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ORIGINAL ARTICLE

Predicting and Measuring Fluid Responsiveness by Hemodynamic Indices Versus Transthoracic Echocardiography in Clinically Shocked Patients

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Background In recent decades, dynamic measures have emerged as a more effective method for assessing fluid responsiveness in patients with clinical 51 shock. Among various dynamic parameters, we chose transthoracic echocardiographic-based measurements of diversity in aortic blood flow and the inferior vena cava diameter.

Aim This study made to determine the agreement of between usual hemodynamic indices and transthoracic echo-measured variations in aortic blood flow and the inferior vena cava diameter upon limited bolus crystalloid infusion in assessing fluid responsiveness in patients with clinical shock.

Patients and Methods This study comprised 51 patients diagnosed with acute circulatory failure secondary to clinical shock. All patients underwent the standard transthoracic echocardiographic assessment of the aortic blood flow (using left ventricular outflow tract velocity time integral) and changes in inferior vena cava diameter during the respiratory cycle. These evaluations were conducted at the time of shock diagnosis and repeated after infusion of a 300mL crystalloid fluid bolus (over 15min). patients were excluded if they had any of these: aortic or mitral stenosis, malignant atrial arrythmia, pregnant ladies, burned patients more than 20% burn of body surface area.

Results A notable distinction was observed between the respondents and non-respondents in terms of aortic flow variation after 15min.

Conclusion Transthoracic echocardiography can be used as an accurate method for foretelling fluid responsiveness in patients with shock after a fluid challenge.

Keywords CVP, Fluid responders, LVOT VTI, Stroke.
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INTRODUCTION

Shock is characterized by the critical inability to provide sufficient oxygen to body tissues, often leading to life-threatening conditions. This can stem from reduced blood flow to tissues, insufficient oxygen saturation in the blood, or heightened tissue oxygen demands, all of which lead to diminished oxygenation and vital organ dysfunction[1].

Without proper intervention, shock leads to persistent dysfunction in multiple organs, causing damage to the vital body systems and potentially resulting in fatalities[2].

Shock is categorized into four primary categories based on its underlying reason: hypovolemic, cardiogenic, obstructive, and distributive[3].

The common clinical indicators/laboratory findings suggestive of shock include hypotension; rapid heartbeat; fast breathing; and altered mental status, characterized by obtundation, cold and clammy extremities, mottled skin, reduced urine output, metabolic acidosis, and elevated lactate levels[4].

Shock is typically diagnosed based on a set of symptoms, the results of physical assessments, and the findings of lab analyses. Due to the lack of sensitivity or specificity in many signs and symptoms of shock, various clinical decision-making tools have been devised to aid in the early detection of shock[5].

Echocardiography is non invasive and widely popular and available in most ICU settings so it could help to differentiate easily between fluid responders and non responders in patient with clinical shock.

Echocardiography provides a comprehensive hemodynamic assessment. Elevated right ventricle (RV) afterload can precipitate obstructive shock. The RV is responsive to both pressure and volume surges. Under a heightened RV afterload, the RV prolongs its systolic duration, causing RV pressure to exceed the left ventricle (LV) pressure at the end of systole[6].

The assessment of RV function is important in sepsis. Often, the RV function is compromised in patients with sepsis due to a combination of factors, such as diminished RV contractility and increased RV afterload (resulting from certain conditions such as acute respiratory distress syndrome (ARDS) and mechanical ventilation). In approximately one-fifth of patients, RV dysfunction emerges as the primary characteristic[7].

Additionally, it is crucial to thoroughly examine for the presence of obstruction in the dynamic left ventricular outflow tract (LVOT)[8].

PATIENTS AND METHODS

Sample size:

Using Raosoft's sample size calculator, with a 10% margin of error, 90% confidence level, population size of 200, and response distribution of 50%, the calculated sample size was 51 patients.

Patients:

This controlled clinical trial was conducted in 51 patients who were admitted to Department of Anesthesiology, Intensive care and Pain management, Ain Shams University between January 2020 and January 2022 (Figure 1).

The echocardiogram was performed by the same physician for all patients.

