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

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Abstract

Background: In the last decades dynamic measures had evolved as a better solution to predict fluid responsiveness in clinically shocked patients, out of multiple dynamic parameters we chose transthoracic echocardiographic based measurements of variations of aortic blood flow and inferior vena caval diameter.

Aim of the Work: to assess accuracy of transthoracic echo measured variation in aortic blood flow and inferior vena caval diameter with limited bolus crystalloid infusion in predicting fluid responsiveness in patients with clinical shock. Patients and Methods: This study included 51 selected Patients with acute circulatory failure secondary to clinical shock admitted to ICU. All patients had undergone basic transthoracic echocardiographic (TTE) assessment of aortic blood flow (using left ventricular outflow tract velocity time integral as a surrogate) and inferior vena caval diameter changes with respiratory cycle, these measures were obtained at time of diagnosis of shock and repeated after infusion of 300 ml crystalloid fluid bolus (over fifteen minutes).

Results: Our results had revealed significant difference between responders and non-responders regarding aortic flow variation after fifteen minutes.

Conclusion: Based on the results obtained in this study, it was proved that transthoracic echocardiography can be used as an accurate method to predict fluid responsiveness in shocked patient with fluid challenge.

Keywords: Fluid responsiveness; Hemodynamic indices; Transthoracic echocardiography; Clinically shocked patients

Introduction

Shock is most commonly defined as the life-threatening failure of adequate oxygen delivery to the tissues and may be due to decreased blood perfusion of tissues, inadequate blood oxygen saturation, or increased oxygen demand from the tissues that results in decreased end-organ oxygenation and dysfunction [1].

If left untreated, shock results in sustained multiple organ dysfunction, and damage to vital organs with possible death [2].

Shock is divided into four main types based on the underlying cause: hypovolemic, cardiogenic, obstructive, and distributive shock [3].

The most common clinical features/labs which are suggestive of shock include hypotension, tachycardia, tachypnea, obtundation or abnormal mental status, cold, clammy extremities, mottled skin, oliguria, metabolic acidosis, and hyperlactatemia [4].

The diagnosis of shock is commonly based on a combination of symptoms, physical examination, and laboratory tests. Many signs and symptoms are not sensitive or specific for shock, and as such many clinical decision- making tools have been developed to identify shock at an early stage [5].

Treatment of shock is based on the likely underlying cause. An open airway and sufficient breathing should be established. Any ongoing bleeding should be stopped, which may require surgery or embolization. Intravenous fluid, such as Ringer's lactate or packed red blood cells, is often given. Efforts to maintain a normal body temperature are also important. Vasopressors may be useful in certain cases [3].

Echocardiography allows to obtain the full hemodynamic evaluation. Increases in RV afterload may lead to obstructive shock. The RV is sensitive to pressure and volume overload. Importantly, the RV prolongs its systolic time in face of an increased RV afterload so that the pressure in the RV exceeds the pressure of the left ventricle (LV) at the end of systole leading to paradoxical septal movement [6].

It is also relevant to evaluate right ventricular function in sepsis. Right ventricular function is also frequently impaired in sepsis due to the combined effects of sepsis associated impairment in RV contractility and elevation in RV afterload (ARDS and mechanical ventilation), In one fifth of the patients, RV dysfunction is the predominant feature [7].

Finally, it is also important to carefully check for the presence of dynamic left ventricular outflow tract or midventricular obstructions [8].

Sepsis and septic shock, in general, are associated with long-term morbidity and mortality, with many of the survivors requiring placement into long-term acute care facilities or post-acute care centers [4].

Patients and Methods:

Patients

The study is a Randomized controlled clinical trials included 51 patients. The study was conducted on patients admitted to the Critical care medicine department of Suez General hospital, with the help of clinicians and staff members working in intensive care unit in this hospital in the time period between January 2020 to January 2022.

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

The protocol was approved by the critical care medicine department ethics committee of faculty of medicine, Ain Shams University (Approval number: FMASU MD 282/2018) and informed consent was obtained from the patients or their next of kin.

Any adult patient admitted to the critical care medicine department and fulfilled all the criteria for a spontaneous breathing activity for whom the attending physician decided to perform a fluid challenge because of the presence of at least one of the following clinical signs of inadequate global perfusion: Mean arterial pressure (MAP) below 60 mmHg, Oliguria (urine output less than 0.5ml/kg per hour for more than 2 h), Delayed capillary refill, mottled skin, and tachycardia (heart rate higher than 100/min).

