

Can We Predict Left Ventricular Dysfunction Induced Weaning Failure? Invasive & Echocardiographic Evaluation

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Abstract: Introduction: Cardiovascular dysfunction may contribute to unsuccessful ventilator discontinuation & promote prolonged ventilator dependence. **Aim:** Studying the relation of weaning failure to development of LV diastolic dysfunction using echo & PA catheter **Methods:** Thirty invasively mechanically ventilated patients fulfilling criteria of weaning from mechanical ventilation & Shifted to SBT (using low PSV (8 cmH₂O)) for 30 minutes. 2 sets of variables were measured at the beginning & end of the SBT; Weaning failure was defined as; Failed SBT, Reintubation and /or ventilation or death within 48 h .Following extubation ,Swan Ganz catheter was inserted to get the right atrial (RAP) ,pulmonary artery (PAP), pulmonary artery occlusion (PAOP) pressures, & cardiac index (CI). Using trans-thoracic Echocardiography; The LV internal diameter at end diastole (LVIDd) & end systole (LVIDs), Ejection fraction (LVEF %), E/A ratio; Deceleration time (DT), Isovolumetric relaxation time (IVRT) in ms were measured & E/E' ratio using tissue Doppler imaging (TDI). **Results:** Mean age was 56.6 ±15.9 years, 53% were males. The outcome of weaning was successful in 76.6% of patients. The pts were subdivided into two groups according to weaning outcome; Group I: 23 pts (successful weaning), Group II: 7 patients (failed weaning). RAP, PAOP&SVO₂ were similar at the start of SBT(6.3±1.9 vs.7.6±2.3,p=0.1), (12±3.7 vs.14.6±3,p=0.4), (72±2.4 vs.71±3.1,p=0.1) between group I & II yet significantly different at the end of SPT; (6.2±2.4 vs.10±3.5,p=0.01), (12.8±3.5 vs.19±5.4,p=0.004), (73±2.8 vs.66.6±7, p=0.009) respectively. CI was similar between group I &II at both ends of the SBT, p=0.5&0.9.Group I & II had similar LVIDs &EF at the beginning of SBT (3±0.7 vs. 3.3±0.5,p=0.2),(68±8vs.62±6,p=0.08) yet different at the end (3±0.6 vs. 3.5±0.5,p=0.048), (66±8vs.58±7, p=0.03) respectively. There was no significant differences in E/A,IVRT,DT yet significant difference in E/E' between group I &group II at both ends of the trial; (1.04±0.4 vs. 0.97±0.3,p=0.78),(1.02±0.4 vs.1.07±0.4,p=0.78),(94±26 vs. 99.6±18, p=0.52),(97±22vs. 91±24,p=0.57),(194±31 vs. 196±30,p=0.98),(197±27 vs.189±33, p=0.6), (8.9±2 vs. 12.2±4, p=0.02), (9.4±2.3 vs. 13±5, p=0.02) respectively. **Conclusions:** LV dysfunction may have an impact on weaning outcome. Invasive monitoring as well as echocardiography and tissue Doppler indices may be reliable in monitoring and detection of LV dysfunction and subsequently may be possibly useful in improving weaning outcome RAP may be particularly a reliable and easy method to monitor during the period of weaning.

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Key words: mechanical ventilation, LV dysfunction, echocardiography & weaning failure

Introduction

Even if its actual incidence is unknown, cardiac tissue imaging variables.^[8] pressures using Doppler transmitral flow and Doppler dysfunction is a leading cause of weaning failure^[1]. The transthoracic approach is the easiest and Breathing in the context of weaning was described as least invasive way to image cardiac structures and as a physical exercise^[2]. The abrupt cessation of positive result of the significantly improved technical quality of pressure ventilation increases venous return and left TTE imaging, the majority of ICU patients can be ventricular (LV) afterload^[3], decreases LV compliance^[4], satisfactorily studied with this modality^[9]. and may even induce cardiac ischemia^[5]. All these factors Numerous clinical studies performed in tend to increase LV filling pressure^[4, 6], and may spontaneously breathing heart failure patients have subsequently result in cardiogenic pulmonary edema. Since uniformly shown that echo Doppler may accurately the classical signs of pulmonary oedema such as crackles predict invasive PAOP.^[10-15] and foamy excretions are usually lacking in this Although echocardiography allows the situation, the confident diagnosis of weaning-induced noninvasive assessment of cardiac function and LV pulmonary oedema is based upon the assessment of an filling pressures, its clinical value in the setting of increase in PAOP^[7]. ventilator weaning has yet to be determined^[16].