The study was approved by the Critical Care Medicine Department Ethics Committee of the Faculty of Medicine, Ain Shams University (approval number: FMASU MD 282/2018), and informed consent was obtained from all patients or their next of kin.

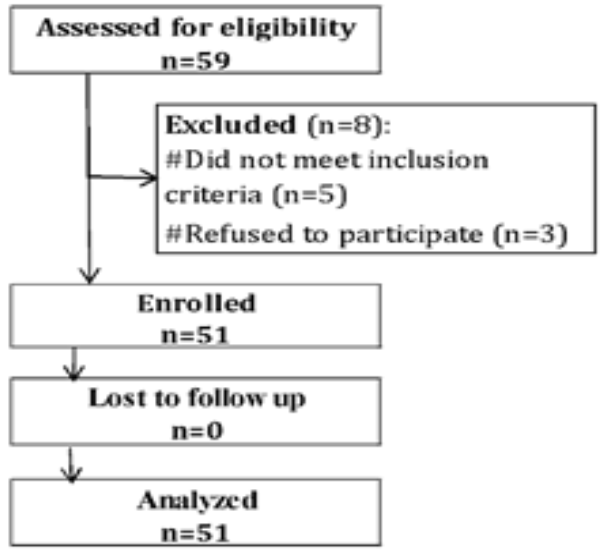

Fig. 1: Flow chart of the studied cases.

Any adult patient admitted to the critical care medicine department who meets all criteria for spontaneous breathing activity and exhibits at least any clinical sign of insufficient global perfusion, such as tachycardia (heart rate (HR) >100 beats per minute), delayed capillary refill, mottled skin, oliguria (urine output less than 0.5mL/kg per h for more than 2h), or a mean arterial pressure (MAP) of <60mmHg, may prompt the attending physician to consider fluid challenges.

Patients with aortic valve disease, mitral regurgitation more than grade 2, Mitral stenosis, tricuspid valve insufficiency of grade 3 or higher, tricuspid stenosis, atrial arrhythmias, RV infarction or failure, and unsatisfactory cardiac echogenicity; pregnant women; and patients with burns on >20% body surface area were excluded from the study.

Patient Characteristics:

Group diagnosis: sepsis or septic shock, Cardiogenic, acute respiratory failure, stroke, acute liver, acute renal failure, hypovolemic.

Methods:

A- Blood pressure readings

The systolic blood pressure (SBP) and MAP were assessed either invasively or non-invasively. The results were expressed in millimeters of mercury (mmHg).

B- Central venous pressure

Central venous pressure (CVP) was measured manually, with the results expressed in centimeters of water (cm H_2O). The zero level was identified as the cardiac apex.

C- Echocardiographic measurements

An echocardiographic assessment was conducted by the same operator utilizing a transthoracic ultrasound device, specifically Siemens Acuson X 300. This device is equipped with a colored echocardiographic feature and employs a 3.5-MHz transducer.

The following measurements were recorded:

1- Stroke volume

The amount of blood ejected by the LV in a single contraction. The LV pumps out approximately two-thirds of its blood with each heartbeat. The normal SV in healthy adults ranges from 60 to 120mL. The SV (in mL) is calculated by multiplying the LVOT velocity time integral (VTI) by the LVOT cross-sectional area. Using parasternal long-axis image, the area of the LVOT was determined. LVOT diameter during systole should be determined before calculating the cross-sectional area. Both the noncoronary and right coronary cusps of the aortic valve should be measured at these locations. The area beneath the envelope of the pulsed-wave Doppler signal acquired at the level of the LVOT annulus was computed to determine the LVOT VTI in the coronal five-chamber view. The LVOT VTI value was calculated as the average of three consecutive measurements. LVOT area was calculated using the formula $\pi\times$ diameter $^2/4$. As the diameter of the aortic orifice was presumed to stay consistent in each patient, measurements were taken once at baseline.

2- Inferior cava diameter

The inferior cava (IVC) diameter was measured in the subcostal view. The end-expiratory values of the IVC diameter were assessed as the maximum diameter [in centimeters (cm)], and the values measured over three respiratory cycles were averaged. For each patient, a single measurement was performed at the beginning of the study.

