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Clinical Research

THE PREDICTION OF PRELOAD RESPONSIVENESS IN MECHANICALLY VENTILATED CRITICALLY ILL PATIENTS BY THE USE OF DYNAMIC CHANGES IN HEPATIC VEIN DOPPLER VELOCITIES :- A NEW HORIZON

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ABSTRACT **Background:** Assessment of dynamic parameters to guide fluid administration is one of the mainstays of current resuscitation strategies. Each test has its own limitations, but passive leg raising (PLR) has emerged as one of the most versatile preload responsiveness tests. The objective of this study was to assess the diagnostic accuracy of hepatic vein Doppler S and D-wave velocities during PLR as a predictor of preload responsiveness. **Methods:** Prospective observational study conducted in two medical–surgical ICUs in Delhi Patients in circulatory failure and connected to controlled mechanical ventilation were included from August to December 2023. A baseline ultrasound assessment of cardiac function was performed. Then, simultaneously, ultrasound measurements of hepatic vein Doppler S and D waves and cardiac output by continuous pulse contour analysis device were performed during a PLR maneuver. **Results:** 33 patients were analyzed. 66% of the patients were preload responsive defined by a 12% increase in CO after passive leg raising. A 21% increase in the maximum S wave velocity after PLR showed the best diagnostic accuracy with a sensitivity of 69.6% (49.1–84.4) and specificity of 92.8 (68.5–99.6) to detect preload responsiveness. **Conclusions:** Hepatic vein Doppler assessment emerges as a novel complementary technique with adequate predictive capacity to identify preload responsiveness in patients in mechanical ventilation and circulatory failure.

KEYWORDS :

BACKGROUND

Fluid resuscitation is generally considered a fundamental aspect in managing critically ill patients because it is intended to help optimize cardiac output and enhance tissue perfusion [1]. However, determining the appropriate volume of fluids to administer can be challenging, as both under- and over-resuscitation can result in significant morbidity and mortality [[2], [3], [4]]. Fluid responsiveness, defined as the ability of a patient's cardiac output to increase in response to a fluid bolus, has become an important concept in guiding fluid therapy [5], both in spontaneously breathing [6] and mechanically ventilated patients [7]. This narrative review will discuss the various methods to predict fluid responsiveness in critically ill patients and their clinical applications. Although fluid responsiveness is an important concept, it is debatable whether it should be solely used to manage fluid therapy in critically ill patients.

Among them, passive leg raising (PLR) has become one of the most used preload responsiveness tests, due to its applicability in a wide array of scenarios, including patients in spontaneous ventilation, low tidal volume ventilation or presence of arrhythmias [5]. Unfortunately, the maneuver requires either real-time CO [6] measurement through devices that are not routinely available in all intensive care units (ICU) [7], or ultrasound assessment of left ventricular outflow tract–velocity time integral (LVOT–VTI) [8], which is not always feasible to measure correctly [9]. This may be accentuated during PLR's fast position change of the thorax, which makes image acquisition more challenging.

The Guytonian model of circulation defines that at a steady state, CO is equal to venous return (VR) [10, 11]. Moreover, directly assessing VR in daily clinical practice has proven elusive [11], even though VR is constantly manipulated by administering both fluids and vasopressors. Recent interest has emerged on the analysis of hepatic vein Doppler (HVD) through surface ultrasound as a novel window to assess physiological and pathological cardiovascular states [12, 13]. It has a specific Doppler envelope, where S and D waves represent antegrade blood flow to the right chambers during the cardiac cycle [14], and presents a high image acquisition rate [15]. Tus, tracking changes in HVD after a dynamic test such as PLR could effectively aid clinicians to identify significant changes in VR (and thus, CO), potentially predicting preload responsiveness. Unfortunately, there is paucity of data assessing this phenomenon.

