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The Role of Ultrasound Guided Internal Jugular Collapsibility Index in Assessment of Fluid Responsiveness in Sepsis: Review Article

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ABSTRACT
Intravascular volume status assessment still among the greatest significant obstacles in critical care management. The restrictions of invasive hemodynamic monitoring are being more apparent. Inferior vena cava (IVC), left internal jugular vein (IJV) collapsibility, pulmonary artery occlusion pressure (PAOP), central venous pressure (CVP), or left ventricular end diastolic area, as well as transpulmonary thermodilution index are all ways to evaluate body volume status. In recent years, the fluid status of a cases has been determined by the ultrasound-guided measurement of the inferior vena cava diameter and its variations with respiration. It is a comparatively cheap and also safe method. It may be employed as an alternative to central venous catheterization to evaluate the volume status of cases. The IVC-collapsibility index was demonstrated to be correlated with clinical and invasive assessments of intravascular volume status and has received increasing attention. It is simple to record and requires minimal training. Nevertheless, IVC measurement isn't achievable in ten to fifteen percent of cases because of obesity, raised intra-abdominal pressure, abdominal surgical dressings, huge amounts of intra-thoracic air, excessive intra-abdominal gas, extrinsic structures compressing IVC, elevated pulmonary artery pressure, tricuspid, or pulmonary valve disease.
Keywords: IJV, Inferior vena cava collapsibility, Fluid management, Sepsis.

INTRODUCTION
The case's outcome is significantly influenced by the management of fluid in the ICU. Ischemia is the result of the inappropriate usage of vasopressors to preserve blood pressure, which results in hypovolemia and reduced organ perfusion. Conversely, fluid overload induces cellular swelling and also lung congestion, which subsequently elevates mortality and morbidity. According to a meta-analysis of 5 researches, central venous pressure's ability to accurately detect fluid responsiveness is limited to fifty-six percent as evidenced by the associations among assessed blood volume, alteration in cardiac index, and stroke volume (SV) as a reaction to volume expansion. Fluid resuscitation is typically advised to enhance cardiac output and enhance tissue hypoperfusion, as sepsis is linked to a decline in effective blood volume.

In cases who experienced septic shock and severe sepsis, early goal-directed management has been established to decrease morbidity as well as mortality by achieving an optimal fluid status. Clinical examination was determined to be unreliable, lacks precision and accuracy in measuring the intravascular volume status of critically ill cases. A variety of non-invasive methods of measuring central venous pressure have been proposed as an alternative to invasive central venous pressure assessment, utilizing portable ultrasonography. The volume status of critically ill cases was extensively investigated using IVC parameters, including collapsibility, caval index as well as diameter as an indirect measurement of central venous pressure. The study's results have been variable and conflicting [(1)].

Our aim was to estimate the effectiveness of IJV-collapsibility index in prediction of fluid reaction in addition to the calculation of volume status in association with internal jugular vein- collapsibility index and invasively monitored central venous pressure (CVP) in intensive care unit sepsis cases

Sepsis
Life-threatening organ dysfunction is the reason of a dysregulated host reaction to infection, which is known as sepsis. Sepsis as well as septic shock are significant healthcare issues that impact millions of individuals globally every year and result in killing of as many as one in four (and frequently more). Septic shock is a subsection of sepsis which is correlated with an elevated likelihood of mortality and circulatory and cellular/metabolic dysfunction, similar to poly-trauma, acute myocardial infarction, or stroke. Mortality rates of up to 50% are linked with septic shock. In the first hours subsequent to the sepsis progress, results are enhanced by early detection and suitable management. In a subsequent revision, parameters were established to support a SIRS response, however SIRS was reserved. The framework for evaluating sepsis was introduced, and it was recommended that laboratory measurements of numerous inflammatory indicators (In addition to abnormalities in the count of white blood cells) to recognize sepsis. The framework was established on the basis of predisposition, host response, pathogen, as well as organ dysfunction[(2)].

