

# CORRELATION OF INFERIOR VENA CAVA COLLAPSIBILITY INDEX WITH LUNG ULTRASOUND AND STROKE VOLUME VARIABILITY IN HYPOTENSIVE CRITICAL CARE PATIENTS

By

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## ABSTRACT

**Background:** Differentiating fluid responders from non-responders is the primary goal when assessing critical care hypotensive patients for fluid responsiveness.

**Objectives:** To assess the fluid responsiveness in critical care hypotensive patients using inferior vena cava (IVC) collapsibility index and correlating its effectiveness with lung ultrasound (US) and stroke volume variation (SVV) induced by passive leg raising (PLR) in prediction of fluid responsiveness.

**Patients and Methods:** After approval of scientific and ethical committees, One hundred critical care hypotensive patients who were admitted to the ICU of El-Hussein University Hospital from November 2018 to March 2021 were included in the study. The following were done: echocardiography on admission, routine hemodynamic monitoring, lung US for assessment for extra volume lung water (EVLW), assessment of IVC variability, assessment of SVV induced by passive leg raising. Patients were classified into fluid responders and non-responders based on SVV. Responders were patients with  $SVV \geq 12\%$ .

**Results:** Caval index for assessment of fluid responsiveness is strongly correlated with lung US for assessment of EVLW and SVV with highest sensitivity and specificity in mechanically ventilated patients on muscle relaxant, and lower sensitivity and specificity in spontaneously breathing patients.

**Conclusion:** Caval index can be used to predict fluid responsiveness, but with different values depending on mechanical ventilation status and use of muscle relaxation.

**Keywords:** Inferior Vena Cave collapsibility index, Lung ultrasound, Stroke Volume Variation, Passive Leg Raising (PLR), Fluid responsiveness.

## INTRODUCTION

Fluid resuscitation of patients with acute circulatory failure aims to increase stroke volume (SV) and consequently improve cardiac output (CO) for better tissue oxygenation. However, this effect

does not always occur. The evaluation of fluid responsiveness before their administration may help to identify patients who would benefit from fluid resuscitation and avoid the risk of fluid overload in the others. The dynamic parameters of fluid responsiveness

evaluation are promising predictive factors. Of these, the echocardiographic measurement of the respiratory variation in the IVC diameter is easy to apply and has been used in the hemodynamic evaluation of intensive care patients (ICU) patients. However, the applicability of this technique has many limitations, and the present studies are heterogeneous and inconsistent across specific groups of patients. Assessment of the IVC using trans-thoracic echocardiography (TTE) is a conventional element of the echocardiographic study of critical patients. The physiological principle behind it is the lung-heart interaction. The variation in transpulmonary pressure during respiration is transmitted to the right heart cavities, which varies the venous return and the IVC diameter. This relationship depends on the ventilatory mode and IVC compliance of the patient (*Furtado and Reis, 2019*).

In non-ventilated patients or those under invasive mechanical ventilation (IMV) with respiratory effort, there is a negative transpulmonary pressure at the beginning of inspiration that induces a variable degree of IVC collapse as a function of its compliance. For example, in patients with high right heart cavity pressure or elevated preload (during the flat phase of the Frank-Starling curve), IVC shows reduced compliance and limited collapse due to the negative transpulmonary pressure transmitted; in fact, collapse may be absent. Among patients with low right heart cavity pressure in hypovolemia (i.e., the ascending phase of the Frank-Starling curve), IVC compliance is high, and collapse is significant during inspiration. By contrast, positive pressure can be

applied to the thorax during inspiration among patients under IMV without respiratory effort (in the controlled mode). This pressure is transmitted to the right heart cavities and the IVC, which stretches as a function of its compliance. Among patients without cardiac reserve due to poor cardiac function and/or those with high preload (i.e., during the flat phase of the Frank-Starling curve), the IVC shows reduced compliance and limited distention, and its diameter may not vary. Conversely, the IVC of patients with cardiac reserve who potentially benefit from the administration of fluids shows significant distension during inspiration (*Funk et al., 2013, Lansdorp et al., 2014 and Widmaier et al., 2016*).

**We aimed by our study to** evaluate the correlation of IVC variability index with lung US and SVV in assessment of fluid responsiveness and fluid tolerance in critical care hypotensive patients.

## PATIENTS AND METHODS

This prospective study was carried out on 100 adult patients who were admitted to the ICU of El-Hussein University Hospital from November 2018 to March 2021.

**Inclusion criteria:** All hypotensive critical care patients, both sexes and hypotension was defined as mean arterial pressure (MAP) less than 65 mm Hg or systolic blood pressure (SBP) less than 90 mm Hg.

**Exclusion criteria:** Patient's refusal, uncooperative patients, patients with poor echo window patients with valvular heart disease, patients with atrial fibrillation, patients with increased intra-abdominal pressure, patients in whom the supine

position was contraindicated, contraindication to PLR including hip or spine surgery, post abdominal surgery.

**Ethical approval:** was obtained from the Faculty of Medicine Al-Azhar University Research/Ethics Committee on 8/10/2018.

Informed consent was obtained from all patients or from their relatives and they received the fullest possible information about the study.