Echocardiography has become a routine tool for evaluating the cardiovascular status in critically ill patients; it's now possible to estimate LV filling

Patients & Methods: This prospective study was conducted over 30 patients admitted to critical care

department, Cairo University from August 2009 to August 2010. The protocol was approved by our institution and consent was obtained from the patients' next of kin.

- The patients were ventilated in the assist-control mode using a Puritan-Bennett 7200a (Puritan-Bennett, Los Angeles, CA) ventilator through a cuffed endotracheal tube.

Inclusion criteria:

We studied patients who had all of the following criteria for weaning From mechanical ventilation^[1]: adequate cough, absence of excessive tracheobronchial secretion, resolution of the acute episode for which the patient was intubated, stable cardio-vascular status (heart rate <120/min, systolic blood pressure >90 mm Hg and <160 mm Hg), stable metabolic status, adequate oxygenation (PaO₂/FIO₂ ≥150, positive end-expiratory Pressure ≤8 cm H₂O), adequate pulmonary function (respiratory rate <35/min, tidal volume >5 mL/kg, no significant respiratory acidosis), no sedation or stable neurologic status, 2) who had pulmonary artery catheter inserted as part of their medical management.

Exclusion criteria:

Patients were excluded if they had severe mitral regurgitation, mitral stenosis, mitral prosthesis, atrial fibrillation, or myocardial ischemia. The spontaneous breathing trial (SBT) was defined as failed if, during the test, at least one of the following signs appears: diaphoresis, use of accessory respiratory muscles, worsening of discomfort, respiratory rate ≥35/min, pulse oxygen saturation ≤90%, heart rate ≥140/min, and systolic arterial pressure ≥180 mm Hg.

Arterial Blood Gas and mechanical ventilation data: Arterial oxygen tension (PaO₂), carbon dioxide tension (Paco₂), pH, and arterial oxygen saturation (SaO₂) were measured after blood sampling in the radial artery. Tidal volume (V_T), respiratory frequency (f), minute ventilation (V_E), peak airway (PIP) and plateau pressures (P_{plat}), airway resistance (R_{aw}), and static compliance (C_{eff}) were measured during the SBT.

Invasive Pressure Measurements: An indwelling arterial line, the distal and proximal ports of the pulmonary artery catheter from EDWARD IFESCIENCES® were connected to a strain-gauge manometer that provided continuous recordings of mean systemic arterial, pulmonary arterial and right atrial pressures, respectively. The pulmonary artery occlusion pressure (PAOP) was determined after balloon inflation and read at end-expiration. Cardiac output (CO) was measured by the thermodilution principle using 10-ml aliquots of saline at room temperature randomly injected throughout the

respiratory cycle, and the average of five measurements was taken.

Echocardiography and Doppler: The echocardiographic examination was performed using a transthoracic ultrasound device (ATL HDI 5000 colored echocardiographic machine with TDI software incorporated in the device using a 3.5 MHz transducer. Echocardiographic images were recorded together with the electrocardiogram. Image recordings and off line analysis were carried out by a physician who was blinded to all bedside variables, hemodynamic data, and clinical outcomes. LV systolic function was assessed using M-mode linear measurement of LV size. Pulsed Doppler was used to record transmitral flow in the apical four chamber view. The following parameters were measured: Peak early (E), atrial (A) flow velocity (cm/s), and E/A ratio. Doppler imaging of the mitral annulus was obtained from the apical four-chamber view. A 1.5-mm sample volume was placed at the septal mitral annulus, and the Ea peak velocity was measured. The E/Ea was then computed.