Circulatory dysfunction, as recognized by a mean arterial pressure of lower than sixty-five-millimeter mercury or a sustained necessity for vasopressor medication in the nonappearance of hypovolemia, and a

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raised serum lactate level (more than two millimoles per liter), are all components of septic shock in Sepsis-3 [3].

lactate level measurement:

Although serum lactate isn't a direct marker of tissue perfusion, it could be used as a surrogate, as rises could indicate tissue hypoxia, faster aerobic glycolysis caused by excessive beta-adrenergic stimulation, or additional factors that are linked to negative results [4].

Obtain blood cultures:

Before antibiotics were introduced, sterilization of cultures may take place throughout minutes of the initial dose of a suitable antimicrobial. Therefore, it is imperative to obtain cultures prior to antibiotic administration in order to enhance the identification of pathogens and enhance results [5].

Administer broad-spectrum antibiotics:

For cases who present with sepsis or septic shock, empiric broad-spectrum treatment with 1 or extra intravenous antimicrobials should be initiated immediately to address all likely pathogens. Once pathogen recognition and sensitivities have been determined, empiric antimicrobial therapy have to be either limited or discontinued if it is determined that the case doesn't have an infection. The connection among early administration of antibiotics to treat suspected infections as well as antibiotic stewardship continues to be a critical component of high-quality sepsis management. Antimicrobials must be stopped if infection is subsequently determined to not occur [6]

Administer intravenous fluid:

Stabilizing sepsis-induced tissue hypoperfusion or septic shock necessitates early and effective fluid resuscitation. Given the urgency of this medical emergency, the primary fluid resuscitation has to be commenced promptly upon the identification of a case who experienced sepsis, hypotension, and raised lactate levels, and ought to be completed within three hours of their recognition [7].

The guidelines suggest that a minimum of thirty milligrams per kilogram of intravenous crystalloid fluid be included. Despite the limited amount of controlled information available in the literature to substantiate this volume, current interventional investigations have defined this as the standard practice in the early phases of resuscitation, alongside observational evidence provides support. The lack of obvious benefits from colloid in comparison with crystalloid solutions in sepsis cases, along with the cost of albumin, strongly recommends the usage of crystalloid solutions for initial resuscitation [8].

Apply vasopressors:

Urgently restore vital organ perfusion pressure during resuscitation. If blood pressure isn't restored, initiate vasopressors within an hour to reach MAP of sixty-five mm Hg. Literature reviews outline vasopressor effects. In cases who experienced severe sepsis as well as septic shock, early goal-directed medication was demonstrated to decrease morbidity and mortality by achieving an optimal fluid status. Clinical examination was determined to be inaccurate also lacks precision and accuracy in evaluating the intravascular volume status of critically ill cases. Likewise, the absence of hypotension or tachycardia doesn't exclude the possibility of significant loss of blood within the initial stages of hemorrhagic shock. Clinical investigation and vital signs are frequently confusing also could result in the loss of appreciated time within resuscitation. The gold standard to measure central venous pressure is the insertion of a central venous catheter; however, this method is time-consuming, invasive, and necessitates specially trained personnel. Furthermore, it isn't viable in pre-hospital conditions or in an emerging resuscitation situation, and it entails as well as complications. The volume status of critically ill cases was widely investigated using IVC parameters, including collapsibility, diameter, and caval index, as an indirect measurement of central venous pressure. The findings of these studies have been not compatible and were varying. [1]

Nevertheless, IVC measurement isn't feasible in ten to fifteen percent cases because of obesity, abdominal surgical dressings, exaggerated intra-abdominal gas, raised intra-abdominal pressure, large amounts of intra-thoracic air, extrinsic structures compressing inferior vena cava, raised pulmonary artery pressure, tricuspid, or pulmonary valve disease. IJV is being progressively employed as an alternative to the inferior vena cava to measuring the central venous pressure indirectly. In routine clinical practice, the height of jugular venous pulsation was observed as an indirect measurement of central venous and right atrial pressure; however, its sensitivity is not particularly high. The right atrial pressure was estimated accurately in only fifty percent of cases through physical investigation [9].

