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Oscillometric Blood Pressure in Predicting Volume Responsiveness Assessed By Passive Leg Raising Test

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Abstract

Introduction: The PLR test can be used to assess if cardiac function will improve with fluid administration, PLR is considered fluid challenge. The aim of the study is to show the role of non-invasive blood pressure in predicting volume responsiveness assessed by passive leg raising test.

Patients and method: This study was conducted on 30 adult mechanically ventilated patients. Initially, while the patient is in the semirecumbent position, heart rate and brachial artery blood pressure were recorded, and stroke volume was obtained using transthoracic echocardiography. Then PLR was performed by changing the patient from the semirecumbent position to flat position with the lower limbs lifted at 45°, and another set of measurements were recorded when the PLR had induced its maximal effects, i.e., within 1 min. Volume responsiveness was defined as an increase in CO >10 % after PLR

Results: as regard areas under the ROC curves, Δ systolic blood pressure and Δ pulse pressure were significant (p =0.008, 0.012) respectively

Conclusion: changes in non-invasive oscillometric pulse pressure and systolic BP induced by PLR showed moderate diagnostic ability for prediction of fluid responsiveness in mechanically ventilated patients.

Keywords: PLR passive leg raising test, pulse pressure, stroke volume, cardiac output, blood pressure.

Introduction

Maintaining an optimum volume status is of great importance in managing the critically ill patient. Clinical researches recommend fluids but a simple, harmless and repeatable method to evaluate volume responsiveness is still ambiguous. static parameters as CVP, while seemingly universal in practice[(1)], do not monitor the intravascular status [(2)]. That is why the passive leg raising (PLR) test has developed.

The PLR test can be used to assess if cardiac function will improve with fluid administration. By auto transfusing a volume of150-300 mL of blood from the peripheral to the central circulation, PLR is considered fluid challenge. This test is safe as no extra volumeis given and the cardiovascular response are rapidly reversible, thereby escaping the complications of volume overload[(3)]. The impact of PLR on CO is short and temporary and lasts for few minutes, since the intravascular fluid status overall is fixed and its distribution within the blood vessels rapidly relocates. The hemodynamic device must monitor rapid and short-lasting variables.[(3)]

In case of unavailable haemodynamic monitor the impact of passive leg raising test on cardiac output is usually evaluated by effect on invasive blood pressure value.However, the preliminary hemodynamic assessment of unstable patients often depends entirely on non-invasive blood pressure. Whether evaluating systemic consequence of PLR using non-invasive blood pressure on cardiac output is unknown[(4, 5)].

The aim of the study is to show the role of non-invasive blood pressure in predicting volume responsiveness assessed by passive leg raising test.

Patients and Methods

This study was conducted on 30 adult mechanically ventilated patients admitted to critical care medicine department in tertiary care institute. The legal representative of each patient gave informed consent before start of the study and the protocol was approved by the institution alethics committee. Patients suffered from hypoxia, central nervous system pathology, amputated leg, pregnant female and patients with intraabdominal hypertension were excluded.

Procedure

Initially, while the patient was in the semirecumbent position, heart rate and brachial artery oscillometric blood pressure were recorded from the bedside monitor, and stroke volume was obtained using transthoracic echocardiography. Then PLR was performed by changing the patient from the semirecumbent position to flat position with the lower limbs lifted at45°, and another set of measurements were recorded when the PLR had induced its maximal effects, i.e., within 1 min. Volume responsiveness was defined as an increase in CO >10 % after PLR.

Cardiac Output measurement

In the apical five chamber view, the aortic velocity time integral (VTI) was measured by pulsed wave Doppler. In the parasternal long axis view the aortic annulus diameter was measured. Aortic area was calculated as ($\pi \times$ annulus diameter^2)/4. Stroke volume was estimated as velocity time integral × Aortic area. Cardiac output (CO) was determined as stroke volume × heart rate.

Data collected:

The following clinical data were recorded: age, gender, ICU admission diagnosis, Acute Physiology and Chronic Health Evaluation (APACHE) II score, and the use of vasopressor drugs.

Statistical analysis

Data are presented as median with interquartile range (IQR). Comparisons between responders and non-responders were done using the Mann–Whitney U test. Accuracy of oscillometric blood pressure parameters in predicting volume responsiveness was assessed by calculating the area under the receiver operating characteristic (ROC) curve. Data were analyzed by Medcalc, Version 18.0. All hypotheses were constructed two-tailed and $P \leq 0.05$ was considered significant.

