



clinical investigations in critical care

Changes in BP Induced by Passive Leg Raising Predict Response to Fluid Loading in Critically Ill Patients*

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Objective: To test the hypothesis that passive leg raising (PLR) induces changes in arterial pulse pressure that can help to predict the response to rapid fluid loading (RFL) in patients with acute circulatory failure who are receiving mechanical ventilation.

Design: Prospective clinical study.

Setting: Two medical ICUs in university hospitals.

Patients: Thirty-nine patients with acute circulatory failure who were receiving mechanical ventilation and had a pulmonary artery catheter in place.

Interventions: PLR for > 4 min and a subsequent 300-mL RFL for > 20 min.

Measurements and main results: Radial artery pulse pressure (PPrad), heart rate, right atrial pressure, pulmonary artery occlusion pressure (PAOP), and cardiac output were measured invasively in a population of 15 patients at each phase of the study procedure (*ie*, before and during PLR, and then before and after RFL). PPrad, PAOP, and stroke volume (SV) significantly increased in patients performing PLR. These changes were rapidly reversible when the patients' legs were lowered. Changes in PPrad during PLR were significantly correlated with changes in SV during PLR ($r = 0.77$; $p < 0.001$). Changes in SV induced by PLR and by RFL were significantly correlated ($r = 0.89$; $p < 0.001$). Finally, PLR-induced changes in PPrad were significantly correlated to RFL-induced changes in SV ($r = 0.84$; $p < 0.001$). In a second population of 24 patients, we found the same relationship between PLR-induced changes in PPrad and RFL-induced changes in SV ($r = 0.73$; $p < 0.001$).

Conclusion: The response to RFL could be predicted noninvasively by a simple observation of changes in pulse pressure during PLR in patients with acute circulatory failure who were receiving mechanical ventilation. (CHEST 2002; 121:1245–1252)

Key words: arterial compliance; arterial pulse pressure; baroreflex; hypovolemia; postural maneuvers; shock; stroke volume; vascular volume expansion

Abbreviations: DAP = diastolic arterial pressure; HR = heart rate; LPBR = low-pressure baroreceptor; MAP = mean arterial pressure; PAOP = pulmonary artery occlusion pressure; PLR = passive leg raising; PPrad = radial artery pulse pressure; RFL = rapid fluid loading; SAP = systolic arterial pressure; SV = stroke volume

In critically ill patients with acute circulatory failure, fluid volume replacement is often necessary to maintain adequate cardiac preload and output.

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The usual clinical and hemodynamic parameters are not reliable indexes of the adequacy of the cardiac preload.¹ Fluid challenge with measurement of cardiac output remains a widely used test to detect cardiac preload dependence but may result in worsening pulmonary edema.

Passive leg raising (PLR) is a reversible maneuver that mimics rapid fluid loading (RFL) by shifting venous blood from the legs² toward the intrathoracic compartment³ and by increasing right⁴ and left^{5,6} ventricular preloads, thereby increasing stroke vol-

ume (SV) and cardiac output.^{4,7-9} Thus, when SV increases with PLR it should increase with RFL as well.

Aortic pulse pressure is proportional to SV and is inversely related to aortic compliance.^{10,11} This led us to propose the following hypotheses: first, because PLR can change SV, it should change aortic pulse pressure proportionally, provided that it hardly changes aortic compliance; and second, during PLR the radial artery pulse pressure (PPrad) might change proportionally with aortic pulse pressure, provided that the PLR does not significantly alter the relationship between the aortic and radial pressures. The aim of our study was to test whether the increase in PPrad during PLR could predict the increase in SV obtained by a subsequent 300-mL RFL in patients with acute circulatory failure.

MATERIALS AND METHODS

We first studied 15 consecutive patients who were admitted to the ICU for acute circulatory failure, which was defined as a systolic arterial pressure (SAP) < 90 mm Hg (or a decrease of > 50 mm Hg in previously hypertensive patients) and urine output of < 0.5 mL/kg/min for at least 2 h. All patients were receiving mechanical ventilation in a volume-controlled mode. Positive end-expiratory pressure was used in two patients and did not exceed 8 cm H₂O. All patients were studied within the first 3 days after admission to the ICU. All had Swan-Ganz catheters (model 93A831H 7.5F; Baxter Healthcare Corp, Edwards Critical Care Division; Irvine, CA) and radial artery catheters in place prior to the study as part of their standard hemodynamic monitoring. Right atrial pressure (RAP) and pulmonary artery occlusion pressure (PAOP) were recorded with patients in the supine position, and cardiac output was measured by four to seven injections at end expiration of 10 mL iced 5% dextrose in water via a closed system (CO-set; Baxter Healthcare Corporation, Edwards Critical Care Division). The result of the first injection was discarded. Cardiac output was determined by averaging the first three values that were within 10% of each other. SAP, diastolic arterial pressure (DAP), and mean arterial pressure (MAP) were measured at end expiration. PPrad was calculated as the SAP minus the DAP. For measurements, we used bedside monitors (Sirecust 126; Siemens Medical Electronics, Inc; Danvers, MA) and pressure transducers (Pressure Monitoring Kit T100209A; Baxter AG; Dietlikon, Switzerland).

