

Original Research Article

ASSESSMENT OF CAROTID DOPPLER INDICES AS A SURROGATE OF FLUID RESPONSIVENESS FOLLOWING PASSIVE LEG RAISE MANOEUVRE

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ABSTRACT

Background: Fluid resuscitation remains central to ICU management but risks fluid overload, complicating outcomes in critically ill patients. Passive leg raising (PLR) is a reversible preload challenge used to assess fluid responsiveness. Carotid Doppler indices, such as corrected carotid flow time (ccFT) and peak systolic velocity (Vmax), may provide non-invasive surrogates of cardiac output (CO) changes, but evidence remains limited. The objective is to assess the correlation between PLR-induced changes in cardiac output (CO) and carotid Doppler indices (ccFT and Vmax) as surrogates of fluid responsiveness in mechanically ventilated ICU patients.

Materials and Methods: This prospective pre-post observational study included 50 mechanically ventilated ICU patients aged 20–60 years. CO was measured using non-invasive esCCO monitoring, and ccFT (using Wodey's correction) and Vmax were recorded via carotid Doppler before and 1 minute after PLR. Patients with valvular heart disease, pregnancy, atrial fibrillation, or inability to tolerate PLR were excluded. Statistical analysis assessed changes in ccFT and Vmax with CO following PLR.

Results: Among 50 patients, 72% were fluid responders ($\geq 10\%$ CO increase post-PLR). Responders showed a mean CO increase of $19.0 \pm 8.0\%$ versus $4.0 \pm 3.0\%$ in non-responders ($p < 0.0001$). $\Delta ccFT$ was significantly higher in responders (49.4 ± 46.4 ms) compared to non-responders (12 ± 18.8 ms; $p = 0.0009$). ROC analysis for $\Delta ccFT$ showed an AUC of 0.71 ($p = 0.024$), with a cut-off > 7.58 ms yielding 71.4% sensitivity and 75% specificity. No significant changes in Vmax were observed between groups ($p = 0.2527$).

Conclusion: PLR-induced changes in ccFT correlate significantly with CO changes, supporting ccFT as a reliable non-invasive surrogate for fluid responsiveness assessment. However, Vmax showed no significant correlation. Further large-scale studies are warranted to validate carotid Doppler indices as standard bedside tools for fluid responsiveness assessment in critical care.

Keywords: Fluid responsiveness, passive leg raise, carotid Doppler, corrected carotid flow time, peak systolic velocity, cardiac output, critical care.

INTRODUCTION

Fluid resuscitation is central to ICU management but carries risks of fluid overload. Despite Rivers et al.^[1] demonstration of benefits from aggressive fluid administration in sepsis, fluid overload is associated with increased mortality, prolonged ventilation, and

worsened outcomes in ARDS, intra-abdominal hypertension, and AKI.^[2-5] The Frank–Starling relationship complicates prediction of fluid responsiveness; CVP has proven unreliable, yet remains widely used. Dynamic markers like PPV and SVV are better but require invasive monitoring.^[6-10]

Passive leg raising (PLR) has emerged as a reversible, repeatable fluid challenge inducing a temporary preload increase equivalent to 300–500 mL fluid bolus, without risks of overloading. Meta-analyses report PLR has pooled sensitivity of 85–88% and specificity of 89–91% when CO is assessed in real-time.

Currently, no fully non-invasive method reliably assesses fluid responsiveness. Echocardiographic LVOT VTI changes post-PLR correlate with CO changes but require high skill. Carotid Doppler indices – corrected carotid flow time (ccFT) and peak systolic velocity (Vmax) – have emerged as promising surrogates, given their ease of acquisition and non-invasiveness. This study aimed to correlate changes in ccFT and Vmax with CO following PLR to assess their utility as surrogates for fluid responsiveness in ICU patients.

Primary Objective: Correlate PLR-induced changes in CO and ccFT.

Secondary Objective: Correlate PLR-induced changes in CO and carotid Vmax.

MATERIALS AND METHODS

A prospective pre-post observational study was conducted in 50 mechanically ventilated ICU patients aged 20–60 years at J.N. Medical College Hospital, Aligarh (2018–2020). Exclusion criteria included valvular heart disease, pregnancy, limb amputation, atrial fibrillation, atherosclerosis, and inability to tolerate PLR. Written informed consent was taken.