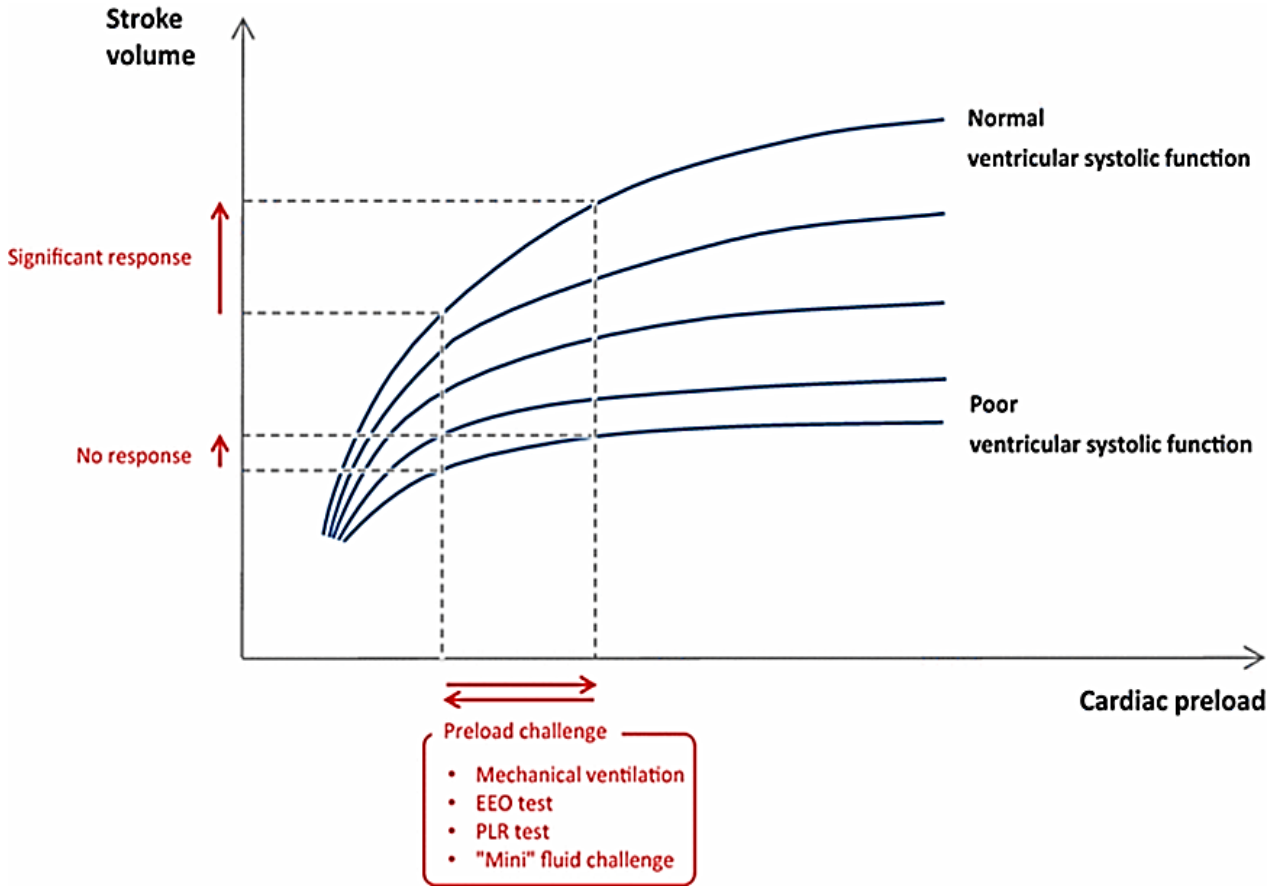
Why it is essential to predict fluid responsiveness?

The decision to administer fluids for cases with acute circulatory failure shouldn't be considered lightly. The risk of excessive fluid administration was definitely indicated. Additionally, cardiac output doesn't always rise as anticipated as a result of volume expansion. Therefore, the initial step in the fluid strategy should be the prediction of fluid responsiveness, particularly following the very first stage and/or when the loss of fluid isn’t readily apparent. A critical therapeutic dilemma may arise as a result of volume expansion, the initial medication for

acute circulatory failure. Contrary to this, the disease severity motivates individuals to start medication in a rapid besides widespread way.