Study Design: An SBT was performed over a thirty minutes period using low pressure support protocol (8cmH₂O). The SBT was stopped if the patient did not tolerate spontaneous breathing. A first set of hemodynamic, blood gas, mechanical ventilation data and echocardiographic measurements was collected at baseline before the SBT while the patient was in a semi recumbent position (45 degree). A second set of measurements was collected at the end of the SBT. The decision to stop the SBT was taken by the attending physician according to the criteria previously detailed, regardless of the changes in PAOP. The attending physician was blinded to the TTE data.

Statistical Analysis: Data were statistically described in terms of range, mean ± standard deviation (± SD), median, frequencies (number of cases) and percentages when appropriate. Comparison of quantitative variables between the study groups was done using Mann Whitney U test for independent samples. Within group comparison between reading 1 and 2 was done using paired t test. Correlations were done using Spearman rank correlation for non normal data. Accuracy was represented by sensitivity and specificity. ROC analysis was done to determine the best cutoff of the diagnostic markers. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using computer programs Microsoft Excel 2007 (Microsoft Corporation, NY, USA) and SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 15 for Microsoft Windows.

Results

A. Characteristics of The studied Subjects:

1) Age& Gender:

The mean age of our patients was 56.6±15.9 (range 24–80 years). Males represented 53%.

2) Underlying diseases:

The most common underlying cause for ICU admission was chest related causes (The most common is pneumonia which represented 23% of patients), followed by severe sepsis& septic shock which represented 13% of patients included in the study.

B. Mechanical Ventilation Data

i. Indication for mechanical ventilation:

The most common indication for mechanical ventilation was refractory hypoxemia which represented 66.7 %. Table (1).

Table 1: Causes of mechanical ventilation

Indication of mechanical ventilation	No	Frequency (%)
Hypoxemia	20	66.7
Type II respiratory failure	5	16.7
Apnea	2	6.7
Need for heavy sedation	2	6.7
Lung collapse	1	3.3
Total	30	100

ii. Outcome of Weaning:

Out of 30 patients, 23 patients passed the SBT and extubated, one Patient experienced post extubation stridor and was reintubated, 6 Patients failed the initial SBT. Figure (1)

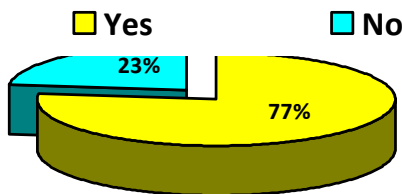


Figure 1: Frequency of weaning failure

i. Causes of failure:

The most common cause of weaning failure was occurrence of severe Bronchospasm and cardiogenic pulmonary oedema which represented 42.9% each. Figure (2).

According to the outcome of weaning, the patients were subdivided into two groups:

- **Group I:** successful weaning (23 patients);
- **Group II:** failed weaning (7 patients).

Comparison between group I and group II regarding their measured variables:

i. Mechanical ventilation and arterial blood gases variables:

1. Volumes and frequency:

Patients who failed the SBT had significantly lower V_T and higher F & V_E at the end of the trial than patients who succeeded. Table (2).

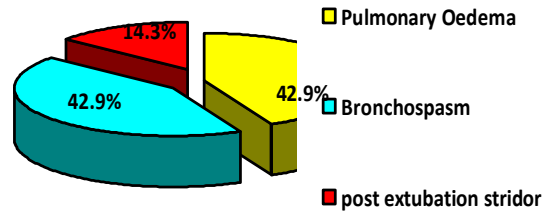


Figure 2: Causes of weaning failure

Table 2: V_T , V_E & F between both groups (significant differences)

	Group I	Group II	P value
V_T 2 (after SBT)	0.47±0.1	0.38±0.1	0.03
F 2	17±4.3	31±5.4	0.0001
V_E 2	8±1.6	11.6±2.2	0.0007

Before the trial, there were no significant differences between both group regarding their V_T , F and V_E . Table (3)/

Table 3: V_T , V_E & F between both groups (insignificant differences)

	Group I	Group II	P value
V_T 1 (before SBT)	0.5±0.1	0.55±0.04	0.27
F 1	13±1.5	11±2.8	0.14
V_E 1	6.7±1.5	6.4±1.8	0.41

We found patients who failed the trial had significantly higher PIP at the end of the trial while they had significantly lower C_{eff} before the SBT. Table (4).