Methodology

- CO Measurement: esCCO (NIHON KOHDEN, Tokyo) providing continuous, non-invasive CO estimation using ECG, SpO₂, and BP.
- Carotid Doppler: 10-5 MHz linear probe (SONOSITE M-Turbo) measured ccFT (using Wodey's correction) and Vmax. Measurements were taken pre-PLR and at 1 minute post-PLR, the time of maximal blood flow.

A learning curve of >20 carotid Doppler measurements was ensured under radiologist supervision before study initiation. Data analysis was blinded.

RESULTS

Demographics

Table 1: Summarises age and gender distribution

Parameter	Responders (n=36)	Non-responders (n=14)	p-value
Mean age (years)	38.2 ± 4.2	36.5 ± 2.6	0.17
Male (%)	53%	71%	0.23
BMI (kg/m ²)	24.6 ± 8.5	21.7 ± 7.0	0.26

No significant demographic differences were observed.

Primary Findings

1. Change in CO:

- Responders showed a mean increase of 19.0 ± 8.0% post-PLR vs 4.0 ± 3.0% in non-responders (p<0.0001).
- 72% patients were fluid responders (>10% CO increase).

2. Change in ccFT (Wodey's correction):

- Mean ΔccFT in responders: 49.4 ± 46.4 ms
- Mean ΔccFT in non-responders: 12 ± 18.8 ms
- Statistically significant (p=0.0009).
- ROC analysis AUC = 0.71 (p=0.024) indicating fair predictive value with a cut-off ΔccFT >7.58 ms showing 71.4% sensitivity and 75% specificity.

3. Change in Vmax

- No significant difference observed between responders and non-responders (p=0.2527).

The study was conducted on 50 patients of either sex, aged between 20–60 years, requiring mechanical ventilation in the ICU. Patients with any valvular heart disease or thrombophlebitis of the upper limbs were not included in this study.

All patients meeting the inclusion criteria were assessed for PLR induced changes in CO, ccFT and Vmax. Later correlation between the changes in carotid Doppler indices (ccFT and Vmax) with the change in standard non-invasive cardiac output (CO) induced by PLR was studied.

Table 2: showing the diagnosis of patients included in the study

	Diagnosis	No. of patients
Gynaecological patients	• Post-normal delivery with shock • Post DNC with shock • Pyoperitoneum • Post- partum haemorrhage • Post delivery case of pregnancy with abruptio placentae. • Pyoperitoneum with sepsis with • AKI	4(08%) 2(04%) 2(04%) 3(06%) 2(04%) 2(04%) TOTAL=15(30%)
Surgical patients	• Fever with altered sensation • Ischemic CVA with systemic HTN with T2DM • Complicated malaria • CLD with altered sensation	1(02%) 2(04%) 1(02%) 2(04%) 1(02%)

	- CLD with HTN with left sided pneumonia. - Age with shock	3(06%) TOTAL=10(20%)
Neurological patients	RTA with HI	2(4%) TOTAL = 2(4%)
Medical patients	- Fever with altered sensation - Ischemic CVA with systemic HTN with T2DM - Complicated malaria - CLD with altered sensation - CLD with HTN with left sided pneumonia. - Age with shock	1(02%) 2(04%) 1(02%) 2(04%) 1(02%) 3(06%) TOTAL=10(20%)
	- Snake bite - Poisoning	2(4%) 2(4%) TOTAL=4(8%)
Respiratory disease	- COPD with type 2 respiratory failure - B/L bronchopneumonia	2(4%) 2(4%) TOTAL=4(8%)
Renal disease	CKD with sys HTN	2(4%) TOTAL=2(4%)

Out of 15[30%] gynaecological patients, majority of patients were of post-normal delivery with shock[n=4(08%)] followed by postpartum haemorrhage[n=3(06%).

In 11[22%] Surgical patients, majority of patients were of postoperative case of perforation peritonitis [3(06%)] followed by chronic pancreatitis 2(04%), post-operative case of ruptured liver abscess 2(04%) and follow up case of of pyoperitonium 2(04%).

Out of 10(20%) medical patients, majority of patients had Acute gastroenteritis (AGE) with shock [3(06%)

followed by Chronic Liver Disease (CLD) with altered sensation [2(04%)] and Ischemic CVA with systemic HTN with T2DM[2(04%)].

Out of 6 patients, majority had poisoning [4(08%)] followed by snake bite [2(06%)].