Study protocol:

The Systolic arterial pressure (SAP), MAP, HR, CVP, LVOT VTI, and IVC diameter were measured at baseline with the patient placed in a semi-recumbent posture. These are the first readings (Reading 1). Finally, the SAP, MAP, HR, CVP, and LVOT VTI measurements were obtained within 15min after the administration of a 300mL fluid bolus (fluid challenge) (Reading 2). All volume challenges involved the introduction of crystalloid fluids. Each volume challenge was performed by the attending physician. A patient who exhibited an SV increase of 15% or higher following volume expansion was referred to as a responder. Patients were categorized as either fluid responders or non-responders through the assessment of static or dynamic parameters and the performance of echocardiography, taking into account the changes in SV and IVC collapsibility after fluid loading.

Statistical analysis:

The Statistical Package for the Social Sciences (version 20.0; SPSS Inc., Chicago, Illinois, USA) was used to analyze the recorded data. Quantitative data were expressed as the mean±standard deviation (SD), while qualitative data were expressed as frequencies and percentages.

The following tests were performed:

Paired samples *t*-tests were used to compare related samples, Wilcoxon Signed-Rank Sum tests for non-parametric data to compare the differences over time, independent samples *t*-tests for comparing the means between two groups, and Mann–Whitney *U* tests for two-group comparisons with non-parametric data. Chi-square (χ^2) tests were employed to balance the proportions among the qualitative parameters. Receiver operating characteristic curve interpretation was employed to analyze the general predictability of the parameters and determine the ideal cut-off value. The sensitivity and specificity at this threshold were also assessed.

The confidence interval was set at 95% with a margin of error of 5%. Therefore, a *p*-value was considered significant as one less than 0.05.

Statistical methods:

The collected data were coded, tabulated, and statistically analyzed using IBM SPSS statistics (Statistical Package for Social Sciences) software version 28.0, IBM Corp., Chicago, USA, 2021. Quantitative data described as mean±SD (standard deviation) and then compared using independent *t*-test. Qualitative data described as number and percentage and then compared using Chi square test and Fisher's Exact test for independence as well as Kappa test for agreement. Bonferoni test used for post hoc comparisons. ROC curve was used to evaluate the performance pre fluid measures. The level of significance was taken at *p*-value ≤0.050 was significant, otherwise was non-significant.

Diagnostic characteristics was calculated as follows:

- Sensitivity= (True positive test/Total positive golden) x100

- Specificity= (True negative test/Total negative golden) x100

• Predictive positive value= (True positive test/Total positive test)x100

• Predictive negative value= (True negative test/Total negative test)x100

RESULTS

The studied 51 cases had hemodynamic measures that revealed 27(52.9%) cases were fluid responders, and had transthoracic echocardiography that gave the same findings in the same cases (kappa for agreement= 1.000, *p*-value <0.001). The following results presented data in the whole cases and compared between responders and non-responders.

Table (1) showed that: Cardiac diagnoses were significantly more frequent in non-responders, while septic diagnoses were significantly more frequent in responders.

Table (2) showed that: Fluid responders had significant lower pre fluid lactate, CVP and IVC, significant higher pre fluid HR and SV, significant lower post fluid CVP and IVC, significant higher post fluid SBP, DBP, MBP, LVOT and SV, significant more elevation and elevation % SBP, DBP, MBP, CVP, IVC LVOT and SV, and significant more reduction and reduction % HR.

From both (Table 3; Figure 2) it showed that: Only pre fluid lactate, CVP, IVC and SV had significant diagnostic performance in predicting being a fluid responder. Lactate and SV had highest sensitivity, lactate had highest NPV, CVP had highest specificity, PPV and YI.

The IVC values (mean±SD) in the pre fluid and post fluid challenge were 3.38±3.05 and 3.39±3.34, respectively, showing a mean difference of 0.01 and a percentage change of 0.29%. No discernible change was found between the pre and post fluid challenge ($p>0.05$).