The hemodynamic status of all patients initially had been stabilized with infusions of fluid and catecholaminergic agents. Less than 30 min before the beginning of the study, the hemodynamic status of patients had worsened, and a fluid challenge was decided on because of the occurrence of at least one of the following signs: a decrease in SAP of < 90 mm Hg or a decrease in cardiac output by > 10% while PAOP was decreased or unchanged.

Study Protocol

Figure 1 illustrates the design of the study. First, with the patient in the supine position, heart rate (HR), SAP, DAP, and MAP were recorded four times over 4 min at 1-min intervals. The first set of cardiac output and filling pressure values was obtained. The lower limbs of the patients then were lifted in a straight

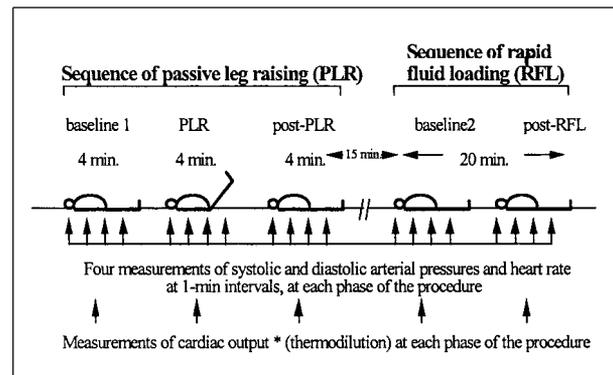


FIGURE 1. Design of the study procedure. * = See text for explanation of measurements of cardiac output.

manner by an assistant to a 45° angle, and HR, SAP, DAP, and MAP were recorded four times at 1-min intervals. A second set of cardiac output and filling pressures values was obtained over this time period. The legs of the patients then were returned to the supine position. Measurements of HR, SAP, DAP, and MAP were recorded once again. A third set of cardiac output and filling pressures was obtained. Fifteen minutes later, a fourth set of hemodynamic measurements was obtained (Fig 1). A fifth set of hemodynamic measurements was obtained after RFL was performed for > 20 min with 300 mL modified fluid gelatin solution (Fig 1).

Because the purpose of the study was to test whether the hemodynamic effect of PLR could predict the hemodynamic response to a subsequent RFL, the specific sequence of PLR followed by RFL, instead of a randomized order, was chosen.

To further validate our hypothesis, we studied a second group of 24 patients with acute circulatory failure, in whom we measured HR and BP via the radial artery catheter through the same sequences of PLR and RFL and measured cardiac output only before and after RFL.

The ventilator settings were maintained at a constant throughout the study period. Patients were sedated by continuous IV midazolam infusion as part of their standard therapy. Midazolam dosages were adjusted to maintain a score of 5 on the Ramsay scale¹² and to avoid voluntary muscular contraction during the procedure. When drugs that might affect cardiac performance or vascular tone (including midazolam) were used, the dosage was not altered throughout the study period.

The study protocol received the approval of the ethics committee of our institution, and informed consent was obtained from the patients' relatives.

Statistical Analysis

Analysis of variance for repeated measurements and the Scheffé F test were used to find significant changes in hemodynamic parameters throughout the study period. Simple linear regressions were used to compare changes in hemodynamic parameters. The results are expressed as the mean ± SD. A p value < 0.05 was chosen as being significant.

RESULTS

The clinical characteristics of the patients studied are listed in Table 1.