All examinations were performed using an echocardiograph (Philips Affiniti 50, USA) with S4-2 Cardiac sector probe.

**The following were done:** Complete history taking, complete physical examination, laboratory investigation and arterial blood gases measurements as well as echocardiography and routine hemodynamic monitoring.

**Pulmonary Assessment for EVLW:** Lung US was performed according to a systematic protocol in supine patients. An increased amount of EVLW was diagnosed by multiple B-lines or comet tails. B-lines were defined as discrete laser-like vertical hyperechoic reverberation artifacts that arise from the pleural line and extended to the bottom of the screen without fading, and moved synchronously with lung sliding. They represented a reverberation artifact through edematous interlobular septa or alveoli (Volpicelli *et al.*, 2012). The echo comet score (ECS) was obtained by the 28-rib interspaces technique, which divided the chest wall into 12 areas on the left (from the 2nd to the 4th intercostal space) and 16 areas on the right (from the 2nd to the 5th intercostal space) anterior and lateral hemithorax (Volpicelli *et al.*, 2012, Zieleskiewicz *et al.*, 2014, and

Bouhemad *et al.*, 2015). The sum of the B-lines found on each of the 28 chest-wall areas yields the ECS, denoting the amount of EVLW.

**Assessment of Inferior Vena Cava Variability:** The patient was maintained in the supine position then the IVC was evaluated in the subcostal (SIVC) view. The IVC was first identified in a transverse plane, with the probe in a subxiphoid position perpendicular to the skin. Then the probe was moved to the right to visualize the IVC in the center of the field. The probe was then rotated by 90° to obtain a longitudinal plane. The in-plane view of the probe showed the IVC in its longitudinal axis draining into the right atrium. The right hepatic vein, the last tributary to join the IVC intra-abdominally was visualized in this view (De Backer and Fagnoul, 2014, Evans *et al.*, 2014, Lang *et al.*, 2015, and Levitov *et al.*, 2016). After confirming the IVC using the PWD mode, the M mode was used to select a plane just distal to the right hepatic vein, approximately 2–3 cm from the junction of the IVC and right atrium. This was to make sure that the IVC caliber was measured intra-abdominally avoiding the intrathoracic region and also for standardization. M mode was used to capture a 10-s cine loop of the IVC over two or three respiratory cycles (De Backer and Fagnoul, 2014, Evans *et al.*, 2014, Lang *et al.*, 2015 and Levitov *et al.*, 2016). Then the maximum IVC diameter (Dmax) and minimum IVC diameter (Dmin) were measured. Three measurements were averaged.

**Caval index (dIVC) was calculated as** 
$$[(D_{\text{max}} - D_{\text{min}}) / ((D_{\text{max}} + D_{\text{min}})/2)] \times 100\%.$$

The caval index was expressed as a percentage, where one end of the spectrum was 0% which indicated minimal collapse of the IVC meaning volume overload and the other end of the spectrum was 100% which indicates almost complete collapse of the IVC meaning volume depletion.

**Assessment of Stroke Volume Variability:** SVV induced by passive leg rising was assessed at the left ventricular outflow tract (LVOT). LVOT diameter first was measured in the parasternal long axis view (PLAX) view and hence LVOT area i.e., cross sectional area (CSA), then LVOT velocity time integral (VTI) was measured using pulsed wave Doppler PWD) in the apical five chamber (AP5) view, while the patient in a semi recumbent position 30°. SV was calculated as the product of the CSA of the LVOT and the LVOT VTI (*Poth et al., 2014*). PLR test was done by elevating the patient lower limbs to 45°(automatic bed elevation or wedge pillow) while at the same time placing the patient in the supine from a 30° semi recumbent position. After a minute of equilibration, while the transducer is in the same position, LVOT VTI was repeated and SV recalculated.

**SVV was calculated as  $[(SV_{max} - SV_{min}) / ((SV_{max} + SV_{min})/2)] \times 100\%$ .**

Patients were classified into fluid responders and non-responders based on SVV. Responders are patients with  $SVV \geq 12\%$  (*Monnet et al., 2011, Brun et al.,*

*2013, Vos et al., 2015, Monnet et al., 2016, Teboul et al., 2016 and Miller and Mandeville, 2016*).

Vital data were recorded before and after PLR test.

**Statistical analysis:** Recorded data were analysed using the statistical package for the social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean $\pm$  standard deviation (SD). Qualitative data were expressed as frequency and percentage.

**The following tests were done:** Independent-samples t-test of significance was used when comparing between two means. Mann Whitney (z) test: for two-group comparisons in non-parametric data. Chi-square ( $\chi^2$ ) test of significance was used in order to compare proportions between qualitative parameters. Pearson's correlation coefficient (r) test was used to assess the degree of association between two sets of variables. Receiver operating characteristic (ROC curve) analysis was used to find out the overall predictivity of parameter in and to find out the best cut-off value with detection of sensitivity and specificity at this cut-off value. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant at  $< 0.05$ .

## RESULTS

We aimed by our study to evaluate the correlation of IVC variability index with lung US and SVV in assessment of fluid responsiveness and fluid tolerance in critical care hypotensive patients and we

applied our study on 100 patients with the same inclusion and exclusion criteria.

