Role of Inferior Vena Cava Diameter as Indicator of Fluid Response in Septic and Cardiogenic Shock in Mechanically Ventilated Patients

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Contents:

1.	List of abbreviations	3
2.	List of figures	6
3.	List of tables	8
4.	Introduction	9
5.	Aim of the study	13
6.	Shock	14
7.	Static indices	48
8.	Dynamic indices	53
9.	Patients and methods	87
10.	Results	93
11.	Discussion	106
12.	Conclusion	108
13.	English summary	110
14.	References	112
15.	Arabic summary	124

List of abbreviations:

Abbrev.	Meaning
AAA	***Abdominal Aortic Aneurysm***
AC	***Alternating Component / pulsatile infra-red signal***
ACES	***Abdominal And Cardiac Evaluation With Sonography In Shock***
APACHE score	***Acute Physiology And Chronic Health Evaluation Score***
ARDS	***Acute Respiratory Distress Syndrome***
AUC	***Area Under Curve***
BMI	***Body Mass Index***
CI	***Confidence Interval***
CO	***Cardiac Output***
CT	***Computed Tomography***
CVP	***Central Venous Pressure***
DC	***Direct or constant component /non pulsatile infra-red signal***
DDIVC	***Distensibility Difference Of Inferior Vena Cava***
dDown	***Delta Down(Expiratory Decrease In Left Ventricle Systolic Pressure***
dIVC	***Distensibility Index If Inferior Vena Cava***
Dmax	***Maximum Diameter Of Inferior Vena Cava***
Dmin	***Minimum Diameter Of Inferior Vena Cava***
DO2	***Oxygen Delivery***
dPOP	***Change In Pulseoximetry Plethysmography***
dUp	***Delta Up(Inspiratory Increase In Systolic Pressure)***
ECG	***Electrocardiogram***

ED	***Emergency Department***
EEO	***End Expiratory Occlusion***
EGDT	***Early Goal Directed Therapy***
EVLW	***Extravascular Lung Water***
FAST	***Focused Assessment With Sonography In Trauma***
FiO2	***Fraction Of Inspired Oxygen***
FTC	***Flow Time Corrected For Heart Rate***
GI	***Gastrointestinal***
H_2O	***Water***
HR	***Heart Rate***
IAH	***Intra-Abdominal Hypertension***
IAP	***Intra-Abdominal Pressure***
ICU	***Intensive Care Unit***
IV	***Intravenous***
IVC	***Inferior Vena Cava***
IVC	***Inferior Vena Cava***
LMWH	***Low Molecular Weight Heparin***
LV SV	***Left Ventricular Stroke Volume***
M- mode	***Motion Mode***
MAP	***Mean Arterial Pressure***
Mm Hg	***Millimeters Mercury***
PaO2	***Partial Arterial Oxygen Pressure***
PAOP	***Pulmonary Artery Occlusion Pressure***
Paw	***Airway Pressure***
PCO2 gap	***Arterio-Venous Carbon Dioxide Gap***
PEEP	***Positive End Expiratory Pressure***
Pes	***Esophageal Pressure***

PI	***Perfusion Index***
PICCO	***Pulse Contour Cardiac Output***
PLR	***Passive Leg Raising***
PPV	***Pulse Pressure Variation***
PRBCs	***Packed red blood cells***
PVI	***Plethysmography Variability Index***
RRT	***Renal Replacement Therapy***
SaO2	***Arterial Oxygen Saturation***
SCVO2	***Central Venous Oxygen Saturation***
SIRS	***Systemic Inflammatory Response Syndrome***
SPmax	***Maximum Systolic Pressure***
SPmin	***Minimum Systolic Pressure***
SPref	***Reference Systolic Pressure At End Expiration***
SR	***Sinus rhythm***
SSPS	***Statistical Package Of Social Science***
SV	***Stroke Volume***
SVC	***Superior Vena Cava***
SVR	***Systemic Vascular Resistance***
SVV	***Stroke Volume Variation***
TV	***Tidal Volume***
UFH	***Unfractionated Heparin***
VO2	***Systemic Oxygen Consumption***
VPV	***Ventilator Induced Plethysmographic Variation***
VTE	***Venous Thromboembolism***
VTI	***Velocity Time Integral***