Table 1—Descriptive Clinical Data*

Patient No.	Age, yr/Sex	Clinical Diagnosis on Hospital Admission	Adrenergic Agents Used During the Study, $\mu\text{g}/\text{kg}/\text{min}$				Oxygen-Derived Data Before the Beginning of the Study	
			Dopamine	Dobutamine	Norepinephrine	Epinephrine	OER	$\text{PaO}_2/\text{FIO}_2$
1	72/M	Septic shock, ischemic CHF	7	5	—	—	15	165
2	66/F	Septic shock, ischemic CHF	—	—	—	0.3	31	192
3	72/M	Mesenteric infarction, ischemic CHF	3	7	—	—	19	119
4	55/F	Septic shock, ARDS	15	—	—	0.8	40	81
5	66/M	Septic shock, ARDS	10	8	—	—	19	134
6	64/M	APE, hypertensive cardiomyopathy	5	7	—	—	19	205
7	60/M	Septic shock, ischemic CHF	13	10	—	—	27	355
8	78/M	Septic shock, mesenteric infarction	5	—	—	—	31	110
9	58/F	Septic shock, ARDS, aspiration pneumonia	5	10	—	—	36	273
10	70/M	Septic shock, peritonitis, ischemic CHF	13	—	—	—	18	204
11	82/M	Septic shock, pneumonia, ischemic CHF	9	7	—	—	35	131
12	58/M	Septic shock, ARDS	7	—	—	—	27	184
13	57/F	Pneumonia, cor pulmonale	5	—	0.2	—	26	160
14	38/F	Peripartum cardiomyopathy	3	17	—	0.2	33	268
15	73/M	Myocardial infarction, cardiogenic shock	10	—	—	—	46	273
16	57/M	Cardiac surgery, shock	8	—	0.3	—	23	162
17	60/M	Cardiac surgery, shock	17	—	1.9	—	32	173
18	83/M	Aortic surgery, shock	5	—	—	—	36	151
19	40/F	Meprobamate intoxication, cardiogenic shock	—	—	—	1	22	150
20	83/M	Septic shock, pneumonia, ischemic CHF	3	16	—	—	18	237
21	78/M	Septic shock, pneumonia	3	12	0.6	—	40	86
22	70/M	Cardiac surgery, shock	4	—	—	—	21	160
23	74/M	Septic shock, pneumonia, ischemic CHF	4	—	—	—	34	51
24	72/M	Thoracic surgery, hypovolemic shock	8	—	—	—	24	160
25	65/M	Septic shock, pneumonia, ARDS	6	—	—	—	16	126
26	69/M	Thoracic surgery, shock	7	—	—	—	18	70
27	21/M	Near-drowning, hypovolemic shock	9	—	—	—	39	115
28	30/F	Pyelonephritis, septic shock, ARDS	13	—	—	—	35	67
29	75/M	Acute pancreatitis, hypovolemic shock, CHF	—	9	—	—	26	168
30	78/M	Septic shock, pneumonia	4	—	—	—	18	91
31	70/M	Cardiac surgery, shock	4	—	—	—	29	89
32	58/M	Cardiac surgery, shock	3	—	—	—	23	131
33	73/F	Cardiac surgery, shock	4	9	—	—	23	147
34	66/M	Cardiac surgery, shock	6	—	—	—	21	116
35	37/F	Pyelonephritis, septic shock	7	5	—	—	23	85
36	76/M	Pneumonia, cor pulmonale, cardiogenic shock	—	5	—	—	20	100
37	83/M	Septic shock, pneumonia, ARDS	15	—	0.6	1	21	136
38	60/M	Cardiac surgery, shock	10	—	—	—	36	294
39	65/F	Septic shock, pneumonia, CHF	12	7	—	—	46	170

*M = male; F = female; FIO_2 = fraction of inspired oxygen; CHF = chronic heart failure; OER = oxygen extraction ratio; APE = acute pulmonary edema.

Analysis of variance detected significant changes in BP that were induced by PLR positioning, by the return of the legs to the supine position, and by RFL, while it detected no change in BP values recorded within each phase of the procedure. Because of the stability of BP (*ie*, SAP, DAP, and MAP) within each phase of the PLR and RFL procedures, we expressed individual BP values as the average of the four measurements performed at each phase of the study protocol.