Table 1: Demographic characteristics among the studied cases and comparison according to fluid responsiveness:

Variables		All cases (Total= 51)	Response		*p*-value
			Responder (Total= 27)	Non responder (*N*= 24)	
Age (years)		63.5±8.6	61.6±9.3	65.8±7.4	^0.086
Sex	Male	28(54.9%)	16(59.3%)	12(50.0%)	#0.507
	Female	23(45.1%)	11(40.7%)	12(50.0%)	
Diagnosis	Cardiac	13(25.5%)	0(0.0%)a	13(54.2%)	§<0.001*
	Septic	11(21.6%)	9(33.3%)a	2(8.3%)b	
	Pulmonary	8(15.7%)	6(22.2%)a	2(8.3%)b	
	Brain	7(13.7%)	5(18.5%)a	2(8.3%)a	
	Liver	5(9.8%)	1(3.7%)a	4(16.7%)a	
	Others	7(13.7%)	6(22.2%)a	1(4.2%)a	

^: Independent *t*-test; #: Chi square test; §: Fishers Exact test; *: Significant (≤0.050); Homogenous diagnoses between responders and non-responders had the same symbol "a and b" based on post hoc Bonferroni test.

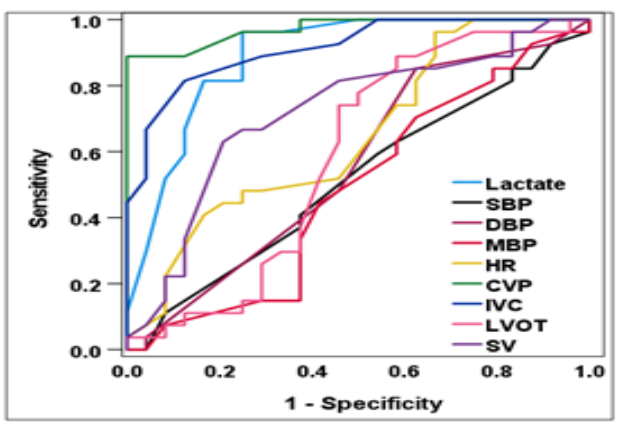

Fig. 2: Receiver operating characteristics (ROC) curve for pre fluid hemodynamic and transthoracic echocardiography parameters in predicting being a fluid responder.

The LVOT VTI values (mean±SD) in the pre fluid and post fluid challenge were 88.70±32.93 and 114.25±41.10, respectively, showing a mean difference of 25.55 and a percentage change of 30.83%. The mean value in the post fluid challenge was significantly higher than that in the pre fluid challenge ($p<0.001$).

The SV (mean±SD) in the pre-fluid and post-fluid challenge were 51.33±10.18 and 67.77±19.76, respectively, showing a mean difference of 16.44 and a change of 31.70%. The mean value in the post fluid challenge was significantly higher than that in the pre fluid challenge ($p<0.001$).

Multivariate analysis revealed that the significant predictors of response outcome were SBP, DBP, HR, CVP, IVC, LVOT VTI, and SV.

Table 2: Hemodynamic and transthoracic echocardiography parameters among the studied cases and comparison according to fluid responsiveness:

Variables		All cases (Total= 51)	Fluid responsiveness		^*p*-value
			Responder (Total= 27)	Non-responder (*N*= 24)	
Pre fluid lactate (mmol/L)		3.4±1.9	2.3±0.5	4.7±2.1	<0.001*
SBP (mmHg)	Pre fluid	74.8±8.4	74.8±8.2	74.8±8.8	0.991
	Post fluid	86.5±14.9	95.0±14.2	77.0±8.8	<0.001*
	Change	11.8±11.9	20.2±10.0	2.3±4.0	<0.001*
	Change %	15.9±15.6	27.1±12.7	3.2±5.7	<0.001*
DBP (mmHg)	Pre fluid	44.4±6.3	43.9±5.8	45.0±6.9	0.535
	Post fluid	49.4±7.8	52.9±7.4	45.4±6.3	<0.001*
	Change	5.0±7.4	9.0±7.3	0.4±4.1	<0.001*
	Change %	12.1±17.6	21.5±18.0	1.6±9.4	<0.001*
MBP (mmHg)	Pre fluid	54.4±6.5	54.1±6.1	54.8±7.1	0.691
	Post fluid	61.6±9.6	66.8±9.0	55.9±6.6	<0.001*
	Change	7.2±8.2	12.7±7.2	1.0±3.3	<0.001*
	Change %	13.6±15.5	23.8±14.0	2.1±6.1	<0.001*
HR (beat/min.)	Pre fluid	109.9±13.7	113.9±11.1	105.5±15.3	0.030*
	Post fluid	104.5±13.9	103.4±12.4	105.8±15.5	0.552
	Change	-5.4±9.4	-10.4±10.6	0.2±2.1	<0.001*
	Change %	-4.6±8.3	-9.0±9.4	0.2±1.8	<0.001*
CVP (cm H2O)	Pre fluid	8.9±6.2	4.1±2.9	14.2±4.5	<0.001*
	Post fluid	10.8±5.1	7.3±2.4	14.7±4.4	<0.001*
	Change	1.9±1.6	3.2±1.2	0.5±0.7	<0.001*
	Change %	71.5±121.0	130.8±142.7	4.7±7.1	<0.001*
IVC (cm)	Pre fluid	1.7±0.5	1.4±0.3	2.1±0.3	<0.001*
	Post fluid	1.9±0.3	1.8±0.2	2.1±0.3	<0.001*
	Change	0.2±0.3	0.3±0.3	0.0±0.1	<0.001*
	Change %	15.4±20.9	27.8±21.5	1.5±6.5	<0.001*
LVOT VTI (cm)	Pre fluid	88.7±32.9	88.6±31.2	88.8±35.5	0.987
	Post fluid	114.1±41.3	134.3±35.8	91.3±35.3	<0.001*
	Change	25.4±24.3	45.7±15.0	2.5±1.9	<0.001*
	Change %	30.5±29.9	54.7±20.5	3.3±2.3	<0.001*
SV (ml)	Pre fluid	47.4±22.3	53.6±10.9	40.5±29.2	0.035*
	Post fluid	67.8±19.8	82.3±14.0	51.4±10.0	<0.001*
	Change	20.4±22.2	28.7±7.9	11.0±28.8	0.007*
	Change %	23.3±54.3	55.0±15.5	-12.3±60.3	<0.001*

Data presented as Mean±SD; Change= post fluid - pre fluid; negative values indicate reduction; ^: Independent *t*-test; *: Significant (≤0.050).

This study made to evaluate the predictive accuracy of transthoracic echocardiographic recorded changes in ABF and IVC diameter with limited bolus fluid infusion in individuals experiencing clinical shock in terms of fluid responsiveness.

This prospective study enrolled 51 patients with acute circulatory failure admitted to the ICU. Each patient underwent a basic TTE assessment, which included the evaluation of ABF using the LVOT VTI as a surrogate. Additionally, the changes in IVC diameter (IVCD) within the respiratory cycle were assessed by measuring the IVCD min during inspiration and IVCD max during expiration. The ΔIVCD was calculated as follows: [(IVCDmax−IVCDmin)/IVCDmax]. Subsequently, a 300mL fluid bolus was administered over 15min, followed by TTE measurement of VTI and ΔIVCD to evaluate the response to fluid administration.

Patients were classified into two groups based on their response to fluid administration: responders and non-responders. The criterion for responsiveness was defined as a ≥15%–20% increase in SV following the infusion of 300mL of fluid.Of the 51 patients involved in the study, 27 were classified as responders and 24 as non-responders. More importantly, age, sex, height, and body weight did not differ significantly among responders and non-responders.

No significant differences were noticed between groups in terms of HR.

Meanwhile, significant differences were noticed between responders and non-responders in terms of the mean VTI, both at baseline and after the infusion of 300mL of fluid.

Table 3: Diagnostic performance and characteristics of pre fluid hemodynamic and transthoracic echocardiography parameters in predicting being a fluid responder:

Pre fluid measures	AUC	*p*-value	Cut point	Sensitivity	Specificity	PPV	NPV	YI
Lactate mmol/l	0.891	<0.001*	≤3.4	96.3%	75.0%	81.3%	94.7%	71.3%
SBP (mmgh)	0.513	0.873	≥74.0	59.3%	45.8%	3.1%	29.3%	5.1%
DBP mmgh	0.559	0.473	≥37.5	96.3%	4.2%	53.1%	50.0%	0.5%
MBP mmgh	0.515	0.858	≥50.7	70.4%	37.5%	55.9%	52.9%	7.9%
HR bpm	0.648	0.070	≥97.5	96.3%	33.3%	61.9%	88.9%	29.6%
CVP cm H_2O	0.972	<0.001*	≤7.5	88.9%	100.0%	100.0%	88.9%	88.9%
IVC (cm)	0.914	<0.001*	≤1.8	81.5%	87.5%	88.0%	80.8%	69.0%
LVOT (cm)	0.586	0.291	≥68.5	88.9%	41.7%	63.2%	76.9%	30.6%
SV (ml)	0.719	0.007*	≥53.5	96.3%	75.0%	75.0%	66.7%	41.7%

AUC: Area under curve; PPV: Positive predictive value; NPV: Negative predictive value; YI: Youdens index; *: Significant (≤0.050).

DISCUSSION

Shock is a severe and potentially fatal condition that causes widespread acute circulatory failure, which reduces the body's ability to use oxygen owing to inadequate oxygen delivery to cells[1].

In this study, we investigated whether the rapid administration of a small volume (300mL) of the normal saline could serve as a foreteller of fluid responsiveness.

Employing a low challenge volume theoretically limits the adverse effects of fluids in non-responders. Based on the Frank–Starling cardiac function curve, fluid responsiveness denotes a substantial increase in SV due to augmented cardiac preload. Additionally, owing to the shape of the curve, the boost in SV is theoretically more pronounced in the steep segment of the Frank–Starling curve, particularly at the onset (specifically, the initial 100mL) of the fluid challenge, particularly when the fluid administration rate increases.

A positive response to volume is characterized by a 15% elevation in cardiac output or cardiac index following a fluid load administered over 15–20min.

TTE express a simple and noninvasive measuring of SV via the calculation of the LVOT VTI and IVC diameter.

Measuring both the size and collapsibility of the IVC enables the identification of hypovolemic patients by estimating the right atrial pressure[9].

The rationale for utilizing aortic blood flow (ABF) variation as a foreteller of fluid responsiveness stems from the understanding that cardiac output results from the multiplication of the HR and SV. SV was determined by multiplying the subaortic VTI, which was recorded echocardiographically using pulse Doppler at the LVOT in the apical five-chamber view, by the subaortic LV area (SV= VTI×LVOT area).

The subaortic LVOT area is calculated using the formula πr^2, where R is the radius of the LVOT, equivalent to half its diameter measured via two-dimensional imaging. Given the assumption of a constant diameter of the LV outflow chamber diameter within a patient and minimal fluctuations in HR, variations in cardiac output are primarily attributed to variations in the VTI. Consequently, measuring the VTI and its variations directly correlates with changes in cardiac output, thus mitigating the potential error associated with measuring the diameter of the LVOT[10].

Inspiration induces negative intrathoracic pressure in normovolemic spontaneously breathing patients, leading to

a reduction in the IVC size. In hypovolemic individuals, this effect is exaggerated, resulting in increased IVC collapse during inspiration[11].

The subcostal window on transthoracic echocardiography provides a view of the IVC in the sagittal plane. M-mode imaging enables high-frame rate measurements of size changes all through the respiratory cycle, thereby facilitating accurate assessments[12].

Patients identified as likely to be fluid responsive, as indicated by an IVC collapse exceeding 50%, exhibited significant enhancements in the catheter-measured cardiac index, cardiac output, and MAP following fluid resuscitation[13].

In conclusion, a noticeable difference was observed between those who responded to fluid load and those who did not. A mean change was observed in ΔIVCD at baseline and after the infusion of 300mL of fluid.

In a study by Monnet *et al.*, ABF both pre and post fluid infusion (500mL normal saline over 10min), was monitored using esophageal Doppler monitoring equipment. ABF was monitored along with flow duration, and the difference between the lowest and maximal values during the respiratory cycle was computed. In 20 individuals (referred to as responders), the ABF increased by at least 15% following volume load. Prior to the administration of fluid, the responders (28±12%) exhibited greater breathing fluctuation in aortic flow compared with the non-responders (12±5%). In responders, it dramatically decreased (18%–11%) following volume augmentation.