The objective of this observational bi-centric prospective study was to assess the diagnostic accuracy of the changes of HVD S and D-wave velocities during a PLR maneuver as predictor of preload responsiveness in mechanically ventilated critically ill patients requiring hemodynamic resuscitation. We hypothesized that changes in HVD S and D-wave velocities would accurately predict preload responsiveness in this study population.

Patients

Subjects older than 18 years of age who were admitted to the ICU of Hospital in Delhi requiring controlled mechanical ventilation, in circulatory failure, and in which the attending physician performed a PLR with cardiac output assessment (PLR–CO) as part of routine clinical care were considered eligible for this study. Circulatory failure was defined as the need for vasoactive drugs to maintain MAP≥65 mmHg and the presence of at least one clinical sign of inadequate tissue perfusion, including: Lactate level>2 mmol/l, capillary refill time>3 s or a mottling score≥1.

Exclusion criteria included the impossibility to obtain an adequate hepatic ultrasound assessment; pregnancy; S-wave reversal in hepatic vein Doppler; confirmed or suspected abdominal hypertension; chronic kidney disease in hemodialysis [17]; patients with limitations of therapeutic effort; Child C cirrhosis [18]; moderate to severe tricuspid regurgitation; cardiac arrhythmias; spontaneous ventilation; extracorporeal life support; severe respiratory failure (PaO_2:FiO_2 ratio of<100 mm Hg) and/ or need of PEEP>15 cm H20.

Ultrasound Measurements

A baseline ultrasound assessment was performed as part of standard hemodynamic ICU management. This included echocardiographic measures such as color Doppler of the tricuspid valve, left ventricular outflow tract– velocity time integral (LVOT–VTI), lateral mitral E/E' ratio, tricuspid annular plane systolic excursion (TAPSE), tricuspid annular systolic velocity by tissue Doppler imaging and ejection fraction (EF), which was calculated using Simpson's biplane or Teicholz method. The middle hepatic vein (MHV) was identified from lateral or mid-subcostal view according to previously published reports [19, 20]. A phased array transducer was used with cardiac presets to facilitate study measurements. The MHV was interrogated 2–3 cm from its junction to the inferior vena cava.

Colour flow Doppler was used to identify high flow parallel to the ultrasound beam and then pulsed wave Doppler was obtained. The sample volume used was 2–3 mm, with velocity range of 65–80 mm/s, and the intercept angle between the Doppler beam and the vessels 'long axis was lower than 45°. Normal hepatic vein morphology follows a triphasic pattern, including two forward and one backward related. S-wave represents forward flow during early–mid systole, D-wave represents forward flow during early–mid diastole, and A-wave represents backward flow during late diastole [12]. The maximum velocity of D and S waves, as well as the VTI of D and S waves were recorded. All Doppler findings were obtained during the end-expiratory phase of the patient's respiratory cycle with concurrent multi-lead ECG tracings. Trained operators performed ultrasound measurements using Mindray M9 (Bio-Medical Electronics Co., Shenzhen, China).

PLR Maneuver

Whenever the attending clinician performed a PLR–CO to assess fluid responsiveness status, the technique was performed according to current recommendations [21]. Baseline patient positioning was in semi-recumbent at 45º angle and ended with the trunk in supine position and both legs elevated [22].