In our first 15 patients, PLR significantly increased PAOP and SV, while MAP and HR were not

altered (Table 2). When PLR changed PAOP, RAP, and SV, these parameters returned to baseline values immediately after the PLR (Fig 2). In most of the patients, PPrad slightly increased during PLR (Fig 2). This increase in PPrad was positively correlated to the increase in SV during PLR ($r = 0.77$; $p < 0.001$) [Fig 3]. There was no significant correlation between PLR-induced changes in SV and values for RAP before PLR ($r = 0.39$). When considering the median value of RAP before PLR (8 mm Hg), we did not find any significant difference in the percentage increase in SV when RAP was either ≤ 8 mm Hg

Table 2—Evolution of Hemodynamic Parameters in 15 Patients Subjected to PLR and Then to RFL*

Parameters	Baseline 1	PLR	Post-PLR	Baseline 2	Post-RFL
HR, beats/min	98 ± 21	97 ± 20	97 ± 20	97 ± 20	94 ± 26
SAP, mm Hg	106 ± 25	113 ± 25†	106 ± 26‡	107 ± 25	109 ± 26
DAP, mm Hg	57 ± 15	60 ± 17†	57 ± 16‡	57 ± 15	58 ± 15
MAP, mm Hg	73 ± 17	77 ± 18	73 ± 18	71 ± 19	73 ± 18
PPrad, mm Hg	49 ± 16	53 ± 17†	49 ± 17‡	50 ± 17	51 ± 17
RAP, mm Hg	9 ± 5	10 ± 6	9 ± 6‡	9 ± 6	12 ± 6§
PAOP, mm Hg	11 ± 6	14 ± 6†	11 ± 6‡	12 ± 6	16 ± 7§
CO, L/min	5.2 ± 1.3	5.6 ± 1.6	5.4 ± 1.4	5.4 ± 1.3	5.6 ± 1.5
SV, mL	56 ± 20	60 ± 23†	57 ± 21	58 ± 18	62 ± 23§

*Values given as mean ± SD. RAP = right atrial pressure; CO = cardiac output.

†PLR vs baseline 1, $p < 0.05$.

‡Post-PLR vs PLR, $p < 0.05$.

§Post-RFL vs baseline 2, $p < 0.05$.

or > 8 mm Hg ($9 \pm 10\%$ vs $4 \pm 8\%$, respectively). There was no significant correlation between PLR-induced changes in SV and the values of PAOP before PLR.

Changes in SV induced by PLR and RFL were significantly correlated ($r = 0.89$; $p < 0.001$) [Fig 4]. Changes in PPrad with PLR were correlated to RFL-induced changes in SV ($r = 0.84$; $p < 0.001$) [Fig 4]. There was no significant correlation between RFL-induced changes in SV and the values of RAP before RFL ($r = 0.40$). When considering the median value of RAP before RFL (8 mm Hg), we did not find any significant difference in the percentage increase in SV when RAP was either ≤ 8 mm Hg or > 8 mm Hg ($10 \pm 8\%$ vs $4 \pm 10\%$, respectively). There was no significant correlation between RFL-induced changes in SV and the values of PAOP before RFL.

In the 24 additional patients, changes in PPrad with PLR were correlated to changes in SV induced by RFL ($r = 0.84$; $p < 0.001$).

When data from all 39 patients were pooled

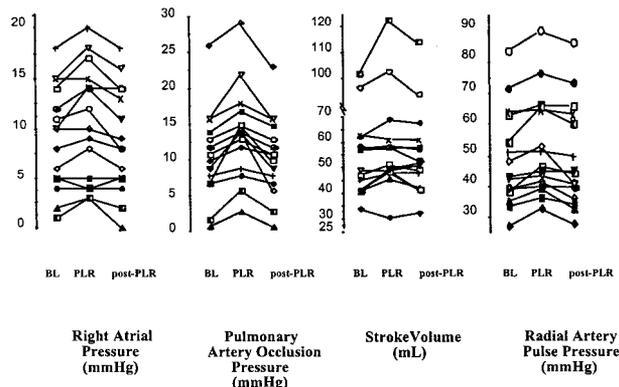


FIGURE 2. Evolution of RAP, PAOP, SV, and PPrad during PLR. BL = baseline.

together, the changes in arterial BP showed the same profiles as those in our 15 first patients (Table 3), and we also found a good correlation between PLR-induced changes in PPrad and RFL-induced changes in SV ($r = 0.74$; $p < 0.001$) [Fig 5].

DISCUSSION

The main finding of our study was that changes in PPrad induced by PLR are proportional to the changes in SV that are obtained with a subsequent RFL. Therefore, simple measurements of BP during PLR could help to detect patients who will positively respond to volume expansion.