A change in aortic flow of at least 18% predicted fluid responsiveness during respiration with 90% sensitivity and 94% specificity prior to volume expansion. Furthermore, in the responders and non-responders, the flow time increased following fluid infusion.

In patients with sinus rhythm who did not show spontaneous breathing, respiratory fluctuations in ABF effectively predicted fluid responsiveness[14].

Another study was conducted at the University Hospital of Nîmes in Nîmes, France, in 2010 by the Staff Anesthesiologist and Intensivist Professor Robert Debre *et al.,* The study reported ABF variability in response to rapid fluid infusion. A prospective study was conducted in 39 critically sick patients who were sedated, ventilated, and experienced acute circulatory failure. Transthoracic echocardiography was used to assess subaortic VTI before fluid infusion (baseline), 1 minute after a 100mL hydroxyethyl starch infusion, and 14min after a second 400mL hydroxyethyl starch infusion. For every patient, they computed the variation in the VTI after infusing 100mL of fluid (VTI100). For the VTI100, receiver operating characteristic curves were generated. The receiver operating characteristic curves for CVP and pulse pressure fluctuation were also developed where available. The VTI increased by ≥15% in 21 patients (54%) who were classified as responders following a 500mL volume augmentation. Fluid responsiveness was predicted by a ΔVTI100 of >10%, with corresponding sensitivity and specificity of 95% and 78%, respectively[15].

The findings of the aforementioned study align with those of our study, albeit with the distinction that we administered fluid infusions to spontaneously breathing patients.

A total of 23 patients with acute circulatory failure associated with sepsis who were on mechanical ventilation for acute lung damage were assessed in a study conducted by Barbier *et al.,* The study was carried out in the Medical and Surgical Intensive Care Units of Hospital St. Germain-en-Laye, France. Using subcostal echocardiography, They assessed the diameter of the IVC at end-expiration (Dmin) and end-inspiration (Dmax). The ratio of (Dmax-Dmin)/Dmin was used to compute the index of IVC distensibility (dIVC), which was then reported as a percentage. The cardiac index (CI) of the pulmonary artery trunk was calculated using the Doppler method. Measurements were obtained at baseline and during the increase of plasma expander volume to 7mL/kg.

Individuals were classified as unresponsive (CI increase <15%) or responsive (CI increase >15%). A sensitivity of 90% and a specificity of 90% were achieved in the discrimination of responders and non-responders, with a threshold dIVC of 18. Research has shown that fluid responsiveness is not well predicted by baseline CVP[10].

A previous review conducted by Westerly and Maldonado at the Mayo Clinic in September 2014 examined multiple studies that attempted to predict fluid responsiveness in patients with septic shock. One study examined the utility of changes in the IVC diameter in patients with spontaneous breathing. In this group, respiratory variation in IVC diameter demonstrated a sensitivity of 70% and a specificity of 80% for predicting fluid responsiveness when a large variation (>40%) in IVC diameter was present.

A vena cava collapsibility index (difference between the maximum and minimum diameters divided by the maximum diameter) of >15% was shown to have a 100% negative predictive value but only had a positive predictive value of 62% in a more recent small-scale study. The positive and negative predictive values increased to 75% and 80%,

respectively, when a cutoff of >50% was employed. The IVC was scanned in M-mode approximately 0.5 to 3cm from the ostium of the right atrium, very close to the point where the hepatic veins converged. In summary, patients are unlikely to respond if there is a variance of at least 15%. However, if the variation exceeds 15%, there remains a great deal of ambiguity.

CONCLUSIONS

Transthoracic echocardiography can be used as an accurate method to estimate the response to fluids in patients with shock.

ACKNOWLEDGEMENT

We would like to thank Ain Shams university hospital for supporting this study.

LIMITATIONS OF STUDY

1. Small number of patients.
2. The results are from single medical center (Ain Shams university Hospitals).
3. Poor Echo window in unstable patients.

ETHICS DECLARATION

The study was approved by the Critical Care Medicine Department Ethics Committee of the Faculty of Medicine, Ain Shams University (approval number: FMASU MD 282/2018), and informed consent was obtained from all patients or their next of kin.

CONFLICT OF INTERESTS

There are no conflicts of interest.

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