Value of right ventricular dimensions' swings on weaning from mechanical ventilation

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Background
Cardiac dysfunction is a common cause of weaning failure. Previous studies focused on the role of LV diastolic dysfunction in the occurrence of weaning failure, and there are no more studies yet interested in RV dysfunction.
Herein we evaluate cardiac changes using tissue Doppler echocardiography in critically ill patients during the weaning process and to compare between left and right ventricular functions as a predictor of weaning failure from mechanical ventilation.
Methods
We recruited 40 mechanically ventilated patients admitted to our ICU in this cross-sectional study. Echocardiography was performed during baseline ventilator settings and during spontaneous breathing trial (SBT) to assess changes in cardiac dimensions and functions. of left and right ventricular contractility and relaxation in this subset of patients.
Results
Among 40 patients included, there was a significant increase in cardiac dimensions during SBT. There was a statistically significant decrease in LV EF with (*P*=0.000) during SBT. Also, there was a statistically significant decrease in RV systolic function during SBT. There was a significant increase in the E/E′ ratio of lateral mitral annulus due to the evolution of impaired LV relaxation and diastolic dysfunction (*P*=0.008). Moreover, the development of right ventricular diastolic dysfunction presented by E/E′ ratio of tricuspid annulus, (*P* value=0.03).
Conclusions
RV dysfunction, as detected by systolic wave velocity of anterior tricuspid annulus (S′) by Doppler tissue imaging during SBT, also has good sensitivity and specificity in weaning failure prediction from mechanical ventilation.

Keywords:
Diastolic dysfunction, echocardiography, failed SBT, right ventricular dysfunction

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Cardiogenic pulmonary edema is a critical cause of weaning failure [1]. The shift of positive intrathoracic pressure to negative pressure is the primary mechanism responsible for weaning failure of cardiac origin, which causes an increase in the right ventricular (RV) preload and then an increase in the left ventricular (LV) preload [2]. Moreover, there is an increase in the LV surrounding pressure, which leads to an increase in LV afterload.

Previous studies have focused on the role of LV diastolic dysfunction in the occurrence of weaning failure, but there are no more studies yet interested in RV dysfunction.

Herein, we evaluated cardiac changes using tissue Doppler echocardiography in critically ill patients during the weaning process and compared between LV and RV functions as a predictor of weaning failure from mechanical ventilation.

Patients and methods

This is a nonblinded cross-sectional comparative study conducted on 40 mechanically ventilated patients admitted to the Critical Care Unit of the Internal Medicine Department of Assiut University Hospital. His study was supported by a fund from Grant office, Faculty of Medicine, Assuit University. Patients included in the study were intubated and mechanically ventilated for more than 48 h, and they

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became ready for undergoing a spontaneous breathing trial (SBT) after resolution of the precipitating cause for intubation, with adequate cough, absence of excessive airway secretion, stable hemodynamic and metabolic status, adequate oxygenation on minimal fractional of inspired oxygen and positive end-expiratory pressure (PEEP), and appropriate pulmonary functions [respiratory rate (RR) less than or equal to 35 breaths/min, tidal volume (TV) more than 5 ml/kg, vital capacity more than 10 ml/kg, rapid shallow breathing index less than or equal to 105 breaths/min/l (RSBI), and no significant respiratory acidosis] [3].

Patients with poor echocardiographic window, complete heart block, severe mitral regurge or mitral stenosis, prosthetic mitral valve, atrial fibrillation, congenital heart disease with a left to right shunt, and not ready for SBT were excluded from the study.

SBT is done for half an hour with pressure support at 8 cmH_2O and PEEP 5 cm water [3].

Transthoracic echocardiography was performed using a Philips EnViosr C HD (Philips Ultrasound, Bothell, WA, USA, www.ultrasound.philips.com) just before the SBT and 30 min after starting the SBT, LV systolic function by eyeball, and ejection fraction (EF) by M-mode at papillary muscle level (Techoliz method). LV diastolic function was assessed by pulsed Doppler over mitral inflow for measurement of peak E and A waves velocity (cm/s) and E/A ratio, isovolumic relaxation time, and pulsed Doppler tissue imaging (DTI) for E' velocity (cm/s) over lateral mitral annulus, with calculation of E/E' ratio which is closely related to LV end-diastolic filling pressure measured by pulmonary artery Swan-Ganz catheter [4].