List of figures

Figure no:	Description	page
1.	*Algorithm for fluid strategy & choice of monitoring of responsiveness*	45
2.	*Frank-Starling curve demonstrating relationship between change in preload to change in Stroke Volume(SV) in a normal and failing ventricles. A given change in preload may cause variable changes in SV, depending on the slope of the curve.*	47
3.	Central venous pressure waveform &ECG correlation	49
4.	*Pulmonary artery occlusion waveforms using Swan-Ganz catheter*	51
5.	*Passive leg raising (PLR) can be performed by elevating the limbs, while placing the patient in the supine position to transfer blood both from the lower limbs as from the abdominal compartment creating a sufficient venous return to significantly elevate biventricular preload.*	56
6.	*Measurement of pulse pressure variation.*	58
7.	*Delta up , delta down systolic pressure variation*	61
8.	*Hemodynamic effects of mechanical ventilation. The cyclic changes in left ventricle (LV) stroke volume are mainly related to the expiratory decrease in LV preload because of the inspiratory decrease in right ventricle (RV) filling*	63
9.	*Arterial waveform analysis during positive pressure ventilation predicts an individual patients' position on their Starling curve and allows optimization of cardiac performance. SVV, stroke volume variation.*	64
10.	*Positive end expiratory occlusion pressure*	65
11.	*Plethysmography Variability Index* ***(PVI)*** *waveform views Perfusion Index (PI) max ,(PI) min*	68

12.	*The Bioreactance technology*	**71**
13.	*Optimization of preload based on dynamic changes in preload responsiveness and extravascular lung water*	**74**
14.	*Intra-abdominal pressure measurement using the pressure transducer technique*	**75**
15.	*Measurement of aortic velocity-time integral (VTI).*	**78**
16.	*Corrected flow time waveform 1/3 cardiac cycle*	**79**
17.	*Measurement of the inferior vena cava (IVC) caval index. .*	**82**
18.	*IVC longitudinal view by sonar.*	**99**
19.	*IVC short axis view by sonar.*	**99**
20.	*IVC distended during inspiration, longitudinal view.*	**100**
21.	*IVC collapsed during expiration, longitudinal view.*	**100**
22.	*The causes of sepsis in group 1*	**101**
23.	*Boxplot showing the median APACHE score in each group.*	**101**
24.	*Types and number of supports given in both groups.*	**102**
25.	*Mean IVC distensibility index(DI) before and after fluid boluses in both groups.*	**103**
26.	*Mean serum lactate before and after fluid boluses in both groups.*	**104**
27.	*Number of fluid boluses (median, minimum and maximum) given in both groups.*	**105**

List of tables

Table no:	Description	Page
1.	*Categories of shock.*	18
2.	*Causes of shock in trauma patients*	37
3.	*Hemodynamic monitoring methods summary.*	42
4.	*Summary of methods predicting preload responsiveness with diagnostic threshold and limitations*	43
5.	*Techniques used to determine fluid responsiveness, used technologies*	43
6.	*Pre-intervention assessment.*	102
7.	*Descriptive analysis of the patients' demographic data in each group.*	103
8.	*Comparison between the 2 groups regarding the vital data before administration of fluid boluses*	103
9.	*Comparison between the 2 groups regarding the vital data after administration of fluid boluses*	104
10.	*Post-intervention assessment.*	104
11.	*Comparison of hemodynamic parameter change in septic shock patients pre and post intervention arranged in descending order*	105
12.	*Comparison of hemodynamic parameter change in cardiogenic shock patients pre and post intervention arranged in descending order*	106

INTRODUCTION

It is important for critical care physicians to address the hemodynamic status of critically ill patients for an appropriate guide to fluid therapy and inotropes. Different techniques are employed for this purpose which include physical examination, central venous pressure (CVP) measurement, biochemical markers, estimate of the vascular pedicle width, pulmonary artery catheters, and sonographic inferior vena cava (IVC) diameter assessment. CVP measurements are the most frequently employed through estimation of the preload; however CVP measurement requires insertion of central venous catheters which can be costly, time consuming and can lead to complications***. (Khalil et al., 2015)***.

Although intravenous fluid is often the initial treatment in hypotensive patients, aggressive volume resuscitation may be detrimental in some patients and in certain types of shock. Accurate diagnosis of shock state can be challenging because physical findings of hypovolemic, distributive, cardiogenic, and obstructive shock often overlap. Pulmonary artery and central venous pressure catheters, which provide physiologic data such as cardiac output and right atrial pressure, are time-consuming, invasive, and carry considerable risks. Central

venous pressure has long been used to guide fluid management; however, data suggest that in critically ill patients, central venous pressure may not correlate with the effective intravascular volume. Furthermore, invasive hemodynamic monitoring has not been shown to benefit patients. ***(Shah et al., 2005)***

Assessment of hemodynamic status in a shock state remains a challenging issue in emergency medicine and critical care. As the use of invasive hemodynamic monitoring declines, bedside-focused ultrasound has become a valuable tool in the evaluation and management of patients in shock. No longer a mean to simply evaluate organ anatomy, ultrasound has expanded to become a rapid and noninvasive method for the assessment of patient physiology. Clinicians caring for critically ill patients should strongly consider integrating ultrasound into their resuscitation pathways. ***(Seif et al., 2012).*** Assessment of the IVC distensibility index in mechanically ventilated patients provides a useful and reliable tool in predicting response to volume in critically ill patients.