This is consistent with the pivotal research conducted by **Rivers *et al.*** [10], which established that the administration of enormous fluids throughout the 1st six hours of resuscitation in cases having septic shock and severe sepsis was linked with a better result. Conversely, it was clearly established that fluid excess has adverse effects. Fluid overload raises the mortality rate of critically ill cases also prolongs mechanical ventilation. This is particularly true for cases with sepsis, intra-abdominal hypertension, acute respiratory distress syndrome (ARDS), and acute renal injury. The potential benefit of volume expansion, which is associated with a rise in cardiac output [11].

(CO) and oxygen delivery, have to be balanced by the risk of exacerbating oedema in the lungs and tissues. Cardiovascular physiology complicates the reaction to a fluid challenge. A fluid challenge may cause either a negligible or significant rise in cardiac output and SV, as the Frank–Starling curve's shape is contingent upon the ventricular systolic function (Fig. 1) [12] .



PLR passive leg elevating, end-expiratory occlusion (EEO).

Figure (1): Frank-Starling relationship. The Frank–Starling curve's slope is contingent upon the systolic function of the ventricular system. Subsequently, fluid responsiveness cannot be predicted using a specific level of cardiac preload. Conversely, dynamic tests involve a preload challenge, which can be either spontaneous, provoked by passive leg elevating, end-expiratory occlusion, or fluid infusion, or encouraged by mechanical ventilation. It is possible to identify preload responsiveness by observing its resulting impact on stroke volume [12].

Definition of fluid responsiveness:

Variations in SV or CO of greater than ten to fifteen percent are indicative of a positive reaction to fluids. This suggests that monitoring only blood pressure is insufficient; blood flow additionally have to be assessed. There are numerous minimally invasive monitoring systems that are capable of continuously monitoring variations in CO and SV. The detection of fluid responsiveness has been previously reliant on filling pressures, absolute values of CVP, and PAOP. However, these variables aren't accurate indicators of cardiac preload or the response of SV or CO challenge. Volumetric parameters, including global end-diastolic volume as well as right ventricle end-diastolic volume, are additionally ineffective in detecting fluid reaction. The most current consensus regarding circulatory shock as well as hemodynamic monitoring expressly don't suggests targeting fluid management on any ventricular filling pressure or volume. The initial condition that defines the need for fluids is an insufficient tissue perfusion, as evidenced through high lactate levels, extended capillary refilling time, mottled skin, hypotension, oliguria, and changed mental status. It is crucial to differentiate between the ability to respond to fluids and the overall need for fluids in the case. Therefore, it is crucial to understand how to provide an appropriate volume without exceeding it [(13)] **.**

Understanding CVP

Could CVP monitoring be utilized as a predictive tool for fluid responsiveness?

It is essential to have a good understanding of what the CVP reveals you and what it doesn't tell you, despite the fact that its predictive value is limited. Initially, CVP doesn't serve as an indicator of volume status, and a low CVP doesn't necessarily suggest the necessity for fluid. During exercise, a case may have a normal or even high cardiac output, despite having a high CVP. In a case with normal cardiac output and excess volume, the CVP may be elevated, while in an individual with minimal excess blood volume but inadequate heart function, it may be elevated. In both scenarios, the heart is likely to be operating within the flat portion of the cardiac function curve, and volume loading won't result in an elevation in CO. The patient may have a high pleural pressure or chronic pulmonary hypertension, which may result in a reduction in the compliance of the right ventricle. In this scenario, the CVP may be elevated further, which could potentially elevate cardiac output. This can result in the heart being compressed and a need for further elevated CVP values to maintain cardiac output, as elevated filtration from mediastinal and thoracic vessels will also occur. The outcome of this positive feedback mechanism is not favorable. Countless researches have established that static cardiac filling pressures are inadequate fluid reaction indicators [(14)].

Dynamic parameters

Dynamic parameters are obtained by stimulating the circulation by altering the heart's loading conditions. In point of fact, this provocation can take the shape of variations in posture or interactions between the heart and the lungs [(15)].

The great veins

The inferior vena cava and SVC's size and size variation throughout the respiratory cycle provide knowledge of their volume status [(16)].