RV systolic function had been evaluated using the RV myocardial performance index (MPI) by pulsed Doppler and DTI, tricuspid annular plane systolic excursion (TAPSE) and S' velocity, and S'/MPI [5], whereas RV diastolic function is carried out by pulsed Doppler of the tricuspid inflow and tissue Doppler of the lateral tricuspid annulus [5].

Parameters of weaning failure

Patients were considered to be failed weaning if they have any of the following signs during half an hour of SBT [3]:

Subjective indices

Agitation, anxiety, depressed mental status, diaphoresis, cyanosis, and evidence of increased respiratory efforts with increased accessory muscle activity.

Objective indices

PaO_2 less than or equal to 50–60 mmHg on FIO_2 more than or equal to 0.5 or SaO_2 less than 90%, $PaCO_2$ more than 50 mmHg or an increase in $PaCO_2$ more than 8 mmHg, pH less than 7.32 or a decrease in pH more than or equal to 0.07 U, RSBI (RR/VT) more than 105 breaths/min/l, RR more than 35 breaths/min or increased by more than or equal to 50%, HR more than 140 beats/min or increased by more than or equal to 20%, systolic blood pressure more than 180 mmHg or increased by more than or equal to 20% or

Table 1 Demographic and clinical characteristics of the studied sample (*N*=40)

Variables	Succeeded (*N*=18)	Failed (*N*=22)	*P* value
Age (years)[a]	63.78±8.07	59.18±9.68	0.272
Sex[b]			
Male	10 (56)	12 (55)	0.6
Female	8 (44)	10 (54)	
History of chronic pulmonary diseases[c]	8 (44)	10 (55)	0.52
History of ischemic heart disease[c]	0	8 (36)	0.000
Duration of mechanical ventilation (days)[c]	4 (2.5–5.5)	3 (1–5)	0.54
Indications of mechanical ventilation[d]			
Massive pneumonia	8 (44.4)	8 (36.4)	0.121
COPD exacerbations	4 (22.2)	4 (18.2)	
Overlap syndrome	2 (11.1)	4 (18.1)	
Postarrest with pneumonia	0	4 (18.2)	
Lupus cerebritis	2 (11.1)	0	
Obesity hypoventilation	2 (11.1)	0	
Massive pleural effusion	0	2 (9.1)	

COPD, chronic obstructive pulmonary disease. [a]Data are expressed in mean±SD; continuous data with normal distribution and compared with independent *t* test. [b]Data are shown in *n* (%); qualitative data and compared with the c^2 test. [c]Data are expressed in median and interquartile range; continuous data with non-normal distribution and compared with the Mann–Whitney test. [d]Data are represented in median and interquartile range; continuous data with non-normal distribution and compared by Fisher exact test.

Table 2 Changes in cardiac dimensions and systolic functions before and during spontaneous breathing trial

	Before SBT	During SBT	*P* value
Left atrial diameter	3.7 (3.42–3.97)	3.76 (3.3–4.2)	**0.009**
Left ventricular ESD	3 (2.75–3.75)	3.2 (2.85–3.55)	**0.001**
Left ventricular EDD	4.58 (4.18–4.98)	4.7 (4.2–5.2)	**0.001**
Right ventricular dimension	2.83 (2.74–2.91)	3.04 (3.01–3.07)	**0.000**
LV EF (%)	66 (60.5–69.5)	64 (61.5–68.5)	**0.000**
TAPSE (cm)	2 (1.52–2.47)	1.71 (1.32–2.1)	**0.000**
MPI of RV by pulsed-wave Doppler	0.33 (0.15–0.51)	0.59 (0.4–0.78)	**0.003**
MPI of RV by pulsed-wave tissue Doppler	0.6 (0.4–0.8)	0.6 (0.48–0.72)	0.357
Tricuspid systolic wave velocity by pulsed tissue Doppler (cm/s)	20 (15–25)	19 (14.5–23.5)	0.804
S'/MPI by DTI	38 (25.87–50.13)	30 (23.01–36.89)	**0.048**

Data expressed in median and interquartile range; continuous data with non-normal distribution and compared with Wilcoxon test. DTI, Doppler tissue imaging; EF, ejection fraction; EDD, end-diastolic pressure; ESD, end-systolic pressure; LV, left ventricular; MPI, myocardial performance index; PW, pulsed wave; RV, right ventricular; SBT, spontaneous breathing trial; TAPSE, tricuspid annular plane systolic excursion. The bold values denote the effects of positive intrathoracic pressure on cardiac dimensions, functions, and the changes in cardiac function during spontaneous weaning trial.