The inferior vena cava (IVC) is a compliant blood vessel, easily distended, especially in cases of hypovolemia. Assessment of the physiologic characteristics of the IVC provides a rapid distinction between low and high volume

states and offers the clinician a rapid, noninvasive way to guide resuscitation in critically ill patients.

The four core types of shock—cardiogenic, hypovolemic, obstructive, and distributive—can readily be identified by echocardiography. Even within each of the main headings contained in the shock classification, a variety of pathologies may be the cause and echocardiography will differentiate which of these is responsible. Increasingly, as a result of more complex and elderly patients, the shock may be multifactorial, such as a combination of cardiogenic and septic shock or hypovolemia and ventricular outflow obstruction.

Measuring the IVC should be part of a hemodynamic assessment specific protocol to evaluate the necessity or not of volume *(**Varas et al., 2015**)*.

Ultrasound of inferior vena cava (IVC) is a tool that can provide a rapid and non-invasive means of gauging preload and the need for fluid resuscitation. ***(Demeria et al., 2004*)**.

Echocardiography is pivotal in the diagnosis and management of the shocked patient. Important characteristics in the setting of shock are that it is non-invasive and can be rapidly applied with competency in basic critical care echocardiography is now regarded as a mandatory part of critical care training with clear guidelines available. The majority of pathologies found in shocked patients are readily

identified using basic level 2D and M-mode echocardiography. **(*McLean, 2016)***.

The diagnostic benefit of echocardiography in the shocked patient is obvious. The increasing prevalence of critical care physicians experienced in advanced techniques means echocardiography often supplants the need for more invasive hemodynamic assessment and monitoring in shock. ***(McLean, 2016)***.

Aim of the study

The objective of this study was to evaluate the inferior vena caval (IVC) distensibility index as an indication for venous capacitance and fluid responsiveness in patients presenting with septic and cardiogenic shock.

Shock

Shock is a state of acute cardiovascular or circulatory failure. It leads to decreased delivery of oxygenated blood to the body's organs and tissues or impaired oxygen utilization by peripheral tissues, resulting in end-organ dysfunction. **(*Richards et al. , 2014).***

The mechanisms that can result in shock are divided into 4 categories: (1) hypovolemic, (2) distributive, (3) cardiogenic, and (4) obstructive. While much is known regarding treatment of patients in shock, several controversies continue in the literature. Assessment begins with identifying the need for critical interventions such as intubation, mechanical ventilation, or obtaining vascular access. Prompt workup should be initiated with laboratory testing (especially of serum lactate levels) and imaging, as indicated. Determining the intravascular volume status of patients in shock is critical and aids in categorizing and informing treatment decisions. This issue reviews the four primary categories of shock as well as special categories, including shock in pregnancy and traumatic shock. Adherence to evidence-based care of the specific causes of shock can optimize a patient’s chances of surviving this life-threatening condition. **(*Richards et al. , 2014).***

The physiologic mechanism of oxygen delivery to peripheral tissues (DO2) is described in the formula in **Equation 1.**

Equation 1

Oxygen Delivery DO2 = (cardiac output) x [(hemoglobin concentration) x SaO2 x 1.39] + (PaO2 x 0.003)

DO2 = COP X (Hb x SaO2 x 1.39)+ (PaO2x 0.003).

DO2, oxygen delivery; PaO2; partial arterial oxygen pressure; SaO2, arterial oxygen saturation. **(*Richards et al. , 2014).***

Blood pressure is not included in this formula; while shock is frequently associated with hypotension, patients may present with "cryptic shock" in which they have a blood pressure typically considered to be within normal ranges, yet they have pathophysiologic signs of shock (particularly early in their clinical course). **(*Richards et al. , 2014).***

Equation 2 Demonstrates the influence that cardiac output has on blood pressure (as evidenced by mean arterial pressure). A mean arterial pressure that decreases below a critical threshold will result in decreased cardiac output and, thereby, decreased DO2. **(*Richards et al., 2014).***

Equation 2 : Mean Arterial Pressure = Cardiac Output X Systemic Vascular Resistance (MAP = CO X SVR). **(*Richards et al., 2014).***