IVC size, which is determined just distal to the hepatic vein, is correlated with RAP in cases who are spontaneously breathing. Ventilated patients exhibit little association among inferior vena cava size and RAP; although, a RAP of fewer than ten millimeters of mercury may be determined if the inferior vena cava is smaller than twelve millimeters. In cases who are spontaneously breathing, the optimal cut-off value for a RAP which is either over or under ten millimeters of mercury is two centimeters. Nevertheless, the presence of a small IVC (less than ten millimeters) indicates that fluid is tolerated [(17)].

Passive leg raising

Fluid responsiveness can be indicated through passive leg raising. The procedure involves the transfer of as much as three hundred milliliters of blood into the central circulation by tilting a case from a forty-five-degree semi-recumbent head-up position to a forty-five-degree leg-up position. Compression of the femoral veins is prevented by tipping the entire bed and not raising the legs. The SV, or VTI, across either outflow tract is assessed prior and one minute following the PLR. FR is suggested by an increase of ten percent [(18)]

Fluid challenge

It is typically known as the rapid administration of two hundred and fifty to five hundred milliliters of intravenous fluid. Echocardiography facilitates the valuation of the flow reaction to a fluid challenge. In order to obtain SV, it is sufficient to measure LVOT VTI rather than the addition of LVOT assessment. The VTI should be measured promptly before and after a fluid challenge. It is recommended that measurements be either end-expiratory or averaged more than a number of consecutive beats. Recent research has demonstrated that a mini-fluid challenge of only one hundred milliliters accurately

indicates FR by utilizing disparities in aortic VTI with TTE. It is important to acknowledge that significant enhancements in SV couldn't be evident in variations in blood pressure; nevertheless, blood flow and assessed oxygen delivery are enhanced [19].

Fluid tolerance

By evaluating signs such as a small IVC, non-dilated right heart chambers, non-displaced interventricular septum, nonappearance of right and left ventricular systolic failure, as well as nonappearance of raised LVEDP in spontaneously breathing and ventilated cases, echocardiography can assist in determining whether a fluid challenge is suitable. This indicates that fluid administration won't result in acute harm [20].

Role of ultrasound in fluid management

Ultrasound has become a tool to direct fluid therapy for critically ill cases in the past few decades. The advantages of this practice are that it gives imaging immediately, it is portable, and without the drawbacks of the ionizing radiation. Yet it is operator dependent. Using ultrasonography as a method for goal directed fluid therapy is gaining wide acceptance for capacity assessment. It gives a real time adjustment of suitable volume administration that differs among patients based on their hemodynamic parameters. It can stabilize hemodynamics to ensure smooth surgery [21].

Inferior vena cava and ultrasound measurements

Ultrasound basics:

Ultrasound is sound with frequency greater than the audible range (more than twenty thousand cycles/sec). Frequencies utilized in the clinical imaging are in the 1- to 20-megahertz (MHz) range. Ultrasound waves have a velocity of 1540 m/sec in the soft tissue (13 μsec/cm tissue back and forth for the total fly-back time of the received echoes). The velocity of sound is obtained by the bulk modulus and density of the medium. Therefore, the speed of sound in a given medium is essentially independent of frequency. The attenuation of ultrasound in soft tissue is approximately 0.75 decibel (dB)/(cm-MHz). This attenuation is primarily from absorption. Other tissues absorb sound waves less (e.g., fluids) or more (e.g., bone) than soft tissue [22].

Characteristics of ultrasound:

The term "ultrasound" refers to a type of mechanical sound energy which is transmitted via a conducting medium (such as body tissue) in the form of a longitudinal wave. In this wave, compression (high pressure) and rarefaction (low pressure) are alternately generated. A sinusoidal waveform may be used to show sound propagation. This waveform has a variety of characteristics, including a wavelength (λ), characteristic pressure (P), period (T), frequency (f), as well as velocity (speed © plus direction) (Fig. 2) [23].