Results

Median age of our patients was 59 year, 57% of the patients were. 7 patients were diagnosed as cardiogenic shock, 8 respiratory failure, 8 septic shock, 2 acute kidney injury and 5 trauma patients. The median of APACHE II score was 13, (table-1).

Table 1: Patients characteristics

Study variable	Median (IQR) / Frequency (%)
Age *(years)*	59 (46 – 69)
Male sex	17 (56.7)
Diagnosis	
Cardiogenic Shock	7 (22.6)
Respiratory Failure	8 (25.8)
Septic Shock	8 (25.8)
AKI	2 (6.5)
Trauma	5 (16.1)
Vasopressor Dose*(µg/ kg/min)*	7 (5 – 13)
APACHE-II score	13 (10 – 15)

As regard hemodynamic data at baseline, we found 16 (53.3%) patients were non-responders and 14 (46.7%) were responders, there were no statistically difference between both groups as regard HR, SBP, DBP, MAP, SV and CO. While there is statistically significant difference between responder and non-responder in pulse pressure at baseline (p = .019), (table 2)

Table 2: Hemodynamic Data at baseline

	Non-Responders (*n* =16)	**Responders** (*n* =14)	***P***
Heart Rate (HR)	94 (81 – 104)	98 (87 – 111)	0.448
Systolic BP *(SBP,mm Hg)*	110 (94 – 117)	99 (81 – 119)	0.101
Diastolic BP *(DBP ,mm Hg)*	63 (59 – 68)	59 (51 – 76)	0.822
Mean arterial pressure *(MAP ,mmHg)*	82 (70 – 84)	72 (61 – 90)	0.473
Pulse Pressure *(PP,mm Hg)*	49 (39 – 56)	40 (31 – 41)	0.019*
Stroke Volume *(SV ,ml)*	71 (64 – 79)	67 (58 – 72)	0.093
Cardiac Output *(CO ,L/min)*	6.6 (5.4 – 7.7)	6 (5.1 – 6.7)	0.224

BP=Blood Pressure
(*) *P*< 0.05 is significant

After PLR, no significant difference between fluid responders and non-responders as regard SBP, DBP, MAP, PP, SV and CO, (table-3).

While changes induced by PLR in SBP, PP, SVand CO were statistically significant difference among responders and non-responders, (table-4).

Table 3: Hemodynamic Data after PLR

	Non-Responders (*n* =16)	**Responders** (*n* =14)	***P***
Heart Rate (HR)	94 (83 – 102)	100 (87 – 113)	0.275
Systolic BP *(SBP , mmHg)*	112 (93 – 115)	100 (81 – 115)	0.473
Diastolic BP *(DBP,mmHg)*	64 (56 – 64)	56 (48 – 66)	0.552
Mean arterial pressure *(MAP,mmHg)*	79 (68 – 80)	73 (59 – 82)	0.400
Pulse Pressure *(PP ,mmHg)*	48 (40 – 51)	42 (32 – 50)	0.294
Stroke Volume *(SV,ml)*	73 (68 – 82)	74 (66 – 78)	0.790
Cardiac Output *(CO ,L/min)*	6.5 (5.6 – 8)	6.9 (5.7 – 7.7)	0.854

BP=Blood Pressure
(*) *P*< 0.05 is significant

Table 4: Changes in hemodynamic data after PLR

	Non-Responders (*n* =16)	Responders (*n* =14)	*P*
Δ Heart Rate *(%)*	0.97 (-1.4 to 2.4)	1.97 (-0.9 to 3.4)	0.142
Δ Systolic BP *(%)*	-2.5 (-4.7 to -2.2)	-2.2 (-2.5 to 4.1)	0.025*
Δ Diastolic BP *(%)*	-5.2 (-5.9 to 7.5)	-5.2 (-5.9 to -5)	0.886
Δ MAP*(%)*	-4.1 (-5.3 to 1.1)	-2.7 (-3.9 to -0.6)	0.608
Δ Pulse Pressure *(%)*	0 (-16 to 5.3)	7.8 (1.5 to 22.1)	0.038*
Δ Stroke Volume *(%)*	6.2 (0 to 6.7)	11.1 (9.6 to 13.6)	<0.001*
Δ Cardiac Output *(%)*	5.6 (1.3 to 7.8)	12.9 (10.9 to 15.7)	<0.001*

BP=Blood Pressure, MAP=Mean Arterial Pressure
(*) *P*< 0.05 is significant

As regard areas under the ROC curves, Δ systolic blood pressure and Δ pulse pressure were significant (*p* =0.008, 0.012) respectively, (table-5, figure-1).