Table 3 Changes in cardiac diastolic functions before and during spontaneous breathing trial

	Before SBT	During SBT	*P* value
E/A ratio	0.83±0.25	0.91±0.34	0.247
E wave velocity of MV inflow (m/s)	0.73±0.26	0.81±0.28	**0.007**
E wave deceleration time of MV inflow (s)	0.16±0.04	0.17±0.05	0.660
E' velocity of lateral mitral valve annulus (m/s)	0.11±0.04	0.10±0.05	0.486
E/E' of lateral mitral valve annulus	7.80±3.73	9.59±4.98	**0.008**
IVRT (s)	0.08±0.04	0.09±0.05	0.234
Tricuspid E/A ratio	0.80±0.3	0.79±0.23	0.529
Tricuspid E wave velocity by PD/E' velocity by DTI	5.10±3.29	5.93±4.62	**0.032**

Data expressed in mean±SD; continuous data with non-normal distribution and compared with Wilcoxon test. DTI, Doppler tissue imaging; IVRT, isovolumic relaxation time; PW, pulsed wave; SBT, spontaneous breathing trial. The bold values denote the effects of positive intrathoracic pressure on cardiac dimensions, functions, and the changes in cardiac function during spontaneous weaning trial.

Table 4 Echocardiographic cardiac dimensions differences before and during spontaneous breathing trial in both succeeded and failed groups

Variable	Before SBT			During SBT		
	Succeeded (*N*=18)	Failed (*N*=22)	*P* value	Succeeded (*N*=18)	Failed (*N*=22)	*P* value
Left atrial	3.68 (3.49–3.86)	3.7 (2.75–4.65)	0.3	3.65 (3.07–4.13)	3.76 (3.01–4.51)	0.300
LVEDD	4.59 (4.47–4.70)	4.5 (3.8–5.19)	0.23	4.7 (4.62–4.78)	5.2 (4.4–6)	**0.019**
LVESD	2.97 (2.85–3.09)	3.23 (2.68–3.78)	0.157	3.15 (3.02–3.27)	3.1±0.8	**0.022**
RV	2.83 (2.81–2.84)	2.83 (2.25–3.41)	0.08	3.04 (3.02–3.05)	3.04 (2.61–3.47)	0.09

Data are expressed in median and interquartile range; continuous data with non-normal distribution and compared with Mann–Whitney test. EDD, end-diastolic pressure; ESD, end-systolic pressure; LV, left ventricular; RV, right ventricular; SBT, spontaneous breathing trial. The bold values denote the effects of positive intrathoracic pressure on cardiac dimensions, functions, and the changes in cardiac function during spontaneous weaning trial.

decreased systolic blood pressure less than 90 mmHg, and cardiac arrhythmias.

Statistical analysis

Statistical analysis was performed using IBM Statistical Package for the Social Sciences (SPSS) Software 19.0.0 (SPSS Inc., Chicago, Illinois, USA). Patients were divided into successful and failed weaning groups. Continuous variables were represented as median and interquartile range (25th–75th percentile), and categorical variables as percentages. Categorical variables were analyzed with a c^2 test or Fisher's exact test. Continuous variables were compared using the nonparametric Mann–Whitney *U* test or the Wilcoxon tests as appropriate concerning each of the echocardiographic parameters. *P* values less than 0.05 were considered statistically significant.

Results

Demographic and clinical characteristics of patients

From 40 patients, 22 (55%) patients had weaning failure during the SBT, whereas 18 (45%) patients succeeded. Patients enrolled in the study had a different indication for mechanical ventilation (Table 1). All patients with ischemic cardiovascular

Table 5 Right and left ventricular systolic and diastolic functions before and during spontaneous breathing trial in both succeeded and failed groups