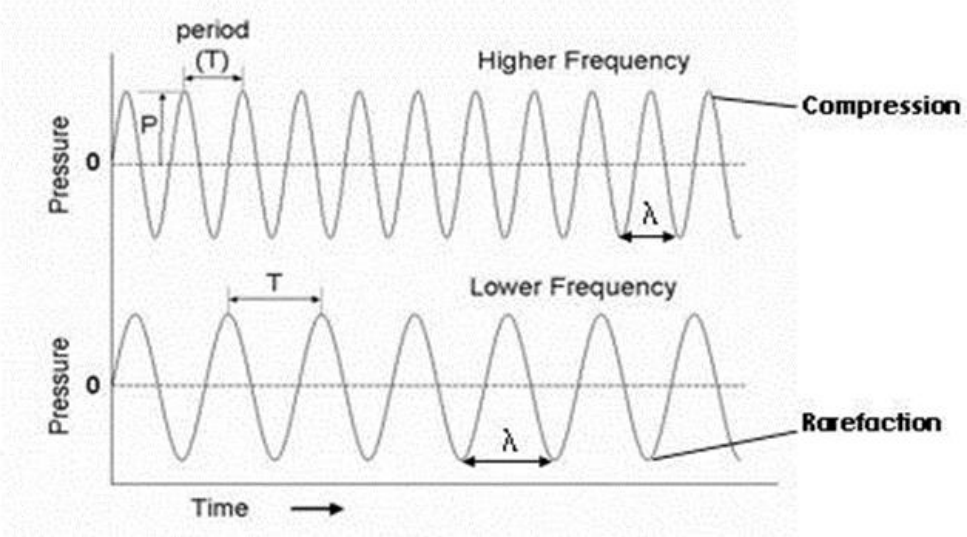


Figure (2): Characteristics of ultrasound waves [23].

The incidence of an ultrasound wave is more than twenty thousand hertz (or twenty kilohertz) and medical ultrasound frequently utilized is in the two points five-to-fifteen-megahertz range. Human hearing is in the twenty to twenty thousand hertz range [24].

Ultrasound tissue interactions:

The amplitude of the original signal is weakened as the depth of infiltration rises as the ultrasound beam travels within tissue layers. Absorption (the translation of acoustic energy to heat), reflection, and scattering at interfaces are the causes of attenuation (energy loss). Absorption, which results in the production of heat, is responsible for eighty percent of the attenuation of the sound wave in soft tissue. The attenuation coefficient of the specific tissue type is used to depict attenuation, which is measured in dB per cm of tissue. The original intensity is reduced by half when the decrease is 3 dB. The greater the attenuation coefficient, the greater weakened the ultrasound wave is through the specified tissue [(23)].

Tissue echogenicity:

Echogenicity is the brightness of a dot on a display when an echo returns to the transducer. The final image is formed by combining all the dots. Strong specular reflections create bright, hyperechoic dots, while weak diffuse reflections produce grey, hypoechoic dots. Dark, anechoic dots, resembling fluid and blood-filled structures, are generated when there is no reflection. The attenuation of deep structures frequently causes them to appear hypoechoic, resulting in weak echoes [(22)].

Transducer:

The quality of ultrasound images is influenced by the characteristics of the transducer, including its frequency and shape. The transducer frequencies employed to observe the IVC range from three to fifteen MHz. In order to generate high-resolution images, linear and curvilinear (or curved) transducers are the major advantageous for imaging of nerve. Sector-phased array transducers can additionally be developed; however, the images are considerably "grainier" [(25)].

Modern transducers are transducers which are intended to produce multiple frequencies and have a broad bandwidth. The piezoelectric transducer is greatest effective in converting electrical energy to acoustic energy and vice versa at the resonance frequency. The thickness of the piezoelectric element determines the resonance frequency [(25)].

Ultrasound measurements of IVC (Fig. 3):

The inferior vena cava is a compliant vessel that expands as well as contracts in response to alterations in pressure and volume. However the absolute inferior vena cava size differs extensively between healthy persons and it couldn't be diagnostic by itself [(26)].