Table 5: Areas under the receiver operating characteristic curves (AUC) in predicting fluid responsiveness

	AUC (95% CI)	Cut-off point	Sensitivity %	Specificity %	*P*
Δ Systolic BP	0.739 (0.547 – 0.881)	> -3.4%	73.7	85	0.008*
Δ Diastolic BP	0.540 (0.350 – 0.723)	≤ -4.7%	85.7	43.7	0.713
Δ Pulse Pressure	0.743 (0.552 – 0.884)	> 1.7%	78.6	68.7	0.012*
Δ MAP	0.556 (0.364 – 0.736)	> -4.2%	85.7	50	0.630

BP=Blood Pressure, MAP=Mean Arterial Pressure
(*) *P*< 0.05 is significant

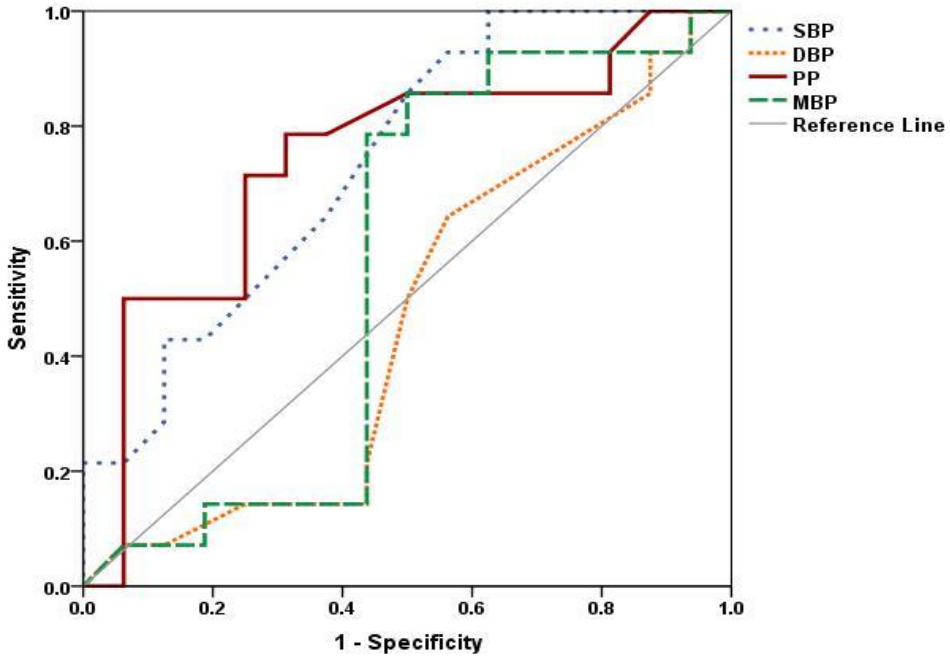

Figure 1: ROC curves for prediction of fluid responsiveness by non-invasive blood pressure monitoring

Discussion

The central result of our research is that the passive leg raising test induced change innon-invasive systolic BP and pulse pressure had a moderate discriminative ability between responders and non-responders in critically ill patients.

Our results showed that PLR-caused change in pulse pressure and systolic BP showed AUC of 0.743 and 0.739 respectively. In accordance with that, Lakhal et al.[6] in a study of 102 critically ill patients found that changes in non-invasive pulse pressure and systolic BP induced by PLR were moderately predictive of volume responsiveness (AUC= 0.70 and 0.72 respectively). On the contrary, Pickett et al.[7] found that pulse pressure measured by non-invasive blood pressure monitoring is not a good predictor of volume responsiveness in healthy volunteers.

The hemodynamic response to the test is different in critically ill mechanically ventilated patients and in healthy volunteers where arterial baroreceptors may be stimulated by painful stimuli, causing vascular compliance to change and thus pulse pressure to inaccurately reflects SV.

Among invasive (i.e measured via arterial catheter) blood pressure parameters; pulse pressure changes induced by PL Rare considered a surrogate for variation in SV. A recent meta-analysis reported an area under the ROC curve of 0.77 ± 0.05[8]. In our study, changes in non-invasive systolic BP showed similar predictive ability as variation in pulse pressure. This can be interpreted by the fact that oscillometric devices measure the mean arterial pressure and then extrapolate both the systolic and the diastolic BP.

Conclusion

Changes in non-invasive oscillometric pulse pressure and systolic BP induced by PLR showed moderate diagnostic ability for prediction of fluid responsiveness in mechanically ventilated patients.

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