Intensive Care Med (2007) 33:1111–1113
DOI 10.1007/s00134-007-0645-8

EDITORIAL

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Can one predict fluid responsiveness in spontaneously breathing patients?

Received: 13 March 2007
Accepted: 27 April 2007
Published online: 17 May 2007

This editorial refers to the articles available at:
http://dx.doi.org/10.1007/s00134-007-0642-y,
http://dx.doi.org/10.1007/s00134-007-0644-9, and
http://dx.doi.org/10.1007/s00134-007-0646-7

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Prediction of fluid responsiveness has become a topic of intense interest. Although measurements of preload, by whatever technique, are still commonly used to guide fluid therapy [1, 2], these fail to estimate the response to fluids in one-half of the patients [3]. Accordingly, many patients may be subjected to the hazards of fluids [4], without benefiting from hemodynamic improvement.

In patients receiving mechanical ventilatory support predictable heart lung interactions can be used to accurately identify fluid responsiveness by noting the arterial pulse pressure or aortic stroke volume variation over three or more breaths [5]. This is because positive-pressure inspiration induces cyclic increases in right atrial pressure causing in turn inverse changes in venous return, right ventricular filling and ejection and ultimately left ventricular preload. In preload-dependent patients these cyclical changes in ventricular filling induce cyclic changes in stroke volume and arterial pulse pressure, provided that both the right and left ventricles are preload responsive. These cyclic changes in pressure and flow are referred to as pulse pressure variation (Δ PP) and stroke volume variation (Δ SV), respectively. In spontaneously breathing patients the situation is more complex. Spontaneous inspiration induces negative cyclic changes in intrathoracic pressure causing right atrial pressure to also decrease increasing the pressure gradient for venous blood flow. In calves with total artificial hearts spontaneous ventilation induced opposite changes in left ventricular stroke volume than mechanical ventilation [6]. However, unlike artificial hearts whose right and left side filling is independent of each other, in patients with normal hearts Δ PP failed to predict preload response during under either pressure support [7, 8] or spontaneously breathing [8]. Various factors may explain this lack of reliability. Both pressure support ventilation and spontaneous breathing are associated with variability in tidal volume, and both Δ PP and Δ SV are dependent on tidal volume [9, 10]. Similarly, spontaneous inspiratory efforts may increase intra-abdominal pressure because of active compression of abdominal muscles, exaggerating the preload response. Finally, sudden increases in right ventricular end-diastolic volume decrease left ventricular diastolic compliance by the process of ventricular interdependence, which may decrease left ventricular filling and stroke volume independent of preload-responsiveness, because even failing hearts need some end-diastolic volume to generate stroke volume. Soubrier et al. [11] in their contribution to *Intensive Care Medicine* now reason that forced expiratory maneuvers (Valsalva maneuver) may identify preload responsiveness in spontaneously breathing patients when normal spontaneous ventilation do not. Using standardized amplitude of respiratory movements they compared the effects of both a Valsalva maneuver and spontaneous

breathing on Δ PP to predict preload responsiveness in 32 critically ill patients. During regular spontaneous breathing activity Δ PP was higher in responders to fluid challenge than in nonresponders, but the test performed poorly with a sensitivity of Δ PP greater than 12% of only 63% although specificity was good (92%). The forced maneuver failed to improve the performance of the test, with a decrease in sensitivity to 21% but a maintained specificity (92%). Importantly, the cutoff value of Δ PP increased to 33% during the forced expiratory maneuver, indicating that during this large swing in pleural pressure moderate fluctuations in arterial pressure may not indicate fluid responsiveness. This may be due to several factors. First, applying larger tidal volumes and more negative pleural pressure may transiently shift the patient to preload dependency, which disappears when the patient breaths normally. Second, the Valsalva maneuver may also affect right and left ventricular afterload, which may contribute to respiratory variations in stroke volume [12]. Thus, regrettably, Δ PP and other derived indices cannot be used in spontaneously breathing patients, as slight and sometimes undetected changes in breathing pattern may affect these variables.