The PLR Preload Effect

It has been known for a long time that PLR can mimic the hemodynamic effects of RFL. However, the effects of PLR on cardiac output are variable,^{4,7,8,13} probably depending on the degree of

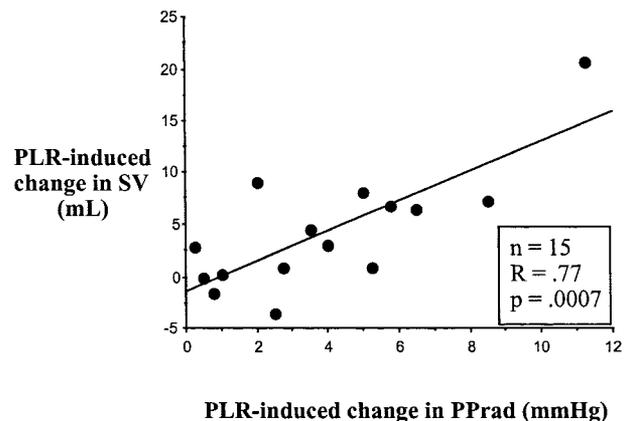


FIGURE 3. Relationship between changes in PPrad and changes in SV both obtained during PLR.

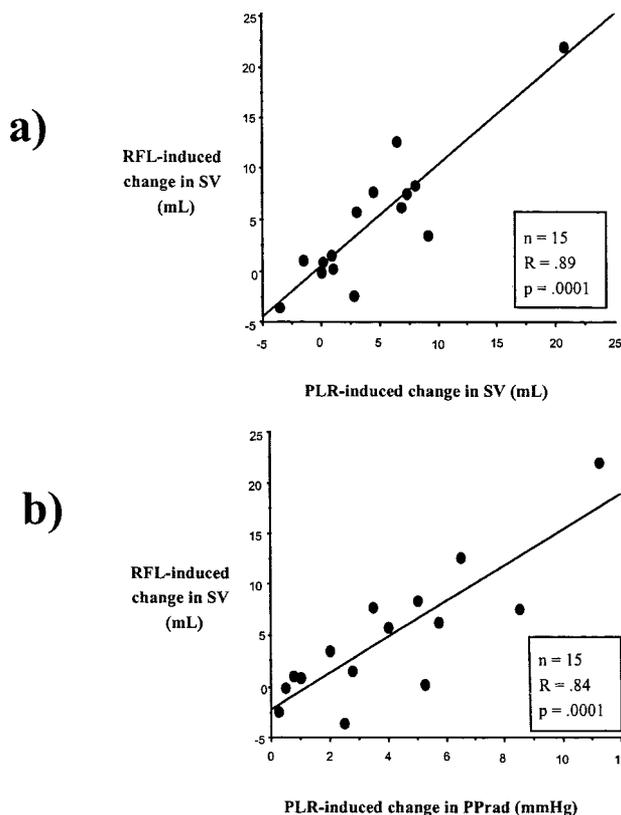


FIGURE 4. *Top, a:* relationship between changes in SV obtained during PLR and with a subsequent RFL. *Bottom, b:* relationship between changes in PPrad during PLR and changes in SV obtained with a subsequent RFL.

leg elevation and on the existence of a cardiac preload dependence. The effects of PLR have been reported to vanish with time, rarely exceeding a 10-min duration.^{4,7,13} We chose the amount of 300 mL for RFL because it is roughly comparable to the volume of blood shifted from the legs toward the central compartment during PLR.^{2,14} In our patients, PLR and RFL, both of which increased PAOP, induced changes in SV that were significantly and positively correlated. We think that these results validate our 4-min, 45° angle PLR procedure as a maneuver that is sufficient to

Table 3—Evolution of Arterial Pressure in Our 39 Patients During the PLR Procedure*

Pressures	Baseline 1	PLR	Post-PLR
MAP, mm Hg	74 ± 17	79 ± 17†	73 ± 16‡
DAP, mm Hg	57 ± 14	60 ± 15†	56 ± 14‡
PPrad, mm Hg	51 ± 18	56 ± 20†	52 ± 18‡
SAP, mm Hg	109 ± 26	116 ± 28†	108 ± 26‡

*Values given as mean ± SD.

†Significant change compared to baseline value ($p < 0.05$).

‡Significant change compared to PLR sequence value ($p < 0.05$).