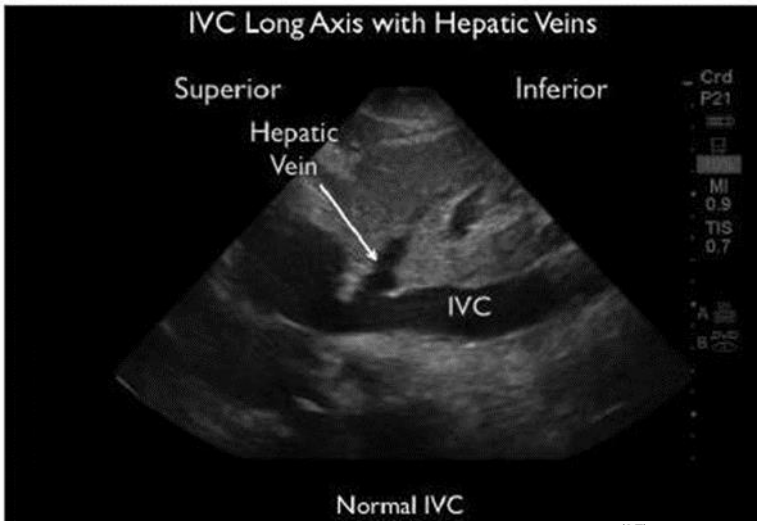


Figure (3): Ultrasound view of normal IVC [(27)].

Inspiration results in a decline in intrathoracic pressure, causing venous blood to be drawn from the lower half of the body into the right atrium. This action leads to a temporary, however normal, decline in the inferior vena cava diameter. The inferior vena cava diameter rises and subsequently returns to its baseline during expiration (Fig. 4). Respirophasic variability is the term used to describe these modifications. The maximum (expiratory) and minimal (inspiratory) inferior vena cava diameters are divided by the maximal diameter to determine the IVC-CI, also referred to as the caval index. The IVC-CI is employed to determine the right atrial pressure in cases who are spontaneously breathing. In cases with minimal respirophasic collapse, the case's inspiratory effort will be distinguished from those with raised right atrial pressure by requiring them to breath forcefully or breathe. The breath method may offer a more precise assessment of volume status; nevertheless, assessments obtained throughout normal respiration are also reasonably accurate. In critical care, fluid responsiveness is a critical concept that aims to prevent the unimportant administration of fluids, which may place the case at risk of volume overload, once a fluid challenge isn't anticipated to enhance organ perfusion and hemodynamics [(28)].

The IVC, that is situated in the retroperitoneum to the right of the aorta, is assessed via a low-incidence phased array transducer (3.5–5 megahertz).

It is distinguished by its respiratory flow variation and narrower walls. The IVC is joined by the hepatic veins and passes posterior to the liver before entering the thoracic cavity and draining to the right atrium.

There is agreement among researchers that the measurement must be taken at or near the hepatic veins' junction. The inferior vena cava is typically measured within three centimeters of the junction with the right atrium and distal to it in the majority of investigations. The inferior vena cava have to be evaluated just proximal to the hepatic veins, that are located about zero point five to three centimeters from the right atrium, regarding the American Society of Echocardiography's guidelines. The probe is located in the subxiphoid 4-chamber position to visualize the inferior vena cava. The probe indicator is oriented laterally to recognize the right ventricle and right atrium. The point at which the inferior vena cava meets the right atrium can be realized as the probe is moved closer to the spine. One should trace the inferior vena cava (IVC) downwards, paying special attention to the point where the hepatic veins join the IVC. The inferior vena cava (IVC) can also be evaluated in the long-axis orientation. The probe is repositioned from a 4-chamber subxiphoid orientation to a 2-chamber subxiphoid orientation, leading to a longitudinal view [(29)].