Variables	Before SBT			During SBT		
	Succeeded (*N*=18)	Failed (*N*=22)	*P* value	Succeeded (*N*=18)	Failed (*N*=22)	*P* value
LV EF (%)	66 (62–70)	64 (59.5–68.5)	0.87	66 (62–69)	63 (59.5–68.5)	0.000
TAPSE (cm)	2.35 (2.13–2.57)	1.6 (1.28–1.92)	0.353	2.01 (1.84–2.17)	1.69 (1.29–2.09)	0.100
MPI of RV by PW Doppler	0.21 (0.02–0.44)	0.4 (0.25–0.55)	0.275	0.57 (0.52–0.62)	0.69 (0.5–0.88)	0.170
MPI of RV DTI	0.57 (0.41–0.75)	0.62 (0.3–0.91)	0.354	0.57 (0.52–0.62)	0.69 (0.5–0.88)	0.586
Tricuspid systolic wave velocity by DTI (S') (cm/s)	22 (18.75–25.25)	16 (10.5–21.5)	0.586	19 (14–24)	18.5 (15–21.99)	**0.038**
S'/MPI by DTI	41.5 (32.6–50.35)	25.8 (0.7–52.3)	0.384	32 (28.55–35.4)	28.9 (12.95–44.85)	0.114
E wave of MV inflow	0.57 (0.47–0.67)	0.7 (0.39–1.101)	0.513	0.72±0.22	0.88±0.31	0.076
E/A ratio	0.74 (0.51–0.96)	0.75 (0.6–0.89)	0.642	0.85±0.22	0.97±0.41	0.261
E wave deceleration time of MV inflow (s)	0.16 (0.15–0.17)	0.15 (0.12–0.18)	0.779	0.18±0.06	0.16±0.04	0.261
E' velocity of lateral mitral valve annulus	0.12 (0.4–0.15)	0.10 (0.1–0.14)	0.870	0.11±0.06	0.10±0.05	0.465
E/E' of lateral mitral valve annulus	6.4 (3.01–7.19)	8.4 (5.95–10.85)	**0.02**	9.11±3.67	11.96±5.54	**0.01**
IVRT (s)	0.07 (0.03–0.11)	0.06 (0.03–0.09)	0.536	0.09±0.03	0.09±0.05	0.864
Tricuspid E/A ratio	0.65 (0.49–0.80)	0.79 (0.53–1.04)	0.624	0.80±0.23	0.79±0.23	0.885
E/E' of anterior tricuspid annulus	3.68 (3.33–4.02)	4 (1.25–6.75)	0.209	4.68±1.9	6.95±5.86	0.124

Data are expressed in median and interquartile range; continuous data with non-normal distribution and compared with Mann–Whitney test. DTI, Doppler tissue imaging; EF, ejection fraction; IVRT, isovolumic relaxation time; LV, left ventricular; MPI, myocardial performance index; PW, pulsed wave; RV, right ventricular; SBT, spontaneous breathing trial; TAPSE, tricuspid annular plane systolic excursion. The bold values denote the effects of positive intrathoracic pressure on cardiac dimensions, functions, and the changes in cardiac function during spontaneous weaning trial.

diseases had failed the first SBT. All patients with massive pleural effusion or postarrest pneumonia were abandoned during the first SBT; however, others showed different percentages.

There was a significant increase in RR, HR, and RSBI and decrease in TV (in both groups) during SBT, with a statistically significance difference (*P*=0.000). Although there was a decrease in minute ventilation, it was not statistically significant (*P*=0.11), and when comparing these cardiorespiratory parameters between succeeded and failed groups before and after SBT, there was no statistically significant difference before SBT, although it was highly statistically significant difference after SBT.

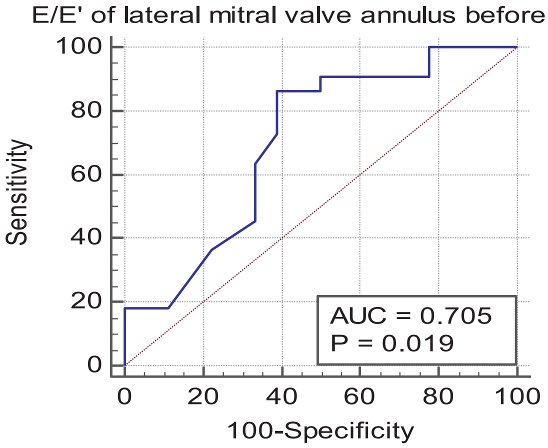
Echocardiographic parameters (cardiac dimensions and systolic and diastolic functions) before and during spontaneous breathing trial

There was a highly statistically significant increase in cardiac dimensions during SBT. Moreover, there was a statistically significant decrease in LV systolic function estimated by M-mode (i.e. EF) during SBT; furthermore, there was a considerable decrease in RV systolic function during SBT as assessed by TAPSE, MPI by pulsed Doppler, and S'/MPI by DTI (Table 2). Regarding LV and RV diastolic function before and during SBT, there was a significant increase in E wave velocity of mitral valve inflow (which represent early diastolic filling and reflects pulmonary venous return) during SBT, with *P* value=0.007, with increase in E/E' ratio of lateral mitral annulus. Moreover, there was a significant decrease in RV diastolic function represented by tricuspid E/E' ratio, with *P* value=0.032 (Table 3).