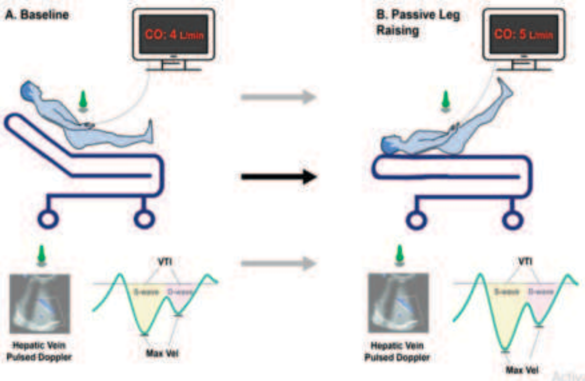

Fig 1 - Study Protocol - VTI Velocity Time Integral

Stroke volume and cardiac output were obtained with a continuous pulse contour analysis device (Argos® Cardiac Output Monitor, Retia Medical, USA). The monitors' refresh rate was set at 20 s to detect meaningful variations during the PLR maneuver. Arterial line transducer adequacy was checked before the PLR maneuver. Te HVD measurements were repeated between 1–2 min after leg elevation, at end expiration as well. We did not measure LVOT–VTI after PLR due to the limited time frame of the maneuver's effect on CO (1–2 min) [6]. Due to this small window of opportunity, we focused on obtaining proper HVD measurements. During the measurement period, adequacy of arterial line transducer and a minimum tidal volume of 8 ml/kg IBW during controlled mechanical ventilation was ensured. Patients were considered preload responders if CO increased 10% or more after PLR, as suggested by previous studies and recent expert recommendations [23–25].

Data Registry

Demographic data such as age, sex, comorbidities, Acute Physiology and Chronic Health Evaluation II (APACHE II), Sequential Organ Failure Assessment (SOFA) score, Charlson comorbidity index, diagnostic group on admission, laboratory tests, mechanical ventilation parameters [23] and clinically relevant outcomes such as ICU length of stay and 28-day mortality were registered.

Statistical Analysis

Demographic variables are presented as frequency (percentage) and mean±standard error (SD) or median [interquartile range] as appropriate, depending on data normality distribution. Receiver operating characteristics (ROC) were constructed to assess the diagnostic accuracy of HVD S and D-wave velocity and VTI changes to predict a preload responsive status. Data are presented as the area under the ROC curve (AUC–ROC)±SD (with a 95% confidence interval), sensitivity (95% CI) and specificity (95% CI). The Youden index (sensitivity+specificity – 1) was calculated to determine the best cutoff values. The gray zone approach was performed to define cases under 90% of sensitivity and specificity (diagnostic tolerance of 10%). AUC–ROC between variables were compared according to Hanley and McNeil method [26]. We performed two exploratory sensitivity analyses: first, using a 15% increase of CO cutoff after PLR, and second, using a 10% cut off on stroke volume (SV).

Considering previous reports [25, 27], we estimated the sample size by comparing an expected AUC–ROC of 0.78 curve with the null hypothesis of 0.50, assuming a two sided α error of 0.05 and power of 80%. In total, at least 36 cases were required to be included. A p value<0.05 was considered statistically significant. Data were analyzed with Graphpad Prism 10.0 statistical package.

RESULTS

From August to December 2023, we screened 50 patients for eligibility and 33 met inclusion and exclusion criteria, as shown in the study flow. Median age was of 59±15 years and most had sepsis-related circulatory dysfunction. Median SOFA score was 8 [6–10], and median norepinephrine dose at measurement time was of 0.15 [0.06–0.37] mcg/kg/min.8% (3/37) of patients were on vasopressin infusion as a second vasopressor.Regarding the tissue perfusion triggers that prompted clinicians to perform a PLR assessment, 40% of patients had abnormal lactate, 46% altered CRT, and 14% an altered mottling score [28].

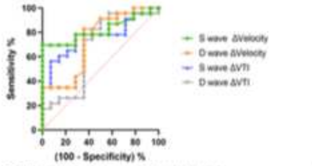

Fig. 2 Receiver operating curves of different hepatic vein Doppler parameters to identify preload responsiveness (defined by an increase of CO > 10%). VTI velocity time integral

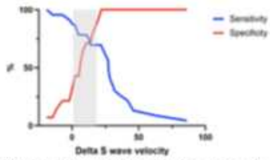

Fig. 3 Grey-zone approach of delta-velocity of S-wave hepatic vein Doppler

62% of the patients were preload responsive defined by a 10% increase in CO after PLR. the result shows key hemo-dynamic, echocardiographic and HV Doppler variables in preload responsive and unresponsive patients. In 11% (4/37) of patients, LVOT–VTI was not accessible due to the poor 5-chamber view window.