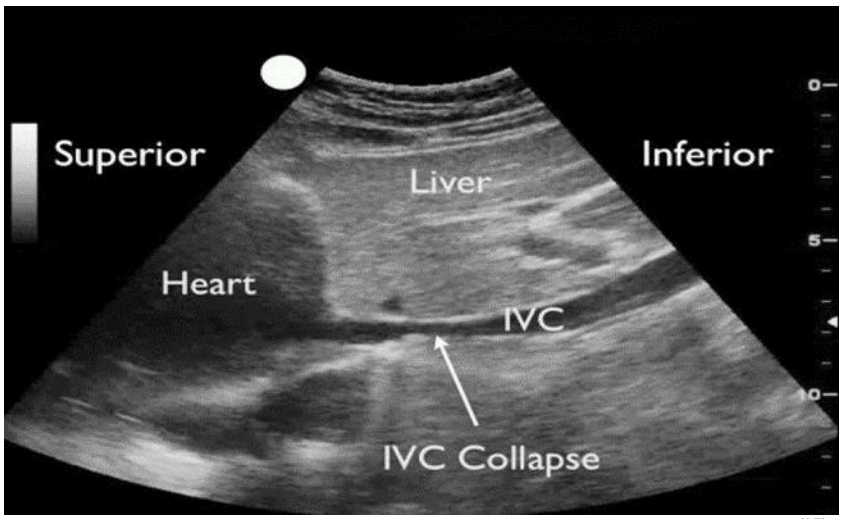


Figure (4): Ultrasound view of IVC min diameter with inspiration [(25)].

During the end of exhalation and inhalation, it is necessary to measure the diameter of the inferior vena cava at a right angle to its long axis. A small-diameter inferior vena cava that collapses significantly during inspiration (high CI) indicates low volume states. In both distributive shock and hypovolemic states, this phenomenon may be noted. In contrast, a large inferior vena cava with minimal collapse (low CI) indicates a high-volume state, like obstructive or cardiogenic shock. Particularly throughout forceful inspiration or breathing, the diaphragm's movement may cause the inferior vena cava to be displaced relative to the probe, thereby complicating the acquisition of comparative measurements at the same site. In order to identify the location measured at expiration, the probe might require to be angled inferiorly throughout inspiration in the short axis. IVC displacement may necessitate inferior and/or lateral angling in the long axis to prevent tangential measurement. The CI is a percentage, with a value near one hundred percent suggesting the likelihood of an almost complete collapse and a value near zero percent suggesting minimal collapse. Assessment of RAP is also used in the management of volume control in cases who experienced congestive heart failure. In order to prevent the inaccurate estimation of vessel size and collapse, it is advised that M-mode sonography be employed following the inferior vena cava variability has been sufficiently visualized in the B-mode. In the severely ill case that caval index is nearing the extremes, the assessment of IVC collapsibility is beneficial. Furthermore, it is possible to repeat caval sonography during resuscitation to assess the enhancement of these parameters [(29)].

Relation between IVC and right atrial pressure:

IVC is a large, high-compliance, thin-walled vein. Nevertheless, the inferior vena cava functions as a blood reservoir as a result of this compliance. The vessel's size is influenced by extrinsic factors and intrinsic visco-elastic properties as s reaction to radial hydrostatic pressure. The vessel wall's mechanical properties, especially its elasticity, are crucial. The volume-pressure connections in large abdominal veins are influenced by delayed compliance, smooth muscle creep, autonomic innervation, sympathetic vasoconstriction, vasodilatation, and circulating vasoactive substances, resulting in complex vascular response to volume infusion [(28)].

Internal jugular vein measurements

Point of care ultrasound modalities of the IJV imaging has been introduced for the valuation of the central venous pressure. **Keller *et al.*** [(31)] discussed the association of internal jugular vein aspect ratio (height/width) to predict a central venous pressure of eight-millimeter mercury in spontaneously breathing cases. The evaluation of end-expiratory internal jugular vein diameter in spontaneously breathing supine case has demonstrated great association with central venous pressure. To identify the internal jugular vein, a gentle compression is utilized to distinguish it from the carotid artery. Afterwards, the probe pressure will be relieved to prevent interfering with the internal jugular vein diameters. The IJV on the transverse axis will be noted over a single respiratory cycle. The collapsibility index will be calculated according to the formula. Limitation for using internal jugular vein collapsibility index include, the possibility of results variation according to the applied pressure, the pulsatile internal carotid artery may affect the measurements, the degree of the head of the bed elevation may cause changes in the hydrostatic pressure within the vein, causing alteration in the measurements, and difficult visualization of the vein in severely dehydrated patient [(32)].

Ethical considerations: All the procedures of the research were approved by the Anesthesia and Intensive Care Department and the Investigation Ethics Committee of Faculty of Medicine, Suez Canal University. Administrative consents required were taken. This study was performed in compliance with the Declaration of Helsinki, the code of ethics of the World Medical Association.

Declarations

- **Funding:** No fund
- **Availability of data and material:** Available
- **Conflicts of interest:** No conflicts of interest.
- **Competing interests:** None

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