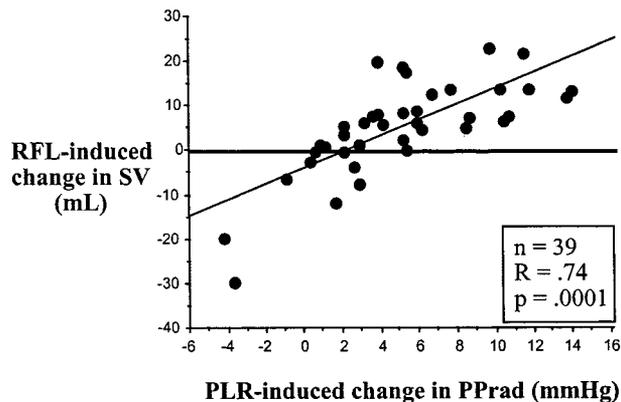


FIGURE 5. Relationship between changes in PPrad observed during PLR and changes in SV obtained by a subsequent RFL when all 39 patients were pooled.

mimic a 300-mL RFL-induced cardiac preload augmentation that would result in SV augmentation in patients with preload dependence. The way by which PLR can alter preload is probably an increase in the mean systemic pressure, the driving force for venous return,^{15,16} due to the gravitational shift of venous blood from unstressed to stressed volume. This mechanism was probably of major importance during PLR in our patients. When mechanical ventilation is required, the volume of blood enclosed by the thoracic and splanchnic beds is already stressed by positive airway pressure, and these vascular compartments are less compliant than when mechanical ventilation is not required.¹⁷ In these conditions, the increase in mean systemic pressure with PLR was expected to be higher in our patients than in patients not receiving mechanical ventilation.

Some treatments used in our patients probably had significant and various effects on the preload effect of PLR. Catecholamines with α -adrenergic properties, by their venous vasoconstrictor effects, could have altered our results. They might have shifted venous blood from an unstressed to a stressed volume¹⁸ and might have amplified the preload augmentation effect of PLR in our patients. However, we think that this phenomenon did not affect the interpretation of our results since the dosages of the catecholamines were not modified during our study. Nevertheless, as in many patients, therapy with catecholamines could have amplified the effects of PLR, so our results must be used with caution in patients who have not been treated with vasoconstrictors.

For reasons mentioned earlier, mechanical ventilation also might have amplified the cardiac preload increase during PLR, so that we cannot extrapolate our results to spontaneously breathing patients.

Throughout our study procedure, we performed numerous bolus injections to measure cardiac output by thermodilution, which could have had a vascular expansion effect and, thereby, could have interfered with the assessment of the true effects of PLR. However, because we used analysis of variance to discover significant changes occurring during our study, which disclosed no change in cardiac output between baseline 1 (*ie*, before PLR) and baseline 2 (*ie*, before RFL), we think that the preload augmentation effect of the bolus injections performed was probably negligible.

SV to PPrad Relationships

In our study, PLR produced a rapid and sustained rise (over a period of 4 min) in PPrad in almost all the patients. It is interesting to note that the increase in PPrad was closely correlated to the increase in SV during PLR.

Our work was based on the hypothesis that PLR *per se* does not alter the viscoelastic properties of the arteries and has no effect on the transmission of the arterial pressure wave from the aorta to the radial artery, so that changes in PPrad could reflect changes in SV. However, PLR, which increases thoracic blood volume, has been shown to dilate the arteries of the upper limbs via the stimulation of the low-pressure baroreceptors (LPBRs).^{19–22} This probably explains why PLR has been shown⁸ to have no effect on MAP, although it increased SV in slightly hypovolemic and spontaneously breathing patients. By contrast, to our knowledge, the LPBR-mediated vasodilation has never been demonstrated in mechanically ventilated patients. Consequently, we put forward that mechanical ventilation with positive intrathoracic pressure, by reducing the stretch of the LPBRs that are located in the pulmonary vessels,^{23–25} could attenuate LPBR stimulation and the subsequent arterial dilation.

However, whatever the degree of LPBR-mediated vasodilation that might have occurred in our patients, we think that it did not affect the significance of our results because the ratio of SV to PPrad, when used as a gross estimate of total arterial compliance in our first 15 patients, gave results before and during PLR that were strongly correlated and in good agreement ($r = 0.98$) [Fig 6].

Thus, we think our results support our hypothesis that the compliance of the arterial system and the relationship between aortic and radial pressures are not significantly altered by PLR.