While patients with aortic valvulopathy, mitral insufficiency greater than grade 2, mitral stenosis, tricuspid valve insufficiency grade 3 or more or tricuspid stenosis, atrial arrhythmias, right ventricular infarction or failure, cardiac echogenicity not satisfactory, pregnant ladies, burn patients more than 20% burn of BSA were excluded from the study.

The Sample Size

Using raosoft sample size calculator with margin of error 10%, confidence level 90%, population size 200 & response distribution 50%, the sample size was 51 patients.

Methods

Blood Pressure Measurements

Systolic arterial pressure (SAP) and mean arterial pressure (MAP) in millimeter of mercury (mmHg) were measured either using an oscillometric cuff system at the brachial artery level or using an artery catheter when available.

Central Venous Pressure

Central venous pressure (CVP) in centimeter water (cm H2O) was measured manually. The zero level was taken at the level of the 4th intercostal space in the mid-axillary line.

C- Echocardiographic measurements

The echocardiographic examination was performed by the same operator using a transthoracic ultrasound device (siemens acuson x 300) colored echocardiographic machine using a 3.5 MHz transducer.

● The following measurements were recorded:

a) Stroke Volume

Stroke volume is the amount of blood pumped by the left ventricle of the heart in one contraction, or with each beat. As you probably already know, only about two-thirds of the blood in the left ventricle is pumped out with each beat. Normal stroke volume in a healthy adult can be anywhere between 60ml and 120ml. The stroke volume (SV) in milliliter (ml) was calculated as the product of the velocity time integral of left ventricular outflow tract (LVOT VTI) multiplied by the LVOT area. To calculate the cross sectional area of the left ventricular outflow tract (LVOT area) we can figure this from the parasternal long axis (PLAX) view. To get the cross sectional area of the LVOT, first measure the LVOT diameter during systole. this measurement should be at the insertion points of the right coronary cusp of the aortic valve and the non-coronary cusp of the aortic valve. Using the apical 5-chamber view the LVOT VTI was computed from the area under the envelope of the pulsed-wave Doppler signal obtained at the level of the LVOT annulus. The LVOT VTI value was averaged over three consecutive measurements. Using the parasternal long axis view, the diameter of the LVOT was measured at the insertion of the aortic cusps and the LVOT area was calculated ($\pi \times \text{diameter}^2/4$). As the diameter of the aortic orifice is assumed to remain constant in each patient, the diameter was measured once at baseline.

Inferior Cava Diameter (IVC)

IVC was identified by a subcostal long axis view. A time-motion record of the IVC diameter was generated by M-mode imaging at 2-3 cm from the right atrium. End expiratory values of IVC diameter were measured in centimeter (cm) as the maximum diameter and values were averaged over 3 respiratory cycles. For each patient, a single measurement was only taken at the start of the study.

Systolic arterial pressure (SAP), Mean arterial pressure (MAP), heart rate (HR), Central venous pressure (CVP), LVOT VTI, as well as IVC diameter were measured at baseline while the patient was in the semirecumbent position (45°). These were the first readings (reading 1). Finally, measurements of SAP, MAP, heart rate, CVP and LVOT VTI measurements were collected After fluid challenge of 300 ml within 15 min of fluid bolus (reading 2). All volume challenges were crystalloid fluid. All volume challenges were performed at the discretion of the attending physician. An increase in SV of 15% or more after volume expansion defined a responder patient. There is one group of patients studied to be fluid responder or non responder either by static, dynamic parameters and by echocardiography based on the response of stroke volume and IVC collapsibility to fluid loading.

Statistical Analysis

Recorded data were analyzed using the statistical package for social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The following tests were done

Paired sample t-test of significance was used when comparing between related sample. Comparison between differences by time for non-parametric data using Wilcoxon Signed-Rank Sum test. Independent-samples t-test of significance was used when comparing between two means. Mann Whitney U test: for two-group comparisons in non-parametric data. Chi-square (x2) test of significance was used in order to compare proportions between qualitative parameters. 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 as the following: P-value <0.05 was considered significant.

Results

Table (1) describes the age and the sex distribution of total study population. Age ranged from 34 to 77 years with mean± SD of 63.55±8.64. As regards sex distribution, there was female predominance with 28 females with percentage 54.9% and 23 males with percentage 45.1%.