Table 4: Lung mechanics between both groups (significant differences)

	Group I	Group II	P value
PIP 2 (cmH ₂ O)	17.7±2.3	20.7±1.8	0.01
C_{eff} 1 (ml/cmH ₂ O)	42±6.8	35±4.5	0.02

Both groups didn't have significant differences in their PIP and R_{aw} before the trial. Table (5).

Table 5: Lung mechanics between both groups (insignificant differences)

	Group I	Group II	P value
PIP 1	28±4	31.6±7	0.45
R_{aw} 1 (cmH ₂ O/L/sec)	10.4±3.5	12±6.8	0.6

2. Respiratory indices:

The patients who failed to be weaned had significantly lower NIP before the trial; at the end of the trial they have significantly lower PaO₂/FiO₂ and higher F/V_T than patients who succeeded. Table (6)

Table 6: Respiratory indices between both groups

	Group I	Group II	P value
P/F 2	275±67	178±41	0.001
NIP	-44.6±5.5	-35.6±5.1	0.006
F/V _T	38.4±13.7	87±29	0.0003

3. Arterial blood gases:

Patients who failed the trial had significantly lower oxygenation variables (PaO₂ and SaO₂) at the end of the trial .Table (7).

Table 7: Arterial blood gases between both groups (significant differences)

	Group I	Group II	P value
PaO ₂ 2	125±24	80±13	0.0002
SaO ₂ 2	97±1.3	92.9±2.1	0.0003

Other variables didn't show statistically significant differences before and at the end of the trial. Table (8).

Table 8: Arterial blood gases between both groups (insignificant differences)

	Group I	Group II	P value
PH 1	7.42±0.05	7.41±0.05	0.7
PH 2	7.43±0.04	7.45±0.09	0.47
PCO ₂ 1	38±6.7	36±5.3	0.52
PCO ₂ 2	37±5.2	34±8.6	0.12
PaO ₂ 1	131±34	109±17	0.085
SaO ₂ 1	97.3±1.3	97.1±1.6	0.74

ii. Hemodynamic variables:

Patients who failed the trial had significantly higher HR, RAP and PAOP and significantly lower SvO₂ at the end of the trial. Table (9).

Table 9: Hemodynamic changes between both groups (significant differences)

	Group I	Group II	P value
HR 2	95±14.5	111±15.6	0.023
SvO ₂ 2	73±2.8	66.6±7	0.009
RAP 2	6.2±2.4	10±3.5	0.015
PAOP 2	12.8±3.5	19±5.4	0.004

Patients who failed to be weaned had lower CI at the end of the trial than before the SBT, while in patients who passed the trial, CI increased with no significant differences. Other variables didn't differ significantly between both groups. Table (10).

Table 10: Hemodynamic changes between both groups (insignificant differences)

	Group I	Group II	P value
SBP 1	125±10.4	127.3±13.8	0.64
SBP 2	129.3±25.4	130±13.5	0.7
DBP 1	74±6	75±7.7	0.9
DBP 2	74.3±10.6	74.9±8.6	0.92
SvO ₂ 1	72±2.4	71±3.1	0.11
HR 1	90±14	90.4±13.8	0.98
RAP 1	6.3±1.9	7.6±2.3	0.096
SPAP 1	28±7.2	33±6.7	0.14
SPAP 2	29±6.6	32±8.2	0.34
PAOP 1	12±3.7	13.8±6.6	0.6
CI 1	3.1±0.5	3.38±0.7	0.53
CI 2	3.21±0.5	3.25±0.6	0.9

Out of seven patients who failed the weaning process; three of them had PAOP ≥18 mmHg at the end of SBT. Table (11)

Table 11: PAOP in failure group before and at the end of the SBT

PAOP at the end of the trial	N.	PAOP Before the trial	PAOP After the trial	P value
≥18mmHg	3	15.6±10.7	22.6±6.4	0.1
<18mmHG	4	12.5±2.4	15.8±1.5	0.014
Total	7	13.8±6.6	19±5.3	0.08