Echocardiographic parameters difference between succeeded and failed groups (cardiac dimensions, systolic, and diastolic functions) before and during spontaneous breathing trial

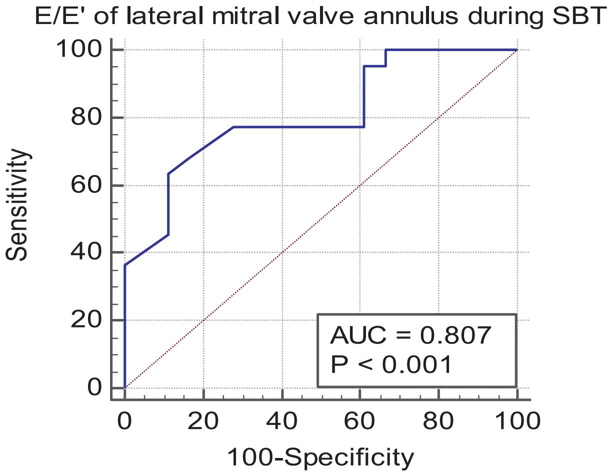
There was no statistically significant difference between successful and failed groups before SBT, but there was only a statistically significant increase in LV dimensions in the failed group during SBT (Table 4).

Figure 1



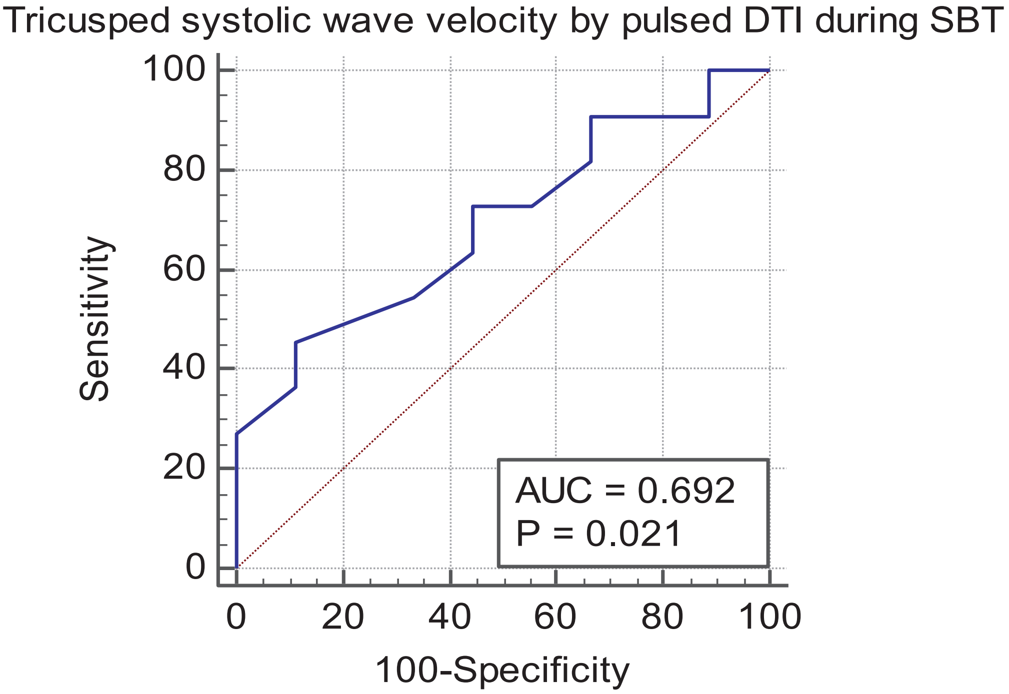
ROC curve shows sensitivity, specificity cut-off point, AUC, and *P* value of E/E’ wave velocity of mitral valve inflow before SBT. AUC, area under the curve; ROC, receiver operating characteristic; SBT, spontaneous breathing trial.

Figure 2



ROC curve shows sensitivity, specificity cut-off point, AUC, and *P* value of E/E’ wave velocity of mitral valve inflow during SBT. AUC, area under the curve; ROC, receiver operating characteristic; SBT, spontaneous breathing trial.

Figure 3



ROC curve shows sensitivity, specificity cut-off point, AUC, and *P* value of tricuspid systolic wave velocity of RV by pulsed DTI during SBT. AUC, area under the curve; DTI, Doppler tissue imaging; ROC, receiver operating characteristic; RV, right ventricular; SBT, spontaneous breathing trial.