Another way to predict fluid responsiveness is to perform an endogenous fluid challenge using passive leg raising. This approach has been used by cardiologists in the cardiac catheterization laboratory for over 50 years. In critically ill patients under mechanical ventilation, with or without spontaneous respiratory movements, an increase in mean aortic flow of more than 10% during passive leg raising reliably predicts the response to fluid challenge [7, 13, 14]. Until now this test has been difficult to apply in conscious and spontaneously breathing patients. Passive leg raising requires the use of a fast response measurement of cardiac output as the increase in cardiac output is transient and may not be maintained when legs are raised for more than a few minutes. Hence esophageal Doppler measures of descending aortic blood flow was used in the three reported studies [7, 13, 14]. Unfortunately, using an esophageal flow probe may be quite uncomfortable in conscious patients. To address this issue both Lamia et al. [15] and Maizel et al. [16] in their contributions to *Intensive Care Medicine* demonstrate that transthoracic echocardiography can be used to measure mean cardiac output during leg raising. They report that an increased change in aortic flow, measured as velocity time interval at the aortic valve (VTIao) by 10–13%, was associated with a positive response to fluid challenge. In both cases the performance of the tests was very good (receiver operating characteristic curve area of 0.96 [15] and 0.90 [16]) with sensitivity and specificity above 80%. Importantly, one-half of the patients in the study by Lamia et al. [15] and all patients in the study by Maizel et al. [16] were spontaneously breathing without any mechanical support.

These studies provide several lessons. First, changes in mean arterial pressure during passive leg raising failed to predict the response to fluid. Similarly, changes in arterial pressure during fluid challenge were only loosely related to changes in cardiac output, again demonstrating that the only way to assess a positive response to fluid in a spontaneously breathing subject is to measure the changes in cardiac output by whatever the technique. Second, changes in left ventricular area or mitral Doppler, reflecting left ventricular preload, were not useful for predicting the response to fluids, indicating again that preload-responsiveness is not the same as preload.

Several limitations of these last two studies should be acknowledged. First, VTIao rather than stroke volume or cardiac output was measured in order to limit errors in calculation. Both studies considered that aortic diameter did not change during passive leg raising and fluid challenge, and thus VTIao reflected stroke volume. However, if passive leg raising induced increased flow and also increased arterial pressure, aortic diameter may also increase, reducing the accuracy of the VTIao to track flow changes [17]. This limitation may not apply to the aortic outflow tract where VTIao is obtained with echocardiography, as this area is somewhat protected by the aortic annulus. Also, although tachycardia is common in hypovolemia, many studies have reported that there is no major change in heart rate during fluid challenge, even in responders, and therefore stroke volume can be used to assess cardiac output changes, which define fluid responsiveness. Second, some patients experienced a significant decrease in cardiac output both during passive leg raising and fluid challenge. This may reflect a stress-induced change in metabolic requirements or reflect vasoconstriction occurring between baseline and subsequent passive leg raising measurements. This underscores the axiom that hemodynamic evaluation should always be performed carefully in conscious patients and that external factors may interfere with the hemodynamic response to an intervention. Finally, although reliable in experts hands (the inter- and intraobserver variability of VTIao were lower than 5% in both studies), investigators less experienced with echocardiographic techniques may not reach this level of accuracy. In particular, small changes in the angle of the echo beam may induce errors in measurements that may be misinterpreted (a 15° angle inducing a 5% error in measurement). More importantly, echocardiography is not always available at any time of the day and cannot be used for continuous monitoring needed for trend analysis. It is likely that other cardiac output measurement techniques, such as pulse contour determinations, would provide similar results and may supplant echocardiography in bedside monitoring of dynamic changes in cardiac output.

Hence the prediction of fluid responsiveness is now feasible in spontaneously breathing patients, but this requires the performance of a passive leg raising test and the dynamic measure of changing cardiac output using fast response measurements techniques. In the hands of

the unskilled, much caution needs to be placed on the interpretation of these parameters, but when the data are accurately collected, it represents a new and powerful tool in the critical care diagnostic armamentarium.