- Trend of PAOP values in failure & successful group

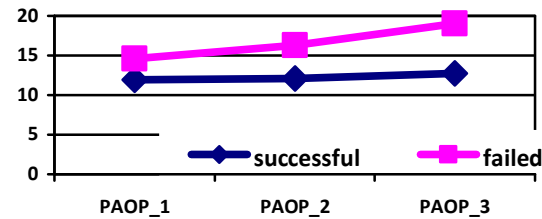


Figure 3: Trend of PAOP values in failure & successful group

iii. Echocardiographic variables:

1) Global systolic function:

EF was significantly lower in patients who failed to be weaned at the end of SBT. Table (12)

Table 12: Global systolic function in both groups (significant differences)

	Group I	Group II	P value
ESD 2	3±0.6	3.5±0.5	0.048
EF 2	66±8	58±7	0.03

We didn't find statistically significant differences between both groups regarding other variables. Tables (13).

Table 13: Global systolic function in both groups

	Group I	Group II	P value
EDD 1	4.8±0.7	4.9±0.7	0.52
EDD 2	4.7±0.7	5.1±0.6	0.069
ESD 1	3±0.7	3.3±0.5	0.2
EF 1	68±8.3	62±5.6	0.08

2) Mitral flow analysis:

Mitral inflow derived variables didn't differ significantly between both group of patients. Nevertheless, E/A increased while IVRT and DT decreased in patients who failed the trial compared to those who succeeded. Tables (14).

Table 14: Mitral flow variables between both groups

	Group I	Group II	P value
E wave 1	0.79±0.2	0.77±0.2	0.8
E wave 2	0.82±0.2	0.81±0.2	0.86
A wave 1	0.79±0.16	0.8±0.16	0.86
A wave 2	0.84±.2	0.77±0.2	0.16
E/A 1	1.04±0.4	0.97±0.3	0.78
E/A 2	1.02±0.4	1.07±0.4	0.78
IVRT 1	94±26	99.6±18	0.52
IVRT 2	97±22	91±24	0.57
DT 1	194±31	193±30	0.98
DT 2	197±27	189±33	0.6

3) Doppler tissue imaging:

Patients who failed the trial had significantly higher E/E' before and after the trial compared to those who succeeded. Table (15).

Table 15: DTI variables between both groups

	Group I	Group II	P value
E' 1	0.09±0.02	0.065±0.02	0.01
E' 2	0.091±0.02	0.063±0.01	0.005
E/E' 1	8.9±2	12.2±4	0.022
E/E' 2	9.4±2.3	13±5	0.023

Correlation between E/E' and both RAP & PAOP:

E/E' correlated well with both RAP and PAOP at the end of the SBT.

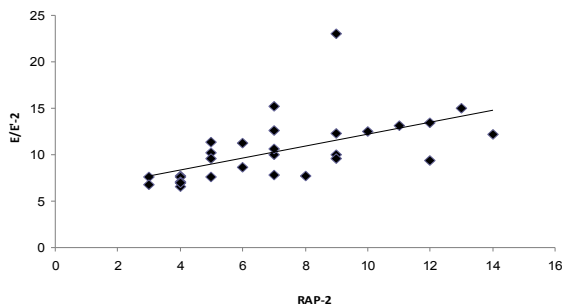


Figure 4: Correlation between RAP-2 and E/E'-2 among study cases (r: 0.74, p<0.001)

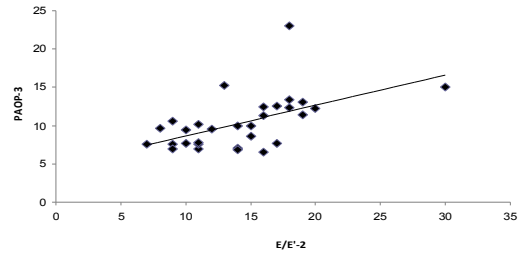


Figure 5: Correlation between E/E'-2 and PAOP-3 among study cases (r: 0.6, p<0.001)

Ability of septal E/E' at the end of the SBT to detect a weaning induced PAOP elevation:

We found a value of E/E' >10.4 at the end of the SBT predicted weaning induced PAOP elevation ≥18mmHg with a sensitivity of 100%, and a specificity of 78%.