Out of 4 respiratory disease patients, two had COPD with type 2 respiratory failure [2(04%)] and other two had B/L bronchopneumonia [2(04%)].

Both the patients of renal disease had CKD with system HTN [2(04%)].

Table 3: Demographic and clinical information of the patients:

Patient Characteristics	Total Patients [n=50]	Responders [n=36]	Non-responders [n=14]	p-Value
Hematocrit, mean $\pm$ SD %	28.23 $\pm$ 6.88	29.15 $\pm$ 7.86	27.97 $\pm$ 6.03	p=0.6154
Positive end-expiratory pressure > 5 cm H ₂ O, %	8(16%)	5 (13.9%)	3 (21.43%)	--
Pressor used, % Noradrenaline	20(40%)	16 (44.4%)	4 (28.57%)	X=2.05 p=0.7266
Combination	5(10%)	4 (11.1%)	1 (7.14%)	
-Adrenaline (Noradrenaline + Adrenaline)	2(4%)	1 (2.8%)	1 (7.14%)	
-Dopamine (Noradrenaline + Dopamine)	2(4%)	1 (2.8%)	1 (7.14%)	
-Vasopressin (Noradrenaline + Vasopressin)	1 (2%)	1 (2.8%)	0	p=0.5151
APACHE-II, mean $\pm$ SD	22.16 $\pm$ 9.21	23.83 $\pm$ 9.85	21.84 $\pm$ 9.03	

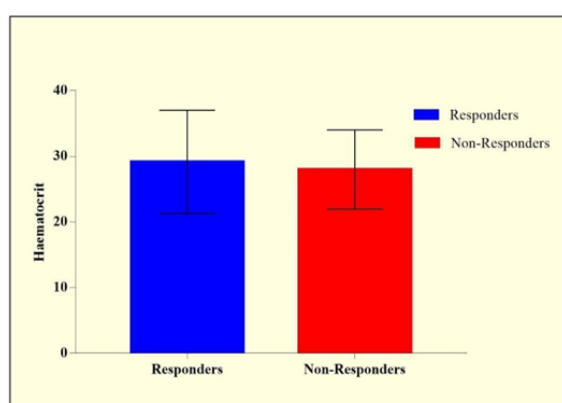


Figure 1: Graphical representation of haematocrit in responder and non-responder

The mean hematocrit was 29.15 $\pm$ 7.86 and 27.97 $\pm$ 6.03 in responder and non-responder respectively. The difference was statistically insignificant (p=0.6154).

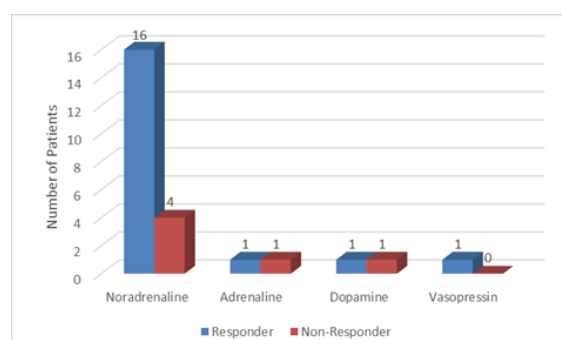


Figure 2: Graphical representation based on type of pressor used.

Noradrenaline was used in 44.4% in responders and 28.57% in non-responders. However, the combinations of pressors were more in non-responders. Although insignificant difference (p=0.726) was observed.

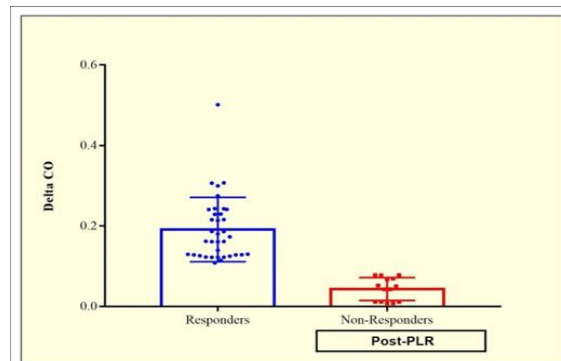
Table 4: Tabular representation of Cardiac Output between the Responders and Non-Responders.