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Intensive Care Med (2007) 33:1133–1138
DOI 10.1007/s00134-007-0642-y

ORIGINAL

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Diagnosis of central hypovolemia by using passive leg raising

Received: 11 October 2006
Accepted: 27 March 2007
Published online: 17 May 2007

This article is discussed in the editorial available at: http://dx.doi.org/10.1007/s00134-007-0645-8.

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Abstract *Objective:* Suspected central hypovolemia is a frequent clinical situation in hospitalized patients, and no simple bedside diagnostic test in spontaneously breathing patients is available. We tested the value of passive leg raising to predict hemodynamic improvement after fluid expansion in patients with suspected central hypovolemia. *Design and setting:* Prospective study in four intensive care units at the Amiens university hospital. Thirty-four spontaneously breathing patients with suspected hypovolemia were included and were classified as responders (cardiac output increased by 12% or more after fluid expansion) or nonresponders. Patients were analyzed in the supine position during 30° leg raising and after fluid expansion. *Measurements and results:* Stroke volume and cardiac output determined by echocardiographic and Doppler techniques and heart rate and blood pressure were measured at baseline, during passive leg raising and after fluid expansion. An increase of cardiac output or stroke volume by 12% or more during passive leg raising was highly predictive of central hypovolemia (AUC 0.89 ± 0.06, 95% CI 0.73–0.97 for cardiac output and AUC 0.9 ± 0.06, 95% CI 0.74–0.97 for stroke volume). Sensitivity and specificity values were 63% and 89% for cardiac output and 69%, 89% for stroke volume respectively. A close correlation ($r = 0.75$; $p < 0.0001$) was observed between cardiac output changes during leg raising and changes in cardiac output after fluid expansion. *Conclusions:* Bedside measurement of cardiac output or stroke volume by Doppler techniques during passive leg raising was predictive of a positive hemodynamic effect of fluid expansion in spontaneously breathing patients with suspected central hypovolemia.

Introduction

In many clinical situations, such as hypotension, shock, functional renal failure, oligoanuria, or clinical and/or laboratory signs of dehydration, central hypovolemia may be suspected, raising the question of whether fluid expansion is required to increase central blood volume [1, 2]. In these clinical situations, volume infusion induces significant increase in cardiac output because the heart (left and right ventricles) works on the stiff portion of the Frank–Starling relationship, and therefore increases organ perfusion and blood flow. The development of a predictive index of preload dependency, and thus of fluid responsiveness, in patients with suspected central hypovolemia therefore constitutes a major clinical challenge. Such indices were recently introduced and validated in intubated and mechanically ventilated patients [3–6]. In spontaneously breathing patients, these indices appear inaccurate because they strongly depend on respiratory status, which is not controlled in this case. Although the

clinical responses to postural maneuvers, such as a large change in pulse, severe dizziness or hypotension, may help clinicians to identify patient with hypovolemia due to blood loss, these criteria demonstrated low sensitivity and specificity for hypovolemia not due to blood loss [1]. On the other hand, blind fluid infusion can be harmful by increasing left-ventricular diastolic pressure, leading to pulmonary edema. We hypothesized that passive leg raising, by mobilizing blood from the lower part of the body to the chest, may increase preload, stroke volume and cardiac output, but only if the patient has true central hypovolemia with a low preload. In the absence of central hypovolemia, stroke volume and cardiac output are not increased by this postural maneuver [7–9]. This maneuver combined with measurement of cardiac output can therefore be used before any volume expansion. The primary aim of this study was to assess the predictive value of stroke volume and noninvasive measurement of cardiac output changes during passive leg raising in spontaneously breathing patients with suspected hypovolemia.