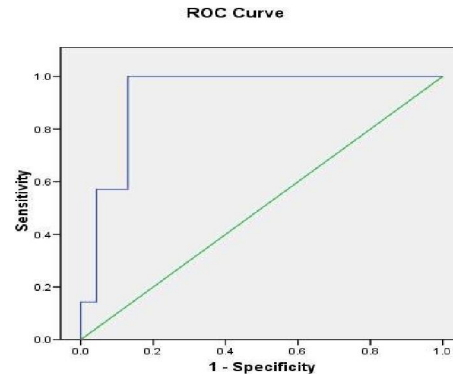


Figure 6: ROC curve

Discussion

The main finding in our study is that a septal E/E' value of more than 10.4 can predict weaning induce PAOP elevation during weaning from mechanical ventilation with a sensitivity of 100% and specificity of 78%.

**Cardiovascular interaction and weaning
Right heart catheterization and Cardiac function alterations in patients with successful and failed weaning outcome**

In our study, throughout the SBT, the subset of patients who failed the trial had a significant higher HR than those who were successful. Sinus tachycardia designates stress and theoretically a higher risk of extubation failure [17].

Esteban et al. [18, 19] noted a significantly higher HR in patients who failed SBTs compared with those who were successful.

In our study, patients who failed to be weaned exhibited significantly higher RAP and PAOP at the

end of SBT. This reflected the impact of increased LV filling pressures and decreased LV compliance on weaning outcome.

This was similar to the results of the Lemaire et al., Jubran et al.,^[4,6].

In the study of Anguel et al. patients with weaning induced pulmonary oedema exhibited significantly larger increases in PAOP^[20].

Lamia et al.^[21] assessed the role of transthoracic echocardiography to diagnose PAOP elevation during weaning from mechanical ventilation; they found all patients who had weaning induced elevated PAOP failed the trial. In our study, there was no significant difference in SvO₂ between patients who either failed or passed the weaning process at the start of the SBT, on the other hand, patients who failed the trial exhibited significant decline in SvO₂ whereas it remained unchanged in success group, this later result was probably explained by an insufficient increase in oxygen transport to meet the global oxygen demand. This was similarly shown by Jubran et al.,^[6] and **Cassiano et al.**^[22] who showed that reduction of central venous O₂ saturation (ScvO₂) by >4.5% was an independent predictor of reintubation with high sensitivity and specificity (88%, 95% respectively).

In our study, before the SBT, no significant difference in CI existed between failure & success group, at the end of the trial, CI increased in the Success group, while in the failure group it didn't change. These findings may demonstrate the inability of the heart in failure group to respond to subsequent alterations in venous return by increasing CO to meet the excess oxygen demands during weaning.

These findings were similar to **Jubran et al.**^[6] and **Anguel et al.**^[20]

In our study, PASP didn't differ significantly between patients who either failed the SBT or who were successful, which may reflect the negligible changes in right ventricular impedance during the SBT. This was in contrast to other studies that reported significant difference between both groups during the SBT^[6, 20& 21]. This could be explained by the small portion of patients with COPD in our study (who are likely to have right heart disease) compared to other studies.

In our study, most of patients whose PAOP was elevated failed to pass the SBT (72.4%) while the failure in patients whose PAOP didn't rise was 8.7%.

Lamia et al.^[21] found that a significantly larger proportion of patients with weaning-induced PAOP elevation than without weaning-induced PAOP elevation failed the SBT (100% vs. 64% respectively)

Anguel et al.^[20] found that a significantly larger proportion of patients with than without weaning-induced pulmonary oedema failed at the weaning trial (100% vs. 47%, this indirectly confirms

that an abnormal cardiac response to weaning contributed to weaning failure in patients with weaning induced PAOP elevation.

✚ **Transthoracic echocardiography and the effect of SBT on hemodynamics**

Weaning induced increase in LV afterload may explain the SBT induced decrease in LVEF. This was demonstrated by Richard et al. but on less no of patients.

For the entire studied patients, SBT induced an increase in E/A ratio and decrease in deceleration time (DT), which reflect that weaning induced alterations in LV diastolic properties.

Caille et al.^[16] found that statistically significant increase in E/A ratio and shortening of DT during SBT was only observed in patients with LV dysfunction (<50%) which suggested further deterioration in LV diastolic properties induced by SBT.