The majority diagnosis of cardiogenic 9 patients (17.6%), followed by septic shock 8 patients (15.7%), Pneumonia and Stroke 5 patients (9.8%) and Abdominal sepsis 3 patients (5.9%).

This table shows that the lactate of the studied group ranged from 1.8 to 9 with mean 3.41 and ±SD 1.95

Systolic blood pressure with the mean & ±SD in each of pre fluid and post fluid was 74.76±8.41, compared to 86.55±14.93 respectively, the mean difference 11.78 with change 15.89%, there was a highly statistically significant higher mean in post fluid compared to Pre fluid, with (p-value <0.001).

Table (1): Demographic data distribution among study group (n=51).

Demographic data	**Total (n=51)**
Sex	
Female	23 (45.1%)
Male	28 (54.9%)
Age (years)	
Range	34 - 77
Mean±SD	63.55±8.64

In CVP with the mean & ±SD in each of pre fluid and post fluid was 8.88±6.21, compared to 10.79±5.06 respectively, the mean difference 1.91 with change 21.52%, there was a highly statistically significant higher mean in post fluid compared to Pre fluid, with (p-value <0.001).

In IVC, the mean & ±SD in each of pre fluid and post fluid was 3.38±3.05, compared to 3.39±3.34 respectively, the mean difference 0.01 with change 0.29%, there is no statistically significant difference between pre and post fluid, with (p-value >0.05).

In LVOT Vti, the mean & ±SD in each of pre fluid and post fluid was 88.70±32.93, compared to 114.25±41.10 respectively, the mean difference 25.55 with change 30.83%, there was a highly statistically significant higher mean in post fluid compared to Pre fluid, with (p-value <0.001).

In SV stroke volume with the mean & ±SD in each of pre fluid and post fluid was 51.33±10.18, compared to 67.77±19.76 respectively, the mean difference 16.44 with change 31.70%, there was a highly statistically significant higher mean in post fluid compared to Pre fluid, with (p-value <0.001).

There were 27 patients (52.9%) of them had “response” and 24 patients (47.1%) of them had “non-response” regarding responder.

The two groups were comparable in age with the Mean±SD in each of response group and non-response group was 61.59±9.27 compared to 65.75±7.45 respectively, as there is no statistically significant difference between the groups with p-value (p=0.086).

This table showed also that sex was comparable in each of response group were 11 patients (40.7%) and 16 patients (59.3%) were Male and Female, respectively compared to non-response group were 12 patients (50%) and 12 patients (50%) were Male and Female respectively in term of Sex, there is no statistically significant difference between the groups with p-value (p=0.507).

This table shows highly statistically significant higher mean lactate in non-response group was mean value 4.67±2.14 compared to response group was mean value 2.26±0.56, with p-value (p<0.001).
This table shows highly statistically significant higher mean change% in response compared to non-response according SBP (mmHg), DBP (mmHg) and CVP with p-value (p<0.001).

Also highly statistically significant lower mean change% in response compared to non-response according to heart rate "beat/min" with p-value (p<0.001).

This table shows highly statistically significant higher mean change% in response compared to non-response according to IVC, LVOT Vti and SV stroke volume with p-value (p<0.001).

Multivariate analysis revealed that the significant predictors of response outcome were Systolic, Diastolic, HR, CVP, IVC, LVOT Vti and SV stroke v were the best independent predictors of response. All variable indices were significant predictors for responder outcome.

Discussion

Shock is a life-threatening, generalised form of acute circulatory failure with inadequate oxygen delivery to, and consequently oxygen utilisation by the cells [1].

Volumetric Parameters: Extravascular Lung Water (EVLW)

Transpulmonary thermodilution has enabled measurement of several new volumetric parameters, which can be obtained with the PiCCOTM and VolumeViewTM devices (Table 2). The relationship of these parameters is explained in the diagram below.

Table (2): Diagnosis distribution among study group (n=51).