Method

Patients

This prospective study was performed in four intensive care units in Amiens university hospital (two medical, one surgical and one cardiac unit). Spontaneously breathing patients in whom the attending physician decided to perform fluid expansion were included consecutively. Inclusion criteria were: hypotension (systolic blood pressure less than 90 mmHg and/or mean blood pressure less than 70 mmHg or blood pressure decreased by more than 40 mmHg compared to usual blood pressure); oligoanuria or acute renal failure; or clinical and biological signs of extracellular fluid volume depletion (associated with an obvious etiology: ketoacidosis, vomiting, diarrhea). Patients with clinical signs of hemorrhage, patients in whom fluid challenge could not be deferred for several minutes, patients with a contraindication to passive leg raising, and patients with arrhythmia were not included. This protocol was approved by the local ethics committee and all patients — when conscious — or their relatives gave their informed consent.

Protocol

All patients were in the supine position for baseline measurements. Arterial blood pressure [systolic (SAP), diastolic (DAP), and mean arterial pressure (MAP)] (Agilent Component Monitoring System, model M1205A, Boeblingen, Germany) was measured with a brachial cuff and heart rate was recorded. Echocardiographic cardiac output was measured in this position. The bed was angled to produce passive leg raising (PLR) of 30 ° and blood pressure, heart rate and cardiac output were measured again after 2 min. The patient was then returned to the supine position and 500 ml of saline solution was administered intravenously over 15 min. Echocardiography was repeated and blood pressure and heart rate were recorded.

Measurements

The following clinical characteristics were recorded: age, gender, SAPS II score, weight, MacCabe score, surgical/medical clinical problems, principal diagnosis, medical history (presence of hypertension, diabetes mellitus, cardiomyopathy, presence of COPD or pulmonary embolism). Echocardiography was performed using HP Sonos 2000 and Philips Envisor (Philips Medical System, Suresnes, France). On a parasternal 2D view, aortic diameter (AoD) was measured at the level of the aortic valve insertion (aortic annulus). Aortic area (AA) was calculated as follows: $AA = (\pi \times AoD^2)/4$. On an apical five-chamber view, aortic blood flow was recorded using pulsed Doppler, with the sample volume placed at the aortic annulus. The velocity–time integral of aortic blood flow (VTI) was calculated. Stroke volume (SV) and cardiac output (CO) were calculated as follows: $CO = SV \times HR$, where HR is heart rate and $SV = VTI \times AA$. Aortic area was considered to be stable throughout the experiment and was measured only at baseline; it was used to calculate CO during leg raising and after fluid infusion. Each reported measurement of VTI was an average of three to five consecutive measurements over one respiratory cycle. All measurements were performed by echocardiography-trained intensivists (J. M. and N. A.)

Statistical analysis

All variables are presented as mean ± standard deviation. A normal distribution was tested by means of a Kolmogorov–Smirnov test. The relationships between various variables were analyzed using a linear regression method. Variables were compared using a Student *t*-test for continuous variables and a chi-square test for percentages.

The reproducibility of CO and SV measurements was tested before the study. These variables were measured twice in 10 patients by the same observer (J. M.; intraobserver reproducibility) and by a second observer (N. A.; interobserver reproducibility) after a 2-min interval in stable, spontaneously breathing patients. The mean difference was calculated and divided by the mean of the two values. Intraobserver reproducibility was $4.2 \pm 3.9\%$ and $4.2 \pm 3.9\%$ for CO and SV, respectively, and interobserver reproducibility was $6.5 \pm 5.5\%$ and $6.2 \pm 4.2\%$, respectively. As all measurements during

this study were performed by the same observer, and in the light of these findings concerning intraobserver reproducibility, patients with an increase of CO $\geq$ 12% or $<$ 12% after fluid expansion compared to baseline were classified as responders and nonresponders, respectively. Absolute values at baseline as well as changes of heart rate, pressures, VTI (ΔVTI), SV (ΔSV), and CO (ΔCO) during leg raising (PLR) were analyzed. The correlation between these variables and changes in CO after fluid expansion as well as their predictive value to diagnose an increase in CO after fluid expansion was calculated by using a receiver operating characteristic (ROC) curve. The area under the curve (AUC) was calculated for all parameters and compared by means of a Hanley–McNeil test. Sensitivity, specificity, negative and positive predictive values, negative and positive likelihood ratio and rate of correct classification were calculated after defining a cut-off value. A p value < 0.05 was considered significant. Statistical analysis was performed using StatView version 5.0 (SAS Institute Inc, Cary, NC) and MedCalc version 8.2.1.0 (MedCalc Software, Mariakerke, Belgium) software.