According to demographic data (age and sex), there was no statistically significant difference between fluid responders and non-responders (**Table 1**).

**Table (1): Comparison between fluid responder and non-responder according to their demographic data**

Fluid responsiveness Demographic data	Responders (n=67)	Non-responders (n=33)	Test	p-value
<b>Age (years)</b>				
Mean±SD	49.12±13.64	49.73±13.31		
Range	19-69	24-68	t=0.045	0.833
<b>Sex</b>				
Male	39 (58.2%)	21 (63.6%)		
Female	28 (41.8%)	12 (36.4%)	x <sup>2</sup> =0.271	0.602

According to the use of mechanical ventilation and muscle relaxation, there was no statistically significant difference

between fluid responders and non-responders (**Table 2**).

**Table (2): Comparison between fluid responder and non-responder according to their mechanical ventilation and muscle relaxantion**

Fluid responsiveness Mechanical ventilation	Responders (n=67)	Non-responders (n=33)	x <sup>2</sup>	p-value
<b>Mechanical ventilation</b>				
Ventilated	43 (64.2%)	24 (72.7%)		
Not ventilated	24 (35.8%)	9 (27.3%)	0.731	0.393
<b>Muscle relaxation</b>				
Yes	30/43 (69.8%)	19/24 (79.2%)		
No	13/43 (30.2%)	5/24 (20.8%)	0.693	0.405

No statistically significant difference between fluid-responders and non-responders according to their blood pressure before & after PLR test, but for the changes in blood pressure with PLR, there were differences between pre and post SBP, DBP and MAP in fluid responder group with a mean of

$6.28 \pm 1.95$ ,  $6.04 \pm 1.47$  and  $6.36 \pm 1.07$  respectively and a mean of  $0.00 \pm 2.66$ ,  $0.79 \pm 1.93$  and  $0.73 \pm 1.40$  respectively in fluid non-responder group. There was highly statistically significant higher mean value in fluid responders compared to non-responders with ( $p$ -value  $<0.001$  highly significant) (Table 3).

**Table (3): Comparison between fluid responder and not responder according to their blood pressure**

Fluid responsiveness Blood pressure	Responders (n=67)	Non-responders (n=33)	Test value	p-value
<b>SBP before PLR</b>				
Mean±SD	$90.76 \pm 9.38$	$93.24 \pm 7.94$	t=1.706	0.195
Range	75-110	77-106		
<b>DBP before PLR</b>				
Mean±SD	$51.70 \pm 8.96$	$54.33 \pm 9.09$	t=1.889	0.172
Range	31-67	31-69		
<b>MAP before PLR</b>				
Mean±SD	$64.55 \pm 8.38$	$67.12 \pm 8.17$	t=2.113	0.149
Range	48-81	47-79		
<b>SBP after PLR</b>				
Mean±SD	$97.04 \pm 9.57$	$93.24 \pm 7.85$	t=3.907	0.051
Range	78-117	75-108		
<b>DBP after PLR</b>				
Mean±SD	$57.75 \pm 9.46$	$55.12 \pm 9.85$	t=1.658	0.201
Range	37-76	30-74		
<b>MAP after PLR</b>				
Mean±SD	$70.91 \pm 8.81$	$67.85 \pm 8.55$	t=2.723	0.102
Range	53-89	48-82		
<b>SBP change with PLR</b>				
Mean±SD	$6.28 \pm 1.95$	$0.00 \pm 2.66$	z=65.673	<0.001
Range	0 - 10	-9 - 5		
<b>DBP change with PLR</b>				
Mean±SD	$6.04 \pm 1.47$	$0.79 \pm 1.93$	z=23.418	<0.001
Range	4 - 9	-2 - 7		
<b>MAP change with PLR</b>				
Mean±SD	$6.36 \pm 1.07$	$0.73 \pm 1.40$	z=71.565	<0.001
Range	4 - 8	-2 - 4		

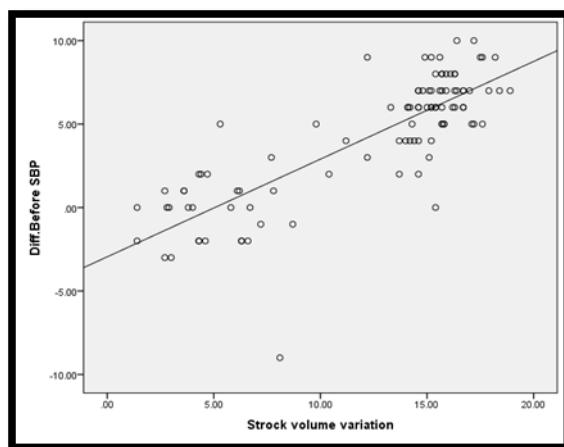
There was statistically significant positive correlation between SVV and blood pressure changes with PLR test

(Difference between blood pressure before & after PLR test) (**Table 4**).