Diagnosis	No.	%
Cardiogenic	9	17.6%
Septic shock	8	15.7%
Pneumonia	5	9.8%
Stroke	5	9.8%
Abdominal sepsis	3	5.9%
Hypovolemic	2	3.9%
Liver cell failure	2	3.9%
Myocardial infarction	2	3.9%
Pulmonary embolism	2	3.9%
20% Burn	1	2.0%
Abortion	1	2.0%
ARDS	1	2.0%
Arrhythmia	1	2.0%
Brain death brain he	1	2.0%
Cardiomyopathy	1	2.0%
Drug poisoning	1	2.0%
Hepatorenal	1	2.0%
Liver cirhosis	1	2.0%
Ruptur uterus	1	2.0%
S/P pelvic exentration	1	2.0%
Splenic rupture	1	2.0%
Stroke +dehydration	1	2.0%
Total	51	100.0%

The most useful of these parameters is extravascular lung water (EVLW). This is an estimation of pulmonary oedema, the fluid accumulated in the interstitial and alveolar spaces [9].

It is calculated indirectly from intrathoracic thermal volume (ITTV) and pulmonary thermal volume (PTV), by subtracting the intrathoracic blood volume from the intrathoracic thermal volume. EVLW is indexed to ideal body weight to produce an EVLW index (EVLWI) measurement. EVLWI measurements are useful in the detection of pulmonary oedema, and in guiding the intensivist with fluid management [10].

This is the ratio of EVLW to pulmonary thermal volume, and reflects the permeability of the capillary-alveolar barrier. Thus PVPI is higher in ALI/ARDS (Table 3) than in hydrostatic pulmonary oedema (Anguel et al., 2007).

Table (3): Lactate distribution among study group (n=51).

Lactate in mmol/L	Total (n=51)
Range	1.8 - 9
Mean±SD	3.41±1.95

Table (4): Comparison between pre fluid and post fluid according to hemodynamic monitoring among study group (n=51).

Hemodynamic monitoring	Pre fluid (n=51)	Post fluid (n=51)	Paired Sample t-test			
			MD±SE	Change%	t-test	p-value
Systolic blood pressure (mmHg)						
Range	57 - 100	60 - 140	11.78±1.66	15.89±2.19	-7.094	<0.001**
Mean±SD	74.76±8.41	86.55±14.93				
Diastolic blood pressure (mmHg)						
Range	30 - 60	35 - 70	4.96±1.03	12.11±2.46	-4.819	<0.001**
Mean±SD	44.41±6.30	49.37±7.83				
Heart rate (beat/min)						
Range	75 - 135	76 - 135	-7.00±1.16	-6.03±1.00	6.013	<0.001**
Mean±SD	110.73±13.99	103.73±13.23				
CVP (cm H2O)						
Range	1 - 22	4 - 23	1.91±0.23	21.52±17.47	-8.376	<0.001**
Mean±SD	8.88±6.21	10.79±5.06				

p-value >0.05 NS; *p-value <0.05 S; **p-value <0.001 HS

Table (5): Comparison between pre fluid and post fluid according to echo dynamics among study group (n=51).

Echo dynamics	Pre fluid (n=51)	Post fluid (n=51)	Paired Sample t-test			
			MD±SE	Change%	t-test	p-value
IVC# Range Mean±SD	0.9 – 3.0 3.38±3.05	1.3 – 2.5 3.39±3.34	0.01±0.16	0.29±1.92	-0.061	0.952
LVOT Vti Range Mean±SD	56 - 212 88.70±32.93	59 - 245 114.25±41.10	25.55±3.39	30.83±4.15	-7.544	<0.001**
SV stroke volume Range Mean±SD	26 - 88 51.33±10.18	26 - 101 67.77±19.76	16.44±2.04	31.70±3.91	-8.045	<0.001**

Wilcoxon test

Table (6): Response distribution among study group (n=51).

Response	No.	%
Response	27	52.9%
Non-response	24	47.1%
Total	51	100.0%

Table (7): Comparison between response and non-response according to demographic data.

Demographic data	Response			
(n=27)	Non-response			
(n=24)	Test value	p-value		
Age (years)				
Range	34-74	45-77	t=-1.751	0.086
Mean±SD	61.59±9.27	65.75±7.45		
Sex				
Male	11 (40.7%)	12 (50%)	x2=0.440	0.507
Female	16 (59.3%)	12 (50%)		

Using: t-Independent Sample t-test; x^2: Chi-square test

Table (8): Comparison between response and non-response according to lactate.