Results

Patient characteristics are summarized in Table 1; 3 patients received only vasoactive agents at the inclusion. Thirty-four patients (19 males and 15 females) with a mean age of 61 $\pm$ 17 years were included in the study due to the presence of hypotension ($n = 17$; 50%), oligoanuria or acute renal failure ($n = 6$; 18%) or clinical and laboratory signs of dehydration ($n = 13$; 38%).

Seventeen (50%) patients were considered to be responders with a $\geq$ 12% increase of CO after saline infusion. The characteristics of the two groups were identical (Table 1). An increase of CO or SV by 5% or more during PLR distinguished responders from nonresponders with high sensitivity, specificity and positive predictive

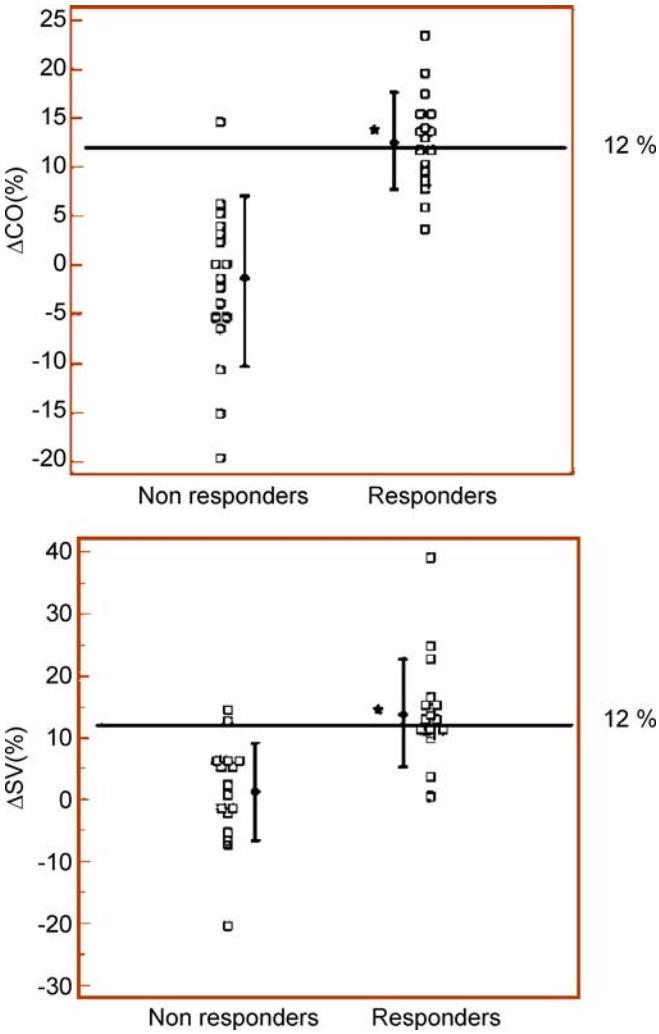

Fig. 1 Changes of cardiac output and stroke volume induced by PLR (expressed as percentage from baseline) in responders and nonresponders. *ΔCO*, Variation of cardiac output between baseline and after passive leg raising; *ΔSV*, variation of stroke volume between baseline and after passive leg raising. $^{*}p < 0.05$ vs. nonresponders