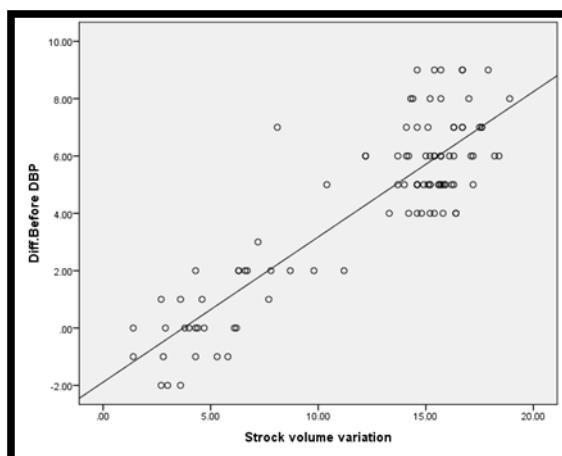
**Table (4): Correlation between patients SVV and blood pressure changes using Pearson Correlation Coefficient**

Parameters	SVV	
	r	p-value
SBP change with PLR	0.814	<0.001
DBP change with PLR	0.876	<0.001
MAP change with PLR	0.949	<0.001

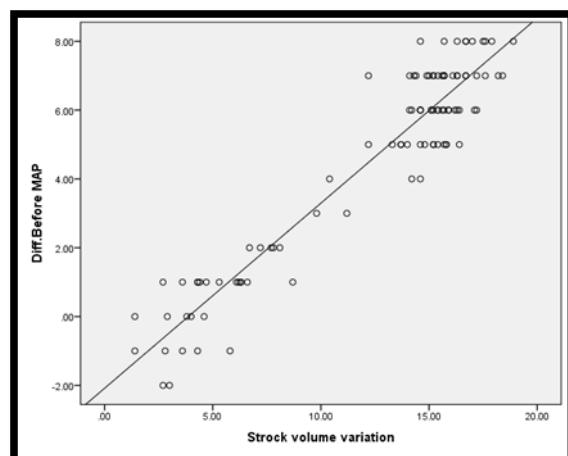
r-Pearson Correlation Coefficient



**Figure (1): Statistically significant positive correlation between SVV and SBP change with PLR test (Difference between SBP before & after PLR test).**



**Figure (2): Statistically significant positive correlation between SVV and DBP change with PLR test (Difference between DBP before & after PLR test).**



**Figure (3): Statistically significant positive correlation between SVV and MAP change with PLR test (Difference between SBP before & after PLR test).**

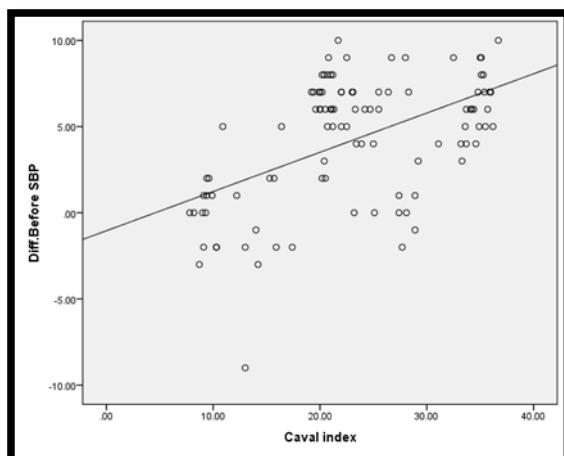
There was statistically significant positive correlation between caval index and blood pressure changes with PLR test

(Difference between blood pressure before & after PLR test) (**Table 5**).

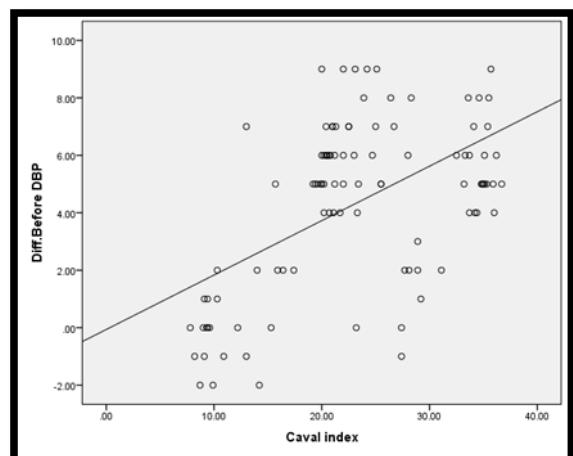
**Table (5): Correlation between patients caval index and blood pressure changes using Pearson Correlation Coefficient**

Parameters	SVV	
	r	p-value
SBP change with PLR	0.520	<0.001
DBP change with PLR	0.538	<0.001
MAP change with PLR	0.596	<0.001

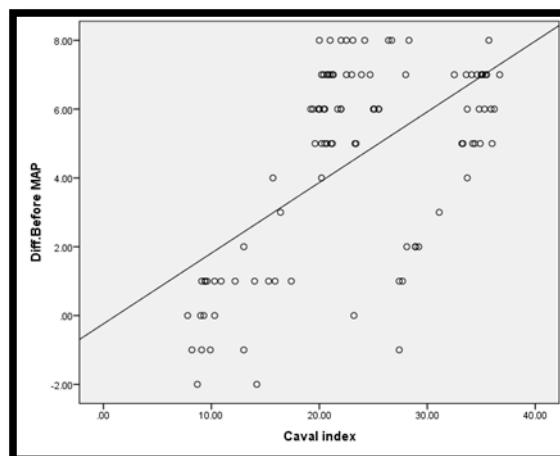
r-Pearson Correlation Coefficient



**Figure (4): Statistically significant positive correlation between Caval Index and SBP change with PLR test (Difference between SBP before & after PLR test)**