In the patients included in our study; LVEF was >50% which explained the insignificant change in DT&E/A in comparison to the significant changes found by **Caille et al.**^[16]

Ait-ouefelle et al.^[23] found that E/A ratio significantly increased also with a significant decrease in DT during the weaning trial.

In our study, E/E' increased significantly during the trial which may reflect the weaning induced increase in LV filling pressure. Similar to **Caille et al.**^[16] and **Lamia et al.**^[21]

In a similar study Campo et al.^[24] there was greater rise in E/E' during failed trials as compared to successful trials. This emphasizes the finding that diastolic dysfunction was more prevalent in patients who failed the SBT as compared to those who passed the trial.

Ability of transthoracic echocardiography to reflect LV filling pressures changes during weaning

In our study, we found a weak correlation between transmitral flow derived variables (E/A, IVRT, and DT) and PAOP, the best correlation was with DT.

This result was consistent with the weak correlation found between PAOP and E/A found in studies including critically ill patients^[7, 15-16], particularly with normal left ventricular ejection fraction^[25, 26].

Vignon et al.^[25] in a study to assess pulmonary artery occlusion pressure in ventilated patients with transesophageal echocardiography reported that correlations between Doppler variables and PAOP values were consistently closer in the subset of patients with depressed LV systolic function, when compared to patients with preserved cardiac

performance, this explain why we found such weak correlations between transmitral flow derived Doppler variables and PAOP as all patients included in our study had preserved LV function(>50%).

Lamia et al.^[21] found that at the end of the SBT, the value of E/A predicted a PAOP \geq 18 mmHg only fairly well.

Doppler tissue imaging and assessment of LV filling pressures during the SBT

The mitral E wave velocity (E) is directly influenced by left atrial pressure and inversely altered by changes in the time constant of relaxation^[27].it is therefore not surprising that by itself, the E wave velocity correlates poorly with left atrial pressure^[28], dividing the E wave velocity (E) by E' provides a method to correct the transmitral velocity for the influence of relaxation^[28].

In our study, we used DTI for assessment of cardiac function alterations, several studies were done validating the accuracy with which Doppler tissue derived variables can reflect left sided filling pressures^[28-29].

It was stated that DTI derived variables may better reflect cardiac filling pressures particularly in patients with preserved LV systolic function^[25].

In our study, we used the septal annulus as it is the preferred site for DTI, because this location is less influenced by the pericardium^[30].

Nevertheless, **Srinivasan et al.**^[31] found that both septal and mitral E/E' showed excellent correlation with PAOP but the lateral E/E' was reported to be better.

Similarly Combes et al.^[29] found that septal E/E' > 9 predicted PAOP \geq 15mmHg, Vignon et al.^[25] and Lamia et al. found the value of lateral E/E' > 8-8.5 predicted PAOP > 18 mmHg.

We also found a significant correlation between septal E/E' and RAP during the SBT, which gives us an insight into how probable is the ability of septal E/E' to reflect right sided filling pressures given their close proximity.

Potential limitations of our study must be discussed, first the small number of studied patients, second, measured end expiratory PAOP could overestimate the transmural PAOP value in actively breathing patients which lead us to the possibility that a few patients were misclassified, third, echocardiography is an operator dependent technique, fourth, the history of diuretic use in the last 48 hours were not taken into consideration which probably led to either soften or harden the effects of SBT on hemodynamics, fifth, we purposely excluded patients with non sinus rhythm because Doppler indices are more challenging to precisely measure in this setting, finally, silent myocardial ischemia can't be confidently

excluded in our patients because real-time ST segment monitoring with 30-min trends wasn't performed.

Conclusion

- Cardiac dysfunction could represent an important cause of weaning failure.
- Right atrial pressure may be a useful tool to monitor cardiac function alterations during the process of weaning.
- Transthoracic echocardiography is a useful noninvasive method to study cardiac function alterations during the process of weaning
- Weaning failure of cardiac origin is mainly diastolic in origin.
- Doppler tissue E/E' is a reliable tool to reflect cardiac dysfunction that may occur and cause weaning failure.

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