Some of the treatments used in our patients, such as the administration of catecholamines with their arterial constrictor effects, could have modified the

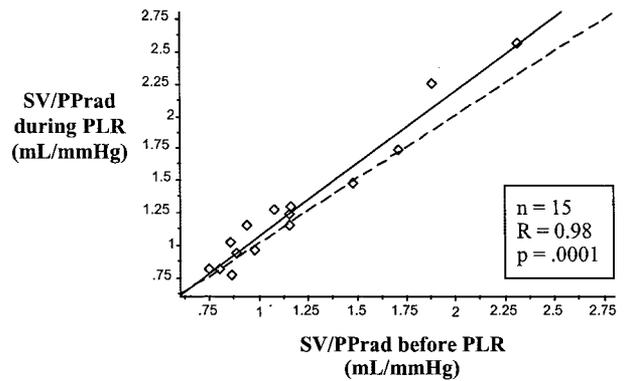


FIGURE 6. Total arterial compliance, assessed by the ratio of SV to PPrad, measured before and during PLR. Dashed line indicates line of identity (*ie*, when there is no change in SV/PPrad with PLR).

transmission of the arterial pressure wave from the aorta to the peripheral arteries, thereby inducing changes in PPrad without any changes in SV (or no changes in PPrad and significant changes in SV). This did not seem to occur in our patients since, as mentioned above, the ratio of SV to PPrad gave results before and during PLR that were strongly correlated (Fig 6).

We also demonstrated that the PLR-induced changes in PPrad were significantly correlated to the changes in SV that had been induced by fluid loading. This finding strongly suggests that PPrad changes induced by PLR could help to differentiate patients who will benefit from volume infusion from patients who will not.

We found that the changes in PAOP induced by PLR were immediately and completely reversible when patients' legs were lowered. This finding suggests that PLR may aid in predicting individual fluid responsiveness while avoiding the hazards of unnecessary fluid loading.

The correlations that we found between changes in PPrad induced by PLR and changes in SV with RFL were good but not very strong ($r = 0.74$ in 39 patients). Indeed, PLR may have different preload augmentation effects among patients, and the shift of blood from the legs to the central compartment may range from 150 to 300 mL.² Some patients in our study probably underwent a shift of blood of much < 300 mL with PLR that led to small increases in SV and PPrad, while exhibiting a clear SV augmentation with a 300-mL volume of fluid loading.

Volume status and peripheral arterial tone may influence peripheral pressure waveforms via complex changes in pressure-wave propagation and reflection.²⁶ We cannot exclude the possibility that patients with marked volume depletion and/or vaso-

constriction could exhibit a certain degree of pressure artifacts. However, we tried to minimize this problem as far as possible by carefully checking the quality of the pressure-wave recordings. Despite unavoidable limitations due to the use of fluid-filled catheters in the peripheral arteries, our results were quite consistent with our hypothesis, confirming the fact that fluid-filled catheter systems may be sensitive enough to give helpful information for the management of the fluid resuscitation of patients at the bedside.

Finally, it has to be underlined that neither baseline RAP nor baseline PAOP were good predictors of volume responsiveness in our patients. These findings are in accordance with results of previous clinical studies²⁷⁻³⁰ showing that values of RAP and/or PAOP before volume expansion were not significantly different in patients who will respond to fluid infusion compared with those who will not respond. This emphasizes the need for an alternative method to the use of a pulmonary artery catheter to manage fluid resuscitation in critically ill patients.

CONCLUSION

Our data suggest that in patients with acute circulatory failure who are receiving mechanical ventilation, the hemodynamic response to fluid loading could be predicted by simply measuring arterial pulse pressure changes during PLR. This may have two practical implications: (1) the existence of a cardiac preload dependence could be detected without the use of a Swan-Ganz catheter; and (2) a potentially harmful fluid-loading procedure could be avoided when unnecessary.

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REFERENCES

- 1 Shippy CR, Appel PL, Shoemaker WC. Reliability of clinical monitoring to assess blood volume in critically ill patients. *Crit Care Med* 1984; 12:107-112
- 2 Rutlen DL, Wackers FJT, Zaret BL. Radionuclide assessment of peripheral intravascular capacity: a technique to measure intravascular volumes changes in the capacitance circulation in man. *Circulation* 1981; 64:146-152
- 3 Reich DL, Konstadt SN, Raissi S, et al. Trendelenburg position and passive leg raising do not significantly improve cardiopulmonary performance in the anesthetized patient with coronary artery disease. *Crit Care Med* 1989; 17: 313-317
- 4 Thomas M, Shillingford J. The circulatory response to a standard postural change in ischaemic heart disease. *Br Heart J* 1965; 27:17-27
- 5 Rocha P, Lemaigre D, Leroy M, et al. Nitroglycerin-induced