**Figure (5): Statistically significant positive correlation between Caval Index and DBP change with PLR test (Difference between DBP before & after PLR test)**



**Figure (6): Statistically significant Positive correlation between Caval Index and MAP change with PLR test (Difference between MAP before & after PLR test).**

The Caval Index in fluid responder group ranged from 0.97-2.111 (mean  $1.53 \pm 0.26$ ). While the Caval Index in fluid non-responder group ranged from 1.78-2.26 (mean  $1.96 \pm 0.14$ ), there was highly

statistically significant higher mean value in fluid responder compared to non-responder with ( $p$ -value  $<0.001$  highly significant) (Table 6).

**Table (6): Comparison between fluid responders and non-responders according to their IVC parameters**

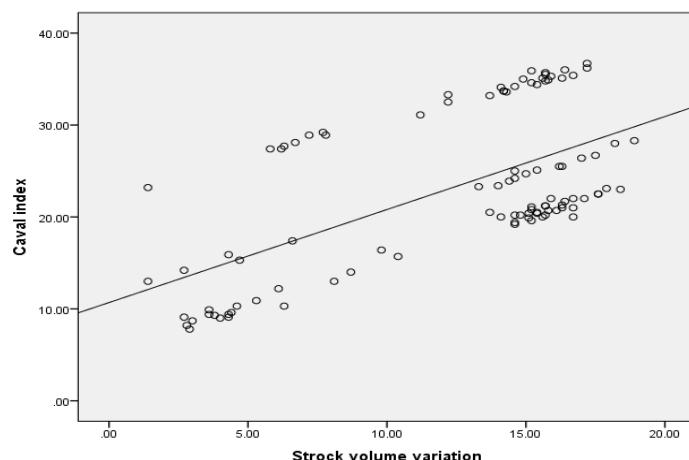
IVC Fluid responsiveness	Responders (n=67)	Non-responders (n=33)	Test value	p-value
<b>D<sub>max</sub></b>				
Mean±SD	1.53±0.26	1.96±0.14	t=78.060	<0.001
Range	0.97-2.11	1.78-2.26		
<b>D<sub>min</sub></b>				
Mean±SD	1.17±0.18	1.67±0.08	t=226.877	<0.001
Range	0.77-1.52	1.51-1.8		
<b>Caval index</b>				
Mean±SD	26.53±6.31	16.06±7.90	z=37.410	<0.001
Range	19.2-36.7	7.8-31.1		

There was statistically significant positive correlation between patients SVV and their Caval index (Table 7).

**Table (7): Correlation between patients SVV and IVC parameters using Pearson Correlation Coefficient**

IVC parameters	SVV	
	r	p-value
D <sub>max</sub>	-0.752	<0.001
D <sub>min</sub>	-0.924	<0.001
Caval index	0.616	<0.001

r-Pearson Correlation Coefficient



**Figure (7): Statistically significant positive correlation between SVV and Caval Index**

The ECS (No. of B lines) in fluid responder group was ranged from 2-20 (mean  $11.51 \pm 3.92$ ), while in the non-responder group it was ranged from 25-58

(mean  $41.15 \pm 8.99$ ), there was highly statistically significant lower mean value in fluid responder compared to non-responder with ( $p$ -value  $<0.001$ ) (Table 8).

**Table (8): Comparison between fluid responders and non-responders according to their EVLW**

Lung US Fluid responsiveness	Responders (n=67)	Non-responders (n=33)	Test	p-value
N. of B lines				
Mean $\pm$ SD	$11.51 \pm 3.92$	$41.15 \pm 8.99$		
Range	2-20	25-58	$z=24.125$	$<0.001$

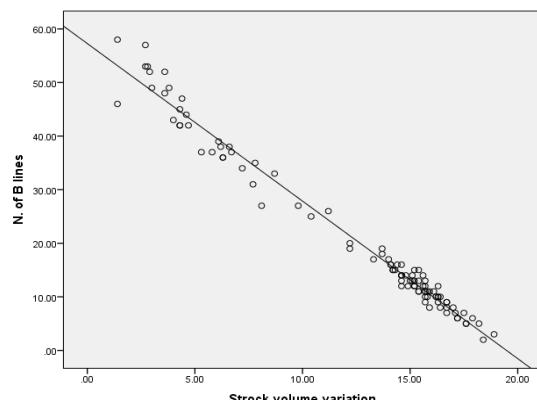
There was statistically significant correlation between SVV and echo comet

score (number of B lines) for detection of EVLW (Table 9).