Lactate	Response (n=27)		Non-response (n=24)		t-test value	p-value
	Mean	±SD	Mean	±SD		
Lactate	2.26	0.56	4.67	2.14	-5.432	<0.001**

Table (9): Comparison between response and non-response according to hemodynamic monitoring.

Hemodynamic monitoring	Response (n=27)		Non-response (n=24)		Test value	p-value
	Mean	±SD	Mean	±SD		
SBP (mmHg)						
Pre fluid	74.78	8.22	74.75	8.80	t=0.012	0.991
Post fluid	95.00	14.21	77.04	8.83	t=5.339	<0.001**
Difference post-pre	20.22	9.96	2.29	4.03	U=-6.006	<0.001**
Change%	27.12	12.73	3.25	5.68	U=-5.926	<0.001**
DBP (mmHg)						
Pre fluid	43.89	5.77	45.00	6.92	t=-0.625	0.535
Post fluid	52.89	7.45	45.42	6.29	t=3.844	<0.001**
Difference post-pre	9.00	7.30	0.42	4.06	U=-4.330	<0.001**
Change%	21.46	18.02	1.60	9.41	U=-4.251	<0.001**
Heart rate (beat/min)						
Pre fluid	115.33	11.09	105.54	15.28	t=2.640	0.011
Post fluid	101.93	10.85	105.75	15.46	t=-1.031	0.308
Difference post-pre	-13.41	6.21	0.21	2.06	U=-5.898	<0.001**
Change%	-11.57	5.19	0.20	1.85	U=-5.891	<0.001**
CVP						
Pre fluid	4.19	2.83	14.17	4.46	U=-9.655	<0.001**
Post fluid	7.31	2.40	14.71	4.36	U=-7.617	<0.001**
Difference post-pre	3.13	0.16	0.54	0.10	U=-5.792	<0.001**
Change%	74.78	20.16	3.82	0.99	U=-5.931	<0.001**

Using: U=Mann-Whitney test; t-Independent Sample t-test

Table (10): Comparison between response and non-response according to echo dynamics.

Echo dynamics	Response (n=27)		Non-response (n=24)		Test value	p-value
	Mean	±SD	Mean	±SD		
IVC						
Pre fluid	2.76	4.98	4.09	7.12	t=-4.691	<0.001**
Post fluid	2.93	4.32	3.92	6.35	t=-3.081	0.002*
Difference post-pre	0.17	0.18	-0.17	0.14	U=-5.406	<0.001**
Change%	6.18	3.01	-4.18	0.91	U=-5.339	<0.001**
LVOT Vti						
Pre fluid	88.63	31.17	88.78	35.49	U=-0.016	0.987
Post fluid	134.30	35.76	91.71	35.00	U=4.288	<0.001**
Difference post-pre	45.67	15.03	2.93	2.71	U=-6.132	<0.001**
Change%	54.71	20.45	3.95	4.07	U=-6.097	<0.001**
SV stroke volume						
Pre fluid	53.59	10.93	48.79	8.80	t=1.714	0.093
Post fluid	82.30	14.04	51.43	9.98	t=8.942	<0.001**
Difference post-pre	28.70	7.94	2.64	4.06	U=-6.062	<0.001**
Change%	55.01	15.49	5.47	8.47	U=-6.040	<0.001**

Table (11): Multivariate binary logistic regression analysis of hemodynamic monitoring and echo dynamics affecting response.

Parametes	β	Wald	Sig.	Odds ratio	95% C.I.	
					Lower	Upper
Constant	2.286	5.336	<0.001**	4.691		
Systolic change%	0.219	7.055	<0.001**	3.086	0.771	2.054
Diastolic change%	0.412	4.456	0.044*	2.091	0.774	2.064
HR change%	0.194	7.189	0.016*	5.106	0.785	2.093
CVP change%	1.659	6.091	0.026*	4.674	3.319	8.844
IVC change%	0.780	9.500	<0.001**	2.897	0.637	1.697
LVOT Vti change%	0.227	8.411	0.047*	1.294	0.919	2.449
SV stroke v change%	0.281	3.400	0.013*	1.248	0.886	2.360

β: Regression coefficient, SE: Standard error, OR: Odds ratio, CI: Confidence interval

In the current study, we tested the hypothesis that a low volume (300 ml) of rapidly delivered crystalloid fluid can predict fluid responsiveness.