There was only a significant decrease in LV systolic function in the failed group during SBT (*P*=0.000); furthermore, there was a substantial decrease in tricuspid systolic wave velocity by pulsed tissue Doppler in the failed group, which is representative of RV systolic function. However, for diastolic function, there was only a significant increase in LV diastolic filling pressure in the failed group before and during SBT (*P*=0.02, 0.01) (Table 5).

By using the receiver operating characteristic (ROC) curve, the E/E’ of lateral mitral annulus is the only significant predictor of LV parameters as a predictor of weaning failure; before SBT the cutoff point was more than 5.2, with sensitivity 86.3%, specificity 61.3% and with significant *P* value of 0.019, whereas during SBT, the cutoff point was more than 9, with sensitivity of 63.6% and specificity of 88.8%, with significant *P* value of 0.001.

On using the ROC curve, the systolic wave velocity of anterior tricuspid annulus (S') during SBT is the only significant predictor of RV parameters as a predictor of weaning failure, with a cutoff point more than 9, having sensitivity of 72.2% and specificity of 55.5%, with substantial *P* value 0.02 (Figs 1–3).

Discussion

Several studies have documented the development of cardiogenic pulmonary edema during weaning from mechanical ventilation [6]. It is well known that weaning from mechanical ventilation is associated with an increase in LV filling pressure, which predisposes to the occurrence of cardiogenic pulmonary edema and weaning failure.

However, weaning effects on RV dimensions and functions are underestimated in almost all previous studies.

We found marked increase in the median and interquartile range of RR, RSBI, of our patients at the end of SBT than before SBT with highly statistically significance (P=0.000), with a reduction in patients' TV. It is noticed that there were marked changes in respiratory parameters at the end of SBT, although all patients met the typical weaning criteria, which reflects that another pathophysiological mechanism leads to pulmonary congestion due to transition from a positive pressure ventilation to a negative pressure ventilation, which leads to increasing overload on cardiac muscle (increase in RV and LV preload and afterload) and, potentially, induces myocardial ischemia, which leads to weaning-induced diastolic dysfunction, increasing in LV end-diastolic filling pressure and finally leads to increase in RR, decrease in TV and causing weaning failure [7].

Moreover, there was a marked increase in the median and interquartile range of heart rate of our patients at the end of SBT than before SBT with a highly statistically significant difference. Tachycardia is one of the major determinants of diastolic dysfunction. It was more pronounced in weaning failure patients in our series and has been documented as numerous features of weaning induced by cardiac dysfunction [8]. Tachycardia could lead to alteration of ventricular diastolic function by reducing ventricular filling time and decreasing coronary perfusion [9].

There was a statistically significant increase in cardiac dimensions in our group of patients at the end of SBT. Herein, we found a statistically significant increase in RV dimensions at the end of SBT owing to the change from positive to negative intrathoracic pressure, which increases venous return to the right side. Left atrium dilatation is also attributed to the shift from positive to negative intrathoracic pressure, which increases venous return to the left atrium, and also the development of LV diastolic dysfunction during weaning [10]. Moreover, there is a statistically significant increase in LV end-diastolic and end-systolic dimensions at the end of SBT owing to shifting from positive intrathoracic pressure, which compresses the LV, to negative intrathoracic pressure, which increases the LV venous return (preload) [2].

When comparing cardiac dimensions between the succeeded and failed groups before the SBT, although there were variations, it was statistically insignificant to exclude confounding factors.

At the end of SBT, the median of LA diameter in the failed group was more than the succeeded group, but it was statistically insignificant owing to the short duration of SBT for the evolution of significant dilatation. This is attributed to higher LA filling pressure in the failed group at the end of SBT measured by E/E' of the lateral mitral valve annulus. Left atrial dilatation is a reflection of the degree and duration of LV diastolic dysfunction, which is associated with an increase in left ventricular end-diastolic pressure (LVEDP) and left atrial pressure [11].

Although there was no statistically significant difference between succeeded and failed group after SBT regarding RV dimensions, it was higher in the failed group, and this is also owing to short duration of SBT for the evolution of significant dilatation, but LV end-diastolic and end-systolic dimensions were significantly higher in the failed than the succeeded group.

Cardiac dysfunction participates in an essential role in the weaning outcome. Decreased LV EF is now considered as a risk factor for weaning failure [12]. Even in patients with preserved LV systolic function, there is increased LV volume and pressure overload during weaning, which may also impair LV diastolic function and induce pulmonary edema, especially in the case of preexisting diastolic dysfunction [13].