**Table (9): Correlation between patients SVV and EVLW using Pearson Correlation Coefficient**

EVLW	SVV	
	r	p-value
N. of B lines	-0.990	$<0.001$

r-Pearson Correlation Coefficient



**Figure (8): Statistically significant negative correlation between SVV and N. of B lines.**

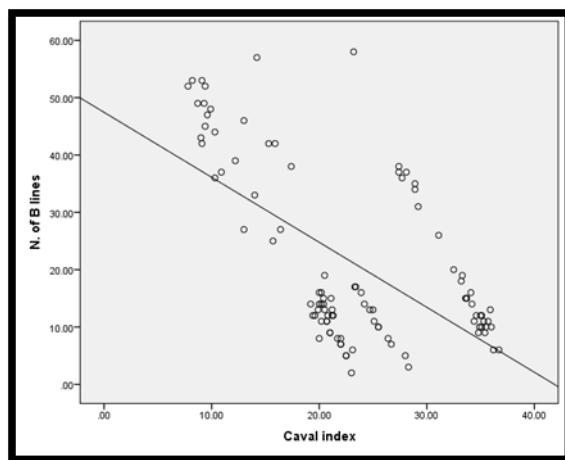
There was statistically significant correlation between caval index and echo

comet score (number of B lines) for detection of EVLW (Table 10).

**Table (10): Correlation between patients Caval index and EVLW using Pearson Correlation Coefficient**

EVLW	Caval index	
	r	p-value
N. of B lines	-0.627	$<0.001$
Semiquantitative score	-0.513	$<0.001$

r-Pearson Correlation Coefficient

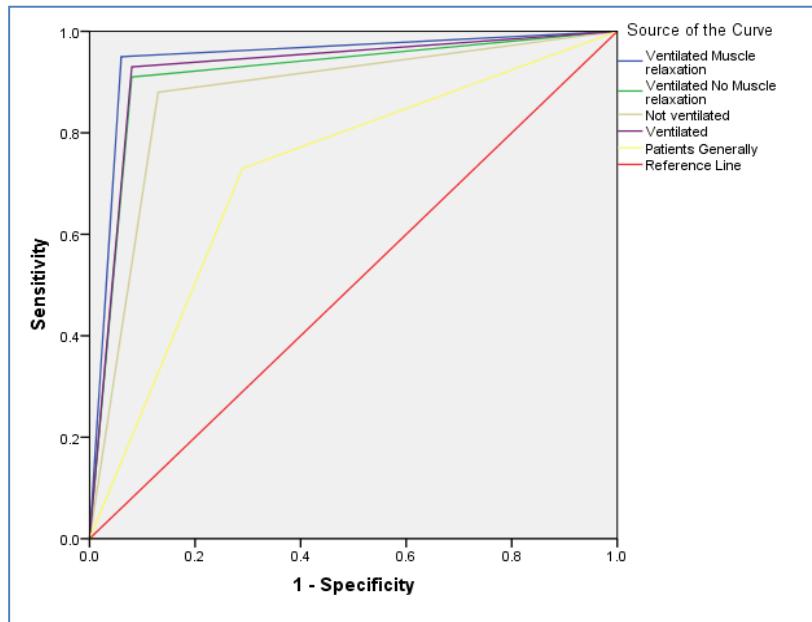


**Figure (9): Statistically significant Negative correlation between Caval Index and N. of B lines.**

#### Prediction of fluid responsiveness using the Caval Index:

Receiver operator characteristics (ROC) curves were constructed for Caval Index indices for prediction of fluid responsiveness in different patient's

categories. In all patient categories it was significant predictor as denoted by the significantly large area under the curves (AUCs); with Caval Index in ventilated patients with muscle relaxant being the most significant predictor (**Figure 10**).



**Figure (10): Receiver-operating characteristic (ROC) curve for prediction of fluid responsiveness using the Caval Index**

There were different values of Caval index for prediction of fluid responsiveness depending on mode of

ventilation and use of muscle relaxation as shown in (**Table 11**).

**Table (11): Caval index value for prediction of fluid responsiveness**

<b>Patient category</b>	<b>Ventilated patients</b>			<b>Not ventilated</b>	<b>All</b>
	<b>With muscle relaxant</b>	<b>Without muscle relaxant</b>	<b>All</b>		
<b>Cut-off value by Caval index</b>	$\geq 16.4$	$\geq 17.4$	$\geq 17.4$	$\geq 31.1$	$\geq 20.2$
<b>Sensitivity</b>	95%	91%	93%	88%	73%
<b>Specificity</b>	94%	92%	92%	87%	71%
<b>PPV</b>	94.1%	91.9%	92.1%	87.1%	71.6%
<b>NPV</b>	94.9%	91.1%	92.9%	87.9%	72.4%
<b>Accuracy</b>	94.5%	91.5%	92.5%	87.5%	72.0%

## DISCUSSION

As to come to our knowledge, this was the first study to evaluate the effectiveness of IVC variability index for assessment of fluid responsiveness in correlation with lung US and SVV using PLR test.

Patients were classified into fluid responders and non-responders based on SVV. Responders are patients with  $SVV \geq 12\%$  (*Monnet et al., 2011, Brun et al., 2013, Vos et al., 2015, Monnet et al., 2016, Teboul et al., 2016* and *Miller and Mandeville, 2016*).

There were 67 (67%) patients with  $SVV \geq 12\%$  and hence they are fluid responders and 33 (33%) patients with  $SVV < 12$  (fluid non-responder). Of the fluid responder group there were 43 patients mechanically ventilated (40 mechanically ventilated with muscle relaxant and 13 without muscle relaxant) and 24 spontaneously breathing patients; while in the non-responder group there were 24 patients mechanically ventilated (19 mechanically ventilated with muscle relaxant and 5 without muscle relaxant) and 9 spontaneously breathing patients.