By using a low volume for this challenge, the deleterious effects of fluid among non-responders would be limited hypothetically. According to the Frank-Starling cardiac function curve, the concept of fluid responsiveness is defined as a significant increase in stroke volume secondary to an increase in cardiac preload. Moreover, because of the form of the curve, the increase in stroke volume theoretically would be greater in the steep portion of the Frank-Starling curve at the beginning (in particular, the first 100 ml) of the fluid challenge, especially when the rate of fluid administration is increased.

A positive response to volume expansion is defined as a 15% increase in cardiac output or cardiac index after a fluid challenge over 15-20 min.

Transthoracic echocardiography provides a rapid, simple, and noninvasive assessment of stroke volume via the measurement of the left ventricular outflow tract velocity time index (VTI) and inferior vena caval diameter [11].

The rationale behind using aortic blood flow variation as a predictor of fluid responsiveness relies on the fact that cardiac output is the product of stroke volume and heart rate. The stroke volume is calculated by the product of the subaortic VTI recorded echocardiographically with pulse Doppler at the left ventricle outflow tract(LVOT) on an apical 5-chamber view and the subaortic left ventricular area (SV=VTI x LVOT area) LVOT area can be measured following the formula: subaortic left ventricular area(LVOT) = πR2 where R means radius of the left ventricular outflow tract which equals half its diameter measured at 2Dimensional imaging.Assuming that the diameter of the left ventricle outflow chamber is constant in a given patient and that variations of heart rate are low, the variations in cardiac output are related to VTI variations. Thus, the measurement of VTI and its variations are directly correlated with variations in cardiac output, avoiding the potential error in the measurement of the left ventricle outflow tract diameter [12].

The Concept of inferior vena cava size and collapsibility can be explained as follow, hypovolemic patients can be identified using measurement of both size and collapsibility of the IVC for estimation of right atrial pressure (RAP) [11].

Inspiration in normovolemic, spontaneously breathing patients causes negative intrathoracic pressure and a decrease in IVC size. An exaggerated response in IVC collapse occurs in patients in the hypovolemic state during inspiration [13].

Routine measurements of size of the IVC and collapsibility with respiration have been used in patients with shock to reliably guide fluid management decisions [14].

The transthoracic echocardiographic subcostal window can be used to view the IVC in the sagittal plane by angling and rotating the transducer to the left from the subcostal four-chamber view. M-mode imaging allows high-frame rate measurements of size changes throughout the respiratory cycle [15].

Patients who were predicted to be fluid responsive (measured IVC collapse >50%) demonstrated statistically significant improvements in catheter-measured cardiac index, cardiac output and mean arterial pressure after fluid resuscitation [16].

The aim of this work is to assess accuracy of TTE measured variation in aortic blood flow and inferior vena caval diameter with limited bolus fluid infusion in predicting fluid responsiveness in patients with clinical shock.

This prospective study included 51 selected Patients with acute circulatory failure admitted to ICU. All patients had undergone basic transthoracic echocardiographic (TTE) assessment of aortic blood flow(ABF)(using LVOT velocity time integral(VTI) as a surrogate) and inferior vena caval diameter changes with respiratory cycle (ΔIVCD) (through measuring $IVCD_{min}$ in inspiration and $IVCD_{max}$ in expiration) where ΔIVCD =[*IVCDmax-IVCDmin]/IVCDmax*) then 300ml of fluid bolus were infused quickly over fifteen minutes followed by TTE measurement of these parameters (VTI and ΔIVCD).

Patients were classified into two groups, responders or non-responders according to fluid responsiveness, The threshold for responsiveness was ≥ 15-20% increase in stroke volume after 300 ml of fluid infusion.

Out of 51 patients included in our study there were 27 responders and 24 non-responders. There was no significant difference between responders and non-responders regarding age, gender, height and body weight.

No significant difference had been observed between both groups concerning heart rate.

A significant difference between responders and non- responders was clear concerning mean VTI at the baseline, after 300 ml fluid infusion.

Finally there was a significant difference between responders and non-responders concerning mean ΔIVCD at the baseline, after 300ml fluid infusion.

