results in infants with BPD may not be entirely accurate due to the limitations of Doppler echocardiography, such as angle dependency and tethering of nearby myocardium.[7] Since more than one parameter is recommended for the assessment of RV diastolic dysfunction,[28] therefore, utilizing PLRAS in addition to conventional echocardiographic parameters of RV diastolic dysfunction such as tricuspid E/e′ would help in the effective detection of RV diastolic dysfunction in infants with BPD.

Atrial reservoir strain has been reported to increase with age in normal infants and children,[20] and our results are in accord with previous findings, as our results show correlation between PLRAS and gestational age and birth weight. In addition, PLRAS has been used as a predictor of adverse events in patients with pulmonary hypertension.[29] In our study, a decrease in PLRAS showed significant correlation with increased duration of mechanical ventilation, while other parameters such as tricuspid E/e′ or maximum RA volume index did not.

Our results are in accord with the previously reported findings regarding correlation of impaired relaxation parameters and increased duration of mechanical ventilation.[6] However, the factors influencing respiratory prognosis in BPD are numerous, interrelated, and complex. For example, the association between PLRAS and duration of mechanical ventilation may further be influenced by how long the duration of high frequency ventilator settings persisted in individual cases, which was not investigated in our study. Another example could be that maternal factors, such as maternal age, maternal diabetes and smoking have been reported to influence late respiratory outcomes in addition to BPD in infants with BPD.[30] Also, the association between PLRAS and the duration of mechanical ventilation may be attenuated by the variability of PLRAS measurements, which may have resulted in overlap of PLRAS values between BPD severity groups.

To date, the association between echocardiographic parameters across the severity of BPD remains unclear. Others have reported no significant difference in echocardiographic

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parameters of RV function, except the eccentricity index, across the severity of BPD.[3] Still other studies regarding infants with BPD and respiratory outcomes have shown associations with parameters of RV function and respiratory outcomes, speculating that RV dysfunction may serve as a mediator of clinical respiratory outcomes.[6] In our study, PLRAS did not differ significantly between the mild and moderate BPD groups. We speculate that the heterogeneity of clinical characteristics of our study population, such as a history of prolonged infection not characterized as sepsis, or other contributing maternal factors such as history of gestational DM, or pregnancy induced hypertension, which was not investigated in our study, might contribute to the lack of a statistically significant difference in PLRAS between the moderate and mild BPD groups.

Our study was a retrospective study, therefore causality between BPD severity and RV dysfunction could not be established. The heterogeneity of the clinical characteristics across the severity of BPD might have affected the associations between BPD severity and PLRAS. Also, due to previously stated unknown confounders, we acknowledge that there are limitations to attempting to depict a more direct association between PLRAS and respiratory outcomes and PLRAS. As mentioned in other studies, we utilized software designed to track ventricles to track PLRAS, which may have affected our results.[20]

In conclusion, PLRAS was significantly decreased in infants with severe BPD compared to the other BPD groups. A decrease in PLRAS was significantly correlated with increased duration of mechanical ventilation duration; however, tricuspid E/e′ and maximum RA volume index were not. Evaluating PLRAS in conjunction with other echocardiographic parameters might help detect RV diastolic dysfunction in infants with BPD. Further studies involving longer follow up in a larger study population may help elucidate the association between PLRAS and respiratory outcomes in infants with BPD.

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