There was strong positive correlation between SVV and Caval index in both responders and non-responders with highly significant p-value  $<0.001$ . Also, there was strong positive correlation between Caval index and EVLW in both responders and non-responders with highly significant p-value  $<0.001$  and blood pressure changes with PLR test in both responders and non-responders with highly significant p-value  $<0.001$  with highly significant p-value  $<0.001$ .

There were different values of Caval index for prediction of fluid responsiveness depending on mode of ventilation and use of muscle relaxation:

In mechanically ventilated patients with muscle relaxation: Caval index  $\geq 16.4\%$  is the value for prediction of fluid responsiveness with 95% sensitivity, 94% specificity, 94.1 positive predictive values and 94.9 negative predictive values. Other studies showed that a distensibility index ( $D_{max} - D_{min}/D_{min}$ ) of  $> 19.4\%$  is the value for prediction of fluid responsiveness with 79.17% sensitivity and 80% specificity with an AUC of 0.886 (*Aboelnile et al., 2020*).

In a systematic review and meta-analysis done by *Long et al.* (*Long et al., 2017*), they found that pooled results of dIVC in mechanically ventilated patients had AUC of 0.79 with a sensitivity of 67% and a specificity of 68%, which is not in line with our results.

In another recent systematic review and meta-analysis done by *Huang et al.* (*Huang et al., 2018*), dIVC has better performance in mechanically ventilated shocked patients with a pooled AUC of 0.82 (95% CI 0.79–0.85) with a specificity of 80% and a sensitivity of 69%.

Additional studies had less consistent results, showing discriminatory powers of AUC = 0.43 (95%CI 0.25 - 0.61) and AUC = 0.69 (95%CI 0.48 - 0.89), respectively (*Charbonneau et al., 2014*) (*Theerawit et al., 2016*).

One potential explanation of this discrepancy compared with previous studies is related to the fact that Charbonneau et al. found a higher percentage of patients receiving laparotomy 23% which might have conditioned the accuracy of the test casting doubts about its use among patients undergoing abdominal surgery (*Charbonneau et al., 2014*). In the case of *Theerawit et al.*, (*Theerawit et al., 2016*) patients with severe sepsis were included, who might have increased intra-abdominal pressure in that context. Intra-abdominal pressure was not monitored and may have biased the results.

Many factors can affect dIVC measurements and cause this difference between studies as respiratory compliance (*Lakhal et al., 2011*), and factors affecting intra-abdominal pressure (*Bendjelid &*

*Romand, 2012*) and (*Santa-Teresa et al., 2012*).

**In mechanically ventilated patients without muscle relaxant:** Caval index  $\geq 17.4\%$  is the value for prediction of fluid responsiveness with 91% sensitivity, 92% specificity, 91.9 positive predictive values and 91.1 negative predictive value.

**In all ventilated patients:** Caval index  $\geq 17.4\%$  is the value for prediction of fluid responsiveness with 93% sensitivity, 92% specificity, 91.1 positive predictive value and 92.9 negative predictive value

**In spontaneously breathing patients:** Caval index  $\geq 31.1\%$  is the value for prediction of fluid responsiveness with 88% sensitivity, 87% specificity, 87.1 positive predictive values and 87.9 negative predictive value.

Other studies have shown that a collapsability index (Dmax - Dmin/Dmax) of  $> 40\%$  among 40 non-ventilated patients with hemorrhagic, hypovolemic or septic shock had a specificity of 80% and a sensitivity of 70%, with an AUC of 0.77 (95%CI 0.60 - 0.88); however, the test was not reliable concerning these patients because the lower limit of the 95%CI of the AUC was  $< 0.75$ . An IVC collapsibility index below 40% does not allow us to exclude fluid responsiveness, and the probability of response increases when the index is above 40% (*Muller et al., 2012*).

*Airapetian et al.* (*Airapetian et al., 2015*) found similar results among 59 non-intubated, non-ventilated patients, in which a collapsibility index (Dmax - Dmin/Dmax) of  $> 42\%$  had a specificity of 97% and a positive predictive value of 90% but low sensitivity and negative

predictive values, with an AUC of 0.62 (95%CI 0.49 - 0.74).

In all patients (mechanically ventilated and spontaneously breathing): Caval index  $\geq 20.2\%$  is the value for prediction of fluid responsiveness with 73% sensitivity, 71% specificity, 71.6 positive predictive value and 72.4 negative predictive value.

In a 2014 meta-analysis of eight studies including 235 patients, either non-ventilated or under IMV, the combined sensitivity was 76% (95% CI = 61 - 86) and the specificity was 86% (95%CI 69 - 95). The combined AUC was 0.84 (95%CI 0.79 - 0.89). The discriminatory value of IVC variation ranged between 12 and 40% across these studies. Of the patients under IMV, better sensitivity (81%; 95%CI 67 - 91) was found for similar specificity (87%; 95%CI 63 - 97) (*Zhang et al., 2014*).