- decrease of carbon monoxide diffusion capacity in acute myocardial infarction reversed by elevating legs. *Crit Care Med* 1987; 15:131-133
- 6 Takagi S, Yokota M, Iwase M, et al. The important role of left ventricular relaxation and left atrial pressure in the left ventricular filling velocity profile. *Am Heart J* 1989; 118:954-962
- 7 Wong DH, Tremper KK, Zaccari J, et al. Acute cardiovascular response to passive leg raising. *Crit Care Med* 1988; 16:123-125
- 8 Wong DH, O'Connor D, Tremper KK, et al. Changes in cardiac output after acute blood loss and position change in man. *Crit Care Med* 1989; 17:979-983
- 9 Albert RK, Schrijen F, Poincelot F. Oxygen consumption and transport in stable patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1986; 134:678-682
- 10 Guyton AC. *Textbook of medical physiology*. 8th ed. Philadelphia, PA: WB Saunders, 1991; 221-233
- 11 Chemla D, Hebert JL, Coirault C, et al. Total arterial compliance estimated by stroke volume-to-aortic pulse pressure ratio in humans. *Am J Physiol* 1998; 274:H500-H505
- 12 Ramsay MAE, Savege TM, Simpson BRJ, et al. Controlled sedation with alpharealone-alphadalone. *BMJ* 1974; 2:656-659
- 13 Gaffney FA, Bastian BC, Thal ER, et al. Passive leg raising does not produce a significant or sustained autotransfusion effect. *J Trauma* 1982; 22:190-193
- 14 Morgan BC, Guntheroth WG, McGough GA. Effect of position on leg volume: case against the Trendelenburg position. *JAMA* 1964; 187:1024-1026
- 15 Guyton AC, Lindsey AW, Abernathy B, et al. Venous return at various right atrial pressures and the normal venous return curve. *Am J Physiol* 1957; 189:609-615
- 16 Guyton AC, Granger HJ, Coleman TG. Autoregulation of the total systemic circulation and its relation to control of cardiac output and arterial pressure. *Circ Res* 1971; 28(suppl):93-97
- 17 Chihara E, Hashimoto S, Kinoshita T, et al. Elevated mean systemic filling pressure due to intermittent positive-pressure ventilation. *Am J Physiol* 1992; 262:H1116-H1121
- 18 Appleton C, Olajos M, Morkin E, et al. Alpha-1 adrenergic control of the venous circulation in intact dogs. *J Pharmacol Exp Ther* 1985; 233:729-734
- 19 Parati G, Grassi G, Coruzzi P, et al. Influence of cardiopulmonary receptors on the bradycardic responses to carotid baroreceptor stimulation in man. *Clin Sci* 1987; 72:639-645
- 20 London GM, Pannier BM, Laurent S, et al. Cardiopulmonary baroreflex control of brachial artery diameter in sustained essential hypertension. *J Hypertension* 1989; 7:879-883
- 21 Grassi G, Giannattasio C, Cuspidi C, et al. Cardiopulmonary receptor regulation of renin release. *Am J Med* 1988; 84:97-104
- 22 London GM, Levenson JA, Safar ME, et al. Hemodynamic effects of head-down tilt in normal subjects and sustained hypertensive patients. *Am J Physiol* 1983; 245:H164-H202
- 23 Cassidy SS, Robertson CH Jr, Pierce AK, et al. Cardiovascular effects of positive end-expiratory pressure in dogs. *J Appl Physiol* 1978; 44:743-750
- 24 Morgan BC, Martin WE, Hornbein TF, et al. Hemodynamic effects of intermittent positive pressure respiration. *Anesthesiology* 1966; 27:584-590
- 25 Rankin JS, Olsen GO, Arentzen CE, et al. The effects of airway pressure on cardiac function in intact dogs and man. *Circulation* 1982; 66:108-120
- 26 O'Rourke MF, Yaginuma T. Wave reflections and the arterial pulse. *Arch Intern Med* 1984; 144:366-371

- 27 Calvin JE, Driedger AA, Sibbald WJ. The hemodynamic effect of rapid fluid infusion in critically ill patients. *Surgery* 1981; 90:61-76
- 28 Reuse C, Vincent JL, Pinsky MR. Measurements of right ventricular volumes during fluid challenge. *Chest* 1990; 98: 1450-1454
- 29 Tavernier B, Makhotine O, Lebuffe G, et al. Systolic

- pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology* 1998; 89: 1313-1321
- 30 Michard F, Boussat S, Chemla D, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000; 162:134-138

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