Table 1 Baseline characteristics of study population

Variable	Value
Age (years)	59 ± 15
Male (%)	54%
Diagnosis (%)	
Sepsis/septic shock	81%
Non-cardiac surgery	8%
Other	11%
SOFA score	8 [6–10]
APACHE 2 score	15 [8–22]
Charlson comorbidity index	2 [0–5]
Lactate (mmol/L)	1.5 [1–2.9]
CRT (secs)	3 [2–7]
ScvO2 (%)	83 [69–88]
Delta pCO2 a-v (mmHg)	7 [5–9]
Norepinephrine dose (mcg/kg/min)	0.15 [0.06–0.37]
CVP (mmHg)	10 [7–13]
Use of Vasopressin (%)	8%
ICU LOS (Days)	8 [4–13]
28-day mortality (%)	32%

CVP Central venous pressure, SOFA Sequential Organ Failure Assessment, ICU LOS Intensive care unit length of stay, CRT Capillary refill time, ScvO2 Central venous oxygen saturation, APACHE II Acute Physiology and Chronic Health Evaluation II

Table 2 Hemodynamic parameters and ultrasound measurements according to preload responsiveness status

	PR +	PR –	P value
Number of patients	62% (23/37)	38% (14/37)	
HR (bpm)	84 ± 21	77 ± 18	0.3
MAP (mmHg)	72 [65–77]	79 [72–88]	0.04
CVP (mmHg)	9 [8–15]	10 [7–13]	0.8
CRT (s)	4 [2–7]	3 [2–5]	0.17
NE dose (mcg/kg/min)	0.1 [0.03–0.23]	0.28 [0.08–0.56]	0.1
Stroke volume (mL)	52.5 ± 21.4	61.1 ± 15.5	0.17
Cardiac output (L/min)	4.8 ± 1.5	5.0 ± 2.1	0.25
LVOT-VTI	16.1 ± 4.3	15.1 ± 4.2	0.31
TAPSE (mm)	19.9 ± 4.1	17.2 ± 3.2	0.045
LVEF (%)	48 ± 13	51 ± 11	0.59
E/E' ratio	7.7 ± 2.5	8.5 ± 2.5	0.5
Baseline S-wave velocity (m/s)	19 [16–25.1]	21 [17–24.7]	0.34
Baseline D-wave velocity (m/s)	18.5 [13.6–17.7]	17.1 [13–20.4]	0.38
Baseline S-wave VTI (cm)	4.1 [3.5–4.9]	4.0 [3.4–5.8]	0.88
Baseline D-wave VTI (cm)	3.7 [1.9–4.6]	3.5 [2.1–4.9]	0.68

HR heart rate, MAP mean arterial pressure, CVP central venous pressure, CRT capillary refill time, NE norepinephrine, LVOT-VTI left ventricular outflow tract-velocity time integral, TAPSE tricuspid annular plane systolic excursion, LVEF Left ventricle ejection fraction, E/E' ratio mitral peak Doppler E wave to peak mitral annulus velocity ratio, VTI velocity time integral, PR preload responsiveness status

DISCUSSION

The main results of our study can be summarized as follows. A change of 20% of S-wave peak velocity or 25% of S-wave VTI may identify preload responsiveness during a PLR maneuver in sedated and mechanically ventilated critically ill patients. Analysis of D-wave velocities had worse predictive capacity.