In a 2017 systematic review and meta-analysis (*Long et al., 2017*) of 17 studies including 533 patients with circulatory failure, the combined sensitivity and specificity values of the IVC variation index to predict fluid responsiveness were 63% (95%CI 56 - 69) and 73% (95%CI 67 - 78), respectively, with a combined AUC of 0.79 (standard error = 0.05). The subgroup of ventilated patients (combined sensitivity = 67% [95% CI = 58 - 75]; specificity = 68% [95%CI 60 - 76]) presented with better results than non-ventilated patients (combined sensitivity = 52% [95%CI 42 - 62]; specificity = 77% [95%CI 68 - 84]).

## CONCLUSION

Fluid therapy increases CO in only approximately two third of patients with acute circulatory failure. Ideally, patients

with acute circulatory failure should be evaluated with regard to fluid responsiveness before its administration to avoid deleterious effects. In intensive care units, the use of IVC variation measured by TTE may play a role in this evaluation; however, it is necessary to guarantee the conditions under which the technique is validated and to consider its limitations, depending on the clinical context, for correct interpretation.

Our study concludes that Caval index is strongly correlated with lung US for assessment of EVLW and SVV and it can be used to predict fluid responsiveness, but with different values depending on mechanical ventilation status and use of muscle relaxation.

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**إرتباط مؤشر الوريد الأجوف السفلي مع الموجات الصوتية للرئة ومعدل اختلاف حجم نفحة القلب في مرضى الرعاية الحرجة الذين يعانون من انخفاض ضغط الدم**

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**خلفية البحث:** تقييم حالة التحميل المسبق للقلب هو الهدف الأساسي قبل إعطاء أي سوائل لمرضى العناية الحرجة الذين يعانون من انخفاض ضغط الدم وذلك للتمييز بين المستجيبين للسوائل وغير المستجيبين.

**الهدف من البحث:** تقييم حالة التحميل المسبق للقلب بإستخدام مؤشر الوريد الأجوف السفلي وتقدير مدى ارتباطه مع الموجات الصوتية للرئة ومعدل اختلاف حجم نفحة القلب في مرضى الرعاية الحرجة الذين يعانون من انخفاض ضغط الدم في مدى استجابتهم وتحملهم للسوائل

**المرضى وطرق البحث:** بعد موافقة اللجان العلمية والأخلاقية لجامعة الأزهر تم اختيار مائة من مرضى العناية الحرجة الذين يعانون من انخفاض ضغط الدم، ثم تم عمل التالي أخذ التاريخ المرضي الكامل للمرضى والفحص البدني الكامل والتحاليل المخبرية وغازات الدم الشريانية وخطيط صدى القلب عند الدخول للعناية المركزية و المتابعة الروتينية للعلامات الحيوية والموجات الصوتية للرئة لتقدير كمية ترشح المياه الزائد و قياس مؤشر الوريد الأجوف السفلي و قياس معدل اختلاف حجم نفحة القلب الناجم عن اختبار رفع الساق السلبي و تم تسجيل العلامات الحيوية قبل وبعد اختبار رفع الساق السلبي. تم تقسيم المرضى إلى مستجيبين للسوائل وغير مستجيبين بناءً على معدل اختلاف حجم نفحة القلب حيث أن المستجيبون هم المرضى الذين لديهم معدل اختلاف حجم نفحة القلب بنسبة  $\geq 12\%$ .

**نتائج البحث:** كشفت نتائج هذه الدراسة أنه يمكن إستخدام مؤشر الوريد الأجوف السفلي لتقييم حالة التحميل المسبق للقلب بحساسية وخصوصية عالية في

المرضى الخاضعين للتهوية الميكانيكية باستخدام مرخى العضلات وبحساسـه وخصوصـية أقل فـى المرضـى الذين يـتنفسـون تلقـائـياـ، كما إنـه يـرتبـط إـرتبـاطـاـ إـيجـابـياـ قـوىـ معـ مـعـدـلـ اختـلاـفـ حـجـمـ نـفـصـةـ القـلـبـ فـيـ كـلـاـ منـ المـسـتـجـيبـينـ وـغـيرـ المـسـتـجـيبـينـ، وـارـتـباطـ سـلـبـاـ قـوـيـاـ مـعـ كـمـيـةـ التـرـشـحـ الرـئـوىـ فـيـ كـلـ منـ المـسـتـجـيبـينـ وـغـيرـ المـسـتـجـيبـينـ.

**الاستنتاج:** يمكن استخدام مؤشر الطـى للوريد الأـجـوـفـ السـفـلـىـ لـتقـيـمـ حـالـةـ التـحـمـيلـ المـسـبـقـ لـلـقـلـبـ وـلـكـنـ بـتقـيـمـ مـخـالـفـهـ إـعـتـمـادـاـ عـلـىـ طـرـيقـةـ التـهـويـةـ وـاسـتـخـادـ مـرـخـىـ العـضـلـاتـ.

**الكلـماتـ الدـالـلـةـ:** مؤـشـرـ الطـىـ لـلـورـيدـ الـأـجـوـفـ السـفـلـىـ، الـمـوـجـاتـ الصـوـتـيـهـ لـلـرـئـةـ، مـعـدـلـ إـختـلاـفـ حـجـمـ نـفـصـةـ القـلـبـ، اـختـبـارـ رـفعـ السـاقـ السـلـبـىـ.