Table 1 Characteristics of the study population

	Global population ($n = 34$)	*Responders* ($n = 17$)	*Nonresponders* ($n = 17$)
Age (years), mean $\pm$ SD	61 $\pm$ 17	64 $\pm$ 17	58 $\pm$ 18
Male, *n* (%)	19 (56)	12 (63)	7 (36)
MacCabe group 0/1/2, *n*	18/14/2	6/10/1	12/4/1
SAPS II, mean $\pm$ SD	39 $\pm$ 16	42 $\pm$ 16	36 $\pm$ 17
Medical/surgical admission, *n* (%)	32 (94)/2 (6)	15 (89)/2 (12)	17 (100)/0 (0)
Medical history, *n* (%):			
Hypertension	17 (50)	7 (41)	10 (59)
Diabetes mellitus	7 (20)	2 (12)	5 (29)
Cardiomyopathy:	13 (38)	7 (41)	6 (35)
- Ischemic	11 (32)	6 (35)	5 (29)
- Hypertensive	4 (12)	1 (6)	3 (18)
COPD	7 (20)	6 (35)	1 (6)
Pulmonary embolism	1 (3)	0 (0)	1 (6)

SAPS II, Simplified Acute Physiology Score II; *COPD*, chronic obstructive pulmonary disease

Table 2 Accuracy of cardiac output and stroke volume changes after passive leg raising to predict fluid responsiveness

Criterion	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Positive likelihood ratio	Negative likelihood ratio	Rate of correct classification
ΔCO > 5%	94%	83%	83%	94%	6	0.1	88%
ΔSV > 5%	88%	67%	70%	86%	3	0.2	76%
ΔCO > 8%	81%	89%	87%	84%	7	0.2	88%
ΔSV > 8%	88%	83%	82%	88%	5	0.1	85%
ΔCO > 10%	69%	89%	85%	76%	6	0.3	82%
ΔSV > 10%	81%	83%	81%	83%	5	0.2	85%
ΔCO > 12%	63%	89%	83%	73%	6	0.4	76%
ΔSV > 12%	69%	89%	85%	76%	6	0.4	82%

ΔCO, variation of cardiac output between baseline and after passive leg raising; *ΔSV*, variation of stroke volume between baseline and after passive leg raising

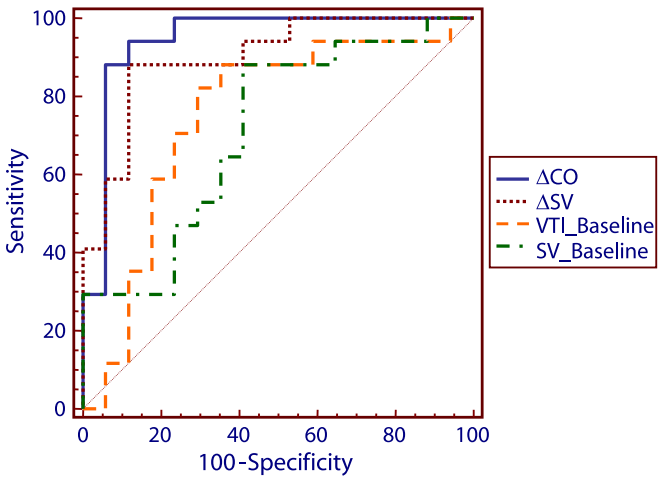

Fig. 2 Receiver operating curves to discriminate responders and nonresponders to volume expansion. *ΔCO*, Variation of cardiac output between baseline and after passive leg raising; *ΔSV*, variation of stroke volume between baseline and after passive leg raising; *AUC*, area under receiver operating curve ± standard error