The hemodynamic effects of a PLR maneuver have been estimated to mimic a fluid challenge of approximately 300 ml [21]. Current expert recommendations suggest performing the test from a semi-recumbent rather than from supine position, to induce shifts from two vascular beds (the legs and the splanchnic region) and enhance diagnostic accuracy [21]. This was hypothesized in the study by Jabot et al., in which sequential postural changes from semirecumbent to supine and from supine to leg elevation had similar net effect to that of the entire maneuver performed at once [22]. The authors inferred that the scaffolded approach isolated the relative contributions of both vascular territories (splanchnic and legs), but unfortunately, it wasn't measured [22]. The current study provides direct assessment on the impact of PLR on splanchnic flow through Doppler assessment, potentially confirming this hypothesis. In this sense, the fact that significant changes in flow-related measurements of the splanchnic region accurately predicted changes in CO, support that the contribution of this venous reservoir is relevant, at least in this clinical context. Future research endeavors should further investigate the relative contribution of each territory in this and other clinical contexts.

Contemporary research has assessed the accuracy of splanchnic Doppler signals to predict preload responsiveness. Cheong et al. analyzed the utility of portal vein pulsatility index (PVPi), finding that a value of PVPi>32% predicted fluid unresponsiveness with a sensitivity 30.8% (17–47.6%) and specificity of 100% (85.8–100) [29]. On the other hand, Wu et al. analyzed absolute values of S and D waves of HVD, finding higher delta S-wave velocity on patients where cardiac output increased after volume expansion as compared to those where it did not (30.1 cm/s±10.2 to 37.1 cm/s±12.5; p<0.003) [30], moreover, no diagnostic accuracy was analyzed. Our study further expands the results obtained by these previous researchers, since they aim specifically at interrogating Doppler waves that represent forward flow to the atrium and are near inferior vena cava drainage, as compared to portal vein. Thus, we believe it has a stronger physiological background and could interrogate more directly changes in VR, explaining their increased accuracy.

Multiple tests to predict preload responsiveness have focused on measuring arterial flow-related variables to identify markers of CO increase [31], such as pulse pressure or stroke volume. Only two proposed preload responsiveness tests have focused on interrogating the venous side of circulation, namely SVC and IVC diameter variability [32, 33]. However, both present technical and conceptual challenges. The former requires transesopha geal ultrasound (US) measurements [34], hindering its' widespread applicability, while the latter has multiple technical drawbacks related to insonation angle, IVC sphericity and venous compliance, limiting its'diagnostic accuracy [35]. The novel approach proposed in this study, which focuses on blood flow variations measured through pulsed Doppler rather than anatomical variations, such as diameter, could help overcome these issues, and approach VR changes indirectly. Due to the angulation required, obtaining fow patterns from the IVC is not feasible, but the HV provides an adequate US assessment site that drains directly into the IVC without major valves or obstructions, becoming a valuable and emerging window for hemodynamic assessment.

Recently, the visual inspection of HVD has also been studied as a marker for venous congestion or food intolerance, being a pivotal component of the VExUS score [36]. S-wave reversal, which represents retrograde flow, has been proposed as a marker of hepatosplanchnic congestion. Altered VexUS has correlated to key hemo dynamic parameters such as high CVP values [37], and could predict the risk of acute kidney injury in different contexts. HVD has also been used as a marker of venous congestion in heart failure contexts with prognostic implications as well [38]. As proposed by Kenny et al., the dynamic assessment of waveform patterns (i.e., increase in D wave or reversion of S wave) through a PLR maneuver or fluid challenge could aid in identifying potential congestion [39, 40]. Tus, HVD could be a fairly unique tool able to assess not only fluid responsiveness as seen in this manuscript, but also assess fluid tolerance, which is of growing clinical importance [41, 42]. Moreover, future research efforts should answer these areas of uncertainty.