Variables	Total	Responders [n=36]	Non- responders [n=14]	p-Value
Non-Invasive Cardiac Output Monitor				
Pre PLR CO, mean $\pm$	5.07 $\pm$	4.90 $\pm$ 1.38	5.31 $\pm$ 0.38	p=0.6520
SD	1.11			
Post PLR CO, mean $\pm$	5.63 $\pm$	5.78 $\pm$ 1.44	5.54 $\pm$ 0.46	p=0.5459
SD	1.13			
Change in CO, mean $\pm$ SD,%	15.0 $\pm$ 10.0	19.0 $\pm$ 8.0	4.0 $\pm$ 3.0	p<0.0001*

Mean Pre-PLR CO among responders and non-responders were 4.90 $\pm$ 1.38 and 5.31 $\pm$ 0.38 respectively. No statistical significant difference (p=0.6520) was found while analysing the mean pre-PLR CO among responders and non-responders group.

The bar-chart shows the mean value. Error bars at the edge of each bar represents standard deviation (SD) of the data at 95% Class-Interval. Each dot represents the individual patient value.

The mean Δ CO among responders and non-responders were 19.0 $\pm$ 8.0 and 4.0 $\pm$ 3.0 respectively and the mean difference was statistically significant (p<0.0001).

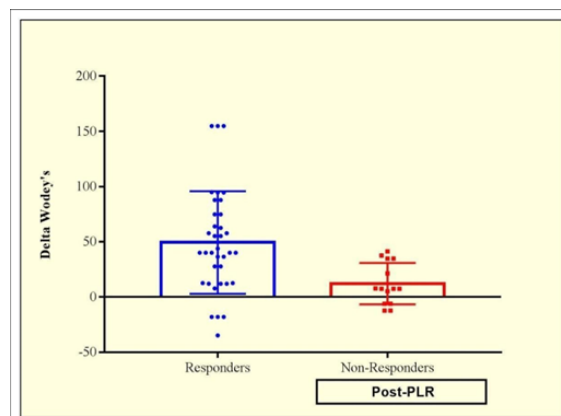
**Figure 3: Graphical representation of Change in mean Δ CO between Responder and Non-responder****Table 5: Tabular representation of mean ccFT (Wodey's) between the Responders and Non-Responders.**

	ccFT (WODEY'S) Pre-PLR		
	Responders [n=36]	Non- Responders [n=14]	P-value
Mean $\pm$ SD, m/s	308.4 $\pm$ 44.38	330 $\pm$ 34.44	U=214 p=0.4191
Minimum	214.5	275.5	
Median	318	329.2	
Maximum	376.8	388.1	
POST-PLR			
Mean $\pm$ SD, m/s	331.4 $\pm$ 69.11	333.5 $\pm$ 47.76	U=172 p=0.0849
Minimum	137.1	263.2	
Median	344.2	320.3	
Maximum	397.4	392.5	
Changes in ccFT (Wodey's) before and after PLR	Δ ccFT(using WODEY'S formula)		U= 103.5 p= 0.0009*
	49.38 $\pm$ 46.43	12 $\pm$ 18.8	

Mean ccFT (using Wodey's formula) Pre-PLR between Responder and Non-responder was 308.4 $\pm$ 44.38 and 330 $\pm$ 34.44 m/s respectively. The difference in ccFT (using Wodey's formula) in terms of measures as mentioned above, between Responders and Non-responders was not significant both pre-PLR (p=0.4191) and post-PLR(p=0.0849).

The bar-chart showed the mean value. Error bars at the edge of each bar representing standard deviation (SD) of the data at 95% Class-Interval. Each dots represents the individual patient value

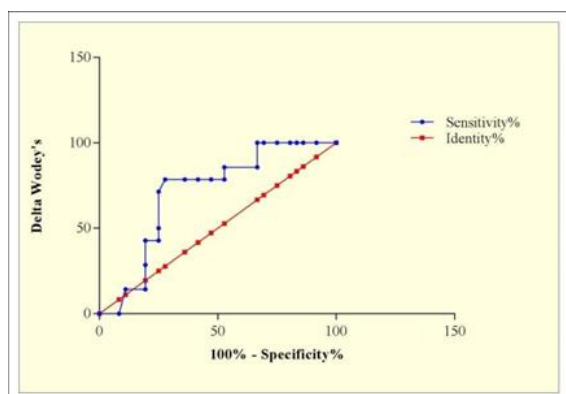
The Δ ccFT (Wodey's) before and after PLR between Responder and Non-responder was 49.38 $\pm$ 46.43 and 12 $\pm$ 18.8 m/s. Statistically significant difference (p=0.0009*) was found while analysing the Δ ccFT (Wodey's) before and after PLR between Responder and Non-responder group.