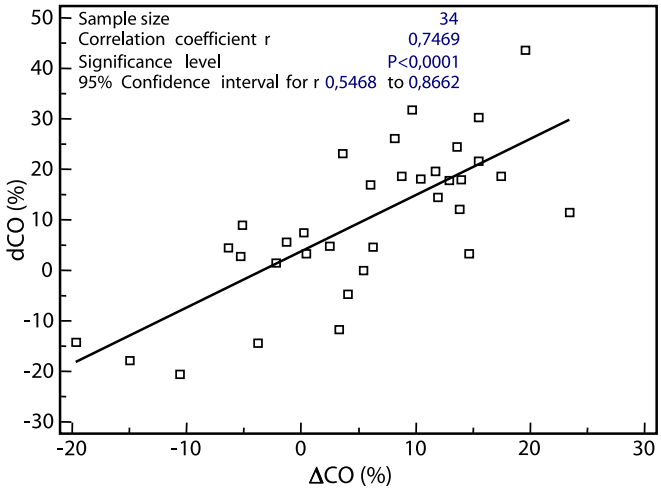

Fig. 3 Relations between changes in CO during passive leg raising (*ΔCO*) and the changes in CO after volume expansion (*dCO*). *dCO*, Variation of cardiac output between baseline and after volume expansion; *ΔCO*, variation of cardiac output between baseline and after passive leg raising

Table 3 Pearson correlation coefficient and area under the receiver operating curve of the different parameters

	Correlation coefficient (*p value*)	*AUC* ± *SE*
SV baseline	–0.38 (0.03)	0.70 ± 0.09
VTI baseline	–0.39 (0.02)	0.75 ± 0.08
ΔCO	0.75 (0.0001)	0.95 ± 0.04
ΔSV	0.56 (0.0006)	0.90 ± 0.06

AUC, area under the receiver operating curve ± standard error; *SV*, stroke volume; *VTI*, velocity time integral of aortic blood flow; *CO*, cardiac output; *Δ*, Evolution between baseline and passive leg raising

value. From a practical point of view, however, taking into account the intraobserver reproducibility, a cut-off of 12% should be proposed with a fair specificity, sensitivity and predictive values (Fig. 1, Table 2). The highest AUC was found for ΔCO (0.89 ± 0.06; 95% confidence interval 0.73–0.97) and ΔSV (0.9 ± 0.06; 95% confidence interval 0.74–0.97) (Fig. 2, Table 3). A significant correlation was observed only between (1) baseline value of VTI and SV, and variations in SV and CO during PLR; and (2) changes in CO after fluid challenge (Table 3, Fig. 3), but not between any other variable and changes in CO after fluid challenge. In contrast, neither absolute value nor changes in blood pressure were predictive of an increase in SV of fluid infusion.

Heart rate, pressures and echocardiographic indices at baseline, during PLR, and after fluid challenge are shown in Table 4. No difference between responders and nonresponders was demonstrated at baseline. After fluid infusion, SAP, SV and CO increased in the responder group, but remained stable in the nonresponder group except for VTI and SV.

Table 4 Evolution of hemodynamic parameters at baseline, after PLR and fluid challenge in responders and nonresponders

	Responders ($n=17$)	*Nonresponders* ($n=17$)	*p*
SAP, mmHg			
Baseline	106 ± 31	116 ± 30	ns
PLR	111 ± 27	121 ± 28	ns
Volume expansion	115 ± 27*	117 ± 23	ns
DAP, mmHg			
Baseline	57 ± 17	57 ± 17	ns
PLR	62 ± 17*	60 ± 17	ns
Volume expansion	58 ± 12	59 ± 15	ns
MAP, mmHg			
Baseline	73 ± 20	76 ± 20	ns
PLR	78 ± 19*	81 ± 18	ns
Volume expansion	77 ± 16	78 ± 16	ns
HR, beats/min			
Baseline	92 ± 19	87 ± 22	ns
PLR	90 ± 20	85 ± 21	ns
Volume expansion	92 ± 20	85 ± 21	ns
VTI, cm			
Baseline	17 ± 3	21 ± 5	0.02
PLR	20 ± 3*	21 ± 5	ns
Volume expansion	21 ± 4*†	21 ± 5	ns
SV, ml			
Baseline	51 ± 10	61 ± 14	0.02
PLR	58 ± 10*	62 ± 15	ns
Volume expansion	62 ± 12*†	62 ± 15	ns
CO, l/min			
Baseline	4.6 ± 1.2	5.3 ± 1.6	ns
PLR	5.2 ± 1.4*	5.2 ± 1.7	ns
Volume expansion	5.6 ± 1.5*†	5.2 ± 1.8	ns