Of note, in this cohort, preload responsive patients had significantly higher baseline TAPSE readings as compared to non-responders (although median values were within normal range). Tus, it could be argued that the difference in preload responsiveness status was determined because patients had better right ventricular function, determining a steeper cardiac function curve. Recent reports such as Zhang et al. have shown that TAPSE correlates with HVD-derived indexes such as the systolic filling fraction [S-wave velocity/(S-wave+D-wave velocities)] [20]. Our results show that delta S-wave velocity correlates with TAPSE (Additional File 3). Both phenomena could be explained by the fact that during ventricular systole the tricuspid annulus moves inward to the cardiac apex, which causes a further anterograde flow during systole (thus reflected in the S wave of HVD) [14]. Moreover, this study was not designed to address this question, and clinical interpretation should be made with caution considering other key echocardiographic results obtained, but certainly it deserves further exploration. This study has several strengths. First, it's bicentric nature. Second, delta S-wave velocity presented an adequate AUC–ROC (>0.8), with less than 40% of measurements in the gray zone of diagnostic tolerance. This becomes particularly relevant when considering the easiness of image acquisition, as compared to LVOT–VTI. In fact, Spiegel et al. only reported 7.9% of inadequate HVD windows in a cohort of patients admitted to ICU [43], a similar rate to that shown by Prager et al. in a cohort of septic shock patients [15]. Even though our study was not designed to assess image acquisition feasibility of HVD, we excluded only 4% of patients for this reason out of the eligible patients screened.This contrasts significantly to the prevalence of inadequate windows to obtain LVOT–VTI, which can amount up to 22% [44, 45].

Added to the non-invasive nature of the technique, no requirement of CO monitor and its'easier learning curve, HVD could emerge as a valuable tool at the bedside assessment of critically ill patients. This study has several limitations. First, as all ultrasound-based assessments, the technique is operator dependent and intra or inter-observer variability could occur. Second, we decided to assess changes in CO through a PLR rather than the more classic design of administering a fluid challenge [25] to assess fluid responsiveness. Even though the fluid challenge has been praised as a gold-standard technique, both the physiological determinants of PLR (±300 ml of autotransfusion) and the diagnostic accuracy of its' derived measurements (sensitivities and specificties > 85%) [24], provide a comparable alternative. This approach has already been used in the contemporary literature of diagnostic accuracy of preload responsiveness tests [46, 47], and has the added benefit of avoiding potential deleterious fluids [48, 49] to patients which are preload unresponsive. Another potential criticism could be the use of non-calibrated CO monitors such as those used in this study. Moreover, the technical determinants (i.e., pulse contour analysis) for tracking relative changes of CO are the same as those used in transpulmonary thermodilution (TPTD), and the least significant changes of CO variability has been estimated around 1%, significantly under our detection threshold of PR of 10% [50]. The multi-beast pulse contour monitoring used has a short refresh rate (20 s), and presents an adequate ability to capture quick and relative changes such as those happening during a PLR, as shown in recent studies [51, 52]. There was a predominance of septic patients, which could present with higher rates of preload responsiveness [53]. Thus, this could preclude extrapolation to other diagnostic groups of circulatory failure. Since our study was the first assessment of the diagnostic accuracy of this novel technique, we decided to avoid potential physiological confounders, such as right sided valvular diseases, light sedation for patients with respiratory efforts, which could introduce bias or error and preclude a correct interpretation of results.

Future studies should confirm these results and test the diagnostic capacity of HVD in broader contexts of critical illness, such as patients with spontaneous breathing, light sedation, arrhythmias or valvular diseases. Aswell, the usefulness of the dynamic assessment of food intolerance through identification of flow patterns of S and D waves in concomitance with preload responsiveness could aid clinicians on identifying the best balance between risk/benefit ratio on fluid administration. Finally, future studies could compare, through hand-tracking devices, the difference in the number of movements and distance traveled between HVD and LVOT–VTI during a PLR maneuver [54].

In conclusion, in sedated and mechanically ventilated critically ill patients, dynamic changes of S-wave on HVD after a PLR maneuver had a suitable predictable capacity to identify preload responsiveness. This technique could become valuable in scenarios of basic hemodynamic monitoring and when cardiac US is not feasible. Future studies should confirm these results and their relationship with dynamic venous congestion assessment.

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