There was a decrease in LV systolic function (expressed by EF) at the end of SBT with statistical significance. This is owing to the development of negative pressure surrounding the LV, which resists blood ejection instead of positive pressure, which compresses the LV. Moreover, the increase in LV afterload is caused by an increase in sympathetic tone, which is created by emotional stress and potentially by hypercapnia and hypoxia [2], and increases in the work of breathing, with increased risk of myocardial ischemia with resulting decrease in LV systolic function.

While comparing the LV systolic function between the succeeded and failed groups during mechanical ventilation (before the SBT), there was no statistically significant difference, but at the end of SBT, there was a statistically significant difference. This was different than the study by Moschietto *et al.* [14], which did not find a difference in LV EF between succeeded and failed groups at the end of SBT, which can be explained by that their second echocardiography was done after 10 min in SBT, which is a short period for development of a decrease in EF by previously described mechanisms. Moreover, they used the Biplane Simpson method, which has a higher incidence of errors, especially in patients with bad echo windows owing to difficulties in optimal visualization of the endocardial border and tracing it.

Regarding the change in RV systolic function during mechanical ventilation (before SBT) and during SBT, there was a significant decrease in RV systolic function during SBT expressed by TAPSE, MPI by pulsed-wave Doppler, and by S’/MPI by DTI. Nevertheless, this is not the same for the MPI of RV by pulsed-wave tissue Doppler and tricuspid systolic wave velocity by pulsed tissue Doppler, as although there was a decrease, it was statistically insignificant. This may be related to the test technique used by the operator. The reduction in RV systolic function occurs because of an increase in RV afterload owing to pulmonary vasoconstriction by hypoxia and hypercapnia [2].

Evaluation of RV systolic function can be assessed by several methods, such as RV MPI, TAPSE by M-mode, RV fractional area change, systolic wave velocity of lateral tricuspid annulus by tissue Doppler (S’), three-dimensional (3D) RV EF, and longitudinal strain and strain rate. Although 3D RV EF seems to be more reliable with fewer errors, there are insufficient data to be used in clinical practice [15].

Weaning is associated with falsely low results of RV MPI owing to shift of positive to negative intrathoracic pressure, which increases right-side preload and elevates right atrial pressure, which will decrease the isovolumic relaxation time. Although TAPSE is a specific measure of RV systolic function, it has low sensitivity [5]. S’ velocity by tissue Doppler has been shown to correlate well with other measures of global RV systolic function, but Doppler cursor should be kept in line with basal segment and the annulus to avoid errors [5], which is challenging to be done in mechanically ventilated patients owing to swinging of heart with respiration, bad echo of the patients, and difficulties to put them in left lateral position. 2D RV fractional area change could not be done in our research because of challenges in tracing the whole RV area, which requires optimal visualization of the endocardial border (with the exclusion of trabeculations) during systole and diastole [5]. 3D RV EF and longitudinal strain and strain rate could not be done, as they are not present in our portable echocardiography device.

Our study showed a significant increase in E wave velocity of mitral valve inflow during SBT, reflecting an increase in mitral inflow and an increase in pulmonary venous return during the weaning process. Moreover, there was a significant increase in LVEDP during the weaning process at the end of SBT expressed by an increase in the E/E’ ratio of lateral mitral valve annulus [16]. The rise in LVEDP is similar to previous studies done by Moschietto *et al.* [14] and Lamia *et al.* [17]. Moreover, Caille *et al.* [18] found an increase in the E/E’ during SBT but without statistical significance. This is owing to the shifting from positive to negative intrathoracic pressure, which increases ventricular preload (venous return) and afterload, as mentioned before [2,19].

The failed group showed a higher E wave velocity of mitral valve inflow, with lower E’ velocity of the lateral mitral annulus, and then higher E/E’ ratio of the lateral mitral annulus before and during SBT, which indicates increased pulmonary venous return and impairment of LV relaxation. This is the same finding in Moschietto and Roche-Campo studies, which showed a marked increase in E/E’ in the failed group, whereas there was no increase in LV filling pressure in succeeded groups before and at the end of SBT.

E/E’ is closely correlated with LV filling pressure measured by pulmonary artery catheter even in mechanically ventilated patients [20]. Increase in E/E’ during stress test is a reliable marker of the impaired diastolic reserve [21] which is defined as normal or mild degree of impaired diastolic function with normal LV filling pressure during rest, whereas during exercise or stress, there is overt diastolic dysfunction with increased LV filling pressure [21]. Weaning failure occurs in some patients during SBT despite normal diastolic function at the baseline owing to the progressive decrease of LV diastolic function during SBT stress in patients lacking diastolic reserve.