Values are expressed as mean ± SD.
PLR, passive leg raising; *SAP*, systolic arterial pressure; *DAP*, diastolic arterial pressure; *MAP*, mean arterial pressure; *PP*, pulse pressure; *HR*, heart rate; *VTI*, velocity time integral of aortic blood flow; *SV*, stroke volume; *CO*, cardiac output
* $p<0.05$ vs. baseline; † $p<0.05$ vs. PLR

Discussion

This study in spontaneously breathing patients with suspected hypovolemia shows that changes in CO or SV during PLR are predictive of central hypovolemia. An increase by more than 12% of cardiac output or SV during PLR was predictive of a positive hemodynamic response after fluid expansion. Therefore, echocardiographic assessment of CO or SV during PLR appears to be reproducible and easy to perform at the bedside. This reversible test avoided fluid expansion in patients in whom it would be harmful.

Many studies have analyzed postural hemodynamic changes to confirm suspected hypovolemia, but the accuracy of previously described clinical signs to diagnose hypovolemia not due to blood loss is very low [1]. Lack of information concerning this population was emphasized in a review published in 1999 [1].

Leg raising was very recently used in selected critically ill patients [7–9]. Monnet et al., using esophageal Doppler, found that an increase in aortic blood flow by more than 12% was predictive of a greater than 15% increase in CO after fluid expansion [8]. Similarly Lafanechere et al. reported that an increase in aortic flow greater than 8% during PLR predicted an increase in aortic blood flow after volume infusion [9]. Both studies were performed in intubated and mechanically ventilated patients and used esophageal Doppler to measure CO [9]. These findings observed in mechanically ventilated patients cannot be extrapolated to nonsedated and nonintubated patients. No information is available concerning the effect of PLR in nonintubated patients. Negative intrathoracic pressure may change the amount of increase in blood central volume and may change baroreceptor and hemodynamic response. In addition, arterial tone is modified during sedation and may change responses to volume increase or decrease. Therefore this concept should be validated in spontaneously breathing patients. Our study was performed in nonintubated patients without any sedation with suspected hypovolemia, and therefore the findings may be applied to patients in whom clinicians wonder whether fluid infusion would improve their hemodynamics and clinical status.

In a previously published study using this concept, esophageal Doppler was used, and this method is far from a gold standard method to assess CO. In addition, the technique cannot be used in nonintubated patients. Echocardiography is a simple and noninvasive method to

evaluate hemodynamics in unstable patients and can be safely used in nonintubated and nonsedated patients [10]. It is widely available and can be used in many hospitals. CO measurement using this method has been extensively validated and can be conducted at the bedside [11].

Lastly, PLR did not induce alterations in heart rate, suggesting the absence of catecholamine stimulation related to the maneuver in awake patients.

This study presents a number of limitations or rise concerns. Firstly, the number of patients appears to be low, but comparable to the sample sizes of other published studies in this field [1, 7, 8] and this study should be considered to be a pilot study. Secondly, the echocardiographic method is not readily available 24 hours a day, 7 days a week, in many centers. Third, we did not assess hemodynamics at a second baseline after PLR and before fluid infusion. This may explain why some patients experienced a high CO at baseline due to the associated stress, which decreased during the procedure, explaining the lower CO in some nonresponders after fluid challenge.

Despite these limitations, this study is the first to demonstrate in nonintubated patients that hemodynamic changes assessed by using a noninvasive, well-validated technique during a simple maneuver may predict the hemodynamic effect of fluid infusion.

Conclusion

In spontaneously breathing patients with suspected hypovolemia, cardiac output or stroke volume measurement using echocardiography during passive leg raising can very accurately discriminate patients who will obtain a hemodynamic benefit from fluid challenge.

Acknowledgements. Grants from PHRC (Programme Hospitalier de Recherche Clinique) were obtained to conduct this clinical study.

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