Our results regarding aortic blood flow variations are consistent with a study published in March 2013 performed by Xavier Monnet et al. [17] a Hôpitaux universitaires Paris-Sud, Hôpital de Bicêtre University hospital medical intensive care to test whether fluid responsiveness can be predicted by the respiratory variation in aortic blood flow and/or the flow time corrected for heart rate monitored with esophageal Doppler. The study included 38 mechanically ventilated patients with sinus rhythm and without spontaneous breathing activity in whom volume expansion was planned. The aortic blood flow was measured using an esophageal Doppler monitoring device before and after fluid infusion (500 ml NaCl 0.9% over 10 min). The variation in aortic blood flow over a respiratory cycle between its minimal and maximal values was calculated. The flow time was also measured. Aortic blood flow increased by at least 15% after volume expansion in 20 patients (defined as responders). Before fluid infusion the respiratory variation in aortic flow was higher in responders than in non-responders (28±12% vs. 12±5%). It significantly decreased after volume expansion (18±11%) in responders only.

A respiratory variation in aortic flow before volume expansion of at least 18% predicted fluid responsiveness with a sensitivity of 90% and a specificity of 94%. Flow time increased with fluid infusion in responders and non-responders.

They concluded that the respiratory variation in aortic blood flow reliably predicts fluid responsiveness in patients with sinus rhythm and without breathing activity [17].

Another study conducted in University hospital of Nîmes in Nîmes, France in 2010 by Staff Anesthesiologist and Intensivist; Professeur Robert Debre et al. assessed Aortic Blood Flow variation in response to rapid fluid infusion, Thirty-nine critically ill ventilated and sedated patients with acute circulatory failure were prospectively studied. Subaortic VTI was measured by transthoracic echocardiography before fluid infusion (baseline), after 100 ml hydroxyethyl starch infusion over 1 min, and after an additional infusion of 400 ml hydroxyethyl starch over 14 min. The authors measured the variation of VTI after 100 ml fluid (VTI100) for each patient. Receiver operating characteristic curves were generated for (ΔVTI100). When available, receiver operating characteristic curves also were generated for pulse pressure variation and central venous pressure. After 500 ml volume expansion, VTI increased ≥15% in 21 patients (54%) defined as responders. ΔVTI100 ≥ 10% predicted fluid responsiveness with a sensitivity and specificity of 95% and 78%, respectively [18].

The results of the above mentioned study are in agreement of ours with the difference that we apply fluid infusion to spontaneously breathing patients.

Concerning the role of respiratory changes in inferior vena cava diameter (IVCD), a study performed by Barbier et al. [16], in medical and surgical intensive care units, Hospital St. Germain-en-Laye France, on twenty-three patients with acute circulatory failure related to sepsis and mechanically ventilated because of an acute lung injury, they measured Inferior vena cava diameter (D) at end-expiration (Dmin) and at end-inspiration (Dmax) by echocardiography using a subcostal approach. The distensibility index of the IVC (dIVC) was calculated as the ratio of Dmax - Dmin / Dmin, and expressed as a percentage. The Doppler technique was applied in the pulmonary artery trunk to determine cardiac index (CI). Measurements were performed at baseline and after a 7 ml/kg volume expansion using a plasma

expander. Patients were separated into responders (increase in CI≥15%) and non-responders (increase in CI <15%) Using a threshold dIVC of 18%, responders and non-responders were discriminated with 90% sensitivity and 90% specificity. They observed that baseline central venous pressure did not accurately predict fluid responsiveness [11].

Westerly and Maldonado in Mayo Clinic in September 2014 had reviewed multiple studies that tried to predict fluid responsiveness in patients with septic shock. In one study the utility of IVC diameter changes has been studied in spontaneously breathing patients. In one group of spontaneously breathing patients, respiratory variation in IVC diameter had only a fair sensitivity of 70% and specificity of 80table% for predicting fluid responsiveness when a large variation (>40%) in diameter was present.

In a more recent small study, vena cava collapsibility index (difference between maximum and minimum diameters divided by the maximum diameter) > 15% had a positive predictive value of only 62%, but negative predictive value of 100%. If a cutoff > 50% was used, the positive predictive value was 75% and negative predictive 80%. The IVC was imaged in M mode just proximal to junction of hepatic veins, about 0.5 cm to 3 cm. from the ostium of the right atrium. Taken together, if a variation of at least 15% is not present, the patient seems unlikely to respond, but there is still considerable uncertainty for those with variation more than 15%.

Conclusion

Based on the results obtained in this study, it was proved that transthoracic echocardiography can be used as an accurate method to predict fluid responsiveness in shocked patients.

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