This assumption is similar to studies done by Moschietto and colleagues and Roche-Campo and colleagues. However, our research and Moschietto and colleagues showed no significant decrease in E' velocity during failed weaning trials; this in contrast with the study by Roche-Campo *et al.* [10], which found a substantial decline in E' during SBT. This discrepancy may be attributed to the time of second echocardiography, as in our series was done after 30 min during SBT, and in the study by Moschietto and colleagues after 10 min, whereas in the trial by Roche-Campo *et al.* [10], the second echocardiography was done at the end of SBT. Moreover, the modality of SBT may play a critical role, as we use 5-cm PEEP with pressure support 8 cm, whereas in the previous study, they used zero PEEP.

Haji *et al.* [22] also showed increasing E wave velocity, E/A, and E/E' ratio in the failed group at the end of SBT, with decreasing in E' velocity.

Assessment of RV diastolic function is done by pulsed-wave Doppler over the tricuspid inflow with measurement of E and A wave ratio, tissue Doppler of the lateral tricuspid annulus, pulsed Doppler of the hepatic vein, and measurements of inferior vena cava (IVC) size and collapsibility [15]. Nevertheless, pulsed Doppler of the hepatic vein and measurements of IVC size and collapsibility cannot be done owing to positive pressure ventilation with dilating IVC and hepatic veins during inspiration instead of collapse. Note that these parameters should be obtained at end-expiration [15], which is difficult to achieve in our patients and may cause an error during estimation.

There was a statistically significant decrease in RV diastolic function before and during SBT by using tricuspid E wave velocity by PD/E' velocity by DTI. This is owing to the increase in RV preload and afterload, as described before. However, there was no significant difference in RV diastolic function in either succeeded or failed groups before or during SBT. Our study is the only study that examined RV systolic and diastolic function by echocardiography before and during SBT to date.

The significant findings of our study are that it shows serial measurement of E/E' may predict weaning failure. E/E' before SBT was significantly higher in the failed group; however, diagnosis in predicting weaning failure, as estimated from the ROC curve, was poor owing to low cutoff point more than or equal to 5. Interestingly, E/E' measured 30 min after starting the SBT showed better diagnostic performance.

These results are suggestive of an increase in LVFP during the SBT in the failed group. These findings are in line with previous studies.

Systolic wave velocity of anterior tricuspid annulus (S') estimated by DTI during SBT is the only significant predictor of RV parameters as a predictor of weaning failure. This is owing to the shift of positive intrathoracic pressure to negative pressure, with an increase in cardiac preload and afterload, which may predispose to acute RV dilatation and failure if RV function is impaired. However, before SBT, there is positive intrathoracic pressure, which decreased RV preload and afterload. Other parameters of estimation of RV function have other limitations, as mentioned before.

Conclusion

We conclude that although changes in LV dimensions and functions during weaning still have a crucial role in the prediction of weaning failure, RV dysfunction, as detected by systolic wave velocity of anterior tricuspid annulus (S') by DTI during SBT, also has a good sensitivity and specificity in weaning failure prediction from mechanical ventilation, which need more attention and wide-scale researches.

Recommendations

Based on the results, we recommend the following:

Using new cardiac parameters by tissue Doppler echocardiography such as E/E' of lateral mitral annulus before and during SBT and S' of anterior tricuspid annulus during SBT as predictors of weaning failure from mechanical ventilation.

It is better to do SBT by using pressure support with minimal PEEP than using a T-piece trial as it causes lesser changes in intrathoracic pressure and then cardiac preload. Postpone weaning till control of all possible predisposing conditions which affect cardiac function, like heart rate, blood pressure, volume overload, arrhythmias, ischemia, especially if the patient had a history of systolic or diastolic dysfunction.

Consider the use of low-dose diuretic before SBT if the patient had a history of cardiac diseases. Avoid using high PEEP unless necessary with gradual reduction before SBT as it causes deterioration of RV function.

Limitations

We could not insert Swan-Ganz catheter to confirm weaning-induced pulmonary congestion, which is the cause of weaning failure, and to determine the mechanism of pulmonary congestion is either diastolic dysfunction or increase in LV afterload. Moreover, the sample size of our population was small.

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Conflicts of interest

There are no conflicts of interest.

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