**Figure 4: Graphical representation of Mean Δ ccFT (using Wodey's formula) between Pre-PLR and post PLR in Responder and Non-responder.****Table 6: Tabular representation of ROC analysis of Δ ccFT (Wodey's).**

Δ ccFTArea under the ROC curve	
Area	0.7073
Std. Error	0.07553
95% confidence interval	0.5593 to 0.8554
P value	0.0240*

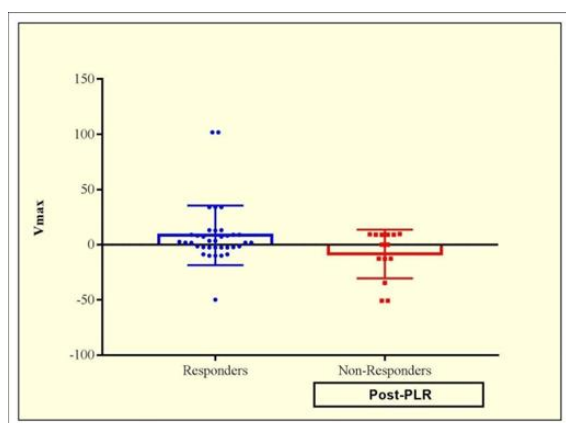
Table 7: Tabular representation of mean Vmax between the Responders and Non-Responders.

	Vmax Pre-PLR		
	Responders [n=36]	Non- Responders [n=14]	P-value
Mean $\pm$ SD, cm/s	68.63 $\pm$ 37.71	85.21 $\pm$ 27.87	U=172 p=0.0849
Minimum	17.9	42.6	
Median	54.65	82.95	
Maximum	130.4	128.4	
POST-PLR			
Mean $\pm$ SD, cm/s	77.07 $\pm$ 44.4	76.74 $\pm$ 33.19	U=238 p=0.7690
Minimum	19.7	8	
Median	68.85	84.1	
Maximum	184.3	115.7	
Changes in Vmax before and after PLR	Δ Vmax		U= 198.5 p= 0.2527
	8.436 $\pm$ 27.04	-8.479 $\pm$ 22.05	

**Figure 5: Graphical representation between ROC analysis of Mean Δ ccFT (Wodey's)**

Receiver Operating characteristic (ROC) curve was analysed for ccFT (Wodey's) showed maximum area under the curve of 0.7073 at 95% confidence interval and found to be significant ($p=0.0240^*$)

Mean Vmax Pre- PLR between Responder and Non-responder was 68.63 ± 37.71 and 85.21 ± 27.87 cm/s respectively. No statistical significant difference ($p=0.0849$) was found while analysing the mean Vmax Pre- PLR between Responder and Non-responder group

**Figure 6: Graphical representation of Mean Δ Vmax between Pre- PLR and post PLR in Responder and Non-responder**

The bar-chart shows the mean value. Error bars at the edge of each bar represents SD of the data at 95% Class-Interval. Each dot represents the individual patient value.

Change in Vmax before and after PLR between Responder and Non- responder was 8.436 ± 27.04 and -8.479 ± 22.05 cm/s. No Statistical significant difference ($p=0.2527$) was found while analysing the changes in Vmax before and after PLR between Responder and Non-responder group.

DISCUSSION

This study confirms PLR is a safe, reversible predictor of fluid responsiveness. A significant positive correlation between Δ ccFT and Δ CO suggests ccFT can serve as a non-invasive surrogate marker of preload responsiveness. Findings are consistent with Barjaktarevic et al. (2018), who demonstrated ccFT changes correlate with CO increases post-PLR with high predictive accuracy. Conversely, Vmax changes were not significant, potentially due to insufficient fluid shift in hypovolemic patients or sample size limitations.^[11-18]

Study Limitations

- Unblinded investigator during data collection, though blinded reanalysis ensured reliability.
- Operator skill may influence Doppler measurements.
- Small sample size limits generalisability of Vmax findings.

CONCLUSION

- PLR reliably predicts fluid responsiveness by transiently increasing preload.
- ccFT (Wodey's corrected) changes correlate well with CO changes post-PLR and can serve as a non-invasive, bedside surrogate marker.
- Vmax changes were not significant in this cohort.
- Further multicentric studies are needed to validate carotid Doppler indices as standard fluid responsiveness predictors in diverse ICU populations.

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